

# **Risk-Assessment Scores, Prevention Strategies, and the Incidence of Pressure Ulcers among the Elderly in Four Canadian Health-Care Facilities**

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Prévoir avec précision l'apparition des ulcères de pression (UP) chez les personnes âgées hospitalisées est une entreprise complexe. Une étude prospective, longitudinale, portant sur une cohorte de 330 patients âgés de plus de 65 ans dans deux hôpitaux d'enseignement de soins tertiaires et dans deux établissements de soins de longue durée du Canada, a porté sur les liens qui existent entre les résultats de l'évaluation des risques, les stratégies de prévention et l'incidence des UP. L'incidence globale des UP s'est établie à 9,7 %, la moitié des sujets ayant développé des UP pendant la première semaine d'hospitalisation. Le taux d'incidence chez les patients à risque s'est établi à 10,1 %, comparable au taux enregistré auprès des patients non à risque (9,3 %). Il se dégage un rapport entre le nombre de stratégies de prévention déployées d'une part et les résultats de l'évaluation des risques et l'apparition des UP d'autre part. Paradoxalement, le taux d'incidence augmente avec le nombre de stratégies de prévention mises en oeuvre. Le score total à l'échelle d'évaluation des risques qui semble être le plus sensible (69 %) et le plus spécifique (55 %) est 19. Quatre des six sous-échelles d'évaluation des risques ont un rapport avec l'apparition des UP. La modélisation de la régression logistique confirme les résultats à une variable voulant que le nombre de stratégies de prévention déployées est le meilleur facteur de prédiction de l'apparition d'ulcères de pression. Les données confirment qu'il est difficile de prédire si tel ou tel patient développera des ulcères de pression. Les résultats donnent à penser que l'utilisation d'une échelle d'évaluation des risques ne suffit pas à elle seule à prédire avec exactitude s'il y aura ou non des ulcères de pression. Le jugement clinique et l'expérience des infirmières s'imposent et complètent les instruments de mesure standards.

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The accurate prediction of pressure ulcer (PU) development among hospitalized elderly patients is a complex endeavour. A prospective, longitudinal, cohort study of 330 patients over age 65 in 2 Canadian tertiary-care teaching hospitals and 2 long-term-care facilities examined the association between risk-assessment scores, prevention strategies, and PU incidence. The overall PU incidence rate was 9.7%, with half of the subjects who developed a PU doing so in the first week of hospitalization. The incidence rate for "at risk" patients (10.1%) was similar to the rate for "not at risk" patients (9.3%). The number of prevention strategies used was related to risk-assessment scores and to PU development. Paradoxically, the incidence rate increased with the number of prevention strategies employed. The total risk-assessment score that appeared to have the best balance of sensitivity (69%) and specificity (55%) was 19. Four of the 6 risk-assessment subscales were associated with PU development. Logistic regression modelling confirmed the univariate results that the number of prevention strategies used was the best single predictor of PU development. The data confirm that predicting PU development for individual patients is difficult at best. Results suggest that use of a risk-assessment scale alone is not sufficient to accurately predict PU development. The clinical judgement and experience of nurses are required in providing supplementary information to standard measurement instruments.

## **Introduction**

Pressure ulcers (PU) are a significant clinical problem among hospitalized elderly patients, in spite of the fact that they are largely preventable. It is a high-volume, high-risk problem in many health-care settings and is often used as an indicator of quality of care (Frantz, 1997). Increasing in-patient acuity levels and the aging of the population have the potential to lead to an increased incidence of skin breakdown in hospitalized individuals (Harrison, Wells, Fisher, & Prince, 1996). Substantial resources are expended in efforts to prevent the development of PU. Myriad risk factors have been identified in the literature as contributing to the development of PU, but, unfortunately, conflicting evidence regarding precise delineation and articulation of the most relevant risk factors make identification somewhat problematic.

Accurate identification of the at-risk patient is an ongoing challenge. Practical constraints preclude the possibility of collecting all potential covariates in every patient. Nurses often independently assume responsibility for assessing patient risk for PU and implementing appropriate prevention strategies. Clinical practice guidelines from the Agency for Health Care Policy and Research (AHCPR) strongly promote the use of risk-assessment instruments, and strong support for the clinical use of these tools is indicated by the results of numerous studies (Bankert, Daughtridge, Meehan, & Colburn, 1996; Bergstrom & Braden, 1992; Foltz-Gray, 1997; Langemo et al., 1991). Accurate assess-

ment of PU risk will allow for the appropriate allocation of physical and human resources.

This paper reports on a cohort study that attempted to address these issues by investigating the association between PU incidence, prevention strategies, and risk-assessment scores in a geriatric in-patient population of four Canadian health-care facilities. The findings are part of a larger study examining prevention strategies and treatment trajectory related to PU (Goodridge, LeDoyen, Sloan, McKenzie, & Knight, 1997). Specific research questions investigated for this manuscript were: (1) *Can risk-assessment scores alone accurately predict the development of PU in older adults?* (2) *Can prevention strategies alter the risk, and therefore the ultimate incidence rate, of PU?*

### **Review of the Literature**

Predicting the development of PU in an individual patient presents a daunting challenge (Bergstrom & Braden, 1992; Burd, Langemo, & Olson, 1992; Harrison et al., 1996; Norton, 1996). A number of studies have identified the factors associated with PU development. It is a different and more difficult matter, however, to collate these risk factors into a practical prognostic index capable of predicting individual patient illness trajectory and identifying which patients will develop PU.

The evidence supporting the contribution of specific risk factors in PU is inconclusive. Age was initially identified as an important risk factor by Bergstrom and Braden (1992) and Spector, Kapp, Tucker, and Sternberg (1988), but was found not to be significant in a large epidemiological study by Brandeis, Ooi, Hossain, Morris, and Lipsitz (1994). Male gender has been implicated as a risk factor in some studies (Brandeis et al., 1994; Spector et al.), but not in others (Guralnik, Harris, White, & Coroni-Huntley, 1988; Smith, Winsemius, & Besdine, 1991; Verdery & Mittlemark, 1990). Maklebust and Magnan (1994), in a study with 2,189 patients, examined a series of risk factors: fecal incontinence, urinary incontinence, malnutrition, impaired mobility, decreased mental status, diabetes mellitus, peripheral vascular disease, spinal cord injury, multiple sclerosis, and metastatic carcinoma. Fecal incontinence was associated with a 22-fold increase in PU and was the second most frequently occurring risk factor. Stepwise logistic regression resulted in a model that included fecal incontinence, impaired mobility, malnutrition, decreased mental status, and an interaction effect between fecal incontinence and impaired mobility. Patients with both fecal inconti-

nence and impaired mobility were 37.5 times more likely to develop PU than patients with neither.

Increasingly sophisticated analyses of large data sets have produced alternative models of PU development specific to various populations. Brandeis, Berlowitz, Hossain, and Morris (1995), using data on 2,011 patients in 270 nursing homes across 10 American states, determined that dependence in transfer or mobility, being bedfast, having diabetes mellitus, and having had a PU in the past were the only factors significantly associated with the development of Stage 2–4 ulcers.

To date, impressive work in this area has included the development of prognostic indices such as the Braden and Norton scales (Harrison et al., 1996; Norton, 1996). These assessment instruments are easy to use and represent minimal clinical intervention. They take a subset of the factors known or widely believed to be involved in PU development and assign the patient numeric scores for presence of risk (Buhrer & Mitchell, 1996). A summative risk score serves as a basis for prediction and prescription of prevention strategies. Clinical use of these risk-assessment instruments is recommended in the AHCPR guidelines (Panel on the Prediction and Prevention of Pressure Ulcers in Adults, 1992).

The Braden risk-assessment tool is widely identified as one of the leading instruments prognostic for PU development (Harrison et al., 1996). It identifies six variables traditionally associated with PU development: sensory perception, moisture, activity, mobility, nutrition, and friction and shear (Bergstrom, Braden, Laguzza, & Holman, 1987). Each variable is rated from 0 to 3 or 4, for a possible total of 23 points, with higher scores indicating low risk. The predictive ability of the Braden Scale has been examined in a number of studies via alternative approaches, with varying results. Sensitivity (the percentage of persons correctly predicted to develop PU) and specificity (the percentage correctly predicted to not develop PU) have varied substantially among studies. Sensitivity has been reported from as low as 40% and 53% (Oot-Giromini, 1993; Salvadalena, Snyder, & Brogdon, 1992) to as high as 100% (Bergstrom, Braden, et al., 1987). A number of studies found more moderate sensitivities, ranging from 67% to 83% (Barnes & Payton, 1993; Bergstrom, Demuth, & Braden, 1987; Braden & Bergstrom, 1994; Capobianco & McDonald, 1996; Harrison et al.) using cut-off scores of 16 or 18. Reported specificity has ranged from 50–59% (Braden & Bergstrom; Oot-Giromini) to 91% (Barnes & Payton). The variation in sensitivity and specificity appears to be due, in part, to the different risk

cut-off points and populations studied. The majority of the cited studies used sample sizes of 60–100.

The quality or type of care provided by a particular agency may also influence the relative significance of individual risk factors, rendering a standardized tool for PU development ineffective across multiple settings. Brandeis et al. (1994), in reviewing data on more than 4,232 nursing-home patients, found differences in the variables associated with PU development in high-incidence (19.3%) and low-incidence (6.5%) nursing homes. In high-incidence homes, fecal incontinence and diabetes mellitus were significantly related to the development of PU. In low-incidence homes, in contrast, risk factors included male gender but not fecal incontinence or diabetes mellitus. In both settings, difficulties with ambulation and self-feeding were significant factors. Age, BMI, transfer ADL, and facility size were not significantly associated with the incidence of PU in either group. These authors suggest that there may be an unknown or unmeasured facility effect on the risk for PU development in addition to the characteristics of a given resident in a particular home. These results corroborate the findings of Rudman, Mattson, Alverno, Richardson, and Rudman (1993), who compared clinical indicators in two nursing homes. Residents of one nursing home were significantly less likely to develop PU than those of the other. The authors attribute the difference to exogenous (environmental and quality-of-care) causes such as higher staffing and greater expenditures on clinical care.

A danger inherent in using risk-assessment scales is oversimplification of a very complex set of interacting factors that produce ulcers in some patients but not in others. A qualitative study conducted with nurses expert in the prevention of PU (Buhrer & Mitchell, 1996) demonstrated the complexity of parameters that expert nurses take into account when determining risk. The nurses included such factors as particular medical conditions (chronic illness, local and systemic infection, respiratory diseases, diabetes), age, serum albumin, and hypotension in their judgement regarding the patient's risk status. They likewise gave preferential consideration to nutrition and activity/mobility as critical elements. While formalized risk-assessment tools often emphasize levels of consciousness, the expert nurses tended to focus on mood, motivation, and social support as key factors in risk status. There remain many unanswered questions about the association between risk-assessment scales and PU prevention strategies.



## Methods

### *Subjects and Settings*

For this prospective, single-arm, longitudinal, observational study, a convenience sample of 330 patients was drawn from patients 65 years of age and older consecutively admitted, within the preceding 48–96 hours, to the medical and geriatric units of two tertiary-care hospitals ( $n = 222$ ) and two long-term-care facilities ( $n = 108$ ) in a large western Canadian city. Exclusion criteria were: pre-existing dermal ulcers, the terminal stages of cancer, and acute or chronic renal failure.

### *Procedures*

Ethical approval was received from the Faculty of Nursing, University of Manitoba, and access approval was received from each of the participating facilities. Research assistants were given both didactic and clinical training in the use of the Braden Scale and the use of demographic, prevention-strategy, and other research instruments. Several meetings were held with staff nurses and the head nurse of each participating unit to inform them of the study protocol.

Potential subjects were identified through daily contact with the head nurse of each participating unit to determine whether eligible patients had been admitted. Patients were given a brief written disclaimer by the research assistant. The disclaimer stated that a project on skin care was being conducted, that data would be gathered from the health record only, and that confidentiality and consistency of care were guaranteed.

If the patient agreed to participate, the research assistant gathered data from the health record within 48–96 hours of admission, employing demographic, medical, risk-assessment (Braden Scale), and prevention-strategy data-collection instruments that had been pilot-tested by the investigators to ensure validity and interrater reliability.

Data were collected bi-weekly for a period of 3 months or until the patient was discharged or transferred from the participating unit, in order to assess the accuracy of the risk-assessment method prospectively. The data were gathered from a health-record review and verified clinically by the nurse who provided care to the subject on the day of collection. The data included medical, demographic, prevention-strategy, and Braden Scale information. Nurses were kept blinded to the Braden Scale information to avoid the possibility of Hawthorne effect. Medical and demographic data included age, medical diagnoses, labo-

ratory values (WBC, hemoglobin, albumin, zinc), height, weight, and medications. Prevention strategies included turning schedule, ambulation schedule, range-of-motion exercises, assistive positioning/moving devices, protective padding, seating assessments, pressure-reducing mattress, pressure-relieving mattress, use of emollients/lubricants/barriers, incontinence management, nutrition management, and patient/family teaching. While many of these strategies serve other purposes in addition to PU prevention, it was considered important to include them as prevention strategies.

Research assistants monitored the subjects for PU. In the case of patients who developed an ulcer, the skin was evaluated a second time after 20 minutes had elapsed to confirm the initial indication. The research nurse at each institution verified the findings of the research assistant; there was 100% agreement between the research nurses and research assistants in terms of PU identification and assessment. Written consent was obtained to continue with the second phase of the study. The research assistants arranged to be present during regularly scheduled dressing changes on a weekly basis in order to visually assess the wound. They continued to collect medical and prevention-strategy data for subjects with PU.

### *Statistical Methods*

Data were analyzed by the research team in collaboration with the Health Services Research Division of the Mayo Clinic in Rochester, Minnesota. Descriptive statistics were used to summarize the incidence of PU and related patient characteristics. Hypothesized differences in subjects with PU and without PU were tested using a variety of parametric and non-parametric tests appropriate for the level of data under consideration. These are detailed under Results. Comparisonwise type I error rates were set at 5%. The sample of 330 provides 80% power to detect small-effect sizes across most subsets, so statistical significance must be interpreted along with a consideration for clinical relevance. For example, comparing the average of two groups of 165 patients on any continuous variable would provide 80% power to detect a small-effect-size difference of 0.33 standard deviations (Cohen, 1988). Sensitivity, specificity, positive and negative predictive values, and performance of the Braden Scale were calculated.

Larson (1986) defines the various parameters typically used to evaluate screening tests such as the Braden Scale. Sensitivity refers to the extent to which a true characteristic is classified correctly (rate of true positives), while specificity indicates the extent to which the absence of

a characteristic is classified correctly (rate of true negatives). A highly sensitive test will identify the majority of individuals who have a given disease or characteristic; a highly specific test will correctly identify individuals who are free of a given characteristic. Both parameters are necessary for a measure of validity. The test that is chosen must provide the best balance between sensitivity and specificity. Since sensitivity and specificity are inversely related, increasing the sensitivity of a test by lowering the point at which it is considered positive (the cut-off point) decreases its specificity.

The predictive value of a positive test is the probability that when it is positive the characteristic is truly present; in other words, those who test positive are the proportion who have the condition. The predictive value of a negative test is the probability that when it is negative the characteristic is truly absent; those who test negative are the proportion who do not have the condition.

Simple correlation measures (Pearson's *r*, Spearman's *rho*) were used to assess the relationship between the Braden score and associated covariates such as age and number of prevention strategies used. A sample of 330 observations provides 80% power to detect a true correlation of 0.16 with a 5% type I error rate and one-sided testing (Cohen, 1988).

Logistic regression modelling procedures were used to examine the relative prognostic value of selected sociodemographic and clinical variables for predicting PU development in individual patients. Variables were selected by univariate associative testing involving Fisher's exact test for categorical predictors (e.g., gender) and Wilcoxon procedures for continuous variables (e.g., age). The dependent variable was the presence or absence of PU for each patient.

## **Results**

Subjects ranged in age from 65 to 101 years ( $\bar{x} = 78.6$ ,  $SD = 8.53$ ). The average number of medical diagnoses was 5.86 ( $SD = 2.47$ ), the most common diagnoses being cardiac disease (55.9%), arthritis (25.6%), and fractures (23.5%). Mean WBC was 13.9 and hemoglobin 126.2 was g/dl. The most frequently prescribed medications were non-narcotic analgesics and laxatives.

Comparisons of average scores of the 298 patients who did not develop PU and the 32 who did develop PU have 80% power to detect a moderate-effect size of 0.5 standard deviations (Cohen, 1988). A total of 1,251 observations was obtained for the 330 patients. Subjects were



assessed an average of eight times (over 2 months), with a range of 2–14 observations per subject. Only two subjects were assessed for less than 1 month.

### *Pressure Ulcer Development*

Of the 330 subjects, 32 (9.7%) developed a total of 62 ulcers within 3 months of admission. Most of these (73.8%) had only one ulcer, 11.9% had two ulcers, and 14.4% had three or more ulcers. Of the 32 subjects who developed PU, 23 were acute-care patients and nine were long-term-care patients. The incidence rate of 10% (95% C.I. of 6.7%, 13.5%) was not significantly different for the two settings. By the end of the first week of hospitalization, 15 subjects had developed ulcers. By the end of the first month, 75% of all ulcers had developed. The mean number of days in which subjects developed an ulcer was 18.5 ( $SD = 21.27$ ). The sacrum/coccyx was the most frequent site of PU, at 27%. The heel was the next most frequent site, at 20.3%. More than half (57.1%) of the total ulcers were Stage 1, while 31% were Stage 2. Stages 3 and 4 ulcers each accounted for 4.8% of the total ulcers in the sample.

Subjects with PU did not weigh significantly less (58.99 kg) than subjects without PU (66.83 kg) (two-sample  $t$  test  $p = 0.09$ ). Subjects with PU did have a significantly greater number of medical diagnoses (6.2) than subjects without PU (5.4) (two-sample  $t$  test  $p = 0.01$ ).

### *Braden Scale Risk Assessment*

For the sample, the mean Braden score on admission was 18.0 ( $SD = 2.75$ ) with a range of 6–24. For those who developed PU, the mean Braden score on admission was 18.0 ( $SD = 2.75$ ) with a range of 6–24. The average Braden score on admission for those who developed PU (mean = 17.5, median = 18) was roughly one point lower than that for patients who did not ultimately develop PU (mean = 18.8, median = 19, Wilcoxon  $p$  value = 0.002). According to the Braden Scale, on admission 172 subjects (52.1%) were assessed as *no risk* ( $\geq 19$ ), while 107 (32.5%) were assessed as *low risk* (16–18) and 39 (11.8%) as *moderate risk*. Only 12 (3.6%) were assessed as *high risk* ( $< 12$ ). Of the *high risk* subjects, nine were from acute-care facilities. Significantly more long-term-care than acute-care patients were classified as *at risk* (chi squared = 10.09,  $df = 1$ ,  $p < .001$ ), although the prevalence rate of PU was marginally lower in the long-term-care setting.

The incidence of PU development was very similar for *at risk* ( $< 19$ ) and *not at risk* ( $\geq 19$ ) patients (Table 1). Furthermore, although a greater

proportion of long-term-care patients were classified as *at risk*, the incidence of PU was similar for the two groups. While 10.1% of the *at risk* subjects went on to develop an ulcer, a similar proportion (9.3%) of the *not at risk* subjects also developed PU. Only 50% of subjects who developed PU had a Braden score indicative of increased risk. Of the 158 subjects with a Braden score less than 19 (*at risk*), 16 (10.12%) went on to develop PU. One might speculate that the prevention strategies instituted at the point of care were thus effective for the majority of subjects. The degree of risk, however, was not associated with frequency of PU. Four (25%) of the 12 *high risk* subjects developed PU, while four (10%) of the *moderate risk* group and eight (7.5%) of the *low risk* group likewise developed PU. Of the 172 subjects classified as *not at risk* (Braden score >18), 16 (9.3%) developed an ulcer (Table 1). The stage distribution for development of PU was the same for *at risk* and *not at risk* patients.

<b>Table 1</b> <i>PU Incidence Rate Classified by Braden Score Risk Status</i>		
<b>Risk Status</b>	<b>Proportion of Subjects (N = 330)</b>	<b>Proportion of Subjects with PU (N = 32)</b>
Not at risk (Braden Score >18)	172 (52%)	16 (50%)
Low risk (Braden Score 16–18)	107 (33%)	8 (25%)
Moderate risk (Braden Score 12–16)	39 (12%)	4 (25%)
High risk (Braden Score <12)	12 (4%)	4 (25%)
Overall	330	32 (10%)

The average Braden score immediately prior to development of PU (17.42) was typically lower by about two points than the scores of subjects without PU (19.33) (two-sample *t* test  $p<0.0001$ ). There was no change in Braden scores for individual patients over the duration of the study from baseline measurements. The score did not change significantly when the PU appeared. The Braden scores of very few subjects dropped immediately preceding PU appearance to indicate increased risk.

Table 2 illustrates the differences in mean Braden score for patients with and without PU at the four sites, using standard one-way ANOVA

testing. In both acute-care facilities, subjects with PU had significantly lower Braden scores ( $\bar{x} = 18.1$ ) than subjects without PU ( $\bar{x} = 19.9$ ). In the long-term-care facilities, there was no significant difference in scores for subjects with and without PU; the mean Braden score was actually higher for subjects with PU (18.4) than for subjects without PU (18.1).

**Table 2** *Mean Braden Scores for Subjects With and Without PU*

	Mean Braden Score (298 Subjects Without PU)	Mean Braden Score (32 Subjects With PU)	One-way ANOVA F-test <i>p</i> value
Acute Care A	20.4	18.9	0.001
Acute Care B	19.5	17.4	0.01
Long-Term Care A	18.1	17.4	0.64
Long-Term Care B	18.2	19.4	0.27

### *Braden Subscale Items*

The Braden subscale items of sensory perception, nutrition, and moisture were not problematic for this sample. In 70.2% of subjects, sensory perception was unimpaired. More than three quarters of the sample (83.7%) were rated as having either excellent or adequate nutrition. However, 69.0% were assessed as having problems with moisture. In contrast, only 38.2% of subjects were free of mobility limitations and only 18.1% walked frequently. Long-term-care subjects were significantly more impaired than acute-care subjects in terms of mobility and activity (chi squared  $p < .001$ ). Almost half (47.2%) of the subjects experienced no apparent problem with friction and shear, although in 43.9% of cases a potential problem was noted.

Differences in factors associated with increased risk for the acute-care subjects were examined via Wilcoxon rank sum testing (Table 3). Nutrition and activity scores were similar for the two groups, but comparison of the remaining Braden subscale scores demonstrated that the long-term-care subjects were more impaired in sensory perception, moisture, mobility, and friction/shear. However, the differences were once again small in terms of clinical significance. The sample size had sufficient power to detect small differences, so that even though statistical significance was observed the differentials were quite small and perhaps clinically insignificant.

**Table 3** *Braden Subscale Means in Acute Versus Long-Term Care*

	Acute Care ( <i>n</i> = 232)	Long-Term Care ( <i>n</i> = 108)	<i>p</i> value
Sensory Perception	3.9	3.5	<0.0001
Moisture	3.7	3.4	<0.005
Activity	2.9	3.0	0.23
Mobility	3.9	3.0	<0.0001
Nutrition	3.0	3.0	0.38
Friction/Shear	2.6	2.2	<0.0001

Table 4 presents the differences between subjects with and without PU in terms of Braden subscale means. Four of the six Braden subscale means (nutrition, activity, mobility, and friction/shear) were statistically associated with PU development, while no relationship with incidence was demonstrated for sensory perception and moisture. Again, the sample size allowed for detection of all but the smallest of differences, so statistical significance should be interpreted with caution.

**Table 4** *Relationship of Braden Scale Items to PU Development*

	Mean (Subjects Without PU) ( <i>n</i> = 298)	Mean (Subjects With PU) ( <i>n</i> = 32)	Wilcoxon rank sum <i>p</i> value
Nutrition	2.9	2.4	0.0003
Sensory Perception	3.7	3.6	0.61
Moisture	3.6	3.4	0.13
Activity	2.8	2.3	0.0001
Mobility	3.4	2.9	0.0003
Friction/Shear	2.4	2.0	0.0003

#### *Sensitivity and Specificity of the Braden Score for PU Development*

Only half of the 32 subjects with PU had Braden scores indicative of increased risk. Sensitivity, specificity, and positive and negative predictive values of the Braden Scale in predicting all stages of PU were infe-

rior to that previously reported (Table 5). Overall sensitivity in this sample using the recommended cut-off of 16 was 22%, while specificity was 86%. The positive predictive value of the Braden Scale using the recommended cut-off was 15%, while the negative predictive value was 91%. Table 5 provides these predictive values at various Braden scores. The Braden score found to have the best balance of sensitivity (69%) and specificity (55%) was a cut-off of 19, although even this cut-off provided relatively weak performance characteristics for predicting PU in individual patients.

**Table 5** *Sensitivity and Specificity of the Braden Scale for Varying PU Risk Cut-Off Values*

Braden Score PU Risk Cut-off	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)
20	81	46	14	96
19	69	55	14	94
18	47	68	14	92
17	38	80	17	92
16	22	86	15	91
15	9	91	10	90
14	9	94	15	91
13	9	97	23	91
12	9	98	22	91
11	0	98	0	90

### **Prevention Strategies**

An average of 3.3 PU prevention strategies were used for patients classified as *no risk*, while significantly (*t* test,  $p < .001$ ) more strategies (6.4) were used for patients with scores of  $< 19$  on the Braden Scale (up to a maximum of 11 strategies per subject). The number of prevention strategies used was correlated (Pearson  $r = -.596$ ,  $p < .001$ ) to Braden scores. Patients with a minimum Braden score indicating *risk* for PU development ( $\geq 19$ ) averaged twice as many prevention strategies as patients *not at risk* (4.0 versus 2.0, respectively, Wilcoxon  $p$  value  $< 0.0001$ ). The mean number of prevention strategies per subject increased by approximately one, from 5.2 to 6.3, following the development of an ulcer.



The number of prevention strategies used tended to be greater for older patients ( $r = .24$ ,  $p < .001$ ). Patients aged 75 or older had slightly more prevention strategies (2.7) than patients under age 75 (2.2) (Wilcoxon  $p$  value = 0.01). The number of prevention strategies used was comparable across genders (Wilcoxon  $p$  value = 0.49).

Prevention strategies were ranked in order of frequency of use. Those most frequently documented were (in descending order): use of a pressure-reduction mattress, use of a barrier cream, diapering, and use of a walker. Frequencies were the same regardless of whether the patient's Braden score indicated risk or whether the patient had a ulcer. A turning schedule was documented for only 38.6% of the *at risk* subjects. Patient and family teaching regarding PU was documented in only 4.4% of cases. A greater number of prevention strategies per subject was documented in the long-term-care settings ( $\bar{x} = 3.7$ ) than in the acute-care settings ( $\bar{x} = 2.7$ ).

A logistic regression modelling process was undertaken to examine the prognostic power of the collected variables for predicting PU in individual patients. From the above-reported univariate analyses, the variables recording the patient's age and total number of prevention strategies used prior to PU observation would seem to be useful supplements for the minimum Braden score in predicting which patients would develop PU. The Braden score at admission was also included, as suggested in the literature. The modelling results are not sufficient for practical purposes, because 90% of the subjects did not have PU. No model produced predicted more than 75% of the cases. Hence, if we just assumed no patients would develop PU we would be correct more often than any of the models constructed empirically. The knowledge derived from this modelling process was therefore used to produce a relative ranking of association of the variables with PU development. Models were run both with and without the gender variable, as the literature suggests that gender may be an important covariate. In none of the models did gender appear to be a useful prognostic factor for PU development. Stepwise modelling resulted in a model that incorporated only the total number of prevention strategies used prior to PU appearance as a prognostic factor for PU development. The odds ratio of PU development was 1.35 per prevention strategy used (chi square  $p$  value = 0.0005). This result supports our contention that the prevention strategies were used more as a prophylactic measure than as a reaction to PU development. None of the other variables (minimum Braden score, Braden score at admission, age) contributed significantly to the predictive power of the model, which correctly predicted 75% of the cases. A saturated model was subsequently used to force the entry of all vari-

ables into the model. The saturated model estimate for the effect of the number of prevention strategies remained unchanged and produced a lower correct prediction percentage. Hence the Braden score and age collectively had lower predictive power for PU than the number of prevention strategies implemented. If the number of strategies used was removed from the model, both age and Braden score became useful predictors. This model correctly predicted only 68% of the cases. The Braden score upon admission added no prognostic value to the modeling process.

### **Discussion**

The incidence of PU (9.7%) in the present study is comparable to that reported for other studies. Overall incidence rates among various sites range from 9.0% to 12.0% (Bergstrom, Braden, Kemp, Champagne, & Ruby, 1996; Langemo et al., 1991). The acute-medicine and geriatric units of the tertiary-care facilities in this study had an incidence of 10.36%, while the long-term-care settings had an incidence of 8.0%. Tertiary-care incidence rates range from 7.4% to 15.0% (Bergstrom et al., 1996; Langemo et al.), while rates in skilled-care facilities and nursing homes are reported to be between 3.4% and 28% (Bergstrom et al., 1996; Langemo et al.; Leshem & Skelskey, 1994).

Thirty-two subjects developed a total of 62 ulcers within 3 months of admission. By the end of the first week, almost half of all subjects who would eventually develop PU had done so, making this first week of hospitalization an especially critical period for both skin assessment and implementation of prevention and treatment measures. Three quarters of the ulcers that were present over the 3-month data-collection period had developed by the end of the fourth week. The time frames for PU development suggested by the present study are somewhat different from those reported by Bergstrom and Braden (1992) and Langemo et al. (1991), who found that 77–80% of subjects developed ulcers within 2 weeks of admission. In the study by Bergstrom and Braden, 92% of the ulcers had developed by the third week.

In terms of ulcer characteristics, our findings are consistent with the ranking of the most common PU sites reported in a recent national prevalence study with 39,874 patients (Barczak, Barnett, Childs, & Bosley, 1997). Sacral ulcers comprised 39% of the total ulcers reported for that study, followed by heel ulcers at 28%. In their study, Bergstrom et al. (1996) found that sacral/coccygeal PU comprised almost 60% of ulcers. More than half (57%) of the ulcers detected in the present study were classified as Stage 1. This is a higher proportion than the previ-

ously reported range of 29.8% to 38% (Barczak et al.; Bergstrom et al., 1996; Maklebust & Magnan, 1994). Stage 2 ulcers were the next most common, at 30%, while stages 3 and 4 accounted for only 4.8% each. The incidence of Stage 2 ulcers in the present study was markedly lower than the 67.3% reported by Bergstrom et al. (1996) but was similar to the 37.5% reported by Maklebust and Magnan and the 39% reported by Barczak et al. The remaining 10% of ulcers in the present study were classified as stages 3 and 4, falling within the range of the 0% reported by Bergstrom et al. (1996) and the 17% cited in the national prevalence study.

Nine of the 12 subjects categorized as *high risk* were acute-care patients. However, a significantly higher proportion of *moderate* and *low risk* subjects, and fewer *no risk* subjects, were in long-term care than in acute care. This finding may reflect the relative stabilization in health status that has occurred by the time a patient enters a long-term-care facility.

Our results support the use of risk-assessment instruments such as the Braden Scale in differentiating between groups of patients in terms of indicating PU development. We found that patients who developed PU did have a lower average risk-assessment score, and the scores did dip slightly just before the PU appeared. The collective average Braden score immediately prior to PU development was almost two points lower on average than the scores of patients who did not develop ulcers. A similar difference in scores is reported by Bergstrom and Braden (1992): mean score of 16.3 for subjects without PU; between 14.1 and 14.5 for subjects with PU.

However, the risk-assessment scores were not successfully prognostic in predicting ulcer development in individual patients. Only half of the patients classified as *at risk* actually developed PU. The scores of the individual subjects changed only very minimally over time, and, furthermore, did not change when a PU developed. These findings suggest that risk-assessment scales alone may not be sensitive to the changes in status that can predispose a patient to an ulcer. Collectively, our results produce an answer for research question #1, indicating that it is not reasonable to expect a simple risk-assessment score to accurately predict PU in individual patients. Risk-assessment scores, then, would seem to have an associative rather than a prognostic role in PU. As such, they can be useful in the context of a comprehensive prevention strategy, in identifying patient subpopulations that may be at greater risk for PU.

In the acute-care setting the mean Braden scores were significantly lower for subjects with PU ( $\bar{x} = 18.1$ ) than without PU ( $\bar{x} = 20.0$ ). This difference disappeared, however, in the long-term-care setting, where the mean Braden scores were actually marginally higher for subjects with PU ( $\bar{x} = 18.41$ ) than without PU ( $\bar{x} = 18.13$ ). It may be that an exogenous variable was especially significant in the development of ulcers in the long-term-care population.

In terms of the relationship of subscale means to PU development, four of the Braden subscales (nutrition, activity, mobility, and friction/shear) were associated with PU development, although the magnitude of the differences was of questionable clinical value. The sensory perception and moisture subscales did not demonstrate an association with PU development. This may reflect the fact that only one third of the sample were impaired in either risk factor, although it seems more likely that these findings support the possibility that models of PU development containing alternative variables are better predictors (Brandeis et al., 1994, 1995; Maklebust & Magnan, 1994; Rudman et al., 1993).

In the current study, the total Braden score that appeared to have the best balance of sensitivity (69%) and specificity (55%) was 19. These results are somewhat lower than but comparable to those of Harrison et al. (1996) and Salvadalena et al. (1992). Langemo et al. (1991) reported that optimal sensitivity (64%) and specificity (87%) were attained at a score of 15 for acute-care settings. For the current study, sensitivity was 9% with a specificity of 91% at a score of 15. This is an unacceptably low rate of accuracy in predicting PU, with the results of the current study being less favourable than those of previous studies using the Braden Scale (Bergstrom, Braden, et al., 1987; Bergstrom, Demuth, et al., 1987; Capobianco & McDonald, 1996).

Harrison et al. (1996) identify a number of factors that could account to some extent for the poor sensitivity and specificity of risk-assessment scores in their study: a large range of patient ages, diagnoses, and severity of condition; cross-sectional design; and varying levels of nursing care and staff between units ranging from critical to long-term care. Similar issues arise in the current study with respect to range of diagnoses, severity, and varying levels of nursing care, but not for age or cross-sectional design. Any of these issues may have contributed to the results. It may, however, be simply an unreasonable expectation for a simple single associative index to have substantial prognostic power.



Capobianco and McDonald (1996) found that the Braden Scale failed to identify four of 14 patients who developed ulcers. They suggest that patients with poor nutrition may be missed by the Braden Scale. In the present study, only three of the 15 subjects who developed ulcers were considered to have poor nutrition, so the poor sensitivity and specificity of the Braden Scale cannot be attributed solely to problems with the nutrition subscale.

In the present study, patients who were *at risk* according to the Braden Scale were found to have significantly more prevention strategies in place than patients who were *not at risk*. In fact, the number of preventive strategies increased upon appearance of the ulcer; this might indicate that the prevention strategies were sometimes used in reaction to the development of PU rather than as a prophylactic measure. It may be that prevention strategies were used to prevent secondary ulcer development, and that these strategies were highly effective in this group, thus possibly accounting for the low sensitivity of the Braden Scale in accurately predicting PU. However, it is difficult to account for the *not at risk* subjects developing ulcers other than by suggesting that fewer prevention strategies were in place for *not at risk* than for *at risk* subjects. This type of reasoning tends to become circular and is not helpful in the clinical setting. The reality is that in terms of our second research question the use of prevention strategies is related to PU incidence rates, although the directionality of the relationship in the clinical settings studied is in question.

A limitation of the study may be the reliance upon staff nurses' identification of new ulcers, particularly Stage 1 ulcers. However, as more than half of the ulcers identified were Stage 1, this does not represent a large concern.

Another limitation relates to interrater reliability testing. Interrater reliability between the three research assistants was not formally assessed, but the 10 patient assessments of each research assistant were reviewed and approved by an expert nurse (YL) prior to data collection.

A further limitation is the lack of availability for some of the potentially concomitant confounding influences. The prevalence of protein-calorie malnutrition is as high as 50% in some health-care settings (Strauss & Margolis, 1996), yet albumin levels (a valuable gauge of nutritional status) were not ordered for the vast majority of patients in this study. Strauss and Margolis note that in multiple cross-sectional and longitudinal studies demonstrating that malnourished people are at greater risk for PU, zinc levels were absent for all subjects. Albumin levels were absent in 88.2% of cases, but the average for available sub-



jects was 32.3 g/l ( $N = 33.45$  g/l). Data on height and weight were not present in the health-care record for half of the subjects. While these data represent potential sources of bias, and while they may have been helpful in building a more comprehensive prognostic model for PU development, there is no evidence to suggest that their inclusion would have altered the basic findings.

A brief comment on the choice of assessment tool is in order to clarify our purpose in this study. It was our goal to examine the degree to which a risk-assessment tool, in concert with prevention strategies and associated demographics, could predict PU. We chose the Braden Scale because it is recognized as one of the best instruments available for PU risk assessment. The primary point to be made here is that our findings indicate the need for a comprehensive approach to predicting PU rather than relying on a simple single index. It is doubtful that our results would be different had we used any other risk-assessment tool. The data indicate the context within which risk-assessment tools such as the Braden Scale can be applied to aid in PU prevention. The results are not an indictment of the Braden Scale, nor do the data suggest that the tool itself is flawed in any way. As per the AHCPR guidelines, the data indicate that risk-assessment tools are an important *part* of a prevention program but cannot stand alone in predicting PU in individual patients.

### **Conclusions**

Accurate prediction of PU is a highly complex endeavour. This conclusion was borne out strongly by our data. In terms of our specific research questions, it is clear that a simple risk-assessment tool alone cannot accurately predict ulcer development. Further, while prevention strategies may indeed alter the incidence of PU, it may well be that many prevention strategies are not employed until PU is imminent and are applied more as a reactive rather than as a prophylactic intervention.

It is not surprising that a simple index encounters difficulty in accurately predicting the development of PU. Besides the patient's functional characteristics described by risk-assessment scales, it may be that variables such as medical status, social support, environment, and quality of care need to be entered into the predictive equation. No doubt there are many individualistic variables that confound the prediction effort. Further work along this line to supplement risk assessments in the development of a reliable, sensitive, and specific prognostic model for PU development in individual patients is indicated.

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### **Acknowledgement**

This research was supported by a grant from the Manitoba Health Organisation.