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The Home Health Clinician's Field Guide to Wound Assessment & Management

Assess Systematically. Stage Accurately. Treat Purposefully.

Agenda Overview

- Understanding Wound Etiologies
- Pressure Injury Staging and Clinical Assessment
- Categories of Wound Dressings
- Evidence-Based Dressing Selection
- Negative Pressure Wound Therapy

The High Stakes of Wound Care in the Home

Diabetic Foot Ulcers (DFUs) are a common and highly morbid complication of diabetes, representing a significant challenge in home health. Mastering advanced wound care is critical to preventing devastating outcomes.

19% to 34%

The lifetime risk of a person with diabetes developing a DFU.

65%

Recurrence rate for DFUs within 3-5 years after healing.

20%

Lifetime lower-extremity amputation incidence after a DFU.

50-70%

5-year mortality rate following a DFU diagnosis, a prognosis comparable to many cancers

(Source: McDermott et al., 2023)

From Assessment to Action: Your Guide to Wound Investigation

Identifying the Culprit

Differentiating the Four Basic Wound Etiologies

Profiling the Wound

Deep Dives into Diabetic Foot Ulcers & Pressure Injuries

Choosing Your Tools

A Guide to Dressings & Advanced Therapies

Every Great Decision Starts with a Great Assessment

Your clinical assessment is the foundation for selecting the right intervention. Before choosing a dressing or therapy, evaluate these key wound characteristics:

Etiology: Is it Neuropathic, Ischemic, or Neuroischemic? The cause dictates the approach.

Dimensions: Measure size and depth to track progress. Note any undermining or tunneling.

Exudate: Quantify the amount (scant, moderate, heavy) and quality. Is the goal to add moisture or absorb it?

Tissue Type: Identify granulation, slough, or eschar. This determines the need for debridement.

Infection: Look for clinical signs. Over 20% of Wagner Grade 3 DFUs may have underlying osteomyelitis. (Source: Newman et al., 1991)

A Visual Guide to Wound Etiology

The appearance of a Diabetic Foot Ulcer provides crucial clues to its underlying cause.

Ischemic & Neuroischemic Ulcer

Often presents with irregular borders, a pale or necrotic base, or as frank gangrene. Commonly found at points of friction like toe joints.

(Source: McDermott et al., 2023)

Neuropathic Ulcer

Classic 'punched out' appearance. Typically painless, found on plantar, weight-bearing surfaces.

(Source: McDermott et al., 2023)

At-a-Glance: The Four Basic Wound Etiologies

Venous Ulcer	Arterial Ulcer	Diabetic/Neuropathic Ulcer	Pressure Injury
Location: Medial lower leg, ankle (gaiter area).	**Location:** Toes, feet, bony prominences.	**Location:** Plantar aspect of foot, pressure points.	**Location:** Over bony prominences (sacrum, heels, hips).
Wound Bed: Ruddy, granular, shallow.	**Wound Bed:** Pale, eschar, "punched-out" look, minimal granulation.	**Wound Bed:** Pink, pale, may have eschar, often surrounded by callus.	**Wound Bed:** Varies by stage; may have slough or eschar.
Exudate: Moderate to heavy.	**Exudate:** Minimal.	**Exudate:** Variable.	**Exudate:** Variable.
Periwound: Edema, hemosiderin staining, stasis dermatitis.	**Periwound:** Thin, shiny, dry skin; hair loss; dependent rubor.	**Periwound:** Callused rim, dry, cracked skin (autonomic neuropathy).	**Periwound:** May be non-blanchable erythema, indurated, or discolored.

Chapter 1 Deep Dive: Decoding the Diabetic Foot Ulcer (DFU)

A DFU is defined as a **break of the epidermis** and at least part **of the dermis** in a person with diabetes.

Lifetime Risk: 19% to 34% of people with diabetes will develop a DFU.

5-Year Mortality: 50–70% after an incident ulcer.

Neuropathic (35%): Caused by sensory loss.
Ischemic (15%): Caused by peripheral artery disease.
Neuroischemic (50%): A combination of both.

Classic Neuropathic Ulcer

Classic Neuroischemic Ulcer

DFU Pathophysiology: The Dual Threat of Neuropathy and Ischemia

Peripheral Neuropathy (PN)

The Insensate Foot

Sensory Loss: Loss of protective sensation leads to unrecognized trauma.

Motor Deficit: Muscle wasting leads to foot deformities (e.g., clawed toes) and new pressure points.

Autonomic Dysfunction: Reduced sweating causes dry, fragile, cracking skin.

"Neuropathy of any kind confers an approximate sevenfold risk of DFU."

Peripheral Artery Disease (PAD)

The Ischemic Foot

Affects smaller, infrapopliteal arteries.

Characterized by medial arterial calcification.

Diagnostic Challenge: Calcification makes standard ankle-brachial index (ABI) unreliable.

"PAD is a contributing factor in 50% to 70% of DFUs."

DFU Grading: The Wagner Classification System At-a-Glance

1 Superficial Ulcer

Partial or full-thickness ulcer confined to skin.

2 Deep Ulcer

Ulcer extends to ligament, tendon, joint capsule, or deep fascia without abscess or osteomyelitis.

3 Deep Ulcer with Abscess or Osteomyelitis

Deeper extension with bone involvement or abscess formation.

4 Localized Gangrene

Gangrene of a digit or part of the forefoot. Described as potentially "wet (swelling, odor, discharge) or dry (blackened, mummified, hard)."

5 Extensive Gangrene

Gangrene of the entire foot, requiring disarticulation.

A Clinician's Quick Guide to Lower Extremity Ulcers

A visual comparison of classification systems, causes, and key characteristics for pressure injuries, diabetic foot ulcers, and venous vs. arterial ulcers.

ULCER CLASSIFICATION SYSTEMS

Pressure Injury Staging (NPIAP)

Stage 1
Intact skin with non-blanchable redness.

Stage 2
Partial-thickness loss with exposed pink/red dermis.

Stage 3
Full-thickness skin loss; fat is visible.

Stage 4
Full-thickness skin & tissue loss; exposed bone, tendon, or muscle.

Unstageable
Depth obscured by slough or eschar.

Deep Tissue Injury
Persistent deep red, maroon, or purple discoloration.

Based on the depth of tissue loss

Diabetic Foot Ulcer Grading (Wagner)

Grade 0
Intact skin, "at-risk" foot with bony deformity.

Grade 1
Superficial ulcer of skin or subcutaneous tissue.

Grade 2
Ulcer extends to tendon, capsule, or bone.

Grade 3
Deep ulcer with abscess or osteomyelitis.

Grade 4
Gangrene of the forefoot or toes.

Grade 5
Gangrene of the entire foot.

Based on ulcer depth, infection,

VENOUS VS. ARTERIAL ULCERS

Venous Ulcers

The Most Common Leg Ulcer

Caused by chronic venous insufficiency leading to venous hypertension.

Arterial Ulcers

A Sign of Ischemia

Caused by peripheral artery disease (PAD) reducing blood supply.

DISTINGUISHING CLINICAL SIGNS

Venous Ulcers			Arterial Ulcers
Venous insufficiency, reflux		Cause	Peripheral Artery Disease (PAD)
Medial lower leg, "gaiter area"		Location	Toes, feet borders, bony prominences
Irregular edges, granulation tissue		Appearance	"Punched-out" appearance, pale base
Aching/heaviness, relieved by elevation		Pain	Severe, agonizing pain
Edema, pigmentation (staining), dermatitis		Associated Signs	Cool skin, pale/cyanotic color, absent puises

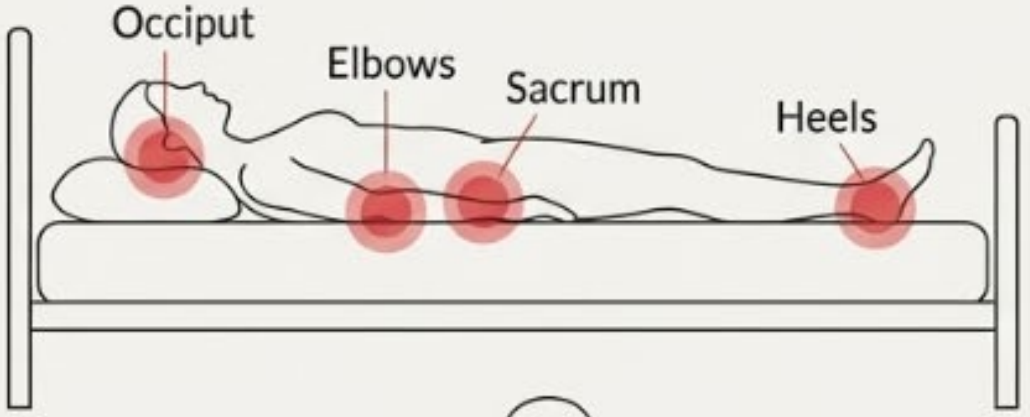
Chapter 2 Deep Dive: Investigating the Pressure Injury (PI)

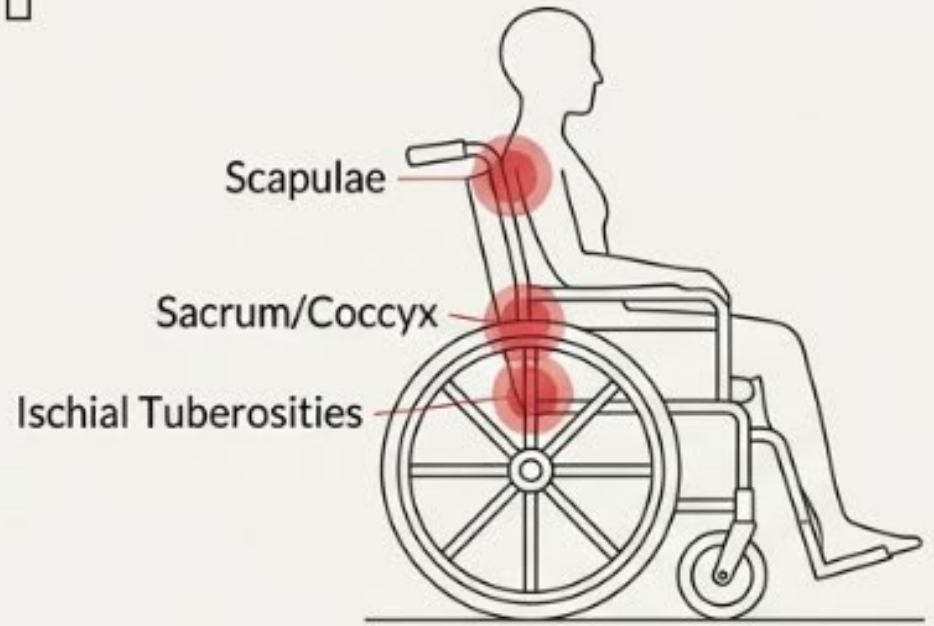
Definition

"A pressure injury is localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device."

Primary Mechanism

The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear.





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Improving Patient Outcomes Through Education, Research and Public Policy

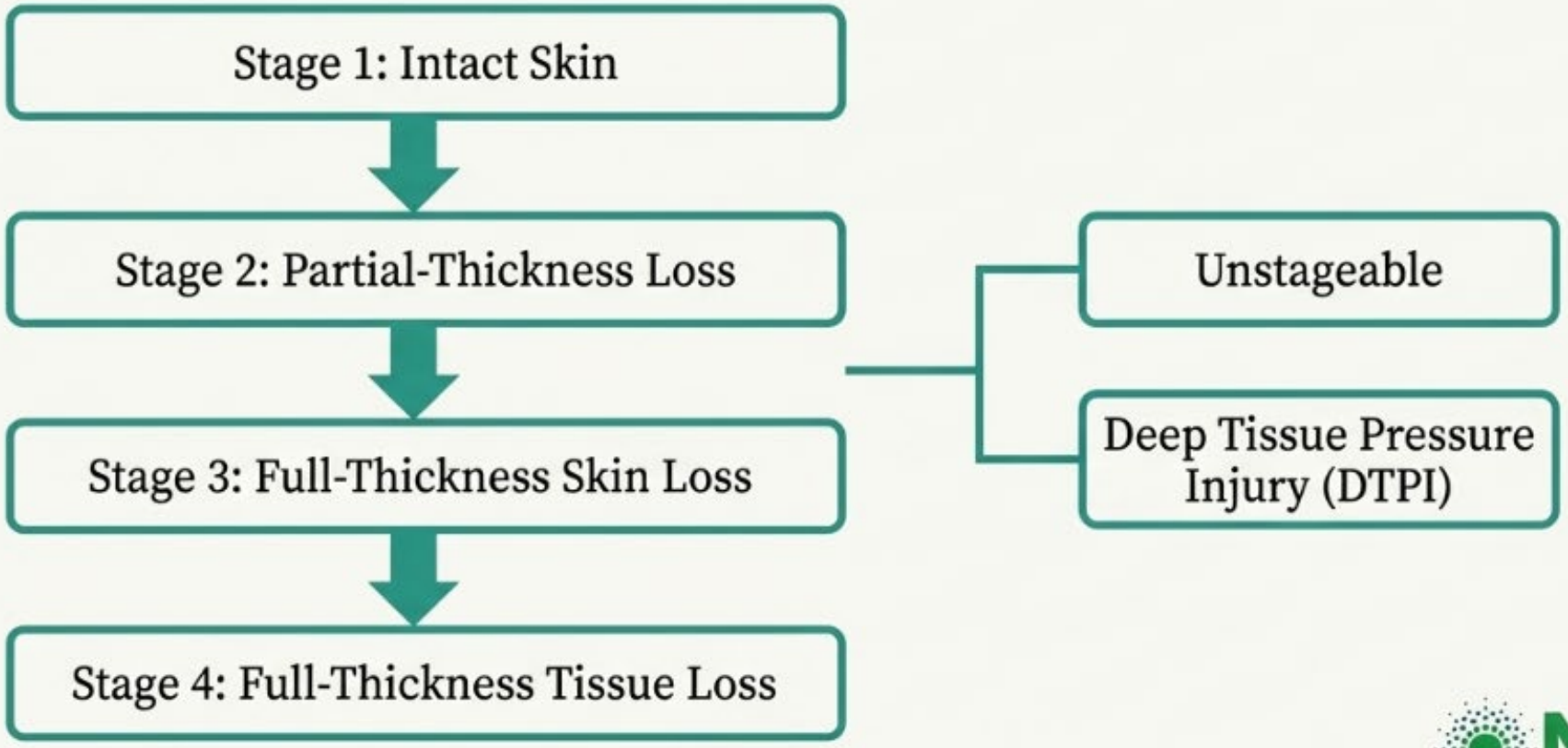
Mastering Pressure Injury Staging

A Practical Guide for Home Health Nurses

Applying the 2019 NPIAP Clinical Practice Guidelines

The NPIAP Framework: A Common Language for Wound Care

The National Pressure Injury Advisory Panel (NPIAP) provides the authoritative, evidence-based system for classifying pressure injuries. This system creates a consistent terminology for assessing and documenting wound severity, ensuring clear communication across the care team.



Source: NPIAP/EPUAP/PPPIA Clinical Practice Guideline, 2019.

NPIAP

Stage 1: Non-Blanchable Erythema of Intact Skin

Stage 1 Pressure Injury: Sacrum

Official NPIAP Definition

Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue.

Key Clinical Descriptors

- Skin is unbroken.
- Redness persists and does not turn white when pressed (non-blanchable).
- Changes in sensation, temperature, or firmness may be the first signs.

Home Health Nurse's Note

In darkly pigmented skin, look for changes in color tone (e.g., purplish, bluish) compared to surrounding skin. Palpate for changes in temperature and tissue consistency **(induration). This is your critical window for prevention and early intervention.**

Stage 2: Partial-Thickness Skin Loss with Exposed Dermis

Stage 2 Pressure Injury: Heel

Official NPIAP Definition

Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister.

Key Clinical Descriptors

- Shallow ulcer with a red-pink, moist wound bed.
- No slough, eschar, or granulation tissue is visible. Adipose tissue is not visible.
- Presents as an abrasion or ruptured blister.

Home Health Nurse's Note

This stage should not be used to describe skin tears, tape burns, perineal dermatitis, or maceration. Accurate differentiation is crucial for selecting the appropriate treatment and avoiding misclassification.

NPIAP

Stage 3: Full-Thickness Skin Loss

Stage 3 Pressure Injury: Sacrum

Official NPIAP Definition

Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible.

Key Clinical Descriptors

- Adipose tissue is visible, but fascia, muscle, tendon, or bone are NOT exposed.
- Slough or eschar may be present but does not obscure the depth of tissue loss.
- Undermining and tunneling may be present.

Home Health Nurse's Note

The depth of a Stage 3 injury varies by anatomical location. An injury on the ear will be shallow, while one on an area with significant adiposity can be extremely deep. Always probe gently for undermining and tunneling to understand the full extent of the wound.

NPIAP

Stage 4: Full-Thickness Skin and Tissue Loss

Stage 4 Pressure Injury: Trochanter

Official NPIAP Definition

Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often occur.

Key Clinical Descriptors

- Exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone.
- Slough or eschar can be present.
- Undermining and tunneling are common.

Home Health Nurse's Note

Exposed bone is an automatic Stage 4 and places the patient at high risk for osteomyelitis. This finding requires prompt communication with the primary physician and likely a surgical or infectious disease consultation.

NPIAP

Unstageable: Obscured Full-Thickness Skin and Tissue Loss

Unstageable Pressure Injury: Heel

Official NPIAP Definition

Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar.

Key Clinical Descriptors

- Wound base is covered by slough (yellow, tan, gray, green, or brown) or eschar (tan, brown, or black).
- The true depth, and therefore stage, is completely obscured.

Home Health Nurse's Note

Crucial distinction: Do not remove stable, dry, adherent eschar on an ischemic limb or heel. It acts as a natural biological cover. Debridement is only necessary once the base is visible or if signs of infection develop.

NPIAP

Deep Tissue Pressure Injury (DTPI): Persistent, Non-Blanchable Deep Discoloration

Deep Tissue Pressure Injury: Heel

Official NPIAP Definition

Intact or non-intact skin with a localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister.

Key Clinical Descriptors

- Discoloration persists when pressed (non-blanchable).
- Pain and temperature changes often precede color changes.
- May evolve rapidly, exposing additional tissue layers, or may resolve without tissue loss.

Home Health Nurse's Note

A DTPI is not a Stage 1 injury. This presentation signals severe underlying tissue damage. Document it as a DTPI and monitor vigilantly for deterioration, as it can quickly become a full-thickness wound.

NPIAP

Beyond the Bed: Staging Medical Device-Related Injuries

- These injuries result from pressure or shear from devices used for diagnostic or therapeutic purposes.
- The injury's shape or pattern typically conforms to the device.

Oxygen Tubing Injury

Urinary Catheter Strap Injury

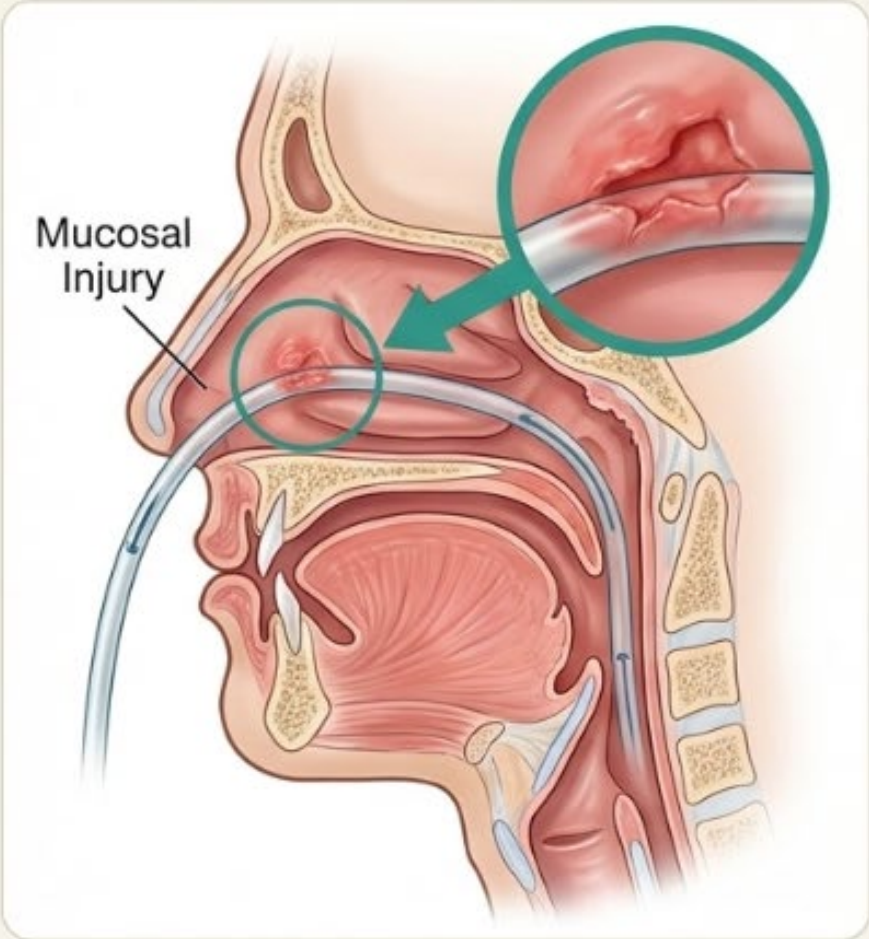
BiPAP Mask Injury

- **How to Stage*:** Use the exact same NPIAP staging system (Stages 1-4, Unstageable, DTPI) to classify the injury. For example, a non-blanchable red line from a catheter is a "Stage 1 Medical Device-Related Pressure Injury."

Home Health Nurse's Note

Proactive assessment is vital. Regularly check the skin under and around all devices. Use prophylactic dressings, such as soft silicone foam, to pad high-risk areas.

A Special Case: Mucosal Membrane Pressure Injuries



- Found on mucous membranes (e.g., nasal passages, mouth, urethra) with a history of medical device use at the site.
- **These injuries cannot be staged** using the standard pressure injury system because mucosal tissue is anatomically different from skin.
- **Documentation:** Describe the injury as a "Mucosal Membrane Pressure Injury" and note its location and appearance.

Home Health Nurse's Note

Common in home care with feeding tubes, oxygen cannulas, and catheters. While you don't stage them, you must document their presence, assess for pain, and monitor for signs of infection.

NPIAP

Is It a Pressure Injury? A Comparative Guide

Pressure Injury	Venous Ulcer	Diabetic Foot Ulcer
		
• Located over a bony prominence. • Caused by pressure +/- shear. • Caused by pressure +/- shear. • Shape is often round or oval.	• Located on the lower leg ('gaiter area'). • Irregular shape, shallow, heavy exudate. • Associated with edema, hemosiderin staining (brown skin).	• Located on weight-bearing surfaces of the foot. • Surrounded by callus. • Patient has neuropathy.

Different Tools for Different Wounds: NPIAP vs. Wagner Scale

NPIAP Staging System

NPIAP staging system can use from pressure and shear. As in Source Serif Pro.

- **Use For:** Assessing tissue damage from pressure and shear.
- **Describes:** The depth of tissue destruction (partial vs. full-thickness).
- **Applies To:** Any pressure injury on any patient.

Wagner Ulcer Classification

Wagner ulcer classification can use foot ulcers . in Source Serif Pro.

- **Use For:** Grading diabetic foot ulcers.
- **Describes:** Depth of the ulcer plus the presence of infection, ischemia, and gangrene.
- **Applies To:** Foot ulcers in patients with diabetes.

Home Health Nurse's Note

A patient can have a Stage 2 pressure injury on their sacrum and a Wagner Grade 2 ulcer on their foot. They are distinct problems and must be assessed and documented using the correct system for each.

Your Pressure Injury Staging Toolkit: Key Principles

Assess Holistically: Look beyond the wound to the patient's risk factors and overall condition.

Location is Key: Pressure injuries are almost always over a bony prominence.

Determine the Deepest Visible Tissue: This defines the stage (Adipose = Stage 3; Bone/Muscle/Tendon = Stage 4).

If the Base is Obscured, It's Unstageable: Never guess the depth beneath slough or eschar.

Deep Red, Maroon, or Purple ≠ Stage 1: This signals a potential Deep Tissue Pressure Injury (DTPI).

Document with Precision: Use the official NPIAP terminology to ensure clarity and consistency.

The Gold Standard Resource

This presentation is based on the 2019 International Clinical Practice Guideline. For comprehensive evidence summaries, implementation considerations, and detailed recommendations, always refer to the full guideline.

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European Pressure Ulcer Advisory Panel (EPUAP)
National Pressure Injury Advisory Panel (NPIAP)
Pan Pacific Pressure Injury Alliance (PPPIA)

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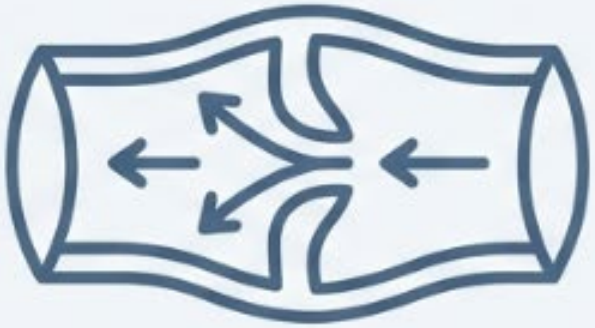
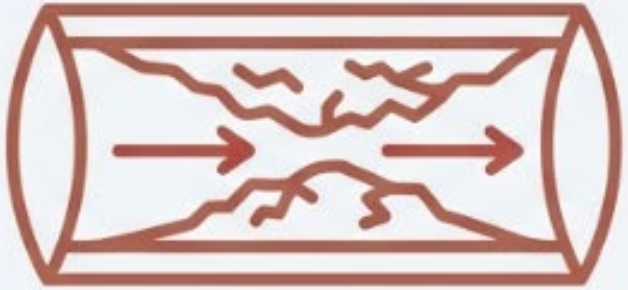
Every Leg Ulcer Presents a Fork in the Road

As a home health nurse, you are the first line of defense and the patient's most trusted guide. A new lower extremity ulcer means the patient is at a critical juncture. Choosing the right path is essential for healing; choosing the wrong path can cause significant harm. This guide is designed to sharpen your diagnostic skills to confidently navigate these two distinct paths.



The Venous Path: A **Drainage** Problem

Caused by venous hypertension and valve failure. Blood can't get out effectively.



The Arterial Path: A Supply Problem

Caused by peripheral artery disease (PAD). Oxygenated blood can't get in effectively.

At-a-Glance: Differentiating Venous vs. Arterial Ulcers

Feature	Venous Ulcer	Arterial Ulcer
Location	Medial side of the leg, typically around the medial malleolus (the "gaiter area").	Lateral side of the leg, over bony prominences, tips of toes, or heels.
Appearance	Shallow with irregular, sloping edges. Wound bed is often red.	Deep, with a "punched-out" appearance. Wound bed can be pale, gray, or necrotic (black eschar).
Exudate	Typically "wet" with moderate to heavy exudate.	Typically "dry," with minimal to no exudate. May be covered by a scab or eschar.
Pain Profile	Moderate pain, often described as aching or heavy. Pain **improves** on elevation.	Often severe, sharp pain, especially at night. Pain **worsens** with elevation (intermittent claudication).
Surrounding Skin	Edematous (swollen). May show lipodermatosclerosis (hardened skin), hyperpigmentation (hemosiderin staining), and atrophie blanche.	Pale, cool to the touch, shiny, and hairless.

Reading the Wound: Visual Cues in the Home Setting

Venous Ulcer

edial "gaiter
ea" location

Irregular, sloping borders

Moderate exudate present

Hemosiderin staining on surrounding skin

Evidence of edema

Arterial Ulcer

Located on pressure point (toe)

Deep, "punched-out" appearance

Minimal exudate, dry wound bed

Pale, shiny periwound skin

Trophic changes (hair loss)

Confirming Your Assessment: The Ankle-Brachial Index (ABI)

The critical step before considering compression.

The ABI compares the blood pressure at the ankle to the blood pressure in the upper arm. A low ABI can indicate blockage or narrowing of the arteries in the legs (Peripheral Artery Disease - PAD), making compression therapy dangerous.

The Formula

$$\frac{\text{Highest Ankle Systolic Pressure}}{\text{Highest Arm Systolic Pressure}} = \text{ABI}$$

The Interpretation Scale

< 0.5	< 0.9	0.9 - 1.3	> 1.3
Severe PAD. Compression is contraindicated. Urgent vascular referral needed.	**Indicates Peripheral Artery Disease (PAD). Compression should be applied with extreme caution or not at all.**	Normal range.	Suggests non-compressible, calcified vessels.

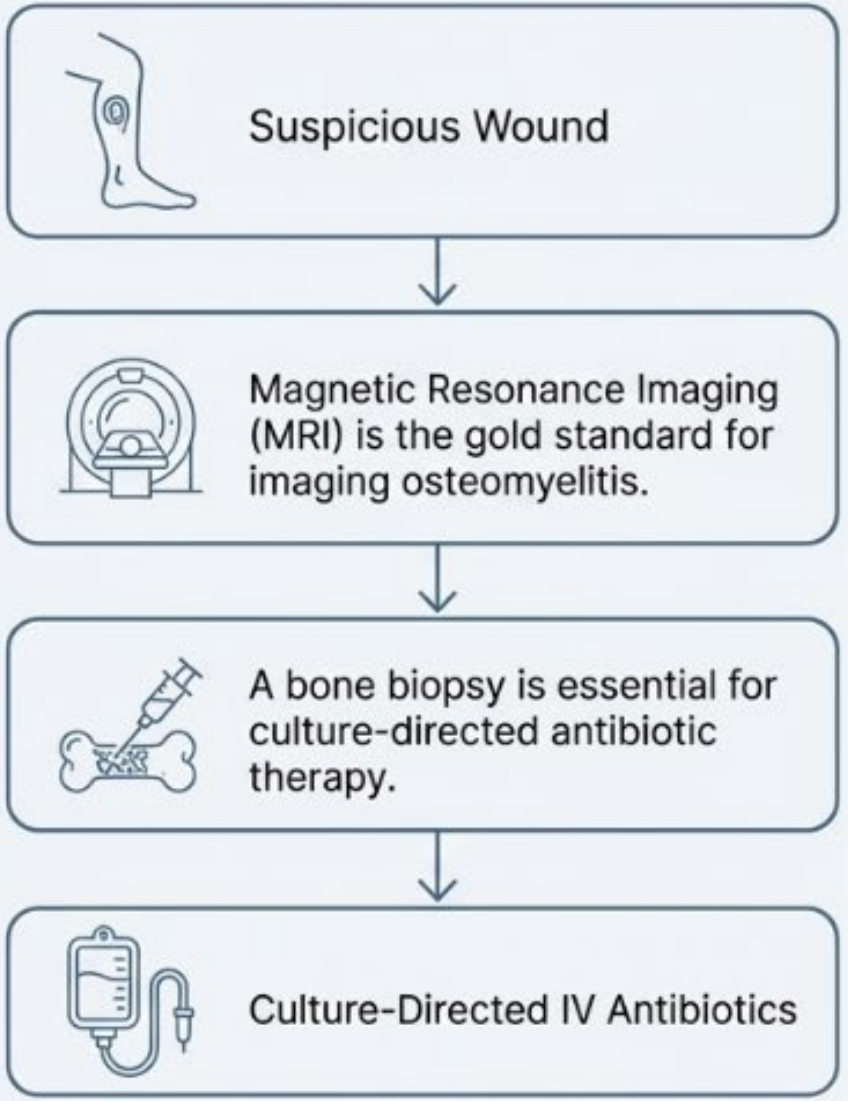
Looking Deeper: When to Suspect Osteomyelitis

A non-healing ulcer, especially one that is deep, has failed to improve after 4-6 weeks of appropriate care, or has exposed bone, should raise a high index of suspicion for osteomyelitis (bone infection). Healing is unlikely until the infection is controlled. Staphylococcus aureus is the most common pathogen.

Key Indicators

- **Probe-to-bone Test**: Bone feels rough or soft when gently probed.
- **Non-healing Wound**: Fails to show signs of healing despite optimal care.
- **Chronic Drainage**: Persistent or reopened draining sinus.

Diagnostic Pathway



The Venous Treatment Pathway: Manage the Drainage, Heal the Ulcer

The goal is to counteract venous hypertension.
Compression is the cornerstone of therapy.

Compression Therapy

Use multi-layer elastic bandages or specifically designed compression boots/stockings. Counters the impact of venous reflux and reduces edema.

Elevation

Encourage the patient to elevate the leg above the heart whenever possible to reduce pressure.

Exercise

Prescribed exercise, in conjunction with compression, is proven to increase healing rates. Focus on ankle/calf muscle pump activation.

Medication

Pentoxifylline can be a useful adjunctive treatment to improve blood flow.

The Arterial Treatment Pathway: Protect the Supply, Save the Limb

DO NOT COMPRESS an ischemic limb.

The goal is to protect the ischemic limb from further injury and facilitate revascularization.

Protection

Offload pressure completely. Use heel suspension devices if applicable. Protect from trauma, friction, and extreme temperatures.

Wound Care Pro-Tip

Do NOT debride stable, hard, dry eschar on ischemic limbs or heels.

This eschar serves as a natural, protective cover. Keep the area clean and dry to prevent infection.

Referral

Urgent referral to a vascular specialist is the primary goal.

The patient may need procedures (angioplasty, stenting, stenting, bypass) to restore blood flow.

Red Flags: When to Escalate Care Immediately

Call your clinical manager or the patient's physician/specialist if you observe:

Signs of Spreading Infection

Signs of Spreading Infection

- Advancing erythema (redness) or cellulitis extending from the ulcer edge.
- Increased warmth, pain, or swelling.
- Purulent drainage or foul odor.
- Systemic signs: Fever (>101°F), malaise, confusion (especially in older adults).

Signs of Critical Limb

Signs of Critical Limb Ischemia

- Sudden, severe increase in pain.
- Leg becomes pale, cold, or numb.
- Loss of pulse (if able to assess).

Signs of Tissue Death

Signs of Tissue Death

- Rapidly expanding area of black, necrotic tissue.
- Development of wet gangrene (swelling, odor, discharge).

You Are the Guide on the Path to Healing

Venous Path

ASSESS: Medial, shallow, wet, edematous.

DIAGNOSE: Confirm with ABI > 0.9.

TREAT: COMPRESS, Elevate, Exercise.

Arterial Path

ASSESS: Lateral/toes, deep, dry, shiny/hairless.

DIAGNOSE: Suspect with ABI < 0.9.

TREAT: DO NOT COMPRESS, Protect, Refer Urgently.

Your expert assessment and decisive action set the course for healing. By correctly identifying the path—venous or arterial—you not only treat the wound but also protect your patient from harm and guide them toward recovery.

The Home Health Clinician's Toolkit: Mastering Advanced Wound Care

A Practical Guide to Dressing Selection and Negative Pressure Wound Therapy

Chapter 3: The Clinician's Toolbox – Wound Bed Preparation

Cleansing

"Cleanse the pressure injury at each dressing change. Use cleansing solutions with antimicrobials only for pressure injuries with suspected or confirmed infection." (NPIAP, 12.1, 12.2)

Debridement

"Debride the pressure injury of devitalized tissue (slough, eschar) and suspected biofilm. Perform maintenance debridement until the wound bed is covered with healthy granulation tissue." (NPIAP, 12.5)

The Ischemic Exception

AVOID disturbing stable, hard, dry eschar in ischemic limbs and heels, unless infection is suspected. (NPIAP, 12.4)

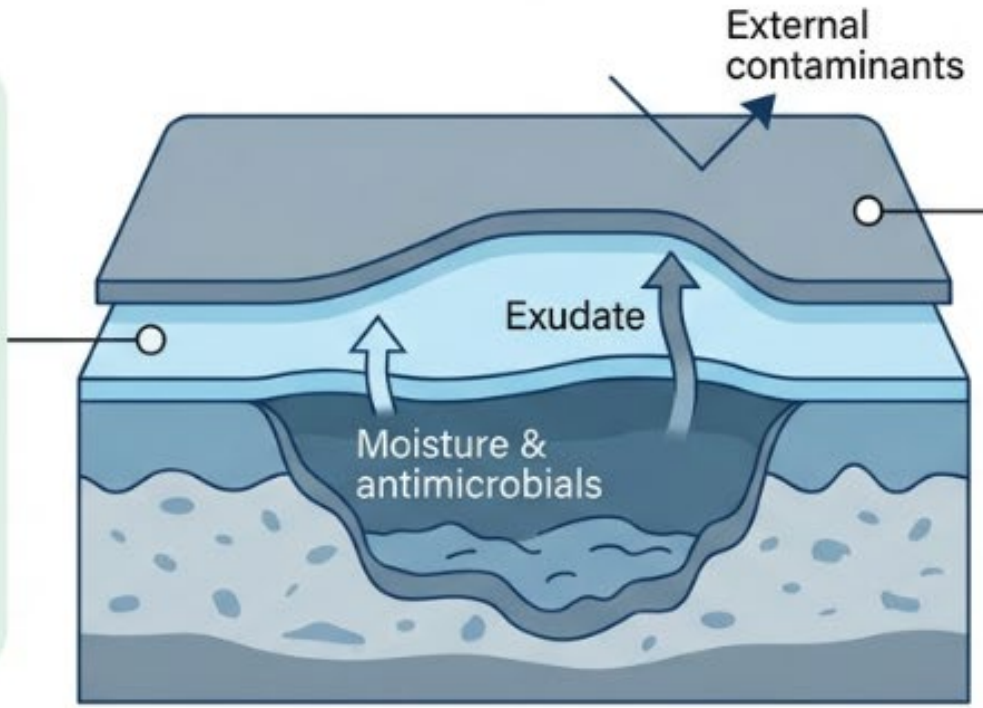
The System of Care: Primary vs. Secondary Dressings

PRIMARY DRESSING

The Therapeutic Layer.

Contact: Direct wound bed.

Function: Donates moisture, absorbs exudate, or delivers antimicrobials.



SECONDARY DRESSING

The Structural Layer.

Contact: Covers primary.

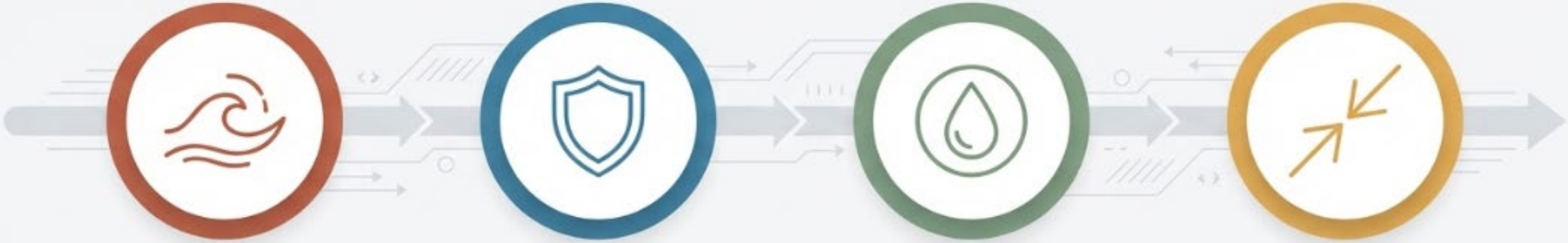
Function: Secures placement, absorbs strike-through, protects from contaminants.

Key Distinction:

Clinical Note: Some dressings can act as both (e.g., adhesive foams), while others (e.g., hydrogels, alginates) always require a secondary cover.

A Framework for the Journey: The TIME Pathway for Wound Bed Preparation

Effective wound management isn't about memorizing a catalog of products. It's a strategic process. The TIME framework provides a clinical pathway to guide your decisions, addressing the key obstacles to healing in a logical sequence.



T - Tissue: Managing Non-Viable Tissue

I - Infection/Inflammation: Controlling Bioburden

M - Moisture: Achieving Moisture Balance

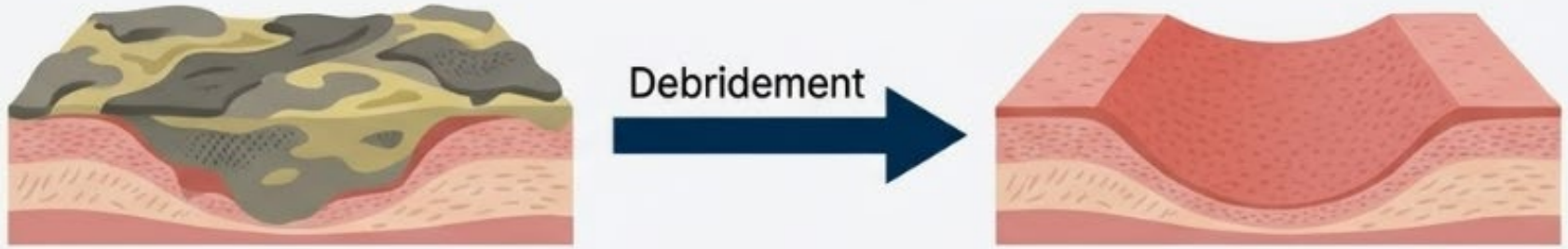
E - Edge: Promoting Epithelial Advancement

We will follow this journey, introducing the right dressings as the specialized tools needed at each step to move the wound toward closure.

T - TISSUE MANAGEMENT

Preparing the Foundation: Preparing the Clearing the Way for Healing — for Healing

A wound cannot heal if the bed is covered with non-viable tissue. Necrotic tissue and slough act as a physical barrier to granulation, a food source for bacteria, and a mask for underlying infection. The first step on the journey is to prepare a clean foundation.



Non-Viable Wound Bed

Prepared Wound Bed

- **Goal:** Remove devitalized tissue (slough, eschar) to reveal a healthy, granulating wound bed.
- **Methods:** This can be achieved through various means, from surgical debridement to autolytic debridement, which is facilitated by certain dressings.

Critical Consideration: "Avoid disturbing stable, hard, dry eschar in ischemic limbs and heels, unless infection is suspected." (Source: NPIAP Guidelines, 2019).

T - TISSUE MANAGEMENT

The Right Tools for Tissue Management

Santyl (Collagenase Ointment)

- **Mechanism of Action:** An enzymatic debriding agent that selectively digests the collagen anchoring necrotic tissue to the wound bed.
- **Primary Use:** Debridement of chronic dermal ulcers and severely burned areas. Effective on both slough and eschar.
- **Clinical Considerations:** Requires a moist environment to be active. Inactivated by certain heavy metals like silver. Change dressing daily.

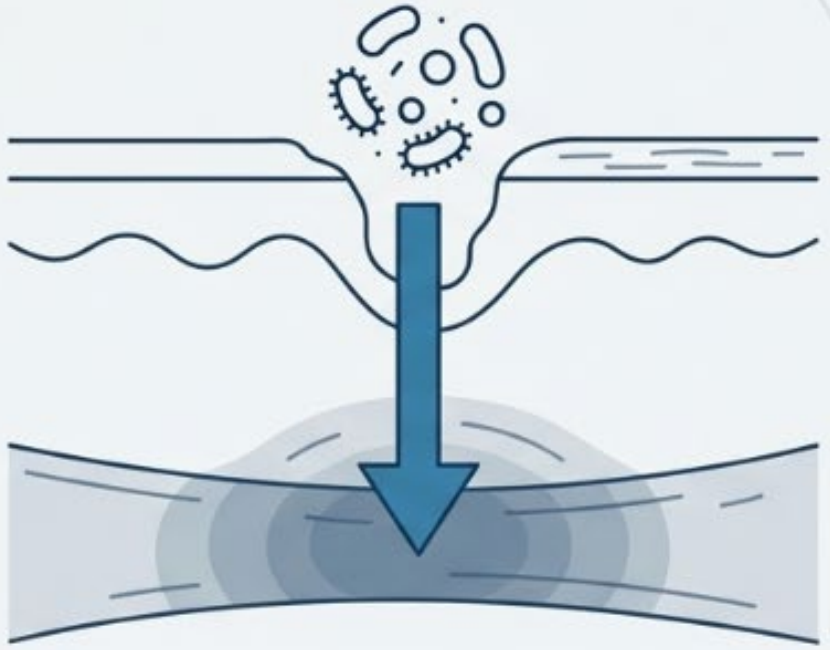
Hydrogels

- **Mechanism of Action:** Donates moisture to dry, necrotic wounds, softening eschar and facilitating autolytic debridement (the body's own enzymatic process).
- **Primary Use:** Dry or minimally exudative wounds requiring hydration. Ideal for softening hard, black eschar.
- **Clinical Considerations:** Available as amorphous gel, sheets, or impregnated gauze. Requires a secondary cover dressing. Can cause maceration if applied to surrounding skin.

I - INFECTION & INFLAMMATION

Gaining Control of Bioburden

The presence of bacteria is a given, but an uncontrolled bacterial load (biofilm, critical colonization, or frank infection) stalls healing, increases inflammation, and can lead to devastating complications. Your goal is to regain control and create an environment where healing can resume.



The Link to Deeper Problems

- **Osteomyelitis**: "Wounds that fail to close, close but reopen, have draining sinuses...warrant a high index of suspicion for osteomyelitis." Bone infection is a primary cause of non-healing wounds. (Source: StatPearls - Hyperbaric Treatment).
- **Evaluation**: If infection is suspected, evaluate for exposed bone or a rough/soft feel on probing. Consider the need for deeper cultures and imaging (MRI is the gold standard). (Source: NPIAP Guidelines, StatPearls).

I - INFECTION & INFLAMMATION

The Antimicrobial & Anti-Inflammatory Toolkit

Silver Dressings

Mechanism: Broad-spectrum; silver ions (Ag+) are released in the presence of exudate, binding to and disrupting bacterial cells.

Use: For infected wounds or those at high risk of infection. Amount of silver released is often proportionate to exudate level.

Cadexomer Iodine

Mechanism: A slow-release formulation that absorbs exudate and debris while providing sustained, broad-spectrum antimicrobial action.

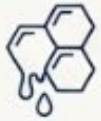
Use: Available as gel, ointment, or pad. Highly effective in sloughy, exudative wounds.

Methylene Blue & Gentian Violet

Mechanism: Antiseptic dyes with broad antimicrobial/antifungal properties and anti-biofilm effects. Strong affinity for dead cells and protein-rich exudate.

Use: Often impregnated in foam dressings. Helps reduce bacterial load and prevent biofilm formation.

Honey (Medical Grade)



Mechanism: Multiple actions: osmotic effect dehydrates bacteria, low pH inhibits growth, and hydrogen peroxide production provides antimicrobial activity. Also has anti-inflammatory properties.

Use: Impregnated into various dressing formats. Effective for a wide range of wound types.

DACC (Dialkylcarbamoyl Chloride)

Mechanism: A unique mechanism that irreversibly binds bacteria to the dressing fibers through hydrophobic interaction, removing them from the wound with each dressing change.

Use: Does not kill bacteria, reducing risk of endotoxin release. For low to high exudating wounds.

M - MOISTURE BALANCE

The ‘Goldilocks’ Principle: Not Too Wet, Not Too Dry

Healing cells—fibroblasts and keratinocytes—need a moist environment to migrate and function. However, excessive moisture (exudate) can damage periwound skin (maceration) and increase bioburden. The goal is to achieve a perfect balance, customized to the wound's needs.

Too Dry (Desiccated)

Balanced
Balanced (Moist Wound Healing)

Too Wet (Macerated)

The Two Core Tasks:

1. **Absorb Excess Exudate**: For wounds that are producing too much fluid.
2. **Donate or Maintain Moisture**: For wounds that are too dry.

M - MOISTURE BALANCE

The Right Tools for High Exudate: Absorbers

Foams

Mechanism of Action: Polyurethane foam layers absorb and hold large amounts of exudate, maintaining a moist environment while protecting the periwound skin.

Primary Use: Moderate to heavy exudating wounds. Can be used as a primary or secondary dressing.

Clinical Considerations: Available in various thicknesses and sizes, with or without adhesive (often silicone) borders. Provides thermal insulation and cushioning over bony prominences.

Alginates

Mechanism of Action: Derived from brown seaweed, these fibers interact with wound exudate to form a soft gel. This gelling action traps exudate and bacteria.

Primary Use: Moderate to heavy exudating wounds. Ideal for packing deep or tunneling wounds.

Clinical Considerations: Available as pads or ropes. Always requires a secondary cover dressing. Should not be used on dry wounds as they will desiccate the wound bed.

M - MOISTURE BALANCE

The Right Tools for Dry Wounds: Hydrators & Protectors

Hydrogels (Revisited)

Donate Moisture

The primary choice for rehydrating dry wound beds covered in slough or eschar to facilitate autolytic debridement.

Hydrocolloids

Maintain Moisture

For low to moderate exudating wounds. The occlusive wafer interacts with exudate to form a gel, maintaining a moist environment and promoting autolysis. Not for use on infected wounds.

Transparent Films

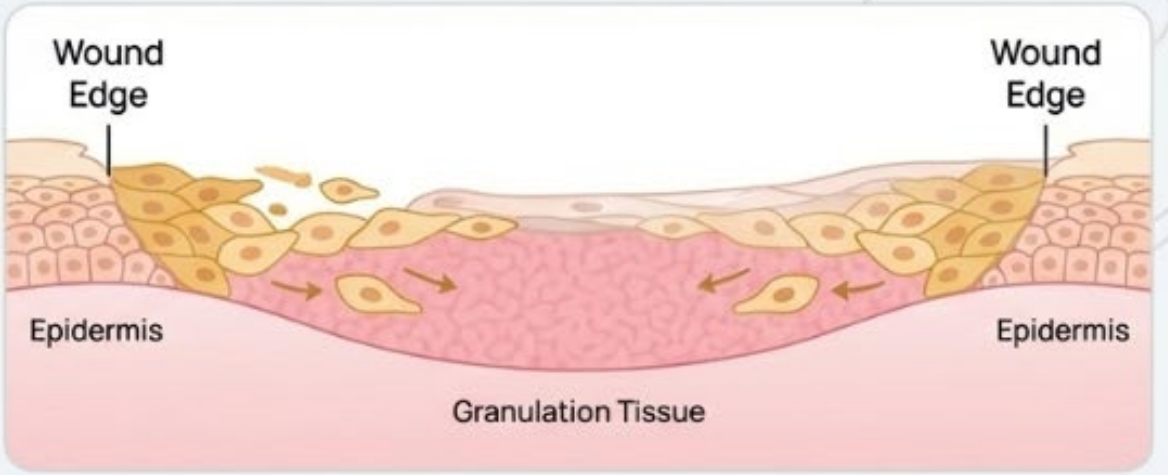
Maintain Moisture (Barrier)

For superficial wounds with little to no exudate (e.g., Stage 1 Pressure Injuries, skin tears, secondary dressing cover). The semi-permeable membrane allows moisture vapor to escape but prevents bacterial entry.

E - EDGE ADVANCEMENT

Supporting the Final Steps to Closure

As the wound bed fills with granulation tissue, the final phase is epithelialization—the migration of keratinocytes across the wound surface. The wound edge (or "rim") and the newly formed tissue are fragile and require protection from trauma and desiccation.



Key Goals at this Stage:

Protect: Shield the wound bed and new epithelial cells from the trauma of dressing changes.

Promote: Provide a structure or environment that supports cell migration.

Manage: Continue to manage exudate without sticking to the wound bed.

E - EDGE ADVANCEMENT

The Right Tools for Protecting the Edge

Contact Layers

Mechanism of Action

A thin, porous, non-adherent sheet placed directly on the wound bed. Protects the tissue from trauma while allowing exudate to pass through to a secondary absorbent dressing.

Primary Use

On any wound where protection from sticking is a priority, including skin grafts, donor sites, and granulating wounds.

Clinical Considerations

Often made of silicone or other flexible materials. Can be left in place for several days.

Collagens

Mechanism of Action

Provides a natural collagen scaffold that encourages the deposition of new tissue and cell migration. May help modulate protease activity in chronic wounds.

Primary Use

Stalled, non-healing wounds that are clean and granulated.

Clinical Considerations

Available as powders, gels, pads, and pastes. Requires a secondary dressing.

Silicone Gel Sheets

Mechanism of Action

Soft, self-adherent sheets that hydrate and occlude scar tissue.

Primary Use

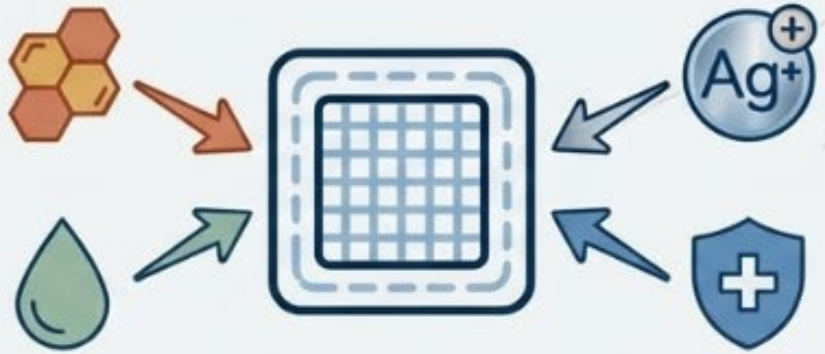
Primarily for post-closure scar management to improve appearance and pliability. Can also protect fragile, newly healed skin.

Clinical Considerations

Used after complete epithelialization. Reusable.

A Versatile Category: Impregnated Dressings

'Impregnated Dressing' is a broad classification rather than a single type. It refers to any dressing material—such as gauze, foam, or non-woven fabric—that has been saturated with a therapeutic **therapeutic** agent. You are already using many of them.



- **Antimicrobials:** Gauze or foam impregnated with agents like honey, iodine, or methylene blue/gentian violet.
- **Moisture Management:** Gauze impregnated with petrolatum (non-adherent) or amorphous hydrogel (moisture donation).
- **Debridement:** Dressings impregnated with saline to facilitate moistening and gentle debridement.

Clinical Pearl: The key is to select the dressing based on the ***action of the impregnated agent*** and the needs of the wound, following the TIME principles.

The Wound Bed Journey: A Decision Support Tool

		Alginates	Antimicrobials	Collagens	Contact Layers	Foams	Hydrocolloids	Hydrogels	Santyl	Transparent Films
TISSUE										
#C05746	Dry Eschar / Necrotic						○	●	●	
	Sloughy / Loose Debris	●	● Antimicrobials (Cadexomer Iodine)					○	●	
INFECTION										
#3D84A8	High Bioburden / Infected		●							
MOISTURE	High Exudate	●				●				
#7A9E7E	Low/Moderate Exudate					●	●			
	No/Minimal Exudate (Dry)						○	●		●
EDGE										
	Granulating / Fragile Bed			○	●	●				
#E5A44B	Epithelializing / Protecting				●	●				●

● Primary Choice ○ Consider

The Dressing Formulary: Matching Product to Purpose

Think of advanced dressings not by brand name, but by function. The goal is to create an optimal healing environment by matching the dressing's properties to the wound bed's needs. Every choice should answer one of these questions:

Does this wound need **moisture?** (Hydrate)

Does this wound need **exudate managed?** (Absorb)

Does this wound need **protection and cover?** (Protect)

Best Practices in Home-Based Wound Care

1. Cleansing the Wound

✅ **Do:** Use potable (tap) water or normal saline (0.9%). Cleanse at each dressing change.

❌ **Don't:** Use harsh solutions like hydrogen peroxide or full-strength Dakin's solution, which can be toxic to healing tissue.

Technique

Technique: Start at the center of the wound and clean in a circular pattern toward the edges to avoid recontamination. Gently pat the surrounding skin dry.

2. Selecting a Dressing

The goal is to maintain a moist wound environment and manage exudate.

For Heavy Exudate (VLUs)

Use calcium alginates, foams, or super-absorbent dressings.

For Dry Wounds (ALUs)

Use hydrogels to donate moisture. Keep stable eschar dry.

For Infected Wounds

Consider dressings with antimicrobials (silver, iodine, Manuka honey) as directed.

Dressing Category: Hydrogels

What It Is

A gel-based dressing composed primarily of water, designed to donate moisture to the wound bed.

Mechanism of Action

Maintains a moist wound environment, which promotes autolytic debridement and facilitates epithelial cell migration.

Primary Indications

- Dry or minimally exuding wounds.
- Wounds with dry eschar or slough that require softening.
- NPIAP Guideline: Recommended for non-infected Category/Stage II, III, and IV pressure injuries with minimal exudate. (Source: NPIAP, 2019)

Nursing Considerations for Home Health

- Requires a secondary dressing to secure it.
- Monitor peri-wound skin for maceration.
- Change frequency depends on the secondary dressing and wound condition.

Dressing Categories: Foams & Hydrocolloids

Foam Dressings

What It Is: Highly absorbent, non-adherent polyurethane pads.

Mechanism: Wicks and holds exudate away from the wound bed, maintaining a moist environment while protecting peri-wound skin.

Indications: Moderate to heavily exuding wounds. NPIAP Guideline: Recommended for Category/Stage II and greater pressure injuries. *(Source: NPIAP, 2019)*

Considerations: Available with or without adhesive borders; can be used under compression.

Hydrocolloid Dressings

What It Is: Occlusive wafer containing gel-forming agents.

Mechanism: Forms a moist gel as it absorbs light exudate, promoting autolytic debridement.

Indications: Lightly exuding wounds. NPIAP Guideline: Recommended for non-infected Category/Stage II pressure injuries. *(Source: NPIAP, 2019)*

Considerations: Not for infected wounds or heavy exudate. Change when the gel bubble migrates to the edge.

Dressing Categories: Alginates & Super-Absorbents

Calcium Alginate Dressings

What It Is: Absorbent, biodegradable dressing derived from seaweed.

Mechanism: Forms a hydrophilic gel upon contact with wound exudate, **absorbing large amounts of fluid.**

Indications: Moderately to heavily exuding wounds, including those with tunneling or undermining. **NPIAP Guideline: Recommended for Category/Stage III and IV pressure injuries.** (Source: NPIAP, 2019)

Considerations: Requires a secondary dressing. Do not use on dry wounds as it will dehydrate the wound bed.

Super-Absorbent Dressings

What It Is: Multi-layer dressing with a core that locks fluid away.

Mechanism: Rapidly absorbs and retains large volumes of exudate, preventing maceration and reducing dressing change frequency.

Indications: Very heavily exuding pressure injuries. (Source: NPIAP, 2019)

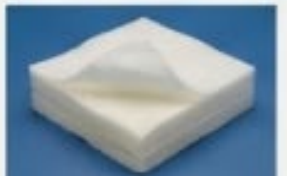
Considerations: Ideal for reducing caregiver burden and cost associated with frequent changes.

The Dressing Selection Matrix: Your At-A-Glance Guide

Wound Bed Need	Dressing Category	Key Characteristics
Needs Moisture (Dry)	Hydrogel	Donates moisture, facilitates autolytic debridement.
Light Exudate	Hydrocolloid	Maintains moist environment, promotes autolysis.
Light to Moderate Exudate	Foam	Versatile absorption, protective cushioning.
Moderate to Heavy Exudate	• Calcium Alginate	High absorption, forms a gel, good for packing tunnels.
Very Heavy Exudate	• Super-Absorbent	Locks away large fluid volumes, reduces change frequency.

The Clinician's Toolbox: Wound Dressings At-a-Glance

"Select the most appropriate dressing based on a clinical assessment of the wound: its size and depth, exudate level, wound bed condition, and periwound skin." (Adapted from NPIAP, 14.1)

Dressing Type	Primary Indication	Function	Visual Example
Hydrocolloids	Non-infected Stage II PI	Maintain moist environment, support autolytic debridement.	
Hydrogels	Dry to minimally exuding Stage II-IV PI	Donate moisture to a dry wound bed.	
Foams (Hydropolymers)	Stage II and greater PI with moderate to heavy exudate	Absorb exudate while maintaining a moist environment.	
Calcium Alginates	Stage III and IV PI with moderate to heavy exudate	Absorb heavy exudate, forming a gel. Good for packing.	
Super-absorbents	Heavily exuding pressure injuries	High capacity for absorption to manage large volumes of fluid.	

The Bioactives: Fighting Infection (Antimicrobials)

Goal: Kill or slow microbe growth to manage bioburden.

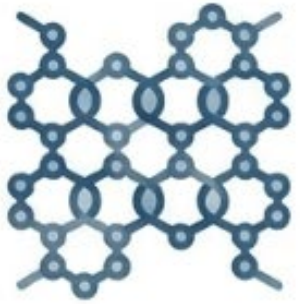
Silver (Ag)

Broad-spectrum. Found in foams/alginates. Use for infected or critically colonized wounds.

Iodine (Cadexomer/Povidone)

Slow release killing. Caution: Thyroid disorders or iodine sensitivity.

PHMB



Bacteria-killing polymer in gauze/foam.

Clinical Rule: Treat local infection with antimicrobial dressings. Systemic infection (fever, spreading redness) requires systemic antibiotics.

The Bioactives: Medical-Grade Honey (Manuka)

Antimicrobial

High sugar/osmotic pressure & low pH inhibits bacteria

Debridement

Osmotic action draws fluid, lifting slough/necrotic tissue

Odor Control

Reduces malodor in necrotic wounds

Indications

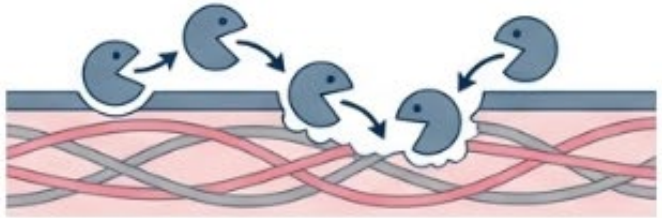
Sloughy, malodorous, or stalled wounds

Application Tip

Warn patient of transient stinging sensation

Jumpstarting Repair: Collagens

The Problem: MMPs (Matrix Metalloproteinases)

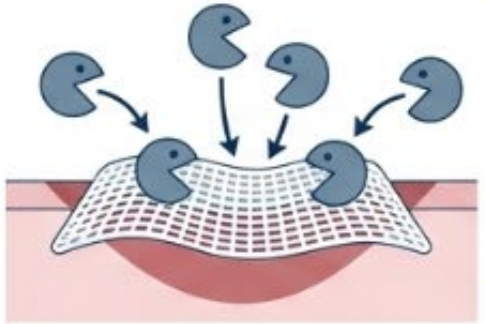


MMPs eat healthy tissue.

The Solution: Collagen Dressings

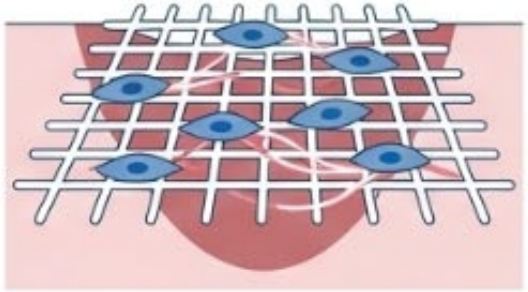
1. Sacrificial Substrate

Distracts MMPs to digest the dressing.



2. Scaffold

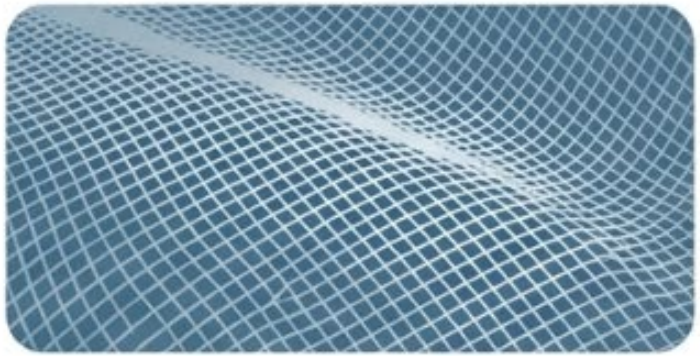
Attracts fibroblasts to build new tissue.



Indication: Clean, stalled chronic wounds free of necrotic tissue. *Distinct from oral supplements.*

Protection & Interface: Treating the Fragile

Contact Layers



Non-adherent mesh (silicone/petrolatum).
Placed directly on wound.

Benefit

- Allows exudate pass-through.
- Prevents sticking.
- Reduces pain during changes.

Transparent Films

Thin, semi-permeable membrane.
Waterproof but breathable.

Benefit

- Visibility of wound.
- Traps moisture for autolytic debridement.

Caution

- Avoid on draining wounds (maceration) or fragile skin (stripping risk).

Specialty Solutions: Packing & Scar Management

Impregnated Dressings

- Gauze/ribbon with petrolatum, bismuth, or saline.
- **Function:** Pack deep wounds (dead space) or keep wound bed moist.
- **Tip:** Pack loosely to avoid pressure damage.

Silicone Gel Sheets

- Scar therapy for Hypertrophic scars or Keloids.
- **Mechanism:** Hydrates stratum corneum, regulates fibroblasts to flatten scars.

Leveling Up: Mastering Negative Pressure Wound Therapy (NPWT)

When wounds are deep, complex, or fail to progress despite optimal dressing management, NPWT offers an active therapeutic solution. This section provides a practical guide to its use in the home health setting.

What is NPWT?

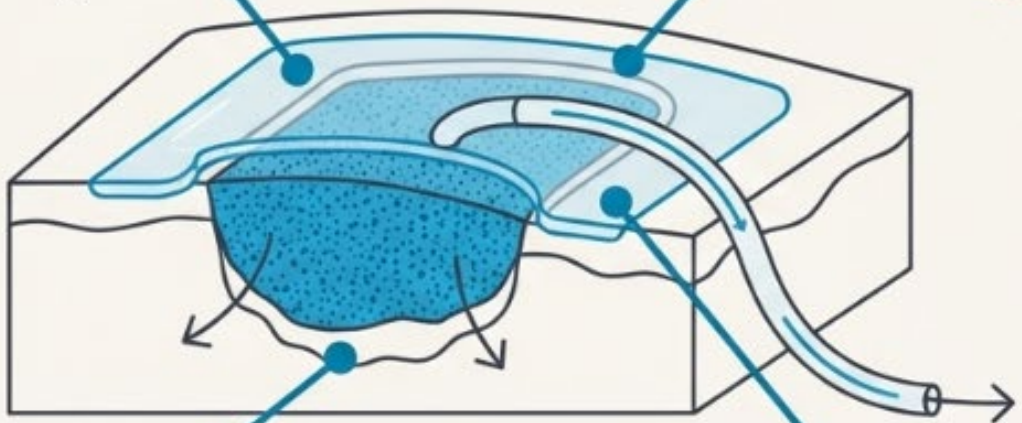
An active wound healing system that uses a vacuum to apply controlled, sub-atmospheric pressure to the wound bed.

The Science of Suction: How NPWT Promotes Healing

NPWT creates a dynamic healing environment through several key mechanisms of action:

Removes Excess Fluid: Continuously draws exudate and infectious material away from the wound bed, reducing edema and bacterial load.

Promotes Perfusion: Increases blood flow to the wound area, delivering more oxygen and nutrients essential for healing.



Draws Wound Edges Together: Provides mechanical forces that encourage contraction and reduce wound volume.

Promotes Granulation: Creates macro- and micro-strain at the cellular level, stimulating the formation of healthy new tissue.

NPWT in Practice: Indications and Critical Contraindications

✓ Common Indications

- Chronic wounds (Diabetic Foot Ulcers, Pressure Injuries)
- Acute and traumatic wounds
- Dehisced surgical wounds
- Skin grafts and flaps
- **NPIAP Guideline:** Consider as an *early adjunct therapy* for reducing the size and depth of Category/Stage III and IV pressure injuries. (Strength B1, ↑ Recommendation) (Source: NPIAP, 2019)

✗ Critical Contraindications

- **Malignancy** in the wound
- **Untreated Osteomyelitis** (Therapy can be considered *after* appropriate surgical debridement)
- Non-enteric and unexplored fistulas
- Necrotic tissue with eschar present (must be debrided first)
- Exposed blood vessels, nerves, or organs

The NPWT Troubleshooting Guide

Problem	Common Causes in the Home Setting	Nursing Actions
Loss of Seal / Air Leak Alarm	• Dressing wrinkles • Uneven skin surface (e.g., near a joint) • Patient movement or diaphoresis	• Smooth out wrinkles; 'picture frame' with extra drape • Use skin barrier strips or stoma paste to build up uneven areas • Ensure all tube connections are tight
Canister Full Alarm	• High volume of wound exudate • Canister has been tipped over	• Change the canister • Keep the NPWT unit on a flat, stable surface
Patient Reports Increased Pain	• Pressure setting may be too high • Foam is in direct contact with sensitive structures (tendon, bone)	• Consult with provider about lowering pressure settings • Apply a non-adherent contact layer before placing foam • Pre-medicate for dressing changes
Bleeding in Canister or Tubing	• Normal granulation tissue bleeding (serosanguinous) • Erosion into a blood vessel (bright red, pulsatile - **EMERGENCY)**	• **For minor bleeding, assess and document.** • **If bleeding is significant or pulsatile: STOP THERAPY IMMEDIATELY. Apply direct pressure. Call for emergency medical assistance.**

Case Study: Putting the Toolkit to Work

Patient: Mr. G, a 72-year-old with poorly controlled Type 2 Diabetes and a Wagner Grade 3 DFU on his plantar surface.

Stage 1: Week 1-4

Initial Management

Assessment: Moderate serosanguinous exudate, 100% slough after initial sharp debridement. High risk of infection.

Action: A calcium alginate dressing was used to manage exudate and fill dead space, changed every 2-3 days. Offloading boot provided.

Stage 2: Week 5

Reassessment & Change in Plan

Assessment: Wound size has not decreased. Edges are stalled. Exudate remains moderate. Underlying osteomyelitis was ruled out.

Action: Plan escalated to include NPWT to stimulate granulation tissue and manage exudate more effectively.

Stage 3: Week 8

Outcome

Assessment: Wound shows significant granulation tissue, a 40% reduction in volume, and minimal exudate. Ready to transition back to advanced moisture-retentive dressings.

Action: NPWT discontinued. Patient transitioned to a foam dressing.

Your Expanded Toolkit for Complex Wounds

Mastering these advanced tools empowers you to manage complex wounds effectively in the home, preventing complications and promoting healing.

ASSESS FIRST: The wound always dictates the treatment. Match the dressing's function (hydrate, absorb, protect) to the wound bed's specific needs.

KNOW YOUR INDICATIONS: Use NPWT as a powerful tool for stalled, complex wounds, but always be vigilant about the critical contraindications.

TROUBLESHOOT WITH CONFIDENCE: Common NPWT issues can be managed at the bedside with systematic problem-solving.

YOU ARE THE KEY: Your advanced skills in wound management are the most critical factor in improving your patients' quality of life and preventing amputations.

Advanced Therapies: Hyperbaric Oxygen (HBO) for Refractory Wounds

Primary Indication

"For chronic refractory osteomyelitis: a bone infection that has not improved after 4-6 weeks of appropriate culture-directed antibiotics and surgical debridement." (Cooper et al.)

What is HBO Therapy?

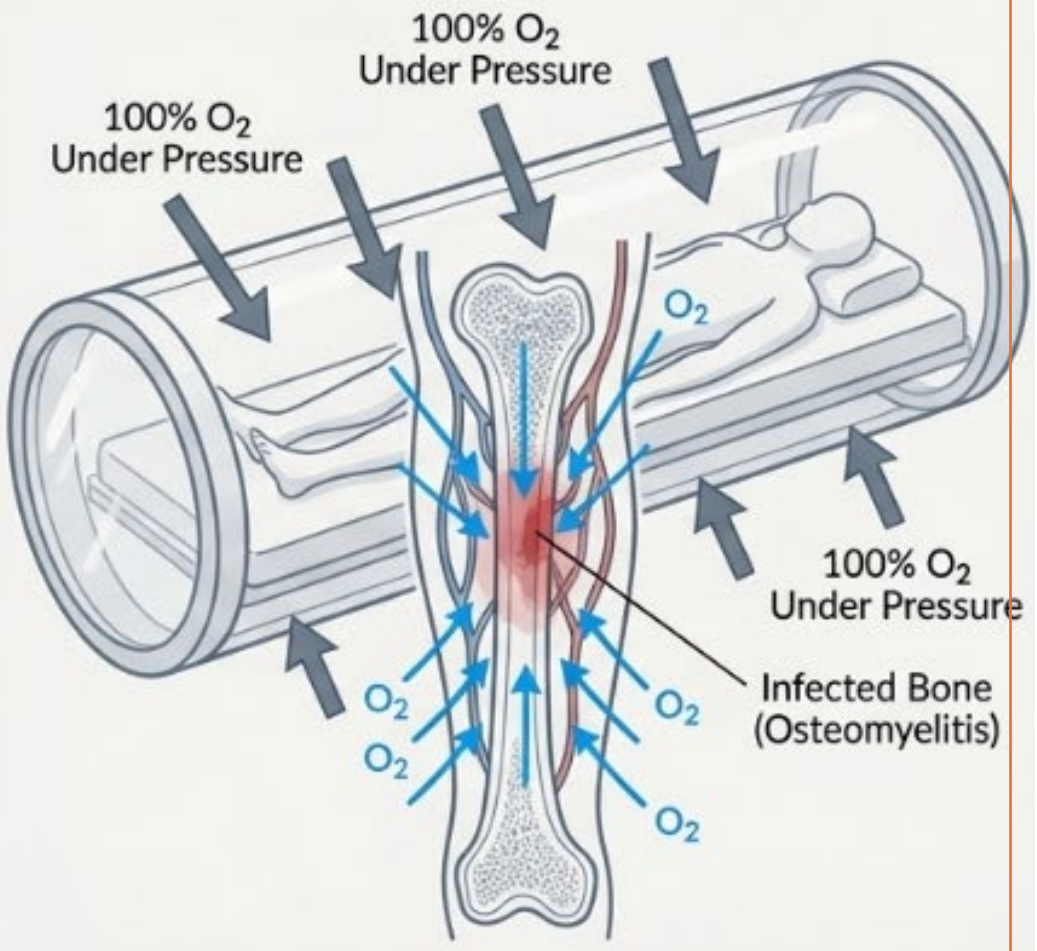
A patient breathes 100% oxygen in a pressurized chamber (2.0-3.0 atmospheres absolute). Treatments are typically 90-120 minutes daily for 20-40 sessions.

How It Works for Osteomyelitis

1. **Enhances Bacterial Killing**: Hyper-oxygenates infected bone, which improves leukocyte-mediated killing of organisms like *S. aureus*.
2. **Boosts Antibiotics**: Improves the penetration of aminoglycosides and cephalosporins into bone tissue.
3. **Stimulates Osteogenesis**: Promotes new bone growth and healing.

Guideline Note

Considered a Class II recommendation by the AHA for treating chronic, refractory osteomyelitis.



Assess Systematitically. Stage Accurately. Treat Purposefully. Collaborate Constantly.

Effective management of complex wounds requires an interprofessional team. The best outcomes are achieved through collaboration between wound specialists, podiatry, surgery, infectious disease, and nutrition. (Adapted from Cooper et al.)

Your Field Guide Checklist

- ❑ Conduct regular, comprehensive skin and risk assessments.
- ❑ Use a consistent classification system (Wagner for DFU, NPIAP for PI).
- ❑ Match treatment (debridement, dressing, offloading) to the wound type and stage.
- ❑ Maintain a high index of suspicion for complications like biofilm and osteomyelitis.
- ❑ Know when to escalate care and consult the wider healthcare team.

References

Abu-Own A, Scurr JH, Coleridge Smith PD. (1994). Effect of leg elevation on the skin microcirculation in chronic venous insufficiency. Journal of Vascular Surgery, 20(5), 705–710. doi: 10.1016/s0741-5214(94)70157-1.

Akers AS. (2023, July 18). Arterial and venous ulcers: characteristics and telling the difference. Medical News Today.

Alandejani T, Marsan J, Ferris W, Slinger R, Chan F. (2009). Effectiveness of honey on Staphylococcus aureus and Pseudomonas aeruginosa biofilms. Otolaryngology–Head and Neck Surgery, 141(1), 114–118. doi: 10.1016/j.otohns.2009.01.005.

Amsler F, Blättler W. (2008). Compression therapy for occupational leg symptoms and chronic venous disorders-a meta-analysis of randomised controlled trials. European Journal of Vascular and Endovascular Surgery, 35(3), 366–372. doi: 10.1016/j.ejvs.2007.09.021.

Bajwa A, Wesolowski R, Patel A, Saha P, Ludwinski F, Smith A, Nagel E, Modarai B. (2014). Assessment of tissue perfusion in the lower limb. *Circulation: Cardiovascular Imaging*, 7(5), 836–843. doi: 10.1161/CIRCIMAGING.113.001476.

Barfett J, Velauthapillai N, Kloeters C, Mikulis DJ, Jaskolka JD. (2012). An en bloc approach to CT perfusion for the evaluation of limb ischemia. *The International Journal of Cardiovascular Imaging*, 28(8), 2073–2083. doi: 10.1007/s10554-012-0059-3.

References

Blair SE, Cokcetin NN, Harry EJ, Carter DA. (2009). The unusual antibacterial activity of medical-grade Leptospermum honey: antibacterial spectrum, resistance and transcriptome analysis. *European Journal of Clinical Microbiology & Infectious Diseases*, 28(10), 1199–1208. doi: 10.1007/s10096-009-0763-z.

Bogdanov S. (2009). Bee product science. In *Book of honey as nutrient and functional food*.

Boulton AJ, Vileikyte L, Ragnarson-Tennvall G, Apelqvist J. (2005). The global burden of diabetic foot disease. *The Lancet*, 366(9498), 1719–1724. doi: 10.1016/S0140-6736(05)67698-2.

Brem H, Kirsner RS, Falanga V. (2004). Protocol for the successful treatment of venous ulcers. *The American Journal of Surgery*, 188(1 Suppl), 1–8. doi: 10.1016/S0002-9610(03)00284-8.

Britto EJ, Nezwek TA, Popowicz P, et al. Wound Dressings. [Updated 2024 Jan 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. https://www.ncbi.nlm.nih.gov/books/NBK470199/

Choucair M, Phillips TJ. (1998). Compression therapy. *Dermatologic Surgery*, 24(1), 141–148. doi: 10.1111/j.1524-4725.1998.tb04067.x.

Comerota AJ. (2011). Intermittent pneumatic compression: physiologic and clinical basis to improve management of venous leg ulcers. *Journal of Vascular Surgery*, 53(4), 1121–1129. doi: 10.1016/j.jvs.2010.08.059.

Cooper, J S, Hanley, M E, & Hendriksen, S (2025). Hyperbaric treatment of chronic refractory osteomyelitis. In *StatPearls [Internet]*. StatPearls Publishing.

References

Cooper RA, Halas E, Molan PC. (2002). The efficacy of honey in inhibiting strains of Pseudomonas aeruginosa from infected burns. *Journal of Burn Care & Rehabilitation*, 23(6), 366–370. doi: 10.1097/00004630-200211000-00002.

Cooper RA, Lindsay E, Molan PC. (2011). Testing the susceptibility to manuka honey of streptococci isolated from wound swab. *Journal of ApiProduct and ApiMedical Science*, 3(3), 117–122.

Cullum N, Nelson EA, Fletcher AW, Sheldon TA. (2000). Compression bandages and stockings for venous leg ulcers. *Cochrane Database of Systematic Reviews*, (2), CD000265. doi: 10.1002/14651858.CD000265.

Dahl PR, Winkelmann RK, Connolly SM. (1995). The vascular calcification-cutaneous necrosis syndrome. *Journal of the American Academy of Dermatology*, 33(1), 53–58. doi: 10.1016/0190-9622(95)90011-8.

Delis KT, Nicolaides AN, Wolfe JH, Stansby G. (2000). Improving walking ability and ankle brachial pressure indices in symptomatic peripheral vascular disease with intermittent pneumatic foot compression: a prospective controlled study with 1-year follow-up. *Journal of Vascular Surgery*, 31(4), 650–661. doi: 10.1067/mva.2000.103969.

Duan Y, Wang X, Yang X, Wu D, Cheng Z, Wu L. (2013). Diagnostic efficiency of low-dose CT angiography compared with conventional angiography in peripheral arterial occlusions. *AJR. American Journal of Roentgenology*, 201(6), W906–W914. doi: 10.2214/AJR.12.10368.

References

Eze AR, Comerota AJ, Cisek PL, Holland BS, Kerr RP, Veeramasuneni R, Comerota AJ Jr. (1996). Intermittent calf and foot compression increases lower extremity blood flow. *The American Journal of Surgery*, 172(2), 130–134. doi: 10.1016/S0002-9610(96)00134-1.

Fletcher A, Cullum N, Sheldon TA. (1997). A systematic review of compression treatment for venous leg ulcers. *BMJ*, 315(7108), 576–580. doi: 10.1136/bmj.315.7108.576.

Fox IJ, Wood EH. (1960). Indocyanine green: physical and physiologic properties. *Proceedings of the Staff Meetings of the Mayo Clinic*, 35, 732–744.

Galanakis N, Maris TG, Kontopodis N, Tsetis K, Kehagias E, Tsetis D. (2022). Perfusion imaging techniques in lower extremity peripheral arterial disease. *The British Journal of Radiology*, 95(1131), 20211203. doi: 10.1259/bjr.20211203.

Galvan L. (2005). Assessing venous ulcers and venous insufficiency. *Nursing*, 35(Suppl), 70. doi: 10.1097/00152193-200507001-00020.

Gethin G, Cowman S. (2009). Manuka honey vs. hydrogel--a prospective, open label, multicentre, randomised controlled trial to compare desloughing efficacy and healing outcomes in venous ulcers. *Journal of Clinical Nursing*, 18(3), 466–474. doi: 10.1111/j.1365-2702.2008.02558.x.

Gethin GT, Cowman S, Conroy RM. (2008). The impact of manuka honey dressings on the surface pH of chronic wounds. *International Wound Journal*, 5(2), 185–194. doi: 10.1111/j.1742-481X.2007.00424.x.

References

Gillespie DL, Writing Group III of the Pacific Vascular Symposium 6. (2010). Venous ulcer diagnosis, treatment, and prevention of recurrences. *Journal of Vascular Surgery*, 52(5 Suppl), 8s–14s. doi: 10.1016/j.jvs.2010.05.088.

Gloviczki P, Comerota AJ, Dalsing MC, Eklof BG, Gillespie DL, Gloviczki ML, Lohr JM, McLafferty RB, Meissner MH, Murad MH, et al. (2011). The care of patients with varicose veins and associated chronic venous diseases: clinical practice guidelines of the Society for Vascular Surgery and the American Venous Forum. *Journal of Vascular Surgery*, 53(5 Suppl), 2s–48s. doi: 10.1016/j.jvs.2011.01.079.

Gordon Y, Partovi S, Müller-Eschner M, Amarteifio E, Baeuerle T, Weber M-A, Kauczor H-U, Rengier F. (2014). Dynamic contrast-enhanced magnetic resonance imaging: fundamentals and application to the evaluation of the peripheral perfusion. *Cardiovascular Diagnosis and Therapy*, 4(2), 147–164. doi: 10.3978/j.issn.2223-3652.2014.03.04.

Guideline for Disinfection and Sterilization in Healthcare Facilities. (2014). *CDC*.

Gupta S, Anderson C, Black J, et al. (2017). Management of chronic wounds: diagnosis, preparation, treatment, and follow-up. *Wounds*, 29(9), S19–S36.

Hackl G, Gary T, Belaj K, Hafner F, Eller P, Brodmann M. (2015). Risk factors for puncture site complications after endovascular procedures in patients with peripheral arterial disease. *Vascular and Endovascular Surgery*, 49(5-6), 160–165. doi: 10.1177/1538574415598144.

Haynes JS, Callaghan R. (2011). Properties of honey: its mode of action and clinical outcomes. *Wounds UK*, 7(2), 50–57.

References

Henriques AF, Jenkins RE, Burton NF, Cooper RA. (2010). The intracellular effects of manuka honey on Staphylococcus aureus. *European Journal of Clinical Microbiology & Infectious Diseases*, 29(1), 45–50. doi: 10.1007/s10096-009-0817-2.

Henriques AF, Jenkins RE, Burton NF, Cooper RA. (2011). The effect of manuka honey on the structure of Pseudomonas aeruginosa. *European Journal of Clinical Microbiology & Infectious Diseases*, 30(2), 167–171. doi: 10.1007/s10096-010-1065-1.

Institute for Clinical Systems Improvement (ICSI). (2012). *Pressure ulcer prevention and treatment protocol. Health care protocol*. Bloomington (MN): Institute for Clinical Systems Improvement (ICSI).

Jervis-Bardy J, Foreman A, Bray S, Tan L, Wormald PJ. (2011). Methylglyoxal-infused honey mimics the anti-Staphylococcus aureus biofilm activity of manuka honey: potential implication in chronic rhinosinusitis. *The Laryngoscope*, 121(5), 1104–1107. doi: 10.1002/lary.21717.

Keneric Healthcare. (n.d.). *RTD wound dressing indications for use: methylene blue and Gentian Violet*. Retrieved from https://www.kenerichealthcare.com/rtd-product-brochure/

Kestrel Health Information, Inc. (2018). Wound cleanser products. *Wound Source Website*. www.woundsource.com/product-category/wound-cleansers/wound-cleanser-products.

Lehrman, J. D. (n.d.). *Pressure ulcer versus pressure injury ICD-10-CM coding*. Intellicure Blog.

References

Moffatt CJ, Franks PJ, Oldroyd M, Bosanquet N, Brown P, Greenhalgh RM, McCollum CN. (1992). Community clinics for leg ulcers and impact on healing. *BMJ*, 305(6866), 1389–1392. doi: 10.1136/bmj.305.6866.1389.

Molan P, Betts J. (2000). Using honey dressings: the practical considerations. *Nursing Times*, 96(49), 36–37.

Molan PC. (2009). Debridement of wounds with honey. *Journal of Wound Technology*, 5, 12–16.

Mölnlycke Health Care. (2025). *Supporting your healing journey: A guide to venous leg ulcer care* (Document ID HQIM006456) [Brochure].

Moody M. (1999). Comparison of Rosidal K and SurePress in the treatment of venous leg ulcers. *British Journal of Nursing*, 8(6), 345–355. doi: 10.12968/bjon.1999.8.6.6660.

Moore OA, Smith LA, Campbell F, Seers K, McQuay HJ, Moore RA. (2001). Systematic review of the use of honey as a wound dressing. *BMC Complementary and Alternative Medicine*, 1, 2. doi: 10.1186/1472-6882-1-2.

Morison MJ. (1992). *A colour guide to the nursing management of wounds*. London: Wolfe.

National Wound Care Strategy Programme. (2023). *Foot ulcer recommendations*.

Necas M. (2010). Duplex ultrasound in the assessment of lower extremity venous insufficiency. *Australasian Journal of Ultrasound in Medicine*, 13(4), 37–45. doi: 10.1002/j.2205-0140.2010.tb00171.x.

References

Nelson EA, Harper DR, Prescott RJ, Gibson B, Brown D, Ruckley CV. (2006). Prevention of recurrence of venous ulceration: randomized controlled trial of class 2 and class 3 elastic compression. *Journal of Vascular Surgery*, 44(4), 803–808. doi: 10.1016/j.jvs.2006.05.051.

NPIAP/EPUAP/Pan-Pacific Pressure Injury Alliance. (2019). *Prevention and treatment of pressure ulcers/injuries: Clinical practice guideline. Quick reference guide 2019*.

O'Meara S, Cullum NA, Nelson EA. (2009). Compression for venous leg ulcers. *Cochrane Database of Systematic Reviews*, (1), CD000265. doi: 10.1002/14651858.CD000265.pub2.

Partsch H, Menzinger G, Mostbeck A. (1999). Inelastic leg compression is more effective to reduce deep venous refluxes than elastic bandages. *Dermatologic Surgery*, 25(9), 695–700. doi: 10.1046/j.1524-4725.1999.98040.x.

Phillips TJ. (2001). Current approaches to venous ulcers and compression. *Dermatologic Surgery*, 27(7), 611–621. doi: 10.1046/j.1524-4725.2001.00195.x.

Pomposelli F. (2010). Arterial imaging in patients with lower extremity ischemia and diabetes mellitus. *Journal of Vascular Surgery*, 52(5 Suppl), 81S–91S. doi: 10.1016/j.jvs.2010.06.009.

Powell CC, Rohrer MJ, Barnard MR, Peyton BD, Furman MI, Michelson AD. (1999). Chronic venous insufficiency is associated with increased platelet and monocyte activation and aggregation. *Journal of Vascular Surgery*, 30(5), 844–853.

References

Shi C, Wang, C. Wang H. et al. Selection of Appropriate Wound Dressing for Various Wounds. Front. Bioeng. Biotechnol.,Vol 8 – 2020: 1-17. -https://doi.org/10.3389/fbioe.2020.00182

Smith & Nephew. (2019). *RENASYS GO patient information* (Document ID RNME17-14397-0119GO) [Brochure].

Smith PC, Sarin S, Hasty J, Scurr JH. (1990). Sequential gradient pneumatic compression enhances venous ulcer healing: a randomized trial. *Surgery*, 108(5), 871–875.

Solventum. (n.d.). V.A.C.® therapy clinical guidelines.

Srisuwan T, Inmutto N, Kattipathanapong T, Rerkasem A, Rerkasem K. (2020). Ultrasound use in diagnosis and management of venous leg ulcer. *International Journal of Lower Extremity Wounds*, 19(4), 305–314. doi: 10.1177/1534734619888995.

Strolin A, Volkert B, Hafner HM, Junger M. (2005). Medical compression stockings in CVI-patients. *Phlebologie*, 34(1), 34–41.

Wound, Ostomy and Continence Nurses Society. (2011). *Ankle brachial index: quick reference guide for clinicians*. Mt. Laurel, NJ: Author.

References

Wound, Ostomy and Continence Nurses Society (WOCN) Wound Committee, et al. (2012). Clean vs. sterile dressing techniques for management of chronic wounds: a fact sheet. Journal of Wound, Ostomy, and Continence Nursing, 39(2 Suppl), S30–S34. doi: 10.1097/WON.0b013e3182478e06.

WoundSource Practice Accelerator. (2018). *Wound infection diagnosis and management: an overview of topical therapies*. Kestrel Health Information, Inc.

Wounds UK. (n.d.). *Best practice statement addressing skin tone bias in wound care: assessing signs and symptoms in people with dark skin tones*.

Yaniv T, Beeckman D, Gefen A (2024) A Markov cost-effectiveness modeling framework for evaluating wound dressings: A concept for practical implementation of economic evaluations in an informed dressing selection process. *J Tissue Viability* 33(4): 938–48