

Chronic Wound Prevention Guidelines

Abridged Version
2009



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The Wound Healing Society

In December 2006, the Wound Healing Society published “Treatment Guidelines for Pressure, Arterial, Venous and Diabetic Ulcers” (*Wound Repair & Regeneration* 14:645-710, 2006). The project was initiated, funded and supported by the Wound Healing Foundation, an organization that shares with the Wound Healing Society the goal of enhancing the quality of care for patients suffering from any type of wounds.

Four work groups gave selflessly of their time to complete the Treatment Guideline project in a timely manner. Their efforts were crowned by the above-mentioned publication, which has become a standard reference. At the completion of the project, two issues became evident: 1) the need to update the guidelines on a regular basis, a task entrusted by the Wound Healing Society to Dr. Martin Robson, and 2) the void in the realm of chronic wound prevention. Clinicians and health care providers are keenly aware of how much easier it is to prevent an ulcer rather than treat one. Furthermore, all have had the disappointment of seeing a wound that was carefully and painstakingly nursed to healing recur in short time.

Before the work groups disbanded, I gave them the challenge of providing a further set of guidelines on prevention of chronic wounds. All groups rose to the challenge, recognizing the void and need for such an exposition. The only change occurred in the Pressure Ulcer Group: leadership for the prevention guidelines was assumed by Joyce K Stechmiller, PhD ARNP, who was a member of the Pressure Ulcer Working group but had a special interest in prevention.

The work groups again have fully delivered on their assigned tasks. Drafts of the Prevention Guidelines were formulated using the same format and parameters as the Treatment Guidelines. Consensus was maintained throughout: in the broad and comprehensive research of existing literature; in the make-up of work groups to include all specialties, disciplines, professional degrees or societies so that clinical fields such as dermatology, endocrinology, vascular surgery, plastic surgery, podiatric medicine, geriatrics, nursing, dietetics/nutrition, rehabilitative services and prosthetics are all represented; in the application of the Delphi process, so that the majority of the group had to be in agreement with any pronouncement or recommendation; and finally, in the seeking of input from all interested parties, societies and industry at a publicly held forum during the Annual Meeting of the Wound Healing Society in April 2007 in Tampa. Characteristically, there is a lack of any “agenda,” as no industrial or other interest group funding was sought.

I again wish to thank the Wound Healing Foundation for their support. My sincerest thanks go also to all panel members who have worked so hard to produce these remarkable documents. Lastly, I think these guidelines also represent a call to action to all scientists, clinicians, health care providers, industry members involved in this field and government funding agencies: the need for more hard data is clear and cannot be acquired unless all interested parties lock arms and work toward this goal.

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March 2008

Risk Factors

- Deep vein thrombosis
- Incompetence of the direct calf and ankle perforating veins
- Elevation of ambulatory venous pressure (venous hypertension)
- Calf pump dysfunction
- Edema
- Cellulitis
- Lipodermatosclerosis

Lower Extremity Compression

1. Patients with signs of increased ambulatory venous pressure (venous hypertension) and/or postphlebotic syndrome should use compression stockings constantly and forever.

Principle: A degree of compression is necessary long term to minimize the pre-existing conditions conducive to venous ulcer formation. Attempts need to be made to aid patient compliance, including teaching the patient techniques to help them don the compression stockings.

2. Exercises to increase calf muscle pump function have been demonstrated to be helpful in long-term maintenance and venous ulcer prevention.

Principle: Calf muscle pump function has been shown to be improved with exercise.

3. Venous thromboembolism prophylaxis after deep vein thrombosis decreases recurrent deep vein thrombosis and the postphlebotic syndrome, and their complications such as venous ulceration.

Principle: Recurrent venous thromboembolic events can lead to the complication of postphlebotic or postthrombotic syndrome. This can be minimized with thrombo-embolism prophylactic therapy.

4. Subfascial endoscopic perforator surgery (SEPS) is the procedure of choice when it is desirable to address the underlying venous pathologic etiology of venous ulceration by preventing backflow from the deep to the superficial venous system. To achieve the greatest effectiveness when using this

procedure, care must be taken to divide all visible perforators. The procedure is not effective if the patient has severe deep venous disease with either deep reflux or obstruction.

Principle: Interruption of incompetent perforating vessels will aid in decreasing ambulatory venous pressure in the leg.

5. Less extensive surgery on the venous system—such as superficial venous ablation, endovenous laser ablation or valvuloplasty, especially when combined with compression therapy—can help decrease venous hypertension.

Principle: Procedures that are less extensive than deep ligation of multiple perforating veins can help decrease venous hypertension when combined with an adequate compression system. Although surgery can help prevent venous ulceration, ulcers that do occur following surgery may be more difficult to treat.

6. Cellulitis (inflammation and infection of the skin and subcutaneous tissue most commonly due to streptococci or staphylococci) in the edematous lower extremity should be treated with systemic antibiotics appropriate for those organisms.

Principle: Edema inactivates the normal bactericidal properties of skin. This renders the skin and subcutaneous tissue susceptible to infection by streptococci and staphylococci.

7. Fibrinolytic enhancement with an anabolic steroid, such as stanozolol, in conjunction with compression therapy, may be useful in treating lipodermatosclerosis of the lower extremity. However, one must be aware of side effects.

Principle: A fibrinolytic agent capable of decreasing extravascular fibrin should be able to decrease induration and inflammation in cases of lipodermatosclerosis.

8. Oral treatment with micronized purified flavonoid fraction (MPFF) may be a useful adjunct to conventional compression therapy in managing chronic venous insufficiency.

Principle: Agents that inhibit synthesis of prostaglandins and free oxygen radicals, decrease microvascular leakage, and inhibit leukocyte trapping and activation should theoretically improve chronic venous insufficiency and prevent venous ulceration.

Risk Factors

- Immobility
- Friction and shear
- Moisture
- Incontinence
- Poor nutrition
- Perfusion
- Age
- Skin condition
- Altered level of consciousness

1. Pressure Ulcer Risk Screening

- 1.1 All patients admitted to the healthcare setting shall undergo a pressure ulcer risk screening (PURS) by or under the supervision of a registered nurse or health care professional with training and expertise in wound care. The screening should be done within the time frame specified by organization policy or as required by regulation within 12 hours of admission. In nursing home settings, the window for screening is the MDS, which should be completed in 7 days.

Principle: The best-practice process of pressure ulcer prevention requires a series of steps with feedback loops. These steps include pressure ulcer risk screening (PURS), pressure ulcer risk assessment (PURA), formulation of a pressure ulcer prevention care plan (PUPCP), implementation of the plan, monitoring and reassessment of the care plan, reevaluation of the healthcare setting, and then either reformulation of the care plan or termination of therapy.

PURS Recommendations:

The **Braden Scale** (1994) has been the most extensively studied. The Braden Scale consists of six parameters (sensory perception, mobility, activity, moisture, nutrition, and friction and shear) with potential scores from 6 to 23. Lower total scores indicate greater risk of developing pressure ulcers. If other major risk factors are present (e.g., age, fever, poor dietary intake of protein, diastolic pressure < 60, and/or hemodynamic instability), advance to next level of risk.

Mild risk = 15-18

Moderate risk = 13-14

High risk = 10-12

Very high risk = 9 or below

The **Norton Scale** (1996) is a pressure ulcer risk assessment scale that consists of five parameters (general physical condition, mental condition, activity, mobility and incontinence) each rated on a scale of 1 to 4, with lower numbers associated with greater impairment and potential total scores ranging from 5 to 20.

Mild risk = 14

Moderate risk = 13

High risk = 12

The reliability and validity of the tool has not been established.

Braden Q Scores (1996): This scale was adapted from the Braden Scale for use in the pediatric population.

Mild risk = 25

Moderate risk = 21

High risk = 16

Resident Assessment Protocol (RAP) for nursing homes:

This is the only assessment tool recognized by CMS for pressure ulcer risk assessment in nursing homes. The Braden Scale does not perform well in settings outside the hospital. Both the Norton Scale and the Braden Scale have good sensitivity (73-92% and 83-100%, respectively) and specificity (61-94% and 64-77%, respectively), but have poor positive predictive value in nursing home residents (around 37% at a pressure ulcer incidence of 20%). In populations with a lower incidence of PUs, such as nursing home residents, the same sensitivity and specificity would produce a positive predictive value of 2%. The Norton and Braden scales show a 0.73 Kappa Statistic agreement among at-risk patients, with the Norton Scale tending to classify patients at-risk when the Braden Scale classified them as not-at-risk. The net effect of poor positive predictive value means that many patients who will not develop pressure ulcers will receive expensive and unnecessary treatment.

- 1.2 The result of the PURS shall be documented, and appropriate assessment and intervention initiated within 24 hours of admission. Nursing homes: 7 days (see Guideline 1.1).

Principle: A skin risk screening assessment tool may be most helpful when used with a combination of strategies, including additional skin assessment policies and procedures, skin care teams, and educational programs.

- 1.3 A procedure for pressure ulcer rescreening should be implemented within 48 hours or when there is a significant change in the individual's condition, such as transfer to ICU, system or organ failure, septicemia, chronic ICU status with prolonged ventilator support, fever, hemodynamic instability, urinary tract infection in nursing home residents, etc.

Principle: Same as 1.1

- 1.4 A schedule for reassessing risk should be based on the acuity of the individual and awareness of when pressure ulcers occur in a particular clinical setting.

Principle: Note that there is limited evidence that risk assessment leads to a reduction in frequency of pressure ulcers. There are limits of risk factor identification; a number of risk factors are not modifiable, such as fecal incontinence, mobility, level of consciousness or even nutrition.

2. Pressure Ulcer Risk Assessment (PURA)

Preamble: Pressure ulcer risk assessment (PURA) plays a significant role in the prevention of pressure ulcers. Patients who are at-risk should be identified by PURA shortly after admission to a health care setting

- 2.1 The PURA shall be performed by or under the supervision of a registered nurse or health care professional with training and expertise in wound care within the time frame specified by organization policy or as required by regulation.

Principle: Each patient shall undergo a thorough PURA by or under the supervision of a registered nurse or health care professional with training and expertise in wound care to determine and assess the risk factors and care needs, and the type of preventive care to be provided (NPUAP, 2002).

2.2 The pressure ulcer risk assessment shall include identification of subjective, objective and psychosocial factors to determine and assess the risk factor, care needs and type of preventative care to be provided. The following key points are recommended for documentation and shall be addressed when appropriate:

For skin status and bony prominences:

- Description of skin changes, as well as any actions taken, recent trauma, friction, shear or immobility.
- Use of special garments, shoes, heel and elbow protectors, orthotic, or orthopedic devices.
- History of pressure ulcers and presence of current ulcer.
- Previous treatments or surgical interventions that increase risk for pressure ulcers.
- Factors that impede healing status, such as comorbid conditions or medications.
- Medical history (history of stroke).
- Body weight, appetite and nutritional status.
- Alcohol and substance abuse; use of tobacco.
- Gastrointestinal and elimination symptoms.
- Recent changes in functional capacities.

Other elements:

- Advanced age (> 75 years of age having an odds ratio of > 12.6).
- African-American race.
- Female gender.
- Disorders of skin integrity and specific vulnerable pressure points.
- Assessment for immobility.
- Assessment for incontinence.
- Admitting diagnosis that may affect skin integrity (gangrene, burns, osteomyelitis, edema and infections) and wound healing (including immune status and diabetes).

PRESSURE ULCER

- Concurrent medical and surgical problems that may affect skin integrity (burns, edema, organ system failure, septicemia, ICU length of stay, ventilator days, advanced cancer, terminal illness and diabetes) and wound healing (including infections, e.g., urinary tract infections, bacterial infections, pneumonia, anemia and immune status).
- Assessment of nutrition status data: weight, BMI, and anthropometric and laboratory evaluations. Other helpful elements: lab values such as serum transferrin, prealbumin, and resting energy expenditure. Nutritional requirements and nutrition support options should be determined as an integral part of the initial risk assessment for each individual.
- Laboratory data as available, which may include but are not limited to: complete blood count with red cell indices, total lymphocyte count, serum electrolytes, blood urea nitrogen, creatinine, serum glucose, serum albumin, pre-albumin, C-Reactive Protein, transferrin, serum cholesterol, serum triglycerides and liver function studies.

Principle: Each patient shall undergo a thorough PURA to assess the risk factors, care needs and type of preventative care to be provided. Additional individual considerations that impact pressure ulcer prevention measures:

- *Age: Seniors (> 65 years old) and children (< 5 years old) are at high risk. In children and neonates, the head (occiput) is the most common site of pressure ulcer occurrence.*
- *Gender and race: Female gender, African-American race, and advanced age are identified as risk factors for pressure ulcer diagnosis in acute care hospitals.*
- *Spinal cord injury (SCI): Patients with SCI are at high risk of developing PUs with high rates of recurrence. Increased risk factors: history of ulcers, younger age at onset and duration of SCI, greater disability and difficulty with practicing good skin care, and extent of paralysis. PUs are least likely among individuals with SCI who maintain a normal weight, return to work and family roles, do not have a history of tobacco use, suicidal behaviors, incarcerations, or alcohol or drug abuse.*

- *Potential sites for pressure ulcers: The most common sites are the sacrum or coccyx and heels; others are the ankle, buttocks and occipital areas.*
- *Critically ill patients: The risk is higher among burn patients with increased moisture as determined by the Braden Scale (wound drainage and incontinence were also identified as contributing factors); ICU patients exposed to moisture, and patients with sensory perception or perfusion problems.*
- *Other factors in critically ill patients include norepinephrine administration, APACHE II score greater than 13, length of stay, anemia and fecal incontinence.*
- *Immobilized low weight patients: Patients with a lower BMI developed PUs.*
- *Postoperative patients with a longer duration of surgery and length of time in a hypotensive state have an increased risk.*
- *Acute care hospitalized patients diagnosed with PUs have three categories of risk factors including skin integrity (presence of gangrene, nutritional deficiencies, diabetes and anemia), system failure (paralysis, senility, respiratory failure, acute renal failure, CVA and congestive heart failure-nonhypertensive) and infections (septicemia, osteomyelitis, pneumonia, bacterial infections and urinary tract infections).*
- *Inadequate nutritional intake: Adequacy of nutritional intake (serum albumin, total lymphocyte count, nitrogen balance, hydration status and micronutrients) is correlated with skin integrity and should be frequently addressed. Note that although poor nutrition is part of total patient care and should be addressed in each patient, no nutritional intervention has shown effectiveness in prevention of pressure ulcers in published studies.*

- 2.3 The subjective and objective assessments of pressure ulcer risk shall be summarized and documented in the patient's medical record.
- a) The patient's skin status and bony prominence assessment shall be summarized based on the findings of the subjective and objective pressure ulcer risk assessment and should include prevention measures (turning-positioning program, bed surface pressure relief therapy, minimizing friction and shear, managing incontinence) for optimal skin integrity requirements.
 - b) The patient's nutritional requirements shall be summarized based on the findings of the subjective and objective nutrition assessments, and should include protein, calorie, fluid, electrolyte and micronutrient requirements.
 - c) The patient and caregiver educational requirements shall be summarized based on the findings of the subjective and objective nutrition assessments, and should include causes and risk factors for pressure ulcer development, and ways to minimize risk.

Principle: The pressure ulcer risk assessment shall be documented and be available to all health care providers.

3. Pressure Ulcer Prevention Care Plan (PUPCP)

Preamble: Development of a pressure ulcer prevention care plan (PUPCP) plays a significant role in the prevention of PUs. Clinical settings and patients who are at risk should have a prevention plan to target prevention efforts to minimize risk.

- 3.1 The policy and procedure for the prevention plan for pressure ulcers shall be formalized and documented. The pressure ulcer prevention plan shall include identification of high-risk settings and groups to target prevention efforts to minimize risk.

Principle: Identify etiologic factors contributing to pressure ulcer occurrence; conduct regular risk screening and assessments using valid and reliable tools; develop, implement and evaluate evidence-based programs for prevention of pressure ulcers (including identification of risk factors, skin and bony prominence assessment and care, demonstration of proper body positioning, selection and use of support surfaces and skin protection devices, and treatments and use of appropriate nutritional interventions).

- 3.2 An interdisciplinary team of health care professionals shall review and evaluate the quality of preventative pressure ulcer care provided at the health care setting at least quarterly. The PUPCP should be developed with an interdisciplinary approach involving the physician, registered nurse, registered dietitian, physical therapist and other healthcare personnel as appropriate, and with the involvement of the patient and/or family whenever possible. It should be based on information from the risk tool and the PURA. The PUPCP information should be completed within 48 hours after the completion of the PURA and incorporated into the overall plan of care in acute care settings. In long-term care settings, an LPN may be assigned to a team, and completion of PUPCP information and overall plan of care will be consistent with the policies and guidelines of long-term care settings.

Principle: An interdisciplinary team of health care providers is the optimal approach to prevention of PUs. See also 3.1.

Although the development of a pressure ulcer may reflect a breakdown in quality of care, it cannot reflect quality of care if the pressure ulcer develops despite consistent application of known interventions. The link to quality of care assumes that consistent application of effective interventions will prevent all pressure ulcers. This has not been demonstrated in the literature. This is a profoundly important issue from a regulatory and medico-legal standpoint.

4. Selection of Prevention Interventions

Preamble: There are many pressure ulcer prevention interventions to consider. Selection should be appropriate to the patient's individualized needs. Guidelines assist the health care provider in making decisions regarding the best cost-effective practice.

- 4.1 The interventions selected to prevent pressure ulcers shall be appropriate to the patient's risk factors, skin status and assessment of bony prominences, nutritional status, mobility, risk of incontinence, pressure, friction and shear, medical condition, and goals expressed by the patient and/or family. If the resident is not competent, the designated individual with durable power of attorney/healthcare shall be an active participant.

Principle: Prevention intervention selections should be tailored to the individual's needs and should be evidence based. Consideration of the least invasive, most cost-effective therapy is preferred.

5. Consideration of Positioning and Support Surfaces

Preamble: Pressure ulcers are areas of localized tissue destruction caused by unrelieved pressure, shear and friction to the skin.

- 5.1 Provide frequent position changes using pillows and wedges to reduce pressure on bony prominences.

Principle: PUs are thought to be caused by unrelieved pressure and compression of soft tissues against bony prominences. Relieving pressure over bony prominences can be achieved. Pillows under calves decrease heel interface pressures. No specific support surface or heel product has been proven superior in decreasing pressure at the heel.

- 5.2 Avoid foam rings or donuts for pressure reduction; they concentrate the pressure to surrounding tissue. Foam wheelchair cushions are recommended.

Principle: Seat cushions reduce pressure in the sitting position. Ring cushions or donut devices have been shown to increase edema and venous congestion.

- 5.3 Pressure prevention interventions shall be provided through use of pressure-reducing or -relieving devices.

Principle: Pressure-reducing or -relieving devices work by redistributing pressure over the bony prominences. Static support surfaces are mattresses or mattress overlays that are applied to a mattress and are filled with air, water, gel, foam or a combination of these. Alternating support surfaces or dynamic support surface mattresses have been associated with lower incidence of PUs compared with standard hospital mattresses. High-specification foam bed surface has been effective in decreasing the incidence of PUs in high-risk patients. A variety of pressure-reducing mattresses or devices lower the incidence of PUs when compared to a standard hospital mattress. At-risk patients should not be placed on an ordinary, guideline hospital foam mattress. Turning every 4 hours in combination with the use of a pressure-reducing mattress was shown to decrease the occurrence of PUs compared to turning every 6 hours on a pressure-reducing mattress or turning every 2-4 hours on a non-pressure-reducing mattress. Bed surfaces that provide pressure reduction include non-powered surface mattress replacements, powered single-zone surfaces, low-air-loss multi-zone customized beds and mattresses; pressure relieving surfaces include air-fluidized beds and lateral rotation dynamic air therapy beds. There is insufficient evidence to support the choice of one device over another for prevention of PUs. Efficacy of these devices is inconclusive; most support surface studies rely only on interface pressure measurements as an outcome.

- 5.4 Avoid ordinary sheepskin for pressure reduction; it provides comfort but does not relieve pressure to tissue. Dense specialized sheepskin is recommended.

Principle: There is limited evidence that dense specialized sheepskin will reduce the incidence of pressure ulcers.

- 5.5 Limit the amount of time the head of the bed is elevated. Lower head of bed 1 hour after meals to prevent pressure over bony prominences; assess the sacral area more frequently if this is not possible.

Principle: Elevation of the head of the bed may result in shear and friction forces between the skin and the bed surface; this may predispose to the development of PUs.

- 5.6 Pressure prevention interventions shall be provided through regular and frequent turning and repositioning for bed- and chair-bound patients. Reposition at least every hour to reduce pressure for chair-bound patients, with attention to the patient's anatomy, postural alignment, distribution of weight and support of feet.

Principle: See 5.1. Reductions in pressure incidence have been achieved by repositioning every 4 hours for bed-bound patients. For the chair-bound patient, tissue pressure between the sitting surface and bony prominence should be relieved. Reposition the sitting individual to relieve pressure at least every hour. For those who can reposition themselves, pressure relief should be encouraged every 15 minutes such as chair push-ups. Constant low-pressure devices and seat cushions have not been proven efficacious in reducing the incidence of PUs for chair-bound individuals. Gel or air-pressure reducing chair cushions are more effective than foam in preventing ischial pressure ulcers. The ROHO cushion was demonstrated to be more effective in relieving pressure at the seating surface when compared to the Jay and PinDot® cushion.

- 5.7 Pressure reduction using specialized foam overlays should be used as a preventative measure in the operating room for patients assessed to be at high risk for pressure ulcer development.

Principle: Convoluted foam, cubed foam and specialized thicker, denser sheepskin overlays are the only overlay surfaces shown to reduce the incidence of pressure ulcers in these settings.

6. Friction and Shear Prevention Interventions

Preamble: Shear and friction to the skin is a contributing risk factor increasing a person's susceptibility to a complex etiology that causes pressure ulcers.

- 6.1 Avoid vigorous massage over bony prominences.

Principle: Friction damage occurs when repetitive friction results in parallel rubbing or sanding of the epidermis.

- 6.2 Use overhead trapeze bars, when possible, to assist with patient mobility.

Principle: Shear force is a key factor in the development of pressure ulcers.

- 6.3 Clean and dry the skin after each incontinence occurrence.
Principle: Friction and shear are enhanced in the presence of moisture.
- 6.4 The use of cornstarch to decrease skin resistance, or protective application of hydrocolloids, hexachlorophene lotion, hyperoxygenated fatty acid preparations or sheet hydrogel dressings may protect vulnerable skin surfaces.
Principle: Friction combined with pressure and moisture results in damage more readily. Friction injuries occur more frequently on elbows or where skin is fragile or macerated.
- 6.5 Use lift sheets or devices to turn or transfer patients to avoid dragging or pulling that can result in friction injuries.
Principle: Same as 6.4.
- 6.6 Maintain head of bed at, or below, 30 degrees or at the lowest degree of elevation consistent with the patient's medical condition to prevent sliding and shear-related injury.
Principle: Elevation of the head of the bed may result in shear and friction forces between the skin and the bed surface; this may predispose to the development of pressure ulcers.

7. Moisture or Incontinence Management

Preamble: Moisture or incontinence is a contributing risk factor increasing the person's susceptibility to a complex etiology that causes pressure ulcers.

- 7.1 Use of gentle skin cleansers designed for intact skin should be used to remove excrement. Products that contain surfactant facilitate the removal of urine and stool, and require less abrasive force.
Principle: Moisture removes oils on the skin, making it friable and contributing to maceration of tissues, and softening of the skin's connective tissue and erosion of the epidermis; increasing the likelihood of pressure sore development. Use of strong soaps (bar soap) emulsifies the lipids in the skin and increases the skin pH, reducing the protective normal skin barriers. AHCPR guidelines recognize that skin exposure to urine and feces increases the risk of PUs.

8. Nutrition Management

Preamble: Managing nutrition is essential to the prevention of PUs. Protein, carbohydrates, vitamins, minerals and trace elements are required for wound healing. There is limited evidence documenting the effectiveness of nutritional management in the prevention of PUs.

- 8.1 The nutrition management plan to prevent pressure ulcers should provide adequate daily calories, protein, carbohydrates, fat, vitamins and minerals to meet individual energy needs. Provide nutrition (parenteral or enteral) appropriate to individual needs, goals of care and patient preferences.

Principle: Nutrition is essential in maintaining skin integrity. If nutritional risk or malnutrition occurs, the patient is at risk for the development of PUs. The impact of nutrition in the prevention of PUs remains controversial.

- 8.2 Provide ongoing weekly nutrition assessment and reassessment to ensure adequate dietary intake in acutely ill patients; nutrition assessment in nursing home residents shall be compatible with guidelines for nursing homes.

Principle: See 8.1. Long-term care residents are at particularly high risk for developing PUs. Risk assessment tools such as the Braden Scale include nutrition assessment as an integral part of overall assessment of risk for pressure ulcer development.

9. Health Care Provider, Patient and/or Caregiver Education

Preamble: Despite many of the advances in wound care, the challenge of preventing PUs remains due to the complexity of predisposing factors. This is compounded by the need to educate health care providers, patients, family, and/or caregivers about pressure ulcer prevention.

- 9.1 The health care provider, patient and/or caregiver should understand the importance of the following in preventing PUs in at-risk adults: regularly inspecting skin and bony prominences; following recommended skin-care regimens; avoiding vigorous massage of reddened areas and bony prominences; preventing friction and shearing forces, including frequent turning, repositioning and the use of

pressure-reducing devices if patient is confined to bed and/or chair; avoiding donut-type devices; maintaining adequate hydration and nutrition; monitoring weight loss, poor appetite or gastrointestinal changes that interfere with eating; and promptly reporting changes in medical status and nutritional problems.

Principle: Health care providers, patients, families and caregivers need to be educated about pressure ulcer risk prevention.

10. Interdisciplinary Approach

Preamble: Preventing PUs is less costly than treating pressure ulcers. The economic significance of PUs necessitates the importance of an interdisciplinary approach for their prevention.

- 10.1 An interdisciplinary team of appropriate health care professionals, including a physician, advanced practice nurse and/or registered nurse/LPN for nursing homes, registered dietitian, physical therapist, occupational therapist, social worker and other health care professionals as appropriate shall be identified to establish, develop, and implement policies and procedures for the prevention of pressure ulcers.

Principle: Efforts to implement pressure ulcer prevention protocols through development of policies and procedures demonstrate a reduction in the prevalence of PUs. Note the literature has not demonstrated an effect in reducing the incidence of pressure ulcers that was sustained in time. No change in pressure ulcer prevalence has been observed since implementation of the Omnibus Budget Reconciliation Act of 1987 in a nationally derived sample of long-term nursing home residents. The relative odds of having a pressure ulcer increased by 6% from 1992-1994 to 1997-1998 for all pressure ulcer stages and increased 21% for stages 2 and greater. The risk-adjusted incidence rate of developing a pressure ulcer in Department of Veterans Affairs nursing facilities in 1997 was similar to the rate in 1990. However, the severity of new pressure ulcers was higher in 1997. Arguably, this represents either a complete failure to implement recommended interventions or a complete failure of the nature of the quality improvement interventions.

Preamble: Patients with diabetes develop wounds as a result of neuropathy, peripheral vascular disease with ischemia or both. In 2002, there were 19.3 million people with diabetes mellitus, for a crude incidence of 9.3%. The diagnosis of diabetes had been established in 6.5%, with 2.8% undiagnosed. There are now an estimated 20.8 million people in the United States with diabetes. As many as 25% of these patients are at risk for ulceration during their lifetimes. Preventing wounds may reduce the likelihood of amputation. Establishing the proper diagnosis is imperative, as is evaluation of the patient for the complications of diabetes. Prevention of wounds in these patients involves addressing these complications prior to ulceration.

1. Identify Complications of Diabetes

- 1.1 In patients with diabetes, clinically significant arterial disease should be ruled out by establishing that pedal pulses are clearly palpable or that the ankle:brachial index (ABI) is > 0.9 . An ABI > 1.3 suggests noncompressible arteries. In elderly patients or patients with an ABI > 1.2 , a normal Doppler derived wave form, a toe:brachial index of > 0.7 or a transcutaneous oxygen pressure of > 40 mm Hg may help to suggest an adequate arterial flow. Color duplex ultrasound scanning provides anatomic and physiologic data confirming atherosclerotic occlusive disease.

Principle: Diabetic ulcers can result from minor trauma in patients with arterial insufficiency. Although clinical history and physical examination can be suggestive of ischemia of the lower extremity in a patient with diabetes, a definitive diagnosis must be established.

- 1.2 The presence of significant neuropathy that can render a patient at risk of foot ulceration can be determined by testing with a 10 gram (5.07) Semmes-Weinstein monofilament. Monofilament testing should be combined with a clinical examination of the lower extremity that focuses on the possible existence of foot deformity and a symmetric sensory level, below which there is reduced sensation to pain, touch and vibration in both limbs.

Principle: The most important causative factor of diabetic foot ulcers is peripheral neuropathy. Neuropathy leads to foot deformity with abnormal pressure on the foot, especially the plantar surface. Lack of protective sensation allows ulceration in areas of high pressure. Autonomic neuropathy may increase the likelihood of skin breakdown.

- 1.3 In patients with diabetes, laboratory values such as hemoglobin A1c should be monitored.

Principle: High glucose concentrations in the blood lead to increased glycation of the hemoglobin molecules to form hemoglobin A1C, which persists in circulation for up to 6 weeks. Therefore, measurement of plasma hemoglobin A1C is the accepted standard for monitoring long-term glucose control. Elevated hemoglobin A1C levels have been correlated with a variety of comorbidities of diabetes, such as cardiovascular and/or coronary heart disease, retinopathy, neuropathy, and nephropathy/renal failure. Elevated hemoglobin A1C has been shown to be a predictive factor in the development of diabetic foot ulcers, but further research is still required.

2. Perform Foot Exam

- 2.1 Patients with diabetes should have an annual foot exam.

Principle: Published guidelines uniformly recommend that all diabetic patients have an annual foot examination that includes assessment for anatomic deformities, skin breaks, nail disorders, loss of protective sensation, diminished arterial supply and improper footwear. People with higher risk for foot ulceration should have more frequent foot exams.

- 2.2 Patients with diabetes should be examined for callus formation.

Principle: Callus formation, particularly with hemorrhage, is a sign of impending skin breakdown and ulceration. Removal of the callus results in lowered plantar pressures.

- 2.3 Patients with diabetes should be examined for fungal toenails.

Principle: Onychomycosis, a fungal infection of the nails, affects approximately one-third of patients with diabetes and is the source of extensive morbidity. Fungal toenails often harbor bacteria that can cause infection after injury to the skin, often initiated by the sharp and brittle nails themselves. Treatment options include oral antifungal agents, topical therapy and mechanical intervention.

3. Surgery to Prevent Ulceration

- 3.1 Increased pressure on areas of the diabetic foot results in callus formation, which can then lead to ulceration. Removal of callus will reduce the likelihood of ulceration.

Principle: Paring callus will reduce pressure in areas at increased risk for ulceration; therefore, all calluses should be removed with few exceptions.

- 3.2 Achilles tendon lengthening decreases forefoot plantar pressure. This procedure may be recommended only for patients with history of repeated foot ulceration in whom all other non-interventional care has failed.

Principle: Decreasing elevated forefoot plantar pressure is associated with a decrease in risk of ulceration.

4. Protect the Diabetic Foot

- 4.1 Protective footwear should be prescribed in any patient at risk for ulceration (significant arterial insufficiency, significant neuropathy or previous amputation).

Principle: Diabetic ulceration may result from an increase in pressure in the diabetic foot because of foot deformity and neuropathy. Offloading reduces the area of high pressure. The incidence of ulceration in diabetic patients at risk can be reduced by using protective footwear. Protective footwear should be prescribed in any patient at risk for amputation (such as significant arterial insufficiency, significant neuropathy, previous amputation, previous ulcer formation, pre-ulcerative callus, foot deformity or evidence of callus formation).

- 4.2 Patients with healed diabetic ulcers should use protective footwear to prevent recurrence.

Principle: Diabetic ulcers of the lower extremity are a chronic problem. Recurrence rates are 8 to 59%. Therefore, long-term maintenance must be addressed even for healed ulcers. Most treatments do not eliminate the underlying increased pressure on the foot, so offloading is necessary long term.

5. Good Foot Care

- 5.1 Good foot care and daily inspection of the feet will reduce the incidence of diabetic ulceration. It may be necessary for a family member or caregiver to help with these tasks.

Principle: Good foot care, including proper bathing and nail trimming, and the use of proper footwear, will reduce ulceration in diabetic feet.

6. Education

- 6.1 Education of patients with diabetes, aimed at preventing foot wounds, may reduce the incidence of ulceration and amputation, especially in high-risk patients.

Principle: Educating patients, using a diabetes educator if available, may help them practice good foot care behavior.

- 6.2 Education of clinicians about patients with elevated risk for lower extremity amputation may reduce the risk of ulceration.

Principle: Clinicians who are aware of patients with increased risk of foot ulceration are more likely to prescribe preventive foot care behaviors.

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Preamble: The current guideline will focus on preventing lower extremity arterial insufficiency ulcers in adults with lower extremity peripheral arterial occlusive disease (PAOD), as well as preventing recurrence in those who have already had an arterial insufficiency ulcer. Clearly, the ideal way to prevent arterial ulcers is to prevent PAOD. These guidelines will focus on measures to prevent lower extremity ulcers in patients who already have PAOD.

PAOD has a high prevalence, especially among people older than 60 years (about 30-40%) and is associated with high risk of fatal and nonfatal cardiovascular events. Patients with PAOD may develop gangrene and spontaneous ulceration (critical limb ischemia) or impaired or failed healing of injury. Although patients with PAOD are at risk for amputation, their risk of death from cardiac causes is much higher.

PAOD is a clear risk factor for cardiovascular mortality. All patients with PAOD should be evaluated for medical treatment with antiplatelet drugs, beta blockers, statin therapy, and ACE inhibitors to reduce the risk of cardiovascular morbidity and mortality. Despite the high risk of death from cardiac causes in patients with PAOD, several studies have demonstrated that healthcare providers often underestimate the risks and provide less comprehensive cardiovascular treatment in patients with a presenting diagnosis of PAOD. Awareness of the implications of PAOD is critical to appropriate treatment of patients with PAOD, outside of the prevention of ulceration.

Awareness of risk factors for PAOD will clearly increase recognition of it and, thus, initiation of appropriate treatment both to reduce mortality and to reduce the incidence of arterial ulcers. Risk factors include smoking, diabetes, age, gender, hyperlipidemia, renal insufficiency or failure, hyperviscosity or hypercoagulable states, non-ambulatory status, CAD, cerebrovascular disease, race, level of awareness, hypertension, and associated vasculitis. Individuals at high risk for PAOD or other cardiovascular disease should be screened and assessed so that efforts can be focused with interdisciplinary plans of care for preventing peripheral arterial ulcers.

1. Peripheral Arterial Ulcer Awareness

Preamble: Peripheral arterial occlusive disease (PAOD) is a common manifestation of atherosclerosis. Intermittent claudication is often thought of as the “classic” symptom of PAOD, but only one-third of patients with PAOD present with this symptom. Only a minority of patients with intermittent claudication progress to arterial ulceration. Most patients with critical limb ischemia had asymptomatic PAOD prior to the development of rest pain or tissue loss.

Ischemic arterial ulcers appear in advanced stages of PAOD with associated high rates of cardiovascular morbidity, mortality, limb amputation, decreased quality of life and increased cost of health care. When compared with other conditions in the spectrum of cardiovascular diseases (i.e., stroke, CAD), PAOD is under-diagnosed and under-treated, even though its presence is associated with similar morbidity, mortality and risks of future cardiovascular events. Awareness and recognition of PAOD by primary care providers has a significant impact on prevention of ischemic arterial ulcers. Implications include the need for regular lower extremity protection and assessment, education about self-care, and referral to specialists when required.

- 1.1 Clinicians should have a high index of suspicion for the presence of PAOD in patients with multiple risk factors, even if they do not have symptomatic disease (e.g. claudication). Patients with PAOD may not ambulate enough to have symptoms of claudication, and symptoms may not develop in patients with peripheral neuropathy, despite advanced disease. Furthermore, symptoms of ischemia may not be recognized among other comorbidities such as arthritis, or pulmonary or cardiac disease. A high index of suspicion is needed to detect asymptomatic disease, using indicators such as trophic changes (e.g., hair loss; thin, shiny skin on the calves or feet; and thickened nails).
- 1.2 Awareness and recognition of the risks and impact of treatment of PAOD on future cardiovascular events in the PAOD population should be emphasized in education and practice.

- 1.3 A family history should be sought and the routine review of systems should include: decreased walking distance, exertional leg pain, numbness or leg fatigue while walking, nocturnal foot pain, and foot numbness at rest. Patients may not mention symptoms of PAOD to health care providers unless prompted. A patient's attitudes and requests regarding treatment impact health care providers' behavior toward preventive interventions.
- 1.4 A large number of educational efforts, including national advertising campaigns, have been aimed at better public awareness of CAD and cerebrovascular disease. Similar efforts aimed at awareness of PAOD have been undertaken. These campaigns should be broadened, expanded and continued. There is a large population with PAOD and associated decreased ABI and skin oxygen levels that is too sedentary, deconditioned, and/or with limited physical activity to present with classic symptoms of PAOD, such as claudication. Patients may also have neuropathy with inability to feel and report rest pain. Such patients may have a minor non-healing injury and not be aware of the implications of this problem or the need to seek medical attention.

2. Peripheral Arterial Ulcer Risk Screening and Prevention

Preamble: Data on ischemic arterial ulcers as an independent event are limited since PAOD is strongly associated with other serious and potentially life-threatening conditions. Given this information, it is common to underdiagnose patients with limb ulcers secondary to PAOD. Therefore, efforts should be made to prevent ulcers in two distinct groups: those with PAOD who have yet to be diagnosed and those who are already identified as having PAOD.

An interdisciplinary team of appropriate health care professionals, including a physician, advanced practice nurse and/or registered nurse or LPN, ostomy/wound care nurse, registered dietitian, physical therapist, occupational therapist, social worker, podiatrist and other health care professionals, as appropriate, should be identified to establish, develop, and implement policies and procedures for the prevention of

peripheral arterial ulcers. A multidisciplinary team is more likely to implement the wide range of therapies that have been found to be beneficial.

Prevention in those identified with PAOD

- 2.1 Patients with an ABI < 0.4 , an ankle pressure less than 50 mm Hg or a toe pressure less than 30 mm Hg are at high risk of developing arterial ulceration. Early referral to a vascular specialist is paramount.

Yearly ABIs and a low threshold for referral back to a vascular specialist seems prudent.

- a) In patients with known PAOD (symptomatic or asymptomatic), progression should be assessed frequently by non-invasive hemodynamic assessments. The Ankle Brachial Index (ABI; ankle systolic pressure divided by brachial systolic pressure) is the most frequently used measurement. It is easy to measure, although its sensitivity is not high. The Toe Brachial Index (TBI) may be more sensitive, especially when tibial artery calcification (non compressibility) is suspected, as is common in diabetics. Absence of toes precludes this measurement; however, a decrease of 0.1 units in TBI or 0.15 units in ABI is considered a significant deterioration. Skin perfusion pressure and transcutaneous oximetry may also be of value in following progression of disease. Providers should have a high index of suspicion for progression of disease, and a low threshold for pursuing further testing or referral (see Guideline 4.1 for indications for vascular intervention).
- b) Twenty percent of patients who undergo surgical revascularization of one extremity for arterial ulceration will progress to arterial ulceration of the other extremity. These patients should be closely followed by a vascular specialist.

Prevention in those not yet diagnosed with PAOD

- 2.2 As with Guideline 2.1, early diagnosis is paramount. Health care providers should be educated in identifying risk factors and routine screening for PAOD. Patients should be referred early to a vascular specialist.

a) ABI should be measured routinely in patients at high risk for vascular disease so as to identify patients with PAOD, ideally as part of the routine annual primary care physical examination. This will enable early institution of prevention programs, along with timely referral for formal vascular evaluation. High risk groups include:

- Advanced age (>75 years have a 30% prevalence of PAOD)
- Known coronary artery disease (angina, prior MI or revascularization)
- Known cerebrovascular disease (stroke, TIA)
- Smokers
- Diabetes mellitus
- Hyperlipidemia
- Dialysis dependent renal failure
- Hyperhomocysteinemia
- Elevated CRP

b) Signs of PAOD usually develop at the leg, ankle or heel level and may progress to ulceration if not recognized. These include:

- Loss of hair
- Shiny or dry skin
- Mummified or dry and black toe
- Devitalized soft tissue with a dry or wet crust
- Thickened toe nails
- Deep purple color (in dependent position)
- Cool skin

Limbs should be covered with a towel or other cover in the clinic to avoid confusion, since a cool limb often results from cold exposure even in patients without PAOD.

3. Preventive Interventions: Medical

Preamble: Prevention of ischemic arterial ulcers should focus on aggressive management of PAOD and prevention of trauma to the legs and feet of patients at risk.

- 3.1 Cardiovascular diseases should be identified and managed with a multidisciplinary, evidence-based approach. Standard treatment guidelines for medical therapy (including beta blockers, statins, ACE inhibitors and calcium channel blockers) will improve outcomes for not only coronary artery disease, but also ischemic arterial ulcers.
- 3.2 Cessation of smoking is by far the most important factor determining the progression of peripheral atherosclerotic disease to arterial ulceration. Interventions to enable patients with PAOD to quit smoking should be consistently addressed. Increased number of pack years of smoking is associated with increased risk of amputations, vascular graft occlusions and mortality. Behavior modification, nicotine replacement therapy and anti-depressive drugs should be advocated, along with referral to smoking cessation programs.
- 3.3 Frequent exercise, usually starting at 30 minutes a day, 3 times a week, leads to increased walking distance and improves the distance to claudication pain, as well as peripheral circulation and pulmonary function. Stabilization of PAOD symptoms is achieved by development of collaterals, adaptation of ischemic muscle and alteration of gait in favor of non-ischemic muscle groups. Effective exercise therapy requires a comprehensive assessment of patient needs and functional status. The role of exercise in preventing new or recurrent arterial ulcers has not been investigated. Exercise, while clearly beneficial, may increase the risk of trauma, pressure and ulceration. Therefore, a supervised exercise program is ideal.
- 3.4 Aspirin therapy delays the rate of progression of peripheral atherosclerotic disease and reduces the need for intervention. Antiplatelet therapy (aspirin or clopidigrel) is also indicated to reduce the risk of myocardial infarction, stroke and vascular death in patients with PAOD.

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- 3.5 Use of lipid-lowering therapies decreases long-term ischemic arterial ulcer development; statin-class drugs are currently preferred, although the benefit of their non-lipid-related mechanisms of actions is not established.
- 3.6 Monitoring of blood sugar and management of diabetes mellitus (DM) using recent guidelines should be continually addressed, including determination of hemoglobin A1C levels. Diabetes control is directly associated with prevention of PAOD ischemic ulcers since DM increases the risk of PAOD three- to four-fold, and can result in infection and immunosuppression.
- 3.7 B vitamins and/or folate were not shown to benefit treatment of hyperhomocysteinemia and improve PAOD. This is, therefore, not recommended.
- 3.8 Lower extremity protection should be aggressively pursued in patients with known or suspected PAOD, whether or not the patient has associated neuropathy from diabetes or other causes. The oxygen requirement for intact skin is significantly lower than for injured skin. The uninjured limb may be stable, but even minor skin trauma may be catastrophic in patients with PAOD.
 - a) Foot protection with soft, conforming, proper fitting shoes (foot deformity/altered biomechanics), casts, orthotics and offloading are key to prevention of ulceration by decreasing tissue trauma. Prevention of injury is essential to limb preservation.
 - b) Leg protection is equally important as foot protection. Patients should be educated on how to prevent even minor injury to the lower extremity.
 - c) Protection of digits and heels in bedridden or hospitalized patients with effective pressure relief using, for example, foam or air cushion boots, should be standard. In patients with PAOD, development of a pressure ulcer frequently leads to amputation.

- d) In patients with co-existing neuropathy, special attention should be paid to foot protection with proper footwear and off-loading. Loss of protective sensation to the foot in PAOD patients, especially when associated with DM and Buerger's disease, increases the risk of injury and may delay recognition of injuries, which increases the risk of amputation.
 - e) Patients with PAOD, particularly if DM is a comorbidity, should exercise extreme caution in cutting their toenails. In general, toenail care for patients with known PAOD should be performed by a podiatrist or other specialist, particularly if the pulse is absent or there is significant neuropathy.
- 3.9 External pneumatic compression has been shown to improve arterial inflow in ischemic limbs and may be of benefit in treating or preventing arterial ulcers.
- 3.10 Passive warming of the extremity, as with a Rooke boot, has been shown to improve perfusion in ischemic limbs and is likely to be beneficial in preventing or treating arterial ulcers.
- 3.11 Venous insufficiency frequently co-exists with PAOD and DM. Compression is the primary treatment for venous insufficiency and should be instituted to the extent possible. The ideal means of achieving adequate compression in patients with PAOD without causing or worsening ulceration or increasing the risk of occlusion or failure after revascularization has not been adequately investigated. This is a crucial area of investigation for prevention of arterial ulcers.
- 3.12 Poor psychosocial status (i.e., patients with serious psychiatric illness, living alone, alcohol abuse, poor body hygiene or malnutrition) is associated with an increased risk of an ischemic arterial ulcer and should be addressed by a multidisciplinary care team.

4. Preventive Interventions: Surgical

- 4.1 There is currently no defined role for lower extremity revascularization (open or endovascular) to prevent the progression of PAOD to arterial ulceration. Because claudication rarely progresses to CLI, surgical revascularization for claudication should be limited to patients with a significant functional disability. The presence of CLI is a clear indication for surgical revascularization to prevent limb loss.

At the present time, however, there is no indication for earlier revascularization, i.e., in patients without CLI. Nonetheless, it is important for the future to identify patients in whom the benefits of surgical management of PAOD (without ulceration or CLI) will outweigh the perioperative risks, which may be substantial. The development and advances in endovascular revascularization procedures may be changing the risk-benefit analysis; a formal evaluation would provide important data needed for determining the appropriate treatment in a given patient. Early referral to a vascular specialist for assessment is critical to the success of interventions. In all patients, whether revascularization is indicated or not, lifestyle modifications remain the most important preventive intervention.

- 4.2 Long-term administration of adjunctive medications should be routine. Anti-platelet agents (aspirin and/or clopidogrel) should be routine after revascularization. Treatment with aspirin and/or clopidogrel improves long-term postoperative patency after peripheral artery bypass grafting, stenting or angioplasty. It has been shown to decrease the progression of femoral artery atherosclerosis.

Other adjunctive agents that are less well studied but may have a role include anticoagulants such as coumadin, and low molecular weight heparins and statin drugs. Levels of evidence for these agents are continually evolving. The PREVENT trial examined the use of an antiproliferative antisense oligonucleotide (E2F) administered to vein bypass grafts at the time of implantation and found that there was no benefit in terms of re-stenosis or graft patency.

Condensed from: A. Barbul et al. Prevention Guidelines,
Wound Rep Reg. 2008; 16: 145-188.

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<http://www3.interscience.wiley.com/journal/119413535/issue>

This pocket guide was abridged by WHS member Stéphanie Bernatchez and donated by 3M. It summarizes the Wound Healing Society's Chronic Wound Prevention Guidelines published in March 2008.



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70-2009-8682-9