

Chronic Wound Identification and Assessment



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Advanced Wound Care

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by Cheryl Carver LPN, WCC, CWCA, CWCP, FACCWS, DAPWCA, CLTC – Wound Educator

Chronic wounds create a strain and major challenge in every health care setting, and these challenges continue to multiply as a result of health care costs and the aging population. The goal of this white paper is to provide a more simplified structured plan to identify and assess chronic wounds correctly in the health care arena. A discussion will be provided on the key essentials of the chronic wound plan of care, with an emphasis on proactive strategies for preventing wound recurrence.

Chronic wounds affect more than 6.5 million people and cost billions of dollars every year in the United States.¹ Wound identification and a comprehensive assessment make up the foundation for gauging an effective treatment plan. Chronic wounds become stagnant through the normal phases of wound healing, and they usually adopt a vicious cycle. This cycle may include numerous comorbidities, mixed etiologies, and intrinsic and extrinsic factors.²

10 Key Aspects Of Chronic Wound Management

In effectively managing chronic wounds and supporting patients on the path to healing, there are a number of aspects of the care plan that need to be addressed. Following are the essential areas of wound management that must be factored into delivering comprehensive and effective care³:

1. Wound etiology
2. Wound assessment
3. Wound bed preparation (cleansing, controlling bioburden)
4. Wound depth and exudate amount
5. Dressing wear time (frequency)
6. Nutritional status
7. Pain management
8. Patient compliance, self-abilities, lifestyle changes
9. Patient and caregiver education (ongoing)
10. Payer source (reimbursement, financial)

Strategies To Simplify Identifying Wound Types

Identifying wound types can be difficult at times. There may be mixed etiologies and underlying factors that do not quite fit the normal characteristics of chronic wound types. You should be consistent with a detailed assessment of wound characteristics, including wound location, wound tissue type, periwound and wound edge shape, temperature, pulses, sensation, exudate color and amount, and wound depth.²

The following reference tool will provide you with an overview of the characteristics and treatment options for the most common types of chronic wound conditions.

Note: This guide is for informational purposes only; please always refer to your facility's protocol in the assessment and treatment of your patient's wounds.



Arterial Ulcer

LOCATION	Between or on the tips of the toes, outer ankle
SHAPE	Punched-out round appearance, unless a mixed etiology of venous and arterial
COLOR	Pale pink, gray, or yellow and periwound edema
DEPTH	Usually deep, full-thickness
PAIN	Typically yes; patient may report pain while in bed or when limb elevated ⁴

Most common causes and risk factors of arterial ulcers are⁴:

- Peripheral vascular disease
- Pyoderma gangrenosum
- Chronic vascular insufficiency
- Sickle cell disease
- Vasculitis
- Obesity
- Diabetes mellitus
- Renal failure
- Hyperlipidemia
- Family history
- Arteriosclerosis
- Atherosclerosis
- Smoking
- Trauma
- Limited joint mobility
- Increased age
- Thromboangiitis

Diagnostic Toolbox

Diagnostic Tests – Arterial Doppler studies with toe pressures if diabetic (include waveforms), Semmes Weinstein monofilament test to rule out neuropathy, and transcutaneous oximetry monitoring or fluorescent angiography to provide tissue perfusion results at the wound site. Ankle-brachial index (ABI) results can be elevated by calcification of vessels. Blood flow impairment; ABI <0.9

Lab Tests – CBC, CRP, and ESR

Imaging – MRI, CTA, and angiogram

Surgical Procedures – Revascularization using endovascular or traditional bypass procedures to promote wound healing⁴

Treatment Plan

- 1 Manage existing comorbidities (cholesterol, blood pressure, controlled blood sugar levels).
- 2 Perform weekly thorough lower extremity skin checks.
- 3 Protect skin to lower extremities to avoid trauma wounds.
- 4 Smoking cessation. Smoking impedes healing and increases risk of ulcers.
- 5 Do not cross legs.
- 6 Wear loose socks and garments to prevent constriction.⁴



Venous Ulcer

LOCATION	Gaiter area of the leg. Above the malleolus to 1 inch below the knee, along distal saphenous vein. Edema in lower extremities
SHAPE	Irregular characteristic
COLOR	Moist, viable red granulation tissue, may or may not have devitalized tissue (slough, eschar)
DEPTH	Shallow and diffuse ^{4,5}

Most common causes and risk factors of venous ulcers are^{4,5}:

- Venous hypertension, incompetent valves
- Varicose veins
- Previous deep vein thrombosis
- Smoking
- Family history
- Lymphedema
- Diabetes mellitus
- Congestive heart failure
- Poor nutrition
- Decreased mobility

Diagnostic Toolbox

Diagnostic Tests – Arterial Doppler studies with toe pressures if diabetic (include waveforms), Semmes Weinstein monofilament test to rule out neuropathy, and transcutaneous oximetry monitoring or fluorescent angiography to provide tissue perfusion results at the wound site. Ankle brachial index (ABI) results can be elevated by calcification of vessels. Adequate blood flow; ABI >0.8 unless mixed etiology with arterial

Lab Tests – CBC, CRP, and ESR

Imaging – MRI, CTA, and angiogram

Surgical Procedures – Revascularization using endovascular or traditional bypass procedures to promote wound healing. Surgical ablation of superficial and/or perforating veins^{4,5}

Treatment Plan

- 1 Manage existing comorbidities (cholesterol, blood pressure, controlled blood sugars, obesity).
- 2 Consistent wear of compression therapy as prescribed.
- 3 Smoking cessation. Smoking impedes healing and increases risk of ulcers.
- 4 Elevate legs above the heart.
- 5 Walk frequently.

Compression Therapy Options⁵:

- 1 **Unna boot** – Zinc oxide plaster. Calamine additive also available. (35–40mmHg) Provides sustained therapeutic compression for up to 7 days. ABI (ankle brachial index) >0.7
- 2 **Two-layer system** – (25–30mmHg, 35–40mmHg) Provides sustained therapeutic compression for up to 7 days. ABI (ankle brachial index) >0.7
- 3 **Four-layer system** – (35–40mmHg) Provides sustained therapeutic compression for up to 7 days. ABI (ankle brachial index) >0.8
- 4 **Compression garments** – Velcro, zipper, and pull-on application available
- 5 **Compression lymphedema pumps** – Dynamic compression devices provide a therapeutic, controlled external compression or pressure cycle to a limb



Diabetic Neuropathic Ulcer

LOCATION	Below ankle, most common plantar aspect of foot
SHAPE	Round and calloused edges (may be referred to as hyperkeratosis, or fibrotic rimmed)
COLOR	Pink, pale, dry
DEPTH	Partial- or full-thickness ^{4,5}

Most common causes and risk factors of diabetic neuropathic ulcers are^{4,5}:

- Uncontrolled blood sugars
- Ischemia, peripheral vascular disease
- Charcot deformity, increased plantar aspect pressure
- Advanced age
- Obesity
- Family history
- Hypertension
- Previous amputation
- Smoking
- Presence of retinopathy
- Absence of vibratory sensation
- Trauma
- Poor fitting shoes – mechanical force causing skin breakdown, callus, and/or ulcer
- Coronary artery disease
- Neuropathy, Insensate

Diagnostic Toolbox

Diagnostic Tests – Arterial Doppler studies with toe pressures (include waveforms), Semmes Weinstein monofilament test to rule out neuropathy, and transcutaneous oximetry monitoring or fluorescent angiography will provide tissue perfusion results at the wound site. Ankle brachial index (ABI) results can be elevated by calcification of vessels. Biothesiometry to determine vibration perception threshold.

Lab Tests – CMP, pre-albumin, ESR, CRP, HgA1c, CBC, screen for leukocytosis and anemia

Imaging – X-ray, MRI, CT angiography, or bone scan as indicated

Surgical Procedures – None^{4,5}

Treatment Plan

- 1 Manage calluses, fissures, corns, hammertoes, bunions, toenail complications, Charcot foot deformities, and chronic non-healing ulcers to prevent limb amputation.
- 2 Daily inspection of feet. Never walk barefoot. Wear dry, high-wicking clean socks. No lotion should be between the toes, thus causing increased risk of bacteria growth.
- 3 Control diabetes per physician recommendations that include a well-balanced diet, regular exercise, and healthy lifestyle.^{4,5}



Surgical Dehiscence

Wound dehiscence is one of the most common complications of surgical wounds, and it involves breaking open the surgical incision along the suture. When wound dehiscence occurs, the edges start to separate, and the wound reopens instead of healing closed as planned.²

Most common causes and risk factors of surgical chronic wounds are⁴:

- Subacute infection
- Poor surgical technique
- Poor perfusion
- Tension to wound edges
- Increased age
- Obesity
- Smoking
- Steroidal therapy
- Poor nutrition
- Diabetes mellitus
- Radiation
- Heart, kidney, and or liver disease

Wound dehiscence can be prevented by taking the following measures:

- 1 Avoid stress or strain to wound site – lifting, exercise, coughing, vomiting, and constipation. Holding onto a pillow at the wound site may help relieve stress.
- 2 Comply with physician post-operative instructions.
- 3 Maintain hydration and a well-balanced diet.⁴



Atypical Wounds

Atypical wounds, also known as wounds of unknown etiology, are wounds caused by conditions or diseases that do not typically form a wound, such as inflammations, infections, malignancies, chronic illnesses, or genetic disorders. Atypical wounds can also stem from rare uncommon causes or occur with an abnormal presentation or location.⁷

Most common causes and risk factors of arterial ulcers are:⁷

Increased age

Pre-existing chronic illness

Infections

Weakened immune system

Medications

Unhealthy lifestyle

Inflammatory Wounds

Pyoderma gangrenosum

Bullous pemphigoid

Hereditary or Genetic Wounds

Scleroderma

Sickle cell anemia

Epidermolysis bullosa

Dermatitis artefacta

Malignancy Wounds

Kaposi sarcoma

Squamous cell carcinoma

Basal cell carcinoma

Radiation necrosis

Vasculopathy Wounds

Vasculitis

Cryoglobulinemia

Infection Wounds

Blastomycosis

Atypical mycobacterial infection



Pressure Ulcer/Injury Staging

LOCATION	Usually bony prominence areas, but can be under medical devices or in mucosal membrane
SHAPE	Usually round, but may be irregular in shape depending on friction and shear
COLOR	Based on tissue level of destruction
DEPTH	Intact, partial-thickness, full-thickness

Most common causes and risk factors of pressure ulcers/injuries are:

- Impaired blood supply from prolonged pressure, friction, or shear
- Lack of sensation
- Moisture
- Immobility
- Insensate
- Poor nutrition
- Chronic medical conditions
- History of pressure ulcer/injury

Diagnostic Toolbox

Lab Tests – CBC, ESR, HgA1c (diabetics), CRP, pre-albumin

Imaging – X-ray, bone scan, MRI

Surgical Procedures – Sharp debridement, surgical debridement

In April 2016, the National Pressure Ulcer Advisory Panel (NPUAP) announced a change in terminology from “pressure ulcer” to “pressure injury” and also updated the stages of pressure injury. Note that there is no entry in the ICD-10-CM alphabetic index for pressure injury.

Pressure Injury (NPUAP Level of Injury) ²	Pressure Ulcer (CMS Terminology) ²
Stage 1	Stage I
Stage 2	Stage II
Stage 3	Stage III
Stage 4	Stage IV
Unstageable Pressure Injury	Unstageable Pressure Ulcer
Deep Tissue Pressure Injury	Suspected Deep Tissue Injury
Medical Device Pressure Injury	—
Mucosal Membrane Pressure Injury	—

Treatment Plan

- 1 Offload pressure: Turn and reposition every 2 hours and as needed while in bed. Individuals confined to a wheelchair or chair should shift weight at least every 15 minutes.
- 2 Follow a well-balanced nutritional diet.
- 3 Perform wound cleansing, and use select dressings that provide a protective barrier for infection and an optimal moist environment.
- 4 Control bioburden (debridement methods).
- 5 Implement patient and caregiver prevention and treatment plan education.

What can we do to reduce friction and shearing in managing our patients?

1. Pad and protect vulnerable areas (transparent, hydrocolloid, composite, foam dressings) as per facility protocol.
2. Use heel or elbow protectors for hospice or palliative patients.
3. Educate caregivers and nursing staff about how to identify key factors for pressure ulcers/injuries.
4. Ensure that support surfaces provide for individual’s particular needs: pressure redistribution, shear reduction, and or microclimate control.
5. Use positioning devices in wheelchairs or chairs to reduce shearing.
6. Establish a risk assessment per facility protocol.
7. Use draw sheets to pull up, transfer, and position your patient. DO NOT DRAG.



Moisture-Associated Skin Damage (MASD)/ Incontinence-Associated Dermatitis (IAD)

When Is it MASD, and When Is It Pressure?

Regular inspection of the skin is essential in identifying damage resulting from moisture and/or pressure. The following chart provides a basic guideline for distinguishing MASD/IAD from the formation of pressure ulcers/injuries⁸:

MASD/IAD	PRESSURE ULCER/INJURY
LOCATION: Diffusely distributed	LOCATION: Usually over a bony prominence
PAIN: Yes	PAIN: May or may not be present
COLOR: Pink or red	COLOR: Red or bluish- purple
DEPTH: Partial-thickness, blistering	DEPTH: Intact, partial- or full-thickness
TISSUE: No slough or eschar	TISSUE: With or without slough or eschar

Moist Wound Healing

Moist wound healing has been shown to improve or reduce healing time. Controlling exudate and bacteria is most effective. Keeping an even moisture balance of the wound bed is vital in healing progress. Moist wound healing increases the speed of the proliferative phase and decreases the intensity and length of the inflammatory phase. Autolytic debridement is also provided with moist wound healing, in return increasing synthesis of collagen and fibroblast proliferation. Less scarring and pain have also been proven with moist wound healing.⁹

Controlling Bioburden

Controlling bioburden and/or biofilm is necessary for a wound to heal. Devitalized tissue (slough and eschar) harbor bacteria and slow down the wound healing process. Most chronic wounds are considered contaminated with biofilm. Biofilm is another culprit that impedes wound healing progress. Most, if not all ulcers develop a biofilm over time. The protective polysaccharide matrix is produced by bacteria. Bioactive wound care dressings can include tissue-engineered products derived from artificial sources or natural tissues (e.g., hyaluronic acid, elastin, silicone, chitosan, alginates, collagen, antimicrobials, skin equivalents, growth factors, cell-free matrices, cell-containing matrices).¹⁰

Periwound And Surrounding Skin

The periwound is as important as the wound. As clinicians, we should carefully assess the wound bed, but we need to remember also to assess the periwound and surrounding skin. The periwound is a protective barrier and extends 4cm from the wound bed. Chronic wounds may manifest any of the following characteristics, depending on wound type: erythema, induration, epibole, ecchymosis, hyperkeratosis; and they can change in shape.¹¹

Five-Step Periwound Assessment

In assessing the tissue surrounding the wound bed, make careful note of the following:

1. Temperature
2. Location
3. Shape
4. Color
5. Wound depth

Simplifying Wound Treatments By Wound Depth And Exudate Amount

Wound Depth	Exudate Amount	Primary Dressing	Secondary Dressing	Frequency
Intact Non-Blanchable	Dry	Moisturizer	Transparent	Incontinent Patients –
		Skin Barrier	Hydrocolloid	Every Episode
Suspected Deep Tissue Injury (Intact)		Skin Prep	Composite	Daily
		Transparent Hydrocolloid	Gauze	Hydrocolloid Every 3 Days
Partial-Thickness Blister	Dry to Minimal	Hydrocolloid	Foam	And PM
		Composite Gauze		
Scab Stage II Pressure		Skin Barrier	Transparent	Daily To Monitor
		Oil Emulsion	Composite	Every 2 Days
Moisture-Associated Skin Damage (MASD)*	Moderate	Petrolatum Gauze	Hydrocolloid	Twice A Week
		Medical Honey Gel	Border Gauze	May Require Higher Dressing Frequency If Incontinent
Stage II Pressure Moisture-Associated Skin Damage (MASD)*		Hydrocolloid		
		Honey Alginate	Composite	Daily
Stage III Pressure		Calcium Alginate	Hydrocolloid	May Require Higher Dressing Frequency If Incontinent
		Gelling Fiber	Gauze	<i>NOTE: Protect Skin And Manage Moisture</i>
Stage IV Pressure		Hydrocolloid	Foam	
		Oil Emulsion	Foam	
Full-Thickness	Dry to Minimal	Petrolatum Gauze	Super Absorbent	Daily
		Collagen (Granulating Wounds)	Composite	Every Other Day
Stage III Pressure		Medical Honey	Foam	Twice A Week
		Hydrogel Filler	Super Absorbent	Daily
Stage IV Pressure		Oil Emulsion	Negative Pressure Wound Therapy (NPWT)	3 Times A Week
		Petrolatum Gauze		
Suspected Deep Tissue Injury (Non-Intact)		Enzymatic Ointment As Indicated (Daily)		
		Collagen (Granulating Wounds)	Hydrocolloid	Daily
Tunneling/Undermining*	Moderate to Heavy	Honey Alginate	Composite	May Require Higher Frequency If Incontinent Or Copious Exudate
		Calcium Alginate	Foam	
Tunneling/Undermining*		Hydrofiber	Super Absorbent	
		Enzymatic Ointment As Indicated (Daily)	Negative Pressure Wound Therapy (NPWT)	3 Times A Week
Tunneling/Undermining*				<i>NOTE: Always Fill Dead Space Of Deep, Undermining, And Tunneling Wounds To Promote Healing</i>

1. Control Biofilm With Debridement Methods, Topical Agents, Antimicrobial, And Or Bacterostatic Dressing As Indicated.
2. Pressure Ulcers/Injuries – Offloading Is Key
3. Non-Bordered And Bordered Secondary Dressing Selection Depending On Location Of Wound And Incontinence.

Patient-Caregiver Education And Communication

Patient-caregiver education and communication are vital in chronic wound prevention and good healing outcomes. There should be collaboration among the patient, the caregiver, and the health care team. You will need to determine the current knowledge base of your patient's condition, support network, and any challenges moving toward their plan of care.

Conclusion

The leading method for preventing a wound from becoming "chronic" is to avoid all risk factors. The primary focus of treatment should be removing the underlying cause if all possible. The correct wound type diagnosis is imperative. Regular monitoring of wounds, appropriate consistent wound care, and maintaining a healthy lifestyle will help in prevention and to enhance the wound healing progress. Patient education, communication, and follow-up are vital in helping prevent chronic wound recurrence.

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HOW TO REACH US

Corporate Office:
P.O. Box 189 – 206 Commerce St., Hinesburg, VT 05461

Phone: (802) 482-4000 – **Fax:** (802) 473-3113

E-mail: info@kestrelhealthinfo.com

WEBSITE: www.kestrelhealthinfo.com, www.woundsource.com

Editorial inquiries: editorial@kestrelhealthinfo.com

Advertising inquiries: sales@kestrelhealthinfo.com

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