Prevention and Treatment of Pressure Ulcers: 
Wound Care and Pain Management –
an extract from the Clinical Practice Guideline
INTRODUCTION

Foreword

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

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

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

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

Suggested Citation

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

Limitations and Appropriate Use of This Guideline

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

Abstract

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

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

This extract focuses on the evidence presented treatment of pressure ulcers, with a focus on management of the wound. The extract includes chapters from the full Clinical Practice Guideline that discuss wound bed preparation, wound bed cleansing, debridement, management of infection and biofilm, wound dressings, biological dressings, growth factors and biophysical agents. The extract also includes a chapter that addresses pain management.
Strengths of Evidence and Strengths of Recommendations

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

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Guideline Website

http://www.internationalguideline.com

The guideline website will remain accessible during the interim period until the next guideline revision. The Quick Reference Guideline, sponsor acknowledgement, and supportive documents (e.g. data extraction tables) to the guideline are available from the website.
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INTRODUCTION

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Translation

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Stakeholders

Special thanks to the many stakeholders who reviewed the guideline processes and drafts. All stakeholder comments were reviewed by the Guideline Development Group and revisions were made based on the comments received. We appreciate the investment of health professionals, researchers, educators and manufacturers from all over the world who took time to share their expertise and thoughtful critique.
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INTERVENTIONS FOR TREATMENT OF PRESSURE ULCERS

WOUND BED PREPARATION

This section is a background to the discussion, evidence and recommendations presented in the next four guideline sections on cleansing; debridement; infection and biofilms; and wound dressings.

Wound bed preparation is a clinical concept encompassing a systematic and holistic approach to wound evaluation and treatment that promotes a wound environment that will allow normal progression toward 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

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, and
- Epithelial edge advancement.

Evaluating and optimizing each of these four components of wound care removes the barriers that are known to delay normal healing in chronic wounds.

Removing devitalized or necrotic tissue and its associated bacterial and cellular burden provides a stimulatory wound environment that promotes healthy tissue growth.2,4 The guideline sections Wound Care: Cleansing and Wound Care: Debridement provides comprehensive discussion and recommendations on cleansing techniques, appropriate use of debridement, selection of debridement techniques, and cautions to consider.

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.2,4 The section Assessment and Treatment of Infection and Biofilms from the full Clinical Practice Guideline provides further discussions and recommendations for clinical practice.

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.1,3 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.1 The guideline section Wound Dressings provides recommendations to guide practice.

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.3 Control of infection and inflammation; removal of cellular burden through debridement; and control of wound moisture are all important considerations in promoting epithelial advancement.1 Sequential monitoring of the advance of epithelium at the wound edge allows health professionals to assess the adequacy of wound bed preparation.

References


WOUND CARE: CLEANSING

Introduction

Wound cleansing is the process of using fluids to remove surface contaminants (debris), remnants of previous dressings and bacteria from the wound and peri-wound surface. Cleansing does not ‘sterilize’ a wound; it is ‘washing out’ a wound. If fibrinous material and detritus/debris cannot be removed gently with fluids, then debridement (i.e. removal of devitalized tissue) may be required (See Wound Care: Debridement section of the guideline).

Research on cleansing of pressure ulcers is sparse. There are no studies that could be found comparing cleansing versus not cleansing pressure ulcers. 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 ulcer wound bed to heal by removing surface debris and dressing remnants and allowing better wound visualization for assessment.

Cleansing must be extremely gentle in re-epithelializing pressure ulcers to prevent disruption of the neoepithelium. However, pressure ulcers with devitalized tissue or suspected biofilm usually require more aggressive use of irrigating solutions or debridement.

Comprehensive systematic reviews2,3 have identified no direct evidence to support the use of any specific wound cleansing solutions or wound cleansing techniques for pressure ulcers.

Recommendations

1. Cleanse the pressure ulcer at the time of each dressing change. (Strength of Evidence = C; Strength of Recommendation = )

   1.1. Cleanse most pressure ulcers with potable water (i.e., water suitable for drinking) or normal saline. (Strength of Evidence = C; Strength of Recommendation = )

   1.2. Consider using an aseptic technique when the individual, the wound or the wound healing environment is compromised. (Strength of Evidence = C; Strength of Recommendation = )

   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 appropriate4 (indirect evidence).

   For clean pressure ulcers (those with no debris or confirmed bacterial infection), potable (drinkable) tap water or normal saline is recommended. Boiled and cooled water is an effective wound cleansing solution if potable water or normal saline is not available (indirect evidence).5-7 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 (indirect evidence).1, 6, 8, 9

   1.3. Consider using cleansing solutions with surfactants and/or antimicrobials to clean pressure ulcers with debris, confirmed infection, suspected infection, or suspected high levels of bacterial colonization. (Strength of Evidence = C; Strength of Recommendation = )

   See the Assessment and Treatment of Infection and Biofilms section of the guideline for information on appropriate selection of topical antiseptics and cytotoxic profiles of different topical preparations.

   For dirty pressure ulcers (those with debris and/or high bacterial colonization), a cleansing solution with a surfactant and/or antimicrobial agent (antibiotic, antiseptic) appropriate for the wound and consistent with current toxicity/efficacy recommendations should be considered.
until the wound bed is clean. For pressure ulcers with suspected biofilm, debridement is the most effective management strategy (indirect evidence and consensus opinion). Avoid cleansing agents that are cytotoxic to fibroblasts or use them for only a short period of time to reduce bioburden. Cleansers that are formulated to remove fecal material (skin cleansers) are cytotoxic, and should not be used in wounds. When an antiseptic is added to a wound cleanser its toxicity increases, and the benefit of adding an antiseptic to wound cleanser has not been documented. 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.

1.4. Cleanse pressure ulcers with sinus tracts/tunneling/undermining with caution. (Strength of Evidence = C; Strength of Recommendation = )

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.

2. Apply cleansing solution with sufficient pressure to cleanse the wound without damaging tissue or driving bacteria into the wound. (Strength of Evidence = C; Strength of Recommendation = )

Pressure ulcer cleansing can be accomplished by irrigating the ulcer with fluid. In order to remove the debris in the ulcer, the force of the irrigation stream has to be greater than the adhesion forces holding the debris to the wound surface. Generally, irrigation pressure between four and 15 pounds per square inch (psi) should be adequate to clean the surface of the pressure ulcer without causing trauma to the wound bed. One way to produce pressurized irrigation is to deliver the irrigant from a syringe through a needle or catheter. For example, with a 19-gauge needle, the pressure generated with a 35ml syringe is 8 psi. There are also many commercially available irrigation devices.

2.1. Contain and properly dispose of used irrigation solution to reduce cross-contamination. (Strength of Evidence = C; Strength of Recommendation = )

Environmental contamination is possible with these devices, and infection-control precautions should be routinely used.

3. Cleanse surrounding skin. (Strength of Evidence = B; Strength of Recommendation = )

Periwound cleansing with normal saline caused a statistically significant decrease in wound and periwound microbial counts of pressure ulcers (17 ulcers at different locations) leading the authors to suggest that daily periwound cleansing was beneficial and should be a part of standard pressure ulcer wound care (Level 4 study). In a second study, Konya et al. (2005) compared cleansing of the periwound skin with normal saline (n = 84) to cleansing with a pH-balanced skin cleanser (n = 90). For pressure ulcers of all Category/Stage, healing time was shorter when 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). Lack of control for the increased potential for excreta in ulcers of the sacrum, ischial tuberosities and coccyx may have influenced the findings (Level 3 study).

References

Introduction

Despite comprehensive literature reviews, very little direct evidence (i.e., studies of debridement of pressure ulcers in humans) was identified to support these recommendations. As many as ten clinical practice guidelines support the expert opinion statements in this section. Limited direct evidence and indirect evidence (i.e., studies of debridement in other types of wounds) are included.

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. In fact, prior to Steed’s 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. Steed’s 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.

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.

Recommendations

1. Debride devitalized tissue within the wound bed or edge of pressure ulcers when appropriate to the individual’s condition and consistent with overall goals of care. (Strength of Evidence = C; Strength of Recommendation = △△)

   Caution: Debridement should only be performed when there is adequate perfusion to the wound (refer to Recommendation 9).

   Devitalized tissue is tissue that 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.

   Necrotic tissue is a nidus for infection, prolonging the inflammatory response, mechanically obstructing contraction, and impeding re-epithelialization. It may mask underlying fluid collections or abscesses and limit full assessment capability in determining ulcer depth. If appropriate to the individual’s condition and consistent with overall goals of care, a thorough initial debridement of the pressure ulcer and the hyperproliferative epithelial edge should be performed to elicit an acute wound-healing response. Maintenance debridement should follow as dictated by the ulcer bed condition.

   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 manner in which it should be accomplished.

2. Debride the wound bed when the presence of biofilm is suspected or confirmed. (Strength of Evidence = C; Strength of Recommendation = △△)

   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. See the Assessment and Treatment of Infection and Biofilms section for more information.

   Wolcott et al. (2010) demonstrated in invitro models and a small scale clinical study that less mature biofilm is more susceptible to topical antimicrobial treatment. Invitro models demonstrated that
biofilm develops tolerance to antimicrobial treatment within 24 to 96 hours and suggests 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 antibiotic therapy between 24 hours and 48 hours post-debridement. By 72 hours, susceptibility had returned to that of mature biofilm samples (indirect evidence).

3. Select the debridement method(s) most appropriate to the individual, the wound bed, and the clinical setting. (Strength of Evidence = C; Strength of Recommendation = □□□)

The most common methods used for debriding pressure ulcers are:
- surgical/sharp,
- conservative sharp,
- autolytic,
- enzymatic,
- larval, and
- mechanical (including ultrasound and hydrosurgical).

**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. 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. 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.

Surgical debridement is most appropriate when there is an urgent need to remove extensive, devitalized tissue. A pressure ulcer 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 ulcer.

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. A non-randomized study comparing hydrosurgery to hydrogel therapy 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 hydrogel in chronic venous leg ulcers (indirect evidence). 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 group (indirect evidence).

**Conservative sharp debridement** employs the use of scalpels, curettes, scissors, forceps, and rongeurs to remove devitalized tissue without pain or bleeding. This method of debridement decreases wound surface bacterial burden and removes senescent cells, converting a chronic wound into an acute wound.

Surgical/sharp and conservative sharp debridement should only be performed in anatomical locations possessing adequate vascularity to support the ability to heal. Knowledge of anatomy and training is vital for a person using sharp debridement techniques. Caution must be exercised with immunocompromised individuals to avoid large open cavities that may serve as portals for opportunistic infection. Additionally, caution must be exerted in those individuals with bleeding disorders and those taking anticoagulants. Access to conservative sharp debridement may be limited in certain care settings.

Saap et al. (2002) developed and tested a Debridement Performance Index (DPI) scoring system. The DPI addresses three parameters: removal of callus; removal of an ulcer’s edge; undermining; and removal of wound bed necrotic or infected tissue. Each parameter is then scored (0 = debridement needed, not performed; 1 = debridement needed and performed; 2 = debridement not needed and
not performed). Using digital images from diabetic foot ulcers previously enrolled in a controlled, randomized bioengineered skin construct clinical trial where the observer was blinded to treatment, Saap et al. (2002)\(^7\) reported that the lower the DPI, the lower the incidence of wound closure. Although the DPI scoring system for debridement performance appears to be a promising predictive tool for determining clinical diabetic foot healing outcomes, a lack of follow up refinement and testing of the tool has precluded its adoption into clinical practice to date (indirect evidence).

Golinko et al. (2009)\(^29\) concur that surgical debridement should be performed until all devitalized tissue is excised. Their retrospective study conducted on pressure ulcers suggested that histopathological analysis of tissue excised during surgical debridement can be used to determine the adequacy of the debridement as visual inspection of tissue alone is 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 5 study).

Williams et al. (2005)\(^9\) in a non-randomized pilot study of individuals with chronic venous leg ulcers, reported that individuals receiving sharp circulator curette debridement exhibited significant healing at four weeks post-debridement when compared to those who did not receive conservative sharp debridement as measured by a decrease in ulcer mean surface area. But 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.

**Autolysis** is a highly selective form of slow debridement occurring naturally in all wound types.\(^19\) 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.\(^19\) The aim is to regulate the wound environment to achieve optimal moisture, pH and humidity in order that autolysis will occur (indirect clinical evidence).

Moisture-retentive 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.\(^20\) In heavily exudating 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\(^30,31\) (Level 2 studies). This suggests that no specific type of amorphous hydrogel is superior to another for achieving autolysis.

In two 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 (2000)\(^32\) reported no difference in healing between collagenase and hydrocolloid use (Level 2 study), while Muller et al. (2001)\(^33\) 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 ulcers (Level 2 study). It is important to note that in Muller’s study, surgical debridement was performed prior to subject randomization. Among individuals with Category/Stage III pressure ulcers, Burgos (2000)\(^32\) 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.

Autolytic debridement is contraindicated in the presence of untreated infection or extensive necrotic tissue, in large ulcers with undermining and sinus tracts, and in individuals with compromised immunity.\(^7,11,12,14-16,21\)

**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.\(^20\) 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.\textsuperscript{19} Bacterial collagenase degrades native collagen with great specificity, yet is not active against keratin, fat, or fibrin.\textsuperscript{19} 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.\textsuperscript{19} Heavy metals may inactivate some enzymes. Follow manufacturer’s directions when using enzymatic debriding agents.

In a RCT (n = 28) comparing papain-urea to collagenase for debriding Category/Stage II to IV pressure ulcers, there was a significantly greater reduction in devitalized tissue (p < 0.0167) and significantly greater amount of granulation (p < 0.0167) for those receiving papain-urea, but the ulcer healing rates were not different (p > 0.05) between groups\textsuperscript{34} (Level 2 study). In a double-blind RCT (n = 135 included, n = 78 results analyzed) comparing collagenase to fibrinolysin/deoxyribonuclease for debriding Category/Stage II to IV pressure ulcers no significant difference (p = 0.164) was found between the two groups for the reduction of devitalized tissue\textsuperscript{35} (Level 2 study). 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 ulcers in individuals in a long term care setting. Approximately 85% of the pressure ulcers managed with collagenase achieved complete debridement at 42 days compared with 29% of those treated with hydrogel wound dressing (p < 0.03). The pressure ulcers debrided with collagenase were also statistically more likely to have achieved complete wound closure within 84 days (69% versus 21%, p = 0.02)\textsuperscript{36} (Level 2 study).

**Mechanical debridement** is often a non-selective form of debridement that can result in the removal of both devitalized as well as viable tissue.\textsuperscript{20} Examples of mechanical debriding agents include:

- wet-to-dry dressings,
- monofilament fiber pads,
- wound irrigation,
- low frequency ultrasound, and
- ultrasonic mist.

Wet-to-dry gauze dressings can be painful, and may remove healthy tissue. 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.\textsuperscript{37, 38} A monofilament fiber pad removes slough and devitalized tissue, and potentially disrupts biofilm within the wound bed; however, more research is required on its effectiveness in promoting wound healing.\textsuperscript{39}

Noncontact low frequency ultrasound (ultrasonic mist) debridement is increasingly being used to remove devitalized tissue. This selective method uses low frequency ultrasound electrical currents converted to mechanical vibrations that stimulate a probe that in turn amplifies the vibrations, converting them into acoustic energy that is transferred to the wound tissue. Ultrasound debridement provides both mechanical and hydrodynamic effects directly in the wound bed due to cavitation.\textsuperscript{18} The application of ultrasound causes creation and destruction of small bubbles in fluid that expand and rapidly collapse (‘imploding gaps’) resulting in turbulent shockwaves and currents that lead to erosion of necrotic tissue and fibrin. Further information on irrigation, whirlpool, and (noncontact low frequency ultrasound is the guideline sections *Wound Care: Cleansing* and *Biophysical Agents in Pressure Ulcer Treatment*.

**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.\textsuperscript{15, 20} 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.\textsuperscript{20, 40}
In a clinical series of individuals with pressure ulcers treated with larval therapy compared with conventional debridement, faster debridement and granulation tissue formation was reported in those treated with maggots (Level 2 study).

4. **Use mechanical, autolytic, enzymatic, and/or biological methods of debridement when there is no urgent clinical need for drainage or removal of devitalized tissue.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

   This recommendation is supported by expert opinion from nine clinical practice guidelines.1, 2, 11-17, 21

5. **Surgical/sharp debridement is recommended in the presence of extensive necrosis, advancing cellulitis, crepitus, fluctuance, and/or sepsis secondary to ulcer-related infection.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

   This recommendation is supported by expert opinion from ten clinical practice guidelines.1, 2, 11-17, 21

6. **Conservative sharp debridement and surgical/sharp debridement must be performed by specially trained, competent, qualified, and licensed health professionals consistent with local legal and regulatory statutes.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

   It is vital that health professionals who perform conservative sharp debridement or surgical/sharp debridement possess knowledge of anatomy and adequate training and experience.11, 15, 17

7. **Use sterile instruments for conservative sharp and surgical/sharp debridement.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

   This recommendation is supported by expert opinion. Although clean dressings may be appropriate for pressure ulcer management,2 the instruments being used for conservative sharp or surgical/sharp debridement should be sterile.1

8. **Use conservative sharp debridement with caution in the presence of:**
   - immune incompetence,
   - compromised vascular supply, or
   - lack of antibacterial coverage in systemic sepsis (Strength of Evidence = C; Strength of Recommendation = ★★★).

   *Caution: Relative contraindications include anticoagulant therapy and bleeding disorders.*

   This recommendation is supported by expert opinion from ten clinical practice guidelines.1, 2, 11-17, 21

9. **Refer individuals with Category/Stage III or IV pressure ulcers 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.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

   This recommendation is supported by expert opinion from nine clinical practice guidelines.1, 2, 11, 12, 14-17, 21

10. **Manage pain associated with debridement.** (Strength of Evidence = C; Strength of Recommendation = ★★★)

    Surgical, conservative sharp, enzymatic, biological and mechanical forms of debridement may result in pain (Refer to *Pain Assessment and Treatment* section of this guideline.) This recommendation is supported by expert opinion from ten clinical practice guidelines.1, 2, 11-17, 21

11. **Perform a thorough vascular assessment prior to debridement of lower extremity pressure ulcers to determine whether arterial status/supply is sufficient to support healing of the debrided wound.** (Strength of Evidence = C; Strength of Recommendation = ★★★)
Surgical/sharp and conservative sharp debridement of avascular tissue should only be performed after adequate perfusion has been established. This recommendation is supported by expert opinion from two clinical practice guidelines.\(^1\)\(^1\) When vascular correction is impossible, the decision on whether to debride should be made between the patient and vascular or wound specialist, in consideration of risks and benefits.

12. Do not debride stable, hard, dry eschar in ischemic limbs. (Strength of Evidence = C; Strength of Recommendation = \(\heartsuit\))

Shannon (2013)\(^42\) conducted a retrospective review in a nursing home population of heel pressure ulcers \((n = 179)\) with entire eschar \((67.8\% \text{ of the sample})\) or blister coverage \((31.8\% \text{ of the sample})\). Of the 155 patients not lost to follow up, 154 of the wounds \((99.3\%)\) healed. Of the heel pressure ulcers covered with eschar, 100\% of wounds healed with an average healing time of 11 weeks \((\text{range 2 to 50 weeks})\). Complications included one patient who developed osteomyelitis \((\text{with eventual healing})\) and two cases of cellulitis and one eventual amputation in a patient with blister coverage of the ulcer \((\text{Level 5 study})\).

12.1. Assess stable, hard, dry eschar at each wound dressing change and as clinically indicated. (Strength of Evidence = C; Strength of Recommendation = \(\heartsuit\)\(\heartsuit\))

Assessment of an ulcer 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 \((\text{i.e., signs of infection})\) in the area around the dressing.

12.2. Consult a medical practitioner/vascular surgeon urgently in the presence of the above symptoms. (Strength of Evidence = C; Strength of Recommendation = \(\heartsuit\))

12.3. Debride the pressure ulcer urgently in the presence of the above symptoms \((\text{i.e, erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodour})\). (Strength of Evidence = C; Strength of Recommendation = \(\heartsuit\))

Supporting expert opinion is expressed in five clinical practice guidelines.\(^1\)\(^,\)\(^2\)\(^,\)\(^12\)\(^,\)\(^14\)\(^,\)\(^15\)

13. Perform maintenance debridement on a pressure ulcer until the wound bed is free of devitalized tissue and covered with granulation tissue. (Strength of Evidence = C; Strength of Recommendation = \(\heartsuit\))

Maintenance debridement is ongoing debridement to help maintain the wound 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 \((\text{biofilms})\) and senescent fibroblasts, allowing a stimulatory environment to be established\(^4\)\(^,\)\(^5\)\(^,\)\(^10\)\(^,\)\(^11\)\(^,\)\(^19\) \((\text{indirect evidence})\). The need for debridement is determined by both clinical parameters and the need to achieve optimal wound bed preparation. In ulcers that appear healthy but do not show evidence of closure, maintenance debridement is indicated.\(^43\)

While acute wounds may only require an initial debridement \((\text{if at all})\) chronic wounds often require maintenance debridement of the base as well as the non-migratory hyperproliferative epithelial edge.\(^4\)\(^,\)\(^5\)\(^,\)\(^10\)\(^,\)\(^11\)\(^,\)\(^19\) \(\text{Continue maintenance debridement until the wound bed is free of devitalized tissue, covered with granulation tissue and progressing towards healing. Maintenance debridement should be resumed in the case of delayed wound healing that suggests presence of biofilm}^{18,22} \text{or with the return of any devitalized tissue or deteriorating granulation tissue (indirect evidence).}\)
References

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ASSESSMENT AND TREATMENT OF INFECTION AND BIOFILMS

Introduction

Bacteria are present on all skin surfaces. When the primary defense provided by intact skin is lost, bacteria will reside on the wound surface. When the bacteria (by numbers or virulence in relation to host resistance) cause damage to the body, infection is present. An impaired host has a reduced ability to combat bacteria. The number of bacteria and their effect on the host can be categorized as:

- contamination,
- colonization,
- critical colonization/topical infection,
- local infection,
- regional spreading infection/cellulitis and
- sepsis.

Sometimes, microorganisms multiply, invade, and damage tissues, delaying healing, and often cause systemic responses. Infection is present when bacteria present in an ulcer impair wound healing.¹

Pressure ulcers are a consequence of ischemia and are more susceptible to the development of infection² as the tissue does not receive normal nutrition, oxygen, immune cells, antibodies, and antibiotics. In addition, risk factors for pressure ulcer development (e.g., protein calorie malnutrition) compromise the host’s defenses. Infection is not common in Category/Stage I or II pressure ulcers, and assessment of infection should focus on Category/Stage III and IV ulcers³ and unstageable pressure ulcers.

Wound infection may be associated with biofilms. Bacterial biofilms are extremely common in the natural environment. They are known to cause chronic inflammation that contributes to the molecular pathologies of many diseases, including periodontal disease, surgical device infections, urinary catheter infections, cystic fibrosis, chronic otitis media, and contact lens associated corneal infections.⁴ Compared to planktonic (free-floating) bacteria, bacteria in biofilms have enhanced resistance to endogenous antibodies and phagocytic cells, as well as by exogenous antibiotics and antiseptics. Approximately 60% of chronic skin wounds contain bacterial biofilms,⁵,⁶ which suggests that biofilms play important roles in maintaining a chronic inflammation state that ultimately leads to the failure of skin wounds to heal. The terms ‘critical colonization’ and ‘localized infection’, which were created to describe wounds that fail to heal even with only low numbers of planktonic bacteria (≤ 10⁵ CFU/gm), may actually be describing wounds that have biofilms. Removal of biofilms by debridement, and prevention of reformation of biofilms using topical antiseptics or antimicrobial dressings may be the optimal treatment to move chronic wounds out of a chronic inflammatory phase and into a healing repair phase.⁷,⁸ Further study is needed.

System Consideration

1. Follow local infection control policies to prevent self-contamination and cross-contamination in individuals with pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ☑️)
Assessment of High Risk Individuals with Pressure Ulcers

1. Have a high index of suspicion of local infection in a pressure ulcer in the presence of:
   - lack of signs of healing for two weeks;
   - friable granulation tissue;
   - malodor;
   - increased pain in the ulcer;
   - increased heat in the tissue around the ulcer;
   - increased drainage from the wound;
   - an ominous change in the nature of the wound drainage (e.g., new onset of bloody drainage, purulent drainage);
   - increased necrotic tissue in the wound bed; and/or
   - pocketing or bridging in the wound bed. (Strength of Evidence = B; Strength of Recommendation =  )

Wound healing is delayed and/or may be abnormal when pressure ulcers have significant bacterial burden and infection. Bridging is the presence of strands of tissue bridging across the ulcer. Pocketing occurs when granulation tissue is not uniform, or heals from the bottom up to the top. These undulating pockets of open tissue can harbor bacteria (indirect evidence).9

Cutting et al. (1994)10 developed criteria to identify infection in granulating wounds of mixed etiology based on a review of the literature. They categorized their characteristics of infected wounds as traditional criteria; that is, cellulitis, abscess and wound discharge (serous, seropurulent, hemopurulent and pus), as well as additional criteria including discoloration, delayed healing, friable granulation, pain and tenderness, malodor, pocketing and bridging. These criteria were later tested in an observational study investigating application of the criteria to wounds by registered nurses in the clinical setting11 and a Delphi process.12

Gardner et al. (2001)13 reported on the validity of 12 clinical signs and symptoms of chronic wound infection (i.e., pain, erythema, edema, heat, and purulent exudates) and those specific to open chronic wounds (i.e., serous drainage with concurrent inflammation, delayed healing, discoloration of granulation tissue, friable granulation tissue, pocketing at the base of the wound, malodor and wound breakdown) in a mixture of chronic wounds that included 19 pressure ulcers, three of which were infected. Wounds were assessed by health professionals blinded to wound biopsy and culture results. The most sensitive measures of infection were delayed healing and friable granulation tissue, with a sensitivity of 0.81 (specificity of 0.64). Over 80% of infected ulcers had these signs. Increasing pain, malodor and heat also had specificity over 0.80. Ulcers that were not infected did not have these signs. All (100%) the wounds that had increasing pain or wound breakdown were clinically infected (Level 2 study).

2. Have a high index of suspicion for the likelihood of infection in pressure ulcers that:
   - have necrotic tissue or a foreign body present;
   - have been present for a long period of time;
   - are large in size or deep; and/or
   - are likely to be repetitively contaminated (e.g., near the anus). (Strength of Evidence = C; Strength of Recommendation = )

Factors within the pressure ulcer can increase the risk of infection. Necrotic tissue contains high levels of both anaerobic and aerobic bacteria, and in greater density than nonnecrotic ulcers have.14, 15 Tarnuzzer et al. (1996)16 have suggested that bacterial colonization in chronic wounds elevates proinflammatory cytokines, such as interleukin-1 and tumor necrosis factor. This condition 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. Fecal matter contains high levels of bacteria, which can create a heavy bacterial burden in the wound bed.17
In a retrospective study of surgical samples in infected pressure ulcers, the predominant organisms were *Enterobacter* (29%), *Staphylococci* (28%), and *Enterococcus faecalis* (16%).18 A cross-sectional prevalence study in Spain reported the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) colonization in pressure ulcers of 1377 participants from nine long term care facilities was 59%.19

3. **Have a high index of suspicion for local wound infection in individuals with:**
   - diabetes mellitus,
   - protein-calorie malnutrition,
   - hypoxia or poor tissue perfusion,
   - autoimmune disease, or
   - immunosuppression. (Strength of Evidence = B; Strength of Recommendation = )

The immune response to bacterial infection of individuals with compromised host defenses is less robust than normal. The majority of Category/Stage III and IV pressure ulcers occur in older adults, who often have many co-morbidities that increase the risk of pressure ulcer development and simultaneously impair healing. Gardner et al. (2001)13 studied the signs of infection in 19 pressure ulcers known to be infected, as confirmed by quantitative tissue culture. Mean tissue oxygen levels were lower in ulcers with infection (Level 5 study). Tissue ischemia has also been found to be closely linked to postoperative20 and diabetic foot wound infection.21

4. **Have a high index of suspicion of biofilm in a pressure ulcer that:**
   - has been present for more than 4 weeks;
   - lacks signs of any healing in the previous 2 weeks;
   - displays clinical signs and symptoms of inflammation;
   - does not respond to antimicrobial therapy. (Strength of Evidence = C; Strength of Recommendation = )

This statement is based on consensus opinion. Biofilm is associated with impaired epithelialization and formation of granulation. When a pressure ulcer has delayed healing (i.e., has been present for four weeks or longer), exhibits clinical signs and symptoms of inflammation and fails to heal despite a standard wound management plan that promotes moist wound healing and/or it does not respond to antimicrobial therapy, the presence of biofilm should be suspected (indirect evidence).8, 22-24 In a small diagnostic study including 15 chronic wounds, 60% of the sample had biofilm identified using epifluorescence microscopy. Of these pressure ulcers, all were of greater than four weeks duration and the mean duration was 108 weeks. Only one third of the pressure ulcers showed signs and symptoms of infection6 (indirect evidence).

There is currently no confirmed noninvasive, macroscopic method through which presence of biofilm can be visibly identified within the wound bed.8, 22-24 The validity of the visual presence in the wound bed of a visible, translucent, thick, slimy film that may be pale yellow or green as a clinical indicator of biofilm is currently debated.22, 25, 26

In one observational study (n = 24 enrolled, n = 16 completed study) macroscopic identification of biofilm was used to assess wound healing associated with a biofilm based wound management plan (debridement and application of polyhexanide and betaine [PHMB]). The researchers defined presence of a shiny, translucent, slimy layer on a non-healing wound surface as indicative of biofilm, and reported statistically significant reduction in macroscopic biofilm over 24 weeks associated with the treatment regimen (61.8 ± 34.6% versus 22.6 ± 36.0% of wound bed, p < 0.01). Although a recognized biofilm based wound management strategy reduced the macroscopic presence of the slime layer, there was no microscopic confirmation that this visual characteristic was attributable to biofilm27 (indirect evidence).

In a case series (n = 9) the researchers reported the presence of a thick, opaque film in chronic wounds with clinical signs of infection, particularly local inflammation. The presence of bacterial burden was confirmed via laboratory swabs in five of the cases. In all cases, reduction or complete eradication of
the macroscopic film was only successful with regular wound debridement, and when this was achieved the wounds progressed to healing. Once again, although reduction of the visual film was achieved using a biofilm based wound management strategy, microscopic diagnosis of biofilm was not made (indirect evidence). Further clinical studies are required to confirm whether experienced clinicians are able to visually identify the presence of wound biofilm.

Diagnosis of Infection

1. Consider a diagnosis of spreading acute infection if the pressure ulcer has local and/or systemic signs of acute infection, such as:
   - erythema extending from the ulcer edge;
   - induration;
   - new or increasing pain or warmth;
   - purulent drainage;
   - increase in size;
   - crepitus, fluctuance, or discoloration in the surrounding skin;
   - fever, malaise, and lymph node enlargement; or
   - confusion/delirium and anorexia (particularly in older adults). (Strength of Evidence = C; Strength of Recommendation = \( \mathbb{2} \mathbb{2} \))

Chronic ulcers can develop into acute spreading infection. There is a more classic appearance of the individual and the wound when acute infection is present. Older adults often do not develop the usual signs of infection; rather, they may develop confusion or delirium, lose general function and become anorexic.

2. Determine the bacterial bioburden of the pressure ulcer by tissue biopsy or quantitative swab technique. (Strength of Evidence = B; Strength of Recommendation = \( \mathbb{C} \))

In the absence of clinical signs of infection, 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 swabs will only reveal the colonizing organism, and may not reflect deeper tissue infection.

In one study, superficial swabs from pressure ulcers (n = 72) were positive for 96% of ulcers tested, whereas the deep tissue aspirates were positive in only 43% of ulcers and deep tissue biopsies were positive in 63% of the same ulcers. Of the 43 pressure ulcers 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 biopsy (Level 2 study).

An acceptable alternative to quantitative tissue culture is the Levine quantitative swab technique (described below). Sapico et al. (1986) compared findings from quantitative and swab cultures of pressure ulcers (n = 25) and reported a mean concordance of 74.5%. The concordance between the central and peripheral portions of the ulcer was 63%, indicating that there is some variability in findings based on the location of the sample (Level 2 study). Bill et al. (2001) reported a 69% concordance between quantitative biopsy and quantitative swab cultures in 39 ulcers, not including pressure ulcers (indirect evidence).
Procedure for performing quantitative swab cultures (Levine method)

- Cleanse wound with normal saline.
- Remove/debride nonviable tissue.
- Wait two to five minutes.
- If ulcer is dry, moisten swab with sterile normal saline.
- Culture the healthiest looking tissue in the wound bed.
- Do not culture exudate, pus, eschar, or heavily fibrous tissue.
- Rotate the end of a sterile alginate-tipped applicator over a 1cm² area for 5 seconds.
- 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.

2.1. Consider using tissue biopsy and microscopy to determine the presence of biofilm. (Strength of Evidence = C; Strength of Recommendation = )

The current gold standard for confirmation of presence of biofilm is microscopic examination using light microscopy, epifluorescence microscopy or scanning electronic microscopy (SEM). However, the value and cost effectiveness of these techniques in routine clinical management of pressure ulcers is yet to be demonstrated.

In one diagnostic study, wedge tissue biopsies from chronic wounds (n = 15, n = 5 were pressure ulcers) 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 (indirect evidence). Similarly, in a study in which culture analysis, light microscopy and SEM were used to assess 37 chronic wounds of mixed etiology (n = 21 pressure ulcers), culture identified eight frequently observed bacterial species compared with 15 frequently occurring species identified used microscopy. Sixty percent of the sample contained biofilm (indirect evidence).

3. Consider a diagnosis of pressure ulcer infection if the culture results indicate bacterial bioburden of ≥ 10⁵ CFU/g of tissue and/or the presence of beta hemolytic streptococci. (Strength of Evidence = B; Strength of Recommendation = )

Wound infection occurs when the virulence factors of one or more wound organisms overwhelm the host’s resistance, resulting in invasion and replication of the organism or production of toxin and local tissue damage. Daltrey et al. (1981) demonstrated that a microbial load greater than 10⁵ organisms per gram of tissue is the critical level for diagnosing infection. Bendy et al. (1964) showed that significant healing of pressure ulcers occurred only when bacterial counts were less than 10⁵. The 10⁵ guideline has been questioned based on the assertion that the interactions among specific types of pathogens may be more important than microbial load in promoting bacterial growth and infection. Supporting this assertion is microbiological evidence that chronic leg wounds contain multiple species of microbial organisms and that those that contain four or more different species have poor healing outcomes. Nonetheless, it is unclear which organisms represent a definitive threat to the wound environment or which interact with others in a synergistic manner.

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Treatment

1. Optimize the host response by:
   - evaluating nutritional status and addressing deficits;
   - stabilizing glycemic control;
   - improving arterial blood flow; and/or
   - reducing immunosuppressant therapy if possible. (Strength of Evidence = C; Strength of Recommendation = ★★★)

   Many systemic factors contribute to the development of pressure ulcers. If these same factors can be improved, the individual’s intrinsic ability to fight infection can usually also be improved. Review the individual’s nutritional intake, modify it if needed and stabilize diabetic glycemic control (see Nutrition for Prevention and Treatment of Pressure Ulcers section from the full Clinical Practice Guideline). Assess arterial blood supply to the wound and institute 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 doses of immunosuppressive agents.

2. Prevent contamination of the pressure ulcer. (Strength of Evidence = C; Strength of Recommendation = ★★★)

   Pressure ulcers near the anus are subject to contamination, especially by bacteria from the colon. Predominant organisms in infected pressure ulcers included Enterobacter species, Proteus species, Escherichia coli, and Enterococcus faecalis. Meticulous skin cleansing and use of dressings or topical agents to prevent exposure to fecal matter are needed. At times, bowel management systems and diversion of ostomies are required due to continuous exposure of the ulcer to feces. The Preventive Skin Care section from the full Clinical Practice Guideline provides further recommendations on continence management.

3. Reduce bacterial load and biofilm in the pressure ulcer as outlined in the Wound Care: Cleansing and Wound Care: Debridement sections. (Strength of Evidence = C; Strength of Recommendation = ★★★)

   Necrotic tissue and slough promote bacterial growth (see section Wound Care: Cleansing, Wound Care: Debridement, and the section Surgery for Pressure Ulcers from the full Clinical Practice Guideline for associated recommendations). Cleansing removes loose debris and planktonic (free-floating) bacteria. Debridement is often required to remove adherent slough and eschar.

   Debridement physically disrupts biofilm growth, providing a window of opportunity in which topical antiseptics can be used more effectively. Once removed, biofilms tend to redevelop. Maintenance debridement should be continued in conjunction with topical antiseptic therapy until the pressure ulcer is clear of biofilm (indirect evidence).

4. Consider the use of tissue appropriate strength, non-toxic topical antiseptics for a limited time period to control bacterial bioburden. (Strength of Evidence = C; Strength of Recommendation = ★★★)

   Warning: Hydrogen peroxide is highly toxic to tissues even at low concentrations and should not be used as a preferred topical antiseptic. Its use should be totally avoided in cavity wounds due to the risk of surgical emphysema and gas embolus.

   Caution: Iodine products should be avoided in patients with impaired renal failure, history of thyroid disorders or known iodine sensitivity. Sodium hypochlorite (Dakin’s solution) is cytotoxic at all concentrations and should be used with caution, at concentrations no greater than 0.025%, for short periods only when no other appropriate option is available. There is a risk of acidosis when acetic acid is used for extended periods over large wound surface areas.
Antiseptics are agents that destroy or inhibit the growth and development 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 even prions. Resistance to antiseptics does develop. Antiseptics commonly used in wounds include:

- iodine compounds (povidone iodine and slow-release cadexomer iodine),
- silver compounds (including silver sulfadiazine),
- polyhexanide and betaine (PHMB),
- chlorhexidine,
- sodium hypochlorite, and
- acetic acid.

Cytotoxicity is the main concern when applying a topical agent on an open wound. Antiseptics have been found, primarily using invitro models, to be cytotoxic to cells essential to the wound healing process, including fibroblasts, keratinocytes, and leukocytes. However, this cytotoxicity appears to be concentration dependent, as several antiseptics in low concentrations are not cytotoxic, although they retain their antibacterial activity invitro. Care should be taken to protect the periwound area from topical antiseptics and to manage pain associated with application.

When there is a delay in wound healing due to suspected infection antimicrobial use overrides the risk of antiseptic toxicity. 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.

Povidone iodine and cadexomer iodine are low cost topical antiseptic options. Invitro studies have found povidone iodine to be toxic to granulocytes in concentrations above 0.05%; 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 (indirect evidence). See the guideline section on Wound Dressings for Treatment of Pressure Ulcers for evidence on cadexomer iodine dressings.

Sodium hypochlorite (Dakin’s solution) appears to have only short lived (up to 24 hours) antibacterial properties and there is conflicting evidence on its toxicity to skin cells. Lineaweaver et al. (1985) demonstrated there is no concentration of acetic acid that is toxic to bacteria whilst preserving fibroblasts. Its short term use in low concentrations (no greater than 0.025%) should only be considered in the absence of other appropriate topical antiseptics (indirect evidence).

One small (n = 30), randomized controlled trial (RCT) conducted by Wild et al. (2012) compared topical cleansing with PHMB to normal saline for treating pressure ulcers (half of which were Category/Stage IV pressure ulcers) with known MRSA colonization. After 14 days of treatment, significantly more pressure ulcers in the PHMB group were eradicated of MRSA compared with the control group (100% versus 66.67%, p < 0.05) (Level 2 study). Invitro studies support the use of PHMB in concentrations up to 2% as a topical antiseptic for managing Pseudomonas species and S. aureus (indirect evidence).

Dilute acetic acid may be of benefit in pressure ulcers infected with Pseudomonas species (indirect evidence).

5. **Consider the use of topical antiseptics in conjunction with maintenance debridement to control and eradicate suspected biofilm in wounds with delayed healing.** (Strength of Evidence = C; Strength of Recommendation = )

Wolcott et al. (2010) demonstrated in invitro models and a small scale clinical study that less mature biofilm is more susceptible to topical antimicrobial treatment. Invitro 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).
**Invitro** studies supports the notion that biofilm develops resistance to topical antiseptics as it matures. Numerous studies have demonstrated susceptibility of immature (three days) *S. aureus*, *S. epidermidis* and *P. aeruginosa* biofilm to povidone iodine in concentrations of 1% to 10% and to cadexomer iodine paste. Silver sulfadiazine has also been demonstrated to reduce, but not eradicate, colonies of immature biofilm in *invitro* studies and has been less successful in reducing mature biofilm colonies. A comparison between iodophors and silver suggested iodophors have a greater role in managing biofilm (indirect evidence).

A small, uncontrolled study conducted in 16 chronic wounds of mixed etiology described as having macroscopic evidence of biofilm that were managed with 0.3% PHMB impregnated wound dressing showed significant increase in granulation of the wound bed (p < 0.04) after 24 weeks of treatment. Seventy-five percent of the wound achieved complete healing (indirect evidence).

6. Consider the use of topical antiseptics for pressure ulcers that are not expected to heal and are critically colonized/topically infected. (Strength of Evidence = C; Strength of Recommendation = )

Critically colonized or topically infected pressure ulcers are those in which bacteria are present in the tissue, resulting in delayed healing, malodor, and increased exudate from the ulcer. Recommended strength antiseptics can be used for maintenance ulcers (i.e., those that are not expected to heal) to control bioburden and to reduce inflammation in the ulcer and surrounding skin. See the **Special Populations: Individuals In Palliative Care** section from the full **Clinical Practice Guideline** for discussion on wound care in individuals whose wound is not expected to heal.

7. Consider use of silver sulfadiazine in heavily contaminated or infected pressure ulcers until definitive debridement is accomplished. (Strength of Evidence = C; Strength of Recommendation = )

**Caution:** 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.

Topical antimicrobial silver offers broad antimicrobial coverage. There is evidence on the use of silver for wound care; however, the majority of studies investigating the use of silver involved burn wounds, leg ulcers, or animal models, and therefore are not directly applicable to pressure ulcers in humans. Strains of bacteria resistant to silver may be emerging (indirect evidence). Silver impregnated dressings are discussed in detail in the **Wound Dressings for Treatment of Pressure Ulcers** section of the guideline.

8. Consider the use of medical-grade honey in heavily contaminated or infected pressure ulcers until definitive debridement is accomplished. (Strength of Evidence = C; Strength of Recommendation = )

**Caution:** Before applying a honey dressing, ensure the individual is not allergic to honey. Individuals who have bee or bee stings allergies are usually able to use properly irradiated honey products.

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 the leg, Fournier’s gangrene, and other skin infections. Because honey produces an alternative product for bacterial metabolism that yields lactic acid rather than ammonia, amines, and sulfur (which are odorous) wound odor is reduced. However, no significant research exists on the bactericidal effects of medical-grade honey and the specific bacteria that may be eradicated with honey.

A recent Cochrane review identified one small (n = 40) RCT comparing medical-grade honey to saline-soaked gauze for healing Category/Stage I and II pressure ulcers. 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 ulcers in this study were described as uninfected at baseline (Level 2 study). Biglari et al. (2012) reported a case series of 20 individuals
with spinal cord injury (SCI) and Category/Stage III or IV pressure ulcers that were treated with Medihoney®. After one week of daily cleansing with Ringer’s solution and application of 3 mm thick Medihoney®, 90% of the pressure ulcers were void of bacterial growth. However, baseline clinical infection status was not reported in the pressure ulcers (Level 5 study). Gunes and Eser 78 conducted a RCT with 26 participants with 68 Category/Stage II and III pressure ulcers. The study compared healing rates in ulcers 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) were the primary outcome measure. The honey treated group’s PUSH scores showed healing at 4 times the rate of the control group (p < .001) (Level 5 study).

Manuka honey should be rated UMF (Unique Manuka Factor) +12 or above for topical dressing products. Use medical-grade gamma irradiated honey, as other sterilising processes will destroy the UMF in the honey. 79

9. **Limit the use of topical antibiotics on infected pressure ulcers, except in special situations where the benefit to the patient outweighs the risk of antibiotic side effects and resistance.** (Strength of Evidence = C; Strength of Recommendation = \(\star\))

In general, topical antibiotics are not recommended for treating pressure ulcers. Individuals with pressure ulcers are clearly a high risk group for the acquisition, harboring, and dissemination of antibiotic-resistant organisms. Reasons for this include inadequate penetration for deep skin infections, development of antibiotic resistance, hypersensitivity reactions, systemic absorption when applied to large wounds, and local irritant effects, all of which can lead to further delay in wound healing.

Short courses of silver sulfadiazine, topical antibiotic solutions, or topical metronidazole can be useful in certain circumstances, for example on wounds that have been debrided and cleansed, yet still have a bacterial bioburden of \(\geq 10^5\) CFU/g of tissue and/or the presence of beta hemolytic streptococci. These wounds are considered infected and may benefit from a short course of topical antibiotic guided by culture results and microbial sensitivity.1,2,81-83

Topical metronidazole might be used for the treatment of malodor in fungating wounds or wounds with anaerobic infection (indirect evidence).

10. **Use systemic antibiotics for individuals with clinical evidence of systemic infection, such as positive blood cultures, cellulitis, fasciitis, osteomyelitis, systemic inflammatory response syndrome (SIRS), or sepsis.** (Strength of Evidence = C; Strength of Recommendation = \(\star\))

Pressure ulcers are a known cause of sepsis and death.84-87 Abscessed or grossly infected pressure ulcers should be drained and debrided to treat ulcer related sepsis or advancing cellulitis. Systemic antibiotics can reach infected tissue in the base of the pressure ulcer, whereas topically applied agents cannot penetrate through necrotic tissue to reach the wound bed below. Antibiotics should be chosen based on confirmed antibiotic susceptibilities of the suspected or known pathogens. For life-threatening infections, empiric antibiotics should be based on local antimicrobial susceptibility patterns, and re-evaluated when definitive cultures become available.1,2,81-83 (indirect evidence). In some instances, the use of antibiotics may be limited by individual preference or advance directives for end-of-life care.

Judicious use of systemic antibiotics remains an important consideration. In a retrospective study including primarily Category/Stage IV pressure ulcers (56 participants with 115 ulcers) referred for surgical consultation, 4% of pressure ulcers 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.88 Cataldo et al. (2011)89 reported a prevalence rate of 15% for MRSA in a convenience sample of older adults with at least Category/Stage III pressure
ulcers (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 Brazil hospital who had Category/Stage II or greater pressure ulcers, 43.5% of participants had a MRSA colonized pressure ulcer and 8.3% had MRSA bacteremia. Approximately 57% of the participants had received at least two classes of antibiotics in the preceding 30 days\(^{(90)}\) (Level 4 study).

11. **Drain local abscesses. (Strength of Evidence = C; Strength of Recommendation = \(\star \star \))**

Local abscesses, the collection of pus, should be incised and drained to prevent local or systemic spread of the infection.

12. **Evaluate the individual for osteomyelitis if exposed bone is present, the bone feels rough or soft, or the ulcer has failed to heal with prior therapy. (Strength of Evidence = C; Strength of Recommendation = \(\star \))**

Osteomyelitis has been reported in up to 32% of individuals with pressure ulcers.\(^{(91-93)}\) Diagnostic assessments may include plain film X-rays, elevated white counts, elevated erythrocyte sedimentation rate (ESR), bone scans, magnetic resonance imaging (MRI), and biopsy, depending on the clinical situation.

Growing research has shown some benefits of using MRI for the diagnosis of osteomyelitis, although there is insufficient evidence on which to base definitive recommendations.\(^{(94-96)}\) A retrospective review of 41 MRI scans conducted on 37 participants with pressure ulcers 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 \(r = 0.82\)). There was high interrater agreement (\(\kappa = 0.92\), 95% confidence interval [CI] 0.84 to 1.01, \(p < 0.0001\)) between radiographers on the likelihood of osteomyelitis\(^{(94-96)}\) (Level 5 study). 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 procedure\(^{(97)}\) (Level 5 study).

Permanent healing of the pressure ulcer is unlikely until osteomyelitis is controlled. Treatment of osteomyelitis is beyond the scope of these guidelines.

**References**

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WOUND DRESSINGS FOR TREATMENT OF PRESSURE ULCERS

Introduction

Wound dressings are a central component of pressure ulcer 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. Occlusive or semi-occlusive wound dressings that maintain wound bed moisture promote re-epithelialization and wound closure. Wound dressings for pressure ulcers are designed to:

• improve wound healing time;
• absorb blood and tissue exudate;
• minimize pain associated with application and removal;
• absorb and control malodor; and
• reduce injury to periwound skin.

Recently, the role of dressings in protecting skin at high risk of pressure ulcers from shear has received an increased focus in clinical practice and research. Recommendations on the use of prophylactic dressings are outlined in Emerging Therapies for Prevention of Pressure Ulcers. Recommendations on negative pressure wound therapy (NPWT) are found in the Biophysical Agents in Pressure Ulcer Treatment section of the guideline.

General Recommendations

1. Select a wound dressing based on the:
   • ability to keep the wound bed moist;
   • need to address bacterial bioburden;
   • nature and volume of wound exudate;
   • condition of the tissue in the ulcer bed;
   • condition of periwound skin;
   • ulcer size, depth and location;
   • presence of tunneling and/or undermining;
   • goals of the individual with the ulcer. (Strength of Evidence = C; Strength of Recommendation = )

This statement is based on expert opinion. When the pressure ulcer is clean and granulating, maintenance of a moist wound bed is an important factor in promoting healing or closure. A dressing that remains in contact with the wound bed or a skin barrier product keeps the periwound dry and prevents maceration. As the ulcer 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 ulcer heals. Several moisture-retentive dressings are available.

2. Protect peri-ulcer skin. (Strength of Evidence = C; Strength of Recommendation = )

This statement is based on expert opinion. Care of skin surrounding the pressure ulcer is outlined in the Wound Care: Cleansing section of the guideline.

3. Assess pressure ulcers at every wound dressing change and confirm the appropriateness of the current dressing regimen. (Strength of Evidence = C; Strength of Recommendation = )

This statement is based on expert opinion. See the section Assessment of Pressure Ulcers and Monitoring of Healing from the full Clinical Practice Guideline for detailed information on assessing pressure ulcers.

4. Follow manufacturer recommendations, especially related to frequency of dressing change. (Strength of Evidence = C; Strength of Recommendation = )
5. Change the wound dressing if feces seep beneath the dressing. (Strength of Evidence = C; Strength of Recommendation = ⬤) 

This statement is based on expert opinion. Entry of feces into the direct wound healing environment increases the risk for infection.

6. The plan of care should guide usual dressing wear times and contain provisional plans for dressing changes as needed (for family, the individual, and staff) due to soilage, loosening, etc. (Strength of Evidence = C; Strength of Recommendation = ⬤) 

7. Ensure all wound dressing products are completely removed with each dressing change. (Strength of Evidence = C; Strength of Recommendation = ⬤) 

This statement is based on expert opinion. See the guideline sections Wound Care: Cleansing Wound Care: Debridement for guidance on preparing the wound bed for dressing application.

Hydrocolloid Dressings

1. Use hydrocolloid dressings for clean Category/Stage II pressure ulcers in body areas where they will not roll or melt. (Strength of Evidence = B; Strength of Recommendation = ⬤) 

2. Consider using hydrocolloid dressing on noninfected, shallow Stage III pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = ⬤) 

3. Consider using filler dressings beneath hydrocolloid dressings in deep ulcers to fill in dead space. (Strength of Evidence = B; Strength of Recommendation = ⬤) 

4. Carefully remove hydrocolloid dressings on fragile skin to reduce skin trauma. (Strength of Evidence = B; Strength of Recommendation = ⬤) 

Hydrocolloid dressings are a common treatment for Category/Stage II pressure ulcers due to their long wear time. The manufacturing of these dressings has advanced, with improvements in the adhesion of dressing edges, addition of antimicrobials to the gel, and development of wound dressing shapes designed for specific anatomical locations. (e.g., heel, sacrum). When dressings were taped to a “hydrocolloid window” around surgical wounds (rather than directly to the skin), Milne et al. (1999) found less damage to periwound skin.

The evidence statement for the use of hydrocolloid dressings for the treatment of pressure ulcers is derived from three meta-analyses comparing hydrocolloid dressing with paraffin gauze and wet-to-dry gauze dressings. Singh et al. (2004) (Level 3 study) analyzed the effect of gauze versus hydrocolloid dressing in pressure ulcers and venous ulcers, and reported that treatment with hydrocolloid dressing resulted in a statistically significant improvement in the complete healing rate of pressure ulcers. In a meta-analysis of five trials, Bradley et al. (1999) concluded that hydrocolloid dressings led to statistically significant improvement in the rate of pressure ulcer healing when compared to traditional treatments. The results from a meta-analysis by Bouza et al. (2005) showed that hydrocolloid dressings improve healing of pressure ulcers when compared to traditional forms of gauze. However, the effect size was small and there was no difference between the healing rates of hydrocolloid dressings and more advanced forms of dressings for pressure ulcers. Today, this information is well-accepted, and wet-to-dry dressings are seldom used because their continuous mechanical debridement prevents healing.

Belmin et al. (2002) conducted an open, randomized, multiple-center, parallel group trial to compare sequential treatment with calcium alginate and hydrocolloid dressings in 110 participants with non-infected and granulating Category/Stage III or IV pressure ulcers. The healing rate was more rapid in the pressure ulcers treated with calcium alginate first, compared to the group treated with hydrocolloid dressings alone (Level 1 study). Graumlich et al. (2003) conducted an eight week single-
blinded randomized controlled trial (RCT) in 65 participants with Category/Stage II or III pressure ulcers, 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 2 study).

Clinical utility of hydrocolloid dressings has been reported, including indications as to their conformance (tested on the heels), absorbency, adhesion, and ease of removal. Bale et al. (1997)\textsuperscript{8} compared hydrocolloid to foam dressings and concluded that there was no difference in mean wear time (Level 2 study). Brown-Etris et al. (2008)\textsuperscript{9} compared hydrocolloids to film dressings containing an absorptive pad, and concluded that the films were more easily placed, removed, and conformable (Level 1 study). Baxter (2000)\textsuperscript{10} (Level 5 study) and Brown-Etris et al. (2008)\textsuperscript{9} reported on the removability of hydrocolloid dressings, addressing the issues with patches of adhesive and dressing remaining on the skin.

### Transparent Film Dressings

1. Consider using film dressings for autolytic debridement when the individual is not immunocompromised. (Strength of Evidence = C; Strength of Recommendation = \textsuperscript{})

2. Consider using film dressings as a secondary dressing for pressure ulcers treated with alginates or other wound filler that will likely remain in the ulcer bed for an extended period of time (e.g., 3 to 5 days). (Strength of Evidence = C; Strength of Recommendation = \textsuperscript{})

3. Carefully remove film dressings on fragile skin to reduce skin trauma. (Strength of Evidence = C; Strength of Recommendation = )

4. Do not use film dressings as the tissue interface layer over moderately to heavily exuding ulcers. (Strength of Evidence = C; Strength of Recommendation = )

5. Do not use film dressings as the cover dressing over enzymatic debriding agents, gels, or ointments. (Strength of Evidence = C; Strength of Recommendation = )

These recommendations are based on expert opinion. Film dressings were originally designed to cover intact skin over intravenous puncture sites. The transparency of these dressings allows inspection of the skin beneath. Plain film dressings do not absorb drainage from a wound bed. Brown-Etris et al. (2008)\textsuperscript{9} compared hydrocolloids to film dressings containing an absorptive pad, and concluded that the films were more easily placed and removed and were conformable. The Wound Ostomy and Continence Nurses Society (WOCNS) and Agency for Health Care Policy and Research (AHCPR) guidelines address the role of film dressings for autolytic debridement.\textsuperscript{11, 12} As outlined in the guideline section \textit{Wound Care: Debridement}, this form of debridement is commonly performed with film dressings to allow easy wound monitoring. There is little other research on the use of film dressings for treating pressure ulcers.

### Hydrogel Dressings

1. Consider using hydrogel dressings on shallow, minimally exuding pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = )

2. Consider using amorphous hydrogel for pressure ulcers that are not clinically infected and are granulating. (Strength of Evidence = B; Strength of Recommendation = )

3. Consider using hydrogel dressings for treatment of dry ulcer beds. (Strength of Evidence = C; Strength of Recommendation = )

4. Consider using hydrogel dressings for painful pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = )
5. Consider using hydrogel sheet dressings for pressure ulcers without depth and contours and/or on body areas that are at risk for wound dressing migration. (Strength of Evidence = C; Strength of Recommendation = )

6. Consider using amorphous hydrogel for pressure ulcers with depth and contours and/or on body areas that are at risk for dressing migration. (Strength of Evidence = C; Strength of Recommendation = )

Hydrogel dressings contain hydrated hydrophilic polymers, which produce a moist environment that promotes wound healing. The increase in moisture in the wound bed facilitates autolytic debridement. Other advantages to hydrogel dressings are reductions in:

• wound pain, as gels do not adhere to the wound surface;
• time taken to perform dressings changes; and
• frequency of dressing changes.

The two most common types of hydrogels are amorphous hydrogels and sheet hydrogels. Amorphous gels are clinically preferred for pressure ulcers 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 ulcers on nonmoving and nondependent body surfaces.

Little formal study of hydrogels could be found. These recommendations are based on evidence from one Level 2 study and expert opinion. Matzen et al. (1999)\cite{13} randomly assigned amorphous hydrogel or a continuously wet dressing in 32 participants with non-infected Category/Stage III or IV pressure ulcers on the sacrum or trochanter. Despite a large loss of sample size, wound volume was significantly smaller in the hydrogel group (p < 0.02). The hydrogel-treated group also needed necrotic tissue to be debrided from the wound (p< 0.03) on significantly fewer occasions (Level 2 study).

Alginate Dressings

1. Consider using alginate dressings for the treatment of moderately and heavily exuding pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = )

2. Consider using alginate dressings in clinically infected pressure ulcers when there is appropriate concurrent treatment of infection. (Strength of Evidence = C; Strength of Recommendation = )

3. Gently remove the alginate dressing, irrigating it first to ease removal if necessary. (Strength of Evidence = C; Strength of Recommendation = )

4. Consider lengthening the interval between wound dressing changes or changing the type of wound dressing if the alginate dressing is still dry at the scheduled time for dressing change. (Strength of Evidence = C; Strength of Recommendation = )

Alginate is able to absorb exudate and maintain ulcer bed moisture. Alginate dressings can often be left on an ulcer for several days, thereby decreasing frequency of dressing changes. Alginate dressings are manufactured in sheet and rope forms. The clinical choice between alginate sheet and rope dressings is based on the depth and shape of the ulcer. Residual alginate fibers are not biodegradable; therefore they should be completely removed from the ulcer bed. See the guideline section Wound Care: Cleansing and Wound Care: Debridement for recommendations on cleaning the wound bed in preparation for a wound dressing application.

The recommendations are derived from two RCTs. Belmin et al. (2002)\cite{6} reported that ulcer surface area of Category/Stage II and III pressure ulcers in geriatric participants was statistically significantly reduced in size with an alginate dressing for four weeks, followed by a hydrocolloid dressing for four weeks, when compared to use of a hydrocolloid dressing alone for eight weeks (Level 2 study). Sayag et al. (1996)\cite{14} conducted a RCT with 92 participants, and also reported that mean healing time was...
reduced in full thickness pressure ulcers when treated with alginate, compared to dextranomer paste (Level 2 study).

Because alginites have minimal antimicrobial properties they are generally not used as the primary or only treatment for infected ulcers. Treatment of infected pressure ulcers is discussed in the Assessment and Treatment of Infection and Biofilms section of the guideline.

**Foam Dressings**

1. Consider using foam dressings on exuding Category/Stage II and shallow Category/Stage III pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = \( \circ \))

2. Avoid using single small pieces of foam in exuding cavity ulcers. (Strength of Evidence = C; Strength of Recommendation = \( \circ \))

3. Consider using gelling foam dressing in highly exuding pressure ulcers. (Level of Evidence C; Strength of Recommendation = \( \circ \))

Foam dressings absorb wound exudate from the pressure ulcer 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)\(^8\) compared hydrocolloid dressings to foam dressings and concluded that the latter managed exudate more effectively, although there was no significant difference in wound dressing wear time. Clinical uses of foam dressings also include application as a cover dressing to extend wear time (Level 2 study).

Diehm et al. (2005)\(^15\) reported a descriptive study of 6,693 participants with chronic exuding ulcers of multiple types, including 1,793 participants with pressure ulcers. Only 4.5% of the ulcers were classified as superficial in this group, and 49% were described as infected. The ulcers were managed with a hydropolymer dressing. At four weeks, there was a 67% reduction in ulcer radius; 39% of the pressure ulcers 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 (Level 5 study).

Parish et al. (2008)\(^16\) conducted a small quasi-experimental study (n = 23) of an adhesive, gelling foam dressing. At 28 days, 4% of pressure ulcers were described as healed, 30% had a marked improvement, 26% showed mild improvement, 4% had mild deterioration and 9% were markedly deteriorated (Level 5 study).

**Silver-Impregnated Dressings**

1. Consider using silver-impregnated dressings for pressure ulcers that are clinically infected or heavily colonized. (Strength of Evidence = B; Strength of Recommendation = \( \circ \))

2. Consider using silver-impregnated dressings for ulcers at high risk of infection. (Strength of Evidence = B; Strength of Recommendation = \( \circ \))

3. Avoid prolonged use of silver-impregnated dressings. Discontinue silver dressings when wound infection is controlled. (Strength of Evidence = C; Strength of Recommendation = \( \circ \))
**Caution: Topical silver products should not be used on patients with silver sensitivities. Silver may have toxic properties, especially to keratinocytes and fibroblasts; the extent of the toxicities has not been fully described.**

There are several forms and formulations of silver-impregnated dressings available for wound care. This section of the guideline addresses wound dressings that are impregnated with silver. The use of topical silver sulfadiazine is discussed in the guideline section *Assessment and Treatment of Infection and Biofilms.*

Metallic silver is relatively inert, but the presence of liquid leads to the release of the silver ion responsible for its biological activity. Silver ions are biocidal at very low concentrations due to the ability of microbial cells to absorb and concentrate silver from very dilute solutions. However, the presence of organic matter significantly diminishes the efficacy of silver. The efficacy of silver dressings remains to be confirmed in the presence of devitalized tissues in a wound bed.

The study of silver dressings in pressure ulcers is still being debated due to the misperception of direct cost effectiveness. The primary aim of treatment with a silver dressing is to reduce bioburden. There is currently little scientific literature upon which to base recommendations on the use of silver in wound care. The studies reported below used silver dressings for a maximum of four weeks.

Vermeulen et al. (2007) conducted a Cochrane systematic review and reported that silver dressings did not lead to pressure ulcer healing. The reviewers confirmed that silver dressings were associated with a reduction in ulcer area. Munter et al. (2006) studied 619 participants with chronic ulcers, including 46 individuals with pressure ulcers. They reported greater reduction in wound area with a silver foam dressing (58.5%, compared with 33.3% for local best practice), less maceration, better handling of exudate, and faster reduction of odor (Level 3 study).

A recent RCT by Trial et al. (2010) that included 42 participants with pressure ulcers showing one or more symptoms of local infection, compared outcomes of ionic silver alginate matrix with that of a silver-free alginate dressing. While the silver dressing appeared to improve the bacteriological status of the wounds, this trial was underpowered to measure clinical effectiveness, and further trials are needed to demonstrate a positive impact on the healing process (Level 2 study).

Chuangsuwanich et al. (2011) conducted a low quality randomized trial with 40 participants comparing silver mesh dressings (n = 20) with silver sulfadiazine cream (n = 20). There was no significant difference (p = 0.093) in reduction in pressure ulcer area between the two types of silver treatments after eight weeks. The cost of silver mesh dressings was reported to be cheaper (Level 2 study).

Beele et al. (2010) compared a silver alginate/carboxymethyl-cellulose antimicrobial wound dressing with a non-silver calcium alginate dressing over four weeks. Participants (n = 36, only n = 12 had pressure ulcers) were considered at risk of infection as assessed using the mASEPSIS tool (a tool in which Likert scores are used to assess signs and symptoms indicative of infection). Beele et al. (2010) reported significant reduction in overall wound surface area (p = 0.017 between wound dressing types). A reduction in mASEPSIS scores was found for the silver alginate dressing, but this was not significantly different from the outcomes observed for the non-silver dressing (indirect evidence).

It is important to acknowledge that silver dressings are intended to reduce bioburden and their use should be discontinued once the pressure ulcer is healing. Silver-resistant strains of bacteria may be emerging. The prophylactic use of a silver dressing as a barrier to microorganisms in wounds at high risk of infection or re-infection should be carefully considered and clearly documented.
Honey Impregnated Dressings

1. Consider using dressings impregnated with medical-grade honey for the treatment of Category/Stage II and III pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ≥)  

   Caution: Before applying a honey dressing, ensure the individual is not allergic to honey. Individuals who have bee or bee stings allergies are usually able to use properly irradiated honey products.  

Honey-impregnated dressings should be compared to alginates, hydrocolloids, silver and other advanced topical treatments for pressure ulcers. Honey produces hydrogen peroxide (H₂O₂), 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. Gunes and Eser conducted a RCT with 26 participants with 68 Category/Stage II and III pressure ulcers. The study compared healing rates in ulcers treated with unprocessed honey with a minimum inhibitory concentration (MIC) of 3.8%, to those treated with ethoxy-diaminoacridine plus a nitrofurazone dressing. The primary outcome measure was Pressure Ulcer Scale for Healing (PUSH) tool scores. The honey-treated group’s mean PUSH tool score showed healing at four times the rate of the control group (p < .001) (Level 5 study).

The use of medical-grade honey as a topical agent under a dressing (i.e., not a honey impregnated dressing) is discussed in the Assessment and Treatment of Infection and Biofilms section of this guideline.

Cadexomer Iodine Dressings

1. Consider using cadexomer iodine dressings in moderately to highly exuding pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ≥)  

   Caution: Iodine products should be avoided in individuals with impaired renal failure, history of thyroid disorders or known iodine sensitivity. It is not recommended for individuals taking lithium, or for pregnant or breast-feeding women. Iodine toxicity has been reported in a few case studies, especially in those individuals with large wounds, in whom dressings were changed often. The risk of systemic absorption increases when iodine products are used on larger, deeper wound or for prolonged periods.

Cadexomer iodine consists of spherical hydrophilic beads of cadexomer starch that contain iodine, are highly absorbent, and release iodine slowly in the wound area. It is available as an ointment, a wound dressing and as a powder. Moberg et al. (1983) conducted a randomized controlled trial on 34 participants with pressure ulcers, comparing cadexomer iodine with standard treatment. Cadexomer iodine significantly reduced pus, debris, and pain of the ulcers and accelerated the healing rate. After eight weeks of treatment, ulcer area was reduced by 76% and 57% in the cadexomer iodine and standard treatment groups, respectively. Six ulcers treated with cadexomer iodine were completely healed, while only one with standard treatment was healed.

Gauze Dressings

1. Avoid using gauze dressings for open pressure ulcers that have been cleansed and debrided because they are labor-intensive, cause pain when removed if dry, and lead to desiccation of viable tissue if they dry. (Strength of Evidence = C; Strength of Recommendation = ≥)  

   Caution: Avoid use of wet-to-dry gauze dressings.

2. When other forms of moisture-retentive dressing are not available, continually moist gauze is preferable to dry gauze. (Strength of Evidence = C; Strength of Recommendation = ≥)
3. Use gauze dressings as the cover dressing to reduce evaporation when the tissue interface layer is moist. (Strength of Evidence = C; Strength of Recommendation = ⚫)

4. Use loosely woven gauze for highly exuding ulcers; use tightly woven gauze for minimally exuding ulcers. (Strength of Evidence = C; Strength of Recommendation = ⚫)

5. Loosely fill (rather than tightly pack) ulcers with large tissue defects and dead space with saline-moistened gauze when other forms of moisture-retainive dressing are not available, to avoid creating pressure on the wound bed. (Strength of Evidence = C; Strength of Recommendation = ⚫)

6. Change gauze packing often enough to manage exudate. (Strength of Evidence = C; Strength of Recommendation = ⚫)

7. Use a single gauze strip/roll to fill deep ulcers; do not use multiple gauze dressings, because retained gauze in the ulcer bed can serve as a source of infection. (Strength of Evidence = C; Strength of Recommendation = ⚫)

8. Consider using impregnated forms of gauze to prevent evaporation of moisture from continuously moist gauze dressings. (Strength of Evidence = C; Strength of Recommendation = ⚫)

Gauze dressings are made of cotton or synthetic fabric that is absorptive and permeable to water, water vapor and oxygen. Practice varies widely in relation to gauze dressings. Increased infection rates, retained dressing particles, and pain have led health professionals in some geographic regions to avoid the use of gauze dressings for open chronic wounds such as pressure ulcers, in favor of advanced wound dressings. Several studies have demonstrated faster healing rates with advanced dressings when compared to saline-moistened gauze or only a dry gauze.

If used, gauze may be used dry; moist; or impregnated with paraffin, petrolatum, antiseptics, or other agents. It is manufactured in varying weaves, and with different size interstices. Gauze dressings today are fairly limited and primarily used as surgical dressings. Due to the need for frequent changes, they have been shown to be costly in health professional time. Although the use of saline impregnated or moistened gauze is preferable to allowing the ulcer to desiccate, the formulary should provide access to advanced wound dressing options.

**Silicone Dressings**

1. Consider using silicone dressings as a wound contact layer to promote atraumatic dressing changes. (Strength of Evidence = C; Strength of Recommendation = ⚫)

2. Consider using silicone dressings to prevent periwound tissue injury when periwound tissue is fragile or friable. (Strength of Evidence = B; Strength of Recommendation = ⚫)

Silicone is chemically inert, and adverse effects from the use of silicone in wound care are rare. Since silicone is inert, it does not chemically interact with the wound. Silicone is insoluble in wound exudate. Silicone dressings are designed to provide a wound contact layer that can be removed without causing trauma to the tissues or pain for the individual. These dressings can also protect friable or newly healed periwound tissue from injury during dressing changes. Meaume et al. (2003) conducted a RCT with 38 participants with Category/Stage II pressure ulcers comparing an adherent foam dressing to a silicone dressing. The silicone dressing was found to be less traumatic to the periwound tissue (Level 2 study).

**Collagen Matrix Dressings**

1. Consider using collagen matrix dressings for nonhealing Category/Stage III and IV pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ⚫)
Collagen is the most prevalent body protein and has been shown to be degraded in chronic wounds by proteases and elastase. Collagen matrix dressings are manufactured from bovine, porcine, or avian collagen and made in sheets and pads, as particles, and as gels.

The application of collagen has been shown to reduce the levels of proteases in chronic wounds. One RCT on collagen dressings and pressure ulcers found no difference in healing rates between ulcers treated with collagen matrix and those treated with a viscose rayon dressing (Level 2 study).

There is one well-designed RCT comparing collagen to hydrocolloid dressings in 65 subjects with 65 Category/Stage II and III pressure ulcers (Level 2 study). After adjusting for baseline depth, there was no significant difference in primary or secondary healing between the groups. Mean healing time in the collagen group was five weeks, compared to six weeks in the hydrocolloid group. Mean linear healing rate of the wound bed was three mm in both groups. Collagen was more expensive than hydrocolloid and required more nursing interventions per week. The ideal individual and ulcer to benefit from collagen dressings is yet to be elucidated.

Composite Dressings

Many of the dressing types listed here are manufactured in combinations. Please refer to the statements about the individual components when considering the use of composites.

Various composite dressings with new components for specific purposes emerge in the wound dressing market. For example, recent research conducted on the effectiveness of an advanced composite dressing containing chitosan (derived from sea crustacean) and polysaccharide alginate showed reduction in size for Category/Stage I to IV pressure ulcers treated for 21 days. The study had methodological limitations, including a high drop-out rate without intention-to-treat analysis and the product is currently not widely available, having been developed in Iran for military use (Level 2 study).

References


BIOLOGICAL DRESSINGS FOR THE TREATMENT OF PRESSURE ULCERS

Introduction

Biological dressings function as protective wound cover and may be cellular (contain living cells) or acellular (biologically inert). They can be composed of: 1

- animal (bovine or porcine) material,
- human (cadaveric skin) cells,
- plant (cellulose) materials,
- synthetic (man-made) material, or
- be a composite (mix of materials of various origin).

Biological dressings include skin substitutes, xenografts, allografts or collagen dressings. These dressings may function as biological modulators, influencing biological processes.3

Recommendations

1. Due to insufficient evidence to support or refute the use of biological dressings in the treatment of pressure ulcers, biological dressings are not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = )

One randomized, controlled, pilot study conducted in patients with Category/Stage III pressure ulcers 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 ulcers treated with collagen wound dressing were healed, compared with 80% treated with the foam wound dressing (Level 2 study).2 Studies conducted in individuals with diabetic (neurotrophic) foot ulcers3 and mixed etiology chronic wounds4 have demonstrated positive wound healing outcomes associated with biological dressings, including a bi-layered cell therapy wound dressing and a hyaluronic acid derivative wound dressing (indirect evidence).

References

GROWTH FACTORS FOR THE TREATMENT OF PRESSURE ULCERS

Introduction

The role of growth factors in the cellular and biochemical events that occur during wound healing includes regulation of cell proliferation and differentiation. Recombinant deoxyribonucleic acid (DNA) technology has been used to produce a recombinant human platelet-derived growth factor (rPDGF, rPDGF-BB, or rhPDGF-BB).

Platelet-rich plasma (also known as platelet-enriched plasma, platelet-rich concentrate, autogenous platelet gel, or platelet releasate) is used by placing supraphysiologic concentrations of autologous platelets at the site of tissue damage. Blood is drawn from the individual and centrifuged to create platelet-rich plasma that is applied to the wound.

Numerous growth factors have been investigated for healing of pressure ulcers including:

- recombinant platelet-derived growth factor (rPDGF)\(^1\)-\(^5\) (Level 2 studies);
- basic fibroblast growth factor (bFGF)\(^6\) (Level 3 study);
- granulocyte-macrophage colony-stimulating factor (GM-CSF)\(^7\) (Level 2 study);
- nerve growth factor (NGF)\(^8\) (Level 2 study);
- interleukin-1 beta (IL-1\(\beta\))\(^9\) (Level 2 study);
- transforming growth factor beta-3 (TGF-\(\beta3\))\(^10\) (Level 2 study);
- autologous platelet factors; and
- bone marrow nuclear cells\(^11\) (Level 5 study).

Recombinant Platelet-Derived Growth Factor

1. Consider using platelet-derived growth factors for treatment of Category/Stage III and IV pressure ulcers that have delayed healing. (Strength of Evidence = B; Strength of Recommendation = \(\geq\))

Three phase II clinical studies reported significantly improved healing of pressure ulcers treated with rPDGF.\(^1\)-\(^3\),\(^3\)-\(^5\) In a multi-centered, double blinded randomized controlled trial (RCT) conducted by Rees et al. (1999)\(^3\) participants (n = 124) with Category/Stage III or IV pressure ulcers were treated with becaplermin gel (a rPDGF) in doses of either 100 µg/g or 300 µg/g. The control group was treated with a placebo gel. Pressure ulcers 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 ulcer volume (Level 1 study).

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 ulcers 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 2 study).\(^4\),\(^5\)

Mustoe et al. (1994)\(^1\) 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 superior reduction in wound volume after 29 days compared with saline dressings. However, this study was small and had a high participant drop out (n = 11) that may have influenced the findings (Level 2 study). In a secondary analysis\(^2\) of a subset of the participants in this trial (n = 20), laboratory analyses demonstrated a significant increase in fibroblast content in pressure ulcers treated with rPDGF-BB compared with placebo (2.81 ± 0.17 versus 2.05 ± 0.24, p = 0.0) (indirect evidence). Process for selection of participants for this secondary analysis was not reported.
Other Growth Factors

1. Due to insufficient evidence to support or refute the use of growth factors (other than recombinant platelet-derived growth factor) in the treatment of pressure ulcers they are not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = ⊃)

Robson et al. (2000) conducted a phase II study (n = 61) comparing effectiveness of a range of growth factor treatments (GM-CSF alone, bFGF alone and sequential GM-CSF/bFGF) with placebo for treating Category/Stage III or IV pressure ulcers. At 36 day follow up, there was no statistically significant difference between any single treatment and placebo for reduction in wound volume or percent wound closure. When the growth factor groups were combined, significantly more (p = 0.03) of those participants achieved at least an 85% reduction in wound volume compared with placebo (Level 2 study).

A double blinded RCT (n = 26) investigated IL-1β in three doses for healing pressure ulcers with a baseline volume of between 10 and 100 cm³. There was no significant difference compared with placebo in decrease in wound volume at 29 day follow up (Level 2 study).

In a small RCT (n = 16), allogeneic platelet gel was shown to trigger early healing (onset of granulation tissue proliferation) in the first two weeks of treatment compared with control treatments (a range of iodine based dressings or negative pressure wound therapy), but no prolonged advantage was observed (Level 2 study). Platelet-rich plasma gel was also used in two case series that both demonstrated improvements in wound area and volume and reduction in undermining/sinus tracts after two weeks of treatment (Level 5 studies). None of these studies followed through to wound closure.

References


BIOPHYSICAL AGENTS IN PRESSURE ULCER TREATMENT

Introduction

Biophysical agents can be used to deliver specific treatment substances to the wound bed. These substances include oxygen via positive (hyperbaric or hyperatmospheric) pressure.

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.

Common Forms of Biophysical Agents

<table>
<thead>
<tr>
<th>Category</th>
<th>Biophysical Agents</th>
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<tbody>
<tr>
<td>Electromagnetic Spectrum</td>
<td>Electrical stimulation (ES)</td>
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<tr>
<td></td>
<td>Electromagnetic fields (EMF)</td>
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<tr>
<td></td>
<td>Pulsed radio frequency energy (PRFE)</td>
</tr>
<tr>
<td></td>
<td>Phototherapy: infrared (IR), ultraviolet (UV), light emitting diode (LED)</td>
</tr>
<tr>
<td>Acoustic</td>
<td>Low frequency ultrasound (LFU) KHz</td>
</tr>
<tr>
<td></td>
<td>High frequency ultrasound (HFU) MHz</td>
</tr>
<tr>
<td>Mechanical/ Kinetic</td>
<td>Subatmospheric such as NPWT but also including suction</td>
</tr>
<tr>
<td></td>
<td>Kinetic (whirlpool, pulsatile lavage, vibration)</td>
</tr>
<tr>
<td></td>
<td>Atmospheric (hyperbaric and topical oxygen)</td>
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</tbody>
</table>

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 by Aaron et al. (2004) and Bassett (1987) indicate that electrical stimulation (capacitive 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. Currently there is insufficient evidence to identify an optimal EMS frequency or wavelength signal for treating pressure ulcers.

Other forms of biophysical energy that have been studied in the management of pressure ulcers 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); pulsed lavage delivers kinetic and mechanical energy and suction (a form of sub-atmospheric pressure). Whirlpool delivers thermal (infrared) and mechanical energy (agitation).

All of these biophysical energies should be delivered to the individual using medical devices that meet local technical and legal requirements 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.

The use of electrical stimulation for preventing pressure ulcers is reported in the section Emerging Therapies for Prevention of Pressure Ulcers from the full Clinical Practice Guideline.
Electrical Stimulation

1. Consider the use of direct contact (capacitive) electrical stimulation to facilitate wound healing in recalcitrant Category/Stage II pressure ulcers as well as any Category/Stage III and IV pressure ulcers. (Strength of Evidence = A; Strength of Recommendation = )

This recommendation is supported by six randomized controlled trials (RCTs), three of them being moderate quality studies published between 2009 and 2012.3-5 Franek et al. (2012)3 conducted a RCT investigating electrical stimulation (ES) compared with standard wound care (SWC) for treating lower extremity Category/Stage II to III pressure ulcers (n = 50). The mean baseline area of the pressure ulcers was 4.54 cm² and 3.97 cm² in the study and control groups respectively and the ulcers had persisted for 2 to 3 months duration. In the ES group (n = 26) participants received SWC, preventive care practices and high-voltage pulsed current (HVPC; monophasic, double-peaked impulses; 100 pps; 100 μs; 100 V; the intensity on sensory level, below the level of muscle contractions) applied for 50 minutes a day, five days a week. Cathodal stimulation applied for the first one to two weeks was replaced by anodal stimulation for the remainder of the treatment period. The SWC group received preventive care and SWC only. After six weeks, wound areas 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 wound area was 88.9% in the ES group and 44.4% in the control group (p < 0.001).3 The limitations of this study included a lack of blinding, and the SWC consisted of a variety of treatments that may not have been consistent between the groups (Level 2 study).

The results of a non-blinded RCT by Franek et al. (2011)4 also showed significant progress in the healing of Category/Stage I to III pressure ulcers in 29 participants treated with HVPC (monophasic, double-peaked impulses; 100 pps; 100 μs; 100 V; the intensity on sensory level, below the level of muscle contractions; 50 minutes a day; five days a week). The mean area and the mean duration of pressure ulcers were 4.45 cm² and 3.17 months respectively in ES group and 4.93 cm² and 2.80 months in the control group. All patients received topical wound treatment (local bath of potassium permanganate, compression of fibrolan, colistin and wet dressings containing 10% sodium chloride) and regular repositioning. After six weeks the mean surface wound area decreased significantly in both groups (p ≤ 0.001 in ES group; p = 0.002 in control group). In the ES group eight of 29 pressure ulcers closed versus four of 29 pressure ulcers in the control group. A mean decrease in surface wound area was 85.38% in the ES group versus 40.08% in the control group (p ≤ 0.001) (Level 2 study).

Another single-blind, parallel-group RCT was carried out by Houghton et al. (2010)5 with 34 participants with spinal cord injury (SCI) who had Category/Stage II to IV pressure ulcers. In the ES group (n = 16), HVPC (monophasic, double-peaked impulses; 50 μs; 50 to 150 V) with the stimulator device programmed to provide 20 minutes at a pulse frequency of 100 pps followed by 20 minutes at 10 pps and then 20 minutes off-cycle for eight hours each day, for a period of at least three months or until the ulcer closed. The polarity of the treatment electrode was initially negative and then alternated weekly. In a treatment period of 12 weeks, all Category/Stage II pressure ulcers closed in both the ES group and the control group. In the ES group, 33.3% of Category/Stage III to IV pressure ulcers closed compared with 7.1% in the control group (p = 0.550). In the ES group, 80% of pressure ulcers decreased in surface area by at least 50%. This result was significantly better than in the control group, where the mean wound area reduction was 36% (p = 0.02). The average percentage decrease in wound surface area at treatment end was significantly greater in the ES group compared with the control 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, there was inconsistent application of the therapeutic ES protocol. In this study, SWC therapies were selected to meet the individual needs of participants, therefore management strategies varied. Specifically, the ES group received silver dressings to facilitate the ES therapy and none of the control group received silver dressings5 (Level 2 study).
Gardner et al. (1999)\(^6\) conducted a meta-analysis of studies investigating pressure ulcers and other wounds to quantify the effect of electrical stimulation on chronic wound healing. The pressure ulcers were analyzed separately and are reported here. There were 216 pressure ulcers studied (130 treated with ES and 86 controls). The percentage of ulcer surface area reduction was 16.63% in the pressure ulcers treated with ES compared to 3.59% in the control groups. Two studies used in the meta-analysis included cross over designs\(^5\), \(^8\) (Level 1 studies), where healing rates of pressure ulcers treated with electrical stimulation after the crossover mimicked the initial healing rates (12.9% per week). Pressure ulcers had the highest rate of healing with a net increase of 13.30% per week; a 403% increase over the control groups. The authors concluded there was more rapid healing in pressure ulcers treated with ES, although no conclusions could be drawn on which type of ES was more efficacious. Complete healing is seldom undertaken as an objective of biophysical agent clinical trials. In addition to reporting wound closure, other objective outcome measures usually reported are percent of healing per unit of time (e.g., week), and/or percent of ulcers healed. Several other studies on non-pressure ulcers or smaller samples have been done as well, but had methodological concerns.\(^9\)-\(^11\)

A Cochrane Review from 2001 of three randomized controlled trials suggested a benefit associated with electrotherapy treatment for pressure ulcers, yet they cautioned that this recommendation was based on three small studies with a total of 140 patients.\(^12\)

**Electromagnetic Agents**

1. **Consider the use of pulsed electromagnetic field (PEMF) treatment for recalcitrant Category/Stage II pressure ulcers as well as any Category/Stage III and IV pressure ulcers.** (Strength of Evidence = C; Strength of Recommendation = \(
\text{**C**}
\))

*Caution: No major adverse effects of electromagnetic therapy were reported in the research included in this review. Manufacturers of devices used to administer electromagnetic therapy do not recommend their use in individuals with pacemakers or other electrical implants, pregnancy or organ transplant. Caution is recommended for individuals with fever, active bleeding, seizures or dehydration.*\(^13\), \(^14\)

This recommendation is based primarily on expert opinion. Four RCTs conducted in the 1990s assessed this modality on chronic wounds and pressure ulcers\(^15\)-\(^17\) (Level 2 studies). While the results were suggestive of safe and accelerated wound healing in Category/Stage II to IV pressure ulcers, methodological flaws were numerous.

A small double-blind RCT was conducted by Gupta et al. (2009)\(^18\) in 12 participants (mean age approximately 27 years) with a total of 24 Category/Stage III and IV pressure ulcers. The PEMF was administered to six participants (13 pressure ulcers) for 30 sessions over six weeks. There was a significant improvement in wound healing as assessed using the Bates-Jensen Wound Assessment Tool (BWAT); however, the control group receiving sham PEMF also achieved significant healing and there was no statistically significant difference between groups.\(^18\)

A systematic review on the effectiveness of pressure ulcer treatments conducted by the Agency for Healthcare Research and Quality\(^19\) included the studies reported above and concluded that the evidence showed a trend toward improved rate of healing associated with electromagnetic therapy, but a lack of demonstrated clinical significance. The review suggested electromagnetic therapy could be considered as an adjunct to other interventions. Three different Cochrane reviews have been done on PEMF. The first in 2001\(^20\) reviewed three small quantitative studies (all on venous ulcers) that were deemed to be weak, and the review concluded that no clear evidence was provided on the benefit of PEMF in chronic wound treatment. The second Cochrane review in 2006\(^21\) of two methodologically limited studies\(^15\), \(^16\) with small samples concluded that the research did not provide evidence of benefit in using PEMF for pressure ulcer treatment and noted further research was needed. The third Cochrane review\(^22\) updated the search of Olyae Manesh et al. (2006)\(^23\) but found no new studies meeting the review inclusion criteria and made no changes to the conclusions.
Pulsed Radio Frequency Energy

Pulsed radio frequency energy (PRFE) has been shown to improve wound healing through promoting progression through inflammation to angiogenesis and tissue remodeling. The radio frequency signal used (27.12 MHz) is non-ionizing and has a non-thermal effect.

1. Consider the use of PRFE in the treatment of recalcitrant Category/Stage II pressure ulcers as well as any Category/Stage III and IV pressure ulcers (Strength of Evidence = C; Strength of Recommendation = △△)

Caution: No major adverse effects of electrotherapy were reported in the research included in this review. Electrotherapy is contraindicated in individuals with electrical implants (e.g., pacemakers) or who are pregnant. Electrotherapy is contraindicated in local anatomical areas of the eye, testes and any malignancy. Electrotherapy should be used with caution in individuals with impaired circulation or devitalized tissue.

Two trials (Level 5 studies) assessed this modality on chronic wounds and pressure ulcers. While the results were suggestive of safe and accelerated wound healing in Category/Stage II to IV pressure ulcers, methodological flaws were present in these studies.

Phototherapy: Laser, Infrared and Ultraviolet

Phototherapeutic agents, as mentioned above, employ energy waves from the infrared (IR), visible and ultraviolet (UV) region of the electromagnetic spectrum. A development in phototherapy involves the use of clusters of laser diodes (LDs), light-emitting diodes (LEDS), super luminescent diodes (SLDs), or a mixture of these light sources (cluster probes). Laser energy differs from that of an LED or SLD because it is emitted in a more narrow beam (collimated), has a single wavelength (monochromatic) and its light waves are all in phase (coherent). Combinations of these technologies are commonly used. The benefit of combining technologies include shorter treatment times, treatment of larger tissue areas, and biologic effects of different waveforms may be accessed.

Infrared Therapy

1. Due to current insufficiency of evidence to support or refute the use of infrared therapy in the treatment of pressure ulcers, infrared therapy is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = △△)

While studies and systematic reviews have been done on infrared therapy with and without heat; overall, findings are mixed. Studies were unclear regarding concurrent management strategies (e.g., the type of support surfaces used and what comprised standard wound care) and sample sizes were small.

Laser

1. Due to current insufficiency of evidence to support or refute the use of laser therapy in the treatment of pressure ulcers, laser therapy is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = △△)

Woodruff et al. (2004) performed a meta-analysis of 24 animal and clinical studies on the effectiveness of laser (including infrared-based units) on wound healing in a variety of ulcers on both animals and humans. They concluded that laser therapy studies had numerous methodological limitations.
Ultraviolet Light Therapy

1. **Consider a short term application of ultraviolet C light (UVC) if traditional therapies fail. (Strength of Evidence = C; Strength of Recommendation = )**

   This recommendation is based primarily on expert opinion. Currently, little evidence exists to support the use of ultraviolet light in the treatment of pressure ulcers. One study by Nussbaum et al. (1994)\(^31\) (Level 2 study) examined the effects of ultraviolet C light (UVC) in combination with ultrasound (n = 6) on pressure ulcer healing as compared to standard care therapy (n = 6) plus low level laser (n = 5) treatment in 17 participants with SCI. The combined UVC and ultrasound treatment enhanced healing over that attained with low level laser and standard care therapy. However, as the two treatment interventions (UVC and US) were combined, no definitive conclusion could be drawn as to their individual efficacy.

   In a more recent study, Nussbaum et al. (2013)\(^32\) compared UVC therapy (n = 30) to placebo light therapy (n = 28) for healing Category/Stage II to IV pressure ulcers in individuals with SCI. In this larger study, there was no statistically significant difference in rate of complete pressure ulcer healing between the two groups (35% UVC group versus 60% placebo group, p = ns). Percent area change between consecutive weeks averaged 16.2% for the UVC group and 5.2% for placebo group (p = not significant [ns]). Although Category/Stage II pressure ulcers treated with UVC therapy showed significantly greater reduction in size from baseline compared to the placebo group at some weekly time points (p < 0.03 to p < 0.05), the study was not powered to measure this effect and the large participant drop out that was excluded from the analysis suggests these findings be considered with caution.

   In an under-powered, double blinded study, Wills et al. (1983)\(^33\) (Level 2 study) reported significantly shorter healing times (6.25 ± 0.55 weeks versus 8.38 ± 0.45 weeks) for superficial ulcers exposed to UV light (n = 8) when compared to a placebo treatment (n = 8; p < 0.02).

   Small studies investigating the use of other light therapies including ultraviolet B light\(^34\) and polarized light\(^35\) have reported positive outcomes on pressure ulcer healing.

2. **Consider a course of ultraviolet light as an adjunctive therapy to reduce bacterial burden in critically colonized Category/Stage III and IV pressure ulcers that have been debrided and cleansed. (Strength of Evidence = C; Strength of Recommendation = )**

   This recommendation is based primarily on expert opinion. A study by Thai et al. (2005)\(^36\) demonstrated a reduction in bacterial numbers in 22 individuals with chronic wounds, only seven of which were pressure ulcers, exposed to 180 seconds of UVC (Level 3 study). *In vitro* and *in vivo* evidence also supports these findings\(^37, 38\) (Level 3 studies), as did a review.\(^39\)

   At this time, there is insufficient evidence to make a definitive conclusion as to the benefit of phototherapy in reducing bacterial numbers in pressure ulcers. Until sufficient evidence exists, phototherapy may be considered an adjunctive therapy to reduce bacterial burden in critically colonized pressure ulcers. However, it should not be used in the absence of other therapies (See *Assessment and Treatment of Infection and Biofilm* section of this guideline).

Acoustic Energy (Ultrasound)

Ultrasound (US) is a mechanical vibration transmitted in a wave formation at frequencies beyond the upper limit of human hearing. Units of measure for US are called Hertz (Hz). One hertz = 1 cycle per second and 1kHz = 1000 cycles per second. This vibratory property affects the tissue cells. Different frequencies are used therapeutically to treat and assess soft tissues.

High frequency US used therapeutically, is delivered between 0.5 and 3 million cycles per second (0.5 to 3 MHz). Thermal and nonthermal properties, as well as cellular effects, are related to all frequencies.
Low frequency US is typically between 20 to 50 kHz. Applications of low frequency include fibrinolysis and debridement of slough. Wound debridement of slough uses 22.5, 25, 35 or 40 kHz depending on the design of the manufacturer.

1. Due to current insufficiency of evidence to support or refute the use of noncontact low frequency (40 kHz) ultrasound spray (NC-LFUS) in the treatment of pressure ulcers, NC-LFUS is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation ▼)

Caution: No major adverse effects attributable to NC-LFUS were reported in the research included in this review. Noncontact low frequency ultrasound spray should not be used near prostheses, near electronic implanted devices (e.g., cardiac pacemakers), over the lower back or uterus in pregnant women; or over areas of malignancy; or on the face/head.

There is limited direct evidence on the efficacy of NC-LFUS in populations with pressure ulcers, although it is not infrequently used for wound care in geographic regions where it is available.

One uncontrolled study was conducted in participants (n = 13; n = 11 completed trial) with Category/Stage III pressure ulcers that had >10⁵ bacterial count to determine bacterial reduction associated with NC-LFUS. The participants received a wound biopsy at baseline and at two weeks, after six treatments of NC-LFUS (mean duration of treatment was four minutes). The per-protocol analysis showed a reduction in mean bacterial burden after two weeks (2 x 10⁷ versus 4 x 10⁷, 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). In the same study, the animal arm showed an overall slight decrease in total bacterial counts over 7 days, with increases in S. aureus and decreases in P. aeruginosa associated with NC-LFUS (p = not reported) (Level 5 study).

Evidence is emerging regarding the effect of NC-LFUS in treating suspected deep tissue injury (SDTI). A retrospective record review (n = 85 participants with 127 SDTIs) investigated the effect of NC-LFUS administered daily for five days then every other day (mean number of treatments = 10) compared with standard management. A non-validated assessment tool was applied retrospectively to photographs of wounds to assess total surface area, skin integrity and wound color/tissue. Scores for individual areas of assessment were combined to give a severity score from 3 to 18 (higher score indicates greater severity). The wound sizes were not comparable at baseline, with the control group having a larger mean total surface area (p = not reported); however there was no difference on severity scale (p < 0.913). The NC-LFUS group achieved significant reduction in severity score at follow up compared to the control group (t = 5.67, p < 0.000); however the study was insufficiently powered. In the treated participants, 18% of SDTI spontaneously resolved, compared with 2% of participants who received no NC-LFUS (Level 4 study).

There is a range of indirect evidence on the efficacy of NCLFU in populations with other types of wounds. The highest quality indirect evidence comes from a double-blind RCT that included participants with diabetic foot ulcers that compared LFUS therapy (40 kHz) to sham therapy. The intention-to-treat analysis (n=123) showed no significant difference in healing rates (26% versus 22%, p = ns). In the per protocol analysis (n=55), the LFUS group had a 40.7% closure rate compared with 14.3% for the control group (p = 0.0366); however, ulcers in the control group were of longer duration at baseline than those in the treatment group. Adverse effects included ulcer enlargement, blister, edema, erythema, pain and infection, but these did not occur at a significantly greater rate than for sham therapy (indirect evidence).
2. Consider use of low frequency (22.5, 25 or 35 kHz) ultrasound for debridement of necrotic soft tissue (not eschar). (Strength of Evidence = C; Strength of Recommendation = ⊳)

3. Consider use of high frequency (MHz) ultrasound as an adjunct for the treatment of infected pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = ⊳)

**Caution:** No major adverse effects of ultrasound were reported in the research included in this review. Its use is not recommended over anatomical areas with implanted materials or devices.

These recommendations are based on expert opinion. Limited evidence exists specific to the efficacy of HFUS or LFUS in the treatment of pressure ulcers. A 2006 Cochrane review\(^5\) identified three trials (n = 146 participants) with methodological limitations that reported on US to treat pressure ulcers, including the trial by McDiarmid et al. (1985)\(^5\) and the trial by Nussbaum et al. (1994)\(^3\) that included treatment with ultraviolet-C. None of the individual trials included in the Cochrane review found a significant effect for treatment with US. The meta-analysis also found no significant effect (risk ratio = 0.80, 95% CI 0.41 to 1.56, \(p = 0.51\)). An update to the review in 2009 did not include any additional studies. The Cochrane review concluded that there is no evidence supporting US in the treatment of pressure ulcers (Level 1 study).\(^5\)

One of the studies reported in the Cochrane review was a low quality RCT conducted by McDiarmid et al. (1985).\(^5\) Forty participants with Category/Stage I and II pressure ulcers were randomized into a treatment and sham treatment non-thermal three MHz US study. Ulcers exposed to sound waves tended to heal more quickly but the difference was not statistically significant \((p = 0.80)\). Ad-hoc analyses comparing effects in “clean” ulcers and in infected pressure ulcers suggested a significant effect in the healing of infected pressure ulcers, but the study was not powered to measure this effect and categorization of ulcers as clean or infected was based on visual appearance only (Level 2 study). A systematic review concluded that the above studies provided low strength evidence that the wound improvement observed with US is similar to that achieved with sham therapy.\(^7\)

Until stronger evidence is available, traditional methods of treatment for infected pressure ulcers should be used and high frequency US can be considered as an adjunct.

**Negative Pressure Wound Therapy**

Negative pressure wound therapy (NPWT) has been in use as a wound treatment modality for decades, and while it did not originate for the treatment of pressure ulcers, there are data to support its use in pressure ulcer treatment. For the past decade, NPWT has been used as a late treatment for recalcitrant wounds. More recently, NPWT has been used as a first-line treatment for ulcers that could achieve benefit; however, more research is needed to identify which participants are most likely to benefit from this therapy.

Negative pressure wound therapy has its greatest efficacy in reducing wound volume,\(^5\) and can serve as an adjuvant therapy when combined with debridement and other treatments that promote healing, such as nutritional support and pressure redistribution. Today, 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. The research on NPWT has focused on the intermediate outcomes of ulcer healing: reduction in wound volume,\(^5\) wound bed preparation for skin grafting or flap closure,\(^5\) ability to use a surface dressing rather than wound packing\(^5\) and rate of healing.\(^5\) Negative pressure wound therapy promotes wound healing through removal of third space edema,\(^5\) thus enhancing nutrient and oxygen delivery,\(^6\) removal of wound exudate, which is the medium for bacterial colonization;\(^6\) promotion of granulation tissue;\(^6\) promotion of angiogenesis; and removal of wound inhibitory factors. Therefore, the intent of NPWT is to facilitate wound closure rather than to fully close or heal a pressure ulcer.
1. Consider NPWT as an early adjuvant for the treatment of deep, Category/Stage III and IV pressure ulcers. (Strength of Evidence = B; Strength of Recommendation = )

Caution: Negative pressure wound therapy is not recommended in inadequately debrided, necrotic or malignant wounds; where vital organs are exposed; in wounds with no exudate; or in individuals with untreated coagulopathy, osteomyelitis or local or systemic clinical infection. Cautious use by an experienced health professional is recommended for individuals on anticoagulant therapy; in actively bleeding wounds; or where the wound is in close proximity to major blood vessels.53

Negative pressure wound therapy has been shown to reduce the depth of pressure ulcers when compared to traditional forms of topical therapies.53 A large retrospective review found that the rate of healing was significantly more rapid with NPWT (0.23 cm$^2$ per day). Healing rates were compared to those reported in a RCT by Ferrell et al. (1993)56 in which the pressure ulcer healing rate in participants on low-air-loss beds or foam mattresses was reported at 0.090 cm$^2$ per day (for the participants on the low-air-loss bed). Joseph et al. (2000)55 conducted a prospective randomized trial comparing NPWT to wet-to-moist gauze dressings covered with a thin film to simulate closed therapy without suction. Wound depth (percent change in depth) in this study was significantly more rapid (p < 0.00001) in the NPWT group (Level 2 study). Tissue biopsy showed more inflammation and fibrosis in the moist gauze dressing group and more granulation tissue in the NPWT group.55

Wanner et al. (2003)57 found no difference in time to reach a 50% decrease in ulcer volume in pelvic pressure ulcers in 22 individuals with SCI in order to allow surgical closure.

In one trial, de Laat et al. (2011)65 investigated the reduction of wound volume using NPWT versus sodium hypochlorite dressings. One of the more notable findings to emerge from this research was a reduction in the median treatment time of 50% (p = 0.001). Wild et al. (2008)66 also investigated reduction in wound area using NPWT versus Redon surgical drain bottles. The results of this research support the idea of using NPWT to improve healing outcomes for pressure ulcers. Findings showed an increase in surface granulation tissue of 54% in the NPWT and a reduction of granulation tissue in the Redon group (p = 0.001).

Wallin et al. (2011)67 conducted a retrospective descriptive study in which data for 87 participants who had received NPWT for wound management was reviewed. The study identified that NPWT had a successful outcome for individuals with acute wounds rather than in those with pressure ulcers (p = 0.001). The results of an observational study by Ho et al. (2010)68 did not find a significant statistical difference between the NPWT group and the non-NPWT group. 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). Nutritional status appears to be important in the effectiveness on NPWT.

2. Debride the pressure ulcer of necrotic tissue prior to the use of NPWT. (Strength of Evidence = C; Strength of Recommendation = )

Negative pressure wound therapy is intended for use in pressure ulcers free of necrotic tissue. Therefore, NPWT therapy should begin after debridement.

3. Follow a safe regimen in applying and removing the NPWT system. (Strength of Evidence = C; Strength of Recommendation = )

Clean technique can be most used for NPWT dressing changes. As NPWT is 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.69 Fill the defect and dead space with dressing and record the number of dressings placed in the ulcer. 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. Optimal negative pressure levels are not well-established, but usually range between 75 and 125 mm Hg. Place the drainage collection system on a level surface.

4. **Evaluate the pressure ulcer with each dressing change.** (Strength of Evidence = C; Strength of Recommendation = )

The optimal dressing change interval is not well-established, and should be 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. If tissue ingrowth into the dressings or tubing is noted, lower pressures may be sufficient to correct this problem. It is expected that granulation tissue will appear in the pressure ulcer; if present, monitor for tissue trauma or pain.

It is also expected that the ulcer will decrease in volume, and tunnels and undermining will resolve. If the ulcer 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 ulcer need to be fully evaluated with any deterioration (see the Assessment of Pressure Ulcers and Monitoring of Healing section from the full Clinical Practice Guideline). If there is no change in ulcer 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 NWPT.

5. **If pain is anticipated or reported consider:**
   - placing a nonadherent interface dressing on the wound bed, underneath the foam;
   - lowering the level of pressure, and/or changing type of pressure (continuous or intermittent); and/or
   - using a moist gauze filler instead of foam. (Strength of Evidence = C; Strength of Recommendation = )

Negative pressure wound therapy set 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. 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.

6. **Educate the individual and his/her significant others about negative pressure wound therapy when used in the community setting.** (Strength of Evidence = C; Strength of Recommendation = )

Negative pressure wound therapy systems can be used in outpatient or home settings. Provide adequate education so that the individual and his/her significant others know what to do if the seal loosens; alarms ring; blood or tissue are seen in the tubing; or local erythema develops. Emergency contacts should be provided.

**Hydrotherapy: Whirlpool and Pulsatile Lavage with and without Suction**

Hydrotherapy uses water (with or without the use of additives) or saline to stimulate wound healing and to cleanse and debride wounds. Warm water (IR energy) provides superficial warming of tissue, and may have beneficial physiological effects of increasing vasodilation and perfusion, thus increasing oxygen delivery to aid in healing. The effectiveness of whirlpool or pulsatile lavage with and without suction on pressure ulcer healing is largely unknown.

**Whirlpool**

Whirlpool is seldom used and is no longer recommended. Whirlpool has become a generic name for a metal or plastic tub with an agitator/turbine attached or built into the tub that is of a suitable size to submerge a body part when filled with heated water. The water hydrates and softens the tissue. Vigorous
rinsing of the wound and skin with potable warm water following immersion to remove bacteria and effluent from the water is required due to the risk of wound contamination.

1. Whirlpool should not be considered for routine use in treating pressure ulcers due to the potential for contamination and the emergence of newer hydrotherapies. (Strength of Evidence = C; Strength of Recommendation = ≤)

*Caution: Individuals with dependent lower extremity edema or peripheral vascular disease,* immunocompromised individuals, those who are mechanically ventilated and lethargic, and incontinent individuals should never be immersed.

Whirlpool has been used in the past for wound cleansing and reducing bacterial bioburden. Due to the risk of exposure to pathogens and potential wound contamination, it is not recommended as a routine treatment for pressure ulcers. Newer hydrotherapies have replaced whirlpool as a recommended treatment option.

One randomized clinical trial (n = 42) provided evidence that whirlpool treatments plus moist wound dressings led to faster healing rates over moist wound dressings alone in surgically debrided, clean, granulating, Category/Stage III and IV pressure ulcers (0.39 cm/week compared to 0.17 cm/week for moist wound dressings alone) (Level 2 study). No adverse effects were reported.

In vitro, adding chloramine-T at 200 ppm for 5 to 20 minutes was effective against three virulent gram-positive bacteria without fibroblast damage.

**Pulsed Lavage with/ without Suction**

When pulsed lavage is used, normal saline may be delivered between 4 and 15 psi (pressurized irrigation) through a mechanical apparatus. Suction (subatmospheric pressure) may be concomitantly employed to aspirate wound debris and remove microorganisms. The use of mechanical energy through a pressurized spray also assists with the removal of wound debris.

1. Consider a course of pulsed lavage with suction for wound cleansing and debridement. (Strength of Evidence = C; Strength of Recommendation = ≤)

One double blind RCT (n = 28, n = 14 treated with lavage) provided moderate quality evidence that daily low pulsed lavage (with 1 liter normal saline at 11 psi applied over 10 to 20 minutes) was associated with faster healing rates for Category/stage II and IV pressure ulcers in patients with SCI compared with sham treatment. Although pressure ulcers treated with pulsed lavage showed significantly greater negative changes over time in depth, width, length and volume (all p < 0.0001), 95% confidence intervals (CI) spanned the null value (Level 2 study).

**Vibration Therapy**

The use of vibration therapy to promote wound healing involves the application of mechanical vibration to part or all of the body. It is thought that this type of therapy stimulates blood flow due to mechanical stresses of endothelial cells resulting in vasodilation.

1. Due to current insufficiency of evidence to support or refute the use of vibration therapy in the treatment of pressure ulcers, vibration therapy is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = ≤)

One non-blinded RCT (n = 31) provided evidence that the application of mechanical vibration led to improved healing rates of Category/Stage I pressure ulcers. This study identified that more pressure ulcers in the experimental group healed compared to the control group (40% versus 9.5% p = 0.033). The mean relative change per day of wound area was superior in the experimental group (20.4 ± 27.2% versus 6.4 ± 6.9%, p = 0.007). The researchers suggested that seasonal variations in microclimate may have influenced the findings. (Level 2 study).
Oxygen for the Treatment of Chronic Wounds

Hyperbaric Oxygen Therapy (HBOT)

1. Due to current insufficiency of evidence to support or refute the use of hyperbaric oxygen therapy in the treatment of pressure ulcers, hyperbaric oxygen therapy is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation = )

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 (3 ATA) may be utilized.

The only study on HBOT for pressure ulcers was conducted by Rosenthal et al. (1971)77 In this study, the HBOT treatment of 18 participants with 38 pressure ulcers was compared to treatment of three controls. Twenty-two pressure ulcers healed completely (58%) and five (13%) had a greater than 50% decrease in wound size. The three controls with six pressure ulcers had no wounds heal and no wounds decreased in size 50% or greater. No inferential statistics were completed and no demographics or wound sizes were compared between groups (Level 3 study).77 Kranke et al. (2004)78 conducted a Cochrane review of the effectiveness of HBOT in treating diabetic foot ulcers with osteomyelitis and reported that a pooled analysis concluded that diabetic wounds treated with HBOT were more likely to heal when compared to wounds treated with traditional therapy (indirect evidence).

Topical Oxygen Therapy

1. Due to insufficient evidence to support or refute the use of topical oxygen in the treatment of pressure ulcers, topical oxygen is not recommended for routine use at this time. (Strength of Evidence = C; Strength of Recommendation )

Topical oxygen is a therapy in which 100% oxygen is applied directly to the wound. Pressures of 22 mm Hg and 50 mm Hg are most often reported in the literature. Heng et al. (2000)79 reported on a descriptive study that the healing rate in 15 individuals with 24 chronic wounds treated with topical oxygen. Nineteen of the wounds were neuropathic, so a maximum of five wounds could have been pressure ulcers. At 12 weeks, 22 of the 24 wounds were healed. The outcomes for the pressure ulcers were not reported separately. In a small study, Edsberg et al. (2002)80 also reported that there was no difference in healing in individuals with pressure ulcers treated with topical HBO compared to those treated with electrical stimulation and topical hyperbaric oxygen.

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CLINICAL PRACTICE GUIDELINE:

TREATMENT: BIOPHYSICAL AGENTS


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PAIN ASSESSMENT AND TREATMENT

Introduction

Pressure ulcers are painful. Individuals with pressure ulcers experience ulcer related pain that can be quantified and differentiated from other pain, and this pain occurs both during procedures and at rest. Dallam et al. (1995)\(^1\) evaluated pressure ulcer pain in 132 hospitalized adults with Category/Stage I or II pressure ulcers. Using a visual analog scale (VAS) or the Faces Rating Scale (FACES) scale, researchers were able to demonstrate that those participants who could respond (n = 44) could quantify their pressure ulcer pain. The average Category/Stage I and II pressure ulcer pain reported was 4 cm and 3.5 cm on a 10cm VAS, respectively. Individuals with Category/Stage IV pressure ulcers had greater pain than those with lower Category/Stage ulcers. In all, 68% of those who responded reported some degree of pressure ulcer pain. However, only 2% of those individuals reporting pressure ulcer pain received timely analgesics after complaints of pain (Level 3 study).

Gorecki et al. (2011)\(^2\) concurred with these findings in their systematic review (four quantitative studies and six qualitative studies) involving participants with pressure ulcer pain (n = 108). Participants with Category/Stage II pressure ulcers reported lower pain severity than those with Category/Stage III and IV pressure ulcers. The outcome of the work by Gorecki et al. (2011)\(^2\) is a conceptual framework of five pain domains: communicating the pain, feeling the pain, impact of pain, self-management, and professional management.

The pain caused by pressure ulcers can be constant and severe, and may be the most distressing pressure ulcer symptom the individual reports.\(^1,3\) Pain related to pressure ulcers can arise from:

- pressure, friction, and/or shear;
- damaged nerve endings;
- inflammation;
- infection;
- procedures/treatments; or
- excoriation from incontinence and muscle spasm.\(^21\)\(^23\)

Pressure ulcer pain can occur at rest, when no procedures are being performed.\(^4,8,11,17,18\)

A prevalence study conducted in long term aged care facilities in seven European countries (n = 4,156) found that presence of a severe pressure ulcer (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.\(^24\) Individuals with Category/Stage IV pressure ulcers experience more pain than individuals with lower Category/Stage ulcers.\(^1,11,16\)

Pressure ulcer related pain may be acute (including hyperalgesia), chronic, or neuropathic. Refer to the Glossary for definitions and further explanation.

Assess for Pressure Ulcer Pain

Data gathered during a pain assessment measures pressure ulcer pain presence, quality and quantity. These data are interpreted to determine the severity of pressure ulcer pain and inform the development of an appropriate management plan.

1. Assess all individuals for pain related to a pressure ulcer or its treatment and document findings. (Strength of Evidence = C; Strength of Recommendation = )

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.\(^20,21,25\) In fact, the U.S. Joint Commission on Accreditation of Hospital Organizations mandates regular and ongoing assessment of pain in all hospitalized individuals in US health facilities.\(^26\)
Pain assessments should be done prior to and during wound procedures, such as dressing changes or debridement, as well as when the dressing is intact and no procedures are in progress.

The assessment for pressure ulcer pain needs to be comprehensive, including objective and subjective assessment. An initial pain assessment should include the following four elements:

- A detailed pain history including the character, intensity and duration of pressure ulcer pain;
- A physical examination that includes a neurological component;
- A psychosocial assessment; and
- An appropriate diagnostic work-up to determine the type and cause of the pain.27

2. Assess for pressure ulcer related pain in adults using a scale that is valid and reliable. (Strength of Evidence = C; Strength of Recommendation = )

The McGill Pain Questionnaire (MPQ) and FACES were used in a study of 47 people with Category/Stage II to IV pressure ulcers, and a statistically significant relationship (Pearson’s r = 0.90) was found between current pain intensity and FACES20 (Level 5 study). Dallam et al. (1995)1 found that the VAS correlated with FACES (r = 0.92) and the Category/Stage of the pressure ulcer (r = 0.37). Pressure ulcer pain intensity correlated with generalized pain (r = 0.59) (Level 3 study). Variability in VAS scores significantly increased as FACES values increased10 (Level 5 study). In addition, the VAS and FACES proved to be highly reliable for pain assessment in individuals with decreased verbal and abstract thinking.10

2.1. Incorporate the individual’s cognitive ability into the selection of a pain assessment tool. (Strength of Evidence = C; Strength of Recommendation =  )

Pain assessment tools should be appropriate to the individual’s cognitive level. Individuals with pressure ulcers are often older and may have cognitive impairment. Studies investigating the use of FACES by cognitively impaired adults report that this population has difficulty using this scale compared with other self-report pain assessment tools.28-33 Likewise, the VAS has been shown to have limited reliability when used by cognitively impaired adults with pain associated with a range of conditions28, 29, 34 (indirect evidence). Current evidence suggests that the MPQ, which is validated in populations with pressure ulcer pain,20 provides the most reliable pain assessment for cognitively impaired individuals.35, 36

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:37-40

- 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?

3. Assess for pain in neonates and children using a validated scale. (Strength of Evidence = C; Strength of Recommendation = )

Children and neonates can experience pressure ulcer related pain,1, 10, 41-45 and assessment for pain is mandated in the US.26

3.1. Use the FLACC (Face, Leg, Activity, Cry, and Consolability) tool for children 2 months to 7 years of age. (Strength of Evidence = C; Strength of Recommendation = "C")

The FLACC tool was found to be valid and have high interrater reliability in a study of 89 children aged from two months to seven years who experienced postoperative pain44 (indirect evidence).
3.2. **Use the CRIES (Crying; Requires O2 for Saturation > 95%; Increasing vital signs; Expression; Sleepless) Scale for neonates up to 6 months. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

For neonates up to six months of age, the CRIES scale is effective.42, 43 The VAS can be used in older children.

4. **Pain assessment tools may not provide sufficient information to guide interventions. Investigate other aspects of the pain in order to provide more effective, individualized interventions. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

4.1. **Incorporate the individual’s body language and nonverbal cues into the assessment of pain. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

For cognitively impaired individuals and those who are nonverbal (including infants), observe for specific behaviors (e.g., change in activity, loss of appetite, guarding, grimacing, withdrawal, crying out and moaning) during wound procedures and movement. The AGS Panel on Pharmacological Management of Persistent Pain in Older Adults (2009)46 recommends looking for behaviors such as facial expressions, verbalizations or vocalizations, body movements, changes in interpersonal interactions, and changes in activity patterns or routines.

1.1. **Incorporate the words used by the individual to express pressure ulcer pain character into the assessment of pain. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

The MPQ includes a broad range of words to describe pain character. Acute pain is associated with pain terms such as quick, sharp and short. Chronic pain is associated with reports of constant or persistent pain.1, 20 Neuropathic pain is associated with terms such as ‘pins and needles’, stabbing, shooting, ‘hot poker’ and electric pulse.20 Health professionals should have high index of suspicion for neuropathy when these terms are reported.

1.2. **Evaluate factors that increase pain frequency and/or intensity when conducting an assessment of pain. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

In conducting a pain assessment, consider activities that influence pain (e.g., wound dressing changes, sharp debridement, movement and touch). Gunes (2008)20 found that individuals with pressure ulcer pain reported increased pain intensity at dressing changes compared with at rest.

4.4. **Evaluate the duration of the pressure ulcer and associated pain when conducting an assessment of pain. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

Gunes (2008)20 used both the MPQ and FACES to assess pressure ulcer pain. For the majority of individuals with pressure ulcers (94.6%, 44 out of 47), pain was constantly present. Pain intensity and constancy was associated with increasing Category/Stage of the pressure ulcer. About half (52%) of participants with Category/Stage II pressure ulcers reported intermittent pain and the majority of individuals with Category/Stage III pressure ulcers (56%) and Category/Stage IV pressure ulcers (67%) pressure ulcers reported constant pain. Pain scores on the MPQ increased as ulcer duration increased. Pain intensity was significantly greater for pressure ulcers of longer duration (p < 0.05) (Level 3 study).

5. **Assess for deterioration of the ulcer or possible infection when the individual reports increasing intensity of pain over time. (Strength of Evidence = C; Strength of Recommendation = ★★★)**

Increasing presence or intensity in pain is an indication that a chronic wound may be infected and a comprehensive assessment of the pressure ulcer should be performed.47-49 See the Assessment and Treatment of Infection and Biofilms section of the guideline for recommendations on assessment and management of infection.
6. Assess the impact of pressure ulcer pain on the individual’s quality of life. (Strength of Evidence = C; Strength of Recommendation = )

Pressure ulcers have measurable and persistent impact on health-related quality of life measures. In one study, participants with pressure ulcers 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 ulcers. Pressure ulcers were found to impact especially on measures of physical functioning (p = 0.001). Perceived pain was of borderline significance (p = 0.06) (Level 5 study).

Prevent Pressure Ulcer Pain

1. Use a lift or transfer sheet to minimize friction and/or shear when repositioning an individual, keeping bed linens smooth and unwrinkled. (Strength of Evidence = C; Strength of Recommendation = )

Repositioning is associated with pain in individuals with both medical and surgical conditions. One observational study (n = 1,395) found individuals experienced a mean score of 4.9 ± 3.1 on a 0 to 10 numerical rating scale when being turned, even when interventions to reduce pain were implemented (indirect evidence).

Hyperalgesia is described by individuals as occurring during repositioning and transfer activities 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. Provide positional support to affected pressure ulcer area where possible. Move gently and listen to the individual to guide movements.

2. Position the individual off the pressure ulcer whenever possible. (Strength of Evidence = C; Strength of Recommendation = )

Pressure ulcers 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 ulcer can result in increased pressure, pain and damage to the area. Keeping the individual off the pressure ulcer will relieve pain and ischemia, enhance soft tissue viability and promote healing of the pressure ulcer.

3. Avoid postures that increase pressure, such as Fowler’s position greater than 30° or 90° side-lying position, or the semi-recumbent position. (Strength of Evidence = C; Strength of Recommendation = )

The Repositioning and Early Mobilization section from the full Clinical Practice Guideline provides detailed recommendations on the role of positioning to both prevent and treat pressure ulcers.

Manage Pressure Ulcer Pain

1. Organize care delivery to ensure that it is coordinated with pain medication administration and that minimal interruptions follow. Set priorities for treatment. (Strength of Evidence = C; Strength of Recommendation = )

Pain management includes performing care after administration of pain medication to minimize pain experienced and interruptions to comfort for the individual.

2. Encourage individuals to request a ‘time out’ during any procedure that causes pain. (Strength of Evidence = C; Strength of Recommendation = )
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 their questions;
- allowing active participation;
- pacing the procedure to the individual’s preferences; and
- allowing time outs as needed.\textsuperscript{54-56}

3. Reduce pressure ulcer pain by keeping the wound bed covered and moist, and using a non-adherent dressing. (Note: Stable dry eschar is usually not moistened). (Strength of Evidence = B; Strength of Recommendation = \textsuperscript{\textbullet\textbullet})

Wounds resurface more quickly in the presence of moist wound healing.\textsuperscript{57} Pressure ulcer pain can be minimized by keeping the pressure ulcer wound bed moist and covered.\textsuperscript{58}

4. Select a wound dressing that requires less frequent changing and is less likely to cause pain. (Strength of Evidence = C; Strength of Recommendation = \textsuperscript{\textbullet\textbullet})

Hydrocolloids, hydrogels, alginates, polymeric membrane foams, foam and soft silicone wound dressings should be considered for management of painful pressure ulcers. A wound dressing that allows for less frequent changing is advised. Nonadherent and/or moist dressings cause less pain and trauma on removal.\textsuperscript{59-68} Gauze dressings are more likely to cause pain.

See the Wound Dressings for Treatment of Pressure Ulcers section of the guideline for further recommendations on wound dressing selection.

4.1. Where available, consider ibuprofen impregnated wound dressings as a topical analgesic treatment for pressure ulcer pain. (Strength of Evidence = C; Strength of Recommendation = \textsuperscript{\textbullet\textbullet})

\textit{n.b. Ibuprofen-impregnated dressings are not available in the U.S.}

Although ibuprofen impregnated dressings have not been tested in pressure ulcers, a recent Cochrane review\textsuperscript{69} provides indirect evidence on the effectiveness of ibuprofen impregnated dressings. The review included two randomized controlled trials conducted on participants with wound of mixed etiology. In one of the studies\textsuperscript{70} ibuprofen impregnated dressing was associated with a 40% reduction in pain from baseline, and a 30% reduction in pain compared with a foam dressing. In the second study,\textsuperscript{71} 19% more participants experienced at least a 50% reduction in their pain levels compared with foam dressing (indirect evidence).

5. Consider the use of non-pharmacological pain management strategies to reduce pain associated with pressure ulcers. (Strength of Evidence = C; Strength of Recommendation = \textsuperscript{\textbullet\textbullet})

This statement is based on expert opinion. A large range of non-pharmacological pain management strategies are used in managing pain including:

- music,
- progressive relaxation,
- position changes,
- meditation and self-hypnosis,
- guided imagery,
- healing touch,
- distraction and conversation,
- warmth applications, and
- electrotherapy therapy (e.g., transcutaneous electrical nerve stimulation [TENS]).
While few studies have been conducted on the effectiveness of these non-pharmacological strategies for managing pressure ulcer pain, their benefit in treating chronic neuropathic pain has been reported.\textsuperscript{22, 55, 72, 73}

6. **Administer pain medication regularly, in the appropriate dose, to control chronic pain following the World Health Organization Pain Dosing Ladder.** (Strength of Evidence = C; Strength of Recommendation = \(\text{	extbullet	extbullet}\))

The World Health Organization (WHO) Pain Dosing Ladder\textsuperscript{74} is a validated and effective method for relieving pain related to cancer and in other individuals with pain.\textsuperscript{27} Step one is the accurate assessment and measurement of the individual’s pain on a validated pain intensity scale, and then matching the pain intensity to the potency of the analgesic, beginning with nonopioid medication and proceeding to an opioid with an adjuvant. On a 10-point scale, mild pain would be 1 to 3, moderate pain 4 to 6, and severe pain 7 to 10. Mild pain would be treated with a nonopioid, moderate pain with a mild opioid with or without a nonopioid or adjuvant, and severe pain with a strong opioid with or without an adjuvant. The WHO Pain Dosing Ladder\textsuperscript{74} is based on the goals of minimizing side effects and maximizing pain relief. Opioids act on the central nervous system by altering the pain perception, while nonopioids act on peripheral nerves to block painful impulses. Adjuvants enhance the effectiveness of analgesics synergistically.\textsuperscript{27} Pain medication needs to be administered regularly in the appropriate dose to control chronic pain. Individuals with chronic wounds, especially older adults and those with dementia, continue to be under-assessed and under-treated.\textsuperscript{18, 75, 76}

To maintain analgesic effects, administer drugs ‘by the clock’ every three to six hours by the least invasive route.\textsuperscript{77}

A study (n = 34) investigating an innovative pressure ulcer 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 care\textsuperscript{78} (Level 2 study). No significant difference was found with regard to safety or tolerability. Further research on such pain management strategies is warranted.

7. **Encourage repositioning as a means to reduce pain, if consistent with the individual’s wishes.** (Strength of Evidence = C; Strength of Recommendation = \(\text{	extbullet	extbullet}\))

This statement is based on expert opinion. Individuals who are in pain do not wish to move, yet repositioning remains a high priority for helping to decrease pain.\textsuperscript{76} Even small changes in position are helpful in decreasing pressure. Providing adequate analgesia 20 to 60 minutes prior to planned movement may be helpful in maintaining repositioning programs.

**Reduce Procedural Pain**

1. **Use adequate pain control measures, including additional dosing, prior to commencing wound care procedures.** (Strength of Evidence = C; Strength of Recommendation = \(\text{	extbullet	extbullet}\))

This statement is based on expert opinion. Wound care procedures including wound manipulation, wound cleansing, debridement and dressing changes are painful. Topical medications are more effective when applied 20 to 30 minutes, and up to 60 minutes, prior to wound treatments.\textsuperscript{79}

2. **Consider using topical opioids (diamorphine or benzydamine 3%) to reduce or eliminate pressure ulcer pain.** (Strength of Evidence = B; Strength of Recommendation = \(\text{	extbullet} \text{	extbullet}\))

*Caution: Topically applied opioids may be associated with increased systemic side effects in individuals taking systemic opioids. Local itching and irritation has been reported, but not more frequently than when a placebo gel is applied.*\textsuperscript{80}
Opioid receptors have been found on peripheral nerves and inflamed tissue, suggesting that topically applied opioids may provide relief of pressure ulcer pain. Availability of these preparations may vary from country to country.

Flock (2003) conducted a randomized, blinded, placebo-controlled crossover pilot trial of seven hospice patients with painful Category/Stage II or III pressure ulcers to compare pre and post treatment pain using IntraSite® and diamorphine gels. Pain scores improved significantly more at one hour (p = 0.003) and 12 hours (p = 0.005) after diamorphine gel application compared with placebo and baseline (Level 2 study). A retrospective study of 15 older individuals with Category/Stage II pressure ulcers was done to assess the effectiveness of diamorphine-IntraSite® gel in relieving pain. Participants showed an improvement on the VAS of a mean of 4 points (9.4 to 4.6, p < 0.02) (Level 3 study).

Twillman et al. (1999) treated nine consecutive participants with a variety of painful skin ulcers with a topical morphine-infused gel dressing. Seven of the nine participants reported substantial relief, another participants reported a lesser (but still significant) degree of analgesia, and the ninth reported no relief for a non-open ulcer (Level 5 study).

3. Consider using topical anesthetics to reduce or eliminate pressure ulcer pain. (Strength of Evidence = C; Strength of Recommendation = 

This statement is based on expert opinion. Topical anesthetics include eutectic mixture of lidocaine and prilocaine (EMLA®, AstraZeneca, Alderley Park, UK), which is applied to the periwound area.

Manage Chronic Pain

1. Refer the individual with chronic pain related to pressure ulceration to the appropriate pain and/or wound clinic resources. (Strength of Evidence = C; Strength of Recommendation = 

2. Work with the multi-disciplinary health care team to develop a holistic plan to manage chronic pressure ulcer pain. (Strength of Evidence = C; Strength of Recommendation = 

These statements are based on expert opinion. Chronic wound pain or persistent, continuous neuropathic pain that is resistant to simple analgesia requires the development of a chronic pain management plan that may incorporate short and long term pharmacological and non-pharmacological interventions. This should be developed with input from a range of health professionals (e.g., pain specialists, medical professionals, nursing and allied health professionals), the individual and his or her caregivers.

Individualized strategies may include:

- local anesthetics;<sup>5, 83</sup>
- adjuvant medication (e.g., tricyclic antidepressants or antiepileptic);<sup>5, 83</sup> and
- non-pharmacological interventions outlined elsewhere in this section.<sup>22, 55, 72</sup>

Educate Individuals, Family and Health Care Providers

1. Educate the individual, caregivers, and health care providers about causes, assessment and management of pressure ulcer pain. (Strength of Evidence = C; Strength of Recommendation = 

The individual and his or her significant other(s) are integral to adequate management of pressure ulcer pain.<sup>84</sup> Educating the individual and family about the cause and expected duration of pain as well as what to do to minimize it can enhance understanding and compliance, and subsequently reduce pain.<sup>4, 8, 11, 15-19, 52, 84-87</sup>
Health professionals may not address pressure ulcer associated pain as a priority; however, the presence of a painful pressure ulcer is restrictive to the individual’s health related quality of life and, therefore, their convalescence.\textsuperscript{50} It is observed that within the interprofessional team, communication regarding the pressure ulcer and associated pain may be lacking.\textsuperscript{88} Balancing concurrent goals of healing the pressure ulcer and enabling participation in usual roles and activities should be considered a priority. Health professionals in the home care setting should assess for pressure ulcer pain on a regular basis and update care plans as the individual’s pain changes.\textsuperscript{88}

References

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GLOSSARY OF TERMS – WOUND CARE AND PAIN MANAGEMENT EXTRACT

Abscess: A localized collection of pus surrounded by inflamed tissue, usually due to an infective process.\(^1\)

Acute wound: A wound that progresses through the normal stages of healing without delay. In normal wound healing, signs of healing should be evident within two weeks.

Adjuvant: A substance (or therapy) that aids or heightens the action of another.

Angiogenesis: The process of developing new blood vessels from pre-existing blood vessels within the wound space; an integral part of wound healing.\(^2\)

Antibacterial: A term used to encompass antibiotics, antiseptics and disinfectants. A substance that inhibits the growth of, or eradicates, micro-organisms.\(^3, 4\)

Antibiotic: A natural or synthetic substance administered systemically or topically that has the capacity to destroy or inhibit bacterial growth.\(^1, 3, 4\)

Antimicrobial: A substance that acts directly on a microorganism to destroy the bacteria and prevent the development of new bacterial colonies.\(^5\) An antimicrobial is a broad term that includes: antiseptics, disinfectants and antibiotics.\(^6\)

Antiseptic: A substance that kills microorganisms.\(^6\)

Aseptic technique: A wound care technique that may be considered when the individual is immunocompromised; the wound enters a sterile body cavity; during the peri-operative period; or when the wound healing environment is compromised.\(^3\) Aseptic technique is designed to prevent the introduction of new microorganisms into the wound, and to reduce cross infection risk and uses sterile products and devices.

Autolysis: see Debridement.

Avascular: Lacking or without blood supply; includes necrotic tissue, slough and eschar.\(^1\)

Bacterial bioburden: The quantity of microorganisms present (e.g., planktonic bacteria or biofilm). It can be categorized as:

- Contamination: The presence of bacteria on the wound surface without bacterial multiplication\(^5\) and with no impairment to health or obvious clinical signs of infection.\(^7\)

- Colonization: The replication of microorganisms on the surface of the wound without invasion into wound tissue and without host immune response.\(^8\)

- Critical colonization (topical infection): Replication of microorganisms in low numbers of planktonic bacteria (\(\leq 10^5\) CFU/gm) and potential presence of biofilm. Bacteria and/or their products have invaded the wound surface and impaired the healing process. Clinical signs of infection may be present.\(^7\)

- Local infection: The presence of bacteria or other microorganisms in sufficient quantity to damage tissue and/or impair healing. A wound is classified as infected when the tissue contains \(\leq 10^5\) CFU/gm microorganisms per gram of tissue. Typical signs and symptoms of infection include purulent exudates, odor, erythema, warmth, tenderness, edema, pain, fever, and elevated white blood count. In some instances, clinical signs of infection may not be present, especially in the immunocompromised individual or individual with poor perfusion.\(^1\)

- Cellulitis (regional infection, spreading infection): Bacteria and/or their products have invaded
surrounding tissues causing diffuse, acute inflammation and infection of skin or subcutaneous tissues.\(^1\),\(^7\)

**Sepsis (bacteremia):** Bacteria and/or their products have entered the bloodstream. Impairment to healing occurs and the individual presents with systemic clinical signs.\(^7\)

**Bacteremia:** see *Bacterial bioburden*.

**Biofilm:** An aggregate of microorganisms known to cause chronic inflammation such as periodontal disease, surgical device infections and urinary catheter infections.\(^9\) Biofilms have enhanced resistance to destruction by endogenous antibodies and phagocytic cells, as well as by exogenous antibiotics and antiseptics. Biofilms play an important role in maintaining a chronic inflammation state ultimately leading to the failure to heal of skin wounds.\(^10\) 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*.

**Bridging:** The presence of strands of tissue across the ulcer bed.

**Callus:** Painless thickening of the skin at locations of pressure or friction, frequently seen on the foot.

**Cellulitis:** see *Bacterial bioburden*.

**Chronic wound:** A wound that does not proceed through the normal stages of healing in an orderly fashion but becomes stuck in one phase of healing.

**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.\(^3\) 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 ulcers.

**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.

**Contraction:** Pulling together wound edges in the healing process.

**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).

**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.\(^4\) 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 wounds\(^11\) and is promoted the use of moisture-retentive dressings.\(^12\)

  **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.\(^{13}\)

Conservative sharp debridement: The removal of devitalized tissue using a sharp instrument (e.g., scalpel, scissors or curette).\(^1\)

Enzymatic debridement: The removal of devitalized tissue by applying exogenous proteolytic or fibrinolytic enzymes.\(^{12}\)

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.\(^{12}\)

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.


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.

Electrical stimulation: The use of an electrical current to transfer energy controlled by an electrical source. In the prevention and treatment of pressure ulcers, 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.

Wound electrical stimulation: electrodes are usually placed over a wet conductive medium (saline soaked gauze, gel, or conductive gel) in the wound bed and on the skin a distance away from the wound or by indirectly by placing electrodes on opposite sides (bracketing) of the wound.

Muscle electrical stimulation: surface electrodes are placed over the (usually gluteal or hamstring) muscle, generally using specially designed clothing with inbuilt electrodes. The electrical current induces intermittent tetanic muscle contractions temporarily reshape the muscle and redistributing pressure.\(^{14}\)

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.

Epidermis: The outermost layer of skin.

Epithelialization: The process of becoming covered with or converted to epithelium. The new epithelial cells advance across the wound bed until they meet epithelial cells coming from the opposite direction.

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 superficial capillaries.\(^1\)

Blanchable erythema: An area of reddened skin that temporarily turns white or pale when pressure is applied to the skin. Over a pressure site, this is due to a normal hyperemic response.\(^{15}\)

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

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.\textsuperscript{1,16}

**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.\textsuperscript{1}

**Fistula:** An abnormal passage from an internal organ to the body surface or between two internal organs.\textsuperscript{1}

**Friable:** Fragile, easily injured, characteristic of newly healed tissue.

**Full thickness skin loss:** Ulceration that extends through the dermis to involve the subcutaneous tissue (Category/Stage III and IV pressure ulcers) and, if a Category/Stage IV pressure ulcer, extends into the muscle and possibly down to the bone.

**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.\textsuperscript{1}

**Growth factors:** Naturally occurring proteins or hormones that stimulate cell growth.

**Hematoma:** A collection of blood as a result of bleeding.

**Hemorrhage:** Bleeding (may be internal or external).

**Host response:** The reaction of the individual to the invasion of the microorganism.

**Hydrotherapy:** The use of a whirlpool or other submersion in water for cleansing.\textsuperscript{1}

**Hyperbaric oxygen:** Therapy in which the individual breathes 100% oxygen at pressure greater than normal atmospheric (sea-level) pressure or more than 1 atmosphere absolute (ATA).

**Induration:** Tissue that is hardened to touch.

**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.

**Intertrigo:** An erythematous skin eruption that occurs on opposing surfaces of skin (e.g., the creases of the neck, folds of the groin and armpit, or beneath pendulous breasts) from moisture, warmth, friction, and/or infectious agents. It occurs more commonly in bariatric individuals.

**Laser:** Coherent and monochromatic light, a phototherapeutic agent that is part of the electromagnetic spectrum.

**Likert scale:** An interval-based multiple-choice style question frequently used in questionnaires.

**Macerate:** To soften by wetting or soaking.

**Maggot therapy:** see *Debridement*.

**Malodor:** An offensive or disagreeable odor.

**Matrix metallopeptase (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.\textsuperscript{2}
**Medical grade honey**: Honey that is filtered, gamma irradiated and produced under exacting standards of hygiene.

**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.

**Osteomyelitis**: The inflammation of bone and bone marrow, usually caused by pathogens that enter the bone during an injury or surgery.¹

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

**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 a 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 in an injured area.

**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.¹⁷

**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.

**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

**Pressure injury**: see *Pressure ulcer*.

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

**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.¹⁸ They play a critical role in regulating the integrated hepatic acute-phase protein response.

**Protease**: A proteolytic enzyme.

**Proteolytic enzyme**: An endogenous substance such as collagenase, alastase, myeloperoxidase, acid hydrolase, and lysozymes that selectively liquefies and separates necrotic tissue and eschar from healthy
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.

Purulent: Containing pus.

Reepithelialization: The replacement of the epithelial layers of the tissue.

Sepsis: see Bacterial bioburden.

Seroma: A collection of serum/plasma within a wound.

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.

Slough: Soft, moist, devitalized (non-viable) tissue. It may be white, yellow, tan, or green, and it may be loose or firmly adherent.

Silver sulfadiazine: A silver-based, rapidly absorbed, and fairly quickly excreted antibacterial agent.

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

Tissue ischemia: The reduction of oxygen levels to below normal.

Topical antibiotic: See Antibiotic.

Surfactant: A surface active agent that reduces the surface tension of fluids to allow greater penetration.

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

Tunneling: See Sinus tract.

Ultrasound: A mechanical vibration (acoustic energy) transmitted in a wave formation at frequencies beyond the upper limit of human hearing. Its vibratory property affects the cells of biologic tissues, and 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.

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

**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 odor and minimization of pain.

**Wound dressing types:**

- **Alginate:** A highly absorbent, biodegradable dressing derived from non-woven absorptive material manufactured from seaweed. They are available in sheet and rope form.\(^1\)

- **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.

- **Collagen matrix:** 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:** A dressing that is a combination of two or more types of dressing.

- **Cover dressing:** Dressing used as the top layer to cover other absorbent dressings.

- **Fiber methylcellulose:** Highly absorbent dressing, chemically similar to a hydrocolloid.

- **Filler dressing:** Dressing material used to fill dead space in a wound bed.

- **Foam:** 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.

- **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.\(^1\)

- **Honey impregnated:** 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.

- **Hydrocolloid:** 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:** A water-based, non-adherent gel that contains hydrated hydrophilic polymers, which produce a moist environment that improves wound healing. The dressing is able to absorb excess exudates from exuding wounds but donate moisture to dry, necrotic tissue or slough. The dressing facilitates autolytic debridement.\(^1\)

- **Polymetric membrane:** 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.\(^1\)
Silicone: 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: 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.

Transparent film: A transparent dressing that is nonabsorptive and polymer-based, making it permeable to oxygen and water vapor but not to water.

Wet-to-dry saline gauze: 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.

References