Special Considerations in Wound Bed Preparation 2011: An Update

Part one of this article published here. Part two will be published in the Summer 2012 issue of *Wound Care Canada.*

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Introduction

This article builds and expands upon the concept of wound bed preparation introduced by Sibbald et al. in 2000 as a holistic approach to wound diagnosis and treatment of the cause and patient-centred concerns such as pain management, optimizing the components of local wound care: *D*ebridement, *I*nfection and persistent inflammation, along with *M*oisture balance before *E*dge effect for healable but stalled chronic wounds.

Background

This article incorporates a framework for assessment, diagnosis and treatment of wounds along the continuum toward optimal healing.[1] The authors will introduce evidence-based and best clinical practice-based strategies for providing holistic and patient-centred care. It is important to treat the whole patient and not just the "hole" in the patient. The preparation and optimization of the wound bed for functional healing may not always result in complete healing, despite clinicians' comprehensive team efforts. It is also important to recognize that some wounds may remain in the static or "stalled" phase of the wound-healing trajectory.

The authors recognize that wound-healing trajectories can be heterogeneous and nonuniform. They will explore several concepts to effectively manage non-healable wounds or a new category the authors term as "maintenance wounds," which are potentially healable but with existing patient or system barriers to effective treatment. These include patient adherence or competence to participate in treatment plans and systems-based errors embracing logistical issues that impede optimal healing. By reading this article, clinicians will comprehend and apply clinical criteria to help select and use the appropriate topical agents for superficial critical colonization versus systemic anti-infective agents for deep and surrounding tissue infection utilizing the mnemonics NERDS and STONEES. Clinicians will also be able to interpret the new bedside diagnostic tests introduced in this article, which may help in the identification of wounds stuck in the inflammatory stage.

This 2011 wound bed preparation update also links evidence-informed practices to the evidence summarized in the recent Best Practice Guidelines from the Registered Nurses' Association of Ontario. To date, 3 best practice documents related to the treatment of wounds (pressure, venous and diabetic) have been issued by the Registered Nurses' Association of Ontario, and the components related to local wound care have been considered for this summary along with updated literature searches. The information includes a quick reference guide of the key bedside assessment and treatment steps organized with the components of the wound bed preparation paradigm (Table 1).

Introduction

As the population ages, acute and chronic wounds will become more frequent and prevalent, with increased chronicity. Any wound older than 6 weeks is considered chronic.[2] Preparing the wound bed was first described in 2000 by Sibbald et al.[3] and Falanga,[4] with sequential updates by Sibbald et al. in 2003[5] and 2006–2007[6] and a reprint by the World Health Organization in 2010.[6] The 2011 updated evidence-informed practice documents are presented here, and link the wound bed preparation paradigm to the evidence-based literature, expert opinion, the clinical environment and organizational context. In Table 1, the 3 components of Sackett's triad have been accommodated: clinical evidence and expert opinion with the need to address patient preference (patient-centred concerns). In addition, the WoundPedia Best Practice summaries (www.woundpedia.com) utilized in this update are meant to provide

a practical, easy-to-follow guide or as a bedside enabler for patient care. The levels of scientific evidence-based grading systems are outlined in Table 2.[7]

For more detailed information on this grading system, the reader is referred to the Registered Nurses' Association of Ontario Best Practice Guidelines (www.rnao.org/bestpractices.com) and/or the designated references.

Chronic wounds: Nonhealable and maintenance wound categories

The holistic approach to healable wound management as outlined in Table 1 stresses an accurate diagnosis and successful treatment with a team approach (see Enabler: persons with healable chronic wound[s]). For wounds that do not have the ability to heal, the approach is different (see Enabler: persons with nonhealable or maintenance wound[s]). In these individuals, the inability to heal (nonhealable wound) may be due to an inadequate blood supply and/or the inability to treat the cause or wound-exacerbating factors. The second category, a maintenance wound, occurs when the patient refuses the treatment of the cause (e.g. will not adhere to compression therapy) or there is a health system error or barrier (e.g. no plantar pressure redistribution is provided in the form of footwear or the patient cannot afford the device). These may change, and periodic re-evaluation may be indicated (see Enabler).

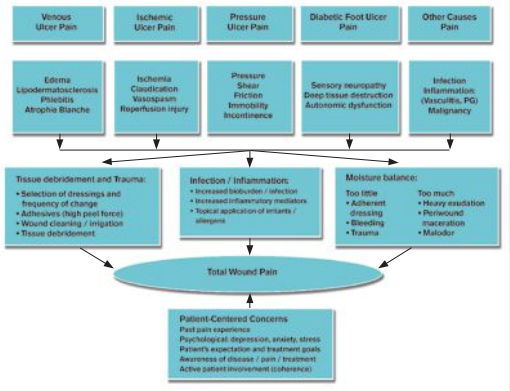
Chronic wounds are disabling and constitute a significant burden on patients' activities of daily living (ADLs) and the healthcare system. Of persons with diabetes, 2–3% develop a foot ulcer annually, while the lifetime risk of a person with diabetes developing a foot ulcer is as high as 25%.[8] It is estimated that venous leg ulcers (VLUs) affect 1% of the adult population and 3.6% of people older than 65 years.[9] As our society continues to age, the problem of pressure ulcers (PUs) is growing. Each of these common types of chronic wounds requires an accurate and concise diagnosis and appropriate treatment as part of holistic care.

Local wound care may also be difficult to optimize. Chronic wounds are often recalcitrant to healing, and may not follow the expected trajectory that estimates a wound should be 30% smaller (surface area) at week 4 to heal in 12 weeks.[10,11] If all 5 components of wound bed preparation have been corrected (cause, patient-centred concerns and the 3 components of local wound care) and a healable wound is stalled, re-evaluation of the diagnosis and treatment plan is necessary to ensure each component has been idealized before considering active local advanced therapies (edge effect). This update will clarify the system outlined above, dividing chronic wounds into healable, maintenance and nonhealable categories. The authors will develop the clinical parameters around critical colonization with any 3 or more of the 5 NERDS mnemonic criteria for topical therapy versus any 3 or more of the 7 STONEES mnemonic criteria associated with the deep and surrounding skin infection for systemic antimicrobial agents.

The updated wound bed preparation 2011quick reference guide is intended for all wound-healing practitioners from basic to intermediate or advanced levels ideally, organized in transdisciplinary teams. To clarify the rationale for the evidence-informed practices, the authors discuss each item individually with reference to key supporting literature and enablers for practice where indicated.

FIGURE 1

The wound-associated pain model: the wound, the cause, the patient



Identify and treat the causes of the wound

1a: Determine if there is adequate blood supply to heal

This is often important, especially for ulcers on the leg or foot. It is important to inspect the foot and lower leg for signs of arterial compromise (dependent rubor, pallor on elevation and loss of hair on the foot or toes), as well as palpating for a plantar pulse (dorsalis pedis or posterior tibial). Practitioners need to remember that a small percentage of patients may have an anomalous or anatomical variance resulting in absence of the dorsalis pedis artery. A palpable pulse indicates a foot arterial pressure of 80 mmHg or higher. The authors record a pulse as present or absent. However, a palpable pulse may not always exclude an arterial etiology. Although a foot pulse might be palpable, the nonhealing wound might be situated in a different angiosome that has to be revascularized in order to induce healing (angiosome model).[12] Doppler examination of the

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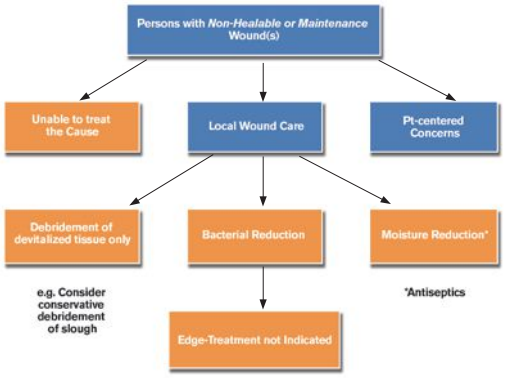
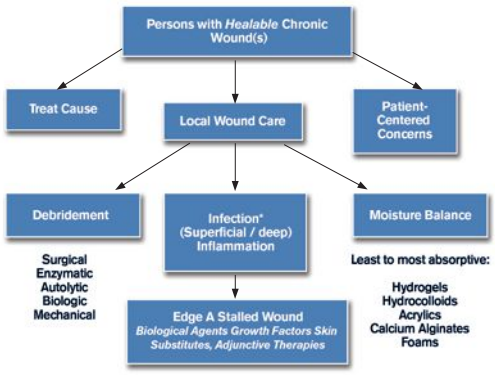
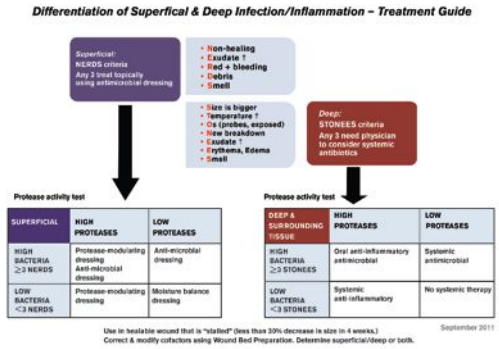
Enabler. Persons with nonhealable chronic wound(s)



Enabler. Persons with healable chronic wound(s)



Enabler. Sibbald cube



Enabler. Healable wounds: Key factors in deciding on the method of debridement

	Surgical	Enzymatic	Autolytic	Biologic	Mechanical
Speed	1	3	5	2	4
Tissue selectivity	3	1	4	2	4
Painful wound	5	2	1	3	4
Exudate	1	4	3	5	2
Infection	1	4	5	2	3
Cost	5	2	1	3	4

**Where 1 is the most desirable and 5 is least desirable

Nonhealable maintenance preferred & newer antiseptics

Agent	Effects
Acetic Acid (0.5% to 5%) *(Hydrochlorous Acid)*	▶ Lowers pH ▶ Effective against pseudomonas ▶ May select out *(Staph aureus)* ▶ May cause local stinging/burning
Chlorhexidene 2% alcohol solution or 0.5% aqueous solution *(PHMB derivatives)*	▶ Active against gm –ve & gm +ve organisms ▶ Low tissue toxicity ▶ Water based formulations best for wounds
Povidone iodine 10% aqueous solution delivers 0.9% iodine at wound bed	▶ Broad spectrum of activity, gram negative, gram positive, anaerobes, fungi, viruses, biofilms ▶ Activity ↓ in the presence of pus or exudate ▶ May be toxic to thyroid with prolonged use over large areas ▶ Slow release formulation may provide autolytic debridement/even lower tissue toxicity
Crystal violet-Methylene Blue	▶ Broad spectrum antimicrobial activity ▶ Lower tissue toxicity with low release form ▶ Can be used with enzymatic agents to prevent secondary bacterial infection

Antiseptic agents where harmful effects may be greater than helpful

Dyes-Scarlet red, Proflavine	Select out gram negative
Na Hypochlorite-Dakins, Eusol	Toxic = Bleach
Hydrogen Peroxide	Action = Fizz
Quatemary Ammonia-Centrimide	Very high tissue toxicity

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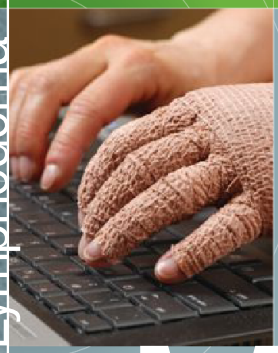

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TABLE 1

Quick reference guide to wound bed preparation, 2011

#	Recommendations for wound bed preparation	RNAO level of evidence
1	**Treat the cause** a. Determine if there is adequate blood supply to heal b. Identify the cause(s) as specifically as possible or make appropriate referrals c. Review cofactors/comorbidities (systemic disease, nutrition, medications) that may delay or inhibit healing d. Evaluate the person's ability to heal: healable, maintenance, non-healable	IV
2	a. Develop an individualized plan of care b. Treat the cause(s) related to specific wound etiology/diagnosis	IV
3	**Patient-centred concerns** Assess and support individualized concerns a. Pain b. Activities of daily living c. Psychological well-being d. Smoking e. Access to care, financial limitations	IV
4	Provide education and support to the person and his/her circle of care (including referral) to increase adherence (coherence) to the treatment plan	IV
5	**Local wound care** Assess and monitor the wound history and physical examination	
6	**Gently cleanse wounds with low-toxicity solutions: saline, water and acetic acid (0.5–1.0%)** Do not irrigate wounds where you cannot see where the solution is going or cannot retrieve (or aspirate) the irrigating solution	Ib
7	Debride: **Healable wounds** – sharp or conservative surgical, autolytic, mechanical, enzymatic, biological (medical maggots); **non-healable and maintenance** – conservative surgical or other methods of removal of nonviable slough	IV
8	Assess and treat the wound for superficial critical colonization/deep infection/abnormal persistent inflammation (mnemonic NERDS), deep infection (mnemonic STONEES) or persistent inflammation: **any 3 NERDS** – treat topically: non-healing, Exudate, Red-friable tissue, Debris, Smell; **any 3 STONEES** – treat systemically: ↑ Size, ↑ Temperature, Os, New breakdown, ↑ Exudate, ↑ Erythema/edema (cellulitis), Smell; persistent inflammation (non-infectious): topical and/or systemic anti-inflammatories	IIa
9	Select a dressing to match the appropriate wound and individual person characteristics **Healable wounds: autolytic debridement:** alginates, hydrogels, hydrocolloids, acrylics **Critical colonization:** silver, iodides, PHMB, honey **Persistent inflammation:** anti-inflammatory dressings **Moisture balance:** foams, Hydrofibers, alginates, hydrocolloids, films, acrylics **Nonhealable, maintenance wounds:** chlorhexidine, povidone-iodine	IV
10	Evaluate expected rate of wound healing: healable wounds should be 30% smaller by week 4 to heal by week 12 Wounds not healing at the expected rate should be reclassified or reassessed, and the plan of care revised	III–IV
11	Use active wound therapies (e.g. skin grafts, biological agents, adjunctive therapies) when other factors have been corrected and healing still does not progress (stalled wound)	Ia–IV
12	**Provide organization support** For improved outcomes, education and evidence-informed practice must be tied to interprofessional teams and improved cost-effective patient care outcomes with the cooperation of healthcare systems	IV

ankle brachial pressure index (ABPI) is indicated if the pulse is not palpable or to assess the appropriateness of high or modified compression bandaging for venous ulcers (Table 2).

The audible Doppler signals may also be useful diagnostically: a triphasic normal sound, a biphasic sound indicative of arterial compromise and the monophasic or absent signal with advanced ischemia. Complete segmental lower-leg arterial Doppler examinations are needed if there is a possibility of a proximal lesion or arterial restriction or blockage that is amenable to surgical bypass or endovascular dilatation. If the blood supply is inadequate or cannot be immediately determined, dressing selection should be based on a maintenance wound program with moisture reduction and bacterial reduction until further assessments are performed.

Toe pressures are useful because about 80% of people with diabetes and 20% of the nondiabetic

When wounds are trapped in the inflammatory phase, debridement is not complete…

Lorsque les plaies sont piégées dans la phase inflammatoire, le débridement n'est pas complet…

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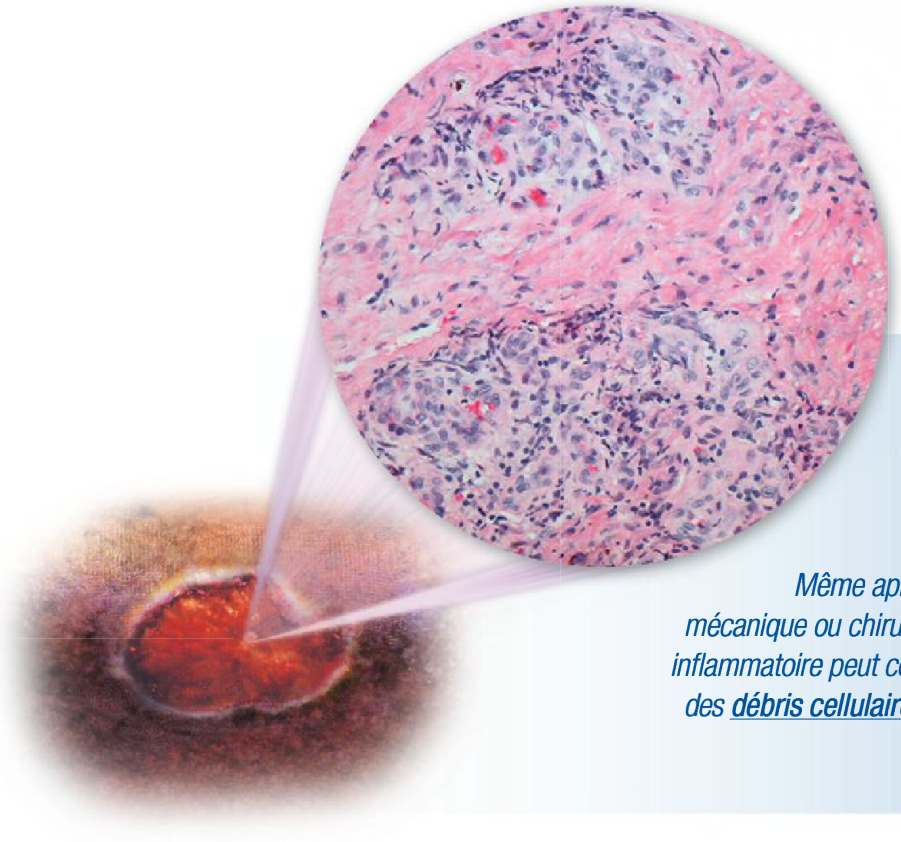

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Occasional slight transient erythema has been noted in surrounding tissue when applied outside the wound. One case of systemic hypersensitivity has been reported after 1 year of treatment with collagenase and cortisone.

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On a noté un érythème occasionnel et léger sur les tissus environnants lorsque l'application de l'onguent dépasse le pourtour de la plaie. Un cas d'hypersensibilité systémique a été rapporté après un an de traitement à la collagénase et à la cortisone.

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CLINICAL PHARMACOLOGY: Santyl® (collagenase) possesses the ability to digest insoluble collagen, undenatured and denatured, by peptide bond cleavage, under physiological conditions of pH and temperature. This ability makes it particularly effective in the removal of detritus from dermal lesions, contributing towards the more rapid formation of granulation tissue and subsequent epithelization of dermal ulcers and severely burned areas. Collagen in healthy tissue or in newly formed granulation tissue is not digested.

INDICATIONS: Santyl® (collagenase) is a sterile ointment indicated for the debridement of dermal ulcers or severely burned areas.

CONTRAINDICATIONS: Application is contraindicated in patients who have shown local or systemic hypersensitivity to collagenase.

WARNINGS: Debilitated patients should be closely monitored for systemic bacterial infections because of the theoretical possibility that debriding enzymes may increase the risk of bacteremia.

PRECAUTIONS: The enzyme's optimal pH range is 6 to 8. Significantly lower pH conditions have a definitive adverse effect on the enzyme's activity, and appropriate precautions should be carefully taken. The enzymatic activity is also adversely affected by detergents, hexachlorophene and heavy metal ions such as mercury and silver that are used in some antiseptics and by cobalt, magnesium and manganese. When it is suspected such materials have been used, the site should be carefully cleansed by repeated washings with normal saline before Santyl® (collagenase) ointment is applied. Soaks containing metal ions or acidic solutions such as Burow's solution should be avoided because of the metal ion and low pH. Cleansing materials such as hydrogen peroxide or Dakin's solution followed by sterile normal saline do not interfere with the activity of the enzyme. The ointment should be confined to the area of the lesion in order to avoid the possible risk of irritation or maceration of normal skin; however, the enzyme does not damage newly forming granulation tissue. A slight erythema has been noted occasionally in the surrounding tissue particularly when the enzyme ointment was not confined to the lesion. This can be readily controlled by protecting the healthy skin with a material such as zinc oxide paste. Since the enzyme is a protein, sensitization may develop with prolonged use.

ADVERSE REACTIONS: Although no allergic sensitivity or toxic reactions have been noted in the recorded clinical investigations to date, one case of systemic manifestations of hypersensitivity has been reported in a patient treated for more than one year with a combination of collagenase and cortisone. Irritation, maceration or erythema has been noted where prolonged contact of normal skin with Santyl® (collagenase) ointment has been allowed, either by application of the ointment to areas of normal skin or by excessive application of ointment to the wound crater with subsequent spread to normal skin when dressings are applied. The reported incidence for this type of reaction was 1.8%.

SYMPTOMS AND TREATMENT OF OVERDOSE: Symptoms: To date, the irritation, maceration or erythema reported on prolonged contact of normal skin with Santyl® (collagenase) ointment constitute the only symptoms of overdosage reported. **Treatment:** Santyl® (collagenase) ointment can be rendered inert by the application of Burow's solution USP (pH 3.6 - 4.4) to the treatment site. If this should be necessary, reapplication should be made only with caution.

DOSAGE AND ADMINISTRATION: For external use only. Santyl® (collagenase) ointment should be applied once daily, or more frequently if the dressing becomes soiled (as from incontinence) in the following manner: **(1)** Prior to application the lesions should be gently cleansed with a gauze pad saturated with sterile normal saline, to remove any film and digested material. If a stronger cleansing solution is required, hydrogen peroxide or Dakin's solution may be used, followed by sterile normal saline. **(2)** Whenever infection is present, as evidenced by positive cultures, pus, inflammation or odor, it is desirable to use an appropriate antibacterial agent. Should the infection not respond, therapy with Santyl® (collagenase) ointment should be discontinued until remission of the infection. **(3)** Santyl® (collagenase) ointment should be applied (using a tongue depressor or spatula) directly to deep wounds, or when dealing with shallow wounds, to a non-adherent dressing or film dressing which is then applied to the wound. The wound is covered with an appropriate dressing such as a sterile gauze pad and properly secured. **(4)** Use of an occlusive or semi-occlusive dressing may promote softening of eschar, if present. Alternatively, crosshatching thick eschar with a #11 blade is helpful in speeding up debridement then cleanse with sterile saline. It is also desirable to remove as much loosened detritus as can be done readily with forceps and scissors. **(5)** All excess ointment should be removed each time the dressing is changed. **(6)** Use of Santyl® (collagenase) ointment should be terminated when debridement of necrotic tissue is complete and granulation is well under way.

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PHARMACOLOGIE CLINIQUE : Santyl® (collagénase) a la capacité de digérer le collagène insoluble, non dénaturé et dénaturé, par clivage de la liaison peptidique à un pH et à une température physiologiques. Cette caractéristique le rend particulièrement efficace dans l'élimination des déchets des lésions dermiques favorisant ainsi la formation du tissu de granulation et l'épithélialisation ultérieure des zones dermiques ulcérées et gravement brûlées. Le collagène des tissus sains ou du nouveau tissu de granulation n'est pas digéré.

INDICATIONS : Santyl® (collagénase) est un onguent stérile indiqué pour le débridement des zones dermiques ulcérées ou gravement brûlées.

CONTRE-INDICATIONS : L'application est contre-indiquée chez les patients ayant présenté une hypersensibilité locale ou systémique à la collagénase.

MISE EN GARDE : Les patients atteints de conditions débilitantes doivent être surveillés étroitement pour éviter la généralisation des infections bactériennes. Les enzymes de débridement augmenteraient le risque de bactériémie.

PRÉCAUTIONS : Le pH optimal de l'enzyme est de 6 à 8. Un pH nettement inférieur à un effet nettement adverse sur l'action de l'enzyme et des précautions appropriées doivent alors être prises. L'action de l'enzyme est également contrariée par les détergents, l'hexachlorophène et les ions de métaux lourds, comme le mercure et l'argent, présents dans certains antiseptiques, et par le cobalt, le magnésium et le manganèse. Quand on soupçonne l'utilisation de ces produits, la zone affectée doit être soigneusement nettoyée par des lavages répétés avec une solution saline avant l'application de l'onguent Santyl® (collagénase). Les bains contenant des ions de métaux ou des solutions acides comme la solution de Burow doivent être évités en raison de l'ion métal et du faible pH. Les solutions nettoyantes comme l'eau oxygénée ou la solution de Dakin suivie d'une solution stérile saline n'entravent pas l'action de l'enzyme. L'application de l'onguent doit se limiter à la zone affectée pour éviter le risque possible d'irritation ou de macération de la peau saine. Cependant, l'enzyme n'altère pas le nouveau tissu de granulation. Un érythème bénin dans le tissu avoisinant pourrait se produire. Cela peut facilement être évité en protégeant la peau saine avec un produit comme de la pâte d'oxyde de zinc. Compte tenu de la nature protéique de l'enzyme présent dans le médicament, son emploi prolongé pourrait amener une sensibilisation.

EFFETS SECONDAIRES : Bien qu'aucune sensibilité allergique ni réaction toxique n'aient été notées à ce jour dans les compte rendus d'études, on a signalé un cas de manifestations systémiques d'hypersensibilité chez un patient traité pendant plus d'un an avec une association de collagénase et de cortisone. On a noté de l'irritation, de la macération ou de l'érythème dans le cas de contact prolongé de la peau normale avec l'onguent Santyl® (collagénase), soit par application de l'onguent sur les régions normales de la peau, soit par application excessive de l'onguent dans le cratère de la plaie, permettant à celui-ci de s'étendre à la peau normale lors de l'application des pansements. L'incidence signalée de ce type de réaction était de 1,8 %.

SYMPTÔMES ET TRAITEMENT DU SURDOSAGE: Symptômes: Jusqu'ici, l'irritation, la macération ou l'érythème signalés en cas de contact prolongé de la peau saine avec l'onguent Santyl® (collagénase) représentent les seuls symptômes signalés de surdosage. **Traitement:** On peut rendre l'onguent Santyl® (collagénase) inerte en appliquant la solution de Burow U.S.P. (pH 3.6-4.4) sur la plaie. La réapplication du produit, si elle est considérée nécessaire, ne se fera qu'avec prudence.

POSOLOGIE ET ADMINISTRATION : Pour usage externe seulement. L'onguent Santyl® (collagénase) doit être appliqué une fois par jour ou plus fréquemment si le pansement se souille (à cause d'incontinence par exemple) de la façon suivante : **(1)** Avant application, les lésions doivent être nettoyées doucement avec une gaze saturée d'une solution stérile saline normale pour enlever toute pellicule et toute matière digérée. Si l'on a besoin d'une solution nettoyante plus puissante, on peut utiliser de l'eau oxygénée ou de la solution de Dakin suivie de solution stérile saline normale. **(2)** En cas d'infection, révélée par la présence de cultures positives, de pus, d'une inflammation ou d'une odeur, il serait souhaitable d'employer un agent antibactérien approprié. Il faut interrompre le traitement au Santyl® (collagénase) jusqu'à rémission de l'infection, si l'infection ne se résorbe pas. **(3)** Appliquer Santyl® (collagénase) directement sur les blessures profondes à l'aide d'un abaisse-langue ou d'une spatule. Pour les plaies superficielles, appliquer l'onguent sur une compresse non adhérente ou un pansement transparent à être déposée sur la plaie; puis recouvrir d'un pansement approprié tel une compresse de gaze stérile adéquatement retenue. **(4)** L'utilisation d'un pansement occlusif ou semi-occlusif peut favoriser le ramollissement de l'escarre, le cas échéant. Ou, si l'on hachure une escarre épaisse à l'aide d'une lame numéro 11, on peut accélérer le débridement. Nettoyer alors avec une solution saline stérile. Il est également souhaitable d'enlever autant de détritus lâches que possible à l'aide de pinces et de ciseaux. **(5)** Enlever tout excès d'onguent à chaque renouvellement du pansement. **(6)** Arrêter les applications de l'onguent Santyl® (collagénase) dès que le tissu nécrosé est suffisamment débridé et que le bourgeonnement est bien entamé.

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population have calcified large leg arterial vessels that are nonpliable and stiff, leading to falsely high ABPI levels, often greater than 1.3.[13] When ABPI levels are this high, no conclusions can be drawn about the quality of limb perfusion without further investigation. In Table 3, the arterial status is related to the vascular testing results.[13,14]

1b: Identify the cause(s) as specifically as possible or make appropriate referrals

A comprehensive wound assessment is required to determine the cause of the wound. In order to achieve this, a holistic approach to the patient assessment is needed. An interprofessional team approach will facilitate a comprehensive review of the whole patient, the environmental factors and the wound. In a recent community, comprehensive interprofessional assessment of leg and foot ulcer patients, more than 60% of diagnoses were changed or made more specific, leading to the implementation of best practices, thus facilitating the optimization of wound bed preparation and improving healing rates of chronic wounds.[15]

1c: Review cofactors/comorbidities (systemic disease, nutrition, medications) that may delay or inhibit healing

Wound healing can be delayed or interrupted in persons with a coexisting systemic disease and the multiple comorbidities associated with chronic wounds. In the case of diabetes, excess glycosylation of hemoglobin due to poor diabetic glucose control can result in a prolonged inflammatory phase in addition to decreased neutrophil and macrophage phagocytosis of bacteria. Furthermore, diabetes affects the ability of erythrocytes to deliver oxygen to the wound, a fundamental step in collagen synthesis and tissue proliferation[16] along with numerous other important factors in wound healing. An original investigation by Markuson et al demonstrated that individuals with lower glycated hemoglobin levels had shorter healing times.[17] This translated to a cost reduction because the closed wounds had a decreased risk of infection compared with the ulcers that were still in the healing phase.

A detailed review and clinical analysis of patient cofactors and comorbidities that may influence healing should be carried out in a systems-based approach. Systemic diseases such as diabetes or autoimmune disease may interfere with the stages of wound healing and stall or prevent healing.

A low protein intake or relative deficiency can prevent the production of granulation tissue and will contribute to a stalled healing environment for the wound. A given albumin measurement in a patient implies the nutritional status over a few months, and these levels are a gross indicator of long-term nutritional deficit. Albumin levels measure the large reservoir of amino acids that serve as the fundamental building blocks for wound

TABLE 2

Levels of evidence employed by RNAO guideline development panels (2005)[7]

Level	Type of evidence
Ia	Evidence obtained from meta-analysis or system review of randomized controlled trials
Ib	Evidence obtained from at least 1 randomized controlled trial
IIa	Evidence obtained from at least 1 well-designed controlled study without randomization
III	Evidence obtained from well-designed, non-experimental descriptive studies such as comparative studies, correlation studies and case studies
IV	Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities

TABLE 3

Arterial status is related to vascular results[13,14]

ABPI	Toe pressure (mmHg)	Toe brachial index	Ankle Doppler waveform	TcPO2 (mmHg)	Diagnosis
>0.8	>80	>0.6	Normal/triphasic	>40	No relevant arterial disease
>0.5	>50	>0.4	Biphasic/monophasic	30–39	Some arterial disease: modify compression
>0.4	>30	>0.2	Biphasic/monophasic	20–29	Arterial disease predominates
<0.4	<30	<0.2	Monophasic	<20	High risk for limb ischemia

Adapted with permission from references 14 and 15.

healing. Several other patient stressors can also influence albumin levels.[18] Normal serum albumin levels are 3.4–5.4 g/dL,[19] and levels of 2.0–3.4 g/dL are associated with potentially delayed healing. Wounds in patients with these levels and may need to be treated as maintenance wounds until the albumin level is corrected.

Prealbumin (transthyretin) is a more sensitive indicator of protein deficiency, reflecting levels over 18–21 days. Transferrin is often thought of as an indirect measure of nutrition; however, levels are elevated in response to infection or inflammation, and results can therefore be misleading in persons with a chronic wound.[20] Cost and access to transferrin testing may be a challenge in some practice settings. Published literature attributes recumbent positioning of patients with a direct decrease in serum liver proteins such as albumin, prealbumin and transferrin.[21,22] Therefore, in utilizing the "whole patient" concept, we should optimize activity and mobilization.

Individualized patient medicine reconciliation should take place as part of any wound management protocol. Several medications that may alter the healing processes on the cellular level need to be identified. Some medications important to note in the assessment of a wound are high doses of systemic steroids, immunosuppressive drugs and antimetabolite cancer chemotherapy. Vitamin E intake of more than the recommended 100 IU daily can impair healing[23] because of its oxygen-scavenging property at the tissue level, opposite to the oxygen-sparing property of vitamin C.

1d: Evaluate the person's ability to heal: Healable, maintenance, nonhealable

Categorizing a wound according to its ability to heal (healability) assists the clinician in determining an accurate diagnosis along with a realistic individualized treatment approach. Adequate tissue perfusion is necessary for a healable wound. As outlined above, decreased vasculature will increase the risk of infection and decrease healability. In order to be classified as a healable wound, the wound should have several attributes including an adequate blood supply; the cause of the wound must be corrected; and existing cofactors, conditions or medications that could potentially delay healing must be optimized or ideally corrected. A maintenance wound is a wound that may be healable but that either healthcare system factors or patient-related issues are preventing from healing. A nonhealable wound is a wound that does not have an adequate blood supply to support healing or the cause cannot be corrected. In nonhealable wounds, moist interactive healing is contraindicated and debridement should be on a conservative basis only (expert opinion for SCALE [Skin Changes at Life's End] document).

The healability percentages of consecutive consenting homecare patients with leg and foot ulcers from Toronto and Mississauga (Ontario, Canada) districts have been tabulated in the final column of Table 4.[24] The results indicated that most subjects (69.0%) had a demonstrated ability to correct the cause and achieve adequate circulation for healing. Determining if a patient has a healable, nonhealable (5.2%) or maintenance (24.9%) wound allows the clinician to identify and address specific individualized challenges, particularly for the nonhealable and maintenance wound patients. Along with the patient's input, the clinician is able to tailor the nonhealable or maintenance care plan, facilitating responsible use of available resources along with realistic treatment goals. In general, advanced active therapies are not indicated for maintenance or nonhealable wounds.

When a healable wound does not progress at the expected rate, a chronic and stalled wound results. These wounds are more prevalent in older adults and are attributed to the aged skin and comorbidities such as neuropathy, coexisting arterial compromise, edema, unrelieved pressure, inadequate protein intake, coexisting malignancy and some medications. Persistent inflammation may be the cause of a stalled wound and in some cases may not be correctable. The presence of multiple comorbidities in some older adult patients implies that healing is not a realistic endpoint.[25] For nonhealable or maintenance wounds, pain and quality of life may be indicated as the primary goals of care. Palliative wound care often includes nonhealable wounds, but patients undergoing palliative care may have maintenance or even healable wounds.

Frequently, skin changes at life's end may be associ-

TABLE 4

Determining the healability of a wound

Wound prognosis	Treat the cause	Blood supply	Coexisting medical conditions/ drugs	No. of wounds (%) with ability to heal*
Healable	Yes	Adequate	Not preventing healing	121 (69.9%)
Maintenance	No*	Adequate	May prevent healing	43 (24.9%)
Nonhealable, including SCALE	No	Usually inadequate	May prevent healing	9 (5.2%)

Modified from © Sibbald, Krasner, Lutz SCALE document 2010.
*Results from a comprehensive interprofessional assessment of leg and foot ulcers.[15]
SCALE = skin changes at life's end

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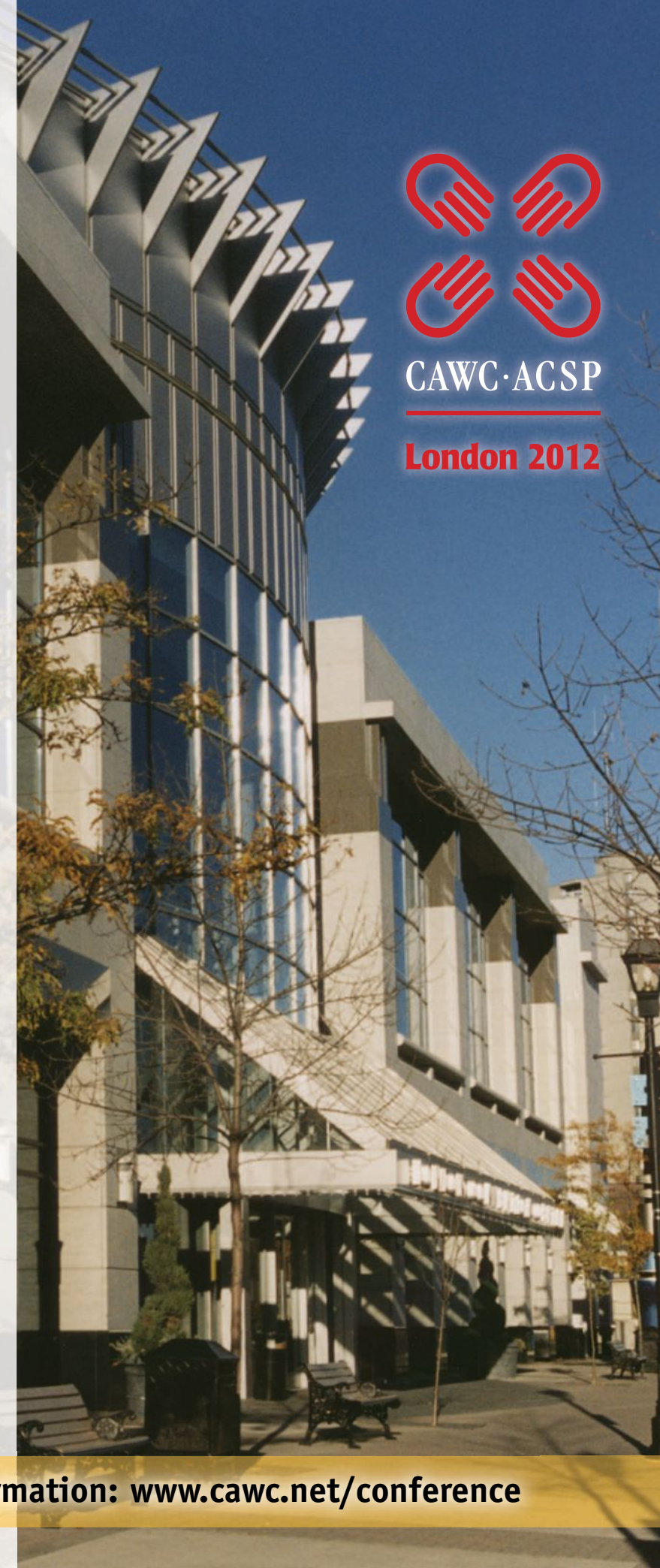

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ated with individual risk factors and comorbidities. In 2009, an 18-member international expert panel explored the issues and research literature surrounding end-of-life skin and wound care, including the Kennedy Terminal Ulcer (case series evidence)[26] and the concept of skin compromise.[27] The panel developed a consensus document entitled "Skin Changes at Life's End" (SCALE). A modified Delphi process with 52 international distinguished reviewers was utilized to reach consensus on the document. The 10 final consensus statements have clarified the authors' views on skin and wound conditions at the end of life.

Of the 10 SCALE consensus statements, statement 1 is key: "Physiologic changes that occur as a result of the dying process may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain. These changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care."[28] The panel explored the work by Kennedy, who published a descriptive study describing the phenomenon of PUs that occur in the sacral area of dying patients in a long-term care facility.[26] Kennedy's work was the first modern descriptive research to discuss this issue, which was first depicted in 1877 by Jean-Martin Charcot and termed "decubitus ominosus."

In an observational study that took place in a 10-bed teaching hospital palliative care unit, the staff reported that 5% of patients had skin changes of reddish-purple discoloration ranging from 2 hours to 6 days prior to death. These areas of intact skin rapidly became full-thickness PUs.[29] The staff turned patients hourly. This study provides observational data on some of the unavoidable skin changes at life's end.

TABLE 5

Types of wounds and treatment

Type of wound	Treatment of the cause in a healable wound
Venous ulcers	Compression therapy wraps for healing and stockings for maintenance
	High compression in absence of arterial disease if the ankle brachial pressure index >0.8 (ABPI or ABI) and modified compression for mixed vascular disease with ABPI 0.65–0.8 (extreme caution when 0.5–0.65)
Arterial ulcers	Revascularization where possible
	Angioplasty, stents or bypass (grafting or synthetic)
Pressure ulcers	Pressure redistribution to reduce pressure, friction and shear forces
	Optimize mobility and incontinence and moisture management
Diabetic foot ulcers	V = Confirm adequate vascular supply
	I = Infection treatment
	P = Plantar pressure redistribution according to local provisions
	S = Sharp surgical serial debridement

2a: Develop an individualized plan of care

Following the wound assessment as described above, an individualized wound plan of care should be developed by the interprofessional team. The plan must be tailored to the individual, taking into consideration his/her unique biopsychosocial needs, including:

- risk factors;
- comorbidities;
- quality-of-life issues;
- support systems/circle of care;
- access to care; and
- personal preferences.

As discussed by Sackett et al, individualized patient preference must be honoured and reflected in the wound care plan.[30] Sackett et al recognized 3 dimensions of equal importance: best available scientific evidence, clinical expertise and patient preference. This model of evidence-based medicine has been 1 of the most important healthcare trends in the past 20 years. Interprofessional, individualized, patient-centred care must drive the care process.[31]

The wound care plan of care should be as follows:

- in writing and part of the permanent healthcare record;
- routinely evaluated and updated; and
- updated with any significant change in the individual's health status.

2b: Treat the cause(s) related to specific wound etiology/diagnosis

Once an accurate type of wound is established, the treatment can be planned and implemented (Table 5). For example, in a person with a venous ulcer, compression therapy is contraindicated when ABPI is 0.5 or less, and a vascular consult is required for limb preservation.[32] Under the care of an expert wound care team, modified compression therapy for patients with ABPI between 0.5 and 0.8 is beneficial and assists perfusion by increasing pulsatile flow,[33] thereby decreasing venous pressure and facilitating the arterial–venous gradient.[34]

Importance of holistic, interprofessional, coordinated and collaborative care

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ful treatments plans can be a challenging undertaking, given the complexity of chronic wounds. A holistic interprofessional approach is required. Each member of the team possesses a unique professional skill set and knowledge base that should contribute to the individualized plan of care. Implemented treatment plans that do not yield wound-healing rates at the expected trajectory require a timely referral to an interprofessional team that can re-evaluate the diagnosis and causative factors. Redefining the treatment goals with input from the patient, family and healthcare provider is also essential.

2c: Modify (if possible) systemic factors/other cofactors that may impair healing: Medications, nutrition, hemoglobin, glycated hemoglobin, blood pressure, creatinine, CHF, LFTs and so on

A good example of a systemic factor that affects wound healing is the hemoglobin level. Because hemoglobin carries the oxygen that is essential for new tissue building, hemoglobin levels should be optimized. Potential negative influences for adequate hemoglobin are common in patients living with chronic illnesses such as renal disease, sickle cell and other anemias, to name a few.

Persons with cardiopulmonary disease, cardiovascular disease (including congestive heart failure) and related conditions have diminished extremity tissue perfusion as a result of reduced ejection fractions. In particular, heart failure and associated decreased tissue perfusion to the periphery results in edema accumulation in the lower extremities, creating a higher risk for lower-leg wound formation or delayed healing in existing wounds. In many cases, an internal medicine or subspecialty referral can optimize heart function and manage fluid balance and edema reduction. The offending co-contributors and cofactors that impede wound healing should be adjusted and corrected. Improving as many factors as possible may contribute to overall improvements in the patient's quality of life, reducing pain, improving mobility and facilitating improved wound outcomes.

A patient with a chronic wound may require a thorough nutritional assessment by a registered dietitian to address any underlying and correctable nutritional deficits. Proteins have a fundamental role throughout the wound-healing cycle, influencing the function of leukocytes, phagocytes, monocytes, lymphocytes and macrophages, all of which are integral to a normal healing trajectory.[35,36] A multinational European, prospective, randomized, controlled, double-blind trial has studied the effects of specific oral nutritional supplementation in non-malnourished patients specific to PU healing. The provision of a high-protein, micronutrient-enriched and arginine-supplemented diet resulted in improved healing rates and less wound care intensity for the care providers.[37]

Medications that may inhibit or delay wound healing should be reviewed, including the benefit, risk and dose of each medication. Refer to section 1c for more detail.

Address and treat individualized concerns

3a: Pain

McCaffery has stated that pain is what the patient says it is.[38] Every person experiences pain differently. Clinicians cannot treat pain that they do not know patients are experiencing. Pain measurement is subjective; however, the universally accepted measurement techniques are visual analogue scales (a 10-cm line with no pain at 1 end and worst possible pain at the other end, and the patient places an "x" at the appropriate point), the Faces Pain Scale (various levels of happy and sad faces) and the numerical rating scale. The numerical rating scale asks if the patient has any pain on a 0- to 10-point scale with the anchors that 0 is no pain, 5 is the pain associated with a bee sting and 10 would be the amount of pain experienced by slamming the car door on your thumb. Even in patients who cannot respond verbally, such as those with dementia, pain still needs to be assessed. There are pain scales for these patients that rely on nonverbal clues such as facial grimaces and pupil dilatation. (Assessment of pain for people with dementia can be found at www.hartfordign.org.) Pain levels should be recorded before dressing change, during dressing change and after dressing reapplication.

Krasner has delineated wound-associated pain at dressing change (intermittent and recurrent) versus incident pain from debridement or the persistent pain between dressing changes. Woo carried the Krasner concept further and demonstrated that anxiety and other patient-related factors could intensify the pain experience. The wound-associated pain model of Woo and Sibbald (Figure 1) defines pain from the cause of the wound as often being persistent or present between dressing changes and distinguishes this pain from the pain associated with local wound care components (dressing change, debridement, infection, lack of moisture balance). All of these factors can be modified by patient-centred concerns, including previous pain experience, anxiety, depression, mobility, awareness or lack of comfort with the setting and the procedure or treatment plan. Pain is an under-recognized and undertreated component of chronic wound care that has been demonstrated to be more important to patients than to healthcare professionals. Causes of pain at dressing change include the dressing material

adhering to wound base, skin stripping from strong adhesives and aggressive trauma from the cleansing technique (e.g. scrubbing with gauze).

Many patients also express chronic persistent pain between dressing changes, even at rest. A systematized approach should examine other systemic and disease factors that may play a role in precipitating and sustaining persistent wound-related pain. Common systemic factors are bacterial damage from superficial critical colonization or deep and surrounding compartment infections, deep structural damage (e.g. acute Charcot foot in patients with diabetes), abnormal inflammatory conditions (e.g. vasculitis, pyoderma gangrenosum) and periwound contact irritant skin damage from enzyme-rich wound exudate.

The impact of chronic unrelenting pain can be devastating, eroding the individual's quality of life and delivering a significant amount of stress. Increased levels of stress have been demonstrated to lower the pain threshold and decrease tolerance. The result is a vicious cycle of pain, stress/anxiety, anticipation of pain and worsening pain. Increased stress also activates the hypothalamus–pituitary–adrenal axis, producing hormones that modulate the immune system and compromising normal wound healing. Medications, including non-narcotic analgesics for moderate pain and narcotic analgesics for moderate to severe pain, are required to treat pain as outlined below. Consultation with a pain and symptom management team may be considered. Comprehensive management should also include careful selection of atraumatic dressing, prevention of local trauma, treatment of infection, patient empowerment, stress reduction and patient education.

The medical treatment of wound-associated pain and other components of pain management are outlined in documents from the World Union of Wound Healing Societies.[39,40] In general, wound-associated pain is nociceptive and stimulus-dependent (e.g. gnawing, aching, tender, throbbing) vs. neuropathic, non–stimulus-dependent or spontaneous pain (e.g. burning, stinging, shooting, stabbing). Nociceptive pain is treated according to the World Health Organization pain ladder, starting with aspirin and nonsteroidal anti-inflammatory drugs and then progressing to weak and strong narcotics.[41] Short-acting agents are often used to determine the dose of longer-acting agents, with short-acting agents then used for breakthrough. Neuropathic pain often responds to tricyclic agents, particularly second-generation agents that are high in anti-noradrenaline activity (nortriptyline and desipramine are often better than amitriptyline). Nonresponders may be treated with alternate agents such as gabapentin, pregabalin or other antiepileptic agents. Neuropathic pain occurs even with the loss of protective sensation and can awaken patients at night with lightning-like flashes of pain.

3b: Activities of daily living

The impact of living with a chronic leg ulcer on ADLs has the largest body of evidence, mainly using qualitative methodology, compared with other ulcer etiologies. Patients have reported numerous negative influences on their ability to carry out ADLs, including, pain, odour, mobility, finances and other aspects of living.[42] Depression and anxiety have been reported in as many as 68% of subjects. Another study highlighted the dominant impact of social isolation in patients suffering from chronic leg ulcers.[43] One study compared patients living with diabetic foot ulcers (DFUs) and those with amputation following foot ulcers, and concluded that a higher quality of life was reported in those who underwent lower-limb amputations.[44] Assessing the unique individual's concerns can be a time-consuming but necessary piece in addressing the patient's holistic needs. This highlights the emotional burden of living with a chronic wound.

3c: Psychosocial well-being

Psychosocial well-being is the dimension of quality of life that most people equate with the quality piece.[45] It includes the individual's psychological perspectives on his/her wound and overall life. It reflects the person's ability to socialize and interact with others.

There are many wound care interventions that can address and support a person's wound-related psychosocial issues. For example:

- If wound odour is an issue, charcoal or other odour-reducing dressings can be utilized.
- Dressing routines can be modified to accommodate individualized hygiene practices. For showers on Mondays, Wednesdays and Fridays, dressing changes can be coordinated to Mondays, Wednesdays and Fridays right after the shower.

3d: Smoking

Cigarette smoking is a leading preventable health problem, causing damage to the endothelial function of arteries throughout the body[46] and contributing to the development of vascular disease of both arterial and venous origin. The direct cutaneous effect of smoking has been clearly stated by Rayner.[47] "Cutaneous blood flow decreases as much as 40% to produce ischemia and impair healing.[48] Smoking a single cigarette creates a vasoconstrictive effect for up to 90 minutes, while smoking a packet results in tissue hypoxia that lasts an entire day."[49] Delayed wound healing for individuals

who use tobacco is attributed to resultant tissue hypoxia.[50] Smoking disrupts the normal healing process at many levels, decreasing cell proliferation and migration across the wound bed.[51–53] Cigarettes contain more than 4,000 substances, including carbon monoxide, nicotine and cyanide derivatives,[54] and each substance can negatively influence wound healing. Useful smoking-cessation strategies, including pharmacological and behavioural aspects and the effectiveness of these programs, are outlined by Ahn et al.[54]

Offering patients these strategies to quit smoking and improve tissue oxygenation may enhance healing.

3e: Access to care, financial limitations

Living with a wound can be a challenge for patients who have limited financial resources or access to care. Patients living with chronic illnesses compounded by a wound may have difficulties with transportation for medical appointments, and many are unemployed or on limited incomes. Depending on where the patient lives, there are differing resources available. Healthcare professionals should advocate for required patient resources. When a wound is determined to be maintenance or nonhealing, the healthcare team, along with the patient, can individualize the care plan to be most efficient for both the patient and the system.

4: Provide education and support to the person and his/her circle of care (including referral) to increase adherence (coherence) to the treatment plan

One strategy to provide support and education to a patient is by developing a therapeutic relationship.[55] Trust implies sharing of information and communication, and open dialogue allows the patient and those in his or her circle of care to understand that each person involved has a meaningful contribution. Active participation by the patient in the development of an individualized plan of care provides reassurance to the patient that the team is working with him or her to achieve the best possible outcome. This helps to enhance adherence to the agreed-upon treatment plan, as there is trust. An additional concept in team dynamics is unit cohesion or the process of "sticking together" for the accomplishment of a mission or task. If the patient provides substantive input into the treatment plan, there is a greater chance that the patient will adhere (cohere) to a given plan. By way of example, patient participation, such as removing the dressing at dressing change, should be encouraged as clinically appropriate. People in the patient's circle of care such as family, caregivers and healthcare professionals should also be part of the plan, including implementation and re-evaluation. Communication is paramount between healthcare sectors and professionals when managing chronic wounds. Once an expert team has determined that a wound is maintenance or nonhealable wound, it is important that this be communicated to prevent unnecessary investigations or interventions that may have already been unsuccessful. Healthcare professionals should review and educate the patient and family after determining their current knowledge gaps. Teaching the patient to report important signs that could indicate a deterioration of the wound is critical. Strategies to improve adherence have been reported in a comprehensive review by Osterberg and Blaschke.[56]

Special Considerations in Wound Bed Preparation 2011: An Update...

To be continued in *Wound Care Canada* Summer 2012

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