Chapter 12. Pressure Ulcers: A Patient Safety Issue

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Background

Pressure ulcers remain a major health problem affecting approximately 3 million adults.[1] In 1993, pressure ulcers were noted in 280,000 hospital stays, and 11 years later the number of ulcers was 455,000.[2] The Healthcare Cost and Utilization Project (HCUP) report found from 1993 to 2003 a 63 percent increase in pressure ulcers, but the total number of hospitalizations during this time period increased by only 11 percent. Pressure ulcers are costly, with an average charge per stay of $37,800.[2] In the fourth annual HealthGrades Patient Safety in American Hospitals Study, which reviewed records from about 5,000 hospitals from 2003 to 2005, pressure ulcers had one of the highest occurrence rates, along with failure to rescue and postoperative respiratory failure.[3] Given the aging population, increasingly fragmented care, and nursing shortage, the incidence of pressure ulcers will most likely continue to rise.

Preventing pressure ulcers has been a nursing concern for many years. In fact, Florence Nightingale in 1859 wrote, "If he has a bedsore, it's generally not the fault of the disease, but of the nursing"[4] (p. 8). Others view pressure ulcers as a "visible mark of caregiver sin"[5] (p. 726) associated with poor or nonexistent nursing care.[6] Many clinicians believe that pressure ulcer development is not simply the fault of the nursing care, but rather a failure of the entire heath care system[7]—hence, a breakdown in the cooperation and skill of the entire health care team (nurses, physicians, physical therapists, dietitians, etc.).

Although the prevention of pressure ulcers is a multidisciplinary responsibility, nurses play a major role. In 1992, the U.S. Agency for Healthcare Research and Quality (AHRQ, formerly the Agency for Health Care Policy and Research) published clinical practice guidelines on preventing pressure ulcers.[8] Much of the evidence on preventing pressure ulcers was based on Level 3 evidence, expert opinion, and panel consensus, yet it served as a foundation for providing care. Although the AHRQ document was published 15 years ago, it still serves as the foundation for providing preventive pressure ulcer care and a model for other pressure ulcer guidelines developed afterward. Nurses are encouraged to review these comprehensive guidelines. The document identifies specific processes (e.g., risk assessment, skin care, mechanical loading, patient and staff education, etc.) that, when implemented, could reduce pressure ulcer development, and the literature suggests that following these specific processes of pressure ulcer care will reduce the incidence of ulcers. Research also suggests that when the health care providers are functioning as a team, the incidence rates of pressure ulcers can decrease.[9] Thus, pressure ulcers and their prevention should be considered a patient safety goal.

Incidence, Mortality, and Costs

The incidence rates of pressure ulcers vary greatly with the health care settings. The National Pressure Ulcer Advisory Panel (NPUAP) says the incidence ranges from 0.4 percent to 38 percent in hospitals, from 2.2 percent to 23.9 percent in skilled nursing facilities, and from 0 percent to 17 percent for home health agencies.[10] There is ample evidence that the majority of pressure ulcers

occur relatively early in the admissions process. For patients in the hospital, they can occur within the first 2 weeks.[11] With the increased acuity of elderly patients admitted and decreased lengths of stay in hospital, new data suggest that 15 percent of elderly patients will develop pressure ulcers within the first week of hospitalization.[12] For those elderly residents admitted to long-term care, pressure ulcers are most likely to develop within the first 4 weeks of admission.[13]

Mortality is also associated with pressure ulcers. Several studies noted mortality rates as high as 60 percent for older persons with pressure ulcers within 1 year of hospital discharge.[14, 15] Most often, pressure ulcers do not cause death; rather the pressure ulcer develops after a sequential decline in health status. Thus, the development of pressure ulcers can be a predictor of mortality. Studies further suggested that the development of skin breakdown postsurgery can lead elders to have major functional impairment post surgical procedure.

The cost to treat pressure ulcers can be expensive; the HCUP study reported an average cost of $37,800.[2] Cost data vary greatly, depending on what factors are included or excluded from the economic models (e.g., nursing time, support surfaces). It has been estimated that the cost of treating pressure ulcers is 2.5 times the cost of preventing them.[16] Thus, preventing pressure ulcers should be the goal of all nurses.

Etiology

Pressure ulcers develop when capillaries supplying the skin and subcutaneous tissues are compressed enough to impede perfusion, leading ultimately to tissue necrosis. Since 1930, we have understood that normal blood pressure within capillaries ranges from 20 to 40mm Hg; 32mm Hg is considered the average.[17] Thus, keeping the external pressure less than 32 mm Hg should be sufficient to prevent the development of pressure ulcers. However, capillary blood pressure may be less than 32 mm Hg in critically ill patients due to hemodynamic instability and comorbid conditions; thus, even lower applied pressures may be sufficient to induce ulceration in this group of patients. Pressure ulcers can develop within 2 to 6 hours.[18, 19] Therefore, the key to preventing pressure ulcers is to accurately identify at-risk individuals quickly, so that preventive measures may be implemented.

Risk Factors

More than 100 risk factors of pressure ulcers have been identified in the literature. Some physiological (intrinsic) and nonphysiological (extrinsic) risk factors that may place adults at risk for pressure ulcer development include diabetes mellitus, peripheral vascular disease, cerebral vascular accident, sepsis, and hypotension.[20] A hypothesis exists that these physiological risk factors place the patients at risk due to impairment of the microcirculation system. Microcirculation is controlled in part by sympathetic vasoconstrictor impulses from the brain and secretions from localized endothelial cells. Since neural and endothelial control of blood flow is impaired during an illness state, the patient may be more susceptible to ischemic organ damage (e.g., pressure ulcers).[21]

Additional risk factors that have been correlated with pressure ulcer development are age of 70 years and older, current smoking history, dry skin, low body mass index, impaired mobility, altered mental status (i.e., confusion), urinary and fecal incontinence, malnutrition, physical restraints, malignancy, history of pressure ulcers, and white race.[22–25] Although researchers have noted that the white race is a predictor of pressure ulcers, the small number of nonwhite patients in

most pressure ulcer studies makes this finding questionable. The few studies that have included sufficient numbers of black people for analysis purposes have found that blacks suffer more severe pressure ulcers than nonblacks.[26, 27] Only one nursing study found that blacks had a higher incidence rate of pressure ulcer than whites.[28] In a study funded by AHRQ using the New York State Inpatient Data Set 1998–2000, Fiscella and colleagues[29] found that African Americans were more likely to develop pressure ulcers than other races in hospitals. Moreover, a 2004 study investigating black/white differences in pressure ulcer incidence found that after controlling for eight resident characteristics and three facility characteristics, race was significantly associated with pressure ulcer incidence (hazard ratio comparing blacks with whites = 1.31, 95% confidence interval = 1.02–1.66).[30]

Risk Assessment

What tool and how often a pressure ulcer risk assessment should be done are key questions in preventing pressure ulcers. Due to the number of risk factors identified in the literature, nurses have found the use of risk assessment tools helpful adjuncts to aid in the identification of patients who may be at high risk. Most health care institutions that use pressure ulcer risk assessment tools use either the Braden Scale or Norton Scale, with the Braden scale being the most widely used in the United States. The Braden Scale is designed for use with adults and consists of 6 subscales: sensory perception, moisture, activity, mobility, nutrition, and friction and shear.[31] It is based on the conceptual schema of linking the above clinical situations to the intensity and duration of pressure or tissue tolerance for pressure.[32] The copyrighted tool is available at http://www.bradenscale.com.braden.pdf. The scores on this scale range from 6 (high risk) to 23 (low risk), with 18 being the cut score for onset of pressure ulcer risk. Research has shown that hospital nurses could accurately determine pressure ulcer risk 75.6 percent of the time after an interactive learning session on the Braden scale.[33] Nurses were best at identifying persons at the highest and lowest levels of risk and had the most difficultly with patients with mild levels of risk (scores of 15–18).[34]

The Norton Scale was developed in the United Kingdom and consists of five subscales: physical condition, mental condition, activity, mobility, and incontinence.[35] The total score ranges from 5 (high risk) to 20 (low risk).

The Braden Scale and Norton Scale have been shown to have good sensitivity (83 percent to 100 percent, and 73 percent to 92 percent, respectively) and specificity (64 percent to 77 percent, and 61 percent to 94 percent, respectively), but have poor positive predictive value (around 40 percent and 20 percent, respectively).[36] The Norton and Braden scales show a 0.73 Kappa statistic agreement among at-risk patients, with the Norton Scale tending to classify patients at risk when the Braden scale classifies them as not at risk. The net effect of poor positive predictive value means that many patients who will not develop pressure ulcers may receive expensive and unnecessary treatment. Moreover, optimal cutoff scores have not been developed for each care setting (e.g., medical intensive care versus operating room). Thus, nurses still need to use their clinical judgment in employing preventive pressure ulcer care. A recent systematic review of risk assessment scales found that the Braden Scale had the optimal validation and the best sensitivity/specificity balance (57.1 percent/67.5 percent) when compared to the Norton Scale (46.8 percent/61.8 percent) and Waterlow Scale (82.4 percent/27.4 percent).[37] It should be noted that the Waterlow skill is a pressure ulcer prediction tool used primarily in Europe.

In recent years, several new prediction tools have been developed (FRAGMMENT Score and

Schoonhoven Prediction Rule); however, these tools lack sufficient evidence to evaluate their predictive validity.[38, 39] Thus, the use of a validated pressure ulcer risk assessment tool like the Braden Scale should be used, given the fair research-based evidence. The U.S. Centers for Medicare and Medicaid Services (CMS) recommends that nurses consider all risk factors independent of the scores obtained on any validated pressure ulcer prediction scales because all factors are not found on any one tool.[40]

The usefulness of clinical informatics to assess and prevent pressure ulcers has been explored. A quality improvement study involving 91 long-term care facilities evaluated the usefulness of Web-based reports alerting nursing staff to a resident's potential risk for pressure ulcers.[41] Only one-third of long-term care facilities used the Web-based reports regularly to identify at-risk patients. Several key characteristics of facilities that were high users emerged:

- Administrative level and nursing staff buy-in and support
- Development of an actual process integrating the risk reports into ongoing quality improvement processes
- Having "facility champions" to keep the effort focused and on track

There is no agreement on how frequently risk assessment should be done. There is general consensus from most pressure ulcer clinical guidelines to do a risk assessment on admission, at discharge, and whenever the patient's clinical condition changes. The appropriate interval for routine reassessment remains unclear. Studies by Bergstrom and Braden[42, 43] found that in a skilled nursing facility, 80 percent of pressure ulcers develop within 2 weeks of admission and 96 percent develop within 3 weeks of admission. The Institute for Healthcare Improvement has recently recommended that in hospitalized patients, pressure ulcer risk assessment be done every 24 hours[44] rather than the previous suggestion of every 48 hours.[45]

Implementing a Prevention Plan

Preventing pressure ulcers can be nursing intensive. The challenge is more difficult when there is nursing staff turnover and shortages. Studies have suggested that pressure ulcer development can be directly affected by the number of registered nurses and time spent at the bedside.[46, 47] In contrast, however, one recent study suggested that there was no correlation between increasing the nurse-to-patient ratio and the overall incidence of pressure ulcers.[48] Donaldson and colleagues[49] noted that this particular study was limited by the fact that the researchers could not affirm compliance with ratios per shift and per unit at all times. Given that the cost of treatment has been estimated as 2.5 times that of prevention, implementing a pressure ulcer prevention program remains essential.

A growing level of evidence suggests that pressure ulcer prevention can be effective in all health care settings. One study examined the efficacy of an intensive pressure ulcer prevention protocol to decrease the incidence of ulcers in a 77-bed long-term care facility.[50] The pressure ulcer prevention protocol consisted of preventive interventions stratified on risk level, with implementation of support surfaces and turning/repositioning residents. The sample included 132 residents (69 prior to prevention intervention and 63 after prevention intervention). The 6-month incidence rate of pressure ulcers prior to the intensive prevention intervention was 23 percent. For the 6-months after intensive prevention intervention, the pressure ulcer incidence rate was 5 percent. This study demonstrated that significant reductions in the incidence of pressure ulcers are possible to achieve within a rather short period of time (6 months) when facility-specific intensive prevention interventions are used. A subsequent study by the same researchers was undertaken to

evaluate the cost effectiveness of the pressure ulcer prevention protocol after a 3-year period. The implementation of a pressure ulcer prevention protocol showed mixed results. Initial reductions in pressure ulcer incidence were lost over time. However, clinical results of ulcer treatment improved and treatment costs fell during the 3 years.[51]

A more recent nursing study examined the effects of implementing the SOLUTIONS program, which focuses pressure ulcer prevention measures on alleviating risk factors identified by the Braden Scale, in two long-term care facilities.[52] The quasi-experimental study found that after 5 months of implementing the SOLUTIONS program, Facility A (150 beds) experienced an 87 percent reduction in pressure ulcer incidence (from 13.2 percent to 1.7 percent), which was highly significant ($P = 0.02$). Facility B (110 beds) experienced a corresponding 76 percent reduction (from 15 percent to 3.5 percent), which was also highly significant ($P = 0.02$). Gunningberg and colleagues[52] investigated the incidence of pressure ulcers in 1997 and 1999 among patients with hip fractures and found significant reductions in incidence rates (55 percent in 1997 to 29 percent in 1999). The researchers attributed these reductions in pressure ulcer incidence rates to performing systematic risk assessment upon admission, accurately staging pressure ulcers, using pressure-reducing mattresses, and continuing education of staff. Thus, the use of comprehensive prevention programs can significantly reduce the incidence of pressure ulcers in long-term care.

The use of quality improvement models, where systematic processes of care have been implemented have also been shown to reduce overall pressure ulcer incidence. In one study involving 29 nursing homes in three States, representatives of the 29 nursing homes attended a series of workshops, shared best practices, and worked with one-on-one quality improvement mentors over 2 years.[53] This study found that six of eight prevention process measures (based on AHRQ prevention guidelines) significantly improved, with percentage differences between baseline and followup ranging from 11.6 percent to 24.5 percent. Another study using similar methods involving 22 nursing homes found 8 out of 12 processes of care significantly improved.[7] Moreover, the study found that pressure ulcer incidence rates decreased in the nursing homes. Nursing homes with the greatest improvement in quality indicator scores had significantly lower pressure ulcer incidence rates than the facilities with the least improvement in quality indicator scores ($P = 0.03$).

In the acute care setting, several studies have attempted to demonstrate that the implementation of comprehensive pressure ulcer prevention programs can decrease the incidence rates. However, no studies could be found that eliminated pressure ulcers. One large study evaluated the processes of care for hospitalized Medicare patients at risk for pressure ulcer development.[7] This multicenter retrospective cohort study used medical record data to identify 2,425 patients ages 65 and older discharged from acute care hospitals following treatment for pneumonia, cerebral vascular disease, or congestive heart failure. Charts were evaluated for the presence of six recommended pressure ulcer prevention processes of care. This study found that at-risk patients who used pressure-reducing devices, were repositioned every 2 hours, and received nutritional consults were more likely to develop pressure ulcers than those patients who did not receive the preventive interventions. One explanation for this finding may be the amount of time (48 hours) before the preventive measures were implemented. Given the acuity of patients entering hospitals, waiting 48 hours may be too late to begin pressure ulcer prevention interventions. Thus, despite this one study, there is significant research to support that implementing comprehensive pressure ulcer prevention programs reduces the incidence of pressure ulcers.

A key component of research studies that have reported reduction of pressure ulcers is how to

sustain the momentum over time, especially when the facility champion leaves the institution. It is clear from the evidence that maintaining a culture of pressure ulcer prevention in a care setting is an important challenge, one that requires the support of administration and the attention of clinicians.

Skin Care

Although expert opinion maintains that there is a relationship between skin care and pressure ulcer development, there is a paucity of research to support that. How the skin is cleansed may make a difference. One study found that the incidence of Stages I and II pressure ulcers could be reduced by educating the staff and using a body wash and skin protection products.[54]

The majority of skin care recommendations are based on expert opinion and consensus. Intuitively nurses understand that keeping the skin clean and dry will prevent irritants on the skin or excessive moisture that may increase frictional forces leading to skin breakdown. Individualized bathing schedules and use of nondrying products on the skin are also recommended. Moreover, by performing frequent skin assessments, nurses will be able to identify skin breakdown at an early stage, leading to early interventions. Although there is a lack of consensus as to what constitutes a minimal skin assessment, CMS recommends the following five parameters be included: skin temperature, color, turgor, moisture status, and integrity.[40]

The search for the ideal intervention to maintain skin health continues. One study compared hyperoxygenated fatty acid compound versus placebo compound (triisotearin) in acute care and long-term care patients.[55] These researchers found that using hyperoxygenated fatty acid significantly (p-0.006) reduced the incidence of ulcers. Pressure ulcer incidence was lower in an intervention group of acute care patients when topical nicotinate was applied (7.32 percent) compared to lotion with hexachlorophene, squalene, and allantoin in the control group (17.37 percent).[56]

There are several key recommendations to minimize the occurrence of pressure ulcers. Avoid using hot water, and use only mild cleansing agents that minimize irritation and dryness of the skin.[8, 57] Avoid low humidity because it promotes scaling and dryness, which has been associated with pressure ulcer development.[23] During skin care, avoid vigorous massage over reddened, bony prominences because evidence suggest that this leads to deep tissue trauma. Skin care should focus on minimizing exposure of moisture on the skin.[58] Skin breakdown caused by friction may be mitigated by the use of lubricants, protective films (e.g., transparent and skin sealants), protective dressings (e.g., hydrocolloids), and protective padding.

Mechanical Loading

One of the most important preventive measures is decreasing mechanical load. If patients cannot adequately turn or reposition themselves, this may lead to pressure ulcer development. It is critical for nurses to help reduce the mechanical load for patients. This includes frequent turning and repositioning of patients.

Very little research has been published related to optimal turning schedules. The first such nursing study was an observational one that divided older adults into three turning treatment groups (every 2 to 3 hours [n = 32], every 4 hours [n = 27], or turned two to four times/day [n = 41]).[59] These researchers found that older adults turned every 2 to 3 hours had fewer ulcers. This landmark nursing study created the gold standard of turning patients at least every 2 hours. Some

researchers would suggest that critically ill patients should be turned more often. However, one survey study investigating body positioning in intensive care patients found that of 74 patients observed, 49.3 percent were not repositioned for more than 2 hours.[60] Only 2.7 percent of patients had a demonstrated change in body position every 2 hours. A total of 80–90 percent of respondents to the survey agreed that turning every 2 hours was the accepted standard and that it prevented complications, but only 57 percent believed it was being achieved in their intensive care units. A more recent study by DeFloor and colleagues[61] suggests that depending on the support surface used, less-frequent turning may be optimal to prevent pressure ulcers in a long-term care facility. Several nurse researchers investigated the effect of four different turning frequencies (every 2 hours on a standard mattress, every 3 hours on a standard mattress, every 4 hours on a viscoelastic foam mattress, and every 6 hours on a viscoelastic foam mattress). The nurse researchers found that the incidence of early pressure ulcers (Stage I) did not differ in the four groups. However, patients being turned every 4 hours on a viscoelastic foam mattress developed significantly less severe pressure ulcers (Stage II and greater) than the three other groups. Although the results of this study may indicate less turning may be appropriate when using a viscoelastic foam mattress, additional studies are needed to examine optimal turning schedules among different populations. Reddy and colleagues[62] have raised questions about the methodology in the Defloor and colleagues study, leading them to recommend that it may be too soon to abandon the every-2-hours turning schedule in favor of every 4 hours based on this one study. Thus, there is emerging research to support the continued turning of patients at least every 2 hours.

How a patient is positioned may also make a difference. Lateral turns should not exceed 30 degrees.[63, 64] One randomized controlled trial that studied a small sample of 46 elderly patients in the 30-degree-tilt position and the standard 90-degree side-lying position found no significant difference in the development of pressure ulcers between the two groups.[65]

Support Surfaces

The use of support surfaces is an important consideration in pressure redistribution. The concept of pressure redistribution has been embraced by the NPUAP.[66] You can never remove all pressure for a patient. If you reduce pressure on one body part, this will result in increased pressure elsewhere on the body. Hence, the goal is to obtain the best pressure redistribution possible.

A major method of redistributing pressure is the use of support surfaces. Much research has been conducted on the effectiveness of the use of support surfaces in reducing the incidence of pressure ulcers. A comprehensive literature review by Agostini and colleagues[67] found that there was adequate evidence that specially designed support surfaces effectively prevent the development of pressure ulcers. However, a major criticism of the current support surface studies was poor methodologic design. Agostini and colleagues noted that many studies had small sample sizes and unclear standardization protocols, and assessments were not blind.

Reddy and colleagues[62] have provided a systematic review of 49 randomized controlled trials that examined the role of support surfaces in preventing pressure ulcers. No one category of support surface was found to be superior to another; however, use of a support surface was more beneficial than a standard mattress. A prospective study evaluating the clinical effectiveness of three different support surfaces (two dynamic mattress replacement surfaces and one static foam mattress replacement) found that an equal number of patients developed pressure ulcers on each surface (three per surface).[68] The researchers concluded no differences in the support surface effectiveness, yet large differences in the cost. (Dynamic mattress replacements cost

approximately $2,000 per mattress, compared to $240 per mattress for static foam mattress replacements.) Given the similar clinical effectiveness, cost should be considered in determining the support surface.

Four randomized controlled trials evaluated the use of seat cushions in pressure ulcer prevention, and found no difference in ulcer incidence among groups except between foam and gel cushions.[62] Despite the dearth of research that correlates seat cushions and preventing pressure ulcers, expert opinion supports the use of seat cushions.

The CMS has divided support surfaces into three categories for reimbursement purposes.[68] Group 1 devices are those support surfaces that are static, they do not require electricity. Static devices include air, foam (convoluted and solid), gel, and water overlays or mattresses. These devices are ideal when a patient is at low risk for pressure ulcer development. Group 2 devices are powered by electricity or pump and are considered dynamic in nature. These devices include alternating and low-air-loss mattresses. These mattresses are good for patients who are at moderate to high risk for pressure ulcers or have full-thickness pressure ulcers. Group 3 devices, also dynamic, comprises only air-fluidized beds. These beds are electric and contain silicone-coated beads. When air is pumped through the bed, the beads become liquid. These beds are used for patients at very high risk for pressure ulcers. More often they are used for patients with nonhealing full-thickness pressure ulcers or when there are numerous truncal full-thickness pressure ulcers. The NPUAP has suggested new definitions for support surfaces that move away from these categories and divide support surfaces into powered or nonpowered.[69] Whether these new definitions will be embraced by CMS is yet to be determined.

There remains a paucity of research that demonstrates significant differences in the effectiveness of the various classifications of support surfaces in preventing or healing pressure ulcers. Therefore, nurses should select a support surface based on the needs and characteristics of the patient and institution (e.g., ease of use, cost). It is imperative to have the pressure redistribution product (e.g., mattress or cushion) on the surface where the patients are spending most of their time, in bed or a chair. However, being on a pressure-redistributing mattress or cushion does not negate the need for turning or repositioning.

Nutrition

Controversy remains on how best to do nutritional assessment for patients at risk for developing pressure ulcers. The literature differs about the value of serum albumin; some literature reports that low levels are associated with increased risk.[70] While the AHRQ pressure ulcer prevention guideline suggests that a serum albumin of less than 3.5 gm/dl predisposes a patient for increased risk of pressure ulcers, one study reveals that current dietary protein intake is a more independent predictor than this lab value.[8, 42] In the revised Tag F-314 guidance to surveyors in long-term care, CMS recommends that weight loss is an important indicator.[40] Evaluation of the patient's ability to chew and swallow may also be warranted.

The literature is unclear about protein-calorie malnutrition and its association with pressure ulcer development.[70] Reddy and colleagues[62] suggested that the widely held belief of a relationship between nutrition intake and pressure ulcer prevention was not always supported by randomized controlled trials. Some research supported the finding that undernourishment on admission to a health care facility increases a person's likelihood of developing a pressure ulcer. In one prospective study, high-risk patients who were undernourished on admission to the hospital were twice as likely to develop pressure ulcers as adequately nourished patients (17 percent and

9 percent, respectively).[71] In another study, 59 percent of residents were undernourished and 7.3 percent were severely undernourished on admission to a long-term care facility. Pressure ulcers occurred in 65 percent of the severely undernourished residents, while no pressure ulcers developed in the mild-to-moderately undernourished or well-nourished residents.[15]

Reddy and colleagues[62] concluded that nutritional supplementation was beneficial in only one of the five randomized controlled trials reviewed in their systematic analysis of interventions targeted at impaired nutrition for pressure ulcer prevention. Older critically ill patients who had two oral supplements plus the standard hospital diet had lower risk of pressure ulcers compared to those who received only the standard hospital diet.[72]

Empirical evidence is lacking that the use of vitamin and mineral supplements (in the absence of deficiency) actually prevents pressure ulcers.[73] Therefore, oversupplementing patients without protein, vitamin, or mineral deficiencies should be avoided. Before enteral or parental nutrition is used, a critical review of overall goals and wishes of the patient, family, and care team should be considered.[74] Despite the lack of evidence regarding nutritional assessment and intervention, maintaining optimal nutrition continues to be part of best practice.

Management of Pressure Ulcers

When a pressure ulcer develops, nursing's patient safety goal is to assist the health care team in closing the ulcer as quickly as possible. Nursing is also concerned with preventing further ulcer deterioration, keeping the ulcer clean and in moisture balance, preventing infections from developing, and keeping the patient free from pain.

Many aspects of managing pressure ulcers are similar to prevention (mechanical loading, support surfaces, and nutrition). Clearly, the health care team has to address the underlying causes (intrinsic and extrinsic) or the pressure ulcer will not close. In 1994, AHRQ published clinical practice guidelines on treating pressure ulcers.[75] Much of the evidence related to treating pressure ulcers was based on Level C evidence, requiring one or more of the following: one controlled trial, results of at least two case series/descriptive studies in humans, or expert opinion. Although the AHRQ document was published 13 years ago, it provides the foundation for treating pressure ulcers. The document identified specific indices (e.g., wound assessment, managing tissue load, ulcer care, managing bacterial colonization/infection, etc.). The following section supplements this document.

Cleansing

Once the pressure ulcer develops, the ulcer should be cleaned with a nontoxic solution. Cleaning the ulcer removes debris and bacteria from the ulcer bed, factors that may delay ulcer healing.[76] No randomized control studies could be found that demonstrated the optimal frequency or agent for cleansing a pressure ulcer. A Cochrane review of published randomized clinical trials found three studies addressing cleansing of pressure ulcers, but this systematic review produced no good trial evidence to support any particular wound cleansing solution or technique for pressure ulcers.[77] Therefore, this recommendation remains at the expert opinion level. Nurses should use cleansers that do not disrupt or cause trauma to the ulcer.[78] Normal saline (0.9 percent) is usually recommended because it is not cytotoxic to healthy tissue.[79] Although the active ingredients in newer wound cleansers may be noncytotoxic (surfactants), the inert carrier may be

cytotoxic to healthy granulation tissue.[80] Thus, nurses should be cognizant of the ingredients in cleansing agents before using them on pressure ulcers.

Assessment and Staging

The nurse should assess and stage the pressure ulcer at each dressing change. Experts believe that weekly assessments and staging of pressure ulcers will lead to earlier detection of wound infections as well as being a good parameter for gauging of wound healing.[40, 75] There are no universal parameters for assessing a pressure ulcer. Most experts agree that when a pressure ulcer develops its location, size (length, width, and depth), and color of the wound; amount and type of exudate (serous, sangous, pustular); odor; nature and frequency of pain if present (episodic or continuous); color and type of tissue/character of the wound bed, including evidence of healing (e.g., granulation tissue) or necrosis (slough or eschar); and description of wound edges and surrounding tissue (e.g., rolled edges, redness, hardness/induration, maceration) should be assessed and documentd.[75, 81] Upon identifying the ulcer characteristics, the initial stage of the should be completed.

The staging system is one method of summarizing certain characteristics of pressure ulcers, including the extent of tissue damage. Hence, whether the nurse observes the epidermis, dermis, fat, muscle, bone, or joint determines the stage of pressure ulcer. Knowing the appropriate stage aids in determining the management of the pressure ulcer. However, staging of pressure ulcers can vary, because different nurses may observe different tissue types. In a survey of nurses' wound care knowledge, less than 50 percent of new nurses (fewer than 20 years of nursing experience) did not feel confident in consistently identifying all stages of pressure ulcers, as compared to 30 percent of the more experienced nurses (more than 20 years of nursing experience).[82] Achieving consistency in staging will provide optimal pressure ulcer management.

Pressure ulcer staging systems differ, depending on geographic location. The Europeans use a four-stage system.[83] For Grade 1, nonblanchable erythema of intact skin, discoloration of the skin, warmth, edema, and induration or hardness may be used as indicators, particularly on individuals with darker skin. For Grade 2, indicators include partial thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion or blister. Grade 3 includes full thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. Grade 4 includes extensive destruction; tissue necrosis; or damage to muscle, bone, or supporting structures, with or without full thickness skin loss.

The most widely used staging system in the United States was developed in 1989 by the NPUAP.[84] This staging system was modified from Shea's original system.[85] The staging system rates the pressure ulcer from superficial tissue damage (Stage I) to full thickness skin loss involving muscle or bone (Stage IV). If the pressure ulcer is covered with necrotic tissue (eschar), it should be noted as unstageable. In skilled nursing facilities, nurses must stage a pressure ulcer covered with necrotic tissue as Stage IV.[86] In home care and nursing homes, nurses must stage pressure ulcers because staging is linked to reimbursement of medical expenses.

In 2007, the NPUAP revised the staging system to include deep tissue injury, an ulcer often described as a purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear.[87] The NPUAP also reclassified blisters and unstageable pressure ulcers. The NPUAP staging definitions were refined with input from an online evaluation of their face validity, accuracy clarity, succinctness, utility, and

discrimination. The new staging system has six stages: suspected deep tissue injury, Stage I, Stage II, Stage III, Stage IV, and Unstageable. Table 1 presents the NPUAP definition, and Table 2 illustrates the differences between the old and new pressure ulcer staging systems.

Table 1. National Pressure Ulcer Definition

	Previous NPUAP Definition	2007 NPUAP Definition	2007 NPUAP Further Descriptions to Accompany Revised Definition
Pressure Ulcer Definition	A localized area of tissue necrosis that develops when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time.	A pressure ulcer is localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction.	A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated.

Table 2. National Pressure Ulcer Staging System

Pressure Ulcer Stage	Previous NPUAP Staging Definitions	2007 NPUAP Definitions	2007 NPUAP Descriptions to Accompany Revised Definitions
Deep Tissue Injury	A pressure-related injury to subcutaneous tissues under intact skin. Initially, these lesions have the appearance of a deep bruise, and they may herald the subsequent development of a Stage III–IV pressure ulcer, even with optimal treatment.	Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear.	• The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler, as compared to adjacent tissue. • Deep tissue injury may be difficult to detect in individuals with dark skin tones. • The area may rapidly evolve to expose additional layers of tissue, even with optimal treatment.
Stage I	An observable pressure-related alteration of intact skin whose indicators as compared to an adjacent or opposite area on the body may include changes in one or more of the following parameters: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel), sensation (pain, itching), and/or a defined area of persistent redness in lightly pigmented skin; in darker skin tones, the ulcer may appear with persistent red, blue, or purple hues.	Intact skin with nonblanchable redness of a localized area, usually over a bony prominence.	• The area may be painful, firm, soft, warmer, or cooler, as compared to adjacent tissue. • Stage I may be difficult to detect in individuals with dark skin tones. • May indicate at-risk persons (a heralding sign of risk).
Stage II	Partial thickness skin loss involving the epidermis and/or dermis. The ulcer is superficial and presents clinically as an abrasion,	Partial thickness loss of dermis presenting as a shallow open ulcer with a red, pink wound bed without slough. May also present as	Presents as a shiny or dry shallow ulcer without slough or bruising. This stage should not be used to describe skin tears, tape burns, perineal

Pressure Ulcer Stage	Previous NPUAP Staging Definitions	2007 NPUAP Definitions	2007 NPUAP Descriptions to Accompany Revised Definitions
	blister, or shallow crater.	an intact or open/ruptured serum-filled blister.	dermatitis, maceration, or excoriation.
Stage III	Full thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.	Full thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are *not* exposed. Slough may be present but does not obscure the depth of tissue loss. *May* include undermining and tunneling.	• The depth of a Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Stage III pressure ulcers. • Bone/tendon is not visible or directly palpable.
Stage IV	Full thickness skin loss with extensive destruction; tissue necrosis; or damage to muscle, bone, or supporting structure (such as tendon, or joint capsule).	Full thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. *Often* includes undermining and tunneling.	• The depth of a Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue, and these ulcers can be shallow. • Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon, or joint capsule), making osteomyelitis likely to occur. • Exposed bone/tendon is visible or directly palpable.
Unstagable		Full thickness tissue loss in which *actual* depth of the ulcer is *completely* obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed.	Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, or adherent, intact without erythema or fluctuance) eschar on the heels serves as the "the body's natural (biological) cover" and should not be removed.

The Stage I pressure ulcer may be more difficult to detect in darkly pigmented skin. A quality improvement study in several nursing homes found that by empowering the nursing assistants with education (skin assessment), use of pen lights to assess darker skin, mirrors, and financial reward, the researchers were able to reduce the Stage I pressure ulcers in residents with darkly pigmented skin.[88] One method for delineating Stage I pressure ulcers in darkly pigmented skin may be the use of high-resolution ultrasound. Although ultrasound is widely used as a safe and cost-effective technique for noninvasive visualization of specific human anatomy, its use for skin assessment is just now available. Ultrasound utilizes the echoes of sound waves to create images of soft tissue anatomy.[89] A probe transmits sound waves into the body. High-frequency ultrasound (20MHZ)

will provide high resolution images of the skin and underlying soft tissue, and because the images are related to tissue density (not pigment), the clinician's assessment ability is enhanced significantly. A recent study strongly suggests that clinicians should consider high-frequency ultrasound as an improved method for identifying and implementing good pressure ulcer preventive care.[90]

The assessment and staging of pressure ulcers remains at the expert opinion level.

Debridement

The presence of necrotic devitalized tissue promotes the growth of pathologic organisms and prevents wounds from healing.[91] Experts believe that debridement is an important step in the overall management of pressure ulcers. No randomized control trials could be found that demonstrated that one debridement technique is superior. Thus, the best method of debridement is determined by the goals of the patient, absence or presence of infection, pain control, amount of devitalized tissue present, and economic considerations for the patient and institution.[92–94] There are five types of debridement: sharp, mechanical, autolytic, enzymatic, and biosurgery.

Sharp debridement (use of scalpel or laser) is probably the most effective type of debridement because of the time involved to remove the devitalized tissue.[95] Sharp debridement should always be considered when the patient is suspected of having cellulites or sepsis.[96] Mechanical debridement uses a nonselective, physical method of removing necrotic tissue and debris from a wound using mechanical force. One common form of mechanical treatment is wet-to-dry gauze to adhere to the necrotic tissue, which is then removed. Upon removal of the gauze dressing, necrotic tissue and wound debris are also removed. The challenge with mechanical debridement is the possibility that healthy granulation tissue may be removed as well, along with the devitalized tissue, thereby delaying wound healing and causing pain. Thus, CMS suggests that this method of debridement be used in limited circumstances.[40]

Autolytic debridement involves the use of semiocclusive (transparent film) and occlusive dressings (hydrocolloids, hydrogels, etc.), which creates an environment for the body's enzymes to break down the necrotic tissue.[97] Enzymatic debridement uses proteolytic enzymes (i.e., papain/urea, collagenase) to remove necrotic tissue.[98] This form of debridement is considered drug therapy; therefore it should be signed on the medication record. Finally, biosurgery (maggot therapy) is another effective and relatively quick method of debridement.[99] This type of debridement is especially effective when sharp debridement is contraindicated due to the exposure of bone, joint, or tendon.[99]

Bacterial Burden

Managing bacterial burden is an important consideration in pressure ulcer care. All pressure ulcers contain a variety of bacteria. Pressure ulcer bacterial contamination should not impair health.[100] Of great concern is when a colony of bacteria reaches 10^5 or 10^6 organisms per gram in the ulcer.[101] At these levels, the pressure ulcer can be considered infected. Healing can be impeded when wounds have high levels of bacteria. Robson and Heggers[101] found in 32 pressure ulcers that spontaneous healing occurred only when the microbial population was controlled.

Experts agree that swab cultures should not be used to determine wound infection.[102] Rather a tissue biopsy should be conducted to determine the qualitative and quantitative assessment of any aerobic and anaerobic organisms present.[103] Clinical signs that the pressure ulcer may be infected

include malodorous, purulent exudate; excessive draining; bleeding in the ulcer; and pain.[104, 105] One study investigating the validity of clinical signs and symptoms used to identify localized chronic wound infections found signs associated with secondary wounds (i.e., serous exudate, delayed healing, discoloration of granulation tissue, friable granulation tissue, pocketing at the base of the wound, foul odor, and wound breakdown) were better predictors of wound infection than the classic signs of infection (i.e., increasing pain, erythema, edema, heat, and purulence).[106] Overall, these researchers concluded that increasing pain and wound breakdown were both sufficient clinical indicators of infected wounds with 100 percent specificity. Thus, when these signs are present, the nurse should seek additional treatments for the patient. This will help to safeguard the patient from further ulcer complications.

The use of oral antibiotics or topical sulfa silverdiazine has also been found to be effective in decreasing the bioburden in the ulcer bed.[107, 108] Treatment using silver-impregnated dressings has been shown to be somewhat effective in decreasing bacterial bioburden load. One in vivo study found that silver-based dressings decreased specific bacteria (e.g., Eschericha coli, Candida albicans, and Staphylococcus aureas).[109] However, a systematic review of the research literature found only three randomized controlled trials covering 847 participants. This Cochrane review determined that based on only three randomized controlled trials, there remains insufficient evidence to recommend the use of silver-containing dressings or topical agents for treatment of infected or contaminated chronic wounds.[110]

The use of antiseptics to reduce wound contamination continues to be a controversial topic. The ideal agent for an infected pressure ulcer would be bactericidal to a wide range of pathogens and noncytotoxic to leukocytres. In vitro studies of 1 percent povidone-iodine have been found to be toxic to fibroblast, but a solution of 0.005% sodium hypochlorite ($P = 0.001$) caused no fibroblast toxicity and was still bactericidal to Staphylococcus aureus.[111] Another common antiseptic with conflicting data is sodium hypochlorite (Dakins solution). Studies suggest that 0.005 percent concentration of sodium hypochlorite to be bactericidal; however, its use can also cause inhibition of fibroblast and neutrophil migration necessary for pressure ulcer healing.[112] Conversely, other in vitro studies suggest that 0.005 percent sodium hypochlorite did not inhibit fibroblasts. McKenna and colleagues examined the use of 0.005 percent sodium hypochlorite, 0.001 percent povidone-iodine, 0.0025 percent acetic acid, and 0.003 percent hydrogen peroxide on various clinical isolates.[111] These researchers found that sodium chlorite significantly inhibited ($P = 0.001$) the growth of all bacteria tested (Staphylococcus aureas, Escherichia coli, Group D enterococci, Pseudomonas aeruginosa, and Bacteroides fragilis) without inhibiting fibroblast activity, whereas povidone-iodine and acetic acid reduced only specific bacteria.

Exudate Management

The use of dressings is a major component in maintaining a moist environment. There are more than 300 different modern wound dressings available to manage pressure ulcers.[113] Most dressings can be broken down into seven classifications: transparent films, foam islands, hydrocolloids, petroleum-based nonadherents, alginates, hydrogels, and gauze. Few randomized controlled studies have been conducted to evaluate the efficacy of dressings within a specific classification. Therefore, no one category of wound dressings (independent of gauze) may be better than another category. Most research evaluating the effects of dressings usually compare gauze (standard) to modern wound dressings (nongauze).[114, 115, 116] These studies are inherently flawed because gauze dressings are not classified as a modern wound dressing; thus equivalent comparisons cannot be

made. The studies usually have small sample sizes; thus inferences can be difficult to make. However, one study investigating wound-healing outcomes using standardized validated protocols found that primarily using nongauze protocols of care matched or surpassed the best previously published results on similar wounds using gauze-based protocols of care, including protocols applying gauze impregnated with growth factors or other agents. Thus, nongauze protocols of care should be used to accelerate pressure ulcer healing.[117]

Nutrition

The use of high-protein diets for patients with protein deficiency is essential to wound healing. One small study (n = 12) has suggested that 1.25 g protein/L/kg/day to 1.50 g protein/L/kg/day is needed to promote wound healing.[118] However, Mulholland and colleagues[119] suggested in a 1943 journal article that as much as 2.0 g protein/L/kg/day is essential for wound healing. To underscore that increasing protein does have a positive effect on wound healing, researchers investigated 28 malnourished patients with a total of 33 truncal pressure ulcers.[120] The researchers found that patients who received the 24-percent protein intake had significant decrease ($P = 0.02$) in truncal pressure ulcer surface area compared to the group on 14-percent protein intake. Clearly, increasing protein stores for patients with pressure ulcers who are malnourished is essential; however, it is unclear from the literature what the optimum protein intake requirement is for patients with pressure ulcers. Most promising: the use of amino acids such as argine, glutamine, and cysteine have been noted to assist in ulcer healing.[121] However, there remains a paucity of data to substantiate these claims; thus their use should be tempered with the overall goals of the patient.

Pain Management

Pressure ulcers can be painful. In particular, patients with Stage IV ulcers can experience significant pain.[122, 123] A cross-sectional study of patients with a mix of chronic wounds found that wound stage was positively related to severity of pain.[123] Moreover, pain catastrophizing was positively related to pain intensity and higher levels of affective distress and depressive symptoms. Hence, the goal of pain management in the patient with pressure ulcers should be to eliminate the cause of pain, to provide analgesia, or both. This goal was supported recently by the World Union of Wound Healing Societies consensus document, *Principles of Best Practice: Minimizing Pain at Wound Dressing-Related Procedures.*[124] Pain at dressing-related procedures can be managed by a combination of accurate assessment, suitable dressing choices, skilled wound management, and individualized analgesic regimens. Dressing removal can potentially cause damage to delicate tissue in the wound and surrounding skin. Thus, clinicians should use multiple methods to address the pressure ulcer pain. This may include using dressing that mitigates pain during dressing changes, such as dressings containing soft-silicone, and administering analgesic prior to dressing changes.

Monitoring Healing

Presently, there are two instruments that are often used to measure the healing of pressure ulcers. The Pressure Ulcer Scale for Healing (PUSH) was developed by the NPUAP in 1997.[125] The PUSH tool is copyrighted and available on NPUAP's Web site.[84] It quantifies the pressure ulcer with respect to surface area, exudate, and type of wound tissue. Using a Likert scale from 1

to 10 for length and width, a Likert scale from 1 to 3 for exudate amount, and a Likert scale from 1 to 4 for tissue type, the nurse can determine whether a pressure ulcer is healing or nonhealing. Each of the three ulcer characteristics is recorded as a subscore, then the subscores are added to obtain the total score. A comparison of total scores measured over time provides an indication of the improvement or deterioration of the pressure ulcer.

Few studies have been published that measure the validity and reliability of the PUSH tool. A study investigating the PUSH tool's content validity found that it had both content validity (P = 0.01) and correlational validity (P = 0.05) to monitor the changing pressure ulcer status.[126] Moreover, a recent prospective study by Gardner and colleagues[106] of 32 pressure ulcers found that 21 ulcers (66 percent) healed during the 6-month study period, and 11 (34 percent) did not heal. The PUSH scores decreased significantly (P = 0.001) over time among the healed ulcers but did not among the unhealed ulcers. Thus, the PUSH tool was shown to be a valid instrument for measuring healing in a clinical setting.

The Bates-Jensen Wound Assessment Tool (BWAT; formerly the Pressure Sore Status Tool, PSST) was developed in 1992 and is also widely used.[127] The BWAT consists of 15 items. The first 2 items are related to location and shape of the ulcer. The remaining 13 items are scored on the basis of descriptors of each item and ranked on a modified Likert scale (1 being the healthiest attribute of the characteristic and 5 being the least healthy attribute of the characteristic). The 13 BWAT characteristics that are scored are size, depth, edges, undermining, necrotic tissue type, necrotic tissue amount, exudate type, exudate amount, skin color surrounding wound, peripheral tissue edema, peripheral tissue induration, granulation tissue, and epithelialization. The 13 item scores are summed to provide a numerical indicator of wound health or degeneration.

There is a paucity of validation studies for the BWAT. However, content validity has been established by a panel of 20 experts. Interrater reliability was established by the use of two wound, ostomy, and continence nurses who independently rated 20 pressure ulcers on 10 patients. Interrrater reliability was established at r = 0.91 for first observation and r = 0.92 for the second observation (P = 0.001).[128] A recent study examined wound-healing outcomes with standardized assessments using the BWAT. Most of the 767 wounds selected to receive the standardized protocols of care were Stage III–IV pressure ulcers (n = 373; mean healing time 62 days). Partial thickness wounds healed faster than same-etiology full thickness wounds.[117] This finding further adds to the validation of the BWAT tool for measuring wound healing.

Adjunctive Therapies

The use of adjunctive therapies is the fastest growing area in pressure ulcer management. Adjunctive therapies include electrical stimulation, hyperbaric oxygen, growth factors and skin equivalents, and negative pressure wound therapy. Except for electrical stimulation, there is a paucity of published research to substantiate the effectiveness of adjunctive therapies in healing pressure ulcers.

Electrical stimulation is the use of electrical current to stimulate a number of cellular processes important to pressure ulcer healing.[129] These processes include increasing the fibroblasts, neutrophil macrophage collagen, DNA synthesis, and increasing the number of receptor sites for specific growth factors.[129] Eight randomized controlled studies were found in the literature. Electrical stimulation appears to be most effective on healing recalcitrant Stages III and IV pressure ulcers.[130] A meta-analysis of 15 studies evaluating the effects of electrical stimulation on the healing of chronic ulcers found that the rate of healing per week was 22 percent for the

electrical stimulation group compared to 9 percent for the control group.[131] Thus, electrical stimulation should be considered for nonhealing pressure ulcers.

Negative pressure wound therapy is widely used, although few randomized controlled trials have been published. This therapy promotes wound healing by applying controlled localized, negative pressure to the wound bed.[132–134] In one prospective study investigating nonhealing pressure ulcers, 24 patients were randomized into two groups (wet-to-moist dressings or vacuum-assisted closure).[133] Those patients receiving negative pressure wound therapy had a 66-percent reduction in wound depth ($P = 0.0001$), compared to the wet-to-moist dressings group, which had a 20-percent wound depth reduction.[133] Much more research is needed on the benefits of negative pressure wound therapy for treating pressure ulcers, but there is emerging evidence that this therapy may be helpful in assisting the healing of pressure ulcers.

The use of growth factors and skin equivalents in the healing of pressure ulcers remains under investigation, although the use of cytokine growth factors (e.g., recombinant platelet-derived growth factor-BB [rhPDGF-BB]) and fibroblast growth factors (bFGF) and skin equivalents have been shown to be effective in diabetic and venous ulcers. Three small randomized controlled trials have suggested that growth factors had beneficial results with pressure ulcers, but the findings warrant further exploration.[135–137] When we learn more about the healing cascade, the appropriate use of growth factors in pressure ulcer treatment may become clearer.

The use of electroceuticals—highly refined electromagnetic fields that can accelerate the body's natural anti-inflammatory response, thereby aiding wounds to heal faster—is showing some promising results. One animal study used a prospective, randomized, double-blind, placebo-controlled design to evaluate the effect of a specific noninvasive radiofrequency-pulsed electromagnetic field signal on tendon tensile strength at 21 days after transection in a rat model.[138] This study found an increase in tensile strength of up to 69 percent (136.4 ± 31.6 kg/cm^2) at the repair site of the rat Achilles' tendon at 3 weeks after transection and repair, compared with the value (80.6 ± 16.6 kg/cm^2) in nonstimulated control animals. Although electroceuticals are promising, additional research is needed to recommend them for pressure ulcer treatment.

The use of therapeutic ultrasound for pressure ulcers has also been explored. A Cochrane review found three published randomized clinical trails using therapeutic ultrasound.[139] It was concluded that there was no evidence of the benefit of ultrasound therapy in the treatment of pressure ulcers. Thus, additional studies are needed before this therapy can be supported.

Evidence-Based Practice Implications

Much progress has been made in identifying patients at risk for pressure ulcers. The use of pressure ulcer prediction tools (e.g., Braden Scale) have led to nursing's sensitivity to earlier preventive measures. Research has shown that using the AHRQ guidelines on pressure ulcer prediction and prevention can lead to decreased incidence of pressure ulcers. Moreover, internalizing these guidelines throughout the health care system can lead to pressure ulcer reductions.

Much progress has been made in understanding effective wound treatments. Treatments range from using traditional therapies (keeping the wound moist, appropriate repositioning, support surfaces, and proper nutrition) to the wise use of adjunctive therapies. Although many studies in pressure ulcer prevention and treatment have small sample sizes, there is a growing body of evidence to suggest that newer wound modalities can be effective in preventing and treating pressure ulcers.

Research Implications

Since the original publications of the AHRQ pressure ulcer prevention and treatment guidelines in 1992 and 1994, some progress has been made in our understanding of pressure ulcer care. Nursing research is needed to address many gaps in our understanding of pressure ulcer prevention and treatment. Many risk factors for pressure ulcer development have been identified; however, a hierarchy of risk factors has not been determined. Thus, research to determine the essential risk factors is still needed. There also remains a dearth of research determining the role that race and ethnicity may have on pressure ulcer development. A small body of research is emerging to suggest that people of color may have an increased risk for pressure ulcer development. Thus, nurses must actively recruit minority participants to further explore this important variable. Another promising area of nursing research is the use of pressure ulcer prediction tools. Although the Braden Scale was originally published nearly two decades ago, it remains the gold standard. As the patient population continues to change, nursing research is needed to develop and validate newer pressure ulcer prediction tools.

There is a paucity of research on the effects of good skin care on pressure ulcer development. Randomized clinical trials are needed to validate specifics aspects of skin care (bathing schedules, cleansing solutions, water temperature, etc.) and their association with pressure ulcer development. Nursing research can also play a major role in closing the knowledge gap regarding optimal turning/repositioning schedules. Emerging research suggests that turning/repositioning every 2 hours may not be necessary when using dynamic support surfaces. However, randomized controlled trials with large numbers of participants are greatly needed. Evidence is still unclear as to whether there are large differences in the effectiveness of various support surfaces (e.g., Group II) to prevent pressure ulcers.

The role of protein-calorie malnutrition and pressure ulcer development remains understudied. Moreover, research into dietary supplements (vitamins, minerals, etc.) in the absence of a dietary deficiency is lacking. Additional nursing studies are needed to investigate whether the use of dietary supplements have any effect on pressure ulcer prevention. Recent nursing studies suggested that a comprehensive approach to prevention can lead to significant decreases in pressure ulcer incidence. However, studies investigating methods to sustain these decreases in pressure ulcer development are greatly needed. Additional research is also needed to further our understanding of risk level and titration of preventive measures

Staging of pressure ulcers remains more of an art than a science. Additional nursing research is needed to determine effective methods of classifying pressure ulcer depth with good validity and reliability. There is also a dearth of nursing research on the optimal solution and frequency for cleansing a pressure ulcer. Moreover, nursing research is needed to determine the optimal method for removing devitalized tissue in a pressure ulcer. No randomized controlled trials could be found that determined the best debridement method for healing pressure ulcers. Nursing research has identified some clinical characteristics of infected pressure ulcers. However, additional research is needed on the most effective method for treating an infected or contaminated pressure ulcer.

Numerous dressings are currently available to manage wound exudate. However, few randomized controlled trials have been conducted to determine optimal dressings within a classification (e.g., hydrocolloid, alginate). Many adjunctive therapies are currently being used, but few have extensive research to substantiate their effectiveness in healing pressure ulcers. Nursing research investigating the role of skin substitutes, growth factors, negative pressure wound therapy, and electroceuticals in healing pressure ulcers is greatly needed. Finally, nursing

research evaluating the cost effectiveness of adjunctive treatments in healing pressure ulcers is warranted, given rising health care costs.

Conclusion

The prevention of pressure ulcers represents a marker of quality of care. Pressure ulcers are a major nurse-sensitive outcome. Hence, nursing care has a major effect on pressure ulcer development and prevention. Prevention of pressure ulcers often involves the use of low technology, but vigilant care is required to address the most consistently reported risk factors for development of pressure ulcers. The literature suggested that not all pressure ulcers can be prevented, but the use of comprehensive pressure ulcer programs can prevent the majority of pressure ulcers. When the pressure ulcer develops, the goals of healing or preventing deterioration and infection are paramount. Randomized controlled trials are needed to determine optimal management strategies dependent on stage and comorbidities/severity of illness. Nursing remains at the forefront of protecting and safeguarding the patient from pressure ulcers.

Search Strategy

The electronic databases MEDLINE® (1980–2007), CINAHL® (1982–2007), and EI Compedex*Plus (1980–2007) were selected for the searches. Evaluations of previous review articles and seminal studies that were published before 1966 were also included. Research conducted worldwide and published in English between the years 1930 and 2007 was included for review. Moreover, studies using descriptive, correlational, longitudinal, and randomized controlled trials were included.

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References

1. Eckman KL. The prevalence of dermal ulcers among persons in the U.S. who have died. Decubitus 1989;2:36–40.

2. Russo CA, Elixhauser A. Hospitalizations related to pressure sores, 2003, Healthcare Cost and Utilization Project. Rockville, MD: Agency for Healthcare Research and Quality; April 2006. http://www.hcup-us.ahrq.gov/reports/statbriefs/sb3.pdf. Accessed December 19, 2006.

3. U.S. hospital errors continue to rise. http://news.yahoo.com/s/hsn/20070402/hl_hsn/ushospitalerrorscontinuetorise. Accessed March 31, 2007.

4. Nightingale F. Notes on nursing. Philadelphia: Lippincott; 1859.

5. Bliss MR, Thomas JM. A basis for future action: applying clinical findings of trials on pressure-relieving supports to practice: kinetic treatment table versus "normal" bed with two-hourly turning. Prof Nurse 1993;8:726,728,730.

6. Bolton LL, van Rijswijk L, Shaffer FA. Quality wound care equals cost-effective wound care: a clinical model. Adv Skin Wound Care 1997;10(4):33-8.

7. Lyder C, Grady J, Mathur D, et al. Preventing pressure ulcers in Connecticut hospitals using the plan-do-study-act model for quality improvement. Jt Comm J Qual Patient Saf 2004;30:205-14.

8. Panel on the Prediction and Prevention of Pressure Ulcers in Adults. Pressure ulcers in adults: prediction and prevention. Clinical Practice Guideline No. 3. Rockville, MD: Agency for Health Care Policy and Research; 1992. AHCPR Publication No. 92-0047.

9. Campell K, Teague L, Hurd, T, et al. Health policy and the delivery of evidence-based wound care using regional wound teams. Healthc Manage Forum 2006;19(2):16-21.

10. Cuddigan J, Berlowitz DR, Ayello EA. Pressure ulcers in America: prevalence, incidence, and implications for the future. Reston VA: National Pressure Ulcer Advisory Panel; 2001.

11. Langemo DK, Olson B, Hunter S, et al. Incidence of pressure sores in acute care, rehabilitation, extended care, home health, and hospice in one locale. Decubitus 1989;4(3):25-26, 28, 30, passim.

12. Lyder CH, Preston J, Grady J, et al. Quality of care for hospitalized Medicare patients at risk for pressure ulcers. Arch Intern Med 2001;161:1549-54.

13. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. J Am Geriatr Soc 1992;40:747-58.

14. Allman RM, Goode PS, Patrick MM, et al. Pressure ulcer risk factors among hospitalized patients with activity limitations. JAMA 1995;273:865-70.

15. Thomas DR, Goode PS, Tarquine PH, et al. Hospital-acquired pressure ulcers and risk of death. J Am Geriatr Soc 1996;44:1435-40.

16. Oot-Giromini B, Bidwell FC, Heller NB, et al. Pressure ulcer prevention versus treatment, comparative product cost study. Decubitus 1989;2(3):52-4.

17. Landis EM. Micro-injection studies of capillary blood pressure in human skin. Heart 1930;15:209.

18. Kosiak M, Kubicek WG, Olson M, et al. Evaluation of pressure as a factor in the production of ischial ulcers. Arch Phys Med Rehabil 1958;39:623-29.

19. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40(2):62-9.

20. Lyder C, Preston, Ahearn D, et al. Medicare Quality Indicator System: Pressure ulcer prediction and prevention module: final report. Bethesda, MD: Qualidigm/U.S. Health Care Financing Administration; 1998.

21. Bliss MR. Hyperaemia. J Tissue Viability 1998;8:4-13.

22. Allman RM, Laprade CA, Noel LB, et al. Pressure sores among hospitalized patients. Ann Intern Med 1986;105:337-42.

23. Guralnik JM, Harris TB, White LR, et al. Occurrence and predictors of pressure ulcers in the National Health and Nutrition Examination Survey follow-up. J Am Geriatr Soc 1988;36:807-12.

24. Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. J Am Geriatr Soc 1989;37:1043-50.

25. Brandeis GH, Morris JN, Nash DJ, et al. Epidemiology and natural history of pressure ulcers in elderly nursing home residents. JAMA 1990;264:2905-09.

26. Fuhrer M, Garber S, Rintola D, et al. Pressure ulcers in community-resident persons with spinal cord injury: prevalence and risk factors. Arch Phys Med Rehabil 1993;74:1172-77.

27. Spector W, Kapp M, Tucker R,et al. Factors associated with presence of decubitus ulcers at admission to nursing homes. Gerontologist 1988;28:830-34.

28. Lyder C, Yu C, Emerling J, et al. The Braden scale for pressure ulcer risk: evaluating the predictive validity in blacks and Hispanic elderly patients. Appl Nurs Res 1999;12(2):60-8.

29. Fiscella K, Meldrum S, Barnett S, et al. Separate and unequal: hospital racial segregation and disparity in pressure ulceres in NYC. http://www.academyhealth.org/2004/ppt/fiscella2.ppt. Accessed April 2007.

30. Baumgarten M, Margolis D, Gruber-Baldini AL, et al. Pressure ulcers and the transition to long-term care**.** Adv Skin Wound Care 2003;16(6):299-304.

31. Bergstrom N. Braden BJ, Laguzza A. The Braden Scale for predicting pressure sore risk. Nurs Res 1987;36(4):205-10.

32. Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. Rehabil Nurs 1987;2(1):8-12.

33. Maklebust J, Sieggreen MY, Sidor D, et al. Computer-based testing of the Braden Scale for predicting pressure sore risk. Ostomy Wound Manage 2005;51(4):40-52.

34. Norton D. Calculating the risk: reflections of the Norton Scale. Decubitus 1989;2(3):24-31.

35. Bergstrom N, Demuth P, Braden B. A clinical trial of the Braden scale for predicting pressure sore risk. Nurs Clin North Am 1987;22:417-28.

36. Pang SM, Wong TK. Predicting pressure sore risk with the Norton, Braden, and Waterlow scales in a Hong Kong rehabilitation hospital. Nurs Res 1998;47:147-153.

37. Pancorbo-Hidalgo PL, Garcia-Fernandez FP, Lopez-Medina IM, et al. Risk assessment scales for pressure ulcer prevention: a systematic review. J Adv Nurs 2006;54(1):94-110.

38. Perneger T, Rae A, Gaspoz J, et al. Screening for pressure ulcer risk in an acute care hospital: development of a brief beside scale. J Clin Epidemiol 2002;55:498-504.

39. Schoonhoven L, Haalboom J, Bousema M, et al. Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. Br Med J 2002;25(797):1-11.

40. Centers for Medicare & Medicaid Services. Tag F-314 pressure ulcers. (Revised). Guidance for surveyors in long term care. Issued Nov 12, 2004. http://www.new.cms.hhs.gov/manuals/download/som107ap_pp_guidelines_ltcf.pdf. Accessed December 13 2006.

41. Teigland C, Gardiner R, Li H, et al. Clinical informatics and its usefulness for assessing risk and preventing falls and pressure ulcers in nursing home environments. In: Advances in Patient Safety: From Research to Implementation. Vol. 3. http://www.ahrq.gov/downloads/pub/advances/vol3/Teigland.pdf Accessed April, 2007.

42. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. J Am Geriatr Soc 1992;40(8):747-58.

43. Bergstrom N, Braden B, Kemp M. et al. Predicting pressure ulcer risk: a multisite study of the predictive validity of the Braden Scale. Nurs Res 1998;47(5):261-69.

44. IHI.ORG. 5 million lives campaign. http://www.ihi.org/IHI/Programs/Campaign/. Accessed December 23, 2006.

45. Ayello EA, Braden B. How and why to do pressure ulcer risk assessment. Adv Skin Wound Care 2002;15(3):125-32.

46. Johnson-Pawlson J, Infeld DL. Nurse staffing and quality of care in nursing facilities. J Gerontol Nurs 1996;22(8):36-45.

47. Horn S, Buerhaus P, Bergstrom N, et al. RN staffing time and outcomes of long stay nursing home residents: pressure ulcers and other adverse outcomes are less likely as RNs spend more time on direct patient care. Am J Nurs 2005;105:58-70.

48. Donaldson N, Bolton LB, Aydin C, et al. Impact of California's licensed nurse-patient ratios on unit-level nurse staffing and patient outcomes. Policy Polit Nurs Pract 2005;6(3):198-210.

49. Xakellis GC, Frantz RA, Lewis A, et al. Cost-effectiveness of an intensive pressure ulcer prevention protocol in long term care. Adv Wound Care 1998;11:22-9.

50. Xakellis GC, Frantz RA, Lewis A, et al. Translating pressure ulcer guidelines into practice: it's harder than it sounds. Adv Skin Wound Care 2001;14(5):249-56, 258.

51. Lyder C, Shannon R, Empleo-Frazier O, et al. A comprehensive program to prevent pressure ulcers: exploring cost and outcomes. Ostomy Wound Manage 2002;48:52-62.

52. Gunningberg L, Linddholm C, Carlsson M, et al. Reduced incidence of pressure ulcers in patients with hip fractures: a 2-year follow-up of quality indicators. Int J Qual Health Care 2001;13(5):399-407.

53. Baier R, Gifford D, Lyder C, et al. Quality improvement for pressure ulcer care in the nursing home setting: the northeast pressure ulcer project. J Am Med Dir Assoc 2003;4:291-301.

54. Thompson P, Langemo D, Anderson J, et al. Skin care protcols for pressure ulcers and incontinence in long-term care: a quasi-experimental study. Adv Skin Wound Care 2005;18:422-9.

55. Torra I, Bou JE, Segovia Gomez T, et al. The effectivenss of a hyperoxygenated fatty acid compound in preventing pressure ulcers. J Wound Care 2005;14:117-21.

56. van der Cammen TJ, O'Callaghan U, Whitefield M. Prevention of pressure sores: a comparison of new and old pressure sore treamtnets. Br J Clin Pract 1987;41:1009-11.

57. Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of the current literature. Ostomy Wound Manage 1999;45:80S-85S.

58. Ek AC, Gustavsson G, Lewis DH. The local skin blood flow in areas at risk for pressure sores treated with massage. Scand J Rehabil Med 1985;17(2):81-6.

59. Norton D, McLaren R, Exton-Smith A. An investigation of geriatric nurse problems in hospitals. Edinburgh UK: Churchill Livingston; 1975.

60. Krishnagopalan S, Johnson EW, Low LL, et al. Body positioning of intensive care patients: Clinical practice versus standards. Crit Care Med 2002;30:2588-92.

61. DeFloor T, De Bacquer D, Grypdonck M. The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. Int J Nurs Stud 2005;42(1):37-46.

62. Reddy M, Gill SS, Rochon PA. Preventing pressure ulcers: a systematic review. JAMA 2006;296:974-84.

63. Seiler WO, Stahelm HB. Influence of the 30 degrees laterally inclined position and the 'super-soft' 3-piece mattress on skin oxygen tension on areas of maximum pressure—implications for pressure sore prevention. Gerontology 1986;32(3):158-66.

64. Young T. The 30 degree tilt position vs the 90 degree lateral and supine positions in reducing the incidence of non-blanching erythema in a hospital inpatient population: a randomized controlled trial. J Tissue Viability 2004;14:88-96.

65. Seiler WO, Stahelm HB. Decubitus ulcer: preventive techniques for the elderly patient. Geriatrics 1985;40(7):53-60.

66. National Pressure Ulcer Advisory Panel Support Surface Standards Initiative. http://www.npuap.org/NPUAP_S3I_TD.pdf Accessed January, 2008.

67. Agostini JV, Baker DI, Bogardus ST. Prevention of Pressure Ulcers in Older Patients. In: Making Health Care Safer: A Critical Analysis of Patient Safety Practices. Evidence Report/Technology Assessment, No. 43 Chapter 27.(Prepared by University of California at San Francisco-Stanford University Evidence-based Practice Center under Contract No. 290-97-0013). Rockville MD: Agency for Healthcare Research and Quality, July 2001. AHRQ Publication No. 01-E058. http://www.ahrq.gov/clinic/ptsafety/chap27.htm. Accessed November, 2006.

68. Ooka M, Kemp MG, Shott S. Evaluation of three types of support surfaces for preventing pressure ulcers in patients in a surgical intensive care unit. J Wound Ostomy Continence Nurs 1995;22:271-9.

69. Centers for Medicare & Medicaid Services. CMS Manual System, Pub. 100-07: State operations, provider certification. Department of Health and Human Services; 2004.

70. Anthony D, Reynolds T, Russell L. An investigation into the use of serum albumin in pressure sore prediction. J Adv Nurs 2000;32:359-65.

71. Thomas DR. Improving outcome of pressure ulcers with nutritional interventions: a review of the evidence. Nutrition 2001;17(2):121-25.

72. Pinchcofsky-Devin GD, Kaminsk MV. Correlation of pressure sores and nutritional status. J Am Geriatr Soc 1986;34(6):435-40.

73. Bourdel-Marchasson L, Barateau M, Rondeau V, et al. GAGE Group. A multi-center trail of the effects of oral nutritional supplementation in critically ill older inpateients. Nutrition 2000;16:1-5.

74. Matthus-Vliegen E. Old age, malnutrition, and pressure sores: an ill-fated alliance. J Gerontol A Biol Sci Med Sci 2004;59:355-60.

75. Panel on the Prediction and Prevention of Pressure Ulcers in Adults. Treatment of pressure ulcers. Clinical Practice Guideline No. 15. Rockville, MD: Agency for Health Care Policy and Research; 1994. AHCPR Publication No. 92-0652.

76. Rodeheaver G. Controversies in topical wound management. Ostomy Wound Manage 1988;58-68.

77. Moore ZE, Cowman S. Wound cleansing for pressure ulcers (Review). The Cochrane Collaboration 2007;1:1-16.

78. Barr JE. Principles of wound cleansing. Ostomy Wound Manage 1995;41:7A.

79. Lam DG, Rastomjee D, Dynan Y. Wound irigation: a simple, reproducible device. Ann R Coll Surg Engl 2000;82(5):346-7.

80. Hellwell TB, Major PA, Foresman PA, et al. A cytotoxicity evaluation of antimicrobial wound cleansers. Wounds 1997;9:15-20.

81. Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: a systemic approach to wound management. Wound Repair and Regen 2003;11:1-28.

82. Ayello EA, Baranoski S, Salati, D. A survey of nurses' wound care knowledge. Adv Skin Wound Care 2005;18(5):268-75.

83. European Pressure Ulcer Advisory Panel. The EPUAP Guide To Pressure Ulcer Grading http://www.epuap.org/grading.html Accessed April, 2007.

84. The National Pressure Ulcer Advisory Panel. http://www.npuap.org/pr2.htm Accessed January , 2008.

85. Shea JD. Pressure sores: classification and management. Clin Orthop Relat Res 1975;112:89-100.

86. Minimum Data Set (MDS)—Version 2.0 for nursing home resident assessment and care screening http://www.cms.hhs.gov/NursingHomeQualityInits/downloads/MDS20MDSAllforms.pdf. Accessed April, 2007.

87. National Pressure Ulcer Advisory Panel. Updated staging system. http://www.npuap.org/pr2.htm. Accessed April, 2007.

88. Rosen J, Mittal V, Degenholtz H, et al. Organizational change and quality improvement in nursing homes: approaching success. J Healthc Qual 2005;27(6): 6-14, 21, 44, (41 ref).

89. Dyson M. Lyder C. Wound management—physical dalities. In: Morsion, M. ed. The prevention and treatment of pressure ulcers. Edinburgh, UK: Harcourt Brace/Mosby International. P. 177-94.

90. Quintavalle PR, Lyder CH, Mertz PJ, et al. Use of high-resolution, high-frequency diagnostic ultrasound to investigate the pathogenesis of pressure ulcer development. Adv Skin Wound Care 2006;19(9):498-505.

91. Robson MC, Heggers JP. Bacterial quantification of open wounds. Mil Med 1969;134(1):19-24.

92. Falanga V. Classification of wound bed preparation and stimulation of chronic wounds. Wound Repair Regen 2000;8:347-52.

93. Falanga V. Wound bed preparation and the role of enzymes: a case for multiple actions of therapeutic agents. Wounds: A Compendium of Clinical Research and Practice 2002;14(2):47-57.

94. Falabella A. Dedridement of wounds. Wounds: A Compendium of Clinical Research and Pratice 1998;10(Suplement C):1C-9C.

95. Leaper D. Sharp technique for wound debridement. World Wide Wounds 2002;5.

96. O'Brien, M. Exploring methods of wound debridement. Br J Community Nurs 2002; Dec: 10-8.

97. Barr JE, Day AL, Weaver VA, et al. Assessing clinical efficacy of a hydrocolloid/alginate dressing on full-thickness pressure ulcers. Ostomy Wound Manage 1995;41(3):28-30, 32, 34-6 passim.

98. Sinclair RD, Ryan TJ. Proteolytic enzymes in wound healing: the role of enzymatic debridement. Australas J Dermatol 1994;35(1):35-41.

99. Wollina U, Liebold K, Schmidt, et al. Biosurgery supports granulation and debridement in chronic wounds—clinical data and remittance spectroscopy measurement. Int J Dermatol 2002;41(10):635-9.

100. Dow G, Browne A, Sibbald RG. Infection in chronic wounds: controversies in diagnosis and treatment. Ostomy Wound Manage 1999;45(8):23-40.

101. Robson MC, Heggers JP. Bacterial quantification of open wounds. Mil Med 1969;134(1):19-24.

102. Robson MC. Lessons gleaned from the sport of wound watching. Wound Repair Regen 1999;7(1):2-5.

103. Heggers JP. Defining infection in chronic wounds: Does it matter? J Wound Care 1998;7(8):452-6.

104. Bergin SM, Wraight P. Silver based wound dressings and topical agents for treating diabetic foot ulcers. Cochrane Database Syst Rev 2006;1:CD005082.

105. Cutting KF, Cardiff KG. Criteria for identifing wound infection. J Wound Care 1994;3(4):198-201.

106. Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized chronic wound infection. Wound Repair Regen 2001;9:178-86.

107. Romanelli M, Magliaro A, Mastronicola D, et al. Systemic antimicrobial therapies for pressure ulcers. Ostomy Wound Manage 2003;49(5A Suppl):25-9.

108. Meaume S, Vallet D, Morere MN, et al. Evaluation of a silver-releasing hydroalginate dressing in chronic wounds with signs of local infection**.** J Wound Care 2005;14:411-9.

109. Thomas S, McCubbin P. A comparison of the antimicrobial effects of four silver-containing dressings on three organisms. J Wound Care 2003;12(3):101-7.

110. Vermeulen H, van Hattem JM, Storm-Versloot MN, et al. Topical silver for treating infected wounds (Review). The Cochrane Collaboration 2007;1:1-36.

111. McKenna P, Lehr GS, Leist P, et al. Antiseptic effectiveness with fibroblast preservation. Ann Plastic Surg 1991;27:265-68.

112. Lineaweaver W, Howard R. Topical antimicrobial toxcity. Arch Surg 1985;120:267-70.

113. Ovington L. Dressings and adjunctive therapies: AHCPR guidelines revisited. Ostomy Wound Manage 1999;45:94S-106S.

114. Colwell J, Foreman MD, Trotter JP. A comparision of the efficacy and cost-effectiveness of two methods of managing pressure ulcers. Decubitus 1993;6(4):28-36.

115. Kerstein MD, Gemmen E, van Rijswijk L et al. Cost and cost effectiveness of venous and pressure ulcer protocols of care. Dis Manage Health Outcomes 2001;46(8):651-63.

116. Xakellis G, Chrischilles EA. Hydrocolloid versus saline-gauze dressings in treating pressure ulcers: a cost effective analysis. Arch Phys Med Rehabil 1992;73:463-69.

117. Bolton L, McNees P, van Rijswijk L, et al. Wound healing outcomes using standardized assessment and care in clinical practice. J Wound Ostomy Continence Nurs 2004;31(2):65-71.

118. Chernoff RS, Milton KY, Lipschitz DA. The effect of a very high-protein liquid formula on decubitus ulcer healing in long term tube-fed institutionalized patients. J Am Geriatr Soc1990;90:A-130.

119. Mulholland JH, Tui C, Wright AM, et al. Protein metabolism and bedsores. Ann Surg 1943;118:1015-23.

120. Breslow RA, Hallfrisch J, Guy DG, et al. The importance of dietary protein in healing pressure ulcers. J Am Geriatr Soc1993;41:357.

121. Posthauer ME. The role of nutrition in wound care. Adv Skin Wound Care 2005;19:43-52.

122. Quirono J, Santos VL, Quednau TJ, et al. Pain in pressure ulcers. Wounds: A Compedium of Clinical Research and Practice 2003;15:381-89.

123. Roth RS, Lowery J, Hamill J. Assessing persistent pain and its relation to affective distress, depressive symptoms and pain catstrophizing in patients with chronic wounds: a pilot study. Am J Phys Med Rehabil 2004;83:827-34.

124. World Union of Wound Healing Societies. Principles of best practice: minimizing pain at wound dressing-related procedures, a consensus document. http://www.wuwhs.org. Accessed October, 2006.

125. Bartolucci AA, Thomas DR. Using principal component analysis to describe wound status. Adv Wound Care 1997;10(5):93-5.

126. Stotts NA, Rodeheaver GT, Thomas DR, et al. An instrument to measure healing in pressure ulcers: development and validation of the Pressure Ulcer Scale for Healing (PUSH). J Gerontol A Biol Sci Med Sci 2001;56A(12): M795-9.

127. Bates-Jensen BM, Vredevoe DL, Brecht M. Validity and reliability of the pressure sore status tool. Decubitus 1992;5(6):20-8.

128. Bates-Jensen BM. The Pressure Sore Status Tool a few thousand assessments later.
Adv Wound Care. 1997 Sep;10(5):65-73.

129. Kloth L, Feeder J. Acceleration of wound healing with high voltage, monophasic, pulsed current. Phys Ther 1988;68:503-8.

130. Kloth LC, McCulloch J. Promotion of wound healing with electrical stimulation. Adv Wound Care 1996;9:42-5.

131. Gardner SE, Frantz RA, Schmidt FL. Effect of electrical stimulation on chronic wound healing: a meta-analysis. Wound Repair Regen 1999;7:495-503.

132. Gupta S, Baharestani M, Baranoski S, et al. Guidelines for managing pressure ulcers with negative pressure wound therapy. Adv Skin Wound Care 2004;17(supplement):1-16.

133. Joseph E, Hamori C, Bergman, et al. A prospective randomized trial of vacuum-assisted closure versus standard therapy of chronic nonhealing wounds. Wounds: A Compendium of Clinical Research and Practice 2000;12:60-7.

134. Mendez-Eastman S. Determining the appropriateness of negative pressure wound therapy for pressure ulcers. Ostomy Wound Manage 2004;50(4A Supple):13-6.

135. Payne WG, Ochs DE, Meltzer DD, et al. Long-term outcome study of growth factor-treated pressure ulcers. Am J Surg 2001;181(1):81-6.

136. Landi F, Aloe L, Russo A, et al. Topical treatment of pressure ulcers with nerve growth factor: a randomized clinical trial. Ann Intern Med 2003;139(8):635-41.

137. Thomas DR. The promise of topical growth factors in healing pressure ulcers. Ann Intern Med 2003;139(8):694-95.

138. Strauch B, Patel MK, Rosen DJ, et al. Pulsed magnetic field therapy increases tensile strength in a rat Achilles' tendon repair model. J Hand Surg [Am] 2006;31(7):1131-5.

139. Babi-Akbari SA, Flemming K, Cullum NA, et al. Theraputic ultrasound for pressure ulcers (Review). The Cochrane Collaboration 2007;1:1-12.

Evidence Table. Pressure Ulcers—Risk, Assessment, and Prevention

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
Allman 1986[22]	Pressure ulcer risk factors	Prospective cohort study	Cross-sectional, pressure ulcer development	Hospital, 21 years and older		Hypoalbuminemia, fecal incontinence, and fractures remained significantly and independently associated with having a pressure sore (odds ratios = 3.0, 3.1, and 5.2, respectively).
Allman 1995[14]	Pressure ulcer risk identification	Prospective cohort study	Prospective cohort study, time to in-hospital development of a Stage II or greater pressure ulcer	Urban teaching hospital		Age of 75 years or more, dry skin, nonblanchable erythema (a Stage I pressure ulcer), previous pressure ulcer history, immobility, fecal incontinence, depleted triceps skin fold, lymphopenia (lymphocyte count < 1.50 x 10(9)/L), and decreased body weight (< 58 kg) were significantly associated with pressure ulcer development by univariate Kaplan-Meier survival analyses ($P < 0.05$ by log-rank test).
Anthony 2000[70]	Pressure ulcer prediction	Prospective cohort study	Noncomparative study, pressure ulcer development	Skilled nursing facility, elderly		Serum albumin (low) can be a useful predictor of pressure ulcer development.
Baier 2003[53]	Pressure ulcer prevention	Quality improvement	Prospective cohort study, implementation of AHRQ guidelines and pressure ulcer development	Skilled nursing facilities, quality improvement teams in 29 nursing homes	Quality improvement teams	Six of eight prevention process measures improved significantly, with percent difference between baseline and followup ranging from 11.6% to 24.5%. Three of four treatment process measures improved significantly, with 5.0%, 8.9%, and 25.9% differences between baseline and followup. For each process measure, between 5 and 12 facilities demonstrated significant improvement between baseline and followup, and only 2 or fewer declined for each process measure.

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
Bates-Jensen 1992[127]	Pressure ulcer healing	Prospective cohort study	Cross-sectional, pressure ulcer healing			The Pressure Sore Status Tool interrater reliability was established at $r = 0.91$ for first observation and $r = 0.92$ for the second observation ($P < 0.001$). Interrater reliability was $r = 0.99$ for rater one and $r = 0.96$ for rater two ($P < 0.001$).
Baumgarten 2003[30]	Pressure ulcer prevalence	Prospective cohort study	Prospective cohort study, pressure ulcer development among newly admitted residents from hospitals, home, or other settings.	Skilled nursing facilities, 65 years and older		Admission from a hospital was significantly associated with pressure ulcer prevalence on admission (OR = 2.2).
Bergstrom 1987[35]	Pressure ulcer prediction	Quality improvement	Prospective cohort study	Acute intensive care unit	The Braden scale score and skin assessment	Twenty-four of 60 consecutively admitted patients developed pressure ulcers with the total score of 16 as the cut-off.
Bergstrom 1992[13]	Pressure ulcer risk identification	Prospective cohort study	Prospective cohort study, pressure ulcer presence or absence	Skilled nursing facility, 65 years and older, 70% female		Best predictors of pressure sore development were the Braden scale score (< 16), diastolic blood pressure, temperature, dietary protein intake, and age.
Bergstrom 1992[42]	Pressure ulcer prevention guidelines	Published guideline	Systematic literature review (Level 11) and consensus reports (Level 16), pressure ulcer prevention	Hospital, skilled nursing facilities, and home care, elderly population		Development of guidelines to prevent pressure ulcers.
Bergstrom 1998[43]	Pressure ulcer prediction	Quality improvement	Prospective cohort study	Tertiary care hospitals, Veterans Administration Medical Centers, and skilled nursing facilities	The Braden scale score and skin assessment	One hundred eight of 843 subjects (12.8%) developed pressure ulcers. Braden scale scores were significantly lower ($P = 0.0001$) in those who acquired pressure ulcers then those who did not. Total score of 18 is the cut-off score for prediction of pressure ulcers.
Berlowitz 1989[24]	Pressure ulcer	Prospective cohort	Cross-sectional,	Skilled nursing		Factors associated with pressure

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
	predictors	study	pressure ulcer development	facility, elderly		ulcer development included altered level of consciousness (OR = 4.1), bed- or chair-bound (OR = 1.9), and hypoalbuminemia (OR = 1.8).
Brandeis 1990[25]	Pressure ulcers and mortality	Retrospective cohort studies	Use of large database, pressure ulcer development and mortality, hospitalization	Skilled nursing facilities, elderly		Pressure ulcers were associated with an increased rate of mortality, but not associated with increased transfers from skilled nursing facilities to hospitals for treatment.
Breslow 1993[120]	Pressure ulcers and dietary protein	Prospective cohort study	Nonrandomized trial, pressure ulcer healing	Skilled nursing facility, patients ages 72 years and older with malnutrition	Dietary supplement	Significant truncal decrease in pressure ulcers sizes when using 24% protein.
Cuddigan 2001[10]	Pressure ulcer incidence	Systematic literature review	Systematic literature review (Level 11), pressure ulcer incidence and prevalence	Hospital, skilled nursing facilities, and home care		Pressure ulcer incidence rates (e.g., hospitals, 0.4% to 38%; skilled nursing facilities, 2.2% to 23.9%; and home care, 0% to 17%).
DeFloor 2004[61]	Pressure ulcers	Prospective cohort study	Randomized controlled trial, pressure ulcer development	Skilled nursing facilities, 60 years and older	Turning every 2, 3, 4, or 6 hours using either standard mattress or viscoelastic foam	The incidence of Stage I pressure ulcers was not different between the groups. However, the incidence of Stage II pressure ulcers and higher in the 4-hour turning group with viscoelastic was 3%, compared with incidence figures in the other groups varying between 14.3% and 24.1%.
Donaldson 2005[48]	Licensed nurse-patient ratios and pressure ulcer development	Retrospective	Cross-sectional, pressure ulcer development	Hospitals, adult, surgical, and definitive-observation units, nurse-patient ratios	Staffing ratios	Impact of mandated nurse-patient ratios did not reveal significant changes in incidence of pressure ulcer development.
Ek 1985[58]	Pressure ulcers and massage	Prospective cohort study	Nonrandomized trial, pressure ulcer development, pressure ulcer development using massage	Hospital, patients older than 60 years with and without cerebral hemorrhage	Massage	The effect of massage over areas at risk for pressure ulcer varies greatly between patients and within patients.

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
Fuhrer 1993[26]	Pressure ulcer development	Prospective cohort study	Prospective cohort study, pressure ulcer development	Community, patients with spinal cord injury (100 men and 40 women)		Thirty-three percent (n = 46) presented with one or more ulcers of at least Stage I severity when visually examined. Twenty-one individuals had more than one ulcer, the maximum number of ulcers being seven. Of 87 ulcers for which severity ratings were available, 30 (34.5%) were Stage I, 33 (37.9%) were Stage II, and 24 (27.6%) were either Stage III or IV. Individuals with an ulcer exhibited more paralysis and were more dependent on others in activities of daily living. A greater proportion of blacks had more severe ulcers (Stages III and IV) than their white counterparts.
Gardner 1999[131]	Pressure ulcers	Meta-analysis	Nonrandomized trial, pressure ulcer healing		Electrical stimulation	Rate of healing per week was 22% for electrical stimulation samples and 9% for control samples. The net effect of electrical stimulation was 13% per week, an increase of 144% over the control rate.
Guralnik 1988[23]	Pressure ulcer predictors	Retrospective cohort studies	Use of large database, pressure ulcer development	Skilled nursing facilities, 55 years and older		Pressure ulcer development was associated with current smokers, inactivity, poor self-assessed health status, and anemia.
Hellwell 1997[80]	Cytotoxicity evaluation	Prospective cohort study		Polymorphonuclear leukocytes (PMNs)	Ten commercial wound cleaners	The non-antimicrobial wound cleansers had toxicity indexes of 10 to 1,000, while the toxicity indexes of antimicrobial wound cleansers were 10,000.
Horn 2005[47]	Nurse-patient ratios and pressure ulcer development	Retrospective	Cross-sectional, pressure ulcer development	Skilled nursing facility, registered nurse-patient ratios	Staffing ratios	More registered nurse care time per resident was associated with the development of fewer pressure ulcers.
Johnson-Pawlson 1996[46]	Nurse-patient ratios and pressure ulcer development	Retrospective	Cross-sectional, pressure ulcer development	Skilled nursing facility, registered nurse patient ratios	Registered nurse staffing ratios	The ratio of registered nurse to residents is directly related to a measure of quality of care deficiencies.

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
Kloth 1988[129]	Pressure ulcer healing	Prospective cohort study	Randomized controlled trial, pressure ulcer healing	Skilled nursing facility, patients ages 20 to 89 years, Stage IV pressure ulcers	High voltage monophasic pulsed current	Patients in treatment group healed at a mean rate of 44.8% a week and healed 100% over a mean period of 7.3 weeks. Patients in the control group increased in area an average of 11.6% a week and increased 28.9% over mean period of 7.4 weeks.
Lyder 1998[20]	Pressure ulcer prediction and prevention	Unpublished research	Retrospective cohort study (Level 9), pressure ulcer development	Hospitals, Medicare beneficiaries		The number of risk factors is associated with pressure ulcer development ($P \leq 0.001$). Patients with ≥ three risk factors were associated with an increased incidence (26.3%) of pressure ulcers, in comparison to those with one or two factors ($P = 0.001$).
Lyder 2001[12]	Pressure ulcer prevention	Quality improvement research	Retrospective cohort study (Level 9), pressure ulcer incidence	Hospital, Medicare beneficiaries	AHRQ pressure ulcer prevention guidelines	Hospital compliance with AHRQ prevention guidelines varied greatly for daily skin assessment (94%), risk identification (22.6%), use of pressure-reducing devices (7.5%), nutritional consult (34.3%), and repositioning patient every 2 hours (66.2%).
Lyder 2002[51]	Pressure ulcer prevention	Retrospective and prospective quasi-experimental	Pressure ulcer prevention program	Two long-term care facilities (A = 150 beds, B = 110 beds)	Pressure ulcer prevention program	An 87% decrease in pressure ulcers in facility A (13.2% to 1.7%) and a 76% decrease in pressure ulcers in facility B (15% to 3.5%).
Lyder 2004[7]	Pressure ulcer prevention	Quality improvement research	Retrospective cohort study (Level 9), pressure ulcer development	Hospitals, Medicare beneficiaries	Implement systematic risk assessment, repositioning, support surfaces	Statistically significant increases in the identification of high-risk patients, repositioning of bed-bound or chair-bound patients, nutritional consults in malnourished patients, and staging of acquired Stage II pressure ulcers from baseline and followup medical record abstractions.
Meaume 2005[108]	Silver in chronic wounds	Prospective cohort study	Randomized (stratification according to	13 centers with 99 participants	Silver-releasing hydroalginate	The study suggests that treating wounds with a high risk of infection with silver-releasing hydroalginate

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
			wound type) opened label multicenter comparative two-arm parallel-group		dressing	dressing had a favorable influence on wound prognosis.
Ooka 1995[68]	Support surfaces	Prospective cohort study	A new-product evaluation with convenience sampling	Surgical intensive care unit	Dynamic and static mattresses	All three mattresses were comparable in effectiveness.
Pang 1998[36]	Pressure ulcer risk identification	Prospective cohort study	Prospective cohort study, validity of pressure ulcer prediction scales	Hospital, 21 years and older, pressure ulcer free	Pressure ulcer prediction scales	Both the Norton and Waterlow scales had relatively high sensitivity (81% and 95%, respectively), whereas the Braden Scale had both high sensitivity (91%) and specificity (62%). All three scales had relatively high negative predictive values (>90%), but the Braden Scale had better positive predictive value.
Perneger 2002[38]	Pressure ulcers	Prospective cohort study	Cross-sectional, pressure ulcer development	Teaching hospital, patients older than 60 years		The FRAGMMENT score (sum of friction, age, mobility, mental status) was linearly related to pressure ulcer risk, and its area under the receiver operating characteristic curve (0.80) was higher than for the Norton (0.74; $P = 0.006$) and Braden (0.74; $P = 0.004$) scores.
Reddy 2006[62]	Pressure ulcer prevention	Systematic literature review, pressure ulcers	Systematic literature review	59 randomized controlled trials grouped into three categories		Giving current evidence, use of support surfaces, repositioning, optimizing nutritional status, and hydration of sacral skin are appropriate.
Romanelli 2003[107]	Pressure ulcer infection	Systematic literature review	Systematic literature review			Use of systemic antibiotics in infected pressure ulcers should be based on culture results. Therapy should be specific to isolated pathogens to avoid widespread use of antimicrobial drugs that contribute to the proliferation of drug-resistant organisms.

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
Rosen 2005[88]	Pressure ulcer prevention	Quality improvement	Prospective cohort study, pressure ulcer development	Skilled nursing facilities, elderly	Staff empowerment, real-time feedback	Empowering staff with real-time feedback led to significant reduction of new pressure ulcers ($P = 0.05$).
Roth 2004[123]	Pressure ulcer and pain	Prospective cohort study	Cross-sectional, pressure ulcer pain			McGill Pain questionnaire was more sensitive to pain experience than a single rating of pain intensity. Moreover, wound stage (larger) was positively related to severity of pain, and pain catastrophizing was positively related to pain intensity.
Schoonhoven 2002[39]	Pressure ulcer risk	Prospective cohort study	Prospective cohort study, pressure ulcer development	Hospitals, patients admitted to surgical, internal, neurological, or geriatric units, 18 years and older		The weekly incidence of patients with pressure ulcers was 6.2% (95% confidence interval 5.2% to 7.2%). The area under the receiver operating characteristic curve was 0.56 (0.51 to 0.61) for the Norton scale, 0.55 (0.49 to 0.60) for the Braden scale, and 0.61 (0.56 to 0.66) for the Waterlow scale; the areas for the subpopulation, excluding patients who received preventive measures without developing pressure ulcers and excluding surgical patients, were 0.71 (0.65 to 0.77), 0.71 (0.64 to 0.78), and 0.68 (0.61 to 0.74), respectively. In this subpopulation, using the recommended cut-off points, the positive predictive value was 7.0% for the Norton, 7.8% for the Braden, and 5.3% for the Waterlow scales.
Stotts 2001[126]	Pressure ulcer healing	Retrospective	Cross-sectional, pressure ulcer healing	Skilled nursing facility		The Pressure Ulcer Scale for Healing tool accounted for 58% to 74% of the wound healing variance over a 10-week period in Study 1, and 40% to 57% of the wound healing variance over a 12-week period in Study 2. Thus the PUSH

Source	Safety Issue Related to Clinical Practice	Design Type	Study Design & Study Outcome Measure(s)	Study Setting & Study Population	Study Intervention	Key Finding(s)
						tool is a valid and sensitive measure of pressure ulcer healing.
Thomas 1996[15]	Pressure ulcers and mortality	Prospective cohort study	Prospective cohort study, time to death from admission to 1-year posthospital discharge.	Urban teaching hospital		Development of an in-hospital pressure ulcer was associated with greater risk of death at 1 year (59.5% versus 38.2%, *P* = 0.02). However, pressure ulcer development did not remain independently associated with decreased survival after adjusting for other predictors of mortality.
Torra 2005[55]	Hyperoxygenated fatty acid	Prospective cohort study	Double-blind randomized clinical trial	Multicenter	Hyper-oxygenated fatty acid preparation	Pressure ulcer incidence during the study was 7.32% in the intervention group versus 17.37% in the placebo group (*P* = 0.006).
Xakellis 1998[49]	Pressure ulcer prevention	Quality improvement research	Cost-effectiveness evaluation	77-bed long-term care facility	A guideline-based pressure ulcer prevention protocol	Pre-protocol: 16 out of 69 patients developed 26 pressure ulcers. Post-protocol: 3 out of 63 patients developed 5 pressure ulcers.
Xakellis 2001[50]	Pressure ulcer guidelines	Retrospective and prospective quasi-experimental longitudinal	Cost effectiveness of a guideline-based pressure ulcer prevention protocol over time	77-bed long-term care facility	A guideline-based pressure ulcer prevention protocol	Time to ulcer development varied among three groups (log rank = 8.81, *P* = 0.01). Time to ulcer healing (log rank = 9.49, *P* = < 0.01). Cost of treatment decreased (F = 5.5, *P* = < 0.01). Cost of prevention increased (F = 15, *P* = < 0.01).