SPINAL CORD MEDICINE

PRESSURE ULCER

CLINICAL PRACTICE GUIDELINE:

Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury:

A Clinical Practice Guideline for Health-Care Professionals

consortium for SPINAL CORD MEDICINE
CLINICAL PRACTICE GUIDELINES

Administrative and financial support provided by Paralyzed Veterans of America

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This guideline has been prepared based on scientific and professional information available in 2000. Users of this guideline should periodically review this material to ensure that the advice herein is consistent with current reasonable clinical practice.

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Contents

Foreword

Pressure ulcers are a frequent, costly, and potentially life-threatening complication of spinal cord injury (SCI). They interfere with the rehabilitation process and are a significant deterrent to participation in activities that contribute to independent, productive, and satisfying lives. Pressure ulcers result in long hospitalization, delayed community reintegration, reduced quality of life, and loss of self-esteem. The etiology of pressure ulcers is complex and multidimensional and includes 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 noncompliance with acknowledged preventive behaviors. Pressure ulcer prevalence rates range from 8 percent 1 year following the onset of SCI to 33 percent for community-resident individuals with SCI.

The prevention and management of pressure ulcers are processes that are inextricably linked across the continuum of care of individuals with SCI. However, current prevention programs, specifically hospital-based education interventions, have had limited success in reducing the occurrence of pressure ulcers. Furthermore, despite the plethora of education and treatment programs and protocols described in the literature, few have been evaluated 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/her home and community.

In the last 40 years, a number of scientific studies have advanced knowledge of the factors that contribute to the formation of pressure ulcers and have provided a basis for improving preventive techniques. However, much of this research has focused on populations other than individuals with spinal cord injury. The purpose of this clinical practice guideline is to describe effective strategies for identifying risk and reducing the incidence, prevalence, and recurrence of this lifelong complication of SCI. The recommendations in this guideline cover a broad spectrum of issues that have been addressed by the multidisciplinary pressure ulcer clinical practice guideline development panel. The significant constructs of this problem are prevention, including risk factors, risk assessment, risk assessment tools, and prevention strategies; nutrition; assessment of the individual with a pressure ulcer and of the ulcer itself; nonsurgical and surgical treatments and their complications; and support surfaces and positioning for managing tissue loads.

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 treatment 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 a consumer guide will be developed shortly after publication of the clinical guideline. 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 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
Panel Chair

Preface

Pressure ulcers have plagued mankind since antiquity. Rowling (1961) observed evidence of pressure ulcers in his study of Egyptian mummies. In ancient China, soldiers used gunpowder to cleanse their battle wounds. In 16th Century Europe, the French surgeon Ambrose Paré described pressure ulcers as "an incurable malady" which could be helped only with "rest, exercise and a good diet" (Levine, 1992). Pare' was an advocate of humanistic treatment of the "bed sore problem," emphasizing psychological and spiritual wellbeing (Levine, 1992). Likewise, traumatic spinal cord injury has a history as old as mankind. Perhaps the earliest description of SCI is found in a 5,000-year-old papyrus in which an Egyptian physician not only characterized the specific symptoms of a complete cervical cord lesion, but also commented on the bleak prognosis of such individuals by admonishing that it is "an ailment not to be treated" (Guttman, 1976). Historically, it wasn't until the end of World War II that the first rehabilitation programs specifically for people with SCI were developed. From pioneers such as Guttmann, Monroe, Rusk, Kessler, and Covalt, as well as the policies and programs of the United States Veterans Administration, emerged a new philosophy of care for individuals with SCI (Clifton et al., 1985; Monroe, 1943). Improved medical care, new technologies, and the development of antibiotics provided innovative interventions for preventing and treating pressure ulcers.

From initial trauma through discharge from the rehabilitation hospital to return to home and community, the person with a spinal cord injury is at risk for pressure ulcer development. When pressure ulcers develop during the acute and rehabilitation phases of care, they delay the achievement of rehabilitation goals; when they occur after reintegration into the community, they result in loss of independence and participating in vocational, educational, and leisure activities. Additionally, pressure ulcers result in prolonged periods of inactivity, consumption of financial resources, and disruption of the lives of the entire family. Prevention strategies can reduce the risk of pressure ulcers but often require the active participation of the person and significant others in the process.

Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury is the 6th in the series of evidence-based clinical practice guidelines developed by the Consortium for Spinal Cord Medicine under the auspices of Paralyzed Veterans of America. The Consortium's mission is to enhance the quality, appropriateness, and cost-effectiveness of care delivered to individuals with SCI through evidence-based practice. The purpose of this guideline, therefore, is to provide guidance and assistance in the decisions required to restore health, independence, control, and self-esteem to people with spinal cord injury. This document provides the conceptual framework within which to develop effective strategies for preventing and treating pressure ulcers.

In preparing this guideline, the panel reviewed an extensive body of literature. It should be noted that most pressure ulcer literature does not focus on persons with SCI. Therefore, where no SCI-specific literature existed with respect to a particular recommendation, the findings from studies of other populations were reported. The panel took a new step in this document in that the reference list provides the "scientific evidence (I–V)" for each graded article. In addition, it lists all references used, including those that were not graded but were essential to the guideline as a whole. This approach puts more resources in the hands of the care providers and, hopefully, will stimulate future researchers and health-care professionals to examine the topic areas that have not yet been subject to rigorous scientific study.

Each clinical practice guideline represents the collaboration of many individuals. Congratulations are in order to the panel members for their outstanding work and to its chair, Susan L. Garber, M.A., O.T.R., F.A.O.T.A., for her exemplary

leadership as the panel prepared this document. This document could not exist without the support of Paralyzed Veterans of America (PVA) and its Health Policy Department. J. Paul Thomas and Dawn M. Sexton provided the guidance and organizational structure for the successful completion of this guideline. Appreciation is extended to all of the reviewers of this guideline who contributed invaluable time and expertise. They have ensured that the content reflects the best practices available in the field.

Kenneth C. Parsons, MD
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Acknowledgments

The panel chair and members of the guideline development panel on pressure ulcer prevention and treatment wish to express special appreciation to the individuals and professional organizations who are members of the Consortium for Spinal Cord Medicine and to the expert health-care professionals, researchers, and health-care providers who critiqued the draft documents. Special thanks go to the consumers, advocacy organizations, and the staffs of the numerous medical facilities and spinal cord injury rehabilitation centers who contributed their time and expertise to the development of the guideline.

Andrea K. Biddle, Ph.D., and her colleagues at the Department of Health Policy and Administration, University of North Carolina at Chapel Hill, served as consultant methodologists. They masterfully conducted the initial and secondary-level literature searches, evaluated the quality and strength of the scientific evidence, constructed evidence tables, and graded the quality of research for all identified literature citations.

Members of the Consortium Steering Committee, representing 19 professional, payer, and consumer organizations, were joined in the guideline development process by 46 expert reviewers. Their clinical analyses and thoughtful comments identified additional supporting evidence from the scientific literature, which was incorporated into this document. The quality of the technical assistance from these dedicated reviewers contributed significantly to the professional consensus building that is achieved through the guideline development process. Attorney William H. Archambault, of Goodman, West & Filetti, PLLC, in Charlottesville, Virginia, conducted a comprehensive analysis of the legal and health policy issues associated with this complex, multifaceted topic.

The Pressure Ulcer Prevention and Treatment clinical practice guideline panel is grateful for the many administrative, organizational, and technical support services provided by various departments of the Paralyzed Veterans of America. The panel recognizes J. Paul Thomas and Dawn M. Sexton in the Consortium coordinating office and Health Policy Department for their untiring efforts in guiding the panel through the guideline development process; Fred Cowell in the Health Policy Department for his cogent comments reflecting the perspective of consumers; James A. Angelo and Patricia E. Scully in the Communications Department for their guidance in writing, editing, and indexing; medical editor Joellen Talbot for her excellent technical review and editing of the clinical practice guideline; and graphic designer Sarah Ornstein for designing and creating art for the document. Appreciation is expressed for the steadfast commitment and enthusiastic advocacy of the entire PVA board of directors and of PVA's senior officers, including National President Homer S. Townsend, Immediate Past President Kenneth C. Huber, Executive Director Gordon H. Mansfield, Deputy Executive Director John C. Bollinger, and John L. Carswell, Associate Executive Director for Health Policy. Their generous financial support has made the consortium and its guideline development process a successful venture.

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Summary of Recommendations

Prevention

Risk Factors, Risk Assessment, and Risk Assessment Tools

1. Conduct comprehensive, systematic, and consistent assessment of pressure ulcer risk factors in individuals with spinal cord injury.
   - 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.
   - Use clinical judgment as well as a risk assessment tool to assess risk.
   - Assess demographic, physical/medical, and psychosocial risk factors associated with pressure ulcer prevention.

Prevention Strategies

2. Implement pressure ulcer prevention strategies as part of the comprehensive management of acute SCI and review all aspects of risk when determining prevention strategies.
   - Avoid prolonged positional immobilization whenever possible.
   - Institute pressure relief as soon as emergency medical condition and spinal stabilization status allow.
   - Initiate intraoperative pressure reduction strategies.
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:
   - Ischii
   - Sacrum/coccyx
   - Trochanters
   - Heels
4. Turn or reposition individuals with SCI initially every 2 hours in the acute and rehabilitation phases if the medical condition allows.
   - Eliminate stretching and folding of soft tissues and prevent shearing when individuals are repositioned.
   - Avoid positioning individuals who are sidelying in bed directly on their trochanter.
5. Evaluate the individual and his/her support environment for optimal maintenance of skin integrity.
   - Apply pressure-reducing support surfaces preventively to protect soft tissues from bruise and injury.
   - Prevent moisture accumulation and temperature elevation at the support surface-skin interface.
   - Apply pillows and cushions to bridge contacting tissues and unload bony prominences; do not use donut-type devices.
   - Establish a mechanism to follow up on equipment performance specific to pressure ulcer prevention (support surfaces for the bed and wheelchair) and determine if changes in medical or health status have altered the effectiveness of the support surface.
6. Provide an individually prescribed wheelchair and pressure-reducing seating system.
   - Establish and initiate a specific pressure relief regimen within the individual's capability.
   - Employ a power weight-shift system when manual pressure relief is not possible.
7. Implement an ongoing exercise regimen for the medically stable SCI individual to promote maintenance of skin integrity, increase strength of paretic and nonparalyzed muscles, improve cardiovascular endurance, and prevent fatigue and deconditioning.
8. 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.

Nutrition

9. Assess nutritional status of all SCI individuals on admission and as needed, based on medical status, including:
   - Dietary intake
   - Anthropometric measurements
   - Biochemical parameters (prealbumin, total protein, albumin, hemoglobin, hematocrit, transferrin, and total lymphocyte count)

10. Provide adequate nutritional intake to meet the individual's needs, especially:
    - Calories (or Energy)
    - Protein
    - Micronutrients (zinc, vitamin C, vitamin A, and vitamin E)
    - Fluids
11. Implement aggressive nutritional support measures if dietary intake is inadequate or if an individual is nutritionally compromised.

Assessment Following Onset of a Pressure Ulcer

Assessment of the Individual with a Pressure Ulcer

12. Perform an initial comprehensive assessment of the individual with a pressure ulcer, to include:
    - Complete history
    - Physical examination and laboratory tests
    - Psychological health, behavior, cognitive status, and social and financial resources
    - Availability and utilization of personal care assistance
    - Positioning, posture, and related equipment

Assessment of the Pressure Ulcer

13. Describe in detail an existing pressure ulcer. Include the following parameters:
    - Anatomical location and general appearance
    - Size (length, width, depth, and wound area)
    - Stage
    - Exudate/odor
    - Necrosis
    - Undermining
    - Sinus tracts
    - Infection
    - Healing (granulation and epithelialization)
    - Wound margins/surrounding tissue

Treatment

Nonsurgical

CLEANSING

14. Cleanse pressure ulcers at each dressing change.
    - Use minimum mechanical force when cleansing with gauze, cloth, or sponge.
    - Use enough irrigation pressure to enhance cleansing without causing trauma to the wound.
    - Use normal saline or wound cleansers.
    - Avoid antiseptic agents.
    - Consider hydrotherapy for ulcers containing large amounts of exudate and necrotic tissue.

DEBRIDEMENT

15. Debride devitalized tissue from pressure ulcers using a method appropriate to the ulcer's status and the individual's condition and goals.
    - Debride areas in which there is eschar and devitalized tissue.

DRESSINGS

16. Use dressings that will keep the ulcer bed continuously moist and the surrounding intact skin dry.
    - Use a dressing that controls exudate, but does not desiccate the ulcer bed or macerate surrounding tissue.
    - Loosely fill pressure ulcer cavities with dressing material to avoid dead space; avoid overpacking the ulcer.
    - Monitor the placement of all dressings, especially those in anatomical areas in which they are difficult to keep intact.
    - Perform dressing changes on a specific schedule based on assessment of the individual, the ulcer, and the condition of the dressing. Consult the dressing manufacturer's package insert for general information about the frequency of dressing changes.

ELECTRICAL STIMULATION

17. Use electrical stimulation to promote closure of stage III or IV pressure ulcers combined with standard wound care interventions.

REASSESSMENT

18. Monitor and assess the pressure ulcer on a consistent, ongoing basis to determine the adequacy of the plan of care.
    - Monitor the pressure ulcer at each dressing change.

- Document ulcer assessment at least weekly and every time the condition of the pressure ulcer or the individual changes.

19. Modify the treatment plan if the ulcer shows no evidence of healing within 2 to 4 weeks.
    - Review individual risk factors when assessing the healing of pressure ulcers.
    - Evaluate healing progress using an instrument or other quantitative measurements.

Surgical

20. Refer appropriate individuals with complex, deep stage III pressure ulcers (i.e., undermining, tracts) or stage IV pressure ulcers for surgical evaluation. When surgery is indicated, include the following tenets of surgical treatment:
    - Excising of ulcer, surrounding scar, bursa, soft tissue calcification, and underlying necrotic or infected bone
    - Filling dead space, enhancing vascularity of the healing wound, and distributing pressure off the bone
    - Resurfacing with a large regional pedicle flap, with suture line away from the area of direct pressure, and one that does not encroach on adjacent flap territories
    - Preserving options for future potential breakdowns

PREOPERATIVE CARE

21. Assess, treat, and optimize the following factors preoperatively:
    - Local wound infection
    - Nutritional status
    - Bowel regulation
    - Severe spasms and contractures
    - Comorbid conditions
    - Previous ulcer surgery
    - Smoking
    - Osteomyelitis
    - Urinary tract infection (UTI)
    - Heterotopic ossification

POSTOPERATIVE CARE

22. Be cognizant of postoperative care procedures.
    - Position the individual in a manner that keeps pressure off a fresh surgical site.
    - Use an air-fluidized bed when pressure on the surgical flap is unavoidable.
    - Progressively mobilize the individual to a sitting position over at least 4 to 8 weeks to prevent reinjury of the ulcer or surgical site.
    - Provide subsequent patient education on pressure management and skin inspection.

Complications of Pressure Ulcers

Nonsurgical

23. Identify the presence of tissue and/or bone infection.
    - Obtain quantitative tissue and/or bone cultures in ulcers not responding to routine therapeutic measures.
    - Obtain a tissue and/or bone biopsy to confirm infection, if necessary.

24. Identify the potential complications of immobility associated with pressure ulcer management and implement preventive and therapeutic measures for:
    - Nutritional deficiencies and dehydration
    - Decreased range of motion
    - Deconditioning (cardiopulmonary, cardiovascular, and musculoskeletal)

25. Manage hypergranulation tissue that may impede ulcer healing.

26. Identify the potential psychosocial impacts of pressure ulcers and immobility and provide referral for therapeutic interventions based upon the individual's characteristics and circumstances. Refer to appropriate resources for problem resolution, including:
    - Vocational rehabilitation services
    - Peer counseling and support groups
    - Formal psychotherapy and/or family therapy

Surgical

27. Identify potential complications of surgical intervention, including:
    - Wound dehiscence/wound separation
    - Delayed infection and abscess
    - Hematoma and seroma

Support Surfaces and Positioning for Managing Tissue Loads

Bed Positioning

28. Use bed-positioning devices and techniques to prevent and treat pressure ulcers. Use devices and techniques that are compatible with the bed type and the individual's health status.

- Avoid positioning individuals directly on a pressure ulcer.
- Avoid positioning individuals directly on the trochanter.
- Use cushions and positioning aids to relieve pressure on 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.
- Limit the amount of time the head of the bed is elevated.
- Develop, display, and use an individualized positioning regimen and repositioning schedule.

Bed Support Surfaces

29. Use pressure-reducing bed support surfaces for individuals who are at risk for or who have pressure ulcers.

- Select a static support surface for individuals who can be positioned without weight bearing on an ulcer and without bottoming out on the support surface.
- Select a dynamic support surface if the individual cannot be positioned without pressure on an ulcer, when a static support surface bottoms out, if there is no evidence of ulcer healing, or if new ulcers develop.
- Use low-air-loss and air-fluidized beds in the treatment of pressure ulcers if one or more of the following conditions exist:
  - Pressure ulcers on multiple turning surfaces
  - Compromised skin temperature and moisture control in the presence of large stage III or IV pressure ulcers

Wheelchair Positioning

30. Prescribe wheelchairs and seating systems according to individualized anthropometric, ergonomic, and functional principles.

- Obtain specific body measurements for optimal selection of seating system dimensions.
- Measure the effects of posture and deformity on interface pressure distribution.
- Prescribe a power weight-shifting wheelchair system for individuals who are unable to independently perform an effective weight shift.
- Use clinical judgment as well as objective data in determining the compatibility of the individual's shape with the seating system.

31. Evaluate the individual's postural alignment, weight distribution, balance, stability, and pressure reduction capabilities to establish a proper sitting schedule.

- Avoid positioning the wheelchair-seated individual directly on a pressure ulcer.
- Allow limited sitting in individuals capable of performing weight shifts every 15 minutes.
- Reposition the wheelchair-seated individual at least every hour; if this is not possible and the individual is unable to perform weight shifts, return the individual to bed.

Wheelchair Support Surfaces

32. Use appropriate wheelchair cushions with all individuals with SCI.

- Inspect and maintain all wheelchair cushions at regularly scheduled intervals.

The Consortium for Spinal Cord Medicine

Seventeen organizations, including PVA, joined together to form a consortium in June 1995 to develop evidence-based clinical practice guidelines in spinal cord medicine. Today, nineteen organizations compose the consortium. A steering committee governs its operation, leads the guideline development process, identifies topics, and selects panels of experts for each topic. The steering committee is composed of one representative with clinical practice guideline experience from each consortium member organization. PVA provides financial resources, administrative support, and programmatic coordination of consortium activities.

After studying the processes used to develop other guidelines, the consortium steering committee unanimously agreed on a new, modified, scientific evidence-based model derived from the Agency for Health Care Policy and Research (AHCPR), now called the Agency for Healthcare Research and Quality (AHRQ). The consortium's model is interdisciplinary, in that it reflects the multiple information needs of the spinal cord practice community; it is responsive, with a 12-month timeline for completion of each set of guidelines; and it is reality-based, in that the time and energy of the busy health-care professionals who serve as panel members and expert reviewers are used efficiently and effectively.

The consortium's approach to the development of evidence-based guidelines is both innovative and cost-efficient. The process recognizes the specialized needs of the national spinal cord medicine community, encourages the participation of both payer representatives and consumers with spinal cord injury, and emphasizes utilization of graded evidence available in the international scientific literature.

The Consortium for Spinal Cord Medicine is unique to the clinical practice guideline development field. It employs highly effective management strategies based on the availability of resources in the health-care community; it is coordinated by a recognized national consumer organization with a reputation for providing effective service and advocacy for people with spinal cord injury and disease; and it includes third-party and reinsurance payer organizations at every level of the development and dissemination processes. The consortium expects to initiate work on several topics per year, with evaluation and revision of previously completed guidelines as new research demands.

Guideline Development Process

The guideline development process adopted by the Consortium for Spinal Cord Medicine consists of twelve steps, leading to panel consensus and organizational endorsement. After the steering committee chooses a topic, a panel of experts is selected. Panel members must have demonstrated leadership in the topic area through independent scientific investigation and publication. Following a detailed explication and specification of the topic by select steering committee and panel members, consultant methodologists review the international literature, prepare evidence tables that grade and rank the quality of research, and conduct statistical meta-analyses and other specialized studies, as needed. The panel chair then assigns specific sections of the topic to the panel members, based on area of expertise. Writing begins on each component using the references and other materials furnished by the methodology support group.

After the panel members complete their sections, a draft document is generated during the first full meeting of the panel. The panel employs an iterative process in document preparation to incorporate new literature citations or other evidence-based information not previously available. At this point, charts, graphs, algorithms, and other visual aids, as well as a complete list of references, are added, and the full document is sent to legal counsel for review.

After legal analysis to consider antitrust, restraint-of-trade, and health policy matters, the draft document is reviewed by clinical experts from each of the consortium organizations plus other select clinical experts and consumers. The review comments are assembled, analyzed, and entered in to a database, and the document is revised to reflect the reviewers' comments via a second panel meeting. Following a second legal review, the draft document is distributed to all consortium organization governing boards. Final technical details are negotiated among the panel chair, members of the organizations' boards, and expert panelists. If substantive changes are required, the draft receives a final legal review. The document is then ready for professional editing, formatting, and preparation for publication.

The benefits of clinical practice guidelines for the spinal cord medicine practice community are numerous. Among the more significant applications and results are the following:

- Clinical practice options and care standards
- Medical and health professional education and training
- Building blocks for pathways and algorithms
- Evaluation studies of guideline use and outcomes
- Research gap identification
- Cost and policy studies for improved quantification
- Primary source for consumer information and public education
- Knowledge base for improved professional consensus building

Methodology

Literature Search

The methodology team's strategy for finding evidence relating to the prevention and treatment of pressure ulcers in individuals with SCI closely resembles the methods recommended by AHCPR and by the National Academy of Sciences' Institute of Medicine. First, a preliminary literature search of the MEDLINE database from 1966 to the 1998 was conducted. The purpose of this initial search was to enable the methodology team to estimate the volume of literature available and to identify the main issues associated with the topic.

The results of this initial search were presented to the panel during an October 1997 conference call. The panel discussed, defined, and further clarified the guideline topic. During the conference call, the group agreed on literature search topic areas, identified the guideline's intended audience, and established inclusion and exclusion criteria for the literature searches. The panel discussed the possibility of including animal studies in the literature search, particularly in the area of skin physiology and the pathophysiology of pressure ulcers. A decision was made to review the available literature using human subjects first to determine the need for animal studies to supplement the evidence base. The literature search criteria included elderly as well as nonelderly adults. Children under the age of 13 were excluded, as were articles involving nontraumatic paralysis. The search was limited to articles published in English. Study designs employing clinical trials (randomized and nonrandomized), cohort studies, case controls, case series, and cross-over studies were included. However, case reports and "n-of-one" studies were excluded as evidence. Studies across the continuum, from point of injury through integration into the home/community, were included, as were studies including pressure ulcers at all stages and anatomical locations.

Review articles and overview articles examining pressure ulcers in individuals with SCI were identified and retrieved if pressure ulcers were the focus of discussion. It is important to note that although review articles were included, they were not intended for use as evidence for the guideline. Rather, they were used to identify "gray literature" and to cross-reference with the literature search to ensure that all relevant articles on the topic had been identified and retrieved for analysis.

Appropriate key words and Index Medicus subheadings (MeSH subheadings) identified by the panel were used to search the MEDLINE database (1966–98) and the CINAHL nursing database (1982–98). These initial literature searches concentrated on articles focusing on pressure ulcers in individuals with SCI. A second search was conducted in the MEDLINE database (1993–98) focusing on the general population with pressure ulcers. The purpose of this second search was to identify literature that had emerged since publication of the AHCPR pressure ulcer guidelines. Whenever possible, "exploded" MeSH subheadings were used, allowing the inclusion of more relevant literature than would be discovered using text word searches. Second-level searches were conducted using the major and minor MeSH subheadings retrieved from relevant articles.

Approximately 1,800 abstracts from the literature searches were reviewed, using the inclusion and exclusion criteria, for relevance to the prevention and treatment of pressure ulcers. Nearly 350 articles were deemed relevant to the guideline and were retrieved. Of these articles, more than 200 clearly met the inclusion and exclusion criteria and were slated for data extraction. Another 23 articles did not have abstracts, or their relevance was unclear, so they were retrieved for further evaluation. Approximately 50 articles were identified that did not have valid study designs but were worthy of retrieval for education and orientation purposes. Finally, relevant review articles (approximately 45 in number) were retrieved.

Standardized data extraction forms were used to extract relevant information from the articles found to meet the inclusion and exclusion criteria. Extracted information was compiled into evidence

tables according to subject area. The methodologists categorized the articles into topic areas and disseminated relevant articles and evidence tables to panel members for study and consideration. Articles identified as relevant by panel members were extracted, and supplemental evidence tables were created and disseminated. Throughout the guideline writing process, the methodologists responded to queries from the panel chair and panel members.

Evidence Analysis

GRADING THE EVIDENCE

A number of approaches exist for evaluating the quality of research studies and the evidence derived from them (Feinstein, 1985; Sackett et al., 1989). Most employ a hierarchy of evidence that places more weight on certain study designs than on others. Generally, the greatest weight is placed on randomized, controlled trials, followed by observational studies, uncontrolled case series, and finally case reports.

For all evidence presented in this report, the methodology team employed 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). These levels of scientific evidence are presented in table 1. Additionally, each study was evaluated for internal and external validity. Factors affecting internal validity (e.g., the extent to which the study provides valid information about the individuals and conditions studied) included 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; and methods of statistical analysis. External validity—the extent to which the study findings are generalizable to conditions other than the setting of the study—was evaluated through an examination of the characteristics of the study population, the clinical setting, and the environment.

If the literature supporting a guideline recommendation came from two or more levels, the number and level of the studies are reported (e.g., in the case of a guideline recommendation that is supported by two studies, one a level III, the other a level V, the "Scientific evidence" would be indicated as "III/V").

TABLE 1
Hierarchy of the Levels of Scientific Evidence

Level	Description
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.

GRADING THE RECOMMENDATIONS

Next, each of the guideline recommendations was classified, depending upon the level of scientific evidence supporting the specific recommendation. The schema used by the panel is shown in table 2 (Sackett, 1989; U.S. Preventive Health Services Task Force, 1996). It should be emphasized that these ratings, like those just described, represent the strength of the supporting evidence, not the strength of the recommendation itself. The strength of the recommendation is indicated by the language in its rationale.

Category A requires that the guideline recommendation be supported by scientific evidence from at least one properly designed and implemented randomized, controlled trial providing statistical results that consistently support the guideline statement. Category B requires that the guideline recommendation be supported by scientific evidence from at least one small randomized trial with uncertain results; this category also may include small randomized trials with certain results where statistical power is low. Category C recommendations are supported by either nonrandomized, controlled trials or by trials for which no controls were used.

If a guideline recommendation is supported by literature that crossed two categories, both categories are reported (e.g., a guideline recommendation that includes both level II and III studies would be classified as categories B/C). In situations where no published literature existed, consensus of the panel members and outside expert reviewers was used to develop the guideline recommendation and is indicated as "Expert consensus."

TABLE 2
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.

GRADING OF PANEL CONSENSUS

After deliberation and discussion of each guideline recommendation and the supporting evidence, the level of panel agreement with the guideline recommendation was assessed as either low, moderate, or strong. In this assessment, each panel member was asked to indicate his or her level of agreement on a 5-point scale, with 1 corresponding to "neutrality" and 5 representing "maximum agreement." The scores were aggregated across the panel members and an arithmetic mean was calculated. This mean score was then translated into low, moderate, or strong, as shown in table 3. A panel member could abstain from the voting process for a variety of reasons, including, but not limited to, lack of expertise associated with the particular guideline recommendation.

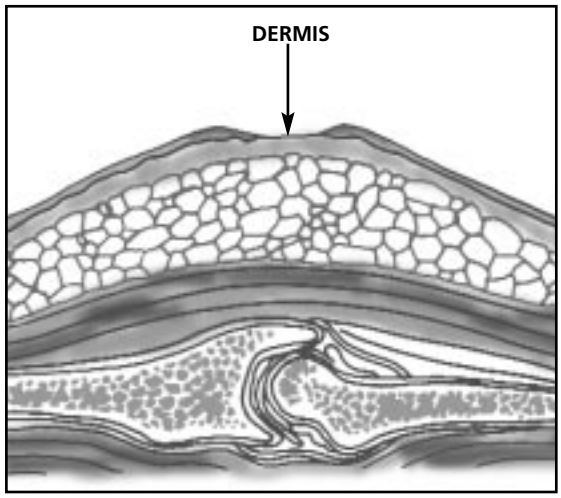
TABLE 3
Levels of Panel Agreement with the Recommendation

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

Introduction

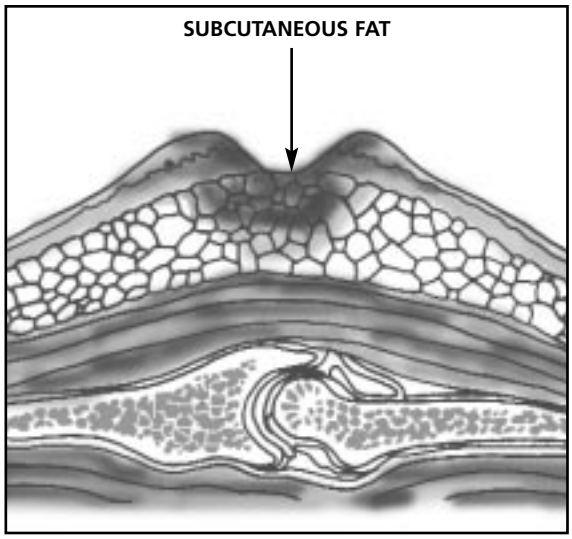
Pressure ulcers are a lifelong, serious complication of spinal cord injury. They have the potential to interfere with physical, psychological, and social well-being and to impact overall quality of life. Although mostly preventable, pressure ulcers frequently disrupt rehabilitation, educational and vocational pursuits, and community reintegration after SCI.

Pressure ulcers are defined as lesions caused by unrelieved pressure resulting in damage of underlying tissue (Bergstrom et al., 1992). They usually occur over bony prominences and are classified as stages by the degree of tissue damage observed. Staging is used primarily for the initial assessment of a pressure ulcer. Described and illustrated below are the stages of pressure ulcers (Cuddigan and Frantz, 1998):



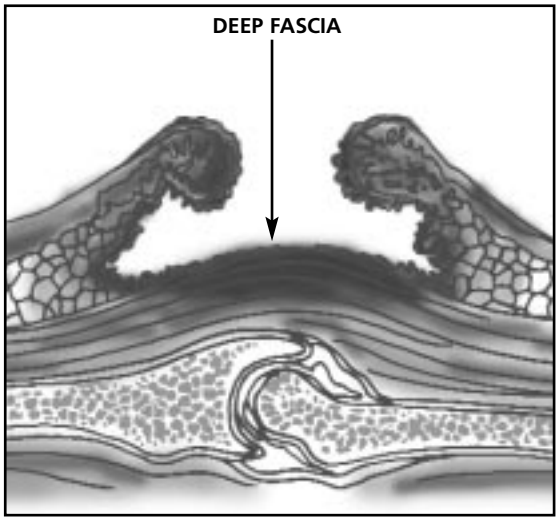
Stage I:

An observable pressure-related alteration of intact skin whose indicators as compared to an adjacent or opposite area on the body may include changes in one or more of the following: skin temperature (warmth or coolness), tissue consistency (firm or boggy feeling), and/or sensation (pain, itching). The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones, the ulcer may appear with persistent red, blue, or purple hues.



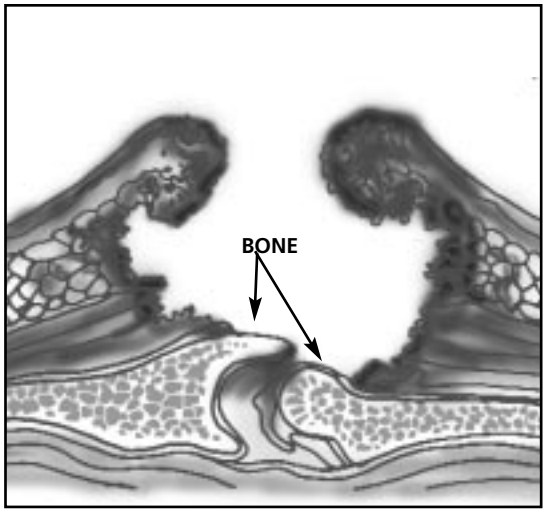
Stage II:

Partial-thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.



Stage III:

Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.



Stage IV:

Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule). Undermining and sinus tracts also may be associated with stage IV pressure ulcers.

Pressure ulcers do not progress from stage I to stage II to stage III and ultimately to stage IV. Rather, they begin deep inside the tissues, close to the bone, and erupt on the surface of the skin. Conversely, healing ulcers do not progress in reverse order of the stages. Muscle tissue is more sensitive than skin to pressure-induced ischemia (Daniel et al., 1981; Nolan and Vistnes, 1980). Clinically the skin may be discolored, but the muscle underneath may be necrotic. When eschar is present, the pressure ulcer cannot be graded accurately until the eschar is removed. Stage I pressure ulcers are not always accurately assessed, especially in people with darkly pigmented skin.

Incidence and Prevalence

The literature is replete with studies describing variables associated with the incidence (new cases appearing during a specific period of time) and prevalence (a cross-sectional count of the number of cases at a specific point in time) of pressure ulcers in people with SCI. In general, these studies categorize the variables as demographic factors, SCI-related factors, and aspects of physical and psychosocial well-being. The demographic factors that have been associated with the occurrence of pressure ulcers include age (younger or older), gender (male), ethnicity (minorities), marital status (single), and level of education (less education). Regarding SCI-related factors, pressure ulcers occur more frequently in people with more extensive paralysis and completeness of SCI, longer duration of the SCI, and less functional independence. Aspects of physical and psychosocial well-being associated with the occurrence of pressure ulcers include lack of responsibility for skin care, poor nutrition, use of tobacco, alcohol and drug abuse, low self-esteem, and feelings of dissatisfaction with life and one's activities. Confounding this literature are other reports that the occurrence of pressure ulcers is not related to age, gender, ethnicity, marital status, or employment status, nor is it related to level or completeness of SCI, tobacco use, or alcohol use.

Reliable data on the incidence and prevalence of pressure ulcers have been difficult to obtain, especially in the SCI population. This derives from a number of methodological problems that prevent generalizability of the statistics. These limitations include:

(1) the use of different classifications of pressure ulcer stages;

(2) inability to compare the varied populations presenting with or developing a pressure ulcer; and

(3) varying methods of obtaining data (i.e., direct observation versus retrospective chart review).

This results, then, in inaccurate or incomplete information that cannot be translated into developing programs to reduce the occurrence of pressure ulcers. Most of the data that are available reflect populations of individuals in acute care hospitals and skilled nursing care facilities. Few statistics exist in the literature that pertain to the actual prevalence rate of pressure ulcers in people with SCI living in the community. One exception—and the source that is perhaps the most reliable—is the database of the Spinal Cord Injury Model Systems. The Model Systems Program, sponsored by the National Institute on Disability and Rehabilitation Research (NIDRR), is a Federal extramural grant program of selected research and demonstration sites that provide exemplary, state-of-the-art care from point of injury through acute medical care, comprehensive rehabilitation, and long-term followup and health maintenance services. To be included in the research database, the individual must be admitted to the system facility within 24 hours of trauma.

Richardson and Meyer (1981) conducted a retrospective study of 549 SCI individuals in the Model Systems program and reported that the prevalence of pressure ulcers was significantly higher in nonsystem individuals compared to those

within the Model Systems. (Nonsystem individuals were those who were admitted to the hospital 72 hours or more from the time of their injury.) They also reported that level and completeness of SCI were the most important factors in calculating the prevalence. Mawson et al. (1988) reported that 32 to 40 percent of all individuals admitted to special SCI units in the United States would develop pressure ulcers during their initial hospital admission period. Young and Burns (1981a, 1981b) reported a 40-percent prevalence of pressure ulcers during initial acute care and rehabilitation in a regional SCI Model Systems facility and 30 percent in each 5-year followup. Fuhrer et al. (1993) reported a 33-percent prevalence of pressure ulcers in their population of 140 community-resident subjects. Those with ulcers were more impaired (based on the level and completeness of their SCI) and more disabled (based on the self-report version of the Functional Independence Measure (FIM)). Yarkony and Heinemann (1995) reported a pressure ulcer prevalence rate of 7.9 percent at the first annual evaluation after discharge from a Model Systems facility and 31.9 percent at 20 years post discharge. The most severe ulcers at one-year post were in the sacral area (25 percent), whereas at 2 years the most severe ulcers were in the ischial tuberosities (24.3 percent). These investigators also reported that 1.6 percent of individuals admitted to a Model Systems facility within 1 day of trauma were admitted with an ulcer, whereas 28.9 percent of individuals whose admission was delayed were admitted with a pressure ulcer. Thirty-seven percent of the pressure ulcers that developed in the Model Systems were sacral and 50 percent of the severe stages III and IV pressure ulcers were sacral. Model Systems data also indicate that individuals admitted promptly to a Model Systems facility had a lower rate of severe ulcers and lower rates generally from injury to discharge (Yarkony and Heinemann, 1995).

According to the 1998 National Spinal Cord Injury Statistical Center (NSCISC) Annual Report, 34 percent of individuals admitted to a Model Systems facility within 24 hours of SCI developed at least one pressure ulcer during acute care or rehabilitation (Yarkony and Heinemann, 1995). On followup, 15 percent had a pressure ulcer at their first annual examination, 20 percent at year 5, 23 percent at year 10, 24 percent at year 15, and 29 percent at year 20. These numbers are based on 4,065 individuals, of whom 2,971 developed pressure ulcers.

Carlson et al. (1992) studied the relation between pressure ulcer history and later pressure ulcer development. A total of 125 individuals were assessed across three time periods. Fifty-eight percent were individuals whose SCI resulted in tetraplegia. Twenty-nine percent developed ulcers during the acute phase, 3 percent during rehabilitation, and 17 percent during followup. The majority of the ulcers (81 percent) were in the pelvic region (sacrum, coccyx, ischium, and trochanter), with the remainder (19 percent) on the feet. A total of 36 percent of the individuals developed 88 ulcers during the study.

Recurrence

Niazi et al. (1997) conducted a retrospective case-controlled study on the recurrence of pressure ulcers in a population of 176 veterans with spinal cord injury. They found that there was a 35-percent recurrence rate regardless of whether the treatment was medical or surgical. Smoking, diabetes, and cardiovascular disease were associated with the highest rates of recurrence. Heilporn (1991) reported in two case studies that recurrence was related to personality and psychological well-being. Yasenchak et al. (1990) reported that people who were employed were less likely to have a recurrence of their pressure ulcer after surgery. However, Rodriguez and Garber (1994) found no relation between employment and recurrence. Disa et al. (1992) reported that younger people were more likely to have a recurrence. However, Rodriguez and Garber (1994) found no relation between age and recurrence.

Costs

Estimates of the costs of preventing and treating pressure ulcers have been difficult to obtain. Most of the data reflect the estimated treatment costs of all individuals who are reported to have had a pressure ulcer in hospitals, rehabilitation centers, and long-term care facilities. Reliable data specific to the costs of treating pressure ulcers in individuals with SCI have not been reported in the published literature. Therefore, it is necessary to extrapolate from existing information what the financial implications are for individuals with SCI.

Miller and DeLozier (1994) investigated and analyzed the costs related to pressure ulcer treatment in hospitals, nursing homes, and home care. Only stages II, III, and IV pressure ulcers were included in the analysis. The researchers reported that the total cost of treating pressure ulcers was $1.335 billion per year. Approximately 69 percent of these costs were incurred for care provided in hospitals and for individuals whose pri-

mary diagnosis was a pressure ulcer, and for acute care patients whose primary diagnosis was hip fracture with a secondary diagnosis of pressure ulcer. Data were based on the experience recorded or extrapolated for 1992. Braun et al. (1992) reported that the cost to heal complex full-thickness pressure ulcers was $70,000. Less serious pressure ulcers cost $20,000 to $30,000 to heal (National Pressure Ulcer Advisory Panel, 1989; U.S. Dept. of Health and Human Services, 1990).

The costs related to preventing pressure ulcers have been infrequently addressed in the literature. Hu et al. (1993) reported that current practice was no different in cost than the implementation of the AHCPR Clinical Practice Guideline *Pressure Ulcers in Adults: Prediction and Prevention* or Bergstrom et al. (1992). However, Xakellis and Frantz (1996) reported that aggressive preventive measures in long-term care were effective in reducing pressure ulcers and required a low level of institutional expenditures. It has not been possible to measure precisely the cost savings that could be obtained by implementing the prevention guideline. However, Miller and DeLozier (1994) reported that although the exact cost savings of implementing the AHCPR prevention guideline could not be determined, it was evident that some savings would occur. Reduced hospital admissions, fewer pressure ulcer-related procedures (debridement), and fewer ulcers requiring surgical intervention have the potential to save almost $50 million per year. It is clear that more studies are needed to determine the cost-effectiveness and cost-benefit of prevention strategies compared to treatment.

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 is part of the insulating system of the body whose function is to maintain the core temperature within a healthy range. Blood flow from the interior of the body to a venous plexus immediately beneath the skin is most efficient 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. Blood flow in the skin is mainly under nervous control rather than local control. Injury to the spinal cord interferes with this control and often is responsible for the fact that individuals with SCI are unable to compensate for extremes of cold and heat, depending on their level of injury.

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 crisscrossed with capillaries. The dermis consists mainly of collagen whereas the epidermis has no collagen. The skin damage that eventually develops into a pressure ulcer starts in the dermis before there is any visible breakdown in the epidermis.

Neurologically Impaired Skin

Individuals with SCI have an altered autonomic nervous system, with the degree of alteration varying with the level of injury. As a consequence of SCI above the neurologic level of T6, the functional properties of skin are altered. For this reason, individuals with SCI are unable to maintain constant body temperature, at least in the early stages following the injury. In normal skin when the skin temperature reaches 32-34 degrees centigrade, visible sweating normally takes place over a wide area of the body; this is called reflex sweating (Cosman, 1971). In people with SCI above the T6 level, reflex sweating is lost. Because of the loss of this reflex, individuals with SCI must be cautious since in a hot environment the body cannot cool itself effectively and can become easily overheated.

Biochemical Factors

Neurologically impaired skin undergoes a number of metabolic changes that do not usually stabilize for three to five years after the injury. These changes include:

- A significant and rapid increase in the overall rate of collagen catabolism immediately after the trauma resulting in abnormal concentrations of collagen metabolites in the urine (Claus-Walker et al., 1977) and a decrease in the amino acid concentration in the insensate skin (Rodriguez and Claus-Walker, 1988).
- A decrease in the activity of lysyl hydroxylase, an enzyme of collagen biosynthesis, in the skin below the level of injury (Rodriguez and Claus-Walker, 1988).

- A decrease in the proportion of Type I to Type III collagen in the skin below the level of injury (Rodriguez and Markowski, 1995).
- A decrease in the density of adrenergic receptors in the skin below the level of injury (Rodriguez et al., 1986).
- A large increase in the excretion of glycosaminoglycans (GAGs) in the urine (Pilonchery et al., 1983; Singh et al., 1977).

The increased urinary excretion of GAGs has a bearing on the biosynthesis of collagen. GAGs are produced by the degradation of proteoglycans, the ground substance surrounding the collagen bundles. Proteoglycans bind water and are thought to influence the type of collagen synthesized by the skin by the proportion of each particular glycosamine present in the parent proteoglycan. A lower content of GAG in the skin robs it of the necessary elasticity to adapt to mechanical insults at the same time that altered collagen biosynthesis decreases the tensile strength.

Rodriguez et al. (1986) found a greater decrease in the density of adrenergic receptors in individuals with tetraplegia compared to individuals with paraplegia. The increased catabolism of collagen coupled with defective collagen biosynthesis produces a more fragile skin below the level of injury. The decrease in density of adrenergic receptors could be the cause of abnormal vascular reactions. It has been reported that normally innervated skin can withstand ischemia three hours longer than neurologically impaired skin (Patterson et al., 1993). There is evidence that oxygen tension over the sacrum as well as over the tibia is lower in individuals with SCI (Bogie et al., 1992; Mawson et al., 1993; Patterson et al., 1993). It also has been noted that individuals with SCI have a reduced blood supply (Bennett et al., 1984), and reduced blood flow below the level of injury (Lindan, 1961), which would affect the delivery of nutrients to the skin. The decrease in the density of adrenergic receptors with time correlates well with other symptoms of vascular dysfunction in SCI, such as the reduced blood supply and deficient circulation mentioned above, as well as the impaired response of individuals with SCI to repeated surface pressure loads (Patterson et al., 1993). Under the same pressure load, individuals with SCI had a reduction of the transcutaneous P02 five times the magnitude of the reduction in able-bodied subjects (Hunt and Connally, 1978; Lindan, 1961). The abnormal vascularity would account for the diminished blood flow, lower nutrient availability, and lower tissue oxygenation, which are conditions that predispose to ulcer formation.

In a similar manner, fibronectin, a glycoprotein present in the connective tissue, may contribute to wound healing by promoting migration and attachment and proliferation of fibroblasts, the cells that secrete collagen. Lower concentrations of plasma fibronectin have been reported in SCI individuals at high risk for developing pressure ulcers (Viziri et al., 1992). The increased catabolism of collagen coupled with defective collagen biosynthesis produces a more fragile skin below the level of injury. Type I collagen produces 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. Its proportional increase contributes to the fragility of neurologically impaired skin. Skin with greater tensile strength has a higher ratio of Type I collagen (Flint et al., 1984). The increased degradation of collagen affects the integrity of the skin. It has been shown that the insensate skin of individuals with SCI who are prone to the development of pressure ulcers has widely spaced collagen fibrils (Donovan et al., 1988).

Mechanical Factors

Although the preponderance of scientific opinion suggests that pressure ulcers start below the surface of the skin and extend upwards, the first indication of a developing ulcer is usually a change in the skin surface. Since changes in color are only useful in people with light skin, various groups have attempted to use other characteristics. Hagisawa et al. (1994) measured changes in blood content and oxygenation in superficial vessels of the skin following an applied pressure of 150 mm of mercury over the trochanter. There was no substantial difference in the reactive hyperemia length and intensity between 10 SCI individuals and 10 able-bodied individuals. They did find a slower reflow rate after pressure in the SCI group. This slower reflow rate was noted by Schubert and Fagrell (1991), who used laser Doppler fluxmetry to measure the response of skin blood cell flow after locally applied pressure over the sacrum and the gluteus maximus muscle in 20 SCI individuals (10 tetraplegic and 10 paraplegic) and 10 able-bodied individuals. Doppler flow measurements are indirect inferences of local blood flow and count the red blood cell mass. Occlusion of blood flow was reached at a lower external pressure over the sacrum than over the gluteus in the able-bodied group. A smaller increase in temperature during occlusion was found in the SCI group with no sensation over the sacrum, compared to individu-

als who had sensation over the sacrum or to the able-bodied group.

Muscle atrophy that is caused by paralysis produces a loss of muscle bulk in the paralyzed parts of the body. The diminished bulk does not provide enough cushioning around bony prominences to protect the skin and absorb some of the mechanical forces. This mechanical deficiency combined with biochemical alterations of the neurogenically impaired skin are, in large part, responsible for the vulnerability of the individual with SCI to develop pressure ulcers.

Pathophysiology of Pressure Ulcers

Pressure ulcers result from the effect of gravity on the body mass in contact with a support surface. The forces at the contact point with the environment generate stress in the tissues, which may be called pressure, if the force is perpendicular, or shear, if the force is tangential to the tissue contact surface. The viscoelastic and microvascular properties of the tissue will determine its response to these forces (Bader, 1990; Bogie et al., 1995; Reddy et al., 1981). Prolonged stress in the tissue collagen network above the capillary and lymphatic vessel tolerance 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).

The primary factors leading to pressure ulcers are associated with pressure, shear (Bennett et al., 1979; Reichel, 1958; Schubert et al., 1994), and the resulting deformation of the soft tissues (Scales, 1990). Other predisposing or contributing factors have been identified including repetitive stress (Hall and Brand, 1979; Manley and Darby, 1980), nutritional deficiency (Bergstrom et al., 1992), biochemical and enzyme activity changes (Ryan, 1990), and collagen degradation (Rodriguez et al., 1989). Continuous stress and ischemia have been noted to contribute to ulceration in immobile individuals in bed, but repetitive stress has been reported to be a more important contributor to ulceration in ambulatory individuals with neurologic disorders (Brand, 1984).

It is clear that spinal cord injury produces profound changes in the structure and physiology of the skin, which contribute to its fragility and increased susceptibility to the development of pressure ulcers. There is evidence of an increase in the degradation of collagen before any ulceration is visible on the surface of the skin. 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. One of the metabolites, characteristic of skin collagen, was preferentially increased. The time elapsed from the start of the increased excretion to the appearance of the ulcer in the epidermis ranged from 2 to 5 months (Rodriguez and Garber, 1994). Some investigators have reported that the actual inception of a pressure ulcer is not in the skin at all, but in the underlying muscle (Daniel et al., 1981; Nolan and Vistnes, 1980). Muscle is more sensitive to ischemia than is skin. Pressure ulcers usually develop over bony prominences. Prolonged pressure on these areas of the body compresses the skin and muscle against the bone occluding some of the blood vessels. Muscle, needing more oxygen and being softer than skin, would be damaged first. This theory would not be in conflict with the findings of Rodriguez and Garber (1994). It is possible that some of the increased collagen excretion resulted from muscle basement membrane or tendons, but the amount from these sources would not be very large and it would probably be impossible to detect its contribution to the total urinary collagen metabolite excretion.

The histopathology of pressure ulcers has received little attention, but Vande Berg and Rudolph (1995) published a very detailed study of 20 stage IV ulcers examined by light and electron microscopy. Common features of the ulcers were an accumulation of fibrin on the inside edge, within which were inflammatory cells and vacuolated fibroblasts (the cells that secrete the collagen). The fibrin appeared to supplant the collagen matrix. Edema was also present near the surface of the ulcer, and there was partial to full occlusion of blood capillaries. These data could act as a reference point when studying the effect of growth factors and other substances in the treatment of chronic pressure ulcers.

Alterations to the skin structure have also been detected by biochemical means. Collagen is the principal component of the organic matrix of the skin and is responsible for its great tensile strength. Immediately after the trauma, SCI individuals excrete large amounts of collagen metabolites in the urine. This excess excretion ceases during the second year after the injury, but it resumes in cases of broken bones, heterotopic ossification, or a developing pressure ulcer (Rodriguez et al., 1989). The amino acid content of biopsies from the skin below the level of injury in SCI individuals was found to be lower than that of biopsies from the skin above the level of injury or from biopsies of able-bodied controls. The activity of lysyl hydroxylase (an enzyme involved in the biosynthesis of col-

lagen) was also lower in biopsies from below the level of injury than in biopsies from above the level of injury or from able-bodied controls (Rodriguez and Claus-Walker, 1988).

Other proteins seem to play a role in the healing of pressure ulcers, although their role is not clear. Collagen type VII biosynthesis seems to be necessary for chronic wounds to heal (Hopkinson et al., 1997). Type VII collagen is found in anchoring fibrils, structures found beneath most epithelial basement membranes. Individuals with elevated plasma fibronectin showed rapid healing of their pressure ulcers (Vaziri et al., 1992). SCI seems to disrupt the balance of the process of protein biosynthesis, thus affecting the integrity of the skin structure and function.

External support forces on the body are oriented obliquely at the contact area and cause both pressure and shear stress 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 will collapse easily from pressure loads, whereas vessels perpendicular to the surface will 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. Externally applied forces also cause change in tissue contours and deformation. 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 mechanical effect of external loads on tissue is resisted by the internal pressure and the strength of the collagen network in the tissue structure. Internal tissue pressure builds up in the interstitial fluid trapped between the cells and the collagen network. Pressure variation will squeeze the fluid from high- to low-pressure regions, causing local volume and contour change in the tissues (Reger et al., 1986). The fluid flow from the high- to low-pressure region will induce cell contact with other cells and the collagen network. The contact will build up stress at the cell boundary and destroy the collagen network. The contact stress will inhibit cellular metabolism and in time will stop new collagen synthesis (Reddy et al., 1981). The larger the pressure difference, the more effective is the squeezing out of the interstitial fluids and the mechanical damage to the cells and the collagen network. With small pressure difference, the immediate damage is more limited, but if it is prolonged, the lack of circulating nutrients and disrupted metabolism can result in the same damage. Change in contour and deformation will also result in pressure and shear stress in the tissues. The forces generating these stresses together will squeeze out the interstitial fluids more effectively and accelerate the tissue degeneration, atrophy, and the potential for ulcer formation (Reger et al., 1990).

It is evident that the precise mechanisms of pressure ulcer formation are not fully understood, despite years of study. What is clear, however, is that pressure ulcer formation in people with SCI is a complex process that transcends the biomechanical aspects of soft tissue's response to mechanical loading. Thus, health-care professionals and researchers are challenged to look beyond the obvious and focus on the intrinsic and extrinsic patient characteristics that influence the integrity of the skin. Diagnosis, tissue history (previous breakdown, surgical repair), body build, and magnitude and distribution of interface pressures are intrinsic to the individual. Number of hours of sitting in a wheelchair or lying in bed, types of activities performed, level of functional independence, type of wheelchair, cushion and bed surface usage, environment (climate, continence), and psychosocial factors such as living arrangement, caregiver support, and finances are extrinsic to the individual.

Recommendations

Prevention

Preventive techniques begin from day one of the onset of a spinal cord injury, from early medical management in acute care to rehabilitation and on to the home and community setting. Intensive education and skills training in the rehabilitation phase focus on the spinal-injured individual learning and using a variety of preventive measures to be carried out in the home/community setting (Basta, 1991).

Risk Factors, Risk Assessment, and Risk Assessment Tools

The etiology of pressure ulcers is multidimensional and includes unrelieved pressure, shear, friction, moisture, poor nutrition, immobility, and psychological, social, and economic factors such as inadequate personal and financial resources and noncompliance with acknowledged preventive behaviors (Krouskop et al., 1983).

All individuals with SCI are at risk for the development of pressure ulcers. Numerous risk factors have been identified and described in the literature. Demographic (e.g., age, marital status, education, ethnicity), physical/medical (e.g., level and completeness of SCI, nutrition, incontinence), and psychosocial factors (e.g., psychological health, cognition, substance abuse) have been investigated.

The results of some of these investigations have been inconclusive and contradictory with respect to the effect of the risk factor on pressure ulcer development, primarily because the investigations focused on only one or two of the variables. Other variables were not controlled, even though they had been identified as significant contributing factors. Level of the spinal cord injury has been associated with the occurrence of pressure ulcers. Conflicting data have been reported with regard to who is at greater risk—individuals with tetraplegia or individuals with paraplegia. Despite differences of opinion among investigators, there is consensus that individuals who have sustained a complete injury are at higher risk, regardless of the level of injury.

Risk factor assessment for pressure ulcers heightens the health-care professional's awareness of pressure ulcer prevention and improves clinical practice (Bergstrom et al., 1996). Deeks (1996) used a risk profile to trigger pressure ulcer prevention protocols. Preventive practices may be based upon specific scales and other risk variables (e.g., turning frequency, use of specialty support surfaces) (Blaylock, 1995; Clifford et al., 1995). In the intensive care setting (ICU), Lowery (1995) conducted studies on risk assessment tools, from which a wound care protocol was developed, including the use of specialty support surfaces, for managing individuals at risk for developing pressure ulcers in the ICU. In a multisite prospective cohort study (N=843), Bergstrom et al. (1996) used logistic regression analysis to predict prescriptive practices based on individual demographics and the Braden scale risk score. In a retrospective chart review (N=47), Blaylock (1995) demonstrated that select criteria from the Braden scale and the total Braden scale score (mean 11.62) were clinically significant for placement of individuals on specialty support surfaces. In a descriptive study involving 16 individuals, Clifford et al. (1995) used a risk assessment scale score, subjective assessment, and case record forms to conclude that an alternating pressure overlay and cushion were effective modalities in immobile individuals at high risk for pressure ulcer development.

1. **Conduct comprehensive, systematic, and consistent assessment of pressure ulcer risk factors in individuals with spinal cord injury.**

   - **Assess and document risk on admission and reassess on a routine basis, as determined by the healthcare setting, institutional guidelines, and changes in the individual's health status.**
   - **Use clinical judgment as well as a risk assessment tool to assess risk.**
   - **Assess demographic, physical/medical, and psychosocial factors associated with pressure ulcer prevention.**

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

Regular assessment should be incorporated into the overall comprehensive assessment of all individuals with SCI. Documentation may vary from every shift in an intensive care unit, to daily or weekly on inpatient units, to variable intervals in the community, based on frequency of outpatient clinic visits. Ooka et al. (1995) reported an 8-percent incidence of stage I and II pressure ulcers in 110 individuals on specialty support sur-

faces and advocated continuous monitoring of the skin condition even when these devices are used. Hoshowsky and Schramm (1994) reported an incidence of 16.8 percent of stage I ulcers in 505 individuals using pressure reduction devices and standard devices intraoperatively.

The trigger for pressure ulcer risk reassessment should be based on 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). 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).

All individuals with SCI are at life-long risk for developing pressure ulcers. Preliminary evidence demonstrates that risk assessment scales may be used successfully to predict pressure ulcers in various populations and result in favorable outcomes (Allman et al., 1995). However, scales reflect the individual's status, not necessarily the quality of care the individual receives.

The predictive value of existing risk assessment tools is imprecise for the SCI population because these were designed for the general medical population. Certain risk factors are not assessed by current tools (e.g., albumin, hemoglobin). Furthermore, the predictive value of tools appropriate to the SCI population in various settings (e.g., community) requires investigation.

An ideal risk assessment tool should have high predictive value for determining risk—i.e., high sensitivity (missing few individuals who are in fact at risk) and high specificity (few false positives for individuals rated as being at risk when they in fact are not. Although the scales, such as those designed by Salzberg et al. (1996) and Braden (Bergstrom et al., 1995; Bergstrom et al., 1996), seem most appropriate to the SCI population, future research is required to establish the predictive merit for SCI risk assessment variables.

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).

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. Most individuals with SCI will be at risk according to the Braden scale. The Braden scale is composed of six subscales measuring intensity and duration of pressure (activity, mobility, sensory perception) and tissue tolerance to pressure (nutrition, moisture, friction, and shear). Subscales are rated from the highest risk (1) to the lowest risk (4). Scores range from 6 to 23; the lower the summative number of points, the greater the individual's risk for pressure ulcers (Bergstrom et al., 1996).

The cut-off point of a scale, indicating the score at which the individual is at risk for a pressure ulcer, should be determined according to the tool, health-care setting, population, and other variables (Edwards, 1994). However, the Braden scale has cut-off points ranging from 11 to 19 in cited studies (Harrison et al., 1996; Jiricka et al., 1995; Ramundo, 1995; Tourtual et al., 1997; VandenBosch et al., 1996). Factors in establishing a cut-off point for the at-risk status of individuals with SCI have not been well established.

Salzberg et al. (1996) and Salzberg et al. (1998) developed pressure ulcer risk assessment scales designed for individuals with SCI in hospital and community care settings. The Salzberg scales have had limited testing for validity or reliability. Lehman (1995) identified common factors related to pressure ulcers for individuals with SCI residing in the community.

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. Widely used in the United Kingdom, the Waterlow Pressure Sore Risk Calculator (Clifford et al., 1995; Edwards, 1995) considers build/weight for height, continence, mobility, and appetite. The Watkinson scale (Watkinson, 1996; Watkinson, 1997), Cubbin and Jackson scale (Hunt, 1993),

and Stratheden scale (McCormack, 1996) also have been cited in the non-SCI literature.

Arnold (1994) compared the risk scores assessed by individuals with SCI to those assessed by health-care providers. The author determined that there was no significant relationship between the assessed scores for developing pressure ulcers in an SCI population. However, some significant relationships were found in examining some individual factors (e.g., mobility and skin circulation).

In a prospective cohort study of 60 men with SCI, Rodriguez and Garber (1994) noted a significant correlation between the individual's belief he was at risk for developing pressure ulcers and the individual's number of prior pressure ulcers. However, the authors reported no significant difference in individuals' perceptions of the importance of various prevention strategies, beliefs about the risk of pressure ulcer development, and beliefs regarding the serious nature of this complication.

Controversy exists in the literature regarding many of the variables for determining risk status. Study limitations, such as small sample size, low statistical power, lack of controls and randomization, differences in pressure ulcer stages, and interrater reliability, should be considered when generalizing findings to individuals with SCI in various health-care settings. This recommendation focuses on three major groups of risks factors: demographics, physical/medical, and psychosocial.

DEMOGRAPHICS

Age:

As the SCI individual ages, there is decreased muscle mass, decreased collagen, altered elastin, and increased comorbidities, which increase the individual's risk for developing pressure ulcers (Bergstrom et al., 1996). Vidal and Sarrias (1991) found that as individuals with SCI age, particularly over 40, the number and seriousness of pressure ulcers tended to increase. Rochon et al. (1993) found that SCI individuals 60 years or older were at more risk than younger individuals with SCI. However, Rochon et al. (1993) indicated that comorbidities may be more important than age in predicting pressure ulcer development. Salzberg et al. (1998) reported that age was not significant in pressure ulcer development in individuals with SCI in the community. Similarly, Mawson et al. (1988) found that age was not a significant risk factor in the SCI population who developed pressure ulcers immediately post-injury.

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 an ulcer. Rodriguez and Garber (1994) did not find a significant association between duration of SCI and recurrence among a population with previous pressure ulcers.

Gender:

Vidal and Sarrias (1991) found there was a 3:1 ratio of males to females, although Salzberg et al. (1996) and Salzberg et al. (1998) found no gender differences in SCI populations. In the non-SCI population, Bergstrom et al. (1996) noted that women had significantly more ulcers than males in a prospective cohort study of 843 individuals. However, Ek et al. (1991), Jiricka et al. (1995), Ooka et al. (1995), and Oot-Giromini (1993) found gender did not pose a significant risk in the general population.

Ethnicity:

Bergstrom et al. (1996) and Jiricka et al. (1995) related that white people may be at greater risk for developing pressure ulcers than the non-white population. Allman et al. (1995), Ek et al. (1991), and Mawson et al. (1988) found that race was not a significant factor in pressure ulcer development. It must be noted that in individuals with darker skin, stage I pressure ulcers may be more difficult to detect than in individuals with lighter skin.

Marital Status:

Several investigators have studied the effect of marital status on the occurrence of pressure ulcers. Unfortunately, there seem to be differences among the findings. Young and Burns (1981a and 1981b) found that, at followup, married people were less likely to have a pressure ulcer. In a study by Thiyagarajan and Silver (1984), the majority of individuals with SCI readmitted to a rehabilitation hospital with pressure ulcers were single. 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.

Education:

Vidal and Sarrias (1991) found a negative relationship between education and pressure ulcer risk. Evidence supports a relationship between education and health outcomes, generally. Davidoff et al. (1990) found that less education predicted more hospital readmissions among a group of 88 people with recent SCIs after their acute rehabilitation. Results from Lloyd et al. (1993) indicate that a lower level of education is linked to less understanding and follow-through with complex recommendations for managing chronic conditions.

Based on a retrospective medical record review involving 884 individuals with SCI, Vidal and Sarrias (1991) profiled 268 individuals with pressure ulcers: sensory and motor complete paraplegia; male; less educated; not standing; not spastic; prone to recurrent ulcers; prone to urinary tract infections; 40 years or older; and comorbidities. Rochon et al. (1993) conducted a retrospective chart review of 364 individuals with SCI to determine the profile of 81 individuals with pressure ulcers. The authors reported the following significant risk predictors for individuals with SCI: low albumin, a higher score on the Cumulative Illness Rating Scale, motor complete, and a history of pressure ulcers. Lehman (1995) reported a higher incidence of pressure ulcers among paraplegics in the community.

PHYSICAL/MEDICAL

Level and Completeness of Injury:

In a study of 549 SCI individuals, Richardson and Meyer (198l) found that the prevalence of pressure ulcers was greatest with cervical complete injuries and that the next highest prevalence was among individuals with thoracic complete injuries. Thiyagarajan and Silver (1984) reported prevalence in 100 SCI individuals with pressure ulcers to be greater in complete SCI, with more tetraplegics in the acute group and paraplegics with thoracic lesions in the chronic group. In two descriptive prospective cohort-designed studies, Tourtual et al. (1997) identified limb weakness as a significant risk factor in pressure ulcer development.

Salzberg et al. (1998) related that complete SCI was a significant risk factor for pressure ulcers and that there tended to be a greater incidence of pressure ulcers in people who had an etiology of traumatic SCI. In an earlier study of 219 Department of Veterans Affairs (VA) patients with SCI, Salzberg et al. (1996) found that complete SCI was significant, but level of injury was not significant. Mawson et al. (1988) and Curry and Casady (1992) found no statistical significance related to the development of pressure ulcers based upon completeness and level of injury immediately post injury.

Activity and Mobility:

Activity, as distinguished from mobility, refers to involvement of the person in recreation, including, but not limited to, athletics. It also refers to social and vocational activity. 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 the analysis of 800 questionnaires of 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. Multiple studies of non-SCI individuals have substantiated immobility as a precursor to pressure ulcer development (Allman et al., 1995; Bergstrom et al., 1996; Berlowitz and Wilking, 1989; Blaylock, 1995; Ek et al., 1991; Maklebust and Magnan, 1994; Oot-Giromini, 1993).

In people comparable in age, spinal cord injury severity, and pre-injury health, athletic involvement was associated with less likelihood of pressure ulcer, as well as other complications and rehospitalizations (Stotts, 1986). Controlling for level and completeness of injury, involvement in social and vocational activity was found to predict survival among a large sample of individuals with SCI over 11-year (Krause and Crewe, 1987) and 15-year periods (Krause et al., 1990).

Bladder, Bowel, and Moisture:

Appropriate bladder and bowel management programs will prevent skin from becoming contaminated with urine and feces. Salzberg et al. (1996) and Salzberg et al. (1998) reported that urinary and fecal incontinence were significant factors in pressure ulcer development in the SCI population. Rochon et al. (1993) did not find fecal or urinary continence to be significant predictors of pressure ulcers in people with SCI. Excess perspiration is seen in people with spinal cord injuries. The presence of moisture on the skin may lead to maceration and compromise the skin's natural barrier to infection.

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. 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 (N=219). In a later study by Salzberg et al. (1998), data obtained from questionnaires of 800 SCI individuals in the community revealed that renal and pulmonary diseases were significant variables, 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 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 a significant risk factor in pressure ulcer development.

In a retrospective chart review of 268 SCI individuals, Vidal and Sarrias (1991) found that recurrence of pressure ulcers was a highly significant risk factor for increased severity of the ulcer. In a descriptive study of 29 SCI individuals in the community, Lehman (1995) reported a 52-percent recidivism for pressure ulcers in the sample. Rochon et al. (1993) and Rodriguez and Garber (1994) reported that a history of pressure ulcers was a significant risk factor among individuals with SCI.

Mawson et al. (1988) reported that SCI individuals who developed pressure ulcers in the immediate post-injury period had significantly lower systolic blood pressure (≈ 99–100 mm Hg) compared to controls (≈ 120–121 mm Hg). Schubert et al. (1995) noted that SCI individuals had a significantly lower systolic blood pressure compared to elderly or control groups. In two studies, autonomic dysreflexia was identified as a significant risk factor for pressure ulcer development among SCI individuals (Salzberg et al., 1996; Salzberg et al., 1998). Allman et al. (1995) related that hypotension was not a significant risk factor. Readers are referred to the clinical practice guideline titled *Acute Management of Autonomic Dysreflexia* and its companion consumer guide, *Autonomic Dysreflexia: What You Should Know*, for more detailed information. (Both publications are available at PVA's website, www.pva.org.)

Select blood chemistry and biochemical tests may be useful for predicting pressure ulcer risk for individuals with SCI. Specific biochemical indices that are associated with the risk of pressure ulcer development include total protein, albumin, hemoglobin, hematocrit, and total lymphocyte count. (*See "Biochemical Parameters" on page 30*).

PSYCHOLOGICAL AND SOCIAL FACTORS

The findings have been mixed regarding pressure ulcer risk factors associated with an individual's psychological health. Some of the factors associated with pressure ulcers involve the behavior of the individual who has the injury and the behavior of those in his/her formal and informal support networks. Many of the recommendations for prevention, such as performing weight shifts, require understanding, cooperation, and initiative. 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), also involves the individual and his/her caregivers in many complex, demanding behaviors. The regimens involved in managing SCI are complex and involve lifestyle changes. 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).

Limited spinal cord injury research is available to indicate that aspects of the person and of the person's social and physical environment can affect behavior and thereby affect the risk of developing pressure ulcers. Rintala (1995) reported that interpretation and generalization of findings about pressure ulcer risk and prevention are limited by differences in sample characteristics, sample sizes, points in the rehabilitation course, and measures and study designs. Intervention studies are conspicuously lacking.

Considerable research has been done on other chronic conditions, investigating the links between the person's behavior and health outcomes and identifying sources of influence on that behavior. Research from the multidisciplinary field of behavioral medicine indicates that if a person has any of the problems described below, that individual's adherence to health behavior recommendations has the potential to be compromised. The following factors should be assessed and any identified problems treated by qualified professionals as part of the effort to prevent pressure ulcers and/or to prevent their recurrence. Citations providing support from the spinal cord injury literature are noted with an asterisk (*); otherwise, citations are from studies of other chronic conditions, such as diabetes or cardiovascular disease, and refer to adherence issues shared across disorders. Psychosocial risk factors associated with pressure ulcer prevention are often overlooked. However, literature supports consideration of these factors when developing prevention strategies.

Psychological Distress:

This includes major depression and anxiety disorders as well as negative self-concept or poorly managed anger and frustration. Any of these may interfere with cooperation between the individual and care providers and can be associated with inactivity, self-neglect, and poor medical adherence (Cox and Gonder-Frederick, 1992; Vidal and Sar-

rias, 1991*; Woolsey, 1985). Krause and Kjorsvig (1992*) found lower survival rates, including deaths from sepsis following pressure ulcer, 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 may result from another comorbidity (e.g., delirium secondary to infection, medication problems, preexisting cognitive dysfunction and learning disability, or substance abuse) (see below). Several studies indicate a relationship between impaired mental status and risk of pressure ulcer. In a study of 219 SCI individuals in a VA setting, Salzberg et al. (1996*) identified impaired cognitive function as a significant risk factor for pressure ulcers. Richards et al. (1991*) reported an increased risk for pressure ulcers among people with SCI who had also sustained a traumatic brain injury. Although Macklebust and Magnan (1994) reviewed data on a large sample of individuals and reported that 51 percent of those who had pressure ulcers also showed evidence of decreased mental status, the authors did not provide a definition of how this was measured. Other investigators (Allman et al., 1995; Rochon et al., 1993) found that level of consciousness was not a significant factor in pressure ulcer development.

The effects of cognitive impairment on pressure ulcer impairment are likely to vary depending on how much responsibility the individual is given for managing self-care. This, in turn, is likely to vary according to such variables as acuteness, severity of physical and sensory impairments, presence of other medical conditions, age, and the setting (e.g., hospital, home, or extended care facility). Investigations examining cognitive impairment and pressure ulcer development are limited because of:

(1) vague or no definitions of mental status,

(2) little or no information about assessment measurements, or

(3) little or no information about important sample characteristics, such as age, acuteness, and severity of physical and sensory impairments.

Thus, it is not possible to draw firm conclusions regarding cognitive impairment as a risk factor for pressure ulcers until further research is conducted.

Substance Abuse:

Substance abuse is a likely risk factor for pressure ulcer development after SCI (Vidal and Sarrias, 1991*). Hawkins and Heinemann (1998*) found increased risk for illicit substance abusers but not heavy drinkers in a sample of 126 individuals with SCI. Substance abuse can lead to impairments in cognition and judgment (Cleaveland and Denier, 1998) and is associated with less adherence to various health regimens (Pablos-Mendez et al., 1997; Umpierrez, et al., 1997). Substance abuse also may affect health directly (e.g., excessive alcohol intake increases the risk of poor nutritional intake, as well as bladder distension and urinary incontinence, all of which are potential risk factors for pressure ulcer). However, findings to date are not consistent (e.g., Salzberg et al. (1996*) did not show alcohol history to be a risk in their sample). Again, variation in methods, measures, and samples limit interpretation.

Based on the known physiological effects of smoking, which could be expected to impair circulation to the skin, health educators often advise SCI individuals against smoking. Evidence of smoking as a risk factor is mixed. Salzberg et al. (1996*) found that individuals with SCI who had pressure ulcers were twice as likely to be current smokers. However, a later study by Salzberg et al. (1998*) did not identify smoking as a risk factor. It is reasonable to assume that smoking is a risk factor for the development of pressure ulcers and would interfere with healing.

Adherence:

A verbal or written commitment from the person to follow through with a health behavior recommendation has been associated with better adherence (Cox and Gonder-Frederick, 1992; Meichenbaum and Turk, 1987), and so such commitment should be sought routinely. Adherence should not be regarded as a global trait; each individual may vary considerably in the degree to which he/she follows one behavioral recommendation versus another, and adherence may change or vary over time (Johnson, 1992; Merbitz et al., 1985*). The person's adherence to distinct aspects of his/her health-behavior regimen can be monitored at different points in time in order to address specific behavioral deficits as they arise.

A consistent predictor of adherence to a health-behavior regimen found in the general behavioral medicine literature is an individual's relationship and communication with the person making the recommendations (Cox and Gonder-Frederick, 1992; Golin et al., 1996; Hulka et al., 1976; Kurtz, 1990; Meichenbaum and Turk, 1987). Unfortunately, little formal investigation of these factors has been conducted in SCI research.

However, the wealth of evidence found in research on other conditions suggests that relationship and communication factors would likely play a significant role in the clinical efficacy of pressure ulcer prevention strategies and interventions.

Individuals often do not understand or retain health behavior instructions the first time such instructions are given. Several studies have found that recall of physicians' recommendations is directly related to the amount of time spent delivering and explaining the advice (Kravitz et al., 1993; Meichenbaum and Turk, 1987). Individuals' recall of recommendations about changes to lifestyle and daily routines is much poorer than recall about medications, and this is paralleled by much poorer adherence to lifestyle recommendations (Kravitz et al., 1993). The individual's understanding of health behavior recommendations can be assessed through direct, specific questions. The person's ability to verbalize his/her health-behavior regimen is a minimum indicator of adherence (i.e., understanding is necessary but not sufficient to produce the recommended behavior) (Rodriguez and Garber, 1994*). (*See Recommendations 8 and 22 for discussions on "Patient Education."*)

Assessment of the SCI individual's agreement with recommendations, as well as identification and clarification of misunderstandings, may be helpful. Individuals with SCI often have many misconceptions about pressure ulcers and the risk factors associated with them. For example, Rodriguez and Garber (1994*) found that 82 percent of their sample of people with SCI who had experienced a previous ulcer did not believe they were at risk of future ulcers. After discharge from hospital rehabilitation, individuals with SCI may not believe that followup appointments at SCI outpatient clinics are worthwhile for preventing secondary complications (Canupp et al., 1997*; Stockton, 1994*). Potential points of disagreement between the health-care provider and the individual 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.

An individual with SCI may perceive insurmountable barriers to adherence. Canupp et al. (1997*) found that individuals who did not return for recommended appointments at an SCI outpatient clinic were more likely to report difficulties with cost, distance, and transportation. Lower socioeconomic status (SES) has consistently been linked to poorer adherence and greater rates of secondary complications for many conditions (Lloyd et al., 1993). Consistent relationships have been found between an individual's reported self-efficacy to follow a particular regimen and better follow-through and better health outcomes (Golin et al., 1996). Investigators have recommended that education efforts be directed at removing or ameliorating reported barriers. Herrick et al. (1994*) found that self-reported problem-solving ability was related to development of pressure ulcers; people with SCI who reported using systematic strategies of problem-solving, such as problem identification and strategy planning, were at significantly less risk. Repeated practice in recommended techniques, particularly in the home, as well as improvements in time management, resources, and the physical environment, may produce better follow-through than lectures attempting to persuade and motivate (Cox and Gonder-Frederick, 1992).

Effective social support has been linked to better adherence to self-care (Cox and Gonder-Frederick, 1992; Lloyd et al., 1993). Some studies have found that individuals reported stronger intentions to adhere to self-care regimens when they believed it was important to family and significant others (Shenkel et al., 1985–6). There is evidence that intervening to increase family awareness and support of recommended regimens can improve adherence and health outcomes (Morisky et al., 1985). Results obtained by Krouskop et al. (1983*) provide some evidence for this among people with SCI, although their study design does not allow the effects of family education to be separated from the effects of patient education.

Prevention Strategies

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

- **Avoid prolonged positional immobilization whenever possible.**
- **Institute pressure relief as soon as emergency medical condition and spinal stabilization status allow.**
- **Initiate intraoperative pressure reduction strategies.**

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

The duration of unrelieved pressure prior to nursing unit admission and the length of time on the spinal board are significant risk variables for pressure ulcer development within the first 8 days post spinal cord injury (Mawson et al.,

1988). However, these factors were not associated with pressure ulcer development during the 30-day observation period. 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 six hours developed pressure ulcers at a significantly greater rate than individuals immobilized for shorter periods of time.

A study on 32 spinal cord-injured individuals 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). Researchers found that all individuals with pressure ulcers had experienced a prolonged immobilization between the onset of injury and admission to the acute care ward. Individuals (N=14) who had pressure ulcers were not turned during the first 2 hours post injury, whereas 13 individuals who did not have pressure ulcers had been turned within 2 hours (N=27). No significant differences were found between the groups in terms of mode of transportation to the hospital, type of immobilization device used en route to the hospital, the occurrence of surgery within 24 hours of the injury, or the time spent in the operating room. The pressure ulcer group did have significantly longer time to reach the hospital, more time in x-ray, and a significantly longer time to reach the acute care unit. In a multiple comparison study, Aung and El Masry (1997) found that paraplegics and tetraplegics who were admitted to an SCI center less than l week post injury were less likely to develop pressure ulcers than those who were admitted more than l week post injury (N=219).

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:**

   - **Ischii**
   - **Sacrum/coccyx**
   - **Trochanters**
   - **Heels**

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

Frequent inspection is essential to detect early warning signs of impending skin breakdown (e.g., nonblanchable erythema). Individuals with lower level injuries (paraplegia) may perform self-inspection with a long-handled mirror; individuals with high levels of paraplegia and tetraplegia must rely on a caregiver or professional for thorough inspection.

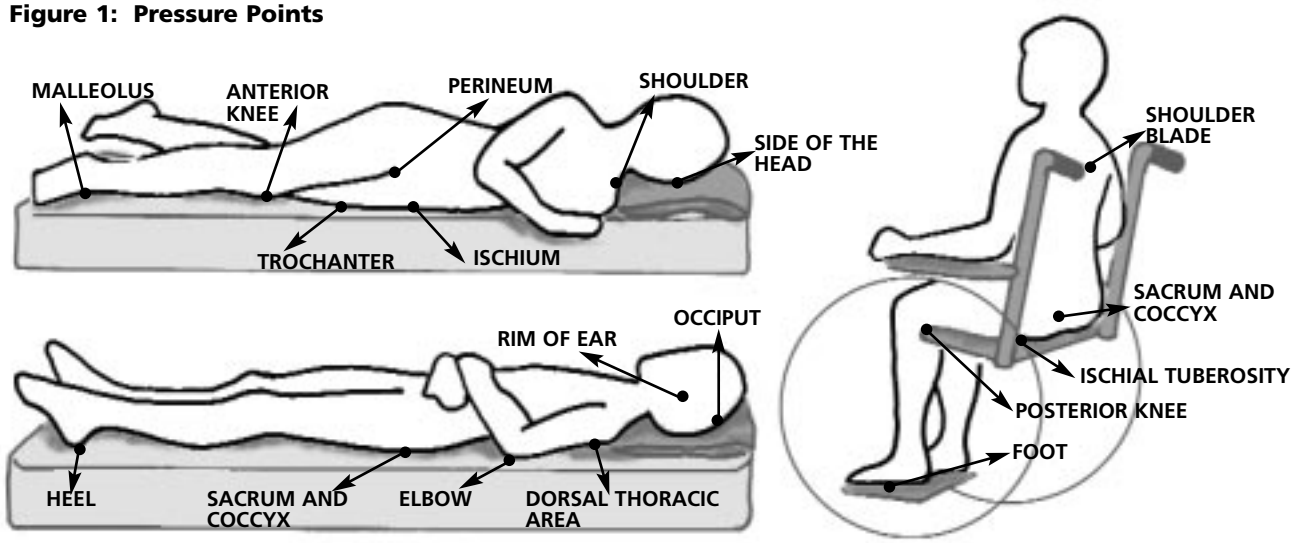
Vidal and Sarrias (1991) reported that the highest incidence of pressure ulcers in the SCI population was on the ischium (28 percent), sacrum (21 percent), and trochanter (20 percent). Lehman (1995) also reported a higher incidence of pressure ulcers in the SCI population in the ischium. In the immediate post-SCI period, Mawson et al. (1988) related that the most common area was on the sacrum (57 percent) and heel (22 percent). Richardson and Meyer (198l) related that among 549 SCI individuals, the most frequent single site for pressure ulcers was the sacral region.

In a community setting in which 23 individuals with SCI were given a structured interview, Garber et al. (1996) reported that 90 percent of paraplegics detected the ulcer by self-inspection or "feeling it," and 92 percent of the tetraplegics reported that someone else detected the ulcer. Although many individuals with paraplegia may detect pressure ulcers, the ulcer may be more advanced by the time it is noted (Garber et al., 1996).

The skin should be visually inspected daily, paying specific attention to bony prominences (Bergstrom et al., 1992), to assess for any changes in skin color (red areas/discolorations, bruises), and 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 ischia, sacrum, coccyx, trochanters, heels, ankles, knees, scapulae, and elbows (Scotzin and Sommer, 1993). *(See Figure 1.)* The recommendation is for the SCI individual to be responsible for carrying out this task. If this is physically or cognitively impossible, it is best if a consistent person (family member or care provider) assist with the task since it is necessary to have a base of comparison in order to detect a change in status. Regardless of a spinal-injured person's physical ability, he/she can still be in control of directing others to assist in this measure.

4. **Turn or reposition individuals with SCI initially every 2 hours in the acute and rehabilitation phases if the medical condition allows.**

Figure 1: Pressure Points



- **Eliminate stretching and folding of soft tissues and prevent shearing when individuals are repositioned.**
- **Avoid positioning individuals who are side-lying in bed directly on their trochanter.**

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

Research is limited in the area of frequent turning. Knox et al. (1994) conducted a quasi-experimental study exploring the length of turning intervals and body position on interface pressures in healthy older adults. The researchers' hypothesis that length of the turning interval and body position would make a significant difference in interface pressure was not supported. Using a Latin-square design, Knox et al. (1994) found a statistically significant increase in skin surface temperature at the end of 2-hour turning intervals (N=48, P=.004, chi-square). Norton et al. (1975) conducted an observational study of older people (originally done in 1962) and demonstrated that individuals turned every 2 to 3 hours had fewer ulcers.

The technique of turning an individual is as important as the frequency of turning. Individuals should never be dragged across surfaces, as skin that is slid or dragged has an increased risk of being damaged by friction. Use turning devices such as sheets, a trapeze, or manual or electric lifts that will reduce the risk of skin damage. These concepts are also important in minimizing skin breakdown during transfers from one surface to another.

Certain physical movements associated with poor turning and transfer techniques and prolonged, unrelieved pressure can result in the development of pressure ulcers. Using lifting devices to assist in moving individuals who cannot assist during transfers or changes in bed position may help to reduce friction or shearing forces exerted on the skin. Friction injuries 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/bed mobility, or ineffective weight-shift techniques could be contributing factors to this problem. An adequate pressure-relieving mattress is recommended for use while lying in bed, as well as a consistent turning schedule to relieve pressure over bony prominences (Lowthian, 1993). 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 abrasive features, such as hard fasteners or studs on rear pockets or double 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). These items may restrict normal blood flow in the body or cause undue friction or shear (Krouskop et al., 1983; Scotzin and Sommer, 1993).

Garber et al. (1982) reported the effect of side-lying and trochanteric interface pressures. Seiler et al. (1986) measured the effect of side-lying positions on transcutaneous oxygen tension. Results of these studies indicated that direct positioning on the trochanter (90-degree angle) produces high interface pressures and low transcutaneous oxygen tension. When individuals were positioned at a 30-degree side-lying angle, the body's transcutaneous oxygen tensions were normal, with significantly reduced interface pressures.

Pillows or foam wedges should be used to keep bony prominences from contacting one another. Skin is to 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 is contraindicated, secondary to orthopedic restrictions, yet should be considered when spinal and respiratory stability is established.

5. Evaluate the individual and his/her support environment for optimal maintenance of skin integrity.

- **Apply pressure-reducing support surfaces preventively to protect soft tissues from bruise and injury.**
- **Prevent moisture accumulation and temperature elevation at the support surface-skin interface.**
- **Apply pillows and cushions to bridge contacting tissues and unload bony prominences; do not use donut-type devices.**
- **Establish a mechanism to follow up on equipment performance specific to pressure ulcer prevention (support surfaces for the bed and wheelchair) and determine if changes in medical or health status have altered the effectiveness of the support surface.**

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

The use of pressure-reducing devices prophylactically is effective in reducing the risk of pressure ulcers (Zernike, 1994). Adequate pressure reduction with minimum contact at the sacrum of the supine individual is recommended (Bogie et al., 1992).

Sitting interface pressures are significantly greater than supine support pressures due to smaller contact area. In the absence of sensation, localized sitting interface pressures much higher than capillary closing pressure have been measured and tolerated well. Both paraplegic and tetraplegic subjects were found to sit and travel for hours without pressure relief and without ulceration (Patterson and Fisher, 1986). The critical factor for tissue survival is perfusion (Souther et al., 1974). Higher intermittent pressures may be tolerated more than uninterrupted continuous lower pressures. Wheelchair cushion performance should be evaluated by its pressure-time effect on tissue function (Rithalia, 1997). Collectively, these and other biochemical 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 endproducts, hypoxia, cell rupture, and necrosis (Reddy, 1990). Therefore, pressure-relieving strategies are best when they follow an individualized approach based on individual and caregiver characteristics with the objectives of prevention, early detection, and affordability (Remsburg and Bennett, 1997).

At constant pressure, 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). The physical condition of the individual is also important.

The purpose of cleansing is to remove foreign material from the surface of the pressure ulcer. Cleansing as a preventive measure has not been studied. However, cleansing on a regular basis and at times of soiling is recommended (Bergstrom et al., 1992; Rodeheaver, 1999). Cleansing agents should minimize the drying of the skin. Guralnik et al. (1988) reported that low humidity promotes dryness and scaling of the skin, which have been associated with pressure ulcers in the elderly. Humidity should also be minimized (Bergstrom et al., 1992).

Moisture is a factor that must be kept to a minimum so that skin can remain dry. Should bowel or bladder incontinence occur, the SCI person should clean and dry the skin as soon as possible. Mild cleansing agents that minimize irritation

and dryness of the skin are recommended; hot water should be avoided. When cleansing the skin, care should be taken not to exert undue force and friction to the tissue. When the 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 may be used (Bergstrom et al., 1992). Wet skin has more of a tendency to adhere to bed linens, possibly causing shearing when the linen is pulled away from the skin (Krouskop et al., 1983). If the spinal-injured person lives in a humid climate or if excessive perspiration is a problem, cotton fiber clothing 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).

Bridging techniques may be used to support a bony prominence with pillows situated above and below the prominence. Bridging allows blood to flow to the area of concern. Pillows support the extremities and maintain the individual's position in both the bed and the wheelchair. Donut-shaped devices are strongly discouraged because they decrease blood flow to the surrounding tissue rather than protect the bony prominence (Bergstrom et al., 1992).

During the SCI indiviudal's initial rehabilitation, the therapist will establish a progressive sitting program. Sitting time is usually set at 30- to 60-minute intervals and is slowly increased every few days, if hyperemia resolves within 30 minutes of returning to bed (Yarkony, 1994). A pressure-reducing cushion of foam, gel, air, or a combination of these materials should be used. Donut cushion-type devices are not to be used (Bergstrom et al., 1992).

Beer (1984) evaluated an Australian home visiting nursing service provided to spinal cord-injured individuals upon discharge to their homes. The goal of the service was primarily preventive nursing care, with demonstrated outcomes of reduced readmissions to the spinal unit. The home visiting nurses provided both education as well as physical and emotional support for individuals. Evaluation of admission for pressure ulcer treatment showed a 34-percent reduction and bed occupancy a 30-percent reduction.

Today, health care is delivered across a continuum of care settings. The use of consistent guidelines is strongly recommended across acute care, subacute, rehabilitation, and long-term care facilities. Ennis and Meneses (1997) state that this is the most ethical and efficient way for managed care systems to ensure integration of all components with consistent approaches. Because the grief reaction differs from person to person, it may be helpful for a home-health nurse to visit a newly discharged SCI individual in his/her own living environment. As described by Beer (1984), this visit could consist of an assessment of the person's integration of physical skills as well as knowledge of self-care tasks in the home setting.

At discharge from the initial hospitalization, some individuals with a spinal cord injury have not accepted the permanence of their disability. A visiting nurse could answer questions that arise when the person is responsible for caring for himself/herself. Should problems arise, the nurse can direct the person to an appropriate professional resource. An outpatient occupational and physical therapy visit may be recommended following discharge to ensure that the equipment and functional skill maneuvers that were prescribed for home care are working in the individual's living environment. Early identification of problems will help to prevent pressure ulcers if such problems can be resolved immediately.

If funding allows, a subsequent inpatient or day-hospital readmission may be sought in order to teach new, more independent skill techniques. Because initial rehabilitation admissions are shorter now, individuals with SCI are returning to the community with less information about self-care, fewer opportunities to reinforce newly learned health-care strategies, and less developed functional abilities (Garber et al., 1996). With improved muscle strength, endurance, and readiness to learn, the person with SCI could benefit from a second rehabilitation phase by upgrading physical and functional skills and improving knowledge of prevention strategies. Lowthian (1993) suggests that when an individual with SCI enters a hospital or long-term care setting for medical care, an "at risk for pressure ulcers" card be issued, similar to those carried by persons taking life-saving medications, so that pressure ulcer risks can be minimized by staff personnel.

Other recommendations pertaining to bed positioning include:

(1) placing any person who is at risk for developing a pressure ulcer on a pressure-reducing device, such as a foam, static air, alternating air, gel, or water mattress;

(2) using devices that totally relieve pressure on the heels, most commonly those that raise the heels off the bed; and

(3) maintaining the lowest degree of elevation consistent with medical conditions and other restrictions and limiting the amount of time

that the head of the bed is elevated (Bergstrom et al., 1992).

These measures are to be followed after the acute hospitalization, whether the place of residence is at home or in a long-term care facility.

Weight control can be a challenge when energy expenditure and efficient exercise are compromised. Fatty tissue is not as well plenished with blood supply as muscle, so although some spinal-injured individuals may believe more body mass will protect them from pressure ulcers, this is not accurate (Krouskop et al., 1983; Waterlow, 1996). A change in body weight may make personally owned 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 or fluid.

Environmental or technical factors may contribute to pressure ulcer formation. AHCPR guidelines recommend minimizing environmental factors, such as low humidity or exposure to cold, that could lead to dry skin (Bergstrom et al., 1992). Moisturizers should be used to treat dry skin. External heat, such as over-exposure to the sun, hot showers, cigarettes, exposed pipes and heaters, heating pads or electric blankets, ovens (conventional, microwave), or hot liquids, could cause skin burns that may lead to ulcer formation (Scotzin and Sommer, 1993). Pressure-relieving cushions that minimize the absorption of heat allow the skin-to-cushion interface to remain at a lower local temperature. This allows for a more stable metabolic state and requires less oxygen demand in the tissues at the interface (Nixon, 1985).

Community-based seating services should be available as part of the continuum of care. Consultations with health-care professionals knowledgeable about the changing needs of individuals with SCI will prevent severe pressure problems, minimize hospital admissions, and shorten home health-care services. Such a program can provide a rapid response to people who are at high risk for serious medical complicatons. Demographic studies show that the SCI population is living longer, and therefore, the demand for services and evaluation of pressure-relieving equipment is increasing (Cox-Martin and Pullen, 1996; Hallett, 1996; Stockton, 1994).

6. Provide an individually prescribed wheelchair and pressure-reducing system.

- **Establish and initiate a specific pressure relief regimen within the individual's capability.**

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

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

Decisions regarding equipment selections are being made more quickly these days because of the shorter length of hospital stays and reductions in funding. For this reason, continued assessment of the compatibility of each individual and his/her equipment is important (Garber and Krouskop, 1997). Routine maintenance and parts replacement lessen the possibility that poor equipment conditions will contribute to pressure ulcers. Unfortunately, the very equipment that has been selected to prevent pressure ulcers may contribute to them if it is inadequate or poorly maintained. Normal wear and tear on wheelchair seat upholstery can result in a "sling or hammock" effect. Hallett (1996) explains that this effect creates increased pressure on the buttocks and can cause the knees to rotate inward, possibly pressing against one another. Excessive "slinging or hammocking" of the back upholstery can create increased pressure or shear on the upper and lower back.

Therapists frequently recommend that SCI individuals perform weight shifts as a part of their pressure ulcer prevention regimen (DeLateur et al., 1976). A variety of techniques can be employed, based on the person's physical and cognitive status. The most frequently used techniques are the full push-up, side or lateral lean, and forward lean, or alternatively use of a mechanical reclining or tilting wheelchair feature that can facilitate the weight shift. Generally, a weight shift every 15 to 30 minutes is recommended to allow the skin to be replenished with oxygen (Bergstrom et al., 1992; Nixon, 1985). In cases of poor skin care behaviors or cognitive deficits, a watch timer can be used to cue the person that a weight shift needs to be performed (Bridle et al., 1992).

7. Implement an ongoing exercise regimen for the medically stable SCI individual to promote maintenance of skin integrity, increase strength of paretic and nonparalyzed muscles, improve cardiovascular endurance, and prevent fatigue and deconditioning.

(Scientific evidence–II/III/V; Grade of recommendation–B/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). After

surgical intervention, range-of-motion recommendations may need to be modified to prevent dehiscence of the wound (Lewis, 1994).

Stotts (1986) has reported that involvement in athletic activities is associated with less vulnerability to pressure ulcer development as well as other complications and rehospitalization in individuals with SCI of comparable age, severity of spinal cord injury, and preinjury health. A number of studies involving individuals with SCI have shown that specific exercise protocols can improve level of physical conditioning and overall quality of life, thereby directly preventing development of pressure ulcers. Arm crank ergometry training in paraplegics is reported to have resulted in statistically significant increases in peak oxygen uptake, performance, and mean dynamic strength (Nilsson et al., 1975). Numerous other upper extremity exercise training regimens have resulted in increased pulmonary ventilation (Miles et al., 1982), as well as improved wheelchair sprinting performance (DiCarlo, 1988; DiCarlo et al., 1983; Hooker and Wells, 1992; Taylor et al., 1986; Whiting et al., 1983). These regimens benefit most SCI wheelchair users because activities of daily living—especially those involving transfers and ramps—require intense, short-duration power output.

In individuals with SCI who have lost voluntary control of their lower extremities, computerized functional electrical stimulation (FES) can be used to facilitate leg cycle ergometry and has been shown to produce a number of positive treatment effects. Ragnarsson et al. (1988) and Rodgers et al. (1991) demonstrated improved quadriceps muscle performance attributed to muscle hypertrophy (Neumayer et al., 1997). Other studies involving lower extremity FES on individuals with SCI have resulted in increased cardiac output and stroke volume (Davis et al., 1990; Figoni, 1993), an increase in left ventricular mass in tetraplegics (Nash et al., 1991), and an increase in tibial bone density (Hangartner et al., 1994; Rodgers et al., 1991).

Research by Anderson and Andberg (1979) indicates that individuals with SCI who engage in productive, meaningful, satisfying life activities experience a lesser incidence of pressure ulcers. Such activities could be public events, family gatherings, education classes, employment, volunteer activities, or avocational pursuits.

Spinal-injured individuals should be responsible for directing their care and maintaining control over their environment by communicating instructions to those assisting them in the home or community. Control over these decisions may lessen the stress of feelings of the loss of control and may result in a more positive health status (Sebern, 1996).

8. 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.

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

In today's health-care environment, individuals experience significantly shorter hospital stays. Health-care providers focus on maximizing functional gains in activities of daily living and mobility. Frequently, education is informal or minimal. 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 implemented for the individual with SCI as well as family and personal care attendants. *(See "Patient Education" on page 51.)*

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). 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 once 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 (Liberman and Chaiken, 1992). Assess the individual with SCI's understanding of health-behavior recommendations through direct, specific questions. Identify points needing clarification and provide explanations, giving special attention to changes in lifestyles and daily routines. *(See "Psychological and Social Factors" on page 20.)* 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.

Nutrition

Malnutrition is a major risk factor related to the development of pressure ulcers in individuals with SCI (Maklebust and Magnan, 1994; Vidal and Sarrias, 1991; Wagner et al., 1996). Specific factors that are significantly associated with the development and prolonged healing of pressure ulcers are impaired nutritional status and decreased nutrient intake. Assessment and monitoring of nutritional status can lead to appropriate interventions for both prevention and treatment of pressure ulcers.

9. Assess nutritional status of all SCI individuals on admission and as needed, based on medical status, including:

- **Dietary intake**
- **Anthropometric measurements**
- **Biochemical parameters (prealbumin, total protein, albumin, hemoglobin, hematocrit, transferrin, and total lymphocyte count)**

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

Nutritional status has been correlated with the development and healing of pressure ulcers (Ek et al., 1991; Strauss and Margolis, 1996). However, no single parameter consistently assesses nutritional status or predicts the effect of nutrition on the prevention and treatment of pressure ulcers. Serial measurements to assess trends over time may be useful for estimating baseline nutritional status, then monitoring the response to a nutritional intervention. Recommended evaluations should be interpreted collectively, with consideration given to possible nonnutritional factors such as age, gender, over- or under-hydration, drug-nutrient interactions, physiologic stress, injury, infection, and concurrent illnesses.

Dietary Intake

A dietary history can illustrate the adequacy of an individual's usual food intake. Factors that contribute to inadequate nutritional intakes are poor appetite, food intolerances and allergies, difficulty with chewing and swallowing, difficulty with food acquisition and preparation, immobility, neglect, lack of knowledge, 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 intakes than do those who do not have pressure ulcers (Bergstrom and Braden, 1992). Normal tissue and skin integrity depend on adequate calorie, protein, and vitamin intake (Krouskop et al., 1983).

Anthropometric Measurements

Standard anthropometric measurements include body weight, body mass index (weight/height^2), triceps skinfold (TSF), and mid-arm muscle circumference. Decreased body weight (< 80 percent of ideal weight) and low body mass index (17.6 ± 4.6) have been correlated with severe malnutrition and pressure ulcer development (Bonnefoy et al., 1995; Ek et al., 1991; Strauss and Margolis, 1996). Weight loss reduces fat and muscle tissue, resulting in elevated pressure over bony prominences and increased damage to microcirculation, thus contributing to pressure ulcer development (Schubert et al., 1994). However, ideal body weight standards have not yet been established for individuals with SCI. To estimate ideal body weight, the New York Metropolitan Life Insurance Company tables have been recommended with the following adjustments: for long-term paraplegics, subtract 4.5 to 7 kg from the guideline for a given height and frame size; for long-term tetraplegics, subtract 7 to 9 kg from the recommended guideline (Peiffer et al., 1981). These adjustments attempt to compensate for muscle atrophy when evaluating weight loss in individuals with SCI, but have not been validated in a prospective, randomized study.

TSF measurements are also significantly lower in individuals with pressure ulcers than in those who do not have them (Bonnefoy et al., 1995; Ek et al., 1991). Depleted TSF, defined as < 3.0 mm for females and < 2.5 mm for males, has been significantly associated with pressure ulcer development and longer hospital lengths of stay (Allman et al., 1995). Although mid-arm muscle circumferences were not measured in their study, Maklebust and Magnan (1994) observed that 60 percent of individuals at high risk for pressure ulcers also had decreased muscle mass.

Anthropometric measurements should be interpreted with caution in individuals with SCI. Standards for comparison with individual anthropometric measurements do not account for skin compressibility, fluid status, or recent changes in nutritional intakes. In addition, these standards are based on a healthy 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.

Biochemical Parameters

Table 4 provides blood chemistry tests that may be useful in assessing nutritional status.

TABLE 4
Blood Chemistry Tests for Nutritional Assessment

Blood Chemistry Test	Range of Normal Values*
Prealbumin	19–43 mg/dL
Total protein	6.0–8.0 g/dL
Albumin	3.5–5.0 g/dL
Hemoglobin	Female: 11.5–15.5 g/dL Male: 14.0–18.0 g/dL
Hematocrit	Female: 33–44 percent Male: 39–49 percent
Serum transferrin	200–400 mg/dL
Total lymphocyte count	1500–4000/mm^3

*Normal reference ranges may vary according to the laboratory consulted, the method used, the population tested, and the conditions of specimen collection and preservation.

Several biochemical parameters have been associated with the development or presence of pressure ulcers.

Prealbumin:

Serum prealbumin levels are reportedly extremely low (13.7 ± 3.8 mg/dL) in people with pressure ulcers (Bonnefoy et al., 1995). Prealbumin levels were significantly lower in people with pressure ulcers than in those without. Prealbumin is the most sensitive indicator for monitoring nutritional adequacy due to its short half-life of 2–3 days (Tuten et al., 1985).

Total Protein and Albumin:

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). Serum albumin levels less than 3.5 mg/dL have also 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 stage of a pressure ulcer and significantly associated with lifetime incidence of pressure ulcers, number of different sites, and recurrences (Salzberg et al., 1996).

Possible explanations for these associations are:

(1) low albumin levels may precipitate interstitial edema, causing the skin to become less elastic and interfering with oxygen and nutrient transport from the blood to the skin, thus contributing to pressure ulcer development (Krouskop et al., 1983); and

(2) edematous areas may experience a temporary loss of blood flow caused by increased tissue pressure resulting in tissue damage (Strauss and Margolis, 1996).

A diet rich in calories and protein is recommended to improve serum albumin levels instead of intravenous albumin administration. Intravenous albumin is deficient in essential amino acids, is very expensive, 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 (Bonnefoy et al., 1995; Segal et al., 1997; Strauss and Margolis, 1996).

Hemoglobin and Hematocrit:

Anemia, assessed by hemoglobin and hematocrit levels, reduces oxygen supply to tissues, thus impairing healing of pressure ulcers. Hemoglobin levels below 12.0–14.0 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 percent 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). In addition, in some studies

hemoglobin and hematocrit levels have not been correlated with the development or healing of pressure ulcers, most likely again due to confounding variables (Allman et al., 1995; Day and Leonard, 1993).

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 nonnutritional factors, such as infections and steroid use, that compromise immunocompetence.

10. Provide adequate nutritional intake to meet individual needs, especially for:

- **Calories (or energy)**
- **Protein**
- **Micronutrients (zinc, vitamin C, vitamin A, and vitamin E)**
- **Fluids**

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

Provision and intake of a nutritionally complete, well-balanced diet are necessary for both the prevention and treatment of pressure ulcers. Specific nutritional factors associated with wound healing include calories, protein, zinc, vitamin C, vitamin A, vitamin E, and fluids.

Calories (Energy)

Calories are required to fuel basic life processes and to spare lean body mass from being used to meet metabolic demands. Healthy SCI individuals commonly have lower energy needs than do their ambulatory controls, most likely due to the accompanying decreased physical activity and muscle atrophy. However, individuals with both SCI and pressure ulcers have higher energy needs than do their SCI counterparts who have similar levels of injury but no pressure ulcers (Alexander et al., 1995; Liu et al., 1996). Pressure ulcer surface area has also been found to be significantly related to percent of predicted energy expenditure (Liu et al., 1996).

A possible explanation for increased energy expenditures with pressure ulcers is the underlying chronic inflammatory processes induced by cytokines and cortisol (Bonnefoy et al., 1995; Segal et al., 1997). Decreased thyroxine levels have been observed in SCI individuals, and therefore do not contribute to this hypermetabolic state (Cheville and Kirshblum, 1995).

Predictive equations may be helpful in estimating energy expenditure in SCI individuals either with or without pressure ulcers. Table 5 presents equations that have been reported in the literature.

TABLE 5
Equations for Estimating Energy Expenditures

Predictive Equation	Reference
30 – 35 kcal/kg of body weight/day (with or without pressure ulcers)	Bergstrom et al., 1994
30 – 40 kcal/kg of body weight/day (with pressure ulcers)	Breslow et al., 1993
Harris-Benedict equation (BEE) x stress factor (1.6 for acute spinal cord injury, without pressure ulcers)	Rodriguez et al., 1997
BEE: Females: 655 + (9.6 x weight in kg) + (1.7 x height in cm) - (4.7 x age in years)	
Males: 66 + (13.7 x weight in kg) + (5 x height in cm) - (6.8 x age in years)	
Harris-Benedict BEE x stress factor (1.2 for stage II ulcer; 1.5 for stage III and IV ulcers)	Chin and Kearns, 1997
PARAPLEGIC	
27.9 kcal/kg of body weight/day	Cox et al., 1985
21.4 ± 0.6 kcal/kg of body weight/day (without pressure ulcers)	Alexander et al., 1995
25.9 ± 1.2 kcal/kg of body weight/day (with pressure ulcers)	
TETRAPLEGIC	
22.7 kcal/kg of body weight/day	Cox et al., 1985
20.9 ± 0.8 kcal/kg of body weight/day (without pressure ulcers)	Liu et al., 1996
24.3 ± 1.1 kcal/kg of body weight/day (with pressure ulcers)	

These predictive equations are often inaccurate because of wide variations among individuals. Indirect calorimetry is the best method of determining energy expenditures in SCI individuals who have pressure ulcers (Alexander et al, 1995; Liu et al., 1996). However, this assessment is not available in all settings.

Protein

Protein is essential for tissue growth, maintenance, and repair. High protein intake is needed for optimal healing of pressure ulcers (Allman et al., 1995). To determine the effect of dietary protein on healing of pressure ulcers, 28 malnourished individuals were randomly assigned to

receive standard diets in addition to liquid supplements containing either 24 percent or 14 percent of calories as protein for 8 weeks (Breslow et al., 1993). Results showed that:

(1) protein intake significantly increased from 1.5 g/kg to 2.1 g/kg in the 24-percent group, while no change was seen in the 14-percent group;

(2) total truncal pressure ulcer surface area decreased significantly in the group receiving the 24-percent protein supplement but not in the group receiving the 14-percent protein supplement; and

(3) changes in pressure ulcer surface area correlated with calorie and protein intake per kilogram of body weight.

Significant changes in body weight and in biochemical indices of nutritional status did not occur in either of the two study groups, indicating that calories and protein may be used preferentially for wound healing.

Table 6 presents recommendations for protein intake for individuals with SCI. These recommendations vary according to the presence or absence of pressure ulcers.

TABLE 6
Recommendations for Protein Requirements

Amount of Protein	Reference
(grams of protein/kilogram of body weight/day)	
1.0–1.25 (without pressure ulcers)	Bergstrom et al., 1994
1.25–1.5 (with pressure ulcers)	Bergstrom et al., 1994
1.5–2.0 (with pressure ulcers)	Breslow et al., 1993
1.2–1.5 (stage II ulcers)	Chin and Kearns, 1997
1.5–2.0 (stage III and IV ulcers)	Chin and Kearns, 1997

Further research is needed to quantify protein requirements for prevention and treatment of pressure ulcers in SCI individuals.

Micronutrients

Deficiencies of micronutrients, especially of zinc, vitamin C, vitamin A, and vitamin E, 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. Vitamin and mineral supplementation may be necessary if deficiencies are suspected due to prolonged inadequate intake or increased needs specific to a disease condition. However, supplementation of micronutrients in individuals who do not have deficiencies does not enhance healing of pressure ulcers.

Zinc

Zinc is known to be involved in the structural integrity of proteins, particularly collagen. However, serum zinc levels are reportedly 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).

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 does not accelerate healing of pressure ulcers in individuals who are deficient in vitamin C (ter Riet et al., 1995). In a study by ter Riet et al. (1995), 88 individuals with pressure ulcers were randomly assigned to receive either 500 mg or 10 mg of vitamin C twice daily for a 12-week period. No significant differences in healing rates were observed between the two groups. Optimal wound healing may occur with intakes of 10 to 20 mg of vitamin C daily, the amount needed to prevent the deficiency state of scurvy in humans (ter Riet et al., 1995). Because a subclinical deficiency state is difficult to diagnose, the minimum intake of the Recommended Dietary Allowance (RDA) of 60 mg of vitamin C has been suggested.

Vitamin A

Vitamin A deficiency can result in delayed wound healing. However, no differences have been reported in the vitamin A intake of individuals who did or did not develop pressure ulcers (Bergstrom and Braden, 1992). Vitamin A supplementation is not recommended because of unproven benefits and potential toxicity.

Vitamin E

Anecdotal reports have claimed that supplemental vitamin E reduces the incidence and improves healing of pressure ulcers. Scientific evidence to support these claims, however, is not available. The role of vitamin E in the prevention and treatment of pressure ulcers may be as an

antioxidant, inhibiting free radical oxidation of unsaturated fatty acids in cell membranes, thus contributing to the maintenance of cell wall integrity. Vitamin E may also work synergistically with other antioxidants such as vitamin C to enhance wound healing. Future research needs to address the potential risks and benefits of supplemental vitamin E in individuals who have or are at risk for pressure ulcers.

Fluids

Inadequate fluid intake is reportedly a risk factor in the development of pressure ulcers (Berglund and Nordstrom, 1995; Ek et al., 1991). Individuals who are treated on air-fluidized beds can easily become dehydrated due to increased evaporative water losses (Breslow, 1994). Therefore, all individuals with SCI should be closely monitored for adequacy of fluid intake and signs of dehydration.

11. Implement aggressive nutritional support measures if dietary intake is inadequate or if an individual is nutritionally compromised.

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

Dietary Intake

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. High calorie and high protein foods (e.g., milk, custard, pudding, cheese) can also be included in the individual's diet to enhance intake. When dietary intakes do not meet estimated requirements, more aggressive interventions, such as those explained below, can be used for nutritional support.

Oral Supplements

To supplement an individual's usual diet, several commercial oral supplements are available in the forms of liquids, solids, puddings, and bars (Himes, 1997). Liquid supplements are provided with different nutrient densities, ranging from 1.0 to 2.0 kilocalories per mL, 13 to 25 percent of total calories as protein, and the RDA for vitamins and minerals in approximately 1,000 to 1,500 mL of formula. Flavored breakfast drink powder mixed with milk is an inexpensive nutritional supplement. Liquid supplements in addition to regular meals and snacks have been shown to prevent pressure ulcer development and improve wound healing. In an experimental 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). Actual dietary intakes of the two groups, however, were not recorded during the study, making it difficult to determine the contribution of the liquid supplement to total intake.

Tube Feeding

When the gastrointestinal tract (GI) is functional but dietary intake is inadequate, enteral nutrition through a feeding tube is the preferred method of nutritional support. Use of the enteral route is always preferable to parenteral feedings due to the possible benefits of more physiologic metabolism and utilization of nutrients, maintenance of gut integrity, decreased risk of bacterial translocation, decreased expense of nutrient delivery, and decreased risk of catheter-related infections.

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. Gastric feedings should not be used in individuals who have impaired gag reflex, gastroesophageal reflux, gastroparesis, or gastric outlet obstruction. Jejunal feedings can potentially reduce the risk of pulmonary aspiration.

Tube feeding formulas differ by calorie and protein density, fiber content, form of nutrients, and amounts of micronutrients. (*See "Oral Supplements" on this page.*) 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. Recognition of the causes of and solutions to these complications can enhance formula tolerance and nutrient delivery.

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. The combination of TPN (to provide nutritional needs) and small amounts of enteral feedings (to preserve gut integrity) may be the ideal method for nutritional support for this subpopulation.

Either central or peripheral veins can be used for delivering TPN. Concentrated TPN solutions (> 900 mOsm) are only tolerated through central venous catheters and may cause thrombophlebitis of peripheral veins. Standard TPN solutions can often be ordered from stock order forms available in an institution's pharmacy. Patient-specific formulas can also be designed to deliver individualized nutrient requirements. Complications of TPN include mineral and electrolyte, acid-base disorders, substrate intolerances, and catheter-related infections. Frequent monitoring and meticulous line care are necessary to detect and treat these complications.

Assessment Following Onset of a Pressure Ulcer

Pressure ulcers are most easily treated when diagnosed early and therapy is initiated promptly.

Assessment of the Individual with a Pressure Ulcer

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

- **Complete history**
- **Physical examination and laboratory tests**
- **Psychological health, behavior, cognitive status, and social and financial resources**
- **Availability and utilization of personal care assistance**
- **Positioning, posture, and related equipment**

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

Individuals with spinal cord injury who present with pressure ulcers should have a complete history and physical examination. The etiology and mechanism of ulcer development should be determined. Risk factors should be assessed (*see recommendation 1, page 16*). The contribution of underlying disease processes and comorbidities should be evaluated. Initial evaluations should also include a psychosocial assessment of cognitive status, depression, and other potentially contributory psychological disorders.

Psychosocial supports and resources 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 should also be determined. If the adequacy of available treatment resources can not be assured, delivery of the prescribed treatment plan will be in jeopardy.

Studies have also demonstrated the importance of adequate nutrition in the promotion of wound healing (Ek et al., 1991). Nutritional status assessment should therefore be included in the assessment of individuals with pressure ulcers.

Although assessment of posture, positioning, and equipment is important in determining the causation of pressure ulcers, such assessment is critical in the development of effective treatment and prevention strategies. Check for the individual's posture and level of pressure on the support surface. Evaluate the individual's efficiency in transferring and performing pressure relief and method of short- and long-distance mobility. Support surfaces should be checked for evidence of moisture at the pressure ulcer and for mechanical integrity, wear-out, and fatigue. There is evidence for the above-listed variables as risk factors for pressure ulcers (Lazarus et al., 1994; Maklebust, 1997). (*For supporting references, see "Risk Factors, Risk Assessment, and Risk Assessment Tools" beginning on page 16*).

Assessment of the Pressure Ulcer

13. Describe in detail an existing pressure ulcer. Include the following parameters:

- **Anatomical location and general appearance**
- **Size (length, width, depth, and wound area)**
- **Stage**
- **Exudate/odor**
- **Necrosis**
- **Undermining**
- **Sinus tracts**
- **Infection**

Healing (granulation and epithelialization)

Wound margins/surrounding tissue

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

An objective and thorough description of pressure ulcers enables the development of an appropriate treatment plan, forms 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.

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), routine clinical assessment should include at least a measurement of length, width, and depth. Length measurement should be along the longest dimension of the wound and width measurement 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 edges of the wound. A number of systems have been developed to describe the anatomic depth of pressure ulcers. The National Pressure Ulcer Advisory Panel system is the most commonly used classification system. Other systems have been developed that use more descriptive criteria and possess good interrater reliability (Yarkony et al., 1990). Assessment of wound depth should also include evaluation of sinus tracts, which may communicate with deeper structures. This typically requires the use of radiographic imaging studies such as sinograms.

"Closed ulcers" are characterized by a small skin surface opening in communication with a larger cavity. Although some measurement of dimension 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).

Wound exudate should be characterized by amount, odor, consistency, and color. The presence of necrotic eschar and its appearance should be documented. A black eschar is indicative of dried necrotic tissue, while a yellow covering of the wound surface is indicative of a fibrin slough. A clean, red appearance of the wound base indicates the absence of necrotic tissue.

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. Infection of the wound should also be assessed. Although it is expected that all open wounds are colonized with bacteria, clinically significant wound infection (as distinct from colonization) is felt to be an important cause of delayed wound healing (Stotts and Hunt, 1997). For this reason, cultures should be taken of pressure ulcers if healing is not evident within 2 weeks of initiation of treatment (*see recommendation 23, page 52*). Gross exudate should not be routinely cultured. Tissue biopsy is the most accurate means of determining soft tissue infection. 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.

Tissues surrounding the pressure ulcer should be examined for erythema, warmth, induration, and swelling. These findings 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. The general appearance of the wound may also be important to document irregular borders. Unusual or irregular topography of the wound base, including evidence of sinus tracts, should be documented. Characteristics of the ulcer margin—including margins that are thickened or rolled under the edges—may inhibit epithelialization and should thus be documented.

Although a narrative description of the wound may suffice, photography can also be useful in creating a record of wound appearance. Because the accurate determination of wound edge position is difficult from plain photographs, a marker pen outline of the wound should 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).

Because individuals with dark 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).

Treatment

A comprehensive treatment plan includes assessment of risk, health status of the individual, and status of the pressure ulcer. The elements of a treatment plan include cleansing, debridement, dressings (*see recommendations 14–16*), surgery (*see recommendations 20–22*), nutrition (*see recommendations 9–11*), and management of tissue loads (*see recommendations 28–32*). These elements represent standard treatment procedures as reflected in current literature and practice. However, new research and innovative approaches are being developed in the areas of adaptive therapies.

Nonsurgical

Wound healing is delayed by the presence of high bacterial counts and devitalized or necrotic tissue (Bendy et al., 1964; Daltrey et al., 1981). Anaerobic organisms are indicated by the presence of foul odor. The initial steps to take are cleaning (to reduce bacterial counts) and debridement (to remove necrotic tissue). Topical antibiotics may be used if routine measures do not result in wound healing after several weeks. Broad spectrum agents, such as 1 percent silver sulfadiazine cream, may be used (Kucan et al., 1981), although cross-sensitivity to other sulfonamides may occur. Mupirocin calcium cream 2 percent may be applied for pressure ulcers infected with *staphylococcus aureus* and *streptococcus pyogenes*. Prolonged use may result in overgrowth of nonsusceptible microorganisms, including fungi.

CLEANSING

14. Cleanse pressure ulcers at each dressing change.

- **Use minimum mechanical force when cleansing with gauze, cloth, or sponge.**
- **Use enough irrigation pressure to enhance cleansing without causing trauma to the wound.**
- **Use normal saline or wound cleansers.**
- **Avoid antiseptic agents.**
- **Consider hydrotherapy for ulcers containing large amounts of exudate and necrotic tissue.**

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

Pressure ulcer cleansing is required to reduce bacterial burden and to remove devitalized tissue, metabolic wastes, and topical agents that can retard wound healing. Wound cleansers are described as products that contain surfactants that lower the surface tension, thereby facilitating removal of exudate and other foreign matter. 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 due to its noncytotoxic effects in the wound. Commercial wound cleansers containing surfactants should meet the requirements of the Food and Drug Administration. Health-care providers must be cautioned that although the active agent in a wound cleanser may be noncytotoxic, the inert carrier may be cytotoxic to granulation tissue (Rodeheaver, 1988). Wound cleanser packaging labels should list all ingredients and should provide indications, directions, and warnings. Hellewell et al. (1997) examined the relative toxicity of a variety of wound cleansers and determined that antiseptic wound cleansing solutions exhibited the greatest toxicity.

Although a preponderance of literature can be found on antiseptic cytotoxicity in animal models, 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 believed to be contraindicated for cleansing ulcers due to cytotoxic effects.

Lineaweaver et al. (1985) introduced three topical antibiotics and four antiseptic agents (1% povidone-iodine, 0.25% acetic acid, 3% hydrogen peroxide, and 0.5% sodium hypochlorite) to cultured human fibroblasts and incised wounds in rats (N=20); all four antiseptics were cytotoxic on fibroblasts, and with the exception of hydrogen peroxide, all antiseptics adversely affected tensile strength and epithelialization. Mulliken et al. (1980) also noted that povidone-iodine inhibited tensile strength recovery in incised rabbit wounds. In another in vitro experiment of the effects of three iodine solutions on *Staphylococcus aureus* and *Escherichia coli* on acute animal wounds, Rodeheaver et al. (1982) demonstrated that aqueous iodine and povidone-iodine significantly reduced the wound's resistance to these bacteria. Using a guinea pig model, Niedner and Schopf

(1986) tested seven antiseptics, including povidone-iodine, in incised acute wounds; all antiseptics showed an inhibition of granulation tissue. In a prospective randomized study of 40 inpatients, Kucan et al. (1981) found that povidone-iodine solution was no more effective than normal saline in reducing the level of organisms in open infected wounds over a 3-week period. Cases have been reported revealing increased serum iodide concentrations due to absorption of povidone-iodine used in dressings; this complication has resulted in hyperchloremic acidosis and other electrolyte abnormalities (Aronoff et al., 1980; Dela Cruz et al., 1987; Zamora, 1986).

Delivery systems of cleansing agents vary from gauze sponges to brushes to irrigation. Caution should be exercised if using a gauze sponge or brush because these may inflict tissue trauma. When using these devices, cleanse centrally to peripherally to avoid contamination (Barr, 1995). Strict adherence to universal precautions is advised to protect the caregiver from bloodborne pathogens.

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 irrigation pressure stream. The bulb syringe, with a pressure below 1 psi, is not a satisfactory irrigation device. Battery-powered, disposable irrigation devices can provide pulsatile lavage to loosen wound debris while removing it by suction (Rodeheaver, 1999). Pulsatile lavage may be a comparable alternative to whirlpool therapy to cleanse wounds in the inflammatory state (Barr, 1995). In a controlled trial of 23 individuals with stage III or IV pressure ulcers, Burke et al. (1998) found that a daily whirlpool significantly enhanced wound healing.

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).

DEBRIDEMENT

15. Debride devitalized tissue from pressure ulcers using a method appropriate to the ulcer's status and the individual's condition and goals.

■ **Debride areas in which there is eschar and devitalized tissue.**

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

Debridement is defined as removal of necrotic or infected tissue that interferes with wound healing. Debridement is performed at the bedside or in the operating room. Debridement allows the extent of the ulcer to be determined. It results in a decrease in the bacterial concentration in the wound and, in conjunction with the removal of necrotic tissue, debridement improves wound healing and decreases the risk of the spread of infection, which could cause cellulitis or sepsis (Witkowski and Parish, 1992; Yarkony, 1994).

Table 7 describes several methods of debridement, including autolytic, enzymatic, mechanical, sharp, and surgical. Autolytic debridement occurs under wounds covered with occlusive dressings. It cannot be used on infected ulcers; and it is slow and reserved for individuals who cannot tolerate other methods. Enzymatic debridement uses proteolytic enzymes to remove necrotic tissue. It is slower and is useful in individuals who are not candidates for surgical debridement. Mechanical debridement is accomplished with wet-to-dry dressings or hydrotherapy. It is slow and can be painful and should be discontinued when necrotic tissue has been removed.

Sharp debridement removes only necrotic tissue. It may be done at the bedside by health-care providers according to their scope of practice. Surgical debridement, performed in the operating room, removes large amounts of necrotic tissue. A wide excision of the ulcer may be performed in preparation for closure. Sharp or surgical techniques are the preferred methods of debridement because they most rapidly remove devitalized tissue. Bleeding, the need for anesthesia and its associated risks, and possible injury to nervous or other viable tissue are the main disadvantages. Surgical debridement of necrotic ulcers with sepsis is essential because it rapidly eliminates the source of infection (Galpin et al., 1976). These techniques can be used in any setting.

Choice of a method of debridement is based on the individual's clinical situation and should consider the caregivers' and the individual's satisfaction. These 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.

TABLE 7:
Methods of Pressure Ulcer Debridement

Method	Definition	Indications	Contraindications
Autolytic	A selective method of natural debridement promoted under occlusive or semi-occlusive moisture-retentive dressings that results in solubilization of necrotic tissue only by phagocytic cells and by proteolytic and collagenolytic enzymes inherent in the tissues.	• Individuals on anticoagulant therapy • Individuals who cannot tolerate other forms of debridement • All necrotic wounds in people who are medically stable	• Infected wounds • Wounds of immunosuppressed individuals • Dry gangrene or dry ischemic wounds
Enzymatic	A selective method of chemical debridement that promotes liquefication of necrotic tissue by applying topical preparations of proteolytic or collagenolytic enzymes to those tissues. Proteolytic enzymes help loosen and remove slough or eschar while collagenolytic enzymes digest denatured collagen in necrotic tissue.	• All moist necrotic wounds • Eschar after cross-hatching • Homebound individuals • People who cannot tolerate surgical debridement	• Ischemic wounds unless adequate vascular status has been determined • Dry gangrene • Clean, granulated wounds
Mechanical	A nonselective method of debridement that not only removes foreign material and devitalized or contaminated tissue by physical forces (wet-to-dry gauze dressing, dextranomers, pulsatile lavage with suction or whirlpool), but may also remove healthy tissue as well.	• Wounds with moist necrotic tissue or foreign material present	• Clean, granulated wounds
Sharp	A selective method of debridement using sterile instruments (scalpel, scissors, forceps, silver nitrate sticks) that sequentially removes only necrotic wound tissue without anesthesia and with little or no bleeding induced in viable tissue.	• Scoring and/or excision of leathery eschar • Excision of moist necrotic tissue	• Clean wounds • Advancing cellulitus with sepsis • When infection threatens the individual's life • Individual on anticoagulant therapy or has coagulopathy
Surgical	The most efficient method of debridement. It is nonselective and is performed by a physician or surgeon using sterile instruments (scalpel, scissors, forceps, hemostat, silver nitrate sticks) in a one-time operative procedure. The procedure usually removes most, if not all, necrotic tissue, but may also remove some healthy tissue in what is termed wide excision. Because there may be associated pain and/or bleeding, the individual may require anesthesia, and the procedure will likely require an operating or special procedures room.	• Advancing cellulitus with sepsis • Immunocompromised individuals • When infection threatens the individual's life • Clean wounds as a preliminary procedure to surgical wound closure line. • Granulation and scar tissue may be excised	• Cardiac disease, pulmonary disease, or diabetes • Severe spasticity • Individuals who cannot tolerate surgery • Individuals who have a short life expectancy • Quality of life cannot be improved

DRESSINGS

Historically, dressings are topical products used for protection of a pressure ulcer from contamination and trauma, application of medications, absorption of drainage, or debridement of necrotic tissue. Today, topical dressing products differ from past materials because they are not passive products. Some take part in the healing process, and some are interactive, working with the environment of the wound (Turner, 1997). Bioactive dressings are currently being introduced into the market and are intended to directly or indirectly stimulate healing (Turner, 1997). Further study is needed on these products.

The selection and use of effective and efficient wound-care dressings is determined by issues such as prospective payment, capitation, length of stay, and the increasing caseloads of health-care professionals (Ovington, 1999). 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, the health-care professional's expertise in assessing the ulcer and his/her level of knowledge about dressing functions can have a direct effect on healing outcomes (Arnold and Weir, 1994).

Few randomized controlled trials have been conducted on the myriad dressings on the market. The following recommendations on dressings for pressure ulcers reflect current available literature.

16. Use dressings that will keep the ulcer bed continuously moist and the surrounding intact skin dry.

- **Use a dressing that controls exudate, but does not desiccate the ulcer bed or macerate surrounding tissue.**
- **Loosely fill pressure ulcer cavities with dressing materials to eliminate dead space; avoid overpacking the ulcer.**
- **Monitor the placement of all dressings, especially those in anatomical areas in which they are difficult to keep intact.**
- **Perform dressing changes on a specific schedule based on assessment of the individual, the ulcer, and the condition of the dressing. Consult the dressing manufacturer's package insert for general information about the frequency of dressing changes.**

(Scientific evidence–I/II; Grade of recommendation–A/B; Strength of panel opinion–Strong)

Numerous dressing products have been marketed in the last decade. Currently, some of the major dressings 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 8. In general, the clinical trials of these products were conducted on individuals in nursing homes or hospitals and not on individuals with SCI.

Moist-Wound Healing versus Dry-Wound Healing

Several studies indicate that wounds heal better and faster in a moist environment when compared with dry wound healing techniques (Fowler and Goupil, 1984, Gorse and Messner, 1987; Kurzuk-Howard et al., 1985; Saydak, 1990; Sebern, 1986). Wet-to-dry dressings are not considered continuously moist dressings and should be used for debridement. In a randomized controlled trial, hydrocolloid occlusive dressings were compared with conventional wet-to-dry dressings (Kim et al., 1996). Although there were no statistically significant differences with regard to healing outcomes, healing time was shorter for individuals treated with the hydrocolloid dressing. The wet-to-dry gauze dressings were also found to be more costly than the occlusive hydrocolloid dressings, even without consideration of labor cost (Kim et al., 1996).

Moist-Wound Healing

Other investigators found no significant differences in healing outcomes when moist saline gauze was used compared to other types of moist wound healing (Alm et al., 1989; Colwell et al., 1993; Neill et al., 1989; Oleske et al., 1986; Xakellis and Chrischilles, 1992). Hydrocolloid dressings have been compared with foams (Bale et al., 1997; Banks et al., 1994; Banks and Harding, 1994; Thomas et al., 1997a); foams have been compared to each other (Banks et al., 1997); and hydrogels have been compared to hydrocolloids and saline-moistened gauze (Mulder et al., 1993). No significant differences in healing rate were reported in the literature. Other studies also reported a reduction in caregiver time and overall cost-effectiveness with moisture retentive dressings (Bolton et al., 1997; Phillips and Davey, 1997).

Controlling Exudate

Although dressings should keep the ulcer bed moist, they should not macerate the surrounding intact skin. Excessive exudate can macerate surrounding tissue and it is associated with prolonged healing time (Xakellis and Chrischilles, 1992). Expert clinical opinion suggests that excessive

TABLE 8
Characteristics of Some Major Dressing Categories

Dressing Category and Definition	Indications	Advantages
Transparent Films Clear, adhesive, semipermeable membrane dressings. Permeable to atmospheric oxygen and moisture vapor yet impermeable to water, bacteria, and environmental contaminants.	• Stage I and II pressure ulcers • Secondary dressing in certain situations • For autolytic debridement • Skin donor sites • Cover for hydrophilic powder and paste preparations and hydrogels	• Visual evaluation of wound without removal • Impermeable to external fluids and bacteria • Transparent and comfortable • Promote autolytic debridement • Minimize friction
Hydrocolloids Adhesive wafers containing hydroactive/absorptive particles that interact with wound fluid to form a gelatinous mass over the wound bed. May be either occlusive or semi-occlusive. Available in paste form that can be used as a filler for shallow cavity wounds	• Protection of partial-thickness wounds • Autolytic debridement of necrosis or slough • Wounds with mild exudate	• Maintain a moist wound environment • Nonadhesive to healing tissue • Conformable • Impermeable to external bacteria and contaminants • Support autolytic debridement • Minimal to moderate absorption • Waterproof • Reduce pain • Easy to apply • Time-saving • Thin forms diminish friction
Hydrogels Water or glycerin-based gels. Insoluble in water. Available in solid sheets, amorphous gels, or impregnated gauze. Absorptive capacity varies.	• Partial- and full-thickness wounds • Wounds with necrosis and slough • Burns and tissue damaged by radiation	• Soothing and cooling • Fill dead space • Rehydrate dry wound beds • Promote autolytic debridement • Provide minimal to moderate absorption • Conform to wound bed • Transparent to translucent • Many are nonadherent • Amorphous form can be used when infection is present
Foams Semipermeable membranes that are either hydrophilic or hydrophobic. Vary in thickness, absorptive capacity, and adhesive properties	• Partial- and full-thickness wounds with minimal to moderate exudate • Secondary dressing for wounds with packing to provide additional absorption • Provide protection	• Insulate wounds • Provide some padding • Most are nonadherent • Conformable • Manage light or moderate exudate • Easy to use • Some newer products are designed for deep cavities
Alginates Soft, absorbent, nonwoven dressings derived from seaweed that have a fluffy cottonlike appearance. React with wound exudate to form a viscous hydrophilic gel mass over the wound area. Available in ropes and pads.	• Wounds with moderate to large amounts of exudate • Wounds with combination exudate and necrosis • Wounds that require packing and absorption • Infected and noninfected exuding wounds	• Absorb up to 20 times their weight in drainage • Fill dead space • Supports debridement in presence of exudate • Easy to apply
Gauze Dressings Made of cotton or synthetic fabric that is absorptive and permeable to water and oxygen. May be used wet, moist, dry, or impregnated with petrolatum, antiseptics, or other agents. Come in varying weaves and with different size interstices.	• Exudative wounds • Wounds with dead space, tunneling, or sinus tracts • Wounds with combination exudate or necrotic tissue WET TO DRY • Mechanical debridement of necrotic tissue and slough CONTINUOUS DRY • Heavily exudating wounds CONTINUOUS MOIST • Protection of clean wounds • Autolytic debridement of slough or eschar • Delivery of topical needs	• Readily available • Can be used with appropriate solutions such as gels, normal saline, or topical antimicrobials to keep wounds moist • Can be used on infected wounds • Good mechanical debridement if properly used • Cost-effective filler for large wounds • Effective delivery of topicals if kept moist

Disadvantages	Considerations
• Nonabsorptive • Application can be difficult • Channeling or wrinkling occurs • Not to be used on wounds with fragile surrounding skin or infected wounds	• Allow 1–2 inch wound margin around wound bed • Shave surrounding hair • Secondary dressing not required • Dressing change varies with wound condition and location • Avoid in wounds with infection, copious drainage, or tracts
• Nontransparent • May soften and change shape with heat or friction • Odor and yellow drainage on removal (melted dressing material) • Not recommended for wounds with heavy exudate, sinus tracts, or infections; wounds that expose bone or tendon; or wounds with fragile surrounding skin • Dressing edges may curl	• Characteristic odor with yellow exudate that looks similar to pus is normal when dressing is removed • Allow 1 to 1 1/2 inch margin of healthy tissue around wound edges • Taping edges will help prevent curling • Frequency of changes depends on amount of exudate • Change every 3 to 7 days and as needed with leakage • Avoid in wounds with infection or tracts
• Most require a secondary dressing • Not used for heavily exudating wounds • May dry out and then adhere to wound bed • May macerate surrounding skin	• Sheet form works well on partial-thickness ulcers • Do not use sheet form on infected ulcers • Sheet form can promote growth of Pseudomonas and yeast • Dressing changes every 8–48 hours • Use skin barrier wipe on surrounding intact skin to decrease risk of maceration.
• Nontransparent • Nonadherent foams require secondary dressing, tape, or net to hold in place • Some newer foams have tape on edges • Poor conformability to deep wounds • Not for use with dry eschar or wounds with no exudate	• Change schedule varies from 1 to 5 days or as needed for leakage • Protect intact surrounding skin with skin sealant to prevent maceration
• Require secondary dressing • Not recommended for dry or lightly exudating wounds • Can dry wound bed	• May use dry gauze pad or transparent film as secondary dressing • Change schedule varies (with type of product used and amount of exudate) from every 8 hours to every 2 to 3 days
• Delayed healing if used improperly • Pain on removal (wet to dry) • Labor-intensive • Require secondary dressing	• Change schedule varies with amount of exudate • Pack loosely into wounds; tight packing compromises blood flow and delays wound closure • Use continuous roll of gauze for packing large wounds (ensures complete removal) • If too wet, dressings will macerate surrounding skin • Use wide mesh gauze for debridement and fine mesh gauze for protection • Protect surrounding skin with moisture barrier ointment or skin sealant as needed

Sources: Maklebust, J., and M. Sieggreen. Pressure Ulcer Treatment. In: *Pressure Ulcers Guidelines for Prevention and Nursing Management, 2nd edition*. Springhouse, PA: Springhouse Corporation, 1996: 104–21.

Erwin-Toth, P, and B. Hocevar. Wound Care: Selecting the right dressing. *Am J Nurs* 95 (1995): 46–51.

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.

In a clinical trial on the management of pressure ulcers in individuals with SCI, Chapuis and Dollfus (1990) treated the ulcers with an alginate fiber. The investigators found that the calcium alginate had a high absorption rate, controlled odor, was easy to remove, and conformed to the wound bed. Sayag et al. (1996) compared an alginate wound dressing to treatment with a dextranomer paste. The results indicated that reduction in wound size and the rate of healing were better with the alginate group than with the dextranomer paste. The pharmacological properties of this product need further investigation.

Packing Wounds and Adherence of Dressings

No clinical studies have been reported that address wound packing methods or consequences. However, expert opinion supports the recommendation that wound cavities be filled enough to be effective, but not so tightly as to prevent effective granulation of the wound base. Additional tissue damage may result if wounds are packed too tightly, causing increased pressure on the tissue in the wound bed (Bergstrom et al., 1994).

Several studies have addressed the issues of adherence of the dressing to some anatomical areas as well as the shape of the dressing. Adherence of dressings in the sacral area may be compromised by inherent moisture from perspiration, incontinence, shear, and friction. Dobrzanski et al. (1990) reported that dressings in the sacral area tended to roll when the individual changed positions. Different shapes of hydrocolloid, transparent film, and foam dressings are manufactured to help increase wear time in the sacral area. Some of the newer dressings have beveled edges that help prevent rolling of the dressing edges and that increase adherence to problematic areas. Day et al. (1995) conducted a prospective, randomized, controlled multicenter clinical study to evaluate the effect on safety, efficacy, and dressing performance of different hydrocolloid dressing shapes—triangle or oval—used in stage II and III sacral pressure ulcers. The triangle-shaped dressing group experienced less bunching and wrinkling and longer wear time than the oval-shaped dressing group. These investigators reported difficulties in applying dressings near the anus and recommended that this area be closely monitored.

Individuals with SCI frequently 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).

No scientific studies address the use of rectal pouches to contain the stool and prevent fecal contamination of the dressings and the wound. However, in clinical practice, rectal pouches are used at times to contain the liquid stool and to prevent fecal contamination of the dressing and the wound.

Dressing Selections

Current knowledge about wound-care principles, assessment parameters, and the variety of dressing options enables health-care professionals to select the right dressing for the wound (Baranoski, 1995; Krasner, 1997). The choice of dressing becomes a clinical decision, based on the assessment of the individual, the pressure ulcer, and the overall expected outcome. Today, health-care professionals can match the wound assessment factors with the characteristics of available dressings. This decision is not static. Rather, the health-care professional must be vigilant in recognizing conditions indicative of the need for modifying the treatment plan and must introduce a different dressing. Some circumstances indicating prompt investigation and 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).

Table 8 lists some dressing products that are most often used today. Clinical trials have been conducted on these categories. However, many new products are being developed and information on their use and effectiveness has not been widely disseminated.

ELECTRICAL STIMULATION

17. Use electrical stimulation to promote closure of stage III or IV pressure ulcers combined with standard wound care interventions.

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

Data from three randomized, controlled clinical trials (RCTs) involving a total of 251 SCI individuals, each with at least one wound, supported the efficacy of electrical stimulation (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). In one study, ulcers treated with ES demonstrated significantly greater percentage decreases in size from their pretreatment size than ulcers in the control group at days 5, 15, and 20 (Griffin et al., 1991). These findings are supported by the RCTs of Stefanovska et al. (1993). Baker et al. (1996) also demonstrated significantly better healing rates for wounds treated with ES compared with control wounds. Two other RCTs involving the use of ES on wounds of non-SCI individuals also report significant increases in healing rate compared to control wounds (Baker et al., 1997; Wood et al., 1993). In response to treatment with ES, 20 insensate diabetic foot ulcers demonstrated increased healing rates by nearly 60 percent over control wounds (Baker et al., 1997). A double-blind multicenter study, in which ES below sensory perception was used to treat pressure ulcers, demonstrated that 58 percent of 43 wounds (25 ulcers) healed in 8 weeks, whereas only 1 of 31 ulcers (3 percent) in the control group healed and most other control wounds increased in size (Wood et al., 1993). In addition, the authors of the latter study hypothesized from an experimental study on guinea pigs that ES enhances wound healing by improving growth and differentiation of repair cells in the skin by increasing cell membrane permeability to calcium.

ADJUNCTIVE THERAPIES

Literature reviews were done for several adjunctive wound therapies, including those that used physical forms of energy, such as ultraviolet radiation, low-energy laser radiation, normothermia, ultrasound, subatmospheric pressure therapy, hyperbaric oxygen, topical agents, cytokine growth factors, and nonantibiotic systemic drugs. These reviews did not provide sufficient supporting evidence to justify recommending them for the treatment of pressure ulcers in individuals with SCI.

Ultraviolet and Laser Therapy

Several studies related to the use of ultraviolet (Conner-Kerr et al., 1998; Dodd et al., 1989; Sullivan et al., 1999; Wills et al., 1983) and low-energy laser radiation (Freytes et al., 1965; Kahn, 1984; MacKinnon and Cleek, 1984; Mester et al., 1985; Scott, 1983; Stillwell, 1971; Surinchak et al., 1983) can be found in the literature. No controlled clinical trials involving pressure ulcers were found for ultraviolet or laser therapy, and existing data specific to treatment of pressure ulcers in SCI are minimal.

Normothermia

Recently, clinical trials related to the use of radiant-heat energy to create a normothermic wound environment have been published (Cherry and Wilson, 1999; Kloth et al., 2000; Price et al., 2000; Santilli et al., 1999). The four studies reported positive treatment outcomes; two of them investigated the effect of radiant-heat energy on pressure ulcer healing. In a 4-week controlled trial on fifteen stage III and IV pressure ulcers (seven SCI patients), Kloth et al. (2000) reported a reduction of 61 percent in mean wound surface area for wounds treated with radiant-heat energy. In six control wounds (three SCI patients) treated with standard wound care, a 19-percent reduction in mean wound surface area occurred over the same time period.

In a 6-week prospective, randomized trial involving 50 subjects with stage III and IV pressure ulcers, Price et al. (2000) reported a 54-percent surface area reduction for 25 wounds treated with normothermia versus a 23-percent reduction for 25 wounds treated with standard wound care. Although these findings are encouraging, they do not provide sufficient evidence to support the efficacy of using this therapy for treatment of pressure ulcers in people with SCI.

Hyperbaric Oxygen and Ultrasound

No controlled clinical studies were found for the treatment of pressure ulcers with hyperbaric oxygen administered inside a pressurized chamber with the individual breathing 100-percent oxygen. Two case studies involving the use of topically applied oxygen therapy to treat pressure ulcers were found (Fisher, 1969; Rosenthal and Schurman, 1971). Data from one additional study suggest that topically applied oxygen does not affect tissue oxygen tension below the superficial dermis (Gruber et al., 1970).

The efficacy of using ultrasound for healing pressure ulcers was evaluated in one controlled trial (McDiarmid et al., 1985) that showed a marginally improved rate of healing for infected

ulcers, but no difference for clean ulcers. The lack of controlled studies on pressure ulcers treated with hyperbaric oxygen or ultrasound that demonstrate a significant positive treatment effect prevents the formulation of any recommendation for treatment of pressure ulcers in people with SCI.

Sub-atmospheric Pressure Therapy

The effects of subatmospheric (negative) pressure on pressure-ulcer healing were identified in two studies (Argenta and Morykwas, 1997; Mullner et al., 1997). Mullner et al. (1997) evaluated the efficacy of negative pressure therapy 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 1 SCI individual, were treated with the vacuum technique for 4 weeks. One ulcer achieved primary closure, 8 ulcers granulated and were closed secondarily by grafting, and 3 ulcers decreased in size by 80 percent. Of the 5 individuals with ulcers that did not granulate, 2 died, and 3 had their ulcers closed by flaps.

Argenta and Morykwas (1997) reported on a case series of 141 stage III and IV pressure ulcers. They found that the length of treatment varied directly with the size of the ulcer. Thirty-two percent of the wounds closed completely in 2 to 16 weeks; and 46 percent closed more than 80 percent and were subsequently treated with either skin grafts, muscle flaps, primary closure, or dressing changes. Fifteen percent decreased in size from 50 percent to 80 percent and were either grafted or flapped. Six individuals died of other causes during the protocol period, and 4 individuals did not respond to negative pressure treatment. Thus, the efficacy of negative pressure for promoting closure of stage III and IV pressure ulcers has not been sufficiently established to permit recommendation of this intervention for SCI individuals.

Nonantibiotic Drugs, Topical Agents, and Skin Equivalents

Three papers were identified that evaluated the effects of several nonantibiotic, systemic drugs on wound healing (Adler, 1991; Baron et al., 1982; Olsson, 1980). These reports do not provide evidence to support the use of systemic vasodilators, hemorrheologics, serotonin inhibitors, or fibrinologic agents in the treatment of pressure ulcers. Three papers were identified that investigated the effects of certain topical agents (zinc acetate, aluminum hydroxide, ascorbic acid) on wound healing (El Zayat, 1989; Motta, 1991; ter Riet et al., 1995). These reports do not provide sufficient data to support the use of these topical agents for pressure ulcer treatment. Two clinical studies were found that examined 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 percent 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 stage III or IV pressure ulcers. They observed that granulation tissue developed early and that epithelialization was complete by 7 weeks in all five individuals. There is insufficient evidence at this time to support the efficacy of cultured dermal substitutes for treatment of pressure ulcers in people with SCI.

Growth Factors

Two independent clinical studies examined the effect of homodimeric recombinant platelet-derived growth factor (rhPDGF-BB) on the healing of pressure ulcers. In a randomized controlled trial, Robson et al. (1992a) treated the pressure ulcers of 20 individuals with 1, 10, or 100 μg/mL concentration of rhPDGF-BB or with a placebo gel. After 28 days, only ulcers treated with 100 μg/mL of rhPDGF-BB decreased to a mean of 6.4 percent of their original volume, compared with placebo-treated ulcers that decreased to 21.8 percent of their original volume.

In a followup, multicenter, randomized double blind study, Mustoe et al. (1994) evaluated the effects of two aqueous concentrations of rhPDGF-BB and an aqueous placebo on stage III and IV pressure ulcers in 45 individuals. After 28 days ulcers treated with 300 μg/mL of rhPDGF-BB decreased to 40 percent of their original volume. Ulcers treated with 100 μg/mL of rhPDGF-BB had a mean ulcer volume reduction of 71 percent, and placebo-treated ulcer volume only decreased a mean of 17 percent. The combined findings from Mustoe et al. (1994) and Robson et al. (1992b) provide evidence that rhPDGF-BB enhanced the healing of only 30 pressure ulcers. Recommendation of growth factors for treatment of pressure ulcers in SCI individuals is precluded by the need for additional randomized controlled clinical trials on larger populations that examine the effect of rhPDGF-BB and many other growth factors on pressure ulcer healing.

REASSESSMENT

18. Monitor and assess the pressure ulcer on a consistent and ongoing basis to determine the adequacy of the plan of care.

- **Monitor the pressure ulcer at each dressing change.**
- **Document ulcer assessment at least weekly, and if the condition of the pressure ulcer or the individual changes.**

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Initial wound assessment and reassessment provide the basis for pressure ulcer management. Although randomized clinical trials have not been specifically related to 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. 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 stage, and other variables (van Rijswijk, 1995). Reassessment intervals may vary for individuals in rehabilitation, acute, subacute, extended care, or home-care settings. Whereas the rationale for reassessment of stage II ulcers is to detect epithelialization, the rationale for reassessment of stage III and IV ulcers may be to detect the signs and symptoms of infection and granulation (van Rijswijk, 1995).

19. Modify the treatment plan if the ulcer shows no evidence of healing within 2 to 4 weeks.

- **Review individual risk factors when assessing the healing of pressure ulcers.**
- **Evaluate healing progress using an instrument or other quantitative measurements.**

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

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

Serial measurements should be taken over time to determine the progress of ulcer healing. This quantitative measurement allows the SCI health-care provider to modify the treatment plan if the ulcer is deteriorating, is at a plateau, or is healing. The method selected should be practical and appropriate for the setting.

Instruments may be used to facilitate this evaluative aspect of pressure ulcer management, although reliability and validity have been established with small sample sizes. Three healing instruments will be briefly discussed: Pressure Ulcer Scale for Healing (PUSH), Sessing scale, and Pressure Sore Status Tool (PSST).

The PUSH tool, initiated by the National Pressure Ulcer Advisory Panel, monitors pressure ulcer healing using 13 variables related to measurement, wound margins, undermining, tissue, exudate, and epithelialization (Thomas et al., 1997b). Bartolucci and Thomas (1997) sought to establish a model of three of these variables in 37 subjects. Acknowledging the need for a larger data set, the authors reported that the model demonstrated reasonable discrimination between time points.

The Sessing scale, identifying 0–6 stages, assesses granulation tissue, infection, necrosis, drainage, odor, surrounding skin, and eschar (Ferrell et al., 1995a). The higher the score, the greater the ulcer improvement. In a cohort of 84 nursing home patients with pressure ulcers, there were strong relationships between changes in the Sessing scale compared to changes in ulcer diameter (Ferrell et al., 1995a).

PSST includes 15 indices for wound location, size, stage, edges, undermining or tunneling, necrotic tissue, exudate, surrounding tissue, granulation, and epithelialization (Bates-Jensen, 1995). Parameters are evaluated using a modified Likert scale 1–5; lower scores indicate improved wound status.

Pathophysiology related to wound healing is complex, depending upon multiple variables. Brown-Etris (1995) related that wound healing in superficial partial-thickness stage II ulcers is by regeneration with tissue identical to that replaced. In these wounds, resurfacing (reepithelialization) occurs as a result of epithelial migration to cover the defect. Wound healing in full-thickness stage III and IV ulcers occurs by reparation with inflammation, granulation, matrix formation, and remodeling (Brown-Etris, 1995; Cooper, 1995). Chronic wounds, such as pressure ulcers, do not heal in the same orderly and timely manner seen in acute surgical wounds (Lazarus et al., 1994).

Authors have addressed rates of healing and wound healing times for individuals with SCI (Nussbaum et al., 1994; Salzberg et al., 1995). However, as with literature concerning the non-SCI population, ulcer stages, location, duration, local treatment plans, cleaning method, risk factors, and other variables are not fully addressed. These factors, compounded by small sample sizes, nonran-

domization, and lack of controls, make it difficult to compare pressure ulcer healing relevant to therapeutic modalities (e.g., types of dressings, electrical stimulation).

Additionally, authors use a variety of terms to describe pressure ulcer healing. This diversity of terminology limits the ability to compare data to determine pressure ulcer healing. Nomenclature commonly used includes:

- Wound survival healing rates of wound surfaces; wound closure probability per unit of time (ter Riet et al., 1996)
- Healing efficacy (Sayag et al., 1996)
- Mean or absolute healing rates; relative healing rates (ter Riet et al., 1995)
- Time to healing (van Rijswijk and Polansky, 1994)
- Absolute or percent change in wound area or volume over a given period of time (Margolis, 1994).

Predictors of wound healing must also be considered. In a large retrospective cohort study of 819 individuals with pressure ulcers in long-term care facilities, Berlowitz et al. (1997) determined that significant predictors of healing included pressure ulcer size, older age, and rehabilitation services. Specifically, pressure ulcers were more likely to heal in residents who were over 75, were admitted to long-term care during the previous 4 weeks, were receiving rehabilitation therapies such as occupational therapy, and had stage II or III ulcers as opposed to stage IV. Berlowitz et al. (1997) related that pressure ulcer healing was less likely if the resident was immobile or incontinent, was receiving oxygen therapy, was dependent in any activities of daily living, or had tetraplegia.

Surgical

20. Refer appropriate individuals with complex, deep stage III pressure ulcers (i.e., undermining, tracts) or stage IV pressure ulcers for surgical evaluation. When surgery is indicated, include the following tenets of surgical treatment:

- **Excising of ulcer, surrounding scar, bursa, soft tissue calcification, and underlying necrotic or infected bone**
- **Filling dead space, enhancing vascularity of the healing wound, and distributing pressure off the bone**
- **Resurfacing with a large regional pedicle flap, with suture line away from the area of direct pressure, and one that does not encroach on adjacent flap territories**
- **Preserving options for future potential breakdowns**

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

The goals of surgical closure of pressure ulcers are:

(1) reduce protein loss through the wound;

(2) prevent progressive osteomyelitis and sepsis;

(3) avoid progressive secondary amyloidosis and renal failure;

(4) lower rehabilitation costs;

(5) improve hygiene and appearance;

(6) avert future Marjolin's ulcer; and

(7) reduce time to healing.

In general, pressure ulcers of stages I and II can be treated nonsurgically, while those of stages III and IV usually require a more aggressive surgical approach. The high recurrence rates and long duration to achieve complete healing are often cited as reasons that surgical closure for stages III and IV ulcers may be the most appropriate. Suitable candidates for operative repair of pressure ulcers are those who have clean granulating wounds of stages III or IV. *(See photos 1 and 2)*. There must also be a reasonable chance for improvement, when continuous pressure can be avoided, and a long life expectancy (Casas and Lewis, 1989). Individuals who cannot medically tolerate surgery, have a short life expectancy, and cannot improve their quality of life by operation are seldom candidates for operative wound closure.

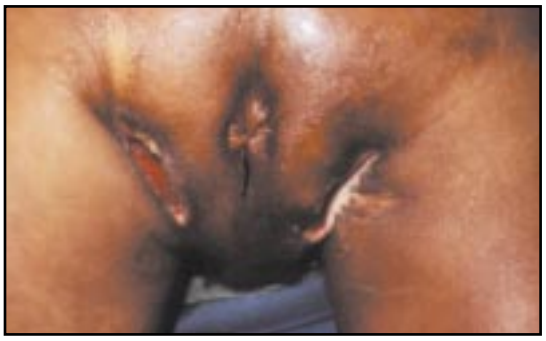


Photo 1: Bilateral ischial stage IV pressure ulcers.

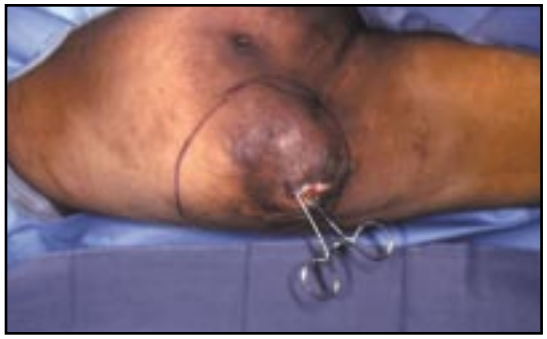


Photo 2: Trochanteric ulcer with extensive undermining.

Operating time may be up to 3 hours, and blood loss may be between 500 and 1,500 ml (Casas and Lewis, 1989). Successful repair is obtainable only with individual participation, and therefore individuals with SCI must exhibit self-motivation and comprehension of the pathogenesis of the ulcers (Stal et al., 1983). However, it is difficult to measure subjective characteristics such as self-motivation, comprehension, quality of life, and probable individual cooperation. Clinical judgment with input by attending health-care providers has to be exercised in making treatment decisions. *(For a discussion of psychological and social factors, see page 20.)*

Although medical knowledge and techniques of muscle and musculocutaneous flap closure have changed, the basic tenets of surgical treatment of pressure ulcers remain essentially unchanged (Buntine and Johnstone, 1988; Conway and Griffith, 1956; Daniel et al., 1979; Ger and Levine, 1976; Hill et al., 1978). Among these guidelines are the following:

- Excise the ulcer and surrounding scar, underlying bursa, and any soft tissue calcification.
- Remove underlying necrotic or infected bone.
- Fill the dead space with fascia or muscle flaps, introduce improved vascularity with flaps and by distributing pressure over bony prominences.
- Resurface with a large regional pedicle flap (a flap should be designed as large as possible, placing the suture line away from the area of direct pressure). *(See photo 3).*
- Use a flap design that does not violate adjacent territory, to preserve all options for coverage in the event of subsequent breakdown or recurrence requiring further reconstruction. *(See photo 3).*

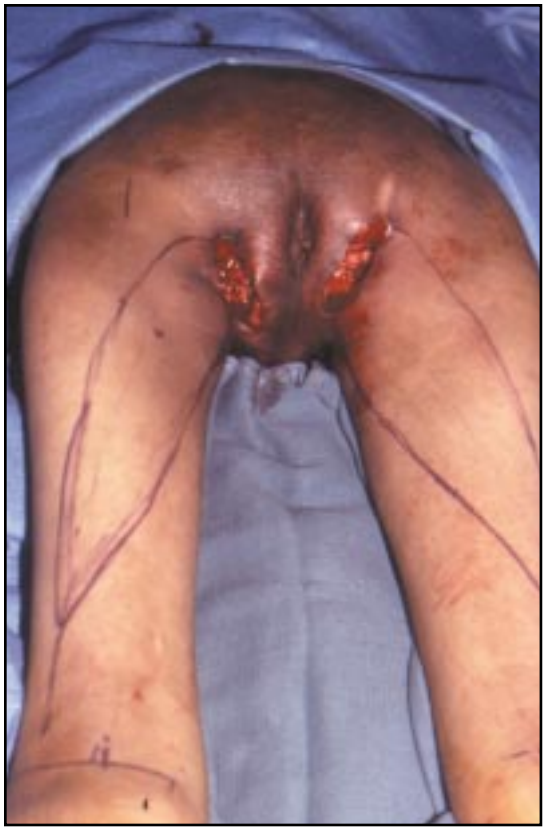


Photo 3: Outline of bilateral posterior thigh V-Y advancement musculocutaneous flap.

Removal of underlying bony prominences as an adjunct to treatment of individuals with pressure ulcers was first reported by Kostrubala and Greeley (1947). The recommendation for excision of sacral promontories and the greater trochanter was subsequently expanded to encompass the entire ischium (Blocksma et al., 1949). Conway and Griffith (1956) reported a decrease in pressure ulcer recurrence from 38 percent after partial ischiectomy to 3 percent following total ischiectomy.

A total ischiectomy does minimize ipsilateral ischial recurrence, but secondary problems often occur. Arregui et al. (1965) evaluated the long-term results of ischiectomy in 94 individuals. A unilateral ischiectomy was performed in 43 individuals and bilateral ischiectomy in 51. In spite of good results in 81 percent, they reported early complications such as hematoma and dehiscence in 16 percent of cases and a 28-percent occurrence of a contralateral ischial ulcer. With one ischium removed, the individual's weight in the erect sitting position is transferred to the opposite side. After bilateral total ischiectomy, weight bearing is transferred to the pubic rami and perineum. With the individual sitting, the pressure that had previously been accepted by the ischial tuberosi-

ties is now borne more directly by the membranous and proximal bulbous urethra leading to perineal ulcers and urethrocutaneous fistulas being reported in up to 58 percent of individuals (Comarr and Bors, 1958; Hackler and Zampieri, 1987; Karaca et al., 1978).

In summary, complete removal of a bony prominence, such as complete ischiectomy, may be protective against long-term local pressure ulcer recurrence. However, there is increased incidence of immediate postoperative complications at the surgical site (such as hematoma and dehiscence) and increased likelihood of developing a contralateral pressure ulcer because of redistribution of pressure to the other side. Thus, radical "prophylactic" removal of bone is no longer indicated. Nonetheless, individuals with proven osteomyelitis or necrosis in the bone immediately underlying the pressure ulcer should be treated with at least partial ostectomy to satisfy the surgical tenet of achieving a satisfactorily debrided wound.

The endpoint of bone debridement should be healthy, bleeding bone, although osteopenia in paraplegics may cloud this goal. Individuals who undergo proximal femoral resection under a trochanteric ulcer experience a pistoning effect of the distal femur that is detrimental to flap survival. However, in the face of infected trochanteric pressure ulcers that communicate to the hip joint, Girdlestone arthroplasty with proximal femoral resection and soft tissue coverage with a vastus lateralis muscle flap is believed to be essential to successful management of those cases (Evans et al., 1993).

PREOPERATIVE CARE

21. Assess, treat, and optimize the following factors preoperatively:

- **Local wound infection**
- **Nutritional status**
- **Bowel regulation**
- **Severe spasms and contractures**
- **Comorbid conditions**
- **Previous ulcer surgery**
- **Smoking**
- **Osteomyelitis**
- **Urinary tract infection (UTI)**
- **Heterotopic ossification**

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

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

Wound Infection

Persistent bacteremia from infected ulcers carries a 50 to 70 percent mortality (Longe, 1986). Adequate wound debridement is an obvious prerequisite to achieving wound closure and perioperative antibiotics are routinely used (Salzberg et al., 1990). Mathes et al. (1983) evaluated the results of muscle flaps to cover 54 infected wounds (following debridement) that had established infection for 6 months and exposed bone or vital structures. Seventeen of these wounds were infected pressure ulcers with underlying outer bone cortical infection. All individuals had wound debridement and excision of the outer exposed bone cortex with immediate coverage with muscle or musculocutaneous flaps. Ninety-three percent of individuals had stable wound coverage without recurrence of infection. It does seem that most pressure ulcer wounds, even in the face of underlying osteomyelitis, can be closed with healing achieved when muscle flaps are utilized in the face of adequate soft tissue and bone debridement.

Nutritional Status

Immobile and debilitated individuals tend to have generalized muscle atrophy and nutritional imbalances (Agris and Spira, 1979; Staas and LaMantia, 1982). It has been recommended that serum protein levels should be above 6g/dl100ml (Agris and Spira, 1979; Herceg and Harding, 1978), hemoglobin above 12 g/dl (Agris and Spira, 1979), and albumin above 3.5mg/dl (Stal et al., 1983). Transferrin levels should be above 180 mg/dl and lymphocyte count above 1500m3 (Lewis, 1990). A positive nitrogen balance should be achieved. While a number of preoperative factors in the past have been assumed to adversely affect the outcome of pressure ulcer repair, recommendations for specific serum levels prior to pressure ulcer closures have not been truly evaluated for their significance on postoperative healing. A low hemoglobin has been thought to be a predictor of poor wound healing (Allman et al., 1995). The standard of care in the 1960s demanded that an individual's hemoglobin be brought up to 10gm/dl with a transfusion of whole blood prior to surgical treatment of a pressure ulcer (Griffith and Schultz, 1961). The reported preoperative hemoglobin range of surgical individuals who had pressure ulcer closure in a recent study was 6.9–16.4g/dl (Goodman et al., 1999). Although the mean hemoglobin count in that group of indi-

viduals was 12.3±2.2g/dl, a specific hemoglobin level was not found to correlate significantly with any of the outcome variables, including those related to wound healing and ulcer recurrence.

The individual's nutritional status has been measured by serum protein levels and inadequate nutritional intake (Griffith and Schultz, 1961). Inadequate nutritional intake has been shown to correlate with new ulcer development (Griffith and Schultz, 1961; Rintala, 1995; Vidal and Sarrias, 1991). Superficial pressure ulcers (stages I and II) treated nonoperatively demonstrated improved outcome and healing when associated with increased protein intake, low lymphocyte count, and small ulcer surface area (Allman et al., 1986). None of these factors, however, was independently predictive of outcome. In a group of surgically treated individuals, serum albumin and total protein levels were used as measures of nutritional status, and it was found that neither level correlated with any postoperative wound healing outcome measurements (Evans et al., 1994; Goodman et al., 1999). Although wound closure is not advised in nutritionally debilitated individuals, a particular serum total protein or albumin level has not been identified that would mitigate against successful wound closure in the surgical individual.

Bowel Regulation

Fecal incontinence must be controlled before surgery (Lewis, 1990). Temporary bowel diversion by colostomy is indicated for individuals with multiple pressure ulcers in close proximity to the anus. This procedure will minimize the risk of flap compromise and infection after surgery. However, in real terms, colostomy in the spinal cord-injured individual is seldom required as a prerequisite to achieve wound healing.

Spasms and Contractures

Hyperreflexia secondary to upper motor neuron lesions may be helpful to maintain some muscle tone and improve the ability to transfer to and from bed, but severe spasticity precludes surgery (Herceg and Harding, 1978). A certain amount of spasticity may also assist in bladder emptying and reflex erection (Herceg and Harding, 1978). However, severe spasms of the lower extremity have led to dehiscence of wound edges following operative repair (Ger and Levine, 1976). Hentz (1979) used oral diazepam or dantrolene to control spasms in 115 individuals who had operative repair of 445 pressure ulcers. Perioperative management has been well summarized by Stal et al. (1983). Their drug of choice for spasticity and reflex spasms was baclofen, but diazepam and dantrolene were also used effectively either adjunctively or alternatively.

Severe flexion contractures may aggravate development of pressure ulcers and also promote recurrence. When contractures are severe, preliminary flexor releases should be done. However, these contractures should not necessarily be released totally because of the risk of vascular compromise from extreme shortening of vein, artery, and nerves across the contracted joints (Haher et al., 1983).

Comorbid Conditions

Cardiac disease, pulmonary disease, and diabetes have all been implicated as factors contributing to poor wound healing. Neither one nor a combination of these conditions significantly correlates with a poor outcome of surgical closure (Goodman et al., 1999). This finding is contrary to a retrospective study published in 1997 (Niazi et al.) of 176 spinal cord-injured individuals in which a positive correlation between cardiovascular disease and ulcer recurrence was found. Nonetheless, all medical comorbid conditions must at least be stabilized before embarking on these major surgical procedures.

Previous Pressure Ulcer Surgery

The published literature with regard to new and recurrent ulcers is also contradictory. 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). Other studies have found that the success of flap closure for pressure ulcers was not affected by previous flap reconstruction (Foster et al., 1997; Goodman et al., 1999). A previous flap reconstruction does not seem to necessarily correlate negatively with any of the surgical outcome variables if an ulcer recurs at the same site (Goodman et al., 1999). However, common sense would dictate that if an individual had multiple previous surgeries for ulcers at different but contiguous sites, flap reconstruction will become more difficult (or may be impossible) because of scar tissue and lack of remaining available flap reconstructive options.

Smoking

Information in the published literature concerning smoking in people with pressure ulcers is contradictory. Carbon monoxide and nicotinic acid in cigarette smoke are potent vasoconstrictors that may compromise arterial flow to the flap used for operative repair. Smoking also increases blood viscosity by stimulating erythocytosis, thrombocytosis, and lymphocytosis, thereby aggravating ischemia (Read, 1984). Some have concluded that smoking leads to a higher recurrence rate (Niazi et al., 1997), while other investigators have found

that a history of chronic smoking did not correlate with increased incidence of recurrence (Goodman et al., 1999; Rodriguez and Garber, 1994). There does not seem to be an increased recurrence rate or complication rate among chronic smokers. However, one cannot support continued smoking in the face of surgery because clearly smoking is known to influence wound healing adversely (Silverstein, 1992). The apparent finding that chronic smokers do not seem to be at greater risk for postoperative complications than nonsmokers most likely results from individuals who are frequently hospitalbound and in bed for at least a 3-week period postoperatively and therefore are obliged to cease smoking during that time. Nicotine patches should not be substituted for smoking because of the long half-life as compared to smoked tobacco (Finchman, 1992).

Osteomyelitis

A negative bone scan rules out osteomyelitis underlying the pressure ulcer, but a positive scan is not always diagnostic of bone infection. Lewis et al. (1988) and Thornhill-Joynes et al. (1986) recommend bone biopsy to definitely diagnose osteomyelitis in the presence of abnormal bone scan. 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 percent and specific in 88 percent. Bone scans and CT scans were expensive and not very sensitive. The most useful single test was needle bone biopsy, with a sensitivity of 73 percent and a specificity of 96 percent. 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.

The presence of osteomyelitis per se, nonetheless, does not result in greater flap failure as has been identified in at least two studies (Bruck et al., 1991; Thornhill-Joynes et al., 1986). However, inadequate surgical debridement followed by attempts to sterilize the infected and necrotic bone with prolonged antibiotic use will allow osteomyelitis to progress (Deloach et al., 1992) and will impair surgical outcome.

Urinary Tract Infection

Spinal cord-injured individuals have a higher than normal risk of urinary tract infections. Due to the risk of sepsis that UTIs generate, adequate preoperative management is essential.

Heterotopic Ossification

Heterotopic ossification may develop in the knees, shoulders, elbows, hips, and spine and may restrict movement, aggravating the propensity to develop pressure ulcers. 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). 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.

POSTOPERATIVE CARE

22. Be cognizant of postoperative care procedures.

- **Position the individual in a manner that keeps pressure off a fresh surgical site.**
- **Use an air-fluidized bed when pressure on the surgical flap is unavoidable.**
- **Progressively mobilize the individual to a sitting position over at least 4-8 weeks to prevent reinjury of the ulcer or surgical site.**
- **Provide subsequent patient education on pressure management and skin inspection.**

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

Positioning and Support Surfaces

Successful postoperative management depends on two important conditions: (1) modifying the factors that contribute to ulcer formation, and (2) teaching "pressure consciousness" to the individual. It is important that no pressure be applied to the site after surgery. The individual should be placed on an air-fluidized bed or on a static support surface when the individual can be maintained on a turn regimen that does not apply pressure to the operative site (Black and Black, 1987). If placed on an air-fluidized 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. Complications of a prone position include confusion, boredom, airway difficulties, especially in tetraplegics, and ulnar nerve compression (Nath and Taylor, 1979). Constipating medications may on occasion be administered and a low-fiber diet may be used to avoid fecal contamination of the surgical site (Black and Black, 1987; Rubayi et al., 1990). Prophylactic antibiotics are commonly used in postoperative care (Salzberg et al., 1990), or else antibiotics appropriate to the immediate preoperative wound cultures can be employed. However, there are no research data to report the efficacy or duration of prophylactic antibiotics.

Mobilization

Timing and degree of postoperative mobilization after reconstruction vary according to the specific protocol in a given center. Bed rest should be maintained postoperatively to allow sufficient time for the development of adequate tensile strength to the healing wound before mobilization. Stal et al. (1983) began sitting individuals 6 weeks after surgery and gradually increased the sitting times over a 2-week period. Rogers and Wilson (1975) at Rancho Los Amigos Medical Center in Downey, California, monitored tissue hyperemia with interval thermister probes in order to gauge the rate of postoperative mobilization and arrived at the following protocol:

1. Operative site: complete rest for 6 weeks.
2. After 6 weeks of satisfactory healing, begin passive range-of-motion exercises in preparation for sitting. If the individual demonstrates markedly prolonged temperature rises around the operative site, then progress will be slower.
3. As hip flexion of 90 degrees is obtained, initiate a progressive sitting program over a 2 1/2 week period until the individual can sit for up to 5 hours.
4. Overall mobilization time from operation: 8 1/2 weeks.

Hentz (1979) had a faster remobilization sequence, beginning sitting the individual who had an uncomplicated course at 3 to 4 weeks postoperatively. Individuals are taught to shift body weight once they are bearing weight on a flap and inspecting the skin with a long-handled mirror (Black and Black, 1987; Di Pirro, 1975).

Patient Education

Education is the mechanism for translating knowledge into effective strategies for prevention and treatment. If education materials and programs are to be effective, they must be relevant to the target audience (Bergstrom et al., 1994). Successful patient education motivates the individual to take responsibility (Rottkamp, 1976). Assessing knowledge and cognitive abilities, planning short- and long-term objectives and teaching strategies, using understandable language, and evaluating the extent to which the learning objectives are achieved are the elements of a therapeutic environment in which successful learning can take place (Rottkamp, 1976). However, even under the most ideal of circumstances, compliance with recommended practices may not be assured.

Contemporary approaches to pressure ulcer management emphasize prevention through education (Bergstrom et al., 1992; Bergstrom et al., 1994). However, most pressure ulcer prevention educational programs are designed for an inpatient general population during initial rehabilitation; rarely is there a mechanism to adequately evaluate the information that has been retained, the behaviors that are practiced, and the perceived value of the prevention strategies in the individual's daily life. Andberg et al. (1983) found that when the individual was an active participant in the rehabilitation and educational processes, there was greater compliance and success in treatment. Maklebust and Magnan (1992) stressed the need to assess the individual's characteristics that influence the teaching-learning process and to test the learner's knowledge and skills.

Chapman et al. (1991) advocate encouraging personal responsibility and individualization of treatment if behavior is to be changed. According to these investigators, noncompliance results when the individual does not believe that the recommendations will help the problem and fears that they will interfere with lifestyle. The investigators suggest further that positive participation will be enhanced if the treatment is individualized to the individual's lifestyle, the regimen is easy to follow, positive reinforcement is provided, support systems are enhanced, and a care agreement that delineates the individual's responsibilities is developed. The person's motivation to comply may depend on beliefs about his/her susceptibility to the problem or about the severity of the problem (Eriksen et al., 1988).

Dai and Catanzaro (1987) pioneered use of the Health Beliefs Model to describe factors that predict adherence to skin care regimens. Their results suggested that skin care education might

be more effective in increasing compliance if it emphasizes information on severity, specific knowledge and techniques, and evidence of efficient skin care. Furthermore, the success of health educational programs is in direct proportion to the degree to which individuals are involved in the process: defining their needs, setting their own priorities, controlling solutions, and evaluating their own progress (Green, 1986).

Complications of Pressure Ulcers

Nonsurgical

23. Identify the presence of tissue and/or bone infection.

- **Obtain quantitative tissue and/or bone cultures in ulcers not responding to routine therapeutic measures.**
- **Obtain a tissue and/or bone biopsy to confirm infection, if necessary.**

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

The clinical signs of infection include erythema, induration, purulence, and foul odor. Swab cultures are not useful in determining the presence of infection in pressure ulcers. They will only reflect the surface bacteria and not the bacteria within the tissue (Rousseau, 1989). If the bacterial count exceeds 10^5 organisms, wound healing will be impaired. The appropriate method of culture is best determined by tissue biopsy (Sapico et al., 1986). Results may vary depending on the site of the lesion biopsied. The irrigation aspirations technique for deep culture will have high concordance with the results obtained via biopsy (Ehrenkranz et al., 1990).

Management of cellulitis, osteomyelitis, and sepsis requires antibiotics. Common causes of sepsis include *Bacteriodes fragilis*, *Staphylococcus aureus*, and gram-negative rods. An appropriate medical workup includes medical history, physical examination, blood cultures, and diagnostic testing to rule out other sources of infection. In a septic individual with a pressure ulcer, early debridement is essential to remove the necrotic tissue that is serving as a nidus for infection (Bryan et al., 1983; Galpin et al., 1976).

Delayed wound healing and recurrence of pressure ulcers may result from underlying osteomyelitis. A recent study indicates that if there is an erythrocyte sedimentation rate of 120mm, a higher white cell count of 15,000/mm3, and plain x-rays that are all positive, there is a high predictive value for osteomyelitis (69 percent). If all three are negative, there is a 96-percent probability that there is no disease present (Lewis et al., 1988).

Bone biopsy remains the definitive method (gold standard) for diagnosis and allows identification of the offending organism (Sugarman, 1987). Nuclear medicine bone scans Technetium—99m are only of value if negative. When negative, osteomyelitis is extremely unlikely. When osteomyelitis is confirmed by bone biopsy, appropriate postoperative antibiotics are generally continued for 6 weeks.

Long-standing ulcers, usually present for 20 years or more, can develop a squamous cell carcinoma, known as Marjolin's ulcers (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).

24. Identify the potential complications of immobility associated with pressure ulcer management and implement preventive and therapeutic measures for:

- **Nutritional deficiencies and dehydration**
- **Decreased range of motion**
- **Deconditioning (cardiopulmonary, cardiovascular, musculoskeletal)**

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

The presence of pressure ulcers can result in losses in protein and fluid into the pressure ulcer exudate, thus aggravating the conditions of malnutrition and hypoalbuminemia (Allman et al., 1995). Pressure ulcers can also induce chronic inflammatory processes, which increase systemic hypermetabolism and possibly result in loss of weight and lean body mass (Bonnefoy et al., 1995; Segal et al., 1997; Strauss and Margolis, 1996). *(See recommendations 7 and 9–11 for detailed recommendations and supporting rationales related to nutritional deficiencies.)*

25. Manage hypergranulation tissue that may impede ulcer healing.

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

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. Kerstein (1995) suggested surgical removal, the use of silver nitrate sticks, and semi-occlusive or nonocclusive dressings that dry the wound to remove or diminish hypergranulation tissue.

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).

26. Identify the potential psychosocial impact of pressure ulcers and immobility and provide referral for therapeutic interventions based upon the individual's characteristics and circumstances. Refer to appropriate resources for problem resolution, including:

- **Vocational rehabilitation services**
- **Peer counseling and support groups**
- **Formal psychotherapy and/or family therapy**

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

Krouskop et al. (1983) reported on the development and evaluation of a comprehensive tissue pressure-management program designed to reduce pressure ulcer occurrence. The program focused on effective patient assessment, individualized equipment prescriptions, and increased awareness by the individual and his/her family of their responsibilities for pressure ulcer prevention. Prior to 1974, 32 percent of individuals who came to outpatient plastic surgery clinics of major rehabilitation hospitals in the Southwest returned with a recurrent breakdown within 24 months of discharge. The recurrence rate dropped to 11 percent with the implementation of a multidisciplinary pressure ulcer prevention clinic (1977), and to 9 percent after 1978. Formalized referrals for psychological counseling were introduced in 1980, further reducing the recurrence rate to 4 percent.

Cross-sectional research by Krause (1998) indicated an association of pressure ulcers with unemployment in a sample of 1,017 people with SCI, though prospective research would be necessary to determine if a pressure ulcer leads to loss of employment, or conversely if employment (or the ability to be employed) reduces pressure ulcer risk.

Among Krause's (1998) SCI subjects, those who had pressure ulcers reported less satisfaction with interpersonal relations and activities; again, due to the cross-sectional design, the direction of the effects is unknown, though Krause's speculations on a bidirectional relationship are logically supportable. SCI subjects of Gordon et al. (1982) who had experienced a pressure ulcer during their inpatient rehabilitation reported poorer social adjustment on a questionnaire administered 1 to 4 years later.

Among Krause's (1998) SCI subjects, those who had pressure ulcers reported more negative emotions. In Harding-Okimoto's (1997) very small sample of people with SCI, those with pressure ulcers reported and described more negative images of themselves. Recognizing the major role of depression in contributing to secondary medical complications including pressure ulcers, readers are referred to the clinical practice guideline titled *Depression Following Spinal Cord Injury: A Guide for Primary Care Professionals* and its companion consumer guide, *Depression: What You Should Know*. (Both publications are available via PVA's website, www.pva.org, or they can be ordered by calling the PVA Distribution Center at (888) 860-7244.)

Surgical

27. Identify potential complications of surgical intervention, including:

- **Wound dehiscence/wound separation**
- **Delayed infection and abscess**
- **Hematoma and seroma**

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Wound separation may occasionally occur acutely, even as soon as in the postoperative recovery room. Such a wound separation in the first few postoperative days is invariably due to mechanical factors from uncontrolled individual spasms or shear resulting from individual positioning and turning.

A wound separation that results after 5 days may be due to one or more of a number of causes.

Ischemia at the flap edges may result in marginal wound necrosis, which will manifest as an eschar and black rim to the edge of the wound. Consequently, sutures will not hold well, and progressive wound separation may develop. Most commonly, late wound separation is a harbinger of a more deeply located problem.

A deep infection may result in a seropurulent drainage through the suture line, leading to a progressive breakdown of the wound edges. The individual may have systemic manifestations, such as a fever, and there may be local wound swelling, erythema, and warmth. However, frequently the local and systemic signs of a deeper wound infection are not very apparent other than the seropurulent drainage. A postoperative hematoma deep to the flap will initially congeal, but after about 10 days will liquefy and a sanguineous very dark drainage may exude from the suture line and lead to wound breakdown.

Early wound separation due to mechanical factors can often be treated by returning the individual to the operating room to resuture the wound. Later wound separations, accompanied by seropurulent or sanguineous drainage, are best treated with dressings and local wound care. Once the residual wound has been cleansed, the individual can often be treated by a return to the operating room and a readvancement of the original flap to close the wound.

Postoperative wound infections following flap surgeries are frequently insidious. Local and systemic signs are often not very obvious. The most frequent manifestation is a late wound dehiscence at about 7 days, with exudation of seropurulent material that is thick and pasty in consistency from the suture line. The wound then needs to be opened further to ensure adequate drainage and to enable dressing changes to be done. This can frequently be performed at the bedside.

A hematoma may develop in the early postoperative period. Frequently it is difficult to diagnose because a hematoma is situated deep to the flap. Excessive bright red bleeding of the wound edges, excessive bloody drainage from the drainage tubes, or excessive swelling of the flap may indicate a postoperative hematoma. It should be treated by returning the individual to the operating room to evacuate the hematoma and control any residual bleeding.

Support Surfaces and Positioning for Managing Tissue Loads

Spinal cord injuries significantly affect an individual's ability to move and maintain changes in position. Involuntary muscle movement (i.e., spasticity) complicates the lack of mobility both in bed and in the wheelchair. Constant pressure from the body's weight causes compression of the tissues between an individual's bones and the surface on which the individual is positioned. Restriction of movement by an individual is one of the most common reasons for pressure-related circulatory complications.

Recommendations for using positioning devices for the bed and wheelchair are based on an individualized assessment of the status of the person with SCI, his or her risk profile, and the presence of a pressure ulcer. The use of these devices is not static. Rather, most likely they will be changed over time. *(See recommendations 5 and 6 starting on page 24.)*

Bed Positioning

28. Use bed-positioning devices and techniques to prevent and treat pressure ulcers. Use devices and techniques that are compatible with the bed type and the individual's health status.

- **Avoid positioning individuals directly on a pressure ulcer.**
- **Avoid positioning individuals directly on the trochanter.**
- **Use cushions and positioning aids to relieve pressure on 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.**
- **Limit the amount of time the head of the bed is elevated.**
- **Develop, display, and use an individualized positioning regimen and repositioning schedule.**

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

Interrupted or reduced pressure on the ulcer will reduce ischemia and will likely improve tissue healing (Bergstrom et al., 1994). In experimental positioning evaluations, the measurements of transcutaneous partial pressure of oxygen and interface pressure indicated low-oxygen partial pressure and high-interface pressures when individuals were positioned side-lying directly on the trochanter (pelvis 90 degrees to horizontal surface). The trochanter interface pressure is the highest, and positioning on it should be avoided (Garber et al., 1982; Seiler et al., 1986). When individuals were repositioned at a 30-degree side-lying angle to horizontal, the interface pressure was reduced (Garber et al., 1982). *(See Figure 2.)* In a similar study, Seiler et al. (1986) found that the transcutaneous partial pressure of oxygen returned to normal.

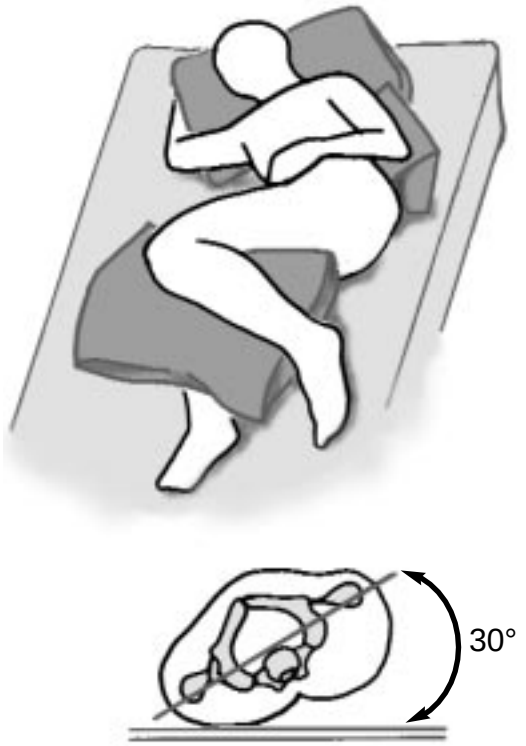


Figure 2: Side-lying at a 30-degree angle.

Application of positioning devices is an effective means of raising the ulcer off the support surface. The 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). Application of positioning devices should also maintain postural alignment and prevent postural deviation. Ring cushions (donuts) 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 prevent pooling of the blood in the center of the wound.

Many published reports have shown the effect of pressure intensity and duration to cause tissue ischemia and ulcer formation *(see "Pathophysiology of Pressure Ulcers" on page 13)*. Thus, any plan implemented to reduce pressure intensity and length of exposure will promote tissue health and ulcer healing. Individual repositioning at planned, displayed intervals will enhance caregiver compliance even when individuals are using pressure-reducing support surfaces. Individuals with SCI and their caregivers should be educated regarding their optimal turning schedule as part of the development of their home program.

Some reports indicate the development of pressure ulcers in people who are being treated for pressure ulcers (Allman et al., 1995; Rochon et al. 1993) and who are using pressure-reducing support surfaces of various types (Jesurum et al., 1996; Ooka et al., 1995). The results indicate the need to maintain careful monitoring and preventive positioning while using therapeutic support surfaces and treating pressure ulcers at all times.

Bed Support Surfaces

29. Use pressure-reducing bed support surfaces for individuals who are at risk for or who have pressure ulcers.

- **Select a static support surface for individuals who can be positioned without weight bearing on an ulcer and without bottoming out on the support surface.**
- **Select a dynamic support surface if the individual cannot be positioned without pressure on an ulcer, when a static support surface bottoms out, if there is no evidence of ulcer healing, or if new ulcers develop.**
- **Use low-air-loss and air-fluidized beds in the treatment of pressure ulcers if one or more of the following conditions exist:**
  - **Pressure ulcers on multiple turning surfaces**
  - **Compromised skin temperature and moisture control in the presence of large stages III or IV pressure ulcers**

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

TABLE 9:
Typical Characteristics for Classes of Support Surfaces

PERFORMANCE CHARACTERISTICS	SUPPORT DEVICES					
	Dynamic Supports			Static Supports		
	Air-Fluidized	Low-Air-Loss	Alternating Air	Static Flotation (air or water)	Foam	Standard Mattress
Increased support area	Yes	Yes	Yes	Yes	Yes	No
Pressure reduction	Yes	Yes	Yes	Yes	Yes	No
Shear reduction	Yes	questionable data	Yes	Yes	No	No
Low moisture retention	Yes	Yes	No	No	No	No
Reduced heat accumulation	Yes	Yes	No	No	No	No
Transfers	Difficult	Routine	Routine	Routine	Routine	Routine
Cost per day	Highest	High	Moderate	Low	Low	Lowest

Adapted from Bergstrom, N., M.A. Bennett, C.E. Carlson, et al., *Clinical Practice Guideline Number 15: Treatment of Pressure Ulcers.* Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, 1994. AHCPR Publication 95-0652.

The individual circumstances of the person with a pressure ulcer must be considered in the prescription of a specialized bed or support surface. These devices may impact the ability to perform functional activities or affect the ability to provide core assistance. These devices may not be appropriate in all home settings due to factors such as weight or operating costs. The availability of these devices is often subject to approval by third-party payers and guidelines established by government agencies.

Many reports indicate that specialized support surfaces will provide an environment in which existing ulcers improve (Charles et al., 1995; Economides et al., 1995; Ferrell et al., 1993) and new ulcer formation is prevented (Blaylock, 1995). In an extensive review of the literature, however, no compelling evidence was found that any specialized support surface performs better than others under all circumstances (Bergstrom et al., 1994). Thus, the health-care provider should consider several factors when selecting a support surface. Consideration should be given to the individual's condition, characteristics of the support surface, type of care setting, available resources, and preference of the individual and the caregiver. Typical performance characteristics of the major types of support surfaces are shown in Table 9 (Bergstrom et al., 1994). Table 10 outlines the common applications, advantages, and disadvantages of the major types of support surfaces.

Static supports are a reasonable choice for individuals with complete immobility or limited mobility or a pressure ulcer and one impairment of nutrition, sensory perception, circulation, or incontinence. Statistically significant differences in outcomes between static support surfaces have not been demonstrated and thus the choice of the support is left for the individual and the caregiver.

The condition of "bottoming out" occurs when a mattress overlay, support, or wheelchair seat cushion is compressed by high pressure. 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 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 mattress overlay, support, or seat cushion is less than 1 inch thick, the prominence has bottomed out. *(See Figure 3.)*

Dynamic supports are options for individuals with stage II pressure ulcers on multiple turning surfaces and a failure to heal on a static support. Similarly, the presence of a large stage III or IV pressure ulcer or a recent tissue graft for ulcer repair may also suggest the use of dynamic supports. (Charles et al., 1995; Day and Leonard, 1993). Good evidence exists for more effective treatment outcomes from the use of dynamic supports than static supports (Ferrell et al., 1993).

In an acute care setting, the healing of large pressure ulcers has been shown to benefit from the use of air-fluidized (high-air-loss) and low-air-loss beds (Bergstrom et al., 1994). The application of air-fluidized beds for home care should be considered in light of their large size, heavy weight, and high power consumption, and the difficulty encoun-

TABLE 10:
Support Surfaces

Major Types	Common Applications	Advantages	Disadvantages
Static Support Surface	• Pressure ulcer prevention • Individual may be kept off pressure ulcer	• Reduces interface pressure • Is cost-effective when properly matched to individual • Does not consume power	• May result in shearing • Moisture and heat build up
Alternating Pressure Surface	• Individual who requires more pressure reduction than a static mattress	• Relieves pressure intermittently	• Intermittent elevated pressure • Moisture retention possible
Low-Air-Loss Surface	• Individual who has more than 1 turning surface impaired due to multiple pelvic pressure ulcers or other factors	• Reduces interface pressure • Manages moisture and heat	• Shear reduction depends on design; possible noise • Complicated activities of daily living maneuvers and transfers
Air-Fluidized Bed	• Post-op flap surgery • Deterioration of multiple pelvic pressure ulcers	• Reduces interface pressure below capillary closing pressure • Manages moisture	• Most expensive • Vulnerable to respiratory or dehydration problems • Premature drying of moist dressings • Significant electric energy requirement • Noise • Limited ability to elevate head of the bed

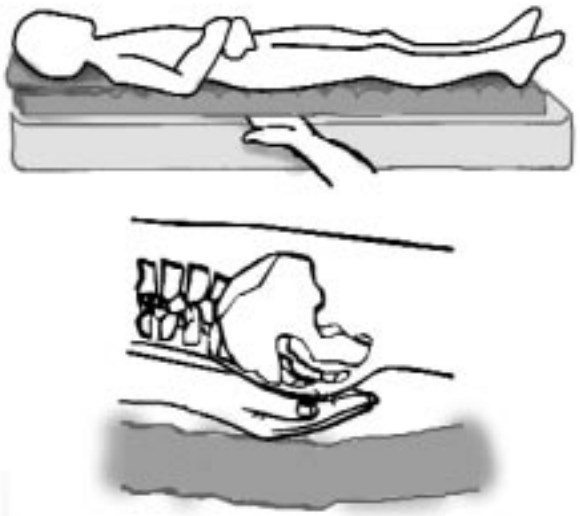


Figure 3: Hand Check of Bottoming Out

tered with individual transfers. In contrast, low-air-loss beds have been easily managed and were effective in pressure ulcer prevention and healing due to their reduced bulk and easier individual positioning and transfer (Charles et al., 1995; Ferrell et al., 1993; Mulder et al., 1994).

Moisture accumulation in the skin is an important factor predisposing to the occurrence of pressure ulcers. Incontinence fluids particularly contribute to the risk of further tissue breakdown (Allman, 1989; Allman et al., 1986).

Moist skin is more likely to abrade and blister (Bergstrom et al., 1994). Use of low-air-loss support surfaces reduces the interface pressure and effectively controls moisture (Scales et al., 1974). The flowing air evaporates skin moisture and keeps a drying environment with reduced temperatures and pressure (Flam et al., 1995) while maintaining a microclimate conducive for tissue healing.

Wheelchair Positioning

30. Prescribe wheelchairs and seating systems according to individualized anthropometric, ergonomic, and functional principles.

- **Obtain specific body measurements for optimal selection of seating system dimensions.**
- **Measure the effects of posture and deformity on interface pressure distribution.**
- **Prescribe a power weight-shifting wheelchair system for individuals who are unable to independently perform an effective weight shift.**
- **Use clinical judgment as well as objective data in determining the compatibility of the individual's body shape with the seating system.**

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

Significant reduction in sitting force can be obtained by using armrests. The armrests support 10 percent of the body weight (combined weight of arm and hand), thereby relieving seating force over the buttocks (Gilsdorf et al., 1991). Wheelchair features that optimize the person's independence in weight shifting, transferring, and propelling, as well as provide postural support, are recommended. Providing spinal-injured people with seating systems that meet individual needs, with emphasis on achieving the highest level of functional and mobility independence, should be the goal (Garber and Krouskop, 1997). 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).

Wheelchairs not only provide mobility, but they can also provide an independent means of performing weight shifts (Nixon, 1985). The purpose of these mechanical pressure relief systems are:

(1) to allow for altering sitting pressures and for change in body pressure distribution to assist in the prevention of pressure ulcers;

(2) to allow for the change of postural alignment while sitting; and

(3) 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 buttocks onto the back. Hobson's (1992) research has shown that a minimum of 45 degrees of tilt/rotation is required for adequate pressure distribution. 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 relief 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 will also reduce pressure without causing shear (Goossens et al., 1997).

Other weight-shifting systems work by reclining the wheelchair back and elevating the legs to approximately horizontal position, helping to redistribute weight-bearing pressure over a larger surface. 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. No evidence was found in the literature to substantiate whether a tilt-in-space or a reclining wheelchair is more effective in preventing pressure ulcers.

Maintaining good postural alignment can facilitate equal weight bearing over the bony prominences of the buttocks (Krouskop et al., 1983). Unequal or excessive pressure over bony areas can contribute to pressure ulcers (Bergstrom et al., 1992). A "plumb line" posture (alignment of the ear/shoulder/hip) keeps normal spinal curves intact. Postures such as slouching forward or leaning to one side place unequal pressure over the buttocks.

Wheelchair footplates need to be level in height and elevation. Footplates that are too high or too low can result in suboptimal sitting pressure distribution between the thighs and the ischial tuberosities. Adjust foot-plate height to ensure that the pelvis is level and the thighs are horizontal. 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.

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 spinal-injured individual must monitor the level of spasticity and seek medical guidance when it becomes detrimental to adequate skin care and function.

An individual physical and functional assessment is recommended for an insensate spinal-injured individual to match the most appropriate seating system with the pressure prevention and functional qualities necessary (Beer, 1984; Lowthian, 1993; Rosenthal et al., 1996). Clinically useful computerized systems have been developed to evaluate the pressure exerted on various cushions. Most 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, and an ability to compare these values in a static and dynamic position (Barr, 1991; Salcido et al., 1996). A limitation of these systems is their inability to measure shear stress (Burman, 1993; Henderson et al., 1994; Salcido et al., 1996). A new sensor intended to measure local shear force is under development and is described by Goossens et al. (1997).

Factors to be considered during a cushion evaluation include pressure- and shear-reducing qualities, comfort, postural support, functional activity level, heat-reducing properties (Knox et al., 1994), adaptability, cost, care, and maintenance

needs (Krouskop et al., 1983). Even the best cushion does not alleviate the need for pressure reduction behaviors. Basta (1991) commented that "in the home or community setting, the SCI individual must be knowledgeable of the proper use and care of the cushion in order for it to provide the desired skin protection properties." The cushion will work well only if it is properly used, maintained, and replaced.

As an individual ages, changes in the skin may increase the vulnerability to pressure ulcer development. Research indicates that the skin tends to become thinner as an individual ages (Knox et al., 1994; Waterlow, 1996). For this reason, a routine cushion reassessment 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).

31. Evaluate the individual's postural alignment, weight distribution, balance, stability, and pressure relief capabilities to establish a proper sitting schedule.

- **Avoid positioning the wheelchair-seated individual directly on a pressure ulcer.**
- **Allow limited sitting in individuals capable of performing weight shifts every 15 minutes.**
- **Reposition the wheelchair-seated individual at least every hour; if this is not possible and the individual is unable to perform weight shifts, return the individual to bed.**

(Scientific evidence–II/III; Grade of recommendation–B/C; Strength of panel opinion–Strong)

Postural instability can result from absent or weakened musculature, imbalanced muscle tone, orthopedic deformities, sensory deficits, or inadequately fitting support devices. It may be necessary to prescribe a specific wheelchair back support to minimize excessive 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 usage. 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." 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).

Interface pressure or ischial tuberosities are higher while sitting than lying down and must be relieved frequently to prevent tissue injury. Assistance with pressure relief is in the form of a reminder or motion assistance and is needed for individuals with impaired sensation or impaired mobility. When the pressure on the ulcer can be relieved by either assisted or self-mobility then limited sitting may be allowed (Bergstrom et al., 1994).

Postural management is a significant determinant of proper seating of individuals. 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).

Weight shifts are a critical element of pressure reduction (DeLateur et al., 1976). Generally weight shifting is recommended every 30 minutes for 30 seconds or every 60 minutes for 1 minute to allow reoxygenation of the cutaneous tissues (Nixon, 1985). Obesity may reduce one's ability to perform adequate pressure relief and safe transfers.

Wheelchair Support Surfaces

32. Use appropriate wheelchair cushions with all individuals with SCI.

- **Inspect and maintain all wheelchair cushions at regularly scheduled intervals.**

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

There are many commercially available, custom-fabricated pressure-reducing wheelchair seat cushions on the market, but not one of them has pressure ulcer prevention capacity for all individuals who have sustained a spinal cord injury (DeLateur et al., 1976; Krouskop et al., 1983). Pressure-reducing cushions include air-, fluid-, and foam-filled varieties, as well as combinations of these materials. 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).

After examining the variety of wheelchair cushions available, health-care professionals should find one that matches the individual's

needs. As with bed supports, the use of donut shaped ring cushions should be avoided. Wheelchair users need to follow a continuous inspection and maintenance schedule for their seat cushions.

FOAM CUSHIONS

Foam cushions are available in either a flat or contoured design. Sitting on contoured foam results in lower pressure and more acceptable pressure distribution than sitting on flat foam. The ability of foam to reduce pressure and to conform to body contours is defined by the *Indentation Load Deflection (ILD)* parameter. Sitting on soft foam (ILD=45lbs) resulted in lower pressure distribution and more acceptable pressure distribution than sitting on stiffer foam (ILD=55lbs) (Garber and Krouskop, 1997). A 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. A cushion that is custom-contoured to the user's tissue shape will provide lower pressures with a more stable base of support while sitting. This will result in less tissue deformity and stress (Sprigle et al., 1990). The advantages of foam cushions are that they are lightweight, lower in cost, easily modified, and available in a variety of forms. The disadvantages are easier fatigue, resulting in a relatively short life expectancy; heat retention; difficult cleaning; and rapid changes in support properties (Garber and Krouskop, 1997).

FLUID-FILLED CUSHIONS

Fluid-filled cushions tend to dampen horizontal motion while conforming to body contours and to reduce peak pressure on bony prominences. When an individual with SCI is active, it is not unusual for layers of tissue to be sliding on each other producing shear forces. Some researchers believe that fluid-filled (gel) cushions are more effective in reducing these shearing forces 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). 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).

AIR CUSHIONS

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 relief. 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. In addition to these qualities, Garber and Krouskop (1997) described the advantages of air-filled cushions: they are lightweight, easy to clean, effective for many people, and reduce shear and peak pressures. The disadvantages are a tendency to puncture, the need for maintenance, difficulty of repair, and enhancement of postural deformities.

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 visco-elasticity. Others use combinations of gel, air, 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.

Recommendations for Future Research

A comprehensive review of the research literature on the prevention and treatment of pressure ulcers after SCI was completed in 1998. To the extent possible, the recommendations in this clinical practice guideline were based on the findings reported in that review. Improvement in the effectiveness of prevention and treatment of pressure ulcers after SCI requires further research in the following areas:

Basic Science/ Biochemistry

- Undertake further study of pressure ulcer etiology following SCI, including:
  - Analysis of tissue above and below SCI lesion
  - Analysis of ethnic differences in skin
  - Assessment of "ideal" body weight parameters for individuals with SCI

Medical

- Develop and evaluate preoperative bowel preparation protocols for SCI individuals.
- Conduct longitudinal, controlled studies to determine the effect of positioning on bladder, bowel, and pulmonary function (wheelchair, bed, and standing in a standing wheelchair).
- Evaluate the effects of passive standing on circulation and tissue perfusion.
- Conduct studies on the management of hypergranulation tissue.
- Analyze post-operative bowel preparation and management.

Psychosocial Factors

- Develop, assess, and refine measures of knowledge and behavioral performance of health maintenance and self-care during and following rehabilitation.
- Assess the relationships between educational interventions and knowledge; between knowledge and behavior; and between behavior and health outcomes (e.g., incidence of pressure ulcers).
- Assess the efficacy of pressure ulcer preventive recommendations. Identify sources of variation, e.g., age, activity, and other health factors, and develop and test new prevention strategies based on these variations.
- Identify predictors of poor adherence and evaluate their impact on risk factors and pressure ulcer development and recurrence.
- Investigate the impact of both financial and social resources and the individual's living environment on pressure ulcer incidence.
- Conduct longitudinal studies to investigate the relationship between vocational activities (e.g., employment, volunteerism, and school attendance) and pressure ulcer incidence.
- Conduct SCI-specific behavioral medicine research investigating strategies to improve health maintenance behavior.

Pressure-Reducing Devices

- Conduct clinical evaluation of pressure-reducing devices for the wheelchair and bed.
- Conduct clinical trials on "specialty support surfaces" and their efficacy in the prevention and treatment of pressure ulcers.
- Conduct studies to determine whether there are postoperative uses for specialty support surfaces.

Dressings

- Conduct clinical evaluation of dressings, especially the newer ones on the market.
- Conduct comparative studies looking at various dressings to determine wound healing rates and costs.

Risk Assessment

- Develop an SCI-specific risk assessment tool suitable for clinical care and home settings.
- Determine the reliability and validity of an SCI-specific risk assessment tool.
- Determine the predictive merit of risk assessment variables on clinical outcomes.

Pressure Ulcer Education

- Develop and evaluate effective pressure ulcer-education programs.
- Determine the most effective means for teaching pressure ulcer prevention and treatment.

Nutrition

- Determine the protein requirements for effective prevention and treatment of pressure ulcers.
- Determine the appropriate level of vitamin C in SCI individuals for effective prevention of pressure ulcers.
- Determine if vitamin E is effective for pressure ulcer prevention and treatment in individuals with SCI.

Wound Healing

- Develop standardized methods for quantifying wound healing and compare the efficacy of these interventions.
- Determine the role cytokines, interleukins, and adhesion molecules play in pressure ulcer wound healing. Is it possible to use the individual's blood level as predictor of pressure ulcer healing rate?
- Develop and clinically evaluate non-invasive techniques for monitoring the status of pressure ulcers (or wound healing).

Cleansing

- Determine the most effective means to wound cleansing without damaging healthy tissue.

Interventions

- Compare the effectiveness of prevention strategies using various support surfaces and devices.
- Determine the effectiveness of platelet-derived growth factors and other growth factors as topical treatments and their effect on the rate of healing of pressure ulcers in SCI individuals.

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 (T-6) 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 interal 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 elestomer 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 cicatrical 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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