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SKIN INTEGRITY

The development and worsening of pressure ulcers comprise one of thirteen indicators of quality of care identified by the Joint Commission for the Accreditation of Health Care Organizations (1987). A recent consensus conference sponsored by the National Pressure Ulcer Advisory Panel (NPUAP) (1989) was convened to delineate the prevalence, cost, and status of risk assessment of this problem. The work of the Consensus Panel and its recommendations will be considered throughout this discussion.

State of the Science

Incidence/Prevalence of the Problem

The incidence (new cases appearing during a specified period) and prevalence (a cross-sectional count of the number of cases at a specific time) of pressure sores in acute-care hospitals, long-term care facilities, and during home care is somewhat difficult to determine due to several methodological barriers that prevent generalization from available data. Methodological limitations to the interpretation of incidence and prevalence studies were broadly categorized by the Consensus Panel as: 1) difficulty comparing the various populations (data collected in tertiary-care hospitals are not likely to reflect community hospital populations); 2) variations in the sources of data, ranging from direct observation of patients by trained research personnel to retrieval of data from patient records; and 3) study methods that confuse incidence and prevalence, include or exclude Stage I sores, and include or exclude segments of the institutional population.

A major problem in data interpretation is the system used to classify pressure sore stages. The debate regarding the appropriateness of including Stage I pressure sores in the calculation of incidence and prevalence was addressed by the Consensus Panel. Stage I pressure ulcers were included in the recommended classification system, recognizing the importance of early identification and treatment of ulcers in the prevention and worsening of pressure sores. The following classification system was recommended by the NPUAP.

Stage I: Nonblanchable erythema of intact skin; the heralding of skin ulceration.

Stage II: Partial thickness skin loss involving epidermis and/or dermis; the ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

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.

Stage IV: Full thickness skin loss with extensive destruction; tissue necrosis or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule).

Clinically, wounds are further described in relation to surface size, depth, location, and descriptors reflecting infected versus healing tissue. The classification and description of pressure ulcers are important to incidence and prevalence studies, but also are equally important to studies comparing prevention and treatment measures. Achieving reliability among observers has been a major concern of one group conducting multi-site studies (Bergstrom, Braden, Champagne, & Kemp, 1988-91). An instrument detailing pressure sore sites to facilitate systematic assessment and staging is recommended. An example of one instrument is presented in Figure 1. (p. 63). Use of a videotape that describes pressure ulcers and provides instruction on staging and measurement, using a staging system virtually identical to that recommended by the NPUAP, has resulted in high interrater reliability (r > 0.90) (Kemp & Bergstrom, 1988). It is important to note that training in systematic skin assessment is essential to achievement of successful results. A more precise measure of the area of an ulcer using stereophotography has been devised by Frantz (1989). This highly sophisticated technique is more appropriate for monitoring advanced pressure sores undergoing treatment than for studies that screen for incidence and prevalence because the method requires a high level of skill in photography and interpretation, and costly equipment.

A number of studies of incidence and prevalence of pressure sores describing the sample population and number, the staging system, and methods used to obtain data have been conducted (Brandis, G.H., Morris, J.N., Nash, D.J. & Lipsitz, L.A., 1990; Kemp, M.G., Keithly, J.K., Smith, D.W. & Morreate, B., 1990; Kennedy, K.L., 1989; Oot-Giromini, B., et al 1989; Raney, J.P., 1989; Shannon, M.L. & Skorga, P., 1989; Clark, M. & Kadhom, J., 1988; Stotts, N.A., 1988; Jensen, T.T. & Junckner, Y., 1987; Nyquist, R. & Hawthorn, P.J., 1987; Pajk, M., Craven, G.A., Cameron-Barry, J., Shipps, T. & Bennum, N.W., 1986; Abildgaad, U & Daugaard, K., 1979; Lowthian, P., 1979; Manley, M.T., 1978; Peterson, N.C. & Bittmann, S., 1971). The incidence of pressure ulcers among medical-surgical patients in hospital settings ranged from 2.7 percent (Gerson, 1975) to nine percent (Bergstrom, Braden, Laguzza, & Holman, 1987). Prevalence ranged from 4 percent (Ek & Bowman, 1982) to 14 percent (Langemo et al., 1989). Incidence in special high-risk populations, for example, spinal cord injuries (Richardson & Meyer, 1981) and fractured hips (Versluysen, 1985), ranged from 60 percent to 82 percent, respectively. Among persons in skilled-care/nursing home-type facilities, the prevalence ranged from 12 percent by six months following admission (Brandeis, Morris, Nash, & Lipsitz, 1989) to 28 percent (Langemo et al., 1989; Young, 1989). These data are particularly difficult to generalize to other skilledcare/nursing home facilities due to the highly variable nature of case mix and staffing. One report found that the incidence of pressure ulcers increases with length of stay (Brandeis et al., 1989). The prevalence and incidence of pressure ulcers in these facilities deserve further attention to determine the magnitude and cost of the problem and to project the resources needed to increase the effectiveness of patient care. The prevalence of pressure ulcers among persons who are cared for in home settings with the supervision or assistance of professionals is not understood fully. Barbanel, Jordan, Nicol, and Clark (1977) reported a prevalence of 8.7 percent of Stage II or worse ulcers and Langemo et al. (1989), reporting on a very small sample, noted that 19 percent had ulcers when they were assessed. The magnitude of the problem among home-care patients remains to be studied.

Older persons may be at disproportionate risk for pressure ulcer development. The studies of incidence/prevalence demonstrate a higher incidence/prevalence in settings with higher mean age subjects (average age over 65) (Lofgren, MacPherson, Granieri, Myllenbeck, & Sprafka, 1989; Versluysen, 1986).

In summary, the incidence and prevalence of pressure ulcers is sufficiently high to warrant concern. Methods for determining actual incidence and prevalence are hampered by a lack of studies that are widely generalizable and sufficiently controlled in data acquisition methods and pressure ulcer classification systems. Studies that use large databases suffer from lack of control of the data acquisition skills of the observers and must be balanced with more accurate data

acquisition using smaller samples. The NPUAP (1989) recommended classification system should be endorsed and used in future studies to increase the generalizability of data. A systematic assessment guide detailing pressure sore sites should be used to prevent errors of omission in measurement. Knowledge of the incidence and prevalence of pressure ulcers by stage and by type of health care facility and diagnosis will permit better planning for and allocation of services to vulnerable populations.

Predicting Pressure Sore Risk

Risk Predictor Tools. A number of instruments have been developed over the last 30 years to predict or assess risk for pressure sore development (Andersen, Jensen, Kvorning, & Bach, 1982; Bergstrom et al., 1987; Gosnell, 1973; Norton, McLaren, & Exton-Smith, 1962; Taylor, 1988). The NPUAP did not endorse any one risk assessment tool, but identified factors most consistently described in relation to risk assessment including immobility, inactivity, malnutrition, fecal and urinary incontinence, and decreased level of consciousness. The instruments that have been tested and refined most extensively were recently printed with commentary from the authors in the Proceedings of the Consensus Conference (NPUAP, 1989). These instruments all include the risk factors identified above and are based on observation and scoring of risk factors and a summative score with suggested cut-off points for diagnosing risk. The Agency for Health Care Policy and Research (AHCPR) Panel developing guidelines for the prediction and prevention of pressure ulcers cite the Braden (Bergstron, et al., 1987) and Norton (Norton, 1990) scales as being more valid. Of these two, the Braden Scale enjoys greater interrater reliability.

The NPUAP Consensus Panel (1989) identified the following characteristics of ideal clinical screening tools: predictive validity, as evidenced by high sensitivity and specificity, and ease of use. Taylor (1988), in a review of clinical studies of screening tools, provided a summary of the sensitivity, specificity, and predictive value of positive and negative tests. These tests of instruments, like studies of prevalence and incidence, are difficult to interpret because different sources of data and timing of data collection are used to make risk assessments and to measure outcome (skin condition). The Norton scale (Norton et al., 1962), the most frequently tested scale, has highly variable sensitivity and specificity and a tendency to overpredict. It has most frequently been tested with elderly patients; its performance with other age groups is uncertain. The Gosnell scale (Gosnell, 1973), when tested with a small sample (N = 30), demonstrated a tendency to underestimate risk. The Braden scale (Bergstrom et al., 1987), the most recently developed and tested scale, has more promising sensitivity and specificity. Further tests are underway to determine the predictive validity of the Braden Scale (see Table 1) with specific populations and to determine the frequency with which risk should be reassessed. Collectively, these pressure sore predictor instruments have been tested more widely than most nursing assessment tools. These studies are consistent with the NPUAP (1989) recommendation that systematic studies be conducted to assess risk predictor tools for the purpose of refining and generalizing to diverse settings and populations.

Risk Factors. Pressure is the foremost factor in the etiology of pressure ulcers, but ulcers can occur with any condition in which the ability to perceive and/or to take or seek meaningful action to relieve pressure is operant. Tissue tolerance for pressure, the ability of the tissue to withstand pressure, is an additional consideration (Braden & Bergstrom, 1987) (see Figure 2). Factors identified as influencing tolerance include moisture from any source, friction and shearing force, nutrition, body temperature, hypotension, and perhaps hypoxia. The NPUAP suggested that a better understanding of the etiology and natural history of pressure ulcers is needed and singled out nutrition as an area needing special attention in relation to the etiology, prevention, and treatment of pressure sores. Few studies have addressed the relationship between nutrition and pressure sore development. Moolten (1972), in studying 50 chronically ill patients with existing pressure sores, related undernutrition to the presence and severity of ulcers. Serum albumin, as a

marker of nitrogen balance, was tested in 46 of the 50 subjects, with levels of 3.5 gm/100 ml or less correlating more closely than body weight with pressure sore formation. Hypoalbuminemia was present in eight of 19 cases of superficial ulcers and in 24 of 31 cases of deep ulcers. Of the 24 subjects with deep ulcers and a serum albumin of < 3.5 gm/100 ml, half were of normal weight or more and half were underweight. It was especially interesting that all of the six patients with a serum albumin less than 2.0 gm/100 ml had deep ulcers and only two were underweight. The hypoalbuminemia found in the subjects with deep ulcers may have resulted, in some measure, from loss of serum protein in the exudates of the ulcer. Therefore, conclusions cannot be drawn regarding the relationship between hypoalbuminemia and initial skin breakdown. How-ever, it is unlikely that the subjects with superficial ulcerations could have lost sufficient serum protein in exudates to account for a serum albumin below 3.5 gm/100 ml in eight of 19 cases reported by Moolten (1972).

Hypoalbuminemia has been related more recently to pressure sores in hospitalized patients (Allman et al., 1986) and in nursing home residents (Bergstrom, Norvell, & Braden, 1988). In Allman's cross-sectional study, subjects in an at-risk group who eventually developed pressure sores had lower serum albumin (2.6 + 0.5 g/dl) than at-risk subjects who did not develop pressure sores (3.1 + 0.7 g/dl) (p = 0.08). The odds of having a pressure sore increased threefold for every gram decrease in the serum albumin. A concurrent prospective study (Bergstrom & Braden, in press) of 200 at-risk (Braden Scale score < 17) nursing home residents who had admission and weekly evaluation of serum albumin showed that 100 percent of the subjects with serum albumin below 2.5 g/dl on admission eventually developed pressure sores. Serum albumin < 3.0g/dl was significantly related to pressure sore development within one week. The role of anemia in the etiology of pressure sores was studied by Moolten (1972) who found significant anemia (hemoglobin < 10 mg/dl) in eight of 31 patients with deep ulcers. Although anemia showed no apparent correlation to body weight, it was highly correlated with hypoalbuminemia. Anemia is generally mentioned as contributing to pressure sore formation due to the diminished oxygencarrying capacity of the blood.

Dietary intake as a marker of nutritional status is addressed as a risk factor in both the Gosnell (1973) and Braden (Bergstrom et al., 1987) scales. Prospective studies are important to confirm the relationship among dietary intake, serum protein, and other nutritional markers. Nutritional outcomes, anthropometric measures (height/weight, triceps skinfold thickness, mid-arm circumference), and blood and serum markers (serum albumin, hemoglobin, serum iron, iron-binding capacity, total lymphocyte counts) have been reported in recent studies (Allman et al., 1986; Bergstrom & Braden, in press; Stotts & Paul, 1988). Reports of hemoglobin and other laboratory tests with sex-specific norms have not separated males and females which makes interpretation of data difficult. Ascorbic acid deficiency, although frequently hypothesized to be an etiological factor, has not been studied prior to the development of ulcers; however, it has been studied in two groups of patients with pressure ulcers. Hunter and Rajan (1971) found that ascorbic acid stores may be reduced in paraplegic patients with ulcers. Taylor, Rimmer, Day, Butcher, and Dymock (1974), in a prospective, double-blind, controlled trial, found that dietary supplementation of ascorbic acid greatly reduced the area of ulceration for the experimental group after one month of supplementation.

Nursing Interventions to Prevent Pressure Ulcers in "At-Risk" Patients

Clinical screening tools identify "at-risk" persons for the purpose of early intervention and pressure sore prevention. The prevention of pressure sores is the desired norm and can be accomplished by intervention strategies employed early in the course of acute hospital stays and nursing home admissions; many pressure ulcers have been documented to occur within two weeks of admission (Andersen et al., 1982; Bergstrom & Braden, in press; Norton et al., 1962). Pressure is the foremost factor in the development of pressure sores; hence, methods to relieve pressure

have received much attention. Frequent turning regimens (usually every two hours) are usually instituted as part of a care plan for at-risk subjects. When more frequent turning is necessary, a scheduled turning regimen may be supplemented with planned small shifts in body weight. The efficacy of this intervention, based on the work of Norton et al. (1962), was described by Horsley, Crane, Haller, and Bingle (1981); they recommended an implementation protocol that was subjected to only one small clinical test (Brown, Boosinger, Black, & Gaspar, 1985). Fifteen patients at risk for pressure sore development (Norton score < 14) in four nursing homes were randomly assigned to receive standard care or standard care plus frequent small shifts in body weight. Despite high-risk status, none of the subjects in the experimental group and only one in the control group developed pressure sores. Although this sample was too small to draw conclusions about the intervention, it appears intuitively promising and deserves further testing.

Special mattresses, mattress overlays, and cushions often are used when frequent turning is either difficult or not possible, and when patients are at higher risk. These devices are classified as static (nonmoving) or dynamic (movement of air currents by electrical systems). The static systems include foam, water, gel, and air-filled devices that are placed over the mattress or chair surface to provide for more even weight distribution, but these systems still require manual turning of patients. The dynamic support systems adjust weight distribution by alternating air currents and include two types: mattress overlays, such as alternating current pads, and air-fluidized beds. Numerous studies comparing these pressure-relieving devices are presented as unpublished reprints by product manufacturers, but most have never been subjected to scientific review. Polystyrene bead pillow beds and cushions were associated with a lower incidence of pressure ulcers than a standard surface (16% vs. 49%) for elderly subjects at substantial risk for developing pressure ulcers (Norton score < 12-13) (Goldstone, Norris, O'Reilly, & White, 1982). In another study, chronic, neurologic patients (N = 32) at high risk for pressure sore development (Norton score, no cutoff for risk was given) were randomly assigned to alternating air or silicone mattresses for three weeks (Daechsel & Conine, 1985). Both groups received standard care with turning every three to four hours while on these mattresses. Four subjects in each group developed pressure sores by the end of three weeks.

The pressure under bony prominences was measured in normal subjects, (Maklebust, Mondoux, & Sieggreen, 1986;), a combination of cancer patients and normal volunteers (Douglass, Holyoke, Goodwin, & Priore, 1983), and rehabilitation patients and employees (Krouskop, Williams, & Krebs, 1984). Douglass et al. (1983) found that pressures under inflated surfaces were not always lower than pressures under static surfaces, and that Chux pads placed over the surface for protection from incontinence did not increase or decrease pressure. A comparison of conventional mattresses, convoluted two-inch foam, Biogard flotation unit, and Sof-Care bed cushions showed that the means of three pressure measurements were lowest over the sacrum and trochanter with Sof-Care and over the heel with Biogard (Maklebust et al., 1986). Krouskop et al. (1984) found no statistically significant difference between the means of three pressure readings when subjects rested on Mediscus or Clinitron beds.

Wheelchair cushions and pads frequently are used for patients confined to chairs for long periods of time. There are many products on the market and numerous studies comparing two or more methods, but few are published. A study using a randomized Latin square design in which three spinal cord-injured patients tested seven cushions and pads for 30 minutes each showed that after 30 minutes of sitting, some redness occurred with each cushion or pad (DeLateur, Berni, Hongladarom, & Giaconi, 1976). One study of 251 para- and quadriplegics, referred to occupational therapy for evaluation of cushions because of pressure sores, prior pressure sores, or surgery to correct pressure sores, evaluated eight different types of wheelchair pads. The Roho pad was used most often and the author, without presenting any data, concluded that it was most effective in providing pressure relief (Garber, 1985). Elderly persons seated on a hard surface have been shown to: 1) have skin blood flow occluded at lower pressures; 2) have higher shear values;

3) benefit from tipping the chair backward 20 degrees; and 4) have greater variability in sitting pressures than younger healthy men (Bennett, Kavner, Lee, Trainor, & Lewis, 1981).

Skin temperature increased from 2.3 to 2.5 degrees under the ischial tuberosities, and from 3.2 to 3.5 degrees under the thighs when normal subjects sat on latex rubber for 30 minutes. A decrease of 3.2 degrees under the ischial tuberosities and 2 degrees under the thighs occurred when water flotation pads were used (Fisher, Syzmke, Apte, & Kosiak, 1978). Subsequent work (Seymour & Lacefield, 1985) showed that spinal cord-injured subjects had higher temperatures than normal subjects and that alternating pressure and foam pads were associated with the highest temperatures. All eight cushions tested had pressures more than two times greater than those reported for occluding circulation.

Treatment of Pressure Ulcers

The choice of treatment for ulcers depends on the stage of the ulcer, the presence or absence of infection and/or necrosis, and the location of the ulcer. According to Parish, Witkowski, and Crissey (1983), over 2,200 topical preparations have been recommended for treatment of ulcers. A review of this myriad of products is not practical. The fact that no standard for treatment is currently accepted by the health care community indicates that none of these methods has enjoyed enduring success.

Non-surgical treatments for pressure ulcers have focused on providing stimuli to promote healing. Several reports suggest that high or low voltage, pulsed electrical current can accelerate the rate of healing of dermal ulcers (Akers & Gabrielson, 1984; Alvarez, Mertz, Smerbeck, & Eaglstein, 1983; Frantz, 1989; Gault & Gatens, 1976; Kloth & Feedar, 1988). It has been hypothesized that electrical stimulation increases cutaneous vasodilatation, thereby increasing the rate of healing. Electrical stimulation has been delivered via low voltage galvanic stimulation (Gault & Gatens, 1976) and more recently by high voltage, low amperage, low density, and short pulse duration (Brown & Gogia, 1987). Animal studies have provided mixed results. Brown and Gogia (1987) reported that rabbits treated with high voltage did not have significantly improved wound healing, and the treatment may have hampered healing for one group. The healing of eleven Yorkshire pigs following dermal injury and treatment with electrical stimulation provided by a nine-volt battery was reported to be greater in treated than nontreated areas of the same animal (Alvarez et al., 1983).



Courtesy, Craig Edmundson, University of Nebraska Medical Center; photo taken at Mercy Care Center, Omaha, NE

Low intensity, direct current has had promising results with healing of ischemic skin ulcers (Gault & Gatens, 1976). Healing of untreated ulcers in six patients occurred at the rate of 14.7 percent per week while treated ulcers in the same patients healed at the rate of 30 percent per week. Akers and

Gabrielson (1984) failed to find a statistically significant difference in healing of pressure sores among 14 subjects divided into three groups who were treated with high voltage stimulation, stimulation plus whirlpool baths, and whirlpool baths alone. The author concluded that high voltage stimulation promoted more regular wound healing, but the three groups were vastly different

in diagnostic and other characteristics. Another study (Kloth & Feedar, 1988) that controlled for pressure-relieving devices, protein supplementation, wound debridement, and dressings (saline-

moistened gauze during debridement; tegaderm after debridement) demonstrated that nine subjects in a group treated with high voltage, pulsed stimuli completely healed while a control group did not heal and worsened. They concluded that electricity enhanced the rate and extent of wound healing, but conclusions were not based on statistical analysis. Based on available data, it is impossible to determine the efficacy of electrical treatment. The intensity and application of stimuli across studies have been highly variable, and patient characteristics only partially controlled. Inadequate numbers of subjects have been studied rendering statistical analysis inadequate when it was attempted. The application of Transcutaneous Electrical Nerve Stimulation (TENS) for the treatment of pressure ulcers may hold promise. Frantz (1989) is using TENS to promote healing of pressure ulcers reported to be resistant to healing. Many confounding variables are being controlled, and preliminary findings appear promising.

Hyperbaric oxygen has been used for treatment; theoretically, it can deliver an increased amount of usable oxygen to the tissues. This may increase the bacteriocidal activity of leukocytes, and increased PO2 may enhance collagen synthesis by fibroblasts (Gabb & Robin, 1987; Rosenthal & Schurman, 1971). An excellent review article (Gabb & Robin, 1987) dubbed hyperbaric oxygen as a "therapy in search of a disease." Chronic ulcers are only one of the 132 past or present indications for this treatment. There have been clinical reports of effective treatment of dermal ulcers and pressure ulcers, but Gabb & Robin concluded that there were no acceptable clinical trials to date.

It is unlikely that any one treatment, mattress or device, topical ointment or dressing, will be

successful in preventing or treating pressure sores. Studies focusing on only one of these preventive or intervention strategies (e.g., one mattress vs. another) are limited in usefulness. Studies are needed that attend to multiple etiological factors. For example, different interventions may be appropriate for persons identified as low, moderate, and high risk for ulcer development. The protocol for interventions may well focus on the etiological factors related to the development of the problem. Pressure-relieving devices or routines will usually be the first and foremost preventive/treatment strategy. The level of risk may determine whether frequent turning or position changes are adequate, or whether low cost mattress overlays or higher cost specialty beds are required to relieve pressure. Other interventions in the protocol will focus on specific risk/etiological factors (e.g., measures to keep incontinent patients dry, nutritional supplementation).

Research Needs and Opportunities

Older persons are at especially high risk for the development of pressure sores; hence, it is appropriate to target elderly populations in all settings for further study. Older persons in home care, during transition to a nursing home, and during long-term care may all be at especially high risk for pressure sore development. Little is known about the effect of accurate risk prediction, staffing levels, case mix, and costs associated with the prevention of pressure sores. These areas all need further intensive investigation. A great deal of work has been done to refine the methods for predicting or assessing risk, and this knowledge is almost ready for diffusion to clinicians. It is appropriate to develop methods for widespread education of nursing staff, clinical implementation, and further testing. Formal research utilization studies are needed. The focus in utilization should be on reliability and validity of assessment. The critical test of the utility of risk assessment will be the success of preventing pressure sores; hence, experimental studies of multifactorial models of prevention strategies are essential. These studies must consider the level of risk and specific risk factors of individuals when planning interventions and must include periodic reassessment of risk and skin condition.

Studies considering the efficacy of one pressure-relieving product over another often have used normal subjects or heterogenous patient populations; have not considered level of risk; have not

controlled for co-variates such as age, sex, race, weight, activity, and mobility; and have tested the product only once during the one-to two-hour study. Longitudinal studies are needed to evaluate clinical interventions for usefulness in clinical practice. Studies should: 1) be prospective versus retrospective or cross-sectional; 2) include control or comparison groups; 3) test appropriate interventions for assessed levels of risk; 4) assess risk with reliable and valid risk predictor tools; 5) provide for documentation of standard care given to control groups and standard care given in addition to the intervention being tested; and 6) evaluate skin condition and other clinical outcomes at specific intervals using trained data collectors rather than information from patient records. Documentation of staffing patterns, nurse-patient ratios, and level of education of the caregiver also would be useful for extrapolation to resources planning.

The development of treatment models may require conducting some basic studies; for example, studies on methods to accurately measure the interface pressure between the skin and mattress and to determine the level of dietary supplementation safe and necessary to improve markers of nutritional status and wound healing. The often-used, available nursing treatment models should be tested systematically to determine their degree of efficacy and to highlight risk factors needing further basic study. Models for treatment of existing pressure sores must be tested in multifactorial controlled tests considering and treating or controlling the multiple risk/co-morbidity factors. Great care must be taken to adequately describe the subject (e.g., risk level, nutritional status, continence) and to control, treat, and document the care provided to reduce all risk factors. Accurate methods for measuring pressure sore healing must be used, and the progress of the subject should be followed after care to document recidivism. Once studies have been conducted to test multifactorial models for prevention and healing of pressure sores, quality standards should be developed that reflect the treatment model of choice, level of staffing given a specified case mix, and cost of prevention and treatment.

Recommendations

Based on the foregoing assessment of research needs and opportunities in "Skin Integrity," the Panel has made the following recommendations concerning research in this area over the next five years.

- Determine the prevalence, incidence, and cost of pressure ulcers by stage and type of health facility to permit better planning for and allocation of service.
- Develop systematic, reliable instruments for assessment of pressure sore risk; this is essential for early treatment and prevention of pressure ulcers. Such instruments must be reliable when used by staff nurses, and this reliability should be demonstrated through clinical trials.
- Identify and investigate etiological factors that may increase the accuracy of prediction and early treatment, for example, the nutrition-pressure sore relationship.
- Conduct comprehensive studies of nursing interventions that consider the level of risk and specific risk factors and the need for both pressure relief and attention to factors influencing tissue tolerance. Studies focused on only one intervention are unlikely to produce clear results.

References

Akers, T. K., & Gabrielson, A. L. (1984). The effect of high voltage galvanic stimulation on the rate of healing of decubitus ulcers. Biomedical Sciences Instrumentation, 20, 99-100.

Abildgaard, U., Daugaard, K., (1979). Pressure sores a prevalence investigation. Ugeskr. Laeg, 141(46), 3147-54.

Allman, R. M., Laprade, C. A., Noel, L. B., Walker, J. M., Moorer, C. A., Dear, M. R., & Smith, C.R. (1986). Pressure sores among hospitalized patients. Annals of Internal Medicine, 105(3), 337-42.

Alvarez, O. M., Mertz, P. M., Smerbeck, R. V., & Eaglstein, W. H. (1983). The healing of superficial skin wounds is stimulated by external electrical current. The Journal of Investigative Dermatology, 81(2), 144-148.

Andersen, K. A., Jensen, O., Kvorning, S. A., & Bach, E. (1982). Prevention of pressure sores by identifying patients at risk. British Medical Journal, 284(6326), 1370-1371.

Barbenel, J. C., Jordan, M. M., Nicol, S. M., & Clark, M. O. (1977). Incidence of pressure sores in the greater Glasgow health board area. Lancet, 2, 548-550.

Bennett, L., Kavner, D., Lee, B. Y., Trainor, F. S., & Lewis, J. M. (1981, August). Skin blood flow in seated geriatric patients. Archives of Physical Medicine and Rehabilitation, 62(8), 392-398.

Bergstrom, N., Norvell, K., & Braden, B. (1988, October). Instant nutritional assessment, serum albumin, and total lymphocyte count as predictors of pressure sore risk. The Gerontologist, 28 (Suppl.), 76A.

Bergstrom, N. (1989). Skin Assessment Tool. Copyright N. Bergstrom, University of Nebraska Medical Center, Omaha, Nebraska 68105-5330.

Bergstrom, N., & Braden, B. (In press). A prospective study of pressure sore risk among institutionalized elderly. Journal of the American Geriatric Society.

Bergstrom, N., & Braden, B. (1990). Nutritional/pressure sore link: Nutritional status during the development and resolution of pressure sores in older persons. In S. Funk, E. Tournquist, & E. Champagne (Eds.), Key aspects of recovery: Improving mobility, rest, and nutrition in infants, adults, and older persons, (pp. 183-187). New York: Springer.

Bergstrom, N., Braden, B.J., Laguzza, A. & Holman, V. (1987). The Braden Scale for predicting pressure sore risk. Nursing Research, 36(4), 205-210.

Bergstrom, N., Braden, B., Champagne, M., & Kemp, M. (1988-1991). Nursing Assessment of Pressure Sore Risk. Funded by NIH, NCNR, 2RO1 NU 01061.

Bergstrom, N., Demuth, P.J., & Braden, B. (1987). A clinical trial of the Braden Scale for predicting pressure sore risk. Nursing Clinics of North America, 22(2), 417-428.

Braden B., & Bergstrom, N. (1987). A conceptual schema for the study of the etiology of pressure sores. Rehabilitation Nursing, 12(1), 8-12,16.

Brandeis, G. H., Morris, J. N., Nash, D. J., & Lipsitz, L. A. (1989). Incidence and healing rates of pressure ulcers in the nursing home. Decubitus, 02(2), 60-62.

Brandeis, G. H., Morris, J.N., Nash, D. J., & Lipsitz, L. A. (1990). The epidemiology and natural history of pressure ulcers in elderly nursing home residents. Journal of the American Medical Association, 265(22), 2905-2909.

Brown, M., & Gogia, P. P. (1987). Effects of high voltage stimulation on cutaneous wound healing

in rabbits. Physical Therapy, 67(5), 662-667.

Brown, M. M., Boosinger, J., Black, J., & Gaspar, T. (1985). Nursing innovation for prevention of decubitus ulcers in long-term care facilities. Plastic Surgical Nursing, 5(1), 57-64.

Clark, M., & Kadhom, J. (1988). The nursing prevention of pressure sores in hospital and community patients. Journal of Advanced Nursing, 13, 365-373.

Daechsel, D., & Conine, T. A. (1985). Special mattresses: Effectiveness in preventing decubitus ulcers in chronic neurologic patients. Archives of Physical Medicine and Rehabilitation, 66(4), 246-248.

DeLateur, B. J., Berni, R., Hongladarom, T., & Giaconi, R. (1976). Wheelchair cushions designed to prevent pressure sores: An evaluation. Archives of Physical Medicine and Rehabilitation, 57(3), 129-135.

Douglass, H. O., Jr., Holyoke, E. D., Goodwin, P. M., & Priore, R. L. (1983). Skin pressure measurements on various mattress surfaces in cancer patients. American Journal of Physical Medicine, 62(5), 217-226.

Eckman, K. L. (1989). The prevalence of dermal ulcers among persons in the U.S. who have died. Decubitus, 2(2), 36-40.

Ek, A. C., & Bowman, G. (1982). A descriptive study of pressure sores: The prevalence of pressure sores and the characteristics of patients. Journal of Advanced Nursing, 7, 51-57.

Fisher, S. V., Szymke, T. E., Apte, S. Y., & Kosiak, M. (1978). Wheelchair cushion effect on skin temperature. Archives of Physical Medicine and Rehabilitation, 59(2), 68-72.

Frantz, R. (1989). The effect of TENS on healing of decubitus ulcers. Proceedings of the scientific sessions, 30th biennial convention of Sigma Theta Tau International (p. 100). Indianapolis, IN.

Gabb, G., & Robin, E. D. (1987). Risk-benefit analysis in chest medicine. Chest, 92(6), 1074-1082.

Garber, S. L. (1985). Wheelchair cushions for spinal cord-injured individuals. American Journal of Occupational Therapy, 39(11), 722-725.

Gault, W. R., & Gatens, P. F. Jr. (1976). Use of low intensity direct current in management of ischemic skin ulcers. Physical Therapy, 56(3), 265-268.

Gerson, L. W. (1975). The incidence of pressure sores in active treatment hospitals. International Journal of Nursing Studies, 12, 201-204.

Goldstone, L. A., & Roberts, B. V. (1980). A preliminary discriminant function analysis of elderly orthopaedic patients who will or will not contract a pressure sore. International Journal of Nursing Studies, 17(1), 17-23.

Goldstone, L. A., & Goldstone, J. (1982). The Norton score: an early warning of pressure sores? Journal of Advanced Nursing, 7(5), 419-26.

Goldstone, L. A, Norris, M., O'Reilly, M., & White, J. (1982). A clinical trial of a bead bed system

for the prevention of pressure sores in elderly orthopaedic patients. Journal of Advanced Nursing, 7, 545-548.

Gosnell, D. J. (1973). An assessment tool to identify pressure sores. Nursing Research, 22(1), 55-59.

Hoenshell-Nelson, N., & Miller, S. M. S. (1988). The validity of the Braden Scale for predicting pressure sore in adult intensive care unit patients. Unpublished Master's Thesis. Omaha: UNMC.

Horsley, J. A., Crane, J., Haller, K. B., & Bingle, J. D. (1981). Preventing decubitus ulcers. New York: Grune & Stratton.

Hunter, T., & Rajan, K. T. (1971). The role of ascorbic acid in the pathogenesis and treatment of pressure sores. Paraplegia, 8, 211-216.

Jensen, T. T., & Juncker, Y. (1987). Pressure sores common after hip operations. Acta Othopaedica Scandinavica, 58(3), 209-1

Kemp, M. G., Keithly, J. K., Smith, D. W., & Morreale, B. (1990). Factors that contribute to pressure sores in surgical patients. Research in Nursing and Health, 13, 293-301.

Kemp, M., & Bergstrom, N. (1988). Assessing skin for pressure sores. University of Nebraska Medical Center, Biomedical Communications.

Kennedy, K. L. (1989). The prevalence of pressure ulcers in an intermediate care facility. Decubitus, 2(2), 44-45.

Kloth, L. C., & Feedar, J. A. (1988). Acceleration of wound healing with high voltage, monophasic, pulsed current. Physical Therapy, 68(4), 503-508.

Krouskop, T., Williams, R., & Krebs, M. (1984, November). The effectiveness of air flotation beds in lowering pressure under the recumbent body. Care, Science and Practice, 4(2), 9-12.

Langemo, D. K., Olson, B., Hunter, S., Burd, C., Hansen, D., & Cathcart-Silberberg, T. (1989). Incidence of pressure sores in acute care, rehabilitation, extended care, home health, and hospice in one locale. Decubitus, 2(2), 42.

Lincoln, R., Roberts, R., Maddox, A., Levine, S., & Patterson, C. (1986). Use of the Norton Pressure Sore Risk Assessment Scoring System with elderly patients in acute care. Journal of Enterostomal Therapy, 13(4), 132-8.

Lofgren R. P., MacPherson, D. S., Granieri, R., Myllenbeck, S., & Sprafka, J.M. (1989). Mechanical restraints on the medical wards: are protective devices safe? American Journal of Public Health, 79(6), 735-8.

Lowthian, P. (1979). Pressure sore prevalence: A survey of sores in orthopaedic patients. Nursing Times, 75(9), 358-360.

Maklebust, J., Mondoux, L., & Sieggreen, M. (1986). Pressure relief characteristics of various support surfaces used in prevention and treatment of pressure ulcers. Journal of Enterostomal Therapy, 13(3), 85-89.

Manley M.T. (1978). Incidence, contributory factors and costs of pressure sores. South African Medical Journal, 53(6), 217-22.

Moolten, S. E. (1972). Bedsores in the chronically ill patient. Archives of Physical Medicine and Rehabilitation, 53(9), 430-438.

National Pressure Ulcer Advisory Panel, Consensus Department Conference Statement. (1989). Decubitus, 2(2), 24-28.

Norton, D., McLaren, R., & Exton-Smith, A. N. (1962). An investigation of geriatric nursing problems in hospitals. London: National Corporation for the Care of Old People.

Nyquist, R., & Hawthorn, P. J. (1987). The prevalence of pressure sores within an area health authority. Journal of Advanced Nursing, 12, 183-187.

Oot-Giromini, B., Bidwell, F. C., Heller, N. B., Parks, M. L., Wicks, P., & Williams, P. M. (1989). Evolution of skin care: Pressure ulcer prevalence rates pre/post intervention. Decubitus, 2(2), 54-55.

Pajk, M., Craven, G. A., Cameron-Barry, J., Shipps, T., & Bennum, N. W. (1986). Investigating the problem of pressure sores. Journal of Gerontological Nursing, 12(7), 11-6.

Parish, L. C., Witkowski, J. A., & Crissey, J. T. (1983). The Decubitus Ulcer. Chicago: Year Book Medical Publishers.

Petersen, N. C., & Bittmann, S. (1971). The epidemiology of pressure sores. Scandinavian Journal of Plastic and Reconstructive Surgery, 5(1), 62-6.

Powell, J. W. (1989). Increasing acuity of nursing home patients and the prevalence of pressure ulcers: A ten year comparison. Decubitus, 2(2), 56-58.

Raney, J. P. (1989). A comparison of the prevalence of pressure sores in hospitalized ALS and MS patients. Decubitus, 2(2), 48-49.

Richardson, R. R., & Meyer, P. R. (1981). Prevalence and incidence of pressure sores in acute spinal cord injuries. Paraplegia, 19, 235-247.

Rosenthal, A. M., & Schurman, A. (1971, September). Hyperbaric treatment of pressure sores. Archives of Physical Medicine & Rehabilitation, 52(9), 413-415, 433.

Seymour, R. J., & Lacefield, W. E. (1985). Wheelchair cushion effect on pressure and skin temperature. Archives of Physical Medicine and Rehabilitation, 66(2), 103-108.

Shannon, M. L., & Skorga, P. (1989). Pressure ulcer prevalence in two general hospitals. Decubitus, 2(4), 38-43.

Stotts, N. A. (1988). Predicting pressure ulcer development in surgical patients. Heart Lung, 17(6 Pt 1), 641-7.

Stotts, N. A., & Paul, S. M. (1988). Pressure ulcer development in surgical patients. Decubitus, 1 (3), 24-30.

Taylor, T. V., Rimmer, S., Day, B., Butcher, J., & Dymock, I. W. (1974). Ascorbic acid supplementation in the treatment of pressure sores. Lancet, 2, 544-546.

Taylor, K. J. (1988). Assessment tools for the identification of patients at risk for the development of pressure sores: A review. Journal of Enterostomal Therapy, 15(5), 201-205.

Versluysen M. (1985). Pressure sores in elderly patients, the epidemiology related to hip operations. Journal of Bone and Joint Surgery, 67-B(1), 10-13.

Versluysen, M. (1986). How elderly patients with femoral fracture develop pressure sores in hospital. British Medical Journal [Clinical Research], 292(6531), 1311-3.

Warner, U., & Hall, D. J. (1986). Pressure sores: a policy for prevention. Nursing Times, 82(16), 59-61.

Young, L. (1989). Pressure ulcer prevalence and associated patient characteristics in one long-term care facility. Decubitus, 2(2), 52.

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CHAPTER 5