Thomas T. Yoshikawa, Section Editor

Infected Pressure Ulcers in Elderly Individuals

Nigel J. Livesley and Anthony W. Chow

Division of Infectious Diseases, Department of Medicine, University of British Columbia and Vancouver Hospital & Health Sciences Centre, Vancouver, British Columbia, Canada

Pressure ulcers in elderly individuals can cause significant morbidity and mortality and are a major economic burden to the health care system. Prevention should be the ultimate objective of pressure ulcer care, and it requires an understanding of the pathophysiology leading to pressure ulcers and the means of reducing both intrinsic and extrinsic risk factors. Clinical manifestations are protean, and early recognition requires a low threshold of suspicion. Clinical examination often underestimates the degree of deep-tissue involvement, and its findings are inadequate for the detection of associated osteomyelitis. Microbiological data, if obtained from deep-tissue biopsy, are useful for directing antimicrobial therapy, but they are insufficient as the sole criterion for the diagnosis of infection. Imaging studies, such as computed tomography and magnetic resonance imaging, are useful, but bone biopsy and histopathological evaluation remain the "gold standard" for the detection of osteomyelitis. The goals of treatment of pressure ulcers should be resolution of infection, promotion of wound healing, and establishment of effective infection control.

Pressure ulcers have important consequences both for patients and for the health care system. They can lead to severe or intolerable pain, are prone to infection, and are associated with high mortality rates [1]. They also inflict a considerable economical burden on the health care system. In a 1996 study, the incremental cost per pressure ulcer (in US dollars) was $2731 [2], and this cost was dramatically higher ($59,000) if the pressure ulcer was associated with osteomyelitis [3]. In The Netherlands, treatment of pressure ulcers is estimated to account for >1% of the total health budget [4].

EPIDEMIOLOGY AND RISK FACTORS

Pressure ulcers are areas of necrosis caused by compression between bony prominences and external surfaces. The damage may be relatively minor, or it may lead to massive destruction of deeper tissues, which can cause significant morbidity and mortality. The incidence and prevalence of pressure ulcers depends on the definition of pressure ulcers used and the patient population studied. The National Pressure Ulcer Advisory Panel has classified pressure ulcers according to 4 stages [5]:

Stage I: Nonblanchable erythema of intact skin.

Stage II: Partial-thickness skin loss involving the epidermis or dermis; lesions may present as an abrasion, blister, or superficial ulcer.

Stage III: Full-thickness skin loss that may extend to, but not through, the fascia; the ulcer may be undermined.

Stage IV: Full-thickness skin loss involving deeper structures, such as muscle, bone, or joint structures.

Among nursing home residents, the prevalence of pressure ulcers classified as stage II or higher is 1.2%–11.3% [1]. In one longitudinal study, 17% of persons admitted to nursing homes had pressure ulcers at the time of admission [6]. Among those persons who did not have a pressure ulcer at the time of admission, the risk of a pressure ulcer developing after admission was 13% in the first year after admission and 21% by the second year [6]. On the other hand, a Canadian study reported an incidence of only ~3% during a 2-year period [7]. In a study of 74 long-term care facilities conducted by the Department of Veterans Affairs, the incidence of new pressure ulcers differed widely from facility to facility and ranged from 0% to 10.9% during a 6-month period [8]. The case-mix of the facilities was found to be an important variable when the pressure ulcer incidences at different facilities were compared. Among patients

Received 30 May 2002; accepted 16 July 2002; electronically published 4 November 2002.

Reprints or correspondence: Dr. Anthony W. Chow, Div. of Infectious Diseases, Vancouver Hospital & Health Sciences Centre, 2733 Heather St., Vancouver, British Columbia, Canada V5Z 3J5 (tonychow@interchange.ubc.ca).

Clinical Infectious Diseases 2002; 35:1390–6

1058-4838/2002/3511-0014$15.00

admitted to acute care hospitals without pressure ulcers, 11.2% of patients aged 70–79 years and 34% of patients aged >90 years eventually developed pressure ulcers [9]. The onset of pressure ulcer development occurred within a median of 9 days after admission to the hospital.

Risk factors for the development of pressure ulcers are either intrinsic or extrinsic. Limited mobility and poor nutrition are the strongest intrinsic predictors of pressure ulcer formation. Incontinence, increased age, diabetes mellitus, stroke, white race, skin abnormalities, and male sex have also been implicated by multivariate analysis in some studies [1]. Extrinsic factors include pressure, friction, shear stress, and moisture; of these, the most important is pressure.

An inverse relationship between the degree of external pressure and the time needed for tissue damage to occur has been demonstrated in several animal models (figure 1) [10]. A patient lying on a hospital mattress can generate pressures of 45–75 mm Hg over such bony prominences as the sacrum, greater trochanters, and heels, where pressure ulcers commonly form (figure 2). If this pressure is sustained for 3–4 h, capillary perfusion pressure within the deep tissues (estimated to be 20–30 mm Hg) is exceeded, and pressure ulcers may result [11]. It is important to recognize that pressure is highest at the muscle/bone interface, and that fat and muscle are more susceptible to pressure-related damage than is skin. Thus, the appearance of the visible skin lesion often results in an underestimation of the degree of deep-tissue involvement (figure 3).

Friction and shear stress can occur when a patient is dragged across a surface or is positioned with the head of the bed in a raised position. Friction can damage superficial skin, and shear stress can crimp the deeper vessels, leading to increased ischemia. Ulcers produced by shear stress can have extensive deep-tissue necrosis and can be much worse than external inspection may suggest. Moisture, such as that resulting from incontinence, can increase the risk of a pressure ulcer developing by 5-fold. It can also serve as a source of bacterial contamination.

INFECTED PRESSURE ULCERS

The epidemiology of infection in pressure ulcers has not been well described. In one prospective study of 16 patients with pressure ulcers who were followed for 2184 days, the incidence of infection was 1.4 cases per 1000 patient-ulcer days [7]. A point prevalence study found that 6% of 532 nursing home residents received treatment for infected pressure ulcers [12]. Because of the protean clinical manifestations, a low threshold of suspicion is essential for recognizing infection associated with pressure ulcers. The approach to management of potentially infected pressure ulcers requires clinical assessment and judgment, microbiological evaluation, imaging studies, and histopathological examination of deep-tissue biopsy specimens.

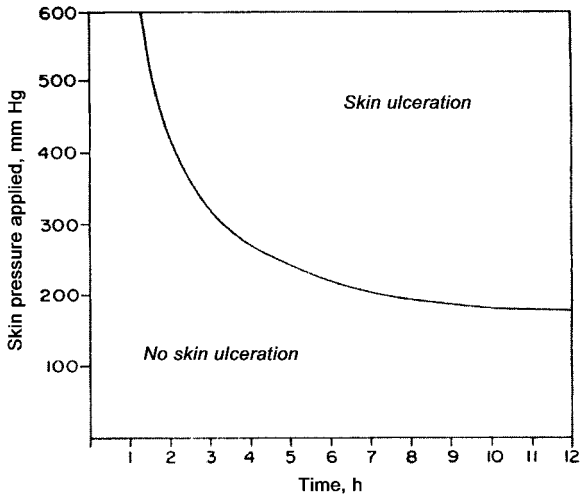


Figure 1. Inverse relationship of pressure to time in the production of pressure ulcers under experimental conditions in an animal model. From [11].

CLINICAL ASSESSMENT

Clinical assessment of pressure ulcers begins with identification of patients considered to be at risk and examination for early signs of pressure sore formation at the anatomical sites where such sores are most commonly encountered (figure 2). A thorough clinical examination is critical to the identification of pressure ulcers that may serve as an occult focus for infection. It is helpful to recognize the typical signs of soft-tissue involvement, such as warmth, erythema, local tenderness, purulent discharge, and presence of foul odor. However, because of associated comorbidities and advanced age, systemic signs, such as fever and leukocytosis, may be minimal or absent, and even local signs of inflammation may not be obvious [13].

Osteomyelitis associated with pressure ulcers is not easily diagnosed clinically. In a study of 36 patients who had suspected osteomyelitis associated with pressure ulcers, the accuracy of clinical examination was only 53%, with a sensitivity of 33% and a specificity of 60% [14]. Even the presence of a nonhealing wound or exposed bone did not always indicate underlying osteomyelitis.

The manifestations of infection in pressure ulcers can be extremely variable. Delayed wound healing may be the only sign of infection that can occur in the presence of $>10^6$ microorganisms per gram of tissue [13]. More serious manifestations of infection are osteomyelitis and bacteremia. Osteomyelitis can present as a poorly healing wound with or without systemic manifestations, such as fever, leukocytosis, and other signs of sepsis. In contrast, bacteremia due to infected pressure ulcers usually presents with signs of a systemic inflammatory response, including fever, chills, confusion, and hypotension,

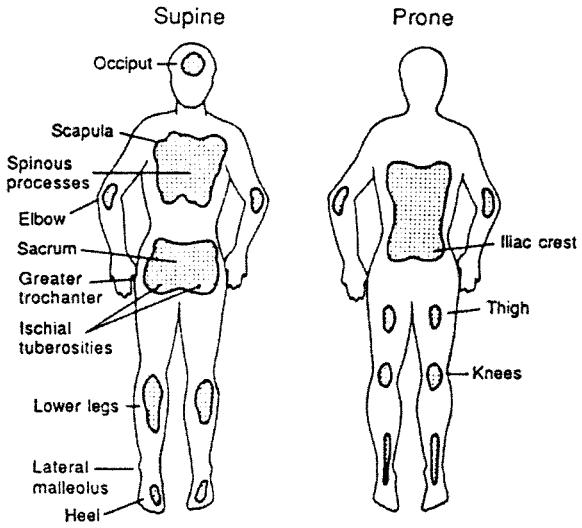


Figure 2. Common locations of pressure ulcers *(stippled areas)* in prone and supine positions. From [10].

and the mortality rate among patients with bacteremia due to infected pressure ulcers approaches 50% [15].

MICROBIOLOGICAL EVALUATION

In a study of 23 consecutively evaluated patients, bacteriological findings for clinically infected pressure ulcers were assessed by both aerobic and anaerobic culture techniques and specialized specimen transport [16]. An average of 4 isolates (3 aerobes and 1 anaerobe) was recovered. Bacteremia was extremely prevalent (79%) among these patients presenting with sepsis manifestations. Aerobes were more commonly isolated from the ulcers than were anaerobes, but twice as many anaerobes were recovered from cultures of blood samples obtained from 19 patients with bacteremia. Isolates recovered from the ulcer included *Proteus mirabilis, Escherichia coli,* enterococci, staphylococci, and *Pseudomonas* species. Anaerobic isolates included *Peptostreptococcus* species, *Bacteroides fragilis,* and *Clostridium perfringens.* The predominant bacteremic isolates were *B. fragilis, Peptostreptococcus* species, *P. mirabilis,* and *Staphylococcus aureus.* In 41% of cases, the bacteremia was polymicrobial. In a 5-year prospective study of bacteremia among residents of a long-term care facility, Muder et al. [17] reported that infected pressure ulcers were the second leading cause of bacteremia (the leading cause was urinary tract infection) and the most likely source of polymicrobial bacteremia. Thus, blood cultures clearly are very important in the initial microbiological assessment of all patients with suspected infection associated with pressure ulcers.

The challenge of microbiological evaluation is to distinguish between bacterial invasion and colonization. Blood cultures or cultures of deep-tissue biopsy specimens generally are more clinically significant than are cultures of superficial swab specimens or aspiration of the pressure ulcer. In one study, positive results were obtained for 97% of cultures of superficial swab specimens, compared with 43% of aspirations and 63% of cultures of deep-tissue biopsy specimens [18]. Concordance was poor between the different bacterial species identified by biopsy and those identified by aspiration and swab culture. It was concluded that swab culture led to too many false-positive results and that aspiration was too insensitive for general use. Another study compared deep-tissue biopsy with aspiration of draining pressure ulcers [19]. In this study, 1 mL of sterile saline was irrigated into the wound margin, and the area was massaged before aspiration. Compared with deep-tissue biopsy, this technique had a sensitivity of 93% and a specificity of 99% [19]. Similar species were identified by irrigation-aspiration and deep-tissue biopsy. However, aspirated samples of clinically noninfected ulcers have also been shown to contain bacteria in 30% of cases [7].

Culture results must also be interpreted with caution in cases of osteomyelitis. In a study of 36 patients with nonhealing pressure ulcers, only 17% of patients were found to have histopathological evidence of chronic osteomyelitis [14]. These patients had positive results of cultures of bone biopsy specimens, but so did 73% of patients without histological evidence of osteomyelitis.

On the basis of the aforementioned information, it can be concluded that (1) superficial swab cultures generally reflect colonization rather than infection and are not useful clinically, (2) needle aspirations are difficult to interpret and either should not be used or should be interpreted with caution, and (3) culture results by themselves, even results of bone culture or culture of other deep-tissue biopsy specimens, should not be used as the sole criterion for infection without clinical or histopathological evidence of infection.

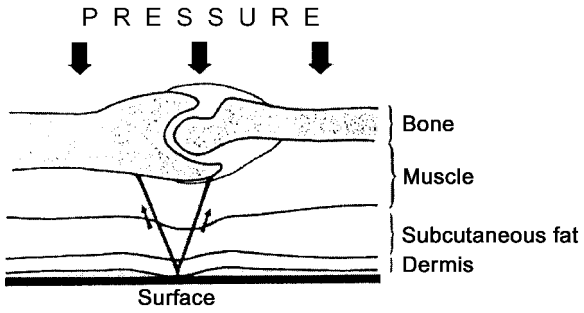


Figure 3. Pressure on any bony prominence is transmitted through the intervening tissues to the skin surface in a 3-dimensional cone-shaped gradient, with the greatest pressure occurring over a wide surface of bone and diminishing pressure occurring toward the skin surface. Thus, the appearance of the visible skin lesion often results in an underestimation of the degree of deep-tissue involvement. From [10].

IMAGING STUDIES

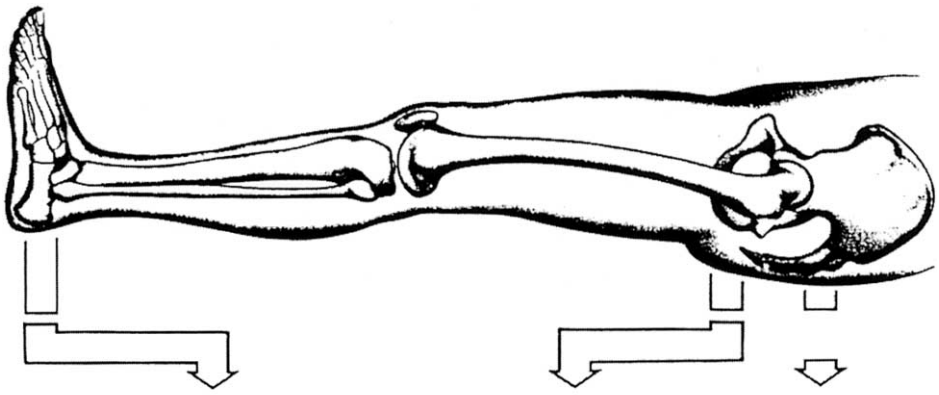
Imaging studies are useful in the evaluation of pressure ulcers for determination of the presence of osteomyelitis and for delineation of the extent of deep-tissue involvement.

Plain radiography. Plain radiographs have a limited role in the evaluation of pressure ulcers. Bony changes, such as periosteal reactive changes and heterotopic new bone formation usually associated with osteomyelitis, can also be present in noninfected pressure ulcers. Furthermore, lytic bony lesions rarely are seen in cases of osteomyelitis associated with pressure ulcers [14]. Sinograms can be useful to define the extent of the ulcer, but their value probably has diminished with the availability of CT and MRI.

CT and MRI. CT may be more useful for definition of the extent of deep soft-tissue damage associated with pressure ulcers. Although highly specific, CT has a relatively low sensitivity for the diagnosis of associated osteomyelitis (11%) [20]. MRI is more useful for the detection of osteomyelitis associated with pressure ulcers. In a study of 42 patients with pressure ulcers associated with spinal cord injuries, MRI demonstrated the presence of osteomyelitis with a sensitivity of 98% and a specificity of 89%, compared with the reference standard of bone biopsy (for 32 patients) and clinical follow-up (for 10 additional patients) [21]. One caveat is that the prevalence of osteomyelitis in the study was extremely high (47 of 59 studies), and this may have colored the interpretation of results. The performance of MRI for the detection of osteomyelitis associated with pressure ulcers in populations with a lower prevalence of disease or in older patients remains unclear and will require further study.

Radionuclide scintigraphy. Although 3-phase technetium-99m diphosphate bone scans and gallium scintigraphy are very useful in the diagnosis of hematogenous osteomyelitis, they have been shown to lack specificity for the detection of osteomyelitis caused by pressure ulcers [22]. Indium-labeled WBC scans may be more specific for patients with contiguous soft-tissue abnormality. In a study of 41 diabetic foot ulcers, indium-labeled WBC scanning had a specificity of 77%, compared with bone biopsy [23]. Unfortunately, this technique has not been adequately studied in patients with pressure ulcers.

In summary, clinical examination may indicate the presence of superficial soft-tissue infection, but it is inadequate for determination of the extent of deep-tissue involvement, and it is not useful in the diagnosis of associated osteomyelitis. Microbiological data, if obtained by deep-tissue biopsy, are useful for directing antimicrobial therapy after the diagnosis of infection. On its own, however, the presence of bacteria (even as detected by deep-tissue culture) is not sufficient for the diagnosis of



Air fluid bed	55–60 mm Hg (with pressure increased to >100 mm Hg)	20 mm Hg	15–20 mm Hg
Air mattress	>100 mm Hg	80 to >100 mm Hg	HOB 45°, >100 mm Hg HOB 30°, 70 mm Hg HOB 15°–50°, 50 mm Hg Flat, 40 mm Hg
Water bed	80 to >100 mm Hg	>100 mm Hg	HOB 45°, >100 mm Hg HOB 30°–50°, >70 mm Hg HOB 15°, 40 mm Hg Flat, 20-25 mm Hg

Figure 4. Degree of pressure reduction, in millimeters of mercury (mm Hg), achieved at different anatomical locations by use of pressure-relieving devices. HOB, head of bed. From [31].

Table 1. Antibiotic regimens for infected pressure ulcers.

Regimen	Recommended dose schedule
Monotherapy	
Cefoxitin	1–2 g iv or im every 6–8 h
Ceftizoxime	1–2 g iv every 8–12 h
Cefotetan	1–2 g iv or im every 12–24 h
Ticarcillin-clavulanate	3.1 g iv every 4–6 h
Piperacillin-tazobactam	2–4 g iv every 6–8 h
Imipenem	0.5–1 g iv every 6–8 h
Meropenem	0.5–1 g iv every 6–8 h
Gatifloxacin	400 mg iv or po daily
Combination therapy	
Clindamycin	450–600 mg iv every 6–8 h or 450 mg po q.i.d.
Plus ciprofloxacin	200–400 mg iv every 12 h or 500 mg po b.i.d.
Plus ofloxacin	200–400 mg iv every 12–24 h or 400 mg po b.i.d.
Metronidazole	500 mg iv every 6–8 h or 500 mg po t.i.d.
Plus ciprofloxacin	200–400 mg iv every 12 h or 500 mg po b.i.d.
Plus ofloxacin	200–400 mg iv every 12–24 h or 400 mg po b.i.d.
Treatment of infection due to MRSA	
Vancomycin	0.5 g iv every 6–8 h
Quinupristin/dalfopristin[a]	7.5 mg/kg iv every 8–12 h
Oxazolidinone[b]	600 mg iv every 12 h

NOTE. MRSA, methicillin-resistant *Staphylococcus aureus*.

[a] Synercid (Aventis Pharmaceutical Products).

[b] Linezolid.

infection. Of the radiographic investigations, MRI and CT may be of some use, but data are insufficient to recommend their general use in the nursing home or elderly population. Indium-labeled WBC scanning is more specific than is technetium or gallium scanning for the detection of underlying osteomyelitis; however, the usefulness of technetium or gallium scanning of elderly patients with infected pressure ulcers remains to be critically evaluated. Bone biopsy remains the "gold standard" and should be used in cases of uncertainty, particularly if prolonged antimicrobial therapy is being contemplated.

THERAPY

The goals of treatment of infected pressure ulcers are to resolve the infection and to aid in wound healing. Implementation of the appropriate therapy requires an understanding of the risk factors and pathophysiology that lead to pressure ulcer formation.

Treatment to aid in wound healing. Attention should be given to promoting healing of the pressure ulcer itself, in addition to treating infection. This requires ameliorating both intrinsic and extrinsic risk factors and providing meticulous local wound care. Although many intrinsic risk factors for the development of pressure ulcers are not amenable to intervention, some comorbid conditions associated with delayed wound healing, such as poor nutritional status, congestive heart failure, and diabetes mellitus, can be optimally controlled [24]. There is evidence that patients who have a high protein intake may experience improved wound healing, compared with patients who have an inadequate caloric intake of protein. Supplementation with enteral feeding, however, has not been demonstrated to improve wound healing or prevent pressure ulcers [25].

Reduction of extrinsic factors—in particular, pressure relief—is a cornerstone of therapy. Mechanical devices to lower pressure can be classified as either static or dynamic. Static devices, such as foam- or fluid-filled mattresses or supports, maintain constant pressure when the patient is not moving, but they disperse pressure over a greater area than do standard bed mattresses. These devices are appropriate for patients who can assume different positions without bearing weight on the ulcer and without compressing the support material [22]. For patients who cannot avoid bearing weight on the ulcer or who are not healing as expected, a dynamic pressure-relieving device, such as an air-fluid bed, may be a better choice. These devices change their support by alternating currents of air to redistribute pressure against the body, and they can achieve a greater degree of pressure reduction than can static devices (figure 4). Thus, most authorities recommend the use of dy-

Table 2. Infection-control recommendations from the Agency for Healthcare Research and Quality (formerly known as the Agency for Health Care Policy and Research) for residents of long-term care facilities who have pressure ulcers [22].

Reduce contamination of pressure ulcers
Sterile instruments should be used to debride pressure ulcers; the act of sharp debridement changes the physiology of the wound and makes it more susceptible to infection.
Clean dressings may be used instead of sterile dressings. Sterile dressings have not been shown to lead to less wound contamination, compared with clean dressings. It is unclear whether this recommendation also applies to newly debrided wounds and to patients with endemic resistant organisms.
Health care workers should use clean gloves for the care of each patient. When treating multiple ulcers on the same patient, the most contaminated ulcer should be treated last. Health care workers also should wash their hands in between contacts with different patients. It may be reasonable to use sterile gloves for contact with newly debrided pressure ulcers.
Ulcers should be protected from sources of contamination, such as feces. Wound healing is delayed in patients with fecal incontinence, although it may be difficult to protect the wound entirely.
Prevent the spread of pathogenic organisms from pressure ulcers by following body-substance precautions or an equivalent system when treating pressure ulcers
Wear gloves for contact with body fluids.
Change gloves and wash hands in between contacts with different patients and after any type of patient contact.
Wear additional barriers, such as gowns, masks, or goggles, when body fluids may come in contact with the clothing or skin.
Place soiled reusable items in securely sealed containers.
Place needles into designated sharps containers.

namic beds for the management of persons with stage III to stage IV pressure ulcers and for those that do not respond to standard therapy [22].

Local wound care is another fundamental component of pressure ulcer therapy. Debridement of necrotic tissue, appropriate dressing selection, and surgical repair are all important aspects of wound care, and these have been extensively reviewed elsewhere [26].

Treatment of infection. A combination of surgical and medical interventions may be required. Surgical debridement is necessary to remove necrotic tissue and drain abscesses. Systemic antimicrobial therapy should be used for patients with serious pressure ulcer infections, including those with spreading cellulitis, osteomyelitis, or bacteremia. Because of the high associated mortality, empiric antibiotics are appropriate if bacteremia or sepsis is suspected. Administration of topical antibiotics is not indicated.

The choice of antibiotics is based on the current understanding of the microbiology of infected pressure ulcers. Because such infections usually are polymicrobial, therapeutic regimens should be directed against both gram-positive and gram-negative facultative organisms as well as anaerobic organisms (table 1). Because of poor tissue perfusion in infected pressure ulcers, antibiotic therapy initially should be administered intravenously to patients with signs of systemic infection.

INFECTION-CONTROL MEASURES

Because most pressure ulcers occur in institutionalized patients, close attention to appropriate infection control is increasingly important. The goals of infection control are to reduce bacterial colonization and prevent infection; to minimize the spread of pathogenic organisms to other patients, staff, or the environment; and to prevent selection of resistant microorganisms. In 1994, the Agency for Healthcare Research and Quality included 5 infection-control recommendations in their treatment guidelines for institutionalized patients with pressure ulcers [22]. Of these recommendations, 4 are designed to reduce bacterial contamination of the wound, and 1 aims to reduce the spread of pathogens (table 2). Each of these recommendations was given a grade of C for strength of evidence indicating expert opinion rather than hard data from clinical trials. Clearly, more investigation in this area is needed [27].

PREVENTION

In an ideal world, the only subheading in a minireview of infected pressure ulcers would be "Prevention." The keys to prevention are identifying patients at risk, improving general health, minimizing external forces, and promoting educational programs about pressure ulcers to caregivers. In this regard,

the importance of quality nursing care in the prevention of pressure ulcers cannot be overemphasized. The incidence of pressure ulcers in a long-term care facility is often a direct measure of the quality of nursing care provided, particularly in the meticulous attention paid to careful positioning and frequent turning of the bedridden patient. Various risk prediction scales (such as the Braden or Norton scales) have been developed to aid in patient assessment and to identify patients for whom early treatment or prevention of pressure ulcers should be considered [28, 29]. Ability to minimize the 4 extrinsic risk factors (pressure, friction, shear stress, and moisture) is crucial to this preventive strategy. Pressure can be reduced through careful positioning and turning. Friction and shear stress can be avoided by not pulling patients over their beds and by paying attention to their positioning. Moisture usually is the result of incontinence; incontinence should be treated, if possible, or its effects should be reduced by the use of absorbent pads. There also is strong evidence that educational programs can lead to a reduction in the incidence of pressure ulcers [30]. A multitude of devices and different dressings and topical agents that have been proposed for the treatment or prevention of pressure ulcers. Unfortunately, well-designed clinical trials to evaluate and support the use of these modalities are extremely rare and clearly are warranted.

References

1. Allman RM. Pressure ulcer prevalence, incidence, risk factors, and impact. Clin Geriatr Med **1997**; 13:421–37.
2. Xakellis GC, Frantz R. The cost of healing pressure ulcers across multiple health care settings. Adv Wound Care **1996**; 9:18–22.
3. Hirshberg J, Rees RS, Marchant B, Dean S. Osteomyelitis related to pressure ulcers: the cost of neglect. Adv Skin Wound Care **2000**; 13: 25–9.
4. Severens JL, Habraken JM, Duivenvoorden S, Frederiks CM. The cost of illness of pressure ulcers in The Netherlands. Adv Skin Wound Care **2002**; 15:72–7.
5. Pressure ulcers prevalence, cost, risk assessment: consensus development conference statement. The National Pressure Ulcer Advisory Panel. Decubitus **1989**; 2:24–8.
6. Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. The epidemiology and natural history of pressure ulcers in elderly nursing home residents. JAMA **1990**; 264:2905–9.
7. Nicolle LE, Orr P, Duckworth H, et al. Prospective study of decubitus ulcers in two long term care facilities. Can J Infect Control **1994**; 9: 35–8.
8. Berlowitz DR, Wilking SVB. Pressure ulcers in the nursing home. In: Rubenstein L, Wieland D, eds. Improving care in the nursing home: comprehensive reviews of clinical research, vol. 4. Newbury Park, CA: Sage Publications, **1993**:102–30.
9. Perneger TV, Heliot C, Rae AC, Borst F, Gaspoz JM. Hospital-acquired pressure ulcers: risk factors and use of preventive devices. Arch Intern Med **1998**; 158:1940–5.
10. Reuler JB, Cooney TG. The pressure sore: pathophysiology and principles of management. Ann Intern Med **1981**; 94:661–6.
11. Woolsey RM, McGarry JD. The cause, prevention, and treatment of pressure sores. Neurol Clin **1991**; 9:797–808.
12. Garibaldi RA, Brodine S, Matsumiya S. Infections among patients in nursing homes: policies, prevalence, problems. N Engl J Med **1981**; 305:731–5.
13. Parish LC, Witkowski JA. The infected decubitus ulcer. Int J Dermatol **1989**; 28:643–7.
14. Darouiche RO, Landon GC, Klima M, Musher DM, Markowski J. Osteomyelitis associated with pressure sores. Arch Intern Med **1994**; 154:753–8.
15. Galpin JE, Chow AW, Bayer AS, Guze LB. Sepsis associated with decubitus ulcers. Am J Med **1976**; 61:346–50.
16. Chow AW, Galpin JE, Guze LB. Clindamycin for treatment of sepsis caused by decubitus ulcers. J Infect Dis **1977**; 735:565–8.
17. Muder RR, Brennen C, Wagener MM, Goetz AM. Bacteremia in a long-term-care facility: a five-year prospective study of 163 consecutive episodes. Clin Infect Dis **1992**; 14:647–54.
18. Rudensky B, Lipschits M, Isaacsohn M, Sonnenblick M. Infected pressure sores: comparison of methods for bacterial identification. South Med J **1992**; 85:901–3.
19. Ehrenkranz NJ, Alfonso B, Nerenberg D. Irrigation-aspiration for culturing draining decubitus ulcers: correlation of bacteriological findings with a clinical inflammatory scoring index. J Clin Microbiol **1990**; 28: 2389–93.
20. Lewis VL Jr, Bailey MH, Pulawski G, Kind G, Bashioum RW, Henfrix RW. The prognosis of osteomyelitis in patients with pressure sores. Plastic Reconstr Surg **1988**; 81:229–32.
21. Huang AB, Schweitzer ME, Hume E, Batte WG. Osteomyelitis of the pelvis/hips in paralyzed patients: accuracy and clinical utility of MRI. J Comput Assist Tomogr **1998**; 22:437–43.
22. Bergstrom N, Bennett MA, Carlson CE. Clinical practice guideline number 15: treatment of pressure ulcers. Rockville, MD: Agency for Health Care Policy and Research (AHCPR), US Department of Health and Human Services, **1994**. AHCPR publication 95–0652.
23. Newman LG, Waller J, Palestro CJ, et al. Unsuspected osteomyelitis in diabetic foot ulcers: diagnosis and monitoring by leukocyte scanning with indium In 111 oxyquinolone. JAMA **1993**; 266:1246–51.
24. Herman LE, Rothman KF. Prevention, care and treatment of pressure (decubitus) ulcers in intensive care unit patients. J Intensive Care Med **1989**; 4:117–23.
25. Thomas DR. The role of nutrition in prevention and healing of pressure ulcers. Clin Geriatr Med **1997**; 13:497–511.
26. Goode PS, Thomas DR. Pressure ulcers: local wound care. Clin Geriatr Med **1997**; 13:543–52.
27. Krasner D. The AHCPR pressure ulcer infection control recommendations revisited. Ostomy Wound Manage **1999**; 45:88–91S.
28. Braden BJ, Bergstrom N. Clinical utility of the Braden scale for predicting pressure sore risk. Decubitus **1989**; 2:44–6, 50–51.
29. Perneger TV, Gaspoz JM, Rae AC, Borst F, Heliot C. Contribution of individual items to the performance of the Norton pressure ulcer prediction scale. J Am Geriatr Soc **1998**; 46:1282–6.
30. Moody BL, Fanale JE, Thompson M, Vaillancourt D, Symonds G, Bonasoro C. Impact of staff education pressure sore development in elderly hospitalized patients. Arch Intern Med **1988**; 148:2241–3.
31. Mulder GD, LaPan M. Decubitus ulcers: update on new approaches to treatment. Geriatrics **1988**; 43:37–9, 44–5, 49–50.