Prevention and Treatment of Pressure Ulcers: 
*Etiology and Risk Factors* – an extract from the Clinical Practice Guideline
INTRODUCTION

Foreword

This document presents an extract of the full Clinical Practice Guideline. The methodology used to appraise research and develop the recommendations is presented in the Clinical Practice Guideline, the abridged Quick Reference Guide, and in the methodology report, all available on the International Pressure Ulcer Guideline website (www.internationalguideline.com).

The full Clinical Practice Guideline presents recommendations and summarizes the supporting evidence for pressure ulcer prevention and treatment. The first edition was developed as a four year collaboration between the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP). In the second edition of the guideline, the Pan Pacific Pressure Injury Alliance (PPPIA) has joined the NPUAP and EPUAP.

The goal of this international collaboration was to develop evidence-based recommendations for the prevention and treatment of pressure ulcers that could be used by health professionals throughout the world. An explicit scientific methodology was used to identify and critically appraise all available research. In the absence of definitive evidence, expert opinion (often supported by indirect evidence and other guidelines) was used to make recommendations. Drafts of the recommendations and supporting evidence were made available to 986 invited stakeholders (individuals and organizations) around the world. The final guideline is based on available research and the accumulated wisdom of the NPUAP, EPUAP, PPPIA and international stakeholders. In this edition of the guideline, a consensus voting process (GRADE) was used to assign a strength to each recommendation. The strength of recommendation identifies the importance of the recommendation statement based on potential to improve patient outcomes. It provides an indication to the health professional of the confidence one can have that the recommendation will do more good than harm, and can be used to assist in prioritizing pressure ulcer related interventions. Printed copies of the English version of the full Clinical Practice Guideline are available through links provided on the following websites:

NPUAP website: www.npuap.org
EPUAP website: www.epuap.org
Wounds Australia (previously Australian Wound Management Association) website: www.woundsaustralia.com.au
New Zealand Wound Care Society (NZWCS) website: www.nzwcs.org.nz
International Pressure Ulcer Guideline website: www.internationalguideline.com

Suggested Citation

The NPUAP, EPUAP and PPPIA welcome the use and adaptation of this guideline at an international, national and local level. We request citation as the source, using the following format for this extract:

Limitations and Appropriate Use of This Guideline

- Guidelines are systematically developed statements to assist health professional and patient consumer decisions about appropriate health care for specific clinical conditions. The recommendations may not be appropriate for use in all circumstances.
- The decision to adopt any particular recommendation must be made by the health professional with consideration to available resources and circumstances of the individual patient. Nothing contained in this guideline is to be considered medical advice for specific cases.
- Because of the rigorous methodology used to develop this guideline, the Guideline Development Group members believe that the research supporting these recommendations is reliable and accurate. Every effort has been made to critically appraise the research contained within this document. However, we do not guarantee the reliability and accuracy of individual studies referenced in this document.
- This guideline is intended for education and information purposes only.
- This guideline contains information that was accurate at the time of publication. Research and technology change rapidly and the recommendations contained in this guideline may be inconsistent with future advances. The health professional is responsible for maintaining a working knowledge of research and technology advances that may affect his or her clinical decision making.
- Generic names of products have been used. Nothing in this guideline is intended as endorsement of a specific product.
- Nothing in this guideline is intended as advice regarding coding standards or reimbursement regulations.
- The guideline does not seek to provide full safety and usage information for products and devices; however commonly available safety and usage tips have been included. Adverse events reported in the included research have been reported in the evidence summaries and caution statements. All products should be used according to manufacturer’s directions.

Abstract

The guideline is the result of a collaborative effort among the National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Injury Alliance (PPPIA). A comprehensive literature review was conducted on pressure ulcer prevention and treatment. A rigorous scientific methodology was used to appraise available research and make evidence-based recommendations for the prevention and treatment of pressure ulcers. Draft guidelines were made available to 986 invited stakeholder individuals and organizations/societies and stakeholder feedback was considered by the guideline developers. In the final development process, the guideline development team used a consensus voting process (GRADE) to assign strengths of recommendation. Strength of recommendations indicate the extent to which one can be confident that adherence to a recommendation will do more good than harm, and are intended to assist the health professional to prioritize interventions.

The full Clinical Practice Guideline includes 575 explicit recommendations and/or research summaries.

This extract presents background on the etiology of pressure ulcers, risk factors for pressure ulcers and risk assessment.
**Strengths of Evidence and Strengths of Recommendations**

Full explanation of the methodology is available in *Appendix 1: Guideline Methodology* in the full *Clinical Practice Guideline*. Individual studies were assigned a ‘level of evidence’ based on study design and quality. The body of evidence supporting each recommendation was given a ‘strength of evidence’. A consensus voting process (GRADE) involving all the experts formally engaged in the guideline development was used to assign a ‘strength of recommendation’ that indicates the confidence the health professional can have that the recommended practice will improve patient outcomes (i.e., do more good than harm). The overall aim of the ‘strength of recommendation’ is to help health professionals to prioritize interventions.

### Strengths of Evidence

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<td>A</td>
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<td>B</td>
<td>The recommendation is supported by direct scientific evidence from properly designed and implemented clinical series on pressure ulcers in humans (or humans at risk for pressure ulcers) providing statistical results that consistently support the recommendation. (Level 2, 3, 4, 5 studies)</td>
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<td>The recommendation is supported by indirect evidence (e.g., studies in healthy humans, humans with other types of chronic wounds, animal models) and/or expert opinion</td>
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### Strengths of Recommendation

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**Guideline Website**

http://www.internationalguideline.com

The guideline website will remain accessible during the interim period until the next guideline revision. The *Quick Reference Guideline*, sponsor acknowledgement, and supportive documents to the guideline (e.g., data extraction tables) are available from the website.
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Special thanks to the many stakeholders who reviewed the guideline processes and drafts. All stakeholder comments were reviewed by the Guideline Development Group and revisions were made based on the comments received. We appreciate the investment of health professionals, researchers, educators and manufacturers from all over the world who took time to share their expertise and thoughtful critique.
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BACKGROUND

THE ETIOLOGY OF PRESSURE ULCERS

Introduction

A pressure ulcer is a localized injury to the skin and/or underlying tissue, usually over a bony prominence, resulting from sustained pressure (including pressure associated with shear).

A number of contributing or confounding factors are also associated with pressure ulcers; the primary of which is impaired mobility.

Pressure Ulcer or Pressure Injury?

Since the first description of the injury referred to above, there has been debate regarding terminology. The oldest term is decubitus, originally described as gangraena per decubitum by Wohleben (1777), which means ‘dead tissue due to lying down’, thus referring to wounds developed by patients while in bed. Etiological research started with the work of Groth (1942) and a number of seminal studies and papers by Kosiak (1959) and Reichel (1958). Groth (1942) used the term decubitus, Reichel (1958) used decubitus ulcers and Kosiak (1959) used several terms including ischemic ulcers. None of these terms are accurately descriptive and the term used by Kosiak (1959) implies an overly limited etiological pathway.

The term bedsore arose following publication of Bedsore Biomechanics an edited book that followed the first international conference on pressure ulcer etiology held in 1975 in Glasgow. This term maintains the association with the bed, despite knowledge at the time that pressure ulcers could be acquired whenever soft tissues are in contact with supporting surfaces, and of the major role played by shear forces and shear deformation. The addition of sore implies a raw or painful place on the body.

In the 1980s the term pressure sore became more popular, thus no longer relating the injury to the bed. Since the early 1990s the term pressure ulcer, referring to an open ulcer at the skin surface that is difficult to heal or fails to heal, has been in common usage. However, this term fails to capture both deep tissue injury, an internal wound under intact skin (see Classification section of the full Clinical Practice Guideline) and Category/Stage I pressure ulcers in which skin remains intact.

All the above terms are still in use by clinicians and/or patients. In Europe and North America the term pressure ulcer is widely used. South-East Asia, Australia and New Zealand have recently adopted the term pressure injury. Although none of these terms accurately describes the full etiology behind the injury, they all refer to the same phenomenon described in the introduction to this guideline section, and terminology is still the subject of ongoing discussion. In this version of the guideline the term pressure ulcer is used.

Mechanical Load: Magnitude and Time

This section defines a number of commonly used mechanical terms.

Mechanical load comprises all types of force that are applied to an individual’s soft tissue as a result of contact between the skin and a solid surface (including air-filled or water-filled support surfaces, medical devices and other body surfaces). It includes forces carried by the bony structures and transmitted through the soft tissue to the supporting surface. Mechanical loads are often characterized as being a normal force (a force perpendicular to the skin surface) or a shear force (a force parallel to the skin surface). In most practical situations, the interacting force is a combination of a normal and a shear force.

Pressure is defined as normal force per unit surface area.
When two surfaces are in contact with each other, they can either be **fixed** (no sliding occurs between the surfaces) or they can slide over each other (in technical literature, referred to as **slip**). The occurrence of fixation or slip depends on surface properties and mechanical loading conditions (a combination of normal and shear forces).

In the technical literature, the term **friction** is used to describe all phenomena that relate to interface properties and sliding of surfaces with respect to each other. In literature related to pressure ulcers, including this guideline, friction is used to define the contact force parallel to the skin surface in case of slip (in technical literature this is called dynamic friction).

Continuous rubbing or sliding of a surface (e.g., a textile) along the skin can result in redness, inflammation or a wound referred to as a **friction blister**. These blisters are not considered to be pressure ulcers; however, they are covered by this clinical guideline because it is not always evident whether the injury to the skin resulted from frictional sliding or from pressure and shear.

When the body is in contact with a supporting surface, such as a wheelchair cushion or mattress, both normal forces and shear forces are generated between the body and the support. As a result, the loaded soft tissues, including skin and deeper tissues (e.g., adipose tissue, connective tissues, and muscle) will deform, resulting in **strain** (a measure of the relative deformation) and **stress** (force transferred per unit area) within the tissues. Excessive internal strains and stresses will hinder transport processes within the tissues (e.g., by reducing blood perfusion and affecting transport in interstitial spaces or transport through cell membranes).

The ways in which tissue is affected by the mechanical load is a complex process that depends on morphology (the size and shape of the different tissue layers) and the mechanical properties of the tissues involved (e.g., stiffness, strength and diffusion properties), as well as the magnitude and distribution of the mechanical force that is applied to the tissue at the point of contact with the supporting surface.

Morphology, mechanical properties and tissue tolerance can all change over time as a result of aging, lifestyle, chronic injury, or disease. In general, external mechanical loading will lead to a highly irregular internal tissue response (i.e., different responses at different locations). This can also be referred to as a **heterogeneous** or **nonhomogeneous** response.

Normal forces will be highly non-uniform across the supported areas in the presence of clinical conditions (e.g., a human body supported by a mattress or cushion), and some shear force always exists. Accordingly, considerable deformations and strains may occur within the skin and deeper tissues. While an individual is sitting in a chair it is common that internal strain levels in the muscle can reach values of 50% and above.  

Techniques available for assessment of internal deformation are magnetic resonance imaging (MRI), elastography, and ultrasound. These imaging modalities can be used in combination with subject-specific theoretical finite element models (a method of solving mechanical problems by means of a computer) to estimate stress and strain throughout the tissue and predict the risk of damage.

Pressure ulcers develop as a result of the internal response to external mechanical load. Understanding the etiology of pressure ulcers relies on an awareness of the internal response to mechanical load and not just what is apparent on the outside of the body or on the skin surface.

### How Tissues Respond to Different Types of Mechanical Loading

The primary cause of pressure ulcers is a sustained mechanical load that is applied to soft biological tissues, generally near a bony prominence. Pressure gradients that induce sustained deformation of skin and subdermal tissues must be present in order for tissue damage that characterizes a pressure injury to occur.

The magnitude of the mechanical load that will lead to tissue damage depends on the duration of time for which the load is applied. Both a high load for a short period and a low load applied for a prolonged period can lead to tissue damage.
**Sustained** loading refers to a load that is applied for a long duration (minutes to hours or even days). In technical terms this is called a quasi-static mechanical loading. At high tissue deformations resulting from pressure and shear, damage to the cells is visible on a microscopic level within a few minutes, although it may take hours of sustained loading to become a deep tissue injury or pressure ulcer.

**Impact damage**, which usually occurs as a result of an accident or trauma, does not fall under the definition of pressure ulcers. Within a fraction of a second a very high mechanical load is applied to the tissue. The mass of the objects plays an important role and inertia effects leading to shock/pressure waves in the tissue may cause very high external and internal damage, all within a fraction of a second. This impact injury is not considered a pressure ulcer.

The threshold function for damage developed by Reswick et al. (1976)\(^\text{17}\) depends on pressure applied to the skin and duration of applied pressure. It was developed, based on observations of superficial damage in humans. Although Reswick et al. (1976)\(^\text{17}\) indicated that the function becomes asymptotic (meaning that it goes to infinity) for short durations of applied pressure, we understand that the absolute limit on pressure magnitude is finite as shown in Figure 1. The Reswick et al. (1976)\(^\text{17}\) curve should be revised to more accurately reflect the risk of tissue damage at the extremes of very short and very long loading times. High loads can almost instantaneously cause damage to tissues at a microscopic level, which can be made visible with MRI or histological techniques. Conversely, very low loads will not lead to damaged tissues even if applied for extended periods of time.

Due to variability in individual tolerance and confounding factors, it is not possible to determine quantitative values for damage thresholds as a function of time and pressure. Therefore Figure 1 does not feature a scale along the axes.\(^\text{14, 17, 18}\) An example of an extrinsic confounding factor that has been shown to have a profound effect on tissue’s tolerance to pressure damage is temperature.\(^\text{19}\) Another intrinsic confounding factor may be arteriole insufficiency related to diabetes.

*Figure 1: Proposal for pressure/time curve according to Linder-Ganz et al. (2006)\(^\text{14}\), Gefen et al. (2008)\(^\text{20}\) and Stekelenburg et al. (2007)\(^\text{18}\)*

Minimizing pressure at the interface between the body and the supporting surface is a valid clinical intervention for reducing the risk of developing a pressure ulcer.\(^\text{21}\) However, pressure alone is not a reliable measure for risk of tissue breakdown. Thus, a damage threshold based on interface pressure alone is not appropriate.\(^\text{5, 22-29}\)

High shear forces at the interface between body and supporting surface can exacerbate the damaging deformation caused by normal stresses alone.\(^\text{3, 11, 29, 30}\) Internal stresses and strains adjacent to bony prominences are substantially higher than those near the surface, and have the potential to cause damage in deep tissues before the superficial tissue is damaged.\(^\text{5, 23, 24, 26-28, 31-33}\)
Friction may disturb the barrier function of the stratum corneum, and therefore represents an extra danger for infection to occur concurrently with pressure ulcers.\textsuperscript{11, 34}

**Mechanisms That Lead to Tissue Damage**

An increasing body of evidence suggests two physiologically relevant deformation thresholds exist. One is a lower threshold leading to occlusion of blood vessels resulting in ischemia-induced damage and the other is a higher threshold leading to direct deformation-induced damage.\textsuperscript{35-41}

Ischemia as a result of sustained deformation of soft tissues will lead to hypoxia, blocking of nutrient supply, and blocking of the removal of waste products. Deprivation of nutrients and change in pH due to waste products will eventually lead to tissue damage.\textsuperscript{10-13, 42, 43}

The duration of time for which tissue cells can endure ischemia without damage differ for muscle, fat, and skin. Muscle tissues are more susceptible to damage than skin tissues.\textsuperscript{10, 15, 44} Skin is much stiffer than muscle and fat and therefore deforms to a lesser degree in most clinical applications. In animal experiments, the first signs of ischemic damage are found in skeletal muscle after two to four hours of sustained deformation.\textsuperscript{10-13, 38, 39, 42, 43}

Muscle deformation at strains higher than 50\% will almost immediately (within minutes) lead to tissue damage at a microscopic scale. At these strains there is a strong correlation between magnitude of the strain and the amount of damage to muscle. It is not clear yet what causes this direct deformation damage. Hypotheses include a direct rupture of the cytoskeleton, stretching of the plasma membrane or internal pathways that cause cell death.\textsuperscript{8, 9, 16, 18, 35, 45}

The balance in the interstitial space, where transport of nutrients and waste products takes place, is critical for healthy tissue homeostasis. Specifically, diffusion of nutrients, waste products, and hormones that regulate muscle metabolism may be hindered by mechanical loading.\textsuperscript{37, 46, 47} Recent laboratory and computational modeling work suggest that the localized sustained large deformations in weight-bearing soft tissues under bony prominences translate to large cellular deformations at the micro-scale and cause distortion of cellular organelles, for example considerable stretching of cellular plasma membranes. The prolonged exposure to large tensional plasma membrane strains may interfere with normal cellular homeostasis, primarily by affecting transport through the plasma membrane which could become more permeable when it is highly stretched. This has been visualized and quantified in cell cultures subjected to physiologically-relevant deformations for periods of two to three hours, using biomolecular fluorescent markers.\textsuperscript{48, 49}

Cell death and tissue necrosis cause local alterations of the mechanical properties of the injured tissues that can in turn distort the distribution of strain and stress, and are likely to exacerbate the injury.\textsuperscript{32, 33, 50} Reperfusion that follows a period of prolonged ischemia may increase the degree of tissue damage because it involves release of harmful oxygen free radicals.\textsuperscript{51-56}

An increasing body of evidence suggests that the microclimate between skin and the supporting surface plays a role in the development of Category/Stage I and II pressure ulcers. Microclimate refers to the humidity and temperature. With an increase in humidity and temperature, the skin becomes weaker (more vulnerable) and less stiff. Excessively dry skin becomes more brittle and liable to break. The importance of these issues and the characteristics of an optimal microclimate are still a matter of debate and ongoing research.

Difference in etiology of superficial pressure ulcers and pressure ulcers in deeper layers continues to be debated. Superficial ulcers may be primarily caused by high shear at the skin surface, while deeper ulcers could result from high pressure at the surface over bony prominences. Although some studies support this proposition, the current evidence is minimal and the precise response of skin to high shear deformation is not yet fully understood.\textsuperscript{57}
Factors That Influence Susceptibility to Pressure Ulcers

A number of factors that may influence an individual’s risk of developing pressure ulcers have been described in relevant research and are discussed in the Risk Factors and Risk Assessment section of this guideline.

Figure 2: Factors influencing the susceptibility of an individual for developing pressure ulcers (adapted from Oomens (1985)\textsuperscript{58}, used with permission in 2009 guideline, continuing work produced this modification which is published in Coleman et al. (2013)\textsuperscript{59}; reproduced with permission)

References


Introduction

Risk assessment is a central component of clinical practice aimed at identifying individuals susceptible to pressure ulcers in order to target appropriate interventions and prevent pressure ulcer development. The following section of the guideline addresses risk factors for pressure ulcers and risk assessment in adult populations. The Special Populations: Pediatric Individuals section of the full Clinical Practice Guideline addresses risk factors and risk assessment in neonates and children.

Individuals with activity/mobility limitations are at risk of developing pressure ulcers (see also the Etiology of Pressure Ulcers section of this guideline). The challenge in clinical practice is to identify individuals with characteristics that increase the probability of pressure ulcer development. Individuals who are at high risk are those characterized by multiple risk factors that affect both the mechanical boundary conditions (i.e., the type, magnitude, time and duration of the mechanical load) and the susceptibility and tolerance of the individual (i.e., individual mechanical properties, geometry, physiology and repair, and transport and thermal properties of the tissues), as detailed in Figure 1. Examples of high risk individuals include:

- older adults,
- those who have experienced trauma,
- those with spinal-cord injuries (SCI),
- those who have sustained a fractured hip,
- those in long-term homes or community care,
- the acutely ill,
- individuals with diabetes, and
- those in critical care settings.

Figure 1: Factors influencing the susceptibility of an individual for developing pressure ulcers (adapted from Oomens (1985)\(^1\), used with permission in 2009 guideline, continuing work produced this modification which is published in Coleman et al. (2013)\(^2\); reproduced with permission)
Epidemiological research has increased considerably in recent years, providing a better understanding of risk factors important in the development of pressure ulcers for each individual. In addition, there has been a plethora of risk assessment tools developed for use in clinical practice, with clinical evaluation of their ability to identify those at high risk. This literature has been systematically reviewed in order to address the following questions:

1. What characteristics of the individual increase the probability of pressure ulcer development?
2. Does the use of risk assessment tools confer any benefits in the prevention of pressure ulcer development over clinical judgment?
3. What is the reliability of risk assessment tools?
4. What is the validity of risk assessment tools?

The methodological considerations for epidemiological studies used to identify risk factors and studies to determine the reliability and predictive validity of risk assessment tools differ from those of the intervention studies used throughout much of this guideline. There are, therefore, distinct review methods to address questions 1, 3 and 4 as stated above, with an exception made to guideline statement developments which are based on two published systematic reviews and updated literature. Refer to Appendix 1: Guideline Methodology section of the full Clinical Practice Guideline and the summary of evidence below.

General Recommendations for Structured Risk Assessment

1. Conduct a structured risk assessment as soon as possible (but within a maximum of eight hours after admission) to identify individuals at risk of developing pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = 

2. Repeat the risk assessment as often as required by the individual’s acuity. (Strength of Evidence = C; Strength of Recommendation = 

3. Undertake a reassessment if there is any significant change in the individual’s condition. (Strength of Evidence = C; Strength of Recommendation = 

These statements are based on expert opinion. Due to the burden and impact of pressure ulcer development on both the individual and the health service, it is accepted practice that risk assessment should be undertaken on individuals, with the aim of identifying those who are at potential risk in order that individualized preventive interventions can be planned and initiated. Risk assessments should be conducted as soon as possible, but within a maximum of eight hours after admission (i.e., at first contact with the health professional) or at first visit in community settings.

An individual’s level of pressure ulcer risk may change with alterations in health status. These changes may occur over time, and should be monitored regularly. Sudden changes in the individual’s condition may result in increased risk and vulnerability to pressure damage. Health professionals must be alert and identify changes in the level of risk, as prevention strategies may need to be intensified accordingly.

4. Include a comprehensive skin assessment as part of every risk assessment to evaluate any alterations to intact skin. (Strength of Evidence = C; Strength of Recommendation = 

This statement is based on expert opinion and indirect evidence. As noted in the full Clinical Practice Guideline section, Skin and Tissue Assessment, a comprehensive skin assessment should be part of every risk assessment. Skin and risk assessment are inextricably linked. There is strong epidemiological evidence that alterations in skin status are associated with both the progression of existing pressure ulcers and the development of new pressure ulcers, making skin assessment an essential part of any risk assessment. (See below for a discussion of skin status as a risk factor for pressure ulcers). Results of a comprehensive skin assessment are also essential in developing an individualized plan for prevention.

5. Document all risk assessments. (Strength of Evidence = C; Strength of Recommendation = 

This statement is based on expert opinion. Accurate documentation is essential. Documentation of risk assessments ensures communication within the multidisciplinary team, provides evidence that care planning is appropriate, and serves as a benchmark for monitoring the individual’s progress.4-6

6. Develop and implement a risk based prevention plan for individuals identified as being at risk of developing pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ⬤ ⬤)

Caution: Do not rely on a total risk assessment tool score alone as a basis for risk based prevention. Risk assessment tool subscale scores and other risk factors should also be examined to guide risk-based planning.

This statement is based upon expert opinion. Once individuals are identified as being at risk of pressure ulcer development, a prevention program should be developed that aims to minimize the impact of factors identified as increasing the individual’s pressure ulcer risk. Failing to provide appropriate prevention strategies when an individual has been identified to be at risk of pressure ulcer development is a failure in the duty of care owed by the health professional and can be deemed as negligence, except in situations where pressure ulcer prevention strategies are not consistent with the individual’s wishes (see the Special Populations: Individuals In Palliative Care section of the full Clinical Practice Guideline). The rationale of care should be explained to the individual and the agreed plan of care documented.

Recent research on pressure ulcer prevention has focused on programs to reduce risk. Risk reduction programs combine risk assessment with components tailored to the individual’s unique risk profile. The section Implementing the Guideline: Facilitators, Barriers and Implementation Strategy of the full Clinical Practice Guideline provides a comprehensive overview on the effectiveness of risk reduction programs and components to consider in their implementation.

Total risk assessment tool scores provide general information on risk status and level of risk. Pressure ulcer incidence progressively increases with increasing level of risk based on Braden Scale total scores.7-8 Total Braden Scale scores9-15 and Norton Scale scores16 have emerged as statistically significant factors in some multivariable models. However, total scores do not provide sufficient information for developing individualized risk-based prevention plans and do not assess all relevant risk factors. Subscale scores and other risk factors should also be examined to guide risk-based planning and more effective utilization of resources.

Structured Risk Assessment

1. Use a structured approach to risk assessment that is refined through the use of clinical judgment and informed by knowledge of relevant risk factors. (Strength of Evidence = C; Strength of Recommendation = ⬤ ⬤)

There is no universally agreed best approach for conducting a risk assessment; however, expert consensus3 suggests that the approach be ‘structured’ in order to facilitate consideration of all relevant risk factors. This guideline provides a summary of key considerations in a structured risk assessment. The first approach involves consideration of characteristics of the individual that increase the probability of pressure ulcer development that have been identified through a comprehensive review of current epidemiological evidence. The second involves consideration of risk assessment tools that incorporate many, but not all, relevant risk factors. Regardless of the structured approach used, clinical judgment is a necessary component of any risk assessment.

Risk Factor Assessment

Our systematic search of the literature to address the question of what characteristics of the individual increase the probability of pressure ulcer development identified one systematic review4 comprising 54 studies5-62 and a further 15 risk factor studies6,12,63-75 identified in the guideline search update. Factors that
have been explored and that emerge in multivariable risk factor modeling as statistically independent risk factors are reported in this section of the guideline.

A review of the epidemiological evidence identifies a number of key risk factor domains associated with the development of pressure ulcers. The literature provides a basis for generic guideline statements regarding risk factor domains that are important in pressure ulcer development. However, the large number of risk factor descriptors utilized in the 69 cohort studies that were identified provides a confusing landscape in terms of how some of the risk factors may be assessed in the clinical setting.

In practice, risk assessment tools have been developed to provide structure and operational definitions for assessment of many of the key risk factors, and these are supplemented by advanced and specialized knowledge which informs clinical judgment. However, it is also acknowledged that some risk factors are not currently considered or operationally defined (e.g., perfusion and oxygenation, as discussed below) and translation into practice requires further development work. In addition, the strength and quality of evidence is variable for each risk factor. Risk factors are presented according to their supporting strength of evidence. Any structured approach to risk assessment should consider all these factors.

1. Use a structured approach to risk assessment that includes assessment of activity/mobility and skin status. (Strength of Evidence = B; Strength of Recommendation = 66)

   Activity and Mobility Limitations

   1.1. Consider bedfast and/or chairfast individuals to be at risk of pressure ulcer development. (Strength of Evidence = B; Strength of Recommendation = 66)

   1.2. Consider the impact of mobility limitations on pressure ulcer risk. (Strength of Evidence = B; Strength of Recommendation = 66)

   These statements are underpinned by high quality epidemiological evidence, bioengineering principles/research and the etiological framework. Immobility descriptors emerge consistently in multivariable modeling demonstrating a strong statistical association between activity and mobility limitations and the development of new pressure ulcers (see Table 1).

   Being bedfast or chairfast are usually described as limitations of activity. A reduction in an individual’s frequency of movement or ability to move is usually described as having a mobility limitation. In terms of the underlying conceptual framework, mobility and activity limitations directly impact upon mechanical boundary conditions (see Figure 1) and exposure to pressure, shear and resulting frictional forces.

   Epidemiological studies consistently identify that activity/mobility limitations increase the probability of pressure ulcer development (Level 2 and 4 studies). Overall, 48 studies entered one or more measure of mobility/activity into multivariable modeling and in 34 (70.8%) of these studies a measure of activity/mobility emerged. Indicators of activity/mobility limitations include descriptors, scales and measures (see Table 1) indicative of exposure to abnormal mechanical loads including:

   - Mobility/activity related activities of daily living (ADLs).
   - The mobility subscale of a risk assessment tool.
   - Descriptors of activity (such as bed/chairfast) or immobility.
   - Factors that affect mobility.
   - General ADLs.
   - The friction and/or shear subscale of a risk assessment tool.
   - The activity subscale of risk assessment tool.
   - Interface pressure.
Table 1: Summary of evidence for measures of mobility/activity as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mobility/activity related activities of daily living (ADLs)</td>
<td>55.5% (5 of 9 studies)</td>
<td>Studies which risk factors were significant in the model24, 46, 51, 53, 65</td>
</tr>
<tr>
<td>Mobility subscale of a risk assessment tool</td>
<td>52.9% (9 of 17 studies)</td>
<td>Studies in which risk factors were significant in the model8, 18, 20, 30, 31, 38, 39, 48, 61</td>
</tr>
<tr>
<td>Descriptors of activity (e.g., bed/chairfast, immobile)</td>
<td>50.0% (7 of 14 studies)</td>
<td>Studies in which risk factors were significant in the model27, 21, 41, 44, 45, 54, 65</td>
</tr>
<tr>
<td>Factors affecting mobility</td>
<td>50% (12 of 24 studies)</td>
<td>Studies in which risk factors were significant in the model8, 16, 20, 23, 49, 52, 60, 63, 64, 67, 70, 74</td>
</tr>
<tr>
<td>General ADLs</td>
<td>50.0% (3 of 6 studies)</td>
<td>Studies in which risk factors were significant in the model21, 24, 65</td>
</tr>
<tr>
<td>Friction and/or shear subscale of a risk assessment tool</td>
<td>33.3% (5 of 15 studies)</td>
<td>Studies in which risk factors were significant in the model8, 27, 35, 48, 59</td>
</tr>
<tr>
<td>Activity subscale of a risk assessment tool</td>
<td>16.6% (3 of 18 studies)</td>
<td>Studies in which risk factors were significant in the model8, 21, 31, 69</td>
</tr>
<tr>
<td>Interface pressures</td>
<td>66.6% (2 of 3 studies)</td>
<td>Studies in which risk factors were significant in the model57, 58</td>
</tr>
</tbody>
</table>

1.3. Complete a comprehensive risk assessment for bedfast and/or chairfast individuals to guide preventive interventions. (Strength of Evidence = C; Strength of Recommendation = ⚫⚫⚫)

This statement is based on expert opinion. Mobility and activity limitations can be considered a necessary condition for pressure ulcer development. In the absence of these conditions, other risk factors should not result in a pressure ulcer. However, pressure ulcers are multi-causal and it is important to identify any other potential contributing factors in immobile individuals in order to implement a comprehensive prevention plan.

Skin status

1.4. Consider individuals with a Category/Stage I pressure ulcer to be at risk of progression or new Category/Stage II and greater pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = ⚫⚫)
1.5. Consider individuals with an existing pressure ulcer (any Category/Stage) to be at risk of additional pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = ⚫ ⚫)

1.6. Consider the general status of skin on pressure ulcer risk. (Strength of Evidence = B; Strength of Recommendation = ⚫)

These statements are based upon high quality epidemiological evidence (see Table 2). The literature identifies that skin/pressure ulcer status emerges consistently in multivariable modeling and demonstrates a strong statistical association with the development of new pressure ulcers.

Epidemiological studies utilizing multivariable modeling consistently identify the presence of non-blanching erythema (a Category/Stage I pressure ulcer) and alterations to intact skin as increasing the probability of pressure ulcer development (see Table 2)\(^2, 12\) (Level 2 and 4 studies). The presence of an existing pressure ulcer of any Category/Stage emerges less consistently as a significant predictor of new pressure ulcer development\(^2, 63, 65, 75\) (Level 2 and 4 studies).

In terms of the underlying conceptual framework, skin status is associated with the susceptibility and tolerance of the skin, indicating that physiology and repair and transport properties of the skin have been disrupted.

Table 2: Summary of evidence for measures of skin status as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Existing Category/Stage I pressure ulcer</td>
<td>100% (4 of 4 studies)</td>
<td>Studies in which risk factors were significant in the model(^17, 42, 43, 30)</td>
</tr>
<tr>
<td>General skin status</td>
<td>90.9% (10 of 11 studies)</td>
<td>Studies in which risk factors were significant in the model(^9, 12, 17, 26, 28, 40, 43, 47, 51, 54)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model(^23)</td>
</tr>
<tr>
<td>Existing pressure ulcer of any Category/Stage</td>
<td>37.5% (3 of 8 studies)</td>
<td>Studies in which risk factors were significant in the model(^19, 28, 65)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model(^15, 43, 59, 63, 75)</td>
</tr>
<tr>
<td>Previous pressure ulcers</td>
<td>25.0% (1 of 4 studies)</td>
<td>Studies in which risk factors were significant in the model(^68)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model(^17, 35, 64)</td>
</tr>
</tbody>
</table>

2. Consider the impact of the following factors on an individual’s risk of pressure ulcer development:
   - perfusion and oxygenation;
   - poor nutritional status; and
   - increased skin moisture. (Strength of Evidence = C; Strength of Recommendation = ⚫)

This statement is based upon primarily moderate and low quality epidemiological evidence. The literature identifies that these three risk factors emerge in epidemiological studies on pressure ulcer risk, demonstrating a moderate association with the development of new pressure ulcers.

Perfusion and Oxygenation

Epidemiological studies consistently identify alterations to tissue perfusion and oxygenation as increasing the probability of pressure ulcer development (Level 2 and 4 studies). Results from a number of epidemiological studies that employed multivariable analyses indicate that various factors affecting tissue perfusion and oxygenation increase the risk of pressure ulcer development (see Table 3).
However, translation into practice, (i.e., how tissue perfusion and oxygenation can be assessed) is complicated by the wide range of descriptors and direct and indirect measures utilized by researchers. Examples include diabetes; cerebrovascular accident (CVA); renal disease; cardiac disease; vascular disease; peripheral vascular disease (PVD); cardiovascular instability/norepinephrine use; pulse pressure; ‘skin circulation’; cyanosis, popliteal and posterior tibial pulses; hematocrit; low diastolic blood pressure; decreased ankle brachial index; hypotension; high systolic blood pressure; hypertension; inotrope administration; cigarette smoking and oxygen use.², ⁶⁶, ⁶⁹-⁷¹, ⁷³, ⁷⁴

In terms of the underlying conceptual framework (see Figure 1), perfusion and oxygenation factors are associated with the susceptibility and tolerance of the skin, with consideration given to the potential impact upon individual physiology and repair; and transport and thermal properties.

**Table 3: Summary of evidence for measures of perfusion and circulation as a risk factor for pressure ulcer development**

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vascular disease</td>
<td>66.6% (4 of 6 studies)</td>
<td>Studies in which risk factors were significant in the model²¹, ³², ⁴¹, ⁶⁰</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model²⁹, ⁵⁹</td>
</tr>
<tr>
<td>Alterations to blood pressure (low or high)</td>
<td>61.5% (8 of 13 studies)</td>
<td>Studies in which risk factors were significant in the model¹⁰, ²³, ²⁵, ⁴², ⁴⁷, ⁶⁰, ⁶⁶, ⁷⁰</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model¹³, ²⁹, ⁴⁵, ⁵⁷</td>
</tr>
<tr>
<td>Diabetes</td>
<td>50.0% (7 of 14 studies)</td>
<td>Studies in which risk factors were significant in the model¹⁴, ²⁴, ⁴³, ⁴⁶, ⁴⁹, ⁷³, ⁷⁴</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model¹⁵, ²¹, ²⁶, ²⁹, ₃₂, ₃⁵, ⁶⁰</td>
</tr>
<tr>
<td>Circulation</td>
<td>50% (5 of 10 studies)</td>
<td>Studies in which risk factors were significant in the model²⁶, ⁴⁴, ⁴⁵, ⁶⁹, ⁷¹</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model²⁸, ³², ⁵⁹, ⁶⁸, ⁷⁰</td>
</tr>
<tr>
<td>Smoking</td>
<td>40% (2 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model⁵⁷, ⁵⁸</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-significant studies²⁹, ³², ⁶⁸</td>
</tr>
<tr>
<td>Edema</td>
<td>20% (1 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model²⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model²⁰, ²⁹, ⁴¹, ⁷³</td>
</tr>
</tbody>
</table>

**Nutrition Indicators**

A number of Level 2 and 4 studies have identified that nutritional deficits increase the probability of pressure ulcer development (see Table 4).², ¹², ⁷², ⁷⁴, ⁷⁵ Indicators of nutritional deficits reported in these studies and considered in the multivariable model included descriptors, scales and measures as follows:

- Study specific descriptors of food intake.
- Presence of malnutrition (e.g., diagnosis of malnourishment recorded in medical record).
- Arm measurements.
- Nutrition assessment scales.
- Low weight and weight loss.
- Low body mass index (BMI).
- Other measures of nutritional status (e.g., nutrition screening resulting in a dietitian referral).

None of the studies included in the multivariable model specifically investigated elevated weight or BMI as a potential risk factor for pressure ulcers. The potential relationship between obesity and pressure
ulcer occurrence is discussed in the Special Populations: Bariatric (Obese) Individuals section of the full Clinical Practice Guideline.

In terms of the underlying conceptual framework (see Figure 1), nutritional deficits are associated with, and may impact upon all four components of the susceptibility and tolerance of the skin, including mechanical properties of the tissue; the geometry (morphology) of the tissues; physiology and repair; and transport and thermal properties.

Table 4: Summary of evidence for measures of nutritional status as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study specific descriptors of food intake</td>
<td>57.1% (4 of 7 studies)</td>
<td>Studies in which risk factors were significant in the model^10, 21, 24, 31 Studies in which risk factors were not significant in the model^20, 27, 28</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>33.3% (1 of 3 studies)</td>
<td>Studies in which risk factors were significant in the model^50 Studies in which risk factors were not significant in the model^29, 55</td>
</tr>
<tr>
<td>Arm measurements</td>
<td>33.3% (1 of 3 studies)</td>
<td>Significant studies^47 Studies in which risk factors were not significant in the model^27, 56</td>
</tr>
<tr>
<td>Low weight and weight loss</td>
<td>28.6% (4 of 14 studies)</td>
<td>Studies in which risk factors were significant in the model^17, 25, 39, 42 Studies in which risk factors were not significant in the model^10, 23, 26, 37, 38, 45, 61, 62, 71, 75</td>
</tr>
<tr>
<td>Low body mass index (BMI)</td>
<td>28.6% (4 of 14 studies)</td>
<td>Studies in which risk factors were significant in the model^13, 14, 72, 74 Studies in which risk factors were not significant in the model^24, 26, 28, 32, 39, 56, 60, 64, 66, 68</td>
</tr>
<tr>
<td>Nutrition assessment scales</td>
<td>6.6% (1 of 15 studies)</td>
<td>Studies in which risk factors were significant in the model^56 Studies in which risk factors were not significant in the model^18, 22, 28, 30, 35, 38, 59, 43, 47, 48, 59-61</td>
</tr>
<tr>
<td>Other measures of nutrition status</td>
<td>33.3% (3 of 9 studies)</td>
<td>Studies in which risk factors were significant in the model^12, 72, 75 Studies in which risk factors were not significant in the model^26, 37, 61, 62, 64, 65</td>
</tr>
</tbody>
</table>

Skin Moisture

General measures of skin moisture, including urinary and fecal incontinence, emerge inconsistently in epidemiological studies as factors which increase the probability of pressure ulcer development (Levels 2 and 4 studies). Indicators of skin moisture utilized in the literature include descriptors, scales and measures as follows (see Table 5)^2, 8, 65:

- Dual incontinence.
- Skin moisture.
- The moisture subscale of a risk assessment tool.
- Fecal incontinence.
- Urinary catheter in situ.
- Urinary incontinence.
- Incontinence (type unspecified).

It should be considered that a certain level of skin hydration is necessary to ensure proper skin function and resistance. The factors listed above refer to excess moisture. In terms of the underlying conceptual...
framework (see Figure 1) excess moisture impacts the susceptibility and tolerance of the skin by affecting the barrier and mechanical properties of the tissue; and physiology and repair.

### Table 5: Summary of evidence for measures of skin moisture as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dual incontinence</td>
<td>60.0% (3 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model 20, 46, 60; Studies in which risk factors were not significant in the model 19, 59</td>
</tr>
<tr>
<td>Skin moisture</td>
<td>60.0% (3 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model 20, 26, 57; Studies in which risk factors were not significant in the model 27, 35</td>
</tr>
<tr>
<td>Moisture subscale of a risk assessment tool</td>
<td>35.7% (5 of 14 studies)</td>
<td>Studies in which risk factors were significant in the model 6, 18, 35, 52, 59; Studies in which risk factors were not significant in the model 20, 22, 28, 30, 38, 48, 60, 61, 68</td>
</tr>
<tr>
<td>Fecal incontinence</td>
<td>25.0% (3 of 12 studies)</td>
<td>Studies in which risk factors were significant in the model 24, 40, 65; Studies in which risk factors were not significant in the model 23, 29, 35, 38, 50, 57, 64</td>
</tr>
<tr>
<td>Urinary catheter insitu</td>
<td>25.0% (1 of 4 studies)</td>
<td>Studies in which risk factors were significant in the model 50; Studies in which risk factors were not significant in the model 21, 26, 65</td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>12.5% (1 of 8 studies)</td>
<td>Studies in which risk factors were significant in the model 57; Studies in which risk factors were not significant in the model 19, 20, 24, 35, 39, 65</td>
</tr>
<tr>
<td>Incontinence (type unspecified)</td>
<td>100% (1 of 1 study)</td>
<td>Studies in which risk factors were significant in the model 57</td>
</tr>
</tbody>
</table>

3. Consider the potential impact of the following factors on an individual’s risk of pressure ulcer development:
   - increased body temperature;
   - advanced age;
   - sensory perception;
   - hematological measures and;
   - general health status (Strength of Evidence = C; Strength of Recommendation = ) 

This statement is based upon high and medium quality epidemiological evidence. The literature identifies that these four risk factors emerge inconsistently in epidemiological studies on pressure ulcer risk, demonstrating a weak association with the development of pressure ulcers. Advanced age, sensory perception and health status are likely confounding factors of characteristics demonstrated to be strong risk factors of pressure ulcer development, particularly immobility.

### Body Temperature

A systematic review by Coleman et al. (2013)² identified eight studies (see Table 6) that included body temperature in multivariable modeling and, of these, three studies reported an independent statistical association between elevated body temperature and pressure ulcer development, one reported an association but not the direction of the relationship and in three studies body temperature did not emerge in multivariable modeling (Level 2 and 4 studies). No new studies were found in the updated
review. This may be an aspect that is considered in risk assessment, but it is an area which requires confirmatory research.

In terms of the underlying conceptual framework, body temperature may impact upon the susceptibility and tolerance of the skin by affecting physiology and repair; and transport and thermal properties.

**Table 6: Summary of evidence for measures of body temperature as a risk factor for pressure ulcer development**

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body temperature</td>
<td>62.5% (5 of 8 studies)</td>
<td>Studies in which risk factors were significant in the model 10, 41, 51, 57, 58</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model 30, 32, 60</td>
</tr>
</tbody>
</table>

**Advanced Age**

Early prevalence surveys established that pressure ulcers are generally associated with advanced age, although it is recognized that pressure ulcers do affect individuals of all ages, including infants and neonates in whom other risk factors are present. A large number of studies (n = 40) have included age within multivariable modelling. Increasing age emerges as an independent risk factor in only 15 (37.5%) studies 2, 71, 73, 75 (Level 2 and 4 studies) (see Table 7). It is suggested that age is a confounding factor and a general indicator of likely deficits in the main areas of risk including mobility/activity; skin status; perfusion and oxygenation; nutrition; and skin moisture. Therefore, in terms of the underlying conceptual framework (see Figure 1), at an individual level age may impact upon both the mechanical boundary conditions and all four components of susceptibility and tolerance of the skin: mechanical properties of the tissue; the geometry (morphology) of the tissue; physiology and repair; and transport and thermal properties.

**Table 7: Summary of evidence for measures of increasing age as a risk factor for pressure ulcer development**

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing age</td>
<td>37.5% (15 of 40 studies)</td>
<td>Studies in which risk factors were significant in the model 10, 11, 14, 34-36, 39, 43, 46, 48, 56, 60, 71, 73, 75</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model 12, 13, 17, 19-21, 24-28, 32, 33, 37, 38, 42, 45, 59, 61-64, 67, 68, 74</td>
</tr>
</tbody>
</table>

**Sensory Perception**

A systematic review by Coleman et al. (2013) 2 identified nine studies which included the Braden sensory perception subscale in multivariable modeling and a further three studies have been identified in this update. 8, 12, 69 Sensory perception emerges in only four (33.3%) of the 12 studies identified, despite widespread clinical recognition that this is an important risk factor (Level 2 and 4 studies). It is likely that in statistical modeling other confounding factors related to sensory deficits, including factors associated with loss of sensation (e.g., diabetic neuropathy and spinal cord injury) and lack of response (e.g., mental capacity or acuity of illness) are dominant.

In terms of the underlying conceptual framework, sensory perception impacts upon the mechanical boundary conditions.
Table 8: Summary of evidence for measures of sensory perception as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory perception subscale of the Braden Scale</td>
<td>33.3% (4 of 12 studies)</td>
<td>Studies in which risk factors were significant in the model⁶, ¹², ²⁸, ³⁵</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model¹⁰, ²², ³⁸, ⁴⁸, ⁵⁹-⁶¹, ⁶⁹</td>
</tr>
</tbody>
</table>

Hematological Measures

A number of Level 2 and 4 studies², ⁶⁴ have reported a statistical association between abnormal hematological measures and pressure ulcer development, including alterations to urea and electrolytes (e.g., creatinine above 1 mg/dl), elevated C-reactive protein, lymphopenia, low albumin, and low hemoglobin. Direct interpretation and application to practice is complicated by the diversity of causes for abnormality in hematological measures ranging from severe malnutrition to blood loss during surgery and the impact upon the tolerance of the tissues may be multi-factorial.

In terms of the underlying conceptual framework (see Figure 1), abnormal hematological measures may impact upon the susceptibility and tolerance of the skin by affecting physiology and repair; and transport and thermal properties.

Table 9: Summary of evidence for hematological measures as a risk factor for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphopenia</td>
<td>100% (2 of 2 studies)</td>
<td>Studies in which risk factors were significant in the model¹⁷, ⁴⁷</td>
</tr>
<tr>
<td>Albumin</td>
<td>58.3% (7 of 12 studies)</td>
<td>Significant studies¹⁶, ³¹, ³⁶, ⁴⁰, ⁴³, ⁵⁰, ⁵⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model¹⁰, ³⁸, ³⁹, ⁵², ⁶⁴</td>
</tr>
<tr>
<td>Hemoglobin (Hb)</td>
<td>54.5% (6 of 11 studies)</td>
<td>Studies in which risk factors were significant in the model¹⁵, ²⁰, ³⁶, ⁴³, ⁴⁵, ⁴⁷</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model³², ³⁴, ⁴², ⁴⁴, ⁵⁶</td>
</tr>
<tr>
<td>Urea and Electrolytes (U&amp;Es)</td>
<td>50% (2 of 4 studies)</td>
<td>Studies in which risk factors were significant in the model²⁵, ⁵², ⁵⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model²¹, ⁴⁴</td>
</tr>
<tr>
<td>Protein (C-reactive protein)</td>
<td>33.3% (1 of 3 studies)</td>
<td>Significant studies³⁶</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model⁶⁰, ⁵³</td>
</tr>
<tr>
<td>Other hematological measures</td>
<td>100% (1 of 1 studies)</td>
<td>Studies in which risk factors were significant in the model³⁶</td>
</tr>
</tbody>
</table>

General Health Status

A number of epidemiological studies have used measures indicating general and mental health status and these have emerged inconsistently in multivariable modeling as predictive of pressure ulcer development (Level 2 and 4 studies). Examples (see Table 10) include number of activities of daily living (ADL) dependencies; do-not-resuscitate (DNR) status; mechanical ventilation illness severity scores, including the APACHE II, Ramsey, and acquired immune deficiency syndrome (AIDS) severity and performance indices; confusion/mental status; acute (versus elective) admission; surgical treatment; various medication treatments; and length of stay.², ⁸, ⁶⁳-⁶⁶, ⁶⁹, ⁷¹, ⁷², ⁷⁴, ⁷⁵
It is suggested that general health status is a confounding factor and a general indicator of likely deficits in the main areas of risk including mobility/activity, skin status and perfusion, nutrition and skin moisture. Therefore, in terms of the underlying conceptual framework, at an individual level, general health status impacts upon both the mechanical boundary conditions and all four components of the susceptibility and tolerance of the skin.

**Table 10: Summary of evidence for measures of mental and general health status as a risk factor for pressure ulcer development**

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>General health status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Society of Anesthesiologists (ASA) score</td>
<td>50% (1 of 2 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{45}) Studies in which risk factors were not significant in the model(^\text{29})</td>
</tr>
<tr>
<td>Chronic wounds</td>
<td>50% (1 of 2 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{43}) Studies in which risk factors were not significant in the model(^\text{42})</td>
</tr>
<tr>
<td>Acute Physiology and Chronic Health Evaluation (APACHE II) score</td>
<td>40% (2 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{62, 63}) Studies in which risk factors were not significant in the model(^\text{24, 26, 29, 45, 55, 60, 62, 68})</td>
</tr>
<tr>
<td>Medication</td>
<td>38.5% (5 of 13 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{15, 20, 41, 72, 74}) Studies in which risk factors were not significant in the model(^\text{24, 26, 29, 45, 60, 62, 68})</td>
</tr>
<tr>
<td>Norton score measures</td>
<td>0% (0 of 3 studies)</td>
<td>Non-significant studies(^\text{28, 30, 48})</td>
</tr>
<tr>
<td>Other factors</td>
<td>41.7% (15 of 36 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{8, 14, 39, 41, 43, 49, 50, 62, 64, 66, 69, 71, 74, 75}) Studies in which risk factors were not significant in the model(^\text{9, 12, 13, 20, 24, 26-29, 32, 35, 37, 42, 44, 52, 53, 56, 61, 65, 68, 73})</td>
</tr>
<tr>
<td>Mental health status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mental status study specific measures</td>
<td>22.2% (2 of 9 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{50, 65}) Non-significant studies(^\text{19, 20, 24, 29, 35, 47, 53})</td>
</tr>
<tr>
<td>Mental status subscale of a risk assessment tool</td>
<td>20% (1 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model(^\text{48}) Studies in which risk factors were not significant in the model(^\text{28, 30, 47})</td>
</tr>
<tr>
<td>Mini Mental State Exam (MMSE)</td>
<td>0.0% (0 of 1 studies)</td>
<td>Studies in which risk factors were not significant in the model(^\text{64})</td>
</tr>
</tbody>
</table>

**Other Potential Risk Factors for Pressure Ulcers**

A number of risk factor studies (see Table 11) have explored the relationship between race and gender and pressure ulcer development, but results from different studies are contradictory and inconclusive. Whilst prevalence data indicates the rate of pressure ulcers is higher in people with darkly pigmented skin\(^\text{76-81}\) only one epidemiological study demonstrated an increased risk in individuals with darkly pigmented skin. It is suggested that the observed increased prevalence rate may be due to delayed detection rather than to a true increase in risk (see the Classification of Pressure Ulcers and Assessment of Pressure Ulcers and Monitoring of Healing sections of the full Clinical Practice Guideline for further discussion).
Table 11: Summary of evidence for demographic characteristics as risk factors for pressure ulcer development

<table>
<thead>
<tr>
<th>Risk factor variables</th>
<th>Percent studies significant in multivariable model</th>
<th>Risk factor significant and non-significant in multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Race</td>
<td>40% (2 of 5 studies)</td>
<td>Studies in which risk factors were significant in the model¹¹,¹⁹</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model⁹,¹²,²⁴</td>
</tr>
<tr>
<td>Gender</td>
<td>30% (6 of 20 studies)</td>
<td>Significant studies²⁰,²⁶,³⁶,⁴⁴,⁶⁷,⁶⁸</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Studies in which risk factors were not significant in the model²¹⁻²³,²⁴,²⁹,³³,³⁷,³⁹,⁵⁶,⁶³,⁶⁴,⁷⁴</td>
</tr>
</tbody>
</table>

Risk Assessment Tools

Most risk assessment tools incorporate many of the risk factors discussed above (e.g., activity, mobility, nutrition, moisture, sensory perception, friction and shear, and general health condition). However, the volume of epidemiological research has increased considerably in recent years, providing for a better understanding of the risk factors important in the development of pressure ulcers, and risk assessment tools do not incorporate these advances in knowledge. If risk assessment tools are selected as a structured approach for risk assessment, additional factors (e.g., perfusion, skin status and other relevant risks) should be considered as part of a comprehensive risk assessment. Table 12 compares the risk factors supported by current epidemiological studies with the risk factors measured by the three most commonly used risk assessment tools, identifying gaps for each tool. Regardless of how the risk assessment is structured, clinical judgment is essential.

Table 12: Comparison of Risk Factors Identified in Both Epidemiological Studies and Commonly Used Risk Assessment Tools

Note: An asterisk (*) indicates that the actual subscale was significant in multivariable modeling in one or more epidemiological study identified. Lack of an asterisk may indicate non-significance in multivariable modeling, but may also indicate that the subscale was not entered in any multivariable modeling studies. “Not included” indicates the risk factor is not included on the risk assessment tool, identifying a gap that health professionals should consider during a comprehensive risk assessment.
Risk Factors from Epidemiological Studies | Braden Scale | Norton Scale | Waterlow Score
--- | --- | --- | ---
Increased body temperature | Not included | Not included | Not included
Advanced age | Not included | Not included | Gender/Age
Sensory perception | Sensory Perception* | Not included | Neurological Deficit
Hematological measures | Not included | Not included | Not included
General health status | Not included | Physical condition | Major Surgery/Trauma
Mental condition* | | Medications |

1. Recognize additional risk factors and use clinical judgment when using a risk assessment tool. (Strength of Evidence = C; Strength of Recommendation = )

Caution: Do not rely on the results of a risk assessment tool alone when assessing an individual’s pressure ulcer risk.

This statement is based upon expert opinion. A risk assessment tool offers a structured approach to assessment, but does not replace a comprehensive assessment conducted by an appropriately qualified health professional using a structured approach to clinical judgment. The majority of the currently available risk assessment tools were developed on the basis of literature review, expert opinion, and/or adaptation of an existing scale. The three most commonly used scales – the Norton Scale© (1962), Waterlow Score© (1985), and the Braden Scale for Predicting Pressure Sore Risk© (1987) – were developed more than twenty years ago without the insight from more recent epidemiological studies.

Risk assessment tools do not necessarily include assessment of all key factors that can increase the risk of pressure ulcer development. Specifically, most risk assessment tools do not include an assessment of tissue perfusion or skin status. As presented under Risk Factors for Pressure Ulcers (see above), epidemiological studies identify these factors as strong indicators of pressure ulcer risk. It is important to consider tissue perfusion and skin status in conjunction with an assessment conducted with a formalized risk assessment tool.

Additionally, most risk assessment tools use a simple ordinal system to score risk. They are limited in their ability to assess any potential differences in the contribution or importance of one risk factor versus another, or to assess the cumulative effect of two or more risk factors. In an attempt to create a simple screening tool for clinical use, the complex interplay of individual and environmental factors has been reduced to a simple score. Therefore, clinical judgment must be exercised to interpret these scores with consideration of the impact of other risk factors and within the context of often-complex individual and clinical factors.

Risk Assessment Tools versus Clinical Judgment

A large number of risk assessment tools have been developed to provide a structured approach for risk assessment in practice, yet the results of studies comparing risk assessment tools to clinical judgment are mixed. Risk assessment tools provide some advantages over clinical judgment alone. For example, they provide:
- a practical framework;
- operational definitions of risk factors that have clinical utility and can be reliably measured;
- clinical reminders (especially for novice nurses); and
• a minimum auditable standard.

A meta-analysis conducted by García-Fernández et al. (2014)\textsuperscript{85} reported relatively poor pooled predictive capacity indicators for clinical judgment as measured by relative risk (RR = 1.95; 95% confidence interval [CI] 0.94 to 4.04) when compared to the Braden Scale (RR = 4.26; 95% CI 3.27 to 5.55), Norton Scale (RR = 3.69; 95% CI 2.64 to 5.16), Waterlow Score (RR = 2.66, 95% CI 1.76 to 4.01) and modified Cubbin-Jackson Scale for critically ill individuals (RR = 3.16; 95% CI 1.49 to 6.71). When 1.0 (null value, i.e. equal odds) is included in the confidence interval (see clinical judgment results), results are considered less than conclusive.

Moore et al. (2014)\textsuperscript{86} conducted a systematic review to determine if using structured systematic pressure ulcer risk assessment tools reduced the incidence of pressure ulcers. Finding only two studies meeting their inclusion criteria,\textsuperscript{75, 87} they concluded that there was no evidence from randomized controlled trials (RCTs) to suggest structured pressure ulcer risk assessment reduces the incidence of pressure ulcers.

One of these trials was a large, blinded randomized trial conducted by Webster et al. (2011)\textsuperscript{75} that compared use of a Waterlow Score (n = 410), the Ramstadius risk screening tool (n = 411) and risk assessment based on the nurse’s clinical judgment (n = 410) for reducing pressure ulcer occurrence in participants located in medical and oncology wards in Australia. After the four day follow up period, facility-acquired pressure ulcer rates were not significantly different between those assessed with the Waterlow Score versus clinical judgment (7.5% versus 6.8%, risk ratio = 1.10, 95% CI 0.68 to 1.81, \(p = 0.69\)) or between those assessed with the Ramstadius risk screening tool versus clinical judgment (5.4% versus 6.8%, risk ratio = 0.79, 95% CI 0.46 to 1.35, \(p = 0.38\)). The difference in pressure ulcer rates between the two groups assessed with risk assessment tools was also not significant (\(p = 0.18\)) (Level 2 study).

In the second of the trials reported in the systematic review by Moore et al. (2014)\textsuperscript{86}, Saleh et al. (2009)\textsuperscript{87} conducted a cluster randomized trial in a military hospital in Saudi Arabia. Participants were considered to be at risk of pressure ulcers (Braden Scale score \(\leq 18\)). The trial, which had three groups, compared the use of the Braden risk assessment tool (group A; n = 74); nurse education on the Braden Scale but risk assessment conducted using clinical judgment alone (group B; n = 76) and risk assessment using clinical judgment without accompanying education (group C; n = 74). After eight weeks, there was no statistically significant difference in pressure ulcer incidence between group A and group B (16 versus 17 pressure ulcers, risk ratio = 0.97; 95% CI 0.53 to 1.77, \(p = 0.91\)). There was also no statistically significant difference in pressure ulcer incidence between group A and group C (16 pressure ulcers in each group, risk ratio = 1.43; 95% CI 0.77 to 2.68, \(p = 0.26\)). The trial was considered to be at high risk of bias (Level 2 study).

An additional clinical trial designed primarily to assess the effectiveness of different repositioning regimens that did not meet inclusion criteria for the review conducted by Moore et al. (2014)\textsuperscript{86} reported on different strategies to assess pressure ulcer risk. Participants (n = 1,772) were assessed using the Norton Scale, the Braden Scale and by nurses using their own clinical judgment\textsuperscript{88} after being randomly allocated to different repositioning regimen groups. Sensitivity of clinical judgment was 25% to 28% lower than assessment using the risk assessment tools and specificity was 20% to 30% higher. Fewer individuals who developed a pressure ulcer were identified as being at risk when clinical judgment was used, but of those individuals identified at risk, more actually developed a pressure ulcer. The two risk assessment tools were essentially equivalent in predicting development of pressure ulcers. Education background and clinical experience of the nurses participating in the study were not reported (Level 5 study).

There are limitations to the current research that prevent a clear comparison between risk assessment tools and clinical judgment alone.\textsuperscript{89} In the majority of studies investigating risk assessment strategies, preventive interventions are initiated on the basis of the risk assessment. These interventions will impact upon pressure ulcer incidence, confounding the evaluation of the risk assessment strategy. Defloor et
al. (2005)\(^8\) highlight that development of a pressure ulcer in an individual assessed as being at risk is primarily an indication that preventive management was insufficient, rather than an indication that the risk assessment strategy was reliable. In the studies conducted by Saleh et al. (2009)\(^7\) and Defloor et al. (2005)\(^8\), there was non-equivalent use of pressure ulcer prevention strategies, in particular the types of support surfaces used, between individuals identified at risk and not at risk and this confounded the findings. In the higher quality study conducted by Webster et al. (2011)\(^5\), non-significant differences in pressure ulcer prevention interventions initiated following the risk assessment is reported.

2. **When using a risk assessment tool, select a tool that is appropriate to the population, is valid and is reliable. (Strength of Evidence = C; Strength of Recommendation = \(\mathbf{\ddagger}\))**

This statement is based upon expert opinion. A review of the evidence on reliability and validity for the most commonly used pressure ulcer risk assessment tools is provided below.

**Reliability**

Reliability refers to the consistency and the ability of scores to differentiate among subjects. Reliability is widely regarded as a necessary condition for validity. There are a large number of studies that specifically address the interrater and intrarater reliability of risk assessment tools and reports of early tool development usually contain some measure of reliability. There are generally high levels of reliability in terms of total scores for the Modified Norton Scale (intraclass correlation coefficient [ICC] = 0.821, 95% CI 0.715 to 0.926)\(^9\) and Braden Scale (ICC range = 0.72 to 0.95).\(^10-95\) Interrater reliability for the Waterlow Score was reported as 1.0 in one study\(^7\) and as 0.36 (95% CI 0.09 to 0.63) in a second study.\(^9\) Interrater reliability for subscale scores varied depending on the subscale and the clarity of the operational definition.\(^90-95\) Ongoing education and competency testing for health professionals administering risk assessment tools are important to support reliability.

**Validity**

Validity refers to the degree to which a tool measures what it claims to measure. Of the many types of validity (e.g., content, construct and criterion), ‘predictive validity’ has received the most attention in relation to risk assessment tools. Rather than focus on the degree to which these tools accurately measure risk factors such as mobility, activity and skin moisture, we have focused on the degree to which they predict a future event (i.e., pressure ulcer development).

A major problem identified in the literature\(^8\) in establishing predictive validity of risk assessment tools is that preventive interventions are initiated in the majority of studies, and these will impact upon the performance of the tool. Studies of predictive validity are prognostic (estimating the likelihood of a future problem) rather than diagnostic (identifying an existing problem). Despite these constraints, most studies of predictive validity report some statistical estimates of likelihood associated with each prognostic method. These include sensitivity, specificity, positive likelihood ratio (PLR), negative likelihood ratio (NLR), area under receiver operator curves (as an indication of the best balance between sensitivity and specificity; area under receiving operator characteristic [AUROC]) and relative risk. Although these measures are imperfect, they provide some insight into the predictive validity of risk assessment tools, especially when considered in light of the intervening preventive strategies.

Table 13 summarizes the estimates for the three most frequently studied risk assessment tools. Data for this table were abstracted from a systematic review that met criteria for guideline inclusion as identified in the *Methodology* section of the guideline.\(^4\) One relevant article was published after the inclusion date (July 2012) for review by Chou et al. (2013)\(^3\) but before the end of the guideline review period (July 2013). In this study,\(^96\) predictive validity was determined for Norton, Braden and Waterlow Scores for 100 surgical participants in New Delhi, India. Specificity for the Norton and Braden scales fell well within the range of those summarized by Chou et al. (2013)\(^3\). Predictive validity of the Waterlow Score was higher than that reported by Chou et al. (2013)\(^3\). At a cutoff score of 10, sensitivity was 95.65%, and specificity was 74.02%.\(^96\)
Table 13: Psychometric qualities of major risk assessment tools based on data from Chou et al. (2013)\(^3\)

Note: The Chou et al. (2013)\(^3\) analysis did not provide estimates of relative risk. These were taken from a meta-analysis conducted by García-Fernández et al. (2014)\(^85\)

<table>
<thead>
<tr>
<th>Scales (cut-off)</th>
<th>Sensitivity Median (range)</th>
<th>Specificity Median (range)</th>
<th>PLR</th>
<th>NLR</th>
<th>AUROC Median (range)</th>
<th>Relative Risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Braden (&lt; 18)</td>
<td>0.74(^a) (0.33 to 1)</td>
<td>0.68(^a) (0.34 to 0.86)</td>
<td>2.31(^a)</td>
<td>0.38(^a)</td>
<td>0.77(^b) (0.55 to 0.88)</td>
<td>4.26(^f) (3.27 to 5.55)</td>
</tr>
<tr>
<td>Norton (&lt; 14)</td>
<td>0.75(^c) (0 to 0.89)</td>
<td>0.68(^c) (0.59 to 0.95)</td>
<td>2.34(^c)</td>
<td>0.37(^c)</td>
<td>0.74(^c) (0.56 to 0.75)</td>
<td>3.69(^g) (2.64 to 5.16)</td>
</tr>
<tr>
<td>Waterlow (&gt; 10)</td>
<td>1.00, 0.88(^d)</td>
<td>0.13, 0.29(^d)</td>
<td>1.15, 1.24(^d)</td>
<td>0.0, 0.41(^d)</td>
<td>0.61(^e) (0.54 to 0.66)</td>
<td>2.66(^h) (1.76 to 4.01)</td>
</tr>
</tbody>
</table>

\(^a\)16 studies, n=5,462  \(^b\)7 studies, n=4,811  \(^c\)5 studies, n=2,809
\(^d\)2 studies, n=419  \(^e\)4 studies, n=2,559  \(^f\)15 studies, n=4,935
\(^g\)15 studies, n=4,935  \(^h\)12 studies, 2,408

Other risk assessment tools have received minimal psychometric testing. Those noted in publications over the five year review period for this guideline revision include the Suriadi and Sanada Scale,58 Risk Assessment Pressure Sore Scale,97 The Modified Norton Scale,90 Ramstadius,75 and Cubbin-Jackson Scales.98, 99 The sections Special Populations: Pediatric Individuals and Special Populations: Individuals in Palliative Care of the full Clinical Practice Guideline discuss population specific risk assessment tools.

Comparison of Risk Tools

The systematic comparative effectiveness review completed by Chou et al. (2013)\(^3\) attempted to answer the question: “How do various risk assessment tools compare with one another in their ability to predict the incidence of pressure ulcers?” The reviewers identified 14 studies that directly compared two or more risk assessment tools in the same population. Six studies\(^23, 28, 48, 100-102\) reported that the AUROCs within each study were comparable. AUROC’s ranged between 0.66 and 0.90 with the exception of one study\(^100\) in which AUROCs ranged between 0.55 and 0.61 which is roughly equivalent to chance (0.50). An AUROC offers the best balance between sensitivity and specificity.

The eight studies\(^23, 28, 100, 101, 103-105\) examining sensitivity and specificity reported very similar findings for comparisons of tools within the same population(s). Sensitivity and specificity vary by the cut-off score used for the tool. Most cutoff scores are selected to optimize sensitivity and specificity; however, clinical judgment is important when considering trade-offs between sensitivity and specificity. A higher sensitivity (but lower specificity) will facilitate identification of more true-positive at-risk individuals, but will also require greater resource utilization as both true-positive and false-positive individuals receive preventive interventions. A higher specificity (but lower sensitivity) will facilitate more efficient utilization of resources as those not developing pressure ulcers are more clearly identified during risk assessment; however, some individuals who may have benefited from prevention will not be identified.

The systematic review conducted by Chou et al. (2013)\(^3\) also examined whether the predictive validity of risk assessment tools differ across clinical settings or according to individual patient characteristics. Few studies addressed these issues and results were inconclusive.

In the midst of these discussions, one must remember that ‘prediction is not destiny’. The outcome for an at-risk individual can often be altered by carefully selected and consistently implemented, risk-based prevention strategies. The best method for identifying risk has not been determined. Available evidence is summarized to guide clinical decision making.
References


70. Man S, Au-Yeung T. Hypotension is a risk factor for new pressure ulcer occurrence in older patients after admission to an acute hospital. Journal of the American Medical Directors Association. 2013 //.


CLINICAL PRACTICE GUIDELINE

RISK FACTORS AND RISK ASSESSMENT


**Albumin:** Albumin makes up 60% of total protein in the blood. It decreases with stress, age, and impaired liver function. Albumin serves to maintain colloid osmotic pressure and as a transport protein for certain ions, hormones, medications, enzymes, fatty acids, amino acids, and bilirubin. It decreases with over-hydration, stress, infection, impaired renal function, and liver disease, among other causes. Normal albumin blood level is 3.5 to 5.4 gm/dL. Normal values may vary depending upon the laboratory performing analysis.

**Blanchable erythema:** see Erythema.

**Body mass index (BMI):** Defined as an individual’s weight in kilograms divided by the square of his height in meters. The term bariatric, derived from the Greek word *baros* meaning heavy and *iatric* relating to the medical treatment of this condition, is used to refer to individuals with a BMI > 30 kg/m².

**Bony prominence:** A bony elevation or projection on an anatomical structure.

**Collagen:** The most abundant protein of the dermis, accounting for 70 to 80% of its dry weight; the main supportive protein of the skin and connective tissue.

**Deep tissue injury (DTI):** See Suspected deep tissue injury.

**Erythema:** Redness of the skin due to dilation of superficial capillaries.

**Blanchable erythema:** An area of reddened skin that temporarily turns white or pale when pressure is applied to the skin. Over a pressure site, this is due to a normal hyperemic response.

**Nonblanchable erythema:** Redness that persists following the application of fingertip pressure, usually over a bony prominence. Darkly pigmented skin may not have visible blanching. This is a sign of a Category/Stage I pressure ulcer.

**Extrinsic factors:** Originating outside of the body.

**Friction (frictional force):** The resistance to motion in a parallel direction relative to the common boundary of two surfaces, e.g., when skin is dragged across a coarse surface, such as bed linens.

**Friction blister:** An area of skin that becomes red, inflamed or broken as a result of rubbing or sliding along a surface. A friction blister is not a considered to be a pressure ulcer.

**Interface pressure:** The force per unit area that acts perpendicularly between the body and a support surface. This parameter is affected by the stiffness of the support surface, the composition of body tissue, and the geometry of the body being supported.

**Intrinsic factors:** Originating within the body.

**Malnutrition:** Malnutrition defined as any nutritional imbalance and is synonymous with the term undernutrition.

**Microclimate:** The local tissue temperature and moisture (relative humidity) level at the body/support surface interface.

**Mobility:** The ability to move oneself from one position to another.

**Partial thickness skin loss:** Skin damage that involves the epidermis and can penetrate into but not through the dermis. Includes Category/Stage I and II pressure ulcers.

**pH:** A measure on a scale from 0 to 14 of the acidity or alkalinity of a solution, with 7 being neutral, greater than 7 is more alkaline and less than 7 is more acidic.
Pressure: Normal force per unit surface area.

Pressure injury: see Pressure ulcer.

Pressure ulcer (pressure injury): a localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of pressure or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors has yet to be elucidated. (See the Etiology of Pressure Ulcers section of the guideline). Previously referred to as decubitus ulcer, bedsore and pressure sore.

Protein: A complex organic compound made up of chains of amino acid molecules. Proteins are responsible for the repair of injured tissue, fluid balance, antibody production, cellular function, and hormonal and enzymatic function. Proteins are a source of building material for muscle and for healing wounds.

Risk assessment: An assessment to determine which, if any, risk factors are present that might contribute to the development of a pressure ulcer.¹

Standard (usual) care: A term most often used in research studies to describe usual care delivered within a facility that is often the comparator intervention when pressure ulcer prevention interventions are being investigated. Standard care varies according to the setting and historical context. Within the context of this guideline, a description of the standard care is provided when available.

Tissue-interface layer: The point at which a dressing is in direct contact with the skin (wound bed).

Suspected deep tissue injury: Purple or maroon localized area of discoloured, intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler than adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with treatment.

Unstageable pressure ulcer: Full thickness tissue loss in which actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black) in the wound bed. Until enough slough and/or eschar is removed to expose the base of the wound, the true depth cannot be determined, but it will be either a Category III or IV pressure ulcer. Stable (dry, adherent, intact, without erythema or fluctuance) eschar on the heels serves as a natural (biological) cover and should not be removed.

References
