Guidelines for the treatment of pressure ulcers

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Health care providers face the challenge of providing effective care for increasing numbers of patients with chronic wounds. Pressure ulcers, one type of chronic wound, are estimated to affect 1.3–3 million individuals in the United States.[1] Prevalence varies among specific clinical populations, with higher percentages reported for the elderly, the acutely ill, and those who have sustained spinal cord injuries.[2,3,4] The first comprehensive clinical practice guidelines for the treatment of patients with pressure ulcers were published by the Agency for Healthcare Research and Quality (AHRQ) in 1994. Since that time, a number of professional groups have also developed and published guidelines.

The acceptance and adoption of guideline recommendations in practice is variable and influenced by several factors, including (1) guideline currency with the most recent and comprehensive evidence, (2) recognition and acceptance of guideline validity, (3) breadth of interprofessional representation in guideline development, and (4) guideline presentation and format.[5] These issues pertain to guidelines in general, but are also applicable to those specific to chronic wounds. Despite many recent advances in wound care, the challenge of managing chronic wounds remains compounded by the current lack of consensus on clearly defined, comprehensive wound care principles and uniformly accepted analytical methods to evaluate outcomes. With these concerns in mind, the following guidelines were developed to facilitate use by multiple groups in the wound care community of clinicians, researchers, industry, governing agencies, and third-party payers.

The guidelines provide recommendations for treatment of pressure ulcers supported by current evidence. However, treatment decisions also depend on specific patient characteristics, pressure ulcer characteristics/stage, patient circumstances, and overall goals. The development of a treatment plan of care begins with the determination of the goals of therapy. In most cases, the goal of therapy is to produce complete healing with restoration of functional skin integrity to the highest extent possible. However, in certain cases, the goal of therapy may not be complete healing of the wound. For example, in patients who are terminally ill, the goal of therapy may be palliative and focused on reducing discomfort or deterioration of the pressure ulcer, rather than complete healing of the wound. In other cases, the treatment may produce added discomfort or increased risk to the patient. Individual evaluation of each case is necessary within the context of the optimum outcome for that patient.

The specific objectives of this project were to:

1. Develop comprehensive, evidence- and consensus-based guidelines for pressure ulcer treatment.
2. Present these guidelines in a clear, simple format designed to enable health care providers to make informed, evidence-supported treatment decisions to manage pressure ulcers appropriately.

METHODS

A search of health care databases for current published evidence-based guidelines addressing the treatment of pressure ulcers was conducted between July 2004 and January 2006 using electronic and online resources. In addition to published guidelines, PubMed, EMBASE, and the Cochrane Database of Systematic Reviews were reviewed for evidence on pressure ulcer treatment. The following guidelines were located and reviewed by the panel and used in the development of the categories of treatment and individual guidelines.

1. American Family Physician Pressure Ulcer Guideline Panel. Pressure Ulcer Treatment. *Am Fam Phys* 1995; 51: 1207–23.
2. American Medical Directors Association (AMDA). *Pressure Ulcers, Clinical Practice Guideline*. 1996. Columbia, MD: American Medical Directors Association.
3. *AMDA Pressure Ulcer Therapy Companion, Clinical Practice Guideline*. 1999. Columbia, MD: American Medical Directors Association.
4. Bergstrom N, Allman RM, Alvarez OM, Bennett MA, Carlson CE, Frantz RA, Garber SL, Jackson BS, Kaminski Jr MV, Kemp MG, Krouskop TA, Lewis Jr VL, Maklebust J, Margolis DJ, Marvel EM, Reger SI, Rodeheaver GT, Salcido R, Xakellis GC, Yarkony GM. *Treatment of pressure ulcers. Clinical Practice Guideline*, No. 15. 1994. AHCPR Publication No. 95-0652. Rockville, MD: U.S. Department of Health and Human Services. Public Health service, Agency for Health Care Policy and Research.

5. Brem H, Lyder C. Protocol for the successful treatment of pressure ulcers. *Am J Surg* 2004 (Suppl. to July 2004); 188: 9S–17S.
6. Consortium for Spinal Cord Medicine. *Pressure Ulcer Prevention and Treatment following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals*. 2001. Washington, DC: Paralyzed Veterans of America.
7. European Guidelines for Pressure Ulcer Treatment (2004). http://www.epuap.org/
8. Folkedahl BA, Frantz R, Goode C. *Evidence-Based Protocol Treatment of Pressure Ulcers*. 2002. Iowa City: The University of Iowa Gerontological Nursing Interventions Research Center Research Dissemination Core (RDC).
9. Panel for the Prediction and Prevention of Pressure Ulcers in Adults. *Pressure ulcers in adults: prediction and prevention*. Clinical Practice Guideline, No. 3 1992. AHCPR Publication No. 92-0047. 1992. Rockville, MD: U.S. Department of Health and Human Services. Public Health service, Agency for Health Care Policy and Research.
10. Royal College of Nursing. Pressure ulcer risk assessment and prevention. 2001. http://www.nelh.nhs.uk/guidelinesdb/html/PrUlcer-fthtm. Accessed 7/5/04.
11. Schols JMGA, Jager-v.d.Ende MA. Nutritional Intervention in Pressure Ulcer Guidelines: An inventory. *Nutrition* 2004; 20: 548–53.
12. Wound Ostomy Continence Nurses Society. *Guideline for Prevention and Management of Pressure Ulcers*. 2003. WOCN: Glenview, IL.

The panel used a consensus process to determine the treatment categories. Subgroups of the panel (two to three individuals) were responsible for the development of specific guidelines and review of evidence within treatment categories. The first complete document was reviewed by the full panel and revised. The guidelines were presented for public comment in a forum hosted on the National Institutes of Health (NIH) campus (October 2005). Guidelines were further revised based on verbal and written comments received during the public forum review process. This revision was submitted to full panel review and additional modification before adoption. Additional revisions are based on review and critique provided by the board members of the Wound Healing Society and Wound Healing Foundation.

Evidence and Scientific Basis for Guidelines

The panel identified six categories of pressure ulcer treatment: positioning and support surfaces, nutrition, infection, wound bed preparation, dressings, and surgery and adjuvant therapies. Specific guidelines and the underlying principle(s) were developed in each category. Evidence references for each standard are listed and coded. The code abbreviations for the evidence citations were as follows:

STAT	Statistical analysis, meta-analysis, consensus statement by commissioned panel of experts
RCT	Randomized clinical trial
CLIN S	Clinical series
COMP	Comparative study
LIT REV	Literature review
RETROS	Retrospective series review
SURV	Survey
EXP	Laboratory or animal study
TECH	Technique or methodology description
COST ANAL	Cost analysis
PATH S	Pathological series review

Classification of Evidence

Our approach differed from the previous approaches used in evidence-based guidelines. In most published guidelines, evidence was based on clinical human studies. Laboratory or animal studies were not cited. Our approach was not limited to human clinical studies or to a specific study design (e.g., RCT). We have used well-controlled animal studies that present proof of principle, especially when a clinical series corroborated the laboratory results. It was also clear that principles that have been validated for other chronic wound types often are applicable to pressure ulcers. Therefore, evidence is included for some guidelines that were not specific for pressure ulcers. Because of these variations, a different system was necessary to grade the evidence weight supporting a given guideline. The strength of evidence supporting a guideline is listed as Level I, Level II, or Level III using the following definitions:

- *Level I:* Meta-analysis of multiple RCTs or at least two RCTs supporting the intervention in the guideline or multiple laboratory or animal experiments with at least two clinical series supporting the laboratory results.
- *Level II:* Less evidence than Level I, but at least one RCT and at least two significant clinical series or expert opinion papers with literature reviews supporting the intervention. Experimental evidence that is quite convincing but without support by adequate human experience is included.
- *Level III:* Suggestive data of proof of principle, but lacking sufficient data such as meta-analysis, RCT, or multiple clinical series.

References:

1. Lyder CH. Pressure ulcer prevention and management. *JAMA* 2003; 289: 223–6.
2. Barrois B, Allaert FA, Colin D. A survey of pressure sore prevalence in hospitals in the greater Paris region. *J Wound Care* 1995; 4: 234–6.
3. Allman RM, Paprade CA, Noel LB, et al. Pressure sores among hospitalized patients. *Ann Intern Med* 1986; 105: 337–42.
4. Walter JS, Sacks J, Othman R, et al. A database of self-reported secondary medical problems among VA spinal cord injury patients: its role in clinical care and management. *J Rehab Res Dev* 2002; 39: 53–61.
5. Shiffman RN, Dixon J, Brandt C, Essahihi A, Hsiao A, Michel G, O'Connell R. The GuideLine implementability appraisal (GLIA): Development and validation of an instrument to identify obstacles to guideline implementation. *BMC Med Inform Dec Making* 2005; 5: 1–23.

RESULTS

1. POSITIONING AND SUPPORT SURFACES

Preamble: Pressure and compression to soft tissue play a role in the etiology of pressure ulcers. Patient positioning and methods to reduce pressure-related tissue damage are recognized as important treatment components. While there are limited definitive studies, the best current evidence and expert opinion suggest the following guidelines.

Guideline #1.1: Establish a repositioning schedule and avoid positioning patients on a pressure ulcer. (Level II)

Principle: Pressure ulcers are thought to result from compression of soft tissues against a bony prominence. It is reasonable to assume that pressure on an ulcer can result in delayed healing. Patients should be repositioned to relieve pressure over bony prominences. The exact turning interval is not known and is derived empirically. Reductions in pressure incidence have been achieved, but positioning is not universally effective.

Evidence:

1. Clark M. Repositioning to prevent pressure sores—what is the evidence? *Nurs Standard* 1998; 13: 56–64. [LIT REV]
2. Defloor T. Less frequent turning intervals and yet less pressure ulcers. *Tijdschrift voor Gerontologie en Geriatrie* 2001; 32: 174–7. [RCT]
3. Knox DM, Anderson TM, Anderson PS. Effects of different turn intervals on skin of healthy older adults. *Adv Wound Care* 1994; 7: 48–56. [COMP]
4. Thomas DR. Are all pressure ulcers avoidable? *J Am Med Directors Assoc* 2001; 2: 297–301. [LIT REV]

Guideline #1.2: Maintain the head of the bed at the lowest degree of elevation consistent with medical conditions and other restrictions. Limit the amount of time the head of the bed is elevated and elevate only when there is a compelling medical indication (e.g., 1–2 hours after tube feeding or with severe respiratory or cardiac compromise). (Level III)

Principle: Elevation of the head of the bed produces shear and friction forces between the skin and the bed surface. Friction and shear may predispose to the development of pressure ulcers.

Evidence:

1. Thomas DR. Management of pressure ulcers. *J Am Med Directors Assoc* 2006; 7(1): 46–59. [LIT REV]

Guideline #1.3: Assess all patients for risk of developing a pressure ulcer. Use a pressure-reducing surface in those patients at risk. A pressure-reducing surface is superior to a standard hospital mattress in reducing the incidence of pressure ulcers. (Level I)

Principle: When compared with a standard hospital mattress, a variety of pressure-reducing devices can lower the incidence of pressure ulcers by about 60 percent.

Evidence:

1. Cullum N, McInnes E, Bell-Syer SEM, Legood R. Support surfaces for pressure ulcer prevention. *The Cochrane Database of Systematic Reviews* 2004; 3. [STAT]
2. Thomas DR. Issues and Dilemmas in Managing Pressure Ulcers. *J Gerontol: Med Sci* 2001; 56: M238–340. [LIT REV]

Guideline #1.4: A static support surface may be appropriate for patients with a pressure ulcer who can assume a variety of positions without placing pressure on the ulcer or "bottoming out." No difference in pressure ulcers outcomes is documented among different types of static devices. (Level I)

Principle: Static pressure-reducing devices are superior to standard hospital mattresses. However, if the patient "bottoms out" (if there is less than one inch of material between the bed and the pressure ulcer when feeling under the support surface with the palm of your hand), the device may be ineffective.

Evidence:

1. Cullum N, McInnes E, Bell–Syer SEM, Legood R. Support surfaces for pressure ulcer prevention. *The Cochrane Database of Systematic Reviews* 2004; 3. [STAT]

Guideline #1.5: A dynamic support surface may be appropriate for patients with a pressure ulcer who cannot assume a variety of positions in bed, or who "bottom out" on a static surface, or whose ulcer is failing to progress toward healing. (Level I)

Principle: Although some patients improve on a static support surface, there is evidence that other patients have an improved outcome on a dynamic support surface. No difference among studied types of dynamic devices has been shown.

Evidence:

1. Cullum N, McInnes E, Bell-Syer SEM, Legood R. Support surfaces for pressure ulcer prevention. *The Cochrane Database of Systematic Reviews* 2004; 3. [STAT]

Guideline #1.6: In patients who have a large stage 3 or stage 4 pressure ulcer, or multiple pressure ulcers involving several turning surfaces, a low-air-loss or air-fluidized bed may be indicated. (Level I)

Principle: Several studies have shown improved outcomes for pressure ulcers in patients treated with a low-air-loss or air-fluidized bed. However, these beds have some limitations, including difficulty for patients in self-positioning or for patients with pulmonary compromise.

Evidence:

1. Allman RM, Walker JM, Hart MK, et al. Air-fluidized beds or conventional therapy for pressure sores: a randomized trial. *Ann Intern Med* 1987; 107: 641–8. [RCT]
2. Cullum N, McInnes E, Bell-Syer SEM, Legood R. Support surfaces for pressure ulcer prevention. *The*

Cochrane Database of Systematic Reviews 2004; 3. [STAT]
3. Economides NG, Skoutakis VA, Carter CA. Evaluation of the effectiveness of two support surfaces following myocutaneous flap surgery. *Adv Wound Care* 1995; 8: 49–53. [RCT]
4. Munro BH, Brown L, Heitman BB. Pressure ulcers: one bed or another? *Geriatric Nurs* 1989; 10: 190–2. [LIT REV]

Guideline #1.7: A patient at risk for a pressure ulcer should avoid prolonged sitting. Postural alignment, distribution of weight, balance, stability, and pressure reduction should be considered in seated individuals. (Level III)

Principle: Tissue compression between the sitting surface and bony prominence should be relieved in at-risk patients. In patients with a pressure ulcer, sitting on the pressure ulcer should be avoided. Reposition the sitting individual to relieve pressure at least every hour. If this schedule cannot be maintained, return the patient to bed. Individuals should be instructed to shift their weight every 15 minutes.

Guideline #1.8: Use a seat cushion based on the needs of the individual who requires pressure reduction in the sitting position. Avoid using doughnut-type devices. (Level III)

Principle: Several seat cushions reduce pressure in sitting individuals. Examine seating cushions and devices for "bottoming out." There is insufficient evidence on the value of seat cushions in the prevention of pressure ulcers. Ring cushions (doughnut) devices increase venous congestion and edema.

2. NUTRITION

Preamble: Protein, carbohydrates, vitamins, minerals, and trace elements are required for wound healing. Nutrition is valued and considered in practice as a significant factor in the prevention and treatment of pressure ulcers. However, there are limited definitive studies documenting the efficacy of nutritional treatments for pressure ulcer healing. The following guidelines reflect the best current evidence and expert opinion.

Guideline #2.1: Nutritional assessment should be performed on entry to a new healthcare setting and whenever there is a change in an individual's condition that may increase the risk of undernutrition. (Level II)

Principle: Nutrition must be adequate to provide sufficient protein to support the growth of granulation tissue. The patient's weight on entry to the healthcare system is a good starting point. Assess body weight whenever there is a change in an individual's condition that may increase the risk of undernutrition. Achieving a weight as close to the ideal body weight as possible is the goal. Assessment of pre-albumin level (reflecting recent protein consumption) and serum albumin level (reflecting long-term protein consumption) is useful to identify patients who are outside the norm. Encourage nutritional support if an individual is undernourished. Undernutrition is associated with poor clinical outcomes, including increased risk of mortality, so early identification of actual or potential nutritional need allows for timely intervention to mitigate nutritional decline. No studies were identified that specifically address the issue of obesity and pressure ulcer development.

Evidence:

1. Allman RM, Laprade CA, Noel LB. Pressure sores among hospitalized patients. *Ann Intern Med* 1986; 105(3): 337–42. [STAT]
2. Baker JP, Detsky AS, Withwell J, Langer B, Jeejeebhoy KN. A comparison of the predictive value of nutritional assessment techniques. *Hum Nutr Clin Nutr* 1982; 36C: 233–41. [CLIN S]
3. Bourdel Marchasson I, Barateau M, Rondeau V, DequaeMerchadou L, SallesMontaudon N, Emeriau JP, Manciet G, Dartigues JF. A multicenter trial of the effects of oral nutritional supplementation in critically ill older inpatients. GAGE Group. Groupe Aquitain Gériatrique d'Evaluation. *Nutrition* 2000; 16: 15. [RCT]
4. Hartgrink HH, Wille J, Konig P, Hermans J, Breslau PJ. Pressure sores and tube feeding in patients with a fracture of the hip. *Clin Nutr* 1998; 17: 287–92. [RCT]
5. Houwing R, Rozendaal M, WoutersWesseling W, Beulens JWJ, Buskens E, Haalboom J. A randomized, double-blind assessment of the effect of nutritional supplementation on the prevention of pressure ulcers in hip fracture patients. *Clin Nutr* 2003; 22(4): 401–5. [RCT]
6. Murden RA, Ainslie NK. Recent weight loss is related to short-term mortality in nursing homes. *J Gen Intern Med* 1994; 9: 648–50. [CLIN S]
7. Rudman D, Feller AG, Nagraj HS, Jackson DL, Rudman IW, Mattson DE. Relation of serum albumin concentration to death rate in nursing home men. *J Parenter Ent Nutr* 1987; 11: 360. [CLIN S]
8. Rypkema G, Adang E, Dicke H, Naber T, de Swart B, Disselhorst L, Goluke-Willemse G, Olde Rikkert M. Cost-effectiveness of an interdisciplinary intervention in geriatric inpatients to prevent malnutrition. *J Nutr Health Aging*. 2004; 8(2): 122–7. [CLIN S]
9. Salzberg CA, Byrne DW, Cayten CG, et al. A new pressure ulcer risk assessment scale for individuals with spinal cord injury. *Am J Phys Med Rehabil* 1996 Mar–Apr; 75(2): 96–104. [CLIN S]
10. Schue RM, Langemo DK. Prevalence, incidence, and prediction of pressure ulcers on a rehabilitation unit. *J Wound Ostomy Continence Nurs* 1999 May; 26(3): 121–9. [RETRO S]
11. Scivoletto G, Fuoco U, Morganti B. Pressure sores and blood and serum dysmetabolism in spinal cord injury patients. *Spinal Cord* 2004 Aug; 42(8): 473–6. [RCT]
12. Sullivan DH, Johnson LE, Bopp MM, Roberson PK. Prognostic significance of monthly weight fluctuations among older nursing home residents. *J Gerontol Ser A: Biol Sci Med Sci* 2004; 59: M633–M639. [RCT]
13. Thomas DR, Verdery RB, Gardner L, Kant AK, Lindsay J. A prospective study of outcome from protein-energy malnutrition in nursing home residents. *J Parenteral Enteral Nutr* 1991; 15: 400–04. [CLIN S]
14. Volkert D, Kruse W, Oster P, Schlierf G. Malnutrition in geriatric patients: diagnostic and prognostic

significance of nutritional parameters. *Ann Nutr Metab* 1992; 36: 97–112. [CLIN S]

Guideline #2.2: Encourage dietary intake or supplementation if an individual who is undernourished is at risk of developing a pressure ulcer. (Level III)

Principle: Nutrients are basic to cellular integrity and data suggest that a nutritional supplement may have a modest effect in preventing the development of pressure ulcers, largely in stage 1 ulcers.

Evidence:

1. Bourdel-Marchasson I, Barateau M, Rondeau V, Dequae-Merchadou L, Salles-Montaudon N, Emeriau JP, Manciet G, Dartigues JF. A multi-center trial of the effects of oral nutritional supplementation in critically ill older inpatients. GAGE Group. Groupe Aquitain Gériatrique d'Evaluation. *Nutrition* 2000; 16: 1–5. [RCT]
2. Delmi M, Rapin CH, Bengoa JM, Delmas PD, Vasey H, Bonjour JP. Dietary supplementation in elderly patients with fractured neck of the femur. *Lancet* 1990; 335(8696): 1013–6. [RCT]
3. Hartgrink HH, Wille J, Konig P, Hermans J, Breslau PJ. Pressure sores and tube feeding in patients with a fracture of the hip: a randomized clinical trial. *Clin Nutr* 1998; 17: 287–92. [RCT]
4. Houwing R, Rozendaal M, Wouters-Wesseling W, Beulens JWJ, Buskens E, Haalboom J. A randomized, double-blind assessment of the effect of nutritional supplementation on the prevention of pressure ulcers in hip-fracture patients. *Clin Nutr* 2003; 22(4): 401–5. [RCT]
5. Langer G, Schloemer G, Knerr A, Kuss O, Behrens J. Nutritional interventions for preventing and treating pressure ulcers. *The Cochrane Database of Systematic Reviews 2003*, Issue 4, Art. No: CD003216. DOI:10.1002/14651858. CD003216. [STAT]

Guideline #2.3: Ensure adequate dietary intake to prevent undernutrition to the extent that this is compatible with the individual's wishes. (Level III)

Principle: Adequate nutrition is essential for life and undernutrition is associated with the development of pressure ulcers. Nonetheless, the nutritional plan needs to be consistent with the individual's personal goals.

Evidence:

1. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. *J Am Geriatr Soc* 1992; 40: 747–58. [CLIN S]
2. Berlowitz DR, Wilking SVB. Risk factors for pressure sores: A comparison of cross-sectional and cohort-derived data. *J Am Geriatr Soc* 1989; 37: 1043–50. [CLIN S]
3. Finucane TE. Malnutrition, tube feeding and pressure sores: data are incomplete. *J Am Geriatr Soc* 1995; 43: 447–51. [LIT REV]
4. Thomas DR. Specific nutritional factors in wound healing. *Adv Wound Care* 1997; 10: 40–3. [LIT REV]
5. Thomas DR. The role of nutrition in prevention and healing of pressure ulcers. *Geriatr Clin North Am* 1997: 13: 497–512. [LIT REV]
6. Thomas DR, Ashmen W, Morley JE, Evans JE. Nutritional management in long-term care: development of a clinical guideline. *J Gerontol: Med Sci* 2000; 55: 725–34. [STAT]
7. Thomas DR, Goode PS, Tarquine PH, Allman R. Hospital-acquired pressure ulcers and risk of death. *J Am Geriatr Soc* 1996; 44: 1435–40. [CLIN S]

Guideline #2.4: If dietary intake continues to be inadequate, impractical, or impossible, nutritional support (usually tube feeding) should be used to place the patient into positive nitrogen balance (approximately 30–35 calories/kg/day and 1.25–1.50 g of protein/kg/day) according to the goals of care. (Level III)

Principle: Anabolism is facilitated with a positive nitrogen balance and when individuals are not able to meet nutritional needs through oral intake, alternative methods should be undertaken to optimize nutritional status.

Evidence:

1. Chernoff RS, Milton KY, Lipschitz DA. The effect of a very high-protein liquid formula on decubitus ulcers healing in long-term tube-fed institutionalized patients. *J Am Diet Assoc* 1990; 90: A-130. [CLIN S]
2. Henderson CT, Trumbore LS, Mobarhan S, Benya R, Miles TP. Prolonged tube feeding in long-term care: Nutritional status and clinical outcomes. *J Am Coll Clin Nutr* 1992; 11: 309. [CLIN S]
3. Long CL, Nelson KM, Akin JM Jr, Geiger JW, Merrick HW, Blakemore WZ. A physiologic basis for the provision of fuel mixtures in normal and stressed patients. *J Trauma* 1990; 30: 1077–86. [CLIN S]
4. Mathus-Vliegen EM. Old age, malnutrition, and pressure sores: an ill-fated alliance. *J Gerontol Ser A, Biol Sci Med Sci* 2004 Apr; 59(4): 355–60. [LIT REV]
5. Mitchell SL, Kiely DK, Lipsitz LA. The risk factors and impact on survival of feeding tube placement in nursing home residents with severe cognitive impairment. *Arch Intern Med* 1997; 157: 327–32. [CLIN S]
6. Thomas DR. Improving the outcome of pressure ulcers with nutritional intervention: a review of the evidence. *Nutrition* 2001; 17: 121–25. [LIT REV]

Guideline #2.5: Give vitamin and mineral supplements if deficiencies are confirmed or suspected. (Level III)

Principle: Supplements of vitamins and minerals that are needed for wound healing should be provided when intake is insufficient or when a deficit is identified. No acceleration in healing has been reported with supplemental Vitamin A, Vitamin C, or zinc. Amino acids supplements have been effective in the healing of some non-pressure-related wounds. Arginine has not been found to accelerate healing in patients with pressure ulcers.

Evidence:

1. Ehrlich HP, Hunt TK. Effects of cortisone and vitamin A on wound healing. *Ann Surg* 1968; 167: 324. [EXP]

2. Goode P, Allman R. The prevention and management of pressure ulcers. *Med Clin North Am* 1989; 73: 1511–1424. [LIT REV]
3. Gregger JL. Potential for trace mineral deficiencies and toxicities in the elderly. In: Bales CW, editor. *Mineral Homeostasis in the Elderly*. New York: Marcel Dekker, 1989: 171–200. [LIT REV]
4. Hallbook T, Lanner E. Serum zinc and healing of leg ulcers. *Lancet* 1972; 2: 780. [RCT]
5. Houwing R, Rozendaal M, Wouters-Wesseling W, Beulens JWJ, Buskens E, Haalboom JR. A randomized, double-blind assessment of the effect of nutritional supplementation on the prevention of pressure ulcers in hip-fracture patients. *Clin Nutr* 2003; 22(4): 401–5. [RCT]
6. Hunt TK. Vitamin A and wound healing. *J Am Acad Dermatol* 1986; 15: 817–21. [LIT REV]
7. Langer G, Schloemer G, Knerr A, Kuss O, Behrens J. Nutritional interventions for preventing and treating pressure ulcers. The Cochrane Database of Systematic Reviews 2003, Issue 4, Art. No: CD003216. DOI: 10.1002/14651858. CD003216. [STAT]
8. Langkamp-Henken B, Herrlinger-Garcia KA, Stechmiller JK, Nickerson-Troy JA, Lewis B, Moffatt L. Arginine supplementation is well tolerated but does not enhance mitogen-induced lymphocyte proliferation in elderly nursing home residents with pressure ulcers. *JPEN* 2000; 24: 280–7. [RCT]
9. Norris JR, Reynolds RE. The effect of oral zinc sulfate therapy on decubitus ulcers. *J Am Geriatr Soc* 1971; 19: 793. [CLIN S]
10. Prasad AS. Discovery of human zinc deficiency and studies in an experimental human model. *Am J Clin Nutr* 1991; 53: 403–12. [RETRO S]
11. Rackett SC, Rothe MJ, Grant-Kels JM. Diet and dermatology. The role of dietary manipulation in the prevention and treatment of cutaneous disorders. *J Am Acad Dermatol* 1993; 29: 447–61. [CLIN S]
12. Sandstead SH, Henrikson LK, Greger JL, et al. Zinc nutrition in the elderly in relation to taste acuity, immune response, and wound healing. *Am J Clin Nutr* 1982; 36(Suppl.): 1046. [CLIN S]
13. Ter Riet G, Kessels AG, Knipschild PG. Randomized clinical trial of ascorbic acid in the treatment of pressure ulcers. *J Clin Epidemiol* 1995; 48: 1453–60. [RCT]
14. Vilter RW. Nutritional aspects of ascorbic acid: uses and abuses. *West J Med* 1980; 133: 485. [LIT REV]
15. Waldorf H, Fewkes J. Wound healing. *Adv Dermatol* 1995; 10: 77–96. [LIT REV]

3. INFECTION

Preamble: Infection results when the bacteria:host defense equilibrium is upset in favor of the bacteria. Infection plays various roles in the etiology, healing, operative repair, and complications of pressure ulcers. Therefore, guidelines are necessary to address the treatment of infection under each of these circumstances.

Guideline #3.1: Treat distant infections (e.g., urinary tract, cardiac valves, cranial sinuses) with appropriate antibiotics in pressure-ulcer-prone patients or patients with established ulcers. (Level II)

Principle: Bacteria entering the bloodstream or lymphatics can lodge in compressed tissue, denervated tissue, edematous tissue, or established wounds by the compromised tissue acting as a *locus minoris resistentiae*.

Evidence:

1. Alison WE, Phillips LG, Linares HA, et al. The effect of denervation on soft tissue infection pathophysiology. *Plast Reconstr Surg* 1992; 90: 1031–35. [EXP]
2. Groth KE. Clinical observations and experimental studies of the pathogens of decubitus ulcers. *Acta Chir Scand* 1942; 87 (Suppl. 76): 198–207. [EXP]
3. Hussain T. An experimental study of some pressure effects on tissues with reference to the bedsore problem. *J Pathol Bacteriol* 1953; 66: 347–58. [EXP]
4. Krizek TJ, Davis JH. Endogenous wound infection. *J Trauma* 1967; 6: 239–48. [EXP]
5. Richardson D. Diagnosis and management of systemic infections and fever in neurological patients. *Semin Neurol* 2000; 20: 387–91. [LIT REV]
6. Ricketts LR, Squire JR, Topley E, et al. Human skin lipids with particular reference to the self-sterilizing power of the skin. *Clin Sci Mol Med* 1951; 10: 89. [EXP]
7. Robson MC, Krizek TJ. The role of infection in chronic pressure ulcerations. In: Fredericks, S, Brody, GS. editors. *Symposium on Neurologic Aspects of Plastic Surgery*. St. Louis: CV Mosby Co, 1978. [EXP]
8. Wall BM, Mangold T, Huch KM, et al. Bacteremia in the chronic spinal cord injury population: risk factors for mortality. *J Spinal Cord Med* 2003; 26: 248–53. [CLIN S]

Guideline #3.2: Remove all necrotic or devitalized tissue by sharp, enzymatic, biological, mechanical, or autolytic debridement. (See detailed discussion of debridement in Wound Bed Preparation section of these guidelines.) (Level I)

Principle: Necrotic tissue is laden with bacteria while devitalized tissue impairs the body's ability to fight infection and serves as a pabulum for bacterial growth.

Evidence:

1. Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: a systematic review. *Health Technol Assess* 1999; 3: 1–78. [STAT]
2. Edlich RF, Rodeheaver GT, Thacker JG, et al. Technical factors in wound management. In: Dunphy, JE, Hunt, TK, editors. *Fundamentals Wound Manage Surg*. South Plainfield, NJ: Chirurgecom, 1977. [EXP]
3. Falanga V. Wound bed preparation and the role for enzymes: a case for multiple actions of therapeutic agents. *Wounds* 2002; 14: 47–57. [LIT REV]
4. Hamer MI, Robson MC, Krizek TJ, et al. Quantitative bacterial analyses of comparative wound irrigations. *Ann Surg* 1975; 181: 819–22. [EXP]
5. Steed D, Donohue D, Webster M, et al. Effect of extensive debridement and rhPDGF-BB (Becaplermin) on the healing of diabetic foot ulcers. *J Am Coll Surg* 1996; 183: 61–4. [RCT]

6. Witkowski, JA, Parrish, LC. Debridement of cutaneous ulcers: Medical and surgical aspects. *Clin Dermatol* 1992; 9: 585–91. [LIT REV]

Guideline #3.3: If there is suspected infection in a debrided ulcer, or if contraction and epithelialization from the margin are not progressing within two weeks of debridement and relief of pressure, determine the type and level of infection in the debrided ulcer by tissue biopsy or by a validated quantitative swab technique. (Level II)

Principle: High levels of bacteria $\geq 1 \times 10^6$ CFU/gram of soft tissue or any tissue level of beta hemolytic streptococci impede the various wound-healing processes and have been demonstrated to impede spontaneous healing and surgical closure of pressure ulcers. Cultures should be performed to isolate both aerobic and anaerobic bacteria.

Evidence:

1. Bendy RH, Nuccio PA, Wolfe E, et al. Relationship of quantitative wound bacterial counts to healing of decubiti. Effect of gentamicin. *Antimicrob Agent Chemo Ther* 1964; 4: 147–55. [RCT]
2. Heggers JP. Variations on a theme. In: Heggers, JP, Robson, MC, editors. *Quantitative Bacteriology: Its Role in the Armamentarium of the Surgeon*. Boca Raton: CRC Press, 1991.
3. Levine NS, Lindberg RB, Mason AD, et al. The quantitative swab culture and smear: a quick method for determining the number of viable aerobic bacteria in open wounds. *J Trauma* 1976; 16: 84–94. [TECH]
4. Nystrom PO. The microbiological swab sampler—a quantitative experimental investigation. *Acta Pathol Microbiol Scand* 1978; 86B: 361–7. [TECH]
5. Robson MC, Stenberg BD, Heggers JP. Wound healing alterations caused by infection. *Clin Plast Surg* 1990; 17: 485–92. [LIT REV]
6. Robson MC. Wound infection: a failure of wound healing caused by an imbalance of bacteria. *Surg Clin North Am* 1997; 77: 637–50. [LIT REV]
7. Sapico FL, Ginnas VJ, Thornhill-Joynes M, et al. Quantitative microbiology of pressure sores in different stages of healing. *Diagn Microbiol Infect Dis* 1986; 5: 31–8. [TECH]
8. Tobin GR. Closure of contaminated wounds: biologic and technical considerations. *Surg Clin North Am* 1984; 64: 639–52. [LIT REV]
9. Stephens P, Wall JB, Wilson MJ, et al. Anaerobic cocci populating the deep tissues of chronic wounds impair cellular wound healing responses in vitro. *Br J Dermatol* 2003; 148: 456–66. [CLIN S]
10. Volenec FJ, Clark GM, Manni MM, et al. Burn wound biopsy bacterial quantification: a statistical analysis. *Am J Surg* 1979; 138: 695–7. [STAT]

Guideline #3.4: For ulcers with $\geq 1 \times 10^6$ CFU/gram of tissue or any tissue level of beta hemolytic streptococci following adequate debridement, decrease the bacterial level with a topical antimicrobial. Once in bacterial balance, discontinue the use of topical antimicrobial to minimize any possible cytotoxic effects due to the antimicrobial agent or bacterial resistance to the agent. (Level I)

Principle: Systemically administered antibiotics do not effectively decrease bacterial levels in granulating wounds. However, topically applied antimicrobials can be effective.

Evidence:

1. Kucan JO, Robson MC, Heggers JP, et al. Comparison of silver sulfadiazine, povidone-iodine, and physiologic saline in the treatment of chronic pressure ulcers. *J Am Geriatr Soc* 1981; 24: 232–5. [RCT]
2. Robson MC, Mannari RJ, Smith PD, et al. Maintenance of wound bacterial balance. *Am J Surg* 1999; 178: 399–402. [RCT]
3. Robson MC, Heggers JP. Surgical infection: II. The β-hemolytic streptococcus. *J Surg Res* 1969; 14: 289–92. [EXP]
4. Robson MC, Edstrom LE, Krizek TJ, et al. The efficacy of systemic antibiotics in the treatment of granulating wounds. *J Surg Res*. 1974; 16: 299–306. [EXP]
5. Robson MC. Wound infection: A failure of wound healing caused by an imbalance of bacteria. *Surg Clin North Am* 1997; 77: 637–50. [LIT REV]
6. Stotts NA, Hunt TK. Managing bacterial colonization and infection. *Clin Ger Med* 1997; 13: 565–73. [LIT REV]

Guideline #3.5: Obtain bacterial balance ($<10^5$ CFU/gram of tissue and no beta hemolytic streptococci) in the pressure ulcer before attempting surgical closure by skin graft, direct wound approximation, pedicled, or free flap. (Level I)

Principle: "A wound containing contaminated foci with $>10^5$ organisms per gram of tissue cannot be readily closed, as the incidence of wound infection that follows is 50–100%."

Evidence:

1. Edlich RF, Rodeheaver GT, Thacker JG, Winn HA, Edgerton MT. Management of soft tissue injury. *Clin Plast Surg* 1977; 4: 191–8. [LIT REV]
2. Krizek TJ, Robson MC, Ko E. Bacterial growth and skin graft survival. *Surg Forum* 1967; 18: 518–9. [RCT]
3. Liedburg NC, Reiss E, Artz CP. The effect of bacteria on the take of split thickness skin grafts in rabbits. *Ann Surg* 1955; 142: 92–6. [EXP]
4. Murphy RC, Robson MC, Heggers JP, et al. The effect of microbial contamination on musculocutaneous and random flaps. *J Surg Res* 1986; 41: 75–80. [EXP]
5. Robson MC, Lea CE, Dalton JB, et al. Quantitative bacteriology and delayed wound closure. *Surg Forum* 1968; 19: 501–2. [RCT]
6. Robson MC, Krizek TJ, Heggars JP. Biology of surgical infection. *Curr Prob Surg* 1972; 10: 1–62. [LIT REV].
7. Tobin GR: Closure of contaminated wounds: biologic and technical considerations. *Surg Clin North Am* 1984; 64: 639–52. [LIT REV]

Guideline #3.6: Obtain bone biopsy for culture and histology in cases of suspected osteomyelitis associated with a pressure ulcer. (Level II)

Principle: The sensitivity and specificity of noninvasive tests for diagnosing osteomyelitis are not as high as direct bone biopsy and are not as useful in determining treatment.

Evidence:

1. Lewis VL, Bailey MH, Pulawski G, et al. The diagnosis of osteomyelitis in patients with pressure sores. *Plast Reconstr Surg* 1988; 81: 229–32. [RCT]
2. Han H, Lewis VL, Wiedrich TA, et al. The value of Jamshidi core needle bone biopsy in predicting postoperative osteomyelitis in grade IV pressure ulcer patients. *Plast Reconstr Surg* 2002; 110: 118–22. [RETRO S]
3. Huang AB, Schweitzer ME, Hume E, et al. Osteomyelitis of the pelvis/hips in paralyzed patients: accuracy and clinical utility of MRI. *J Comput Assist Tomogr* 1998; 22: 437–43. [CLIN S]
4. Sugarman B. Pressure sores and underlying bone infection. *Arch Int Med* 1987; 147: 553–5. [CLIN S]
5. Turk EE, Tsokos M, Delling G. Autopsy-based assessment of extent and type of osteomyelitis in advanced-grade sacral decubitus ulcers: a histopathologic study. *Arch Pathol Lab Med* 2003; 127: 1599–602. [PATH S]

Guideline #3.7: Once confirmed, osteomyelitis underlying a pressure ulcer should be adequately debrided and covered with a flap containing muscle or fascia. (Antibiotic choice, guided by culture results, should be used for three weeks.) (Level I)

Principle: Muscle, musculocutaneous, and fasciocutaneous flaps effectively control bacterial levels and antibiotics have been demonstrated by meta-analysis not to show additional efficacy beyond three weeks.

Evidence:

1. Calderon W, Chang N, Mathes SJ. Comparison of the effect of bacterial inoculation in musculocutaneous and fasciocutaneous flaps. *Plast Reconstr Surg* 1986; 77: 785–94. [EXP]
2. Chang N, Mathes SJ. Comparison of the effect of bacterial inoculation in musculocutaneous and random flaps. *Plast Reconstr Surg* 1982; 70: 1–10. [EXP]
3. Ger R. Muscle transposition for treatment and prevention of chronic post-traumatic osteomyelitis of the tibia. *J Bone Joint Surg* 1977; 59A: 784. [CLIN S]
4. Gosain A, Chang N, Mathes S, et al. A study of the relationship between blood flow and bacterial inoculation in musculocutaneous and fasciocutaneous flaps. *Plast Reconstr Surg* 1990; 86: 1152–62. [EXP]
5. Lazzarini L, Lipsky BA, Mader JT. Antibiotic treatment of osteomyelitis: what have we learned from 30 years of clinical trials? *Int J Infect Dis* 2005; 9: 127–38.
6. Mathes SJ, Feng LJ, Hunt TK: Coverage of infected wounds. *Ann Surg* 1983; 198: 420–9. [CLIN S]
7. Stengel D, Bauwens K, Sehoul J, et al. Systematic review and meta-analysis of antibiotic therapy for bone and joint infections. *Lancet Infect Dis* 2001; 1: 175–88. [STAT]
8. Thornhill-Joynes, M, Gonzales F, Stewart CA, et al. Osteomyelitis associated with pressure ulcers. *Arch Phys Med Rehabil* 1986; 67: 314–8. [RETRO S]

4. WOUND BED PREPARATION

Preamble: Wound bed preparation is defined as the management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures. The aim of wound bed preparation is to convert the molecular and cellular environment of a chronic wound to that of an acute healing wound.

Guideline #4.1: Examination of the patient as a whole is important to evaluate and correct the causes of tissue damage. It is important to examine the patient's systemic diseases and medications. (Level I)

Principle: General medical history, including a medication record, will help in identifying and correcting systemic causes of impaired healing. Any major illness, systemic disease, or drug therapies that cause alterations in immune functioning, metabolism, nutrition, and tissue perfusion will interfere with wound healing. Systemic disease, such as systemic sepsis, organ failure (hepatic, renal, respiratory, gut), major trauma/burns, diabetes, autoimmune diseases, and drug therapies such as immunosuppressive drugs and systemic steroids, will delay wound healing. Autoimmune diseases such as rheumatoid arthritis, systemic lupus, uncontrolled vasculitis, or pyoderma gangrenosum can impair healing and may require systemic steroids or immunosuppressive agents for adequate control before local wound healing can occur. Patients undergoing major surgery have diminished wound-healing capacity. Smoking is associated with impaired wound healing and increased risk of infection.

Evidence:

1. William DT, Harding K. Healing responses of skin and muscle in critical illness. *Crit Care Med* 2003 Aug 31 (8 Suppl.): S547–57. [LIT REV]
2. Beer HD, Fassler R, Werner S. Glucocorticoid-regulated gene expression during cutaneous wound repair.*Vitam Horm* 2000; 59: 217–39. [EXP]
3. Vaseliso M, Guaitero E. A comparative study of some anti-inflammatory drugs in wound healing of the rat. *Experientia* 1973 Oct 15; 29(10): 1250–1. [EXP]
4. Jorgensen LN, Kallehave F, Karlsmark T, Gottrup F. Reduced collagen accumulation after major surgery. *Br J Surg* 1996 Nov; 83(11): 1591–4. [CLINICAL S]
5. Sorensen LT, Nielsen HB, Kharazmi A, Gottrup F. Effect of smoking and abstention on oxidative burst and reactivity of neutrophils and monocytes. *Surgery* 2004 Nov; 136(5): 1047–53. [RCT]
6. Sorensen LT, Karlsmark T, Gottrup F. Abstinence from smoking reduces incisional wound infection: a randomized controlled trial. *Ann Surg* 2003; 238: 1–5. [RCT]
7. Lazarus GS, Cooper DM, Knighton DR, et al. Definitions and guidelines for assessment of wounds and evaluations of healing. *Arch Dermatol* 1994; 130: 489–93. [STAT]

8. Mustoe T. Understanding chronic wounds. A unifying hypothesis on their pathogenesis and implications for therapy. *Am J Surg*; 187 (5A): 65s–70s. [LIT REV]

Guideline #4.2: Examination of the patient as a whole is important to evaluate and correct causes of tissue damage. It is important to examine the patient's nutritional status. (Level II)

Principle: Nutrition must be adequate to provide sufficient protein to support the growth of granulation tissue. Encourage nutritional support if an individual is undernourished. (Detailed discussion of nutrition is in Nutritional Guidelines.)

Evidence:

1. Allman RM, Laprade CA, Noel LB. Pressure sores among hospitalized patients. *Ann Intern Med* 1986 Sep; 105(3): 337–42. [STAT]
2. Bourdel Marchasson I, Barateau M, Rondeau V, DequaeMerchadou L, SallesMontaudon N, Emeriau JP, Manciet G, Dartigues JF. A multicenter trial of the effects of oral nutritional supplementation in critically ill older inpatients. GAGE Group. Groupe Aquitain Gériatrique d'Evaluation. *Nutrition* 2000; 16: 15. (RCT-multicenter study)
3. Hartgrink HH, Wille J, Konig P, Hermans J, Breslau PJ. Pressure sores and tube feeding in patients with a fracture of the hip. *Clin Nutr* 1998; 17: 287–92. [RCT]
4. Houwing R, Rozendaal M, WoutersWesseling W, Beulens JWJ, Buskens E, Haalboom J. A randomized, double-blind assessment of the effect of nutritional supplementation on the prevention of pressure ulcers in hip fracture patients. *Clin Nutr* 2003; 22(4): 401–5. [RCT]
5. Salzberg CA, Byrne DW, Cayten CG, et al. A new pressure ulcer risk assessment scale for individuals with spinal cord injury. *Am J Phys Med Rehabil* 1996 Mar–Apr; 75(2): 96–104. [CLIN S]
6. Schue RM, Langemo DK. Prevalence, incidence, and prediction of pressure ulcers on a rehabilitation unit. *J Wound Ostomy Continence Nurs* 1999 May; 26(3): 121–9. [RETRO S]
7. Scivoletto G, Fuoco U, Morganti B. Pressure sores and blood and serum dysmetabolism in spinal cord injury patients. *Spinal Cord* 2004 Aug; 42(8): 473–6. [RCT]

Guideline #4.3: Examination of the patient as a whole is important to evaluate and correct causes of tissue damage. It is important to examine the patient's tissue perfusion and oxygenation. (Level I)

Principle: Adequate tissue perfusion and oxygenation: Wounds will heal in an environment that is adequately oxygenated. Oxygen delivery to the wound will be impaired if tissue perfusion is inadequate. Dehydration and factors that increase sympathetic tone such as cold, stress, or pain will decrease tissue perfusion. Cigarette smoking decreases tissue oxygen by peripheral vasoconstriction.

Evidence:

1. Chang N, Goodson WH 111, Gottrup F, Hunt TK. Direct measurement of wound and tissue oxygen tension in postoperative patients. *Ann Surg* 1983 Apr; 197(4): 470–8. [CLIN S]
2. Gottrup F. Oxygen in wound healing and infection. *World J Surg* 2004 Mar; 28(3): 312–5. Epub 2004 Feb 17. [LIT REV]
3. Gottrup F. Prevention of surgical—wound infections (editorial). *N Engl J Med* 2000; 342: 202–4. [LIT REV]
4. Greif R, Akca O, Horn EP, Kurz A, Sessler DI. Supplemental perioperative oxygen to reduce the incidence of surgical-wound infection. Outcomes research group. *N Engl J Med* 2000; 342 (3): 161–7. [RCT]
5. Hunt TK, Aslam RS. Oxygen 2002: Wounds. *Undersea Hyperb Med* 2004; Spring; 31(1): 147–53. [LIT REV]
6. Hopf H, Hunt TK, West JM, et al Wound tissue oxygen tension predicts the risk of wound infection in surgical patients. *Arch Surg* 1997; 132(9): 997–1004. [CLIN S]
7. Hunt TK, Hopf HW. Wound healing and wound infection. What surgeons and anesthesiologists can do. *Surg Clin North Am* 1997 Jun; 77(3): 587–606. [LIT REV]
8. Jensen JA, Goodson WH, Hopf HW, Hunt TK. Cigarette smoking decreases tissue oxygen. *Arch Surg* 1991 Sep; 126(9): 1131–4. [RCT]
9. Jonsson K, Jensen JA, Goodson WH 3rd, Scheuenstuhl H, West J, Hopf HW, Hunt TK. Tissue oxygenation, anemia, and perfusion in relation to wound healing in surgical patients. *Ann Surg* 1991; 214(5): 605–13. [RCT]
10. Knighton DR, Halliday B, Hunt TK. Oxygen as an antibiotic. A comparison of the effects of inspired oxygen concentration and antibiotic administration on in vivo bacterial clearance. *Arch Surg* 1986 Feb; 121(2): 191–5. [EXP]

Guideline #4.4: Initial debridement is required to remove the obvious necrotic tissue, excessive bacterial burden, and cellular burden of dead and senescent cells. Maintenance debridement is needed to maintain the appearance and readiness of the wound bed for healing. The health care provider can choose from a number of debridement methods including sharp, mechanical, enzymatic, and autolytic. More than one debridement method may be appropriate. (Level I)

Principle: Necrotic tissue, excessive bacterial burden, senescent cells, and cellular debris can all inhibit wound healing. The method of debridement chosen may depend on the status of the wound, the capability of the health provider, the overall condition of the patient, and professional licensing restrictions.

1. Ayello EA, Cuddigan J. Debridement: controlling the necrotic/cellular burden. *Adv Skin Wound Care* 2004 March; 17(2): 66–75. [LIT REV]
2. Gottrup, F. Wound debridement. In: *The Oxford European Wound Healing Course Handbook*. Positif Press, Oxford, 2002: 116–120. [SURV]
3. Mosher BA, Cuddigan J, Thomas DR, Boudreau DM. Outcomes of 4 methods of debridement using a decision analysis methodology. *Adv Wound Care* 1999 Mar; 12(2): 81–8. [TECH]

4. Saap LJ, Falanga V. Debridement performance index and its correlation with complete closure of diabetic foot ulcers. *Wound Rep Regen* 2002 Nov–Dec; 10(6): 354–9. [RCT]
5. Sibbald RG, Williamson D, et al. Preparing the wound bed—debridement, bacterial balance, and moisture balance. *Ostomy/Wound Manage* 2000; 46(11): 14–35. [LIT REV]
6. Sieggreen MY, Maklebust J. Debridement: choices and challenges. *Adv Wound Care* 1997 Mar–Apr; 10(2): 32–7. [LIT REV]
7. Steed DL. Debridement. *Am J Surg* 2004 May; 187(5A): 71S–74S. [LIT REV]

Surgical/Sharp Debridement: involves the use of instruments (scissors, scalpels, forceps) or laser to remove necrotic tissue from the wound. Debridement of large amounts of necrotic tissue should be performed in the operating room. Surgical debridement is indicated when the goal is to achieve fast and effective removal of large amounts of necrotic tissue. Surgical debridement is contraindicated if there is lack of expertise in this method, inadequate vascular supply to the wound, and absence of systemic antibacterial coverage in systemic sepsis. Relative contraindication is bleeding disorders or anticoagulation therapy.

Evidence:

1. Sorensen JL, Jorgensen B, Gottrup F. Surgical treatment of pressure ulcers. *Am J Surg* 2004 Jul; 188(1A Suppl.): 42–51. [LIT REV]
2. Steed DL, Donohue D, Webster MW, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. Diabetic Ulcer Study Group. *J Am Coll Surg* 1996 Jul; 183(1): 61–4. [RCT]

Mechanical Debridement: physically removes necrotic tissue with wet-to-dry dressings, wound irrigation, and whirlpool techniques. Wet-to-dry dressing may induce mechanical separation of eschar but can be painful and if dry, may damage viable newly formed tissue. High- or low-pressure streams or pulsed lavage may be quite effective in removing loose necrotic tissue, provided the pressure does not cause trauma to the wound bed. Effective ulcer irrigation pressures range from 4 to 15 psi of pressure. A 30-ml syringe filled with saline can be used to flush a wound through an 18-gauge catheter. Irrigation pressures below 4 psi may not be effective to cleanse the wound and pressures greater than 15 psi may cause trauma and drive the bacteria into the tissue. Whirlpools may be used initially to loosen and remove debris, bacteria, exudates, and necrotic tissue. Prolonged use and periods of wetness may macerate the tissue or may be associated with bacterial contamination.

Evidence:

1. Capasso VA, Munroe BH. The cost and efficacy of two wound treatments. *AORN J* 2003 May; 77(5): 984–92, 995–7, 1000–4. [RETRO S]
2. Hamer MI, Robson MC, Krizek TJ, et al. Quantitative bacterial analysis of comparative wound irrigations. *Ann Surg* 1975; 181: 819–22. [EXP]
3. Mulder GD. Cost-effective managed care: gel versus wet-to-dry for debridement. *Ostomy Wound Manage*. 1995 Mar; 41(2): 68–70, 72, 74 passim. [RCT]
4. Palmier S, Trial C. Use of high-pressure waterjets in wound debridement. In: Teot L, Banwell PE, Ziegler UE, editors. *Surgery in Wounds*. Berlin: Springer 2004: 72–6. [SURV]
5. Xakellis GC, Chrischilles EA. Hydrocolloid versus saline-gauze dressings in treating pressure ulcers: a cost-effectiveness analysis. *Arch Phys Med Rehabil* 1992 May; 73(5): 463–9. [RCT]

Enzymatic Debridement: is achieved by topical application of exogenous enzymes to the wound surface to remove necrotic tissue.

Evidence:

1. Alvarez OM, Fernandez-Obregon A, Rogers RS, et al. A prospective, randomized, comparative study of collagenase and papain-urea for pressure ulcer debridement. *Wounds* 2002; 14: 293–30. [RCT]
2. Boxer AM, Gottesman N, Bernstein H, Mandl I. Debridement of dermal ulcers and decubiti with collagenase. *Geriatrics* 1969 Jul; 24(7): 75–86. [RCT]
3. Falanga V. Wound bed preparation and the role of enzymes: a case for multiple actions of the therapeutic agents. *Wounds* 2002; 14: 47–57. [LIT REV]
4. Rao DB, Sane PG, Georgiev EL. Collagenase in the treatment of dermal and decubitus ulcers. *J Am Geriatr Soc* 1975 Jan; 23(1): 22–30. [RCT]
5. König M, Vanscheidt W, Augustin M, Kapp H. Enzymatic versus autolytic debridement of chronic leg ulcers: a prospective randomised trial. *J Wound Care* 2005; 14(7): 320–3. [RCT]
6. Wright JB, Shi L. Accuzyme papain-urea debriding ointment: a historical review. *Wounds* 2003; 15 (Suppl.): 2S–12. [LIT REV]

Autolytic Debridement: is accomplished by moist interactive dressings. These dressings allow the natural wound fluid and its endogenous enzymes to soften and liquefy slough and promote granulation. The wound needs to be cleansed after debridement to remove the necrotic debris.

If tissue autolysis is not apparent in 1–2 weeks, another debridement method should be used. Autolytic debridement is not recommended for infected wounds or very deep wounds that require packing.

Evidence:

1. Alvarez OM, Mertz PM, Eaglstein WH. The effect of occlusive dressings on collagen synthesis and reepithelialization in superficial wounds. *J Surg Res* 1983; 35(2): 142–8. [EXP]
2. Barnett SE, Varley SJ. The effects of calcium alginate on wound healing. *Ann R Coll Surg Engl* 1987; 69: 153–5. [EXPT]
3. Barr JE, Day AL, Weaver VA, Taylor GM, Dombranski S, et al. Assessing clinical efficacy of a hydrocolloid/alginate dressing on full-thickness pressure ulcers. *Ostomy Wound Manage* 1995 Apr; 41(3): 28–30, 32, 34–6 passim. [CLIN S]

4. Kim YC, Shin JC, et al. Efficacy of hydrocolloid occlusive dressing technique in decubitus ulcer treatment: a comparative study. *Yonsei Med J* 1996 Jun; 37(3): 181–5. [RCT]

Guideline #4.5: Wounds should be cleansed initially and at each dressing change using a neutral, nonirritating, nontoxic solution. Routine wound cleansing should be accomplished with a minimum of chemical and/or mechanical trauma. (Level III)

Principle: Cleansing the wound removes loose impediments to wound healing. Clinical experience has shown that mild soap (non-perfumed, without added antibacterials, and at skin pH: 4.5–5.7) and water for cleansing, used regularly, is effective, safe, and cheap. Sterile saline or water is recommended. Tap water should only be used if the water source is reliably clean. Wound antiseptic agents, e.g., hydrogen peroxide, hypochlorite solution, acetic acid, chlorhexamide, providone/iodine, cetrimide, and others have antibacterial properties but are all toxic to healthy granulation tissue.

Evidence:

1. Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of current literature. *Ostomy Wound Manage* 1999 Jan; 45 (1A Suppl.): 80S–85S; quiz86S–87S. [LIT REV]
2. Rodeheaver GT. Wound cleansing, wound irrigation, wound disinfection. In: Krasner D, Kane D, editors. *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*, 2nd ed. Wayne, PA: Health Management Publications, Inc.; 1997: 97–108. [LIT REV]
3. Rodeheaver GT, Kurtz L, Kircher BJ, et al. Pluronic F-68: a promising new skin wound cleanser. *Ann Emerg Med* 1980; 9: 572–6. [EXP]

Guideline #4.6: Infection control should be achieved by reducing wound bacterial burden and achieving wound bacterial balance. (For detailed guidelines, see Infection.) (Level I)

Principle: Infection will cause wound-healing failure, often with progressive deterioration of the wound. Systemically administered antibiotics do not effectively decrease bacterial levels in granulating wounds. Other methods that may be suitable include enhancing host defense mechanisms, debridement, wound cleaning, and topical antimicrobials. For ulcers with 1×10^6 or higher CFU/gram of tissue or any tissue-level beta hemolytic streptococci following adequate debridement, decrease the bacterial level by a topical antimicrobial. Once in bacterial balance, i.e., 10^5 CFU or less/gram of tissue, and no beta hemolytic streptococci in the ulcer, discontinue the use of topical antimicrobial to minimize the possibility of emergence of resistance. In chronic wounds, the pathogen species may be more important than the number of bacteria. Obtain bone biopsy for culture and histology (gold standard) in case of suspected osteomyelitis. Treat confirmed debrided osteomyelitis with flap containing muscle or fascia and culture-determined antibiotics.

Evidence:

1. Bendy RH, Nuccio PA, Wolfe E, et al. Relationship of quantitative wound bacterial counts to healing of decubiti. Effect of gentamicin. *Antimicrob Agent Chemo Ther* 1964; 4: 147–55. [RCT]
2. Bowler PG, Duerden BL, Armstrong DG. Wound microbiology and associated approaches to wound management. *Clin Microbiol Rev* 2001; 14: 244–69. [LIT REV]
3. Kucan JO, Robson MC, Heggers JP, et al. Comparison of silver sulfa-diazine, povidone-iodine, and physiologic saline in the treatment of chronic pressure ulcers. *J Am Geriatr Soc* 1981; 24: 232–5. [RCT]
4. Nystrom PO. The microbiological swab sampler—a quantitative experimental investigation. *Acta Pathol Microbiol Scand* 1978; 86B: 361–7. [TECH]
5. Robson MC. Wound infection: a failure of wound healing caused by an imbalance of bacteria. *Surg Clin North Am* 1997; 77: 637–50. [LIT REV]
6. Robson MC, Edstrom LE, Krizek TJ, et al. The efficacy of systemic antibiotics in the treatment of granulating wounds. *J Surg Res* 1974; 16: 299–306. [EXP]
7. Robson MC, Mannari RJ, Smith PD, et al. Maintenance of wound bacterial balance. *Am J Surg* 1999; 178: 399–402. [RCT]
8. Robson MC, Stenberg BD, Heggers, JP. Wound healing alterations caused by infection. *Clin Plast Surg* 1990; 17: 485–92. [LIT REV]
9. Sapico FL, Ginnas VJ, Thornhill-Joynes M, et al. Quantitative microbiology of pressure sores in different stages of healing. *Diagn Microbiol Infect Dis* 1986; 5: 31–8. [CLIN S]
10. Stengel D, Bauwens K, Sehoul J, et al. Systematic review and meta-analysis of antibiotic therapy for bone and joint infections. *Lancet Infect Dis* 2001; 1: 175–88. [STAT]
11. Stotts NA, Hunt TK. Pressure ulcers. Managing bacterial colonization and infection. *Clin Geriatr Med* 1997 Aug; 13(3): 565–73. [LIT REV]
12. Thornhill-Joynes M, Gonzales F, Stewart CA, et al. Osteomyelitis associated with pressure ulcers. *Arch Phys Med Rehabil* 1986; 67: 314–18. [RETRO S]
13. Volenec FJ, Clark GM, Manni MM, et al. Burn wound biopsy bacterial quantification: a statistical analysis. *Am J Surg* 1979; 138: 695–7. [STAT]

Guideline #4.8: Achieve local moisture balance by management of exudate. (Level I)

Principle: Local moisture balance is necessary to facilitate granulation and reepithelialization of the ulcer. A moist wound environment accelerates wound healing with more rapid epithelialization. Many dressings now combine wound bed preparation, i.e., debridement and/or antimicrobial activity, with moisture control. Moist wound dressings should keep the ulcer bed continuously moist and at the same time control the exudate to prevent desiccation of the ulcer bed and maceration of the peri-ulcer skin. Use clean, dry dressings for 8–24 hours after sharp debridement associated with bleeding; then reinstitute moist dressings. Clean dressings may also be used in conjunction with mechanical or enzymatic debridement techniques.

(For detailed guidelines, see Dressings.)

Evidence:

1. Alm A, Hornmark AM, Fall PA, et al. Care of pressure sores: a controlled study of the use of a hydrocolloid dressing compared with wet saline gauze compresses. *Acta Derm Venereol* (Stockholm) 1989; 149 (Suppl.): 1–10. [RCT]
2. Agren MS, Karlsmark T, Hansen JB, Rygaard J. Occlusion versus air exposure on full-thickness biopsy wounds. *J Wound Care* 2001; 10(8): 301–4. [RCT]
3. Breuing K, Eriksson E, Liu P, Miller DR. Healing of partial thickness porcine skin wounds in a liquid environment. *J Surg Res* 1992; 52: 50–8. [EXP]
4. Colwell JC, Foreman MD, Trotter JP. A comparison of the efficacy and cost-effectiveness of two methods of managing pressure ulcers. *Decubitus* 1993; 6: 28–36. [RCT]
5. Gorse GJ, Messner RL. Improved pressure sore healing with hydrocolloid dressings. *Arch Dermatol* 1987; 123: 766–71. [RCT]
6. Mulder G, Altman M, Seeley J, et al. Prospective randomized study of the efficacy of hydrogel, hydrocolloid, and saline moistened dressings on the management of pressure ulcers. *Wound Rep Reg* 1993; 1: 213–8. [RCT]
7. Svensjo T, Pomahac B, Yao F, Slama J, Eriksson E. Accelerated healing of full-thickness skin wounds in a wet environment. *Plast Reconstr Surg* 2000; 106: 602–12. [EXP]
8. Vranckx JJ, Slama J, Preuss S, et al. Wet wound healing. *Plast Reconstr Surg* 2002; 110: 1680–7. [CLIN S]
9. Winter GD, Scales, JT. Effect of air drying and dressings on the surface of a wound. *Nature* 1963; 197: 91. [EXP]
10. Neill K, Conforti C, Kedas A, et al. Pressure sore response to a new hydrocolloid. *Wounds* 1989; 1(3): 173–85. [RCT]

Guideline #4.9: There should be an ongoing and consistent documentation of wound history, recurrence, and characteristics (location, staging, size, base, exudates, infection condition of surrounding skin, and pain). The rate of wound healing should be evaluated to determine whether treatment is optimal. (Level III)

Principle: Ongoing evaluations of wound bed preparation are necessary because if the ulcer is not healing at the expected rate, interventions for wound bed preparation need to be reassessed. The longer the duration of the ulcer, the more difficult it is to heal. If an ulcer is recurrent, patient education or issues of prevention and long-term maintenance need to be reassessed.

Evidence:

1. Brown GS. Reporting outcomes for stage IV pressure ulcer healing: a proposal. *Adv Skin Wound Care* 2000 Nov–Dec; 13(6): 277–83. [RETRO S]
2. Lazarus GS, Cooper DM, Knighton DR, Margolis DJ, Pecoraro RE, Rodeheaver G, Robson MC. Definitions and guidelines for assessment of wounds and evaluation of healing. *Arch Dermatol* 1994 Apr; 130(4): 489–93. [STAT]
3. Krasner D. Wound Healing Scale, version 1.0: a proposal. *Adv Wound Care* 1997 Sep; 10(5): 82–5. [LIT REV]
4. Saap LJ, Falanga V. Debridement performance index and its correlation with complete closure of diabetic foot ulcers. *Wound Rep Reg* 2002 Nov–Dec; 10(6): 354–9. [RCT]

5. DRESSINGS

Preamble: There is a plethora of choices for topical treatment of pressure ulcers. Many dressings now combine wound bed preparation, i.e., debridement and/or antimicrobial activity, with moisture control. Guidelines assist the clinician in making decisions regarding the value and best use of these advanced wound care products.

Guideline #5.1: Use a dressing that will maintain a moist wound-healing environment. (Level I)

Principle: A moist wound environment physiologically favors migration and matrix formation while accelerating healing of wounds by promoting autolytic debridement. Moist wound healing also reduces wound pain.

Evidence:

1. Breuing K, Eriksson E, Liu P, Miller DR. Healing of partial thickness porcine skin wounds in a liquid environment. *J Surg Res* 1992; 52: 50–8. [EXP]
2. Gorse GJ, Messner RL. Improved pressure sore healing with hydrocolloid dressings. *Arch Dermatol* 1987; 123: 766–71. [RCT]
3. Svensjo T, Pomahac B, Yao F, Slama J, Eriksson E. Accelerated healing of full-thickness skin wounds in a wet environment. *Plast Reconstr Surg* 2000; 106: 602–12. [EXP]
4. Thomas DR, Goode PS, LaMaster K, Tennyson T. Acemannan hydrogel dressing versus saline dressing for pressure ulcers. A randomized, controlled trial. *Adv Wound Care* 1998 Oct; 11(6): 273–6. [RCT]
5. Vranckx JJ, Slama J, Preuss S, et al. Wet wound healing. *Plast Reconstr Surg* 2002; 110: 1680–7. [CLIN S]
6. Winter GD, Scales, JT. Effect of air drying and dressings on the surface of a wound. *Nature* 1963; 197: 91. [EXP]
7. Ovington, LG. Hanging wet-to-dry dressings out to dry. *Home Healthcare Nurse* 2001; 19: 477–84. [LIT REV]
8. Kerstein, MD, Gemmen, E, van Rijswijk, L, Lyder, CH, Golden, K, Harrington, C. Cost and cost effectiveness of venous and pressure ulcer protocols of care. *Dis Manage Health Outcomes* 2001; 9: 651–6. [COST ANAL]

Guideline #5.2: Use clinical judgment to select a moist wound dressing. (Level I)

Principle: Results from existing studies have not demonstrated any specific moisture retentive topical therapy to

be superior in terms of healing rate. Wet-to-dry dressings are not continuously moist and are an inappropriate wound-dressing selection.

Evidence:

1. Alm A, Hornmark AM, Fall PA, et al. Care of pressure sores: a controlled study of the use of a hydrocolloid dressing compared with wet saline gauze compresses. *Acta Derm Venereol* (Stockholm) 1989; 149 (Suppl.): 1–10. [RCT]
2. Blair SD, Jarvis P, Salmon M, McCollum C. Clinical trial of calcium alginate haemostatic swabs. *Br J Surg* 1990; 77: 568–70. [RCT]
3. Bouza C, Saz Z, Munoz A, Amate JM. Efficacy of advanced dressings in the treatment of pressure ulcers: a systematic review 2005; 14: 193–9. [STAT]
4. Bradley M, Cullum N, Nelson EA, et al. Systematic reviews of wound care management: (2) dressings and topical agents used in the healing of chronic wounds. *Health Technol Assess* 1999; 3: 1–35. [STAT]
5. Colwell JC, Foreman MD, Trotter JP. A comparison of the efficacy and cost-effectiveness of two methods of managing pressure ulcers. *Decubitus* 1993; 6: 28–36. [RCT]
6. Geronemus RG, Robins P. The effect of two new dressings on epidermal wound healing. *J Derm Surg Oncol* 1982; 8: 850–2. [EXP]
7. Graumlich JF, Blough LS, McLaughlin RG, Milbrandt JC, Calderon CL, Agha SA, Scheibel LW. Healing pressure ulcers with collagen or hydrocolloid: a randomized, controlled trial. *J Am Geriatr Soc* 2003 Feb; 51(2): 147–54. [RCT]
8. Kim YC, Shin JC, Park CI, et al. Efficacy of hydrocolloid occlusive dressing technique in decubitus ulcer treatment: a comparative study. *Yonsei Med J* 1996; 37: 181–5. [RCT]
9. Mulder G, Altman M, Seeley J, et al. Prospective randomized study of the efficacy of hydrogel, hydrocolloid, and saline moistened dressings on the management of pressure ulcers. *Wound Rep Reg* 1993; 1: 213–8. [RCT]
10. Neill K, Conforti C, Kedas A, et al. Pressure sore response to a new hydrocolloid. *Wounds* 1989; 1(3): 173–85. [RCT]
11. Sayag J, Meaume S, Bohbot S. Healing properties of calcium alginate dressings. *J Wound Care* 1996; 5: 357–62. [RCT]
12. Thomas DR, Goode PS, LaMaster K, Tennyson T. Acemannan hydrogel dressing versus saline dressing for pressure ulcers. A randomized, controlled trial. *Adv Wound Care* 1998; 11: 273–6. [RCT]
13. Xakellis GC, Chrischilles EA. Hydrocolloid versus saline-gauze dressings in treating pressure ulcers: a cost-effectiveness analysis. *Arch Phys Med Rehabil* 1992; 73: 463–9. [RCT]

Guideline #5.3: Select a dressing that will manage the wound exudate and protect the peri-ulcer skin. (Level I)

Principle: Peri-wound maceration and continuous contact with wound exudate can enlarge the wound and impede healing.

Evidence:

1. Bucalo B, Eaglstein WH, Falanga V. Inhibition of cell proliferation by chronic wound fluid. *Wound Rep Reg* 1993; 1: 181–6. [EXP]
2. Chapuis A, Dollfus P. The use of a calcium alginate dressing in the management of decubitus ulcers in patients with spinal cord lesions. *Paraplegia* 1990; 28: 269–71. [CLIN S]
3. Ladwig GP, Robson MC, Liu R, Kuhn MA, Muir DF, Schultz GS. Ratios of activated matrix metalloproteinase-9 to tissue inhibitor of matrix metalloproteinase-1 in wound fluids are inversely correlated with healing of pressure ulcers. *Wound Rep Reg* 2002; 10: 26–37. [CLIN S]
4. Maume S, Van De Looverbosch D, Heyman H, Romanelli M, Ciangherotti A, Charpin S. A study to compare a new self-adherent soft silicone dressing with a self-adherent polymer dressing in stage II pressure ulcers. *Ostomy Wound Manage* 2003; 49: 44–51. [RCT]
5. Sayag J, Meaume S, Bohbot S. Healing properties of calcium alginate dressings. *J Wound Care* 1996; 5: 357–62. [RCT]
6. Trengrove NJ, Stacey MC, MacAuley S, Bennett N, Gibson J, Burslem F, Murphy G, Schultz G. Analysis of the acute and chronic wound environments: the role of proteases and their inhibitors. *Wound Rep Reg* 1999; 7: 442–52. [EXP]
7. Xakellis GC, Chrischilles EA. Hydrocolloid versus saline gauze dressings in treating pressure ulcers: a cost-effectiveness analysis. *Arch Phys Med Rehabil* 1992; 73: 463–9. [RCT]
8. Yager DR, Zhang LY, Liang HX, Diegelmann RF, Cohen IK. Wound fluids from human pressure ulcers contain elevated matrix metalloproteinase levels and activity compared to surgical wound fluids. *J Invest Derm* 1996; 107(5): 743–8. [EXP]

Guideline #5.4: Select a dressing that remains in place and minimizes shear, friction, skin irritation, and additional pressure. (Level II)

Principles: Wound location, peri-wound skin quality, incontinence of urine or stool, and patient activity can all affect the choice of dressing. Some dressings have been designed to be self-adherent, some are designed to fill a cavity. Additional tissue damage may result if the dressing causes increased pressure on the wound or damages adjacent tissue.

Evidence:

1. Day A, Dombranski S, Farkas C, et al. Managing sacral pressure ulcers with hydrocolloid dressings: results of a controlled clinical study. *Ostomy Wound Manage* 1995; 41: 52–65. [RCT]
2. Dobrzanski S, Kelly CM, Gray JI, et al. Granuflex dressings in treatment of full thickness pressure sores. *Prof Nurse* 1990; 5: 594–9. [RCT]
3. Sasseville D, Tennstedt D, Lachapelle JM. Allergic contact dermatitis from hydrocolloid dressings. *Am J Contact Dermat* 1997; 8: 236–8. [CLIN S]
4. Gallenkemper G, Rabe E, Bauer R. Contact sensitization in chronic venous insufficiency: modern

wound dressings. *Contac Dermatitis* 1998; 38: 274–8. [CLIN S]

Guideline #5.5: Select a dressing that is cost effective. (Level I)

Principles: Because the initial cost of moist gauze is lower than advanced wound care products, there is a perception that moist gauze is more cost effective. When determining cost efficacy, it is important to take into consideration health care provider time, patient care goals and resources, ease of use and healing rate, as well as the unit cost of the dressing.

Evidence:

1. Bolton LL, van Rijswijk L, Shaffer FA. Quality wound care equals cost-effective wound care: a clinical model. *Adv Wound Care* 1997; 10: 33–8. [LIT REV]
2. Chang KW, Alsagoff S, Ong KT, SIm PH. Pressure ulcers randomized controlled trial comparing hydrocolloid and saline gauze dressings. *Med J Malaysia* 1998; 53: 428–31. [RCT]
3. Kim YC, Shin JC, Park CI, et al. Efficacy of hydrocolloid occlusive dressing technique in decubitus ulcer treatment: a comparative study. *Yonsei Med J* 1996; 37: 181–5. [RCT]
4. Severn MD. Pressure ulcer management in home health care: efficacy and cost-effectiveness of moisture vapor permeable dressing. *Arch Phys Med Rehabil* 1986; 67: 726–9. [RCT]
5. Xakellis GC, Chrischilles EA. Hydrocolloid versus saline gauze dressings in treating pressure ulcers: a cost-effectiveness analysis. *Arch Phys med Rehabil* 1992; 73: 463–9. [RCT]
6. Ovington LG. Hanging wet-to-dry dressings out to dry. *Home Healthcare Nurse* 2001; 19: 477–84. [LIT REV]
7. Kerstein MD, Gemmen E, van Rijswijk L, Lyder CH, Golden K, Harrington C. Cost and cost effectiveness of venous and pressure ulcer protocols of care. *Dis Manage Health Outcomes* 2001; 9: 631–6. [COST ANAL]

6. SURGERY

Preamble: Surgical treatment of pressure sores is a final invasive choice for wounds refractory to less aggressive care or for use when rapid closure is indicated. Peri-operative morbidity and greater risk of complications are inherent to the use of surgical options. Surgical procedures can be divided into those that prepare the patient for successful healing, and those that provide definitive closure. Reports of randomized clinical trials for operative treatment of pressure ulcers are almost nonexistent in the literature. However, given the magnitude of these treatment options, guidelines are mandatory to address their appropriate use.

Guideline #6.1: Irregular wound extensions, forming sinuses or cavities, must be explored and unroofed and treated. (Level III)

Principle: Tissue not exposed to treatment agents or devices cannot be expected to respond to the regimen and proceed to healing.

Evidence:

1. Jones, NF, Wexler, MR. Delineation of the pressure sore burns using methylene blue and hydrogen peroxide. *Plast Reconstr Surg* 1981; 68: 798–9. [TECH]

Guideline #6.2: Necrotic tissue must be debrided. (Level I) See Guideline #4.4 in Wound Bed Preparation.

Principle: Nonviable tissue is detrimental to wound healing. Therefore, it should be debrided to allow the wound to proceed to closure. (See Wound Bed Preparation.)

Evidence:

1. Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: a systematic review. *Health Technol Assess* 1999; 3: 1–78. [STAT]

Guideline #6.3: Infected tissue must be treated by topical antimicrobials, systemic antibiotics, or surgical debridement. (Level I)
(See Guidelines #3.2 and #3.4 in Infection.)

Principle: Infected soft tissue or bone will prevent wound healing, whether it is spontaneous or with the aid of surgical intervention. Only tissue with a low bacterial count ($\leq 10^5$/gm tissue) and with no β-hemolytic streptococcus will proceed to closure.

Evidence: See Guidelines #3.2, #3.3, #3.4, #3.5, #3.6, and #3.7 in Infection.

Guideline #6.4: Underlying bony prominence and fibrotic bursa cavities should be removed. (Level II)
Principle: Soft tissue compression between the skeleton and support surfaces leads to pressure necrosis. Removal of prominences, without excessive excision, alleviates pressure points.

Evidence:

1. Blocksma R, Kostrubala JG, Greeley PW. The surgical repair of decubitus ulcers. *Plast Reconstr Surg* 1947; 2: 403. [CLIN S]
2. Blocksma R, Kostrubala JG, Greeley PW. The surgical repair of decubitis ulcers in paraplegics: further observations. *Plast Reconstr Surg* 1949; 4: 123. [CLIN S]
3. Phillips LG, Robson MC. Pathobiology and treatment of pressure ulcerations. In: Jurkiewicz MJ, editor. *Plastic Surgery Principles and Practice*. St. Louis: Mosby, 1990; 1223. [LIT REV]
4. Vasconez LO, Schenider WJ, Jurkiewicz MJ. Pressure sores. *Curr Probl Surg* 1977; 14: 1–62. [LIT REV]
5. Consortium for Spinal Cord Medicine. *Pressure Ulcer Prevention and Treatment Guideline for Healthcare Professionals*. Paralyzed Veterans of America, 2000. [STAT]

Guideline #6.5: Bone excision must not be excessive. (Level III)

Principle: Extensive bone excision, especially at the ischial location, can expose deeper structures such as the urethra, or cause a shift of weight bearing, resulting in excessive pressure elsewhere.

Evidence:

1. Arregui J, Cannon B, Murray JE, et al. Long-term evaluation of ischiectomy in the treatment of pressure ulcers. 1965; 36: 583–90. [RETRO S]
2. Blocksma R, Kostrubala JG, Greeley PW. The surgical repair of decubitus ulcers. *Plast Reconstr Surg* 1947; 2: 403. [CLIN S]
3. Blocksma R, Kostrubala JG, Greeley PW. The surgical repair of decubitis ulcers in paraplegics: further observations. *Plast Reconstr Surg* 1949; 4: 123. [CLIN S]
4. Phillips LG, Robson MC. Pathobiology and treatment of pressure ulcerations. In: Jurkiewicz, MJ, editor. *Plastic Surgery Principles and Practice*. St. Louis: Mosby, 1990; 1223. [LIT REV]
5. Vasconez LO, Schenider WJ, Jurkiewicz MJ. Pressure sores. *Curr Probl Surg* 1977; 14: 1–62. [LIT REV]
6. Hackler RH, Zampari, TA. Urethral complications following ischiectomy in spinal cord injury patients: a urethral pressure study. *J Urol* 1987; 137: 253–5. [CLIN S]

Guideline #6.6: Fecal and urinary diversions are rarely needed to obtain a healed wound. (Level II)

Principle: Unless a fistulous track has developed, urinary or fecal contamination commonly occurs on the surface. Use of a bowel program or catheterization can divert urine and fecal material without the need for additional surgery.

Evidence:

1. Controller, Department of Medicine and Surgery: Mortality report in spinal cord injury; Reports and Statistics Service, Veterans Administration, Nov. 13, 1958. [STAT]
2. Conway H, Griffith BH. Plastic surgery for closure of decubitus ulcers in patients with paraplegia; based on experience with 1,000 cases. *Am J Surg* 1956; 91: 946–75. [CLIN S]
3. Conway H, et al. The plastic surgical closure of decubitus ulcers in patients with paraplegia. *Surg Gynecol Obstet* 1947; 85: 321. [CLIN S]
4. Phillips LG, Robson MC. Pathobiology and treatment of pressure ulcerations. In: Jurkiewicz, MJ, editor. *Plastic Surgery Principles and Practice*. St. Louis: Mosby, 1990; 1223. [LIT REV]
5. Vasconez LO, Schenider WJ, Jurkiewicz MJ. Pressure sores. *Curr Probl Surg* 1977; 14: 1 62. [LIT REV]

Guideline #6.7: Consider radical procedures such as amputation or hemicorporectomy only in the rare and extreme cases. (Level II)

Principle: Amputation, hemipelvectomy, or hemicorporectomy have significant morbidity and mortality, shift pressure points, and rarely address the underlying problem leading to extensive, recurrent pressure sores.

Evidence:

1. Phillips LG, Robson MC. Pathobiology and treatment of pressure ulcerations. In: Jurkiewicz, MJ, editor. *Plastic Surgery Principles and Practice*. St. Louis: Mosby, 1990; 1223. [LIT REV]
2. Royer J, Pickrell K, Georgiade N, et al. Total thigh flaps for extensive decubitus ulcers. A 16 year review of 41 total thigh flaps. *Plast Reconstr Surg* 1969; 54: 109–18. [RETRO S]
3. Vasconez LO, Schneider WJ, Jurkiewicz MJ. Pressure sores. *Curr Prob Surg* 1977; 14: 1 62. [LIT REV]
4. Chan JW, Virgo KS, Johnson FE. Hemipelvectomy for severe decubitus ulcers in patients with previous spinal cord injury. *Am J Surg* 2003; 185: 69–73. [STAT]

Guideline #6.8: A pressure sore should be closed surgically if it does not respond to wound care and there is no other contraindication to the surgical procedures. Exceptions may include the elderly or patients with a fatal illness, for whom palliative, local wound care is more appropriate. (Level II)

Principle: Wound closure decreases protein loss, fluid loss, the possibility of wound infection, and the later development of malignancy in the wound.

Evidence:

1. Dumurgier C, Pujol G, Chevalley J, et al. Pressure sore carcinoma: a late but fulminant complication of pressure sores in spinal cord injury patients: case reports. *Paraplegia* 1991; 29: 390–5. [CLIN S]
2. Evans GR, Lewis VL, Jr, Manson PN, et al. Hip joint communication with pressure sore: the refractory wound and the role of Girdlestone arthroplasty. *Plast Reconstr Surg* 1993; 91: 288–94. [CLIN S]
3. Grotting JC, Bunkis J, Vasconez LO. Pressure sore carcinoma. *Ann Plast Surg* 1987; 18: 527–32. [CLIN S]
4. Turba RM, Lewis VL, Green D. Pressure sore anemia: response to erythropoietin. *Arch Phys Med Rehabil* 1992; 73: 498–500. [CLIN S]
5. Consortium for Spinal Cord Medicine. *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Healthcare Professionals*. Paralyzed Veterans of America, 2000. [STAT]
6. Bergstrom N, Bennett MA, Carlson CE, et al. *Treatment of Pressure Ulcers. Clinical Practice Guideline # 15*. Rockville, MD: U.S. Dept. Health and Human Services, Agency for Healthcare Policy and Research, 1994. [STAT]

Guideline #6.9: Composite tissue closure leads to the best chance of sustained wound closure, although recurrence and recidivism are continuing problems. (Level II)

Principle: The most durable wound closure fills the ulcer with bulk and provides padding over the underlying structures with a tension-free closure.

Evidence:

1. Bruck JC, Buttemeyer R, Grabosch A, et al. More arguments in favor of myocutaneous flaps for the treatment of pelvic pressure sores. *Ann Plast Surg* 1991; 26: 85–8. [CLIN S]
2. Conway H, Griffith BH. Plastic surgery for closure of decubitus ulcers in patients with paraplegia; based on experience with 1,000 cases. *Am J Surg* 1956; 91: 946–75. [RETRO S]

3. Conway H, et al. The plastic surgical closure of decubitus ulcers in patients with paraplegia. *Surg Gynecol Obstet* 1947; 85: 321. [CLIN S]
4. Daniel RK, Faibisoff B. Muscle coverage of pressure points—the roll of myocutaneous flaps. *Ann Plast Surg* 1982; 8: 446–52. [CLIN S]
5. Disa JJ, Carlton JM, Goldberg NH. Efficacy of operative cure in pressure sore patients. *Plast Reconstr Surg* 1992; 89: 272–8. [CLIN S]
6. Evans GR, Dufresne CR, Manson PN. Surgical correction of pressure ulcers in an urban center: is it efficacious? *Adv Wound Care* 1994; 7: 40–6. [CLIN S]
7. Foster RD, Anthony JP, Mathes SJ, et al. Ischial pressure sore coverage: a rationale for flap selection. *Br J Plast Surg* 1997; 50: 374–9. [CLIN S]
8. Foster, RD, Anthony, JP, Mathes, SJ, et al. Flap selection as a determinant of success in pressure sore coverage. *Arch Surg* 1997; 132: 868–73. [CLIN S]
9. Rogers J, Wilson LF. Preventing recurrent tissue breakdowns after "pressure sore" closures. *Plast Reconstr Surg* 1975; 56: 419–22. [CLIN S]
10. Yamamoto Y, Ohura T, Shintomi Y, et al. Superiority of the fasciocutaneous flap in reconstruction of sacral pressure sores. *Ann Plast Surg* 1993; 30: 116–21. [CLIN S]
11. Consortium for Spinal Cord Medicine. *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Healthcare Professionals*. Paralyzed Veterans of America 2000. [STAT]
12. Bergstrom N, Bennett MA, Carlson CE, et al. *Treatment of Pressure Ulcers. Clinical Practice Guideline #15*. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Policy and Research, 1994. [STAT]

Guideline #6.10: Management to address muscle spasm and fixed contractures must occur preoperatively and continue at least until the wound is completely healed. (Level III)

Principle: Spasm may put traction on a wound to cause dehiscence of the suture line. Spasms and fixed contractures may limit postoperative positioning and leave the patient at risk for new pressure sore formation.

Evidence:

1. Stal S, Serure A, Donovan W, et al. The perioperative management of the patient with pressure sores. *Ann Plast Surg* 1983; 11: 347–56. [CLIN S]
2. Daniel RK, Hall EJ, MacLeod MK. Pressure sores—a reappraisal. *Ann Plast Surg* 1979; 3: 53–63. [RETRO S]
3. Conway H, Griffith BH. Plastic surgery for closure of decubitus ulcers in patients with paraplegia; based on experience with 1,000 cases. *Am J Surg* 1956; 91: 946–75. [RETRO S]
4. Conway H, et al. The plastic surgical closure of decubitus ulcers in patients with paraplegia. *Surg Gynecol Obstet* 1947; 85: 321. [CLIN S]
5. Vasconez LO, Schenider WJ, Jurkiewicz MJ. Pressure sores. *Curr Probl Surg* 1977; 14: 1–62. [LIT REV]
6. Haher JN, Haher TR, Devlin VJ, et al. The release of flexion contractures as a prerequisite for the treatment of pressure sores in multiple sclerosis: a report of ten cases. *Ann Plast Surg* 1983; 11: 246–9. [CLIN S]
7. Davis, R. Spasticity following spinal cord injury. *Clin Orthop Related Res* 1975; 112: 66–75. [LIT REV]

7. ADJUVANT AGENTS (TOPICAL, DEVICE, SYSTEMIC)

Preamble: Emerging evidence on adjuvant therapies suggests potential benefit for pressure ulcer healing. To date, there are insufficient studies demonstrating superiority over other more traditional wound treatments. Until further evidence of efficacy is established, consider the use of adjuvant therapy after evaluating individual patient and ulcer characteristics and when (1) healing fails to progress using conventional therapy and (2) under circumstances where the economic or physical burden of adjuvant therapy is consistent with patient goals and circumstances.

Topical Agents

Guideline # 7a.1: Consider the use of growth factor therapy for pressure ulcers that are not responsive to initial comprehensive therapy and/or before surgical repair. (Level II)

Principles: Growth factors are required for normal healing, and chronic wounds have shown growth factor deficiencies and imbalances. Achievement of some degree of ulcer closure, even if not complete, increases the ease of surgical closure. However, to date, no growth factor has received approval for pressure ulcer treatment.

Evidence:

1. Hirshberg J, Coleman J, Marchant B, Rees RS. TGF-beta3 in the treatment of pressure ulcers: a preliminary report. *Adv Skin Wound Care* 2001; 14(2): 91–5. [RCT]
2. Robson MC, Maggi SP, Smith PD, Wassermann RJ, Mosiello GC, Hill DP, Cooper DM. Ease of wound closure as an endpoint of treatment efficacy. *Wound Rep Reg* 1999; 7: 90–6. [RCT]
3. Kawai K, Suzuki S, Tabata Y, Nishimura Y. Accelerated wound healing through the incorporation of basic fibroblast growth factor-impregnated gelatin microspheres into artificial dermis using a pressure-induced decubitus ulcer model in genetically diabetic mice. *Br J Plast Surg* 2005 June 9; 58:1115–23. [EXP]
4. Kallianinen LK, Hirshberg J, Marchant B, Rees RS. Role of platelet-derived growth factor as an adjunct to surgery in the management of pressure ulcers. *Plast Reconstr Surg* 2000; 106: 1243–8. [RCT]
5. Ladwig GP, Robson MC, Liu R, Kuhn MA, Muir DF, Schultz GS. Ratios of activated matrix metalloproteinase-9 to tissue inhibitor of matrix metalloproteinase-1 in wound fluids are inversely correlated with healing of pressure ulcers. *Wound Rep Reg* 2002; 10: 26–37. [CLIN S]
6. Landi F, Aloe L, Russo A, Cesari M, Onder G, Bonini S, Carbonin PU, Bernabei R. Topical treatment of pressure ulcers with nerve growth factor: a randomized clinical trial. *Ann Intern Med* 2003 Oct 21; 139(8): 635–41. [RCT]

7. Mustoe TA, Cutler NR, Allman RM, Goode PS, Deuel TF, Prause JA, Bear M, Serdar CM, Pierce GF. A phase II study to evaluate recombinant platelet-derived growth factor-BB in the treatment of stage 3 and 4 pressure ulcers. *Arch Surg* 1994; 129: 213–9. [RCT]
8. Payne WG, Ochs DE, Meltzer DD, Hill DP, Mannari RJ, Robson LE, Robson MC. Long-term outcome study of growth factor-treated pressure ulcers. *Am J Surg* 2001 Jan; 181(1): 81–6. [RCT]
9. Pierce GF, Tarpley JE, Allman RM, Goode PS, Serdar CM, Morris B, Mustoe TA, Vande Berg J. Tissue repair processes in healing of chronic pressure ulcers treated with recombinant platelet-derived growth factor BB. *Am J Pathol* 1994; 145: 1399–410. [RCT]
10. Rees RS, Robson MC, Smiell JM, Perry BH. Becaplermin gel in the treatment of pressure ulcers: a phase II randomized, double-blind, placebo-controlled study. *Wound Rep Reg* 1999 May–Jun; 7(3): 141–7. [RCT]
11. Robson MC, Hill DP, Smith PD, Wang X, Meyer-Siegler K, Ko F, VandeBerg JS, Payne WG, Ochs D, Robson LE. Sequential cytokine therapy for pressure ulcers: clinical and mechanistic response. *Ann Surg* 2000 Apr; 231(4): 600–11. [RCT]
12. Brem H, Lyder C. Protocol for the successful treatment of pressure ulcers. *Am J Surg* 2004 (Suppl. to July 2004); 188: 9S–17S.

Devices

Guideline #7b.1: Consider using negative pressure wound therapy (NPWT) for stage III or IV pressure ulcers that fail to progress in healing with conventional therapy. (Level I)

Principle: NPWT applies negative pressure to the wound removing wound exudates and debris. Current evidence indicates that NPWT may support pressure ulcer healing by increasing wound perfusion and formation of granulation tissue and by reducing bacterial load.

Evidence:

1. Evans D, Land L. Topical negative pressure for treating chronic wounds. *The Cochrane Database of Systematic Reviews* 2001, Issue 1. Art. No.: CD001898. DOI: 10.1002/14651858.CD001898. [STAT]
2. Ford CN, Reinhard ER, Yeh D, Syrek D, De Las Morenas A, Bergman SB, Williams S, Hamori CA. Interim analysis of a prospective, randomized trial of vacuum-assisted closure versus the healthpoint system in the management of pressure ulcers. *Ann Plast Surg* 2002; 49: 55–61. [RCT]
3. Joseph E, Hamori CA, Bergman S, Roaf E, Swann NF, Anastasi GW. A prospective, randomized trial of vacuum-assisted closure versus standard therapy of chronic nonhealing wounds. *Wounds* 2000; 12: 60–7. [RCT]
4. Morykwas MJ, Argenta LC, Shelton-Brown EI, McGuirt W. Vacuum-assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg* 1997; 38: 553–62. [EXP]
5. Wanner MB, Schwarzl F, Strum B, Zaech GA, Pierer G. Vacuum-assisted wound closure for cheaper and more comfortable healing of pressure sores: a prospective study. *Scand J Plast Reconst Surg* 2003; 37: 28–33. [RCT]

Guideline #7b.2: Electrical stimulation may be useful in the treatment of pressure ulcers that have not healed with conventional therapy. (Level I)

Principle: Improvement in the healing of chronic wounds is reported in response to electrical stimulation. The most effective type of electrical stimulation treatment and specific types of chronic wounds that are most likely to respond to this therapy have not been determined.

Evidence:

1. Gardner SE, Frantz RA, Schmidt FL. Effect of electrical stimulation on chronic wound healing: a meta-analysis. *Wound Rep Reg* 1999; 7: 495–503. [STAT]
2. Kloth LC, Feedar JA. Acceleration of wound healing with high voltage, monophasic, pulsed current. *Phys Ther* 1988; 68: 503–8. [RCT]
3. Reger SI, Hyodo A, Negami S, Kambic HE, Sahgal V. Experimental wound healing with electrical stimulation. *Artif Organs* 1999; 23: 460–2. [EXP]
4. Wood JM, Evans PE III, Schallreuter KU, Jacobson WE, Sufit R, Newman J, White C, Jacobson M. A multicenter study on the use of pulsed low-intensity direct current for healing chronic stage II and stage III decubitus ulcers. *Arch Dermatol* 1993; 129: 999–1009. [RCT]

Systemic

Guideline # 7c.1: Hyperbaric oxygen therapy has *not* been shown to have a statistically significant effect on pressure ulcer healing. Further studies are needed to evaluate the efficacy of hyperbaric oxygen in pressure ulcers. (Level I)

Evidence:

1. Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. *The Cochrane Database of Systematic Reviews 2004*, Issue 1. Art. No.:CD004123.pub2. DOI:10.1002/14651858.CD004123.pub2. [STAT]
2. Roeckl-Wiedmann I, Bennett M, Kranke P. Systematic review of hyperbaric oxygen in the management of chronic wounds. *Br J Surg* 2005; 92: 24–32. [STAT]

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National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers: Quick Reference Guide. Emily Haesler (Ed.).Â The goal of this guideline is to provide evidence based recommendations for the prevention and treatment of pressure ulcers that can be used by health professionals throughout the world. The purpose of the prevention recommendations is to guide evidence based care to prevent the development of pressure ulcers and the purpose of the treatment focused recommendations is to provide evidence-based guidance on the most effective strategies to promote pressure ulcer healing. For optimal treatment of pressure ulcers there are 4 main concerns: 1. Underlying pathology of the pressure ulcer must be treated if. possible. 2. Pressure must be relieved or removed by appropriate measures to prevent further injury.Â However, weekly assessments provide an opportunity for the health care professional to detect early complications and the need for changes in the treatment plan. The treatment needs of a pressure ulcer change over time. Treatment strategies should be continuously re-evaluated based on the current status of the ulcer. 16 17. Wound infection. Treatment of Pressure Ulcers. Clinical Practice Guideline Number 14. Agency for Health Care Policy and Research, Public Health Service. Rockville, MD: US Department of Health and Human Services; 1994. AHCPR Publication No. 95-0642.: Frantz RA. Pressure ulcer costs in long term care.Â Deloach ED, DiBenedetto RJ, Womble L, Gilley JD. The treatment of osteomyelitis underlying pressure ulcers. Decubitus. 1992 Nov. 5(6):32-41. [Medline]. Yarkony GM. Pressure ulcers: a review.