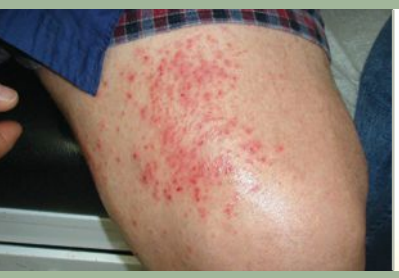
SPRING 2012 PRINTEMPS 2012
Vol.10 No.2 Vol.10 numéro 2
CAN $9.95 9,95 $ CAN

Wound Care

The Official Publication of the Canadian Association of Wound Care
La revue officielle de l'Association canadienne du soin des plaies

Soins des plaies



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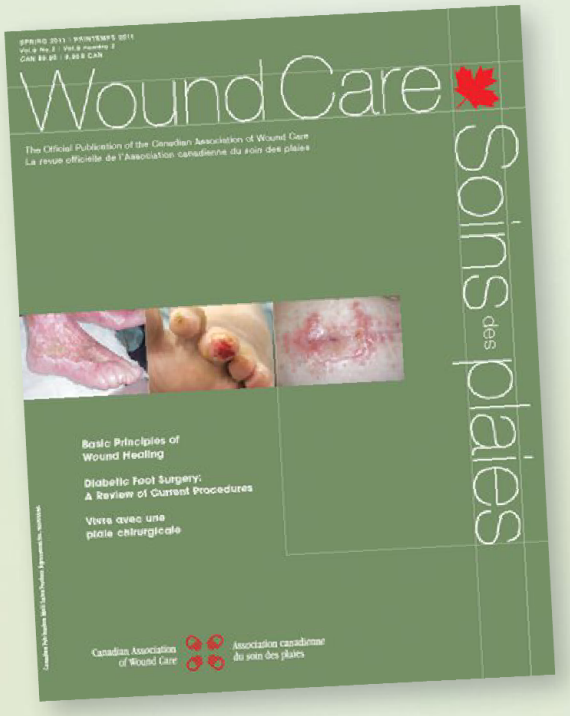

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Canadian Association of Wound Care
Association canadienne du soin des plaies

Wound Care / Soins des plaies

Spring 2012 / Printemps 2012
Volume 10, Number 2
Volume 10, Numéro 2
ISSN 1708-6884

Publication Management/
Gestion de la publication
BCS Communications Ltd.
255 Duncan Mill Road, Suite 803
Toronto, ON M3B 3H9

Wound Care Canada is published by BCS Communications Ltd., on behalf of the Canadian Association of Wound Care. Canada's first publication devoted entirely to wound care, *Wound Care Canada* addresses the needs of clinicians, patients, caregivers and industry.

Canadian Publication Mail
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Wound Care Canada is printed on acid-free paper that contains a minimum of 20 per cent post-consumer fibre.

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The Canadian Association of Wound Care is a non-profit organization of health-care professionals, industry participants, patients and caregivers dedicated to the advancement of wound care in Canada.

The CAWC was formed in 1995, and its official meeting is the CAWC annual conference held in Canada each year. The association's efforts are focused on five key areas: public policy, clinical practice, education, research and connecting with the international wound-care community. The CAWC works to significantly improve patient care, clinical outcomes and the professional satisfaction of wound-care clinicians.

L'Association canadienne du soin des plaies est un organisme sans but lucratif regroupant des professionnels de la santé, des gens de l'industrie, des patients et des membres du personnel soignant fortement intéressés à l'avancement des connaissances pour le soin des plaies au Canada.

Fondée en 1995, l'ACSP organise, chaque année, au Canada, un congrès qui lui tient lieu de réunion officielle, le Congrès annuel de l'ACSP. L'association consacre ses efforts dans cinq domaines particuliers : les politiques gouvernementales, la pratique clinique, la formation, la recherche et la création de liens avec la communauté internationale directement impliquée dans le soin des plaies. L'Association canadienne du soin des plaies vise une amélioration significative du soin donné au patient, des résultats cliniques et de la satisfaction professionnelle des spécialistes en soin des plaies.

Wound Care Alliance Canada Formed

A new advocacy group – the Wound Care Alliance Canada – was formed recently in an effort to raise the profile of wound care as a major health issue in Canada. Founding members of the Alliance are the Canadian Association of Wound Care (CAWC), the Canadian Association of Enterostomal Therapists (CAET), the Ontario Wound Interest Group (ONTWIG) and MEDEC (the national association representing the Canadian medical technology industry).

"We hope to bring key stakeholders to the table."

The group will be pursuing a national wound management innovation agenda, says Maureen Latocki, Acting Executive Director of the Alliance. "Through that agenda, we hope to bring key stakeholders to the table to explore how we may use innovation in wound prevention and care to drive forward opportunities to create more value in the healthcare system," she adds.

The Alliance has identified a natural alignment between improved value-for-money in wound care with government priorities at both the federal and provincial levels. Indeed, says Latocki, "Our initial step will be to speak with one voice to governments to disseminate key information regarding the costs associated with wound care, and how that information can be used by healthcare administrators and government policymakers to make evidence-based decisions regarding how wounds are prevented and managed in practice."

Future initiatives include the development of a Canadian Wound Innovation Network, which would bring together clinical, academic, industry and government expertise, to drive forward opportunities to leverage Canadian assets to generate world-class research and innovation in the field of wound management.

"We are pleased to be a founding member of the Wound Care Alliance Canada," says Peggy Ahearn, Executive Director of the CAWC. "Through this partnership with like-minded organizations, we can foster the policies and procedures necessary to deliver optimal wound care to those in need."

Wound Care Canada Publisher Appointed

The Canadian Association of Wound Care is pleased to announce that Douglas Queen has been appointed Publisher of the association's flagship publication, *Wound Care Canada*. Douglas has been in the wound care arena for more than 25 years. He completed his PhD in biomedical engineering in 1986, studying wound care dressings; he completed his MBA in 1996.

Douglas spent 13 years working for ConvaTec in both the Research & Development and Sales & Marketing areas. His career with ConvaTec gave him both European and North American experience. Currently, Douglas runs a medical device consultancy business and is also Editor of *International Wound Journal*.

"We are pleased to welcome Douglas to this position," says Peggy Ahearn, Executive Director of the Canadian Association of Wound Care. "His breadth of knowledge, and commitment to the prevention and management of wounds will serve the association well."

Douglas Queen: "I very much look forward to the opportunity to work with Canadian wound care clinicians."

Diabetes, Healthy Feet and You: Phase 2 Launch

Phase 2 of the Diabetes, Healthy Feet and You initiative has now rolled out across Canada! All 10 provinces across Canada will be delivering peer-led educational workshops that will motivate people with diabetes and their loved ones to learn about good foot care practices and how to apply them in their daily life. The workshops are entitled *PEP Talk: Diabetes, Healthy Feet and You*.

Along with the community peer leaders, the workshops will also be facilitated by a healthcare professional from each province. The focus of the program is to empower people living with diabetes to "discover and use their own innate abilities to gain mastery of their diabetes" and foot care.[1]

Recognizing the signs and symptoms of diabetic foot complications and accessing timely care can save limbs!

For dates and location information regarding sessions in your area, please visit www.diabetespeptalk.ca. For further information about the program, please contact Andrea Martin, Coordinator, Diabetes Healthy Feet and You Project, Canadian Association of Wound Care, by telephone (416-485-2292, ext. 221) or email (andrea@cawc.net).

Reference:

1. Funnell MM, Nwankwo R, Gillard ML, et al. Implementing an empowerment-based diabetes self-management education program. *Diabetes Educ.* 2005;31:53-61.

Collaborative Wound Care Education Program Launched By Canadian Association of Wound Care and Bayshore Home Health

The Canadian Association of Wound Care and Bayshore Home Health are pleased to announce a unique and exciting educational initiative to provide Bayshore nurses across the country with advanced wound care education and training. The initiative will ensure that Bayshore clients receive optimal wound care management, which is crucial to reducing both healthcare and economic burdens.

Earlier this year, 8 facilitators from Bayshore's nursing staff were trained by the CAWC to conduct the CAWC Institute of Wound Management and Prevention L-Series program. The CAWC L-Series offers intensive, hands-on education regarding the prevention and management of acute and chronic wounds.

The facilitators, with representation from across Canada, were carefully chosen based on their experience, expertise and leadership skills. In early 2012, the facilitators will attend the CAWC L-1 to L-3 Series meetings as regional wound care leaders, and will provide their expertise and hands-on knowledge. They will then be fully prepared to roll out the L-Series program to Bayshore's 3,500 nurses across the country.

To ensure quality control as the L-Series launches with Bayshore staff, a CAWC Institute faculty member will attend each facilitator's initial presentation. "That's part of our commitment to ensuring that the program is successful," says Peggy Ahearn, the CAWC's Executive Director.

"Thanks to the CAWC, we are now equipped to provide point in time education to Bayshore nurses, which is critical," says Katherine Grant-Brown, Clinical Practice Leader, Skin Health and Wound Care, Bayshore Home Health. "Our goal is that each and every Bayshore nurse will undergo this training."

Peggy Ahearn: "The partnership between the CAWC and Bayshore represents a substantial commitment to wound care prevention and management initiatives."

"This is a sizeable commitment on the part of Bayshore," says Peggy. "Recognizing that wound care is a substantial part of the day-to-day work of these nurses is an important step in the prevention and management of chronic wounds."

CAWC Welcomes RQSP

The CAWC congratulates the Regroupement québécois en soins de plaies (RQSP) on their first scientific day, held March 31 in Montreal, Quebec. "We look forward to working in partnership with the RQSP," says Peggy Ahearn, Executive Director, CAWC. "The RQSP meets the very unique needs of Quebec wound care providers. I know our colleagues at the Canadian Association for Enterostomal Therapy join us in wishing much success to this new organization."

L'ACSP souhaite la bienvenue au RQSP

L'ACSP félicite le Regroupement québécois en soins de plaies (RQSP) de la tenue de sa première journée scientifique à Montréal le 31 mars. « Nous nous réjouissons de notre partenariat avec le RQSP », a déclaré madame Peggy Ahearn, directrice administrative de l'ACSP. « Le RQSP répond aux besoins très particuliers des fournisseurs de soins des plaies du Québec. Je sais que nos collègues de l'Association canadienne des stomothérapeutes se joignent à nous pour souhaiter beaucoup de succès à cette nouvelle organisation. »

2012 CAWC Institute Educational Events

The CAWC Institute of Wound Management and Prevention is offering Levels 1 to 3 Series educational sessions at the following locations. Register now!

- Thunder Bay, ON: March 29–April 1
- Montreal, QC: May 3–6 (delivered in French)
- Toronto, ON: June 21–24
- Kelowna, BC: October 11–14
- Toronto, ON: November 29–December 2

The CAWC Institute will also administer the Level 4 International Interprofessional Wound Care Course at the following location:

Michener Institute, Toronto, ON: September 20–23, 2012.

For course descriptions and registration information, please visit: http://cawc.net/index.php/educational/.

Looking at *p* Values: Did the Study Results Happen by Chance Alone?

By
M. Gail Woodbury
PhD BScPT
and
Janet Kuhnke
BSN MS ET

INTRODUCTION

This article outlines the role of *p* values in research. It presents a streamlined discussion of a complex topic, with the aim of supporting wound care practitioners in reading research articles effectively. We recommend two easy-to-read books, *Medical Stats Made Easy*[1] and *PDQ Statistics*,[2] to help you increase your ability to read and use research effectively. A relevant research text might also be of use.

Wound care practitioners and specialists read research articles – the background, methods used, sample size, analyses, results, discussion and interpretation – looking for implications for practice. Scientific studies in the literature often contain a statement such as the following, quoted from the Canadian leg-ulcer care community study, referring to before and after implementation of an evidence-based service: "The proportion of daily visits…dropped from 38% to 6% (Pearson χ^2 test 60.1, $p<0.001$)."[3] Even if you know that the *p* value is **significant**, what does that actually mean? If the *p* value had not been significant (i.e. greater than 0.05), what would that mean?

The *p* value refers to **probability**. Probability ranges from 0 to 1.00. By convention the *p* value is often set at 0.05, which means that a result or statistic as large as that found in the sample would have occurred by chance alone five times out of 100.[4]

Samples and hypotheses

Whenever a research study is done, a sample is used to represent the target population the researcher wants to understand; it is rarely possible to test the whole population. The sample provides an **estimate**, such as a mean or proportion, of the true value of the mean or proportion for the target population.

The investigator starts a study by stating the **null hypothesis**, or H_0, as well as the alternative or **research hypothesis**, H_A or H_1. Let us consider the example of comparing two products or therapeutic approaches, A and B, and determining a mean (i.e. average) value for each. The null hypothesis (H_0) states there is no difference between the means of the products, i.e.:

$A = B$

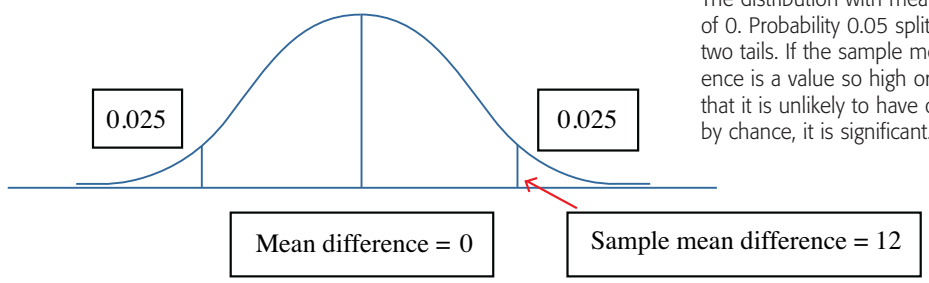
Or $mean_A = mean_B$

The alternative hypothesis (H_A) states there is a difference, i.e.:

$A \neq B$

Or $mean_A \neq mean_B$

FIGURE 1



The distribution with mean difference of 0. Probability 0.05 split between two tails. If the sample mean difference is a value so high or so low that it is unlikely to have occurred by chance, it is significant.

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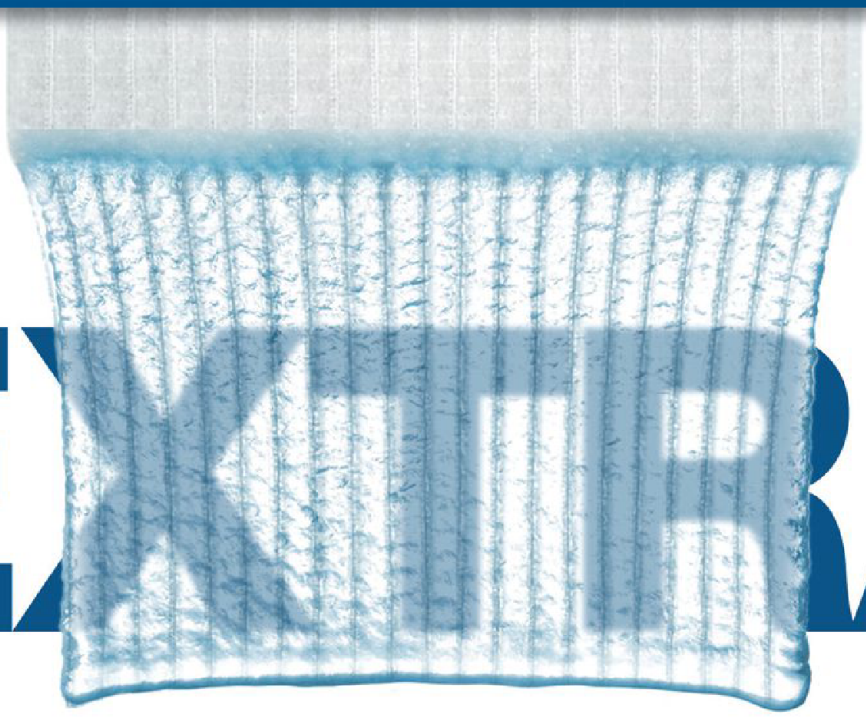

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[a]As compared to original AQUACEL® dressing.

Reference: 1. Preliminary assessment of the physical properties of AQUACEL® EXTRA vs AQUACEL® & DURAFIBER™. *Scientific Background Report*. WHRI3461 TA214. 2011, Data on File, ConvaTec Inc.

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After the study data have been collected and the mean values calculated, the means are compared by hypothesis testing (in this instance, the hypothesis test would be a Student's t-test to compare 2 mean values). Based on the *p* value associated with the hypothesis test, a decision has to be made to either reject the null hypothesis or not reject the null hypothesis. (Statisticians do not talk about accepting the null hypothesis, only **not rejecting** it, because a non-significant hypothesis test does not provide proof of the validity of the null hypothesis.)

Error and significance

When the sample data indicate a big difference such that the *p* value is small (i.e. a value less than 0.05) then the null hypothesis is rejected and there is a risk of type I error. The extent of risk of **type I error** depends upon the significance level of the test, which is set by convention at 0.05. The **significance level** is also called the alpha level and represented by α. When the data indicate there is no difference between groups and the null hypothesis is not rejected, then there is a risk of **type II error**, the amount of which is set by the beta level (represented by β). The statistical power of the hypothesis test, denoted $1 - \beta$, is the probability of finding a significant difference when there is a true difference in the population. These concepts are illustrated in Table 1, which is found in many statistical textbooks.[5–7]

TABLE 1

Type I and type II error

Decision based on sample data and hypothesis test	Reality	
	H_0 is true	H_0 is not true
Not statistically significant (do not reject H_0)	Correct conclusion $1 - \alpha$	Type II error β
Statistically significant (reject H_0)	Type I error α	Correct conclusion $1 - \beta$ (power)

Level of significance indicates the level of risk the investigator is prepared to take in falsely finding significance (i.e. type I error). The researcher must consider how serious the consequences would be if a difference were found by chance that was not true in the population. When the alpha level is set at 0.05, as it often is, the researcher is willing to accept that 5 times out of 100, significance will be found by chance alone. When the alpha level is set at 0.1, the researcher is prepared to accept that risk 10 times out of 100 (i.e. 10% of the time).

Type I and II error

- Type I error refers to the error or risk of rejecting a true null hypothesis, i.e. finding significance where there is not a true difference.
- Type II error refers to the error or risk of not rejecting a false null hypothesis, i.e. not finding significance where there is a true difference.

Let us consider the distribution in Figure 1, with the difference between the means being zero. Think of it as the distribution of all possible samples of the same size from the target population. Our study with the same sample size would be just one example. Let us pretend the data indicate a mean difference of 12 and the hypothesis test has a probability of p=0.015. This sample mean would be unlikely under the null hypothesis and would fall at the extreme end of the distribution, as shown. This result is unlikely to have occurred by chance, so we conclude that we can reject the null hypothesis and say the result is significant.

Conclusion

In summary, *p* values indicate the probability or risk of error – the likelihood that the difference occurred by chance alone. Therefore, when we read the results of a study and they are significant, we know there is still a risk that the result occurred by chance. This illustrates the importance of interpreting evidence from a body of literature as opposed to from just a single study. It also highlights the importance of systematic reviews with meta-analysis for making important clinical decisions.

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7. Elzey F. *An Introduction to Statistical Methods in the Behavioral Sciences*. Monterey, CA: Brooks/Cole Publishing Company; 1976.

Interprétation des valeurs *p* : les résultats de l'étude sont-ils uniquement le fruit du hasard?

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INTRODUCTION

Le présent article porte sur le rôle des valeurs *p* dans la recherche. Il explique un sujet complexe en termes simples pour aider les praticiens du soin des plaies à bien comprendre les comptes rendus de recherches. Les auteurs recommandent au lecteur deux livres faciles à lire, intitulés *Medical Stats Made Easy*[1] et *PDQ Statistics*[2], qui lui permettront de mieux comprendre les comptes rendus et d'utiliser plus efficacement les renseignements qu'ils contiennent. Un livre pertinent sur la recherche pourrait aussi être utile.

Les praticiens et spécialistes du soin des plaies lisent les comptes rendus de recherches – contexte, méthodes employées, taille des échantillons, analyses, résultats, discussion et interprétation – pour comprendre comment ils modifient la pratique. Les études scientifiques publiées contiennent souvent un énoncé comme le suivant, tiré de l'étude canadienne menée en milieu communautaire sur le soin des ulcères de jambe et portant sur la différence entre avant et après la mise en place d'un service fondé sur des données probantes : « La proportion des visites quotidiennes [...] est passée de 38 % à 6 % (test du chi carré de Pearson : 60,1; $p < 0{,}001$). »[3] On sait que la valeur *p* est **significative**, mais qu'est-ce que ça veut dire exactement? Et si la valeur *p* n'avait pas été significative (soit supérieure à 0,05)?

La valeur p désigne la **probabilité**. La probabilité va de 0 à 1,00. Par convention, la valeur p est souvent fixée à 0,05, ce qui veut dire qu'un résultat ou une statistique aussi important que celui retrouvé dans l'échantillon aurait pu être uniquement le fruit du hasard cinq fois sur 100[4].

Échantillons et hypothèses

Les études de recherche portent toujours sur des échantillons qui représentent la population cible que les chercheurs désirent comprendre, car il est rarement possible d'évaluer l'ensemble de la population. L'échantillon donne une **estimation**, par exemple une moyenne ou une proportion, de la valeur réelle de la moyenne ou de la proportion dans la population cible.

Au début d'une étude, le chercheur formule **l'hypothèse nulle**, soit H_0, et **l'hypothèse de recherche** alternative, soit H_A ou H_1. Prenons l'exemple de la comparaison entre deux médicaments ou démarches thérapeutiques, A et B, et de la détermination d'une valeur moyenne pour chacune. Selon l'hypothèse nulle (H_0), il n'y a pas de différence entre les moyennes des produits :

$A = B$

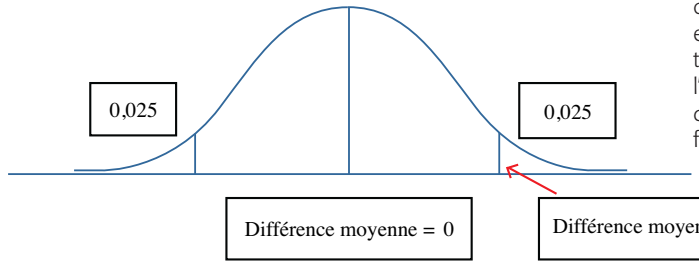
ou $\text{moyenne}_A = \text{moyenne}_B$

Selon l'hypothèse alternative (H_A), il y a une différence :

$A \neq B$

ou $\text{moyenne}_A \neq \text{moyenne}_B$

FIGURE 1



Distribution avec différence moyenne de 0. Probabilité de 0,05 partagée entre les deux queues de la distribution. Si la différence moyenne dans l'échantillon est si grande ou si petite qu'elle est peu susceptible d'être le fruit du hasard, elle est significative.

Une fois les données recueillies et les valeurs moyennes calculées, on compare les moyennes au moyen d'un test d'hypothèse (dans le cas qui nous intéresse, le test d'hypothèse serait un test de Student pour comparer deux valeurs moyennes). Selon la valeur *p* associée au test d'hypothèse, on décide de rejeter ou de ne pas rejeter l'hypothèse nulle. (Les statisticiens ne parlent pas d'acceptation, mais plutôt de non-rejet de l'hypothèse nulle, parce qu'un test d'hypothèse non significatif ne prouve pas la validité de l'hypothèse nulle.)

Erreur et signification

Quand les données d'échantillon indiquent qu'il y a une grande différence, donc que la valeur *p* est faible (c'est-à-dire inférieure à 0,05), l'hypothèse nulle est rejetée et il y a un risque **d'erreur de type I**. L'importance du risque d'erreur de type I dépend du **seuil de signification** du test, qui est fixé par convention à 0,05. Le seuil de signification est aussi appelé seuil alpha (représenté par α). Quand les données indiquent qu'il n'y a pas de différence entre les groupes et que l'hypothèse nulle n'est pas rejetée, il y a un **risque d'erreur de type II**, dont l'importance dépend du seuil bêta (représenté par β). La puissance statistique du test d'hypothèse (soit 1 - β) est la probabilité d'observer une différence significative quand il y a une différence réelle dans la population. Ces concepts sont illustrés au tableau 1, qu'on retrouve dans de nombreux manuels de statistique[4–7].

Le seuil de signification indique l'importance du risque que la signification observée soit fausse (soit une erreur de type I) que le chercheur est prêt à accepter. Le chercheur doit se demander si les conséquences seraient graves si une différence observée était le fruit du hasard, et donc n'était pas réelle. Un seuil alpha fixé à 0,05, ce qui est souvent le cas, indique qu'un chercheur est prêt à accepter que cinq fois sur 100, la signification observée soit uniquement le fruit du hasard. Un seuil alpha de 0,1 indique que le chercheur est prêt à accepter que la signification soit uniquement le fruit du hasard dix fois sur 100 (soit dans 10 % des cas).

Examinons la distribution présentée dans la figure 1, selon laquelle la différence entre les moyennes est de zéro. Cette distribution est comme celle de tous les échantillons possibles de la même taille dans la population cible. Notre étude, qui porte sur un échantillon de la même taille, ne serait qu'un exemple. Supposons que les données indiquent que la différence est de 12 et que le test d'hypothèse donne une probabilité (valeur *p*) de 0,015. Cette moyenne d'échantillon est peu probable selon l'hypothèse nulle et se situe à la limite extrême de la distribution, comme le montre la figure. Comme un tel résultat est peu susceptible d'être le fruit du hasard, on peut conclure qu'il convient de rejeter l'hypothèse nulle et que le résultat est significatif.

TABLEAU 1

Erreur de type I et erreur de type II

Décision fondée sur les données d'échantillon et le test d'hypothèse	Réalité	
	H_0 est vraie	H_0 n'est pas vraie
Pas de différence statistiquement significative (non-rejet de H_0)	Bonne conclusion 1 - α	Erreur de type II β
Différence statistiquement significative (rejet de H_0)	Erreur de type I α	Bonne conclusion 1 - β (puissance)

Erreur de type I et erreur de type II

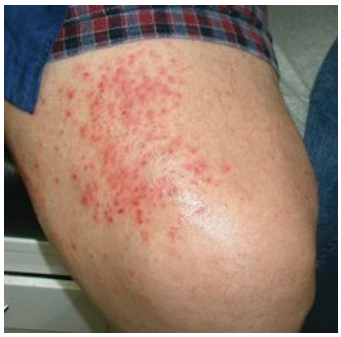
- L'erreur de type I désigne le risque de rejeter une hypothèse nulle vraie, c'est-à-dire d'observer une différence significative quand il n'y en a pas en réalité.
- L'erreur de type II désigne le risque de ne pas rejeter une hypothèse nulle fausse, c'est-à-dire de ne pas observer de différence significative quand il y en a une en réalité.

Conclusion

En résumé, la valeur *p* indique la probabilité ou le risque d'erreur, soit la probabilité que la différence soit uniquement le fruit du hasard. Par conséquent, quand on lit dans un compte rendu que les résultats d'une étude sont significatifs, on sait qu'il est quand même possible que ces résultats soient le fruit du hasard. C'est pourquoi il est important d'examiner les données probantes d'un ensemble d'études, et pas seulement d'une seule. C'est aussi pourquoi les importantes décisions cliniques doivent être fondées sur des examens méthodiques et des méta-analyses.

Références (voir page 8)

What is the Cause of this Unusual Rash?



By Rob Miller
MD FRCPC
and
Cathy Burrows
BScN MScN

Introduction

This 55-year-old man had a below-knee amputation due to peripheral vascular disease. One year post-amputation he developed an asymptomatic red scaly rash at the amputation site. He was concerned that the rash was spreading up his leg.

Rob Miller and **Cathy Burrows** are with the Wound Care Clinic, QEII Halifax Infirmary Site, Halifax, Nova Scotia.

What is the cause of this rash?

This patient has developed a fungal infection (also known as ringworm or tinea). When it occurs on the feet, it is known as athlete's foot.

How would you confirm the diagnosis?

In this case, fungal scrapings were taken with a small #15 scalpel blade and sent to the mycology laboratory. The scrapings were initially examined with a potassium hydroxide preparation under the microscope. Some of the scrapings were also cultured, and grew *Trichophyton* rubrum (a common cause of athlete's foot).

What is the treatment?

Topical therapy with an antifungal cream (e.g. clotrimazole) applied twice daily for 6 weeks will usually clear the rash. However, recurrence is common. For patients who do not respond to topical agents, oral therapy with terbinafine for 3–4 weeks is recommended.

Moist Wound Healing: Past and Present

A historical perspective on the importance of moist wound healing

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Abstract

Local wound care involves the key priorities of debridement, control of infection and prolonged inflammation, and moisture balance. Mindful of a holistic approach, these priorities must not be addressed in isolation, but rather as an evolving continuum dependent on the phase of wound healing, the etiology of the wound and the influence of intrinsic and extrinsic factors affecting healing. A moist wound environment is one of the many requirements for wound healing, and must be consistently assessed and supported in clinical practice to ensure that wounds are not subject to unnecessary and potentially detrimental effects from a lack or excess of moisture. The clinician must be armed with knowledge of the important effects of moisture at the cellular level throughout the phases of wound healing. This article traces the influence of moist wound healing from the work of George Winter and includes a historical overview of animal and human models used in this area. Applications to modern-day practice are presented in a review of occlusive and semi-occlusive dressings, and their ability to enhance or modify the wound environment by influencing oxygen and moisture is discussed.

Introduction

Local wound care involves the key priorities of debridement, control of infection and prolonged inflammation, and moisture balance.[1-3] The holistic approach suggests that these priorities must not be addressed in isolation, but rather as an evolving continuum dependent on the phase of wound healing, the etiology of the wound, and the influence of intrinsic and extrinsic factors affecting wound healing. A fairly recent framework that has influenced wound care professionals is the model of wound bed preparation (WBP).[4,5] It is critical to create a foundation in which healing can occur[6] and WBP supports this by providing a structured approach to wound management.[7] WBP is defined as wound management that accelerates healing or facilitates the effectiveness of other therapeutic measures.[4,5,8] The TIME mnemonic is an easy-to-remember framework for local wound care within the WBP model (Table 1).[9] WBP involves a holistic wound assessment, consisting of examining the underlying etiology and the patient's psychosocial needs.[5] Within this framework, moist wound healing (MWH) is a central pillar.

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An operational definition of MWH is reviewed by Bolton, and is related to the processes (e.g. dressings) that keep a wound moist.[10] Dressings are discussed later in this article. It is clear that MWH is regarded as the ideal environment for optimal healing.[11] A clear definition of MWH is not well established because of the inability to quantify optimal moisture levels. In the absence of MWH, the dermis dehydrates. This supports a scab formation that creates a barrier to epithelial migration, as the cells must move deep under the scab to migrate to a viable wound surface.[12] Wound healing is achieved when epidermal cells migrate and mature across wound margins to achieve epithelialization.[13]

Early research

An enlightening history of early wound treatment from the prehistoric era through to the Middle Ages has been described by Forrest.[14] A central theme throughout history and today is the management of moisture. Galen of Pergamum (120–201 AD) was a dominant Greek figure who treated the wounds of Roman gladiators. He provided a moist environment, described as a cotton

cloth and sponge, to optimize healing.[15] MWH was not scientifically studied until the mid-20th century. Gilge reported treating venous ulcers with adhesive tape and demonstrated quicker healing with occlusion.[16] Bloom incorporated semi-permeable cellophane dressings to cover human burn wounds suffered in war to prevent protein losses and infection.[17] The cellophane was covered with cotton wool and gauze, and later removed to demonstrate a steaming effect of water transuding through the dressing in severe burns. Bloom reported that the time between the application of the cellophane and complete healing was 9 days, with patient reports of pain disappearing after application. The report by Bloom was not a randomized controlled trial, but did include a relatively large sample of 55 cases.

Bull and colleagues experimented with semi-occlusive dressings on humans and were able to demonstrate fewer bacteria collected under occluded skin.[18] In 1950, Schilling et al. found a statistically significant difference in the healing rates ($p<0.0001$) of full-thickness lacerations, abrasions and puncture wounds with a nylon dressing permeable to water vapour vs. with waterproof dressings.[19]

Research and pigs

Current awareness of the concept of MWH has been mostly influenced by the research of Winter, who used an experimental design to examine acute superficial wounds in pedigree pigs.[3] Winter used pigs to compare 1 acute wound covered with a polymer film dressing, protecting the wound from dehydration, with another exposed to air. The results revealed that prevention of scab formation from the film dressing significantly increased the rate of epithelialization compared with the scab in the control, which delayed epithelialization ($p<0.0001$).

Buchan et al. suggested there are limitations in comparing humans with pigs because pigs are poorly vascularized and possess apocrine glands, which humans only have in the axillae and groin.[20] In addition, eccrine glands are present in humans but not in pigs. More recently, Sullivan et al. described similarities in the anatomy and physiology of pigs and humans, including a comparable dermal–epidermal thickness ratio of 10:1 in humans and 13:1 in pigs.[21] Winter used 12- to 14-week-old pigs, which typically have a dermis thickness of 2 mm when 15 weeks old.[22] This minimized a variety of intrinsic and extrinsic factors affecting wound healing. Acute animal models are not typical of the chronic wounds seen in practice.[23]

Human research

The effect of air exposure and occlusion on experimental human skin wounds was studied by Hinman and Maibach[24] in an extension of Winter's work. This experiment was conducted on an undisclosed sample size of healthy adult male volunteers serving as their own controls. The authors reported little to no demographic information for this convenience sample, limiting the generalizability to the wider population. However, the results supported the suggestion that re-epithelialization of acute wounds occurred at a faster rate with occlusion than in those left open to the air – although topical neomycin was used, which may have been a confounding variable in the healing outcome.

Intact functional skin contains low transepidermal water loss to maintain a hydrated or moisturized surface barrier.[25] Removal of the stratum corneum increases transepidermal water loss, contributing to cell death and desiccation.[26]

TABLE 1

The TIME mnemonic: key points on which to focus in wound bed preparation

T	Tissue (healthy or unhealthy)
I	Inflammation or infection (presence or absence)
M	Moisture balance
E	Edge (non-advancing or non-migrating)

Inflammatory phase

MWH enhances the migration of polymorphonuclear neutrophils (leukocytes), enabling bacteria and debris to be destroyed by phagocytosis. Migration of PMNs accelerates healing through autolytic debridement, defined as assisting the separation of dead or damaged tissue.[4,27] Saymen et al. experimented with adult female rats and established that grafting wounds aided in PMN infiltration compared with ungrafted wounds, where infiltration was limited in the desiccated wound.[28] MWH management of split-thickness skin grafts has demonstrated advantages in healing, pain and infection rates.[29] The benefits of a moist occlusive environment were highlighted by Buchan et al. as wound exudate from pigs and humans recovered under occlusion identified the activity of bactericidal neutrophils.[20] This work by Buchan et al. was an extension of the influential work by Winter.[3] In occluded wounds in 6 female pigs, Buchan et al. demonstrated increased levels of globulins and lysozyme under occlusion, enhancing the killing mechanism of the PMNs.[20]

Later research by Dyson et al., in another example of replication of Winter's work, examined the effects of moisture in acute pig wounds covered with a semi-permeable film compared with dry wounds covered in gauze.[30] The wounds were randomly selected with a sample size of 7. Dyson et al. demonstrated a timely

progression from the inflammatory phase to the proliferative phase of wound healing in moist wounds with quantitative histological results. This was supported by a decrease in neutrophil count and increase in fibroblast count in the moist wounds by day 7, compared with a higher number of neutrophils and lower number of fibroblasts in the dry wounds. Ten days after injury, 60% more fibroblasts ($p<0.0001$) were identified in the moist wounds compared with the dry wounds. Fibroblasts were assessed to be aligned, suggesting progressive differentiation into the myofibroblasts contributing to wound contraction. The presence of proteinase activity in the occluded moist surgical excisional wounds of pigs enhanced eschar removal and prepared the wound for re-epithelialization.[30]

Growth factors

Moisture in the wound aids in the transport of growth factors, which is essential for autolysis or the breakdown of necrotic tissue in preparation for healing.[31] Cytokines and growth factors are the signalling proteins that affect the normal wound healing process.[32] Macrophages eliminate debris through phagocytosis, generate chemotactic factors, and synthesize and release regulatory and growth factors essential for repair.[33] Occlusion with hydrocolloids in acute wound models of young pigs was hypothesized by Chen et al. to promote an environment where growth factors could accumulate to facilitate wound healing.[34] Platelet-derived growth factor-like activities in wound fluid from the full-thickness wounds also promoted the growth of cultured fibroblasts. Although the study by Chen et al. involved daily dressing changes for only 4 post-operative days, wound fluid was suggested to contain basic fibroblast growth factor-like factors that contribute to granulation formation. The benefits of occlusion were suggested by Chen et al. to include the retention of functional growth factors modifying the environment to facilitate healing.[34]

Proliferation and remodelling

Dyson et al. examined the effects of dermal repair and angiogenesis, comparing moist conditions in full-thickness excised lesions in pigs achieved by polyurethane film dressings with dry conditions achieved by air exposure through the use of gauze dressings.[35] Angiogenesis involves the formation of new capillaries, enabling the restoration of nutrients and oxygen delivery to the wound.[36] Using computerized image analysis to measure the number of blood vessels, angiogenesis was observed to be increased in the early stage of healing in moist wounds at 3 days. Vessel formation decreased after 7 days, suggesting a timely transition to the remodelling phase, as blood vessel sprouting begins at this time.[36]

Kunugiza et al. examined the effect of MWH using rat excisional wound models, comparing hydrocellular foam dressing and gauze.[37] It is questionable as to why gauze was chosen; Rogers et al. demonstrated in vivo that granulation tissue grows into the structure of the gauze dressing, contributing to adherence and tissue trauma during removal.[38] The use of gauze does not facilitate a constant moist environment and dries out.[39] Foam dressings, on the other hand, absorb exudate and provide an MWH environment. The results from the study by Kunugiza et al. demonstrated, with wound tracing, no reduction in the area of the gauze-treated wounds on day 3 versus a 21% reduction in the area of the foam-dressed wounds.[37] In addition, there was a statistically significant increase in new blood vessels in the foam group compared with controls on day 6 ($p<0.05$).

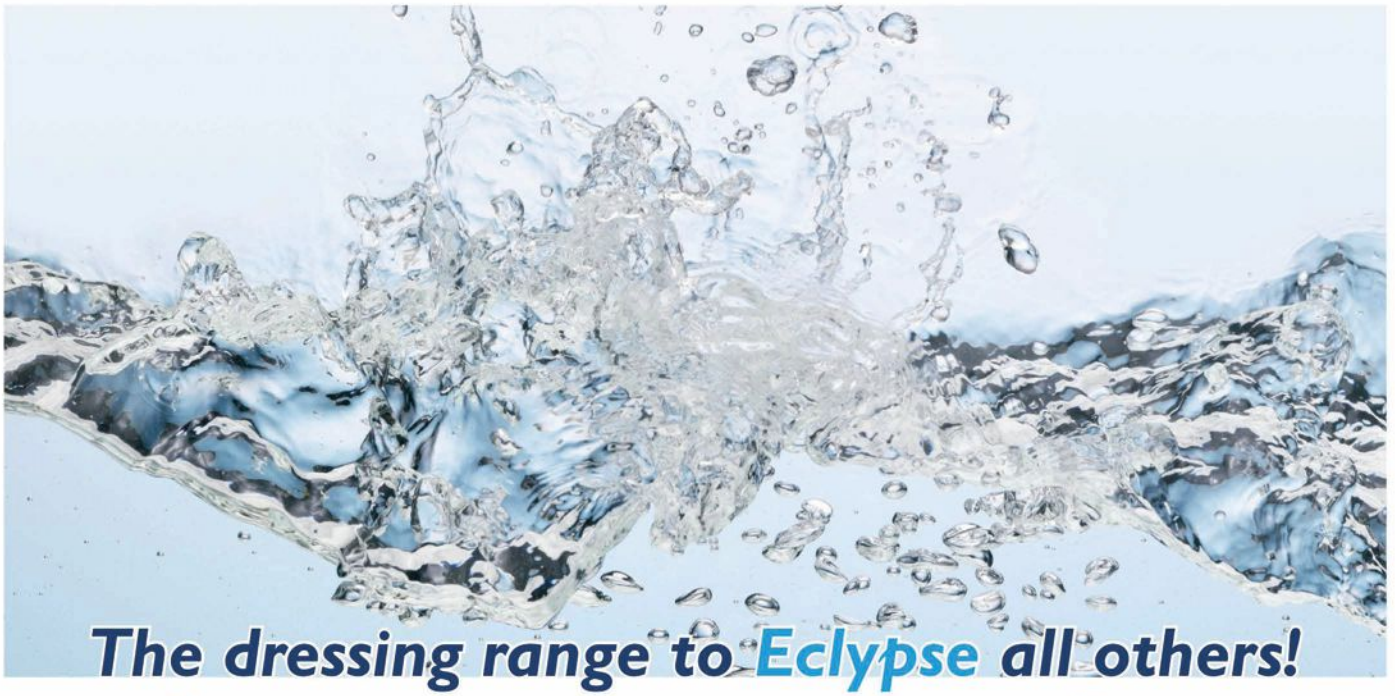
The remodelling phase of wound healing extends from 3 weeks to months or even years, depending on the influence of intrinsic and extrinsic factors affecting healing.[40] It is important to remember that remodelling is occurring, even after the wound is re-epithelialized.

Occlusion and bacteria

Resistance by clinicians to the use of occlusive dressings was based on concerns that bacteria would proliferate in a moist environment.[24,41] Hutchinson and McGuckin reviewed controlled studies investigating the incidence of infection under occlusion, and found that the infection rate was 7.6% with non-occlusive dressings versus 3.2% with occlusive dressings ($p<0.001$).[42] Although wound infections occurred with both treatments, the risk for infection was lower in the occlusive group by preventing invasion of bacteria and fostering a favourable environment for neutrophil activity, enhancing the host's defence. A subsequent review by Hutchinson and Lawrence provided evidence from 50 controlled trials, 48 of them using hydrocolloid dressings. Again, lower rates of infection were reported with occlusive dressings compared with conventional dressings (3.3% versus 5.4%, respectively, $p<0.001$).[43] However, controversy remains regarding the measurement of infection in chronic wounds, as this can be determined by laboratory parameters, microbiology, clinical signs or combinations of each.[44]

Application to clinical practice

There is a gap in the literature regarding the optimal quantity of wound fluid or moisture required for healing.[31,45] Bishop et al. reinforced the importance of moisture balance at the interface between the wound



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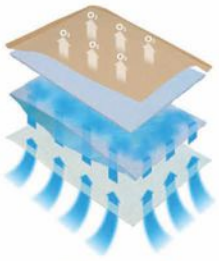

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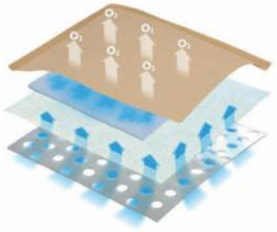

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and the dressing, although the "not too wet and not too dry" approach may be practical but lacks rigour.[46] Excessive exudate can cause malodour and leakage, which can contribute to psychological distress, depression and social isolation.[47,48] The management of excess exudate during infection or in chronic wounds is of equal importance to conserving water in the healing wound.[49] Clinicians must appreciate that there are varied amounts of exudate through the phases of wound healing, with higher levels during the inflammatory and proliferative phases,[50] and variations in the amounts between different wound types and locations.

Important practice point

Sufficient arterial inflow is required to support MWH and occlusion, especially in arterial ulcers. Where there is sufficient arterial circulation to support wound healing, then a moist environment should be maintained. Eschar should be kept dry until the underlying circulation is appropriately assessed or revascularization occurs.[51]

Using MWH as a principle for successful wound healing, when appropriate, provides a framework to guide management of tissue, infection, moisture balance and the wound edge.[4] In the 1990s, the focus of moisture management was geared toward managing excess; from 2000, this focus shifted to moisture balance. Early research supports the modern-day clinical WBP paradigm[52] that links treatment to the underlying cause, patient-centred concerns and local wound care involving debridement, control of infection and prolonged inflammation, and moisture balance.

MWH has influenced a shift in focus to patient-centred concerns by addressing pain control. Advances in MWH dressings have contributed to minimizing pain by decreasing adherence of the dressing to the wound and soothing nerve endings.[39,53] Pain at the wound site can be minimized with moisture-retentive dressings.[54] Consensus documents provide the principles of best practice to minimize pain during wound-dressing-related procedures. Managing parameters such as allergy potential, absorbency capacity and the ability to be traumatic to the wound and periwound, as well as the ability of a dressing to maintain a moist environment, can aid in pain management.[55] Modern dressings that manage large amounts of exudate effectively can also decrease the number of dressing changes required, theoretically improving the health-related quality of life for individuals.[56]

Modern wound dressings not only support wound healing clinically, but are also economically sound.[56,57] Absorbent dressings have evolved, allowing excess moisture to evaporate from the surface of the dressing through varied moisture vapour transfer rates.[58] To be considered moisture retentive, a dressing must have a moisture vapour transfer rate of less than 840 g/m^2/24 hours.[59] Semi-occlusive polyurethane film dressings are permeable to moisture vapour, oxygen and other gases, and provide a microbial barrier.[60–62] Occlusive dressings are impermeable to water vapour and oxygen, and trap the moisture in the wound, preventing trauma, desiccation and transmission of microbes from the environment to the wound.[61–63] Jones eloquently summarized that the quest for the optimal dressing is a perfection that can never be achieved.[26]

Despite the plethora of evidence supporting MWH, its application in modern-day practice continues to vary.[31] Education in wound care is still required in all healthcare disciplines. Physician education in this area is lacking, while registered nurses – with an average of 50–60% of their workload in wound care – consider their wound care education to be insufficient.[64–66] All wound care clinicians should be aware of the impact of MWH, as this is a critical aspect of healing. As practitioners, we should continually assess the prevalence of this aspect in practice to ensure that wounds are not subject to unnecessary and potentially detrimental effects from lack of moisture.

The use of dry dressings has been argued to be based on tradition rather than evidence, and the culture of practice may therefore need to be questioned.[67] The prevalence of dry dressings in wound care is well documented. In a retrospective chart review, Cowan and Stechmiller found that wet-to-dry dressings were ordered 42% of the time; 69% of these wounds were surgical.[68] The science behind wound healing continues to flourish, and so too must the application of MWH to clinical practice.

Conclusion

The evidence provided confirms that the early work of Winter[3] has had a fundamental influence on modern-day clinical wound management. Although Winter's research involved the acute wound, MWH has been extrapolated to include chronic wound management. Modern wound dressings have flourished, aiding the management of moisture to support MWH. The effects of MWH considerably enhance the cellular phases of wound healing, contribute to pain relief and address patient-centred concerns.

When someone says to you, "The wound is OK and it's dry," stop and think. Are we doing all that we can? Are we applying the science to practice to facilitate timely transitions in the phases of wound healing?

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Considering the increase in the prevalence of chronic, non-healing wounds and the characteristics of our patients (who are living longer with comorbid conditions and weakened immune systems), MWH must be assessed and implemented in practice. Several of the articles on early research are truly a fascinating read. Examine the practice around you and apply your knowledge to improve the lives of those with wounds.

Acknowledgement

This paper was adapted from a paper written in 2010 for the MSc Wound Healing and Tissue Repair course through Cardiff University, Wales, UK.

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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).

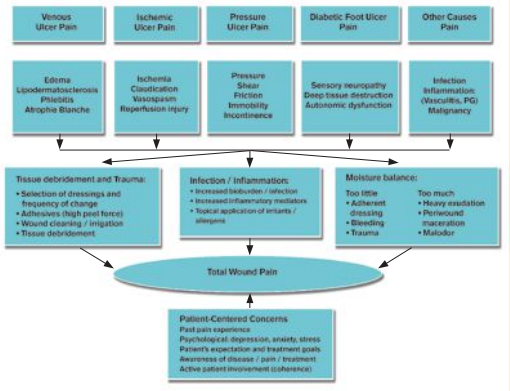
Chronic wounds are disabling and constitute a significant burden on patients' activities of daily living (ADLs) and the healthcare system. Of persons with diabetes, 2–3% develop a foot ulcer annually, while the lifetime risk of a person with diabetes developing a foot ulcer is as high as 25%.[8] It is 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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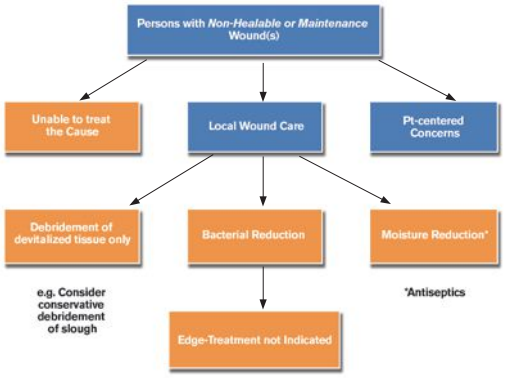
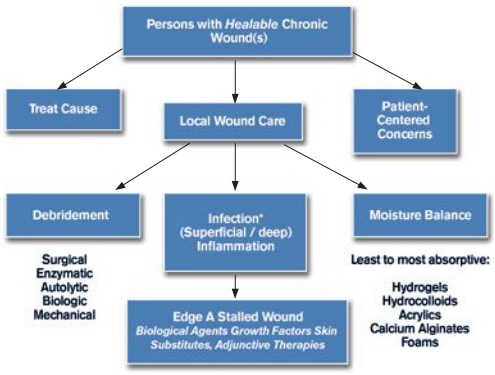
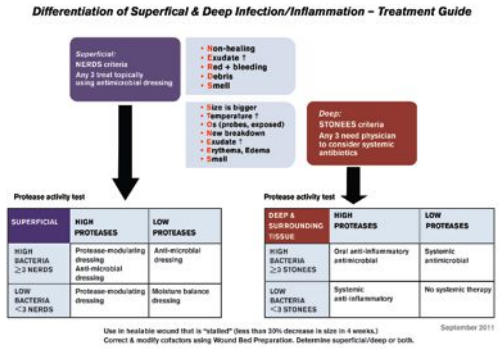
Enabler. Persons with nonhealable chronic wound(s)



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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Lymphedema

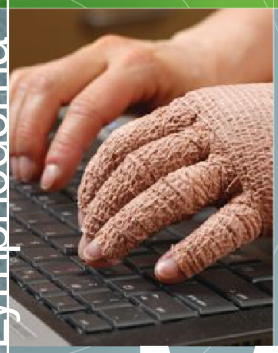

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TABLE 1

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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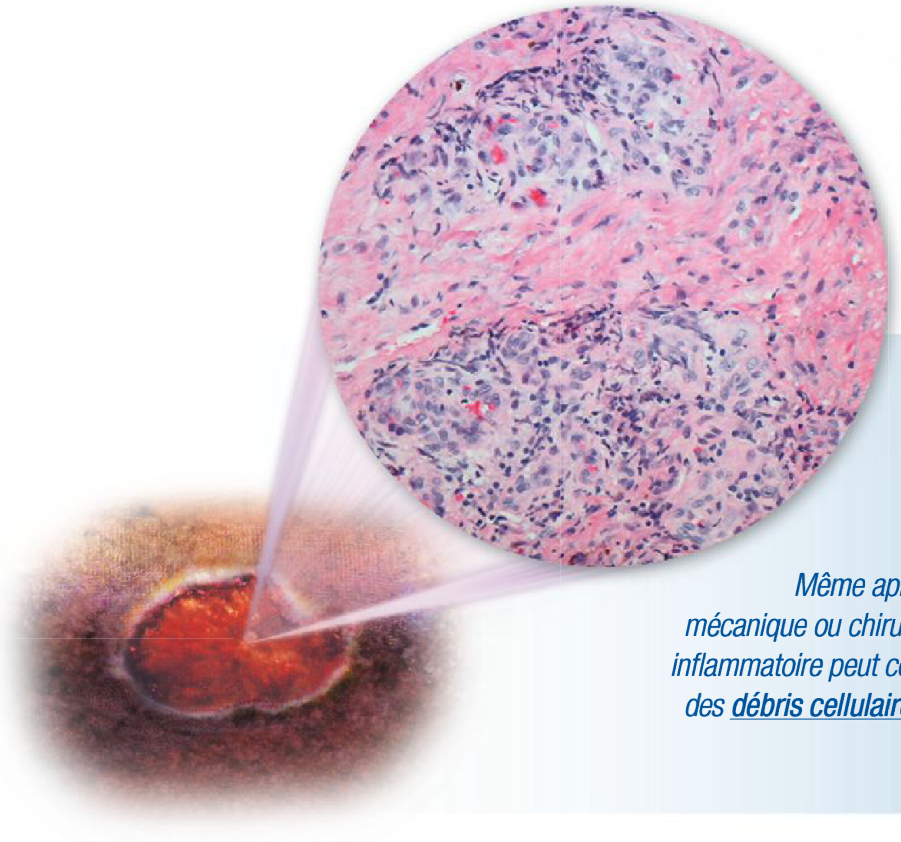

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Occasional slight transient erythema has been noted in surrounding tissue when applied outside the wound. One case of systemic hypersensitivity has been reported after 1 year of treatment with collagenase and cortisone.

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Please see complete Prescribing Information on adjacent page.

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.

L'utilisation de l'onguent SANTYL® avec collagénase devrait être cessée lorsque le débridement est complété et que la granulation est bien entamée.

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DESCRIPTION: Santyl® (collagenase) ointment is a sterile topical enzymatic debriding agent that contains 250 units of collagenase per gram of white petrolatum USP. The enzyme collagenase is derived from the fermentation of *Clostridium histolyticum*. It possesses the unique ability to selectively digest denatured and undenatured collagen that binds necrotic debris to the wound surface.

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.

HOW SUPPLIED: Available in 30 gram tubes of ointment. Sterile until opened. Contains no preservative. Do not store above 25°C.

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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.

PRÉSENTATION : Disponible en tubes de 30 grammes d'onguent. Stérile dans l'emballage non ouvert. Aucun agent de conservation. Ne pas entreposer au-dessus de 25°C.

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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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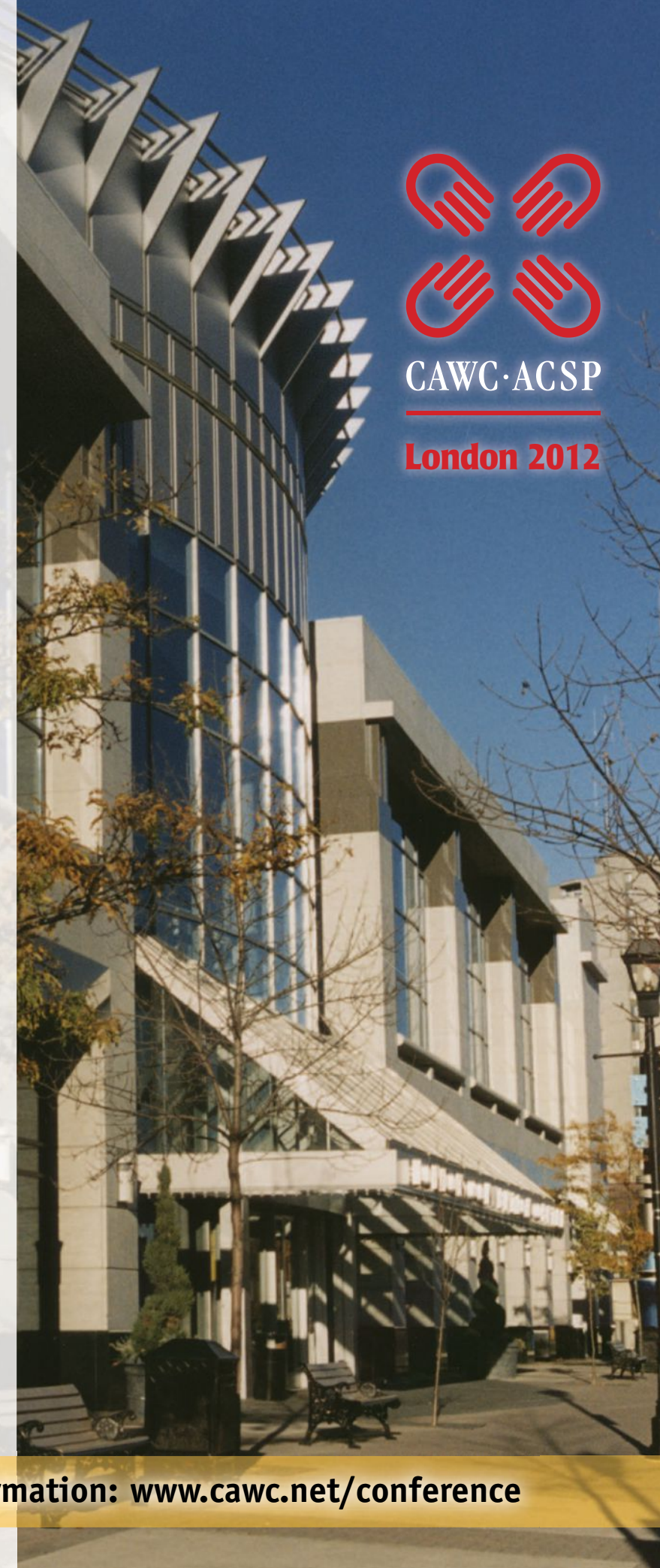

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ated with individual risk factors and comorbidities. In 2009, an 18-member international expert panel explored the issues and research literature surrounding end-of-life skin and wound care, including the Kennedy Terminal Ulcer (case series evidence)[26] 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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Plaie chirurgicale de fasciite nécrosante causée par le pyoderma gangrenosum

Rapport d'un cas

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Résumé

Le pyoderma gangrenosum est une maladie ulcérative rare dont la prise en charge médicale est souvent difficile. Elle est caractérisée par le phénomène de pathergie. Nous présentons le cas d'une femme atteinte de pyoderma gangrenosum présentant une infection nécrosante des tissus mous au niveau de la cuisse et du périnée. Un débridement extensif a été pratiqué et il y a eu une détérioration importante de la plaie malgré la thérapie par pression négative. Il y a également eu de multiples surinfections bactériennes à *Pseudomonas aeruginosa* multirésistant. Nous décrivons la prise en charge multidisciplinaire, à l'aide d'immunomodulateurs, de corticostéroïdes et d'immunoglobulines, de même que de greffes cadavériques et d'autogreffes cutanées. Puisqu'il s'agissait d'un traitement de longue durée, une collaboration entre les équipes soignantes, de même qu'un personnel dévoué et la motivation de la patiente ont été des éléments essentiels.

Introduction

Le pyoderma gangrenosum (PG) est une maladie inflammatoire ulcérative rare qui fait partie des dermatoses neutrophiliques d'étiologie non infectieuse. Il a été signalé pour la première fois par Brusting *et al*. en 1930, qui ont décrit cinq patients présentant des lésions ulcérées avec bordures surélevées et violacées, de même qu'un périmètre érythémateux inflammatoire[1]. Sa physiopathologie est encore inconnue, mais on soupçonne fortement que la maladie est d'origine immunologique, compte tenu de ses liens avec des maladies systémiques (néoplasies, maladies inflammatoires de l'intestin, polyarthrite rhumatoïde, etc.) dans près de 70 % des cas[2, 3]. Le traitement est d'abord et avant tout médical avec en première ligne, pour les cas d'intensité légère (pustules ou papules et petits ulcères superficiels), les immunosuppresseurs topiques, tels le tacrolimus, la cyclosporine et les corticostéroïdes locaux. Les agents systémiques, tels les corticothérapies systémiques et les immunomodulateurs, sont réservés aux cas sévères et réfractaires. Leurs effets secondaires en limitent l'utilisation. Des traitements de rechange, telles les immunoglobulines intraveineuses, sont à l'étude et donnent des résultats prometteurs[4]. Les soins des plaies font également partie intégrale de la prise en charge du PG, y compris par l'utilisation de matrices extra-cellulaires[5]. En cas d'infection, une antibiothérapie appropriée (locale ou systémique) est primordiale. Le PG est caractérisé par le phénomène de pathergie, qui désigne une réaction démesurée aux traumatismes légers qui mène rapidement au développement d'ulcères[1].

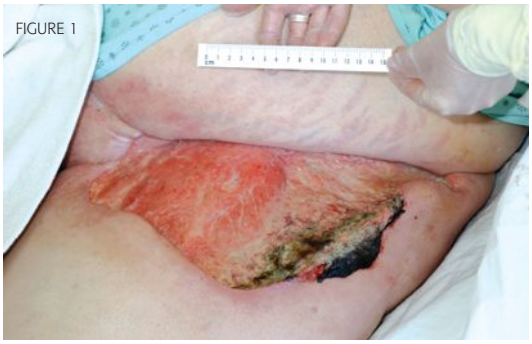


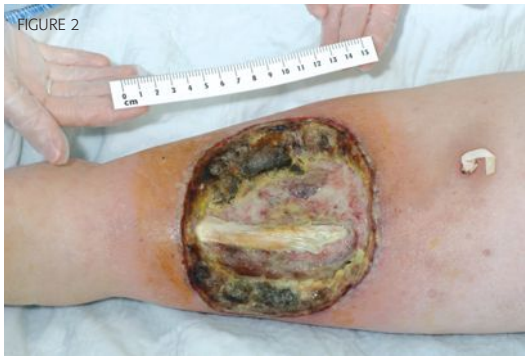
FIGURE 1
Plaie inguinale gauche, juin 2010.

Rapport de cas

La patiente est une femme de 44 ans souffrant de polychondrite (arthrite, uvéite, sclérite, purpura, chondrite et vasculite leucocytoblastique) réfractaire à l'infliximab, à la cyclosporine et au mycophénolate

mofétil. Elle a également présenté des effets secondaires au méthotrexate (pneumonite) et au cyclophosphamide (thrombopénie). De plus, elle présente un syndrome de Cushing iatrogène et une neuropathie périphérique et est traitée pour un épisode de dépression majeure. Elle a été prise en charge dans un centre hospitalier régional pour une plaie inguinale gauche (30 sur 20 sur 2 cm) ayant progressé rapidement à partir d'une folliculite (juin 2009) et contre laquelle des débridements locaux, effectués parce qu'on soupçonnait une fasciite nécrosante, ont été utiles (figure 1). La patiente présente aussi une plaie pré-tibiale gauche (10 sur 10 sur 1 cm) avec exposition tendineuse d'étiologie inconnue et ayant progressé sous thérapie par pression négative et débridements (figure 2). Il y a eu quelques surinfections de ces plaies à *Pseudomonas aeruginosa* multirésistant.

FIGURE 2

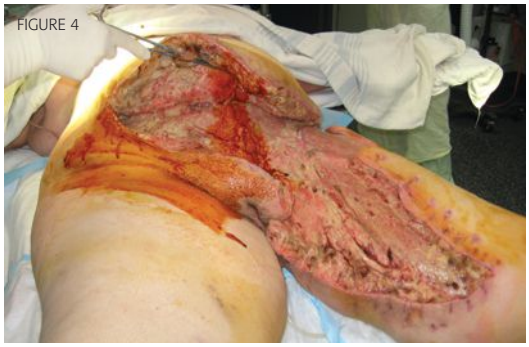


Plaie pré-tibiale gauche, juin 2010.

En juin 2010, le service de dermatologie de notre centre universitaire a poussé l'investigation et pris la patiente en charge. Elle a été hospitalisée pour une approche multidisciplinaire faisant intervenir les services de rhumatologie, de médecine interne et de soins de plaies. On lui a administré une thérapie pulsée à la méthylprednisolone, à la prednisone et au méthotrexate et on a changé ses pansements à l'iode deux fois par jour. Comme son état ne s'améliorait pas, en juillet 2010, on a procédé à un débridement par application d'un onguent enzymatique (Santyl® [collagénase]) et utilisé une matrice extracellulaire d'origine porcine (Oasis®). On a par la suite noté une amélioration de la granulation des plaies. En août 2010, la patiente a présenté une surinfection à *P. aeruginosa* qui a répondu à la tobramycine intraveineuse. L'équipe chirurgicale est intervenue en septembre 2010 en raison d'un abcès périvulvaire gauche accompagné d'une détérioration de l'état général de la patiente et d'une septicémie. La tomographie axiale a confirmé la présence d'un abcès pelvien profond et soulevé un doute quant à l'intégrité du fascia sous-jacent (figure 3).

Devant la détérioration rapide de l'état de la patiente, on l'a conduite en salle d'opération où on a diagnostiqué une infection nécrosante des tissus mous (confirmée par la pathologie) de l'aine gauche, de la paroi abdominale antérieure, du périnée, de même que de la cuisse gauche. Un débridement extensif a été pratiqué

FIGURE 4

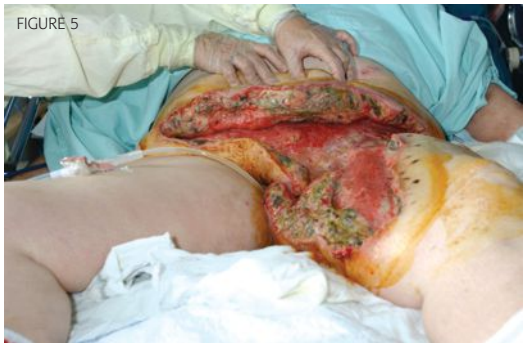


Deux semaines après un débridement chirurgical extensif pour une infection nécrosante des tissus mous, octobre 2010.

(figure 4), laissant une plaie de 53 sur 50 sur 2 cm.

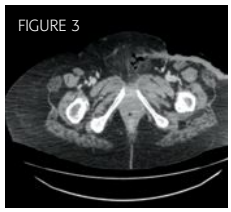
À cause d'importantes douleurs, les pansements ont été changés toutes les 48 heures au bloc opératoire sous anesthésie générale. Sept jours après l'opération, on a décidé d'instaurer une thérapie par pression négative, mais il y a eu une détérioration importante de la plaie et on a abandonné la thérapie cinq jours plus tard. Devant les phénomènes pathergiques, on a repris le traitement par les corticostéroïdes, les immunosuppresseurs (méthotrexate; la patiente n'a pas présenté de pneumonite) et les immunomodulateurs, et des immunoglobulines intraveineuses ont été administrées. Une autre surinfection à *P. aeruginosa* a été traitée par la tobramycine et la colistine (figure 5). Une fois la plaie stabilisée, plusieurs semaines plus tard (6 novembre 2010 et 10 décembre 2010), on a procédé à des greffes cadavériques comme pansements biologiques dans le but de réduire la surface de la plaie (figure 6).

FIGURE 5



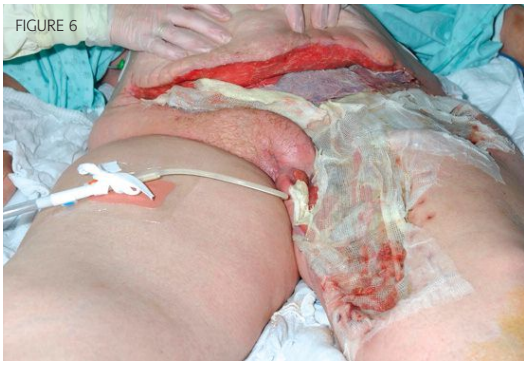
Surinfection de la plaie abdominale à *P. aeruginosa*, octobre 2010.

FIGURE 3



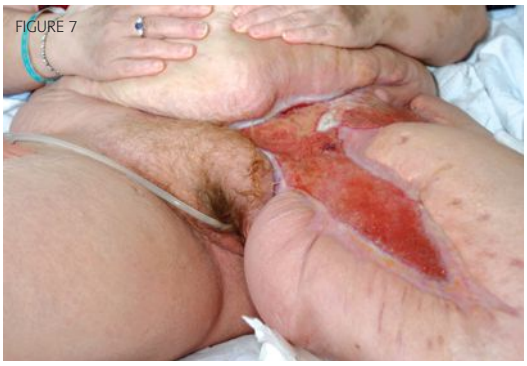
Tomographie axiale démontrant un abcès pelvien de même qu'une fasciite périnéale et de la cuisse gauche.

FIGURE 6



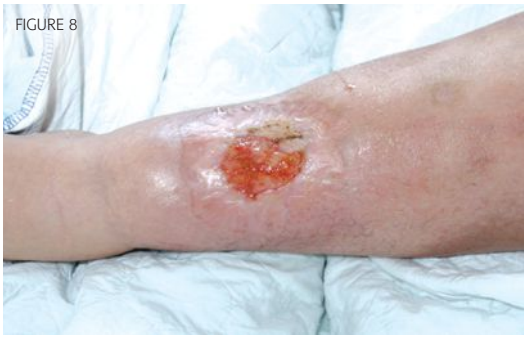
Greffes cadavériques en place, novembre 2010.

FIGURE 7



Évolution de la plaie inguinale, mars 2011.

FIGURE 8



Évolution de la plaie pré-tibiale, mars 2011.

Malheureusement, la patiente a rejeté les greffons à deux reprises, mais on a constaté une contraction de la plaie et une granulation importante qui a rendu possible la mise en place de greffons cutanés autologues d'épaisseur partielle en janvier et février 2011.

Après une hospitalisation de dix mois (en mars 2010), la patiente a été transférée vers son centre régional d'origine (figures 7 et 8). Sa plaie inguinale était alors de 31 cm transverse sur 30 cm craniocaudale. Elle a été traitée par l'infliximab, la prednisone, le méthotrexate et des gammaglobulines.

Discussion

Le PG a été diagnostiqué tardivement chez la patiente, soit un an après la formation de la première plaie. Le PG a été réfractaire aux immunosuppresseurs chez cette patiente recevant une immunothérapie pour une polychondrite. La coexistence d'une infection invasive des tissus mous et d'un PG est parfois signalée dans la littérature[1,6,7]. Les facteurs d'immunosuppression favorisent le développement d'une telle infection et les signes cliniques sont tardifs. En raison d'une septicémie grave, cette patiente a dû subir un débridement très important. Les phénomènes pathergiques provoqués par les multiples débridements et la thérapie par pression négative ont produit une détérioration spectaculaire de la plaie. Bien que selon certains la thérapie par pression négative soit utile contre le PG, dans notre cas, elle a été un facteur aggravant[8, 9]. Une fois la thérapie abandonnée, la maîtrise de l'infection et du PG a permis une contraction et une granulation progressive de la plaie. La mise en place de greffes cadavériques comme pansements biologiques a été bénéfique pour cette patiente et a rendu possible la mise en place d'autogreffes cutanées d'épaisseur partielle. La patiente a toujours maintenu une attitude combative, malgré les douloureux changements de pansements quotidiens et l'hospitalisation prolongée de même que la réhabilitation de longue durée en cours, ce qui a beaucoup contribué à la réussite du traitement. La prise en charge multidisciplinaire et un personnel dévoué ont grandement contribué à l'évolution favorable de l'état de cette patiente.

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John Gregory: I have a newfound appreciation for the rehabilitation required after surgery.

A Patient's Recovery from Emergency Surgery

John Gregory discusses his surgery and the physical and psychological ramifications of a protracted recovery

John has 20 years of healthcare industry experience in the UK, the US and Canada, half of which have been spent in wound care. He completed the International Interprofessional Wound Care Course in 2008. He has served as an industry representative to the CAWC and was chair of the MEDEC Wound Care Committee from 2007 to 2011. He presented an oral poster at the 2009 CAWC conference regarding the use of a blog as an enabler for lower limb assessment. He is currently relocating from Milton, Ontario, to the UK with his wife, Sarah Quart.

Patient history

In October 2010, I presented at a walk-in clinic in Jasper, Alberta, with excruciating abdominal pain. I was referred to the emergency department and airlifted to Edmonton 40 minutes later, where I presented with a sigmoid volvulus. I underwent a sigmoid resection 4 days after my arrival at Misericordia Community Hospital in Edmonton. From the time of the initial emergency airlift, 2 weeks of my life disappeared.

My wife, Sarah, flew immediately from Toronto to join me in Edmonton. We were impressed that the hospital organized homecare follow-up in Milton, and we received a call from the Mississauga and Halton Community Care Access Centre (CCAC) even before we left Edmonton. Despite a physician order stating I was fit to fly, the airline would not allow me to do so and it was challenging to return home.

Prior to discharge, some of the staples were removed on postoperative day 4, creating a dehisced surgical wound that later became infected with a 9 cm tunnel. Although I was cared for daily by homecare, I was fortunate to be referred as a patient to Dr. Gary Sibbald, a dermatologist and wound-healing expert in Mississauga, Ontario. His intervention was analytical, decisive and aggressive. At his clinic, I became both the patient and the student. He excised the wound on 2 occasions to open it up and create a new acute wound. The wound finally closed in the middle of January. I can't imagine how much longer it would have taken without his intervention.

Sarah took time off work and drove me around for weeks after we returned home. I can appreciate how hard this is for any spouse or caregiver. I now also recognize the challenges of integrating back into the workforce, both physically and – more importantly – psychologically.

Physical ramifications

The first 6–8 weeks after my surgery were very tough. I shuffled around for the first couple of weeks, and getting in and out of the car was difficult and painful. I lost 15 pounds as a result of the surgery as I had no appetite; by Christmas, my ribs were visible. Moreover, eating caused distention and bloating, which increased the wound pain and made me even less inclined to eat anything. I wanted nothing other than soup. In addition, sleep was painful and a challenge back in the comfort of my own home. I was uncomfortable at night, no doubt a result of the polypharmacy and the dressing itself.

During the day, I would return exhausted from a round of CCAC, doctor's office and pharmacy visits, collapse and fall asleep. For many weeks I felt sick each morning, often getting up and then lying down again 30 minutes later. It felt so insignificant that I had a wound only a few inches long given the profound effect it was having on how I felt. Prior to the surgery, I was doing about 10 hours of exercise per week, a combination of kayaking, biking, yoga and gym. In the first months of 2011, I experienced dizziness at times. The dizziness eventually disappeared, and 10 months

I recognize the challenges of integrating back into the workforce, both physically and – more importantly – psychologically.

later a cardiologist investigated my unusually low pulse rate with electrocardiography and a Holter monitor. In addition, the surgery and medications resulted in changes to the skin of my trunk and face, making me self-conscious about my appearance. A plastic surgeon recommended that I massage the scar with moisturizer and then use scar treatments.

Five months post-op, I again started yoga, kayaking and spinning, albeit at a gentler intensity. Various healthcare professionals have made me rather anxious about returning to a high level of exercise too quickly, thereby risking a hernia. My goal was to be fit to play at the 2011 Canadian Canoe Polo National Championships in Sudbury, Ontario.

Psychological ramifications

I recognize the strong psychological component to trauma and recovery. I had all this time on my hands, but the fatigue demotivated me to start or do anything. I desperately wanted to use my newfound time productively, but felt weak, drained and lethargic until Christmas. This was depressing. I had moments where I wished to just get back to normal and saw flashes of all the things I could have been doing with the time at my disposal; however, I felt too lethargic to do anything significant. After 8 weeks, I finally felt motivated to get back into a normal morning routine. Only after the wound closed was I again able to shower easily and as often as I wanted.

Reflections

I have a newfound appreciation for the rehabilitation required after surgery. As a manager, I now have a deeper empathy and understand that someone cannot be expected to return to work a week after surgery. I was very fit and healthy going into the surgery, and it took me at least 8 weeks to gain any sense of normality. My own employer was excellent in ensuring that I did not return too quickly. I feel fortunate that at the age of 40, my recovery was relatively short and I was soon able to return to my previous levels of mobility and activity. For elderly patients, I can appreciate that it may take 6–12 months to recover from the surgery itself.

I also have a new respect for talking to patients, and understanding their history and changes in wound appearance. We often discuss addressing patient-centred concerns as part of our wound-healing paradigm to complement treating the cause and local wound care. Being the patient has given me a new perspective on these patient-centred concerns. I was impressed to find a couple of graduates from the International Interprofessional Wound Care Course among the hospital and homecare nurses.

Over the last decade, much has been made of pain at dressing changes. I experienced pain from the use of stainless-steel forceps to pack the wound. Wound care practitioners must do everything possible at the dressing change to avoid damage to the newly vascularized granulation tissue in the wound bed and pain to the patient from the process of removing the wound contact layer, irrigation, assessment and repacking. I identified some potential room for improvement in patient education – particularly at discharge – in terms of expectations for healing, knowing when to seek further medical help and the implications on everyday life, including driving and showering.

Finally, I learned the value of sleep in a hospital environment. As a patient, all I wanted to do was sleep – more than anything else. Product-selection committees should pay attention to patient factors, particularly the noise generated by a piece of equipment. We are lucky to live in a healthcare system with such medical technology; however, at 2:00 am I just wanted to sleep and wished all the equipment was silent and not lit up like a Christmas tree!

ConvaTec Introduces AQUACEL® BURN and AQUACEL® Ag BURN Dressings with Hydrofiber® Technology for Partial Thickness Burns

Innovative dressing technology designed to reduce the need for frequent and painful dressing changes in burn patient care

ConvaTec, a world-leading developer of innovative medical technologies for community and hospital care, announced the introduction of AQUACEL® BURN and AQUACEL® Ag BURN dressings for the management of partial thickness burns (PTBs) and donor sites.

Incorporating Hydrofiber® Technology, AQUACEL® BURN and AQUACEL® Ag BURN dressings create a moist wound environment and are designed to detach during healing and re-epithelialization. The dressing can be left in place for up to 21 days, helping reduce painful dressing changes and the risk of exposure to pathogens in the atmosphere.[1,2] AQUACEL® Ag BURN dressing[a] has been shown to provide activity against antimicrobial-resistant bacteria and potentially reduce the risk of infection.[3b]

Different sizes and shapes from child to adult are available, including gloves. Dressings are reinforced with Nylon stitching to offer patients maximum flexibility and mobility. For more information, call our Customer Relations Center at 1-800-465-6302 or visit www.convatec.ca.

Advanced Wound Dressings with Hydrofiber® Technology from ConvaTec

ConvaTec dressings with Hydrofiber® Technology are soft, absorbent, non-woven wound dressings which gel on contact with fluid, enabling the dressing to lock in exudate and its harmful components[2,4,5c] and to micro-contour to the wound bed,[6c] in response to changing wound conditions.

a. AQUACEL® Ag BURN dressing is the same Hydrofiber® Technology as AQUACEL® Ag dressing **b.** As demonstrated *in vitro* **c.** Applies to AQUACEL® and AQUACEL® Ag dressings

Hydrofiber and AQUACEL are registered trademarks of ConvaTec Inc. AP-012262-CA

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ConvaTec Introduces AQUACEL® EXTRA™ Wound Dressing with Hydrofiber® Technology in Canada

Dressing designed to be nine times stronger and 39% more absorbent than current dressing[1]

ConvaTec, a world-leading developer of innovative medical technologies for community and hospital care, announced the availability of AQUACEL® EXTRA™ wound dressing with strengthening fibers in Canada.

Composed of two layers of Hydrofiber® Technology stitched together, AQUACEL® EXTRA™ wound dressing is designed to be nine times stronger and to give an increased absorbency of 39% over current AQUACEL® wound dressing.[1] Cleared for the same indications as AQUACEL® wound dressing, AQUACEL® EXTRA™ wound dressing will be specifically suitable when managing moderate to highly exuding wounds.

"Clinicians managing acute or chronic wounds know the benefits of Hydrofiber® Technology and this family of products, supported by 15 years of clinical heritage demonstrating our dressings'[a] efficacy," explains Fabien Paquette, Business Director, ConvaTec Canada.

AQUACEL® EXTRA™ wound dressings come in three sizes: 5x5cm, 10x10cm, and 15x15cm. For more information, call our Customer Relations Center at 1-800-465-6302 or visit www.convatec.ca.

Advanced Wound Dressings with Hydrofiber® Technology from ConvaTec

ConvaTec dressings with Hydrofiber® Technology are soft, absorbent, non-woven wound dressings which gel on contact with fluid, enabling the dressing to lock in exudate and its harmful components,[2-4b] and to micro-contour to the wound bed,[5b] in response to changing wound conditions.

a. Applies to AQUACEL® and AQUACEL® Ag dressings **b.** As demonstrated *in vitro*

Hydrofiber and AQUACEL are registered trademarks of ConvaTec Inc. AP-012260-CA

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Systagenix Globally Announces European CE mark of WOUNDCHEK™ Protease Status, the First Point-of-care Diagnostic Test for Chronic Wounds

WOUNDCHEK™ Protease Status is the world's first rapid, point-of-care diagnostic test developed specifically for chronic wounds. WOUNDCHEK™ Protease Status has the potential to revolutionise wound care by enabling early, targeted intervention and cost-effective use of advanced therapies designed to modulate protease activity.

Designed to form part of routine wound assessment, WOUNDCHEK™ Protease Status is easy-to-use and provides results in just 15 minutes at the point of care. This enables the test to immediately influence treatment decisions and help clinicians target advanced wound care therapies more effectively by identifying when elevated protease activity (EPA) exists in chronic wounds.

Although widely understood and recognised as a key marker in wound healing[1], today EPA in chronic wounds goes undetected, as there are no visual cues for it[2]. A recently published study showed that chronic wounds with EPA have a 90% probability they will not heal without appropriate intervention[3]. The estimated 30 million worldwide chronic wounds treated each year account for approximately 3% of total health expenditure[4]. With almost 30% of non-healing wounds having EPA, the addition of a test could result in more effective treatment choices, leading to significant cost savings to healthcare providers.

Health Canada submission is currently in process.

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TCC Gold Standard Made Even Easier!

Total contact casting is the most effective offloading device in healing neuropathic foot wounds, as overwhelmingly validated by clinical results, and is in fact considered Gold Standard by most diabetic foot specialists. Unfortunately, it is not widely used, for different perceived reasons. In order to facilitate its use, BSN Medical developed the **Total Contact Cast Kit** in partnership with healthcare professionals dealing with diabetic foot ulcers every day. The TCC Kit combines specifically chosen and proven casting materials to provide an intimate comfortable close fit and optimized healing environment for a cost-effective treatment.

Today, BSN Medical is proud to launch a new improved version of the kit, the **Cutimed® TCC Kit**, which is even easier to use! In addition to the advantages of the original version, the new Cutimed® TCC Kit now identifies each product for step-by-step application, and includes pre-cut slabs to replace some of the rolls for faster application. Cutimed® TCC Kit simplifies the implementation of the pressure offloading gold standard in the treatment of diabetic foot ulcers.

For optimal results in managing diabetic foot ulcers, BSN Medical recommends the use of **Cutimed® Sorbact®**. This unique and effective antimicrobial dressing with microbe-binding action can be used, without risks involved, as it does not contain any chemical agents.

Please contact your BSN local representative, or our customer service at 1-877-978-5526, to find out more about our unique solution for diabetic foot ulcers.

3M™ Coban™ 2: Redefining Compression Bandaging for Lymphedema Patients

3M™ has expanded the Coban™ 2 Compression line to include new sizes specifically for lymphedema therapy. This was a result of many years of research in Europe and Canada and the launch was accompanied by the publication of numerous clinical articles.

Until now, compression bandaging has been physically and emotionally taxing for patients and clinicians. 3M™ Coban™ 2 Compression System materials enable new application techniques for a better therapy experience and a higher standard of care.

Clinicians can easily adapt application to accommodate any size, shape or tissue consistency with only two layers of conformable, cohesive materials. The compression layer is applied at full stretch to reduce application variability and consistently deliver the right amount of pressure.

Coban™ 2 is a disposable single-use system that is cost-effective and eliminates the time and expense of washing and re-rolling bandages while minimizing the risk associated with potential contamination. The materials used in the two thin layers of the system are safe for skin and were developed with unique stretch and cohesion properties to provide ideal compression and help patients overcome the challenges of wearing bandages during the intensive phase of treatment for lymphedema.

> *"They are so much easier for the therapist to put them on and honestly you don't know they are on when she has finished." – Patient P3, Canada1*

> *"The cumulative results from these research studies support that the 'Coban 2 Compression System' is clinically effective and is set to fundamentally change the field of lymphoedema." – Christine Moffatt CBE PhD, Lead Researcher, Derby Hospitals, Honorary Professor in Nursing & Health Care, Glasgow University*

For more information, visit www.3m.ca/coban2layer.

Covidien Offers the Canadian Wound-care Community a Valuable Tool in the Management of Wounds

Covidien is proud to offer a complete line of traditional and advanced wound-care products to meet the needs of Canadian clinicians. But what sets us apart from other wound-care companies is our line of polyhexamethylene biguanide (PHMB) impregnated dressings.

Part of a proven prophylactic infection prevention program, PHMB has gained popularity amongst many clinicians across the country and has recently been added to the CAWC Product Picker as a safe and effective tool in wound management.

PHMB has been in general use for approximately 60 years, with no evidence of the development of resistance. Exerting little toxicity, it has been found to be safe and effective. PHMB is a bacterial agent that is effective against Gram-negative and Gram-positive bacteria, as well as fungi and yeast. It acts to kill bacteria by integrating into the cell membrane and reorganizing the membrane structure. This structural change prevents the cell from pumping PHMB out of the membrane, thus bactericidal concentrations are maintained in the cell. PHMB's mode of action attacks the bacteria by disrupting the cytoplasmic membrane of the microorganism, as the bacteria is absorbed into the dressing (Coutts, 2009).

Our foam dressings, including our recently launched Kendall™ AMD foam with border, are available in a 0.5% PHMB concentration. Our various gauze formats, including Kerlix™ AMD rolls and sponges, Curity™ AMD packing strips and Excilon™ AMD IV sponges, are also available in a 0.2% PHMB concentration.

As a proud member of the CAWC, Covidien participates in all of the L-Series educational courses held across Canada. We look forward to seeing you in November at the CAWC National Conference in London.

Coloplast Canada Launches Biatain® Silicone and Biatain® Silicone Lite!

At Coloplast we are pleased to introduce two new products to our Canadian customers. Biatain Silicone and Biatain Silicone Lite. Biatain Silicone is a Barrier Free Foam™ that combines the superior absorbency of Biatain foam with the gentleness of a silicone adhesive, delivering three unique benefits:

Silicone only where you need it, on the border
The unique design of Biatain Silicone has silicone adhesive only on the border and not on the foam, leaving the foam barrier free. This Barrier Free Foam™ is measured to have superior absorption and retention properties – even under compression/pressure.

Ultra flexible design for superior fit to wound and body
With only two layers, Biatain Silicone is far more flexible, creating a perfect fit anywhere on the body – even in hard to dress areas.

Easy to apply even with gloves
Biatain Silicone has a unique three part opening. This allows for the clinician to put the dressing safely on the patient. The dressing is easy to apply even with gloves!

To get more information on Biatain Silicone and Biatain Silicone Lite, go to our website www.coloplast.ca.

To request samples, please email or call us.

NUTRITION SOLUTIONS
FOR PRESSURE ULCER MANAGEMENT

Pressure ulcers are a common, costly and debilitating form of chronic wounds which occur with relative frequency in all care settings. Defined by the National Pressure Ulcer Advisory Panel (NPUAP) as "an area of localized damage to the skin and underlying tissue caused by pressure, shear, friction, moisture or a combination of these factors"[1], pressure ulcers are a preventable condition and a major concern for patients and healthcare facilities alike.

In Canada, as many as 30% of individuals in non-acute care centres (rehabilitation, long term care, complex continuing care) and 25% of those in acute care hospitals develop pressure ulcers at some point during their stay[2].

Common Characteristics of Patients/Residents with Pressure Ulcers include[3]:

- Over 70 years of age
- Poor nutritional status
- Prolonged periods of immobilization; bed or chair-bound
- Incontinence
- Poorly controlled diabetes
- Circulatory problems
- Fracture recovery

The burden of pressure ulcers extends beyond the obvious significant patient suffering and decreased quality of life. Increased caregiver anguish, extra work for healthcare providers and ultimately millions spent in Canadian healthcare dollars make pressure ulcers a serious clinical concern[4].

Nutrition and pressure ulcer healing are closely linked. Research shows that malnutrition negatively affects wound healing by prolonging early phases of the healing process and by decreasing the production of collagen and other essential healing components[5]. Malnutrition has also been associated with increased wound infections which can further delay healing.

Elderly individuals with pressure ulcers are likely to be malnourished[6]. Assessing nutritional status is an important first step in designing a successful nutrition care plan and providing for optimal pressure ulcer healing. As metabolic rates are increased in the presence of chronic wounds such as pressure ulcers, without adequate energy intake, muscle breakdown will occur to provide amino acids and energy. The result is an increased risk for malnutrition, poor healing capabilities and a persistent non-healing chronic ulcer. The NPUAP-EPUAP recommended guideline for energy to optimize pressure ulcer healing, is 30-35 Kcal/kg/day and is further increased to 35-40 Kcal/kg/d for those who are underweight or are losing weight[7,8].

All stages of wound healing require protein. Severe protein depletion is associated with decreased wound strength and increased wound infection rates[5]. Older adults require an increased protein intake above the current Dietary Reference Intake (DRI) due to the increased risk of muscle loss (sarcopenia) and decreased immune function[9]. It has been shown that increased protein intake is associated with enhanced wound healing rates.[5,10] NPUAP-EPUAP guidelines suggest that 1.2 - 1.5 g protein/kg/d is required for healing pressure ulcers[8]. Intakes as high as 2.0 g/kg/day have been suggested for multiple or large, full thickness pressure ulcers[7].

Micronutrient supplements such as Vitamin C and Zinc are recommended only when a dietary deficiency has been demonstrated or diagnosed[8]. High doses of vitamin C have not been shown to accelerate wound healing and high-dose zinc supplementation may in fact interfere with healing[11]. Supplementation of vitamins and minerals beyond the DRI's has not been proven as beneficial.

Management of pressure ulcers requires a multidisciplinary team approach. All patients with pressure ulcers should be nutritionally assessed, have their food intake, hydration and blood glucose monitored frequently, and be provided with the means to meet their elevated protein and energy needs for healing. Oral nutritional supplements are beneficial to help achieve these needs, combat under-nutrition and enhance healing[7].

Nestlé Healthcare Nutrition has a variety of nutrition solutions to help with pressure ulcer healing. The MedPass program with **Resource 2.0** provides 480 Kcal and 20g protein per day to support elevated nutritional needs for pressure ulcer healing. In addition, **Beneprotein**, a concentrated source of 100% whey protein, can help to meet the protein needs of your patients and residents.

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