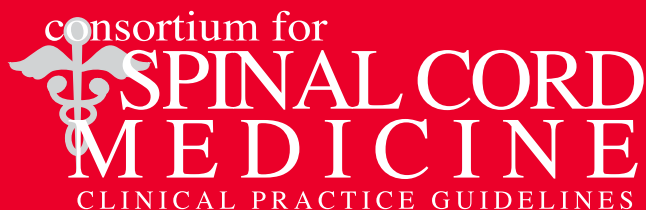


Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury:

**A Clinical Practice Guideline
for Health-Care Professionals**

SECOND EDITION



Consortium for Spinal Cord Medicine Member Organizations

Academy of Spinal Cord Injury Professionals

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Christopher and Dana Reeve Foundation

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International Spinal Cord Society

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Rick Hansen Institute

Society of Critical Care Medicine

U. S. Department of Veterans Affairs

United Spinal Association

CLINICAL PRACTICE GUIDELINE

Spinal Cord Medicine

Pressure Ulcer Prevention and Treatment Following Injury: A Clinical Practice Guideline for Health-Care Providers

SECOND EDITION



Consortium for Spinal Cord Medicine

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This guideline has been prepared based on scientific and professional information available in 2014. Users of this guideline should periodically review this material to ensure that the advice herein is consistent with current reasonable clinical practice. The websites noted in this document were current at the time of publication; however, because web addresses and the information contained therein change frequently, the reader is encouraged to stay apprised of the most current information.

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Foreword

Pressure ulcers are a frequent, costly, and potentially life-threatening complication of spinal cord injury (SCI). They complicate the rehabilitation process and are a significant deterrent to participation in activities that contribute to independent, productive, and satisfying lives. Pressure ulcers result in prolonged hospitalizations, delayed community reintegration, reduced quality of life, and loss of self-esteem. The etiology of pressure ulcers is complex and multidimensional. Biochemical, mechanical, and environmental and contextual factors interact at various times to damage tissue. Clinicians and researchers focus on unrelieved pressure, shear, friction, moisture, poor nutrition, immobility, and psychological, social, and economic factors, such as drug abuse, depression, inadequate personal and financial resources, and non-compliance to acknowledged preventive behaviors as the most significant aspects of pressure ulcer development. Every person with SCI is at risk for the development of pressure ulcers and almost all will develop at least one serious pressure ulcer during their lifetime.

The prevention and management of pressure ulcers are processes that are inextricably linked across the continuum of care of individuals with SCI. Current prevention programs, specifically hospital-based education interventions, have had limited success in reducing the occurrence of pressure ulcers especially after these individuals return to their families and community. This may be due, in part, to very short hospital stays, currently 3 to 4 weeks, in many rehabilitation facilities, resulting in extremely limited pressure ulcer prevention education. Furthermore, despite the plethora of education and treatment programs and protocols described in the literature, few have been validated for their ability to promote the preventive behaviors that reduce the occurrence or recurrence of pressure ulcers, especially after the person has returned to his or her home and community where out-patient educational resources are very limited.

Since the publication of the original clinical practice guideline *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals (2000)*, a number of present scientific studies have advanced our knowledge of the factors that contribute to the formation of pressure ulcers and have provided new directions for improving preventive techniques and treatment. The purpose of this new/updated clinical practice guideline is to present the current state of the science in pressure ulcer research and clinical practice and scientifically sound strategies that are effective in identifying risk and reducing the incidence, prevalence, and recurrence of this lifelong complication of SCI. More than 225 new articles specific to pressure ulcers among persons with spinal cord injury have been reviewed and graded. The recommendations in this guideline cover a broad spectrum of issues that have been addressed by the new multidisciplinary pressure ulcer clinical practice guideline development panel and several consultants. The significant constructs of this problem are risk and risk assessment; prevention strategies across the continuum of care; assessment and reassessment, following the onset of a pressure ulcer, of the individual with a pressure ulcer and of the ulcer itself; nonsurgical and surgical treatments interventions and their complications; and pressure redistribution and support surfaces and positioning for managing tissue loads for the bed and wheelchair.

The recommendations are based on an extensive review and analysis of the available scientific literature and represent the most current understanding of the interventions applied in clinical practice. Where the scientific literature failed to provide guidance in the development of this document, the panel

members based their recommendations on expert consensus. The panel was conscientious in identifying areas where knowledge gaps exist so that future research can be directed toward enhancing prevention and efforts.

The guideline is designed to be used by physicians in a number of specialties (including internal medicine, plastic surgery, and physical medicine and rehabilitation), nurses, physical and occupational therapists, social workers, and psychologists. It also may be useful to individuals with SCI, their families, and significant others, although the original consumer guide will be updated as well. Additionally, this guideline has implications for administrators, personal care attendants, third-party payers, and those who direct public policy.

The Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury Clinical Practice Guideline, 2nd Edition is the result of a collaborative effort among a group of professionals with extensive experience in studying and treating pressure ulcers. Their dedication is reflected in the pages of this document.

Susan L. Garber, MA, OTR, FAOTA, FACRM
Panel Chair

Preface

As chairman of the Steering Committee of the Consortium for Spinal Cord Medicine, it is a great pleasure for me to introduce the revision of the clinical practice guideline *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury*. The initial clinical practice guideline for *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury* was published in 2000. We were very fortunate to have the chairman of the initial guideline, Susan L. Garber, MA, OTR, FAOTA, return to spearhead the revision of this critically important topic.

These guidelines provide comprehensive recommendations for the prevention, assessment and management of pressure ulcers. Pressure ulcers, a secondary complication for individuals of all ages with spinal cord injuries, are unfortunately too common. Pressure ulcers are associated with significant morbidity and mortality throughout the lifespan of those with spinal cord injuries. In order to improve the quality of life and participation of individuals with spinal cord injuries, prevention and timely management of pressure ulcers are critical. The prevention of pressure ulcers begins within the first few hours after an injury and continues throughout the lifespan.

On behalf of the Consortium and Steering Committee, I want to acknowledge Susan Garber's expert, passionate, and committed leadership and our distinguished guideline development panel. Each panel member brought to the guideline development process an immense amount of energy and dedication for the care of people with spinal cord injuries.

Special thanks also goes to the representatives of the Consortium's 22 member organizations who thoughtfully and critically reviewed this draft in its various forms. Their contributions were essential to making this document one that will improve both the quality of care and quality of life for persons with spinal cord injury.

The development of this clinical practice guideline is dependent upon the exceptional administrative support and other services provided by Paralyzed Veterans of America. The Consortium is profoundly grateful to Paralyzed Veterans of America, led by 2015 National President Al Kovach, and to the Paralyzed Veterans of America Research and Education Department. Maureen Simonson, director of Research and Education, and Kim S. Nalle, manager of Clinical Practice Guidelines, were instrumental during all aspects of the development of these guidelines, from inception of the topic to dissemination. We could not have done it without them.

Lawrence C. Vogel, MD
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Acknowledgments

As Paralyzed Veterans of America continues its vital role sponsoring the development of clinical practice guidelines, much is owed to the hard work and extensive experience of the Research and Education Department, composed of Maureen Simonson, director; Marietta Jimmerson, grants portfolio manager; and Kim S. Nalle, manager clinical practice guidelines. Without their hard work and tireless ability to guide this complicated process, the update of this clinical practice guideline, could not have been completed.

We would like to acknowledge Attorney William H. Archambault for conducting a comprehensive analysis of the legal and health policy issues associated with this complex, multifaceted topic.

We extend our appreciation to the Communications Department of Paralyzed Veterans and Timothy Merrill for their excellent technical review and editing of this clinical practice guideline and to Project Design Company for design of this publication.

Appreciation is expressed to the Paralyzed Veterans Board of Directors and senior officers, including National President Al Kovach; Immediate Past President Bill Lawson; Executive Director Homer S. Townsend, Jr.; Associate Executive Director Lana McKenzie; and Director of Research and Education Maureen Simonson.

We have been supported in this work by many unnamed colleagues who have reviewed sections of the guideline and made helpful suggestions. Thank you.

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Summary of Recommendations

Risk and Risk Assessment

1. Conduct an assessment of pressure ulcer risk factors in individuals with SCI at every appropriate opportunity.
 - Assess and document risk on admission and reassess on a routine basis, as determined by the health-care setting, institutional guidelines, and changes in the individual's health status.
 - Demographic
 - SCI-related, such as incontinence
 - Comorbid medical
 - Nutritional
 - Psychological, cognitive, contextual, and social
 - Support surface for bed, wheelchair, and all durable medical equipment (DME) surfaces, such as shower/commode chair or bathroom equipment related
 - Use both a validated risk-assessment tool and clinical judgment to assess risk.

Prevention Strategies Across the Continuum of Care

2. Implement pressure ulcer prevention strategies as part of the comprehensive management of acute and chronic SCI and review all aspects of risk when determining prevention strategies.
 - Initiate pressure redistribution as soon as emergency medical conditions and spinal stabilization status allow.
3. Conduct daily comprehensive visual and tactile skin inspections with particular attention to the areas most vulnerable to pressure ulcer development, including, but not limited to, the following:
 - Ischial tuberosities
 - Sacrum
 - Coccyx
 - Greater trochanters
 - Ankles (malleoli)
4. Turn or reposition individuals with SCI initially every 2 hours in the acute and rehabilitation phases if the medical condition allows.
 - Knees (medial aspect especially during side-lying position)
 - Occiput
 - Calcaneous
5. Evaluate the individual and his or her support surface environment for optimal maintenance of skin integrity.
 - Avoid overstretching and folding of skin/soft tissues while positioning and shearing when individuals are repositioned or transferred.
 - Avoid positioning individuals who are side-lying in bed directly on their trochanters.
 - Prevent moisture accumulation and temperature elevation at the skin-support surface interface.
 - Utilize pressure redistribution support surfaces preventively to protect soft tissues from injury.
 - When off-loading the calcaneus with pillow or cushion, ensure the proper position of the pillow or cushion. It should be placed lengthwise under the lower extremity from the malleolus to the knee, thus lifting the calcaneus off the bed surface.
 - Do not use donut-type devices.
 - Monitor the performance, i.e., continued effectiveness, of support surfaces for the bed and wheelchair specific to pressure ulcer prevention.
6. Provide an individually prescribed seating system designed to redistribute pressure.
 - Employ a power weight-shift system when manual pressure redistribution is not possible.
7. Implement an ongoing exercise regimen to promote maintenance of skin integrity and prevent contractures.
8. Assess nutritional status, including dietary intake, anthropometric measurements, biochemical parameters (prealbumin, total protein, albumin, hemoglobin, hematocrit, and total lymphocyte count) fasting blood sugar, liver function panel, folate, and vitamin B12.

9. Provide adequate nutritional intake to meet individual needs, especially for calories (or energy), protein, micronutrients (zinc, vitamin C, vitamin A, and iron), and fluids.
10. Provide individuals with SCI, their families, significant others, and health-care professionals with specific information on effective strategies for the prevention and treatment of pressure ulcers. This should include the following:
 - Pressure ulcer etiology
 - Reducing pressure ulcer risk
 - Skin cleansing and care techniques
 - Management of incontinence
 - Frequency and techniques of skin inspection
 - Frequency, duration, and techniques of recommended position changes
 - Frequency, duration, and techniques of recommended pressure redistribution
 - Nutrition as it relates to maintaining skin integrity
 - Use and maintenance of support surfaces (mattresses and cushions)
 - Skin changes to be reported to the health-care team

Assessment and Reassessment Following Pressure Ulcer Onset

Assessment of the Individual with a Pressure Ulcer

11. Perform an initial comprehensive assessment of the individual with a pressure ulcer, to include the following:
 - Complete history and physical examination
 - Complete skin assessment
 - Laboratory tests (evaluate for infection and nutritional status)
 - Psychological health, behavior, cognitive status, and social and financial resources
 - Availability and utilization of personal care assistance
 - Positioning, posture, and equipment
 - Nutritional status

- Activities of daily living (ADLs), mobility, and transfer skills, as related to maintaining skin integrity

Assessment and Reassessment of the Pressure Ulcer

12. Describe and document in detail an existing pressure ulcer and its treatment. Include the following parameters:
 - Anatomical location and general appearance
 - Category/Stage
 - Characteristics of the wound base
 - Viable tissue (granulation, epithelialization, muscle, bone, or subcutaneous tissue)
 - Nonviable tissue (necrotic, slough, eschar)
 - Size of wound—length x width x depth
 - Exudate amount and type
 - Odor
 - Wound edges
 - Periwound skin
 - Wound pain
 - Documentation of current treatment strategies and outcomes to date.
13. Monitor, assess, document, and report any observable/visible change in wound status.
 - Monitor the pressure ulcer with each dressing change or if there is no dressing, then routinely.
 - Conduct a comprehensive assessment as described in recommendation 12 at regular intervals

Treatment

Nonsurgical

CREATING A PHYSIOLOGIC WOUND ENVIRONMENT

14. Cleanse pressure ulcer with each dressing change without harming healthy tissue on the wound bed:
 - Use normal saline, sterile water, pH-balanced wound cleansers, or lukewarm potable tap water.
 - Use diluted sodium hypochlorite $\frac{1}{4}$ strength to $\frac{1}{2}$ strength solution for wounds with heavy bioburden for limited time only, until clinical evidence of bioburden is resolved.

- Use the following mechanical wound cleansing techniques to remove wound debris, exudates, surface pathogens, bacteria, and residue from topical creams and ointments.

- 4–15 pounds per square inch (psi) pressure irrigation with angiocatheter attached to syringe, spray bottle, or pulsatile lavage.
- Gentle scrubbing of the wound bed with wet gauze.

- Cleanse periwound skin with normal saline, sterile water, pH-balanced skin cleanser, or lukewarm potable tap water with dressing changes.

DEBRIDEMENT

15. Debride devitalized tissue using a method or a combination of debridement methods appropriate to the ulcer's status.

- Debride eschar and devitalized tissue with the exception of a stable heel eschar.
- Debride areas in which there is unstable eschar and devitalized tissue.

SELECTION OF WOUND CARE DRESSING

16. Use a dressing that achieves a physiologic local wound environment that maintains an appropriate level of moisture in the wound bed:

- Control exudate
- Eliminate dead space
- Control odor
- Eliminate or minimize pain
- Protect the wound and the periwound skin
- Remove nonviable tissue
- Prevent and manage infection

ELECTRICAL STIMULATION

17. Use electrical stimulation (ES) to promote closure of category/stage III or IV pressure ulcers, unless contraindicated in the cases of untreated, underlying osteomyelitis or infection.

ONGOING MONITORING AND MODIFICATION OF TREATMENT PLAN

18. Modify the treatment plan if the ulcer shows no evidence of healing within 2 to 4 weeks. Review individual factors associated with non-healing of pressure ulcers, such as the following:

- Incontinence
- Infection
- Carcinoma

- Abnormal wound healing
- Nutrition
- Medication
- Support surfaces
- Transfers
- Noncompliance

Surgery for Pressure Ulcers

REFERRAL FOR PRESSURE ULCER SURGERY

19. Refer individuals with deep category/stage III and category/stage IV pressure ulcers for operative intervention. For persons deemed appropriate candidates for surgical reconstruction, adhere to the following tenets of surgical treatment:

- Reverse any pressure ulcer risk factor if possible (e.g., impaired nutritional status) and address pre-op medical risk.
- Prior to surgery, treat osteomyelitis or cellulitis. This may need to be combined with excision of infected bone during surgery.
- Fill dead space and enhance the blood supply of the healing wound by mobilizing well-vascularized soft tissues flaps.
- Contour bony prominences to yield larger, flatter surfaces to augment pressure distribution.
- Reconstruct soft tissue defects with large regional pedicle flaps, placing suture lines as far away from the area of direct pressure as possible and with minimum tension. Avoid encroaching on adjacent flap territories.
- Preserve options for future potential breakdowns.

PREOPERATIVE ASSESSMENT

20. Address the following factors to enhance the effectiveness of pressure ulcer surgery:

- Presence of osteomyelitis
- Wound bioburden
- Nutritional status
- Bowel and Bladder management
- Spasticity and contracture
- Heterotopic ossification
- Comorbid medical conditions
- Anesthesia
- Previous ulcer surgery
- Smoking Cessation
- Urinary tract infection

Pressure Redistribution and Support Surfaces

Bed Positioning

21. Use bed positioning devices and techniques that are compatible with the bed type and the individual's health status.
 - Avoid positioning individuals directly on pressure ulcers regardless of the pressure ulcer anatomical location (trochanter, ischium, sacrum, and heel) unless such position is necessary for performance of ADLs, such as eating or hygiene.
 - Use pillows, cushions, and positioning aids to reduce pressure on existing pressure ulcers or vulnerable skin areas by elevating them away from the support surface.
 - Avoid closed cutouts or donut-type cushions.
 - Prevent contact between bony prominences.
 - Elevate the head of the bed no higher than 30 degrees unless medically necessary.
 - Reposition individuals in bed at least every 2 hours.

Bed Support Surfaces

22. Use pressure-redistribution bed support surfaces for individuals who are at risk for or who have pressure ulcers (see Table 1: Support Surfaces).
 - Select a reactive support surface for individuals who are able to reposition themselves enough to avoid weight bearing on all areas at risk for pressure ulceration and who have a stable spine.
 - Select an active support surface for individuals who are unable to reposition themselves.
 - Select an active support surface or a high air-loss (air-fluidized) reactive support surface for individuals who have pressure ulcers on multiple turning surfaces and/or are status post flap/skin graft within the past 60 days.

Wheelchair Positioning – Pressure Redistribution Surfaces

23. Prescribe wheelchairs and seating systems specific to the individual that allow that individual to redistribute pressure sufficiently to prevent the development of pressure ulcers.
 - Obtain specific body measurements for optimal selection of seating system dimensions (postural alignment, weight distribution, balance, stability, and pressure redistribution capabilities).
 - Prescribe a power weight-shifting wheelchair system for individuals who are unable to independently perform an effective pressure relief.
 - Use wheelchair tilt-in-space and/or recline devices effective enough to offload tissue pressure.
 - Use standing wheelchairs to remobilize individuals with existing pelvic pressure ulcers.
 - Full-time wheelchair users with pressure ulcers located on a sitting surface should limit sitting time and use a gel or air surface that provides pressure redistribution.
 - Maintain an offloaded position from the seating surface for at least 1 to 2 minutes every 30 minutes.
24. Prescribe wheelchair seating systems for each person with a spinal cord injury individualized to anthropometric fit, to provide optimal ergonomics, and to provide maximal function. Prescribe wheelchair seating systems that—
 - Redistribute pressure
 - Minimize shear
 - Provide comfort and stability
 - Reduce heat and moisture
 - Enhance functional activity
 - Inspect and maintain all wheelchair cushions at regular scheduled intervals.
 - Replace wheelchair seating systems that are no longer effective.
25. Prescribe padded toilet and bathing durable medical equipment items for pressure redistribution and skin protection during use.
26. Prescribe skin protection devices and pressure redistribution seating systems for use with recreational equipment, other motorized or manually powered vehicles, and specialty wheelchairs.

The Consortium for Spinal Cord Medicine

The Consortium is a collaboration of professional and consumer organizations funded and administered by PVA. The Steering Committee, administratively supported by PVA's Research and Education Department, is comprised of one representative from each Consortium-Member Organization. The Consortium's mission is to direct the development and dissemination of evidence-based CPGs and companion consumer guides. This mission is solely directed to improving the health care and quality of life for persons with SCI/D.

Guideline Development Process

The process used to develop the guidelines is based on the model derived from the Agency for Health Research and Quality (AHRQ). The model is—

- Interdisciplinary, to reflect the multiple information needs of the spinal cord medicine practice community.
- Responsive, with a well-managed timeline for completion of each guideline.
- Reality-based, making use of the scientific literature where it exists and using practical and clinical expertise where there are gaps in the scientific literature.

This innovative and cost-efficient approach recognizes the specialized needs of the United States' spinal cord injury medical community, encourages the participation of both payer representatives and consumers, and emphasizes utilization of graded evidence drawn from the international scientific literature.

The Consortium's CPG development process involves specific review steps to ensure scientific and liability integrity for Steering Committee Members, Panelists, and field reviewers. This process involves extensive field review as well as a legal review against copyright, trademark, and restraint of trade issues.

Methodology

An independent scientific consulting firm is contracted by PVA to provide methodological support for evidence grading. This Methodology Team has three (3) main functions:

- (1) conducting a review of the literature;
- (2) grading of scientific evidence and conducting meta-analyses; and
- (3) drafting the methodological section for the guideline document.

After a review of the literature is conducted, each recommendation receives two objective ratings:

- 1) "Scientific evidence"—this reflects the level of evidence rating for each article cited in the rationale and
- 2) "Grade of recommendation"—this is calculated based on the scientific evidence. Additionally, each recommendation receives a subjective rating, "Strength of panel opinion."

Note: The grading of recommendations may change based on best practices consistent with specific scientific principles appropriate to each subject matter.

Literature Review and Drafting of Recommendations

After a CPG topic is explicated and reviewed, Panel Members provide search terms and parameters relating to their sub-topic to the Methodology Team to PVA's Research and Education Department. PVA will transmit the search terms and parameters to the Methodology Team who will then be responsible for providing PVA's Research and Education Department journal article citations based on the specific search terms and parameters. PVA's Research and Education Department will then transmit these citations to Panel Members. Panel members must also note knowledge gaps and unique features of the sub-topic literature. While Panel Members are reviewing journal articles cited by the Methodology Team, they are to individually write sub-topic recommendations and supporting rationales according to each area of the Panel Members' expertise. A few select opinion-based recommendations may be suggested to provide sub-topic continuity; however, for the most part,

recommendations and supporting rationales will reflect the scientific literature review. Conversing with other Panel Members is encouraged regarding proposed recommendations as long as individual recommendations are put forward to be discussed in the larger Panel discussion. Panel members are also responsible for providing any rewrites/edits of their individual sections as decided during full Panel discussions.

Rating the Scientific Evidence

The Methodology Team conducts a review of the literature based on parameters developed by the Panel for each CPG topic. The product of the Methodology Team's literature review is a graded list of relevant scientific publications that fall within the established search parameters. PVA's Research and Education Department is then provided with the list of graded articles by the Methodology Team.

Panel Members are not limited to the use of the articles retrieved initially in the review process. If Panel Members wish to use literature that falls outside the search parameters, this literature will be graded by the Methodology Team and included in the CPG as deemed appropriate by the Panel.

The Methodology Team begins its grading by employing the hierarchy first discussed by Sackett (1989) and later enhanced by Cook et al. (1992) and the U.S. Preventive Health Services Task Force (1996), presented in Table 1. Additionally, each study is evaluated for internal and external validity. Factors affecting internal validity (i.e., the extent to which the study provided valid information about the individuals and conditions studied) includes:

- Sample size and statistical power
- Selection bias and inclusion criteria
- Selection of control groups, if any
- Randomization methods and comparability of groups
- Definition of interventions and/or exposures
- Definition of outcome measures
- Attrition rates
- Confounding variables
- Data collection methods and observation bias
- Methods of statistical analysis

External validity (i.e., the extent to which the study findings were generalized to conditions other than the setting of the study) is evaluated

through an examination of the characteristics of the study population, the clinical setting and environment, and the investigators and providers of care. The resulting rankings, below, are provided to the panel members during the writing and deliberation process.

Levels of Scientific Evidence

- I. Large randomized trials with clear-cut results (and low risk of error)
- II. Small randomized trials with uncertain results (and moderate to high risk of error)
- III. Nonrandomized trials with concurrent or contemporaneous controls
- IV. Nonrandomized trials with historical controls
- V. Case series with no controls

Sources: Sackett, D.L. Rules of evidence and clinical recommendations on the use of antithrombotic agents. *Chest* 95 (2 Suppl) (1989): 25–45; and the U.S. Preventive Health Services Task Force. Guide to clinical preventive services: *An assessment of the effectiveness of 169 interventions*. Baltimore, MD: Williams and Wilkins, 1996.

These five levels of evidence do not directly describe the quality or credibility of evidence. Rather, they indicate the nature of the evidence being used. Decisions must often be made in the absence of published evidence. In these situations, it is necessary to use the opinion of experts based on their knowledge and clinical experience.

Rating of the evidence performed by Methodology Team may change depending on the best practices consistent with scientific principles appropriate to each subject matter.

Grading the Recommendations

Each recommendation is graded according to the level of scientific evidence supporting it by the Panel. The framework used is outlined in Table 2. These ratings represent the strength of the supporting evidence cited in the rationale for the recommendation, not the strength of the recommendation itself

Table 1. Categories of the Strength of Evidence Associated with the Recommendations

Category	Description
A	The guideline recommendation is supported by one or more level I studies
B	The guideline recommendation is supported by one or more level II studies
C	The guideline recommendation is supported only by level III, IV, or V studies

Sources: Sackett, D.L. Rules of evidence and clinical recommendations on the use of antithrombotic agents. *Chest* 95 (2 Suppl) (1989): 25–45; and the U.S. Preventive Health Services Task Force. Guide to clinical preventive services: *An assessment of the effectiveness of 169 interventions*. Baltimore, MD: Williams and Wilkins, 1996.

If the literature supporting a recommendation comes from two or more levels, the number and level of the studies are reported (e.g., in the case of a recommendation that is supported by two studies, one a level III, the other a level V, the “Scientific evidence” is indicated as “III/V”). In situations in which no published literature exists, consensus of the Panel Members and the Steering Committee’s recommended field reviewers is used to develop the recommendation and its rationale.

The rationale section supports the recommendation statement based on scientific evidence.

Each Panel Member votes based on his/her opinion on the strength of the recommendation (i.e., a vote taken at the final panel meeting). The level to which Panel Members agree on the strength of each recommendation is assessed as either low, moderate, or strong. Each Panel Member is asked to indicate his or her level of agreement on a 5-point scale, with 1 corresponding to neutrality and 5 representing maximum agreement. Scores are aggregated and an arithmetic mean is calculated. This mean score is then translated into low, moderate, or strong, as shown in Table 3. Panel Members may abstain from the voting process if they lack the expertise associated with a particular recommendation. After the Panel votes on the grading of the recommendation, the strength of the opinion will be added to the document.

Table 2. Levels of Panel Agreement with the Recommendation

Level	Mean Agreement Score
Low	1.0–2.32
Moderate	2.33–3.66
Strong	3.67–5.0

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Methods (Second Edition)

Specific to this clinical practice guideline, a systematic search of the medical literature was conducted in MEDLINE (via PubMed) and Embase to identify relevant English-language publications describing pressure ulcers in patients with spinal cord injuries. The search identified publications using specific keywords and Index Medicus subheadings (MeSH subheadings) in MEDLINE, or equivalent Emtree terms in Embase related to pressure ulcer or synonyms such as bedsores, and terms related to spinal cord injury or types of paralysis, such as paraplegia or quadriplegia. Keywords had to be located in the title or abstract of full-length publications, and studies had to be conducted in humans and published from January 1997 to October 2010.

Articles were included if they reported on patients aged 13 or older with spinal cord injuries. Publications reporting the results of randomized or non-randomized clinical trials, observational cohort studies, case-control studies, and case series were included. Included publications described studies of pressure ulcers of all categories/stages and any anatomical location, and at any stage of treatment. Articles were excluded if they did not report on an adult population with spinal cord injuries, or if they did not report studies conducted in humans. Articles also were excluded if they did not report on a patient population with pressure ulcers, or if they reported results for patients with non-traumatic paralysis. Letters, reviews, case reports, commentaries, “n-of-one” studies, editorials, and publications without abstracts also were excluded. Articles were included in the final review if they did not meet any of the exclusion criteria during full-text review. Subsequent to the systematic search conducted for literature published through October 2010, additional articles were recommended for inclusion in the clinical practice guidelines by the expert panel members.

Several guidelines exist to inform an evaluation of the quality of published research. Generally, these guidelines suggest a hierarchy of evidence based on study design, with systematic

reviews of randomized controlled trials (RCT) presenting the highest quality evidence, followed by individual RCTs, observational cohort studies, and case-series, with editorials/expert opinions representing the lowest-quality evidence.

A single researcher evaluated the level of evidence for each study using criteria from the University of Oxford’s Centre for Evidence-Based Medicine (CEBM) Levels of Evidence guidelines (2009).^{1,2} The guidelines from 2009 were used to grade articles recommended by the panel members in 2010; when the CEBM guidelines for determining the levels of evidence were updated in 2011, these guidelines were used to grade subsequent articles recommended for inclusion by the panel members. The 2011 levels of evidence guidelines were designed to be simpler to use than the previous version (from 2009), and to be more closely aligned with the process of clinical decision-making.^{2,3} The grading of the evidence focused on study design, but also was influenced by other measures of study quality, such as sample size, statistical analyses, imprecision, bias, and how well the analyses aligned with the research question of the study. Some publications were not graded according to these guidelines; these included websites, handbooks, and studies reporting the results of pre-clinical investigations, in vitro analyses, instrument validation, and guidelines or consensus statements.

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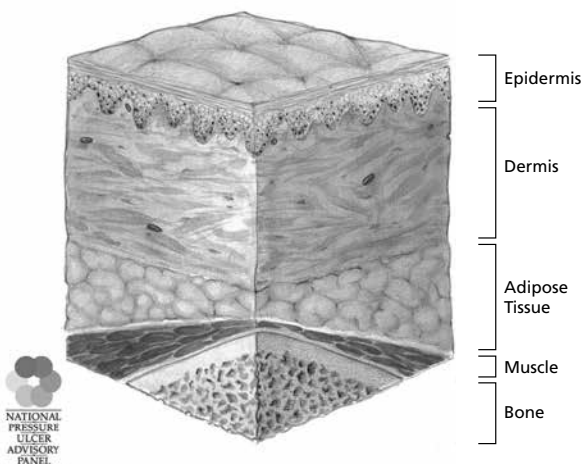
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Introduction

Normal Skin

Skin is the largest single organ of the body. Its main function is to isolate and protect the body from the environment. When the skin barrier is broken, it is no longer impregnable to environmental trauma. Skin also helps insulate the body, maintaining the core temperature within a healthy range as regulated by the autonomic nervous system. Blood flow from the interior of the body to a venous plexus immediately beneath the skin is the most efficient method for dissipating heat from the interior of the body to the skin surface. Cold receptors in the skin activate reflexes to raise the body temperature, if needed, in part by promoting vasoconstriction.

Skin consists of two layers: the epidermis and the dermis. The epidermis is the outermost layer, which is in a constant state of renovation, shedding old cells and acquiring new cells that move upward from the dermis. The dermis is a much thicker layer where hair cells, sebaceous and sweat glands, and nerve receptors are based; it is dense with capillaries. The dermis consists mainly of collagen whereas the epidermis has no collagen.



Changes in Skin after SCI

COLLAGEN SYNTHESIS AND DEGRADATION

Collagen is the principal component of the organic matrix of the dermis and is responsible for its tensile strength. Soon after spinal cord injury, increased levels of collagen metabolites are excreted through urine, a marker of collagen catabolism (Claus-Walker et al., 1977). This

excess excretion seems to cease during the second year after the injury, but often increases again with the development of a pressure ulcer (Rodriguez et al., 1989). A prospective, controlled study of 60 men with SCI who had a history of pressure ulcers showed an increase in the urinary excretion of two collagen metabolites. The metabolite characteristic of skin collagen was preferentially increased. The time elapsed from the start of increased excretion to the appearance of an ulcer in the epidermis ranged from 2 to 5 months (Rodriguez and Garber, 1994).

Type I collagen has the thicker, stronger fibrils, which are responsible for the great tensile strength of normal skin. Type III collagen has much thinner, weaker fibrils, with a certain degree of elasticity. After SCI, skin biopsies show a decrease in the proportion of type I to type III collagen in the skin below the level of SCI (Rodriguez and Markowski, 1995). This proportional increase in type III collagen contributes to the fragility of skin affected by denervation after SCI. Skin with greater tensile strength has a higher ratio of type I collagen to other types (Flint et al., 1984).

When skin biopsies taken below the level of injury are compared to skin biopsies taken from above the level of injury or from biopsies taken from individuals who have not experienced SCI, reductions have been found both in the total amino acid content of skin as well as the activity of enzymes involved in the biosynthesis of collagen within the skin. (Rodriguez and Claus-Walker, 1988).

ADRENERGIC RECEPTOR DENSITY

The density of adrenergic receptors in skin below the level of injury is decreased as compared to adrenergic receptors in skin above the level of injury (Rodriguez et al., 1986). This decrease in density of adrenergic receptors is likely related to changes seen in the vasomotor control of the skin, as the decrease in the density of adrenergic receptors seems to correlate with other symptoms of vascular dysfunction in SCI, such as the reduced blood supply and deficient circulation, as well as the impaired response of individuals with SCI to repeated surface pressure loads (Patterson et al., 1993).

CHANGES IN UNDERLYING SOFT TISSUE COMPOSITION AFTER SCI

Makhsous et al. (2008) used ultrasound to quantitatively measure changes in soft tissue

stiffness, thickness, and deformation at four different anatomical locations—the ischial tuberosity, greater trochanter, posterior mid-thigh, and biceps brachii—in both persons without SCI and in those with chronic SCI. Significant differences were observed within the various anatomical locations in both groups as well as between the two groups. Those with SCI were found to have significantly softer tissue over the ischial tuberosity and mid-thigh areas. This result may be attributable to denervation and atrophy of muscles leading to a greater proportion of fat to muscle.

CHANGES IN SKIN, SOFT TISSUE VASCULARITY, AND OXYGENATION AFTER SCI

Individuals with spinal cord injury have an altered autonomic nervous system, with the degree of alteration varying with the level and completeness of injury. Injury to the spinal cord interferes with this autonomic control and often is responsible for a person with SCI to be unable to compensate for extremes of cold and heat. For example, in persons with complete injuries, sweating is markedly decreased below the level of injury, resulting in a decrease in the body's natural cooling ability.

Deitrick and colleagues (2007) studied small vessel blood flow in the lower limbs of persons with SCI using duplex Doppler sonography of the common femoral artery and laser Doppler flowmetry of the foot. Cutaneous blood flow measured during both the supine and sitting positions was found to be decreased in persons with SCI as much as 50% or more while sitting as compared to blood flow in persons without SCI. This confirms what others have noted previously: that individuals with SCI have a reduced blood supply (Bennett et al., 1984) and reduced blood flow below the level of injury (Lindan, 1961).

Hagisawa et al. (1994) measured changes in blood content and oxygenation in superficial vessels of the skin following an applied pressure over the trochanter in individuals with and without spinal cord injury. No substantial differences were found in the reactive hyperemia duration and intensity between groups. However, there was a slower reflow rate after pressure was removed in the SCI group. This slower reflow rate was also noted by Schubert and Fagrell (1991), who used laser Doppler flow measurements to determine the response of skin blood cell flow after local pressure was applied over the sacrum and the gluteus maximus muscle in individuals with and without SCI. A smaller increase in temperature during occlusion

was found in the SCI group with no sensation over the sacrum, compared to individuals who had sensation over the sacrum or to the able-bodied group.

Studying transcutaneous oxygen tension, Liu reported the subjects with paraplegia, without pressure ulcers, had reduced tissue oxygenation below the level of the spinal cord lesion compared to ambulatory controls (Liu et al., 1999). Others have shown that under the same pressure load, individuals with SCI have a reduction of the transcutaneous oxygen tension five times the magnitude of the reduction measured in those who did not have a spinal cord injury (Hunt and Connally, 1978).

Pathophysiology of Pressure Ulcers

A pressure ulcer is defined as localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of direct pressure or shear (Black et al., 2007) and the resulting deformation of the underlying soft tissues (Scales, 1990). In addition to the necessary direct pressure or shear, there are multiple systemic, internal, and external factors that contribute to the development of pressure ulcers, including but not limited to such divergent factors as skin moisture level, nutritional factors, psychosocial, and cognitive issues. To complicate this further, even for the necessary factors of pressure and shear, there is no known threshold value above which a pressure ulcer will definitely occur, especially since other quantifying factors, such as the duration of pressure, all come into play.

Pressure ulcers result from the effect of gravity on the body mass in contact with a support surface. The forces at the contact point are defined as direct pressure if the force vector is perpendicular or shear pressure if the force vector is tangential to the tissue contact surface. The viscoelastic and microvascular properties of the tissue determine its response to these forces (Bader, 1990; Bogie et al., 1995; Reddy et al., 1981). Prolonged stress due to pressure in the tissue collagen network above the capillary and lymphatic vessel tolerance to remain patent (open) can result in occluded blood and interstitial fluid flow, ischemia, pain, necrosis, and sloughing of the dead tissues (Bennett et al., 1984; Schubert et al., 1994).

Histologic features of chronic pressure ulcers that have extended beyond the dermis into underlying tissues include an accumulation of fibrin on the inside edge, within which are

inflammatory cells and vacuolated fibroblasts (the cells that secrete the collagen). Fibrin appears to supplant the collagen matrix seen in intact tissues. Edema is also present near the surface of the ulcer, and there is often partial to full occlusion of blood capillaries (Vande Berg and Rudolph (1995).

Susceptibility of skin and soft tissue to pressure and shear

The first visible indication that a pressure ulcer is developing usually is a change in the skin surface or change in temperature or turgor in darker skinned individuals, which can be assessed by palpation. However repeated investigations have shown that muscle tissue is more sensitive than skin to pressure-induced ischemia and that this is where the tissue damage is initiated which ultimately leads to pressure ulcer development (Daniel et al., 1981; Nola and Vistnes, 1980; Salcido, 1994).

Vascular deformation by direct pressure and shear

Support surfaces contacting the body are for the great proportion of the contact surface, oriented obliquely at the contact area. This causes both direct pressure and shear stress to occur at the same time. The orientation of the blood vessels relative to the load-bearing skin surface determines the response of the vessel to the surface loads. In general, the major vessels and their branches are oriented either parallel or perpendicular to the skin surface. This pattern repeats for successive branches of the arterial and venous circulation (Agris and Spira, 1979). Vessels parallel to the surface collapse easily from pressure loads whereas vessels perpendicular to the surface bend and collapse from shear loads applied to the weight-bearing tissue. Vessels most vulnerable to occlusion by shear stress are those penetrating through the interfaces between the tissue planes. Blood flow to the distal capillaries is impaired when capillaries collapse and occlude as a result of tissue layers that slip and vessels that bend between tissue layers. Thus, both pressure and shear loads can cause ischemia and necrosis in the layers of the skin and subcutaneous tissues.

The deformative mechanical effect of external loads on tissue is resisted by the internal cellular and interstitial pressures and the strength of the collagen network within the tissue structure. With the application of external pressure, internal tissue pressure builds up first

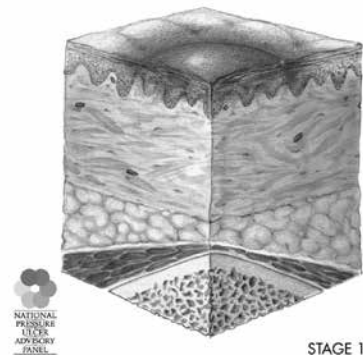
within the interstitial fluid trapped between the cells and the collagen network. Pressure gradients cause the fluid to move from the high- to low-pressure regions, causing local volume and contour change in the cells and supporting structural tissues stroma, which can lead to alterations in cellular metabolism (including collagen synthesis) and, if extreme, cell boundary and collagen network destruction (Reger et al., 1986; Reddy et al., 1981).

Category/Staging of Pressure Ulcers

Pressure ulcers are described by a category/staging system based on the extent of anatomical tissue loss. The category/staging system for pressure ulcers most commonly used is the consensus classification developed by the National Pressure Ulcer Advisory Panel in 1989 and updated in 2007 and illustrated below (Black et al., 2007).

CATEGORY/STAGE I

Category/Stage I damage is defined by non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; however, its color may differ from the surrounding area. The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Category/stage I damage may be difficult to detect in individuals with dark skin tones.

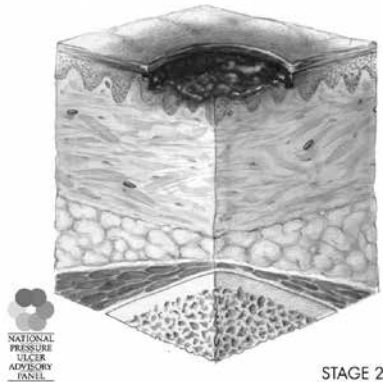


CATEGORY/STAGE II

Moisture or incontinence-associated dermatitis should not be confused with a pressure ulcer. The anatomical location of the lesion, e.g., whether or not it is located over bony prominence, can often help in making this determination.

A category/stage II ulcer is the partial loss of the dermis presenting as a shallow open ulcer with red pink wound bed, without slough. It also may present as an intact or open/ruptured serum-filled or sero-sanguineous-filled blister. It presents

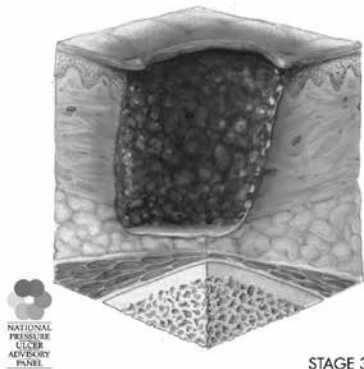
as a shiny or dry shallow ulcer without slough or bruising. The presence of bruising indicates deep tissue injury (see below) and the wound should not be staged as a category/stage II ulcer. This category should not be used to describe skin tears, tape burns, incontinence associated dermatitis, maceration, or excoriation.



STAGE 2

CATEGORY/STAGE III

A category/stage III ulcer is notable for full thickness skin loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are not exposed. Slough may be present but it does not obscure the depth of tissue loss. It may include undermining and tunneling. The depth of a category/stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have significant subcutaneous tissue, and here a category/stage III ulcer can be shallow. In contrast, areas of significant adiposity can develop extremely deep category/stage III pressure ulcers. Bone and tendon are not visible or directly palpable.

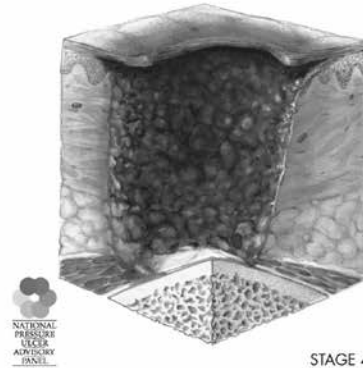


STAGE 3

CATEGORY/STAGE IV

A category/stage IV ulcer shows full thickness skin loss with exposed bone, cartilage, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. It often includes undermining and tunneling.

The depth of a category/stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have adipose or subcutaneous tissue and these ulcers are typically shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule), making osteomyelitis or osteitis likely to occur. Exposed tendon, bone, or muscle is visible or directly palpable.



STAGE 4

UNSTAGEABLE/UNCLASSIFIED

An unstageable/unclassified ulcer shows full thickness tissue loss in which the actual depth of the ulcer is obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined. When the true depth is determined, typically a category/stage III or IV ulcer will be revealed. Stable (dry, adherent, intact, with no erythema or fluctuance) eschar on the heels serves as the body's natural (biological) cover and should not be removed.

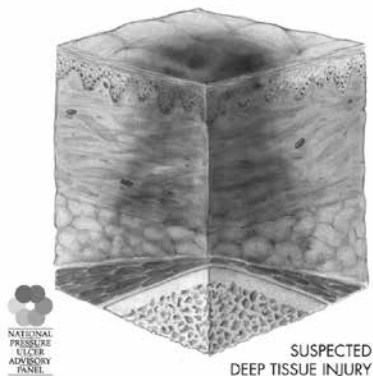


UNSTAGEABLE

SUSPECTED DEEP TISSUE INJURY

Suspected deep tissue injury (sDTI) may present as a purple or maroon localized area of discolored skin (which may otherwise appear

normal) or a blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler as compared to adjacent tissue. sDTI may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.



The Use of Staging in Clinical Practice

Staging of a pressure ulcer using the criteria outlined previously is widely accepted as a proxy for severity. However, it should be noted that the term “staging” is an anatomical description of a wound, rather than a physiological term, and that pressure ulcer staging is only appropriate for defining the maximum anatomic depth of tissue damage.

It is correct to restage a worsening pressure ulcer from visit to visit. If a patient with a known category/stage II pressure ulcer presents at the next visit with exposure of the adipose layer, where previously it had only been partial thickness skin loss, the ulcer should be restaged as a category/stage III ulcer. Similarly, if a previously assessed sDTI develops eschar, which precludes an assessment of the true ulcer depth, it should be restaged as unstageable.

The progressive numerical identification of staging can be misleading in that it seems to imply that a wound must progress sequentially through each category/stage. This is reinforced by the observation that a category/stage IV wound seems to exhibit more tissue damage than a similarly sized category/stage I wound over the same area. Although tissue damage may appear superficial, it may actually begin deep inside the tissues, close to the bone, and only later manifest

on the skin. Visually, the skin may appear intact yet discolored, but the muscle, unseen underneath the skin, may actually be damaged or even necrotic.

Conversely, with regard to healing, deep partial and full thickness stages II, III, and IV pressure ulcers do not heal by restoration of individual tissue layers (i.e., restoration of the adipose layer followed by restoration of the dermis and then the epidermis), but rather by reparation with inflammation, granulation, matrix formation, and remodeling (Brown-Etris, 1995; Cooper, 1995). Therefore, it is never correct to “reverse” stage pressure ulcers from category/stage IV to category/stage III to category/stage II to category/stage I. The tissue defect of a category/stage IV ulcer that has been replaced by collagen scar should be referred to as a healing category/stage IV ulcer if tissue integrity is not yet restored, and ultimately as a healed category/stage IV pressure ulcer when the area is fully epithelialized. It should be noted, however, that a superficial partial thickness ulcer (category/stage II) may heal by re-epithelialization with epithelial migration without scar formation and the skin may return to its normal state without evidence of tissue damage (Brown-Etris, 1995).

It is extremely important to correctly stage a wound upon first presentation as differently staged wounds have different natural histories and prognoses for healing. For example, while sDTI may not appear severe upon initial observation, one case series noted that deterioration may be rapid with 26% of sDTIs becoming full thickness wounds an average of 6 days later, despite the use of pressure redistribution support surfaces and ulcer prevention education in nearly all, and 52% of sDTIs becoming full thickness wounds (category/stage III and IV) within 1 week in another different case series (Richbourg, 2011).

Prevalence of Pressure Ulcers in Persons with SCI

Historically, more than one-third of individuals admitted to specialized SCI units develop pressure ulcers during acute care or rehabilitation (Yarkony and Heinemann, 1995; Mawson et al. 1988; Young and Burns (1981a, 1981b)) and the prevalence rates of pressure ulcers for persons with SCI residing in the community have ranges from 17%-33% (Carlson et al., 1992; Fuhrer, et al., 1993).

An increased prevalence of pressure ulcers is seen in persons with longer SCI durations, especially among those 10 years or more post-injury (Chen, et. al., 2005). Among veterans with SCI, over 1/3 report having pressure ulcers during the previous year (Smith, et al., 2008). Pressure ulcers remain the second leading cause of rehospitalization after urinary causes for persons with SCI enrolled in the US SCI Model Systems Database accounting for 17% of the rehospitalizations in the first year after injury, 23% at 5 years, 29% at 10 years, and 38% at 20 years post injury (NSCISC, 2012).

Costs of Pressure Ulcers

The costs related to the treatment of pressure ulcers include direct costs (e.g., costs of dressings, medications, nursing care, physical therapy, hospitalization, surgery), as well as the much more difficult to ascertain indirect costs, which include caregiver burden, loss of vocational productivity, and psychological stress.

Few published reports address cost of treating pressure ulcers among populations of persons with SCI. Brem et al., (2010) reported the cost of the hospitalization of patients with category/stage IV pressure ulcers at a tertiary, university hospital in the United States to be, on average, more than \$124,000, regardless of whether they were hospital-acquired or community acquired. This included the costs for

the treatment of the category/stage IV pressure ulcers and their associated complications. Dealey et al. (2012) estimated that the cost of healing a non-complicated category/stage IV pressure ulcer to be the equivalent of \$23,000 while the cost of treatment of a patient with a category/stage IV ulcer with underlying osteomyelitis to be the equivalent of \$65,000. The high costs were attributed to both the higher daily costs of treatment, as well as the longer length of stay in the hospital. Therefore, it is universally recognized that the direct costs associated with the treatment of pressure ulcers to be very high. This is not even taking into account the indirect costs to the patients, their families and society.

Two recent reports have examined the direct costs of the treatment of pressure ulcers in persons with SCI. Chan et al. (2013) studied the costs of treating chronic pressure ulcers among community-dwelling SCI individuals in Ontario, Canada. The average monthly cost was found to be \$4,745 per community dwelling SCI individual. Costs for hospital admission were found to make up for the largest percentage of the total costs. Stroupe et al. (2011) studied the costs of treating pressure ulcers among veterans with SCI. The total inpatient cost per year for SCI veterans with pressure ulcers was \$91,341 while that for a veteran without pressure ulcer was \$13,754. The total outpatient cost per year for SCI veterans with pressure ulcers was \$19,844, compared to \$11,829 for those without a pressure ulcer.

Recommendations

Risk and Risk Assessment

All individuals with SCI are at risk for the development of pressure ulcers. Among the numerous risk factors that have been identified are demographic characteristics, SCI-related factors, medical comorbidities, and psychosocial factors. Identifying an individual's risk factors is the first step toward preventing pressure ulcers. The literature is replete with studies that describe the differences of opinion among researchers and clinicians regarding the significance of various risk factors. These differences derive from the various ways studies were conducted, many of which did not control for the numerous variables that contribute to risk.

1. **Conduct an assessment of pressure ulcer risk factors in individuals with SCI at every appropriate opportunity.**
 - **Assess the following risk factors for the development of pressure ulcers.**
 - **Demographic**
 - **SCI-related, such as incontinence**
 - **Comorbid medical**
 - **Nutritional**
 - **Psychological, cognitive, contextual, and social**
 - **Support surface for bed, wheelchair, and all durable medical equipment (DME) surface such as shower/commode chair or bathroom equipment related**
 - **Use both a validated risk-assessment tool and clinical judgment to assess risk.**

(Scientific evidence—I/II/III/V; Grade of recommendation—A/B/C; Strength of panel opinion—Strong)

Demographic Risk Factors

AGE

As individuals with SCI age, particularly after age 40, the number and severity of pressure ulcers that develop increase (Vidal and Sarrias (1991)). Muscle mass, collagen, and normal elastin decreases with age, increasing the risk for developing pressure ulcers (Bergstrom et al., 1996). Rochon et al. (1993) found that SCI

individuals 60 years or older were at higher risk than younger individuals with SCI but observed that comorbidities may be more important than age in predicting this pressure ulcer development. Salzberg et al. (1998) and Mawson et al. (1988) did not find associations between age and pressure ulcer development in persons with SCI in either the community or immediately post injury in their studies. Chen (2005) found pressure ulcers were more common in persons with SCI who were older than 50 as compared to those who were younger.

GENDER

Almost 81% of SCIs occur among males (NSCI SC 2/2013, published in 2014).

Although Vidal and Sarrias (1991) found there was a 3:1 ratio of males to females in developing pressure ulcers, Salzberg et al. (1996) and Salzberg et al. (1998) found no gender differences in the SCI population with regard to pressure ulcer development.

ETHNICITY

Bergstrom et al. (1996) and Jiricka et al. (1995) reported that caucasians may be at greater risk for developing pressure ulcers than nonwhites; however, Allman et al. (1995), Ek et al. (1991), and Mawson et al. (1988) reported that race was not a significant factor in pressure ulcer development. Chen (2005) in a large Model Systems cohort reported a greater risk for African Americans. In a multisite randomized controlled trial of veterans with SCI, the strongest predictor of pressure ulcer recurrence was African American race (multivariate logistic regression, odds ratio=9.3) Guihan, Garber et al. 2008.

MARITAL STATUS

Several investigators have studied the effect of marital status on the occurrence of pressure ulcers. Young and Burns (1981a and 1981b) found that, at follow-up, married people were less likely to have a pressure ulcer. Conversely, Vidal and Sarrias (1991) reported that married people were more likely than single people to have a pressure ulcer. Carlson et al. (1992) and Fuhrer et al. (1993) found no relation between marital status and having an ulcer, while Chen et al. (2005) found a higher prevalence in singles.

EDUCATION

Davidoff et al. (1990) found that less formal education predicted more hospital readmissions among a group of 88 people with a recent SCI

after their acute rehabilitation. Results from Lloyd et al. (1993) indicate that a lower level of education is linked to less understanding of and follow-through on complex recommendations for managing chronic conditions. Supporting this hypothesis, Chen et al. (2005) reported a higher prevalence of ulcers in those with less than a high school education.

ENVIRONMENT/FACILITY IMMEDIATELY POST SCI

In a study of 100 persons admitted to a specialty SCI unit in England, investigators reported that. Individuals admitted within one week of injury were significantly less likely to develop pressure ulcers than those admitted after one week (Aung and el Masry, 1997). Persons admitted to specialty SCI units of US SCI Model Systems within 72 hours of injury have been shown to have a lower prevalence of ulcers with lesser severities than those admitted later (Richardson and Meyer (1981); Yarkony and Heinemann, 1995). This has been attributed to the familiarity of the clinicians in the SCI units with the potential secondary complications of SCI including pressure ulcers and the implementation of preventive measures.

SCI Related Risk Factors

LEVEL AND COMPLETENESS OF INJURY

In one analysis of the data collected on pressure ulcers within the US Model Systems, persons with complete injuries seemed to be at greater risk than those with incomplete injuries and those with cervical injuries seemed to be at greater risk than those with lower level injuries (Richardson and Meyer (1981)). In another analysis, level of injury was not found to be a significant risk factor (Chen 2005). In a study of VA patients with SCI, Salzberg et al. (1996) found that complete SCI was significant, but level of injury was not. Mawson et al. (1988) and Curry and Casady (1992) found no statistical significance related to the development of pressure ulcers based upon completeness or level of injury immediately post injury. However, recently, Verschueren et al. (2011) found tetraplegia and motor completeness of injury to be significant risk factors for developing pressure ulcers.

DURATION OF INJURY

Whiteneck et al. (1985) reported that individuals with high tetraplegia and longer duration of SCI were more likely to have an ulcer. Hirschwald et al. (1990) and Furher et al. (1993) also found that longer duration of SCI was associated with greater likelihood of having an

ulcer. Smith et al. (2008) showed that among veterans completing a health survey, having an injury more than 30 years was associated with the presence of pressure ulcers. Chen and colleagues (2005) conducted a multicenter cohort study of more than 3000 individuals from 9 US Model systems for SCI followed annually to examine age-time period-duration patterns of pressure ulcer prevalence among persons with spinal cord injury residing in the community. They found a significant trend toward increasing pressure ulcer prevalence in recent years (1994–2002) compared to the years 1984–1993 which could not be explained by aging, years since injury, or demographic and clinical factors. Pressure ulcer risk appeared to be steady during the first 10 years post-injury and increased 15 years post-injury.

USE OF MEDICAL DEVICES

Black et al. (2010) in a study of pressure ulcers in a medical center found that approximately 1/3 were medical-device related. Apold and Rydrych (2012) analyzed pressure ulcer data from 34 hospitals and found the following distribution for pressure ulcers associated with medical devices: cervical orthoses (22%), other types of immobilizers (17%), oxygen tubing (13%), stockings or positioned boots (12%), and nasogastric tubes (8%). The authors attribute these device-related pressure ulcers to the non-removal of these devices due to the lack of awareness of the need to periodically remove or reposition them to maintain skin integrity and the lack of guidance on when and how to remove them. Factors associated with cervical orthosis related pressure ulcers are the time period to cervical orthosis removal and the orthosis type (Ackland et al., 2007).

ACTIVITY AND MOBILITY

Activity, as distinguished from mobility, refers to involvement of the person in recreation, such as athletics, social pursuits as well as vocational endeavors. In an investigation of 219 individuals with SCI, Salzberg et al. (1996) related that a decreased level of activity was the most significant risk factor in developing pressure ulcers. In a subsequent study involving analysis of 800 individuals with SCI, Salzberg et al. (1998) similarly reported that a restricted level of activity was the most significant risk factor for pressure ulcer development. In 2004, Krause et al. reported the results of a survey of over 800 non-ambulatory subjects followed in a large subspecialty hospital. Factors found to be protective against developing ulcers included being fit and participating in planned exercise.

In people of comparable age, SCI severity, and pre-injury health, athletic involvement was shown to be protective of pressure ulcer development (Stotts, 1986).

HISTORY OF PRIOR PRESSURE ULCERS

Individuals with SCI who have had a history of pressure ulcers, especially if treatment included surgery, are at high risk for recurrence. Descriptive studies of different populations, including veterans have reported recurrence rates which range from 35% to 63% (Bates-Jensen et al., 2009; Lehman (1995); Niazi et al. (1997).

Vidal and Sarrias (1991) found that recurrence of pressure ulcers was a highly significant risk factor for increased severity of the ulcer. Recurrence has also been associated with younger age, black race, unemployment, nursing home residence, previous pressure ulcer surgery, smoking, diabetes, and cardiovascular disease (Niazi et al., 1997; Disa et al., 1992; Guihan et al., 2008). Lack of social supports, inadequate pressure ulcer prevention knowledge, ineffective or nonparticipation in preventive practices, and poor psychological well-being also have been associated with recurrence (Disa et al., 1992; Jones et al., 2003; Heilporn, 1991).

Recurrence following pressure ulcer surgery has been associated with poor patient compliance, the lack of control of comorbidities, and incomplete presurgical debridement (Sorensen et al., 2004). For those who have undergone previous pressure surgeries, the median recurrence time is 4 months post pressure ulcer surgery (Bates-Jensen et al., 2009).

Hospital-based educational programs are insufficient to prepare an individual with a SCI to integrate preventive behaviors into his/her lifestyle post discharge. In a randomized controlled trial of 49 male veterans with SCI, investigators determined that individualized education and structured monthly contacts have been shown to delay or reduce the frequency of pressure ulcer recurrence after surgical repair (Rintala et al., 2008).

BLADDER, BOWEL, AND MOISTURE CONTROL

Appropriate bladder and bowel management programs prevent the skin from becoming contaminated with urine and feces. Salzberg et al. (1996) and Salzberg et al. (1998) reported that bacteria found in stool is destructive to the skin and that urinary and fecal incontinence were significant factors in pressure ulcer development in the SCI population. Control of moisture is extremely important in preventing incontinence associated dermatitis, which is not to be confused with a pressure ulcer.

Comorbid Medical Risk Factors

MEDICAL COMORBIDITIES

In a retrospective chart review of 81 SCI individuals with pressure ulcers, Rochon et al. (1993) reported that having more than seven ICD-9-CM codes on the discharge summary was significantly associated with pressure ulcer development. Guihan and Garber in 2008 similarly reported that persons with higher burden of illness as measured with the Charlson comorbidity index were at increased risk for developing pressure ulcers. In a retrospective chart review by Vidal and Sarrias (1991) of 268 individuals with SCI, a high incidence of urinary tract infections was associated with pressure ulcers. Salzberg et al. (1996) related that the number of comorbidities—cardiac disease or abnormal EKG, diabetes, renal disease, pulmonary disease, and sepsis/infection—was a risk factor in an SCI population. In a later study by Salzberg et al. (1998), data obtained from 800 individuals with SCI in the community revealed that renal and pulmonary diseases were significant risk factors, but cardiac disease, diabetes, and impaired cognitive function were not significant risk factors. Mawson et al. (1988) found that diabetes mellitus and peripheral vascular disease were insignificant factors for pressure ulcer development in the immediate SCI post-injury period. In contrast, both Chen et al. (2005) and Smith et al. (2008) report in large studies of individuals followed in Model System and VA systems respectively that having diabetes is a significant risk factor for developing pressure ulcers. Verschueren et al. (2011) found that pneumonia and/or pulmonary disease during acute rehabilitation is a risk factor for pressure ulcer development.

In some individuals with SCI, friction and shear may be of concern due to increased spasticity, particularly with higher level injuries, and the contact of the skin and tissues with the support surface. In a retrospective study of 268 individuals with SCI, Vidal and Sarrias (1991) reported that decreased spasticity was also a significant risk factor in pressure ulcer development.

Mawson et al. (1988) reported that individuals with SCI who developed pressure ulcers in the immediate post-injury period had significantly lower systolic blood pressure (≈ 100 mm Hg) compared to controls (≈ 120 mm Hg). In two studies, autonomic dysreflexia was associated with pressure ulcer development among individuals with SCI (Salzberg et al., 1996; Salzberg et al., 1998).

Nutrition

Specific biochemical indices of nutrition that are associated with the risk of pressure ulcers include total protein, albumin, hemoglobin, hematocrit, and total lymphocyte count. See the nutrition section of the “Prevention Strategies” chapter for more details.

Psychological, Cognitive, Contextual, and Social Factors: Substance Abuse and Adherence/Compliance

PSYCHOLOGICAL FACTORS

Major depression and anxiety disorders, as well as negative self-concept and poorly managed anger and frustration can interfere with cooperation between the individual and his or her care providers and can be associated with inactivity, self-neglect, and poor medical adherence (Cox and Gonder-Frederick, 1992; Vidal and Sarrias, 1991; Woolsey, 1985). Krause and Kjorsvig (1992) found lower survival rates, including death from sepsis following pressure ulcers, among people with SCI who had reported lower life satisfaction and adjustment and greater psychological distress 4 years earlier.

COGNITIVE IMPAIRMENT

Cognitive impairments among people with SCI may result from brain injury accompanying the SCI or from medication use, substance abuse, preexisting cognitive dysfunction or learning disability, or delirium secondary to infection. In a study of more than 200 individuals with SCI in a VA setting, Salzberg et al. (1996) identified impaired cognitive function as a significant risk factor for pressure ulcers, while Richards et al. (1991) reported an increased risk for pressure ulcers among people with SCI who also had sustained a traumatic brain injury.

SOCIAL ENVIRONMENTAL CONTEXTUAL AND CIRCUMSTANTIAL FACTORS

In recent years, there have been attempts to identify risk factors, other than obvious medical and SCI-related ones, within a person's life that contribute to the development of pressure ulcers, interfere with healing, and fail to prevent recurrence. For example, in a disadvantaged population cohort for whom life context may include incarceration, homelessness, and gang membership, it has been shown that such factors such as perpetually being in a state of danger; experiencing frequent disruptions or changes of daily routine; choosing to participate in high risk activities; and lacking access to care, services, and supports can indeed influence

the development of pressure ulcers (Clark, et al., 2001; Clark, Rubayi et al., 2006, Jackson et al., 2010). In persons with chronic SCI and pressure ulcers, it is important to assess the individual's motivation to stay ulcer-free (Clark et al 2006; Jackson et al, 2010). Motivation drives the daily actions or inactions of the individual. Lack of motivation to perform preventative activities may be associated to depression, lack of social support or poverty. Conflicting motivations, such as wanting to stay fully engaged in either vocational or recreational pursuits, may interfere with a maintaining a strict regimen of pressure ulcer prevention. Understanding life context, daily routines and central daily activities should result in a balance between ones daily occupations and pressure ulcer preventative routines. Therefore, in order to most effectively prevent and treat pressure ulcers in any individual, it is important to not just evaluate the usual risk factors as described throughout in this guideline, but also to evaluate the context in which the individual with SCI lives, for, if these other factors are not addressed as well, any intervention is not likely to be successful.

SUBSTANCE ABUSE

Substance abuse is a risk factor for pressure ulcer development after SCI (Vidal and Sarrias, 1991). Substance abuse can lead to impairments in cognition and judgment (Cleaveland and Denier, 1998) and is associated with less adherence to health regimens (Pablos-Mendez et al., 1997; Umpierrez, et al., 1997). Substance abuse may be a direct or indirect risk factor (e.g., excessive alcohol intake can increase the risk of poor nutritional intake or it can cause bladder distension and potential urinary incontinence). Hawkins and Heinemann (1998) found increased risk in illicit substance abusers in a sample of 126 individuals with SCI; while data collected by Krause et al. (2001, 2004) suggest that hospitalization for pressure ulcer treatment is associated with reported alcohol or drug treatment.

Based on the known physiological effects of smoking, it is reasonable to assume that smoking is a risk associated with the development of pressure ulcers and would interfere with healing. Salzberg et al. (1996) found that individuals with SCI who had pressure ulcers were twice as likely to be current smokers. Krause, in turn, (2001, 2004) reported that pressure ulcers are more likely to occur in persons who use pain medications and in those who smoke (past or present).

ADHERENCE/COMPLIANCE

Adherence/compliance relates to the success of an individual in following through consistently with health recommendations for preventing and treating pressure ulcers. A major factor that has been associated with suboptimal adherence is a misconception about risk. Rodriguez and Garber (1994) found that more than 80% of a sample of people with SCI who had experienced a previous ulcer did not believe they were at risk of future ulcers. As a verbal or written commitment from a person to follow through with a health behavior recommendation has been associated with better adherence (Cox and Gonder-Frederick, 1992; Meichenbaum and Turk, 1987), such commitment should be sought routinely. The person's ability to verbalize his or her health behavior regimen seems to be a minimum indicator of adherence (i.e., understanding is necessary but not sufficient to produce the recommended behavior) (Rodriguez and Garber, 1994). Potential points of disagreement between the health-care provider and the individual with regard to recommendations offered should be assessed directly by the provider because, at a minimum, this gives the provider a chance to learn where and how to provide more information about and a rationale for a given recommendation.

The regimens involved in managing SCI are complex and require lifestyle changes. Many of the recommendations for prevention, such as performing pressure redistribution, require understanding, cooperation, and initiative. Some of the factors associated with pressure ulcers may involve the behavior of the individual who has the injury as well as the behavior of those in formal and informal support networks if assistance is needed in order to perform certain activities or tasks. Management of other factors, which can complicate the prevention and treatment of pressure ulcers (e.g., comorbidities such as diabetes, or complications of SCI such as incontinence), may also involve demanding and complex procedures. Evidence from the behavioral medicine literature indicates that complex regimens and/or those involving lifestyle changes are associated with poor adherence (Ary et al., 1986; Glasgow et al., 1992; Hulka et al., 1976). Studies by Clark, (2001) and, Jackson, (2010) describe the intricate balance between buffers (protective behaviors and contexts) that reduce the risk of recurring ulcers and liabilities (negative behaviors and circumstances) that increase risk of developing recurring ulcers. These studies shed light on the complex and highly individualized scenario of a person's ulcer history and future interventions. These studies

suggest that to help individuals decrease the risk of pressure ulcer recurrence, the health-care provider must engage in a meaningful dialogue that takes into account individual detailed of habits and routines. The individual with SCI must be empowered to embed preventive behaviors that are doable within their context. For example, the health-care provider may help the person with tetraplegia come up with timed alerts to remind them to recline while being engaged in distracting activities.

Support Surfaces for Bed and Wheelchair

The factors related to support surface are similar for both prevention and management. Please refer to the chapter "Pressure Redistribution and Support Surfaces" for details.

Risk-Assessment Tools

Almost all individuals with SCI are at lifelong risk for developing pressure ulcers. Risk-assessment scales distinguish those who are at risk for developing a pressure ulcer and determine the extent to which a person exhibits a specific risk factor. Early risk assessment prompts the immediate, targeted implementation of preventive and risk-reduction interventions.

There is evidence that risk-assessment scales may be used successfully to predict pressure ulcers in various populations and result in favorable outcomes (Allman et al., 1995). Results of risk-assessment measures and their ability to predict pressure ulcers vary according to the measure (Arnold, 1994; Hunt, 1993), to the patient population (Bergstrom et al., 1996; McCormack, 1996), and to the person who assesses the individual (Edwards, 1994). Some risk variables for which there is research evidence or strong clinical support are not well represented among existing risk-assessment tools. Specifically, these variables include psychosocial factors, such as substance abuse, adherence to recommended behaviors, depression, degree of cognitive impairment, and degree of social support. Additionally, since health status and risk for pressure ulcers can change rapidly, clinical judgment is required to guide decisions when further assessment should be performed. Formal assessment tools have many limitations and therefore patient care prevention strategies based upon the health-care professional's judgment in conjunction with tool use are justified (VandenBosch et al., 1996; Watkinson, 1997).

Several instruments designed to predict the risk of pressure ulcers have been reported in the literature. These instruments use summative rating scales based on contributing and confounding factors and specific critical scores for identifying those at risk. Currently, there is no consensus or specific recommendation as to which risk-assessment scale should be used for persons with SCI.

The Braden scale (Bergstrom et al., 1995; Bergstrom et al., 1996; Bergstrom, 1997) is the risk-assessment tool that has been tested widely in populations other than people with SCI. The scale evaluates risk for pressure ulcer development based on six domains or subscales:

- (1) sensory perception,
- (2) moisture,
- (3) activity,
- (4) mobility,
- (5) nutrition, and
- (6) friction and shear.

Each subscale is rated from the highest risk (score 1) to the lowest risk (score 4). The total score can range from 6 to 23, and individuals are classified as follows: very high risk (score of 9 and below), high risk (score of 10 to 12), moderate risk (score of 13 to 14), low risk (score from 15 to 18), and no risk (score of 19 to 23). It was recommended that for the SCI population, a score below 10 on the Braden scale would be the cutoff score, indicating the score at which the individual is at risk for a pressure ulcer (Salzberg et al., 1999). Factors in establishing a cutoff point for the at-risk status of individuals with SCI have not been well established.

The Spinal Cord Injury Pressure Ulcer Scale (SCIPUS) developed by Salzberg et al. (1996), a measure of risk for pressure ulcer development during the acute rehabilitation phase, assesses seven domains:

- (1) level of activity;
- (2) level of mobility;
- (3) severity of SCI;
- (4) urinary incontinence or constant moisture;
- (5) other factors, such as age, tobacco use/smoking, pulmonary disease, cardiac disease abnormal blood glucose control, renal disease, and impaired cognitive function;
- (6) residence in a nursing home or hospital; and
- (7) nutrition (to uncover malnutrition and anemia).

As a follow-up to this, to address the risk of pressure ulcer development in the acute hospital, the SCIPUS Acute (SCIPUS-A) was designed to measure the risk of pressure ulcer development during acute hospitalization. It measures six domains:

- (1) extent of paralysis;
- (2) incontinence;
- (3) nutrition (measuring serum creatinine and albumin);
- (4) pre-existing conditions, such as pulmonary disease;
- (5) mobility; and
- (6) level of activity (Salzberg et al., 1999).

The Salzberg scales have had limited validity testing.

The Norton scale (Berglund and Nordström, 1995; Norton, 1989) uses five variables to assess risk: activity, mobility, incontinence, physical condition, and mental condition. Other scales have been successfully used on a limited basis with the SCI population in various health-care settings. Arnold (1994) modified the Gosnell scale (Gosnell, 1989), which identified activity, mobility, incontinence, nutrition, and mental status as risk factors, while the Waterlow Pressure Sore Risk Calculator (Clifford et al., 1995; Edwards, 1995) considers build/weight for height, continence, mobility, and appetite.

One trigger for pressure ulcer risk reassessment should be based on the deterioration or improvement in the individual's health status. As the number of comorbidities increases, individuals may be at greater risk for an ulcer (Rochon et al., 1993; Salzberg et al., 1996; Tourtual et al., 1997; Smith et al. 2008; Guihan, Garber et al., 2008). Deterioration of the skin may occur rapidly in acute situations, such as when an individual with a suspected SCI is placed on a spinal board, or may be gradual in nature, such as when an individual becomes malnourished over time (Maklebust and Magnan, 1994; Vidal and Sarrias, 1991).

Although it is widely acknowledged that regular assessment should be incorporated into the overall comprehensive assessment of individuals with SCI, there is no consensus as to what these regular assessment intervals should be. In many civilian acute care settings, risk assessment is done upon admission and every 48 hours, or whenever the patient's condition changes. In contrast, in many civilian rehabilitation settings, risk assessment is done on admission, once weekly thereafter, and when the

patient's condition changes, while in U.S. Department of Veterans Affairs Medical Center SCI units, risk assessment (using the Braden Scale) is performed daily during the first week of admission and then weekly thereafter until discharge, transfer, or change in medical status (VHA Handbook 1180.02).

Prevention Strategies Across the Continuum of Care

Pressure Redistribution

2. **Implement pressure ulcer prevention strategies as part of the comprehensive management of acute and chronic SCI and review all aspects of risk when determining prevention strategies.**

- **Initiate pressure redistribution as soon as emergency medical conditions and spinal stabilization status allow.**

(Scientific evidence— I, II, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Pressure ulcer prevention begins during the acute phase of SCI management and this includes pressure relief strategies in the emergency department (ED) (if patient is on a spinal board) and the operating room. In the operating room, factors related to positioning, the immobility during the intraoperative and immediate postoperative period (Cherry Moss, 2011; St-Amaud & Paquin, 2009), anesthesia duration and the total time of the diastolic pressure less than 50 mm Hg (Connor et al., 2010), the duration of surgery, and patient-related factors all have been shown to affect pressure ulcer development (Walton-Geer, 2009). All persons undergoing surgery should be considered at-risk for pressure ulcer development and perioperative use of dynamic pressure-relieving devices is recommended. Nevertheless, it should be noted that Hoshowsky and Schramm (1994) reported an incidence of 16.8% of category/stage I ulcers in 505 individuals who had been positioned with pressure reduction devices and standard devices intraoperatively.

A rigid backboard should be used for as short a period of time as possible for initial inpatient evaluation and stabilization (Vickery, 2001). Prompt removal from the backboard, after transport to an ED and initial spine stabilization, is required to reduce pressure ulcer formation. For patients with a confirmed SCI, transfer the

patient off the backboard onto a firm padded surface, ideally within 2 hours, continuing precautions to protect the spinal column and skin. Those who have extended transport to the ED or who are delayed in transfer to the intensive care unit are at increased risk of skin breakdown. (Early Acute Management in Adults with Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals, 2008)

The duration of unrelieved pressure prior to a nursing unit admission and the length of time on the spinal board have been shown to be significant risk variables for pressure ulcer development within the first 8 days post spinal cord injury (Mawson et al., 1988). In one study, individuals who developed ulcers during the first 8 days after injury spent an average of 20 hours unturned compared to 11 hours unturned in the control groups. In a study of 49 individuals with SCI immediately post injury, Curry and Casady (1992) found that individuals immobilized longer than 6 hours developed pressure ulcers at a significantly greater rate than individuals immobilized for shorter periods of time.

A study of 32 spinal individuals with SCI with and without pressure ulcers determined that those individuals with pressure ulcers were more likely to have had a prolonged immobilization in the immediate post-injury period (Linares et al., 1987).

Visual and Tactile Skin Inspections

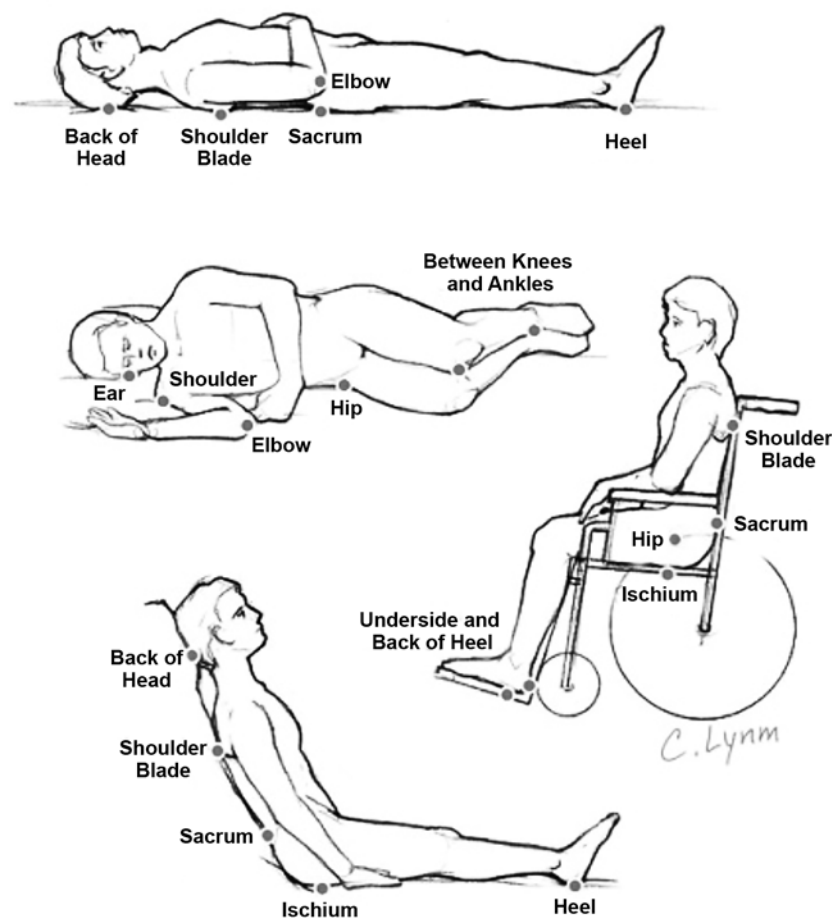
3. **Conduct daily comprehensive visual and tactile skin inspections with particular attention to the areas most vulnerable to pressure ulcer development, including, but not limited to the following:**

- **Ischial tuberosities**
- **Sacrum**
- **Coccyx**
- **Greater trochanters**
- **Ankles (malleoli)**
- **Knees (medial aspect especially during side-lying position)**
- **Occiput**
- **Calcaneous**

(Scientific evidence—III, V; Grade of recommendation—C; Strength of panel opinion—Strong)

Frequent inspection is essential to detect early skin breakdown (e.g., nonblanchable erythema). Individuals with lower level injuries (paraplegia) may perform self-inspection with a

Figure 1: Common Locations of Pressure Ulcers



long-handled mirror or camera, whereas individuals with higher levels of paraplegia and tetraplegia who are unable to perform self-inspection must rely on a caregiver or professional for thorough inspection.

Vidal and Sarrias (1991) reported that the areas of the body with the highest prevalence of pressure ulcers in the SCI population were the ischium (28%), sacrum (21%), and trochanter (20%). In the immediate post-SCI period, Mawson et al. (1988) related that the most common area were the sacrum (57%) and heel (22%). Richardson and Meyer (1981) related that among 549 individuals with SCI, the most frequent single site of pressure ulcers was the sacral region.

In a community sample of persons with SCI, Garber et al. (1996) reported that 90% of persons with paraplegia detected the ulcer by self-inspection or "feeling it," while 92% of persons

with tetraplegia reported that someone else detected the ulcer first.

The skin should be visually inspected daily with specific attention to bony prominences (Bergstrom et al., 1992) to assess for any changes in skin color (red areas, discolorations, bruises) and in texture (dryness, raised areas, cracks, scabs, blisters, rashes, shiny areas). The skin should be touched to assess for warmth, wetness, hardness, or softness (Pires and Muller, 1991). Bony prominences of the body to be inspected are the ischial tuberosities, sacrum, coccyx, greater trochanters (hips), heels, malleoli (ankles), knees, scapulae, and elbows (Scotzin and Sommer, 1993). (See Figure 1.) The recommendation is for the individual with SCI to be responsible for carrying out this task. If this is physically or cognitively impossible, it is best if one or two persons, such as a family member or care provider, consistently assist with the task

because it is necessary to have a basis of comparison in order to detect a change in status. Regardless of the physical ability of a person with SCI, he or she can still be in control of directing others to assist in this task unless the person with the SCI is cognitively or intellectually impaired.

Although pressure ulcers occur most commonly over a bony prominence such as the sacrum, ischial tuberosities, trochanter, and calcaneus, areas where a person's weight is concentrated while sitting or lying, they can develop anywhere on the body, especially in places that are related to the use of medical devices, such as casts, splints, cervical orthoses, drainage tubing, and other immobilizers. Identifying a category/stage I pressure ulcer in individuals with darker skin tone is challenging because redness and color changes are not easily detectable. Other pressure-related skin changes should therefore be assessed. These include changes in skin consistency, sensation, temperature, and moisture. For example, skin can be firm or boggy, there may be pain and itching, and the skin may feel warm or moist to touch.

Bates Jensen et al. (2009), in a cohort study of nursing home residents, performed visual skin assessment and measured subepidermal moisture (SEM) using a surface capacitance dermal phase meter at the right and left buttocks and sacrum weekly and found that SEM was associated with future pressure ulcer occurrence in persons with dark skin tones. Guihan et al. (2008) studied the feasibility of detecting early pressure ulcer damage by measuring SEM on the sacrum, right and left heels, trochanter, ischium, and buttocks using a handheld dermal phase meter on persons with SCI and found that SEM was higher in persons who had category/stage I pressure ulcers, suggesting a possible relationship between SEM and skin damage.

Turning and Repositioning

4. **Turn or reposition individuals with SCI initially every 2 hours in the acute and early rehabilitation phases, adjusting by medical conditions and/or risk factors.**
 - **Avoid overstretching and folding of skin/soft tissues while positioning and shearing when individuals are repositioned or transferred.**
 - **Avoid positioning individuals who are side-lying in bed directly on their trochanters.**

(Scientific evidence—III, V; Grade of recommendation—C; Strength of panel opinion—Strong)

Current practice is to reposition at-risk persons at least every 2 hours to prevent capillary and venous occlusion. Kosiak (1961) recommended a frequency of repositioning of 1 to 2 hours based upon findings found in uninjured individuals. Norton et al. (1975) conducted an observational study of older individuals without SCI and demonstrated that individuals turned every 2 to 3 hours had fewer ulcers than those who turned less frequently. Individuals without SCI have very different response to constant applied load as compared to most with SCI. (Bader, 1990)

When in bed, the lowest degree of head of bed elevation should be maintained consistent with the medical condition and other restrictions that may limit the amount of time that the head of the bed is elevated. Raising the head of bed to 30 degrees or higher increases the peak interface pressure between the skin at the sacral area and the support surface. As the a head of a bed is elevated more than 45 degrees the affected area's skin-bed interface pressure that is greater or equal to 32 mm Hg increases as well (Peterson et al., 2008).

Significantly increased skin-bed interface pressures have also been shown to apply to positioning in a lateral position with and without elevation. Peterson et al. (2010) found that raising the head of the bed to 30 degrees in the lateral position significantly increased the interface pressures at the sacrum, trochanter, and buttock regions, and increased when using wedges as compared to pillows to support lateral positioning. Garber et al. (1982) reported the effect of side-lying on trochanteric interface pressures and Seiler et al. (1986) measured the effect of side-lying positions on transcutaneous oxygen tension finding that direct positioning on the trochanter (90 degree angle) produces high interface pressures and low transcutaneous oxygen tension, but when individuals are positioned at a 30-degree side-lying angle, the body's transcutaneous oxygen tensions are normal, with significantly reduced interface pressures.

The technique of turning an individual is as important as the frequency of turning. A person should always be lifted as opposed to dragged across a surface in order to prevent shear related injuries. Use of sheets can eliminate or reduce shear, thus reducing the risk of skin damage. Avoiding shear is also important in minimizing skin breakdown during transfers from one surface to another. Using lifting devices to assist in moving individuals who cannot assist during

transfers can reduce friction or shearing forces exerted on the skin. Friction may be minimized by the use of lubricants, protective films, protective dressings, and protective padding. Massage over bony prominences should be avoided due to its possible harmful effects (Bergstrom et al., 1992). Bumping or scraping the body during transfers, poor sitting posture, frequent shearing against bed surfaces during dressing or bed mobility, or ineffective pressure redistribution techniques can be contributing factors to this problem.

Proper pillow placement behind the back and between the legs will help to relieve the pressure of bony areas touching one another or the surface of the bed (Land, 1995; Lowthian, 1993). Clothes should not fit tightly anywhere they could restrict circulation or cause friction or shear to the skin. Clothing materials that have a rough texture or include abrasive features, such as hard fasteners or studs on rear pockets or double-welted rear seams, like those on blue jeans, can contribute to skin abrasions. The types of fabrics that are best for the skin do not hold heat in to the body. Lightweight cotton fabrics are better than nylon or wool. Also to be avoided are tight-fitting shoes, socks, stockings, braces, splints, and leg bag straps (Pires and Muller, 1991) as these items may restrict normal blood flow in the body or cause undue friction or shear (Krouskop et al., 1983; Scotzin and Sommer, 1993).

During rehabilitation hospitalization, persons with SCI are turned according to a specific 24 hour protocol. However, over time, the over-night turning schedule may be modified to reflect a person's ability to withstand pressure on vulnerable areas of the body for longer periods of time. This is extremely important once the person is transitioned home so that there are fewer sleep disturbances for both the person with SCI and his/her family or caregiver. This is determined by the person's medical and skin condition. As tissue tolerance increases, persons with SCI should not have to be turned so often especially during the night.

Effective Support Environment

5. Evaluate the individual and his or her support surfaces for optimal maintenance of skin integrity.

- **Prevent moisture accumulation and temperature elevation at the skin-support surface interface.**
- **Utilize pressure redistribution support surfaces preventively to protect soft tissues from bruise and injury.**

- **When off-loading the calcaneus with pillow or cushion, ensure the proper position of the pillow or cushion. It should be placed lengthwise under the calcaneus off the bed surface.**
- **Do not use donut-type devices.**
- **Monitor the performance, i.e., continued effectiveness, of support surfaces for the bed and wheelchair specific to pressure ulcer prevention.**

(Scientific evidence–V; Grade of recommendation–C; Strength of panel opinion–Strong)

Cleansing the skin on a regular basis and at times of incontinence is recommended (Bergstrom et al., 1992; Rodeheaver, 1999). Mild cleansing agents that minimize irritation and dryness of the skin are recommended; while hot water should be avoided. When cleansing the skin, clinicians and caregivers should take care not to exert undue force and friction to the tissues. When sources of moisture—whether from incontinence, perspiration, or wound drainage—cannot be controlled, underpads or briefs made of materials that absorb moisture and present a quick-drying surface should be used (Bergstrom et al., 1992). Wet skin tends to adhere to bed linens, possibly causing shearing when the linen is pulled away from the skin (Krouskop et al., 1983). If someone lives in a humid climate or if excessive perspiration is a problem, cotton fiber clothing and/or a change of clothing during the day may need to be considered in order for the skin to remain dry (Krouskop et al., 1983; Nixon, 1985; Scotzin and Sommers, 1993).

At constant tissue pressures, temperature reduction can diminish tissue damage (Romanus, 1976); conversely, temperature elevation will increase tissue injury from continuous or repetitive stress (Finestone et al., 1991; Vistnes, 1980).

Those at risk for pressure ulcer development should be placed on a pressure-redistribution bed support surface. (See recommendation 22 for detail on types of support surfaces). The use of pressure-redistributing devices prophylactically is effective in reducing the risk of pressure ulcers (Zernike, 1994). Heels should be offloaded from the bed surface.

Pillows or foam wedges should be used to keep bony prominences from contacting one another. Skin should be inspected between turns to ensure tolerance (Bergstrom et al., 1992). All body positions (supine, side-lying, prone) should be used, as tolerated, for bed positioning

(Yarkony, 1994). Initially following injury, prone positioning may be contraindicated, secondary to orthopedic or medical restrictions, yet should be considered when spinal and respiratory stability is established.

A change in body weight may make previously prescribed wheelchairs and cushions contributing factors to excessive pressure if they become too small or large for the person's body. Increases or decreases in weight could lead to excessive pressure being exerted on cushion surfaces, especially those that are filled with air, fluid, and foam.

Individualized Pressure Redistribution System

6. Provide an individually prescribed seating system designed to redistribute pressure.

- **Employ a power weight-shift system when manual pressure redistribution is not possible.**

(Scientific evidence—I, III, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Sitting interface pressures are significantly greater than supine support interface pressures due to smaller contact areas. Higher intermittent pressures may be tolerated more than uninterrupted continuous lower pressures. Wheelchair cushion performance should be evaluated in relation to the pressure-time effect on tissue viability (Rithalia, 1997). Collectively, these and other biochemical and biomechanical observations (Claus-Walker et al., 1977; Rodriguez and Claus-Walker, 1988) suggest that tissue response to external load is controlled by many factors, influencing microcirculation and interstitial fluid flow (Bader, 1990; Reddy et al., 1981). The deep tissue expression of surface stresses is mediated passively by tissue stiffness, connective tissue structure, and the collagen matrix (Bogie et al., 1995; Reddy, 1990). In addition to passive effects, muscular activity (Schubert et al., 1995) will influence interstitial fluid pressure, blood and lymphatic capillary flow and the accumulation of metabolic end products, hypoxia, cell rupture, and necrosis (Reddy, 1990). Therefore, pressure-reducing strategies are best when they follow an individualized approach based on individual and caregiver characteristics with the objectives of prevention, early detection, ease of maintenance, and affordability (Rensburg and Bennett, 1997).

Thorfinn et al. (2009) compared subcutaneous tissue oxygen and glucose levels

in individuals without SCI sitting on a wheelchair cushion and a hard surface. Both tissue oxygenation and glucose levels were significantly lower while sitting on a wheelchair cushion as compared to not sitting, but were profoundly reduced while sitting on the hard surface consistent with the theory that subcutaneous adipose tissue covering the ischial tuberosities becomes ischemic during sitting.

In a study of persons with and without SCI that measured the characteristics of seat loading in manual wheelchair users, it was found that individuals with SCI have a higher pressure distribution over a smaller area, a much smaller contact area, and a load distribution that is asymmetrical in comparison to persons without SCI, putting individuals with SCI at higher risk for pressure ulcer development (Gutierrez et al., 2004). Karatas et al. (2008), in studying the relationship of dynamic sitting stability of persons with and without SCI and its relationship to pressure ulcer development, found that the center-of-pressure displacements in unsupported forward, backward, and right- and left-sided leaning were smaller in persons with SCI than in those without SCI. This can be attributed to loss of function of the trunk, abdominal, hip, and lower extremity muscles in individuals with cervical and thoracic injuries. Based upon these findings it can be hypothesized that improving one's ability to shift in all planes could potentially help in preventing pressure ulcers.

Reenalda et al. (2009) analyzed the sitting position interface pressure distribution and subcutaneous tissue oxygenation of persons without SCI and found that subjects shifted posture an average of 8 times per hour in the sagittal plane (80%) and frontal plane (20%). These posture shifts caused an increase of 2.2% in the subcutaneous tissue oxygen saturation, suggesting potential increased tissue viability.

Depending on a person's cognitive and physical status, a variety of pressure relief and redistribution techniques can be performed including push-up lifts (commonly discouraged in favor of other techniques due to stress on the shoulders and wrists and risk of long term musculoskeletal complications), side leans, and forward leans. Use of a mechanical reclining or tilting in space wheelchair feature can also facilitate pressure redistribution. Tilt, recline, and standing systems should be considered as a means of achieving adequate pressure redistribution for all wheelchair users (Sprigle et al., 2010). The full range of tilt of a power wheelchair should be utilized to maximize the

potential for significant blood flow increases and pressure relief at the ischial tuberosities (Sonenblum and Sprigle, 2011). When this movement is provided by a powered mechanism, the individual acquires the ability to perform pressure redistribution independently (Henderson et al., 1994).

Traditionally, a weight shift every 15 to 30 minutes has been recommended to allow the skin and underlying tissues to be replenished with oxygen (Bergstrom et al., 1992; Nixon, 1985; Ho and Bogie, 2007). Analyzing subcutaneous tissue oxygen measurements of newly injured and individuals with chronic SCI (n=46), Coggrave and Rose (2003) found that the mean duration of pressure relief required to raise tissue oxygen to unloaded levels was 1 minute and 51 seconds. This duration of pressure relief was more successfully accomplished by the subjects leaning forward, side to side or having the wheelchair tilted back at greater than 65 degrees compared to performing a push-up lift (Coggrave and Rose, 2003). This duration of time is much longer than the previously recommended 15–30 seconds. Individualized attention must be given to determine the method of weight shifting that allows the person to consistently perform the maneuver and sustain the off-loaded pressure for the duration of time recommended. Each individual must be assessed for the weight shifting method that is optimal for their performance. Re-education regarding this significant change in duration of off-loaded pressure is important to communicate during out-patient visits to medical personnel who are providing care to the person who has been living with SCI and when reassessing seating and mobility equipment. Retraining in alternative methods or a change in mobility equipment may be necessary if the person cannot sustain the increased off-loading time. A referral to an occupational or physical therapist may be necessary for this re-assessment and training to learn a new weight shift method.

Henderson (1994) studied average ischial tuberosity pressures for different postures, including upright resting posture, tilting back to 35 and 65 degrees, and 45 degree forward lean. Forward leaning demonstrated a significant reduction of ischial tuberosity pressures.

Hobson (1992) found that an ischial pressure reduction could be obtained by forward leaning but that an increase in ischial tuberosity pressure was noted with up to 30 degrees of forward flexion prior to when any reduction began to occur. Henderson (1994) studied average ischial

tuberosity pressures for different postures, including upright resting posture, tilting back to 35 and 65 degrees, and 45 degree forward lean. Forward leaning demonstrated a significant reduction of ischial tuberosity pressures. During lateral leaning, a 32% to 38% decrease in average pressure on the opposite side was found to occur yet no indication of the pressures seen on the weighted side were noted. Lateral trunk leaning to 15 degrees reduced pressure on the unweighted side, but the impact on the weighted side was not reported (Henderson et al., 1994). Sonenblum and Sprigle (2011) showed that the seated tilt of power wheelchairs creates pressure redistribution and increased blood flow during maximum tilts at 45 to 60 degrees in more than 80% of subjects.

A study which compared the efficacy of a dynamic wheelchair cushion and a tilt-in-space wheelchair with conventional cushion in providing pressure redistribution for patients with tetraplegia showed that both a dynamic cushion wheelchair and a tilt-in-space wheelchair with conventional cushion provided similar pressure redistribution over the ischial tuberosities (Burns and Betz, 1999).

For individuals with SCI who are dependent on a wheelchair for all mobility, it is crucial that they use a wheelchair and seating system that has been customized for their unique physical and functional needs. This wheelchair and seating system is essential to contributing to positive health maintenance as well as unencumbered participation in life by providing optimal mobility. An individualized prescribed wheelchair chosen specifically for the user can facilitate healthy tissue viability, symmetrical and balanced posture, and optimal mobility, allowing for mobility-related activities of daily living.

A specially selected wheelchair seat cushion and back support should be used at all times when persons are out of bed. Pressure ulcers occurring at the ischial tuberosities and the sacrum are likely to be a result of being seated in a wheelchair. A seat cushion and solid back that relieves and redistributes pressure and reduces risk of pressure ulcer formation is an important aspect of prevention (Bogie et al., 1995).

In meeting the complex seating needs of persons with SCI, it is advisable to obtain an evaluation from a specialized seating clinic that employs a comprehensive approach (Coggrave and Rose, 2003). This approach should include a review of medical history, postural and functional assessment, skin history assessment, visual inspection of skin of sitting surfaces,

pressure mapping, discussion of home and community environments, exposure to seating and mobility options, actual trial of various systems, education of the person with SCI and his or her caregivers, and collaboration of the therapeutic seating team with a knowledgeable complex medical equipment supplier, preferably those with the assistive technology professional (ATP) designation.

Ongoing assessments of the compatibility of each individual and his or her equipment are important as people change over time (Garber and Krouskop, 1997; Chen et al., 2005). Routine maintenance and replacement of parts reduce the possibility that poor equipment conditions will contribute to pressure ulcers. The very equipment that has been selected to prevent pressure ulcers may contribute to them if it is inappropriate, inadequate, or poorly maintained.

Exercise

7. Implement an ongoing exercise regimen to promote maintenance of skin integrity and prevent contractures.

(Scientific evidence—III, V; Grade of recommendation—C; Strength of panel opinion—Strong)

Maintaining physical endurance, mobility, and joint range-of-motion is an appropriate goal for most individuals (Bergstrom et al., 1992). Immobility can adversely affect pulmonary function, joint range of motion as well as muscle strength and bulk. For individuals confined to bed while they are being treated medically for pressure ulcers, facilities may have protocols for providing these patients with appropriate exercises. Physical and occupational therapists often are asked to intervene to prevent the serious loss of function and independence that results from imposed bed-rest. After pressure ulcer surgical intervention, the rehabilitation team, including the plastic surgeon, will modify range-of-motion recommendations to prevent dehiscence of the wound (Lewis, 1994). Facility-specific protocols introducing range of motion of the hips, are implemented when the patient is cleared by. In addition, sitting on toilet seats can put extreme on comprised or newly healed tissues over the ischial tuberosities.

Participation in athletic activities have been associated with less pressure ulcer development in individuals with SCI of comparable age, severity of spinal cord injury, and pre-injury health. (Stotts, 1986).

Nutrition

8. Assess nutritional status, including dietary intake, anthropometric measurements, biochemical parameters (prealbumin, total protein, albumin, hemoglobin, hematocrit, and total lymphocyte count) fasting blood sugar, liver function panel, folate, and vitamin B12.

(Scientific evidence—II, III, IV, V; Grade of recommendation—B; Strength of panel opinion—Strong)

Although there is consensus that malnutrition is a major risk factor related to the development of pressure ulcers as well as the prolonged healing of pressure ulcers in individuals with SCI (Maklebust and Magnan, 1994; Vidal and Sarrias, 1991; Wagner et al., 1996), there is no universal agreement of the definition of malnutrition. The Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) has recommended two or more of the following characteristics for the diagnosis of malnutrition:

- (1) Insufficient energy intake,
- (2) weight loss,
- (3) loss of muscle mass,
- (4) loss of subcutaneous fat,
- (5) localized or generalized fluid accumulation that may sometimes mask weight loss, and
- (6) diminished functional status as measured by hand grip strength (White, J.V., 2012).

Although malnutrition and overall nutritional status have been correlated with the development and healing of pressure ulcers (Ek et al., 1991; Strauss and Margolis, 1996), no single biomarker, biochemical parameter or otherwise, of nutrition has been identified as a strong predictor related to prevention or healing of pressure ulcers. Serial measurements of multiple biomarkers of nutritional status to assess trends over time may be the most fruitful method for estimating baseline nutritional status and assessing the response to a nutritional intervention. Recommended evaluations should be interpreted collectively, with consideration given to possible non-nutritional factors, such as age, gender, economic, psychosocial issues, over- or under-hydration, drug-nutrient interactions, physiologic stress, infection, and concurrent illnesses. This comprehensive assessment and monitoring of nutritional status can lead to appropriate interventions for both prevention and healing of pressure ulcers.

The poor nutritional state of many patients often goes unrecognized by health professionals who receive little training on nutritional issues (Wong et al. 2012: *Spinal Cord* 50, 446–451). The use of a SCI specific nutrition screening tool, such as the Spinal Nutrition Screening Tool (SNST) may improve the identification of persons at nutritional risk for developing pressure ulcers. The SNST assesses eight criteria, of which the majority are recognized predictors or symptoms of undernutrition: history of recent weight loss, body mass index (BMI), age, level of SCI, presence of co-morbidities, skin condition, appetite, and ability to eat. Each step of screening has a score of up to 5, and the total score reflects the patient's degree of risk. A score of 0–10 indicates a low risk of undernutrition, 11–15 indicates moderate risk of undernutrition, and greater than 15 indicates high risk of undernutrition (Wong et al. 2013, *Eur j Clin Nutr* 66, 382–387). However, in a recent multicenter study from the United Kingdom on malnutrition risk, the percentage of patients identified at risk of undernutrition using the SNST was only slightly greater at 44.6% compared with 40.0% using a generic nutrition screening tool (Wong et al., 2012 *Br J Nutr* 108, 918–923).

DIETARY INTAKE

A dietary history can illustrate the adequacy of an individual's usual food intake. Factors that contribute to inadequate nutritional intake are poor appetite, food intolerances and allergies, difficulty with chewing and swallowing, difficulty with food acquisition and preparation, immobility, social neglect, lack of knowledge about healthy food choices, depression, and poverty (Waterlow, 1996).

Inadequate intake of food and a consecutive 3-day worsening of appetite have been identified as significant predictors of pressure ulcer development (Berglund and Nordstrom, 1995; Bergstrom and Braden, 1992; Ek et al., 1991; Tourtual et al., 1997). Individuals who develop pressure ulcers have significantly lower calorie and protein intake than do those who do not have pressure ulcers (Bergstrom and Braden, 1992). Successful dietary management of malnutrition often includes advice regarding meal planning, assistance with meal preparation, use of assistive eating devices, and change of meal patterns to six small feedings daily. Healthy high calorie and high protein foods (e.g., coconut milk, nuts/nut butters, avocados, whole grain pastas and whole grain breads, dried fruits, whey protein, and cottage cheese) can also be included in the individual's diet to enhance intake. When dietary

intakes do not meet estimated requirements, interventions are necessary to provide required nutritional support.

ORAL SUPPLEMENTS

Commercial oral supplements are available in liquid and solid forms as well as in puddings and bars to supplement an individual's usual diet (Himes, 1997). Liquid supplements are provided with different nutrient densities, ranging from 1.0 to 2.0 kilocalories per mL, with 13% to 25% of total calories as protein, and the recommended dietary allowance for vitamins and minerals in approximately 1,000 to 1,500 mL of formula. Commercial flavored breakfast drinks are not recommended as these drinks are often laden with sugar, artificial flavors, and colors.

In one study, 200 mL of liquid nutritional supplement given twice daily in addition to a standard hospital diet was associated with the development of fewer pressure ulcers and the healing of existing pressure ulcers to a greater extent than in an unsupplemented control group (Ek et al., 1991).

ENTERAL FEEDING

When the gastrointestinal tract (GI) is functional but oral dietary intake is inadequate, enteral nutrition through a feeding tube is the preferred method of nutritional support. The decision regarding the route of enteral access depends on the anticipated duration of tube feeding and the risk of pulmonary aspiration of stomach contents. Short-term access (less than 4 to 6 weeks) is possible through the nasogastric, nasoduodenal, or nasojejunal routes. For long-term access (greater than 6 weeks), surgical or percutaneous endoscopic gastrostomy or jejunostomy tubes can be inserted.

Use of enteral feedings should be strongly considered if an individual is unlikely to meet his or her estimated nutritional needs within 3–5 days.

Enteral feeding formulas differ by calorie and protein density, fiber content, form of nutrients, and amounts of micronutrients. Selection of the appropriate formula depends on the individual's digestive and absorptive capacity and on specific indications for the formula. Formulas can be administered by bolus, intermittent, cyclic, or continuous methods. The most common complications associated with tube feedings are diarrhea and tube obstruction.

TOTAL PARENTERAL NUTRITION (TPN)

Parenteral nutrition support is indicated in the presence of mechanical obstruction of the GI tract, prolonged ileus, severe GI hemorrhage, severe diarrhea, intractable vomiting, and high-output GI fistula. Concentrated TPN solutions (> 900 mOsm) are only tolerated through central venous catheters and may cause thrombophlebitis of peripheral veins. Patient-specific formulas can be designed to deliver individualized nutrient requirements. Complications of TPN include mineral and electrolyte imbalances, acid-base disorders, and catheter-related infections.

ANTHROPOMETRIC MEASUREMENTS

Commonly assessed anthropometric measurements include body weight, BMI (weight/height²), and triceps skinfold thickness (TSF). Published anthropometric standards are based on a non-SCI population and do not consider the body composition changes (water shifts, muscle atrophy from disuse, increased percentage of body fat) that normally occur in individuals with SCI. As ideal body weight standards have not yet been established for individuals with SCI, the Academy of Nutrition and Dietetics Spinal Cord Injury Evidence-Based Nutrition Practice Guideline suggests utilizing the Metropolitan Life Insurance tables to estimate target body weight for individuals with spinal cord injury with estimated adjustments. Reported methods of adjustment for tetraplegia include a reduction of 10–15% or a decrease of 15–20 pounds from the table weight, while for persons with paraplegia, a reduction of 5–10% or 10–15 pounds lower than table weight has been recommended.

Decreased body weight (< 80% of ideal weight) and low BMI (17.6 ± 4.6) have been correlated with severe malnutrition and pressure ulcer development (Bonney et al., 1995; Ek et al., 1991; Strauss and Margolis, 1996). It is thought that the weight loss reduces fat and muscle tissue, resulting in elevated pressures over bony prominences and increased damage to microcirculation, thus contributing to pressure ulcer development (Schubert et al., 1994). Nevertheless, BMI should be used with caution to measure body composition in persons with SCI due to concerns with accuracy (Jones et al., 2003). If available, bioelectric impedance analysis (BIA) or dual-energy X-ray absorptiometry (DEXA) should be used to assess body composition for persons with SCI who are medically stable. Evidence suggests that BIA and DEXA correlate with measures of total body water (TBW) when labeled water is used to provide a reference value for TBW.

TSF measurements have been found to be significantly lower in individuals with pressure ulcers than in those who do not have them (Bonney et al., 1995; Ek et al., 1991). Depleted TSF, defined as less than 3.0 mm for females and less than 2.5 mm for males, has been significantly associated with pressure ulcer development and longer hospital lengths of stay (Allman et al., 1995). However, TSF measurements should also be used with caution in persons with SCI due to concerns about accuracy, as any changes found may be due to SCI rather than malnutrition as Maggioni et al. (2003) demonstrated in comparing skinfold measurements and dual X-ray absorptiometry (DXA) between individuals with and without SCI finding that the skinfold method did not differ between the two groups. However, fat mass was significantly greater in the SCI group compared to controls when DXA was used.

BIOCHEMICAL PARAMETERS

Several biochemical parameters have been associated with the development or presence of pressure ulcers. Normal reference ranges may vary according to the laboratory consulted.

Prealbumin

Serum prealbumin levels are usually lower (14 ± 4 mg/dL) in people with pressure ulcers as compared to those without ulcers (Bonney et al., 1995). Prealbumin is a sensitive indicator for monitoring nutritional adequacy due to its short half-life of 2–3 days (Tuten et al., 1985). However, there are some limitations of interpreting prealbumin levels. Prealbumin is synthesized in the liver and therefore synthesis is depressed in the presence of hepatic disease. Rather than monitoring prealbumin level for a specific range, monitoring any trend in serial prealbumin levels may be more useful.

Albumin and Total Protein

Historically, serum albumin levels have been used as an indicator of nutrition status. Albumin is synthesized in the liver, has a long half-life (12–21 days), a large body pool, and may be influenced by many non-nutrition related factors (i.e., Inflammation, hepatic disease, fluid status). A decrease in serum albumin levels cannot always be correlated with visceral protein losses. Serum albumin levels less than 3.5 mg/dL have been significantly associated with an increased incidence of pressure ulcers (Blaylock, 1995; Ek et al., 1991; Lehman, 1995; Rochon et al., 1993; Salzberg et al., 1996). Individuals who have serum albumin levels of 3.5 g/dL or higher have lower incidences of pressure ulcers than do individuals with albumin levels lower than 3.5 g/dL (Bergstrom and Braden, 1992; Tourtual et

al., 1997). Serum albumin levels have also been inversely related to the worst category/stage of a pressure ulcer and significantly associated with lifetime incidence of pressure ulcers, number of different sites, and recurrences (Salzberg et al., 1996).

A diet rich in calories and protein is recommended to improve serum albumin levels instead of intravenous albumin administration because intravenous albumin is deficient in essential amino acids and provides only transient increases in serum albumin (Fuoco et al., 1997).

Nutritional status as measured by serum albumin has not been correlated with the development or healing of pressure ulcers in some studies (Allman et al., 1995; Day and Leonard, 1993). No significant differences in serum concentrations of total protein or albumin were found between individuals with “slow” (no healing within 5 weeks after initiating therapy) and “fast” (healing within 5 weeks of treatment) healing ulcers (Segal et al., 1997). These conflicting findings could be explained by factors other than nutritional status. Factors associated with hypoalbuminemia include losses of protein and albumin into the pressure ulcer exudate (Allman et al., 1995) and the presence of a chronic cytokine-induced inflammatory state (Bonney et al., 1995; Segal et al., 1997; Strauss and Margolis, 1996).

Albumin and prealbumin are negative acute phase reactants. They are inversely influenced by inflammatory and stress responses. C-reactive protein, a protein that increases with stress, is useful in interpreting whether albumin and prealbumin are being affected or decreased by inflammation and stress.

Serum total protein levels less than 6.4 g/dL have been associated with pressure ulcer development (Blaylock, 1995; Salzberg et al., 1996; Tourtual et al., 1997).

Hemoglobin and Hematocrit

Anemia, assessed by hemoglobin and hematocrit levels, reduces oxygen supply to tissues, thus impairing healing of pressure ulcers. Hemoglobin levels below 12–14 g/dL are associated with increased incidence of pressure ulcers (Lehman, 1995; Rochon et al., 1993; Salzberg et al., 1996; Tourtual et al., 1997). Hematocrit levels below 36% have also been inversely associated with lifetime total pressure ulcers, the depth of the ulcer, and the number of different sites (Salzberg et al., 1996). Iron therapy is not necessarily recommended to correct hemoglobin and hematocrit levels because anemia could result from an inability to use iron

stores rather than from iron deficiency (Fuoco et al., 1997).

Total Lymphocyte Count

Decreased total lymphocyte count (< 1500/mm³) is an independent significant risk factor associated with the development of pressure ulcers (Allman et al., 1995; Lehman, 1995). Lymphopenia can also result from non-nutritional factors, such as infections and steroid use that compromise immunocompetence.

9. Provide adequate nutritional intake to meet individual needs, especially for calories (or energy), protein, micronutrients (zinc, vitamin C, vitamin A, and iron), and fluids.

(Scientific evidence— I, II, III, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

CALORIES (ENERGY)

Calories are required to fuel basic life processes and to spare lean body mass from being used to meet metabolic demands. Individuals with SCI commonly have lower energy needs than matched individuals without SCI, in part due to decreased metabolic demand by denervated muscles. An estimate of the difference in basal energy expenditure between persons with SCI who have severe pressure ulcers and those who do not have pressure ulcers is approximately 5 Kcal/Kg of body weight per day. This has been demonstrated in several studies using indirect calorimetry and has been replicated in other populations with and without pressure ulcers (Alexander et al. (1995); Liu et al. (1996); Sergi (2007)). Both pressure ulcer surface area and severity have been found to be significantly related to percent of predicted energy expenditure (Liu et al., 1996; Sergi 2007). One possible explanation for the increased energy expenditure found in those with pressure ulcers is the underlying chronic inflammatory processes induced by cytokines and cortisol (Bonney et al., 1995; Segal et al., 1997).

PROTEIN

Protein is essential for tissue growth, maintenance, and repair. A high protein intake is needed for optimal healing of pressure ulcers (Allman et al., 1995). Breslow et al. (1993) reported that administration of supplemental nutritional formulas containing 24% protein instead of 14% administered for 8 weeks in a malnourished nursing home population can result in a decrease in pressure ulcer surface area correlated to both dietary protein and calorie intake per kilogram of body weight.

Recommendations for increased protein requirements in persons with pressure ulcers range from 1.25 to 2 grams protein/Kg of body weight per day with the higher requirements suggested for those with ulcers of greater severity (Bergstrom et al., 1994; Breslow et al., 1993; Chin and Kearns, 1997).

Protein recommendations need to be individualized and take into consideration if there is concurrent hepatic or renal dysfunction for which excess protein consumption can be harmful.

AMINO ACIDS

One observational study comparing healing rates of pressure ulcers in persons with SCI who were administered 9 grams of a commercial powered arginine supplement per day with historical controls reported a significantly shorter mean healing time in the intervention group (Brewer 2010).

MICRONUTRIENTS

Deficiencies of micronutrients, especially of zinc, vitamin C, and vitamin A, are associated with poor wound healing. However, strong evidence does not exist to demonstrate that biochemical or dietary deficiencies of micronutrients are major risk factors for pressure ulcer development. Moreover, supplementation of micronutrients in individuals who do not have deficiencies has not been shown to enhance healing of pressure ulcers.

Zinc

Zinc is known to be involved in the structural integrity of proteins, particularly collagen. Cruse et al. (2000) found in a small study that serum zinc levels are lower in those with SCI and pressure ulcers as compared to those with SCI without ulcers (52 mcg/dl as compared to 82 mcg/dl). Others, however, have found serum zinc levels to be similar in people who develop and do not develop pressure ulcers (Bergstrom and Braden, 1992). Evidence also does not support the idea that oral zinc sulfate supplements (220 mg daily) will affect the healing of pressure ulcers within 2 to 3 months (Brewer et al., 1967). Long-term consumption of high amounts of zinc may have adverse physiological effects, such as impaired copper metabolism, which may induce a state of copper deficiency and anemia (Eleazer et al., 1995). However, use for a limited period of time may be considered to correct a deficiency.

Vitamin C

Vitamin C plays a well-known role in the hydroxylation of proline and lysine during collagen formation. However, dietary intake of vitamin C does not predict pressure ulcer development (Bergstrom and Braden, 1992). In addition, supplementation of vitamin C at a dose of 500mg per day has not been shown to accelerate healing of pressure ulcers in individuals who are deficient in vitamin C (ter Riet et al., 1995). Because a subclinical deficiency state is difficult to diagnose, the minimum intake of the RDA of 60 mg of vitamin C has been suggested.

Vitamin A

In one study of 110 SCI individuals, those without a pressure ulcer over a 12-month period were found to have higher vitamin A levels (Moussavi et al., 2003). Vitamin A deficiency can result in delayed wound healing. However, no differences have been reported in the vitamin A intake of individuals who do or do not develop pressure ulcers (Bergstrom and Braden, 1992).

Iron, Vitamin B12 and Folate

Anemia assessed by hemoglobin and hematocrit levels reduces oxygen supply to tissues, thus impairing healing of pressure ulcers. If low hemoglobin concentration is a result of iron, Vitamin B12 or folate deficiency anemia, it may be a factor in tissue hypoxia and impaired wound healing. Supplementation should be provided as indicated to correct iron, Vitamin B12, or folate deficiency anemias if found.

FLUIDS

Inadequate fluid intake is a risk factor in the development of pressure ulcers (Berglund and Nordstrom, 1995; Ek et al., 1991). The Academy of Nutrition and Dietetics evidence-based guideline for the non-SCI population, specifically 30 mL to 40 mL per kilogram body weight or a minimum of 1 mL per kilocalorie per day. For those with pressure ulcers, additional fluid loss may come from wound drainage and evaporative losses caused by fever. A 10 mL to 15 mL per kilogram additional amount of fluid may be required with the use of air fluidized beds set at a high temperature (more than 31° to 34°C or more than 88° to 93°F) due to resultant increased evaporative water losses (similar to fever) (Breslow, 1994). Additional consideration should be given to fluid intake in individuals with conditions in which fluid needs to be restricted, such as in renal and cardiac disease.

Education—Health Care Professionals, Persons with SCI, Family, Caregivers

10. Provide individuals with SCI, their family, significant others, and health-care professionals with specific information on effective strategies for the prevention and treatment of pressure ulcers. This should include the following:

- **Pressure ulcer etiology**
- **Reducing pressure ulcer risk**
- **Skin cleansing and care techniques**
- **Management of incontinence**
- **Frequency and techniques of skin inspection**
- **Frequency, duration, and techniques of recommended position changes**
- **Frequency, duration, and techniques of recommended pressure redistribution**
- **Nutrition as it relates to maintaining skin integrity**
- **Use and maintenance of support surfaces (mattresses and cushions)**
- **Skin changes to be reported to the health-care team**

(Scientific evidence—II, III, IV, V; Grade of recommendation—B; Strength of panel opinion—Strong)

Education of the patient, caregivers, and family members is critical for pressure ulcer prevention and management. Persons with SCI have a lifetime need for education regarding skin care and prevention and treatment of pressure ulcers. It is essential that individuals be provided with the basic knowledge necessary to return them to home and community (Fowler and Pelfrey, 1993). Learning styles should be identified and appropriate teaching strategies to include appropriate content material, time of dissemination, and recurrent education when needed should be implemented for the individual with SCI as well as family and personal care assistants.

Hospital-based education programs, especially during initial rehabilitation following a SCI, are inadequate to effect implementation of preventive practices once the person has returned to his home and community. Rarely is formal or informamal education programs assessed for their effectiveness in reducing ulcers (Garber et al., 1996). Information about the complex management of SCI cannot be absorbed

during the short hospital stays. Individuals are leaving the hospital with less information about self-care and there are very few opportunities for reinforcement of the information presented during hospitalization. Patients may return home with reams of papers instructing them on everything from maintaining nutrition to managing bowel and bladder. Usually, it is only after the appearance of the first skin breakdown that attention is turned to addressing the pressure ulcer problem. Often, the person does not even know whom to contact and delays treatment (Garber et al., 1996).

In a study by Schubart et al. (2008), investigators identified several educational needs of persons with SCI:

- (1) an awareness of lifelong risks for developing pressure ulcers, including the ability to assess risk factors and how risk changes over time;
- (2) an ability to take charge of skin care regimen and to partner with health-care providers;
- (3) an adoption of prevention strategies consistently that fit level of functioning and activity and an ability to update practices as risk changes; and
- (4) an ability to coordinate social supports.

Patient education for SCI patients is a shared responsibility of all professionals caring for the patient. All educational programs should provide current evidence-based information for the patient and family. Educational materials for patients must be at an appropriate reading level, and should also target family members. Garber et al. (2002) reported that 4 hours of structured individualized education on the prevention of pressure ulcers during hospitalization for surgical repair of a pressure ulcer and up to 24 months after discharge was effective in improving a patient's pressure ulcer knowledge. Computer-aided instruction was found to be more effective as compared to traditional educational methods in increasing the initiation and performance or pressure-relieving techniques for individuals with SCI in one study (Pellerito, 2003). Hoffman et al. (2011) compared in-person education forums for persons with SCI to Internet versions of the forums for a one-year period and participants reported learning new information from the online format 88% of the time as compared to 96% for the in-person format; 91% reported that video was more effective than text for presenting educational information.

One of the most frequently taught preventive behaviors in the acute care, rehabilitation, home, and long-term care settings is daily visual and

tactile skin inspections (Burman, 1993). Daily inspection of skin areas of the body has been found to be associated with decreased risk of pressure ulcers. Those who perform daily skin inspection are able to detect pressure or shear damage early and modify their self-management routine accordingly (Raghavan et al., 2003).

Individuals should learn to describe the most frequently affected body locations and the normally accepted descriptions of pressure ulcer stages, so that more accurately reported information can be communicated should a problem occur after the individual has been discharged to a home or community setting. The more accurately a person is able to describe a skin area, the more likely it is that the person receiving the report can make appropriate recommendations for actions to be taken (Garber et al., 1996).

An individual's poor understanding of health behavior instructions could result from many sources: lack of education, cognitive impairment, inadequate education from health professionals, distraction due to psychological distress, and/or distorted and biased processing of unwelcome information (Lieberman and Chaiken, 1992). Assessing an individual's understanding of health behavior recommendations through direct, specific questions is thought to be an effective approach. Additionally, identifying points needing clarification and providing explanations and giving special attention to changes in lifestyles and daily routines should be incorporated into any intervention. Krouskop et al. (1983) observed a decreased yearly incidence of pressure ulcers among individuals of a large SCI outpatient clinic following application of a systematic prevention program; rates dropped again by half when components of psychological counseling and patient/family education were introduced. Peer support groups can help persons better understand their needs through peers and education, when available, they should be offered. Well-timed and recurrent education and support will help persons with SCI be more successful in self-management.

What distinguishes persons with SCI who have pressure ulcers from other vulnerable populations, is the fact that persons with SCI are encouraged to take responsibility for self care, either directly or indirectly. Certainly, persons with paraplegia are able to perform independently many of their activities of daily living. Persons with tetraplegia are strongly encouraged to be active in directing another person in managing their many needs. This is

especially important with regard to skin inspection and the early detection of potential pressure ulcers (Garber, et al., 1996).

Assessment and Reassessment Following Pressure Ulcer Onset

Assessment of the Individual with a Pressure Ulcer

11. Perform an initial comprehensive assessment of the individual with a pressure ulcer, to include the following:

- Complete history and physical examination
- Complete skin assessment
- Laboratory tests (evaluate for infection, anemia, diabetes, and nutritional status)
- Psychological health, behavior, cognitive status, and social and financial resources
- Availability and utilization of personal care assistance (family, caregiver support, financial)
- Positioning, posture, and all durable medical equipment
- Nutritional status
- Activities of daily living (ADLs), mobility, and transfer skills, as related to maintaining skin integrity

(Scientific evidence–N/A; Grade of recommendation–N/A; Strength of panel opinion–Strong)

Individuals with SCI who present with pressure ulcers should have a complete history and physical examination. The etiology and mechanism of ulcer development should be determined and risk factors should be assessed (see recommendation 1). The contribution of underlying disease processes and co-morbidities should be evaluated.

Initial evaluation should also include a psychosocial assessment of cognitive status, depression, substance abuse, other potentially contributory psychological disorders, as well as an evaluation of psychosocial support systems. Generally, these are part of the facility-specific admission protocol following the onset of a pressure ulcer.

Common findings in patients with pressure ulcers are anemia with reduced serum iron, transferrin, total iron-binding capacity, increased ferritin, erythrocyte sedimentation rate, C-reactive protein, white blood cells, and reduced lymphocytes, total protein, albumin, and zinc (Gurcay, E., et al., 2009). Animal studies have shown the value of obtaining serum and urine biomarkers of pressure-induced deep tissue injury. Creatine kinase, myoglobin, heart-type fatty acid binding protein, myosin, and troponin-I have been found to show a rapid increase of concentration upon compression, which may provide an indication of deep tissue injury (Makhsous et al., 2010). Further studies will be needed before biomarkers for deep tissue injury should be used for routine clinical practice.

Malnourished patients are at high risk for pressure ulcer development and with the presence of pressure ulcers are likely to experience delayed healing. Serum measurements of visceral proteins (albumin, total protein, and prealbumin) levels estimate the adequacy of the individual's nutritional intake.

Assessments of posture, positioning, and equipment are important in determining the causation of pressure ulcers; such assessments are critical in developing effective prevention and treatment strategies. It is important to identify all seating surfaces a person might use including shower/commode chairs, lounge chairs, desk chairs, etc. An individual's posture and level of pressure on the support surface should be evaluated as well as an individual's efficiency in transferring and performing pressure redistribution. All support surfaces on any seating device should be checked for moisture at pressure points and for mechanical integrity, deterioration, and fatigue.

Psychosocial supports (family, friends, caregivers) and resources (financial, medical) should be explored since they may be of critical importance in sustaining the optimal treatment plan. The individual's self-care capability and the availability of appropriate personal care assistance also should be determined. If the adequacy of available treatment resources cannot be assured, delivery of the prescribed treatment plan will be in jeopardy.

Assessment and Reassessment of the Pressure Ulcer

12. Describe and document in detail an existing pressure ulcer and its treatment. Include the following parameters:

- **Anatomical location and general appearance**
- **Category/Stage**
- **Characteristics of the wound base**
 - **Viable tissue (granulation, epithelialization, muscle, bone, or subcutaneous tissue)**
 - **Nonviable tissue (necrotic, slough, eschar)**
- **Size of wound – length x width x depth**
- **Exudate amount and type**
- **Odor**
- **Wound edges**
- **Periwound skin**
- **Wound pain**
- **Documentation of current treatment strategies and outcomes to date.**

An objective and thorough description of pressure ulcers enables the development of an appropriate treatment plan, form the basis for serial assessment to determine response to treatment, and provides a reliable means of communicating wound status among health-care professionals. A description of location, size, and severity of the ulcer is important in assessing mechanism of injury and positional restrictions. These restrictions should be incorporated into the treatment plan.

(Scientific evidence–N/A; Grade of recommendation–N/A; Strength of panel opinion–Strong)

13. Monitor, assess, document, and report any observable/visible change in wound status.

- **Monitor the pressure ulcer with each dressing change, or if there is no dressing then routinely.**
- **Conduct a comprehensive assessment of the parameters listed in Recommendation No. 12 at regular intervals**

(Scientific evidence–II, V; Grade of recommendation–B; Strength of panel opinion–Strong)

Initial wound assessment and subsequent reassessment provide the basis for pressure ulcer management. Although randomized clinical

trials have not identified the optimal frequency of reassessment, this parameter is integral to wound evaluation. Bergstrom et al. (1994) recommend weekly reassessment of pressure ulcers to determine the individual's response to the care plan, while van Rijswijk and Braden suggested that healing should be monitored during each dressing change and reassessed at least weekly (van Rijswijk and Braden, 1999). Lazarus et al. (1994) advised that wound changes always be correlated with changes in the individual's health status.

When determining reassessment intervals, consideration should be given to the individual's health status, care setting, pressure ulcer category/stage, and other variables (van Rijswijk, 1995). Reassessment intervals may vary for individuals in rehabilitation, acute, subacute, extended care, or home-care settings. Furthermore the goal of reassessment may differ among wounds, whereas the rationale for reassessment of category/stage II ulcers may be to detect epithelialization, the rationale for reassessment of category/stage III and IV ulcers may be to detect the signs and symptoms of infection and granulation (van Rijswijk, 1995).

An objective and thorough description of pressure ulcers enables the development of an appropriate treatment plan, forms the basis for serial assessment to determine the response of the wound to treatment, and provides a reliable means of communicating wound status among health-care professionals.

The anatomic location of a wound should be clearly delineated and specified. Pressure ulcer locations should contain the name of the bone against which pressure is applied, for example, the plantar aspect of the foot would not be adequate, but the metatarsal head would be an accurate representation of location; the medial malleolus instead of the ankle; the trochanter, instead of the hip. The extent of tissue loss guides the selection of interventions and helps the clinician determine the potential healing time.

Before assessing the wound it is important to remove all wound debris by thorough cleansing of wound and periwound skin. Skin involvement may be full thickness or partial thickness. If the wound's etiology is pressure, the wound should be staged accordingly (see "Staging of Pressure Ulcers").

The characteristics of the wound base may vary within the wounds. Tissue types should be described in percentages. Descriptors of tissue type may include: granulation tissue, epithelial tissue, muscle tissue, subcutaneous tissue,

eschar, or slough. There may be viable tissue, such as granular, epithelial, muscle, or subcutaneous tissue, as well as nonviable tissue, such as eschar, slough, or clean, nongranulating wound base. For example, a description of "20% adherent and loose necrotic slough, 30% nongranulating, and 50% granulation tissue" may indicate the extent that a wound is or is not progressing if performed serially. Biofilm on the wound base should raise concern. Biofilm consists of polysaccharide polymers bound together by metal ions creating a viscous gel-like substance that acts as a physical barrier impermeable and resistant to the action of antimicrobial agents. Biofilms can appear as a stubborn, slimy film frequently overlying the granulation tissue. The presence of granulation tissue is evidence of healing in the ulcer base and is typically beefy red, bumpy, or pearly and shiny. Epithelialization is the regrowth of epidermis across the surface of the pressure ulcer. The presence of eschar and its appearance should be documented. A black eschar is indicative of dried necrotic tissue, while a yellow covering of the wound surface may be indicative of a fibrin slough. A clean, red appearance of the wound base indicates the absence of necrotic tissue. Wound measurement techniques include

- (1) simple linear measurements of length by width and depth;
- (2) traditional wound tracings to determine surface area;
- (3) digital photo-planimetry calculating a wound area from digital photographs;
- (4) computerized assisted tablets using a wound tracing retraced into a digital tablet; and
- (5) a combination of a hand-held personal digital assistant (PDA) and laser beams, referred to as a scanner, to correct image scale and skin curvature (Romanelli et al., 2012).

Wound dimensions provide a valuable indicator of healing progression. Although sophisticated wound size measurement techniques (direct measurement of volume, tracing planimetry, and so forth) may provide the most precise measurements of wound size (Cutler et al., 1993; Griffin et al., 1993; Hayward et al., 1993; Hooker et al., 1988; Haghpanah et al., 2006), routine clinical assessment should include measuring wound size (length, width, and depth) with a ruler. Length should be along the longest dimension of the wound and width is the maximum dimension perpendicular to the length axis. The depth of the wound should be measured from the deepest point to the imaginary surface

in continuity with the wound edges. Undermining, tracks, and tunneling can be documented by measuring depth and noting the location using the face of the clock as a guide. Undermining may be circumferential or specific to a location, using the individual's head as the landmark for the 12 o'clock position, 90 degrees to the right for the 3 o'clock position, the feet for the 6 o'clock position, and 270 degrees clockwise from the head for the 9 o'clock position.

Although a narrative description of the wound may suffice, photography can also be useful in creating a record of wound appearance (Dufrene, C. 2009). Because the accurate determination of wound edge position is difficult from plain photographs, a marker pen outline of the wound can be made on a transparent dressing if photography is used to document wound size. Inclusion of a calibrated grid or measuring scale in the photograph is also recommended (Cutler et al., 1993). Wound photo documentation provides an objective assessment of wound status at any point in time from two-dimensional and three-dimensional wound measurement systems to digital photo-planimetry systems that can accurately document the course of healing of wounds (Wendelken et al., 2011) and can be imported to electronic medical records. Wound photo documentation should clearly show the wound bed characteristics, and where there is undermining or tunneling, a measuring tool such as a swab should be inserted into the undermined area or tunnel to indicate its full extent with a visible ruler on top of the skin paralleling that swab located in the undermined or tunneled area to reflect the exact measurement. When using photographs to document a wound, it is important to be consistent with lighting, distance of the wound from the camera lens. Use a ruler for perspective and document the anatomical location of the wound. This allows more accurate assessment and comparison of the wound picture to other pictures.

The composition of wound exudate indicates the wound healing status and varies according to the individual's health. Wound exudate is assessed to include amount (none, light, moderate, or heavy), type (serous, serosanguineous, purulent yellow, tan, or green), and odor (none, mild, moderate, or strong).

Examination of the wound edges should be performed to determine the presence of undermining, wherein the ulcer cavity extends laterally beneath the edges of the wound. Rolled wound edges (epibole) indicate that the epithelial tissues have migrated down and around the

wound edges. In this case, it is unlikely that the wound will close spontaneously. Debridement of the wound edges usually needs to be done to facilitate progression to wound closure.

Ulcers manifesting as a small skin surface opening in communication with a larger cavity are sometimes referred to as "closed ulcers." Although some measurement of dimension for these ulcers can be obtained with the use of cotton-tipped applicator probes, accurate determination of closed ulcer dimension requires the use of sinography (Hooker et al., 1988; Hooker and Sibley, 1987).

Periwound skin should be assessed for color (erythema, white, blue), texture (moist dry, indurated, boggy, macerated), temperature (warm, cool), and presence of denudement, maceration, excoriation, stripping, erosion, papules, pustules, and lesions. Erythema, warmth, induration and swelling may be indicative of cellulitis. Maceration of surrounding skin may be the result of feces, urine, or wound drainage contamination. The presence of maceration may pose a significant risk for wound deterioration and enlargement.

Because individuals with darkly pigmented skin may not show evidence of reactive hyperemia at the early stages of pressure ulcer development, other methods of determining skin damage need to be used. In these individuals, areas of damaged skin appear darker than surrounding skin and may be taut and shiny, indurated, and warm to the touch. Color changes may range from purplish to blue and when compressed, pressure-damaged intact dark skin does not blanch (Bennett, 1995).

The extent of bacterial burden is classified as contamination, colonization, critical colonization, and infection. **Contamination** is the presence of microorganisms on the surface of the ulcer. All category/stage II-IV pressure ulcers have some level of contamination. In **colonization**, microorganisms attach to the ulcer surface and replicate but do not impair healing or cause signs and/or symptoms of infection. All chronic category/stage II-IV pressure ulcers have some level of colonization. With **critical colonization**, microorganisms attach to the ulcer surface, replicate and multiply to a level that affects pressure ulcer healing without provoking systemic signs of infection. The organisms remain on the wound bed and have not yet infected the soft tissue. There are no systemic responses, such as fever or leukocytosis at this point. Wound bed may appear clean but nongranular. **Infection** occurs when microorganisms on the ulcer surface

invade the healthy tissue, overwhelm host resistance, and create cellular injury leading to local or systemic symptoms. The classic signs of infection are increased purulent exudate, induration, warmth, pain, tenderness, and periwound erythema. Chronic wounds may not necessarily show signs of infection, but its presence is evident from delayed healing, discolored granular tissue, breakdown at the wound base, and foul odor. Clinically significant wound infection (as distinct from colonization) is felt to be an important cause of delayed wound healing. Gross exudate should not be routinely cultured. Deep tissue biopsy is the most accurate means of determining soft tissue infection.

Wound pain may indicate infection or wound deterioration in persons with preserved sensation at the level of the pressure ulcer. Pain should be measured routinely and frequently using validated pain assessment scale such as the International Pain Basic Data Set (<http://www.iscos.org.uk/international-sci-pain-data-sets>). In persons with SCI who are prone to autonomic dysreflexia from a noxious stimulus occurring below the level of injury, the development of autonomic dysreflexia especially when there is coincident irritation of a pressure ulcer from either direct or shear pressure, indicates that something should be done emergently to relieve this pressure. In this situation, autonomic dysreflexia may be considered a proxy to pain as may be experienced in persons with greater preservation of the pain pathways. For more information on autonomic dysreflexia, see *Acute Management of Autonomic Dysreflexia: A Clinical Practice Guideline for Health-Care Professionals* (2001).

Initial wound assessment and reassessment provide the basis for pressure ulcer management. While reassessment intervals may vary for individuals in rehabilitation, acute, subacute, extended care, or home-care settings, it should nevertheless be performed on a regular basis according to a consistent protocol. Inadequate healing progression, such as a stalled or worsening wound, should trigger a re-evaluation of the current plan of care and wound management strategy.

Quantitative measurements can be achieved by using any of the well-established tools published in the literature (Mullins et al., 2005) reporting the validity, reliability, strengths, and limitations of each, to monitor healing: (1) Pressure Ulcer Scale for Healing (PUSH) Tool, (2) Sessing scale, and (3) the Bates-Jensen Wound Assessment Tool (BWAT), previously known as the Pressure Sore Status Tool (PSST).

The PUSH tool, designed to assess ulcer progression over time, monitors healing using three domains:

- (1) surface, by multiplying the greatest length and width;
- (2) the exudate amount; and
- (3) the types of tissue that are present in the wound bed. It has been found to be easy to use (Berlowitz et al., 2005) and to be a valid measure of the healing progress (Gardner et al., 2005; Stotts et al., 2001).

The Sessing scale is an observational scale that assigns a numerical value (0 to 6) associated with seven descriptions of the wound surface, without including the size and depth of the ulcer. The scale assesses the following:

1. granulation tissue
2. infection
3. necrosis
4. drainage
5. odor
6. surrounding skin
7. eschar

The scale is scored by calculating the change in numerical value over successive wound assessments over a period of time (Ferrell et al. (1995a).

The Bates-Jensen Wound Assessment Tool (BWAT) contains 13 items that assess the following:

1. wound size
2. wound depth
3. wound edges
4. undermining
5. necrotic tissue type
6. necrotic tissue amount
7. exudate type
8. exudate amount
9. periwound skin color
10. peripheral tissue edema
11. peripheral tissue induration
12. granulation tissue
13. epithelialization (Harris, et al., 2010)

With the advances in technology the assessment and monitoring of pressure ulcers may be performed via telemedicine.

Treatment – Nonsurgical

A comprehensive treatment plan begins with an assessment of risk, health status, and status of the pressure ulcer. The elements of the treatment plan should address cleansing, debridement, dressings, surgery, nutrition, and positioning and support surface use.

Creating a Physiologic Wound Environment

Preparing the wound bed was a concept introduced by Sibbald et al. (2006) who defined it as the promotion of wound closure through diagnosis and appropriate treatment of the cause, attention to patient-centered concerns, and correction of the systemic and local factors that may be delaying healing.

The choice and use of wound cleansing agents, dressings, forms of debridement and adjunctive therapies are guided by the goal of creating a physiologic wound environment for healing. Wound management must focus on the manipulation of the wound to create a physiologic wound environment. This means that any action/decision taken, type of dressing used, use of adjunctive therapies, or combination of therapies, should bring about a wound environment that mimics the healthy and normal function of the skin. Therefore the objectives in creating a physiologic wound environment consist of

- (1) preventing and managing infection,
- (2) cleansing the wound,
- (3) removing nonviable tissue,
- (4) maintaining appropriate level of moisture,
- (5) eliminating dead space,
- (6) controlling odor,
- (7) eliminating or minimizing pain, and
- (8) protecting the wound and periwound skin.

CLEANSE

14. Cleanse pressure ulcer with each dressing change without harming healthy tissue on the wound bed:

- Use normal saline, sterile water, pH-balanced wound cleansers, or lukewarm potable tap water.

- Use diluted sodium hypochlorite ¼ strength to ½ strength solution for wounds with heavy bioburden for limited time only, until clinical evidence of bioburden is resolved.
- Use the following mechanical wound cleansing techniques, to remove wound debris, exudates, surface pathogens, bacteria, and residue from topical creams and ointments.
 - 4–15 pounds per square inch (psi) pressure irrigation with angiocatheter attached to syringe, spray bottle, or pulsatile lavage.
 - Gentle scrubbing of the wound bed with wet gauze.
- Cleanse periwound skin with normal saline, sterile water, pH-balanced skin cleanser, or lukewarm potable tap water with dressing changes.

(Scientific evidence—I, II, III, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Cleansing, a vital part of wound bed preparation, is required to reduce bacterial burden and to remove biofilm, devitalized tissue, metabolic wastes, and residual topical agents that can retard wound healing. There are no randomized controlled trials regarding frequency of cleansing or product use; however, expert clinical opinion indicates that ulcers should be cleansed prior to each dressing change without causing chemical or mechanical trauma to the wound (Barr, 1995).

Normal saline (0.9% NaCl), an irrigant without a preservative, is recommended for wound cleansing due to its noncytotoxic effects in the wound. Although wound healing and infection rates as well as bacterial burden have not been found to be different when normal saline is used as an irrigant as compared to tap water (Griffiths et al., 2001; Svoboda, S.J. et al., 2008).

Although a preponderance of literature can be found on antiseptic cytotoxicity in animal models, (Lineaweaver et al. (1985); Mulliken et al. (1980); Rodeheaver et al. (1982); Niedner and Schopf (1986)) in vitro testing, and acute incised wounds, a minimal amount of literature addresses the effects of antiseptics on chronic human wounds. Michael (1985) described pressure ulcer healing in a small sample of individuals with SCI treated with povidone-iodine. However, currently antiseptic solutions are not recommended for cleansing ulcers on a routine basis due to cytotoxic effects, e.g., povidone iodine has been

found to be cytotoxic to fibroblasts, and lack of increased effectiveness as compared to normal saline (Kucan et al. (1981)). In addition, cases have been reported revealing increased serum iodide concentrations due to absorption of povidone-iodine used in dressings resulting in hyperchloremic acidosis and other electrolyte abnormalities (Aronoff et al., 1980; Dela Cruz et al., 1987; Zamora, 1986).

For wounds with heavy bacterial burden, stronger wound cleansing solutions may be used of various dilutions for limited periods of time until the bacterial burden or biofilm is controlled. As skin cleansers are quite toxic to wound cells (Foresman et al. (1993)), their use should be limited to intact skin. Wound cleansers, described as products that contain surfactants that lower the surface tension thereby facilitating removal of exudate and other foreign matter, are generally not toxic to the wound cells at concentrations that have toxicity indices of less than 10 as measured by standard means (Wilson et al. (2005)).

Diluted sodium hypochlorite solution is most commonly used as a wound cleanser at a concentration of 1/2 to 1/4 strength or even 1/12 strength. A stabilized form of hypochlorous acid with a pH of 3.5 to 4.0 has also been found to be effective in controlling the bacterial bioburden in a wound without inhibiting wound healing, when used for 15 to 30 minutes and followed with another wound application (Robson et al., 2007).

Cleansing techniques generally used include irrigation and appropriate debridement. The goal of cleansing is to remove wound debris and to eliminate the biofilm that forms on the wound bed. Irrigation may be accomplished using a syringe, squeezable bottle with a tip, or battery-powered irrigation device. Bergstrom et al. (1994) recommended using a 35 mL syringe and 19-gauge needle to create 8 psi (pounds per square inch) irrigation pressure stream. Use of a bulb syringe, which provides a pressure below 4 psi, is generally not an effective irrigation device. Battery-powered, disposable irrigation devices can provide pulsatile lavage to loosen wound debris while removing it by suction (Rodeheaver, 1999). In another trial Ho et al. (2012) found that low pressure pulsatile lavage was effective in the enhancement of pressure ulcer healing rate in persons with SCI.

Gentle scrubbing can be performed with moistened gauze. Caution should be exercised if using a gauze sponge or brush because these may inflict tissue trauma. When using these devices, cleansing should proceed from centrally to periphery to avoid contamination (Barr, 1995).

Cleansing with ultrasonic mist also has been described (Fernandez et al., 2007). In addition, bathing, showering, and washing the affected area can be effective in removing wound debris.

In addition to the pressure of cleansing delivery methods, variables such as efficiency, cost, time, potential for blood and body fluid exposures, and caregiver/patient satisfaction should be considered (Weller, 1991).

A significant amount of skin debris exists in the periwound skin more than in normal skin. This debris consists of protein, lipids, and water-soluble substances. In addition, staph aureus, beta hemolytic streptococcus, and pseudomonas are among the predominant microorganisms present. It is critical to clean the periwound skin once every 24 hours and with dressing changes in order to reduce the periwound debris and avoid critical wound bed contamination (Konya et al., 2005).

DEBRIDEMENT

15. Debride devitalized tissue using a method or a combination of debridement methods appropriate to the ulcer's status.

- Debride eschar and devitalized tissue with the exception of a stable heel eschar.
- Debride areas in which there is unstable eschar and devitalized tissue.

(Scientific evidence—II, III, IV, V; Grade of recommendation—B; Strength of panel opinion—Strong)

Debridement is defined as removal of necrotic or infected tissue. Debridement can be performed in the clinic, at the bedside or in the operating room. Debridement allows the true extent of the ulcer to be determined. Debridement can suppress biofilm reformation by disrupting the biofilm's ability to reconstitute itself and reattach to its host to become metabolically active (Wolcott et al., 2009). It results in a decrease in the bacterial concentration in the wound and may decrease the risk of infection, which could cause cellulitis or sepsis (Witkowski and Parish, 1992; Yarkony, 1994). While rarely reported, necrotizing soft tissue infection from an existing pressure ulcer can occur as a serious complication in the presence of multiple comorbidities, for which rapid diagnosis of soft tissue infection and aggressive debridement are the mainstays of treatment.

Autolytic debridement

Autolytic debridement typically takes place under occlusive dressings. This type of debridement is the function of one's own white

blood cells and proteolytic, fibrinolytic, and collagenolytic enzymes that enter the wound to digest the necrotic tissue present in the wound. It is most active in a moist wound. Adequate leukocyte function and number are essential. If wound exudate is left undisturbed, neutrophils and macrophages continue their phagocytic work and the production of growth factors in the wound exudate continues. If autolytic debridement is used as a sole form of debridement, it should be used only in noninfected wounds. It should not be used in the presence of neutropenia (neutrophil count of $<500 \text{ mm}^3$) and/or wounds with advancing cellulitis. Diminished neutrophils can result in increased bacteria in the wound bed. In the case of infection caused by anaerobic bacteria, the presence of fluid in the wound bed and the use of an occlusive dressing will increase the growth of anaerobic bacteria.

Mechanical debridement

Mechanical debridement is most commonly performed with the application of a wet dressing to a wound, which is subsequently removed when dry, thus the aptly named wet-to-dry dressing. It can be painful for those who are sensate in the area of their ulcers. It is a nonselective form of debridement with the potential of removing healthy granulation and epithelial tissue when the dressing is removed along with necrotic tissue. Other forms of mechanical debridement, such as low-pressure pulsatile lavage, and intermittent irrigation using a syringe can also be effective in clearing the wound bed of debris.

A hydrosurgery system of debridement, similar to high-pressure pulsatile lavage, enables the precise removal of necrotic tissue. This system projects a high-velocity waterjet across the operating window into an evacuation collector. The suction permits the surgeon to hold and cut targeted tissue while at the same time aspirating debris from the wound. In a case series, (Gurunluoglu 2007) found that this type of debridement might be a useful alternative for soft tissue debridement in preparing wounds for reconstructive surgery, although more studies are needed to support its efficacy and cost-effectiveness.

Enzymatic debridement

Enzymatic debridement works by the topical application of commercially produced biologic enzymatic agents that digest the components of slough and dissolve the collagen that anchors necrotic tissue to the wound bed, and spares the non-necrotic tissues. Enzymatic debridement is often helpful in wounds where the necrotic tissue

is so adherent to the wound base that it is difficult to be safely removed by mechanical or sharp debridement. However, the debridement would take more time to be achieved. It is safe to use in wounds with high bacterial loads.

Sharp debridement

Sharp debridement utilizes a scissors or scalpel or curette to cut or scrape away necrotic tissue and biofilm from the wound bed. Bleeding and injury to viable tissue are the main risks. This is the more rapid way of selectively removing necrotic tissue and biofilm. One exception to the recommendation for sharp debridement of necrotic tissue and eschar is when stable eschar is present over the heel. These stable heel ulcers with eschar should not be debrided (Black et al. 2007). However, the presence of edema, erythema, fluctuance or drainage would indicate eschar debridement is necessary.

Surgical debridement

Surgical debridement is the most efficient method of debridement for removing large amounts of tissue in a single session. It is performed as an operative procedure and may involve excising the skin over the area of undermining to allow for debridement of underlying necrotic tissue from infected wounds and areas of fibrosis, removal of callus from wound edges, removal of all grossly infected tissue, and obtaining a biopsy of the deep tissue after debridement of all nonviable or infected tissue for culture and pathology to determine the presence of infection, fibrosis, and granulation tissue. Debridement may be performed using a scalpel, electrocautery, rongeur, or curette for debridement of bone. Because there may be associated pain and/or bleeding, as well as autonomic dysreflexia, anesthesia is often used. Bleeding, the need for anesthesia and its associated risks, and possible injury to nervous or other viable tissue are the main disadvantages. It is indicated in cases of advancing cellulitis with sepsis, immunocompromised individuals, and in cases of life-threatening infections. Surgical debridement of necrotic ulcers associated with sepsis can rapidly eliminate the source of infection (Galpin et al., 1976). Using surgical debridement, the surgeon makes a wide excision of the ulcer in preparation for closure. Serial debridement is required for wounds with heavy bioburden; maintenance debridement performed within 24 to 72 hours of initial debridement is recommended to control bacterial bioburden.

Maggot debridement

Maggot debridement therapy (MDT) or the use of maggots for debridement of wounds has been approved in the United States by the Food and Drug Administration as a medical device. Maggots are precise in their debridement. The larvae excrete through their oral, cutaneous, and fecal matter, proteolytic enzymes, including collagenase, that break down necrotic tissue. Maggots also ingest bacteria along with liquefied necrotic tissue subsequently killing them in their digestive tract. They inhibit the pro-inflammatory responses of human monocytes (van der Plas et al., 2009). Medicinal maggots also stimulate healing, and inhibit and eradicate biofilm (Sherman et al., 2009). MDT has been shown to be significantly faster than conventional treatment in debriding wounds (not pressure ulcers) during the first week of treatment (Opletalova et al., 2012) and faster than hydrogel or autolytic debridement (Dumville et al., 2009). Sherman et al. (2007) reported the successful use of MDT in two SCI patients with wounds unresponsive to conventional therapy, and where surgical debridement was considered too risky.

Choice of a method of debridement is based on the individual's clinical situation. Techniques can often be combined. Although it is beneficial to remove devitalized tissue as quickly as possible, the clinical circumstances will determine the most appropriate method. All methods of debridement should be discontinued when the necrotic tissue and/or biofilm has been removed.

SELECTION OF WOUND CARE DRESSING (TABLE 3)

16. Use a dressing that achieves a physiologic local wound environment that maintains an appropriate level of moisture in the wound bed:

- Control exudate
- Eliminate dead space
- Control odor
- Eliminate or minimize pain
- Protect the wound and the periwound skin
- Remove nonviable tissue
- Prevent and manage infection

(Scientific evidence—I, II; Grade of recommendation—A; Strength of panel opinion—Strong)

Dressings are topical products used for protection of a pressure ulcer from contamination and trauma, application of medication, debridement of necrotic tissue, and optimally provide a physiologic local wound environment. A physiologic wound environment is a local

environment in which tissue hydration levels and the viability of the wound tissue and various cells within the wound space (growth factors, platelets, etc.) are maintained by something other than the skin. The wound dressing can be viewed as the substitute skin.

Dressing selection should be based upon a thorough wound assessment and history, dressing interactions, patient and caregiver needs, as well as cost (Baranoski, 1995; Krasner, 1997). It should be noted, however, that caregiver time and the associated labor costs required for wound care significantly impact the overall cost of caring for individuals with pressure ulcers and may exceed the cost of wound management supplies (Bolton et al., 1997). Therefore a dressing that may cost more on a daily basis but does not need frequent dressing changes may be the more cost-effective one in the long run. Ultimately, the attributes of the dressing should match the needs of the wound.

Control exudate

Exudate can be attributed to bacterial colonization and increased bioburden and therefore can impair wound healing; thus, it needs to be minimized. Although dressings should keep the ulcer bed moist, they should not cause over hydration leading to maceration of the surrounding intact skin. Excessive exudate causing macerated surrounding tissue is associated with prolonged healing time (Xakellis and Chrischilles, 1992). Excessive exudate can be managed by using an absorptive wound dressing designed to control exudate and avoid periulcer maceration. Exudate should be absorbed away from the ulcer bed (Bergstrom et al., 1994). A number of techniques are used to protect the surrounding intact skin from excessive moisture, including applying moisture barrier creams, skin barriers, or skin sealants. Changing the dressing if excessive drainage is observed, keeping the dressing in the wound bed and not on the intact skin, and using a rectal pouch if fecal contamination is anticipated, are other techniques used to protect the periulcer skin.

Sayag et al. (1996) compared an alginate wound dressing to treatment with a dextranomer paste. Reduction in wound size and the rate of healing were better with the alginate group than with the dextranomer paste. In an open-label, randomized, parallel group study, investigators compared the effects of a newly formulated dextranomer paste with saline-soaked dressings. Significantly greater improvement in ulcer drainage (25%) was found with dextranomer paste compared to saline treatment (73% versus 13% of treated ulcers) (Ljungberg, 1998).

Table 3. Selection of Wound Care Dressing

Dressing Category and Definition	Indications	Advantages	Disadvantages	Considerations, Usage, and Precautions
PASSIVE WOUND CARE PRODUCTS/THERAPIES				
<p>Alginates Non-woven spun fibers of brown seaweed used as a non-occlusive primary dressing. They are shaped as ropes or pads that conform to the shape of wound. They have a fluffy cottonlike appearance.</p>	<ul style="list-style-type: none"> • Primary Dressing for prophylaxis on high-risk intact skin • Secondary dressing to other products such as foam, or alginates • Superficial wounds with minimal or no exudate • Eschar covered wounds when autolysis is indicated often used as secondary dressing to alginates and foam 	<ul style="list-style-type: none"> • Absorbent up to 20 times its weight • Fills dead space • Easy to use • Supports debridement in the presence of exudate 	<ul style="list-style-type: none"> • Requires a secondary dressing • Not recommended for dry wounds or eschar covered wound • Not recommended for deep undermined ulcers in which wound edges are at risk of collapsing • Can desiccate the wound bed • Not indicated for patients with known sensitivity to alginates • Not recommended for wounds in which the dressing is not easily retrievable as it may break into pieces and be a nidus of infection if retained 	<ul style="list-style-type: none"> • Not recommended in non-draining wounds • Loosely pack into a wound • May be layered into a deep wound • Requires a secondary dressing to secure – may use gauze or transparent film as secondary dressing
<p>Transparent Film Transparent polyurethane sheets coated on one side with an acrylic hypoallergenic adhesive film that is impermeable to fluids and bacteria.</p>	<ul style="list-style-type: none"> • Primary Dressing for prophylaxis on high-risk intact skin • Secondary dressing to other products such as foam, or alginates • Superficial wounds with minimal or no exudate • Eschar covered wounds when autolysis is indicated often used as secondary dressing to alginates and foam 	<ul style="list-style-type: none"> • Visual evaluation of wound without removal • Impermeable to external fluids and bacteria • Transparent and comfortable • Promotes autolytic debridement • Minimizes friction 	<ul style="list-style-type: none"> • Nonabsorbent • Difficult to apply • Channeling and wrinkling occurs • May dislodge in high-friction areas • Not to be used on wounds with moderate to heavy drainage because they do not absorb • Not to be used on wounds with fragile surrounding skin or infected wound – cause skin stripping 	<ul style="list-style-type: none"> • Should not be used in infected wounds that require frequent monitoring • Allow 4-5 cm overlap from wound margin to the surrounding skin. • May leave undisturbed up to 7 days.
<p>Hydrocolloid An occlusive or semiocclusive dressing composed of materials such as gelatin, pectin, and carboxymethylcellulose. They provide a moist wound bed forming a gelatinous mass over the wound bed that allows clean wounds to granulate and necrotic wounds to debride autolytically. Available in paste form that can be used as filler for shallow cavity wounds. Available in variety of shapes, widths, sizes, contours, and thickness.</p>	<ul style="list-style-type: none"> • Primary or secondary dressing • Partial- and full-thickness wounds • Necrotic wounds or wounds with slough • Minimal – moderate exudates • May be used in combination with other dressing materials (e.g., pastes, alginates) 	<ul style="list-style-type: none"> • Impermeable to bacteria and other contaminants • Waterproof • Easy to apply and time-saving • Thin forms diminish friction and minimizes “rolling” or “curling up” • Conformable, self-adherent, and absorptive • Facilitates autolytic debridement • May be used in combination with compression for venous ulcers 	<ul style="list-style-type: none"> • Not recommended for use in wounds with heavy exudate, depth or friable periwound skin, wounds with exposed tendon or bone • Its occlusive property limits gas exchange between the wound and the environment • May curl at the edges and could injure fragile skin upon removal • May contribute to hypergranulation tissue 	<ul style="list-style-type: none"> • Select a dressing with a minimum of 2-3 cm overlap from the margin of the wound • May be cut to conform to difficult areas • Change dressings up to 3 times a week

Table 3. Selection of Wound Care Dressing

Dressing Category and Definition	Indications	Advantages	Disadvantages	Considerations, Usage, and Precautions
PASSIVE WOUND CARE PRODUCTS/THERAPIES				
<p>Hydrogel Water- or glycerin-based amorphous gels delivered from a tube or impregnated into strip packing materials, gauzes, or sheet dressings. It donates moisture / fluid into the wound. Absorptive capacity varies.</p>	<ul style="list-style-type: none"> • Primary dressing (amorphous and impregnated gauzes) or as primary or secondary dressings (sheets) • Partial- and full-thickness wounds • Deep wounds • Dry to minimal exudate • Used in combination with other dressing materials 	<ul style="list-style-type: none"> • Promotes autolytic debridement • Conformable to wound space • Fills in dead space • Rehydrates the wound bed • Soothing and reduces pain • Amorphous form can be used when infection is present 	<ul style="list-style-type: none"> • Not recommended for wounds with heavy exudate • Dehydrates easily if not covered • Requires secondary dressing • May be difficult to secure • May cause maceration of periwound skin or candidiasis from inappropriate usage 	<ul style="list-style-type: none"> • Sheets without adhesive border or wound fillers are changed up to once per day • Apply skin protective wipe to periwound skin to prevent maceration • Sheets with adhesive covers are changed up to 3 times per week
<p>Foams Semi-permeable hydrophilic foam. Available in varying thicknesses, absorptive capacity, and adhesive properties. Available in pads, sheets, wafers, rolls, pillows, surfactant impregnated or with an odor absorbent charcoal layer.</p>	<ul style="list-style-type: none"> • Primary dressing for absorption and insulation • Secondary dressing for wounds with packing and provide additional absorption and to absorb drainage around tubes • Partial- and full-thickness wounds • Minimal to heavy exudate • Infected wounds • Used in combination with other dressing materials such as films, alginates, pastes, and powders 	<ul style="list-style-type: none"> • Non adherent and protects periwound skin • Conformable to body contours • Insulates wounds and provides padding • Repels contaminants • Easy to apply and remove 	<ul style="list-style-type: none"> • Not recommended for desiccated wounds or those with sinuses unless packing is added • Cavity dressing pillows should not be cut • Nonadherent foam requires secondary dressing, tape, or net to hold in place • Poor conformability to deep wounds 	<ul style="list-style-type: none"> • Select a dressing approximately 2-3 cm larger than the wound • Dressing change may be up to 3 times per week • Usual dressing change for foam wound fillers is up to once per day
<p>Gauze Woven or non-woven material that may include cotton, rayon, and / or polyester. Available sterile or non-sterile. May be used wet, moist, dry, or impregnated with petrolatum, antiseptics, or other agents (see below). Comes in varying weaves with different size interstices.</p>	<ul style="list-style-type: none"> • Partial- and full-thickness wounds • Infected wounds and those with combination exudate or necrotic tissue • Exudative wounds • Wounds with cavities or dead space, tunneling, or sinus tracts 	<ul style="list-style-type: none"> • Loosely filling large wounds • May use in combination with other wound products such as hydrogels 	<ul style="list-style-type: none"> • Adheres to wound tissue for nonselective debridement • May lint or shred if cut • Labor intensive approach • Wound may desiccate 	<ul style="list-style-type: none"> • Fluff gauze and avoid pressure or tight packing • Monitor for desiccation or saturation • Dressing interval depends upon level of saturation
<p>Impregnated Gauze Woven sponges that are impregnated with chemical compounds and agents (hypertonic or NS, petrolatum, zinc, iodoform) to deliver antimicrobial, medication, nutrients and moisture</p>	<ul style="list-style-type: none"> • Partial- and full-thickness wounds • Infected wounds and those with combination exudate or necrotic tissue • Exudative wounds • Wounds with cavities or dead space, tunneling, or sinus tracts 	<ul style="list-style-type: none"> • Dressing dependent: Petrolatum makes gauze nonadherent; hypertonic dry sponges provide absorption, and antimicrobials decrease bioburden 	<ul style="list-style-type: none"> • Dressing dependent 	<ul style="list-style-type: none"> • Loose packing • Monitor for exudate to avoid maceration • Choose appropriate size and ingredients for dressing

Table 3. Selection of Wound Care Dressing

Dressing Category and Definition	Indications	Advantages	Disadvantages	Considerations, Usage, and Precautions
PASSIVE WOUND CARE PRODUCTS/THERAPIES				
<p>Composites Composed of impermeable barrier, an absorptive layer other than an alginate, foam, hydrocolloid or hydrogel, a semi-adherent or nonadherent property for covering the wound and an adhesive border.</p>	<ul style="list-style-type: none"> • Primary or secondary dressing • Partial- and shallow full-thickness wounds • Minimal exudate (dressing dependent and when used in combination with another dressing such as an alginate) • Healthy granulation tissue, or necrotic tissue (slough or moist eschar), or mixed wounds with granular and necrotic base 	<ul style="list-style-type: none"> • Easy to use – application and removal • Combines the advantages from more than one dressing group to address the characteristics of the wound • Molds well • May be used on infected wounds • Includes an adhesive border 	<ul style="list-style-type: none"> • Requires a border of intact skin for anchoring the dressing • Dependent upon the type of composite dressing. Read the package labeling for specific information, and proper use based on wound bed characteristics 	<ul style="list-style-type: none"> • A paper-backing liner is removed and the dressing is applied to the wound • Usual composite dressing change is up to 3 times per week, one wound cover per dressing change
<p>Contact Layers A single layer, nonadherent, woven polyamide net that is placed in contact with the wound base. It allows the passage of wound exudate to a secondary dressing, usually a gauze or foam dressing.</p>	<ul style="list-style-type: none"> • Primary dressing for partial- and full-thickness wounds • Wounds with minimal, moderate, and heavy exudate • In combination with negative pressure wound therapy 	<ul style="list-style-type: none"> • Protects wound base from trauma during dressing changes • For use with large or deep wounds • Antimicrobials may be applied under the dressing 	<ul style="list-style-type: none"> • Not recommended for use in shallow or dry wounds in the presence of viscous exudate • Not recommended for wounds covered with eschar • Requires a secondary dressing 	<ul style="list-style-type: none"> • Applied to a wound base with a secondary absorbent dressing cover (e.g., gauze or foam) • Contact layer stays in place up to 7 days while the absorbent layers are changed as needed
<p>Wound Fillers Absorbent materials composed of starch copolymers in paste, powder or bead form. It requires a secondary dressing.</p>	<ul style="list-style-type: none"> • Partial and shallow full-thickness wounds • Minimal to moderate exudate • Necrotic wounds with moisture • Infected wounds 	<ul style="list-style-type: none"> • Absorbent filling material • May be combined with other dressings to extend wear time 	<ul style="list-style-type: none"> • Not recommended for dry wounds, wounds covered with eschar, deep wounds, or those with tunneling 	<ul style="list-style-type: none"> • Apply to fill a shallow defect in a wound • Apply a secondary dressing • Change dressing up to once per day
<p>Wound Pouches Adapted from ostomy care with an integrated skin barrier and a drainage spout that can be connected to a straight drainage. Odor proof pouch film</p>	<ul style="list-style-type: none"> • Highly exudative wound • Malodorous exudate 	<ul style="list-style-type: none"> • Pouch wear time 4-7 days • Skin protection from wafer • Ease of use 	<ul style="list-style-type: none"> • Pouching system is expensive and requires extensive education of caregiver to minimize application errors 	<ul style="list-style-type: none"> • Apply in similar fashion as an ostomy pouch • Educate caregiver • Dressing change is up to 3 times per week

Table 3. Selection of Wound Care Dressing

Dressing Category and Definition	Indications	Advantages	Disadvantages	Considerations, Usage, and Precautions
ACTIVE WOUND CARE PRODUCTS/THERAPIES				
<p>Antimicrobial Topical wound care product derived from agents such as silver, iodine, and polyhexethylene biguanide. Delivers antimicrobial or antibacterial action to the wound (e.g., silver dressing or iodine). Available as ointments, impregnated gauzes, pads, island dressings, and gels.</p>	<ul style="list-style-type: none"> • Primary Dressing for effective barrier to bacterial penetration • Primary or secondary dressing • Provides sustained release of antiseptic agent at the wound surface for long-lasting antimicrobial action • Acute and chronic wounds • To reduce infection and manage wounds with moderate to heavy exudates in partial- and full-thickness wounds 	<ul style="list-style-type: none"> • Provides broad range of antimicrobial or antibacterial activity • Reduces and prevents infection • Alters metalloproteinases (MMP) within wounds 	<ul style="list-style-type: none"> • Not indicated to control heavy bleeding • Not indicated for patients with known sensitivity to silver • Not indicated for use on patients during MRI examination • Incompatible with oil-based products such as petrolatum • May cause staining on wound and intact skin with silver dressings 	<ul style="list-style-type: none"> • Must be removed before start of long-term radiation treatment with x-rays, ultrasound, diathermy, or microwaves • When using silver or iodine dressings, do not apply or combine with collagenase enzymatic debrider because silver and iodine will inhibit the enzymatic activity
<p>Collagen Derived from bovine, porcine, or avian sources. Non adherent pouches or vials, gels loaded into syringes, pads, powders, and freeze-dried sheets. Requires a secondary dressing.</p>	<ul style="list-style-type: none"> • Primary dressings for partial- and full-thickness wounds • Contaminated and Infected wounds, tunneling wounds • Minimal to moderate exudate 	<ul style="list-style-type: none"> • Accelerates wound repair • Nonadherent to wound • Some forms may be left in wound for 7 days • May be used in combination with topical agents • Conforms well to wound bed • Easy to apply and remove 	<ul style="list-style-type: none"> • Not recommended for patients with sensitivity to bovine materials • Requires a secondary dressing 	<ul style="list-style-type: none"> • Refer to package insert. Each form of collagen dressing has specific usage instructions
<p>Enzymatic Debriding Agents Proteolytic enzymes, fibrinolytic enzymes, collagenase applied to wound to digest necrotic tissue.</p>	<ul style="list-style-type: none"> • Partial – and full-thickness wounds • Eschar or necrotic tissue in wound bed 	<ul style="list-style-type: none"> • Conservative debriding agent • Easy to understand • May be used in many care settings 	<ul style="list-style-type: none"> • Use with caution in patients with coagulation disorders 	<ul style="list-style-type: none"> • Use gauze as a secondary dressing • When granulation tissue is present, may discontinue product use unless maintenance debridement is planned
<p>Negative Pressure Wound Therapy Open cell reticulated foam or gauze dressing placed into the wound, sealed with semi-occlusive drape and attached to subatmospheric pressure through an evacuation tube connected to a computerized pump device.</p>	<ul style="list-style-type: none"> • Acute and chronic open wounds with depth • Partial- and full-thickness wounds 	<ul style="list-style-type: none"> • Control for fluid and isolation of the wound is provided while dressing is in place • Suction provides wound contraction to promote wound closure 	<ul style="list-style-type: none"> • Use with caution in patients with active bleeding, clotting disorders, and those on anticoagulants 	<ul style="list-style-type: none"> • Follow strict protocol • Dressing changes varies based on wound usually every 24 up to 72 hours

Eliminate dead space

Wound cavities should be loosely filled. In order to promote debridement closure and healing, the sinus tracts or tunnels, if they are large enough, can be filled with a single piece of moistened gauze or ribbon. The material filling the wound cavity should be easily retrieved. Filling materials that can break up or separate should not be used as they can break and inadvertently left in the tunnel, making them a foreign body and the source of infection that will prevent healing and closure. Enough dressing material should be used to ensure that the dressing is in contact with the wound bed, but not so tightly as to prevent effective granulation of the wound base. Tissue damage may result if wounds are filled too tightly, causing increased pressure on the tissue in the wound bed (Bergstrom et al., 1994). If there is a risk of loss of dressing material within a cavity, a single piece of dressing material should be used with one end easily accessible for removal at the next dressing change.

Control odor

Odor from a pressure ulcer may be a sign of infection. Monitoring the wound for infection and using the correct dressing for the specific type of wound will help combat odor from the pressure ulcer.

Protect the wound and the periwound skin

Individuals with SCI have fragile skin, especially if they have had prior pressure ulcers. It is important to consider the quality of the dressing adhesive when the skin surrounding the ulcer is fragile. Dressings with low adhesive or no adhesive may be selected when the goal is not to place an adhesive in contact with the wound margins or surrounding skin. The term "epidermal stripping" refers to the removal of the epidermis by mechanical means (Wysocki and Bryant, 1992). Epidermal stripping can be prevented by recognition of fragile skin, appropriate application and tape, avoidance of unnecessary tape, and the use of skin sealant or solid wafer skin barriers under the adhesive (Wysocki and Bryant, 1992). Silicone-based dressings may decrease the risk of skin stripping caused by adhesive products. Wound dressings have evolved to address the issue of epidermal stripping. A silicone bordered dressing differs from adhesive bordered dressing. Silicone is applied to and will only adhere to clean, dry, and intact skin. Its use allows the health-care provider to peel or lift the border to view the wound bed. If the periwound skin is denuded, it would be best to apply a hydrocolloid to the

periwound skin in order to promote healing and any adhesive sheet, tape, etc. should be secured directly to the hydrocolloid.

No scientific studies address the use of rectal pouches or fecal management systems to contain the stool and prevent fecal contamination of the dressings and the wound. However, in clinical practice, rectal pouches and fecal management systems are used at times to contain the liquid stool and to prevent fecal contamination of the dressing and the wound. When using these systems, extra care must be provided to avoid the development of recto-anal ulceration or autonomic dysreflexia caused by pressure from the device.

Dressing Selection

Numerous dressing products have been marketed in the last decades. Currently, some of the dressing categories include transparent films, hydrocolloids, hydrogels, foams, alginates, or gauze dressings. A summary of these products and their indications, advantages, disadvantages, and considerations for use is presented in Table 1. In general, the clinical trials of these products were conducted on individuals in nursing homes or hospitals and not on individuals with SCI. It should be noted that a choice of a dressing should be dynamic in keeping with changing condition of the wound. Some circumstances that may indicate the need for a dressing change include discomfort or the presence of pain; change in the extent of edema, erythema, or skin temperature; seepage of exudate through the dressing; or strong odor from the dressing (Krasner, 1997).

Most wound dressings today are semi-occlusive rather than occlusive. Semi-occlusive dressings are designed to protect the wound and periwound skin from microbial and physical insult. They provide thermal insulation, odor control, compression, and deliver antimicrobial agents. Moisture retentive dressings consistently retain moisture at the wound site by interfering with the natural evaporative loss of moisture vapor. Please see Table 1 for a detailed description of currently used dressings.

ELECTRICAL STIMULATION**17. Use electrical stimulation (ES) to promote closure of category/stage III or IV pressure ulcers, unless contraindicated in the cases of untreated, underlying osteomyelitis or infection.**

(Scientific evidence—I, II, III, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Electrical stimulation to promote wound closure

Electrical stimulation may facilitate pressure ulcer healing by multiple mechanisms: maintenance of appropriate transepithelial potential in the non-intact skin, antibacterial effects, as well as promotion of angiogenesis through vascular endothelial growth factor (VEGF), promotion of granulation and re-epithelialization.

Houghton et al. (2010) reported the results of a single blind parallel group randomized control study comparing community based standard wound care (SWC) to SWC plus 3 months of high voltage pulsed ES applied to the ulcer wound bed an average of 3 hours per day for three months in thirty-four persons with SCI and category/stage II to IV pressure ulcers. Those in the SWC plus ES group had a decrease in wound surface area of 70% as compared to the SWC group, which had a decrease of 36%. In another small randomized sample (n=7) of persons with SCI and category/stage IV pressure ulcers, investigators reported that interrupted direct current stimulation accelerated healing of pressure ulcers when used in conjunction with routine nursing care (Adegoke and Bardmos, 2001). Data from three randomized, controlled clinical trials involving more than 250 individuals with SCI, each with at least one wound, supported the efficacy of ES by accelerating the healing rate of pressure ulcers that had not responded favorably to standard wound care (Baker et al., 1996; Griffin et al., 1991; Stefanovska et al., 1993). Other controlled trials also demonstrated significantly better healing rates for wounds treated with ES compared with control wounds ((Stefanovska et al. (1993), Baker et al. (1996)). A double-blind multicenter study, in which ES below sensory perception was used to treat pressure ulcers, demonstrated that more than 50% of wounds healed in 8 weeks, whereas only 3% of ulcers in the control group healed and most other control wounds increased in size (Wood et al., 1993).

Electrical stimulation to promote muscle bulk and tissue health

When continuous ES and intermittent ES to the bilateral gluteal muscles are compared, both treatments are found to reduce pressure around the ischial bony prominences and provide significant sustained increases in tissue oxygenation (Gyawali et al., 2011).

There is sufficient evidence supporting only the efficacy of ES for a recommendation to be made. Literature reviews were done for several adjunctive wound therapies. These include negative pressure wound therapy, ultrasound, laser therapy, skin substitutes, growth factors, and autologous platelet rich plasma

Negative Pressure Wound Therapy

Negative pressure wound therapy (NPWT) is a mechanical wound care treatment that uses controlled negative pressure to accelerate wound healing by evacuating wound exudate, stimulating granulation tissue formation, reducing the wound bacterial bioburden, increasing blood flow in the wound and adjacent tissue, and maintaining a moist wound environment Morykwas et al. (1997). Intermittent negative pressure application when compared to continuous negative pressure application has been shown to increase blood flow as well as improve wound contraction and granulation formation to a greater degree (Malmsjo et al. (2012); Lindstedt et al. (2010).

Mullner et al. (1997) evaluated the efficacy of NPWT on the healing of pressure ulcers, acute traumatic wounds, and infected soft tissue wounds in 45 individuals. Seventeen of the 45 individuals with infected sacral pressure ulcers, including one with SCI, were treated with NPWT for 4 weeks. Of these, one ulcer achieved primary closure, eight ulcers granulated and were closed secondarily by grafting, and three ulcers decreased in size by 80%. Argenta and Morykwas (1997) reported on a case series of 141 category/stage III and IV pressure ulcers using variable treatment durations in which 32% of the wounds closed completely in 2 to 16 weeks; 46% decreased in size more than 80% and were subsequently treated with either skin grafts, muscle flaps, primary closure, or dressing changes, while another 15% decreased in size from 50% to 80% and were either grafted or flapped. Published results demonstrating effectiveness of the treatment of pressure ulcers in persons with SCI with NPWT is not robust. If nutritional status is poor in persons with SCI and pressure ulcers, Ho et al. (2010) reported that NPWT is not effective.

NPWT has risks. In 2011 the FDA issued a death and injury report of 6 deaths and 97 injuries for a total of 12 deaths and 174 injuries reports since 2007 with NPWT devices. The most serious complication was bleeding which occurred in patients with blood vessel grafts in the leg, breastbone and groin wounds, and patients being managed with anticoagulation. In addition, wound infections occurred in more than half of the cases and were related to the retention of dressing pieces in the wounds, resulting in a delayed recovery and requiring wound exploration, surgical removal of dead tissue, and drainage. Additionally, Citak et al. (2010) reported a rare complication of necrotizing fasciitis during and after NPWT for category/stage IV pressure ulcer in an individual with paraplegia. Contraindications for NPWT include necrotic tissue with eschar present, untreated osteomyelitis, non-enteric and unexplored fistulas, malignancy in the wound, exposed vasculature, exposed nerves, exposed anastomotic site, and exposed organs.

ADJUNCTIVE THERAPIES AND BIOLOGICS

There are a number of interventions that are in use in some facilities but that do not have strong supporting scientific evidence. However, we would be remiss if we did not acknowledge that they are in use clinically. The mention of these products is to inform the reader/clinician/researcher that these are being used but not to the extent that they could be recommended because the evidence is not strong.

Ultrasound Therapy

Low-frequency, low-intensity ultrasound (as opposed to high frequency ultrasound, which is used in imaging and musculoskeletal therapy) delivers energy through mechanical vibrations in the form of sound waves to cause cavitation and streaming. Mechanical wound treatment with low frequency ultrasound disrupts bacterial biofilm and mechanically removes necrotic tissue.

In a case series involving 5 patients with sacral pressure ulcers and compromised mobility (SCI, ventilator/mobility dependent or persistent vegetative state), low frequency low intensity ultrasound therapy resulted in 100% granulation and decreased wound size in four of the patients (Schmuckler, 2008). It has been shown to rapidly debride unstageable pressure ulcers (Medrano and Beneke, 2008).

Laser Therapy

Lasers have been in use since the 1960s without solid evidence for effectiveness. Taly et al. (2004), in a randomized double-blind controlled trial, treated 35 subjects with category/stage III and IV pressure ulcers with multiwavelength light therapy. The intervention did not influence overall healing of the pressure ulcers.

Skin Substitutes

Skin substitute biomaterials are commonly referred to by terms that include bioengineered skin equivalents, tissue-engineered skin, tissue-engineered skin constructs, biological skin substitutes, bioengineered skin substitutes, skin substitute bioconstructs, living skin replacements, and bioengineered alternative tissue. Intended to mimic the histological structure of normal skin or the properties of the extracellular matrix, their key role is to deliver growth factors, provide extracellular matrix proteins, and to attract differentiated cells (e.g., fibroblasts, endothelial cells) or stem cells to the wound. They accelerate and augment the intrinsic healing process in the wound and support wound bed preparation.

Two clinical studies have shown the effects of cultured skin equivalents on closure of pressure ulcers. Phillips and Pachas (1994) applied autologous cultured keratinocyte grafts to 17 pressure ulcers on seven individuals and found that 65% of the ulcers closed completely after an average of two graft applications. Yamashita et al. (1999) evaluated granulation tissue formation and epithelialization following application of an allogeneic cultured dermal substitute to five cases of category/stage III or IV pressure ulcers. They observed that granulation tissue developed early and that epithelialization was complete by 7 weeks in all the individuals.

Growth Factors

Two clinical studies have examined the effect of recombinant platelet-derived growth factor (PDGF) on the healing of pressure ulcers. In a randomized controlled trial, Robson et al. (1992a) treated the pressure ulcers of 20 individuals with different concentrations of PDGF or placebo. After 28 days, only ulcers treated with 100 $\mu\text{g}/\text{mL}$ of PDGF decreased their volume to a mean of 6% of their original volume as compared with placebo-treated ulcers that decreased to 22% of their original volume. In a follow-up, multicenter, randomized double blind study, Mustoe et al. (1994) evaluated the effects of two aqueous concentrations of PDGF and an aqueous placebo on category/stage III and IV pressure ulcers in 45 individuals. After 28 days ulcers treated with 300 $\mu\text{g}/\text{mL}$ of PDGF decreased to 40% of their

original volume. Ulcers treated with 100 $\mu\text{g/mL}$ of PDGF had a mean ulcer volume reduction of 71%, and placebo-treated ulcer volume only decreased a mean of 17%.

Autologous Platelet-Rich Plasma

Blood platelets adhere, aggregate, and release numerous growth factors, adhesion molecules, and lipids that regulate the migration, proliferation, and functions of keratinocytes, fibroblasts, and endothelial cells. Platelet-rich plasma contains is an autologous preparation of platelets in concentrated plasma, rich in growth factors that promote tissue regeneration. Although there is some limited evidence that platelet-rich plasma may be effective, the evidence is not strong enough to recommend its use as a therapy.

Modification of Treatment Plan

18. Modify the treatment plan if the ulcer shows no evidence of healing within 2 to 4 weeks. Review individual factors associated with non-healing of pressure ulcers, such as the following:

- Incontinence
- Infection
- Carcinoma
- Abnormal wound healing
- Nutrition
- Medication
- Support surfaces
- Transfer
- Noncompliance

(Scientific evidence—I, III, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Pressure ulcers receiving effective/adequate treatment should show signs of healing within 2 to 4 weeks. Inadequate healing should prompt a reassessment of the treatment plan, compliance with treatment recommendations, and other sources of treatment failure (Bergstrom et al., 1994).

INCONTINENCE

Urine and stool contamination of pressure ulcers interferes with their healing (Wilczweski et al., 2012). If there is urinary incontinence, a reevaluation of the bladder management program must be performed and use of an external catheter, indwelling catheter, more frequent intermittent

catheterization, or change in medication should be entertained. If there is bowel incontinence, a reevaluation of the bowel program must be performed. Causes of incontinence can include bowel impaction, infection, inadequate evacuation, and loose stool due to colonic denervation. Persistent incontinence of stool that is unable to be controlled and which contaminates a non-healing pressure ulcer is a strong indication for a colostomy.

INFECTION

Most chronic pressure ulcers become colonized with bacteria. When the number of bacteria reaches a critical threshold on the wound bed, they are thought to inhibit wound healing and damage wound tissues. This can be considered local infection. At this point it is thought that biofilm is present which harbors bacterial colonies within the wound. The signs of infection may be subtle manifesting as only a lack of healing progress or with increased wound drainage, epithelial bridging, malodor, a color change of the wound bed, or friable granulation tissue (Leaper 2012).

To treat local infection, clinicians need to control the bacteria burden and the biofilm must be removed (see recommendation 14). Cleansing the wound adequately is the mainstay of treatment; however, topical antibiotics and antiseptics such as silver sulfadiazine cream, honey, polyhexamethylene biguanide, cadexomer iodine are often useful adjunct treatments if progress in healing is not being made (Kucan et al., 1981; Leaper DJ 2012). Mupirocin calcium cream 2% may be applied for pressure ulcers infected or colonized with staphylococcus aureus and streptococcus pyogenes resistant to other topical agents. Prolonged use, however, may result in overgrowth of nonsusceptible microorganisms, including fungi.

If the infection spreads into deeper tissues, the signs of infection become more overt with erythema, induration, purulence, and foul odor. Cellulitis and osteomyelitis may occur. Swab cultures of wound surfaces are not be useful in determining the presence of deep tissue infection related to pressure ulcers as they typically reflect the surface bacteria and not the particular bacteria within the tissue (Rousseau, 1989). Deep tissue biopsy is the commonly used method for obtaining a culture of bacteria within tissue (Sapico et al., 1986). Results may vary depending on the site of the lesion biopsied.

Poor wound healing and recurrence of pressure ulcers may result from underlying osteomyelitis. In a prospective blind trial involving

61 individuals with pressure ulcers, 52 of them had confirmed histopathologic diagnosis of osteomyelitis, and the value of some common tests in making the diagnosis of osteomyelitis (namely, white cell count, erythrocyte sedimentation rate, plain X-ray, Tc99 M bone scan, CT scan, and needle bone biopsy) was evaluated (Lewis et al., 1988). The most practical and least invasive evaluations involved a combination of white blood count, sedimentation rate, and two-view pelvic X-ray. This protocol was sensitive in 89% and specific in 88%. Bone scans and CT scans were expensive and were not found to be very sensitive. The most useful single test was needle bone biopsy, with a sensitivity of 73% and a specificity of 96%. MRI scanning may have an emerging use in diagnosis and evaluation of the extent of osteomyelitis. It may show bone necrosis in the presence of chronic osteomyelitis. However, in the absence of bone necrosis, the diagnosis of osteomyelitis by MRI scanning remains problematic. Furthermore, it has the advantage of showing soft tissue concerns related to pressure ulcers and osteomyelitis, e.g., deep abscess, significant undermining/tunneling.

Bone biopsy remains the definitive method for diagnosis and allows identification of the offending organism (Sugarman, 1987). When osteomyelitis is confirmed by bone biopsy, debridement may be necessary, in conjunction with appropriate postoperative antibiotics which are generally continued for 6 weeks.

CARCINOMA

Long-standing ulcers, usually present for 20 years or more, can develop a squamous cell carcinoma, known as a Marjolin's ulcer (Dumurgier et al., 1991; Schlosser et al., 1956; Treves and Pack, 1930). Warning signs include pain, increasing discharge, bleeding, foul odors, and verrucous hyperplasia. A tissue biopsy is essential when suspected. Metastasis to inguinal nodes is common (Berkwits et al., 1986).

ABNORMAL WOUND HEALING

Full-thickness wounds heal by a process of granulation, epithelialization, and contraction. Granulation tissue, normally granular and uneven, indicates the growth of new capillary loops and a matrix of collagen and ground substance in the wound base (Flanagan, 1998). Healthy granulation tissue is bright red, moist, and shiny; rapidly proliferates; and does not bleed easily (Flanagan, 1998).

Granulation tissue extending above the wound margins is termed hypergranulation. This "exuberant" tissue delays wound healing by retarding epithelialization (Kiernan, 1999). The

etiology of this clinical finding is unclear. Several methods are used to manage hypergranulation including use of silver nitrate sticks, silver dressings, sharp debridement, and semi-occlusive or nonocclusive dressings that dry the wound. In a prospective, noncontrolled, correlational study, Harris and Rolstad (1994) found a 2mm significant decrease in height of granulation tissue within 2 weeks of using a polyurethane foam dressing to treat hypergranulation (N=12 wounds).

NUTRITION

See nutrition section under recommendation 8.

MEDICATION

Corticosteroids affect almost every phase of wound healing. However, doses greater than 40 mg per day of prednisone are needed to adversely affect fibroplasia and collagen remodeling when taken for more than 3 days (Karukonda 2000). Immunosuppressants, such as azathioprine, cyclophosphamide, cyclosporine, and methotrexate, in general have no significant inhibition of wound healing (Karukonda 2000).

SUPPORT SURFACES

Inappropriate, ineffective, or worn-out support surfaces can prevent pressure ulcers from healing (Wilczweski et al., 2012).

TRANSFERS

Poor transfer technique with inadequate body clearance over obstacles, such a wheelchair tire, can result in friction and shear pressure damage to tissues, including existing ulcers, impacted during the transfer. Areas of the body most commonly affected by poor transfer technique include the ischium, trochanters, and sacrum.

NONCOMPLIANCE

Noncompliance to recommended best practices such as the regular performance of pressure redistribution, position change, use of pressure relieving support surfaces, and proper transfer technique can all contribute to the non-healing of pressure ulcers regardless of the intensity of treatment.

Treatment – Surgical

Referral for Pressure Ulcer Surgery

19. Refer individuals with deep category/stage III and category/stage IV pressure ulcers for operative intervention. For persons deemed appropriate candidates for surgical reconstruction, adhere to the following tenets of surgical treatment:

- Reverse any pressure ulcer risk factor if possible (e.g., impaired nutritional status) and address pre-op medical risk.
- Prior to surgery, treat osteomyelitis or cellulitis. This may need to be combined with excision of infected bone during surgery.
- Fill dead space and enhance the blood supply of the healing wound by mobilizing well-vascularized soft tissues flaps.
- Contour bony prominences to yield larger, flatter surfaces to augment pressure distribution.
- Reconstruct soft tissue defects with large regional pedicle flaps, placing suture lines as far away from the area of direct pressure as possible and with minimum tension. Avoid encroaching on adjacent flap territories.
- Preserve options for future potential breakdowns.

(Scientific evidence—I, III, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Achieving a successful outcome after surgical treatment of a pressure ulcer depends on proper patient selection, pre-operative optimization, operative procedure selection, post-operative management including graduated mobilization, and a supportive post-hospitalization program. The best approach to accomplish these objectives is through a multidisciplinary team. A comprehensive, interdisciplinary protocol for the surgical management of pressure ulcers is strongly recommended. This protocol includes in-depth assessment of medical status, i.e., co-morbidities/medical conditions, thoughtful planning for specific surgical procedures based on anatomical area and size of soft tissue defect, consideration of underlying bony anatomy, and appropriate follow-up care. A definitive set of criteria for the selection of patients for surgical repair of pressure ulcers does not exist, however, several articles with decision guidelines have been published (Sørensen et al., 2004; Bauer, 2008; Tchanque-Fossuo, 2011). In general, indications for surgery should be strict and treatment goals realistic with the ultimate goal of sustained improvement of quality of life.

Just as there are no specific criteria for selecting patients for surgery, there is no definitive algorithm to determine which flap to use for pressure ulcer repair. In general, the least demanding procedure with the greatest potential for successfully achieving the agreed upon pre-operative goals should be preformed, based on anatomic location, comorbidities, and

psychosocial analysis. Myocutaneous and fasciocutaneous flaps have superior success rates (Bauer, 2008). Regardless of selection, the flap should be as large as possible with placement of suture lines away from areas of direct pressure; flap design should allow for future re-mobilization and should not violate adjacent flap territories, both of which will preserve all future options for coverage of subsequent ulcers (Foster et al., 1997). In addition, the surgeon should be flexible and plan for several alternate flaps to accommodate unexpected findings in the operating room.

Goals of surgical intervention include:

- Restoration of skin integrity and function
- Elimination of unstable scar tissue
- Recontouring of bony prominences to improve soft tissue pressure distribution
- Diagnosis and treatment of osteomyelitis
- Reduction of healing time
- Reversal of chronic inflammatory state and restoration of anabolic homeostasis
- Prevention of progressive secondary amyloidosis and renal failure
- Prevention of future malignant transformation of ulcer (Marjolin's ulcer)
- Improvement of hygiene and appearance
- Reduction of health-care costs

In general, category/stage I and II pressure ulcers can be treated non-surgically, while category/stage III and IV ulcers are more likely to require surgical intervention to achieve closure and healing. The high recurrence rates after pressure ulcers are allowed to heal by secondary intention (spontaneous healing) and long duration to achieve complete healing are often cited as reasons that surgical closure for category/stage III and IV ulcers may be the most appropriate. Some clinicians argue that the recurrence rate after surgical closure is high as well, however, this is very difficult to assess since the recurrence rate after surgical repair of pressure ulcers reported in the literature across multiple studies ranges from 19-90% (Tchanque-Fossuo et al., 2011). The truth of the matter is that there is a significant recurrence rate whether a pressure ulcer is healed surgically or non-surgically (Guihan et al., 2008). These data indicate that obtaining pressure ulcer closure and preventing any recurrence is not always an achievable goal in all patients.

Because the risk factors that predispose the development of pressure ulcers are the similar to

those that are associated with ulcer recurrence after surgery, patient selection and pre-operative preparation are paramount (Tchanque-Fossuo et al., 2011). Successful surgical repair of pressure ulcers is largely determined by adherence to appropriate dressing change routine and pressure-relief protocol including the use of appropriate pressure re-distributing support surfaces, maintenance of nutritional health, and management of co-morbid medical conditions. The surgical procedure is often time consuming and may be associated with significant blood loss and anesthetic challenges (Bauer, 2008). Successful repair does not end in the recovery room post surgery. It is dependent on personal behaviors and active participation by the patient. Individuals with SCI must exhibit self-motivation to avoid deleterious actions and comprehend the pathogenesis of the ulcers to avoid redevelopment (Stal et al., 1983). It is difficult to measure subjective characteristics such as self-motivation, comprehension, quality of life, and probable individual cooperation. Clinicians must use clinical judgment with input by the entire interdisciplinary health care team in making treatment decisions.

The operative plan requires debridement immediately prior to closure even for wounds that appear clean. All contaminated and heavily scarred tissue should be removed, including partial osteotomy of exposed bone, producing as fresh a wound as possible. All bony irregularities that would cause extreme pressure points should be eliminated (Tchanque-Fossuo et al., 2011). Since pressure ulcers represent tissue loss and an overall tissue deficiency, reconstruction with the interposition of a well-vascularized flap is the reconstructive strategy of choice most of the time. To reiterate, there is no strong evidence favoring the use of any specific anatomic flap (since every ulcer is unique with its own challenges); however, in general most surgeons prefer to close ischial ulcers with a leg flap first (tensor fascia lata, posterior thigh, hamstring, gracilis), sacral ulcers with a gluteal flap, and trochanteric ulcers with a tensor fascia lata or vastus lateralis flap (Tchanque-Fossuo et al., 2011; Bauer, 2008). In all cases the closure should be tension-free with closed-suction drainage for prevention of fluid collection under the flap.

Preoperative Assessment

20. Address the following factors to enhance the effectiveness of pressure ulcer surgery:

- Presence of osteomyelitis
- Wound bioburden
- Nutritional status
- Bowel and bladder management
- Spasticity and contracture
- Heterotopic ossification
- Comorbid medical conditions
- Anesthesia
- Previous ulcer surgery
- Urinary tract infection
- Smoking cessation

(Scientific evidence—I, II, III, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

Several conditions need to be optimized or corrected prior to operative repair of pressure ulcers. Surgery should be delayed until the individual is in optimum condition.

OSTEOMYELITIS

Bacterial infection of bone occurs by introduction of microorganisms via hematogenous seeding, contiguous spread from surrounding structures, or direct inoculation from surgery or trauma. Osteomyelitis associated with pressure ulcers most likely results from bacterial contamination of exposed bone in category/stage IV ulcers, or from translocation from the ulcer bed of a category/stage III ulcer. Clinical staging of adult osteomyelitis (as opposed to pediatric osteomyelitis which predominantly occurs by hematogenous seeding) based on anatomic type was classical described by Cierny in 1985, and has hence been called the Cierny-Mader Classification System (Cierny, 2003). This system divides osteomyelitis into four anatomic types: Type I – medullary (central), Type II – superficial (surface), Type III – localized (full-thickness of cortex), and Type IV – diffuse (circumferential disease). Osteomyelitis arising from deep pressure ulcers is mostly Type II and may only rarely develop into Type III or Type IV if left untreated for a prolonged time (Darouiche et al., 1994). The traditional treatment of osteomyelitis with 4-6 weeks of parenteral antibiotics was established by extrapolation from animal models in 1970s and 1980s (Fraimow, 2009). However, there is more recent evidence that in the absence of sepsis, the osteomyelitis associated with pressure ulcers is of limited clinical consequence (Türk, 2003) and

may be treated with a lesser duration of antibiotics (Marriott, 2008). Appropriate antibiotic selection requires the identification of the causative organism from bone culture.

Successful treatment of osteomyelitis is based on a combination of medical and surgical modalities, the balance determined by the extent of disease. Debridement is the basis of surgical treatment, and should be direct and atraumatic with the ultimate goal of reconstruction. All necrotic (de-vascularized) and infected bone should be removed, unless the goal of treatment is non-curative. Osteomyelitis of the greater trochanter of the femur associated with trochanteric pressure ulcers represents a special case. Significant disease or communication with the hip joint requires a Girdlestone Procedure or resection arthroplasty of the hip (popularized by British surgeon Gathorne Robert Girdlestone in the early 20th century for the treatment of late septic arthritis of the hip) to eradicate the infection. The resulting wound is typically repaired with soft tissue coverage using a vastus lateralis muscle flap and is considered essential to successful management of those cases (Evans et al., 1993). Girdlestone surgeries present unique, seating challenges due to the sitting surface being reduced to a smaller area. When a girdlestone procedure is performed, the thigh/femur is no longer a viable load bearing surface. It is no longer connected to the body by a bony structure, therefore, it cannot offer support. A referral to a specialized seating clinic is recommended following this procedure so that the most appropriate seating system accommodations can be prescribed for maximal sitting stability and skin protection over the weight bearing pelvic surface.

Myocutaneous flaps have been long established as the preferred method of reconstructing wounds in the presence of infected bone including pelvic pressure ulcers (Bruck et al., 1991). Well-vascularized muscle tissue increases the antimicrobial potential of the wound by supplying oxygen, nutrients, and antibiotics to a previously hypoxic wound bed. There is some controversy over the timing of reconstructive surgery, with some advocating for treatment and complete resolution of osteomyelitis prior to flap coverage (Han et al., 2002), presumably to reduce infectious complications following reconstruction. This requires either a pre-operative core needle bone biopsy and culture or a two-stage reconstruction with operative debridement and bone biopsy as a first stage, followed by 4–6 weeks of parenteral antibiotics. Clearly, cases of acute suppurative infections

should be debrided and drained, but the overwhelmingly more common chronic superficial osteomyelitis found with pressure ulcers can be successfully treated as a one-stage surgical procedure by adequate debridement of diseased bone with biopsy of the remaining healthy bone surface (to determine any residual bacterial contamination and to direct post-operative antibiotic therapy) and immediate flap reconstruction. (Larson et al., 2011; Marriott, 2008; Darouishe et al., 1994). In fact, a recent published case series of 101 patients over a span of five years found no correlation between positive bone cultures and surgical complications or ulcer recurrence (Larson et al., 2012).

WOUND BIOBURDEN

The bioburden of a wound refers to the absolute number of microorganisms with which it is contaminated. Bacteria in a wound may originate from normal body flora, enteric sources, or from the environment. There is a wide spectrum of bacterial activity level in wounds, ranging from contamination or colonization (proliferating colonies without host response) to overt suppurative infection (bacterial invasion of healthy tissue) or cellulitis. Colonization generally does not impact wound healing, however, at a certain point critical colonization occurs based on bacterial number or virulence, with the beginning of a host inflammatory response and impaired wound healing. Clearly, purulent wounds must be drained. All non-viable and necrotic tissue must be debrided and removed as a nutrient source for bacteria.

The administration of systemic antibiotics (oral or parenteral) should be reserved for cases of objective findings of infection (or for persons with immunodeficiency). Swab biopsy of a wound is of no value in the diagnosis of wound infection and antibiotic selection since these cultures are invariably polymicrobial and reflective only of surface contamination and do not isolate the invasive bacteria causing the infection. (Levi, 2007; Bauer, 2008). To diagnose infection of the tissue below the wound surface, a quantitative culture is taken.

Prior to definitive flap closure, the wound bioburden should be reduced as much as possible. This involves wound debridement as discussed earlier, which is further performed in the operating room with copious irrigation immediately prior to flap inseting. Additionally, the bioburden can be controlled using topical antimicrobials (not systemic antibiotics) such as sodium hypochlorite (Dakin's solution), and

silver- or iodine-based products for local wound care during the pre-operative period.

NUTRITIONAL STATUS

The healing of any wound, including surgical incisions, requires adequate resources both caloric and protein. This will help ensure that the body is in an anabolic (protein-building) condition and not in a catabolic state (protein-destroying) state. To augment clinical findings of malnutrition (decreased subcutaneous tissue, nail/hair changes, decreased body mass index, etc.) biochemical data such as serum albumin have been used for objective assessment of nutritional status. While a national VA surgical risk study found that decreasing levels of serum albumin are associated with increased morbidity and mortality rate in general surgery, non-cardiac thoracic surgery, and orthopaedic surgery cases (Gibbs J et al., 1999), recommendations for specific serum levels prior to pressure ulcer closures have not been truly evaluated for their significance on postoperative healing.

Conventional wisdom is that malnutrition should be corrected through the administration of protein and caloric supplementation (and micronutrients such as vitamins and minerals to correct specific deficiencies) to achieve positive nitrogen balance prior to surgical repair of pressure ulcers. Improvement of nutritional status can be monitored via the weekly measurement of serum pre-albumin and transferrin, both of which have much shorter half-lives than albumin. In reality, it is often difficult to attain positive protein balance and a normal serum albumin level above 3.5g/dL due to the increased protein consumption by the sustained chronic inflammatory state found with most chronic pressure ulcers (Scivoletto et al., 2004). While a pilot study found that the anabolic steroid oxandrolone may stimulate pressure ulcer healing through its metabolic effects, this was not borne out by a follow-up multicenter trial (Bauman et al., 2013). In fact, it has been shown that the metabolic abnormalities found with large pressure ulcers are most reliably corrected after surgical intervention with debridement and flap coverage (Scivoletto et al., 2004; Larson et al., 2012).

BOWEL MANAGEMENT

The maintenance of healthy and noninfected tissue is essential in the management of pressure ulcers. Clearly this entails prevention of contamination by fecal soilage. If someone with SCI and pressure ulcers does not have volitional control of defecation and experiences fecal incontinence, a bowel routine should be implemented as described in *Neurogenic Bowel*

Management in Adults with Spinal Cord Injury. March 1998. Some clinicians strongly recommend a colostomy to achieve a cleaner perineal milieu and contend that the elimination of chronic constipation and complex bowel care regimens improves overall quality of life (Munck et al., 2008). Arguments used against elective stoma creation suggest that it is associated with significant morbidity and complication rates, however, modern operative techniques using minimally invasive procedures have greatly reduced the morbidity and mortality rates compared to historical reports (de la Fuente et al., 2003).

Fecal incontinence must be controlled before surgery (Lewis, 1990). Preoperative evacuation of the colon and rectum, especially on the morning of operation, with the use of oral laxatives and enemas will reduce the risk of early intraoperative and early postoperative wound contamination. Again, temporary bowel diversion via a colostomy may be indicated for individuals with a pressure ulcer in close proximity to the anus and is performed routinely at some institutions (Rubayi, 1999). This procedure will minimize the risk of flap compromise and infection after surgery and overall healing complications, since the healing of surgical incisions is impaired by fecal exposure.

SPASTICITY AND CONTRACTURES

Hyperreflexia secondary to upper motor neuron lesions may be helpful in preventing muscle atrophy and improving the ability to transfer to and from bed, but severe spasticity precludes surgery (Herceg and Harding, 1978). Muscle spasms may be sufficiently severe enough to rip open fresh surgical incisions (Bauer, 2008). Therefore, spasticity control should be optimized before surgical intervention for pressure ulcers.

Oral pharmacological treatment includes the use of baclofen, tizanidine, and occasionally benzodiazepines. Muscle blocks with botulinum toxin and nerve blocks using alcohol or phenol can be effective for targeting specific spastic muscle groups. More invasive treatments may include placement of an intrathecal pump for the administration of baclofen and as a last resort, dorsal rhizotomy.

Severe flexion joint contractures that result in the tightening of muscles and joint capsules and the limiting of range of motion may aggravate development of pressure ulcers and also promote recurrence. These contractures can limit patient positioning and make relieving pressure on bony prominences difficult for caretakers (Bauer, 2008). Contractures of the lower extremities are especially prone to the development of pressure

ulcers on the trochanters, knees, and ankles. When contractures are severe, preliminary flexor tendon releases should be considered; however, these contractures should not necessarily be totally released because of the risk of flail extremities and vascular compromise from extreme shortening of vein, artery, and nerves across the contracted joints (Hafer et al., 1983).

HETEROTOPIC OSSIFICATION

Heterotopic ossification is a specific cause of contracture that may develop in the knees, shoulders, elbows, hips, and spine and may restrict joint mobility, aggravating the propensity to develop pressure ulcers due to the limited ability to adopt appropriate supine and sitting postures for effective pressure distribution. Diagnostic findings include elevated alkaline phosphatase and evidence on X-ray and triple-phase bone scan or on computerized tomography (Bressler et al., 1987). Mature heterotopic ossification can be removed to restore joint motion, but removal of immature bone may result in increased risk of recurrence of heterotopic ossification. Extensive bone resection may lead to considerable blood loss (Rubayi et al., 1992). As is the case with uncontrollable muscle spasms and joint contractures, heterotopic ossification can affect seating positioning and range of motion. It may increase the risk of pressure ulcer development and affect treatment options. Its impact must be assessed prior to surgical intervention.

Heterotopic ossification may also be involved within a pressure ulcer itself. One of the proposed stimuli for the formation of heterotopic ossification is inflammation, a condition intimately involved with chronic wounds like pressure ulcers. The ectopic bony tissue can create abnormal pressure points and may be the nidus of osteomyelitis. In the operating room, heterotopic ossification may be encountered during pressure ulcer repair, ranging from small spicules to ankylosis across a joint. As much of the heterotopic bone should be removed as possible.

COMORBID CONDITIONS

Cardiovascular disease, pulmonary disease, peripheral vascular disease, and diabetes have all been implicated as factors contributing to poor wound healing. Neither one nor a combination of these conditions was found to correlate significantly with a poor outcome of surgical closure in one study which looked at them, however (Goodman et al., 1999). Nonetheless, all medical comorbid conditions must be addressed and optimized in order to minimize surgical and anesthetic risk prior to any operation.

ANESTHESIA

Airway management and positioning is a challenge in the operating room. Because the majority of pelvic pressure ulcers occur on the dorsal surface of the body, ensuring adequate surgical exposure necessitates placing the patient in a prone position, and often flexed in order to achieve tension-free closure of incisions. General anesthesia with endotracheal intubation is typically required for airway control and to mitigate aspiration and bronchospasm, and to maintain proper patient positioning on the operating table.

The administration of anesthesia is further complicated by potential autonomic dysfunction common with persons with SCI, which may manifest as bradycardia and hypotension or tachycardia and hypertension depending on level of injury and whether or not sympathetic tone is preserved. In addition, the paralytic agent succinylcholine should not be used in patients with SCI as there is a lifetime risk of serious hyperkalemia. These factors mandate that anesthesia be administered by experienced personnel, most often found in specialized SCI centers (Bauer, 2008).

PREVIOUS PRESSURE ULCER SURGERY (RECURRENCE)

An analysis of pressure ulcers that occur after previous surgical closure, specifically at the same anatomic location, is difficult because there is no clear consensus on the definition and natural history of these recurrent ulcers. Do these ulcers arise *de novo* or do they represent incomplete surgical healing? One can assume that early recurrences are due to incomplete healing and late recurrences are separate entities from the previous ulcer, but what time period is used to define early versus late? Late ulcer recurrence has been associated with unmodified patient factors such as spasticity, pressure relief behavior, and psychosocial status and not with surgical flap design (Bates-Jensen et al., 2009; Keys et al., 2010).

How does one stage ulcers that occur at the same location as a previous ulcer closed surgically staged? Presumably all of the ulcerous and scar tissue was excised at the time of the prior operation with correctly performed approximation of incisional edges resulting in the reconstitution of normal anatomic tissue layers. This is not the case for ulcers that recur at locations where a previous ulcer was allowed to heal by secondary intention with scar tissue. If the fascial layer was re-established with use of a myocutaneous or faciocutaneous flap for ulcer repair, then it should be appropriate to stage the

recurrent ulcer as a *de novo* ulcer even when it recurs along a healed tension-free surgical incision. This is not true for ulcers that recur within the scar of an ulcer healed non-surgically, which should be staged with the same stage as the previous ulcer.

The distinction between early ulcer recurrence (from tissue loading with incomplete surgical healing) and late recurrence (new ulcer formation) is key to interpreting the contradictory literature evidence. One study stated that the history of a surgically repaired pressure ulcer was a marker for poor wound healing and outcome (Allman et al., 1995). A more recent study reported that any history of prior same-site dehiscence or recurrence increased the rate of long-term flap failure from 40% to 52% (Keys et al., 2010). Other studies have found that the success of flap closure for pressure ulcers was not affected by previous flap reconstruction (Foster et al., 1997; Kierny et al., 1998). A previous flap reconstruction does not seem necessarily to correlate negatively with any of the surgical outcome variables if an ulcer recurs at the same site (Goodman et al., 1999). If an individual had multiple previous surgeries for ulcers at different but contiguous sites, flap reconstruction will become more difficult (perhaps even impossible) because of the amount of scar tissue and the lack of remaining available flap reconstructive options. The amount of ulcer-free time achieved after surgical closure should also factor in patient selection for subsequent flap repair in a cost (morbidity/mortality risk, resource utilization) versus benefit analysis. For individuals with recurrent pressure ulcers despite multiple previous flap surgeries, where reconstructive surgery is no longer indicated or possible, operative intervention (i.e., debridement) may be required to control bioburden or treat infection as a palliative wound management measure.

SMOKING

While there may be some controversy about the overall impact of smoking and nicotine on the development, healing, and recurrence of pressure ulcers, it is well known by plastic surgeons that cigarette smoking is associated with impaired healing of skin flaps (Kreuger, 2001). The proposed biological mechanisms by which smoking impairs wound healing include vasoconstriction, displacement of oxygen from hemoglobin binding sites by carbon dioxide, increased platelet aggregation, impairment of inflammatory cell oxidative burst, reduced collagen deposition, endothelial damage, development of atherosclerosis, and increased

blood viscosity. Oxygen is essential in all aspects of healing, and any condition that decreases the delivery of oxygen to the wound is detrimental. After smoking for just 10 minutes, the levels of oxygen in the skin are reduced by 22–48% (Jensen, 1991). Numerous studies have been published establishing that smokers are at increased risks of cardiopulmonary and wound-related postoperative complications; one study found that flap necrosis occurred three times more frequently than in patients smoking one pack of cigarettes a day compared to non-smokers, and six times more frequently in patients smoking two packs a day (Moller et al., 2002).

Several studies have shown that pre-operative smoking cessation reduces the risk of smoking-associated complications. A published review of multiple randomized controlled trials supports smoking cessation at least four weeks before surgery (Thomsen et al., 2009). Taking all this data in mind, it has been recommended that patients be nicotine abstinent for at least four weeks prior to flap reconstruction for pressure ulcer repair, with some surgeons advocating for verification using nicotine/cotinine testing.

URINARY TRACT INFECTION

Individuals with SCI have a higher than normal risk of urinary tract infections. Due to the risk of bacteremia and sepsis that UTIs generate, adequate preoperative management is essential. The difficulty is in differentiating infection from colonization as is the case for chronic wounds like pressure ulcers.

Postoperative Care

Clinical reports and anecdotal information indicate that the most successful centers provide post-operative care after flap closure based on strict multidisciplinary protocols. These protocols have changed dramatically in recent years, primarily because of the significant decrease in length of hospital stay after surgery as well as the development of new technology. While the specifics may differ between each facility and across the published literature, certain commonalities do exist: strict post-operative pressure relief and bed rest on a pressure redistributing support surface, graduated resumption of activity, modification of the risk factors for pressure ulcer development (and recurrence), and education of the patient and the patient's family and caregivers about pressure ulcer vigilance (Kierney et al., 1998; Levi, 2007; Bauer, 2008; Tchanque-Fossuo et al., 2011). In actuality, the last two themes of risk modification

and education should be a part of pre-operative planning in the same vein as the aphorism that has stood the test of time, that discharge planning begins on hospital admission.

POSTOPERATIVE POSITIONING AND SUPPORT SURFACES

It is important that no pressure be applied to the operative site after surgery as this decreases vascular perfusion and blood supply. As with all wounds, healing surgical incisions are hypermetabolic and require a sufficient supply of oxygen and nutrients. Premature tissue loading greatly increases the risk of dehiscence and poor healing. In addition, the application of shear and friction across healing incisions can overcome their burst strength and directly cause a dehiscence. The selection of post-operative support surface used during the period of bed rest should be based on the modern concepts of immersion and envelopment to maximize pressure distribution. Traditionally an air-fluidized bed has been used. A more recently developed fluid immersion simulation air mattress system may prove equally effective, however, there is no high-level evidence supporting the use of any specific support surface (Tchanque-Fossuo et al., 2011). The postoperative patient should be maintained on a turning regimen that does not apply pressure to the operative site. While in bed, the head of the bed should not be elevated by more than 15 degrees in people recovering from sacral or ischial repairs since this position increases the risk of shear on the repaired ulcer site. Prone positioning has been suggested in the past, but is currently used less often given the advances in bed and mattress technology, and should only be used with consideration of airway maintenance and ventilation which are both problematic with immersive surfaces.

POSTOPERATIVE MEDICAL CARE

The use of constipating medications administered in the acute post-op period and a low-fiber diet to avoid fecal contamination of the surgical site has been described in the past by a few centers (Black and Black, 1987; Rubayi et al., 1990), but is not in widespread use. Caregivers should be vigilant to the development of postoperative ileus with regular bowel regimens restarted as soon as possible.

The use of prophylactic antibiotics was commonly advocated in the past; however, modern antibiotic therapy mandates culture-directed selection. Antibiotics may be used in conjunction with surgical management of pressure ulcers. Adequate wound debridement prior to flap coverage should excise all potential

niduses of infection (Bauer, 2008). A single dose of a broad spectrum antibiotic with coverage of skin flora and enteric bacteria (e.g., piperacillin/tazobactam) should be used within two hours of initial skin incision (Tchanque-Fossuo, 2011). Post-operative antibiotic therapy should be reserved for cases of osteomyelitis proven by bone biopsy obtained after bone debridement immediately prior to flap coverage with an appropriate culture-specific antibiotic (Larson et al., 2011; Larson et al., 2012).

Deep venous thrombosis prophylaxis should be initiated consistent with clinical practice guidelines for SCI. However, a recent article (Rimler et al., 2011) analyzed a 5-year, 260 case series in which no pre-operative DVT prophylaxis was given for patients with chronic SCI and found that there was a zero incidence of peri-operative DVT. This is important because chemical DVT prophylaxis can increase the risk of bleeding and hematoma associated with surgical flaps due to the large tissue surface areas involved with flap mobilization.

Indwelling urinary catheters are frequently used in the post-op period to prevent contamination of the surgical site by urine. Persons with higher levels of SCI are at high risk for pulmonary complications including atelectasis and pneumonia in this period of enforced bed rest and chest percussion and postural drainage as well as measures to maintain alveolar expansion such as incentive spirometry should be implemented in appropriate at risk individuals.

POSTOPERATIVE MOBILIZATION

Experienced centers reporting the best outcomes for pressure ulcer surgery follow a standardized protocol or clinical pathway in keeping with the common general theme for surgical repair of pressure ulcers (Bauer, 2008; Keys et al., 2010; Tchanque-Fossuo et al., 2011; Larson et al., 2012). Again, while the specifics may differ, these reported post-flap protocols involve a period of strict bed rest immediately after surgery and passive range of motion when healing permits, followed by a graduated and progressive sitting regimen with a seating assessment.

The length of time of strict bed rest is not completely arbitrary, but rather based on the time course of the healing of primarily closed wounds taking into account tensile strength and flap vascularity. The healing of flaps also involves the formation and maturation of new vascular anastomoses between the flap and the recipient bed, with 90% of final flap circulation achieved after 3 weeks. In the past, persons who had

pressure ulcer surgery were usually confined to bed for 6 weeks while the surgical site healed (Stal et al, 1983). Given modern constraints on resource utilization, many centers have trialed shorter courses of bed rest and observed that there was no difference in outcome; more recent articles now report bed rest periods of only 3 weeks (Tchanque-Fossuo et al., 2011; Larson et al., 2012).

Since prolonged bed rest may result in the stiffening of joints, tendons, and ligaments, passive ranging of joints should be conducted prior to sitting (Kierney et al., 1998; Keys et al., 2010). Because ranging of the hips and knees may result in tension to the flaps used to close ischial and sacrococcygeal ulcers, range of motion therapy should only be initiated when the incisions are deemed strong enough to tolerate this. Hip and knee range of motion should be performed slowly, gently, and incrementally to avoid flap dehiscence. The ultimate goal of range of motion exercises is to assess whether or not an appropriate seating posture can be achieved and to attain this permissive joint motion. Though optimal seating posture requires at least 90-degree flexion and the hip and knee, more limited angles may be accommodated by certain wheelchair modifications to open the back angle. Prolonged bed rest decreases strength and endurance. An upper limb bed exercise program must begin when the person is medically cleared. This may include wrist weights, barbells, and/or elastic resistance bands.

Wheelchair activity is initiated after the mandated period of bed rest and when hip and knee range of motion has been optimized. Once again there is variation on the actual lengths of time used (e.g., initial seating for 30 minutes versus 60 minutes, etc.), however, the important facet is that there is a graduated progression of seated activity with skin re-assessment after each sitting episode (Kierney et al., 1998; Keys et al., 2010; Larson et al., 2012). During the first episode of post-operative seating, the patient should undergo a wheelchair and cushion assessment, with or without interface pressure mapping, to determine if any modifications are required to achieve optimal pressure distribution. There is no consensus on how rapidly to advance sitting time, however, any increase in time must be predicated on whether or not the flap and incisions are tolerating the loading pressure (Keys et al., 2010). Consistent weight shifting must be employed during any episode of sitting longer than 15-30 minutes. Optimal functional endpoints

are the ability to transfer and conduct activities of daily living (Kierney et al., 1998).

POSTOPERATIVE PATIENT EDUCATION AND PSYCHOLOGICAL SUPPORT

In many facilities, persons who have undergone pressure ulcer surgery are educated to perform weight shifts once they are bearing weight on a flap and inspecting the skin with a long-handled mirror or camera as well as proper skin hygiene, must be employed for comprehensive care (Black and Black, 1987; Kierney et al., 1998). They also are encouraged to inform home-based caregivers about preventing recurrence. Clinicians also must be concerned about psychological issues specifically depression following surgery. Persons confined to bed for long periods of time during healing may be at risk for depression (Smith et al. 2008). These individuals should be encouraged to engage in activities that allow them to interact with others and in activities that are enjoyable. Individualized education and structured follow-up have been shown to reduce the frequency of or delay the recurrence of pressure ulcers after surgical repair (Rintala, et al. 2008). All the principles of prevention discussed in the earlier chapter should be reviewed.

Complications of Pressure Ulcer Surgery

The most common complication following surgical repair of a pressure ulcer is dehiscence, specifically suture-line dehiscence or separation. Most surgeons differentiate the more common superficial suture-line dehiscence that can be treated with local wounds care from deeper disruptions that require re-operative closure (Keys et al., 2010; Larson et al., 2012). Superficial suture-line dehiscence may occur as a consequence of shear or friction from uncontrolled muscular spasms or inattentive patient positioning or turning. Deeper disruptions of surgically close tissue are more reflective of systemic problems with wound healing (e.g., impaired perfusion or hyperglycemia), infection, or poor surgical technique. These more serious dehiscences may require debridement and flap re-advancement.

Seromas occur when interstitial fluid or transudate collects in potential dead spaces that were not obliterated during surgery. Well-placed surgical drains maintained until drainage has appropriately decreased are usually sufficient prevention. In time, most small seromas are resorbed by the body (unless secondary infection

causes abscess formation), however, large and persistent seromas may require repeated percutaneous needle drainage (Levi, 2007).

A hematoma may develop with uncontrolled bleeding filling potential dead spaces similar to seromas. Hematomas that occur immediately after surgery can either be due to bleeding vessels missed during final closure of incisions in the operating room, or as the result of clot or cauterized eschar being displaced from previously controlled bleeding points due to episodes of extreme hypertension that may result from recovery from anesthesia or with autonomic dysfunction from painful stimuli. Internal bleeding may also result from disruption of tissue layers and vasculature due to shear injury. The clinical signs of active bleeding with hematoma formation include persistent bleeding into surgical drains or from the suture or staple line, and a palpable fluid collection. This collection of blood creates a very good culture medium and thus is prone to secondary infection. Hematomas should be evacuated in the operating room with control of any active bleeding sites (Levi, 2007).

As with any surgical procedure, suppurative wound infection is a potentially serious complication after flap reconstruction of pressure ulcers. Infection of the superficial skin structures (cellulitis) presents as peri-incisional erythema and disruption of the incision and is apparent from physical exam. The incision should be opened in order to achieve effective drainage of the infection, and to rule out any deeper infection. Infections of deeper structures can be insidious with late presentation of purulent drainage from the incision. This can be due to accumulation of purulent material with the potential dead space underneath the flap, forming a contained abscess that will eventually seek spontaneous drainage. The incision must be opened further to ensure adequate drainage and to enable dressing changes to be done. This can frequently be performed at the bedside; however, in rare cases extensive infection may require washout in the operating room (Levi, 2007).

Pressure Redistribution and Support Surfaces

The effects of pressure on the soft tissue are determined by three factors: the magnitude of the pressure, the duration of the pressure, and the ability of the skin and its supporting structures to endure the pressure without adverse effects. Preventive measures targeting the intensity of the

pressure include selecting an appropriate support surface and facilitating a body posture that minimizes areas of high pressures on the supporting surfaces with or without appropriate postural support. The duration of pressure is addressed by turning, weight-shifting effectiveness and frequency, and the use of an active support surface, such as an alternating pressure support surface, that can actively redistribute pressure on the body surfaces (Sprigle and Sonenblum, 2011).

Bed Positioning

21. Use bed positioning devices and techniques that are compatible with the bed type and the individual's health status.

- Avoid positioning individuals directly on pressure ulcers regardless of the pressure ulcer anatomical location (trochanter, ischium, sacrum, and heel) unless such position is necessary for performance of ADLs, such as eating or hygiene.
- Use pillows, cushions, and positioning aids to reduce pressure on existing pressure ulcers or vulnerable skin areas by elevating them away from the support surface.
- Avoid closed cutouts or donut-type cushions.
- Prevent contact between bony prominences.
- Elevate the head of the bed no higher than 30 degrees unless medically necessary.
- Reposition individuals in bed at least every 2 hours.

(Scientific evidence—II, V; Grade of recommendation—B; Strength of panel opinion—Strong)

The use of a positioning device is an effective means of raising the ulcer off the support surface. A bridging technique may be used to support bony prominences with pillows proximal and distal to the prominence. Adequate pressure relief with no support contact at the sacrum of the supine individual was accomplished using this technique by Bogie et al. (1992). Proper placement of cushions behind the back and between the legs will assist in pressure relief of bony prominences that may contact each other or the surface of the bed (Land, 1995; Lowthian, 1993). Positioning devices should also maintain postural alignment and prevent postural deviation. Avoid ring cushions (donuts) as they are more likely to cause pressure ulcers than to prevent them (Crewe, 1987). Pressure-relieving cutouts should be open to the edge of the cushion to allow blood flow to the surrounding tissue and

Table 4. Support Surfaces

Type of Support Surface	Characteristics of Support Surface	Indications	Precautions, Contraindications, and Disadvantages
Reactive (static) A powered or non-powered support surface that changes its load distribution properties only in response to an applied load, such as a patient lying, sitting, or moving on it Examples: foam, air, combination foam/air surfaces.	Foam: <ul style="list-style-type: none"> Conforms to bony prominences to redistribute pressure and reduce shear Viscoelastic foam is temperature-sensitive conforming to the body only when the temperature of the foam gets close to body temperature 	Foam: <ul style="list-style-type: none"> Individuals able to reposition themselves Individuals at risk of pressure ulcer development Multiple Category/stage II pressure ulcers 	Foam: <ul style="list-style-type: none"> Tends to increase skin temperature Foam degrades and loses resilience over time Lacks airflow and can allow moisture to accumulate between an individual's body surface and the support surface
	Air- or gel-filled: <ul style="list-style-type: none"> Low surface tension Conforms to bony prominences to redistribute pressure and reduce shear Airflow is delivered in a continuous pattern Easy to clean 	Air- or gel-filled: <ul style="list-style-type: none"> Individuals able to reposition themselves Individuals at risk for pressure ulcer development Individuals who are heavy or rigid and difficult to reposition Multiple Category/stage II pressure ulcers 	Air- or gel-filled: <ul style="list-style-type: none"> Ineffective if overinflated, underinflated, or punctured Inflation must be checked daily
Reactive (static) A powered or non-powered support surface that changes its load distribution properties only in response to an applied load, such as a patient lying, sitting, or moving on it	Low air-loss (LAL): <ul style="list-style-type: none"> Connected air-filled pillows across the support surface Cover is porous to allow leaking of air to the patient's skin surface May be powered with a pump to provide continuous airflow Calibrated according to the patient's height and weight Easy to clean Cover is impermeable to bacteria 	Low air-loss (LAL): <ul style="list-style-type: none"> Used to manage heat and humidity (microclimate) of the skin Pressure ulcer prevention Multiple Category/stage II pressure ulcers 	Low air-loss (LAL): <ul style="list-style-type: none"> Contraindicated for individuals with an unstable spine If support surface is not calibrated with an individual's height and weight or if mattress and bed frame size do not match, there is a risk of entrapment of individual between the edge of the support surface and bed side rails Powered support surfaces may be noisy
	Air Fluidization or High air-loss: <ul style="list-style-type: none"> Bed contains silicone-coated beads covered by a porous sheet and when air is pumped through the beads, the surface behaves like a liquid on which an individual floats Some hybrid beds have the upper portion as a low air-loss surface and the lower portion as a high air-loss surface 	Air Fluidization or High air-loss: <ul style="list-style-type: none"> After myocutaneous flap reconstructive surgeries Multiple pressure ulcers on two or more turning surfaces such as the sacrum and trochanter For re-warming individuals who are experiencing hypothermia Individuals with severe debilitating pain who cannot be repositioned Category/stage III or IV Pressure Ulcers 	Air Fluidization or High air-loss: <ul style="list-style-type: none"> Ensure that the support surface is pressurized at all times Individuals still need to be repositioned laterally at 30 degrees every 2 hours with head raised up no more than 30 degrees
Active (dynamic) Powered support surface with the capability to change its load distribution with or without an applied load. It is intended to change the magnitude and duration of the applied load	Alternating Pressure Mattress: <ul style="list-style-type: none"> Chambers or pillows are arranged throughout the entire length of the mattress A powered pump fills the pillows with air in periodic cycles of inflation and deflation, thus redistributing pressure by shifting the pressure points and actively shifting the body weight This may be combined with pulsating pressure 	Alternating Pressure Mattress: <ul style="list-style-type: none"> Individuals at high risk for pressure ulcer development Any category/stage of pressure ulcer Category/stage III or IV on trunk or pelvis History of pressure ulcer and potential for recurrence Acutely ill and immobile individuals Worsened wounds with reactive surface After myocutaneous flap reconstructive surgeries 	Alternating Pressure Mattress: <ul style="list-style-type: none"> Individuals still need to be repositioned laterally at 30 degrees every 2 hours with head raised up no more than 30 degrees

prevent pooling of the blood in the center of the wound. Further rationale for avoiding raising of the head of the bed and repositioning every 2 hours are described in “Prevention Strategies Across the Continuum of Care.”

Individual repositioning at planned, displayed intervals will enhance caregiver compliance even when individuals are using pressure-reducing support surfaces. Individuals with SCI, as well as their caregivers, should be educated regarding the optimal turning schedule as part of the development of their home program.

Some reports indicate the development of new pressure ulcers in people who are being treated for other pressure ulcers (Allman et al., 1995; Rochon et al., 1993) and in those who are already using pressure redistribution support surfaces of various types (Jesurum et al., 1996; Ooka et al., 1995). This indicates the need to maintain careful monitoring and preventive positioning while using pressure redistribution bed surfaces and to ensure they are working as designed to redistribute pressure.

Bed Support Surfaces

22. Use pressure-redistribution bed support surfaces for individuals who are at risk for or who have pressure ulcers (see Table 4: Support Surfaces).

- Select a reactive support surface for individuals who are able to reposition themselves enough to avoid weight bearing on all areas at risk for pressure ulceration and who have a stable spine.
- Select an active support surface for individuals who are unable to reposition themselves.
- Select an active support surface or a high air-loss (air-fluidized) reactive support surface for individuals who have pressure ulcers on multiple turning surfaces and/or are status post flap/skin graft within the past 60 days.

(Scientific evidence—I, II, IV, V; Grade of recommendation—A; Strength of panel opinion—Strong)

There are two types of support surfaces: **reactive support surfaces** and **active support surfaces**. A reactive support surface can change its load distribution properties in response to an applied load, such as a person’s movement. It can be powered or nonpowered. An active support surface has the capability to change its load distribution with or without an applied load. Active support surfaces are powered and dynamically redistribute pressure at the body-support surface

interface by changing both the magnitude and duration of the applied load. Active support surfaces redistribute pressure with or without tissue load; the cell inflation-deflation cycle occurs whether or not the individual moves.

Support surfaces redistribute pressure through immersion and envelopment. Immersion is the depth of penetration (sinking) into a support surface (NPUAP, 2007). Immersion allows the pressure to be spread out over the surrounding area rather than directly over a bony prominence. A support surface’s ability to provide immersion is based on its stiffness, thickness, and the flexibility of its cover. Envelopment is the ability of a support surface to conform, so as to fit or mold around irregularities in the body (NPUAP, 2007) without a substantial increase in pressure.

“Bottoming out” is the term used to describe the sinking of the individual into the support surface (bed or wheelchair) while lying flat on the mattress or sitting up in a wheelchair so much so that the underlying surface produces an unintended force upon the body, negating the effect of the support surface. This occurs if (1) the pressure setting, also known as the cell inflation pressure in the support surface is too low or inadequately set to support the patient’s weight; or (2) the support surface is losing pressure from an air leak. A subjective estimate of the compression can be made by palpation of the support thickness at the bony prominence. Bottoming out is tested by either placing the caregiver’s hand under the mattress overlay or wheelchair cushion with palms toward the individual or by placing the open hand with palm against the individual’s skin or clothing at the prominence and estimating the seat cushion or support thickness below by finger flexion and extension. If the support surface is less than 2.5 cm thick at the measured bony prominence, the cushion has “bottomed out.”

REACTIVE SUPPORT SURFACES

A low-air loss mattress is an example of a reactive support surface. It consists of a series of connected air filled pillows that run across the support surface. The amount of pressure in each pillow is controlled and can be calibrated to individual’s weight and height. A pump provides airflow in a continuous pattern and since the covering of the mattress is porous, it allows for leakage of air that controls the microclimate of the patient’s skin. The flowing air evaporates skin moisture (Scales et al., 1974) and reduces temperature (Flam et al., 1995) while maintaining a microclimate conducive for tissue healing.

If individuals can reposition themselves or can assume a variety of positions without bearing weight on the ulcer and without bottoming out the support surface, a reactive support surface is an appropriate option.

In an acute-care setting, the healing of large pressure ulcers has been shown to benefit from both the use of air-fluidized (high-air-loss) and low-air-loss beds (Bergstrom et al., 1994). Although home versions of air-fluidized beds and hybrid designs of low-air-loss and air-fluidized beds are available, low-air-loss beds are more commonly used in the home care setting because of the larger size, heavier weight, high power consumption, and the difficulty encountered with individual transfers for high-air-loss beds. Moreover, low-air-loss beds have been shown to be easily managed and are effective in pressure ulcer prevention and healing due to their reduced bulk and facilitation of individual positioning and transfers (Charles et al., 1995; Ferrell et al., 1993; Mulder et al., 1994).

ACTIVE SUPPORT SURFACES

An alternating pressure mattress is an example of an active support surface. It is designed with chambers or cylinders arranged in various patterns. Air or fluid pumped through these chambers at periodic intervals creates alternating deflation and inflation in opposite phases. It redistributes pressure through cyclical changes in loading and unloading characterized by frequency, duration, amplitude, and rate of change parameters. Alternating pressure mattresses have been associated with a lower incidence of pressure ulcers. Jan et al. (2011) compared the effect of alternating and constant pressure on weight-bearing tissue perfusion in people with SCI. They found that alternating pressure increased skin perfusion of weight-bearing tissues as compared to constant pressure supporting the concept of using an alternating pressure support surface to reduce pressure ulcer risk in the SCI population.

In alternating pressure systems, comfort is related primarily to cell inflation pressure and the rate of change of pressure during the cycle. A high inflation pressure prevents an individual's pressure points from "bottoming out" but leads to discomfort and high peak contact pressures. On the other hand, a low inflation pressure can increase comfort, but limits weight carrying capacity to support the individual, therefore increasing the likelihood of "bottoming out."

If someone cannot assume a variety of positions without bearing weight on the ulcer, compresses the reactive support surface

(experiencing elevated contact pressures), or the ulcer does not show evidence of healing, an active support surface should be used.

Active support surfaces are options for individuals with category/stage II pressure ulcers on multiple turning surfaces and a failure to heal on a reactive support surface. Similarly, the presence of a large category/stage III or IV pressure ulcer or a recent tissue graft for ulcer repair may also suggest the use of an active support surface (Charles et al., 1995; Day and Leonard, 1993).

ADDITIONAL CONSIDERATIONS

The individual circumstances of the person with a pressure ulcer must be considered in the prescription of a specialized bed or support surface as these devices may impact the ability to perform functional activities or affect the ability to provide core assistance. For example, it is often more difficult for someone with a higher level SCI to perform bed mobility or transfers on a active support surface than on a reactive one that may limit his or her functional independence and even require him or her to have additional help that might not have been needed on certain firmer reactive support surfaces. Any of these devices may not be appropriate in all home settings due to such factors as weight or operating costs.

Wheelchair Seating and Positioning

23. Prescribe wheelchairs and seating systems specific to the individual that allow that individual to redistribute pressure sufficiently to prevent the development of pressure ulcers.

- Obtain specific body measurements for optimal selection of seating system dimensions (postural alignment, weight distribution, balance, stability, and pressure redistribution capabilities).
- Prescribe a power weight-shifting wheelchair system for individuals who are unable to independently perform an effective pressure relief.
- Use wheelchair tilt-in-space and/or recline devices effective enough to offload tissue pressure.
- Use standing wheelchairs to remobilize individuals with existing pelvic pressure ulcers.

- Full-time wheelchair users with pressure ulcers located on a sitting surface should limit sitting time and use a gel or air surface that provides pressure redistribution
- Maintain an offloaded position from the seating surface for at least 1 to 2 minutes every 30 minutes.

(Scientific evidence—I, II, III, IV, IV; Grade of recommendation—A; Strength of panel opinion—Strong)

An effective wheelchair and seating system can help promote skin health, sitting balance stability, symmetrical posture, greater upper limb use, and enhanced functional performance. Wheelchair features that optimize independence in performing pressure redistribution, transferring, and propelling, as well as providing optimal postural support and minimizing the risk of developing pressure ulcers, are recommended (Garber and Krouskop, 1997). An individual physical and functional assessment by a clinician with specific expertise in all these areas and in complex mobility equipment is necessary to achieve the best outcomes (Beer, 1984; Lowthian, 1993; Rosenthal et al., 1996; Coggrave & Rose, 2003). In addition to an objective evaluation and clinical judgment by the clinician, it is essential to incorporate direct feedback from the individual requiring the wheelchair into the decision making process for determining what is the most appropriate wheelchair and seating system (Garber and Dyerly 1991; Garber 1985).

Sitting postures can significantly affect ischial pressures, and lateral pelvic tilt can affect pressure distribution over the buttocks; therefore, postural management is crucial when selecting a seating system (Koo et al., 1996). The loss of innervation to muscles of the body results in abnormal postures being assumed to achieve sitting stability within wheelchairs and seating systems. A common postural compensation seen in persons with SCI is posterior rotation of the pelvis, flattened lumbar curve, C-shaped thoracolumbar spinal curve, and extended cervical spine. Left unattended, these asymmetrical postures can result in deformity, excessive sitting pressures and shear, as well as loss of function (Hobson, 1992; Koo et al., 1996). Maintaining good postural alignment can facilitate equal weight bearing over the bony prominences of the buttocks (Krouskop et al., 1983). Unequal or excessive pressure or shear over bony areas, as well as impaired dynamic sitting stability can contribute to pressure ulcers (Bergstrom et al., 1992; Karatas et al., 2008). A “plumb line” posture (alignment of the ear/shoulder/hip) keeps normal spinal curves intact. Slouching forward or

leaning to one side places unequal pressure over the buttocks.

An interface pressure-mapping device is an array of sensors contained in a flexible mat that measure interface pressure between the user and the underlying support surface that can be used as a tool to help determine the wheelchair cushion and seating system to obtain that will best minimize the risk of pressure ulcer development. Most interface pressure-mapping devices provide a value of relative peak pressure values at the interface of the buttocks and the various wheelchair cushions, a visual display of uniformity of the weight-bearing surface, surface contact area and an ability to compare these values in a static and dynamic position (Bar, 1991; Salcido et al., 1996; Barnett & Shelton, 1997). As there is no absolute interface pressure that predicts the development of pressure ulcers (Sprigle and Sonenblum, 2011), interpretation of the data is only one aspect of determining pressure redistribution equipment. Interface pressure-mapping can be used to rule out the least desirable surface but cannot solely determine the optimal cushion surface for a person. One of the most beneficial applications of using a interface-pressure mapping system is to educate wheelchair users through the use of imagery observed during pressure relief techniques, the impact of posture changes and functional movements, and set up of the cushion, especially in the case of air floatation cushions (Coggrave & Rose, 2003; Henderson et al., 1994). There are multiple contributing factors to pressure ulcer incidence, and they must be taken into consideration additionally (Ho & Bogie, 2007; Makhsous et al., 2007). These factors include skin moisture, friction, shear, nutrition, age, and arterial pressure. Variability in body habitus, such as weight, muscle tone, body fat content, and skeletal frame size, also impact interface pressure (Gefen, 2007; Henderson et al., 1994; Hamanami et al., 2004; Barnett & Shelton, 1997). How people transfer into wheelchairs and how they position and reposition themselves within the seating system have a direct impact of pressure or shear forces that occur to the buttock-surface interface (Barnett & Shelton, 1997). Even the age, condition, and type of the cushion and the surface it is placed upon can factor into the development of pressure ulcers.

Wheelchairs not only provide mobility, but they can also provide an independent means of performing pressure redistribution (Nixon, 1985, Sprigle and Sonenblum, 2011) for those persons who are unable to physically lift their body from

the sitting surface, to forward lean, or to side-to-side lean (Sprigle, et al., 2010).

The purposes of mechanical pressure redistribution systems are—

- to allow for altering sitting pressures and for change in body pressure distribution to assist in the prevention of pressure ulcers;
- to allow for the change of postural alignment while sitting; and
- to allow the person to function for a longer period of time while sitting, given the ability to change positions for comfort and function.

Some wheelchairs have a fixed seat-to-back angle, which can be tilted in space to redistribute pressure by shifting weight off of the ischium onto the back. Typical tilting wheelchairs can obtain tilt angles from 45 degrees to as much as 60 degrees from a horizontal plane. Sprigle et al. (2010) in a small sample study of spinal injured subjects who used tilting wheelchairs found a 46% decrease in seat load when at full tilt.

Hobson's (1992) research has shown that a minimum of 45 degrees of tilt/rotation is required for adequate pressure distribution. Henderson, et al. (1994) report 65 degrees of tilt has significant reduction in maximum point pressure at ischial tuberosities, whereas 35 degrees of tilt did not demonstrate significant reduction of ischial tuberosity pressure. These systems are frequently selected if a significant problem with spasticity exists. When the body is in the tilted position, pressure is reduced without change in hip and knee flexion. With these systems pressure redistribution can be achieved without requiring passive movement of the hip and knee joint, thereby avoiding a stimulus to spasticity. Goossens et al. (1997) discusses how local shear stress is affected by changes in body posture, including head and arm movements while sitting. Tilt-in-space systems will also minimize frictional shear (Goossens et al., 1997; Hobson 1992).

Bladder drainage while in the tilted position must be assessed so as to avoid medical complications that can occur with urine backflow. Tilt systems facilitate consistent positioning and access to secondary switches mounted onto the wheelchair throughout the arc of movement (Sprigle et al., 2010).

Other pressure redistribution systems work by reclining the wheelchair to elevate the legs to approximately horizontal position, helping to redistribute weight-bearing pressure over a larger body surface. Power reclining systems must be evaluated for the possible effect of increasing and eliciting extensor spasticity in the body due to this

tendency when moving from a static to dynamic position (Sprigle et al., 1997). The reduced shear reclining-back style—one that allows the back to slide during the reclining movement—is preferred due to reduced risk of shearing to the skin of the back when the seat and back angle are changed. Backrest recline decreased mean maximal pressure of the ischial tuberosities but caused the greatest ischial tuberosity shift, up to 6 cm (Henderson, et al., 1994).

Upon opening the hip-to-back angle during the recline phase, the stretch of muscles surrounding the pelvis can be placed on a quick stretch. This movement may result in the loss of pelvic and spinal support, which may lead to increased pressure and shearing over the bony areas of the pelvis. In cases where pressure redistribution is not as effective with the use of a tilt system as compared to a recline system, a combination tilt and recline system may be considered. The individual is recommended to initially move to a fully tilted position to stabilize the pelvis, and then follow with activation the recline system so as to minimize loss of postural stability (Kreutz, 1997). The subsequent activation order is then reversed to return to upright sitting to minimize loss of stability.

When using power recline or combination tilt/recline systems for pressure redistribution, clinicians often instruct wheelchair users to lean forward away from the wheelchair back before returning to upright sitting position. This helps to reduce potential skin shearing that can occur along the chair's back (Gilsdorf et al, 1990; Sprigle et al. 1997).

There is no consensus as to whether a tilt-in-space or a reclining wheelchair is more effective in preventing pressure ulcers. However, it seems that a tilt and recline combination provides the most pressure redistribution when used on a consistent basis. Aissaoui et al. (2001) conducted a study of pressure distribution and sliding on 10 able-bodied subjects by assuming 12 postures on a simulator chair. Posterior tilt angle ranged from 0 degrees to 45 degrees and the recline angle varied from 90 degrees to 120 degrees. Back and seat force sensing array mats were used to measure the interface pressure at the seat and back with subjects seated on a flat foam cushion. Mean pressure, peak pressure, and peak pressure gradient were calculated for all positions tested. The study found that when subjects sat at 45 degrees of tilt and recline of 120 degrees, a 40% maximum reduction in peak pressure under the ischial tuberosities was found. These findings concluded that the highest reduction of pressure

was obtained when varying both seat tilt and recline as compared to initial values. In another study, 11 wheelchair users with SCI were assigned to one of six protocols of various wheelchair tilt-in-space and recline angles, and it was found that the greatest increase in skin perfusion and decrease in interface pressure over the ischial tuberosity was obtained when a wheelchair tilt-in-space was 35 degrees combined with a 100 degree recline and when a tilt-in-space was at least 25 degrees combined with a recline at 120 degrees (Jan et al., 2010).

Karatas et al. (2008) evaluated the center of pressure displacement and the dynamic sitting stability of SCI patients and their relation to pressure ulcer development. They found that the center of pressure displacement during dynamic unsupported right, left, forward, and backward leaning was smaller than in individuals without SCI participating in the study as controls. This difference can be explained by the loss of muscle function in individuals with cervical and thoracic SCI compared to individuals without SCI who had fully functional trunk, abdominal, hip and lower extremity muscles required to perform dynamic sitting.

Significant reduction in sitting force can be obtained by using armrests. The armrests support 10% of the body weight (combined weight of arm and hand), thereby relieving seating forces over the buttocks (Gilsdorf et al., 1991). Wheelchair footplate position needs to be addressed when adjusting the wheelchair. Foot-plate height should be adjusted to ensure that peak pressures over the pelvis are minimized. Footplates that are too high can result in a suboptimal sitting pressure distribution between the thighs and the ischial tuberosities (i.e., pressure that is increased over the ischial tuberosities). Footplates that are too low can result in the body sliding forward on the seat contributing to excessive shear and pressure to the ischial tuberosities (Gilsdorf et al., 1990). Excessive pressure of the posterior thighs can result in lower body edema. However when positioned in the chair with slight forward pelvic rotation, some pressure is usually shifted to the posterior thighs, which offloads the ischial tuberosities. Stable trunk support will prevent excessive shearing over the scapulae or sacral areas, which can occur if the person is not adequately supported in the wheelchair.

Standing wheelchairs, manual or power, are available for independent mobility. Standing systems can be utilized to allow for extended pressure redistribution over the seat and backrest areas, therefore allowing mobilization of persons

with or without existing pressure ulcers. Careful consideration must be used when determining the viability of using a standing wheelchair, such as orthopedic status, cardiovascular stability, spasticity, range of motion, and balance. In a small study of load distribution in a standing position, Sprigle et al. (2010) determined that a 61% reduction in seat force could be attained with a standing position of 75 degrees or full recline, as compared to 46% decrease in seat load at full tilt. Standing to achieve pressure redistribution may be seen as more socially acceptable than tilting or reclining back in community settings and therefore may be more likely to be utilized in such environments. The physical space needed for a wheelchair to assume a standing position as compared to tilting/reclining is less, thus can be performed in smaller areas where accessibility is an issue. ADLs performed in the standing position allow for pressure redistribution to be built into normal daily routines, resulting in a greater frequency of performance.

It is suggested that having power wheelchair features, such as power tilt/recline/standing, is not enough to facilitate use to prevent pressure ulcers. Consumers must be specifically educated on how to best utilize these features in order that they be used in the optimal manner. One study indicated that even though 97.5% of individuals had power tilt and/or recline on their wheelchairs and used these functions daily, less than 35% used them for the purpose of pressure redistribution rather using them to reduce pain and increase comfort (Lacoste et al., 2003).

Spasticity should be monitored and managed so as to prevent the effects of skin shearing when the body rubs against firm surfaces. Some of these surfaces may be bed linens, wheelchair parts, shoes, or braces and splints. After discharge to home or community, the individual with SCI must monitor the level of spasticity and seek medical guidance when it becomes detrimental to adequate skin care and function.

Sitting-acquired pressure ulcers occur within the soft tissues compressed between weight bearing bony prominences and the supporting surface of the wheelchair. More than 65% of sitting-acquired pressure ulcers occur on the ischial tuberosities, sacrum, coccyx, and trochanters (Gefen, 2007).

Reenalda et al. (2009) analyzed the sitting position interface pressure distribution and subcutaneous tissue oxygenation of 25 persons without SCI. They found that their subjects shifted posture an average of 7.8 times an hour in the

sagittal plane (80%) and frontal plane (20%). These posture shifts caused an increase of 2.2% in the subcutaneous tissue oxygen saturation (SO₂), suggesting increased tissue viability.

Pressure relieving and redistributing maneuvers are a critical element of pressure reduction (DeLateur et al., 1976). Historically, pressure redistribution have been recommended every 30 minutes for 30 seconds or every 60 minutes for 1 minute to allow reoxygenation of the cutaneous tissues (Nixon, 1985). Research has shown that the previously accepted duration parameters may not be sufficient to allow for raising transcutaneous oxygen tension to unloaded levels and that a duration of almost 2 minutes may be required (Coggrave & Rose, 2003; Barnett & Shelton, 1997). Obesity may reduce one's ability to perform adequate pressure redistribution and safe transfers. Obesity associated with excess rolls of soft tissue can lead to the development of skin breakdown on other parts of the body, due to these skin folds retaining moisture and bacteria and causing pressure on other areas.

Wheelchair Support Surfaces

24. Prescribe wheelchair seating systems for each person with a spinal cord injury individualized to anthropometric fit, to provide optimal ergonomics, and to provide maximal function.

- **Prescribe wheelchair seating systems that—**
 - Redistribute pressure
 - Minimize shear
 - Provide comfort and stability
 - Reduce heat and moisture
 - Enhance functional activity
- **Inspect and maintain all wheelchair cushions at regular scheduled intervals.**
- **Replace wheelchair seating systems that are no longer effective.**

(Scientific evidence—II, III, IV, V; Grade of recommendation—B; Strength of panel opinion—Strong)

Interface pressures at the ischial tuberosities are higher while sitting than lying down and must be relieved frequently to prevent tissue injury. When the pressure on the ulcer can be relieved by either assisted or self-mobility, limited sitting may be allowed (Bergstrom et al., 1994).

It may be necessary to prescribe a specific wheelchair back support to minimize unequal

weight bearing or shearing over the pelvis from an unstable trunk posture. Standard wheelchair seat and back upholstery is made of materials that tend to “sling and stretch” with use. Yarkony and Chen (1996) state “one of the most common problems arises from the basic wheelchair design, since the basic sling seat and back can result in pelvic obliquity and kyphotic posture, with increased risk of pressure ulcers, deformity, and discomfort.” Postural management is a significant determinant of proper seating of individuals. Postural instability can result from absent or weakened musculature, imbalanced muscle tone, orthopedic deformities, sensory deficits, or inadequately fitting support devices. Sitting posture was found to influence ischial pressure, and the final pressure distribution over the sitting surface was dependent on lateral pelvic tilt (Hobson, 1992; Koo et al., 1996). The use of contoured back supports can minimize unequal weight bearing, reduce shear forces on the pelvis, and stabilize trunk posture. Proper selection of seat and back supports can effectively solve postural seating problems (Buschbacher et al., 1996). Health-care professionals involved in the recommendation of wheelchair back supports should be knowledgeable about solving problems related to postural seating difficulties (Buschbacher et al., 1996).

Improving center of pressure weight shifts, especially in the sagittal plane should be incorporated into rehabilitation programs for persons with SCI. Evidence suggests that impaired dynamic sitting stability is associated with pressure ulcer development (Karatas et al., 2008).

There are many commercially available, custom-fabricated wheelchair seat cushions on the market, but not one of them has pressure ulcer prevention capacity for all individuals who have sustained SCI (DeLateur et al., 1976; Krouskop et al., 1983). The primary purpose of cushion use is to reduce excessive pressure over the bony prominences and thereby aid in the prevention of pressure ulcer formation. The initial cushion prescribed for an individual may not be appropriate over the lifetime of the user (Garber, 1985; Krouskop et al., 1983) and should be reassessed on a frequent basis for function, fit, and condition. Cushion selection should be based on a combination of clinical knowledge, pressure mapping, skin tolerance history, history of pressure ulcers, and other individual characteristics. Factors to be considered during a cushion evaluation include pressure-redistribution and shear-reducing qualities, comfort, postural support, functional activity level, ADL

performance, heat-reducing properties (Knox et al., 1994), adaptability, cost, care, and maintenance needs (Krouskop et al., 1983). Because no cushion universally produces acceptable pressure redistribution, individualized assessments of interface pressures, tissue tolerance, and skin assessment are essential for maximum protection from pressure ulcer risk (Burns & Betz, 1999). After examining the variety of wheelchair cushions available, health-care professionals should determine one that matches the individual's needs. As with bed supports, the use of donut-shaped ring cushions should be avoided. Sitting-acquired pressure ulcers are caused by chronic sitting associated with central nervous system disorders. Most manufacturers use the interface pressures between the patient's body and the sitting surface as the criterion for support surfaces for beds and wheelchair cushions. This is typically compared to the capillary closing pressure of 32 mm Hg. This criterion ignores the possibility that compression forces in the muscle-bone interface can reach up to 300 mm Hg at the ischial tuberosities (Gefen, 2007). There is no valid justification for the use of an arbitrary threshold for applied interface pressure. The important factors are the combination of time, pressure and substrate, i.e. tissue resilience.

The first study to quantify interface shear stress, interface pressure, and cushion horizontal stiffness was done by Akins et al. (2011). In evaluating 21 commercial wheelchair seat cushions, they found that interface shear stress increased significantly with increased displacement and that viscous fluid cushions resulted in the least amount of interface shear stress, followed by air cell, elastic foam, and honeycomb-type cushions. But even the best cushion does not alleviate the need for pressure redistribution behaviors. Pressure-redistributing cushions include foam, fluids, air, gel, thermoplastic cellular matrix (honeycomb), as well as combinations of these materials. Cushion covers are specifically designed to work with a cushion and should not be substituted by pillowcases, towels, plastic bags, or other cover surfaces.

FOAM CUSHIONS

Two types of foam, polyurethane and latex, have been the most frequently used to fabricate cushions. Two common cell structures seen are open and closed cell foams. The open cell foams allow airflow between intertwined, perforated membranes which allow for greater air ventilation. Open cell foams are more likely to absorb fluids making them more difficult for

cleaning and hygiene. Closed cell foams have internal structures that are encapsulated in a membrane therefore are more dense with less air flow. They are typically used for stable bases for other cushioning materials. Viscoelastic foam and matrix have high viscosity and exhibit slow accommodation with load duration. They have "memory" and return back to their original non-compressed state in a slow fashion, which is their hallmark trait. They present with good envelopment and improved thermal qualities as compared to polyurethane or latex foams. Foam cushions are available in either a flat or contoured design. More pliable softer foam will wrap around the buttocks and develop more contact with body contours (enveloping). This will result in a larger contact area and a more uniform distribution of pressure. Some foam material, however, may be too soft and may result in bottoming out, or sinking in too deeply and totally compressing the foam, resulting in increased interface pressures. Persons who use foam cushions should check the ability of the foam to recover its shape when not in use. If it appears compressed, the foam is fatigued and will no longer redistribute the patient's weight. Custom contoured foam cushions are more effective than flat foam cushions in achieving the lowest interface pressures and the most stable base of support in persons with SCI (Brienza et al. (1998 and 1999; Sprigle et al. 1990). In addition to custom contoured seat cushions, there are cushions custom designed for complete offloading of a bony area with redistribution at another area. These cushions are effective for those patients who are not able to achieve good immersion or distribution on commercially available cushions.

AIR CUSHIONS

Air-filled cushions are made of a sealed compartment membrane that holds air. An air-filled cushion may have one singular air chamber or may be divided into multiple compartments to allow for air flow movement. These types of cushions allow for pressure to move from high to low pressure areas of a person's body. Air-filled cushions allow for sinking immersion (floating) of the buttocks into the cushion, with increasing interface surface of the same tissue pressure. Initial adjustments of inflation pressure are important to establish proper immersion of the body into the cushion. Ongoing maintenance and vigilant assessment of cushion condition, including inflation level, are required to ensure adequate pressure redistribution. Bottoming out must be avoided to prevent a rapidly forming ulcer (Remsberg and Bennett, 1997). Postural

control must be assessed due to the inherently unstable nature of air cushions to horizontal forces, affecting balance, function, and body stability. Advantages of air-filled cushions include being lightweight, easy to clean, effective for many people, slower to deteriorate, and reduction of shear and peak pressures. The disadvantages are a possibility of puncturing, the need for maintenance, difficulty of repair, and postural instability enhancement of postural deformities (Garber and Krouskop (1997)). Many factors influence both the maximum pressure at the buttocks/seat interface as well the overall pressure distribution. Cushion geometry (size, shape, air capacity, material, cover, actual air pressure) and patient characteristics (body weight; size and shape of buttocks; posture; spinal deformities, such as scoliosis; sitting balance; level; and completeness of the SCI) and the wheelchair. Using an interface pressure mapping system to change the cushion inflation pressure individually for each patient may optimize the effectiveness of the air-filled cushion to reduce the risk of pressure ulcers. Hamanami (2004) used a interface pressure mapping system to determine the effectiveness of an air-filled cushion, reporting that optimal reduction in interface pressure was just before bottoming out on the cushion.

FLUID-FILLED CUSHIONS

Fluid-filled cushions tend to lessen horizontal motion of the pelvis while conforming to body contours and to reduce peak pressure over bony prominences. When an individual with SCI is sitting and actively moving, it is not unusual for layers of buttock tissue to be sliding over one another producing shear forces. Some researchers believe that fluid-filled (gel) cushions are more effective in reducing these shearing forces at the buttock-cushion interface by conforming and reducing the horizontal forces exerted on a cushion. Fluid-filled cushions may not always provide the lowest interface pressure, and therefore the limitations must be considered in the selection process (Nixon, 1985). These cushions have good dampening and thermal properties and provide a more stable base of support. They may be affected by external temperatures particularly cold weather temperatures where they may freeze. Fluid-filled cushions usually are covered by an easy-to-clean material, are effective with a wide range of users, promote more uniform distribution of pressure, and provide better skin temperature control (Garber and Krouskop, 1997). Viscous cushions can be heavier than other cushion mediums

although recent technological advances in design have addressed some of these weight issues. Fluid encased within its container can flow to areas under the pelvis where there is less pressure being exerted on the cushion surface, therefore allowing for a bottoming out condition to develop under the areas of greatest pressure. This shifting of the fluid from high to low pressure areas of the cushion may lead to bony prominences coming in contact with the harder cushion base that supports the fluid pad. It is important for the fluid to be kneaded and redistributed evenly across the cushion base prior to its use in order to avoid this occurrence.

GEL

Gel products are made of silicone, polyvinyl chloride used alone or in combination with foam. Gels have been found to be effective in preventing shear. Gel surfaces are easy to clean, but they tend to be heavy and difficult to repair. Because they lack airflow for moisture control, they may increase skin temperature after periods of unrelieved sitting.

CELLULAR MATRIX (HONEYCOMB)

Thermoplastic elastomer materials are seen in "honeycomb-like" cushions, which flex when pressure is applied to its surface to mold to the person's shape. These cushions have good resiliency and allows for good air flow between the open cells of the cushion. This increased air flow helps to wick moisture away from the body-cushion interface (Sprigle, 2001). The cushions can be constructed with different amounts of structural support and stiffness. They are lightweight and easy to clean. They must be visually inspected to detect collapse of the honeycomb-like structures that indicate deterioration with continued use.

COMBINATION CUSHIONS

Recently, a number of cushions have been developed combining a variety of materials and designs. Some use foams of various densities, stiffness, and viscoelasticity. Others use combinations of gel, air, viscous fluid and foam materials. These designs may incorporate cutouts, inserts, and modular components. The combinations are usually intended for rapid individualization of the cushion to the user in the clinical setting. This category of cushions tends to provide good envelopment and thermal properties, pressure redistribution and enhanced dynamic stability to the user. With the use of a dense base with pressure redistribution modular components imbedded, more postural asymmetry can be addressed to attempt to normalize the

pelvis and distribute pressure across a greater surface area.

Findings from a study using persons without SCI determined that when a cushion base with firm thigh support is used, lowering the footrest length as much as possible causes a levering motion by lifting the pelvis thereby reducing ischial tuberosity pressures (Gilsdorf, et al., 1990).

DYNAMIC (ACTIVE SUPPORT) CUSHIONS

A dynamic cushion system has bellows under a cushion surface base that uses a recycling air pump powered by a battery to inflate and deflate on a sequentially set pattern to increase or decrease pressure to the sitting surface. Historically, dynamic cushions have not been used widely because of their added weight, particularly when used on manual wheelchairs; reliance on a power source for activation; the need for battery recharging; and cost. Developments in technology have allowed for many of these concerns to be addressed with smaller, lighter weight components. Most of these units provide a cycle of alternating rows or groups of air cells inflating and deflating at a predetermined cadence, typically of 4 to 5 minute duration (Ho & Bogie, 2007). There is little research establishing the most effective cycle duration and pressure variations for clinical use. For users who find it difficult to remember to perform a weight shift maneuver, these dynamic alternative air cushions may help achieve the necessary offloading of pressure to facilitate pressure ulcer healing.

ADDITIONAL CONSIDERATIONS

A cushion will work well only if properly used, maintained, and replaced. As individuals age, changes in the skin may increase vulnerability to pressure ulcer development: Research indicates that the skin tends to become thinner (Knox et al., 1994; Waterlow, 1996). For this reason, a routine cushion reassessment, every two to three years, is recommended to ensure that the currently used cushion is appropriate and to determine if a change is indicated (Garber, 1985; Salcido et al., 1996).

Wheelchair users need to follow a continuous inspection and maintenance schedule for their wheelchairs and seat cushions. Cushions should be inspected and maintained regularly regardless of the brand or model of the support surface. Foam, gel, thermoplastic, and viscous fluid cushions should be checked monthly, and air cushions weekly or earlier should any problems develop. The cover should be washed and inspected regularly. If the cushion is custom contoured to the patient's skeletal structure, it

should fit properly on the wheelchair in the right direction. The cushion should be evaluated for tears, holes, or flaking in the foam. The covers should be inspected to ensure that the nonskid surface is not worn or torn. Cracked or torn covers and cushion base, and a fluid leak, indicate a need for replacement. Components and seating structures of wheelchairs deteriorate with use and age. Decomposition of wheelchair supporting structures can have an effect on the support of the body and may affect pressure redistribution qualities of the wheelchair thus require attention and replacement on a regular basis. Persons with SCI have complex medical and functional needs necessitating customized medical equipment. Their needs are best met when assessed by clinicians who employ a holistic, comprehensive team approach including education and training of the person and their caregivers (Coggrave & Rose, 2003). Consultations with persons with specific knowledge of complex seating and positioning equipment should be sought in order to achieve optimal outcomes.

Support Surfaces for Bathing and Toileting

25. Prescribe padded toileting and bathing durable medical equipment items for pressure redistribution and skin protection during use.

(Scientific evidence—III; Grade of recommendation—C; Strength of panel opinion—Strong)

In addition to the wheelchair seating surface, frequently used bathroom equipment, such as shower commode chairs, transfer bath benches, shower benches, raised toilet seats, and others, should also be evaluated for adequate padding of these sitting surfaces. When an individual with SCI is seated in a wheelchair, shower commode chair, or any other seating surface, the weight of the body is concentrated over small skin surface areas, which can lead to high localized pressures. Management of bladder or bowel routines may take duration of time that exceeds acceptable local tissue load tolerance, which, in turn, will more quickly contribute to tissue damage. Research studies have reported defecation time from 45 minutes to 2 hours in spinal injured persons with the goal being of many individuals with SCI to complete bowel care in less than 1 hour (Davis et al., 1986; Nelson et al., 1993). Performing part of the bowel routine in bed to minimize sitting on the closed ring of the toilet seat will decrease sitting time on the toilet. Place suppository and wait for it to work while

positioned in bed, and then transfer to the toilet to evacuate bowel. A comprehensive approach for skin care must include addressing these equipment item surfaces, which are frequently ignored when identifying culprits contributing to skin ulceration. As with other mobility equipment, these items must be inspected on a regular basis for signs of deterioration and replaced for continued quality performance. Utilizing interface pressure mapping of padded durable medical equipment can provide a higher level of scrutiny for these equipment surfaces. Employing interface pressure mapping with this equipment will raise the individual's awareness for recognizing that pressure management is just as important with these devices as with their wheelchair cushioning. It is important to work with therapists and nurses who are familiar with bowel care and pressure redistribution. It is important that the bathroom equipment used by persons with SCI is prescribed by professionals who understand the pressure redistribution qualities of these durable medical equipment items.

Other Support Surfaces

26. Prescribe skin protection devices and pressure redistribution systems for use with recreational equipment, other motorized or manually powered vehicles, and specialty wheelchairs.

(Scientific evidence–N/A; Grade of recommendation–N/A; Strength of panel opinion–Strong)

The concept of using a seating system that redistributes pressure, minimizes shear, provides comfort and stability, reduces heat and moisture, and enhances functional activity does not only apply to wheelchairs. This concept should be applied for seating and positioning on any piece of recreational equipment or vehicle, whether it is an automobile, snowmobile, or tractor, as well as specialty sport wheelchairs or other conveyance, such as a handcycle. The concept should also apply to any prescribed orthosis, exoskeletal device, or standing device.

Future Research

A comprehensive review of the research literature on the prevention and treatment of pressure ulcers after SCI was completed in 1998 and again in 2013 (in order to update changes in the field). To the extent possible, the recommendations in this clinical practice guideline were based on the findings reported in these reviews. Improvements in prevention and treatment require future research. Potential research questions include the following:

- What is the risk threshold that should determine recommendation of a specific type of pressure redistribution support surface for a person with SCI?
- Can a universally accepted validated measure of pressure ulcer risk be developed and validated for a spinal cord injury?
- What are the biomarkers of pressure ulcer formation?
- Can noninvasive imaging techniques be developed and validated for early pressure ulcer detection?
- What are biomarkers of pressure ulcer healing?
- What is the effectiveness of the use of negative pressure wound therapy or skin substitutes as treatments for pressure ulcers in persons with SCI?
- What is the effectiveness of the use of stem cells, tissue derived growth factors, or platelet-rich plasma as treatments for pressure ulcers?
- Which dressing is the most effective for a particular category/stage and condition of a wound?
- What is the most effective means for teaching pressure ulcer prevention and treatment?
- What is the optimal pathway for conservative management of a pressure ulcer?
- What is the optimal pathway for surgical management of a pressure ulcer?
- What types of outpatient programs are effective in reducing pressure ulcer recurrence among persons with spinal cord injury?

References

The following list of references includes all sources used by the guideline development panel in supporting topic recommendations. It provides the level of scientific evidence (I-V or N/A) for each graded article. A graded article is one that was evaluated by the methodologists to determine whether it met inclusion criteria established by the panel. If an article is labeled "Scientific Evidence—N/A," it was evaluated by the methodologists but did not meet the level of evidence criteria. If a citation is not labeled, it was not evaluated by the methodologists. Citations labeled N/A or unlabeled are included because they were considered by the panel to enhance the understanding of the guideline.

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Glossary

amyloidosis: a disease characterized by extracellular accumulation of amyloid in various organs and tissues; may be primary or secondary

autonomic dysreflexia: also known as hyperreflexia, an uninhibited sympathetic nervous response to a variety of noxious stimuli occurring in individuals with spinal cord injury at the thoracic 6 (T6) level and above.

closed ulcers: a full-thickness wound that has closed by secondary intention.

collagen type VII biosynthesis: the formation, by the cells, of type VII collagen, which is found principally in basement membrane and anchoring fibrils of the epithelium.

cytokine-induced inflammatory state: an injury, infection, or wound, such as a pressure ulcer, that results in the release of cytokines by cells. Cytokines activate the systemic inflammatory state, characterized by specific changes in nutrient metabolism, nutrient requirements, and body composition.

debridement: excision of devitalized tissue and foreign matter from a wound.

Doppler fluxmetry: a method of measuring the flow of liquids in tissue with blood cells reflecting sound waves, used in measuring velocity of flow.

electrical stimulation: a modality that delivers a therapeutic dosage of electrical charge (200–800 microcoulombs) to wound tissues to accelerate closure of the wound.

enteral nutrition: the provision of nutrients via the gastrointestinal tract. Oral enteral nutrition is taken through the mouth; tube enteral nutrition is the delivery of nutrients directly through a tube inserted into the stomach, duodenum, or jejunum.

epithelialization: formation of epithelium over a denuded tissue surface.

evidence-based guidelines: clinical practice guidelines that have been developed using research findings that have been graded for scientific strength.

exudate: any fluid that passes out of a body structure or tissues because of injury or inflammation.

grading of evidence: a standardized method for evaluating the strength of research literature used in development of a clinical practice guideline or other evidence-based document.

granulation: the formation of minute, rounded, fleshy connective tissue projections and capillary buds on the surface of a wound, ulcer, or inflamed tissue surface in the process of healing.

heterotopic ossification: abnormal bone formation in soft tissue; common locations include the hip and/or knee, which can restrict flexion to less than 90%.

hydrocolloid occlusive dressing: a wound dressing consisting of absorbent sodium carboxymethylcellulose, pectin, gelatin, and elastomer held in a fine suspension on a polyurethane foam or film backing using to hermetically seal a wound.

hydroxylation: placing of a hydroxyl group on a compound in a position where one did not exist previously.

hyperchloremic acidosis: an abnormal amount of chloride ions in circulating blood or tissue.

hypergranulation: excessive growth of granulation tissue above the cutaneous border of a wound that heals by secondary intention.

hyperreflexia—See autonomic dysreflexia

hypoalbuminemia: the below-normal concentration of albumin in the blood.

lymphopenia: a reduction in the number of lymphocytes in the circulating blood.

Marjolin's ulcer: an aggressive, well-differentiated squamous cell carcinoma, occurring in cicatricial tissue at the epidermal edge of a sinus draining underlying osteomyelitis.

methodology team: a group (usually university-based) who performs literature reviews, grades the evidence, and completes specialized studies in support of evidence-based clinical practice guideline development.

micronutrients: vitamins, minerals, and trace elements.

necrosis: pathologic death of cells, or a portion of tissue or organ, resulting from irreversible damage.

nonblanchable erythema: redness of the skin that persists when fingertip pressure is applied; a symptom of a stage I pressure ulcer.

paraplegia: impairment or loss of motor and/or sensory function in the lower extremities due to damage of the neural elements within the thoracic, lumbar, or sacral segments of the spinal cord.

parenteral feedings: the provision of nutrients intravenously. Peripheral parenteral nutrition is delivered through small peripheral veins; central or total parenteral nutrition is delivered through a large central vein, usually the superior vena cava.

periulcer maceration: maceration of the skin surrounding the ulcer.

poiklothermia: capable of existence and growth in mediums of varying temperatures.

sinus tracts: blind ending tracts that open onto the epithelial surface; may indicate presence of a foreign body or abscess located in the deep tissues.

tetraplegia: impairment or loss of motor and/or sensory function in all four extremities due to damage of the neural elements within the cervical segments of the spinal cord.

transcutaneous oxygen tension: the partial pressure of oxygen in tissue beneath the skin.

undermining: a measurable opening in the sidewall of a full-thickness wound, beginning at the wound edge, running beneath the skin, and either parallel or tangential to the skin surface for a variable distance.

wound dehiscence: a bursting open, splitting, or gaping along natural or sutured lines.

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