
Risk Factors for Pressure Ulcers – Can They Withstand the Pressure?

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Abstract

Evidence for risk factors for pressure ulceration was graded as follows:

A: supported by two or more prospective studies;

B: supported by prospective or retrospective studies;

C: anecdotal evidence, case reports or studies which do not all support the risk factor but consensus expert opinion provides support, or

D: conflicting evidence not supported by consensus expert opinions.

Among the Pressure Ulcer Interest Subcommittee of the Australian Wound Management Association (AWMA), expert consensus was reached: no risk factors were supported by evidence graded A; reduced mobility and activity, shear and friction and older age were supported by evidence graded B; moisture (especially faecal incontinence), nutritional factors, vitamin C status, oxygen delivery, diabetes, dry skin and previous pressure ulceration were supported by evidence rated C, and a rating of D given evidence supporting zinc status, male gender, race, temperature (skin and core), cancer and other chronic illnesses, recent hospital transfer, use of antipsychotic medications and psycho-social factors. Evidence-based preventive approaches will be most cost-effective but more data must be gathered to determine whether evidence for risk factors not currently well-supported can be strengthened.

Introduction

Factors which expose skin to excessive pressure or diminish tolerance to pressure are risk factors for pressure ulceration. A large body of literature on this topic has developed, with numerous risk factors described. However, a critical appraisal of the evidence for or against risk factors is rarely included. Some so-called risk factors simply make good sense, so, despite a lack of supportive evidence, they are assumed to indeed be risk factors. Others are supported by very weak evidence, such as one multi-variate analysis. Many risk factors are not independent of other factors and may simply be markers of the presence of other, more fundamental factors. It is, however, vital to know precisely which risk factors are well-supported, since pressure ulcer prevention depends on such accurate understanding.

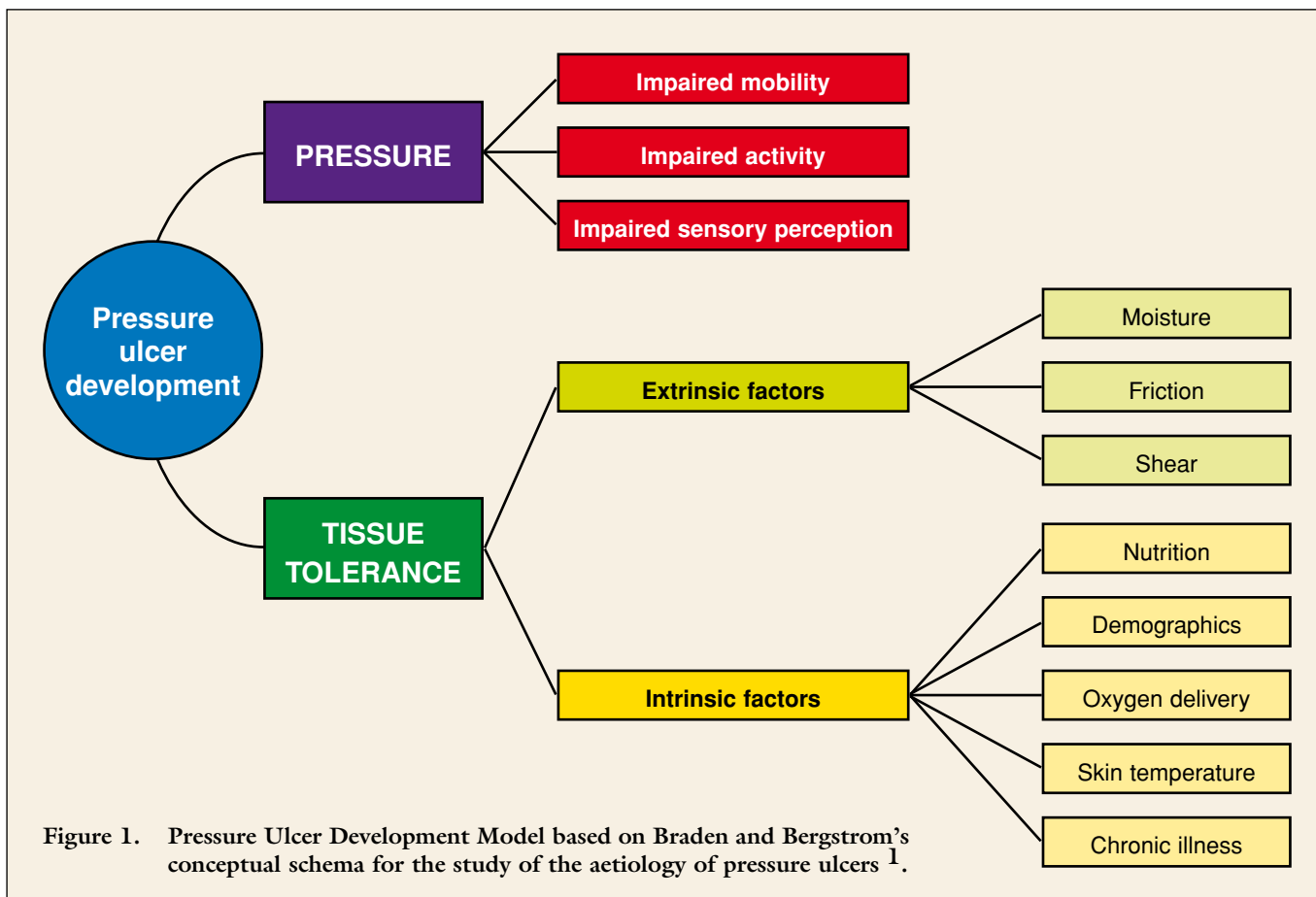
Expensive interventions to modify a weakly supported factor may be in-effective and can divert resources away from more successful approaches.

In this article, a model for risk factors will be presented, followed by an appraisal of the more recent literature, to determine how well-supported risk factors are.

Model

A framework for pressure ulcer development has been described by Braden and Bergstrom¹ (Figure 1). This model will be used in presenting the evidence for risk factors. Some of them overlap and have been combined, since the evidence usually treats them as a single risk factor; for instance, impaired mobility and activity, and friction and shear. Other risk factors in the model have been expanded or better described. Impaired sensory perception includes altered consciousness (which also can reduce mobility and activity). Surgery is also a risk factor best included under impaired sensory perception and people with fractures are included here, although immobility also contributes. Moisture is largely due to incontinence, but sweating and discharge from fistulae and wounds can contribute. Nutritional status includes vitamin C and zinc status, while demographics include age,

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gender and race. Chronic illness includes diabetes, metastatic cancer and other diseases. Other factors demonstrated in some studies but not noted in the model include dry skin and excessive skin-washing, psychological factors, previous pressure ulceration, recent hospital transfer and the use of antipsychotic medications.

Methodology

Several rating scales have been developed for the strength of evidence supporting clinical practice guidelines. However, these apply to the administration of a preventive or treatment intervention and are not as readily applicable to the strength of evidence for a risk factor. Thus, for this study, a new scale of strength of evidence was developed by the Pressure Ulcer Interest Subcommittee of the AWMA. It is summarised in Table 1.

Evidence rated A would come from prospective randomised studies demonstrating that risk factor prevention or treatment reduces the risk of pressure ulcers or facilitates healing. Evidence rated B identifies the risk factor as an independent factor in most prospective or retrospective studies, but no intervention trials have been conducted. Evidence rated C identifies the risk factor

only in some studies, generally retrospective. In the absence of sufficient supportive data, theoretical support or expert consensus is necessary for C-rated evidence. Expert consensus for the evidence rating of C in this study was reached by the members of the Pressure Ulcer Interest Subcommittee of the AWMA, with the opinion of the United States Agency for Health Care Policy and Research also considered. For evidence rated D, the

Table 1. Strength of supporting evidence.

A	Two or more prospective studies to support the evidence – good research base to support the risk factor.
B	Retrospective or prospective studies – fair research base to support the risk factor.
C	Anecdotal evidence, case reports or retrospective and prospective studies which do not all support the risk factors. Designation as a risk factor is based on expert and committee consensus.
D	Conflicting evidence not supported by consensus expert or committee opinion.

risk factor is identified in no or very few studies and conflicting evidence may exist. While experts cannot reach consensus on the role of this risk factor, further study may increase the strength of D-rated evidence. In this paper, no potential risk factors have been included if evidence for them fails to achieve a D-rating.

Research studies on evidence for risk factors were retrieved from published papers using the following methods:

- MEDLINE searches;
- hand searches of journals not referenced in computer databases;
- reference lists from review articles;
- personal files from AWMA subcommittee members, and
- recommendations from peers.

The strength of the evidence will now be rated, with justifications for this, and pivotal studies summarised.

Results

Immobility and diminished activity – strength of evidence B

Immobility and diminished activity show up as risk factors in most prospective and retrospective studies. Berlowitz and Wilking², for instance, reviewed the medical records of 301 admissions to a chronic care hospital and found that 100 had a pressure ulcer on admission. Factors significantly associated with initial presence of a pressure ulcer included being chair- or bed-bound (odds ratio (OR) = 2.4, 95 per cent; confidence intervals (CI) 1.2, 4.9). Of 185 patients without a pressure ulcer on admission in a prospective arm of their study, 20 (that is, 10.8 per cent) developed an ulcer subsequently. Factors significantly associated with this new development included, again, being chair- or bed-bound (OR = 3.8, 95 per cent; CI 1.0, 14.0) and a history of a cerebrovascular accident (OR = 5.0, 95 per cent; CI 1.7, 14.5). The latter factor could be at least partly explained by reduced mobility in stroke survivors.

Allman *et al*³ conducted a prospective study of 286 patients aged over 55 who, while free of pressure ulcers when admitted to a university hospital, were expected to be confined to a chair for at least 5 days or had a new hip fracture. Of those, 37 (that is, 12.9 per cent) subsequently developed a pressure ulcer, after a median period of 9 days from admission. The risk ratio (RR)

for the development of a stage 2 (or greater) pressure ulcer was 2.36 (95 per cent; CI 1.14, 4.85) for immobility compared with mobility.

Brandeis *et al*⁴, in a prospective study of 4232 nursing home residents free of pressure ulcers, divided the homes into ‘high-incidence’ (19.3 per cent of residents developed pressure ulcers over 21 months) and ‘low-incidence’ (6.5 per cent of residents over 21 months) facilities. The OR for development of pressure ulcers was 3.3 (95 per cent; CI 2.0, 5.3) in the high-incidence and 3.6 (95 per cent; CI 1.7, 7.4) in the low-incidence homes for those with ambulation difficulty.

Other studies⁵⁻¹⁰ have demonstrated similar findings. Pase¹⁰, in a prospective study of hospitalised elderly patients, demonstrated that the more mobile or active the individual the lower the incidence of pressure ulcers.

Sensory loss/altered consciousness – strength of evidence C

Impaired sensation or reduced ability to respond to discomfort or pain predispose the individual to prolonged and intense pressure¹. Risk factors related to this include spinal injury, impaired cognition and altered level of consciousness.

In the cross-sectional study of Berlowitz and Wilking², based in a chronic care hospital and looking at the presence of a pressure ulcer on admission, altered level of consciousness was a significant predictive factor (OR 4.1, 95 per cent; CI 2.1, 8.1). Altered consciousness was not, however, a prospective risk factor for new ulcer development, whereas cerebrovascular accident was (see above), and this may have affected sensation or level of consciousness.

Manley¹¹, in a prospective study of 772 hospitalised patients, found a higher incidence of pressure ulcers in hemiplegic (four of 23) than paraplegic (nil of six) patients, but it was unlikely these were independent risk factors; for instance, the hemiplegic patients were older. Interestingly, there was a higher incidence in semiconscious than unconscious patients, perhaps because the latter were turned routinely.

Studies in paraplegic patients have shown sensory loss as a risk factor for pressure ulceration. For instance, Rochon *et al*¹², in a retrospective chart review at a spinal cord injury referral centre, found that in 81 of the 364 patients with pressure ulcers, patients with no or only some sensation below the level of the cord lesion had an OR for pressure ulceration of 5.7 (95 per cent; CI 2.8, 11.9) compared to those with no

sensory loss. Vidal and Sarrias ¹³, in a cross-sectional study of 884 spinal cord injured patients, found complete sensory loss to be significantly associated with the presence of a pressure ulcer ($p < 0.0002$); 79.4 per cent of all ulcers were in this group, which represented 62.7 per cent of admissions.

Impaired cognitive state has also been examined in some studies for its role as a risk factor, but it is generally not found to be an independent factor. For instance, in a large nursing home study by Brandeis *et al* ⁴, disorientation was included as a potential risk factor but was not predictive in either the high or low pressure ulcer incidence homes. Decreased mental status was, however, a predictive factor in Makleburst and Magnam's study ⁸ of 270 patients with pressure ulcers.

Surgery is associated with immobility and sensory loss (anaesthesia) and is a risk factor for the development of pressure ulcers, with post-operative incidences ranging from 13 to 66 per cent ¹⁴⁻¹⁶. Factors within surgery identified as increasing pressure ulcer risk include time on the operating table ¹⁷, older age ^{17, 18}, nutritional state ¹⁸ and use of extra-corporeal circulation ¹⁷, although Papantonio *et al* ¹⁸ failed to confirm that longer bypass time increased the risk for cardiac surgical patients. Fractures requiring surgery are associated with increased risk of developing pressure ulcers – approximately two-thirds of patients hospitalised with hip fractures developed pressure ulcers, mainly on the heels, in a study by Versluisen ¹⁶. However, such patients have multiple risk factors and, although immobility is probably the major risk, its independence from these other factors has not been established. Rochon *et al* ¹² did not find the presence of a fracture a significant risk factor for the presence of a pressure ulcer; nor did Berlowitz and Wilking ² find a lower extremity fracture to be a predictive risk factor.

Moisture – strength of evidence C

Moisture is thought to alter the resilience of the epidermis to external forces. Exposure of the epidermis to moisture for prolonged periods causes maceration, making it more friable and susceptible to injury and infections. The moisture can be in the form of urine, faeces, perspiration or drainage from fistulae or wounds ¹.

Although urinary and faecal incontinence are widely cited as risk factors for pressure ulcer formation, the published evidence is conflicting. There are sound theoretical reasons for urinary incontinence being a significant risk factor, with one cross-

sectional study of 147 patients with pressure ulcers suggesting that it is ¹⁹, but its role as an independent factor has not been established. Larger studies referred to above ^{2-4, 10} have all failed to show urinary incontinence is an independent risk factor. Faecal incontinence was found to be an independent risk factor in studies by Brandeis *et al* ⁴, but only in the high-incidence nursing homes (OR = 2.5, 95 per cent; CI 1.6, 4.0) and a study by Allman *et al* ³ (as part of a univariate Kaplan-Meier survival analysis, $p < 0.05$). In the study using pooled data from five hospital-wide audits in which Makleburst and Magnam ⁸ identified 270 patients with a total of 570 pressure ulcers, faecal incontinence was a clear risk factor, with an OR of 22 (95 per cent; CI 9.20 to 52.70). Thus, faecal incontinence approaches a B rating. Although the Braden score, based on the conceptual model used here ¹, includes moisture, two large subsequent prospective studies by the authors ^{20, 21} did not separately look at exposure to moisture as a risk factor, so it is unclear whether it is an independent risk factor.

Shear and friction – strength of evidence B

Shear is highly likely to be a risk factor, since shear forces occlude capillaries and reduce tissue blood supply, causing similar effects to direct pressure. This theory is supported by sound evidence from models ²²⁻³⁰. Bennett *et al* ²³ showed that shearing forces generated in seated elderly and paraplegic patients were three times those in healthy young adults, with blood flow only a third as much, despite similar contact pressures. There is, however, little modern evidence for the independence of this risk factor; indeed, this may be because it is so soundly based that no well-managed patients are left subject to shearing forces for any significant period of time.

Likewise, friction has been demonstrated as increasing the skin's susceptibility to pressure ^{26, 29} and may cause the initial skin break that becomes a pressure ulcer; again, however, there is little modern retrospective or prospective data to clearly establish it as an independent risk factor. The reasoning behind rating these risk factors as B is given in the discussion section.

Nutrition – strength of evidence C

As the strength-of-evidence rating differs for some of the factors that can be grouped under nutrition, there are separate ratings for vitamin C and zinc status. The broad heading of nutrition includes low albumin, poor oral intake, low weight or body mass index and recent weight loss.

Defined this way, nutritional status has been shown to be an independent risk factor in several studies. In the cross-sectional arm in that by Berlowitz and Wilking², impaired nutritional intake (OR = 1.9, 95 per cent; CI 1.0, 3.7) and hypoalbuminaemia (OR = 1.8 for 10 gm/l decrease, 95 per cent; CI 1.1, 3.1) were risk factors. In the prospective incidence arm, impaired nutritional intake (OR 2.8, 95 per cent; CI 1.0, 17.9) was a risk factor. In the study by Allman *et al*³, decreased body weight (OR 2.18, 95 per cent; CI 1.05, 4.52) was a significant risk factor, as was lymphopaenia (OR 4.86, 95 per cent; CI 1.70, 13.89), which is a marker of poor nutrition. In the Brandeis *et al* study⁴, difficulty feeding oneself (OR 2.2, 95 per cent; CI 1.5, 3.3) was a risk factor, while in Makleburst and Magnam's⁸, malnutrition was a predictive risk factor. Breslow and Bergstrom³¹ reviewed several studies, concluding that the presence of pressure ulcers is associated with inadequate energy and protein intake, being underweight, low triceps skinfold measurement, low serum albumin and low cholesterol and haemoglobin levels. The prospective studies they reviewed demonstrate only that low energy or protein intake, and possibly low serum albumin, are associated with the development of pressure ulcers. However, Finucane³², in an analysis of all published studies, noted that low albumin was a risk factor in seven studies, while in five it was not. More importantly, he notes that no randomised trials of tube feeding to prevent (or treat) pressure ulcers has been completed. Thus, there is probably still insufficient support for nutritional status as a B-rated factor.

Vitamin C status – strength of evidence C

Several studies identify vitamin C deficiency as a risk factor. For instance, Goode *et al*³³, in a small prospective study of 21 consecutive elderly patients with fractured necks of femur, found that a low concentration of leucocyte vitamin C was associated with subsequent development of pressure ulcers – the mean level was 6.3 mg/10⁸ cells in those who developed pressure ulcers compared with 12.8mg/10⁸ cells in those who did not (p<0.001). However, studies measuring serum vitamin C, such as that of Bergstrom and Braden²⁰, have failed to show that this measure of vitamin C status is a risk factor, although older studies^{34, 35} tend to. No studies of supplemental vitamin C have shown that pressure ulcers are prevented by this intervention.

Zinc status – strength of evidence D

It is difficult to identify zinc deficiency accurately – the most useful level is the zinc available to the pressure ulcer itself. Serum zinc is a poor predictor of pressure ulcer risk. Bergstrom and Braden²⁰, for instance, found it was not a significant risk factor. In the study on ulcer development after fractured necks of femur, Goode *et al*³³ found plasma zinc was actually lowest in those who did not develop pressure ulcers, with the leucocyte zinc concentration similar in both those who did and did not develop pressure ulcers. Muscle zinc concentration, which may be a better measure of tissue zinc availability, correlated significantly with the leucocyte zinc concentration. Supplementation with zinc has not been shown to reduce pressure ulcer risk, although some studies have shown that zinc supplements may promote wound, and presumably pressure ulcer, healing.

Older age – strength of evidence B

Most studies identify older age as a risk factor, although they vary in the rigour with which they determine whether older age is independent from other risk factors. Manley¹¹ showed a linear relationship between increasing age and incidence of pressure ulcers, while Papantonio *et al*¹⁸ – in their prospective study of 136 patients followed after cardiac surgery, 27.2 per cent of whom developed sacral ulcers – found that older age was a risk factor. Bergstrom *et al*²¹, in their prospective study of 843 patients in a mixture of nursing homes and hospitals, found older age was a risk factor for subsequent pressure ulcer development, although Brandeis *et al*⁴ did not find it to be a risk factor in either the high- or low-incidence nursing homes they studied.

Male gender – strength of evidence D

Brandeis *et al*⁴ found maleness to be a risk factor in the high-incidence nursing homes (OR 1.9, 95 per cent; CI 1.2, 3.6). However, Bergstrom *et al*²¹ did not find gender a risk factor. Most other studies have not examined gender as a risk factor.

Race – strength of evidence D

Very few studies have examined this. Brandeis *et al*⁴ did not find race a risk factor in either the high- or low-incidence nursing homes, whereas Bergstrom *et al*²¹ did find white race to be predictive in their multi-site study.

Oxygen delivery – strength of evidence C

Conditions included under this risk factor are hypotension, anaemia, peripheral vascular disease, cigarette smoking and spinal cord autonomic dysfunction.

In a study of 60 hospitalised patients, 30 with pressure ulcers, Schubert³⁶ found that those with pressure ulcers had lower systolic blood pressures (130±17 mmHg) compared to those without pressure ulcers (140±20 mmHg; $p<0.05$). The lower systolic blood pressure correlated with low mean blood pressure and reduced post-occlusive reactive hyperaemia. Bergstrom and Braden²⁰ found lower diastolic and lower systolic blood pressure (both $p<0.001$) predicted subsequent pressure ulcer development. Pase¹⁰ found that lower diastolic blood pressure was associated with a greater risk of subsequent pressure ulcer development, but levels of significance were not given. Schubert³⁶ did not find diastolic blood pressure to be a predictor. Intra-operative diastolic hypotensive episodes were not shown to predict subsequent pressure ulcer development in the study of 125 surgical patients by Kemp *et al*¹⁷.

Blood flow recovery time after removing pressure, a probable measure of tissue oxygen delivery, was studied by Meijer *et al*³⁷. They found a slower blood flow recovery time correlated significantly with the risk of subsequent development of pressure ulcers.

Anaemia (low haemoglobin or low haematocrit) was shown to be associated with pressure ulcer presence by Berlowitz and Wilking² – mean haemoglobin was 115±17g/l in those with ulcers compared with 120±16 in those without ($p=0.02$). However, in the multiple logistic regression model, lower haemoglobin did not remain a significant factor. Breslow *et al*³⁸ compared baseline factors in tube-fed nursing home residents and found that those with pressure ulcers had significantly lower haemoglobin (117±5) compared to those without any ulcers (132±5). Rochon *et al*¹² found a haemoglobin less than 140g/l was associated with the presence of pressure ulcers (OR 2.5, 95 per cent; CI 1.5, 4.1 compared with haemoglobin above 140g/l). Other research has not shown anaemia to be a risk factor¹⁸.

Peripheral vascular disease has been shown as a risk factor in some studies. For instance, in their univariate analysis Maklebust and Magnam⁸ found its presence associated with the presence of a pressure ulcer ($p<0.001$). Few other studies have specifically included peripheral vascular disease as a potential risk factor.

Cigarette smoking has been shown in some studies to be a risk factor³⁹⁻⁴¹, but few of the larger general studies included it as a potential risk factor.

Transcutaneous oxygen tension at the sacrum, partially related to autonomic dysfunction, was measured in 21 spinal cord

injured patients by Mawson *et al*⁴², who found lower oxygen tensions in five of the 10 patients with pressure ulcers compared with only one of the 11 without ulcers ($p=0.055$).

Temperature – strength of evidence D

Both skin and core temperatures have been examined. Skin temperature elevation has been associated with pressure ulcer development in several studies⁴³⁻⁴⁷, while elevated core (body) temperature was found to be a risk factor in the prospective nursing home study of Bergstrom and Braden²⁰. The association has not been fully explained; however, it may be related to increased oxygen demand. With each degree centigrade rise in temperature there is an increase in tissue metabolism and oxygen demand of 10 per cent⁴⁷. Pressure-relieving cushions have been shown to lower skin temperature⁴⁵ but no prospective studies have examined the effect of lowering skin temperature on pressure ulcer development.

Diabetes – strength of evidence C

Most studies which include diabetes as a potential risk factor for pressure ulcers confirm that it is indeed one. Brandeis *et al*⁴ found diabetes to be a significant predictor in high-incidence homes (OR 1.7; 95 per cent CI 1.2, 2.5) but not in low-incidence facilities. In a univariate analysis, Maklebust and Magnan⁸ found diabetes a significant risk factor ($p<0.001$), but it did not end up in the best fitting predictive model developed by stepwise logistic regression.

Cancer – strength of evidence D

Only a small number of studies have included cancer as a potential risk factor. For instance, Maklebust and Magnan⁸ found metastatic cancer significant in their univariate analysis ($p=.002$) but did not include it in their predictive model. Berlowitz and Wilking² did not find cancer a risk factor.

Other chronic illnesses – strength of evidence D

Studies vary in the illnesses they include as potential risk factors. Those mentioned in reviews⁴⁸ include renal disease, arthritis, congestive cardiac failure and lymphoedema, but in studies that have examined for them these illnesses do not generally show up as significant factors. For instance, Berlowitz and Wilking² did not find congestive cardiac failure a risk factor and Papantonio *et al*¹⁸ found renal disease not to be a significant risk factor in the development of sacral sores after cardiac surgery; however, they did find respiratory illness to be a risk factor (RR 2.28, 95 per cent; CI 1.25, 4.17).

Other factors

Although not specified in the model, several other factors have been examined as risks for pressure ulceration, as follows.

Dry skin – strength of evidence C

Allman *et al*³ found dry skin a risk for subsequent pressure ulcer development (RR 2.31, 95 per cent; CI 1.02, 5.21). There is strong expert consensus on the role of dry skin as a risk factor.

Excessive skin washing – strength of evidence D

Although there is some theoretical support for this as a risk factor, there is none from published studies.

Transfer from hospital – strength of evidence D

Papantonio *et al*¹⁸ found recent hospital transfer a risk factor for the development of sacral ulcers after cardiac surgery (RR 2.08, 95 per cent; CI 1.13-3.80). Berkowitz and Wilking² did not find transfer from a hospital into the nursing home where they based their study a risk factor; however, since 87 per cent of their patients were from an acute hospital it may have been difficult to demonstrate this. Vidal and Sarnas¹³ did not find transfer from a hospital into their spinal cord unit a risk factor for pressure ulceration on admission.

Previous pressure ulceration – strength of evidence C

Several studies have found this to be a risk factor. Allman *et al*³ found previous ulceration a risk factor in univariate Kaplan-Meier survival analysis, but not after Cox regression analysis. Rochon *et al*¹² found a positive past history to be predictive (Chi square, $p < 0.01$), although it did not remain in the logistic regression model.

Use of antipsychotic medication – strength of evidence D

Brandeis *et al*⁴ did not find antipsychotic drug use a significant risk factor in either low- or high-risk homes. However, excessive sedation and the possibly reduced nutrition associated with these medications results in them being potential risk factors.

Psychosocial factors – strength of evidence D

Psychological considerations can also be risk factors; for example, stress can increase cortisol levels and this affects skin healing (as in Cushing's disease and those treated with steroids). Other potential factors include poor self-assessed health status and intellectual disability. Heilporn⁴⁹ described two paraplegic patients with psychological disabilities who experienced recurrent ulceration, and Anderson and Andberg

⁵⁰ further developed the theory. Unemployment and lack of formal education were associated with greater risk of pressure ulceration in a prospective study by Rodriguez⁴¹ of patients with spinal cord injuries. Evidence from cross-sectional or prospective trials is, however, largely lacking.

Discussion

Perhaps the most surprising finding is that no risk factor was supported by A-rated evidence. Probably the closest was immobility and diminished activity but, surprisingly, there were no well-designed and sufficiently powered prospective intervention studies demonstrating reduced risk from increased activity/mobility alone. Certainly, reducing pressure through pressure-relieving or -reducing surfaces has been shown repeatedly to be an effective intervention, but surfaces were not included as potential risk factors in this model.

It was also surprising that moisture achieved a rating of only C. The evidence was most convincing for faecal incontinence, which in addition to moisture provides bacteria and toxins that may damage skin. As with all risk factors, there may be overlap with other significant factors; for instance, immobility increases the risk of incontinence.

Shear and friction were both reasonably well-supported by models but there have been no large prospective trials in recent times, since patient management strategies aimed to reduce shear and friction. The strength of the theoretical and consensus support for these risk factors was such that an evidence rating of B was applied, even though the data is of the same strength as that supporting risk factors given lower ratings.

Nutrition has been well-analysed in the paper by Finucane³². Prospective pressure ulcer prevention trials using nutritional support as the intervention are likely to be published and positive results may move the strength of evidence to B, if not A. Indeed, randomised controlled intervention trials with any modifiable risk factor that produce positive results could strengthen the evidence for the risk factor, even if retrospective or prospective observational studies conflict or are lacking.

The weakness of the evidence for micronutrient status is perhaps surprising, as vitamin C and zinc are involved in collagen formation and tissue repair. Again, intervention studies could strengthen the evidence, but to date such studies have failed to produce positive results.

Older age is a risk factor but may not be independent of others.

Older age is associated with increased risk of hospitalisation, chronic illness, poor peripheral perfusion and loss of peripheral sensation. Age-related changes in the skin, such as loss of dermal vessels, thinning of the epidermis, flattening of the dermal-epidermal junction, loss of elastic fibres and increased skin permeability, may increase the susceptibility of the skin to breakdown⁵¹.

Male gender was a risk factor. Interestingly, female gender has been shown by Bergstrom and Braden²¹ to predict the use of pressure-relieving devices. The male propensity to pressure ulcers may be related to nutritional and other health status. Races with darker skins have been postulated by some as at increased risk of pressure ulcers, since early warning signs (non-blanchable erythema) may be less easily seen. However, this was not supported by the evidence.

Oxygen delivery has a sound theoretical basis and is also a marker of delivery of other nutrients and removal of toxic metabolic waste products. Although there are many supportive studies, the results are not always so and there have been no intervention studies. Nevertheless, the evidence for this risk factor comes very close to achieving a B rating.

Diabetes is associated with vascular disease, sensory loss, episodes of reduced mobility (various diabetic comas) and increased risk of infection. It would be interesting to see whether tighter diabetic control in the long and short term reduced pressure ulceration. The weak evidence for other illnesses is not surprising and there may have been considerable overlap with immobility and nutritional status.

Skin hygiene appears as a risk factor in most reviews but there is little support for it from published studies. Dry skin may reduce the tissues' resistance to mechanical forces such as pressure, shear and friction⁵². Excessive skin washing or the use of soap and detergents can lead to chemical and physical irritation and also compromise the skin's water-holding capacity⁵³.

Patients transferred from hospitals are generally sicker and have been exposed to a high-risk environment, so the presence or subsequent development of pressure ulcers is no surprise.

A previous history of any disease is a risk factor for subsequent recurrence, and pressure ulceration is no exception. The challenge is to identify any ongoing risk factors and modify them where possible.

Antipsychotic medications have a range of adverse effects in older people and there are many reasons to minimise their use, apart, perhaps, from reducing the risk of pressure ulcers.

The evidence for the role of psychosocial factors is not strong but should not be written off; further studies may strengthen this evidence.

Conclusion

Prevention of pressure ulcers is an important health priority, one that requires clear identification of risk factors. Much work has already been done, but further studies are needed to confirm – or indeed reject – factors with a theoretical role but as yet only weakly supportive evidence. Preventive approaches that are evidence-based will be the most cost-effective and further clarification of risk factors is essential.

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