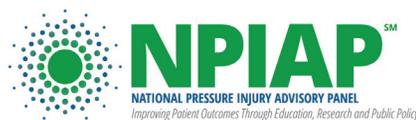


Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline

The International Guideline 2019



Copyright © European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance
ISBN 978-0-6480097-8-8

First published 2009
Second edition published 2014
Third edition published 2019

Published by European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance

All rights reserved. Apart from any fair dealing for the purposes of private study, research or review, as permitted under the Copyright Act, no part may be reproduced or copied in any form or by any means without written permission. Requests to reproduce information can be emailed to admin@internationalguideline.com



Suggested citation:

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline. Emily Haesler (Ed.). EPUAP/NPIAP/PPPIA: 2019.

Disclaimer:

This clinical practice guideline was developed by the European Pressure Ulcer Advisory Panel, the National Pressure Injury Advisory Panel, and the Pan Pacific Pressure Injury Alliance. It presents a comprehensive review and appraisal of the best available evidence at the time of literature search related to the assessment, diagnosis, prevention and treatment of pressure injuries. The recommendations in this clinical practice guideline are a general guide to appropriate clinical practice, to be implemented by qualified health professionals subject to their clinical judgment of each individual case and in consideration of the patient consumer's personal preferences and available resources. The guideline should be implemented in a culturally aware and respectful manner in accordance with the principles of protection, participation and partnership.

Printed copies of the English version of this quick reference guide can be ordered, and PDFs downloaded, from the following websites:

NPIAP	npiap.com
EPUAP	epuap.org
PPPIA	pppia.org
International Guideline	internationalguideline.com

INTRODUCTION

Foreword

This *Clinical Practice Guideline* presents recommendations, good practice statements, implementation considerations for pressure injury prevention and treatment. This *International Clinical Practice Guideline* (2019 edition) was developed as a collaboration between the Partner Organizations—European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP) and the Pan Pacific Pressure Injury Alliance (PPPIA). Additionally, 14 wound organizations from 12 countries joined the project as Associate Organizations contributing to the development, under the direction and oversight of the Partner Organization Guideline Governance Group (GGG) and a methodologist. The full development team consisted of 181 academic and clinical experts in the pressure injury field, including the 12-member GGG, the methodologist and 168 small working group members.

This edition of the guideline used the most recent methodological standards in guideline development. The methodology has been pre-published and peer reviewed. An updated literature search identified research published up to August 2018 that was critically appraised and analysed. New research has been combined with research from previous editions to extend the guideline scope and produce recommendations reflecting the most recent evidence. This third edition provides 114 evidence-based recommendations supported by an overview of the underpinning research. Implementation considerations providing practical guidance are provided to assist health professionals to implement the recommendations in clinical practice. A detailed analysis and discussion of available research, and a critical evaluation of the assumptions and knowledge in the field is included to provide further context. A consensus voting process was used to assign a strength to each recommendation. The strength of recommendation identifies the importance of the recommendation based on potential to improve patient outcomes. It provides an indication of the confidence one can have that the recommended practice will do more good than harm, and can be used to assist in prioritizing pressure injury related interventions. Many topics of relevance to pressure injury prevention and treatment have not been researched extensively. To address gaps in care, the GGG has also developed 62 good practice statements intended to further assist health professionals to deliver quality pressure injury prevention and treatment.

Engagement of patients, informal caregivers (families and friends) and other stakeholders has been extensive throughout the guideline development. An online survey of patient consumers and informal caregivers was conducted to identify care goals, priorities and education needs. Responses from 1,233 patients and their families from around the world were incorporated into the guideline development. Drafts of the recommendations and supporting evidence were made available to 699 stakeholders (individuals and organizations) around the world who registered and reviewed the documents.

A *Quick Reference Guide* presenting an abridged version of this guideline is also available. The *Quick Reference Guide* is intended for busy health professionals who require a quick reference in the clinical setting. Because the full Clinical Practice Guideline contains greater context and critical analysis, **users should not rely on the *Quick Reference Guide* alone in implementing pressure injury prevention and treatment.**

Abstract

This guideline is a collaborative effort from Partner Organizations, the European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP), and Pan Pacific Pressure Injury Alliance (PPPIA), and 14 additional wound organizations around the world. A comprehensive literature review was conducted on pressure injury prevention and treatment and a rigorous methodology was used to appraise the research and make evidence-based recommendations. This work builds on the work of the previous two editions of this guideline (2009 and 2014). Further, an international survey of patient consumers and their informal caregivers was undertaken to capture the perspectives of these individuals in the prevention and treatment of pressure injuries. The research evidence was summarized and evaluated using evidence-to-decision frameworks. Where sufficient research evidence was available, recommendations to guide clinical practice were developed. In areas without sufficient research, good practice statements were developed to promote comprehensive care. Both the recommendations and good practice statements are accompanied by implementation considerations supported by the research and/or expert opinion. The draft recommendations were made available to stakeholders for feedback. There were 699 health professionals, industry representatives, peak body organizations, researchers, policy makers, patient consumers and informal caregivers who reviewed and/or commented on the document. After refining the content, the final stage of the guideline development consisted of a consensus voting process to assign strengths of recommendations. 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 health professionals to prioritize interventions. The guideline includes

discussion of the science, followed by 114 recommendations and 62 good practice statements to guide practice in risk assessment, pressure injury prevention and treatment, and issues in implementing best practice. The needs of specific population groups are discussed, including individuals with obesity, critically ill individuals, older adults, neonates and children, and individuals in palliative care, operating room and community settings. The guideline also discusses the patient perspective of pressure injuries, and covers topics of importance to individuals, including pain and quality of life. Recommendations on supporting and engaging the patient consumer in their care are made. Finally, 20 quality indicators are provided to assist in monitoring implementation of the guideline.

Limitations and Appropriate Use of This Guideline

- Guidelines assist health professionals, patient consumers and informal caregivers to make decisions about healthcare for specific clinical conditions. The evidence-based recommendations and good practice statements may not be appropriate for use in all circumstances.
- The decision to adopt any recommendation must be made by the multidisciplinary healthcare team, in collaboration with patients and informal caregivers, and with consideration of available resources and circumstances. Nothing contained in this guideline replaces medical advice for specific cases.
- Because of the rigorous methodology used to develop this guideline, the Guideline Governance Group members believe that the research supporting the recommendations is accurate. Every effort has been made to critically appraise the research contained within this document. However, we do not guarantee the reliability 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 evidence-based recommendations and good practice statements 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 their clinical decision making.
- Generic names of products have been used, with descriptions of products taken from the research. Nothing in this guideline is intended as endorsement of a specific product.
- Nothing in this guideline is intended as advice regarding credentialing standards, 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. All products should be used according to manufacturer's directions.

Strengths of Evidence and Strengths of Recommendations

Individual studies were assigned a **level of evidence** based on study design. The body of evidence supporting each recommendation was given a **strength of evidence** based on evidence quantity, levels and consistency. A consensus voting process was used to assign a **strength of recommendation** that indicates the confidence the health professional can have that the recommended practice will improve outcomes (i.e., do more good than harm). The **strength of recommendation** can be used by health professionals to prioritize interventions. Full details are available in *Appendix One*.

Strengths of Evidence	
A	<ul style="list-style-type: none"> • More than one high quality Level I study providing direct evidence • Consistent body of evidence
B1	<ul style="list-style-type: none"> • Level 1 studies of moderate or low quality providing direct evidence • Level 2 studies of high or moderate quality providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
B2	<ul style="list-style-type: none"> • Level 2 studies of low quality providing direct evidence • Level 3 or 4 studies (regardless of quality) providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
C	<ul style="list-style-type: none"> • Level 5 studies (indirect evidence) e.g., studies in normal human subjects, humans with other types of chronic wounds, animal models • A body of evidence with inconsistencies that cannot be explained, reflecting genuine uncertainty surrounding the topic
GPS	<p>Good Practice Statement</p> <ul style="list-style-type: none"> • Statements that are not supported by a body of evidence as listed above but considered by the GGG to be significant for clinical practice.

Strengths of Recommendation	
↑↑	Strong positive recommendation: Definitely do it
↑	Weak positive recommendation: Probably do it
↔	No specific recommendation
↓	Weak negative recommendation: Probably don't do it
↓↓	Strong negative recommendation: Definitely don't it

Accessing the Guideline and Support Material

Access to digital and print copies of the *Clinical Practice Guideline* are available on the following websites:

NPIAP website	www.npiap.com
EPUAP website	www.epuap.org
PPPIA website	www.pppia.org
International Pressure Injury Guideline website	www.internationalguideline.com

The International Pressure Injury Guideline website (www.internationalguideline.com) is accessible until the next guideline revision. The website hosts additional supportive material including:

- The *Quick Reference Guide*
- Detailed methodology
- Evidence to Decision frameworks
- Future research needs
- Companion resources
- Sponsor acknowledgement
- Announcements and news from the GGG.

Translations of the *Quick Reference Guide* and information about the translation process are available from the EPUAP website. For more information contact translation@internationalguideline.com.

For enquiries regarding use of the guideline, review the *Permissions of Use* statement on the guideline website. For more information contact admin@internationalguideline.com.

GUIDELINE DEVELOPERS

Guideline Governance Group (GGG)

Jan Kottner, PhD (EPUAP Chair)

Scientific Director Clinical Research, Clinical Research Center for Hair and Skin Science, Department of Dermatology and Allergy, Charité-Universitätsmedizin, Germany

Ghent University, Faculty of Medicine and Health Sciences, Belgium

Janet Cuddigan, PhD (NPIAP Chair)

Professor, University of Nebraska Medical Center College of Nursing, USA

Keryln Carville, PhD (PPPIA Chair)

Professor, Primary Health Care and Community Nursing, Silver Chain Group and Curtin University, School of Nursing, Midwifery and Paramedicine, Australia

Katrin Balzer, PhD

Professor, University of Lübeck, Nursing Research Unit, Germany

Dan Berlowitz, MD, MPH

Professor, Boston University School of Medicine, USA

Center for Healthcare Organization and Implementation Research (CHOIR), Bedford VA Hospital, USA

Yee Yee Chang

Singapore General Hospital, Singapore

Siu Ming Susan Law, MScN

Nurse Consultant, Princess Margaret Hospital, Hong Kong.

Mary Litchford, PhD

President, CASE Software & Books, Greensboro, NC USA.

Pamela Mitchell, MN

Clinical Nurse Consultant, Christchurch Hospital, New Zealand.

Zena Moore, PhD

Professor, Royal College of Surgeons in Ireland, Ireland

Monash University, Faculty of Medicine, Nursing and Health Sciences, Australia

Ghent University, Department of Public Health, Faculty of Medicine and Health Sciences, Belgium

Lida Institute, China

Cardiff University, Wales, UK

Joyce Pittman, PhD

Associate Professor, University of South Alabama, USA

Dominique Sigaud-Roussel, PhD

Director of Research, Laboratory of Tissue Biology and Therapeutic Engineering, National Scientific Research Center (CNRS), University of Lyon, France

Methodologist and Editor-in-Chief

Emily Haesler, PhD

Adjunct Associate Professor, Curtin University, School of Nursing, Midwifery and Paramedicine, Australia

Australian National University, ANU Medical School, Academic Unit of General Practice, Australia

La Trobe University, Australian Centre for Evidence Based Aged Care, School of Nursing and Midwifery, Australia

Guideline Organizations

Partner Organizations

European Pressure Ulcer Advisory Panel

National Pressure Injury Advisory Panel

Pan Pacific Pressure Injury Alliance

Associate Organizations

Brazilian Association of Enterostomal Therapists: Wound, Ostomy and Continence Care (SOBEST)

Canadian Collaboration of Association for Enterostomal Therapy and Wounds Canada

Chinese Nursing Association

Indonesian Collaboration of Indonesian Wound Care Clinician Association and Indonesian Wound Ostomy and Continence Nursing Association

Japanese Society for Pressure Ulcers

Jiangsu Nursing Association

Korean Association of Wound Ostomy Continence Nurses

Malaysian Society of Wound Care Professionals

Philippine Wound Care Society

Saudi Chapter of Enterostomal Therapy

Taiwan Wound Ostomy and Continence Nurse Association

Thai Enterostomal Therapy Society

World Council of Enterostomal Therapists

Small Working Group (SWG) Members

Etiology: Amit Gefen, David Brienza, Laura Edsberg, Wendy Milton, Christine Murphy, Cees W. J. Oomens, Lin Perry, Yunita Sari • **Populations with Specific Pressure Injury Related Needs (chapter and recommendations throughout the guideline):** Jill Cox (leader), Ann Marie Nie (leader), Tracy Nowicki (leader), Mary Ellen Posthauer (leader), Maarit Ahtiala, Boonchuen Aimmak, Rehab Al-Dossari, Paulo Alves, Yufitriana Amir, Carina Bååth, Katrin Balzer, Terrie Beeson, Margaret Birdsong, Carmel Boylan, Jill Campbell, Fiona Coyer, Amy Darvall, Erik De Laat, Christantie Effendy, Aimee Garcia, Ailing Hu, Budi Anna Keliat, Sandra Korge, Janet Kuhnke, Siew Ling Lim, Mary Litchford, Sheau Lan Loh, Jeanine Maguire, Ambili Nair, Sun Young Nam, Paula Cristina Nogueira, Gordana Petkovska, Rina Pijpker, Wendy Sansom, Emil Schmidt, Emer Shanley, Aamir Siddiqui, Mary Sieggreen, Khristina Simon, Sue Templeton, Ann Tescher, Valentina Vanzi, Jaraspas Wongviseskarn • **Risk Factors and Risk Assessment:** Jane Nixon (leader), Susanne Coleman, Emily Haesler, Katrin Balzer, Virginia Capasso, Janet Cuddigan, Claudia Rutherford, Lisette Schoonhoven, Nancy Stotts • **Skin and Tissue Assessment:** Mary Jo Conley (leader), Ida Marie Bredesen, Reba J. Giles, Nanthakumahrie D/O

Gunasegaran, Ulrika Källman, Eleanor Letran, Kathren Puyk, Yajuan Weng, Huo Xiaorong • **Preventive Skin Care:** Mary Jo Conley (leader), Ida Marie Bredesen, Reba J. Giles, Nanthakumahrie D/O Gunasegaran, Ulrika Källman, Eleanor Letran, Kathren Puyk, Yajuan Weng, Huo Xiaorong • **Nutrition in Pressure Injury Prevention and Treatment:** Emanuele Cereda (co-leader), Nancy Munoz (co-leader), Merrilyn Banks, Angela Liew, Mary Ellen Posthauer Siriluck Siripanyawat, Jos Schols • **Repositioning and Early Mobilization:** Tracey Yap (leader), Liesbet Demarré, Lena Gunningberg, Susan Kennerly, Linda Norton, Sofia Macedo, Shuk Yi Pang, Johanna Van Rooyen • **Heel Pressure Injuries:** Jill Cox (leader), Sarah Dallimore, Barbara Delmore, Marie-Line Gaubert-Dahan, Manfred Mak, Tina Meyers, Reynaldo Rey-Matias • **Support Surfaces:** David Brienza (leader), Virginia Capasso, Misako Dai, Qixia Jiang, Sue Monaro, Katherine Rae, Steven Smet, Peter R. Worsley • **Device Related Pressure Injuries:** Rachel M. Walker (leader), Elizabeth A. Ayello, Suk Chu Chan, Aihua Chen, Ann Marie Nie, Valentina Vanzi, Peter R. Worsley • **Classifying Pressure Injuries:** Hin Moon Chong, Idramsyah, Yun Jin Lee, Andrea Pokorná, Catherine Ratliff, Mary Sieggreen, Nicole Walsh • **Assessment of Pressure Injuries and Monitoring of Healing:** Kerrie Coleman, Patricia Davies, Suhaida Binte Ramli, Ann Marie Nie, Catherine Ratliff • **Pain Assessment and Treatment:** Clarissa Young (leader), Widasari Sri Gitarja, Chak Hau Pang, Barbara Pieper, Tina Meyers, Andrea Pokorná, Valentina Vanzi • **Cleansing and Debridement** Shan Bergin, Patricia Davies, Rosemary Hill, Harikrishna Nair, Wan Yin Ping, Pamela Scarborough, David Voegeli • **Infection and Biofilms:** Robyn Rayner (leader), Evan Call, Emma Daza, Jeannie Donnelly, Dea Kent, Gojiro Nakagami, Lea Whittington • **Wound Dressings:** Maria Ten Hove (leader), Mikyung Cho, Reba J. Giles, David Voegeli, Tan Wei Xian, Saldy Yusuf • **Biological Dressings:** Laura Edsberg (leader), Michelle Carr, Elizabeth Faust, Eun Jin Han, Takafumi Kadono, Anna Polak, Jakub Taradaj, Quek Yanting • **Growth Factors:** Laura Edsberg (leader), Michelle Carr, Elizabeth Faust, Eun Jin Han, Takafumi Kadono, Anna Polak, Jakub Taradaj, Quek Yanting • **Biophysical Agents:** Sharon Boxall, Anna Polak, Hiske Smart, Gregory M. Toy • **Pressure Injury Surgery:** Emily Haesler (leader), Aamir Siddiqui, Rebecca Iseli, Julie Jordan-O'Brien • **Measuring Pressure Injury Prevalence and Incidence:** Dan Berlowitz, Janet Cuddigan, Emily Haesler • **Implementing Best Practice in Clinical Settings:** Kimberly Le Blanc (leader), Dimitri Beeckman, Maria Helena Larcher Caliri, Kathleen Finlayson, Bonnie Fraser, Patrícia Homem-Silver, Hongyang Hu, Mei-Yu Hsu, Wen-Pei Huang, Crystal McCallum, Jill Trelease, Louise Webber, Tracey Yap • **Heath Professional Education:** Emily Haesler (leader), Katie Capitulo, Margaret Edmondson, Ednalda Maria Franck, Aimee Garcia, Patrícia Homem-Silva, Jung Yoon Kim, Tamara Page, Diane Maydick Youngberg • **Quality of Life, Self-care and Education:** Emily Haesler (leader), Bernadette McNally, Sivagame Maniya, Lena Gunningberg, Denise Hibbert, Ann Marie Kassab, Yuwadee Kestsumpun, Lynn Tabor • **Quality Indicators:** Joyce Pittman, Emily Haesler, Ruud Halfens

ACKNOWLEDGEMENTS

Acknowledgements & In-Kind Support

Special acknowledgement and thanks to the 2009 and 2014 Guideline Development Groups and Small Working Group members from EPUAP, NPIAP and PPIA who developed the first two editions of this guideline. The work in this *International Guideline* edition builds on research that was appraised and summarized by earlier guideline development teams.

Emily Haesler, PhD

Interim Methodologist (literature update, review and analysis during the interim between formal guideline development activities [2013 to 2017])

Jan Kottner, PhD

Lead organizer and convener of the Guideline Governance Group

Paul Haesler, BSc (Hons)

Web development and IT support for guideline management and evidence appraisal online platform, patient consumer survey, stakeholder review process and strength of recommendation online platform

Knightime Designs

Classification illustrations (*Chapter 12*) and repositioning illustrations (*Chapter 8*)

McKenna Management

Management of guideline administration and marketing

La Trobe University, Australia

Electronic database, journal access and interlibrary loan services

Australian National University, Australia

Ethics approval for patient consumer survey

Photography in Chapter 12

Category/Stage I pressure injury: reproduced with permission from NPIAP and available for purchase from the NPIAP website

Category/Stage II pressure injury: Joyce Black, used with permission

Category/Stage III and IV and unstageable pressure injuries: Keryln Carville, used with permission

Suspected Deep Tissue Injury: available for free download from Wounds Australia website

Mucosal membrane pressure injury: Tracy Nowicki, used with permission

Special thanks go to Emily Haesler who did an extraordinary job in managing the complexities of an

international, comprehensive, systematic review of the research literature and development of this revised and expanded guideline on pressure injury prevention and treatment.

Translation

The following experts completed data extraction for papers in languages other than English:

Jan Kottner

Takafumi Kadono

Maria Helena Larcher Caliri

Patient Consumers and Stakeholders

Special thanks to over 1,200 patient consumers and their informal/family caregivers who contributed to the guideline development through participation in the international patient consumer survey.

Special thanks to the many stakeholders who reviewed the guideline processes and drafts. All stakeholder comments were reviewed by the GGG and revisions were made based on the comments received. We appreciate the investment of health professionals, researchers, educators and industry from all over the world who took time to share their expertise and thoughtful critique.

SPONSOR ACKNOWLEDGEMENTS

The European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP) and the Pan Pacific Pressure Injury Alliance (PPPIA) gratefully acknowledge the contributions of the following individuals and groups for financially supporting the presentation and dissemination of the guideline. All financial contributions were made after the guideline development phase and in *no way influenced* the development of the guideline or its final content. Financial contributions are being used for the printing and dissemination of the guideline and associated educational products. The following companies provided unrestricted education grants:

Gold Level Sponsor

Mölnlycke®

Silver Level Sponsors

ARJO

Medela, LLC

Smith + Nephew

TABLE OF CONTENTS

1	Introduction	
	Foreword.....	1
	Abstract.....	1
	Limitations and Appropriate Use of this Guideline	2
	Strengths of Evidence and Strengths of Recommendation	2
	Accessing the Guideline.....	3
	Guideline Developers.....	4
	Acknowledgements	6
	Sponsor Acknowledgements	7
	Table of Contents.....	8
	Table of Figures and Tables	9
	Guideline Background	11
	Background	
2	The Etiology of Pressure Injuries	16
3	Populations with Specific Pressure Injury Related Needs.....	28
	Prevention of Pressure Injuries	
4	Risk Factors and Risk Assessment	38
5	Skin and Tissue Assessment	73
6	Preventive Skin Care	84
	Interventions for Prevention and Treatment of Pressure Injuries	
7	Nutrition in Pressure Injury Prevention and Treatment.....	94
8	Repositioning and Early Mobilization.....	115
9	Heel Pressure Injuries.....	145
10	Support Surfaces.....	155
11	Device Related Pressure Injuries.....	181
	Treatment of Pressure Injuries	
12	Classifying Pressure Injuries.....	194
13	Pressure Injury Assessment and Monitoring of Healing	209
14	Pain Assessment and Treatment.....	224
15	Supporting Healing.....	235
16	Cleansing and Debridement.....	237
17	Infection and Biofilms.....	251
18	Wound Dressings for Treatment of Pressure Injuries.....	267
19	Biological Dressings for Treatment of Pressure Injuries.....	279
20	Growth Factors for Treatment of Pressure Injuries	282
21	Biophysical Agents.....	286
22	Pressure Injury Surgery	300
	Implementing the Guideline	
23	Measuring Pressure Injury Prevalence and Incidence	315
24	Implementing Best Practice in Clinical Settings	322
25	Health Professional Education.....	340
26	Quality of life, Self-care and Education	349
27	Quality Indicators for this Guideline	362
28	Further Research Needs	373
29	Glossary	
30	Appendices	
	Appendix One: Guideline Methodology	391

TABLE OF FIGURES AND TABLES

Figure 2.1	Factors influencing the susceptibility of an individual for developing pressure injuries	17
Figure 2.2	Schematic depiction of the process of cell damage	19
Figure 2.3	Tolerance behavior of soft tissues subjected to sustained mechanical loads	20
Figure 2.4	Contributors to cell damage and tissue necrosis in pressure injuries	23
Figure 4.1	Factors influencing the susceptibility of an individual for developing pressure injuries	38
Figure 8.1	30° tilt	124
Figure 8.2	Maximum head of bed elevation	125
Figure 8.3	Sitting upright in an armchair with the feet on the ground	130
Figure 8.4	Sitting back in an armchair with the lower legs on a rest	130
Figure 13.1	Patient consumer and informal caregiver goals of care	211
Figure 17.1	International Wound Infection Institute Wound Infection Continuum	252
Figure 23.1	Formula for calculating point prevalence	315
Figure 23.2	Formula for calculating cumulative incidence	315
Figure 23.3	Formula for calculating incidence density	316
Figure 23.4	Formula for calculating facility-acquired pressure injury rate	316
Figure 27.1	Quality indicators (QI) for pressure injuries based on recommendations and good practice statements in this guideline	363

Tables

Table 4.1	Major risk factor categories and their influence on components of the conceptual framework	39
Table 4.2	Summary of evidence for measures of mobility and activity as risk factors for pressure injuries	41
Table 4.3	Summary of evidence for measures of skin status as risk factors for pressure injuries	43
Table 4.4	Summary of evidence for measures of perfusion, circulation and oxygenation as risk factors for pressure injuries	45
Table 4.5	Summary of evidence for measures of nutritional status as a risk factor for pressure injury development	47
Table 4.6	Summary of evidence for measures of skin moisture as a risk factor for pressure injury development	48
Table 4.7	Summary of evidence for increased body temperature as a risk factor for pressure injury development	49
Table 4.8	Summary of evidence for measures of older age as a risk factor for pressure injury development	50
Table 4.9	Summary of evidence for measures of sensory perception as a risk factor for pressure injury development	50
Table 4.10	Summary of evidence for hematological measures as risk factors for pressure injuries	51
Table 4.11	Summary of evidence for mental and general health status measures as risk factors for pressure injuries	52
Table 4.12	Summary of evidence for demographic characteristics as risk factors for pressure injuries	53
Table 4.13	Summary of evidence for factors specific to adults in the operating room	55
Table 4.14	Summary of evidence for factors specific to individuals in critical care	56
Table 4.15	Summary of evidence for factors specific to neonates and children	58
Table 4.16	Summary of evidence for risk assessment tools	63
Table 4.17	Comparison of risk factors identified in epidemiological studies and commonly used risk assessment tools	65
Table 4.18	Psychometric qualities of major risk assessment tools	66
Table 5.1	Psychometric properties of techniques to assess erythema	76
Table 5.2	Psychometric properties of different devices for SEM/edema measurements	79
Table 7.1	Summary of nutrition screening tool validation studies	96
Table 7.2	Recommendations on protein requirements for populations at pressure injury risk	99
Table 7.3	Recommendations on energy requirements for populations at pressure injury risk	100
Table 7.4	Pediatric nutrition screening and assessment tools	108

Table 8.1	Example of a progressive seating protocol	134
Table 8.2	Pressure points of particular concern in different surgical positions (with illustrations)	137
Table 10.1	Consensus on characteristics that constitute a single layer high specification foam mattress	161
Table 12.1	Healthy skin	197
Table 12.2	Pressure injury Category/Stages: photographs and illustrations	198
Table 12.3	Mucous membrane pressure injury	199
Table 12.4	Reliability of categorization/staging using various pressure injury classification systems	200
Table 12.5	Comparison of pressure injury definitions in classification systems commonly used in Europe and the Pan-Pacific	203
Table 12.6	Comparison of pressure injury definitions in classification systems commonly used in the US	205
Table 13.1	Pressure injury healing times reported in the literature	213
Table 13.2	Pressure injury assessment and monitoring tools	219
Table 14.1	Selection of pain assessment tools appropriate for assessing pressure injury pain	226
Table 16.1	Examples of irrigation pressure for wound cleansing	238
Table 16.2	Topical antimicrobial therapies	239
Table 17.1	Procedure for using Levine technique for wound	255
Table 17.2	Topical wound infection therapies	259
Table 21.1	Forms of biophysical agents	286
Table 23.1	Ranges of pressure injury prevalence and incidence reported in selected literature	318
Table 24.1	Summary of evidence supporting stakeholder engagement as an initiative to reduce pressure injuries	329
Table 24.2	Summary of evidence for using evidence-based information systems as an initiative to reduce pressure injuries	330
Table 24.3	Summary of evidence for decision support tools as an initiative to reduce pressure injuries	332
Table 24.4	Summary of evidence for clinical leadership as an initiative to reduce pressure injuries	333
Table 24.5	Summary of evidence for education as an initiative to reduce pressure injuries	334
Table 24.6	Summary of evidence for ongoing evaluation as an initiative to reduce pressure injuries	335
Table 24.7	Summary of evidence for promoting the initiative as an impetus to reduce pressure injuries	336
Table 25.1	Selection of assessment tools appropriate for assessing knowledge or attitudes	342
Table 25.2	Outcomes for multi-faceted pressure injury prevention and treatment education programs	343
Table 25.3	Components used in multi-faceted pressure injury prevention and treatment education programs	344
Table 25.4	Outcomes for didactic/lecture-style pressure injury prevention and treatment education programs	345
Table 25.5	Outcomes for computer-based pressure injury prevention and treatment education programs	346
Table 26.1	Knowledge and HRQoL assessment tools tested in individuals with or at high risk of pressure injuries	352
Table 26.2	Impact of education and lifestyle skills interventions on outcome measures of significance	354
Table 26.3	Intervention components that demonstrated a benefit on at least one outcome measure of significance	356
Table 26.4	Patient consumer rating of importance for pressure injury education topics	359
Table 30.1	Critical domains for systematic reviews to meet for inclusion	394
Table 30.2	Relationship between appraisal criteria and quality domains for risk factor studies	396
Table 30.3	Level of evidence for intervention studies	397
Table 30.4	Adapted levels of evidence for diagnostic studies	397
Table 30.5	Adapted levels of evidence for prognostic studies	397
Table 30.6	Strength of evidence rating for each recommendation	399
Table 30.7	Five types of recommendations	401
Table 30.8	Guideline development team strategies to promote consumer engagement	403

GUIDELINE BACKGROUND

Significance

Pressure injuries are a frequently occurring health problem throughout the world. They are a painful, costly, and often preventable complication for which many individuals are at risk.

Around the world, pressure injury prevalence in healthcare settings ranges from 0%¹ to 72.5%,²⁻⁴ with large variations observed between different geographic and clinical settings. A recent systematic review reported global point prevalence of pressure injuries in acute hospitals at 14.8% and period prevalence at 11.6%, with a mean incidence of 6.3%.⁴ In general acute care, there appears to be a gradual and ongoing decline in pressure injury prevalence over the two decades,⁵ driven in part by growing international health policy focus on pressure injury prevention. Prevalence and incidence rates are generally higher in unique populations who are at elevated risk, such as those receiving palliative care,⁶ those with spinal cord injuries,⁷ neonates and children,^{8,9} and individuals in critical care¹⁰⁻¹² or palliative care.¹³ Although direct comparisons between prevalence studies are confounded by different methodologies and clinical contexts, the evidence indicates that pressure injuries are a commonly occurring health concern globally.¹²

Pressure injuries represent a major burden of sickness and reduced quality of life for patient consumers and their care givers.¹⁴⁻²² Increased morbidity and mortality associated with pressure injury development in hospitalized individuals is documented in multiple studies.^{16,23-25} Hospital lengths of stay, readmission rates, and financial costs of care are greater in individuals who develop a pressure injury than in those remaining pressure injury free.²⁶⁻²⁸ Additionally, the personal burden associated with a chronic wound, includes pain and discomfort,^{20,29} stress, anxiety and depression,^{16,23,24} and declines in autonomy,¹⁴ security and spiritual wellbeing¹⁴ and social functioning,²⁴ Further, individuals at risk or having pressure injuries identify pain as one of their most significant concerns.^{30,31}

Pressure injuries increase hospital costs significantly. However, there is a paucity of robust data on costs of pressure injury care. The available evidence presents health economic reports that use different methodologies and currencies, and represent costs associated with care in different clinical settings, and geographic locations and healthcare systems. In the US, pressure injury care is estimated to approach \$11.6 billion annually (USD in the period 2000 to 2012).³²⁻³⁶ Cost of individual patient care ranged between \$500 (USD) and \$152,000 (USD).^{32,33,35,37} In Australia, the mean hospital stay for pressure injuries is 4.3 days, at a financial cost of \$699 to \$840 (AUD) per hospital bed day,³⁸ suggesting mean costs of \$3,600 (AUD) for inpatient care for a pressure injury. Costs per pressure injury were similar in a Canadian cost analysis conducted in the community that reported estimated costs of treating one pressure injury at \$4,745 (Canadian dollars, 2013).³⁹ Reports from Singapore show costs of treating one pressure injury between S\$4,546 and \$13,138 (Singapore dollars in 2016) depending on wound severity.^{40,41} European economic models suggest the cost of illness associated with pressure injuries consumes up to 1.4% of healthcare expenditure in the Netherlands^{42,43} or between \$362 million and \$2.8 billion annually (USD in 2009).⁴³ Proportional costs are higher in the United Kingdom (UK) where pressure injuries are reported to cost up to 4% of the annual healthcare budget.^{44,45} When community healthcare costs are added to hospital costs, pressure injury treatment consumes up to £2.1 billion of the National Health Service (NHS) budget.⁴⁶ In New Zealand, total cost of pressure injury treatment is estimated at \$694 million annually (New Zealand dollars, 2015).⁴⁷ Although direct comparison is not possible due to the different cost outcomes reported, these studies demonstrate the high economic burden of pressure injuries around the world, that is likely to continue increasing as populations age.⁴⁸

Purpose and Scope

The goal of this guideline is to provide evidence-based recommendations for the prevention and treatment of pressure injuries that can be used by health professionals, patient consumers and informal caregivers throughout the world. The guideline is intended for the use of all health professionals, regardless of clinical discipline, who are involved in the care of individuals who are at risk of developing pressure injuries, or those with an existing pressure injury. The guideline is intended to apply to all clinical settings, including acute care, rehabilitation care, long term care, assisted living at home, and unless specifically stated, can be considered appropriate for all individuals with or at risk of pressure injuries. The guideline includes further guidance for population groups with additional needs, including those in palliative care, critical care, community, or operating room settings, individuals with obesity, individuals with spinal cord injury, and neonates and children. When applicable, the specific needs of older adults are addressed, although it is acknowledged that much of the research on prevention and treatment of pressure injuries is conducted with older adults. Classification of mucosal membrane pressure ulcers is beyond the scope of this guideline.

Guideline Development

The European Pressure Ulcer Advisory Panel (EPUAP), the National Pressure Injury Advisory Panel (NPIAP), and the Pan Pacific Pressure Injury Alliance (PPPIA) collaborated to produce this evidence-based guideline on the prevention and treatment of pressure injuries. This *International Guideline* edition builds on the evidence presented in the two previous editions of this guideline.

The guideline was produced by an interprofessional Guideline Governance Group (GGG) and numerous Small Working Groups (SWGs), each consisting of representatives of the partner and associate organizations. The GGG determined and monitored the guideline development process with the assistance and management of a guideline methodologist.

The first step in the guideline development process was refining the methodology. The GGG reviewed recent approaches to clinical guideline development to ensure that recognized international standards were met in the guideline development. The methodology from the previous editions of the guideline was reviewed, updated and published in a peer-reviewed journal.⁴⁹ Next, the GGG commissioned a comprehensive review of the literature on pressure injury prevention and treatment in several electronic databases, using a sensitive search strategy to identify research published since the previous guideline edition. All retrieved references were screened on pre-determined inclusion criteria and preliminary data extraction tables were completed. In a second step, the retrieved evidence was evaluated, and thereafter the full texts were divided according to specific topic of interest related to pressure injuries. With the assistance of the methodologist, the SWG members conducted critical appraisal of the evidence, assigned a level of evidence to each study using a pre-determined scale and refined the evidence tables. The evidence was then combined in evidence-to-decision frameworks addressing the pre-determined clinical questions. Each SWG formulated conclusions about the body of available evidence and developed recommendations that emerged from the evidence. Recommendations from the second edition of the guideline were reviewed and revised based on insights from new evidence and an analysis of the current cumulative body of evidence. The strength of the body of evidence was determined based on the volume, level and consistency of the evidence. This rating identifies the strength of cumulative evidence supporting a recommendation. Evidence to decision frameworks, recommendations and evidence summaries were reviewed by the GGG. For important areas of practice in which evidence was lacking, the GGG made good practice statements to guide clinical practice. The guideline was made available to registered stakeholders on the guideline website for further input and feedback.

The GGG also conducted an international survey of patient consumers and informal caregivers to gain insight on priority issues for patients.^{30,31} The project, which included development of a survey designed with attention to the needs of the target population, received approval from the Australian National University Human Research Ethics Committee (Protocol 2018/066). The survey was promoted internationally by pressure injury organizations and the SWG members.

The final stage involved determining the strength of each recommendation statement. The body of evidence underpinning each recommendation was ranked by the SWG based on volume, consistency and level of evidence, resource implications, acceptability and priority to stakeholder and feasibility to implement. Each member of the guideline development team was invited to review this data for every recommendation and to participate in a web-based consensus process to assign the strength of recommendations. The recommendation strength represents the confidence a health professional can place in each recommendation, with consideration to the strength of supporting evidence; clinical risks versus benefits; cost effectiveness; and systems implications.

More detail on the methodological process is outlined in *Appendix 1: Guideline Methodology*, and available from the guideline website and the peer-reviewed publication.⁴⁹

Guideline Recommendations and Good Practice Statements

Recommendations are systematically developed statements to assist health professionals, patient consumers and informal caregiver to make decisions about appropriate health care for specific clinical conditions. The recommendations and good practice statements may not be appropriate for use in all contexts, settings and circumstances. The guidance provided should not be considered medical advice for specific cases.

The recommendations and good practice statements in this guideline are a general guide to appropriate clinical practice, to be implemented by qualified health professionals subject to their clinical judgment of each individual case, and in consideration of the patient consumer's preferences and available resources. The guideline should be implemented in a culturally aware and respectful manner in accordance with the principles of protection, participation and partnership.

This guideline, and any recommendations within, are intended for educational and informational purposes only. Generic names of products are provided. Nothing in this guideline is intended as an endorsement of a specific product.

The guideline consists of five main sections: background material, prevention of pressure injuries, topics relevant to prevention and treatment of pressure injuries, treatment of pressure injuries and implementing the guideline.

The *Background* chapters provide an introductory section to the guideline. The etiology chapter focuses on basic science. The needs of specific patient populations is identified in the *Purpose and Scope* also outlined in the *Background* chapters. The specific risks faced by specific population groups are discussed, with reference to sections of the guideline that are of particular significance to different population groups.

The *Prevention* chapters of the guideline comprises four topics: pressure injury risk and assessment, assessment of skin and tissue and preventive skin care. Risk assessment is an essential component of clinical practice that aims to identify individuals who are susceptible to pressure injury development, in order to plan and implement care that addresses the individual's risks. Skin assessment is crucial in pressure injury prevention because skin status is a significant risk factor for pressure injury development. Further, the skin can serve as an indicator of early pressure damage and guides evaluation of preventive care. The identification and differentiation of erythema and considerations when assessing individuals with darkly pigmented skin are discussed throughout this chapter. Preventive skin care, which focuses on promotion of skin integrity and protecting the skin from damage, is a key component of pressure injury prevention discussed in the *Prevention* guideline chapters.

The guideline chapters making up the section on *Interventions for Prevention and Treatment of Pressure Injuries* focus on five areas of care (nutrition, repositioning and early mobilization, heel pressure injuries, support surfaces and device related pressure injuries) that are important in both prevention and treatment of pressure injuries. Comprehensive recommendations on strategies to promote nutritional status, thereby reducing pressure injury risk and/or promoting wound healing are provided. The importance of ensuring adequate caloric, protein, vitamin, mineral and water/fluid intake is highlighted. Repositioning and its frequency should be considered for all at risk individuals and must take into consideration the clinical condition of the individual and the support surface in use. The importance of correct manual handling technique, pressure relief schedules and early mobilization is discussed. Heel pressure injuries are a challenge to prevent and manage. The small surface area of the heel is covered by a small volume of subcutaneous tissue that can be exposed to high mechanical load in individuals on bedrest. The recommendations in this section of the guideline address the importance of assessment, positioning and heel protection, while avoiding potential complications. The recommendations in the support surfaces chapter address mattress/overlay and bed use, seats and cushions, and other forms of support surface. Guidance on product selection, use safety and maintenance is provided; however, manufacturers' information should always be followed. Devices (both medically related and general objects/furniture) are associated with a high risk of pressure injuries. These pressure injuries often conform to the pattern or shape of the device/object and develop due to prolonged, unrelieved pressure on the skin. The recommendations in this section address assessment, device selection, strategies to redistribute pressure, and skin protection.

The guideline chapters on *Treatment of Pressure Injuries* discuss assessment and treatment once a pressure injury has occurred. The importance of accurate diagnosis, classification and assessment of pressure injuries is highlighted. Pressure injury assessment and monitoring of healing provides an evaluation of care that informs the development of a comprehensive, ongoing treatment plan. Pressure injuries are painful; however, pain is often under-recognized and under-treated. Recommendations in the guideline chapter on pain focus on assessment and treatment of pain in the context of providing quality care that addresses the individuals care goals. The treatment chapters of the guideline discuss the overarching principle of wound bed preparation and research evidence specific to providing wound care for pressure injuries. Critical approaches necessary to prepare the wound bed for healing including cleansing, debridement, selection of the most appropriate wound dressings, infection management and other treatments for the wound bed (e.g. biophysical agents) are discussed. These topics are relevant to the management of all chronic wounds; however, the intention of this guideline is to discuss the research specific to pressure injuries. Finally, the treatment chapters also include discussion of the management of an individual undergoing pressure injury surgery, which may be required for non-healing pressure injuries and/or when the individual has clinical signs of severe worsening infection or sepsis.

The guideline chapters on *Implementing the Guideline* address organization and professional level strategies for effective implementation of the clinical recommendations in this guideline. This includes implementation strategy (including facilitators and barriers), health professional education, supporting the patient consumers and their caregivers, measurement on monitoring pressure injuries within a facility/organization, and quality indicators for monitoring guideline implementation. Finally, the guideline includes discussion of ongoing research needs. The paucity of high quality research on prevention and treatment of pressure injuries was highlighted in this guideline update. And areas for which there is a strong need of good quality evidence attained from well-designed studies are identified.

References

1. Hiser B, Rochette J, Philbin S, Lowerhouse N, Terburgh C, Pietsch C. Implementing a pressure ulcer prevention program and enhancing the role of the CWOCN: impact on outcomes. *Ostomy Wound Management*, 2006; 52(2): 48-59.
2. Samaniego IA. A sore spot in pediatrics: risk factors for pressure ulcers. *Pediatr Nurs*, 2003; 29(4): 278-82.
3. Tubaihat A, Papanikolaou P, Anthony D, Habiballah L. Pressure ulcers prevalence in the acute care setting: A systematic review, 2000-2015. *Clin Nurs Res*, 2018; 27(6): 643-659.
4. Al Mutairi KB, Hendrie D. Global incidence and prevalence of pressure injuries in public hospitals: A systematic review. *Wound Medicine*, 2018; 22: 23-31.
5. Goldberg M, *General Acute Care*, in *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future*. B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
6. Tippet AW. Wounds at the end of life. *Wounds: A Compendium of Clinical Research & Practice*, 2005; 17(4): 91-98.
7. Houghton PE, Campbell KE, CPG Panel, Canadian Best Practice Guidelines for the Prevention and Management of Pressure Ulcers in People with Spinal Cord Injury. A resource handbook for clinicians. 2013, Ontario Neurotrauma Foundation: <http://www.onf.org>.
8. Baharestani MM, Ratliff CR. Pressure ulcers in neonates and children: an NPUAP white paper. *Adv Skin Wound Care*, 2007; 20(4): 208.
9. Baharestani M, *Pressure Ulcers in Pediatric Populations*, in *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future*, B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
10. Vangilder C, Amlung S, Harrison P, Meyer S. Results of the 2008 - 2009 International Pressure Ulcer Prevalence Survey and a 3-year, acute care, unit-specific analysis. *Ostomy Wound Management*, 2009; 55: 39-45.
11. Cuddigan J, *Critical Care*, in *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future*, B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
12. Chaboyer WP, Thalib L, Harbeck EL, Coyer FM, Blot S, Bull CF, Nogueira PC, Lin FF. Incidence and prevalence of pressure injuries in adult intensive care patients: A systematic review and meta-analysis. *Crit Care Med*, 2018; 07: 07.
13. Artico M, Dante A, D'Angelo D, Lamarca L, Mastroianni C, Petitti T, Piredda M, De Marinis MG. Prevalence, incidence and associated factors of pressure ulcers in home palliative care patients: A retrospective chart review. *Palliat Med*, 2017: 269216317737671.
14. Degenholtz H, Rosen J, Castle N, Mittal V, Liu D. The association between changes in health status and nursing home resident quality of life. *Gerontologist*, 2008; 48(5): 584-584.
15. Essex HN, Clark M, Sims J, Warriner A, Cullum N. Health-related quality of life in hospital inpatients with pressure ulceration: Assessment using generic health-related quality of life measures. *Wound Repair Regen*, 2009; 17(6): 797-805.
16. Galhardo VAC, Magalhaes MG, Blanes L, Juliano Y, Ferreira LM. Health-related quality of life and depression in older patients with pressure ulcers. *Wounds: A Compendium of Clinical Research & Practice*, 2010; 22(1): 20-26.
17. Thein H-H, Gomes T, Krahn MD, Wodchis WP. Health status utilities and the impact of pressure ulcers in long-term care residents in Ontario. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care and Rehabilitation*, 2010; 19(1): 81-89.
18. Yarkin O, Tamer S, Gamze O, Irem M, Huseyin B. Effect of surgery on psychiatric states and quality of life of paraplegics and quadriplegics with pressure sores and their primary caregivers. *Eur J Plast Surg*, 2009; 32(4): 173-176.
19. Gorecki C, Lamping DL, Brown JM, Madill A, Firth J, Nixon J. Development of a conceptual framework of health-related quality of life in pressure ulcers: A patient-focused approach. *Int J Nurs Stud*, 2010; 47(12): 1525-1534.
20. Gorecki C, Nixon J, Madill A, Firth J, Brown JM. What influences the impact of pressure ulcers on health-related quality of life? A qualitative patient-focused exploration of contributory factors. *Journal of Tissue Viability*, 2012; 21(1): 3-12.
21. Jackson DE, Durrant LA, Hutchinson M, Ballard CA, Neville S, Usher K. Living with multiple losses: Insights from patients living with pressure injury. *Collegian*, 2017.
22. Latimer S, Chaboyer W, Gillespie B. Patient participation in pressure injury prevention: Giving patient's a voice. *Scand J Caring Sci*, 2014; 28(4): 648-656.
23. Degenholtz H, Rosen J, Castle N, Mittal V, Liu D. The association between changes in health status and nursing home resident quality of life. *Gerontologist*, 2008; 48(5): 584-584.
24. Essex HN, Clark M, Sims J, Warriner A, Cullum N. Health-related quality of life in hospital inpatients with pressure ulceration: assessment using generic health-related quality of life measures. *Wound Repair and Regeneration*, 2009; 17(6): 797-805.
25. Allman R, Goode PS, Burst N, Bartolucci A, Thomas DR. Pressure ulcers, hospital complications, and disease severity: implications on hospital costs and length of stay. *Advances in Wound Care*, 1999; 12(1): 22-30.
26. Wilson J, Arnold P, Singh A, Kalsi-Ryan S, Fehlings M. Clinical prediction model for acute inpatient complications after traumatic cervical spinal cord injury: a subanalysis from the Surgical Timing in Acute Spinal Cord Injury Study. *J Neurosurg Spine*, 2012; 17(1): 46-51.
27. Wu Q, Ning GZ, Li YL, Feng HY, Feng SQ. Factors affecting the length of stay of patients with traumatic spinal cord injury in Tianjin, China. *J Spinal Cord Med*, 2013; 36(3): 237-242.
28. Lyder CH, Wang Y, Metersky M, Curry M, Kliman R, Verzier NR, Hunt DR. Hospital-acquired pressure ulcers: results from the national medicare patient safety monitoring system study. *Journal of the American Geriatrics Society*, 2012; 60(9): 1603-1608.
29. Gorecki C, Closs SJ, Nixon J, Briggs M. Patient-reported pressure ulcer pain: a mixed-methods systematic review. *J Pain Symptom Manage*, 2011; 42(3): 443-459.
30. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.

31. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis
32. Padula WV, Mishra MK, Makic MBF, Sullivan PW. Improving the quality of pressure ulcer care with prevention: a cost-effectiveness analysis. *Med Care*, 2011; 49(4): 385-392.
33. Young D, Shen J, Estocado N, Landers M. Financial impact of improved pressure ulcer staging in the acute hospital with use of a new tool, the NE1 Wound Assessment Tool. *Adv Skin Wound Care*, 2012; 25(4): 158-166.
34. Cuddigan J, Berlowitz D, Ayello E. Pressure ulcers in America: prevalence, incidence, and implications for the future. An executive summary of the National Pressure Ulcer Advisory Panel monograph. *Adv Skin Wound Care*, 2001; 14(4): 208-15.
35. Berlowitz D, Vandeusen Lukas C, Parker V, Niederhauser A, Silver J, Logan C, Ayello E. *Preventing pressure ulcers in hospitals: A toolkit for improving quality of care*. 2011 [cited September 2019]; Available from: <https://www.ahrq.gov/sites/default/files/publications/files/putoolkit.pdf>.
36. Padula WV, Pronovost PJ, Makic MBF, Wald HL, Moran D, Mishra MK, Meltzer DO. Value of hospital resources for effective pressure injury prevention: a cost-effectiveness analysis. *BMJ Quality & Safety*, 2018; 10: 10.
37. Banks M, Bauer J, Graves N, Ash S. Malnutrition and pressure ulcer risk in adults in Australian health care facilities. *Nutr Clin Pract*, 2010; 26(9): 896-901.
38. Norman RE, Gibb M, Dyer A, Prentice J, Yelland S, Cheng Q, Lazzarini PA, Carville K, Innes-Walker K, Finlayson K, Edwards H, Burn E, Graves N. Improved wound management at lower cost: A sensible goal for Australia. *Int Wound J*, 2016; 13(3): 303-316.
39. Chan BC, Nanwa N, Mittmann N, Bryant D, Coyte PC, Houghton PE. The average cost of pressure ulcer management in a community dwelling spinal cord injury population. *Int Wound J*, 2013; 10(4).
40. Tan B-K, Chong S-J, Chang VJ. An economic evaluation of chronic wound management in tertiary hospital. *Wound Pract Res*, 2016; 24(3): 130-136.
41. Lim ML, Ang SY. Impact of hospital-acquired pressure injuries on hospital costs: Experience of a tertiary hospital in Singapore. *Wound Pract Res*, 2017; 25(1): 42-47.
42. Severens J, Habraken JM, Duivenoorden S, Frederiks CMA. The cost of illness of pressure ulcers in the Netherlands. *Adv Skin Wound Care*, 2002; 15(2): 72-7.
43. Schuurman JP, Schoonhoven L, Defloor T, van Engelshoven I, van Ramshorst B, Buskens E. Economic evaluation of pressure ulcer care: A cost minimization analysis of preventative strategies. *Nurs Econ*, 2009; 27(6): 390-415.
44. Bennett G, Dealey C, Posnett J. The cost of pressure ulcers in the UK. *Age Ageing*, 2004; 33(3): 230-5.
45. Phillips CJ, Humphreys I, Fletcher J, Harding K, Chamberlain G, Macey S. Estimating the costs associated with the management of patients with chronic wounds using linked routine data. *Int Wound J*, 2015.
46. Large J. A cost-effective pressure damage prevention strategy. *Br J Nurs*, 2011; 20(6): S22-S25.
47. KPMG. *The case for investment in: A quality improvement programme to reduce pressure injuries in New Zealand*. 2015. [cited September 2019]; Available from: <https://www.hqsc.govt.nz/assets/Pressure-Injuries/PR/KPMG-pressure-injury-report-Jan-2016.pdf>.
48. Kim GH, Lee JY, J. K, Hyun JK, Park J. Prevalence of pressure injuries nationwide from 2009 to 2015: Results from the National Inpatient Sample Database in Korea. *Int J Environ Res Public Health*, 2019; 16(5): 704.
49. Kottner J, Cuddigan J, Carville K, Balzer K, Berlowitz D, Law S, Litchford M, Mitchell P, Moore Z, Pittman J, Sigaudou-Rousel D, Chang YY, Haesler E. Prevention and treatment of pressure ulcers/injuries: The protocol for the second update of the international Clinical Practice Guideline 2019. 28, 2019; 2: 51-58.

THE ETIOLOGY OF PRESSURE INJURIES

Introduction

A pressure injury is defined as localized damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear. Pressure injuries usually occur over a bony prominence but may also be related to a medical device or other object.

A pressure injury can occur due to the forces of a patient's bodyweight, or as a result of externally exerted forces such as those applied by a medical device or other object, or by a combination of these. The injury can present as intact (or unbroken) skin or an open wound and may be painful. The tissue damage occurs as a result of intense and/or prolonged exposure to sustained deformations in compression (perpendicular to the tissue surface), tension or shear (parallel to the tissue surface), or a combination of these loading modes. The tolerance of soft tissue for sustained deformations differs by tissue type and may also be affected by microclimate, perfusion, age, health status (either chronic or acute), comorbidities, and conditions of the soft tissues.

It is important to note that pressure injuries affecting the surface of a patient's body are not limited to the skin. For example, pressure injuries can occur on, within or underneath a mucous membrane, which is the moist lining of body cavities including the respiratory, gastrointestinal and genitourinary tracts.¹ Mucosal pressure injuries are primarily related to medical devices, typically caused by tubes and/or their stabilization equipment exerting sustained compressive and shear forces on these vulnerable mucosa and underlying tissues.¹

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

Different injury mechanisms impact various tissues, including cell deformation damage (in single cells), inflammation-related damage (in cells and tissues), and ischemia and reperfusion injury (also at cell and tissue levels). Sustained deformation of cells, vasculature and tissues are the driving force for all these damage pathways, from integrity and function of cell organelles to destruction of tissues and organs. For example, deformations may cause direct damage to the structures of cells, but also trigger inflammation and development of edema, distort the capillary network and reduce supply of nutrients to tissues, or cause lymphatic obstructions which will compromise clearance of metabolic waste products. Hence, the exposure to sustained cell and tissue deformations directly and indirectly cause formation and progression of cell and tissue damage in these multiple, interacting and escalating pathways.

Pressure Ulcer or Pressure Injury?

Since the first description of such injuries there has been debate regarding terminology. The oldest term is **decubitus**, originally described as **gangraena per decubitum** by Wohlleben (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 Groth² and a number of seminal studies and papers by Kosiak³ and Reichel.⁴ Groth² used the term **decubitus**, Reichel⁴ used **decubitus ulcers** and Kosiak³ used several terms including **ischemic ulcers**. None of these terms are accurately descriptive and Kosiak's³ 'ischemic ulcer' implies the limited etiological pathway that was assumed at that time.

The term **bedsore** was documented by Florence Nightingale in 1859,⁵ and became more commonly used following publication of *Bedsore Biomechanics*,⁶ 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, losing the apparent relation between the injury and a bed. Since the early 1990s the term **pressure ulcer**, has been commonly used; however, the term ulcer describes an open wound at the skin surface. This omits both deep tissue injury, an internal wound under intact/unbroken skin (see *Classification* section of this guideline) and Category/Stage I pressure injuries in which skin remains intact.

All the above terms are still in use by clinicians and/or patients. In Europe the term **pressure ulcer** is widely used, whilst in South-East Asia, Australia and New Zealand the term **pressure injury** has been adopted. The United States is transitioning to the term pressure injury; this is currently recommended by the US National Pressure Injury Advisory Panel and supported by many wound care organizations and regulatory bodies; however, discussions regarding terminology continue.⁷ Although none of these terms comprehensively describes the full etiology of these wounds,

they all refer to the same phenomenon described in the introduction to this guideline section. The terminology remains the subject of ongoing discussion and debate. In this version of the guideline the term **pressure injury** is used.

Factors That Influence Susceptibility to Pressure Injuries

A number of factors that may influence an individual's risk of developing pressure injuries have been described in relevant research and are discussed in the *Risk Factors and Risk Assessment* chapter of this guideline. The work of Coleman and colleagues (2014)⁸ has classified the relevant risk factors into two groups (Figure 2.1):

1. Mechanical boundary conditions, including magnitudes and time durations of the applied mechanical loads, and their mode of action, such as compressive or shear.
2. Tolerance of the individual (internal anatomy including the prominence of bony structures, tissue morphology, mechanical properties of tissues, tissue repair capacity and transport and thermal properties of tissues).

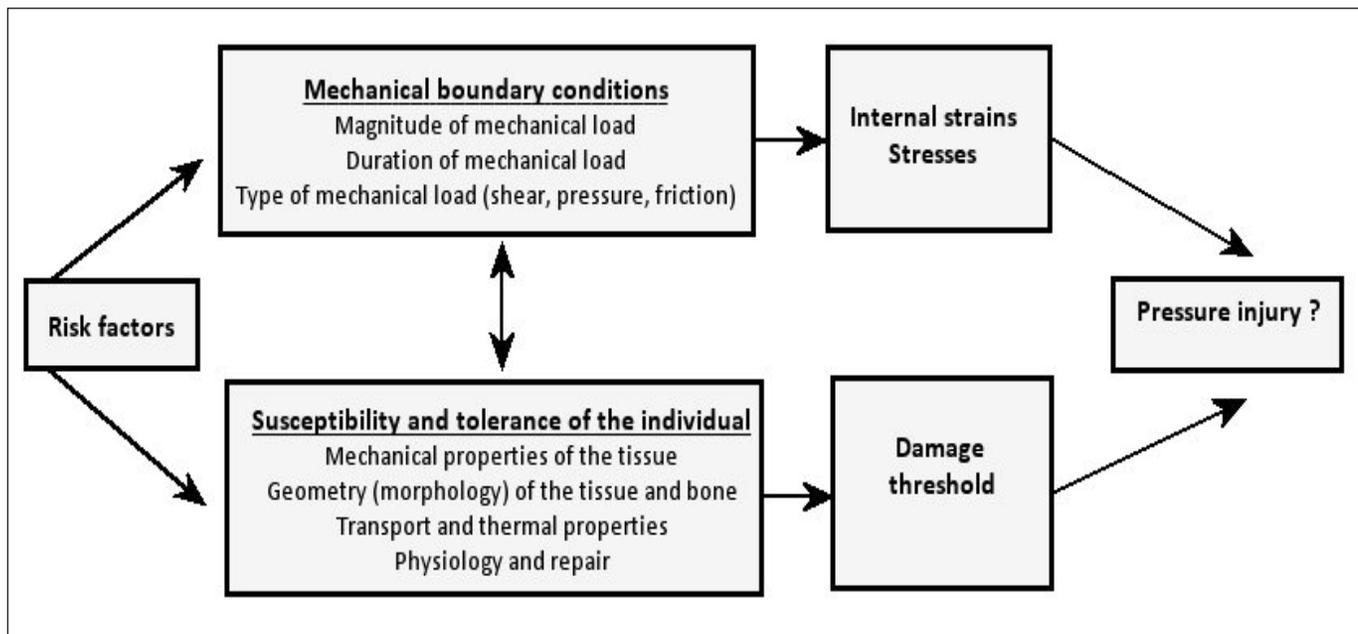


Figure 2.1: Factors influencing the susceptibility of an individual for developing pressure injuries (Adapted from Oomens (1985)⁹ by Coleman et. al. (2014)⁸ and reproduced with permission)

The first group of factors dictates the internal deformations, strains and stresses within soft tissues as well as the quality of vascular perfusion and lymphatic drainage under the applied loads. The latter set of factors determines the tissue injury threshold of the individual. Acting together, the two factor groups determine the time taken for a pressure injury to develop in the individual, and the extent and severity to which it will develop if that individual or an affected body part of the individual is immobile and/or insensate.

Mechanical Boundary Conditions

Magnitude and Duration of the Mechanical Load

This section defines a number of commonly used mechanical terms.

Mechanical load comprises all types of force that are applied to the soft tissues of an individual as a result of contact between the skin and a solid surface (including air-filled or water-filled support surfaces, medical devices and other types of surfaces). It includes bodyweight forces transferred through bony structures which are transmitted through the soft tissue to the supporting surface. External 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 almost all real-world scenarios, the interacting force is a combination of a normal and a shear force.

Pressure is defined as normal force per unit surface area (of skin or underlying tissue).

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 such as the micro-roughness and wetness level 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 specifically to pressure injuries, including this guideline, friction is used to define the contact force parallel to the skin surface due to internal bodyweight loads or forces exerted by a medical device. In either case, the applied frictional forces may be static (if there is no relative movement between the skin and the contacting surface/material) or dynamic (when such relative movement occurs).^{10,11}

Continuous or repetitive movement, rubbing or sliding of a material (e.g., a textile) or another body part along the skin (i.e. including skin-to-skin contact e.g., where the lower extremities lie on top of each other) can result in redness, inflammation or a lesion referred to as a **friction blister**. These blisters are not considered to be pressure injuries. However, 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 distort and deform, resulting in a **strain** (a measure of the relative deformation) and **stress** (force transferred per unit area) within the tissues. Excessive internal strains and stresses in tissues may impair transport phenomena in cells by causing damage to cell structures such as the cytoskeleton or plasma membrane and may also hinder transport processes within the tissues (e.g., by reducing blood perfusion, impairing lymphatic function and affecting transport in interstitial spaces). Cell death, in turn, triggers an inflammatory response that causes increased permeability of the vasculature as gaps develop between endothelial cells, resulting in inflammatory edema^{12,13} that further increase the mechanical loads on cells and tissues through a rise in interstitial pressures (see *Figure 2.2*).

The specific ways by which cells and tissues are affected by mechanical loads are a complex process that depends on anatomical structure and morphology (the size and shape of the different tissue layers) and the biophysical and mechanical properties of the tissues involved (e.g., density and composition, water contents, stiffness, strength and diffusion properties), as well as the magnitudes and distributions of the mechanical forces that are applied to the tissue at the regions of contact with the supporting surface or medical device.

Morphology, mechanical properties and tissue tolerance can all change over time as a result of aging, lifestyle, chronic injury, or disease.^{14,15} In general, external mechanical loading, even of a uniform nature, 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 or an oxygen mask pushing against the face), and some shear force will always exist. Accordingly, considerable deformations and strains may occur within the skin and deeper tissues in weight-bearing postures such as when lying in bed or sitting on a chair.

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 computational (finite element) models (a method of solving mechanical problems by means of a powerful computer and dedicated software) to estimate deformations, strains and stresses throughout the tissue structures, and predict the risk of cell and tissue damage.

Pressure injuries develop as a result of the internal response to bodyweight forces or external mechanical load. Understanding the etiology of pressure injuries relies on a knowledge concerning the internal cell and tissue responses to mechanical loads and not just what is apparent on the outside of the body or on the skin surface.¹⁶

Figure 2.2 presents a schematic description of the process of cell damage due to bodyweight forces and the resulting pressure and shear on skin that cause sustained mechanical deformations in tissues, including the contribution of inflammatory edema to the damage cascade. Pressure and shear loads can also originate from a medical device or other object that is in contact with the skin. Frictional forces (bodyweight-related shear) can be either static (when the body or a body part is stationary) or dynamic (such as during spontaneous or repetitive movements/rubbing, sliding in bed due to gravity or while repositioning the patient). Noteworthy is that this schematic description depicts the immediate and short-term damage factors; however, ischemia is also a factor which may be involved later in the damage cascade. Ischemic conditions may be caused by sustained vascular deformations. There is continuous interaction between ischemia and the inflammatory process described here, as inflammation affects the function of endothelial cells which comprise the capillary walls. The ischemic conditions may be further exacerbated by the build-up of localized edema and the associated increased interstitial pressures, as shown.

Type of loading and the tissue response

The primary cause of pressure injuries is sustained mechanical loads that are applied to soft biological tissues, generally but not necessarily near a bony prominence. The said mechanical loads can originate from bodyweight forces (body mass pulled by gravity) or from the environment, for example delivered by a medical device (e.g., a ventilation mask or a pulse oximeter which apply sustained forces and deformations to tissues that are in contact with these devices). Such medical devices are typically considerably stiffer than the skin, and the mismatch in mechanical properties between device and skin as well as underlying soft tissues causes focal deformations and mechanical stress concentrations in tissues near the contact sites with the device.^{17,18} Deformations of skin and/or deeper tissues due to bodyweight loads or exerted from the environment (e.g. a medical device) must be sustained for the tissue damage that characterizes a pressure injury to occur.

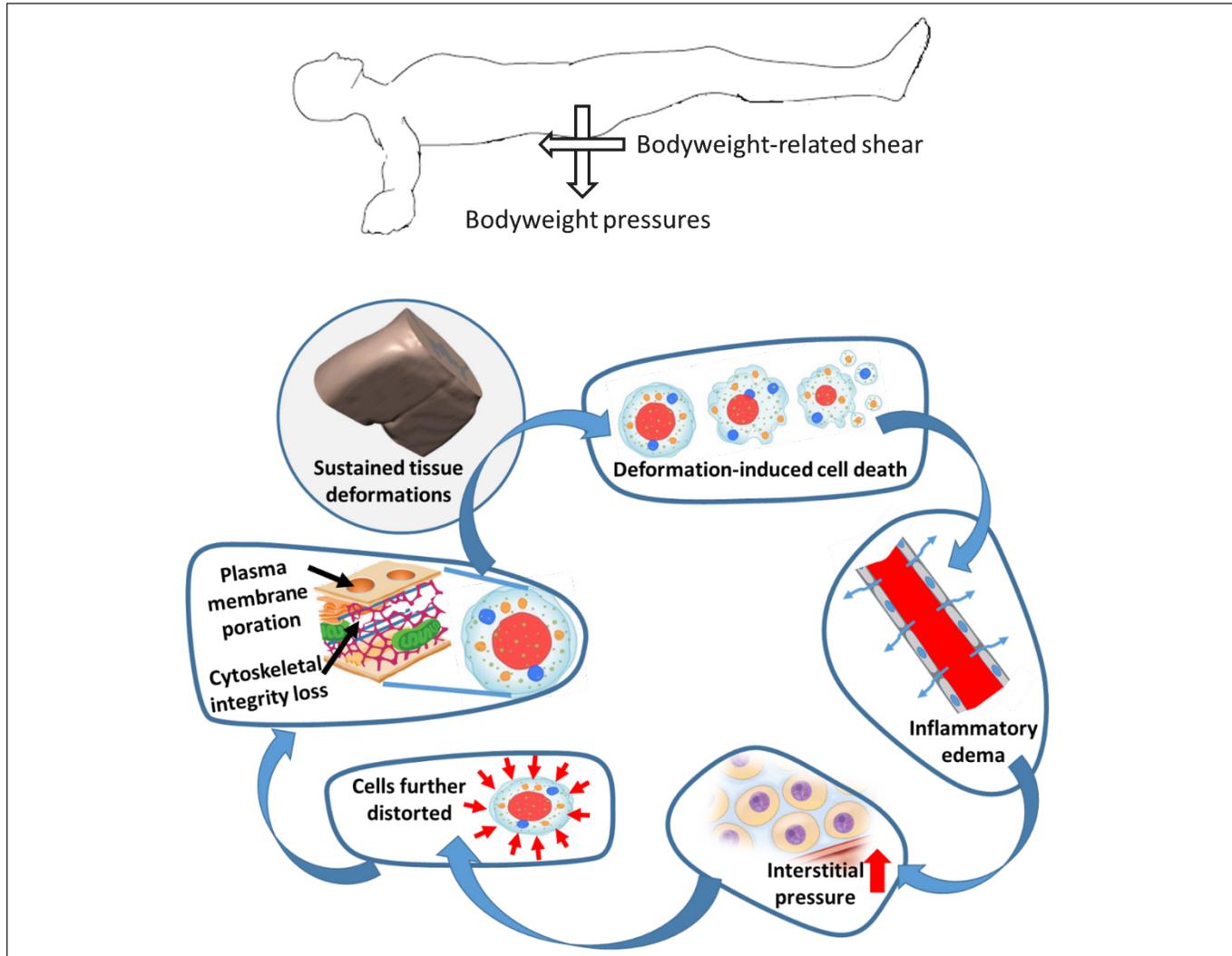


Figure 2.2: Schematic depiction of the process of cell damage (Reproduced with permission from Gefen¹⁹)

The magnitude of the *internal* mechanical load required to lead to tissue damage depends on the duration of time for which the load is applied, as well as on the specific biomechanical tolerance of the tissue subjected to the loading (which is a function of age, morphology, health conditions, and body system function including the tissue repair capacity). Application of both a high load for a short period or a low load for a prolonged period can lead to tissue damage.^{2,3,20-29}

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 minutes in a cell culture and tissue engineering models, although it may take hours of sustained loading before this is clinically visible as a pressure injury.^{30,31}

By contrast, **impact damage** resulting from rapid, high magnitude loading such as would occur as a result of an accident or trauma, does not fall under the definition of pressure injuries (although it similarly injures cells and tissues by application of mechanical loads). The etiological difference is essentially the time of exposure to the mechanical loads. With impact damage, a very high mechanical load is applied to the tissues and organs within a fraction of a second. The mass of the objects plays an important role and inertia effects leading to shock/pressure waves in the tissue may cause high external and internal damage, all within a fraction of a second.³² Impact damage is not considered a pressure injury because the primary damage occurs within fractions of a second.

The historical threshold function for damage developed by Reswick et. al.²⁹ depends on pressure applied to the skin and duration of applied pressures. Its development was based on observations of superficial (skin) damage in humans. Although Reswick et. al.²⁹ indicated that the function becomes asymptotic (meaning that it goes to infinity) for short durations of applied pressure, we now understand that the absolute limit on pressure magnitude is finite as shown in *Figure 2.3*. The Reswick curve²⁹ is therefore incorrect in the sense that it does not reflect the risk of tissue damage at the extremes, particularly for very short loading times.^{33,34} Sufficiently high loads can almost instantaneously cause (traumatic) damage to tissues at a microscopic level, which can be made visible with MRI or histological techniques. High loads applied for durations in the order of minutes can also cause cell death and tissue damage events, as known from clinical experience and as also supported by MRI and histological data from living model systems (animals and tissue engineering models). High loads are not necessarily traumatic impact loads but can be intense tissue deformation episodes that may occur in common clinical conditions (e.g., when using transfer boards, toilet seats, shower stools, overtightening oxygen masks, leaning against the rails of the bed etc.).^{17,28,30,35} Conversely, it is known empirically that low loads such as those experienced by healthy individuals daily (e.g., from the weight of clothing, or from wearing glasses, watches or jewelry) will not lead to damaged tissues even if applied for extended periods of time.^{33,34}

Due to variability in individual anatomies, tissue tolerances and confounding factors, it is not possible to determine generic quantitative values for tissue damage thresholds as a function of the pressure and exposure time; therefore, the axes of *Figure 2.3* are not scaled.^{26,29,35} Temperature, for example, has been shown to have a profound effect on the tolerance of tissue to pressure damage, and this plays an important role in interactions with support surfaces and medical devices.^{36,37} Other intrinsic confounding factors include macrovascular and microvascular diseases.

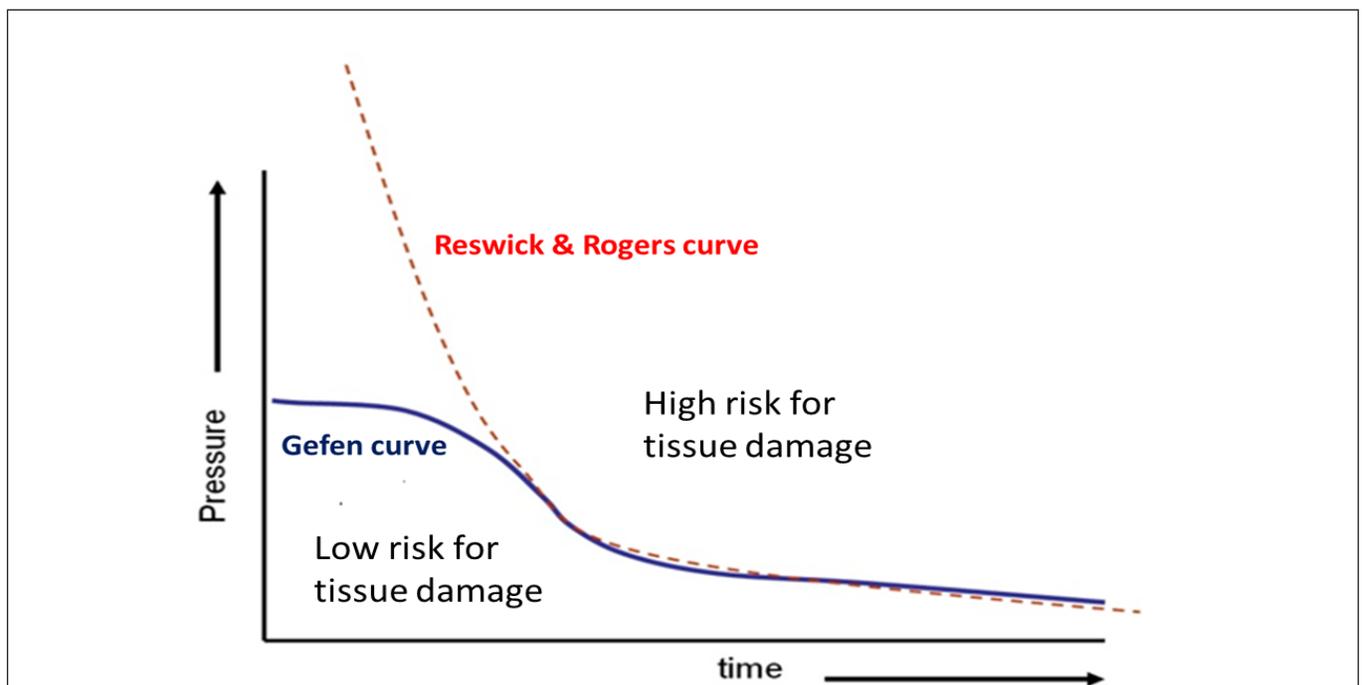


Figure 2.3: Tolerance behavior of soft tissues subjected to sustained mechanical loads

Model formulated by Linder-Ganz et al. (2006)³⁸ and based on experimental data from animal and tissue engineered models, respectively (marked "Gefen curve"), compared to the historic Reswick & Rogers pressure-based damage threshold proposed in the 1970s.

Minimizing compressive (pressure) and shear loads at the interface between the body and the supporting surface or between the body and a medical device are valid clinical interventions for reducing the risk of developing pressure injuries.³⁹ However, pressure measurements alone are not a reliable measure for risk of tissue breakdown, given that similar interface pressure magnitudes will translate to different internal tissue loads in different individuals depending on their internal anatomy (curvature of bony prominences, masses and composition of soft tissues, and soft tissue mechanics properties).⁴⁰ It is therefore not appropriate to judge an individual's risk of tissue damage based on interface pressure alone, or even exposure to interface pressures over time.^{16,41-49}

Elevated shear forces at the interface between the body and a support surface or medical device can exacerbate the damaging deformation caused by normal stresses (pressures) alone.^{4,23,49,50} Internal strains and stresses adjacent to bony prominences are substantially higher than those near the surface, and rise with the level of sharpness of the bony prominence, due to stress concentration effects.⁵¹⁻⁵³ These stress concentrations have the potential to cause damage in deep tissues before the superficial tissue is damaged, and before damage is visible to the naked eye.^{42-44,46-48,54-56}

Friction injuries such as blisters and abrasions may disturb the barrier function of the epidermis. Consequently, support surfaces characterized by high coefficients of friction, or for which the coefficient of friction increases substantially due to wetness (perspiration, exudate and body fluids), represent an extra danger for skin tears, skin breakdown and for infection to occur concurrently with pressure injuries.^{11,23,57} Sustained weight-bearing, by itself or in combination with moisture and wetness, may affect the skin micro-topography (roughness) features, which in turn affects the coefficient of friction of skin with the contacting surface.⁵⁸ These biomechanical interactions are complex and are beyond the scope of this chapter, though it is worth noting that these interactions help explain why moisture associated skin damage is commonly confused with pressure injury.⁵⁹ Moisture associated skin damage may compromise the epidermis barrier function and hence predispose tissue to pressure injury. Detailed review is available of the behavior of skin subjected to frictional forces in a moist environment and the associated risk for skin breakdown.^{10,11,60}

Susceptibility and Tolerance of the Individual

The current understanding is that there are fundamental differences in etiology of superficial pressure injuries compared to pressure injuries in deeper tissue layers.⁶¹ Superficial pressure injuries are primarily caused by high shear at the skin surface, while deeper pressure injuries predominantly result from high pressure in combination with shear at the surface over bony prominences.^{53,62-72} The characteristics of the individual will determine the magnitudes of tissue mechanical loads, the distributions of loads in tissues, the exposure time of tissues to the sustained loads and the tolerance of the affected tissues to the loads. Two physiologically-relevant deformation thresholds exist. One is a lower threshold leading to occlusion of blood vessels resulting in ischemia-induced damage; the other is a higher threshold leading to direct deformation-induced cell damage.^{31,73-78}

Ischemia as a result of sustained deformation of soft tissues will lead to hypoxia, reduced nutrient supply, and impaired removal of metabolic waste products. Deprivation of nutrients and decrease in the pH level towards a more acidic extracellular environment due to accumulation of waste products will eventually lead to cell death and tissue damage.^{22-25,79,80} Bodyweight forces and sustained deformations may alter the stiffness properties of skin even if no injury has been caused, e.g. due to changes in skin hydration.⁵⁸ If an injury has been formed due to the sustained deformations, soft tissue properties may abnormally change. For example, skeletal muscle may exhibit localized 'rigor-mortis' sites of stiffening (i.e., localized pathological contractions due to destruction of muscle fiber membranes) that adds inhomogeneity to the load patterns and promotes intramuscular stress concentrations that endanger adjacent tissues.^{66,81} Sustained deformations may also obstruct lymphatic vessels and reduce the lymphatic flow during and after periods of sustained loading, which further contributes to the biochemical stress at the distorted tissues.⁸² Exposure to ischemic conditions including an acidic extracellular environment (low pH) have shown to slow cell migration in cell culture models, particularly of fibroblasts,⁸³ which may compromise the body's attempts to repair micro damage and hence contribute to an overall accelerated rate of tissue damage in pressure injuries.¹⁹ In elderly patients and those with a central nervous system injury, the capillary density is reduced which typically compromises tissue perfusion, and often, in addition, chronic tissue inflammation is present.^{14,19} Both these factors reduce tissue tolerance as well as repair capacity (e.g. recruitment rate of tissue-repairing cells), in addition to the biomechanical contribution of low-strength atrophied skin and subdermal tissues to the risk of tissue breakdown.¹⁴

The time duration during which cells and tissues can endure ischemia without occurrence of irreversible damage differ for the various tissues that are potentially involved in pressure injuries (i.e., muscle, fat, and skin), with temperature and absorbed moisture for skin. Muscle tissues are more susceptible to damage than skin tissues.^{22,27,84} Skin is much stiffer than muscle and fat and therefore deforms to a lesser degree in most clinically-relevant scenarios, such as bodyweight forces during prolonged sitting or lying in bed. In animal experiments, the first signs of ischemic damage are found in skeletal muscle after two to four hours of sustained deformation.^{22-25,38,75,76,79,80} Muscle deformation at strains greater than 50% will almost immediately (within minutes) lead to tissue damage at a microscopic scale.³⁰ At

this degree of strain there is a strong correlation between magnitude of the strain and the amount of damage to muscle cells/fibers. This direct deformation-inflicted damage to cells is very likely the result of loss of integrity and structural support provided to the cell body by the cytoskeleton. It is probably also related to stretching of the plasma membrane, which increases when the structural support provided to the membrane by the cytoskeleton diminishes, and internal signaling pathways related to these excessive deformations causing apoptotic cell death.^{20,21,28,35,73,85-88} Recent work is applying findings from the rapidly growing field of mechanobiology to pressure injury etiology.

Ongoing Research: Current and Future Perspectives

The rapidly growing field of mechanobiology provides important new insights in how direct deformation results in cell injury and death. First, mechanobiology generates new basic understanding of the mechanisms leading to apoptotic cell death in pressure injury formation and progression as a result of the gradual degradation of cell structures subjected to bodyweight or external forces.⁸⁶ Second, mechanobiological work cited above has a strong translational aspect, in offering a new avenues for interventions at the cell level to eventually increase the tolerance of cellular structures and organelles to sustained mechanical loads. Third, mechanobiology work has recently indicated that stimulating cells mechanically by applying low-level, non-damaging mechanical deformations/strains, accelerates cell migration into damage sites (in laboratory cell cultures).⁸⁹ Given that a pressure injury forms when the rate of cell and tissue death is greater than their rate of regeneration (i.e., through cell proliferation, migration and differentiation), mechanobiology research may reveal optimal stimuli to promote these repair processes, particularly migration of cells at different tissue depths into a micro-damage site at the onset of a pressure injury.⁸⁹

Another recent focus of work is the balance in the interstitial space, where transport of nutrients and waste products that is critical for healthy tissue homeostasis takes place. In a forming pressure injury, that balance is influenced by the interactions of direct deformation damage, inflammatory damage and ischemic damage. Specifically, diffusion of nutrients, clearance of waste products, and hormones that regulate muscle metabolism may be hindered by mechanical loading.^{31,90-94} Recent laboratory (e.g. cell culture and tissue engineering) and computational (e.g. finite modelling) 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.⁹³⁻⁹⁹ The prolonged exposure to large tensional plasma membrane strains may interfere with normal cellular homeostasis, primarily by affecting transport through the plasma membrane that 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.^{86,100,101}

The progression of cell death and tissue necrosis cause gradual 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, e.g., through the development of inflammatory edema and localized rigor mortis in skeletal muscles.^{19,55,56,66,102} Localized inflammatory edema, one of the earliest signs of cell death in pressure injuries, is detectable via measurement of a biophysical marker called the biocapacitance of tissues.^{12,13} Reperfusion that follows a period of prolonged ischemia may increase the degree of tissue damage because it involves release of harmful oxygen free radicals and pro-inflammatory cytokines.¹⁰³⁻¹⁰⁸

An increasing body of evidence suggests that the **microclimate** between skin and the supporting surface plays a role in the development of pressure injuries. Microclimate refers to the temperature, humidity and airflow next to the skin surface. Skin microclimate influences the temperature and hydration of the skin. With an increase in temperature and humidity, the skin becomes weaker (more vulnerable) and less stiff. Excessively dry skin is also undesirable as dry skin becomes more brittle and liable to cracks (fissures). The skin microclimate affects the skin structure and function and the response to mechanical loading, and is relevant for all pressure injuries, not just the superficial ones. Microclimate conditions at the skin affect, for example, the load transfer from the skin to deep tissue and hence the risk for a deep tissue injury.

The characteristics of an optimal microclimate are still a matter of debate and ongoing research.¹⁰⁹ Wetness of the interface plays an important role in the mechanical interactions between the skin and a support surface, a medical device or clothing. Materials with a higher wettability generally show a larger increase in friction coefficient when exposed to warm and moist conditions.¹¹⁰ The temperature of the body influences the production of perspiration. Given that the evaporation of perspiration from the body depends on the local and ambient humidity as well as interactions with contacting materials (e.g., clothing, bedsheets, wheelchair cushion cover and wound dressings),¹¹¹ microclimate eventually impacts:

- The frictional properties of skin
- The magnitudes of frictional forces acting on the body
- Tissue deformations resulting from any frictional sliding movement and shear force between the skin and a support surface or a medical device.

This is discussed by Gefen and colleagues.^{10,11,37,60} Overall, there are strong connections between microclimate and friction, and hence surface and internal tissue loads, and the exposure of living cells to these mechanical loads.

The damage cascade in pressure injuries, illustrated in *Figure 2.4*, includes the sequential damage associated with direct deformation, inflammatory response, and ischemia.¹⁹ The additive nature of these damages highlights the importance of minimization of exposure to sustained tissue deformations and early detection of cell and tissue damage for effective pressure injury prevention. The risks associated with prolonged surgery (sustained soft tissue deformations in the immobile individual's body), and certain medications that affect the functions of the inflammatory system (e.g., steroids and chemotherapy) or vascular system (e.g., vasopressors) should also be interpreted in light of the above-described damage cascade and its three main constituents; namely, deformation, inflammation and ischemia. Examples of current pressure injury prevention technologies can be classified into those that minimize exposure to sustained tissue deformations and those that target the biomarkers of early cell death to prevent progression of the damage.

The translation of basic science findings to medical technologies is based on understanding the etiology of pressure injuries. This then facilitates the development of better devices and protocols for pressure injury prevention. The review of such relevant medical technologies and devices is outside the scope of this chapter. The connections between pressure injury etiology and preventive strategies is explored in other chapters of this guideline.

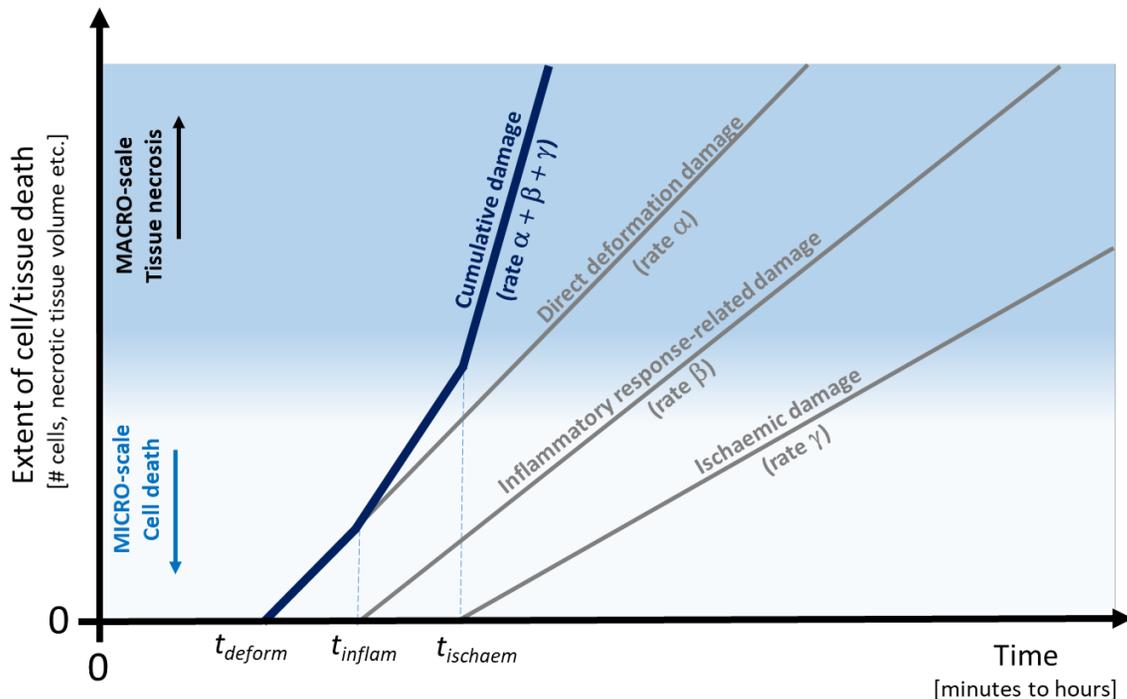


Figure 2.4: Contributors to cell damage and tissue necrosis in pressure injuries¹⁹

¹⁹ There are three major contributors to cell damage and tissue necrosis that have been identified in pressure injuries in cell culture models, animal models, tissue engineering work and computational modeling:

- Direct deformation, inflammatory response, and ischemia. Direct deformation is the initial factor that begins to inflict cell and tissue damage at time point t_{deform} and progresses at a rate α .
- Inflammatory response-related damage occurs second at time point t_{inflam} and develops at a rate β .
- Ischemic damage is the next to appear at time point $t_{ischaem}$ and evolves at a rate γ .

The combined contributions of these three contributors to damage at sequential time points explains the non-linear nature of the cumulative cell and tissue damage in pressure injuries. This damage will accelerate from the micro-scale to the macro-scale and eventually exacerbate at a rate of $\alpha+\beta+\gamma$. Reproduced with permission.

There are also complex interactions (not shown here) which may occur between the above three damage components. For example ischemia may be produced by vascular deformations as the direct cause, but ischemia is then further influenced by the evolving inflammatory process as endothelial cells (which make the walls of capillaries) are developing cell-cell gaps, in response to inflammatory signaling.

References

1. Coyer FM, Stotts NA, Blackman VS. A prospective window into medical device-related pressure ulcers in intensive care. *Int Wound J*, 2014; 11(6): 656-64.
2. Groth KE. Klinische Beobachtungen und experimentelle studien ber die Entstehung des Dekubitus. *Acta Chirurgica Scandinavica*, 1942; 87(supp 76).
3. Kosiak M. Etiology and pathology of ischemic ulcers. *Arch Phys Med Rehabil*, 1959; 40(2): 62-69.
4. Reichel S. Shearing force as a factor in decubitus ulcers in paraplegics. *Journal of the American Medical Association*, 1958; 166(7): 762-763.
5. Nightingale F, *Notes on Nursing: What It Is, And What It Is Not*. 1859, London: Harrison and Son.
6. Kenedi JM, ed. *Bedsore Biomechanics*. 1976, University Park Press.
7. Gefen A. Time to challenge the continued use of the term 'pressure ulcers'? *Br J Nurs*, 2017; 26: S20-S22.
8. Coleman S, Nixon J, Keen J, Wilson L, McGinnis E, Dealey C, Stubbs N, Farrin A, Dowding D, Schols JMGA, Cuddigan J, Berlowitz D, Jude E, Vowden P, Schoonhoven L, Bader DL, Gefen A, Oomens CWJ, Nelson EA. A new pressure ulcer conceptual framework. *J Adv Nurs*, 2014; 70(10): 2222-2234.
9. Oomens CWJ, A Mixture Approach to the Mechanics of Skin and Subcutis - a Contribution to Pressure Sore Research, in Pesis 1985, University of Twente: Enschede, The Netherlands.
10. Shaked E, Gefen A. Modeling the effects of moisture-related skin-support friction on the risk for superficial pressure ulcers during patient repositioning in bed. *Front Bioeng Biotechnol*, 2013; 14(1): 9.
11. Schwartz D, Magen YK, Levy A, Gefen A. Effects of humidity on skin friction against medical textiles as related to prevention of pressure injuries. *Int Wound J*, 2018; 15(6): 866-874.
12. Gefen A. The sub-epidermal moisture scanner: The principles of pressure injury prevention using novel early detection technology. *Wounds Int*, 2018; 9(3): 10-15.
13. Gefen A, Gershon S. An Observational, Prospective Cohort Pilot Study to Compare the Use of Subepidermal Moisture Measurements Versus Ultrasound and Visual Skin Assessments for Early Detection of Pressure Injury. *Ostomy Wound Manage*, 2018; 64(9): 12-27.
14. Gefen A. Tissue changes in patients following spinal cord injury and implications for wheelchair cushions and tissue loading: a literature review. *Ostomy Wound Manage*, 2014; 60(2): 34-45.
15. Gefen A. Why is the heel particularly vulnerable to pressure ulcers? *Br J Nurs*, 2017; 26(Sup20): S62-S74.
16. Gefen A, Levine J. The false premise in measuring body-support interface pressures for preventing serious pressure ulcers. *J Med Eng Technol*, 2007; 31(5): 375-80.
17. Lustig M, Levy A, Kopplin K, Ovadia-Blechman Z, Gefen A. Beware of the toilet: The risk for a deep tissue injury during toilet sitting. *J Tissue Viability*, 2018; 27(1): 23-31.
18. Levy A, Kopplin K, Gefen A. Device-related pressure ulcers from a biomechanical perspective. *J Tissue Viability*, 2017; 26(1): 57-68.
19. Gefen A. The future of pressure ulcer prevention is here: Detecting and targeting inflammation early. *EWMA Journal* 2018 19(2).
20. Breuls RG, Bouten CV, Oomens CW, Bader DL, Baaijens FP. Compression induced cell damage in engineered muscle tissue: an in vitro model to study pressure ulcer aetiology. *Annals of Biomedical Engineering*, 2003a; 31(11): 1357-1364.
21. Breuls RG, Bouten CV, Oomens CW, Bader DL, Baaijens FP. A theoretical analysis of damage evolution in skeletal muscle tissue with reference to pressure ulcer development. *Journal of Biomechanical Engineering*, 2003b; 125(6): 902-909.
22. Daniel RK, Priest DL, Wheatley DC. Etiologic factors in pressure sores: an experimental model. *Archives of Physical Medicine and Rehabilitation*, 1981; 62(10): 492-498.
23. Dinsdale SM. Decubitus ulcers: role of pressure and friction in causation. *Archives of Physical Medicine and Rehabilitation*, 1974; 55(4): 147-152.
24. Gawlitta D, Li W, Oomens CW, Baaijens FP, Bader DL, Bouten CV. The relative contributions of compression and hypoxia to development of muscle tissue damage: an in vitro study. *Annals of Biomedical Engineering*, 2007; 35(2): 273-284.
25. Gawlitta D, Oomens CW, Bader DL, Baaijens FP, Bouten CV. Temporal differences in the influence of ischemic factors and deformation on the metabolism of engineered skeletal muscle. *Journal of Applied Physiology*, 2007; 103(2): 464-473.
26. Linder-Ganz E, Engelberg S, Scheinowitz M, Gefen A. Pressure-time cell death threshold for albino rat skeletal muscles as related to pressure sore biomechanics. *Journal of Biomechanics*, 2006; 39(14): 2725-2732.
27. Salcido R, Donofrio JC, Fisher SB, LeGrand EK, Dickey K, Carney JM, Schosser R, Liang R. Histopathology of pressure ulcers as a result of sequential computer-controlled pressure sessions in a fuzzy rat model. *Advances in Wound Care*, 1994; 7(5): 23-24, 26, 28.
28. Stekelenburg A, Oomens CW, Strijkers GJ, Nicolay K, Bader DL. Compression-induced deep tissue injury examined with magnetic resonance imaging and histology. *J Appl Physiol*, 2006; 100(6): 1946-1954.
29. Reswick JB, Rogers JE, *Experience at Rancho Los Amigos Hospital with devices and techniques that prevent pressure sores.*, in *Bedsore Biomechanics* R.M. Kenedi and J.M. Cowden, Editors. 1976, The Macmillan Press. p. 301-310.
30. Gefen A, van Nierop B, Bader DL, Oomens CW. Strain-time cell-death threshold for skeletal muscle in a tissue-engineered model system for deep tissue injury. *J Biomech*, 2008; 41(9): 2003-12.
31. Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. *Ostomy Wound Manage*, 2008; 54(10): 26-35.
32. Friedman R, Haimy A, Epstein Y, Gefen A. Evaluation of helmet and goggle designs by modeling non-penetrating projectile impacts. *Comput Methods Biomech Biomed Engin*, 2009; 22(3): 229-242.

33. Gefen A. Reswick and Rogers pressure-time curve for pressure ulcer risk. Part 1. *Nurs Stand*, 2009; 23(45): 64, 66, 68 passim.
34. Gefen A. Reswick and Rogers pressure-time curve for pressure ulcer risk. Part 2. *Nurs Stand*, 2009; 23(46): 40-4.
35. Stekelenburg A, Strijkers GJ, Parusel H, Bader DL, Nicolay K, Oomens CW. Role of ischemia and deformation in the onset of compression-induced deep tissue injury: MRI-based studies in a rat model. *J Appl Physiol*, 2007; 102(5): 2002-2011.
36. Lachenbruch C, Tzen Y-T, Brienza DM, Karg PE, Lachenbruch PA. The relative contributions of Interface Pressure, shear, stress and temperature on tissue ischemia: a cross-sectional pilot study. *Ostomy Wound Management*, 2013; 59(3): 25-34.
37. Zeevi T, Levy A, Brauner N, Gefen A. Effects of ambient conditions on the risk of pressure injuries in bedridden patients-multi-physics modelling of microclimate. *Int Wound J*, 2018; 15(3): 402-416.
38. Linder-Ganz E, Engelberg S, Scheinowitz M, Gefen A. Pressure-time cell death threshold for albino rat skeletal muscles as related to pressure sore biomechanics. *J Biomech*, 2006; 39(14): 2725-32.
39. Brienza DM, Karg PK, Geyer MJ, Kelsey S, Trefler E. The relationship between pressure ulcer incidence and buttock-seat cushion interface pressure in at-risk elderly wheelchair users. *Archives of Physical Medicine and Rehabilitation* 2001; 82(4): 529-533.
40. Gefen A. The Compression Intensity Index: a practical anatomical estimate of the biomechanical risk for a deep tissue injury. *Technol Health Care*, 2008; 16(2): 141-149.
41. Breuls RG, Sengers BG, Oomens CW, Bouten CV, Baaijens FP. Predicting local cell deformations in engineered tissue constructs: a multilevel finite element approach. *Journal of Biomechanical Engineering*, 2002; 124(2): 198-207.
42. Chow WW, Odell EI. Deformations and stresses in soft body tissues of a sitting person. *Journal of Biomechanical Engineering*, 1978; 100: 79-87.
43. Linder-Ganz E, Shabshin N, Itzchak Y, Gefen A. Assessment of mechanical conditions in sub-dermal tissues during sitting: a combined experimental-MRI and finite element approach. *Journal of Biomechanics*, 2007; 40(7): 1443-1454.
44. Mak AF, Liu GH, Lee SY. Biomechanical assessment of below-knee residual limb tissue. *Journal of Rehabilitation Research & Development*, 1994; 31(3): 188-198.
45. Oomens CW, van Campen DH, Grootenboer HJ. A mixture approach to the mechanics of skin. *Journal of Biomechanics*, 1987; 20(9): 877-885.
46. Todd BA, Thacker JG. Three-dimensional computer model of the human buttocks, in vivo. *Journal of Rehabilitation Research & Development*, 1994; 31(2): 111-119.
47. Zhang JD, Mak AF, Huang LD. A large deformation biomechanical model for pressure ulcers. *Journal of Biomechanical Engineering*, 1997; 119(4): 406-408.
48. Dabnichki PA, Crocombe ZD, Hughes SC. Deformation and stress analysis of supported buttock contact. *Proceedings of the Institution of Mechanical Engineers. Journal of Engineering in Medicine*, 1994; 208: 9-17.
49. Linder-Ganz E, Gefen A. The effects of pressure and shear on capillary closure in the microstructure of skeletal muscles. *Annals of Biomedical Engineering*, 2007; 35(12): 2095-2107.
50. Knight SL, Taylor RP, Polliack AA, Bader DL. Establishing predictive indicators for the status of loaded soft tissues. *Journal of Applied Physiology* 2001; 90(6): 2231-2237.
51. Akins JS, Valley JJ, Karg PE, Kopplin K, Gefen A, Poojary-Mazzotta P, Brienza DM. Feasibility of freehand ultrasound to measure anatomical features associated with deep tissue injury risk. *Med Eng Phys*, 2016; 38(9): 839-844.
52. Brienza D, Valley J, Karg P, Akins J, Gefen A. An MRI investigation of the effects of user anatomy and wheelchair cushion type on tissue deformation. *J Tissue Viability*, 2018; 27(1): 42-53.
53. Linder-Ganz E, Gefen A. Stress analyses coupled with damage laws to determine biomechanical risk factors for deep tissue injury during sitting. *J Biomech Eng*, 2009; 131(1): 011003.
54. Oomens CW, Bressers OF, Bosboom EM, Bouten CV, Blader DL. Can loaded interface characteristics influence strain distributions in muscle adjacent to bony prominences? *Computer Methods in Biomechanics and Biomedical Engineering*, 2003; 6(3): 171-180.
55. Linder-Ganz E, Gefen A. Mechanical compression-induced pressure sores in rat hindlimb: muscle stiffness, histology, and computational models. *Journal of Applied Physiology* 2004; 96(6): 2034-2049.
56. Gefen A, Gefen N, Linder-Ganz E, Margulies SS. In vivo muscle stiffening under bone compression promotes deep pressure sores. *Journal of Biomechanical Engineering*, 2005; 127(3): 512-524.
57. Dinsdale SM. Decubitus ulcers in swine: light and electron microscopy study of pathogenesis. *Archives of Physical Medicine and Rehabilitation* 1973; 54(2): 51-56.
58. Dobos G, Gefen A, Blume-Peytavi U, Kottner J. Weight-bearing-induced changes in the microtopography and structural stiffness of human skin in vivo following immobility periods. *Wound Repair Regen*, 2015; 23(1): 37-43.
59. Vilhena L, Ramalho A. Friction of human skin against different fabrics for medical use. *Lubricants*, 2016; 4(1): 6.
60. Sopher R, Gefen A. Effects of skin wrinkles, age and wetness on mechanical loads in the stratum corneum as related to skin lesions. *Med Biol Eng Comput*, 2011; 49(1): 97-105.
61. Kottner J, Gefen A, Lahmann N. Weight and pressure ulcer occurrence: a secondary data analysis. *Int J Nurs Stud*, 2011; 48(11): 1339-48.
62. Agam L, Gefen A. Pressure ulcers and deep tissue injury: a bioengineering perspective. *J Wound Care*, 2007; 16(8): 336-42.
63. Gefen A. Risk factors for a pressure-related deep tissue injury: a theoretical model. *Med Biol Eng Comput*, 2007; 45(6): 563-73.
64. Gefen A. The biomechanics of sitting-acquired pressure ulcers in patients with spinal cord injury or lesions. *Int Wound J*, 2007; 4(3): 222-31.
65. Gefen A. Bioengineering models of deep tissue injury. *Adv Skin Wound Care*, 2008; 21(1): 30-6.
66. Gefen A. Deep tissue injury from a bioengineering point of view. *Ostomy Wound Manage*, 2009; 55(4): 26-36.
67. Gefen A, Farid KJ, Shaywitz I. A review of deep tissue injury development, detection, and prevention: shear savvy. *Ostomy Wound Manage*, 2013; 59(2): 26-35.

68. Linder-Ganz E, Yarnitzky G, Yizhar Z, Siev-Ner I, Gefen A. Real-time finite element monitoring of sub-dermal tissue stresses in individuals with spinal cord injury: toward prevention of pressure ulcers. *Ann Biomed Eng*, 2009; 37(2): 387-400.
69. Peko Cohen L, Levy A, Shabshin N, Neeman Z, Amit A. Sacral soft tissue deformations when using a prophylactic multilayer dressing and positioning system *J Wound Ostomy Continence Nurs*, 2018; 45(5): 432-437.
70. Shabshin N, Ougortsin V, Zoizner G, Gefen A. Evaluation of the effect of trunk tilt on compressive soft tissue deformations under the ischial tuberosities using weight-bearing MRI. *Clin Biomech*, 2010; 25(5): 402-8.
71. Shoham N, Gefen A. Deformations, mechanical strains and stresses across the different hierarchical scales in weight-bearing soft tissues. *J Tissue Viability*, 2012; 21(2): 39-46.
72. Sopher R, Nixon J, Gorecki C, Gefen A. Effects of intramuscular fat infiltration, scarring, and spasticity on the risk for sitting-acquired deep tissue injury in spinal cord injury patients. *J Biomech Eng*, 2011; 133(2): 021011.
73. Ceelen KK, Stekelenburg A, Loerakker S, Strijkers GJ, Bader DL, Nicolay K, Baaijens FP, Oomens CW. Compression-induced damage and internal tissue strains are related. *Journal of Biomechanics*, 2008; 41(16): 3399-3404.
74. Gefen A, Cornelissen LH, Gawlitta D, Bader DL, Oomens CW. The free diffusion of macromolecules in tissue-engineered skeletal muscle subjected to large compression strains. *Journal of Biomechanics*, 2008; 41(4): 845-853.
75. Loerakker S, Manders E, Strijkers GJ, Nicolay K, Baaijens FPT, Bader DL, Oomens CWJ. The effects of deformation, ischemia, and reperfusion on the development of muscle damage during prolonged loading. *Journal of Applied Physiology* 2011; 111(4): 1168-1177.
76. Loerakker S, Oomens CWJ, Manders E, T. S, D.L. B, F.P. B, Nicolay K, Strijkers GJ. Ischemia-Reperfusion Injury in Rat Skeletal Muscle Assessed with T-2-Weighted and Dynamic Contrast-Enhanced MRI. *Magnetic Resonance in Medicine*, 2011; 66(2): 528-537.
77. Loerakker S, Stekelenburg A, Strijkers GJ, Rijpkema JJ, Baaijens FP, Bader DL, Nicolay K, Oomens CW. Temporal effects of mechanical loading on deformation-induced damage in skeletal muscle tissue. *Annals of Biomedical Engineering*, 2010; 38(8): 2577-2587.
78. Oomens CWJ. A multi-scale approach to study the aetiology of pressure ulcers. *Wound Repair and Regeneration*, 2010; 18(4): A74.
79. Kosiak M. Etiology of decubitus ulcers. *Archives of Physical Medicine and Rehabilitation* 1961; 42: 19-29.
80. Bader DL, Barnhill RL, Ryan TJ. Effect of externally applied skin surface forces on tissue vasculature. *Archives of Physical Medicine and Rehabilitation* 1986; 67(11): 807-811.
81. Gefen A, Gefen N, Linder-Ganz E, Margulies SS. In vivo muscle stiffening under bone compression promotes deep pressure sores. *J Biomech Eng*, 2005; 127: 512-524.
82. Gray RJ, Worsley PR, Voegeli D, Bader DL. Monitoring contractile dermal lymphatic activity following uniaxial mechanical loading. *Med Eng Phys*, 2016; 38(9): 895-903.
83. Topman G, Lin FH, Gefen A. The influence of ischemic factors on the migration rates of cell types involved in cutaneous and subcutaneous pressure ulcers. *Ann Biomed Eng*, 2012; 40: 1929-1939.
84. Nola GT, Vistnes LM. Differential response of skin and muscle in the experimental production of pressure sores. *Plastic and Reconstructive Surgery*, 1980; 66(5): 728-733.
85. Stekelenburg A, Gawlitta D, Bader DL, Oomens CW. Deep tissue injury: how deep is our understanding? *Archives of Physical Medicine and Rehabilitation*, 2008; 89(7): 1410-1413.
86. Gefen A, Weihs D. Cytoskeleton and plasma-membrane damage resulting from exposure to sustained deformations: A review of the mechanobiology of chronic wounds. *Med Eng Phys*, 2016; 38(9): 828-833.
87. Slomka N, Gefen A. Relationship between strain levels and permeability of the plasma membrane in statically stretched myoblasts. *Ann Biomed Eng*, 2012; 40(3): 606-618.
88. Leopold E, Gefen A. Changes in permeability of the plasma membrane of myoblasts to fluorescent dyes with different molecular masses under sustained uniaxial stretching. *Med Eng Phys*, 2013; 35(5): 601-7.
89. Toume S, Gefen A, Weihs D. Low-level stretching accelerates cell migration into a gap. *Int Wound J*, 2017; 14(4): 698-703.
90. Krouskop TA. A synthesis of the factors that contribute to pressure sore formation. *Medical Hypotheses*, 1983; 11(2): 255-267.
91. Krouskop TA, Reddy NP, Spencer WA, Secor JW. Mechanisms of decubitus ulcer formation--an hypothesis. *Medical Hypotheses*, 1978; 4(1): 37-39.
92. Gefen A, Cornelissen LH, Gawlitta D, Bader DL, Oomens CW. The free diffusion of macromolecules in tissue-engineered skeletal muscle subjected to large compression strains. *J Biomech*, 2008; 41(4): 845-853.
93. Ruschkewitz Y, Gefen A. Cell-level temperature distributions in skeletal muscle post spinal cord injury as related to deep tissue injury. *Med Biol Eng Comput*, 2010; 48(2): 113-22.
94. Ruschkewitz Y, Gefen A. Cellular-scale transport in deformed skeletal muscle following spinal cord injury. *Comput Methods Biomech Biomed Engin*, 2011; 14(5): 411-24.
95. Leopold E, Gefen A. Stretching affects intracellular oxygen levels: three-dimensional multiphysics studies. *J Biomech Eng*, 2012; 134(6): 064501.
96. Leopold E, Gefen A. A simple stochastic model to explain the sigmoid nature of the strain-time cellular tolerance curve. *J Tissue Viability*, 2012 21(1): 27-36.
97. Leopold E, Sopher R, Gefen A. The effect of compressive deformations on the rate of build-up of oxygen in isolated skeletal muscle cells. *Med Eng Phys*, 2011; 33(9): 1072-1078.
98. Slomka N, Gefen A. Confocal microscopy-based three-dimensional cell-specific modeling for large deformation analyses in cellular mechanics. *J Biomech Eng*, 2010 43(9): 1806-16.
99. Slomka N, Or-Tzadikario S, Sassun D, Gefen A. Membrane-stretch-induced cell death in deep tissue injury: Computer model studies. *Cell Mol Bioeng*, 2009; 2: 118-132.

100. Slomka N, Gefen A. Relationship between strain levels and permeability of the plasma membrane in statically stretched myoblasts. *Annals of Biomedical Engineering*, 2012; 40(3): 606-618.
101. Leopold E, Gefen A. Changes in permeability of the plasma membrane of myoblasts to fluorescent dyes with different molecular masses under sustained uniaxial stretching. *Medical Engineering and Physics*, 2013; 35(5)(5): 601-7.
102. Edsberg LE, Cutway R, Anain S, Natiella JR. Microstructural and mechanical characterization of human tissue at and adjacent to pressure ulcers. *Journal of Rehabilitation Research & Development*, 2000; 37(4): 463-471.
103. Houwing R, Overgoor M, Kon M, Jansen G, van Asbeck BS, Haalboom JR. Pressure-induced skin lesions in pigs: reperfusion injury and the effects of vitamin. *Journal of Wound Care*, 2000; 9(1): 36-40.
104. Ikebe K, Kato T, Yamaga M, Hirose J, Tsuchida T, Takagi K. Increased ischemia-reperfusion blood flow impairs the skeletal muscle contractile function. *Journal of Surgical Research*, 2001; 99(1): 1-6.
105. Peirce SM, Skalak TC, Rodeheaver GT. Ischemia-reperfusion injury in chronic pressure ulcer formation: a skin model in the rat. *Wound Repair and Regeneration*, 2000; 8(1): 68-76.
106. Reid RR, Sull AC, Mogford JE, Roy N, Mustoe TA. A novel murine model of cyclical cutaneous ischemia-reperfusion injury. *Journal of Surgical Research*, 2004; 116(1): 172-180.
107. Tsuji S, Ichioka S, Sekiya N, Nakatsuka T. Analysis of ischemia-reperfusion injury in a microcirculatory model of pressure ulcers. *Wound Repair and Regeneration*, 2005; 13(2): 209-215.
108. Unal S, Ozmen S, Demir Y, Yavuzer R, Latifoglu O, Atabay K, Oguz M. The effect of gradually increased blood flow on ischemia-reperfusion injury. *Annals of Plastic Surgery*, 2001; 47(4): 412-416.
109. Kottner J, Black J, Call E, Gefen A, Santamaria N. Microclimate: A critical review in the context of pressure ulcer prevention. *Clin Biomech*, 2018; 59: 62-70.
110. Klaassen M, de Vries EG, Masen MA. The static friction response of non-glabrous skin as a function of surface energy and environmental conditions. *Biotribology*, 2017; 11: 124-131.
111. Gefen A. How do microclimate factors affect the risk for superficial pressure ulcers: a mathematical modeling study. *J Tissue Viability*, 2011; 20(3): 81-8.

POPULATIONS WITH SPECIFIC PRESSURE INJURY RELATED NEEDS

Introduction

While most of the recommendations included throughout this guideline are relevant to all individuals with or at risk of pressure injuries, it is acknowledged that some individuals have specific pressure injury-related needs, due to their clinical condition, age or care setting. The previous edition of this guideline included chapters specific to a range of special populations. However, in most cases specific pressure injury-related needs for special populations are in addition to, rather than a replacement for, more general evidence-based care practices. Evidence arising from studies in one population are frequently relevant to other groups at risk or have pressure injuries, and many care recommendations are extrapolated from evidence in a range of special populations. In this edition of the guideline, recommendations specific to special populations have been incorporated into the relevant guideline chapters. Where specific and unique evidence exists for prevention and treatment of pressure injuries in a special population, recommendations specific to that population are presented in the relevant chapter. The implementation considerations in this guideline also note any variation to implementing the recommendation when caring for special populations. The special populations that have been specifically considered include:

- Critically ill individuals
- Individuals with spinal cord injury
- Individuals receiving palliative care
- Individuals with obesity
- Neonates and children
- Individuals in community, aged care and rehabilitation settings
- Individuals in the operating room
- Individuals in transit.

This chapter will provide a background on issues of significance to the special populations that have been considered in the development of this guideline.

Critically Ill Individuals

Critically ill people, cared for in intensive care units (ICUs), are a unique subset of hospitalized individuals and represent the sickest patients in the health care system. Critical illness can render an individual physiologically unstable requiring treatment with invasive modalities such as mechanical ventilation, vasopressor agents, extracorporeal membrane oxygenation, intraaortic balloon pump, left ventricular assist devices, or continuous renal replacement therapy.¹ Clinical parameters such as hypotension, tachypnea, tachycardia or bradycardia, hypoxemia, hypo or hyperthermia, prolonged capillary refill time, oliguria, and altered mental status are all indicators of physiologic and hemodynamic instability,² and may be the result of conditions such as acute blood loss, shock states or decreased systemic vascular resistance from sepsis. The development of a pressure injury presents an additional comorbid threat for an already severely compromised person.

Pressure injury rates in the critical care population are reported as one of the highest among hospitalized individuals.³⁻⁵ A comprehensive review of pressure injury prevalence studies up to 2013 reported a range of 13.1%⁶ to 45.5%.⁷ More recently, a state-wide prevalence survey (18 facilities) conducted in Australian Level I to III ICUs reported prevalence of Category/State II or greater pressure injuries at 11%, which was 3.8 times (relative risk [RR] 95% confidence interval [CI] 2.7 to 5.4 higher than for non-intensive care units in the same facilities.⁸ Another recent survey conducted over 8.5 years in an Australian hospital (n = 5,280) reported a 10-fold higher incidence of facility-acquired pressure injuries in ICU settings compared to general hospital wards.⁹

The higher rate of pressure injuries in critical care settings is attributed to the high level of disease/illness burden; hemodynamic instability requiring the use of vasoactive medications; poor tissue perfusion and oxygenation; coagulopathy and repeated confrontations with multiple, concomitant risk factors for pressure injury development experienced by this population.^{10,11} Additionally, implementation of some preventive measures may be limited or even contraindicated. For critically ill individuals experiencing multi-organ system failure, impaired tissue oxygenation and perfusion can contribute to 'skin failure', thus the development of a pressure related injury may be an unavoidable outcome and skin damage may occur independent of external pressure on non-loading surfaces.

Critically ill individuals at-risk for pressure injuries should be provided with preventive measures described throughout this guideline. However, due to the additional (and often non-modifiable) risk factors faced by this special population, risk-based interventions often need to be intensified, and interventions specific to the unique needs of critically ill individuals must be provided. In particular, the *Repositioning and Early Mobilization* chapter outlines recommendations that are specific to critically ill individuals.

Several preventive interventions have been studied in critical care populations, and this research underpins general recommendations throughout the guideline. Notably, current research on the clinical efficacy and cost effectiveness of prophylactic sacral and heel dressings in critically ill individuals is presented in the *Preventive Skin Care* chapter. The chapter on *Device Related Pressure Injuries* includes discussion of current research on prevention of pressure injuries under devices commonly found in critically ill populations (e.g., endotracheal tubes, tracheostomy tubes). While the recommendations are considered relevant to all people with or at risk of pressure injuries, they are particularly relevant to critical care settings.

Individuals with Spinal Cord Injury

Individuals with spinal cord injury (SCI) are at an increased risk of pressure injuries due to immobility, decreased sensation and altered pathophysiology that predisposes the skin to breakdown.¹² Duration of time since experiencing the SCI also influences pressure injury risk, with pressure injuries significantly more likely to occur within twelve months of the SCI than in individuals with long-standing SCI.¹³ Lifestyle factors including smoking, alcohol and medication use,¹⁴ level of physical activity, compliance with a pressure injury prevention plan,¹⁵ knowledge of pressure injury prevention strategies,^{15,16} and access to appropriate support surfaces¹⁵ have all been associated with pressure injury incidence and healing outcomes in individuals with SCI.

During the stage of hospitalization, prevalence rates vary. In a large US database study, prevalence of hospital-acquired Category/Stage III or IV pressure injuries was reported as 1.48% (95% CI 1.14% to 1.92%).¹⁷ In a seven-year study conducted in six trauma centers in the US (n = 411), pressure injuries were reported as a complication for 2.6% of individuals with SCI.¹⁸ In a point prevalence study that also relied on database entries, prevalence of pressure injuries in an SCI-specialized facility in the US was 12%.¹⁹ These prevalence rates are all location specific, and interpretation is complicated by the population selection and various different methods for identifying pressure injuries and calculating prevalence rates. After discharge, prevalence of pressure injuries in the community is harder to ascertain. In a cohort study set in Thailand, 42% of wheelchair-bound participants (n = 50) had experienced a pressure injury within six months of their discharge from a rehabilitation center.²⁰ A second study conducted in individuals with SCI in Thailand (n = 129) reported a prevalence rate of 26.4% based on a self-reported survey.²¹ However, the wide variety of care models and access to resources reduces the generalizability of prevalence rates.

The risk of pressure injuries affects individuals with SCI at every stage of their care. Ploumis et al. (2011)¹⁹ found that receiving acute care in an SCI-specific facility at the time of spinal injury significantly decreased the risk of having a pressure injury by the time the individual reached the rehabilitative stage of their care (12% versus 34% for individuals cared for in a non-SCI acute care facility, p < 0.001). However, Richard-Denis et al. (2016)²² did not observe the type of care facility to have an impact on pressure injury risk. In their five year study of individuals in rehabilitative care (n = 123), the odds ratio [OR] of experiencing a pressure injury in a specialized facility compared to a non-specialized facility was 0.059 (95% CI 0.01 to 0.27).²²

In the acute stages, management strategies for suspected SCI increase risks of many adverse outcomes, one of which is skin breakdown associated with pressure and shear. Recommendations on back board use for reducing spinal mobility in the acute care phase are included in the guideline chapter on *Support Surfaces*. Recommendations on the use of cervical collars for stabilizing the spine are included in the guideline chapter on *Device Related Pressure Injuries*.

During the recovery and rehabilitation stages, having a shorter length of stay in acute care reduces the risk of developing a pressure injury,^{13,19,23,24} if an individual does develop a pressure injury, the length of stay in acute care becomes significantly longer, lengthening the recovery period.^{18,23} A large observation study of individuals at least one year post traumatic SCI (n = 1871) demonstrated that individuals with more severe SCI have a trend towards worse pressure injury-related outcomes than those with less severe SCI.¹⁴ More severe immobility and limitations in performing activities of daily living (ADLs) has also been associated with higher rates of pressure injury¹⁶ and pressure injury infection²⁵ in other studies.

Unlike many other individuals for whom pressure injuries are no longer a risk following their discharge from a health care facility, individuals with SCI face a life-long risk that impacts on their daily living. In a longitudinal study in which in-depth interviewing was conducted with 30 individuals with SCI in the US, Jackson et al. (2010)²⁶ identified that the risk of pressure injuries was perceived as a perpetual danger, and individuals often faced tension between living a full life and avoiding situations that put them at higher risk. Being motivated, initiating positive lifestyle changes, identifying goals and understanding pressure injury risk were all associated with a more positive pressure injury risk

status for individuals with SCI in both Jackson et al.'s study²⁶ and others.²⁷⁻²⁹ However, individuals with SCI frequently report barriers to accessing care, services, resources and support.²⁶⁻²⁸

Mathew (2013)¹⁶ found that 65% of all pressure injuries could be attributed to poor pressure relief practices in a cohort of individuals (n = 108) with SCI receiving rehabilitation. Promoting the use of appropriate equipment, particularly wheelchairs and pressure redistribution cushions, and use of regular and effective repositioning are basic requirements for individuals living with SCI. The guideline chapters *Support Surfaces* and *Repositioning and Early Mobilization* provide recommendations of particular relevance to individuals with SCI, including recommendations on equipment selection, repositioning in seated positions and pressure relief maneuvers. Addressing education and lifestyle needs are also an ongoing requirement in order to promote self-efficacy in pressure injury prevention. The guideline chapter on *Quality of Life, Self Care and Education* includes recommendations that are pertinent to individuals with SCI. The recommendations included in other sections of the guideline are also generally appropriate to individuals with SCI.

Up to 95% of individuals with SCI will experience a pressure injury at some stage during their life.¹² The experience of having a pressure injury is described by individuals with SCI (n = 19) in a qualitative exploration conducted by Dunn et al. (2009).²⁹ Participants described underestimating the danger of pressure injuries and a lack of knowledge regarding prevention and management. They described avoiding social discomfort as a contributing factor to pressure injury development, as well as inadequate medical assistance, and the competing demand of managing comorbidities.²⁹ In a Canadian study,³⁰ the high financial cost associated with management of community-based individuals with SCI who experience a full thickness pressure injury was reported. Monthly costs of approximately \$4,700 (Canadian dollars in 2010) were reported, of which 59% were attributed to the cost of health professional care and hospital admissions.³⁰ Making care decisions to promote healing, particularly addressing the difficulty of healing ischial and sacral pressure injuries, is discussed in the guideline chapter on *Repositioning and Early Mobilization*.

Individuals Receiving Palliative Care

Both palliative care and hospice care offer a multidisciplinary approach providing comfort and support to individuals with a serious or chronic illness. The World Health Organization (WHO) defines palliative care as “an approach that improves the quality of life of patients and their families facing the problem associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual.”³¹ Palliative care can begin at diagnosis, and at the same time as treatment for serious illness. Hospice care offers supportive care to individuals in the final phase of a terminal illness and focuses on comfort and quality of life, rather than cure. The goal is to enable patients to be comfortable and free of pain, so that they live each day as fully as possible. End-of-life/hospice care begins after treatment of the disease is stopped and when it is clear that the person is not going to survive the illness (usually when the person is expected to live 6 months or less).

Hospice care is a high risk setting for pressure injury development, as individuals at the end of life experience organ system failure. Skin is the largest organ of the body, and it is subject to failure like any organ. Because of this, skin breakdown is inevitable for many individuals at the end of life,³²⁻⁴⁰ and healing is often not a realistic goal.^{38,41-44} In addition, new pressure injuries may occur in this vulnerable population.⁴⁵

Areas of pressure injury prevention and treatment with notable relevance to individuals in palliative care settings include establishing the individual and/or informal caregivers' goals of care, which are discussed in the guideline chapter *Pressure Injury Assessment and Monitoring of Healing*. The chapter on *Pain Assessment and Treatment* is also particularly relevant to individuals receiving palliative care.

Individuals with Obesity

Obesity has increased dramatically in the last few decades.⁴⁶ Currently 65% of the global population live in countries in which being overweight or obese is associated with greater mortality than being underweight.⁴⁷ The World Health Organization (WHO) defines overweight and obesity as abnormal or excessive fat accumulation that may impair health.⁴⁷ In clinical settings, obesity is defined by body mass index (BMI), body composition assessment or another validated approach. Three classifications of overweight severity are identified by the WHO when using BMI to define obesity:⁴⁷

- Obese I: BMI 30.0 to 34.9 kg/m²
- Obese II: BMI of 35.0 to 39.9 kg/m²
- Obese III: BMI ≥ 40.0 kg/m².

Caring for the individual with obesity can be challenging for both patients and primary caregivers. These patients have specific health care requirements different to those of standard sized individuals. These challenges are influenced by the patient's altered integumentary system, mobility status, body shape, weight and other health-related comorbidities.

Limited or lack of availability of equipment such as type-specific bed, support surfaces, and mobility devices are additional challenges for the primary caregivers.

A lack or limited availability of equipment for individuals with obesity lead to difficulty in management of existing pressure injuries and in preventing further skin breakdown. In addition, occupational health risk consideration should be provided to primary caregivers in choosing equipment for these individuals.

Being aware of the specific needs of the individual with obesity will ensure a proactive approach is embraced to prevent harm to skin and improve the quality of care for this specific patient group.

Obesity is associated with various skin and tissue health problems and diseases;⁴⁸ however, precise causal relationships between obesity and pressure injury development are unclear. Based on finite element modeling,⁴⁹ epidemiological data,⁵⁰ and clinical experience, there appears to be a U-shaped relationship between BMI and pressure injury occurrence. Both very thin individuals and those who are overweight to obese are at higher pressure injury risk compared to individuals within a normal BMI range. However, while the association between underweight and increased pressure injury risk is established, evidence supporting the relationship with obesity seems to be less clear.

Epidemiological studies have demonstrated strong, weak or no relationship between obesity and pressure injuries.⁵¹⁻⁵³ Compher et. al.⁵⁴ conducted a secondary analysis of a cohort study (n = 3214) on risk factors for pressure injuries and found a reduced odds ratio (OR) for pressure injuries in obese individuals (adjusted OR = 0.70, 95% CI 0.40 to 1.0), indicating that obesity might be a protective factor. Possible explanations for these findings are that non-comparable skin areas, non-comparable pressure injury Categories/Stages, and use of different BMI cut-offs and categories.

Shear and friction are often increased as the individual with obesity is inclined to drag their heels and sacrum when getting out of bed. The increased pressure on the bowel and bladder from abdominal weight increases the risk of stress incontinence and diaphoresis, which increases the risk of skin maceration. Obesity can also compromise respiration due to impaired diaphragmatic movement and subsequent impaired tissue perfusion.

There is a small but growing body of research on pressure injury prevention and treatment in obese populations that has informed general recommendations and implementations considerations throughout the guideline. The chapter on *Support Surfaces* includes good practice statements and recommendations on selecting support surfaces for individuals with obesity.

Neonates and Children

Pressure injuries are a significant concern for the pediatric population. Recognition of the risk of pressure injuries in neonates and children is important. A lack of awareness of the risk or a perception that pressure injuries are not of concern to this special population can lead health professionals and informal caregivers to overlook the importance of skin assessment and preventive care.⁵⁶

Pressure injury incidence rates of 0.29% to 27% have been reported in the literature.⁵⁷ Pediatric pressure injury prevalence rates reported in the international literature since 2000 range from 0.47%⁵⁸ to as high as 75%,⁵⁹ with the highest prevalence reported in neonates and children with chronic illness.⁵⁷ For example, Habiballah et al. (2016)⁶⁰ reported that 90.9% of pressure injuries identified in a prevalence study across two pediatric hospitals were in children in critical care wards. Pediatric individuals with medical devices are also at higher risk of pressure injuries.⁶¹

Skin in fullterm neonates generally has a well developed epidermis and stratum corneum, although it is still developing.⁶² Premature neonates have underdeveloped skin that has fewer stratum corneum layers and therefore performs an immature barrier function and increased fragility.^{62,63} Immature skin has impaired thermal properties and increased permeability, leading to water and electrolyte imbalance.⁶² The skin's protective and absorption properties are of particular concern in gestational ages under 32 weeks. Skin may be more dry and scaly, which is generally addressed through humidity management in the neonate intensive care setting. Immature skin places the neonate at higher risk of skin damage from pressure and shear.^{62,63} Neonates and children are also at risk of pressure injuries due to their relatively larger skin surface area, and their larger head circumference, which increases the risk of occiput pressure injuries.^{56,60,64}

Risk factors for pressure injuries specific to neonates and children, particularly those in the intensive care setting, are discussed in the guideline chapter *Risk Factors and Risk Assessment*. That chapter of the guideline also discusses assessing pressure injury risk, presenting general principles that apply to both adults and neonates and children, as well pediatric-specific pressure injury risk assessment tools. The particular risk to neonates and younger children presented by medical devices is discussed in the chapter *Device Related Injuries*.

Neonates and children are at higher risk of nutritional deficiencies due to having an increased nutritional requirement per unit weight to meet normal growth needs, as well as having smaller appetites and dietary intake. Additionally,

children with or at risk of a pressure injury for the most part have other severe acute or chronic comorbidities that influence both nutritional needs and the ability to meet these needs.⁶⁵ The guideline chapter *Nutrition Assessment and Treatment* addresses the nutritional issues to consider in the care of neonates and young children in the context of pressure injury prevention and treatment.

Immature skin is also at increased risk of damage due to epidermal stripping from wound dressings or toxicity from exposure to topical agents.^{62,63} These risks should always be considered when managing skin care and applying products to the skin of neonates and young children.⁶³ This context should be considered when applying the recommendations in the guideline chapter on *Wound Dressings*, and those relating to prophylactic dressings discussed in the guideline chapters, *Preventive Skin Care*, *Device Related Injuries* and *Heel Pressure Injuries*. Suitability of products for application to vulnerable immature skin should also be considered when applying the recommendations in the guideline chapter *Infection and Biofilm*.

Informal caregivers (e.g., parents and other family members) play a significant role in establishing care goals, care planning and, particularly when the child is community-based, in delivering care to prevent and treat pressure injuries. Engagement of the child's family or legal guardian in all aspects of care is crucial. The guideline chapter addressing patient and informal caregiver education, *Quality of Life, Self-care and Education*, is of particular significance to this population group.

Individuals in Community, Aged Care and Rehabilitation Settings

Community settings refer to individuals who are being cared for within the community. The term 'community setting' covers a wide range of settings that vary between geographic regions. In different geographic contexts, the term community setting can include home-based care, supported accommodation, residential aged or disability care, general practice, palliative care units, rehabilitation care and visiting medical/nursing services.⁶⁶ Much of the research on individuals in the community includes older adults living in aged care providing various levels of clinical support, and individuals in rehabilitation. In many of these community settings, pressure injuries are complex to prevent, assess and treat due to the wide range of both contributing factors and demographics of individuals who may be at risk or experience a pressure injury. Throughout the discussion in this section of the guideline, descriptions of the setting as reported in the literature has been used.

Community-based populations identified as having a higher risk of pressure injuries include older adults,⁶⁷ individuals living with a spinal cord injury (SCI),^{15,29,30,68-72} and people with an intellectual or physical disability.⁷³ Prevalence and incidence surveys report highly varied statistics on individuals living in the community. Variations in prevalence and incidence rates relate to the type of community-based population studied, the methods of identifying pressure injuries (e.g., patient surveys versus clinical inspection) and the timing of surveys (e.g., screening community-based individuals on admission to hospital versus an ad-hoc survey of community-based home care services), among other with methodological variations. Accurate prevalence and incidence rates for pressure injuries in the community are impossible to establish because not all cases will be known to health professionals; however, the following studies report recent prevalence and/or incidence surveys and provide an indication of the extent of pressure injuries in different community contexts.

In a community-based pressure injury prevalence study conducted in the UK (n = 1,680), Stevenson et. al.⁶⁶ reported 0.77 per 1,000 individuals in residential homes, palliative care units, nursing homes and rehabilitation facilities had a Category/Stage I or greater pressure injury. For individuals living in their own home, the pressure injury prevalence rate was 0.40 per 1,000.⁶⁶ Another study⁷⁴ reported point prevalence of pressure injuries in one UK district (population 254,000). Participants were those known to aged care facilities, general practices, walk-in clinics and community nursing services, with additional participants identified through a wound dressing scheme operating in the region. Prevalence of any wound was 1.07 per 1,000 district residents, with pressure injuries accounting for 13% of these wounds. In this study, 38% of identified pressure injuries were Category/Stage III or IV.⁷⁴ In a study⁷⁵ set in one hospital in the US, pressure injury inspections were conducted for all admissions over a 12-month period (n = 44,202). There were 1,435 (3.04%) pressure injuries identified, of which 71% were in individuals admitted from a community dwelling (as compared to an institutional dwelling). While this provides an indication of the prevalence of pressure injuries in that community,⁷⁵ only individuals requiring acute hospital care (for any reason) were included in the survey. In Taiwan, a clinical audit of individuals receiving care in their home reported an incidence of new pressure injuries of 14.3% during the 4-6-week follow-up period. 20.8% of the new pressure injuries were Category/Stage I pressure injuries, 75% were Category/Stage II pressure injuries, and 4.2% were Category/Stage III.⁷⁶ In this study,⁷⁶ individuals who were readmitted to hospital for any reason were excluded from the survey, resulting in potential under-reporting.

One of the challenges in addressing pressure injury prevention and treatment in the community setting is understanding the health care environment in which the person was receiving care when the pressure injury developed.^{75,77} While

some individuals develop a pressure injury in the community setting, many others return to the community with a pressure injury that developed in another health care setting.

When a pressure injury develops, a community-based individual could receive ongoing pressure injury assessment and treatment in a wide range of clinical settings including wound care clinics, urgent care/emergency departments, geriatric or rehabilitation units, home-based care and convalescence care.^{66,78} Regardless of where the pressure injury is acquired, treatment of pressure injuries until complete resolution is rarely achieved in acute, emergency or rehabilitation care, meaning individuals often receive ongoing management in their usual home environment. Readmission to hospital is not uncommon, particularly for older adults. In one cross sectional survey (n = 1,038),⁶⁷ individuals with a pressure injury were almost three times more likely to require readmission to hospital from a community dwelling (odds ratio [OR] 2.9, 95% CI 1.5 to 5.7), and about 1.5 times more likely to be readmitted from a nursing home (OR 1.6, 95% CI 1.2 to 2.1). A significant pressure injury (or injuries) may take many months to heal in the community; and for some individuals complete healing might never occur. A cohort trial conducted over four years in Korea (n = 184) found that the probability of a Category/Stage I or II pressure injury healing each month was 5.12%, and only 10% of community-based pressure injuries completely healed within 12 months.⁷⁹ A second community-based cohort study, conducted in the UK, reported complete healing within 12 months of 69% of Category/Stage II pressure injuries, 41% of Category/Stage III pressure injuries and 21% of Category/Stage IV pressure injuries, with a mean time to healing of 5.4 months.⁸⁰ Transitioning between health care settings for treatment was identified in one qualitative study (n = 12) as a time when individuals with a pressure injury feel more vulnerable and lacking control, and concerns about continuity of care arise.^{77,81}

Individuals at risk of or living with a pressure injury in the community face specific challenges. Access to health services in the community is not always easy or possible and varies widely across geographic regions. In some regions a wide range of different community-based care options are available, introducing variation in the type of care delivered and increased transition between care services. In one UK survey, 60% of individuals requiring management of a pressure injury had initial contact with a general practitioner, 14% initially consulted a practice nurse and 8% were managed by another type of health professional.⁸⁰ Another UK-based study reported pressure injuries being managed in aged care, rehabilitation care, general practice, walk-in clinics, community nursing services and by individuals and their informal caregivers, indicating the wide range of care models available in some locations.⁷⁴ However, the individual's knowledge of available care options, local referral requirements, caseloads and appropriate transportation to a service all influence accessibility, if local care or support services exist at all.

Use of appropriate equipment (including support surfaces and heel off-loading devices) is a major challenge. For many individuals, access to equipment is limited due to financial cost or availability. In a mixed-methods study conducted in the UK (n = 90),^{77,81} only 31% of participants had used the equipment they had been recommended. In interviews (n = 12), participants reported that unsuitability to the home environment and discomfort were responsible for their poor uptake of equipment.^{77,81} For those individuals with a pressure injury, health professional services and wound dressings represent another significant challenge. Being available for community-nurse visits or accessing a wound service on a regular basis for wound management while maintaining a normal lifestyle routine can be a problem. The financial cost of both health professional services and wound dressings is substantial, and this is met by the patient in many geographic settings. A recent cost analysis reported a mean cost of £8,720 (UK, 2018) for treating a pressure injury in the community, of which nursing services accounted for 80% of the costs.⁸⁰ In Canada, the total average cost for an individual with SCI and a Category/Stage II, III or IV pressure injury was reported at \$4,748 per month (Canada, 2013), with 59% of costs attributed to health professional services and hospital admissions.³⁰ While community-based funding packages are available in some geographic regions, these are often limited to individuals with specific medical diagnoses or care needs, and the vast majority of community-based individuals at risk of pressure injury have no access to funding support.

Throughout the guideline, implementation considerations specific to individuals in community settings have been provided when applicable. The *Nutrition in Pressure Injury Prevention and Treatment* chapter includes tools appropriate for assessing nutritional status in community-based older individuals. The *Repositioning and Early Mobilization* chapter includes implementation considerations for individuals in the community, particularly those spending prolonged time in seated positions. The importance of weight shift and pressure relief maneuvers is highlighted. These interventions should be implemented in conjunction with selection of appropriate chairs/wheelchairs and pressure redistribution cushions, as discussed in the *Support Surfaces* chapter. The *Support Surfaces* chapter also includes implementation considerations when selecting beds, mattresses and cushions for use in community settings. The guideline chapter on *Quality of Life, Self-Care and Education* is of particular significance to community-based individuals, as this chapter outlines strategies to assess and promote self-care skills, which are essential for many individuals in the community who have lower levels of contact with health professionals. The chapter discusses education needs of patient individuals, and includes data collected from an international survey of patients and informal caregivers on their knowledge needs. The recommendations throughout the guideline are generally of particular relevance to older adults living in aged care settings. In most chapters of the guideline, research supporting recommendations has been undertaken in aged care settings, particularly high level care.

Individuals in the Operating Room

Pressure injuries frequently occur in individuals in surgical units or wards. Pressure injury incidence directly attributable to the operating room ranges between 4% and 45%.^{57,82,83} It is generally assumed that pressure injuries that become visible during the early postoperative period began during the intraoperative (surgical) period.⁸³ The pressure injury incidence data should be interpreted with some caution, as attribution related to causation can be nebulous. The time between development of a pressure injury and the point when a pressure injury becomes visible at the skin varies between several hours to three-to-five days.⁸⁴ However, some lesions are so clearly related to restraints, devices or posture during surgery, or occur so shortly after surgery, that there can be little doubt about the causation. Research also shows that pressure injuries caused during surgery can be misdiagnosed as burns.⁸⁵ There is a need for heightened vigilance in all care units where pressure injuries develop. The care unit discovering a pressure injury should not bear sole responsibility for prevention or sole attribution of causation. Prior to visualization of the pressure injury at the skin level, the duration and intensity of pressure that occurred in the prior 48 to 72 hours needs to be explored. At all levels there is a need for transparency and understanding that facility acquired pressure injuries are a system problem that is shared by all care units and disciplines.

During surgery, patients are immobile, positioned on a relatively hard surface, are not able to feel the pain or discomfort caused by pressure and shearing forces, and are unable to change their position in order to relieve pressure. The duration of immobility is generally not limited to the duration of the surgery; individuals are already immobile during the preoperative period and often remain in the same position until their arrival in the recovery room. The clinical circumstances surrounding the pre, intra and post-operative experience of the individual give rise to additional pressure injury risk factors to consider in individuals undergoing surgery. As with other care delivery settings, risk assessment tools and care bundles are being developed specifically for the operating room. The guideline chapter on *Risk Factors and Risk Assessment* includes evidence-based recommendations specific to individuals in the operating room.

Given the individual's immobility during the operative period, consideration given to positioning, the opportunity to reposition, the support surface and use of additional positioning aids (e.g., facial pillows) is crucial. The guideline chapters on *Repositioning and Early Mobilization* and *Support Surfaces* include recommendations specific to individuals in the operating room. Of particular significance to individuals in the operating room is prevention of heel pressure injuries, and the guideline chapter *Heel Pressure Injuries* details pertinent evidence-based recommendations relevant to the operating room setting.

Individuals in Transit

Individuals in transit to or between clinical care settings (e.g., in an ambulance or awaiting admission in emergency department) are often at high risk of pressure injuries due to immobility or higher morbidity. During the time spent being transported and awaiting admission individuals can spend extended periods of time immobilized, and this duration frequently precedes care in a clinical setting that is associated with high risk of pressure injuries, such as the operating room or critical care. Research focused on the risks of pressure injuries during transportation is sparse. One study⁸⁶ conducted in Sweden reported an audit of care provided in the ambulance, emergency department and ward for 183 older adults (aged more than 70 years) requiring emergency transportation to hospital due to neurological symptoms or a reduced general condition. Using a skin inspection protocol that was being explored in this study, during their stay in the emergency department, 60% of individuals were identified to be at risk for pressure injuries. Despite being admitted to the study with no pressure injury present, 8.2% of individuals developed a heel pressure injury during their time in the emergency department, after a median ambulance time of 25 minutes (median duration of emergency department stay was not reported).⁸⁶ Although all these individuals had received a standard hospital trolley in the ambulance and emergency department, this was not found to be statistically significantly related to developing a heel pressure injury (only 1.6% of all participants received a bed rather than a trolley). In an Australian study,⁸⁷ a random sample of adults (n = 212) arriving to the emergency department by ambulance received a skin inspection within one hour of triage. Prevalence of pressure injuries on presentation to the emergency department was 5.2% (95% CI 2.6% to 9.1%). The pressure injuries were primarily Category/Stage I (42.8%) and occurred most often on the sacrum, buttock and ear. The time spent in the ambulance was statistically significantly longer for individuals who developed a pressure injury, although the effect size was small ($r = 0.14$, $p = 0.046$).⁸⁷ These prevalence studies highlight the importance of early risk identification and preventive care for vulnerable populations during transit.

Some evidence on the advantages of initiating preventive pressure injury care earlier in the care journey is available,⁸⁸⁻⁹⁰ however, this population has received minimal research focus. Following stabilization, initiating a pressure injury risk assessment and a skin inspection is important. The *Risk Factors and Risk Assessment* chapter discusses risk screening, which is important in promptly identifying individuals who require more a rapid comprehensive assessment and preventive pressure injury care, concurrent with managing critical morbidity.^{86,88,89} The *Support Surfaces* chapter

includes recommendations for individuals in transportation, including individuals who require immobilization due to suspected spinal injury. The guideline chapter on *Device Related Pressure Injuries* includes recommendations on use of immobilization devices. The *Heel Pressure Injuries* and *Preventive Skin Care* guideline chapters are also of particular relevance to individuals in transit, as recent research supports the early implementation of preventive care, particularly for the heels.^{86,88}

References

1. Nates JL, Nunnally M, Kleinpell R, Blosser S, Goldner J, Birriel B, Fowler CS, Byrum D, Miles WS, Bailey H, Sprung CL. ICU admission, discharge, and triage guidelines: A framework to enhance clinical operations, development of institutional policies, and further research. *Crit Care Med*, 2016; 44(8): 1553-602.
2. Sevransky J. Clinical assessment of hemodynamically unstable patients. *Curr Opin Crit Care*, 2009; 15(3): 234-8.
3. Shahin ESM, Dassen T, Halfens RJG. Incidence, prevention and treatment of pressure ulcers in intensive care patients: a longitudinal study. *International Journal of Nursing Studies*, 2009; 46(4): 413-421.
4. Shahin ESM, Dassen T, Halfens RJG. Pressure ulcer prevention in intensive care patients: guidelines and practice. *Journal of Evaluation In Clinical Practice*, 2009; 15(2): 370-374.
5. Vangilder C, Amlung S, Harrison P, Meyer S. Results of the 2008 - 2009 International Pressure Ulcer Prevalence Survey and a 3-year, acute care, unit-specific analysis. *Ostomy Wound Management*, 2009; 55: 39-45.
6. Langemo DK, Anderson J, Volden C. Uncovering pressure ulcer incidence. *Nurs Manage*, 2003; 34(10): 54-57.
7. Zhao G, Hiltabidel E, Liu Y, Chen L, Liao Y. A cross-sectional descriptive study of pressure ulcer prevalence in a teaching hospital in China. *Ostomy Wound Management*, 2010; 56: 38-42.
8. Coyer F, Miles S, Gosley S, Fulbrook P, Sketcher-Baker K, Cook JL, Whitmore J. Pressure injury prevalence in intensive care versus non-intensive care patients: A state-wide comparison. *Aust Crit Care*, 2017.
9. Nowicki JL, Mullany D, Spooner A, Nowicki TA, McKay PM, Corley A, Fulbrook P, Fraser JF. Are pressure injuries related to skin failure in critically ill patients? *Aust Crit Care*, 2017.
10. Catala Espinosa AI, Hidalgo Encinas Y, Cherednichenko T, Flores Lucas I, Gonzalez Tamayo R, Garcia-Martinez MA, Herrero-Gutierrez E. [Correlation between body mass index and development of pressure ulcers in intensive care medicine]. *Enferm Intensiva*, 2014; 25(3): 107-13.
11. Cox J, Roche S, Murphy V. Pressure injury risk factors in critical care patients: A descriptive analysis. *Adv Skin Wound Care*, 2018; 31(7): 328-334.
12. Houghton PE, Campbell KE, CPG Panel, Canadian Best Practice Guidelines for the Prevention and Management of Pressure Ulcers in People with Spinal Cord Injury. A resource handbook for clinicians. 2013, Ontario Neurotrauma Foundation: <http://www.onf.org>.
13. Van Der Wielen H, Post MWM, Lay V, Glasche K, Scheel-Sailer A. Hospital-acquired pressure ulcers in spinal cord injured patients: Time to occur, time until closure and risk factors. *Spinal Cord*, 2016; 54(9): 726-731.
14. Li C, DiPiro ND, Krause J. A latent structural equation model of risk behaviors and pressure ulcer outcomes among people with spinal cord injury. *Spinal Cord*, 2017; 55(6): 553-558.
15. Morita T, Yamada T, Watanabe T, Nagahori E. Lifestyle risk factors for pressure ulcers in community-based patients with spinal cord injuries in Japan. *Spinal Cord*, 2015.
16. Mathew A, Samuelkamaleshkumar S, Radhika S, Elango A. Engagement in occupational activities and pressure ulcer development in rehabilitated South Indian persons with spinal cord injury. *Spinal Cord*, 2013; 51(2): 150-155.
17. Hoh DJ, Rahman M, Fargen KM, Neal D, Hoh BL. Establishing standard hospital performance measures for cervical spinal trauma: A nationwide in-patient sample study. *Spinal Cord*, 2016; 54(4): 306-313.
18. Wilson J, Arnold P, Singh A, Kalsi-Ryan S, Fehlings M. Clinical prediction model for acute inpatient complications after traumatic cervical spinal cord injury: a subanalysis from the Surgical Timing in Acute Spinal Cord Injury Study. *J Neurosurg Spine*, 2012; 17(1): 46-51.
19. Ploumis A, Kolli S, Patrick M, Owens M, Beris A, Marino RJ. Length of stay and medical stability for spinal cord-injured patients on admission to an inpatient rehabilitation hospital: a comparison between a model SCI trauma center and non-SCI trauma center. *Spinal Cord*, 2011; 49(3): 411-415.
20. Wannapakhe J, Arrayawichanon P, Saengsuwan J, Amatachaya S. Medical complications and falls in patients with spinal cord injury during the immediate phase after completing a rehabilitation program. *J Spinal Cord Med*, 2015; 38(1): 84-90.
21. Kovindha A, Kammuang-Lue P, Prakongsai P, Wongphan T. Prevalence of pressure ulcers in Thai wheelchair users with chronic spinal cord injuries. *Spinal Cord*, 2015; 53(10): 767-771.
22. Richard-Denis A, Thompson C, Bourassa-Moreau E, Parent S, Mac-Thiong JM. Does the acute care spinal cord injury setting predict the occurrence of pressure ulcers at arrival to intensive rehabilitation centers? *Am J Phys Med Rehabil*, 2016; 95(4): 300-308.
23. Wu Q, Ning GZ, Li YL, Feng HY, Feng SQ. Factors affecting the length of stay of patients with traumatic spinal cord injury in Tianjin, China. *J Spinal Cord Med*, 2013; 36(3): 237-242.
24. Costa RC, Caliri MHL, Costa LS, Gamba MA. Associated factors to the occurrence of pressure ulcer in spinal cord injured patients. *Revista Neurociencias*, 2013; 21(1): 60-68.
25. Chopra T, Marchaim D, Awali RA, Levine M, Sathyaprakash S, Chalana IK, Ahmed F, Martin ET, Sieggreen M, Sobel JD, Kaye KS. Risk factors and acute in-hospital costs for infected pressure ulcers among gunshot-spinal cord injury victims in southeastern Michigan. *Am J Infect Control*, 2016; 44(3): 315-319.

26. Jackson J, Carlson M, Rubayi S, Scott MD, Atkins MS, Blanche EI, Saunders-Newton C, Mielke S, Wolfe MK, Clark FA. Qualitative study of principles pertaining to lifestyle and pressure ulcer risk in adults with spinal cord injury. *Disabil Rehabil*, 2010; 32(7): 567-578.
27. Ghaisas S, Pyatak EA, Blanche E, Blanchard J, Clark F. Lifestyle changes and pressure ulcer prevention in adults with spinal cord injury in the pressure ulcer prevention study lifestyle intervention. *Am J Occup Ther*, 2015; 69(1): 1-10.
28. Schubart JR, Hilgart M, Lyder C. Pressure ulcer prevention and management in spinal cord-injured adults: analysis of educational needs. *Adv Skin Wound Care*, 2008; 21(7): 322-329.
29. Dunn CA, Carlson M, Jackson JM, Clark FA. Response factors surrounding progression of pressure ulcers in community-residing adults with spinal cord injury. *Am J Occup Ther*, 2009; 63(3): 301-309.
30. Chan BC, Nanwa N, Mittmann N, Bryant D, Coyte PC, Houghton PE. The average cost of pressure ulcer management in a community dwelling spinal cord injury population. *Int Wound J*, 2013; 10(4).
31. World Health Organization. *WHO Definition of Palliative Care*. 2019; Available from: <http://www.who.int/cancer/palliative/definition/en/>
32. Bennett RG, O'Sullivan J, DeVito EM, Remsburg R. The increasing medical malpractice risk related to pressure ulcers in the United States. *Journal of the American Geriatrics Society*, 2000; 48(1): 73-81.
33. Chaplin J. Pressure sore risk assessment in palliative care. *J Tissue Viability*, 2000; 10(1): 27-31.
34. De Conno F, Ventafridda V, Saita L. Skin problems in advanced and terminal cancer patients. *J Pain Symptom Manage*, 1991; 6(4): 247-256.
35. Gilchrist B, Corner, J. Pressure sores: Prevention and management - a nursing perspective. *Palliative Medicine*, 1989; 3: 257-261.
36. Hanson DS, Langemo D, Olson B, Hunter S, Burd C. Evaluation of pressure ulcer prevalence rates for hospice patients post-implementation of pressure ulcer protocols. *American Journal of Hospice & Palliative Care*, 1994; 11(6): 14-9.
37. Moss RJ, La Puma J. The ethics of pressure sore prevention and treatment in the elderly: a practical approach. *Journal of the American Geriatrics Society*, 1991; 39(9): 905-8.
38. Walding M, Andrews C. Preventing and managing pressure sores in palliative care. *Professional Nurse*, 1995; 11(1): 33-4, 37-8.
39. Waller A, Caroline NL. *Pressure Sores*, in *Handbook of Palliative Care in Cancer*. 2000, Butterworth Heinemann: Boston. p. 91-98.
40. Sibbald G, Krasner D, Lutz J. Tip the SCALE toward quality end-of-life skin care. *Nursing Management*, 2011; 42(3): 24-32.
41. Chaplin J, McGill M. Pressure sore prevention. *Palliative Care Today*, 1999; 8(3).
42. Dealey C, *The care of wounds: a guide for nurses*. 1999, Oxford: Blackwell.
43. McDonald A, Lesage P. Palliative management of pressure ulcers and malignant wounds in patients with advanced illness. *J Palliat Med*, 2006; 9(2): 285-295.
44. Tippet AW. Wounds at the end of life. *Wounds: A Compendium of Clinical Research & Practice*, 2005; 17(4): 91-98.
45. Alvarez OM, Meehan M, Ennis W, Thomas DR, Ferris FD, Kennedy KL, Rogers R, Bradley M, Baker JJ, Fernandez-Obregon A, Rodeheaver G. Chronic wounds: palliative management for the frail population. *Wounds: A Compendium of Clinical Research & Practice*, 2002; 14(8): 45-27s.
46. Organization for Economic Co-operation and Development. *OECD: Obesity*. 2012 [cited August 6, 2013]; Available from: <http://www.oecd.org/health/49716427.pdf>.
47. World Health Organization. *10 facts on obesity*. 2013 [cited 2013]; Available from: <http://www.who.int/features/factfiles/obesity/en/>
48. Shipman A, Millington G. Obesity and the skin. *Br J Dermatol*, 2011; 165(4): 743-50.
49. Sopher R, Nixon J, Gorecki C, Gefen A. Exposure to internal muscle tissue loads under the ischial tuberosities during sitting is elevated at abnormally high or low body mass indices. *J Biomech*, 2010; 43(2): 280-286.
50. Kottner J, Gefen A, Lahmann N. Weight and pressure ulcer occurrence: a secondary data analysis. *International Journal of Nursing Studies*, 2011; 48(11): 1339-48.
51. Cai S, Rahman M, Intrator O. Obesity and pressure ulcers among nursing home residents. *Med Care*, 2013; 51(6): 478-86.
52. VanGilder C, MacFarlane G, Meyer S, Lachenbruch C. Body mass index, weight, and pressure ulcer prevalence: an analysis of the 2006-2007 international pressure ulcer prevalence surveys. *J Nurs Care Qual*, 2009; 24(2): 127-135.
53. Swanson MS, Rose MA, Baker G, Drake DJ, Engelke M, Pokorny M, Watkins F, Waters W. Braden subscales and their relationship to the prevalence of pressure ulcers in hospitalized obese patients. *Bariatric Surg Pract Patient Care*, 2011; 6(1): 21-23.
54. Compher C, Kinoshian BP, Ratcliffe S, Baumgarten M. Obesity reduces the risk of pressure ulcers in elderly hospitalized patients. *Journal of Gerontology: Medical Sciences*, 2007; 62(11): 1310-1312.
55. Rose MA, Pokorny M, D. D. Braden subscales and their relationship to the prevalence of pressure ulcers in hospitalized obese patients. *Bariatric Nursing and Surgical Patient Care* 2009; 4(3): 221-226.
56. Baharestani MM, Ratliff CR. Pressure ulcers in neonates and children: an NPUAP white paper. *Adv Skin Wound Care*, 2007; 20(4): 208.
57. Pieper B, National Pressure Ulcer Advisory Panel, eds. *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future*. 2012, NPUAP: Washington, DC.
58. Baldwin KM. Incidence and prevalence of pressure ulcers in children. *Adv Skin Wound Care*, 2002; 15(3): 121-4.
59. Samaniego IA. A sore spot in pediatrics: Risk factors for pressure ulcers. *Pediatr Nurs*, 2003; 29(4): 278-82.
60. Habiballah L, Tubaishat A. The prevalence of pressure ulcers in the paediatric population. *J Tissue Viability*, 2016; 25(2): 127-134.
61. August D, Kandasamy Y. Significance of antenatal glucocorticoid exposure for pressure injury prevalence in neonates. *J Neonatal Perinatal Med*, 2016; 9(1): 23-29.

62. Visscher M, Narendran V. Neonatal infant skin: Development, structure and function. *Newborn Infant Nurs Rev*, 2014; 14(4): 135-141.
63. Oranges T, Dini D, Romanelli M. Skin physiology of the neonate and infant: Clinical implications. *Adv Wound Care*, 2015; 4(10): 587-595.
64. Razmus I, Lewis L, Wilson D. Pressure ulcer development in infants: State of the science. *J Healthc Qual*, 2008; 30(5): 36-42.
65. Mehta N, Compher C, ASPEN Board of Directors. A.S.P.E.N. Clinical guidelines: Nutrition support of the critically ill child. *J Parenter Enteral Nutr*, 2009; 33(3): 260-276.
66. Stevenson R, Collinson M, Henderson V, Wilson L, Dealey C, McGinnis E, Briggs M, Nelson EA, Stubbs N, Coleman S, Nixon J. The prevalence of pressure ulcers in community settings: An observational study. *Int J Nurs Stud*, 2013.
67. Bogaisky M, Dezieck L. Early hospital readmission of nursing home residents and community-dwelling elderly adults discharged from the geriatrics service of an Urban teaching hospital: Patterns and risk factors. *J Am Geriatr Soc*, 2015; 63(3): 548-552.
68. Arora M, Harvey LA, Gliinsky JV, Chhabra HS, Hossain S, Arumugam N, Bedi PK, Lavrencic L, Hayes AJ, Cameron ID. Telephone-based management of pressure ulcers in people with spinal cord injury in low-and middle-income countries: A randomised controlled trial. *Spinal Cord*, 2017; 55(2): 141-147.
69. Clark F, Pyatak EA, Carlson M, Blanche EI, Vigen C, Hay J, Mallinson T, Blanchard J, Unger JB, Garber SL, Diaz J, Florindez LI, Atkins M, Rubayi S, Azen SP. Implementing trials of complex interventions in community settings: The USC-Rancho Los Amigos Pressure Ulcer Prevention Study (PUPS). *Clinical trials* 2014; 11(2): 218-29.
70. Street JT, Noonan VK, Cheung A, Fisher CG, Dvorak MF. Incidence of acute care adverse events and long-term health-related quality of life in patients with TSCI. *Spine Journal*, 2015; 15(5): 923-932.
71. Gould LJ, Olney CM, Nichols JS, Block AR, Simon RM, Guihan M. Spinal cord injury survey to determine pressure ulcer vulnerability in the outpatient population. *Med Hypotheses*, 2014; 83(5): 552-558.
72. Brewer S, Desneves K, Pearce L, Mills K, Dunn L, Brown D, Crowe T. Effect of an arginine-containing nutritional supplement on pressure ulcer healing in community spinal patients. *Journal of Wound Care*, 2010; 19(7): 311-316.
73. Rimmer JH, Yamaki K, Lowry BMD, Wang E, Vogel LC. Obesity and obesity-related secondary conditions in adolescents with intellectual/developmental disabilities. *J Intellect Disabil Res*, 2010; 54(9): 787-794.
74. Hopkins A, Worboys F. Establishing community wound prevalence within an inner London borough: Exploring the complexities. *J Tissue Viability*, 2015; 24(1): 42-49.
75. Corbett LQ, Funk M, Fortunato G, O'Sullivan DM. Pressure injury in a community population: A descriptive study. *J Wound Ostomy Continence Nurs*, 2017; 44(3): 221-227.
76. Tsai Y-C, Lin S-Y, Liu Y, Wang R-H. Factors related to the development of pressure ulcers among new recipients of home care services in Taiwan: A questionnaire study. *Int J Nurs Stud*, 2012; 49(11): 1383-1390.
77. Jackson D, Durrant L, Bishop E, Walthall H, Betteridge R, Gardner S, Coulton W, Hutchinson M, Neville S, Davidson PM, Usher K. Health service provision and the use of pressure-redistributing devices: mixed methods study of community dwelling individuals with pressure injuries. *Contemp Nurse*, 2017; 53(3): 378-389.
78. Jones D. Pressure ulcer prevention in the community setting. *Nurs Stand*, 2013; 28(3): 18-24.
79. Lee E. Longitudinal outcomes of home care in Korea to manage pressure ulcers. *Res Nurs Health*, 2017; 40(3): 255-262.
80. Guest JF, Fuller GW, Vowden P, Vowden KR. Cohort study evaluating pressure ulcer management in clinical practice in the UK following initial presentation in the community: Costs and outcomes. *BMJ Open*, 2018; 8 (7) (no pagination)(e021769).
81. Jackson DE, Durrant LA, Hutchinson M, Ballard CA, Neville S, Usher K. Living with multiple losses: Insights from patients living with pressure injury. *Collegian*, 2017.
82. Bulfone G, Marzoli I, Wuattrin R, Fabbro C, Palese A. A longitudinal study of the incidence of pressure sores and the associated risks and strategies adopted in Italian operating theatres. *Journal of Perioperative Practice*, 2012; 22(2): 50-56.
83. Schoonhoven L, Defloor T, Grypdonck MH. Incidence of pressure ulcers due to surgery. *Journal of Clinical Nursing*, 2002; 11(4): 479-487.
84. Reddy NP, *Effects of mechanical stresses on lymph and interstitial fluid flows.*, in *Pressure sores: clinical practice and scientific approach*, D. Bader, Editor. 1990, MacMillan: London. p. 203-220.
85. Schultz A, Bien M, Dumond K, Brown K, Myers A. Etiology and incidence of pressure ulcers in surgical patients. *AORN Journal*, 1999; 70(3): 434, 437-434, 439.
86. Muntlin Athlin Å, Engström M, Gunningberg L, Bååth C. Heel pressure ulcer, prevention and predictors during the care delivery chain - when and where to take action? A descriptive and explorative study. *Scand J Trauma Resusc Emerg Med*, 2016; 24(1): 134-134.
87. Fulbrook P, Miles S, Coyer F. Prevalence of pressure injury in adults presenting to the emergency department by ambulance. *Aust Crit Care*, 2018; <https://doi.org/10.1016/j.aucc.2018.10.002>.
88. Santamaria N, Gerdtz M, Liu W, Rakis S, Sage S, Ng AW, Tudor H, McCann J, Vassiliou J, Morrow F, Smith K, Knott J, Liew D. Clinical effectiveness of a silicone foam dressing for the prevention of heel pressure ulcers in critically ill patients: Border II Trial. *J Wound Care*, 2015; 24(8): 340-345.
89. Faulkner HR, Dowse C, Pope H, Kingdon-Wells C. The emergency department's response to pressure ulcer crisis. *Wounds UK*, 2015; 11(2): 20.25.
90. Cubit K, McNally B, Lopez V. Taking the pressure off in the emergency departments: evaluation of the prophylactic application of a low shear, soft silicon sacral dressing on high risk medical patients. *Int Wound J*, 2012; 10(5): 579-584.

RISK FACTORS AND RISK ASSESSMENT

Introduction

Risk assessment is a central component of clinical practice and a necessary first step aimed at identifying individuals who are susceptible to pressure injuries. Assessment of pressure injury risk should then inform the second step—the development and implementation of an individualized management plan to mitigate modifiable risk factors and prevent pressure injury development. Both modifiable and non-modifiable risk factors should be included in an overall assessment of pressure injury risk; however, pressure injury prevention focuses on modifiable risk.

Risk assessment aims to identify individuals with characteristics that increase the probability of pressure injury development (see *Figure 4.1*). Individuals who are at high risk are those characterized by multiple risk factors that affect:

- Exposure to damaging mechanical boundary conditions (i.e., the type, magnitude, time and duration of the mechanical load)
- The susceptibility and tolerance of the individual (i.e., mechanical properties, geometry, physiology and repair, and transport and thermal properties of the skin and tissues).

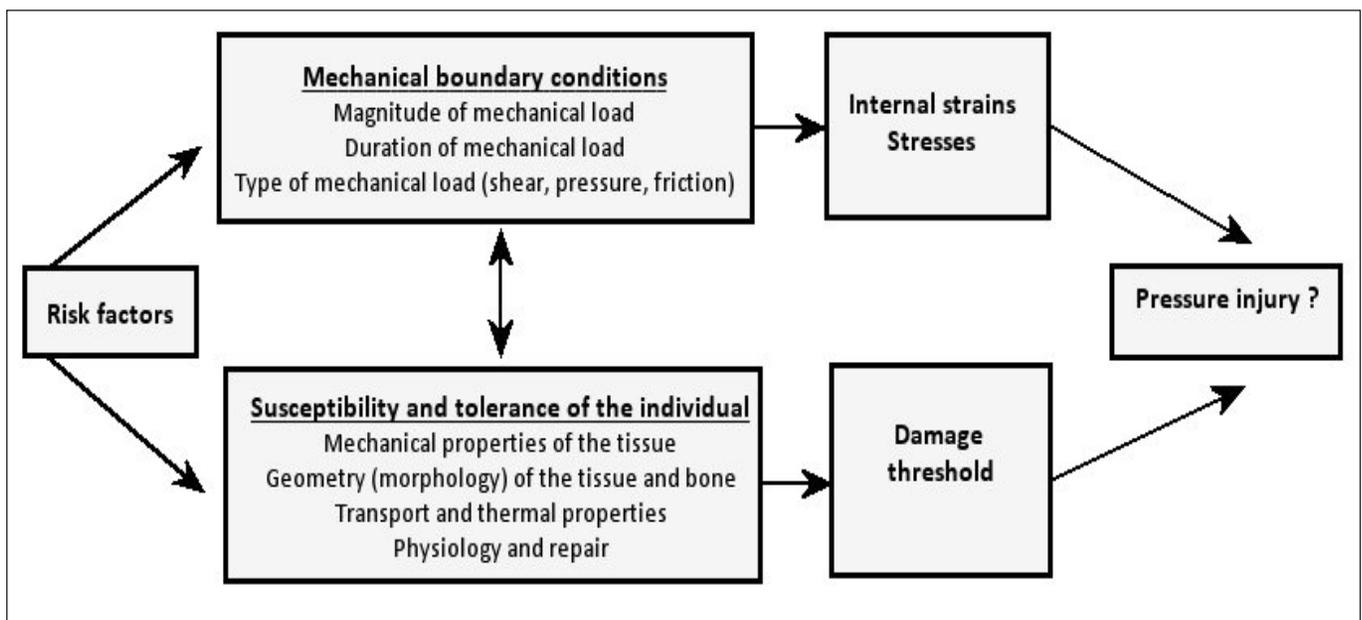


Figure 4.1: Factors influencing the susceptibility of an individual for developing pressure injuries (Adapted from Oomens1 by Coleman et. al.2 and reproduced with permission)

Examples of populations potentially characterized by multiple risk factors include those who:

- Are acutely ill and/or in critical care
- Have sustained a fractured hip
- Have spinal-cord injuries (SCI)
- Have chronic neurological conditions
- Have diabetes mellitus
- Are older
- Are in long-term care homes or community care
- Experience trauma and/or prolonged surgery.

The challenge in clinical practice is to identify individuals within clinical populations who have characteristics that increase the probability of pressure injury development. The epidemiological literature contains many risk factors related to pressure injury development. A rigorous methodology was used to identify the independent predictors of pressure injury development at the individual level (i.e., not organizational factors). However, “independent” is a statistical determination that does not imply causality. Independent factors were categorized into domains,

analyzed for congruence with the conceptual framework and examined for plausible physiological linkages to pressure injury development. This evidence and analysis have informed the guideline recommendations that follow. The recommendations on risk factors are presented according to the strength of supporting evidence at three levels:

- **Consider to be at risk:** there is a **high** likelihood that the presence of this risk factor influences the individual's susceptibility to pressure injury.
- **Consider the impact:** there is a **moderate** likelihood that the presence of this risk factor influences the individual's susceptibility to pressure injury. Clinical judgment is required to determine the importance of this factor to individuals.
- **Consider the potential impact:** there is a **weak** likelihood that the presence of this risk factor influences the individual's susceptibility to pressure injuries. Clinical judgment is required to determine the importance of this factor to individuals.

Where there is a moderate statistical association in heterogeneous risk factor sub-domains or a lack of epidemiological evidence, but the importance of a risk factor is supported by expert opinion and the conceptual framework, good practice statements have been made.

Clinical questions

- What factors put individuals at risk for pressure injury development?
- What are the unique pressure injury risk factors to consider for special populations (if any)?
- What are accurate and effective methods for pressure injury risk assessment?

Risk Factors for Pressure Injuries

Literature for evidence on factors that put an individual at risk of pressure injuries used multivariable modelling to identify independent pressure injury risk factors. Individual factors have been synthesized into categories that influence the two key components of the conceptual framework—mechanical boundary conditions (MBC) or susceptibility and tolerance of the individual (ST).

Table 4.1: Major risk factor categories and their influence on components of the conceptual framework

Risk factor categories	Mechanical boundary conditions (MBC)	Susceptibility and tolerance of the individual (ST)
Activity and mobility limitations	✓	
Skin status		✓
Perfusion, circulation and oxygenation factors		✓
Nutrition indicators		✓
Moisture	✓	✓
Body temperature		✓
Older age	✓	✓
Sensory perception limitations	✓	
Blood markers		✓
General and mental health status	✓	✓
Additional risk factors for specific populations	MBC	ST
Individuals in the operating room	✓	✓
Critically ill individuals	✓	✓
Neonates and children	✓	✓

Activity and Mobility Limitations as Risk Factors for Pressure Injuries in Adults

1.1: Consider individuals with limited mobility, limited activity and a high potential for friction and shear to be at risk of pressure injuries.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)

Evidence Summary

Fifty prognostic studies included factors associated with immobility in a multivariable analysis of risk factors. A large volume of evidence reported measures of mobility/activity limitations as significant in multivariable analyses, including one high³ and five moderate quality⁴⁻⁸ Level 1 studies, and one high quality,⁹ four moderate quality¹⁰⁻¹³ and 27 low/very low quality¹⁴⁻⁴⁰ Level 3 studies. Overall, 76% (38/50) of the prognostic studies reported at least one measure of mobility and activity limitation was a significant risk factor for pressure injuries.

Twelve studies (24%) were unable to establish any measure of mobility/activity as a significant risk factor, including two high quality^{41,42} and one moderate quality⁴³ Level 1 studies and nine low/very low quality⁴⁴⁻⁵² Level 3 studies.

The wide range of clinical settings and types of participants, selection of different risk factors for modeling and range of assessment strategies explain the varied results between studies. Overall, a large body of evidence supports a recommendation to consider the impact of mobility/activity/friction and shear when assessing pressure injury risk.

Implementation Considerations

- Consider mobility and activity limitations to be a necessary condition for pressure injury development (*Expert opinion*).
- Risk assessment subscales for mobility,^{3,11,19-25} friction and shear,^{3,7,11,35,36} and activity^{11,25} may be used as clinical indicators of mobility and activity limitations (*Levels 1 and 3*).
- Consider bedfast/chairfast^{4,14,26-32} individuals to be at pressure injury risk, especially when mobility is also impaired and the potential for friction and shear with movement is increased^{3,7,11,35,36} (*Levels 1 and 3*).
- Consider the requirement to assess population-specific criteria to fully evaluate the individual's types and degree of mobility and activity impairment (e.g., SCI) (*Expert opinion*).
- Consider the duration of mobility limitations on pressure injury risk. Mobility may be impaired on a temporary basis (e.g., sedation,^{4,53} surgery,^{39,54-58} limb fractures,⁵ restraints, guarding with pain, etc.) or permanent basis (e.g., SCI,^{38,40} other paralysis, etc.) (*Levels 1 and 3*).
- Refer to the guideline chapter on *Repositioning and Early Mobilization* for discussion on the use of pressure mapping to evaluate interface pressure.

Evidence Discussion

The recommendations are underpinned by epidemiological evidence (see *Table 4.2*), 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 injuries.

Table 4.2: Summary of evidence for measures of mobility and activity as risk factors for pressure injuries

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Mobility/activity related activities of daily living (ADLs)	MBC	70% (7 of 10 studies)	7 studies in which risk factors were significant in the model ^{9,10,14-18} 3 studies in which risk factors were not significant in the model ^{12,20,48}
Mobility subscale of a risk assessment tool	MBC	52.9% (9 of 17 studies)	9 studies in which risk factors were significant in the model ^{3,11,19-25} 8 studies in which risk factors were not significant in the model ^{13,28,34-36,45,46,50}
Descriptors of activity (e.g., bed/chairfast, immobile)	MBC	56.2% (9 of 16 studies)	9 studies in which risk factors were significant in the model ^{4,14,26-32} 7 studies in which risk factors were not significant in the model ^{6,7,18,20,44,48,49}
Other factors affecting mobility	MBC	45.5% (5 of 11 studies)	5 studies in which risk factors were significant in the model ^{5,12,20,33,34} 6 studies in which risk factors were not significant in the model ^{7,17,27,36,38,41}
General ADLs	MBC	42.8% (3 of 7 studies)	3 studies in which risk factors were significant in the model ^{6,18,20} 4 studies in which risk factors were not significant in the model ^{9,27,28,51}
Friction and/or shear subscale of a risk assessment tool	MBC	33.3% (5 of 15 studies)	5 studies in which risk factors were significant in the model ^{3,7,11,35,36} 10 studies in which risk factors were not significant in the model ^{19-23,28,34,41,45,47}
Activity subscale of a risk assessment tool	MBC	11.8% (2 of 17 studies)	2 studies in which risk factors were significant in the model ^{11,25} 15 studies in which risk factors were not significant in the model ^{3,13,19-24,34-36,41,42,45,50}
Interface pressure	MBC	66.6% (2 of 3 studies)	2 studies in which risk factors were significant in the model ^{8,37} 1 study in which risk factors were not significant in the model ⁵²
Factors affecting mobility related to SCI	MBC	66.6% (4 of 6 studies)	4 studies in which risk factors were significant in the model ^{13,38-40} 2 studies in which risk factors were not significant in the model ^{43,49}

Activity and mobility are specific components of functioning.⁵⁹ Activity refers to the execution of a task or action by an individual.⁵⁹ An activity impairment refers to problems with body function or structure that leads to a reduction or deviation in the individual's type or frequency of activity.⁵⁹ Mobility refers to the ability to change and control one's body position.⁶⁰ A mobility impairment refers to a reduction or deviation in type of frequency of movement. This includes moving in the bed and chair, and ability to maintain specific body positions (e.g. 30° side lying position). In the absence of mobility and activity limitations, other risk factors should not result in a pressure injury. Clinical indicators of limitations to activity and mobility reported in the literature include, but are not limited to:

- General activities of daily living (ADL) function^{6,18,20} (Levels 1 and 3)
- Activity/mobility related ADLs^{9,10,14-18} (Level 3)
- Spinal cord classification^{38,40} (Level 3)
- Limb fracture⁵ (Level 1)
- Length of surgery^{39,54-58} (see Recommendation 1.17) (Level 3).

In terms of the underlying conceptual framework, mobility and activity limitations directly impact upon MBC (see Figure 4.1) and increase the individual's exposure to pressure, shear and resulting frictional forces.

Skin Status as a Risk Factor for Pressure Injuries

1.2: Consider individuals with a Category/Stage I pressure injury to be at risk of developing a Category/Stage II or greater pressure injury.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)

1.3: Consider the potential impact of an existing pressure injury of any Category/Stage on development of additional pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

1.4: Consider the potential impact of a previous pressure injury on additional pressure injury development
(Good Practice Statement)

1.5: Consider the potential impact of alterations to skin status over pressure points on pressure injury risk.
(Good Practice Statement)

1.6: Consider the potential impact of pain at pressure points on pressure injury risk.
(Good Practice Statement)

Evidence Summary

Twenty-four prognostic studies included factors associated with skin status in multivariable analysis of risk factors. Six prognostic studies provided evidence that Category/Stage I pressure injuries are a prognostic factor for Category/Stage II or greater pressure injuries and no studies found this factor to be non-significant. Evidence from two high quality Level 1 studies^{42,61} and one high quality⁶² and three low quality^{26,47,63} Level 3 studies supported the recommendation. Odds ratio of experiencing a Category/Stage II or greater pressure injury after experiencing a Category/Stage I pressure injury ranged from 1.95 to 7.02.

Of eight studies which included existing pressure injury in multivariable modelling, only three report this variable as significant, including one high quality and one moderate quality Level 1 studies,^{6,41} and a very low quality Level 3 study.¹⁸ The remaining five studies, including one high quality Level 1 prognostic study⁴² and four low/very low quality Level 3 studies,^{36,44,64,65} did not find existing pressure injury to be a significant predictor of a new pressure injury. This measure emerges less consistently than other measures of skin status. An existing pressure injury is de facto evidence that the individual can develop a pressure injury. If the risk factors contributing to the initial pressure injury are still present, the individual should be considered at risk for additional pressure injuries.

Twelve of fourteen (85.7%) prognostic studies that reported variations in skin condition as a significant variable in multivariable modelling of pressure injury risk including three high quality Level 1 studies^{41,42,61} and nine low/very low Level 3 prognostic studies.^{16,26,30,39,46,50,66-68} Reported alterations in skin integrity were varied and often poorly defined (e.g., 'unhealthy skin', 'skin type', 'having previous skin problems'). Only two low quality studies^{33,47} did not find an alteration in skin condition to be a significant risk factor.

Implementation Considerations

- Evaluate whether the risk factors and conditions contributing to an initial pressure injury have been adequately addressed with preventive interventions. If these risk factors are still present, the individual is at risk of additional pressure injuries^{6,18,41} (*Levels 1 and 3*).

- Re-evaluate risk factors and the adequacy of preventive measures when additional pressure injuries develop (*Expert opinion*).
- Use clinical judgment to evaluate the potential clinical significance to the individual of alterations to intact skin, including localized erythema over pressure points (*Expert opinion*).
- Consider individuals with a history of pressure injuries to be at risk for breakdown of scar tissue (pressure injury recurrence) in anatomical locations with evidence of a healed Category/Stage III or IV pressure injury^{69,70} (*Expert opinion*).
- Reassess skin at pressure points or other loading surfaces (including those under medical devices) if the individual reports pain.⁶¹ Consider the recommendations included in the guideline chapter *Pain Assessment and Treatment* when assessing and managing pain at pressure points (*Expert opinion*).

Evidence Discussion

The literature identifies that skin/pressure injury status emerges consistently in multivariable modeling and demonstrates a strong statistical association with the development of new pressure injuries where there is an existing Category/Stage I pressure injury (see *Table 4.3*). There is a weak statistical association between the presence of a previous pressure injury and only one study⁶¹ has explored the association between localized skin pain and pressure injuries. Both risk factors require additional research exploration, but are considered clinically important; therefore, a good practice statement has been made. 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. Skin status is a specific risk factor relevant to neonate populations (see *Recommendation 1.19*).

There is a strong statistical association between various descriptors and pressure injury development (e.g., mottled skin, dry skin, alterations to intact skin, skin quality and previous skin problems). However, the reported outcome measures were varied and poorly defined. Measures of skin status included alterations to intact skin, variations to skin condition, skin type, skin quality, having previous skin problems, skin redness, sub-epidermal moisture, dry skin and mottled skin. For many of the descriptors of variation in skin status the physiological mechanism through which pressure injury risk is increased is unclear; therefore, a good practice statement is made regarding the impact of variations in skin status on pressure injury risk.

Table 4.3: Summary of evidence for measures of skin status as risk factors for pressure injuries

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Existing Category/Stage I pressure injury	ST	100% (6 of 6 studies)	6 studies in which risk factors were significant in the model ^{26,42,47,61-63}
Existing pressure injury of any Category/Stage	ST	37.5% (3 of 8 studies)	3 studies in which risk factors were significant in the model ^{6,18,41} 5 studies in which risk factors were not significant in the model ^{36,42,44,64,65}
Alterations in skin status	ST	85.7% (12 of 14 studies)	12 studies in which risk factors were significant in the model ^{16,26,30,39,41,42,46,50,61,66-68} 2 studies in which risk factors were not significant in the model ^{33,47}
Previous pressure injury	ST	33.3% (1 of 3 studies)	1 study in which risk factors were significant in the model ⁷⁰ 2 studies in which risk factors were not significant in the model ^{26,35}
Skin pain	ST	100% (1 of 1 study)	1 study in which risk factors were significant in the model ⁶¹

Perfusion, Circulation and Oxygenation as Risk Factors for Pressure Injuries

1.7: Consider the impact of diabetes mellitus on the risk of pressure injuries.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)

1.8: Consider the impact of perfusion and circulation deficits on the risk of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

1.9: Consider the potential impact of oxygenation deficits on the risk of pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Overall 49 prognostic studies reported a risk factor relating to perfusion, circulation and oxygenation as a significant risk factor for pressure injuries in multivariable modelling. Eight of nineteen (42%) prognostic studies reported a diagnosis of diabetes mellitus was a significant risk factor for pressure injuries including two high quality Level 1 studies,^{42,71} one high quality Level 3 study⁹ and three moderate^{10,12,57} and two low/very low^{72,73} quality Level 3 studies. Eleven studies reported the diabetes variable as non-significant in multivariable modelling including one high quality Level 1 study⁶¹ and ten low/very low quality Level 3 studies.^{15,27,34,35,48,65,67,74-76}

Of 34 prognostic studies which included a perfusion/circulation variable in multivariable modelling, 22 (64.7%) reported one or more perfusion and circulation variables as significant including three moderate quality Level 1 studies,^{4,8,77} and 19 low quality^{27-29,32-34,37,50,51,58,63,64,67,72,73,76,78-80} Level 3 studies. Twelve prognostic studies reported no variables of perfusion and circulation to be significant in multivariable analyses including one high quality Level 1 study,⁴¹ three moderate quality Level 3 studies^{11,12,81} and eight low/very low quality Level 3 studies.^{20,22,31,36,48,49,82,83}

Of 12 prognostic studies that included an oxygenation variable in multivariable modelling, six of the studies^{20,73,74,78,80,84} reported that a measure of oxygenation was significant. All these six studies were Level 3 low/very low prognostic studies.^{20,73,74,78,80,84} The other studies, including a moderate quality Level 1 study⁴ and five moderate and low/very low Level 3 studies,^{20,31,38,40,67} reported an oxygenation variable to be non-significant. The outcome measures varied, with some representing more long term oxygenation impairment and others representing short and medium term deficits, and studies demonstrating significance tended to have larger sample sizes.

Implementation Considerations

- Consider diabetic individuals with associated macro- or microvascular disease to be at risk. Both sensory perception deficits (e.g., peripheral neuropathy) and perfusion deficits associated with diabetes can increase risk of pressure injuries (*Expert opinion*).
- Consider the relevance of medical history of vascular disease (e.g., cerebrovascular accident, cardiac disease, vascular disease and/or peripheral vascular disease)^{4,27,28,34,73,76,78} or respiratory disease⁸⁰ when assessing an individual's pressure injury risk (*Level 1 and 3*).
- Use clinical judgment to determine the relevance measures of circulatory status (e.g. skin circulation, pulse pressure and ankle or toe brachial pressure index and blood pressure)^{29,32-34,50,51,58,63,64,67,77-80} to assessing the individual's pressure injury risk (*Level 3*).
- Consider the impact of peripheral vascular disease when assessing skin on the heels (see the guideline chapter *Heel Pressure Injuries*).
- Use clinical judgment to assess the impact of cigarette smoking^{8,37,72} with vasoconstrictive effects of nicotine on the individual's pressure injury risk (*Level 1 and 3*).
- Use clinical judgment to assess the impact on the individual's pressure injury risk of edema⁶⁷ with changes in interstitial transmural pressure that affect tissue perfusion (*Level 3*).
- Use clinical judgment to assess the impact of mechanical ventilation^{73,74,78,84} and oxygen use²⁰ on pressure injury risk (*Level 3*).

Evidence Discussion

The literature identifies that perfusion and circulation status, and most particularly a diagnosis of diabetes mellitus, emerge as risk factors in epidemiological studies on pressure injury risk. High quality studies demonstrate a strong statistical association between diabetes and the development of new pressure injuries (*Levels 1 and 3*). It is likely that diabetes is a surrogate indicator of the presence of circulatory disease affecting perfusion (i.e., impacts susceptibility and tolerance of the skin). Presence of neuropathy affects exposure to adverse mechanical boundary conditions. Both aspects require consideration in risk assessment.

Moderate and low quality studies demonstrate a moderate statistical association between perfusion and circulation status and the development of new pressure injuries (see *Table 4.4*). However, translation into practice, (i.e., how tissue perfusion and oxygenation can be assessed) is complicated by the wide range of direct and indirect outcome measures examined by researchers. Given the large number of potential measures of perfusion and circulation status, clinical judgment is required when assessing risk associated with factors affecting central (e.g., cerebrovascular accident, cardiac disease, blood pressure, etc.) and peripheral circulation (e.g., peripheral vascular/arterial disease).

Moderate and low quality studies demonstrate a weak statistical association between surrogate measures of oxygenation and pressure injury development. The surrogate measures did not include precise measurement of oxygenation status and may be confounded with other key risk factors, including activity/mobility limitations and illness severity.

In terms of the underlying conceptual framework (see *Figure 4.1*), perfusion, circulation 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. In the context of poor perfusion (e.g. peripheral vascular disease), some tissue may already be injured as a result of hypoperfusion, rendering the tissue more susceptible to the injurious effects of pressure.

Perfusion and oxygenation specific risk factors are also particularly relevant to critically ill individuals (see *Recommendation 1.18*) and neonates and children (see *Recommendation 1.19*).

Table 4.4: Summary of evidence for measures of perfusion, circulation and oxygenation as risk factors for pressure injuries

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Diabetes	ST	42.0% (8 of 19 studies)	8 studies in which risk factors were significant in the model ^{9,10,12,42,57,71-73} 11 studies in which risk factors were not significant in the model ^{15,27,34,35,48,61,65,67,74-76}
Vascular disease	ST	46.6% (7 of 15 studies)	7 studies in which risk factors were significant in the model ^{4,27,28,34,73,76,78} 8 studies in which risk factors were not significant in the model ^{11,12,31,36,48,49,64,81}
Alterations to blood pressure	ST	57.9% (11 of 19 studies)	11 studies in which risk factors were significant in the model ^{33,34,50,51,58,63,64,77-80} 8 studies in which risk factors were not significant in the model ^{22,32,37,48,49,51,73,83}
Circulation (e.g. skin circulation, pulse pressure, ankle-brachial pulse index, etc.)	ST	37.5% (3 of 8 studies)	3 studies in which risk factors were significant in the model ^{29,32,67} 5 studies in which risk factors were not significant in the model ^{36,41,64,76,78}
Smoking	ST	50% (3 of 6 studies)	3 studies in which risk factors were significant in the model ^{8,37,72} 3 studies in which risk factors were not significant in the model ^{48,76,82}

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Edema	ST	25% (1 of 4 studies)	1 study in which risk factors were significant in the model ⁶⁷ 3 studies in which risk factors were not significant in the model ^{4,20,48}
Mechanical ventilation	ST	50% (4 of 8 studies)	4 studies in which risk factors were significant in the model ^{73,74,78,84} 4 studies in which risk factors were not significant in the model ^{4,31,40,67}
Oxygen use	ST	100% (1 of 1 study)	1 study in which risk factors were significant in the model ²⁰
Respiratory disease	ST	25% (1 of 4 studies)	1 study in which risk factors were significant in the model ⁸⁰ 3 studies in which risk factors were not significant in the model ^{13,20,38}

Nutrition Indicators as Risk Factors for Pressure Injuries

1.10: Consider the impact of impaired nutritional status on the risk of pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

A total of 50 prognostic studies explored the relationship between one or more nutrition related variable and pressure injury development. In only 20 (40%) studies including three of high quality (Level 1 and 3),^{9,62,71} three of moderate quality (Level 1 and 3),^{5,57,77} and 14 of low/very low quality (all Level 3)^{22,25-27,46,49,50,53,63,79,83,85-87} was a nutritional variable reported as a significant predictor in multivariable modelling. In 30 studies no measure of nutrition was found to be a significant risk factor. This included three high^{3,41,42} and one moderate⁷ Level 1 studies and one moderate¹¹ and 25 low/very low^{19-21,23,24,28,32-36,39,44,45,47,48,51,54,58,67,76,82,84,88,89} Level 3 studies. There are several limitations associated with measures used to estimate nutritional status and study quality.

Implementation Considerations

- The guideline chapter *Nutrition in Pressure Injury Prevention and Treatment* includes comprehensive discussion on strategies for assessing nutritional status.

Evidence Discussion

Indicators of nutritional deficits considered in multivariable models included a range of descriptors, scales and tools (see *Table 4.5*). The literature demonstrates a moderate statistical association between nutritional status and the development of new pressure injuries. However, translation into practice is complicated by the large range of descriptors used, some of which have stronger physiological links to pressure injury development than others. The insufficient numbers of participants with extremes of weight or body mass index (BMI) in the study populations, and variations in the quality of the evidence also contribute to inconsistencies in the evidence.

In terms of the underlying conceptual framework (see *Figure 4.1*), 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. In patients characterized by extremes of weight/BMI this may also affect exposure to adverse mechanical boundary conditions.

Table 4.5: Summary of evidence for measures of nutritional status as a risk factor for pressure injury development

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Food intake	ST	46.6% (7 of 15 studies)	7 studies in which risk factors were significant in the model ^{5,9,25,27,77,83,85} 8 studies in which risk factors were not significant in the model ^{7,20,23,28,41,53,67,86}
Malnutrition	ST	33.3% (1 of 3 studies)	1 study in which risk factors were significant in the model ⁶² 2 studies in which risk factors were not significant in the model ^{48,54}
Arm measurements (skin fold thickness and arm circumference)	ST	33.3% (1 of 3 studies)	1 study in which risk factors were significant in the model ⁵⁰ 2 studies in which risk factors were not significant in the model ^{26,86}
Weight	ST	20.7% (4 of 13 studies)	4 studies in which risk factors were significant in the model ^{22,26,63,79} 9 studies in which risk factors were not significant in the model ^{21,23,32,33,67,77,84,88,89}
BMI	ST	23.5% (4 of 17 studies)	4 studies in which risk factors were significant in the model ^{49,53,57,71} 13 studies in which risk factors were not significant in the model ^{9,11,22,34,35,41,44,58,67,76,82,83,86}
Nutrition assessment scales	ST	6.3% (1 of 16 studies)	1 study in which risk factors were significant in the model ⁸⁶ 15 studies in which risk factors were not significant in the model ^{3,11,19,21-24,34-36,41,42,45,47,50}
Other measures of nutrition status	ST	22.2% (2 of 9 studies)	2 studies in which risk factors were significant in the model ^{46,87} 7 studies in which risk factors were not significant in the model ^{23,39,44,51,67,88,89}

Moisture as a Risk Factor for Pressure Injuries

1.11: Consider the potential impact of moist skin on the risk of pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Of a total of 33 prognostic studies that included one or more measure of moisture in a multivariable analysis of pressure injury risk factors, 18 studies (54.5%) reported a measure of moisture as significant in multivariable analysis including two high,^{9,62} four moderate,^{10,11,13,55} and 12 low/very low quality^{14,18-20,34-37,50,66,67,72} Level 3 studies. The reported factors included various measures related to incontinence or catheterization, moisture and assessment on a moisture subscale of a risk assessment tool. In 15 studies no measure of moisture was found to be significant. This included two high quality^{3,41} and two moderate quality^{6,7} Level 1 studies and 11 low/very low quality studies^{21,23,24,26,27,32,33,45,47-49} Level 3 studies.

The conflicting findings on the prognostic value of measures of moisture could relate to the diverse range of study participants, differences in methodology and the range of variables included in the modelling (i.e. urinary incontinence, fecal incontinence, dual incontinence, other incontinence, urinary catheter, skin moisture and moisture subscales).

Implementation Considerations

- Assess individuals who have urinary, fecal, dual or unspecified incontinence for pressure injury risk in the presence of mobility and activity impairments^{10,18,20,34,50,66,72} (Level 3).

- Risk assessment subscales for skin moisture could be used as clinical indicators of skin moisture^{19,35,36} (Level 3).

Evidence Discussion

The literature demonstrates a moderate statistical association between excess skin moisture and the development of new pressure injuries (see *Table 4.6*). It is suggested that incontinence is a likely confounding factor in populations characterized by immobility and poor skin status. It is also likely that as well as moisture, there is a skin irritant component to many of the factors where moisture is present. In terms of the underlying conceptual framework, (see *Figure 4.1*) the presence of moisture may impact both the mechanical boundary condition (type of load) and the susceptibility and tolerance of the skin (mechanical properties of the tissues). The coefficient of friction is shown to be greater over moist skin.⁹⁰⁻⁹²

Table 4.6: Summary of evidence for measures of skin moisture as a risk factor for pressure injury development

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Dual incontinence	MBC ST	60.0% (3 of 5 studies)	3 studies in which risk factors were significant in the model ^{10,20,34} 2 studies in which risk factors were not significant in the model ^{6,36}
Skin moisture	MBC ST	66.7% (4 of 6 studies)	4 studies in which risk factors were significant in the model ^{20,37,55,67} 2 studies in which risk factors were not significant in the model ^{7,35}
Moisture subscale of a risk assessment tool	MBC ST	38.5% (5 of 13 studies)	5 studies in which risk factors were significant in the model ^{11,13,19,35,36} 8 studies in which risk factors were not significant in the model ^{3,20,21,23,24,34,41,45}
Fecal incontinence	MBC ST	30.7% (4 of 13 studies)	4 studies in which risk factors were significant in the model ^{9,18,66,72} 9 studies in which risk factors were not significant in the model ^{6,26,32,33,35,37,48,49,62}
Urinary catheter in situ	MBC ST	40.0% (2 of 5 studies)	2 studies in which risk factors were significant in the model ^{14,62} 3 studies in which risk factors were not significant in the model ^{27,47,67}
Urinary incontinence	MBC ST	14.3% (1 of 7 studies)	1 study in which risk factors were significant in the model ³⁴ 6 studies in which risk factors were not significant in the model ^{6,9,13,20,35,48}
Incontinence (type unspecified)	MBC ST	100% (2 of 2 study)	2 studies in which risk factors were significant in the model ^{14,50}

Body Temperature as a Risk Factor for Pressure Injuries

**1.12: Consider the impact of increased body temperature on the risk of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑)**

Evidence Summary

Seven of 12 studies (58.3%) reported that raised body temperature is a prognostic factor for pressure injuries in multivariable modelling. Two moderate quality level 1 studies,^{8,77} a moderate quality Level 3 study⁵⁵ and four low/very low Level 3 studies^{16,37,47,52} found body temperature to be a risk factor in multivariable modelling. The odds ratios for higher body temperature ranged from 1.44⁵² to 8.45.⁵⁵ Conversely, a moderate quality Level 1 study⁴ reported a significant negative association between high body temperature ($\geq 38.5^{\circ}\text{C}$) and pressure injuries in multivariable modelling. The remaining four studies, all low quality Level 3^{24,34,76,85} reported body temperature as a nonsignificant prognostic factor.

Implementation Considerations

- Assess pressure injury risk in individuals with increased body temperature in the presence of mobility and activity impairments^{8,16,37,47,52,77} (*Levels 1 and 3*).
- Consider the impact of an increased body temperature on other pressure injury risk factors, for example, increased perspiration leading to skin moisture (*Expert opinion*).

Evidence Discussion

The literature demonstrates a moderate statistical association between body temperature and the development of new pressure injuries (see *Table 4.7*). 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 4.7: Summary of evidence for increased body temperature as a risk factor for pressure injury development

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Increased body temperature	ST	58.3% (7 of 12 studies)	7 studies in which higher body temperature was significant in the model ^{8,16,18,37,44,47,77} 1 study in which higher body temperature significantly reduced risk ⁴ 4 studies in which risk factors were not significant in the model ^{24,34,76,85}

Older Age as a Risk Factor for Pressure Injuries

1.13: Consider the potential impact of older age on the risk of pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

In total, 51 prognostic studies included age as a factor in multivariable analyses. Of these, 19 studies (37.3%) reported that increasing age was a significant prognostic factor for pressure injuries. Studies that reported a significant relationship included four high quality Level 1 studies,^{3,42,71,93} and one moderate quality Level 1 study.⁷⁷ Two moderate^{10,94} and 12 low/very low studies^{14,22,31,35,72,73,83,84,86,87,95,96} supported the findings.

Conversely, one low quality Level 3 study³⁴ reported a significant negative association between increased age and pressure injuries in multivariable models. The remaining 31 studies reported age as a non-significant factor including one was a high quality Level 1 study⁴¹ and two were moderate quality Level 1 studies.^{6,7} and one high quality,⁹ two moderate quality,^{11,57} and 25 low/very low quality.^{6,15,20,21,23,26,27,32,36,40,49,58,63,64,67,74,76,79,82,85,88,89,97-99} Level 3 studies. The studies used either categorical or continuous measures of age and were conducted in a range of different populations.

Evidence Discussion

The literature demonstrates a weak statistical association between advanced age and the development of new pressure injuries (see *Table 4.8*). 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, circulation and oxygenation; nutrition; and skin moisture. Therefore, in terms of the underlying conceptual framework (see *Figure 4.1*), 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 4.8: Summary of evidence for measures of older age as a risk factor for pressure injury development

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Older age	MBC ST	39.2% (20 of 51 studies)	20 studies in which risk factors were significant in the model ^{3,10,14,22,31,34,35,42,71-73,77,83,84,86,87,93-96} 31 studies in which risk factors were not significant in the model ^{6,7,9,11,15,20,21,23,26,27,32,36,40,41,44,49,57,58,63,64,67,74,76,79,82,85,88,89,97-99}

Sensory Perception as a Risk Factor for Pressure Injuries

1.14: Consider the potential impact of impaired sensory perception on the risk of pressure injuries. (Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Of the 12 prognostic studies that included sensory perception as a factor in multivariable analyses, only four (25%) reported that this measure was a significant factor in the model including one high quality Level 1 study,⁴¹ and one moderate quality¹¹ and two low/very low quality^{35,46} Level 3 studies. The remaining eight studies consisted of a high quality³ Level 1 study and low/very low^{19,21,23,28,34,36,45} Level 3 studies. All studies used the Braden Scale sensory perception subscale to measure this variable.

Implementation Considerations

- Consider sensory perception in individuals with diagnoses associated with local sensory impairment^{11,35,41,46} or the ability to perceive pressure-related discomfort (e.g., diabetes, SCI and peripheral arterial disease) (*Levels 1 and 3*).
- Consider assessing sensory perception in individuals with diagnoses associated with central sensory impairment or the ability to respond to pressure-related discomfort (e.g., coma, sedation, anesthesia, paralysis). (*Level 1 and 3*).
- Consider the use of sensory perception risk assessment tool subscales for assessing sensory perception impairment.^{11,35,41,46} (*Levels 1 and 3*).

Evidence Discussion

The literature demonstrates a weak statistical association between sensory perception deficits and the development of new pressure injuries (see *Table 4.9*). It is likely that sensory perception deficits are confounded with other key risk factors, including activity/mobility limitations, diabetes and illness severity, and these factors dominate in statistical modeling. In terms of the underlying conceptual framework, sensory perception deficits affect exposure to adverse mechanical boundary conditions.

Table 4.9: Summary of evidence for measures of sensory perception as a risk factor for pressure injury development

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Sensory perception subscale of the Braden Scale	MBC	33.3% (4 of 12 studies)	4 studies in which risk factors were significant in the model ^{11,35,41,46} 8 studies in which risk factors were not significant in the model ^{3,19,21,23,28,34,36,45}

Blood Markers as a Risk Factor for Pressure Injuries

1.15: Consider the potential impact of laboratory blood test results on the risk of pressure injuries. (Strength of Evidence = C; Strength of Recommendation = ↔)

Evidence Summary

Twenty-eight prognostic studies included one or more blood test variables in their multivariable model. Of these studies, 17 (60.7%) reported a variable as significant in the model, including one high quality⁴² and one moderate quality⁵ Level 1 prognostic studies and one high quality,⁶² one moderate quality¹³ and thirteen low/very low Level 3^{20,25,26,32,50,51,63,65,66,72,85,86,95} prognostic studies. The largest body of evidence relates to albumin and hemoglobin. The remaining 11 studies, including one moderate quality level 1 prognostic study⁷⁷ and one moderate⁹⁴ and nine low/very low quality^{17,21,22,27,29,44,75,76,83} Level 3 prognostic studies did not find any variable to be important in multivariable modelling.

Evidence Discussion

The literature demonstrates a moderate statistical association between serum albumin and hemoglobin levels and the development of new pressure injuries (see *Table 4.10*). Direct interpretation and application to practice is complicated by the availability of test results and the diversity of causes for abnormality in measures ranging from severe malnutrition to blood loss during surgery. The impact upon the tolerance of the tissues may be multi-factorial. In terms of the underlying conceptual framework (see *Figure 4.1*), blood test results may impact upon the susceptibility and tolerance of the skin by affecting physiology and repair; and transport and thermal properties as follows:

- Low hemoglobin^{20,32,42,50,51,65,72,85,95} (reduces oxygen carrying capacity of the blood and the health of tissues) (Levels 1 and 3).
- Elevated C-reactive protein⁹⁵ (an indicator of inflammation that may affect the health of tissues) (Level 3).
- Low serum albumin^{5,25,62,63,66,86,95} (creates interstitial edema that decreases transmural pressure and perfusion to tissues) (Levels 1 and 3).

Table 4.10: Summary of evidence for hematological measures as risk factors for pressure injuries

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Lymphopenia	ST	100% (2 of 2 studies)	2 studies in which risk factors were significant in the model ^{26,50}
Albumin	ST	50% (7 of 14 studies)	7 studies in which risk factors were significant in the model ^{5,25,62,63,66,86,95} 7 studies in which risk factors were not significant in the model ^{13,21,22,44,75,77,85}
Hemoglobin (Hb)	ST	56.3% (9 of 16 studies)	9 studies in which risk factors were significant in the model ^{20,32,42,50,51,65,72,85,95} 7 studies in which risk factors were not significant in the model ^{29,63,75,76,83,86,94}
Urea and Electrolytes (U&Es)	ST	50% (2 of 4 studies)	2 studies in which risk factors were significant in the model ^{13,86} 2 studies in which risk factors were not significant in the model ^{27,29}
Inflammatory marker (C-reactive protein)	ST	33.3% (1 of 3 studies)	1 study in which risk factors were significant in the model ⁹⁵ 2 studies in which risk factors were not significant in the model ^{17,66}

General and Mental Health Status as Risk Factors for Pressure Injuries

1.16: Consider the potential impact of general and mental health status on pressure injury risk. (Good Practice Statement)

Implementation Considerations

- Where available in clinical records consider the use of population-specific on general health assessment tools and scores when considering the impact of the individual's general and mental health status (e.g. American Society of Anesthesiologists (ASA) Physical Status Classification, Risk of Mortality score, Simplified Acute Physiology Score, Glasgow Coma Scale, Injury Severity Scale, the Acute Physiology and Chronic Health Evaluation (APACHE II) score, etc.)^{12,15,57,83,84,89,98,100} (Level 3).
- In individuals with mobility and activity limitations, consider the impact of mental health status upon sensory perception and ability to independently reposition (*Expert opinion*).

Discussion

The literature demonstrates moderate statistical association between some measures of general health status and a very weak statistical association between mental health status and pressure injury risk (see *Table 4.11*). Variables considered as measures of general health status were wide-ranging and included health status scales, presence of urinary tract or respiratory infection, having a chronic wound, number of nursing interventions, duration of hospital stay, specific medical diagnoses (e.g., cardiac arrest, pulmonary disease, and malignancy) and taking medications (e.g., steroids, vasopressors and sedatives). The wide range of variables used as an indicator of general health status likely contributes to the overall conflicting findings on significance of general health status as a predictor of pressure injuries. A good practice statement has been made because it is likely that general health and mental health status are confounding factors and general indicators of likely deficits in the main areas of risk including mobility/activity, skin status and perfusion, nutrition, skin moisture and sensory perception. Therefore, in terms of the underlying conceptual framework, at an individual level, general health and mental health status may impact upon both the mechanical boundary conditions and all four components of the susceptibility and tolerance of the skin.

Table 4.11: Summary of evidence for mental and general health status measures as risk factors for pressure injuries

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Chronic wounds	ST	50% (1 of 2 studies)	1 study in which risk factors were significant in the model ⁴² 1 study in which risk factors were not significant in the model ⁶³
Medication	MBC ST	35% (7 of 20 studies)	7 studies in which risk factors were significant in the model ^{4,20,53,57,65,78,80} 13 studies in which risk factors were not significant in the model ^{9,32,34,40,47,48,54,67,73,74,81,89}
Norton Scale measures (general physical condition, social activity)	MBC ST	0% (0 of 3 studies)	3 studies in which risk factors were not significant in the model ^{3,24,41}
Infection	ST	44.4% (4 of 9 studies)	4 studies in which risk factors were significant in the model ^{12,38,40,89} 5 studies in which risk factors were not significant in the model ^{28,67,73,74,84}
Length of hospital stay	MBC ST	28.5% (4 of 14 studies)	4 studies in which risk factors were significant in the model ^{22,44,50,64} 10 studies in which risk factors were not significant in the model ^{7,36,65,78,83,84,86,87,97,98}

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Other general health measures	MBC ST	40% (16 of 40 studies)	16 studies in which risk factors were significant in the model ^{4,7,14,15,42,47,62,64,66,71-73,78,80,96,98} 24 studies in which risk factors were not significant in the model ^{9,12,13,20,23,28,29,31,35,38,39,41,48,49,51,54,67,68,74,76,83-85,97}
Other health scales	MBC ST	50% (5 of 10 studies)	5 studies in which risk factors were significant in the model ^{15,57,83,84,98} 5 studies in which risk factors were not significant in the model ^{4,23,44,67,73}
Mental status study specific measures	MBC	18.2% (2 of 12 studies)	2 studies in which risk factors were significant in the model ^{18,62} 10 studies in which risk factors were not significant in the model ^{6,9,17,20,28,35,44,48,50,51}
Mental status subscale of a risk assessment tool	MBC	20% (1 of 5 studies)	1 study in which risk factors were significant in the model ³ 4 studies in which risk factors were not significant in the model ^{24,41,48,50}

Demographic Characteristics as Risk Factors for Pressure Injuries

Prognostic studies have investigated demographic factors as risk factors for pressure injuries (see *Table 4.12*). The findings do not demonstrate a relationship between race and gender and pressure injury risk; therefore, no recommendations have been made. Although prevalence data indicates the rate of pressure injuries is higher in people with darkly pigmented skin,¹⁰¹⁻¹⁰⁶ only one of seven epidemiological studies demonstrated an increased risk in people with darker skin tones. 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 guideline chapter *Skin and Tissue Assessment* for further discussion). In relation to gender, there is no evidence that it is a pressure injury risk factor.

Table 4.12: Summary of evidence for demographic characteristics as risk factors for pressure injuries

Risk factor variables	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
1 study identified increased risk for Caucasian skin, 1 study identified increased risk for dark skin tones	28.6% (2 of 7 studies)	2 studies in which risk factors were significant in the model ^{6,9,33} 5 studies in which risk factors were not significant in the model ^{9,68,82,83,97}
5 studies identified increased risk for males, 2 studies identified increased risk for females	26.9% (7 of 26 studies)	7 studies in which risk factors were significant in the model ^{20,29,67,70,73,95,98} 19 studies in which risk factors were not significant in the model ^{6,9,15,22,33,40,44,48,49,64,82,83,85,86,88,93,96,97,99}

Additional Risk Factors for Individuals in the Operating Room

The pressure injury risk factor domains already presented in this chapter of the guideline are relevant to all adults, including those undergoing surgery. In addition to the previously presented domains, additional risk factors have been identified in individuals in the operating room setting, particularly timing of surgery, duration of surgery, and the individual's clinical severity as determined by ASA classification.

1.17: Consider the impact of time spent immobilized before surgery, the duration of surgery and the American Society of Anesthesiologists (ASA) Physical Status Classification on surgery-related pressure injury risk. (Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Duration of time from admission to surgery

One moderate quality Level 1 study⁴⁴ and two moderate quality Level 3 prognostic studies^{11,12} reported the duration of time prior to surgery was a significant risk factor for development of a pressure injury following surgery. Individuals who were immobile before surgery and had their surgical procedure delayed beyond 12 hours were 1.6 to 1.7 times more likely to develop a pressure injury.^{11,12} Two additional low quality cohort studies^{107,108} indicated that individuals who had delayed surgery were more likely to develop a pressure injury. However, a much smaller moderate quality Level 3 study⁹⁴ found the time between an emergency department admission and having hip surgery was not a significant risk factor.

Duration of surgery

Six low quality Level 3 prognostics studies^{39,54-58} reported multivariable analyses that found the duration of surgery to be a significant prognostic factor for development of a Category/Stage I or greater pressure injury in adults. The studies reported that risk of developing a pressure injury was up to eight times greater for surgeries of longer duration.⁵⁵ Studies with higher odds ratio used categorical outcome of surgery over 5 or 6 hours in duration,^{39,55} and studies with lower odds ratio used surgery/anesthesia length as a continuous outcome measure.^{54,56-58} One additional low quality Level 3 prognostic study⁵⁶ conducted with both adults and children also found surgery duration was a significant risk factor. Although one low quality Level 2 study⁷⁶ and five moderate⁵⁷ and low/very quality Level 3 studies^{44,52,58,75} found duration of surgery was not significant, these studies were generally smaller than those with significant findings.

American Society of Anesthesiologists (ASA) Physical Status Classification

One moderate quality Level 3 prognostic study¹² reported that the individual's classification on the ASA (American Society of Anesthesiologists) Physical Status Classification System was a significant risk factor for development of a pressure injury following surgery. Individuals with a Classification of III (severe systemic disease) or IV (severe systemic disease that is a constant threat to life) on the ASA were more than four times more likely to develop a pressure injury. A smaller, low quality Level 2 study⁴⁸ found ASA Classifications of II, III or IV were not associated with significantly higher pressure injury risk.

Implementation Considerations

- Surgery-specific risk factors should be considered in light of all other risk factors discussed in this guideline chapter (*Expert opinion*).
- Identify individuals with planned lengthy operative times in advance to enable the use of support surfaces, positioning devices, positioning strategies and prophylactic dressings to reduce pressure injury risk. Refer to the relevant chapters of the guideline.
- Where possible, minimize the time of immobilization before and after surgery (*Expert opinion*).

Evidence Discussion

The pressure injury risk factor domains already presented in this chapter of the guideline are relevant to all adults, including those undergoing surgery. In addition to the previously presented domains, additional risk factors have been identified in individuals in the operating room setting (see *Table 4.13*), particularly timing of surgery, duration of surgery, and the individual's clinical severity as determined by ASA classification. In terms of the underlying conceptual framework (see *Figure 4.1*), factors associated with undergoing surgery may impact upon both the mechanical boundary conditions and all the susceptibility and tolerance of the skin.

A long duration of time spent immobilized prior to undergoing surgery is an identified additional risk factor for individuals in the operating room. Studies on the association between delay in surgery and pressure injury risk have been conducted in older adults with hip fractures,¹² and also in mixed surgical populations admitted to intensive care following surgery.^{11,44} (Level 3). Essentially, the duration between admission and surgery reflects the length of time an individual has a mobility/activity limitation, which is discussed as a distinct risk factor earlier in this chapter. Using duration of time to surgery as a measure of mobility/activity limitation is relevant for individuals in the operating room.

Table 4.13: Summary of evidence for factors specific to adults in the operating room

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Time to surgery	MBC	75% (3 of 4 studies)	3 studies in which risk factors were significant in the model ^{11,12,44} 1 study in which risk factors were not significant in the model ⁹⁴
Duration of surgery/ anesthetic	MBC	60% (6 of 10 studies)	6 studies in which risk factors were significant in the model ^{39,54,55,57,58,66} 4 studies in which risk factors were not significant in the model ^{44,52,75,76}
American Society of Anesthesiologists (ASA) score	ST	50% (1 of 2 studies)	1 study in which risk factors were significant in the model ¹² 1 study in which risk factors were not significant in the model ⁴⁸
Other surgical factors (e.g., anesthesia type, positioning, number of surgeries)	MBC ST	25% (4 of 16 studies)	4 studies in which risk factors were significant in the model ^{22,44,57,96} 12 studies in which risk factors were not significant in the model ^{7,38,52,54,63,73,75,76,78,85,86,94}

The longer the surgical procedure, the greater the risk of developing a pressure injury. Two prognostic studies measured surgical duration as a continuous measure and found a statistically significant association between developing a pressure injury and more hours on the operating table. For adults undergoing spinal surgery (n = 209) the risk of developing a Category/Stage I or greater pressure injury was over eight times greater for operations over five hours in duration (OR = 8.12, p = 0.005) (*Level 3*).³⁹ Yoshimura et al. (2015)⁵⁵ reported similar results in a prognostic study exploring risk factors in adults (n = 277) undergoing neurosurgery. For individuals spending six hours or greater in the operating room, the OR of developing a Category/Stage I or greater pressure injury was 8.45 (95% CI 3.04 to 27.46 p < 0.001) (*Level 3*).

Six prognostic studies (all *Level 3*) reported that a surgery duration over five or six hours was a risk factor for pressure injury. The largest odds ratio (OR = 8.45, 95% CI 3.04 to 27.46 p<0.001) was reported for surgery duration over 360 minutes/core temperature >38.1°C as a composite factor in individuals undergoing neurosurgery (n = 277).⁵⁵ Lin et al. (2017)³⁹ reported a similar result (OR 8.12, p = 0.005) for surgery duration over 300 minutes in adults undergoing spinal surgery (n=209). Schoonhoven et al. (2002)⁵⁴ followed 208 individuals undergoing surgery of four hours or longer and reported an OR of 1.0006 (95% CI 1.0037 to 1.0087) for developing a Category/Stage II or greater pressure injury. Chen et al. (2013)⁵⁶ reported similar results (OR = 1.005, 95% CI 1.000 to 2.022, p = 0.036) with an analysis that included both adults and children (n = 286), as did Connor et al. (2010)⁵⁸ (OR = 1.005, 95% CI 1.000 to 1.010, p = 0.038) in an analysis of a larger population of adults (n = 538) undergoing urology surgery. The largest study, conducted in 3,225 adults in intensive care who had undergone surgery, also reported a similar OR (1.07, 95% CI 1.03 to 1.11, p < 0.001).⁵⁷

The ASA Physical Status Classification is a measure of clinical severity. Therefore the significance of this classification score as a predictive risk factor for pressure injuries, as reported by Rademakers et al. (2007)¹² in an analysis of adults undergoing hip fracture surgery (n = 722), is consistent with the data from other studies indicating that overall clinical status is significant to pressure injury risk (*Level 3*).

Additional Risk Factors for Individuals in Critical Care

1.18: Consider the following factors to be population specific pressure injury risk factors for critically ill individuals:

- Duration of critical care unit stay
- Mechanical ventilation
- Use of vasopressors
- Acute Physiology and Chronic Health Evaluation (APACHE II) score.

(Good Practice Statement)

Evidence Summary

Duration of intensive care unit admission

Length of ICU stay was included in 13 multivariable analyses. Seven prognostic (53.8% of studies) Level 3 studies of low^{17,31,89} and very low^{72,88,98,100} quality found that a longer length of time in the ICU was a significant risk factor for development of pressure injuries. The studies reported OR between 1.1 and 1.831. However, five low^{49,54,65,67,81} and one very low⁸⁴ quality Level 3 studies found ICU duration to be non-significant.

Mechanical ventilation

Eight prognostic studies included mechanical ventilation in a multivariable analysis. Of these, four level 3 prognostic studies^{73,74,78,84} of low or very low quality reported mechanical ventilation was a significant risk factor, with odds ratio ranging from 1.042 to 23.604. However, one moderate quality Level 1 study⁴ and four low and very low quality Level 3 studies^{31,40,67,84} found mechanical ventilation was not a significant risk factor.

Acute Physiology and Chronic Health Evaluation (APACHE II) score

Five prognostic studies^{4,67,88,89,100} included APACHE II score as a potential risk factor for pressure injuries in a multivariable analysis. Two of these studies, low⁸⁹ and very low¹⁰⁰ Level 3 studies, found the APACHE II score was a significant prognostic factor, with OR ranging from 1.06 to 16.19. However, a moderate quality level 1 study⁴ and low⁶⁷ and very low⁸⁸ Level 3 studies found the APACHE II score was not a significant prognostic factor for pressure injuries in critically ill individuals.

Vasopressor Use

Eight studies included use of vasopressors in a multivariable analysis. Of these, four Level 4 prognostic studies of moderate⁵⁷ or low/very low quality^{53,78,80} reported use of vasopressors was a significant risk factor, with odds ratio ranging from 1.33 to 4.816. However, one moderate quality Level 3 study⁸¹ and three low/very low quality Level 3 studies^{67,73,89} found vasopressor use was not a significant risk factor.

Implementation Considerations

- Consider risk factors specific to individuals in critical care in light of all other risk factors discussed in this guideline chapter (*Expert opinion*).

Evidence Discussion

The pressure injury risk factor domains already presented in this chapter of the guideline are relevant to all adults, including those who are critically ill. In addition to the previously presented domains, additional risk factors have been identified in individuals in critical care (see *Table 4.14*), particularly duration of intensive care (ICU) admission, being mechanically ventilated, using vasopressors, and the individual's clinical severity as determined by Acute Physiology and Chronic Health Evaluation (APACHE II) score. In terms of the underlying conceptual framework (see *Figure 4.1*), risk factors specific to critically ill individuals impact both the mechanical boundary conditions and the susceptibility and tolerance of the skin.

Table 4:14: Summary of evidence for factors specific to individuals in critical care

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Length of ICU stay	MBC ST	53.8% (7 of 13 studies)	7 studies in which risk factors were significant in the model ^{17,31,72,88,89,98,100} 6 studies in which risk factors were not significant in the model ^{49,54,65,67,81,84}
Mechanical ventilation	ST	50% (4 of 8 studies)	4 studies in which risk factors were significant in the model ^{73,74,78,84} 4 studies in which risk factors were not significant in the model ^{4,31,40,67}
Acute Physiology and Chronic Health Evaluation (APACHE II) score	MBC ST	40% (2 of 5 studies)	2 studies in which risk factors were significant in the model ^{89,100} 3 studies in which risk factors were not significant in the model ^{4,67,88}
Vasopressors	ST	50% (4 of 8 studies)	4 studies in which risk factors were significant in the model ^{53,57,78,80} 4 studies in which risk factors were not significant in the model ^{67,73,81,89}

Additional Risk Factors Specific for Neonates and Children

Many of the risk factors reported throughout this chapter are likely to be relevant to neonates and children as well as adult populations. However, there is a lack of research from high quality prognostic studies available on pressure injury risk in neonates and children. The conceptual model presented in *Figure 4.1* is relevant to all populations at risk of pressure injury and can be assumed to reflect the etiology in neonates and children. Some prognostic studies are available that report multivariable analyses conducted in younger populations and support recommendations specific to neonates and children that can be considered in addition to risk factors discussed throughout this chapter.

1.19: Consider the impact of skin maturity, perfusion and oxygenation, and presence of a medical device on pressure injury risk in neonates and children.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

1.20: Consider the impact of illness severity and the duration of critical care unit stay on pressure injury risk in neonates and children.

(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Evidence from a high quality Level 1 prognostic study¹⁰⁹ indicated that skin texture/maturity is a risk factor for pressure injuries in neonates. Two moderate quality studies providing Level 1¹⁰⁹ and Level 3¹¹⁰ evidence indicated that birth weight was not a significant risk factor for either neonates¹⁰⁹ or children.¹¹⁰ Evidence from a moderate quality Level 1 prognostic study¹⁰⁹ and a moderate quality Level 3 prognostic study¹¹⁰ demonstrated in multivariable analyses the significance of measures of perfusion and oxygenation as a risk factor for pressure injuries in children. The outcome measures for oxygenation and perfusion included the presence of a wide range of oxygen delivery systems, which also increase pressure injury risk due to presence of medical devices.¹¹⁰ Evidence from a moderate quality Level 3 prognostic study¹¹⁰ demonstrated in multivariable analyses the significance of severity of illness and duration of hospital stay as risk factors for pressure injuries in children and neonates.

Implementation Considerations

- Risk factors specific to neonates and children should be considered in light of all other risk factors discussed in this guideline chapter (*Expert opinion*).
- Consider the impact of respiratory support devices (e.g., endotracheal tube, continuous positive airway pressure etc.) in relation to pressure injury risk factors of perfusion and oxygenation status and presence of a medical device¹⁰⁹ (*Level 1*).
- The Dubowitz Neonatal Maturity Assessment Scale provides a measure of skin texture/maturity in neonates.¹⁰⁹ (*Level 1*).

Evidence Discussion

In neonates, skin texture/maturity was identified as a significant pressure injury risk factor¹⁰⁹ (*Level 1*). Skin maturity is directly related to a neonate's age. At 23 to 24 weeks gestation, the stratum corneum is not developed, and by 30 weeks gestation it has only two to three cell layers. The skin appears as transparent, and is particularly fragile.¹¹¹ Thus, the skin of younger infants provides an inadequate barrier and, as indicated in risk studies, is highly susceptible to breakdown (*Level 3*). In terms of the underlying conceptual framework (see *Figure 4.1*), both skin texture and measures of oxygen and perfusion are indicators of susceptibility and tolerance of the skin and reflects mechanical properties of the tissue; the geometry (morphology) of the tissues; physiology and repair; and transport and thermal properties.

The included studies^{109,110} used a range of respiratory support interventions that can be considered measures of perfusion and oxygenation in neonates and children. Presence of a medical device is a risk factor of specific consequence to neonates and children, who may have an increased risk due to their size, weight, and skin immaturity. Further discussion of the risks associated with medical devices is in the guideline chapter *Device Related Pressure Injuries*.

Table 4.15: Summary of evidence for factors specific to neonates and children

Risk factor variables	Conceptual framework component	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Skin texture/maturity	ST	50% (1 of 2 studies)	1 study in which risk factors were significant in the model ¹⁰⁹ 1 study in which risk factors were not significant in the model ¹¹⁰
Perfusion and oxygenation measures	ST	100% (2 of 2 studies)	2 studies in which risk factors were significant in the model ^{109,110}
Presence of a medical device	MBC	100% (2 of 2 studies)	2 studies in which risk factors were significant in the model ^{109,110}
Severity of illness	MBC ST	100% (1 of 1 study)	1 study in which risk factors were significant in the model ¹¹⁰
Length of ICU stay	MBC ST	100% (1 of 1 study)	1 study in which risk factors were significant in the model ¹¹⁰

Pressure Injury Risk Screening and Assessment

1.21: Conduct a pressure injury risk screening as soon as possible after admission to the care service and periodically thereafter to identify individuals at risk of developing pressure injuries.
(Good Practice Statement)

1.22: Conduct a full pressure injury risk assessment as guided by the screening outcome after admission and after any change in status.
(Good Practice Statement)

1.23: Develop and implement a risk-based prevention plan for individuals identified as being at risk of developing pressure injuries.
(Good Practice Statement)

Implementation Considerations

- A screening should be undertaken at first contact with a health professional after admission to the care service, noting that this also includes individuals admitted to community care. (*Expert opinion*).
- When conducting pressure injury risk screening, follow a structured approach that considers major risk factors for pressure injury development in the target population and can be rapidly conducted in all individuals of this population¹¹² (*Expert opinion*).
- For individuals screened as being (very likely) at pressure injury risk, undertake a full pressure injury risk assessment.¹¹² A full pressure injury risk assessment should include an in-depth evaluation of major risk factors and additional condition-specific risk factors for pressure injury development (*Expert opinion*).
- If the full pressure injury risk assessment confirms the 'at risk' status, develop an individualized prevention care plan taking into account the risk factors identified (*Expert opinion*).
- Discuss risk status with the individual and recommended prevention strategies. Modify the pressure injury prevention plan according to individual goals and preferences (*Expert opinion*).
- Repeat the risk assessment as often as required by the individual's acuity. Undertake a reassessment if there is any significant change in the individual's condition (*Expert opinion*).
- Depending on the patient population and clinical setting, develop local standards and policies to address how and by whom the risk assessment is to be conducted. (*Expert opinion*).
- Document all risk assessments (*Expert opinion*).

Discussion

Due to the burden and impact of pressure injury 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. For practical reasons, it is recommended that a risk assessment is organized into two stages:

- (i) Screening to identify individuals who are (very likely) at risk of getting a pressure injury, followed by
- (ii) A full pressure injury risk assessment in those individuals screened as being (very likely) at risk.

Screening for pressure injury risk

The first step, pressure injury risk screening, aims to identify very rapidly and with a minimum of diagnostic effort, those individuals admitted to hospital or any other care service (e.g., aged care facility, home care agency, rehabilitation facility, etc.) who are very likely at risk of developing a pressure injury.¹¹³ Thus, the main purpose of screening is to identify those individuals for whom a certain level of pressure injury risk cannot be ruled out instantly and for whom a full risk assessment is required. Undertaking risk screening should help target resources to those individuals actually in need of a full risk assessment and preventive interventions but should also safeguard that all of individuals who are at risk are identified early and accurately detected.

The pressure injury risk screening should follow a structured and replicable approach, which considers relevant pressure injury risk factors in the target population, local health care infrastructure and procedures, and the training and scope of practice of health professionals in the facility. Conducting a risk assessment should be included in health professional pressure injury education (see the guideline chapter *Health Professional Education*).

To satisfy the screening purposes, screening has to rely on only a few but highly predictive risk factors for pressure injuries. The major risk factors recommended for risk assessment in this guideline chapter provide a theoretically and empirically justified reference framework for pressure injury risk screening (see *Table 4.1* and *Recommendations 1.1 to 1.20*). However, not all of the identified risk factors are equally predictive in all target populations. Risk factors that are highly prevalent in a particular population of interest (e.g., older adult nursing home residents) do not help distinguish between older individuals likely to be at risk of developing a pressure injury and those likely not at risk. The screening should only consider those factors which have the potential to accurately identify those at risk in the population of interest. However, it should always include risk factors such as mobility or activity limitations and measures of impaired skin status (especially the presence of a Category/ Stage I pressure injury) that directly indicate a currently increased and/or insufficiently tolerated exposure towards mechanical loads,^{112,114} and are supported by a high level of evidence.

There are also target populations who may not require any formal screening stage because the presence of major pressure injury risk factors is associated with their reason for admission. For example, in geriatric populations, critically ill individuals, premature/critically ill neonates, or individuals with SCI, several major risk factors are so obviously present and highly prevalent, that any formal screening is superfluous or can be regarded as being automatically completed, indicating 'at risk' status, at admission.

As the pressure injury risk screening must be conducted rapidly, the process should rely on easily accessible information related to the individuals' health history and current health status, either from the individual themselves, their relatives or informal caregivers, other health professionals or health records. For example, existing mobility and activity limitations may be observable directly at the first contact or inferred from existing information on the individual's needs for assistance. Information on the individual's skin status and other major risk categories may be taken from health records or admission papers. Furthermore, the outcome of screening is usually dichotomous (i.e., it indicates that a risk factor of interest is (very likely) present or that risk factor is not present). Individuals should be assumed as being (very likely) at pressure injury risk if the screening results indicate that any of the risk factors included in the screening are (very likely) present.¹¹⁴ Existing mobility or activity limitations or existing pressure injuries should always be regarded as an indication to "at risk" status.^{112,114}

A pressure injury risk screening should be conducted **as soon as possible** (i.e., at first contact with the health professional) or at first visit in community settings. In individuals screened as having a low risk of developing a pressure injury, the screening should be repeated as soon as the risk exposure has increased or is likely to increase due to changes in the health condition or treatment (e.g., surgery). In individuals screened as being (very likely) at risk, a full pressure injury risk assessment should be undertaken immediately.

Full risk assessment, care planning, and re-assessment

A **full risk assessment** aims to thoroughly examine an individual's risk exposure as indicated by the screening outcome, resulting in either confirmation and nearer qualification of the assumed 'at risk' status and underlying risk factors, or non-confirmation of this assumption. For this in-depth evaluation, all of the major risk factors recommended in this guideline chapter (*Table 4.1, Recommendations 1.1 to 1.19*), as well as consideration to additional condition-specific risk factors, e.g. risk factors for heels (see the guide chapter *Heel Pressure Injuries*) or risk factors associated with devices (see the guide chapter *Device Related Pressure Injuries*) should be taken into account. A risk assessment identifies both modifiable and non-modifiable pressure injury risk factors to provide an overall indication of the individual's risk status. The presence and impact of each factor on the individual's risk exposure should be evaluated by means of deliberate, comprehensive risk assessment methods as recommended below (see *Recommendation 1.23*).

Once individuals are confirmed as being at risk of pressure injury development, a prevention program that aims to minimize the impact of modifiable risk factors identified as increasing the individual's pressure injury risk should be developed. The rationale and measures of care should be explained to and agreed with the individual, and the agreed plan of care should be documented. Although a risk assessment identifies both modifiable and non-modifiable risk factors, prevention interventions only address modifiable risks. An individual's level of pressure injury 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 modified accordingly. If the full assessment does not confirm the 'at risk' assumption indicated by the screening, respective individuals should be screened again if the risk exposure has likely increased or is going to increase due to changes in the health conditions or treatment (e.g. surgery).

Documentation

Accurate documentation of risk assessments and prevention plans 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.¹¹⁵⁻¹¹⁷

1.24: When conducting a pressure injury risk assessment:

- Use a structured approach
- Include a comprehensive skin assessment
- Supplement use of a risk assessment tool with assessment of additional risk factors
- Interpret the assessment outcomes using clinical judgment.

(Good Practice Statement)

Implementation Considerations

- When using a risk assessment tool, select a tool that is appropriate to the population, is valid and is reliable. (*Expert opinion*)
- 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 (*Expert opinion*).

Discussion

As noted in the guideline chapter *Skin and Tissue Assessment*, a comprehensive skin assessment should be part of every risk assessment. Skin and risk assessment are inextricably linked. As noted earlier in this chapter, there is some epidemiological evidence that alterations in skin status, specifically the presence of an existing pressure injury, are associated with development of new pressure injuries, making skin assessment an essential component of any risk assessment. Additionally, pressure injury risk factors such as skin moisture and pain at pressure points can be identified in a skin assessment. Results of a comprehensive skin assessment are also essential in developing an individualized plan for prevention.

Structured approach

There is no universally agreed best approach for conducting a risk assessment; however, expert consensus¹¹⁸ 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 injury 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. However, regardless of how the risk assessment is structured, **clinical judgment is essential**.

Clinical judgment

Regardless of the structured approach used, clinical judgment is a necessary component of any risk assessment. Clinical judgment has been defined as an overarching concept integrating all reasoning tasks and actions performed by health professionals to describe and assess a health condition of interest.¹¹⁹ Related to pressure injury risk assessment, clinical judgment is therefore be viewed in this guideline as the sum of cognitive actions carried out by health professionals to interpret and synthesize information on the health status of individuals in order to derive a diagnosis about their pressure injury risk and needs for prevention.

This reasoning may be undertaken explicitly or implicitly in clinical practice, and it usually comprises various types of health information, irrespective of the source and the methods of data collection. Thus, it can consider information self-reported by the individual or reported by relatives as well as the health professional's own assessment findings and/or that by other professionals. Likewise, it may draw on:

- Natural health metrics (e.g. body mass index, body temperature or laboratory parameters)
- Scales or scores used to quantify and qualify pressure injury risk or single risk factors (e.g., mobility, pain, nutritional status, etc.)
- The professionals' own observation and examination of the individual without use of any tool.

Clinical judgment is a key ability of health professionals. Clinical judgment should be rooted in up-to-date knowledge on the subject of interest (i.e., pressure injury development), careful and repeated diagnostic inquiries using several information sources and methods, team collaboration and constant evaluation and critical reflection of the professionals' performance.

Thus, clinical judgment, as understood in this guideline, is not limited to the pressure injury risk assessment. Instead, it refers to the entirety of the health professional's diagnostic reasoning actions carried out to interpret and integrate available information on an individual's pressure injury risk and is indivisibly inherent to any risk assessment task.

Risk assessment tools

As noted above, 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 inform the clinical judgment. A risk assessment tool is one form of assessment on which a health professional draws when using their clinical judgment.

The majority of the currently available risk assessment tools were developed based on literature review, expert opinion, and/or adaptation of an existing scale. A small number of tools are underpinned by conceptual frameworks.^{8,60,112,120} The three most commonly used scales – the Norton Scale[©] (1962),¹²⁰ Waterlow Score[©] (1985),¹²¹ and the Braden Scale for Predicting Pressure Sore Risk[©] (1987)¹²² – were developed more than 30 years ago without the insight from more recent epidemiological studies. Additionally, numerous lesser known risk tools some of which are designed for use in specific clinical settings and/or patient populations are available, including but not limited to, the Ramstadius Risk Screening Tool, Suriadi and Sanada Scale,⁸ Risk Assessment Pressure Sore Scale,¹²³ The Modified Norton Scale,¹²⁴ the PURPOSE T,¹¹² EVARUCI Scale,^{125,126} COMHON,^{126,127} Perioperative Risk Assessment Measure for Skin (PRAMS),¹²⁸ Spinal Cord Injury Pressure Ulcer Scale (SCIPUS),^{129,130} Braden Q¹³¹ and the Cubbin-Jackson Scale.^{132,133}

Risk assessment tools do not necessarily include assessment of all key factors that can increase the risk of pressure injury development. Specifically, most risk assessment tools do not include an assessment of tissue perfusion or skin status. As presented under *Risk Factors for Pressure injuries* (see above), epidemiological studies identify these factors as strong indicators of pressure injury 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.

Total scores on standardized risk assessment tools (e.g., Braden Scale, Norton Scale and Waterlow Score) provide general information on risk status and level of risk.^{5,11,46,49,65,68,71,77,93,134} Total scores on standardized risk assessment tools

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 (see *Selecting and Using a Risk Assessment Tool* below).

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
- Focus on modifiable risk factors
- Subscale scores that can be used as a basis for risk-based intervention planning
- Clinical reminders (especially for novice nurses)
- A minimum auditable standard.

A meta-analysis conducted by García-Fernández et al. (2014)¹³⁵ reported relatively poor pooled predictive capacity indicators for clinical judgment alone as measured by relative risk. For clinical judgment, relative risk was 1.95 (95% confidence interval [CI] 0.94 to 4.04). In comparison the relative risk for risk assessment tools ranged between relative risk of 2.66 to 8.63 (see *Table 4.18*). 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. In this review, the definition and description of clinical judgment is lacking, and the process used for risk assessment that did not include a risk assessment tool is not precisely clear.

Moore and Patton (2019)¹³⁶ conducted a systematic review to determine if using structured systematic pressure injury risk assessment tools reduced the incidence of pressure injuries. Finding only two studies meeting their inclusion criteria,^{87,137} they concluded that the low, or very low certainty of evidence available from the included studies is not sufficiently reliable to suggest that the use of structured and systematic pressure injury risk assessment tools reduces the incidence, or severity of pressure injuries. As previously discussed, an assessment tool alone cannot reduce the incidence of pressure injuries; use of the risk assessment tool to develop and implement risk-based preventive interventions is an essential step in achieving a positive outcome. In this review, clinical judgment was considered to be a health professional's judgment made with no aid of a risk assessment tool, which differs from the definition of clinical judgment used throughout this clinical guideline.

One of the trials included in the above review was a large, blinded randomized controlled trial (RCT) conducted by Webster et al. (2011)⁸⁷ that compared use of a Waterlow Score (n = 410), the Ramstadius tool (n = 411) and risk assessment based on the nurse's clinical judgment (n = 410) for reducing pressure injury occurrence in participants located in medical and oncology wards. Based on the data from the study by Webster et al. (2011),⁸⁷ Moore and Patton (2019)¹³⁶ determined that risk assessment using the Waterlow Score may make little or no difference to pressure injury incidence, or to pressure injury severity, when compared to risk assessment using clinical judgment alone (pressure injuries of all stages: RR 1.10, 95% CI 0.68 to 1.81; 821 participants), or risk assessment using the Ramstadius tool (pressure injuries of all stages: RR 1.41, 95% CI 0.83 to 2.39; 821 participants). Similarly, risk assessment using the Ramstadius tool may make little or no difference to pressure injury incidence, or to pressure injury severity, when compared to risk assessment using clinical judgment (pressure injuries of all stages: RR 0.79, 95% CI 0.46 to 1.35; 820 participants). Moore and Patton (2019)¹³⁶ assessed the certainty of this evidence as low due to methodological limitations and lack of precision (*Level 1*).

In the second of the trials reported in the systematic review¹³⁶, Saleh et al. (2009)¹³⁷ conducted a cluster RCT in a military hospital. Participants were at risk of pressure injuries (Braden Scale score ≤ 18). The trial, which had three groups, compared the use of the Braden Scale (group A; n = 74); risk assessment based on clinical judgment of a nurse who had received education on the Braden Scale (group B; n = 76) and clinical judgment without accompanying education (group C; n = 74). Based on the result of this study, Moore and Patton (2019)¹³⁶ concluded that it was uncertain whether use of the Braden Scale makes any difference to pressure injury incidence, compared to risk assessment using clinical judgment and training (risk ratio (RR) 0.97, 95% confidence interval (CI) 0.53 to 1.77; 150 participants), or compared to risk assessment using clinical judgment alone (RR 1.43, 95% CI 0.77 to 2.68; 180 participants). However, this study was also considered to provide very low certainty of the evidence due to methodological limitations and lack of precision¹³⁶ (*Level 1*).

An additional clinical trial designed primarily to assess the effectiveness of different repositioning regimens reported on different strategies to assess pressure injury risk. Participants (n = 1,772) were assessed using the Norton Scale,

the Braden Scale and by nurses using their own clinical judgment.⁴¹ Sensitivity of clinical judgment was 25% to 28% lower than assessment using the risk assessment tools. However, specificity was 20% to 30% higher with clinical judgment. Fewer individuals who developed a pressure injury were identified as being at risk when clinical judgment was used, but of those individuals identified at risk, more actually developed a pressure injury. The two risk assessment tools were essentially equivalent in predicting development of pressure injuries. Education background and clinical experience of the nurses participating in the study were not reported (*Level 1*).

There are limitations to the current research that prevent a clear comparison between risk assessment tools and clinical judgment alone.¹³⁶ 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 injury incidence, confounding the evaluation of the risk assessment strategy. Defloor et al. (2005)¹³⁸ highlight that development of a pressure injury 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)¹³⁷ and Defloor et al. (2005)¹³⁸, there was non-equivalent use of pressure injury prevention strategies between individuals identified at risk and not at risk (in particular, the types of support surfaces used) and this confounded the findings. In the study conducted by Webster et al. (2011)⁸⁷, non-significant differences in pressure injury prevention interventions initiated following the risk assessment is reported.

Selecting and Using a Risk Assessment Tool

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 injuries. Many risk assessment tools do not incorporate these advances in knowledge.

Total Braden Scale scores^{46,49,65,68,71,73,75,77,82,93} (*Levels 1 and 3*) Norton Scale scores^{5,100} (*Levels 1 and 3*), Waterlow Score⁵¹ (*Level 3*) and scores on the Cubbin-Jackson Scale⁷⁴ (*Level 3*) have emerged as statistically significant factors in some multivariable models (see Table 4.16). Pressure injury incidence progressively increased with increasing level of risk based on Braden Scale total scores in a number of studies.^{11,134} However, in other Level 1 and 3 studies commonly used risk assessment tools were not significant in predicting the development of a pressure injury. Total scores may not always emerge as significant factors in multivariable models; however, the subscale scores identifying specific risk factors (e.g., mobility, activity, moisture etc.) have provided support for analysis of individual risk factors, as previously discussed in this chapter.

As discussed, appropriate application of a risk tool requires the findings to inform the development and implementation of a risk prevention plan, and this likely influences the results of multivariable modelling. Other factors, including the knowledge and experience of the health professional using the tool, may also contribute to the mixed findings on significance of risk assessment tools in predicting pressure injuries.

Table 4.16: Summary of evidence for risk assessment tools

Risk factor variables	Percent studies significant in multivariable model	Risk factor significant and non-significant in multivariable model
Braden Scale score	39% (11 of 28 studies)	11 studies in which risk factors were significant in the model ^{49,57,65,68,71,73,75,77,82,93,97} 17 studies in which risk factors were not significant in the model ^{11,19-21,23,31,36,39,47,48,58,78,81,86,89,98,99}
Norton Scale score	66.6% (2 of 3 studies)	2 studies in which risk factors were significant in the model ^{5,100} 1 study in which risk factors were not significant in the model ⁶⁵
Waterlow Score	33.3% (1 of 3 studies)	1 study in which risk factors were significant in the model ⁵¹ 2 studies in which risk factors were not significant in the model ^{17,67}
Cubbin-Jackson Scale score	100% (1 of 1 study)	1 study in which risk factors were significant in the model ⁷⁴
Other risk scale scores	83.3% (5 of 6 studies)	4 studies in which risk factors were significant in the model ^{5,50,53,88} 1 study in which risk factors were not significant in the model ²²

When using a risk assessment tool, also consider the risk factors not measured by the tool. For example, the Braden Scale subscale scores address risk factors related to mobility, activity, friction and shear, nutrition, moisture and sensory perception. Many facilities use the subscale scores to identify modifiable risk factors as a basis for risk-based prevention planning. However, a comprehensive risk assessment should also include risk factors (both modifiable and non-modifiable) that are not represented in risk assessment tool. In the case of the Braden Scale, this would also include consideration of skin status, diabetes, perfusion and oxygenation, increased body temperature, advanced age, relevant laboratory blood tests and general health status.¹³⁹ *Table 4.17* compares risk factors supported by current epidemiological studies (as presented above under *Risk Factors for Pressure Injuries*) with the risk factors that are included on commonly used risk assessment tools and those for specific populations. The *Table* identifies gaps in the risk assessment that health professionals should be aware of when using these risk tools, indicating factors that should be considered in a risk assessment in addition to those included on the tool.

Psychometric Properties of Risk Assessment Tools

Reliability

Reliability refers to the consistency and reproducibility of scores when performed by different reviewers. Reliability is widely regarded as a necessary condition for validity. There are a large number of studies that specifically address the interrater reliability of risk assessment tools and reports of early tool development usually contain some measure of reliability. Reported measures typically are a kappa value or intraclass correlation coefficient. 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)¹²⁴ and Braden Scale (ICC range = 0.72 to 0.95).¹⁴⁰⁻¹⁴⁴ Interrater reliability for the Waterlow Score was reported as 0.36 (95% CI 0.09 to 0.63) in one study.¹⁴⁰ Interrater reliability for subscale scores varied depending on the subscale and the clarity of the operational definition.^{124,140-144} 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 injury development).

A major problem identified in the literature⁴¹ 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 operating characteristic (AUROC) curves (as an indication of discrimination or the best balance between sensitivity and specificity)
- 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 4.18* summarizes the estimates from meta-analyses^{118,135} or when unavailable, the largest most recent studies,^{130,145} for commonly used risk assessment tools and tools for specific population. Most risk assessment tools have received minimal psychometric testing.

Table 4.17: Comparison of risk factors identified in epidemiological studies and commonly used risk assessment tools

Note: An asterisk (*) indicates that the actual risk tool subscale was significant in multivariable modeling in one or more epidemiological study. 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.

Epidemiological study risk factors	Braden Scale ¹⁴⁶	Norton Scale ¹⁴⁷	Waterlow Score ¹⁴⁸	Cubbin-Jackson Scale ¹⁴⁹ (critically ill individuals)	SCIPUS ¹⁵⁰ (individuals with SCI)	Braden Q Scale ¹⁵¹ (children)
Activity and mobility limitations	<ul style="list-style-type: none"> • Mobility* • Activity* • Friction-shear* 	<ul style="list-style-type: none"> • Mobility* • Activity* 	Mobility	<ul style="list-style-type: none"> • Mobility • Hygiene 	<ul style="list-style-type: none"> • Mobility • Level of activity • Complete SCI • Autonomic dysreflexia/ severe spasticity 	<ul style="list-style-type: none"> • Mobility* • Activity* • Friction-shear*
Skin status	Not included	Not included	Skin type (in visual areas, partial measure of skin status)	General skin condition	Not included	Not included
Diabetes	Not included	Not included	Not included	Not included	Blood glucose levels	Not included
Perfusion and oxygenation	Not included	Not included	Special Risk (partial measure of perfusion)	<ul style="list-style-type: none"> • Oxygen requirements • Respiration • Hemodynamics 	<ul style="list-style-type: none"> • Tobacco use • Cardiac disease 	<ul style="list-style-type: none"> • Tissue perfusion oxygenation
Poor nutritional status	Nutrition	<ul style="list-style-type: none"> • Food intake • Fluid intake (modified scale) 	<ul style="list-style-type: none"> • Appetite • Build (weight for height) 	<ul style="list-style-type: none"> • Weight/tissue viability • Nutrition 	Not included	Nutrition
Increased skin moisture	Moisture*	Incontinence	Continence	Incontinence	Urine incontinence or constant moistness	Moisture*
Increased body temperature	Not included	Not included	Not included	Not included	Not included	Not included
Advanced age	Not included	Not included	Gender/Age	Age	Age	Not included
Sensory perception	Sensory perception*	Not included	Neurological Deficit	Not included	Not included	Sensory perception*
Abnormal laboratory blood results	Not included	Not included	Not included	Not included	<ul style="list-style-type: none"> • Albumin • Hematocrit 	<ul style="list-style-type: none"> • Not included
General health status	Not included	<ul style="list-style-type: none"> • Physical condition • Mental condition* 	<ul style="list-style-type: none"> • Major Surgery/Trauma • Medications 	<ul style="list-style-type: none"> • Mental condition • Past medical condition 	<ul style="list-style-type: none"> • Respiratory disease • Renal disease • Impaired cognitive function 	<ul style="list-style-type: none"> • Not included

Table 4.18: Psychometric qualities of major risk assessment tools

Scales (cut-off)	Sensitivity Median (range)	Specificity Median (range)	Positive likelihood ratio	Negative likelihood ratio	AUROC Median (range)	Relative Risk (95% CI)
Braden (≤ 18) ^{118,135}	0.74 ^a (0.33 to 1)	0.68 ^a (0.34 to 0.86)	2.31 ^a	0.38 ^a	0.77 ^b (0.55 to 0.88)	4.26 ^f (3.27 to 5.55)
Norton (≤ 14) ^{118,135}	0.75 ^c (0 to 0.89)	0.68 ^c (0.59 to 0.95)	2.34 ^c	0.37 ^c	0.74 ^c (0.56 to 0.75)	3.69 ^g (2.64 to 5.16)
Waterlow (≥ 10) ^{118,135}	1.00, 0.88 ^d	0.13, 0.29 ^d	1.15, 1.24 ^d	0.0, 0.41 ^d	0.61 ^e (0.54 to 0.66)	2.66 ^h (1.76 to 4.01)
Cubbin-Jackson (≤ 24) ^{135,145}	0.72 ^j	0.68 ^j	—	—	0.763 ^j	8.63 ^k (3.02 to 24.66)
SCIPUS (≥ 8) ¹³⁰	0.85 ^m	0.38 ^m	1.4 ^m	—	0.64 ^m (0.59 to 0.70)	—
Braden Q (≤ 13) ¹⁵²	0.86 ^p (0.76 to 0.96)	0.59 ^p (0.55 to 0.63)	2.09 ^p (0.95 to 4.58)	—	0.72 ^p (0.76 to 0.78)	—
	^a 16 studies, n=5,462 ^d 2 studies, n=419 ^g 15 studies, n=4,935 ^k 2 studies, n=151		^b 7 studies, n=4,811 ^e 4 studies, n=2,559 ^h 12 studies, n=2,408 ^m 1 study (n=759)		^c 5 studies, n=2,809 ^f 31 studies, n=7,137 ^j 1 study, n=829 ^p 1 study, n=625	

Comparison of Risk Tools

The systematic comparative effectiveness review completed by Chou et al. (2013)¹¹⁸ compared the ability of different risk assessment tools in predicting pressure injury incidence. Fourteen studies that directly compared two or more risk assessment tools in the same population were identified. Six studies^{3,33,41,153-155} reported that the AUROCs within each study were comparable. AUROCs ranged between 0.66 and 0.90 with the exception of one study¹⁵³ in which AUROCs ranged between 0.55 and 0.61 which is slightly better than chance (0.50). A high AUROC indicates that the risk assessment tool is better able to discriminate among individuals who will and those who will not develop a pressure injury.

Seven studies^{33,41,153,154,156-158} 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 injuries 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.¹⁵⁹ 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.

Risk Assessment Based on Decision Support Systems

Data from electronic health records (EHRs) have been used over the past decade to identify risk factors for pressure injury development. More recently, advances in data mining, machine learning and Bayesian networking strategies have facilitated the analysis of multiple risk factors in thousands of patients to develop more sophisticated models for predicting pressure injuries.¹⁶⁰⁻¹⁶⁵ After testing and validation, these predictive models can be integrated into EHRs to provide real-time analysis of patient-level data to screen for pressure injury risk status. Advantages of these decision support systems include:

- Ability to analyze a greater number of risk factors
- Ability to use data already available in the EHR

- Real-time analysis of risk status on admission and when the individual's clinical condition changes
- Potentially improved discrimination of risk status in high risk populations than possible with general risk assessment scales.¹⁶⁵

As with all risk assessment methods, clinical judgment is critical. Risk factors that are statistically significant in a computer-generated model may not be clinically significant. The algorithms used in the design of such systems should be explicit or periodically validated in the case of machine learning. There should be a plausible physiological link between risk factors identified in the model and what is known about the etiology of pressure injuries. Consideration should be given to how the analysis is presented to the clinician and how it integrates with clinical workflow. A global risk score is of limited benefit. Modifiable risk factors should be identified as a basis for planning preventive interventions.

Conclusions

Amid these discussions, one must remember that 'prediction is not destiny'. The outcome for an at-risk individual can often be altered by carefully selecting and consistently implementing risk-based prevention strategies. Although the best method for identifying risk has not been determined, the available evidence presented above provides a guide to clinical decision making.

Risk assessment tools provide a structure to guide a pressure injury risk assessment. Currently there is no single risk assessment tool that reflects all relevant risk factors, and the true predictive validity of tools is unclear. Thus, total and subscale scores provide limited information about risk factors. The degree of risk exposure for a specific individual must be interpreted in light of the clinical judgment of the individual's medical condition and history.

Having a strong understanding of relevant pressure injury risk factors is crucial to identifying individuals who are at risk. The risk assessment must draw on various information sources and assessment methods, including standardised measurements (when available), to capture all individually relevant risk factors. The health professional must evaluate all the available data to make the best possible clinical judgment on those risk factors that are of significance to the individual, and then develop an individualized pressure injury prevention plan to address those factors that are modifiable. Addressing these factors using the evidence-based interventions outlined throughout this guideline is key to effective pressure injury prevention.

References

1. Oomens CWJ, A Mixture Approach to the Mechanics of Skin and Subcutis - a Contribution to Pressure Sore Research, in Pesis 1985, University of Twente: Enschede, The Netherlands.
2. Coleman S, Gorecki C, Nelson EA, Closs SJ, Defloor T, Halfens R, Farrin A, Brown J, Schoonhoven L, Nixon J. Patient risk factors for pressure ulcer development: Systematic review. *Int J Nurs Stud*, 2013.
3. Perneger T, Gaspoz J, Borst F, Vitek O. Screening for pressure ulcer risk in an acute care hospital: development of a brief bedside scale. *J Clin Epidemiol*, 2002; 55(5): 498-504.
4. Nijs N, Toppets A, Defloor T, Bernaerts K, Milisen K. Incidence and risk factors for pressure ulcers in the intensive care unit. *J Clin Nurs*, 2009; 18(9): 1258-66.
5. Bourdel-Marchasson I, Barateau M, Rondeau V, Dequae-Merchadou L, Salles-Montaudon N, Emeriau J-P, Manciet G, Dartigues J-F, Group ftG. A multi-center trial of the effects of oral nutritional supplementation in critically ill older inpatients. *Nutrition*, 2000; 16: 1-5.
6. Baumgarten M, Margolis D, van DC, Gruber-Baldini A, Hebel J, Zimmerman S, Magaziner J. Black/White differences in pressure ulcer incidence in nursing home residents. *J Am Geriatr Soc*, 2004; 52(8): 1293-1298.
7. De Laat E, Pickkers P, Schoonhoven L, Verbeek A, Feuth T, Van Achterberg T. Guideline implementation results in a decrease of pressure ulcer incidence in critically ill patients. *Crit Care Med*, 2007; 35(3): 815-820.
8. Suriadi, Sanada H, Sugama J, Thigpen B, Subuh M. Development of a new risk assessment scale for predicting pressure ulcers in an intensive care unit. *Nurs Crit Care*, 2008; 13(1): 34.
9. Brandeis G, Ooi W, Hossain M, Morris J, Lipsitz L. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *Journal of the American Geriatric Society*, 1994; 42: 388-93.
10. Ooi W, Morris J, Brandeis G, Hossain M, Lipsitz L. Nursing home characteristics and the development of pressure sores and disruptive behaviour. *Age Ageing*, 1999; 28(1): 45-52.
11. Tescher AN, Branda ME, Byrne TJ, Naessens JM. All at-risk patients are not created equal: Analysis of Braden pressure ulcer risk scores to identify specific risks. *J Wound Ostomy Continence Nurs*, 2012; 39(3): 282-291.
12. Rademakers L, Vainas T, van Zutphen S, Brink P, van Helden S. Pressure ulcers and prolonged hospital stay in hip fracture patients affected by time-to-surgery. *Eur J Trauma Emerg Surg*, 2007; 33(3): 238-44.
13. Salzberg C, Byrne D, Kabir R, van Niewerburg P, Cayten C. Predicting pressure ulcers during initial hospitalization for acute spinal cord injury. *Wounds*, 1999; 11(2): 45-57.
14. Chiari P, Forni C, Guberti M, Gazineo D, Ronzoni S, D'Alessandro F. Predictive factors for pressure ulcers in an older adult population hospitalized for hip fractures: A prognostic cohort study. *PLoS ONE [Electronic Resource]*, 2017; 12(1): e0169909.

15. Gonzalez-Mendez MI, Lima-Serrano M, Martin-Castano C, Alonso-Araujo I, Lima-Rodriguez JS. Incidence and risk factors associated with the development of pressure ulcers in an intensive care unit. *J Clin Nurs*, 2018; 27(5-6): 1028-1037.
16. Rose P, Cohen R, Amsel R. Development of a scale to measure the risk of skin breakdown in critically ill patients. *Am J Crit Care*, 2006; 15(3): 337-41.
17. Sayar S, Turgut S, Dogan H, Ekici A, Yurtsever S, Demirkan F, Doruk N, Tasdelen B. Incidence of pressure ulcers in intensive care unit patients at risk according to the Waterlow scale and factors influencing the development of pressure ulcers. *J Clin Nurs*, 2009; 18(5): 765-774.
18. Bergquist-Beringer S, Gajewski BJ. Outcome and assessment information set data that predict pressure ulcer development in older adult home health patients. *Adv Skin Wound Care*, 2011; 24(9): 404-414.
19. Baldwin K, Ziegler S. Pressure ulcer risk following critical traumatic injury. *Adv Wound Care*, 1998; 11(4): 168-73.
20. Bergquist S, Frantz R. Pressure ulcers in community-based older adults receiving home health care. Prevalence, incidence, and associated risk factors. *Adv Wound Care*, 1999; 12(7): 339.
21. Kemp M, Kopanke D, Tordecilla L, Fogg L, Shott S, Matthiesen V, Johnson B. The role of support surfaces and patient attributes in preventing pressure ulcers in elderly patients. *Res Nurs Health*, 1993; 16(2): 89-96.
22. Lindgren M, Unosson M, Fredrikson M, Ek A. Immobility: A major risk factor for development of pressure ulcers among adult hospitalized patients: a prospective study. *Scand J Caring Sci*, 2004; 18(1): 57-64.
23. Watts D, Abrahams E, MacMillan C, Sanat J, Silver R, VanGorder S, Waller M, York D. Insult after injury: Pressure ulcers in trauma patients. *Orthop Nurs*, 1998; 17(4): 84-91.
24. Ek A. Prediction of pressure sore development. *Scandinavian Journal of Caring Sciences*, 1987; 1(2): 77-84.
25. Ek AC, Unosson M, Larsson J, von Schenck H, Bjurulf P. The development and healing of pressure sores related to the nutritional state. *Clin Nutr*, 1991; 10(5): 245-250.
26. Allman R, Goode P, Patrick M, Burst N, Bartolucci A. Pressure ulcer risk factors among hospitalized patients with activity limitation. *J Am Med Assoc*, 1995; 273(11): 865-70.
27. Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. *J Am Geriatr Soc*, 1989; 37(11): 1043-1050.
28. Kwong EW-y, Pang SM-c, Aboo GH, Law SS-m. Pressure ulcer development in older residents in nursing homes: Influencing factors. *J Adv Nurs*, 2009; 65(12): 2608-2620.
29. Okuwa M, Sanada H, Sugama J, Inagaki M, Konya C, Kitagawa A, Tabata K. A prospective cohort study of lower-extremity pressure ulcer risk among bedfast older adults. *Adv Skin Wound Care*, 2006; 19(7): 391-39.
30. Schnelle JF, Adamson GM, Cruise PA, Al-Samarrai N, Sarbaugh FC, Uman G, Ouslander JG. Skin disorders and moisture in incontinent nursing home residents: Intervention implications. *J Am Geriatr Soc*, 1997; 45(10): 1182-1188.
31. Tayyib N, Coyer F, Lewis P. Saudi Arabian adult intensive care unit pressure ulcer incidence and risk factors: a prospective cohort study. *Int Wound J*, 2015.
32. Olson B, Langemo D, Burd C, Hanson D, Hunter S, Cathcart-Silberberg T. Pressure ulcer incidence in an acute care setting. *J Wound Ostomy Continence Nurs*, 1996; 23(1): 15-20.
33. Boyle M, Green M. Pressure sores in intensive care: defining their incidence and associated factors and assessing the utility of two pressure sore risk assessment tools. *Aust Crit Care*, 2001; 14(1): 24-30.
34. Vanderwee K, Grypdonck M, Bacquer D, Defloor T. The identification of older nursing home residents vulnerable for deterioration of grade 1 pressure ulcers. *J Clin Nurs*, 2009; 18(21): 3050-58.
35. Halfens R, Van Achterberg T, Bal RM. Validity and reliability of the Braden scale and the influence of other risk factors: a multicentre prospective study. *Int J Nurs Stud*, 2000; 37(4): 313-319.
36. Tourtual D, Riesenber L, Korutz C, Semo A, Asef A, Talati K, Gill R. Predictors of hospital acquired heel pressure ulcers. *Ostomy Wound Management*, 1997; 43(9): 24-34.
37. Suriadi, Sanada H, Sugama J, Kitagawa A, Thigpen B, Kinoshita S, Murayama S. Risk factors in the development of pressure ulcers in an intensive care unit in Pontianak, Indonesia. *Int Wound J*, 2007; 4(3): 208-15.
38. Joseph C, Nilsson Wikmar L. Prevalence of secondary medical complications and risk factors for pressure ulcers after traumatic spinal cord injury during acute care in South Africa. *Spinal Cord*, 2016; 54(7): 535-9.
39. Lin S, Hey HWD, Lau ETC, Tan KA, Thambiah JS, Lau LL, Kumar N, Liu KPG, Wong HK. Prevalence and predictors of pressure injuries from spine surgery in the prone position. *Spine (Phila Pa 1976)*, 2017; 42(22): 1730-1736.
40. Brienza D, Krishnan S, Karg P, Sowa G, Allegretti AL. Predictors of pressure ulcer incidence following traumatic spinal cord injury: a secondary analysis of a prospective longitudinal study. *Spinal Cord*, 2017; 12: 12.
41. Defloor T, Grypdonck M. Pressure ulcers: Validation of two risk assessment scales. *J Clin Nurs*, 2005; 14(3): 373-82.
42. Nixon J, Nelson EA, Cranny G, Iglesias CP, Hawkins K, Cullum NA, Phillips A, Spilsbury K, Torgerson DJ, Mason S, PRESSURE Trial Group. Pressure relieving support surfaces: a randomised evaluation. *Health Technol Assess*, 2006; 10(22): iii-x, 1.
43. Van Der Wielen H, Post MWM, Lay V, Glasche K, Scheel-Sailer A. Hospital-acquired pressure ulcers in spinal cord injured patients: Time to occur, time until closure and risk factors. *Spinal Cord*, 2016; 54(9): 726-731.
44. Baumgarten M, Rich S, Shardell M, Hawkes W, Margolis D, Langenberg P, Orwig D, Palmer M, Jones P, Sterling R, Kinoshian B, Magaziner J. Care-related risk factors for hospital-acquired pressure ulcers in elderly adults with hip fracture. *J Am Geriatr Soc*, 2012; 60(2): 277-283.
45. Bostrom J, Mechanic J, Lazar N, Michelson S, Grant L, Nomura L. Preventing skin breakdown: nursing practices, costs, and outcomes. *Appl Nurs Res*, 1996; 9(4): 184-188.
46. Chan WS, Pang SMC, Kwong EWY. Assessing predictive validity of the modified Braden Scale for prediction of pressure ulcer risk of orthopaedic patients in an acute care setting. *J Clin Nurs*, 2009; 18(11): 1565-1573.

47. Demarre L, Verhaeghe S, Van Hecke A, Clays E, Grypdonck M, Beeckman D. Factors predicting the development of pressure ulcers in an at-risk population who receive standardized preventive care: Secondary analyses of a multicentre randomised controlled trial. *J Adv Nurs*, 2015; 71(2): 391-403.
48. Donnelly J, A randomised controlled trial comparing the Heelift Suspension Boot with standard care in the prevention of pressure ulcers on the heels of older people with fractured hips. Thesis dissertation. 2006, University of Belfast. .
49. Fife C, Otto G, Capsuto E, Brandt K, Lyssy K, Murphy K, Short C. Incidence of pressure ulcers in a neurologic intensive care unit. *Crit Care Med*, 2001; 29(2): 283-90.
50. Pancorbo Hidalgo P, Garcia Fernandez F. Risk factors for the development of pressure ulcers among hospitalized elderly patients. *Gerokomos*, 2001; 12(4): 175-84.
51. Sternal D, Wilczynski K, Szewieczek J. Pressure ulcers in palliative ward patients: Hyponatremia and low blood pressure as indicators of risk. *Clin Interv Aging*, 2017; 12: 37-44.
52. Yoshimura M, Nakagami G, Iizaka S, Yoshida M, Uehata Y, Kohno M, Kasuya Y, Mae T, Yamasaki T, Sanada H. Microclimate is an independent risk factor for the development of intraoperatively acquired pressure ulcers in the park-bench position: A prospective observational study. *Wound Repair Regen*, 2015; 23(6): 939-947.
53. Roca-Biosca A, Velasco-Guillen M, Rubio-Rico L, Garcia-Grau N, Anguera-Saperas L. [Pressure ulcers in the critical patient: Detection of risk factors]. *Enferm Intensiva*, 2012 23(4): 155-163.
54. Schoonhoven L, Defloor T, van der Tweel I, Buskens E, Grypdonck MH. Risk indicators for pressure ulcers during surgery. *Appl Nurs Res*, 2002; 15(3): 163-173.
55. Yoshimura M, Iizaka S, Kohno M, Nagata O, Yamasaki T, Mae T, Haruyama N, Sanada H. Risk factors associated with intraoperatively acquired pressure ulcers in the park-bench position: A retrospective study. *International Wound Journal*, 2015.
56. Chen HL, Shen WQ, Xu YH, Zhang Q, Wu J. Perioperative corticosteroids administration as a risk factor for pressure ulcers in cardiovascular surgical patients: A retrospective study. *Int Wound J*, 2013.
57. Tschannen D, Bates O, Talsma A, Guo Y. Patient-specific and surgical characteristics in the development of pressure ulcers. *Am J Crit Care*, 2012; 21(2): 116-126.
58. Connor T, Sledge JA, Bryant-Wiersema L, Stamm L, Potter P. Identification of pre-operative and intra-operative variables predictive of pressure ulcer development in patients undergoing urologic surgical procedures. *Urol Nurs*, 2010; 30(5): 289-305.
59. World Health Organization, Towards a common language for functioning, disability and health: The international classification of functioning, disability and health. 2002, WHO: Geneva.
60. Braden B, Bergstrom N. A conceptual schema for the study of the etiology of pressure sores. *Rehabil Nurs*, 1987; 12(1): 8-12.
61. Smith IL, Brown S, McGinnis E, Briggs M, Coleman S, Dealey C, Muir D, Nelson EA, Stevenson R, Stubbs N, Wilson L, Brown JM, Nixon J. Exploring the role of pain as an early predictor of category 2 pressure ulcers: A prospective cohort study. *BMJ Open*, 2017; 7(1): e013623.
62. Reed R, Hepburn K, Adelson R, Center B, McKnight P. Low serum albumin levels, confusion, and fecal incontinence: Are these risk factors for pressure ulcers in mobility-impaired hospitalized adults? *Gerontology*, 2003; 49(4): 255-59.
63. Nixon J, Cranny G, Bond S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: A cohort study. *Int J Nurs Stud*, 2007; 44(5): 655-63.
64. Man Sp, Au-Yeung Tw. Hypotension is a risk factor for new pressure ulcer occurrence in older patients after admission to an acute hospital. *J Am Med Dir Assoc*, 2013.
65. Stordeur S, Laurent S, W. DH. The importance of repeated risk assessment for pressure sores in cardiovascular surgery. *J Cardiovasc Surg (Torino)*, 1998; 39(3): 343-9.
66. Marchette L, Arnell I, Redick E. Skin ulcers of elderly surgical patients in critical care units. *Dimens Crit Care Nurs*, 1991; 10(6): 321-29.
67. Compton F, Hoffmann F, Hortig T, Strauss M, Frey J, Zidek W, Schafer JH. Pressure ulcer predictors in ICU patients: Nursing skin assessment versus objective parameters. *J Wound Care*, 2008; 17(10): 417.
68. Bates-Jensen B, McCreath H, Kono A, Apeles N, Alessi C. Subepidermal moisture predicts erythema and stage 1 pressure ulcers in nursing home residents: a pilot study. *J Am Geriatr Soc*, 2007; 55(8): 1199-1205.
69. Tew C, Hettrick H, Holden-Mount S, Grigsby R, Rhodovi J, Moore L, Ghaznavi AM, Siddiqui A. Recurring pressure ulcers: Identifying the definitions. A National Pressure Ulcer Advisory Panel white paper. *Wound Repair Regen*, 2014; 22(3): 301-304.
70. de Souza D, de Gouveia SV. Incidence of pressure ulcers in the institutionalized elderly. *Journal of Wound, Ostomy, and Continence Nursing: Official Publication of the Wound, Ostomy and Continence Nurses Society / WOCN*, 2010; 37(3): 272-276.
71. Schultz AA, Bien M, Dumond K, Brown K, Myers A. Etiology and incidence of pressure ulcers in surgical patients. *AORN J*, 1999; 70(3): 434-449.
72. Nassaji M, Askari Z, Ghorbani R. Cigarette smoking and risk of pressure ulcer in adult intensive care unit patients. *Int J Nurs Pract*, 2014; 20(4): 418-23.
73. Ranzani OT, Simpson ES, Japiassu AM, Noritomi DT, Amil Critical Care G. The challenge of predicting pressure ulcers in critically ill patients. A multicenter cohort study. *Annals of the American Thoracic Society*, 2016; 13(10): 1775-1783.
74. Apostolopoulou E, Tselebis A, Terzis K, Kamarinou E, Lambropoulos I, Kalliakmanis A. Pressure ulcer incidence and risk factors in ventilated intensive care patients. *Health Science Journal*, 2014; 8(3): 333-342.
75. Chen HL, Zhu B, Wei R, Zhou ZY. A retrospective analysis to evaluate seasonal pressure injury incidence differences among hip fracture patients in a tertiary hospital in East China. *Ostomy Wound Management*, 2018; 64(2): 40-44.
76. Feuchtinger J, de Bie R, Dassen T, Halfens R. A 4-cm thermoactive viscoelastic foam pad on the operating room table to prevent pressure ulcer during cardiac surgery. *Journal of Clinical Nursing*, 2006; 15(2): 162-167.

77. Bergstrom N, Braden B. A prospective study of pressure sore risk among institutionalized elderly. *J Am Geriatr Soc*, 1992; 40(8): 747-58.
78. Cox J, Roche S. Vasopressors and development of pressure ulcers in adult critical care patients. *Am J Crit Care*, 2015; 24(6): 501-510.
79. Cobb G, Yoder L, Warren J. Pressure ulcers: Patient outcomes on a KinAir Bed or EHOB Waffle mattress. 1997, TriService Nursing Research Program (TSNRP).
80. Bly D, Schallom M, Sona C, Klinkenberg D. A model of pressure, oxygenation, and perfusion risk factors for pressure ulcers in the intensive care unit. *Am J Crit Care*, 2016; 25(2): 156-164.
81. Borghardt AT, Prado TN, Bicudo SD, Castro DS, Bringuento ME. Pressure ulcers in critically ill patients: incidence and associated factors. *Rev Bras Enferm*, 2016; 69(3): 460-467.
82. Matozinhos FP, Velasquez-Melendez G, Tiensoli SD, Moreira AD, Gomes FSL. Factors associated with the incidence of pressure ulcer during hospital stay. *Rev Esc Enferm USP*, 2017; 51: e03223.
83. Ham HW, Schoonhoven LL, Schuurmans MM, Leenen LL. Pressure ulcer development in trauma patients with suspected spinal injury: The influence of risk factors present in the emergency department. *Int Emerg Nurs*, 2017; 30: 13-19.
84. Manzano F, Navarro MJ, Roldán D, Moral MA, Leyva I, Guerrero C, Sanchez MA, Colmenero M, Fernández-Mondejar E. Pressure ulcer incidence and risk factors in ventilated intensive care patients. *J Crit Care*, 2010; 25(3): 469-476.
85. Dhandapani M, Dhandapani S, Agarwal M, Mahapatra AK. Pressure ulcer in patients with severe traumatic brain injury: Significant factors and association with neurological outcome. *J Clin Nurs*, 2014; 23(7-8): 1114-1119.
86. Serpa L, Santos V. Assessment of the nutritional risk for pressure ulcer development through Braden scale. *Journal of Wound Ostomy and Continence Nursing*, 2007; 34(3S): S4-S6.
87. Webster J, Coleman K, Mudge A, Marquart L, Gardner G, Stankiewicz M, Kirby J, Vellacott C, Horton-Breshears M, McClymont A. Pressure ulcers: Effectiveness of risk-assessment tools. A randomised controlled trial (the ULCER trial). *British Medical Journal Quality & Safety*, 2011; 20(4): 297-306.
88. Inman KJ, Dymock K, Fysh N, Robbins B, Rutledge FS, Sibbald WJ. Pressure ulcer prevention: a randomized controlled trial of 2 risk-directed strategies for patient surface assignment. *Adv Wound Care*, 1999; 12(2): 72-80.
89. Yepes D, Molina F, Leon W, Perez E. Incidence and risk factors associated with the presence of pressure ulcers in critically ill patients. *Med Intensiva*, 2009; 33(6): 276.
90. Vilhena L, Ramalho A. Friction of human skin against different fabrics for medical use. *Lubricants*, 2016 4(1): doi.org/10.3390/lubricants4010006
91. Klaassen M, Schipper D, Masen M. Influence of the relative humidity and the temperature on the in-vivo friction behaviour of human skin. *Biotribology*, 2016; 6: 21-28.
92. Bernatchez SF, Mengistu GE, Ekholm BP, Sanghi S, Theiss SD. Reducing Friction on Skin at Risk: The Use of 3MTM Cavilon™ No Sting Barrier Film. *Adv Wound Care*, 2015; 4(12): 705-710.
93. Bergstrom N, Braden B, Kemp M, Champagne M, Ruby E. Multi-site study of incidence of pressure ulcers and the relationship between risk level, demographic characteristics, diagnoses, and prescription of preventive interventions. *J Am Diet Assoc*, 1996; 44(1): 22-30.
94. Gunningberg L, Lindholm C, Carlsson M, Sjoden P. Reduced incidence of pressure ulcers in patients with hip fractures: A 2-year follow-up of quality indicators. *International Journal for Quality Health Care* 2001; 13(5): 399-407.
95. Hatanaka N, Yamamoto Y, Ichihara K, Mastuo S, Nakamura Y, Watanabe M, Iwatani Y. A new predictive indicator for development of pressure ulcers in bedridden patients based on common laboratory tests results. *J Clin Pathol*, 2008; 61(4): 514-518.
96. Shaw LF, Chang PC, Lee JF, Kung HY, Tung TH. Incidence and predicted risk factors of pressure ulcers in surgical patients: experience at a medical center in Taipei, Taiwan. *BioMed Research International*, 2014; 2014: 416896.
97. Chan EY, Tan SL, Lee CK, Lee JY. Prevalence, incidence and predictors of pressure ulcers in a tertiary hospital in Singapore. *J Wound Care*, 2005; 14 (8): 383-388.
98. Cremasco MF, Wenzel F, Zanei SS, Whitaker IY. Pressure ulcers in the intensive care unit: the relationship between nursing workload, illness severity and pressure ulcer risk. *J Clin Nurs*, 2013; 22(15-16): 2183-91.
99. Goodridge DM, Sloan JA, LeDoyen YM, McKenzie JA, Knight WE, Gayari M. Risk-assessment scores, prevention strategies, and the incidence of pressure ulcers among the elderly in four Canadian health-care facilities. *Can J Nurs Res*, 1998 30(2): 23-44.
100. Almirall Solsona D, Leiva Rus A, Gabasa Puig I. Apache III Score: A prognostic factor in pressure ulcer development in an Intensive Care Unit. *Enferm Intensiva*, 2009; 20(3): 95-103.
101. Lyder CH, Yu C, Emerling J, Mangat R, Stevenson D, Empleo Frazier O, McKay J. The Braden Scale for pressure ulcer risk: evaluating the predictive validity in Black and Latino/Hispanic elders. *Applied Nursing Research*, 1999; 12(2): 60-8.
102. Lyder CH, Yu C, Stevenson D, Mangat R, Empleo-Frazier O, Emerling J, McKay J. Validating the Braden Scale for the prediction of pressure ulcer risk in blacks and Latino/Hispanic elders: a pilot study. *Ostomy Wound Management*, 1998; 44(3A Suppl): 42S.
103. Meehan M. Multisite pressure ulcer prevalence survey. *Decubitus*, 1990; 3(4): 14-7.
104. Meehan M. National pressure ulcer prevalence survey. *Advances in Wound Care*, 1994; 7(3): 27-30, 34, 36-8.
105. Barczak CA, Barnett RI, Childs EJ, Bosley LM. Fourth national pressure ulcer prevalence survey. *Advances in Wound Care*, 1997; 10(4): 18-26.
106. VanGilder C, MacFarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. *Ostomy Wound Manage*, 2008; 54(2): 40-54.
107. Stahel PF, Vanderheiden T, Flierl MA, Matava B, Gerhardt D, Bolles G, Beauchamp K, Burlaw CC, Johnson JL, Moore EE. The impact of a standardized "spine damage-control" protocol for unstable thoracic and lumbar spine fractures in severely injured patients: A prospective cohort study. *The Journal of Trauma and Acute Care Surgery*, 2013; 74(2): 590-6.

108. Sasabuchi Y, Matsui H, Lefor AK, Fushimi K, Yasunaga H. Timing of surgery for hip fractures in the elderly: A retrospective cohort study. *Injury.*, 2018.
109. Fujii K, Sugama J, Okuwa M, Sanada H, Mizokami Y. Incidence and risk factors of pressure ulcers in seven neonatal intensive care units in Japan: a multisite prospective cohort study. *Int Wound J*, 2010; 7(5): 323-328.
110. Schindler CA, Mikhailov TA, Kuhn EM, Christopher J, Conway P, Ridling D, Scott AM, Simpson VS. Protecting fragile skin: Nursing interventions to decrease development of pressure ulcers in pediatric intensive care. *Am J Crit Care*, 2011; 20(1): 26-35.
111. Razmus I, Lewis L, Wilson D. Pressure ulcer development in infants: State of the Science. *J Healthc Qual*, 2008; 30(5): 36-42.
112. Coleman S, Smith IL, McGinnis E, Keen J, Muir D, Wilson L, Stubbs N, Dealey C, Brown S, Nelson EA, Nixon J. Clinical evaluation of a new pressure ulcer risk assessment instrument, the Pressure Ulcer Risk Primary or Secondary Evaluation Tool (PURPOSE T). *J Adv Nurs*, 2017; 23: 23.
113. Speechley M, Kunnilathu A, Aluckal E, Balakrishna MS, Mathew B, George EK. Screening in public health and clinical care: Similarities and differences in definitions, types, and aims: A systematic review. *J Clin Diagn Res*, 2017; 11(3): LE01-LE04.
114. National Institute for Health and Clinical Excellence (NICE), Pressure ulcers: Prevention and Management Clinical Guideline [CG179] Published date: April 2014, NICE: <https://www.nice.org.uk/guidance/cg179>.
115. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
116. Panel for the Prediction and Prevention of Pressure Ulcers in Adults, Pressure ulcers in adults: prediction and prevention. Clinical practice guideline number 3. AHCPR Publication No. 92-0047. 1992, Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services: Rockville.
117. Royal College of Nursing (RCN), National Institute for Health and Clinical Excellence (NICE), The management of pressure ulcers in primary and secondary care. 2005, RCN and NICE,: London.
118. Chou R, Dana T, Bougatsos C, Blazina I, Starmer A, Reitel K, Buckley D, Pressure ulcer risk assessment and prevention: Comparative effectiveness review no. 87. 2013, Agency for Healthcare Research and Quality, : Rockville MD.
119. Chin-Yee B, Upshur R. Clinical judgement in the era of big data and predictive analytics. *J Eval Clin Pract.* , 2018; 24(3): 638-645.
120. Norton D, Exton-Smith AN, McLaren R, *An investigation of geriatric nursing problems in hospital*. 1962, London: National Corporation for the Care of Old People.
121. Waterlow J. A risk assessment card. *Nurs Times*, 1985; 81: 24-27.
122. Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden Scale for predicting pressure sore risk. *Nurs Res*, 1987; 36(4): 205-10.
123. Fossum M, Olle Söderhamn O, Cliffordson C, Söderhamn U. Translation and testing of the Risk Assessment Pressure Ulcer Sore scale used among residents in Norwegian nursing homes. *BMJ Open*, 2012; 2:e001575 doi:10.1136/bmjopen-2012-001575.
124. Bååth C, Hall-Lord M-L, Idvall E, Wiberg-Hedman K, Wilde Larsson B. Interrater reliability using Modified Norton Scale, Pressure Ulcer Card, Short Form-Mini Nutritional Assessment by registered and enrolled nurses in clinical practice. *J Clin Nurs*, 2008; 17(5): 618-626.
125. Lospitao-Gómez S, Sebastián-Viana T, González-Ruiz JM, Álvarez-Rodríguez J. Validity of the current risk assessment scale for pressure ulcers in intensive care (EVARUCI) and the Norton-MI scale in critically ill patients. *Appl Nurs Res*, 2017; 38: 76-82.
126. Leal-Felipe MLA, Arroyo-Lopez MDC, Robayna-Delgado MDC, Gomez-Espejo A, Perera-Diaz P, Chinea-Rodriguez CD, Garcia-Correa N, Jimenez-Sosa A. Predictive ability of the EVARUCI scale and COMHON index for pressure injury risk in critically ill patients: A diagnostic accuracy study. *Aust Crit Care*, 2017; 06: 06.
127. Fulbrook P, Anderson A. Pressure injury risk assessment in intensive care: comparison of inter-rater reliability of the COMHON (Conscious level, Mobility, Haemodynamics, Oxygenation, Nutrition) Index with three scales. *J Adv Nurs*, 2016; 72(3): 680-92.
128. Meehan AJ, Beinlich NR, Hammonds TL. A nurse-initiated perioperative pressure injury risk assessment and prevention protocol. *AORN J*, 2016; 104(6): 554-565.
129. Krishnan S, Brick RS, Karg PE, Tzen YT, Garber SL, Sowa GA, Brienza DM. Predictive validity of the Spinal Cord Injury Pressure Ulcer Scale (SCIPUS) in acute care and inpatient rehabilitation in individuals with traumatic spinal cord injury. *NeuroRehabilitation*, 2016; 38(4): 401-409.
130. Delparte JJ, Scovil CY, Flett HM, Higgins J, Laramée MT, Burns AS. Psychometric Properties of the Spinal Cord Injury Pressure Ulcer Scale (SCIPUS) for pressure ulcer risk assessment during inpatient rehabilitation. *Arch Phys Med Rehabil*, 2015; 96(11): 1980-5.
131. Vocci MC, Toso LAR, Fontes CMB. Application of the Braden Q scale at a pediatric intensive care unit. *Journal of Nursing UFPE / Revista de Enfermagem UFPE*, 2017; 11(1): 165-172.
132. Cubbin B, Jackson C. Trial of a pressure area risk calculator for intensive therapy patients. *Intensive Care Nurs*, 1991; 7: 40-4.
133. Kosmidis D, Koutsouki S. Pressure ulcers risk assessment scales in ICU patients: validity comparison of Jackson/Cubbin (revised) and Braden scales. *Nosileftiki*, 2008; 47(1): 86-95.
134. Bergquist-Beringer S, Gajewski B, Davidson DJ, *Pressure Ulcer Prevalence and Incidence: Report from the National Database of Nursing Quality Indicators® (NDNQI®)*, in *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future.* , B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
135. García-Fernández F, Pancorbo-Hidalgo P, Soldevilla J. Predictive capacity of risk assessment scales and clinical judgment for pressure ulcers. *J Wound Ostomy Continence Nurs*, 2014; 41(1): 1-11.
136. Moore ZEH, Patton D. Risk assessment tools for the prevention of pressure ulcers. *Cochrane Database Syst Rev*, 2019; 1: Art. No.: CD006471. DOI: 10.1002/14651858.CD006471.pub4.
137. Saleh M, Anthony D, Parboteeah S. The impact of pressure ulcer risk assessment on patient outcomes among hospitalised patients. *J Clin Nurs*, 2009; 18(13): 1923-29.
138. Defloor T, De Bacquer D, Grypdonck MHF. The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. *Int J Nurs Stud*, 2005; 42(1): 37-46.

139. Berlowitz D, VanDeusen Lukas C, Parker V, Niederhauser A, Silver J, Logan C, Ayello P, Zulkowski K, Preventing pressure ulcers in hospitals: A toolkit for improving quality of care. 2014, Agency for Healthcare Research and Quality: <https://www.ahrq.gov/professionals/systems/hospital/pressureulcertoolkit/index.html>.
140. Kottner J, Dassen T. Pressure ulcer risk assessment in critical care: interrater reliability and validity studies of the Braden and Waterlow scales and subjective ratings in two intensive care units. *Int J Nurs Stud*, 2010; 47(6): 671-77.
141. Kottner J, Halfens R, Dassen T. An interrater reliability study of the assessment of pressure ulcer risk using the Braden scale and the classification of pressure ulcers in a home care setting. *Int J Nurs Stud*, 2009; 46(10): 1307-1312.
142. Rogenski NMB, Kurcgant P. Measuring interrater reliability in application of the Braden Scale. *Acta Paulista de Enfermagem*, 2012; 25(1): 24-28.
143. Kottner J, Dassen T. An interrater reliability study of the Braden Scale in two nursing homes. *Int J Nurs Stud*, 2008 45(10): 1501-1511.
144. Simão C, M,, Caliri M, H,L, , Dos Santos C, B. Agreement between nurses regarding patients' risk for developing pressure ulcer. *Acta Paulista de Enfermagem*, 2013; 26(1): 30-5.
145. Kim E, Choi M, Lee J, Kim YA. Reusability of EMR data for applying Cubbin and Jackson pressure ulcer risk assessment scale in critical care patients. *Healthcare Informatics Research*, 2013; 19(4): 261-270.
146. Braden B, Bergstrom N. *Braden Scale For Predicting Pressure Sore Risk*. 1988 [cited August 2019]; Available from: <https://ww2.health.wa.gov.au/~media/Files/Corporate/general%20documents/safety/PDF/Bradenscale.pdf>.
147. *The Norton Pressure Sore Risk-Assessment Scale Scoring System*. [cited August 2019]; Available from: https://shrtn.on.ca/norton_pressure_sore_risk_assessment.
148. Waterlow J. *Waterlow Score Card*. Available from: <http://www.judy-waterlow.co.uk/the-waterlow-score-card.htm>.
149. Ahtiala MH, Soppi E, Kivimäki R. Critical evaluation of the Jackson/Cubbin Pressure Ulcer Risk Scale: A secondary analysis of a retrospective cohort study population of intensive care patients. *Ostomy Wound Management* 2016; 62(2): 24-33.
150. *Spinal Cord Injury Pressure Ulcer Scale*. 2019 [cited August 2019]; Available from: <https://www.sralab.org/rehabilitation-measures/spinal-cord-injury-pressure-ulcer-scale>.
151. *Braden Q Scale*. [cited August 2019]; Available from: <https://paws.gru.edu/pub/Nursing%20Portal/Resources/Documents/bradenQscale.pdf>.
152. Curley MAQ, Hasbani NR, Quigley SM, Stellar JJ, Pasek TA, Shelley SS, Kulik LA, Chamblee TB, Dilloway MA, Caillouette CN, McCabe MA, Wypij D. Predicting Pressure Injury Risk in Pediatric Patients: The Braden QD Scale. *J Pediatr*, 2018; 192: 189-195.e2.
153. Schoonhoven L, Defloor T, Grypdonck MH. Incidence of pressure ulcers due to surgery. *Journal of Clinical Nursing*, 2002; 11(4): 479-487.
154. Seongsook RNJ, Ihnsook RNJ, Younghee RNL. Validity of pressure ulcer risk assessment scales; Cubbin and Jackson, Braden, and Douglas scale. *International Journal of Nursing Studies*, 2004; 41(2): 199-204.
155. Kim E, Lee S, Eom M. Comparison of the predictive validity among pressure ulcer risk assessment scales for surgical ICU patients. *Australian Journal of Advanced Nursing*, 2009; 26(4): 87-94.
156. Pang SM, Wong TK. Predicting pressure sore risk with the Norton, Braden, and Waterlow scales in a Hong Kong rehabilitation hospital. *Nurs Res*, 1998; 47(3): 147-153.
157. Wai-Han C, Kit-Wai C, French P, Yim-Sheung L, Lai-Kwan T. Which pressure sore risk calculator? A study of the effectiveness of the Norton scale in Hong Kong. *International Journal of Nursing Studies*, 1997; 34(2): 165-169.
158. van Marum RJ, Ooms ME, Ribbe MW, van Eijk JT. The Dutch pressure sore assessment score or the Norton scale for identifying at risk nursing home patients? . *Age & Ageing*, 2000; 29(1): 63-68.
159. Chou R, Dana T, Bougatsos C, Blazina I, Starmer A, Reitel K, Buckley D, Pressure Ulcer Risk Assessment and Prevention: Comparative Effectiveness Review No. 87. (Prepared by Oregon Evidence-based Practice Center under Contract No. 290-2007-10057-I.) AHRQ Publication No. 12(13)-EHC148-EF. 2013, Agency for Healthcare Research and Quality, : Rockville MD.
160. Raju D, Su X, Patrician PA, Loan LA, McCarthy MS. Exploring factors associated with pressure ulcers: A data mining approach. *Int J Nurs Stud*, 2014.
161. Li HL, Lin SW, Hwang YT. Using nursing information and data mining to explore the factors that predict pressure injuries for patients at the end of life. *Comput Inform Nurs*, 2019; 37(3): 133-141.
162. Kaewprag P, Newton C, Vermillion B, Hyun S, Huang K, Machiraju R. Predictive models for pressure ulcers from intensive care unit electronic health records using Bayesian networks. *BMC Med Inform Decis Mak*, 2017; 17(Suppl 2): 65.
163. Cramer EM, Seneviratne MG, Sharifi H, Ozturk A, Hernandez-Boussard T. Predicting the Incidence of pressure ulcers in the intensive care unit using machine learning. *EGEMS (Wash DC)*, 2019; 7(1): 49.
164. Cho I, Park I, Kim E, Lee E, Bates DW. Using EHR data to predict hospital-acquired pressure ulcers: A prospective study of a Bayesian Network model. *Int J Med Inform*, 2013.
165. Alderden J, Pepper GA, Wilson A, Whitney JD, Richardson S, Butcher R, Jo Y, Cummins MR. Predicting pressure injury in critical care patients: A machine-learning model. *Am J Crit Care*, 2018; 27(6): 461-468.

SKIN AND TISSUE ASSESSMENT

Introduction

Skin and soft tissue assessment is a key component of pressure injury prevention, classification, diagnosis, and treatment. The condition of the skin and underlying tissue can serve as an indicator of early signs of pressure damage.¹ Routine skin and tissue assessment provides an opportunity for early identification and treatment of skin alterations, especially pressure injuries.

As discussed in the guideline chapter on *Risk Factors and Risk Assessment*, various skin alterations (e.g., dryness, moisture, thinning or inflammation) appear to be associated with pressure injury development. Variation in skin status weakens the skin barrier and increases susceptibility to a wide range of skin problems, including pressure injuries. Advanced age, medications (e.g., steroids) or chronic disease (e.g., diabetes mellitus) all impact on both mechanical boundary conditions and the susceptibility and tolerance of the individual's skin to conditions that increase the risk of damage. Excess moisture on the skin surface (e.g., due to increased perspiration or incontinence) also increases skin vulnerability to damage related to skin maceration, pressure and shear forces.²

Clinical Questions

The clinical questions that guided the development of this chapter were:

- Are scale/ tools effective methods to assess the skin and soft tissue?
- What are effective methods of assessing erythema?
- Is ultrasound an effective method for assessing the skin and soft tissue?
- Is evaluation of skin and tissue moisture an effective method of assessing the skin and soft tissue?
- Is evaluation of skin and tissue temperature an effective method of assessing the skin and soft tissue?
- What additional technologies are accurate and effective methods of assessing skin and soft tissue?
- What methods are effective for assessing skin and soft tissue in individuals with darkly pigmented skin?

Conducting Skin and Tissue Assessment

2.1: Conduct a comprehensive skin and tissue assessment for all individuals at risk of pressure injuries:

- As soon as possible after admission/transfer to the healthcare service
- As a part of every risk assessment
- Periodically as indicated by the individual's degree of pressure injury risk
- Prior to discharge from the care service.

(Good Practice Statement)

Implementation Considerations

- **Skin inspection should be a high priority and performed as soon as possible following admission to a healthcare service (*Expert opinion*).**
- At the organizational level, ensure that a complete skin assessment is part of the risk assessment screening policy in the care service (*Expert opinion*).
- Conduct a head-to-toe assessment with particular focus on skin overlying bony prominences, including the sacrum, heels, hip, pubis, thighs and torso.^{3,4} Include the occiput in a head-to-toe skin assessment for neonates and young children (*Expert opinion*).
- Assess the skin for signs of maceration, paying attention to skin folds, especially in individuals who have obesity (*Expert opinion*).
- Inspect the skin for erythema before repositioning. Avoid positioning the individual on an area of erythema wherever possible (*Expert opinion*).
- Assess the skin and soft tissues underneath medical devices as a part of routine skin assessment (*Expert opinion*). See the guideline chapter on *Device Related Pressure Injuries* for more information about assessing skin under and around devices

- Assess the skin underneath prophylactic dressings (*Expert opinion*). See the guideline chapter on *Preventive Skin Care* for more discussion.
- Increase the frequency of skin assessments in response to deterioration in the individual's overall condition (*Expert opinion*).
- Assess localized pain at every skin assessment. Localized pain at pressure points is a risk factor for pressure injuries.⁵ See the guideline chapter on *Risk Factors and Risk Assessment* for more discussion (*Level 1*).
- Document the findings of all skin assessments (*Expert opinion*).

Discussion

Skin and soft tissue assessment is the basis of pressure injury prevention and treatment. Skin and tissue assessment is an essential component of any pressure injury risk assessment and should be conducted as soon as possible after admission, as a component of a full risk assessment (see the guideline chapter on *Risk Factors and Risk Assessment*). Each time the individual's clinical condition changes, a comprehensive skin and tissue assessment should be conducted to identify any alterations to skin characteristics or integrity, and to identify any new pressure injury risk factors. Finally, a comprehensive skin and soft tissue assessment should be conducted on discharge, to ensure that an appropriate pressure injury prevention and treatment plan is in place.

A comprehensive skin and soft tissue assessment consists of a head-to-toe assessment with particular focus on skin overlying bony prominences including the sacrum, ischial tuberosities, greater trochanters and heels.^{6,7} Include the occiput in a skin assessment for neonates and young children because their comparatively larger head circumference places them at higher risk for occipital pressure injuries than older children and adults.^{8,9} Strategies to use when performing the head-to-toe skin assessment are discussed throughout this chapter. Additionally, several studies also offer some indication that pain over the site was a precursor to tissue breakdown.^{5,10-12} Strategies for evaluating pain are discussed in the guideline chapter *Pain Assessment and Treatment*. There was no evidence available on the effectiveness of formal tools or scales to assess the skin and soft tissues.

Performing skin and tissue assessment in the individual with obesity is similar to that for standard sized individuals; however, there are additional considerations and challenges.¹³ Increased body weight and skin folds of the obese individual makes it difficult to view bony prominences and the skin. The weight of the pannus (the abdominal fat and the skin fold apron) can cause pressure injuries to develop in areas such as the sacrum, heels, hip, pubis, thighs and torso.¹⁴ In obese individuals, pressure injuries may also result from tissue pressure across the buttocks and other areas of high adipose tissue concentration, in addition to the bony prominences. Pressure injuries may develop in unique locations, such as underneath folds of skin and in locations where tubes and other devices have been compressed between skin folds. A particular feature of severe obesity is maceration, inflammation, and tissue/skin necrosis, especially in large and deep skin folds. An increased tissue weight exerts additional load on dependent tissues and causes vascular occlusion and tissue deformation. This, in conjunction with a fragile vascular and lymphatic framework and increased diaphoresis, is responsible for additional skin and tissue complications in individuals who are obese.^{14,15} The combination of moisture trapped under skin folds, pressure of skin folds on the underlying skin, and friction and shear between the skin surfaces are all factors that contribute to pressure injury formation underneath folds of skin. Therefore, care should be made to perform inspection of the full skin surface when undertaking a comprehensive skin and soft tissue assessment.

In addition to comprehensive skin assessment, a brief skin assessment of the pressure points should be undertaken during repositioning. Check the pressure points on which the individual has been positioned to identify any alterations in condition and to evaluate the effectiveness of the repositioning regimen. Presence of persistent erythema can indicate a need to increase frequency of repositioning. Check pressure points onto which the individual will be repositioned to ensure that the skin and tissue has fully recovered from previous loading.

A policy outlining a structured skin and tissue assessment approach relevant to the clinical setting should be implemented at the organizational level to promote the performance of regular assessment, including as a component of risk assessments. The policy should include the timing of assessment and reassessments and include anatomical locations to target. Accurate documentation is essential for monitoring the progress of the individual and aiding communication between health professionals,³ and organizational policy and health professional education should address documentation requirements.

**2.2: Inspect the skin of individuals at risk of pressure injuries to identify presence of erythema.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)**

2.3: Differentiate blanchable from non-blanchable erythema using either finger pressure or the transparent disk method and evaluate the extent of erythema.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Ongoing skin assessment is necessary to detect early signs of pressure injury. Evidence from three Level 1 studies, one Level 2 study and a Level 3 study indicates that the presence of non-blanching erythema, a Category/Stage I pressure injury is predictive of development of a Category/Stage II or greater pressure injury.^{5,16-19} Evidence from three Level 3 studies²⁰⁻²² indicates that the presence of reddened skin other than blanchable erythema is associated with Stage/Category II pressure injury development. Identifying presence of erythema alerts health professionals to the need for further assessment and potential development of a pressure injury prevention and/or treatment plan. Identification of erythema is a component of a skin inspection.

Evidence from a high quality Level 1 study indicates that presence of non-blanching erythema is predictive of development of a Category/Stage II pressure injury.^{5,16-19} Evidence from high quality Level 2 and 3 studies indicated that the finger pressure method has strong psychometric properties for differentiating blanching and non-blanching erythema.^{23,24,25} A low quality Level 4 study indicated that using the finger pressure method may be more reliable than the transparent disk method.²⁵

Implementation Considerations

- Inspection of the skin should include a visual inspection in conjunction with other skin assessment techniques such as touch and palpation for differences in temperature and tissue consistency²⁶ (Level 2).
- Ensure adequate tangential lighting during visual inspection of the skin²⁶ (Level 2).
- To perform the finger pressure method, a finger is pressed on the erythema for three seconds and blanching is assessed following removal of the finger on intact skin^{23,25} (Levels 2 and 4).
- To perform the transparent disk method, a transparent disk is used to apply pressure equally over an area of erythema and blanching can be observed underneath the disk during its application^{23,25} (Levels 2 and 4).
- If there is difficulty in differentiating between a Category/Stage I pressure injury and reactive hyperemia, relieve the pressure area for 30 minutes, then repeat the skin inspection (*Expert opinion*).
- Large skin areas require several measurement points (*Expert opinion*).
- Document the findings of all skin assessments (*Expert opinion*).
- It is not always possible to identify erythema on darkly pigmented skin (*Expert opinion*). Further guidance on assessing darkly pigmented skin in which detection of erythema is more difficult is provided throughout this chapter.

Evidence Discussion

Skin redness is known as erythema. The redness is classified as either blanchable or non-blanchable. **Blanchable erythema** is visible skin redness that becomes white when light pressure is applied and reddens when pressure is relieved. Blanchable erythema may result from normal reactive hyperemia that should disappear within several hours, or it may result from inflammatory erythema with an intact capillary bed.^{23,27} **Non-blanchable erythema** is visible skin redness that persists with the application of pressure. It indicates structural damage to the capillary bed/microcirculation.

Initial and ongoing assessment of the skin is necessary to detect early signs of pressure damage. A visual assessment for erythema is the first component of every skin inspection. Skin redness, in conjunction with tissue edema resulting from capillary occlusion, is a response to pressure, especially over bony prominences.

Presence of non-blanchable erythema is an indication for a Category/Stage I pressure injury.²⁷ As discussed in the guideline chapter *Risk Factors and Risk Assessment*, when a Category/Stage I pressure injury forms the individual is at risk of developing a Category/Stage II or greater pressure injury. Across five prognostic studies,^{5,16-19} the risk of developing a more severe pressure injury was between three and five times' higher once a Category/Stage I pressure injury had been identified (odds ratio [OR] ranged from 3.1 to 7.98) (Level 1, 2 and 3 evidence).

Presence of blanchable erythema has also been identified as a predictor for development of a pressure injury. In a large (n = 698) prognostic study in acute care, critical care and non-surgical care, presence of erythema was associated with a more than two-fold increase in the risk of pressure injuries of Category/Stage II or greater. However, in the same

study, presence of hyperemic skin showed no significant associations²⁰ (*Level 3*). In an aged care setting, severity of blanchable erythema was associated with an increased incidence of Category/Stage II or greater pressure injuries,²² and the same findings were noted for identification of erythema on individuals in acute, critical and surgical care settings²¹ (both *Level 3*). The prognostic studies^{5,16-22} that reported blanchable and non-blanchable erythema as predictors were conducted in acute care, chronic care and aged care settings, suggesting skin status influences the susceptibility and tolerance to strains and stresses that may cause pressure injury of individuals in a wide range of clinical settings.

Therefore, identifying erythema is an imperative, so that an appropriate prevention and treatment plan can be developed and initiated.

Assessing Erythema

Inspection of the skin should include a visual inspection in conjunction with other skin assessment techniques, performed under good lighting conditions. A visual inspection for erythema at bony prominences should be performed when repositioning the individual, so positioning on an area of erythema can be avoided. There are two commonly used methods to assess erythema:^{23,25}

- Finger pressure method — a finger is pressed on the erythema for three seconds and blanching is assessed following removal of the finger
- Transparent disk method — a transparent disk is used to apply pressure equally over an area of erythema and blanching can be observed underneath the disk during its application.

Vanderwee et al. (2006)²³ investigated the reliability of both the finger press and the transparent disk methods of assessing erythema in a cohort of participants from an acute care geriatric ward ($n = 265$). Assessments were conducted by a researcher and nurses, all of whom were provided with training at the commencement of the study. Participants were assessed independently by a researcher and a nurse within 30 minutes of each other and using both assessment methods. Both assessment techniques had high interrater reliability between nurses and researchers. Sensitivity and specificity ranged based on the anatomical location being assessed, as noted in *Table 5.1*. The researchers noted that the transparent disk method has some advantages over the finger press method because the level of pressure applied to the skin is less variable between assessors, and blanching is observable immediately on application of pressure, which increases ease of assessment in individuals with rapid vascular refill (*Level 2*).

Kottner et al. (2009)²⁵ compared prevalence of Category/Stage I pressure injuries in hospitals and nursing homes using two different identification methods. Facilities involved in the prevalence survey were randomly assigned to use either the finger press method ($n = 5,095$ assessments) or to using the transparent disk method ($n = 4,657$ assessments). The finger method was more likely to identify a Category/Stage I pressure injury (OR = 1.80, 95% confidence interval [CI] 1.49 to 2.18, $p < 0.001$) (*Level 4*).

Table 5.1: Psychometric properties of techniques to assess erythema

	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value	Interrater Reliability
Finger Pressure Method	65.3% to 73.1% depending on anatomical location ^{23,24}	93.9% to 95.5% depending on anatomical location ^{23,24}	75% ²³	95.1% ²³	<ul style="list-style-type: none"> • $\kappa = 0.20$ performed by independent assessors with unreported qualifications²⁷ • $\kappa = 0.62$ to $\kappa = 0.72$ performed by nurses and related to their level of experience²³
Transparent Disk Method	74.5% ²³	95.6% ²³	79.5% ²³	94.2% ²³	<ul style="list-style-type: none"> • $\kappa = 0.88$ to 0.89 performed by researchers and nurses and related to their qualifications²⁴ • $\kappa = 0.68$ to $\kappa = 0.76$ performed by nurses and related to their level of experience²³

Using both visual identification of erythema and a physical assessment of the type of erythema is the most reliable method by which to discriminate between blanching and non-blanching erythema. Sterner et al. (2011)²⁷ noted the importance of performing a physical assessment of erythema in their study conducted with older individuals with hip fractures ($n = 78$). Both a visual inspection and a finger press test of sacral skin were conducted daily for up to five days. Interrater reliability was lower on day one for the finger press test ($\kappa = 0.44$, 95% CI 0.21 to 0.67) than for visual inspection alone ($\kappa = 0.67$, 95% CI 0.5 to 0.82). By day five, interrater reliability for the finger press test decreased to 0.20 (95% CI -0.06 to 0.46), while interrater reliability increased slightly for the visual inspections ($\kappa = 0.76$, 95% CI 0.61 to 0.91) (*Level 3*). However, type of erythema cannot be established by a visual assessment alone.

2.4: Assess the temperature of skin and soft tissue.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence from a high quality Level 1 study²⁸ indicated that cooler temperature in the center of an area of skin discoloration was predictive of pressure injury development. A moderate quality Level 3 study²⁹ supported this finding. The research was primarily conducted in Caucasian women.²⁸ The evidence on feasibility and acceptability of implementing routine skin and soft tissue temperature assessment was mixed and evidence on resource requirements for various methods of skin temperature measurement in different clinical settings is also lacking.

Implementation Considerations

- Consider the findings of skin and tissue assessment in the context of the individual's overall presentation and pressure injury risk profile. Relative skin temperature changes over areas of inflammation can present as warmer than surrounding skin and tissue. Relative skin temperature changes over areas of ischemia can present as colder than surrounding skin and tissue (*Expert opinion*).
- Provide health professionals with education and experience in assessing skin temperature with the hand to increase their skills in identifying small temperature differences²⁶ (*Level 2*).
- Consider using an infrared thermographic imaging device^{28,29} (*Levels 1 and 3*) or an infrared thermometer as an adjunct to clinical examination of the skin³⁰ (*Level 3*).
- Health professionals should undertake appropriate education and training before using infrared thermal imaging to monitor the temperature of skin and soft tissues (*Expert opinion*).

Evidence Discussion

Localized heat, edema and change in tissue consistency in relation to surrounding tissue (e.g., induration/hardness) have all been identified as warning signs for pressure injury development.^{23,31} Early identification of changes in skin and tissue color, temperature and consistency enables implementation of an appropriate prevention and treatment plan.

Inspection, touch and palpation are the most commonly used techniques to assess temperature and turgor/consistency of the skin and soft tissue. Palpation of the skin and soft tissues to detect changes in temperature requires skilled examination. One study demonstrated that use of palpation and touch can detect between 1°C and 3°C differences in temperature, with higher accuracy in trained health professionals.³² This suggests that providing education and skills reinforcement for health professionals is important to developing and maintaining their practical skills. Rosen et al. (2006)²⁶ demonstrated that conducting a comprehensive assessment of skin color, texture and warmth can lead to significant improvements in pressure injury rates in an aged care setting. In this study, health professionals were provided education on conducting a skin assessment and identifying subtle differences in skin characteristics. The skin assessment protocol that was initiated emphasized the use of touch to perceive skin warmth at pressure points, and the use of adequate lighting (using a penlight) to detect changes in skin texture and color. Within 12 weeks, there was a significant reduction in pressure injuries compared to baseline ($p < 0.05$) (*Level 2*).

More recently, objective measures of skin temperature using infrared imaging have become more accessible to health professionals in some geographic and clinical settings. These techniques can be used as an adjunct to clinical examination skills to assess skin temperature.

Cox et al. (2016)²⁸ investigated the prognostic value of infrared thermography in a prospective study set in skilled nursing facilities ($n = 67$ participants). Discolored skin was observed for between 7 and 14 days. At 14-day follow up, 45% of discolored skin completely resolved and 32% had become an area of necrosis. Discolored skin that was cooler in the center at the initial observation was significantly more likely to be classified as necrosis within seven days (OR = 18.8, 95% CI 1.04 to 342.44). This suggests that the initial infrared thermography was successful in identifying areas of deep tissue injury²⁸ (*Level 1*). However, there were wide confidence intervals in this study, and the participants were primarily Caucasian. This reduces the certainty and generalizability of the findings.

Farid et al. (2012)²⁹ reviewed records for individuals ($n = 85$) with intact pressure-related skin discoloration for whom skin temperature was measured during skin assessment. All temperature assessments were conducted using a handheld infrared thermographic device. Skin temperature was measured at both pressure-related discolored areas and adjacent normal skin. At the time of the initial skin assessment, approximately 65% of participants had a lower skin temperature in the pressure-related discolored skin region compared to the adjacent skin. The pressure-related

discolored region was significantly more likely to progress to skin necrosis within seven days than in participants with higher skin temperature in the discolored skin region (OR = 31.8, 95% CI 3.8 to 263.1, $p = 0.001$)²⁹ (Level 3). The wide confidence interval suggests that there is some uncertainty in these findings; however, the findings are consistent with the more recent study by Cox et al. (2016) establishing that changes in skin temperature are associated with areas of deep tissue injury.²⁸

Judy et al. (2011)³⁰ used an infrared thermal imaging device to evaluate skin temperature as an objective measure of pressure injury risk. In a repeated-measures study, infrared scans of the sacrum and heels were conducted daily ($n = 100$ participants). Infrared imaging results were able to predict the development of 100% ($n = 5$) of pressure injuries that developed during the study period using a variance of 1.5°C in skin temperature as the predictor. In comparison, the Braden Scale score predicted 60% of pressure injuries.³⁰ The less than ideal interrater reliability achieved in this study ($\kappa = 0.40$ to $\kappa = 0.42$)³⁰ supports the suggestion that education and ongoing experience in using infrared devices is important to achieving accurate results (Level 3).

2.5: Assess edema and assess for change in tissue consistency in relation to surrounding tissues.
(Good Practice Statement)

2.6: Consider using a sub-epidermal moisture/edema measurement device as an adjunct to routine clinical skin assessment.
(Strength of Evidence = B2; Strength of Recommendation = ↔)

Evidence Summary

Evidence from a high quality Level 2 study,³³ moderate and low quality Level 3 studies³⁴⁻³⁸ and a moderate quality Level 4 study³⁹ indicated that a sub-epidermal moisture (SEM) measurement can be used as a measure for tissue edema. In a high quality Level 2 study,³³ SEM measurements strongly correlated to a visual skin assessment at the sacrum, but measures taken at the heel had a moderate to low correlations with the visual assessment. Some evidence from moderate quality Level 3 studies^{34,35} suggested that SEM measurements are predictive of Category/Stage I or greater pressure injuries occurring within one week. Studies showed high sensitivity and specificity, and high interrater reliability for SEM measurements,^{39,40} but low quality and indirect evidence on repeat-measure reliability was conflicting.^{36,40} There was no evidence on the correlation between SEM measurements and palpation.

Implementation Considerations

- Provide health professionals with education and experience in assessing edema and changes in skin consistency with the hand to increase their skills in identifying clinically significant changes²⁶ (Level 2).
- Health professionals require training in using a device that measures SEM/edema to facilitate consistency in measurement method over time and between users (*Expert opinion*).

Evidence Discussion

As previously noted, inspection and palpation of the skin are used most often to detect edema and changes to skin texture and consistency. Ongoing education has been shown to increase clinical skills in skin assessment, leading to reduction in pressure injury incidence²⁶ (Level 2).

Bates-Jensen et al.^{34,35} introduced the concept of SEM as a tissue parameter. Sub-epidermal moisture is a measure of soft tissue edema below the skin surface. Hydration of sub-epidermal tissues is normal. However, inflammatory processes associated with tissue damage lead to increases in SEM in soft tissues.⁴¹ Change in SEM is therefore a marker for inflammation and tissue damage.⁴¹

Numerous small studies have explored the use of SEM measurement in predicting the incidence of blanchable erythema and Category/Stage I pressure injuries, primarily in older adults. In the earliest study conducted by Bates-Jensen et al. (2008), higher SEM measurements were established in older adults ($n = 31$) with skin that was visually assessed as being damaged. Odds ratio of developing a Category/Stage I pressure injury within one week after a predictive SEM measurement was 1.003 (95% CI 1.000 to 1.006). Likewise, the risk of developing a Category/Stage II pressure injury was also statistically significantly increased (OR = 1.32) after a high SEM reading (Level 3). In a follow-up study, SEM measurements predicted the incidence within one week of both Category/Stage I pressure injuries (OR ranged from 2.11 to 5.31 depending on skin tone) and Category/Stage II pressure injuries (OR ranged from 4.30 to 8.51

depending on skin tone)³⁵ (*Level 3*). More recent studies conducted in older adults³⁷ and individuals with jaundice³⁸ have demonstrated that SEM readings increase by statistically significant amounts as the extent of skin damage increases^{37,38} (*Level 3*).

A number of studies^{36,42-44} have reported psychometric properties of different devices for SEM measurements performed by nurses or other trained operators at a range of anatomical locations, including the sacrum, trochanters and heels (see *Table 5.2*).

There is limited information on the resource impact of using SEM to assess soft tissues. The National Institute for Health and Care Excellence (2019)⁴⁵ reported the cost of one device as £5,835 (UK pound, 2019). In one study, putting in place appropriate pressure injury prevention plans based on SEM data was estimated to save 1,420 nursing hours, with an estimated revenue increase of £53,000 based on admissions saved.⁴⁶ However, costs vary widely based on clinical and geographic location and there is insufficient data on resource implications of using SEM measurements.

Table 5.2: Psychometric properties of different devices for SEM/edema measurements

	Sensitivity	Specificity	Interrater Reliability
SEM/edema Measurements	100% (95% CI 83.89% to 100%) ⁴⁴	83% (95% CI 75.44% to 89.51%) ⁴⁴	<ul style="list-style-type: none"> • ICC = 0.80 (trained operators)⁴² • r=0.86 to r=0.92 (observers with unspecified qualifications)⁴³

Assessing Darkly Pigmented Skin and Soft Tissue

There is evidence that Category/Stage I pressure injuries are under-detected in individuals with darkly pigmented skin.⁴⁷ It appears that this is related to areas of redness being more difficult to see on darker skin tones.

In a large study⁴⁸ conducted in 59 nursing homes (n = 1,938 participants) the rate of Category/Stage II to IV pressure injuries was statistically significantly higher in individuals with darker skin tones when compared to individuals with lighter skin (0.56 versus 0.35 per person per year, p < 0.001). This translated to a higher pressure injury risk for individuals with darker skin (hazard ratio [HR] = 1.31, 95% CI 1.02 to 1.66, p = 0.032) when controlling for other characteristics. Other studies have also suggested this racial disparity in pressure injury incidence. Rosen et al. (2006)²⁶ reported a baseline prevalence of pressure injuries in an aged care facility prior to introducing a preventive intervention focused on skin assessment. The baseline prevalence of Category/Stage I to IV pressure injuries was 0.47 per 100 bed days in individuals with dark skin tones compared to 0.28 per 100 bed days for those with light skin (p = 0.098). However, VanGilder et al. (2008)⁴⁷ noted more subtleties when examining the relationship between pressure injuries and skin tone in an international pressure injury prevalence study. As noted in other studies, individuals with darker skin tones experienced higher rates of Category/Stage III and Category/Stage IV pressure injuries. However, Category/Stage I pressure injuries were proportionately lower in individuals with dark skin tones (13%) compared with individuals who had medium skin tones (32%) and light skin tones (38%).⁴⁷

It is likely that Category/Stage I pressure injuries are under-reported in individuals of darker skin tone due to failure to identify early differences in skin color. Delayed identification of early skin damage prolongs implementation of preventive care, which may explain the higher incidence of more severe pressure injuries in dark skin individuals reported in the above studies.^{47,48} Astute assessment of intact skin in dark-skinned individuals is critical in reversing this trend.

2.7: When assessing darkly pigmented skin, consider assessment of skin temperature and sub-epidermal moisture as important adjunct assessment strategies.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

2.8: Evaluate the relevance of performing an objective assessment of skin tone using a color chart when conducting a skin assessment.
(Strength of Evidence = B2; Strength of Recommendation = ↔)

Evidence Summary

One small, moderate quality Level 3 study²⁹ identified that cooler pressure injury related intact skin was more likely to develop into skin necrosis within seven days; and darker skinned individuals had 3.8 times higher likelihood of developing skin necrosis.²⁹ One moderate quality Level 2 study²⁶ identified that an intervention focused on educating health professionals in conducting a comprehensive skin assessment that included using touch to identify changes in skin temperature was associated with a significant reduction in pressure injuries in dark skinned individuals.

There is evidence from one, small, moderate quality Level 3 study³⁵ indicating that SEM measurements are able to identify tissue edema one week prior to pressure injury development in individuals with dark skin tone. No evidence was available on the resource requirements for implementing SEM scanning for all dark-skinned individuals.

Evidence from a logistic regression reported in a moderate quality Level 3 study showed that skin tone classification on a Munsell color chart was a significant predictor of Category/Stage I pressure injuries (but not more severe pressure injuries). Ethnicity/race was not a significant predictor of pressure injuries. Interrater and intrarater reliability was high for Munsell-based skin tone classifications, especially in individuals with dark skin tones.⁴⁹

Implementation Considerations

- Carefully inspect any discoloration over pressure areas in individual with darkly pigmented skin. Areas of discoloration in relation to surrounding skin should be assessed more closely for temperature changes, edema, changes in tissue consistency and pain (*Expert opinion*).
- Tangential light and slightly moistening the skin may aid in the detection of early pressure injury in darker skinned individuals (*Expert opinion*).
- To achieve a reliable visual assessment of skin tone, the skin should be clean and free of skin products⁴⁹ (*Level 3*).

Evidence Discussion

Although the importance of comprehensive skin and soft tissue assessment in dark skinned individuals is acknowledged,⁴⁷ it is also recognized that assessment in dark skinned individuals can be more complex due to difficulty observing subtle changes in skin color. As it is not always possible to identify erythema on darkly pigmented skin, localized heat, edema, and change in tissue consistency in relation to surrounding tissue (e.g., induration/hardness) are important indicators of early pressure damage in skin of darker tones. As reported above, there is evidence to support inclusion of these criteria in all comprehensive skin assessments.

In the skin assessment intervention initiated by Rosen et al. (2006) in an aged care setting (see above), providing education to health professionals to improve their clinical examination skills in identifying changes in skin temperature by hand was effective in reducing pressure injury incidence after 12 weeks. The results of this study specifically indicated that increasing assessment of skin temperature was associated with a significant reduction in Category/Stage I to IV pressure injuries for individuals with dark skin tones ($p < 0.004$) (*Level 2*).

Also noted above, the record review by Farid et al. (2012)²⁹ identified that temperature assessments conducted using a handheld infrared thermographic device were able to detect skin temperature variations that were outside the norm. These areas were more likely to progress to skin necrosis within seven days, indicating that infrared thermography can identify deep tissue injury. Individuals with dark toned skin were 3.8 times more likely than light skinned individuals to develop skin necrosis after identification of cool skin areas with the handheld infrared thermography device (*Level 3*).

The study by Bates-Jensen et al. (2009)³⁵ reported above noted that SEM measurement was predictive of pressure injury incidence, indicating that SEM measurement is effective in identifying areas of inflammation and tissue damage. The findings were particularly relevant to dark skinned individuals ($n = 11$) who had an odds ratio of 1.88 ($p < 0.005$) for a Category/Stage I pressure injury and 1.02 (95% CI 1.001 to 1.02, at 1 dermal phase unit) for Category/Stage II or greater pressure injuries (*Level 3*).

One study reported on the use of skin tone charts to aid the assessment of skin in older adults from a range of ethnic backgrounds ($n = 417$). The 5YR Munsell Color Chart was used by assessors to evaluate skin tone at baseline and at 16 weeks. Skin tone categorization using the Munsell Color Chart was more accurate at predicting Category/Stage I pressure injuries than using ethnicity as a prognostic factor. However, the Munsell Color Chart categorization was not predictive of Category/Stage II or greater pressure injuries. For all ethnic groups, interrater reliability for Munsell Color Chart ratings at the buttocks was high (ICC = 0.97, $\kappa = 0.84$, $p < 0.001$), with highest reliability noted in African American individuals ($r = 0.93$, $p < 0.001$)⁴⁹ (*Level 3*). Use of color charts appears to augment a visual assessment of the skin, particularly when assessing darker skin tones.

Other Skin Assessment Techniques

A smaller body of evidence is available on other adjunct skin assessment techniques including ultrasound, photoplethysmogram (PPG), laser Doppler flowmetry (LDF) and measures of transcutaneous oxygen and other biophysical variables. There was insufficient evidence on any of these skin assessment methods to make specific recommendations; however, their use in clinical practice is increasingly popular as devices become more accessible.

Ultrasound is an acoustic therapy in which mechanical vibration is transmitted in a wave formation at frequencies beyond the upper limit of human hearing. Ultrasonic devices are used to detect objects and measure distances. Ultrasound imaging or sonography is often used for diagnostic purposes. In some studies, ultrasound has been used as a non-invasive method of detecting deep tissue injury prior to visible appearance of the injury on the skin.^{50,51} When the ultrasound waves reaches tissues in the body energy is reflected back or absorbed depending on the different characteristics of the tissue. It has been proposed that early detection of deep tissue injury could help identify whether tissue injury was present on admission.⁵⁰ Because ultrasound is portable and more accessible than magnetic resonance imaging (MRI, the gold standard method of tissue deformation measurement), exploration of the validity, reliability and feasibility of its use in early tissue assessment is important.⁵¹

There is only a small body of evidence on use of ultrasound for skin and soft tissue assessment that was insufficient to make a recommendation. Low frequency and high frequency ultrasound examine tissues at different depths, with low frequency ultrasound detecting deeper tissues. Use of low frequency ultrasound in one diagnostic study⁵⁰ showed good sensitivity, specificity and accuracy in diagnosing deep tissue injury. The ultrasound assessment was confirmed with visual assessment and pressure injury classification by a clinician conducted up to seven days after the ultrasound investigation⁵⁰ (*Level 1*). In another study, there was low to moderate correlation between an abnormal high frequency ultrasound result and being classified as having a pressure injury risk based on visual assessment and application of the Braden Scale friction/shear subscales. However, this study had insufficient pressure injury events to evaluate the ability of ultrasound to predict a pressure injury developing⁵² (*Level 3*). Indirect evidence suggests that tissue deformation associated with pressure injury is identifiable, with interrater reliability reported as high for identifying deformation in muscle, tendon/muscle and skin/fat layers but low for measures in the fat and skin layers⁵¹ (*Level 5*). Other studies⁵³⁻⁵⁵ also showed that ultrasound can detect abnormal tissues, including identification of edematous tissue under intact skin⁵⁵ (all *Level 5*). However, validation against gold standard assessment technique (i.e., MRI) is lacking. Further work is required to determine how ultrasound findings correlate with various stages of skin breakdown^{53,54} (e.g., with validation against the gold standard of MRI assessment) and how to interpret results.⁵²

Measurement of the flow of blood can provide an indication of tissue health. Laser Doppler flowmetry and photoplethysmography both provide non-invasive methods to assess circulation at different tissue depths. In studies conducted in healthy volunteers both laser Doppler flowmetry and photoplethysmography were able to measure changes in blood flow in situations with and without pressure^{56,57} (*Level 5*). However, evidence on its use in individuals with or at risk of pressure injuries is currently lacking.

Transcutaneous oxygen monitoring provides an evaluation of tissue oxygen saturation using a subcutaneous light beam that penetrates the tissue. This provides an indication of the perfusion of tissues, particularly in response to loading. However, in studies conducted in healthy volunteer assessment of skin and tissue using tissue oxygenation showed mixed results. Transcutaneous tissue oxygenation failed to identify any significant difference during four hours in supine position on a pressure redistribution support surface in one study.⁵⁸ A second study⁵⁹ showed significant increases in tissue oxygenation at the sacrum ($p > 0.05$) and the ischial tuberosity ($p < 0.01$) after 15 minutes. However, in the second study transcutaneous tissue oxygenation measures failed to identify significant differences in perfusion after 15 minutes in a sitting position⁵⁹ (*Level 5*). The mixed results indicate that more research is required on the use of transcutaneous oxygenation monitoring in the assessment of the skin and soft tissues.

One study reported on biophysical measures for assessing epidermal lipids and melanin. Melanin contributes to skin color and epidermal lipids contribute to the barrier function of the stratum corneum, therefore are a marker of skin hydration. In the study population of older adults ($n = 38$) there was a strong positive correlation between visual assessment of the skin and measure of skin wetness at sacrum, ischia and trochanters ($p < 0.01$) using the diagnostic tool. There was also a strong positive correlation between visual assessment and measures of skin pigmentation (melanin) ($p = 0.01$) and erythema ($p = 0.01$) taken using the diagnostic tool⁶⁰ (*Level 5*). However, this was a small study and the relationship between the skin assessment criteria and development of pressure injuries requires further investigation to ascertain the diagnostic tool's reliability, validity and role in predicting pressure injuries.

References

1. Dealey C, *The Care of Wounds: A Guide for Nurses*. 2005, Oxford: Blackwell Publishing Ltd.
2. Defloor T, Grypdonck MH. Sitting posture and prevention of pressure ulcers. *Appl Nurs Res*, 1999; 12(3): 136.
3. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
4. European Pressure Ulcer Advisory Panel, *Pressure Ulcer Treatment Guidelines*. 1998, EPUAP, : Oxford, England.
5. Smith IL, Brown S, McGinnis E, Briggs M, Coleman S, Dealey C, Muir D, Nelson EA, Stevenson R, Stubbs N, Wilson L, Brown JM, Nixon J. Exploring the role of pain as an early predictor of category 2 pressure ulcers: A prospective cohort study. *BMJ Open*, 2017; 7(1): e013623.
6. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
7. Wounds Australia, *Standards for Wound Prevention and Management*. 2016, Cambridge Media: Osborne Park, WA.
8. Baharestani MM, Ratliff CR. Pressure ulcers in neonates and children: an NPUAP white paper. *Adv Skin Wound Care*, 2007; 20(4): 208.
9. Razmus I, Lewis L, Wilson D. Pressure ulcer development in infants: State of the science. *J Healthc Qual*, 2008; 30(5): 36-42.
10. Hopkins A, Dealey C, Bale S, Defloor T, Worboys F. Patient stories of living with a pressure ulcer. *J Adv Nurs*, 2006; 56(4): 345-353.
11. Langemo DK, Melland H, Hanson D, Olson B, Hunter S. The lived experience of having a pressure ulcer: A qualitative analysis. *Adv Skin Wound Care*, 2000; 13(5): 225-235.
12. Spilsbury K, Nelson A, Cullum N, Iglesias C, Nixon J, Mason S. Pressure ulcers and their treatment and effects on quality of life: Hospital inpatient perspectives. *J Adv Nurs*, 2007; 57(5): 494-504.
13. Rose MA, Pokorny M, D. D. Braden subscales and their relationship to the prevalence of pressure ulcers in hospitalized obese patients. *Bariat Nurs Surg Pat*, 2009; 4(3): 221-226.
14. Rush A. Bariatric pressure ulcer prevention. *Bariat Nurs Surg Pat*, 2008; 3(2): 125-128.
15. Shipman A, Millington G. Obesity and the skin. *Br J Dermatol*, 2011; 165(4): 743-50.
16. Nixon J, Cranny G, Bond S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: A cohort study. *Int J Nurs Stud*, 2007; 44(5): 655-63.
17. Reed R, Hepburn K, Adelson R, Center B, McKnight P. Low serum albumin levels, confusion, and fecal incontinence: Are these risk factors for pressure ulcers in mobility-impaired hospitalized adults? *Gerontology*, 2003; 49(4): 255-59.
18. Allman R, Goode P, Patrick M, Burst N, Bartolucci A. Pressure ulcer risk factors among hospitalized patients with activity limitation. *J Am Med Assoc*, 1995; 273(11): 865-70.
19. Demarre L, Verhaeghe S, Van Hecke A, Clays E, Grypdonck M, Beeckman D. Factors predicting the development of pressure ulcers in an at-risk population who receive standardized preventive care: Secondary analyses of a multicentre randomised controlled trial. *J Adv Nurs*, 2015; 71(2): 391-403.
20. Compton F, Hoffmann F, Hortig T, Strauss M, Frey J, Zidek W, Schafer JH. Pressure ulcer predictors in ICU patients: Nursing skin assessment versus objective parameters. *J Wound Care*, 2008; 17(10): 417.
21. Marchette L, Arnell I, Redick E. Skin ulcers of elderly surgical patients in critical care units. *Dimens Crit Care Nurs*, 1991; 10(6): 321-29.
22. Schnelle JF, Adamson GM, Cruise PA, Al-Samarrai N, Sarbaugh FC, Uman G, Ouslander JG. Skin disorders and moisture in incontinent nursing home residents: Intervention implications. *J Am Geriatr Soc*, 1997; 45(10): 1182-1188.
23. Vanderwee K, Grypdonck M, Bacquer D, Defloor T. The reliability of two observation methods of nonblanchable erythema, Grade 1 pressure ulcer. *Appl Nurs Res*, 2006; 19: 156-162.
24. Vanderwee K, Grypdonck M, Bacquer D, Defloor T. The identification of older nursing home residents vulnerable for deterioration of grade 1 pressure ulcers. *J Clin Nurs*, 2009; 18(21): 3050-58.
25. Kottner J, Dassen T, Lahmann N. Comparison of two skin examination methods for grade 1 pressure ulcers. *J Clin Nurs*, 2009; 18(17): 2464-2469.
26. Rosen J, Mittal V, Degenholtz H, Castle N, Mulsant B, Nace D, Rubin F. Pressure ulcer prevention in black and white nursing home residents: A QI initiative of enhanced ability, incentives, and management feedback. *Adv Skin Wound Care*, 2006; 19(5): 262-269.
27. Sterner E, Lindholm C, Berg E, Stark A, Fossum B. Category I pressure ulcers: How reliable is clinical assessment? *Orthop Nurs*, 2011; 30(3): 194-205.
28. Cox J, Kaes L, Martinez M, Moles D. A prospective, observational study to assess the use of thermography to predict progression of discolored intact skin to necrosis among patients in skilled nursing facilities. *Ostomy Wound Manage*, 2016; 62(10): 14-33.
29. Farid K, Winkelman C, Rizkala A, Jones K. Using temperature of pressure-related intact discolored areas of skin to detect deep tissue injury: An observational, retrospective, correlational study. *Ostomy Wound Manage*, 2012; 58(8): 20-31.
30. Judy D, Brooks B, Fennie K, Lyder C, Burton C. Improving the detection of pressure ulcers using the TMI ImageMed system. *Adv Skin Wound Care*, 2011; 24(1): 18-24.
31. Bennett M. Report of the task force on the implications for darkly pigmented intact skin in the prediction and prevention of pressure ulcers. *Adv Wound Care*, 1995; 8(6): 34-35.
32. Walker JR, Marcellin-Little DJ, Goulet R, Ru H. Detection of skin temperature differences using palpation by manual physical therapists and lay individuals. *J Man Manip Ther*, 2018; 26(2): 97-101.
33. O'Brien G, Moore Z, Patton D, O'Connor T. The relationship between nurses assessment of early pressure ulcer damage and sub epidermal moisture measurement: A prospective explorative study. *Journal of Tissue Viability*, 2018.

34. Bates-Jensen BM, McCreath HE, Pongquan V, Apeles NCR. Subepidermal moisture differentiates erythema and stage I pressure ulcers in nursing home residents. *Wound Repair Regen*, 2008; 16(2): 189-197.
35. Bates-Jensen BM, McCreath HE, Pongquan V. Subepidermal moisture is associated with early pressure ulcer damage in nursing home residents with dark skin tones: Pilot findings. *J Wound Ostomy Continence Nurs*, 2009; 36(3): 277-284.
36. Harrow JJ, Mayrovitz HN. Subepidermal moisture surrounding pressure ulcers in persons with a spinal cord injury: A pilot study. *J Spinal Cord Med*, 2014; 37(6): 719-728.
37. Kim C, Park S, Ko J, Jo S. The relationship of subepidermal moisture and early stage pressure injury by visual skin assessment. *J Tissue Viability*, 2018; 27(3): 130-134.
38. Park S, Kim CG, Ko JW. The use of sub-epidermal moisture measurement in predicting blanching erythema in jaundice patients. *J Wound Care*, 2018; 27(5): 342-349.
39. Guihan M, Jenson B, Chun S, Parachuri R, A.S. C, McCreath HE. Assessing the feasibility of subepidermal moisture to predict erythema and stage 1 pressure ulcers in persons with spinal cord injury: A pilot study. *Journal of Spinal Cord Medicine*, 2012; 35(1): 46-52.
40. Clendenin M, Jaradeh K, Shamirian A, Rhodes SL. Inter-operator and inter-device agreement and reliability of the SEM Scanner. *Journal of Tissue Viability*, 2015; 24(1): 17-23.
41. Moore Z, Patton D, Rhodes SL, O'Connor T. Subepidermal moisture (SEM) and bioimpedance: A literature review of a novel method for early detection of pressure-induced tissue damage (pressure ulcers). *Int Wound J*, 2016; 14(2): 331-337.
42. Clendenin M, Jaradeh K, Shamirian A, Rhodes SL. Inter-operator and inter-device agreement and reliability of the SEM Scanner. *J Tissue Viability*, 2015; 24(1): 17-23.
43. Guihan M, Jenson B, Chun S, Parachuri R, A.S. C, McCreath HE. Assessing the feasibility of subepidermal moisture to predict erythema and stage 1 pressure ulcers in persons with spinal cord injury: A pilot study. *J Spinal Cord Med*, 2012; 35(1): 46-52.
44. O'Brien G, Moore Z, Patton D, O'Connor T. The relationship between nurses assessment of early pressure ulcer damage and sub epidermal moisture measurement: A prospective explorative study. *J Tissue Viability*, 2018; 27: 4.
45. National Institute for Health and Care Excellence. *SEM Scanner for pressure ulcer prevention: Medtech innovation briefing [MIB182]*. 2019; Available from: <https://www.nice.org.uk/advice/mib182>.
46. Fletcher J, Moore Z, Smit G. Early detection technology transforms care and releases productivity: An NHS case study. *Wounds UK*, 2017; 13(1): 74-78.
47. VanGilder C, MacFarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. *Ostomy Wound Manage*, 2008; 54(2): 40-54.
48. Baumgarten M, Margolis D, van DC, Gruber-Baldini A, Hebel J, Zimmerman S, Magaziner J. Black/White differences in pressure ulcer incidence in nursing home residents. *J Am Geriatr Soc*, 2004; 52(8): 1293-1298.
49. McCreath HE, Bates-Jensen BM, Nakagami G, Patlan A, Booth H, Connolly D, Truong C, Woldai A. Use of Munsell color charts to measure skin tone objectively in nursing home residents at risk for pressure ulcer development. *J Adv Nurs*, 2016; 72(9): 2077-2085.
50. Scheiner J, Farid K, Raden M, Demisse S. Ultrasound to detect pressure-related deep tissue injuries in adults admitted via the emergency department: A prospective, descriptive, pilot study. *Ostomy Wound Manage*, 2017; 63(3): 36-46.
51. Swaine JM, Moe A, Bredahl W, Bader DL, Oomens CWJ, Lester L, O'Loughlin E, Santamaria N, Stacey MC. Adaptation of a MR imaging protocol into a real-time clinical biometric ultrasound protocol for persons with spinal cord injury at risk for deep tissue injury: A reliability study. *J Tissue Viability*, 2018; 27(1): 32-41.
52. Helvig EI, Nichols LW. Use of high-frequency ultrasound to detect heel pressure injury in elders. *J Wound Ostomy Continence Nurs*, 2012; 39(5): 500-508.
53. Porter-Armstrong AP, Adams C, Moorhead AS, Donnelly J, Nixon J, Bader DL, Lyder C, Stinson MD. Do High Frequency Ultrasound Images Support Clinical Skin Assessment? *International Scholarly Research Network Nursing*, 2013: 1-5.
54. Grap MJ, Schubert CM, Burk RS, Lucas V, Wetzel PA, Pepperl A, Munro CL. High frequency ultrasound sacral images in the critically ill: Tissue characteristics versus visual evaluation. *Intensive Crit Care Nurs*, 2017; 42: 62-67.
55. Quintavalle PR, Lyder CH, Mertz PJ, et al. Use of high-resolution, high-frequency diagnostic ultrasound to investigate the pathogenesis of pressure ulcer development. *Adv Skin Wound Care*, 2006; 19(9): 498-505.
56. Hagblad J, Folke M, Linden M. Long term monitoring of blood flow at multiple depths - Observations of changes. *Stud Health Technol Inform*, 2012; 177: 107-12.
57. Hagblad J, Lindberg LG, Kaisdotter Andersson A, Bergstrand S, Lindgren M, Ek AC, Folke M, Linden M. A technique based on laser Doppler flowmetry and photoplethysmography for simultaneously monitoring blood flow at different tissue depths. *Med Biol Eng Comput*, 2010; 48(5): 415-422.
58. Ceylan B, Gunes UY, Uyar M. Examination of sacral tissue oxygen saturation among immobile patients. *J Clin Nurs*, 2017; 08: 08.
59. Kim JT, Wang X, Ho C, Bogie K. Physiological measurements of tissue health; Implications for clinical practice. *Int Wound J*, 2012; 9(6): 656-64.
60. Borzdynski CJ, McGuinness W, Miller C. Comparing visual and objective skin assessment with pressure injury risk. *Int Wound J*, 2016; 13(4): 512-8.

PREVENTIVE SKIN CARE

Introduction

Maintaining skin integrity is essential in the prevention of pressure injuries. Maintaining healthy skin requires comprehensive assessment and care planning. Nutrition and hydration, addressed in the *Nutrition* chapter of this guideline, play an important role in skin health. Appropriate management of other skin conditions (e.g., eczema, incontinence-associated dermatitis) is also an imperative in maintaining skin integrity and ability to protect underlying tissues.

This chapter addresses direct care of the skin to reduce the risk of pressure injuries. Preventive skin care not only protects the skin and promotes comfort, but also provides an opportunity to conduct a skin assessment and identify areas at risk that may require further preventive care and/or changes to the individual's overall pressure injury prevention plan.

Clinical Question

The clinical questions that guided the development of this chapter were:

- Is massage effective in preventing pressure injuries?
- Are topical products effective in preventing pressure injuries?
- Are prophylactic dressings effective for preventing pressure injuries?
- Are continence management strategies effective in preventing and treating pressure injuries?
- Are low friction or microclimate control fabrics effective for preventing pressure injuries?

Skin Hygiene

3.1: Implement a skin care regimen that includes:

- Keeping the skin clean and appropriately hydrated
- Cleansing the skin promptly after episodes of incontinence
- Avoiding use of alkaline soaps and cleansers
- Protecting the skin from moisture with a barrier product.

(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

Two studies^{1,2} provided evidence to support a recommendation to implement structured skin care regimen that includes regular cleansing (particularly after episodes of incontinence). A low quality Level 2 study¹ found that a structured hygiene program was associated with a lower incidence of pressure injuries than standard care. A low quality level 4 observational study² noted that skin was assessed as being healed or healing when a structured skin care regimen was implemented. A moderate quality Level 1 study³ reported significant reductions in erythema and broken skin when a pH-balanced (pH 5.5) foam cleanser was used, as compared to standard hospital soap. The structured skin care regimen reported in the low quality level 2 study¹ also included replacing soap with a pH balanced (pH not reported) foam cleanser.

Implementation Considerations

- Frequency of cleansing should be individualized (*Expert opinion*).
- When washing, drying and applying a barrier product, avoid vigorous massage or rubbing of the skin that can damage the skin due to friction^{4,5} (*Level 1 and Level 5*).
- Consider using a non-rinse skin cleanser (*Expert opinion*).

Evidence Discussion

Cleansing the skin removes dirt, sebum and other unwanted substances from the skin's surface. Frequency of cleansing should be individualized; over-cleansing can cause the skin to become dry due to impairment of the skin's natural moisturizing factor and barrier function.⁶

Ensure the skin is dry after cleansing, paying particular attention to skin folds and select soft fabrics for washers and towels to prevent damage from friction during drying. It is important to note that skin damage from moisture is not a pressure injury, but that presence of skin damage from moisture may increase the risk of pressure injuries. The mechanical properties of the stratum corneum are changed by the presence of moisture and as a function of temperature. The stiffness of the stratum corneum strongly depends on the stratum corneum hydration. The strain at which the stratum corneum breaks is approximately four times higher at 100% humidity, compared to dry skin. Humidity also increases the friction coefficient between the skin and supporting surface, thus enhancing the risk of shear damage.⁷⁻¹⁴

Structured skin hygiene programs

Two studies^{1,2} explored the effectiveness of structured skin hygiene programs in preventing pressure injuries. In the first study,¹ individuals in critical care who had fecal incontinence (n = 76) received either a structured skin care regimen that included use of mild washing with minimal friction, use of wet tissue cloth, regular perineal cleansing with a foaming cleanser followed by a barrier cream and moisturizing or standard care (details not reported). Over seven days, there was a significantly lower incidence of pressure injuries in the group receiving the structured skin hygiene protocol (13.2% versus 50%, p = 0.001). There was no blinded assessment and the nurses delivering the intervention were responsible for assessing outcomes¹ (Level 2).

Additional evidence comes from a case series study conducted in individuals with a moderate or high pressure injury risk on the Braden Scale (n = 20). The participants received one of two different structured skin hygiene regimens based on the assessment of their skin condition (erythema versus moisture lesion versus combination). All regimens included washing, use of a foam cleansing spray, a barrier product and fecal incontinence management based on type on the Bristol Stool Chart. Individuals with an existing moisture lesion received a prophylactic dressing. After between 3 to 28 days, all individuals had skin classified as healed (80%) or healing (20%)² (Level 4).

Skin cleansers

The pH of the skin at the surface measured when refraining from washing or using cleansers ranges from 4.0 to 7.0 (slightly acidic to neutral).¹⁵ Avoiding use of an alkaline soap or cleanser reduces potential dryness, erythema and irritation that can arise due to interaction between high pH soap products and the proteins and lipids on the skin's surface.⁶

As noted above, the study by Park et al. (2014)¹ demonstrated a statistically significant reduction in pressure injuries associated with a skin hygiene regimen that included a pH balanced (pH 5.5) foam cleanser (Level 2). Cooper et al. (2001)³ investigated standard hospital soap (1% aqueous solution with a pH of 9.5 to 10.5; n = 49) compared to a foam no-rinse cleanser (combination of an emollient, water-repellant deodorant and water-repellant barrier with a pH of 5.5; n = 44). The study was conducted over 14 days and participants all had some form of incontinence or catheterization. Skin was assessed using the Stirling Pressure Severity Scale and classified as broken skin (Category/Stage II or greater pressure injury), erythematous (Category/Stage I pressure injury) or healthy (no alterations to skin integrity). Overall, skin condition was maintained or improved for more participants receiving the skin cleanser compared with soap (66% versus 37%, p = 0.05). Participants who were classified as having healthy skin at commencement of the trial experienced more erythema (30.3% versus 15.1%, p = not reported) and more broken skin (12.1% versus 0%, p = not reported) when their skin was cleansed with soap and water. Although the median lengths of stay in care facilities were significantly different between the groups, the condition of skin was not significantly different between the groups on entry to the study³ (Level 1).

Skin moisturizing

The small body of evidence on products to moisturize and protect the skin primarily compares different products. One randomized controlled trial (RCT) found that a hyperoxygenated fatty acid moisturizer was not more effective than a placebo product for reducing pressure injuries¹⁶ (Level 1). Three RCTs¹⁷⁻¹⁹ indicated that there is also no statistically significant difference between different moisturizer or emollient products in preventing pressure injuries in individuals at moderate to high risk (Level 1).

However, the evidence comparing moisturizing the skin to not moisturizing the skin as a strategy to prevent pressure injuries is conflicting. One study conducted with individuals in a medical ward found that application of a silicone-based dermal nourishing moisturizer was more effective in reducing the pressure injury incidence than no emollient or moisturizer (7% versus 31%, p = 0.008)²⁰ (Level 3). A second study conducted in a community hospital with individuals at high risk of pressure injuries (Braden Scale score ≤ 15) compared a hyperoxygenated fatty acid moisturizer to placebo cream for preventing pressure injuries. There was no statistically significant difference between the hyperoxygenated fatty acid moisturizer and placebo cream (6.1% versus 7.4%, p = 0.94)¹⁶ (Level 1). However, the research is difficult to interpret due to the range of different products used, as well as methodological limitations of the studies.

Although research directly linking skin moisturizing to reduction in pressure injury incidence is lacking, one epidemiological study in hospitalized individuals with limited mobility (n = 286) noted that dry skin was a significant and independent risk factor for pressure injuries in a multivariate analysis²¹ (*Level 3 prognostic*). Regular application of a moisturizer in a skin hygiene regimen is suggested for promoting skin hydration and preventing other adverse skin conditions, including dry skin and skin tears.

3.2: Avoid vigorously rubbing skin that is at risk of pressure injuries.
(Good Practice Statement)

Discussion

In the past, massage has been used as a method of pressure injury prevention.^{4,22,23} Vigorous massage has the potential to damage tissue. Various types of massage use combinations of different stroke types including:²⁴

- Effleurage – slow, gentle gliding strokes that use firm pressure
- Pertissage – forceful kneading and skin rolling used on fleshy body regions
- Tapotement – striking and percussive movements
- Friction – compressive, penetrating pressure
- Vibration – shaking or vibrating motions.

Friction massage involves the use of penetrating pressure and is a vigorous type of rubbing described in older nursing texts.²⁵ As well as being painful, it can cause mild tissue destruction or provoke inflammatory reactions, particularly in frail older adults. Early work by Dyson et al. (1978)⁵ examining skin biopsies taken at post-mortem found cellular damage in areas where the skin had been rubbed more vigorously compared to biopsies taken from individuals who had not had their skin rubbed (*Level 5*). However, even less vigorous massage techniques are contraindicated in the presence of acute inflammation and where there is the possibility of damaged blood vessels or fragile skin.^{24,26}

In a randomized crossover trial,^{4,27} older adults (n = 79) were assigned to three study groups. One group received massage with a placebo cream, another group received massage with 5% dimethyl sulfoxide cream (DMSO), and the control group received no massage or cream application. The massage was conducted using soft, circular motions with a gloved hand (effleurage) to the coccyx, heels and ankles. No significant difference in overall pressure injury incidence was found between the three regimens. The researchers found no benefit from the use of massage; and there may have been some advantage to not massaging the individual.²⁷ There was a statistically significantly higher incidence of heel and ankle (but not coccyx) pressure injuries in the group that received massage with 5% DMSO cream (odds ratio [OR] 8.80, 95% confidence interval [CI] 2.61 to 29.6)⁴ (*Level 1*).

Continence Management

3.3: Use high absorbency incontinence products to protect the skin in individuals with or at risk of pressure injuries who have urinary incontinence.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

A low quality Level 1 study²⁸ provided evidence that highly absorbent disposable continence devices that lock moisture away from the skin are associated with a lower incidence of pressure injuries than reusable quilted incontinence pads. A low quality Level 3 study²⁹ reported a 67% reduction in risk of a pressure injury associated with using a highly absorbent incontinence diaper for ten weeks.

Implementation Considerations

- Together with the individual and their informal caregivers, develop a continence management plan that facilitates individualized toileting and/or regular skin care and change of continence pads to protect the skin from the impact of excess moisture and chemical irritants (*Expert opinion*).
- When barrier products (e.g., zinc and petrolatum) are applied excessively moisture wicking and absorbency properties of incontinence products may be inhibited. Review and follow the manufacturer information on incontinence products (*Expert opinion*).

Evidence Discussion

Incontinence can lead to prolonged skin exposure to excess moisture and chemical irritants in urine and feces. In addition, occlusion resulting from the use of an incontinence aid can alter the microclimate of the skin. The overall result can be inflammation, erythema, erosion, and denudation with decreased tolerance to other forms of skin damage, such as that associated with prolonged exposure to pressure or shear.³⁰ The relationship between skin moisture (and potentially skin pH from exposure to urine and feces) from incontinence is discussed in the guideline chapter on *Risk Factors and Risk Assessment*. An incontinence management plan aims to reduce the incidence of incontinent episodes. Together with prompt cleansing (see *Recommendation 3.1*), use of high absorbency incontinence products reduces the duration of skin exposure to irritants. Two studies^{28,29} provide evidence to suggest this also translates to a reduction in pressure injury incidence.

In a large trial, individuals in medical and surgical wards (n = 462) who had fecal and/or urinary incontinence were randomized to receive either disposable waterproof super-absorbent incontinence products or a reusable quilted underpad made from moderately absorptive fabric and a waterproof back.²⁸ The participants were older adults (mean age around 79 years) and all had incontinence associated dermatitis at the commencement of the study. The pressure injury incidence was statistically significantly lower in the group receiving the highly absorbent disposable incontinence product (4.8% versus 11.5%, p = 0.02). However, the findings were confounded by the large between-group differences in the use of other continence management interventions (e.g., toileting program, indwelling catheters and fecal incontinence devices), the significantly shorter mean hospital stay in the disposable product group (6 days versus 8 days, p = 0.02) and the lower incidence of pressure injuries in the disposable product group at baseline (33% versus 44%, p = 0.03)²⁸ (*Level 1*).

Teerawattananon et al. (2015)²⁹ conducted a small cohort study in incontinent adults in a rehabilitation setting (n = 71). High absorption disposable incontinence products were used over a period of ten weeks. Over time, the risk that a pressure injury would be present significantly decreased compared to baseline by week six (58% reduction in risk, 95% CI 8% to 75%) and week ten (67% reduction in risk, 95% CI 16% to 78%). By ten weeks, the mean difference compared to baseline in Braden Scale score was 0.19 (95% CI -0.42 to 0.79). The confidence intervals were wide, and one spanned the null value,²⁹ and the researchers noted that the high cost of the intervention (approximately \$650 million US in Thailand in 2015) was not sustainable (*Level 3*).

In individuals with significant incontinence, catheterization is sometimes implemented to promote skin hygiene. However, indwelling catheters (IDCs) are associated with increased risk of medical device related pressure injuries³¹ and urinary tract infections,³² so the benefits versus the risk of harm should be considered carefully according to the individual's clinical condition. If an IDC is used, the recommendations in the chapter on *Device Related Pressure Injuries* provide guidance on minimizing the risk of medical device related pressure injuries.

Fecal Incontinence

Two studies provided evidence on management of fecal incontinence; however, neither provided sufficient evidence that any specific intervention is more effective in preventing pressure injuries than standard care consisting of regular hygiene. In one RCT conducted in acutely ill individuals with neurogenic fecal incontinence (n = 100),³³ a significant reduction in Category/Stage I pressure injuries was associated with use of a suspension positioning device that elevates the perianal region, thereby reducing exposure to urinary and fecal material. There was no statistically significant reduction in Category/Stage III or IV pressure injuries. Although this strategy was somewhat effective, the feasibility and acceptability of the intervention was not explored (*Level 1*).

In a second RCT, Pittman et al. (2012)³⁴ explored the use of two fecal management devices, a bowel management system catheter (BMS group, n = 21) and a rectal trumpet utilized as a fecal incontinence device (RT group, n = 20) compared with usual care (n = 18). The study was conducted until the end-point of device failure (three or more incontinent stools in a 24 hour period), device complications (including rectal bleeding) or discharge from the critical care unit. There was no significant difference in the number of pressure injuries present in any of the groups (BMS 42.9% versus RT 35% versus usual care 27.8%, p = 0.63). The relatively high rate of pressure injuries was contributed to by the high level of pressure injuries present on entry to the study (32% of participants). There was also a wide variation in time spent in the study (from 2 days to 60 days), which may have influenced the findings (*Level 1*).

Bed Linen

3.4: Consider using textiles with low friction coefficients for individuals with or at risk of pressure injuries. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Implementation Considerations

- Introduction of low co-efficient linen may require facility-level inventory change that should be accompanied by education for health professionals, patient consumers and informal caregivers³⁵ (*Level 1*).

Evidence Summary

The evidence for the recommendation to consider using a low friction coefficient textile is primarily based on studies exploring the effectiveness of silk-like fabrics that reduce shear stress, minimizes skin irritation and dries quickly when compared to a cotton or cotton-blend fabric. One moderate quality Level 1 study reported a hazard ratio of 0.23 (with wide confidence intervals) for Category/Stage II or greater pressure injuries associated with silk-like fabric compared to cotton blend fabric.³⁵ A moderate quality Level 2 study³⁶ also found that silk-like fabric is associated with lower pressure injury incidence than standard cotton sheets. One moderate quality Level 3 study reported that a synthetic fiber was associated with a lower pressure injury risk due to its management of moisture compared to cotton sheets.³⁷ Three additional Level 3 studies (two moderate quality³⁸ and two of low quality^{39,40}) reported lower incidence of pressure injuries (both Category/Stage I and Category/Stage II and greater) in cohorts that were cared for on silk-like fabric sheets compared to standard linen. The range of effect varied between the studies but favored the low coefficient silk-like product. One cohort study reported that silk-like sheets cost more than double that of cotton-blend sheets, but lasted more than three times as long⁴⁰ A low quality cost analysis indicated there was a small cost saving associated with using silk-like fabrics.³⁹

Evidence Discussion

Six studies³⁵⁻⁴⁰ reported on the relationship between low friction coefficient textiles and pressure injuries in acute care,³⁶⁻³⁸ critical care^{39,40} and aged care settings.³⁵ Low friction coefficient textiles are synthetic silk-like fiber fabrics manufactured to create a smooth, quick-drying fabric interface. Used for either bed linen or clothing (or both), the individual using low friction fabrics experiences a reduction in friction force and shear stress as they move or slide on the bed linen surface.³⁸ The synthetic clothing, fiber sheets and underpads and clothing can have more than one layer that absorbs moisture from perspiration away from the skin, reducing heat insulation and influencing that microclimate.³⁷ This is important because the coefficient of friction has been shown to be greater over moist skin.⁴¹⁻⁴³ Although concern has been expressed regarding a risk of slips or falls from a low friction coefficient fabric, the available evidence has shown no increase in adverse events related to falling or sliding.^{35,38}

In a multivariable analysis from a prognostic study (n = 71),³⁷ the type of sheeting the individual received was one of two significant factors for experiencing a pressure injury (the other being Braden Scale score). Individuals who received 100% cotton sheeting were more likely to develop a pressure injury than those who had a synthetic fiber sheet (OR 0.11, 95% CI 0.012 to 1.032, p = 0.00).³⁷ However, the confidence interval spans the null value, suggesting caution in considering the results (*Level 3*).

Five comparative studies^{35,36,38-40} provided evidence for the effectiveness of low friction coefficient bed linen and clothing. In a randomized trial (n = 46) conducted in older adults in a nursing home setting,³⁵ individuals were assigned to either low coefficient, silk-like linen (i.e., bed sheets, under pads and pillowcases) or to usual care that consisted of plain weave linen. Incontinent individuals also received incontinence undergarments that differed in construction between the two groups, with a higher absorbency product used in the intervention group. At the 20-week follow-up the intervention group had experienced significantly fewer pressure injuries of any Category/Stage (hazard ratio [HR] = 0.31, 95% CI 0.12 to 0.78, p = 0.0125). The low coefficient fabric group also experienced fewer Category/Stage II or greater pressure injuries (HR = 0.23, 95% CI 0.078 to 0.69, p = 0.0084).³⁵ The confidence intervals for both these results were wide. Use of higher absorbency incontinence products may have contributed to the effect of the fabric, as this product would contribute to the microclimate changes at the skin-surface interface. Consideration to the type of continence product used might be important in maximizing the impact of specialist fabrics.³⁵ Additionally, the intervention included an extensive education program for health professionals and patient consumers, and required changes to inventory and customized sheets for individuals in the intervention group who required specialty beds.³⁵ These factors are considerations in generalizing the effectiveness of the intervention and feasibility in its implementation in other settings.

In a second study,³⁶ an eight-week, controlled trial, silk-like linen was associated with a lower incidence of pressure injuries among individuals in medical/surgical wards, as compared to cotton-blend linen. In the medical unit, the incidence of new pressure injuries was statistically significantly lower (4.6% versus 12.3%, $p = 0.01$) and there was a non-significant reduction in the average length of stay (5.31 versus 5.97 days, $p = 0.07$) in the group receiving silk-like sheets. The surgical ICU unit showed similar results with a statistically significant decrease in pressure injury incidence (0% versus 7.5%, $p = 0.01$), and a non-significant reduction in the average length of stay ($p = 0.33$) (Level 2).

A cohort study³⁹ specifically explored the use of low friction coefficient fabric undergarments or booties ($n = 165$) compared to regular hospital garments ($n = 204$). The incidence of hospital acquired pressure injuries was significantly lower in the second cohort (25% versus 41%, $p = 0.02$). There was also a lower rate of wound deterioration in the low friction fabric cohort for individuals with a pre-existing pressure injury (6% versus 25%, $p = 0.001$)³⁹ (Level 3).

Two cohort studies compared silk-like fabrics to a cotton blend fabric. In the first of these cohort studies ($n = 1,427$)³⁸ individuals in an historical cohort receiving usual cotton blend linen experienced a higher incidence of hospital-acquired Category/Stage I pressure injuries than individuals on the silk-like bed linen (5.6% versus 2.3%, $p < 0.001$). The silk-like bed linen was also associated with a statistically significantly lower rate of Category/Stage II pressure injuries (0.8% versus 5.95%, $p < 0.001$) (Level 3). In the second of these cohort studies ($n = 1,647$)⁴⁰ individuals in intensive care receiving low coefficient fabric sheets and underpads experienced fewer pressure injuries over 20 weeks compared to individuals who received cotton blend linen (5.26% versus 7.71%, $p = 0.002$). The incidence of Category/Stage II or greater pressure injuries was also lower for the cohort receiving low coefficient fabric (2.82% versus 5.25%, $p < 0.001$) (Level 3). This cohort study reported that the intervention was associated with a \$3.9 million (USD in 2015) cost saving due to the shorter hospitalization duration achieved by preventing pressure injuries.⁴⁰ However, this cost analysis included no details of the costs considered in the analysis. A more detailed cost analysis³⁹ that considered community hospital costs, support surfaces and wound dressings estimated a cost savings £63,000 per 100 at risk patients (UK pounds in 2010) from using a low friction coefficient linen. Therefore, potential cost savings could vary across clinical and geographic settings.

Prophylactic Dressings

3.5: Use a soft silicone multi-layered foam dressing to protect the skin for individuals at risk of pressure injuries. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence supporting the effectiveness of a multi-layer silicone foam dressing in protecting the skin and preventing pressure injuries comes from one high quality,⁴⁴ and four moderate quality^{18,45-47} Level 1 studies, a high quality Level 2 study,⁴⁸ high⁴⁹ and low quality^{50,51} Level 3 studies, all of which reported statistically significantly lower pressure injury incidence compared to using no prophylactic dressing in individuals who were at moderate to very high risk of pressure injury. In one of the moderate quality Level 1 studies,¹⁸ the results were only significant in individuals with a Braden Scale score below 12 (i.e., high risk of pressure injuries). Another low quality Level 3 study⁵¹ reported a reduction in sacral pressure injuries (particularly Category/Stage III and IV pressure injuries) when a multi-layer silicone foam dressing was used, although the difference compared to no prophylactic dressing was not statistically significant. The highest quality study reported an 88% reduction in pressure injury incidence associated with including a multi-layer silicone foam dressing in a skin care bundle.⁴⁴ Two studies (Level 2⁵² and Level 3⁵³) reported reductions in pressure injury incidence using a multi-layer silicone foam dressing that were not statistically significant compared to no prophylactic dressing, and both were low quality. Only one study⁵⁴ offered a comparison between a multi-layer silicone foam dressing and other prophylactic dressings; this high quality Level 2 study⁵⁴ found a multi-layer silicone foam dressing was associated with a statistically significantly lower pressure injury incidence compared to a polyurethane film dressing. Two economic analyses conducted in the US⁵⁰ and Australia^{55,56} suggested that introduction of a multi-layer silicone foam dressing to preventive care could be associated with substantial cost savings.

Implementation Considerations

- Continue to implement other measures (e.g., regular repositioning and support surfaces) to prevent pressure injuries when using a prophylactic dressing (*Expert opinion*).
- Continue to assess the skin under a prophylactic dressing at least daily to evaluate the effectiveness of the preventive care regimen. Many dressings have features that facilitate regular skin assessment (e.g. transparency,⁵⁷ silicone borders,^{46,49} non-adhesive edges,^{58,59} etc.) (Levels 1 and 3).

- When selecting a prophylactic dressing consider:
 - Potential benefit of using a dressing
 - Appropriateness of the size and design of the dressing
 - Ability to manage the microclimate
 - Ease of application and removal
 - Ability to maintain the dressing in situ
 - Ability to regularly assess the skin under the dressing
 - The individual's preferences, comfort and any allergies
 - Coefficient of friction at the skin-dressing interface
 - Cost-effectiveness and accessibility of dressings (*Expert opinion*).
- Replace the prophylactic dressing if it becomes dislodged, loosened or excessively moist,^{46,49} if the dressing or skin underneath become soiled, and according to the manufacturer's instructions (*Levels 1 and 3*).
- For individuals at high risk of pressure injuries, application of a prophylactic dressing should be initiated as early as possible in the care pathway when feasible (e.g., applied in the ambulance or emergency room)⁴⁶ (*Level 1*). Use of a prophylactic dressing on the heels is discussed in more detail in the chapter *Heel Pressure Injuries* and the chapter *Device Related Injuries* discusses use of a prophylactic dressing to prevent pressure injuries associated with devices.

Evidence Discussion

Ohura et al. (2005)⁶⁰ used an in vitro porcine model to measure pressure and shear forces on skin and subcutaneous tissue. Shear forces on both layers of tissue were lower when various prophylactic dressings were applied to the skin. The results of this study⁶⁰ were supported in a study with healthy volunteers.⁶¹ The study⁶¹ showed that ten different prophylactic dressings made from a variety of products including multi-layer foam and hydrocolloid, all contributed to a lower interface pressure compared to no prophylactic dressing. Characteristics of prophylactic dressings were further explored in laboratory studies that explored specific qualities that contributed to their ability to absorb shear and friction force.^{62,63} In their laboratory based study of prophylactic dressings, Call et al. (2013, 2015)^{62,63} reported that features such as an elastic-type adhesive (e.g., silicone), multiple layer construction and the size of the dressing all contribute to its ability to protect the skin^{62,63} (all *Level 5*). Research also indicates that application of a prophylactic dressing can influence the microclimate. Call et al. (2013)⁶³ noted that the construction of prophylactic dressings significantly influences moisture trapping and humidity close to the skin. Accumulation of moisture at the skin surface decreased the ability of some dressings to transpire. Although skin surface temperature was shown to increase with the application of prophylactic dressings, the heat rise was considered insufficient to place the skin at additional risk of injury⁶³ (*Level 5*). Prophylactic dressings differ in their qualities; therefore, it is important to select a dressing that is appropriate to the individual and the clinical use. Foam dressings have superior absorptive capacity than other types of prophylactic dressings, and most are designed to be easy to lift^{31,46,49} in order to assess the skin without causing trauma.^{45,64} This is of particular significance to older adults with fragile skin and neonates with immature skin, as discussed in the guideline chapter on *Special Populations*, which notes the risk of epidermal stripping from dressing products.

Clinical evidence on effectiveness of adding prophylactic dressings to prevention injury prevention regimens that include appropriate support surfaces and repositioning continues to grow. Most studies exploring prophylactic dressings for general use (i.e., applied to the heels, coccyx and sacrum) have compared multi-layered silicone foam dressings to no prophylactic dressing. Evidence on other types of prophylactic dressings, for example hydrocolloid or film dressings, specifically addresses prevention of heel pressure injuries or use to prevent pressure injuries associated with medical devices (particularly film dressings, which are thinner and can be more easily applied under devices). This evidence is discussed in the guideline chapters *Heel Pressure Injuries* and *Device Related Injuries*. However, in the single comparative study currently available, a multilayered silicone foam dressing was more effective than a film dressing in reducing the incidence of Category/Stage I pressure injuries (3% versus 11%, $p = 0.027$) in individuals undergoing surgery ($n = 100$)⁵⁴ (*Level 2*). The evidence discussed below relates to multi-layered silicone foam products compared to no prophylactic dressing.

The most studied population is immobile critically ill individuals in intensive care, in which six studies have demonstrated an association between prophylactic dressings and a lower incidence of pressure injuries compared to no dressing^{44-46,48,49,52} Kalowes et al. (2016)⁴⁴ reported an 88% reduction in risk of developing a pressure injury (hazard ratio [HR] = 0.12, 95% CI 0.02 to 0.98, $p = 0.048$) when a multi-layered silicone foam dressing was added to the critical care unit's pressure injury prevention bundle (*Level 1*). In a study in which a prophylactic dressing was applied to heels and sacrum on admission to the emergency department and use continued in the critical care unit ($n = 440$)⁴⁶ there was a significant reduction compared to usual care in overall pressure injury incidence (4.3% versus 17.8%, $p = 0.002$),

as well as significant decreases in sacral pressure injuries (1.2% versus 5.2%, $p = 0.05$) and heel pressure injuries (3.1% versus 12.5%, $p = 0.002$). The number-needed-to-treat (NNT) was 10 to prevent any pressure injury⁴⁶ (Level 1). Park (2014)⁴⁸ showed a lower pressure injury incidence for multi-layered foam dressing compared to no dressing (6% versus 46%, $p < 0.001$)⁴⁸ (Level 2) and in a cohort study ($n = 302$), Santamaria et al. (2015) noted that a multi-layer silicone foam dressing completely prevented heel pressure injuries in critically ill individuals (0% versus 9.2%, $p < 0.001$) (Level 3).

Only a few studies failed to demonstrate a significant effect for a multi-layered silicone foam dressing. In a study by Brindle and Wegelin (2012),⁴⁵ there was a reduction in sacral pressure injuries associated with application of the prophylactic dressings, but the finding was not statistically significant (2% versus 11.7%, $p > 0.05$). However, the trend indicated a positive effect, and the small study ($n = 85$) was underpowered to measure significance⁴⁵ (Level 1). Another study⁵² failed to show significant reduction in pressure injuries for critically ill individuals receiving a multi-layer silicone foam dressing to the sacrum, buttock and coccyx (incident rate ratio [IRR] ranged from 0.41 to 0.54, $p > 0.05$). However, this study was of lower quality⁵² (Level 2).

Acutely ill individuals in medical or surgical wards also appear to achieve benefit from a multi-layered silicone foam dressing. Padula et al. (2017)⁵⁰ reported a significant reduction in Category/Stage III, IV or unstageable pressure injuries for a multi-layered silicone foam dressings compared to no dressing (1.2 ± 0.045 versus 1.5 ± 0.125 , $p = 0.0063$)⁵⁰ (Level 3). No individuals undergoing trauma surgery who received a prophylactic dressing experienced a Category/Stage III or IV pressure injuries compared to rates of 2.5% and 5% specifically in cohorts not receiving a dressing (Level 3). However, it might be that benefit is only achieved for individuals who are assessed as having a high risk of pressure injuries (Level 3).¹⁸ In a study by Aloweni et al. (2017),¹⁸ a comparison between a multi-layered foam prophylactic dressing and no dressing was only significant upon restricting the analysis to individuals with a Braden Scale score indicative of high pressure injury risk (Braden Scale score ≤ 12 , 0% versus 4.8%, $p = 0.048$)¹⁸ (Level 1). Cubit et al. (2013)⁵³ failed to demonstrate a reduction in sacral pressure injuries for hospitalized individuals ($n = 109$) treated with a multi-layer silicone foam dressing compared to no dressing. However, there was a non-significant trend toward reduction (1.96% versus 10.3%, $p < 0.08$) and level of pressure injury risk for participants was unclear⁵³ (Level 3).

Finally, in older adults ($n = 1,888$) there also appears to be a place for using prophylactic dressings.⁴⁷ There was a statistically significantly lower incidence of Category/Stage I or greater pressure injuries of the sacrum or heels compared to no dressing (2.1% versus 10.6%, $p = 0.004$). However, when the analysis was restricted to specific anatomical locations, there was a significant reduction in sacral pressure injuries (1.45% versus 8.67%, $p = 0.007$) but not pressure injuries of the heels only ($p > 0.05$)⁴⁷ (Level 1). This suggests that further research on refining the use of prophylactic dressings to both population groups and anatomic locations to determine who will achieve the most benefit is required.

References

1. Park KH, Kim KS. Effect of a structured skin care regimen on patients with fecal incontinence: A comparison cohort study. *J Wound Ostomy Continence Nurs*, 2014; 41(2): 161-167.
2. Bateman SD, Roberts S. Moisture lesions and associated pressure ulcers: Getting the dressing regimen right. *Wounds UK*, 2013; 9(2): 97-102.
3. Cooper P, Gray D. Comparison of two skin care regimes for incontinence. *Br J Nurs*, 2001; 10(6): S6-S20.
4. Duimel-Peeters I, Halfens R, Ambergen A, Houwing R, Berger P, Snoeckx L. The effectiveness of massage with and without dimethyl sulfoxide in preventing pressure ulcers: A randomized, double-blind cross-over trial in patients prone to pressure ulcers. *Int J Nurs Stud*, 2007; 44(8): 1285-1295.
5. Dyson R. Bed sores - the injuries hospital staff inflict on patients. *Nurs Mirror*, 1978; 146(24): 30-32.
6. Ananthapadmanabhan K, Moore D, Subramanyan M, Meyer F. Cleansing without compromise: The impact of cleansers on the skin barrier and the technology of mild cleansing. *Dermatol Ther*, 2004; 17: 16-25.
7. Koutroupi KS, Barbenel JC. Mechanical and failure behaviour of the stratum corneum. *J Biomech*, 1990; 23(3): 281-287.
8. Gardner TN, Briggs GA. Biomechanical measurements in microscopically thin stratum comeum using acoustics. *Skin Res Technol*, 2001; 7(4): 254-261.
9. Yuan Y, Verma R. Measuring microelastic properties of stratum corneum. *Colloids and Surfaces B: Biointerfaces*, 2006; 48(1): 6-12.
10. Nicolopoulos CS, Giannoudis PV, Glaros KD, Barbenel JC. In vitro study of the failure of skin surface after influence of hydration and preconditioning. *Archives of Dermatological Research*, 1998; 290(11): 638-640.
11. Papir YS, Hsu KH, Wildnauer RH. The mechanical properties of stratum corneum. I. The effect of water and ambient temperature on the tensile properties of newborn rat stratum corneum. *Biochim Biophys Acta*, 1975; 399(1): 170-180.
12. Park AC, Baddiel CB. Rheology of stratum corneum-I: A molecular interpretation of the stress-strain curve. *Journal of the Society of Cosmetic Chemist*, 1972; 23(): 3-12
13. Wildnauer RH, Bothwell JW, Douglass AB. Stratum corneum biomechanical properties. I. Influence of relative humidity on normal and extracted human stratum corneum. *J Invest Dermatol*, 1971; 56(1): 72-78.

14. Wilkes GL, Brown IA, Wildnauer RH. The biomechanical properties of skin. *CRC Crit Rev Bioeng*, 1973; 1(4): 453-495.
15. Schreml S, Szeimies R-M, Karrer S, Heinlin J, Landthaler M, Babilas P. The impact of the pH value on skin integrity and cutaneous wound healing. *J Eur Acad Dermatol Venereol*, 2009; 24(4): 373-78.
16. Verdú J, Soldevilla J. IPARZINE-SKR study: Randomized, double-blind clinical trial of a new topical product versus placebo to prevent pressure ulcers. *Int Wound J*, 2012; 9(5): 557-565.
17. Lupianez-Perez I, Uttumchandani SK, Morilla-Herrera JC, Martin-Santos FJ, Fernandez-Gallego MC, Navarro-Moya FJ, Lupianez-Perez Y, Contreras-Fernandez E, Morales-Asencio JM. Topical olive oil is not inferior to hyperoxygenated fatty acids to prevent pressure ulcers in high-risk immobilised patients in home care. Results of a multicentre randomised triple-blind controlled non-inferiority trial. *PLoS One*, 2015; 10(4).
18. Aloweni F, Lim ML, Chua TL, Tan SB, Lian SB, Ang SY. A randomised controlled trial to evaluate the incremental effectiveness of a prophylactic dressing and fatty acids oil in the prevention of pressure injuries. *Wound Practice & Research*, 2017; 25(1): 24-34.
19. Bou J, Segovia G, Verdu S, Nolasco B, Rueda L, Perejamo M. The effectiveness of a hyperoxygenated fatty acid compound in preventing pressure ulcers. *J Wound Care*, 2005; 14(3): 117-21.
20. Shannon RJ, Coombs M, Chakravarthy D. Reducing hospital-acquired pressure ulcers with a silicone-based dermal nourishing emollient-associated skincare regimen. *Adv Skin Wound Care*, 2009; 22(10): 461-467.
21. Allman R, Goode P, Patrick M, Burst N, Bartolucci A. Pressure ulcer risk factors among hospitalized patients with activity limitation. *J Am Med Assoc*, 1995; 273(11): 865-70.
22. Acaroglu R, Sendir M. Pressure ulcer prevention and management strategies in Turkey. *Journal of Wound Ostomy and Continence Nursing*, 2005; 32(4): 230-7.
23. Panagiotopoulou K, Kerr S. Pressure area care: An exploration of Greek nurses' knowledge and practice. *J Adv Nurs*, 2002; 40(3): 285-296.
24. Duimel-Peeters I, Halfens R, Berger P, Snoeckx L. The effects of massage as a method to prevent pressure ulcers. A review of the literature. *Ostomy Wound Management*, 2005; 51(4): 70-80.
25. Houghton M, *Aids to practical nursing*. 1940, London: Bailliere, Tindall & Cox.
26. Holey E, Cook E, *Evidence-based therapeutic massage: A practical guide for therapists*. 2nd ed. 2003, Edingburgh: Churchill Livingstone.
27. Houwing R, van der Zwet W, van Asbeck S, Halfens R, Arends JW. An unexpected detrimental effect on the incidence of heel pressure ulcers after local 5% DMSO cream application: A randomized, double-blind study in patients at risk for pressure ulcers. *Wounds*, 2008; 20(4): 84-88.
28. Francis K, ManPang S, Cohen B, Salter H, Homel P. Disposable versus reusable absorbent underpads for prevention of hospital-acquired incontinence associated dermatitis and pressure injuries. *J Wound Ostomy Continence Nurs*, 2017; 44(4): 374-379.
29. Teerawattananon Y, Anothaisintawee T, Tantivess S, Wattanadilokkul U, Krajaistri P, Yotphumee S, Wongviseskarn J, Tonmukayakul U, Khampang R. Effectiveness of diapers among people with chronic incontinence in Thailand. *Int J Technol Assess Health Care*, 2015; 31(4): 249-255.
30. Gray M, Beeckman D, Bliss DZ, Fader M, Logan S, Junkin J, Selekof J, Doughty D, Kurz P. Incontinence-associated dermatitis: A comprehensive review and update. *J Wound Ostomy Continence Nurs*, 2012; 39(1): 61-74.
31. Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Call E, Clark M. Use of wound dressings to enhance prevention of pressure ulcers caused by medical devices. *Int Wound J*, 2015; 12: 322-327.
32. Mody L, Greene MT, Meddings J, Krein SL, McNamara SE, Trautner BW, Ratz D, Stone ND, Min L, Schweon SJ, Rolle AJ, Olmsted RN, Burwen DR, Battles J, Edson B, Saint S. A national implementation project to prevent catheter-associated urinary tract infection in nursing home residents. *JAMA Internal Medicine*, 2017; 177(8): 1154-1162.
33. Su MY, Lin SQ, Zhou YW, Liu SY, Lin A, Lin XR. A prospective, randomized, controlled study of a suspension positioning system used with elderly bedridden patients with neurogenic fecal incontinence. *Ostomy Wound Management*, 2015; 61(1): 30-39.
34. Pittman J, Beeson T, Terry C, Kessler W, Kirk L. Methods of bowel management in critical care. *Journal of Wound Ostomy and Continence Nursing*, 2012; 39(6): 633-639.
35. Twersky J, Montgomery T, Sloane R, Weiner M, Doyle S, Mathur K, Francis M, Schmader K. A randomized, controlled study to assess the effect of silk-like textiles and high-absorbency adult incontinence briefs on pressure ulcer prevention. *Ostomy Wound Manage*, 2012; 58(12): 18-24.
36. Coladonato J, Smith A, Watson N, Brown AT, McNichol L, Clegg A, Griffin T, McPhail L, Montgomery TG. Prospective, nonrandomized controlled trials to compare the effect of a silk-like fabric to standard hospital linens on the rate of hospital-acquired pressure ulcers. *Ostomy Wound Manage*, 2012; 58(10): 14-31.
37. Yusuf S, Okuwa M, Shigeta Y, Dai M, Iuchi T, Sulaiman R, Usman A, Sukmawati K, Sugama J, Nakatani T, Sanada H. Microclimate and development of pressure ulcers and superficial skin changes. *Int Wound J*, 2013.
38. Smith A, McNichol LL, Amos MA, Mueller G, Griffin T, Davis J, McPhail L, Montgomery TG. A retrospective, nonrandomized, before and after study of the effect of linens constructed of synthetic silk-like fabric on pressure ulcer incidence. *Ostomy Wound Management*, 2013; 59(4): 28-34.
39. Smith G, Ingram A. Clinical and cost effectiveness evaluation of low friction and shear garments. *J Wound Care*, 2010; 19(12): 535-542.
40. Freeman R, Smith A, Dickinson S, Tschannen D, James S, Friedman C. Specialty linens and pressure injuries in high-risk patients in the intensive care unit. *Am J Crit Care*, 2017; 26(6): 474-481.
41. Vilhena L, Ramalho A. Friction of human skin against different fabrics for medical use. *Lubricants*, 2016 4(1): doi.org/10.3390/lubricants4010006

42. Klaassen M, Schipper D, Masen M. Influence of the relative humidity and the temperature on the in-vivo friction behaviour of human skin. *Biotribology*, 2016; 6: 21-28.
43. Bernatchez SF, Mengistu GE, Ekholm BP, Sanghi S, Theiss SD. Reducing Friction on Skin at Risk: The Use of 3MTM Cavilon™ No Sting Barrier Film. *Adv Wound Care*, 2015; 4(12): 705-710.
44. Kalowes P, Messina V, Li M. Five-layered soft silicone foam dressing to prevent pressure ulcers in the intensive care unit. *Am J Crit Care*, 2016; 25(6): e108-e119.
45. Brindle CT, Wegelin JA. Prophylactic dressing application to reduce pressure ulcer formation in cardiac surgery patients. *J Wound Ostomy Continence Nurs*, 2012; 39(2): 133-142.
46. Santamaria N, Gerdtz M, Sage S, McCann J, Freeman A, Vassiliou T, De Vincentis S, Ng AW, Manias E, Liu W, Knott J. A randomised controlled trial of the effectiveness of soft silicone multi-layered foam dressings in the prevention of sacral and heel pressure ulcers in trauma and critically ill patients: The Border trial. *Int Wound J*, 2015; 12(3): 302-308.
47. Santamaria N, Gerdtz M, Kapp S, Wilson L, Gefen A. A randomised controlled trial of the clinical effectiveness of multi-layer silicone foam dressings for the prevention of pressure injuries in high-risk aged care residents: The Border III Trial. *Int Wound J*, 2018; 15(3): 482-490.
48. Park KH. The effect of a silicone border foam dressing for prevention of pressure ulcers and incontinence-associated dermatitis in intensive care unit patients. *J Wound Ostomy Continence Nurs*, 2014; 41(5): 424-429.
49. Santamaria N, Gerdtz M, Liu W, Rakis S, Sage S, Ng AW, Tudor H, McCann J, Vassiliou J, Morrow F, Smith K, Knott J, Liew D. Clinical effectiveness of a silicone foam dressing for the prevention of heel pressure ulcers in critically ill patients: Border II Trial. *J Wound Care*, 2015; 24(8): 340-345.
50. Padula WV. Effectiveness and value of prophylactic 5-layer foam sacral dressings to prevent hospital-acquired pressure injuries in acute care hospitals: An observational cohort study. *Journal of Wound, Ostomy, and Continence Nursing*, 2017; 44(5): 413-419.
51. Richard-Denis A, Thompson C, Mac-Thiong JM. Effectiveness of a multi-layer foam dressing in preventing sacral pressure ulcers for the early acute care of patients with a traumatic spinal cord injury: Comparison with the use of a gel mattress. *Int Wound J*, 2017.
52. Byrne J, Nichols P, Sroczyński M, Stelmaski L, Stetzer M, Line C, Carlin K. Prophylactic sacral dressing for pressure ulcer prevention in high-risk patients. *Am J Crit Care*, 2016; 25(3): 228-234.
53. Cubit K, McNally B, Lopez V. Taking the pressure off in the Emergency Department: Evaluation of the prophylactic application of a low shear, soft silicon sacral dressing on high risk medical patients. *Int Wound J*, 2013; 10(5): 579-584.
54. Yoshimura M, Ohura N, Tanaka J, Ichimura S, Kasuya Y, Hotta O, Kagaya Y, Sekiyama T, Tannba M, Suzuki N. Soft silicone foam dressing is more effective than polyurethane film dressing for preventing intraoperatively acquired pressure ulcers in spinal surgery patients: The Border Operating room Spinal Surgery (BOSS) trial in Japan. *Int Wound J*, 2018; 15: 2.
55. Santamaria N, Liu W, Gerdtz M, Sage S, McCann J, Freeman A, Vassiliou T, Devincentis S, Ng AW, Manias E, Knott J, Liew D. The cost-benefit of using soft silicone multilayered foam dressings to prevent sacral and heel pressure ulcers in trauma and critically ill patients: A within-trial analysis of the Border trial. *Int Wound J*, 2014; epub.
56. Santamaria N, Santamaria H. An estimate of the potential budget impact of using prophylactic dressings to prevent hospital-acquired PUs in Australia. *J Wound Care*, 2014; 23(11): 583-589.
57. Souza TS, Reichembach Danski MT, Johann DA, Marques De Lazzari LS, Mingorance P. Prevention's pressure ulcers heel with transparent polyurethane film. *Acta Paulista de Enfermagem*, 2013; 26(4): 345-352.
58. Forni C, Loro L, Tremosini M, Mini S, Pignotti E, Bigoni O, Guzzo G, Bellini L, Trofa C, Guzzi M. Use of polyurethane foam inside plaster casts to prevent the onset of heel sores in the population at risk. A controlled clinical study. *J Clin Nurs*, 2011; 20(5/6): 675-680.
59. Torra I Bou JE, Rueda López J, Camañes G, Herrero Narváez E, Blanco Blanco J, Ballesté Torralba J, Martínez-Esparza EH, García LS, Soriano JV. Preventing pressure ulcers on the heel: a Canadian cost study. *Dermatol Nurs*, 2009; 21(5): 268-272.
60. Ohura N, Ichioka S, Nakatsuka T, Shibata M. Evaluating dressing materials for the prevention of shear force in the treatment of pressure ulcers. *J Wound Care*, 2005; 14(9): 401-404.
61. Matsuzaki K, Kishi K. Investigating the pressure-reducing effect of wound dressings. *J Wound Care*, 2015; 24(11): 512-517.
62. Call E, Pedersen J, Bill B, Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Clark M. Enhancing pressure ulcer prevention using wound dressings: What are the modes of action? *Int Wound J*, 2015; 12(4): 408-413.
63. Call E, Pedersen J, Bill B, Oberg C, Ferguson-Pell M. Microclimate impact of prophylactic dressings using in vitro body analog method. *Wounds* 2013; 25(4): 94-103.
64. Walsh NS, Blanck A, Smith L, Cross M, Andersson L, Polito C. Use of a sacral silicone border foam dressing as one component of a pressure ulcer prevention program in an intensive care unit setting. *J Wound Ostomy Continence Nurs*, 2012; 39(2): 146-149.

NUTRITION IN PRESSURE INJURY PREVENTION AND TREATMENT

Introduction

Nutrition plays a vital role in the prevention and treatment of pressure injuries because all organ systems require macro and micronutrients to meet nutrient requirements for growth, development, maintenance, and repair of body tissues. Well-nourished individuals are at lower risk for developing pressure injuries than malnourished individuals, however both well-nourished and undernourished individuals may develop skin integrity issues under certain circumstances.¹

Malnutrition is a condition in which a nutritional deficiency or an excess or imbalance of energy, protein, and other nutrients causes measurable adverse effects on tissue, body structure, body function, and clinical outcomes. Adult malnutrition usually occurs along a continuum of inadequate intake and/or increased nutrition requirements, impaired absorption, altered transport, and compromised nutrient utilization. Individuals may also have hypermetabolic and/or hypercatabolic and inflammatory conditions. The Academy and ASPEN² defines adult malnutrition as the presence of two or more of the following characteristics:

- Insufficient energy intake
- Unintended weight loss
- Loss of muscle mass
- Loss of subcutaneous fat
- Localized or generalized fluid accumulation
- Decreased functional status.

Unintended weight loss is a marker for malnutrition. One of the hallmarks of declining nutritional status is unplanned weight loss, which has been linked with increased risk for mortality in older adults.^{4,5,7,8} Moreover, anorexia of aging, a syndrome defined by a reduction in appetite and/or food consumption, weight loss, and altered metabolic state in older adults, can increase the risk for malnutrition and negative health outcomes.³ However, despite the frequent association of malnutrition with underweight, it is important to note that adults with obesity may also be poorly nourished.

The Academy and ASPEN⁴ defines pediatric malnutrition as the presence of one or more of the following characteristics when only one data point is available:

- Weight-for-height z score
- BMI-for-age z score
- Length/height-for -age z score
- Mid-upper arm circumference.

Primary indicators to use for pediatric individuals when two or more data points are available:

- Weight gain velocity (< 2 yr age)
- Weight loss (2-20 yrs of age)
- Deceleration in weight for length/height z score
- Inadequate nutrient intake.

Malnutrition and Pressure Injuries

Malnutrition can impact pressure injury development and healing. Both inadequate nutritional intake and undernutrition have been linked to the development of pressure injuries, pressure injury severity, and protracted healing.^{5-8 9-11}

As discussed in detail in the guideline section *Risk Factors and Risk Assessment*, poor nutritional status (malnutrition) and variables that indicate potential malnutrition (e.g., low body weight and poor oral food intake) are independent risk factors for the development of pressure injuries.¹²⁻¹⁴ Moreover, it appears that many individuals who are at risk of developing pressure injuries or have an established pressure injury suffer from unintended weight loss.^{5,12,14-16} International research identifies the association between nutritional status and pressure injuries. A study in the US evaluating the care process for hospitalized older adults at risk for pressure injuries (n = 2,425) noted that 76% were malnourished.¹⁷ In an Australian study, Banks et al. (2010)¹⁸ reported the odds ratio (OR) of having a pressure injury was 2.6 (95% confidence interval [CI] 1.8 to 3.5) for adults with malnutrition in acute care and 2.0 (95% CI 1.5 to 2.7) for adults with malnutrition in aged care. Iizaka et al. (2010)⁶ reported that the rate of malnutrition in older adults

receiving home care in Japan was significantly higher in those with a pressure injury (58.7% versus 32.6%, $p < 0.001$). From a large study ($n = 1,188$) in Belgium, the OR of an older adult with a pressure injury being malnourished was 5.02 (95% CI 1.69 to 14.92, $p < 0.01$).¹¹

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are accurate and effective methods for assessing nutritional status of individuals with or at risk of pressure injuries?
- What nutritional interventions are effective in preventing pressure injuries?
- Is there an ideal nutritional regimen to reduce the risk of pressure injuries, and if so, what should it include?
- Are any nutritional supplements (e.g. formulas, specific vitamins/minerals) effective in reducing risk of pressure injury development?
- What nutritional interventions are effective in supporting pressure injury healing?
- Is there an optimal nutritional regimen to promote healing of pressure injuries, and if so, what should it include?
- Are any specific oral nutritional supplements or formula effective in promoting healing of pressure injuries?

Nutrition Screening

4.1: Conduct nutritional screening for individuals at risk of a pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Direct evidence from a moderate quality Level 1 prognostic study¹⁹ and two Level 3 prognostic studies^{20,21} indicates that being identified as malnourished or at risk for malnutrition through nutritional screening is associated with being more likely to be at pressure injury risk and more likely to develop a pressure injury. Evidence from a low quality Level 3 study²² suggests that implementation of nutritional interventions occurs faster in individuals identified at nutritional risk through nutritional screening, and this is associated with up to 50% reduction in pressure injury rates, decreased length of hospital stay, which could lead to decreased healthcare costs.

Implementation Considerations

- Use a simple, valid and reliable nutritional screening tool²³ (Level 5).
- The standards of practice for the registered dietitian/nutritionist, through the nutrition care process, recommend for individuals to be screened on admission to a health care setting. It is advised to re-screen individuals with each significant change in their clinical condition; and/or if pressure injury healing trajectory is not as expected^{2,24} (*Expert opinion*).
- Any qualified member of the health care team may complete nutrition screening²³ (*Expert opinion*).
- The Mini Nutritional Assessment full version (MNA®) and the Malnutrition Universal Screening Tool (MUST) screening tools have good psychometric properties when used to screen nutritional status of individuals with or at risk of pressure injuries^{19,20} (Levels 1 and 4).
- The Nutrition Risk Screening (NRS) 2002, Rapid Screen and the Short Nutrition Assessment Questionnaire (SNAQ) have good psychometric properties when used to screen nutritional status of older adults^{23,25-27} (Level 5).
- The Seniors in the Community: Risk Evaluation for Eating and Nutrition (SCREEN-II AB) has good psychometric properties when used to screen nutritional status of older adults in community settings^{23,28} (Level 5).
- The Canadian Nutrition Screening Tool (CNST) has good psychometric properties when used to screen nutritional status of adults in acute care²⁹ (Level 5).
- Individuals identified as malnourished, with pressure injuries, at risk for developing pressure injuries, or with significant change in condition should be referred to the registered dietitian/nutritionist for an in-depth nutrition assessment^{2,24} (*Expert opinion*).

Evidence Discussion

Poor outcomes, including the risk of morbidity and mortality, are associated with malnutrition, hence the need to quickly identify and treat malnutrition when pressure injuries are present. Nutrition screening identifies individuals who require a comprehensive nutrition assessment due to characteristics that put them at potential nutritional risk.

The nutrition screening tool should be valid, reliable, and relevant to the patient group being assessed. The screening tool must be appropriate for establishing nutritional risk in all types of individuals, including those with fluid disturbances and those in whom weight and height cannot be easily measured.^{30,31} Any qualified member of the health care team may complete nutrition screening, and it should be conducted on admission to the facility, or at first visit in community settings. Commonly used screening tools to assess risk for malnutrition in adults include the MNA[®]^{19,26,32-35}, MUST²⁶ NRS,^{30,31} and SNAQ,²⁵ SCREEN[®],^{28,36} and CNST³⁷ all of which have been explored in validation studies (see *Table 7.1*). Additional discussion on nutrition screening in neonates and children, including screening tools for different child populations, is discussed in the section on *Nutrition Management in Neonates and Children* at the end of this chapter.

Table 7.1: Summary of nutrition screening tool validation studies

Nutrition Screening Tool	Evidence for identifying pressure injury risk status	Evidence for identifying factors associated with pressure injury risk	Clinical setting and level of evidence
Mini Nutritional Assessment full version (MNA [®]) ³⁸	Yes	Yes	Older adults in community settings ¹⁹ (Level 1) Older adults in long term care ³³ (Level 4) Older adults with pressure injuries and multiple comorbidities ³² (Level 4) Older adults at nutritional risk in long term care and community settings ³⁵ (Level 5) Older adults in acute care, long term care and community settings ²⁶ (Level 5)
Malnutrition Universal Screening Tool (MUST) ³⁹	No	Yes	Older adults in acute care, long term care and community settings ²⁶ (Level 5)
Nutrition Risk Screening (NRS) 2002 ⁴⁰	No	No	Adults in acute care ³⁰ (Level 5) Older adults in acute care, long term care and community settings ²⁶ (Level 5)
Short Nutrition Assessment Questionnaire (SNAQ) ^{41,42}	No	No	Adults in acute care ²⁵ (Level 5) Older adults in residential care ²⁵ (Level 5)
Seniors in the Community: Risk Evaluation for Eating and Nutrition (SCREEN [®]) ^{28,36}	No	No	Older adults in community settings ²⁸ (Level 5)
Canadian Nutrition Screening Tool (CNST) ³⁷	No	No	Adults in acute care ²⁹ (Level 5)

Nutrition Assessment

4.2: Conduct a comprehensive nutrition assessment for adults at risk of a pressure injury who are screened to be at risk of malnutrition and for all adults with a pressure injury.
(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

One low quality Level 2 study⁴³ provided evidence that a nutrition assessment, as one component of a complex nutritional intervention program, contributed to increased pressure injury healing as measured on the Bates-Jensen Wound Assessment Tool. Recognized standards of practice suggest that a comprehensive nutrition assessment involves a systematic process of collecting, verifying, and interpreting data related to nutritional status.⁴⁴

Implementation Considerations

- It is recommended that nutrition assessment is performed by a registered dietitian/nutritionist in collaboration with an interprofessional nutrition team⁴³ (*Level 2*).
- Include the following in a comprehensive nutrition assessment:
 - Food history and adequacy of nutritional intake
 - Anthropometric measures (height, weight and body mass index [BMI])
 - Weight history
 - Biochemical data (based on patient's diagnosis/conditions)
 - Medical tests and procedures
 - Nutrition focused physical assessment that includes muscle wasting, edema, micronutrient deficiencies, and functional status (e.g., handgrip)
 - Ability to eat independently⁴⁵ (*Level 3*).
- The following items are not recommended for establishing sensitive indicators/markers of nutritional status:
 - Serum albumin, prealbumin and other laboratory values may be useful in establishing overall prognosis but do not correlate well with clinical observation of nutritional status^{2,24,46-50} (*Level 5 and expert opinion*).
 - Serum protein levels may be affected by inflammation, renal function, hydration, and other factors so are not a good indicator of nutritional status^{2,24,49-51} (*Indirect evidence and expert opinion*).
- Inflammatory biomarkers are not recommended for diagnosis of malnutrition^{2,52} (*Expert opinion*).

Evidence Discussion

All adults screened to be at risk of malnutrition, as well as all individuals with a pressure injury should be referred to a registered dietitian/nutritionist or an interprofessional nutrition team (including, but not limited to, a physician, nurse, speech pathologist, occupational therapist, physical therapist and dentist), for a comprehensive nutrition assessment.⁵³ A comprehensive nutrition assessment is defined as a systematic process of collecting, verifying, and interpreting data related to nutritional status, and forms the basis for all nutrition interventions.⁴⁴ Research investigating the use of an interprofessional nutritional protocol for adults in aged care (n = 100) with Category/Stage II or III pressure injuries demonstrated that nutrition assessment is associated with improved pressure injury healing⁴³ (*Level 2*).

Serum albumin and prealbumin are generally not considered reliable indicators of nutritional status. Research demonstrates that changes in acute phase proteins do not consistently or predictably change with weight loss, calorie restriction or nitrogen balance.⁴⁴ They appear to reflect severity of inflammatory response rather than nutritional status. Marked inflammation increases the risk of malnutrition by increasing or altering the metabolism and utilization of protein. Thus the relevance of laboratory values as indicators of malnutrition is limited.² Additional discussion on nutrition assessment of neonates and children is discussed in the section on *Nutrition Management in Neonates and Children* at the end of this chapter.

Nutrition Care Planning

4.3: Develop and implement an individualized nutrition care plan for individuals with or at risk of a pressure injury who are malnourished or who are at risk of malnutrition.
(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

One low quality Level 2 study⁴³ provided evidence that a multidisciplinary nutritional intervention that included individualized care planning contributed to increased pressure injury healing as measured on the Bates-Jensen Wound Assessment Tool. The standards of practice for the registered dietitian/nutritionist, through the nutrition care process, recommend the development of individualized care plan for individuals with compromised nutritional status needing specific interventions to resolve nutrition diagnosis.²

Implementation Considerations

- When developing an individualized nutrition care plan, follow relevant and evidence-based guidelines on nutrition and hydration for individuals who exhibit nutritional risk and who are at risk of pressure injuries or have an existing pressure injury⁵⁴⁻⁶² (*Level 5*).

- Monitoring and evaluation of nutritional status is an ongoing process.⁶³ Weigh the individual weekly³⁷ or according to local policy. (*Expert opinion*).
- The individual's management plan should be adjusted with each change in clinical condition⁶³ (*Expert opinion*).

Evidence Discussion

A registered dietitian/nutritionist, in consultation with the interprofessional team, should develop and document an individualized nutrition intervention plan based on the individual's nutritional needs, feeding route and clinical goals of care, as determined by the nutrition assessment and the individual's goals. Allen (2013)⁴³ demonstrated that individualized nutrition assessment and care planning for older adults with Category/Stage II or III pressure injuries (n = 100) is associated with improved wound healing compared with standardized nutrition plans (37% versus 23.4%, $p < 0.05$)⁴³ (*Level 2*).

Energy and Protein Intake for Individuals at Risk of Pressure Injuries

4.4: Optimize energy intake for individuals at risk of pressure injuries who are malnourished or at risk of malnutrition.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

4.5: Adjust protein intake for individuals at risk of pressure injuries who are malnourished or at risk of malnutrition.
(Good Practice Statement)

Evidence Summary

Indirect evidence suggests that individuals at risk of pressure injuries and with malnutrition who receive nutritional supplementation have improved energy intake.^{64,65} One low quality level 3 study⁶⁶ in which individuals were provided with individualized energy intake calculated using the Harris-Benedict equation, there was a reduced incidence of pressure injuries. Analyses indicate that this intervention is cost effective in some geographic locations.⁶⁷⁻⁶⁹

Additional provision of protein is recommended for individuals with acute and chronic disease,⁷⁰ and older adults.⁷¹ There is currently no research evidence to indicate if higher protein intake reduces the incidence of pressure injuries in individuals at risk. Reputable guidelines^{56-60,70,71} suggest that increasing protein intake in individuals with or at risk of malnutrition who may be at pressure injury risk due to illness and/or older age represents good clinical practice.

Implementation Considerations

Energy intake

- Refer to reputable nutritional guidelines for recommended dietary intake (e.g., relevant guidelines produced by The Institute of Medicine (now known as The National Academies of Sciences, Engineering and Medicine[NASEM]),⁶² The Academy of Nutrition and Dietetics,⁷² European Food Safety Authority,⁵⁵ The European Society for Clinical Nutrition and Metabolism [ESPEN],^{57,58} The American Society for Parenteral and Enteral Nutrition [ASPEN],^{59,60,73} and The Australian National Health and Medical Research Council [NHMRC] together with the New Zealand Ministry of Health.⁵⁶ These guidelines recommend calculating energy intake based on individualized circumstances (e.g., medical condition, lifestyle, BMI, etc.) (*Level 5 and expert opinion*).
- Provide and encourage individuals at risk of pressure injuries to consume a balanced diet that includes nutrient dense foods using the recommended dietary intake for that individual as outlined in reputable nutritional guidelines.^{62,72,55-60,73} (*Level 5 and expert opinion*).
- When dietary intake is inadequate, or deficiencies are suspected or confirmed, provide a vitamin and mineral supplement⁷⁴ (*Expert opinion*).
- Individualized energy intake should be based on underlying medical conditions (*Expert opinion*).
- Dietary restrictions should be revised or modified/liberalized when limitations result in decreased food and water/fluid intake. These adjustments should be made in consultation with a medical professional and managed by a registered dietitian whenever possible⁷⁵ (*Expert opinion*).
- Energy intake should be adjusted based on weight change or level of obesity or the individual's diagnosis/conditions (*Expert opinion*).

- In individuals who are underweight or who have had significant unintended weight loss, additional energy intake may be required (*Expert opinion*).
- In end-of-life/hospice and palliative care settings, strive to maintain adequate nutrition and hydration compatible with the individual's condition and wishes. Adequate nutritional support is often not attainable when the individual is unable or refuses to eat, based on certain disease states^{54,76,77} (*Expert opinion*).

Protein intake

- Refer to reputable nutritional guidelines for recommended dietary intake (e.g. relevant guidelines produced by The PROT-AGE Study Group,⁷⁰ The Society for Sarcopenia, Cachexia and Wasting Disease,⁷¹ The NASEM,⁶² The Academy of Nutrition and Dietetics,⁷² European Food Safety Authority,⁵⁵ The ESPEN,^{57,58} The ASPEN,^{59,60,73} and The Australian NHMRC and New Zealand Ministry of Health⁵⁶ (see *Table 7.3* for guidance) (*Level 5 and expert opinion*).
- Protein intake should be 1 to 1.5 g/kg body weight/day for older adults⁷¹ (*Expert opinion*).
- Protein intake of 1.2 to 1.5 g/kg body weight/day is recommended for older adults with acute or chronic disease⁷⁰ (*Level 5*).
- Provide adequate protein for positive nitrogen balance for adults at risk of a pressure injury⁶³ (*Expert opinion*).
- Assess renal function to ensure that high levels of protein are appropriate for the individual, and reassess when the individual's clinical condition changes⁷⁸ (*Expert opinion*).

Evidence Discussion

Numerous studies have examined the benefits of providing a higher energy and protein intake to adults at risk of pressure injuries who have or are at risk of malnutrition as a strategy to prevent pressure injury development. The studies, conducted with adults in palliative care,⁶⁶ aged care⁶⁴ and acute care,⁶⁵ used a variety of modalities including encouraging eating,⁶⁶ supplemental snacks,⁶⁶ oral nutritional supplements,^{65,66} tube-feeding,⁶⁴ peripheral nutrition,⁶⁶ and parenteral nutrition.⁶⁶ However, the nutritional regimens and interventions were poorly characterized and no firm conclusion could be derived.

There is currently no high quality research evidence to indicate if a higher protein and higher energy intakes reduces the incidence of pressure injuries in people at risk. However, evidenced based clinical guidelines have been published for adults who do not have a chronic wound. These guidelines recommend protein intake for an adult of at least 1 g/kg body weight/day^{56,57,71} (*Level 5*). These recommendations are summarized in *Table 7.2* and *Table 7.3*.

Although adequate water/fluid intake and maintenance of serum protein levels are needed for wound healing, this is not always achievable in the frail elderly or an individual at end of life.^{79,80} Additional assistance at mealtimes is often required by adults and children to prevent weight loss that may increase the risk of pressure injury and poor healing.⁸¹

Table 7.2: Recommendations on energy requirements for populations at pressure injury risk (all Level 5)

Guideline	Target Population	Energy Recommendation
Trans-Tasman Pressure Injury guideline (2011) ³⁴	Adults with pressure injuries at moderate to high risk for delayed healing ³⁴	30 to 35 kcalories/ body weight/day 125 to 145 kjoules/kg body weight/day
PROT-AGE Study Group guideline (2013) ⁷⁰	Older adults with kidney disease who are at risk of protein-energy wasting ⁷⁰	30 to 35 kcalories/kg
	Older adults with severe injury or disease ⁷⁰	Use indirect calorimetry to estimate energy needs, if unavailable, use an appropriate predictive equation For individuals with obesity, refer to the ASPEN standards for critically ill adults with obesity.

Guideline	Target Population	Energy Recommendation
ASPEN guidelines (2016 and 2017) ^{59,60}	Critically ill adults ⁶⁰	Use indirect calorimetry to estimate energy needs, if unavailable, use an appropriate predictive equation or weight based formula 25 to 30 Kcalories/kg/day
	Critically ill individuals with obesity ⁶⁰	Use indirect calorimetry to estimate energy needs, if unavailable, use weight-based equation BMI >30 to 50: 11-14 kcalories/kg actual body weight/day BMI >50: 22-25 kcalories/kg ideal body weight/day
	Critically ill children ⁵⁹	Use indirect calorimetry to estimate energy needs, if unavailable, use Schofield ⁸² weight-height or weight equations or WHO equations ⁸³
ESPEN guidelines (2018) ^{57,58}	Critically ill adults ⁵⁸	Use indirect calorimetry to estimate energy needs, if unavailable, use weight-based equation of 25 kcalories/kg/day increasing to target
	Older adults ⁵⁷	30 kcalories/kg body weight/day, individually adjusted based on nutrition assessment

Table 7.3: Recommendations on protein requirements for populations at pressure injury risk (all Level 5)

Guideline	Target Population	Protein Recommendation
Trans-Tasman Pressure Injury guideline (2011) ³⁴	Adults with pressure injuries at moderate to high risk for delayed healing ³⁴	1.25 to 1.5 g/kg body weight/day
Society for Sarcopenia, Cachexia and Wasting Disease (2010) ⁷¹	Older adults	1 to 1.5 g/kg body weight/day
PROT-AGE Study Group guideline (2013) ⁷⁰	Older adults with acute or chronic disease ⁷⁰	1.2 to 1.5 g/kg body weight/day
	Older adults with severe injury or disease ⁷⁰	2.0 g/kg body weight/day
ASPEN guidelines (2016 and 2017) ^{59,60}	Critically ill adults ⁶⁰	1.2 g/kg body weight/day
	Critically ill individuals with obesity ⁶⁰	BMI >30 to 40: 2.0 g/kg ideal body weight/day BMI >40: 2.5 g/kg ideal body weight/day
	Critically ill children ⁵⁹	1.5 g/kg body weight/day
ESPEN guidelines (2018) ^{57,58}	Critically ill adults ⁵⁸	1.3 g/kg body weight/day achieved progressively
	Older adults ⁵⁷	1.2 g/kg body weight/day

Energy and Protein Intake for Individuals with Pressure Injuries

4.6: Provide 30 to 35 kcalories/kg body weight/day for adults with a pressure injury who are malnourished or at risk of malnutrition.

(Strength of Evidence = B1; Strength of Recommendation = ↑)

4.7: Provide 1.25 to 1.5 g protein/kg body weight/day for adults with a pressure injury who are malnourished or at risk of malnutrition.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Direct evidence from a low quality Level 1 study⁸⁴ showed no significant difference in complete healing associated with increasing caloric and protein intake using the Harris-Benedict equation with a higher stress factor. A moderate quality Level 1 study⁸⁴ and low quality level 3 studies showed improvements in some measures of healing (e.g. DESIGN-R scores).^{8,85} A moderate quality economic analysis⁸⁶ indicated that, although substantial resources may be required, there may be overall cost savings (depending on the geographic and clinical setting) associated with optimizing energy intake achieved through a reduction in pressure injury days and an increase in quality-adjusted life years. Individuals and their informal caregivers identified knowing more about dietary requirements associated with healthy skin as a priority.^{87,88}

A low quality Level 1 study⁸⁹ reported a significant 12% absolute reduction in pressure injury PUSH scores associated with protein supplementation compared to placebo. A moderate quality Level 1 study⁸⁴ noted that high intake of protein was associated with significant improvements in pressure injury size and depth compared to low protein intake. A third Level 1 study⁹⁰ reported reduction in pressure injury size associated with increasing mean protein intake from 1.2 g/kg body weight to a 1.4 g/kg/bodyweight; however the intervention also included added arginine, zinc and antioxidants. A high quality level 2 study⁹¹ reported a significant correlation between pressure injury surface area and dietary protein intake. These findings were supported by low quality Level 3 studies^{8,85} that reported significant improvements in tissue type rated on DESIGN-R⁸⁵ and general pressure injury condition⁸ associated with increasing protein intake. In these studies, there was no impact on renal function of protein intake up to 1.5 g/kg body weight/day, although in one Level 1 study a small number of participants experienced minor gastrointestinal intolerance.⁹² A high quality economic analysis⁸⁶ indicated that a nutrition intervention that included increased protein intake delivered for 16 weeks was associated with reduction in pressure injury days, reduction in care costs and increase in quality-adjusted life years.

Implementation Considerations

Energy intake

- Individualized energy intake should be based on underlying medical condition and level of activity (*Expert opinion*).
- Dietary restrictions should be revised or modified/liberalized when limitations result in decreased food and water/fluid intake. These adjustments should be made in consultation with a medical professional and managed by a registered dietitian/nutritionist whenever possible (*Expert opinion*).
- Fortified foods should be offered when nutritional requirements cannot be achieved by normal dietary intake (*Expert opinion*).
- Oral nutritional supplements and artificial nutrition should be considered as strategies for reaching the individual's caloric intake goals⁸⁴ (*Level 1*).
- Energy intake should be adjusted based on level of obesity or on the individual's diagnosis/conditions (*Expert opinion*).
- In adults who are underweight or who have had significant unintended weight loss, additional energy intake may be required (*Expert opinion*).

Protein intake

- Provide adequate protein for positive nitrogen balance for adults with a pressure injury⁶³ (*Expert opinion*).
- Assess renal function to ensure that high levels of protein are appropriate for the individual, and reassess when the individual's clinical condition changes⁷⁸ (*Expert opinion*).

Evidence Discussion

In the last three decades, several studies have directly and indirectly addressed the importance of sufficient energy and protein intake in treating pressure injuries. These studies have generally been conducted in adults of normal weight range; evidence for nutritional requirements for individual with pressure injuries who are severely underweight, obese and for neonates and children, is lacking.

Findings demonstrate the interrelationship between meeting energy and protein requirements. For example, Breslow et al. (1993)⁹¹ established that individuals receiving higher protein, higher energy diets achieved statistically significantly greater reductions in pressure injury surface area compared to baseline than did individuals receiving a standard diet ($p < 0.02$). Furthermore, a change in pressure injury surface area was correlated with both dietary protein ($r = 0.50$, $p < 0.01$) and energy intake ($r = -0.41$, $p < 0.03$)⁹¹ (*Level 2*). Iizaka et al. (2014)⁸⁵ observed not only that meeting energy and protein requirements was associated with changes in weight, arm muscle circumference and serum albumin level, but also that energy and protein intake was associated with wound healing for deep pressure

injuries ($p = 0.013$ for both energy and protein)⁸⁵ (Level 3). Protein intake varied in these studies from 0.95 g/kg body weight/day⁹¹ to 2.1 ± 0.9 g/kg body weight/day.⁸⁵

Quantifying requirements for energy and protein has been the focus of numerous studies. Lee (2006)⁸⁹ reported that providing concentrated, fortified, collagen protein hydrolysate supplement three times daily (each dose 1.5 fluid ounce dose unit, with each unit containing 15 g hydrolyzed protein) compared to placebo resulted in a 60% reduction in PUSH scores after eight weeks of treatment compared to a 48% reduction in the control group ($p < 0.05$) (Level 1). Yamamoto et al. (2009)⁸ demonstrated an improvement in healing of pressure injuries when adults consumed more than 30 kcalories/kg body weight/day, while those consuming no greater than 20 kcalories/kg body weight/day experienced worsening or no improvement in healing. Furthermore, a significant difference in daily protein intake was observed between adults achieving improvements in wound condition and the group with unimproved pressure injuries (always > 45 g/day versus ~ 20 g/day, $p < 0.005$) (Level 3). An RCT⁸⁴ ($n = 60$) investigated the effectiveness of enteral feeding based on a predictive basal energy equation (BEE) combined with a higher intake of protein 1.62 g/kg body weight/day, compared to a control group receiving a daily protein intake of 1.24 g/kg body weight/day. Pressure injuries healed within 12 weeks for seven subjects in the intervention group and four subjects in the control group. Pressure injury depth decreased steadily in the intervention group ($p < 0.05$). Although the researchers concluded that calculating nutritional requirements using *BEE x activity factor 1.1 x stress factor 1.3 to 1.5* may be associated with improved pressure injury healing, the results were limited to immobile older adults receiving enteral feeding (Level 1). Cereda et al. (2009)⁹⁰ explored a high energy, high protein (30 kcalories/kg body weight/day with protein 1.5 g/kg body weight/day) nutritional approach in a small RCT ($n = 28$). This regimen resulted in faster pressure injury healing compared to a high calorie, normal protein diet (30 kcalories/kg body weight/day with protein 1.2 g/kg body weight/day). Nonetheless, individuals allocated to the high energy, high protein intervention also received additional micronutrients (arginine, zinc and antioxidants) that may play an active role in wound healing, therefore residual confounding could not be excluded⁹⁰ (Level 1).

The findings reported in the studies above and the recommendations made in this guideline are supported by a meta-analysis⁹³ focusing on measured energy needs in adults with pressure injuries, as well as other reputable guidelines.³⁴ In this meta-analysis, Cereda et al. (2011)⁹³ reported that after adjusting measured (indirect calorimetry) resting energy expenditure for a 1.3 physical activity correction factor (for individuals confined to bed), mean total daily energy needs are about 30 kcalories/kg body weight/day.

It should be noted that a normal protein diet (16% of total energy) of 30 kcalories/kg body weight/day provides at least 1.2 grams of protein/kg body weight daily. The amount of protein intake increases to 1.4 g/kg body weight with a 35 calories/kg body weight/day diet. In a high protein nutritional support (20% of total energy) the amount of protein provided to individuals would amount to 1.5 to 1.75 g/kg body weight/day.⁹⁰ Protein intake should be adjusted according to kidney function and long term high protein intake should be avoided due to potentially detrimental effects on kidney and liver function. In absence of relevant co-morbidities, a return to an ideal protein intake of about 1.0 to 1.2 g/kg body weight/day could be advised after complete pressure injury healing has occurred. Nevertheless, it is important to consider that an insufficient intake of energy increases the individual's protein requirement.⁵⁷

Nutritional Supplementation

Oral nutritional supplements (ONS), enhanced foods, and food fortifiers can be used to combat unintended weight loss and malnutrition in individuals who are unable to consume estimated requirements by spontaneous (normal) food intake. Oral nutritional supplements include products that supply nutrients including protein, carbohydrates, fat, vitamins, minerals, and/or amino acids. Health professionals are advised to review the nutrition labeling on oral and enteral supplements, to determine micronutrient adequacy.

Nutrition supplementation for neonates and children is discussed in the section on *Nutrition Management in Neonates and Children* at the end of this chapter.

4.8: Offer high calorie, high protein fortified foods and/or nutritional supplements in addition to the usual diet for adults who are at risk of developing a pressure injury and who are also malnourished or at risk of malnutrition, if nutritional requirements cannot be achieved by normal dietary intake.
(Strength of Evidence = C; Strength of Recommendation = \uparrow)

4.9: Offer high calorie, high protein nutritional supplements in addition to the usual diet for adults with a pressure injury who are malnourished or at risk of malnutrition, if nutritional requirements cannot be achieved by normal dietary intake.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

4.10: Provide high-calorie, high-protein, arginine, zinc and antioxidant oral nutritional supplements or enteral formula for adults with a Category/Stage II or greater pressure injury who are malnourished or at risk of malnutrition.

(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Nutrition supplements for adults at risk of pressure injuries

One low quality Level 1 study⁹⁴ found that high-calorie, high-protein supplements were associated with a significant reduction in the incidence of pressure injuries in individuals at risk. This finding was supported by a large, low quality Level 3 study⁴⁵ and favorable but non-significant results from a smaller low quality Level 1 study.⁹⁵ However, other high quality⁹⁶ and low quality^{5,97} Level 1 studies showed no significant effect in reducing pressure injury incidence for high calorie, high protein nutritional supplements. The body of evidence is inconsistent, reflecting uncertainty as to the likelihood that the expected benefits will be achieved. However, there are no known undesired effects, and moderate quality economic analyses⁶⁷⁻⁶⁹ reported cost-savings, including shorter hospital stays, associated with the intervention. Individuals and their informal caregivers identified knowing more about dietary requirements associated with healthy skin as a care priority.^{87,88}

Nutrition supplements for adults with pressure injuries

A large low quality Level 1 study⁵ reported a mean of approximately 42% pressure injuries reached complete healing when high calorie, high protein supplements were provided, which was around 10% more than for standard diet. A high quality Level 1 study⁹² reported complete healing rate of around 10%. Differences in healing rates reported in Level 1 studies might be explained by the large variation in intervention duration of between 3 and 26 weeks. Significant reduction in mean pressure injury surface area and significant improvement in PUSH scores was reported in a low quality Level 1⁹² and Level 2⁹¹ studies for high calorie, high protein supplementation compared with standard diets or placebo supplements. Few adverse events were experienced in studies and an economic analysis⁸⁶ indicated that supplementation was associated with reductions in costs per individual and increases in quality-adjusted life years associated with more pressure injury-free days. More than two thirds of individuals who have experienced a pressure injury indicated that receiving guidance on diet to promote health was a priority.⁸⁷

There is evidence from a high quality Level 1 study,⁹² to suggest that high-calorie, high-protein oral nutritional supplements containing arginine, zinc and antioxidants are related to significant improvements in measures of pressure injury healing and are more effective than high-calorie, high-protein oral nutritional supplements without specific nutrients. The high quality Level 1 study showed more than three times greater likelihood of a pressure injury healing when a high-calorie, high-protein oral nutritional supplement containing arginine, zinc and antioxidants is provided for more than four weeks.⁹² Three moderate quality level 1 studies,^{90,98,99} a low quality Level 1 study¹⁰⁰ and low quality Level 4 studies¹⁰¹⁻¹⁰³ provided evidence for improvements in other wound healing measures including surface area reduction and improvements on PUSH scale. There are no known adverse events. A high quality cost analysis¹⁰⁴ showed the treatment is associated with cost savings to heal a pressure injury compared with no disease-specific nutrient supplementation.

Implementation Considerations

- Supplements should be offered when nutritional requirements cannot be achieved by normal dietary intake^{89,92} (*Level 1*).
- Supplements should be provided between meals¹⁰⁵ (*Level 5*).
- Supplement dose should be two bottles/day serving an energy density of 1.5 to 2.4 kcalories/mL (*Expert opinion*).
- Continue supplementation for at least four weeks (*Expert opinion*).

Evidence Discussion

Nutritional interventions providing adequate calories and protein are believed to play a pivotal role in reducing the risk of pressure injuries in individuals with poor nutritional status and/or nutritional deficits. Nonetheless, pressure injuries themselves are responsible for a deterioration in nutritional status due to increased energy and protein expenditure and nutrient loss associated with a chronic wound. The provision of extra energy is an important strategy to improve anabolism. However, individuals with pressure injuries are frequently characterized by impairment in spontaneous (normal) food intake. Nutritional supplementation is one strategy in satisfying nutritional needs. When oral feeding is still feasible and safe, ONSs are the first-line strategy to cope with inadequate protein and calorie intake. Evidence in this area focuses on multi-nutrient supplementation. Limited early evidence^{106,107} exploring the use of a single vitamin has failed to demonstrate a benefit for individuals with or at risk of pressure injuries.

A study conducted by Wilson et al. (1986)¹⁰⁸ indicated that healthy older adults who consumed high energy, high protein ONS between meals experienced better absorption of nutrients, with the least interference to meal intake (*Level 5*). This suggests that supplementation should be administered between meals. Consistent with this, a systematic review¹⁰⁹ has found that adherence to ONS, commonly provided between meals, is generally good, especially with higher energy density ONS, resulting in improvements in the total energy intakes of individuals that has been linked with clinical benefits.¹⁰⁹

Nutritional supplements for adults at risk of pressure injuries

Identifying the independent contribution of ONS as a nutrition intervention to reduce the risk of pressure injuries in high risk and older populations is challenging because of the multifactorial nature of pressure injury risk reduction. Both retrospective cohort trials⁴⁵ (*Level 3*) and RCTs^{5,94-97} (*Level 1*) have mixed findings, with uncertainty surrounding the efficacy of supplementation in the prevention of pressure injuries. Studies with larger population groups (626 participants⁴⁵ and over 1,500 participants⁹⁴ reported significant findings (*Level 1 and Level 3*) compared with non-significant findings in studies with smaller population groups (less than 500 participants^{5,95-97}) (all *Level 1*). In one of the smaller RCTs,⁹⁵ the ONS intervention resulted in a significantly higher favorable overall clinical course, but a non-significant reduction in incidence of pressure injuries. These studies were all conducted in older adults in acute or long term care settings and the studies ranged in length from two weeks to six months, with no obvious association between study length and results.^{5,45,94-97}

However, a quantitative synthesis of the trials reported above has shown that the use of ONS that provides 250 to 500 kcalories for up to 26 weeks is associated with a lower incidence of pressure injuries in individuals at risk of pressure injuries compared to routine care (OR 0.75, 95% CI 0.62 to 0.89).¹¹⁰ Nonetheless, all studies were consistent for an increase in protein-calorie intake from this intervention, which is in substantial agreement with available literature on the use of ONS, particularly when energy dense formula is used.¹⁰⁹

It is worth noting that economic modelling conducted in Australia and based on RCTs found that nutritional support is cost effective in preventing pressure injuries in hospitalized individuals who are at high risk of pressure injuries. Compared with standard diet, ONS was associated with a predicted mean decrease in length of hospital stay of 0.52%, leading to cost efficiencies.⁶⁷⁻⁶⁹ (*Moderate quality economic analysis*).

Nutritional supplements for adults with pressure injuries

Evidence on the efficacy of extra protein and energy provision in the healing of pressure injuries is substantial. Several RCTs^{5,89,92} (*Level 1*) and a non-randomized control trial⁹¹ (*Level 2*) conducted in hospitals, long term care and community care settings have consistently demonstrated significant improvement in healing of pressure injuries in individuals receiving high energy, high protein ONS in addition to a usual diet compared to control groups. There is growing evidence that supports a positive effect on pressure injury healing of adding arginine and micronutrients (zinc and antioxidants) to high calorie, high protein nutritional supplementation via either ONS or tube-feeding.^{99,103}
^{90,98,99,102,111} (*Levels 1 and 3*).

Two small RCTs (28⁹⁰ and 43 participants⁹⁸) investigating a disease specific nutritional approach as a strategy to promote pressure injury healing reported improved healing as defined by decreasing PUSH scores, in individuals with Category/Stage II or greater pressure injuries in health care centers, hospitals, and long term care facilities. The intervention in both trials was a high energy, high protein formula fortified with arginine and other micronutrients.^{90,98} Not only was the decrease in PUSH score statistically significantly different between the treatment and control groups in both studies,^{90,98} but the van Anholt et al. (2010)⁹⁸ study reported significantly fewer dressings were required per week in the ONS group compared with the control ($p = 0.045$), and less time was spent per week changing the dressings ($p = 0.022$). The researchers concluded that a high protein nutritional supplement with added arginine and micronutrients administered for at least eight weeks may be associated with improved pressure injury healing in older adults who do not have pre-existing malnutrition.^{90,98} (*Level 1*).

To clarify the independent role of specific nutrients in the healing process, the Oligo-Element Sore Trial (OEST),⁹² a double-blind RCT with 200 participants, compared a high energy, high protein nutritional formula enriched with arginine, zinc and antioxidants with an active isocaloric, isonitrogenous control formula (500 kcalories and 40 g protein). After eight weeks of intervention, malnourished adults with (primarily) Category/Stage III and IV pressure injuries had a greater mean reduction in wound surface area compared with a high energy, high protein ONS with no specific nutrients (disease-specific, 60.9%, 95% CI 54.3 to 67.5 versus control, 45.2%, 95% CI 38.4 to 52.0; $p = 0.026$). This equated to an adjusted treatment effect of 18.7% (95% CI 5.7 to 31.8, $p = 0.017$). A greater proportion of complete healing was also observed in the treatment group (16.9%, 95% CI, 8.2 to 25.6 versus 9.7%, 95% CI, 2.1 to 17.3, $p = 0.10$). This equated to an adjusted treatment effect with odds ratio of 2.16 (95% CI 0.88 to 5.39, $p = 0.097$). For individuals remaining in the study for at least four weeks, the adjusted odds ratio was 3.71 (95% CI, 1.05 to 13.16, $p = 0.042$)⁹² (*Level 1*). However, a similar smaller study ($n = 50$) of only two weeks' duration did not result in a significant difference in change in total PUSH score¹⁰⁰ (*Level 1*). Overall, based on the studies of arginine, zinc and antioxidants, there is a moderate-to-high body of evidence supporting the positive effect of using high energy, high protein supplements with arginine and micronutrients to promote pressure injury healing.

Evidence from trials suggest that the length of intervention should be at least four weeks and administration up to complete healing is advisable. The duration of supplementation has been examined in community dwelling individuals with spinal cord injury (SCI) resulting in mixed findings. Brewer et al. (2010)¹⁰¹ found that an intervention group that took 9 g arginine daily experienced superior healing compared to a control group (10.5 ± 1.3 weeks to complete healing versus 21.1 ± 3.7 weeks, $p = 0.006$) (*Level 4*). Another observational study¹⁰² examining the effects of 9 g arginine/day found no statistically significant difference in time to healing between individuals who ceased treatment early and those who completed when the analysis was confined to the Category/Stage of pressure injury. However, when combining analysis of Category/Stage III and IV pressure injuries, a 2.5-fold greater rate of healing was observed for individuals who continued supplementation until full healing compared with those who ceased taking the supplement (8.5 ± 1.1 weeks versus 20.9 ± 7.0 weeks, $p = 0.04$) (*Level 4*).

Moreover, dispensable amino acids (i.e., arginine) become conditionally indispensable amino acids during periods of physiological stress. Quantifying the optimal level of arginine has been explored in a small RCT¹¹² that compared different doses of arginine for healing pressure injuries in acute inpatient participants. The results indicated there was no difference in healing outcomes between a 4.5 g/day and a 9 g/day of arginine supplementation (*Level 1*).

The economic benefits of the disease-specific ONS formula enriched with arginine, zinc, and antioxidants compared to a high-calorie, high-protein oral support are substantial.⁹² Although a disease-specific formula cost more money than a high calorie, high protein ONS without specific nutrients ($p < 0.001$), in a cost analysis that included direct care costs (equipment, tests and staffing) administering this ONS resulted in a significant reduction of overall cost of care ($-\text{€}74.30$, 95% CI -126.1 to -22.5 , $p = 0.013$), with a substantial incremental cost-effectiveness ratio (ICER; $\geq 95\%$ of points were in the 'more effective/less expensive' quadrant)¹⁰⁴ (*High quality economic analysis*).

Artificial Nutrition: Enteral and Parenteral Feeding

There are individuals who cannot meet their nutritional requirements via normal oral intake, even when ONSs are provided.

4.11: Discuss the benefits and harms of enteral or parenteral feeding to support overall health in light of preferences and goals of care with individuals at risk of pressure injuries who cannot meet their nutritional requirements through oral intake despite nutritional interventions.
(Good Practice Statement)

4.12: Discuss the benefits and harms of enteral or parenteral feeding to support pressure injury treatment in light of preferences and goals of care for individuals with pressure injuries who cannot meet their nutritional requirements through oral intake despite nutritional interventions.
(Strength of Evidence = B1; Strength of Recommendation = \uparrow)

Evidence Summary

Due to obvious ethical reasons, there are no randomized trials comparing provision of artificial nutrition (enteral or parenteral) to no intervention in individuals unable to satisfy requirements by spontaneous (normal) oral feeding. In

these clinical situations, administering nutrition via other routes (e.g., naso-enteric tube, PEG or parenteral nutrition) may be discussed with the individual and informal caregivers.

A low quality level 1 study⁹⁷ and three low quality level 3 studies^{45,64,113} indicate that enteral or parenteral feeding have limited impact on pressure injury incidence in individuals at risk. Although current evidence does not support the use of enteral or parenteral feeding to prevent pressure injuries, consideration should be given to the individual's care goals, overall health and clinical needs beyond pressure injury prevention and treatment.

In adults with pressure injuries, two moderate quality level 1 studies showed that high calorie, high protein enteral or parenteral supplements lead to improvements in some measures of pressure injury healing compared to standard formulas.^{84,90} A moderate quality level 3 study had conflicting findings; however, these findings could be because pressure injuries were often more severe in individuals who received enteral feeding in the clinical studies. For example, Breslow et al. (1991)¹¹⁴ found a significant positive correlation between amount of enteral formula received and pressure injury surface area ($r = 0.59$, $p < 0.04$).

Implementation Considerations

- Parenteral and enteral feeding should be administered by qualified professionals using a monitoring protocol⁶⁰ (*Expert opinion*).
- Tolerance of enteral feeding should be evaluated daily through physical examination, regularity of stool and flatus, and experience of gastrointestinal signs and symptoms (e.g., vomiting, abdominal distension, nausea, discomfort, etc.)⁶⁰ (*Expert opinion*).
- Routine assessment should confirm that individuals are actually receiving the amount of tube-feeding solution prescribed (*Expert opinion*).

Evidence Discussion

If oral intake is inadequate, enteral or parenteral nutrition may be recommended if consistent with the individual's wishes. Enteral (tube) feeding is the preferred route if the gastrointestinal tract is functioning. The risks and benefits of nutrition support should be discussed with the individual and informal caregivers early on and should reflect the individual's preferences and goals for care.

Special considerations in palliative care settings

The overriding concerns in palliative care and end of life/hospice care are to provide comfort and minimize symptoms. If providing supplemental nutrition assists in providing comfort to the individual and is mutually agreed upon by the individual, family caregivers, and health professional, then supplemental nutrition (in any form) is very appropriate for palliative or end of life/hospice wound care. If the individual's condition is such that to provide supplemental nutrition (in any form) increases discomfort and the prognosis is expected to be poor, then providing supplemental nutrition should not be a concern and is not appropriate for palliative or end of life/hospice wound care. An individual receiving palliative care who does not have pressure injury healing as a goal can be allowed to consume the type and amount of food and fluids as desired.⁷⁹

Parenteral/enteral feeding in individuals at risk of pressure injuries

The limited volume of research evaluating the benefits of utilizing enteral and parenteral nutrition for the prevention of pressure injuries shows that this modality has no impact on the incidence of pressure injuries^{45,64,113} (*Levels 1 and 3*). The high acuity of the acute care and long term care populations in the trials appears to be a factor that both increases risk of pressure injuries, and contributes to the decision to trial enteral or parenteral feeding.^{45,64,113} For example, in one study,¹¹³ individuals who received enteral feeding were at significantly greater risk of pressure injuries and had significantly lower BMI than individuals who did not receive enteral feeding. The lack of comparable populations therefore confounded the overall finding that enteral feeding did not contribute to a decrease in incidence of pressure injuries¹¹³ (*Level 3*).

Other factors might also contribute to the lack of significant findings in the available research. One of the trials was conducted over only two weeks;⁹⁷ however, the other trials were of eight weeks,¹¹³ twelve weeks⁴⁵ and four years in duration.⁶⁴ In one of the studies, a number of other factors beyond the nutrition intervention were associated with incidence of pressure injuries in individuals in long term care, including, water/fluids provided, medications, and staffing patterns⁴⁵ (*Level 3*). Comorbidities that may have influenced the results were not reported in another study⁶⁴ (*Level 3*).

Due to the quality of the research available, it remains unclear if timely and sufficient parenteral or standard enteral feedings provided to individuals at risk for pressure injuries would reduce prevalence.

Parenteral/enteral feeding in individuals with pressure injuries

There is a limited volume of research evaluating the benefits of utilizing enteral and parenteral nutrition for the treatment of pressure injuries that demonstrates this modality has a positive impact on the healing of pressure injuries.^{64,84,90} (Levels 1 and 3). Although small (60 participants⁸⁴ and 28 participants⁹⁰), both high level studies emphasize a link between nutrition support in the form of high protein and disease specific enteral feedings and a decrease in incidence of pressure injuries in older adults^{84,90} (Level 1). Although one cohort study failed to identify a link between PEG supplementation and significant improvements in pressure injury healing compared to an oral diet, the individuals received PEG feeding had substantially more severe pressure injuries at commencement of the intervention¹¹⁵ (Level 3).

Using aggressive nutrition support may not be beneficial in all individuals and it is not risk free. Individuals receiving enteral nutrition via a PEG or nasogastric tube had significantly more major complications (e.g. weight loss, pneumonia and death) that were deemed to be related to the intervention compared to individuals receiving an oral diet (61% versus 34%, $p < 0.01$) in one study¹¹³ (Level 3). Harvey et al. (2016)¹¹⁶ reported no significant change in mortality rate when utilizing parenteral nutrition support over enteral nutrition support in critically ill individuals (Level 1). Teno et al. (2012)¹¹⁵ reported that feeding via PEG tubes that may be related to increased diarrhea, increased immobility or comorbidities, but this was not investigated (Level 3).

Hydration

Water serves as the solvent for vitamins, minerals, glucose and other nutrients. Water is also needed to transport nutrients through the body, and to eliminate waste products. In healthy individuals who are adequately hydrated, water released from food and metabolism accounts for 20% or more of total water intake.¹¹⁷ Total water needs include the water content of food.¹¹⁷ Note that not all fluids contain water; requirements are based on water needs.

Calculate individual water/fluid requirements. Various formulas have been used to calculate adequate daily water/fluid needs. Evidence-based guidelines recommend that water requirements be calculated as 1 mL/kcalorie/day.^{34,118} Individuals with elevated temperature, vomiting, profuse sweating, diarrhea, and/or heavily exuding wounds often require additional water/fluid intake to replace losses.³⁴ Individuals consuming high levels of protein may also require additional water intake.

Oral nutritional supplements and enteral feedings normally contain 75% water from its total volume. For the specific amount of free water in each enteral formula, refer to each product's nutrition label.

4.13: Provide and encourage adequate water/fluid intake for hydration for an individual with or at risk of a pressure injury, when compatible with goals of care and clinical condition.
(Good Practice Statement)

Implementation Considerations

- Provision of hydration must be consistent with the individual's comorbid conditions and goals (*Expert opinion*).
- In healthy people, water/fluid intake should be approximately 30 mL/kg body weight/day or 1 mL/kcalorie/day.^{34,118} Fluid intake is often restricted for individuals with heart or renal failure (*Expert opinion*).
- Provide additional water/fluid for individuals with dehydration, elevated temperature, vomiting, profuse sweating, diarrhea, or heavily exuding wounds. Individuals consuming high levels of protein may also require additional water/fluid (*Expert opinion*).
- Monitor individuals for signs and symptoms of dehydration including change in weight, skin turgor, urine output, elevated serum sodium, and/or calculated serum osmolality (*Expert opinion*).

Nutrition Management in Neonates and Children

Neonates and children (individuals up to the age of 18 years) are at high risk of nutritional deficiencies due to having an increased nutritional requirement per unit weight to meet normal growth needs, as well as having smaller appetites and dietary intake. Additionally, children at risk of or with a pressure injury for the most part have other severe acute or chronic comorbidities (including malnutrition) that influence both nutritional requirements and the ability to absorb and utilize these nutrients.^{119,120}

Although this section includes recommendations specific to neonates and children, information throughout the rest of this chapter is also relevant to child populations. The section in this chapter on *Nutrition Care Planning* is appropriate

to the care of neonates and children. The section in this chapter on *Energy and Protein Intake for Individuals at Risk of Pressure Injuries* includes guidance on intake requirements for children. The discussion and recommendations in the chapter sections *Artificial Nutrition: Enteral and Parenteral Feeding* and *Hydration* are also broadly relevant to neonates and children.

Nutrition assessment, selection of the appropriate mode of feeding, frequent monitoring, strategies to promote adequate intake in an appealing manner, and, when required, nutritional supplements or nutritional support, are all important considerations in the promotion of wound healing in children.^{121,122}

4.14: Conduct age appropriate nutritional screening and assessment for neonates and children at risk of pressure injuries.
(Good Practice Statement)

Implementation Considerations

- Use a simple, valid and reliable nutritional screening tool appropriate for the child population group.¹²³ (*Expert opinion*).
- Critically ill children admitted to the intensive care require a comprehensive nutrition assessment within 48 hours of admission⁵⁹ (*Level 5*).
- Regularly reassess the nutritional requirements of critically ill neonates and children who have, or are at risk of, a pressure injury. Conduct a nutrition assessment at least weekly for critically ill children^{59,123} (*Level 5*).
- For neonates and children in critical care, measure and document body weight, height/length and (in children aged less than 3 years) head circumference. Use z-scores for BMI for age to screen for neonates or children at the extreme⁵⁹ (*Level 5*).
- For premature infants, adjust and correct measurements for gestational age¹²² (*Expert opinion*).

Discussion

A pediatrician, dietitian or other qualified health professional should conduct an age appropriate nutritional screening and assessment to identify nutritional requirements for neonates and children with, or at risk of pressure injuries. Early identification of neonates and children with or at risk of malnutrition is important to enable prompt intervention.¹²² The Academy and ASPEN⁴ definition of pediatric malnutrition is reported in the *Introduction* to this chapter. *Table 7.4* lists commonly used pediatric nutrition screening and assessment tools.

Table 7.4: Pediatric nutrition screening and assessment tools

Nutrition Screening Tool	Evidence for identifying pressure injury risk status	Screening (S) or Assessment (A) tool	Clinical setting and level of evidence
Subjective Global Nutritional Assessment for Children (SGNA) ¹²⁴	No	S, A	Hospitalized children aged 1 month to 18 years ¹²⁵ (<i>Level 5</i>) Children with cerebral palsy aged 1 to 12 years. ¹²⁶ (<i>Level 5</i>)
Paediatric Nutrition Screening Tool (PNST) ¹²⁷	No	S, A	Hospitalized neonates and children aged birth to 16 years ¹²⁸ (<i>Level 5</i>)
Screening Tool for the Assessment of Malnutrition in Pediatrics (STAMP) ¹²⁹	No	S, A	Hospitalized neonates and children aged 2 to 7 years ¹³⁰ (<i>Level 5</i>) Children with spinal cord injury ¹³¹ (<i>Level 5</i>)
Paediatric Yorkhill Malnutrition Score (PYMS) ¹³²	No	S	Hospitalized children aged 1 to 16 years ¹³³ (<i>Level 5</i>)
Screening Tool for the Risk of Impaired Nutritional Status and Growth (STRONGkids) ¹³⁴	No	S	Hospitalized children from birth to 17 years ¹³⁵ (<i>Level 5</i>) Hospitalized children aged ≥ 1 year and not in critical care ¹³⁶ (<i>Level 5</i>)

When undertaking nutritional screening and assessment in neonates and children, anthropometric measurements and growth charts can be used to determine if the child is developing within expected growth patterns.^{119,121} However, also consider the influence of edema and fluid shifts on measures made in critically ill children.^{119,124}

4.15: For neonates and children with or at risk of pressure injuries who have inadequate oral intake, consider fortified foods, age appropriate nutritional supplements, or enteral or parenteral nutritional support. (Good Practice Statement)

Implementation Considerations

- Where possible enteral nutrition should be preferred over other methods of providing nutrition requirements⁵⁹ (Level 5).
- Ensure all neonates and children maintain adequate hydration¹²² (*Expert opinion*).

Discussion

A pediatrician, pediatric dietitian or other qualified health professional should be involved in planning an appropriate, individualized nutrition plan, and providing caregivers with strategies to promote nutritional intake.¹²¹ Energy needs should be individualized and determined with consideration to energy expenditure in order to avoid overfeeding or underfeeding. Energy and protein intake should be determined in consideration of:

- Requirements for normal growth and development
- Any nutritional deficiency
- Altered needs associated with critical illness or comorbidities
- Needs associated with wound healing.¹²¹

Critically ill children should have their energy expenditure assessed regularly in order to determine appropriate energy needs. Consider that standard equations are often unreliable in estimating energy expenditure in children^{119,137} because they are often derived from measurements in healthy children or adults.^{122,137} For these reasons, energy requirements in neonates and children with a chronic wound are often underestimated.¹³⁸ When direct measurement cannot be made, ensure that any energy expenditure equation that is used to estimate needs is appropriate to the child's age and clinical condition appropriate.¹³⁷ In a review of cohort studies conducted in critically ill children, the variability of metabolic state and thus the inappropriate nature of providing recommendations on specific intake goals was highlighted (Level 5);^{59,119} however, it has been suggested that oral intake should be sufficient to prevent weight loss and complications associated with nitrogen wasting.¹³⁹ Increases in protein and micronutrients might be appropriate if wound healing does not progress on the expected trajectory¹³⁸ (*Expert opinion*). There is insufficient evidence to make specific recommendations on the macro- and micronutrient requirements for neonates and children who have a chronic wound.^{59,119} For individuals with inadequate oral intake, enteral or parenteral nutrition should be commenced,¹³⁹ when consistent with the care goals of the child and their informal caregivers. Due to insufficient evidence to support their use in children, supplemental immune-enhancing nutrients (e.g., arginine, glutamine, antioxidants, etc.) are not recommended for use in critically ill children¹¹⁹ (*Expert opinion*).

References

1. Lagua RT, Claudio VS. *Nutrition and Diet Therapy Reference Dictionary: Malnutrition*. 1995, New York: Chapman & Hall.
2. White J. Consensus Statement: AND and ASPEN: Characteristics recommended for the identification and documentation of adult malnutrition (undernutrition). *J Acad Nutr Diet*, 2012; 112(5): 730-738.
3. Thomas DR. Loss of skeletal muscle mass in aging: Examining the relationship of starvation, sarcopenia and cachexia. *Clin Nutr*, 2007; 26(4): 389-399.
4. Becker P, Carney LN, Corkins MR, Monczka J, Smith E, Smith SE, Spear BA, White JV. Consensus statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Indicators recommended for the identification and documentation of pediatric malnutrition (undernutrition). *Nutr Clin Pract*, 2015; 30(1): 147-161.
5. Ek AC, Unosson M, Larsson J, von Schenck H, Bjurulf P. The development and healing of pressure sores related to the nutritional state. *Clin Nutr*, 1991; 10(5): 245-250.
6. Iizaka S, Okuwa M, Sugama J, Sanada H. The impact of malnutrition and nutrition-related factors on the development and severity of pressure ulcers in older patients receiving home care. *Clin Nutr*, 2010; 29(1): 47-53.
7. Green CJ. Existence, causes and consequences of disease-related malnutrition in the hospital and the community and clinical and financial benefits of nutritional intervention. *Clin Nutr*, 1999; 18(suppl. 2): 3-28.
8. Yamamoto T, Fujioka, Kitamura R, Yakabe A, Hironori K, K. Y, Nagatomo H. Evaluation of nutrition in the healing of pressure ulcers: Are the EPUAP nutritional guidelines sufficient to heal wounds? *Wounds*, 2009; 21(6): 153-157.
9. Fry DE, Pine M, Jones BL, Meimban RJ. Patient characteristics and the occurrence of never events. *Arch Surg*, 2010; 145(2): 148-151.
10. Wojcik A, Atkins M, Mager DR. Dietary intake in clients with chronic wounds. *J Can Diet Assoc*, 2011; 72(2): 77-82.

11. Verbrugghe M, Beeckman D, Van Hecke A, Vanderwee K, Van Herck K, Clays E, Bocquaert I, Derycke H, Geurden B, Verhaeghe S. Malnutrition and associated factors in nursing home residents: A cross-sectional, multi-centre study. *Clin Nutr*, 2013; 32(3): 438-443.
12. Mathus-Vliegen E. Nutritional status, nutrition and pressure ulcers. *Nutr Clin Pract*, 2001; 16: 286-291.
13. Guenter P, Malyszec R, Bliss DZ, Steffe T, O'Hara D, LaVan F, Monteiro D. Survey of nutritional status in newly hospitalized patients with stage III or stage IV pressure ulcers. *Adv Skin Wound Care*, 2000; 13(4 Pt 1): 164-168.
14. Thomas DR, Verdery RB, Gardner L, Kant A, Lindsay J. A prospective study of outcome from protein-energy malnutrition in nursing home residents. *JPEN J Parenter Enteral Nutr*, 1991; 15(4): 400-404.
15. Kerstetter JE, Holthausen BA, Fitz PA. Malnutrition in the institutionalized older adult. *J Am Diet Assoc*, 1992; 92(9): 1109-1116.
16. Schols J. Nutrition in nursing home patients with pressure ulcers: Knowing is not yet doing. *Tijdschrift voor Verpleeghuisgeneeskunde*, 2000; 24(1): 9-12.
17. Lyder CH, Preston J, Grady JN, Scinto J, Allman R, Bergstrom N, Rodeheaver G. Quality of care for hospitalized Medicare patients at risk for pressure ulcers. *Arch Intern Med*, 2001; 161(12): 1549-1554
18. Banks M, Bauer J, Graves N, Ash S. Malnutrition and pressure ulcer risk in adults in Australian health care facilities. *Nutr Clin Pract*, 2010; 26(9): 896-901.
19. Grattagliano I, Marasciulo L, Paci C, Montanaro N, Portincasa P, Mastronuzzi T. The assessment of the nutritional status predicts the long term risk of major events in older individuals. *Eur Geriatr Med*, 2017; 8(3): 273-274.
20. Tsaousi G, Stavrou G, Ioannidis A, Salonikidis S, Kotzampassi K. Pressure ulcers and malnutrition: Results from a snapshot sampling in a University Hospital. *Med Princ Pract*, 2015; 24(1): 11-16.
21. Yatabe MS, Taguchi F, Ishida I, Sato A, Kameda T, Ueno S, Takano K, Watanabe T, Sanada H, Yatabe J. Mini nutritional assessment as a useful method of predicting the development of pressure ulcers in elderly inpatients. *J Am Geriatr Soc*, 2013; 61(10): 1698-1704.
22. Meehan A, Loose C, Bell J, Partridge J, Nelson J, Goates S. Health system quality improvement: Impact of prompt nutrition care on patient outcomes and health care costs. *J Nurs Care Qual*, 2016; 31(3): 217-23.
23. Skipper A, Ferguson M, Thompson K, Castellanos VH, Porcari J. Nutrition screening tools: An analysis of the evidence. *JPEN J Parenter Enteral Nutr*, 2012; 36(3): 292-8.
24. Field LB, R.K. H. Differentiating malnutrition nutrition screening and assessment: A nutrition care process perspective. *J Acad Nutr Diet*, 2015; 15: 824-828.
25. Neelemant F, Kruizenga HM, de Vet HC, Seidell JC, Butterman M, Van Bokhorst-de van der Schueren MA. Screening malnutrition in hospital outpatients. Can the SNAQ malnutrition-screening tool also be applied to this population? *Clin Nutr*, 2008; 27(3): 439-446.
26. Poulia KA, Yannakoulia M, Karageorgou D, Gamaletsou M, Panagiotakos DB, Sipsas NV, Zampelas A. Evaluation of the efficacy of six nutritional screening tools to predict malnutrition in the elderly. *Clin Nutr*, 2012; 31(3): 378-385.
27. Visvanathan R, Penhall R, Chapman I. Nutritional screening of older people in a sub-acute care facility in Australia and its relation to discharge outcomes. *Age Ageing*, 2004; 33: 260-265.
28. Keller HH, Goy R, Kane SL. Validity and reliability of SCREEN II (Seniors in the Community: Risk Evaluation for Eating and Nutrition, Version II). *Eur J Clin Nutr*, 2005; 59(1149-1157).
29. Laporte M, Keller HH, Payette H, Allard P, Duerksen DR, Bernier P, Jeejeebhoy K, Gramlich L, Davidson B, Vesnaver E, Teterina A. Validity and reliability of the new Canadian Nutrition Screening Tool in the 'real-world' hospital setting. *Eur J Clin Nutr*, 2015; 69(5): 558-564.
30. Kondrup J, Rasmussen HH, Hamberg I, Stanga Z. Nutritional risk screening (NRS 2002): A new method based on an analysis of controlled clinical trials. *Clin Nutr*, 2003; 3: 321-336.
31. Elia M, Zellipour L, Stratton RJ. To screen or not to screen for adult malnutrition? *Clin Nutr*, 2005; 24(6): 867-884.
32. Hengstermann S, Fischer A, Steinhagen-Thiessen E, Schulz RJ. Nutrition status and pressure ulcer: what we need for nutrition screening. *JPEN J Parenter Enteral Nutr*, 2007; 31(4): 288.
33. Langkamp-Henken B, Hudgens J, Stechmiller JK, Herrlinger-Garcia KA. Mini nutritional assessment and screening scores are associated with nutritional indicators in elderly people with pressure ulcers. *J Am Diet Assoc*, 2005; 105(10): 1590-1596.
34. Trans Tasman Dietetic Wound Care Group, Evidence based practice guidelines for the nutritional management of adults with pressure injuries. 2011, www.ttdwgc.org
35. Tsai AC, Chang TL, Wang YC, Liao CY. Population-specific short-form mini nutritional assessment with body mass index or calf circumference can predict risk of malnutrition in community-living or institutionalized a people in Taiwan. *J Am Diet Assoc*, 2010; 110(9): 1328-1334.
36. Flintbox. *SCREEN®: Seniors in the Community Risk Evaluation for Eating and Nutrition*. 2016 [cited May 2019]; Available from: <https://www.flintbox.com/public/project/2750>.
37. Canadian Nutrition Task Force. *Canadian Nutrition Screening Tool*. 2014 [cited August 2019]; Available from: <http://nutritioncareinCanada.ca/sites/default/uploads/files/CNST.pdf>.
38. Nestle Nutrition Institute. *Mini Nutritional Assessment MNA®*. 1994 [cited May 2019]; Available from: https://www.mna-elderly.com/forms/mini/mna_mini_english.pdf.
39. BAPEN. *Malnutrition Universal Screening Tool*. 2003 [cited May 2019]; Available from: <https://www.bapen.org.uk/pdfs/must/must-full.pdf>.
40. Kondrup J, Allison SP, Elia M, Vellas B, Plauth M. ESPEN guidelines for nutrition screening 2002. *Clinical Nutrition* (available online <http://espen.info/documents/Screening.pdf>), 2003; 22(4): 415-421.
41. Dutch Malnutrition Steering Group. *SNAQ English*. 2019 [cited May 2019]; Available from: <https://www.fightmalnutrition.eu/?s=SNAQ+English>.

42. Dutch Malnutrition Steering Group. *SNAQ (various languages)*. 2019 [cited May 2019]; Available from: <https://www.fightmalnutrition.eu/?s=SNAQ>.
43. Allen B. Effects of a comprehensive nutritional program on pressure ulcer healing, length of hospital stay, and charges to patients. *Clin Nurs Res*, 2013; 22(2): 186-205.
44. American Dietetic Association, *International Dietetics and Nutrition Terminology (IDNT) Reference Manual: Standardized Language for the Nutrition Care Process*. 2nd ed. 2009, Chicago, IL: American Dietetic Association.
45. Horn SD, Bender SA, Ferguson ML, Smout RJ, Bergstrom N, Taler G, Cook AS, Sharkey SS, Voss AC. The National Pressure Ulcer Long-Term Care Study: Pressure ulcer development in long-term care residents. *J Am Geriatr Soc*, 2004; 52(3): 359-367.
46. Covinsky KE, Covinsky MH, Palmer RM, Sehgal AR. Serum albumin concentration and clinical assessments of nutritional status in hospitalized older people: Different sides of different coins? *J Am Geriatr Soc*, 2002; 50(4): 631-637.
47. Ferguson RP, O'Connor P, Crabtree B, Batchelor A, Mitchell J, Coppola D. Serum albumin and prealbumin as predictors of clinical outcomes of hospitalized elderly nursing home residents. *J Am Geriatr Soc*, 1993; 41(5): 545-549.
48. Shenkin A. Serum prealbumin: Is it a marker of nutritional status or of risk of malnutrition? *Clin Chem*, 2006; 52(12): 2177-2179.
49. Tobert CM, Mott SL, Nepple KG. Malnutrition diagnosis during adult inpatient hospitalizations: Analysis of a multi-institutional collaborative database of academic medical centers. *J Acad Nutr Diet*, 2018; 118(1): 125-131.
50. Hand RK, Murphy WJ, Filed LB, Lee JA, Parrott JS, GFerguson M, Skipper A, Steiber AL. Validation fo the Academy/A.S.P.E.P. malnutrition clinical characteristics. *J Acad Nutr Diet*, 2016; 116(5): 856-863.
51. Fuhrman MP, Charney P, Mueller CM. Hepatic proteins and nutrition assessment. *J Am Diet Assoc*, 2004; 104(8): 1258-1264.
52. Cederholm T, Bosaeus I, Barazzoni R, Bauer J, Van Gossum A, Klek S, Muscaritoli M, Nyulasi I, Ockenga J, Schneider SM, de van der Schueren MAE, Singer P. ESPEN endorsed recommendation: Diagnostic criteria for malnutrition - An ESPEN Consensus Statement. *Clin Nutr*, 2015; 34: 335-340.
53. Posthauer ME. The role of nutrition in wound care. *Adv Skin Wound Care*, 2006; 19(1): 43-52.
54. American Nurses Association Center for Ethics and Human Rights, Position Statement : Nutrition and Hydration at the End of Life. 2017, American Nurses Association: https://www.nursingworld.org/~4af0ed/globalassets/docs/ana/ethics/ps_nutrition-and-hydration-at-the-end-of-life_2017june7.pdf.
55. European Food Safety Authority (EFSA), Dietary Reference Values for nutrients: Summary report. 2017, EFSA: <https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/sp.efsa.2017.e15121> (accessed 14 Jan 2019).
56. National Health and Medical Research Council, Australian Government Department of Health and Ageing, New Zealand Ministry of Health, Nutrient Reference Values for Australia and New Zealand. 2017, National Health and Medical Research Council: Canberra.
57. Volkert D, Beck AM, Cederholm T, Cruz-Jentoft A, Goisser S, Hooper L, Kiesswetter E, Maggio M, Raynaud-Simon A, Sieber CC, Sobotka L, van Asselt D, Wirth R, Bischoff SC. ESPEN guideline on clinical nutrition and hydration in geriatrics. *Clin Nutr*, 2018.
58. Singer P, Blaser AR, Berger MM, Alhazzani W, Calder PC, Casaer MP, Hiesmayr M, Mayer K, Montejo JC, Pichard C, Preiser JC, van Zanten ARH, Oczkowski S, Szczeklik W, Bischoff SC. ESPEN guideline on clinical nutrition in the intensive care unit. *Clin Nutr*, 2018.
59. Mehta NM, Skillman HE, Irving SY, Coss-Bu JA, Vermilyea S, Farrington EA, McKeever L, Hall AM, Goday PS, Braunschweig C. Guidelines for the provision and assessment of nutrition support therapy in the pediatric critically ill patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition. *J Parenter Enteral Nutr*, 2017; 41(5): 706-742.
60. McClave SA, Taylor BE, Martindale RG, Warren MM, Johnson DR, Braunschweig C, McCarthy MS, Davanos E, Rice TW, Cresci GA, Gervasio JM, Sacks GS, Roberts PR, Compher C. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). *JPEN J Parenter Enteral Nutr*, 2016; 40(2): 159-211.
61. Panel on Dietary Reference Intakes for Electrolytes and Water, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine, Dietary Reference Intakes: Water, Potassium, Sodium, Chloride, and Sulfate. 2005, The National Acaemies Press: Washington, D.C.
62. Institute of Medicine, Dietary Reference Intakes: The Essential Guide to Nutrient Requirements. 2006, National Academy of Sciences,: Washington DC.
63. Mueller CM, *The ASPEN Adult Nutrion Support Core Curriculum*. Vol. USA 2017 American Society for Enteral and Parenteral Nutrition.
64. Bourdel-Marchasson I, Dumas F, Pinganaud G, Emeriau JP, Decamps A. Audit of percutaneous endoscopic gastrostomy in long-term enteral feeding in a nursing home. *Int J Qual Health Care*, 1997; 9(4): 297-302.
65. Roberts S, Chaboyer W, Leveritt M, Banks M, Desbrow B. Nutritional intakes of patients at risk of pressure ulcers in the clinical setting. *Nutrition*, 2014; 30(7-8): 841-846.
66. Amano K, Morita T, Baba M, Kawasaki M, Nakajima S, Uemura M, Kobayashi Y, Hori M, Wakayama H. Effect of nutritional support on terminally ill patients with cancer in a palliative care unit. *Am J Hosp Palliat Care*, 2013; 30(7): 730-733.
67. Banks MD, Graves N, Bauer JD, Ash S. Cost effectiveness of nutrition support in the prevention of pressure ulcer in hospitals. *Eur J Clin Nutr*, 2013; 67(1): 42-46.
68. Tuffaha HW, Roberts S, Chaboyer W, Gordon LG, Scuffham PA. Cost-effectiveness and value of information analysis of nutritional support for preventing pressure ulcers in high-risk patients: Implement now, research later. *Appl Health Econ Health Policy*, 2015; 13(2): 167-179.
69. Tuffaha HW, Roberts S, Chaboyer W, Gordon LG, Scuffham PA. Cost-effectiveness analysis of nutritional support for the prevention of pressure ulcers in high-risk hospitalized patients. *Adv Skin Wound Care*, 2016; 29(6): 261-267.
70. Bauer J, Biolo G, Cederholm T, Cesari M, Cruz-Jentoft AJ, Morley JE, Phillips S, Sieber C, Stehle P, Teta D, Visvanathan R, Volpi E, Boirie Y. Evidence-based recommendations for optimal protein intake for older people: a position paper from the PROT-AGE Study Group. *J Am Med Dir Assoc*, 2013; 14(8): 542-559.

71. Morley JE, Argiles JM, Evans WJ, Bhasin S, Cella D, Deutz NEP, Doehner W, Fearon KCH, Ferrucci L, Hellerstein MK, Kalantar-Zadeh K, Lochs H, MacDonald N, Mulligan K, Muscaritoli M, Ponikowski P, Posthauer ME, Rossi Fanelli F, Schambelan M, Schols AMWJ, Schuster MW, Anker SD, Society for Sarcopenia C, Wasting D. Nutritional recommendations for the management of sarcopenia. *J Am Med Dir Assoc*, 2010; 11(6): 391-396.
72. Academy of Nutrition and Dietetics. *Nutrition Care Manual*. 2019 [cited 2019]; Available from: <http://www.nutritioncaremanual.org>.
73. Choban P, Dickerson R, Malone A, Worthington P, Compher C. A.S.P.E.N. Clinical guidelines: nutrition support of hospitalized adult patients with obesity. *JPEN J Parenter Enteral Nutr*, 2013; 37(6): 714-44.
74. Marra MV, Bailey RL. Position of the Academy of Nutrition and Dietetics: Micronutrient supplementation. *J Acad Nutr Diet*, 2018; 118(11): 2162-2173.
75. Dorner B, Friedrich EK. Position of the Academy of Nutrition and Dietetics: Individualized nutrition approaches for older adults: Long-term care, post-acute care, and other settings. *J Acad Nutr Diet*, 2018; 118(4): 724-735.
76. Del Rio MI, Shand B, Bonati P, Palma A, Maldonado A, Taboada P, Nervi F. Hydration and nutrition at the end of life: a systematic review of emotional impact, perceptions, and decision-making among patients, family, and health care staff. *Psychooncology*, 2012; 21(9): 913-21.
77. Posthauer ME. Ethical and legal issues in feeding and hydration. *J Acad Nutr Diet*, 2013; 113(6): 828-33.
78. K/DOQI, National Kidney Foundation. Clinical practice guidelines for nutrition in chronic renal failure. *Am J Kidney Dis*, 2000; 35(6 Suppl 2): S1-140.
79. Alvarez OM, Meehan M, Ennis W, Thomas DR, Ferris FD, Kennedy KL, Rogers R, Bradley M, Baker JJ, Fernandez-Obregon A, Rodeheaver G. Chronic wounds: palliative management for the frail population. *Wounds: A Compendium of Clinical Research & Practice*, 2002; 14(8): 45-27s.
80. Bates-Jensen BM, MacLean CH. Quality indicators for the care of pressure ulcers in vulnerable elders. *Journal of the American Geriatrics Society*, 2007; 55 Suppl 2: S409-16.
81. Kayser-Jones J, Kris AE, Lim K, Walent RI, Halifax E, Paul SM. Pressure ulcers among terminally ill nursing home residents. *Res Gerontol Nurs*, 2008; 1(1): 14-24.
82. Schofield WN. Predicting basal metabolic rate, new standards and review of previous work. *Hum Nutr Clin Nutr*, 1985; 39(suppl 1): 5-41.
83. FAO/WHO/UNU Expert Group. Energy and protein requirements. Report of a joint FAO/WHO/UNU Expert Consultation. *World Health Organ Tech Rep Ser*, 1985; 724: 1-206.
84. Ohura T, Nakajo T, Okada S, Omura K, Adachi K. Evaluation of effects of nutrition intervention on healing of pressure ulcers and nutritional states (randomized controlled trial). *Wound Repair Regen*, 2011; 19(3): 330-336.
85. Iizaka S, Kaitani T, Nakagami G, Sugama J, Sanada H. Clinical validity of the estimated energy requirement and the average protein requirement for nutritional status change and wound healing in older patients with pressure ulcers: A multicenter prospective cohort study. *Geriatr Gerontol Int*, 2014.
86. Hisashige A, Ohura T. Cost-effectiveness of nutritional intervention on healing of pressure ulcers. *Clin Nutr*, 2012; 31(6): 868e74.
87. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
88. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis
89. Lee SK, Posthauer ME, Dorner B, Redovian V, Maloney MJ. Pressure ulcer healing with a concentrated, fortified, collagen protein hydrolysate supplement: a randomized controlled trial. *Adv Skin Wound Care*, 2006; 19(2): 92-96.
90. Cereda E, Gini A, Pedrolli C, Vanotti A. Disease-specific, versus standard, nutritional support for the treatment of pressure ulcers in institutionalized older adults: A randomized controlled trial. *J Am Geriatr Soc*, 2009; 57(8): 1395-1402.
91. Breslow RA, Hallfrisch J, Guy DG, Crawley B, Goldberg AP. The importance of dietary protein in healing pressure ulcers. *J Am Geriatr Soc*, 1993; 41 (4): 357-362.
92. Cereda E, Klersy C, Seriola M, Crespi A, D'Andrea F. A nutritional formula enriched with arginine, zinc, and antioxidants for the healing of pressure ulcers: A randomized trial. *Ann Intern Med*, 2015; 162(3): 167-174.
93. Cereda E, Klersy C, Rondanelli M, Caccialanza R. Energy balance in patients with pressure ulcers: A systematic review and meta-analysis of observational studies. *J Am Diet Assoc*, 2011; 111: 1868-1876.
94. Bourdel-Marchasson I, Barateau M, Rondeau V, Dequae-Merchadou L, Salles-Montaudon N, Emeriau J-P, Manciet G, Dartigues J-F, Group ftG. A multi-center trial of the effects of oral nutritional supplementation in critically ill older inpatients. *Nutrition*, 2000; 16: 1-5.
95. Delmi M, Rapin CH, Bengoa JM, Delmas PD, Vasey H, Bonjour JP. Dietary supplementation in elderly patients with fractured neck of the femur. *Lancet*, 1990; 335(8696): 1013-1016.
96. Houwing RH, Rozendaal M, Wouters-Wesseling W, Beulens JW, Buskens E, Haalboom JR. A randomised, double-blind assessment of the effect of nutritional supplementation on the prevention of pressure ulcers in hip-fracture patients. *Clin Nutr*, 2003; 22(4): 401-405.
97. Hartgrink HH, Wille J, Konig P, Hermans J, Breslau PJ. Pressure sores and tube feeding in patients with a fracture of the hip: A randomized clinical trial. *Clin Nutr*, 1998; 17 (6): 287-292.
98. van Anholt R, Sobotka L, Meijer E, Heyman H, Groen H, Topinková E, van Leen M, Schols J. Specific nutritional support accelerates pressure ulcer healing and reduces wound care intensity in non-malnourished patients. *Nutrition*, 2010; 26(9): 867-872.
99. Desneves KJ, Todorovic BE, Cassar A, Crowe TC. Treatment with supplementary arginine, vitamin C and zinc in patients with pressure ulcers: A randomised controlled trial. *Clin Nutr*, 2005; 24(6): 979-987.

100. Banks MD, Ross LJ, Webster J, Mudge A, Stankiewicz M, Dwyer K, Coleman K, Campbell J. Pressure ulcer healing with an intensive nutrition intervention in an acute setting: A pilot randomised controlled trial. *J Wound Care*, 2016; 25(7): 384-392.
101. Brewer S, Desneves K, Pearce L, Mills K, Dunn L, Brown D, Crowe T. Effect of an arginine-containing nutritional supplement on pressure ulcer healing in community spinal patients. *J Wound Care*, 2010; 19(7): 311-316.
102. Chapman BR, Mills KJ, Pearce LM, Crowe TC. Use of an arginine-enriched oral nutrition supplement in the healing of pressure ulcers in patients with spinal cord injuries: An observational study. *Nutr Diet*, 2011; 68(3): 208-213.
103. Frias Soriano L, Lage Vazquez MA, Pérez-Portabella Maristany C, Xandri Graupers JM, Wouters-Wesseling W, Wagenaar L. The effectiveness of oral nutritional supplementation in the healing of pressure ulcers. *J Wound Care*, 2004; 13(8): 319-322.
104. Cereda E, Klersy C, Andreola M, Pisati R, Schols JM, Caccialanza R, D'Andrea F. Cost-effectiveness of a disease-specific oral nutritional support for pressure ulcer healing. *Clin Nutr*, 2017; 36(1): 246-252.
105. Wilson MG, Purushothaman R, Morley JE. Effect of liquid dietary supplements on energy intake in the elderly. *Am J Clin Nutr*, 2002; 75(5): 944-947.
106. ter Riet G, Kessels AG, Knipschild PG. Randomized clinical trial of ascorbic acid in the treatment of pressure ulcers. *J Clin Epidemiol*, 1995; 48(12): 1453-60.
107. Langer G, Fink A. *Nutritional interventions for preventing and treating pressure ulcers*. *Cochrane Database Syst Rev*, 2014. DOI: 10.1002/14651858.CD003216.pub2.
108. Wilson AP, Treasure T, Sturridge MF, Grüneberg RN. A scoring method (ASEPSIS) for postoperative wound infections for use in clinical trials of antibiotic prophylaxis. *Lancet*, 1986; 1(8476): 311-313.
109. Hubbard GP, Elia M, Holdoway A, Stratton RJ. A systematic review of compliance to oral nutritional supplements. *Clin Nutr*, 2012; 31(3): 293-312.
110. Stratton RJ, Ek AC, Engfer M, Moore Z, Rigby P, Wolfe R, Elia M. Enteral nutritional support in prevention and treatment of pressure ulcers: A systematic review and meta-analysis. *Ageing Res Rev*, 2005; 4(3): 422-450.
111. Heyman H, Van De Looverbosch DEJ, Meijer EP, Schols J. Benefits of an oral nutritional supplement on pressure ulcer healing in long-term care residents. *J Wound Care*, 2008; 17(11): 476-478.
112. Leigh B, Desneves K, Rafferty J, Pearce L, King S, Woodward MC, Brown D, Martin R, Crowe TC. The effect of different doses of an arginine-containing supplement on the healing of pressure ulcers. *J Wound Care*, 2012; 21(3): 150-156.
113. Arinzon Z, Peisakh A, Berner YN. Evaluation of the benefits of enteral nutrition in long-term care elderly patients. *J Am Med Dir Assoc*, 2008; 9(9): 657-662.
114. Breslow RA, Hallfrisch J, Goldberg AP. Malnutrition in tubefed nursing home patients with pressure sores. *J Parenter Enteral Nutr*, 1991; 15 (6): 663-668.
115. Teno J, Gozalo P, Mitchell S, Kuo S, Fulton A, Mor V. Feeding tubes and the prevention or healing of pressure ulcers. Natural history of feeding-tube use in nursing home residents with advanced dementia. *Arch Intern Med*, 2012; 172(9): 697-701.
116. Harvey SE, Parrott F, Harrison DA, Zia Sadique M, Grieve RD, Canter RR, McLennan BKP, Tan JCK, Bear DE, Segaran E, Beale R, Bellingan G, Leonard R, Mythen MG, Rowan KM. A multicentre, randomised controlled trial comparing the clinical effectiveness and cost-effectiveness of early nutritional support via the parenteral versus the enteral route in critically ill patients (CALORIES). *Health Technol Assess*, 2016; 20(28): 1-143.
117. Institute of Medicine. *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate* 2004 [cited 2013]; Available from: <http://www.iom.edu/Reports/2004/Dietary-Reference-Intakes-Water-Potassium-Sodium-Chloride-and-Sulfate.aspx>.
118. Clark M, Schols JMGA, Benati G, Jackson P, Engfer M, Langer G, Kerry B, Colin D. Pressure ulcers and nutrition: A new European guideline. *J Wound Care*, 2004; 13(7): 267-272.
119. Mehta N, Compher C, ASPEN Board of Directors. A.S.P.E.N. Clinical guidelines: Nutrition support of the critically ill child. *J Parenter Enteral Nutr*, 2009; 33(3): 260-276.
120. Mehta N, Corkins M, Lyman B, Malone A, Goday P, et al. Defining pediatric malnutrition: A paradigm shift towards etiology-related definitions. *J Parenter Enteral Nutr*, 2013; 37(4): 460-481.
121. Ranade D, Collins N. Children with wounds: the importance of nutrition. *Ostomy Wound Manage*, 2011; October: 14-24.
122. Rodriguez-Key M, Alonzi A. Nutrition, skin integrity, and pressure ulcer healing in chronically ill children: An overview. *Ostomy Wound Manage*, 2007; 53(6): 56-62.
123. Joosten KF, Hulst JM. Nutritional screening tools for hospitalized children: Methodological considerations. *Clin Nutr*, 2014; 33(1): 1-5.
124. Secker DJ, Jeejeebhoy KN. How to perform Subjective Global Nutritional assessment in children. *J Acad Nutr Diet*, 2012; 112(3): 424-431.e6.
125. Secker DJ, Jeejeebhoy KN. Subjective Global Nutritional Assessment for children. *Am J Clin Nutr*, 2007; 85(4): 1083-9.
126. Minocha P, Sitaraman S, Choudhary A, Yadav R. Subjective Global Nutritional Assessment: A reliable screening tool for nutritional assessment in cerebral palsy children. *Indian J Pediatr*, 2018; 85(1): 15-19.
127. Dietetics and Food Services of Queensland Children's Hospital. *Paediatric Nutrition Screening Tool*. 2013 [cited May 2019]; Available from: <https://www.childrens.health.qld.gov.au/wp-content/uploads/PDF/pnst-form.pdf>.
128. White M, Lawson K, Ramsey R, Dennis N, Hutchinson Z, Soh XY, Matsuyama M, Doolan A, Todd A, Elliott A, Bell K, Littlewood R. Simple nutrition screening tool for pediatric inpatients. *J Parenter Enteral Nutr*, 2016; 40(3): 392-398.
129. British Dietetic Association, Centra Manchester University Hospitals. *Screening Tool for the Assessment of Malnutrition in Pediatrics* 2010 [cited May 2019]; Available from: http://www.stampscreeningtool.org/data/pdfs/stamp_tool.pdf.
130. McCarthy H, Dixon M, Crabtree I, Eaton-Evans M, McNulty H. The development and evaluation of the Screening Tool for Assessment of Malnutrition in Paediatrics (STAMP) for use by healthcare staff. *J Hum Nutr Diet*, 2012; 25: 311-318.

131. Wong S, Graham A, Hirani SP, Grimble G, Forbes A. Validation of the Screening Tool for the Assessment of Malnutrition in Paediatrics (STAMP) in patients with spinal cord injuries (SCIs). *Spinal Cord*, 2013; 51(5): 424-9.
132. Nutrition Tool Steering Group Women and Children's Directorate NHS Greater Glasgow and Clyd. *Paediatric Yorkhill Malnutrition Score: Information and User's Guide* 2009 [cited May 2019]; Available from: <http://www.knowledge.scot.nhs.uk/media/2592959/pyms%20user%20and%20info%20guide.pdf>.
133. Lestari NE, Nurhaeni N, Wanda D. The Pediatric Yorkhill Malnutrition Score is a reliable malnutrition screening tool. *Compr Child Adolesc Nurs*, 2017; 40(sup1): 62-68.
134. Ling R, Hedges V, Sullivan P. Nutritional risk in hospitalised children: An assessment of two instruments. *E Spen Eur E J Clin Nutr Metab* (available from [https://clinicalnutrition.espen.com/article/S1751-4991\(11\)00008-4/pdf](https://clinicalnutrition.espen.com/article/S1751-4991(11)00008-4/pdf)), 2011(6): e153-e157.
135. Huysentruyt K, Alliet P, Muyschont L, Rossignol R, Devreker T, Bontems P, Dejonckheere J, Vandenplas Y, De Schepper J. The STRONG(kids) nutritional screening tool in hospitalized children: A validation study. *Nutrition*, 2013; 29(11-12): 1356-61.
136. Hulst JM, Zwart H, Hop WC, Joosten KF. Dutch national survey to test the STRONGkids nutritional risk screening tool in hospitalized children. *Clin Nutr*, 2010; 29(1): 106-11.
137. Skillman J, Wischmeyer P. Nutrition therapy in critically ill infants and children. *J Parenter Enteral Nutr*, 2008; 32(5): 520-534.
138. Thompson KL, Leu MG, Drummond KL, Popalisky J, Spencer SM, Lenssen PM. Nutrition interventions to optimize pediatric wound healing: An evidence-based clinical pathway. *Nutr Clin Pract*, 2014; 29(4): 473-482.
139. Burd RS, Coats RD, Mitchell BS. Nutritional support of the pediatric trauma patient: A practical approach. *Respir Care Clin N Am*, 2001; 7(1): 79-96.

REPOSITIONING AND EARLY MOBILIZATION

Introduction

Repositioning and mobilizing individuals is an important component in the prevention of pressure injuries.¹ The underlying cause and formation of pressure injuries is multifaceted; however, by definition, pressure injuries cannot form without loading, or pressure, on tissue. Extended periods of lying or sitting on a particular part of the body and failure to redistribute the pressure on the body surface can result in sustained deformation of soft tissues and, ultimately, in tissue damage² (see the guideline chapter *Etiology of Pressure Injuries*).

Typically, a painful stimulus caused by the pressure on the tissue will motivate the individual to change position. Therefore, two primary concerns are the individual's ability to feel pain, and the person's actual physical ability to move or reposition.² Repositioning involves a change in position of the lying or seated individual undertaken at regular intervals, with the purpose of relieving or redistributing pressure and enhancing comfort. Mobilization involves assisting or encouraging a person to move or shift into a new position. Individuals who cannot reposition themselves will require assistance in this activity.

Recommendations in this section of the guideline address the role of repositioning and early mobilization in both the prevention and treatment of pressure injuries. The recommendations are relevant to all individuals with or at risk of pressure injuries, unless otherwise stated. Repositioning in relation to heel pressure injuries are discussed in a separate guideline chapter, *Heel Pressure Injuries*.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- How often should repositioning be performed to reduce the risk of pressure injuries?
- What criteria should be used to determine and monitor frequency of turning?
- What positioning techniques are most effective in redistributing pressure and preventing shear?
- Do programs of early mobilization affect pressure injury rates?

General Repositioning for All Individuals

5.1: Reposition all individuals with or at risk of pressure injuries on an individualized schedule, unless contraindicated.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Evidence from one high quality Level 1 study² and one moderate quality Level 1 study³ demonstrated that repositioning individuals more regularly is associated with a lower incidence of pressure injuries. However, the evidence is conflicting regarding potential differences between different turning frequencies. Evidence from two high^{4,5} and one moderate⁶ quality Level 1 studies showed no significant reduction in pressure injury incidence associated with more frequent repositioning. However, in one of these high quality Level 1 studies,⁵ all repositioning regimens were associated with pressure injury incidence below 3.1%. A moderate quality Level 3 study⁷ reported statistically significant difference between different repositioning frequencies, reporting an incidence rate ratio of 1.12 (95% confidence interval [CI] 0.52 to 2.42) for frequent repositioning compared with infrequent repositioning.

The Level 1 studies²⁻⁶ demonstrated that different repositioning frequencies (e.g. two, three or four hourly) are all at least somewhat effective. Reported variations in pressure injury incidence for different repositioning frequencies could be explained by the range of pressure injury risk for individuals in the studies, and the support surfaces used. Mattresses used in early studies may also be less effective than contemporary support surfaces. Adverse events associated with repositioning were a possibility of the individual experiencing increased pain during repositioning.^{8,9} High quality Level 1 evidence and moderate quality Level 3 evidence reported adherence to repositioning regimens ranging between 53% and 82%.⁵⁻⁷ Two high quality economic analyses demonstrated that costs of implementing frequent repositioning in aged care facilities were not substantial and were related to improvement in quality-adjusted life years.^{10,11} Level 5 evidence suggested that patients and informal caregivers place high importance on understanding more about the role of repositioning in preventing pressure injuries.¹²

Optimal offloading maximizes the redistribution of pressure away from bony prominences. However, individual anatomy may vary. Some positions may offload pressure points in one individual but be inadequate in offloading pressure for another individual.

Implementation Considerations

- The individual's physical, cognitive and psychological condition, and type of support surface in use should be considered when planning repositioning needs. See recommendations in this guideline chapter on factors to consider when assessing the need for repositioning and when determining an individualized repositioning schedule.
- Encourage and educate individuals who are able to regularly reposition themselves when in bed and seated. For individuals with SCI, provide education on repositioning during initial rehabilitation and regularly thereafter (*Expert opinion*).
- Establish and document individualized pressure relief schedules that prescribe the frequency and duration of weight shifts (*Expert opinion*).
- Reconsider the frequency and method of repositioning if the individual is not responding as expected to the repositioning regimen (*Expert opinion*). See the guideline chapter on Skin and Soft Tissue Assessment recommendations on assessments of the skin that can be used to evaluate the skin's response.
- Record when the individual was repositioned, specifying frequency and position adopted, and include an evaluation of the outcome (*Expert opinion*).

Evidence Discussion

Repositioning is undertaken to reduce the duration and magnitude of pressure over vulnerable areas of the body and to contribute to comfort, hygiene, dignity, and functional ability. Because repositioning is considered a necessary intervention to relieve pressure, most studies do not compare repositioning to no repositioning. However, one randomized controlled trial (RCT)² (n = 838) has been conducted in a nursing home in which standard repositioning was not a component of the routine preventive care received by a control group. In this study, the control group (n = 576) received a range of high specification support surfaces including water mattresses, alternating mattresses, sheepskins or gel cushions but no standard repositioning. Four intervention groups received preventive regimens that included a range of repositioning intervals and different support surfaces. Two groups received a standard foam mattress and were turned either two hourly (n = 65) or three hourly (n = 65). Two groups received a viscoelastic polyurethane foam mattress and were turned four hourly (n = 67) or six hourly (n = 65). The incidence of Category/Stage II or greater pressure injuries was significantly lower in the group receiving four hourly repositioning (odds ratio [OR] = 0.12; 95% CI 0.03 to 0.48), and there was a significant increase in the time to pressure injury development for the four hourly turning group compared to all other groups (p = 0.001).² However, there were several confounding factors, including the large variety of support surfaces used and the possibility that the time spent implementing repositioning regimens in the intervention groups impacted upon the care of individuals in the control group (*Level 1*).

In study conducted by Moore et al. (2011)³ among older adults in 12 aged care facilities, a frequent (three hourly) repositioning regimen was compared to an infrequent repositioning regimen (six-hourly). The experimental group were repositioned every three hours using the 30° tilt position (left side, back, right side, back) between 8pm and 8am (n = 99). In the control group, participants received routine repositioning every six hours between 8pm and 8am using 90° lateral rotation (n = 114). Day time care remained routine for all participants. Fewer participants in the experimental group developed a pressure injury (3% versus 11%; p = 0.03, intraclass correlation [ICC] = 0.001; incidence rate ratio [IRR] = 0.27 (95% CI 0.08 to 0.93, p = 0.038, ICC = 0.001). The OR of a pressure injury in the experimental group was 0.243 (95% CI 0.067 to 0.879, p = 0.034).³ The six hourly frequency of repositioning in the control group may not be considered as standard care in many facilities (*Level 1*).

Numerous studies⁴⁻⁷ have explored the influence of different repositioning frequencies on pressure injury incidence in aged care, acute care and critical care settings, but report conflicting findings. In the critical care setting, Manzano et al. (2014)⁶ compared repositioning two hourly or four hourly among individuals who were mechanically ventilated (n = 164). Participants were repositioned either two or four hourly in three positions (left and right 30° tilt position plus supine with 30° elevation). No significant difference was observed in pressure injury incidence between the two hourly turning group (10.3%) and the four hourly group (13.4%; unadjusted hazard ratio [HR] 0.89, 95% CI 0.46 to 1.71, p = 0.73). Although repositioning was interrupted for those experiencing hemodynamic or respiratory instability, compliance with the assigned regimen did not differ significantly between the groups (both approximately 60%). As with the Vanderwee et al. (2007)⁴ study, this RCT also lacked statistical power to measure a clinically significant effect (*Level 1*).

Vanderwee et al. (2007)⁴ followed 235 individuals in aged care who all received a viscoelastic foam mattress. In the intervention group, individuals were repositioned to alternate two hours in a lateral position and four hours in a

supine position. The control group were repositioned every four hours, first in lateral and then in supine. There was no statistically significant difference in incidence of Category/Stage II or greater pressure injuries (16.4% versus 21.2%, $p = 0.40$). However, the study did not recruit enough participants to meet the desired power⁴ (*Level 1*). Also conducted in an aged care setting, Bergstrom et al.'s (2013)⁵ Turning for Ulcer Reduction (TURN) study sought to determine optimal repositioning frequency for older adults ($n = 942$). Residents at moderate or high risk for pressure injuries according to the Braden Scale were randomly allocated using risk stratification to a repositioning schedule of two, three or four hourly. All individuals were placed on high density foam mattresses, although the brand of mattress varied among study sites. There was no significant difference in pressure injury incidence according to repositioning regimen (2-hourly, 2.5%; 3-hourly, 0.6%; 4-hourly, 3.1%, $p = 0.68$), nor was there a statistically significant difference in the incidence of pressure injuries between the high and moderate risk groups ($p = 0.79$). The study was limited to three weeks and relied on documentation and monthly fidelity checks to determine if there was compliance with the assigned regimens (*Level 1*).

In a cohort study investigating the association between being frequently repositioned and pressure injury incidence in an acute care setting,⁷ individuals at high risk based on Braden Scale score had a lower incidence of pressure injuries if they were frequently turned (IRR = 0.39, 95% CI 0.08 to 1.84). In this three-week study, turning was considered frequent if there were at least 12 repositionings per day. The investigators relied upon nursing documentation as the sole indicator that an individual had been repositioned (*Level 3*).

For a very limited number of medical conditions where movement destabilizes the condition of those who are critically ill, it may not be safe to turn the individual.¹³ Recommendations specific to repositioning critically ill individuals are included in this chapter.

Many individuals may have some mobility limitations but are still able to actively redistribute pressure. For example, most individuals with SCI can actively participate in pressure redistribution through repositioning, unless they have comorbidities that interfere with bed and seating mobility. Bed mobility (e.g., rolling, side-lying, prone positioning and recumbent positioning) and seated weight redistribution (shifting, pelvic and leg repositioning) should be taught during initial rehabilitation and then retrained and reinforced during ongoing admissions and contacts with health professionals.¹⁴ The guideline chapter *Quality of Life, Self-Care and Education* includes evidence on education needs and strategies.

Repositioning Frequency

5.2: Determine repositioning frequency with consideration to the individual's level of activity and ability to independently reposition.

(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

A moderate quality Level 4 study¹⁵ provided evidence that when individuals can reposition independently, they experienced no pressure injuries. The study showed that individuals repositioned themselves within a four-hour duration.¹⁵ Indirect evidence from observational studies¹⁶⁻¹⁹ showed that many hospitalized adults are independently mobile and active. Observed individuals reposition themselves regularly in bed (or in the chair if they are wheelchair bound). One study classified hospitalized adults as sedentary although they continued to perform self-initiated activity frequently. Understanding the individual's level of activity and ability to reposition themselves helps determine the frequency and amount of assistance they will require in repositioning.

Implementation Considerations

- Undertake an assessment of the individual's level of activity and ability to reposition independently as a part of every risk assessment.²⁰⁻²⁸ Information on indicators of mobility and activity are discussed in the guideline chapter *Risk Factors and Risk Assessment*.
- Ensure that self-repositioning is effective in adequately off-loading bony prominences (*Expert opinion*). Refer to *Recommendation 5.14* for more discussion on pressure relieving maneuvers that can be used by individuals who are able to self-reposition in a seated position.
- Recognize that some individuals may be damaging tissues with excessive movement. For example, individuals with agitation or who regularly drag when self-repositioning can expose the skin and tissue to shear forces (*Expert opinion*).

Evidence Discussion

When planning an individual's repositioning schedule, it is important to first assess risk of pressure injuries, paying particular attention to level of activity and mobility, because individuals with reduced activity and mobility are more prone to pressure injury damage.

A 3-month prospective case series conducted in an acute care setting¹⁵ explored whether self-repositioning by individuals (n = 112) was sufficient to prevent pressure injuries. A continuous pressure mapping system was used to detect pressure distribution of individuals who had the ability to move in bed without assistance. Only two participants had a duration of four hours or more with no movement, indicating that self-repositioning occurred regularly. No individuals experienced a pressure injury. However, 61% percent of participants were aged under 65 years and 75% had a Braden Scale score above 18, suggesting the study population had relatively low risk for pressure injuries. Additionally, 84% of participants were followed for 24 hours or less (*Level 4*).

A number of studies¹⁶⁻¹⁹ provide indirect evidence analysing individuals who are able to self-reposition. Although these studies do not provide evidence on the relationship between pressure injury incidence and activity, they provide information on the repositioning patterns of individuals in a variety of clinical settings.

McInnes et al. (2013)¹⁸ established that hospitalized individuals (n = 26) assessed as being at risk of developing a pressure injury reposition themselves regularly throughout the day. Movement data collected by trained observers showed the median number of position changes was 3.0 (IQR 2.50, range 1 to 9) on day shift, 4.0 (IQR 3.0, range 0 to 7) on afternoon shift and 4.0 times (IQR 3.0, range 1 to 8) overnight. Participants primarily assumed the supine (46° to 90°) position or sat out of bed in the early part of the day and were more often observed in the supine position (1° to 45°) in the latter part of the day¹⁸ (*Level 5*). In an analysis of 52 participants in hospitals and nursing homes, Källman, et al. (2015)²⁹ explored factors associated with repositioning induced by nursing staff, as well as the individual's own spontaneous movement frequency. There was a large variation in the frequency of spontaneous movements during both day (median 16, Q1 5 to Q3 52) and night (median 10, Q1 4 to Q3 33). Analgesics were positively related to the spontaneous movement frequency and psycholeptics had a negative association with self-repositioning. No relationship was established between staff induced repositioning and the frequency of spontaneous movement; however, nurses more often repositioned individuals assessed as having a high risk pressure injury risk²⁹ (*Level 5*). Chaboyer et al. (2015)¹⁶ observed hospitalized individuals at risk of pressure injury (n = 84) in order to describe physical activity patterns (duration of activity, repositioning frequency). Participants with reduced mobility wore a physical activity monitor for a continuous 24-hour period. Data showed that most time was spent in sedentary activity (94% ± 3%), although the median number of position changes (change of posture at least of 10° maintained five minutes or longer) was 94 times over 24 hours (range 11 to 154).¹⁶ These findings indicate that repositioning of individuals with some degree of restricted mobility occurred regularly; however, it was not known whether this was performed independently or with assistance (*Level 5*). Finally, Sonenblum et al. (2016)¹⁹ reviewed the pattern of self-positioning of individuals with spinal cord injury (SCI, n = 28) who were independently mobile in wheelchairs. Individuals weight-shifted an average of 2.4 ± 2.2 times per hour and performed pressure reliefs an average of 0.4 ± 0.5 times per hour. These repositioning patterns represented primarily male individuals with a mean age of 41 years who were active in their wheelchairs for between four and ten hours per day.¹⁹ It was unclear if the extent of repositioning adequately provided relief to the tissues.

5.3: Determine repositioning frequency with consideration to the individual's:

- Skin and tissue tolerance
- General medical condition
- Overall treatment objectives
- Comfort and pain.

(Good Practice Statement)

Implementation Considerations

- Regularly assess the individual's skin condition and general comfort. Reconsider the frequency and method of repositioning if the individual is not responding as expected to the repositioning regime (*Expert opinion*).
- Refer to the *Skin and Tissue Assessment* chapter for evidence-based recommendations on assessing skin and tissue tolerance.
- Evaluate the need for analgesia prior to scheduled repositioning. When required, pre-medicate the individual 20 to 30 minutes prior to assisting with repositioning. Refer to the *Pain Assessment and Management* chapter for evidence-based recommendations on assessing and managing pain.

Discussion

No support surface provides complete pressure relief. Pressure is always applied to some area of the skin. Turning and repositioning for pressure redistribution must therefore occur regularly. The frequency of turning may vary with the pressure redistribution capacity of the support surface; however, the individual's response to pressure should always guide turning frequency. High risk individuals with poor tissue tolerance may require more frequent turning.

Frequent assessment of the individual's skin condition facilitates timely identification of the early signs of pressure damage and, as such, their tolerance of the repositioning regimen. A number of Level 1 and Level 2 prognostic studies³⁰⁻³⁴ indicate that skin changes are associated with increased risk of pressure injuries. Odds ratio of developing a Category/Stage II or greater pressure injury when non-blanchable erythema was identified ranged from 3.25 (95% CI 2.17 to 4.86)³⁰ to 7.98 (95% CI 2.36 to 39.97).³² Identifying skin changes early by conducting a skin assessment enables health professionals to adjust repositioning (and other interventions) to prevent pressure injuries. If changes in skin condition should occur, the repositioning care plan needs to be re-evaluated.

Ongoing assessment of the skin is necessary in order to detect additional skin damage. Skin that is still reddened from a previous episode of loading may be damaged and undergoing an inflammatory response or may still be in the process of tissue reperfusion. Slower and/or diminished reactive hyperemic responses have been demonstrated in individuals at risk of pressure injuries, including the elderly,³⁵⁻³⁷ critically ill individuals,³⁸ smokers,³⁹ individuals with diabetes mellitus,⁴⁰⁻⁴² and individuals with SCI.⁴³⁻⁴⁶ The rate of reperfusion is slower after the area is unloaded, and reperfusion may ultimately be inadequate to offset the oxygen debt created during periods of loading. These individuals may require either a longer recovery time before reloading a body surface and/or a support surface with better pressure redistribution.

General medical condition can influence how often it is possible to reposition the individual. Individuals who are critically ill may experience dyspnea or hemodynamic instability unless a specific position is maintained. It is also important to take into consideration the individual's overall treatment objectives. For example, certain medical conditions, such as respiratory or cardiac disorders, may mean that the individual becomes very dyspneic or hemodynamically unstable unless cared for in a particular position.

When determining repositioning frequency, consideration should be given to the individual's experience of pain, including both comfort and pain lying in one position and any pain experienced during repositioning.^{8,9} Evidence from a study in general hospital populations (i.e., individuals without pressure injuries) showed that pain is experienced during repositioning. The pain mean score on an 11-point numerical rating scale during repositioning was 4.9 ± 3.1^8 (Level 5). The experience of pain during repositioning was also reported in a qualitative study conducted in individuals with multiple sclerosis and pressure injuries. Participants reported pain during movement and pain related to repositioning equipment⁹ (Level 5). Comfort is of primary importance and may supersede preventive repositioning for individuals who are actively dying or have conditions causing them to have only a single position of comfort.

5.4: Implement repositioning reminder strategies to promote adherence to repositioning regimens.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Two Level 1 studies, one of high quality⁴⁷ and one of moderate quality⁴⁸ demonstrated that a facility-based intervention could improve health professional compliance with repositioning, leading to a reduction in pressure injury incidence. Auditory or visual feedback systems (in the evidence – wearable patient sensors⁴⁷ and musical chimes⁴⁸) can cue health professionals to round or undertake required repositioning. Health professional compliance with repositioning was significantly increased by 20% when the intervention was implemented in one of the studies.⁴⁷ Compliance with repositioning regimens was sub-optimal, reported at 67% in a study that implemented a facility-wide reminder system,⁴⁷ with indirect evidence suggesting the individual's gender, body mass index (BMI), age and Braden Scale score influence compliance rates.⁴⁹ Resource requirements and feasibility are likely to vary widely based on the type of intervention selected and the facility's location.

Implementation Considerations

- Select a repositioning reminder system (e.g., audio system or visual feedback) that is customized to the care setting. (*Expert opinion*)
- Reminder systems have been successfully used as a component in a multi-faceted quality improvement bundle⁵⁰⁻⁵³ (see the guideline chapter on *Implementing Best Practice in Clinical Settings*).

Evidence Discussion

A number of studies^{4,49} indicate that adherence by health professionals to repositioning regimens can be less than optimal. Pickham et al. (2018)⁴⁹ conducted a secondary descriptive study of turning data obtained from a RCT conducted in critical care. Data on turn angle magnitude or depressurization time was obtained for individuals (n = 555) with a wearable sternal sensor. A turn was categorized as having been performed when an angle above 20° was attained and maintained for at least one minute after turning. Compliance was recorded as 54%, with 39% of observed turns reaching the minimum angle threshold and 38% of individuals remaining in position for at least 15 minutes (depressurization). Nurses were less likely to be compliant with the repositioning regimen for individuals with a high BMI, those with lower Braden Scale score and for males. The findings reported by Pickham et al. (2018)⁴⁹ were similar to those of Vanderwee et al. (2007),⁴ who reported adherence to two and four hourly repositioning regimens by health professionals in an aged care setting as approximately 60%.

Interventions have been developed to promote compliance of health professionals with repositioning regimens. Yap et al. (2013)⁴⁸ trialed an intervention designed to prompt nurses and ancillary staff to reposition or encourage mobilization for individuals every two hours in a 12-month RCT conducted in ten long-term care facilities (n = 1,928). The intervention, which consisted of musical cues played over the public announcement system every two hours during daytime hours, was delivered in four facilities for 12 months. Four control facilities provided standard care for six months, then delivered the intervention for six months. A further two facilities provided standard care for the full 12-month study period. Odds ratio of a new pressure injury were lower in the intervention facilities (p = 0.08) for Minimum Data Set (MDS) 2.0 assessments and were significantly lower (p = 0.05) for MDS 3.0 assessments. Mean odds ratios suggested individuals in intervention facilities were 45% less likely than individuals in the comparison facilities to develop a new pressure injury (*Level 1*).

Cues to undertake repositioning, including a visual turn clock placed at the bedside⁵³ (*Level 2*), musical cues played in the facility^{50,51} (*Level 2*), and visual note flagging the bed of individuals at high risk of pressure injuries⁵² (*Level 2*) have been included as components in comprehensive, multi-faceted quality improvement programs. However, the contribution to reduction in pressure injuries from these individual interventions is hard to determine due to numerous prevention initiatives being used in these pressure injury prevention bundles. Further discussion is included in the guideline chapter *Implementing Best Practice in Clinical Settings*.

Two trials^{47,54} have explored the use of sensors and feedback data to assist health professionals to adhere to a repositioning regimen. In one study,⁵⁴ a pressure mapping device was placed under individuals at high risk of pressure injuries (Braden Scale score ≤ 12) and pressure images were relayed to a monitor. An alarm sounded in the nurses' station at a time interval set by the facility (in this study, every two hours). When reinforced by regular education and mandatory staff meetings, the system increased the frequency of repositioning from a mean of 240 minutes to a mean of 164 minutes. However, the study did not report the intervention's influence on pressure injury incidence (*Level 5*). Pickham et al. (2018)⁴⁷ conducted a large RCT (n = 1,312) in two intensive care units (ICUs) to assess whether a wearable sensor promotes optimal turning practices through increasing compliance of health professional with repositioning regimens, thereby preventing pressure injuries. The intervention group wore a sensor placed on the sternum that transmitted data regarding the individual's positioning, allowing analysis of the frequency and extent of repositioning. The group wearing the sensor experienced significantly fewer pressure injuries than the control group receiving usual care (0.7% versus 2.3%, OR = 0.33, 95% CI 0.12 to 0.90, p = 0.031). The total time with turning compliance was statistically significantly higher in the intervention group (67% versus 54%, 95% CI 0.08 to 0.13, p < 0.001), with the effect being more pronounced in individuals at high risk of pressure injuries (67% versus 47%, p < 0.001). However, turning magnitude and time with adequate pressure relief to tissues (in this study, at least 15 minutes) were not statistically different between groups (p > 0.05 for both) (*Level 1*).

Repositioning Techniques

5.5: Reposition the individual in such a way that optimal offloading of all bony prominences and maximum redistribution of pressure is achieved.
(Good Practice Statement)

Implementation Considerations

- Check all pressure points when repositioning the individual to ensure that pressure has been adequately offloaded according to repositioning goals (i.e., check that pressure points are receiving the relief that repositioning aims to provide) (*Expert opinion*).

- When repositioning individuals in the lateral side-lying position, offload the sacrococcygeal area without placing pressure on the trochanter (*Expert opinion*).
- Teach individuals who are able to provide some or all of their own pressure relief to reposition correctly and to do ‘pressure relief lifts’ or other pressure relieving maneuvers as appropriate (*Expert opinion*).
- Pay particular attention to the individual’s heels, which can be inadvertently exposed to continuous pressure even when the individual is repositioned frequently. The guideline chapter *Heel Pressure Injuries* provides evidence-based recommendations on repositioning the heels.
- In individuals who are sedated and ventilated, particularly neonates and infants, frequently reposition the head (*Expert opinion*).
- Avoid positioning the individual directly onto medical devices, such as tubes, drainage systems or other foreign objects. The guideline chapter *Device Related Pressure Injuries* provides evidence-based recommendations relevant to repositioning.

Additional considerations for individuals with a pressure injury

- Avoid positioning the individual on bony prominences with existing non-blanchable erythema (*Expert opinion*).

Discussion

When choosing a position for the individual, it is important to assess whether the pressure is actually relieved or redistributed. For example, it is possible to inadvertently place the individual in a position such that smaller areas of the body, such as the heels, are continuously exposed to pressure. Assessment of the individual’s skin condition will indicate areas of the body that are exposed to sustained pressure. Non-blanchable erythema is an indication of the early signs of pressure damage. Continued positioning on non-blanchable erythema will worsen the damage and result in more severe pressure injuries.

It is possible to inadvertently position an individual directly on top of a tube, drainage system or other foreign object (e.g., eating utensils, remote controls). This will cause a localized area of pressure that, if not corrected early enough, will result in development of a pressure injury. It is important to check that individuals are not lying directly on a medical device or foreign object (see the chapter *Device Related Pressure Injuries*).

5.6: Reposition the individual to relieve or redistribute pressure using manual handling techniques and equipment that reduce friction and shear.

(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

One low quality Level 2⁵⁵ study reported lower rates of pressure injuries associated with low friction turn sheets compared to a standard turning technique. One moderate quality Level 4 study⁵⁶ provided evidence that pressure injury incidence is around 5 to 7% lower in facilities that provide more powered manual handling equipment. Individuals in facilities with fewer mechanical lifting devices were more likely to be assessed as bedbound, increasing their pressure injury risk. However, having more powered mechanical lifts was associated with a small but statistically significant increase in fall incidents, which translated to an increased rate of fractures.⁵⁶ There was no evidence available on resource requirements or acceptability to individuals or their caregivers.

Implementation Considerations

- Avoid dragging when repositioning the individual because this may result in friction and shear (*Expert opinion*).
- Use moving and handling equipment to reposition the individual. Appropriate equipment assists in lifting the individual and reduces unintended drag (*Expert opinion*).
- Promote comfort when repositioning individuals (*Expert opinion*). Refer to *Recommendation 11.3* in the guideline chapter *Pain Assessment and Treatment* for further discussion of the role of repositioning in managing pressure injury pain and in balancing comfort and repositioning at end-of-life.
- Do not leave moving and handling equipment under the individual after use, unless the equipment is specifically designed for this purpose (*Expert opinion*).
- Moving and handling equipment manufactured from fabrics designed to reduce the risk of pressure injuries may be available.⁵⁷ The guideline chapter on *Preventive Skin Care* presents evidence on low friction fabrics.
- Provide individuals with assistive devices to promote bed and seated mobility¹⁴ (*Expert opinion*).

Evidence Discussion

Pressure injuries occur because of sustained mechanical loading and shear forces. Principles of safe manual handling that prevent exposure of skin to pressure and shear forces should be used to ensure safety of both the individual and the health professional. Individuals should never be dragged during transfer or repositioning. Rather, use devices and techniques that reduce risk of friction and shear (e.g., mechanical lifts, transfer sheets, two- to four-person lifts, and turn-assist features on beds). Use of a low friction turn sheet to reposition individuals in a trauma unit (n = 59) was associated with a lower rate of pressure injuries compared to positioning using standard manual handling techniques (20% versus 3.4%, p = 0.04). However, the study results could have been influenced by the use of different positioning aids (i.e., wedges and pillows of different quality) between the groups⁵⁵ (Level 2). A cross-sectional survey of 271 long term care facilities reported a significantly lower prevalence of pressure injuries in individuals at high risk in facilities with more than eight powered mechanical lifting (PML) aids compared with facilities that had four or less PML aids (14.94% versus 9.74%, p < 0.001). Methodological limitations of the study included self-selection of participating facilities and reliance on self-reported data⁵⁶ (Level 4).

Moving and handling equipment may create areas of localized pressure resulting in additional tissue damage.¹ Prolonged sitting on a transfer sling may increase heat, moisture and pressure. Sling material may interfere with pressure redistributing qualities of a support surface. Thus, transfer devices should not remain under the individual after use, unless the equipment has been specifically designed for this purpose (e.g., low friction textiles, as discussed in the *Preventive Skin Care* chapter).

Many individuals with impaired mobility may still be able to actively participate in repositioning, for example individuals with SCI. Individuals should be encouraged to lift rather than drag their bodies during repositioning and transfers. Appropriate assistive devices (e.g., sliding boards, bed rails or trapeze bars) assist in minimizing shear and friction during repositioning.¹⁴

5.7: Consider using continuous bedside pressure mapping as a visual cue to guide repositioning.
(Strength of Evidence = C; Strength of Recommendation = ↔)

Evidence Summary

The evidence on effectiveness of continuous bedside pressure mapping in preventing pressure injuries was mixed. A high quality Level 1 study⁵⁸ found no significance on the incidence or severity of pressure injuries when pressure mapping was implemented in a medical ward. However, a high quality Level 2 study⁵⁹ and a low quality Level 3 study⁶⁰ both reported significant reductions in pressure injury incidence in medical ICUs when pressure mapping was used. Patient consumers provided evidence that pressure mapping was not uncomfortable⁵⁸ on the bed and health professionals identified the intervention as both helpful in performing repositioning and easy to use,⁶⁰⁻⁶² but highlighted that education and training is required to implement pressure mapping.⁶³ No evidence on resource requirements was identified.

Implementation Considerations

- Health professionals require education, training and coaching when a continuous bedside pressure mapping system is implemented in the facility.⁶³ An education intervention that included 15 minutes of verbal instruction and hands on practice over a one week period significantly improved nursing knowledge⁶⁴ (Level 5).
- A continuous bedside pressure mapping system may inhibit the technical function of low-air-loss, air fluidized and other specialty beds by impeding air flow and/or the moisture vapor transmission^{59,60} (*Expert opinion*). Excess linen or incontinence padding could interfere with the accuracy of data from a continuous bedside pressure mapping system⁵⁹ (*Expert opinion*).

Evidence Discussion

As discussed in detail in the guideline chapter on *Risk Factors and Risk Assessment* (See *Recommendation 1.1*), interface pressure was a significant factor in the majority of studies that included this outcome in multivariable analyses exploring pressure injury risk factors. Three studies⁵⁸⁻⁶⁰ provided direct evidence on the effectiveness of pressure mapping with respect to preventing pressure injuries, and two studies^{63,64} provided additional indirect evidence on acceptability and use of pressure mapping. All studies used the same continuous bedside pressure mapping system that displays pressure points in real-time color imagery, visualizing the distribution of pressure at the body–mat interface.⁵⁸

Gunningberg et al. (2017)⁵⁸ conducted a pragmatic RCT (n = 190) evaluating the impact of pressure mapping, testing the hypothesis that using real time feedback would improve staff attention to repositioning. The system gave

immediate feedback to staff about the individual's pressure points, facilitating the implementation of repositioning. Both the intervention and control groups received standard pressure injury prevention care; the intervention group additionally received the pressure injury mapping system from admission to discharge (or 14 days at most). No significant difference in the prevalence or incidence of pressure injuries (at any anatomical site) was demonstrated. The incidence rate ratio between the groups was 1.13 (95% CI 0.34 to 3.79) (*Level 1*).

However, some studies have demonstrated advantages to using pressure mapping. Behrendt et al. (2014)⁵⁹ conducted a prospective controlled study (n = 422) in a medical ICU. Participants were assigned to beds equipped with a continuous bedside pressure mapping system or to standard care only, which included repositioning every two hours. Pressure injury incidence was 0.9% in the intervention group and 4.8% in the control group (p = 0.02) (*Level 2*). A retrospective chart review,⁶⁰ also set in a medical ICU, reported similar results. Incidence of pressure injuries in a cohort (n = 307) placed on a continuous bedside pressure mapping system was 0.3% compared to 5% in a historical control group (n = 320; p = 0.001)⁶⁰ (*Level 3*). These studies used less robust designs than the study reported by Gunningberg et al. (2017), and both confounding factors (e.g., the clinical condition of the participants that was poorly reported) and education over time may have contributed to the results.

Studies^{58,60,62,63} reporting the perceptions of health professionals who have used a continuous bedside pressure mapping system suggest that the technology provides increased awareness for health professionals regarding the importance of pressure injury prevention. Therefore, the intervention can be used as an adjunct to regular repositioning. Health professionals in a number of studies reported that continuous bedside pressure mapping was easy to use, and the real-time feedback assisted in selecting positions that relieved pressure^{58,60,62,63} (*Level 5*). Continuous bedside pressure mapping was demonstrated to contribute to the knowledge of health professionals when used as a component of an education program. Health professionals showed statistically significant improvement in knowledge scores (p = 0.002). Attitudes toward pressure injury prevention were unchanged; however, the health professionals scored highly in this area at the study commencement. After delivery of the training and introduction of bedside pressure mapping, peak interface pressures were significantly lower (p = 0.016), and health professionals implemented more preventive interventions (p = 0.012) (*Level 5*).

Repositioning Individuals in Bed

5.8: Use the 30° side lying position in preference to the 90° side lying position when positioning.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

The evidence comparing side lying positions is mixed. A moderate quality Level 1 study⁶⁵ reported use of repositioning regimen that included the 30° side lying position was associated with a significant reduction in pressure injury incidence. People who were positioned using a 90° side lying position were 3.7 times more likely to experience a pressure injury than those who were positioned using a 30° side lying position (OR = 0.27).⁶⁵ A low quality Level 1 study found no significant difference in pressure injury rates between the two positions. A moderate⁶⁶ quality Level 4 study indicated that the 30° side lying position was associated with lower mean skin temperature over the trochanter than in the 90° *side lying position*. A low¹⁷ quality Level 4 study indicated that the interface pressure was significant lower in the 30° side lying position compared to the 90° side lying position. A moderate quality economic analysis indicated that a repositioning intervention that used a 30° side lying position and three hourly repositioning was associated with lower costs than a repositioning intervention that used a 90° side lying position with six hourly repositioning.⁶⁵ Individuals and their caregivers rated positioning in bed as a high priority education topic.¹²

Implementation Considerations

- Encourage individuals who can reposition themselves to sleep in a 20° to 30° side lying position or flat in bed if not contraindicated (*Expert opinion*).
- Avoid lying positions that increase pressure, such as the 90° side lying position^{17,67} (*Level 5*).
- Instruct individuals to follow the recommended regimen of repositioning when they are independent with bed mobility. When the individual is dependent for bed mobility, health professionals and informal caregivers should be encouraged to follow this regimen (*Expert opinion*).

Evidence Discussion

Two studies reported a possible association between the level of lateral tilt and pressure injury incidence. In one study, the effectiveness of different positioning in reducing pressure injury incidence was examined in older adults (n = 213)

residing in long-term care.⁶⁵ Individuals in a control group were repositioned according to standard care (90° side lying position with repositioning every six hours over night). The intervention group were positioned three hourly overnight using 30° tilt (see *Figure 8.1*) and supine position. The 30° side lying position with three hourly repositioning was associated with significantly fewer Category/Stage I and II pressure injuries (3% versus 11%, $p = 0.03$, IRR 0.27, 95% CI 0.08 to 0.93, ICC = 0.001).⁶⁵ The more frequent repositioning used for the intervention group likely influenced the findings (*Level 1*). In the second study,⁶⁸ acutely ill older adults ($n = 46$) were randomized to receive three hourly repositioning overnight in either 30° side lying position or 90° side lying position. At 24 hours' follow-up, there was no significant difference in the rate of Category/Stage I pressure injuries (9% for the 90° side lying position versus 4% for the 30° side lying position, $p > 0.05$). There were two sacral pressure injuries in the 90° side lying position and one sacral pressure injury in the 30° side lying position. Two trochanter pressure injuries occurred in the 30° side lying position. However, this study was underpowered and had a short follow-up duration. Greater difficulty in attaining and remaining in the 30° side lying position and more individuals in that group reported difficulty maintain the position due to joint stiffness, anxiety or pain⁶⁸ (*Level 1*). A Cochrane review⁶⁹ pooled the findings from these two RCTs and established that there was no statistically significant difference between using 30° tilt three-hourly overnight or using 90° lateral rotation overnight (risk ratio [RR] 0.62, 95% CI 0.10 to 3.97, $p = 0.62$).

In an investigation of six different lying positions (supine 30° tilt, supine 0°, semi-fowler with 30° head-of-bed elevation, semi-fowler with 30° elevation of head and legs, lateral 30° and lateral 90°), Källman et al. (2013)⁶⁶ found that tissue blood flow and skin temperature over bony prominences was significantly lower in both lateral 30° and 90° side-lying positions compared to supine tilt positions, with neither position showing superior effect on outcomes⁶⁶ (*Level 4*).

Laboratory studies measuring indirect outcomes in healthy volunteers ($n = 83$),⁶⁷ showed that the 30° tilt position and the prone position resulted in lower interface pressure measurements than 90° side-lying position, which gave the highest interface pressure measurement (*Level 5*). A small study,⁷⁰ also in healthy subjects, showed that a 20° to 30° tilt position is optimal for reducing peak muscle and fat strain, with the ideal angle varying according to individual factors including BMI⁷⁰ (*Level 5*).



Figure 8.1: 30° tilt

5.9: Keep the head of bed as flat as possible.
 (Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

A small, low quality Level 1 study⁷¹ reported no new pressure injuries associated with using a head of bed elevation of 30° for one day and 45° for the next day. A small, high quality Level 4 study⁷² reported a rate of new pressure injuries of 9.1% when the head of bed was limited to 30° elevation for a median duration of ten days. The inconsistent findings could be related to the study durations.

Indirect evidence reporting interface pressures as an outcome measure were also inconsistent. The largest study showed no increase in interface pressure at the sacrum or trochanters when the head of bed was elevated, and scapula interface pressures decreased as elevation increased.⁷³ In other studies, as the angle of head of bed elevation increased the interface pressure increased at the sacrum⁷⁴⁻⁷⁶ and heels⁷⁶ and interface pressure decreased at the scapulas.⁷⁶ In another study, sacral interface pressure decreased as the angle of head of bed elevation increased.⁷⁷ Additional factors to the angle of head of bed elevation, including BMI, alertness and type of support surface, could influence interface pressures and explain variations in the findings in the literature.

A low quality Level 1 study⁷¹ reported that intubated individuals with gastric tubes had better tolerance for a 30° head of bed elevation compared with a 45° head of bed elevation.⁷¹ However, a high quality Level 4 study reported a compliance rate of only 53.6% with limiting the head of bed to a 30° elevation.⁷²

Implementation Considerations

- Maintaining a flat position should be evaluated with consideration to the individual's clinical needs and comfort. When elevating the head of bed, maintain elevations at 30° or lower to minimize soft tissue deformation (*Expert opinion*).
- Investigate alternatives to sitting in bed (e.g., sitting out of bed for some duration or during meals or gastric feeds) (*Expert opinion*).

- Avoid slouched positions that can increase pressure and shear on the sacrum and coccyx⁷⁸ (Level 5).
- Consider the individual's preferences and medical condition when positioning (Expert opinion).

Additional considerations for individuals with a pressure injury

- Avoid seating an individual with an ischial pressure injury in a fully erect posture in bed (Expert opinion).

Evidence Discussion

In supine position with the head-of-bed elevated, the sacrum is subjected to shear stress/strain and pressure. Pressure and shear are reduced when the head-of-bed is elevated at less than a maximum of 30° (see Figure 8.2).^{67,74,79-81}

Numerous studies^{56-58,64,74,82} provide indirect evidence that interface pressure at the sacrum increases when the head-of-bed is raised. This evidence suggests that the head-of-bed should be maintained as flat as possible, or below 30° elevation. In critically ill individuals at high risk of pressure injuries, Grap et al. (2016)⁸² established that interface pressure at the trochanter and sacrum increases as the head-of-bed elevation increases. The extent of interface pressure increases is also influenced by knee angle, BMI and the individual's movement (Level 5). In immobile, older adults at high risk of pressure injuries (n = 42), sacral and tuberosity interface pressures were significantly greater (all p < 0.001) with the head-of-bed elevated at 30°, 45° and 60° compared with a flat position (0° elevation). Head-of-bed elevation of 15° resulted in a non-significant increase in sacral and tuberosity interface pressure⁷⁴ (Level 5).

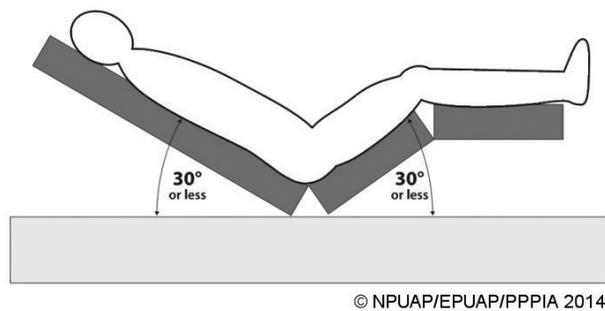


Figure 8.2: Maximum head of bed elevation

during ventilation (e.g., pneumonia). Elevation of 30° was compared to 40° head-of-bed elevation.⁷¹ No pressure injuries occurred in the study; however, the small sample size, short trial duration and cross-over trial design with an insufficient wash out period were significant limitations (Level 1). In a larger (n = 276) observational study,⁷² the head-of-bed elevation was measured three times daily for between three and 28 days (mean 20 days). Approximately 45% of the 6,894 head-of-bed elevation measurements made in the study were 30° or lower. The incidence of pressure injuries over the course of the study was 9.1%. The primary reasons cited by nurses for non-compliance with maintaining the head-of-bed below 30° were care needs and the clinical condition of the individual⁷² (Level 4).

Elevating the head of the bed may be medically necessary to facilitate breathing and/or prevent aspiration and ventilator associated pneumonia.^{71,72} In these cases, semi-Fowler's position is preferred.⁶⁷ Individuals should be positioned and supported to prevent sliding down in the bed and creating shear forces. A reclined or slouched posture should be avoided, as this causes weight bearing and shear on the sacrum and/or coccyx. Flexing the knees and positioning with pillows under the arms may prevent some sliding and slouching when the head-of-bed is elevated. For individuals with a pressure injury on the sacrum and/or coccyx, sitting erect on the side of the bed while eating may be a preferred option. Some integrated bed systems transform into a chair position; if such a bed is used, ensure that pressure is not placed directly on the pressure injury in this position, and place pillows under the arms to prevent slouching and sliding.

One study⁸⁴ investigated a positioning maneuver designed to decrease interface pressure and increase comfort for individuals in a high Fowler's position. The low technology trunk release maneuver was used in healthy, community-dwelling individuals (n = 117). In this maneuver, the individual's trunk was pulled forward and away from the support surface without lifting the buttocks from the support surface. There was a significant reduction in the peak pressure index associated with use of the trunk release maneuver (59.6 mmHg versus 79.9 mmHg, p = 0.002) and no differences in discomfort as rated by participants. Using the trunk release maneuver may reduce interface pressure for individuals sitting upright in bed⁸⁴ (Level 5). However, this repositioning strategy requires further exploration in individuals at risk of pressure injuries.

These findings are supported by laboratory-based studies conducted with healthy volunteers,^{75-77,83} all of which established that there is a significant increase (p < 0.05) in trochanter,⁷³ sacral^{73,75-77} and heel^{73,76} interface pressure associated with raising the head-of-bed to 30° or higher when the individual is positioned in either supine⁷⁵⁻⁷⁷ or 30° tilt positions⁸³ (Level 5).

Further, two studies^{71,72} set in ICUs have identified a direct relationship between head-of-bed elevation and pressure injuries. In one study,⁷¹ 11 individuals at high risk of pressure injuries were followed for two days to explore the feasibility of head-of-bed elevation to prevent clinical complications

Prone Position

5.10: Avoid extended use of prone positioning unless required for management of the individual's medical condition.
(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

One low quality Level 1 study⁸⁵ reported increases in pressure injury incidence in the prone position. In this study,⁸⁵ conducted in critically ill individuals, there was higher incidence of pressure injuries in prone position compared to supine position based on days in intensive care and days using mechanical ventilation; however when controlling for confounders the difference was not significant. One low quality Level 1 study,⁸⁶ and two moderate quality Level 4 studies^{87,88} reported incidence of pressure injuries experienced in the prone position was between 5% and 15% in critically ill individuals or individuals positioned in prone for surgical interventions. Understanding the influence of positioning on pressure injuries is considered an important topic by individuals and their informal caregivers. However, other factors, including medical condition or surgical procedure, influence the need to use prone positioning. Use of appropriate support surfaces and pillows⁸⁶ and repositioning as soon as feasible is important when the prone position cannot be avoided.

Implementation Considerations

- Use a pressure redistribution support surface or positioning devices to offload pressure points on the face and body while in the prone position.⁸⁶ (*Level 1*) Refer to the *Support Surfaces* chapter for further recommendations on effectiveness of different support surfaces.
- Once positioned check for uneven distribution of pressure and positioning of medical devices if possible. Pay particular attention to breast region, knees, toes, penis, clavicles, iliac crest and symphysis pubis. (*Expert opinion*)
- Consider using additional pressure injury prevention strategies such as prophylactic dressing under devices and over bony prominences (e.g., the iliac crest, ribs and patella). The guideline chapter on *Preventive Skin Care* presents evidence-based recommendations on the use of prophylactic dressings.
- At each rotation, assess the face and other body areas (i.e., breast region, knees, toes, penis, clavicles, iliac crest and symphysis pubis) that may be at risk when individuals are in the prone position. Refer to the *Skin and Soft Tissue Assessment* chapter for more information on conducting a skin assessment.

Evidence Discussion

For most individuals, limiting time spent in a prone position is a feasible intervention to prevent pressure injuries. However, some individuals have medical conditions or surgery that require use of prone position. Prone positioning is used more often in surgical settings and in critical care settings in which individuals may have medical conditions requiring use of prone positioning. For example, prone positioning is recommended for more than 12 hours/day for individuals with severe acute respiratory distress syndrome.⁸⁹

In healthy volunteers (n = 83), Defloor (2000)⁶⁷ found that average interface pressures were lower in prone position compared with 30° side lying position and 90° side lying position⁶⁷ (*Level 5*). However, individuals placed in the prone position may be at increased risk for the development of facial pressure injuries. In one small case series report (n = 15) conducted in a critical care setting, 13% (2/15) of participants with severe acute respiratory distress syndrome who were positioned in a prone position for ventilation (mean time in prone position 55 ± 7 hours) developed a Category/Stage II pressure injury on the face⁸⁷ (*Level 4*). Another study⁸⁶ conducted in the operating room using different facial support surfaces reported an incidence of facial pressure injuries of 15.1%. Non-blanchable erythema was found on 75% of the iliac and chest pressure points at the end of surgery, and between 5% and 10% of those pressure injuries persisted at 30 minutes following surgery⁸⁶ (*Level 1*).

An RCT⁸⁵ comparing early, long standing (≥ 16 hours) prone positioning to supine positioning explored whether extended positioning in prone was associated with pressure injuries. The study,⁸⁶ conducted in individuals with severe acute respiratory distress syndrome (ARDS; n = 466) found the incidence of new pressure injuries was significantly higher in prone position compared to supine position when measured by days in ICU (13.92 versus 7.72 per 1,000 ICU days, p = 0.002). After controlling for confounders, pressure injury incidence was statistically significantly different between groups at day seven (prone 42.5% versus supine 57.1%, p = 0.005), but was not statistically significantly different when measured as incidence at time of discharge from the ICU (prone 44.4% versus supine 37.8%, p = 0.151)⁸⁶ (*Level 1*).

The use of appropriate support surfaces and pillows is important to reduce pressure injury risk for individuals in the prone position. The effectiveness of different facial pillows for pressure injury prevention has been explored.^{86,88} Grisell and Place (2008)⁸⁶ conducted an RCT comparing three different facial support pillows (n = 66) in individuals requiring prone positioning for surgery. Time in prone position ranged from one hour to 12 hours. Forty-five percent of participants who received a disposable polyurethane foam facial pillow developed non-blanchable erythema or Category II pressure injuries. This compared to no facial pressure injuries experienced by individuals using either a protective helmet system of polyurethane foam or a neoprene air filled device (p = not reported) (*Level 1*). An observational study⁸⁸ measured presence of pressure injuries both immediately after and 30 minutes following surgery in prone positioning in individuals undergoing spinal surgery (n = 30). Individuals either received a high density 10 cm thick foam mattress or a 2 cm thick viscoelastic pad. Regardless of the type of support surface used, 75% of individuals experienced non-blanchable erythema of the iliac and chest pressure points immediately after surgery⁸⁸ (*Level 4*).

Repositioning and Pressure Redistribution for Seated Individuals

Pressure and shear forces are important considerations in the development of pressure injuries in seated individuals. Although consideration to optimal seated positioning is important for all individuals with or at risk of pressure injuries, these recommendations are of particular significance to people who spend extended time seated out of bed and have a high risk of pressure injuries due to reduced mobility and/or reduced sensory perception, such as people with SCI and older adults.

5.11: Promote seating out of bed in an appropriate chair or wheelchair for limited periods of time. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

A low quality Level 1 study⁹⁰ showed that limiting the duration of sitting sessions to a maximum of two hours for individuals at high risk of pressure injuries can reduce the incidence of pressure injuries compared with allowing individuals to sit out of bed for an unlimited duration. If an individual has an ischial pressure injury, sitting out of bed should be considered cautiously.

Implementation Considerations

- Limit time spent sitting out of bed for individuals at high risk of pressure injuries⁹⁰ (*Level 1*).
- Take psychosocial needs into account in balancing periods of bed rest and sitting in a chair or wheelchair (*Expert opinion*).
- Encourage individuals who spend time in seated position to implement weight shifts and pressure relief maneuvers. See *Recommendation 5.14*.
- Refer individuals with or at risk of pressure injuries who spend prolonged periods in a chair or wheelchair to a seating specialist (*Expert opinion*).
- Select a seated posture and chair that:
 - Is acceptable for the individual
 - Minimizes the pressures and shear exerted on the skin and soft tissues at high risk areas
 - Provides adequate support
 - Maintains stability
 - Maintains the individual's full range of activities (*Expert opinion*).
- Refer to the recommendations on seating position within this guideline for more information about positioning in a seated posture (*Expert opinion*).
- Use an appropriate seating support cushion when an individual is seated out of bed. Refer to the *Support Surfaces* chapter of the guideline for recommendations on selecting an appropriate support cushion.
- Do not use ring or donut-shaped positioning devices. The edges of these devices create areas of high pressure that may damage tissue. These devices may also impair circulation and create edema^{91,92} (*Expert opinion*).
- Perform regular skin and risk assessments when an individual is seated out of bed to determine the most appropriate duration of seating sessions. Refer to the guideline chapters on *Skin and Soft Tissue Assessment* and *Risk Factors and Risk Assessment* for more information on performing skin and risk assessments.

- Avoid sitting on medical devices if possible. Refer to the *Device Related Pressure Injuries* chapter of the guideline for recommendations on preventing device-related pressure injuries.
- Avoid leaving an individual seated on a bed pan, commode or toilet for longer than necessary (*Expert opinion*).

Additional considerations for individuals with a pressure injury

- Avoid seating an individual directly on a pressure injury (*Expert opinion*). Consider periods of bed rest to promote healing of ischial and sacral pressure injuries (Refer to *Recommendation 5.16* for further discussion).
- If sitting in a chair or wheelchair is necessary for an individual with pressure injuries on the sacrum, coccyx or ischia, limit sitting sessions to three times a day for durations of 60 minutes or less (*Expert opinion*).
- Modify sitting time schedules and re-evaluate the seating surface and the individual's posture if the pressure injury worsens or fails to improve (*Expert opinion*).

Transferring the individual to seating

- Use appropriate equipment such as a split leg sling mechanical lift when available to transfer an individual into a chair or wheelchair when the individual needs total assistance to transfer. Remove the sling immediately after transfer (*Expert opinion*).
- Do not leave moving and handling equipment under the individual after use, unless the equipment is specifically designed for this purpose (*Expert opinion*).
- Use transfer equipment manufactured from fabrics designed to reduce the risk of pressure injuries⁵⁷ (*Level 5*).

Evidence Discussion

Repositioning an individual to allow stability and full range of activities may be complex. For example, in an armchair that tilts back, the use of a footrest with the heels offloaded may be a suitable position in terms of pressure redistribution but this position may impede transferring to and from the chair.

A small RCT (n = 57)⁹⁰ investigated the effect of a sitting protocol restricted to two hours per session for participants who either had a fracture or had recently had major orthopedic surgery. All participants were placed on large-celled alternating mattress. Significantly fewer pressure injuries (7%) developed in individuals with fractures who sat for two hours or less per session than in those sitting in a chair for unlimited periods of time (63%) (p < 0.001) (*Level 1*).

Pressure and shear forces can cause sustained tissue deformation and reduced perfusion to tissues. While sitting is important for overall health, every effort should be made to avoid or minimize pressure and static shear force on an existing pressure injury. Continued pressure on an existing pressure injury will delay healing and may cause additional deterioration. In situations where positioning on the pressure injury cannot be avoided (e.g., when the individual has multiple pressure injuries on multiple surfaces), limit the amount of time the individual is positioned on the pressure injury, change support surfaces to provide better pressure redistribution, and use positioning techniques that redistribute pressure off the pressure injury as much as possible (e.g., use specially designed contour seating surfaces or 'bridging' areas around the pressure injury with positioning devices that offload the pressure injury and redistribute pressure to surrounding tissue). Because any intense pressure reduces blood flow and impairs healing, sitting time must be limited to one hour three times daily; the sitting time should correspond to mealtime. Sitting times can be increased or decreased based on the improvement or deterioration of the pressure injury. Periodic shifting, tilting forward, or lift-offs (pressure relief maneuvers) while sitting may also facilitate some reperfusion.

5.12: Select a reclined seated position with the individual's legs elevated. If reclining is not appropriate or possible, ensure that the individual's feet are well-supported on the floor or on footrests when sitting upright in a chair or wheelchair.

(Strength of Evidence = B2; Strength of Recommendation = ↑)

5.13: Tilt the seat to prevent the individual sliding forward in the chair or wheelchair.

(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

A low quality Level 4 study⁹³ provided evidence that skin perfusion significantly increases when tilt-in-space is combined with a reclined position. Additional indirect evidence from two studies conducted in individuals with SCI^{94,95} and two studies^{78,96} conducted in healthy volunteers demonstrated that interface pressure at the sacrum is significantly lower when a reclined seating position is adopted. Supporting the individual's feet prevents sliding down in the chair and slouching, which indirect evidence indicated were both associated with increased pressure.⁷⁸

Indirect evidence^{94,95} also indicates that interface pressure at the sacrum, ischial tuberosities and coccyx is reduced when the seating surface is tilted, with more significant reductions in pressure attained with tilts of at least 30°. Shear forces from sliding forward in the chair or wheelchair are likely to be reduced when the individual is tilted to the rear.

Implementation Considerations

- Avoid seating an individual with an ischial pressure injury in a fully erect posture in chair/wheelchair⁷⁸ (*Level 5*).
- Do not use seat recline or seat tilt as a method of restraining an individual (*Expert opinion*).
- In most situations, 30° seat tilt is adequate to prevent sliding and to redistribute pressure and reduce shear forces. Tilt the wheelchair before reclining. (*Expert opinion*).
- Develop an individualized plan for repositioning using dynamic weight shifting that is informed by:
 - Pressure mapping
 - Evaluation of the skin's response to pressure
 - The individual's functional ability and lifestyle¹⁴ (*Expert opinion*).
- Adjust the footrests and armrests to maintain the individual's posture and to maximize pressure redistribution (*Expert opinion*).
- Use a pressure redistribution cushion. Refer to the *Support Surfaces* chapter for discussion regarding selection of wheelchairs, seats and cushions.

Evidence Discussion

Dynamic weight shifting uses assistive technologies to relieve pressure when the individual has limited ability to effectively perform intentional weight shifting due to paralysis. Use of dynamic weight shifting is of particular significance for individuals with reduced mobility who spend prolonged periods of time in a chair/wheelchair, such as those with spinal cord injury (SCI).

One study compared center of pressure displacement in individuals with SCI to that of healthy volunteers. The participants sat in a static position on a hard, backless chair with appropriate foot support while pressure mapping was performed. Center of pressure displacement was significantly lower in individuals with SCI than healthy volunteers ($p < 0.05$), indicating some impairment in dynamic sitting stability. Significant differences were noted in center of pressure displacements during forward and backward leaning for individuals with SCI who had a past history of pressure injuries⁹⁷ (*Level 5*). Thus, impaired ability to dynamic weight shift appears to contribute to redistribution of pressure in seated positions.

The ischial tuberosities bear intense pressure when the individual is seated. Indirect evidence conducted in both healthy volunteers and individuals with SCI reporting interface pressure as an outcome measure has demonstrated significant reductions in peak pressure at the ischia, sacrum and back associated with both reclined seating,^{78,94,96,98} and with seat tilt and recline in combination^{93,94} (all *Level 5*). In one of these studies conducted with individuals with SCI ($n = 18$),⁹⁴ relative reduction in interface pressure was systematically measured at the ischial tuberosities and sacrum through 10° increments of tilt in a manual wheelchair. A reduction in sacral pressure did not occur until a 30° tilt⁹⁴ (*Level 5*). A minimum tilt of 30° is needed to achieve a clinically important reduction in pressure at the ischial tuberosities^{78,94,96} (*Level 5*).

However, clinical research showing direct support for the use of reclined seating and, where possible, a rear-tilted seat, is limited. Evidence demonstrates a positive effect on increasing blood perfusion of the skin compared to seating without tilt or recline. As discussed in the *Etiology* chapter of this guideline, reduced vascular flow from occlusion of blood vessels results in ischemia-induced damage.⁹⁹ Thus, a measurable increase in skin perfusion plausibly suggests a reduction in conditions that support development of pressure injuries. Skin perfusion was measured at the ischial tuberosities in individuals with SCI ($n = 11$) who used a powered wheelchair in one observational study.⁹³ After trialing six different protocols with combinations of different angles of tilt-in-space and recline, the evidence showed that skin perfusion increases when there is a seat tilt of 15°, 25° or 35° combined with a 120° recline, or when a 35° seat tilt is combined with 100° recline⁹³ (*Level 5*). A small study in individuals with SCI ($n = 13$), demonstrated significant reductions in ischial, sacral and coccygeal interface pressures when tilt and recline were used in combination. Interface

pressure varied at the different anatomical locations depending on the angles of tilt and recline⁹⁵ (*Level 5*). This suggests that seating position should be adjusted based on the individual's needs, and that pressure can be redistributed to some degree by adjusting tilt and recline angles. Pressure mapping, functional ability assessment, evaluation of the skin response to pressure relief and consideration to the individual's lifestyle should all contribute to development of an individualized repositioning plan.¹⁴

Maintenance of proper positioning and postural control is important. To avoid shear and friction, select a seat with an appropriate seat-to-floor height for the individual. When the feet do not rest on the floor, the body slides forward out of the chair. Defloor et al. (1999)⁷⁸ established that interface pressure is significantly lower ($p < 0.001$) when the feet of an individual seated in an upright position are on the ground compared with supporting the legs with a rest (*Level 5*). Miller et al. (2014)⁹⁶ achieved similar results, demonstrating significant reductions compared to upright seating in average and maximum sacral interface pressure when the legs of healthy volunteers were elevated with a support, and the seat was reclined (*Level 5*). Having the feet unsupported may cause excessive pressure behind the knee, impeding circulation. An armchair helps maintain posture and is associated with lower pressure than an armless chair (see *Figure 8.3*).⁷⁸

If the individual's feet cannot be positioned directly on the ground, footrest height should be adjusted so as to slightly tilt the pelvis forward by positioning the thighs slightly lower than horizontally. This position transfers weight (e.g., load) of the upper body onto the posterior thigh. When the footrest is too high, the load is applied to the posterior pelvic region, placing the stress back onto the ischia and coccyx, which may add stress to the feet. Seat depth should be sufficient to allow maximum pressure redistribution over the thighs⁷⁸ (see *Figure 8.3* and *Figure 8.4*) (*Level 5*).

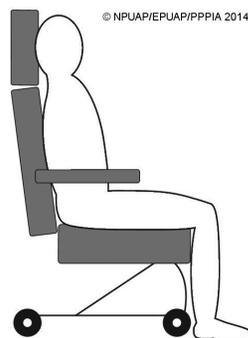


Figure 8.3: Sitting upright in an armchair with the feet on the ground

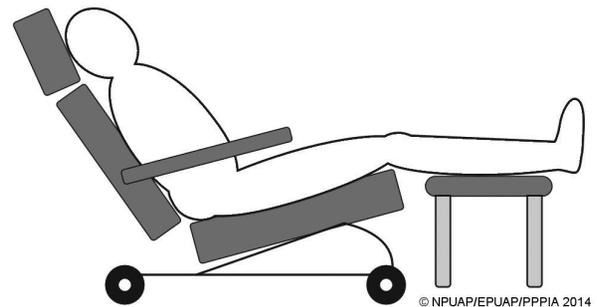


Figure 8.4: Sitting back in an armchair with the lower legs on a rest

Pressure Relieving Maneuvers

5.14: Teach and encourage individuals who spend prolonged durations in a seated position to perform pressure relieving maneuvers.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Evidence from a moderate quality Level 2¹⁰⁰ and 4 studies¹⁰¹ shows that performing intermediate or full leans while seated in a wheelchair significantly increases ischial blood flow. However, the evidence^{102,103} on an association between performing pressure relieving maneuvers and experiencing a pressure injury is mixed and does not include any comparative intervention studies. In one low quality Level 4 study¹⁰² individuals with spinal cord injury (SCI) who did not experience pressure injuries performed significantly more weight shifts per hour (effect size 0.39) than individuals who experienced a pressure injury, but in the same study¹⁰² there was no significant relationship between frequency of in-seat movements and experiencing a pressure injury. Additionally, a high quality Level 3 study¹⁰³ showed no significant relationship between pressure relief maneuvers and experiencing a pressure injury.

Implementation Considerations

- To effectively relieve pressure, pressure relieving maneuvers must at least partially unload the buttocks¹⁰⁰. This is of particular importance to individuals who spend prolonged time in a chair or wheelchair (e.g., individuals with impaired mobility living in the community) (*Level 4*).

- Teach individuals who spend prolonged periods in a seated position (e.g., wheelchair bound individuals with SCI and other immobile adults) to incorporate weight shifts into their daily routine as a part of functional activities (e.g., reaching and leaning),¹⁰² as well as performing intentional pressure relief maneuvers on a regular schedule¹⁴ (*Expert opinion*). The guideline chapter on *Quality of Life, Self-care and Education* discusses factors to consider when developing and delivering education for patient individuals and their informal caregivers.
- Develop an individualized pressure relief schedule outlining the frequency and duration of weight shifts based on the individual's routine and ability^{14,100} and on an assessment of the skin's response¹⁴ (*Expert opinion*).

Evidence Discussion

Individuals spending prolonged time in a chair or wheelchair, particularly those with SCI, should be encouraged to regularly relieve pressure. Pressure relieving maneuvers include intentional exercises, as well as weight shifting that occurs during functional activities (e.g., during leaning, reaching and propelling a wheelchair). Pressure relieving exercises include:^{14,100,101,104}

- Intentional weight shifting (leaning forward or sideward to a small, intermediate or full degree)
- Intermittent standing using assistive devices or with assistance
- Arm lifts/ pushups.

One study¹⁰⁰ compared the effects of a dynamic sitting protocol to a pushup protocol on tissue perfusion and interface pressure for individuals with paraplegia (n = 20) and tetraplegia (n = 20), with an additional control group of healthy individuals (n = 20). The dynamic sitting protocol consisted of upright sitting alternated every ten minutes with an offloaded seating configuration. The pushup protocol alternated normal sitting with a standard wheelchair pushup performed once every 20 minutes. Transcutaneous oxygen measured with an oximeter at the buttocks and ischial tuberosities significantly increased during pushups and increased at the ischial tuberosities in the offloaded sitting position for all individuals. However, the researchers noted that tissue perfusion recovery time was significantly longer in individuals with SCI compared with healthy controls (p < 0.001), suggesting efficacy is related to the performance ability¹⁰⁰ (*Level 2*). This suggests that selection of pressure relieving activities should be individualized according to strength and skill.

Two observational studies^{102,103} provide evidence on a possible relationship between performing pressure relief maneuvers and pressure injury incidence. In one study, participants were individuals with SCI of at least two years' duration who had either not experienced a history of recurrent pressure injuries (n = 12) or had experienced at least two pelvic pressure injuries (n = 17). Based on pressure mapping, individuals with a pressure injury history performed weight shifts significantly less frequently than the group who did not experience pressure injuries (2.5 times [95% CI 1.0 to 3.6] per hour versus 1.0 [95% CI 0.4 to 1.9] time per hour, p = 0.037, effect size [ES] = 0.39). The no pressure injury group also performed more in-seat movements per hour; however, the difference was not statistically significant (46.5 times [95% CI 28.7 to 76.7] per hour versus 39.6 [95% CI 24.3 to 49.7] times per hour, p = 0.352, ES = 0.17)¹⁰² (*Level 4*). In the second study,¹⁰³ comparison was made between individuals with SCI admitted to a rehabilitation center for treatment of a pressure injury (n = 31) and a cohort with SCI dwelling in the community (n = 30). In this study the group with pressure injuries spent a longer duration (hours per day) in a wheelchair (p = 0.002), but there was no significant difference in the number of pressure relief maneuvers performed each hour (pressure injury group 2.2 ± 3.3 versus no pressure injuries 1.8 ± 1.6, p = 0.664). The data collection relied on participant diaries and recall, which may have influenced the results. This study also found that individuals without pressure injuries were able to identify significantly more methods to relieve pressure (1.3 ± 0.6 versus 2.4 ± 1.4, p < 0.0001),¹⁰³ suggesting a relationship between knowledge, compliance and pressure injury incidence (*Level 3*).

The duration and frequency of pressure relieving strategies should be individualized. For example, active individuals with SCI will perform frequent, unintentional weight-shifting throughout the day while engaging in functional activities and may require less frequent intentional weight shifting maneuvers. Individuals who are less active in their normal daytime activity should perform intentional pressure relief more frequently. Regular inspection of the skin should also be used to guide the frequency and intensity of intentional pressure relief maneuvers.¹⁴

Early Mobilization

5.15: Implement an early mobilization program that increases activity and mobility as rapidly as tolerated. (Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Two low quality Level 2 studies^{105,106} reported significant reduction in unit-acquired pressure injuries associated with early mobilization programs. In these studies, there was a reduction of about 2-3% in the unit-acquired pressure injury rates after introduction of the mobility programs. However, a moderate quality Level 2 study¹⁰⁷ reported a significant increase in unit and facility acquired pressure injury rates associated with an early mobilization program, and another low quality Level 2 study¹⁰⁸ reported an early mobilization program had no impact on pressure injury rates. Three of the reported mobilization programs incorporated individualized, tolerance-based, assisted mobilization and exercise and were conducted in units with high patient acuity.¹⁰⁵⁻¹⁰⁷ The fourth intervention (delivered in a general medical unit) appeared to focus on providing individuals with encouragement to engage in mobility activities.¹⁰⁸ Evaluation of resources required to deliver an early mobility program in an 18-bed high acuity unit estimated costs of 12 nurse technician hours/day (plus staff education costs).¹⁰⁹ Early mobilization programs were associated with high patient satisfaction¹⁰⁵ and high staff adherence,¹⁰⁷ and individuals and their informal caregivers rated receiving information about positioning as a priority topic.

Implementation Considerations

- Evaluate the safety of individuals as they commence and increase mobilization and provide adequate supervision for individuals. Informal care givers and non-clinical staff can assist in reminding individuals to mobilize regularly¹¹⁰ (*Expert opinion*).
- Use appropriate mobilization techniques to avoid increased shear forces (*Expert opinion*).
- Facilitate access to appropriate mobility aids and footwear to promote safe mobility (*Expert opinion*).

Evidence Discussion

Individuals on bedrest should progress to sitting and ambulation as rapidly as they can tolerate. Ambulation schedules may help offset the clinical deterioration often seen in patients subjected to prolonged bedrest. Scheduled periods of ambulation (or supported standing when ambulation is not possible) may be viable alternatives to complete bedrest for individuals with ischial and sacral pressure injuries who cannot tolerate sitting.

One team of researchers^{107,109,110} reported on an intervention to increase mobilization in individuals in surgical intensive care. The intervention, which was facilitated by the employment of an additional health professional and delivery of education sessions, provided a protocol of increased mobilization from passive range of movement exercises, to dangling limbs over the side of the bed, sitting out of bed, standing and walking (all three times per day). Three months following the introduction of the intervention the ICU reported a significant increase in facility-acquired pressure injuries (6.1% versus 5.45% $p = 0.009$, adjusting for length of stay). The intervention was also associated with an increased length of stay in both the unit ($p < 0.001$) and the hospital ($p = 0.002$) (*Level 2*). Wood et al. (2014)¹⁰⁸ also found no impact on rates of pressure injuries for an early mobilization program delivered in a medical intensive care unit. Individuals in the intervention group participated in either active or passive range of movement, moving from bed to chair or walking (*Level 2*).

However, two other studies^{105,106} also exploring early mobilization programs in intensive care units, did demonstrate the intervention was associated with a reduction in pressure injuries. Individuals in the study conducted by Klein et al. (2015)¹⁰⁶ were inpatients in a neurological ICU who progressed through mobility milestones with assistance from a clinical technician. There were significantly fewer pressure injuries in the group exposed to this intervention (3.8% versus 1.1%, $p = 0.026$) (*Level 2*). In the study conducted by Azuh et al. (2016)¹⁰⁵ participants from a medical intensive care unit progressed through a mobility program with assistance from mobility nurses and nursing assistants, depending on their levels of tolerance. The intervention group had a lower rate of pressure injury occurrence (9.2% vs 6.1%, $p = 0.0405$) (*Level 2*).

The conflicting findings of the evidence should be viewed with consideration to confounding factors. In the early studies conducted by Dickinson et al. (2013)¹⁰⁷ participants exposed to the early mobilization intervention had higher pressure injury risk and a potential increased acuity compared to the control group. Although there was no statistically significant difference in Acute Physiology and Chronic Health Evaluation (APACHE) scores between the two cohorts, the group exposed to the intervention had a significantly higher risk of pressure injuries as determined by Braden

scores (15.66 versus 15.24, $p < 0.001$). The intervention promoted raising the head-of-bed to 30° to 45°, which may also have contributed to the increase in pressure injuries. Thus, the results of this study are hard to interpret. In all the available studies, participant exclusion criteria were limited, and little mention was given to the ability of the individuals, all of whom were in critical care environments, to participate in the mobilization programs due to hemodynamic stability. None of the studies presented a comparison of outcomes based on the level of participation in the program. Due to the study designs, data collectors were not blinded and clinicians facilitating the mobility programs had different levels of experience in program delivery.

Repositioning for Individuals with Existing Pressure Injuries

5.16: For individuals with an ischial or sacral pressure injury, evaluate the benefit of periods of bed rest in promoting healing versus the risk of new or worsening pressure injuries and the impact on lifestyle, physical and emotional health.
(Good Practice Statement)

Implementation Considerations

- Avoid seating an individual with an ischial pressure injury in a fully erect posture in chair or bed. A minimum tilt of 30° is needed to achieve a clinically significant reduction in pressure at the ischial tuberosities⁹⁴ (*Level 5*).
- For individuals with an ischial or sacral pressure injury, implement a progressive seating schedule based on the response of the pressure injury and surrounding skin, and the individual's tolerance¹⁴ (*Expert opinion*).
- Where possible, develop a management plan in conjunction with a seating professional (*Expert opinion*).
- Minimize and redistribute pressure using dynamic weight shifting (tilt and recline), noting that sitting in a non-upright position (e.g., slouching) applies greater pressure to the sacrum while pressure is applied to the ischia when the individual sits upright⁹⁴ (*Expert opinion*).
- Pressure relieving maneuvers can be used to relieve interface pressure and promote tissue perfusion and oxygenation (see *Recommendation 5.14*).
- Assess the skin after each sitting period to evaluate the regimen. Sitting times can be increased or decreased based on the improvement or deterioration of the pressure injury (*Expert opinion*).

Discussion

Ideally, ischial pressure injuries should heal in an environment in which the pressure injury is free of pressure and other mechanical stress. However, prolonged bedrest can have detrimental impact on the individual's physical, social and psychological health. Balancing needs of the individual against the need for total pressure offloading (i.e., total bed rest) creates a challenging dilemma for the individual and the professional. Potential complications associated with prolonged bed rest include, but are not limited, to:

- Muscle wasting and joint contracture
- Loss of bone density
- Deconditioning
- Respiratory complications
- Malnutrition
- Psychological harm
- Social isolation
- Financial challenges for the individual and their family.¹¹¹

In one RCT,¹¹² it was found that individuals with limited mobility with Category/Stage III and IV pressure injuries ($n = 207$), healed faster when they sat out of bed in a tilted wheelchair with a reactive pressure redistribution cushion for up to four hours daily compared with bed rest on either a foam overlay or low-air-loss bed. Healing, as measured on the Pressure Sore Status Score at four weeks, was significantly better for the seating protocol ($p < 0.0001$), and fewer individuals in the seating protocol group withdrew from the study due to pressure injury deterioration (*Level 1*). However, these results were obtained under conditions of precise seating surface prescriptions in carefully selected individuals. Similar results may not be possible in settings without an experienced seating specialist and the availability of appropriate pressure redistribution surfaces. In a small community-based study that reported an interdisciplinary pressure management protocol compared with strict bed rest for three months for healing a Category/Stage III or IV pressure injury, Chan et al. (2013)¹¹³ noted no difference in wound healing outcomes between the two management

options. The study, which was primarily an economic analysis of community-based care, also showed no significant difference in hours spent in bed or activity levels for the participants (n = 12), all of whom had SCI.

Advanced pressure redistribution support surfaces, ability to use dynamic weight shifting to offload pressure (see *Recommendations 5.12 and 5.13*) and the use of pressure relief maneuvers (see *Recommendations 5.14*) are important adjuvants to an effective supported sitting regimen. Total bed rest may be required for:

- Individuals without access to contemporary advanced support surfaces or tilt-in-space seating options
- Individuals following flap reconstruction surgery (see the guideline chapter *Pressure Injury Surgery*)
- Individuals for whom a seated position would disrupt wound healing.¹¹⁴

A progressive sitting protocol should be developed in consideration of the individual's tolerance and response of the pressure injury. A seating professional should be involved in assessing the individual, selecting an appropriate chair/wheelchair and pressure redistribution support cushion and developing an individualized supported sitting plan.

Houghton (2013)¹⁴ provides an example plan for progressive wheelchair/chair seating (see *Table 8.1*). Developed for individuals with SCI following flap reconstruction surgery to commence at approximately three weeks post-surgery, the protocol it also appropriate for adapting to the needs of individuals with a pressure injury.

Table 8.1: Example of a progressive seating protocol¹⁴

Step [#]	Activity	Duration	Seating Day
1	Sitting on the edge of bed	10 minutes twice daily	1 to 3
2	Sitting in wheelchair with a pressure redistribution cushion	5 to 10 minutes twice daily, increasing by 5 minutes each day	4 to 7
3	Sitting in wheelchair with a pressure redistribution cushion	30 minutes twice daily, increasing by 10 minutes each day to a maximum of 60 minutes twice daily	8 to 10
4	Sitting in wheelchair with a pressure redistribution cushion	Increasing by 15 minutes each day to a maximum of 4 hours twice daily	from day 11

[#]Progress to the next step only after a skin assessment confirms no new skin breakdown

Repositioning Individuals in Critical Care

In addition to the recommendations, good practice statements and implementation considerations discussed above, individuals in critical care have specific care needs. Additional preventive repositioning considerations arise from the physiological instability of critically ill individuals (see the chapter *Populations with Specific Pressure Injury Related Needs*) that increases risk of pressure injuries (See the *Risk Factors and Risk Assessment* chapter).

5.17: Reposition unstable critically ill individuals who can be repositioned using slow, gradual turns to allow time for stabilization of hemodynamic and oxygenation status.
(Good Practice Statement)

5.18: Initiate frequent small shifts in body position for unstable critically ill individuals who are too unstable to maintain a regular repositioning schedule, and to supplement regular repositioning.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Implementation Considerations

- Small shifts in body position do not replace selection of a more appropriate pressure redistribution support surface when needed, or regular repositioning (i.e., major shifts in body position) (*Expert opinion*).
- Assess tolerance to frequent, small, gradual turns on an ongoing basis, allowing the individual at least ten minutes to attain equilibrium before determining whether the position change is tolerated¹¹⁵ (*Expert opinion*).

- In critically ill individuals who cannot tolerate slow incremental turns, repositioning strategies that should be implemented include weight shifts, passive range of motion (ROM), position changes of the extremities, head rotation, heel floating and lower angle turns. The use of these interventions should be based on individual tolerance¹¹⁵ (*Expert opinion*).
- Revise the repositioning schedule in response to assessment of the individual's tolerance. Perform a trial of repositioning at least every eight hours or more frequently to determine if a regular repositioning schedule can be re-established¹¹⁵ (*Expert opinion*).
- Resume regular repositioning as soon as the individual's hemodynamic and oxygenation status stabilize (*Expert opinion*).

Evidence Summary

The current empirical evidence supporting the evidence-based recommendation consists of studies that support indirectly the benefit of supplementing regular repositioning with frequent small shifts in body weight. The studies demonstrated that small weight shifts redistribute pressure in healthy individuals and in the critically ill population.^{116,117} Improvements in sacral blood flow from small weight shifts were demonstrated in critically ill individuals.¹¹⁷ It is uncertain if the outcome is sufficient to prevent pressure injuries and the overall low volume of evidence precludes the ability to determine if this intervention will have an overall effect on pressure injury prevention or reduction.

Evidence Discussion

Oertwich et al. (1995)¹¹⁷ found that small, supplemental shifts in body weight when in the supine position significantly increased capillary perfusion measured by laser Doppler flow at both the sacrum and trochanter. Small shifts of body weight were effective in significantly reducing interface pressure at the trochanter and sacrum in both supine position and lateral oblique position (*Level 5*). A study in healthy individuals supported the finding that small shifts in posture are associated with reductions in interface pressure. In this study, statistically significant reductions of between 1.3 mmHg and 1.5 mmHg ($p < 0.05$) were attained by small weight shifts in 28 different positions.¹¹⁶ There is no direct evidence supporting this interface pressure reduction as having a clinically significant impact on pressure injury risk (*Level 5*).

Hemodynamic instability with mobilization can occur in the critical care population. The critically ill individual often possesses poor vascular tone, a dysfunctional autonomic feedback loop, and/or low cardiovascular reserve.¹¹⁸ Autonomic dysfunction may be more pronounced in individuals with diabetes. The individual's illness and care activities may lead to an imbalance of oxygen supply and demand if the requirements during mobility and/or care activities exceed supply.¹¹⁹ Finally, cardiovascular instability is often seen during position change in individuals who have experienced prolonged bedrest.

Turning the individual more slowly or in small increments that allow adequate time for stabilization of vital signs should be considered when possible.^{115,120} Care activities should be planned to allow for sufficient physiological rest to meet the oxygen demand that mobilization will place on the body. Allow the critically ill individual ten minutes to attain equilibrium before assessing tolerance to a position change.^{121,122} If manual turning is not tolerated, as evidenced by a sustained drop in blood pressure, oxygen saturation, an increase in heart rate, and/or cardiac arrhythmias, return the individual to the supine position. In addition, for individuals who cannot tolerate incremental repositioning, small weight shifts every 30 minutes can be implemented, as well as manually lifting the extremities, occiput and hips on an hourly basis unless contraindicated. Assessment of tolerance to frequent, small weight shifts should be conducted on an ongoing basis, allowing the individual at least ten minutes to attain equilibrium before determining whether the position change is tolerated.¹¹⁵ Prolonged periods in a stationary position increase the likelihood of hemodynamic instability when repositioned.

Few individuals are truly too unstable to turn. However, there may be situations that temporarily preclude turning and repositioning, which should be clearly documented in the individual's clinical record and discussed with the interprofessional team. Assess each clinical situation on an individual basis. Re-establish turning and repositioning as the individual's condition allows. For individuals who have been unable to tolerate full repositioning, Brindle et al. (2013)¹²² suggest performing a trial of repositioning at least every eight hours or more frequently to determine if a regular repositioning schedule can be re-established.

Repositioning Individuals in the Operating Room

In addition to the recommendations, good practice statements and implementation considerations discussed above, individuals in the operating room have specific care needs arising from their immobility during the operative period (see the chapter *Populations with Specific Pressure Injury Related Needs*). The increased risk of pressure injuries for individuals undergoing surgery (see the *Risk Factors and Risk Assessment* chapter) suggest that additional diligence is required in positioning in the operating room setting.

5.19: Position the individual in such a way as to reduce the risk of pressure injury development during surgery by distributing pressure over a larger body surface area and offloading bony prominences.
(Good Practice Statement)

Implementation Considerations

- Evaluate individuals who enter the perioperative setting with medical devices in situ to determine how positioning and instrumentation may impact the potential risk of pressure injury related to the devices (*Expert opinion*).
- Do not position the individual directly on a medical device unless it cannot be avoided (*Expert opinion*).
- Where possible, reposition the individual during surgery. This need not include a full body movement. This will not always be possible and will be determined by the type of surgery, the surgical position, the duration of the surgery and the individual's clinical condition (*Expert opinion*).
- Follow institutional policies and standard safety practices when positioning an individual for surgery. Select the appropriate position and positioning strategies given the type of surgery and the need to protect the individual from any injury (including but not limited to pressure injury)¹²³ (*Expert opinion*).
- Document the position in which the individual was placed during surgery (*Expert opinion*).
- Pay particular attention to pressure points that are unique to the intraoperative position when positioning the individual for surgery and when assessing the skin post operatively (e.g., forehead, nose, chin, breasts, iliac crest, genitalia, knees and toes in prone position) (*Expert opinion*).
- Use pressure redistributing support devices and padding to assist in positioning the individual (e.g., use facial pillows⁸⁶ and chest padding¹²⁴ when in prone position). See the *Repositioning and Early Mobilization* chapter of this guideline for evidence-based recommendations on devices and padding.
- Consider using prophylactic dressings to protect bony prominences. See the chapters on *Preventive Skin Care* and *Heel Pressure Injuries* for evidence-based recommendations on use of prophylactic dressings.
- Use a heel offloading device that provides support for the calves and does not place pressure on the Achilles tendon.^{125,126} See the *Heel Pressure Injuries* chapter of this guideline for evidence-based recommendations on preventing heel pressure injuries.
- Pressure mapping may be used as a visual cue to guide repositioning in the operating room (*Expert opinion*).
- When possible, position the individual in a different posture preoperatively and postoperatively than the posture adopted during surgery (*Expert opinion*).

Discussion

It is usually not possible to reduce the length of time that the skin and tissues are subjected to pressure during a surgical procedure, therefore, positioning the individual so as to distribute pressure over a larger body surface area and protecting bony prominences are key strategies to reducing pressure injury risk in the operating room. Positioning the individual in such a way as to reduce the risk of pressure injuries can be challenging given the need to ensure a stable, visible and accessible operative field for the surgical procedure. Depending on the requirements of the surgery, positioning options may be limited. However, start with the appropriate position for the type of surgery, then use padding and support devices to maximally redistribute pressure and reduce shear.

The position in which the individual is placed on the operating table is generally dictated by surgical needs. In laboratory studies, both Defloor (2000)⁶⁷ and Scott et al. (1999)¹²⁷ demonstrated that interface pressure was lowest when an individual was positioned in the supine position, compared to other surgical positions (*Level 5*). When the surgical position cannot be changed, strategies should be implemented to reduce pressure injury risk. *Table 8.2* presents pressure points associated with common surgical positions.

Many individuals will be immobilized for an extended period of time during surgery. This can cause reduced tissue perfusion at the pressure points.¹²⁸ Consideration should be given to reducing pressure on bony prominences when possible. Pressure points for particular consideration in different commonly used surgical positions are presented in *Table 8.2*. When accessible, pressure mapping systems can assist in identifying pressure points during positioning and enable interventions (e.g., adjusting the individual's position or using support surfaces, pads or pillows) to be implemented to reduce pressure.

When possible, positioning the individual in a different posture pre-operatively and post-operatively allows for rotation in pressure points to be loaded. Thus, the length of the period in which tissue is compromised is shortened, and the risk of developing a pressure injury decreases.⁶⁷ In order for health professionals working in the post anesthesia

care unit and hospital ward to monitor the individual’s skin condition and to select appropriate positions following surgery, documentation of positioning during surgery is required.

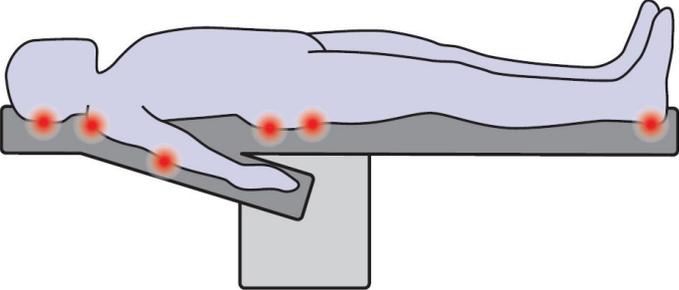
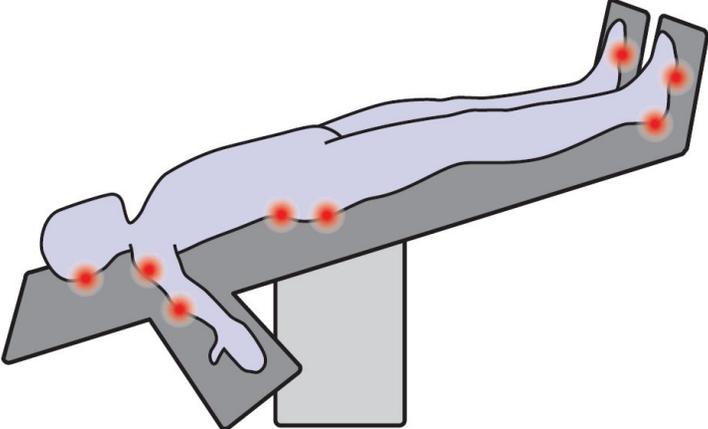
Ideally, heels should be free of all pressure in the operating room—a state sometimes called ‘floating heels’. Pressure can be relieved by elevating the lower leg and calf from the mattress with by using a heel suspension device that floats the heels or, when unavailable, placement of a pillow under the lower legs. Consequently, the pressure will instead spread to the lower legs and the heels will no longer be subjected to pressure. When elevating the heels, careful consideration should be given to the position of the individual’s lower legs. Hyperextension of the knee causes obstruction of the popliteal vein, and this could predispose an individual to deep vein thrombosis (DVT). Positioning the knees in slight flexion prevents popliteal vein compression and decreases the risk of perioperative DVT. The guideline chapter on *Heel Pressure Injuries* provides evidence-based recommendations on positioning the heels in all clinical settings and is particularly relevant to individuals being positioned for surgery.

Individuals with medical devices are at an increased risk of pressure injuries. The heavy burden of technology and equipment utilized in the operating room renders the individual particularly vulnerable to the risk for device related pressure injuries. Additionally, the individual undergoing surgery may be at risk for medical device related pressure injuries due to an increase in risk factors, including impaired sensation, moisture under the device, poor perfusion, altered tissue tolerance, and edema.¹²⁹ The guideline section on *Device Related Pressure Injuries* includes additional recommendations for reducing risk associated with external devices in a variety of clinical settings, including the operating room.

Table 8.2: Pressure points of concern in different surgical positions (with illustrations)

Note: Straps and positioning pads, pillows or wedges made of various materials (e.g., foam or gel) are often used to secure the individual, redistribute pressure and prevent nerve injury. Some, but not all of these devices are depicted in the diagrams below. Securement straps may be a source of medical device-related pressure injuries in all positions.

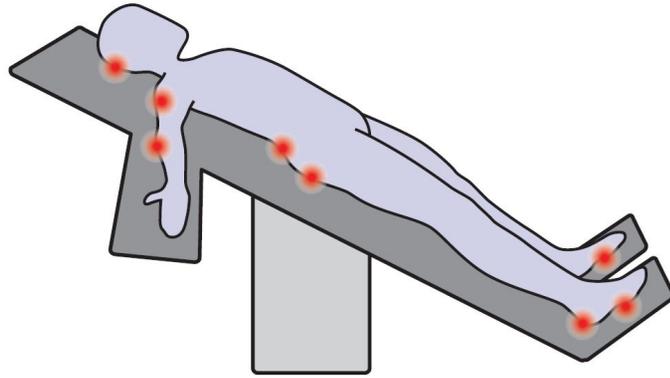
“Hidden” pressure points on the underside of the individual are marked lighter with dotted outlines

Position and pressure points of specific concern	Illustrative position noting pressure points
<p>Supine</p> <ul style="list-style-type: none"> • Occipital • Shoulder blade (scapula) • Elbows • Sacrum • Coccyx • Buttocks • Heels 	
<p>Trendelenburg</p> <p>As per supine position PLUS:</p> <ul style="list-style-type: none"> • Shoulders 	

Reverse Trendelenburg

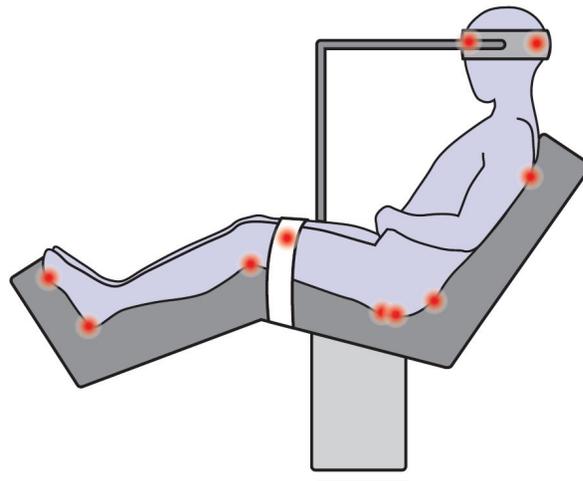
As per supine position PLUS:

- Soles of the feet

**Sitting/modified sitting**

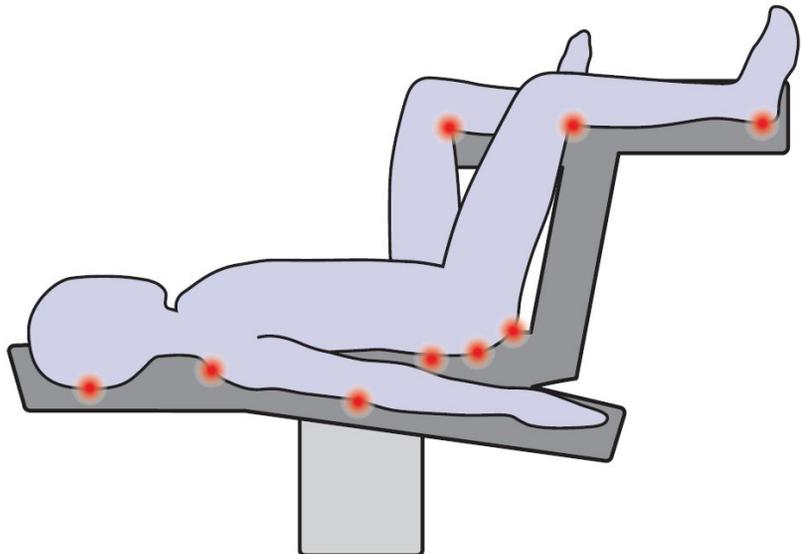
As per supine position BUT ESPECIALLY:

- Buttocks
- Ischium
- Coccyx
- Sacrum
- Back of knees
- Heels

**Lithotomy**

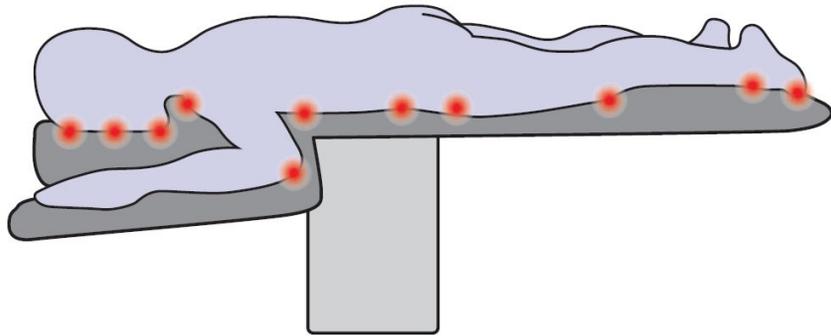
As per supine position BUT ESPECIALLY:

- Sacrum
- Coccyx
- Back of knees

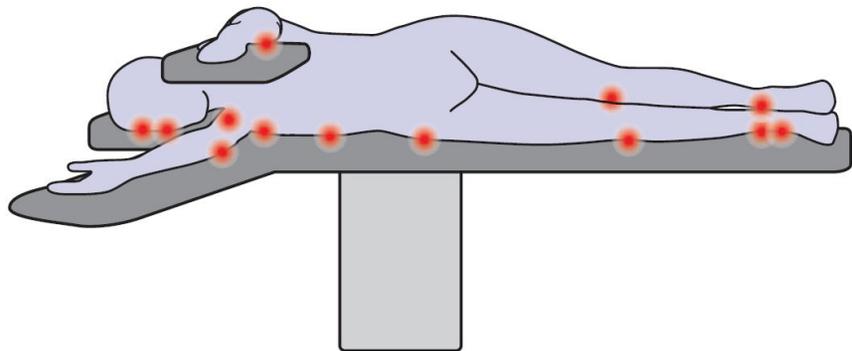


Prone

- Forehead
- Chin
- Cheeks
- Shoulder (anterior)
- Elbow
- Chest (breasts)
- Genitalia
- Anterior pelvic bones (iliac crests & ischium)
- Knees (patella)
- Dorsal feet and toes
- Nose (if positioned incorrectly)

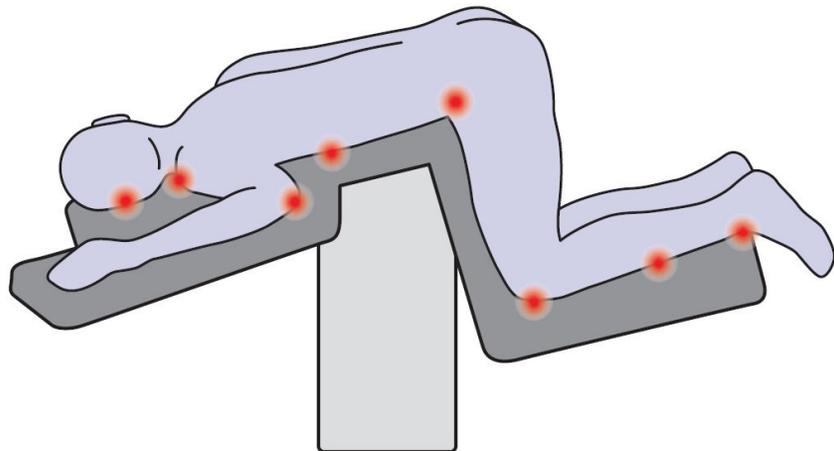
**Lateral**

- Lateral face and ear
- Elbow
- Shoulder
- Axilla
- Superior and dependent arms
- Ribs
- Hips (trochanter)
- Malleoli
- Bent lower leg
- Knees
- Ankles

**Kneeling position
(knee/chest position)**

As per prone position BUT
ESPECIALLY:

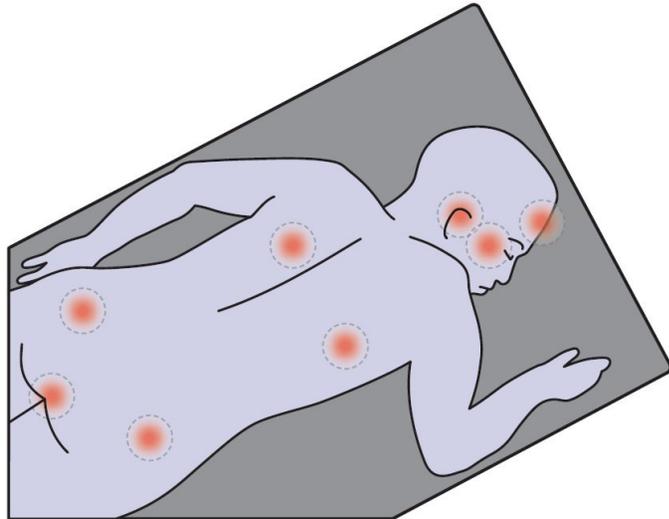
- Face and ear
- Anterior chest
- Elbows
- Anterior pelvic bones (iliac crests and ischium)
- Knees
- Anterior tibia
- Anterior ankle



Freestyle/swimming position

As per prone position BUT
ESPECIALLY:

- Lateral face and ear



References

1. Royal College of Nursing (RCN), National Institute for Health and Clinical Excellence (NICE), The management of pressure ulcers in primary and secondary care. 2005, RCN and NICE, London.
2. Defloor T, De Bacquer D, Grypdonck MHF. The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. *Int J Nurs Stud*, 2005; 42(1): 37-46.
3. Moore Z, Cowman S, Conroy RM. A randomised controlled clinical trial of repositioning, using the 30° tilt, for the prevention of pressure ulcers. *J Clin Nurs*, 2011; 20(17/18): 2633-2644.
4. Vanderwee K, Grypdonck MH, De BD, Defloor T. Effectiveness of turning with unequal time intervals on the incidence of pressure ulcer lesions. *J Adv Nurs*, 2007; 57(1): 59-68.
5. Bergstrom N, Horn SD, Rapp MP, Stern A, Barrett R, Watkiss M. Turning for Ulcer Reduction: a multisite randomized clinical trial in nursing homes. *J Am Geriatr Soc*, 2013; 61(10): 1705-1713.
6. Manzano F, Colmenero M, Pérez-Pérez AM, Roldán D, Jiménez-Quintana Mdel M, Mañas MR, Sánchez-Moya MA, Guerrero C, Moral-Marfil MÁ, Sánchez-Cantalejo E, Fernández-Mondéjar E. Comparison of two repositioning schedules for the prevention of pressure ulcers in patients on mechanical ventilation with alternating pressure air mattresses. *Intensive Care Med*, 2014; 40(11): 1679-87.
7. Rich SE, Margolis D, Shardell M, Hawkes WG, Miller RR, Amr S, Baumgarten M. Frequent manual repositioning and incidence of pressure ulcers among bed-bound elderly hip fracture patients. *Wound Repair Regen*, 2011; 19(1): 10-18.
8. Faigeles B, Howie-Esquivel J, Miaskowski C, Stanik-Hutt J, Thompson C, White C, Wild LR, Puntillo K. Predictors and Use of Nonpharmacologic Interventions for Procedural Pain Associated with Turning among Hospitalized Adults. *Pain Manag Nurs*, 2013; 14(2): 85-93.
9. McGinnis E, Nelson A, Gorecki C, Nixon J. What is different for people with MS who have pressure ulcers: A reflective study of the impact upon people's quality of life? *Journal of Tissue Viability*, 2015; 24(3): 83-90.
10. Paulden M, Bergstrom N, Horn SD, Rapp M, Stern A, Barrett R, Watkiss M, Krahn M. Turning for ulcer reduction (TURN) study: An economic analysis. *Ont Health Technol Assess Ser*, 2014; 14(12): 1-24.
11. Pechlivanoglou P, Paulden M, Pham B, Wong J, Horn SD, Krahn M. Turning high-risk individuals: An economic evaluation of repositioning frequency in long-term care. *J Am Geriatr Soc*, 2018; 66(7): 1409-1414.
12. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
13. De Laat E, Pickkers P, Schoonhoven L, Verbeek A, Feuth T, Van Achterberg T. Guideline implementation results in a decrease of pressure ulcer incidence in critically ill patients. *Crit Care Med*, 2007; 35(3): 815-820.
14. Houghton PE, Campbell KE, CPG Panel, Canadian Best Practice Guidelines for the Prevention and Management of Pressure Ulcers in People with Spinal Cord Injury. A resource handbook for clinicians. 2013, Ontario Neurotrauma Foundation: https://onf.org/wp-content/uploads/2019/04/Pressure_Ulcers_Best_Practice_Guideline_Final_web4.pdf.
15. Gammon HM, Shelton CB, Siegert C, Dawson C, Sexton E, Burmeister C, Gnam G, Siddiqui A. Self-turning for pressure injury prevention. *Wound Medicine*, 2016; 12: 15-18.
16. Chaboyer W, Mills PM, Roberts S, Latimer S. Physical activity levels and torso orientations of hospitalized patients at risk of developing a pressure injury: An observational study. *Int J Nurs Pract*, 2015; 21(1): 11-7.
17. Källman U, Engstrom M, Bergstrand S, Ek AC, Fredrikson M, Lindberg LG, Lindgren M. The effects of different lying positions on interface pressure, skin temperature, and tissue blood flow in nursing home residents. *Biol Res Nurs*, 2015; 17(2).
18. McInnes E, Chaboyer W, Allen T, Murray E, Webber L. Acute care patient mobility patterns and documented pressure injury prevention: An observational study and survey. *Wound Practice Research*, 2013; 21(3): 116-125.
19. Sonenblum SE, Sprigle SH, Martin JS. Everyday sitting behavior of full-time wheelchair users. *J Rehabil Res Dev*, 2016; 53(5): 585-598.

20. Tescher AN, Branda ME, Byrne TJ, Naessens JM. All at-risk patients are not created equal: Analysis of Braden pressure ulcer risk scores to identify specific risks. *J Wound Ostomy Continence Nurs*, 2012; 39(3): 282-291.
21. Ek AC, Unosson M, Larsson J, von Schenck H, Bjurulf P. The development and healing of pressure sores related to the nutritional state. *Clin Nutr*, 1991; 10(5): 245-250.
22. Chiari P, Forni C, Guberti M, Gazineo D, Ronzoni S, D'Alessandro F. Predictive factors for pressure ulcers in an older adult population hospitalized for hip fractures: A prognostic cohort study. *PLoS ONE [Electronic Resource]*, 2017; 12(1): e0169909.
23. Gonzalez-Mendez MI, Lima-Serrano M, Martin-Castano C, Alonso-Araujo I, Lima-Rodriguez JS. Incidence and risk factors associated with the development of pressure ulcers in an intensive care unit. *J Clin Nurs*, 2018; 27(5-6): 1028-1037.
24. Ooi W, Morris J, Brandeis G, Hossain M, Lipsitz L. Nursing home characteristics and the development of pressure sores and disruptive behaviour. *Age Ageing*, 1999; 28(1): 45-52.
25. Rose P, Cohen R, Amsel R. Development of a scale to measure the risk of skin breakdown in critically ill patients. *Am J Crit Care*, 2006; 15(3): 337-41.
26. Sayar S, Turgut S, Dogan H, Ekici A, Yurtsever S, Demirkan F, Doruk N, Tasdelen B. Incidence of pressure ulcers in intensive care unit patients at risk according to the Waterlow scale and factors influencing the development of pressure ulcers. *J Clin Nurs*, 2009; 18(5): 765-774.
27. Brandeis G, Ooi W, Hossain M, Morris J, Lipsitz L. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *Journal of the American Geriatric Society*, 1994; 42: 388-93.
28. Bergquist-Beringer S, Gajewski BJ. Outcome and assessment information set data that predict pressure ulcer development in older adult home health patients. *Adv Skin Wound Care*, 2011; 24(9): 404-414.
29. Källman U, Bergstrand S, Ek AC, Engström M, Lindgren M. Nursing staff induced repositionings and immobile patients' spontaneous movements in nursing care. *Int Wound J*, 2016; 13(6): 1168-1175.
30. Smith IL, Brown S, McGinnis E, Briggs M, Coleman S, Dealey C, Muir D, Nelson EA, Stevenson R, Stubbs N, Wilson L, Brown JM, Nixon J. Exploring the role of pain as an early predictor of category 2 pressure ulcers: A prospective cohort study. *BMJ Open*, 2017; 7(1): e013623.
31. Reed R, Hepburn K, Adelson R, Center B, McKnight P. Low serum albumin levels, confusion, and fecal incontinence: Are these risk factors for pressure ulcers in mobility-impaired hospitalized adults? *Gerontology*, 2003; 49(4): 255-59.
32. Nixon J, Cranny G, Bond S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: A cohort study. *Int J Nurs Stud*, 2007; 44(5): 655-63.
33. Demarre L, Verhaeghe S, Van Hecke A, Clays E, Grypdonck M, Beeckman D. Factors predicting the development of pressure ulcers in an at-risk population who receive standardized preventive care: Secondary analyses of a multicentre randomised controlled trial. *J Adv Nurs*, 2015; 71(2): 391-403.
34. Allman R, Goode P, Patrick M, Burst N, Bartolucci A. Pressure ulcer risk factors among hospitalized patients with activity limitation. *J Am Med Assoc*, 1995; 273(11): 865-70.
35. Hagsawa S, Barbenel JC, Kenedi RM. Influence of age on postischaemic reactive hyperaemia. *Clin Phys Physiol Meas*, 1991; 12(3): 227-237.
36. Schubert V, Schubert PA, Breit G, Intaglietta M. Analysis of arterial flowmotion in spinal cord injured and elderly subjects in an area at risk for the development of pressure sores. *Paraplegia*, 1995; 33(7): 387-97.
37. Schubert V, Fagrell B. Evaluation of the dynamic cutaneous post-ischaemic hyperaemia and thermal response in elderly subjects and in an area at risk for pressure sores. *Clin Physiol*, 1991; 11(2): 169-182.
38. Haisjackl M, Hasibeder W, Klauzner S, Altenberger H, Koller W. Diminished reactive hyperemia in the skin of critically ill patients. *Crit Care Med*, 1990; 18(8): 813-818.
39. Noble M, Voegeli D, Clough GF. A comparison of cutaneous vascular responses to transient pressure loading in smokers and nonsmokers. *J Rehabil Res Dev*, 2003; 40(3): 283-288.
40. Franzeck UK, Stengele B, Panradl U, Wahl P, Tillmanns H. Cutaneous reactive hyperemia in short-term and long-term type I diabetes- Continuous monitoring by a combined laser Doppler and transcutaneous oxygen probe. *Vasa*, 1990; 19(1): 8-15.
41. Mayrovitz HN, Sims N. Effects of support surface relief pressures on heel skin blood flow in persons with and without diabetes mellitus. *Adv Skin Wound Care*, 2004; 17(4 Pt 1): 197-201.
42. Sprigle S, Linden M, Riordan B. Characterizing reactive hyperemia via tissue reflectance spectroscopy in response to an ischemic load across gender, age, skin pigmentation and diabetes. *Med Eng Phys*, 2002; 24(10): 651-661.
43. Hagsawa S, Ferguson Pell M, Cardi M, Miller SD. Assessment of skin blood content and oxygenation in spinal cord injured subjects during reactive hyperemia. *J Rehabil Res Dev*, 1994; 31(1): 1-14.
44. Thorfinn J, Sjoberg F, Lidman D. Sitting pressure and perfusion of buttock skin in paraplegic and tetraplegic patients, and in healthy subjects: A comparative study. *Scand J Plast Reconstr Surg Hand Surg*, 2002; 36(5): 279.
45. Mawson AR, Siddiqui FH, Connolly BJ, Sharp CJ, Summer WR, Biundo JJ, Jr. Sacral transcutaneous oxygen tension levels in the spinal cord injured: risk factors for pressure ulcers? *Arch Phys Med Rehabil*, 1993; 74(7): 745-751.
46. Schubert V, Fagrell B. Postocclusive reactive hyperemia and thermal response in the skin microcirculation of subjects with spinal cord injury. *Scand J Rehabil Med*, 1991; 23(1): 33-40.
47. Pickham D, Berte N, Pihulic M, Valdez A, Mayer B, Desai M. Effect of a wearable patient sensor on care delivery for preventing pressure injuries in acutely ill adults: A pragmatic randomized clinical trial (LS-HAPI study). *Int J Nurs Stud*, 2018; 80: 12-19.
48. Yap T, Kennerly S, Simmons M, Buncher R, Miller E, Kim J, Yap W. Multidimensional team-based intervention using musical cues to reduce odds of facility-acquired pressure ulcers in long-term care: A paired randomized intervention study. *J Am Geriatr Soc*, 2013; 61: 1552-1559.
49. Pickham D, M. P, Valdez A, Mayer B, Duhon P, Larson B. Pressure injury prevention practices in the intensive care unit: Real-world data captured by a wearable patient sensor. *Wounds*, 2018; 30(8): 229-234.

50. Bales I, Padwojski A. Reaching for the moon: achieving zero pressure ulcer prevalence. *J Wound Care*, 2009; 18(4): 137-144.
51. Bales I, Duvendack T. Reaching for the moon: achieving zero pressure ulcer prevalence, an update. *J Wound Care*, 2011; 20(8): 374-377.
52. Shieh DC, Berringer CM, Pantoja R, Resurreccion J, Rainbolt JM, Hokoki A. Dramatic Reduction in Hospital-Acquired Pressure Injuries Using a Pink Paper Reminder System. *Adv Skin Wound Care*, 2018; 31(3): 118-122.
53. Baldelli P, Paciella M. Creation and implementation of a pressure ulcer prevention bundle improves patient outcomes. *Am J Med Qual*, 2008; 23(2): 136-142.
54. Pompeo M. Pressure map technology for pressure ulcer patients: Can we handle the truth? *Wounds*, 2013; 25(2): 34-40.
55. Powers J. Two Methods for Turning and Positioning and the Effect on Pressure Ulcer Development: A Comparison Cohort Study. *Journal of Wound, Ostomy, & Continence Nursing*, 2016; 43(1): 46-50.
56. Gucer PW, Gaitens J, Oliver M, McDiarmid MA. Sit-stand powered mechanical lifts in long-term care and resident quality indicators. *J Occup Environ Med*, 2013; 55(1): 36-44.
57. Webb J, Twiste M, Walton LA, Hogg P. The impact of hoist sling fabrics on interface pressure whilst sitting in healthy volunteers and wheelchair users: A comparative study. *J Tissue Viability*, 2017.
58. Gunningberg L, Sedin IM, Andersson S, Pingel R. Pressure mapping to prevent pressure ulcers in a hospital setting: A pragmatic randomised controlled trial. *Int J Nurs Stud*, 2017; 72: 53-59.
59. Behrendt R, Ghaznavi AM, Mahan M, Craft S, Siddiqui A. Continuous bedside pressure mapping and rates of hospital-associated pressure ulcers in a medical intensive care unit. *Am J Crit Care*, 2014; 23(2): 127-133.
60. Siddiqui A, Behrendt R, Lafluer M, Craft S. A continuous bedside pressure mapping system for prevention of pressure ulcer development in the medical ICU: A retrospective analysis. *Wounds*, 2013; 25(12): 333-339.
61. Gunningberg L, Carli C. Reduced pressure for fewer pressure ulcers: can real-time feedback of interface pressure optimise repositioning in bed? *Int Wound J*, 2014.
62. Scott RG, Thurman KM. Visual feedback of continuous bedside pressure mapping to optimize effective patient repositioning. *Adv Wound Care*, 2014; 3(5): 376-382.
63. Gunningberg L, Baath C, Sving E. Staff's perceptions of a pressure mapping system to prevent pressure injuries in a hospital ward: A qualitative study. *J Nurs Manag*, 2017; 29: 29.
64. Hultin L, Olsson E, Carli C, Gunningberg L. Pressure mapping in elderly care: A tool to increase pressure injury knowledge and awareness among staff. *J Wound Ostomy Continence Nurs*, 2017; 44(2): 142-147.
65. Moore Z, Cowman S, Posnett J. An economic analysis of repositioning for the prevention of pressure ulcers. *J Clin Nurs*, 2013; 22(15-16): 2354-2360.
66. Källman U, Bergstrand S, Ek AC, Engström M, Lindberg LG, Lindgren M. Different lying positions and their effects on tissue blood flow and skin temperature in older adult patients. *J Adv Nurs*, 2013; 69(1): 133-144.
67. Defloor T. The effect of position and mattress on interface pressure. *Appl Nurs Res*, 2000; 13(1): 2-11.
68. Young T. The 30 degree tilt position vs the 90 degree lateral and supine positions in reducing the incidence of non-blanching erythema in a hospital inpatient population: A randomised controlled trial. *J Tissue Viability*, 2004; 14(3): 88, 90, 92-6.
69. Gillespie BM, Chaboyer WP, McInnes E, Kent B, Whitty JA, Thalib L. Repositioning for pressure ulcer prevention in adults. *Cochrane Database Syst Rev*, 2014; 4: CD009958.
70. Oomens CW, Broek M, Hemmes B, Bader DL. How does lateral tilting affect the internal strains in the sacral region of bed ridden patients? A contribution to pressure ulcer prevention. *Clin Biomech*, 2016; 35: 7-13.
71. Schallom M, Dykeman B, Metheny N, Kirby J, Pierce J. Head-of-bed elevation and early outcomes of gastric reflux, aspiration and pressure ulcers: A feasibility study. *Am J Crit Care*, 2015; 24(1): 57-66.
72. Llauro-Serra M, Ulldemolins M, Fernandez-Ballart J, Guell-Baro R, Valentí-Trulls T, Calpe-Damians N, Piñol-Tena A, Pi-Guerrero M, Paños-Espinosa C, Sandiumenge A, Jimenez-Herrera MF, Coloma-Gómez B, Vallejo-Henao LM, Molina-Ramos S, Bordonado-Pérez L, Fortes-Del-Valle ML, Peralvo-Bernat M, Mariné-Vidal J, Alabart-Segura Y, Hidalgo-Margalef S, Portal-Porcel C, Alabart-Lorenzo X, López-Gil A, Cepero-Martí R, Martínez-Reyes L, Tapia-Vallejo C, Arévalo-Rodríguez A, Llasat-Ramón R, Sánchez-Borras E, Rodríguez-Coma E, López-Calvo J, Zahino-Ortega N, Castellano-Nofuentes S, Varo-Matito R, Valeiras-Valero A, Préstamo-Pereira B, Sanchís-López M, Hernández-López I, Martínez-Díaz T, Manzano-Montero S, López-González H, Sánchez-Sánchez MC, Esteban-López M, Treso-Geira MI, Mulet-Fumado A, Salvado-Regolf I, Sospedra-Beltran MC, Adell-Ginovart MT, Ventura-Moratalla MT, Rodríguez-Corbaton R, Sabate-Ortigueas R, Curto-Castellà MC, Guardiola-Cid R, Ventura-Rosado A, Braga-Díaz FD, Carrascal-Alcaide F, Blanco-Sánchez I, Palomino-González L, Domenech-Aguilera M, González-Engroba R, Pérez-Martín RM, Alfonso-Arias C, Álvarez-Martínez C, Vargas-López C. Related factors to semi-recumbent position compliance and pressure ulcers in patients with invasive mechanical ventilation: An observational study (CAPCRI study). *Int J Nurs Stud*, 2016; 61: 198-208.
73. Grap MJ, Munro CL, Wetzel PA, Schubert CM, Pepperl A, Burk RS, Lucas V. Backrest elevation and tissue interface pressure by anatomical location during mechanical ventilation. *Am J Crit Care*, 2016; 25(3): e56-e63.
74. Chung CHK, Lau MCH, Leung TYC, Yui KYS, Chan SHS, Chan SLS, Chan PL. Effect of head elevation on sacral and ischial tuberosities pressure in infirm patients. *Asian Journal of Gerontology and Geriatrics*, 2012; 7(2): 101-106.
75. Lippoldt J, Pernicka E, Staudinger T. Interface pressure at different degrees of backrest elevation with various types of pressure-redistribution surfaces. *Am J Crit Care*, 2014; 23(2): 119-126.
76. Pepperl AA, Rooney MB, Parker A, Burk RS, Kabban CS, Wetzel PA, Grap MJ. Effect of alertness level and backrest elevation on skin interface pressure. *Wounds*, 2014; 26(12): 334-341.
77. Crane BA, Wininger M, Kunsman M. Proxy study on minimizing risk of sacral pressure ulcers while complying with ventilator-associated pneumonia risk reduction guidelines. *Adv Skin Wound Care*, 2015; 28(12): 541-550.
78. Defloor T, Grypdonck MH. Sitting posture and prevention of pressure ulcers. *Appl Nurs Res*, 1999; 12(3): 136.

79. Bergstrom N, Allman, R., Carlson, C., et al. , *Pressure ulcers in adults: prediction and prevention. Clinical practice guideline No. 3.* 1992, Rockville, MD: Agency for Healthcare Policy and Research, AHCPR Pub. No. 920047.
80. PVA. Pressure ulcer prevention and treatment following spinal cord injury: a clinical practice guideline for health-care professionals. *J Spinal Cord Med*, 2001; 24 Suppl 1: S40.
81. Wound Ostomy and Continence Nurses Society (WOCNS), *Wound Ostomy and Continence Nurses Society. Guideline for the Prevention and Management of Pressure Ulcers.* WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: Wound Ostomy and Continence Nurses Society.
82. Grap MJ, Munro CL, Wetzel PA, Schubert CM, Pepperl A, Burk RS, Lucas V. Tissue interface pressure and skin integrity in critically ill, mechanically ventilated patients. *Intensive Crit Care Nurs*, 2017; 38: 1-9.
83. Peterson M, Schwab W, McCutcheon K, van Oostrom JH, Gravenstein N, Caruso L. Effects of elevating the head of bed on interface pressure in volunteers. *Crit Care Med*, 2008; 36(11): 3038-3042.
84. Best K, Desharnais G, Boily J, Miller W, Camp P. The effect of a trunk release maneuver on peak pressure index, trunk displacement and perceived discomfort in older adults seated in a high Fowler's position: A randomized controlled trial. *BMC Geriatric*, 2012; 12(1): 72.
85. Girard R, Baboi L, Ayzac L, Richard JC, Guerin C. The impact of patient positioning on pressure ulcers in patients with severe ARDS: Results from a multicentre randomised controlled trial on prone positioning. *Intensive Care Med*, 2014; 40(3): 397-403.
86. Grisell M, Place HM. Face tissue pressure in prone positioning: a comparison of three face pillows while in the prone position for spinal surgery. *Spine (Phila Pa 1976)*, 2008; 33(26): 2938-2941.
87. Romero CM, Cornejo RA, Galvez LR, Llanos OP, Tobar EA, Berasain M, Arellano DH, Larrondo JF, Castro J. Extended prone position ventilation in severe acute respiratory distress syndrome: A pilot feasibility study. *J Crit Care*, 2009; 24(1): 81-88.
88. Wu T, Wang ST, Lin PC, Liu CL, Chao YF. Effects of using a high-density foam pad versus a viscoelastic polymer pad on the incidence of pressure ulcer development during spinal surgery. *Biol Res Nurs*, 2011; 13(4): 419-424.
89. Fan E, Del Sorbo L, Goligher EC, Hodgson CL, Munshi L, Walkey AJ, Adhikari NKJ, Amato MBP, Branson R, Brower RG, Ferguson ND, Gajic O, Gattinoni L, Hess D, Mancebo J, Meade MO, McAuley DF, Pesenti A, Ranieri VM, Rubenfeld GD, Rubin E, Seckel M, Slutsky AS, Talmor D, Thompson BT, Wunsch H, Uleryk E, Brozek J, Brochard LJ. An Official American Thoracic Society/European Society of Intensive Care Medicine/Society of Critical Care Medicine Clinical Practice Guideline: Mechanical Ventilation in Adult Patients with Acute Respiratory Distress Syndrome. *Am J Respir Crit Care Med*, 2017; 195(9): 1253-1263.
90. Gebhardt K, Bliss MR. Preventing pressure sores in orthopaedic patients. Is prolonged chair nursing detrimental? *J Tissue Viability*, 1994; 4: 51-54.
91. Bergstrom N, Allman, R., Carlson, C., et al. , *Pressure ulcers in adults: prediction and prevention. Clinical practice guideline No. 3.* . 1992, Rockville, MD: Agency for Healthcare Policy and Research, AHCPR Pub. No. 920047.
92. Crewe RA. Problems of rubber ring nursing cushions and a clinical survey of alternative cushions for ill patients. *Care: Science and Practice*, 1987; 5(2): 9-11.
93. Jan YK, Crane BA. Wheelchair tilt-in-space and recline does not reduce sacral skin perfusion as changing from the upright to the tilted and reclined position in people with spinal cord injury. *Arch Phys Med Rehabil*, 2013; 94(6): 1207-1210.
94. Giesbrecht EM, Ethans KD, Staley D. Measuring the effect of incremental angles of wheelchair tilt on interface pressure among individuals with spinal cord injury. *Spinal Cord*, 2011; 49(7): 827-831.
95. Chen Y, Wang J, Lung CW, Yang TD, Crane BA, Jan YK. Effect of tilt and recline on ischial and coccygeal interface pressures in people with spinal cord injury. *Am J Phys Med Rehabil*, 2014; 93(12): 1019-1026.
96. Miller SK, Aberegg L, Blasiolo K, Parker M, Fulton J. A prospective assessment of sacral pressures in healthy volunteers seated upright and reclined with legs elevated in a recliner. *Ostomy Wound Manage*, 2014; 60(9): 52-59.
97. Karatas GK, Tosun A, Kanatl U. Center-of-pressure displacement during postural changes in relation to pressure ulcers in spinal cord-injured patients. *Am J Phys Med Rehabil*, 2008; 87(3): 177-182.
98. Li CT, Huang KY, Kung CF, Chen YN, Tseng YT, Tsai KH. Evaluation of the effect of different sitting assistive devices in reclining wheelchair on interface pressure. *Biomed Eng Online*, 2017; 16 (1) (no pagination)(108).
99. Loerakker S, Oomens CWJ, Manders E, T. S, D.L. B, F.P. B, Nicolay K, Strijkers GJ. Ischemia-reperfusion injury in rat skeletal muscle assessed with T-2-weighted and dynamic contrast-enhanced MRI. *Magn Reson Med*, 2011; 66(2): 528-537.
100. Makhous M, Priebe M, Bankard J, Rowles D, Zeigler M, Chen D, Lin F. Measuring tissue perfusion during pressure relief maneuvers: insights into preventing pressure ulcers. *J Spinal Cord Med*, 2007; 30(5): 497-507.
101. Sonenblum SE, Vonk TE, Janssen TW, Sprigle SH. Effects of wheelchair cushions and pressure relief maneuvers on ischial interface pressure and blood flow in people with spinal cord injury. *Arch Phys Med Rehabil*, 2014; 95(7): 1350-1357.
102. Sonenblum SE, Sprigle SH. Some people move it, move it... for pressure injury prevention. *J Spinal Cord Med*, 2016: 1-5.
103. Morita T, Yamada T, Watanabe T, Nagahori E. Lifestyle risk factors for pressure ulcers in community-based patients with spinal cord injuries in Japan. *Spinal Cord*, 2015.
104. Consortium for Spinal Cord Medicine, Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals. 2014, Consortium for Spinal Cord Medicine: https://pva-cdnendpoint.azureedge.net/prod/libraries/media/pva/library/publications/cpg_pressure-ulcer.pdf.
105. Azuh O, Gammon H, Burmeister C, Frega D, Nerenz D, DiGiovine B, Siddiqui A. Benefits of Early active mobility in the medical intensive care unit: A pilot study. *Am J Med*, 2016; 129(8): 866-871.e1.
106. Klein K, Mulkey M, Bena JF, Albert NM. Clinical and psychologic effects of early mobilization in patients treated in a neurologic ICU: A comparative study. *Crit Care Med*, 2015; 43: 865-873.
107. Dickinson S, Tschannen D, Shever LL. Can the use of an early mobility program reduce the incidence of pressure ulcers in a surgical critical care unit? *Crit Care Nurs Q*, 2013; 36(1): 127-140.

108. Wood W, Tschannen D, Trotsky A, Grunawalt J, Adams D, Chang R, Kendziora S, Diccion-MacDonald S. A mobility program for an inpatient acute care medical unit. *Am J Nurs*, 2014; 114(10): 34-42.
109. Knoblauch DJ, Bettis MA, Lundy F, Meldrum C. Financial implications of starting a mobility protocol in a surgical intensive care unit. *Crit Care Nurs Q*, 2013; 36(1): 120-126.
110. Dammeyer J, Dickinson S, Packard D, Baldwin N, Ricklemann C. Building a protocol to guide mobility in the ICU. *Crit Care Nurs Q*, 2013; 36(1): 37-49.
111. Norton L, Sibbald G. Is bed rest an effective treatment modality for pressure ulcers? *Ostomy Wound Management*, 2004; September.
112. Rosenthal MJ, Felton RM, Nastasi AE, Naliboff BD, Harker J, Navach JH. Healing of advanced pressure ulcers by a generic total contact seat: 2 randomized comparisons with low air loss bed treatments. *Arch Phys Med Rehabil*, 2003; 84(12): 1733-1742.
113. Chan BC, Nanwa N, Mittmann N, Bryant D, Coyte PC, Houghton PE. The average cost of pressure ulcer management in a community dwelling spinal cord injury population. *Int Wound J*, 2013; 10(4).
114. Houghton PE, Campbell KE, CPG Panel, Canadian Best Practice Guidelines for the Prevention and Management of Pressure Ulcers in People with Spinal Cord Injury. A resource handbook for clinicians. 2013, Ontario Neurotrauma Foundation: <http://www.onf.org>.
115. Brindle CT, Malhotra R, O'Rourke S, Currie L, Chadwick D, Falls P, Adams C, Swenson J, Tuason D, Watson S, Creehan S. Turning and repositioning the critically ill patient with hemodynamic instability: A literature review and consensus recommendations. *Journal of Wound, Ostomy and Continence Nursing*, 2013; 40(3): 254-67.
116. Tsuchiya S, Sato A, Azuma E, Urushidani H, Osawa M, Kadoya K, Takamura M, Nunomi M, Mitsuoka A, Nishizawa Yokono T, Sugama J. The effectiveness of small changes for pressure redistribution; Using the air mattress for small changes. *Journal of Tissue Viability*, 2016; 25(2): 135-142.
117. Oertwich PA, Kindschuh AM. The effects of small shifts in body weight on blood flow and interface pressure. *Res Nurs Health*, 1995; 18(6): 481-8.
118. Doering L. The effect of positioning on hemodynamics and gas exchange in the critically ill: a review. *American Journal of Critical Care*, 1993; 2(3): 208-16.
119. Shacknell E, Gillespie M. The oxygen supply and demand framework: A tool to support integrative learning. *Dynamics*, 2009; 20(4): 15-9.
120. Almirall S, Leiva R, Gabasa P. Apache III Score: A prognostic factor in pressure ulcer development in an intensive care unit. *Enferm Intensiva*, 2009; 20(3): 95-103.
121. Vollman KM. Understanding critically ill patients hemodynamic response to mobilization: using the evidence to make it safe and feasible. *Crit Care Nurs Q*, 2013; 36(1): 17-7.
122. Brindle CT, Malhotra R, O'Rourke S, Currie L, Chadwick D, Falls P, Adams C, Swenson J, Tuason D, Watson S, Creehan S. Turning and repositioning the critically ill patient with hemodynamic instability: A literature review and consensus recommendations. *J Wound Ostomy Cont Nurs*, 2013; 40(3): 254-267.
123. MacDonald J, Washington S. Positioning the surgical patient. *Anaesthesia and Intensive Care Medicine*, 2012; 13(11): 528-532.
124. Wu T, Wang ST, Lin PC, Liu CL, Chao YF. Effects of using a high-density foam pad versus a viscoelastic polymer pad on the incidence of pressure ulcer development during spinal surgery. *Biological Research For Nursing*, 2011; 13(4): 419-424.
125. Malkoun M, Huber J, Huber D. A comparative assessment of interface pressures generated by four surgical theatre heel pressure ulcer prophylactics. *Int Wound J*, 2012; 9(3): 259-263.
126. Donnelly J. A randomised controlled trial comparing the Heelift Suspension Boot with standard care in the prevention of pressure ulcers on the heels of older people with fractured hips. Thesis dissertation. 2006, University of Belfast. .
127. Scott EM, Baker EA, Kelly PJ, Stoddard EJ, Leaper DJ. Measurement of interface pressures in the evaluation of operating theatre mattresses. *J Wound Care*, 1999; 8(9): 437-441.
128. Schoonhoven L, Defloor T, van der Tweel I, Buskens E, Grypdonck MH. Risk indicators for pressure ulcers during surgery. *Appl Nurs Res*, 2002; 15(3): 163-173.
129. Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Call E, Clark M. Use of wound dressings to enhance prevention of pressure ulcers caused by medical devices. *Int Wound J*, 2015; 12(3): 322-327.

HEEL PRESSURE INJURIES

Introduction

The heel is one of the two most common anatomical sites for pressure injuries. In a European survey on pressure injury prevalence (n = 5,947),¹ almost 80% of all Category/Stage IV pressure injuries were found at the sacrum and heels (39.9% and 38.5%, respectively). The most severe pressure injury was typically found at the sacrum (44.8%) or the heels (24.2%).¹ In a national pressure injury prevalence study conducted in 2014 in France (n = 21,538),² the heel and the sacrum were the two most common anatomical sites affected by pressure injuries in intensive acute care. A study conducted in Spain from 2012 to 2015 reported the heel was the most common anatomical location for pressure injuries in children and neonates.³ The heel is not only a frequent anatomical location for pressure injuries, but it experiences some of the most severe pressure injuries.

Older people, individuals in critical care, children and neonates are at particularly high risk for heel pressure injuries. The prevalence of heel pressure injuries among individuals in acute care has been reported to be between 21% and 46%.⁴ The highest rates of heel pressure injuries were found in rehabilitation units in a national prevalence study conducted in different clinical settings in France.² In a prospective study conducted in two geriatric rehabilitation centers, the prevalence of heel pressure injuries was 12%,⁵ while heel pressure injury prevalence was as high as 28.6% in children aged between 7 and 12 years attending primary health care consultations.³

The reduction of pressure and shear at the heel is an important point of interest in clinical practice. The posterior prominence of the heel sustains intense pressure, even when a pressure redistribution surface is used. Because the heel is covered with a small volume of subcutaneous tissue, mechanical loads are transmitted directly angular to the bone. Finite element modeling suggests that the shape of the individual's calcanei influences the strain on muscles and tissue at the heel.^{6,7} Given the small surface area of the heel, it is challenging to redistribute load from the heel.

The recommendations and good practice statements in this chapter present evidence specific to preventing and treating heel pressure injuries. The recommendations discuss heel-specific skin and tissue assessment, positioning the heels, and prophylactic dressings applied to the heels. Limited evidence on skin care practice specific to the heels was available. However, the recommendations and studies^{8,9} on skin care presented in the guideline chapter *Preventive Skin Care* are particularly relevant to preventive skin care for heels and should be referred to when developing a care plan to prevent heel pressure injuries.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What factors put individuals at risk for heel pressure injury development?
- What are accurate and effective methods for assessing heel skin and tissue?
- What are effective local management strategies (e.g., skin care, prophylactic dressings) in preventing heel pressure injuries?
- What heel repositioning interventions are effective in preventing heel pressure injuries?
- What support surfaces and devices are effective in preventing heel pressure injuries?
- What are effective strategies for treating heel pressure injuries?
- What factors affect healing of heel pressure injuries?

Assessing the Heels

The heel is considered a vulnerable area to pressure damage as compared to other areas of the body due to factors such as the heel anatomy, disease burden, comorbid conditions, and the aging process.¹⁰ Due to this vulnerability, it is prudent to regularly assess the heel for pressure damage, especially in individuals who are medically complex.^{11,12} Heel assessment should include the physical assessment of the heel as well as noting the individual's past clinical history, any previous heel pressure injuries, and current physical and medical status. Additionally, risk factors that can contribute to the development of heel pressure injuries or delay their healing (e.g., peripheral vascular disease) should be noted and addressed. The guideline chapter *Risk Factors and Risk Assessment* identifies key risk factors to consider in a risk assessment. The guideline chapter *Skin and Tissue Assessment* contains comprehensive discussion and recommendation on assessing the skin.

6.1: Assess the vascular/perfusion status of the lower limbs, heels and feet when performing a skin and tissue assessment, and as part of a risk assessment.
(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

Evidence from one moderate quality Level 3 study¹³ indicated that having peripheral arterial disease increases the risk of heel pressure injuries, possibly due to decreased blood flow to the heel. Evidence from a low quality Level 3 prognostic study¹⁴ showed that heel pressure injuries were less likely to heal when the individual had peripheral vascular disease.

Implementation Considerations

- Inspect the skin of the heels regularly, even if a prophylactic dressing is in situ (*Expert opinion*).
- Health professionals undertaking comprehensive vascular assessment should be trained in using appropriate assessment techniques and equipment (*Expert opinion*).
- When assessing the lower limbs/heels/feet as part of an overall skin assessment, the following parameters should be included, with a goal of evaluating vascular supply to the heel and differentiating between arterial and venous disease:
 - Skin temperature
 - Presence or absence of peripheral pulses that perfuse the heel (particularly posterior tibial artery that supplies the heel)
 - Skin color (e.g., pale, hemosiderin staining, etc.)
 - Quality and appearance of skin (e.g., shiny, presence or absence of hair, etc.)
 - Sensation to touch or individual reports of diminished sensation (*Expert opinion*).
- Pending the availability of equipment, the following assessments may be considered:
 - Ankle brachial pressure index (ABPI) or toe brachial pressure index (TPBI) as a measure of foot perfusion¹⁵
 - Monofilament testing as a measure of sensation (*Expert opinion*).
- In addition to physical inspection of the heel, consider an individual's clinical condition, medical history and risk factors for heel pressure injury development, including vascular disease, diabetes mellitus, and having previously experienced heel pressure injuries (*Expert opinion*).
- Consider referring individuals with a suspected or known compromised vascular status to a vascular specialist as indicated (*Expert opinion*).

Evidence Discussion

Due to vascular disease and tissue thinning associated with aging, and the presence of avascular fat in the heel, the heel is at risk of pressure injuries. As noted in the guideline chapter on *Risk Factors and Risk Assessment*, alterations to perfusion and circulation (including diabetes mellitus) increases the risk of pressure injuries. Vascular status is particularly significant to the prevention and treatment of pressure injuries of the heel,¹⁰ and should be included when conducting a comprehensive assessment of the heels.

One study¹⁶ indicated that having peripheral arterial disease increases the risk of heel pressure injuries, possibly due to decreased blood flow to the heel. In individuals in a community hospital (n = 30), peripheral arterial disease was a significant factor for heel pressure injuries in a multivariable analysis (odds ratio [OR] 11, 95% confidence interval [CI] 1.99 to 60.57)¹⁶ (*Level 3*).

In individuals in acute care who had hospital and community acquired pressure injuries (n = 337),¹⁰ diabetes mellitus, vascular disease, immobility, and an admission Braden Scale score of 18 or less were all significant risk factors for heel pressure injuries in a univariate analysis (*Level 3*).

Additionally, a prognostic study¹⁴ showed that heel pressure injuries were less likely to heal when the individual had peripheral vascular disease. In individuals with heel pressure injuries (n = 140 with 183 pressure injuries), presence of peripheral arterial disease was a significant factor in a multivariable analysis (hazard ratio [HR] = 0.40, 95% CI 0.20 to 0.81, p = 0.010)¹⁴ (*Level 3*).

Along with other comorbid conditions, research has emphasized how compromised blood flow or disease to the heel is a risk factor for heel pressure injury occurrence and/or healing. Therefore, it is prudent to consider vascular status as part of the heel assessment to determine if vascular compromise or disease may be present. A comprehensive heel assessment will provide a more informed prevention and/or treatment plan that is tailored to the individual.

Health professionals performing vascular assessments require education and training in assessment skills and using equipment or should make a referral to an experienced health professional. Commonly used non-invasive investigations for vascular assessment include (but are not limited to) ABPI, TBPI and continuous wave Doppler ultrasound. The evidence for use of these examinations specifically to assess heel vascularization in relation to pressure injury prevention and healing is limited. However, there is a strong body of evidence for use of these investigations to assess lower limb vascularization in other contexts.

The ABPI is commonly used to assess large vessel peripheral vascular disease; however, its use in assessing small vessel disease of the foot is considered to be limited.¹⁵ Additionally, caution is required when interpreting outcomes in individuals with diseases such as diabetes mellitus.^{15,17} With respect to assessment in relation to heel pressure injuries, a retrospective chart review completed for 83 participants with 92 heel pressure injuries¹⁸ found that ABPI may not be an accurate and reliable measure of arterial flow to the heel. Nearly 47% of cases had non-compressible vessels, and in 50% of cases with compressible vessels, the ABPI did not measure an artery that directly reflected heel perfusion (i.e., the posterior tibial artery) (*Level 4*). Therefore, TBPI or Doppler ultrasound may provide a more accurate vascular assessment of the foot.¹⁵

Positioning to Prevent and Treat Heel Pressure Injuries

6.2: For individuals at risk of heel pressure injuries and/or with Category/Stage I or II pressure injuries, elevate the heels using a specifically designed heel suspension device or a pillow/ foam cushion. Offload the heel completely in such a way as to distribute the weight of the leg along the calf without placing pressure on the Achilles tendon and the popliteal vein.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Evidence from moderate^{19,20} and low²¹ quality Level 1 studies demonstrates that elevating the heels reduces the risk of pressure injuries. Incidence of pressure injuries were lower using both a regular foam cushion²¹ and using a foam heel suspension boot^{19,20} than when heels were not elevated. As well as being more effective than normal care with no heel elevation,^{19,20} a foam heel suspension boot was also shown to be more effective in reducing pressure injuries in one high quality Level 1 study,²² and more effective for improving condition of reddened heels in a low quality Level 2 study.²³ One low quality Level 4 study showed reductions in any heel pressure injuries of 43.8% and a 67% reduction in Category/Stage III or IV heel pressure injuries.²⁴ Two low quality Level 4 studies^{25,26} provided evidence supporting air filled heel elevation boots²⁵ and low friction fabric heel elevation boots.²⁶

Implementation Considerations

- Ensure the heels are free from the surface of the bed (*Expert opinion*).
- When selecting a heel suspension device consider:
 - The individual's clinical condition, including factors that may increase movement of the legs (e.g., agitation and muscles spasms)
 - Skin integrity and presence of edema
 - Anatomical appearance/alignment of the hip, foot and lower leg
 - Plan of care
 - The individual's tolerance of the device
 - The manufacturer's guidelines. (*Expert opinion*).
- Knee should be in slight (5° to 10°) flexion (*Level 5*).
- Avoid areas of high pressure, especially under the Achilles tendon. Distribute pressure along the full length of the calves (*Expert opinion*).
- Consider selecting a device with a positioning block if the individual's foot is not aligned in optimal positioning (e.g., if the foot falls into lateral or external rotation) (*Expert opinion*).

- Apply heel suspension devices according to the manufacturer's instructions (*Expert opinion*).
- Remove the heel suspension device periodically (at least twice/day) to assess skin integrity and perfusion status. Remove the positioning device more frequently if edema or fluid shifting is present (*Expert opinion*).

Evidence Discussion

Heel elevation

Pressure can be relieved by elevating the lower leg and calf from the mattress. Ideally, heels should be free of all pressure — a state sometimes called 'floating heels'. Huber et al. (2008)²⁷ documented significant increases in tissue blood flow to the heels measured using laser Doppler when the heels of non-hospitalized volunteers with and without peripheral vascular disease were elevated (*Level 5*). Three studies¹⁹⁻²¹ provide clinical evidence that elevation of the heels is more effective at preventing pressure injuries than a care regimen that does not include heel elevation.

A multi-center randomized controlled trial (RCT) conducted by Bååth et al. (2016)¹⁹ compared the effect of elevation of the heel with a foam heel suspension boot applied in the ambulance (intervention group) with normal care (control group) on the incidence of heel pressure injuries at time of discharge from hospital. A total of 405 older adults (≥ 70 years) being transferred to hospital via ambulance were recruited. The assigned heel management protocol was continued on the ward until discharge. The mean hospital length of stay was 7.9 days for the intervention group and 10.4 days for the control group. Analysis of 183 participants at study conclusion indicated a significantly lower incidence of pressure injuries in the intervention group compared to the control group (14.6% versus 30%, $p = 0.017$). Inter-rater reliability between assessors was not determined, although all the assessors received standard education.¹⁹ The study was significantly powered and the results support the elevation of heels for the prevention of pressure injuries (*Level 1*).

Donnelly et al. (2011)²⁰ conducted an RCT comparing complete offloading of the heel using a commercial heel suspension device to standard care that did not include heel offloading. Older adults who were admitted to a trauma unit with hip fractures occurring within the previous 48 hours and who were pressure injury free ($n = 239$) were recruited. The primary outcome of interest was the occurrence of a new Category/Stage I or greater pressure injury of the heels (or other sites). The intervention group receiving heel elevation developed no pressure injuries on the ankles, feet or heels. However, the control group with no heel elevation experienced 29 foot/heel pressure injuries ($p < 0.001$). Kaplan-Meier survival curves indicated individuals in the control group were more likely to suffer pressure damage at all points in time (log rank, $p = 0.001$). Sensitivity analysis showed that when individuals lost to follow-up were assigned to the pressure injury outcome, the intervention group was still less likely to develop a pressure injury than those in the control group ($p = 0.0001$). The hazard analysis indicated that when considering the effect of multiple clinical and pathological factors that might be specific risk factors, participants randomized to the treatment group were five times less likely to develop pressure damage (HR = 0.21, 95% CI 0.008 to 0.54) than the control group (HR = 1.00). There are some limitations to this study given the frequent protocol violations in relation to support surface upgrades by the nursing staff (*Level 1*).

In an RCT, Cadue et al. (2008)²¹ evaluated the efficacy of placing a foam cushion under the legs to 'float' the heels free from the bed surface. Seventy individuals in intensive care were recruited, with half receiving heel elevation and the remainder receiving no intervention at the heels. Fewer heel pressure injuries developed among the group receiving heel elevation (8.5% compared with 54.2% in the control group). There was also a longer heel pressure injury free time in the heel elevation group (time to development of heel pressure injury was 5.6 days in the heel elevation group and 2.8 days in the control group). Although small, this study suggests the value of removing all pressure from the heels (*Level 1*). Interpretation is constrained by its lack of a formal power calculation and uncertain subject selection criteria.

Two low quality studies,^{24,26} both conducted in the UK hospital setting, reported cost analyses suggesting that elevating the heels with a specifically designed device is associated with cost savings over 12 months²⁶ and longer.²⁴ The studies^{24,26} reported actual or projected cost savings to facilities when heel suspension boots were included in a heel pressure prevention regimen. However, cost implications and potential savings are likely to be highly specific to geographic location, clinical setting and the level of pressure injury risk of the population, as suggested by the variable cost savings reported between facilities in the studies.^{24,26}

Methods for elevating the heels

Elevation of the heels such that they are completely free of the support surface can be achieved using a pillow or foam cushion under the lower legs, or by using a heel suspension device that floats the heel. Pressure will be distributed over the larger surface area of the lower legs and the heels, will no longer be subjected to pressure.

The most accessible method of floating the heels is using a pillow or foam cushion to elevate the lower legs and completely free the heels from the mattress surface. Pillows/foam cushions placed under the full length of the calves to elevate heels are appropriate for short-term use in alert and cooperative individuals who are able to maintain their legs in the appropriate positioning. Pillows or foam cushions used for heel elevation should extend the length of the calf to avoid areas of high pressure, particularly under the Achilles tendon. In the RCT by Cadue et al. (2008) reported above, use of a foam cushion to elevate the heels was associated with significantly fewer facility-acquired heel pressure injuries than when heels were not elevated (8.5% versus 54.2%). However, the participants were in a critical care setting and had high rates of sedation and mechanical ventilation. For individuals who are more mobile or who have agitation or other clinical conditions that increase movement of the lower legs, and for individuals with dementia, positioning with a pillow or foam cushion may be inadequate. In this case, and for individuals with Category/Stage III or greater heel pressure injuries (see *Good Practice Statement 6.3* below), a heel suspension device may provide more reliable heel elevation support.

Heel suspension devices are preferable for long term heel elevation, or for individuals who are not likely to keep their legs on the pillows/foam cushions. Heel suspension devices vary in design and material (e.g., foam heel suspension boots, air inflated heel suspension boots etc.). An assessment of the individual is required to select the most appropriate heel suspension device. Consideration should be given to skin integrity, presence of edema, anatomical appearance/alignment of the foot and lower leg (e.g., deformities or contractures), mobility status, comfort, tolerance of the device and the manufacturer's guidelines.

Most studies exploring effectiveness of heel suspension devices for preventing or treating pressure injuries report on different designs of foam heel suspension boots. The RCT by Bååth et al. (2016)¹⁹ that is discussed above demonstrated significantly fewer pressure injuries with use of a foam heel suspension boot when compared to normal care (14.6% versus 30%, $p = 0.017$) (*Level 1*). The RCT by Donnelly et al. (2011)²⁰ also reported above compared the effectiveness of a foam heel suspension boot plus pressure redistributing support surface (intervention group) to standard care that included a pressure redistributing surface (control group). There were significantly fewer pressure injuries on the ankles, feet or heels in the intervention group compared to the control group (0% versus 24%, $p < 0.01$)²⁰ (*Level 1*). An RCT conducted by Meyers et al. (2017)²² also demonstrated that heel elevation with a foam heel suspension boot was associated with significantly fewer pressure injuries than with regular pillows in critically ill individuals (0% versus 41%, $p < 0.001$)²² (*Level 1*).

Cheneworth et al. (1994)²³ conducted a quasi-experiment comparing the outcomes of Category/Stage I heel pressure injuries between a foot wrap consisting of gauze pads and undefined dressings wrapped around the heel and a laminated foam heel suspension boot. Healing and stabilization of the heel pressure injury was seen in 13 of 14 individuals wearing the heel suspension device, while pressure injuries deteriorated or remained the same in the foot wrap group²³ (*Level 2*).

Bales (2012)²⁸ demonstrated that a foam heel suspension boot was associated with significantly fewer pressure injuries than use of intravenous bags for elevating the heels (0 versus 40%, $p = 0.006$) (*Level 2*).

Meyers et al. (2010)²⁹ reported a foam heel suspension boot was associated with no new heel pressure injuries occurring and a 55% reduction in heel pressure injuries (described as "abnormal heels" in the study) between admission and discharge (*Level 4*). A retrospective analysis conducted by Rajpaul et al. (2016)²⁴ investigated the incidence of pressure injuries in high-risk individuals in two hospitals. Heel elevation with a foam heel suspension boot was associated with a 43% reduction in heel pressure injuries in one hospital and a 67% reduction in Category/Stage III or IV heel pressure injuries in the second hospital²⁴ (*Level 4*).

Use of air filled²⁵ and low friction fabric²⁶ heel suspension boots has also been explored in small observational studies. One observational study ($n = 17$)²⁵ suggested that using a four celled, air inflation heel suspension boot can prevent pressure injuries in individuals receiving rehabilitation care (*Level 4*). However, the study was short (two weeks) and provided no comparison between either not elevating heels, or using a different type of heel suspension device, limiting the inferences that can be made from the results. A second observational study²⁶ conducted with hospitalized individuals reported a reduction in the rate of avoidable heel pressure injuries observed in the facility from 32% to 27.3% over four years after the introduction of a low friction fabric suspension boot²⁶ (*Level 4*).

A variety of heel suspension devices are used in the operating room; however, evidence was limited to a cross-over quasi experiment³⁰ comparing the use of a prototype device to a viscous gel heel block and various support surfaces. Heel offloading devices (both the prototype device and heel blocks) were associated with significant reductions in interface pressure at the heel than comparator support surfaces³⁰ (*Level 5*).

Some support surfaces include technologies that allow for reduction of pressure to the heels. For example, some alternating pressure air mattresses incorporate features that enable reduction of the support surface pressure at the heels.³¹ Research is required to evaluate the effectiveness of these support surfaces in preventing heel pressure injuries.

Heel elevation positioning

Flex the knee slightly to avoid popliteal vein compression and increased risk of deep vein thrombosis (DVT). There is indirect evidence that hyperextension of the knee may cause obstruction of the popliteal vein, and this could predispose an individual to DVT. Huber et al. (2009)³² studied the popliteal veins of 50 individuals under general anesthesia using heel elevators. Using duplex ultrasonography to examine the incidence of popliteal vein compression when the knees were flexed and extended, they found a significant reduction in popliteal vein diameter in extension compared with the diameter in flexion ($p < 0.001$).³²

Heel suspension devices should be applied so as to avoid creating areas of increased pressure under the device. Ensure that the heel suspension device is not too tight and does not create additional pressure damage, particularly in individuals with deformities, contractures or other factors influencing positioning.

Evaluating heel elevation

The skin under a heel suspension device should be checked routinely for device-related pressure damage. Check the skin more frequently and adjust the device in individuals with, or likely to develop, lower extremity edema, individuals with peripheral vascular disease, individuals with neuropathy/reduced sensation, and individuals with reduced ability to communicate points of pressure or pain.

Comfort for the individual is an important factor when selecting a heel suspension device. Bååth et al. (2016)¹⁹ found that a foam heel suspension boot was associated with lower ratings of pain compared to standard care that did not include heel elevation. However, in the same trial individuals described a foam suspension boot as too warm, sweaty, itchy and uncomfortable in side-lying positions.¹⁹ Participants in other studies reported similar concerns^{23,25} (*Levels 1 and 4*). This highlights the importance of evaluating the individual's tolerance to the device and selecting a different type of device if necessary.

6.3: For individuals with a Category/Stage III or greater heel pressure injury, elevate the heels using a device specifically designed for heel suspension, offloading the heel completely in such a way as to distribute the weight of the leg along the calf without placing pressure on the Achilles tendon and the popliteal vein. (Good Practice Statement)

Implementation Considerations

- Due to the time required for healing full thickness pressure injuries, a device that completely offloads the pressure injury and prevents foot drop is preferred (*Expert opinion*).

Discussion

Once a pressure injury develops, pressure relief on the heel is needed to promote perfusion and healing. Pressure on Category/Stage III, IV, and unstageable heel pressure injuries and deep tissue pressure injuries of the heel should be completely offloaded as much as possible. Heel suspension devices are preferable for long-term use, or for individuals who are not likely to keep their legs on the pillows; elevation of the heel on a pillow is usually inadequate. Due to the time required for healing deeper pressure injuries, a device that completely offloads the heel and prevents foot drop is preferred.

Prophylactic Dressings for the Heels

Prophylactic dressings are dressings that are applied to intact skin over a pressure point with the aim of preventing a pressure injury. Use of prophylactic dressings should be an adjunct to, rather than a replacement for heel elevation. Different types of prophylactic dressings are available, including those designed specifically for application to the heel. In the laboratory setting, prophylactic dressings have been found to reduce the forces of pressure, friction and shear through multiple layer construction;³³⁻³⁵ protect fragile skin from shear with specially designed adhesives;³³ and influence microclimate.³⁶ A full discussion of specific characteristics of prophylactic dressings and the ways they protect pressure points is presented in the guideline chapter on *Preventive Skin Care*.

6.4: Use a prophylactic dressing as an adjunct to heel offloading and other strategies to prevent heel pressure injuries. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

The recommendation is supported by moderate³⁷ and low³⁸ quality Level 1 studies, and high³⁹ and moderate⁴⁰ quality Level 3 studies providing evidence on two different foam dressings designed for application to the heels and one low quality Level 2 study⁴¹ providing evidence for a transparent polyurethane film. The different types of prophylactic foam dressings, a multi-layered soft silicone foam dressing^{37,39} and a polyurethane foam dressing^{38,40} were both associated with statistically significantly fewer heel pressure injuries than standard care that included either no prophylactic dressing^{37,39,40} or gauze padding and bandage.³⁸ In both the Level 1 studies, the heel pressure injury incidence rate was around 3% when using either type of prophylactic foam dressing,^{37,38} while the rate of pressure injuries using the polyurethane film was around 6%.⁴¹

Implementation Considerations

- Continue to implement other measures (e.g., heel offloading and regular repositioning) to prevent heel pressure injuries when using a prophylactic dressing (*Expert opinion*).
- When selecting a prophylactic dressing consider:
 - Appropriateness of the size and design of the dressing to apply to a heel
 - Ability to manage the microclimate
 - Ease of application and removal
 - Ability to maintain the dressing on the heel
 - Ability to regularly assess the skin under the dressing
 - The individual's preferences, comfort and any allergies
 - Coefficient of friction at the skin-dressing interface
 - Cost and accessibility of devices. (*Expert opinion*).
- Continue to assess the skin under a prophylactic dressing at least daily to evaluate the effectiveness of the preventive care regimen. Many dressings have features that facilitate regular skin assessment (e.g. transparency,⁴¹ silicone borders,^{37,39} non-adhesive edges,^{38,40} etc.) (*Levels 1 and 3*).
- Replace the prophylactic dressing if it becomes dislodged, loosened or excessively moist,^{37,39} if the dressing or skin underneath become soiled, and according to the manufacturer's instructions (*Levels 1 and 3*).
- A tubular bandage can assist to keep the prophylactic dressing in place³⁷⁻³⁹ (*Levels 1 and 3*).
- For individuals at high risk of pressure injuries, application of a prophylactic dressing should be initiated as early as possible in the care pathway when feasible (e.g., applied in the ambulance or emergency room)³⁷ (*Level 1*).
- Follow manufacturers' instructions for use (*Expert opinion*).

Evidence Discussion

Prophylactic dressings differ in their qualities; therefore, it is important to select a dressing that is appropriate to the individual and the clinical use. Evidence supporting the application of a prophylactic dressing to the heels of individuals at risk of developing heel pressure injuries includes reports on multi-layered soft silicone foam dressings,^{37,39,42} polyurethane foam hydrocellular dressings,^{38,40} polyurethane film⁴¹ and heel silicone pads.⁴³ The available evidence provides comparison of different heel prophylactic dressings to each other, or to a standard care regimen that explicitly included heel elevation. There are no studies meeting inclusion criteria comparing the relative effects of applying a heel prophylactic dressing when the heels are elevated. Prophylactic dressings should be considered an adjunct to positioning and elevation.

There is indirect evidence indicating that applying a prophylactic dressing reduces the interface pressure at the heel. Reduction in interface pressure has been demonstrated with a silicone foam border prophylactic dressing in an observational study⁴² conducted with healthy volunteers (n = 50) that showed effectiveness in reducing interface pressure within four minutes, which was significant compared to no dressing in situ⁴² (*Level 5*). Other studies demonstrate that the reduction in interface pressure translates to a reduced risk of pressure injuries.

Foam prophylactic dressings

Effectiveness of the same prophylactic dressing in reducing heel pressure injury incidence has been demonstrated in critical care settings.^{37,39} Santamaria et al. (2013)³⁷ conducted an RCT in which adults admitted to an intensive care unit (ICU) were randomized in the emergency department to receive either a multi-layered soft silicone foam dressing applied to the heels (and sacrum) or to a control group receiving standard pressure injury prevention (not described). A tubular bandage was also used to secure and protect the heel prophylactic dressing. After transfer to the ICU, skin assessments were performed every two to four hours. The prophylactic heel dressing was changed every three days

or earlier if dislodged or soiled. There was a significant reduction in overall (i.e., heels and sacrum) pressure injury incidence (4.3% versus 17.8%, $p = 0.002$) and in heel pressure injury incidence (3.1% vs 12.5%, $p = 0.002$) associated with the prophylactic dressing. The study and analysis were non-blinded and the Category/Stage of pressure injuries that occurred was not reported (*Level 1*). Additionally, in an historical control cohort study of 302 individuals in trauma and critical care Santamaria et al. (2015)³⁹ found lower pressure injury incidence among individuals in the intervention group ($n = 150$) receiving the same type of prophylactic heel dressing held in place with a tubular bandage, as compared to the control group receiving standard prevention interventions only (interventions not described) (0% vs. 9.2%; $p < 0.001$)³⁹ (*Level 3*).

In a study of a polyurethane foam hydrocellular dressing,³⁸ there was a significant reduction in heel pressure injuries for a group treated with the foam dressing compared to a group receiving a protective heel bandage that covered the ankle articulation. Participants ($n = 133$, $n = 111$ completed the study) were recruited from three long term facilities and three home care programs in Spain. Approximately 3% of individuals in the foam prophylactic dressing group developed pressure injuries compared to 44% in the protective bandaging group. Relative risk of developing a heel pressure injury was 13.42 (95% CI 3.31 to 54.3) for the bandaging group compared to the prophylactic dressing group. It should be noted that heel flotation was not used as a preventive management strategy in this study, and the bandaging intervention used as a control is not considered best practice (*Level 1*).

In a retrospective cohort study in Italy, Forni and colleagues (2011)⁴⁰ investigated the effect of a sterile polyurethane foam pad. Participants were 156 individuals in an orthopedic ward who required the use of a plaster cast to the foot and who had an existing Category/Stage I pressure injury. The study found that the use of a polyurethane foam pad in contact with the heel prior to the application of the plaster cast resulted in fewer heel pressure injuries upon removal of the cast as compared to the control group who did not have a polyurethane foam dressing applied (3.6% intervention group vs. 42.9% control group). The difference equated to a 92% reduction (95% CI 58% to 97%) in heel pressure injuries with the use of the foam dressing⁴⁰ (*Level 3*).

Film prophylactic dressings

Souza et al. (2013)⁴¹ studied the efficacy of a polyurethane film dressing in a study of 100 individuals in an ICU who acted as their own controls. The overall heel pressure injury rate was 32% for this study. The intervention foot (left heel), which received a prophylactic polyurethane film dressing in addition to standard care, experienced significantly lower pressure injury rates as compared to the right heel that received standard treatment only (standard treatment not defined) (6% vs. 18%; $p < 0.001$)⁴¹ (*Level 2*).

Silicone pad prophylactic dressings

In a study exploring prevention of heel pressure injuries in 14 long term care residents assessed as being at risk or at high risk, Knowles et al. (2013)⁴³ investigated the use of a silicone pad held in place with a tubular bandage as a prophylactic dressing (intervention). The comparator group received an alternative polymer-based heel pad, no pad or a wool pad. Photography and high definition ultrasound were used to assess dermal water content and the extent of edema as indicators of tissue inflammation. Comparisons were made between results for the heels compared to adjacent normal skin. In heels receiving the prophylactic dressing and those in the control group, ultrasound results indicated high levels of edema were present at the trial commencement, after a six-week run in period in which standard care (no dressings) was delivered. After four weeks, heels receiving the prophylactic dressing demonstrated reduction in edema, indicating that sub-dermal inflammation had substantially lessened. The comparator group showed no change in ultrasound measurements over time⁴³ (*Level 2*).

The above results indicate that using a prophylactic dressing is likely to reduce the risk of pressure injuries in individuals at high risk as compared to not using a prophylactic dressing. Selection of prophylactic dressing for use on the heels requires consideration of factors influencing application of the dressing and maintaining it in place, the ability to regularly assess the heel by lifting the dressing, the ability to manage microclimate and the comfort of the dressing, as determined by the individual.

Treatment of Heel Pressure Injuries

Before treating a heel pressure injury, the healing status of the wound (healable, maintenance, or non-healable) should be considered along with the individual's care goals.⁴⁴ Vascular status of the lower limb should be assessed, and peripheral vascular disease should be addressed before any treatment of heel pressure injuries (see *Good Practice Statement 6.1*). Wound bed preparation practices⁴⁵ that should be adopted when treating heel pressure injuries include those outlined in the guideline chapters *Cleansing and Debridement* and *Infection and Biofilm*.

Of particular significance to treatment of heel pressure injuries is the management of heel eschar. As discussed in the guideline chapter *Cleansing and Debridement*, stable heel eschars or eschars in the presence of untreated peripheral

vascular disease should not be debrided. However, if there is high suspicion of infection, heel eschars should be debrided to reveal the base of heel pressure injury to enable comprehensive assessment and treatment of the heel pressure injury. Non-viable, necrotic or infected tissue should be removed via appropriate methods of debridement.

Selection of appropriate heel offloading is critical to treating heel pressure injuries (see *Recommendation 6.2* and *Good Practice Statement 6.3*).

Selection of an appropriate wound dressing for the heel is often complicated by the difficulty in applying wound dressings to this anatomical location. Some research has explored padded wound dressings designed specifically for the heels. In one study⁴⁶ exploring the effectiveness of padded dressings for heel ulcers, there was a significant difference in healing in a cohort receiving the padded dressing (n = 20) compared to a cohort that did not receive the padded dressing (n = 20; 100% vs 65%, p < 0.01). The padded heel dressing in this study consisted of a non-adherent mesh dressing applied beneath two rolls of cast padding. There was also significantly lower nursing cost associated with using the padded heel dressing (\$114,080 CAD versus \$245,055, p < 0.001) (*Level 3*).⁴⁶ The study did not state clearly the etiology of the heel ulcers and selection criteria for participants was not reported.

Bateman (2014)⁴⁷ conducted an observational study evaluating foam cushions in treating heel pressure injuries. Participants (n = 50) were recruited from aged care, respiratory and orthopedic wards in a UK hospital. In this study, 100% of Category/Stage I pressure injuries, 80% of Category/Stage II pressure injuries, 100% of Category/Stage III pressure injuries and 66% of Category/Stage IV pressure injuries improved when a foam cushion was used to protect the heel during healing⁴⁷ (*Level 4*). Minimal information on how the foam heel cushion was used was provide in the report and there was no statistical analysis of the results.

Ultimately as with any wound, treatment should be evidence-based, reflective of the wound bed characteristics, and be consistent with the patient's goals of care. Treatment regimens should be re-evaluated on a regular basis and the treatment plan adjusted accordingly.⁴⁸

References

1. Vanderwee K, Clark M, Dealy C, Gunningberg L, Defloor T. Pressure ulcer prevalence in Europe: A pilot study. *J Eval Clin Pract*, 2007; 13(2): 227-232.
2. Barrois B, Colin D, Allaert FA. Prevalence, characteristics and risk factors of pressure ulcers in public and private hospitals care units and nursing homes in France. *Hosp Pract*, 2018; 15: 15.
3. Sanchez-Lorente MM, Sanchis-Sanchez E, Garcia-Molina P, Balaguer-Lopez E, Blasco JM. Prevalence of pressure ulcers in the paediatric population and in primary health care: An epidemiological study conducted in Spain. *J Tissue Viability*, 2018.
4. Tubaishat A, Papanikolaou P, Anthony D, Habiballah L. Pressure ulcers prevalence in the acute care setting: A systematic review, 2000-2015. *Clin Nurs Res*, 2018; 27(6): 643-659.
5. Gaubert-Dahan ML, Castro-Lionard K, Blanchon MA, Fromy B. Severe sensory neuropathy increases risk of heel pressure ulcer in older adults. *J Am Geriatr Soc*, 2013; 61: 2050-2052.
6. Luboz V, Perrier A, Bucki M, Diot B, Cannard F, Vuillerme N, Payan Y. Influence of the calcaneus shape on the risk of posterior heel ulcer using 3D patient-specific biomechanical modeling. *Ann Biomed Eng*, 2015; 43(2): 325-335.
7. Bucki M, Luboz V, Perrier A, Champion E, Diot B, Vuillerme N, Payan Y. Clinical workflow for personalized foot pressure ulcer prevention. *Med Eng Phys*, 2016; 38(9): 845-853.
8. Lupianez-Perez I, Uttumchandani SK, Morilla-Herrera JC, Martin-Santos FJ, Fernandez-Gallego MC, Navarro-Moya FJ, Lupianez-Perez Y, Contreras-Fernandez E, Morales-Asencio JM. Topical olive oil is not inferior to hyperoxygenated fatty acids to prevent pressure ulcers in high-risk immobilised patients in home care. Results of a multicentre randomised triple-blind controlled non-inferiority trial. *PLoS One*, 2015; 10(4).
9. Houwing R, van der Zwet W, van Asbeck S, Halfens R, Arends JW. An unexpected detrimental effect on the incidence of heel pressure ulcers after local 5% DMSO cream application: A randomized, double-blind study in patients at risk for pressure ulcers. *Wounds*, 2008; 20(4): 84-88.
10. Delmore B, Lebovits S, Suggs B, Rolnitzky L, Ayello EA. Risk factors associated with heel pressure ulcers in hospitalized patients. *J Wound Ostomy Continence Nurs*, 2015; 42(3): 242-248.
11. Delmore B, Ayello EA, Smith D, Chu AS, Rolnitzky L. Refining heel pressure injury risk factors in the hospitalized patient. *Adv Skin Wound Care*, in Press.
12. Cox J. Pressure injury risk factors in adult critical care patients: A review of the literature. *Ostomy Wound Manage*, 2017; 63(11): 30-43.
13. Twilley H, Jones S. Heel ulcers - Pressure ulcers or symptoms of peripheral arterial disease? An exploratory matched case control study. *Journal of Tissue Viability*, 2016; 25(2): 150-156.
14. McGinnis E, Greenwood DC, Nelson EA, Nixon J. A prospective cohort study of prognostic factors for the healing of heel pressure ulcers. *Age Ageing*, 2014; 43(2): 267-271.
15. Tehan PE, Santos D, Chuter VH. A systematic review of the sensitivity and specificity of the toe-brachial index for detecting peripheral artery disease. *Vasc Med*, 2016; 21(4): 382-389.
16. Twilley H, Jones S. Heel ulcers - Pressure ulcers or symptoms of peripheral arterial disease? An exploratory matched case control study. *J Tissue Viability*, 2016; 25(2): 150-156.

17. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FGR. Inter-society consensus for the management of peripheral arterial disease (TASC II). *J Vasc Surg*, 2007; 45(1): S5-S67.
18. Crowell A, Meyr AJ. Accuracy of the ankle-brachial index in the assessment of arterial perfusion of heel pressure injuries. *Wounds*, 2017; 29(2): 51-55.
19. Bååth C, Engstrom M, Gunningberg L, Muntlin Athlin A. Prevention of heel pressure ulcers among older patients - from ambulance care to hospital discharge: A multi-centre randomized controlled trial. *Appl Nurs Res*, 2016; 30: 170-5.
20. Donnelly J, Winder J, Kernohan WG, Stevenson M. An RCT to determine the effect of a heel elevation device in pressure ulcer prevention post-hip fracture. *J Wound Care*, 2011; 20(7): 309.
21. Cadue JF, Karolewicz S, Tardy C, Barrault C, Robert R, Pourrat O. [Prevention of heel pressure sores with a foam body-support device. A randomized controlled trial in a medical intensive care unit]. *La Presse Médicale*, 2008; 37(1 Pt 1): 30-36.
22. Meyers T. Prevention of heel pressure injuries and plantar flexion contractures with use of a heel protector in high-risk neurotrauma, medical, and surgical intensive care units: A randomized controlled trial. *J Wound Ostomy Continence Nurs*, 2017; 44(5): 429-433.
23. Cheneworth CC, Hagglund KH, Valmassoi B, Brannon C. Portrait of practice: healing heel ulcers. *Adv Wound Care*, 1994; 7(2): 44-48.
24. Rajpaul K, Acton C. Using heel protectors for the prevention of hospital-acquired pressure ulcers. *Br J Nurs*, 2016; 25(6 Suppl): S18-26.
25. Jones NJ, Ivins N, Ebdon V, Clark M. An evaluation of the use of the Maxxcare Pro Evolution Heel Boot in a rehabilitation care setting. *Wounds UK*, 2017; 13(4): 100-106.
26. Gleeson D. Heel pressure ulcer prevention: A 5-year initiative using low-friction booties in a hospital setting. *Wounds UK*, 2016; 12(4): 80-87.
27. Huber J, Reddy R, Pitham T, Huber D. Increasing heel skin perfusion by elevation. *Adv Skin Wound Care*, 2008; 21(1): 37-41.
28. Bales I. A Comparison between the use of intravenous bags and the heelift suspension boot to prevent pressure ulcers in orthopedic patients. *Adv Skin Wound Care*, 2012; 25(3): 125-131.
29. Meyers TR. Preventing heel pressure ulcers and plantar flexion contractures in high-risk sedated patients. *J Wound Ostomy Continence Nurs*, 2010; 37(4): 372-378.
30. Malkoun M, Huber J, Huber D. A comparative assessment of interface pressures generated by four surgical theatre heel pressure ulcer prophylactics. *Int Wound J*, 2012; 9(3): 259-263.
31. Masterson S, Younger C. Using an alternating pressure mattress to offload heels in ICU. *Br J Nurs*, 2014; 23(15): S44-S49.
32. Huber DE, Huber JP. Popliteal vein compression under general anaesthesia. *Eur J Vasc Endovasc Surg*, 2009; 37(4): 464-469.
33. Call E, Pedersen J, Bill B, Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Clark M. Enhancing pressure ulcer prevention using wound dressings: What are the modes of action? *Int Wound J*, 2015; 12(4): 408-413.
34. Levy A, Gefen A. Computer Modeling Studies to Assess Whether a Prophylactic Dressing Reduces the Risk for Deep Tissue Injury in the Heels of Supine Patients with Diabetes. *Ostomy Wound Manage*, 2016; 62(4): 42-52.
35. Levy A, Frank MBO, Gefen A. The biomechanical efficacy of dressings in preventing heel ulcers. *J Tissue Viability*, 2015; 24(1): 1-11.
36. Call E, Pedersen J, Bill B, Oberg C, Ferguson-Pell M. Microclimate impact of prophylactic dressings using in vitro body analog method. *Wounds* 2013; 25(4): 94-103.
37. Santamaria N, Gerdtz M, Sage S, McCann J, Freeman A, Vassiliou T, De Vincentis S, Ng AW, Manias E, Liu W, Knott J. A randomised controlled trial of the effectiveness of soft silicone multi-layered foam dressings in the prevention of sacral and heel pressure ulcers in trauma and critically ill patients: The Border trial. *Int Wound J*, 2015; 12(3): 302-308.
38. Torra I Bou JE, Rueda López J, Camañes G, Herrero Narváez E, Blanco Blanco J, Ballesté Torralba J, Martínez-Esparza EH, García LS, Soriano JV. Preventing pressure ulcers on the heel: a Canadian cost study. *Dermatol Nurs*, 2009; 21(5): 268-272.
39. Santamaria N, Gerdtz M, Liu W, Rakis S, Sage S, Ng AW, Tudor H, McCann J, Vassiliou J, Morrow F, Smith K, Knott J, Liew D. Clinical effectiveness of a silicone foam dressing for the prevention of heel pressure ulcers in critically ill patients: Border II Trial. *J Wound Care*, 2015; 24(8): 340-345.
40. Forni C, Loro L, Tremosini M, Mini S, Pignotti E, Bigoni O, Guzzo G, Bellini L, Trofa C, Guzzi M. Use of polyurethane foam inside plaster casts to prevent the onset of heel sores in the population at risk. A controlled clinical study. *J Clin Nurs*, 2011; 20(5/6): 675-680.
41. Souza TS, Reichembach Danski MT, Johann DA, Marques De Lazzari LS, Mingorance P. Prevention's pressure ulcers heel with transparent polyurethane film. *Acta Paulista de Enfermagem*, 2013; 26(4): 345-352.
42. Miller SK, Sharma N, Aberegg LC, Blasiole KN, Fulton JA. Analysis of the pressure distribution qualities of a silicone border foam dressing. *J Wound Ostomy Continence Nurs*, 2015; 42(4): 346-351.
43. Knowles A, Young S, Collins F, Hampton S. Report on a clinical evaluation of the KerraPro Heel silicone heel pad. *J Wound Care*, 2013; 22(11): 599-607.
44. Sibbald RG, Ovington LG, Ayello EA, Goodman L, Elliott JA. Wound bed preparation 2014 update: Management of critical colonization with a gentian violet and methylene blue absorbent antibacterial dressing and elevated levels of matrix metalloproteases with an ovine collagen extracellular matrix dressing. *Adv Skin Wound Care*, 2014; 27(3 (Suppl 1)): 1-6.
45. Schultz GS, Sibbald RG, Falanga V, Ayello EA, Dowsett C, Harding K, Romanelli M, Stacey MC, Teot L, Vanscheidt W. Wound bed preparation: a systematic approach to wound management. *Wound Repair Regen*, 2003; 11 Suppl 1: S1-28.
46. Campbell NA, Campbell DL, Turner A. A Retrospective quality improvement study comparing use versus nonuse of a padded heel dressing to offload heel ulcers of different etiologies. *Ostomy Wound Manage*, 2015; 61(11): 44-52.
47. Bateman SD. Utilising a foam positioning device for preventing pressure ulcers on the feet. *Wounds UK*, 2014; 10(1): 78-83.
48. Sibbald RG, Goodman L, Woo KY, Krasner DL, Smart H, Tariq G, Ayello EA, Burrell RE, Keast DH, Mayer D, Norton L. Special considerations in wound bed preparation 2011: An update. *Adv Skin Wound Care*, 2011 24(9): 415-436.

SUPPORT SURFACES

Introduction

Support surfaces are:

“specialized devices for pressure redistribution designed for management of tissue loads, microclimate, and/or other therapeutic functions (i.e., any mattress, integrated bed system, mattress replacement, overlay, or seat cushion, or seat cushion overlay).”¹

In this context, pressure refers to distribution of forces on the individual’s body surface that is in contact with the device. As an individual immerses (sinks) into the support surface, their weight can be redistributed over a larger area. If the surface also envelops (i.e., conforms to the shape of) the individual, the pressure on the individual’s body will be more evenly distributed and less concentrated over bony prominences where pressure injuries typically develop. Periodic recovery from deformation might be achieved if the surface actively reduces tissue loads in vulnerable areas. In practice, as a person lies or sits on a support surface their weight causes both the support surface and their own soft tissue to deform. The extent to which forces are concentrated in small areas will determine the degree of potentially damaging deformation in the underlying skin and soft tissues. Friction is a force resisting an individual’s body from sliding on the top of the support surface and can contribute to tissue deformation. Friction is in part dependent on moisture, which might be controlled by targeted support surface features.

Terminology describing types of support surfaces and their features have been developed.² A **reactive support surface** is a powered or non-powered support surface with the ability to change its load distribution properties only in response to an applied load. An **active support surface** is a powered support surface that has the ability to change its load distribution properties with or without an applied load.² Support surfaces with alternating pressure features; that is, a feature that provides pressure redistribution via cyclic changes in loading and unloading (i.e. inflation and deflation of air-filled cells) as characterized by frequency, duration, amplitude, and rate of change parameters are an example of an active support surface.²

Support surfaces are typically constructed from a range or combination of materials including, but not limited to, air, foam, gel and fluid and incorporate specific structures (e.g., bladders and modules that may be arranged in zones corresponding to anatomical locations). Support surfaces can either be powered or non-powered. Power is used in some devices to alter the immersion and envelopment characteristics of the surface, to control the microclimate or to periodically redistribute pressure. Powered features designed to influence the microclimate include heating, cooling and controlling moisture dissipation. A powered feature intended to affect microclimate is low air loss. Low air loss describes a feature where air is circulated beneath a water vapor permeable cover to control the humidity at the interface between the individual and the support surface. A low air loss feature might be included on either an active or reactive support surface. Powered features designed to change load bearing characteristics include air fluidization of granular materials (e.g., beads). Another example of a powered support surface is a support surface that adjusts air volume within bladders in response to the weight and/or morphology of the individual.

Support surface characteristics such as immersion, envelopment, and microclimate modification will vary substantially from device to device both within and across categories (active or reactive), if they are powered or non-powered, or if they implement such features as alternating pressure and low air loss. Standard tests that quantify performance characteristics have been developed to assist in matching users’ needs to support surface capabilities. The Rehabilitation Engineering and Assistive Technology Association of North America (RESNA) in collaboration with the American National Standards Institute and the National Pressure Ulcer Advisory Panel (NPUAP) has published standard test methods for quantifying most of these characteristics for mattresses.² The RESNA standard² includes methods to measure immersion, envelopment, heat and water vapor dissipation, and horizontal stiffness. Importantly, the standard is intended to offer methods of bench testing to identify clinically meaningful metrics of support surfaces for comparison. A consensus document from The Tissue Viability Society provides guidance on the measurement of interface pressures applied by active therapy support surfaces.³ International standards have also been published to describe performance characteristics for seat cushions.⁴ None of these standards include thresholds to represent specific levels of performance, as these requirements generally vary from person to person. Standards also serve manufacturers as a product development guide and to enhance product quality. Refer to the *Glossary* for selected terms and definitions associated with support surfaces.

This chapter addresses support surface recommendations for individuals at risk of pressure injuries or with existing pressure injuries, including individuals with a range of population-specific needs (e.g., neonates and children, obese

individuals and critically ill individuals). The guideline chapter on *Pressure Injury Surgery* includes discussion on support surface use following surgical repair of pressure injuries.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What reactive support surfaces are effective in preventing pressure injuries?
- What active support surfaces are effective in preventing pressure injuries?
- When should an active support surface be used to prevent pressure injuries?
- What is the most effective seating support surface for preventing pressure injuries?
- What reactive support surfaces are effective in supporting pressure injury healing?
- What active support surfaces are effective in supporting pressure injury healing?
- When should an active support surface be used to support pressure injury healing?
- What is the most effective seating support surface for preventing pressure injuries?

Support Surface Selection and Use

Support surfaces are an important element in pressure injury prevention and treatment because they can prevent damaging tissue deformation and provide an environment that enhances perfusion of at risk or injured tissue. Support surfaces alone neither prevent nor heal pressure injuries, but support surfaces play a significant role in an individualized comprehensive management plan for pressure injury prevention and treatment. Pressure injury risk factors vary from person to person. Choosing a support surface for an individual should take into account their specific needs.

7.1: Select a support surface that meets the individual's need for pressure redistribution based on the following factors:

- Level of immobility and inactivity
- Need to influence microclimate control and shear reduction
- Size and weight of the individual
- Number, severity and location of existing pressure injuries
- Risk for developing new pressure injuries.

(Good Practice Statement)

Implementation Considerations

- Continue to reposition individuals regardless of the type of pressure redistribution support surface being used. Advise individuals to perform regular offloading and repositioning as much as possible when spending prolonged periods on any support surface (*Expert opinion*).
- Choose a support surface that is compatible with the care setting. For individuals in the community, consider the impact support surfaces might have on the home environment, sleeping arrangements and safety. Make selections for support surfaces used in the home in consultation with the individual and their informal caregivers (*Expert opinion*).
- Some support surfaces can reduce mobility and egress from the bed. Balance the need to prevent pressure injuries with promotion of early mobilization and activity (*Expert opinion*).
- Choose positioning devices, incontinence pads, clothing and bed linen that are compatible with the support surface (*Expert opinion*). Bed linen is discussed in more detail in the guideline chapter *Preventive Skin Care*.
- Limit the amount of linen and pads placed on the support (*Expert opinion*).
- Routinely check for 'bottoming out' of the support surface. The weight of an individual, as well as weight distribution and shape, may cause support surface deformation beyond critical immersion whereby effective pressure redistribution is lost (*Expert opinion*).
- Examine the functionality of the support surface on every encounter with the individual and identify potential complications (*Expert opinion*).
- Evaluate the appropriateness of the support surface after every risk or skin assessment (*Expert opinion*).
- Before use, verify that the support surface is being used within its functional life span, as indicated by the manufacturer's recommended test method (or other industry recognized test method) (*Expert opinion*).

Selecting a Support Surface in All Care Settings

Immobility is the key condition that increases risk of pressure injuries. This risk is increased when immobile individuals are unable to turn or reposition themselves, are experiencing pain and discomfort on movement, or when they are unaware of the need to move about in bed. The key support surface characteristics to consider when selecting a support surface are those features and characteristics that affect pressure redistribution, friction and shear force management, and microclimate. Friction and shear force management are particularly important for individuals using a support surfaces with the head of the bed raised or who are immobile and may be dragged across the support surface. Friction and shear can be reduced with a low friction fabric⁵ (see the guideline chapter *Preventive Skin Care*). Individuals with damp skin (e.g., from perspiration, moisture trapping, fever and incontinence) may benefit from microclimate features. The coefficient of friction is greater over moist skin, potentially resulting in greater tissue damage.^{6,7}

When selecting a support surface, consideration should be given to where the support surface and/or bed will be placed. Consider:

- Weight of the bed
- Structure of the building, including width of doors
- Availability of uninterrupted electrical power
- Safe location for the pump or motor, including its ventilation.

Contingency plans for power failure should be in place.

Powered support surfaces can generate heat, noise and motion. One trial conducted in older women confined to bed (n = 10) reported that automated tilted beds were associated with a non-significant change in high frequency components of the heart rate; however, this is an infrequent occurrence⁸ (*Level 5*). These factors have varying levels of acceptability.

Beds that produce air flow at the skin interface (i.e., specifically air fluidized beds) can accelerate the evaporation of perspiration.⁹ In some cases this may lead to dehydration; however modern bed systems often have features to control air flow. This insensible loss should be considered in daily fluid status assessment. Beds that lead to a sensation of floating may lead to disorientation and confusion; in such cases, reorientation and explanations of the bed's function may be helpful.

Individuals should not lie on a pressure injury. However, there are situations in which the individual cannot be positioned off a pressure injury because there are pressure injuries on multiple anatomical sites. For individuals with existing full thickness pressure injuries (i.e., Category/Stage III or IV pressure injuries, unstageable pressure injuries and deep tissue pressure injuries), perfusion to injured tissue may benefit from support surfaces with additional features (e.g., alternating pressure or air fluidized). Other support surfaces may be adequate for partial thickness pressure injuries (i.e., Category/Stage I and II pressure injuries). See below for recommendations on selecting support surfaces specifically for individuals with existing pressure injuries.

After selecting a support surface its appropriateness should be verified with the individual using the support surface. Any support surface can fail, be less than adequate for an individual's clinical needs, or be uncomfortable.

7.2: Ensure that the bed surface area is sufficiently wide to allow turning of the individual.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

There is indirect evidence¹⁰ to suggest that individuals with obesity require a wider bed to adequately turn/be repositioned from one side to the other. Although there is no documented research, it is logical that providing a wider bed surface for individuals with overweight or obesity would reduce the risk of pressure on the skin and tissue from bed rails. There is no direct evidence to empirically demonstrate that providing a bariatric hospital bed prevents pressure injuries from occurring.

Implementation Considerations

- Bed sides or rails, bedside furniture and equipment at the bedside can be a source of device related pressure injuries if there is insufficient clearance between the individual and the edge of the bed (*Expert opinion*).

- Be aware of manual handling risk to the caregiver (i.e., staff leaning/reaching further to care for an individual) when using extra wide beds (*Expert opinion*).
- Use equipment (e.g., beds, chairs, transfer equipment etc.) that is sufficiently wide and strong to accommodate the individual's girth and weight (*Expert opinion*).
- Ensure the bed is an adequate length to enable correct positioning of the individual on the support surface (*Expert opinion*).

Evidence Discussion

Standard beds are 32 to 36 inches (81 to 91 cm) in width. Individuals who fill the width of the bed may be restricted in their ability to turn side-to-side or into positions that offload the sacral area. Selection of support surfaces should also consider the individual's body dimensions, ensuring there is adequate space for repositioning. When selecting equipment for individuals with obesity, sometimes body weight or body mass index (BMI) can be misleading. Different body shapes may indicate that equipment is appropriate based on weight; however, there may be inadequate breadth/width. In a laboratory study with overweight or obese volunteers,¹⁰ waist circumference was the best predictor of the surface width required for the individual to turn from one side to the other. Body mass index (BMI) was correlated with the surface width required to turn from supine onto one side. The study found that for individuals with a BMI over 40 kg/m², a 50 inch (127 cm) bed surface is required for adequate repositioning. Individual with a BMI below 35 kg/m² required a 36 inch (91 cm) wide bed surface¹⁰ (*Level 5*). The measure of an individual's hip breadth should also be considered when selecting the most appropriate equipment for the individual.

Most facilities will require a range of bariatric equipment to accommodate different body shapes, sizes and mobility status. The equipment needs of a larger, mobile individual are different than those of an individual with obesity who is immobile or unconscious. Diagnostic equipment (e.g., magnetic resonance imaging) often does not accommodate the breadth of an individual with obesity. Measure the individual's physical dimensions prior to proceeding with imaging procedures.

7.3: For individuals with obesity, select a support surface with enhanced pressure redistribution, shear reduction and microclimate features.
(Good Practice Statement)

Discussion

Individuals with obesity are at increased risk of pressure injuries. Obese individuals often experience increased shear and friction, and increased difficulty in redistributing pressure. People with obesity are also at increased risk of stress incontinence and diaphoresis, as well as heat and moisture trapping between the body and the support surface. A support surface that optimizes pressure redistribution and microclimate control is required.

In a small observational study (n = 21), Pemberton et al. (2009)¹¹ provided a low air loss, continuous lateral rotation bed with advanced microclimate technology to individuals with obesity (BMI > 35 kg/m², mean BMI was 51.4 ± 10.3 kg/m²) and pressure injuries. The individuals spent an average of 4.8 ± 2.5 days (range two to eight days) on the specialized support surface. Over the study period no new pressure injuries developed, and existing pressure injuries decreased from an average size of 5.2 ± 2.6 cm² to an average size of 2.6 ± 5.0 cm² (p = not reported). Mean participant comfort rating for the surface was 3.9 out of 4 (*Level 4*).

Using Support Surfaces in All Care Settings

After selecting a support surface, in all cases, the manufacturer's recommendations for the use and maintenance should be followed. It is widely recognized that support surfaces have a finite life span. Determining the condition of a support surface can be accomplished through contractual support surface performance verification conducted by the manufacturer, or by staff trained in the use of industry recognized test methods.^{12,13}

Proper selection and operation of support surfaces is the key to preventing complications. Correctly fitting the mattress to the bed base will mitigate entrapment risks. Overlays placed on top of existing mattresses can elevate the surface to the level of side rails. The top of the side rail should be more than 8.66 inches (220 mm) above the uncompressed mattress (International Electrotechnical Commission [IEC] 60601-2-52). The additional height may make it difficult to transfer onto the bed from a seated position. High beds may be difficult to get in and out of, increasing the risk of falling and injury.

Caregivers and users should follow the supplier's instructions regarding maintenance schedules and care and use of the support surface. Caregivers and users must monitor for power failure and 'bottoming out' (i.e., exceeding the critical immersion threshold) and implement the contingency plan if needed. To prevent falls, electrical cords should be kept away from transfer/walk areas. Support surface pumps and motors should not be obstructed by pillows, bedding, blankets, or clothing. The obstructed motor may overheat and fail to operate. These considerations are especially important for individuals in the home care setting and should be reviewed with the individual or caregiver. Safety suggestions for using powered support surfaces include:

- Avoiding the use of an electric blanket
- Ensuring hot equipment (e.g., hair dryer, heaters), candles and smoking is not within the vicinity of the mattress
- Not overloading power sockets
- Installing smoke detection systems.^{14,15}

Fire retardant properties must meet local standards. However, some materials used to reduce fire risk may compromise management of tissue loads and microclimate. Comprehensive guidance on safety is included in the international standard on general requirements for support surfaces.¹⁶ Specific guidance on the flammability of seating support surfaces can be found in the international standards for wheelchairs and wheelchair seating.⁴

Devices with sharp edges should not be used near support surfaces. Foam positioning wedges can be used to raise the head of the bed in some air fluidized beds. Bed linen, foam devices, and incontinence pads may be necessary to manage comfort, positioning, and moisture. Consider the individual's condition and the types of support surfaces being utilized in order to determine the type and amount of linen to be used. A general rule of thumb is "less is best." In one laboratory study, the impact of adding various combinations of incontinence pads and linen layers to a low-air-loss and to a therapeutic foam support surface was investigated using a pelvic indenter model. The findings indicated statistically significant ($p < 0.0001$) increases in peak sacral interface pressure for all combinations of additional bed linen and/or incontinence pads compared with a single fitted sheet. The percentage increase in peak sacral interface pressure was larger in low-air-loss bed compared to a high specification foam mattress¹⁷ (*Level 5*).

When selecting linen and incontinence pads to place on support surfaces with low air loss features, avoid impeding airflow as this will interfere with the thermal performance properties of the surface. If plastic-backed incontinence pads must be used, use them for dignity when the individual is ambulating and remove them when in bed, or place the pad loosely against the skin to promote as much air flow as possible.¹⁸

Repositioning is still required for pressure relief and comfort when a support surface is in use. The *Repositioning and Early Mobilization* chapter provides recommendations and discussion on repositioning.

Mattress and Bed Support Surfaces for Individuals at Risk of Pressure Injuries

Support surfaces can mitigate pressure injury risk by redistributing pressure, managing friction and shear, managing the microclimate. Pressure redistribution is achieved by either increasing the body surface area that comes in contact with the support surface through immersion and envelopment (to reduce concentrations of weight over bony prominences) or sequentially altering the parts of the body that bear load, thus reducing the duration of loading at any given anatomical site. Measures of interface pressure (pressure at the interface between the body and the support surface) have been frequently reported as surrogate indicators of support surface pressure redistribution efficacy. However, the relevance of interface pressure measurement on individuals is questionable given wide inter-individual responses to applied loads and uncertainty of the relationship between surface pressure and potentially damaging stress and strain in deeper tissues. Recently published US standards² suggest that immersion and envelopment should be used to characterize pressure redistribution and provide test methods for these parameters. In addition to tests for immersion and envelopment, the standards² provide three methods as options for assessing microclimate management:

- Measurement of evaporative capacity (expressed in units of $g/(m^2 \cdot hr)$) and resistance to heat (expressed in units of W/m^2)
- Measurement of the volume of water transferred from a heated water bladder to the surface
- Measurement of relative humidity and temperature after applying a source of water vapor through a simulated user.

Finally, the standards,² American National Standard for Support Surfaces - Volume 1: Requirements and Test Methods for Full Body Support Surfaces (ANSI/RESNA SS-1:2019), provide a test method for assessing the resistance to horizontal displacement that is representative of those forces that might occur if an individual were tending to slide down the support surface with the head of the bed raised.

Single Layer High Specification Foam Mattresses

Characteristics of a high specification single layer foam mattress are presented in Table 10.1.

7.4: Use a high specification reactive single layer foam mattress or overlay in preference to a foam mattress without high specification qualities for individuals at risk of developing pressure injuries. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence from six moderate and low quality Level 1 studies¹⁹⁻²⁴ showed that for individuals at risk of pressure injuries, a high specification foam mattress or overlay is associated with significantly fewer pressure injuries compared with a standard hospital mattress. While three low quality Level 1 studies²⁵⁻²⁷ reported there was no significant differences between a high specification and a standard foam mattress, these studies were of short duration, and one reported no pressure injuries occurred in the study period.²⁵ Additionally, one of these studies²⁶ showed that individuals on a high specification mattress had a significantly longer period of time before a pressure injury occurred. Evidence from two moderate and low quality studies^{28,29} showed there is no significant differences between different types of high specification mattresses or overlays. Evidence from one moderate quality Level 2 study³⁰ found no difference between a high specification foam pad and a foam pad and when pooled, these two reactive support surfaces were not significantly different to an alternating pressure mattress for preventing pressure injuries. Two low quality Level 1 studies^{23,29} reported that individuals rated a high specification mattress as more comfortable than a standard hospital mattress. No recent cost analyses are available to indicate the resource requirements to implement this recommendation; however, ensuring high specification foam mattresses are available could have substantial cost implications in settings or facilities where these support surfaces are not standard.

Implementation Considerations

- Continue to reposition individuals placed on a pressure redistribution support surface. See the guideline chapter on *Repositioning and Early Mobilization* for evidence-based recommendations on repositioning.
- Select a high specification support surface for premature infants and younger children to prevent occipital pressure injuries³¹ (Level 5).
- Not all foam mattresses meet high specification standards (see Table 10.1) (*Expert opinion*).
- Audit all support surfaces use for pressure injury prevention in the facility and ensure they are appropriate for use (*Expert opinion*). The guideline chapter *Implementing Best Practice in Care Settings* provides further information on reviewing facility equipment.

Evidence Discussion

The studies investigating reactive support surfaces that compared standard foam and alternative foam mattresses provided evidence of varying quality. Generally, the studies fail to adequately describe the “standard hospital mattress” used as a comparator, and many studies have limited information on the experimental support surfaces and their mechanisms of action. Some studies excluded Category/Stage I pressure injuries in the reported incidence rates. The variations in interventions and outcome measures, the diverse range of study participants and limitations in reporting make comparisons and interpretation of the evidence difficult. However, the evidence suggests that high specification foam mattresses are associated with a significant reduction in pressure injuries in at-risk individuals when compared to standard hospital foam mattresses. Standard hospital foam mattresses may be made from inferior foam that is not highly resilient, has a lower or higher support factor, or together with the cover does not allow for sufficient moisture vapor transmission.

Studies exploring the effectiveness of high specification foam mattresses have demonstrated large reductions in pressure injury incidence in populations reported to be at moderate to high risk of pressure injuries. Park et al. (2017)¹⁹ reported that hospitalized individuals at moderate to high pressure injury risk receiving a high specification viscoelastic foam overlay placed on a standard mattress (n = 55) experienced statistically significantly fewer Category/Stage I or greater pressure injuries than individuals receiving only a standard hospital foam mattress (n = 55) (3.6% vs 27.3%, p = 0.001) (Level 1). In another study conducted in hospitalized individuals with moderate to high pressure injury risk (n = 170), a high specification mattress was associated with significantly lower incidence of Category/Stage II pressure injuries after ten days compared with a standard hospital mattress (7% versus 34%)²³ (Level 1). In the orthopedic surgery setting, individuals (n = 36) with a high pressure injury risk experienced a significantly lower incidence of Category/Stage II or greater pressure injuries with a high specification cubed foam mattress compared to a standard hospital foam mattress (24% versus 68%).²⁴ (Level 1).

Although some randomized controlled trials (RCTs)^{20,26} failed to demonstrate a statistically significant reduction in pressure injury incidence associated with providing a high specification foam mattress to individuals at moderate to high risk of pressure injury, these studies did follow up at longer duration or until pressure injury onset. In a large RCT (n = 1,168), Russell et al. (2003)²⁰ presented a seven-day survival analysis demonstrating a statistically significant decrease (p = 0.042) in Category/Stage I pressure injuries associated with a high specification viscoelastic polymer foam mattress compared to a standard hospital foam mattress. However, although there was a decrease in the incidence of Category/Stage I pressure injuries that occurred in the high specification viscoelastic polymer foam mattress group, this was not significant (10.9% to 8.5%, p = 0.17)²⁰ (Level 1). Likewise, a large RCT (n = 1,729) by Berthe et al. (2007)²⁶ showed that the time to develop a pressure injury was longer in the individuals receiving a high-specification foam mattress with block structure compared to those receiving a standard hospital foam mattress (31 days versus 18 days, p < 0.001),²⁶ although overall pressure injury incidence was not significantly different (p = 0.154) (Level 1).

Some smaller studies with methodological limitations, reported no significant effect on overall pressure injury incidence. Additionally, the study populations may not have had a moderate to high risk of pressure injuries. In an RCT, Gray and Smith (2000)²⁷ compared a high specification foam mattress (n = 50) to a standard 5 inch (130 mm) thick hospital foam mattress (n = 50) in individuals from surgical, orthopedic, and medical wards. There was no significant difference between the two groups in Category/Stage II to IV pressure injury incidence (2% in both groups).²⁷ (Level 1). A small RCT (n = 90) that appears to be underpowered for the study design failed to demonstrate a statistically significant difference in Category/Stage I or greater pressure injury incidence for seven different high specification mattresses each compared to a standard hospital foam mattress. No pressure injuries occurred in the trial.²⁵ (Level 1)

A systematic review³² pooled the results of five RCTs conducted between 1994 and 2003 comparing foam alternatives with the standard hospital foam mattress. McInnes et al. (2015)³² concluded that high specification foam mattresses reduced the incidence of pressure injuries in individuals at risk (risk ratio [RR] 0.40).

A number of RCTs^{28,29,33} have conducted comparisons between different types of high specification foam mattresses and overlays. The studies, conducted in critically ill,²⁸ orthopedic³⁰ and geriatric^{29,33} populations, provided evidence that no type of high specification foam mattress is superior to any other high specification foam mattresses. In a systematic review, McInnes et al. (2015)³² pooled RCTs comparing different higher specification foam mattresses and found no apparent difference in the incidence of pressure injuries. Additionally, studies reported no statistically significant differences in pressure injury incidence between a high specification foam mattress and either a reactive air mattress (4.8% versus 17.1%, p = 0.08)²² or an alternating pressure air mattress (risk ratio 0.90, 95% CI 0.51 to 1.58)³⁰ (Level 2).

Characteristics of a single layer high specification foam mattress

Table 10.1 outlines consensus opinion,³⁴ describing characteristics for a single layer foam support surface to be considered a high specification foam mattress. It cannot be assumed that the support surfaces reported in the studies outlined above conform to these specifications. We encourage readers to refer to the studies on high specification foam mattresses, and the relevant manufacturer websites to review the characteristics of the surfaces used in these studies. This knowledge can guide selection of comparable high specification foam mattress for clinical use.

Table 10.1: Consensus on characteristics that constitute a single layer high specification foam mattress³⁴

Characteristic	Explanation	High specification mattress
Foam type	Polyurethane foams vary in cell structure and density. High resilience (HR) foam has relatively uniform and dense cell structure that allows it to provide good support while loaded, and also to maintain its shape when unloaded. ²⁷	Type HR ^{35,36} - high resilience ^{26,27,37}
Density	Foam density is expressed in units of lb/ft ³ or Kg/m ³ . Density affects many foam characteristics including resistance to heat flow, durability, resilience, stiffness and strength, among others.	> 35 kg/m ³ (2.18 lb/f ³ ³⁵)
Hardness	Foam hardness characterizes the ability of foam to 'push back' and carry weight. Foam hardness is expressed as the amount of force in Newtons (N) required to indent a sample of the foam by a specific percentage of the original thickness, using a small flat disk indenter. This measure is known as indentation force deflection (IFD). ³⁸ In Australia and Europe, hardness is characterized by 40% IFD. In the US, stiffness is characterized at 25% IFD. ³⁶ The cover and bed linens used can affect IFD if they impede immersion and envelopment.	40% IFD ≥ 130 N ³⁵

Characteristic	Explanation	High specification mattress
Support factor	Support factor (also known as compression modulus) is the ratio of 65% IFD to 25% IFD. Support factor is used to characterize the foam mattresses stiffness. Support factor relates to the perceived comfort, where a higher value usually indicates a softer feel and good base support. ³⁸	Support factor in the range of 1.75 to 2.4 ³⁵
Thickness	Mattress thickness should be sufficiently deep to manage upper body weight and prevent 'bottoming out'. Sufficient thickness will depend on patient weight ³⁹ and other characteristics of the foam (e.g., density and hardness).	5.9 inches (150 mm)
Water vapor permeability	Trans-epidermal water loss (TEWL) and sweating may result in the accumulation of moisture between the individual and the support surface if the surface does not allow for transmission or evaporation of the moisture away from the interface. The combination of a foam mattress cover and the foam itself creates resistance to moisture transmission. Moisture accumulation will increase the friction between the individual and the surface. A mattress cover with high moisture vapor transmission rate (MVTR) potentially allows the moisture to transpire through the cover. ⁴⁰ A lower mattress cover MVTR protects the foam from moisture degradation. Selecting a cover based on MVTR becomes a compromise between managing skin microclimate and the individual's TEWL.	MVTR \geq 300 g/m ² /24hrs (equivalent to normal TEWL) ⁴¹

Current foam support surfaces are rarely made of a single type and layer of foam. Multi-layering of various grades or types of foam alters the design features of the mattress, which might impact effectiveness. An RCT (n = 206) conducted in older adults with a moderate to high pressure injury risk failed to demonstrate the superiority of a high specification multilayered foam overlay over a standard hospital mattress. There was a non-statistically significant higher incidence of Category/Stage II or greater pressure injuries after 12 weeks for individuals receiving the multilayered foam overlay (8.7% versus 4.9%, $p > 0.05$)²¹ (*Level 1*). However, most of the available evidence fails to adequately describe the structure and characteristics of high specification foam mattresses or overlays, particularly when the structure is complex. The evidence presented above indicates that homogenous high specification foam mattresses are superior to standard foam mattresses. Translating this evidence to more complex foam mattresses made from multiple layers and including multiple zones, may be possible by adequately describing foam characteristics and characterizing support surfaces using standard tests for immersion, envelopment, and microclimate management.

Some alterations in design of mattresses aim to promote bed mobility and safety. For example, inclusion of a border or stiffener at the mattress edge (side walls) increases firmness, which may assist bed mobility and transfers. Concave shaping (safety sides) are designed to reduce risk of falls; however, this may reduce bed mobility and be contrary to a restraint-free policy.

High specification foam mattresses for neonates and children

Occipital pressure injuries are a specific concern for neonates and very young children. The head composes a greater percentage of the body surface area than in adults and the occiput is a primary pressure point for children in the supine position.^{42,43} In a survey of seven neonatal intensive care units (NICUs), Fuji et al. (2010)⁴⁴ reported that approximately 7% of pressure injuries were in the occipital region. Schindler et al. (2011)⁴⁵ supported these findings in a survey of nine pediatric intensive care units (PICUs) that reported 6% of pressure injuries were occipital.

Turnage-Carrier et al. (2008)³¹ investigated interface pressure at the occipital bony prominence in healthy premature neonates with no history of pressure injuries and who were close to hospital discharge (n = 11). The neonates were placed on five different support surfaces and interface pressure was measured under the occiput after five minutes. The neonates were consecutively placed on a gel mattress, a gel pillow, a water pillow and a standard crib/cot mattress with 2.75 inches (7 cm) thick foam overlay and a standard (undefined) crib/cot mattress. The high specification foam overlay was associated with the lowest interface pressure (31 mmHg versus 86.8 mmHg for the standard crib/cot mattress, $p < 0.001$), although all the high specification support surfaces were associated with significantly lower interface pressure ($p < 0.001$) compared to a standard crib/cot mattress (*Level 5*).

Reactive Air Pressure Mattresses

Reactive air mattresses redistribute pressure by deforming in response to an individual's weight on the surface. Reactive air mattresses may include low air loss features or other cushioning materials (e.g., foam), but they do not include alternating pressure features.

7.5: Consider using a reactive air mattress or overlay for individuals at risk for developing pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Four level 1 studies of moderate⁴⁶ and low quality^{22,47,48} found no significant effect for a reactive air mattress compared to a standard hospital mattress,^{22,47} other reactive support surfaces,^{46,48} or an alternating pressure air mattress.⁴⁸ Risk ratio for a reactive air mattress compared to an active (alternating pressure) air mattress was 3.08.⁴⁸ However, a low quality Level 1 study⁴⁹ reported a significant reduction in Category/Stage I or greater pressure injuries with a constant low pressure air mattress compared to a standard foam mattress, with a relative risk of 0.06. One low quality Level 3 study conducted in children⁵⁰ reported a significant relative reduction in incidence of Category/Stage I or greater pressure injuries of approximately 17% associated with selecting a reactive air mattress rather than a standard foam mattress. A moderate quality Level 3 study⁵¹ reported an incidence rate for Category/Stage II or greater pressure injuries of around 5% when using a reactive air mattress, and a low quality Level 4 study reported incidence of 3%,⁵² but there were no comparators in these two studies.⁵¹ A moderate quality Level 1 study reported that a reactive air overlay was more cost-effective than a microfluid overlay, however the analysis was based on rental of some products and the relative costs could vary widely based on facility and geographic region.⁴⁶ Ratings of comfort made by individuals indicated that reactive air mattresses are as comfortable as other support surfaces.⁴⁶

Implementation Considerations

- Reactive air mattresses may require a power source. Evaluate accessibility to power and safety of power cords, especially in home care settings (*Expert opinion*).
- Reactive air mattresses require regular inspection to ensure the mattress is intact and the inflation mechanism/ electric pump is correctly functioning (*Expert opinion*).
- Verify that reactive air mattresses are used within their functional life span, as indicated by the manufacturer's recommended test method (or other industry recognized test method) (*Expert opinion*).
- Evaluate the individual's comfort when using a reactive air mattress or overlay. Powered reactive air mattresses and overlays can be noisy and generate heat or motion that may be uncomfortable (*Expert opinion*).
- Evaluate the safety of reactive air mattresses and overlays when in use. Some individuals may experience difficulty getting into and out of the bed when a reactive air mattress or overlay is in use (*Expert opinion*).

Evidence Discussion

After high specification foam mattresses, reactive air mattresses appear to be the second most researched reactive support surface. These trials used a range of both experimental and comparator mattresses and overlays. A broad range of participants were represented in the research including hospitalized adults at pressure injury risk,^{46,52} critically ill individuals,^{48,49} older adults,^{22,47,51} children⁵⁰ and neonates.⁵⁰ However, most of these studies were small (less than 100 participants) and follow up periods range from a few days to 12 months.

Only one small RCT⁴⁹ conducted with individuals at moderate to high risk of pressure injuries provided a comparison between a reactive air mattress and a standard hospital foam mattress. In the study, individuals in an ICU (n = 40) received either a reactive air mattress or a standard hospital foam mattress for up to 14 days. The reactive air mattress was associated with significantly fewer Category/Stage I or greater pressure injuries (0% versus 37%, p < 0.005, relative risk 0.06, 95% CI 0 to 0.99)⁴⁹ (*Level 1*). A third small study (n = 30)⁵⁰ compared a reactive air mattress consisting of double air-cell construction in three different compartments to an undefined standard care support surface provided in an historical cohort; this comparator could be assumed to be a standard hospital foam mattress. Participants were neonates and children aged up to 10 years. The study mattress was available in two sizes, one for children weighing 1.1 lb/500 g to 13.2 lb/6 kgs (n = 4) and one for children above 13.2 lb/6 kgs (n = 26). The reactive air mattress was associated with a significant reduction in facility-acquired pressure injuries (3.3% versus 20%, 95% CI 0.08 to 17.2%, p = 0.021)⁵⁰ (*Level 3*).

However, another RCT failed to demonstrate superiority of a reactive air mattress or overlay to a standard hospital foam mattress. In older adults (n = 66), a reactive air overlay was not statistically significantly different to a standard foam mattress for reducing incidence of Category/Stage II or greater pressure injuries (16% versus 15%, p > 0.05)⁴⁷ (*Level 1*).

Studies with no comparator groups reported varied pressure injury incidence associated with reactive air mattresses. In older adults (n = 176), use of air mattresses and seating cushions was associated with a Category/Stage I pressure injury

incidence of 23.3% and incidence of Category/Stage II or greater pressure injuries of 5.1%⁵¹ (Level 3). In hospitalized individuals (n = 61), a reactive support surface combining air cells with high specification foam was associated with a pressure injury incidence of 3%⁵² (Level 4).

Comparisons between reactive air mattresses/overlays and high specification foam mattresses

Two studies^{22,48} compared a reactive air mattress to support surfaces that provide superior pressure redistribution than a standard hospital foam mattress. A small RCT²² compared a high specification polyether foam mattress to a reactive air overlay in nursing home residents (n = 83). Fewer participants on the air mattress overlay developed Category/Stage II or greater pressure injuries, but the difference was not statistically significant (p = 0.088)²² (Level 1). In individuals in a surgical ICU, no statistically significant difference in pressure injury reduction was established between an alternating pressure air mattress and the pooled results for two groups receiving reactive support surfaces (one group received a reactive air mattress and the other received a water mattress) (RR 0.43, 95% CI 0.04 to 4.29).⁴⁸ (Level 1).

Comparisons between reactive air mattresses/overlays and other reactive or active support surfaces

Finally, two studies provided comparison of a reactive air mattress to other reactive support surfaces.^{46,48} In the largest RCT (n = 110), Vermette et. al. (2012)⁴⁶ compared an air overlay with a microfluid overlay for preventing pressure injuries in participants at moderate to high risk of pressure injuries in acute care wards (medical, surgical, geriatric and intensive care). There were no statistically significant differences between the two overlays for pressure injury incidence (4% for the air inflated overlay versus 11% for the microfluid overlay, p = 0.2706) or participant rated comfort (p = 0.7129). The micro-fluid overlay was reported to be more expensive (p ≤ 0.001)⁴⁶ (Level 1). In individuals in a surgical ICU, no significant difference was established between a reactive air mattress and a water mattress for preventing pressure injuries (RR 0.43, 95% CI 0.04 to 4.29).⁴⁸ (Level 1).

Overall, these studies present a conflicting overview of the effectiveness of reactive air mattresses in comparison to standard foam mattresses, or the comparative effectiveness of different types of reactive mattresses in preventing pressure injuries. It is possible that the design of the individual product could influence the results; however, the small size of the trials and the low methodological quality likely contributes to the variation in reported results.

Medical Grade Sheepskins

7.6: Assess the relative benefits of using a medical grade sheepskin for individuals at risk of developing pressure injuries.

(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

Three Level 1 studies of high,⁵³ moderate⁵⁴ and low quality⁵⁵ indicated that a medical grade sheepskin was effective in reducing the incidence of pressure injuries in individuals at risk. The risk of developing a Category/Stage I or II sacral pressure injury was approximately 40% less when a medical grade sheepskin was used.⁵⁴ No adverse events were reported. However, a small percent of individuals in one trial (4.58%) requested the sheepskin be removed due to discomfort (primarily feeling too hot).⁵⁴ In the high quality Level 1 study a minority of individuals reported the sheepskin was itchy or tickly, and one third (33%) of the individuals reported the sheepskin was too warm, which accounted for 69% of trial withdrawals.⁵³ Medical grade sheepskins may not be available in all geographic locations, and the requirement for specialist laundering to disinfect the sheepskin⁵⁴ might limit their use (e.g., individuals who are incontinent or with heavily exuding wounds).

Implementation Considerations

- Before using a medical grade sheepskin to reduce pressure injury risk, ensure the product has been manufactured to Australian Standard AS4480.1. Not all sheepskins are of an equivalent quality.⁵⁴ A medical grade sheepskin is derived from animals (not synthetic). Medical grade sheepskins have a uniform pile, a high density of wool fibers and greater resilience to laundering (Level 1).
- Consider potential impact of a medical grade sheepskin on the function of the support surface on which it is placed. Adding an additional layer between the individual and the support surface can affect the pressure redistribution properties of the support surface by creating surface tension (sometimes referred to as hammocking) (*Expert opinion*).
- Assess temperature and moisture at the skin-surface interface and evaluate the individual's comfort. Medical grade sheepskins have been associated with excess warmth^{53,54} (Level 1).

- Medical grade sheepskins require specialist laundering to achieve thermal disinfection. This may reduce their feasibility in some clinical settings and for some individuals (e.g., individuals who are incontinent)⁵⁴ (*Level 1*).
- Natural medical grade sheepskins are an animal-based product and as such may not be acceptable to all individuals (*Expert opinion*).

Evidence Discussion

Three RCTs studies⁵³⁻⁵⁵ provided evidence on medical grade sheepskins for preventing pressure injuries. Mistiaen et al. (2010)⁵³ conducted a larger RCT (n = 588) in eight nursing homes comparing a medical grade sheepskin to usual care. A range of basic mattresses were used for most participants, although some of the intervention group (0.9%) and the control group (15.1%, p = 0.06) received a pressure redistributing mattress. In the 30-day follow up period, the intervention group receiving the sheepskin had a statistically significantly lower incidence of sacral pressure injuries (8.9% versus 14.7%, p = 0.035). When adjusting for Braden scale score, age and gender, the odds ratio (OR) of experiencing a pressure injury for the intervention group was 0.53 (95% CI 0.29 to 0.95). The disproportionate use of mattresses with high specification characteristics trended toward favoring the intervention group and might have influenced the outcomes. Jolley et al. (2004)⁵⁴ reported an RCT (n = 441) involving participants at low to moderate risk for pressure injury development, comparing the use of an Australian medical grade sheepskin with standard nursing care (any pressure redistributing strategy decided by a nurse). The pressure injury incidence was 9.6% in the group receiving a medical grade sheepskin, compared with 16.6% in the comparator group. The relative risk was 0.58 (95% CI 0.35 to 0.96).⁵⁴ However, these results should be treated with caution, as there were numerous methodological flaws, and there is a risk of bias (*Level 1*). McGowan et al. (2000)⁵⁵ performed a RCT involving 297 individuals in an orthopedic setting. The experimental group (n = 155) had both an Australian medical sheepskin and a standard hospital mattress, and the comparator group (n = 142) had a standard hospital mattress with or without other low-technology constant pressure supports. The pressure injury incidence in the control group was 30.3% and 9% in the experimental group (p < 0.0001).⁵⁵ Some methodological limitations should be recognized (*Level 1*). In the systematic review by McInnes et al. (2015)³² Australian medical grade sheepskins are suggested for preventing pressure injuries, with a meta-analysis reporting a risk ratio [RR] of 0.56. In the studies exploring subjective evaluations of the medical grade sheepskin, large proportions of the participants reported the sheepskin was too warm, leading to study withdrawals and participants indicating they would not recommend the sheepskin to others^{53,54} (*Level 1*). However, the effectiveness of this support surface might relate to the way in which it influences the microclimate.

Other Reactive Support Surfaces

A small volume of studies provide evidence on other less frequently used reactive support surfaces (e.g., water mattresses, bean mattresses and gel filled mattresses). There is currently insufficient evidence indicating a strong effect for other reactive support surfaces in reducing the incidence of pressure injuries; therefore, no recommendations on the use of these support surfaces can be made.

Alternating Pressure Support Surfaces

7.7: Assess the relative benefits of using an alternating pressure air mattress or overlay for individuals at risk of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

The evidence on alternating pressure air mattresses is mixed but could be explained by the range of different mattresses and the different concurrent interventions implemented in the studies. One moderate quality Level 1 study⁵⁶ reported that at any given time approximately 7.5 times more people on a standard mattress will experience a pressure injury compared with an alternating pressure air mattress (hazard ratio 7.57). A larger moderate quality Level 1 study⁵⁷ showed no significant difference between an alternating pressure air mattress and a viscoelastic polyurethane foam mattress in preventing pressure injuries; however, participants were not regularly repositioned in this trial. A low quality Level 1 study⁵⁸ found no significant effect for a single cell alternating pressure air mattress compared to a polyester mattress, but in the same study a double cell alternating pressure air mattress was associated with significantly reduced pressure injuries. A number of low quality Level 4 studies⁵⁹⁻⁶¹ reported no pressure injuries with alternating pressure air mattresses used for up to six months. High quality Level 1 studies showed no differences between alternating pressure air mattresses and overlays⁶² or between different cell cycle regimens,⁶³ in terms of pressure injury incidence. However, one moderate quality Level 2 study⁶⁴ found an alternating pressure air mattress was more effective than an alternating pressure air overlay. Evaluations from individuals using alternating pressure air mattresses and overlays generally indicated satisfaction.^{56,61,62} A low quality cost analysis⁵⁹ showed reductions in care

costs using a hybrid alternating air/foam mattress and a high quality cost analysis⁶⁵ showed alternating air mattresses were more cost effective than overlays.⁶⁵ Adverse events (e.g., falls) were infrequent and reported more often with an alternating pressure air mattress than with an overlay, although this difference was not significant.⁶⁶

Implementation Considerations

- For many individuals at risk for pressure injuries an alternating support surface may not be applicable because a high specification foam mattress meets the individual's clinical and comfort needs (*Expert opinion*).
- Ensure that the individual's height, weight and age are consistent with the manufacturer's recommendations when placing a child on an alternating pressure support surface. Using a bed designed for an adult presents a safety hazard and may be ineffective in smaller sized children (*Expert opinion*).
- Alternating pressure air support surfaces differ with respect to cell size (height and width) and cell cycle patterns and duration. There is insufficient evidence to recommend any specific design as having greater efficacy in terms of preventing pressure injuries (*Expert opinion*).
- Alternating pressure air mattresses and alternating pressure air overlays have similar efficacy in terms of preventing pressure injuries⁶² (*Level 1*).
- An alternating pressure air mattress overlay requires a quality base mattress. Substandard base mattresses may affect performance (*Expert opinion*).
- Where possible, continue a regular turning and repositioning regimen, with frequency based on the needs of the individual⁵⁶ (*Level 1*).
- All alternating pressure air mattresses require a power source. Evaluate accessibility to power and backup power, and safety of power cords, especially in home care settings (*Expert opinion*).
- Alternating pressure air mattresses require regular inspection to ensure the mattress is intact and the inflation mechanism/electric pump is correctly functioning (*Expert opinion*).
- Verify that alternating pressure air mattresses are used within their functional life span, as indicated by the manufacturer's recommended test method (or other industry recognized test method) (*Expert opinion*).
- Evaluate the individual's comfort when using an alternating pressure air mattress or overlay. Powered alternating pressure air mattresses and overlays can be noisy and generate heat or motion that may be uncomfortable (*Expert opinion*).
- Evaluate the safety of alternating pressure air mattresses and overlays when in use. Some individuals may experience difficulty getting into and out of the bed when an alternating pressure air mattress or overlay is in use (*Expert opinion*).

Evidence Discussion

Studies investigating alternating air pressure mattresses for preventing pressure injuries have been conducted with older adults,⁵⁶⁻⁵⁸ critically ill individuals,⁵⁹ individuals in home care⁶⁷ and rehabilitation⁶⁰ and in medical wards.⁶¹ These studies evaluated alternating pressure air mattresses and overlays that used different designs in terms of cell size, cycles and cell cycle durations, and the preventive care regimens differed across the studies.

Two RCTs⁵⁶ compared an alternating pressure air mattress to a high specification foam mattress. Sauvage et al. (2017)^{56,57} compared an alternating pressure air mattress on a six minute cycle to a high specification foam mattress for preventing pressure injuries in bed-bound older adults (n = 76). The cumulative risk of developing a pressure injury over 30 days was significantly higher in viscoelastic group (38.91%, 95% CI 24.66 to 57.59) compared with alternating pressure air mattress group (6.46%, 95% CI 1.64 to 23.66, p = 0.001). This equated to 7.5 times (95% CI 1.79 to 35.21, p = 0.006) higher risk of developing a pressure injury when using the high specification foam mattress. However, repositioning was performed infrequently in both groups (mean repositioning times over 17 hours was 1.42 ± 2.02 times in the air mattress group versus 1.68 ± 2.17 times in the foam mattress group).⁵⁶ Although the difference was not statistically significant, failure to regularly reposition may have influenced the findings of this study (*Level 1*).

Vanderwee et al. (2005)⁵⁷ compared alternating pressure air mattresses with no turning protocol to high specification foam mattresses with four-hourly repositioning in surgical, internal medicine, and geriatric settings (n = 447). This study showed no statistically significant difference in incidence of Category/Stage II or greater pressure injuries among individuals cared for on either an alternating pressure air mattress (15.6%) compared to a high specification foam mattress (15.3%, p = 1.00). However, there were more heel pressure injuries in the group receiving the foam mattress, but the group receiving the alternating pressure air mattress experienced more severe pressure injuries.⁵⁷ The high incidence of pressure injury development and presence of full thickness pressure injuries in both groups must be acknowledged (*Level 1*).

Comparisons between different types of alternating pressure air mattresses and overlays

Alternating pressure air mattresses and overlays have different designs, types of air cells and deflation/inflation cycles. However, the evidence generally suggests that different active support mattresses and overlays have a similar efficacy in terms of reducing pressure injury incidence.^{58,62,63,68} Only one study⁶⁴ reported a significant difference between different types of alternating pressure air mattresses, and the delay of five years between trialing the two interventions may mean that other components of the care regimen contributed to the observed differences between the support surfaces. The study, conducted in ventilated individuals in the ICU (n = 221), reported an alternating pressure air mattress was more effective in reducing Category/Stage II or greater pressure injuries than a small cell alternating pressure air overlay (OR 0.44, 95% CI: 0.21 to 0.92, p = 0.038)⁶⁴ (Level 2).

Nixon et al. (2006)^{62,66} undertook a large, multicenter RCT (n = 1,971) to compare the effectiveness of alternating pressure mattresses and alternating pressure air overlays for individuals admitted to vascular, orthopedic, medical, and geriatric wards. The incidence of Category/Stage II or greater pressure injuries for those on an alternating pressure air overlay was 10.7% compared with 10.3% for those on an alternating pressure air mattress (p = 0.75), showing no significant difference between the two products for pressure injury incidence. In the mattress group, the mean time to pressure injury development was 10.64 days longer (p > 0.05). Although this was not statistically significantly different, there was a non-significant cost saving associated with using the mattress, primarily due to shorter hospitalization. Although individuals expressed general satisfaction with both the mattress and the overlay, more individuals on the overlay requested to be changed to another support surface. Adverse events deemed to be associated with the support surfaces (e.g. falls and incidents associated with bed rails) were very infrequent, and these occurred more frequently in the mattress group^{62,66} (Level 1). Another RCT included individuals who had experienced a stroke, were recovering from surgery or who had a terminal illness and high risk of pressure injuries (n = 82). Participants using a single cell alternating pressure air mattress had no statistically significant difference in Category/Stage II or greater pressure injuries than individuals using a double cell alternating pressure overlay (13.8% versus 3.8%, p > 0.05)⁵⁸ (Level 1).

In an RCT conducted in 25 hospital wards in Belgium, Demarré et al. (2012)⁶³ compared alternating pressure air mattresses with differing deflation/inflation cycles. The experimental group (n = 298) were cared for on alternating pressure air mattresses with a multi-stage deflation/inflation cycle of between 10 and 12 minutes. The control group (n = 312) had alternating pressure air mattresses with a standard 10-minute deflation/inflation cycle. There was no significant difference between the two groups in the cumulative incidence of Category/Stage II to IV pressure injuries (5.7% versus 5.8%, p = 0.97) or the time to develop a pressure injury (five days versus eight days, p = 0.182).⁶³ There appears to be no benefit of alternating low pressure mattress with multi-stage inflation/deflation cycles over a standard cycle alternating low pressure air mattress in preventing pressure injuries (Level 1). This work continued with a meta-analysis⁶⁸ that pooled results from two RCTs comparing different alternating pressure air mattresses. The results showed no statistically significant difference between an alternating pressure air overlay and a one-stage alternating pressure air mattress (OR = 0.40, 95% CI 0.14 to 1.10). Although fewer pressure injuries developed when using a multi-stage alternating pressure air mattress compared to the alternating pressure air overlay (OR = 0.08, 95% CI 0.01 to 0.83),⁶⁸ the low OR suggests the difference has limited clinical significance.

Alternating pressure support surfaces are designed to support the weight of an adult over a larger number of air cells than will be required to support a child's surface area, resulting in inappropriate pressures. The child's smaller limbs can lodge between alternating pressure air cells, and the sacrum region can rest between cells in the sitting position.⁶⁹ This results in a need to more regularly reposition the child appropriately on the alternating pressure cells.⁶⁹

Although powered support surfaces can be noisy and some individuals dislike a moving support surface, the individuals participating in the above studies generally reported satisfaction^{56,61,67} with alternating pressure air mattresses. Both mattresses and overlays were rated positively by older adults with respect to ease of movement, temperature and sleep disruption⁵⁶ (Level 1).

Low Air Loss Features in Pressure Injury Prevention

The evidence on beds with low air loss features is of low quality and has conflicting findings. One low quality Level 1 study⁷⁰ showed no difference in incidence of Category/Stage II to IV pressure injuries with a bed with low air loss features compared to a range of standard reactive surfaces and alternating mattresses; however, there was a statistically significant reduction in Category/Stage I pressure injuries. The bed used in this study was a prototype design that is no longer available.⁷⁰ A low quality Level 2 study⁷¹ showed similar results, with two analyses finding no statistically significant difference in pressure injury incidence between a low air loss bed and a variety of mattresses described as standard. However, in another low quality Level 1 study⁷² there was an 18% likelihood that individuals would experience a single pressure injury at the same rate on a low air loss bed as on a standard intensive care bed. Another low quality Level 2 study⁷³ showed that a low air loss bed was associated with significant reduction in pressure injury incidence compared to an integrated pressure air distribution mattress. In one Level 1 study⁷⁰ the

majority of individuals who completed a rating of a low air loss bed described it as uncomfortable, and nurses rated the stakeholder acceptability as low, though it should be noted there was no comparison made with the alternate bed in this study. All these studies were small and of low quality, and the results between studies are conflicting and at least one used a prototype bed that is not available.

The manufacturer's weight recommendations for low air loss beds should be followed. Beds with low air loss features have pressure redistribution configurations that are designed for adults. When children are placed on an adult bed, their head is frequently positioned in an area with pressures designed for an adult's trunk.⁷⁴

Support Surfaces to Prevent Pressure Injuries in the Operating Room

7.8: Use a pressure redistribution support surface on the operating table for all individuals with or at risk of pressure injuries who are undergoing surgery.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

One high quality Level 1 study⁷⁵ indicated that a high specification (reactive) support surface (i.e., a visco-elastic polymer [VEP] pad) used in the operating room was associated with a lower incidence of surgery-related pressure injuries when compared to a standard operating mattress. However, a VEP pad was not superior when compared to a high density foam (HDF) pad in a Level 3, moderate quality study.⁷⁶ A moderate quality Level 1 study⁷⁷ found that two different high specification reactive support surfaces had a similar efficacy in preventing pressure injuries, while two low quality Level 1 studies^{78,79} had conflicting results on whether an alternating pressure air mattress was superior to a high specification reactive support surface in preventing pressure injuries in the operating room.

Implementation Considerations

- Low profile alternating pressure overlays may be used in the operating room (*Expert opinion*).
- Review manufacturer guidelines regarding use of the different support surfaces. Some surgical procedures may have specific requirements based on duration, positioning, instrumentation and stability of the operative field (*Expert opinion*).
- Support surfaces used in the perioperative setting require regular maintenance and replacement. Refer to manufacturer guidelines (*Expert opinion*).
- To minimize the time and magnitude of pressure and shear exerted on pressure points, individuals at risk of pressure injuries should also be placed on a pressure redistributing support surface both preoperatively and postoperatively (*Expert opinion*).

Evidence Discussion

In the operating room, the duration of time the individual will spend in one position is generally determined by the surgical procedure (refer to *Recommendation 1.17* in the guideline chapter *Risk Factors and Risk Assessment*). This increases the importance of the support surface in reducing pressure injury risk.⁸⁰ Interface pressures on an operating table can be very high. In a laboratory study of interface pressure measurements among healthy volunteers, Defloor et al. (2000)⁸⁰ found that in four different surgical positions, maximum interface pressure was lowest on viscoelastic foam mattresses, compared to foam mattresses and gel mattresses (*Level 5*). In another laboratory study,⁸¹ sacral interface pressure was measured in healthy volunteers on four foam mattresses with different density, hardness and/or covers. The foam mattress with lowest density and a neoprene cover resulted in the lowest interface pressure. However, other factors including positioning (supine versus Lloyd Davies position) and the individual's body mass index (BMI) influenced interface pressures as much as the type of support surface (*Level 5*).

Several operating room support surfaces that encourage pressure redistribution have been developed. Nixon et al, (1998)⁷⁵ conducted an RCT (n = 446) in individuals undergoing a range of elective surgeries planned to be at least 1.5 hours in duration. The provision of a warming mattress was standardized for all participants, and the support surface was randomized to either a viscoelastic polymer pad or a standard table mattress. The pressure injury incidence in the viscoelastic polymer pad group was significantly lower than in the standard mattress group (11% versus 20%, OR = 0.46, 95% CI 0.26 to 0.82, p = 0.010)⁷⁵ (*Level 1*). However, a non-randomized trial in individuals in the prone position during surgery (n = 30) found that a high specification viscoelastic polymer pad was associated with a lower but not significantly different rate of iliac pressure injuries compared to high density foam pad (10% versus 5%, OR 0.47, 95% CI 0.11 to 1.99, p > 0.05). Although over 75% of individuals undergoing surgery experienced non-blanchable erythema immediately after surgery, this reduced to 10% or fewer cases within 30 minutes⁷⁶ (*Level 2*).

A number of RCTs compared different high specification foam support surfaces for effectiveness in preventing surgery-related pressure injuries. The incidence of pressure injuries in these studies ranged from 0% to 17.6%.⁷⁷⁻⁷⁹ Feuchtinger et al. (2006)⁷⁷ performed a RCT in individuals undergoing cardiac surgery for a minimum of 1.5 hours (n = 175). The experimental group were given a thermoactive viscoelastic foam overlay combined with a water-filled warming mattress while the control group received a water-filled warming mattress. There was a non-significant increase in pressure injuries in the intervention group compared with the control group (17.6% versus 11.1%, p = 0.22) (Level 1). Two RCTs evaluated the use of alternating pressure air mattresses (a multi-segmented pad with more than 2,500 air cells enclosed in a waterproof cover) during and after surgery of at least four hours' duration. In both control groups participants were on a gel mattress during surgery and a standard mattress after surgery.^{78,79} Aronovitch et al. (1999)⁷⁸ reported a pressure injury incidence of 8.7% in the control group and no pressure injuries in the intervention group (p < 0.005) (Level 1). Russell and Lichtenstein (2000)⁷⁹ studied 198 individuals undergoing cardiothoracic surgery, reporting pressure injury incidence of 7% in the control group and 2% in the intervention group (p = 0.17) (Level 1). However, from these studies, it cannot be concluded whether the reduction in pressure injuries was related to the multi-segmented alternating pressure air mattress or to the postoperative pressure redistribution, or to a combination of both.

Mattress and Bed Support Surfaces for Individuals with Existing Pressure Injuries

Individuals with an existing pressure injury are at higher risk for developing additional pressure injuries.^{65,82-86} In many cases, a small Category/Stage I or II pressure injury can be easily offloaded with repositioning, such as turning side to side (for sacral pressure injuries) or using heel elevation. However, clinical judgment may lead the health professional to review the support surface in high risk or hemodynamically unstable individuals with Category/Stage I or II pressure injuries, particularly if there are multiple injuries at multiple sites or the individual cannot be moved off the pressure injury.

7.9: For individuals with a pressure injury, consider changing to a specialty support surface when the individual:

- Cannot be positioned off the pressure injury
- Has pressure injuries on two or more turning surfaces (e.g., the sacrum and trochanter) that limit repositioning options
- Has a pressure injury that fails to heal or that deteriorates despite appropriate comprehensive care
- Is at high risk for additional pressure injuries
- Has undergone flap or graft surgery
- Is uncomfortable
- 'Bottoms out' on the current support surface.

(Good Practice Statement)

Implementation Considerations

- Wherever possible, do not position an individual on an existing pressure injury (*Expert opinion*).
- Before replacing the existing support surface evaluate the effectiveness of previous and current prevention and treatment plans (*Expert opinion*).
- Before replacing the existing support surface set treatment goals consistent with the individual's goals, values, and lifestyle (*Expert opinion*).
- Specialty support surfaces to consider for individuals with a pressure injury include alternating pressure air mattresses, mattresses with a low-air-loss feature and air fluidized beds (*Expert opinion*).

Discussion

Unless the individual's clinical condition has changed (e.g., the individual is now mobile, awake, and has adequate perfusion), the support surface on which the pressure injury developed usually does not provide an appropriate environment for healing. A different support surface is often required to provide better pressure redistribution, friction and shear force management and modification of microclimate, thus reducing further ischemia or deformation induced pressure injuries. 'Bottoming out' on a support surface (i.e., when the support surface deformation is beyond critical immersion whereby effective pressure redistribution is lost) is a clear indication that pressure redistribution is inadequate, and the support surface must be changed. When pressure injuries deteriorate or fail to heal, the health professional should consider replacing the existing support surface with one that will provide a properly matched support surface environment that reduces related risk factors. However, changing the support surface is only one of

several strategies to consider. More frequent repositioning of the individual may be needed. Preventive interventions and local wound care should also be intensified as needed. Specialty support surfaces have additional technology (e.g., alternating pressure, air fluidized or loss air loss features) designed to further redistribute pressure, reduce shear and influence the microclimate.

When the individual has pressure injuries on two or more sites on the trunk of the body, options for repositioning are diminished. When pressure injuries are present on two or more turning surfaces (e.g., the sacrum and trochanters) the individual will need to be repositioned on the pressure injuries, because they cannot continuously lie on the same turning surface.^{62,87} The individual will spend relatively more time on unaffected areas of the body therefore prevention becomes even more crucial in individuals at risk of developing additional pressure injuries.

Non-Powered Reactive Support Surfaces for Individuals with a Pressure Injury

Few studies investigate the effectiveness of reactive support surfaces for individuals with an existing pressure injury. A low quality Level 1 study⁸⁸ and a low quality Level 3 study⁸⁹ provide some evidence that use of reactive support surfaces increases pressure injury healing rates. However, evidence was presented in a low quality Level 3 study⁸⁹ that suggested that reactive support surfaces are inferior to alternating pressure air support surfaces with regards to number of pressure injuries healed. However, in this study the alternating pressure air mattress group had a non-significantly higher rate of new pressure injuries, leading the authors⁸⁹ to conclude that overall, the reactive surface was superior. High level evidence for effectiveness in pressure injury healing in comparison to active support surfaces, and comparisons between different reactive support surfaces (e.g., reactive air mattresses, high specification foam mattresses etc.) is lacking. The reviewed evidence has limitations brought about by other pressure injury risk factors such as end-of-life care, restrictions in positioning and impact of microclimate that may have confounded results and led to inconsistencies in the evidence base. Therefore, no specific recommendation is made on using reactive surfaces for individuals with an existing pressure injury.

Specialty Support Surfaces for Individuals with a Pressure Injury

Numerous studies compare healing rates for Category/Stage III and IV pressure injuries on a range of different specialty support surfaces. It is difficult to make definitive recommendations for some of these support surfaces based on the available studies due to differences in the support surfaces tested, variations in outcome measures (i.e., complete healing, time to healing, reduction in wound size, or assessment of wound improvement/deterioration), small sample sizes, and other methodological inconsistencies. Most of these studies were published over 30 years ago. Since that time, technology has improved for both powered and non-powered surfaces and the support surfaces often used as comparators.

Use of specialty support surfaces for individuals with suspected deep tissue injury with intact skin has not been rigorously examined. The true level and degree of tissue damage may be difficult to determine until the deep tissue injury fully demarcates. At early stages of evolution (when the skin is still intact), offloading and pressure redistribution may allow injured tissue to repair, limiting the extent of infarcted or dead tissue. Infarcted tissue is not salvageable. For all practical purposes, evolving deep tissue injury should be provided the same level of support surface intervention as a Category/Stage III or IV pressure injury. Once the pressure injury has fully demarcated, support surface needs should be re-evaluated. These surfaces are also used for individuals undergoing surgical reconstruction of a pressure injury (see the guideline chapter *Pressure Injury Surgery*).

Alternating pressure air mattresses and low air loss beds for individuals with a pressure injury

Mattresses and overlays with alternating pressure features are recommended and used by health professionals for individuals with existing pressure injuries. However, the available evidence for these support surfaces is limited and conflicting.

Evidence for alternating pressure air mattresses is conflicting and limited to studies reporting change in condition of the pressure injury using poorly described subjective scales, thus no recommendations could be made. A moderate quality Level 1 study⁹⁰ showed an alternating pressure air mattress was not significantly different to a reactive fluid overlay. A high quality Level 1 study⁶² reporting a comparison between two different alternating pressure air mattresses reported complete healing for both mattresses in 35% of Category/Stage I and II pressure injuries. Two Level 1 moderate quality studies^{79,91} comparing different alternating pressure air mattresses also found no difference between products and reported complete healing rates ranging from 35.7% (4 week follow-up) to 91.5% (19 month follow-up). A moderate quality Level 3 study⁶⁷ reported that approximately one-third of Category/Stage III pressure injuries improved in condition after 90 days with an alternating pressure air mattress. However, three low quality Level 4 studies⁹²⁻⁹⁴ reported between only 50% to 69% of Category/Stage I to III pressure injuries were classified as improved in condition after between 19 days and 7 months of follow-up. A Cochrane review⁹⁵ reported a meta-analysis of RCTs comparing alternating pressure air mattresses to standard hospital mattresses. There was no significant difference

between the support surfaces for complete pressure injury healing at four weeks (relative 0.57, 95% CI 0.26 to 1.27, $p = 0.17$) or for decrease in pressure injury size at four weeks (RR 0.99, 95% CI 0.90 to 1.09, $p = 0.31$).⁹⁵

Beds with low-air-loss features are commonly used by health professionals for individuals with existing pressure injuries; however, the evidence is also conflicting. A moderate quality Level 1 study⁹⁶ showed no difference in complete healing after 44 days compared to a foam overlay for Category II pressure injuries, and a Level 4 low quality study¹¹ reported complete healing for Category I pressure injuries of 23.8% after seven days. Moderate quality⁹⁶ and a low quality⁹⁷ Level 1 studies reported significant reduction in surface area for Category/Stage II and greater pressure injuries compared to baseline, but comparisons to control study groups was not reported. A low quality Level 1 study⁹⁸ reported no significant difference in reduction in wound size for Category/Stage II pressure injuries on a low air loss bed compared to a standard foam mattress.

Air fluidized beds for individuals with a pressure injury

Air fluidized beds are characterized by relatively high levels of envelopment and immersion compared to other support surfaces. As a result of these pressure redistributing characteristics, the users may be more restricted in terms of their ability to move on the surface while the fluidization feature is active. Some bed designs limit air fluidization to high risk zones such as sacrum in an effort to facilitate mobility. Transfers on and off the bed can be facilitated by deactivating the fluidization feature. Air fluidized beds also tend to have relatively high MVTR. These rates need to be considered relative to the needs of the individual for humidity at the skin and support surface interface.

7.10: Assess the relative benefits of using an air fluidized bed to facilitate healing while reducing skin temperature and excess hydration for individuals with Category/Stage III or IV pressure injuries. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Both a moderate quality⁹⁹ and a low quality¹⁰⁰ Level 1 study reported a significant reductions in surface area for Category/Stage III and IV pressure injuries compared with an alternating pressure air mattress used for 13 days⁹⁹ and compared to an unspecified support surface plus a sheepskin for 15 days.¹⁰⁰ A low quality Level 3 study showed statistically significantly greater healing for Category/Stage III and IV pressure injuries with an air fluidized bed compared to reactive support surfaces and compared to active support surfaces.¹⁰¹ In moderate⁹⁹ and low quality^{102,103} Level 1 studies significantly more pressure injuries in individuals with an air fluidized bed were rated as having improved in condition. In the moderate quality Level 1 study,⁹⁹ improvements in surface area and wound condition were greater in pressure injuries that were larger in size (more than 7.8 cm²) at baseline. Compared with the unspecified support surface, the moderate quality Level 1 study⁹⁹ showed an air fluidized bed was associated with a statistically significantly lower rate of pain. A low quality cost analysis¹⁰² conducted in 1991 found an air fluidized bed was associated with statistically significantly lower costs for hospital and physician care, possibly due to a lower rate of hospitalization. Another early low quality Level 1 study¹⁰³ also reported statistically significantly shorter hospital stays associated with an air fluidized bed.

Implementation Considerations

- A support surface with characteristics that redistribute pressure, reduce shear and decrease skin temperature and hydration are suggested for individuals for whom pressure cannot be relieved by repositioning (*Expert opinion*).
- Air fluidized beds have been associated with reductions in pressure injury pain compared with other specialty support surfaces⁹⁹ (*Level 1, moderate quality*).
- Air fluidized beds have been associated with dry skin¹⁰² (*Level 1*). Individuals might require increased skin moisturizing (*Expert opinion*).
- Some individuals report feelings of floating and disorientation and some individuals have difficulties repositioning themselves in an air fluidized bed. These adverse effects have been mitigated by newer air fluidized bed designs (*Expert opinion*).

Evidence Discussion

None of the studies that explored the use of air fluidized beds reported complete healing as an outcome measure; however, four small studies reported changes in surface area or wound condition.⁹⁹⁻¹⁰³ In adults who had undergone surgery and who had a pressure injury (primarily Category/Stage III or IV, $n = 65$), an air fluidized bed was associated with statistically significantly greater reduction in median wound surface area compared with an alternating pressure air mattress (-1.2cm^2 versus $+0.5\text{cm}^2$, 95% CI -9.2cm^2 to -0.6cm^2 , $p = 0.01$) over a mean of 13 days. The difference

was greater in pressure injuries that were above 7.8cm² at baseline (-5.3cm² versus +4.0cm², 95% CI -42.2 to -3.2cm², $p = 0.01$)⁹⁹ (Level 1). In another RCT conducted in hospitalized adults ($n = 45$), mean percent change in surface area for Category/Stage II or III pressure injuries at 15 days was a 43.5% reduction with an air fluidized bed versus an increase of 40% with an unspecified standard mattress plus a sheepskin ($p = 0.05$)¹⁰⁰ (Level 1). Two additional RCTs^{102,103} reported overall better outcomes with respect to surface area and wound bed characteristics for individuals receiving an air fluidized bed compared to a standard hospital mattress; however, there was minimal inter-group comparisons or statistical analysis in these studies (Level 1). The comparator mattresses in these studies were poorly defined and may not reflect contemporary support surfaces. Finally, a retrospective study conducted in older adults ($n = 664$) showed superior rates of healing for Category/Stage III and IV pressure injuries with an air fluidized bed (mean healing 3.1cm²/week) compared with reactive support surfaces (mean healing 0.6cm²/week) and compared with active support surfaces (mean healing 0.6cm²/week, $p = 0.0211$). The support surfaces used in both comparator groups were varied. For example, individuals in the comparator group received a low air loss bed or an alternating pressure air mattress or a powered or non-powered overlay. The range of different comparators made the results difficult to interpret (Level 3).

Seating Support Surfaces for Individuals with or at Risk of Pressure Injuries

When an individual is seated, their body weight is supported by a relatively small surface area (i.e., buttocks, thighs, and feet), leading to relatively high interface pressures combined with limited opportunities to redistribute body weight to other anatomical sites. Prolonged sitting results in a strong predisposition to pressure injury development, particularly in the ischial area.

7.11: Select a seat and seating support surface that meets the individual's need for pressure redistribution with consideration to:

- Body size and configuration
- Effects of posture and deformity on pressure distribution
- Mobility and lifestyle needs.

(Good Practice Statement)

Implementation Considerations

- Choose a support surface that is compatible with the care setting (*Expert opinion*).
- Some support surfaces or seating positions (e.g., tilt and recline) can reduce mobility and egress from the chair. Balance the need to prevent pressure injuries with promotion of early mobilization and activity (*Expert opinion*).
- Continue to reposition individuals regardless of the type of pressure redistribution support surface cushion being used (*Expert opinion*).
- Select a stretchable/breathable cushion cover that fits loosely on the top surface of the cushion and is capable of conforming to the body contours (*Expert opinion*).
- Before use, inspect and maintain all aspects of a seat and seating support surface to ensure proper functioning and meeting of the individual's needs. Regularly inspect other commonly used seating surfaces (e.g., travel seats, commode, shower bench, etc.) (*Expert opinion*).
- Assess the cushion and cover for heat dissipation. Select a cushion and cover that permit air exchange to minimize temperature and moisture at the buttock interface (*Expert opinion*).
- Provide training on use and maintenance of a seating support surface (including wheelchairs) and cushion devices delivered to the individual (*Expert opinion*).

Discussion

Chairs and wheelchairs

Use of a wheelchair is imperative for some individuals, most particularly those with spinal cord injury (SCI). Selection of a chair should be based on an individual assessment of functional ability and needs. Chair/wheelchair selection requires consideration to body size, configuration, posture, deformity, mobility and lifestyle needs, and should be based on an individualized assessment that includes pressure mapping.^{104,105} For individuals at risk of pressure injuries who spend significant periods of time in seated positions (e.g., those with SCI) referral to a seating specialist is recommended.

The individual's ability to weight shift in various seated positions should be assessed and considered in selection of an appropriate wheelchair/seating system. As noted in the chapter on *Repositioning and Early Mobilization*, impaired

ability to dynamic weight shift, as observed in individuals with SCI, influences pressure redistribution¹⁰⁶ (*Level 5*). This should be considered in selection of both a chair/wheelchair and cushion.

Other commonly used seating surfaces (e.g., commodes, toilets, shower bench, travel seats and recreational seating) should be reviewed to ensure they meet the individual's pressure redistribution needs (e.g., appropriate padding and well-fitted cushions). Ensure there is no specific risk to skin (e.g., from broken surfaces). All equipment should be periodically reassessed as the individual's posture and deformity, functional ability, comorbidities, preferences and needs change over time.¹⁰⁵

Seating cushions

Cushion construction achieves pressure redistribution in one of two methods: immersion/envelopment or redirection/off-loading. Envelopment is the capability of a support surface to deform around and encompass the contour of the body. Cushions that utilize envelopment must deflect and deform to immerse the buttocks in the material. Depending on the shape of the cushion, some designs will require more deflection than others to achieve the same immersion (i.e., flat cushions must deflect more than contoured cushions). The anthropometrics of the pelvis require immersion and/or a precontoured shape for load to be transferred from the inferior position of the ischial tuberosities (assuming there is no asymmetry in the pelvis) to other load bearing anatomical surfaces (e.g., buttocks and thighs). The depth of immersion and contour is in the range of 1.6 to 1.7 inches (40 to 45 mm) for most individuals.¹⁰⁷ Some cushion designs that redirect loads like this accomplish the redirection via relief areas in the cushion surface. Some require customization. Off-loading cushions generally require that the individual sit on the cushion in a specific manner. Therefore, the clinical assessment must include a determination on the individual's ability to consistently reproduce this position and confirmation that no significant functional tradeoffs occur.

A tight, non-stretch cover will adversely affect cushion performance. Covers that fit loosely on the top surface and those that are manufactured from a stretchable material are better suited to allow the cushion to deform as intended for body immersion. Evidence suggests that a rise in tissue temperature increases the susceptibility to pressure injuries.^{108,109} Evaluating heat dissipation and selecting a cushion and cover that promote air flow can reduce moisture between the cushion and skin.

Seating cushions should be inspected for signs of wear on a daily basis. The support surface (chairs and wheelchairs) should be inspected according to the manufacturer's recommendations.

7.12: Use a pressure redistribution cushion for preventing pressure injuries in people at high risk who are seated in a chair/wheelchair for prolonged periods, particularly if the individual is unable to perform pressure relieving maneuvers.

(Strength of Evidence = B1, Strength of Recommendation = ↑)

7.13: Assess the relative benefits of using an alternating pressure air cushion for supporting pressure injury healing in individuals who are seated in a chair/wheelchair for prolonged periods, particularly if the individual is unable to perform pressure relieving maneuvers.

(Strength of Evidence = B1, Strength of Recommendation = ↑)

7.14: Use a bariatric pressure redistribution cushion designed for the individuals with obesity on seated surfaces.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

The desirable effects of pressure redistribution cushions for the prevention of pressure injuries is supported by five clinical studies of moderate and low quality. A moderate quality Level 1 study¹¹⁰ suggested individuals at high risk of pressure injuries may experience fewer ischial pressure injuries when seated on either an air cushion, a viscous fluid and foam cushion or a gel and foam cushion rather than a standard foam cushion, but there was no significant difference in pressure injury incidence when sacral pressure injuries were included.¹¹⁰ These results were supported by a low quality Level 1 study comparing a pressure redistribution cushion to a standard foam cushion.¹¹¹ A low quality Level 2 study reported a statistically significant reduction in overall pressure injury incidence with a pressure redistribution

cushion compared to standard foam. One Level 4 study comparing outcomes for single- and multi-compartment air cushions reported low pressure injury incidence rates (less than 5%).¹¹² However, in another low quality Level 4 study the rate of Category/Stage I pressure injuries in individuals seated on pressure redistributing cushion consisting of memory foam plus a gel was 33%.¹¹³ Variation in the research findings may relate to the quality of the chair on which the cushion was placed and the frailty of the individuals included in the studies.¹¹⁴

There is direct evidence from one low quality Level 1 study¹¹⁵ that revealed some improvements in measures of pressure injury healing with an alternating pressure air cushion compared to a standard seating cushion plus using pressure relieving maneuvers. The improvements included reaching 30% closure of a pressure injury in a mean of 5 days faster as well as faster wound closure, but did not extend to significantly greater rates of complete pressure injury closure overall. The intervention required a full seating system that was described in the study as potentially having low cost-effectiveness and accessibility may be limited.

There is indirect evidence to suggest that individuals with obesity experience stresses and forces that increase tissue loading, particularly on a harder seating surface.^{116,117} The indirect evidence suggests tissue loading is lower on a softer seating surface,¹¹⁸ and that an air cell-based cushion reduces the increase in fat/tissue strain experienced by individuals with obesity. It is feasible that a pressure-redistributing seating surface that reduces tissue loading would lead to a reduction in risk for pressure injuries; however, there is no direct evidence that demonstrates this empirically.

Implementation Considerations

- Refer individuals at high risk of pressure injuries who spend prolonged periods in a chair/wheelchair to a seating specialist (*Expert opinion*).
- Advise individuals who spend prolonged periods in a chair/wheelchair to perform regular off-loading maneuvers (*Expert opinion*).
- Perform regular skin and risk assessments for individuals who spend prolonged periods sitting in a chair/wheelchair (*Expert opinion*).
- Regularly inspect air cushions and their covers for signs of wear and tear¹¹⁹ (*Expert opinion*).
- Evaluate the stability of wheelchair users who are seated on air cushions. Weigh the benefits of off-loading against the potential for instability and shear based on the construction and operation of the cushion (*Expert opinion*).
- Advise individuals who spend prolonged periods in a wheelchair with a pressure redistribution cushion to use a pressure redistribution with other seating, for example when traveling (e.g., in motor vehicle, airplane or train).¹²⁰
- In individuals with a pressure injury, take psychosocial needs into account in balancing periods of bed rest and sitting in a chair/wheelchair (*Expert opinion*).
- In individuals with a pressure injury, assess the pressure injury for deterioration related to time spent seated in a chair/wheelchair and adjust the amount of time spent in the chair accordingly (*Expert opinion*).

Evidence Discussion

Two studies^{110,111} found significant reduction in ischial tuberosity pressure injury incidence associated with pressure redistribution cushions, although neither study demonstrated a statistically significant reduction in overall (i.e., ischial tuberosity plus sacral, coccyx and buttock) pressure injury incidence. Brienza et al. (2010)¹¹⁰ conducted an RCT with individuals living in a nursing home (n = 180) over a six month period. Participants were all provided with a fitted wheelchair and randomized into skin protection seated on an air, viscous fluid and foam cushion, a gel and foam cushion or a high specification foam cushion (n = 113) or a control receiving a standard foam cushion (n = 119). The experimental group experienced a significantly lower incidence of ischial tuberosity pressure injuries (0.0% versus 6.7%, p = 0.04). However, when the analysis combined ischial tuberosity and sacral pressure injuries, the incidence was not significantly different between groups (17.6% control versus 10.6% experimental, p = 0.14). Kaplan Meier methods did not demonstrate significant differences in the cumulative incidence of pressure injuries between the groups.¹¹⁰ There was no control for conditions outside of chair time and frequency of repositioning was not reported (*Level 1*). Geyer et al. (2001)¹¹¹ conducted a small, pilot RCT involving 32 elderly nursing home residents who could tolerate six hours daily sitting in a wheelchair. The experimental group (n = 15) received a pressure reducing cushion (type not reported clearly), and the control group (n = 17) received a foam cushion. In total, 50% of study participants developed pressure injuries, with no significant differences between the groups. However, as with the study by Brienza et al. (2010),¹¹⁰ the ischial tuberosity pressure injuries incidence was significantly lower in the pressure redistribution cushion group (p < 0.005)¹¹¹ (*Level 1*).

Collins (1999)¹²¹ performed a controlled trial involving older adults in acute care (n = 40). The experimental group had armchairs with pressure redistribution cushions, padded armrests, and side wings to support the head, and the control group had standard armchairs with foam on the seat. The experimental group developed significantly fewer pressure injuries (p < 0.0001).¹²¹ The importance of adequate postural support to prevent shear in coccyx, sacrum and buttocks should be noted (*Level 2*).

Defloor and Grypdonck (2000)¹²² investigated different types of cushion including air, water, hollow fiber, foam, combination gel and foam, and sheepskin (n = 28 cushions) in a laboratory study involving healthy volunteers. Interface pressure was measured after one hour of immobilization. When cushions were combined according to type, the air cushion category had the lowest interface pressure (t = -6.40, 95% CI -9.17 to -4.65, p < 0.01 versus armchair with no cushion). However, water cushions and foam cushions did not differ significantly to air cushions. Within the foam cushion category (n = 9 cushions) there was a significant difference between the various cushion types, with the two visco-elastic foam cushions having maximum interface pressures approximately 38% higher than the armchair with no cushion (p < 0.01). Cushions with the lowest maximum interface pressure were described by the manufacturers as polyethylene-urethane (7 cm/2.75 inches; 85 kg/m³), polymer (no specifications), vinyl (no specifications) and shock absorbing polyester foam (60 kg/m³). Many of the gel cushions, combination cushions and the synthetic sheepskin had negligible impacts on interface pressures (all p = ns versus armchair)¹²² (Level 5).

Alternating pressure air cushions

Alternating pressure air cushions have been used in many clinical settings.¹²³ A study by Burns and Betz (1999)¹²⁴ concluded that there is a similar relief in pressure over the ischial tuberosities between a dynamic cushion during the low pressure phase compared with a tilt-in-space wheelchair with a conventional cushion. However, individual responses to the high pressure phase may vary (Level 5).

Wheelchairs equipped with an individually adjusted automated seat providing cyclic pressure relief using a protocol of ten minutes normal sitting and ten minutes offloaded sitting may enhance pressure injury closure and decrease wound surface area. An RCT (n = 44) conducted by Makhsous et al. (2009)¹¹⁵ found significantly more improvement in pressure injury area closure and Pressure Scale for Healing (PUSH) score in individuals using an automated, cyclic relief seat compared with individuals in a standard wheelchair who performed arm push-ups for pressure relief every 20 or 30 minutes. The group using the cyclic pressure relief seating system achieved a mean 45 ± 21% improvement in mean pressure injury surface area compared with 10.2 ± 34.8% improvement in the control group (p < 0.001).¹¹⁵ As the study did not address possible differences between groups in preventive measures provided when the individuals were not seated, differences in wound care/dressings, and pressure injury size at baseline, it was not possible to recommend an adjusted automated seat above a standard wheelchair with a manual pressure relief regimen (Level 1).

Seating for individuals with obesity

Biomechanical modeling studies suggest an increased risk of suspected deep tissue injury in the seated obese individual. In a biomechanical modeling investigation, Elsner and Gefen (2008)¹¹⁶ used finite element models to demonstrate that a higher BMI is associated with an increase in internal muscle tissue load under the ischial tuberosities. Sopher et al. (2010)¹¹⁷ continued this investigation using finite element models representing the same individual modeled with BMIs ranging from less than 16.5 kg/m² up to 40 kg/m². The study results showed that the percentage volume of muscle tissue under the ischial tuberosities increased over five times as BMI increased from 19 kg/m² to 40 kg/m² (Level 5).

In laboratory modeling, increases on internal muscle load were shown to be of a greater magnitude when sitting on a hard surface compared with a soft chair.^{116 116} Levy et al. (2016)¹¹⁸ established that average strain and stress in fat and skin tissues were mildly decreased when modeling individuals with normal to obese BMI sitting on an air cell-based (ACB) cushion. The effect was more pronounced modeling individuals with diabetes and the same range of BMI. The ACB has the potential to protect tissues of individuals with BMI of 30kg/m² by preventing strain and stress in fat and skin tissues from exceeding a 20% increase above individuals with a normal range BMI (Level 5).

Support Surface Use During Transportation

Individuals may be at risk for pressure injury in all circumstances where they are in contact with a support surface and have a degree of immobility or inactivity. This includes vehicle transportation and while waiting for a clinical review and/or admission in the emergency department.

7.15: For individuals with or at risk for a pressure injury, consider using a pressure redistributing support surface during transit.
(Good Practice Statement)

7.16: Transfer the individual off a spinal hard board/back board as soon as feasible after admission to an acute care facility in consultation with a qualified health professional.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

A high quality Level 4 study¹²⁵ reported an incidence of pressure injuries for individuals with suspected cervical spine injuries who remained on a spinal hardboard for a median of four hours as 28.3%. Indirect evidence suggests that sacral tissue oxygenation is significantly lower after 30 minutes immobilization on a rigid long board.¹²⁶ Although indirect evidence^{127,128} suggests that using a vacuum spine board or a gel overlay is associated with lower interface pressure than a spinal hardboard, a small high quality Level 3 study found no significant difference in pressure injury incidence.¹²⁹

Implementation Considerations

- Transfer individuals with suspected spinal injury using protective equipment deemed appropriate by local policies and procedures.^{130,131} In most instances, an ambulance transport stretcher used in conjunction with straps provides sufficient spinal protection and adequately restricts the individual's spinal motion, particular when the individual is conscious and cooperative¹³⁰ (*Expert opinion*).

Evidence Discussion

Undertaking a comprehensive pressure injury risk assessment during transit is often not possible, particularly in the emergency vehicle when the care team have competing priorities (e.g., respiratory and cardiac stabilization). Using a pressure redistributing support surface for all individuals in transit or, if pressure injury risk screening has been possible, for those screened as having a possible pressure injury risk, is suggested (see *Recommendation 1.21* on risk screening). Initiating other preventive interventions (e.g., prophylactic dressings) as early as possible in the care pathway when feasible has been demonstrated to contribute to a reduction in pressure injuries¹³² (see *Recommendation 6.4* in the guideline chapter *Heel Pressure Injuries*).

Historically, individuals with suspected spinal cord injury (SCI) have been managed prior to hospitalization with an extrication collar and long spine board or spinal back board to restrict spinal motion. Restriction of spinal motion (particularly on a long spine board) is associated with increased adverse events, including pressure injuries (also see *Recommendation 8.8* related to cervical collar use in the guideline chapter *Device Related Pressure Injuries*). A survey evaluating pressure injury incidence in individuals with suspected SCI (n = 254) admitted to an emergency department with a backboard and extrication collar had a pressure injury incidence of 28.3% (95% confidence interval [CI] 22.8% to 34.3%), of which 21.1% were Category/Stage IV pressure injuries. The buttocks (42.1%) and heels (33.4%) were the most common anatomical locations for pressure injuries¹²⁵ (*Level 4*). Another study has shown that restriction of spinal motion using a back board is associated with a decrease in sacral tissue oxygen saturation (*Level 5*).

While a number of small studies have made comparisons between a rigid spine back board and alternative support surfaces, there is insufficient evidence to make specific recommendations. No identified studies made comparisons between a long spine board and other motion restriction techniques for reducing pressure injuries; however, a recent position paper of the National Association of Emergency Medical Service (EMS) Physicians¹³⁰ recommended that a long spine board be avoided for transportation of most individuals with suspected SCI due to the high risk of adverse events. The American College of Emergency Physicians¹³¹ has recommended judicious use of spinal motion restriction based on validated indicators. Indirect evidence in healthy volunteers (n = 42) showed that a padded spine board was as effective as a long spine board for immobilizing the head, and the reduced effectiveness in immobilizing the pelvis and sternum was not considered to be clinically significant¹³³ (*Level 5*).

Comparisons between a rigid spine back board and a vacuum spine board used for intercontinental air transportation of individuals with suspected spinal injuries showed no significant difference in pressure injury incidence (13% on a rigid board versus 10% on a vacuum board, p = 0.70)¹²⁹ (*Level 3*). The presumed longer transportation durations may reduce the generalizability of these results. Indirect evidence from trials conducted in healthy volunteers has shown significantly lower interface pressures associated with both a vacuum mattress¹²⁸ and a viscous gel overlay¹²⁷ compared to a rigid spine back board (*Level 5*). Until further research is available to guide practice in transporting individuals immobilized due to suspected SCI, ensure individuals admitted on a rigid spinal board are transferred off the board as soon as an appropriately qualified health professional deems the transfer to be safe.

References

1. NPUAP. *National Pressure Ulcer Advisory Panel Support Surface Standards Initiative - Terms and Definitions Related to Support Surfaces*. 2007; Available from: http://www.npuap.org/NPUAP_S3I_TD.pdf.
2. RESNA (2019). *American National Standard for Support Surfaces - Volume 1: Requirements and Test Methods for Full Body Support Surfaces (ANSI/RESNA SS-1:2019)*. 2019; Available from: <https://www.resna.org/standards/support-surfaces/support-surfaces>.
3. Tissue Viability Society. Laboratory measurement of the interface pressures applied by active therapy support surfaces: A consensus document. *Journal of Tissue Viability*, 2010; 19(1): 2-6.

4. British Standards, BS ISO 16840-10:2014 Wheelchairs. Resistance to ignition of non-integrated seat and back support cushions. Requirements and test methods. 2014: <https://doi.org/10.3403/30280575U>.
5. Rithalia S, Kenny L. Review Hospital bed mattresses: an overview of technical aspects. *J Med Eng Technol*, 2003; 24(1): 32-39.
6. Vilhena L, Ramalho A. Friction of human skin against different fabrics for medical use. *Lubricants*, 2016 4(1): doi.org/10.3390/lubricants4010006
7. Klaassen M, Schipper D, Masen M. Influence of the relative humidity and the temperature on the in-vivo friction behaviour of human skin. *Biotribology*, 2016; 6: 21-28.
8. Futamura M, Sugama J, Okuwa M, Sanada H, Tabata K. Evaluation of comfort in bedridden older adults using an air-cell mattress with an automated turning function: Measurement of parasympathetic activity during night sleep. *J Gerontol Nurs*, 2008; 34(12): 20-26.
9. McNabb LJ, Hyatt J. Effect of an air-fluidized bed on insensible water loss. *Crit Care Med*, 1987; 15(2): 161-162.
10. Wiggermann N, Smith K, Kumpar D. What bed size does a patient need? The relationship between body mass index and space required to turn in bed. *Nurs Res*, 2017; 66(6): 483-489.
11. Pemberton V, Turner V, VanGilder C. The effect of using a low-air-loss surface on the skin integrity of obese patients: Results of a pilot study. *Ostomy Wound Management*, 2009; 55(2): 44-48.
12. Bain DS, Ferguson-Pell MW, Davies P. An instrument for in-service testing of mattresses. *J Tissue Viability*, 2001; 11(4): 161-165.
13. Heule EJC, Goossens RHM, Mugge R, Dietz E, Heule F. Using an indentation measurement device to assess foam mattress quality. *Ostomy Wound Management*, 2007; 53(11): 56-62.
14. Yim GH, Clark M, Gray D, Stephen-Haynes J, Jeffery SLA. The hazards of dynamic airflow mattresses and overlays. *Burns*, 2014; 40(4): 782-783.
15. Greater Manchester Fire and Rescue Service. *Dynamic Air Flow Pressure Relieving Mattresses Information and Fire Prevention Guidance for Partner Organisations*. Available from: <https://www.rbsab.org/UserFiles/Docs/Dynamic%20Air%20Flow%20Pressure%20Relieving%20Mattresses%20Guidance%20for%20partners.pdf>.
16. International Organization for Standardization, ISO 20342-1:2019. Assistive products for tissue integrity when lying down — Part 1: General requirements. 2019: <https://www.iso.org/standard/67750.html>.
17. Williamson R, Lachenbruch C, Vangilder C. The effect of multiple layers of linens on surface interface pressure: Results of a laboratory study. *Ostomy Wound Management*, 2013; 59(6): 38-48.
18. Fader M, Bain DS, Cottenden A. Effects of absorbent incontinence pads on pressure management mattresses. *Journal Of Advanced Nursing*, 2004; 48(6): 569-574.
19. Park KH, Park J. The efficacy of a viscoelastic foam overlay on prevention of pressure injury in acutely ill patients: A prospective randomized controlled trial. *Journal of Wound, Ostomy, and Continence Nursing*, 2017; 44(5): 440-444.
20. Russell LJ, Reynolds TM, Park C, Rithalia S, Gonsalkorale M, Birch J, Torgerson D, Iglesias C, Group. P-S. Randomized clinical trial comparing 2 support surfaces: Results of the Prevention of Pressure Ulcers Study. *Adv Skin Wound Care*, 2003; 16(6): 317-327.
21. van Leen M, Halfens R, Schols J. Preventive effect of a microclimate-regulating system on pressure ulcer development: A prospective, randomized controlled trial in Dutch nursing homes. *Adv Skin Wound Care*, 2018; 31(1): 1-5.
22. van Leen M, Hovius S, Neyens J, Halfens R, Schols J. Pressure relief, cold foam or static air? A single center, prospective, controlled randomized clinical trial in a Dutch nursing home. *Journal of Tissue Viability*, 2011; 20(1): 30-34.
23. Gray D, Campbell M. A randomised clinical trial of two types of foam mattresses. *Journal of Tissue Viability*, 1994; 4(4): 128-132.
24. Hofman A, Geelkerken RH, Wille J, Hamming JJ, Hermans J, Breslau PJ. Pressure sores and pressure-decreasing mattresses: Controlled clinical trial. *Lancet*, 1994; 343(8897): 568-71.
25. Collier ME. Pressure-reducing mattresses. *J Wound Care*, 1996; 5(5): 207-211.
26. Berthe JV, Bustillo A, Melot C, de Fontaine S. Does a foamy-block mattress system prevent pressure sores? A prospective randomised clinical trial in 1729 patients. *Acta Chir Belg*, 2007; 107(2): 155-161.
27. Gray DG, Smith M. Comparison of a new foam mattress with the standard hospital mattress. *J Wound Care*, 2000; 9(1): 29-31.
28. Ozyurek P, Yavuz M. Prevention of pressure ulcers in the intensive care unit: A randomized trial of 2 viscoelastic foam support surfaces. *Clin Nurse Spec*, 2015; 29(4): 210-7.
29. Ricci E, Roberto C, Ippolito A, Bianco A, Scalise MT. *A randomized study on the effectiveness of a new pressure-relieving mattress overlay for the prevention of pressure ulcers in elderly patients at risk*. *EWMA journal*, 2013. 13, 27-32.
30. Stapleton M. Preventing pressure sores -- An evaluation of three products...foam, ripple pads, and Spenco pads. *Ger Nurs*, 1986; 6(2): 23-5.
31. Turnage-Carrier C, McLane KM, Gregurich MA. Interface pressure comparison of healthy premature infants with various neonatal bed surfaces. *Adv Neonatal Care*, 2008; 8(3): 176-184.
32. McInnes E, Jammali-Blasi A, Bell-Syer SEM, Dumville JC, Middleton V, Cullum N. Support surfaces for pressure ulcer prevention. *Cochrane Database Syst Rev*, 2015; 9(CD001735).
33. Van Leen M, Hovius S, Halfens R, Neyens J, Schols J. Pressure relief with visco-elastic foam or with combined static air overlay? A prospective, crossover randomized clinical trial in a Dutch nursing home. *Wounds*, 2013; 25(10): 287-292.
34. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
35. Standards Australia, Committee PL/36 on Flexible Polyurethane, Australian Standard® AS2281-1993 Flexible cellular polyurethane for seat cushioning and bedding. 1993: Homebush, NSW.
36. Polyurethane Foam Association. *Joint Industry Foam Standards And Guidelines, Section 1*. 1994 [cited 2013]; Available from: <http://www.pfa.org/jifsg/jifsgs1.html>.
37. British Standard, Hospital bedding - Part 2: Combustion modified, flexible polyurethane, general purpose foam mattress cores - specification, BS 5223-2. 1999.
38. Polyurethane Foam Association. *Joint Industry Foam Standards And Guidelines, Section 4*. 1994 [cited 2013]; Available from: <http://www.pfa.org/jifsg/jifsgs4.html>.

39. Brienza D, Geyer M. Understanding support surface technologies. *Advances in Skin & Wound Care*, 2000; 13(5): 237-243.
40. Thomas S, *Surgical Dressings and Wound Management*. 2010, Great Britain: Medetec Publications.
41. Clubb M. Water vapour permeable materials for mattress coverings. *Journal of Tissue Viability*, 1998; 8(1): 12-14.
42. Quigley S, Curley M. Skin integrity in the pediatric population: Preventing and managing pressure ulcers. *J Spec Pediatr Nurs*, 1996; 1(1): 7-18.
43. Razmus I, Lewis L, Wilson D. Pressure ulcer development in infants: State of the Science. *J Healthc Qual*, 2008; 30(5): 36-42.
44. Fujii K, Sugama J, Okuwa M, Sanada H, Mizokami Y. Incidence and risk factors of pressure ulcers in seven neonatal intensive care units in Japan: a multisite prospective cohort study. *Int Wound J*, 2010; 7(5): 323-328.
45. Schindler CA, Mikhailov TA, Kuhn EM, Christopher J, Conway P, Ridling D, Scott AM, Simpson VS. Protecting fragile skin: Nursing interventions to decrease development of pressure ulcers in pediatric intensive care. *Am J Crit Care*, 2011; 20(1): 26-35.
46. Vermette S, Reeves I, Lemaire J. Cost effectiveness of an air-inflated static overlay for pressure ulcer prevention: A randomized, controlled trial. *Wounds: A Compendium of Clinical Research & Practice*, 2012; 24(8): 207-214.
47. Lazzara DJ, Buschmann MBT. Prevention of pressure ulcers in elderly nursing home residents: Are special support surfaces the answer? . *Decubitus*, 1991; 4(4): 42-6.
48. Sideranko S, Quinn A, Burns K, Froman RD. Effects of position and mattress overlay on sacral and heel pressures in a clinical population. . *Res Nurs Health*, 1992; 15(4): 245-51.
49. Takala J, Varmavuo S, Soppi E. Prevention of pressure sores in acute respiratory failure: A randomised controlled trial. *Clin Intensive Care*, 1996; 7(5): 228-235.
50. García-Molina P, Balaguer-López E, Torra I Bou JE, Alvarez-Ordiales A, Quesada-Ramos C, Verdú-Soriano J. A prospective, longitudinal study to assess use of continuous and reactive low-pressure mattresses to reduce pressure ulcer incidence in a pediatric intensive care unit. *Ostomy Wound Management*, 2012; 58(7): 32-39.
51. Serraes B, Beeckman D. Static air support surfaces to prevent pressure injuries: A multicenter cohort study in Belgian nursing homes. *Journal of Wound, Ostomy, & Continence Nursing*, 2016; 43(4): 375-8.
52. Newton H. A 6 month evaluation of a non-powered hybrid mattress replacement system. *Br J Nurs*, 2015; 24 (Suppl 20): S32-S36.
53. Mistiaen P, Achterberg W, Ament A, Halfens R, Huizinga J, Montgomery K, Post H, Spreeuwenberg P, Francke AL. The effectiveness of the Australian Medical Sheepskin for the prevention of pressure ulcers in somatic nursing home patients: a prospective multicenter randomized-controlled trial (ISRCTN17553857). *Wound Repair Regen*, 2010; 18(6): 572-579.
54. Jolley DJ, Wright R, McGowan S, Hickey MB, Campbell DA, Sinclair RD, Montgomery KC. Preventing pressure ulcers with the Australian Medical Sheepskin: An open-label randomised controlled trial. *Med J Aust*, 2004; 180(7): 324-327.
55. McGowan S, Montgomery K, Jolley D, Wright R. The role of sheepskins in preventing pressure ulcers in elderly orthopaedic patients. *Primary Intention*, 2000; 8: 127-134.
56. Sauvage P, Touflet M, Pradere C, Portalier F, JMichel J-M, Charru P, Passadori Y, Fevrier R, Hallet-Lezy A-M, Beauchêne F, Scherrer B. Pressure ulcers prevention efficacy of an alternating pressure air mattress in elderly patients: E²MAO a randomised study. *J Wound Care*, 2017; 26(6): 304-312.
57. Vanderwee K, Grypdonck MH, Defloor T. Effectiveness of an alternating pressure air mattress for the prevention of pressure ulcers. *Age Ageing*, 2005; 34(3): 261-267.
58. Sanada H, Sugama J, Matsui Y, Konya C, Kitagawa A, Okuwa M, Omote S. Randomised controlled trial to evaluate a new double-layer air-cell overlay for elderly patients requiring head-elevation. *Journal of Tissue Viability*, 2003; 13(3): 112-118.
59. Fletcher J, Tite M, Clark M. Real-world evidence from a large-scale multisite evaluation of a hybrid mattress. *Wounds UK*, 2016; 12(3): 54-61.
60. Gleeson D. Evaluating an alternating mattress on an elderly rehabilitation ward. *Br J Nurs*, 2015; 24(12): S42, S44-7.
61. Gleeson D. Effectiveness of a pressure-relieving mattress in an acute stroke ward. *Br J Nurs*, 2016; 25(20): S34-S38.
62. Nixon J, Cranny G, Iglesias C, Nelson EA, Hawkins K, Phillips A, Torgerson D, Mason S, Cullum N. Randomised, controlled trial of alternating pressure mattresses compared with alternating pressure overlays for the prevention of pressure ulcers: PRESSURE (pressure relieving support surfaces) trial. *Br Med J*, 2006; 332(7555): 1413-1413.
63. Demarré L, Beeckman D, Vanderwee K, Defloor T, Grypdonck M, Verhaeghe S. Multi-stage versus single-stage inflation and deflation cycle for alternating low pressure air mattresses to prevent pressure ulcers in hospitalised patients: A randomised-controlled clinical trial. *Int J Nurs Stud*, 2012; 49(4): 416-426.
64. Manzano F, Pérez AM, Colmenero M, Aguilar MM, Sánchez-Cantalejo E, Reche AM, Talavera J, López F, Barco SFD, Fernández-Mondejar E. Comparison of alternating pressure mattresses and overlays for prevention of pressure ulcers in ventilated intensive care patients: A quasi-experimental study. *J Adv Nurs*, 2013; Epub 2013 Jan 24.
65. Nixon J, Nelson EA, Cranny G, Iglesias CP, Hawkins K, Cullum NA, Phillips A, Spilsbury K, Torgerson DJ, Mason S, PRESSURE Trial Group. Pressure relieving support surfaces: a randomised evaluation. *Health Technol Assess*, 2006; 10(22): iii-x, 1.
66. Iglesias C, Nixon J, Cranny G, Nelson EA, Hawkins K, Phillips A, Torgerson D, Mason S, Cullum N. Pressure relieving support surfaces (PRESSURE) trial: Cost effectiveness analysis. *BMJ*, 2006; 332: 1416.
67. Meaume S, Marty M. Pressure ulcer prevention and healing using alternating pressure mattress at home: The PARESTRY project. *J Wound Care*, 2015; 24(8): 359-365.
68. Demarré L, Verhaeghe S, Van Hecke A, Grypdonck M, Clays E, Vanderwee K, Beeckman D. The effectiveness of three types of alternating pressure air mattresses in the prevention of pressure ulcers in Belgian hospitals. *Res Nurs Health*, 2013; 36(5): 439-452.
69. Law J. Transair® paediatric mattress replacement system evaluation. *Br J Nurs*, 2002; 11(5): 343-346.
70. Bennett R, Baran P, DeVone L, Bacetti H, Kristo B, Tayback M, Greenough Wr. Low airloss hydrotherapy versus standard care for incontinent hospitalized patients. *J Am Geriatr Soc*, 1998; 46(5): 569-576.
71. Johnson J, Peterson D, Campbell B, Richardson R, Rutledge D. Hospital-acquired pressure ulcer prevalence--Evaluating low-air-loss beds *J Wound Ostomy Cont Nurs*, 2011; 38(1): 55-60.
72. Inman KJ, Sibbald WJ, Rutledge FS, Clark BJ. Clinical utility and cost-effectiveness of an air suspension bed in the prevention of pressure ulcers. *JAMA*, 1993; 269(9): 1139-43.

73. Black J, Berke C, Urzandowski G. Pressure ulcer incidence and progression in critically ill subjects: Influence of low air loss mattress versus a powered air pressure redistribution mattress. *J Wound Ostomy Cont Nurs*, 2012; 39(3): 267-73.
74. McLane K, Krouskop TA, McCord S, Fraley K. Comparison of interface pressures in the pediatric population among various support surfaces. *J Wound Ostomy Continence Nurs*, 2002; 28: 242-251.
75. Nixon J, McElvenny D, Mason S, Brown J, Bond S. A sequential randomised controlled trial comparing a dry visco-elastic polymer pad and standard operating table mattress in the prevention of post-operative pressure sores. *Int J Nurs Stud*, 1998; 35(4): 193-203.
76. Wu T, Wang ST, Lin PC, Liu CL, Chao YF. Effects of using a high-density foam pad versus a viscoelastic polymer pad on the incidence of pressure ulcer development during spinal surgery. *Biological Research For Nursing*, 2011; 13(4): 419-424.
77. Feuchtinger J, de Bie R, Dassen T, Halfens R. A 4-cm thermoactive viscoelastic foam pad on the operating room table to prevent pressure ulcer during cardiac surgery. *Journal of Clinical Nursing*, 2006; 15(2): 162-167.
78. Aronovitch SA, Wilber M, Slezak S, Martin T, Utter D. A comparative study of an alternating air mattress for the prevention of pressure ulcers in surgical patients. *Ostomy Wound Management*, 1999; 45(3): 34-44.
79. Russell JA, Lichtenstein SL. Randomized controlled trial to determine the safety and efficacy of a multi-cell pulsating dynamic mattress system in the prevention of pressure ulcers in patients undergoing cardiovascular surgery. *Ostomy Wound Management*, 2000; 46(2): 46-45.
80. Defloor T, De Schuijmer JD. Preventing pressure ulcers: an evaluation of four operating-table mattresses. *Appl Nurs Res*, 2000; 13(3): 134-141.
81. Scott EM, Baker EA, Kelly PJ, Stoddard EJ, Leaper DJ. Measurement of interface pressures in the evaluation of operating theatre mattresses. *J Wound Care*, 1999; 8(9): 437-441.
82. Smith IL, Brown S, McGinnis E, Briggs M, Coleman S, Dealey C, Muir D, Nelson EA, Stevenson R, Stubbs N, Wilson L, Brown JM, Nixon J. Exploring the role of pain as an early predictor of category 2 pressure ulcers: A prospective cohort study. *BMJ Open*, 2017; 7(1): e013623.
83. Reed R, Hepburn K, Adelson R, Center B, McKnight P. Low serum albumin levels, confusion, and fecal incontinence: Are these risk factors for pressure ulcers in mobility-impaired hospitalized adults? *Gerontology*, 2003; 49(4): 255-59.
84. Nixon J, Cranny G, Bond S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: A cohort study. *Int J Nurs Stud*, 2007; 44(5): 655-63.
85. Demarre L, Verhaeghe S, Van Hecke A, Clays E, Grypdonck M, Beeckman D. Factors predicting the development of pressure ulcers in an at-risk population who receive standardized preventive care: Secondary analyses of a multicentre randomised controlled trial. *J Adv Nurs*, 2015; 71(2): 391-403.
86. Allman R, Goode P, Patrick M, Burst N, Bartolucci A. Pressure ulcer risk factors among hospitalized patients with activity limitation. *J Am Med Assoc*, 1995; 273(11): 865-70.
87. Bergstrom N, Bennett, M.A., Carlson, C.E., et al, *Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. AHCPH Pub. No. 95-0653*. 1994, Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, Agency for Healthcare Policy and Research.
88. Cassino R, Ippolito AM, Cuffaro C, Corsi A, Ricci E. A controlled, randomised study on the efficacy of two overlays in the treatment of decubitus ulcers. *Minerva Chir*, 2013; 68(1): 105-16.
89. Valente SA, Greenough III WB, DeMarco SL, Andersen RE. More expensive surfaces are not always better. *Kuwait Med J*, 2012; 44(1): 40-45.
90. Russell L, Reynolds TM, Towns A, Worth W, Greenman A, Turner R. Randomized comparison trial of the RIK and the Nimbus 3 mattresses. *Br J Nurs*, 2003; 12(4): 254-259.
91. Devine B. Alternating pressure air mattresses in the management of established pressure sores. *J Tissue Viability*, 1995; 5: 94-8.
92. Ward L, Fenton K, Maher L. The high impact actions for nursing and midwifery 4: Your skin matters. *Nurs Times*, 2010; 106(30): 14-15.
93. Fletcher J, Harris C, Mahoney K, Crook H, Moore JO. A small-scale evaluation of the Dolphin Fluid Immersion Simulation® mattress. *Wounds UK*, 2014; 10(1): 97-100.
94. Stephen-Haynes J, Callaghan R. A 100 patient clinical evaluation of an alternating pressure replacement mattress in a home-based setting. *Br J Nurs*, 2017; 26: 554-560.
95. McInnes E, Jammali-Blasi A, Bell-Syer SEM, Leung V. Support surfaces for treating pressure ulcers. *Cochrane Database Syst Rev*, 2018(Issue 10. Art. No.: CD009490. DOI: 10.1002/14651858.CD009490.pub2).
96. Ferrell BA, Osterweil D, Christenson P. A randomized trial of low-air-loss beds for treatment of pressure ulcers. *J Am Med Assoc*, 1993; 269(4): 494-7.
97. Mulder GD, Taro N, Seeley J, Andrews K. A study of pressure ulcer response to low air loss beds vs. conventional treatment. *Journal of Geriatric Dermatology*, 1994; 2(3): 87-91.
98. Day A, Leonards F. Seeking quality care for patients with pressure ulcers. *Decubitus*, 1993; 6(1): 32-43.
99. Allman RM, Walker JM, Hart MK, Laprade CA, Noel LB, Smith CR. Air-fluidized beds or conventional therapy for pressure sores. A randomized trial. *Ann Intern Med*, 1987; 107(5): 641-8.
100. Munro BH, Brown L, Heitman BB. Pressure ulcers: One bed or another? How does an air-fluidized bed compare with pads and other devices on a standard bed? *Geriatric Nursing* 1989; 10: 190-2.
101. Ochs RF, Horn SD, van Rijswijk L, Pietsch C, Smout RJ. Comparison of air-fluidized therapy with other support surfaces used to treat pressure ulcers in nursing home residents. *Ostomy Wound Management*, 2005; 51(2): 38-68.
102. Strauss MJ, Gong J, Gary BD, Kalsbeek WD, Spear S. The cost of home air-fluidized therapy for pressure sores. A randomized controlled trial. *The Journal of Family Practice*, 1991; 33(1): 52-59.
103. Jackson BS, Chagares R, Nee N, Freeman K. The effects of a therapeutic bed on pressure ulcers: An experimental study. *J Enterostomal Ther*, 1988; 15(6): 220-226.
104. Regan M, Teasell R, Keast D, Aubut J, Foulon B, Mehta S, Pressure Ulcers Following Spinal Cord Injury. 2010, Spinal Cord Injury Rehabilitation Evidence (SCIRE): Vancouver, BC.

105. Houghton PE, Campbell KE, CPG Panel, Canadian Best Practice Guidelines for the Prevention and Management of Pressure Ulcers in People with Spinal Cord Injury. A resource handbook for clinicians. 2013, Ontario Neurotrauma Foundation: https://onf.org/wp-content/uploads/2019/04/Pressure_Ulcers_Best_Practice_Guideline_Final_web4.pdf.
106. Karatas GK, Tosun A, Kanatl U. Center-of-pressure displacement during postural changes in relation to pressure ulcers in spinal cord-injured patients. *Am J Phys Med Rehabil*, 2008; 87(3): 177-182.
107. Sprigle S, Press L. Reliability of the iso wheelchair cushion test for loaded contour depth. *Assist Technol*, 2003; 15(2): 145-150.
108. Patel S, Knapp CF, Donofrio JC, Salcido R. Temperature effects on surface pressure-induced changes in rat skin perfusion: Implications in pressure ulcer development. *J Rehabil Res Dev*, 1999; 36(3): 189-201.
109. Kokate JY, Leland KJ, Held AM, Hansen GL, Kveen GL, Johnson BA, Wilke MS, Sparrow EM, Iazzo PA. Temperature-modulated pressure ulcers: a porcine model. *Arch Phys Med Rehabil*, 1995; 76(7): 666-673.
110. Brienza D, Kelsey S, Karg P, Allegretti A, Olson M, Schmeler M, Zanca J, Geyer MJ, Kusturiss M, Holm M. A randomized clinical trial on preventing pressure ulcers with wheelchair seat cushions. *J Am Geriatr Soc*, 2010; 58(12): 2308-2314.
111. Geyer M, Brienza D, Karg P, Trefler E, Kelsey S. A randomized control trial to evaluate pressure-reducing seat cushions for elderly wheelchair users. *Adv Skin Wound Care*, 2001; 14: 120-129.
112. Meaume S, Marty M, Colin D. Prospective observational study of single- or multi-compartment pressure ulcer prevention cushions: PRESCAROH project. *J Wound Care*, 2017; 26(9): 537-544.
113. Mossman B, Hampton S. Effectiveness of a pressure-redistributing cushion for low- to medium-risk patients in care homes. *Br J Community Nurs*, 2016; S29-30, S32-6.
114. Brealey G, James E, Hay K. Pressure cushions in a home environment: How effective are they at reducing interface pressure and does the chair surface count? A pilot study. *Wound Practice & Research*, 2017; 25(4): 180-187.
115. Makhsous M, Lin F, Knaus E, Zeigler M, Rowles DM, Gittler M, Bankard J, Chen D. Promote pressure ulcer healing in individuals with spinal cord injury using an individualized cyclic pressure-relief protocol. *Adv Skin Wound Care*, 2009; 22(11): 514-521.
116. Elsner JJ, Gefen A. Is obesity a risk factor for deep tissue injury in patients with spinal cord injury? *J Biomech*, 2008; 41(16): 3322-3331.
117. Sopher R, Nixon J, Gorecki C, Gefen A. Exposure to internal muscle tissue loads under the ischial tuberosities during sitting is elevated at abnormally high or low body mass indices. *J Biomech*, 2010; 43(2): 280-286.
118. Levy A, Kopplin K, Gefen A. A computer modeling study to evaluate the potential effect of air cell-based cushions on the tissues of bariatric and diabetic patients. *Ostomy Wound Management*, 2016; 62(1): 22-30.
119. Sprigle S. Visual inspections of wheelchair cushions after everyday use. *Assist Technol*, 2013; 25(3): 176-180.
120. McClure IA, McCarthy SK, Frisina P, Kirshblum SC. Comparison of cushions used by travelers with spinal cord injury. *J Spinal Cord Med*, 2013; ISSUE: 559-560.
121. Collins F. The contribution made by an armchair with integral pressure-reducing cushion in the prevention of pressure sore incidence in the elderly, acutely ill patient. *J Tissue Viability*, 1999; 9(4): 133-137.
122. Defloor T, Grypdonck MH. Do pressure relief cushions really relieve pressure? *West J Nurs Res*, 2000; 22(3): 335-350.
123. Clark M, Donald I. *A randomised controlled trial comparing the healing of pressure sores upon two pressure-redistributing seat cushions*. in *In: Proceedings of the 7th European Conference on Advances in Wound Management*. 1999. London: Macmillan Magazines Ltd.,.
124. Burns SP, Betz KL. Seating pressures with conventional and dynamic wheelchair cushions in tetraplegia. *Arch Phys Med Rehabil*, 1999; 80(5): 566-571.
125. Ham WHW, Schoonhoven L, Schuurmans MJ, Leenen LP. Pressure ulcers in trauma patients with suspected spine injury: A prospective cohort study with emphasis on device-related pressure ulcers. *International Wound Journal*, 2016.
126. Berg G, Nyberg S, Harrison P, Baumchen J, Gursz E, Hennes E. Near-infrared spectroscopy measurement of sacral tissue oxygen saturation in healthy volunteers immobilized on rigid spine boards. *Prehosp Emerg Care*, 2010; 14(4): 419-424.
127. Nemunaitis G, Roach MJ, Boulet M, Nagy JA, Kaufman B, Mejia M, Hefzy MS. The effect of a liner on the dispersion of sacral interface pressures during spinal immobilization. *Assist Technol*, 2015; 27(1): 9-17.
128. Pernik MN, Seidel HH, Blalock RE, Burgess AR, Horodyski MB, Rehtine GR, Prasarn ML. Comparison of tissue-interface pressure in healthy subjects lying on two trauma splinting devices: The vacuum mattress splint and long spine board. *Injury*, 2016; 46(8): 1801-1805.
129. Mok JM, Jackson KL, Fang R, Freedman BA. Effect of vacuum spine board immobilization on incidence of pressure ulcers during evacuation of military casualties from theater *Spine Journal*, 2013; 13(12): 1801-1808.
130. White CC, Domeier RM, Millin MG. EMS spinal precautions and the use of the long backboard –resource document to the position statement of the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma. *Prehosp Emerg Care*, 2014; 18(2): 306-314.
131. American College of Emergency Physicians Board of Directors. EMS management of patients with potential spinal injury. *Ann Emerg Med*, 2015; 66(4): 445.
132. Santamaria N, Gerdtz M, Sage S, McCann J, Freeman A, Vassiliou T, De Vincentis S, Ng AW, Manias E, Liu W, Knott J. A randomised controlled trial of the effectiveness of soft silicone multi-layered foam dressings in the prevention of sacral and heel pressure ulcers in trauma and critically ill patients: The Border trial. *Int Wound J*, 2015; 12(3): 302-308.
133. Weber SR, Rauscher P, Winsett RP. Comparison of a Padded Patient Litter and Long Spine Board for Spinal Immobilization in Air Medical Transport. *Air Med J*, 2015; 34(4): 213-217.

DEVICE RELATED PRESSURE INJURIES

Introduction

Device related pressure injuries result from the use of devices designed and applied for diagnostic or therapeutic purposes. Non-medical devices (e.g., bed clutter, furniture and equipment) can also result in pressure injuries when they (usually inadvertently) remain in contact with skin and tissues. The resultant pressure injury generally closely conforms to the pattern or shape of the device.¹ Potential sources of device related pressure injuries include, **but are not limited to:**^{2,3}

- Respiratory devices, for example:
 - tracheostomy faceplates and securement devices
 - masks used to deliver non-invasive positive pressure ventilation (NIV) (e.g., biphasic positive airway pressure [Bi-PAP], continuous positive airway pressure [CPAP])
 - endotracheal (ET) and nasotracheal tubes
 - oximeter probes
 - oxygen tubing/nasal cannulas.
- Orthopedic devices, for example:
 - cervical collars
 - halo devices
 - helmets
 - external fixators
 - immobilizers
 - braces
 - plaster casts.
- Urinary/fecal collection devices, for example:
 - indwelling urinary catheters
 - fecal containment devices
 - bedpans and bottles.
- Repositioning devices, for example:
 - heel lifts
 - slings and transfer boards.
- Device securements
- Nasogastric and feeding tubes
- Extra-corporeal membrane oxygenation (ECMO) cannulas
- Surgical drains
- Chest tubes
- Central venous and dialysis catheters
- Intravenous catheters and components
- Arterial lines
- Intra-aortic balloon pumps
- Retention sutures
- Blood pressure cuffs
- Intermittent pneumatic compression device sleeves
- Compression stockings and bandaging systems
- Restraints
- Devices and objects without a medical function (e.g., mobile phones, objects) left in the bed/chair.

Prolonged exposure to mechanical loads can lead to device related pressure injuries. Many medical devices that attach to the skin are based on generic designs employing traditional stiff polymer materials, which are secured via tape and strapping. The mismatch in mechanical properties between a stiffer device and softer skin and underlying tissues creates focal deformations and mechanical stress concentrations in tissues near the contact sites with the device.^{4,5} In addition, a medical device can result in an altered microclimate at the skin-device interface. One large prevalence study identified that medical device related pressure injuries (MDRPIs) occur more quickly following admission to a facility than non-MDRPIs (12 days versus 15 days, $p < 0.05$).³ Risk for MDRPIs may increase as a result of impaired sensation, moisture under the device, poor perfusion, altered tissue tolerance, poor nutritional status and edema.¹ Other factors that contribute to the formation of device related injuries include poor positioning and ill-fitting devices or incorrect device use.

Diagnostic or therapeutic devices are typically fixed to the skin surface with straps or taping creating high pressure and shear forces at the device interface.¹ In some instances, the generic design of the medical equipment can also contribute to pressure injury development.⁶⁻⁸ In critical care settings (e.g., adult and pediatric intensive care), the heavy burden of technology and equipment utilized in the environment renders the individual particularly vulnerable to the risk for device related pressure injuries. For example, in individuals with serious pathology, the functionality of the device is critical to life, necessitating its use for prolonged periods.

Mucosal pressure injuries are found on mucous membranes. The mucous membrane is the moist lining of body cavities that communicates with the exterior. These tissues line the tongue, oral mucosa, gastrointestinal (GI) tract, nasal passages, urinary tract, tracheal lining and vaginal tract. Pressure applied to this tissue can cause sustained deformation leading to ischemia. Mucosal tissues are especially vulnerable to pressure from medical devices, such as oxygen tubing, endotracheal tubes and tube holders, bite blocks, orogastric and nasogastric tubes, urinary catheters, and fecal containment devices.

Whenever a pressure injury occurs due to a medical device, removal or changing the device should be considered when clinically feasible. If the device must remain in-situ, strategies to relieve pressure should be implemented. Assessment and treatment for medical device related pressure injuries follow the current guidelines for pressure injury management.

Classifying Device Related Pressure Injuries

'Device related' refers to pressure injuries arising from devices. It also includes pressure injuries arising from regular items (e.g., cutlery, pens and phones), furniture or equipment that may have inadvertently applied pressure to the individual's skin. Except for those occurring on mucosal membranes, device related pressure injuries should be recorded as arising from a device and then classified using a pressure injury classification system. The term 'device related pressure injury' describes the etiology of the pressure injury; it should not replace the Category/Stage designation.^{9,10}

Classification systems for pressure injuries of the skin cannot be used to categorize mucosal pressure injuries.¹¹ Where pressure is a significant factor in the etiology of the mucosal wound, it should still be considered to be a pressure injury; however, it is inappropriate to use a pressure injury classification system to categorize/stage. Non-blanchable erythema cannot be seen in mucous membranes. Shallow open mucosal ulcers indicating superficial tissue loss of the non-keratinized epithelium are so shallow that the naked eye cannot distinguish them from deeper, full thickness pressure injuries. Soft coagulum seen in mucosal pressure injuries looks like slough that is often present in Category/Stage III pressure injuries. However, this is actually a soft blood clot. Exposed muscle is rarely visible in mucosal pressure injuries. These factors render classification systems designed for pressure injuries of the skin inappropriate in the classification of mucosal pressure injuries.¹¹ Although mucosal membrane pressure injuries are not classified using a pressure injury classification system, they should still be identified, monitored, reported and tracked in prevalence and incidence surveys. See the guideline chapter *Classification of Pressure Injuries* for further discussion.

Prevalence and Risk for Medical Device Related Pressure Injuries

In a survey³ of almost 100,000 individuals with complete medical records from 115 facilities in the US and Canada, the annual MDRPI prevalence was 0.60% (601/99,876). The rate of MDRPIs present on admission was 0.15%. Nasal oxygen devices were the most common devices to be associated with a MDRPI (32%), with the MDRPI most often occurring where the oxygen delivery device touched the ears. Casts and splints (12%), NIV devices (9%), intermittent compression therapy devices (7.7%), NGT (5%) and ET (7.5%) tubes, tracheostomy plates (5.5%) and cervical collars (2.4%) were other reported devices associated with MDRPIs. Overall, 51% of MDRPIs occurred on the face/head, with the ears (29%) being the most common anatomical location. However, there was no significant difference in the distribution of MDRPIs across anatomical locations ($\chi^2 = 4,800$, $p < 0.001$).³ Although MDRPIs occurred significantly more often in long term acute care settings ($\chi^2 = 91$, $p < 0.001$) compared with acute care, long term care, hospice care or rehabilitation, the researchers cautioned that there were fewer records from this setting to include in the analysis (Level 4).

Another large prevalence survey² included 106,722 patient days in an analysis of MDRPI events in three US aged care facilities. Between 35% and 50% of the pressure injuries observed in these facilities were associated with a medical device. Consistent with the findings of Kayser et al. (2018),³ MDRPIs occurred most often on the ears (71%) and were most often caused by casts/splints (20%) or oxygen tubing (15%)² (Level 4).

Other surveys highlight the risks associated with specific medical devices. Hobson et al. (2017)¹² reported 2.2% of individuals in intensive care settings experienced a compression stocking related pressure injury (Level 4). Schallom et al. (2018)¹³ noted a significantly higher rate of MDRPIs associated with oximetry sensors used on the forehead compared to nasal oximetry sensors (50% versus 9.7%, $p = 0.006$) in critical care settings (Level 4). From a sample of 2,136 surgical admissions, Asti et al. (2017)¹⁴ reported 4.8% of individuals with a NG tube experienced a nasal pressure injury. The primary factor associated with developing a NG tube-associated pressure injury was surgery duration, with surgery time of over four hours (12.6%, 95% confidence interval [CI] 9.2 to 17.1) having higher MDRPI rates than surgeries of less than two hours' duration (2.3%, 95% CI 9.2 to 17.1). Age, gender, type of NG tube and duration of tube use were not significantly associated with MDRPIs (Level 4).

A secondary analysis of data from eight quarterly point prevalence studies conducted in a US medical center ($n = 2,500$)¹⁵ investigated risk factors for facility-acquired pressure injuries. In a sub-population of adults in medical, surgical and step-down units who did not have a pressure injury on admission ($n = 2,079$), 1.4% of individuals had a MDRPI. For individuals in intensive care who had a facility-acquired pressure injury ($n = 83$ with 113 pressure injuries), 34.5% of the pressure injuries were deemed to be medical device related. Individuals with a medical device were significantly more likely ($\chi^2 = 6.98$, $p = 0.008$) to develop a pressure injury than those who had no medical device. Presence of a medical device indicated that the individual would be 2.4 times more likely (95% CI 1.2 to 4.8, $p = 0.10$) to develop a pressure injury of any kind¹⁵ (Level 4).

Turjanica et al. (2011)¹⁶ found a similarly high rate of medical device related pressure injuries in a convenience sample of individuals receiving oxygen via nasal cannula recruited in a medical/surgical unit ($n = 100$). In this sample, 37% of individuals experienced skin breakdown, predominantly classified as a Category/Stage I pressure injury. In a multivariate analysis, lack of oxygen use prior to hospital admission was the only factor significantly associated with increased likelihood of developing a pressure injury of the ear ($\chi^2 = 6.113$, $p = 0.013$) (Level 4).

Medical Device Related Pressure Injuries in Neonates and Children

Medical device related pressure injuries are an important consideration in children. While MDRPI prevalence and incidence rates vary according to methodology, clinical population and types of devices on which studies focused, neonates and children are consistently identified as a population with significant risk of MDRPIs. In a prevalence study conducted in a Spanish intensive care unit (ICU; $n = 47$),¹⁷ 22.7% of neonates experienced a pressure injury related to NIV masks (Level 4). In an observational study that included all children ($n = 61$) treated with a pinless halo over a nine-year period, pressure injuries were associated with the device in 4.9% of cases¹⁸ (Level 4). Su et al. (2014)¹⁹ reported a MDRPI rate of 6.25% in a sample of 32 children treated with brace fixation for club foot in one facility over four years (Level 4). In a prospective point prevalence study conducted in children hospitalized for at least 24 hours ($n = 412$; aged 24 hours to 18 years)²⁰ reported that 40% of 412 children with an external medical device were assessed as having a pressure injury related to the device (Level 4).

In a retrospective review of children (mean age 45 months \pm 8.7 months) who underwent a tracheostomy over a 15-month period in a US pediatric medical center ($n = 65$), Jaryszak et al. (2011)²¹ reported the rate of tracheostomy related pressure injuries as 29.2%. Multivariate analysis found that the type (design) of tracheostomy tube ($p = 0.003$) and being in a lower age group (under 12 months versus over 12 months) were significant risk factors for a MDRPI (Level 4).

In a prospective cohort study conducted in seven neonatal intensive care units ($n = 81$; mean age 32.5 weeks gestation), Fujii et al. (2010)²² reported that 86% of pressure injuries were associated with CPAP or nasal direction positive airway pressure (DPAP). A multivariate analysis showed an odds ratio (OR) of 4.0 (95% CI 1.04 to 15.42, $p = 0.047$) for pressure injuries in children undergoing ET intubation. In this study most of the neonates were extremely underweight, which is also a factor associated with increased pressure injury risk (Level 1).

Schindler et al. (2011)²³ conducted a multivariate analysis of risk factors for pressure injuries from retrospective data collected in seven pediatric intensive care units and trauma centers ($n = 5,346$). A number of factors associated with medical devices were significantly associated with an increased risk of pressure injuries including mechanical ventilation (OR = 1.334, 95% CI 1.031 to 1.726, $p = 0.03$); Bi-PAP or CPAP (OR = 2.004, 95% CI 1.509 to 2.661, $p < 0.001$); high frequency oscillatory ventilation (OR = 2.057, 95% CI 1.208 to 5.134, $p = 0.01$) and extracorporeal membrane oxygenation (OR = 2.490, 95% CI 1.208 to 5.134, $p = 0.01$) (Level 3).

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What factors should be considered when selecting and fitting a medical device?
- What local management strategies are effective in preventing MDRPIs?
- Is a prophylactic dressing effective for preventing MDRPIs? If so, what factors should be considered when selecting a prophylactic dressing?

Recommendations for Selecting, Fitting and Securing a Medical Device

8.1: To reduce the risk of medical device related pressure injuries, review and select medical devices with consideration to:

- The device's ability to minimize tissue damage
- Correct sizing/shape of the device for the individual
- Ability to correctly apply the device according to manufacturer's instructions
- Ability to correctly secure the device.

(Strength of Evidence = C; Strength of Recommendation = ↑↑)

Evidence Summary

There is direct evidence that medical device design, shape and sizing are associated with MDRPIs. The evidence from moderate and low quality Level of Evidence 2 to 4 studies reported that adjusting the type of device or securements used is associated with reduction in MDRPI incidence.^{21,24-28} Evidence from moderate and low quality Level 3 and 4 studies indicated that devices that were incorrectly sized or shaped were associated with increased MDRPIs in adults and children.^{29,30} Recent research indicates that individuals and their informal caregivers consider information on prevention and treatment of MDRPIs to be an important issue.

Implementation Considerations

- Review the range of medical devices and securements methods available in the facility and select stock to provide a wide range of devices associated with less skin damage from friction and shear.²⁷⁻²⁹ The guideline chapter on *Implementing Best Practice in the Clinical Settings* has further discussion on reviewing equipment as a part of organizational level quality improvement.
- Health professionals who apply and fit medical devices should have appropriate training (*Expert opinion*).
- Where safe and appropriate, modifying the device by adjusting or removing non-essential components that create a poor fit might be an option in some cases⁷ (*Expert opinion*).
- Provide information to individuals and caregivers about the purpose of selecting/changing a medical device to increase the acceptability to individuals and their caregivers of medical devices that might differ in appearance from their expectations²⁵ (*Expert opinion*).

Evidence Discussion

Medical devices provided within a facility should be selected with the input of health professionals and with consideration to the device's ability to minimize the risk of skin damage. This may include selection of softer, more flexible devices,²⁸ different device designs,²⁵ devices with a low-friction interface, or selecting a device model that has a smaller skin-device interface.²⁶

In a case series report,²¹ an audit of tracheostomy use in children²¹ identified that the type of tracheostomy tube in situ was associated with MDRPI rates, suggesting that careful selection of the type of device used could reduce pressure injury incidence²¹ (*Level 4*). Boesch et al. (2012)²⁵ also investigated the influence of tracheostomy device type in an exploration of a multi-faceted intervention to reduce tracheostomy related pressure injuries in 834 pediatric individuals. Interventions included the introduction of a hydrophilic foam dressing, in addition to the incorporation of a moisture and pressure-free device interface and an extended tracheostomy tube. Significant reductions in tracheostomy related pressure injury rates ($p = 0.007$) and in the number of days with an existing tracheostomy related pressure injury ($p < 0.0001$) were associated with the introduction of the extended tracheostomy tube and changes to tracheostomy care (*Level 2*). In one large ($n = 6,103$) quality improvement study²⁸ conducted in a US trauma center, similar success in reducing MDRPIs by changing the type of device or securement was demonstrated. In this study, the number of mucosal pressure injuries associated with ET tubes decreased with introduction of an institutional change in the brand of ET tube securement device (*Level 2*). The impact of selecting an appropriate securement method was

also demonstrated in a quasi-experiment conducted in long term care facilities over 12 months (n = 106,722 patient days). In this trial, individuals with an ET tube received either a commercial NG tube securement device (n = 115) or securement with regular adhesive tape wrapped around the NG tube and fixed to the nasal bridge (n = 83). The commercial device was associated with fewer MDRPIs (4% versus 23%, p < 0.0001)³¹ (Level 2). In contrast, another study reported an increase in oral pressure injuries when a commercial ET tube securement device was introduced in an intensive care unit. Prior to the securement device being introduced, a cloth securement device with six hourly position adjustment was used in the unit. On its introduction, the commercial securement device was adjusted every two hours. Incidence of ET tube-associated pressure injury rates increased from 1.98/100 to 4.03/100 (incident rate ratio [IRR] 2.03, 95% CI 1.17 to 3.51, p = 0.02). These findings may have been related to the new device, the change in management protocols or increased surveillance as a result of the study³² (Level 2). There is currently minimal evidence for specific securement devices, and health professionals need to work with facilities to explore securement options that might reduce pressure injury rates.

Using medical devices with the lowest skin-device interface appears to reduce the incidence of MDRPIs. In a quasi-experiment²⁶ in which CPAP delivered via a facial mask (with a prophylactic dressing in situ) was compared to an oxygen helmet in babies (aged 3 to 11 months, n = 40), lower MDRPI incidence was associated with the helmet (0% versus 75%, p = 0.002). Despite wearing the helmet for a significantly longer mean duration than the facial mask (10.8 ± 2.0 hours versus 6.4 ± 1.8 hours, p = 0.001), the smaller skin-device interface reduced skin injury. This result was achieved without compromising gas exchange, and with a significantly lower rate of device intolerance requiring sedation. However, of 97 potential participants, only 20 children met the selection criteria to use the CPAP helmet, indicating that practical use of the device may be limited (Level 2). In an observational study (n = 74) conducted in an ICU, adults receiving oxygen therapy who switched early in the course of treatment from a standard facial mask to a total face mask with lower skin-device contact were less likely to sustain a facial pressure injury (24% versus 87%, p = 0.0002). However, the time spent wearing the different masks was not equivalent²⁷ (Level 4).

Careful selection of a medical device sized correctly for the individual is important because ill-fitting devices have been shown to contribute to device malfunction and to an increase in pressure at the skin-device interface. Respiratory masks used to deliver NIV should be fitted sufficiently that air leaks are prevented without creating pressure injuries. In one retrospective, observational study in 410 children, the presence of an ill-fitting helmet was reported to be a contributory factor to pressure injury development in 10.5% of children wearing helmets³⁰ (Level 4). A prospective cohort study (n = 50) conducted over three years showed that the incidence of MDRPIs associated with facial oxygen masks was higher in children with cranio-facial abnormalities due to ill-fitting masks positioned over bony prominences.²⁹ Method of participant selection of and their assignment to groups was unclear in the reporting (Level 3).

In some cases, when it is appropriate and safe, medical devices may need to be adjusted or modified in order to prevent pressure injuries. In one study of complications associated with halo use in children (n = 68), the researchers found that cutting or trimming the offending portion of the halo vest reduced discomfort and relieved pressure in most cases (Level 4).⁷ However, it is essential that making adjustments to the device or its securements does not alter the device function.

8.2: Regularly monitor the tension of medical device securements and where possible seek the individual's self-assessment of comfort.

(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

There is currently no evidence that reducing the tension in medical device securements decreases pressure injuries. However, there is evidence from two studies in healthy volunteers that show that increasing the tension of medical device securements is associated with unfavorable changes in indirect outcome measures, including increased interface pressure,^{8,33} increases in some markers of inflammatory response³³ and increased discomfort.^{8,33} Recent research indicates that patient consumers and their informal caregivers consider information on prevention and treatment of MDRPIs to be an important issue.^{34,35}

Implementation Considerations

- The tension of medical device securements may need to be monitored and adjusted more often (greater than twice daily) in individuals vulnerable to fluid shifts and/or exhibiting signs of localized or generalized edema¹ (*Expert opinion*).
- Do not compromise the function of the device when adjusting the device securements (*Expert opinion*).

Evidence Discussion

In healthy volunteers, increasing oxygen mask strap tension by 5mm and greater was significantly and positively associated with interface pressure ($p < 0.01$) and greater discomfort ratings on an unreported subjective scale ($p < 0.05$).³³ However, reducing the tension of oxygen mask straps had no significant impact on either skin temperature or humidity values ($p > 0.05$).³³ An increase in tension of oxygen mask straps was also associated with an increase in interleukin 1 alpha (IL-1 α) concentrations as measured by a device used to assess skin surface sebum levels at the nose bridge (median ratio of 1.34 at highest strap tension, $p < 0.05$).³³ There were inconsistent trends in changes in interleukins (IL-1 β , IL-8, IL-2, IL-6 and IL-10) and interferon (IFN- γ) associated with changes in tension of oxygen mask straps³³ (Level 5).

In a second trial in healthy volunteers, increasing interface pressures were associated with greater cervical collar tension in two different collar models ($p < 0.01$).⁸ There was also a significant difference in the comfort scores ($p < 0.01$), with the greatest discomfort associated with the highest tension in the cervical collar securement. While the comfort scores between two different collar designs were not significant ($p > 0.05$), the ratio of cytokine concentrations from pre-collar to post-collar increased. However, there was no significant difference between the design or the tension of the collars being tested ($p > 0.05$)⁸ (Level 5).

There was no evidence reported on potential adverse events of reducing the tension of medical device securements. It is important that adjusting securements does not reduce the efficacy of the medical device or put the individual at clinical risk.

Recommendations for Assessment of the Skin and Medical Device

8.3: Assess the skin under and around medical devices for signs of pressure related injury as part of routine skin assessment.
(Good Practice Statement)

Implementation Considerations

- Safely remove medical devices (if possible) without interfering with medical treatment when conducting a skin assessment (*Expert opinion*).
- Continue to assess the skin regularly when a prophylactic dressing is in place under the medical device¹ (*Expert opinion*).
- Conduct more frequent skin assessments (greater than twice daily) at the skin-device interface in individuals vulnerable to fluid shifts and/or exhibiting signs of localized or generalized edema¹ (*Expert opinion*).
- Assess under the medical device for moisture from diaphoresis or excessive secretions that can cause tissue maceration and contribute to development of pressure injuries¹ (*Expert opinion*).
- Be aware of devices that can become entrapped in skin folds resulting in skin damage, especially in the individuals who are overweight or obese³⁶ (*Expert opinion*).

Discussion

Conducting frequent skin assessments is considered best practice, although there is no high quality scientific evidence to support this practice in preventing MDRPIs. Regular assessment of the skin allows prompt detection of pressure related injury. By identifying risks early, strategies to redistribute pressure can be implemented. Frequently inspect the skin beneath adjustable medical devices and continue to lift and/or adjust or remove the medical device for pressure relief where possible. The guideline chapter *Skin and Soft Tissue Assessment* provides further guidance on when and how to perform a skin assessment.

Changes in fluid volume status or hypoproteinemic states can result in localized or generalized edema causing a medical device that initially fits properly to exert external pressure to the skin that leads to pressure injury formation.³⁷ The health professional should apply an interface material that can accommodate expansion and worsening edema in those individuals at risk of tissue volume changes. Depending on the type/purpose of the device and the extent of volume changes, loosening, replacing or removing the device may be advised.

Individuals in the home setting fitted with a medical device should continue to perform routine skin assessments on a regular basis, inspecting under or around the device between health professional appointments.⁷

Recommendations for Prevention of Medical Device Related Pressure Injuries

8.4: Reduce and/or redistribute pressure at the skin-device interface by:

- Regularly rotating or repositioning the medical device and/or the individual
- Providing physical support for medical devices in order to minimize pressure and shear
- Removing medical devices as soon as medically feasible.

(Good Practice Statement)

Implementation Considerations

- Simple changes in posture of the individual and device positioning may be used to minimize pressure and shear created by medical devices (*Expert opinion*).
- When repositioning, do not position the individual directly on a medical device unless it cannot be avoided (*Expert opinion*).
- Endotracheal tubes may be repositioned laterally to prevent a MDRPI. Always validate that the insertion depth of the ET tube does not change when the tube is repositioned (e.g., using a centimeter marking at the teeth or lips) (*Expert opinion*).

Discussion

In order to reduce pressure injury risk associated with the use of a medical device, individuals should be routinely assessed for the continued need for the device, and the device should be removed as soon as it is no longer clinically indicated. Hard/rigid extrication cervical collars should be removed and replaced with soft acute care collars as soon as feasible^{38,39} (see *Recommendation 8.8*).

Pressure injuries may develop under medical devices that have been compressed under the individual causing a localized area of pressure. If positioning an individual on a medical device is unavoidable, regularly reposition the individual to redistribute pressure away from the device. Repositioning strategies may vary depending on the individual and the medical device. Simple changes in degree of lateral rotation, head of bed elevation, knee elevation and the positioning of devices may be used to minimize the magnitude and duration of the pressure and shear at the skin-device interface. For example, ensuring a device is not dependent after repositioning may minimize its gravitational pull on skin and other tissues.

Wherever possible, a medical device should be regularly repositioned or rotated. Oximetry probes can be rotated to a different finger/toe or positioned on the hand, ear lobe or forehead, for either constant or periodic monitoring. Endotracheal tubes can be moved laterally to redistribute pressure over different parts of the oral cavity and lips. Care should be taken to prevent ET tube migration via reference to documented insertion depth measurement.

8.5: Use a prophylactic dressing beneath a medical device to reduce the risk of medical device related pressure injuries.

(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

There is direct evidence in a range of populations that application of a prophylactic dressing at the skin-device interface reduces the incidence of MDRPIs. The desirable effects of prophylactic dressings used in conjunction with medical device application is supported by several Level 1, 2 and 3 studies of moderate quality. The evidence included effectiveness in reducing pressure injury incidence when prophylactic dressings were used with tracheostomies,^{25,40} ET tubes,⁴¹ ventilation prongs and masks,^{42,43} and under casts.⁴⁴ A range of different types of prophylactic dressings were evaluated in the literature, including hydrocolloid dressings,^{40,42} foam dressings,^{25,40,44,45} silicone gel sheeting⁴³ and transparent films.⁴² No cost effectiveness studies for prophylactic dressings used in conjunction with a medical device were identified. Recent research indicates that patient consumers and their informal caregivers consider information on prevention and treatment of MDRPIs to be an important issue.

Implementation Considerations

- Evaluate the vulnerability of the individual's skin and the potential benefit of using a prophylactic dressing. Consider the increased fragility of neonatal skin and that of older adults. (*Expert opinion*).

- Avoid excessive layering of prophylactic dressings under a medical device because layering may increase pressure at the skin-device interface¹ (*Expert opinion*).
- Ensure the functionality of the medical device is not compromised by the prophylactic dressing (*Expert opinion*).
- Consider the following factors when selecting a prophylactic dressing:
 - Ability of the prophylactic dressing to manage moisture and microclimate,¹ especially when used with a medical device that may be in contact with bodily fluids/drainage (e.g., percutaneous endoscopic gastrostomy tube)
 - Ease of application and removal¹
 - Ability to regularly assess the condition of the skin¹
 - Thickness of the dressing under tightly-fitting devices¹
 - Anatomical location of the medical device
 - Type/purpose of the medical device
 - The individual's preferences, comfort and any allergies
 - Coefficient of friction at the skin-dressing interface (*Expert opinion*).
- Replace the prophylactic dressing if it becomes dislodged, loosened or excessively moist,^{46,47} if the dressing or skin underneath become soiled, and according to the manufacturer's instructions (*Levels 1 and 3*).
- Follow manufacturers' instructions for use (*Expert opinion*).

Evidence Discussion

In one randomized controlled trial (RCT),⁴³ the use of a silicone gel sheeting was found to be effective in reducing the occurrence of nasal injuries in preterm infants. The study investigated prevention of nasal injuries (described in the study as bleeding, crusting, excoriation and columella necrosis) using 1.8 mm thick silicone gel sheeting applied to the nares of premature neonates during nasal CPAP. Compared to no intervention (n = 97), the prophylactic gel sheeting (n = 92) was associated with significantly fewer nasal injuries (14.9% versus 4.3%, OR = 3.43, 95% CI 1.1 to 10.1, p < 0.05) and fewer cases of columella necrosis at one month follow up (1.08% versus 6.8%, OR = 6.34, 95% CI 0.78 to 51.6, p < 0.05). Infants that developed a nasal injury had a much longer mean duration of ventilation (19.6 ± 10.6 days versus 4 ± 3.3 days), but injuries developed more rapidly in those without gel sheeting. Methods of randomization, allocation concealment and blinding were not clearly reported and the variance in therapy duration between groups confounded the findings (*Level 1*).

Using a quasi-experiment design, O'Toole et al. (2017)⁴⁰ compared regular management (no standard protocol, n = 183) of new tracheostomies to a standardized protocol that included consecutive use of two prophylactic dressings (n = 155). Immediately following surgery, a hydrocolloid dressing was placed under the tracheostomy flare. After one week, sutures were removed, and the hydrocolloid dressing was replaced with a polyurethane foam dressing, applied with neck in a neutral position. Following the introduction of this protocol, incidence of tracheostomy related pressure injuries was significantly lower (1.29% versus 10.93%, p = 0.003). The pressure injuries occurring in the intervention phase were deemed to be related to non-adherence to the protocol. Although skin assessment was performed daily, classification of pressure injuries was performed on a monthly basis, which may have confounded the reporting of some events⁴⁰ (*Level 2*).

In a controlled clinical trial, Forni et al. (2011)⁴⁴ reported a significant difference in the development of Category/Stage I heel pressure injuries (defined as "sore skin" in the study) between a group receiving a polyurethane foam dressing applied under the heel pad of a casted limb (n = 71) and a control group receiving no prophylactic dressing (n = 85). Less than 4% of the participants receiving the foam dressing developed a Category/Stage I heel pressure injury compared to almost 43% (p < 0.0005) in the control group. This equated to a relative risk of 0.08 (95% CI 0.02 to 0.33) of developing a heel pressure injury when a prophylactic polyurethane foam dressing was applied. However, the duration for cast wearing was not reported and it was unclear if it was equivalent between the groups (*Level 2*).

Boesch et al. (2012)²⁵ implemented a quality improvement program in a children's hospital over 10,132 tracheostomy days (n = 834) to reduce tracheostomy related pressure injuries. The intervention included application of a hydrophilic polyurethane foam dressing under the tracheostomy flare. Compared to a pre-intervention period, the quality improvement intervention was associated with a reduction in the mean rate of tracheostomy related pressure injuries (from 8.1% to 2.6% over 12 months). There were other components to the intervention, including changing to an extended tracheostomy device design, conducting staff education on risk and skin assessment, and incorporating tracheostomy related pressure injury prevention into the electronic workflow of nurses. Therefore, it is unclear whether the prophylactic dressing specifically influenced the reduction in pressure injury incidence²⁵ (*Level 2*). In another pediatric-based quality improvement project,⁴⁸ introduction of an adhesive foam dressing under CPAP masks in children requiring non-invasive ventilation or during prone surgery reduced the incidence of facial pressure injuries

to zero. In this study, nurses and respiratory therapists used a multi-disciplinary approach to development of the care bundle⁴⁸ (Level 3).

Whitley et al. (2017)⁴¹ demonstrated a reduction in MDRPI associated with ET tubes in a comparison between a twill securement used with silicone pressure reducing strip (n = 38) and the twill securement alone (n = 77). The participants were children and adults recruited in a burn center requiring ventilation (age range 0 to 92 years). Medical device related pressure injuries reduced after introduction of the prophylactic silicone strips (20.7% versus 5.2% p = 0.032). (Level 3)

Kuo et al. (2013)⁴⁵ reported findings from a retrospective cohort study investigating effectiveness of a silver foam dressing used for preventing skin breakdown. The dressing was applied beneath the tracheostomy and ties. The study showed that the use of the prophylactic dressing was significantly associated with a reduction in tracheostomy site pressure injuries in a sample of 134 children undergoing tracheotomies in a tertiary care pediatric hospital. No skin breakdown developed in the prophylactic dressing group as compared to the 11.8% of the comparison cohort group (p = 0.02) experiencing skin breakdown. The method of skin assessment and pressure injury classification was not reported and with a six-year study period, other practice changes may also have influenced the study outcomes. (Level 3).

In a smaller, quasi-experimental study⁴⁹ conducted in 18 nasally intubated participants undergoing head/neck surgery, the use of a hydrocolloid dressing in combination with a soft liner made from a composite conformable material used for denture cushioning was found to be effective in reducing the rate of pressure injuries associated with nasal intubation (60% versus 100%, p = not reported) (Level 2).

In a comparative study conducted in Taiwan by Weng (2008),⁴² individuals requiring NIV received either a hydrocolloid dressing (n = 30), a transparent film dressing (n = 30) or no prophylactic dressing (n = 30). The dressings were placed on the nasal bridge prior to application of a respiratory face mask. Category/Stage I device related pressure injuries were significantly reduced in individuals treated prophylactically with the hydrocolloid dressing compared to controls receiving no prophylactic dressing (40% versus 96.7%, p < 0.01), demonstrating an absolute risk reduction of greater than 50%. There was also a significant reduction in Category/Stage I pressure injuries with the transparent film dressing compared to no prophylactic dressing (53.3% versus 96.7%, p < 0.01). The study showed a very high rate of pressure injuries in the control group, which may have been influenced by the darker skin tone of the participants that is reported to delay pressure injury identification.⁵⁰ Time to occurrence of a Category/Stage I pressure injury was significantly increased (p < 0.01) for both prophylactic dressings (no dressing 1111 ± 2169 minutes versus transparent film dressing 2628 ± 1655 minutes versus hydrocolloid dressing 3272 ± 2566 minutes). A potential mechanism for this effect is that the dressing reduced sliding of the mask on the individual's skin and reduced skin irritation caused by pressure from tight restraining straps (Level 3).

However, it is of note that prophylactic dressings differ in their qualities;^{51,52} therefore it is important to select a dressing that is appropriate to the individual and the clinical use. A transparent film dressing is less able to manage drainage, and may not adhere to the skin as effectively as a hydrocolloid dressing.⁴² Foam dressings have greater ability to absorb moisture than film or hydrocolloid dressings.¹ Some dressings are more able to manage humidity and moisture at the skin surface than others. One laboratory study found that for some dressings, accumulation of moisture reduced the ability of the dressing to transpire.⁵² When prophylactic dressings are used, consider the fragility of the individual's skin and the ease of removal of the dressing when performing routine skin assessments. Detrimental effects such as epidermal stripping may occur with frequent removal of adhesive-based dressings,⁵¹ particularly in more vulnerable skin of older adults and immature skin of neonates (see the *Special Populations* chapter of the guideline). Some dressings are designed to adhere well to the skin. However, if they are not removed carefully there is increased risk of damage to fragile skin.^{51,53,54} Dressings with a soft silicone border may be more easily lifted for regular skin assessment, and appear to absorb shear forces more efficiently.⁵¹

Further discussion on properties of prophylactic dressings is in the guideline chapter *Preventive Skin Care*.

8.6: If appropriate and safe, alternate the oxygen delivery device between correctly fitting mask and nasal prongs to reduce the severity of nasal and facial pressure injuries for neonates receiving oxygen therapy.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

8.7: If appropriate and safe, alternate the oxygen delivery device between correctly-fitting mask(s) and nasal prongs to reduce the severity of nasal and facial pressure injuries for older children and adults receiving oxygen therapy.
(Good Practice Statement)

Evidence Summary

There is direct evidence from a high quality Level 1 study⁵⁵ that rotation between mask and nasal prongs every four hours reduced nasal and facial pressure injuries (described in the study as skin excoriation and erythema). The evidence for effectiveness of alternating oxygen delivery methods for reducing MDRPIs was conducted in extremely low birth weight neonates in critical care. There are no reported adverse events and a cost analysis was not undertaken.

Although there is no direct evidence on the influence on MDRPI incidence of rotating oxygen delivery systems in older children and adults, evidence can be extrapolated from the studies in neonates, from a Level 2 study comparing different oxygen delivery systems conducted in babies,²⁶ and from a Level 4 study comparing different oxygen delivery systems in adults.²⁷ Alternating the type of oxygen delivery device can rotate the anatomical areas in contact with a medical device, providing skin and soft tissue with intermittent pressure relief.

Implementation Considerations

- Ensure access to a wide range of mask and nasal prong sizes to allow an optimal fit (*Expert opinion*).
- Ensure oxygen delivery devices are well-fitted to the individual and correctly secured (*Expert opinion*).
- Continue to evaluate the skin at frequent time intervals and during each change of the oxygen delivery system (*Expert opinion*).
- Monitor oxygen saturation levels to ensure that the individual continues to receive adequate oxygen when the delivery device is changed (*Expert opinion*).

Evidence Discussion

In an RCT conducted in low birthweight neonates,⁵⁵ rotating the oxygen therapy delivery option (changing between mask and nasal prongs) was associated with significantly lower 'skin excoriation' scores (as described on the Neonatal Skin Condition Scale [NSCS], scale of 1 to 3) than receiving oxygen therapy by the same device (1.10 [alternating regimen] versus 1.18 [prongs alone] versus 1.19 [mask alone], $p = 0.007$). Rotating oxygen therapy delivery options between mask and nasal prongs was also associated with significantly lower erythema scores on the NSCS than using the same oxygen delivery device (1.18 [alternating regimen] versus 1.12 [prongs alone] versus 1.31 [mask alone], $p = 0.007$), without any adverse events⁵⁵ (*Level 1*).

Although there is no direct evidence in older children and adult populations, regular rotation of oxygen delivery devices may reduce nasal and facial pressure injuries. Two studies in these populations demonstrated that selecting an oxygen delivery system with a lower skin-device interface was associated with fewer MDRPIs. The first study (reported above),²⁶ demonstrated that a helmet oxygen delivery system for babies aged 3 to 11 months was associated with a lower MDRPI incidence than a facial or nasal mask, while attaining adequate oxygen saturation levels (*Level 2*). In the second study,²⁷ adults in critical care who switched from a regular oxygen face mask to a total face mask earlier in the course of treatment experienced fewer facial pressure injuries (24% versus 87%, $p = 0.0002$) (*Level 4*). While neither study explicitly explored rotating between different oxygen delivery systems, it should be considered as a treatment option for older children and adults who require longer-term oxygen therapy and who have a high risk of developing pressure injuries.

8.8: In consultation with a qualified health professional, replace an extrication cervical collar with an acute care rigid collar as soon as feasible and remove cervical collars as soon as possible, as indicate by clinical condition. (Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Evidence from high^{56,57} and moderate³⁹ quality Level 4 studies indicates that incidence of pressure injuries associated with extrication collars is high, with one study reporting an incidence rate of over 75%.⁵⁶ In an observational study in which an extrication collar was replaced with an acute care collar within eight hours, pressure injury incidence was around 7%.³⁹ There were no comparative studies demonstrating effects of removing an extrication cervical collar. Indirect evidence demonstrated no clinically significant differences in interface pressure between different models of acute care cervical collars.⁵⁸

Implementation Considerations

- In consultation with a qualified health professional, remove cervical collars in adults who are awake and alert and who have no neurological deficit, no evidence of intoxication, no neck pain/tenderness and demonstrate full

cervical spine range of motion. Undertake cervical spine screening (e.g., computer tomography) when unstable cervical spine injury is suspected⁵⁹⁻⁶¹ (*Expert opinion*).

- Follow the manufacturer's directions when sizing and applying a cervical collar⁵⁸ (*Expert opinion*).
- Assess skin under the cervical collar regularly (e.g., at least twice/day)³⁹ and change collar pads according to the manufacturer's instructions (*Expert opinion*).

Evidence Discussion

Extrication collars are applied to individuals with suspected spinal cord injury (SCI) for reducing spinal range of motion in the acute phase of injury prior to hospitalization.^{56,57} Used in conjunction with a spinal back board (see *Recommendation 7.16* in the guideline chapter *Support Surfaces*) and head blocks, an extrication collar maintains the spine in straight alignment by preventing rotation at the neck.⁵⁶ To achieve protection of the spine from (further) injury, extrication collars are tightly fitted and therefore increase risk of tissue and skin damage due to pressure and friction.^{39,56}

The available evidence on the relationship between cervical collars used in the acute phase of (suspected) spinal cord injury (SCI) and pressure injuries is derived from observational studies, which generally report high pressure injury incidence. In one study conducted in individuals admitted to a trauma unit (n = 342), the incidence of pressure injuries after removal of the extrication collar was 78.4% (95% CI 73.6% to 82.6%). In a pain assessment that was performed while undertaking the skin assessment, 38.5% of individuals reported severe pain (rated ≥ 7 on a 10 point scale) that was deemed to be associated with wearing the extrication collar. In addition, 64.6% (95% CI 59.3% to 69.7%) had indentation marks that matched the extrication collar location. The pressure injuries reported in this study were primarily Category/Stage I and II pressure injuries⁵⁶ (*Level 4*). A second study⁵⁷ conducted in individuals (n = 254) admitted to a trauma unit with a back board, extrication collar and headblocks in situ reported a pressure injury incidence rate of 28.3% (95% CI 22.8% to 34.3%). Approximately 90% of these pressure injuries were deemed to be associated with a medical device and of these, 55.7% (95% CI 44.7% to 66.3%) were related to immobilizing devices, primarily cervical collars. Approximately 40% of the pressure injuries reported in the study were full thickness pressure injuries⁵⁷ (*Level 4*). In a third observation study,³⁹ the incidence of collar-associated pressure injuries in trauma patients (n = 484) over the full duration of their hospital admission was 6.8%. In this study, all individuals admitted with an extrication collar had that collar replaced by an acute care collar within eight hours of admission³⁹ (*Level 4*). Pressure injuries associated with cervical collars occur most often at the back, shoulders and chest,⁵⁶ as well as the chin and occiput⁵⁷ (*Level 4*).

Extrication collars are recommended by many paramedic protocols for application until cervical injury has been ruled out or stabilized, although increasingly their relationship to adverse events including pressure injuries is being recognized and the benefits of their use re-evaluated.⁶² Extrication cervical collars should be removed as soon as feasible and the requirement for a cervical collar should be assessed. When a continuing use of a cervical collar is deemed necessary, use either an acute care rigid collar or a soft collar.⁶² One study⁵⁸ conducted in health volunteers (n = 48) found no statistically significant difference between four different acute care collars for ability to restrict movement (p < 0.001). Although one model of collar was associated with statistically significantly lower interface pressure at the mandible and occiput, the small difference was deemed to have no clinical significance.⁵⁸ This study also noted that interface pressures were high in individuals with a higher body mass index (BMI), highlighting the importance of selecting an appropriately sized collar⁵⁸ (*Level 5*).

References

1. Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Call E, Clark M. Use of wound dressings to enhance prevention of pressure ulcers caused by medical devices. *Int Wound J*, 2015; 12: 322-327.
2. Arnold-Long M, Ayer M, Borchert K. Medical device-related pressure injuries in long-term acute care hospital setting. *J Wound Ostomy Continence Nurs*, 2017; 44(4): 325-330.
3. Kayser S, VanGilder C, Ayello E, Lachenbruch C. Prevalence and analysis of medical device-related pressure injuries: Results from the International Pressure Ulcer Prevalence Survey. *Adv Skin Wound Care*, 2018; 31(6): 276-285.
4. Lustig M, Levy A, Kopplin K, Ovadia-Blechman Z, Gefen A. Beware of the toilet: The risk for a deep tissue injury during toilet sitting. *J Tissue Viability*, 2018; 27(1): 23-31.
5. Levy A, Kopplin K, Gefen A. Device-related pressure ulcers from a biomechanical perspective. *J Tissue Viability*, 2017; 26(1): 57-68.
6. Skillman J, Thomas S. An audit of pressure sores caused by intermittent compression devices used to prevent venous thromboembolism. *J Perioper Pract*, 2011; 21(12): 418-20.
7. Limpaphayom N, Skaggs DL, McComb G, Krieger M, Tolo VT. Complications of halo use in children. *Spine (Phila Pa 1976)*, 2009; 34(8): 779-784.
8. Worsley PR, Stanger ND, Horrell AK, Bader DL. Investigating the effects of cervical collar design and fit on the biomechanical and biomarker reaction at the skin. *Med Devices (Auckl)*, 2018; 11: 87-94.

9. Edsberg LE, Black JM, Goldberg M, McNichol L, Moore L, Sieggreen M. Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System. *J Wound Ostomy Continence Nurs*, 2016; 43(6): 585-597.
10. National Pressure Ulcer Advisory Panel. *NPUAP Pressure Injury Stages*. 2016; Available from: <http://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/>.
11. National Pressure Ulcer Advisory Panel. *Mucosal Pressure Ulcers: An NPUAP Position Statement*. 2008; Available from: http://www.npuap.org/wp-content/uploads/2012/01/Mucosal_Pressure_Ulcer_Position_Statement_final.pdf.
12. Hobson DB, Chang TY, Aboagye JK, Lau BD, Shihab HM, Fisher B, Young S, Sujeta N, Shaffer DL, Popoola VO, Kraus PS, Knorr G, Farrow NE, Streiff MB, Haut ER. Prevalence of graduated compression stocking-associated pressure injuries in surgical intensive care units. *J Crit Care*, 2017; 40: 1-6.
13. Schallom M, Prentice D, Sona C, Arroyo C, Mazuski J. Comparison of nasal and forehead oximetry accuracy and pressure injury in critically ill patients. *Heart Lung*, 2018; 47(2): 93-99.
14. Asti E, Sironi A, Milito P, Bonavina G, Bonitta G, Bonavina L. Prevalence and risk factors of nasal pressure ulcers related to nasogastric intubation: an observational study. *Eur Surg*, 2017: 1-4.
15. Black JM, Cuddigan JE, Walko MA, Didier LA, Lander MJ, Kelpel MR. Medical device related pressure ulcers in hospitalized patients. *Int Wound J*, 2010; 7(5): 358-365.
16. Turjanica MA, Clark L, Martini C, Miller P, Turner BL, Jones S. Incidence, correlates, and interventions used for pressure ulcers of the ear. *Medsurg Nurs*, 2011; 20(5): 241-247.
17. Bonell-Pons L, García-Molina P, Balaguer-López E, Montal MÁ, Rodríguez MC. Neonatal facial pressure ulcers related to noninvasive ventilation: Incidence and risk factors. *EWMA Journal*, 2014; 14(2): 33-33.
18. Bakhshi H, Kushare I, Banskota B, Nelson C, Dormans JP. Pinless halo in the pediatric population: Indications and complications. *J Pediatr Orthop*, 2015; 35(4): 374-8.
19. Su Y, Nan G. Manipulation and brace fixing for the treatment of congenital clubfoot in newborns and infants. *BMC Musculoskeletal Disord*, 2014; 15: 363.
20. Schluer AB, Halfens RJ, Schols JGA. Pediatric pressure ulcer prevalence: A multicenter, cross-sectional, point prevalence study in Switzerland. *Ostomy Wound Manage*, 2012; 58(7): 18-31.
21. Jaryszak EM, Shah RK, Amling J, Peña MT. Pediatric tracheotomy wound complications: Incidence and significance. *Arch Otolaryngol*, 2011; 137(4): 363-366.
22. Fujii K, Sugama J, Okuwa M, Sanada H, Mizokami Y. Incidence and risk factors of pressure ulcers in seven neonatal intensive care units in Japan: a multisite prospective cohort study. *International Wound Journal*, 2010; 7(5): 323-328.
23. Schindler CA, Mikhailov TA, Kuhn EM, Christopher J, Conway P, Ridling D, Scott AM, Simpson VS. Protecting fragile skin: Nursing interventions to decrease development of pressure ulcers in pediatric intensive care. *Am J Crit Care*, 2011; 20(1): 26-35.
24. Ambutas S, Staffileno BA, Fogg L. Reducing nasal pressure ulcers with an alternative taping device. *Medsurg Nurs*, 2014; 23(2): 96-100.
25. Boesch RP, Myers C, Garrett T, Nie A, Thomas N, Chima A, McPhail GL, Ednick M, Rutter MJ, Dressman K. Prevention of tracheostomy-related pressure ulcers in children. *Pediatrics*, 2012; 129(3): e792-e797.
26. Chidini G, Calderini E, Pelosi P. Treatment of acute hypoxemic respiratory failure with continuous positive airway pressure delivered by a new pediatric helmet in comparison with a standard full face mask: A prospective pilot study. *Pediatr Crit Care Med*, 2010; 11(4): 502-508.
27. Lemyze M, Mallat J, Nigeon O, Barrailler S, Pepy F, Gasan G, Vangrunderbeeck N, Grosset P, Tronchon L, Thevenin D. Rescue therapy by switching to total face mask after failure of face mask-delivered noninvasive ventilation in do-not-intubate patients in acute respiratory failure. *Crit Care Med*, 2013; 41(2): 481-488.
28. Zaratkiewicz S, Whitney JD, Lowe JR, Taylor S, O'Donnell F, Minton-Foltz P. Development and implementation of a hospital-acquired pressure ulcer incidence tracking system and algorithm. *J Healthc Qual Res*, 2010; 32(6): 44-51.
29. Visscher MO, White CC, Jones JM, Cahill T, Jones DC, Pan BS. Face masks for noninvasive ventilation: Fit, excess skin hydration, and pressure ulcers. *Respir Care*, 2015; 60(11): 1536-1547.
30. Wilbrand JF, Wilbrand M, Malik CY, Howaldt HP, Streckbein P, Schaaf H, Kerkmann H. Complications in helmet therapy. *Journal of Cranio-Maxillofacial Surgery*, 2012; 40(4): 341-346.
31. Ambutas S, Staffileno BA, Fogg L. Reducing nasal pressure ulcers with an alternative taping device. *Medsurg Nursing*, 2014; 23(2): 96-100.
32. Hampson J, Green C, Stewart J, Armitstead L, Degan G, Aubrey A, Paul E, Tiruvoipati R. Impact of the introduction of an endotracheal tube attachment device on the incidence and severity of oral pressure injuries in the intensive care unit: a retrospective observational study. *BMC Nurs*, 2018; 17: 4.
33. Worsley PR, Prudden G, Gover G, Bader D. Investigating the effects of strap tension during non-invasive ventilation mask application: A combined biomechanical and biomarker approach. *Med Devices (Auckl)*, 2016; 9: 409-16.
34. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis
35. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
36. Mathison CJ. Skin and wound care challenges in the hospitalized morbidly obese patient. *J Wound Ostomy Continence Nurs*, 2003; 30(2): 78-83.
37. Murray J, Noonan C, Quigley S, Curley M. Medical device-related hospital-acquired pressure ulcers in children: An intergrative review. *J Pediatr Nurs*, 2013; 28(6): 585-595.

38. Webber-Jones JE, Thomas CA, Bordeaux Jr RE. The management and prevention of rigid cervical collar complications. *Orthopaedic Nursing*, 2002; 21(4): 19-27.
39. Powers J, Daniels D, McGuire C, Hilbish C. The incidence of skin breakdown associated with use of cervical collars. *J Trauma Nurs*, 2006; 13(4): 198-200.
40. O'Toole TR, Jacobs N, Hondorp B, Crawford L, Boudreau LR, Jeffe J, Stein B, LoSavio P. Prevention of tracheostomy-related hospital-acquired pressure ulcers. *OTO Open*, 2017; 156(4): 642-651.
41. Whitley AB, Nygaard RM, Endorf FW. Reduction of pressure-related complications with an improved method of securing endotracheal tubes in burn patients with facial burns. *J Burn Care Res*, 2017; 31.
42. Weng M. The effect of protective treatment in reducing pressure ulcers for non-invasive ventilation patients. *Intensive Crit Care Nurs*, 2008; 24(5): 295-299.
43. Günlemez A, Isken T, Gökalp A, Türker G, Arisoy E. Effect of silicon gel sheeting in nasal injury associated with nasal CPAP in preterm infants. *Indian Pediatr*, 2010; 47: 265-267.
44. Forni C, Loro L, Tremosini M, Mini S, Pignotti E, Bigoni O, Guzzo G, Bellini L, Trofa C, Guzzi M. Use of polyurethane foam inside plaster casts to prevent the onset of heel sores in the population at risk. A controlled clinical study. *J Clin Nurs*, 2011; 20(5/6): 675-680.
45. Kuo C, Wootten CT, Tylor D, Werkhaven J, Huffman K, Goudy S. Prevention of pressure ulcers after pediatric tracheostomy using a Mepilex Ag dressing. *Laryngoscope*, 2013: doi:10.1002/lary.24094.
46. Santamaria N, Gerdtz M, Liu W, Rakis S, Sage S, Ng AW, Tudor H, McCann J, Vassiliou J, Morrow F, Smith K, Knott J, Liew D. Clinical effectiveness of a silicone foam dressing for the prevention of heel pressure ulcers in critically ill patients: Border II Trial. *J Wound Care*, 2015; 24(8): 340-345.
47. Santamaria N, Gerdtz M, Sage S, McCann J, Freeman A, Vassiliou T, De Vincentis S, Ng AW, Manias E, Liu W, Knott J. A randomised controlled trial of the effectiveness of soft silicone multi-layered foam dressings in the prevention of sacral and heel pressure ulcers in trauma and critically ill patients: The Border trial. *Int Wound J*, 2015; 12(3): 302-308.
48. Clay P, Cruz C, Ayotte K, Jones J, Fowler SB. Device related pressure ulcers Pre and post identification and intervention. *J Pediatr Nurs*, 2018; 31: 31.
49. Huang TT, Tseng CE, Lee TM, Yeh JY, Lai YY. Preventing pressure sores of the nasal ala after nasotracheal tube intubation: From animal model to clinical application. *J Oral Maxillofac Surg*, 2009; 67(3): 543-551.
50. Bliss DZ, Gurvich O, Savik K, Eberly LE, Harms S, Mueller C, Wyman JF, Garrard J, Virnig B. Are There Racial-Ethnic Disparities in Time to Pressure Ulcer Development and Pressure Ulcer Treatment in Older Adults After Nursing Home Admission? *J Aging Health*, 2014.
51. Call E, Pedersen J, Bill B, Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Clark M. Enhancing pressure ulcer prevention using wound dressings: what are the modes of action? *Int Wound J*, 2013; epub.
52. Call E, Pedersen J, Bill B, Oberg C, Ferguson-Pell M. Microclimate impact of prophylactic dressings using in vitro body analog method. *Wounds* 2013; 25(4): 94-103.
53. McNichol L, Lund C, Rosen T, M. G. Medical adhesives and patient safety: state of the science: consensus statements for the assessment, prevention, and treatment of adhesive-related skin injuries. *Orthopaedic Nursing* 2013; 32(5): 267-81
54. Cutting K. Impact of adhesive surgical tape and wound dressings on the skin, with reference to skin stripping. *Journal of Wound Care*, 2008; 17(4): 157-62.
55. Newnam KM, McGrath JM, Salyer J, Estes T, Jallo N, Bass WT. A comparative effectiveness study of continuous positive airway pressure-related skin breakdown when using different nasal interfaces in the extremely low birth weight neonate. *Appl Nurs Res*, 2015; 28(1): 36-41.
56. Ham WHW, Schoonhoven L, Schuurmans MJ, Leenen LPH. Pressure ulcers, indentation marks and pain from cervical spine immobilization with extrication collars and headblocks: An observational study. *Injury*, 2016; 47: 1924-1931.
57. Ham WHW, Schoonhoven L, Schuurmans MJ, Leenen LP. Pressure ulcers in trauma patients with suspected spine injury: A prospective cohort study with emphasis on device-related pressure ulcers. *Int Wound J*, 2016.
58. Tescher AN, Rindfleisch AB, Youdas JW, Terman RW, Jacobson TM, Douglas LL, Miers AG, Austin CM, Delgado AM, Zins SM, Lahr BD, Pichelmann MA, Heller SF, Huddleston PM. Comparison of cervical range-of-motion restriction and craniofacial tissue-interface pressure with 2 adjustable and 2 standard cervical collars. *Spine (Phila Pa 1976)*, 2016; 41(6): E304-E312.
59. Panczykowski DM, Stone JG, Okonkwo DO, *The Management of Traumatic Spinal Cord Injury*, in *Neurocrit Care*, L. Shutter and B. Molyneaux, Editors. 2018, Oxford University Press.
60. Yorkgitis B, McCauley D. Cervical spine clearance in adult trauma patients. *Journal of the American Academy of PAs*; 32(2).
61. Patel M, Como J, Haut E. The Devil Is in the Details When Removing Cervical Collars After Blunt Trauma. *JAMA Surg*, 2018; 153(7): 632-633.
62. Rogers L. Rigid cervical collar in pre-hospital care. *Journal of Paramedic Practice*, 2017; 9(6): 1-5.

CLASSIFICATION OF PRESSURE INJURIES

Introduction

A pressure injury is defined as localized damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear. Pressure injuries usually occur over a bony prominence but may also be related to a device or other object. Open wounds from various etiologies (e.g., venous ulcers, arterial ulcers, neuropathic ulcers, incontinence associated dermatitis, skin tears and intertrigo) may appear similar to a pressure injury. The treatment of any wound begins with a comprehensive understanding of its etiology. Therefore, differentiating pressure injuries from other wounds is the first step in assessing the wound and developing a treatment plan.

As with many other types of wounds, terminology has been developed to describe the severity of pressure injuries. A pressure injury classification system describes the extent of skin and tissue damage presenting as a pressure injury. Numerous classification systems have been developed and used over the years, informed by the evolving understanding of the etiology of pressure injuries. Anatomical knowledge of the skin, subcutaneous fat, fascia and muscle layers (as well as supporting structures such as tendon, ligament and bone) is essential to accurate classification. The type (histology) and depth of tissues may vary depending on the anatomical site. Current classification systems are based on visual inspection of types of tissue and to some extent palpation for tissue consistency and temperature differences. Unfortunately, the true extent of tissue injury cannot always be detected with visual and palpable assessment. This is an inherent limitation of all current classification systems. There are several assessment technologies that may improve diagnostic accuracy in the future. These are described in the chapter on *Skin and Tissue Assessment*.

This chapter will discuss differential diagnosis and classification of pressure injuries and provide an overview of classification systems that are commonly used around the world to identify the extent and type of tissue damage of presenting pressure injuries. Terminology and classification systems continue to evolve as more research informs knowledge of pressure injury etiology. Throughout this guideline terminology from the International NPUAP/EPUAP Pressure Ulcer Classification System (2014)¹ is used to describe Category/Stage of pressure injuries. However, the term 'injury' has been adopted in place of 'ulcer' due increasing international recognition.^{2,3} *Tables 12.5 and 12.6* in this chapter provide a crosswalk between terminologies used across different geographic and clinical settings.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are the most commonly recognized and used pressure injury classification systems and how do they relate to one another?
- What are the recognized characteristics of each pressure injury category (i.e. Category/Stage I to IV, unstageable pressure injury and deep tissue pressure injury or suspected deep tissue injury)?

Differential Diagnosis

**9.1: Differentiate pressure injuries from other types of wounds.
(Good Practice Statement)**

Implementation Considerations

- Identify the most likely etiology of the wound. In the case of pressure injuries, the injured area will usually be on a pressure loading surface in an individual with a history of mobility impairment or will be under a medical device (*Expert opinion*).
- Provide education on the etiology and clinical presentation of wounds commonly confused with pressure injuries to enhance diagnostic accuracy (*Expert opinion*). Refer to the guideline chapter *Health Professional Education* for more information on the role of education in assessment of pressure injuries.
- Conduct a comprehensive assessment of the individual to inform differential diagnosis (*Expert opinion*). The guideline chapter *Assessment of Pressure Injuries and Monitoring Healing* provides recommendations on factors to consider when assessing the individual with a wound.

- When applicable (e.g., particularly for lower limb wounds), conduct a vascular assessment to inform differential diagnosis (*Expert opinion*). The guideline chapter *Heel Pressure Injuries* includes recommendations on performing vascular assessment.

Discussion

Accurate assessment of wound etiology is essential for the development of an appropriate and comprehensive treatment plan that addresses underlying pressure injury risk factors. Accurate assessment of wounds and identification of their etiology is also important to inform the development and evaluation of pressure injury quality improvement programs, track pressure injury incidence and prevalence, evaluate quality indicators and, in some geographic jurisdictions, to calculate reimbursement and/or fines applied to facilities.⁴

Assessment of wound etiology is informed by the presentation of the wound, including its anatomical location and visual appearance. For example, wounds appearing over a bony prominence are more likely to be associated with pressure and shear, indicating a possible pressure injury.⁴ Differentiation is also informed by a comprehensive assessment of the individual to identify comorbidities related to wound development and wound healing (e.g., diabetes mellitus, vascular disease or malnutrition) and to evaluate pressure injury risk factors (e.g., mobility and continence). Environmental factors, particularly the presence of devices or equipment, are also a consideration in determining the etiology of the wound. It A device related pressure injury usually conforms to the shape of the device, equipment or furniture that has applied pressure to the skin and tissue.⁵

Hart et al. (2006)⁶ reported on a study of the accuracy of nurses' assessments of the etiology of pressure injuries and other open wounds. The participants (n = 256) evaluated seven photographs of wounds, including pressure injuries, arterial ulcers and diabetic foot ulcers. The participants ranged in experience, but only 16% were described as wound care/skin nurses and 17% were reported to have certification in wound, ostomy and continence care. There was moderate agreement between the participants on identification of wound type ($\kappa = 0.56$, standard deviation (SD) = 0.22). Identification of pressure injuries was more accurate than differentiation of other wound etiologies, and agreement increased when limiting the analysis to nurses specialized in wound care ($\kappa = 0.92$, SD = 0.15 for wound specialist nurses). This study highlights the importance of ensuring access to appropriately trained health professionals and clinical leaders specialized in wound care (see the guideline chapter *Implementing Best Practice in Clinical Settings*). This study was conducted in 2006 and is reported to be one of the earliest studies that utilized web-based digital photography for testing differential wound diagnosis and pressure injury classification. The quality and presentation of the images (e.g. the monitors used by nurses in their facilities) may have influenced the findings, although the researchers attempted to address this potential by providing contextual information (e.g., wound dimensions and depth) in a narrative (*Level 4*).

However, Mahoney et al. (2011)⁴ reported low agreement between nurses in differentiating wounds. In an online survey delivered to participants recruited through the WOCN Society, nurses (n = 100) classified nine color digital photographs of gluteal cleft and buttock wounds. Wound photographs presented to the nurse participants included pressure injuries, moisture lesions, incontinence-associated dermatitis and skin tears. There was an overall lack of consensus amongst the participants in identifying wound etiology ($\kappa = 0.1708$, 99% confidence interval [CI] 0.163 to 0.1786). Only one-third of the photographs achieved agreement on etiology above 75%. Although recruitment was through the WOCN Society, information on the participants was not reported, so the level of experience and education in wound differentiation is unclear (*Level 4*).

Two studies that explored the interrater reliability of the EPUAP classification system also included photographs of moisture-associated dermatitis.^{7,8} In the first study, photographs of pressure injuries and moisture associated dermatitis were presented to researchers, staff nurses and pressure injury nurses in order to develop a set of clear photographs for larger interrater and intrarater studies. In the second study, the 56 photographs that were selected in the earlier study were presented to 473 nurses to establish interrater reliability and 86 nurses who performed repeat assessments for the interrater reliability analysis. In both these studies the accuracy and reliability for nurses attempting to distinguish moisture-associated dermatitis from Category/Stage II pressure injuries was low. Moisture-associated dermatitis was incorrectly identified as a pressure injury in 44.3% of assessments^{7,8} (both *Level 4*). There is evidence that the accuracy of pressure injury classification can be improved with education, and training (see discussion below).⁹⁻¹⁴

Using a Pressure Injury Classification System

9.2: Use a pressure injury classification system to classify and document the level of tissue loss. (Good Practice Statement)

Implementation Considerations

- Consistently use the same pressure injury classification system to assess the pressure injury (*Expert opinion*).
- Use a pressure injury classification system to classify and document the level of tissue loss in a device-related pressure injury (*Expert opinion*).
- Do not use a pressure injury classification system to describe tissue loss in a mucosal membrane pressure injury (*Expert opinion*).
- Do not use a pressure injury classification system to describe tissue loss in other types of wounds (*Expert opinion*).
- When classifying Category/Stage I pressure injuries and suspected deep tissue injury in individuals with darkly pigmented skin, rely on assessment of skin temperature, sub-epidermal moisture, change in tissue consistency and presence of skin pain rather than identification of erythema. Assessment of these factors should also be included when evaluating the severity and extent of Category/Stage II to IV pressure injuries in individuals with darkly pigment skin^{15,16} (*Level 3*).

Discussion

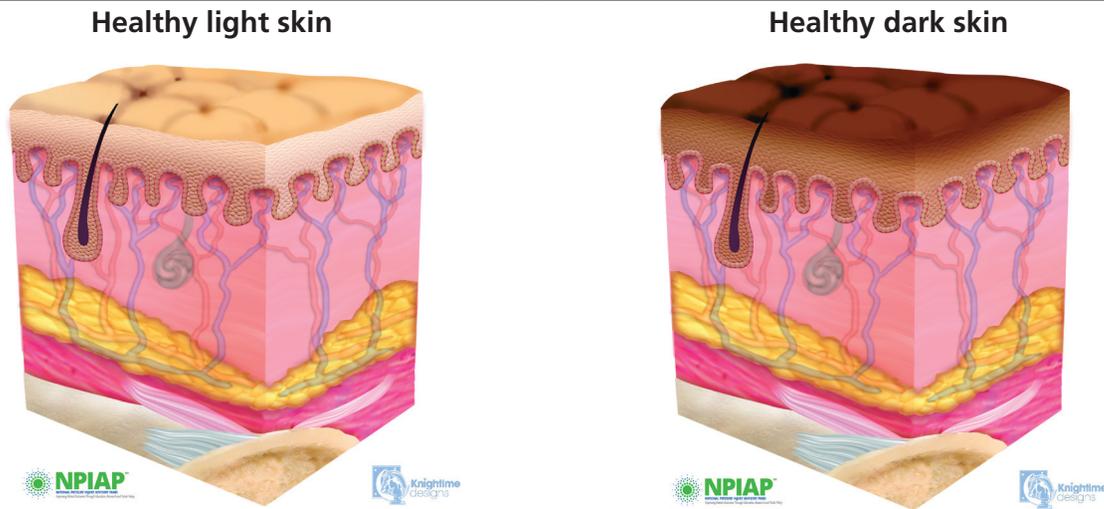
Pressure injuries are classified according to the amount of visible tissue loss using a pressure injury classification system. The use of a pressure injury classification system:

- Contributes to development of a pressure injury prevention plan
- Informs the selection of pressure injury treatments
- Improves communication between health professionals
- Allows for comparison of data between institutions
- Improves the methodological quality of pressure injury research.

Pressure injury classification systems describe the extent of tissue involvement of a pressure injury. *Table 12.1* presents illustrations of healthy skin, and tissue in deeper structures, identifying five layers:

- Epidermis
- Dermis
- Adipose tissue
- Muscle
- Bone

Table 12.2 presents photographs and illustrations of pressure injury Categories/Stages, demonstrating the extent of tissue loss indicative of each Category/Stage.

Table 12.1: Healthy skin (see the guideline Introduction for credits)

Pressure injury classification is based on the visual and palpatory identification of tissues including skin, subcutaneous fat, bone, muscle, tendon, and ligament. Necrotic tissue (slough and eschar) appears in full-thickness pressure injuries. Granulation tissue becomes present as a full-thickness pressure injury heals. In contrast, Category/Stage II pressure injuries do not have necrotic tissue and heal with epithelialization rather than granulation tissue. Healing tissues include scar, granulation tissue, and epithelium.

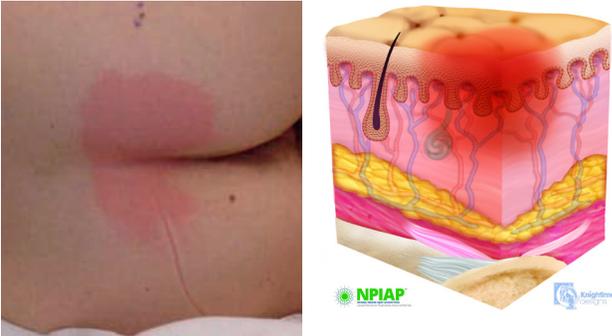
Pressure injury depth varies by anatomical site and relying on depth alone to determine whether a pressure injury is Category/Stage III or IV can be misleading. In anatomical locations with little adipose tissue (e.g., the bridge of the nose, the occiput, behind the ear, the sacrum, and the malleolus) shallow pressure injuries can be Category/Stage IV. In contrast, in anatomical locations with greater adipose tissue (e.g., buttocks and ischium) a pressure injury may be deep but not reach the muscle or bone, and therefore would be classified as a Category/Stage III pressure injury.

The description of a pressure injury should be supplemented with other findings. Indicating the exact anatomical location of the pressure injury is important, including clearly identifying location over a bony prominence as applicable. Historical information, such as the conditions under which the pressure injury developed, the history of prior treatment, and the trajectory of healing or non-healing of the pressure injury (if known) should be documented and considered when evaluating the effectiveness of the treatment plan. Loss of skin integrity over areas of previously healed full thickness pressure injuries may be difficult to classify. The National Pressure Injury Advisory Panel (NPIAP) recommends that these injuries be classified as “reopened, recurrent or new” depending on the length of time since the previous pressure injury closed and the maturation of the scar tissue.¹⁷

Classifying pressure injuries in darkly pigmented skin

Visual inspection of the skin and open wound may be augmented with an assessment of skin temperature and sub-epidermal moisture, especially in dark skinned individuals. As discussed in detail in the guideline chapter *Skin and Tissue Assessment*, areas of erythema are more difficult to identify and differentiate in darkly pigmented skin,¹⁸⁻²⁵ leading to a failure to detect Category/Stage I pressure injuries in dark skinned individuals.^{18,19,26} Additionally, identification of cellulitis might also be delayed or missed. Therefore, localized heat, edema/sub-epidermal moisture, change in tissue consistency in relation to surrounding tissue (e.g., induration/hardness), and localized skin pain are all important indicators of pressure damage in skin of darker tones. The guideline chapter on *Skin and Tissue Assessment* provides evidence-based recommendations on assessment techniques (e.g., skin temperature,¹⁵ sub-epidermal moisture measurement¹⁶ and skin tone color charts²⁷) that should be used when assessing the skin and tissues and classifying pressure injuries, particularly in darker skin tones. The guideline chapter on *Pain Assessment and Treatment* includes recommendations on assessing pain.

Table 12.2: Pressure injury Category/Stages: photographs and illustrations
(see the guideline Introduction for credits)

<p style="text-align: center;">Category/Stage I Pressure Injury</p>  <p>The photograph shows a localized area of non-blanchable redness on the skin. The illustration shows a cross-section of the skin with the epidermis intact but showing redness and some swelling in the superficial layers.</p>	<p style="text-align: center;">Category/Stage II Pressure Injury</p>  <p>The photograph shows a partial-thickness skin loss with a red, moist wound bed. The illustration shows a cross-section where the epidermis is partially missing, exposing the underlying dermis.</p>
<p style="text-align: center;">Category/Stage III Pressure Injury</p>  <p>The photograph shows a full-thickness skin loss with visible yellow, white, or tan slough or eschar. The illustration shows a cross-section where the epidermis and dermis are missing, exposing the subcutaneous fat.</p>	<p style="text-align: center;">Category/Stage IV Pressure Injury</p>  <p>The photograph shows a full-thickness skin loss with exposed muscle, bone, or other structures. The illustration shows a cross-section where the epidermis, dermis, and subcutaneous fat are missing, exposing the underlying muscle and bone.</p>
<p style="text-align: center;">Unstageable Pressure Injury (covered in eschar or slough)</p>  <p>The photograph shows a pressure injury completely covered by a thick, dark, necrotic eschar. The illustration shows a cross-section where the epidermis and dermis are missing, and the wound is filled with necrotic tissue, obscuring the underlying structures.</p>	<p style="text-align: center;">Suspected Deep Tissue Injury</p>  <p>The photograph shows a localized area of skin that is purple, magenta, or dark red, with a mottled appearance. The illustration shows a cross-section where the epidermis is intact but the underlying dermis and subcutaneous fat are dark purple, indicating deep tissue damage.</p>

Device related pressure injuries

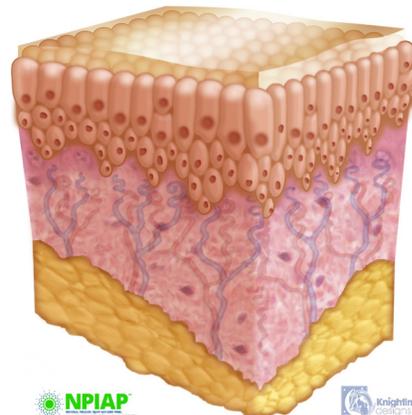
Device related pressure injuries result from medical devices, equipment, furniture and everyday objects that have applied pressure to the skin, either as an unintended consequence of their therapeutic use or inadvertently due to unintended skin-device contact. When the pressure injury is the result of a device designed and applied for diagnostic or therapeutic purposes it is referred to as a medical device related pressure injury (MDRPI). The resultant pressure injury generally conforms to the pattern or shape of the device.⁵ The term 'device related' describes the etiology of the pressure injury rather than its severity or extent of tissue loss.²⁸ Medical device related pressure injuries should be staged using a recognized classification system, as for other pressure injuries.²⁸

Mucosal membrane pressure injuries

Mucosal membrane pressure injuries are pressure injuries of the moist membranes that line the respiratory, gastrointestinal and genitourinary tracts.²⁹ Mucosal membrane pressure injuries are primarily caused by medical devices (generally tubing and stabilization equipment) exerting sustained compressive and shear forces on the mucosa. In the mucosa of the respiratory tract (i.e., lips, mouth, nasal passages, etc.), pressure injuries are typically caused by ventilation or feeding tubes and/or their stabilization equipment.²⁹ Gastrointestinal tract pressure injuries³⁰ and pressure injuries of the genitourinary tract (i.e., penile, ureteral, etc.)³¹ are primarily caused by feeding tubes or ostomy appliances and catheters.

Table 12.3: Mucous membrane pressure injury (see the guideline Introduction for credits)

Mucous Membrane Pressure Injury



Classification systems for pressure injuries of the skin and underlying tissues cannot be used to categorize mucosal pressure injuries.^{28,32} Where pressure is a significant factor in the etiology of the mucosal wound, it should still be considered a pressure injury; however, it is inappropriate to use a pressure injury classification system to categorize/stage. Non-blanchable erythema cannot be seen in mucous membranes. Shallow open mucosal ulcers indicating superficial tissue loss of the non-keratinized epithelium are so shallow that the naked eye cannot distinguish them from deeper, full thickness pressure injuries. Soft coagulum seen in mucosal pressure injuries looks like slough that is often present in Category/Stage III pressure injuries. However, this is actually a soft blood clot. Exposed muscle is rarely visible in mucosal pressure injuries. These factors render classification systems designed for pressure injuries of the skin inappropriate in the classification of mucosal pressure injuries.³³ More recently, development of the first classification system for mucosal membrane pressure injuries has been undertaken, with testing of reliability performed in an intensive care unit (ICU) setting.³⁴ The Reaper Oral Mucosa Pressure Injury Scale (ROMPIS), which describes three stages of mucosal membrane pressure injuries, showed moderate interrater reliability (α coefficient = 0.307, 95% CI 0.2000 to 0.409) when tested in 52 ICU nurses. Reliability was slightly higher in more experienced ICU nurses (α coefficient = 0.494). This classification system is still undergoing further validation.³⁴

**9.3: Verify that there is clinical agreement in pressure injury classification amongst the health professionals responsible for classifying pressure injuries.
(Good practice Statement)**

Implementation Considerations

- Include use of a pressure injury classification system in pressure injury related education³⁵ (*Level 4*). Refer to the guideline chapter *Health Professional Education* for more information on the role of education in classification of pressure injuries.
- Establish interrater reliability of pressure injury classification determinations for pressure injury prevalence and incidence studies (*Expert opinion*). Refer to the guideline chapter *Measuring Pressure Injury Prevalence and Incidence*.
- Consider two-clinician verification of differential diagnosis and classification in both prevalence and incidence studies and routine clinical practice as needed (*Expert opinion*).

Discussion

Numerous published studies have examined clinical agreement in pressure injury categorization/staging and reported interrater reliability for various pressure injury classification systems (see *Table 12.4*). These studies have either compared bedside evaluations of wounds or evaluated assessments of pressure injuries based on photographs. Across studies, interrater reliability varied, but was generally good to excellent for all classification scales. As noted in *Table 12.4*, there appears to be limited variation in reliability of assessment based on the observer/rater's level of experience,^{8,35} although in most of the reported studies, observers were registered/accredited nurses or health professionals with specific expertise in wound care and/or pressure injuries.

Table 12.4: Reliability of categorization/staging using various pressure injury classification systems

Classification system	Type of observation	Observers/raters	Interrater reliability unless otherwise stated
NPUAP classification system	Clinical assessment	Nurses (n = 180, n = 591 observations)	$\kappa = 0.60$ for Category/Stages I to IV ³⁶ $\kappa = 0.61$ for Category/Stage II to IV ³⁶
		WOCNs in a hospital setting (n = 3)	$\kappa = 0.78$, $p < 0.39$ to 0.58 , $p < 0.001$ ³⁷ Agreement 55% to 62% ³⁷
	Photograph assessment	Nurses (n = 180, n = 591 observations)	$\kappa = 0.69$ for Category/Stages I to IV ³⁶
EPUAP classification system 1999	Photograph assessment	Wound researchers (n = 7; n = 56 photographs)	$\kappa = 0.80$, $p < 0.01$ ⁷
	Photograph assessment	RNs (n = 20; n = 56 photographs)	$\kappa = 0.80$, $p < 0.01$ ⁷
	Photograph assessment	Pressure injury nurses (n = 17; n = 56 photographs)	$\kappa = 0.78$, $p < 0.01$ ⁷
	Photograph assessment	Nurses (n = 473; n = 56 photographs)	$\kappa = 0.41$ to $\kappa = 0.50$ ⁸ Average agreement 55.6% ⁸
	Photograph assessment	Nurses and physicians in emergency department (n = 54; n = 20)	$\kappa = 0.58$ ¹²
	Photograph assessment	Nurses working in a spinal cord injury setting (n = 414; n = 50 photographs)	Agreement varied from 67% to 100% between Category/Stages ³⁸
Pressure Ulcer Card (PUC) classification system	Photograph assessment	Registered nurse (RN) pairs (n = 114 assessments)	$\kappa = 0.364$ to 0.637 by anatomical location ³⁵
	Photograph assessment	Enrolled nurse (EN) pairs (n = 114 assessments)	$\kappa = 0.322$ to 0.607 by anatomical location ³⁵
	Photograph assessment	RN and EN pairs (n = 228 assessments)	$\kappa = 0.394$ to 0.755 by anatomical location ³⁵

Classification system	Type of observation	Observers/raters	Interrater reliability unless otherwise stated
PRESSURE Trial classification tool	Clinical assessment	Research nurses and ward-based nurse (n = 378 pairs)	21% disagreement, 82% of the disagreements falling within one Category/Stage of each other ³⁹
Un-named classification system	Clinical assessment	Nurses in nursing homes (n = 344 pairs)	$\kappa = 0.97^{40}$
	Clinical assessment	Nurses in hospitals (n = 674 pairs)	$\kappa = 0.81^{40}$
	Clinical assessment	Nurses in home healthcare (n = 1,348 pairs)	$\kappa = 0.49^{40}$
N.E. One Can Stage digital system	Photograph assessment	Health professionals and students (n = 101)	intraclass coefficient [ICC] = 0.794, 95% CI 0.697 to 0.862 ¹⁰
N.E.1 Wound Assessment Tool	Photograph assessment	Registered nurses (n = 94; n = 30 photographs)	ICC = 0.892, 95% CI 0.840 to 0.927 ⁴¹

Improving the pressure injury classification skills of health professionals

Numerous studies provide evidence that health professional education increases accuracy of pressure injury classification. For example, Briggs (2006)⁹ established markedly improved categorization/staging by registered nurses after participation in an education program consisting of theoretical background information and practice classifying photographs of pressure injuries. Young et al. (2011)¹⁰ also found that participating in an education program was associated with significant improvements in classification/staging compared with pre-education for both health professionals (correct responses rates 63.5% versus 70.7%) and nursing students (52.3% versus 67%). Ham et al. (2015)¹² noted significant improvements in use of the EPUAP classification system by emergency department nurses to identify and categorize/stage pressure injuries (in photographs) following a lecture that included visual illustrations of pressure injuries.¹² Similar results were shown in a study by Lee et al. (2016)¹³ in which clinical nurses (n = 407) showed significant improvements in differential diagnostic ability ($p < 0.001$) and classification ($p < 0.001$) using pressure injury photographs. The education in this study consisted of a didactic lecture and case studies¹³ (all Level 5).

Beeckman et al. (2010)¹⁴ reported effectiveness of an education course called the Pressure Ulcer Classification (PUCLAS) education tool designed to improve differential diagnosis and pressure injury classification. Clinical nurses were randomly selected to either a control group (n = 559) that received 15 minutes of education on the EPUAP classification system or to the intervention group (n = 658) receiving the PUCLAS education package. The package included a 60 minute lecture and photographs and video of pressure injuries representing the classification definitions. Both groups undertook a pre- and post-test in which they classified 40 pressure injury and moisture-associated dermatitis photographs. There was no significant difference between the two groups in diagnostic and classification skills at baseline ($p = 0.82$); however, the intervention group showed superior skills compared to the control group following the intervention (62.8% versus 53%, $p < 0.0001$ odds ratio [OR] 1.50, 95% CI 1.40 to 1.61)¹⁴ (Level 5). Since this study, this education program has been further developed as an e-learning package (PuClas4).⁴²

Tschannen et al. (2016)¹¹ suggested that augmenting a standard didactic lecture with participation in a facility's monthly pressure injury risk, prevalence and incidence surveillance program significantly improves the ability of nursing students to correctly identify the Category/Stage of pressure injuries. In a comparative study, nursing students receiving both the lecture and practical skills education correctly identified pressure injuries (all Category/Stage) more often than nursing students receiving only the lecture 69.47% versus 60.29%, $p < 0.0001$). Improvements were also significant for identification of Category/Stage II pressure injuries ($p = 0.001$) and suspected deep tissue pressure injuries ($p = 0.006$)¹¹ (Level 5).

Commonly Used Pressure Injury Classification Systems

Little comparative data exists on the accuracy of different pressure injury classification systems. Russell et al. (2001)⁴³ examined the accuracy and precision of classification of 30 photographs of pressure injuries by pressure injury experts, tissue viability experts and clinical nurse specialists (n = 200). Two classification systems were explored — the EPUAP system and the full four-digit Stirling classification tool.⁴³ This study⁴³ demonstrated a lack of consensus between classifications using the two systems, with higher levels of consensus (61.9% versus 30.2%) when using the EPUAP system. However, this study explored classification systems that are no longer commonly used, and classification of pressure injuries has advanced considerably since this early 2000s study. Most notably, the study noted that individuals with specialist training in pressure injuries or tissue viability had higher levels of interest in furthering their knowledge on classification systems (*Level 4*).

Generally, a specific healthcare system tends to adopt a single pressure injury classification system. *Tables 12.5 and 12.6* present the most commonly used classification systems in Europe, the Pan Pacific and the US. These systems include those derived from classifications developed by the National Pressure Ulcer Advisory Panel (NPUAP, now NPIAP) in 1989, 2007 and 2016;^{28,44,45} the NPUAP-EPUAP International Classification System which was adapted from the 2007 NPUAP System and published in the 2009 and 2014 international guideline; and those developed by the World Health Organization (WHO) for international standards of disease reporting. In clinical settings, classification systems derived from NPIAP systems are most commonly used, while administrative coders usually adopt WHO-ICD classification systems. As noted in *Tables 12.5 and 12.6*, there are additional country-specific and setting-specific variations in terminology between classification systems. As our knowledge of pressure injury etiology and the clinical assessment has advanced, continuous improvements have been made in pressure injury classification systems. Current systems are similar, with minor differences in terminology and definitions. These systems are compared and contrasted below. Clinician are encouraged to use the system adopted by their healthcare system to ensure consistency within systems and comparability in reporting.

Table 12.5: Comparison of pressure injury definitions in classification systems commonly used in Europe and the Pan-Pacific (continues over page)

International NPUAP/EPUAP Pressure Ulcer Classification System (2009, 2014) ¹	WHO ICD-10 (2016) ⁴⁶	WHO ICD-11 (2018) ⁴⁷ <i>Synonyms: pressure ulcer, pressure injury, decubitus ulcer</i>	ICD-11-Australian Modified (AM) (2019) ⁴⁸
<p>Category/Stage I pressure ulcer: Non-blanchable erythema</p> <p>Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Category/Stage I may be difficult to detect in individuals with dark skin tones. May indicate "at risk" individuals (a heralding sign of risk).</p>	<p>L89.0 Stage I decubitus ulcer and pressure area</p> <p>The ulcer appears as a defined area of persistent redness (erythema) in lightly pigmented skin, whereas in darker skin tones, the ulcer may appear with persistent red, blue or purple hues, without skin loss. Decubitus [pressure] ulcer limited to erythema only</p>	<p>EH90.0 Pressure ulceration grade 1</p> <p>Pressure ulceration grade 1 is a precursor to skin ulceration. The skin remains intact but there is non-blanchable redness of a localized area, usually over a bony prominence. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. It can be difficult to detect in individuals with dark skin but affected areas may differ in color from the surrounding skin. The presence of pressure ulceration grade 1 may indicate persons at risk of progressing to frank ulceration.</p>	<p>L89.0 Pressure injury, stage I</p> <p>Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding areas. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue.</p>
<p>Category/Stage II pressure ulcer: partial thickness skin loss</p> <p>Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising. * This Category/Stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation.</p> <p><i>*Bruising indicates suspected deep tissue injury.</i></p>	<p>L89.1 Stage II decubitus ulcer</p> <p>Decubitus [pressure] ulcer with abrasion, blister, partial thickness skin loss involving epidermis and/or dermis, skin loss NOS</p>	<p>EH90.1 Pressure ulceration grade 2</p> <p>Pressure injury with partial thickness loss of dermis. It presents as a shallow open ulcer with a red or pink wound bed without slough or as a serum-filled or serosanguinous blister which may rupture. This category should not be used to describe skin tears, tape burns, incontinence associated dermatitis, maceration or excoriation.</p>	<p>L89.1 Pressure injury, stage II</p> <p>Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.</p>
<p>Category/Stage III: Full thickness skin loss</p> <p>Full thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling. The depth of a Category/Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.</p>	<p>L89.2 Stage III decubitus ulcer</p> <p>Decubitus [pressure] ulcer with full thickness skin loss involving damage or necrosis of subcutaneous tissue extending to underlying fascia</p>	<p>EH90.2 Pressure ulceration grade 3</p> <p>Pressure ulcer with full thickness skin loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. There may be undermining and tunneling into adjacent structures. The depth varies by anatomical location: grade 3 pressure ulcers can be shallow in areas with little or no subcutaneous fat (e.g. bridge of the nose, ear, occiput and malleolus). In contrast, grade 3 pressure ulcers can be extremely deep in areas of significant adiposity.</p>	<p>L89.2 Pressure injury, stage III</p> <p>Full thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss.</p>

<p>International NPUAP/EPUAP Pressure Ulcer Classification System (2009, 2014)¹</p>	<p>WHO ICD-10 (2016)⁴⁶</p>	<p>WHO ICD-11 (2018)⁴⁷ <i>Synonyms: pressure ulcer, pressure injury, decubitus ulcer</i></p>	<p>ICD-11-Australian Modified (AM) (2019)⁴⁸</p>
<p>Category/Stage IV pressure ulcer: Full thickness tissue loss</p>	<p>L89.3 Stage IV decubitus ulcer</p>	<p>EH90.3 Pressure ulceration grade 4</p>	<p>L89.3 Pressure injury, stage IV</p>
<p>Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling. The depth of a Category/Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Category/Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.</p>	<p>Decubitus [pressure] ulcer with necrosis of muscle, bone or supporting structures (i.e., tendon or joint capsule)</p>	<p>Pressure ulcer with visible or directly palpable muscle, tendon or bone as a result of full thickness loss of skin and subcutaneous tissue. Slough or eschar may be present. The depth varies by anatomical location: grade IV pressure ulcers can be shallow in areas with little or no subcutaneous fat (e.g. bridge of the nose, ear, occiput and malleolus) but are typically deep and often undermine or tunnel into adjacent structures.</p>	<p>Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling. The depth of a stage IV pressure injury varies by anatomical location.</p>
<p>Unstageable: Depth unknown</p>	<p>No comparable classification</p>	<p>EH90.5 Pressure ulceration, ungradable</p>	<p>L89.4 Pressure injury, unstageable, so stated</p>
<p>Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough or eschar is removed to expose the base of the wound, the true depth, and therefore Category/ Stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover' and should not be removed.</p>	<p>—</p>	<p>Pressure ulcer with full thickness skin 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 are removed to expose the base of the wound, it is not possible to determine whether the ulcer is grade 3 or grade 4.</p>	<p>Full-thickness skin and tissue loss in which the base of the injury is covered by slough (yellow, tan, grey, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough or eschar is removed to expose the base of the wound, the stage cannot be determined. Excludes pressure injury reclassified to stage III of IV after exposure/debridement.</p>
<p>Suspected deep tissue injury: Depth unknown</p>	<p>No comparable classification</p>	<p>EH90.4 Suspected deep pressure-induced tissue damage, depth unknown</p>	<p>L89.5 Suspected deep tissue injury, depth unknown, so stated</p>
<p>Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. 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 optimal treatment.</p>	<p>—</p>	<p>An area of soft tissue damage due to pressure or shear which is anticipated to evolve into a deep pressure ulcer but has not yet done so. The affected skin is typically discolored purple or maroon and may display hemorrhagic blistering. It may be painful and edematous. It can be either warmer or cooler than adjacent tissue. Evolution into a deep ulcer may be rapid even with optimal treatment.</p>	<p>Purple or maroon localised area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. 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 optimal treatment. Excludes pressure injury reclassified to stage I of IV after exposure/debridement.</p>

Table 12.6: Comparison of pressure injury definitions in classification systems commonly used in the US (continues over page)

<p>NPUAP Classification System^a (April 2016)^{28,49}</p> <p>Pressure Injury: A pressure injury is localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.</p> <p>Stage 1 Pressure Injury: Non-blanchable erythema of intact skin</p> <p>Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.</p>	<p>U.S. CMS Long-Term Care^v (October 1, 2019)⁵⁰</p> <p>Pressure Ulcer/Injury: A pressure ulcer/injury is localized injury to the skin and/or underlying tissue, usually over a bony prominence, as a result of intense and/or prolonged pressure or pressure in combination with shear. The pressure ulcer/injury can present as intact skin or an open ulcer and may be painful. (Page M-4)</p> <p>Stage 1 Pressure Injury (M0300A)</p> <p>An observable, pressure-related alteration of intact skin whose indicators, as compared to an adjacent or opposite area on the body, may include changes in one or more of the following parameters: skin temperature (warmth or coolness); tissue consistency (firm or boggy); sensation (pain, itching); and/or a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones, the injury may appear with persistent red, blue, or purple hues. (Page M-11)</p>	<p>U.S. ICD-10-CM (2020 Release) Oct 1, 2019 –Sept 30, 2020⁵¹</p> <p>The ICD-10-CM classifies pressure ulcer stages based on severity, which is designated by stages 1-4, deep tissue pressure injury, unspecified stage, and unstageable. (Page 55)</p> <p>Stage 1 Pressure Ulcer (L89.XX1)</p> <p>Definition unchanged from previous ICD-10 versions. XX=code for anatomical location.</p>
<p>Stage 2 Pressure Injury: Partial-thickness skin loss with exposed dermis</p> <p>Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and heel in the heel. This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSi), or traumatic wounds (skin tears, burns, abrasions).</p>	<p>Stage 2 Pressure Ulcer (M0300B)</p> <p>Partial thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed, without slough or bruising. May also present as an intact or open/ ruptured blister. (Page M-12)</p>	<p>Stage 2 Pressure Ulcer (L89.XX2)</p> <p>Definition unchanged from previous ICD-10 versions. XX=code for anatomical location.</p>
<p>Stage 3 Pressure Injury: Full-thickness skin loss</p> <p>Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.</p>	<p>Stage 3 Pressure Ulcer (M0300C)</p> <p>Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining or tunneling. (Page M-14)</p>	<p>Stage 3 Pressure Ulcer (L89.XX3)</p> <p>Definition unchanged from previous ICD-10 versions. XX=code for anatomical location.</p>
<p>Stage 4 Pressure Injury: Full-thickness skin and tissue loss</p> <p>Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.</p>	<p>Stage 4 Pressure Ulcer (M0300D)</p> <p>Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often includes undermining and tunneling. (Page M-18)</p>	<p>Stage 4 Pressure Ulcer (L89.XX4)</p> <p>Definition unchanged from previous ICD-10 versions. XX=code for anatomical location.</p>

<p>NPUAP Classification System^a (April 2016)^{28,49}</p>	<p>Unstageable Pressure Injury: Obscured full-thickness skin and tissue loss</p> <p>Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.</p>	<p>U.S. CMS Long-Term Care^y (October 1, 2019)⁵⁰</p> <p>Unstageable Pressure Ulcer Related to Slough and/or Eschar (M0300F)</p> <p>Known but not stageable due to coverage of wound bed by slough or eschar. Slough tissue: Non-viable yellow, tan, gray, green or brown tissue; usually moist, can be soft, stringy and mucinous in texture. Slough may be adherent to the base of the wound or present in clumps throughout the wound bed. Eschar Tissue: Dead or devitalized tissue that is hard or soft in texture; usually black, brown, or tan in color, and may appear scab-like. Necrotic tissue and eschar are usually firmly adherent to the base of the wound and often the sides/ edges of the wound. (Page M-21)</p>	<p>U.S. ICD-10-CM (2020 Release) Oct 1, 2019 -Sept 30, 2020⁵¹</p> <p>Unstageable Pressure Ulcer (L89.XX0)</p> <p>Assignment of the code for unstageable pressure ulcer (L89.XX0) should be based on the clinical documentation. These codes are used for pressure ulcers whose stage cannot be clinically determined (e.g., the ulcer is covered by eschar or has been treated with a skin or muscle graft). This code should not be confused with the codes for unspecified stage (L89.XX9). When there is no documentation regarding the stage of the pressure ulcer, assign the appropriate code for unspecified stage (L89.XX9). (Page 55)</p>
<p>Deep Tissue Pressure Injury: Persistent non-blanchable deep red, maroon or purple discoloration</p> <p>Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.</p>	<p>Unstageable Pressure Injury Related to Deep Tissue Injury (M0300G)</p> <p>Deep Tissue Injury: Purple or maroon area of discolored intact skin due to damage of underlying soft tissue. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue. (Page M-24)</p>	<p>Pressure-induced Deep Tissue Damage (L89.XX6)</p> <p>For pressure-induced deep tissue damage or deep tissue pressure injury, assign only the appropriate code for pressure-induced deep tissue damage (L89.XX6). (Page 56)</p>	
<p>No comparable classification</p>	<p>Unstageable Pressure Ulcers/ Injuries Related to Non-removable Dressing/ Device (M0300E)</p> <p>Known but not stageable due to non-removable dressing/device. Non-removable Dressing/Device: Includes, for example, a primary surgical dressing that cannot be removed, an orthopedic device, or cast. (Page M-20)</p>	<p>No comparable classification</p>	

^y Although definitions for long-term care have been provided, CMS has harmonized pressure ulcer/injury definitions across all post-acute care settings

References

1. National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), Pan Pacific Pressure Injury Alliance (PPPIA), Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. 2014: Emily Haesler (Ed.) Cambridge Media: Osborne Park, WA.
2. Ayello EA, Cordero GM, Sibbald RG. Survey Results from Canada and Some Latin America Countries: 2016 National Pressure Ulcer Advisory Panel Changes in Terminology and Definitions. *Adv Skin Wound Care*, 2017; 30(2): 71-76.
3. Ayello EA, Delmore B, Smart H, Sibbald RG. Survey results from the Philippines: NPUAP changes in pressure injury terminology and definitions. *Adv Skin Wound Care*, 2018; 31(1): 601-606.
4. Mahoney M, Rozenboom B, Doughty D, Smith H. Issues related to accurate classification of buttocks wounds. *J Wound Ostomy Continence Nurs*, 2011; 38(6): 635-642.
5. Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Call E, Clark M. Use of wound dressings to enhance prevention of pressure ulcers caused by medical devices. *Int Wound J*, 2015; 12: 322-327.
6. Hart S, Bergquist S, Gajewski B, Dunton N. Reliability testing of the National Database of Nursing Quality Indicators pressure ulcer indicator. *J Nurs Care Qual*, 2006; 21(3): 256.
7. Defloor T, Schoonhoven L. Inter-rater reliability of the EPUAP pressure ulcer classification system using photographs. *J Clin Nurs*, 2004; 13(8): 952-959.
8. Defloor T, Schoonhoven L, Katrien V, Weststrate J, Myny D. Reliability of the European Pressure Ulcer Advisory Panel classification system. *J Adv Nurs*, 2006; 54(2): 189-198.
9. Briggs S-L. How accurate are RGNs in grading pressure ulcers? *Br J Nurs*, 2006; 15(22): 1230-1234.
10. Young DL, Estocado N, Landers MR, Black J. A pilot study providing evidence for the validity of a new tool to improve assignment of national pressure ulcer advisory panel stage to pressure ulcers. *Adv Skin Wound Care*, 2011; 24(4): 168-175.
11. Tschannen D, McKay M, Steven M. Improving pressure ulcer staging accuracy through a nursing student experiential intervention. *J Nurs Educ*, 2016; 55(5): 266-70.
12. Ham WHW, Schoonhoven L, Schuurmans MJ, Veugelers R, Leenen LPH. Pressure ulcer education improves interrater reliability, identification, and classification skills by emergency nurses and physicians. *J Emerg Nurs*, 2015; 41(1): 43-51.
13. Lee YJ, Kim JY. Effects of pressure ulcer classification system education programme on knowledge and visual differential diagnostic ability of pressure ulcer classification and incontinence-associated dermatitis for clinical nurses in Korea. *Int Wound J*, 2016; 13(1): 26-32.
14. Beeckman D, Schoonhoven L, Fletcher J, Furtado K, Heyman H, Paquay L, De Bacquer D, Defloor T. Pressure ulcers and incontinence-associated dermatitis: effectiveness of the Pressure Ulcer Classification education tool on classification by nurses. *Qual Saf Health Care*, 2010; 19(5): e3.
15. Farid K, Winkelman C, Rizkala A, Jones K. Using temperature of pressure-related intact discolored areas of skin to detect deep tissue injury: An observational, retrospective, correlational study. *Ostomy Wound Manage*, 2012; 58(8): 20-31.
16. Bates-Jensen BM, McCreath HE, Pongquan V. Subepidermal moisture is associated with early pressure ulcer damage in nursing home residents with dark skin tones: Pilot findings. *J Wound Ostomy Continence Nurs*, 2009; 36(3): 277-284.
17. Tew C, Hettrick H, Holden-Mount S, Grigsby R, Rhodovi J, Moore L, Ghaznavi AM, Siddiqui A. Recurring pressure ulcers: Identifying the definitions. A National Pressure Ulcer Advisory Panel white paper. *Wound Repair Regen*, 2014; 22(3): 301-304.
18. Baumgarten M, Margolis D, van DC, Gruber-Baldini A, Hebel J, Zimmerman S, Magaziner J. Black/White differences in pressure ulcer incidence in nursing home residents. *J Am Geriatr Soc*, 2004; 52(8): 1293-1298.
19. VanGilder C, MacFarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. *Ostomy Wound Manage*, 2008; 54(2): 40-54.
20. Meehan M. Multisite pressure ulcer prevalence survey. *Decubitus*, 1990; 3(4): 14-7.
21. Meehan M. National pressure ulcer prevalence survey. *Adv Wound Care*, 1994; 7(3): 27-30, 34, 36-8.
22. Lyder CH, Yu C, Stevenson D, Mangat R, Empleo-Frazier O, Emerling J, McKay J. Validating the Braden Scale for the prediction of pressure ulcer risk in blacks and Latino/Hispanic elders: a pilot study. *Ostomy Wound Manage*, 1998; 44(3A Suppl): 42S.
23. Lyder CH, Yu C, Emerling J, Mangat R, Stevenson D, Empleo Frazier O, McKay J. The Braden Scale for pressure ulcer risk: evaluating the predictive validity in Black and Latino/Hispanic elders. *Appl Nurs Res*, 1999; 12(2): 60-8.
24. Barczak CA, Barnett RI, Childs EJ, Bosley LM. Fourth national pressure ulcer prevalence survey. *Adv Wound Care*, 1997; 10(4): 18-26.
25. Baumgarten M, Margolis DJ, Selekof JL, Moye N, Jones PS, Shardell M. Validity of pressure ulcer diagnosis using digital photography. *Wound Repair Regen*, 2009; 17(2): 287-290.
26. Rosen J, Mittal V, Degenholtz H, Castle N, Mulsant B, Nace D, Rubin F. Pressure ulcer prevention in black and white nursing home residents: A QI initiative of enhanced ability, incentives, and management feedback. *Adv Skin Wound Care*, 2006; 19(5): 262-269.
27. McCreath HE, Bates-Jensen BM, Nakagami G, Patlan A, Booth H, Connolly D, Truong C, Woldai A. Use of Munsell color charts to measure skin tone objectively in nursing home residents at risk for pressure ulcer development. *J Adv Nurs*, 2016; 72(9): 2077-2085.
28. National Pressure Ulcer Advisory Panel. *NPUAP Pressure Injury Stages*. 2016 [cited September 2019]; Available from: https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/npuap_pressure_injury_stages.pdf.
29. Coyer FM, Stotts NA, Blackman VS. A prospective window into medical device-related pressure ulcers in intensive care. *Int Wound J*, 2014; 11(6): 656-64.
30. Teno JM, Gozalo P, Mitchell SL, Kuo S, Fulton AT, Mor V. Feeding tubes and the prevention or healing of pressure ulcers. *Arch Intern Med*, 2012; 172(9): 697-701.

31. Bell MA. Severe indwelling urinary catheter-associated urethral erosion in four elderly men. *Ostomy Wound Manage*, 2010; 56(12): 36-39.
32. National Pressure Ulcer Advisory Panel. *Mucosal Pressure Ulcers: An NPUAP Position Statement*. 2008; Available from: http://www.npuap.org/wp-content/uploads/2012/01/Mucosal_Pressure_Ulcer_Position_Statement_final.pdf.
33. Mucous Membrane Task Force of the NPUAP. *Mucosal Pressure Ulcers: An NPUAP Position Statement*. 2008 [cited October 2019]; Available from: https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/position_statements/mucosal_pressure_ulcer_posit.pdf.
34. Reaper S, Green C, Gupta S, Tiruvoipati R. Inter-rater reliability of the Reaper Oral Mucosa Pressure Injury Scale (ROMPIS): A novel scale for the assessment of the severity of pressure injuries to the mouth and oral mucosa. *Aust Crit Care*, 2017; 30(3): 167-171.
35. Bååth C, Hall-Lord M-L, Idvall E, Wiberg-Hedman K, Wilde Larsson B. Interrater reliability using Modified Norton Scale, Pressure Ulcer Card, Short Form-Mini Nutritional Assessment by registered and enrolled nurses in clinical practice. *J Clin Nurs*, 2008; 17(5): 618-626.
36. Bergquist-Beringer S, Gajewski B, Dunton N, Klaus S. The reliability of the National Database of Nursing Quality Indicators pressure ulcer indicator: A triangulation approach. *J Nurs Care Qual*, 2011; 26(4): 292-301.
37. Jesada EC, Warren JI, Goodman D, Iliuta RW, Thurkauf G, McLaughlin MK, Johnson JE, Strassner L. Staging and defining characteristics of pressure ulcers using photographs by staff nurses in acute care settings. *J Wound Ostomy Continence Nurs*, 2013; 40(2): 150-156.
38. Sarhan F. Use of digital images in the assessment and treatment of pressure ulcers in patients with spinal injuries in community settings. *J Telemed Telecare*, 2010; 16(4): 207-210.
39. Nixon J, Thorpe H, Barrow H, Phillips A, Andrea Nelson E, Mason SA, Cullum N. Reliability of pressure ulcer classification and diagnosis. *J Adv Nurs*, 2005; 50(6): 613-623.
40. Bours GJ, Halfens RJ, Lubbers M, Haalboom JR. The development of a national registration form to measure the prevalence of pressure ulcers in The Netherlands. *Ostomy Wound Manage*, 1999; 45(11): 28.
41. Lilly D, Estocado N, Spencer-Smith JB, Englebright J. Validation of the NE1 wound assessment tool to improve staging of pressure ulcers on admission by registered nurses. *J Nurs Meas*, 2014; 22(3): 438-50.
42. Beeckman D., European Pressure Ulcer Advisory Panel. *PuClas4 eLearning Module*. 2017 [cited October 2019]; Available from: <https://puclas4.ucvvgent.be/>.
43. Russell LJ, Reynolds TM. How accurate are pressure ulcer grades? An image-based survey of nurse performance. *J Tissue Viability*, 2001; 11(2): 67.
44. No author listed. Pressure ulcers prevalence, cost and risk assessment: consensus development conference statement-The National Pressure Ulcer Advisory Panel. *Decubitus*, 1989; 2(2): 24-28.
45. Black J, Baharestani MM, Cuddigan J, Dorner B, Edsberg L, Langemo D, Posthauer ME, Ratliff, C., Taler G, National Pressure Ulcer Advisory Panel. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. *Adv Skin Wound Care*, 2007; 20(5): 269-274.
46. World Health Organization. *ICD-10 (Version : 2016)*. 2016 [cited January 2019]; Available from: <https://icd.who.int/browse10/2016/en>.
47. World Health Organization. *ICD-11 for Mortality and Morbidity Statistics (Version : 04/2019)*. 2018 [cited January 2019]; Available from: <https://icd.who.int/browse11/l-m/en>.
48. National Centre for Classification in Health, *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification*. 11th ed. 2019.
49. Edsberg LE, Black JM, Goldberg M, McNichol L, Moore L, Sieggreen M. Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System: Revised Pressure Injury Staging System. *J Wound Ostomy Continence Nurs*, 2016; 43(6): 585-597.
50. Centers for Medicare & Medicaid Services, Long-Term Care Facility Resident Assessment Instrument 3.0 User's Manual. 2019. Version 1.17:129. 2019: https://downloads.cms.gov/files/mds-3.0-rai-manual-v1.17.1_october_2019.pdf.
51. Centers for Disease Control and Prevention, ICD-10-CM Official Guidelines for Coding and Reporting - FY 2020. 2019: https://www.cdc.gov/nchs/data/icd/10cmguidelines-FY2020_final.pdf.

ASSESSMENT OF PRESSURE INJURIES AND MONITORING OF HEALING

Introduction

Comprehensive assessment of the individual and their pressure injury informs the development of the most appropriate and comprehensive management plan. Ongoing monitoring of wound healing provides an evaluation of the pressure injury treatment plan, as well as the overall management plan. Effective assessment and monitoring of wound healing are based on scientific principles, as described in this section of the guideline.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are accurate and effective strategies for evaluating/assessing pressure injuries?
- What are accurate and effective strategies for monitoring healing over time?
- What are the most commonly recognized and used pressure injury assessment/monitoring tools/scales and how do they relate to one another?
- Which pressure injury monitoring tools are most responsive to change over time and most accurately describe the healing trajectory of the wound (i.e., healing, deteriorating, and stalled)?

Assessment of the Individual with a Pressure Injury

Assessment of the individual, their ability to heal and the risk for developing additional pressure injuries should be conducted, in addition to assessment of the pressure injury itself (see *Recommendations 10.4 to 10.8*).

10.1: Conduct a comprehensive initial assessment of the individual with a pressure injury.
(Good practice statement)

Implementation Considerations

- Include the following areas in a comprehensive assessment:
 - A complete health/medical, psychological and social history
 - A focused physical examination
 - Nutritional status
 - Pain related to pressure injuries
 - Risk for developing additional pressure injuries
 - Health related quality of life (HRQoL), self-care skills and knowledge
 - Functional capacity
 - Resources and supports available to the individual and the healing environment
 - Values and goals of care of the individual and their informal caregivers
 - Ability to adhere to a prevention and management plan (*Expert opinion*).
- A comprehensive assessment includes patient interview, a focused physical examination and laboratory tests (e.g., pathology tests) and radiological imaging as indicated.

Discussion

An assessment of the individual includes identification and assessment of comorbidities and other intrinsic factors that could contribute to the individual's ability to heal. This should include medications, nutritional status, vascular status, mobility and activity, posture, continence status and psychosocial status. Comprehensive guidance on specific areas of patient assessment are outlined in other chapters of the guideline, including:

- Vascular assessment, including perfusion and sensation (see the chapter *Heel Pressure Injuries*)
- Pain assessment (see the chapter *Pain Assessment and Treatment*)
- Nutritional status assessment (see the chapter *Nutrition in Pressure Injury Prevention and Treatment*)
- Pressure injury risk assessment (see the chapter *Risk Factors and Risk Assessment*)
- Mobility and activity (see the chapter *Repositioning and Early Mobilization*)
- Health-related QoL, psychosocial status and knowledge (see the chapter *Quality of Life, Self-care skills and Education*).

Assessment of the individual also includes an assessment of the environment and resources that will influence the individual's ability to heal. Consideration to resources available to promote healing is of particular significance to individuals living in the community who may not have easy access to equipment. The availability of pressure redistribution support surfaces, mobility aids and appropriate seating all contribute to the individual's healing environment. Access to wound care supplies may be limited in some healthcare settings. Considerations for the home environment are discussed in the guideline chapter on *Support Surfaces*.

Psychosocial factors, knowledge and beliefs about pressure injuries influence the individual's ability to adhere to a treatment plan and engage in self-care skills. For individuals living in the community, the support available from social networks should be evaluated. These factors are discussed in detail in the guideline chapter *Quality of Life, Self-care Skills and Education*.

Determining the wishes, goals and concerns of the individual and their informal caregiver is an important part of the patient assessment.¹⁻⁹

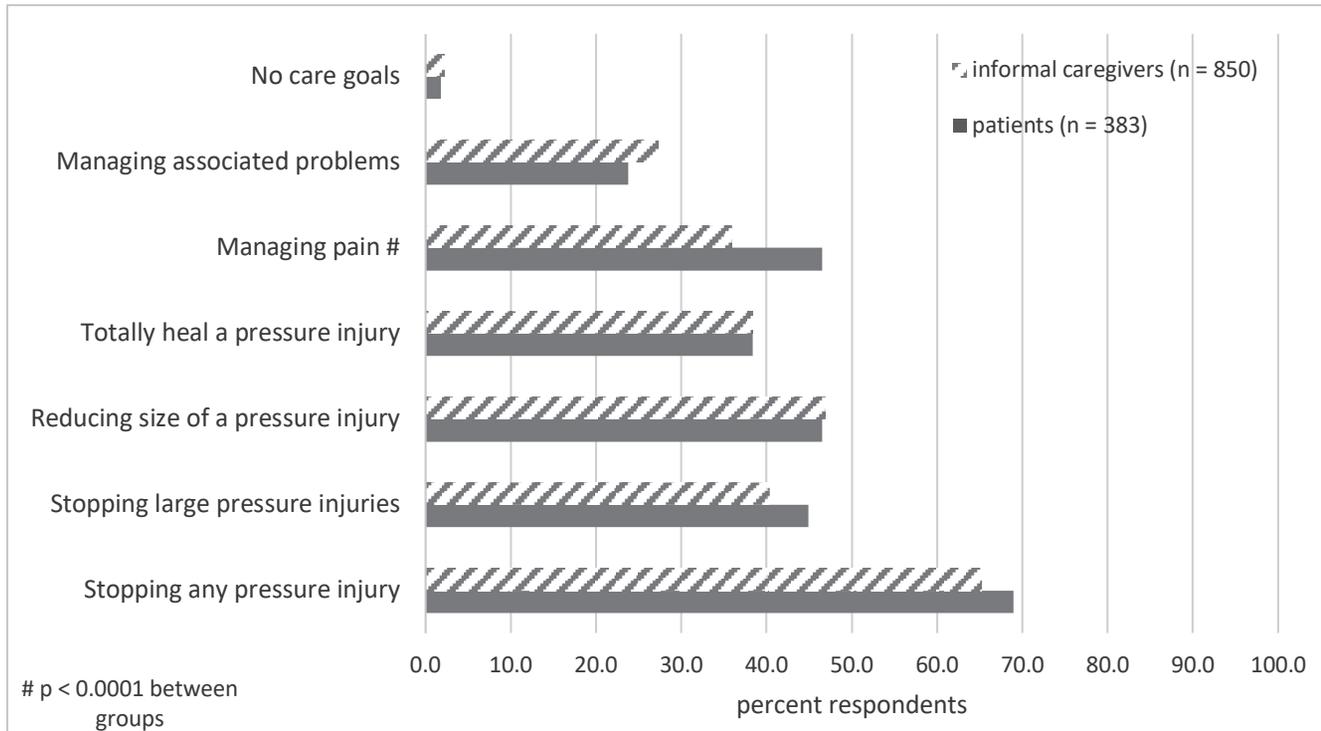
10.2: Set treatment goals consistent with the value and goals of the individual, with input from the individual's informal caregivers, and develop a treatment plan that supports these values and goals.
(Good practice statement)

Implementation Considerations

- The pressure injury treatment plan should be appropriate to the clinical condition of the pressure injury and address the goals of care. Local treatment options for pressure injuries are discussed in the guideline chapters on *Wound Care, Wound Dressings for Treatment of Pressure Injuries, Biological Dressings for Treatment of Pressure Injuries, Growth Factors for Treatment of Pressure Injuries and Biological Agents*.
- For individuals in end-of-life or palliative care, consider using a population-specific tool that has been tested for validity and reliability (e.g., Toronto Symptom Assessment System for Wounds) to determine concerns of the individual that should be addressed, including pain, exudate, odor, itchiness and cosmetic appearance¹⁰ (*Expert opinion*).
- If the pressure injury cannot be healed or treatment does not lead to complete closure/healing, consider other care goals including reduction in the pressure injury size,^{11,12} enhancement of quality of life¹³ and/or limiting the impact of the wound¹³ and associated problems (e.g., exudate and odor)^{11,12} (*Level 5*).

Discussion

As a part of this guideline development, an international survey of patient consumers and informal caregivers was undertaken to establish goals of care.^{11,12} There were 1,233 respondents (n = 383 individuals with or at risk of pressure injuries; n = 850 caring for such an individual) to the survey. Individuals were asked to select a maximum of three care goals (see *Figure 10.1*). Very few respondents identified that they did not have a goal of care; highlighting the importance of health professionals working with the individual and their informal caregivers to determine needs and values and to align care goals. The survey results indicated that with respect to treating pressure injuries, individuals with a pressure injury were more likely to have a goal to reduce the size of a pressure injury than to achieve total healing of a pressure injury. Managing pain was identified as a care goal as frequently as was reducing the size of a pressure injury. Significantly more patients than informal caregivers identified pain management as a goal of care (p < 0.0001), suggesting that the experience of pressure injury related pain might be overlooked by caregivers^{11,12} (*Level 5*).

Figure 13.1: Patient consumer and informal caregiver goals of care^{11,12}

Setting treatment goals in palliative/end-of-life care

If consistent with the individual's wishes, healing the pressure injury can be a care goal, even in the palliative care stages. If the pressure injury cannot be healed or treatment does not lead to closure/healing, focus on goals to enhance quality of life. Refer to the guideline chapter *Populations with Specific Pressure Injury Related Needs* for background discussion of the various needs of individuals in palliative care versus end-of-life care.

A multidisciplinary approach that addresses medical symptomology, nutrition and wound management may improve the potential for pressure injuries to heal in palliative care settings. Ruggeri et al. (2016)¹⁴ followed participants with advanced cancer who were admitted to a home palliative services with a pressure injury. In this study, 42.3% of all pressure injuries healed using a multidisciplinary management approach, with a further 46% having a reduction in pressure injury size (Level 4). Sankaran et al. (2015)¹⁵ found that when education was provided on hygiene, nutrition and repositioning to people with cancer receiving homecare (n = 108), 42.9% of pressure injuries healed and 23.8% reduced in size (Level 4).

Some, but not all, pressure injuries in individuals receiving end-of-life or palliative care will heal.^{16,17} Non-healing, chronic pressure injuries remain in an inflammatory state, further interfering with the potential to heal.¹⁸ However, Masaki et al. (2007)¹⁹ found no statistically significant difference for pressure injury healing time between individuals with and without cancer (Level 3). McNees and Meneses (2007)²⁰ analyzed 36,000 wound assessments, half conducted on participants with and half on participants without cancer. The two groups were sub-divided equally into those with and those without a pressure injury. They found that significantly more individuals without cancer had healed pressure injuries compared with those with cancer (78% versus 44%, p = 0.018). Individuals with cancer and non-healing wounds had significantly more risk factors than those with a wound that healed (mean 6.46 versus 2.78). However, it is important to note that pressure injuries *did* heal in 44% of participants with cancer²⁰ (Level 3).

An individual receiving end-of-life care whose body systems are shutting down often lacks the physiological resources necessary for complete healing of the pressure injury. As such, the goal of care may be to maintain or improve the status of the pressure injury rather than heal it.¹⁹ As the individual nears death, the skin may be one of the first organs to be compromised and may eventually fail along with other organs.¹³

In a prospective study of participants with advanced disease (n = 282), Maida et al. (2012)²¹ found that 18.9% of participants with Category/Stage I pressure injuries and 10.4% of participants with Category/Stage II pressure injuries achieved complete healing before death. However, only 4% (one participant) with a Category/Stage III pressure injury showed complete healing, and none of the participants with Category/Stage IV or unstageable pressure injuries achieved healing²¹ (Level 3). In a similar study, terminally ill nursing home residents receiving end-of-life care (n =

117, 64 of whom had pressure injuries) were followed to evaluate healing and factors that contributed to pressure injury development.²² This study found that some Category/Stage I, II or III pressure injuries healed before death (46%, 29.8% and 20% respectively); however, no Category/Stage IV or unstageable pressure injuries achieved healing by the time of death (*Level 4*).

Thus, while healing remains unlikely for some individuals receiving palliative care, it should not be assumed that all pressure injuries in people receiving end-of-life care will not heal. For individuals receiving end-of-life care, monitoring the pressure injury is an important step toward providing comfort, reducing wound pain, and addressing symptoms such as malodor and exudate. In many cases, the pressure injury may worsen as death approaches and as the individual's condition worsens. As the physical condition of the individual deteriorates, less frequent pressure injury assessment may assist in minimizing pain for the individual.

10.3: Conduct a comprehensive reassessment of the individual if the pressure injury does not show some signs of healing within two weeks despite appropriate local wound care, pressure redistribution, and nutrition. (Strength of Evidence = B2, Strength of Recommendation = ↑↑)

Evidence Summary

There is evidence from two Level 3 studies^{23,24} that pressure injuries receiving appropriate care will demonstrate signs of healing within two weeks. One study²³ indicated that Category/Stage III and IV pressure injuries demonstrate as much as 45% reduction in size within the first two weeks of treatment.

Implementation Considerations

- Expect some signs of pressure ulcer healing within two weeks^{23,24} (*Level 3*).
- Adjust expectations for healing in the presence of multiple factors that impair wound healing^{23,25-34} (*Level 4*).

Evidence Discussion

If progress toward healing is not seen within two weeks, the individual, the pressure injury, and the plan of care should be re-evaluated. General signs of healing include decreased length, width, and depth of the pressure injury; progressively less exudate; and changes in tissue type from less devitalized tissues (e.g., eschar and slough) to healthy regenerative tissues (e.g., granulation tissue and epithelialization). The health professional should be particularly alert to these signs when making a clinical judgment regarding the healing progress of the pressure injury.

There are no definitive answers regarding the time it takes a pressure injury to heal because contextual factors affecting healing vary from study to study, just as they do from individual to individual.³³ There is some evidence for using two weeks as a benchmark for expecting some healing. In a longitudinal study (n = 119 individuals with 153 pressure injuries), van Rijswijk et al. (1993)²³ noted that pressure injuries that did not show at least a 45% reduction in size at two weeks or a 77% reduction at four weeks were less likely to heal within the 15 month study duration. In this study, pressure injuries were treated with 3% hydrogen peroxide, saline rinse and a hydrocolloid dressing, with provision of pressure redistribution support surfaces and repositioning only provided to individuals who had received these interventions prior to study enrollment (*Level 3*). Although the pressure injury treatment described does not reflect current best practice, the study provides evidence that failure to show signs of healing within two weeks is an indicator that the pressure injury has a higher likelihood of taking extensive healing time without treatment review. In a second study²⁴ including Category/Stage III and IV pressure injuries (n = 48 individuals with 56 pressure injuries), the percent reduction in pressure injury surface area at two weeks of treatment was statistically significantly associated with likelihood of reaching complete healing (hazard ratio [HR] = 7.67, 95% CI 2.271 to 25.96. p = 0.01)²⁴ (*Level 3*).

Studies have noted that the largest increment in healing for pressure injuries occurs during the first three months, suggesting that signs of improvement should be evident in the early weeks of treatment.^{24,35} This is supported by a small analysis of Category/Stage IV pressure injury healing (n = 10), that reported the proportion of healing time taken to reach a 50% reduction in size was between 26.7% and 42.2% (variations based on initial wound size).²⁵

Variables influencing time taken for a pressure injury to heal

Healing rates and outcomes vary according to a myriad of factors. Some factors that could influence healing rates include:

- Category/Stage of the pressure injury^{32,33}
- Initial size of the pressure injury^{23,25-31,36}

- Nutritional status^{24,30,34}
- Co-morbidities³¹
- Presence of wound infection and biofilm (see the guideline chapter *Infection and Biofilms*)
- Adequacy of the treatment plan.^{26,30,33}

Category/Stage II pressure injuries take less time to heal than Category/Stage III and IV pressure injuries. *Table 13.1* summarizes healing times reported in the literature, noting the differences in healing duration based on Category/Stage. The analysis by Lynn et al. (2007)³² reporting healing times for pressure injuries in nursing home residents only included pressure injuries that had persisted for at least 30 days (i.e., pressure injuries healing in less than 30 days were excluded). The researchers noted that Category/Stage II pressure injuries in this study almost universally healed within a few weeks, leading to their exclusion from analysis and confounding the overall findings (*Level 4*). In the study by Bolton et al. (2004),³³ mean healing times were derived from those pressure injuries that achieved full healing within the 12 week study duration, which was 61% of Category/Stage II pressure injuries and 36% of Category/Stage III and IV pressure injuries ($p < 0.001$ between Category/Stages)³³ (*Level 4*).

Table 13.1: Pressure injury healing times reported in the literature (median days, unless otherwise stated)

Clinical setting	Category/Stage II	Category/Stage III and IV
Aged care ³² (<i>Level 4</i>)	51 – 52	140 – 150
Aged care, acute care and community-based care ³³ (<i>Level 4</i>)	31 ± 41	62 ± 54
Aged care ³¹ (<i>Level 3</i>)	46 (95% CI 27 – 36)	
Aged care, acute care and community-based care ²⁷ (<i>Level 3</i>)	18 (Average 22.9 [95% CI 20.47 – 25.37])	
Acute care, aged care and rehabilitation ²⁴ (<i>Level 4</i>)	69	

The initial size of the pressure injury also influences the time it will take to heal. In a retrospective study of 774 older adults with Category/Stage II pressure injuries ($n = 774$),³¹ the median time to healing a Category/Stage II pressure injury was 46 days (95% confidence interval [CI] 42 to 50). Bivariate survival analysis identified that the initial size of the pressure injury was significantly associated with time to reach complete healing, with small (≤ 1 cm²) pressure injuries taking 33 days (95% CI 27 to 36 days), medium (> 1 to 4 cm²) pressure injuries taking 53 days (95% CI 41 to 66 days) and large (> 4 cm²) pressure injuries taking 73 days (95% CI not established)³¹ (*Level 3*). Palese et al. (2015)²⁷ also noted that healing was associated with pressure injury size, with Category/Stage II pressure injuries of surface areas less than 3.1cm² having statistically significantly shorter average healing times than those of higher surfaces areas (19.2 days, 95% CI 1.6 to 21.8 days versus 31.0 days, 95% CI 26.4 to 35.6 days, $p < 0.001$)²⁷ (*Level 3*). In a longitudinal study by Edsberg et al. (2011)³⁶ investigating strategies to predict wound healing times, Category/Stage III and IV pressure injuries were assessed daily for ten days then weekly until study end (42 days). Pressure injury size at baseline was a significant predictor of time to heal ($p = 0.023$), with smaller pressure injuries taking less time to heal. Average daily healing was significantly related to with the initial pressure injury size ($p = 0.3537$)³⁶ (*Level 4*). Brown et al. (2000)²⁵ noted a strong association between baseline pressure injury size and healing rates for Category/Stage IV pressure injuries (correlation co-efficient = 0.806), however with only ten pressure injuries included, no conclusion can be drawn from this analysis (*Level 4*).

Studies reporting the influence of comorbidities and nutritional status on healing times report mixed findings.^{24,27,28,30} In the bivariate survival analysis conducted by Bergstrom et al. (2008),³¹ comorbidities and nutritional status that were significantly associated with time to complete healing of Category/Stage II pressure injuries included peripheral edema ($p = 0.006$), a temperature above 100°F (37.7°C; $p < 0.001$), agitation ($p < 0.001$), having oral eating problems ($p < 0.001$) and having obesity ($p = 0.03$). Other co-morbidities, including cognitive impairment, impaired ability to perform activities of daily living (ADLs), creatinine and albumin levels, diabetes mellitus or heart failure were not statistically significantly related to healing times (all $p > 0.05$)³¹ (*Level 3*). Modeling presented by van Rijswijk et al. (1994)²⁴ that included 56 Category/Stage III and IV pressure injuries noted that poor nutritional status at baseline was significantly related to less likelihood of reaching complete healing (HR = 0.21, 95% CI 0.052 to 0.85, $p = 0.02$). Being coherent (mean 54 ± 9.1 days) was also associated with faster healing than having confusion or disorientation (mean 139 ± 28.7 days, $p = 0.047$)²⁴ (*Level 3*). In an aged care setting, Bliss et al. (2017)²⁸ found that ADL deficits were significantly related to less likelihood of healing in an analysis of Category/Stage II to IV pressure injuries ($n = 10,861$). Additionally, the individual's racial/ethnic group was identified as being significantly associated with healing. Individuals with darkly pigmented skin had lower than expected healing rates compared to those with lighter skin²⁸ (*Level 3*). Palese et al. (2015)²⁷ reported a bivariate analysis including 270 Category/Stage II pressure injuries that found no statistically significant relationships between healing times and diabetes mellitus, steroid therapy and having two

or more comorbidities²⁷ (*Level 3*). Thus, comorbidities identified as influencing healing times differed across studies, and may be related to the clinical care, the severity of pressure injuries, and the selection of covariates included in each analysis.

Pressure Ulcer Assessment and Monitoring

10.4: Assess the pressure injury initially and re-assess at least weekly to monitor progress toward healing. (Good practice statement)

Implementation Considerations

- With each wound dressing change, observe the pressure injury for signs that indicate a change in treatment is required (e.g., wound improvement, wound deterioration, changes in exudate, signs of infection or other complications) (*Expert opinion*).
- Address signs of wound deterioration immediately (*Expert opinion*).
- Use the findings of pressure injury assessment to develop interventions that will best promote healing, and to evaluate the effectiveness of these interventions over time (*Expert opinion*).
- Consider further diagnostic investigations of wound bed tissue when healing does not progress according to expectations (e.g., tissue biopsies) (*Expert opinion*).
- Document pressure injury assessment in a manner that enables ongoing comparison to determine the wound's progress towards healing (see *Tools for Monitoring Pressure Injury Healing* below).
- Teach patients and their informal caregivers to monitor the condition of their pressure injury and to identify signs and symptoms that should be reported to the health professional (e.g., signs of local and systemic infection) (*Expert opinion*).

Discussion

A two-week period is recommended for evaluating progress toward healing. However, weekly assessments or more frequently if clinically indicated, provide an opportunity for the health professional to assess the pressure injury more regularly, detect complications as early as possible, and adjust the treatment plan accordingly. The health professional who undertakes the pressure injury assessment should be appropriately trained to undertake wound assessment, be familiar with the anatomy in the affected area and tissue types and be able to evaluate the progress of the wound toward healing.

Pressure injury assessment includes an evaluation of the pressure injury size and physical characteristics, both of which are discussed in more detail throughout this chapter. Infrequently (e.g., in more complex pressure injuries or those that have failed to heal over an extended time), additional diagnostic investigations may be required. Tissue biopsies can assist in differential diagnosis (e.g., if malignancy is suspected) and can improve understanding of the healing process and potential for healing. Differential expression levels of specific wound proteins assayed by mass spectrometry and multiplexed microassays are predictive of healing in the wound. In a longitudinal study, Edsberg et al. (2012)³⁷ identified significant differences in levels of 21 wound proteins in various parts (periphery versus interior) of the wound tissue between pressure injuries that healed and those that did not. For example, pyruvate kinase isozymes M1/M2, profilin-1, Ig lambda-1 chain C regions, and Ig gamma-1 chain C region, were in lower levels in the periphery of pressure injury wound beds compared to the interior of the wound bed. Keratin, type II cytoskeletal 6A (KRT6A), keratin, type I cytoskeletal 14, S100 calcium binding proteins A7, alpha1-antitrypsin precursor, hemoglobin subunit alpha, and hemoglobin subunit were seen in higher levels in the wound bed periphery compared to the interior of the wound bed (*Level 5*). Similarly, Taverna et al. (2015)³⁸ demonstrated different calcium modulated protein profiles between Category/Stage IV pressure injuries that did and did not heal. For example, calgranulin A was more detectable in non-healing wounds but not in healing wounds (*Level 5*).

Pressure injury status can change rapidly. Improvement or deterioration indicated by change in pressure injury dimensions, tissue quality or wound exudate levels, signs of infection (see the guideline chapter *Infection and Biofilms*) or other complications all provide indications of the effectiveness of the current treatment plan. Signs of deterioration should be addressed immediately. Individuals with pressure injuries and/or their informal caregivers, especially those in the community who have less frequent contact with a health professional, should be provided with information about the expected healing trajectory. Individuals should be made aware of how to identify signs of healing or deterioration in the pressure injury and alerted to signs and symptoms that should be brought to the health professional's attention.^{39,40}

Assessing and Monitoring the Size of the Pressure Injury

10.5: Select a uniform, consistent method for measuring pressure injury size and surface area to facilitate meaningful comparisons of wound measurements across time.
(Strength of Evidence = B2, Strength of Recommendation = ↑↑)

Evidence Summary

Evidence from two Level 4 studies of moderate⁴¹ and low⁴² quality indicated that wound tracing methods achieve similar results to ruler methods for calculating a pressure injury size. Evidence from a low quality Level 4 study⁴³ showed that two different methods of tracing the wound circumference and calculating the wound surface area achieved significantly different results for the wound surface area; however, both methods were equally effective in monitoring change in wound size over time. These studies suggest that various wound measurement methods are acceptable but using the same technique for repeated measures is important.

Implementation Considerations

- Cleanse the wound before surface measurement or probing for depth, undermining or tunneling (*Expert opinion*).
- Use standard aseptic technique/clean technique (rather than surgical aseptic technique) for measurement of most pressure injuries. Rulers that touch the surface of the wound should be clean and single-use to avoid cross-contamination of micro-organisms. Instruments or swabs that probe depth, undermining or tunnelling should be sterile (*Expert opinion*). Review the *Glossary* for definitions of asepsis.
- Use wound tracing in preference to the ruler method if the pressure injury is irregularly shaped^{42,44,45} (*Levels 3 and 4*).
- Position the individual in a consistent neutral position for wound measurement (*Expert opinion*).
- Document the individual's position during pressure injury measurement to enable replicability during repeated measurements (*Expert opinion*).
- Care should be taken to avoid causing tissue damage when probing the depth of the wound bed or determining the extent of undermining or tunneling (*Expert opinion*).
- Health professionals who perform digital wound measurement and photography for assessment and monitoring of healing should be appropriately trained in use of equipment (*Expert opinion*).
- Document wound measurements in a manner that enables ongoing comparison to determine the wound's progress towards healing. Valid and reliable tools should be used to monitor change in pressure injury size (see *Tools for Monitoring Pressure Injury Healing* below).

Evidence Discussion

Quantitative measurement of the pressure injury is the most accurate method to evaluate the wound size, including surface area and volume. Regular reassessment using the same measurement method allows for an objective evaluation of progress toward healing. For clinical practice, a method of wound measurement that balances validity, reliability, and clinical utility should be selected and consistently used. For research purposes, a more labor intensive method of measuring the wound may be desirable for greater precision.

Evidence comparing different wound measurement techniques suggests that there is good to excellent correlation between different methods of measuring the wound size and depth. In studies in which there is discrepancy between single measurements performed using different methods, the estimation of change in wound size over time was consistent between the methods. Thus, using a uniform and consistent method to measure the pressure injury is a greater imperative than choosing between different measurement techniques.

It is possible to distort soft tissue with variations in positioning yielding a larger or smaller measurement depending on position of the individual. Selecting a neutral position based on the anatomical location of the pressure injury increases accuracy of the measurement. Using the same position for repeated measures increases consistency. For example, it may be helpful to note that a sacral pressure injury was measured with the individual turned at a 90° angle on his/her hip with legs extended. Leg flexion and variations in the turning angle can distort tissue and result in very different measurements.

Techniques for measuring pressure injury size and/or surface area

Commonly used pressure injury measurement techniques include:

- Manually measuring the length and width of the wound (ruler method) using a consistent method (e.g., head-to-toe axis for length and perpendicular at 90° for width)
- Tracing the circumference of the wound onto transparent acetate film
- Taking a digital photograph of the wound and tracing the wound circumference
- Computer-assisted planimetry using a digital photograph and computerized software of the wound to trace or measure the wound circumference.

Using the ruler method, wound surface area is estimated by multiplying the length by the width of the wound. Using either tracing method, wound surface area is calculated using manual or digitized planimetry.

The ruler method assumes the wound is a standard shape. Ruler method generally overestimates surface area by 10% to 44%,⁴⁶ with accuracy increasing as the wound size increases. The ruler method that yields the least overestimation for various wound shapes is to measure the longest length of the pressure injury head-to-toe, and longest width side-to-side, perpendicular (at 90°) to the length.⁴¹ Measuring the longest length of the pressure injury (regardless of orientation) and a perpendicular width is more sensitive in monitoring wounds with changing shapes and configurations; however, this method increases the risk of overestimation, and potentially introduces variability in the selection of the longest length⁴¹ (*Level 4*).

Cutler et al. (1993)⁴² compared ruler method, computer-assisted planimetry from acetate film tracings and computer-assisted planimetry from digitized wound photographs. Measurements were made weekly for four weeks on Category/Stage III and IV pressure injuries (n = 17). There was a strong correlation (correlation co-efficient > 0.94, p = 0.01) between the measurement methods for determining the wound surface area. The mean difference in surface area was only 1.5cm², with the area slightly over-estimated using the ruler method⁴² (*Level 4*). Another study found that there was a range of accuracy for all wound measurement techniques, especially when measuring irregularly-shaped wounds, suggesting that different measurement methods are better suited to different wound shapes.⁴⁴ Bilgin et al. (2013)⁴⁵ confirmed these findings. In their study,⁴⁵ 80 pressure injuries were classified as larger and irregularly shaped or smaller and rounder/oval. All the pressure injuries were measured using ruler method, acetate film wound tracing with manual calculation of the wound area and thirdly, acetate wound tracing with digitized planimetry. There was a strong correlation between the three methods when measuring the regularly shape wounds (ICC = 0.95) than when comparing the irregularly shaped wounds (ICC = 0.75)⁴⁵ (*Level 3*). Wound tracing and planimetry provides a more accurate estimation of the surface area when measuring an irregularly shaped pressure injury.^{42,44,45}

An observational study⁴³ compared wound tracing on an acetate transparent film to digitized wound tracings (i.e., tracing of a photograph of the pressure injury) for determining the wound surface area of Category/Stage II or greater pressure injuries (n = 20). With both methods, the same software was used to calculate the wound area. Calculation of wound area was significantly different between the two measurement methods (p < 0.0001). However, the results also indicated that both methods were equally accurate in measuring improvement over time, with no significant difference in the calculation of wound improvement over one week (p = 0.9429). Neither method included consideration of undermining in calculating wound area⁴³ (*Level 4*). In the study by Cutler et al (1993), wound tracings from photographs produced more variable results than both the other measurement techniques⁴² (*Level 4*). It should be noted that this study was conducted in the 1990s, and since then, photography technology has significantly advanced. Studies have shown that when health professionals receive appropriate training there is high interrater and intrarater reliability for commercial wound tracing, digital photography and planimetry equipment⁴⁷⁻⁴⁹ (all *Level 4*).

Techniques for measuring pressure injury depth, tunneling and undermining

Measurement of pressure injury depth and measurement of areas of tunneling and undermining are typically performed through the very gentle insertion of a pre-moistened (with normal saline or sterile water) cotton-tipped applicator to the gentle point of resistance. The applicator is then marked off at the point that it meets skin level, then removed and held alongside a ruler to determine depth measurement. Other methods of measuring depth and undermining involve filling the wound cavity (e.g., with a malleable impression material or sterile fluid) to determine the volume.

Cutler et al. (1993) conducted a comparison between two different methods of establishing pressure injury depth—wound impression using a hydrocolloid material and the standard manual measurement technique. Measurements were made weekly for four weeks on Category/Stage III and IV pressure injuries (n = 17). There was a strong positive association between results obtained from the two techniques (r = 0.892). The impression technique generally produced slightly smaller results for wound volume in larger pressure injuries (≥ 10 cm²)⁴² (*Level 4*).

One study, conducted with 30 individuals with pressure injuries, reported a method of measuring wound undermining. Undermining was measured with a probe at four points of the wound corresponding to 3, 6, 9 and 12 on a clock face, with 12 positioned at the patient's head). Excellent interrater reliability (intraclass coefficient [ICC] = 0.996 (95% CI 0.992 to 0.999) and intrarater reliability (ICC = 0.998, 95% 0.996 to 0.999) were reported, with measurements for both falling within an error margin of 0.3 cm approximately 80% of the time for reliability measures⁵⁰ (Level 4).

Technological advances have enabled the development of laser or infrared thermography assisted wound measurement devices for 2- and 3-dimensional wound measurement. Reliability and validity data have been published for some, but not all of these systems.^{43,47,51-54} Clinicians should examine available reliability and validity data before adopting any such system.

Assessing and Monitoring Characteristics of the Pressure Injury

10.6: Assess the physical characteristics of the wound bed and the surrounding skin and soft tissue at each pressure injury assessment.
(Good practice statement)

Implementation Considerations

- Assess and document physical characteristics of the pressure injury, including:
 - Anatomical location
 - Category/Stage
 - Size and surface area (see *Recommendation 10. 2*)
 - Tissue type(s)
 - Color
 - Periwound condition
 - Wound edges
 - Sinus tracts, undermining, and tunneling
 - Exudate
 - Odor (*Expert opinion*).
- For Category/Stage II to IV and unstageable pressure injuries in individuals with darkly pigmented skin, prioritize assessment of skin heat, tenderness, changes in surrounding tissue consistency and pain. Strategies to assess skin and tissue is discussed in detail in the guideline chapter *Skin and Tissue Assessment*.
- Consider using valid and reliable wound assessment tools/scales to guide and document the findings and the assessment of the wound characteristics, and to monitor healing progress (*Expert opinion*).
- Consider using serial digital photography to document the wound's condition and to monitor healing progress (*Expert opinion*).
- Include an assessment of wound pain in every pressure injury assessment. Refer to the guideline chapter *Pain Assessment and Treatment* for more guidance in this area.

Discussion

Clinical judgment is used to assess physical characteristics of the pressure injury. These characteristics provide an indication of the pressure injury condition and its progress towards healing. Signs of healing include decreasing exudate amount, decreasing wound size, and improvement in wound bed tissue. The assessment of the physical characteristics of the pressure injury can also identify signs of wound deterioration (e.g., due to infection) that requires re-evaluation of the treatment plan. Assessment and treatment of infection is discussed in the guideline chapter *Infection and Biofilms*.

Inflammatory redness from cellulitis and deeper tissue damage may be difficult to detect in individuals with darkly pigmented skin. Just as Category/Stage I pressure injuries and deep tissue injury in intact skin may go undetected in dark skinned individuals,⁵⁵⁻⁶² the full extent and severity of Category/Stage II to IV, and unstageable, pressure injuries may be overlooked without a comprehensive assessment of the surrounding skin (or where this is not possible or clear, the skin on the opposite side of the body). Diagnosis and treatment of cellulitis and/or undermining may be delayed or missed with an assessment of the surrounding skin. Warm, firmer skin that is tender or painful may indicate infection, cellulitis or undermining/tunneling in the adjacent pressure injury.

Experienced health professionals are often astute in monitoring progress toward healing in wounds; however, there is room for variability when multiple health professionals (or those with less experience) are evaluating the pressure injury over time. Use of a pressure injury assessment tool/scale might achieve a more accurate assessment of the pressure injury, as discussed below. When relying on clinical judgment to assess progress toward healing, there should be clear documentation and ongoing communication among the various health professionals providing care for the individual.

Tools for Monitoring Pressure Injury Healing

10.7: Monitor the pressure injury healing progress.
(Good Practice Statement)

10.8: Consider using a validated tool to monitor pressure injury healing.
(Strength of Evidence = B2, Strength of Recommendation = ↑)

Evidence Summary

Evidence from a moderate quality Level 4 study⁶³ showed that assessment of a pressure injury made using a monitoring tool had only low correlation with clinical judgment. However, another study⁶⁴ provided indirect evidence showing a high correlation between the same tool and clinical judgment when assessing acute and chronic wounds. One moderate quality⁶⁵ and two low quality^{66,67} Level 3 studies showed that the score on a pressure injury monitoring tool is associated with whether the pressure injury eventually heals or otherwise. Scores in weeks 0 to 5 appear to be more indicative of eventual healing. Level 4 studies⁶⁸⁻⁷¹ provided evidence for the interrater and intrarater reliability of various pressure injury monitoring tools.

Implementation Considerations

- Compare assessment findings to previous assessments of the pressure injury to monitor progress towards healing (*Expert opinion*).
- Use the findings from pressure injury assessments and the trends identified in monitoring over time to guide treatment (*Expert opinion*).
- With signs of deterioration or stalled healing, conduct a comprehensive reassessment of the individual, the pressure injury and the healing environment and adjust the pressure injury treatment plan accordingly. With ongoing failure to heal, consider advanced diagnostic techniques (e.g., tissue biopsy) and the appropriateness of referral (e.g., referral for a surgical review) (*Expert opinion*).

Evidence Discussion

Pressure injury assessment tools/scales have been designed to aid in assessing the progress of pressure injury healing. George-Saintilus et al. (2009)⁶³ established poor correlation between clinical judgment of health professionals and PUSH© scores ($K = 0.11$ to 0.32) in the assessment of 48 individuals (370 total assessments) with Category/Stage II to IV pressure injuries. Considering the strong correlations that have been established between PUSH© scores and objective outcomes (e.g., wound tracings),^{67,72} the study suggests caution should be taken when relying on clinical judgment alone to assess wound progress. However, a more recent study reported a high correlation ($K = 0.97$) between and PUSH© scores and clinical judgment when acute and chronic wounds of mixed etiology (only 2% of the 541 participants had a pressure injury) were evaluated by experienced wound care nurses⁶⁴ (Level 5). Therefore, conclusions on the accuracy of clinical judgment alone are difficult to reach.

Commonly used pressure injury assessment and monitoring tools/scales that have been tested for validity and/or reliability are reported in *Table 13.2*. These tools/scales provide the basis for a structured assessment that includes a range of wound characteristics. All the tools⁷³⁻⁷⁶ below include a calculation of an overall assessment score that can be used to monitor overall progress towards healing.

Table 13.2: Pressure injury assessment and monitoring tools

	Pressure injury specific	Tool Description	Psychometric Properties
DESIGN-R ⁷³	Yes	7 items scored on numeric/description scales that combine to produce an overall severity score	<ul style="list-style-type: none"> • Strong correlation with BWAT (correlational co-efficient = 0.91)⁷⁰ • Interrater reliability when assessing clinical wounds, r = 0.91⁷⁰ • Interrater reliability when assessing photographed wounds, r = 0.94⁷⁰
Bates-Jensen Wound Assessment Tool (BWAT) ⁷⁴	Yes	15 items (13 wound characteristics scored on a Likert scale and 2 unscored items)	<ul style="list-style-type: none"> • Interrater reliability when assessing clinical wounds, ICC = ranged from 0.78⁶⁸ to 0.92⁶⁹ • Intrarater reliability when assessing clinical wounds, ICC = ranged from 0.89⁶⁸ to 0.99⁶⁹ • Moderate correlation with Category/Stage of pressure injury (r = 0.55, p = 0.001)⁷⁷ • Strong correlation with PUSH©, with correlation increasing in repeated measures over time (r = 0.72 to 0.95)⁶⁶
Pressure Ulcer Scale for Healing (PUSH©) ⁷⁵	Yes	3 items scored on numeric/description scales that combine to produce an overall total healing score	<ul style="list-style-type: none"> • Total PUSH© score explains 31% of variation in pressure injury over time⁶⁷ • Good correlation with wound tracings (r = 0.63, p = 0.01)⁷² • Strong correlation with BWAT, with correlation increasing in repeated measures over time (r = 0.72 to 0.95)⁶⁶
Spinal Cord Impairment Pressure Ulcer Monitoring Tool (SCI-PUMT) ⁷⁶	Yes	7 items scored on numeric/description scales that combine to produce an overall severity score	<ul style="list-style-type: none"> • Interrater reliability when assessing clinical wounds, r = 0.79⁷⁸ • Intrarater reliability when assessing clinical wounds, r = 0.81 to 0.99⁷⁸

The DESIGN-R tool was developed by the Japanese Society of Pressure Ulcers as a method of accurately distinguishing pressure injury healing rates. The tool, which is a revised version of the original DESIGN tool, scores the severity of a pressure injury and monitors its healing process. The DESIGN-R includes seven scales to assess depth, exudate, size, inflammation/infection, granulation tissue, necrotic tissue and undermining. The original DESIGN tool was developed using a consensus process. The DESIGN-R revision was developed in a prospective prognostic study that estimated the probability of healing at 12 months to establish a weighting for each item on DESIGN-R to establish severity ratings for the tool.⁷⁹ Interrater reliability has been reported as high for individual subscales of the DESIGN-R⁷¹ and for the overall DESIGN-R severity score,^{70,71} when used by trained health professionals.^{70,71} A positive change of at least one point in DESIGN-R score is significantly associated with complete wound healing within 30 days⁶⁵ (*Levels 3 and 4*).

The PUSH© was developed by the National Pressure Ulcer Advisory Panel (NPUAP, now NPIAP) as a method of monitoring pressure injury healing. Using existing research databases, a principal components analysis was conducted to determine the factors most predictive of pressure injury healing or deterioration. Three factors (length by width, exudate amount, and predominant tissue type) explained 39% of the variance at weeks 0 through 6 for one study sample, and 57% of healing variance from weeks 0 to 12 in a second study sample, with good discrimination between time points.⁶⁷ Because there are limited items on the tool, the PUSH© does not provide adequate information to serve as a basis for a comprehensive treatment plan. However, it does provide an efficient mechanism for monitoring change over time and evaluating whether the pressure injury is deteriorating or improving^{32,66,80,81} (*Levels 3 and 4*).

The BWAT (previously the Pressure Sore Status Tool), is a 15-item tool with 13 wound characteristics scored using a Likert scale and an additional two unscored items. Items included on the tool include size, depth, edges, undermining, necrotic tissue type and amount, exudate type and amount, skin color surrounding the pressure injury, peripheral tissue edema and induration, granulation tissue and epithelialization. The additional unscored item describe the anatomical location and the shape of the pressure injury.⁷⁴ The BWAT score correlates with the severity of the pressure injury, with higher scores indicating more severe pressure injuries according to Category/Stage.⁷⁷ The BWAT has undergone extensive validation^{68,69,77} and reliability testing among health professionals^{68,69} (*all Level 4*).

The SCI-PUMT is specifically designed to evaluate pressure injuries in individuals with spinal cord injury (SCI) with a recognition that pressure injuries can take significantly longer to heal in this populations.⁸² The tool evaluates seven items: wound surface area, depth, edges, tunneling, undermining, exudate type, and necrotic tissue amount.⁷⁸ The SCI-PUMT was developed by a consensus panel, informed by the content of both the PUSH© and BWAT. Validity and reliability of SCI-PUMT has been tested in assessment of veterans with SCI and Category II to IV pressure injuries⁷⁸ (*Level 4*). Evaluation of the acceptability of the tool to SCI-specialized health professionals indicate it is considered to be useful, convenient, easy to use, efficient and objective, and there is a strong likelihood that health professionals who use the tool make clinical decision based on the assessment outcome⁸² (*Level 5*).

Serial digital photography

Serial digital photography is more frequently being used to support the clinical assessment and monitoring of pressure injuries as digital photography becomes more accessible. Digital photography can provide supportive evidence of the progress towards healing; however, consideration should be given to the accuracy of wound photography. For example, in a small study (n = 19 pressure injuries),⁸³ the accuracy of digital wound photography was influenced by angle skew, especially when assessing wound dimensions. Errors of approximately 4% with a 10° angle skew were noted, but errors were below 2% with no skew⁸³ (*Level 4*). In a study⁸⁴ comparing digital photography of Category/Stage III and IV pressure injuries to a bedside assessment, reliability varied widely across a range of assessment criteria. For example, interrater reliability for length measurement was only slight ($\kappa = 0.075$, $p = 0.003$) but interrater reliability for presence of undermining was excellent ($\kappa = 0.85$, $p < 0.001$). Intrarater reliability reached statistical significance for only 55% of the different assessment criteria⁸⁴ (*Level 4*). It should be noted that digital photography technology is rapidly changing, and the finding of these studies may not reflect currently available photography techniques. However, the underlying principles of using appropriate equipment, standardized technique and ensuring health professionals are adequately trained are generalizable.

Emerging Pressure Injury Assessment Strategies

A small body of evidence explores the use of thermography, ultrasound and wound bed color assessment as strategies for assessing pressure injuries and monitoring progress towards healing. The evidence on these assessment methods is insufficient to make specific recommendations. Use of these strategies require access to appropriate equipment and training, which is currently limited in most clinical and geographic settings. Additionally, further exploration of the interpretation of findings is required.

Nakagami et al. (2010)⁸⁵ utilized thermography to predict pressure injury healing. In this small prognostic study (n = 33), the relative risk for delayed healing in pressure injuries with a wound temperature above the temperature of surrounding skin was 2.25 (95% CI 1.13 to 4.47, $p = 0.021$). The study was of only three weeks duration and the sensitivity of the thermography in detecting pressure injuries that would be slow to heal was 0.56 (*Level 4*). In a second trial,⁸⁶ thermography was combined with ultrasound to predict healing in Category/Stage I (n = 10) and II (n = 27) pressure injuries. The findings indicated that pressure injuries that displayed an increased temperature compared to surrounding skin and that also had an unclear layered structure on ultrasound assessment were 6.85 times more likely to experience delays in healing.⁸⁶ Although not explicitly used to assess individuals with darkly pigmented skin, the population was of Asian background, and further development of such thermographic imaging may prove useful in aiding pressure injury assessment in dark skinned individuals. Thermography use in evaluating pressure damaged intact skin (i.e., Category/Stage I pressure injuries and deep tissue pressure injuries) is discussed further in the guideline chapter *Skin and Tissue Assessment*.

Assessment of wound bed color as a predictor of healing for pressure injuries has been reported in a number of small prognostic trials.^{87,88} The studies explore the use of a redness value of wounds, indicative of the percent of granulation tissue, that is derived from digital imagery of the wound bed. In the most recent and larger trial, evaluation of 68 Category/Stage III and IV pressure injuries was undertaken using the DESIGN-R, a ranking of wound depth on a 6-point scale, wound imaging, and evaluation of nutritional status and anemia. A granulation redness index was calculated from the digital images using image editing software applied by an experienced researcher. This study⁸⁸ identified relationships between the granulation redness index and nutritional status, diabetic status and hemoglobin levels (*Level 3*). However, further work is required on the practical application of these assessments before the assessment methods are feasible in clinical settings.

Ultrasound as a strategy to monitor pressure injury healing has also been investigated. Aoi et al.⁸⁹ undertook exploration of 20 Category/Stage I and II pressure injuries (n = 12) and suspected deep tissue injuries (n = 8) to identify loss of dermo-epidermal interface and presence of hypoechoic lesions in subcutaneous fat and/or deep muscle using intermediate frequency (10MHz) ultrasound. All the pressure injuries exhibited at least one ultrasonic change indicating pressure damage extending beyond the epidermis. 100% of Category/Stage II and pressure injuries and deep tissue pressure injuries and 63% of Category/Stage I pressure injuries exhibited disruption of the dermo-epidermal interface.⁹⁰ This study indicated that tissue damage beyond that visible to the clinician exhibits sign of damage and

could be used to evaluate healing progress (*Level 3*). The previous studies^{89,91} also indicated that characteristics of the fascia and deep tissue that are detectable using ultrasound may be predictive of deterioration of a pressure injury versus its healing. However, the accessibility and feasibility of using ultrasound evaluation in clinician practice requires exploration, and significant investment in equipment and training is likely required before ultrasonic evaluation is adopted into standard pressure injury assessment.

References

1. Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden Scale for predicting pressure sore risk. *Nurs Res*, 1987; 36(4): 205-10.
2. Brown G. Long-term outcomes of full-thickness pressure ulcers: Healing and mortality. *Ostomy Wound Management*, 2003; 49(10): 42-50.
3. Pang SM, Wong TK. Predicting pressure sore risk with the Norton, Braden, and Waterlow scales in a Hong Kong rehabilitation hospital. *Nurs Res*, 1998; 47(3): 147-153.
4. Chaplin J. Pressure sore risk assessment in palliative care. *J Tissue Viability*, 2000; 10(1): 27-31.
5. De Conno F, Ventafridda V, Saita L. Skin problems in advanced and terminal cancer patients. *J Pain Symptom Manage*, 1991; 6(4): 247-256.
6. Ferrell BA, Josephson K, Norvid P, Alcorn H. Pressure ulcers among patients admitted to home care. *J Am Geriatr Soc*, 2000; 48(9): 1042-1047.
7. Hanson D, Langemo DK, Olson B, Hunter S, Sauvage TR, Burd C, Cathcart Silberberg T. The prevalence and incidence of pressure ulcers in the hospice setting: analysis of two methodologies. *Am J Hosp Palliat Care*, 1991; 8(5): 18-22.
8. Henschel I, Gustafsson M. Pressure ulcers in palliative care: Development of a hospice pressure ulcer risk assessment scale. *Int J Palliat Nurs*, 2003; 9(11): 474-484.
9. Perneger TV, Gaspoz JM, Rae AC, Borst F, Heliot C. Contribution of individual items to the performance of the Norton pressure ulcer prediction scale. *J Am Geriatr Soc*, 1998; 46(10): 1282.
10. Maida V, Ennis M, Kuziemycki C, Corban J. Wounds and survival in cancer patients. *Eur J Cancer*, 2009; 45(18): 3237-3244.
11. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
12. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis
13. Bates-Jensen B, Early, L, Seeman, S., *Skin Disorders*, in *Textbook of Palliative Nursing*, B.R. Ferrell and N. Coyle, Editors. 2004, Oxford University Press, Inc: New York, NY.
14. Ruggeri E, Martotoni AA, Malavasi I, Agostini F, Piccinelli E, Mazzetti M, Tenace A, Campullu L, Boin B, Pannuti F. The treatment of pressure ulcers in advanced cancer patient: The importance of a dedicated team. *Acta Vulnologica*, 2016; 14(2): 65-77.
15. Sankaran BM, Chakraborty S, Patil VM, Raghavan SN, Thomas S, Sen S. Burden and outcomes of pressure ulcers in cancer patients receiving the kerala model of home based palliative care in India: Results from a prospective observational study. *Indian J Palliat Care*, 2015; 21(2): 152-7.
16. McDonald A, Lesage P. Palliative management of pressure ulcers and malignant wounds in patients with advanced illness. *J Palliat Med*, 2006; 9(2): 285-295.
17. Tippet AW. Wounds at the end of life. *Wounds: A Compendium of Clinical Research & Practice*, 2005; 17(4): 91-98.
18. Mast BA, Schultz GS. Interactions of cytokines, growth factors, and proteases in acute and chronic wounds. *Wound Repair Regen*, 1996; 4(4): 411-20.
19. Masaki F, Riko K, Seiji H, Shuhei Y, Aya Y. Evaluation of pressure ulcers in 202 patients with cancer -- do patients with cancer tend to develop pressure ulcers? Once developed, are they difficult to heal? *Wounds: A Compendium of Clinical Research & Practice*, 2007; 19(1): 13-19.
20. McNees P, Meneses KD. Pressure ulcers and other chronic wounds in patients with and patients without cancer: a retrospective, comparative analysis of healing patterns. *Ostomy Wound Management*, 2007; 53(2): 70-78.
21. Maida V, Ennis M, Corban J. Wound outcomes in patients with advanced illness. *Int Wound J*, 2012; 9(6): 683-92.
22. Kayser-Jones J, Kris AE, Lim K, Walent RI, Halifax E, Paul SM. Pressure ulcers among terminally ill nursing home residents. *Res Gerontol Nurs*, 2008; 1(1): 14-24.
23. van Rijswijk L. Full-thickness pressure ulcers: Patient and wound healing characteristics. *Decubitus*, 1993; 6(1): 16-21.
24. van Rijswijk L, Polansky M. Predictors of time to healing deep pressure ulcers. *Ostomy Wound Manage*, 1994; 40(8): 40-2, 44, 46-8 passim.
25. Brown G. Reporting outcomes for stage IV pressure ulcer healing: A proposal. *Adv Skin Wound Care*, 2000; 13(6): 277-83.
26. Jones KR, Fennie K. Factors influencing pressure ulcer healing in adults over 50: An exploratory study. *J Am Med Dir Assoc*, 2007; 8(6): 378-87.
27. Palese A, Luisa S, Ilenia P, Laquintana D, Stinco G, Di Giulio P. What is the healing time of Stage II pressure ulcers? Findings from a secondary analysis. *Adv Skin Wound Care*, 2015; 28(2): 69-75.
28. Bliss DZ, Gurvich O, Savik K, Eberly LE, Harms S, Mueller C, Garrard J, Cunanan K, Wiltzen K. Racial and ethnic disparities in the healing of pressure ulcers present at nursing home admission. *Arch Gerontol Geriatr*, 2017; 72: 187-194.
29. Banks MD, Ross LJ, Webster J, Mudge A, Stankiewicz M, Dwyer K, Coleman K, Campbell J. Pressure ulcer healing with an intensive nutrition intervention in an acute setting: A pilot randomised controlled trial. *J Wound Care*, 2016; 25(7): 384-392.
30. Bergstrom N, Horn SD, Smout RJ, Bender SA, Ferguson ML, Taler G, Sauer AC, Sharkey SS, Voss AC. The National Pressure Ulcer Long-Term Care Study: Outcomes of pressure ulcer treatments in long-term care. *J Am Geriatr Soc*, 2005; 53(10): 1721-1729.

31. Bergstrom N, Smout R, Horn S, Spector W, Hartz A, Limcangco MR. Stage 2 pressure ulcer healing in nursing homes. *J Am Geriatr Soc*, 2008; 56(7): 1252-1258.
32. Lynn J, West S, Hausmann D, Gifford R, Nelson P, McGann P, Bergstrom N, Ryan JA. Collaborative clinical quality improvement for pressure ulcers in nursing homes. *J Am Geriatr Soc*, 2007; 55(10): 1663-9.
33. Bolton L, McNees P, van Rijswijk L, de Leon J, Lyder C, Kobza L, Edman K, Scheurich A, Shannon R, Toth M, Wound Outcomes Study Group. Wound healing outcomes using standardized assessment and care in clinical practice. *J Wound Ostomy Continence Nurs*, 2004; 31(2): 65-71.
34. Iizaka S, Kaitani T, Nakagami G, Sugama J, Sanada H. Clinical validity of the estimated energy requirement and the average protein requirement for nutritional status change and wound healing in older patients with pressure ulcers: A multicenter prospective cohort study. *Geriatr Gerontol Int*, 2015; 15(11): 1201-1209.
35. Brandeis G, Morris J, Nash D, Lipsitz L. The epidemiology and natural history of pressure ulcers in elderly nursing home residents *J Am Med Assoc*, 1990; 264(22): 2905-2909.
36. Edsberg LE, Wyffels JT, Ha DS. Longitudinal study of Stage III and Stage IV pressure ulcer area and perimeter as healing parameters to predict wound closure. *Ostomy Wound Management*, 2011; 57(10): 50-62.
37. Edsberg LE, Wyffels JT, Brogan MS, Fries KM. Analysis of the proteomic profile of chronic pressure ulcers. *Wound Repair Regen*, 2012; 20(3): 378-401.
38. Taverna D, Pollins AC, Sindona G, Caprioli RM, Nanney LB. Imaging mass spectrometry for assessing cutaneous wound healing: Analysis of pressure ulcers. *J Proteome Res*, 2015; 14(2): 986-996.
39. Hartigan I, Murphy S, Hickey M. Older adults' knowledge of pressure ulcer prevention: a prospective quasi-experimental study. *Int J Older People Nurs*, 2012; 7(3): 208-218.
40. Brace JA, Schubart JR. A prospective evaluation of a pressure ulcer prevention and management e-learning program for adults with spinal cord injury. *Ostomy Wound Management*, 2010; 56(8): 40-50.
41. Langemo D, Anderson J, Hanson D, Hunter S, Thompson P. Measuring wound length, width, and area: which technique? *Adv Skin Wound Care*, 2008; 21(1): 42-5.
42. Cutler NR, George R, Seifert RD, Brunelle R, Sramek JJ, McNeill K, Boyd WM. Comparison of quantitative methodologies to define chronic pressure ulcer measurements. *Decubitus*, 1993; 6(6): 22-30.
43. Gabison S, McGillivray C, Hitzig SL, Nussbaum E. A study of the utility and equivalency of 2 methods of wound measurement: Digitized tracing versus digital photography. *Adv Skin Wound Care*, 2015; 28(6): 252-258.
44. Bryant J, Brooks T, Schmidt B, Mostow E. Reliability of wound measuring techniques in an outpatient wound center. *Ostomy Wound Manage*, 2001; 47(4): 44-51.
45. Bilgin M, Güneş Ü. A comparison of 3 wound measurement techniques. *J Wound Ostomy Continence Nurs*, 2013; 40(6): 590-593.
46. Chang AC, Dearman B, Greenwood JE. A comparison of wound area measurement techniques: Visitrak versus photography. *Eplasty*, 2011; 11: e18.
47. Langemo D, Spahn J, Spahn T, Pinnamaneni VC. Comparison of standardized clinical evaluation of wounds using ruler length by width and Scout length by width measure and Scout perimeter trace. *Adv Skin and Wound Care*, 2015; 28(3): 116-121.
48. Sugama J, Matsui Y, Sanada H, Konya C, Okuwa M, Kitagawa A. A study of the efficiency and convenience of an advanced portable Wound Measurement System (VISITRAK). *J Clin Nurs*, 2007; 16(7): 1265-1269.
49. Haghpanah S, Bogie K, Wang X, Banks PG, Ho CH. Reliability of electronic versus manual wound measurement techniques. *Arch Phys Med Rehabil*, 2006; 87(10): 1396-1402.
50. Arora M, Harvey LA, Chhabra HS, Sharawat R, Glinsky JV, Cameron ID. The reliability of measuring wound undermining in people with spinal cord injury. *Spinal Cord*, 2017; 55: 304-306.
51. Langemo DK, Spahn JG. A Reliability Study Using a Long-Wave Infrared Thermography Device to Identify Relative Tissue Temperature Variations of the Body Surface and Underlying Tissue. *Adv Skin Wound Care*, 2017; 30(3): 109-119.
52. Langemo D, Spahn J, Snodgrass L. Accuracy and Reproducibility of the Wound Shape Measuring and Monitoring System. *Adv Skin Wound Care*, 2015; 28(7): 317-323.
53. Bhedi A, Saxena A, Gadani R, Patel R. Digital photography and transparency-based methods for measuring wound surface area. *Indian J Surg*, 2013; 75(2): 111-114.
54. Anghel EL, Kumar A, Bigham TE, Maselli KM, Steinberg JS, Evans KK, Kim PJ, Attinger CE. The reliability of a novel mobile 3-dimensional wound measurement device. *Wounds*, 2016; 28(11): 379-386.
55. VanGilder C, MacFarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. *Ostomy Wound Manage*, 2008; 54(2): 40-54.
56. Baumgarten M, Margolis D, van DC, Gruber-Baldini A, Hebel J, Zimmerman S, Magaziner J. Black/White differences in pressure ulcer incidence in nursing home residents. *J Am Geriatr Soc*, 2004; 52(8): 1293-1298.
57. Rosen J, Mittal V, Degenholtz H, Castle N, Mulsant B, Nace D, Rubin F. Pressure ulcer prevention in black and white nursing home residents: A QI initiative of enhanced ability, incentives, and management feedback. *Adv Skin Wound Care*, 2006; 19(5): 262-269.
58. Barczak CA, Barnett RI, Childs EJ, Bosley LM. Fourth national pressure ulcer prevalence survey. *Adv Wound Care*, 1997; 10(4): 18-26.
59. Lyder CH, Yu C, Emerling J, Mangat R, Stevenson D, Empleo Frazier O, McKay J. The Braden Scale for pressure ulcer risk: evaluating the predictive validity in Black and Latino/Hispanic elders. *App Nurs Res*, 1999; 12(2): 60-8.
60. Lyder CH, Yu C, Stevenson D, Mangat R, Empleo-Frazier O, Emerling J, McKay J. Validating the Braden Scale for the prediction of pressure ulcer risk in blacks and Latino/Hispanic elders: a pilot study. *Ostomy Wound Manag*, 1998; 44(3A Suppl): 42S.
61. Meehan M. Multisite pressure ulcer prevalence survey. *Decubitus*, 1990; 3(4): 14-7.
62. Meehan M. National pressure ulcer prevalence survey. *Adv Wound Care*, 1994; 7(3): 27-30, 34, 36-8.

63. George-Saintilus E, Tommasulo B, Cal CE, Hussain R, Mathew N, Dlugacz Y, Pekmezaris R, Wolf-Klein G. Pressure ulcer PUSH score and traditional nursing assessment in nursing home residents: do they correlate? *J Am Med Dir Assoc*, 2009; 10(2): 141-144.
64. Choi EP, Chin WY, Wan EY, Lam CL. Evaluation of the internal and external responsiveness of the Pressure Ulcer Scale for Healing (PUSH) tool for assessing acute and chronic wounds. *J Adv Nurs*, 2016; 72(5): 1134-43.
65. Iizaka S, Sanada H, Matsui Y, Furue M, Tachibana T, Nakayama T, Sugama J, Furuta K, Tachi M, Tokunaga K, Miyachi Y, Scientific Education Committee of the Japanese Society of Pressure Ulcers. Predictive validity of weekly monitoring of wound status using DESIGN-R score change for pressure ulcer healing: a multicenter prospective cohort study. *Wound Repair Regen*, 2012; 20(4): 473-81.
66. Gardner SE, Frantz RA, Bergquist S, Shin CD. A prospective study of the Pressure Ulcer Scale for Healing (PUSH). *J Gerontol A Biol Sci Med Sci*, 2005; 60(1): 93-97.
67. Stotts NA, Rodeheaver GT, Thomas DR, Frantz RA, Bartolucci AA, Sussman C, Ferrell BA, Cuddigan J, Maklebust J. An instrument to measure healing in pressure ulcers: development and validation of the pressure ulcer scale for healing (PUSH). *J Gerontol A Biol Sci Med Sci*, 2001; 56(12): M795-9.
68. Bates-Jensen BM, McNees P. Toward an intelligent wound assessment system. *Ostomy Wound Manage*, 1995; 41(7A Suppl): 80s-86s.
69. Bates-Jensen BM, Vredevoe DL, Brecht ML. Validity and reliability of the Pressure Sore Status Tool. *Decubitus*, 1992; 5(6): 20-8.
70. Sanada H, Moriguchi T, Miyachi Y, Ohura T, Nakajo T, Tokunaga K, Fukui M, Sugama J, Kitagawa A. Reliability and validity of DESIGN, a tool that classifies pressure ulcer severity and monitors healing. *J Wound Care*, 2004; 13(1): 13-18.
71. Zhong X, Nagase T, Huang L, Kaitani T, Iizaka S, Yamamoto Y, Kanazawa T, Sanada H. Reliability and validity of the Chinese version of DESIGN-R, an assessment instrument for pressure ulcers. *Ostomy Wound Manage*, 2013; 59(2): 36-43.
72. Hon J, Lagden K, McLaren A, O'Sullivan D, Orr L, Houghton PE, Woodbury MG. A prospective, multicenter study to validate use of the Pressure Ulcer Scale for Healing (PUSH®) in patients with diabetic, venous, and pressure ulcers. *Ostomy Wound Manage*, 2010; 56(2): 26.
73. Japanese Society of Pressure Ulcers. *DESIGN-R Scoring Manual*. 2014 [cited September 2019]; Available from: http://www.jspu.org/pdf/DESIGN-R_manual_eng.pdf.
74. Bates-Jensen B. *Bates-Jensen Wound Assessment Tool*. 2001 [cited September 2019]; Available from: <http://www.woundcare.ca/Uploads/ContentDocuments/BWAT.pdf>.
75. National Pressure Ulcer Advisory Panel. *Pressure Ulcer Scale for Healing (PUSH) 2019* [cited September 2019]; Available from: <https://npuap.org/page/PUSHTool>.
76. US Department of Veterans Affairs. *Quality Enhancement Research Initiative: Spinal Cord Impairment Pressure Ulcer Monitoring Tool*. 2012 [cited September 2019]; Available from: <https://www.queri.research.va.gov/tools/sci-pumt/>.
77. Bates-Jensen BM. The Pressure Sore Status Tool a few thousand assessments later. *Adv Wound Care*, 1997; 10(5): 65-73.
78. Thomason SS, Luther SL, Powell-Cope GM, Harrow JJ, Palacios P. Validity and reliability of a pressure ulcer monitoring tool for persons with spinal cord impairment. *J Spinal Cord Med*, 2014; 37(3): 317-327.
79. Matsui Y, Furue M, Sanada H, Tachibana T, Nakayama T, Sugama J, Furuta K, Tachi M, Tokunaga K, Miyachi Y. Development of the DESIGN-R with an observational study: an absolute evaluation tool for monitoring pressure ulcer wound healing. *Wound Repair Regen*, 2011; 19(3): 309-315.
80. Ratliff CR, Rodeheaver GT. Use of the PUSH tool to measure venous ulcer healing. *Ostomy Wound Manage*, 2005; 51(5): 58-60, 62-53.
81. Günes UY. A prospective study evaluating the Pressure Ulcer Scale for Healing (PUSH Tool) to assess Stage II, Stage III, and Stage IV pressure ulcers. *Ostomy Wound Manage*, 2009; 55(5): 48-52.
82. Thomason SS, Graves BA, Madaris L. A pilot study to evaluate the role of the Spinal Cord Impairment Pressure Ulcer Monitoring Tool (SCI-PUMT) in clinical decisions for pressure ulcer treatment. *Ostomy Wound Manage*, 2014; 60(12): 28-36.
83. Sprigle S, Nemeth M, Gajjala A. Iterative design and testing of a hand-held, non-contact wound measurement device. *J Tissue Viability*, 2011.
84. Terris DD, Woo C, Jarczok MN, Ho CH. Comparison of in-person and digital photograph assessment of stage III and IV pressure ulcers among veterans with spinal cord injuries. *J Rehabil Res Dev*, 2011; 48(3): 215-224.
85. Nakagami G, Sanada H, Iizaka S, Kadono T, Higashino T, Koyanagi H, Haga N. Predicting delayed pressure ulcer healing using thermography: a prospective cohort study. *J Wound Care*, 2010; 19(11): 465-472.
86. Nakagami G, Sanada H, Higashino T, Kadono T, Uchida G, Fujita H, Ogawa Y, Yamamoto Y, Iizaka S, Koyanagi H, Sasaki S, Haga N. Combination of ultrasonographic and thermographic assessments for predicting partial-thickness pressure ulcer healing. *Wounds*, 2011; 23(9): 285-292.
87. Iizaka S, Kaitani T, Sugama J, Nakagami G, Naito A, Koyanagi H, Konya C, Sanada H. Predictive validity of granulation tissue color measured by digital image analysis for deep pressure ulcer healing: A multicenter prospective cohort study. *Wound Repair Regen*, 2013; 21(1): 25-34.
88. Iizaka S, Koyanagi H, Sasaki S, Sekine R, Konya C, Sugama J, Sanada H. Nutrition-related status and granulation tissue colour of pressure ulcers evaluated by digital image analysis in older patients. *J Wound Care*, 2014; 23(4): 198-206.
89. Aoi N, Yoshimura K, Kadono T, Nakagami G, Iizaka S, Higashino T, Araki J, Koshima I, Sanada H. Ultrasound assessment of deep tissue injury in pressure ulcers: possible prediction of pressure ulcer progression. *Plast Reconstr Surg*, 2009; 124(2): 540-550.
90. Aliano KA, Stavrides S, Davenport T. The use of hemoglobin saturation ratio as a means of measuring tissue perfusion in the development of heel pressure sores. *Surg Technol Int*, 2013; 23: 69-71.
91. Yabunaka K, Iizaka S, Nakagami G, Aoi N, Kadono T, Koyanagi H, Uno M, Ohue M, Sanada S, Sanada H. Can ultrasonographic evaluation of subcutaneous fat predict pressure ulceration? *J Wound Care*, 2009; 18(5): 192-196.

PAIN ASSESSMENT AND TREATMENT

Introduction

Pressure injuries are painful. A prevalence study conducted in long term aged care facilities in seven European countries (n = 4,156) found that presence of a severe pressure injury (odds ratio [OR] = 2.03, 95% confidence interval [CI] 1.51 to 2.72, p < 0.01) was a significant correlate in the experience of pain.¹

Individuals with pressure injuries experience wound-related pain that can be quantified and differentiated from other pain, and this pain occurs both during procedures and at rest.²⁻⁶ Dallam et al. (1995)⁷ evaluated pressure injury pain in hospitalized adults with Category/Stage I or II pressure injuries (n = 132) using two previously validated tools, a visual analog scale (VAS) and the Wong-Baker FACES® Pain Rating Scale (FRS). Participants in the study who could respond (n = 44) were able to quantify their pressure injury pain. The average pain level reported by individuals with Category/Stage I and II pressure injuries was 4 cm and 3.5 cm respectively, on a 10 cm VAS. Individuals with Category/Stage IV pressure injuries had greater levels of pain. In all, 68% of participants who responded reported some degree of pressure injury pain. However, only 2% of individuals experiencing pressure injury pain received timely analgesics after reporting their pain (*Level 1*). Gorecki et al. (2011)⁸ concurred with these findings in a systematic review that included four quantitative studies and six qualitative studies involving participants with pressure injury pain (n = 107). Participants with Category/Stage II pressure injuries reported lower pain severity than those with Category/Stage III and IV pressure injuries.

The pain caused by pressure injuries can be constant and severe, and may be the most distressing pressure injury symptom the individual reports.^{2-7,9-21} Pain related to pressure injuries can arise from:

- Pressure, friction, and/or shear
- Damaged nerve endings
- Inflammation and/or infection
- Procedures/treatments
- Excoriation from incontinence
- Muscle spasm.²²⁻²⁴

More recently, Kim et al. (2016)²⁵ have proposed a biopsychosocial conceptual framework describing the relationships between concepts of pain experience. The framework notes that numerous factors influence the individual's experience of pressure injury pain, including:

- Severity of the pressure injury (i.e., Category/Stage)
- Sociocultural factors (e.g., ethnicity and social support)
- Psychological factors (e.g., anxiety, depression, fatigue and coping strategies)
- Biological factors (e.g., inflammation, infection and comorbidity)
- Environmental factors (e.g., nurse/patient ratio and frequency of wound dressing changes).²⁵

Despite this background, individuals with chronic wounds, especially older adults and those with dementia, continue to be under-assessed and under-treated.^{26,27} As noted by Gorecki et al. (2011),⁸ failure to identify and treat pain can be exacerbated when the individual has a limited ability to express their problems and pain experience. Additionally, factors specific to the individual's experience of pressure injuries that influence and interact with the pain experience that should be assessed and addressed in holistic management of the individual and their pain are discussed in the guideline chapter *Quality of Life, Self-Care and Education*.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are accurate and effective methods to assess pressure injury pain?
- What are effective non-pharmacologic interventions for reducing pressure injury pain?
- What are effective pharmacologic interventions for reducing pressure injury pain?

Pressure Injury Pain Assessment

Data gathered during a pain assessment measures pressure injury pain presence, quality and quantity. These data should be interpreted to determine the severity of pressure injury pain and to inform the development of an

appropriate management plan. Pressure injury related pain may be acute (including hyperalgesia), chronic, nociceptive or neuropathic. Refer to the *Glossary* for definitions and further explanation.

11.1: Conduct a comprehensive pain assessment for individuals with a pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Managing pain is a priority for people with pressure injury pain. In the US it is mandated that people in hospitals receive regular, ongoing pain assessment.²⁸ No evidence was identified indicating that conducting a pain assessment contributes to pressure injury healing or management of pressure injury pain. However one high quality Level 1 diagnostic study^{7,14} established that pressure injury pain can be identified using two well-established pain assessment tools, a VAS and FRS. A low quality Level 3 diagnostic study¹⁹ and two Level 5 diagnostic studies^{6,21} suggested that pressure injury pain can be identified using the well-established pain assessment tool, the McGill Pain Questionnaire (MPQ). A range of pain assessment tools are easily accessible and feasible to implement in most clinical settings.

Implementation Considerations

- A single pain assessment tool alone may not provide sufficient information to guide selection of pain management interventions (*Expert opinion*).
- If using a pain assessment tool, select a tool that has been designed and trialed in the relevant population (*Expert opinion*).
- Pain assessments should be done prior to and during wound procedures, such as wound dressing changes or debridement, as well as when no procedures are in progress (*Expert opinion*).
- Include the following in a comprehensive pain assessment:
 - Character, intensity and duration of the pressure injury pain²¹ (*Level 5*)
 - Change in the severity or quality of pressure injury pain over time²⁹⁻³¹ (*Expert opinion*)
 - Physical examination that includes a neurological component (*Expert opinion*)
 - Appropriate diagnostic work-up to determine the type and cause of the pain³² (*Expert opinion*)
 - Severity and duration of the pressure injury^{21,33,34} (*Level 3*)
 - Psychosocial assessment (*Expert opinion*)
 - Activities associated with experiencing pressure injury pain^{6,21,35,36} (*Level 5*)
 - Activities associated with reducing pressure injury pain^{6,21,35,36} (*Level 5*).
- Pay attention to non-verbal language and body cues, particularly in individuals who cannot express themselves verbally (e.g., neonates, children and individuals with impaired cognitive and abstract thought ability such as those with dementia or psychological/psychiatric disorders) (*Expert opinion*).
- Pain over a pressure area may be an indicator of early pressure injury that should be more fully assessed³⁷ (*Level 1*). Assessment of pain as a risk factor for pressure injuries is discussed in the chapter *Risk Factors and Risk Assessment*.
- Reports of increasing pressure injury pain over time should prompt a re-assessment of the pressure injury²⁹⁻³¹ (*Expert opinion*).

Evidence Discussion

The most reliable indicator of pain is the individual's report of pain. Systematic ongoing assessment of pain provides direction for the pain treatment plan, with modifications based on the response of the individual.^{22,38,39} The U.S. Joint Commission on Accreditation of Hospital Organizations mandates regular and ongoing assessment of pain in all hospitalized individuals, including neonates and children, in US health facilities.²⁸

Assess for pressure injury related pain using a scale or tool that is valid and reliable, and appropriate to the individual being assessed. However, a single pain assessment tool may not provide sufficient information to guide interventions. It is important to investigate other aspects of the pain in order to provide more effective, individualized interventions.

Three pain assessment tools have been tested in individuals with pressure injuries. The FRS score was highly correlated with pain intensity in individuals (n = 47) with Category/Stage II to IV pressure injuries (Pearson's r = 0.90)³⁹ (*Level 5*). A moderate correlation has also been established between VAS and Category/Stage of the pressure injury (r = 0.37) and, in the same study, pressure injury pain assessed on the VAS was strongly correlated with pain assessment on FACES (r

= 0.92)⁷ (Level 1). In addition, the VAS and FACES have also been shown to be highly reliable for pain assessment in individuals with decreased verbal and abstract thinking.¹⁴ Pain assessment on the McGill Pain Questionnaire (MPQ) is also shown to be valid for identifying pain intensity in individuals with pressure injuries, with pain intensity being significantly greater for pressure injuries of greater severity and of longer duration ($p < 0.05$)³⁹ (Level 5). However, other pain assessment tools are likely to be appropriate for individuals with pressure injury pain. Selection of a tool should be most appropriate to the individual's level of development, understanding and communication. *Table 14.1* outlines some pain assessment tools that are appropriate for identifying and assessing pressure injury pain; however, this is not a complete list of the tools available.

Table 14.1: Selection of pain assessment tools appropriate for assessing pressure injury pain

Pain Assessment Tool	Evidence for Identifying Pressure Injury Pain	Evidence for Identifying Other Types of Pain	Clinical Setting and Level of Evidence
Visual Analog Scale (VAS)	Yes	Yes	Adults ^{7,14} (Level 1, high quality) Hospitalized adults ⁴⁰ (Level 5, low quality)
McGill Pain Questionnaire (MPQ)	Yes	Yes	Older adults in long term care settings ¹⁹ (Level 3, low quality) Adults ²¹ (Level 5, moderate quality) Adults ⁶ (Level 5, moderate quality)
Wong-Baker FACES® Pain Rating Scale (FRS)	Yes	Yes	Adults with pressure injuries ^{7,14} (Level 1, high quality) Cognitively impaired adults with other types of pain ⁴¹⁻⁴⁶ (Level 5)
Abbey Pain Scale ⁴⁷	No	Yes	Cognitively impaired adults with other types of pain (Level 5)
FLACC (Face, Leg, Activity, Cry, and Consolability)	No	Yes	Children 2 months to 7 years of age with postoperative pain ⁴⁸ (Level 5)
CRIBS (Crying; Requires O ₂ for Saturation > 95%; Increasing vital signs; Expression; Sleepless) Scale	No	Yes	Neonates up to 6 months ^{49,50} (Level 5)

In conducting a pain assessment, consider activities that influence pain frequency, duration or intensity (e.g., wound dressing changes, debridement or movement/touch). Günes (2008)²¹ found that individuals with pressure injury pain reported increased pain intensity at wound dressing changes compared with at rest and individuals with more severe pressure injuries experienced constant pain. The role of anticipating pain has not been explored in individuals with pressure injuries.

Evaluate the words used by the individual to express pressure injury pain character. Acute pain is associated with pain terms such as 'quick', 'sharp' and 'short' while chronic pain is often associated with reports of constant or persistent pain.^{7,39} Neuropathic pain is associated with terms such as 'pins and needles', 'stabbing', 'shooting', 'hot poker' and 'electric pulse'.³⁹ In contrast, nociceptive pain is often described as 'nagging', 'throbbing' or 'gnawing'.⁵¹

Assess the impact of pressure injury pain on the individual's quality of life. Pressure injuries have measurable and persistent impact on health-related quality of life (HRQoL) measures. In one study, participants with pressure injuries were found to have significantly lower overall scores on Short Form Health Survey (SF-36) and EQ-5D™ ($p < 0.001$) than participants without pressure injuries. Pressure injuries were also found to impact on measures of physical functioning ($p = 0.001$).⁴⁰ (Level 5). The guideline chapter *Quality of Life, Self-Care and Education* includes a more extensive discussion of quality of life and its assessment.

Assess for deterioration of the pressure injury or possible infection if the individual reports increasing intensity of pain over time. Increasing presence or intensity of pain is an indication that a chronic wound may be infected and a comprehensive assessment of the pressure injury should be performed.²⁹⁻³¹ See the chapter *Infection and Biofilms* for recommendations on assessment and management of infection.

Assessing Pain in Non-Verbal Individuals and/or Individuals with Cognitive Impairment

For individuals who are nonverbal including infants and cognitively impaired adults, observe for specific behaviors during wound procedures and movement (e.g., change in activity, loss of appetite, guarding, grimacing, withdrawal,

crying out and moaning). Observing for behaviors such as facial expressions, verbalizations or vocalizations, body movements, changes in interpersonal interactions, and changes in activity patterns or routines is recommended.⁵²

Pain assessment tools should be appropriate to the individual's development stage and cognitive level. Individuals with pressure injuries are often older and may have cognitive impairment. Studies investigating the use of FACES by cognitively impaired adults report that this population sometimes has difficulty using this scale compared with other self-report pain assessment tools.⁴¹⁻⁴⁶ Likewise, the VAS has been shown to have limited reliability when used by cognitively impaired adults with pain associated with a range of conditions^{41,42,53} (*Level 5*). The MPQ is considered to provide a reliable pain assessment for cognitively impaired individuals.^{54,55} However, the most appropriate pain assessment tool for use in cognitively impaired older adults has not been determined.⁵⁶ Consider existing evidence-based guidelines for pain assessment in individuals with cognitive impairment and/or limitations in verbalizing pain.^{57,58}

Some researchers have reported the ability of mildly to moderately cognitively impaired older adults to respond to a simple direct yes/no question, such as:⁵⁹⁻⁶¹

- Do you have pain?
- Where is your pain?
- Can you point to or touch the area of pain?
- Do you have wound pain every day?
- Does wound pain keep you from sleeping?
- Does wound pain keep you from doing activities you enjoy?

Non-pharmacologic Interventions for Preventing and Managing Pain

11.2: Use non-pharmacologic pain management strategies as a first line strategy and adjuvant therapy to reduce pain associated with pressure injuries.
(Good Practice Statement)

Implementation Considerations

- Non-pharmacologic strategies can be used in conjunction with pharmacologic interventions, particularly during wound care procedures (*Expert opinion*).
- When choosing non-pharmacologic strategies to reduce pressure injury pain, consider the individual's development stage and cognitive ability and overall health status (*Expert opinion*).
- Provide education to individuals and their informal caregivers about non-pharmacologic strategies to manage pain⁶² (*Level 5*).
- Coordinate care delivery with administration of analgesia (*Expert opinion*).
- Encourage the individual to request a 'time out' from any procedure that causes pain (*Expert opinion*).
- Pay attention to nonverbal language and body cues when using non-pharmacologic strategies to evaluate the effectiveness of the strategy being used (*Expert opinion*).

Using non-pharmacologic pain management strategies to reduce pain associated with pressure injuries reflects good practice. There is no direct evidence from the literature search on the effectiveness of non-pharmacologic pain management strategies for treating pain associated with pressure injuries; however, non-pharmacologic pain management strategies are well-acknowledged as being useful in pain management.

The individual and their informal caregivers are integral to adequate management of pressure injury pain.⁶³ Educating the individual and family about the cause and expected duration of pain, as well as what to do to minimize pain can enhance understanding and compliance, and subsequently reduce pain.^{2-6,18,20,63-67} Discuss different non-pharmacologic interventions with the individual and explore strategies that are feasible and acceptable to the individual and the care setting to promote shared decision-making and the individual's adherence to plans. Shared decision-making can be facilitated by consultation decision aids.⁶⁸

Individuals who experience wound-related pain may also experience anxiety associated with wound care. Anxiety is influenced by both physiological and psychological factors. Anxiety can be ameliorated, at least to some degree, by:

- Talking with individuals about their wound-related pain
- Providing a detailed explanation of each procedure

- Answering questions
- Allowing active participation
- Pacing the procedure to the individual's preferences
- Allowing time outs as needed.^{7,69-71}

A large range of non-pharmacologic pain management strategies are used in managing pain including:

- Position changes
- Distraction and conversation
- Healing touch
- Music therapy
- Warmth applications
- Progressive relaxation
- Meditation and self-hypnosis
- Guided imagery
- Electrotherapy (e.g., transcutaneous electrical nerve stimulation [TENS])
- Virtual reality/computer simulation immersion.

Few studies have been conducted on the effectiveness of these non-pharmacologic strategies for managing pressure injury pain; however, their benefit in treating acute wound pain^{72,73} and chronic neuropathic pain has been reported^{23,69,70,74,75} in both adults and children.

Wound care procedures including wound manipulation, wound cleansing, debridement and dressing changes are painful. Structure wound care procedures around the analgesia regimen to minimize interruptions to comfort. For example, wait at least 20 to 30 minutes (up to a maximum of 60 minutes) after applying topical analgesia before commencing wound treatments.⁷⁶

**11.3: Use repositioning techniques and equipment with consideration to preventing and managing pressure injury pain.
(Good Practice Statement)**

Implementation Considerations

- Reposition an individual so that pressure is kept off the pressure injury wherever possible. Continued positioning on a pressure injury can result in increased pressure, pain and damage to the area^{64,77} (*Level 5*).
- Consider the need for analgesia prior to repositioning an individual with a pressure injury (*Expert opinion*).
- Use a lift, transfer sheet or slide sheet to minimize friction and/or shear when repositioning an individual (*Expert opinion*).
- When selecting appropriate repositioning techniques, consider the individual's development level, conscious state and cognitive ability. Repositioning (turn procedures) should be made by at least two health professionals, and when turning unconscious individuals to a prone position there should be at least four health professionals (*Expert opinion*).
- Do not under treat pain in individuals receiving palliative care (*Expert opinion*).

Discussion

Pressure injuries are caused, at least in part, by unrelieved pressure and the resulting ischemia of tissues that occurs between an external surface and underlying bone. Continued positioning on a pressure injury can result in increased pressure, pain and damage to the area. Keeping the individual off the pressure injury will relieve pain and ischemia, enhance soft tissue viability and promote healing of the pressure injury.^{64,77} The chapter *Repositioning and Early Mobilization* provides detailed recommendations on the role of repositioning to both prevent and treat pressure injuries. However, there is indirect evidence that repositioning and turning can cause both generalized pain and pressure injury pain,^{36,78} especially in individuals with chronic pain, limited cognitive ability or receiving end-of-life care.

Evidence from an observational study conducted in a general hospital population without pressure injuries (n = 1,395) showed that pain is experienced during repositioning. The mean pain score on an 11-point numerical rating

scale during repositioning was 4.9 ± 3.1 , suggesting that repositioning is associated with moderate pain⁷⁸ (Level 5). In a qualitative study conducted in people with multiple sclerosis and pressure injuries, participants described their experience of pain during movement and related to use of repositioning equipment³⁶ (Level 5). Individuals who are in pain often do not wish to move, yet repositioning remains a high priority for helping to relieve pain.²⁷ Even small changes in position are helpful in decreasing pressure.

Despite the relationship between positioning and pressure injury pain, there is insufficient evidence from the literature search on the most appropriate repositioning techniques to avoid triggering pain. Being aware of care interventions that cause increased pain and planning to prevent pain through premedication and careful use of equipment, is a component of good practice. Hyperalgesia is described by individuals as occurring during repositioning and transfer activities⁸ and should be addressed prior to commencing movement. Using a lift or transfer sheet can minimize shear when repositioning an individual in bed. Keeping bed linens smooth and unwrinkled can promote comfort and decrease pressure. Avoid postures that increase pressure, such as Fowler's position greater than 30° or 90° side-lying position, or the semi-recumbent position. Use small adjustments in position and provide positional support to affected pressure injury area where possible. Move gently and listen to the individual to guide movements.

Balancing comfort with repositioning at end-of-life

Relief of pain is especially important for individuals in palliative or end-of-life care with pressure injuries, as a primary goal is to provide comfort and improve their quality of life. When an individual is actively dying comfort is of primary importance. Interventions to prevent and/or treat a pressure injury are often superseded by the need to promote comfort by minimizing turning and repositioning and allowing the individual to determine frequency of turning and choice of position.⁷⁹⁻⁸³ Many individuals receiving palliative care prefer a single position for comfort, and turning and positioning may only serve to increase their pain and discomfort.^{27,79,80,82,83}

Work with the individual and their informal caregivers to develop a flexible, individualized patient-directed approach. A repositioning schedule should be based on the individual's goals, wishes, comfort and tolerance; their clinical status; and, the pressure redistribution characteristics of the support surface. Consider the individual's choices in turning, including whether they have a position of comfort, after explaining the rationale for repositioning. Document the individual's turning and repositioning, as well as the factors influencing these decisions (e.g., individual wishes or medical needs).

More frequent position changes may be possible for individual's receiving end-of-life care with the use of opiates and/or sedatives to control pain. It is important to weigh the pros and cons of medication administration, as it can lead to a decrease in spontaneous movements, which in turn is often counter to proper pain relief and promotion of comfort.⁸⁴

11.4: Use the principles of moist wound healing to reduce pressure injury pain.
(Good Practice Statement)

Implementation Considerations

- Keep the wound bed covered and moist⁸⁵ (*Expert opinion*).
- Select a wound dressing that is non-adherent, requires less frequent changes, and manages the level of exudate in the wound⁸⁵ (*Expert opinion*).
- Warm up topical products to room temperature before applying them to the wound bed (*Expert opinion*).

Discussion

Wounds re-epithelialize more quickly in the presence of a moist environment.⁸⁶ Pressure injury pain can be minimized by keeping the wound bed moist and covered.⁸⁷

Attempt to minimize wound dressing changes as much as possible because dressing removal is the activity most associated with wound pain.⁷¹ Consider using dressings that allow for less frequent change due to higher absorbency capacity, including but not exclusively, alginates, gelling fiber dressings, polymeric membrane foams, soft silicone edged wound dressings. Nonadherent and/or moist dressings cause less pain and trauma on removal.^{71,88-90} A plain gauze dressing is more likely to cause pain and will require more frequent changing to maintain the moist wound bed.⁷¹ See the *Wound Dressings for Treatment of Pressure Injuries* chapter of the guideline for further recommendations on dressing selection.

Pharmacologic Treatment for Managing Pressure Injury Pain

**11.5: Consider applying a topical opioid to manage wound-related pressure injury pain, if required and when there are no contraindications.
(Strength of Evidence = B1; Strength of Recommendation = ↔)**

Evidence Summary

There is evidence from small Level 1 studies^{12,20} and lower levels of evidence^{9,91} that use of a topical opioid can decrease pressure injury pain by at least four points on a VAS at five days,⁹ which is likely to be a clinically significant reduction in pain for most individuals. There was insufficient evidence to make recommendations on other topical products that are used to manage wound-related pressure injury pain (e.g., anti-inflammatory preparations and anaesthetics).

Implementation Considerations

- Individual who experience significant ongoing pain may require a review by a pain specialist (*Expert opinion*).
- Topical opioids generally have minimal systemic effect; however, they may be associated with increased systemic side effects in individuals taking systemic (e.g., oral) opioids. Local itching and irritation have been reported, but not more frequently than when a placebo gel is applied¹² (*Level 1*).
- Topical opioids and other topical analgesia are more effective when applied 20 to 30 minutes, and up to 60 minutes, prior to wound care⁷⁶ (*Expert opinion*).
- Topical opioids and other analgesia might be used prior to performing wound care (particularly when the pressure injury requires debridement) or other activities associated with pressure injury pain (e.g., repositioning) (*Expert opinion*).

Evidence Discussion

Opioid receptors have been found on peripheral nerves and inflamed tissue, suggesting that topically applied opioids may provide relief of pressure injury pain, with limited to no systemic effect.⁹² Availability of these preparations varies between different geographic locations and may require a prescription from a licensed health professional and/or review from a pain specialist.

Flock (2003)¹² conducted a randomized, blinded, placebo-controlled crossover pilot trial of seven hospice patients with painful Category/Stage II or III pressure injuries to compare pre and post treatment pain between diamorphine gel and a placebo gel. One hour after application, pain scores had improved significantly more for pressure injuries treated with diamorphine gel compared with placebo gel and compared with baseline scores ($p = 0.003$). The significant effect was still evident 12 hours after application ($p = 0.005$)¹² (*Level 1*). Zeppetella et al. (2003)²⁰ compared topical morphine sulphate (10 mg) applied daily to sacral pressure injuries to a placebo gel. After two days of treatment, all participants ($n = 5$) reported lower VAS scores with the topical opioid compared to placebo, with no participants experiencing adverse events attributed to opioid use.²⁰ (*Level 1*). Both these studies were very small, and both used cross-over designs that may be inappropriate for assessing pain even when a washout period has been used. A retrospective observational study of older individuals with Category/Stage II pressure injuries ($n = 15$) also demonstrated the effectiveness of diamorphine gel (5 to 10 mg) applied every 12 to 24 hours. Participants showed mean improvement over time in VAS scores of 4 points (9.4 to 4.6, $p < 0.02$)⁹ (*Level 4*).

Other topical analgesia

Other topical agents are sometimes used in managing wound-related pain, including topical anti-inflammatory preparations and topical anaesthetics. However, the evidence for these interventions for treating pressure injury pain is very limited and no recommendations could be made. These products are not universally accessible in all geographic locations.

One study ($n = 30$) conducted in a palliative care setting with participants with painful pressure injuries (Category/Stage not reported) compared topical 3% benzydamine hydrochloride cream to a placebo gel. The topical preparations were applied to intact peri-wound skin. Over 24 hours, neither the average improvement in pain on a 10 cm VAS nor the maximum pain reduction were significantly different between the anti-inflammatory cream and the placebo (mean VAS reduction 23.5mm \pm 22.5 versus 15.8 \pm 22.5mm, $p = 0.41$).¹⁵ (*Level 1*) Topical anti-inflammatories can also be delivered via impregnated wound dressings, although there is no evidence for the effectiveness in managing pain in pressure injuries. A systematic review conducted in individuals with venous leg ulcers is available,⁹³ but reports only two randomized controlled trials. In the first study a 40% reduction in pain from baseline was associated with an

ibuprofen dressing⁹⁴ and in the second study,⁹⁵ 19% more participants experienced at least a 50% reduction in their pain levels compared with a foam dressing (*Level 5*).

**11.6: Administer analgesia regularly to control pressure injury pain.
(Good Practice Statement)**

Implementation Considerations

- Analgesia needs to be administered regularly in the appropriate dose to control pain. To maintain analgesic effects, administer pain medication 'by the clock' every 3 to 6 hours according to the individual's needs, by the least invasive route⁹⁶ (*Expert opinion*).
- Organize care delivery to ensure that it is coordinated with pain medication administration and that minimal interruptions follow. Set priorities for treatment (*Expert opinion*).
- The World Health Organization (WHO) Pain Dosing Ladder is a valid and effective method for relieving cancer pain, pain at end-of-life and acute pain.³² However, the WHO Pain Dosing Ladder⁹⁷⁻¹⁰⁰ may not be appropriate for individuals with chronic (i.e., more than 12 weeks) pressure injury pain.¹⁰¹ Refer the individual with chronic pain related to pressure injuries to pain and/or wound specialists and/or clinics. (*Expert opinion*).
- Consider involving a pain specialist in developing the pain management plan for an individual with ongoing pressure injury pain (*Expert opinion*).

Discussion

The WHO Pain Dosing Ladder⁹⁷⁻¹⁰⁰ is a valid and effective method for relieving cancer pain, pain at end-of-life and acute pain.³² The WHO Pain Dosing Ladder^{99,100} is based on the goals of minimizing side effects and maximizing pain relief. After conducting a pain assessment and determining the severity of the individual's pain, the WHO Pain Dosing Ladder suggests commencing with non-opioid analgesics, and if pain is not controlled, adding a weak opioid. A step up to a stronger opioid could be considered if pain remains unrelieved.⁹⁷⁻¹⁰⁰

Non-opioid analgesics include simple analgesics (e.g., paracetamol or aspirin) and Non-steroidal anti-inflammatory drugs (NSAIDs, e.g., ibuprofen, naproxen and diclofenac). Non-opioids act on peripheral nerves to block painful impulses.

Opioids act on the central nervous system by altering the pain perception.^{99,100,102} Weak opioids can be added (not substituted) to the pain management regimen when a non-opioid option is ineffective.¹⁰³ For severe pain, a stronger opioid might be required. However, long term use of opioids is associated with adverse physical, psychological and social outcomes that may also be exacerbated by polypharmacy.^{101,104} Additionally, evidence suggests that long term opioid use (i.e., greater than 12 weeks) is ineffective in managing chronic pain.^{101,104} For chronic pressure injury pain, multi-modal management plans that include psychological interventions are suggested, and may require the involvement of a pain specialist.^{101,104}

Adjuvants enhance the effectiveness of analgesics synergistically; however, adjuvants should be individualized based on the individual's concurrent diagnoses and type of pain. Examples of adjuvants include antibiotics to manage spreading or systemic infection (which indirectly addresses pain associated with inflammation), tricyclic antidepressants or antiepileptics to manage neuropathic pain or selective serotonin reuptake inhibitor (SSRIs) to manage concurrent depression.^{104,105}

A study (n = 34) investigating an innovative pressure injury pain management strategy found that a nitrous oxide/oxygen mixture administered five minutes prior to, and throughout wound care significantly reduced (p < 0.001) pain assessed on validated pain tools compared to morphine (1 mg/ 10 kg body weight) administered 30 minutes prior to wound care¹⁰⁶ (*Level 1*). No significant difference was found with regard to safety or tolerability. Further research on such pain management strategies is warranted.

References

1. Lukas A, Mayer B, Fialova D, Topinkova E, Gindin J, Onder G, Bernabei R, Nikolaus T, Denking MD. Pain characteristics and pain control in european nursing homes: Cross-sectional and longitudinal results from the services and health for elderly in long term care (SHELTER) study. *J Am Med Assoc*, 2013; 14(6): 421-428.
2. Langemo DK, Melland H, Hanson D, Olson B, Hunter S. The lived experience of having a pressure ulcer: A qualitative analysis. *Adv Skin Wound Care*, 2000; 13(5): 225-235.
3. Bale S, Dealey C, Defloor T, Hopkins A, Worboys F. The experience of living with a pressure ulcer. *Nurs Times*, 2007; 103(15): 42-43.

4. Fox C. Living with a pressure ulcer: A descriptive study of patients' experiences. *Br J Community Nurs*, 2002; 7(6 Suppl): 10.
5. Spilsbury K, Nelson A, Cullum N, Iglesias C, Nixon J, Mason S. Pressure ulcers and their treatment and effects on quality of life: Hospital inpatient perspectives. *J Adv Nurs*, 2007; 57(5): 494-504.
6. Szor JK, Bourguignon C. Description of pressure ulcer pain at rest and at dressing change. *Journal of Wound, Ostomy, and Continence Nursing*, 1999; 26(3): 115-120.
7. Dallam L, Smyth C, Jackson BS, Krinsky R, O'Dell C, Rooney J, Badillo C, Amella E, Ferrara L, Freeman K. Pressure ulcer pain: Assessment and quantification. *J Wound Ostomy Continence Nurs*, 1995; 22(5): 211-218.
8. Gorecki C, Closs SJ, Nixon J, Briggs M. Patient-reported pressure ulcer pain: a mixed-methods systematic review. *J Pain Symptom Manage*, 2011; 42(3): 443-459.
9. Abbas SQ. Diamorphine-Intrasite dressings for painful pressure ulcers. *J Pain Symptom Manage*, 2004; 28(6): 532-534.
10. European Pressure Ulcer Advisory Panel, Pressure Ulcer Treatment Guidelines. 1998, EPUAP, : Oxford, England.
11. de Laat E, Scholte op Reimer W, van Achterberg T. Pressure ulcers: diagnostics and interventions aimed at wound-related complaints: a review of the literature. *Journal of Clinical Nursing*, 2005; 14(4): 464-472.
12. Flock P. Pilot study to determine the effectiveness of diamorphine gel to control pressure ulcer pain. *J Pain Symptom Manage*, 2003; 25(6): 547-554.
13. Freedman G, Cean C, Duron V, Tarnovskaya A, Brem H. Pathogenesis and treatment of pain in patients with chronic wounds. *Surgical Technology International*, 2003; 11: 168-179.
14. Freeman K, Smyth C, Dallam L, Jackson B. Pain measurement scales: a comparison of the visual analogue and faces rating scales in measuring pressure ulcer pain. *J Wound Ostomy Continence Nurs*, 2001; 28(6): 290-296.
15. Prentice WM, Roth LJ, Kelly P. Topical benzydamine cream and the relief of pressure pain. *Palliat Med*, 2004; 18(6): 520-524.
16. Price P. An holistic approach to wound pain in patients with chronic wounds. *Wounds: A Compendium of Clinical Research & Practice*, 2005; 17(3): 55-57.
17. Quirino J, Santos VLC, Quednau TJP, Martins APF, Lima P, Almeida MRM. Pain in pressure ulcers. *Wounds: A Compendium of Clinical Research & Practice*, 2003; 15(12): 381-389.
18. Rastinehad D. Pressure ulcer pain. *Journal of Wound, Ostomy, and Continence Nursing*, 2006; 33(3): 252-256.
19. Roth RS, Lowery JC, Hamill JB. Assessing persistent pain and its relation to affective distress, depressive symptoms, and pain catastrophizing in patients with chronic wounds: a pilot study. *Am J Phys Med Rehabil*, 2004; 83(11): 827-834.
20. Zeppetella G, Paul J, Ribeiro MD. Analgesic efficacy of morphine applied topically to painful ulcers. *J Pain Symptom Manage*, 2003; 25(6): 555-558.
21. Günes UY. A descriptive study of pressure ulcer pain. *Ostomy Wound Management*, 2008; 54(2): 56-61.
22. American Society of Pain Management Nurses, *Core Curriculum for Pain Management Nursing*. 2002, Philadelphia, PA: Saunders.
23. Reddy M, Keast D, Fowler E, Sibbald RG. Pain in pressure ulcers. *Ostomy Wound Management*, 2003; 49(4 Suppl): 30-35.
24. Thomas S. Pain and wound management. *Community Outlook*, 1989; 12: 11-5.
25. Kim J, Ahn H, Lyon DE, Stechmiller J. Building a biopsychosocial conceptual framework to explore pressure ulcer pain for hospitalized patients. *Healthcare (Basel)*, 2016; 4(1).
26. Alvarez OM, Kalinski C, Nusbaum J, Hernandez L, Pappous E, Kyriannis C, Parker R, Chrzanowski G, Comfort CP. Incorporating wound healing strategies to improve palliation (symptom management) in patients with chronic wounds. *J Palliat Med*, 2007; 10(5): 1161-89.
27. Alvarez OM, Meehan M, Ennis W, Thomas DR, Ferris FD, Kennedy KL, Rogers R, Bradley M, Baker JJ, Fernandez-Obregon A, Rodeheaver G. Chronic wounds: Palliative management for the frail population. *Wounds: A Compendium of Clinical Research & Practice*, 2002; 14(8): 45-27s.
28. Joint Commission, *Pain assessment and management: an organizational approach*. 2000, Oakbrook Terrace, IL: The Joint Commission.
29. Cutting KF, Harding KG. Criteria for identifying wound infection. *J Wound Care*, 1994; 3(4): 198-201.
30. Cutting KF, White RJ, Mahoney P, Harding KG, *Clinical identification of wound infection: a Delphi approach.*, in *European Wound Management Identifying criteria for wound infection. EWMA Position Document*. 2005, London: MEP Ltd. p. 6-9.
31. Gardner TN, Briggs GA. Biomechanical measurements in microscopically thin stratum comeum using acoustics. *Skin Res Technol*, 2001; 7(4): 254-261.
32. Jacox A, Carr D.B., Payne, R., et al. , *Management of cancer pain*. 1994, Rockville, MD: The Agency for Health Care Policy and Research (AHCPR), now Agency for Healthcare Research and Quality (AHRQ).
33. Ahn H, Stechmiller J, Horgas A. Pressure ulcer-related pain in nursing home residents with cognitive impairment. *Advances in Skin and Wound Care*, 2013; 26(8): 375-380.
34. Ahn H, Stechmiller J, Fillingim R, Lyon D, Garvan C. Bodily pain intensity in nursing home residents with pressure ulcers: analysis of national minimum data set 3.0. *Res Nurs Health*, 2015; 38(3): 207-12.
35. Jackson D, Durrant L, Bishop E, Walthall H, Betteridge R, Gardner S, Coulton W, Hutchinson M, Neville S, Davidson PM, Usher K. Pain associated with pressure injury: A qualitative study of community based, home-dwelling individuals. *J Adv Nurs*, 2017.
36. McGinnis E, Nelson A, Gorecki C, Nixon J. What is different for people with MS who have pressure ulcers: A reflective study of the impact upon people's quality of life? *Journal of Tissue Viability*, 2015; 24(3): 83-90.
37. Smith IL, Brown S, McGinnis E, Briggs M, Coleman S, Dealey C, Muir D, Nelson EA, Stevenson R, Stubbs N, Wilson L, Brown JM, Nixon J. Exploring the role of pain as an early predictor of category 2 pressure ulcers: A prospective cohort study. *BMJ Open*, 2017; 7(1): e013623.
38. Berry PH, Covington ED, Dahl J, Pain: Current understanding of assessment, management, and treatment. 2006, National Pharmaceutical Council, Inc. 2006: Reston, VA.
39. Gunes UY. A descriptive study of pressure ulcer pain. *Ostomy Wound Management*, 2008; 54(2): 56-61.
40. Essex HN, Clark M, Sims J, Warriner A, Cullum N. Health-related quality of life in hospital inpatients with pressure ulceration:

- Assessment using generic health-related quality of life measures. *Wound Repair Regen*, 2009; 17(6): 797-805.
41. Closs SJ, Barr B, Briggs M, Cash K, Seers K. A comparison of five pain assessment scales for nursing home residents with varying degrees of cognitive impairment. *J Pain Symptom Manage*, 2004; 27: 196-205.
 42. Closs SJ, Cash K, Barr B, Briggs M. Cues for identification of pain in nursing home residents. *Int J Nurs Stud*, 2005; 42: 3-12.
 43. Pautex S, Herrmann F, Le Lous P, Fabjan M, Michel JP, Gold G. Feasibility and reliability of four pain self-assessment scales and correlation with an observational rating scale in hospitalized elderly demented patients. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 2005; 60A: 524-529.
 44. Pautex S, Michon A, Guedira M, Emond H, Lous PL, Samaras D, Michel JP, Herrmann F, Giannakopoulos P, Gold G. Pain in severe dementia: Self-assessment or observational scales? *Journal of the American Geriatric Society*, 2006; 54: 1040-1045.
 45. Scherder E, Bouma A. Visual analogue scales for pain assessment in Alzheimer's disease. *Gerontology*, 2000; 46: 47-53.
 46. Scherder E, van Manen F. Pain in Alzheimer's disease: nursing assistants' and patients' evaluations. *J Adv Nurs*, 2005; 52: 151-158.
 47. Abbey J, De Bellis A, Piller N, Esterman A, Giles L, Parker D, Lowcay B. *Abbey Pain Scale*. 2002 [cited May 2019]; Available from: https://apsoc.org.au/PDF/Publications/Abbey_Pain_Scale.pdf.
 48. Merkel SI, Voepel-Lewis T, Shayevitz JR, Malviya S. The FLACC: A behavioral scale for scoring postoperative pain in young children. *Pediatr Nurs*, 1997; 23(3): 293-297.
 49. Bildner J. CRIES Instrument Assessment Tool of Pain in Neonates. 1997, City of Hope Pain/Palliative Care Resource Center: Missouri.
 50. Krechel SW, Bildner J. CRIES: A new neonatal postoperative pain measurement score. Initial testing of validity and reliability. *Paediatr Anaesth*, 1995; 5(1): 53-61.
 51. White R, Harding K. *Trauma and Pain in Wound Care*. 2006, Wounds UK: Aberdeen.
 52. AGS Panel on Pharmacological Management of Persistent Pain in Older Adults. Pharmacological management of persistent pain in older adults. *J Am Geriatr Soc*, 2009; 57: 1331-1346.
 53. Closs SJ, Barr B, Briggs M, Cash K, Seers K. Evaluating pain in care home residents with dementia. *Nursing and Residential Care*, 2003; 5: 32-35.
 54. Ferrell BA, Ferrell, B.R., Rivera L. Pain in cognitively impaired nursing home patients. *J Pain Symptom Manage*, 1995; 10: 591-598.
 55. Wynne CF, Ling SM, Remsburg R. Comparison of pain assessment instruments in cognitively intact and cognitively impaired nursing home residents. *Geriatric Nursing*, 2000; 21: 20-23.
 56. Husebo BS, Achterberg W, Flo E. Identifying and managing pain in people with alzheimer's disease and other types of dementia: A systematic review. *CNS Drugs*, 2016; 30(6): 481-497.
 57. National Institute for Health and Clinical Excellence (NICE). *Dementia: Assessment, Management And Support For People Living With Dementia And Their Carers (NG97)*. 2018, NICE: <https://www.nice.org.uk/guidance/ng97/chapter/Recommendations>.
 58. Hadjistavropoulos T, Herr K, Prkachin KM, Craig KD, Gibson SJ, Lukas A, Smith JH. Pain assessment in elderly adults with dementia. *Lancet Neurol*, 2014; 13(12): 1216-1227.
 59. Chu L, Schnelle JF, Cadogan MP, Simmons SF. Using the minimum data set to select nursing home residents for interview about pain. *J Am Geriatr Soc*, 2004; 52(12): 2057-2061.
 60. Herr K. Pain assessment in cognitively impaired older adults. *Am J Nurs*, 2002; 102(12): 65-67.
 61. Jensen MP. The validity and reliability of pain measures in adults with cancer. *J Pain*, 2003; 4(1): 2-21.
 62. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis
 63. Barnes SH. Patient/family education for the patient with a pressure necrosis. *Nurs Clin North Am*, 1987; 22(2): 463-74.
 64. Bergstrom N, Bennett, M.A., Carlson, C.E., et al, *Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. AHCPR Pub. No. 95-0653*. 1994, Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, Agency for Healthcare Policy and Research.
 65. Hopkins A, Worboys F. Establishing community wound prevalence within an inner London borough: Exploring the complexities. *Journal of Tissue Viability*, 2015; 24(1): 42-49.
 66. Moffatt CJ. Pain at Wound Dressing Changes: Understanding Wound Pain and Trauma: An International Perspective. EWMA Position Document. 2002, Medical Education Partnership, London UK.
 67. Montague A, *Touching the human significance of the skin*. 1971, Columbia, NY: Columbia University Press.
 68. Agoritsas T, Heen AF, Brandt L, Alonso-Coello P, Kristiansen A, Akl EA, Neumann I, Tikkinen KA, Weijden TV, Elwyn G, Montori VM, Guyatt GH, Vandvik PO. Decision aids that really promote shared decision making: The pace quickens. *BMJ (Clinical research ed)* 2015; 350: g7624.
 69. Krasner D. The chronic wound pain experience: a conceptual model. *Ostomy Wound Management*, 1995; 41(3): 20.
 70. Krasner D, *Caring for the person experiencing chronic wound pain.*, in *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*, D. Krasner, D. Rodeheaver, and G. Sibbald, Editors. 2001, HMP Communications: Wayne, PA. p. 79-89.
 71. Brown A. Strategies to reduce or eliminate wound pain. *Nursing Times*; , 2014; 110(15): 12-15.
 72. McSherry T, Atterbury M, Gartner S, Helmsold E, Searles D, Schulman C. Randomized, crossover study of immersive virtual reality to decrease opioid use during painful wound care procedures in adults. *J Burn Care Res*, 2018; 39(2): 278-285.
 73. Hoffman H, Chambers G, Meyer 3rd W, et al. Virtual reality as an adjunctive non-pharmacologic analgesic for acute burn pain during medical procedures. *Ann Behav Med*, 2011; 41(2): 183-191.
 74. Tan G, Alvarez JA, Jensen MP. Complementary and alternative medicine approaches to pain management. *J Clin Psychol*, 2006; 62(11): 1419-1431.

75. Tick H, Nielsen A, Pelletier K, Bonakdar R, Simmons S, Glick R, Ratner E, Lemmon R, Wayne P, Zador V. The Pain Task Force of the Academic Consortium for Integrative Medicine and Health. Evidence-based nonpharmacologic strategies for comprehensive pain care: The consortium pain task force white paper. *Explore*, 2018; 14: 177-211.
76. Evans E, Gray M. Do topical analgesics reduce pain associated with wound dressing changes or debridement of chronic wounds? *Journal of Wound, Ostomy, and Continence Nursing*, 2005; 32(5): 287-290.
77. Wound Ostomy and Continence Nurses Society (WOCNS), *Wound Ostomy and Continence Nurses Society. Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: Wound Ostomy and Continence Nurses Society.
78. Faigeles B, Howie-Esquivel J, Miaskowski C, Stanik-Hutt J, Thompson C, White C, Wild LR, Puntillo K. Predictors and Use of Nonpharmacologic Interventions for Procedural Pain Associated with Turning among Hospitalized Adults. *Pain Manag Nurs*, 2013; 14(2): 85-93.
79. Brink P, Smith TF, Linkewich B. Factors associated with pressure ulcers in palliative home care. *Journal of Palliative Medicine*, 2006; 9(6): 1369-1375.
80. Eisenberger A, Zeleznik J. Pressure ulcer prevention and treatment in hospices: A qualitative analysis. *J Palliat Care*, 2003; 19(1): 9-14.
81. Henoch I, Gustafsson M. Pressure ulcers in palliative care: development of a hospice pressure ulcer risk assessment scale. *Int J Palliat Nurs*, 2003; 9(11): 474-484.
82. Langemo D, Anderson J, Hanson D, Thompson P, Hunter S. Understanding palliative wound care. *Nursing*, 2007; 37(1): 65-66.
83. Langemo DK. When the goal is palliative care. *Adv Skin Wound Care*, 2006; 19(3): 148-154.
84. Masaki F, Riko K, Seiji H, Shuhei Y, Aya Y. Evaluation of pressure ulcers in 202 patients with cancer -- do patients with cancer tend to develop pressure ulcers? Once developed, are they difficult to heal? *Wounds: A Compendium of Clinical Research & Practice*, 2007; 19(1): 13-19.
85. Keast DH, Parslow N, Houghton PE, Norton L, Fraser C. Best practice recommendations for the prevention and treatment of pressure ulcers: Update 2006. *Adv Skin Wound Care*, 2007; 20(8): 447-60.
86. Hinman CD, Maibach H. Effect of air exposure and occlusion on experimental human skin wounds. *Nature*, 1963; 200: 377-378.
87. Queen D, Woo K, Schulz VN, Sibbald RG. Chronic wound pain and palliative cancer care. *Ostomy Wound Management*, 2005; 51(11A): 9-11.
88. Bale S, Squires D, Varnon T, Walker A, Benbow M, Harding KG. A comparison of two dressings in pressure sore management. *J Wound Care*, 1997; 6(10): 463-466.
89. Matzen S, Peschardt A, Alsbjorn B. A new amorphous hydrocolloid for the treatment of pressure sores: A randomised controlled study. *Scand J Plast Reconstr Surg Hand Surg*, 1999; 33(1): 13-5.
90. Meaume S, Van De Looverbosch D, Heyman H, Romanelli M, Ciangherotti A, Charpin S. A study to compare a new self-adherent soft silicone dressing with a self-adherent polymer dressing in stage II pressure ulcers. *Ostomy Wound Management*, 2003; 49(9): 44-51.
91. Twillman RK, Long TD, Cathers TA, Mueller DW. Treatment of painful skin ulcers with topical opioids. *J Pain Symptom Manage*, 1999; 17(4): 288-292.
92. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
93. Briggs M, Nelson EA, Martyn-St James M. Topical agents or dressings for pain in venous leg ulcers. *Cochrane Database Syst Rev*, 2012; 11.
94. Gottrup F, Jørgensen B, Karlsmark T, Sibbald G, Rimdeika R, Harding K, Price P, Venning V, Vowden P, Jünger M, Wortmann S, Sulcaite R, Vilkevicius G, Ahokas TL, Ettlner K, M. A. Reducing wound pain in venous leg ulcers with Biatain Ibu: A randomised controlled double blind clinical investigation on performance and safety. *Wound Repair Regen*, 2008; 16: 615-25.
95. Arapoglou V, Katsenis D, Syrigos KN, Dimakakos EP, Zakopoulou N, Gjødsbøl K, Glynn C, Schäfer E, Petersen B, Tsoutos D. Analgesic efficacy of an ibuprofen-releasing foam dressing compared with local best practice for painful exuding wounds. *J Wound Care*, 2011; 20(7): 319-25.
96. Reddy M, Kohr R, Queen D, Keast D, Sibbald RG. Practical treatment of wound pain and trauma: A patient-centered approach. An overview [corrected] [published erratum appears in *Ostomy Wound Management* 2003 May;49(5):8]. *Ostomy Wound Management*, 2003; 49(4A): 2.
97. Best Practice Advocacy Centre New Zealand. WHO Analgesic Ladder: Which weak opioid to use at step two? *Best Practice Journal*, 2008; 18.
98. Vargas-Schaffer G. Is the WHO analgesic ladder still valid? Twenty-four years of experience. *Can Fam Physician*, 2010; 56(6): 514-517.
99. World Health Organization, *Cancer pain relief and palliative care*. 1990, Geneva, Switzerland: World Health Organization.
100. World Health Organization, *Cancer pain relief*. 2nd ed. 1996, Geneva, Switzerland: WHO.
101. British Medical Association, *Chronic pain: Supporting safer prescribing of analgesics*. 2017, British Medical Association, : London, UK.
102. Busse JW, Craigie S, Juurlink D, Buckley N, Wang L, Couban RJ, Agoritsas T, Akl EA, Carrasco-Labra A, Cooper L, Cull C, da Costa BR, Frank JW, Gus Grant G, Iorio A, Persaud N, Stern S, Tugwell P, P.O. V, Guyatt GH. The 2017 Canadian Guideline for Opioids for Chronic Non-Cancer Pain. *CMAJ*, 2017; 189(18): E659-E666.
103. Best Practice Advocacy Centre New Zealand. Pharmacological management of chronic pain. *Best Practice Journal*, 2008; 16.
104. Best Practice Advocacy Centre New Zealand. Understanding the role of opioids in chronic non-malignant pain. 2018.
105. Best Practice Advocacy Centre New Zealand. Strong opioids for pain management in adults in palliative care. *Best Practice Journal*, 2012; 49.
106. Paris A, Horvath R, Basset P, Thiery S, Couturier P, Franco A, Bosson JL. Nitrous oxide-oxygen mixture during care of bedsores and painful ulcers in the elderly: A randomized, crossover, open-label pilot study. *Journal of Pain & Symptom Management*, 2008; 35(2): 171-176.

SUPPORTING WOUND HEALING

Wound bed preparation is a clinical concept encompassing a systematic and holistic approach to wound evaluation and treatment that create a wound environment that will promote normal wound healing. The overall goal of wound bed preparation is to promote a well-vascularized wound bed, free from non-viable tissue and excess exudate, and with a reduced bacterial burden and reduced edema, that is optimal for development of healthy granulation tissue.^{1,2} This section provides a background to the discussion, evidence and recommendations presented in chapters of the guideline that focus on preparing the wound bed and promoting healing.

Wound bed preparation incorporates four major aspects of wound care, represented by the acronym TIME:¹⁻⁴

- Tissue management
- Infection and inflammation control
- Moisture balance
- Epithelial edge advancement.

More recently the TIME acronym has been updated to **TIMERS** to include:⁵

- Repair and regeneration
- Social factors and factors related to the individual

Evaluating and optimizing each of these components of wound care addresses barriers that are known to delay normal healing in chronic wounds.

Tissue Management

Removing devitalized or necrotic tissue and its associated bacterial and cellular burden provides a stimulatory wound environment that promotes healthy tissue growth.^{1,3} The guideline chapter *Cleansing and Debridement* provides comprehensive discussion and recommendations on cleansing techniques, appropriate use of debridement, selection of debridement techniques, and cautions to consider.

Infection and Inflammation Control

Treatment of bacterial burden is a significant consideration in chronic wounds that are often heavily colonized. The role of biofilm in delaying healing is also a concern. Treatment of infection reduces bacterial counts, inflammatory cytokines and protease activity; and increases growth factor activity in the wound bed, promoting health healing.^{1,3} The guideline section *Infection and Biofilms* provides further discussions and recommendations for clinical practice.

Moisture Balance

Promoting a warm, moist wound bed prevents desiccation, stimulates growth factor activity and promotes accelerated re-epithelialization, but does not increase infection. Control of excessive moisture prevents maceration of surrounding tissue.^{2,4} Appropriate selection of moisture-retentive dressings and use of absorptive dressings in heavily exuding wounds plays a key role in promoting moisture balance to promote healing.² The *Wound Dressings* chapter provides recommendations to guide practice.

Epithelial Edge Advancement

Failure of the epithelium to advance indicates that barriers to healing have not been adequately removed and further preparation of wound bed is needed. A non-advancing wound edge, or undermining, can be due to abnormalities in the cellular matrix, hypoxia of the wound bed or abnormal protease activity.⁴ Control of infection and inflammation; removal of cellular burden through debridement; and control of wound moisture are all important considerations in promoting epithelial advancement.² Sequential monitoring of the advance of epithelium at the wound edge allows health professionals to assess the adequacy of wound bed preparation.

Repair and Regeneration

Repair and regeneration addresses support of wound healing through therapies that support and stimulate the wound healing process. In the absence of infection/biofilm or unaddressed co-morbidities (e.g. vascular disease), therapies that promote development of the extra-cellular matrix and stimulate the activity of cells involved in healing processes are an option for some individuals.⁵ The chapters *Biological Dressings* and *Growth Factors* provide recommendations on wound therapies that are designed to support the repair and regeneration of the tissue and skin, for example

collagen therapy, topical growth factors and bioengineered dermal substitutes.⁵ In some individuals with full thickness pressure injuries, surgical repair of the wound may be required. Recommendations on supporting the individual through the surgical journey are discussed in the chapter *Pressure Injury Surgery*.

Social and Individual-related Factors

Social factors and individual factors are acknowledged as making an important contribution to the ability of the wound to heal.⁵ A wide range of factors related to the individual are recognized as contributing to the pressure injury risk, and many of these factors will also influence ability to heal (e.g., skin status, nutrition status, mobility, hematological status etc.). The *Risk Factors and Risk Assessment* chapter provides an overview of intrinsic factors that influence pressure injury risk that also must be addressed to enable existing pressure injuries to heal and to prevent recurrence. Co-morbidities beyond pressure injury risk factors can also play a part in the ability of the individual to adhere to a treatment plan and/or for the wound to heal.⁵

Complex factors related to the individual's environment, psychological status, sleep, knowledge and education, and social supports also influence pressure injury prevention and treatment.⁵ The limited evidence available on strategies to address social and individual-related factors that can impact both pressure injury prevention and treatment is discussed in the chapter *Quality of Life, Self-Care and Education*.

Emerging science is continuously identifying more factors that put individuals at risk of delayed healing, leading to exploration of other factors that could be addressed to promote faster healing, for example social determinants, immune-based therapies⁶ and genetic factors.⁷

References

1. Falanga V, *Wound bed preparation: science applied to practice*, in European Wound Association Position Document: Wound Bed Preparation in Practice. 2004, Medic: London.
2. Halim AS, Khoo TL, Mat Saad AZ. Wound bed preparation from a clinical perspective. *Indian J Plast Surg*, 2012; 45(2): 193-202.
3. Leaper DJ, Schultz GS, Carville K, Fletcher J, Swanson T, Drake R. Extending the TIME concept: what have we learned in the past 10 years? *Int Wound J*, 2012; 9(Suppl 2): 1-19.
4. Dowsett C. Using the TIME framework in wound bed preparation. *Br J Community Nurs*, 2008; 13(6): S15-S20.
5. Atkin L, Bucko Z, Conde Montero E, Moffatt CJ, Probst A, Romanelli M, Schultz GS, Tettlebach W. Implementing TIMERS: the race against hard-to-heal wounds. *J Wound Care*, 2019; 28(3 Suppl 3): S1-S49.
6. Larouche J, Sheoran S, Maruyama K, Martino MM. Immune regulation of skin wound healing: Mechanisms and novel therapeutic targets. *Adv Wound Care.*, 2018; 7(7): 209–231.
7. Li H, Duann P, Lin PH, Zhao L, Fan Z, Tan T, Zhou X, Sun M, Fu M, Orange M, Sermersheim M, Ma H, He D, Steinberg SM, Higgins R, Zhu H, John E, Zeng C, Guan J, Ma J. Modulation of wound healing and scar formation by MG53 protein-mediated cell membrane repair. *J Biol Chem*, 2015; 290(40): 24592-245603.

CLEANSING AND DEBRIDEMENT

Introduction

Cleansing and debridement are important aspects of wound bed preparation and for creating a wound environment that promotes healing. There is a large volume of evidence on wound cleansing and debridement for chronic wounds of different etiologies. This chapter specifically focuses on the research available on these aspects of wound care in the context of managing pressure injuries.

Clinical Question

The clinical question that guided the development of this chapter was:

- What local pressure injury treatments are effective for supporting healing (i.e., cleansing, debridement, topical agents, wound dressings, etc.)?

Wound Cleansing

Wound cleansing is the process of using fluids to remove surface contaminants (debris), remnants of previous dressings and microorganisms from the wound and peri-wound surface.¹ Cleansing does not ‘sterilize’ a wound; instead, it ‘washes’ a wound. If fibrinous material and detritus/debris cannot be removed gently with fluids, then an evaluation for the appropriateness of debridement (i.e., removal of devitalized tissue) is required.

Research on cleansing of pressure injuries is sparse. Most clinical articles regarding cleansing speak to general cleansing principles for any type of wound bed preparation. Cleansing is an important first step in preparing the pressure injury wound bed for healing by removing surface debris and dressing remnants, which facilitates better wound visualization for assessment.

12.1: Cleanse the pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

There is only a small body of evidence on cleansing pressure injuries. Two small, moderate² and low³ quality Level 1 studies provide evidence that cleansing a pressure injury is associated with statistically significant reduction in wound size and improvements in pressure injury severity than when cleansing is not performed. In one study, low pressure pulsatile lavage was more effective than no lavage,² and in the second study cleansing was more effective than allowing the wound bed to dry.³

Implementation Considerations

- The type and frequency of cleansing should be based on the wound size, Category/Stage, and wound bed characteristics (e.g., exudate) (*Expert opinion*).
- Manage pain before cleansing the pressure injury (*Expert opinion*). See the guideline chapter *Pain Assessment and Treatment* for information on managing pain.
- Cleanse most pressure injuries and the peri-wound skin with potable water (i.e., water suitable for drinking) or normal saline⁴⁻⁷ (*Level 5*).
- In some countries and organizations, individuals with chronic wounds can bath or shower to cleanse the wound, if there is access to non-shared, appropriately cleaned, bathing facilities.⁸ In other countries or organizations, the use of sterile water or cleansing solutions is mandated. Develop a cleansing regimen with consideration to local standards⁴ and policies, cleanliness of facilities, the anatomical location of the pressure injury and the individual’s preferences⁸ (*Expert opinion*).
- Consider using an aseptic technique when the individual, the wound or the wound healing environment is compromised^{4,5,8} (*Expert opinion*).
- Cleanse pressure injuries with sinus tracts/tunneling/undermining with caution⁴ (*Expert opinion*).
- Apply cleansing solution with enough pressure to cleanse the wound bed without damaging tissue or driving bacteria into the wound, generally between 4 and 15 pounds per square inch (psi) (*Expert opinion*).

Evidence Summary

Ho et al. (2012)² demonstrated the effectiveness of cleansing Category/Stage III and IV pressure injuries (n = 28) using low pressure (maximum 11psi) pulsatile lavage. Compared with sham lavage, cleansing with normal saline at a low pressure for 10 to 20 minutes was associated with greater reduction in pressure injury depth, width, length and volume (all p < 0.01). However, this study was conducted over only three weeks, with weekly pressure injury measurement and did not follow pressure injuries to complete closure.² The mean between group differences were small, despite being statistically significant, and confidence intervals spanned the null value (*Level 1*). Another study³ (n = 50) comparing a regimen that included wound cleansing to a regimen that did not include wound cleansing also reported significant improvements in both pressure injury size and wound bed condition measured as a Pressure Ulcer Scale for Healing (PUSH) score. Category/Stage II and III pressure injuries that received regular cleansing showed significantly greater reduction in surface area after 28 days compared with pressure injuries that were air-dried to promote scabbing (p < 0.05). There was a 92% improvement in overall PUSH score for the cleansed pressure injuries compared with a 60% improvement for the air-dried pressure injuries (p < 0.01) (*Level 1*).

For clean pressure injuries (those with no debris or confirmed bacterial infection), potable (drinkable) tap water or normal saline is recommended, although some organizations mandate the use of sterile solutions and aseptic technique. Boiled and cooled water is an effective wound cleansing solution if potable water or normal saline is not available.⁴⁻⁷ No differences in rates of infection and healing between potable water and normal saline have been noted in the cleansing of chronic wounds in adults or children^{6,9,10} (*Level 5*). Aseptic technique using sterile products should be considered when the individual is immunocompromised; or if the wound enters a sterile body cavity or when the wound healing environment is compromised; otherwise, clean wound management technique is appropriate.^{4,5} When the wound bed cannot be visualized due to sinus tracts/tunneling/undermining there is a possibility that the cleansing solution may not be retrieved.⁴

Pressure injury cleansing can be accomplished by irrigating the wound bed with fluid. Cleansing must be extremely gentle in re-epithelializing pressure injuries to prevent disruption of the neoepithelium. However, in order to remove the debris in the wound bed, the force of the irrigation stream has to be greater than the adhesion forces holding the debris to the wound surface.¹¹ Generally, irrigation pressure between 4 and 15 psi should be adequate to clean the surface of the pressure injury without causing trauma to the wound bed.^{4,11,12} Appropriate irrigation can be achieved with selection of different sized syringes and needles (see *Table 16.1*) or alternatively, there are also many commercially available irrigation devices. Wound cleansing should only be performed when clinically indicated. Frequency of cleansing should be based on the need to remove exudate, debris, wound dressing residue or loosely adhered necrotic tissue,^{11,13} and/or the need to better visualize the wound bed.

Table 16.1: Examples of irrigation pressure for wound cleansing^{1,14,15}

Syringe	Needle Gauge	Irrigation Pressure
35 cc (ml)	19-gauge	8 psi
20 cc (ml)	28-gauge angio catheter	12 psi
12 cc (ml)	22-gauge	13 psi

Contain and properly dispose of used irrigation solution to reduce cross-contamination. Environmental contamination is possible with irrigation devices and fluids, and infection-control precautions should be routinely used.¹⁶

12.2: Use cleansing solutions with antimicrobials to clean pressure injuries with suspected or confirmed infection. (Good Practice Statement)

Implementation Considerations

- Surfactant antimicrobial cleansing solutions for use on the wound bed include polyhexamethylene biguanide (PHMB) and octenidine dihydrochloride (OCT).⁵ (*Expert opinion*).
- Antiseptic cleansing solutions for use on the wound bed include super oxidised solutions with hypochlorous acid (HOCl)¹⁷ and sodium hypochlorite (NaOCl) and povidone iodine.⁵ (*Expert opinion*).
- Cytotoxicity of cleansing solutions may be concentration-dependent. Select a product with a sustained release of antimicrobial agent at concentrations sufficiently low to minimise toxicity, and use for the shortest time duration to reduce bioburden.⁵ (*Expert opinion*).

- Refer to local antimicrobial resistance policies for guidance when using antiseptics (*Expert opinion*).
- Refer to the guideline chapter *Infection and Biofilm* for more information on using antimicrobial solutions.

Evidence Discussion

Pressure injuries with devitalized tissue or suspected biofilm usually require more aggressive use of irrigating solutions or debridement. Comprehensive systematic reviews^{18,19} have identified no direct evidence to support the use of any specific wound cleansing solutions or wound cleansing techniques.

Cleansers with antimicrobial properties assist in managing bioburden. Some cleansers combine an antimicrobial with a surfactant that lowers surface tension and promotes the spread of the liquid across the wound bed, facilitating separation of loose, non-viable tissue and bioburden. For dirty pressure injuries (those with debris and/or confirmed high bacterial colonization), a cleansing solution with a surfactant and/or antimicrobial agent appropriate for the wound and consistent with current toxicity/efficacy recommendations should be considered until the wound bed is clean.^{4,5}

One study comparing an aloe vera, silver chloride and decylglucoside to isotonic saline for pressure injuries (n = 82) reported the aloe vera spray was superior in achieving improvements on the Pressure Sore Status Tool (PSST) (p = 0.02) after 14 days²⁰ (*Level 1*). Another study¹⁷ compared use of HOCl to normal saline for irrigation of chronic wounds (n = 17 individuals, n = 12 had pressure injuries). Irrigation was performed in conjunction with ultrasonic debridement and followed by use of silver dressings for seven days in both groups. Fewer wound complications were observed in the HOCl group (35% versus 80%). This study had numerous methodological limitations (*Level 1*).

Some wound cleansers are cytotoxic to fibroblasts if used in high concentrations¹ (See *Table 16.2*). Cleansers that are formulated to remove fecal material (skin cleansers) are cytotoxic, and should not be used in wounds.¹ Avoid products intended for use only on intact skin. Solutions that are at room temperature when applied to the wound are reported to be less painful.

For pressure injuries with suspected biofilm, debridement is the most effective management strategy,^{21,22} as discussed in the *Debridement* section of this chapter.

Table 16.2: Topical antimicrobial therapies (Reproduced with permission from the International Wound Infection Institute⁵)

Solution	Type	Cytotoxicity	Effect on biofilm	Comments
Sterile normal saline	Isotonic ²³	None	None	<ul style="list-style-type: none"> • Sterile, non-antiseptic solution²⁴
Sterile water	Hypotonic	None	None	<ul style="list-style-type: none"> • Sterile, non-antiseptic solution²⁴
Potable tap water	Varies in content	Unknown/ variable	None	<ul style="list-style-type: none"> • Not sterile²⁴
Polyhexamethylene Biguanide (PHMB)	Surfactant anti-microbial	Low to none ²²	Surfactant qualities disrupt biofilm attachments ²²	<ul style="list-style-type: none"> • Lowers liquid surface tension allowing greater spread and facilitating separation of non-viable tissue²² • Does not promote bacterial resistance²²
Octenidine dihydrochloride (OCT)	Surfactant anti-microbial	<ul style="list-style-type: none"> • In-vitro tests show high toxicity²⁵ • Lack of absorption suggests no systemic effects²⁵ • Not been shown to disrupt healing 	<ul style="list-style-type: none"> • Prevents formation of new biofilm for at least 3 hours²⁶ • Inhibits planktonic and bacterial biofilm growth for up to 72 hours²⁶ 	<ul style="list-style-type: none"> • Comes in a gel and irrigation preparation that can be used together or separately²⁵ • Lowers liquid surface tension allowing greater spread and facilitating separation of non-viable tissue²⁶

Solution	Type	Cytotoxicity	Effect on biofilm	Comments
Super oxidised solution with hypochlorous acid(HOCL) and Sodium hypochlorite (NaOCL)	Antiseptic	May vary depending on concentrations	<ul style="list-style-type: none"> • Penetrates biofilm rapidly, killing formations from within²⁴ • Does not promote resistant bacteria strains²⁴ 	<ul style="list-style-type: none"> • Purported to provide de-sloughing and antimicrobial activity • Comes in a gel and irrigation preparation that can be used together or separately
Povidone Iodine	Antiseptic	Varies depending on concentration ²⁷	<ul style="list-style-type: none"> • Inhibits development of new biofilm²⁸ • Eradicates young biofilm colonies²⁸ • Significantly reduces mature biofilm colonies²⁸ 	<ul style="list-style-type: none"> • Modulates redox potentials and enhances angiogenesis thereby promoting healing²⁹ • May inhibit excess protease levels in chronic wounds²⁹

12.3: Cleanse the skin surrounding the pressure injury.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Research supporting the recommendation to cleanse skin surrounding the pressure injury comes from a low quality Level 2 study³⁰ that found faster healing was associated with peri-wound cleansing for Category/Stage II pressure injuries. Additionally, a low quality Level 4 study³¹ suggested that peri-wound cleansing is associated with a reduction in skin microbials for up to 24 hours.

Implementation Considerations

- Evaluate the skin surrounding the pressure injury and determine the risk for damage to peri-wound skin (*Expert opinion*).
- If the peri-wound skin is vulnerable, consider applying a barrier product to protect the skin from medical adhesive related skin injury (*Expert opinion*).

Evidence Discussion

In a non-randomized clinical trial in older adults (n = 189), Konya et al. (2005)³² compared cleansing of the peri-wound skin with normal saline to cleansing with a pH-balanced (pH not specified) skin cleanser. For pressure injuries of all Category/Stage, healing time was shorter when the peri-wound skin was cleansed with a pH-balanced skin cleanser and water. However the decreased healing time was only statistically significant for Category/Stage II pressure ulcers (median healing 15 days versus 20 days, p = 0.002).³² Lack of control for the increased potential for excreta in pressure injuries of the sacrum, ischial tuberosities and coccyx may have influenced the findings (*Level 2*). In an observational study with older adults (n = 5),³¹ there was a significant reduction in peri-wound skin microbial counts immediately after cleansing (p < 0.05), but microbial levels returned to baseline within 24 hours³¹ (*Level 4*). This suggests that cleansing the peri-wound skin daily or more often is appropriate for reducing microbes that may colonize the wound bed.

Debridement

Devitalized tissue is nonviable or necrotic. It is normally moist, yellow, green, tan, or gray and may become thick and leathery with dry black or brown eschar. Debridement of devitalized tissue is an essential component of wound bed preparation.^{22,33-37} Debridement in the presence of adequate wound bed vascularity is believed to hold a key role in wound bed preparation, addressing not only the barriers to chronic wound healing but also providing potential stimulatory effects.^{22,35,36}

There are few clinical studies conducted in pressure injuries providing evidence on the use of debridement; the ethics of such trials are dubious. Prior to Steed et al.'s 1996³⁸ pivotal post hoc analysis of a non-randomized comparison of debridement rates in wound healing centers participating in a recombinant growth factor study, there was no experimental clinical data to support the commonly accepted view and clinical practice that debridement was beneficial to wound healing.^{34,38} Steed's³⁸ finding that aggressive debridement of diabetic foot ulcers was associated

with increased wound closure set the stage for investigation into the cascade of benefits afforded by initial and maintenance debridement.^{34,38}

Despite the lack of research evidence, there is strong informed clinical consensus to support the role of debridement in wound bed preparation, with a large volume of clinical practice guidelines, care standards and research in other wound types supporting the concept of debridement as a necessary component of effective wound bed preparation.^{4,34-36,38-51}

12.4: Avoid disturbing stable, hard, dry eschar in ischemic limbs and heels, unless infection is suspected. (Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

One low quality Level 3 study⁵² supports the recommendation to avoid disturbing stable eschar. When heel eschar was left intact, 99.3% of heel pressure injuries healed in an average duration of 11 weeks.

Implementation Considerations

- Assess stable, hard, dry eschar at each wound dressing change and as clinically indicated⁴ (*Expert opinion*).
- Consult a medical practitioner/vascular surgeon urgently in the presence of erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor (i.e., signs of infection) in the area around the wound dressing (*Expert opinion*).
- Debride the pressure injury urgently in the presence of, erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor. Surgical sharp debridement or conservative sharp debridement should be used because these are the fastest methods to debride the wound^{4,39-42,53} (*Expert opinion*).

Evidence Discussion

Assessment of a pressure injury covered with dry, stable eschar should be performed at each dressing change and as clinically indicated to detect the first signs of any developing infection. Clinical indications that the dry, stable eschar requires assessment and intervention include signs of erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor (i.e., signs of infection) in the area around the dressing.^{4,39-42,53}

Shannon et al. (2013)⁵² conducted a retrospective review in a nursing home population of heel pressure injuries (n = 179) with entire eschar (67.8% of the sample) or blister coverage (31.8% of the sample). Of the 155 participants not lost to follow up, 154 of the pressure injuries (99.3%) healed. Of the heel pressure injuries covered with eschar, 100% of wounds healed within an average healing time of 11 weeks (range 2 to 50 weeks). Complications included one individual who developed osteomyelitis (with eventual healing) and two cases of cellulitis, one leading to amputation (Level 3).

12.5: Debride the pressure injury of devitalized tissue and suspected or confirmed biofilm and perform maintenance debridement until the wound bed is free of devitalized tissue and covered with granulation tissue. (Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

There is strong informed clinical consensus to support the role of debridement in wound bed preparation, despite the ethically understandable lack of randomized controlled trials directly comparing debridement to no debridement in human subjects.^{34,36,38,39,41,43-50,53-56} Direct evidence on debridement primarily offers comparisons between different types of debridement rather than demonstrating that debriding a wound is more effective than not performing debridement. One study provided indirect evidence that sharp debridement is effective in increasing susceptibility of wound bacteria in chronic wounds to antibiotic therapy for short periods (up to 72 hours).⁵⁷ One high quality Level 3 study⁵⁷ demonstrated improvement in wound condition reported on the Bates-Jensen Wound Assessment Tool (BWAT) with enzymatic debridement compared to no debridement or sharp debridement (it was unclear how many controls received no debridement in the study).

Comparisons between different types of debridement generally demonstrate no statistically significant differences between methods. One low quality Level 1 study,⁵⁸ and one low quality Level 3 study⁵⁹ demonstrated that enzymatic

debridement is as effective as autolytic debridement and sharp debridement in achieving improvements in wound surface area. Two high quality Level 3 studies^{57,60} also demonstrated enzymatic debridement is associated with improvements in wound condition (increase in granulation tissue and improvement in scores on a BWAT). Three low Level 1 studies⁶¹⁻⁶³ provided evidence that autolytic debridement with different dressings are as effective as each other and other forms of debridement in achieving improvement in pressure injury condition. A number of small economic analyses of high,⁶⁴ moderate^{58,65} and low⁶⁶ quality indicated that enzymatic debridement may be a more cost effective debridement method, but this finding is influenced by geographic location, clinical setting and duration of use.

One study in wounds of different etiologies provided indirect evidence that debridement weekly or more frequently was associated with increased hazard ratios for healing when compared with less than weekly debridement (HR = 4.26 (95% confidence interval [CI] 4.20 to 4.31)).⁶⁷ Additional indirect evidence indicated that wound bacterial sensitivity to antibiotics decreases to non-significant levels by 48 hours and returns to pre-debridement levels within 72 hours,²¹ suggesting maintenance debridement is required to treat biofilm (*Indirect evidence*).

Implementation Considerations

- Debridement should only be performed when there is adequate perfusion to the wound. Perform a vascular assessment prior to debridement of lower extremity pressure injuries to determine whether arterial status/supply is sufficient to support healing of the debrided wound.³⁷ See the guideline chapter *Heel Pressure Injuries* for more information on vascular assessment of the feet and heels (*Expert opinion*).
- Conservative sharp debridement, surgical/sharp debridement and ultrasonic debridement must be performed by specially trained, competent, qualified, and licensed health professionals consistent with local legal and regulatory statutes³⁷ (*Expert opinion*).
- Manage pain before commencing wound debridement.^{4,39-46,53,68} See the guideline chapter *Pain Assessment and Treatment* for information on managing pain.
- Use sterile instruments for conservative sharp, surgical/sharp and ultrasonic debridement³⁷ (*Expert opinion*).

Selecting the method of debridement

- Select the debridement method(s) most appropriate to the individual, the wound bed and the clinical setting (*Expert opinion*).
- Perform surgical/sharp debridement in the presence of extensive necrosis, advancing cellulitis, crepitus, fluctuance, and/or sepsis secondary to ulcer-related infection^{4,39,41-46,53,68} (*Expert opinion*).
- Perform conservative sharp or sharp debridement cautiously if the individual has immune incompetency, compromised vascular supply, or when there is a lack of antibacterial coverage in systemic sepsis. Relative contraindications include anticoagulant therapy and bleeding disorders^{4,39-46,53,68} (*Expert opinion*).
- Use mechanical, autolytic, enzymatic, and/or biological methods of debridement when there is no urgent clinical need for drainage or removal of devitalized tissue^{4,39-42,44-46,53,68} (*Expert opinion*).
- Refer individuals with Category/Stage III or IV pressure injuries with undermining, tunneling/sinus tracts, and/or extensive necrotic tissue that cannot be easily removed by other debridement methods for surgical evaluation as appropriate to the individual's condition and goals of care^{39-45,53,68} (*Expert opinion*).

Evidence Summary

Necrotic tissue is a nidus for infection, prolonging the inflammatory response, mechanically obstructing contraction, and impeding re-epithelialization.⁶⁹ It may mask underlying fluid collections or abscesses and limit full assessment capability in determining ulcer depth.⁶⁹ If appropriate to the individual's condition and consistent with overall goals of care, a thorough initial debridement of the pressure injury^{39-45,68} and the hyperproliferative epithelial edge should be performed to elicit an acute wound-healing response. Maintenance debridement should follow as dictated by the wound bed condition.^{33,35,36} Additionally, when a wound has delayed healing (i.e., four weeks or more) and fails to respond to standard wound care and/or antimicrobial therapy, have a high index of suspicion of the presence of biofilm and consider debriding the pressure injury. See the guideline chapter *Infection and Biofilms* for more information.

In cases where individuals are receiving palliative care, their overall quality of life should be taken into consideration when deciding whether to debride and the most appropriate type of debridement.^{41,53}

Methods of debridement

The most common methods used for debriding pressure injuries are:

- Sharp debridement (e.g., surgical/sharp or conservative sharp)
- Autolytic

- Enzymatic
- Biological
- Mechanical.

Sharp debridement

Sharp debridement includes surgical/sharp debridement and conservative sharp debridement. Surgical/sharp debridement is rapid wound debridement in which devitalized tissue is removed from the wound using scalpel and scissors under general or local topical anesthetic.³⁷ Surgical debridement extends into viable tissue, and the resultant bleeding stimulates the production of bloodborne endogenous growth factors acting as chemo attractants for inflammatory cells and mitogens for both fibroblasts and epithelial cells.^{33,50} It is usually confined to specialist inpatient clinics that have the capacity for anesthesia and the ability to maintain strict asepsis and control bleeding, and is performed by a surgeon, other qualified medical doctor, podiatrist, or advanced practitioner.³⁷

Surgical debridement is most appropriate when there is an urgent need to remove extensive, devitalized tissue. A pressure injury should be surgically debrided when there is a clinical need for extensive debridement; the degree of undermining and sinus tract/tunneling cannot be determined; there is advancing cellulitis; bone and infected hardware must be removed; and/or the individual is septic secondary to the pressure injury.^{37,70} Relative contraindications include anticoagulant therapy and bleeding disorders.

Conservative sharp debridement employs the use of scalpels, curettes, scissors, forceps, and rongeurs to remove devitalized tissue with limited pain or bleeding.⁷¹ This method of debridement decreases wound surface bacterial burden and removes senescent cells, converting a chronic wound into an acute wound.³³

Surgical/sharp and conservative sharp debridement should only be performed in anatomical locations possessing adequate vascularity to support the ability to heal.^{4,40,46,50} The chapter *Heel Pressure Injuries* includes information on conducting a vascular assessment of the lower extremities. When inadequate vascularity is present and vascular correction is impossible, the decision on whether to debride should be made between the individual, their family and a vascular or wound specialist, in consideration of risks and benefits.

Knowledge of anatomy and training is vital for a health professional using sharp debridement techniques. Caution must be exercised with immunocompromised individuals to avoid large open cavities that may serve as portals for opportunistic infection.³³ Additionally, caution must be exerted in those individuals with bleeding disorders and those taking anticoagulants.^{33,45,72} Access to conservative sharp debridement may be limited in certain care settings. Competency and/or certification to perform conservative sharp debridement may be mandated in some geographic regions.^{4,40,46}

Anvar and Okonkwo (2017)⁷³ reported outcomes for sacral, sacro-coccygeal, and coccygeal, ischial and trochanteric pressure injuries (n = 190) following conservative sharp debridement performed at the bedside by surgeons and surgical assistants. Mean surface area debrided was 3.2 inch² (20.8 cm²). At 12 week follow up, 73% of the pressure injuries demonstrated an improvement in surface area, with an average reduction in size of 40%⁷³ (Level 3). However, the severity of the pressure injuries was not reported and visual identification of biofilm was used to determine debridement requirements, which is not considered a plausible or valid method of biofilm identification⁵. Golinko et al. (2009)⁷⁴ note that surgical debridement should be performed until all devitalized tissue is excised. Their retrospective study conducted on pressure injuries suggested that histopathological analysis of tissue excised during surgical debridement can be used to determine the adequacy of the debridement because visual inspection of tissue alone may be inadequate. Study results demonstrated that using visual assessment alone, hyperkeratotic and fibrotic tissue and osteomyelitis remained even following surgical debridement undertaken by experienced surgeons (Level 4).

Williams et al. (2005)⁴⁹ in a non-randomized pilot study of individuals with chronic venous leg ulcers, reported that wounds receiving sharp circulator curette debridement exhibited significant healing at four weeks post-debridement when compared to those not receiving conservative sharp debridement, as measured by a decrease in mean ulcer surface area. There were no differences in infection rates between the two groups or a significant difference in mean surface area at 20 weeks. It is important to acknowledge that given the use of a less rigorous design, the groups are less homogenous, which may explain some of the variability. Those in the control group received no sharp debridement, and at baseline had no slough or devitalized tissue. They presented with 15 to 20% granulation, while those receiving debridement at baseline had slough and no granulation.

It is vital that health professionals who perform conservative sharp debridement or surgical/sharp debridement possess knowledge of anatomy and adequate training and experience.^{4,37,41,44,45} Although clean wound dressings may be appropriate for pressure ulcer management,³⁹ the instruments being used for conservative sharp or surgical/sharp debridement should be sterile.^{4,37,40}

Autolytic debridement

Autolysis is a highly selective form of slow debridement occurring naturally in all wound types.^{33,37} Macrophages phagocytize bacteria, and endogenous proteolytic enzymes such as collagenase, elastase, myeloperoxidase, acid hydrolase, and lysozymes selectively liquefy and separate devitalized tissue and eschar from healthy tissue.³³ The aim is to regulate the wound environment to achieve optimal moisture, pH and humidity in order that autolysis will occur.

Moisture-retentive wound dressings such as hydrocolloids, transparent films, and hydrogels rehydrate dry devitalized tissue and provide a moist environment for the body's own proteolytic enzymes and phagocytic cells to debride necrotic tissue.⁶⁹ In heavily exuding wounds, absorption dressings (e.g., calcium alginate, cellulose fiber) are more appropriate.

In two small, randomized controlled trials (RCTs) comparing amorphous hydrogels, no difference was noted in rates of debridement or healing^{61,63} (*Level 1*). This suggests that no specific type of amorphous hydrogel is superior to another for achieving autolysis.

In two small RCTs comparing autolytic debridement using hydrocolloids to enzymatic debridement using a topical enzyme (collagenase) varying results were reported. Among individuals with Category/Stage III pressure ulcers, Burgos et al. (2000) reported no difference in healing between collagenase and hydrocolloid use (*Level 1*), while Müller et al. (2001)⁶² found collagenase to be faster in achieving debridement of soft necrotic tissue and wound healing after removing the hard eschar in Category/Stage IV calcaneal pressure injuries (*Level 1*). Among individuals with Category/Stage III pressure injuries, Burgos et al. (2000)⁶² reported that those treated with collagenase exhibited a positive trend toward healing (83.3% healed) compared to (73.7%) in those treated with hydrocolloid, but this difference did not reach significance ($p = 0.754$). It is important to note that in Müller's 2001 study,⁶⁶ surgical debridement was performed prior to subject randomization. Both these studies were small, non-blinded studies that used subjective evaluations of wound size, and in the study by Burgos et al. (2000)⁶² greater than 30% of the participants withdrew from the study.

Autolytic debridement is contraindicated in the presence of untreated infection or extensive necrotic tissue, in large pressure injuries with undermining and sinus tracts, and in individuals with compromised immunity.^{39,41-43,45,53,68} Natural wound healing can be delayed in older adults.⁷⁵

Enzymatic debridement

Enzymatic debridement is accomplished by the application of exogenous proteolytic or fibrinolytic enzymes to the ulcer surface that will work synergistically with the body's own endogenous enzymes.^{37,69} The availability of enzymatic debriding agents may vary by country, and their properties and benefits in debridement vary. Fibrinolysin/deoxyribonuclease (DNAse) breaks down fibrin components of blood clots, inactivates fibrinogen and other clotting factors, and dilates the blood vessels, allowing macrophages to debride the devitalized tissue.³³ Bacterial collagenase degrades native collagen with great specificity, yet is not active against keratin, fat, or fibrin.³³ Papain, a proteolytic enzyme, is inactive against collagen and digests devitalized tissue through the liquefaction of fibrinous debris. Papain requires an activator to function; urea serving as an activator assists in denaturing nonviable protein, making it amenable to proteolysis.³³ Heavy metals may inactivate some enzymes. Follow manufacturer's directions when using enzymatic debriding agents.

A number of RCTs and cohort studies provide evidence on the use of collagenase for enzymatic debridement of pressure injuries. One retrospective database review⁵⁹ explored the use of collagenase as an adjunct to selective debridement for treating Category/Stage IV pressure injuries. It was assumed by the researchers that selective debridement method used was sharp debridement. Compared to pressure injuries that received only selective debridement, those that also received enzymatic debridement healed in a statistically significantly faster time frame (mean 456 days for collagenase versus mean 589 days for no collagenase, $p < 0.0001$), which translated to a hazard ratio of 1.85 (95% CI 1.28 to 2.68, $p = 0.001$). The healing rates overall were low in this study; only 11% of the non-collagenase group and 22% of the collagenase group achieved complete closure within 12 months⁵⁹ (*Level 3*). Another retrospective study compared Category/Stage III and IV pressure injuries receiving collagenase as an adjunct to negative pressure wound therapy (NPWT; $n = 67$) to those receiving only NPWT ($n = 47$). All pressure injuries received additional sharp debridement as required. There was no significant difference between the cohorts for change in wound surface area ($p = 0.322$); however, the pressure injuries that received adjunct collagenase treatment had significantly better scores ($p = 0.022$) on the Bates-Jensen Wound Assessment Tool that includes evaluation of wound bed characteristics (*Level 3*).⁵⁷ Thus the evidence on the benefits of adding enzymatic debridement with collagenase to a treatment regimen that includes sharp debridement is mixed.

Comparisons of collagenase to other enzymatic debridement agents (i.e., papain-urea⁷⁶ and fibrinolysin/deoxyribonuclease)⁷⁷ in Category/Stage II to IV pressure injuries also provide somewhat mixed evidence. While an RCT ($n = 28$) that compared to collagenase papain-urea reported the papain-urea product was superior in achieving

reduction in devitalized tissue ($p < 0.0167$) and improvement in granulation ($p < 0.0167$), overall healing rates were not different (*Level 1*). A second RCT ($n = 135$ included, $n = 78$ results analyzed) no significant difference ($p = 0.164$) was found between the two groups for the reduction of devitalized tissue⁷⁷ (*Level 1*). Both studies included small sample sizes and had methodological limitations (e.g., non-blinded outcomes measurement).

Comparisons have also been made between enzymatic debridement with collagenase and autolytic debridement. A small RCT ($n = 27$) demonstrated superior debridement of wounds with collagenase and a semi-occlusive dressing compared with a hydrogel dressing for Category/Stage III and IV pressure injuries. Approximately 85% of the pressure injuries managed with collagenase achieved complete debridement at 42 days compared with 29% of those treated with hydrogel dressing ($p < 0.03$). The pressure injuries debrided with collagenase were also statistically more likely to have achieved complete wound closure within 84 days (69% versus 21%, $p = 0.02$)⁵⁸ (*Level 1*). A larger study⁶⁰ made retrospective comparisons between collagenase ($n = 446$) and medicinal honey ($n = 341$) for debriding pressure injuries (primary Category/Stage III) in the outpatient setting. Fewer treatment visits were required for debridement with collagenase (9.1 ± 9.9 versus 12.6 ± 16.6 , $p < 0.001$). Pressure injuries treated with collagenase were 38% more likely to be fully granulated after 12 months (odds ratio [OR] 1.384, 95% CI 1.057 to 1.812, $p = 0.018$). Although the collagenase treated pressure injuries were statistically significantly more likely to be fully healed (complete epithelialization) at 12 months, the complete healing rate for both groups was low, and the difference might not be clinically significant (28.2% versus 21.3%, $p = 0.009$; OR 1.467, 95% CI 1.051 to 2.047, $p = 0.024$)⁶⁰ (*Level 3*).

Numerous economic analyses conducted in the US have reported cost-savings associated with using collagenase for pressure injury debridement. In a study on the care of 557 pressure injuries, Mearns et al. (2017)⁶⁴ reported lower costs over one year for debridement with collagenase compared to honey. Carter et al. (2017)⁶⁵ found cost savings to the US health system by performing debridement with collagenase as compared to sharp debridement. Waycaster et al. (2013)⁵⁸ found collagenase was a 3.2 times more cost effective method for debridement than a hydrogel dressing. In the Netherlands, Muller et al. (2001)⁶⁶ found costs associated with using collagenase were 5% lower than using a hydrocolloid dressing for autolytic debridement. These findings from the US and the Netherlands may not extrapolate to other geographic locations. Decision regarding the most appropriate method of debridement should be based foremost on the clinical condition of the wound and the individual's preferences, as well as the health professional's experience and accessibility of different treatment options.

Mechanical debridement

Mechanical debridement is often a non-selective form of debridement that can result in the removal of both devitalized as well as viable tissue.^{4,69} Examples of mechanical debriding agents include:

- Wet-to-dry dressings
- Monofilament/microfiber debridement pads
- Low frequency ultrasound (contact and non-contact)
- Hydrosurgery.

Wet-to-dry gauze dressings can be painful and may remove healthy tissue.⁴ Wet-to-dry gauze dressings are being used less frequently. Research suggests they are associated with slower wound healing and are costly in professional time due to the need for frequent wound dressing changes.^{78,79}

A monofilament/microfiber debridement pad removes slough and devitalized tissue, and potentially disrupts biofilm within the wound bed.^{80,81} Research on the use of monofilament/microfiber debridement pad is limited to its application in debriding the wound bed ($n = 13$) to enable better visualization of the wound bed for classifying the pressure injury⁸¹ (*Level 4*). In that study, the debriding process took no longer than four minutes; however, clinical outcomes for the wounds and tolerance of the individual were not reported. More research is required on its effectiveness in promoting wound healing.⁸⁰

Low frequency ultrasound (LFUS) debridement is increasingly being used to remove devitalized tissue. Low frequency ultrasonic debridement provides both mechanical and hydrodynamic effects directly in the wound bed due to cavitation.²² The application of ultrasound causes creation and destruction of small bubbles in irrigation fluid that expand and rapidly collapse ('imploding gaps') resulting in turbulent shockwaves and currents that lead to erosion of necrotic tissue and fibrin. Devices for both contact LFUS and non-contact LFUS are both used in wound debridement, with access to and licensing of different types of ultrasonic debridement devices varying across geographic locations. Evidence is primarily in other wound types, particularly venous leg ulcers and diabetic foot ulcers. Randomized controlled trials (RCTs) have demonstrated significant improvements in the wound bed status in wounds of other etiologies associated with both contact^{82,83} and non-contact⁸⁴⁻⁸⁶ LFUS debridement. The chapter *Biophysical Agents* has more information on ultrasound therapies used to promote healing.

The hydrosurgical water knife is an alternative tool to achieve surgical-type debridement. It can be regulated to precisely control the depth of debridement through pressure-setting calibration.^{87,88} Clinical evidence in wounds of different etiology indicates that hydrosurgery can achieve faster debridement than other methods. For example, a non-randomized study in chronic venous leg ulcers comparing hydrosurgery to autolytic debridement using a hydrogel wound dressing reported no difference in healing rates, but the time to achieve complete debridement was 1.3 ± 0.6 days for hydrosurgery, compared to 4.3 ± 3.9 days for hydrogel⁸⁹ (Level 5). Additionally, a retrospective study using historical controls of acute and chronic wounds having received hydrosurgery compared to conventional sharp debridement reported fewer surgical procedures being required in the hydrosurgery group⁹⁰ (Level 5). More recently, Ferrer-Sola et al. (2017)⁸⁸ utilized hydrosurgery to debride 29 chronic wounds (23% of which were pressure injuries) exhibiting slow wound healing and requiring rapid debridement. Approximately 73% of the chronic wounds required only one debridement session to stimulate healing, and the researchers noted a correlation between baseline surface area of the wound and the number of hydrosurgical debridement sessions required ($r = 3.07$).⁸⁸ Notably, this study reported that hydrosurgery was associated with low pain ratings (less than 5 on a 10-point scale) from participants when local or block anesthetic or systemic analgesia was used (Level 5).

Biological debridement

Biological debridement (larval therapy) consists of application of sterile fly larvae to the devitalized ulcer bed. Sterile maggots produce a mixture of proteolytic enzymes including collagenase, allantoin, and other agents with broad-spectrum antibacterial activity.^{33,69} Biological therapy should not be used where there are exposed blood vessels; acute infections that are limb- or life-threatening; ulcers requiring frequent inspections; necrotic bone or tendon tissues; or circulatory impairment significant enough to impair ability to heal.^{69,91}

In a clinical series of individuals with pressure injuries (n = 103 participants with n = 145 pressure injuries) treated with larval therapy compared with conventional debridement, faster debridement and granulation tissue formation was reported in those treated with maggots (0.8cm² versus 1.2cm² per week, p = 0.003)⁹¹ (Level 3).

Maintenance Debridement

Maintenance debridement is ongoing debridement to help maintain the wound bed in a healing mode. Beyond the obvious removal of devitalized tissue, research on other chronic wounds has shown that conservative sharp debridement or surgical debridement in particular effaces the wound bed of excess exudates and disassembles or detaches bacterial colonies (biofilms) and senescent fibroblasts, allowing a stimulatory environment to be established.^{33,35,36,50,72} The need for debridement is determined by both clinical parameters and the need to achieve optimal wound bed preparation. In pressure injuries that appear healthy but do not show evidence of closure, maintenance debridement is indicated.⁹²

While acute wounds may only require an initial debridement (if at all), chronic wounds often require maintenance debridement of the base as well as the non-migratory hyperproliferative epithelial edge.^{33,35,36,50,72,92} In a large observational study of chronic wounds of various etiologies (n = 312,744, 16% of which were pressure injuries) more frequent debridement was associated with improved healing. Wounds that were debrided weekly or more frequently were over four times more likely to heal than wounds receiving debridement less often than weekly (hazard ratio [HR] 4.26, 95% confidence interval [CI] 4.20 to 4.31).⁶⁷ (Level 5). Wolcott et al. (2010)²¹ demonstrated in invitro models and a small scale clinical study (n = 3 chronic wounds) that immature biofilm is more susceptible to topical antimicrobial treatment. Invitro models demonstrated that biofilm develops tolerance to antimicrobial treatment within 24 to 96 hours. Biofilm samples from venous leg ulcers subjected to conservative sharp debridement showed peak susceptibility to antibiotic therapy between 24 hours and 48 hours post-debridement. By 72 hours, susceptibility had returned to that of mature biofilm samples²¹ (Level 5). This suggests that when biofilm is suspected or confirmed in the pressure injury, maintenance debridement should be performed at least every 72 hours to promote sensitivity to antiseptic or antibiotic therapy by removing active cells from the surface of biofilm and exposing dormant bacteria (Level 5).

Continue maintenance debridement until the wound bed is free of devitalized tissue, covered with granulation tissue and progressing towards healing. Maintenance debridement should be resumed in the case of delayed wound healing that suggests presence of biofilm^{5,21,22} or with the return of any devitalized tissue or deteriorating granulation tissue.

Topical Agents for Pressure Injury Healing

There is a small body of evidence on a range of different topical products applied to the wound bed to promote healing (e.g., through promoting blood flow to the wound bed, reducing inflammation etc.). Topically applied products that have been explored in the pressure injury research include sildenafil,⁹³ atorvastatin,⁹⁴ insulin,⁹⁵ phenytoin,⁹⁶ nitric oxide,⁹⁷ hemoglobin,⁹⁸ hyaluronate,⁹⁹ and a range of different herbal/Chinese medicine preparations.¹⁰⁰⁻¹⁰⁴ The studies are small, and few studies make a comparison to contemporary wound care practices. No single product is reported in more than one study, and most studies are of low quality, therefore evaluation of clinical efficacy of topical products is not possible and no recommendations could be made.

References

1. Rodeheaver GT, Ratliff CR, *Wound Cleansing, Wound Irrigation, Wound Disinfection in Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*, D.I. Krasner, G.T. Rodeheaver, and R.G. Sibbald, Editors. 2007, HMP Communications: Malvern, PA.
2. Ho CH, Bensitel T, Wang X, Bogie KM. Pulsatile lavage for the enhancement of pressure ulcer healing: A randomized controlled trial. *Phys Ther*, 2012; 92(1): 38-48.
3. Luan XR, Li WH, Lou FL. Applied analysis of humanized nursing combined with wet healing therapy to prevent bedsore. *Eur Rev Med Pharmacol Sci*, 2016; 20(19): 4162-4166.
4. Wounds Australia, *Standards for Wound Prevention and Management*. 2016, Cambridge Media: Osborne Park, WA.
5. International Wound Infection Institute (IWII), *Wound Infection in Clinical Practice*. 2016, Wounds International.
6. Fernandez R, Griffiths R. Water for wound cleansing *Cochrane Database Syst Rev*, 2012; 2 (Art. No.: CD003861).
7. Joanna Briggs Institute. *Solutions, techniques and pressure for wound cleansing*. Best Practice, 2006; 10(2): 1-4.
8. Haesler E, Thomas L, Morey P, Barker J. A systematic review of the literature addressing asepsis in wound management. *Wound Pract Res*, 2016; 24(4): 208-246.
9. Beam JW. Wound cleansing: water or saline? *J Athl Train*, 2006; 41(2): 196-197.
10. Valente JH, Forti RJ, Freundlich LF, Zandieh SO, Crain EF. Wound irrigation in children: Saline solution or tap water? *Ann Emerg Med*, 2003; 41(5): 609-616.
11. Weir D. Clean it like you mean it! *Wound Management & Prevention* 2019; 65(2).
12. Fernandez R, Griffiths R, Ussia C. Effectiveness of solutions, techniques and pressure in wound cleansing. *JBHI Reports*, 2004; 2(7): 231-270.
13. Flanagan M, *Principles of wound management*, in *Wound Healing and Skin Integrity: Principles and Practice*, M. Flanagan, Editor. 2013, Wiley-Blackwell: Chichester.
14. Rahul S, Paul MK, Barreto E, Sreekar H, Dawre S. Syringe-based wound irrigating device. *Indian J Plast Surg*, 2012; 45(3): 590-591.
15. Wound Wise Editor. A fount of wound irrigation tips. *Nursing Made Incredibly Easy!*, 2007; 5(1): 14-15.
16. Maragakis LL, Cosgrove SE, Song X, Kim D, Rosenbaum P, Ciesla N, Srinivasan A, Ross T, Carroll K, Perl TM. An outbreak of multidrug-resistant *Acinetobacter baumannii* associated with pulsatile lavage wound treatment. *JAMA*, 2004; 292(24): 3006-3011.
17. Hiebert JM, Robson MC. The immediate and delayed post-debridement effects on tissue bacterial wound counts of hypochlorous acid versus saline irrigation in chronic wounds. *Eplasty*, 2016; 16: e32.
18. Moore Z, Cowman S. Wound cleansing for pressure ulcers. *Cochrane Database of Systematic Reviews*, 2013; 3.
19. Moore Z, Cowman S. A systematic review of wound cleansing for pressure ulcers. *Journal of Clinical Nursing*, 2008; 17(15): 1963-1972.
20. Bellingeri R, Attolini C, Fioretti O, Forma P, Trapedini M, Costa M, al. e. Evaluation of the efficacy of a preparation for the cleansing of cutaneous injuries. *Minerva Med*, 2004; 95(Suppl): 1-9.
21. Wolcott RD, Rumbaugh KP, James G, Schultz GS, Phillips P, Yang Q, Watters C, Stewart PS, Dowd SE. Biofilm maturity studies indicate sharp debridement opens a time- dependent therapeutic window. *J Wound Care*, 2010; 19(8): 320-328.
22. Leaper DJ, Schultz GS, Carville K, Fletcher J, Swanson T, Drake R. Extending the TIME concept: What have we learned in the past 10 years? *Int Wound J*, 2012; 9(Suppl 2): 1-19.
23. Reddi BAJ. Why Is saline so acidic (and does it really matter?). *Int J Med Sci*, 2013; 10(6): 747-750.
24. Edwards-Jones V, Flanagan M, Wolcott R. Technological advancements in the fight against antimicrobial resistance. *Wounds Int*, 2015; 6(2): 47-51.
25. Braun M, McGrath A, Downie F. Octenilin® range made easy. *Wounds UK*, 2013; 9(4).
26. Cutting K, Westgate S. The use of cleansing solutions in chronic wounds. *Wounds UK* 2012; 8(4): 130-133.
27. Drosou A, Falabella A, Kirsner RS. Antiseptics on wounds: An area of controversy. *Wounds* 2003; 15(5): 149-166.
28. Wound Healing and Management Group. Evidence summary: Wound infection: Iodophors and biofilms. *Wound Practice and Research*, 2013; 21(2): 86-87.
29. Leaper DJ, Durani P. Topical antimicrobial therapy of chronic wounds healing by secondary intention using iodine products. *Int Wound J*, 2008; 5(2): 361-368.
30. Konya C, Sanada H, Sugama J, Okuwa M, Kitagawa A. Does the use of a cleanser on skin surrounding pressure ulcers in older people promote healing? *J Wound Care*, 2005; 14(4): 169-171.
31. Konya C, Sanada H, Sugama J, Kitayama Y, Ishikawa S, Togashi H, Tamura S. Skin debris and micro-organisms on the periwound skin of pressure ulcers and the influence of periwound cleansing on microbial flora. *Ostomy Wound Management*, 2005; 51(1): 50-59.
32. Konya C, Sanada H, Sugama J, Okuwa M, Kitagawa A. Does the use of a cleanser on skin surrounding pressure ulcers in older people promote healing? *J Wound Care*, 2005; 14(4): 169-171.
33. Schultz GS, Sibbald RG, Falanga V, Ayello EA, Dowsett C, Harding K, Romanelli M, Stacey MC, Teot L, Vanscheidt W. Wound bed preparation: A systematic approach to wound management. *Wound Repair Regen*, 2003; 11(2): S1-28.
34. Saap LJ, Falanga V. Debridement performance index and its correlation with complete closure of diabetic foot ulcers. *Wound Repair Regen*, 2002; 10(6): 354-359.
35. Falanga V, *Wound bed preparation: science applied to practice*, in *European Wound Association Position Document: Wound Bed Preparation in Practice*. 2004, Medic: London.

36. Falanga V. Classifications for wound bed preparation and stimulation of chronic wounds. *Wound Repair Regen*, 2000; 8(5): 347-352.
37. Brown A. The role of debridement in the healing process. *Nurs Times*, 2013; 109(40): 16-19.
38. Steed D, Donohoe D, Webster M, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *J Am Coll Surg*, 1996; 183: 61-64.
39. Bergstrom N, Bennett, M.A., Carlson, C.E., et al, *Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. AHCPR Pub. No. 95-0653*. 1994, Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, Agency for Healthcare Policy and Research.
40. Keast DH, Parslow N, Houghton PE, Norton L, Fraser C. Best practice recommendations for the prevention and treatment of pressure ulcers: Update 2006. *Adv Skin Wound Care*, 2007; 20(8): 447-60.
41. European Pressure Ulcer Advisory Panel, *Pressure Ulcer Treatment Guidelines*. 1998, EPUAP: Oxford, England.
42. Wound Ostomy and Continence Nurses Society (WOCNS), *Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: WOCNS.
43. Royal College of Nursing (RCN), National Institute for Health and Clinical Excellence (NICE), *The management of pressure ulcers in primary and secondary care*. 2005, RCN and NICE: London.
44. University of Iowa College of Nursing (UICN), *Gerontological Nursing Interventions Research Center. Evidence-Based Practice Guideline Treatment of Pressure Ulcers*. 2000, UICN: University of Iowa.
45. Whitney J, Phillips L, Aslam R, Barbul A, Gottrup F, Gould L, Robson MC, Rodeheaver G, Thomas D, Stotts N. Guidelines for the treatment of pressure ulcers. *Wound Repair Regen*, 2006; 14(6): 663-679.
46. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
47. Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: A systematic review. *Health Technology Assessment (Winchester, England)*, 1999; 3(17 Pt 1): iii.
48. Hebda PA, Lo C. Biochemistry of wound healing: The effects of active ingredients of standard debriding agents papain and collagenase on digestion of native and denatured collagenous substrates, fibrin and elastin. *Wounds*, 2001; 13(5): 190-194.
49. Williams D, Enoch S, Miller D, Harris K, Price P, Harding KG. Effect of sharp debridement using curette on recalcitrant nonhealing venous leg ulcers: A concurrently controlled, prospective cohort study. *Wound Repair Regen*, 2005; 13(2): 131-137.
50. Zacur H, Kirsner RS. Debridement: Rationale and therapeutic options. *Wounds*, 2002; 14(7): 2E.
51. Gray D, Acton C, Chadwick P, Fumoral S, Leaper D, Morris C, Stang D, Vowden KR, Vowden P, Young T. Consensus guidance for the use of debridement techniques in the UK. *Wounds UK*, 2011; 7(1): 77-84.
52. Shannon MM. A retrospective descriptive study of nursing home residents with heel eschar or blisters. *Ostomy Wound Manage*, 2013; 59(1): 20-27.
53. AMDA, *American Medical Directors Association. Pressure Ulcers in the Long-Term Care Setting Clinical Practice Guideline*. 2008, Columbia, MD: AMDA.
54. Keast DH, Parslow N, Houghton PE, Norton L, Fraser C. Best practice recommendations for the prevention and treatment of pressure ulcers: Update 2006. *Adv Skin Wound Care*, 2007; 20(8): 447-60.
55. Falanga V, *Wound bed preparation: science applied to practice.*, in *European Wound Association Position Document: Wound bed preparation in practice*. 2004, Medic: London.
56. Wound Ostomy and Continence Nurses Society (WOCNS), *Wound Ostomy and Continence Nurses Society. Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: Wound Ostomy and Continence Nurses Society.
57. McCallon SK, Frilot C. A retrospective study of the effects of clostridial collagenase ointment and negative pressure wound therapy for the treatment of chronic pressure ulcers. *Wounds*, 2015; 27(3): 44-53.
58. Waycaster C, Milne CT. Clinical and economic benefit of enzymatic debridement of pressure ulcers compared to autolytic debridement with a hydrogel dressing. *J Med Econ*, 2013; 16(7): 976-986.
59. Carter MJ, Gilligan AM, Waycaster CR, Fife CE. Treating pressure ulcers with clostridial collagenase ointment: Results from the US Wound Registry. *Wound Repair Regen*, 2016; 24(5): 904-912.
60. Gilligan AM, Waycaster CR, Bizier R, Chu BC, Carter MJ, Fife CE. Comparative effectiveness of clostridial collagenase ointment to medicinal honey for treatment of pressure ulcers. *Adv Wound Care*, 2017; 6(4): 125-134.
61. Bale S, Banks V, Haglestein S, Harding KG. A comparison of two amorphous hydrogels in the debridement of pressure sores. *J Wound Care*, 1998; 7(2): 65-68.
62. Burgos A, Gimenez J, Moreno E, Lanberto E, Utrera M, Urraca EM, Vélez FJ, López E, Martínez MA, Gómez MJ, García L. Cost, efficacy, efficiency and tolerability of collagenase ointment versus hydrocolloid occlusive dressing in the treatment of pressure ulcers: A comparative, randomized, multicentre study. *Clin Drug Investig*, 2000; 19(5): 357-365.
63. Colin D, Kurring PA, Quinlan D, Yvon C. Managing sloughy pressure sores. *J Wound Care*, 1996; 5(10): 444-446.
64. Mearns ES, Liang M, Limone BL, Gilligan AM, Miller JD, Schaum KD, Waycaster CR. Economic analysis and budget impact of clostridial collagenase ointment compared with medicinal honey for treatment of pressure ulcers in the US. *Clinicoecon Outcomes Res*, 2017; 9: 485-494.
65. Carter MJ, Gilligan AM, Waycaster CR, Schaum K, Fife CE. Cost effectiveness of adding clostridial collagenase ointment to selective debridement in individuals with stage IV pressure ulcers. *J Spinal Cord Med*, 2017; 20(3): 253-265.
66. Muller E, van Leen MW, Bergemann R. Economic evaluation of collagenase-containing ointment and hydrocolloid dressing in the treatment of pressure ulcers. *Pharmacoeconomics*, 2001; 19(12): 1209-1216.
67. Wilcox JR, Carter MJ, Covington S. Frequency of debridements and time to heal: A retrospective cohort study of 312 744 wounds. *JAMA Dermatol*, 2013; 149(9): 1050-1058.

68. Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Pressure ulcer prevention and treatment following spinal cord injury: A clinical practice guideline for health-care professionals. *J Spinal Cord Med*, 2001; 24 (Suppl 1): S40.
69. Baharestani M, *The clinical relevance of debridement*, in *The Clinical Relevance of Debridement*, M. Baharestani, P. Holstein, and W. Vanscheidt, Editors. 1999, Springer-Verlag: Heidelberg, Germany. p. 1-15.
70. Sieggreen MY, Maklebust J. Debridement: Choices and challenges. *Adv Wound Care*, 1997; 10(2): 32-37.
71. Tomaselli N. WOCN position statement. Conservative sharp wound debridement for registered nurses. *J Wound Ostomy Continence Nurs*, 1995; 22(1): 32A-34A.
72. Whitney JD, Salvadlena G, Higa L, Mich M. Treatment of pressure ulcers with noncontact normothermic wound therapy: Healing and warming effects. *J Wound Ostomy Continence Nurs*, 2001; 28(5): 244-252.
73. Anvar B, Okonkwo H. Serial surgical debridement of common pressure injuries in the nursing home setting: Outcomes and findings. *Wounds*, 2017; 29(7): 215-221.
74. Golinko MS, Clark S, Rennett R, A. F, Boulton AJ. Wound emergencies: The importance of assessment, documentation, and early treatment using a wound electronic medical record. *Ostomy Wound Manage*, 2009; 55(5): 54-61.
75. Gould L, Abadir P, Brem H, Carter M, Conner-Kerr T, Davidson J, DiPietro L, Falanga V, Fife C, Gardner S, Grice E, Harmon J, Hazzard WR, High KP, Houghton P, Jacobson N, Kirsner RS, Kovacs EJ, Margolis D, McFarland Horne F, Reed MJ, Sullivan DH, Thom S, Tomic-Canic M, Walston J, Whitney JA, Williams J, Zieman S, Schmader K. Chronic wound repair and healing in older adults: Current status and future research. *J Am Geriatr Soc*, 2015; 63(3): 427-38.
76. Alvarez OM, Fernandez-Obregon A, Rogers RS, Bergamo L, Mazzo J, Black M. A prospective, randomized, comparative study of collagenase and papain-urea for pressure ulcer debridement. *Wounds*, 2002; 14(8): 293-301.
77. Pullen R, Popp R, Volkens P, Füsgen I. Prospective randomized double-blind study of the wound-debriding effects of collagenase and fibrinolysin/deoxyribonuclease in pressure ulcers. *Age Ageing*, 2002; 31(2): 126-130.
78. Matzen S, Peschardt A, Alsbjorn B. A new amorphous hydrocolloid for the treatment of pressure sores: A randomised controlled study. *Scand J Plast Reconstr Surg Hand Surg* 1999; 33(1): 13-15.
79. Singh A, Halder S, Menon GR, Chumber S, Misra MC, Sharma LK, Srivastava A. Meta-analysis of randomized controlled trials on hydrocolloid occlusive dressing versus conventional gauze dressing in the healing of chronic wounds. *Asian J Surg*, 2004; 27(4): 326-332.
80. Strohal R, Apelqvist J, Dissemmond J. EWMA document: Debridement. *J Wound Care*, 2013; 22(Suppl 1): S1-S52.
81. Dowsett C, Swan J, Orig R. The changing NHS and the role of new treatments: Using a monofilament fibre pad to aid accurate categorisation of pressure ulcers. *Wounds UK*, 2013; 9(4): 122-127.
82. Murphy C, Houghton P, Brandys T, Rose G, Bryant D. The effect of 22.5 kHz low-frequency contact ultrasound debridement (LFCUD) on lower extremity wound healing for a vascular surgery population: A randomised controlled trial. *Int Wound J*, 2018; 15: 460-472.
83. Herberger K, Franzke N, Blome C, Kirsten N, Augustin M. Efficacy, tolerability and patient benefit of ultrasound-assisted wound treatment versus surgical debridement: A randomized clinical study. *Dermatology*, 2011; 222(3): 244-249.
84. Ramundo J, Gray M. Is ultrasonic mist therapy effective for debriding chronic wounds? *J Wound Ostomy Cont Nurs*, 2008; 35(6): 579-583.
85. Ennis WJ, Formann P, Mozen N, Massey J, Conner-Kerr T, Meneses P. Ultrasound therapy for recalcitrant diabetic foot ulcers: Results of a randomized, double-blind, controlled, multicenter study [correction published erratum appears in *Ostomy Wound Manage* 2005;51(9):14]. *Ostomy Wound Management*, 2005; 51(8): 24-39
86. Kavros SJ, Schenck EC. Use of noncontact low-frequency ultrasound in the treatment of chronic foot and leg ulcerations: A 51-patient analysis. *J Am Podiatr Med Assoc*, 2007; 97(2): 95-101.
87. Attinger CE, Janis JE, Steinberg J, Schwartz J, Al-Attar A, Couch K. Clinical approach to wounds: Debridement and wound bed preparation including the use of dressings and wound-healing adjuvants. *Plast Reconstr Surg*, 2006; 117(7 Suppl): 725-109s.
88. Ferrer-Sola M. Hydrosurgery as a safe and efficient debridement method in a clinical wound unit. *J Wound Care*, 2017; 26(10): 593-599.
89. Mosti G, Mattaliano VI. The debridement of chronic leg ulcers by means of a new, fluidjet-based device. *Wounds*, 2006; 18(8): 227-337.
90. Granick MS, Posnett J, Jacoby M, Noruthun S, Ganchi PA, Datiashvili RO. Efficacy and cost-effectiveness of a high-powered parallel waterjet for wound debridement. *Wound Repair Regen*, 2006; 14(4): 394-397.
91. Sherman R, A. Maggot versus conservative debridement therapy for the treatment of pressure ulcers. *Wound Repair Regen*, 2002; 10(4): 208-214.
92. Falanga V, Brem H, Ennis WJ, Wolcott RD, Gould LJ, Ayello E. Maintenance debridement in the treatment of difficult-to-heal chronic wounds. *Ostomy Wound Manage*, 2008; June(Suppl): 2-15.
93. Farsaei S, Khalili H, Farboud ES, Khazaeipour Z. Sildenafil in the treatment of pressure ulcer: A randomised clinical trial. *Int Wound J*, 2015; 12(1): 111-117.
94. Farsaei S, Khalili H, Farboud ES, Karimzadeh I, Beigmohammadi MT. Efficacy of topical atorvastatin for the treatment of pressure ulcers: A randomized clinical trial. *Pharmacotherapy*, 2014; 34(1): 19-27.
95. Stephen S, Agnihotri M, Kaur S. A randomized, controlled trial to assess the effect of topical insulin versus normal saline in pressure ulcer healing. *Ostomy Wound Manage*, 2016; 62(6): 16-23.
96. Inchingolo F, Vermesan D, Inchingolo AD, Malcangi G, Santacroce L, Scacco S, Benagiano V, Girolamo F, Cagiano R, Caprio M, Longo L, Abbianante A, Inchingolo AM, Dipalma G, Tarullo A, Tattoli M. Bedsores successfully treated with topical phenytoin. *Acta Biomedica*, 2017; 88(1): 45-48.
97. Saidkhani V, Asadizaker M, Khodayar MJ, Latifi SM. The effect of nitric oxide releasing cream on healing pressure ulcers. *Iran J Nurs Midwifery Res*, 2016; 21(3): 322-330.

98. Tickle J. A topical haemoglobin spray for oxygenating pressure ulcers: A pilot study. *Br J Community Nurs*, 2015; Suppl Wound Care: S1-S18.
99. Felzani G, Spoletini I, Convento A, Di Lorenzo B, Rossi P, Miceli M, Rosano G. Effect of lysine hyaluronate on the healing of decubitus ulcers in rehabilitation patients. *Adv Ther*, 2011; 28(5): 439-445.
100. Buzzi M, Freitas F, Winter Me. Pressure ulcer healing with Plenusermax® Calendula officinalis L. extract. *Rev Bras Enferm*, 2016; 69(2): 250-257.
101. Li W, Ma Y, Yang Q, Pan Y, Meng Q. Moist exposed burn ointment for treating pressure ulcers. *Medicine (United States)*, 2017; 96 (29) (no pagination)(e7582).
102. Liu X, Meng Q, Song H, Zhao T. A traditional Chinese herbal formula improves pressure ulcers in paraplegic patients: A randomized, parallel-group, retrospective trial. *Exp Ther Med*, 2013; 5(6): 1693-1696.
103. Niu J, Han L, Gong F. Therapeutic effect of external application of ligustrazine combined with holistic nursing on pressure sores. *Med Sci Monit*, 2016; 22: 2871-2877.
104. Sipponen A, Jokinen JJ, Sipponen P, Papp A, Sarna S, Lohi J. Beneficial effect of resin salve in treatment of severe pressure ulcers: a prospective, randomized and controlled multicentre trial. *Br J Dermatol*, 2008; 158(5): 1055-1062.

INFECTION AND BIOFILMS

Introduction

Microorganisms are present on all skin surfaces. When the primary defense provided by intact skin is lost and a wound results, the microorganisms will contaminate and colonize the wound. When the microorganisms (by numbers or virulence in relation to host resistance) cause damage to the body, infection is present. Wound healing is delayed and/or may be abnormal when a pressure injury has a significant microbial burden or the host has an impaired immune system.

Pressure injuries have a high susceptibility to the development of infection¹ due to the presence of ischemia. Ischemic tissue does not receive an adequate supply of nutrition, oxygen, immune cells and antibodies, thus limiting the ability to respond to microorganism contamination. In addition, risk factors for pressure injury development (e.g., protein calorie malnutrition) compromise the host's defenses. Therefore, infection is not uncommon, particularly in Category/Stage III and IV pressure injuries and Unstageable pressure injuries.² A cross-sectional prevalence study set in nine long term care facilities in Spain reported the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) colonization in pressure injuries (n = 1,377) was 59%, highlighting the extent of pressure injury infection.³

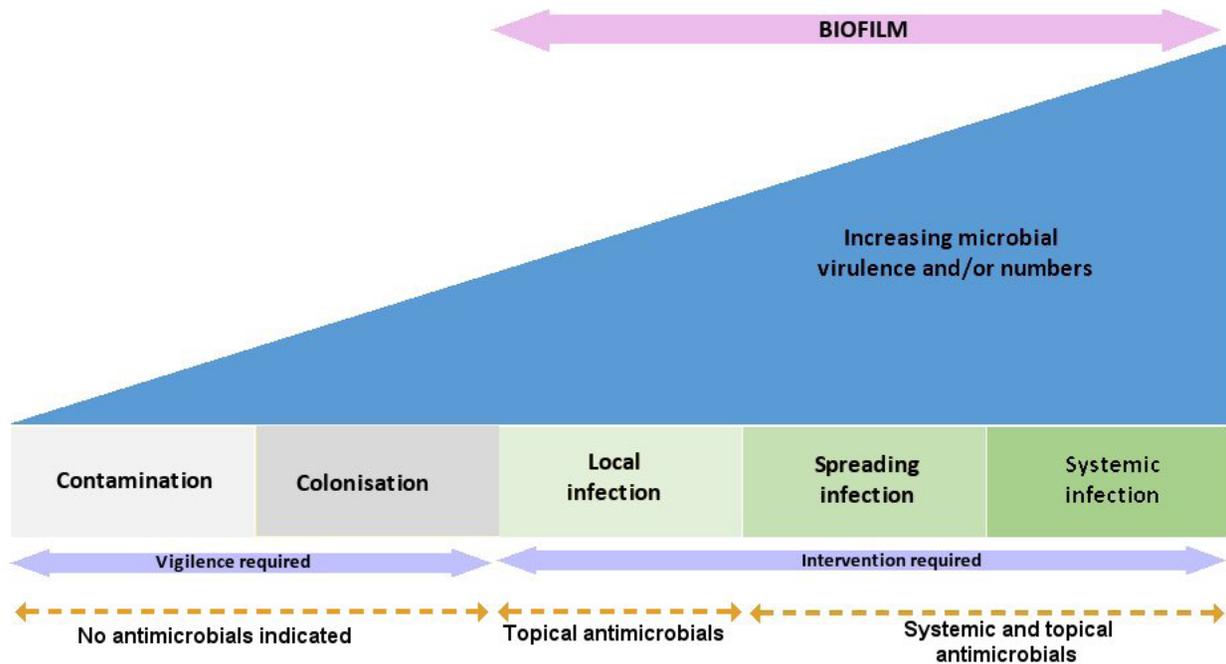
Necrotic tissue can increase the risk of infection because it contains high levels of both anaerobic and aerobic bacteria, in greater density than observed in non-necrotic pressure injuries.^{4,5} Tarnuzzer and Schultz (1996)⁶ have suggested that bacterial colonization in chronic wounds elevates proinflammatory cytokines, such as interleukin-1 and tumor necrosis factor. This in turn increases the levels of matrix metalloproteases (MMPs), decreases the level of tissue inhibitors of MMPs, and decreases the production of growth factors and fibroblast activity.

It is generally recognized that wound infection gradually increases over time with respect to the number and virulence of microorganisms. Conceptually, wound infection can be a continuum between a state of contamination to systemic pervasion of the host. Conceptualization of this continuum and the terminology used to describe the clinical characteristics continue to advance. Most recently, the International Wound Infection Institute (IWII) undertook a consensus study⁷⁻⁹ in which wound infection experts reached agreement on the continuum presented in *Figure 17.1*. As noted on the continuum, the number of microorganisms and their effect on the host can be categorized in the following stages:

- Contamination
- Colonization
- Local infection
- Spreading infection
- Systemic infection.

Sometimes, microorganisms multiply, invade, and damage tissues, which delays healing, and often causes systemic responses. Infection is present when there are microorganisms of sufficient numbers or virulence to impair wound healing and provoke clinical signs and symptoms in the individual.¹⁰ The types of microorganism seen in infected pressure injuries is varied. For example, in a retrospective study of surgical samples taken from infected pressure injuries, the predominant organisms were *Enterobacter* (29%), *Staphylococci* (28%), and *Enterococcus faecalis* (16%).¹¹ In Category/Stage II or greater pressure injuries evaluated in a hospital in Brazil,¹² 74% contained mixed flora, 49% were colonized with *Enterobacter*, 49% had *Escherichia coli*, and less than 10% had *Staphylococcus aureus*. In a hospital setting in Italy, Teseschi et al. (2017)¹³ reported *Staphylococcus aureus*, *Proteus mirabilis* and *Pseudomonas aeruginosa* as the most commonly seen pathogen in surgically repaired Category/Stage III and IV pressure injuries (n = 116). Bacterial profiles are influenced by the individual's geographic and clinical setting, for example, *Staphylococcus aureus* occurs more commonly in individuals in hospital settings.

Figure 17.1: International Wound Infection Institute Wound Infection Continuum
(reproduced with permission)^{9,14-16}



Biofilm may also be present in an infected pressure injury. Bacterial biofilms are extremely common in the natural environment.^{9,17-21} Biofilms are known to cause chronic inflammation that contributes to the molecular pathologies of many conditions, including periodontal disease, surgical device infections, urinary catheter infections, cystic fibrosis, chronic otitis media, and contact lens associated corneal infections.²² Compared to planktonic (free-floating) bacteria, bacteria in biofilms have enhanced resistance to endogenous antibodies and phagocytic cells, as well as to exogenous antibiotics and certain antiseptics. Approximately 60% of chronic skin wounds contain bacterial biofilms,^{23,24} which suggests that biofilms play important roles in chronic inflammation that ultimately leads to the failure of skin wounds to heal.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are accurate and effective methods to assess the presence of infection in a pressure injury?
- What are the accurate and effective methods to assess the presence of biofilm in a pressure injury?
- What is the role of topical agents in preventing and treating infection?
- What wound dressings are effective in reducing infection and/or biofilm?
- How should biofilm be treated?

Assessment of Infection or Biofilm

13.1: Have a high index of suspicion of local infection in a pressure injury in the presence of:

- Delayed healing
- Lack of signs of healing in the preceding two weeks despite appropriate treatment
- Larger size and/or depth
- Wound breakdown/dehiscence
- Necrotic tissue
- Friable granulation tissue
- Pocketing or bridging in the wound bed
- Increased exudate, or change in the nature of the exudate
- Increased warmth in the surrounding tissue
- Increased pain
- Malodor.

(Strength of Evidence = B1; Strength of Recommendation = ↔)

13.2: Have a high index of suspicion of biofilm in a pressure injury in the presence of:

- Failure to heal despite appropriate antibiotic therapy
- Recalcitrance to appropriate antimicrobial therapy
- Delayed healing despite optimal treatment
- Increased exudate
- Increased poor granulation or friable hypergranulation
- Low level erythema and/or low level chronic inflammation
- Secondary signs of infection.

(Good Practice Statement)**13.3: Consider a diagnosis of spreading infection if the individual with a pressure injury has local and/or systemic signs of acute infection including but not limited to:**

- Delay in healing
- Erythema extending from the wound edge
- Wound breakdown/dehiscence
- Induration
- Crepitus, fluctuance or discoloration of the surrounding skin
- Lymphangitis
- Malaise/lethargy
- Confusion/delirium and anorexia (particularly in older adults).

(Good Practice Statement)**Evidence Summary**

One high quality Level 1 study²⁵ demonstrated the classic signs and symptoms of infection have low sensitivity and specificity for determining conclusive presence of pressure injury infection when diagnosed with culture of exudate obtained via percutaneous aspiration. An earlier high quality Level 1 study found secondary signs of infection had stronger sensitivity and specificity for determining presence of infection than do classic signs of infection.²⁶ These studies suggest that the classic and secondary signs of infection listed in *Recommendation 13.1* indicate possible local wound infection that should be further investigated and confirmed.

Implementation Considerations

- Consider an individual to be at high risk of developing pressure injury infection if they have comorbidities associated with poor tissue perfusion and/or they are immunosuppressed⁹ (*Expert opinion*).
- Be alert to indirect indicators of infection in older adults (e.g., confusion/delirium and anorexia) who may not display classic signs of spreading infection²⁷ (*Expert opinion*).
- Consider pressure injuries to be at higher risk of infection if they are in anatomical locations with an increased risk of repeated contamination (e.g., sacral)^{9,28} (*Expert opinion*).
- Assess the environment and mitigate factors that increase risk of wound infection, including but not limited to unclean surfaces and bathing facilities, presence of dust or mold, poor hand hygiene and delayed management of incontinence episodes (*Expert opinion*).
- Include identification of classic and secondary signs and symptoms that increase suspicion of infection or biofilm in a pressure injury in health professional education (*Expert opinion*) (refer to the guideline chapter *Health Professional Education*).
- Teach individuals with pressure injuries and their caregivers to identify signs and symptoms of local and spreading infection, and ensure they have a plan to inform their health professional and adjust their treatment (*Expert opinion*) (refer to the guideline chapter *Quality of Life, Self-Care Skills and Education*).

Evidence Discussion

Blanco-Blanco et al. (2017)²⁵ undertook a prospective study exploring the concordance between classic signs of infection (heat, erythema, edema and purulent discharge) and wound infection confirmed via culture of wound exudate obtained from percutaneous aspiration. Of the 117 chronic wounds, 78% of which were pressure injuries, 58% exhibited at least one classic sign of infection. Signs of infection occurred more often in Category/Stage IV pressure injuries, and erythema ($p = 0.018$) and purulent exudate ($p = 0.024$) were significantly more likely to occur

in Category/Stage III and IV pressure injuries. Amongst these, 50.4% of the pressure injuries were confirmed to be infected via positive culture. Sensitivity of classic signs of infection to a positive culture was 0.36, specificity was 0.55, positive likelihood ratio was 0.79 and negative likelihood ratio was 1.17. The positive predictive value of classic signs of infection had a positive predictive value of 0.45 and a negative predictive value of 0.45.²⁵ These findings suggest that classic signs and symptoms have a poor ability to determine a true positive or true negative diagnosis of wound infection. These signs and symptoms should be considered in conjunction with secondary signs and symptoms and confirmed using wound culture (*Level 1*).

Gardner et al. (2001)²⁶ established similar results when reporting on the sensitivity of clinical signs and symptoms of chronic wound infection in chronic wounds, 53% of which were pressure injuries (n = 19). Sensitivities for the classic signs of infection, edema (0.64), erythema (0.55) and pain (0.36), were moderate to good. Sensitivities for heat (0.18) and purulent exudate (0.18) were low. Secondary signs of wound infection specific to chronic wounds had stronger sensitivities, including delayed healing (0.81), presence of friable granulation (0.82), discoloration (0.64), serous exudate with inflammation (0.55), wound breakdown (0.46), and malodor (0.36). Specificities ranged between 0.56 to 1.00. All (100%) the wounds that displayed increasing pain or wound breakdown/dehiscence were clinically infected²⁶ (*Level 1*).

'Bridging' is the presence of strands of epithelial tissue that form bridges across the wound bed.²⁹ Pocketing occurs when granulation tissue deposition is uneven and the undulating pockets of open tissue can harbor bacteria.³⁰ Bridging and pocketing have been confirmed as signs of potential wound infection in other types of chronic wounds (*Level 5*),³¹ and wound infection experts have reached agreement on the relationship of these characteristics to wound infection in consensus studies.^{9,32}

In a recent consensus study,⁷⁻⁹ wound infection experts reached agreement that the criteria listed in *Recommendation 13.2* are indicative of potential presence of biofilm that should be investigated further.

Chronic pressure injuries can develop acute spreading infection with resultant cellulitis and increased infection markers in the individual.^{9,33} It should be noted that older adults with infection may develop confusion or delirium, lose general function and become anorexic.²⁷

Some individuals may be at increased risk of developing pressure injury infection and biofilm. The immune responses required to combat infection amongst individuals with compromised host defenses are less robust and autoimmune disease and immunosuppression should be considered when evaluating the possibility of pressure injury infection.⁹ Category/Stage III and IV pressure injuries often occur in individuals with co-morbidities that both increase the risk of pressure injury development and simultaneously impair healing. Co-morbidities including diabetes mellitus, protein-calorie malnutrition, and hypoxia²⁶ all contribute to inadequate supply of nutrition and oxygen to the wound bed. Additionally, there is an increased risk of infection from environmental contaminants or fecal or urinary contamination.⁹

Diagnosis of Infection and Biofilm in a Pressure Injury

13.4: Determine presence of microbial burden in the pressure injury by tissue biopsy or semi-quantitative swab technique and microscopy.
(Good Practice Statement)

13.5: Determine presence of biofilm in the pressure injury by tissue biopsy and high resolution microscopy.
(Good Practice Statement)

Implementation Considerations

- Consider supplementing clinical assessment of the individual and the pressure injury with wound specimen collection and microbiological investigation:
 - When there is a high index of suspicion of local infection in an immunocompromised individual
 - When there are signs of spreading or systemic infection
 - When a pressure injury showing signs of infection or biofilm fails to respond to antimicrobial therapy or biofilm-based wound care

- o For compliance with local protocols for surveillance of drug-resistant microbials
- o Prior to undertaking some surgical procedures.⁹ (*Expert opinion*).
- Use the Levine technique (refer to *Table 17.1*) to take a wound swab of a pressure injury^{34,35} (*Level 5*).
- Identification of biofilm requires high resolution microscopy (e.g., fluorescence microscopy, confocal scanning microscopy, scanning electronic microscopy or transmission electron microscopy) (*Level 5 and expert opinion*).^{17,20,36-39}
- Microbiology results should be interpreted by an experienced health professional with consideration to the context of the individual and their clinical signs and symptoms. Consider consulting a microbiologist or infectious disease specialist for assistance in interpreting results and guidance in developing a treatment plan.⁹ (*Expert opinion*).

Discussion

The quantity of organisms (microbial load) is believed to be the best indicator of wound infection. The gold standard method for examining microbial load is quantitative culture of viable biopsied wound tissue. Wound tissue is viewed as the most valid specimen for quantitative tissue culture because tissue biopsies reflect organisms invading the wound, not those contaminating the wound surface. Surface wound swabs will only reveal colonizing organisms and may not reflect deeper tissue infection. This was demonstrated in a study by Rudensky et al. (1992)⁴⁰ in which 72 pressure injuries were evaluated for presence of infection using a range of diagnostic techniques. Wound swabs were positive for 96% of the pressure injuries tested, whereas the deep tissue aspirates were positive in only 43% of pressure injuries, and deep tissue biopsies were positive in 63% of the same wounds. Of the 43 pressure injuries that were assessed using all three methods, 98% screened positive via swab culture, 53% had positive deep tissue aspirate and 63% screened positive using tissue biopsy⁴⁰ (*Level 2*).

An acceptable alternative to quantitative culture of biopsied tissue is a semi-quantitative wound swab. In a small study (n = 25), Sapico et al. (1986)⁴¹ compared quantitative culture results from wound swabs taken using an unspecified technique and tissue biopsies taken during wound debridement. There was a mean concordance of 74.5% in the quantitative culture results. The concordance between samples taken from the central and peripheral wound bed was 63%, indicating that there is some variability based on the location in the wound bed from which the sample is taken (*Level 2*). Research undertaken in other types of chronic wounds concurs with this finding.⁴²

Research conducted in acute and chronic wounds has compared different swab techniques.^{34,35} Although studies comparing swab techniques are not definitive, the Levine technique is recommended for attaining a wound swab (refer to *Table 17.1*). This technique recovers microbial samples at and below the wound surface, increasing the ability to distinguish between wound colonization and local infection.^{9,34,35,43}

Table 17.1: Procedure for using Levine technique for wound swab^{9,34}

- Inform the individual about the procedure and address pain as required.
- Cleanse pressure injury with warm normal saline.
- Remove/debride nonviable tissue.
- Repeat cleansing of the pressure injury.
- Wait two to five minutes.
- If the wound bed is dry, moisten the wound swab tip with sterile normal saline.
- Culture the healthiest looking tissue in the wound bed.
- Do not culture exudate, pus, eschar, or heavily fibrous tissue.
- Place the wound swab into the wound, firmly press and rotate over 1 cm² of the wound.
- Apply sufficient pressure to swab to cause tissue fluid to be expressed.
- Use sterile technique to break tip of swab into a collection device designed for quantitative cultures.
- Label the specimen, including as much relevant history as appropriate.
- Apply a wound dressing.
- Transport the sample to the laboratory for processing within four hours.

While the validity of presence in the wound bed of characteristics indicative of biofilm (e.g., a slimy film) has been discussed in the literature,⁴⁴ there is agreement that there is currently no confirmed non-invasive, macroscopic method through which presence of biofilm can be visibly identified.^{9,17-21} The current gold standard for confirmation of presence of biofilm is microscopic examination using fluorescence microscopy, confocal scanning microscopy, scanning electronic microscopy or transmission electron microscopy.^{17,20,36-39}

The inadequacy of wound swabbing for evaluating the presence of biofilm has been demonstrated in studies of chronic wounds. In one diagnostic study, wedge tissue biopsies from chronic wounds (n = 15, n = 5 were pressure injuries) were analyzed using standard culture, gene sequencing and epifluorescence microscopy in order to inform

a taxonomy classification of biofilm organisms. Standard culture identified an average of three bacterial species in each sample compared with an average of 17 species identified using gene sequencing. Epifluorescence microscopy identified biofilm in 60% of samples.⁴⁵ Similarly, in a study in which culture analysis, light microscopy and scanning electron microscopy were used to assess 37 chronic wounds of mixed etiology (n = 21 pressure injuries), culture identified eight frequently observed bacteria species compared with 15 frequently occurring species identified used microscopy. Sixty percent of the sample contained biofilm⁴⁶ (both *Level 5*).

Despite these research findings, the value and cost effectiveness of using tissue biopsy and higher resolution microscopy in routine clinical evaluation of pressure injuries is yet to be demonstrated. Most geographic and clinical settings have limited or no access to these diagnostic techniques. Undertaking biofilm-based wound care in the presence of signs and symptoms indicative of biofilm in the pressure injury represents best clinical practice, despite the absence of confirmatory microbiology results.

Research continues to be undertaken on non-invasive and bed-side techniques for identifying biofilm in a wound.^{21,47,48} For example, wound blotting techniques that utilize blotted nitrocellulose membranes have been explored in 23 pressure injuries. The researchers compared the wound blot laboratory analysis with clinical development of slough in more than 10% of the wound bed at day seven (considered to be a clinical indicator of biofilm). The odds ratio of pressure injury with a biofilm-positive wound blot developing increased slough was 9.37 (95% confidence interval 2.47 to 35.5, p = 0.001).⁴⁷ Recent research has also explored the sensitivity and accuracy of bed-side bacterial fluorescence to assist health professionals in identifying bioburden in the wound bed.⁴⁸ Both these diagnostic techniques are still being further researched.

Diagnosing Osteomyelitis

13.6: Evaluate the pressure injury for presence of osteomyelitis in the presence of exposed bone and/or if the bone feels rough or soft, or if the pressure injury has failed to heal with appropriate treatment. (Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Two moderate quality Level 4 studies^{49,50} reported protocols that included pre-operative assessment for osteomyelitis and deep infection using tissue cultures and radiograms for all pressure injuries scheduled for surgical repair. Seven moderate and low quality studies reported protocols in which bone samples were taken for culture and sensitivity when osteomyelitis was suspected;⁵¹ when bone was exposed;⁵² when bone was rough or soft; or for all pressure injuries.^{49,53-56}

A high quality Level 2 study⁵⁷ comparing magnetic resonance imaging (MRI) to bone culture found 86% agreement on the presence or otherwise of osteomyelitis in the pressure injuries scheduled for surgical repair. Three low quality Level 4 studies⁵⁸⁻⁶⁰ reported good to excellent agreement on diagnosis of osteomyelitis using MRI scans.

Implementation Considerations

- Use plain film X-rays, white blood cell counts, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), bone scans, magnetic resonance imaging (MRI), computer tomography (CT) scan, and/or bone and tissue biopsy to investigate for osteomyelitis, depending on the clinical situation⁵⁹⁻⁶¹ Plain film X-rays, MRI and nuclear bone scan are considered the most useful^{57-60,62,63} (*Level 2, 3 and 4*).
- For individuals undergoing pressure injury surgery, investigations for osteomyelitis may be undertaken either pre-operatively^{49,50,64} or during the operative procedure^{49,51-56,64} (*Level 3 and 4*).

Evidence Discussion

Osteomyelitis has been reported in up to 32% of individuals with pressure injuries.⁶⁵⁻⁶⁷ Diagnostic assessments may include plain film X-rays, elevated white blood cell counts, elevated erythrocyte sedimentation rate (ESR), bone scans,⁶⁸ magnetic resonance imaging (MRI),⁵⁷ and biopsy, depending on the clinical situation.

Diagnostic assessment for pressure injury-related osteomyelitis includes clinical signs, laboratory evaluation and imaging, with selection determined by the clinical situation. Clinical signs include exposed bone, persistent sinus tracts, tissue necrosis and signs of local and systemic infection. Laboratory evaluation includes positive blood cultures and elevated erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP).^{62,63} It should be noted that bone biopsy with a culture is the definitive diagnostic tool,⁶² and most surgical protocols for pressure injury repair include harvesting bone samples during the surgical procedure when osteomyelitis is suspected^{49,51-56,64} (*Levels 3 and 4*). Pre-

operative imaging may also be utilized to identify soft tissue infection with or without bone involvement. Plain film X-rays, magnetic resonance imaging (MRI) and nuclear bone scan are considered the most useful.^{62,63}

Growing research demonstrates effectiveness of using MRI for the diagnosis of osteomyelitis. Brunel et al. (2016)⁵⁷ compared the accuracy of MRI scans to microbiological and pathological examination of bone samples for 44 pressure injuries. Histology was positive in 86.4% of pressure injuries (n = 38) and bone culture was positive for 93.2%. There was a positive MRI scan for 90.9% of the pressure injuries. Agreement between positive microbiology and histology was good (88.6%, $\kappa = 0.55$); however, agreement between MRI and composite criterion was lower (79.5%, $\kappa = 0.20$). Sensitivity for MRI was 94.3%, specificity 22.2%, and negative predictive value 50%.⁵⁷ A retrospective review of 41 MRI scans conducted on 37 individuals with pressure injuries showed a significant association between an intermediate to high probability of osteomyelitis and both cortical bone erosion (Pearson's $r = 0.84$) and abnormal bone marrow edema (Pearson's $r = 0.82$) on MRI scan. There was high interrater agreement ($\kappa = 0.92$, 95% CI 0.84 to 1.01, $p < 0.0001$) between radiographers on the likelihood of osteomyelitis⁵⁸⁻⁶⁰ (Level 4). However, a retrospective case-controlled study of individuals (n = 65) with osteomyelitis undergoing flap reconstruction determined that a diagnostic preoperative MRI scan did not significantly alter clinical or surgical management of the individual, nor patient outcomes compared to diagnosis through bone cultures taken during the surgical procedure⁶⁴ (Level 4).

Permanent healing of the pressure injury is unlikely until osteomyelitis is controlled. Treatment of osteomyelitis is beyond the scope of these guidelines.

Treatment for Pressure Injury Infection and Biofilm

13.7: Optimize potential for healing by:

- Evaluating the individual's nutritional status and addressing deficits
- Evaluating the individual's comorbidities and promoting disease control
- Reducing the individual's immunosuppressant therapy if possible
- Preventing contamination of the pressure injury
- Preparing the wound bed through cleansing and debridement.

(Good Practice Statement)

Implementation Considerations

- Undertake a comprehensive assessment of the individual, the pressure injury and the healing environment to identify comorbidities that can negatively impact wound healing and increase the risk of infection⁶⁹ (see the guideline chapter *Assessment of the Pressure Injury and Monitoring of Healing*).
- Maintain the pressure injury free of necrotic tissue and slough through regular cleaning and debridement (see the guideline chapter *Cleansing and Debridement*).
- Follow local infection control policies to prevent self-contamination and cross-contamination in individuals with pressure injuries⁶⁹ (*Expert opinion*).

Discussion

Many systemic factors contribute to the development of pressure injuries. If these same factors can be mitigated or improved, the individual's intrinsic ability to fight infection can usually increase. Review the individual's nutritional intake, modify it if needed and stabilize diabetic glycemic control (see the guideline chapter *Nutrition in Pressure Injury Prevention and Treatment*). Assess vascular supply to the pressure injury (see the guideline chapter *Heel Pressure Injuries*) and instigate appropriate management for peripheral arterial disease (e.g., management of blood pressure and cholesterol, encouraging the individual to cease smoking and medical or surgical management as appropriate). If possible, reduce immunosuppressive agents.

Necrotic tissue and slough promote bacterial growth. Cleansing removes loose debris and planktonic (free-floating) bacteria. Debridement is often required to remove adherent slough, eschar and biofilm. Refer to the guideline chapters *Cleansing and Debridement* and *Pressure Injury Surgery* for recommendations on debridement of the wound bed.

Pressure injuries near the anus are subject to contamination, especially by bacteria from the colon. Predominant organisms in infected pressure injuries include *Enterobacter* species, *Proteus* species, *Escherichia coli*, and *Enterococcus faecalis*.^{11,13} Meticulous skin cleansing and use of wound dressings or topical agents to prevent exposure to fecal matter reduces the risk of contamination. At times, bowel management systems and diversion ostomies could be considered to reduce continuous exposure to feces of the pressure injury (or the surgical site following a surgical repair).^{70,71} The *Preventive Skin Care* section of the guideline provides further recommendations on structured skin

hygiene programs^{72,73} to reduce the risk of contamination to the wound bed.

13.8: Use topical antiseptics in tissue-appropriate strengths to control microbial burden and to promote healing in pressure injuries that have delayed healing.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence from one moderate⁷⁴ quality and one low⁷⁵ quality Level 1 study and three low quality Level 4⁷⁶⁻⁷⁸ studies provides support for microbial effect of various topical antiseptics in reducing microbials of a range of different topical antiseptics in reducing bioburden in pressure injuries. The effect size is difficult to estimate due to the small sample sizes in studies and the failure to diagnose wound infection at the outset in studies.^{75,77} Additional low quality, older studies conducted in small samples provide support for this recommendation.^{2,77,79-83} Many topical antiseptics are toxic to tissues and should be used at the lowest possible concentrations and shortest duration to reduce the risk of adverse effects. Evidence on resource requirements is lacking. Not all contemporary and emerging antiseptics are universally available in all geographic or clinical settings.

Implementation Considerations

- Refer to international wound infection management guidelines to select the most appropriate topical antiseptic for the individual and the pressure injury⁹ (*Expert opinion*).
- Consider the use of topical antiseptics for pressure injuries that are not expected to heal and are critically colonized/topically infected (*Expert opinion*).
- Use antiseptics at the lowest effective concentrations to reduce the risk of harm to the wound bed. Some antiseptics are cytotoxic to skin and tissue cells in higher concentrations.^{9,84-86}
- Use a topical antiseptic for two weeks before evaluating its effectiveness in reducing infection.^{9,87,88} A two-week challenge is recommended; however, if the pressure injury deteriorates, re-evaluate the treatment plan earlier (*Expert opinion*).
- Some antiseptics can be painful on application. Treat pain prior to undertaking the wound care procedure. Pain management options are discussed in the guideline chapter *Pain Assessment and Treatment* (*Expert opinion*).
- Consider the individual's allergies, sensitivities, clinical history and preferences when selecting a topical antiseptic. Some topical antiseptics have contraindications^{9,89-95} (see below for an overview and carefully review manufacturers' instructions before using a topical antiseptic) (*Expert opinion*).

Evidence Discussion

Antiseptics are agents that destroy or inhibit the development and growth of microorganisms in or on living tissue. Unlike antibiotics that act selectively on a specific target, antiseptics have multiple targets and a broader spectrum of activity that includes bacteria, fungi, viruses, protozoa, and prions. Antiseptics that are commonly used in wounds (either for cleansing or infection treatment or both) include:^{9,84,96,97}

- Iodine (e.g., povidone iodine and slow-release cadexomer iodine)
- Silver (e.g., salts, metallics, ionic silver combined with antibiofilm agents)
- Enzyme alginogels
- Polyhexamethylene biguanide (PHMB)
- Medical-grade honey
- Super oxidized solution with hypochlorous acid (HOCL)
- Surfactants.

Older antiseptics that are commonly used in some geographic and clinical settings include chlorhexidine, sodium hypochlorite and acetic acid. *Table 17.2* includes a summary of the characteristics of antiseptics used for treating infection. Additionally, *Table 16.2* in the guideline chapter *Cleansing and Debridement* includes information on antiseptics that are primarily used as cleansers (e.g., PHMB and HOCL).

Cytotoxicity is the main concern when applying a topical agent to an open wound. Antiseptics have been found, primarily using *in vitro* models, to be cytotoxic to cells essential to the wound healing process, including fibroblasts, keratinocytes, and leukocytes.^{9,84,96,97} However, cytotoxicity appears to be concentration dependent, as several antiseptics in low concentrations are not cytotoxic, although they retain their antibacterial activity *in vitro*. Newer

antiseptics do not have the same cytotoxicity concerns. Care should be taken to protect the peri-wound area from topical antiseptics and to manage pain associated with application. Topical antiseptics should be discontinued when infection is managed, or the wound starts to heal, or if the patient experiences any adverse reaction to the agent.⁹⁸

Table 17.2: Topical wound infection therapies (reproduced with permission from IWII)⁹

Antimicrobial agent	Type	Biofilm Efficacy	Guidance for use
Enzyme alginogel	Alginate gel with two enzymes: <ul style="list-style-type: none"> Lactoperoxidase Glucose oxidase 	<ul style="list-style-type: none"> Prevents formation of biofilms at concentration $\leq 0.5\%$ (w/v)^{99,100} Inhibits growth of established biofilms at higher concentrations Does not disrupt biofilm biomass^{99,100} 	<ul style="list-style-type: none"> Concentrations of alginate of 3% and 5% depending on level of exudate^{99,100}
Iodine (povidone and cadexomer)	<ul style="list-style-type: none"> Solution Impregnated wound dressings Powder and paste 	<ul style="list-style-type: none"> Inhibits development of new biofilm^{101,102} Eradicates young biofilm colonies^{101,103} Significantly reduces mature biofilm colonies^{101,102} 	<ul style="list-style-type: none"> Contraindicated in individuals sensitive to iodine or with thyroid or renal disorders¹⁰¹ Contraindicated in extensive burns¹⁰¹
Honey	<ul style="list-style-type: none"> Medical grade Honey impregnated dressings 	<ul style="list-style-type: none"> Inhibits biofilm growth^{100,104,105} Reduces biofilm colony formation¹⁰⁶ Inhibits quorum sensing of biofilm thereby reducing ability to proliferate¹⁰⁷ 	<ul style="list-style-type: none"> Select products that have been gamma irradiated¹⁰⁶ Leptospermum species is more effective than other types¹⁰⁶
Silver	<ul style="list-style-type: none"> Salts (e.g., silver sulfadiazine, silver nitrate, silver sulphate, silver CMC) Metallic (e.g. nanocrystalline, silver-coated nylon fibers) Impregnated wound dressings 	<ul style="list-style-type: none"> Denatures existing bacterial biofilm in concentrations $\geq 5\mu\text{g/ml}$ 	<ul style="list-style-type: none"> Change more frequently in wounds with heavy exudate Avoid in individuals with silver sensitivities¹⁰⁸
Ionic silver combined ethylenediamin tetraacetate (EDTA) and benzethonium chloride (BEC) (antibiofilm agents)	<ul style="list-style-type: none"> Carboxymethylcellulose gelling dressing impregnated with ionic silver enhanced with EDTA and BEC 	<ul style="list-style-type: none"> Combines antibiofilm and antimicrobial components that work in synergy to disrupt biofilm and expose associated microorganisms to the broad-spectrum antimicrobial action of ionic silver¹⁰⁹ Eradicates mature biofilm within 5 days¹¹⁰ Prevents biofilm formation¹¹⁰ Associated improvement in healing rates¹¹¹ 	<ul style="list-style-type: none"> Change more frequently in wounds with heavy exudate Avoid in individuals with sensitivities to silver, EDTA or BEC¹¹²
Surfactant	Concentrated surfactant gels with antimicrobial preservatives	<ul style="list-style-type: none"> Prevents biofilm formation¹¹³ Increases antibiotic efficacy Eradicates mature biofilm 	<ul style="list-style-type: none"> Can be used between and post-debridement to prevent re-establishment of biofilm May require daily application for the first few days

The evidence base for most antiseptic products is in wounds of other etiologies (particularly venous leg ulcers and diabetic foot ulcers) and therefore does not meet the inclusion criteria for this guideline. A recent Cochrane review also noted a paucity of research on the use of antiseptics in treatment of infection in pressure injuries, noting that evidence is inconsistent when comparing different treatment regimens. Most studies are small and have methodological limitations.¹¹⁴ Guideline users are encouraged to review local, national and international⁹ wound infection guidelines

and product information when evaluating the effectiveness of antiseptics and their appropriateness for use with specific individuals. Evidence conducted in pressure injury populations and a broad product overview is provided below.

Enzyme alginogels

Enzyme alginogels are a newer class of antiseptic that combine hydrogels, alginates and antimicrobial enzymes (e.g., lactoperoxidase and glucose oxidase). The enzymes work by destroying bacterial cell walls, therefore these products are able to prevent formation of new biofilms and inhibit the growth of existing biofilm colonies.^{99,100}

Iodine

Povidone iodine and cadexomer iodine are low cost topical antiseptic options available as solutions, impregnated wound dressings, powders and pastes. *In vitro* studies have found povidone iodine to be toxic to granulocytes in concentrations above 0.05%;^{84,115} however, animal and clinical studies in mixed etiology wounds have found no reduction in healing rates for povidone iodine in concentrations up to 10% compared with normal saline.^{89,115,116} Iodine is effective in inhibiting the development of new biofilm and reducing both young and mature biofilm in the wound, increasing its usefulness in biofilm-based wound care¹⁰¹⁻¹⁰³ (*all Level 5*). Iodine products should be avoided in individuals with renal failure, history of thyroid disorders or known iodine sensitivity.^{93,94}

The Cochrane review¹¹⁴ reports a summary of four small trials meeting inclusion for this guideline⁸⁰⁻⁸³ that explore use of iodine products for treating pressure injuries. The review¹¹⁴ reports risk ratios from 0.64 (95% CI 0.43 to 0.97) to 0.81 (95% CI 0.48 to 1.37) when compared to other antiseptics for eradicating infection and a risk ratio of 6.0 (95% CI 0.80 to 45.20) when compared to not using an antiseptic.^{82,114} The studies are small and considered to have a high risk of bias.

Honey

Topical medical-grade honey offers broad antimicrobial coverage. A growing body of literature has shown benefit to using medical-grade honey for infected wounds of various etiologies.^{77,117-119} Manuka honey should be rated UMF (Unique Manuka Factor) +12 or above for topical dressing products. Use medical-grade gamma irradiated honey, as other sterilizing processes destroy the UMF in the honey.⁶⁹ Because honey produces an alternative product for bacterial metabolism that yields lactic acid rather than ammonia, amines, and sulfur (which are odorous) wound odor can be reduced through its use. Medical-grade honey can be also used in heavily contaminated or infected pressure injuries until definitive debridement is accomplished.

Yapuca Gunés and Eser¹²⁰ conducted an RCT that included 26 participants with 68 Category/Stage II and III pressure injuries. The study compared healing rates in pressure injuries treated with unprocessed honey that had a minimum inhibitory concentration (MIC) of 3.8%, to those treated with ethoxy-diaminoacridine plus nitrofurazone dressing. Scores on the Pressure Ulcer Scale for Healing Tool (PUSH®) showed the honey group healed at four times the rate of the control group ($p < 0.001$) (*Level 1*). A Cochrane review¹²¹ identified one small RCT ($n = 40$)¹²² comparing medical-grade honey to saline-soaked gauze for healing Category/Stage I and II pressure injuries. Although mean time to healing favored the honey treated group ($p =$ not reported), no outcome measures specifically investigated the effect of honey on controlling infection, and the pressure injuries in this study were described as uninfected at baseline (*Level 1*). Biglari et al (2012)⁷⁷ reported a case series of 20 individuals with spinal cord injury (SCI) and Category/Stage III or IV pressure injuries that were treated with medical-grade honey. After one week of daily cleansing with Ringer's solution and application of 3 mm thick honey, 90% of the pressure injuries were void of bacterial growth. However, baseline clinical infection status was not reported (*Level 4*).

Silver

Silver comes in a range of different types (e.g., salts, metallics and ionic preparations) and formulas (e.g. impregnated dressings and pastes). Silver may have toxic properties, especially to keratinocytes and fibroblasts; the extent of the toxicities is not fully described. Topical silver products should not be used on individuals with silver sensitivities, and silver sulfadiazine products are not recommended for people with sulfur sensitivities.¹²³ Also consider sensitivities to other ingredients (e.g., silver is available in combination with antibiofilm agents: ethylenediaminetetraacetate and benzethonium chloride).

Ionic silver combined with antibiofilms has antibiofilm and antimicrobial components that disrupt biofilm and expose microorganisms to the broad-spectrum action of silver.¹⁰⁹ It is reported to rapidly eradicate mature biofilm and prevent new biofilm colonies.¹¹⁰ Silver salts, metallics and impregnated dressings are also reported to be active against biofilm. However, clinical research on the use of silver in pressure injuries is limited. The Cochrane review by Norman et al. (2016)¹¹⁴ identified one small ($n = 26$) study⁸¹ comparing silver sulfadiazine to povidone iodine, reporting a risk ratio of 0.65 (95% CI 0.41 to 1.01).¹¹⁴ Evidence from a small case study⁷⁸ ($n = 20$) exploring a silver-containing dressing indicated

that a hydrofiber silver dressing is associated with an 80% reduction in bioburden in the wound bed after seven days of treatment, as evaluated using tissue biopsy (*Level 4*).

Biofilm-Based Wound Care

Biofilm-based wound care describes treatment in which biofilm growth is disrupted through debridement and, in the window of opportunity following removal of more mature biofilm, antiseptic treatment is used to prevent reformation of biofilm.

13.9: Use topical antiseptics that are active against biofilm in tissue-appropriate strengths in conjunction with regular debridement to control and eradicate suspected (or confirmed) biofilm in pressure injuries with delayed healing.

(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence summary

A low quality Level 5 study¹²⁴ conducted in chronic wounds with biofilm showed significant increase in wound bed granulation after management with debridement and 0.3% PHMB, with 75% of wounds reaching complete healing. Indirect evidence from Level 5 studies has demonstrated biofilm susceptibility to povidone iodine in concentrations of 1% to 10%,^{103,125,126} to cadexomer iodine paste,¹²⁷ and to a lesser extent, silver sulfadiazine,^{103,128,129} all in laboratory studies. A laboratory based comparison between iodophors and silver suggested iodophors are more effective in decreasing biofilm.¹³⁰ These findings are supported by international consensus documents.^{9,10,98}

Implementation Considerations

- Refer to *Recommendation 12.5* for evidence on the effectiveness of debridement in addressing biofilm in the pressure injury and implementation considerations in performing debridement.
- Refer to *Recommendation 13.8* for evidence on the effectiveness of topical antiseptics in reduction of microbial burden in pressure injuries with biofilm and implementation considerations in using antiseptics.
- In chronic pressure injuries with suspected biofilm (or biofilm diagnosed with high resolution microscopy), undertake biofilm-based wound care for at least 72 hours or until the pressure injury progresses towards healing¹³¹ (*Level 5*).
- Resume biofilm-based wound care in the case of delayed healing that suggests returned presence of biofilm^{9,131,132} (*Level 5*).

Evidence Discussion

Biofilm-based wound care is the current optimal treatment to disrupt the chronic inflammatory phase of a pressure injury and promote transition to a healing repair phase. However, biofilm tends to redevelop, and maintenance debridement in conjunction with topical antiseptic therapy must continue until the pressure injury is clear of biofilm.^{18,19,88,99,133,134} Indirect evidence from laboratory studies has demonstrated susceptibility of biofilm to a range of antiseptics, as outlined in *Table 17.2*; however, *in vitro* studies support the notion that biofilm develops resistance to topical antiseptics as it matures (*Level 5*). Therefore, removing mature biofilm from the wound bed optimizes the impact of antiseptics on developing and immature biofilm colonies. The guideline chapter on *Cleansing and Debridement* includes an extended discussion on the role of maintenance debridement in the ongoing treatment of biofilm, including the optimal timeframe in which to treat the pressure injury.

Despite consensus on the role of biofilm-based wound care in treating biofilm, there is a paucity of clinical evidence on its effectiveness. The large body of evidence primarily focuses on laboratory exploration of the response of biofilm to different antiseptics. However, Eberlein et al. (2012)¹²⁴ demonstrated effectiveness of biofilm-based wound care using a 0.3% PHMB impregnated wound dressing and maintenance debridement in 16 chronic wounds with clinical signs of biofilm. In this study, a significant increase in granulation of the wound bed ($p < 0.04$) was demonstrated after 24 weeks of treatment, with 75% of the chronic wounds reaching complete healing in this timeframe¹²⁴ (*Level 5*).

Wolcott et. al.¹⁹ demonstrated in *in vitro* models and a small-scale clinical study that less mature biofilm is more susceptible to topical antimicrobial treatment. *In vitro* models demonstrated that biofilm develops tolerance to antibiotic treatment within 24 to 96 hours and suggested that removal of active cells from the surface of biofilm exposes dormant bacteria that have increased susceptibility to treatment. Biofilm samples from venous leg ulcers subjected to conservative sharp debridement showed peak susceptibility to topical antibiotics between 24 hours and 48 hours post debridement. By 72 hours, susceptibility had reduced to that of mature biofilm samples (indirect evidence).

Antibiotic Therapy for Pressure Injury Infection

Due to rising rates of antibiotic resistance, antibiotic therapy should only be used when indicated. In general, topical antibiotics are not recommended for treating pressure injuries. Short courses of topical antibiotic solutions or topical metronidazole can be useful in some very limited circumstances; for example on pressure injuries that have been debrided and cleansed, yet still have a high bacterial bioburden and/or the presence of beta hemolytic Streptococci.^{9,133,135} However, judicious use of systemic antibiotics remains an important consideration when the individual shows signs of spreading or systemic infection.

**13.10: Use systemic antibiotics to control and eradicate infection in individuals with pressure injuries and clinical evidence of systemic infection.
(Good Practice Statement)**

Implementation Considerations

- Consider using systemic antibiotics in the presence of positive blood cultures, cellulitis, fasciitis, osteomyelitis, systemic inflammatory response syndrome (SIRS), or sepsis (*Expert opinion*).
- Local abscesses (the collection of pus) should be incised and drained to prevent local or systemic spread of the infection (*Expert opinion*).
- Limit the use of topical antibiotics on infected pressure injuries, except in special situations where the benefit outweighs the risk of antibiotic side effects and antibiotic resistance (*Expert opinion*).
- Follow local protocols for the selection and commencement of antibiotic therapy (*Expert opinion*).

Evidence Discussion

Pressure injuries are a known cause of sepsis and death.¹³⁶⁻¹³⁹ Abscessed or grossly infected pressure injuries should be drained and debrided to treat pressure injury related sepsis or advancing cellulitis.

Systemic antibiotics can reach infected tissue in the base of the pressure injury, whereas topically applied agents cannot penetrate through necrotic tissue to reach the wound bed below. Systemic antibiotics should be chosen based on confirmed antibiotic susceptibilities of the pathogens. For life-threatening infections, empiric antibiotics should be based on local antimicrobial susceptibility patterns, and re-evaluated when definitive cultures become available^{9,10,140,141} In some instances, the use of antibiotics may be limited by individual preference or advance directives for end-of-life care.

In a retrospective study¹⁴² including primarily Category/Stage IV pressure injuries (56 participants with 115 pressure injuries) referred for surgical consultation, 4% of participants had clinical signs of infection and 13% of participants were positive for MRSA colonization, despite 96% of participants undertaking a course of antibiotics in the preceding two weeks. This study highlighted the issue of over prescription of antibiotics and development of antibiotic-resistant bacterial strains.¹⁴² Cataldo et al. (2011)¹⁴³ reported a prevalence rate of 15% for MRSA in a convenience sample of older adults with at least Category/Stage III pressure injury (n = 32) in home care in Italy. Almost 38% of the participants had received systemic antibiotic therapy in the preceding 90 days. In a retrospective study conducted in participants (n = 145) in a Brazilian hospital who had Category/Stage II or greater pressure injuries, 43.5% of participants had an MRSA colonized pressure injury and 8.3% had MRSA bacteremia. Approximately 57% of the participants had received at least two classes of antibiotics in the preceding 30 days.¹⁴⁴

References

1. Chao R, Greer DM, McCorvey DL, Wright JK, Garza JR, *Management of Pressure Sores in Wound Care Practice*, S. Sheffield, Fife, Editor. 2004, Best Publishing Co: Flagstaff, AZ. p. 335
2. Konya C, Sanada H, Sugama J, Okuwa M, Kitagawa A. Does the use of a cleanser on skin surrounding pressure ulcers in older people promote healing? *J Wound Care*, 2005; 14(4): 169-171.
3. Manzur A, Gavaldà L, Ruiz de Gopegui E, Mariscal D, Dominguez MA, Perez JL, Segura F, Pujol M. Prevalence of methicillin-resistant Staphylococcus aureus and factors associated with colonization among residents in community long-term-care facilities in Spain. *Clin Microbiol Infect*, 2008; 14(9): 867-872.
4. Stotts NA, Hunt TK. Pressure ulcers. Managing bacterial colonization and infection. *Clinics in Geriatric Medicine*, 1997; 13(3): 565-573.
5. Sapico FL, Ginunas VJ, Thornhill Joynes M, Canawati HN, Capen DA, Klein NE, Khawam S, Montgomerie JZ. Quantitative microbiology of pressure sores in different stages of healing. *Diagnostic Microbiology and Infectious Disease* 1986; 5(1): 31-8.
6. Tarnuzzer RW, Schultz GS. Biochemical analysis of acute and chronic wound environments. *Wound Repair Regen*, 1996; 4(3): 321-325.

7. Haesler E, Ousey K. Evolution of the wound infection continuum. *Int Wound J*, 2018; 9(4): 6-10.
8. Haesler E, Swanson T, Ousey K, Carville K. Clinical indicators of wound infection and biofilm: Reaching international consensus. *J Wound Care*, 2019; 28(Supp3b): s4-s12.
9. International Wound Infection Institute (IWII), *Wound Infection in Clinical Practice*. 2016, Wounds International.
10. Keast DH, Parslow N, Houghton PE, Norton L, Fraser C. Best practice recommendations for the prevention and treatment of pressure ulcers: update 2006. *Advances in Skin & Wound Care*, 2007; 20(8): 447-60.
11. Heym B, Rimareix F, Lortat-Jacob A, Nicolas-Chanoine M. Bacteriological investigation of infected pressure ulcers in spinal cord-injured patients and impact on antibiotic therapy. *Spinal Cord*, 2004; 42(4): 230-234.
12. Braga IA, Brito CS, Filho AD, Filho PP, Ribas RM. Pressure ulcer as a reservoir of multiresistant Gram-negative bacilli: risk factors for colonization and development of bacteremia. *Braz J Infect Dis*, 2017; 21(2): 171-175.
13. Tedeschi S, Negosanti L, Sgarzani R, Trapani F, Pignanelli S, Battilana M, Capirossi R, Brillanti Ventura D, Giannella M, Bartoletti M, Tumietto F, Cristini F, Viale P. Superficial swab versus deep-tissue biopsy for the microbiological diagnosis of local infection in advanced-stage pressure ulcers of spinal-cord-injured patients: A prospective study. *Clin Microbiol Infect*, 2017; 08.
14. Edwards R, Harding KG. Bacteria and wound healing. *Curr Opin Infect Dis*, 2004; 17(2): 91-6.
15. Lipsky BA, Hoey C. Topical antimicrobial therapy for treating chronic wounds. *Clin Infect Dis*, 2009; 49(10): 1541-9.
16. World Union of Wound Healing Societies (WUWHS), *Principles of best practice: Wound infection in clinical practice. An international consensus*. 2008, MEP Ltd: London. Available from www.mepltd.co.uk.
17. Metcalf D, Bowler P. Biofilm delays wound healing: A review of the evidence. *Burns & Trauma*, 2013; 1(1): 5-12.
18. Wolcott RD, Kennedy J, Dowd SE. Regular debridement is the main tool for maintaining a healthy wound bed in most chronic. *J Wound Care*, 2009; 18(2): 54-56.
19. Wolcott RD, Rumbaugh KP, James G, Schultz GS, Phillips P, Yang Q, Watters C, Stewart PS, Dowd SE. Biofilm maturity studies indicate sharp debridement opens a time- dependent therapeutic window. *J Wound Care*, 2010; 19(8): 320-328.
20. Percival SL, Hill KE, Williams DW, Hooper SJ, Thomas DW, Costerton JW. A review of the scientific evidence for biofilms in wounds. *Wound Repair Regen*, 2012; 20: 647-657.
21. Wu YK, Cheng NC, Cheng CM. Biofilms in chronic wounds: Pathogenesis and diagnosis. *Cell Press Reviews*, 2019; 37(5): 505-517.
22. Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clinical Microbiology Reviews*, 2002; 15(2): 167-193.
23. James GA, Swogger E, Wolcott R, Pulcini Ed, Secor P, Sestrich J, Costerton JW, Stewart PS. Biofilms in chronic wounds. *Wound Repair and Regeneration*, 2008; 16(1): 37-44.
24. Han A, Zenilman J, Melendez J, Shirliff M, Agostinho A, James G, Stewart PS, Mongodin E, Rao D, Rickard A, Lazarus G. The importance of a multifaceted approach to characterizing the microbial flora of chronic wounds. *Wound Repair and Regeneration*, 2011; 19: 532-541.
25. Blanco-Blanco J, Gea-Sanchez M, Valenzuela-Pascual F, Barallat-Gimeno E, Espart A, Escobar-Bravo MA. Are the classic signs of infection in concordance with results from percutaneous aspiration to diagnose infection in pressure injuries? *J Adv Nurs*, 2017; 73(6): 1433-1442.
26. Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized chronic wound infection. *Wound Repair Regen*, 2001; 9(3): 178-186.
27. Mouton CP, Bazaldua OV, Pierce B, Espino DV. Common infections in older adults. *Am Fam Physician*, 2001; 63(2): 257-268.
28. European Wound Management Association, *Position Document: Identifying Criteria for Wound Infection*. 2005: EWMA.
29. Healy B, Freedman A. Infections. *BMJ (Clinical research ed)*, 2006; 332(7545): 838-841.
30. Harding K, Carville K, Cuddigan J, Fletcher J, Fuchs P, Harding K, Ishikawa O, Keast D, Leaper D, Lindholm C, Moodley P, Ricci E, Schultz G, Vasquez J. Wound infection in clinical practice: Shaping the future. An International Consensus Document. *International Wound Journal* 2008; 5(Supplement 3): 1-11.
31. Cutting KF. Identification of infection in granulating wounds by registered nurses. *J Clin Nurs*, 1998; 7(6): 539-546.
32. Cutting KF, White RJ, Mahoney P, Harding KG. *Clinical identification of wound infection: A Delphi approach, in European Wound Management Identifying criteria for wound infection. EWMA Position Document*. 2005, London: MEP Ltd. p. 6-9.
33. Harding K, Carville K, Cuddigan J, Fletcher J, Fuchs P, Harding K, Ishikawa O, Keast D, Leaper D, Lindholm C, Moodley P, Ricci E, Schultz G, Vasquez J. Wound infection in clinical practice: Shaping the future. An International Consensus Document. *Int Wound J*, 2008; 5(Supplement 3): 1-11.
34. Angel DE, Lloyd P, Carville K, Santamaria N. The clinical efficacy of two semi-quantitative wound-swabbing techniques in identifying the causative organism(s) in infected cutaneous wounds. *Int Wound J*, 2011; 8(2): 176-185.
35. Gardner SE, Frantz RA, Saltzman CL, Hillis SL, Park H, Scherubel M. Diagnostic validity of three swab techniques for identifying chronic wound infection. *Wound Repair Regen*, 2006; 14(5): 548-57.
36. Wilson SM, Antony B. Preparation of plant cells for transmission electron microscopy to optimize immunogold labeling of carbohydrate and protein epitopes, Table 1: Advantages and limitations of different microscopy techniques. *Nat Protoc*, 2012; 7: 1716-27.
37. Davidson MW. *Microscopy U*. 2016; Available from: <http://www.microscopyu.com/>.
38. Bell DC, Thomas WK, Murtagh KM, Dionne CA, Graham AC, Anderson JE, Glover WR. DNA base identification by electron microscopy. *Microsc Microanal*, 2012; 18(5): 1049-53.
39. Almeida C, Azevedo NF, Santos S, Keevil CW, Vieira MJ. Discriminating multi-species populations in biofilms with peptide nucleic acid fluorescence in situ hybridization (PNA FISH). *PLoS One*, 2011; 6(3): e14786.
40. Rudensky B, Lipschits M, Isaacsohn M, Sonnenblick M. Infected pressure sores: comparison of methods for bacterial identification. *South Med J*, 1992; 85(9): 901-3.
41. Sapico FL, Ginunas VJ, Thornhill Joynes M, Canawati HN, Capen DA, Klein NE, Khawam S, Montgomerie JZ. Quantitative microbiology of pressure sores in different stages of healing. *Diagn Microbiol Infect Dis*, 1986; 5(1): 31-8.

42. Bill TJ, Ratliff CR, Donovan AM, Knox LK, Morgan RF, Rodeheaver GT. Quantitative swab culture versus tissue biopsy: A comparison in chronic wounds. *Ostomy Wound Manage*, 2001; 47(1): 34-37.
43. Copeland-Halperin LR, Kaminsky AJ, Bluefeld N, Miraliakbari R. Sample procurement for cultures of infected wounds: A systematic review. *J Wound Care*, 2016; 25(4): S4-S10.
44. Hurlow J, Bowler P. Potential implications of biofilm in chronic wounds: a case series. *J Wound Care*, 2012; 21(3): 116-119.
45. Han A, Zenilman J, Melendez J, Shirliff M, Agostinho A, James G, Stewart PS, Mongodin E, Rao D, Rickard A, Lazarus G. The importance of a multifaceted approach to characterizing the microbial flora of chronic wounds. *Wound Repair Regen*, 2011; 19: 532-541.
46. James GA, Swogger E, Wolcott R, Pulcini Ed, Secor P, Sestrich J, Costerton JW, Stewart PS. Biofilms in chronic wounds. *Wound Repair Regen*, 2008; 16(1): 37-44.
47. Nakagami G, Schultz G, Gibson DJ, Phillips P, Kitamura A, Minematsu T, Miyagaki T, Hayashi A, Sasaki S, Sugama J, Sanada H. Biofilm detection by wound blotting can predict slough development in pressure ulcers: A prospective observational study. *Wound Repair Regen*, 2017; 25(1): 131-138.
48. Serena T, Harrell K, Serena L, Yaakov R. Real-time bacterial fluorescence imaging accurately identifies wounds with moderate-to-heavy bacterial burden. *J Wound Care*, 2019; 28(6): 346-347.
49. Grassetti L, Scalise A, Lazzeri D, Carle F, Agostini T, Gesuita R, Di Benedetto G. Perforator flaps in late-stage pressure sore treatment: outcome analysis of 11-year-long experience with 143 patients. *Ann Plast Surg*, 2014; 73(6): 679-85.
50. Huang K, Guo Q. Surgical repair involving tissue flap transplantation with vascular pedicle in treating refractory pressure ulcers around hip and sacral region. *Curr Signal Transduct Ther*, 2015; 10(1): 36-40.
51. Tadiparthi S, Hartley A, Alzweri L, Mecci M, Siddiqui H. Improving outcomes following reconstruction of pressure sores in spinal injury patients: A multidisciplinary approach. *J Plast Reconstr Aesthet Surg*, 2016; 69(7): 994-1002.
52. Wettstein R, Tremp M, Baumberger M, Schaefer DJ, Kalbermatten DF. Local flap therapy for the treatment of pressure sore wounds. *Int Wound J*, 2013.
53. Bertheuil N, Huguier V, Aillet S, Beuzeboc M, Watier E. Biceps femoris flap for closure of ischial pressure ulcers. *Eur J Plast Surg*, 2013; 36(10): 639-644.
54. Greco M, Marchetti F, Tempesta M, Ruggiero M, Marcasciano M, Carlesimo B. Cutaneous flaps in the treatment of 338 pressure sores: a better choice. *Ann Ital Chir*, 2013; 84(6): 655-9.
55. Larson DL, Hudak KA, Waring WP, Orr MR, Simonelic K. Protocol management of late-stage pressure ulcers: A 5-year retrospective study of 101 consecutive patients with 179 ulcers. *Plast Reconstr Surg*, 2012; 129(4): 897-904.
56. Marriott R, Rubayi S. Successful truncated osteomyelitis treatment for chronic osteomyelitis secondary to pressure ulcers in spinal cord injury patients. *Ann Plast Surg*, 2008; 61(4): 425-429.
57. Brunel AS, Lamy B, Cyteval C, Perrochia H, Teot L, Masson R, Bertet H, Bourdon A, Morquin D, Reynes J, Le Moing V, Almeras I, Thanh AD, Goelis A, Reynaud C, Rouays H, Trial C, Fluieraru S, Herlin C, Lavigne JP, Delfour C, Baron MP, Viala P, Mura T, Goeraud P. Diagnosing pelvic osteomyelitis beneath pressure ulcers in spinal cord injured patients: A prospective study. *Clin Microbiol Infect*, 2016; 22(3): 267.e1-267.e8.
58. De Heredia L, Hauptfleisch J, Hughes R, Graham A, Meagher T. Magnetic resonance imaging of pressure sores in spinal cord injured patients: Accuracy in predicting osteomyelitis. *Topics in Spinal Cord Injury Rehabilitation*, 2012; 18(2): 146-148.
59. Hauptfleisch J, Meagher TM, Hughes RJ, Singh JP, Graham A, Lopez de Heredia L. Interobserver Agreement of Magnetic Resonance Imaging Signs of Osteomyelitis in Pelvic Pressure Ulcers in Patients With Spinal Cord Injury. *Arch Phys Med Rehabil*, 2013.
60. Luis, Hauptfleisch J, Hughes R, Graham A, Meagher TMM. Magnetic Resonance Imaging of Pressure Sores in Spinal Cord Injured Patients: Accuracy in Predicting Osteomyelitis. *Topics in Spinal Cord Injury Rehabilitation*, 2012; 18(2): 146-148.
61. Lopez de Heredia L, Hauptfleisch J, Hughes R, Graham A, Meagher T. Magnetic resonance imaging of pressure sores in spinal cord injured patients: Accuracy in predicting osteomyelitis. *Topics in Spinal Cord Injury Rehabilitation*, 2012; 18(2): 146-148.
62. Hatzenbuehler J, Pulling T. Diagnosis and Management of Osteomyelitis. *Am Fam Physician*, 2011; 84(9): 1027-1033.
63. American Society of Plastic Surgeons. *Evidence-Based Clinical Practice Guideline: Chronic Wounds of the Lower Extremity*. 2011 [cited August 2019].
64. Daniali LN, Keys K, Katz D, Mathes DW. Effect of preoperative magnetic resonance imaging diagnosis of osteomyelitis on the surgical management and outcomes of pressure ulcers. *Ann Plast Surg*, 2011; 67(5): 520-525.
65. Sugarman B, Hawes S, Musher DM, Klima M, Young EJ, Pircher F. Osteomyelitis beneath pressure sores. *Archives of Internal Medicine*, 1983; 143(4): 683-8.
66. Thornhill Joynes M, Gonzales F, Stewart CA, Kanel GC, Lee GC, Capen DA, Sapico FL, Canawati HN, Montgomerie JZ. Osteomyelitis associated with pressure ulcers. *Archives of Physical Medicine and Rehabilitation*, 1986; 67(5): 314-8.
67. Darouiche RO, Landon GC, Klima M, Musher DM, Markowski J. Osteomyelitis associated with pressure sores [see comments]. *Archives of Internal Medicine* 1994; 154(7): 753-8.
68. Heiba SI, Stempler L, Sullivan T, Kolker D, Kostakoglu L. The ideal dual-isotope imaging combination in evaluating patients with suspected infection of pelvic pressure ulcers. *Nucl Med Commun*, 2017; 38(2): 129-134.
69. Wounds Australia, Standards for Wound Prevention and Management. 2016, Cambridge Media: Osborne Park, WA.
70. Deshmukh GR, Barkel DC, Sevo D, Hergenroeder P. Use or misuse of colostomy to heal pressure ulcers. *Dis Colon Rectum*, 1996; 39(7): 737-738.
71. Saltzstein RJ, Romano J. The efficacy of colostomy as a bowel management alternative in selected spinal cord injury patients. *J Am Paraplegia Soc*, 1990; 13(2): 9-13.
72. Bateman SD, Roberts S. Moisture lesions and associated pressure ulcers: Getting the dressing regimen right. *Wounds UK*, 2013; 9(2): 97-102.
73. Park KH, Kim KS. Effect of a structured skin care regimen on patients with fecal incontinence: A comparison cohort study. *J Wound Ostomy Continence Nurs*, 2014; 41(2): 161-167.
74. Wild T, Bruckner M, Payrich M, Schwarz C, Eberlein T, Andriessen A. Eradication of methicillin-resistant *Staphylococcus aureus* in

- pressure ulcers comparing a polyhexanide-containing cellulose dressing with polyhexanide swabs in a prospective randomized study. *Adv Skin Wound Care*, 2012; 25(1): 17-22.
75. Sipponen A, Jokinen JJ, Sipponen P, Papp A, Sarna S, Lohi J. Beneficial effect of resin salve in treatment of severe pressure ulcers: A prospective, randomized and controlled multicentre trial. *Br J Dermatol*, 2008; 158(5): 1055-1062.
 76. Dryden M, Dickinson A, Brooks J, Hudgell L, Saeed K, Cutting KF. A multi-centre clinical evaluation of reactive oxygen topical wound gel in 114 wounds. *J Wound Care*, 2016; 25(3): 140, 142-6.
 77. Biglari B, Linden PH, Simon A, Aytac S, Gerner HJ, Moghaddam A. Use of Medihoney as a non-surgical therapy for chronic pressure ulcers in patients with spinal cord injury. *Spinal Cord*, 2012; 50(2): 165-169.
 78. Ciliberti M, De Lara F, Serra G, Tafuro F, Iazzetta FM, De Martino V, et al. Effective management of pressure ulcers using Hydrofibre technology with silver ions. *Wound Medicine*, 2014; 5: 40-44.
 79. Yapuca Günes U, E er I. Effectiveness of a honey dressing for healing pressure ulcers. *J Wound Ostomy Continence Nurs*, 2007; 34(2): 184-190.
 80. Kaya AZ, Turani N, Akyüz M. The effectiveness of a hydrogel dressing compared with standard management of pressure ulcers. *J Wound Care*, 2005; 14(1): 42-4.
 81. Kucan JO, Robson MC, J.P. H, Ko F. Comparison of silver sulfadiazine, povidone-iodine and physiologic saline in the treatment of chronic pressure ulcers. *J Am Geriatr Soc*, 1981; 29(5): 232-235.
 82. Moberg S, Hoffman L, Grennert ML, Holst A. A randomized trial of cadexomer iodine in decubitus ulcers. *J Am Geriatr Soc*, 1983; 31(8): 462-465.
 83. Nisi G, Brandi C, Grimaldi L, Calabrò M, D'Aniello C. Use of a protease-modulating matrix in the treatment of pressure sores. *Chir Ital*, 2005; 57(4): 465-468.
 84. Lineaweaver W, Howard R, Soucy D, McMorris S, Freeman J, Crain C, Robertson J, et al. Topical antimicrobial toxicity. *Arch Surg*, 1985; 120(3): 267-270.
 85. Wilson J, Mills J, Prather I, Dimitrijevic S. A toxicity index of skin and wound cleansers used on in vitro fibroblasts and keratinocytes. *Adv Skin Wound Care*, 2005; 18(7): 373-378.
 86. Heggors J, Sazy J, Stenberg B, Strock L, McCauley R, Hernom D, Robson M. Bacterial and wound healing properties of sodium hypochlorite solutions: The 1991 Lindberg Award. *Journal of Burn Care & Research*, 1991; 12(5): 420-424.
 87. Edwards-Jones V, Flanagan M, Wolcott R. Technological advancements in the fight against antimicrobial resistance. *Wounds Int*, 2015; 6(2): 47-51.
 88. Leaper DJ, Schultz G, Carville K, Fletcher J, Swanson T, Drake R. Extending the TIME concept: what have we learned in the past 10 years? *Int Wound J*, 2012; 9(Suppl 2): 1-19.
 89. Gruber R, Vistnes L, Pardoe R. The effect of commonly used antiseptics on wound healing. *Plast Reconstr Surg*, 1975; 55(4): 472-6.
 90. Reid C, Alcock M, Penn D. Hydrogen peroxide - a party trick from the past? *Anaesthesia and Intensive Care Journal*, 2011; 39: 1004-8.
 91. Hussain-Khan Z, Soleimani A, Farzan M. Fatal gas embolism following the use of intraoperative hydrogen peroxide as an irrigation fluid. *Acta Med Iran*, 2004; 42(2): 151-153.
 92. Echague C, Hair P, Cunnion K. A comparison of antibacterial activity against Methicillin-Resistant *Staphylococcus aureus* and gram-negative organisms for antimicrobial compounds in a unique composite wound dressing. *Adv Skin Wound Care*, 2010; 23(9): 406-13.
 93. Leaper DJ, Durani P. Topical antimicrobial therapy of chronic wounds healing by secondary intention using iodine products. *Int Wound J*, 2008; 5(2): 361-8.
 94. Sibbald RG, Leaper DJ, Queen D. Iodine made easy. *Wounds International*, 2011; 2(2): S1-6.
 95. Ward R, Saffle J. Topical agents in burn and wound care. *Phys Ther*, 1995; 75: 526-538.
 96. Brennan S, Leaper D. The effect of antiseptics on the healing wound: a study using the rabbit ear chamber. *Br J Surg*, 1985; 72(10): 780-2.
 97. Johnson AR, White A, McAnalley B. Comparison of common topical agents for wound treatment: cytotoxicity for human fibroblasts in culture. *Wounds: A Compendium of Clinical Research & Practice*, 1989; 1(3): 186-92.
 98. World Union of Wound Healing Societies, Principles of Best Practice: Wound Infection in Clinical Practice: An International Consensus. 2008, MEP Ltd: London.
 99. Cooper RA, Bjarnsholt T, Alhede M. Biofilms in wounds: a review of present knowledge. *J Wound Care*, 2014; 23(11): 570-80.
 100. Cooper R, Jenkins L, Hooper S. Inhibition of biofilms of *Pseudomonas aeruginosa* by Medihoney in vitro. *J Wound Care*, 2014; 23(3): 93-104.
 101. Wound Healing and Management Group. Evidence summary: Wound infection: Iodophors and biofilms. *Wound Practice and Research*, 2013; 21(2): 86-87.
 102. Suman E, Madhavi R, Shashidhar Kotian M. Role of bacterial biofilms in chronic non-healing ulcers and effect of subinhibitory concentrations of betadine and hydrogen peroxide on biofilms. *J Hosp Infect*, 2009; 73: 87-9.
 103. Hill K, E., Malic S, McKee R, Rennison T, Harding K, et al., Williams D. An in vitro model of chronic wound biofilms to test wound dressings and assess antimicrobial susceptibilities. *J Antimicrob Chemother*, 2010; 65: 1195-206.
 104. Roberts A, Maddocks SE, Cooper RA. Manuka honey is bactericidal against *Pseudomonas aeruginosa* and results in differential expression of *oprF* and *algD*. *Microbial Pathogenicity*, 2012; 158: 3005-13.
 105. Majtan J, Bohova J, Horniackova M, Klaudiny J, Majtan V. Anti-biofilm effects of honey against wound pathogens *Proteus mirabilis* and *Enterobacter cloacae*. *Phytother Res*, 2014; 28(1): 69-75
 106. Lu J, Turnbull L, Burke CM, Liu M, Carter DA, Schlothauer RC, Whitchurch CB, Harry EJ. Manuka-type honeys can eradicate biofilms produced by *Staphylococcus aureus* strains with different biofilm-forming abilities. *PeerJ*, 2014; 2: e326.
 107. Wang R, Starkey M, Hazan R, Rahme LG. Honey's ability to counter bacterial infections arises from both bactericidal compounds and QS inhibition. *Front Microbiol*, 2012; <http://journal.frontiersin.org/article/10.3389/fmicb.2012.00144/full>.
 108. International consensus, Appropriate use of silver dressings in wounds. An expert working group consensus. Download from: www.woundhealing.com.

- woundsinternational.com. 2012, Wounds International, London.
109. Bowler P, Parsons D. Combatting wound biofilm and recalcitrance with novel anti-biofilm Hydrofiber wound dressing. *Wound Medicine*, 2016; 14: 6-11.
 110. Parsons D, *Designing a dressing to address local barriers to wound healing*, in *Next-generation antimicrobial dressings: AQUACEL™ Ag+ Extra and Ribbon*. 2014, Wounds International 2014: London.
 111. Metcalf D, Parsons D, Bowler P. A next-generation antimicrobial wound dressing: A real-life clinical evaluation in the UK and Ireland *J Wound Care*, 2016; 25(3): 132-138.
 112. AQUACEL®, Ag+ Extra Dressing. Instructions for use. 2016: ConvaTec Limited.
 113. Yang Q, Larose C, Della Porta AC, Schultz GS, Gibson DJ. A surfactant based wound dressing can reduce bacterial biofilms in a porcine skin explant model. *Int Wound J*, 2017; 14(2): 408-413.
 114. Norman G, Dumville J, Moore ZEH, Tanner J, Christie J, Goto S. Antibiotics and antiseptics for pressure ulcers. *Cochrane Database Syst Rev*, 2016; Issue 4(Art. No.: CD011586).
 115. Burks RI. Povidone-iodine solution in wound treatment. *Phys Ther*, 1998; 78(2): 212-8.
 116. Fumal I, Braham C, Paquet P, Pierard-Franchimont C, Pierard GE. The beneficial toxicity paradox of antimicrobials in leg ulcer healing impaired by a polymicrobial flora: A proof-of-concept study. *Dermatol Nurs*, 2002; 204(Supp 1): 70-74.
 117. Gethin G, Cowman S. Bacteriological changes in sloughy venous leg ulcers treated with manuka honey or hydrogel: an RCT. *J Wound Care*, 2008; 17(6): 241.
 118. Efem SE. Recent advances in the management of Fournier's gangrene: preliminary observations. *Surgery*, 1993; 113(2): 200-204.
 119. Molan PC. Re-introducing honey in the management of wounds and ulcers - theory and practice. *Ostomy Wound Management*, 2002; 48(11): 28-40.
 120. Günes UY, Eer I. Effectiveness of a honey dressing for healing pressure ulcers. *Journal of Wound, Ostomy and Continence Nursing*, 2007; 34(2): 184-190.
 121. Jull AB, Walker N, Deshpande S. Honey as a topical treatment for wounds. *Cochrane Database Syst Rev*, 2013; 2: CD005083.
 122. Weheida SM, Naguib HH, El-Banna HM, Marzouk S. Comparing the effects of two dressing techniques on healing of low grade pressure ulcers. *Journal of the Medical Research Institute*, 1991; 12(2): 259-78.
 123. Toy L, Macera L. Evidence-based review of silver dressing use on chronic wounds. *J Am Acad Nurse Pract*, 2011; 23: 183-92.
 124. Eberlein T, Haemmerle G, Signer M, Gruber-Moesenbacher U, Traber J, Mittlboeck M, al. e. Comparison of PHMB-containing dressing and silver dressings in patients with critically colonised or locally infected wounds. *J Wound Care*, 2012; 21(1): 12-20.
 125. Oduwole KO, McCormack DJ, Glynn AA, O'Gara P, Molony DC, Murray D, al. e. Anti-biofilm activity of sub-inhibitory povidone-iodine concentrations against *Staphylococcus epidermidis* and *Staphylococcus aureus*. *J Orthop Res*, 2010; 28: 1252-6.
 126. Presterl E, Suhomel M, Eder M, Reichmann S, Lassnigg A, Graninger W, al. e. Effects of alcohols, povidone-iodine and hydrogen peroxide on biofilms of *Staphylococcus epidermidis*. *J Antimicrob Chemother*, 2007; 60: 417-20.
 127. Brett D. Testing of cadexomer iodine against a variety of micro-organisms grown in single and mixed species biofilms. *J Wound Ostomy Continence Nurs*, 2011; 38(35): S2-S115
 128. Bjarnsholt T, Kirketerp-Møller K, Kristiansen S, Phipps R, Nielsen A, Jensen PK, al. e. Silver against *Pseudomonas aeruginosa* biofilms. *Acta Pathologica, Microbiologica, et Immunologica Scandinavica*, 2007; 115: 921-8.
 129. Akiyama H, Yamasaki O, Kanzaki H, Tada J, Arata J. Effects of sucrose and silver on *Staphylococcus aureus* biofilms. *J Antimicrob Chemother*, 1998; 42: 629-34.
 130. Thorn R, Austin A, Greenman J, Wilkins J, Davis P. In vitro comparison of antimicrobial activity of iodine and silver dressings against biofilms. *J Wound Care*, 2009; 18: 343-6.
 131. Wolcott RD, Rumbaugh KP, James G, Schultz GS, Phillips P, Yang Q, Watters C, Stewart PS, Dowd SE. Biofilm maturity studies indicate sharp debridement opens a time- dependent therapeutic window. *J Wound Care*, 2010; 19(8): 320-328.
 132. Leaper DJ, Schultz GS, Carville K, Fletcher J, Swanson T, Drake R. Extending the TIME concept: What have we learned in the past 10 years? *Int Wound J*, 2012; 9(Suppl 2): 1-19.
 133. Wolcott RD. Disrupting the biofilm matrix improves wound healing outcomes. *J Wound Care*, 2015; 24(8): 366-71.
 134. Bianchi T, Wolcott RD, Peghetti A, Leaper D, Cutting K, Polignano R, Rosa Rita Z, Moscatelli A, Greco A, Romanelli M, Pancani S, Bellingeri A, Ruggeri A, Postacchini L, Tedesco S, Manfredi L, Camerlingo M, Rowan S, Gabrielli A, Pomponio G. Recommendations for the management of biofilm: a consensus document. *J Wound Care*, 2016; 25(6): 305-317.
 135. Wound Healing Society, *Chronic wound care guidelines. Abridged version*. 2007, Maitland, FL: The Wound Healing Society.
 136. Bryan CS, Dew CE, Reynolds KL. Bacteremia associated with decubitus ulcers. *Arch Intern Med*, 1983; 143(11): 2093-5.
 137. Wall BM, Mangold T, Huch KM, Corbett C, Cooke CR. Bacteremia in the chronic spinal cord injury population: risk factors for mortality. *J Spinal Cord Med*, 2003; 26(3): 248.
 138. Cafferkey MT, Hone R, Keane CT. Sources and outcome for methicillin-resistant *Staphylococcus aureus* bacteraemia. *J Hosp Infect*, 1988; 11(2): 136-143.
 139. Redelings MD, Lee NE, Sorvillo F. Pressure ulcers: more lethal than we thought? *Adv Skin Wound Care*, 2005; 18(7): 367-372.
 140. European Pressure Ulcer Advisory Panel, *Pressure Ulcer Treatment Guidelines*. 1998, EPUAP; Oxford, England.
 141. European Wound Management Association. *Position Document: Management of Wound Infection*. 2006.
 142. Buck DW, Goucher JH, Lewis JVL. The Incidence of Methicillin-Resistant *Staphylococcus aureus* in Pressure Ulcers. *Adv Skin Wound Care*, 2012; 25(11): 509-12.
 143. Cataldo MC, Bonura C, Caputo G, Aleo A, Rizzo G, Geraci DM, Calá C, Fasciana T, Mattaliano AR, Mammina C. Colonization of pressure ulcers by multidrug-resistant microorganisms in patients receiving home care. *Scand J Infect Dis*, 2011; 43(11-12): 947-952.
 144. Nery Silva Pirett CC, Braga IA, Ribas RM, Gontijo Filho PP, Filho AD. Pressure ulcers colonized by MRSA as a reservoir and risk for MRSA bacteremia in patients at a Brazilian university hospital. *Wounds*, 2012; 24(3): 67-75.

WOUND DRESSINGS FOR TREATMENT OF PRESSURE INJURIES

Introduction

Wound dressings are a central component of pressure injury care. Since the 1960s, it has been accepted that wound healing is optimized when the wound is kept in a moist environment rather than air-dried or dried with heat lamps or topically applied drying agents.¹ Occlusive or semi-occlusive wound dressings that maintain wound bed moisture promote re-epithelialization and wound closure.

The role of dressings in protecting skin at high risk of pressure injuries from shear has received an increased focus in clinical practice and research. Recommendations on the use of prophylactic dressings are outlined in the chapter *Preventive Skin Care*. Recommendations on negative pressure wound therapy (NPWT) are found in the *Biophysical Agents* chapter of the guideline.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What wound dressings are effective for supporting healing of partial thickness pressure injuries?
- What wound dressings are effective for supporting healing of full thickness pressure injuries?
- What wound dressings are effective for pressure injuries with higher levels of exudate?
- Which wound dressings are the most cost-effective for healing pressure injuries?

Selecting a Wound Dressing

14.1: For all pressure injuries, select the most appropriate wound dressing based on goals and self-care abilities of the individual and/or their informal caregiver and based on clinical assessment, including:

- Diameter, shape and depth of the pressure injury
- Need to address bacterial bioburden
- Ability to keep the wound bed moist
- Nature and volume of wound exudate
- Condition of the tissue in the wound bed
- Condition of the peri-wound skin
- Presence of tunneling and/or undermining
- Pain

(Good Practice Statement)

Implementation Considerations

- For older adults, select wound dressings with consideration to the potential impact on fragile skin, especially related to skin trauma on dressing removal² (*Level 1*).
- Assess pressure injuries every time the wound dressing is changed and confirm the effectiveness and appropriateness of the dressing regimen (*Expert opinion*).
- Follow the manufacturer's recommendations when using a wound dressing, especially related to frequency of dressing change (*Expert opinion*).
- The plan of care should guide usual wear times for wound dressings and contain provisional plans for wound dressing changes as needed (*Expert opinion*).
- Replace wound dressings if they are leaking or visibly soiled (*Expert opinion*).
- Ensure all wound dressing products are completely removed and replaced with each dressing change (*Expert opinion*).
- For individuals in the community, ensure there is an adequate wound dressing supply to accommodate unplanned wound dressing changes due to soiling or leakage (*Expert opinion*).

Discussion

The goals of wound care are important to consider in selecting a wound dressing, because different wound dressings have different methods of action. Selecting the right wound dressing for a specific pressure injury requires a comprehensive evaluation of the pressure injury, the individual and the environment each time the wound dressing is changed. Wound dressings for pressure injuries are designed to:

- Improve wound healing time
- Absorb blood and tissue exudate
- Minimize pain, including pain associated with application and removal
- Minimize shear strain
- Protect the wound and surrounding skin and tissue
- Absorb and control malodor
- Reduce injury to peri-wound skin
- Promote autolytic debridement (if this is a care goal)
- Address bioburden (see the guideline chapter *Infection and Biofilm*).

Selection of a wound dressing should be individualized and based on the condition of the wound and the individual's goals of care, self-care and preferences. For example, consideration be given to:

- The care setting (e.g., how long before the wound dressing will be changed?)
- The location of the pressure injury (e.g., will the dressing be contaminated frequently?)
- The individual's mobility (e.g., will the dressing stay in place if the individual is mobile?)
- Skill required to change the wound dressing (e.g., who will change the dressing, and do they possess the necessary knowledge and skills?)
- The individual's preferences (e.g., will the wound dressing be visible? Is the dressing comfortable?)
- Accessibility of different wound dressings (e.g., what is available?).

When the pressure injury is clean and granulating, maintenance of a moist wound bed is an important factor in promoting healing or closure and reducing wound-related pain. The peri-wound skin can be kept dry and free from maceration using a wound dressing that remains in contact with the wound bed and absorbs the anticipated amount of exudate, or by using a skin barrier product. As the pressure injury either heals or deteriorates over time, the type of wound dressing most appropriate for promotion of healing may change. For example, exudate usually decreases as the pressure injury heals, reducing the requirement for a wound dressing with high absorption.

Potential trauma to the skin and surrounding tissue is another consideration when selecting a wound dressing, particularly when the peri-wound skin is fragile.³ Wound dressings with strong adhesive properties present a risk to skin, particularly for individuals with vulnerable and fragile skin (e.g. older adults and premature neonates). This damage to the epidermis reduces the skin's barrier function and can lead to inflammation and infection.³ Removal of wound dressings with strong medical adhesive can cause detachment of the stratum corneum that is exacerbated with repeated wound dressing removals over time.^{3,4} Some wound dressings are designed specifically to be atraumatic to the skin. Wound dressings with a silicone interface are designed to provide a wound contact layer that can be removed without causing trauma to the tissues or pain to the individual. Silicone is chemically inert, and adverse effects from its use in wound care are rare. Wound dressings with a silicone interface can also protect friable or newly healed wound tissue. Meaume et al. (2003)⁵ conducted an RCT with 38 participants with Category/Stage II pressure injuries comparing an adherent foam dressing to a foam dressing with a silicone interface. The silicone foam dressing was found to be less traumatic to the peri-wound tissue (*Level 1*). Barrier films are also used to protect peri-wound skin,³ although there were no included studies evaluating their effectiveness in populations with pressure injuries.

Many of the wound dressing types discussed throughout this chapter are manufactured in combinations. Please refer to the statements about the individual components when considering the use of composite dressings.

14.2: Evaluate the cost effectiveness of wound dressings at a local level, with consideration to direct and indirect costs to the health care system and to the individual with a pressure injury. Advanced wound dressings that promote moist wound healing are more likely to be cost-effective due to faster healing times and less frequent dressing changes.
(Good Practice Statement)

Discussion

Evidence of cost effectiveness of wound dressing for healing pressure injuries is reported in low to moderate quality cost analyses and/or low quality clinical studies.⁶⁻⁹ Costs may have large variations between geographic locations. Direct costs of a wound dressing vary substantially depending on the geographic and clinical settings and the types of wound products used. Formal cost benefit analysis compares the financial value of health benefits to financial costs, requiring a value judgment of different benefits (e.g., cost of disability). To meaningfully inform local clinical practice and choice of individuals, comparative costs should accurately reflect the local setting.¹⁰

Variation in costs between different locations is demonstrated in the recent evidence. A moderate quality cost analysis conducted in an outpatient setting in Thailand in 2010 reported a mean cost of wound dressings (including staffing costs, wound debridement and products) for a Category/Stage III or IV pressure injury as \$16.13 for a silver dressing (USD in 2013).⁸ This compared to average costs of \$12.34 (standard deviation [SD] 11.24) (Category/Stage III pressure injury) and \$5.84 (SD 7.02) (Category/Stage IV pressure injury) reported by a low quality cost analysis set in a US teaching hospital (USD in 2016).⁷ Another moderate quality cost analyses reported a wound dressing performed in the intensive care setting in Brazil (mean cost \$11.90, SD 7.40, USD in 2015).⁹ Further, wound dressing costs vary according to the Category/Stage of the pressure injury. Lima et al. (1999)⁷ reported cost ranges from \$19.18 ± 11.80 for dressing a Category/Stage I pressure injury to \$5.84 (SD 7.02, USD in 1999) for dressing a Category/Stage IV pressure injury. However as noted, different settings, severity of wounds, ranges of products and analysis techniques prevent direct comparisons of cost. Cost effectiveness of different dressing options must therefore be evaluated with consideration to geographic and clinical setting, the severity of the pressure injury, the duration of the wound and the methodology used to explore cost effectiveness.

Low quality evidence suggests that advanced wound dressings that promote moist wound healing are associated with lower costs than gauze dressings.⁶ Lower costs relate to less frequent dressing changes and faster healing rates.⁶ As an example, this study showed that advanced moist wound dressings (e.g., foam, silver foam, silver sulfadiazine and ibuprofen-releasing foam dressings) required a mean 49.5 ± 29.6 dressing changes compared to a mean of 222.6 ± 101.9 (p < 0.0001) dressing changes for a gauze dressing, while achieving significantly faster healing (85.56 ± 52.1 days versus 121.4 ± 52.2 days, p = 0.0001).⁶ However, there were inconsistent findings regarding direct costs related to the severity of pressure injury,⁷ and costs are likely to have large variations between geographic locations.

Advanced Wound Dressings for Category/Stage I and II Pressure Injuries

14.3: Use hydrocolloid dressings for non-infected Category/Stage II pressure injuries as indicated by the clinical condition of the pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

14.4: Use hydrogel dressings for non-infected Category/Stage II pressure injuries as indicated by the clinical condition of the pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

14.5: Use polymeric membrane dressings for non-infected Category/Stage II pressure injuries as indicated by the clinical condition of the pressure injury.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence supports the use of different types of advanced wound dressings for Category/Stage II wound dressings based on the wound bed condition. Evidence from low quality Level 1 studies^{2,11,12} suggests that pressure injury healing rates when a hydrocolloid dressing is applied to Category/Stage II pressure injuries do not differ significantly from healing rates when other contemporary wound dressings are used. There is limited information on current costs for using a hydrocolloid dressing. Although there is evidence to suggest that hydrocolloid dressings are acceptable options for health professionals, with a favorable profile for ease of removal, low residue and conformability, individuals with pressure injuries tended to rate hydrocolloids lower than other wound dressings with respect to comfort during wear and during removal,² and higher rates of erythema have been reported.¹³

There is evidence from a low quality Level 1 study that Category/Stage II pressure injuries are more likely to heal with a hydrogel dressing compared to a standard moist gauze dressing, although healing rates may not be substantially faster.¹⁴ A second study had conflicting results.¹⁵ Inconsistencies in the findings may relate to the severity of the pressure injuries (one study¹⁵ included full thickness pressure injuries), differing regimens, or different active components in the dressings (one study¹⁵ used an aloe vera-based product). Ratings from people with pressure injuries suggested a hydrogel dressing is a comfortable wound dressing choice¹⁶ and wound dressing changes may be performed less frequently than with a moistened gauze dressing.¹⁴

There is evidence from very small, low quality Level 1 studies^{17,18} showing that a polymeric membrane dressing is associated with improvements in some measures of wound healing, including scores on the Pressure Ulcer Scale for Healing (PUSH) tool. The low level evidence indicated that a Category/Stage I pressure injury could heal within 19 days with a polymeric dressing, and a Category/Stage II pressure injury could heal within 61 days.¹⁸ Indirect evidence from a laboratory study provided support for the capacity of a polymeric membrane to absorb exudate.¹⁹ There was no evidence available on potential adverse effects or resource requirements.

Implementation Considerations

Hydrocolloid dressings

- Use hydrocolloid dressings on anatomical locations where the dressing will not roll or melt (*Expert opinion*).
- Use hydrocolloid dressings cautiously in older adults who are at higher risk for skin trauma due to aging skin. Consider using a barrier film product to protect the peri-wound skin (*Expert opinion*).
- Remove hydrocolloid dressings carefully from fragile skin to reduce skin trauma and discomfort during removal. Hydrocolloid wound dressings can deposit residue on the wound bed and/or peri-wound skin, the removal of which can cause trauma² (*Level I*).
- Hydrocolloid dressings require less frequent dressing changes¹² (*Level I*).
- Provide a “hydrocolloid window” to the peri-wound skin to prevent further trauma to the skin²⁰ (*Expert opinion*).

Hydrogel dressings

- Use amorphous hydrogel for pressure injuries that are not clinically infected and are granulating (*Expert opinion*).
- Consider using a hydrogel dressing for treatment of a dry wound bed when the goal of care is hydration or autolytic debridement²¹ (*Level 5*).
- When there are high levels of exudate present, a hydrogel dressing could increase the risk of maceration and therefore may not be the most appropriate dressing choice (*Expert opinion*).
- Consider use of hydrogel sheets dressings for pressure injuries without depth and contours and/or on body areas that are at risk for wound dressing migration (*Expert opinion*).
- Protect the peri-wound skin (e.g., with a barrier product) to prevent maceration²² (*Expert opinion*).

Evidence Discussion

Hydrocolloid dressings are a common treatment for Category/Stage II pressure injuries. The manufacturing of these dressings has advanced, with improvements in the adhesion and beveling of dressing edges, and development of different shapes designed for specific anatomical locations (e.g., heel, sacrum).

Graumlich et al. (2003)¹² conducted an eight-week single-blinded RCT in 65 participants with Category/Stage II or III pressure injuries, comparing collagen and hydrocolloid dressings. There was no difference in complete healing between the two groups. However, there was no stratification based on initial ulcer size (*Level 1*).

Clinical utility of hydrocolloid dressings has been reported, including indications as to their conformability, adhesion, and ease of removal. While Bale et al. (1997)¹¹ compared hydrocolloid to foam dressings and concluded that there was no difference in mean wear time (*Level 1*). Graumlich et al. (2003)¹² found that a hydrocolloid dressing had longer wear times compared to a collagen dressing (*Level 1*). Brown-Etris et al. (2008)² compared a hydrocolloid to a film dressing containing an absorptive pad and concluded that the hydrocolloid dressing was less easily placed and removed, and less conformable and comfortable during wearing (*Level 1*). However, these studies are dated, and the design and manufacture of hydrocolloid dressings has advanced substantially.

As noted above, medical adhesives such as those used on self-adhesive hydrocolloid dressings have been associated with skin injury, particularly in individuals with vulnerable skin. Strategies to reduce medical adhesive related skin injury should be implemented when using hydrocolloid dressings. For example, when wound dressings were taped to a “hydrocolloid window” around wounds rather than directly to the skin, Milne et al. (1999)²⁰ reported less damage to peri-wound skin. A hydrocolloid window is created by applying strips of hydrocolloid dressing around the wound

and affixing the primary wound dressing to the “hydrocolloid window”. The hydrocolloid strips can remain in place for up to five days while the primary dressing is replaced as required. This reduces skin injury from repeated wound dressing removal. The guideline chapter *Cleansing and Debridement* also includes discussion on protecting the skin surrounding the pressure injury.

Hydrogel dressings contain hydrated hydrophilic polymers, which produce a moist environment that promotes wound healing. The two most common types of hydrogels are amorphous hydrogels and sheet hydrogels. Amorphous gels are clinically preferred for pressure injuries where the wound dressing is likely to be displaced (e.g., on gravity-dependent body areas such as the lower leg). Hydrogel sheets are clinically preferred for pressure injuries on non-moving and non-dependent body surfaces. The increase in moisture in the wound bed facilitates autolytic debridement.

One 10-week RCT reported the effectiveness of an aloe vera-based hydrogel in older adults. Participants (n = 30) had Category/Stage II, III and IV pressure injuries (47% were Category/Stage II pressure injuries) that had no local clinical infection, sinus tracts or undermining. Pressure injuries were treated daily with either the hydrogel or a saline gauze dressing. There was no significant difference between the two groups in percent of pressure injuries reaching complete healing during the study period (63% vs 64%) odds ratio [OR] 0.93, 95% confidence interval [CI] 0.16 to 5.2, p = 0.92¹⁵ (*Level 1*). However, a second RCT¹⁴ reported a hydrogel was superior to povidone-iodine gauze for promoting healing in non-infected pressure injuries in individuals (n = 27, with n = 49 pressure injuries) with spinal cord injury (SCI). Significantly more pressure injuries reached complete healing in the hydrogel dressing group (84% vs 54.2%, p = 0.04), although there was no significant difference in the time taken to reach complete healing (p = 0.06) or in the rate of healing (hydrogel 0.12 ± 0.16 cm²/day versus povidone-iodine gauze 0.09 ± 0.05cm²/day, p = 0.97). This study also included Category/Stage I pressure injuries for which a hydrogel is generally not indicated¹⁴ (*Level 1*). Neither of these studies^{14,15} provided a detailed description of the pressure injuries (e.g., level of exudate), and this factor may contribute to the studies’ findings.

These RCTs provide low quality evidence from small and under-powered studies suggesting hydrogel dressings are not inferior to simple wound dressings when used on non-infected pressure injuries. A systematic review of studies reporting hydrogel dressing used for Category/Stage II or greater pressure injuries concluded that the evidence was of insufficient quantity or quality to make clear comparisons between hydrogel dressings and other advance dressings or simple wound dressings.²¹

Hydrogel dressings generally require less frequent dressing changes¹⁴ (*Level 1*). Amorphous or non-adhesive sheet hydrogels have been associated with lower levels of wound pain due to the lack of an adherent surface, and have been rated by individuals with pressure injuries as more comfortable to wear than a saline-soaked gauze dressing¹⁶ (*Level 1*).

Polymeric membrane dressings consist of a hydrophilic, polyurethane matrix that contains wound cleanser, glycerin and absorbent polymer. This type of wound dressing is designed to manage moisture^{19,23} and claimed to subdue activity of nociceptive neuron, thereby preventing the spread inflammation and edema.^{23,24} In laboratory-based studies, a polymeric membrane dressing was demonstrated to absorb fluid to an 83% increase in its weight over 24 hours, suggesting ability to manage exudate.¹⁹

However, published, peer-reviewed clinical evidence on the effectiveness of polymeric dressings is limited. One very small study¹⁷ reported 67% of three Category/Stage I pressure injuries healed in 19 days or fewer, and 67% of nine Category/Stage II pressure injuries healed in 61 days or fewer when treated with a polymeric dressing. The older adults in this study had a range of comorbidities and received unspecified nutritional interventions to promote healing¹⁷ (*Level 4*). There was no comparison group; however, the mean time for healing is within the range that has been identified as the usual healing time for a Category/Stage II pressure injury (range 33 days to 73 days).²⁵ In older adults (n = 44) with non-infected Category/Stage II pressure injuries, a polymeric dressing was associated with significantly greater improvements in score on the PUSH tool compared to antibiotic ointment plus a dry dressing (mean score improvement 3.24 ± 2.32 vs 1.61 ± 1.61637, p < 0.0001). In this non-blinded RCT, participants received nutritional supplements, advanced support surfaces and frequent repositioning.¹⁸ However, the comparator care regimen was not considered to be standard wound care (*Level 1*).

Advanced Wound Dressings for Full Thickness Pressure Injuries

Category/Stage III and IV pressure injuries are deeper wounds with a cavity. To prevent premature superficial closure, wounds with a deep cavity, undermining or tunneling usually require packing with a moist filler dressing (e.g., moist gauze, amorphous gel, alginate etc.) underneath a secondary wound dressing.²⁶ Filler dressings also play a role in exudate management. Selection of the filler dressing and the secondary dressing is based on the considerations outlined in *Recommendation 14.1*.

14.6: Use hydrogel dressings for non-infected Category/Stage III and IV pressure injuries with minimal exudate. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Evidence from one low quality Level 1 study¹⁶ suggests that over 12 weeks a Category/Stage III or IV pressure injury treated with a hydrogel is more likely to have reduction in depth and less likely to require regular weekly debridement than a pressure injury treated with standard wet saline gauze. Ratings from individuals with pressure injuries suggested a hydrogel dressing is a comfortable wound dressing choice and wound dressing changes might be required less frequently than with a moistened gauze dressing.¹⁶

Implementation Considerations

- Hydrogel dressings can be used for autolytic debridement¹⁶ (*Level 1*). Review the guideline chapter *Cleansing and Debridement* for guidance on the use of hydrogels for wound debridement.
- Consider using hydrogel dressing for treatment of a dry wound bed.²¹ When there are high levels of exudate present, use of a hydrogel dressing could increase the risk of maceration (*Level 5*).
- Consider use of hydrogel sheets dressings for pressure injuries without depth and contours and/or on body areas that are at risk for wound dressing migration (*Expert opinion*).
- Protect the peri-wound skin (e.g., with a barrier product) to prevent maceration²² (*Expert opinion*).

Evidence Discussion

Matzen et al. (1999)¹⁶ randomly assigned amorphous hydrogel or a continuously wet dressing in 32 participants with non-infected Category/Stage III or IV pressure injuries on the sacrum or trochanter. Despite a large loss of sample size, wound volume was significantly smaller in the hydrogel group at 12 weeks ($26 \pm 20\%$ versus $64 \pm 16\%$, $p < 0.02$). As previously discussed in this chapter, hydrogel dressings promote autolytic debridement. In the study by Matzen et al. (1999),¹⁶ fewer Category/Stage III pressure injuries treated with the hydrogel-dressing required weekly debridement to remove necrotic tissue from the wound bed compared with those treated with the moist saline gauze (21% versus 7%, $p < 0.03$) (*Level 1*). These findings are consistent with reports from research conducted in wounds of mixed etiology. For example, Zoellner et al. (2007)²⁷ found that mean wound surface area covered in slough reduced from 63% to 34% after three wound dressing changes when a hydrogel dressing was used in chronic wounds²⁷ (*Level 5*).

Individuals with Category/Stage III and IV pressure injuries have reported no difference between hydrogel dressing and saline soaked gauze dressing for management of wound pain or malodor¹⁶ (*Level 1*).

14.7: Use calcium alginate dressings for Category/Stage III and IV pressure injuries with moderate exudate. (Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

There is evidence from low and moderate quality Level 1 studies^{28,29} plus additional lower level evidence³⁰ indicating that full thickness pressure injuries treated with an alginate dressing will have greater reduction in surface area and depth compared with some other contemporary wound dressings. After eight weeks of treatment using a sequential calcium alginate/hydrocolloid dressing regimen, reduction in wound surface area could be about 26% greater than using a hydrocolloid dressing alone.²⁸ Individuals with pressure injuries rated alginate dressings as less painful to remove than a hydrocolloid dressing.²⁸

Implementation Considerations

- Irrigate the calcium alginate dressing to facilitate full removal of the dressing material.²² Residual alginate material could cause potential injury if product remains in wound bed. The low tensile strength of a moist calcium alginate dressing makes it unsuitable for use in narrow sinus tracts. (*Expert opinion*). Refer to the guideline chapter *Cleansing and Debridement* for more discussion on cleansing a pressure injury.
- Calcium alginate dressings are available in rope or sheet form; selection is based on the depth, shape and level of exudate of the wound bed (*Expert opinion*).
- Avoid using calcium alginate dressings if the wound bed is dry²² (*Expert opinion*).

Evidence Discussion

Calcium alginate is a highly absorbent alginate dressing derived from seaweed. Alginate dressings maintain a physiologically moist microenvironment that promotes healing and the formation of granulation tissue. Alginate dressings can often be left on a pressure injury for several days, thereby decreasing frequency of dressing changes. Residual alginate fibers should be completely removed from the wound bed.

In an RCT, Belmin et al. (2002)²⁸ reported that non-infected Category/Stage II and III pressure injuries in older adults (n = 110) were statistically significantly reduced in size with a dressing regimen that included an alginate dressing for four weeks followed by a hydrocolloid dressing for four weeks when compared to a regimen of a hydrocolloid dressing alone for eight weeks. The mean between-group difference in surface area reduction was 26.5% (95% CI 10.62 to 42.38) (*Level 1*). In a second RCT, Sayag et al. (1996)²⁹ also reported that the mean healing time was reduced in full thickness pressure injuries (n = 92) treated with a calcium alginate dressing compared to those receiving 3 mm thick dextranomer paste. In the calcium alginate dressing group, 74% of pressure injuries achieved at least 40% reduction in surface area, compared to 42% of the dextranomer group (p = 0.002). The rate of healing was also significantly faster for those pressure injuries treated with calcium alginate (2.39 ± 3.54cm²/week vs 0.27 ± 3.21cm²/week, p = 0.0001). However, this trial did not report an endpoint of complete wound healing, and there was high attrition in both groups (21% in the alginate group and 49% in the dextranomer group) (*Level 1*). In an uncontrolled observational study, individuals with Category/Stage III and IV pressure injuries receiving initial treatment of 4 to 6 weeks with a calcium alginate dressing followed by foam dressing experienced a significant reduction in mean surface area over time. At 12 weeks, the mean absolute surface area reduced from 12.5 ± 7.5cm² to 3.7 ± 5.2cm² (p < 0.001). At 4 weeks, 40% of the pressure injuries had achieved a mean surface area reduction of at least 50%, and by the end of the trial at 12 weeks, 75% of the pressure injuries had a mean surface area of 50% or greater³⁰ (*Level 4*).

In one of the RCTs above, individuals rated pain during removal of the calcium alginate dressing as significantly lower compared to removal of a hydrocolloid dressing (p = 0.03) and health professionals rated calcium alginate and hydrocolloids as equivalent with respect to ease of removal (p = 0.11). However, the study was conducted in 2002 and the findings may not accurately reflect products on the market today²⁸ (*Level 1*).

Wound Dressings for Pressure Injuries with High Exudate

14.8: Use foam dressings (including hydropolymers) for Category/Stage II and greater pressure injuries with moderate/heavy exudate.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

There is evidence from a low quality Level 1 study¹¹ and a number of low quality Level 4 studies^{6,31,32} suggesting that foam dressings offer an improvement in measures of pressure injury healing,^{6,11,31} management of peri-wound skin³² and reduction in wound exudate^{11,31} in pressure injuries with high levels of exudate. Adverse effects appear to be minimal. Ratings from both individuals with pressure injuries¹¹ and health professionals³¹ indicate that foam dressings are likely to be an acceptable wound dressing choice.

Implementation Considerations

- A gelling foam dressing or hydropolymer can be used for managing pressure injuries with high exudation (*Expert opinion*).
- Avoid using small pieces of foam in cavity pressure injuries (*Expert opinion*).
- Filler dressings should be used beneath foam dressings in deep ulcers to fill in dead space and prevent fluid accumulation. Lightly pack the wound cavity, including tunneling and undermining (*Expert opinion*).
- Clinical uses of foam dressings include application as a cover dressing to extend wear time (*Expert opinion*).

Evidence Discussion

Foam dressings absorb wound exudate from the wound bed. Simple foam dressings wick exudate from the wound bed and translocate it to the surface of the wound dressing. Complex foam dressings absorb wound exudate by dispersing it throughout the wound dressing for retention away from the skin. Gelling foam dressings manage excess wound exudate and protect surrounding skin from prolonged exposure to wound or body fluids. Foam dressings also promote moisture evaporation, thereby allowing more drainage to be wicked away from the wound bed and surrounding skin.

Bale et al. (1997)¹¹ compared a gelling foam dressing to a hydrocolloid for promoting healing and managing exudate in Category/Stage II and III pressure injuries of < 11 cm diameter. The researchers concluded that the foam dressing managed exudate significantly more effectively based on subjective ratings of absorbency from the study participants. However, there was no significant difference in the percent of pressure injuries in each group that reached complete wound healing within the 30-day study period (foam 24% versus hydrocolloid 16%) (*Level 1*).

Souliotis et al. (2016)⁶ conducted an RCT (n = 100) comparing a range of moist wound healing foam dressings, including non-medicated foam, silver foam, silver-sulfadiazine foam and ibuprofen foam with gauze dressings in the management of Category/Stage III or IV pressure injuries. Pressure injuries treated with foam dressings had significantly faster healing times (85.56 ± 52.09 days vs 121.4 ± 5 2.21 days, p = 0.0001), and required fewer dressing changes compared to those treated with plain gauze (p < 0.0001). In this study antiseptic solutions were used on pressure injuries that showed clinical signs of local wound infection, and some of the foam dressing had antimicrobial properties; these confounding factors may have influenced the study results (*Level 1*).

Diehm and Lawall (2005)³¹ reported a descriptive study of 6,693 participants with chronic exuding ulcers of multiple etiologies, including 1,793 participants with pressure injuries. 4.5% of the pressure injuries were classified as superficial, and 49% were described as infected. The pressure injuries were managed with a hydropolymer foam dressing. At four weeks, there was a 67% reduction in ulcer radius; 39% of the pressure injuries had healed, and 56% were improved. At 12 weeks, there was an 87.5% reduction in wound radius, with 58% healed and 43.9% improved. At 12 weeks, only 3.8% of the pressure injuries had moderate to large exudate volumes, compared to 42.4% at study commencement (*Level 4*).

Parish et al. (2008)³² conducted a small observational study (n = 23) of an adhesive, gelling foam dressing used to treat Category/Stage II pressure injuries ≥ 2cm² and Category/Stage III and IV pressure injuries. At 28 days, 4% of pressure injuries were described as healed, 30% had a marked improvement, 26% showed mild improvement, 4% had mild deterioration and 9% were markedly deteriorated. The gelling foam was associated with 65% improvement in the peri-wound skin condition, indicating effective management of exudate (*Level 4*).

In a meta-analysis of three Level 1 studies that were of low quality, Walker et al. (2017)³³ identified that foam dressings were neither inferior nor superior to hydrocolloid dressings for promoting pressure injury healing. Relative risk of healing when a foam dressing was applied was 0.85 (95% confidence interval [CI] 0.54 to 1.34, p = 0.77), which was not statistically significantly different from using a hydrocolloid dressing.³³ There was also no difference in adverse events between the two dressing types (RR = 0.88, 95% CI 0.37 to 2.11). The primary inquiry of this review was impact of foam dressings on healing, and some studies in the meta-analysis included pressure injuries with low exudate levels. Another Cochrane meta-analysis of low quality Level 1 studies reported foam dressings were superior to saline gauze dressings for pressure injury healing (RR 1.52, 95% CI 1.03 to 2.26), although there was low certainty for this evidence.³⁴

Evaluation of the acceptability of foam dressings to individuals with pressure injuries found foam dressings to be rated significantly better than a hydrocolloid dressing (p = 0.018) for their ability to conform to the body and easier to remove¹¹ (*Level 1*).

14.9: Use super-absorbent wound dressings with a high capacity for absorption to manage heavily exuding pressure injuries.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

There is low level evidence from one moderate quality Level 4 study³⁵ suggesting that a super-absorbent dressing is associated with healing of heavily exuding pressure injuries. Individuals with pressure injuries reported improvements in quality of life and reduction of pain by approximately three points on an 11-point scale when a super-absorbent dressing is used. There is no evidence on possible adverse events or the resource requirements for his wound dressing.

Implementation Considerations

- Do not use super-absorbent dressings for wounds with mild to moderate exudate due to the risk of the wound bed becoming too dry³⁶ (*Expert opinion*).
- Evaluate the structural integrity of the super-absorbent dressing regularly to ensure the dressing is appropriate for the volume of exudate.³⁶ (*Level 5*).

Evidence Discussion

Super-absorbent dressings are complex multilayer dressings that provide either a semi-adherent quality or a non-adherent layer, combined with highly absorptive fibre layers from various materials that have increased ability to wick moisture (e.g., cellulose, cotton, rayon or gel particles). Super-absorbent dressings are designed to absorb larger volumes of wound exudate while locking the fluid away from skin to prevent maceration, skin breakdown, strike through and leakage.^{35,36}

The body of evidence for super-absorbent dressings for managing pressure injuries is small, although there is a wider body of evidence for the use of these dressings in other wound types.^{37,38} In pressure injuries the evidence is limited to a case study by Van Leen et al. (2014)³⁵ using super-absorbent dressings in a small convenience sample of participants (n = 11) with Category/Stage II to IV pressure injuries and venous leg ulcers (n = 20). Mean PUSH Tool scores decreased from 11.05 at baseline to 5.0 over eight weeks, and there was a mean reduction in wound surface area from 15.27 cm² to 7.63 cm². Participant-rated levels of pain and negative social impact of having a wound also reduced over the course of the study (*Level 4*).

Basic Wound Dressings

The types of contemporary wound dressings that are available have rapidly expanded in recent years. Modern wound dressings have been designed to maximize wound healing while promoting comfort of the individual and reducing the need for frequent wound dressing changes. However, access to contemporary wound dressing products is limited in many geographic regions. The recommendations in this section should be considered in the context of geographic regions that do not have access to modern wound dressings. These recommendations may not represent best practice in a healthcare system in which modern wound dressings are relatively accessible.

14.10: Use moist gauze dressings to maintain an appropriately moist wound environment when advanced wound dressings are not an option.
(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

Two low quality Level 1 studies^{6,16} indicated that moist gauze dressings are associated with complete pressure injury healing, although healing took approximately 30% longer than compared to more modern wound dressings (e.g., hydrocolloids and foams).⁶ A low quality Level 1 study reported no significant difference in healing with a moist gauze dressing compared to a hydrogel, with healing rates of about 66% over 10 weeks in both groups.¹⁵ Therefore, moist wound dressings can achieve healing in the absence of an advanced wound dressing option. Moist gauze dressings were rated by individuals with pressure injuries as less comfortable than more advanced dressings¹⁶ and a low quality economic analysis suggested that the requirement for more frequent dressing changes was associated with increased costs for using a moist gauze dressing.⁶

Implementation Considerations

- Avoid the use of wet-to-dry gauze dressings. Vigilance is required in maintaining moisture of the dressing and wound bed when using moist gauze dressings (*Expert opinion*).
- Monitor the wound and peri-wound skin for maceration (*Expert opinion*).
- Consider using impregnated forms of gauze dressings to prevent evaporation of moisture (*Expert opinion*).
- Use loosely woven gauze for highly exuding pressure injuries and use more tightly woven gauze for pressure injuries with minimal exudate (*Expert opinion*).
- When filling cavities of deeper pressure injuries, loosely fill (rather than tightly pack) the dead space with saline-moistened gauze, to avoid creating pressure on the wound bed (*Expert opinion*).
- Use a single gauze strip/roll to fill deep wounds. Do not use multiple gauze dressings because retained gauze in the wound bed can serve as a source of infection (*Expert opinion*).

Evidence Discussion

Gauze dressings are made of cotton or synthetic fabric that is absorptive and permeable to water, water vapor and oxygen. Clinical practice varies widely in relation to gauze dressings. Increased infection rates, retained dressing particles, and pain have led health professionals to avoid the use of gauze dressings for open chronic wounds such as pressure injuries, in favor of advanced wound dressings.^{16,39,40} Due to the need for frequent changes, gauze dressings have been shown to be costly in health professional time and resources⁶ (*Level 1*).

A number of studies used moist gauze as a comparator dressing for Category/Stage II and greater pressure injuries.^{6,15,16} In one randomized controlled trial (RCT), saline-soaked gauze was not significantly different to a hydrogel for complete healing rates ($p = 0.92$)¹⁵ (*Level 1*). However, a second RCT showed that more advanced moist wound healing foam dressings were associated with faster average healing times ($p = 0.0001$) for Category/Stage III and IV pressure injuries compared to a plain gauze dressing⁶ (*Level 1*). Another RCT conducted with Category/Stage III and IV pressure injuries showed that compared to a hydrogel dressing, saline gauze was associated with a need for more frequent debridement ($p < 0.03$) and slower reduction in wound volume ($p < 0.02$)¹⁶ (*Level 1*).

Because studies^{6,16} indicate they can achieve faster wound healing, the formulary should provide access to modern moist wound dressing options. However, the use of saline impregnated or moistened gauze is preferable to allowing the wound bed to desiccate. In the absence of modern wound dressings, gauze may be used dry; moist; or impregnated with paraffin, petrolatum, antiseptics, or other agents. Gauze is manufactured in varying weaves, and with different size interstices. Selection of the most appropriate gauze product is important to prevent damage to the wound bed, minimize wound pain and avoid loss of product in large and deep ulcers.

14.11: Use a transparent film dressing as a secondary dressing when advanced wound dressings are not an option.
(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

One low quality Level 1 study² provided evidence that healing rates of Category/Stage II pressure injuries with a transparent film secondary dressing were not significantly different to those when a hydrocolloid dressing was used. Subjective ratings from individual with pressure injuries and health professionals showed film dressings are preferable to some other wound dressings for comfort, conformability, ease of removal, residue and ability to assess the wound. Transparent film dressings are considered a low-cost option for treating pressure injuries that are an appropriate choice in some clinical situations.

Implementation Considerations

- Transparent film dressings can be used for autolytic debridement when the individual is not immunocompromised. Review the guideline chapter *Cleansing and Debridement* for guidance on the use of film dressings in wound debridement (*Expert opinion*).
- Transparent film dressings should not be used as the tissue interface layer for pressure injuries with moderate or heavy exudate (*Expert opinion*).
- Transparent film dressings can be used as a secondary dressing to secure wound fillers (*Expert opinion*).
- Remove transparent film dressings carefully from the skin to reduce skin trauma (*Expert opinion*).
- Transparent film dressings should not be used over enzymatic debriding agents, gels or ointments (*Expert opinion*).

Evidence Discussion

Film dressings were originally designed to cover intact skin over intravenous puncture sites. The transparency of these dressings allows inspection of the skin beneath.² Plain film dressings do not absorb exudate from a wound bed; generally a transparent film dressing is used as a secondary dressing to secure a primary dressing that fills the wound bed and absorbs exudate.

In an RCT, Brown-Etris et al. (2008)² compared hydrocolloids to film dressings containing an absorptive pad for Category/Stage II pressure injuries and shallow Category/Stage III pressure injuries. There were no significant differences in pressure injuries that reached complete healing within the 56-day study period ($p = 0.96$), healing rates ($p = 0.65$) or peri-wound skin maceration ($p = 0.27$) between the two dressing regimens. Subjective ratings from the health professionals using the products ranked the film dressing as significantly easier to apply ($p = 0.005$), more conformable ($p = 0.026$) and easier to remove ($p < 0.001$). Individuals with pressure injuries ranked the film dressing as more comfortable ($p < 0.001$). The researchers concluded that film dressings can be used for shallow pressure injuries² (*Level 1*).

The Wound Ostomy and Continence Nurses Society and Agency for Health Care Policy and Research (AHCPR) guidelines address the role of film dressings for autolytic debridement.^{41,42} As outlined in the guideline chapter *Cleansing and Debridement*, autolytic debridement is commonly performed with film dressings to allow easy wound monitoring.

14.12: Consider the available evidence and guidance on using local resource dressings when selecting wound dressings in geographic regions with limited access to resources.
(Good Practice Statement)

Implementation Considerations

- Evidence on local resource wound dressings can be accessed using a research database established by the World Health Organization to promote access to health literature in low- and middle-income countries (Hinari, <http://www.who.int/hinari/en/>) (*Expert opinion*).

Discussion

Health professionals practicing in resource limited areas need to evaluate product availability and review the efficacy and potential risks of locally available products in order to make choices with patient consumers and their informal caregivers regarding the most appropriate wound management.⁴³ Evidence for efficacy is available for local resource wound dressings (e.g., banana leaf dressing,⁴⁴ potato dressing⁴⁵ etc.) in wounds of different etiologies and could be extrapolated to pressure injuries (*Level 5*).

References

1. Winter GD. Formation of the scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. *Nature*, 1962; 193: 293-294.
2. Brown-Etris M, Milne C, Orsted H, Gates JL, Netsch D, Punchello M, Couture N, Albert M, Attrell E, Freyberg J. A prospective, randomized, multisite clinical evaluation of a transparent absorbent acrylic dressing and a hydrocolloid dressing in the management of Stage II and shallow Stage III pressure ulcers. *Adv Skin Wound Care*, 2008; 21(4): 169-174.
3. Stephen-Haynes J. The outcomes of barrier protection in periwound skin and stoma care. *Br J Nurs*, 2014; 23(5): S26-S30.
4. Kohta M, Iwasaki T. The effect of concentration of tackifying agent on adhesive and skin-protective properties of ceramide 2-containing hydrocolloid dressings. *J Wound Care*, 2015; 24(1): 41-8.
5. Meaume S, Van De Looverbosch D, Heyman H, Romanelli M, Ciangherotti A, Charpin S. A study to compare a new self-adherent soft silicone dressing with a self-adherent polymer dressing in stage II pressure ulcers. *Ostomy Wound Management*, 2003; 49(9): 44-51.
6. Souliotis K, Kalemikerakis I, Saridi M, Papageorgiou M, Kalokerinou A. A cost and clinical effectiveness analysis among moist wound healing dressings versus traditional methods in home care patients with pressure ulcers. *Wound Repair Regen*, 2016; 24(3): 596-601.
7. Lima AF, Castilho V, Baptista CM, Rogenski NM, Rogenski KE. Direct cost of dressings for pressure ulcers in hospitalized patients. *Rev Bras Enferm*, 2016; 69(2): 290-297.
8. Chuangsuwanich A, Chotrakarnkij P, Kangwanpoom J. Cost-effectiveness analysis in comparing alginate silver dressing with silver zinc sulfadiazine cream in the treatment of pressure ulcers. *Archives of Plastic Surgery*, 2013; 40(5): 589-596.
9. Silva DRA, Bezerra SMG, Costa JP, Luz M, Lopes VCA, Nogueira LT. Pressure ulcer dressings in critical patients: A cost analysis. *Rev Esc Enferm USP*, 2017; 51: e03231.
10. Dhaliwal I. *Comparative Cost-Effectiveness to Inform Policy in Developing Countries*. 2011 [cited May 2019]; Available from: <https://www.povertyactionlab.org/sites/default/files/Lecture%20Cost%20Effectiveness.pdf>.
11. Bale S, Squires D, Varnon T, Walker A, Benbow M, Harding KG. A comparison of two dressings in pressure sore management. *J Wound Care*, 1997; 6(10): 463-466.
12. Graumlich JF, Blough LS, McLaughlin RG, Milbrandt JC, Calderon CL, Agha SA, Scheibel LW. Healing pressure ulcers with collagen or hydrocolloid: A randomized, controlled trial. *J Am Geriatr Soc*, 2003; 51(2): 147-154.
13. Hao DF, Feng G, Chu WL, Chen ZQ, Li SY. Evaluation of effectiveness of hydrocolloid dressing vs ceramide containing dressing against pressure ulcers. *Eur Rev Med Pharmacol Sci*, 2015; 19(6): 936-941.
14. Kaya AZ, Turani N, Akyüz M. The effectiveness of a hydrogel dressing compared with standard management of pressure ulcers. *J Wound Care*, 2005; 14(1): 42-4.
15. Thomas DR, Goode PS, LaMaster K, Tennyson T. Acemannan hydrogel dressing versus saline dressing for pressure ulcers. A randomized, controlled trial. *Adv Wound Care*. , 1998; 11(6): 273-6.
16. Matzen S, Peschardt A, Alsbjorn B. A new amorphous hydrocolloid for the treatment of pressure sores: A randomised controlled study. *Scand J Plast Reconstr Surg Hand Surg*, 1999; 33(1): 13-5.
17. Carr RD, Lalagos DE. Clinical evaluation of a polymeric membrane dressing in the treatment of pressure ulcers. *Decubitus*, 1990; 3(3): 38-42.
18. Yastrub DJ. Relationship between type of treatment and degree of wound healing among institutionalized geriatric patients with stage II pressure ulcers. *Care Management*, 2004; 5(4): 213-8.
19. Gorska A, Dorozynski P, Weglarz WP, Jasinski K, Kurek M, Jachowicz R, Klaja J, Kulinowski P. Spatiotemporal characterization of hydration process of asymmetric polymeric wound dressings for decubitus ulcers. *Journal of Biomedical Materials Research Part B Applied Biomaterials*, 2017.

20. Milne CT, Barrere CC, McLaughlin T, Moore A. Surgical hip dressings: A comparison of taping methods. *Orthop Nurs*, 1999; 18(3): 37-42.
21. Dumville JC, Stubbs N, Keogh SJ, Walker RM *Hydrogel dressings for treating pressure ulcers*. Cochrane Database Syst Rev, 2014. DOI: 10.1002/14651858.CD011226.
22. Adkins CL. Wound care dressings and choices for care of wounds in the home. *Home Healthc Nurse*, 2013; 31(5): 259-267.
23. Denyer J, Agathangelou C, White R, Ousey K, Nair H. POLyMem dressing made easy. *Wounds International*, 2015.
24. Gefen A. Managing inflammation by means of polymeric membrane dressings in pressure ulcer prevention. *Wounds International*, 2018; 9(1): 22-28.
25. Bergstrom N, Smout R, Horn S, Spector W, Hartz A, Limcangco MR. Stage 2 pressure ulcer healing in nursing homes. *J Am Geriatr Soc*, 2008; 56(7): 1252-1258.
26. Dabiri G, E. D, Phillips T. Choosing a wound dressing based on common wound characteristics. *Adv Wound Care*, 2016; 5(1): 32-41.
27. Zoellner P, Kapp H, Smola H. Clinical performance of a hydrogel dressing in chronic wounds: a prospective observational study. *J Wound Care*, 2007; 16(3): 133-6.
28. Belmin J, Meaume S, Rabus MT, Bohbot S, The Investigators of the Sequential Treatment of the Elderly with Pressure Sores T. Sequential treatment with calcium alginate dressings and hydrocolloid dressings accelerates pressure ulcer healing in older subjects: A multicenter randomized trial of sequential versus nonsequential treatment with hydrocolloid dressings alone. *Journal of the American Geriatrics Society*, 2002; 50(2): 269-74.
29. Sayag J, Meaume S, Bohbot S. Healing properties of calcium alginate dressings. *J Wound Care*, 1996; 5(8): 357-62.
30. Ausili E, Paolucci V, Triarico S, Maestrini C, Murolo D, Focarelli B, Rendeli C. Treatment of pressure sores in spina bifida patients with calcium alginate and foam dressings. *Eur Rev Med Pharmacol Sci*, 2013; 17(12).
31. Diehm C, Lawall H. Evaluation of Tielle hydropolymer dressings in the management of chronic exuding wounds in primary care. *Int Wound J*, 2005; 2(1): 26-35.
32. Parish LC, Dryjski M, Cadden S. Prospective clinical study of a new adhesive gelling foam dressing in pressure ulcers. *Int Wound J*, 2008; 5(1): 60-67.
33. Walker RM, Gillespie BM, Thalib L, Higgins NS, Whitty JA. Foam dressings for treating pressure ulcers. Cochrane Database Syst Rev, 2017; 2017 (10) (no pagination)(CD011332).
34. Westby MJ, Dumville JC, Soares MO, Stubbs N, Norman G. Dressings and topical agents for treating pressure ulcers. Cochrane Database Syst Rev, 2017; 2017 (6) (no pagination)(6): CD011947.
35. Van Leen M, Rondas A, Neyens J, Cutting K, Schols JMGA. Influence of superabsorbent dressings on non-healing ulcers: A multicentre case series from the Netherlands and the UK. *J Wound Care*, 2014; 23(11): 543-550.
36. Browning P, White RJ, Rowell T. Comparative evaluation of the functional properties of superabsorbent dressings and their effect on exudate management. *J Wound Care*, 2016; 25(8): 452-462.
37. Faucher N, Safar H, Baret M, Philippe A, Farid R. Superabsorbent dressings for copiously exuding wounds. *Br J Nurs*, 2012; 21(12): S22, s24, s26-8.
38. Wahab N, Wray K. *The use of a superabsorbent three layer wicking wound dressing to manage diabetic wounds in a long-term care facility*. 2015; Available from: <http://wce.lasvegaswebsolution.com/wp-content/uploads/2016/06/Qwick-2015-Wahab-LIT031WC.pdf>.
39. Singh A, Halder S, Menon GR, Chumber S, Misra MC, Sharma LK, Srivastava A. Meta-analysis of randomized controlled trials on hydrocolloid occlusive dressing versus conventional gauze dressing in the healing of chronic wounds. *Asian Journal of Surgery*, 2004; 27(4): 326-332.
40. Kordestani S, Shahrezaee M, Tahmasebi MN, Hajimahmodi H, Ghasemali DH, Abyaneh MS. A randomised controlled trial on the effectiveness of an advanced wound dressing used in Iran. *Journal of Wound Care*, 2008; 17(7): 323-327.
41. Bergstrom N, Bennett, M.A., Carlson, C.E., et al, *Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. AHCPH Pub. No. 95-0653*. 1994, Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, Agency for Healthcare Policy and Research.
42. Wound Ostomy and Continence Nurses Society (WOCNS), *Wound Ostomy and Continence Nurses Society. Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: Wound Ostomy and Continence Nurses Society.
43. Bolton LL. Resources for optimising wound outcomes in low-resource settings. *Wound Practice and Research*, 2017; 25(3): 127-132.
44. Wound Healing and Management Node Group. Wound management - Low resource communities: Banana leaf dressing. *Wound Practice and Research*, 2017; 25(3): 156-157.
45. Haesler E, Watts R, Solomons T. Wound management - Low resource communities: Potato peel dressings. *Wound Practice and Research*, 2017; 25(3): 154-155.

BIOLOGICAL DRESSINGS FOR TREATMENT OF PRESSURE INJURIES

Introduction

Biological dressings function as protective wound cover and may be cellular (contain living cells) or acellular (biologically inert). They can be composed of:

- Animal (bovine or porcine) material (e.g. collagen)
- Human (cadaveric skin) cells
- Plant (cellulose) materials
- Synthetic (man-made) material
- Composite of materials (e.g. collagen and oxidized regenerated cellulose [ORC]).¹

Biological dressings include skin substitutes, xenografts, allografts or collagen dressings. The majority of research on biological dressing use in pressure injuries has been conducted on collagen-based dressings.

Clinical Question

The clinical question that guided the development of this chapter was:

- What biological dressings are effective for supporting healing of pressure injuries?

Collagen Matrix Dressings

Collagen dressings are mostly derived from bovine, porcine, or avian skin made into sheets and pads, as particles, and as gels. Collagen is a protein produced by fibroblasts that promotes the reduction of protease activity while encouraging angiogenesis, epithelization and granulation during the wound healing process. Proteases such as metalloproteinases (MMPs) and elastase are enzymes that break down proteins into peptides and amino acids. An imbalance of proteases, reactive oxygen species, and protease inhibitors within the wound microenvironment would delay the wound healing process. During the inflammatory phase, barriers against microorganisms and damaged tissues will be created via the actions of MMPs.²⁻⁴ Excessive proteases can prolong the inflammatory stage, thereby inhibiting the progression to the proliferative phase and delaying the wound healing process. Furthermore, collagen promotes dermal fibroblast proliferation, stimulates the migration of the cell, and growth of the capillary bed.³⁻⁵ These actions encourage the growth of healthy tissues in the wound environment and aid in the repair process. In chronic wounds such as pressure injuries, collagen lowers the elastase level, altering the chronicity of the wound and heightening the healing process.³⁻⁵

15.1: Consider applying collagen dressings to nonhealing pressure injuries to improve rate of healing and decrease signs and symptoms of wound inflammation.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

There is direct evidence from low, moderate and high quality Level 1 studies to suggest that collagen matrix dressings are as effective as other contemporary wound dressings (e.g. hydrocolloid, hydropolymer and foam) in promoting healing.⁶⁻⁸ In one low quality Level 1 study,⁹ a collagen matrix dressing out-performed a hydropolymer dressing in achieving reduction in pressure injury surface area and in other studies, indicators of wound inflammation were more favorable in pressure injuries treated with collagen.^{6,9} Older studies indicated that collagen matrix dressings cost more with respect to product and labor than other types of wound dressings;⁸ however, a cost-benefit analysis is not available. Consideration should be given to resource availability because collagen matrix dressings might be more costly than other contemporary wound dressings^{6,8} and/or difficult to access. Consideration should also be given to patient preferences (collagen is derived from animal products).³⁻⁵

Implementation Considerations

- Collagen dressings are animal-based products¹⁰ and may not be acceptable to all individuals. Check the product description for more information on its derivation and discuss the individual's treatment preferences. (*Expert opinion*).
- Collagen dressings are not appropriate for pressure injuries with dry eschar^{11,12} (*Indirect evidence*).

- Collagen creates a gel residue in the wound bed. If gel residue is visible, the collagen dressing should remain untouched until the next dressing review (generally every third day or more frequently for wounds with heavy exudate)^{3,11} (*Expert opinion and indirect evidence*).
- Optimize the individual's ability to heal by addressing nutritional status, management of co-morbidities and pressure offloading through repositioning and use of appropriate support surfaces (*Expert opinion*).

Evidence Discussion

In a 2016 randomized, controlled trial (RCT), Kloeters et al. (2016)⁹ recruited 33 people with pressure injuries and randomly included them into two groups: collagen/ORC matrix (n = 23) or an absorbent hydropolymer foam dressing (n = 10). Pressure injuries treated with collagen/ORC matrix showed a significant reduction in wound surface area compared to the hydropolymer dressing group (65 ± 13% versus 41 ± 11%, p < 0.05). Protease activity was also explored. Compared to baseline, there was a significant reduction in elastase reactivity at days 5, 14, 28, 42 and 56 for the collagen/ORC dressing group (p < 0.05 for all), and this reduction in elastase activity was significant compared to the hydropolymer dressing group at day 5 and day 14. Plasmin activity was also significantly reduced at days 5 and 14 for pressure injuries treated with collagen/ORC matrix compared to the control group (p < 0.05 for both)¹³ (*Level 1*).

Graumlich et al. (2003)⁸ presented an RCT comparing collagen wound dressing to a hydrocolloid wound dressing. Participants (n = 65), who were recruited from 11 nursing homes, (about 80% of participants had Category/Stage II pressure injuries and 20% of participants had Category/Stage III pressure injuries). The mean duration of pressure injuries was from 3 to 6.5 weeks. Individuals were randomized to receive the collagen wound dressing that consisted of application of sterile saline, collagen sprinkled in a thin continuous layer over the wound bed and application of gauze (n = 35). The comparator group received a hydrocolloid wound dressing (n=35). After eight weeks of treatment, it appeared that the collagen wound dressing was as effective as the hydrocolloid wound dressing in achieving complete wound healing (mean difference 1%; 95% confidence interval [CI] 26 to 29%; p = 0.893). However, the cost analysis showed the collagen wound dressing was more expensive than hydrocolloid wound dressing (average per patient cost, hydrocolloid \$222 USD versus collagen \$627 USD in 2003). Collagen wound dressings required seven nursing interventions per week versus two interventions per week for hydrocolloid wound dressings⁸ (*Level 1*).

Eighty individuals with Category/Stage II to IV pressure injuries received either a collagen matrix dressing or a viscoeyan dressing in another RCT.⁷ The pressure injuries were debrided and treated with a topical antimicrobial until there were no signs of local infection before commencing the dressing trial. There was no significant difference in the percent of pressure injuries that were completely healed at six months (90% collagen group versus 70% control group, p = 0.59). Time to healing was reported to be faster for pressure injuries treated with collagen (2 to 6 weeks versus 2 to 8 weeks), and this led to a shorter hospitalization period for the collagen matrix group. The collagen group also need fewer wound dressing changes. Although this suggests that a collagen matrix dressing may reduce resource use, no cost analysis was conducted⁷ (*Level 1*).

One pilot RCT conducted in individuals with Category/Stage III pressure injuries of at least four weeks duration (n = 10) demonstrated that a collagen wound dressing was associated with a significant positive effect on angiogenesis (p < 0.05) compared with a foam wound dressing. After 21 days of second-daily dressings, 100% of the pressure injuries treated with collagen wound dressing were healed, compared with 80% treated with the foam wound dressing.⁶ In the same study,⁶ it was observed that a collagen dressing may have some effect on reducing inflammatory factors. After seven days of treatment, the concentration of metalloproteinases-9 (MMP-9) was lower in pressure injuries treated with collagen wound dressing than in pressure injuries treated with foam wound dressing (p < 0.04)⁶ (*Level 2*).

Available collagen dressings contain different types of collagen and the dressings come in different formats (e.g. gel versus dressing sheet versus particles).¹⁰ Different collagen types attract different MMPs in the wound, therefore the biological effect may vary, although the general principles underlying the effect of the collagen on wound healing are the same.¹⁰ Different collagen dressing formats are primarily a choice of ease of application, although larger size of dressing pores may allow more rapid concentration of MMPs within the dressing.¹⁰ Exploration of potential differences between different collagen types and different collagen dressing formats has not been undertaken in pressure injuries.

Other Biological Dressings

Some evidence is available on other types of biological dressings for treating pressure injuries, including hyaluronic acid derivative wound dressing,¹⁴ a bi-layered cell therapy wound dressing¹⁵ and an amniotic membrane dressing.¹⁶ The volume of evidence for these biological dressing interventions is currently insufficient to make any specific recommendations.

References

1. Wounds International, International consensus. Acellular matrices for the treatment of wounds. An expert working group review. 2010, Wounds International: London.
2. Wu S, Applewhite AJ, Niezgodá J, Snyder R, Shah JP, Cullen B, Schultz G, Harrison J, Hill R, Howell M, Speyrer M, Utra H, de Leon J, Lee W, Treadwell T. Oxidized Regenerated Cellulose/Collagen Dressings: Review of Evidence and Recommendations. *Advances in Skin & Wound Care*, 2017; 30: S1-S81.
3. Grothier L. Understanding the use of collagen/oxidised regenerated cellulose dressings. *Wounds Int*, 2015; 6(2): 34-40.
4. Fleck CA, Simman R. Modern collagen wound dressings: Function and purpose. *J Am Col Certif Wound Spec*, 2010 2(3): 50-4.
5. Wu S, Applewhite AJ, Niezgodá J, Snyder R, Shah JP, Cullen B, Schultz G, Harrison J, Hill R, Howell M, Speyrer M, Utra H, de Leon J, Lee W, Treadwell T. Oxidized regenerated cellulose/collagen dressings: Review of evidence and recommendations. *Adv Skin Wound Care*, 2017; 30: S1-S81.
6. Piatkowski A, Ulrich D, Seidel D, Abel M, Pallua N, Andriessen A. Randomised, controlled pilot to compare collagen and foam in stagnating pressure ulcers. *J Wound Care*, 2012; 21(10): 505-511.
7. Nisi G, Brandi C, Grimaldi L, Calabrò M, D'Aniello C. Use of a protease-modulating matrix in the treatment of pressure sores. *Chir Ital*, 2005; 57(4): 465-468.
8. Graumlich JF, Blough LS, McLaughlin RG, Milbrandt JC, Calderon CL, Agha SA, Scheibel LW. Healing pressure ulcers with collagen or hydrocolloid: A randomized, controlled trial. *J Am Geriatr Soc*, 2003; 51(2): 147-154.
9. Kloeters O, Unglaub F, de Laat E, van Abeelen M, Ulrich D. Prospective and randomised evaluation of the protease-modulating effect of oxidised regenerated cellulose/collagen matrix treatment in pressure sore ulcers. *Int Wound J*, 2016; 13(6): 1231-1236.
10. Brett D. A review of collagen and collagen-based wound dressings. *Wounds* 2008; 20(12): 347-356.
11. Rao H, Pai A, Hussein I, Arun A, Ram HS, Pai A, Pai SR, Pain SG. A comparative study between collagen dressings and conventional dressings in wound healing. *Int J Collab Res Intern Med Public Health*, 2012; 4(5): 611-23.
12. Haesler E. Biological dressings: Collagen-based dressings. *Wound Practice and Research*, 2018; 26(4): 210-212.
13. Kloeters O, Unglaub F, de Laat E, van Abeelen M, Ulrich D. Prospective and randomised evaluation of the protease-modulating effect of oxidised regenerated cellulose/collagen matrix treatment in pressure sore ulcers. *International Wound Journal*, 2016; 13(6): 1231-1236.
14. Caravaggi C, Grigoletto F, Scuderi N. Wound bed preparation with a dermal substitute (Hyalomatrix (R) PA) facilitates re-epithelialization and healing: Results of a multicenter, prospective, observational study on complex chronic ulcers (The FAST study). *Wounds*, 2011; 23(8): 228-235.
15. Karr J. Utilization of living bilayered cell therapy (Apligraf) for heel ulcers. *Adv Skin Wound Care*, 2008; 21(6): 270-274.
16. Dehghani M, Azarpira N, Mohammadkarimi V, Mossayebi H, Esfandiari E. Grafting with cryopreserved amniotic membrane versus conservative wound care in treatment of pressure ulcers: A randomized clinical trial. *Bull Emerg Trauma*, 2017; 5(4): 249-258.

GROWTH FACTORS FOR TREATMENT OF PRESSURE INJURIES

Introduction

The role of growth factors in the cellular and biochemical events that occur during wound healing includes regulation of cell proliferation and differentiation. Different types of growth factors have different actions in the wound healing process. Growth factors commonly stimulate the proliferation of neutrophils, macrophages and keratinocytes, all of which are active in different stages of wound healing.¹ Development of exogenous growth factor products for wound management is based on expectations that appropriately timed application of these preparations to the wound bed will increase healing rates. Five families of growth factor have been identified as having significance in wound healing processes:^{1,2}

- Epidermal growth factor
- Transforming growth factor beta
- Platelet derived growth factor
- Insulin-like growth factor
- Fibroblast growth factor.

Clinical Question

The clinical question that guided the development of this chapter was:

- What growth factors are effective for supporting healing of pressure injuries?

Platelet Rich Plasma

Platelet-rich plasma (PRP) is also known as platelet-enriched plasma, platelet-rich concentrate, autogenous platelet gel, or platelet releasate. Platelet rich plasma is autologous blood that has a high platelet concentration and a high concentration of growth factors.² Platelet-rich plasma is used by placing supraphysiologic concentrations of autologous platelets at the site of tissue damage. To create the PRP, blood is drawn from the individual and centrifuged to separate the red and white blood cells, creating a plasma that has high concentrations of platelets.^{3,4} Additional centrifugation is usually conducted to concentrate the platelets.⁴ Growth factors are then activated from the platelets using one of numerous different chemical processes that promote physical lyses through disruption of cell membranes (platelet agonists).⁴ The resulting plasma gel is applied to the wound.

**16.1: Consider applying platelet-rich plasma for promoting healing in pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↔)**

Evidence Summary

Evidence supporting this recommendation comes from two low quality Level 1 studies^{5,6} that indicate that application of PRP is effective in supporting healing of pressure injuries. Compared to placebo or standard care, Category/Stage II and III pressure injuries completely healed at significantly faster rates after between two and seven weeks of treatment.^{5,6} When two applications of PRP were applied, complete healing rates were over 30% greater compared to standard care.⁵ Low quality level 1 studies^{5,6} and lower level studies⁷⁻¹⁰ showed that applying PRP was also associated with reductions in wound surface area, improvements in tissue type and improvement in scores on the Pressure Ulcer Scale for Healing (PUSH) after two weeks and one month. The improvements in other outcome measures were less substantial than those seen for complete pressure injury healing. Relative risk (RR) of an adverse effect occurring after application of a PRP to any type of wound has been reported as 0.44 (95% confidence interval [CI] 0.05 to 3.85, $p = 0.46$),³ suggesting undesirable effects are probably not substantial. There is no information available on cost-effectiveness; however, PRPs are usually manufactured in laboratory settings and require skilled technicians and specialist resources that are likely to be limited in most clinical settings.

Implementation Considerations

- Platelet rich plasma should be prepared using standardized protocols by health professionals who have received appropriate training⁴ (*Expert opinion*).
- Platelet rich plasma should not be used on infected pressure injuries. (*Expert opinion*).

- Before applying PRP, the wound bed should be prepared using cleansing and debridement⁵ (*Level 1*).
- Optimize the individual's ability to heal by addressing nutritional status, management of co-morbidities and pressure offloading through repositioning and use of appropriate support surfaces (*Expert opinion*).

Evidence Discussion

Preparation of PRP can be done using a range of different protocols, including commercially available kits in some geographic regions.⁴ The randomized controlled trials (RCTs) and non-randomized trials reported below all used autologous blood samples, withdrawn with an anticoagulant (e.g., sodium citrate,^{5,6} or citrate phosphate dextrose adenine^{9,10}) and centrifuged for 8 minutes⁵, 10 minutes,^{9,10} or 15 minutes.⁶ Only one study specifically reported using a second centrifugation in the preparation process.^{9,10} Calcium chloride 10% was used as the activator in all of the RCTs.^{5,6,9,10} It has been proposed that method of PRP preparation, including the volume of whole blood used and the number and speed of centrifugal spins, influences the concentration of the PRP, which may contribute to efficacy of the final product. However, these factors were not reported in sufficient detail in the PRP pressure injury trials⁴ to evaluate.

Because growth factors stimulate different wound healing processes, timing of their application to a wound could be important. The literature provided limited guidance on selecting an appropriate time to apply PRP. The studies reporting PRP use were generally conducted in chronic pressure injuries.^{5-8,10-12} One study focused on pressure injuries that showed clinical signs of local wound infection.⁹ Regimens varied widely, from application only once or twice,⁵ weekly or twice weekly applications,^{7,9,10} three applications over seven weeks,⁶ to daily application.¹²

An RCT (n = 320)⁶ conducted in chronic (more than six months) pressure injuries that compared two different types of PRP reported complete healing rates of approximately 50%. In a group receiving PRP plus gelatin hydrogen, complete wound healing at seven weeks was 51.8%, with 20% completely healed at one week and 30% healed by four weeks. In the comparator group receiving PRP plus collagen ointment, healing rate at seven weeks was 53.75%, with 35% healed at one week and 40% healed at four weeks. This study also reported a safety analysis in which no significant adverse events were attributed to treatment with PRP.⁶ The study did not include a group receiving a placebo or standard wound care (*Level 1*).

In a non-blinded RCT,⁵ different regimens that included PRP were compared to standard treatment (liquid hydrogel dressing) on 124 Category/Stage II and III pressure injuries. After cleansing and debridement, PRP gel was applied either once on day zero or on day zero and day 15 before application of the standard treatment. A third experimental group received PRP in combination with hyaluronic acid and standard treatment. Compared to standard treatment (0%), there were significant improvements in percent of pressure injuries that reached complete healing by day 36 in the pressure injuries receiving PRP on day zero (8%, p = 0.023), those receiving two applications of PRP (32%, p = 0.001) and for PRP plus hyaluronic acid (37.5%, p ≤ 0.001). All the experimental groups also showed significantly better percent reduction in wound surface area, and no adverse events were experienced⁵ (*Level 1*).

The effectiveness of a PRP dressing in reducing infection and promoting healing was compared to a saline gauze dressing.^{9,10} Participants (n = 25) in the study had spinal cord injury (SCI) and at least two Category/Stage II to IV pressure injuries. All pressure injuries in the PRP experimental group were classified as Category/Stage IV, while those in the control group were Category/Stage II to IV pressure injuries. Dressings were changed twice weekly, with a weekly wound evaluation for five weeks.^{9,10} At five weeks, the pressure injuries treated with PRP were less likely to be critically colonized, as measured by wound swab with culture (24% versus 76%, p = 0.006).⁹ The pressure injuries treated with PRP showed a mean decrease in wound surface area compared to baseline (p < 0.001) but those in the control group had no significant change.¹⁰ Overall, 96% of PRP-treated pressure injuries were considered to have improved status, compared to 68% of control group¹⁰ (*Level 2*).

Platelet-rich plasma gel was also used in two case series^{7,8} that both demonstrated improvements in wound surface area and wound volume, and reduction in undermining/sinus tracts after two to three weeks of treatment (*Level 4*). A third case series reported the use of PRP applied directly to Category/Stage III pressure injury fistulas immediately prior to surgical closure of the ulcer. Imaging confirmed complete closure of fistulas at three weeks¹¹ (*Level 4*).

Although the studies reported above did not provide an indication of the cost of treatment or any economic analyses, commercial PRP kits are considered to be costly;⁴ preparation time is much longer than other dressings and topical agents; and specific training is required to prepare and administer PRP. These factors are likely to reduce the feasibility of using PRP in many clinical settings.

Recombinant Platelet-Derived Growth Factor

Recombinant deoxyribonucleic acid (DNA) technology has been used to produce a recombinant human platelet-derived growth factor (rPDGF, rPDGF-BB, or rhPDGF-BB). Platelet-derived growth factor is available as a commercial topical wound product, becaplermin gel.

16.2: Consider applying platelet-derived growth factor for promoting healing in Category/Stage III and IV pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

A high quality Level 1 study showed 23% more Category/Stage III and IV pressure injuries reached complete healing with application of PDGF gel.¹² Low quality Level 1 studies provided evidence for significantly greater reduction in pressure injury depth associated with PDGF gel,^{13,14} although results were mixed for measures of wound volume.¹³⁻¹⁵ One high quality economic analysis¹⁶ based on clinical outcomes from a high quality Level 1 study estimated that treatment of one pressure injury required approximately three tubes of PDGF gel at a cost of \$920/tube. Over 12 months, individuals would need to pay \$298 USD to gain one additional pressure-injury free week with PDGF gel compared to placebo.¹⁶

Implementation Considerations

- Prepare the wound bed with debridement and ensure the pressure injury is infection-free before application^{13,14} (*Level 1*).
- Optimize the individual's ability to heal by addressing nutritional status, management of co-morbidities and pressure offloading through repositioning and use of appropriate support surfaces (*Expert opinion*).

Evidence Discussion

Three clinical studies reported significantly improved healing of pressure injuries treated with PDGF.¹²⁻¹⁵ All the trials are small studies conducted over 20 years ago; none provide a comparison between PDGF and contemporary wound dressings. More research has been conducted in wounds of other etiologies.

In a multi-centered, double blinded RCT conducted by Rees et. al.¹² participants (n = 124) with Category/Stage III or IV pressure injuries were treated with becaplermin gel (rPDGF) in doses of either 100 µg/g or 300 µg/g. Two control groups were treated with a placebo gel (either daily or twice daily). Pressure injuries treated with rPDGF were more likely to achieve complete healing compared with those treated with placebo gel (placebo gel 0%; 100 µg/g daily 23%, p = 0.005; 300 µg/g daily 19%, p = 0.008). Significant findings were also achieved for other wound healing end points including median relative pressure injury volume (*Level 1*). A secondary economic analysis of PDGF gels used data from two of the randomized groups in the Rees et al. (1999) study¹² (becaplermin gel 100 µg/g daily and placebo daily). The analysis considered direct treatment costs including gel, saline gauze, nursing time and physician reimbursements, all calculated in US dollars in 2016. The study found that actual treatments costs were \$3,827 USD for becaplermin gel and \$1,297 USD for placebo gel. A Markov model that considered the state of having a pressure injury or being completely healed or 90% healed over 52 weeks showed that the cost of one additional week with a healed pressure injury was \$298 USD, and the cost of one additional week with a 90% healed pressure injury was \$150 USD.¹⁶

Another double blind RCT (n = 20) demonstrated superiority of rPDGF-BB 100 µg/g in achieving reduction in wound depth at 29 day follow up for Category/Stage III and IV pressure injuries of up to 67 months duration compared with a placebo gel (14.1 ± 7.4% of day 0 depth versus 34.9 ± 6.7% of day 0 depth, p ≤ 0.05). However, the findings were not significant for reduction in wound volume (*Level 1*).^{13,14}

Mustoe et. al.¹⁵ compared 300 µg/ml aqueous rPDGF-BB (n = 12); 100 µg/ml aqueous rPDGF-BB (n = 15) and saline-soaked gauze dressings (n = 14) in a multi-center RCT. The rPDGF-BB was associated with reduction in wound volume after 29 days compared with saline dressings (p = 0.056). However, this study was small and had a high participant drop out (n = 11) that may have influenced the findings (*Level 1*). In a secondary analysis¹⁷ of a subset of the participants in this trial (n = 20), laboratory analyses demonstrated a significant increase in fibroblast content in pressure injuries treated with rPDGF-BB compared with placebo (2.81 ± 0.17 versus 2.05 ± 0.24, p < 0.01) The process for selection of participants for this secondary analysis was not reported.

Other Growth Factors

Some studies are available on other types of growth factors for treating pressure injuries. For example, one RCT (n = 61)¹⁸ evaluated the effectiveness of a range of growth factor treatments including granulocyte-macrophage colony-stimulating factor (GM-CSF) alone, basic fibroblast growth factor (bFGF) alone and sequential GM-CSF/bFGF for treating Category/Stage III or IV pressure injuries and reported favorable, statistically significant findings for some measures of wound healing (e.g., at least an 85% reduction in wound volume)¹⁸ (*Level 1*). Another small RCT (n = 26)¹⁹ investigated interleukin-1 beta (IL-1β) in three doses for healing pressure injuries but found no significant difference compared with placebo in reducing wound volume (*Level 1*).¹⁹

The volume of evidence on other growth factors for healing pressure injuries is insufficient to make any specific recommendations. Growth factors have more research supporting their use in wound types other than pressure injuries.

References

1. Traversa B, Sussman G. The role of growth factors, cytokines and proteases in wound management. *Primary Intention*, 2001; 9(4): 161-7.
2. Lubkowska A, Dolegowska B, Banfi G. Growth factor content in PRP and their applicability in medicine. *J Biol Regul Homeost Agents*, 2012 26(2 Suppl 1): 35-225.
3. Martinez-Zapata MJ, Martí-Carvajal AJ, Solà I, Expósito JA, Bolibar I, Rodríguez L, Garcia J, Zaror C. Autologous platelet-rich plasma for treating chronic wounds. *Cochrane Database Syst Rev*, 2016; 5(DOI: 10.1002/14651858.CD006899.pub3).
4. Dhurat R, Sukesh M. Principles and methods of preparation of platelet-rich plasma: A review and author's perspective. *J Cutan Aesthet Surg*, 2014; 7(4): 189-197.
5. Ramos-Torrecillas J, Garcia-Martinez O, Luna-Bertos ED, Ocana-Peinado FM, Ruiz C. Effectiveness of platelet-rich plasma and hyaluronic acid for the treatment and care of pressure ulcers. *Biological Research in Nursing*, 2015; 17(2).
6. Yu Q, Han FJ, Lv DS. To compare the healing of pressure sores by the use of combination therapy with platelet rich plasma and gelatin hydrogel versus platelet rich plasma and collagen. *Biomed Res (India)*, 2017; 28(3): 1216-1222.
7. Frykberg RG, Driver VR, Carman D, Lucero B, Borris-Hale C, Fylling CP, Rappl LM, Clausen PA. Chronic wounds treated with a physiologically relevant concentration of platelet-rich plasma gel: a prospective case series. *Ostomy Wound Manage*, 2010; 56(6): 36.
8. Rappl LM. Effect of platelet rich plasma gel in a physiologically relevant platelet concentration on wounds in persons with spinal cord injury. *Int Wound J*, 2011; 8(2): 187-195.
9. Singh R, Dhayal RK, Sehgal PK, Rohilla RK. To evaluate antimicrobial properties of platelet rich plasma and source of colonization in pressure ulcers in spinal injury patients. *Ulcers*, 2015; 2015(749585).
10. Singh R, Rohilla RK, Dhayal RK, Sen R, Sehgal PK. Role of local application of autologous platelet-rich plasma in the management of pressure ulcers in spinal cord injury patients. *Spinal Cord*, 2014; 52(11): 809-16.
11. Biglari B, Reitzel T, Swing T, Buchler A, Gerner HJ, Schmidmaier G, Moghaddam A. A pilot study on the effectiveness of platelet-rich plasma and debridement for the treatment of nonhealing fistulas in spinal cord-injured patients. *Adv Skin Wound Care*, 2015; 28(3): 123-128.
12. Rees RS, Robson MC, Smiell JM, Perry BH. Becaplermin gel in the treatment of pressure ulcers: A phase II randomized, double-blind, placebo-controlled study. *Wound Repair Regen*, 1999; 7(3): 141-147.
13. Robson MC, Phillips LG, Thomason A, Altrock BW, Pence PC, Hegggers JP, Johnston AF, McHugh TP, Anthony MS, Robson LE, Odom LL, Yanagihara D, Pierce GF. Recombinant human platelet-derived growth factor-BB for the treatment of chronic pressure ulcers. *Ann Plast Surg*, 1992a; 29(3): 193-201.
14. Robson MC, Phillips LG, Thomason A, Robson LE, Pierce GF. Platelet-derived growth factor BB for the treatment of chronic pressure ulcers. *Lancet*, 1992b; 339(8784): 23-5.
15. Mustoe TA, Cutler NR, Allman RM, Goode PS, Deuel TF, Prause JA, Bear M, Serdar CM, Pierce GF. A phase II study to evaluate recombinant platelet-derived growth factor-BB in the treatment of stage 3 and 4 pressure ulcers. *Arch Surg*, 1994; 129(2): 213-219.
16. Gilligan AM, Waycaster CR, Milne CT. Cost effectiveness of becaplermin gel on wound closure for the treatment of pressure injuries. *Wounds*, 2018; 30(6): 197-204.
17. Pierce G, Tarpley JE, Allman RM, Goode PS, Serdar CM, Morris B, Mustoe TA, Vande Berg J. Tissue repair processes in healing chronic pressure ulcers treated with recombinant platelet-derived growth factor BB. *Am J Pathol*, 1994; 145(6): 1399-410.
18. Robson MC, Hill DP, Smith PD, Wang X, Meyer-Siegler K, Ko F, VandeBerg JS, Payne WG, Ochs D, Robson LE. Sequential cytokine therapy for pressure ulcers: clinical and mechanistic response. *Ann Surg*, 2000; 231(4): 600-611.
19. Robson MC, A. , Abdullah A, Burns BF, Phillips LG, Garrison L, Cowan W, Hill D, Vandeberg J, Robson LE, Scheeler S. Safety and effect of topical recombinant human interleukin-1beta in the management of pressure sores. *Wound Repair Regen*, 1994; 2(3): 177-81.

BIOPHYSICAL AGENTS

Introduction

Biophysical agents deliver specific treatment to the wound bed, including oxygen via positive (hyperbaric or hyperatmospheric) pressure, electromagnetic, acoustic waves and mechanical energy.

Common Forms of Biophysical Agents

Table 21.1: Forms of biophysical agents

Category	Biophysical Agents
Electromagnetic Spectrum	Electrical stimulation (ES)
	Electromagnetic fields (EMF)
	Pulsed radio frequency energy (PRFE)
	Phototherapy: laser, infrared, ultraviolet, light emitting diode
Acoustic	Non-contact low frequency ultrasound (NC-LFUS) KHz
	Low frequency ultrasound (LFUS) KHz
	High frequency ultrasound (HFUS) MHz
Mechanical/ Kinetic	Subatmospheric: negative pressure wound therapy, suction, tension
	Kinetic: whirlpool, pulsatile lavage, vibration
	Atmospheric: hyperbaric oxygen therapy topical oxygen

The electromagnetic spectrum (EMS) is an energy source that affects living systems. The EMS comprises infrared (thermal radiation), ultraviolet light (invisible light), laser (coherent and monochromatic light) and electrical/electromagnetic stimulation. The various modalities of EMS energy differ from each other only in their wavelength or frequency, and often overlap with adjacent areas of the EMS.

Electrical and magnetic fields are two component properties of electromagnetic radiation that travel perpendicular to each other and are always present together. Properties of these two fields may be altered by the device design so that one is dominant. *In vitro* studies^{1,2} indicate that electrical stimulation (ES) and electromagnetic fields (EMF) induce similar physiological responses that are important for wound healing; however, there are sufficient distinctions between the two to categorize and evaluate them independently.

Other forms of biophysical energy that have been studied in the management of pressure injuries include acoustic, mechanical, and kinetic energy. Some delivery devices provide more than one form of biophysical energy. For example, megahertz (MHz) and kilohertz (kHz) ultrasound devices respectively transmit high and low frequency acoustic (sound) waves and kinetic energy (pressure waves).

Negative pressure wound therapy is a commonly used wound treatment through which negative pressure (a vacuum) is applied to the wound bed through a dressing in a closed environment,³ promoting removal of third space edema.

If used, biophysical energies should be delivered using medical devices that meet local technical and legal requirements and as appropriate to the individual's health and wound condition. Use of biophysical agents should be directed by and under the supervision/management of an appropriately licensed health professional educated and trained in safe and effective selection, application, and monitoring methods.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- Is electrical stimulation an effective intervention for treating pressure injuries?
- Is electromagnetic field therapy an effective intervention for treating pressure injuries?
- Is pulsed radio frequency energy an effective intervention for treating pressure injuries?
- Is phototherapy an effective intervention for treating pressure injuries?
- Is ultrasound therapy (low frequency, high frequency, non-touch) an effective intervention for treating pressure injuries?

- Is subatmospheric therapy (e.g. negative pressure wound therapy, suction, tension) an effective intervention for treating pressure injuries?
- Is kinetic therapy (e.g. whirlpool, pulsatile lavage, vibration) an effective intervention for treating pressure injuries?
- Is atmospheric therapy (e.g. hyperbaric oxygen therapy, topical oxygen therapy) an effective intervention treating pressure injuries?
- Are there other biophysical agents that are effective for treating pressure injuries?
- Are any biophysical agents effective for preventing pressure injuries?

Electrical Stimulation

Direct and Pulsed Current Electrical Stimulation

Electrical stimulation (ES) for treatment of wounds utilizes subsensory amplitudes of direct current (DC) and pulsed current (PC), or sensory ES at a threshold below muscle contraction. Sensory ES utilizes high-voltage monophasic pulsed current (HVMPC), low-voltage monophasic pulsed current (LVMPC), or low-voltage biphasic pulsed current (LVBPC).^{4,5}

Direct current is the continuous, unidirectional flow of charged particles. Direct current ES is applied at a subsensory intensity of not more than 1 mA (usually 20 μ A to 600 μ A). Although an early trial⁶ on DC ES at a subsensory level noted a positive effect on healing of Category/Stage III and IV pressure injuries, later trials failed to show an effect or produced ambiguous results.⁷⁻⁹ Currently there is no scientific evidence to recommend ES at a subsensory level for treatment of pressure injuries and contemporary research has focused on potential healing benefits achieved using PC ES at a sensory intensity.

Monophasic and Biphasic Current Electrical Stimulation

Pulsed current is the brief unidirectional (monophasic pulsed current) or bidirectional (biphasic pulsed current) flow of electrons or ions in which each pulse is separated by a period with no current flow. A monophasic pulse represents a very brief movement of electric particles away from the isoelectric line, returning to the zero line after a finite period, which makes up the duration of the pulse. Monophasic pulsed current waveforms include the rectangular or square waveforms of LVMPC and the twin-peaked waveforms of HVMPC. The duration of low-voltage and high-voltage pulses used in wound treatment are very short (usually between 50 μ s and 200 μ s), and they do not cause pH changes that could be harmful to the tissues.

The biphasic PC waveform is bidirectional and consists of two phases. One phase leaves the isoelectric line, and on its return to baseline the second phase leaves the isoelectric line in the opposite direction. The biphasic waveforms may be asymmetric or symmetric about the isoelectric line. When applying biphasic current, the risk of electrochemical tissue damage is low due to a lack of electrochemical changes in the tissues.

Anodal and Cathodal Electrical Stimulation

Electrodes (anode and cathode) are the conductive elements of an electrical circuit that deliver ES to wound tissues. The anode is a positive electrode that attracts negatively polarized electrical particles (anions), creating an acid environment. The cathode is a negative electrode into which positively charged electric particles (cations) flow, forming an alkaline environment. When using monophasic current ES to treat a pressure injury, the treatment electrode is placed on the wound surface and the electrode closing the electrical circuit is applied to intact skin at least 15 cm from the wound edge.

Recent evidence indicates that there is no statistically significant difference in healing outcomes between cathode-only or cathode-anode ES,⁴ therefore selection of the therapeutic electrode is based on the phase of wound healing and the goals of wound care. Studies show that electrical currents increase the synthesis of nitric oxide in the endothelium of blood vessels, which leads to increased blood flow. Electrical current can also inhibit the activity of pro-inflammatory cytokines and increase the synthesis of anti-inflammatory cytokines and growth factors. Monophasic electric current can stimulate the migration of cells important in the healing process. Anodal ES facilitates cells involved in the inflammatory phase of wound healing, while cathodal ES promotes cellular proliferation.^{4,5} Both anode and cathode can stimulate cellular processes that enhance growth of blood vessels.

17.1: Administer pulsed current electrical stimulation to facilitate wound healing in recalcitrant Category/Stage II pressure injuries and Category/Stage III or IV pressure injuries.
(Strength of Evidence = A; Strength of Recommendation = \uparrow)

Evidence Summary

There was consistent evidence from eight Level 1 studies of high quality^{4,5,10} moderate quality¹¹⁻¹³ and low quality^{14,15} that application of electrical stimulation to Category/Stage II to IV pressure injuries for between two weeks and eight weeks is associated with a greater reduction in wound surface area than either sham therapy^{4,5,15} or standard wound care.¹⁰⁻¹³ Studies reported relative wound surface area reduction of 25% to 82%^{4,5,10-13,15} greater with electrical stimulation regimens than with comparator treatments. One high quality Level 1 study¹⁰ showed that statistically significantly more pressure injuries healed after six weeks of electrical stimulation compared with standard wound care. Two small, low quality Level 1 studies^{15,16} provided evidence that 100% of Category/Stage II to IV pressure injuries treated with high voltage electrical stimulation were able to completely heal in 20 days¹⁵ and in seven weeks.¹⁶ A low quality Level 3 study¹⁷ reported a 23% complete healing rate for Category/Stage II to IV pressure injuries treated for between two and four weeks. Three moderate¹⁸ and low quality^{16,19} Level 1 studies reported statistically significantly faster wound healing rates associated with electrical stimulation compared with sham treatment^{16,18} or standard wound care.¹⁹ No adverse events were reported in the studies. The reported regimens varied with respect to characteristics of the electrical stimulation, but generally administered using high voltage monophasic electrical current,^{4,5,10-17} for between 30 minutes to two hours daily (generally one hour daily), generally for five days per week for up to eight weeks.^{4,5,10-20} The treatment was usually administered by physical therapists, physiotherapists or trained researchers in a range of inpatient and outpatient settings.

Implementation Considerations

- Electrical stimulation may not be the first-line therapy depending on the clinical care setting (*Expert opinion*).
- Use of electrical stimulation should be directed by and under the supervision/management of an appropriately licensed health professional who has been educated and trained in safe and effective selection, application, and monitoring methods (*Expert opinion*).

Evidence Discussion

While some earlier studies report on LVBPC,^{19,21} the majority of studies on ES for improving pressure injury healing report the effectiveness of HVMP. ^{4,5,10-17} The highest quality evidence comes from two recent RCTs^{4,5} comparing ES to sham/placebo ES for promoting healing in older adults with Category/Stage II to III⁵ (or to IV⁴) pressure injuries. In both studies, the ES regimen consisted of HVMP (100 pps, 154 μ s, 0.25 V) delivered for 50 minutes/day, five days/week. Placebo ES was delivered on the same regimen. In one of the trials,⁴ two treatment groups received the ES, one group receiving cathode-only therapy and the second group receiving a combination of cathode-anode ES. Across both trials, all groups receiving HVMP demonstrated statistically significantly greater reductions in wound surface area compared to placebo ($p < 0.05$).^{4,5} In one trial, reduction in wound surface area in pressure injuries receiving ES was over double that in the placebo at week three of treatment (45% versus 20.32% reduction in wound surface area, $p < 0.032$).⁵ The second study supported these findings, with wound surface area reduction reported at six weeks being superior in wounds received ES (82.34% cathode only group, 70.77% cathod-anode group versus 40.53% placebo group, $p < 0.05$ for treatment groups versus placebo).⁴ Neither of these studies followed pressure injuries to complete healing; however, ES is generally used as an adjunct therapy with a primary goal of promoting faster reductions in wound size in the early stages of healing (*Level 1*).

Another recent, small RCT¹⁴ ($n = 35$) compared HVMP ES (100 pps, 100 μ s, 50 – 100 V) delivered for 60 minutes three times per week to 3 MHz high frequency ultrasound for treating Category/Stage II to IV pressure injuries. After four to 12 weeks of treatment, there was a 63% reduction in wound surface area for the ES group ($p < 0.001$ compared to baseline) compared to a 43% reduction in the ultrasound group, which was not a statistically significant difference. Limitations that may have contributed to the lack of significant difference between the two interventions included the pressure injuries in the ES group being significantly more severe with respect to size and Category/Stage¹⁴ (*Level 1*). However, other evidence suggests that ultrasound at 3 MHz may have insufficient penetration of tissues to deliver optimal therapeutic effect.^{22,23}

Franek et. al.¹² conducted an RCT investigating ES compared with standard wound care for treating Category/Stage II and III pressure injuries that had persisted for two to three months ($n = 50$). In the ES group ($n = 26$), participants received standard wound care, preventive practices, and HVMP ES (100 pps, 100 μ s, 100 V). The ES was applied for 50 minutes per day, five days a week. Cathodal stimulation was applied for the first one to two weeks, after which anodal stimulation was used. The comparator group received preventive care and standard wound care only. After six weeks, mean wound surface area decreased significantly in both groups ($p < 0.001$ in both groups). Granulation tissue increased compared with baseline in both groups, but the difference was statistically significant only in the ES group ($p = 0.0006$). The mean decrease in surface area was 88.9% in the ES group and 44.4% in the control group ($p < 0.001$).¹² The limitations of this study included a lack of blinding, and the treatments provided as standard wound care may not have been consistent (*Level 1*).

The results of another RCT (n=29) by Franek et. al.¹³ also showed significant progress in healing of Category/Stage I to III pressure injuries treated with HVMP (100 pps, 100 μ s, 100 V, 50 minutes/day, five days/week). All participants received regular repositioning and topical wound treatment (local bath of potassium permanganate, compression of fibrolan, colistin and moist saline dressings). After six weeks, the mean wound surface area decreased significantly compared to baseline in both groups (85.38% reduction, $p \leq 0.001$ in ES group and 40.08% reduction, $p = 0.002$ in control group). The difference between groups was significant ($p \leq 0.001$)¹³ (Level 1).

An RCT⁶ (n = 34) was conducted with individuals with spinal cord injury (SCI) who had Category/Stage II to IV pressure injuries compared ES to standard wound care. In the ES group (n = 16), HVMP (50 μ s, 50 to 150 V) was applied for 20 minutes at 100 pps followed by 20 minutes at 10 pps and then 20 minutes off-cycle for eight hours each day, for at least three months or until complete healing. The polarity of the treatment electrode was initially negative and then polarity was alternated weekly. All Category/Stage II pressure injuries closed in both groups. In the ES group, 33.3% of Category/Stage III to IV pressure injuries closed compared with 7.1% in the control group ($p = 0.550$). The average decrease in wound surface area at conclusion of treatment was statistically significantly greater in the ES group compared to the standard wound care group (70.0% versus 36%; $p = 0.048$). A major achievement of this study was its finding that ES can be effectively delivered in the community or at home, without direct oversight by health professionals, with ES applied for approximately 5.3 hours per day, typically overnight. However, the study had limitations. Application of the therapeutic ES protocol was inconsistent because wound care therapies were individualized and therefore varied, and only the ES group received silver dressings¹¹ (Level 1).

Ultrasound Therapy

Ultrasound is an acoustic therapy in which mechanical vibration is transmitted in a wave formation at frequencies beyond the upper limit of human hearing. Units of measure for ultrasound are called hertz (Hz). This vibratory property of the ultrasound waves affects the tissue cells. Different frequencies are used therapeutically to assess and to treat soft tissues, with therapeutic HFUS delivered at between 0.5 to 3 MHz, and LFUS typically between 20 to 50 kHz. Non-contact low frequency ultrasound uses acoustic waveforms at low frequencies to transmit energy into the skin and tissues through atomized saline, without contact with the wound or tissues. The transmitted energy is reported to create bubbles in cell fluids, thereby promoting interstitial movement through the cell membrane that is thought to promote healing activities at a cellular level.²⁴

Non-Contact Low Frequency Ultrasound Therapy

17.2: Consider using non-contact low frequency ultrasound therapy as an adjunct therapy to facilitate healing in Category/Stage III and IV pressure injuries and suspected deep tissue injuries.
(Strength of Evidence = B2; Strength of Recommendation = \leftrightarrow)

Evidence Summary

The available evidence is from small studies at high risk of bias. One low quality Level 3 study²⁵ and one low quality Level 4 study²⁴ provided evidence that treatment with NCLFUS therapy is associated with complete resolution of 18%²⁵ to 23%²⁴ of deep tissue injuries. Three low quality Level 3 and Level 4 studies provided evidence for an association between NCLFUS therapy and reduction in wound surface area. Two low quality Level 4 studies^{24,26} reported that two weeks of treatment with NCLFUS therapy was between 26% reduction²⁶ and 41.4% reduction²⁴ in the mean deep tissue injury or Category III pressure injury surface. A high quality Level 3 study²⁷ also demonstrated a significantly greater reduction in deep tissue surface area associated with NCLFUS therapy compared with standard treatment. No adverse events were reported and no studies reported comparisons of different NCLFUS therapy regimens.

Implementation Considerations

- Noncontact low frequency ultrasound is not recommended for use near prostheses or electronic implanted devices (e.g., cardiac pacemakers), over the lower back or uterus in pregnant women, over areas of malignancy, or on the face/head²⁵ (Level 3).
- Use of NCLFUS should be directed by and under the supervision/management of an appropriately licensed health professional who has been educated and trained in safe and effective selection, application, and monitoring methods (Expert opinion).

Evidence Discussion

The body of evidence on the efficacy of NCLFUS is expanding with its increasing use in clinical practice. The research on its use for treating pressure injuries is in increasingly larger sample sizes and adds to the larger body of evidence

on use of NCLUS therapy that includes studies in other wound etiologies, particularly diabetic foot ulcers. In particular, the body of evidence demonstrating efficacy in treating suspected deep tissue injury (SDTI) is increasing, although current studies provide only a low level of evidence supporting its effectiveness in promoting more rapid healing.

One small observational study²⁶ was conducted in participants (n = 13; n = 11 completed trial) with Category/Stage III pressure injuries that had $>10^5$ bacterial count to determine bacterial reduction associated with NCLFUS. The participants received a wound biopsy at baseline and at two weeks, after six treatments of NCLFUS (mean duration of treatment was four minutes). The per-protocol analysis showed a reduction in mean bacterial burden after two weeks (2×10^7 versus 4×10^7 , p = not reported). The study also reported a 26% reduction in mean wound area (p = not reported) and a 20% reduction mean wound volume (p = not reported)²⁶ (Level 4).

The growing evidence regarding the effect of NCLFUS therapy in treating SDTI consists of three cohort studies.^{24,25,27} The first retrospective cohort study²⁵ (n = 85 participants with 127 SDTIs) investigated the effect of NCLFUS therapy administered daily for five days then every other day (mean number of treatments = 10) compared with standard treatment. A non-validated assessment tool was applied retrospectively to photographs of wounds to assess total surface area, skin integrity and tissue color. Scores for individual areas of assessment were combined to give a severity score. The tissue injuries were not comparable at baseline, with the control group having a larger mean total surface area (p = not reported). There was no difference in scores on the severity scale (p < 0.913). The NCLFUS therapy group achieved significant reduction in severity score at follow up compared to the control group (t = 5.67, p < 0.0001); however, the study was insufficiently powered. In the treated participants, 18% of SDTI spontaneously resolved, compared with 2% of participants who received no NCLFUS²⁵ (Level 3).

Honaker et al. (2016)²⁷ followed this study with a second comparison of NCLFUS therapy compared to standard care in two cohorts each of 30 individuals with SDTI. Suspected deep tissue injuries treated with NCLFUS therapy showed a mean decrease in surface area of 8.8 cm² compared to a mean decrease in surface area of 0.3 cm² in the standard care group²⁷ (Level 3).

A retrospective observational study²⁴ was performed to determine the impact of non-contact low-frequency ultrasound (NCLFU) on SDTI (both hospital acquired, and those present on admission to the hospital). In this study, which included review of 44 adults, all SDTIs demonstrated a statistically significant improvement in surface area. This study reported resolution in deep tissue injuries in 23% of all cases, and in 63% of cases where the individual had not been discharged early from the hospital or expired during hospital course.²⁴ There are noted limitations to this study, including the retrospective design, relatively small sample size, and lack of a comparison to standard care (e.g., pressure relief and redistribution, standard wound dressing changes etc). The exclusion of individuals with multiple deep tissue pressure injuries may have skewed the results of this study (Level 3).

High Frequency Ultrasound Therapy

17.3: Consider using high frequency ultrasound therapy at 1 MHz as an adjunct therapy to facilitate healing in Category/Stage III and IV pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↔)

Evidence Summary

Two high quality Level 1 studies^{10,28} provided evidence supporting the use of high frequency ultrasound (HFUS) therapy at 1 MHz frequency reducing wound surface area. In both studies,^{10,28} mean wound surface area reduction was approximately 30% greater with the use of HFUS therapy (1 MHz), compared with standard therapy alone, which was a statistically significant improvement in both studies. In one study,¹⁰ approximately 46% of Category/Stage II to IV pressure injuries completely healed with HFUS therapy (1MHz) for six weeks and in the second high quality Level 1 study²⁸ approximately 38% of Category/Stage II or III pressure injuries completely healed; however, neither of these results was statistically significant compared to standard therapy.

Evidence from three low quality Level 1 studies^{14,29,30} showed that HFUS therapy at 3MHz is associated with statistically significant reductions in wound surface area but other studies showed no statistically significant improvements in wound healing rates³¹ or complete wound healing.^{31,32} Ultrasound waves at 3MHz have shallower tissue penetration compared to ultrasound waves at 1MHz, and may not treat a pressure injury at sufficient tissue depth to achieve clinically significant outcomes.^{22,23}

Implementation Considerations

- High frequency ultrasound is not recommended for use near prostheses or electronic implanted devices (e.g., cardiac pacemakers), or for treating pressure injuries located on the occiput or other areas of the skull (*Expert opinion*).
- In cases of pressure injuries located directly above the bone (up to 1 cm), use HFUS at 3 MHz frequency, in other cases use 1 MHz frequency (*Expert opinion*).
- Use of HFUS should be directed by and under the supervision/management of an appropriately licensed health professional who has been educated and trained in safe and effective selection, application, and monitoring methods (*Expert opinion*).

Evidence Discussion

The evidence for HFUS is somewhat mixed, but when reviewed by frequency, there is a small body of evidence supporting the use of ultrasound at 1 MHz. Results from some studies failed to demonstrate consistent and statistically significant improvements for HFUS treatment for pressure injuries.^{31,32} However, recent hypotheses suggest this may be related to the frequency at which therapy was delivered.¹⁰ The 3 MHz US wave is short and only penetrates the tissues at shallow depths, and is therefore appropriate for treating surface tissues at a depth of 1cm to 1.5 cm. The 1 MHz US wave is longer and therefore penetrates tissue at depths above 1cm, suggesting it may be more appropriate for treating pressure injuries. Studies confirm that even in the case of clinically superficial pressure injuries (i.e., Category/Stage I and II), deeper tissues may be injured.²³ Therefore, the ultrasonic wave with the frequency of 1 MHz may be more beneficial in the treatment of PUs than the wave with a frequency of 3 MHz.

This hypothesis was supported in two successive RCTs that reported consistent results demonstrating a positive effect of 1MHz HFUS on pressure injury healing.^{10,28} The same methodology of sonotherapy was used in both studies, and in both studies all participants received standard pressure injury care, with the experimental group receiving additional HFUS. An US frequency of 1 MHz was applied (SATP 0.5 W/cm², 20% duty cycle, SATA 0.1 W/cm²), for one to three minutes per cm² to the wound bed and around the pressure injury.^{10,28} In the first study (n = 42),²⁸ the HFUS had a significantly greater change in surface area compared with the control group (68.8 ± 37.23 cm² versus 37.24 ± 57.04 cm², p = 0.047) after six weeks of treatment. In the second study (n = 77),¹⁰ pressure injury healing was compared between the control group, the HFUS and a third group receiving ES (HVMPC, 154 μs, 100 pps, 100 V, 250 μC/sec, 50 minutes / day). At 6 months, pressure injury surface area reduction was 77.48% in the US group, 76.19% in the ES group and 48.87% in the control group. The US and ES groups were not significantly different to each other (p = 0.99), but the results obtained in the US group were statistically significantly higher than in the control group (p = 0.024).

Negative Pressure Wound Therapy

Negative pressure wound therapy has been in use as a wound treatment modality for decades. Categorized as an atmospheric therapy intervention, NPWT has been used as a first-line treatment for wounds that could achieve benefit, and while it did not originate for the treatment of pressure injuries, there is a growing body of evidence that supports its use. There was no evidence available on the use of other atmospheric therapy interventions for treating pressure injuries (e.g., suction or tension).

17.4: Consider negative pressure wound therapy as an early adjunct therapy for reducing the size and depth of Category/Stage III or IV pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Most evidence on NPWT focuses on its effectiveness in reducing the wound size, as this is the primary purpose for applying NPWT. Only a low quality Level 4 study³³ provided evidence on complete wound healing, reporting no difference to standard wound care. Two low quality Level 4 studies had conflicting findings on the association between NPWT and reduction in wound surface area.^{33,34} However, high³⁵ and low^{36,37} quality Level 1 studies provided evidence suggesting NPWT is associated with reduction in pressure injury dimensions, including depth and volume, which was supported lower level studies.^{21,34} Relative reduction in wound depth compared with standard wound care ranged from 22% to 48% after six to nine weeks of treatment.³⁵⁻³⁷ Additional evidence^{21,36,37} suggested NPWT has a role in promoting reduction in slough and increase in epithelialisation. Significant reductions in wound dimensions and improvements in wound characteristics (e.g., tissue type and exudate level) were evident early in treatment, with studies reporting significant effects observable within two to three weeks.^{21,34,36,37} One moderate quality Level 1 study³⁸ reported significantly faster healing of Category/Stage IV pressure injuries when NPWT was implemented, and a low

quality Level 1 study suggested NPWT was associated with a significant reduction in inflammatory markers.³⁶ Adverse events were reported in the literature, including retention of a foam dressing (Level 5), osteomyelitis, calcaneal fractures, arterial bleeding and clinical infection. Some adverse events may be due to improper use of NPWT devices. However, when compared to the rate of adverse events occurring with standard wound care in high³⁵ and moderate³⁸ quality Level 1 studies, NPWT was not associated with an increased risk of adverse events. Most studies reported use of a commercially available NPWT system; some studies reported custom-made systems. In most comparison studies, comparator groups received saline soaked gauze dressings (in one study,³⁸ sodium hypochlorite dressing was the comparison) attended twice or three times daily rather than comparison to contemporary wound dressings. In two limited cost evaluations,^{21,36,37} NPWT was cheaper to deliver than moist gauze dressings. However, use of NPWT requires application by a trained health professional, along with specialized medical equipment that may not be accessible in all clinical or geographic settings.

Implementation Considerations

- If active bleeding develops suddenly or in large amounts during NPWT, or if frank (bright red) blood is seen in the tubing or in the canister stop NPWT (e.g., stop the suction), take measures to stop the bleeding and seek immediate expert advice (*Expert opinion*).
- If the pressure injury is located on the heel or foot, establish the presence of adequate vascular blood supply to the lower limb before commencing NPWT³⁹ (*Expert opinion*).
- Consider anatomical structures and their location when using NPWT (*Expert opinion*).
- Negative pressure wound therapy is not recommended for:
 - malignant wounds
 - use when vital organs or large vascular structures are exposed
 - wounds with no exudate
 - in individuals with untreated osteomyelitis, local or systemic clinical infection^{40,41} (*Expert opinion*).
- Cautious use by an experienced health professional is recommended for individuals on anticoagulant therapy, in actively bleeding wounds, or where the wound is near major blood vessels (*Expert opinion*).
- Debride the pressure injury of necrotic tissue prior to applying NPWT.^{34-38,42} Instillation therapy augments debridement (*Levels 1 and 4*).
- Use the interface dressing recommended by the manufacturer. Gauze interfaces should be used strictly according to manufacturer's guidelines and with equipment designed for use with gauze interfaces (*Expert opinion*).
- Avoid placing wound interface dressings on intact skin³⁵ (*Level 1*).
- Diligently remove the entire tissue interface layer at each wound dressing change to prevent dressing retention^{35,43} (*Levels 1 and 5*).
- Pay attention to positioning of the NPWT tubing due to the risk of medical-device related pressure injuries. The guideline chapter *Device Related Pressure Injuries* provides relevant additional recommendations (*Expert opinion*).
- Optimal negative pressure levels are not well-established, but usually range between 75 and 125 mmHg^{34,44,45} (*Levels 3 and 4*).
- Place the drainage collection system on a level surface (*Expert opinion*).
- Record and document the volume and appearance of exudate in the drainage collection system (*Expert opinion*).
- If pain is anticipated or reported consider:
 - placing a nonadherent interface dressing on the wound bed underneath the foam dressing, and/or
 - lowering the level of pressure, and/or
 - changing the type of pressure (continuous or intermittent) (*Expert opinion*).
- Evaluate the pressure injury with each dressing change to determine response of the wound and appropriate intervals for wound dressing changes (*Expert opinion*).
- Provide the individual and their informal caregivers with instructions for managing the NPWT, especially for those residing in community settings (*Expert opinion*).

Evidence Discussion

Negative pressure wound therapy has its greatest efficacy in reducing wound volume,^{34,35,44} therefore it can serve as an adjuvant therapy when combined with debridement and other treatments that promote healing, such as nutritional support and pressure redistribution. Negative pressure wound therapy promotes wound healing through removal of third space edema⁴⁶ thereby enhancing nutrient and oxygen delivery.⁴⁷ The intervention also reduces wound exudate, which is the medium for bacterial colonization;⁴⁸ promotes granulation tissue⁴⁷ and angiogenesis, and reduces wound inhibitory factors.⁴⁹ Therefore, the intent of NPWT is to facilitate wound closure rather than to fully close or heal a

pressure injury. Due to these goals of therapy, the research on NPWT has focused on the intermediate outcomes of ulcer healing including reduction in wound volume,^{35,44} wound bed preparation for skin grafting or flap closure,⁴⁴ ability to use a surface dressing rather than wound packing,⁴⁴ and rate of healing.³⁸

The results of an observational study by Ho et. al.³³ conducted with individuals with SCI and Category/Stage III pressure injuries (n = 86) are the only evidence reporting complete wound healing as the primary outcome measure. This study did not find a statistically significant difference between the NPWT group and the non-NPWT group for complete healing or for reduction in wound surface area. However, in the NPWT group the non-healing subgroup had significantly lower serum albumin levels than the healing subgroup (2.9 ± 0.4 versus 3.3 ± 0.5 mg/dl, $p < 0.05$), suggesting nutritional status may be important in the effectiveness on NPWT³³ (Level 4).

However, NPWT has been shown to reduce wound dimensions, and particularly the depth of pressure injuries when compared to traditional forms of topical therapies in numerous other studies.^{34,35,44} In an RCT,³⁵ NPWT was compared to wet-to-moist gauze dressings covered with a thin film to simulate closed therapy without suction. Percent change in wound depth in this study was significantly more rapid ($p < 0.00001$) in the NPWT group. In this study, a tissue biopsies also showed more inflammation and fibrosis in the moist gauze dressing group and more granulation tissue in the NPWT group³⁵ (Level 1). An observational study³⁴ in immobilized individuals with Category/Stage IV pressure injuries (n = 10) also reported that mean wound surface area was reduced by 55.1% after seven weeks of treatment with NPWT (Level 4).

A number of other studies also showed NPWT was associated with improvement in pressure injury size and depth. Srivastava et al. (2014)²¹ conducted a controlled trial to compare pressure injury healing with negative pressure to conventional dressings. All Category/Stage III and IV pressure injuries (n = 36) were cleaned and packed with saline gauze, with wound dressings changed twice daily. Participants in intervention group received NPWT for 9 weeks (mean pressure -80 mm Hg, range -60 to -120 mm Hg). Ulcer size and depth decreased significantly ($p = 0.0001$) for pressure injuries treated with NPWT, but there were no statistically significant differences for surface area or depth in pressure injuries receiving a standard wound dressing. Use of NPWT was ineffective in low sacral pressure injuries in which the wound bed involved the area close to the natal cleft because the adhesive dressing could not be properly applied to obtain an airtight seal (Level 2).

Dwivedi's research team (2016, 2017)^{36,37} conducted an RCT exploring the effectiveness of negative pressure devices compared to standard wound dressings for promoting pressure injury closure in individuals with paraplegia. Participants received either standard care consisting of normal saline and sterile gauze packing changed once or twice daily, or NPWT using a sterile foam and transparent film dressing changed weekly. Mean wound exudate level was significantly lower in the NPWT group after the third week of treatment. Conversion of slough into red granulation tissue was significantly higher in NPWT after week six. By week nine, pressure injuries in the NPWT group had achieved 79.7% reduction in wound dimensions. This compared with a 54.7% reduction in wound dimensions in the standard wound dressing comparator group. In this study, conducted in India, nine weeks of NPWT cost \$105 USD compared to \$200 USD for standard care.^{36,37} It appears that NPWT is a reasonable treatment for promoting closure of Category/Stage IV pressure injuries and is cost effective in a low resource setting. However, feasibility of using NPWT will be influenced by the anatomical location of the pressure injury, and ability to secure NPWT in order to attain adequate seal on the wound dressing²¹ (Level 1). Wild et al. (2008)⁴² also investigated reduction in wound area using a commercial NPWT system versus non-commercial NPWT delivered using surgical drain bottles. Findings showed an increase in surface granulation tissue of 54% of pressure injuries receiving a commercial NPWT and a reduction of granulation tissue in the non-commercial NPWT group ($p = 0.001$)⁴² (Level 1).

One of the more notable findings to emerge from a trial conducted by de Laat et. al.³⁸ that investigated the reduction of wound volume using NPWT versus sodium hypochlorite dressings was a 50% reduction in the median treatment time ($p = 0.001$) (Level 1). Wanner et al. (2003)⁵⁰ found no difference in time to reach a 50% decrease in volume between NPWT therapy and either wet-to-dry dressings or wet-to-wet dressings for Category/Stage II or deeper pelvic pressure injuries in 22 individuals with SCI. Mean time to achieve 50% reduction in size was around 27 days for all groups. In this very small trial that lacked statistical power, the NPWT group had significantly larger pressure injuries at baseline,⁵⁰ which might indicate that the treatment was clinically useful in achieving reduction in size more quickly for larger pressure injuries (Level 2).

Negative pressure wound therapy is intended for use in pressure injuries free of necrotic tissue. Therefore, NPWT therapy should begin after debridement. In the research studies reported above, pressure injuries received some form of debridement before application of the NPWT,^{34,36-38} generally sharp^{33,35} or surgical debridement.^{42,44,50}

Clean technique can be used for most NPWT dressing changes. As NPWT is most commonly used in deep wounds, the health professional must be diligent in removing the entire previous tissue interface layer to prevent retained packing. One case study reported a retained foam dressing.⁴³ Fill the defect and dead space with wound dressing and record the number of dressings placed in the wound bed. Most available NPWT wound contact layers are foam or gauze,

and current research has increased our understanding of how the fillers interact with the wound. Use caution to avoid placing wound interface dressings on intact skin. Clear film dressings should cover the wound interface dressing and a 3 to 5 cm border of intact periwound skin. Protect fragile periwound tissue with barrier films or dressings. Position the dressing tubing on flat body surfaces and away from the perineal areas, bony prominences, or pressure areas.²¹

Optimal negative pressure levels are not well-established, but usually range between 75 and 125 mm Hg. Negative pressure wound therapy on intermittent suction settings has been associated with clinical reports of pain. Lower levels of NPWT (75 to 80 mmHg) have been reported to reduce pain without compromising efficacy.³⁴ Nonadherent silicon mesh tissue interface dressings have been used effectively to reduce pain with dressing removal. The use of petrolatum or emulsion based dressings reduces efficacy of wound fluid transfer.

The optimal dressing change interval is not well-established and is ideally based on characteristics of the individual and the wound. Dressing change intervals can range from every 12 hours (wounds with heavy exudate) to twice weekly (wounds with light exudate), with the most common frequency being three times a week. A reduction in the frequency of wound dressing changes compared to standard wound care has been noted in numerous studies,^{36,37,45} including the additional benefit of reduction in pain and discomfort associated with dressing changes⁴⁵ (*Level 2*), as well as reduction of the socioeconomic burden of dressing changes^{36,37} (*Level 1*). If tissue ingrowth into the wound dressing or tubing is noted, using lower pressures may be sufficient to correct this problem.³⁴ It is expected that granulation tissue will appear in the pressure injury; if present, monitor for tissue trauma or pain.

It is also expected that the wound bed will decrease in volume,^{21,36,37} and tunnels and undermining will resolve. If the pressure injury appears clinically infected (e.g., erythema or purulence) or the individual presents with signs of infection (e.g., fever, malaise and/or leukocytosis), NPWT should not be reapplied. The individual and pressure injury need to be fully evaluated with any deterioration (see the guideline chapter *Pressure Injury Assessment and Monitoring of Healing*). If there is no change in wound dimensions (1 cm in any dimension) within two weeks, reassess for continuation of NPWT. If there is no exudate or the wound bed approaches skin level, consider discontinuation of the NPWT.

When monitoring exudate, consider the type and volume of exudate in both the tubing and canister. The exudate color may change from serous to serosanguineous and some sanguineous or bloody drainage may also be noted during NPWT. The change in wound drainage characteristics may be related to disruption of capillary buds of granulation tissue.

Negative pressure wound therapy systems can be used in outpatient or home settings. Provide adequate education so that the individual and his/her informal caregivers know what to do if the wound dressing seal loosens; alarms ring; blood or tissue are seen in the tubing; or local erythema develops. Emergency contacts should be provided.

Other Types of Electromagnetic Spectrum Therapy

Pulsed Electromagnetic Field Therapy

Pulsed, nonthermal, low frequency (usually < 100 Hz) electromagnetic field therapy is the delivery of magnetic field to the wound bed with a goal of delivering therapeutic effect. Although the precise mechanism of the physiological effect of PEMF therapy is unclear, increase in keratinocyte growth, reduction in inflammation, increased collagen, and fibrin deposits in the wound bed are all proposed as possible outcomes.^{51,52}

The evidence on PEMF therapy for treating pressure injuries is at high risk of bias, mode of operation has not been clearly established and there is a lack of recent research in this field, therefore, no recommendation could be made on its use. One small, low quality Level 1 study⁵³ provided evidence that Category/Stage II and III pressure injuries have better rates of complete healing with PEMF therapy compared with sham therapy after up to 12 weeks of treatment. The study indicated that over 40% more Category/Stage II pressure injuries could achieve complete healing with PEMF therapy as compared to sham therapy.⁵³ One small, moderate quality Level 1 study⁵³ indicated that PEMF therapy is associated with larger reduction in Category/Stage II pressure injury surface area than sham treatment after one week of treatment. Another moderate quality Level 1 study⁵⁴ indicated that four different PEMF therapy regimens were associated with statistically significant reductions in Category/Stage II and III pressure injury surface area compared to baseline after four weeks of treatment, with no differences in outcomes associated with any specific PEMF therapy regimen. Evidence for PEMF therapy being associated with greater improvements in wound characteristics compared with sham therapy was provided by two Low quality Level 1 studies^{55,56} In these studies, no adverse events were associated with PEMF therapy, although individuals with potential contraindications, including medical device implants, fever and seizures were excluded from participating.⁵³⁻⁵⁶

Pulsed Radio Frequency Energy Therapy

Pulsed radio frequency energy (PRFE) therapy is a nonthermal, non-invasive, method of delivering electromagnetic energy in pulsed athermal doses to a wound bed to promote healing.^{57,58} Invitro cell studies have demonstrated that waveform energy is associated with optimized fibroblast and epithelial cell proliferation.⁵⁷ Evidence on PRFE therapy is limited to retrospective analyses of wound registries maintained by the product manufacturer that are at high risk of bias, therefore no recommendation can be made on its use. Two low quality Level 4 studies^{57,58} reported a mean/median decrease in wound surface area of around 45 to 50% after four weeks of treatment with pulsed radio frequency energy therapy. The pressure injuries reported in both analyses ranged from 100% healing to increase in area by almost four times.^{57,58} Neither study reported adverse events. Pulsed radio frequency energy therapy was administered either by an individual with a pressure injury or a health professional for two 30-minute sessions each day, with therapy administered through the wound dressing.^{57,58}

Phototherapy

Phototherapy is therapy that involves exposure of the wound to a source of light, including daylight, low level laser therapy (LLLT), other laser therapies, light emitting diodes and ultraviolet light. Although the mechanism are unclear, phototherapy is thought to reduce inflammation, increase lymphatic circulation and increase tissue regeneration.⁵⁹ The evidence on effectiveness of phototherapy (laser, ultraviolet and infrared light therapies) is conflicting and no recommendations can be made on the use of any type of phototherapy. Differences may relate to the type of light therapy used or the regimen implemented. Only one study compared different types of phototherapy and the results from this low quality Level 1 study²⁹ suggested ultraviolet C light may be superior to laser therapy; however, there was a high risk of bias.

One high quality Level 1 study⁶⁰ provided evidence that laser therapy is associated with significantly better rates of complete healing for Category/Stage II and III pressure injuries compared to a placebo therapy. Approximately 30% more pressure injuries achieved complete healing with one month of treatment and approximately 50% more pressure injuries were completely healed at three-month follow-up. A low quality Level 1 study supported this finding.⁶¹ However, three low quality Level 1 studies^{29,61,62} reported that laser therapy was not associated with superior effects compared to standard wound care when the outcome measure was reduction in wound surface area or healing rates. The rate of undesirable outcomes did not significantly differ from standard wound care.⁶²

One high quality Level 1 study⁶³ reported no statistically significant effect in achieving complete wound healing for ultraviolet C light compared to placebo therapy. A low quality Level 1 study⁶⁴ reported slightly a higher healing rate in a group receiving infrared light therapy compared to placebo therapy; however, the approximate 4% difference in complete healing rates did not appear to be clinically significant and statistical significance was not reported. Evidence from small, low quality Level 1^{29,65} and Level 2⁶⁶ studies suggested that ultraviolet B or C light is associated with statistically significant superior effects for reduction in wound surface area and healing rates. Evidence from moderate⁶⁷ and low quality⁶⁴ Level 1 studies provided conflicting evidence on the effectiveness of infrared light for promoting faster wound healing. One study reported adverse events associated with infrared light including tingling, pain, bleeding and skin redness.⁶⁴

Phototherapy requires a trained health professional and is generally conducted once or twice daily for five days per week until the wound heals. This regimen may be inaccessible in many clinical or geographic settings. High attrition was noted in some studies, suggesting that some phototherapy interventions may not be acceptable to individuals or may lack feasibility in some settings.^{29,63,64,67}

Oxygen Therapies

Hyperbaric oxygen therapy (HBOT) is a therapy in which the individual breathes 100% oxygen at pressures greater than normal atmospheric (sea level) pressure or more than 1 atmosphere absolute (ATA). Pressures of up to three times normal atmospheric pressure may be utilized. There was insufficient evidence to make a recommendation on the use of HBOT to treat pressure injuries. A low quality Level 3 study⁶⁸ indicated superior outcomes for complete wound healing and reduction in wound surface area associated with use of a hyperbaric oxygen chamber for two hours per day compared with frequent wound dressings. There was no comparison to contemporary wound dressings. No adverse events were reported. The intervention required trained health professionals delivering therapy for 120 minutes daily using specialized equipment,⁶⁸ which may reduce feasibility in some clinical and geographic settings.

Oxygen-based therapies are hypothesized to stimulate wound healing in hypoxic wounds by improving angiogenesis. Topical oxygen is a therapy in which 100% oxygen is applied directly to the wound, usually at pressures between 22 mm Hg and 50 mm Hg. There was insufficient evidence to make a recommendation on the use of topical oxygen therapy to treat pressure injuries. A moderate quality Level 1 study⁶⁹ indicated that topical oxygen therapy delivered directly to the wound bed with an oxygen catheter for a total of 60 minutes daily over three sessions is associated with

significantly better reductions in wound surface area and higher rates of complete healing compared to saline-soaked gauze dressings. There was no comparison to contemporary wound dressings. No adverse events were reported. The intervention required trained health professionals delivering therapy for 60 minutes daily using specialized equipment,⁶⁹ which may reduce feasibility in some clinical and geographic settings.

Kinetic Therapies

Whirlpool is a form of hydrotherapy in which warm water circulation is used to promote wound cleansing, including removal of necrotic tissue and debris in the wound bed. Either the individual is submerged in a whirlpool bath, or the limb is submerged and the water may or may not be agitated.⁷⁰ Whirlpool has been used in the past for wound cleansing and reducing bacterial bioburden but is seldom used in contemporary wound care. Due to the high risk from adverse events and the low certainty of desired effects, no recommendation can be made regarding whirlpool therapy for the treatment of pressure injuries. One low quality Level 1 study⁷¹ reported that whirlpool therapy for two weeks was associated with faster healing compared to a moist saline wound dressing. This study was at a high risk of bias. Indirect evidence from a review⁷⁰ that included outcomes for research conducted in other types of wounds highlighted the risks of whirlpool therapy including wound infection, cross contamination and increased vascular hypertension and vascular congestion.

Pulsatile lavage is mechanical hydrotherapy that involves delivering normal saline at between 4 and 15 psi (pressurized irrigation) through a mechanical apparatus with a goal of removing wound debris using mechanical energy. Usually suction is used in conjunction with pulsed lavage creating negative pressure that is thought to stimulate granulation. Due to the very small volume of evidence, no recommendation can be made on the use of pulsatile lavage for treating pressure injuries. One moderate quality Level 1 study⁷² suggested that pulsatile lavage for three weeks is associated with faster reductions in wound length, width, depth and volume than sham treatment. However, all confidence intervals included the null value, decreasing confidence in the significance of the findings.

Vibration therapy attempts to use vibration to promote blood flow, thereby improving wound healing from vasodilation. It is proposed that this type of therapy stimulates blood flow due to mechanical stresses of endothelial cells resulting in vasodilation.⁷³ There is insufficient evidence to make a recommendation on the use of vibration therapy to treat pressure injuries. One moderate quality Level 2 study⁷⁴ suggested that vibration therapy for up to seven days might be associated with superior outcomes for wound healing compared to standard wound care alone. The study was at a high risk of bias. There were no adverse events reported. Delivery of vibration therapy was performed three times per day with each session lasting 15 minutes. This regimen may not be feasible in all clinical settings, and access to equipment may be limited in some geographic regions.

Electrical Stimulation for Prevention of Pressure Injuries

A number of studies⁷⁵⁻⁷⁸ have explored the role of ES in preventing pressure injuries in individuals at high risk (primarily with SCI). Electrical stimulation induces intermittent tetanic muscle contractions that redistributes the loading and stiffness of the deformed soft tissues as demonstrated by significant reductions in ischial interface pressure observed in a number of studies. The intervention appears to be practical in daily life and is well tolerated;^{75,76} however, the current evidence is insufficient to make a recommendation.

A number of small clinical experiments,⁷⁵⁻⁷⁸ investigated the effect of ES-induced activation of the gluteal and hamstring muscles on the sitting interface pressure in individuals with SCI, and a smaller number of studies have explored the effect of ES on tissue perfusion and oxygenation^{77,79} and pressure injury incidence.⁸⁰ The majority of studies explored surface application of ES in which stimulation is applied to the gluteal and hamstring muscles, generally through specially designed ES-shorts with built-in electrodes.⁷⁵⁻⁷⁸ or pads applied to the skin.⁸⁰ The current amplitude across studies ranged from 70 to 115 mA, with ES applied for 1 to 3 hours/day with a 50 pps current in an intermittent cycle: 3 min of stimulation (including 1-second on: 1-second off or 1-second on: 4-seconds off) and 17 minutes of rest in most studies.⁷⁵⁻⁷⁸ One study⁷⁹ compared surface ES with sacral nerve root application of ES, reporting the latter to be associated with significantly better tissue perfusion and oxygenation outcomes, although both surface and sacral nerve root ES regimens demonstrated improvements compared to no intervention⁷⁹ (*Level 4*).

The above studies⁷⁵⁻⁷⁸ concluded that ES-induced tetanic contractions of the gluteal and hamstring muscles in sitting individuals with SCI causes a temporary decrease in ischial interface pressure and improved pressure distribution⁷⁵⁻⁷⁸ (all *Level 5*). The evidence on effectiveness in improving tissue perfusion and oxygenation was mixed, with one study reporting no statistically significant effect,⁷⁷ and the other finding both surface and deep nerve root ES improving blood flow⁷⁹ (both *Level 4*). In the single study that reported pressure injury incidence, no pressure injuries were experienced in the study period, although it was unclear if this was associated with the intervention, other preventive care strategies, of the relatively short study duration⁸⁰ (*Level 3*).

References

1. Aaron RK, Boyan BD, Ciombor DM, Schwartz Z, Simon BJ. Stimulation of growth factor synthesis by electric and electromagnetic fields. *Clin Orthop Relat Res*, 2004(419): 30-37.
2. Bassett CA. Low energy pulsing electromagnetic fields modify biomedical processes. *Bioessays*, 1987; 6(1): 36-42.
3. Dumville Jo C, Land L, Evans D, Peinemann F. Negative pressure wound therapy for treating leg ulcers. *Cochrane Database of Systematic Reviews*, 2015; 7(Art No. CD011354).
4. Polak A, Kloth LC, Blaszcak E, Taradaj J, Nawrat-Szoltysik A, Ickowicz T, Hordynska E, Franek A, Kucio C. The efficacy of pressure ulcer treatment with cathodal and cathodal-anodal high-voltage monophasic pulsed current: A prospective, randomized, controlled clinical trial. *Phys Ther*, 2017; 97(8): 777-789.
5. Polak A, Kloth LC, Blaszcak E, Taradaj J, Nawrat-Szoltysik A, Walczak A, Bialek L, Paczula M, Franek A, Kucio C. Evaluation of the healing progress of pressure ulcers treated with cathodal high-voltage monophasic pulsed current: Results of a prospective, double-blind, randomized clinical trial. *Adv Skin Wound Care*, 2016; 29(10): 447-459.
6. Karba R, Šemrov D, Vodovnik L, Benko H, S'avrin R. DC electrical stimulation for chronic wound healing enhancement Part 1. Clinical study and determination of electrical field distribution in the numerical wound model. *Bioelectrochem Bioenerg*, 1997; 43(2): 265-270.
7. Adunsky A, Ohry A, DDCT Group. Decubitus direct current treatment (DDCT) of pressure ulcers: Results of a randomized double-blinded placebo controlled study. *Arch Gerontol Geriatr*, 2005; 41(3): 261-269.
8. Ullah M. A study to detect the efficacy of micro-current electrical therapy on decubitus wound. *J Med Sci*, 2007; 7(8): 1320-1324.
9. Stefanovska A, Vodovnik L, Benko H, Turk R. Treatment of chronic wounds by means of electric and electromagnetic fields. Part 2. Value of FES parameters for pressure sore treatment. *Med Biol Eng Comput*, 1993; 31(3): 213-20.
10. Polak A, Taradaj J, Nawrat-Szoltysik A, Stania M, Dolibog P, Blaszcak E, Zarzeczny R, Juras G, Franek A, Kucio C. Reduction of pressure ulcer size with high-voltage pulsed current and high-frequency ultrasound: A randomised trial. *J Wound Care*, 2016; 25(12): 742-754.
11. Houghton PE, Campbell KE, Fraser CH, Harris C, Keast DH, Potter PJ, Hayes KC, Woodbury MG. Electrical stimulation therapy increases rate of healing of pressure ulcers in community-dwelling people with spinal cord injury. *Arch Phys Med Rehabil*, 2010; 91(5): 669-678.
12. Franek A, Kostur R, Polak A, Taradaj J, Szlachta Z, Blaszcak E, Dolibog P, Dolibog P, Koczy B, Kucio C. Using high-voltage electrical stimulation in the treatment of recalcitrant pressure ulcers: Results of a randomized, controlled clinical study. *Ostomy Wound Manage*, 2012; 58(3): 30-44.
13. Franek A, Kostur R, Taradaj J, Blaszcak E, Szlachta Z, Dolibog P, Dolibog P, Polak A. Effect of high voltage monophasic stimulation on pressure ulcer healing: Results from a randomized controlled trial. *Wounds*, 2011; 23(1): 15-23.
14. Karsli PB, Gurcay E, Karaahmet OZ, Cakci A. High-voltage electrical stimulation versus ultrasound in the treatment of pressure ulcers. *Adv Skin Wound Care*, 2017; 30(12): 565-570.
15. Griffin JW, Tooms RE, Mendius RA, Clifft JK, Vander Zwaag R, el Zeky F. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. *Phys Ther*, 1991; 71(6): 433-42.
16. Kloth LC, Feedar JA. Acceleration of wound healing with high voltage, monophasic, pulsed current. *Phys Ther*, 1988; 68(4): 503-8.
17. Gentzkow GD, Alon G, Taler GA, Eltorai IM, Montroy RE. Healing of refractory Stage III & IV pressure ulcers by a new electrical stimulation device. *Wounds*, 1993; 5(3): 160-171.
18. Wood JM, Evans PE, Schallreuter KU, Jacobson WE, Sufit R, Newman J, White C, Jacobson M. A multicenter study on the use of pulsed low-intensity direct current for healing chronic stage II and stage III decubitus ulcers. *Arch Dermatol*, 1993; 129(8): 999-1009.
19. Jercinovic A, Karba R, Vodovnik L, Stefanovska A, Kroselj P, Turk R, Dzidic I, Benko H, Savrin R. Low frequency pulsed current and pressure ulcer healing. *IEEE Trans Rehabil Eng*, 1994; 2(4): 225-233.
20. Lawson D, Petrofsky J. The Effect of monophasic vs. biphasic current on healing rate and blood flow in people with pressure and neuropathic ulcers. *J Acute Care Phys Ther*, 2013; 4(1): 26-33.
21. Srivastava RN, Dwivedi MK, Bhagat AK, Raj S, Agarwal R, Chandra A. A non-randomised, controlled clinical trial of an innovative device for negative pressure wound therapy of pressure ulcers in traumatic paraplegia patients. *Int Wound J*, 2014.
22. Maeshige N, Fujiwara H, Honda H, Yoshikawa Y. Evaluation of the combined use of ultrasound irradiation and wound dressing on pressure ulcers. *J Wound Care*, 2010; 19: 63-8.
23. Aliano K, Low C, Stavrides S, Luchs J, Davenport T. The correlation between ultrasound findings and clinical assessment of pressure-related ulcers: is the extent of injury greater than what is predicted? *Surg Technol Int*, 2014; 24: 112-6.
24. Wagner-Cox P, Duhamel HM, Jamison CR, Jackson RR, Fehr ST. Use of noncontact low-frequency ultrasound in deep tissue pressure injury: A retrospective analysis. *J Wound Ostomy Continence Nurs*, 2017; 44(4): 336-342.
25. Honaker JS, Forston MR, Davis EA, Wiesner MM, Morgan JA. Effects of non contact low-frequency ultrasound on healing of suspected deep tissue injury: A retrospective analysis. *Int Wound J*, 2013; 10(1): 65-72.
26. Serena T, Lee SK, Lam K, Attar P, Meneses P, Ennis W. The impact of noncontact, nonthermal, low-frequency ultrasound on bacterial counts in experimental and chronic wounds. *Ostomy Wound Manage*, 2009; 55(1): 22-30.
27. Honaker JS, Forston MR, Davis EA, Wiesner MM, Morgan JA, & Sacca E. The effect of adjunctive non-contact low frequency ultrasound on deep tissue pressure injury. *Wound Repair Regen*, 2016.
28. Polak A, Franek A, Blaszcak E, Nawrat-Szoltysik A, Taradaj J, Wiercigroch L, Dolibog P, Stania M, Juras G. A prospective, randomized, controlled, clinical study to evaluate the efficacy of high-frequency ultrasound in the treatment of Stage II and Stage III pressure ulcers in geriatric patients. *Ostomy Wound Manage*, 2014; 60(8): 16-28.

29. Nussbaum EL, Biemann I, Mustard B. Comparison of ultrasound/ultraviolet-C and laser for treatment of pressure ulcers in patients with spinal cord injury. *Phys Ther*, 1994; 74(9): 812-23.
30. Shanmuga RP, Suryanaryana Reddy V, Venkat R, Sachin G, Bhagya SS. A study to evaluate the effectiveness of continuous ultrasound therapy in healing of pressure sores - A prospective randomized clinical trial. *Indian J Physiother Occup Ther*, 2017; 11(3): 136-140.
31. McDiarmid T, Burns PN, Lewith GT, Machin D. Ultrasound and the treatment of pressure sores. *Physiotherapy*, 1985; 71(2): 66-70.
32. ter Riet G, Kessels AG, Knipschild P. A randomized clinical trial of ultrasound in the treatment of pressure ulcers. *Phys Ther*, 1996; 76(12): 1301-11.
33. Ho CH, Powell HL, Collins JF, Bauman WA, Spungen AM. Poor nutrition is a relative contraindication to negative pressure wound therapy for pressure ulcers: preliminary observations in patients with spinal cord injury. *Adv Skin Wound Care*, 2010; 23(11): 508-516.
34. Isago T, Nozaki M, Kikuchi Y, Honda T, Nakazawa H. Negative-pressure dressings in the treatment of pressure ulcers. *J Dermatol*, 2003; 30(4): 299-305.
35. Joseph E, Hamori CA, Bergman S, Roaf E, Swann NF, Anastasi GW. New therapeutic approaches in wound care. A prospective randomized trial of vacuum-assisted closure versus standard therapy of chronic nonhealing wounds. *Wounds*, 2000; 12(3): 60-67.
36. Dwivedi MK, Bhagat AK, Srivastava RN, Jain A, Baghel K, Raj S. Expression of MMP-8 in pressure injuries in spinal cord injury patients managed by negative pressure wound therapy or conventional wound care. *J Wound Ostomy Continence Nurs*, 2017; 44(4): 343-349.
37. Dwivedi MK, Srivastava RN, Bhagat AK, Agarwal R, Baghel K, Jain A, Raj S. Pressure ulcer management in paraplegic patients with a novel negative pressure device: A randomised controlled trial. *J Wound Care*, 2016; 25(4): 199-207.
38. de Laat EHEW, van den Boogaard MHWA, Spauwen PHM, van Kuppevelt DHJM, van Goor H, Schoonhoven L. Faster wound healing with topical negative pressure therapy in difficult-to-heal wounds: A prospective randomized controlled trial. *Ann Plast Surg*, 2011; 67(6): 626-631.
39. Weir GR, Smart H, van Marle J, Cronje FJ. Arterial disease ulcers, Part 1: Clinical diagnosis and investigation. *Adv Skin Wound Care*, 2014; 27(9): 421.
40. Cardinal Health. *Cardinal Health™ Negative Pressure Wound Therapy CATALYST™ Clinician User Manual*. 2018; Available from: <https://www.cardinalhealth.com/content/dam/corp/web/documents/Manual/cardinal-health-catalyst-clinician-user-manual.pdf>.
41. KCI. *V.A.C.® Therapy™ Clinical Guidelines: A reference source for clinicians*. 2014; Available from: <http://research.vuse.vanderbilt.edu/srdesign/2005/group27/KCI%20operating%20procedures.pdf>.
42. Wild T, Stremitzer S, Budzanowski A, Hoelzenbein T, Ludwig C, Ohrenberger G. Definition of efficiency in vacuum therapy - A randomised controlled trial comparing with V.A.C. therapy. *Int Wound J*, 2008; 5(5): 641-647.
43. Fox A, Tadros A, Perks AG. An unusual complication of vacuum assisted closure in the treatment of a pressure ulcer. *J Wound Care*, 2004; 13(8): 344-5.
44. Deva AK, Buckland GH, Fisher E, Liew SC, Merten S, McGlynn M, Gianoutsos MP, Baldwin MA, Lendvay PG. Topical negative pressure in wound management. *Med J Aust*, 2000; 173(3): 128-131.
45. Wallin A-M, Bostrom L, Ulfvarson J, Ottosson C. Negative pressure wound therapy - A descriptive study. *Ostomy Wound Manage*, 2011; 57(6): 22-29.
46. Timmers M, Le Cessie S, Banwell P. The effects of varying degrees of pressure delivered by negative-pressure wound therapy on skin perfusion. *Ann Plast Surg*, 2005; 55(6): 665-71; 1097-98.
47. Morykwas MJ, Argenta LC, Shelton-Brown EI, McGuirt W. Vacuum-assisted closure: a new method for wound control and treatment: Animal studies and basic foundation. *Ann Plast Surg*, 1997; 38(6): 553-562.
48. Fabian T, Kaufman H, Lett E. The evaluation of subatmospheric pressure and hyperbaric oxygen in ischemic full-thickness wound healing. *Am J Surg*, 2000; 66(12): 1136-43.
49. Greene A, M. Puder M, Roy R. Microdeformational wound therapy: Effects on angiogenesis and matrix metalloproteinases in chronic wounds of three debilitated patients. *Ann Plast Surg*, 2006; 56(4): 418-22.
50. Wanner MB, Schwarzl F, Strub B, Zaech GA, Pierer G. Vacuum-assisted wound closure for cheaper and more comfortable healing of pressure sores: A prospective study. *Scand J Plast Reconstr Surg Hand Surg*, 2003; 37(1): 28-33.
51. Mostafa J, Ali Y, Zohre R, Samaneh R. Electromagnetic fields and ultrasound waves in wound treatment: A comparative review of therapeutic outcomes. *Biosciences Biotechnology Research Asia*, 2015; 12: 185-195.
52. Aziz Z, Bell-Syer SEM. Electromagnetic therapy for treating pressure ulcers. *Cochrane Database Syst Rev*, 2015; 9(Art. No.: CD002930).
53. Salzberg CA, Cooper Vastola SA, Perez F, Viehbeck MG, Byrne DW. The effects of non-thermal pulsed electromagnetic energy on wound healing of pressure ulcers in spinal cord-injured patients: a randomized, double-blind study. *Ostomy Wound Manage*, 1995; 41(3): 42-4, 46, 48
54. Seaborne D, Quirion-DeGirardi C, Rousseau M, Rivest M, Lambert J. The treatment of pressure sores using pulsed electromagnetic energy (PEME). *Physiother Can*, 1996; 48(2): 131-137.
55. Comorosan S, Vasilco R, Arghiropol M, Paslaru L, Jieanu V, S. S. The effect of Diapulse therapy on the healing of decubitus ulcer. *Rom J Physiol*, 1993; 30(1-2): 41-45.
56. Gupta A, Taly AB, Srivastava A, Kumar S, Thyloth M. Efficacy of pulsed electromagnetic field therapy in healing of pressure ulcers: A randomized control trial. *Neurol India*, 2009; 57(5): 622.
57. Frykberg RG, Driver VR, Lavery LA, Armstrong DG, Isenberg RA. The use of pulsed radio frequency energy therapy in treating lower extremity wounds: Results of a retrospective study of a wound registry. *Ostomy Wound Manage*, 2011; 57(3): 22-29.

58. Conner-Kerr T, Isenberg RA. Retrospective analysis of pulsed radiofrequency energy therapy use in the treatment of chronic pressure ulcers. *Adv Skin Wound Care*, 2012; 25(6): 253-60.
59. Chen C, Hou W-H, Chan Edwin SY, Yeh M-L, Lo Heng-Lien D. Phototherapy for treating pressure ulcers. *Cochrane Database Syst Rev*, 2014(7).
60. Taradaj J, Halski T, Kucharzewski M, Urbanek T, Halska U, Kucio C. Effect of laser irradiation at different wavelengths (940, 808, and 658 nm) on pressure ulcer healing: Results from a clinical study. *Evid Based Complement Alternat Med*, 2013.
61. Shojaei H, Sokhangoei Y, Soroush MR. Low level laser therapy in the treatment of pressure ulcers in spinal cord handicapped veterans living in Tehran. *Janbazan Medical eac Engineering Research Centre* 2008; 33(1): 44–8.
62. Lucas C, van Gemert MJ, de Haan RJ. Efficacy of lowlevel laser therapy in the management of stage III decubitus ulcers: A prospective, observer-blinded multicentre randomised clinical trial. *Lasers Med Sci*, 2003; 18 (2): 72–7.
63. Nussbaum E, Flett H, Hitzig S, McGillivray C, Leber D, Morris H, Jing F. Ultraviolet-C irradiation in the management of pressure ulcers in people with spinal cord injury: A randomized, placebo-controlled trial. *Arch Phys Med Rehabil*, 2013; 94(4): 650-9.
64. Dehlin O, Elmstahl S, Gottrup F. Monochromatic phototherapy in elderly patients: A new way of treating chronic pressure ulcers? *Aging Clin Exp Res*, 2003; 15(3): 259–63.
65. Wills EE, Anderson TW, Beattie BL, Scott A. A randomized placebo-controlled trial of ultraviolet light in the treatment of superficial pressure sores. *J Am Geriatr Soc*, 1983; 31(3): 131-3.
66. Onigbinde AT, Olafimihan KF, Ojoawo A, Adedoyin RA, Omiyale O, Mothabeng J. The effect of ultraviolet radiation (type B) on decubitus ulcers. *Internet J Allied Health Sci Pract*, 2010; 8(1): 1-6.
67. Schubert V. Effects of phototherapy on pressure ulcer healing in elderly patients after a falling trauma. A prospective, randomized, controlled study. *Photodermatol Photoimmunol Photomed*, 2001; 17(1): 32-38.
68. Rosenthal AM, Schurman A. Hyperbaric treatment of pressure sores. *Arch Phys Med Rehabil*, 1971; 52(9): 413-5.
69. Azimian J, Nayeri ND, Pourkhaleghi E, Ansari M. Transdermal wound oxygen therapy on pressure ulcer healing: A single-blind multi-center randomized controlled trial. *Iran Red Crescent Med J*, 2015; 17(11).
70. Tao H, Butler JP, Luttrell T. The role of whirlpool in wound care. *J Am Coll Clin Wound Spec*, 2012; 4(1): 7–12.
71. Burke DT, Ho CH, Saucier MA, Stewart G. Effects of hydrotherapy on pressure ulcer healing. *Am J Phys Med Rehabil*, 1998; 77(5): 394-8.
72. Ho CH, Bensitel T, Wang X, Bogie KM. Pulsatile lavage for the enhancement of pressure ulcer healing: A randomized controlled trial. *Phys Ther*, 2012; 92(1): 38-48.
73. Maloney-Hinds C, Petrofsky J, Zimmerman G, Hessinger DA. The role of nitric oxide in skin blood flow increases due to vibration in health adults and adults with type 2 diabetes. *Diabetes Technol Ther*, 2009; 11(1): 39-43.
74. Arashi M, Sugama J, Sanada H, Konya C, Okuwa M, Nakagami G, Inoue A, Tabata K. Vibration therapy accelerates healing of stage I pressure ulcers in older adult patients. *Adv Skin Wound Care*, 2010; 23(7): 321.
75. Janssen T, de Koning A, Legemate K, Smit C. Electrical stimulation-induced gluteal and hamstring muscle activation can reduce sitting pressure in individuals with a spinal cord injury. *Assistive Technology Research Series*, 2010: 332-334.
76. Smit C, Haverkamp G, de Groot S, Stolwijk-Swuste J, Janssen T. Effects of electrical stimulation-induced gluteal versus gluteal and hamstring muscles activation on sitting pressure distribution in persons with a spinal cord injury. *Spinal Cord*, 2012; 50(8): 590-4.
77. Smit CA, Zwinkels M, van Dijk T, de Groot S, Stolwijk-Swuste JM, Janssen TW. Gluteal blood flow and oxygenation during electrical stimulation-induced muscle activation versus pressure relief movements in wheelchair users with a spinal cord injury. *Spinal Cord*, 2013; 51(9): 694-9.
78. Smit CAJ, Legemate KJA, de Koning A, de Groot S, Stolwijk-Swuste JM, Janssen TWJ. Prolonged electrical stimulation-induced gluteal and hamstring muscle activation and sitting pressure in spinal cord injury: Effect of duty cycle. *J Rehabil Res Dev*, 2013; 50(7): 1035-1046.
79. Liu LQ, Ferguson-Pell M. Blood perfusion changes during sacral nerve root stimulation versus surface gluteus electrical stimulation on in seated spinal cord injury. *Assist Technol*, 2017; 29: 1-8.
80. Kane A, Warwaruk-Rogers R, Ho C, Chan M, Stein R, Mushahwar VK, Dukelow SP. A feasibility study of intermittent electrical stimulation to prevent deep tissue injury in the intensive care unit. *Adv Wound Care*, 2017; 6(4): 115-124.

PRESSURE INJURY SURGERY

Introduction

Category/Stage III and IV pressure injuries are often difficult to heal using conventional wound healing techniques. Surgical management includes surgical sharp debridement or pressure injury excision, with or without a split skin graft or flap closure to cover the wound. Surgical management becomes a treatment option when a pressure injury does not respond to traditional management strategies including debridement, infection management, and advanced wound dressings, in conjunction with pressure redistribution. In some cases, surgical management becomes an urgent necessity, for example due to suspected sepsis or osteomyelitis.

This chapter focuses on preoperative, intraoperative, and postoperative recommendations for surgical management of pressure injuries, particularly reconstruction surgery that involves flap closure. Surgical sharp debridement is discussed in the guideline chapter *Cleansing and Debridement*. This chapter does not address specific surgical techniques; those decisions are more appropriately made by an experienced surgeon who understands the unique needs of the individual, the pressure injury requiring surgical management and the wound healing environment.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What indicators are appropriate for considering eligibility for surgical intervention for a pressure injury?
- What preoperative interventions are effective for supporting the individual undergoing surgical intervention for a pressure injury?
- What intraoperative interventions are effective for supporting the individual undergoing surgical intervention for a pressure injury?
- What postoperative interventions are effective for supporting the individual undergoing surgical intervention for a pressure injury?
- What interventions are effective for reducing recurrence of a pressure injury following surgical intervention?

Selection of Individuals for Pressure Injury Surgery

18.1: Obtain a surgical consultation for an individual with a pressure injury that:

- Has advancing cellulitis or is a suspected source of sepsis
- Has undermining, tunneling, sinus tracts and/or extensive necrotic tissue not easily removed by conservative debridement
- Is Category/Stage III or IV and not closing with conservative treatment.

(Good Practice Statement)

Implementation Considerations

- When sepsis is suspected, surgical consultation should be obtained urgently (*Expert opinion*).
- When the individual with a pressure injury experiences fever with no obvious other foci of infection, suspect bacteremia associated with the pressure injury¹ (*Expert opinion*).
- In the presence of clinical signs of infection, dry, stable eschar requires assessment by a medical practitioner/vascular surgeon and possible urgent surgical sharp debridement (*Expert opinion*).
- Surgical referrals should be appropriate to the individual's clinical condition and their goals of care (*Expert opinion*).

Discussion

Pressure injuries are a known cause of sepsis and death.¹⁻⁶ For pressure injuries with advancing cellulitis, abscess or gross infection, a surgical consultation should be made urgently for draining and/or debridement. Bacteremia or sepsis originating from a pressure injury occurs infrequently—one 32-year cohort study reported an incidence of 1.70 per 10,000 adult discharges in a teaching hospital in Spain¹—but should it occur, urgent surgical management may be required.

Surgical consultation should be obtained in some clinical presentations that benefit from surgical sharp debridement. As discussed in the guideline chapter on *Cleansing and Debridement* (see *Recommendation 12.5*), stable, hard, dry eschar should not be debrided when there is insufficient blood supply to support infection control or healing.⁷ However, urgent surgical sharp debridement of dry, stable eschar may be required in the presence of clinical signs of infection. In this case, surgical sharp debridement may be appropriate as it is a rapid means of moving non-viable tissue.⁸⁻¹³ Additionally, pressure injuries with undermining, tunneling/sinus tracts, and/or extensive necrotic tissue that cannot be easily removed by other debridement methods should be reviewed by the surgical team for possible surgical sharp debridement.⁸⁻¹³ The rationale for a surgical review includes improved anesthesia, analgesia, clinical monitoring and access to instrumentation equipment and ancillary staff.

A surgical consultation should also be initiated to evaluate the eligibility for surgical repair of the pressure injury for individuals with non-healing severe pressure injuries. Category/Stage III and IV pressure injuries are missing large amounts of skin, subcutaneous fat and sometimes muscle. Exposed bone can also be present, increasing the risk of osteomyelitis. With conservative treatment, Category/Stage III and IV pressure injuries may take months to years to heal.^{14,15}

18.2: Consider the following factors when assessing the individual's eligibility for pressure injury surgery:

- Likelihood of healing with conservative treatment versus surgical intervention
- The individual's goals of care
- The individual's clinical condition
- Motivation and ability of the individual to comply with the treatment regimen
- Risk of surgery for the individual.

(Good Practice Statement)

Implementation Considerations

- Consider using a formal assessment tool to evaluate risk of mortality (e.g., American Society of Anesthesiologists [ASA] classification system, the Acute Physiology and Chronic Health Evaluation II [APACHE II] and the Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity [POSSUM])¹⁶ (*Expert opinion*).
- Evaluate factors that may impair the individual's ability to heal following surgery including nutritional status, smoking status, continence, co-morbidities, infection and psychosocial factors (*Expert opinion*).
- In planning surgery, discuss with the individual and their informal caregivers:
 - Goals of care
 - Expected outcomes
 - Risks and benefits
 - Pre- and post-operative treatment regimen (*Expert opinion*).

Discussion

When consistent with the individual's goal of care, surgical excision and repair of the pressure injury achieves rapid closure of the defect and establishes durable, thick, soft tissue coverage and revascularization. Surgical excision and repair also contribute to improved functional capacity and quality of life. In the research reporting pressure injury surgery, some studies reported using specific selection criteria for selection of surgical candidates. Factors that should be evaluated include the need for surgery (e.g., likelihood that the wound will heal through conservative treatment), individual's clinical condition, goals of care and motivation, as well as surgical risks and potential surgical outcomes.

Healing and recurrence

It is important that the surgeon determines and communicates the potential for healing prior to undertaking surgical intervention. Most individuals initially heal from pressure injury surgery. In observational studies reported in the literature, complete healing rates were generally high. Ljung et al. (2017)¹⁷ noted that 96% of a cohort undergoing Category/Stage III or IV pressure injury surgery in Sweden were completely healed within four weeks (*Level 3*). Huang et al. (2015)¹⁸ reported a 100% complete healing rate by final follow-up (between four months and three years) for a sample of individuals undergoing surgical reconstruction for a Category/Stage III or IV pressure injury in China (*Level 4*). Srivastava et al. (2009)¹⁹ reported an 87% total healing rate (mean follow up of 15.4 ± 7.45 months) for 25 individuals with spinal cord injuries (SCI) undergoing surgery for Category/Stage III, IV or unstageable pressure injuries in India (*Level 4*). The difference in healing rates reported across studies may be due to severity of illness, duration of follow up, the surgical procedure used in the primarily single center studies or due to differences in how complete healing was defined. In a multivariate logistic regression of date from individuals undergoing surgery ($n =$

137 individuals with $n = 231$ flap surgeries), having had previous flap surgery at the same anatomical site (OR = 3.84, 95% CI 1.23 to 11.94, $p = 0.02$), poor diabetes control (OR = 3.84, 95% CI 1.11 to 4.19, $p = 0.024$) and being aged less than 45 years at the time of surgery (OR = 4.89, 95% CI 1.19 to 20.08, $p = 0.028$) were associated with impaired healing requiring further surgery²⁰ (Level 3).

Although most individuals appear to reach complete healing following pressure injury surgery, recurrence of the pressure injury is a significant concern, especially for individuals who are at high risk of pressure injuries (e.g., individuals with SCI). Individuals who have failed to adhere to a pressure injury prevention program in the past may be at risk of recurrence, especially if there is no ongoing change in pressure injury prevention behavior following surgery.²¹ Thus, the likelihood of a recurring or a new pressure injury should be considered and discussed with the individual and his or her informal caregivers when discussing surgical eligibility and rehabilitation plan.

A long term cohort study ($n = 33$)¹⁷ conducted in Switzerland reported the three-to-ten year recurrence rate following pressure injury surgery was 27%, and an additional 18% of individuals had a new pressure injury at a different anatomical location. Of these, 9% had undergone repeat surgery (Level 3). Other studies reported recurrence rates of between 0% and 39% over mean follow up periods of between 14 months to 12 years¹⁸⁻²⁹ (Levels 3 and 4).

In a multivariate analysis²⁰ of factors associated with recurrence after pressure injury surgery (137 individuals with 231 pressure injuries), having had surgery on an ischial pressure injury (OR = 2.87, 95% CI 1.5 to 5.6) and having had previous failed surgery at the same anatomical site (OR = 3.3, 95% CI 1.4 to 7.6) were associated with recurrence over a mean period of 4.4 years²⁰ (Level 3). Similar findings were noted in another prognostic study²⁹ conducted over 20 years ($n = 276$) that analyzed pressure injury recurrence following surgery. Factors that were significant in pressure injury recurrence included having had surgery on an ischial pressure injury (RR = 3.46, 95% CI 1.76 to 6.81, $p < 0.01$), having a body mass index (BMI) below 18.5 kg/m² (RR = 3.13, 95% CI 1.34 to 7.27, $p < 0.01$) and being a smoker (RR = 2.33, 95% CI 1.16 to 4.7, $p = 0.0018$). Age, diabetic status, size of the pressure injury at time of surgical repair and presence of osteomyelitis were not significantly related pressure injury recurrence²⁹ (Level 3). A third multivariable analysis³⁰ that included pressure injury surgery cases with a mean follow up of 55 months ($n = 181$ individuals) also identified having had surgery on an ischial pressure injury as a significant risk factor for recurrence (OR = 3.02, 95% CI 1.32 to 6.93, $p = 0.009$). Other factors that were related to pressure injury recurrence included paraplegia (OR = 2.42, 95% CI 1.29 to 4.56, $p = 0.006$) and albumin level (OR = 2.09, 95% CI 1.11 to 3.91, $p = 0.021$) (Level 3).

Surgical risks and complications

Although high rates of complete healing were reported in the studies above,¹⁷⁻¹⁹ rates of general surgical complications, wound dehiscence and pressure injury recurrence have also been reported as high in many studies. These potential outcomes and the individual's physical and psychological ability to overcome complications and setbacks during healing, should be considered and discussed with the individual and their informal caregivers when assessing eligibility for surgery.

Individuals with wounds who undergo surgery with general anesthetic are reported to have more comorbidities and greater general surgical risk than an average individual undergoing surgery.³¹ Individuals with medical conditions that would be worsened by general anesthesia, blood loss, systemic stress, or immobility following surgery are usually not candidates for surgical pressure injury repair. General anesthesia is required for T-6 paraplegics and tetraplegics to control hyperreflexia and autonomic dysfunction. General anesthesia is also required when the individual is positioned prone for the operation. Operative procedures may last up to three hours and may result in blood loss requiring transfusion. Time undergoing surgery also places individuals at risk of new pressure injuries, with longer surgical durations associated with a greater risk.³²⁻³⁷ See the guideline chapter *Risk Factors and Risk Assessment* for further discussion on pressure injury risk associated with undergoing any surgery.

In a multivariate analysis of risks for general post-operative complications in individuals undergoing surgical closure of a pressure injury, Thiessen et al. (2011)²⁴ reported a significantly lower risk for individuals who had no pre-operative paralysis (odds ratio [OR] = 0.081, 95% confidence interval [CI] 0.009 to 0.706, $p = 0.02$). There was also a lower risk of general complications for individuals who were not hospitalized when their pressure injury developed (OR = 0.108, 95% CI 0.0021 to 0.563, $p = 0.008$). There was no relationship between the type of flap closure performed and risk of post-operative complications (Level 3).

Ljung et al. (2017)¹⁷ reported four-week outcomes for individuals with SCI undergoing surgery for Category/Stage IV pressure injuries ($n = 51$). In this cohort the general complication rate was 4% and the local complication rate was 6%. Local complications included local bleeding, minor flap necrosis that healed within three months and delay in healing beyond two months.¹⁷ This study also reported long term outcomes at ten years following surgery for 33 individuals. Although the pressure injury recurrence rate at ten years was 27%, health scores measured on the EQ-5D@ tool³⁸ were a median of 70 (100 point visual analog scale) compared with the median pre-operative score of 30. This suggests that despite the risk of complications and the high recurrence rate, overall the individuals undergoing surgery appeared to have large improvements in health status¹⁷ (Level 3).

In a larger cohort study conducted in the US (n = 276) a complication rate of 58.7% was reported for individuals undergoing surgery for a pressure injury. In this study, wound dehiscence (31.2%) and flap infection (6.5%) were commonly reported²⁹ (Level 3). Across the other included studies, complication rates following pressure injury surgery were variable. Post-surgical wound dehiscence rates from 0 to 49% were reported^{18-21,39-44} and flap complications (e.g., infection and necrosis) ranged from 0% to 37.5%^{18,21,24-28,40-42,44-48} (Levels 3 and 4).

Variations in complication rates across the studies above likely relate to the wide range of clinical settings, surgical procedures, methods of defining and measuring outcomes and the length of follow-up, which varied from weeks to decades. Tashiro et al. (2016)⁴⁹ reported a risk-adjusted multi-variable analysis exploring patient factors associated with flap complications. Records from a national US database for individuals who underwent surgical reconstruction of a pressure injury site (n = 2,749) were included in the analysis. The study reported that risks of flap complications significantly increased for females (OR = 1.64, 95% CI 1.10 to 2.44, p = 0.02), individuals with obesity (OR = 1.90, 95% CI 1.02 to 3.55, p = 0.04), individuals from an Asian background (OR = 4.78, 95% CI 1.40 to 16.32, p = 0.01) and individuals with renal failure (OR = 4.99, 95% CI 2.23 to 11.16, p < 0.001). There was also variations in risk based on the anatomical location of the surgical repair, with trochanteric flaps (OR = 4.54, 95% CI 2.38 to 8.33, p < 0.001) and sacrococcygeal flaps (OR = 1.72, 95% CI 1.02 to 2.86, p = 0.04) associated with higher rates of flap complication than gluteal flaps⁴⁹ (Level 3). A second, smaller prognostic study (n = 276)²⁹ reported that having a diagnosis of diabetes mellitus was a significant risk factor for post-operative infection (relative risk [RR] = 4.34, 95% CI 1.15 to 16.43, p = 0.031) and having osteomyelitis was a significant risk factor for wound dehiscence (RR = 2.78, 95% CI 1.51 to 5.13, p < 0.01). However, age, BMI, smoking status were not significant risk factors for either complication²⁹ (Level 3).

Tools for assessing surgical eligibility

Various tools are used by surgeons, anesthesiologists and anesthesiologists to assist in an assessment of the individual's fitness to undergo surgery and general anesthetic. Commonly used tools include (but are not limited to) the ASA classification system, the APACHE II and the POSSUM.¹⁶

Only one study in the included literature investigated the use of a surgical checklist with individuals undergoing surgical sharp debridement, split skin grafts or flap closures. Kurita et al. (2009)⁵⁰ investigated the use of POSSUM and O-POSSUM (a version of POSSUM adapted for assessing eligibility for orthopedic surgery) for predicting of mortality risk for those pressure injury surgery. A cohort of individuals undergoing pressure injury surgery (n = 50 individuals; n = 71 surgeries) was compared to a cohort of individuals undergoing surgery that was not related to chronic wounds (n = 62 individuals; n = 62 surgeries). The study found that individuals receiving pressure injury surgery had higher predicted mortality scores than individuals undergoing other types of surgery (p < 0.001 on both the POSSUM and O-POSSUM). Both scales were considered valid predictive methods. However, the results may have been influenced by the difference in mean age between the cohorts (72.1 ± 17.5 years in the pressure injury surgery group versus 47.2 ± 20.8 years in the non-pressure injury surgery group) (Level 3).

Motivation to adhere to management plan

While clinical condition, including preparation of the wound bed, infection status and nutritional parameters,²⁴ are important to determining whether surgery is an option, motivation is also reported as an important consideration. In one study conducted in individuals with spinal cord injury (SCI) undergoing surgery for Category/Stage IV pressure injuries (n = 51), surgical candidates were selected based on their motivation and ability to follow treatment, as well as the expectation of failure to heal with conservative treatment.¹⁷ (Level 3). In another study, individuals undergoing pressure injury surgery (n = 158) were selected for the procedure with consideration to their ability to adhere to the pre and post-operative treatment program²³ (Level 4). Willingness to adhere to an ongoing management plan²¹ including positioning requirements, daily skin inspection, and progressive rehabilitation must be confirmed with the individual and their informal caregivers. Adequate preoperative education may facilitate to program adherence (see Recommendation 18.3).

Preparing the Individual for Pressure Injury Surgery

18.3: Evaluate and mitigate physical and psychosocial factors that may impair surgical wound healing or influence recurrence of a pressure injury.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Five Level 3 studies of high,²⁹ moderate^{20,49,51} and low⁵² quality identified comorbidities, including diabetes,^{20,29} renal disease,⁴⁹ obesity,⁴⁹ prealbumin levels⁵¹ and laboratory blood results indicative of clinical condition^{51,52} as being significantly related to an increased risk of post-surgical wound/flap complications. Five Level 4 studies of moderate^{22,25}

and low^{18,27,28} quality reported interventions that included optimization of the individual's clinical condition, including nutritional status^{18,22,27,28} and continence management,^{25,28} prior to surgery were associated with wound complication rates of between 15% and 38% but overall high positive healing rates following surgery. An additional moderate quality Level 4 study²³ reported on nutritional support provided prior to pressure injury surgery, with outcomes of 25% recurrence rate reported. Three moderate quality Level 4 studies^{19,22,23} reported providing education to the individual and their informal caregivers. Moderate quality Level 4 studies reported that assessment of home circumstances,²² and promotion of access to social support²³ were components of management plans.

Implementation Considerations

- Optimize control of diabetes prior to pressure injury surgery^{20,29} (Level 3).
- Optimize nutritional status prior to pressure injury surgery^{18,22,27,28,51,53-55} (Level 3 and 4). The guideline chapter on *Nutrition in Pressure Injury Prevention and Treatment* provides comprehensive guidance on nutritional optimization.
- Diagnosis of osteomyelitis could be undertaken preoperatively^{18,25,56} or intraoperatively^{22,25,40,44,56-59} depending on the individual's clinical course and available resources (Levels 3 and 4). Refer to the guideline chapter *Infection and Biofilms* for discussion of diagnostic tests and treatment strategies for osteomyelitis.
- Manage incontinence prior to pressure injury surgery^{25,28} (Level 3 and 4).
- Confirm the individual's end-of-life preferences if anticipating surgery (*Expert opinion*).
- Provide access to preoperative and ongoing education on the surgical procedure, the postoperative regimen and ongoing pressure injury prevention^{18,19,22,23,60} (Levels 1 and 4). The guideline chapter on *Quality of Life, Self-Care and Education* provides guidance on education interventions.
- Optimize healthy lifestyle choices and a supportive social network preoperatively and prior to discharge. The guideline chapter on *Quality of Life, Self-Care and Education* has more information on psychosocial factors in pressure injury treatment and prevention.

Evidence Discussion

Expectations of the operation and the ability of the individual to tolerate surgery and surgical recovery should be discussed and understood. Palliative care can include surgery for the treatment of pain and control of odor when the risk-benefit ratio is favorable.⁴

Conducting a pre-operative assessment of factors that may influence the individual's recovery and risk of recurrence (see *Recommendation 18.2*) enables identification and address of potential intraoperative and postoperative complications.

Management of comorbidities and medication use can promote more rapid healing and reduce the risk of complications. Assessment and management of diabetes mellitus,^{20,29} muscular spasms and tobacco use can promote healing and reduce risk of suture line disruption. Individuals taking cortisone, chemotherapy, antiproliferative, or immunosuppressive drugs may have a higher complication rate and a longer healing duration. When appropriate, reducing the dosages of these medications (if feasible) may promote wound healing.

Nutritional status must be optimized to promote healing. Nutritional optimization and support was a component of comprehensive pressure injury surgery protocols (Levels 3 and 4)^{17,18,21-25,27,28} and a high calorie, high protein diet was commenced in the pre-operative period in some reported protocols^{18,21,27,28} (Level 4). In consultation with the individual, dietary supplementation or tube feeding may be indicated to achieve nutritional optimization.

Control of factors that may increase the risk of post-operative infection is also important. Continence management is a significant issue because urine and feces have potential to contaminate the surgical site. Diarrhea should be controlled to prevent fecal contamination. Control of diarrhea may require fecal containment systems, medication, or diverting colostomy.^{61,62} Encourage individuals with SCI preparing for surgery to perform intermittent clean self-catheterization to prevent urinary contamination of sacral pressure injuries^{25,28} (Level 3 and 4) or consider insertion of an indwelling catheter.

Wound infection and osteomyelitis

Management of wound infection and biofilm prior to surgery maximizes the individual's potential for healing. Some management protocols reported in the literature included pre-operative assessment for deep infection using wound cultures and radiograms for all pressure injuries scheduled for a surgical repair^{18,25} (Level 4). Appropriate diagnosis and pre-operative wound infection management was reported as a helpful component of numerous surgical protocols^{18,19,26,59} (Levels 3 and 4) and a requirement for surgical eligibility in some centers^{24,63} (Level 3). The guideline chapter *Infection and Biofilm* discusses assessment and treatment strategies that can be initiated preoperatively.

Osteomyelitis has been reported in up to 32% of individuals with pressure injuries.^{64,65} Analysis of records from individuals undergoing pressure injury surgery (n = 276) indicated that presence of osteomyelitis in the wound is associated with an increase in wound dehiscence (RR = 2.78, 95% CI 1.51 to 5.13, p < 0.01), but is not statistically associated with post-operative wound infection rates or recurrence²⁹ (*Level 3*).

Some studies have explored the comparative benefits of identifying osteomyelitis pre-operative compared with waiting to undertake an intra-operative bone sample for culture. In observational studies, both pre-operative diagnosis (e.g., using plain film X-rays or magnetic resonance imaging [MRI]) and harvesting of bone samples during the surgical procedure have been used, with no apparent differences in management or outcomes between the two strategies. For example, in a review of individuals diagnosed for osteomyelitis in conjunction with pressure injury surgery (n=47), conducting diagnostic preoperative MRI did not lead to a statistically significant difference in the rate of pre-operative antibiotic therapy compared to individuals who did not receive an MRI (26.9% versus 23.8%, OR = 1.2, p = 0.81). There was also no statistically significant difference in post-surgical infection between those with osteomyelitis diagnosed by MRI and those with osteomyelitis diagnosed by bone culture (7.7% versus 14.3%, OR = 0.50, p = 0.44)⁵⁶ (*Level 3*). These findings suggest that evaluation and treatment of osteomyelitis could be identified either pre or intraoperatively depending on the individual's clinical course and available resources.

Psychosocial and education factors

Prior to the surgical procedure, establishing the psychosocial and educational needs of the individual and their informal caregivers is essential to optimizing potential for healing.²²

Having a strong knowledge about pressure injuries, their prevention and the treatment the individual will need to engage in is an imperative to achieving adherence to the protocol and instilling behaviors that prevent recurrence. In one retrospective review (n = 168 individuals), Schryvers et al. (2000)³⁹ reported that most participants undergoing pressure injury surgery at one center were paraplegic, unemployed men who had a low level of education.³⁹ These men were primarily undertaking their own care, even if they lived with family. This indicates a cohort with high educational support needs (*Level 4*). Some of the surgical management protocols reported in the literature noted the inclusion of education initiatives for both patient individuals and their informal caregivers, commencing either preoperatively^{19,22,23} or postoperatively prior to discharge¹⁸ (*all Level 4*).

One RCT⁶⁰ compared the effectiveness of an education intervention in preventing pressure injury recurrence following pressure injury surgery. Individuals (n = 38 analyzed) were randomized to one of three groups in conjunction with standard preoperative and postoperative care. The different interventions were an enhanced, structured education program delivered monthly for two years following discharge, monthly consultations for skin assessment but no education for two years and follow-up by mail every three months for two years. The group receiving structured education had a significantly lower rate of pressure injury recurrence compared to the other two groups (33% versus 60% versus 90%, p = 0.007), which translated to an odds ratio of 0.228 (95% CI 0.080 to 0.647, p = 0.003)⁶⁰ (*Level 1*). However, this study had an insufficient sample size for statistical power and the education intervention was not described in detail.

A positive psychological status, strong self-care skills, family involvement and good social networks can improve short- and long-term outcomes. Yarkin et al. (2009)⁶⁶ investigated psychiatric state and quality of life for individuals with SCI undergoing pressure injury surgery (n = 17) and their informal family caregivers (n = 18). Prior to surgery, the individuals with pressure injuries had significantly lower scores (p < 0.05) on all components of the Short Form-36 (SF-36) compared with the national (US) average of the general population. Informal caregivers had significantly lower scores for the SF-36 components of social function, emotional difficulty and mental health, but no statistically significant difference in physical function, physical role difficulty, pain, general health or energy levels (*Level 3*). This suggests that living with a chronic condition (such as SCI) and managing a pressure injury significantly impacts the quality of life of both individuals and their families. In the six month follow up after surgery, Yarkin et al (2009)⁶⁶ demonstrated significant improvements in SF-36, Beck Depression Inventory and Trait Anxiety Inventory scores for both individuals who underwent surgery and their informal caregivers (all p < 0.05) demonstrating that undergoing surgery itself has potential to improve the individual's psychosocial status (*Level 3*). This may be due to relief of depression, anxiety and the burden of caring for a pressure injury. However, the study also showed an association between higher levels of anxiety and recurrence, highlighting the importance of providing psychosocial support throughout the recovery period.

Some of the surgical management protocols described in the literature incorporated interventions to optimize psychosocial status prior to discharge. Tadiparthi et al. (2016)²² undertook preoperative assessments of the individual's home circumstances to enable early address of psychosocial needs prior to discharge²² (*Level 4*). In a retrospective review, Kierney, 1998²³ reported a recurrence rate of 19% through delivery of a surgical management protocol that included a strong focus optimization of social support that extended beyond the individual's discharge from hospital. This was achieved by promoting establishment of a viable social support network through introduction to

other individuals undergoing similar surgery (*Level 4*). Srivastava et al. (2009)¹⁹ provided ongoing counselling during hospitalization and after discharge to strengthen the capacity of the individual to adhere to the prevention and treatment plan (*Level 4*).

Intraoperative Management of the Individual and the Pressure Injury

The recommendations in this section focus on surgical considerations in repairing a pressure injury. During the intraoperative period, universal pressure injury prevention is required to prevent new pressure injuries. This includes conducting risk and skin assessments, attention to positioning, use of appropriate support surfaces and pads (including facial if in the prone position) and preventing medical device related pressure injuries. Recommendations in other chapters of this guideline are also appropriate to the intraoperative period.

18.4: Fully excise the pressure injury, including abnormal skin, granulation and necrotic tissue, sinus tracts, bursa and involved bone to the extent possible.

(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

Two moderate quality Level 3 studies^{17,24} and nine Level 4 studies of moderate^{22,25,40,44,46,57} and low^{18,27,47} quality reported procedures that included full excision of the wound bed, including sinus tracts, necrosis and bursa. One moderate quality Level 3 study²⁴ reported procedures that included full excision of the wound bed, including sinus tracts, necrosis and bursa. One moderate quality Level 3 study²⁴ and three moderate and low^{27,47,59} quality Level 4 studies reported resection and evening out uneven bony surfaces as a component of the surgical procedure.

Evidence Discussion

Removal of nonviable tissues is key to the surgical procedure to promote healing. Many studies reported that full excision of the pressure injury defect was part of the surgical procedure used in the centers reporting surgical outcomes^{17,18,22,24,25,27,40,44,46,47,57} (*Levels 3 and 4*). Aggressive removal of nonviable bone down to a level where the bone bleeds during the surgical procedure is generally advised.^{59,67}

In studies reporting comparisons, healing rates were similar for pressure injury surgeries that used single-stage or multiple-stage operations.^{63,68} Laing et al. (2010)⁴⁵ reported on individuals (n = 41) who underwent surgical debridement followed by closure using negative pressure wound therapy, approximately 50% of whom required definitive surgical reconstruction after a mean of 4.3 weeks. Following the surgical debridement, 12% of the individuals experienced bleeding and required transfusion, leading the researchers to propose that a multi-stage procedure may facilitate hemostasis and prevent hematoma formation (*Level 4*).

Osteomyelitis has been reported in up to 32% of individuals with pressure injuries.^{64,65} Permanent healing of the pressure injury or successful surgical closure are unlikely until osteomyelitis is controlled because bacterial infection of the bone has a profoundly detrimental effect on the viability of the flap.⁵⁹ Analysis of records from individuals undergoing pressure injury surgery (n = 276) indicated that presence of osteomyelitis in the wound is associated with an increase in wound dehiscence (RR = 2.78, 95% CI 1.51 to 5.13, p < 0.01), but is not statistically associated with post-operative wound infection rates or recurrence²⁹ (*Level 3*). Effective management can be achieved through aggressive surgical removal of non-viable bone (e.g., bone that feels rough or soft). Bone samples for culture and sensitivities should be considered for a definitive diagnosis of osteomyelitis and targeted antibiotic therapy. In the literature, management protocols included taking bone samples when osteomyelitis was "suspected",²² when bone was exposed,⁵⁸ when bone felt soft, or for all pressure injuries.^{25,40,44,57,59} The guideline chapter on *Infection and Biofilm*, discusses treatment of osteomyelitis.

18.5: When designing a flap:

- Select tissue with a good quality blood supply
- Use composite tissues to increase durability
- Use a flap as large as possible
- Minimize violation of adjacent skin and tissue
- Locate the suture line away from areas of direct pressure
- Minimize tension on the incision at closure.

(Good Practice Statement)

Implementation Considerations

- Consider using a hand-held Doppler prior to surgery to detect perforators providing good flap vascularization^{25,41,47} (*Level 4*).

Discussion

The most durable wound closure technique fills the pressure injury defect with tissue bulk to provide padding and protect underlying structures. This is generally achieved using flap repair. A flap consists of tissue that has its own vascular supply⁴⁷ and therefore does not require the same level of vascular support from the wound bed for survival as does a graft. Harvesting a flap requires identification of strong vasculature that will also be dissected with the tissue, which is usually a more complicated and time-consuming process than a graft. In many clinical situations a flap is the preferred surgical repair method because the wound bed of a deep pressure injury is generally not sufficiently healthy to support a graft.²¹

Flaps used for pressure injury reconstruction can be local flaps or free flaps. A local (pedicled) flap consists of freed tissue that remains attached to the body at its base and is rotated, maintaining its blood supply intact, to cover the pressure injury defect. A free flap is tissue that is removed with its blood supply and both the tissue and vascularization are reconnected over the pressure injury site. A free flap is best used to repair a large pressure injury that has insufficient local tissue to adequately cover the defect.⁶⁹ Regardless of whether a local or free flap is used, selection of the flap tissue should be made with consideration to preserving adjacent tissue for potential future options, while ensuring the chosen flap is as large as possible and sufficiently covers the defect.

Flaps are also described by the type of tissue they contain. For example, a myocutaneous flap (also called a musculocutaneous flap) consists of muscle, subcutaneous tissue and skin. A fasciocutaneous flap consists of deep fascia, subcutaneous tissue and skin.²⁶ Designing a flap from composite tissues improves the durability of the flap and provides enough tissue to adequately fill the defect.²³ Flaps should also be designed to locate the suture line away from an area of direct pressure to promote post-operative healing, and tension on the incisions at the time of closure should be minimized.

Selection of the type of tissue to use for a flap will be made with consideration to the individual's clinical condition, available tissue for harvest, the anatomical location of the pressure injury and the surgeon's preference.²⁸ Reports that compare different types of flaps, many of which were not included for this guideline due to their primary focus on surgical technique, suggest that clinical outcomes are comparable between different flap types.^{18,26,28,30,42,69} In a retrospective cohort study, outcomes for pressure injuries repaired with myocutaneous flaps, fasciocutaneous flaps, or free perforator-based flaps ($n = 181$) were reported. At follow up (mean 55 months), the complication rate was similar between different flap types (ranged between 44.2% and 48.8%). The pressure injury recurrence rate was also similar between the three flap types (range 15.1% and 18.6%)³⁰ (*Level 3*). Thiessen et al. (2011) compared myocutaneous flaps and fasciocutaneous flaps ($n = 94$), reporting no statistically significant difference based on type of flap for hospital stay ($p = 0.059$), wound dehiscence ($p = 0.835$), infection ($p = 0.135$), flap necrosis ($p = 0.735$), requirement for additional surgical procedures ($n = 0.648$) or recurrence ($p = 0.648$)²⁴ (*Level 3*). Other observational studies have reported similar outcomes between free and local flaps,⁶⁹ and between flaps of different tissue types^{18,26,42} (all *Level 4*).

Post-operative Management

18.6: Regularly monitor the wound and immediately report signs of flap failure.
(Good Practice Statement)

Implementation Considerations

- Regularly monitor wound drainage systems (*Expert opinion*).
- Report signs of flap failure to the surgeon immediately, including:
 - Pallor of poor arterial supply
 - Slow capillary refill
 - Mottling
 - Bluish-purple tissue of venous engorgement
 - Purple-maroon tissue of deep tissue injury (*Expert opinion*).
- Consider using advanced monitoring devices (e.g., Doppler systems, infrared spectroscopy and surface temperature monitoring) to evaluate the health of the flap following surgery (*Expert opinion*).

Discussion

Flap failure can occur due to loss of arterial blood supply or impairment of venous return. Gold standard technique for monitoring flaps is the clinical observation of color and capillary refill.⁷⁰ Arterial inflow appears as pallor or mottling in the flap. Venous engorgement, which is fairly rare (except in free flaps), presents as a swollen or bluish-purple tissue. The purple-maroon color typical of deeper tissue injury is a rare but ominous sign of flap failure. Devices may be used to monitor flaps, including implantable Doppler, infrared spectroscopy and surface temperature monitoring.^{70,71} However, there was no included evidence that evaluated the use of these monitoring devices in pressure injury flap surgery, and not all clinical and geographic locations have these advanced technologies available for routine postoperative use.

Suture line dehiscence is one of the most common complication after pressure injury surgery. Observational studies reported rates of up to 48.5%, although higher quality studies tended to report higher dehiscence rates^{18-21,24,25,29,40,41,47,72} (Levels 3 and 4). In one multivariable analysis that included analysis of records for individuals following flap surgery (n = 227), higher rates of dehiscence occurred in individuals with poorly controlled diabetes mellitus (OR = 15.9, 95% CI 2.0 to 127), individuals aged below 45 years OR = 4.9, 95% CI 1.2 to 20.1) and individuals who had previously had failed flap surgery at the same anatomical location (OR = 3.8, 95% CI 1.2 to 11.9)²⁰ (Level 3).

Implement frequent monitoring and early referral of complications to the individual's treating physician. Protocols reported in observational studies included regular monitoring of the flap following surgery^{17,22,28,73} (Level 4).

Drainage systems are placed to remove fluid from dead space and to prevent seroma and hematoma formation in the surgical site.^{22,27,58} Collections of blood and serum can become a source of infection, and fluid accumulation can place tension on the wound. Use of suction drains also help the flap adhere to the underlying wound bed.

Drain tubes should be regularly checked for kinking/clogging or other sources of occlusion. Drainage from wounds should be recorded, and those records should guide decisions for drain removal. Drains may remain in situ for up to three weeks.^{22,27,58} Drain tubes can be a source of pressure and shear forces that increase the risk of new pressure injuries. The guideline chapter *Medical Device Related Pressure Injuries* provides comprehensive guidance on reducing the risk of pressure injuries associated with drain tubes and other devices.

18.7: Use a specialty support surface in the immediate post-operative period.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Most of the evidence on support surfaces following pressure injury surgery report use of an air fluidized bed. One low quality Level 1 study⁷⁴ reported post-operative healing rates of 78% for an air fluidized bed and 86% for an alternating pressure air mattress. These results show similar outcomes between the two types of specialty support surface, but no statistical comparison was made. Seven moderate and low quality Level 3^{17,63} and 4^{23,25,44,57,58} observational studies reported management protocols that included use of air fluidized beds, sometimes commencing in the pre-operative period. In these studies, use of air fluidized beds was for between two and four weeks. The studies report a range of different outcome measures including complete healing rates of 61% to 96%,^{17,44} complication rates of 3% to 26%^{25,57,58,63} and recurrence rates of 25%.²³ Feasibility of using air fluidized beds is influenced by resources and accessibility.

Implementation Considerations

- Consider using an alternating pressure air mattress^{40,74} (Levels 1 and 4), an air fluidized bed^{17,23,25,44,58,63,74} (Levels 1 and 4) or a bed with a low air loss feature (*Expert opinion*).
- Prior to surgery, procure appropriate support surfaces for the postoperative period^{23,28} (Level 4).
- Preoperatively undertake an assessment of the individual's living environment and commence procurement of support surfaces and other equipment that will be required on discharge. This might be facilitated by a referral to an appropriately trained health professional (e.g., seating specialist, occupational therapist, physiotherapist and/or other trained professional) (*Expert opinion*).
- Use a specialty support surface for at least two to four weeks following surgery^{17,23,25,44,58,63,74} (Level 4).
- Avoid transferring the post-surgical individual onto a non-high specification support surface unless clinically indicated (*Expert opinion*).

- Inspect the bed for hard edges that may increase pressure or shear as the individual transfers or sits on the edge of the bed. When the individual is ready to sit up consider a support surface that provides easy egress (*Expert opinion*).
- See the guideline chapter on *Support Surfaces* for more information about support surfaces.

Evidence Discussion

Individuals should be placed on a specialty support surface immediately following surgery. Risks and benefits must be evaluated. If the individual is placed on hard surfaces such as gurneys, stretchers, and x-ray tables in the early postoperative period there is a serious risk of flap disruption or necrosis from increased pressure and shear. Any transfer from the specialty support surface should be carefully managed to prevent injury during transfer. Time spent on surfaces that lack adequate pressure redistribution characteristics should be avoided or severely limited. Individuals who have undergone surgical closure of a pressure injury are at higher risk for developing additional pressure injuries. Their reduced mobility and limited positioning options following surgery increase pressure injury risk. Therefore, the support surface that is used postoperatively should be appropriate for preventing new pressure injuries, as well as able to distribute pressure away from the operative site, reduce shear and limit tension on the incision to prevent flap necrosis or delayed healing.

Air fluidized beds are commonly used after surgical repair. Successful use of air fluidized beds following flap surgery was reported in a number of observational studies^{17,23,25,44,58,63} (*Level 4*). However, there is limited evidence on their relative effectiveness compared to other support surfaces. In a small pilot study (n = 37) Finnegan et al. (2008)⁷⁴ compared healing following pressure injury surgery between an air fluidized bed and an alternating pressure air mattress that was modified. Individuals received the specialty support surfaces in conjunction with standardized care during the acute stage of their postoperative recovery and outcome measures were reported on discharge to rehabilitation (mean duration of eight days). In this time, 98% of the individuals receiving the air fluidized bed were assessed as having an intact and healthy surgical site compared with 87% of individuals receiving the alternating pressure air mattress (p = not reported). Patients and health professionals rated both surfaces as comfortable and exceeding expectations. The air fluidized bed was 52% more expensive to use; however, costs may have related to facility-specific procurement arrangements. This was a small study with a short follow-up period and subjective outcomes measured by non-blinded clinical staff (*Level 1*). Additional research in non-surgical populations with Category/Stage III and IV pressure injuries has demonstrated superior reduction in wound size with air fluidized beds compared to standard hospital mattresses⁷⁵⁻⁷⁷ and an alternating pressure air mattress⁷⁸ (*Level 1*). However, there was minimal inter-group comparison in some of these studies, individuals were not followed to complete healing and the generalizability of the results to postoperative individuals was not demonstrated.

Two studies reported successful use of an alternating pressure air mattress for treating individuals following flap surgery^{40,74} (*Levels 1 and 4*). In one of the studies (results above⁷⁴) the alternating pressure air mattress was modified to permanently deflate single mattress cells beneath the surgical site and the alternating pressure function was not used at the flap site. The method to redistribute pressure at the surgical site was not reported. No data were provided on methods used to ensure individuals remained aligned with the deflated portion of the bed (*Level 1*).

Beds with a low-air-loss feature are also commonly used for the post-surgical individual; however, there were no included studies on their use. A small body of evidence reports effectiveness of low air loss beds for preventing new pressure injuries and promoting healing in individuals with pressure injuries.

Equipment procurement

Any support surface system for postoperative use should be procured prior to surgery.^{23,28} Optimally, the individual should be cared for on the specialty support surface prior to surgery to determine the individual's tolerance of the bed (e.g., dyspnea and weightlessness). In one surgical protocol, participants awaiting pressure injury surgery were encouraged to use prone positioning on a specialty support system during the preoperative period in preparation for the recovery period²⁸ (*Level 4*). Appropriate seating support surfaces should also be organized prior to surgery.

Use of appropriate pressure redistribution support surfaces in the home setting or usual living environment is essential for long term recovery and for reducing the risk of recurrence. Prior to surgery, an assessment should be undertaken of the individual's wheelchair, support cushion and other devices (e.g., shower chair). Assistance in attainment of appropriate equipment and education in its maintenance should be offered. Considerations for equipment in the home setting are discussed in the guideline chapter *Support Surfaces*.

18.8: Position and transfer the individual in such a way as to avoid pressure on, and disruption to, the surgical site.
(Good Practice Statement)

Implementation Considerations

Positioning

- Avoid placing any pressure or body weight on the surgical site^{28,30,48,63} (*Level 3 and 4*).
- Maintain bed rest in a flat position as much as possible following flap surgery^{26,40,44,57,58} (*Level 4*).
- Minimize truncal flexion to 30° to 40° (e.g., raising the head of the bed) until the surgical site is sufficiently healed to permit progressive sitting^{44,58} (*Level 4*).
- Use the prone position or position the individual laterally^{27,48} (*Level 4*).
- When positioning the individual off the surgical site is not possible, repositioning should be conducted on an hourly basis⁶³ (*Level 3*).
- Consider using pressure mapping to guide positioning the individual in such a way that all pressure from the surgical site is relieved²² (*Level 4*).
- Be extremely cautious in positioning an individual on a bedpan and removing the bedpan following flap surgery (*Expert opinion*).

Transferring

- When transferring the individual from the operating table immediately following surgery, ensure there is adequate assistance to avoid disruption of the flap. Maintain the same diligence when transferring the individual in the post-surgical healing stages (*Expert opinion*).
- Ensure that the individual is appropriately clothed/protected when using slide boards to prevent injury to the flap (*Expert opinion*).

Discussion

Flaps rely on the blood supply in the tissues that is carried along with the tissues. Blood flow to the flap is provided through the pedicle. Some flaps have deep blood vessels supplying the overlying tissue (e.g., gluteal flaps) and in others the blood flow is more superficial (e.g., latissimus dorsi flaps). This blood supply that feeds the flap (classically called the 'pedicle' of the flap) can be damaged by shear forces (e.g., pulling on the skin) and pressure to the skin. It is important to know where the blood supply is coming and how close to the surface the blood supply is located. The circulation to parts of the flap distal from the pedicle can also be compromised by pressure and shear. This should also be anticipated and managed by the treating physician and the multidisciplinary team.

Positioning

Positioning is generally determined by the surgeon's preferences and the needs of the individual. There was no research providing experimental comparisons between different postoperative positioning following pressure injury surgery.

Following surgery the individual should be positioned in such a way as to avoid pressure on the surgical site.^{28,30,48,63} Numerous observational studies reported that individuals were maintained in a flat position following surgery, usually until the surgical site was sufficiently healed to permit gradual pressure and stress to the site. Until this point of healing is reached, minimize truncal flexion^{44,58} (*Level 4*). Elevating the head of the bed can have unintended consequences on flap healing. Head of bed elevation should only be undertaken with a full understanding of the associated risks and benefits. Many hospitals have initiated evidence-based protocols to limit aspiration pneumonia and ventilator-associated pneumonia by elevating the head of the bed for individuals confined to bed. However, following flap surgery, elevating the head of the bed increases tension on the incision from hip flexion, and increases interface pressure and shear, all of which place the individual at a serious risk of flap disruption or necrosis. It is important to understand the immediate and long term consequences of both options prior to applying a patient specific intervention. When head of bed elevation commences, coordinate with meals and selfcare to promote the individual's function.

Expert opinion on the use of bedpans for individuals with new pelvic flaps varies. They should be used with extreme caution, as they create pressure on the pelvic flap and can create shear forces with removal.

Ongoing repositioning is important to prevent development of new or recurring pressure injuries. Appropriate repositioning options should be planned by the treating physician, and changes to the regimen should be driven by the individual's progress toward healing. Further recommendations on repositioning are provided in the guideline section *Repositioning and Early Mobilization*.

Transferring

Immediately following surgery, it is important to avoid manual handling techniques that involve moving individuals from one surface to another by pulling on the buttocks and hips. Instead, lift the individual from the operating room table onto the bed rather than sliding or pulling. Immediately following surgery and throughout the postoperative period, use of proper manual handling techniques and equipment (e.g., turning sheet, adequate turning team) will limit the need to drag the individual and reduce risk of disrupting the flap from shear and friction. Tension on the suture lines must be avoided when turning the individual in bed. Holding the individual's legs and back is preferred to pulling on the hips and buttocks.

Hospital gowns that are open in the back permit the skin of the thighs and buttocks to drag on transfer devices or slide boards (i.e., for transfer into wheelchairs). Individuals should be adequately clothed to protect the skin during transfers. Clothing with zippers, buttons, or snaps should not be used over the surgical site or pressure points.

18.9: When the surgical site is sufficiently healed commence a progressive sitting protocol.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

Evidence Summary

Two moderate and low quality Level 3 studies^{17,63} and four high, moderate and low quality Level 4^{19,22,23,25,26,58} studies reported post-operative management plans that included initiation of a progressive sitting protocol. The studies reported healing rates of 87% to 96%,^{17,19} complication rates of 10 to 26%,^{19,25,26,58,63} and recurrence rates of between 7% and 25%.^{22,23,26} In these studies, the progressive sitting was commenced at between ten days and eight weeks post-operatively.^{17,19,22,23,25,26,58,63}

Implementation Considerations

- Maintain upper body strength and prevent hazards of immobility and by engaging the individual in passive and active upper body exercises during periods of bed rest^{21,23,25,27} (Level 4).
- Progressive sitting should be initiated according to the surgeon's post-operative orders (*Expert opinion*).
- Initiate a progressive sitting protocol at between two and eight weeks post-operatively based on the individual's clinical condition and the healing of the surgical site^{17,19,22,23,25,26,58,63} (Level 3 and 4).
- Select and use an appropriate pressure redistribution cushion for when the individual is seated out of bed.^{17,23,25,28} (Levels 3 and 4). The guideline chapter on *Support Surfaces* includes more extensive discussion regarding use of pressure redistribution cushions.
- Consider using pressure mapping to assist in selection of the most appropriate pressure redistribution cushion for the individual⁴⁰ (Level 4).
- Teach the individual to initiate pressure relief maneuvers every 15 minutes when engaging in progressive sitting protocol^{23,25} (Level 4). More information about pressure relief maneuvers is included in the guideline chapter *Repositioning and Early Mobilization*.

Evidence Discussion

As noted above, in the immediate postoperative period the individual should be positioned in a way that prevents pressure and tension on the surgical site. The hazards of immobility are well reported. Individuals on pressure redistribution surfaces still require repositioning and skin inspection for new pressure injuries, and the usual postoperative interventions for pulmonary hygiene and to prevent blood stasis.

Range-of-motion exercises of the arms and upper body can reduce the hazards of immobility and start preparing the individual for rehabilitation. In observation studies,^{21,23,25,27} postoperative management protocols were sometimes reported as important components of the individual's regimen. These included passive and active limb mobilization^{21,23,25,27} and upper body strengthening exercises^{23,25,27} (Level 4). However, the studies did not report comparison to other management strategies or provide details of the regimens. Flexion of the hips during upper body mobilization should be avoided until approved by the surgeon.

When weight bearing on the operative site is permitted, it should be graduated and progressive.^{17,63 19,22,23,25,26,58} The postoperative progressive sitting protocol focuses on gradual increase in both pressure and tension being placed on the surgical site. Seating should commence only after a comprehensive assessment of seating support surface needs^{17,23,25,28} (Levels 3 and 4) and selection of an appropriate seat/chair. A seating professional should be involved in assessing the individual, selecting an appropriate chair/wheelchair and pressure redistribution support cushion and developing an individualized supported sitting plan. The guideline chapter on *Support Surfaces* discusses factors to consider when selecting chairs and pressure redistribution cushions. In a report on the outcomes of individuals undergoing flap surgery for Category/Stage IV pressure injuries (n = 45), Ljung et al. (2017)¹⁷ provided a detailed description of a postoperative progressive sitting protocol. At week three, individuals commenced 30 minutes seating in a wheelchair, three times daily on a pressure redistribution cushion. Duration of sitting increased over the week to a maximum of two hours, three times daily. In week four, individuals commenced sitting in bed with the legs extended and the hip angle at 90°, avoiding forward lean. The positioning restrictions were recommended for the first six months following surgery¹⁷ (Level 3). Inclusion of pressure relief maneuvers, which are reported in more detail in the guideline chapter *Repositioning and Early Mobilization*, was reported as a component of some rehabilitation protocols^{23,25} (Level 4).

Sitting should gradually increase in duration if no erythema is noted over pressure points. Skin tolerance to pressure over the surgical site should be assessed after each period of sitting. Refer to the guideline chapter on *Skin and Tissue Assessment* for comprehensive discussion of skin assessment techniques. If healing is slow or other confounding factors exist (e.g., obesity, multiple pressure injuries, or high level of paralysis) then weight bearing may be delayed until incisions are completely healed.

References

1. Espejo E, Andres M, Borrallo RM, Padilla E, Garcia-Restoy E, Bella F. Bacteremia associated with pressure ulcers: a prospective cohort study. *Eur J Clin Microbiol Infect Dis*, 2018; 37(5): 969-975.
2. Bryan CS, Dew CE, Reynolds KL. Bacteremia associated with decubitus ulcers. *Arch Intern Med*, 1983; 143(11): 2093-5.
3. Cafferkey MT, Hone R, Keane CT. Sources and outcome for methicillin-resistant *Staphylococcus aureus* bacteraemia. *J Hosp Infect*, 1988; 11(2): 136-143.
4. Lee KF, Ennis WJ, Dunn GP. Surgical palliative care of advanced wounds. *Am J Hosp Palliat Care*, 2007; 24(2): 154-160.
5. Redelings MD, Lee NE, Sorvillo F. Pressure ulcers: more lethal than we thought? *Adv Skin Wound Care*, 2005; 18(7): 367-372.
6. Wall BM, Mangold T, Huch KM, Corbett C, Cooke CR. Bacteremia in the chronic spinal cord injury population: risk factors for mortality. *J Spinal Cord Med*, 2003; 26(3): 248.
7. Shannon MM. A retrospective descriptive study of nursing home residents with heel eschar or blisters. *Ostomy Wound Manage*, 2013; 59(1): 20-27.
8. AMDA, *American Medical Directors Association. Pressure Ulcers in the Long-Term Care Setting Clinical Practice Guideline*. 2008, Columbia, MD: AMDA.
9. Bergstrom N, Bennett, M.A., Carlson, C.E., et al, *Treatment of Pressure Ulcers. Clinical Practice Guideline, No. 15. AHCPR Pub. No. 95-0653*. 1994, Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, Agency for Healthcare Policy and Research.
10. European Pressure Ulcer Advisory Panel, *Pressure Ulcer Treatment Guidelines*. 1998, EPUAP, : Oxford, England.
11. Keast DH, Parslow N, Houghton PE, Norton L, Fraser C. Best practice recommendations for the prevention and treatment of pressure ulcers: Update 2006. *Adv Skin Wound Care*, 2007; 20(8): 447-60.
12. Wound Ostomy and Continence Nurses Society (WOCNS), *Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: WOCNS.
13. Wounds Australia, *Standards for Wound Prevention and Management*. 2016, Cambridge Media: Osborne Park, WA.
14. van Rijswijk L, Polansky M. Predictors of time to healing deep pressure ulcers. *Ostomy Wound Management*, 1994; 40(8): 40-2, 44, 46-8 passim.
15. Lee E. Longitudinal Outcomes of Home Care in Korea to Manage Pressure Ulcers. *Res Nurs Health*, 2017; 40(3): 255-262.
16. Wolters U, Wolf T, Stutzer H, Schroder T. ASA classification and perioperative variables as predictors of postoperative outcome. *British Journal of Anaesthesia*, 1996; 77: 217-22.
17. Ljung AC, Stenius MC, Bjelak S, Lagergren JF. Surgery for pressure ulcers in spinal cord-injured patients following a structured treatment programme: a 10-year follow-up. *Int Wound J*, 2017; 14(2): 355-359.
18. Huang K, Guo Q. Surgical repair involving tissue flap transplantation with vascular pedicle in treating refractory pressure ulcers around hip and sacral region. *Curr Signal Transduct Ther*, 2015; 10(1): 36-40.
19. Srivastava A, Gupta A, Taly AB, Murali T. Surgical management of pressure ulcers during inpatient neurologic rehabilitation: outcomes for patients with spinal cord disease. *J Spinal Cord Med*, 2009; 32(2): 125-131.
20. Keys KA, Daniali LN, Warner KJ, Mathes DW. Multivariate predictors of failure after flap coverage of pressure ulcers. *Plast Reconstr Surg*, 2010; 125(6): 1725-1734.
21. Di Caprio G, Serra-Mestre JM, Ziccardi P, Scioli M, Larocca F, Nunziata V, Grella R, D'Andrea F. Expanded flaps in surgical treatment of pressure sores: Our experience for 25 years. *Ann Plast Surg*, 2014.
22. Tadiparthi S, Hartley A, Alzweri L, Mecci M, Siddiqui H. Improving outcomes following reconstruction of pressure sores in spinal injury patients: A multidisciplinary approach. *J Plast Reconstr Aesthet Surg*, 2016; 69(7): 994-1002.

23. Kierney PC, Engrav LH, Isik FF, Esselman PC, Cardenas DD, Rand RP. Results of 268 pressure sores in 158 patients managed jointly by plastic surgery and rehabilitation medicine. *Plast Reconstr Surg*, 1998; 102(3): 765-72.
24. Thiessen FE, Andrades P, Blondeel PN, Hamdi M, Roche N, Stillaert F, Van Landuyt K, Monstrey S. Flap surgery for pressure sores: should the underlying muscle be transferred or not? *J Plast Reconstr Aesthet Surg*, 2011; 64(1): 84-90.
25. Grasseti L, Scalise A, Lazzeri D, Carle F, Agostini T, Gesuita R, Di Benedetto G. Perforator flaps in late-stage pressure sore treatment: outcome analysis of 11-year-long experience with 143 patients. *Ann Plast Surg*, 2014; 73(6): 679-85.
26. Ahluwalia R, Martin D, Mahoney JL. The operative treatment of pressure wounds: a 10-year experience in flap selection. *Int Wound J*, 2009; 6(5): 355-358.
27. Estrella EP, Lee EY. A retrospective, descriptive study of sacral ulcer flap coverage in nonambulatory patients with hypoalbuminemia. *Ostomy Wound Management*, 2010; 56(3): 52-59.
28. Singh R, Singh R, Rohilla RK, Magu NK, Goel R, Kaur K. Improvisations in classic and modified techniques of flap surgery to improve the success rate for pressure ulcer healing in patients with spinal cord injury. *International Wound Journal*, 2013; 10(4): 455-60.
29. Bamba R, Madden JJ, Hoffman AN, Kim JS, Thayer WP, Nanney LB, Spear ME. Flap Reconstruction for Pressure Ulcers: An Outcomes Analysis. *Plast Reconstr Surg Glob Open*, 2017; 5(1): e1187.
30. Chiu YJ, Liao WC, Wang TH, Shih YC, Ma H, Lin CH, Wu SH, Perng CK. A retrospective study: Multivariate logistic regression analysis of the outcomes after pressure sores reconstruction with fasciocutaneous, myocutaneous, and perforator flaps. *Journal of Plastic, Reconstructive and Aesthetic Surgery*, 2017; 30.
31. O'Neill DK, Robins B, Ayello EA, Cuff G, Linton P, Brem B. Regional anaesthesia with sedation protocol to safely debride sacral pressure ulcers. *Int Wound J*, 2012; 9: 525-43.
32. Lin S, Hey HWD, Lau ETC, Tan KA, Thambiah JS, Lau LL, Kumar N, Liu KPG, Wong HK. Prevalence and predictors of pressure injuries from spine surgery in the prone position. *Spine (Phila Pa 1976)*, 2017; 42(22): 1730-1736.
33. Schoonhoven L, Defloor T, van der Tweel I, Buskens E, Grypdonck MH. Risk indicators for pressure ulcers during surgery. *Appl Nurs Res*, 2002; 15(3): 163-173.
34. Yoshimura M, Iizaka S, Kohno M, Nagata O, Yamasaki T, Mae T, Haruyama N, Sanada H. Risk factors associated with intraoperatively acquired pressure ulcers in the park-bench position: A retrospective study. *International Wound Journal*, 2015.
35. Chen HL, Shen WQ, Xu YH, Zhang Q, Wu J. Perioperative corticosteroids administration as a risk factor for pressure ulcers in cardiovascular surgical patients: A retrospective study. *Int Wound J*, 2013.
36. Tschannen D, Bates O, Talsma A, Guo Y. Patient-specific and surgical characteristics in the development of pressure ulcers. *Am J Crit Care*, 2012; 21(2): 116-126.
37. Connor T, Sledge JA, Bryant-Wiersema L, Stamm L, Potter P. Identification of pre-operative and intra-operative variables predictive of pressure ulcer development in patients undergoing urologic surgical procedures. *Urol Nurs*, 2010; 30(5): 289-305.
38. EuroQol. *EQ-5D*® 2019 [cited August 2019]; Available from: <https://euroqol.org/>.
39. Schryvers OI, Stranc MF, Nance PW. Surgical treatment of pressure ulcers: 20-year experience. *Arch Phys Med Rehabil*, 2000; 81(12): 1556-1562.
40. Larson DL, Hudak KA, Waring WP, Orr MR, Simonelic K. Protocol management of late-stage pressure ulcers: A 5-year retrospective study of 101 consecutive patients with 179 ulcers. *Plast Reconstr Surg*, 2012; 129(4): 897-904.
41. Isken T, Alagoz MS, Onyedi M, Izmirli H, Isil E, Yurtseven N. Preoperative color Doppler assessment in planning of gluteal perforator flaps. *Ann Plast Surg*, 2009; 62(2): 158-163.
42. Kim YH, Kim SW, Kim JT, Kim CY. Tensor Fascia Lata Flap Versus Tensor Fascia Lata Perforator-Based Island Flap for the Coverage of Extensive Trochanteric Pressure Sores. *Ann Plast Surg*, 2013.
43. Diamond S, Moghaddas HS, Kaminski SS, Grotts J, Ferrigno L, Schooler W. National outcomes after pressure ulcer closure: Inspiring surgery. *Am Surg*, 2016; 82(10): 903-906.
44. Bertheuil N, Huguier V, Aillet S, Beuzeboc M, Watier E. Biceps femoris flap for closure of ischial pressure ulcers. *Eur J Plast Surg*, 2013; 36(10): 639-644.
45. Laing TA, Ekpete N, Oon S, Carroll SM. Surgical reconstruction of pressure ulcer defects: a single- or two-stage procedure? *Journal of Wound, Ostomy, and Continence Nursing*, 2010; 37(6): 615-618.
46. Chang JW, Lee JH, Choi MSS. Perforator-based island flap with a peripheral muscle patch for coverage of sacral sores. *Journal of Plastic, Reconstructive and Aesthetic Surgery*, 2016; 69(6): 777-782.
47. Bonomi S, Salval A, Brenta F, Rapisarda V, Settembrini F. The Pacman Perforator-Based V-Y Advancement Flap for Reconstruction of Pressure Sores at Different Locations. *Ann Plast Surg*, 2016; 77(3): 324-31.
48. Mathur BS, Tan SS, Bhat FA, Rozen WM. The transverse lumbar perforator flap: An anatomic and clinical study. *Journal of Plastic, Reconstructive and Aesthetic Surgery*, 2016; 69(6): 770-776.
49. Tashiro J, Gerth DJ, Thaller SR. Pedicled flap reconstruction for patients with pressure ulcers: Complications and resource utilization by ulcer site. *JAMA Surgery*, 2016; 151(1): 93-94.
50. Kurita M, Ichioka S, Tanaka Y, Umekawa K, Oshima Y, Ohura N, Kinoshita M, Harii K. Validity of the orthopedic POSSUM scoring system for the assessment of postoperative mortality in patients with pressure ulcers. *Wound Repair Regen*, 2009; 17(3): 312-317.
51. Kenneweg KA, Welch MC, Welch PJ. A 9-year retrospective evaluation of 102 pressure ulcer reconstructions. *J Wound Care*, 2015; 24 Suppl 4a: S12-21.
52. Han HH, Ko JG, Rhie JW. Factors for postoperative complications following pressure ulcer operation: Stepwise multiple logistic regression analysis. *Int Wound J*, 2017.

53. Lopez de Heredia L, Hauptfleisch J, Hughes R, Graham A, Meagher T. Magnetic resonance imaging of pressure sores in spinal cord injured patients: Accuracy in predicting osteomyelitis. *Topics in Spinal Cord Injury Rehabilitation*, 2012; 18(2): 146-148.
54. Hauptfleisch J, Meagher TM, Hughes RJ, Singh JP, Graham A, Lopez de Heredia L. Interobserver Agreement of Magnetic Resonance Imaging Signs of Osteomyelitis in Pelvic Pressure Ulcers in Patients With Spinal Cord Injury. *Arch Phys Med Rehabil*, 2013.
55. Luis, Hauptfleisch J, Hughes R, Graham A, Meagher TMM. Magnetic Resonance Imaging of Pressure Sores in Spinal Cord Injured Patients: Accuracy in Predicting Osteomyelitis. *Topics in Spinal Cord Injury Rehabilitation*, 2012; 18(2): 146-148.
56. Daniali LN, Keys K, Katz D, Mathes DW. Effect of preoperative magnetic resonance imaging diagnosis of osteomyelitis on the surgical management and outcomes of pressure ulcers. *Ann Plast Surg*, 2011; 67(5): 520-525.
57. Greco M, Marchetti F, Tempesta M, Ruggiero M, Marcasciano M, Carlesimo B. Cutaneous flaps in the treatment of 338 pressure sores: a better choice. *Ann Ital Chir*, 2013; 84(6): 655-9.
58. Wettstein R, Tremp M, Baumberger M, Schaefer DJ, Kalbermatten DF. Local flap therapy for the treatment of pressure sore wounds. *Int Wound J*, 2013.
59. Marriott R, Rubayi S. Successful truncated osteomyelitis treatment for chronic osteomyelitis secondary to pressure ulcers in spinal cord injury patients. *Ann Plast Surg*, 2008; 61(4): 425-429.
60. Rintala DH, Garber SL, Friedman JD, Holmes SA. Preventing recurrent pressure ulcers in veterans with spinal cord injury: impact of a structured education and follow-up intervention. *Arch Phys Med Rehabil*, 2008; 89(8): 1429-1441.
61. Deshmukh GR, Barkel DC, Sevo D, Hergenroeder P. Use or misuse of colostomy to heal pressure ulcers. *Dis Colon Rectum*, 1996; 39(7): 737-738.
62. de la Fuente SG, Levin LS, Reynolds JD, Olivares C, Pappas TN, Ludwig KA, Mantyh CR. Elective stoma construction improves outcomes in medically intractable pressure ulcers. *Dis Colon Rectum*, 2003; 46(11): 1525-1530.
63. Han HH, Choi EJ, Choi JY, Rhie JW. Efficacy of one-stage surgical treatment and clinical features in patients with multiple pressure ulcers. *Int Wound J*, 2016; 13: 7-12.
64. Sugarman B, Hawes S, Musher DM, Klima M, Young EJ, Pircher F. Osteomyelitis beneath pressure sores. *Archives of Internal Medicine*, 1983; 143(4): 683-8.
65. Thornhill Joynes M, Gonzales F, Stewart CA, Kanel GC, Lee GC, Capen DA, Sapico FL, Canawati HN, Montgomerie JZ. Osteomyelitis associated with pressure ulcers. *Arch Phys Med Rehabil*, 1986; 67(5): 314-8.
66. Yarkin O, Tamer S, Gamze O, Irem M, Huseyin B. Effect of surgery on psychiatric states and quality of life of paraplegics and quadriplegics with pressure sores and their primary caregivers. *Eur J Plast Surg*, 2009; 32(4): 173-176.
67. American Society of Plastic Surgeons. *Evidence-Based Clinical Practice Guideline: Chronic Wounds of the Lower Extremity*. 2011 [cited August 2019].
68. Foster RD, Anthony JP, Mathes SJ, Hoffman WY, Young D, Eshima I. Flap selection as a determinant of success in pressure sore coverage. *Arch Surg*, 1997; 132(8): 868-73.
69. Lemaire V, Boulanger K, Heymans O. Free flaps for pressure sore coverage. *Ann Plast Surg*, 2008; 60(6): 631-634.
70. Perng C-K. Recent advances in postoperative freemicrovascular flap monitoring. *Formosan Journal of Surgery*, 2013; 46(5): 145-148.
71. Poder T, Fortier P-H. Implantable Doppler in monitoring free flaps: A cost-effectiveness analysis based on a systematic review of the literature. *Eur Ann Otorhinolaryngol Head Neck Dis*, 2013; 130(2): 79-85.
72. Foster RD, Anthony JP, Mathes SJ, Hoffman WY. Ischial pressure sore coverage: a rationale for flap selection. *Br J Plast Surg*, 1997; 50(5): 374-379.
73. Black JM. Surgical options in wound healing. *Crit Care Nurs Clin North Am*, 1996; 8(2): 169-182.
74. Finnegan MJ, Gazzero L, Finnegan JO, Lo P. Comparing the effectiveness of a specialized alternating air pressure mattress replacement system and an air-fluidized integrated bed in the management of post-operative flap patients: A randomized controlled pilot study. *Journal of Tissue Viability*, 2008; 17(1): 2-9.
75. Munro BH, Brown L, Heitman BB. Pressure ulcers: One bed or another? How does an air-fluidized bed compare with pads and other devices on a standard bed? *Geriatric Nursing* 1989; 10: 190-2.
76. Jackson BS, Chagares R, Nee N, Freeman K. The effects of a therapeutic bed on pressure ulcers: An experimental study. *J Enterostomal Ther*, 1988; 15(6): 220-226.
77. Strauss MJ, Gong J, Gary BD, Kalsbeek WD, Spear S. The cost of home air-fluidized therapy for pressure sores. A randomized controlled trial. *The Journal of Family Practice*, 1991; 33(1): 52-59.
78. Allman RM, Walker JM, Hart MK, Laprade CA, Noel LB, Smith CR. Air-fluidized beds or conventional therapy for pressure sores. A randomized trial. *Ann Intern Med*, 1987; 107(5): 641-8.

MEASURING PRESSURE INJURY PREVALENCE AND INCIDENCE

Introduction

The effectiveness of the implementation of this guideline in preventing and treating pressure injuries is measurable through well-designed research and/or quality improvement initiatives. These initiatives may rely on some measure of pressure injury frequency. Measures of pressure injury frequency have typically relied on describing the rates and proportions of pressure injury incidence and prevalence. Understanding the basic properties of prevalence and incidence, and how best to use these measures, is essential in evaluating how well the guideline is being implemented. The most commonly used approaches in measuring pressure injury prevalence or incidence are discussed in the chapter.

Defining Prevalence and Incidence

Prevalence

Pressure injury prevalence is the proportion of individuals within a defined population (e.g., individuals within a specific geographic region, a facility or a ward) that have a pressure injury within a defined period of time. Prevalence rates provide insights regarding the burden of pressure injuries and the resources needed to address that burden.

Point prevalence is the number of individuals with a pressure injury at a specific point in time (usually on a specific day). The pressure injuries may have developed recently or may have been present for an extended period of time. For inpatients, these pressure injuries may have been present on admission to the facility.^{1,2}

Figure 23.1: Formula for calculating point prevalence

<p>Point prevalence (%)</p> $\frac{\text{Number of individuals with pressure injury at a specific point in time}}{\text{Total number of individuals in the study population at a specific point in time}} \times 100$
--

Period prevalence is also commonly reported. Period prevalence is the number of individuals who have a pressure injury over a specified period of time (usually days or weeks). Period prevalence is often used in preference to point prevalence because of the time it takes to collect data for a pressure injury prevalence study. Like point prevalence, period prevalence describes all existing pressure injuries rather than just newly acquired pressure injuries. However, period prevalence identifies pressure injuries present during a specified time period rather than at a specific point in time. Period prevalence is therefore a combination of prevalent and incident pressure injuries.^{1,2} When reporting prevalence determined over a period of time, it should be referred to as **period prevalence** and the time period should be specified.

Incidence

Pressure injury incidence is the proportion of pressure injury free individuals that develop a pressure injury over a specific period of time. Incidence therefore provides an indication of the rate at which new pressure injuries occur in a specified population. Incidence rates provide insights into the effectiveness of prevention measures; however, incidence studies are more labor intensive and costly to implement than prevalence studies.

Cumulative incidence is the proportion of a specified population that develops a new pressure injury within a specified time period (usually weeks or months). In calculating cumulative incidence, a population free of pressure injuries is identified and then followed for a specified time period, with periodic determinations of the presence of pressure injuries for each individual.^{1,2}

Figure 23.2: Formula for calculating cumulative incidence

<p>Cumulative incidence (%)</p> $\frac{\text{Number of individuals developing pressure injury during a specific time period}}{\text{Total number of individuals in study population over a specific time period}} \times 100$
--

Incidence density is also sometimes used as a measure of pressure injury incidence. In using this measure, the denominator is not the total number of patients. Instead, the number of patient days of care in the facility or service is used. This calculation better accounts for length of stay and is particularly useful in long-term care facilities where individuals may reside for months or years. Incidence density is often described as the number of new pressure injury cases per 1,000 patient days of care.

Figure 23.3: Formula for calculating incidence density of individuals per time

<p>Incidence density (No. per 1000 patient days)*</p> $\frac{\text{Number individuals developing a new pressure injury}}{\text{Total patient-days without a pressure injury}}$ <p>* If reporting per 1,000 patient days, multiply by 1,000</p>

Facility-acquired Pressure Injuries

Facility-acquired pressure injury (FAPI) rates measure the number of individuals with pressure injuries at a specific point in time that were acquired within a given facility (also referred to as nosocomial, hospital-acquired [HAPI] or healthcare-acquired pressure injuries). Unlike point prevalence, this term describes only those individuals with pressure injuries that were acquired within the facility after admission. Facility-acquired pressure injury rates are often determined in conjunction with point prevalence. Existing pressure injuries are documented to determine prevalence rates. Admission documentation is then examined to determine if the pressure injury was present on admission. If not documented on admission, the pressure injury is considered to be facility-acquired. Facility-acquired pressure injury rates are often calculated to evaluate the effects of prevention protocols in decreasing the pressure injuries acquired in the facility without undertaking a more time consuming and expensive incidence study. Examining admission records to differentiate facility-acquired versus community-acquired pressure injuries as part of a point prevalence study can provide insights into the burden of pressure injuries, the required resources (prevalence), and the effectiveness of prevention protocols with the facility (FAPI).

Figure 23.4: Formula for calculating facility-acquired pressure injury rate

<p>Facility-Acquired (%) †</p> $\frac{(\text{No. individuals with pressure injury at time point}) - (\text{No. individuals with pressure injury on admission})}{(\text{No. in population at time point}) - (\text{No. with pressure injury on admission})} \times 100$ <p>† Some FAPI methods include individuals with a pressure injury on admission if they also develop a new pressure injury after admission</p>

An accurate FAPI rate requires an accurate, documented skin assessment on admission to the facility for all individuals in the defined population in order to exclude pre-existing pressure injuries. The time frame for admission documentation may vary between studies. Some methodologies for FAPI consider those with a pressure injury present-on-admission (POA) as a case and exclude those individuals from FAPI calculations, regardless of new additional pressure injuries. Other methodologies include individuals with POA pressure injuries who develop new pressure injuries following admission. Variations in methodology should be considered before interpreting the results of any study and especially when comparing results between studies.^{1,3}

Interpreting Pressure Injury Rates

When interpreting pressure injury prevalence and incidence rates, there is no best approach. Different approaches tell you something different and will vary depending on the purpose of measurement and the intended use of the data. Data collected during prevalence and incidence studies is used for a variety of purposes, including to:

- Estimate disease prevalence
- Determine resource needs
- Monitor effectiveness of quality improvement initiatives
- Benchmark among facilities
- Report quality indicators to stakeholders
- Calculate reimbursement rates that are linked to quality indicators.

By necessity, larger databases used for benchmarking within a country or across countries are less granular or detailed and depend more heavily on medical record documentation than bedside observation. The method developed

or selected should strive for the best balance of reliability, validity, responsivity to changing rates over time and practicality, with consideration to the intended use of the data. Pressure injury e-measures that rely on data extracted from electronic health records (EHRs) are currently being developed; however, reliability and validity concerns have not been fully addressed.^{4,5}

When used for internal or external benchmarking, consistency in the methods being compared is critical. Rates determined by different approaches or using different data sources are not comparable. No method is more correct; however, FAPI rates provide a better indication of the effectiveness of pressure injury prevention programs than raw prevalence rates. Incidence measures are even more suitable to measure effectiveness. Interpretation of prevalence and incidence studies is complicated by:^{1,2,6}

- The method used to calculate pressure injury rates (e.g., prevalence versus incidence versus facility-acquired rates)
- Criteria used to define the population (e.g., clinical setting, type of individual and their pressure injury risk, inclusion/exclusion criteria)
- Definitions and classifications used for pressure injuries
- Inclusion or exclusion of Category/Stage I pressure injuries, deep tissue pressure injuries, unstageable pressure injuries and medical device related pressure injuries (MDRPIs)
- Strategies used to determine presence of a pressure injury (e.g., clinical assessment, patient self-report or medical record review)
- Accuracy of pressure injury classification and differential diagnosis
- Methods and time frames used in determining whether a pressure injury was POA
- Variations in time periods over which studies are conducted
- Random variation
- In some settings, whether the pressure injuries were considered avoidable or unavoidable, and the criteria used to make that determination.

Experience in both the UK and the US illustrates the hazards of not considering these complicating factors. In the UK, monitoring of the quality and safety of National Health Service (NHS) Trusts includes several different reporting systems for pressure injuries. The Safety Thermometer assesses pressure injury prevalence monthly, the Incident Reporting System is intended to capture new pressure injury incidents, and the Strategic Executive Information System captures those pressure injuries that are serious incidents. Yet when compared to a pressure injury wound audit that involved direct examination of patients' skin, the sensitivity of both the Safety Thermometer and the Incident Reporting System was around 50%, suggesting that both these measures missed many cases of pressure injuries.⁷ Among the reasons for poor performance included NHS Trusts varying in their inclusion of MDRPIs, unstageable pressure injuries, and deep tissue pressure injuries. Differences across the NHS Trusts in the definition of POA was also an issue⁸ In the US, government agencies use hospital pressure injury rates in a variety of different programs. The Centers for Medicare and Medicaid Services use administrative codes to calculate hospital-acquired pressure injury rates as part of its Hospital-Acquired Conditions Initiative. This initiative ensures hospitals are not reimbursed for care of individuals with medical complications. The initiative's Hospital-Acquired Condition Reduction Program penalizes hospitals with high complication rates.⁹ Meanwhile, the Agency for Healthcare Research and Quality tracks hospital-acquired complications, including pressure injuries, through medical record reviews as part of the Medicare Patient Safety Monitoring System. When comparing the different data sources, the pressure injury rate calculated from administrative data was approximately one-twentieth the rate based on medical record data. These international experiences demonstrate the ongoing issues in interpreting pressure injury rates.

Summary of Prevalence and Incidence Rates

These variations in methodological design and rigor continue to confound analysis of prevalence and incidence studies. There is a strong need for consistency in design and reporting in order to enable more reliable international benchmarking. Particularly where the effectiveness of pressure injury prevention programs is being investigated, facility-acquired pressure injury rates should be reported. *Table 23.1* provides indicative data on the prevalence and incidence rates reported in the literature since 2000 for different clinical settings and/or populations. Where available, data from recent systematic reviews has been used. Whilst this review included multiple studies and systematic reviews, a range of values inclusive of all studies is reported below.

Table 23.1: Ranges of pressure injury prevalence and incidence reported in selected literature

Setting or Population		Prevalence Rates	Incidence/ FAPI Rates
Acute care		6 to 18.5% ¹⁰	0% ¹¹ to 12% ¹¹
Critical care		95% CI: 10.0 to 25.9% ¹²	95% CI: 16.9 to 23.8% ¹²
Aged care		4.1% ¹³ to 32.2% ¹⁴	1.9% ¹⁵ to 59% ¹⁶
Pediatric care	Primary health care	1.75% (95% CI: 1.71 to 1.73) ¹⁷	--
	General acute care	1.8% ¹⁸ to 4.0% ¹⁸	0.57% ²⁰ to 21.4% ¹⁸
	Critical care	32.8% ¹⁸	0.25% ²¹ to 27% ²²
	Mixed settings	0.47% ¹⁹ to 7.1% ¹⁸	0.29% ¹⁹ to 27.7% ²³
Operating room		—	5% ²⁴ to 53.4% ²⁵

Tubaishat et al. (2015)¹⁰ reported a meta-analysis of pressure injury prevalence studies conducted in acute care settings (excluding studies that reported mixed clinical settings). Ten studies that used the EPUAP Classification System reported prevalence ranging from 7.8% to 54% when Category/Stage I pressure injuries were included. Prevalence was 3.4% to 20.3% when Category/Stage I pressure injuries were excluded. Eight studies that used the NPUAP Classification System reported prevalence ranging from 6% to 22% when Category/Stage I pressure injuries were included, and 6% to 11% excluding Category/Stage I pressure injuries. The researchers combined data from these studies with one additional study that used the Torrance Classification System to estimate the international pressure injury prevalence rate reported in *Table 23.1*. This estimation was based on prevalence rates reported in prospective studies that included Category/Stage I pressure injuries and in which trained observers conducted skin assessments, with outlier results removed.¹⁰

Chaboyer et al. (2018)¹² conducted a meta-analysis of pressure injury data from 10 studies (8,168 individuals) reporting cumulative incidence and 8 studies (13,144 individuals) reporting pressure injury prevalence. The studies included in the review were conducted in critical care settings internationally and ranged in methodological quality. The majority of the studies (70% of the incidence studies and 100% of the prevalence studies) reported that pressure injuries were identified by skin inspections. When limiting the incidence data analysis to those studies that used skin inspection, the 95% confidence interval (CI) was 9.4% to 27.5%. When limiting the analysis to studies evaluated as having a low risk of bias, the 95% CI for cumulative incidence was 6.6% to 36.8% (n = 4 studies, 640 individuals) and 95% CI for prevalence was 12.2% to 24.5% (n = 5 studies, 4,036 individuals).¹²

The range of pressure injury prevalence and incidence observed in pediatric populations is somewhat varied according to the clinical setting, as presented in *Table 23.1*. Few prevalence and incidence studies conducted in pediatric populations are reported in the literature; however, two of the more recent surveys have surveyed large populations. Razmus et al. (2017)²⁰ surveyed 678 pediatric facilities in the US (n = 39,984), reporting FAPI rates of between 0.57% (all settings combined) to 3.7% (pediatric intensive care unit [PICU]) and 4.6% (rehabilitation settings).²⁰ Montserrat Sánchez-Lorente et al. (2018)¹⁷ surveyed 65,359 individuals in Spanish primary health care settings, reporting a prevalence rate of 1.75% (95% CI 1.71 to 1.73).¹⁷ Most studies report that the majority of pressure injuries are Category/Stage I.^{18,19,22,23} For example, in the study by Curley et al. (2003)²² set in a PICU, 97% of pressure injuries were Category/Stage I or II, and in the study by de Souza et al. (2017)¹⁸ in Brazil, 55% of the pressure injuries were Category/Stage I. However, the incidence rate of 0.25% that Murdoch et al. (2002)²¹ reported in a PICU only considered Category/Stage III and IV pressure injuries. Because the various pressure injuries staging systems used across geographic locations and over time have varied, direct comparison of prevalence and incidence rates across studies may not be accurate. Across the studies, there appears to be a consistent trend for younger children to have higher prevalence and incidence rates. Curley et al. (2003)²² calculated an odds ratio of 1.27 (95% CI 1.02 to 1.57, p = 0.03) of experiencing a pressure injury compared to a child in an older age group. The exception to this was in the study conducted in primary health care¹⁷ in which the 1 to 30 day age range experienced the lowest incidence of all children (0.72% compared with 1.6% in the 9 to 18 year age range).¹⁷ For hospitalized children, MDRPIs remain a significant challenge, with one study reporting as many as 84% of pressure injuries to be associated with a device.²³ The guideline chapter on *Device Related Pressure Injuries* includes discussion of higher risks for pediatric populations.

In aged care, ranges of both prevalence and incidence remain unchanged since the previous guideline. The largest most recent study²⁶ was a population-based prevalence study conducted across one geographic region in Japan (n = 37,855 people, 37.7% aged 65 years or older). Prevalence was derived from a mandatory public database containing records for all individuals receiving community or facility based long term care. The prevalence rate of pressure injuries was 20.3 per 1,000 population in those aged 65 years or over (95% CI 18.1 to 22.7), and 44.6 per 1,000 population in those aged 80 years or over (95% CI 39.5 to 50.2). This was substantially higher than that in the population aged 18 to 64 years (9.2 per 1,000 population, 95% CI 39.5 to 50.2), and demonstrates the increasing risk of pressure injuries

associated with aging.²⁶ Other recent studies^{27,28} set in aged care have highlighted a higher rate of pressure injuries in individuals with darker skin. In a database prevalence study in aged care the US (n = 116,460 admissions), prevalence was 14% overall (excluding Category/Stage I pressure injuries). However, dark skinned individuals had a prevalence rate 1.7 times higher than light skinned individuals.²⁷ This finding might introduce additional considerations when making comparisons of pressure injuries prevalence and incidence across geographic regions. The guideline chapter on *Skin and Tissue Assessment* discusses assessing darker toned skin.

Undertaking Prevalence and Incidence Studies

Pressure injury prevalence and incidence studies provide valuable data to drive:

- Quality improvement on a ward/unit and facility level
- Policy decisions on a national level
- Research agendas on an international scale.

As discussed in this chapter, significant variations in study methods and methodological rigor limit the value of prevalence and incidence data in directing quality, policy and future research. The following recommendation and implementation considerations are based on sound epidemiological principles and are designed to guide greater consistency and rigor in the design, implementation and reporting of pressure injury prevalence and incidence studies in clinical settings.

19.1: Use a rigorous methodological design and consistent measurement variables when conducting and reporting pressure injury prevalence and incidence studies.
(Good Practice Statement)

Implementation Considerations

- Compare results against organizational, national and/or international data sets that used a similar methodology to develop a clearer understanding of pressure injury prevalence and incidence (*Expert opinion*).
- Use FAPI rates rather than prevalence rates to evaluate pressure injury prevention programs (*Expert opinion*).
- Present results by pressure injury risk level when reporting prevalence and incidence studies (*Expert opinion*).
- Include the common anatomical locations of pressure injuries when reporting prevalence and incidence studies (*Expert opinion*).
- Present prevalence and incidence data by the pressure injury Category/Stage. Clearly indicate whether:
 - Category/Stage I pressure injuries were included or excluded in the final calculation of prevalence and incidence rates
 - Suspected deep tissue injuries and unstageable pressure injuries were included or excluded in the overall prevalence and incidence rate and if so, how they were considered (e.g., combined with another Category/Stage or as a separate designation) (*Expert opinion*).
- Include mucosal membrane pressure injuries in prevalence and incidence data. However, these pressure injuries should not be categorized/staged. (*Expert opinion*).
- Determine whether pressure injuries were related to devices. Device related pressure injuries should be reported by Category/Stage (*Expert opinion*).

Discussion

Prevalence rates include all individuals in the facility/health service with pressure injuries, including those with pressure injuries that were POA to the health service. Facility-acquired pressure ulcer rates identify individuals with pressure injuries that developed after admission; therefore, these rates provide a better estimate of the adequacy of pressure injury preventive care within a given facility. Prospective incidence measures would provide an even more accurate evaluation of prevention; however, this methodology is often too resource intensive for facilities to implement.

Prevalence and incidence studies should clearly report their methodological design. Attempts should be made to use a standardized methodology to allow risk adjustment and benchmarking. A rigorous study should include:

- Clear definition of the study population prior to collecting data
- Provision of surveyor education
- Establishment of interrater reliability
- Skin inspections to categorize/stage pressure injuries
- Two surveyors per skin inspection.

Prevalence rates based on medical record audits or administrative data sources may be less reliable than data obtained from skin inspections conducted by qualified health professionals.²⁹

A simple description of pressure injury rates within various pressure injury risk levels may help refine quality improvement initiatives. It allows for more accurate comparison between facilities and may serve as a basis for risk adjustment. It is useful to distinguish population features that might relate to pressure injury risk (e.g., mean age) in clinical settings that incorporate varying population profiles (e.g., critical care, aged care and pediatric units). A description of the population serviced by the facility can also assist in comparison (e.g., specifying the type of 'aged care facility', such as older adults in supported care versus high level aged care).

Reporting results by Category/Stage is critical to a full understanding of pressure injury concerns within a facility. Reporting pressure injury prevalence by anatomical location (e.g., sacrum, heels and occiput) can assist in identifying components of a pressure injury prevention program that may require more intensive resources and/or education. Several authors have noted reassuring declines in overall pressure injury rates yet increases in Category/Stage III and IV pressure injuries.^{9,30} When identified, these patterns and trends require a more targeted approach to preventing more severe pressure injuries.

The term 'device-related' describes an etiology rather than the severity of the pressure injury. Device related pressure injuries should be categorized/staged, with the exception of those occurring on mucosal membranes. Identifying the related device may provide additional guidance for quality improvement decisions related to selection and use of devices (see the guideline chapter on *Device Related Pressure Injuries* for more discussion). Mucosal membrane pressure injuries should be recorded, but not categorized/staged. Refer to the guideline chapter *Classification of Pressure Injuries* for further discussion.

References

1. International Guidelines, Pressure Ulcer Prevention: Prevalence and Incidence in Context. A Consensus Document. 2009, MEP Ltd London.
2. Berlowitz D, *Prevalence, Incidence, and Facility-Acquired Rates, in Pressure Ulcers: Prevalence, Incidence, and Implications for the Future.*, B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
3. Bergquist-Beringer S, Davidson J, Cuddigan J. *Pressure Injury Training 6.0.* 2019 [cited October 2019; Available from: <https://members.nursingquality.org/NDNQIPressureUlcerTraining/>].
4. Warren JJ, Dunton N, eMeasure Development Process for NQF Recognition to be used by NDNQI. 2014, American Nurses Association: Silver Springs, MD.
5. National Quality Forum, Patient Safety, Spring 2019 Cycle, CDP Report: Draft Report for Comment. US Department of Health and Human Services: Washington, DC.
6. Black J, Langemo D, *Pressure Ulcer Staging/Categorization, in Pressure Ulcers: Prevalence, Incidence, and Implications for the Future,* B. Pieper and National Pressure Ulcer Advisory Panel, Editors. 2012, NPUAP: Washington, DC.
7. Smith IL, Nixon J, Brown S, Wilson L, Coleman S. Pressure ulcer and wounds reporting in NHS hospitals in England part 1: Audit of monitoring systems. *Journal of Tissue Viability*, 2016; 25(1): 3-15.
8. Coleman S, Smith IL, Nixon J, Wilson L, Brown S. Pressure ulcer and wounds reporting in NHS hospitals in England part 2: Survey of monitoring systems. *Journal of Tissue Viability*, 2016; 25(1): 16-25.
9. Smith SK, Snyder A, McMahon L, Petersen L, Meddings J. Success in hospital-acquired pressure ulcer prevention: A tale in two data sets. *Health Affairs*, 2018; 37(11): 1787-1796.
10. Tubaishat A, Papanikolaou P, Anthony D, Habiballah L. Pressure ulcers prevalence in the acute care setting: A systematic review, 2000-2015. *Clin Nurs Res*, 2018; 27(6): 643-659.
11. Bales I, Padwojski A. Reaching for the moon: achieving zero pressure ulcer prevalence. *Journal of Wound Care*, 2009; 18(4): 137-144.
12. Chaboyer WP, Thalib L, Harbeck EL, Coyer FM, Blot S, Bull CF, Nogueira PC, Lin FF. Incidence and prevalence of pressure injuries in adult intensive care patients: A systematic review and meta-analysis. *Critical Care Medicine*, 2018; 07: 07.
13. Wilborn D, Grittner U, Dassen T, Kottner J. The National Expert Standard Pressure Ulcer Prevention in Nursing and pressure ulcer prevalence in German health care facilities: a multilevel analysis. *Journal of Clinical Nursing*, 2010; 19(23-24): 3364-71.
14. Abel RL, Warren K, Bean G, Gabbard B, Lyder CH, Bing M, McCauley C. Quality improvement in nursing homes in Texas: results from a pressure ulcer prevention project. *Journal of the American Medical Directors Association*, 2005; 6: 181-8.
15. Igarashi A, Yamamoto-Mitani N, Gushiken Y, Takai Y, Tanaka M, Okamoto Y. Prevalence and incidence of pressure ulcers in Japanese long-term-care hospitals. *Archives of Gerontology and Geriatrics*, 2013; 56(1): 220-226.

16. Geyer M, Brienza D, Karg P, Trefler E, Kelsey S. A randomized control trial to evaluate pressure-reducing seat cushions for elderly wheelchair users. *Advances in Skin & Wound Care*, 2001; 14: 120-9.
17. Montserrat Sánchez-Lorente M, Sanchis-Sánchez E, García-Molina P, Balaguer-López E, Blasco JM. Prevalence of pressure ulcers in the paediatric population and in primary health care: An epidemiological study conducted in Spain. *Journal of Tissue Viability*, 2018; 27(4): 221-225.
18. de Souza Pellegrino DM, Ferreira Chacon JM, Blanes L, Ferreira LM. Prevalence and incidence of pressure injuries in pediatric hospitals in the city of Sao Paulo, SP, Brazil. *Journal of Tissue Viability*, 2017; 26(4): 241-245.
19. Baldwin KM. Incidence and prevalence of pressure ulcers in children. *Advances in Skin & Wound Care*, 2002; 15(3): 121-4.
20. Razmus I, Bergquist-Beringer S. Pressure Injury Prevalence and the Rate of Hospital-Acquired Pressure Injury Among Pediatric Patients in Acute Care. *J Wound Ostomy Continence Nurs*, 2017; 44(2): 110-117.
21. Murdoch V. Pressure care in the paediatric intensive care unit. *Nursing Standard*, 2002; 17(6): 71-4, 76.
22. Curley M, Quigley S, Lin M. Pressure ulcers in pediatric intensive care: incidence and associated factors. *Pediatric Critical Care Medicine*, 2003; 4(3): 284-90.
23. Schluer AB, Cignacco E, Muller M, Halfens RJ. The prevalence of pressure ulcers in four paediatric institutions. *Journal of Clinical Nursing*, 2009; 18(23): 3244.
24. Conner T, Sledge JA, Bryant-Wiersema L, Stamm L, Potter P. Identification of pre-operative and intra-operative variables predictive of pressure ulcer development in patients undergoing urologic surgical procedures. *Urologic Nursing*, 2010; 30(5): 289-95,305.
25. Schuurman JP, Schoonhoven L, Keller BP, van Ramshorst B. Do pressure ulcers influence length of hospital stay in surgical cardiothoracic patients? A prospective evaluation. *Journal of Clinical Nursing*, 2009; 18(17): 2456-63.
26. Nakashima S, Yamanashi H, Komiya S, Tanaka K, Maeda T. Prevalence of pressure injuries in Japanese older people: A population-based cross-sectional study. *PLoS ONE*, 2018; 13 (6) (no pagination)(e0198073).
27. Harms S, Bliss DZ, Garrard J, Cunanan K, Savik K, Gurchich O, Mueller C, Wyman J, Eberly L, Virnig B. Prevalence of pressure ulcers by race and ethnicity for older adults admitted to nursing homes. *Journal of Gerontological Nursing*, 2014; 40(3): 20-26.
28. Bliss DZ, Gurchich O, Savik K, Eberly LE, Harms S, Mueller C, Wyman JF, Garrard J, Virnig B. Are There racial-ethnic disparities in time to pressure ulcer development and pressure ulcer treatment in older adults after nursing home admission? *J Aging Health*, 2014.
29. Prentice J, An evaluation of clinical practice guidelines for the prediction and prevention of pressure ulcers, in School of Surgery and Pathology, Faculty of Medicine, Dentistry and Health Science. 2007, The University of Western Australia.
30. Kayser SA, VanGilder CA, Lachenbruch C. Predictors of superficial and severe hospital-acquired pressure injuries: A cross-sectional study using the international pressure ulcer prevalence survey. *International Journal of Nursing Studies*, 2018; 89: 46-52.

IMPLEMENTING BEST PRACTICE IN CLINICAL SETTINGS

Introduction

Research associated with pressure injury prevention and treatment has grown exponentially over the past two decades, as has the commitment by policy developers, educators and healthcare administrators to promote and implement best practice. However, there is still a gap between research and practice. Knowledge transfer has its own body of research aimed at exploring effective strategies for translating research evidence into practice. Synthesizing information from the implementation literature illuminates strategies that have contributed to successful best practice implementation based on barriers and facilitators described in literature.

Research conducted at a national level has shown that an organization's ongoing involvement in quality improvement initiatives is associated with lower pressure injury incidence, although other organizational factors also play a role.¹⁻³ Lahmann et al. (2010)¹ reported that aged care facilities (n = 60) and hospitals (n = 82) in Germany that were repeatedly involved in national level quality improvement initiatives were more likely to have lower facility-acquired pressure injuries rates than facilities without a quality improvement program. However, the difference in pressure injury incidence only reached significance in acute care hospitals that maintained their initiatives for at least three years (mean 10.2% versus mean 5.2%, $p < 0.05$).¹ In the US, an analysis from the National Database of Nursing Quality Indicators® (NDNQI®) noted that hospitals that had attained Magnet status¹ were more likely to have lower facility-acquired pressure incidence rates than non-Magnet facilities. However, when the work environment of individual units/wards was considered, the difference was not significant.² In a study conducted in nursing homes in the US (n = 35 facilities, n = 1,065 nurses), Berlowitz et al. (2003)⁴ reported that implementation of a quality improvement program was associated with increased workplace satisfaction for nursing staff (mean improvement of 0.83 on a 5-point scale, $p < 0.001$) and increased adoption of clinical guidelines ($p < 0.001$). These improvements did not translate to adherence to best practice ($p = 0.37$) or statistically significant changes in pressure injury incidence ($p = 0.19$), although there was a trend toward lower rates.⁴ Also in the US,³ a retrospective review of hospitals and medical centers (n = 55) noted that engagement in quality improvement initiatives doubled within the audited facilities over the five-year period up to 2012. Although this engagement with quality improvement was associated with significant decreases in facility-acquired pressure injuries, the single greatest factor associated with pressure injury reduction was changes to funding models occurring over the same time period.³ The findings of these national level studies suggest that at the organizational level, a quality improvement program designed to facilitate implementation of best practice requires an innovative workplace culture⁴ and sustained engagement to achieve stronger results.

This chapter of the guideline includes recommendations on how best an organization can facilitate sustained implementation of best practice, including **facilitators and barriers** to this process. The recommendations identify actions that can be implemented at the organization or professional level. Organizational implementation is a multi-level construct that refers to strategies with a focus on the shared readiness of organization members to implement a change, and the shared belief in their collective capability to do so. At a professional level, implementation focuses on strategies that relate to individual professionals. A sound underpinning at both the organizational and professional level is essential for effective introduction and ongoing promotion of best practice (as outlined in this guideline) in prevention and treatment of pressure injuries.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What organizational level interventions/quality improvement programs are effective in attaining sustained pressure injury prevention?
- What are the professional and organizational level components of interventions/quality improvement programs that are effective in attaining sustained pressure injury prevention?
- What organizational level factors facilitate or are barriers to implementing best practice in pressure injury prevention and treatment?

Assessing Facilitators and Barriers to Best Practice

Before developing a quality improvement plan it is critical to assess the environment in which the plan will be delivered. This includes identifying strengths within the organization that can be leveraged when introducing the quality improvement plan, as well as weakness in the organization that could impede effective implementation of the plan. The research identified the following organizational level factors as the primary issues that can facilitate practice or create barriers:

- Staffing characteristics (e.g., the skills mix in the facility)⁵⁻¹⁴
- Knowledge¹⁵⁻¹⁸ and attitudes¹⁹⁻²¹ of the organization's workforce
- Access to appropriate equipment and resources.^{12,18,19,22,23}

Other barriers may be present that require attention, and the facility may have other strengths that will assist in guideline implementation. Barriers and facilitators are specific to the organization; therefore, assessment at a local level is required in order to develop an implementation plan that meets the facility's needs.

20.1: At an organizational level, assess and maximize workforce characteristics as part of a quality improvement plan to reduce pressure injury incidence.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

The recommendation to assess and maximize workforce characteristics is underpinned by several studies that provided evidence that the skills mix (i.e., ratio of registered nurses to licensed/enrolled nurse) and staffing levels contributes to the pressure injury incidence. Two low quality Level 3 studies⁵⁻⁷ demonstrated that understaffing,⁷ number of registered nurses per resident per day⁶ and number of hours of care by a licensed practical nurse (LPN)⁵ are prognostic factors for developing a pressure injury. A low quality Level 3 study,²⁴ and moderate^{8,9,25} and low quality¹⁰ Level 4 studies also demonstrated relationships between workforce characteristics and pressure injury incidence. Higher pressure injury rates were associated with the organization having with fewer qualified nurses, fewer nursing hours and lower rates of staff permanency. Two low quality Level 3 studies^{5,24} and three Level 4 studies^{11,25,26} showed that workforce characteristics (including skills mix, number of registered nurse working hours and staff permanency) were not statistically significantly associated with pressure injury incidence.

Implementation Considerations

- Increasing the number of hours of care provided by qualified nurses might improve implementation of a quality improvement program and decrease pressure injury incidence^{6,8,24,25} (*Levels 3 and 4*).
- Ensure there is a sufficient number of qualified staff to provide consistent, preventive care to individuals at risk of pressure injuries (*Expert opinion*). Ensure there is appropriately educated health professionals to provide clinical leadership in pressure injury prevention and management (see *Recommendation 20.9*).

Evidence Discussion

Workforce characteristics includes skills mix, staffing levels and workforce permanency. Staffing hours, number of hours of care provided by qualified (registered/licensed/enrolled) nurses and the level of permanency of staff are factors that influence pressure injury prevention and treatment that have been identified as a potential barrier to implementing best practice by health professionals^{12-14,25,27} (*Levels 4 and 5*).

Cohort studies have identified some workforce characteristics as prognostic factors for pressure injuries. Patrician et al. (2017)⁵ explored the relationship between pressure injuries and variations in nursing care hours in 69 hospitals in the US. In medical-surgical units, the number of hours of care/patient/day provided by an LPN on day three was significantly predictive of pressure injury development (hazard ratio [HR] = 0.27, $p < 0.01$). However, hours of care provided by a registered nurse (RN) and nursing assistants were not associated with pressure injury rates, and there were no associations between workforce characteristics and pressure injuries in the critical care units ($n = 13$) in this study⁵ (*Level 3*). Konetzka et al. (2009)⁶ used an online survey to assess workforce characteristics in US aged care facilities ($n = 1,366$), including skills mix and number of RN hours/resident/day. After adjusting for resident clinical conditions and facility level factors (e.g., Medicare status), there was a significant decrease in pressure injury prevalence associated with increased RN hours/resident/day ($p < 0.05$), but not with overall skills mix ($p > 0.05$) (*Level 3*). Hart and Davis (2011)²⁴ assessed staffing characteristics in five US hospitals and also reported a significant relationship between pressure injury prevalence and RN hours/patient/day ($r = -0.525$, $p < 0.05$), total nursing hours/patient ($r = -0.485$,

$p < 0.05$), and total RN hours staffed by agency nurses ($r = 0.586$, $p = 0.022$) (*Level 3*). Observational studies (*Level 4*) conducted in nursing homes also suggested that rates of staffing with registered nurses,⁸ the length of time the nursing home administrator had been in the role ($p < 0.05$),¹⁰ and the length of time the director of nursing had been in the role ($p < 0.05$)¹⁰ have statistically significant influences on the incidence of pressure injuries. The conflicting findings from some of these studies may relate to the type of unit (e.g., fewer statistically significant relationships were established in critical care units).^{5,24} Further research in this area is required.

20.2: At the organizational level, assess the knowledge health professionals have about pressure injuries to facilitate implementation of an education program and a quality improvement program.
(Strength of Evidence = B1; Strength of Recommendation = ↑)

20.3: At an organizational level, assess and maximize workforce attitudes and cohesion to facilitate implementation of a quality improvement program.
(Good Practice Statement)

Evidence Summary

The recommendation to assess staff knowledge to facilitate education and quality improvement programs is supported by three studies providing high quality Level 1 evidence¹⁵ and low quality Level 2 evidence.^{16,17} In all three studies,¹⁵⁻¹⁷ knowledge survey results were used to develop organization-specific education interventions as a component of multi-faceted quality improvement programs that achieved reductions in pressure injury incidence. Additionally, one low quality Level 2 study²⁸ that demonstrated significant reduction in pressure injury incidence implemented a multi-faceted health professional education program that was based on the results of a knowledge assessment. In addition, evaluation and maximization of workforce attitudes and cohesion is reflective of best practice.

Implementation Considerations

- Use tools that have been shown to have good psychometric properties to assess pressure injury knowledge. The guideline chapter on *Health Professional Education* provides information on tools used to assess staff knowledge.
- The Attitude Towards Pressure Ulcer Prevention Instrument (APuP) has good validity in measuring workforce attitudes towards pressure injury prevention^{29,30} (*Level 5*).
- Use the results of organizational level knowledge surveys to develop targeted education initiatives to meet the knowledge needs of the workforce²⁸ (*Level 2*).

Evidence Discussion

Knowledge

Assessment of health professionals' knowledge related to pressure injury prevention and treatment identifies potential barriers to mitigate, or facilitators to enhance, when introducing education programs or quality improvement programs. Understanding the knowledge needs of health professionals provides information that assists in the development of organization-specific education and training initiatives.^{15,16,28}

Knowledge surveys have been used in several quality improvement programs to establish education needs of the workforce. In a randomized controlled trial (RCT), Beekman et al. (2013)¹⁵ assessed the knowledge of health professionals using a validated pressure injury knowledge assessment tool to identify knowledge gaps. The results were used to inform the development of interactive education interventions and a range of other strategies to improve pressure injury prevention and treatment. Price et al, (2017)²⁸ conducted a pre-intervention knowledge survey of health professionals working in aged care. The results were used to develop the content for a multi-faceted education program. After delivery of the targeted education, there was a significant increase in health professional knowledge and measures of competency, and a significant and sustained reduction in pressure injuries (*Level 2*).

Antonio and Conrad (2013)¹⁶ noted that evaluating staff wound care knowledge using a skills and knowledge survey provided benchmark data for a quality improvement initiative set in a regional health service. The results informed the development of organization-specific resources on the wound products available in the organization, as well as training in best practice pressure injury prevention (*Level 2*). Baldelli et al. (2008) reported that a staff knowledge survey on identification of pressure injuries and strategies for their prevention was a first-line initiative

in a quality improvement program in US hospitals. Establishing that the staff had a low understanding that pressure injury incidence in the facilities was above national average provided a rationale for the improvement program that followed¹⁷ (Level 2). Although neither of these studies^{16,17} evaluated the impact of the education programs on staff knowledge, organizations in both studies experienced a decrease in pressure injury incidence.

The guideline chapter on *Health Professional Education* includes recommendations on training and education.

Attitudes and cohesion

There is a small volume of evidence that demonstrates a relationship between pressure injury incidence and attitudes of the workforce.^{19,20} In an observational study conducted in Finland (n= 66 facilities and 724 nurses), Pekkarinen et al. (2008)²⁰ assessed the relationships between pressure injury prevalence and nurse perspectives on management decisions and time pressure in the workplace. A statistically significant association ($p = 0.05$) between an increase in pressure injuries and the nurses' ranking of time pressure within their unit was established.²⁰ (Level 4). However, Bosch et al. (2011)³¹ found no relationship between pressure injury rates and organizational culture in nursing homes and hospitals (n = 61 facilities and n = 460 health professionals) in the Netherlands. Evaluated factors included team climate and competing values, point prevalence of pressure injuries and evidence-based quality indicators. After adjusting for confounders, the odds ratio of experiencing a pressure injury ranged from 0.99 to 1.02 for four different types of organizational cultures (group, developmental, rational and hierarchical)³¹ (Level 4).

Qualitative studies have noted a focus on collaborative care by health professionals. In a study conducted with medical-surgical nurses in India (n = 100),¹⁹ teamwork and collaboration were identified by the nurses as the most important facilitator for providing pressure injury prevention¹⁹ (Level 5). In long term aged care in Greece, nurses identified interdisciplinary conflict as a barrier to their practice, and proposed a collaborative approach as being more consistent with best practice²¹ (Level 5).

Other studies indicate that nurse attitudes towards pressure injuries are influenced by years of experience in nursing and knowledge levels,^{22,32,33} which may explain the conflicting results of the studies above.

20.4: At an organizational level, assess and maximize the availability and quality of equipment and standards for its use as part of a quality improvement plan to reduce the incidence of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Evidence supporting the recommendation comes from one high quality Level 1 study,¹⁵ one moderate³⁴ and two low³⁵⁻³⁷ quality Level 2 studies, and additional Level 4 studies.³⁸⁻⁴⁰ The studies were conducted in a range of clinical and geographic locations and all reported quality improvement programs that demonstrated reduction in pressure injury incidence and/or prevalence after commencement of the program. All the quality improvement programs incorporated an assessment of equipment and/or products in the facility as a component of the program, including reviewing, replacing and/or changing procurement arrangements for equipment and/or products. The resources required to conduct an equipment review were not clear, but in one moderate quality Level 4 study⁴⁰ that measured compliance, there was a very high level of delivery of the initiative by health professionals.

Implementation Considerations

- Review availability of and access to support surfaces, and establish protocols for procurement that ensure timely access for individuals at risk of, or with an existing pressure injury^{15,40-42} (Levels 1, 2 and 4).
- Review the equipment available within the facility to ensure appropriate options are available to meet the needs of special populations, including individuals with obesity, frail older adults, neonates and children (*Expert opinion*). More information is available in the guideline chapter *Populations with Specific Pressure Injury Related Needs*.
- Review and select medical devices available within the facility based on the devices' ability to induce the least degree of damage from the forces of pressure and/or shear^{43,44} while still achieving their intended function (Levels 2 and 4).
- Review the product formulary associated with skin and wound care^{16,34} (Level 2).

Evidence Discussion

Multiple studies^{15,34-40} indicated that including an evaluation of equipment (e.g., support surfaces, medical devices and wound care products) and its procurement as a component of a quality improvement initiative is associated

with reductions in pressure injuries. In a number of studies in which surveys and interviews were conducted,^{12,19,22,23,27} health professionals identified inappropriate or inadequate equipment as a barrier to implementing best practice. In contrast, having easy access to equipment was perceived as a facilitator to performing best practice (*Level 5*). An audit identified that there was a high level of compliance by health professionals with equipment reviews when introduced as a component of a quality improvement program⁴⁰ (*Level 4*).

Beeckman et al. (2013)¹⁵ included an assessment of the availability and quality of pressure injury preventive resources as part of a multi-faceted approach that was associated with a reduction in pressure injury prevalence in aged care facilities (7.1% versus 14.6%) (*Level 1*). Tippet (2009)³⁴ evaluated the support surfaces available within an aged care facility as part of a quality improvement program that reported sustained reduction in pressure injury prevalence over four years (*Level 2*). Sving et al. (2104)¹⁸ undertook a review of the facility equipment inventory as part of a quality improvement program; however this program saw no significant reduction in pressure injuries (8.4% versus 9%, $p > 0.05$)¹⁸ (*Level 2*). Lower level studies that initiated support surface reviews also reported reduction in pressure injuries^{39,40} (*Level 4*). Reviewing the equipment includes identifying the mattresses, cushions, beddings systems, chairs and manual handling equipment within the facility that is and is not contributing to optimal pressure injury prevention. Many of the studies on pressure injury prevention initiatives that led to a reduction in pressure injuries reported that the equipment available within the facility was changed (e.g., increasing, upgrading or replacing) as a result of a comprehensive review.³⁵⁻³⁸ National standards have been developed and approved in the US to test various support surface characteristics including immersion, envelopment, heat and water vapor dissipation characteristics, heat and moisture dissipation characteristics, and horizontal stiffness.^{45,46}

In some facilities, access to support surfaces may be limited outside of regular business hours (e.g., if the facility uses rental/contract equipment). Organizations should review access to support surfaces and develop written guidelines that identify the fastest way to procure support surfaces when a need is identified outside of business hours (e.g. on holidays, nights and weekends). Four studies^{15,40-42} (*Levels 1 and 4*) reporting on quality improvement programs included review of support surface procurement arrangements. In these studies, the contracts for acquisition of pressure injury prevention equipment (primarily support surfaces) was reviewed and optimized, sometimes in conjunction with the rental contractor.⁴² All these quality improvement programs reported a sustained reduction in pressure injuries.

Institutions should also review the medical devices in the facility and select devices that will minimize skin damage. This may include selection of softer, more flexible devices (e.g., tubes and airways)⁴³ or products of different design and/or sizing^{35,43,44} (*Levels 2 and 4*). Reviewing the product formulary also allows the organization to ensure evidence-based wound care treatment is available and sourced in a manner that maximizes ability of health professionals to implement best practice in chronic wound management^{16,34} (*Level 2*).

Quality Improvement Initiatives

20.5: At an organizational level, develop and implement a structured, tailored and multi-faceted quality improvement program to reduce the incidence of pressure injuries.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)

Evidence Summary

Evidence from two high^{15,47} and two moderate^{48,49} quality Level 1 studies indicated that a multi-faceted quality improvement program is associated with reductions in facility-acquired pressure injuries. This was supported by 17 Level 2 studies^{16-18,34-37,43,50-58} of high, moderate and low quality; five Level 3 studies^{3,59-62} of moderate and low quality and 11 Level 4 studies^{38-42,44,63-67} of high, moderate and low quality. The studies were conducted in a range of facilities including acute medical-surgical hospitals, critical/intensive care facilities, nursing homes, community care and pediatric hospitals. The studies were also delivered in a range of geographic locations including the US, Europe, the Middle East and the Pan-Pacific. The interventions in all studies included a range of initiatives that were tailored to the facility and often increased as the quality improvement program continued. Reported effectiveness varies and is likely contributed to by the baseline pressure injury incidence and factors discussed throughout this chapter. One high quality economic analysis⁶⁸ and four lower quality economic analyses^{41,57,61,69} indicated that the resources required to implement a quality improvement program are substantial, but lead to cost savings through prevention of pressure injuries. Qualitative studies indicated that health professionals^{23,70,71} and individuals and their informal caregivers⁷² find quality improvement programs to be acceptable.

Implementation Considerations

- Assess barriers and facilitators at professional and organizational levels before implementing a pressure injury prevention program within the organization (see *Recommendations 20.1 to 20.4*).

Evidence Discussion

Evidence provides support for the introduction of a multi-faceted quality improvement program (often called a 'bundle') that addresses the specific needs of the facility for improving pressure injury prevention and treatment. Most of the successful approaches reported in the literature incorporated strategies at both the professional and organizational level, suggesting that adopting a multi-faceted approach is an effective strategy that appears to increase the involvement of health professionals,¹⁵ minimize resistance to change,¹⁵ and lead to sustained reduction in facility-acquired pressure injuries.^{3,15-17,24,34-41,43,44,48-67,71}

Selection of specific interventions to include in a multi-faceted pressure injury prevention program should be based on a thorough evaluation of the barriers and facilitators specific to the organization,¹⁵ as discussed earlier in this chapter.

Different approaches are reported in the literature. Chaboyer et al. (2016)⁴⁷ evaluated a multi-faceted program in eight hospitals in Australia. The bundle included promoting patient engagement in pressure injury prevention, nurse education and promotional material. Although there was no statistically significant difference in pressure injury rates at the patient level compared to standard care (6.1% versus 10.5%, $p > 0.05$), there was a significant 52% reduction in hospital-acquired pressure injuries associated with the multi-faceted pressure injury bundle⁴⁷ (*Level 1*). Beeckman et al.'s (2013)¹⁵ bundle included a wide range of components at the professional and organizational level aimed at reducing pressure injuries in nursing homes in Belgium ($n = 11$ facilities with $n = 646$ residents). Over the course of the four-month study, Category/Stage I and greater pressure injury rates decreased from 14.6% to 7.1%. Although nursing knowledge about pressure injuries did not change, the comprehensive bundle demonstrated a positive impact on the attitude of health professionals toward pressure injury prevention¹⁵ (*Level 1*). Also set in nursing homes, Rantz et al. (2012)⁴⁹ found that introduction to US facilities of a comprehensive bundle that included education, clinical resources and mentoring was associated with a reduction in pressure injury incidence over a two year period (odds ratio [OR] 1.23, 95% confidence interval 1.00 to 1.51). High staff turn-over was noted to increase the cost of the intervention due to regular staff education⁴⁹ (*Level 1*). Multi-faceted bundles have also been implemented with success in critical care settings. Tayyib et al. (2015)⁴⁸ included evidence-based guidelines, education, risk assessment and routine repositioning in a bundle for Saudi Arabian critical care units. There was a 70% lower likelihood of pressure injuries compared to prior to the bundle's introduction (7.14% versus 32.86%, $p < 0.001$)⁴⁸ (*Level 1*).

In addition to these *Level 1* studies, a large volume of *Level 2*,^{16-18,34-37,43,50-58} *Level 3*,^{3,59-62} and *Level 4*^{38-42,44,63-67} studies conducted in critical care, acute care, aged care, community care and pediatric care provided evidence that a multi-faceted quality improvement program was associated with a significant reduction in pressure injury incidence. These studies were all conducted at an organizational level and reported reduction in pressure injury incidence or prevalence that was sustained over at least 12 months. Components that were included in these successful quality improvement programs are discussed in the recommendations that follow.

The literature on implementing quality improvement programs included one analysis providing high quality evidence. This study demonstrated that the intervention reported in a *Level 1* study by Chaboyer et al. (2016)⁴⁷ (see above) was associated with an estimated net monetary benefit of $-\$2,320$ (95% CI $-\$3,900$ to $-\$1,175$) per individual patient (AUD in 2016).⁶⁸ In another cost analysis, a program delivered in a hospital in Denmark that achieved a 9.3% reduction in pressure injuries had an estimated net savings per patient of €38.62 (Euros in 2013).⁶⁹ A program delivered in 12 US nursing homes achieved a 59% reduction in monthly pressure injury incidence and an approximate cost saving of \$20,800 per 100 residents (USD in 2014).⁶¹ Other studies in US and New Zealand also reported moderate and large cost savings across facilities and health networks.^{41,57} There is a wide variation in clinical and geographic settings in which these cost analyses are conducted; however, the evidence consistently reported financial savings over time.

20.6: At an organizational level, engage all key stakeholders in oversight and implementation of the quality improvement program to reduce the incidence of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

Key stakeholders include management, health professionals and untrained staff, patients and families/informal caregivers. The recommendation is underpinned by a high quality Level 1 study that included a partnership between management and interdisciplinary care staff and promotion of team decision-making into a successful quality

improvement program⁴⁹ as well as a low quality Level 2 study that incorporated a regional level steering committee with management and clinical staff.⁵¹ A low quality Level 4 study also showed benefits of a regional oversight committee that included management and care staff.⁶³ A moderate quality Level 2 study³⁴ and a moderate quality Level 3 study⁵⁹ both included interdisciplinary team engagement in a quality improvement initiative. Patient engagement in quality care delivery was a primary focus of a quality initiative reported in a high quality Level 1 study⁴⁷ and was also a component of programs reported in Level 2^{16,43} and Level 4 studies.⁴¹ In surveys providing indirect evidence,^{19,22} nursing staff identify barriers to implementing quality care when the patient individual is unable or unwilling to be involved in care, suggesting patient engagement is both important and acceptable to health professionals.

Implementation Considerations

- Engage in strategic partnerships at the organization level to develop, implement, promote and evaluate a quality improvement in pressure injury prevention and treatment^{49,51,63} (*Levels 1, 2 and 4*).
- Engage patients and their families/informal caregivers in pressure injury prevention initiatives, facilitated by provision of education and information^{16,41,43,47} (*Levels 1, 2 and 4*).
- Promote interdisciplinary team decision-making to develop and implement quality improvement in pressure injury prevention and treatment^{34,49,59} (*Levels 1, 2 and 3*).

Evidence Discussion

The evidence suggests that an effective quality improvement program should be underpinned by strong leadership and should actively engage with managers/administrators, health professionals, patient individuals and informal caregivers. Strategies used in various studies to achieve engagement at all levels of staffing in the facility, and with patient stakeholders, are summarized in *Table 24.1*.

At the managerial level, regional level steering committees were a component of two successful quality improvement programs, one of which reported a reduction in facility-acquired pressure injuries from 53% to 12% over 16 months⁶³ (*Level 3*), and the second of which reported a decrease in pressure injuries of 1.37 per 1,000 patient days.⁵¹ In both studies, management from a number of facilities within the same region worked together to develop, implement and promote a pressure injury prevention program (*Levels 2 and 4*).

Engaging staff through partnerships with management, and promotion of interdisciplinary involvement was also a component in a number of quality improvement programs.^{34,59} Tippet (2009)³⁴ reported a successful quality improvement program delivered by an interdisciplinary leadership team that included director of nursing and supervisors, as well as allied health professionals, physicians, wound nurses and the supply clerk (*Level 2*). Sving et al. (2014)¹⁸ reported engagement of first-line managers with nurses and care staff in regular evaluation of pressure injury prevalence and the facility-level prevention program¹⁸ (*Level 2*). Set in Australian surgical units, the initiative reported by Burston et al. (2015)⁵⁹ consisted of a manager, nurses and allied health professionals working together to lead the introduction of the quality improvement program (*Level 3*).

Engaging patient consumers in pressure injury prevention was a key component of numerous successful multi-faceted interventions.^{16,41,43,47} In the bundle reported by Chaboyer et al. (2016)⁴⁷ (*Level 1*, reported above), three simple evidence-based messages were conveyed to patients and their informal caregivers through face-to-face education, DVDs and posters to promote their engagement in pressure injury prevention. In addition to a facility-level reduction in pressure injuries,⁴⁷ high levels of patient engagement were achieved (>96% of patients received at least one component of education),⁷³ and patients reported that the personal contact from health professionals and a newfound understanding of pressure injuries were important in enhancing their engagement⁷² (*Levels 1 and 5*).

Table 24.1: Summary of evidence supporting stakeholder engagement as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Management engagement	
Regional level steering committee	US regional hospital network (<i>Level 2</i>) ⁵¹ US hospital and local inpatient facilities (<i>Level 4</i>) ⁶³
Staff engagement	
Partnership between management and interdisciplinary care staff	US nursing homes (<i>Level 1</i>) ⁴⁹ US regional hospital network (<i>Level 2</i>) ⁵¹ US nursing home (<i>Level 2</i>) ³⁴ Australian surgical units (<i>Level 3</i>) ⁵⁹ US hospital and local inpatient facilities (<i>Level 4</i>) ⁶³ Acute hospitals in Sweden (<i>Level 2</i>) ¹⁸
Promotion of team decision-making	US nursing homes (<i>Level 1</i>) ⁴⁹
Patient engagement	
Patient engagement in pressure injury prevention	Australian tertiary care hospitals (<i>Level 1</i>) ⁴⁷ Australian acute and aged care (<i>Level 2</i>) ¹⁶
Face-to-face patient education	Australian tertiary care hospitals (<i>Level 1</i>) ⁴⁷
Patient information leaflets	US pediatric hospital (<i>Level 2</i>) ⁴³ New Zealand hospitals (<i>Level 4</i>) ⁴¹

20.7: At an organizational level, include evidence-based policies, procedures and protocols and standardized documentation systems to reduce the incidence of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

This recommendation is underpinned by one moderate quality Level 1 study,⁴⁸ one moderate quality Level 2 study,⁵⁰ seven low quality Level 2 studies,^{16,17,35,51,53,54,74} one low quality Level 3 study³ and two low quality Level 4 studies.^{38,39} All these studies reported multi-faceted quality improvement programs that included policies, procedures and protocols that were underpinned by evidence-based guidelines. In one of these studies, nurse-generated care plans based on evidence were implemented,³⁵ and in another program evidence appraisals were undertaken.³⁸ In all the studies, the multi-faceted quality improvement program was associated with a reduction in pressure injuries.

Implementation Considerations

- Use evidence-based clinical guidelines to underpin local policies, procedures and protocols.^{17,48,50,54} (*Levels 1 and 2*).

Evidence Discussion

Underpinning a quality improvement program with an evidence-based information system (i.e., policies, procedures, protocols, information systems and documentation systems) reinforces best practice. Studies reporting programs based on evidence are summarized in *Table 24.2*. Four studies^{17,38,50} reported successful evidence-based pressure injury bundles that were introduced into critical care settings in Saudi Arabia,⁴⁸ USA,^{17,50} and the UK.³⁸ In the study providing the highest level of evidence supporting this recommendation,⁴⁸ the introduction of a pressure injury prevention bundle based on international clinical guidelines was associated with a 70% reduction in the likelihood of a pressure injury ($p < 0.001$). The intervention was also associated with significantly fewer Category/Stage I ($p = 0.002$) and Category II/Stage II ($p = 0.026$) pressure injuries compared to standard care⁴⁸ (*Level 1*). Other studies in critical care implementing evidence-based bundles together with other interventions reported large reductions in pressure injury incidence—a 50% reduction in one study¹⁷ (*Level 2*) and 63% reduction in a second study³⁸ (*Level 4*). A critical care evidence-based bundle also contributed to an increase in staff adherence to repositioning ($p = 0.015$) and heel elevation ($p < 0.001$)⁵⁰ (*Level 2*).

Table 24.2: Summary of evidence for using evidence-based information systems as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Evidence-based clinical guidelines	Saudi Arabian critical care units (<i>Level 1</i>) ⁴⁸ US acute and critical care hospital (<i>Level 2</i>) ¹⁷ US long term acute care hospital (<i>Level 2</i>) ⁵⁴ US intensive care units (<i>Level 2</i>) ⁵⁰ US acute care hospitals (<i>Level 2</i>) ⁷⁴
Evidence-based pressure injury protocols	US regional hospital network (<i>Level 2</i>) ⁵¹ Australian acute and aged care (<i>Level 2</i>) ¹⁶ US hospitals and medical centers (<i>Level 3</i>) ³ US pediatric hospital (<i>Level 4</i>) ⁴⁴ Australian acute and sub-acute units (<i>Level 4</i>) ³⁹
International pressure injury staging system	Lebanese medical center (<i>Level 2</i>) ⁵³ US pediatric hospital (<i>Level 4</i>) ⁴⁴
Evidence appraisals	UK intensive care units (<i>Level 4</i>) ³⁸
Changes to/standardization documentation system (often electronic)	US long term acute care hospital (<i>Level 2</i>) ⁵⁴ US acute care hospitals (<i>Level 2</i>) ³⁵ US long term face facilities (<i>Level 2</i>) ⁵² US pediatric hospital (<i>Level 4</i>) ⁴⁴ US hospital and local inpatient facilities (<i>Level 4</i>) ⁶³

Five studies demonstrated effectiveness of evidence-based pressure based injury bundles in acute care settings in the US,^{3,51,74} Lebanon⁵³ and Australia³⁹. In analysis of data from 55 tertiary hospitals, Padula et al. (2016)³ noted an association between the number of evidence-based quality improvement activities and a hospital-acquired pressure injury rates, although change to funding and coding policies over the same period of time also contributed to decreases in pressure injuries (*Level 3*). Another large study in 21 US hospitals noted that introduction of an evidence-based pressure injury bundle, overseen by a regional committee, led to an average decrease in pressure injury incidence over four years of 1.37 per 1,000 patient days⁵³ (*Level 2*). Introduction of the use of a pressure injury classification system from an evidence-based international guideline was associated with a significant reduction in pressure injuries (6.63% versus 2.47%, $p < 0.01$), when combined with electronic reporting and staff education⁵³ (*Level 2*). Use of international clinical guidelines to develop a standardized prevention bundle for individuals at extremely high risk of pressure injuries was associated with a 67% reduction in hospital-acquired pressure injuries over four years in two US acute care hospitals (pre-intervention 12/1,000 patient days versus post-intervention 0.4/1,000 patient days)⁷⁴ (*Level 2*). Introduction of annual reviews of the way in which the facility translated evidence into practice was associated with decreases in pressure injury incidence in an acute care setting in Australia³⁹ (*Level 4*).

In acute and residential care in Australia, Antonio and Conrad (2013)¹⁶ implemented evidence-based interventions, together with leadership initiative and extensive health professional education, leading to a reduction in combined pressure injury point prevalence from 11% to 3.7% over three years and a bed/day savings' improvement from \$882,740 to \$4,427,684 (Australian in 2013) over one year¹⁶ (*Level 2*). In an aged care facility in the US, introduction of protocols based on a pressure injury guideline led to a reduction in pressure injuries from 41% to 4.2% over 12 months (*Level 2*).

Standardized documentation

Standardized documentation systems have been included into the facility information system as a part of numerous multi-faceted quality improvement programs. In a multi-center study conducted in eleven long term aged care facilities, Horn et al. (2010)⁵² introduced a standardized, computer documentation system that incorporated weekly automated electronic reports on completeness of records and identified individuals at high risk of pressure injury (*Level 2*). Electronic medical records were commonly used in quality improvement programs to document pressure injury assessment,^{35,44,54,63} (*Levels 2 and 4*), including in conjunction with computerized internal reporting⁵⁴ (*Level 2*) and automated referrals to a wound ostomy and continence nurse (WOCN)³⁵ (*Level 2*). Standardized documentation is considered to improve the quality and accuracy of documentation and to promote communication across the interdisciplinary care team.⁷⁵

20.8: At an organizational level, provide clinical decision support tools as part of a quality improvement plan to reduce the incidence of pressure injuries.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

This recommendation is underpinned by one high¹⁵ and one moderate⁴⁸ quality Level 1 study, one moderate⁵² and three low quality^{35-37,53} Level 2 studies, two Level three studies^{60,61} and three Level 4 studies^{38,41,42}. The studies, which all reported reductions in pressure injuries associated with the introduction of a multi-faceted quality improvement program, reported the use of computer-generated reports,^{15,36,37,52} risk assessment decision support protocols,^{36,37,48,53} and support surface selection algorithms,³⁵ to promote clinical decision-making by individual health professionals and the multidisciplinary team.

Implementation Considerations

- Use clinical decision support tools that are aligned with evidence-based guidelines (*Expert opinion*).
- Consider implementing a protocol or algorithm to assist health professionals to select appropriate support surfaces for the individual^{35,41,42,60} (*Levels 2, 3 and 4*).

Evidence Discussion

Algorithms and decision support tools or protocols are used to assist health professionals in their selection of appropriate care strategies and equipment for preventing and treating pressure injuries. Clinical decision support tools should be aligned with up-to-date evidence and present a range of management options that assist in decision-making between the health professional, individual, informal caregiver and interdisciplinary team regarding appropriate pressure injury prevention and management. Tools might include flow charts, algorithms, reports or other aids, either in electronic or paper form.⁷⁶

Such resources have been reported as a component of several successful quality improvement programs (see *Table 24.3*). More advanced computerized decision support tools are becoming more accessible; however, they are currently only reported as a component of quality improvement in a small number of implementation studies. In one study, Beeckman et al. (2013)¹⁵ evaluated the effectiveness of an electronic system to support the decision-making of health professionals developing individual-specific pressure injury prevention programs. The electronic system was part of a bundle of initiatives introduced in six long term care facilities. There was a significant reduction in Category/Stage I to IV pressure injuries compared to the control facilities that received only a hard copy of a pressure injury prevention guideline (7.1% versus 14.6%, $p < 0.05$) (*Level 1*). The comprehensive quality improvement program reported by Bales et al. (2011)³⁶ included a computerized decision support tool for initial assessment of a pressure injury and development of an appropriate management plan. In this study, the decision support tool was used by a WOCN to direct clinical care, with a resulting relative reduction in hospital acquired pressure injuries of 12%^{36,37} (*Level 2*). In an aged care setting, a weekly generated report of individuals with incomplete electronic medical records, a high-risk status or abnormal skin observations was successfully used to assist health professionals to identify individuals requiring prevention plans and follow-up, leading to reduction in pressure injuries⁵² (*Level 2*). Additionally, *Level 3*⁶¹ and *Level 4*⁴⁰ studies have described successful quality improvement programs that incorporated computerized decision support tools.

Table 24.3: Summary of evidence for decision support tools as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Computerized clinical decision support tools	Belgian nursing homes (<i>Level 1</i>) ¹⁵ US community hospital (<i>Level 2</i>) ^{36,37} US long term care facilities (<i>Level 2</i>) ⁵² US nursing homes (<i>Level 3</i>) ⁶¹ Australian hospitals (<i>Level 4</i>) ⁴⁰
Risk assessment protocols	Saudi Arabian critical care units (<i>Level 1</i>) ⁴⁸ Lebanese medical center (<i>Level 2</i>) ⁵³ US community hospital (<i>Level 2</i>) ^{36,37} UK intensive care units (<i>Level 4</i>) ³⁸
Support surface use selection protocol	US acute care hospitals (<i>Level 2</i>) ³⁵ Dutch nursing homes (<i>Level 3</i>) ⁶⁰ UK Hospital Trust (<i>Level 4</i>) ⁴² New Zealand hospitals (<i>Level 4</i>) ⁴¹

20.9: Provide clinical leadership in pressure injury prevention and treatment as part of a quality improvement plan to reduce pressure injuries.

(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

A large volume of evidence supports the recommendation to provide clinical leadership as a part of a quality improvement program. A high quality¹⁵ and a moderate quality⁴⁸ Level 1 study both included appointment of a wound champion as a part of a successful component of a multi-faceted improvement program. A second moderate quality Level 1 study⁴⁹ included clinical leadership delivered by an onsite research nurse. Seven low quality Level 2 studies,^{16,17,35-37,53-55} three Level 3 studies^{59,60} and three Level 4 studies^{41,44,65} included clinical leadership from a wound champion, a clinical nurse educator, an aged care trained nurse, specialist allied health professionals or a wound care team. The studies were conducted in critical care, acute care, aged care, community care and pediatric care, providing evidence that including clinical leadership in a quality improvement program is associated with pressure injury incidence reduction in many clinical settings.

Implementation Considerations

- Engage a wound care champion/coach/clinical educator to provide clinical leadership^{15-17,35-37,41,48,53,59,60} (*Levels 1, 2, 3 and 4*).
- Consider incorporating referrals to a wound ostomy continence nurse (WOCN) into the organization's quality improvement initiative^{35,50,59} (*Level 2*).
- Consider developing a wound care team within the organization^{54,65} (*Level 2 and 4*).

Evidence Discussion

Clinical leadership initiatives identified as components of successful quality improvement programs included identification and delegation of health professionals (usually nurses) with specialized knowledge and skills in pressure injury prevention and treatment. These health professionals were variably referred to as wound champions, coaches, clinical educators and clinical specialists, and they took on a range of roles including education, auditing, care planning and wound care in the quality improvement programs reported in the literature (see *Table 24.4*). Clinical leaders were often specifically trained for their roles through certification programs³⁵⁻³⁷ or national-level competency programs.^{36,37,53}

Table 24.4: Summary of evidence for clinical leadership as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Wound champions/coaches	Belgian nursing homes (Level 1) ¹⁵ Saudi Arabian critical care units (Level 1) ⁴⁸ Lebanese medical center (Level 2) ⁵³ US acute care hospitals (Level 2) ³⁵ US community hospital (Level 2) ^{36,37} Dutch nursing homes (Level 3) ⁶⁰ New Zealand hospitals (Level 4) ⁴¹
Referral of patient to WOCN/allied health	US acute care hospitals (Level 2) ³⁵ US intensive care units (Level 2) ⁵⁰ Australian surgical units (Level 3) ⁵⁹
Clinical nurse educators	Australian acute and aged care (Level 2) ¹⁶ US hospital (Level 2) ¹⁷
Wound care team	US long term acute care hospital (Level 2) ⁵⁴ US aged care facility (Level 4) ⁶⁵
Specialist tracheostomy and respiratory staff members	US pediatric hospital (Level 4) ⁴⁴
Phone and email support from aged care trained nurse	US nursing homes in one State (Level 2) ⁵⁵
Onsite consultation with a research nurse	US nursing homes (Level 1) ⁴⁹
WOCN-generated care planning	US acute care hospitals (Level 2) ³⁵ US community hospital (Level 2) ^{36,37}

**20.10: At a professional level, provide education in pressure injury prevention and treatment as part of a quality improvement plan to reduce the incidence of pressure injuries.
(Strength of Evidence = A; Strength of Recommendation = ↑↑)**

Evidence Summary

The recommendation is supported by two high quality^{15,47} and one moderate quality⁴⁸ Level 1 studies, four moderate quality^{34,48,50,57} and five low quality^{16,17,43,53,54} Level 2 studies and an additional seven Level 4 studies,^{38-41,44,63,65} all of which included an education initiative in a quality improvement program that was successful in reducing pressure injury incidence. Education initiatives included didactic presentations, hands-on/bedside teaching, peer-to-peer teaching and web-based teaching.

Implementation Considerations

- Education programs delivered as a component of a multi-faceted quality improvement program have included didactic education, competency-based education, bedside/hands-on teaching, peer-to-peer teaching and e-learning^{15-17,34,36-41,43,44,47,48,50,53,54,57,63,65} (Levels 1, 2 and 4).
- Consider mandating pressure injury-related education and recording attendance on the health professional's employment record^{34,36,37,40} (Level 2).
- For more information on education programs, see the guideline chapter *Health Professional Education*.

Evidence Discussion

Tailored health professional education was included in most of the quality improvement programs. Education was provided using a range of delivery methods, including, but not limited to:

- Didactic/classroom based^{16-18,44,47,54}
- Bed-side^{16,17,48,50}
- Peer-peer⁵⁷
- Active participation^{15,53,54}
- Web-based^{15,39-41,43,44,65}
- Written.^{15,41,44,47,48,53,63}

Many programs incorporated a range of education delivery methods, increasing accessibility of new information for health professionals with different learning style preferences. Some studies noted that the education program was competency-based,^{16,65} and mandatory attendance and/or recording of attendance in staff records^{34,40,44} was used in some studies to encourage compliance. Recommendations on health professional education are discussed in detail in chapter *Health Professional Education*. Table 24.5 provides an overview of the different types of education initiatives reported in the evidence translation research.

Table 24.5: Summary of evidence for education as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Competency based staff education program	Australian acute and aged care (Level 2) ¹⁶ US aged care facility (Level 4) ⁶⁵
Education program (some mandatory and/or recorded on staff record)	Belgian nursing homes (Level 1) ¹⁵ Australian tertiary care hospitals (Level 1) ⁴⁷ Saudi Arabian critical care units (Level 1) ⁴⁸ Australian acute and aged care (Level 2) ¹⁶ Lebanese medical center (Level 2) ⁵³ US hospital (Level 2) ¹⁷ US long term acute care hospital (Level 2) ⁵⁴ US nursing home (Level 2) ³⁴ Acute hospitals in Sweden (Level 2) ¹⁸ New Zealand hospitals (Level 4) ⁴¹ UK intensive care units (Level 4) ³⁸ US pediatric hospital (Level 4) ⁴⁴ US hospital and local inpatient facilities (Level 4) ⁶³
Bedside/hands-on teaching	Saudi Arabian critical care units (Level 1) ⁴⁸ US intensive care units (Level 2) ⁵⁰ Australian acute and aged care (Level 2) ¹⁶ US hospital (Level 2) ¹⁷
Peer-peer teaching	US acute care hospitals (Level 2) ⁵⁷
Web-based learning	Belgian nursing homes (Level 1) ¹⁵ US pediatric hospital (Level 2) ⁴³ New Zealand hospitals (Level 4) ⁴¹ US aged care facility (Level 4) ⁶⁵ Australian acute and sub-acute units (Level 4) ³⁹ Australian in-patient services (Level 4) ⁴⁰ US pediatric hospital (Level 4) ⁴⁴

20.11: At an organizational level, regularly monitor, analyze and evaluate performance against quality indicators for pressure injury prevention and treatment.
(Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

This recommendation is supported by one high quality Level 1 study,¹⁵ one moderate quality⁵⁵ and six low quality^{16,17,36,37,51,53,56} Level 2 studies, a Level 3 study⁶² and two Level 4 studies.^{39,66,67} The studies reported multi-faceted quality improvement programs that were associated with reduction in pressure injury incidence and/or prevalence that included evaluation as one of the program components. Evaluation initiatives reported in the studies included auditing/surveillance, use of computer-based pressure injury monitoring systems, evaluation of facilitators and barriers to best practice, engagement of a data analysis team, and daily program evaluation.

Implementation Considerations

- Regularly monitor and analyze facility-acquired pressure injury rates^{15,16,18,39,51,53,56} (Level 1 and 2). Review the chapter *Measuring Pressure Injury Prevalence and Incidence* for related recommendations.

- Consider implementing an electronic system to report and track pressure injury incidence and prevalence^{15,53,56} (*Level 1 and 2*).
- Use appropriate quality indicators to monitor pressure injury prevention and treatment⁵⁵ (*Expert opinion*).
- Consider implementing benchmarking to promote continuous quality improvement^{66,67} (*Level 4*).
- Undertake a regular evaluation of the quality improvement initiative^{18,36,37,39} (*Levels 2 and 4*).
- Quality indicators to evaluate implementation of best practice outlined in this clinical guideline are included in the chapter *Quality Indicators for this Guideline*.

Evidence Discussion

Many studies reported monitoring facility-acquired pressure injury rates as a method for evaluating the success of quality improvement programs^{15-18,36,37,39,51,53,56} (see *Table 24.6*).

Computer-based monitoring was used in several quality improvement programs.^{15,53,56,62} In an RCT that demonstrated sustained reduction in Category/Stage I to IV pressure injuries (7.1% versus 14.6%, $p < 0.05$) a multi-faceted intervention that included a computerized monitoring system was implemented. The system allowed staff to enter the results of clinical audits of pressure injury rates and to undertake computer analysis and presentation of the facility's ongoing progress¹⁵ (*Level 1*). Sebastian-Viana et al. (2016)⁵⁶ used a computer reminder system that enabled documentation of pressure injuries together with pressure injury risk care initiatives, allowing staff to see automatic monitoring data whenever the computer was turned on. This initiative was included in a multi-faceted program that was associated with a relative risk reduction for pressure injuries of 29.4% and a number needed to treat of 333⁵⁶ (*Level 2*). Mallah et al. (2014)⁵³ introduced an electronic pressure injury reporting system to support a comprehensive quality improvement program that led to a relative reduction in pressure injury incidence of 4.16% ($p < 0.01$).⁵³ Other computer-based initiatives reported in the literature included a data linkage system that matched nurse and patient data in a computerized care planning program⁶² (*Level 3*) and a web-based quality reporting program that enabled benchmarking between nursing homes within the same regional network^{66,67} (*Level 4*).

A large range of quality indicators, many of which relate to local accreditation processes, are used to monitor pressure injury care. The chapter *Quality Indicators for this Guideline* details a set of quality indicators that can be used to audit organizational performance against the recommendations in this guideline.

Table 24.6: Summary of evidence for ongoing evaluation as an initiative to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Regular auditing/surveillance of pressure injuries	Australian acute and aged care (<i>Level 2</i>) ¹⁶ Australian acute and sub-acute units (<i>Level 4</i>) ³⁹ Belgian nursing homes (<i>Level 1</i>) ¹⁵ US community hospital (<i>Level 2</i>) ^{36,37} US hospitals (<i>Level 2</i>) ¹⁷ Acute hospitals in Sweden (<i>Level 2</i>) ¹⁸
Computer-based pressure injury monitoring system	Belgian nursing homes (<i>Level 1</i>) ¹⁵ Spanish hospital (<i>Level 2</i>) ⁵⁶ Lebanese medical center (<i>Level 2</i>) ⁵³
Nurse/patient data linkage in computerized care planning system	US hospitals (<i>Level 3</i>) ⁶²
Quality indicator tracking system	US nursing homes in one State (<i>Level 2</i>) ⁵⁵
Web-based quality reporting for benchmarking	US nursing homes (<i>Level 4</i>) ^{66,67}
Daily nurse-led quality rounds	US cardiac wards (<i>Level 2</i>) ⁵⁸
Evaluating facilitators and barriers to best practice	US regional hospital network (<i>Level 2</i>) ⁵¹ Acute hospitals in Sweden (<i>Level 2</i>) ¹⁸
Data analysis action team	US regional hospital network (<i>Level 2</i>) ⁵¹
Annual review of quality program	Australian acute and sub-acute units (<i>Level 4</i>) ³⁹
Daily safety update in bedside handover	Australian surgical units (<i>Level 3</i>) ⁵⁹

20.12: At an organizational level, use feedback and reminder systems to promote the quality improvement program and its outcomes to stakeholders.
(Strength of Evidence = B2; Strength of Recommendation = ↑)

Evidence Summary

One high quality Level 1 study,¹⁵ one moderate quality and four low quality Level 2 studies,^{16,17,43,74} and moderate and low quality Level 3⁵⁹ and Level 4^{36,37,39,41} studies provided evidence supporting this recommendation. The studies reported on multi-faceted quality improvement programs associated with reduction in pressure injury incidence and/or prevalence that included initiatives that promoted the program to staff and/or patients and informal caregivers. Feedback initiatives included brochures and posters, reporting of outcomes, rewards and/or staff recognition for participation. Reminder systems included visual cues to care staff to implement preventive care.

Implementation Considerations

- Use brochures/flyers and posters to provide health professionals, individuals and informal caregivers with information about the organization's quality improvement initiative^{36,37,41,42} (Levels 2 and 4).
- Regularly give feedback to stakeholders on the progress and achievements of the organization's quality improvement initiative^{16,17,39,43} (Level 2 and 4).
- Consider introducing recognition and rewards to encourage health professionals to actively engage in the organization's quality improvement initiative^{36,37,59} (Level 2 and 3).
- Consider using reminder systems (e.g. visual or auditory cues) to promote preventive care implementation and health professionals' engagement in the organization's quality improvement initiative^{15,74} (Level 1 and 2).

Evidence Discussion

Several successful facility-wide pressure injury prevention programs have included regular (e.g., weekly and/or monthly) reporting of the program initiatives and/or the pressure injury occurrence to stakeholders through newsletters, posters, flyers or computer-generated reports.^{15-17,36,37,39,41-43,59} Table 24.7 summarizes some of the promotional strategies that were successfully used in quality improvement programs delivered in different clinical contexts.

Reminder systems have also been used to encourage health professionals to implement preventive care. Individuals assessed as having an extremely high pressure injury risk were identified in the facility with a visual reminder at the bedside. Combined with a guideline-based preventive care bundle, this reminder initiative was associated with a 67% reduction in hospital-acquired pressure injuries over four years⁷⁴ (Level 2). Ongoing pressure injury prevention campaigns that are promoted to all stakeholders on a regular basis could assist in maintaining awareness and vigilance in implementing best practice.

Table 24.7: Summary of evidence for promoting the initiative as an impetus to reduce pressure injuries

Quality Improvement Initiative	Setting and Level of Evidence
Brochures outlining Care protocol features	New Zealand hospitals (Level 4) ⁴¹
Reporting of outcomes/improvements	Australian acute and aged care (Level 2) ¹⁶ US hospital (Level 2) ¹⁷ US pediatric hospital (Level 2) ⁴³ Australian acute and sub-acute units (Level 4) ³⁹
Rewards and recognition	US community hospital (Level 2) ^{36,37} Australian surgical units (Level 3) ⁵⁹
Posters	US community hospital (Level 2) ^{36,37} UK Hospital Trust (Level 4) ⁴²
Reminders	Belgian nursing homes (Level 1) ¹⁵ US acute care hospitals (Level 2) ⁷⁴

References

1. Lahmann NA, Halfens RJG, Dassen T. Impact of prevention structures and processes on pressure ulcer prevalence in nursing homes and acute-care hospitals. *Journal of Evaluation in Clinical Practice*, 2010; 16(1): 50-56.
2. Ma C, Park SH. Hospital Magnet Status, Unit Work Environment, and Pressure Ulcers. *Journal of Nursing Scholarship*, 2015; 47(6): 565-73.
3. Padula WV, Gibbons RD, Valuck RJ, Makic MBF, Mishra MK, Pronovost PJ, Meltzer DO. Are evidence-based practices associated with effective prevention of hospital-acquired pressure ulcers in US Academic Medical Centers? *Medical Care*, 2016; 54(5): 512-518.
4. Berlowitz DR, Young GJ, Hickey EC, Saliba D, Mittman BS, Czarnowski E, Simon B, Anderson JJ, Ash AS, Rubenstein LV, Moskowitz MA. Quality improvement implementation in the nursing home. *Health Serv Res*, 2003; 38(1 Pt 1): 65-83.
5. Patrician PA, McCarthy MS, Swiger P, Raju D, Breckenridge-Sproat S, Su X, Randall KH, Loan LA. Association of Temporal Variations in Staffing With Hospital-Acquired Pressure Injury in Military Hospitals. *Research in Nursing & Health*, 2017; 40(2): 111-119.
6. Konetzka RT, Stearns S, Park J. The staffing-outcomes relationship in nursing homes. *Health Services Research*, 2009; 43(3): 1025.
7. Twigg DE, Gelder L, Myers H. The impact of understaffed shifts on nurse-sensitive outcomes. *J Adv Nurs*, 2015; e-pub.
8. Lee HY, Blegen MA, Harrington C. The effects of RN staffing hours on nursing home quality: A two-stage model. *International Journal of Nursing Studies*, 2014; 51(3): 409-417.
9. Choi J, Staggs VS. Comparability of nurse staffing measures in examining the relationship between RN staffing and unit-acquired pressure ulcers: A unit-level descriptive, correlational study. *International Journal of Nursing Studies*, 2014.
10. Decker FH, Castle NG. Relationship of the Job Tenure of Nursing Home Top Management to the Prevalence of Pressure Ulcers, Pain, and Physical Restraint Use. *Journal of Applied Gerontology*, 2011; 30(5): 539-561.
11. Bae SH, Kelly M, Brewer CS, Spencer A. Analysis of nurse staffing and patient outcomes using comprehensive nurse staffing characteristics in acute care nursing units. *Journal of Nursing Care Quality*, 2014; 29(4): 318-326.
12. Ilesanmi RE, Olabisi P. Assessment of common interventions and perceived barriers to pressure ulcer prevention in Southwest Nigeria. *Journal of Wound, Ostomy and Continence Nursing*, 2014; 41(3): 242-246.
13. Cho E, Chin DL, Kim S, Hong O. The Relationships of Nurse Staffing Level and Work Environment With Patient Adverse Events. *Journal of Nursing Scholarship*, 2016; 48(1): 74-82.
14. Choi KR, Ragnoni JA, Bickmann JD, Saarinen HA, Gosselin AK. Health Behavior Theory for Pressure Ulcer Prevention: Root-Cause Analysis Project in Critical Care Nursing. *Journal of Nursing Care Quality*, 2016; 31(1): 68-74.
15. Beeckman D, Clays E, Van Hecke A, Vanderwee K, Schoonhoven L, Verhaeghe S. A multi-faceted tailored strategy to implement an electronic clinical decision support system for pressure ulcer prevention in nursing homes: A two-armed randomized controlled trial. *International Journal of Nursing Studies*, 2013; 50(4): 475-486.
16. Antonio T, Conrad K. Clinical and economic improvements in pressure injury care at Ballarat Health Services. *Wound Practice & Research*, 2013; 21(1): 4-10.
17. Baldelli P, Paciella M. Creation and implementation of a pressure ulcer prevention bundle improves patient outcomes. *American Journal of Medical Quality*, 2008; 23(2): 136-142.
18. Sving E, Högman M, Mamhidir AG, Gunningberg L. Getting evidence-based pressure ulcer prevention into practice: A multi-faceted unit-tailored intervention in a hospital setting. *International Wound Journal*, 2014.
19. Anand R, Kumari V, Nair R. Nurses' practice related to prevention of pressure ulcer among patients and factors inhibiting and promoting these practices. *International Journal of Nursing Education*, 2014; 6(1): 229-233.
20. Pekkarinen L, Sinervo T, Elovainio M, Noro A, Finne-Soveri H. Drug use and pressure ulcers in long-term care units: do nurse time pressure and unfair management increase the prevalence? *Journal of Clinical Nursing*, 2008; 17(22): 3067-3073.
21. Kaba E, Kelesi M, Stavropoulou A, Moustakas D, Fasoi G. How Greek nurses perceive and overcome the barriers in implementing treatment for pressure ulcers: 'against the odds'. *Journal of Wound Care*, 2017; 26(9): S20-S26.
22. Strand T, Lindgren M. Knowledge, attitudes and barriers towards prevention of pressure ulcers in intensive care units: a descriptive cross-sectional study. *Intensive & Critical Care Nursing: The Official Journal of the British Association of Critical Care Nurses*, 2010; 26(6): 335-342.
23. Tayyib N, Coyer F, Lewis P. Pressure injury prevention in a Saudi Arabian intensive care unit: Registered nurse attitudes toward prevention strategies and perceived facilitators and barriers to evidence implementation. *Journal of Wound, Ostomy, & Continence Nursing*, 2016; 43(4): 369-74.
24. Hart P, Davis N. Effects of nursing care and staff skill mix on patient outcomes within acute care nursing units. *Journal of Nursing Care Quality*, 2011; 26(2): 161-168.
25. Hickey EC, Young GJ, Parker VA, Czarnowski EJ, Saliba D, Berlowitz DR. The effects of changes in nursing home staffing on pressure ulcer rates. *J Am Med Dir Assoc*, 2005; 6(1): 50-3.
26. Needleman J, Buerhaus P, Mattke S, Stewart M, Zelevinsky K. Nurse-staffing levels and the quality of care in hospitals. *N Engl J Med*, 2002; 346(22): 1715-1722.
27. Mirshekari L, Tirgari B, Forouzi MA. Intensive care unit nurses' perceived barriers towards pressure ulcer prevention in south east Iran. *Journal of wound care*, 2017; 26(3): 145-151.
28. Price K, Kennedy KJ, Rando TL, Dyer AR, Boylan J. Education and process change to improve skin health in a residential aged care facility. *International Wound Journal*, 2017.
29. Beeckman D, Vanderwee K, Demarre L, Paquay L, Van Hecke A, Defloor T. Pressure ulcer prevention: development and psychometric validation of a knowledge assessment instrument. *International Journal of Nursing Studies*, 2010; 47(4): 399-410.

30. Florin J, Bååth C, Gunningberg L, Mårtensson G. Attitudes towards pressure ulcer prevention: A psychometric evaluation of the Swedish version of the APuP instrument. *International Wound Journal*, 2014.
31. Bosch M, Halfens RJG, van der Weijden T, Wensing M, Akkermans R, Grol R. Organizational culture, team climate, and quality management in an important patient safety issue: nosocomial pressure ulcers. *Worldviews On Evidence-Based Nursing / Sigma Theta Tau International, Honor Society Of Nursing*, 2011; 8(1): 4-14.
32. Tubaishat A, Aljezawi M. Exploring pressure ulcer care in Jordan: Nurses' knowledge and practice. *Journal of the Dermatology Nurses' Association*, 2014; 6(3): 115-123.
33. Ünver S, Fındık ÜY, Özkan ZK, Sürücü Ç. Attitudes of surgical nurses towards pressure ulcer prevention. *Journal of Tissue Viability*, 2017; 26(4): 277-281.
34. Tippet AW. Reducing the incidence of pressure ulcers in nursing home residents: a prospective 6-year evaluation. *Ostomy Wound Management*, 2009; 55(11): 52-58.
35. McInerney JA. Reducing hospital-acquired pressure ulcer prevalence through a focused prevention program. *Advances in Skin & Wound Care*, 2008; 21(2): 75-78.
36. Bales I, Duvendack T. Reaching for the moon: achieving zero pressure ulcer prevalence, an update. *Journal of Wound Care*, 2011; 20(8): 374-377.
37. Bales I, Padwojski A. Reaching for the moon: achieving zero pressure ulcer prevalence. *Journal of Wound Care*, 2009; 18(4): 137-144.
38. Richardson A, Peart J, Wright SE, McCullagh IJ. Reducing the incidence of pressure ulcers in critical care units: A 4-year quality improvement. *International Journal for Quality in Health Care*, 2017; 29(3): 433-439.
39. Smith SK, Ashby SE, Thomas L, Williams F. Evaluation of a multifactorial approach to reduce the prevalence of pressure injuries in regional Australian acute inpatient care settings. *International Wound Journal*, 2017; 07: 07.
40. Asimus M, Maclellan L, Li PI. Pressure ulcer prevention in Australia: the role of the nurse practitioner in changing practice and saving lives. *International Wound Journal*, 2011; 8(5): 508-513.
41. Lewis H, Hughes D, Madell D, Coomarasamy C, Villa L, Hayward B. Estimated reduction in expenditure on hospital-acquired pressure injuries after an intervention for early identification and treatment. *New Zealand Medical Journal*, 2017; 130(1461): 42-46.
42. Hall S, Ryan E. How a mattress selection matrix helped to sustain pressure ulcer prevention and also cut costs. *Wounds UK*, 2015; 11(3): 16-21.
43. Boesch RP, Myers C, Garrett T, Nie A, Thomas N, Chima A, McPhail GL, Ednick M, Rutter MJ, Dressman K. Prevention of tracheostomy-related pressure ulcers in children. *Pediatrics*, 2012; 129(3): e792-e797.
44. Peterson J, Adlard K, Walti BI, Hayakawa J, McClean E, Feidner SC. Clinical Nurse Specialist Collaboration to Recognize, Prevent, and Treat Pediatric Pressure Ulcers. *Clinical Nurse Specialist*, 2015; 29(5): 276-82.
45. National Pressure Ulcer Advisory Panel. *Support Surface Standards Initiative*. 2019 [cited October 2019; Available from: <https://npuap.org/page/S31?&hhsearchterms=%22standards%22>
46. Rehabilitation Engineering and Assistive Technology Society of North America (RESNA). *RESNA Standards Committee on Support Surfaces (SS)*. 2019 [cited October 2019; Available from: <https://www.resna.org/standards/support-surfaces/support-surfaces>.
47. Chaboyer W, Bucknall T, Webster J, McInnes E, Gillespie BM, Banks M, Whitty JA, Thalib L, Roberts S, Tallott M, Cullum N, Wallis M. The effect of a patient centred care bundle intervention on pressure ulcer incidence (INTACT): A cluster randomised trial. *International Journal of Nursing Studies*, 2016; 64: 63-71.
48. Tayyib N, Coyer F, Lewis PA. A two-arm cluster randomized control trial to determine the effectiveness of a pressure ulcer prevention bundle for critically ill patients. *Journal of Nursing Scholarship*, 2015; 47(3): 237-47.
49. Rantz MJ, Zwygart-Stauffacher M, Hicks L, Mehr D, Flesner M, Petroski GF, Madsen RW, Scott-Cawiezell J. Randomized multilevel intervention to improve outcomes of residents in nursing homes in need of improvement. *Journal of the American Medical Directors Association*, 2012; 13(1): 60-68.
50. Anderson M, Finch Guthrie P, Kraft W, Reicks P, Skay C, Beal AL. Universal Pressure Ulcer Prevention Bundle With WOC Nurse Support. *Journal of Wound, Ostomy & Continence Nursing*, 2015; 42(3): 217-225.
51. Crawford B, Corbett N, Zuniga A. Reducing hospital-acquired pressure ulcers: A quality improvement project across 21 hospitals. *Journal of Nursing Care Quality*, 2014; 29(4): 303-310.
52. Horn SD, Sharkey SS, Hudak S, Gassaway J, James R, Spector W. Pressure ulcer prevention in long-term-care facilities: a pilot study implementing standardized nurse aide documentation and feedback reports. *Advances in Skin & Wound Care*, 2010; 23(3): 120-131.
53. Mallah Z, Nassar N, Kurdahi Badr L. The Effectiveness of a Pressure Ulcer Intervention Program on the Prevalence of Hospital Acquired Pressure Ulcers: Controlled Before and After Study. *Applied Nursing Research*, 2014.
54. Milne CT, Trigilia D, Houle TL, DeLong S, Rosenblum D. Reducing pressure ulcer prevalence rates in the long-term acute care setting. *Ostomy Wound Management*, 2009; 55(4): 50-59.
55. Rantz MJ, Cheshire D, Flesner M, Petroski GF, Hicks L, Alexander G, Aud MA, Siem C, Nguyen K, Boland C, Thomas S. Helping nursing homes "at risk" for quality problems: a statewide evaluation. *Geriatric Nursing*, 2009; 30(4): 238-249.
56. Sebastian-Viana T, Losa-Iglesias M, Gonzalez-Ruiz JM, Lema-Lorenzo I, Nunez-Crespo FJ, Salvadores Fuentes P, team A. Reduction in the incidence of pressure ulcers upon implementation of a reminder system for health-care providers. *Applied Nursing Research*, 2016; 29: 107-12.
57. Beinlich N, Meehan A. Resource nurse program: A nurse-initiated, evidence-based program to eliminate hospital-acquired pressure ulcers. *Journal of Wound, Ostomy and Continence Nursing*, 2014; 41(2): 136-141.

58. Fisher K, Grosh A, Felty V. Using nurse-led rounds to improve quality measures related to HAPUs. *Nursing*, 2016; 46(11): 63-68.
59. Burston S, Chaboyer W, Gillespie B, Carroll R. The effect of a transforming care initiative on patient outcomes in acute surgical units: a time series study. *J Adv Nurs*, 2015; 71(2): 417-29.
60. Van Leen MWF, Schols JMGA, Hovius SER, Halfens RJG. The effect of a simple 3-step pressure relieving strategy for preventing pressure ulcers: An explorative longitudinal study from 2002-2011. *Wounds*, 2014; 26(10): 285-292.
61. Olsho LEW, Spector WD, Williams CS, Rhodes W, Fink RV, Limcangco R, Hurd D. Evaluation of AHRQ's on-time pressure ulcer prevention program: A facilitator-assisted clinical decision support intervention for nursing homes. *Medical Care*, 2014; 52(3): 258-266.
62. Stifter J, Yao Y, Lodhi MK, Lopez KD, Khokhar A, Wilkie DJ, Keenan GM. Nurse Continuity and Hospital-Acquired Pressure Ulcers: A Comparative Analysis Using an Electronic Health Record "Big Data" Set. *Nursing research*, 2015; 64(5): 361-371.
63. Thomas ME. The providers' coordination of care: a model for collaboration across the continuum of care. *Professional Case Management*, 2008; 13(4): 220-227.
64. Tzeng H-M, Grandy GA, Yin C-Y. Staff response time to call lights and unit-acquired pressure ulcer rates in adult in-patient acute care units. *Contemporary Nurse*, 2013; 45(2): 182-187.
65. Young DL, Borris-Hale C, Falconio-West M, Chakravarthy D. A Single Long-Term Acute Care Hospital Experience with a Pressure Ulcer Prevention Program. *Rehabil Nurs*, 2014.
66. Baier R, Butterfield K, Patry G, Harris Y, Gravenstein S. Identifying star performers: the relationship between ambitious targets and nursing home quality improvement. *Journal of the American Geriatrics Society*, 2009; 57(8): 1498-1503.
67. Baier RR, Butterfield K, Harris Y, Gravenstein S. Aiming for star performance: the relationship between setting targets and improved nursing home quality of care. *Journal of the American Medical Directors Association*, 2008; 9(8): 594-598.
68. Whitty JA, McInnes E, Bucknall T, Webster J, Gillespie BM, Banks M, Thalib L, Wallis M, Cumsille J, Roberts S, Chaboyer W. The cost-effectiveness of a patient centred pressure ulcer prevention care bundle: Findings from the INTACT cluster randomised trial. *Int J Nurs Stud*, 2017; 75: 35-42.
69. Mathiesen ASM, Norgaard K, Andersen MFB, Moller KM, Ehlers LH. Are labour-intensive efforts to prevent pressure ulcers cost-effective? *Journal of Medical Economics*, 2013; 16(10): 1238-1245.
70. Roberts S, McInnes E, Wallis M, Bucknall T, Banks M, Chaboyer W. Nurses' perceptions of a pressure ulcer prevention care bundle: a qualitative descriptive study. *BMC Nursing*, 2016; 15: 64.
71. Chaboyer W, Gillespie BM. Understanding nurses' views on a pressure ulcer prevention care bundle: A first step towards successful implementation. *Journal of Clinical Nursing*, 2014; 23: 3415-3423.
72. Roberts S, Wallis M, McInnes E, Bucknall T, Banks M, Ball L, Chaboyer W. Patients' Perceptions of a Pressure Ulcer Prevention Care Bundle in Hospital: A Qualitative Descriptive Study to Guide Evidence-Based Practice. *Worldviews Evid Based Nurs*, 2017.
73. Roberts S, McInnes E, Bucknall T, Wallis M, Banks M, Chaboyer W. Process evaluation of a cluster-randomised trial testing a pressure ulcer prevention care bundle: a mixed-methods study. *Implement Sci*, 2017; 12(1): 18.
74. Shieh DC, Berringer CM, Pantoja R, Resurreccion J, Rainbolt JM, Hokoki A. Dramatic Reduction in Hospital-Acquired Pressure Injuries Using a Pink Paper Reminder System. *Advances in Skin & Wound Care*, 2018; 31(3): 118-122.
75. Mykkänen M, Miettinen M, Saranto K. Standardized nursing documentation supports evidence-based nursing management. *Stud Health Technol Inform*, 2016; 225: 466-470.
76. Trevena L, McCaffery K, Salkeld G, Glasziou P, Del Mar C, Doust J, Hoffman T. Clinical decision-making tools: How effective are they in improving the quality of health care? 2014, Deeble Institute for Health Policy Research, Australian Government National Lead Clinicians Group, : <https://ahha.asn.au/publication/issue-briefs/clinical-decision-making-tools-how-effective-are-they-improving-quality>.

HEALTH PROFESSIONAL EDUCATION

Introduction

Knowledge is an essential component of delivering evidence-based practice. The process of evidence-based care requires health professionals to access and evaluate evidence, make clinical decisions based on their knowledge and experience, perform clinical skills with expertise and evaluate outcomes. Each of these stages requires specific knowledge. In a health world in which the volume of evidence continuously expands at an exponential rate, maintaining a contemporary knowledge base is critical. In many geographic and professional settings, health professionals are required to engage in continuous professional development in areas of knowledge consistent with their regular areas of clinical practice.

A large body of evidence evaluates knowledge levels regarding pressure injuries and their prevention and treatment. In general, there are mixed findings from descriptive studies that assessed existing knowledge levels of health professionals (primarily nurses) with respect to pressure injury prevention and treatment. Reported knowledge scores range from low (score below 50%)¹⁻³ to moderate (score 50 to 79%)⁴⁻²⁰ and high (score above 79%).²¹ Studies reported significant differences in knowledge based on geographic location,²² levels or type of health professional training,^{1,12,14,17} and wound certification.²¹ However, comparing study results is not possible because many different instruments have been used to conduct knowledge assessments. Furthermore, methodological limitations (e.g., selection bias, the use of non-validated knowledge assessment tools, and single site data collection) are common.

Attitudes are the principles and beliefs that influence an individual and the way they act. Attitude includes confidence, trust, value and self-direction.²³ Frameworks often identify attitudes as a key component in attaining competency in tasks both within healthcare and more broadly, thereby suggesting that there is a relationship between knowledge and attitude.²³⁻²⁶ Beeckman et al. (2010)²⁷ proposed that where a health professional holds positive beliefs about the potential of their actions to have an impact on pressure injury outcomes, it is more likely that they will engage in that positive behavior. A small volume of studies has explored this relationship with respect to pressure injury prevention and management. In one of these studies, Demarré et al. (2012)² explored the relationship between attitudes to pressure injuries and the knowledge of nurses and nursing assistants (n = 145) working in nursing homes in Belgium. Approximately 80% of the participants had more than five years' experience working in aged care. In this sample, there was no significant correlation between knowledge of pressure injuries and attitudes towards them, and knowledge level was not a significant predictor of compliance to best practice. However, both compliance with best practice and knowledge levels were both very low in this study, with 26.9% of individuals receiving no preventive pressure injury care, and the mean knowledge score of health professionals being 28.9%.² Another study⁸ identified that direct care health professionals in an acute rehabilitation facility in Saudi Arabia (n = 105) with average knowledge displayed unsatisfactory attitudes towards pressure injuries; however, there were no significant relationships between attitudes and either education level or years of clinical experience.⁸ In contrast, one study⁵ found there was a significant positive correlation (p < 0.01) between knowledge and attitudes. However, this study⁵ was conducted in nursing students with minimal experience in pressure injury prevention and care.

Other studies have noted a significant relationship between pressure injury knowledge and behavior/skill performance, suggesting that improving knowledge might have a direct impact on the level of preventive pressure injury care in which a health professional engages.^{6,7,28} These studies provide some support for provision of education for health professionals on pressure injury prevention and treatment. However, the findings indicating that improvements in knowledge lead to increased implementation of pressure injury prevention and treatment are not supported by other studies.²⁹ Only a small number of studies have explored the effectiveness of education intervention in achieving sustained improvement in knowledge or competency, and an even smaller number of studies have explored the influence of education on reducing the incidence of pressure injuries.

The available evidence on assessing and delivering health education is discussed throughout this chapter. Studies in which health professional education was one component of a multi-faceted quality improvement program are discussed in the guideline chapter on *Implementing Best Practice in Clinical Settings*. Due to the multi-faceted nature of quality improvement programs, the specific influence of education cannot be determined. The evidence in this current chapter focuses specifically on delivery of health professional education programs with a goal of achieving a sustained improvement in relevant outcome measures.

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What valid and reliable assessment methods are available to evaluate health professional knowledge of pressure injury prevention and treatment?
- What interventions/programs are effective in attaining sustained improvements in health professional knowledge of pressure injury prevention and treatment?
- What interventions/programs are effective in attaining sustained improvements in health professional competency in pressure injury prevention and treatment?

Assessing Health Professional Knowledge and Attitudes

21.1: At the organizational level, assess the knowledge health professionals have about pressure injuries to facilitate implementation of an education program and a quality improvement program. (Strength of Evidence = B1; Strength of Recommendation = ↑↑)

Evidence Summary

The recommendation to assess staff knowledge to facilitate education and quality improvement programs is supported by three studies providing high quality Level 1 evidence³⁰ and low quality Level 2 evidence.^{31,32} In all three studies,³⁰⁻³² knowledge survey results were used to develop organization-specific education interventions as a component of multi-faceted quality improvement programs that achieved reductions in pressure injury incidence. Additionally, one low quality Level 2 study³³ that demonstrated significant reduction in pressure injury incidence implemented a multi-faceted health professional education program that was based on the results of a knowledge assessment.

Implementation Considerations

- Use tools that have been shown to have good psychometric properties to assess pressure injury knowledge. *Table 25.1* summarizes tools designed to assess knowledge related to pressure injury prevention and treatment.
- The Attitude Towards Pressure Ulcer Prevention Instrument (APuP) has good validity in measuring workforce attitudes towards pressure injury prevention^{34,35} (*Indirect evidence*).
- Use the results of organizational level knowledge surveys to develop targeted education initiatives to meet the knowledge needs of the workforce³³ (*Level 2*).

Evidence Discussion

Assessment of health professionals' knowledge related to pressure injury prevention and management identifies potential barriers to mitigate or facilitators to enhance when introducing either an education program or a quality improvement program. Understanding the knowledge needs of health professionals provides information that assists in the development of organization-specific education and training initiatives.^{30,31,33}

Knowledge surveys have been used in several quality improvement programs to establish education needs of the workforce. In a randomized controlled trial (RCT), Beeckman et al. (2013)³⁰ assessed the knowledge of health professionals using a validated pressure injury knowledge assessment tool to identify knowledge gaps. The results were used to inform the development of interactive education interventions and a range of other strategies to improve pressure injury prevention and management practice. Price et al. (2017)³³ conducted a pre-intervention knowledge survey of health professionals working in aged care. The results were used to develop the content for a multi-faceted education program. After delivery of the targeted education, there was a significant increase in health professional knowledge and measures of competency, and a significant and sustained reduction in pressure injuries.

The guideline chapter *Implementing Best Practice in Clinical Settings* includes this recommendation and presents discussion of health professional knowledge assessment in the context of quality improvement programs.

Table 25.1: Selection of assessment tools appropriate for assessing knowledge or attitudes

Assessment Tool	Tool characteristics			Psychometric testing
	tool design	content validity	test-retest reliability	
Pressure Ulcer Knowledge Assessment Tool (PUKAT) ³⁴	26 multiple-choice items in six themes	$\alpha = 0.77$	ICC = 0.88	Nurses and nursing students in Belgium and the Netherlands ³⁴
Pressure Ulcer Knowledge Assessment Tool 2.0 (PUKAT 2.0) ³⁶	25 multiple-choice items in six themes	-----	ICC = 0.69	Students and nurses in Belgium ³⁶
Pieper Pressure Ulcer Knowledge Test (PPUKT) ^{16,37}	47 items in three subscales	$\alpha = 0.91$ ³⁷	ICC = 0.96 ¹⁶	Critical care nurses in US ³⁷ Modified Turkish version in acute care nurses ¹⁶
Pieper Zulkowski Pressure Ulcer Knowledge Assessment Tool (PZ-PUKT) ³⁸	72 items in three subscales	$\alpha = 0.80$	-----	Conference attendees in US ³⁸
Questionnaire Adherence to Recommendations for Preventing Pressure Ulcers (QARPPU) ³⁹	18 items in four factors	$\alpha = 0.89$	-----	Nurses in Spain ³⁹
Attitude towards Pressure Ulcer Prevention tool (APuP) ²⁷	13 items in five factors	$\alpha = 0.79$	ICC = 0.88	Nurses and nursing students in Belgium and the Netherlands ²⁷

Delivery of Health Professional Education

21.2: At an organizational level, develop and implement a multi-faceted education program for pressure injury prevention and treatment.

(Strength of Evidence = B2; Strength of Recommendation = ↑↑)

Evidence Summary

Three low quality Level 2 studies^{33,40,41} demonstrated that a multi-faceted education program delivered to health professionals in a range of clinical and geographic settings was associated with a reduction in pressure injury incidence for 3 months,⁴¹ 12 months³³ and 24 months.⁴⁰ Two of the low quality Level 2 studies^{33,40} demonstrated sustained improvement in health professional knowledge about pressure injuries for 12 months or longer, and the fourth low quality study reported improvement in knowledge after three months.⁴¹ A Level 5 study also demonstrated that a multi-faceted pressure injury education program improves knowledge level in the short term.⁴² Additionally, all three low quality Level 2 studies^{33,40,41} reported improvements in a measure of health professional competency was associated with the education programs, including increased time spent performing pressure injury prevention skills^{33,41} and increased performance of risk assessment.^{33,40} Indirect evidence also showed that the more recently an education session has been attended by the health professional, the more positive their attitudes are toward pressure injury prevention and treatment.⁴³ Patient individuals and their informal caregivers have identified the knowledge levels of their professional caregivers as being of high priority.⁴⁴

Implementation Considerations

- Incorporate a range of teaching styles, training opportunities and resources into a multi-faceted education program. *Table 25.3* outlines components of multifaceted programs that were associated with sustained increase in health professional knowledge and competency and a decrease in pressure injury incidence^{33,40-42} (*Levels 2 and 5*).
- Consider including a computer-based component of education into a multi-faceted education program (*Expert opinion*).

- Tailor the content of pressure injury prevention and treatment education to the needs of both the health professional team and the organization. Education program content that was reported as successful in achieving sustained (at least 12 months) improvement in knowledge and/or competency is discussed in this chapter (*Expert opinion*).
- The chapter on *Implementing Best Practice in Clinical Settings* includes extensive discussion regarding the effectiveness of multi-faceted quality improvement programs that included an educational component in reducing pressure injuries.

Evidence Discussion

A multi-faceted education program is one in which education is delivered using more than one type of education delivery method. Four studies^{33,40-42} presented evidence regarding the effectiveness of multi-faceted education programs, reporting outcome measures that included health professional knowledge, measures of health professional competency and, in some studies, pressure injury incidence or prevalence (*Levels 2 and 5*). *Table 25.2* provides an overview of the studies, including their clinical settings, duration of follow-up and impact of the multi-faceted education program on different outcome measures.

Table 25.2: Outcomes for multi-faceted pressure injury prevention and treatment education programs

	Setting	Follow up duration	Impact on health professional knowledge	Impact on health professional competency	Impact on pressure injury prevalence
Multi-faceted education program (<i>Level 2</i>) ³³	Aged care in Australia	1 year	Positive	Positive	Positive
Multi-faceted education program (<i>Level 2</i>) ⁴⁰	Surgery and emergency rooms in China	2 years	Positive	Positive	Positive
Multi-faceted education program (<i>Level 2</i>) ⁴¹	Aged care in Hong Kong	3 months	Short term [‡] positive	Short term [‡] positive	Short term [‡] positive
Multifaceted education program (<i>Level 5</i>) ⁴²	Acute hospitals in Nigeria	3 months	Short term [‡] positive	---	---

[‡] Short term describes outcomes measured at 3 months or earlier

In a study conducted in operating rooms and emergency departments in China,⁴⁰ a multi-faceted education program was developed with a goal of decreasing pressure injury incidence. A baseline training questionnaire was completed primarily by nurses (n = 275). Initiatives were introduced by an education steering committee, including a training course for individuals who would lead education delivery, training in standardized practices, access to a wound expert and a knowledge contest to reward successful individuals. This multi-faceted program was associated with an increase in average score pressure injury knowledge scores (47% baseline versus 81% at 24 months, p<0.001) and significantly improved use of the Braden Scale for risk assessment. Improvements in knowledge and competency translated to a significant decrease in pressure injury prevalence at 24 months (from 0.07% to 0.03%)⁴⁰ (*Level 2*).

Price et al. (2017)³³ undertook a baseline knowledge survey in two aged care facilities in Australia. The results were used to develop appropriate and targeted education programs for registered nurses and personal care workers. All clinical staff received ongoing access to a wound expert, online training modules, training in prevalence surveys, didactic education sessions and written education material appropriate to their employment level. Patient consumer engagement in the education was achieved through bedside teaching and workshops, and the patients also received their own written education material and presentations and consumer meetings. To evaluate the program, clinical staff maintained a diary of their activities and undertook a knowledge test 12 months after the program commencement. After 12 months, pressure injury prevalence decreased from 12.5% to 6.8% (p = 0.01). Knowledge levels were significantly increased for both nursing staff (p < 0.01), but unchanged for personal care workers (p = 0.30). The diaries indicated that nursing staff spent significantly more time on wound prevention and care (p < 0.001) and pressure injury risk assessment (p = 0.01). The number of individuals with a pressure injury prevention plan was unchanged; however, this was high at baseline, with 93% of prevention plans being documented. Personal care workers spent more time on wound prevention and care (p < 0.001) and repositioning (p < 0.001)³³ (*Level 2*).

Two additional studies^{41,42} provided evidence for multi-faceted education programs in decreasing pressure injuries,^{41,42} improving health professional knowledge⁴¹ and improving skills.⁴¹ However, these two studies^{41,42} were only of three months' duration; therefore, the sustainability of the education program and the positive outcomes was only demonstrated in the short term (*Levels 2 and 5*).

A range of different education initiatives were used in the multi-faceted education programs (see *Table 25.3*). These included different education delivery styles as well as different initiatives to support the education. Because all studies used a different range of education initiatives it was not possible to identify specific components of the multi-faceted education programs that may have had greater impact or otherwise. A recent review⁴⁵ of effective teaching strategies in nursing skills noted that active and innovative strategies are received in a significantly more positive light by nurse learners than didactic/traditional lecture-style teaching. Overall, providing a range of different strategies^{45,46} that considers the type of skills/knowledge to be transferred, the health professional's learning style, and practicalities (e.g., teaching space and time, number of participants and accessibility of teaching resources) is important in the design of a multi-faceted education program.

Didactic/Traditional Lecture Education Programs

Five studies reported pressure injury prevention and treatment education programs that implemented didactic/lecture/classroom style education only (see *Table 25.4*). Only one of these studies⁴⁷ provided evidence that delivering an didactic-style education program is associated with a statistically significant decrease in pressure injuries over time. However, this study was only three months in duration; therefore, could not demonstrate sustainability of the education program or its results, and the study was also unable to demonstrate impact of the education on knowledge levels⁴⁷ (*Level 2*). Two studies^{48,49} showed that didactic-style education was associated with short term (6 months⁴⁹ and 2 months⁴⁸) increases in health professional knowledge (*Levels 2 and 5*), and one of the studies also demonstrated increase in competency demonstrated by improvements in documentation of wound size, exudate and tissue type (but no improvement in documentation of interventions).⁴⁸ However, neither of these studies evaluated pressure injury incidence. A further two studies^{50,51} showed increases in health professional knowledge immediately after education, but the positive improvements in knowledge levels were not sustained for the full length of the short (3 months⁵¹ and 5 months⁵⁰) studies (both *Level 5*).

Table 25.3: Components used in multi-faceted pressure injury prevention and treatment education programs

Type of Education Initiative	Setting and Level of Evidence
Didactic presentations	Aged care in Australia (<i>Level 2</i>) ³³ Acute hospitals in Nigeria (<i>Level 5</i>) ⁴² Aged care in Hong Kong (<i>Level 2</i>) ⁴¹
Computer-based/online education modules	Aged care in Australia (<i>Level 2</i>) ³³
Written education material	Aged care in Australia (<i>Level 2</i>) ³³ Acute hospitals in Nigeria (<i>Level 5</i>) ⁴²
Hands-on/bedside teaching	Aged care in Australia (<i>Level 2</i>) ³³ Emergency and surgery setting in China (<i>Level 2</i>) ⁴⁰ Aged care in Hong Kong (<i>Level 2</i>) ⁴¹
"Train the Trainer" for individuals delivering education	Emergency and surgery setting in China (<i>Level 2</i>) ⁴⁰
Group work/discussion	Acute hospitals in Nigeria (<i>Level 5</i>) ⁴²
Access to education and support from a wound care expert/champion	Aged care in Australia (<i>Level 2</i>) ³³ Emergency and surgery setting in China (<i>Level 2</i>) ⁴⁰
Patient consumer facilitation of education	Aged care in Australia (<i>Level 2</i>) ³³
Incentives (e.g. CPD points, awards, rewards)	Aged care in Australia (<i>Level 2</i>) ³³ Emergency and surgery setting in China (<i>Level 2</i>) ⁴⁰

Table 25.4: Outcomes for didactic/lecture-style pressure injury prevention and treatment education programs

	Setting	Follow up duration	Impact on health professional knowledge	Impact on health professional competency	Impact on pressure injury prevalence
Didactic/lecture-style program [#] (Level 2) ⁴⁹	Community hospital in US	6 months	Short term [¥] positive	---	---
Didactic/lecture-style program* (Level 2) ⁴⁷	Aged care in US	3 months	No change	Short term [¥] positive	Short term [¥] positive
Didactic/lecture-style program [#] (Level 5) ⁵¹	Acute hospitals in US	3 months	Short term [¥] positive Not sustained	---	---
Didactic/lecture-style program* (Level 5) ⁵⁰	Critical care in New Zealand	5 months	Short term positive Not sustained	---	---
Didactic/lecture-style program* (Level 5) ⁴⁸	Long term care in US	2 months	Short term [¥] positive	Short term [¥] positive	---

[#] Compared to computer-based education

[¥] Short term describes outcomes measured at 3 months or earlier

* No comparator, change over time

The evidence was insufficient to make any recommendations on education programs that deliver didactic-style education only. None of these studies were of sufficient length to evaluate a sustained effect of education, and all the studies had significant methodological limitations, including self-selection of participants into the studies, small sample sizes, use of non-validated evaluation methods and lack of control for the effect of independent learning. Further, it is unclear to what extent the education delivery method influences the effectiveness of the education program. Therefore, the lack of a specific recommendation should not be interpreted as an indication that didactic-style education is ineffective. Didactic-style education presentation was included in the majority of the multi-faceted education programs^{33,41,42} reported earlier in this chapter), suggesting that this style of education may have a positive impact, especially when reinforced by other education techniques. Additionally, in one study that compared computer-based learning to didactic lecture presentation, health professionals undertaking the didactic-style education reported higher levels of satisfaction (97.6% vs 93.3%, $p = 0.042$)⁴⁹ (Level 2).

Computer-based Education Programs

Four studies^{49,51-54} reported the implementation of a computer-based education program for improving health professional pressure injury knowledge. Two of these studies^{49,51} reported a direct comparison between computer-based education and didactic/classroom style education delivery. The four studies are summarized in *Table 25.5*. All of the studies^{49,51-54} were of short duration (less than six months), therefore no sustained effect of the intervention was demonstrated, and none evaluated the effect of the intervention on either health professional competency or pressure injury prevalence. All of the studies^{49,51-54} reported short term improvements in health professional knowledge. One comparison study found that there was no significant difference between computer-based and classroom style education for achieving improvement of knowledge in acute care nurses in US ($n = 43$).⁴⁹ The second comparison study, conducted in nurses (primarily in critical care) in the US ($n = 60$), reported that computer-based learning was less effective than traditional classroom based education for improving knowledge.⁵¹ However, both these studies were small and of short duration.^{49,51}

Due to the lack of studies demonstrating that computer-based education programs alone are effective in achieving significant and sustained improvement in health professional knowledge, no recommendation can be made.

Table 25.5: Outcomes for computer-based pressure injury prevention and treatment education programs

	Setting	Follow up duration	Impact on health professional knowledge	Impact on health professional competency	Impact on pressure injury prevalence
Computer-based program [#] (Level 2) ⁴⁹	Community hospital in US	6 months	Short term [¥] positive	---	---
Computer-based program* (Level 5) ⁵²	Acute and aged care in Norway	3 months	Short term [¥] positive Not sustained	---	---
Computer-based program* (Level 5) ^{53,54}	University setting in Spain	Immediate	Short term [¥] positive	---	---
Computer-based program [#] (Level 5) ⁵¹	Acute hospitals in US	3 months	Short term [¥] positive	---	---

[#] Compared to didactic/classroom style education

[¥] Short term describes outcomes measured at 3 months or earlier

* No comparator, change over time

Pressure Injury Education Program Content

There are no studies that directly evaluate the topics and content of education programs. It is acknowledged that education programs should be evidence-based and reflect best practice, therefore their content should be drawn from evidence-based guidelines.

Educational programs reported in the literature vary in terms of their content, and many studies did not report the education program in detail.

A suggested list of topics includes, but is not limited to:^{46,55}

- Etiology and risk factors for pressure injuries
- Differential diagnosis and classification of pressure injuries
- Risk assessment
- Skin assessment and skin care
- Nutrition
- Repositioning, including manual handling and use of equipment
- Selection and use of support surfaces
- Pressure injury assessment
- Wound care (including wound dressing selection)
- Documentation
- Importance of an interprofessional approach
- Working with the individual and their informal caregivers
- Prevalence and incidence measurement
- Implementing best practice and quality improvement.

In addition to general pressure injury related education, health professionals should undertake appropriate education and training before undertaking advanced clinical skills (e.g., debridement). In many geographic and clinical regions, requirements for additional and or advanced education are mandated. Local guidance on certification, credentialing and/or training requirements should be reviewed and implemented. Health professionals should also undertake appropriate education and training before using woundcare products and specialized equipment (e.g., diagnostic tools and support surfaces).

References

1. Aydin A, Karadağ A. Assessment of nurses' knowledge and practice in prevention and management of deep tissue injury and stage I pressure ulcer. *Journal of Wound, Ostomy, and Continence Nursing*, 2010; 37(5): 487-494.
2. Demarré L, Vanderwee K, Defloor T, Verhaeghe S, Schoonhoven L, Beeckman D. Pressure ulcers: knowledge and attitude of nurses and nursing assistants in Belgian nursing homes. *J Clin Nurs*, 2012; 21(9/10): 1425-1434.

3. Qaddumi J, Khawaldeh A. Pressure ulcer prevention knowledge among Jordanian nurses: A cross-sectional study. *BMC Nurs*, 2014; 13(1).
4. Cox J, Roche S, Gandhi N. Critical care physicians: Attitudes, beliefs, and knowledge about pressure ulcers. *Advances in Skin and Wound Care*, 2013; 26(4): 168-176.
5. Simonetti V, Comparcini D, Flacco ME, Di Giovanni P, Cicolini G. Nursing students' knowledge and attitude on pressure ulcer prevention evidence-based guidelines: A multicenter cross-sectional study. *Nurse Educ Today*, 2015.
6. Tallier PC, Reineke PR, Asadoorian K, Choonoo JG, Campo M, Malmgreen-Wallen C. Perioperative registered nurses knowledge, attitudes, behaviors, and barriers regarding pressure ulcer prevention in perioperative patients. *Appl Nurs Res*, 2017; 36: 106-110.
7. Lee YJ, Kim JY, Korean Association of Wound Ostomy Continence N. Effects of pressure ulcer classification system education programme on knowledge and visual differential diagnostic ability of pressure ulcer classification and incontinence-associated dermatitis for clinical nurses in Korea. *Int Wound J*, 2016; 13(1): 26-32.
8. Kaddourah B, Abu-Shaheen AK, Al-Tannir M. Knowledge and attitudes of health professionals towards pressure ulcers at a rehabilitation hospital: A cross-sectional study. *BMC Nurs*, 2016; 15(1).
9. Chianca T, Rezende J, Borges E, Nogueira V, Caliri M. Pressure ulcer knowledge among nurses in a Brazilian university hospital. *Ostomy Wound Management*, 2010; 56(10): 58-64.
10. Iranmanesh S, Abdoli Tafti A, Rafiei H, Dehghan M, Razban F. Orthopaedic nurses' knowledge about pressure ulcers in Iran: A cross-sectional study. *J Wound Care*, 2013; 22(3): 138-143.
11. El Enein NYA, Zaghoul AA. Nurses' knowledge of prevention and management of pressure ulcer at a health insurance hospital in Alexandria. *Int J Nurs Pract*, 2011; 17(3): 262-268.
12. Gupta N, Loong B, Leong G. Comparing and contrasting knowledge of pressure ulcer assessment, prevention and management in people with spinal cord injury among nursing staff working in two metropolitan spinal units and rehabilitation medicine training specialists in a three-way comparison. *Spinal Cord*, 2012; 50(2): 159-164.
13. Miyazaki M, Caliri M, Santos C. Knowledge on Pressure Ulcer Prevention Among Nursing Professionals. *Rev. Latino-Am. Enfermagem*, 2010; 18(6): 1203-1211.
14. Galvao NS, Serique MA, Santos VL, Nogueira PC. Knowledge of the nursing team on pressure ulcer prevention. *Rev Bras Enferm*, 2017; 70(2): 294-300.
15. Gill EC, Moore Z. An exploration of fourth-year undergraduate nurses' knowledge of and attitude towards pressure ulcer prevention. *J Wound Care*, 2013; 22(11): 618-627.
16. Gul A, Andsoy, II, Ozkaya B, Zeydan A. A descriptive, cross-sectional survey of Turkish nurses' knowledge of pressure ulcer risk, prevention, and staging. *Ostomy Wound Manage*, 2017; 63(6): 40-46.
17. Gunningberg L, Mårtensson G, Mamhidir AG, Florin J, Muntlin Athlin A, Bååth C. Pressure ulcer knowledge of registered nurses, assistant nurses and student nurses: A descriptive, comparative multicentre study in Sweden. *Int Wound J*, 2013.
18. Miller DM, Neelon L, Kish-Smith K, Whitney L, Burant CJ. Pressure Injury Knowledge in Critical Care Nurses. *J Wound Ostomy Continence Nurs*, 2017.
19. Rafiei H, Abdar ME, Iranmanesh S, Lalegani H, Safdari A, Dehkordi AH. Knowledge about pressure ulcer prevention, classification and management: A survey of registered nurses working with trauma patients in the emergency department. *International Journal of Orthopaedic and Trauma Nursing*, 2014; 18(3): 135-142.
20. Rafiei H, Mehralian H, Abdar ME, Madadkar T. Pressure ulcers: how much do nursing students really know? *Br J Nurs*, 2015; 24(6): S12, S14-7.
21. Zulkowski K, Ayello EA, Wexler S. Certification and education: do they affect pressure ulcer knowledge in nursing? *J Nurs Adm*, 2010; 40(10 Suppl): S28-S32.
22. Meesterberends E, Wilborn D, Lohrmann C, Schols JMGA, Halfens RJG. Knowledge and use of pressure ulcer preventive measures in nursing homes: A comparison of Dutch and German nursing staff. *J Clin Nurs*, 2014; 23(13-14): 1948-1958.
23. Mallidou AA, Pat Atherton P, Chan L, Frisch N, Glegg S, Scarrow G. Core knowledge translation competencies: a scoping review. *BMC Health Serv Res*, 2018; 18: article 502.
24. Organization for Economic Cooperation and Development (OECD). *The OECD Learning Compass 2030*. 2019 [cited August 2019]; Available from: <https://www.oecd.org/education/2030-project/>.
25. Barry MM, Battel-Kirk B, Dempsey C. The CompHP core competencies framework for health promotion in Europe. *Health Educ Behav*, 2012; 39(6): 648-662.
26. Cutcliffe JR, Sloan G. Towards a Consensus of a competency framework for clinical supervision in nursing: Knowledge, attitudes, and skills. *The Clinical Supervisor*, 2014; 33(2): 182-203.
27. Beekman D, Defloor T, Demarre L, Van Hecke A, Vanderwee K. Pressure ulcers: development and psychometric evaluation of the Attitude towards Pressure Ulcer Prevention instrument (APuP). *Int J Nurs Stud*, 2010; 47(11): 1432-1441.
28. Saleh MYN, Al-Hussami M, Anthony D. Pressure ulcer prevention and treatment knowledge of Jordanian nurses. *Journal of Tissue Viability*, 2013; 22(1): 1-11.
29. Gallant C, Morin D, St-Germain D, Dallaire D. Prevention and treatment of pressure ulcers in a university hospital centre: A correlational study examining nurses' knowledge and best practice. *Int J Nurs Pract*, 2010; 16(2): 183-187.
30. Beekman D, Clays E, Van Hecke A, Vanderwee K, Schoonhoven L, Verhaeghe S. A multi-faceted tailored strategy to implement an electronic clinical decision support system for pressure ulcer prevention in nursing homes: A two-armed randomized controlled trial. *Int J Nurs Stud*, 2013; 50(4): 475-486.
31. Antonio T, Conrad K. Clinical and economic improvements in pressure injury care at Ballarat Health Services. *Wound Practice & Research*, 2013; 21(1): 4-10.

32. Baldelli P, Paciella M. Creation and implementation of a pressure ulcer prevention bundle improves patient outcomes. *Am J Med Qual*, 2008; 23(2): 136-142.
33. Price K, Kennedy KJ, Rando TL, Dyer AR, Boylan J. Education and process change to improve skin health in a residential aged care facility. *Int Wound J*, 2017.
34. Beeckman D, Vanderwee K, Demarre L, Paquay L, Van Hecke A, Defloor T. Pressure ulcer prevention: development and psychometric validation of a knowledge assessment instrument. *Int J Nurs Stud*, 2010; 47(4): 399-410.
35. Florin J, Bååth C, Gunningberg L, Mårtensson G. Attitudes towards pressure ulcer prevention: A psychometric evaluation of the Swedish version of the APuP instrument. *Int Wound J*, 2014.
36. Manderlier B, Van Damme N, Vanderwee K, Verhaeghe S, Van Hecke A, Beeckman D. Development and psychometric validation of PUKAT 2.0, a knowledge assessment tool for pressure ulcer prevention. *Int Wound J*, 2017.
37. Pieper B, Mattern JC. Critical care nurses' knowledge of pressure ulcer prevention, staging and description. *Ostomy Wound Management*, 1997; 43(2): 22-31.
38. Pieper B, Zulkowski K. The Pieper-Zulkowski pressure ulcer knowledge test. *Adv Skin Wound Care*, 2014; 27(9): 413-9.
39. Moya-Suárez AB, Morales-Asencio JM, Aranda-Gallardo M, Enriquez de Luna-Rodríguez M, Canca-Sánchez JC. Development and psychometric validation of a questionnaire to evaluate nurses' adherence to recommendations for preventing pressure ulcers (QARPPU). *Journal of Tissue Viability*, 2017; 26(4): 260-270.
40. Feng H, Li G, Xu C, Ju C. Educational campaign to increase knowledge of pressure ulcers. *Br J Nurs*, 2016; 25(12): S30-S35.
41. Kwong E, Lau A, Lee R, Kwan R. A pressure ulcer prevention programme specially designed for nursing homes: does it work? *J Clin Nurs*, 2011; 20(19/20): 2777-2786.
42. Ekama Ilesanmi R, Morohunfoluwa Oluwatosin O. A quasi-experimental study to Assess an interactive educational intervention on nurses' knowledge of pressure ulcer prevention in Nigeria. *Ostomy Wound Management*, 2016; 62(4): 30-40.
43. Aslan A, Yavuz van Giersbergen M. Nurses' attitudes towards pressure ulcer prevention in Turkey. *Journal of Tissue Viability*, 2016; 25(1): 66-73.
44. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
45. Staykova MP, Stewart DV, Staykov DI. Back to the basics and beyond: Comparing traditional and innovative strategies for teaching in nursing skills laboratories. *Teaching and Learning in Nursing*, 2017; 12(2): 152-157.
46. Ayello EA, Zulkowski K, Capezuti E, Jicman WH, Sibbald RG. Educating nurses in the United States about pressure injuries. *Adv Skin Wound Care*, 2017; 30(2): 83-94.
47. Wogamon CL. Exploring the effect of educating certified nursing assistants on pressure ulcer knowledge and incidence in a nursing home setting. *Ostomy Wound Management*, 2016; 62(9): 42-50.
48. Thomas A. Assessment of nursing knowledge and wound documentation following a pressure ulcer educational program in a long-term care facility: A capstone project. *Wound Practice and Research*, 2012; 20(3): 142-155.
49. Esche CA, Warren JI, Woods AB, Jesada EC, Iliuta R. Traditional classroom education versus computer-based learning: how nurses learn about pressure ulcers. *J Nurses Prof Dev*, 2015; 31(1): 21-7.
50. Tweed C, Tweed M. Intensive care nurses' knowledge of pressure ulcers: Development of an assessment tool and effect of an educational program. *Am J Crit Care*, 2008; 17(4): 338.
51. Cox J, Roche S, Van Wynen E. The effects of various instructional methods on retention of knowledge about pressure ulcers among critical care and medical-surgical nurses. *J Contin Educ Nurs*, 2011; 42(2): 71-78.
52. Bredesen IM, Bjoro K, Gunningberg L, Hofoss D. Effect of e-learning program on risk assessment and pressure ulcer classification: A randomized study. *Nurse Educ Today*, 2016; 40: 191-7.
53. Monteiro AKC, Monteiro AKC, Andrade EMLR, Luz MHBA, Cavalcanti PAL. Distance continuing education on prevention of pressure ulcer. *Revista Enfermagem*, 2016; 24(1).
54. Veredas FJ, Ruiz-Bandera E, Villa-Estrada F, Rufino-González JF, Morente L. A web-based e-learning application for wound diagnosis and treatment. *Comput Methods Programs Biomed*, 2014; 116(3): 236-248.
55. Agency for Healthcare Research and Quality. *Pressure Injury Prevention in Hospitals Training Program*. 2017 [cited October 2019]; Available from: <https://www.ahrq.gov/patient-safety/settings/hospital/resource/pressure-injury/index.html>.

QUALITY OF LIFE, SELF-CARE AND EDUCATION

Introduction

Health-related quality of life (HRQoL) refers to an individual's overall wellbeing and satisfaction in life. Health-related QoL is associated with mental, physical and spiritual wellbeing, self-perceived health, engagement in health behaviors, engagement in society and work, financial wellbeing, and environmental factors specific to the individual.¹ As noted in the guideline chapter *Supporting Wound Healing*, complex factors associated with the individual's environment, psychological status, education and social supports all influence pressure injury prevention and treatment.²

Quality of Life

Pressure injuries are negatively associated with all domains of HRQoL³⁻⁸. Having a pressure injury has been associated with significant declines in autonomy,³ security and spiritual wellbeing³ (*Level 4*). Compared to individuals without a pressure injury, those with a pressure injury have poorer physical functioning,^{4,5} poorer psychological functioning,^{4,5,7} greater levels of depression,⁴ poorer emotional status^{4,7} and lower functioning in social roles⁴ (*Level 3*). Impaired HRQoL has been shown to persist after a pressure injury has healed³ (*Level 4*). Because these studies look at associations, cause cannot be inferred. For example, it is not known whether the decline in functioning occurs before, after or at the same time as the pressure injury.

Qualitative studies⁹⁻¹¹ have explored the experience of having a pressure injury in more detail. In interviews conducted with 12 primarily community-based individuals with pressure injuries, Jackson et al. (2017)¹⁰ identified loss as having a significant influence on HRQoL. Major themes identified in this study included loss of mobility and independence, loss of privacy and dignity, loss of personal control and autonomy and loss of social engagement and preferred activities. These areas are all noted as being factors that embody the concept of HRQoL,¹ and identification of their loss suggests individuals with pressure injuries experience substantial decline in wellbeing¹⁰ (*Level 5*).

Gorecki et al. (2012)⁹ explored factors that influenced HRQoL for individuals (n = 30) living with pressure injuries either in hospital or in the community. Interviews identified that both the way care was experienced, and individual patient factors influenced HRQoL. Individual factors that were noted as contributing to HRQoL included comorbidities, coping ability, motivation, health-seeking behavior, knowledge, support, preoccupation with the pressure injury and beliefs about its cause, financial status and involvement of a partner in care. These factors interacted with the individuals' care experiences. Their own adherence to treatment, the experience of hospitalization, inconsistencies in care, time spent on wound care and satisfaction with care all influenced HRQoL.⁹ The researchers concluded that these factors not only directly contribute to pressure injury HRQoL, but also interact with each other, resulting in a complex relationship between HRQoL and contributory factors (*Level 5*).

A third qualitative study conducted by Latimer et al. (2014)¹¹ reported interviews from hospitalized individuals (n = 20), one-third of whom had a pressure injury. One of the major themes focused on the individual's emotions related to having a pressure injury. Pain, malodor, unpleasant memories, and emotional responses such as fear, anger and abandonment were noted to be a part of the pressure injury experience. The study also identified factors that influence engagement in self-care and resourcing pressure injury prevention and treatment as important to the experience of having a pressure injury. Latimer et al. (2014)¹¹ confirmed the conclusion of Gorecki et al. (2012)⁹ that the patient experience of pressure injuries is complex and HRQoL is influenced by a wide range of experiences and the individual's response to these.

Noting the complexity of HRQoL,^{9,11} it would be prudent to promote maximal function in all HRQoL domains (i.e., physical, psychological, social and spiritual). However, there was insufficient research on strategies focused on improving HRQoL explicitly and no specific recommendations have been made. However, the recommendations below on patient consumer education and promotion of self-care and lifestyle skills are relevant to HRQoL, and some of the studies^{12,13} reported the impact of interventions on a measure of HRQoL.

Knowledge Levels

The patient consumer has an important role in pressure ulcer prevention. Knowledge of pressure injuries and their prevention is important and requires a special emphasis for those at high risk. However, community dwelling individuals, as well as those with traumatic injury, generally lack information about pressure injuries.¹⁴⁻¹⁸ Thietje et al. (2011)¹⁶ explored pressure injury related knowledge development of individuals with a new spinal cord injury (SCI) over the course of an admission of greater than three months' duration. Baseline general knowledge about pressure injuries was poor. However, at discharge 47.2% of the individuals achieved a score on a knowledge test that

represented having good knowledge (30.4% average and 22.4% poor). The mean increase in knowledge score was significant over time (mean score 5.44 versus 11.24, ($p < 0.001$); however, there was a slight fall in knowledge levels after 30 months (mean score 10.8). The individuals in this study most often identified their rehabilitation physician as being the most important source of information. Other important sources of information were physiotherapists, nurses, other health professionals, in-hospital education courses and the internet. Support groups and families were not considered to be important knowledge sources¹⁶ (*Level 5*). This study highlights the significance that health professionals play in transferring knowledge to patient consumers outside of formal education programs, but also notes that the majority of individuals with SCI and at high risk of pressure injuries were discharged with less than optimal self-care knowledge.

Self-Care Skills

In one qualitative study, individuals with SCI ($n = 16$) identified the need for empowerment, education and approaches to coordination of social support as priorities for ongoing management of their condition. The significant themes included promotion of self-care through facilitating access to appropriate equipment and services within the community; providing education and support for managing pressure injury risk; and ensuring that the individual and their caregivers have a realistic perception of the risk of pressure injuries¹⁸ (*Level 5*). These findings were supported by a second qualitative study in which the need to advocate for one's self and to balance prevention and lifestyle concerns were highlighted by participants ($n = 19$) as important for ongoing care. In this study, providing information about wound care clinics, community-based resources and consumer support groups, facilitating access to medical help, and providing education were all considered valuable by individuals¹⁹ (*Level 5*). Both of these studies were conducted in small samples of community-based individuals with SCI. Although individuals with SCI are at high risk of pressure injuries and their experiences may be generalizable to other people at risk of pressure injuries, other burdens of disease are intricately linked to the ability of individuals with SCI to engage in self-care in ways that vary from individuals without severely debilitating chronic disease.

Ghaisas et al. (2015)²⁰ explored the relationship between lifestyle change and pressure injury healing in another cohort of individuals with SCI and pressure injuries ($n = 25$). The individuals were classified as having achieved either positive, minor or no change in three lifestyle domains: adjusting daily routine, changing the physical environment and developing an awareness of pressure injury risk in everyday living. The pressure injury status of the individuals was classified as either improving or worsening. Most of the cohort ($n = 19$) had a positive classification for both their pressure injury status and making lifestyle changes. Individuals with worsening pressure injury status were all classified as having made few or no lifestyle changes. This study²⁰ highlights the significance of being engaged in self-care and adapting one's way of living to accommodate pressure injury care. The authors described individuals who achieved both lifestyle change and improvement in the status of their pressure injuries as being motivated. They were also noted to have identified personal goals and support to achieve these goals. Positive lifestyle changes had helped to improve pressure injury healing. However, specific interventions to promote this engagement were not reported or evaluated in this study²⁰ (*Level 3*).

Clinical Questions

The clinical questions that guided the development of this chapter were:

- What are effective strategies for promoting HRQoL for individuals with or at risk of pressure injuries?
- What are effective strategies for engaging individuals in pressure injury prevention and treatment?

Measuring Quality of Life, Self-Care Skills and Knowledge

22.1: Assess the health-related quality of life, knowledge and self-care skills of individuals with or at risk of pressure injuries to facilitate the development of a pressure injury care plan and education program.
(Good Practice Statement)

Implementation Considerations

- Consider using a pressure-injury specific assessment tool to measure health-related quality of life (HRQoL), knowledge and self-care skills when available.²¹⁻²⁵ Some generic assessment tools designed for use in individuals in health settings have also been tested for reliability and validity for use in pressure injury-related care²⁶⁻²⁸ (*Level 4*).

- Consider all aspects of HRQoL (e.g., physical, psychological, social and spiritual) and their interactions when conducting an assessment (*Expert opinion*).
- Use the findings of HRQoL, knowledge and self-care skills to develop an individualized care plan that meets the individual's needs (*Expert opinion*).
- Assess the knowledge and care skills of informal caregivers to assist in care planning (*Expert opinion*).

Discussion

Measuring HRQoL, knowledge and self-care skills provides insight into the individual's needs and is intrinsic to delivering holistic care. Tracking these outcomes over time provides an indication of the effectiveness and acceptability of treatment.¹ Only a few tools^{21,25,29} have been developed for assessing HRQoL, knowledge and/or care skills specifically in individuals with or at risk of pressure injuries. Some generic HRQoL tools have also been evaluated and found to be reliable and valid when used in populations with or at risk of pressure injuries (see *Table 26.1*). The available evidence does not evaluate the impact of these assessments on pressure injury prevention or healing.

Gorecki et al. (2013)²⁹ developed a QoL assessment tool, the PU-QoL, specific to individuals with pressure injuries. The sample participating in the tool development and initial psychometric testing were based in hospitals, community centers and a hospice. The PU-QoL measures patient-reported outcomes in domains that were identified as significant to individuals with pressure injuries in previous studies.⁹ The tool includes scales that measure symptoms, physical functioning, psychological well-being and social participation. The research team developed and evaluated a revised version of their tool in 2018,²⁵ including nine pressure injury specific outcomes measured on three symptom scales and six function scales. Internal consistency for the sub-scales was good to excellent ($\alpha = 0.795$ to 0.97)²⁵ (both *Level 4*).

Kisala et al. (2015)²⁴ and Gélis et al. (2011)³⁰ both reported on the development and psychometric testing of tools for evaluation of individuals with SCI. The Spinal Cord Injury Quality of Life Pressure Ulcer Scale (SCI-QOL) reported by Kisala et al. (2015),²⁴ which focuses on HRQoL for individuals with SCI and an existing pressure injury, includes 12 items relating to the impact of pressure injury symptoms and the influence of pressure injuries on socialization, activity and work. The tool has good test-retest reliability (intraclass coefficient (ICC) = 0.79, 95% confidence interval [CI] 0.74 to 0.84) (*Level 4*). The Skin Management Needs Assessment Checklist reported by Gélis et al. (2011)³⁰ includes 12 questions measuring self-care skills related to preventing pressure injuries and other wounds. The tool has excellent reliability (ICC = 0.899, 95% CI 0.862 to 0.927) and is an easy-to-use, self-administered questionnaire³⁰ (*Level 4*).

Chaboyer et al. (2017)²¹ developed the Patient Participation in Pressure Injury Prevention (PPPIP) scale, which measures self-perceived knowledge, access to information, involvement in decision making and care, satisfaction with care and involvement of informal caregivers. The tool was developed and tested in individuals in acute hospital care. Internal consistency was excellent ($\alpha = 0.86$)²¹ (*Level 4*).

Table 26.1: Knowledge and HRQoL assessment tools tested in individuals with or at high risk of pressure injuries

Tool	Tool Type	Tool Topic	Tool content	Pressure Injury Population Tested In
Pressure Ulcer Quality of Life scale (PU-QOL and PUQOL-P)	Pressure injury specific	QOL	Ten items (later revised to nine items) measuring symptoms, physical functioning, psychological well-being and social participation specific to pressure injuries ²⁹	Adults in in community settings (Level 4) ²⁹ Adults in a secondary care hospital setting (Level 4) ²⁵
Spinal Cord Injury Quality of Life Pressure Ulcer Scale (SCI-QOL)	Pressure injury specific	QOL	Twelve items targeted at individuals with spinal cord injury (SCI) measuring psychological state, comfort, social and work activities ²⁴	Adults with SCI in community settings (Level 4) ²⁴
EQ-5D ^{®31}	Generic	Health-related QOL	Five domains measuring mobility, selfcare, usual activities, pain/discomfort and anxiety/depression ²⁸	Adults with pressure injuries in acute care and community settings (Level 4) ²⁸
Medical Outcomes Study (MOS) 36-Item Short Form Health Survey ³² (SF-36)	Generic	Physical and mental health	36 items measuring vitality, physical functioning, pain, health perceptions, emotional and physical roles in functioning, social role, mental health ³²	Adults with spinal cord injury and pressure injuries in the community (Level 4) ²⁷
SF-6D ³³	Generic	Physical and mental health	A shorter version of SF-36 Six domains measuring physical functioning, social functioning, role limitations, pain, mental health and vitality ²⁸	Adults with pressure injuries in acute care and community settings (Level 4) ²⁸
Patient Participation in Pressure Injury Prevention (PPPIP)	Pressure injury specific	Self-care skills	Seven items measuring knowledge, engagement with staff, receiving information, family assistance, acceptability of prevention plan	Hospitalized adults (Level 4) ²¹
Patient Activation Measure ³⁴ (PAM-score)	Generic	Self-care skills	Thirteen items measuring knowledge, skills and confidence for self-management	Individuals with paraplegia in rehabilitation setting (Level 4) ²⁶
Skin Management Needs Assessment Checklist (SMnac)	Pressure injury specific	Knowledge Self-care skills	12 items measuring knowledge on preventing wounds, conducting skin checks and preventive behavior ³⁵	Individuals with SCI with and without pressure injuries living in community (Level 4) ³⁰

Engaging Individuals in Pressure Injury Prevention and Treatment

22.2: Provide pressure injury education, skills training and psychosocial support to individuals with or at risk of pressure injuries.
(Strength of Evidence = C; Strength of Recommendation = ↑)

Evidence Summary

Two high quality,^{12,36} one moderate quality^{37,38} and one low quality^{39,40} Level 1 studies reported the impact of patient consumer education and lifestyle programs on preventing pressure injuries. One of the studies³⁶ reported fewer pressure injuries developed compared to a group receiving written education, but the incidence rate was very low in both groups and follow-up was only eight weeks. A second study^{39,40} found lower pressure injury recurrence rates at 24 months in a group of individuals who received an enhanced education program compared to groups receiving less or no education. Two studies^{12,37,41} reported no significant reduction in pressure injuries associated with education interventions compared to usual care, at either six months^{37,41} follow-up or at 24 months.¹² The findings from two moderate quality Level 1 studies¹³ and one low quality Level 3 study⁴² reporting the relationship between patient consumer education programs and healing of pressure injuries were also mixed. A high quality Level 1 study,³⁶ a low quality Level 1 study^{39,40} and a Level 5 study¹⁵ indicated that education programs have positive impacts on patient consumer knowledge levels in the short and long term.^{15,36} Three high quality^{12,13,36} and one low quality⁴³ Level 1 studies and indirect evidence⁴⁴ reported improvements in self-care skills following participation in education and lifestyle programs for up to 24 months. However, a study with six months' follow-up showed no effect on self-care skills for individualized compared to standardized telephone support.⁴¹ Quality of life outcomes were reported less frequently, but findings were also mixed. One high quality Level 1 study¹³ reported HRQoL improvements associated with education¹³ and a second high quality Level 1 study¹² finding improvements over time, but these were not different to usual care. The mixed results reported in these studies could relate to the varied program delivery methods, content of the programs, duration and intensity of education and follow-up periods, outcome measurement methods or characteristics of the participants.

Implementation Considerations

- Discuss prevention and treatment of pressure injuries with the patient consumer and their informal caregivers as a part of routine care (*Expert opinion*).
- Promote and facilitate self-management, particularly for individuals with ongoing risk (e.g., individuals with SCI) (*Expert opinion*).
- Use and recommend evidence-based education resources. Discuss the use of internet-based education with patient consumers and their informal caregivers and encourage access to reputable sources of information (*Expert opinion*).
- Consider education level, cognitive and psychological status, clinical condition and physical abilities when determining the most appropriate education and support interventions (*Expert opinion*).
- Access reputable guidance and research^{18,45-47} on strategies for developing health education material (*Expert opinion*).
- Where possible, use multiple education and support delivery methods (e.g., verbal, internet, telephone and written)^{12,36,39,40} (*Level 1*).
- Engage informal caregivers in education, skills training and psychosocial support interventions^{39,40} (*Level 1*).
- Regularly refresh and reinforce education and skills acquisition (*Expert opinion*).

Evidence Discussion

Although some studies^{44,48} supporting this recommendation were conducted in general at-risk populations in tertiary care, the majority of evidence for education, skills training and psychosocial support is drawn from studies conducted with individuals with SCI either with or at risk of pressure injuries. These participants have been recruited in the community, rehabilitation and acute post-surgical settings. Individuals with SCI have a high need for education and development of care and lifestyle skills due to their ongoing risk of pressure injuries. A number of outcome measures are of interest. First, a successful education program should demonstrate a sustained improvement in knowledge of pressure injuries and preventive behavior. Demonstration that acquisition of knowledge and skills translates to engagement in care through behavioral change is also an important outcome for education programs, as is demonstration that improvement in knowledge and skills leads to a direct influence on pressure injury prevention and/or healing. Patient-relevant outcomes (e.g., improved HRQoL) are also important. *Table 26.2* outlines the impact that different education and lifestyle skills programs had on the more widely reported outcome measures.

Table 26.2: Impact of education and lifestyle skills interventions on outcome measures of significance

	Follow up duration	Impact on QoL	Impact on knowledge	Impact on self-care skills	Impact on pressure injuries
Multi-faceted education program [#] (Level 1) ^{39,40}	24 months	---	Positive ⁴⁰ Sustained		Positive ³⁹ Sustained
Evidence-based pamphlets [#] (Level 5) ⁴⁴	2 days	---	---	Short term positive [¥]	---
E-learning program* (Level 5) ¹⁵	2 weeks	---	Short term positive [¥]	---	---
Positioning skills training program [#] (Level 1) ⁴³	1 day	---	---	Short term positive [¥]	---
Multi-faceted lifestyle skills program (Level 1) ¹²	24 months	Sustained positive* No difference [#]	No difference	Sustained positive* ^{#,¥}	No difference [#]
Multi-faceted lifestyle-skills program [#] (Level 1) ³⁶	8 weeks	---	Short term positive [¥]	Short term positive [¥]	Short term positive [¥]
Telephone-based lifestyle skills program* (Level 1) ¹³	12 weeks	Short term positive [¥]	---	Short term positive [¥]	Short term positive [¥]
Telephone-based (automated) education and support program* (Level 1) ^{37,38}	6 months	---	---	---	No difference
Telephone-based lifestyle skills and motivational interviewing program** (Level 1) ⁴¹	6 months	---	---	No difference	No difference
Smoking cessation program [#] (Level 3) ⁴²	6 months	---	---	---	Short term positive [¥]

[#] Compared to no education/skills program or standard care; + Compared to written education; ++ compared to standardized education

[¥] Short term describes outcomes measured at less than 12 months

* No comparator, change over time

When planning and delivering education and skills training consideration should be given to the psychological and cognitive status of the individual. Many studies excluded participants who had cognitive impairment^{12,13,36,39,41-44} and/or mental health impairments.³⁶ Guihan et al. (2014)⁴¹ noted depression rates of 40% in their study that failed to demonstrate either high engagement in the intervention or significant improvements in self-care behaviors in individuals receiving telephone support and motivational interviewing. Psychological status might negatively influence motivation to implement learned skills, even with regular telephone coaching and support.⁴¹ Education level, literacy, primary language and sensory impairments are just some factors that influence ability to engage in education. These factors are largely unexplored in relation to pressure injury education for patient consumers.¹⁸

Many of the education and lifestyle skills programs reported in the literature^{12,36,39,40} included more than one educational component, combining different education activity and methods of delivery. Table 26.3 presents a summary of strategies reported in the literature that demonstrated a benefit on at least one outcome measure of significance. The literature and programs are discussed in more detail throughout this chapter.

The development and delivery processes for some of the education material and programs explored in the research is reported in brief in some studies. Many programs were based on pressure injury clinical guidelines^{41,44} and literature reviews.¹² Most programs delivered by a trained health professional.^{12,13,39,41,43} This suggests that content of the programs was likely to reflect best practice. In a survey of hospitalized adults (n = 51), McInnes et al. (2014)⁴⁹ explored patient

perspectives on strategies to prevent pressure injuries, providing some insight on education delivery. The patient consumers identified strategies to help engage individuals in education, including managing pain, working together, and providing ongoing education (*Level 5*). This information underpinned the development of a patient engagement program that was reported by Chaboyer et al. (2017)²¹ as a component of a multi-faceted quality improvement program (see the guideline chapter *Implementing Best Practice in Clinical Settings*). A wide body of research and guidance is available on education principles and effective strategies in designing and delivering patient education. This guidance is likely to be relevant to the pressure injury field.

No cost-benefit analyses for education and lifestyle programs were identified in the literature. Costs for delivering one of the multi-faceted programs to individuals with SCI was reported as \$5,200 (USD in 2015) per participant. The program included one-on-one education and lifestyle skills counselling delivered in the individual's home by a trained health professional for 12 months, with additional support provided by telephone.¹² Costs are likely to vary greatly based on education format, resources, clinical setting and geographic location.

Education Programs

A multi-faceted pressure injury prevention program conducted in Australian acute and rehabilitation hospitals included a patient education program designed to promote engagement of individuals in pressure injury prevention. The program incorporated a digital resource, face-to-face education and posters. There was a significant reduction in pressure injury incident rate ratio associated with the intervention (IRR = 0.48, 95% CI 0.33 to 0.69, $p < 0.0001$).⁴⁸ The program⁴⁸ included other organizational level and staff-focused interventions that are discussed in detail in the guideline chapter *Implementing Best Practice in Clinical Settings (Level 1)*. The specific effect of patient education could not be isolated from other components of the intervention;⁴⁸ however, this study highlighted the importance of engaging individuals and their informal caregivers to collaborate with health professionals in addressing pressure injury prevention.

Rintala et al. (2008)^{39,40} developed a multi-faceted education program for veterans with SCI and multiple sclerosis (MS) who were recovering from pressure injury reconstruction surgery. Individuals were assigned to a multi-faceted education program delivered over four hours plus telephone support and education provided for two years ($n = 20$), a control group that received a monthly contact with a health professional without education intervention ($n = 11$), or to a second control group receiving infrequent support calls to track progress ($n = 10$). The intervention group received four, one-hour face-to-face education sessions, monthly structured follow-up, written material and telephone-based education when required. Their caregivers also received education and training. At the end of the program, knowledge acquisition was reinforced with a survey and question/answer session. The program received ongoing reinforcement through monthly telephone surveys that included reminders on positive lifestyle change. At 24 months' follow-up, significantly fewer individuals in the intervention group had experienced a pressure injury recurrence than either the monthly contact group or the minimal contact group (33% versus 60% versus 90%, $p = 0.007$). Odds ratio (OR) for experiencing a pressure injury recurrence for the intervention group was 0.228 (95% CI 0.080 to 0.647, $p = 0.03$)³⁹ (*Level 1*). Knowledge scores improved significantly over time between admission and discharge for all groups, but the intervention group showed statistically significantly better improvements (time by group, $F = 4.72$, $p < 0.04$).⁴⁰ The knowledge tests had not undergone psychometric testing (*Level 1*).

Two small trials^{14,15} have evaluated an e-learning program focused on prevention and treatment of pressure injuries. In two small cohorts of individuals with SCI, completion of the two-week internet-based education program was associated with improvement in knowledge, with median test scores increasing from 65% to 92.5% in one study,¹⁴ and from 80% to 89% in the second study.¹⁵ Participants rated the e-learning program positively on generic scales for evaluating the utility of internet-based resources¹⁵ (both *Level 5*). Translation of knowledge into behavioral change and reduction in pressure injuries were not measured in these studies.

Table 26.3: Intervention components that demonstrated a benefit on at least one outcome measure of significance

Type of Education Initiative	Setting and Level of Evidence
Group education sessions	<ul style="list-style-type: none"> Older men with multiple sclerosis or SCI undergoing pressure injury surgery in US^{39,40} (Levels 1 and 5) Individuals with SCI in rehabilitation in Korea³⁶ (Level 1)
Computer-based/online education modules	<ul style="list-style-type: none"> Older men with multiple sclerosis or SCI undergoing pressure injury surgery in US^{39,40} (Levels 1 and 5) Individuals with SCI in rehabilitation settings in US^{14,15} (Level 5) Individuals with SCI in rehabilitation in Korea³⁶ (Level 1) Individuals with SCI in rehabilitation settings in US⁵⁰ (Level 5)
Written education material	<ul style="list-style-type: none"> Older men with multiple sclerosis or SCI undergoing pressure injury surgery in US^{39,40} (Levels 1 and 5) Community-based individuals with SCI and pressure injuries in US⁴² (Level 3) Individuals undergoing surgery in Sweden⁴⁴ (Level 5) Community-based individuals with SCI in US¹² (Level 1)
Training informal caregivers	<ul style="list-style-type: none"> Older men with multiple sclerosis or SCI undergoing pressure injury surgery in US^{39,40} (Levels 1 and 5)
Telephone support and education	<ul style="list-style-type: none"> Older men with multiple sclerosis or SCI undergoing pressure injury surgery in US^{39,40} (Levels 1 and 5) Community-based individuals with SCI in US¹² (Level 1) Community-based individuals with SCI in Bangladesh⁵¹ (Level 1) Community-based individuals with SCI in Bangladesh¹³ (Level 1) Individuals with SCI in rehabilitation in Korea³⁶ (Level 1)
One-on-one education and/or counselling	<ul style="list-style-type: none"> Community-based individuals with SCI in US¹² (Level 1) Individuals with SCI in rehabilitation in Korea³⁶ (Level 1) Community-based individuals with SCI in Bangladesh⁵¹ (Level 1)
Therapist-consumer matching to promote rapport	<ul style="list-style-type: none"> Community-based individuals with SCI in US¹² (Level 1)
Skills training	<ul style="list-style-type: none"> Individuals with SCI in rehabilitation in Korea³⁶ (Level 1) Hospitalized individuals with SCI in US⁴³ (Level 1)

A small trial (n = 31)⁴⁴ set in a surgical ward demonstrated that providing written, evidence-based patient resources at the bedside was associated with significantly more individuals reporting having received education about pressure injury risks (13% vs 28%, p = 0.013), causative factors (13% vs 48%, p=0.001) and prevention strategies 14% vs 47%, p = 0.001). Of the group of individuals to the ward after adoption the education intervention, 46% reported engaging in preventive behaviors (e.g., regularly changing position and moving in the bed)⁴⁴ (Level 5). The follow-up was two days after provision of education material, and the data for self-care engagement relied on self-report. The low level of implementation by recipients of the advice provided in the written pamphlets suggests that more active methods of delivering education may be required to change behavior.

Lifestyle Skills Programs

Some studies^{12,36} have explored interventions that include both education resources and skills training, usually providing active learning opportunities for participants to promote behavior change. These programs, broadly referred to as lifestyle skills programs in this guideline, incorporate education, practical skills demonstration and practice (e.g., skin checks, positioning and pressure relief maneuvers), psychological support/counselling, and support in accessing services and social opportunities. These holistic programs are generally delivered using a multi-faceted approach combining various modes of content delivery to accommodate different learning styles and preferences.

Kim and Cho (2017)³⁶ investigated a multi-faceted self-efficacy program for improving knowledge and self-care and preventing pressure injuries in individuals with SCI in rehabilitation. The intervention (n = 24) consisted of small group education, skills training, computer-based education and both face-to-face and telephone-based one-on-one counselling. The control group (n = 23) received a written education booklet. Outcome measures were self-care knowledge, self-efficacy, self-care behavior and pressure injury incidence. The one pressure injury that occurred in this study was in the control group (p = 0.489). Both groups showed statistically significant improvements over time for knowledge, self-efficacy and self-care. The improvements were greater in the intervention group compared to

the control group for knowledge (18.83 ± 1.61 versus 15.78 ± 2.50 , $p = 0.004$), self-efficacy (45.21 ± 3.37 versus 41.78 ± 4.58 , $p < 0.001$) and self-care behavior (92.29 ± 5.21 versus 77.1 ± 12.81 , $p < 0.001$).³⁶ The follow-up duration was only eight weeks; therefore, the sustainability of improvements is unclear, and specific content of the program was not reported (*Level 1*).

However, a longer study conducted over 24 months failed to demonstrate sustainable improvements in pressure injury rates associated with a multi-faceted lifestyle-skills program. In a trial conducted in community-based individuals with SCI who had experienced a full thickness pressure injury in the preceding five years ($n = 170$),¹² individuals were randomized to receive either a multifaceted lifestyle skills program or usual care (clinic visits for skin checks). The intervention was based on clinical guidelines and consisted of six modules that included general knowledge, lifestyle skills, exercises, activities and adaptation of information to the individual's circumstances. The intervention was delivered by health professionals in the individual's home over six months, with an additional phase of six months in which the intervention was tapered. Support was also provided via telephone. At the final 24-month follow-up, there was no significant difference in the annualized rate of Category/Stage III or IV pressure injuries between the intervention and control groups (0.44 versus 0.39, rate ratio 1.14, 95% CI 0.72 to 1.82, $p > 0.05$). Participants in both groups experienced improvements over time in measures of HRQoL, with no significant between-group differences. No improvements were apparent for measures of pressure injury knowledge in either group.¹² However, the lifestyle intervention was associated with improved performance of preventive behavior over time ($p = 0.005$), and compared to the usual care group ($p = 0.01$) (*Level 1*).

Telephone-based programs

Telephone-based programs have been explored in more detail in the research;^{13,37,41,42,51} however, their impact on preventing pressure injuries is largely unquantifiable. Findings in the research are mixed and limited by methodological quality of the available evidence and the highly varied programs and geographic settings in which they have been delivered.

In one trial ($n = 158$),⁴² introduction of a smoking cessation program was demonstrated to have a positive influence on pressure injury healing. Individuals with SCI and pressure injuries were engaged in consultations that included use of the 5As (i.e., Ask, Advise, Assess, Assist and Arrange) to promote and support smoking cessation. This program included one-on-one education and provision of written material. More individuals exposed to the program had ceased smoking within six months compared to a cohort not receiving the program (44% versus 21%, $p = 0.03$). The study also showed that individuals who ceased smoking had statistically significantly superior healing outcomes at six months in terms of number of pressure injuries healed and reduction in pressure injury size compared with never-smokers and continuing smokers.⁴² Confirmation of smoking cessation was via verbal report and the reliability of self-report may have influenced the results (*Level 3*).

Houlihan et al. (2013)³⁷ trialed an automated, voice response telephone-based education and support program for individuals with SCI. Individuals in the intervention group ($n = 71$) received education, cognitive behavioral interventions, screening and referrals to health professionals. The control group ($n = 71$) received usual care consisting of an education resource book. After controlling for baseline number of pressure injuries, age and gender, there was no significant between-group difference at six months in pressure injury prevalence. There were some benefits demonstrated for depression outcomes, and the researchers noted that the intervention had potential cost-savings, although a cost-analysis was not presented. The researchers also proposed that an individualized telephone-based service might provide greater success (*Level 1*).

However, the research on individualized telephone-based education and support services is mixed.^{13,41} Arora et al. (2017)¹³ conducted a randomized controlled trial (RCT) to evaluate a multi-faceted telephone-based program for individuals with SCI and pressure injuries living in the community ($n = 120$). The intervention consisted of a written pressure injury prevention pamphlet, weekly individualized phone calls from a health professional for 12 weeks and engagement of families in education. Advice on equipment, psychosocial support and assistance to develop weekly goals was provided over the telephone. The control group received only the written education. At 12 weeks' follow-up, the individualized telephone support group showed significantly greater reduction in pressure injury size (between group difference 2.3cm^2 , 95% CI -0.3 to 4.9 ; $p = 0.008$). The intervention group also reported greater confidence in managing their pressure injury (mean between group difference of 1.7 on a 10-point scale, 95% CI 1.0 to 2.3, $p < 0.001$) and greater improvements in QoL measured on the EQ-5D (between-group difference of 10.5 on 100-point scale, 95% CI 4.5 to 16.6; $p = 0.001$) (*Level 1*).

However, Guhan et al. (2017)⁴¹ failed to demonstrate additional benefits of a telephone-based motivational interviewing plus a skills training group ($n = 71$) compared to telephone-based education plus group education ($n = 72$). In this study, individuals with SCI and existing Category/Stage III or IV pressure injuries were randomized to one of the two groups, both of which received a minimum of four support calls over six months. Over six months, neither group showed a significant increase in implementation self-care behaviors ($p = 0.45$). There was also no significant

between-group difference for percent of self-care behaviors being performed (mean 85.0% \pm 15.2% versus 83.0% \pm 14.6%, $p = 0.41$). Difference at six months in pressure injuries described as having worsened was also not statistically significant ($p = 0.86$)⁴¹ (*Level 1*).

Guihan et al. (2017)⁴¹ reported high feasibility for both interventions reported above (over 80% of support calls were made in both study groups). However, there was low engagement by participants in the calls in both groups. In the telephone-based motivational interviewing plus skills training group 36% of participants engaged in all support calls compared to 22% in the comparator group ($p = 0.07$)⁴¹ (*Level 1*). This contrasted to the findings of Houlihan et al. (2013),³⁷ who reported that 78% of participants receiving their automated telephone-based education and support intervention adhered to the intervention (*Level 1*). A secondary analysis³⁸ of Houlihan's (2013)³⁷ trial reported high levels of satisfaction with the automated telephone service, with 70% of participants rating the intervention as 'most useful'. Less than 10% of participants rated the comparator, a written education resource, as useful³⁸ (*Level 1*). In a pilot RCT ($n = 30$), Hossain et al. (2017)⁵¹ explored the feasibility of delivering a telephone-based education and support by a health professional every two weeks for 12 months, followed by monthly for 12 months. The researchers deemed that the intervention was feasible to deliver to individuals with SCI in Bangladesh, with 87% of support calls and 100% of home visits being conducted in this study⁵¹ (*Level 1*).

Engaging patient consumers and their informal caregivers in quality improvement programs within facilities is also significant in preventing facility-acquired pressure injuries. Patient engagement in multi-facet facility-based quality improvement programs is recommended in this guideline and is discussed in the guideline chapter *Implementing Best Practice in Clinical Settings*.

Education Content for Patient Consumers and Informal Caregivers

There was no evidence in which education programs with explicitly different content were compared. Few studies reported their program content in detail. Topics that were included in education interventions reported in the literature included:^{12,13,17,18,40,52}

- Etiology of pressure injuries, including skin anatomy and how pressure injuries form
- Nutrition and healthy diet choices
- Healthy lifestyle choices (e.g., alcohol and tobacco use)
- Hygiene and continence management
- Pressure redistribution strategies for sitting and lying
- Skin inspection and skin care
- Wound care
- Exercise (appropriate to co-morbidities)
- Use of wheelchairs
- Use of cushions and mattresses
- Equipment maintenance
- Appropriate clothing and shoes
- Lifestyle skills (e.g., financial management)
- Advocacy, self-help, life planning and goals
- Well-being, social support and coping strategies.

International Survey of Patient Consumer Education Needs

As a part of this guideline development, an international survey of patient consumers and informal caregivers was undertaken to establish areas of education need. Of the 1,233 respondents ($n = 383$ individuals with or at risk of pressure injuries; $n = 850$ caring for such an individual), more than 80% rated 14 pressure injury education topics as being important or very important to receive information on for their care (see *Table 26.4*). These results suggest that individuals with or at risk of pressure injuries place a high priority on education and consider a range of pressure injury related topics to be relevant to their education needs. However, the participants were self-selecting and may have a high interest in education. Most participants who had experienced or were at risk of a pressure injury (63%) were aged below 50 years and resided in Asiatic countries (90.86%), which may not be representative of an average individual with pressure injuries. However, representation from a wide range of countries ($n = 16$) suggested that patient consumers and their informal caregivers across the world place similar priority on receiving education on pressure injury prevention and treatment^{53,54} (*Level 5*).

Table 26.4: Patient consumer rating of importance for pressure injury education topics (n = 383)^{53,54}

	No response (%)	Not at all important (%)	Not important (%)	Neutral (%)	Important (%)	Very important (%)
How pressure injury happens	9.66	6.53	1.31	5.48	33.42	43.60
Risk factors	9.92	6.27	0.26	6.53	32.11	44.91
Hospital plans to prevent pressure injuries	10.44	5.48	0.78	4.44	33.68	45.17
Carers knowing about pressure injuries	9.66	5.48	0.78	1.57	35.25	47.26
Where to get more information	12.27	5.74	1.57	8.88	35.77	35.77
Diet	10.44	5.48	1.57	10.70	33.94	37.86
Beds and chairs	12.01	5.22	2.87	11.23	35.25	33.42
Positioning	9.92	5.74	2.09	6.01	32.90	43.34
Medical devices	15.14	6.79	2.09	9.14	32.38	34.46
Skin assessment/skin checks	11.75	5.22	1.57	8.88	33.42	39.16
Skin care	12.01	4.96	0.52	3.13	31.33	48.04
Immobile patients	9.40	5.74	1.31	3.66	30.03	49.87
How to help pressure injuries heal	11.75	5.22	0.52	4.96	32.11	45.43
Assessing pressure injuries	11.23	4.96	1.31	6.27	34.46	41.78
Dressings	13.05	5.48	0.78	8.62	31.85	40.21
Pain management	11.23	6.27	0.52	7.57	32.11	42.30

References

- Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Adult and Community Health, Measuring Healthy Days: Population Assessment of Health-Related Quality of Life. 2000, US Department of Health and Human Services <https://www.cdc.gov/hrqol/pdfs/mhd.pdf>.
- Atkin L, Bucko Z, Conde Montero E, Moffatt CJ, Probst A, Romanelli M, Schultz GS, Tettlebach W. Implementing TIMERS: the race against hard-to-heal wounds. *J Wound Care*, 2019; 28(3 Suppl 3): S1-S49.
- Degenholtz H, Rosen J, Castle N, Mittal V, Liu D. The association between changes in health status and nursing home resident quality of life. *Gerontologist*, 2008; 48(5): 584-584.
- Essex HN, Clark M, Sims J, Warriner A, Cullum N. Health-related quality of life in hospital inpatients with pressure ulceration: Assessment using generic health-related quality of life measures. *Wound Repair Regen*, 2009; 17(6): 797-805.
- Galhardo VAC, Magalhaes MG, Blanes L, Juliano Y, Ferreira LM. Health-related quality of life and depression in older patients with pressure ulcers. *Wounds: A Compendium of Clinical Research & Practice*, 2010; 22(1): 20-26.
- Thein H-H, Gomes T, Krahn MD, Wodchis WP. Health status utilities and the impact of pressure ulcers in long-term care residents in Ontario. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care and Rehabilitation*, 2010; 19(1): 81-89.
- Yarkin O, Tamer S, Gamze O, Irem M, Huseyin B. Effect of surgery on psychiatric states and quality of life of paraplegics and quadriplegics with pressure sores and their primary caregivers. *Eur J Plast Surg*, 2009; 32(4): 173-176.
- Gorecki C, Lamping DL, Brown JM, Madill A, Firth J, Nixon J. Development of a conceptual framework of health-related quality of life in pressure ulcers: A patient-focused approach. *Int J Nurs Stud*, 2010; 47(12): 1525-1534.
- Gorecki C, Nixon J, Madill A, Firth J, Brown JM. What influences the impact of pressure ulcers on health-related quality of life? A qualitative patient-focused exploration of contributory factors. *Journal of Tissue Viability*, 2012; 21(1): 3-12.
- Jackson DE, Durrant LA, Hutchinson M, Ballard CA, Neville S, Usher K. Living with multiple losses: Insights from patients living with pressure injury. *Collegian*, 2017.
- Latimer S, Chaboyer W, Gillespie B. Patient participation in pressure injury prevention: Giving patient's a voice. *Scand J Caring Sci*, 2014; 28(4): 648-656.
- Carlson M, Vigen CL, Rubayi S, Blanche EI, Blanchard J, Atkins M, Bates-Jensen B, Garber SL, Pyatak EA, Diaz J, Florindez LI, Hay JW, Mallinson T, Unger JB, Azen SP, Scott M, Cogan A, Clark F. Lifestyle intervention for adults with spinal cord injury: Results of the USC-RLANRC Pressure Ulcer Prevention Study. *J Spinal Cord Med*, 2017: 1-18.
- Arora M, Harvey LA, Glinsky JV, Chhabra HS, Hossain S, Arumugam N, Bedi PK, Lavrencic L, Hayes AJ, Cameron ID. Telephone-based management of pressure ulcers in people with spinal cord injury in low-and middle-income countries: A randomised controlled trial. *Spinal Cord*, 2017; 55(2): 141-147.

14. Brace JA, Schubart JR. A prospective evaluation of a pressure ulcer prevention and management e-learning program for adults with spinal cord injury. *Ostomy Wound Management*, 2010; 56(8): 40-50.
15. Schubart J. An e-learning program to prevent pressure ulcers in adults with spinal cord injury: a pre- and post- pilot test among rehabilitation patients following discharge to home. *Ostomy Wound Manage*, 2012; 58(10): 38-49.
16. Thietje R, Giese R, Pouw M, Kaphengst C, Hosman A, Kienast B, Hirschfeld S. How does knowledge about spinal cord injury-related complications develop in subjects with spinal cord injury? A descriptive analysis in 214 patients. *Spinal Cord*, 2011; 49(1): 43-48.
17. Hartigan I, Murphy S, Hickey M. Older adults' knowledge of pressure ulcer prevention: a prospective quasi-experimental study. *Int J Older People Nurs*, 2012; 7(3): 208-218.
18. Schubart JR, Hilgart M, Lyder C. Pressure ulcer prevention and management in spinal cord-injured adults: Analysis of educational needs. *Adv Skin Wound Care*, 2008; 21(7): 322-329.
19. Dunn CA, Carlson M, Jackson JM, Clark FA. Response factors surrounding progression of pressure ulcers in community-residing adults with spinal cord injury. *Am J Occup Ther*, 2009; 63(3): 301-309.
20. Ghaisas S, Pyatak EA, Blanche E, Blanchard J, Clark F. Lifestyle changes and pressure ulcer prevention in adults with spinal cord injury in the pressure ulcer prevention study lifestyle intervention. *Am J Occup Ther*, 2015; 69(1): 1-10.
21. Chaboyer W, Harbeck E, Bucknall T, McInnes E, Thalib L, Whitty J, Wallis M, Gillespie B. Initial psychometric testing and validation of the Patient Participation in Pressure Injury Prevention scale. *J Adv Nurs*, 2017.
22. Gelis A, Stefan A, Coudeyre E, Fattal C. Therapeutic education for pressure ulcer care management in paraplegics: The ETP SOFMER guide. *Annals of Physical and Rehabilitation Medicine*, 2014; 57: e58-e59.
23. Gorecki C, Nixon J, Lamping DL, Alavi Y, Brown JM. Patient-reported outcome measures for chronic wounds with particular reference to pressure ulcer research: A systematic review. *International Journal of Nursing Studies*, 2014; 51(1): 157-165.
24. Kisala PA, Tulsy DS, Choi SW, Kirshblum SC. Development and psychometric characteristics of the SCI-QOL Pressure Ulcers Scale and short form. *J Spinal Cord Med*, 2015; 38(3): 303-314.
25. Rutherford C, Brown JM, Smith I, McGinnis E, Wilson L, Gilberts R, Brown S, Coleman S, Collier H, Nixon J. A patient-reported pressure ulcer health-related quality of life instrument for use in prevention trials (PU-QOL-P): Psychometric evaluation. *Health Qual Life Outcome*, 2018; 16(1): 227.
26. de Laat HE, de Munter AC, van der Burg MJ, Ulrich DJ, Kloeters O. A cross-sectional study on self-management of pressure ulcer prevention in paraplegic patients. *J Tissue Viability*, 2017; 26(1): 69-74.
27. Lourenco L, Blanes L, Salomé GM, Ferreira LM. Quality of life and self-esteem in patients with paraplegia and pressure ulcers: A controlled cross-sectional study. *J Wound Care*, 2014; 23(6): 331-337.
28. Palfreyman S, Mulhern B. The psychometric performance of generic preference-based measures for patients with pressure ulcers. *Health and Quality of Life Outcomes*, 2015; 13(1).
29. Gorecki C, Brown JM, Cano S, Lamping DL, Briggs M, Coleman S, Dealey C, McGinnis E, Nelson AE, Stubbs N, Wilson L, Nixon J. Development and validation of a new patient-reported outcome measure for patients with pressure ulcers: The PU-QOL instrument. 2013: 95.
30. Gélis A, Daures JP, Benaim C, Kennedy P, Albert T, Colin D, Joseph PA, Pelissier J, Fattal C. Evaluating self-reported pressure ulcer prevention measures in persons with spinal cord injury using the revised Skin Management Needs Assessment Checklist: Reliability study. *Spinal Cord*, 2011; 49(5): 653-658.
31. EuroQol. *EQ-5D® 2019* [cited August 2019]; Available from: <https://euroqol.org/>.
32. RAND Health Care. *36-Item Short Form Survey (SF-36)*. 2019 [cited August 2019]; Available from: https://www.rand.org/health-care/surveys_tools/mos/36-item-short-form.html.
33. University of Sheffield. *Measuring and Valuing Health*. 2019 [cited August 2019]; Available from: <https://www.sheffield.ac.uk/scharr/sections/heds/mvh/sf-6d>.
34. Insignia Health. *Patient Activation Measure®(PAM®) 13*. 2015 [cited August 2019]; Available from: http://solihulltogether.co.uk/images/Patient_Activation/UK_PAM_13.pdf.
35. Spinal Cord Injury Research Evidence Professional. *Skin Management Needs Assessment Checklist (SMNAC)*. 2017 [cited August 2019]; Available from: <https://scireproject.com/outcome-measures/outcome-measure-tool/skin-management-needs-assessment-checklist-smnac/#1467983894080-2c29ca8d-88af>.
36. Kim JY, Cho E. Evaluation of a self-efficacy enhancement program to prevent pressure ulcers in patients with a spinal cord injury. *Jpn J Nurs Sci*, 2017; 14(1): 76-86.
37. Houlihan BV, Jette A, Friedman RH, Paasche-Orlow M, Ni P, Wierbicky J, Williams K, Ducharme S, Zazula J, Cuevas P, Rosenblum D, Williams S. A pilot study of a telehealth intervention for persons with spinal cord dysfunction. *Spinal Cord*, 2013; 51(9): 715-20.
38. Mercier HW, Ni P, Houlihan BV, Jette AM. Differential impact and use of a telehealth intervention by persons with MS or SCI. *Am J Phys Med Rehabil*, 2015; 94(11): 987-99.
39. Rintala DH, Garber SL, Friedman JD, Holmes SA. Preventing recurrent pressure ulcers in veterans with spinal cord injury: impact of a structured education and follow-up intervention. *Arch Phys Med Rehabil*, 2008; 89(8): 1429-1441.
40. Garber SL, Rintala DH, Holmes SA, Rodriguez GP, Friedman J. A structured educational model to improve pressure ulcer prevention knowledge in veterans with spinal cord dysfunction. *J Rehabil Res Dev*, 2002; 39(5): 575-588.
41. Guihan M, Bombardier CH, Ehde DM, Rapacki LM, Rogers TJ, Bates-Jensen B, Thomas FP, Parachuri R, Holmes SA. Comparing multicomponent interventions to improve skin care behaviors and prevent recurrence in veterans hospitalized for severe pressure ulcers. *Arch Phys Med Rehabil*, 2014; 95(7): 1246-1253.e3.
42. Lane CA, Selleck C, Chen Y, Tang Y. The impact of smoking and smoking cessation on wound healing in spinal cord-injured patients with pressure injuries: A retrospective comparison cohort study. *Journal of Wound, Ostomy, & Continence Nursing*, 2016; 43(5): 483-7.

43. Rottkamp BC. An experimental nursing study: A behavior modification approach to nursing therapeutics in body positioning of spinal cord-injured patients. *Nurs Res*, 1976; 25(3): 181-186.
44. Schoeps LN, Tallberg AB, Gunningberg L. Patients' knowledge of and participation in preventing pressure ulcers- an intervention study. *Int Wound J*, 2017; 14(2): 344-348.
45. Royal Australian College of General Practice (RACGP). *Guidelines for Preventive Activities in General Practice. Patient Education and Literacy*. 2016; Available from: <https://www.racgp.org.au/FSEDEDEV/media/documents/Clinical%20Resources/Guidelines/Red%20Book/Guidelines-for-preventive-activities-in-general-practice.pdf>.
46. Agency for Healthcare Research and Quality (AHRQ). *The Patient Education Materials Assessment Tool (PEMAT) and User's Guide An Instrument To Assess the Understandability and Actionability of Print and Audiovisual Patient Education Materials*. [cited August 2019]; Available from: <https://www.ahrq.gov/ncepctr/tools/self-mgmt/pemat.html>.
47. DeWalt DA, Callahan LF, Hawk V, Broucksou KA, Hink A, Rudd R, Brach C, *Health Literacy Universal Precautions Toolkit. AHRQ Publication No. 10-0046-EF*. 2010, <https://www.ahrq.gov/sites/default/files/wysiwyg/professionals/quality-patient-safety/quality-resources/tools/literacy-toolkit/healthliteracytoolkit.pdf>: Agency for Healthcare Research and Quality (AHRQ).
48. Chaboyer W, Bucknall T, Webster J, McInnes E, Gillespie BM, Banks M, Whitty JA, Thalib L, Roberts S, Tallott M, Cullum N, Wallis M. The effect of a patient centred care bundle intervention on pressure ulcer incidence (INTACT): A cluster randomised trial. *Int J Nurs Stud*, 2016; 64: 63-71.
49. McInnes E, Chaboyer W, Murray E, Allen T, Jones P. The role of patients in pressure injury prevention: a survey of acute care patients. *BMC Nurs*, 2014; 13(1): 41.
50. Hilgart M, Ritterband L, Baxter K, Alfano A, Ratliff C, Kinzie M, Cohn W, Whaley D, Lord H, Garber S. Development and perceived utility and impact of a skin care internet intervention. *Internet Interventions*, 2015; 1(3): 149-157.
51. Hossain MS, Harvey LA, Rahman MA, Bowden JL, Islam MS, Taylor V, Muldoon S, Herbert RD. A pilot randomised trial of community-based care following discharge from hospital with a recent spinal cord injury in Bangladesh. *Clin Rehabil*, 2017; 31(6): 781-789.
52. Krause JS, Broderick L. Patterns of recurrent pressure ulcers after spinal cord injury: Identification of risk and protective factors 5 or more years after onset. *Arch Phys Med Rehabil*, 2004; 85(8): 1257-1264.
53. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
54. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis

QUALITY INDICATORS FOR THIS GUIDELINE

Introduction

Quality indicators are used across healthcare to assess, measure, and improve the quality of care delivered to patient consumers and their families.^{1,2} The quality indicators presented in this section of the guideline are examples intended to assist healthcare organizations to implement and monitor the strategies recommended in this clinical guideline for the prevention and treatment of pressure injuries. The quality indicators have been developed to reflect recommendations and current best practice outlined in this clinical guideline. The indicators are not intended to be prescriptive, or to replace other quality indicators in common use. Healthcare organizations that choose to use these guideline-specific quality indicators may use them in isolation or in addition to other local, national or international quality indicators.

Continuous quality improvement is a process by which healthcare organizations ensure systematic and intentional improvement of services to their consumers. Clinical guidelines are developed to improve quality of care through promotion of the most effective and safe interventions for managing clinical conditions. They provide recommendations on care that the clinician and healthcare organization can use to deliver quality care, in conjunction with clinical judgment and the consumer's personal wishes. Therefore, using clinical guidelines are an efficient, appropriate and resource-saving approach to derive quality indicators.¹

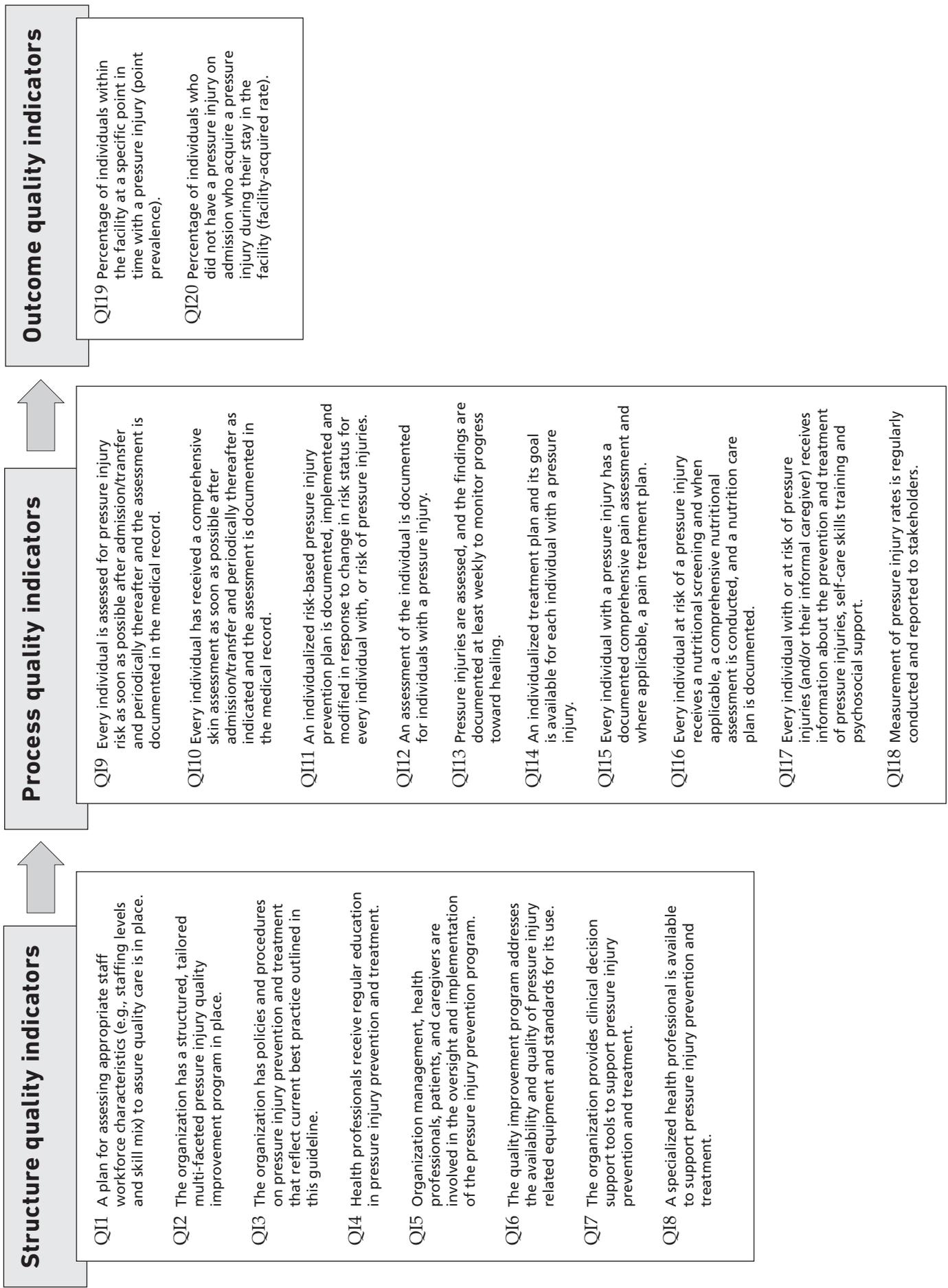
To gain insight into whether the quality of care being delivered reflects the best practice outlined in this clinical guideline and effectively addresses the individual's care needs, some form of evaluation is required. Quality indicators are developed as a measure of care to monitor quality and initiate future improvements.

Quality indicators

Quality indicators measure a range of different indicators of the quality of care delivered within an organization. One way in which quality indicators can be distinguished is as internal or external indicators. Internal quality indicators are used by healthcare providers to monitor and improve the outcomes of their care processes. Health professionals and managers can use these data to investigate where potential problems lie, and how they may be approached. On the basis of such analyses, care processes may be redesigned, and the indicators can then be used to monitor the influence of these improvement initiatives. Progress toward meeting internal quality indicators may be maintained confidentially, or used to benchmark against other organizations. In contrast, external indicators are used by various stakeholders (e.g., governments, accreditors and consumer organizations) to assess quality of care and cost-effectiveness. Comparison of results among organizations provides an indication of performance on a local, national or international level, and indicates how an organization benchmarks and performs against others. Often external quality indicators are publicly accessible.

The quality indicators in this chapter are presented using the now commonly accepted categorization developed by Donabedian (1988)³ that relates to the type of care delivery a given indicator addresses: structure, process or outcome. Structure indicators are related to attributes of the care setting, including organizational structure, material resources (e.g., environment, technology and tools), and human resources. These attributes or characteristics of the clinical setting often provide insight into sustainability of quality improvement initiatives. Process indicators measure activities and tasks required to implement patient care at the care level (e.g., clinical procedures, documentation methods and use of clinical tools). Outcome indicators describe the healthcare effects at the individual patient level (e.g., measurement, prevalence, and incidence).^{3,4}

Figure 27.1: Quality indicators (QI) for pressure injuries based on recommendations and good practice statements in this guideline



Structure Quality Indicators

QI 1: A plan for assessing appropriate staff workforce characteristics (e.g., staffing levels and skill mix) to assure quality care is in place.

Description	The organization has a plan for assuring appropriate staffing levels and staff skill mix that reflects current best practice.
Question	Does the organization have a plan for appropriate staffing levels and skill mix that reflects current best practice?
Definition(s)	The organization's staff workforce plan should reflect the current best practice to assure quality of care is provided.
Source	Organization.
Measurement level	Organizational.
Rationale	A plan that reflects evidence-based staff workforce levels and skill mix ensures there is an adequate workforce of appropriately trained care staff to prevent and treat pressure injuries in accordance with best available evidence.
Evidence rationale	See <i>Recommendation 20.1</i> .

QI 2: The organization has a structured, tailored multi-faceted pressure injury quality improvement program in place.

Description	The organization has a structured, tailored multi-faceted pressure injury quality improvement program in place.
Question	Does the organization have a tailored multi-faceted (e.g., bundle), pressure injury quality improvement program in place?
Definition(s)	The organization's pressure injury quality improvement program includes a structured, tailored multi-faceted bundle of prevention strategies/interventions that addresses the specific needs of facility stakeholders.
Source	Organization.
Measurement level	Organizational.
Rationale	A pressure injury prevention program that reflects evidence-based strategies to address the specific needs of the facility stakeholders to prevent and treat pressure injuries in accordance with best available evidence.
Evidence rationale	See <i>Recommendation 20.5</i> .

QI 3: The organization has policies and procedures on pressure injury prevention and treatment that reflect the current best practice outlined in this guideline.

Description	The organization has policies and procedures governing the prevention and treatment of pressure injuries that reflects current best practice and that is relevant to the clinical setting.
Question	Does the organization have a policy/protocol governing the prevention and treatment of pressure injuries that reflects current best practice?
Definition(s)	The organization's policies and procedures governing the prevention and treatment of pressure injuries should reflect the current best practice outlined in this international guideline and relevant local requirements.
Source	Organization.
Measurement level	Organizational.
Rationale	Policies and procedures that reflect current best practice in the prevention and treatment of pressure injuries determine interventions for preventing and treating pressure injuries and promote care delivery that is in accordance with best available evidence.
Evidence rationale	See <i>Recommendation 20.7</i> .

QI 4: Health professionals receive regular education in pressure injury prevention and treatment.

Description	Knowledge and skills of health professionals regarding pressure injury prevention and treatment must be current. This can be accomplished by providing regular access to mandatory evidence-based education.
Question	Have all health professionals attended recent evidence-based training in pressure injury prevention and treatment?
Definition(s)	Education and training refer to evidence-based education in pressure injuries.
Source	Training calendar/health professional records.
Measurement level	Organizational and/or departmental.
Rationale	Receiving mandatory education on a regular basis promotes evidence-based knowledge and care delivery.
Evidence rationale	See <i>Recommendation 21.2</i> .

QI 5: Organization management, health professionals, patients, and caregivers are involved in the oversight and implementation of the pressure injury prevention program.

Description	Management, health professionals, patients, and caregivers are involved in the oversight and implementation of the pressure injury prevention program.
Question	Are management, health professionals, patients, and caregivers involved in the oversight and implementation of the pressure injury prevention program?
Definition(s)	Management, health professionals, patients, and caregivers participate in the development, oversight and implementation of the pressure injury prevention program.
Source	Organization or department.
Measurement level	Organizational and/or departmental.
Rationale	Engagement of management, interdisciplinary care staff, patients/caregivers and key stakeholders promotes team-decision making, patient-centered care and successful quality improvement.
Evidence rationale	See <i>Recommendation 20.6</i> .

QI 6: The quality improvement program addresses the availability and quality of pressure injury related equipment and standards for its use.

Description	A protocol for ensuring availability and allocation of pressure injury related equipment (e.g., pressure redistribution support surfaces) that are of an appropriate quality based on a national or international guideline, regulation or standard, must be available.
Question	Is a protocol for ensuring availability and allocation of pressure injury related equipment based on a national or international guideline, regulation or standard available?
Definition(s)	Pressure injury related equipment includes preventive equipment (e.g., pressure redistribution support surfaces, repositioning devices and wound care supplies) that is of a quality appropriate for its use based on relevant equipment standards and testing protocols.
Source	Document management system.
Measurement level	Departmental.
Rationale	Implementation of a quality improvement program will support decision making related to use of pressure injury related equipment, and equipment of an appropriate quality will be provided in a timely manner to enable deliver of quality care.
Evidence rationale	See <i>Recommendation 20.4</i> .

QI 7: The organization provides clinical decision support tools to support pressure injury prevention.

Description	Clinical decision support tools to guide pressure injury prevention are available to healthcare staff.
Question	Are clinical decision support tools to guide pressure injury prevention available to healthcare staff?
Definition(s)	Clinical decision support tools may include computer-generated reports, risk assessment decision support protocols, support surface algorithms, and other technology-based tools integrated into the electronic health record.
Source	Document management system.
Measurement level	Departmental.
Rationale	Availability of clinical decision support tools will guide decision making related to pressure injury prevention best practice.
Evidence rationale	See <i>Recommendation 20.8</i> .

QI 8: A specialized health professional is available to support pressure injury prevention and treatment.

Description	A specialized health professional (i.e., a wound expert clinical leader) is available to support pressure injury prevention and treatment.
Question	Is a specialized health professional (i.e., a wound expert clinical leader) available to support pressure injury prevention and treatment?
Definition(s)	Identification and delegation of a wound expert clinical leader to lead and support pressure injury prevention and treatment.
Source	Organization.
Measurement level	Organizational.
Rationale	Availability of a wound expert clinical leader has been shown to be a key component in the successful delivery of pressure injury prevention and treatment.
Evidence rationale	See <i>Recommendation 20.9</i> .

Process Quality Indicators**QI 9: Every individual is assessed for pressure injury risk as soon as possible after admission/transfer and periodically thereafter and the assessment is documented in the medical record.**

Description	The percentage of individuals assessed for risk of developing a pressure injury is documented as soon as possible following admission or transfer to the healthcare service and periodically thereafter using a structured approach that considers major risk factors, a valid and reliable risk assessment instrument, clinical judgment and is replicable.
Numerator	Number of individuals whose risk for developing a pressure injury is assessed and documented as soon as possible (i.e., first contact with a health professional, or at the first visit for those in community care).
Denominator	All admissions of at least 24 hours' duration.
Definition(s)	The risk for developing a pressure injury is determined using a structured approach that incorporates assessment of the multiple epidemiological factors that increase the risk of pressure injury development and clinical judgment.
Inclusion criteria	All admissions of at least 24 hours' duration.
Source	Medical records.
Measurement level	Patient.
Rationale	Assessing the risk of pressure injury on admission facilitates the (timely) application of individualized preventive measures to reduce the risk of pressure injury.
Evidence rationale	See <i>Recommendations 1.21 to 1.23</i> .

QI 10: Every individual has received a comprehensive skin assessment as soon as possible after admission/transfer and periodically thereafter as indicated and the assessment is documented in the medical record.

Description	The percentage of individuals whose skin is assessed and documented as soon as possible after admission/transfer to the healthcare service and periodically thereafter.
Numerator	Number of individuals whose skin is assessed and documented as soon as possible after admission (i.e., first contact with a health professional for inpatients, or at the first visit for those in community care).
Denominator	All admissions of at least 24 hours' duration.
Definition(s)	Alterations to skin integrity provide an indication of pressure injury risk. A comprehensive head-to-toe skin assessment identifies any existing pressure injuries and contributes to a risk assessment.
Inclusion criteria	All admissions at least 24 hours' duration.
Source	Medical records.
Measurement level	Patient.
Rationale	Assessing the skin on admission facilitates the (timely) application of pressure injury prevention strategies and appropriate wound care and contributes to the development of an individualized pressure injury prevention plan.
Evidence rationale	See <i>Recommendation 2.1</i> .

QI 11: An individualized risk-based pressure injury prevention plan is developed, documented, implemented and modified in response to change in risk status for every individual at risk of, or with, pressure injuries.

Description	The percentage of individuals at risk of, or with, pressure injuries for whom an individualized risk-based pressure injury prevention plan has been developed, documented and implemented. The plan should be modified as risk status worsens or improves.
Numerator	The number of individuals at risk of, or with, pressure injuries for whom an individualized risk-based pressure injury prevention plan has been developed, documented and implemented.
Denominator	The number of individuals at risk of, or with, pressure injuries.
Definition(s)	An individualized risk-based pressure injury prevention plan should consider, at a minimum , the individual's specific pressure injury risk factors, nutrition, repositioning, pressure redistribution support surfaces, and topical skin care. The plan should be consistent with the individual's goals and wishes. The risks and benefits of pressure injury prevention should be explained to the individual and/or family so that informed decisions can be made.
Exclusion criteria	Individuals who have documented consistent informed refusal of preventive care are excluded.
Source	Medical records.
Measurement level	Patient.
Rationale	Developing and implementing individualized preventive measures reduces the risk of developing a new pressure injury.
Evidence rationale	See <i>Recommendation 1.24</i> . The guideline chapters <i>Preventive Skin Care, Nutrition in Pressure Injury Prevention and Treatment, Support Surfaces and Repositioning and Early Mobilization</i> detail appropriate components of a risk-based pressure injury prevention plan.

QI 12: An assessment of the individual is documented for individuals with a pressure injury.

Description	The percentage of individuals with a pressure injury for whom there is a documented comprehensive assessment.
Numerator	The number of individuals with a pressure injury for whom there is a documented comprehensive assessment.
Denominator	The number of individuals with a pressure injury.
Definition(s)	A comprehensive assessment must meet the criteria as described in <i>Recommendation 10.1</i> in this guideline.
Inclusion criteria	Individuals with pressure injuries.
Source	Medical records.
Measurement level	Patient.
Rationale	A comprehensive assessment provides information on the individual characteristics (e.g., physical status, nutrition, medical/social history, values, care goals, pain, functional capacity, health related quality of life [HRQoL], self-care skills, and ability to adhere to prevention/treatment plan) and availability of resources and support that impact on the health status of the individual and their ability to heal. This underpins development of an individualized treatment plan that meets the goals of the individual.
Evidence rationale	See <i>Recommendation 10.1</i> .

QI 13: Pressure injuries are assessed and the findings are documented at least once a week to monitor progress toward healing.

Description	The percentage of individuals with a pressure injury who have a documented wound assessment in their record at least once a week.
Numerator	The number of individuals with a pressure injury who have a documented wound assessment in their record at least once a week.
Denominator	The number of individuals with a pressure injury.
Definition(s)	A pressure injury evaluation consists of: <ul style="list-style-type: none"> • Assessment of pressure injury characteristics as outlined in <i>Recommendation 10.4 to 10.6</i> • A uniform and consistent method of measuring pressure injury size and surface area to facilitate meaningful monitoring of the pressure injury healing over time • The use of a pressure injury assessment tool that has been tested for validity and reliability.
Inclusion criteria	Individuals with pressure injuries.
Source	Medical records.
Measurement level	Patient.
Rationale	Evaluating healing determines whether treatment is yielding the desired clinical outcome. If not, reassess the individual, the pressure injury and the plan of care every two weeks.
Evidence rationale	See <i>Recommendations 10.4 to 10.7</i> .

QI 14: An individualized treatment plan and its goal, is available for each individual with a pressure injury.

Description	The percentage of individuals with a pressure injury for whom an individualized treatment plan and its goal has been documented.
Numerator	The number of individuals with a pressure injury for whom an individualized treatment plan has been documented.
Denominator	The number of individuals with a pressure injury.
Definition(s)	The treatment plan should be developed using a multidisciplinary approach and should include pressure injury treatment, nutrition, pain management, pressure relief and redistribution, and education. Treatment goals should be consistent with patient values and goals.
Inclusion criteria	Individuals with a pressure injury.
Source	Medical records.
Measurement level	Patient.
Rationale	Developing individualized treatment plans allows for evidence-based treatment of the individual and the pressure injury, which supports ongoing evaluation of intervention effectiveness.
Evidence rationale	See <i>Recommendation 10.2</i> .

QI 15: Every individual with a pressure injury has a documented comprehensive pain assessment and where applicable, a pain management plan.

Description	The percentage of individuals with a pressure injury for whom pain assessment and management plan has been documented.
Numerator	The number of individuals with a pressure injury for whom pain assessment and management plan has been documented.
Denominator	The number of individuals with a pressure injury.
Definition(s)	A pain assessment is conducted using an appropriate valid and reliable scale that considers non-verbal expression of pain. An evidence-based treatment plan is developed and documented for individuals who experience pain.
Inclusion criteria	Individuals with a pressure injury.
Source	Medical records.
Measurement level	Patient.
Rationale	Developing an individualized pain management plan promotes comfort and quality of life.
Evidence rationale	See <i>Recommendations 11.1 to 11.6</i> .

QI 16: Every individual at risk of a pressure injury receives nutritional screening and when applicable, a comprehensive nutritional assessment is conducted, and a nutrition care plan is documented.

Description	The percentage of individuals at increased risk of pressure injuries who receive nutritional screening and if required, a comprehensive nutrition assessment and plan.
Numerator	The number of individuals at increased risk of a pressure injuries who received nutritional screening (and assessment and a treatment plan as required).
Denominator	Number of individuals at increased risk of a pressure injury.
Inclusion criteria	Individuals at increased risk of pressure injury.
Source	Medical records.
Measurement level	Patient.
Rationale	Nutritional screening for those at risk of pressure injuries results in faster identification of individual with or at risk of malnutrition who require comprehensive assessment. Nutritional assessment is associated with more rapid implementation of nutritional interventions and reduction in pressure injury rates/increased pressure injury healing.
Evidence rationale	See <i>Nutrition in Pressure Injury Prevention and Treatment</i> section of the guideline.

QI 17: Every individual with, or at risk of pressure injuries (and/or their informal caregiver) receives information about the prevention and treatment of pressure injuries, self-care skills training and psychosocial support.

Description	The percentage of individuals at increased risk of pressure injuries (and/or caregivers) who received information on preventing and treating pressure injury, self-care skills training, and psychosocial support.
Numerator	The number of individuals at increased risk of a pressure injuries (and/or caregivers) who received information on preventing and treating pressure injury, self-care skills training, and psychosocial support.
Denominator	Number of individuals at increased risk of a pressure injury.
Inclusion criteria	Individuals at increased risk of pressure injury.
Source	Medical records and/or patient consultation.
Measurement level	Patient.
Rationale	Providing individuals with or at risk of a pressure injury with access to evidence-based education increases knowledge and skills; motivation to engage in self-care; and promotes the likelihood that appropriate care will be provided. Education delivery may be varied (verbal, printed or electronic formats, duration and intensity). Patients and their informal caregivers place a high priority on education and consider a range of pressure injury related topics to be relevant to their education needs.
Evidence rationale	See <i>Recommendation 22.2</i> .

QI 18: Measurement of pressure injury rates is regularly conducted and reported to stakeholders.

Description	A rigorous and consistent method for measurement of pressure injury rates is regularly conducted.
Numerator	The number of data collection surveys.
Denominator	The number of months in a year.
Definition(s)	Prevalence and incidence studies should clearly report their methodological design. A rigorous and standardized methodology should be used to allow risk adjustment and benchmarking. The results should be communicated to all stakeholders using feedback loops. The method and frequency of pressure injury monitoring and reporting used within an organization may vary. Event reporting systems and electronic health record-based dashboards can provide more immediate feedback. Some evaluation of pressure injury rates is encouraged on at least a monthly basis.
Exclusion criteria	N/A
Source	Internal and external quality reporting mechanisms (i.e., facility or national benchmarking data). Facility communication records.
Measurement level	Organization.
Rationale	Pressure injury prevalence and incidence studies provide valuable data to drive quality improvement, policy decisions on a national level and research agendas on an international scale.
Evidence rationale	See <i>Recommendations 19.1 and 20.12</i> .

Outcome Quality Indicators

QI 19: Percentage of individuals within the facility at a specific point in time with a pressure injury (point prevalence).

Description	The percentage of individuals with a pressure injury.
Numerator	The number of individuals at a specific point in time with a pressure injury.
Denominator	The number of individuals in the facility at the specific point in time.
Definition(s)	See the guideline chapter <i>Classification of Pressure Injuries</i> for definitions of Categories/Stages of pressure injuries.
Exclusion criteria	Exclusions should be clearly reported (e.g., specific departments, such as outpatients or short-stay surgery, individuals on leave from the facility at the time of audit).
Source	Assessment of individuals using a consistent classification system.
Measurement level	Patient.
Rationale	The prevalence of pressure injuries gives a general indication of the effectiveness of preventive and treatment strategies for pressure injuries and an estimate of resources needed to address pressure injury treatment.
Evidence rationale	See <i>Recommendation 19.1</i> and the guideline chapter <i>Classification of Pressure Injuries</i> . Refer to the guideline chapter <i>Measuring Pressure Injury Prevalence and Incidence</i> for additional methodology considerations.

QI 20: Percentage of individuals who did not have a pressure injury on admission who acquire a pressure injury during their stay in the facility (facility-acquired rate).

Description	Percentage of individuals who did not have a pressure injury on admission who acquire a pressure injury during their stay in the facility.
Numerator	The number of individuals who did not have a pressure injury on admission who acquire a pressure injury during their stay in the facility.
Denominator	The number of individuals who did not have a pressure injury on admission.
Definition(s)	See the guideline chapter <i>Classification of Pressure Injuries</i> for definitions of Categories/ Stages of pressure injuries.
Exclusion criteria	Exclusions should be clearly reported (e.g., specific departments).
Source	Assessment of individuals using a consistent pressure injury classification system. Clinical audit provides a more reliable indication of facility-acquired pressure injury rates than review of medical records.
Measurement level	Patient.
Rationale	Facility-acquired rates provide a clearer indication of the effectiveness of the preventive measures used for pressure injuries.
Evidence rationale	See <i>Recommendation 19.1</i> and the guideline chapter <i>Classification of Pressure Injuries</i> . Refer to the guideline chapter <i>Measuring Pressure Injury Prevalence and Incidence</i> for additional methodology considerations.

References

1. Kotter T, Blozik E, Scherer M. Methods for the guideline-based development of quality indicators--a systematic review. *Implement Sci*, 2012; 7: 21.
2. Becker M, Breuing J, Nothacker M, Deckert S, Steudtner M, Schmitt J, Neugebauer E, Pieper D. Guideline-based quality indicators—a systematic comparison of German and international clinical practice guidelines: protocol for a systematic review. *Systematic Reviews*, 2018; 7(5): DOI 10.1186/s13643-017-0669-2.
3. Donabedian A. The quality of care. How can it be assessed? *Journal of the American Medical Association*, 1988; 26(12): 1743-1748.
4. Mainz J. Defining and classifying clinical indicators for quality improvement. *International Journal for Quality in Health Care*, 2003; 15(6): 523-530.

FURTHER RESEARCH NEEDS

Introduction

The literature search underpinning the development of this guideline highlighted the paucity of research investigating the prevention and treatment of pressure injuries using study designs that are at low risk of bias. Most of the research appraised in this guideline was at a moderate to high risk of bias. The Guideline Governance Group recommends that future research in the area of pressure injuries prioritize the areas listed below.

Priorities for Future Research

This list outlines priorities for future research, but it is not a prioritized list.

Research Methods

- Standardized approaches to measure and report prevalence and incidence of pressure injuries should be adopted in order to facilitate national and international benchmarking. See the guideline chapter *Measuring Pressure Injury Prevalence and Incidence* for recommendations on approaches to measuring and reporting pressure injury rates. Standardized approaches to measure and report pressure injury healing should be adopted.
- Consistent implementation of study designs and processes that are at low risk of bias are required for intervention trials, including:
 - Correctly powered trials.
 - Use of true randomization, wherever appropriate and possible.
 - Concealed allocation to study groups.
 - Blinding of the following, wherever possible: participants, outcome assessors and data analysts to study groups and/or outcome measurement.
 - Use of intention-to-treat analysis.

Etiology of Pressure Injuries

- The concept of skin failure versus pressure injury requires further investigation.
- An increasing body of evidence suggests that the microclimate at the individual's skin and the support surface plays a role in the development of pressure injuries. The importance of these issues and the characteristics of an optimal microclimate continue to require further research.
- Although there is some evidence that high shear forces at the skin surface cause superficial injuries and high pressure is responsible for wounds in deeper tissue layers, further investigation into the precise mechanisms by which damage occurs when skin is loaded is required.
- There is a substantial knowledge base regarding skeletal muscle and skin; however, there is a paucity of knowledge regarding the mechanical properties and damage thresholds of adipose tissue. This area continues to be one for further research.
- The role of the occlusion of lymph vessels, its relationship to tissue edema and influence on pressure injury development is an area that warrants future research.
- Further guidance on concerns over the potentially unavoidable nature of some pressure injuries is strongly recommended.

Risk Assessment and Early Detection

- As sensor technology has become more sophisticated, cost effective and easy to use, and the ability to integrate this technology into clothing, wound dressings and linen has developed, it is worthwhile investigating new opportunities for risk assessment, early detection and screening of individuals.
- As technology is becoming more accessible, there is a necessity to explore which biophysical and biochemical markers are the target markers for this risk screening. Fundamental research is needed using *in vitro* and *in vivo* model systems as well as pre-clinical studies with human volunteers. This area has progressed since the 2014 guideline, but future exploration is warranted.
- The role of the following factors as a risk for pressure injuries is under-explored in the current evidence base: having an existing pressure injury, presence of moisture, deficits of oxygenation and perfusion, impaired sensory perception, age, laboratory blood tests and nutritional status.

- The role of pain as a risk factor or early indicator for pressure injury development warrants future research. In consensus studies, 76.92% of Australian participating experts¹ and 76.92% of European participating experts² rated pain as a prognostic factor for pressure injuries as a high priority for future research.
- There is minimal good quality research investigating the effectiveness of formal risk assessment tools in identifying individuals at risk of pressure injuries. Investigation into, and development of, risk screening and assessment tools that incorporate newer research on factors that are associated with increased pressure injury risk is warranted.
- There is a paucity of high quality evidence on pressure injuries occurring during surgery. More research is needed on frequency of pressure injury incidence and risk factors (e.g., specific diagnoses, length of surgery and positioning) during surgery.
- From the perspective of pressure injury experts participating in a consensus study, 92.31% of the Australia participants¹ and 69.23% of European participants² rated a meta-analysis of studies investigating risk factors in adult sub-populations (e.g. individuals in operating room or neonates) as a priority for pressure injury research.

Skin and Tissue Assessment

- Although there have been recent advances on the role of sub-epidermal moisture (SEM) measurement in assessing the skin and tissues, more research is needed in this area. Research on the differing threshold that might apply to SEM measurements taken at different anatomical locations would assist clinicians in interpreting SEM measurement results.³ High quality studies with larger sample sizes are still required to establish the psychometric properties of SEM measurement devices and to investigate potential impact of comorbidities that influence edema and/or inflammation (e.g., cardiac failure).³
- Given the potential benefits of being able to more accurately identify individuals at high risk of pressure injuries (and with early tissue injury) and adapt their management accordingly, research on methods to assess skin temperature and thermography are a high priority. More research is needed in larger samples that include younger participants and individuals with dark skin, and research evaluating interrater reliability and validity of different methods is needed. Research on assessing skin and tissue for pressure injury risk in neonates is very limited and could be explored more comprehensively, including the use of different assessment devices as noted above. From the perspective of pressure injury experts participating in a consensus study, 69.23% of Australian participants¹ and 100% of European participants² rated strategies to assess the skin and tissues as a research priority.

Preventive Care

- The body of research on the role of skin hygiene and continence management in preventing pressure injuries is small and is a future research priority. More research is needed in larger samples, and exploration of interventions for younger participants (e.g., neonates and children) is warranted.
- The body of research on the role of low co-efficient fabrics in preventing pressure injuries is small and is a future research priority.
- There is a need for further good quality research studies on the use of prophylactic dressings, including comparative studies between dressing types and exploration on the use of prophylactic dressings in special populations (e.g., neonates). In consensus studies with pressure injury experts, 69.23% of participants from Australia¹ and 53.85% of participants from Europe² rated comparisons between different types of prophylactic dressings to be a research priority. Additionally, 84.62% of Australian participants¹ and 61.54% of participants from Europe² considered the efficacy and cost-effectiveness of using prophylactic dressing in populations and clinical settings other than critical care as being a high research priority. However, since these consensus studies were conducted, some evidence on use of prophylactic dressings in aged care has become available. Research on the use of prophylactic dressings in the operating room, with neonates and in community settings remains limited.

Nutrition

- There is minimal research on the effectiveness of conducting nutritional screening in reducing pressure injuries. Direct relationships are difficult to establish as screening leads to implementation of nutritional interventions. Few studies have measured the psychometric properties of nutritional screening tools in individuals with or at risk of pressure injuries, except in older adults. A focus on nutrition screening in neonates and children is warranted.
- There is currently a paucity of research on the role of energy and protein supplementation in prevention of pressure injuries. Further research could investigate the use of supplementation in populations considered at high risk of pressure injuries. In consensus studies, 53.85% of pressure injury expert participants in Australia¹ and Europe² rated efficacy of high calorie and protein intake in preventing pressure injuries as an area of research with high priority.

- The role of multivitamin and arginine supplementation in pressure injury prevention and healing requires further research. In consensus studies, almost 70% of participating pressure injury experts in both Australia¹ and Europe² considered the role of other nutritional supplements (e.g., arginine) in preventing pressure injuries or promoting healing as being a research priority.
- Nutritional requirements for preventing pressure injuries in neonates and children has not been explored in scientific studies. This area has been rated as a high research priority by 46.15% of Australian pressure injury participants¹ and 76.92% of experts in Europe² who participated in consensus studies. Research could focus on establishing the efficacy of nutritional interventions and providing guidance on optimal nutritional regimens to promote chronic wound healing in neonates and children.
- Nutritional screening, assessment and treatment for individuals with obesity who are at risk of, or who have a pressure injury has not been explored. Future research in this area is a high priority.

Repositioning and Early Mobilization

- A fundamental way in which health professionals contribute to the prevention and treatment of pressure injuries is by repositioning those individuals who are unable to reposition independently. Although repositioning is a practice with good validity, there are a limited number of clinical trials that have examined its effect. It is important to undertake robust research investigating the most effective repositioning regimens, in combination with the various pressure redistribution support surfaces commonly used in clinical practice today. These studies should consider specific patient populations (e.g., older adults and neonates) and should report outcome measures of significance for patient consumers (e.g., sleep, pain and comfort) alongside pressure injury incidence and healing. In consensus studies, 61.54% of pressure injury expert participants in Australia¹ and 76.92% of pressure injury expert participants in Europe² rated effectiveness of repositioning regimens in all populations (e.g., acute care, older adults and neonates) as an area of research with high priority.
- Although there is a high incidence and prevalence of heel pressure injuries, they have been the direct focus of very few studies. Research is needed to examine whether heel elevation strategies are more effective when matched to a specific individual's characteristics (e.g., duration and level of immobility and frequency and force of leg movement). In consensus studies, 76.92% of participating pressure injury experts in Australia¹ and in Europe² considered studies exploring heel pressure off-loading and shear reduction strategies to be a research priority.
- Strategies for relieving pressure while seated are an area that has received very minimal research, particularly in older, immobile adults for whom pressure relief maneuvers may be more difficult to perform. In consensus studies, 53.8% of pressure injury expert participants in Australia¹ and 84.62% of pressure injury expert participants in Europe² rated optimal pressure relief lift protocols as an area of research with high priority.
- It is well-established that mobile individuals are at low risk of pressure injuries and generally will not require repositioning assistance. Future research is required on accurate methods to assess mobility in the context of pressure injury risk.

Support Surfaces

- Research on the effectiveness of pressure redistribution surfaces for individuals with obesity with, or at risk of pressure injuries is required. In consensus studies, pressure injury experts in Australia¹ and Europe² rated strategies to reduce pressure injuries in individuals with obesity as an area of research with high priority. In an international consensus process, 86% of participants rated efficacy of support surfaces as a top priority for wound research.⁴
- There is little evidence on pressure injury prevention in the seated population. In consensus studies, 46.15% of pressure injury expert participants in Australia¹ and 84.62% of pressure injury expert participants in Europe² rated effectiveness of various seating selections (chairs and cushions) in individuals with spinal cord injury as a priority for future pressure injury research.
- Further robust studies should be undertaken to evaluate use of natural sheepskin as a support surface in pressure injury prevention.

Device Related Pressure Injuries

- Research on preventing device related pressure injuries is required. In consensus studies, 76.92% of pressure injury expert participants in Australia¹ and 76.92% of pressure injury expert participants in Europe² rated strategies to prevent and treat medical device related pressure injuries (MDRPIs) as an area of research with high priority. There is limited high quality evidence on qualities of medical devices that might influence pressure injury rates, or considerations to make when selecting medical devices to reduce pressure injury rates. Evidence on specific

qualities of medical devices that are associated with medical device related pressure injuries could inform the future design of medical devices.

Pain Management

- A solid evidence base on the experience of pain on the individual with the pressure injury has been documented. Further research on the most effective pharmacological and non-pharmacological strategies to manage pain associated with pressure injuries is required.
- Research on patient consumer care goals identified that pain management is a care goal for 46.5% of individuals with or at risk of a pressure injury, and for 35.9% of their informal caregivers.⁵ Strategies to ensure health professionals address pain associated with pressure injuries are an imperative.

Biophysical Agents

- The role of hyperbaric oxygen therapy in healing pressure injuries has had minimal exploration using robust research designs and appropriate outcome measures. Research on a potential role of this therapy is suggested.
- The effectiveness of phototherapy and laser therapy, and the selection of individuals for whom these therapies are likely to have a clinical effective outcome require further investigation using robust study designs and appropriate outcome measures.
- Although recent research has commenced exploration of the topic, further research on the role of electrical stimulation of the muscles of individuals with spinal cord injury in order to create involuntary movement that relieves pressure and alters the loading characteristics of muscle is warranted based on the pilot trials reported in this guideline.

Promoting Healing and Managing Infection and Biofilms

- The impact of antibiotic resistant bacteria on the healing of chronic wounds, and strategies to reduce the unnecessary use of antibacterial treatments should be prioritized. This includes the role of topical agents in managing pressure injuries.
- Minimal robust research has been conducted on best practice for preventing, diagnosing and eradicating biofilm in pressure injuries and other chronic wounds and this is a priority for ongoing research. Recent advances in this area are promising and will inform clinical practice.
- The role of traditional treatments and medicines (e.g., those used by Indigenous groups and Eastern and African cultures) is under represented in the literature on pressure injuries. There is a strong need for robust research on traditional interventions.

Education and Engagement

- Further research on effective strategies to educate health professionals in pressure injury risk and prevention, especially in settings in which this clinical area is not a primary focus (e.g., patient transportation services and emergency departments) is important. In consensus studies, 69.23% of Australian pressure injury expert participants,¹ and 61.54% of European pressure injury expert participants² rated frequency and content of staff education for sustained knowledge and best practice as a research priority. Further well-designed studies are required to determine optimal education program design and delivery.
- Further research on educating patient consumers and their informal caregivers, and actively engaging them in care is warranted. There is limited evidence on strategies to promote self-care skills, and to engage patient consumers in preventive care in the in-patient setting. Evidence in this area should focus on different strategies that could be implemented in specific populations (e.g. specific clinical settings and specific patient populations). In consensus studies 76.92% of participating Australian pressure injury experts¹ and 61.54% of participating European pressure injury experts² rated research on the frequency and content of consumer education for sustained knowledge and best practice in pressure injury prevention and treatment as a priority for pressure injury research.

References

1. Haesler E, Carville K, Haesler P. Priority issues for pressure injury research: An Australian consensus study. *Res Nurs Health*, 2018; 08: 08.
2. Haesler E, Carville K, Haesler P. Consensus priorities for pressure ulcer research in Europe, in 5th World Congress of the World Union of Wound Healing Societies. 2016: Florence.
3. Guihan M, Jenson B, Chun S, Parachuri R, A.S. C, McCreath HE. Assessing the feasibility of subepidermal moisture to predict erythema and stage 1 pressure ulcers in persons with spinal cord injury: A pilot study. *Journal of Spinal Cord Medicine*, , 2012; 35(1): 46-52.
4. Cowman S, Gethin G, Clarke E, Moore Z, Craig G, Jordan-O'Brien J, McLain N, Strapp H. An international eDelphi study identifying the research and education priorities in wound management and tissue repair. *J Clin Nurs*, 2011; 21: 344-353.
5. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis.

GLOSSARY OF TERMS

Abrasion: A loss of the epidermis through some mechanical process, such as friction or trauma.

Abscess: A localized collection of pus surrounded by inflamed tissue, usually due to an infective process.¹

Active support surface: A powered support surface, with the capability to change its load distribution properties, with or without applied load.²

Acute wound: A surgical or traumatic wound that heals by primary or secondary intention and which proceeds through an orderly and timely reparative process that results in sustained restoration of anatomical integrity.³

Adjunct therapy: A substance (or therapy) that aids or heightens the action of another therapy, thereby maximizing prevention or treatment.

Adjuvant therapy: See adjunct therapy.

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.

Alginate wound dressing: A highly absorbent, biodegradable dressing derived from non-woven absorptive material manufactured from seaweed. They are available in sheet and rope form.¹

Anasarca: Diffuse, systemic edema arising from an accumulation of fluid in the interstitial space. Anasarca often occurs in congestive heart failure, liver failure, or renal disease.

Angiogenesis: The process of developing new blood vessels from pre-existing blood vessels within the wound space; an integral part of wound healing.⁴

Antibacterial: A term used to encompass antibiotics, antiseptics and disinfectants. A substance that inhibits the growth of, or eradicates bacteria.⁵⁻⁷

Antibiotic: A natural or synthetic substance administered systemically or topically that has the capacity to destroy or inhibit bacterial growth.^{5,6,8}

Antimicrobial: A substance that acts directly on a microorganism to destroy bacteria, fungi, spores or viruses and prevents the development of new growth.⁹ An antimicrobial is a broad term that includes: antiseptics, disinfectants, antibiotics and antifungals.

Antiseptic: A substance that kills microorganisms.¹⁰

Artificial nutrition: Artificial nutrition is a medical intervention through which nutrition and hydration is delivered by a route other than oral (i.e., enterally or parenterally) when an individual is unable to take nutrition and hydration by mouth. Enteral nutrition options include nasogastric tube, nasogastrojejunal tube, percutaneous endoscopic gastrostomy (PEG) and jejunostomy (PEGJ), as well as other surgical options. Parenteral nutrition option involves feeding via a peripheral intravenous line or central venous line. Both parenteral and enteral nutrition require the consent of the individual or his or her family.¹¹⁻¹³

Aseptic technique: A wound care technique designed to prevent introduction of new microorganisms into the wound, and to reduce cross infection risk and uses sterile products and devices.

Surgical aseptic technique: required for complex/longer wound procedures (i.e. longer than 20 minutes) involving larger open wounds, multiple wounds, or wounds without entirely visible wound beds. Sterile gloves, non-touch technique and a critical sterile field are used.^{7,14}

Standard aseptic/clean technique: used for simple wound dressing procedures that are shorter in duration (less than 20 minutes) and involve few key sites or key parts. Non-sterile gloves can be used, and a non-touch technique and general aseptic field are used.^{7,14}

Autolysis: see *Debridement*.

Avascular: having few or no blood vessels.

Bacterial bioburden: The quantity of microorganisms present (e.g., planktonic bacteria or biofilm). It can be categorized as:

Contamination: The presence of non-proliferating microbes on the wound surface⁹ and with no impairment to health or obvious clinical signs of infection.^{6,15}

Colonization: The replication of microorganisms on the surface of the wound without invasion into wound tissue and without host immune response.¹⁶ The microbial growth does not impede wound healing.⁶

Local infection: The presence of bacteria or other microorganisms in sufficient quantity to damage tissue and/or impair healing. The classic (overt) signs and symptoms of infection include purulent exudates, malodor, erythema, local warmth, tenderness/pain, edema, and elevated white blood count. Covert signs may also provide early indicators of local infection including hypergranulation, bleeding friable granulation, epithelial bridging, new or increasing wound pain, increasing malodor and delayed wound healing.⁶

Spreading infection: Microbial growth invades the tissue surrounding the wound. Signs include induration with or without erythema, crepitus, wound dehiscence, lymph gland swelling and malaise.⁶

Systemic infection: Infection invades the bloodstream and other organs leading to severe sepsis, organ failure, septic shock and death.⁶

Barrier film/cream/ointment: Substance or leave-on product used as a protective layer (barrier) to prevent skin irritation.

Biofilm: Biofilm is an aggregate structure of microbes with genetic diversity that creates behaviours and defences to produce unique chronic infection. Biofilms are characterised by significant tolerance to antibiotics and biocides whilst remaining protected from host immunity.⁶ Biofilms play an important role in maintaining a chronic inflammation state ultimately leading to the failure to heal of skin wounds.¹⁷ Also see *Bacterial bioburden*.

Biophysical agent: An agent used to deliver a specific treatment substance to a wound, e.g., oxygen, negative pressure wound therapy, pulsatile lavage with suction, electrical stimulation or whirlpool, among many others.

Blanchable erythema: see *Erythema*.

Body configuration: The general form and appearance of the individual's body and body parts. Body configuration can be influenced by any factor that influences ability to maintain posture and balance, including but not limited to contractures, spasticity, deformities, amputation and paralysis.

Body mass index (BMI): Defined as an individual's weight in kilograms divided by the square of his height in meters.

Bolster pad: A pad used as a support.

Bony prominence: A bony elevation or projection on an anatomical structure.¹

Bottoming out: The state of support surface deformation beyond critical immersion whereby effective pressure redistribution is lost.²

Bridging: The presence of strands of tissue across the ulcer bed.

Cadexomer iodine dressing: A dressing consisting of spherical hydrophilic beads of cadexomer-starch that contain iodine. It is highly absorbent and releases iodine slowly in the wound area. Cadexomer iodine is also available as a topical cream.

Callus: Reactive hyperkeratosis, usually due to friction and/or pressure, leading to enhanced skin markings.¹⁸

Cellulitis (regional infection, spreading infection): Diffuse, spreading infections of skin and soft tissues by a range of bacterial organisms, most commonly Beta-hemolytic Streptococci and Staphylococcus aureus. The clinical presentation is dependent not only on the organism but also on the way it invades the tissues.¹⁹

Chronic wound: A wound with slow progression through the healing phase; or delayed, interrupted or stalled healing due to intrinsic and extrinsic factors.⁶

Clean technique: A wound care technique that is designed to minimize the number of organisms introduced to a wound and to reduce the risk of cross infection.²⁰ Wound cleaning is performed using clean, potable water with either clean or sterile products (depending on local protocols). As most chronic wounds have some level of bacterial colonization, clean technique is appropriate for most pressure injuries if the host is not compromised or the wound does not enter a sterile organ or joint.

Clinical judgment: An overarching concept integrating all reasoning tasks and actions performed by health professionals to describe and assess a health condition of interest.²¹ It describes the sum of cognitive actions carried out by health professionals to interpret and synthesize information to derive a diagnosis and management plan for an individual.

Coefficient of friction: A measurement of the amount of friction existing between two surfaces.²

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.

Cohen's kappa (κ): interrater or interrater reliability coefficient.

Collagen matrix wound dressing: A dressing manufactured from bovine, porcine, or avian collagen that has been shown to reduce the levels of proteases in chronic wounds. It is available in sheets and pads, and as particles and gels.

Composite wound dressing: A dressing that is a combination of two or more types of dressing.

Confidence interval (CI): for a given statistic calculated for a sample of observations (e.g., the mean), the confidence interval is a range of values around that statistic that are believed to contain, with a certain probability (e.g. 95%), the true value of that statistic (i.e., the population value).²²

Contraction: Pulling together wound edges in the healing process.

Contour seating: A seating product that increases contact area with the body by providing a contour that resembles the typical human form.

Cover dressing: Dressing used as the top layer to cover other absorbent dressings.

Crepitus: A cracking, crunchy, or popping sensation upon palpation of soft tissue related to underlying gas in the tissue released by anaerobes; indicative of the presence of air bubbles in the tissue.

Culture: A laboratory test involving the growth of bacteria or other cells in a special growth medium. Cultures are grown to identify an organism as well as which antibiotics are effective in combating the organism(s).

Cytokine: see *Proinflammatory cytokines*

Cytotoxic: A substance that damages or kills living cells.

Dead space: An area of tissue loss in a cavity or tract.

Debridement: The removal of devitalized (non-viable) tissue from or adjacent to a wound.⁵ The process effaces the wound bed of exudates, detaches bacterial colonies, and allows a stimulatory environment to be established.

Autolytic debridement (autolysis): A highly selective form of slow debridement that occurs naturally in wounds²³ and is promoted the use of moisture-retentive dressings.²⁴

Biological debridement (larval therapy): The use of sterile fly larvae to remove devitalized tissue. Larvae are believed to secrete a proteinase enzyme that degrades necrotic tissue, digests bacteria, and stimulates granulation tissue.²⁵

Conservative sharp debridement: The removal of devitalized tissue using a sharp instrument (e.g., scalpel, scissors or curette) without pain or bleeding.¹

Enzymatic debridement: The removal of devitalized tissue by applying exogenous proteolytic or fibrinolytic enzymes.²⁴

Maintenance debridement: Repeated debridement until devitalized (non-viable) tissue is removed from the wound bed.

Mechanical debridement: Non-selective removal of devitalized tissue by physical forces.²⁴

Surgical/sharp debridement: rapid wound debridement in which devitalized tissue is removed from the wound using scalpel and/or scissors under general or local topical anesthetic.

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

Denuded: Loss of epidermis.

Desiccation: The drying of the wound bed.

Devitalized tissue: Tissue that is devoid of vitality or life (non-viable). It is normally moist, yellow, green, tan, or gray and may become thick and leathery with dry black or brown eschar.

Dialkylcarbamoyl chloride impregnated (DACC) dressings: employs the principles that some pathogenic microorganisms are hydrophobic and will bind to a hydrophobic acetate or cotton dressing surface coated with DACC.⁶

Direct current: A term describing a feature of electrical stimulation, direct current is the continuous, unidirectional flow of charged particles.

Electrical stimulation: The use of an electrical current to transfer energy controlled by an electrical source. In the prevention and treatment of pressure injuries, electrical stimulation is used as a wound healing therapy and is emerging as a therapy to stimulate muscles in individuals who are unable to reposition.

Electromagnetic spectrum (EMS): is an energy source that affects living systems. The EMS comprises infrared (thermal radiation), ultraviolet light (invisible light), laser (coherent and monochromatic light) and electrical/electromagnetic stimulation.

Emollient: Cosmetic ingredient remaining on and in the skin surface to retain water within the stratum corneum.

Enhanced food: see *Fortified food*.

Enteral nutrition: Nutritional support given via a nasogastric, naso-enteral, or percutaneous tube. Enteral nutrition is used when the gastrointestinal tract is functioning.²⁶

Envelopment: The ability of a support surface to conform, so to fit or mold around irregularities in the body.²

Epibole: A condition that exists when the edges of the top layers of epidermis have rolled down and healing stops.¹

Epidermis: The outermost layer of skin.

Epithelialization: The process of becoming covered with or converted to epithelium.

Eschar: Black or brown necrotic, devitalized tissue. The tissue can be loose or firmly adherent and hard, soft, or somewhat soggy.¹

Erythema: Redness of the skin due to dilation of blood vessels.¹

Blanchable erythema: An area of reddened skin that temporarily turns white or pale when light pressure is applied to the skin and reddens when pressure is relieved. Over a pressure site, this is due to a normal hyperemic response.²⁷

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

Excoriation: A loss of the epidermis and a portion of the dermis due to scratching or an exogenous injury.¹⁸

Extrinsic factors: Originating outside of the body.

Exudate: Fluid extruded from a tissue or capillaries that can include fluid, cells, or cellular debris that has escaped from blood vessels and been deposited in tissue surfaces. It may contain serum, cellular debris, bacteria, and leukocytes.^{1,28}

Fascia: A sheet or band of fibrous tissue that lies deep below the skin or encloses muscles and various organs of the body.

Fatigue (of a support surface): The reduced capacity of a surface or its components to perform as specified. This change may be the result of intended or unintended use and/or prolonged exposure to chemical, thermal, or physical forces.²

Fiber methylcellulose wound dressing: Highly absorbent dressing, chemically similar to a hydrocolloid.

Fibroblast: The cells from which connective tissue develops. Fibroblasts proliferate in the deeper parts of a wound and begin synthesizing small amounts of collagen, which serves as a scaffold for migration of cells and further fibroblast proliferation.¹

Filler dressing: Dressing material used to fill dead space in a wound bed.

Fistula: An abnormal passage from an internal organ to the body surface or between two internal organs.¹

Flap: A flap is a surgical relocation of tissue from one part of the body to another part in order to reconstruct a primary defect. Flaps may be skin flaps, cutaneous flaps or composite flaps. The flap is often cut and rotated to a neighboring site.

Float: A method used to off-load a body part, such as the heel, of pressure.

Foam wound dressing: A sponge-like polymer dressing that may be impregnated or coated with other materials and has some absorptive properties. Simple foams wick drainage from the wound bed and move it to the surface of the dressing. Complex polyurethane foam dressings absorb the fluid, move it throughout the dressing, and retain it. Foam dressings also allow fluid to evaporate.

Fortified foods: Normal food enriched with specific nutrients, in particular with energy and /or protein, minerals, vitamins, or trace elements.

Frequent small shifts: Frequent shifts in the position of the individual, which may be only 10° to 15° at a time; a procedure used to reposition an individual who may be hemodynamically unstable.

Friable: Fragile, easily injured, characteristic of newly healed tissue.

Friction (frictional force): The resistance to motion in a parallel direction relative to the common boundary of two surfaces.²

Friction blister: A blister within the skin caused by repeated friction.

Full thickness skin loss: Ulceration that extends through the dermis to involve the subcutaneous tissue (Category/Stage III and IV pressure injuries) and, if a Category/Stage IV pressure injury, extends into the muscle and possibly down to the bone.

Functionality: This refers to the intended, proper use for which the product was designed.

Functional life span: The designated time period for which a medical device such as support surfaces were designed and intended to fulfill its original function.

Gauze: A woven dressing, usually made from cotton or synthetic material, that is absorptive and permeable to water, water vapor, and oxygen. Gauze can be impregnated with petrolatum, antiseptics, or other agents.¹

Gelling fiber dressing: Carboxymethyl cellulose or polyvinyl alcohol sun into fibers and manufactured into sheet or ribbon packing dressings.²⁹

Granulation tissue: The pink/red, moist, shiny tissue that glistens and is composed of new blood vessels, connective tissue, fibroblasts, and inflammatory cells that fills an open wound when it begins to heal. It typically appears deep pink or red with an irregular, granular surface.¹

Growth factors: Naturally occurring proteins or hormones that stimulate cell growth.

Health related quality of life: Health related quality of life (HRQoL) is an individual's perceived physical or mental health.³⁰

Hematoma: Circumscribed, usually palpable hemorrhage into the skin or soft tissues.¹⁸

Hemorrhage: Bleeding (may be internal or external).

High specification reactive foam mattress: A high specification reactive foam mattress is one with superior qualities of immersion, envelopment and microclimate control. Characteristics of reactive foam mattresses that relate to the qualities of immersion, envelopment and microclimate control include foam type, density, hardness, support factor, thickness and water vapor permeability. Studies reporting on the effectiveness of reactive foam mattresses generally provide a limited description of the characteristics of support surfaces used in the research. However, values for these

characteristics that are currently considered to contribute to a mattress being considered “high specification” are discussed in the *Support Surfaces* chapter. Standards and performance measures for reactive foam mattresses continue to evolve and provide ongoing insight into selection of support surfaces.

Honey impregnated wound dressing: A dressing that produces hydrogen peroxide, contains antioxidants, and releases anti-inflammatory products. Odor is reduced because the honey produces an alternative product for bacterial metabolism that yields lactic acid rather than ammonia, amines, and sulfur, which are odorous. Honey must be of medical-grade.

Host response: The reaction of the individual to the invasion of the microorganism.

Hydrocolloid wound dressing: A flexible dressing containing gel-forming agents, such as sodium carboxymethylcellulose (NaCMC), pectin and gelatin. In many products, these are combined with elastomers and adhesives and applied to a carrier (usually polyurethane foam or film) to form an absorbent, self-adhesive, waterproof wafer.¹

Hydrogel wound dressing: A water-based, non-adherent gel that contains hydrated hydrophilic polymers, which produce a moist environment that improves wound healing. The dressing can absorb excess exudates from exuding wounds but donate moisture to dry, necrotic tissue or slough. The dressing facilitates autolytic debridement.¹

Immersion: Penetration (sinking) into a support surface, measured by depth.²

Incidence: Proportion or rate of occurrence of a given medical condition in a population within a specified period of time.

Incontinence-associated dermatitis (IAD): Irritant contact dermatitis from prolonged contact with urine or feces as a result of incontinence.³¹

Induration: Firm texture in the absence of calcification or bone formation.¹⁸

Infection: The presence of bacteria or other microorganisms in sufficient quantity to damage tissue or impair healing. Clinical signs of infection may not be present in the immunocompromised individual or the individual with a chronic wound. See *Bacterial bioburden*.

Infrared therapy: Treatment using thermal radiation, a phototherapeutic agent that is part of the electromagnetic spectrum.

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.²

Integrated bed system: A bed frame and support surface that are combined into a single unit whereby the surface is unable to function separately.²

Intertrigo: Intertriginous dermatitis (intertrigo) is a form of irritant contact dermatitis of the skin folds (axillary, submammary, genitocrural, abdominal apron) caused by repetitive shearing forces of skin on skin. Sweat, other body fluids, occlusion and obesity all contribute to its development.³²

Intrinsic factors: Originating within the body.

Kaplan-Meier survival curves: A curve plot that is generated in a survival analysis. A survival analysis is used to investigate the amount of time it takes until participants a trial develop a specific clinical outcome or end point (e.g., development of a pressure injury).³³

Laser: Coherent and monochromatic light, a phototherapeutic agent that is part of the electromagnetic spectrum.

Lateral rotation therapy: A feature of a support surface that provides rotation about a longitudinal axis as characterized by degree of patient turn, duration, and frequency.²

Lift (pressure relief lift): The lifting of oneself or the body from a seated surface to temporarily relieve pressure.

Likert Scale: Bipolar adjectival scale in that the descriptors range from none or little of the attribute at one end to a lot or the maximal amount at the other. Used in questionnaires or psychological tests.³⁴

Lipido-colloid dressings: Wound dressing comprised of poly-absorbent fibers and a lipido-colloid matrix creating a gel that aids autolytic debridement or lipido-colloid matrix with nano-oligo saccharide polymers Forms a gel in the presence of exudate and facilitates protease inhibition.²⁹

- Low air loss:** A feature of a support surface that uses a flow of air to assist in managing the heat and humidity (microclimate) of the skin.²
- Maceration of skin:** softening and breaking down of skin resulting from prolonged exposure to moisture.
- Maggot therapy:** see *Debridement*.
- Malnutrition:** Malnutrition defined as any nutritional imbalance³⁵ and is synonymous with the term undernutrition.
- Malodor:** An offensive or disagreeable odor.
- Matrix metalloprotease (MMP):** A cell protein that plays an essential role in wound healing, including contraction of the wound matrix through the use of myofibroblasts, implementation of angiogenesis, cell migration, remodeling of scar extracellular matrix (ECM), and removal of damaged ECM.⁴
- Medical grade honey:** Honey that is filtered, gamma irradiated and produced under exacting standards of hygiene.
- Medical grade sheepskin:** A sheepskin that complies with the Australian Standard AS4480.1-1998.⁵
- Microclimate of skin:** Temperature, humidity and air flow next to the skin surface.³⁶
- Micronutrient:** A micronutrient is a chemical element or substance required in very small amounts for normal growth and development.
- Mobility:** The ability to move oneself from one position to another.
- Multivariable model:** a statistical model that has multiple independent variables that investigates the independent relationships between variables.³⁷
- Multivariate model:** a statistical model that has two or more dependent or outcome variables, often derived from longitudinal studies in which outcomes are measured on the same individual multiple times.³⁷
- Necrosis:** The death of tissue.
- Necrotic tissue:** Tissue that has died, also called devitalized or non-viable tissue.
- Negative-pressure wound therapy (NPWT):** A wound treatment modality that promotes healing through the removal of third space edema, thus enhancing nutrient and oxygen delivery; removal of wound exudates, which is the medium for bacterial colonization; promotion of granulation tissue; promotion of angiogenesis; and removal of wound inhibitory factors.
- Nutritional supplement:** A commercial or other prepared food or beverage that supplements energy, protein, carbohydrate, and/or fiber.
- Odds ratio (OR):** The ratio of one odds to another odds, for example, the ratio of the odds of an event in one group to the odds of an event in another group; an odds ratio of 1.0 indicates no differences between groups.³⁸
- Offload:** To remove pressure from any area.
- Oral nutritional supplement:** A commercial or other prepared food or beverage that supplements nutrient and caloric intake.
- Osteomyelitis:** The inflammation and infection of bone and bone marrow, usually caused by pathogens that enter the bone during an injury or surgery.¹
- Overlay:** An additional support surface designed to be placed directly on top of an existing surface.²
- P value (p):** In statistical testing, the probability that the obtained results are due to chance alone.³⁸
- Palliative care:** Care focused on holistically supporting the individual for comfort rather than cure, or healing of the wound, while enhancing the quality of living and dying.³⁹
- Pannus:** A hanging flap of abdominal tissue in a bariatric individual.
- Parenteral nutrition:** The provision of macronutrients, vitamins, minerals, electrolytes, and fluids via a central or peripheral vein that is indicated when the gastrointestinal tract cannot be used for nutritional support. Total parenteral nutrition (TPN) provides all essential nutrients and is delivered through of central vein.

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

Pearson's r (r): a measure of the strength and direction of the linear relationship between two metric variables (correlation).

Periwound: The area immediately adjacent to the wound edge and extending out as far as the tissue color and consistency changes extend.

pH: A measure on a scale from 0 to 14 of the acidity or alkalinity of an aqueous solution, with 7 being neutral, greater than 7 is more alkaline and less than 7 is more acidic.

Phagocytosis: The process of the ingestion and digestion of bacteria, cells, necrotic tissue, or debris by white blood cells.

Phototherapy: An agent that employs energy waves from the infrared, visible, and ultraviolet region of the electromagnetic spectrum. Combinations of these technologies are often used.⁴⁰

Planktonic bacteria: Free-floating bacteria. Also see *Bacterial bioburden*.

Pocketing: This occurs when granulation tissue does not grow in a uniform manner across the entire wound or when healing does not progress from the bottom up to the top of the wound. Pockets can harbor bacteria.

Potable water: Water that is fit for consumption by humans and animals.

Polyhexamethylene biguanide (PHMB): Also known as polyhexanide and polyaminopropyl biguanide, a cationic polymeric biguanide similar to chlorhexidine that is an antimicrobial agent.²⁹

Polymeric membrane wound dressing: A foam dressing combined with glycerin to soften devitalized tissue in the ulcer and starch to wick away exudates. The dressing also contains a surfactant that loosens necrotic tissue from the wound bed.⁴¹

Pounds per square inch (PSI): A unit of pressure exerted by a stream of fluid against one square inch of skin or wound surface.¹

Prealbumin: A body protein whose function is to transport thyroxine and complexes with retinol-binding protein for Vitamin A transport. The normal level is 15 to 36 mg/dL, but it can vary with the laboratory determining the level.

Pressure: The force per unit area exerted perpendicular to the plane of interest.²

Pressure injury: Localized damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear. Pressure injuries usually occur over a bony prominence but may also be related to a medical device or other object.

Pressure ulcer: see *Pressure injury*

Prevalence: The proportion/percentage of individuals in a defined population who have a medical condition at a specified point in time.

Point pressure injury prevalence: Measures the proportion of a defined population (e.g., individuals in a hospital) who have a pressure injury at a specific moment in time (e.g., on a specific day).⁴²

Period pressure injury prevalence: Measures the proportion of a defined population (e.g., individuals in a hospital) who have a pressure ulcer over a period of time (e.g., over a week).

Pressure point: a point on the surface of the body that is sensitive to pressure (e.g., a bony prominence).

Proinflammatory cytokines: A body substance liberated in the presence of inflammation and infection, e.g., interleukin-1 and tumor necrosis factor, which in turn increases the levels of matrix metalloproteases (MMPs), decreases the level of inhibitors in tissue against the MMPs, and decreases the production of growth factors and fibroblast activity.⁴³ They play a critical role in regulating the integrated hepatic acute-phase protein response.

Prophylactic dressing: A dressing that is placed onto the skin before any skin damage is evident with a goal of preventing skin breakdown due to pressure, shear and alternations in the skin's microclimate. Features such as an elastic adhesive type (e.g. silicone), the number of dressing layers and their construction, and the size of the selected dressing all contribute to its ability to protect the skin.⁴⁴

Protease: A proteolytic enzyme.

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.

Protectant (skin): A substance or product applied externally to the skin to protect it from harmful substances.

Protein-calorie malnutrition: This occurs when both protein and energy intake are insufficient to meet an individual's metabolic demands. The wasting and excessive loss of lean body mass resulting from too little energy being supplied to the body tissue can be reversed solely by the administration of nutrients.⁴⁵

Proteolytic enzyme: An endogenous substance such as collagenase, elastase, myeloperoxidase, acid hydrolase, and lysozymes that selectively liquefies and separates necrotic tissue and eschar from healthy tissue.²³

Pulsatile lavage: The delivery of irrigation fluid in rapid, discrete pulses via a disposable, battery-powered unit that delivers variable irrigation pressures with or without concurrent suction. The pulsation of the irrigation fluid may increase the amount of debris removed. Concurrent suction immediately removes irrigation fluid that has been contaminated by contact with the wound.¹

Pulsed current: A term describing a feature of electrical stimulation, pulsed current is the brief unidirectional (monophasic pulsed current) or bidirectional (biphasic pulsed current) flow of electrons or ions in which each pulse is separated by a period with no current flow.

Pulsed electromagnetic field therapy: The delivery of magnetic field to the wound bed with a goal of delivering therapeutic effect.

Purulent: Containing pus.

Quality indicator: Quality indicators (QIs) are used in assessing the quality of healthcare. Evidence-based clinical practice guidelines (CPGs) are relevant sources for generating QIs.⁴⁶

Quality of life: An individualized, qualitative measure of the impact of disease, treatment, and/or disability on the individual's ability to lead a fulfilling life.⁴⁷

Reactive hyperemia: A reddening of the skin caused by blood rushing back into hypoxic tissue after pressure offloading.

Reactive support surface: A powered or non-powered support surface with the capability to change its load distribution properties only in response to applied load.²

Receiver Operator Curve, Area Under (AUROC): a measure of an overall accuracy of a specific test, with a value approaching 1.0 indicating a high sensitivity and specificity.³⁷

Relative risk (risk ratio): The risk of a particular outcome (e.g., a pressure injury) occurring in the presence of a particular exposure (e.g., a pressure injury prevention program) compared to the risk without the particular exposure. A RR of 1.0 indicates no difference in outcome risk between exposure and non-exposure.

Reepithelialization: The replacement of the epithelial layers of the tissue.

Reposition: A change in position in the lying or seated individual, with the purpose of relieving or redistributing pressure and enhancing comfort, undertaken at regular intervals.

Semi-Fowler position: A position in which the individual is supine and the head of the bed is elevated 30°.

Sensitivity: The proportion of individuals with disease or condition who test positive when undergoing a particular test. Thus, sensitivity indicates how well a particular test detects a specific condition that is actually present.⁴⁸

Sepsis: A life-threatening organ dysfunction caused by a dysregulated host response to infection.⁴⁹

Seroma: A collection of serum/plasma within a wound.

Shear (shear stress): The force per unit area exerted parallel to the perpendicular plane of interest.²

Silicone wound dressing: A dressing composed of silicone, which is chemically inert and, therefore does not chemically interact with the wound. It is insoluble in wound exudates. This dressing provides a wound contact layer that can be removed atraumatically and without pain for the individual.

Silver impregnated wound dressing: A dressing product impregnated with ionic silver for immediate or sustained release of silver into the wound bed. Silver provides a barrier to bacterial penetration.⁵⁰

Silver sulfadiazine: A silver-based, antibacterial agent.

Sinus tract: A course or path of tissue destruction, sometimes called a tunnel, occurring in any direction from the surface or edge of a wound. It results in dead space with a potential for abscess formation. A sinus can be distinguished from undermining in that it involves only a small portion of the wound edge whereas undermining involves a significant portion of the wound edge.¹

Skin integrity: Skin integrity is the combination of an intact cutaneous structure and a functional capacity that is high enough to preserve it.⁵¹

Slough: Soft, moist, devitalized (non-viable) tissue. It may be white, yellow, tan, or green, and it may be loose or firmly adherent.¹

Specialty support surface: Specialty support surfaces are support surfaces that have additional technology features designed to further redistribute pressure, reduce shear and influence the microclimate (e.g., alternating pressure, air fluidized or loss air loss features).

Specificity: The proportion of individuals without disease or condition who test negative when undergoing a particular test. Thus, specificity indicates how well a particular test rules out a specific condition when the condition is not present.⁴⁸

Standard hospital mattress: A term used to describe the standard mattress provided within a facility and generally used as the comparative intervention in research trials investigating the effectiveness of pressure redistribution support surfaces. As such, the qualities of a standard hospital mattress vary according to historical and clinical context and are rarely reported in detail in clinical trials. In most cases it is assumed that a standard hospital mattress is a non-powered foam or spring-based mattress.²

Statistical significance: A term indicating that the results from an analysis of sample data are unlikely to have been the result of chance at a specified level of probability.³⁸

Strain: A measurement of relative deformation.

Stress: Force transferred per unit area.

Support surface: A specialized device for pressure redistribution designed for management of tissue loads, microclimate, and/or other therapeutic functions. Support surfaces include, but are not limited to, mattresses, integrated bed systems, mattress replacements or overlays, or seat cushions and seat cushion overlays.²

Surfactant (surface active agent): A substance which lowers the surface tension of the medium in which it is dissolved, and/or the interfacial tension with other phases, and, accordingly, is positively adsorbed at the liquid/vapour and/or at other interfaces. The term surfactant is also applied to sparingly soluble substances, which lower the surface tension of a liquid by spreading spontaneously over its surface.⁵²

Suspected deep tissue injury: Purple or maroon localized area of discolored, intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, 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.⁵³

Tensile strength: The maximum force or pressure that can be applied to a wound without causing it to break apart.

Tilt-in-space: A positioning of a seat (usually a wheelchair) that changes the orientation of the body while maintaining the positioning of the hip, knee and feet at the angles achieved in usual seated position. This contrasts to a recline position in which the upper torso is angled backward at the hip, leaning the individual backwards.^{54,55}

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

Tissue ischemia: The reduction of oxygen levels to below normal.

Topical antibiotic: See *Antibiotic*.

Transfer aid: Any agent that aids in transferring an individual (e.g. a sheet, mechanical lift).

Transparent film dressing: A transparent dressing that is nonabsorptive and polymer-based, making it permeable to oxygen and water vapor but not to water.¹

Transpire: A process through which moisture passes into the atmosphere as water vapour. Ability to transpire is a characteristic that may be used to describe wound dressing that have a semi-permeable or permeable quality that allows excess moisture to pass into the atmosphere as vapour.

Tunneling: See *Sinus tract*.

Turn: The act of changing position.

Ultrasound: A mechanical vibration (acoustic energy) transmitted in a wave formation at frequencies beyond the upper limit of human hearing. When applied as a therapeutic agent, its vibratory property affects the cells of biologic tissues. Ultrasound can be used to assess and treat soft tissues.

Ultraviolet light therapy: A form of therapy that uses an invisible light that is part of the electromagnetic spectrum and can be used as a phototherapeutic agent.

Undermining: An area of tissue destruction extending under intact skin along the periphery of a wound commonly seen in shear injuries. It can be distinguished from a sinus tract in that it involves a significant portion of wound edge.¹

Undernutrition: see *Malnutrition*.

Unintentional weight loss: Gradual, unintended weight loss over time.

Unstable individual: an individual physiologically unstable requiring treatment with invasive modalities such as mechanical ventilation, vasopressor agents, extracorporeal membrane oxygenation, intra-aortic balloon pump, left ventricular assist device, or continuous renal replacement therapy.⁵⁶

Pressure injury, ungradable: Pressure injury with full thickness skin loss in which actual depth of the wound 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 are removed to expose the base of the wound, it is not possible to determine whether the pressure injury is Category/Stage III or IV.⁵⁷

Viscoelastic foam (memory foam): A type of porous polymer material which conforms in proportion to the applied weight. The material exhibits dampened elastic properties when load is applied.²

Wet-to-dry saline gauze dressing: A technique whereby gauze is moistened with normal saline, applied wet to the wound, and allowed to dry, then removed when adhered to the wound bed. As the dressing is removed, the wound is non-specifically debrided.¹

Whirlpool: A hydrotherapy approach using water with or without additives or saline to stimulate wound healing and to cleanse and debride chronic wounds.

Wound dressing: A material applied to a wound for a variety of reasons, including promotion of healing, protection, absorption and drainage of exudate, control of malodor and minimization of pain.

References

1. Wound Ostomy and Continence Nurses Society (WOCNS), *Wound Ostomy and Continence Nurses Society. Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: Wound Ostomy and Continence Nurses Society.
2. National Pressure Ulcer Advisory Panel. *National Pressure Ulcer Advisory Panel Support Surface Standards Initiative - Terms and Definitions Related to Support Surfaces*. 2018; Available from: http://www.npuap.org/NPUAP_S3I_TD.pdf.
3. Lazarus G, Cooper C, Knighton D, Margolis D, Pecoraro R, Rodeheaver G, Robson M. Definitions and guidelines for assessment of wounds and evaluation of healing. *Arch Dermatol*, 1994; 130: 489-493.
4. Jones V, Harding K, Stechmiller JK, Schultz G, *Acute and Chronic Wound Healing*, in *Wound Care Essentials: Practice Principles*, S. Baranoski and E. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 64-76.
5. Australian Wound Management Association (AWMA), *Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury*. 2012, Osborne Park, WA: Cambridge Media.
6. International Wound Infection Institute (IWII), *Wound Infection in Clinical Practice*. 2016, Wounds International.
7. Wounds Australia, *Standards for Wound Prevention and Management*. 2016, Cambridge Media: Osborne Park, WA.
8. Wound Ostomy and Continence Nurses Society (WOCNS), *Guideline for the Prevention and Management of Pressure Ulcers*. WOCN Clinical Practice Guideline Series. 2010, Mount Laurel, NJ: WOCNS.
9. Gardner SE, Frantz RA, *Wound Bioburden*, in *Wound Care Essentials: Practice Principles*, S. Baranoski and E. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 111-3.

10. Vowden P, Vowden K, Carville K. Antimicrobials Made Easy. *Wounds International*, 2011; 2(1).
11. Mueller CM, *The ASPEN Adult Nutrition Support Core Curriculum*. Vol. USA 2017 American Society for Enteral and Parenteral Nutrition.
12. Cederholm T, Bosaeus I, Barazzoni R, Bauer J, Van Gossum A, Klek S, Muscaritoli M, Nyulasi I, Ockenga J, Schneider SM, de van der Schueren MAE, Singer P. ESPEN endorsed recommendation: Diagnostic criteria for malnutrition - An ESPEN Consensus Statement. *Clin Nutr*, 2015; 34: 335-340.
13. Druml C, Ballmer P, Druml W, Oehmichen F, Shenkin A, Singer P, Soeters P, Weimann A, Bischoff SC. ESPEN guideline on ethical aspects of artificial nutrition and hydration. *Clin Nutr*, 2016; 35(3): 545-556.
14. National Health and Medical Research Council, Australian Guidelines for the Prevention and Control of Infection in Healthcare. 2010, Commonwealth of Australia.
15. AWMA, *Bacterial Impact on Wound Healing: From Contamination to Infection. Position Paper*. 2011, <http://www.awma.com.au/publications/publications.php>: AWMA.
16. Bowler PG. Wound pathophysiology, infection, and therapeutic options. *Annals of Internal Medicine*, 2002; 34: 419-27.
17. James GA, Swogger E, Wolcott R, Pulcini Ed, Secor P, Sestrich J, Costerton JW, Stewart PS. Biofilms in chronic wounds. *Wound Repair and Regeneration*, 2008; 16(1): 37-44.
18. Nast A, Griffiths C, R. Hay R, Sterry W, Bologna JL. The 2016 International League of Dermatological Societies' revised glossary for the description of cutaneous lesions. *Br J Dermatol*, 2016; 174: 6.
19. World Health Organization. ICD 11: 1B70 Bacterial cellulitis, erysipelas or lymphangitis.
20. AWMA, Standards for Wound Management. 2010, The Australian Wound Management Association Inc Australia.
21. Chin-Yee B, Upshur R. Clinical judgement in the era of big data and predictive analytics. *J Eval Clin Pract*. , 2018; 24(3): 638-645.
22. Field A, *Discovering Statistics using SPSS*. 2017: Sage.
23. Schultz GS, Sibbald RG, Falanga V, Ayello EA, Dowsett C, Harding K, Romanelli M, Stacey MC, Teot L, Vanscheidt W. Wound bed preparation: a systematic approach to wound management. *Wound Repair and Regeneration*, 2003; 11(2): S1-28.
24. Baharestani M, *The clinical relevance of debridement*, in *The Clinical Relevance of Debridement*, M. Baharestani, P. Holstein, and W. Vanscheidt, Editors. 1999, Springer-Verlag: Heidelberg, Germany. p. 1-15.
25. Ayello EA, Baranoski S, Cuddigan J, Sibbald RG, Kerstein MD, *Wound Debridement Essentials: Practice Principles*, in *Wound Care* S. Baranoski and E.A. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 119-35.
26. ASPEN Board of Directors and the Clinical Guidelines Task Force. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *Journal of Parenteral and Enteral Nutrition*, 2002; 26: 225A - 245A.
27. Vanderwee K, Grypdonck M, Bacquer D, Defloor T. The reliability of two observation methods of nonblanchable erythema, Grade 1 pressure ulcer. *Appl Nurs Res*, 2006; 19: 156-162.
28. Baranoski S, Ayello EA, Langemo DK, *Wound assessment*, in *Wound Care Essentials: Practice Principles*, S. Baranoski and E.A. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 77-92.
29. Carville K, *Wound Care Manual*. 2017, Osborne Park, Western Australia: Silver Chain Foundation.
30. National Center for Chronic Disease Prevention and Health Promotion Division of Population Health. *Health-Related Quality of Life (HRQOL)*. 2018 [cited October 2019]; Available from: <https://www.cdc.gov/hrqol/index.htm>.
31. World Health Organization. ICD 11: EK02.22 Irritant contact dermatitis due to incontinence.
32. World Health Organization. ICD 11: EK02.20 Intertriginous dermatitis due to friction, sweating or contact with body fluids.
33. Rich J, Neely JG, Paniello RC, Voelker CCJ, Nussenbaum B, Wang EW. A practical guide to understanding Kaplan-Meier curves. *Otolaryngology-Head and Neck Surgery*, 2010; 143: 331-336.
34. Streiner D, Norman G, Cairne J, *Health Measurement Scales: A Practical Guide to Their Development and Use*. 5th ed. 2014: Oxford University Press.
35. Dorland, Dorland's Illustrated Medical Dictionary. 2011, Elsevier Health Sciences Division.: New York, NY.
36. Imhof RE, De Jesus ME, Xiao P, Ciortea L, Berg E. Closed-chamber transepidermal water loss measurement: microclimate, calibration and performance. *Int J Cosmet Sci*, 2009; 31(2): 97-118.
37. Agency for Health Care Policy and Research, *Pressure ulcers in adults: prediction and prevention. Clinical Practice Guideline No. 3. US Dept. of health & Human Services, Public Health Service, (Pub. No. 92-0047)*. 1992, Rockville, MD: AHCPR.
38. Polit D, Beck C, *Nursing Research: Generating and Assessing Evidence for Nursing Practice*. 10th ed. 2016: Wolters Kluwer.
39. Langemo DK, *Palliative wound care*, in *Wound Care Essentials: Practice Principles*, S. Baranoski and E.A. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 448.
40. Conner-Kerr T, *Phototherapy in Wound Management*, in *Wound Care: A collaborative Practice Manual for Health Professionals*, C. Sussman and B. Bates Jensen, Editors. 2007, Lippincott, Williams, and Wilkins. p. 591-611.
41. Yastrub DJ. Relationship between type of treatment and degree of wound healing among institutionalized geriatric patients with stage II pressure ulcers. *Care Management*, 2004; 5(4): 213-8.
42. Pieper B, National Pressure Ulcer Advisory Panel, eds. *Pressure Ulcers: Prevalence, Incidence, and Implications for the Future*. 2012, NPUAP: Washington, DC.
43. Tarnuzzer RW, Schultz GS. Biochemical analysis of acute and chronic wound environments. *Wound Repair and Regeneration*, 1996; 4(3): 321-325.
44. Call E, Pedersen J, Bill B, Black J, Alves P, Brindle CT, Dealey C, Santamaria N, Clark M. Enhancing pressure ulcer prevention using wound dressings: what are the modes of action? *Int Wound J*, 2013; epub.
45. Bergstrom N, Allman, R., Carlson, C., et al. , *Pressure ulcers in adults: prediction and prevention. Clinical practice guideline No. 3. . 1992, Rockville, MD: Agency for Healthcare Policy and Research, AHCPR Pub. No. 920047.*

46. Becker M, Breuing J, Nothacker M, Deckert S, Steudtner M, Schmitt J, Neugebauer E, Pieper D. Guideline-based quality indicators—a systematic comparison of German and international clinical practice guidelines: protocol for a systematic review. *Systematic Reviews*, 2018; 7(5): DOI 10.1186/s13643-017-0669-2.
47. Pierce P. Defining and measuring quality of life. *Journal of Wound Care*, 1996; 5(3): 139-40.
48. Attia J. Moving beyond sensitivity and specificity: using likelihood ratios to help interpret diagnostic tests. *Australian Prescriber*, 2003; 26(5): 111-113.
49. World Health Organization. ICD 11: 1G40 Sepsis without septic shock.
50. Baranoski S, Ayello EA, McIntosh A, Galvan L, Scarborough P, *Wound treatment options*, in *Wound Care Essentials: Practice Principles*, S. Baranoski and E.A. Ayello, Editors. 2008, Lippincott: Philadelphia. p. 136-71.
51. Kottner J, Beeckman D, Vogt A, Blume-Peytavi U, *Skin Health and Integrity*, in *Innovations and Emerging Technologies in Wound Care*, A. Gefen, Editor. 2019, Elsevier Academic Press,.
52. International Union of Pure and Applied Chemistry. *Manual of Symbols and Terminology for Physicochemical Quantities and Units*. 2001 [cited August 2019]; Available from: https://old.iupac.org/reports/2001/colloid_2001/manual_of_s_and_t/node36.html.
53. World Health Organization. ICD 11: EH90 Pressure ulceration.
54. Lange ML. Tilt in space versus recline--New trends in an old debate. *Technology Special Interest Section Quarterly*, 2000; 10(1-3).
55. Leonard RB. To tilt or recline. *Top Spinal Cord Inj Rehabil*, 1995; 1(1): 17-22.
56. Nates JL, Nunnally M, Kleinpell R, Blosser S, Goldner J, Birriel B, Fowler CS, Byrum D, Miles WS, Bailey H, Sprung CL. ICU admission, discharge, and triage guidelines: A framework to enhance clinical operations, development of institutional policies, and further research. *Crit Care Med*, 2016; 44(8): 1553-602.
57. World Health Organization. ICD 11: EH90.5 Pressure ulceration, ungradable.

APPENDIX ONE: GUIDELINE METHODOLOGY

Introduction

The following methodology was used for the development of this edition of the guideline. The methodology was made available to all stakeholders via a peer reviewed publication,¹ and on the guideline website (www.internationalguideline.com).

The methodology for this edition of the guideline is revised from 2014 to ensure current international standards in guideline development are addressed and rigorous guideline development is maintained. The guideline continues to focus on primary evidence, and included a consensus voting process to assign a '*strength of recommendation*' to each evidence-based recommendation statement. This process is intended to provide an indication of the confidence a health professional can have that implementation of the recommendation will promote positive outcomes. The rating can be used to prioritize interventions. This Appendix provides an abridged overview of the process, further information (e.g., conflict of interest forms) is available on the guideline website.

Guideline Website

<http://www.internationalguideline.com>

The guideline website was established to publish documents associated with the Guideline. The Guideline website is used to disseminate the *Quick Reference Guideline*, acknowledge sponsors, and publish supportive documents, updates and statements from the GGG.

Participants in the Guideline Development

All members of the development team were screened for experience, expertise and potential conflicts of interest through an expression of interest and application process. In the interest of transparency, all contributors to the guideline were required to identify potential conflicts of interest (COIs) and their approximate value. Potential COIs were declared and managed based on an adapted version of the *Guidelines International Network Principles*.² Conflict of interest declarations were completed annually in writing and reported to the methodologist whenever a new conflict arose. The final COI declarations will be published on the guideline website. Participants with a 'moderate' to 'very high' COI according to *Appendix Table 2* in Schünemann et. al. (2015)² abstained from reviewing and critically appraising any research in their area of the conflict, and were excluded from group discussions, chapter preparation and strength of evidence rating.

Member Organizations

This revision of the guideline was overseen by the Member Organizations: European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP) and the Pan Pacific Pressure Injury Alliance (PPPIA).

Associate Organizations

Other international not-for-profit pressure injury organizations that share the mission, values and purposes of the Member Organizations were invited to apply to join the development process through designation as Associate Organizations. The following organizations were successful:

- Chinese collaboration of: Chinese Nursing Association and Jiangsu Nursing Association
- Indonesian collaboration of: Indonesian Wound Ostomy and Continence Nursing Association and Indonesian Wound Care Clinician Association
- Canadian collaboration of: Canadian Association for Enterostomal Therapy and Wounds Canada
- Japanese Society for Pressure Ulcers
- Korean Association of Wound Ostomy Continence Nurses
- Malaysian Society of Wound Care Professionals
- Philippine Wound Care Society
- Saudi Chapter of Enterostomal Therapy
- Taiwan Wound Ostomy and Continence Nurse Association
- Thai Enterostomal Therapy Society

- World Council of Enterostomal Therapists
- Brazilian Association of Enterostomal Therapists: Wound, Ostomy and Continence Care.

Guideline Governance Group (GGG)

The GGG monitored each step of the guideline development process and manage the guideline dissemination strategy. Each of the three Member Organizations nominated four representatives each, to form the 12-member GGG. The four nominated representatives for each member organization appointed a Chair. The GGG members all voted during joint deliberations, with the majority deciding. Examination of the evidence and consensus building preceded all voting.

Small Working Groups (SWG)

The guideline content was divided into working topic areas and SWGs formed to review the evidence. The SWG members were selected based on experience and expertise. Representatives of industry were excluded. The SWGs were formed based on the principle of equal contribution from the Member Organizations and representation from at least one Associate Organization. Guideline development was an iterative process, with GGG and SWG members maintaining communication via the methodologist.

Patient Consumers and Their Informal Caregivers

Consumers (patients and caregivers) were invited to engage in the development process. At commencement of the project, an international survey of consumers was undertaken to establish consumer needs, consumer interest in outcome measures and inform development of the clinical questions. This process is reported elsewhere in this methodology summary.

Methodologist

The guideline process was overseen by an experienced guideline methodologist. The methodologist assisted the SWG members in implementing the documented methodology, appraising and summarizing the new literature, and guided review and development of recommendations. The methodologist managed the confidential consensus voting process and COIs. The methodologist provided a link between the GGG and Associate Organizations, and between the GGG and the SWGs. The methodologist attended GGG and SWG meetings, but did not participate in any voting in meetings or in the strength of recommendation process

Stakeholders

The process of development was made available to stakeholders on the guideline website. A stakeholder is anyone who has interest in pressure injuries and wishes to contribute by reading the methodology, search strategies, references under consideration, and commenting on the draft guideline. Anyone may register as a stakeholder, either as an individual or as a representative for a society/organization.

Methods

The steps for the guideline development process are delineated briefly below. For simplicity and clarity, the process is described as linear and sequential; however, the actual process was iterative, with multiple drafts developed and progressively improved.

Identifying the Evidence

Databases

The GGG identified clinical questions to guide literature searches. To identify the scientific literature on pressure injury prevention and treatment, several electronic databases were consulted, including:

- AMED
- MEDLINE
- EMBASE
- Scopus
- The Cochrane Database of Systematic Reviews
- The Cochrane Central Register of Controlled Trials
- Health Technology Assessment

The search dates for this update were 1st July 2013 through 31st August 2018. Evidence from previous editions of the guideline has also been retained in this edition.

Search strategy

A sensitive search strategy was developed and made available on the guideline website. The SWGs conducted additional focused searches to ensure the full depth and breadth of their topic area has been covered, if required. All references retrieved by the electronic literature search were screened by the methodologist based on the inclusion and exclusion criteria that follows.

General eligibility criteria

- The articles must be primarily focused on pressure injury prevention, risk assessment, or pressure injury treatment in human subjects.
- The articles must have been published in a peer reviewed journal.
- An abstract should be available.

Inclusion criteria for primary research:

- The studies should have used one of the following designs:
 - Randomized controlled trials (RCTs)
 - Prospective controlled clinical trials (CCTs)
 - Prospective cohort studies with a control group
 - Pre-test/post-test studies
 - Retrospective cohort studies
 - Observational studies
 - Cross-sectional studies
 - Survey studies
 - Case-control studies
 - Case series.
- At least ten subjects must have been included in any case series.
- Studies using established qualitative methodologies were considered as appropriate to the clinical question (e.g., the individual's experiences, such as pain).

Inclusion criteria for synthesized research:

- Systematic reviews and meta-analyses were only for comparative discussion, clearly delineated as supportive content in the guideline layout. These sources of evidence were not included in the strength of evidence rating.
- Identified systematic reviews and meta-analyses will be screened for eligibility using the AMSTAR 2 tool. For inclusion, these evidence sources were required to meet all of the critical domains listed in *Table 30.1*.³
- SWG members reviewed the original articles cited in systematic reviews and meta-analyses.
- Other forms of synthesized evidence (e.g. other clinical guidelines) were considered only to support background discussion, good practice statements or implementation considerations, as required.

Research published in languages other than English was screened by evaluating the English abstract. There was no restriction based on the language. A pool of translators was identified among guideline participants. Publications were selected for appraisal and data extraction by one translator only when there was a likelihood of providing unique, high level evidence not available in the current body of evidence.

Table 30.1: Critical domains for systematic reviews to meet for inclusion (adapted from Shea et. al.³)

<ul style="list-style-type: none"> • Adequate literature search conducted as per criteria for YES on AMSTAR 2 tool (item 4) • Studies read in full but excluded are individually listed, with justification for excluding each individual study (Amstar 2, item 7) • Risk of bias in individual included studies is evaluated as per criteria on AMSTAR 2 tool. This includes assessment of risk of bias from specific items as per AMSTAR 2 tool. (item 9) • Appropriate meta-analytical methods are used as per criteria on AMSTAR 2 tool, with justification for combining in meta-analysis and use of an appropriate weighting technique and adjustment for heterogeneity when present (item 11) • Consideration is given to risk of bias in individuals studies when interpreting the review results by either including only RCTs at low risk of bias or if RCTs have a moderate or high risk of bias or non-randomized trials are included, the impact of this is discussed (Amstar 2 item 13) • An assessment is conducted of presence and likely impact of publication bias as per criteria on AMSTAR 2 tool i.e. for reviews with a quantitative synthesis, graphical or statistical tests for publication bias are performed and discussed the likelihood and magnitude of impact of publication bias (item 15)
--

Exclusion criteria:

- Non-systematic literature reviews, narrative papers, opinion, commentary, other clinical guidelines and descriptive papers. Papers falling into this category were only used only to support background discussion, good practice statements or implementation considerations, as required.
- Case series with less than 10 participants.
- Conference abstracts or o short papers with insufficient detail to enable appraisal.
- Duplicate reports of research.
- Computational modeling and other research conducted in non-human subjects, except to support background discussion.
- Systematic reviews and meta-analyses not meeting all the critical domains listed in Table 1.
- Papers without a substantial focus on pressure injury prevention or treatment or risk assessment.
- Papers in lanugages other than English for which the abstract did not indicate unique, high-level evidence.

Eligibility criteria for research reporting on quality improvement and education

In addition to the criteria outlined above, additional inclusion criteria were:

- Time series design with at least three outcome measurement time points, with data covering at least 12 months.
- Project should be institution-wide (i.e., not individual units).
- Outcomes should be incidence or facility-acquired pressure injury rates.
- Quality improvement projects should be described in sufficient detail to enable replication (i.e., specific methods used, barriers and facilitators).
- Publications before January 2008 were not considered, unless identified as seminal by the SWG or GGG.

Eligibility criteria for research reporting on pressure injuries risk factors

In the 2014 guideline, a systematic review by Coleman et al. (2013)⁴ was used as a basis for literature selection to identify patient characteristics that increase the probability of pressure injury development. This review was supplemented by a search extended to literature published up to 31st August 2018 for the 2019 edition. The inclusion and exclusion criteria applied by Coleman et. al. (2013)⁴ are applied to all literature:

Inclusion criteria:

- Primary research
- Adult patients (aged \geq 18 years)
- Outcome was the development of a new pressure injury
- Prospective cohort, retrospective record review (where the risk factor preceded the pressure injury) or controlled trials
- Length of follow-up at least three days, with the exception of operating room studies
- Outcome clearly defined as Category/Stage I or greater pressure injury or equivalent
- Multivariable analyses undertaken to identify factors affecting pressure injury outcome
- The unit of analysis was the individual patient.

Exclusion criteria:

- Cross-sectional, case-study, patient recall or self-report, analysis of general practitioner records.
- Duplicate publication of a patient dataset
- Cohort studies (prospective and record reviews) in which more than 20% of the study sample were excluded from analysis for reasons including withdrawal, death, loss to follow-up and missing records
- Controlled trials in which these minimum criteria did not apply: randomised allocation to treatment and intention to treat analyses.

Evaluating the Evidence

The methodological quality of each study was evaluated by two reviewers. Where large discrepancy of opinion was noted (such that the paper's overall quality was rated differently by the two reviewers), a third reviewer evaluated the paper.

The methodological quality of each study was assessed using methodology checklists. Evaluation of study quality focused on the study internal and external validity of the studies. The following broad quality criteria was considered: internal validity; clear and appropriate research question(s); selection of subjects; allocation; baseline comparability; outcomes; blinding; confounding factors; statistical analysis; overall assessment of the study; and potential bias. Specific critical appraisal tools were used for different research designs, and these tools are described in more detail in the full methodology report available on the guideline website.

Each criterion on critical appraisal checklists was assessed as being met, not met not reported/unclear, or not applicable. Unless alternate methods were stated on specific tools, studies will be described as high, moderate, or low quality using the following criteria:

- High quality studies: fully meeting at least 80% of applicable criteria
- Moderate quality studies: fully meet at least 70% of applicable criteria
- Low quality studies: did not fully meet at least 70% of applicable criteria.

Appraisal of methodological quality for risk factor research

Coleman et. al. (2013)⁴ used an assessment framework based upon guidelines for assessing quality and risk of bias in prognostic studies and methodological considerations in the analysis, meta-analysis and publication of observational studies. Because this guideline built on Coleman et al.'s⁴ review, evidence was appraised using the same methods, which evaluated the following factors:

- Baseline study sample (i.e. individuals entering the study) dequately described for key characteristics
- Clear definition or description of the risk factor measured (e.g., including dose, level, duration of exposure and clear specification of the method of measurement)
- Continuous variables used or appropriate (i.e., not data-dependent) cut-points for continuous data.
- Risk factor measurement valid and reliable
- Adequate proportion of sample had complete data for risk factors
- Range of potential risk factors measured (i.e., key variables in conceptual model; potential confounders accounted for in study design)
- Range of potential risk factors are accounted for in the analysis (i.e., appropriate the adjustment, potential confounders accounted for in analysis)
- Appropriate imputation
- No selective reporting.

In addition, specific consideration was given to the following quality domain:

- Is there sufficient number of events (rule of thumb: more than 10 events per risk factor)?
- Is there sufficient presentation of data to assess the adequacy of method and analysis?
- Is the strategy for model building (i.e., inclusion of variables) appropriate and based upon a conceptual framework?
- Is the selected model adequate for the design?

Table 30.2: Relationship between appraisal criteria and quality domains for risk factor studies (From Coleman, used with permission)

CRITERIA 1-8	QUALITY DOMAINS 1-4			
	1. Is there sufficient number of events (rule of thumb: more than 10 events per risk factor)?	2. Is there sufficient presentation of data to assess the adequacy of method and analysis?	3. Is the strategy for model building (i.e., inclusion of variables) appropriate and based upon a conceptual framework?	4. Is the selected model adequate for the design?
1.The baseline study sample is adequately described for key characteristics.		X		
2. A clear definition/ description of the risk factor measured is provided and a clear definition/ description of how the risk factor was measured is provided		X	X	X
3. Continuous variables used or appropriate (i.e. not data-dependent) cut-points for continuous data.		X	X	
4.An adequate proportion of sample has complete data for risk factors.		X	X	X
5.Range of potential risk factors are measured			X	X
6.Range of potential risk factors are accounted for in the analysis			X	X
7.Appropriate imputation			X	X
8.No selective reporting		X	X	X

Each of the above four quality domains were assessed as being met (yes/no/partial/unsure) using the criteria as outlined in *Table 30.2*. Studies will be classified as high, moderate, low and very low quality using the following criteria:

- High quality studies: 'yes' for all quality domains
- Moderate quality studies: 'yes' for quality domain 1 and at for least two other quality domains
- Low quality studies: 'no' for criteria 1 and 'no' or 'partial yes' for two other quality domains
- Very low quality studies: 'no' for criteria 1 and 'no' or 'partial yes' for all three remaining quality domains

Level of evidence

The '*level of evidence*' for individual intervention studies will be noted for each study containing direct evidence, using a classification system adapted from The Joanna Briggs Institute^{5,6} (see *Table 30.3*).

Levels of evidence are typically applied to intervention studies (e.g., RCTs, CCTs or case series studies) because these studies are regarded as important knowledge sources for clinical decision making. However, there are many more study designs (e.g., epidemiological or descriptive studies) that provide valuable evidence to guide practice yet cannot be classified with an intervention-based level of evidence system.

Table 30.3: Level of evidence for intervention studies^{5,6}

Level 1	Experimental Designs <ul style="list-style-type: none"> • Randomized trial
Level 2	Quasi-experimental design <ul style="list-style-type: none"> • Prospectively controlled study design • Pre-test post-test or historic/retrospective control group study
Level 3	Observational-analytical designs <ul style="list-style-type: none"> • Cohort study with or without control group • Case-controlled study
Level 4	Observational-descriptive studies (no control) <ul style="list-style-type: none"> • Observational study with no control group • Cross-sectional study • Case series (n=10+)
Level 5	Indirect evidence: studies in normal human subjects, human subjects with other types of chronic wounds, laboratory studies using animals, or computational models

Studies on diagnostic and prognostic validity of pressure injury risk and classification form an important body of knowledge that were appraised independently from intervention studies. Diagnostic accuracy studies are studies in which results of index tests are compared with results from reference standards at the same point in time.⁷ Therefore, cross-sectional designs are needed to establish the concurrent existence of both index test and reference standard results. Most studies in pressure injury risk research are not diagnostic accuracy studies according to this widely agreed upon definition, because the measured pressure injury risk is often compared with subsequent pressure injury occurrence. These designs resemble those of prognostic studies or diagnostic accuracy studies with imperfect reference standards.⁸

Comparable to different phases of intervention research, phases of diagnostic and prognostic research can also be distinguished. In diagnostic research, Phase I and II studies focus on differentiation between individuals with the target from those without. Phase III studies are typical diagnostic accuracy studies whereas phase IV research investigates the clinical impact of diagnostic procedures.⁹ Prognostic studies are comparable with diagnostic accuracy studies with the difference that, based on factors or diagnostic cues, future events are predicted. These types of studies are typically used to develop prognostic models. Prognostic models (e.g., pressure injury risk assessment tool scores) are used to predict the probability of future events in individuals or groups.¹⁰

Test accuracy and validity estimates are only surrogate measures for clinical effectiveness.¹¹ The clinical effectiveness of diagnostic test procedures can only be adequately investigated by diagnostic RCTs.^{12,13} In case of diagnostic or prognostic RCTs the described level of evidence hierarchy of intervention studies is used. Corresponding 'level of evidence' hierarchies for diagnostic and prognostic accuracy have been proposed^{12,14} and have been adopted by the GGG since the 2014 guideline edition (see *Tables 30.4 and 30.5*).

Table 30.4: Adapted levels of evidence for diagnostic studies^{12,14}

Level 1	Individual high quality (cross sectional) studies according to the quality assessment tools with consistently applied reference standard and blinding among consecutive persons.
Level 2	Non-consecutive studies or studies without consistently applied reference standards.
Level 3	Case-control studies or poor or non-independent reference standard.
Level 4	Mechanism-based reasoning, study of diagnostic yield (no reference standard). Low and moderate quality cross sectional studies.

Table 30.5: Adapted levels of evidence for prognostic studies^{12,14}

Level 1	A prospective cohort study.
Level 2	Analysis of prognostic factors amongst persons in a single arm of a randomized controlled trial.
Level 3	Case-series or case-control studies, or low quality prognostic cohort study, or retrospective cohort study.

Data Extraction

The full papers of included references were obtained and made available to SWGs on a web-based platform. A data extraction template was used to extract relevant data from individual papers, including study design; participant description; study groups and interventions; outcome measures; length of follow up; study results; limitations and general comments. Data extraction tables were completed by one reviewer and checked by at least one additional reviewer. The GGG reviewed 20% of data extraction tables and established a high level of accuracy. Evidence tables from previous guideline editions were made available to SWGs to ensure the full body of scientific literature was compiled and reviewed.

Terminology and Product Names

Terminology to describe pressure injuries is varied across geographic jurisdictions and, in some regions, across professional boundaries. Terminology describing both a pressure injury, and the severity of a pressure injury has been adapted in many geographic regions and is considered to be an ongoing area of evolution. For clarity, consistent terminology was required throughout this document. In the absence of a single, validated classification system, the GGG voted at the outset of the development of this edition of the guideline to use the term '*pressure injury*' and to use the classification terminology that was internationally agreed on and published in the International EPUAP/NPUAP Pressure Ulcer Classification System (2014).¹⁵ A crosswalk of classification systems is available in the guideline to assist health professionals in converting terminology to the recognized system in their jurisdiction.

The term '*individual*' was selected to describe the patient, client, resident, or person with a pressure injury or at risk for a pressure injury.

The terms '*health professional*' and '*interprofessional team*' were selected for use when referring to health professionals and non-professional healthcare workers providing formal healthcare services to the individual. The disciplines of professionals/healthcare workers performing a given service may vary from country to country based on the laws and regulations governing healthcare providers.

The term '*informal caregiver*' was selected to describe people providing care to the individual outside the context of formal healthcare services. This generally refers to family members and friends.

The guideline does not endorse or be seen to endorse the use of any specific products, manufacturers, services or companies. Consistent with best practice in developing clinical guidelines, brand/product names have not been used in recommendation statements or the discussion.^{16,17} Where available, generic names or product classifications was used. The guideline includes descriptions of the features of products that may relate to their effectiveness (or otherwise) when reported in study results. Descriptions of products used in the appraised research was used in reporting because this is what has been presented in the peer reviewed evidence. More information was sought from the manufacturer's product information as required. In evidence tables, product names were used to describe intervention and control products used in a specific trial on the first time the product/s was referenced.

Drafting and Revising Recommendations

Each SWG formulated conclusions about the body of evidence available to address each clinical question. These conclusions were based on the evidence tables, critical appraisals and levels of evidence. The process was facilitated using an adapted evidence-to-decision framework^{18,19} that was finalized by the GGG. An evidence-to-decision framework presents a summary of the evidence, together with the relative pros and cons for an intervention. The evidence-to-decision framework guided the development of recommendations addressing the clinical questions and allowed a concise summary of the evidence underpinning each recommendation. The SWGs evaluated the volume and consistency of evidence, the probability of benefits and harms, and additional evaluations that would be used to assign strengths of recommendation (see *Assigning Strength of Recommendation Ratings*). Evidence-to-decision frameworks were used to summarize evidence underpinning recommendations based on prognostic or diagnostic studies; however, the evaluation of probability of benefits and harms was not relevant. Other evaluations, used to assign '*Strength of Recommendation*', were made.

A first draft of recommendations was developed by the SWGs and/or the methodologist. The GGG reviewed the draft recommendations, making revisions as necessary. To ensure uniformity and internal consistency in the final guideline, the GGG applied the following guidance:

- Each recommendation should start with a direct action verb and be a simple, short, direct, declarative statement, free of jargon
- Recommendation statements should be broad recommendations on clinical practice (e.g., broad, directive statements). Additional subsequent statements with more detail (e.g., how, when or how often) that support recommendations could be included as implementation considerations

- Recommendations should be specific and unambiguous
- When available from the evidence, information on health benefits, side effects and risks should be provided.

The GGG reviewed all recommendations to ensure the wording of the recommendations accurately translates available research into best practice while being sensitive to the many different individual cultures and professional standards represented among the international audience for these guidelines. This will additionally be reviewed by the Consumer SWG.

Assigning Strength of Evidence Ratings

The SWGs summarized the evidence supporting each recommendation. An explicit link between the recommendation and supporting evidence was expected. '*Strength of evidence*' ratings were assigned to recommendations. This rating identifies the strength of cumulative body of evidence supporting each recommendation. *Table 30.6* outlines the strength of evidence rating system (adapted from NHMRC methodology).²⁰

Table 30.6: Strength of evidence rating for each recommendation (adapted from NHMRC)²⁰

A	<ul style="list-style-type: none"> • More than one high quality Level I study providing direct evidence • Consistent body of evidence
B1	<ul style="list-style-type: none"> • Level 1 studies of moderate or low quality providing direct evidence • Level 2 studies of high or moderate quality providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
B2	<ul style="list-style-type: none"> • Level 2 studies of low quality providing direct evidence • Level 3 or 4 studies (regardless of quality) providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
C	<ul style="list-style-type: none"> • Level 5 studies (indirect evidence) e.g., studies in normal human subjects, humans with other types of chronic wounds, animal models • A body of evidence with inconsistencies that cannot be explained, reflecting genuine uncertainty surrounding the topic
Good practice statement	<ul style="list-style-type: none"> • Statements by the GGG that are not supported by a body of evidence as listed above but considered significant for clinical practice.

The strengths and limitations of this body of evidence was clearly described. All recommendations with a '*strength of evidence*' rating required an explicit evidence summary (justification). For '*strength of evidence*' rating A or B (1 or 2), the evidence summary required one or more studies conducted with human subjects with or at risk of pressure injuries. The '*level of evidence*' for each study and its quality rating was identified in the evidence summary.

An attempt had been made to explicitly identify evidence gaps. The systematic searches identified indirect evidence from studies of normal subjects, studies with intermediate or surrogate outcomes, studies of humans with other types of chronic wounds, and animal studies. Indirect evidence was used to support '*strength of evidence C*' ratings or good practice statements. A clarification was made for '*Strength of evidence*' (SoE) of C regarding whether the recommendation was supported by:

- SOE C: indirect evidence from studies of normal subjects
- SOE C: studies with intermediate or surrogate outcomes
- SOE C: studies of humans with other types of chronic wounds, and animal studies or other basic bench research.

In the absence of adequate evidence, a good practice statement was made for clinical practice areas considered very important. Good practice statements were made when they were perceived by the GGG to be necessary. A good practice statement is designed to should assist health professionals to take appropriate actions in areas of uncertainty.²¹ Good practice statements were based on expert opinion, supported by other evidence-based clinical guidelines or other types of research that did not meet the criteria for an evidence-based recommendation. Appropriate referencing was used to support good practice statements.

The '*strength of evidence*' supporting the recommendation is not the same as the '*strength of the recommendation*'. For example, there may be no RCTs in individuals with pressure injuries evaluating commonly implemented practices. Therefore, a recommendation may have a relatively low '*strength of evidence*' supporting the recommendation, yet the recommendation may be strongly recommended in many clinical situations based on evidence from studies of other types of chronic wounds, proof of principle from basic science research, and/or expert opinion.

Stakeholder Review Process

After developing recommendations and good practice statements, implementations and the chapter discussion, stakeholder feedback was sought. A register of stakeholders who had specifically indicated their interest by completing their details at the guideline website were invited to participate. This register contained over 1,200 known interested stakeholders. In addition, the stakeholder review process was advertised through social media by the Partner and Associate Organizations. The stakeholder review process was also available to any person or organization who was interested to participate.

The guideline website contained the evidence-to-decision framework and full chapters for stakeholder review. The majority of guideline content was available for comment from stakeholders for four weeks (four chapters were available for only seven days due to time constraints). A total of 699 independent stakeholders (either individuals or representing organizations) accessed the guideline chapters and made comments or voted on whether they agreed with the recommendations in each chapter. Stakeholders were also given opportunity to identify any research meeting the eligibility criteria that may have been missed in the searches.

Comments from the stakeholder feedback process were reviewed by the GGG, and when applicable were also reviewed by the SWG members. Changes to the guideline content were made when the stakeholder feedback warranted changes or clarification.

Assigning Strength of Recommendation Ratings

As previously discussed, *'strength of evidence'* ratings identify the strength of cumulative evidence supporting the recommendation. In contrast, *'strength of recommendation'* ratings require a different analysis. The evidence-based recommendations are rated based on their importance and their potential to improve individual patient outcomes. The good practice statements were not given a strength of recommendation, consistent with current best practice in guideline development.²¹

The *'strength of recommendation'* is the extent to which a health professional can be confident that adherence to the recommendation will do more good than harm.

The rating is not necessarily related to the strength of internal or external evidence. The overall aim is to help health professionals to prioritize interventions. The following points have been considered:^{18,19,22-24}

- The balance between benefits and harms. The larger the difference between both, the higher the likelihood for giving a strong recommendation. However, this consideration is not applicable to prognostic and diagnostic recommendations
- The overall quality of evidence across all studies upon which the recommendation is based. The higher the quality, the higher the likelihood that a strong recommendation is warranted
- Translation of the evidence into practice in specific clinical settings or uncertainty of baseline risk in the populations of interest
- The higher the financial costs of an intervention, the greater the resources consumed, the lower the likelihood that a strong recommendation is warranted, unless cost effectiveness can be demonstrated
- The acceptability of the intervention to stakeholders, including patient consumers and health professionals
- The priority that patient consumers place on the outcomes the recommendation will achieve.

All SWG and the GGG members were invited to take part in the consensus voting process, each voting on every recommendation in the guideline. The consensus voting process was conducted on a purpose-built website platform, with each team member provided with a unique identification. The participants were required to confirm their understanding of the procedure before commencing. The voting criteria were made using an adapted GRADE grid^{18,19,22-24} (See *Table 30.7*). The process was facilitated using the evidence-to-decision framework^{18,19} that was finalized by the GGG. For each recommendation to be evaluated, voters were presented with a tabulated summary of the evidence relevant to the following questions:¹⁹

Derived from the reviewed evidence:

- How substantial are the desirable anticipated effects?

- How substantial are the undesirable anticipated effects?
- What is the overall certainty of the evidence of effects?
- Does the balance between desirable and undesirable effects favor the intervention or the comparison?
- How substantial are the resource requirements (costs)?

Derived from the reviewed evidence, input from SWG responsible for relevant chapter and stakeholder review:

- Is the intervention feasible to implement?

Derived from the reviewed evidence, with input from the consumer survey conducted as a part of the guideline development:

- Is there important uncertainty about or variability in how much patient consumers and their informal caregivers value the main outcomes?
- Is the intervention acceptable to patient consumers and their informal caregivers?
- Is the intervention feasible for patient consumers and their informal caregivers?

Table 30.7: Five types of recommendations^{18,19,22-24}

Recommendation	Symbol	Description	Implications
Do it: Strong recommendation for an intervention (We recommend offering this option)	↑↑	Indicates a judgment that most well informed people would make.	<i>For patient consumers</i> —Most people would want the recommended course of action and only a small proportion would not. <i>For health professionals</i> —Most people should receive the intervention. If health professionals choose not to follow the recommendation, they should document their rationale. <i>For quality monitors</i> —Adherence to this recommendation could be used as a quality criterion or performance indicator.
Don't do it: Strong recommendation against an intervention (We recommend against offering this option)	↓↓		
Probably do it: Conditional recommendation for using an intervention (We suggest offering this option)	↑	Indicates a judgment that a majority of well informed people would make, but a substantial minority would not.	<i>For patient consumers</i> —Most people would want the suggested course of action, but many would not. <i>For health professionals</i> —Examine, and be prepared to discuss, the evidence with patients, as well as their values and preferences. <i>For quality monitors</i> —Clinicians' discussion and consideration of pros and cons of the intervention, and documentation of discussion, could be used as a quality indicator.
Probably don't do it: Conditional recommendation against using an intervention (We suggest not offering this option)	↓		
No specific recommendation: Conditional recommendation for either the intervention or the comparison (We make no suggestion on offering this option)	↔	Trade-offs between risk and benefit unclear or lack of agreement between voting participants.	The advantages and disadvantages are equivalent; and/or the target population has not been identified; and/or there is insufficient evidence on which to formulate a 'strength of recommendation'.

After reviewing the evidence-to-decision framework, voters were asked to select a '*strength of recommendation*' rating from the options presented in *Table 7* (or to abstain from voting, with reason provided). Votes were recorded and calculated using a software program designed for the purpose. Rules were determined based on previous applications of this consensus voting process (ie., 2014 voting), and a desire to obtain significant consensus. Determination of the final '*strength of recommendation*' was made according to the following rules:

- To achieve a strong positive (do it) or strong negative (don't do it) recommendation, 100% of votes must be cast in the same direction (positive or negative), with at least 70% voting for a strong recommendation, and 0% voting in the opposite direction
- To achieve a weak positive (probably do it) or weak negative (probably don't do it) recommendation, at least 70% of votes must cast in the same direction (positive or negative), and less than 20% voting in the opposite direction
- Any other combination of voting results in 'no specific recommendation'.

Implementation Considerations and Quality Indicators

An implementation consideration is a suggestion on how to implement the core recommendations in the guideline that aim to provide guidance on clinical questions. Implementation considerations may describe how, when, where, who or how often to implement a recommended practice, or may identify core principles to consider when implementing the recommendation. Implementation considerations cover supplemental information considered pertinent to practice and are supported by level 1 to 5 evidence, other clinical guidance resources or consensus of the SWG and/or GGG.

Additionally, quality indicators that could be used to monitor the implementation of this guideline have been developed. A wide range of clinical indicators are currently used around the world as part of ongoing health service accreditation programs, international benchmarking projects and at local levels for monitoring ongoing quality improvement. The quality indicators have been designed to monitor the specific recommendations for practice that are included in this guideline. They were selected based on expert opinion on their intrinsic value as an indicator of quality care for prevention and treatment of pressure injuries, with consideration to practicalities of ongoing auditing. The indicators are proposed for use in health facilities/services in addition to other quality indicators as a measure of effectiveness in implementing the guideline locally.

Consumer Engagement in the Guideline

Consumer engagement is recognized as a requirement for high quality, international clinical guidelines.²⁵⁻²⁸ In the context of this Guideline, consumer engagement refers to involvement in guideline development from the following groups:

- Patient consumers (i.e. individuals with or at risk of a pressure injury)
- Informal caregivers (i.e. individuals caring in an informal capacity such as family members or friends)
- Consumer stakeholders (i.e. professional consumer representatives).

Recognising international standards, the goals of consumer engagement in this guideline development process were to:²⁹

- Promote the relevance of recommendations and guideline content to patient consumers
- Promote patient consumer values and preferences
- Acknowledge and respond to the needs of specific populations groups
- Evaluate and respond to consumer education/information needs
- Promote consumer awareness of the guideline.

The primary audience of the guideline is health professionals, academics and organizations/facilities, and the content and terminology are appropriate to this audience. Input from patient consumers sought to provide guidance on the development of companion resources for the guideline, including topics for region-specific patient consumer education.

The Guideline Governance Group (GGG) recognizes the diverse range of barriers guideline development teams face in promoting consumer engagement. The literature identifies a wide range of barriers to consumer engagement,^{26,28,29} including discrepancies between health professional experts and consumer perspectives regarding topics of interest; difficulty integrating consumer opinion into recommendation development; consumer recruitment and retention issues; limitations in consumer understanding of technical terminology; time and financial constraints; resistance to change; feelings of being undervalued; and cultural (e.g. language), health (e.g. sensory impairment) and physical (e.g. lack of internet) barriers.

The GGG considered the above factors in developing a consumer engagement strategy. Strategies to promote consumer engagement were developed based on recommendations on the literature on promoting consumer engagement.^{26,28-30} Consumer engagement strategies were developed for each of the guideline steps²⁶ (see *Table 30.8*).

Table 30.8: Guideline development team strategies to promote consumer engagement ^{26,28-30}

Guideline step	Processes to promote patient engagement
Recruitment	<ul style="list-style-type: none"> Information about the guideline development process will be publicly available. Both patient consumers and consumer stakeholders will be eligible and invited to participate. Diverse patient consumers will be encouraged by recruiting in all countries involved in the guideline development. Diverse health professionals will be recruited to promote consideration of the needs of diverse patient consumers.
Preparation	<ul style="list-style-type: none"> Patient consumers will receive background information about the guideline and goals of consumer participation.
Logistics	<ul style="list-style-type: none"> Methods for providing opportunity for patient consumer contribution are pre-identified in this protocol. The methodologist will co-ordinate consumer engagement (e.g. send invitations, administer survey, collect written feedback, etc.). Contribution from patient consumers will be sought in an online survey, with consideration to simplicity in language, questions, methods of response and time to complete the survey. Updates on the guideline development process are published on the International Guideline website.

Consumer Survey

At commencement of the project, an international survey of consumers was undertaken to establish consumer needs, consumer interest in outcome measures and inform development of the clinical questions. Broad consumer input will be sought, with a goal of collecting information from consumers in all geographic regions participating in the guideline. Consumers were recruited to complete the consumer survey and/or to register as a stakeholder for the stakeholder review process. Consumer engagement was invited through website invitations, social media and invitations to consumer stakeholder groups known to GGG members in all geographic regions.

The consumer survey received ethics approval from the Australian National University Human Research Ethics Committee (HREC Protocol 2018/066) and the methodology and findings will be published independent to the guideline. Presentation of the results has already been publically made at relevant international conferences.^{31,32}

In brief, the consumer survey was made available from 24 April 2018 to 30 October 2018 at a purpose-built website. The survey was presented in simple language and translated into languages other than English by the SWG translators. The online survey explored:

- Topics of patient priority
- Relevance of clinical questions to patient needs
- Patient care goals and education needs
- Invitation to register as stakeholder.

The survey achieved participation from 1,233 patient consumers (n = 383) and informal caregivers (n = 850) from 27 countries. Information collected via the survey was to review and revise the clinical questions, to contribute to the evidence-to decision-framework evaluations and will be used to develop priorities for consumer education material. The consumer survey results also informed specific recommendations, particularly surrounding care goals and education needs.

Guideline Monitoring and Updates

The GGG will continue to monitor guideline implementation after the guideline is published, encouraging translation of the guideline into non-English languages for maximum dissemination. The 2009 Quick Reference Guide was translated into 17 different languages, the 2014 full guideline was translated into two different languages and the Quick Reference Guide was translated into 11 different languages.

The GGG will continue to monitor the pressure injury literature after the 2019 guideline has been published. This is an ongoing process in the interim between published guideline editions and seeks to prepare for the next guideline edition, as well as identify any pertinent advances of knowledge. In case of the latter, the GGG publish statements on the guideline website to clarify interpretation of the guideline in the context of important new knowledge. The next published revision of this guideline is planned for 2024.

References

1. Kottner J, Cuddigan J, Carville K, Balzer K, Berlowitz D, Law S, Litchford M, Mitchell P, Moore Z, Pittman J, Sigauco-Roussel D, Yee CY, Haesler E. Prevention and treatment of pressure ulcers/injuries: The protocol for the second update of the international Clinical Practice Guideline 2019. *J Tissue Viability*, 2019; 28(2): 51-58.
2. Schünemann HJ, Al-Ansary LA, Forland F, Kersten S, Komulainen J, Kopp IB, Macbeth F, Phillips SM, Robbins C, van der Wees P, Qaseem A, Network. BoTotGl. Guidelines International Network: Principles for disclosure of interests and Management of conflicts in guidelines. *Ann Intern Med*, 2015; 163(7): 548-53.
3. Shea BJ, Reeves BC, Wells G, Thuku M, Hamel C, Moran J, Moher D, Tugwell P, Welch V, Kristjansson E, Henry DA. AMSTAR 2: a critical appraisal tool for systematic reviews that include randomised or non-randomised studies of healthcare interventions, or both. *BMJ* 2017; 358: j4008.
4. Coleman S, Gorecki C, Nelson A, Closs SJ, Defloor T, Halfens R, Farrin A, Brown J, Schoonhoven L, Nixon J. Patient risk factors for pressure ulcer development: Systematic review. *International Journal of Nursing Studies*, 2013; e-pub.
5. Joanna Briggs Institute, Reviewers' Manual 2014. 2014, Joanna Briggs Institute,; Adelaide.
6. Joanna Briggs Institute, Reviewers' Manual 2014: Summary of Findings Tables for Joanna Briggs Systematic Reviews. 2014, Joanna Briggs Institute: Adelaide.
7. Bossuyt PM, Reitsma JB, Bruns DE, Gatsonis CA, Glasziou PP, Irwig LM, Moher D, Rennie D, de Vet HC, Lijmer J, Standards for Reporting of Diagnostic Accuracy. The STARD statement for reporting studies of diagnostic accuracy: explanation and elaboration. *Annals of internal medicine*, 2003; 138(1): W1-12.
8. Rutjes AW, Reitsma JB, Coomarasamy A, Khan KS, Bossuyt P. Evaluation of diagnostic tests when there is no gold standard. A review of methods. *Health Technology Assessment*, 2007 11(50): iii, ix-51.
9. Sackett DL, Haynes RB. The architecture of diagnostic research. *British Medical Journal*, 2002; 324(7336): 539-41.
10. Altman DG, Vergouwe Y, Royston P, Moons KG. Prognosis and prognostic research: validating a prognostic model. *British Medical Journal*, 2009; 338(b605).
11. Ferrante di Ruffano L, Hyde CJ, McCaffery KJ, Bossuyt PM, Deeks JJ. Assessing the value of diagnostic tests: a framework for designing and evaluating trials. *British Medical Journal*, 2012; 344: e686.
12. Merlin T, Weston A, Toohar R. Extending an evidence hierarchy to include topics other than treatment: revising the Australian 'levels of evidence'. *BMC Medical Research Methodology*, 2009; 9: 34.
13. Schunemann H, Oxman A, Brozek J, Glasziou P, Jaeschke R, Vist G, Williams J, Kunz R, Craig J, Montori V, Bossuyt P, Guyatt G, GRADE Working Group. Grading quality of evidence and strength of recommendations for diagnostic tests and strategies. *British Medical Journal*, 2008; 336(7653): 1106-10.
14. OCEBM Levels of Evidence Working Group. *The Oxford 2011 Levels of Evidence*. 2011; Available from: <http://www.cebm.net/index.aspx?o=5653>.
15. National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), Pan Pacific Pressure Injury Alliance (PPPIA), Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. 2014: Emily Haesler (Ed.) Cambridge Media: Osborne Park, WA.
16. National Institute for Health and Care Excellence (NICE), NICE style guide: scientific and medical terms. 2017, NICE: <https://www.nice.org.uk/corporate/ecd1/chapter/scientific-and-medical-terms>.
17. Cochrane Style Manual Working Group, Cochrane Style Manual: Names and common terms. 2016, Cochrane Collaboration: http://community.cochrane.org/book_pdf/224.
18. Alonso-Coello P, Schünemann HJ, Moberg J, Brignardello-Petersen R, Akl EA, Davoli M, Treweek S, Mustafa RA, Rada G, Rosenbaum S, Morelli A, Guyatt GH, Oxman AD, GRADE Working Group. GRADE Evidence to Decision (EtD) frameworks: a systematic and transparent approach to making well informed healthcare choices. 1: Introduction. *BMJ*, 2016; 353: i2016.
19. Alonso-Coello P, Oxman AD, Moberg J, Brignardello-Petersen R, Akl EA, Davoli M, Treweek S, Mustafa RA, Vandvik PO, Meerpohl J, Guyatt GH, Schünemann HJ, GRADE Working Group. GRADE Evidence to Decision (EtD) frameworks: a systematic and transparent approach to making well informed healthcare choices. 2: Clinical practice guidelines. *BMJ*, 2016; 353: i2089.
20. NHMRC GAR consultants, NHMRC additional levels of evidence and grades for recommendations for developers of guidelines 2009, National Health and Medical Research Council Canberra.
21. Guyatt GH, Schunemann HJ, Djulbegovic B, Ak EA. Guideline panels should not GRADE good practice statements. *Journal of Clinical Epidemiology*, 2015; 68: 597-600.
22. Atkins D, Best D, Briss P, Eccles M, Falck-Ytter Y, Flottorp S, Guyatt G, Harbour R, Haugh M, Henry D, Hill S, Jaeschke R, Leng G, Liberati A, Magrini N, Mason J, Middleton P, Mrukowicz J, O'Connell D, Oxman A, Phillips B, Schünemann H, Edejer T, Varonen H, Vist G, Williams J, Zaza S, GRADE Working Group. Grading quality of evidence and strength of recommendations. *British Medical Journal*, 2004; 328(7454): 1490.
23. Guyatt G, Oxman A, Kunz R, Falck-Ytter Y, Vist G, Liberati A, Schünemann H, GRADE Working Group. Going from evidence to recommendations. *British Medical Journal*, 2008; 336(7652): 1049-51.
24. Jaeschke R, Guyatt G, Dellinger P, Schunemann H, Levy M, Kunz R, Norris S, Bion J, GRADE Working Group. Use of GRADE grid to reach decisions on clinical practice guidelines when consensus is elusive. *BMJ*, 2008; 337: a744.
25. Patient-Centered Outcomes Research Institute (PCORI), PCORI Methodology Standards. 2015, PCORI: <https://www.pcori.org/research-results/about-our-research/research-methodology/pcori-methodology-standards>.
26. Armstrong MJ, Mullins CD, Gronseth GS, Gagliardi AR. Recommendations for patient engagement in guideline development panels: A qualitative focus group study of guideline-naïve patients. *PLOS ONE*, 2017; e0174329.
27. Qaseem A, Forland F, Macbeth F, Ollenschlager G, Phillips SM, van der Wees P, et al. Guidelines International Network: toward international standards for clinical practice guidelines. *Ann Intern Med*, 2012; 156(7): 525-531

28. National Health and Medical Research Council (NHMRC), Statement on Consumer and Community Involvement in Health and Medical Research. 2016, Consumers Health Forum of Australia: https://www.nhmrc.gov.au/_files_nhmrc/file/publications/16298_nhmrc_-_statement_on_consumer_and_community_involvement_in_health_and_medical_research-accessible.pdf.
29. Légaré F, Boivin A, Trudy van der Weijden T, Christine Pakenham C, Burgers J, Légaré J, Sylvie St-Jacques S, Gagnon S. Patient and public involvement in clinical practice guidelines: A knowledge synthesis of Existing Programs. *Medical Decision Making*, 2011; 31(6).
30. Patient-Centered Outcomes Research Institute (PCORI), Engagement Rubric for Applicants. 2015, PCORI: <https://www.pcori.org/sites/default/files/Engagement-Rubric.pdf>.
31. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in guideline development: Surveying patients in 30 countries in 14th Guideline International Network (G-I-N) Conference. 2018: Manchester.
32. Haesler E, Cuddigan J, Kottner J, Carville K, Guideline Governance Group, International consumer engagement in pressure injury/ulcer guideline development: Global survey of patient care goals and information needs, in National Pressure Ulcer Advisory Panel 2019 Annual Conference. 2019: St Louis



www.internationalguideline.com