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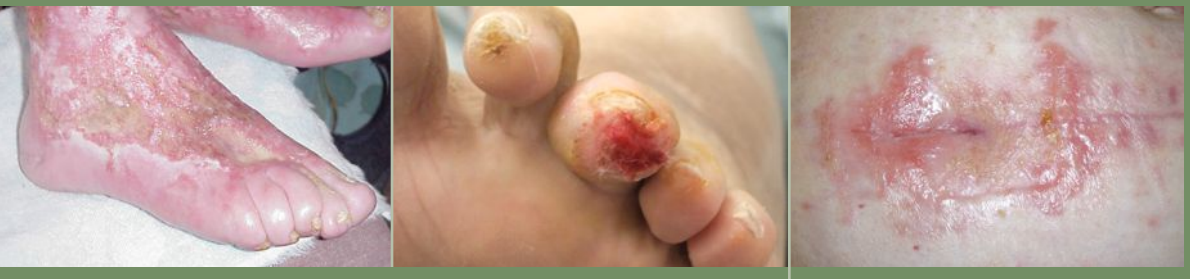
Wound Care

Soins des plaies

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



Basic Principles of Wound Healing

Diabetic Foot Surgery: A Review of Current Procedures

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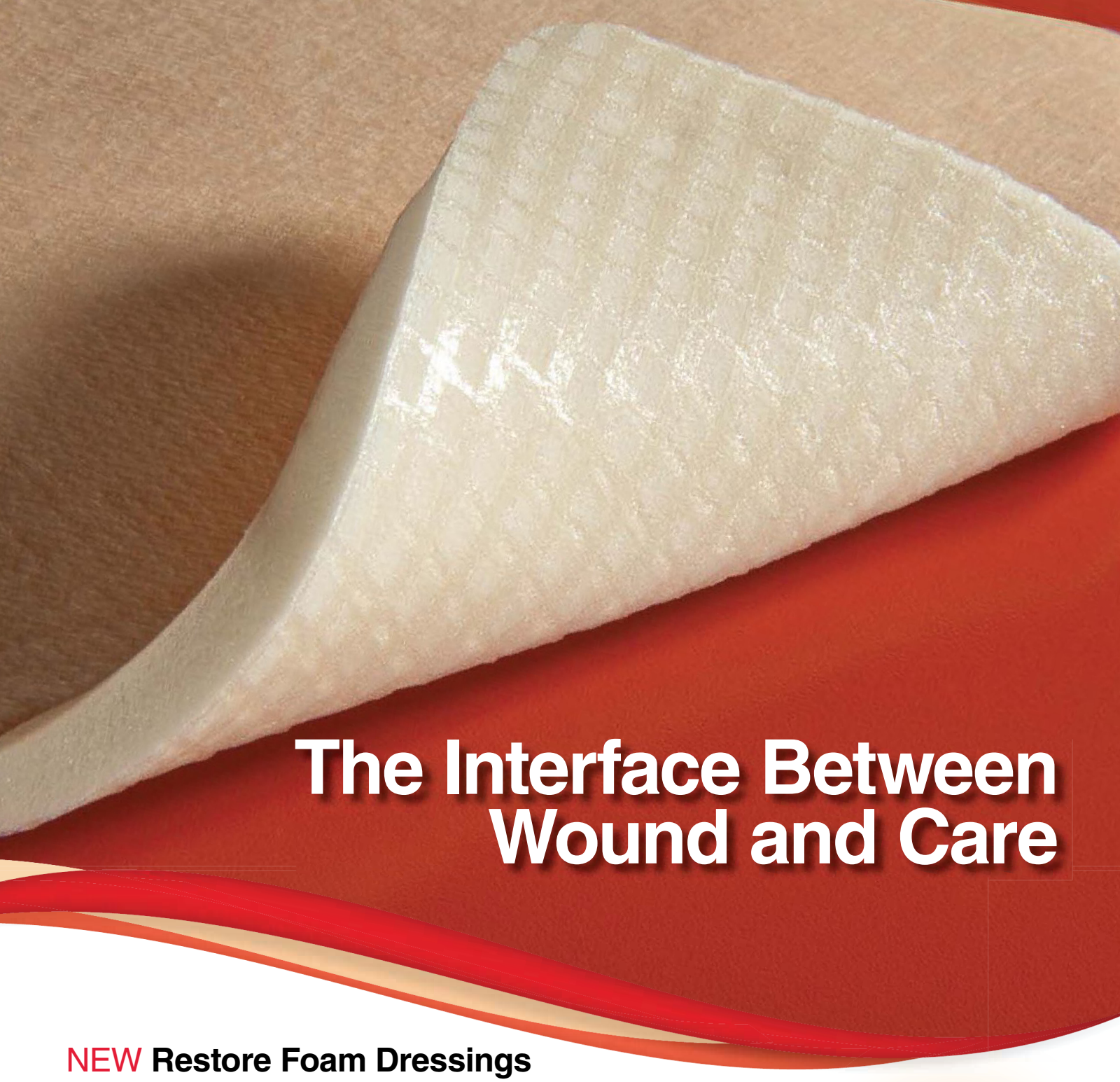

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The Canadian Association of Wound Care is a non-profit organization of health-care professionals, industry participants, patients and caregivers dedicated to the advancement of wound care in Canada.

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.

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Basic Principles of Wound Healing

An understanding of the basic physiology of wound healing provides the clinician with the framework necessary to implement the basic principles of chronic wound care

BY

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Introduction

Wound healing is a complex and dynamic process, with the wound environment changing with the shifting health status of an individual. Knowledge of the physiology of the normal wound healing trajectory through the phases of hemostasis, inflammation, granulation and maturation provides a framework for understanding the basic principles of wound healing. Through this understanding, the healthcare professional can develop the skills required to care for a wound and the patient can be helped with the complex task of tissue repair.

Wounds that do not heal as expected should prompt the healthcare professional to search for unresolved underlying causes. A wound that does not heal as expected requires care that is patient-centred, holistic, interprofessional, collaborative, cost-effective and evidence-based.

This paper addresses the following topics: Why do wounds occur? How do they heal? What factors interfere with healing? When is a wound considered chronic? What is the nature of good chronic wound care? It is hoped that the explanations regarding these basic principles will provide a framework for further study and exploration into the complex area of wound management.

Why do wounds occur?

In any natural disaster, the damaging forces must be identified and stopped before repair work can begin. So, too, in wound care must the basic underlying cause(s) of a wound be identified and controlled as best as possible before wound healing can begin. Common underlying causes of tissue damage are listed in Table 1.

How do wounds heal?

Research regarding acute wounds in animal models demonstrates that wounds heal in 4 phases. It is believed that chronic wounds also undergo 4 basic phases of healing (although some authors combine the first 2 phases). These are:[1]

- hemostasis;
- inflammation;
- proliferation (also known as granulation and contraction); and
- remodelling (also known as maturation).

Kane's analogy to the repair of a damaged house provides a visual understanding of and connection to the basic physiology of wound repair (Table 2).[2]

Hemostasis

Once the source of damage to a house has been removed and before work can start, utility workers must cap damaged gas or water lines. So, too, in wound healing must damaged blood vessels be sealed. In wound healing, the platelets are the cells that act as utility workers sealing off the damaged blood vessels. The blood vessels themselves constrict

TABLE 1

Common underlying causes of tissue damage

- Trauma (initial or repetitive)
- Scalds and burns (thermal and chemical)
- Animal bites or insect stings
- Pressure
- Vascular compromise (arterial, venous, lymphatic or mixed)
- Immunodeficiency
- Malignancy
- Connective tissue disorders
- Metabolic disease, including diabetes
- Nutritional deficiencies
- Psychosocial disorders
- Adverse effects of medications

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TABLE 2

The 4 phases of wound healing

Phase of healing	Time post injury	Cells involved in phase	Function or activity	Analogy to house repair
Hemostasis	Immediate	Platelets	Clotting	Utility workers cap-off broken utilities
Inflammation	Day 1–4	Neutrophils Macrophages	Phagocytosis	Unskilled labourers clean up the site Contractors direct activity
Proliferation (granulation and contraction)	Day 4–21	Macrophages Lymphocytes Angiocytes Neurocytes Fibroblasts Keratinocytes	Fill defect Re-establish skin function Closure	Subcontractors start work: • framers; • plumbers; • electricians; and • roofers and siders
Remodelling (maturation)	Day 21–2 years	Fibrocytes	Develop tensile strength	Interior finishing

in response to injury, but this spasm ultimately relaxes. The platelets secrete vasoconstrictive substances to aid this process, but their prime role is to form a stable clot sealing the damaged vessel.

Under the influence of ADP (adenosine diphosphate) leaking from damaged tissues, the platelets adhere to the exposed type 1 collagen.[3] They become activated and secrete adhesive glycoproteins, leading to platelet aggregation. They also secrete factors that interact with and stimulate the intrinsic clotting cascade through the production of thrombin, which in turn initiates the formation of fibrin from fibrinogen. The fibrin mesh strengthens the platelet aggregate into a stable hemostatic plug.

Finally, platelets also secrete growth factors such as platelet-derived growth factor, which is recognized as one of the first factors in initiating the subsequent healing steps. These growth factors recruit neutrophils and monocytes (initiating the next phase of wound healing), stimulate epithelial cells and recruit fibroblasts. Hemostasis occurs within minutes of the initial injury unless the patient has underlying clotting disorders.

Inflammation

Clinically, inflammation (the second stage of wound healing) presents as erythema, swelling and warmth often associated with pain, the classic "rubor et tumor cum calore et dolore." This stage usually lasts up to 4 days post injury. In the damaged house analogy, once the utilities are capped the second job is to clean up the debris. This is a job for unskilled labourers. In a wound, these unskilled labourers are the neutrophils (polymorphonucleocytes).

The inflammatory response causes the blood vessels to become leaky, releasing plasma and neutrophils into the surrounding tissue.[4] The neutrophils phagocytose debris and microorganisms and provide the first line of defence against infection. As they digest bacteria and debris, neutrophils die and release intracellular enzymes into the surrounding matrix, which further digest tissue. As fibrin is broken down as part of this clean-up, the degradation products attract the next cells involved such as fibroblasts and epithelial cells. They are aided by local mast cells.

The task of repairing a house is complex and requires someone, such as a contractor, to direct this activity. Similarly, wound repair requires coordinated cell activity and good cell-to-cell communication. Cells communicate through soluble proteins call cytokines and growth factors. These cytokines and growth factors are released by 1 cell and bind to a receptor on a target cell. Once a cytokine binds to a target cell it stimulates the cell to move. Growth factors, on the other hand, stimulate the target cell to either divide and produce more cells or synthesize and release substances such as collagen, which is required to form the extracellular matrix. The extracellular matrix also plays an active role in wound healing by interacting with the cells through receptors called integrins, leading to platelet activation, epithelial migration and fibroblast movement.[5]

In wound healing, cells known as macrophages act as the "contractors." Circulating monocytes differentiate into macrophages after they exit the blood vessels and come in contact with the extracellular matrix. Macrophages are able to phagocytose bacteria and provide a second line of defence. Macrophages also secrete extracellular enzymes to degrade necrotic tis-

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TABLE 3

Differentiating inflammation from infection[7]

Inflammation	Factor	Infection
Coexisting systemic disease	**Comorbidity**	Decreased host resistance
Constant, onset	**Pain**	Increasing with lesions
Multiple sites, symmetric	**Location**	Single location, asymmetric
Palpable purpura, livedo pattern, rolled border, focal necrosis satellite lesions	**Morphology**	Classic or subtle signs of infection, soft tissue crepitus
Local	**Erythema**	Advancing
Normal or warm	**Skin temperature**	Warm or hot

sue at the wound site. These enzymes belong to a family of substances called matrix metalloproteases (MMPs). MMPs require calcium to form a functional shape and zinc for the active site.

About 20 different types of MMPs are secreted by many different cells — including neutrophils, macrophages, epithelial cells and fibroblasts — under the influence of inflammatory cytokines such as tumour necrosis factor-alpha and interleukin-1 and -6. MMPs act on all components of the extracellular matrix and are responsible for removing devitalized tissue, repairing lost or damaged tissue and remodelling. MMPs are balanced by tissue inhibitors of metalloproteases (TIMPs), which are released locally by cells and inactivate MMPs by reversibly binding to them. Uncontrolled MMPs can degrade newly formed tissue or destroy growth factors.

Macrophages secrete a variety of cytokines and growth factors — such as fibroblast growth factor, epidermal growth factor, transforming growth factor-beta and interleukin-1 – which appear to direct the next stage.[6]

Inflammation – the body's response to trauma — can be confused with infection. However, inflammation is a normal response to tissue injury, but with increased bacterial burden and decreased host resistance (Table 3).[7]

Proliferation

The proliferation phase starts approximately 4 days after wounding and usually lasts until day 21 in acute wounds, depending on the size of the wound and the health of the patient. It is characterized by angiogenesis, collagen deposition, granulation tissue formation, wound contraction and epithelialization. Clinically, proliferation is observed by the presence of pebbled red tissue or collagen in the wound base and involves replacement of dermal tissues and sometimes subdermal tissues in deeper wounds, as well as contraction of the wound. In the house analogy, once the site has been cleared of debris under the direction of the contractor, framers move in to build the framework of the new house. Subcontractors can now install new plumbing and wiring on the framework and siders and roofers can finish the exterior of the house.

The "framer" cells are fibroblasts, which secrete the collagen framework on which further dermal regeneration occurs. Specialized fibroblasts are responsible for wound contraction. The "plumber" cells are the pericytes, which regenerate the outer layers of capillaries, and the endothelial cells, which produce the lining. This process is called angiogenesis. The "roofer" and "sider" cells are the keratinocytes, which are responsible for epithelialization. In the final stage of epithelialization, contracture occurs as the keratinocytes differentiate to form the protective outer layer or stratum corneum.

In a healing wound, the cells under the influence of growth factors divide to produce new cells, which migrate to where they are needed under the influence of cytokines. There is a balance between the MMPs and TIMPs so that there is a net production of new tissue. In chronic wounds, in contrast, in which healing is stalled, cell division and migration are suppressed, there are high levels of inflammatory cytokines and MMPs, and low levels of TIMPs and growth factors. Cells are often senescent and unresponsive to the growth factors. This lack of response is characteristic of a chronic inflammatory state. It may be caused by an increased bacterial burden, the presence of devitalized tissue, chronic ischemia or repetitive trauma.

Infection = (number of organisms × virulence of organisms) ÷ (host resistance)

Remodelling

Once the basic structure of the house is completed, interior finishing may begin. Similarly, in wound repair, the healing process involves remodelling and realignment of the collagen tissue to produce greater tensile strength. In addition, cell and capillary density decrease. The main cells involved in this process are the fibroblasts. Remodelling can take up to 2 years after wounding. This explains why closed wounds can quickly breakdown if attention is not paid to the initial causative factors.

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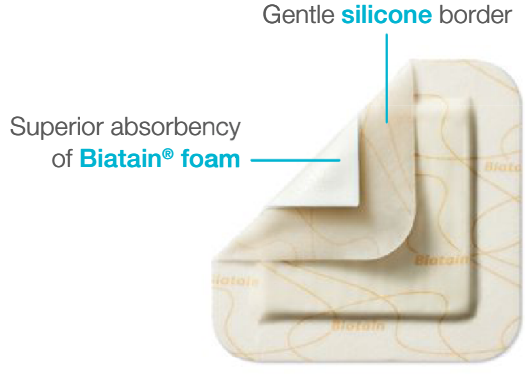

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TABLE 4

Healability of wounds

Type of wound	Characteristics	Examples
Healable	• Causes and cofactors that can interfere with healing have been removed • Wound healing occurs in a predictable fashion • May be acute or chronic	Pressure ulcer, where pressure and other factors such as shear are managed
Maintenance	Causes and cofactors that can interfere with healing are removable but have not been removed due to patient or system factors	Pressure ulcer, where pressure is not managed
Nonhealable	Causes and cofactors that can interfere with healing cannot be removed	Gangrenous foot or malignant wound

determine the type of wound they are caring for to set realistic goals:

- *Acute wounds:* Heal in a normal, orderly sequence of repair as described above.
- *Chronic wounds:* Fail to progress through a normal, orderly and timely sequence of repair, usually because of unresolved factors that interfere with healing. These wounds may eventually pass through the repair process without restoring sustained anatomical and functional results.

Factors that can interfere with healing

Louis Pasteur stated: "The germ is nothing. It is the terrain in which it is found that is everything." It is very similar with wounds! Factors that interfere with wound healing must be addressed in a holistic fashion looking, as Pasteur suggested, at the terrain in which the wound is found. The individual with a wound has a wide terrain, from the local wound environment to the environment in which he or she lives, and that terrain may determine healability. In other words, wounds do not exist in isolation from the patient as a whole.

Factors that may interfere with healing in the local wound environment include infection, necrotic tissue and the vascular supply. In addition, coexisting physical and psychological factors such as nutritional status, disease states (e.g. diabetes, cancer, arthritis) and mental health problems can all impact wound healing.

The next environment is the home in which the patient lives. This may raise concerns regarding immobility, cleanliness and family support. The broader community environment can also impact wound healability through the availability of support services such as personal care in the home, the financial cost of supplies and services, and the availability of skilled personnel and facilities.

Once healability is determined, wounds can be classified as healable, maintenance or nonhealable (Table 4).

When is a wound considered chronic?

In healthy individuals with no underlying factors, an acute wound should heal within 3 weeks with remodelling occurring over the next year or so. If a wound does not follow the normal trajectory then it may become stuck in 1 of the 4 phases, becoming chronic. Chronic wounds are thus defined as wounds that have "failed to proceed through an orderly and timely process to produce anatomic and functional integrity, or proceeded through the repair process without establishing a sustained anatomic and functional result."[8]

The presence of a chronic wound should trigger the clinician to search for underlying causes that may not have been addressed. Better yet, an understanding of the causative factors should lead clinicians to proactively address these factors in at-risk populations so that chronic wounds are prevented.

Best practice and wound healing

Wound healing is a science, but due to the complex nature of the patient it is also an art. The care required to support wound healing needs to be guided by both the available evidence and clinical judgment. Clinical decision-making also involves considering patient preferences, circumstances, values and rights.

Once the clinical problem has been identified and a wound-healing outcome determined, there are 3 key steps:[9]

1. Identify the best evidence available for treatment.
2. Evaluate the client, patient or resident risk factors.
3. Recognize limitations in: available resources; staff and human resources; equipment and supplies; and assessment tools and techniques.

Best practice = consideration of the best evidence + patient risk + available resources

Evaluation of healing

Clinicians must remember that wound closure is only 1 outcome parameter. Patients with wounds that are unlikely to heal (e.g. maintenance or nonhealable wounds) must have alternative outcome expectations. These might be wound stabilization, reduced pain, reduced bacterial load, decreased dressing changes or a return to normal daily routines and activities.[10]

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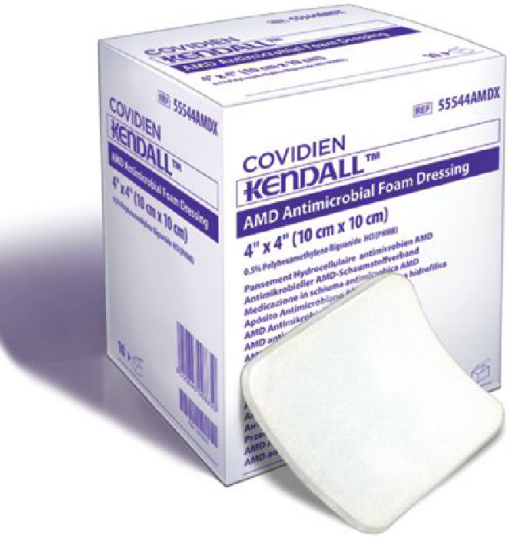

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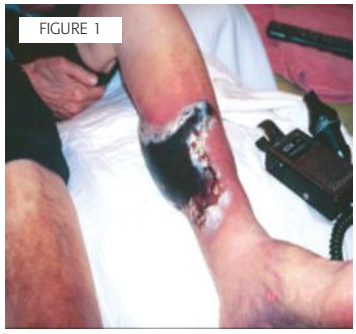

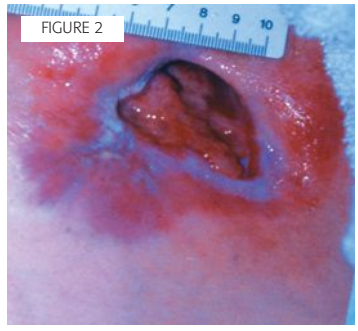

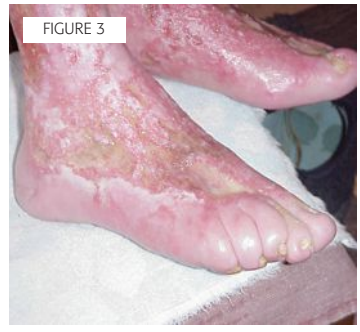

Figure 1. An elderly patient who suffered trauma when she banged her leg on a coffee table. She is on warfarin, which contributed to the injury becoming a large black hematoma. What is the safest way to support the healing of this wound?

Figure 2. A young spinal-cord-injured patient with a chronic pressure ulcer surrounded by erythema. Is the erythema caused by infection, irritation of wound fluid, incontinence or continual pressure to the area?

Figure 3. Chronic ulcers in a frail elderly woman with a long history of lower leg edema related to decreased mobility. The ulcer drains copious amounts of chronic wound drainage causing irritation to the surrounding skin. The patient sits for most of the day, which worsens the leg edema. Although compression is now reducing the edema, how can the wound fluid be controlled to enable healing?

Wound-healing challenges

The clinician working in wound care needs to become a detective. All possible factors and cofactors that may influence healing must be identified. Due to the multifactorial nature of chronic wounds, a thorough health and physical assessment is mandatory (Figures 1, 2 and 3).

Summary and conclusions

The wound-healing approach must incorporate the following themes:

- *Patient centred:* The clinician should remember that he or she is treating with a person who happens to have a chronic wound. A comprehensive wound-management plan can be developed, but without patient buy-in it is doomed to fail.
- *Holistic:* Best practice requires the assessment of the "whole patient," not just the "hole in the patient." All possible contributing factors must be explored.
- *Interprofessional:* Wound care is a complex business that requires skills from many disciplines. Nurses, physiotherapists, occupational therapists, dietitians, chiropodists or podiatrists, orthotists and physicians (both generalists and specialists) should all be included in the team. In some clinical settings, other professionals such as social workers or rehabilitation specialists may also be involved in care. Management and administration should be a further part of the team to support the team and provide required resources or practice change.
- *Evidence-informed:* In today's healthcare environment, treatment must be informed by the best available evidence and demonstrate cost-effectiveness.

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Diabetic Foot Surgery:

A Review of Current Procedures

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Introduction

Foot pathology related to diabetes is complex, debilitating and costly to our healthcare system. The most frequent underlying etiologies are neuropathy, trauma, deformity, high plantar pressures and peripheral arterial disease (PAD).

There is consistent evidence that early identification and aggressive management of people with diabetes—through an integrated, multidisciplinary approach—can prevent problems becoming exacerbated and reduce the incidence of amputation. Treatment should always be patient-centred, and goals should include prompt lesion healing, preventing recurrence and quality of life factors.

Surgical management of diabetic foot ulcers can be either preventive or curative, depending upon the nature of the defect. This paper reviews the surgical procedures most often performed for foot pathologies resulting from diabetes, namely revascularization, surgical offloading and amputation.

Revascularization

People with diabetes have an increased incidence and severity of PAD compared with the general population.[1,2] Every 1% increase in glycated hemoglobin in people with diabetes corresponds to a 26% increase in the risk of PAD.[3]

PAD occurs when arterial plaques or stenoses form within the arterial lumen and impede blood flow. In the lower extremities, this process occurs slowly and over a wide area of the artery, most often occurring distally in the femoral-popliteal and tibial arteries in people with diabetes.[4-6] Blood flow can be temporarily diverted through smaller arterial branches via the collateral circulation. In moderate to severe stages of disease blood flow is severely restricted (i.e. stenosed) or completely blocked (i.e. occluded).

Clinical manifestations typically begin with intermittent claudication in the form of slow walking, calf cramping and leg fatigue.[7] In 50% of patients, however, symptoms of claudication are not present because of reduced activity or diabetes-associated peripheral neuropathy.[8] The symptoms may progress to disabling pain at rest, ulceration with or without infection, gangrene, limb loss and death.[9] Given the severity of outcomes, it is imperative that people with diabetes are monitored for the presence or worsening of PAD and appropriate referrals or interventions are undertaken to prevent and manage the complications of PAD.

Treatment of peripheral arterial disease

The treatment of PAD is dependent on the stage and extent of the disease, but may include risk-factor modification, exercise programs, consistent foot care, antibiotic use and surgical intervention.[4,6] Patients should be carefully monitored for changes to arterial circulation in the form of decreased or impalpable pulses, necrotic ulcerations, abnormal ankle–brachial index measurements (<0.9), disabling intermittent claudication, critical limb ischemia, rest pain or tissue loss (including ulceration). Any of these warrant further arterial testing and referral.[6,7,10]

Referral to a vascular surgeon is required to determine whether surgical intervention is needed to improve the vascular supply. Primary indications for revascularization include disabling claudication, critical limb ischemia, rest pain or tissue loss (including ulceration) refractive to conservative therapy.[6,8,10] Wound-related revascularization can be identified by the wound location and the artery preferentially requiring the return of arterial supply.[11] These wound

TABLE 1

Wound-related revascularization[11]

Location of wound	Artery that should be preferentially revascularized
Heel	Peroneal or posterior tibial
Plantar foot	Posterior tibial
Lateral ankle	Peroneal
Dorsal foot	Anterior tibial

locations and arteries can be identified by the angiosomes (anatomic areas and artery source) in Table 1. This may be helpful to clinicians treating wounds in people with PAD and an ischemic foot (i.e. a foot with impaired arterial blood flow).[11]

Endovascular interventions require determining the evidence, stage and extent of disease, as well understanding the expected benefits and risks related to these interventions.[6,10,12] The most common surgical interventions include arterial bypass and percutaneous transluminal angioplasty.[6,10,12] These are described in Table 2.

People with diabetes and PAD often have an altered distribution of lower-extremity disease, with severe arterial occlusive disease below the knee in the runoff vessels (collateral circulation). As this worsens, the success of percutaneous intervention declines.[4] Generally, percutaneous transluminal angioplasty with or without stenting is preferred for patients with focal disease and restorable runoff, while surgery (arterial bypass) is preferred for patients with diffuse disease and poor runoff.[4,6,12] Outcomes following arterial surgery are becoming increasingly positive as techniques are refined.[13–15]

Some limbs cannot be revascularized due to the lack of a target vessel, the unavailability of autogenous vein or irreversible gangrene beyond the midfoot (Figure 1).[12] In addition, those in poor general health may be at high risk of death if major revascularization is attempted. In these situations, a choice must be made between primary amputation and prolonged medical therapy.[6,10,12]

Conservative therapy for limbs with a questionable arterial supply or those that cannot be revascularized includes pain management, avoidance of further tissue damage and infection, avoidance of sharp debridement and moist wound healing, and the use of broad-spec-

TABLE 2

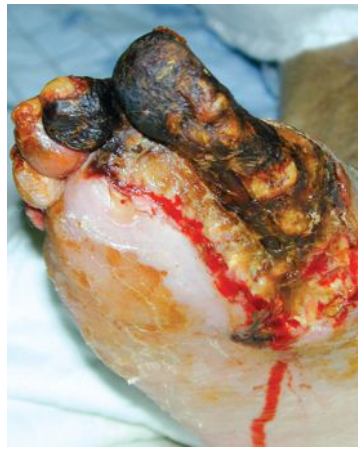
Comparison of revascularization procedures for peripheral arterial disease[6,10,12]

	Arterial bypass	Percutaneous transluminal angioplasty
Surgical definition	An open surgical procedure where a graft or new blood vessel is bypassed around an area of narrowing or blockage A healthy vein or synthetic material is used as a graft	Endovascular: a small percutaneous incision is made in the groin. The procedure os completed inside the artery using a catheter and interventional radiology May involve: • angioplasty—a balloon-tipped catheter is inflated and deflated, pressing plaque against the artery wall and opening a narrowed area; • atherectomy—a tiny blade, laser or rotating burr is inserted through the catheter to remove plaques; or • stenting—a mesh tube is placed into the vessel, using a catheter to reperfuse vessel
Expected benefit	Revascularization of all lesions	Focal disease (stenosis of larger, more proximal vessels) and improved claudication symptoms
Common arteries	Popliteal Tibial	Iliac Femoral
Durability	Greater durability	Long-term efficacy uncertain
Risks	Association with morbidity, mortality and graft occlusion is higher for people with diabetes Other problems may include bleeding, blood clots, infection, heart or lung complications, kidney problems and loss of the toe or foot	Restenosis, lower-limb salvage rates in people with diabetes, rupture of artery, bleeding, blood clots, heart or lung complications, kidney problems, loss of toe or foot and death

trum antiseptics.[16,17] In people with diabetes and advanced PAD, close collaboration is required between the patient, his/her family and caregivers, and the wound care practitioner, physician and vascular surgeon.[6,10,16]

People with diabetes and PAD require careful and ongoing monitoring to identify arterial changes in the progression of PAD that place a limb at risk for amputation.[6,12] Risk factor modification (lowering of blood pressure, cholesterol and blood glucose levels, smoking cessation and exercise), regular foot care and avoidance of foot injury are necessary to prevent and manage PAD.[6,10,17,18] Referral to a vascular surgeon for evaluation of the arterial supply should not be delayed when an arterial impairment is identified.[12] Surgical interventions may be necessary to improve the arterial supply, and outcomes are improving for those with diabetes and PAD.[10,12]

FIGURE 1



A person with diabetes and PAD presenting with significant necrotic 1 and 2 digits. This person was not a candidate for revascularization because of ill health.

Surgical offloading

Deformity, peripheral neuropathy and trauma often lead to foot ulceration.[19,20] Structural alterations in the foot resulting from deformity lead to high plantar foot pressures and increased dorsal, medial or lateral pressure on the foot. This can consequently place the foot at risk for ulceration.[20]

Surgical correction of problematic deformities may prevent ulcers from occurring and help to heal those that are present.[19] Conversely, the disadvantages of surgical offloading include a risk of complications, including postoperative wound infection, induction of acute neuro-osteoarthropathy and development of ulcers at other sites. Such risks can be minimized through careful patient selection and a thorough evaluation of comorbidities. Table 3 outlines the American Diabetes Association's risk classification system.[21]

Sharp surgical debridement

Sharp surgical debridement has multiple advantages, including appropriate evaluation of the wound bed, penetration of topical agents, changing the wound stage from a chronic to acute wound and reducing plantar pressure.[22]

Patients with foot ulcers with an adequate blood supply can undergo sharp surgical debridement of devitalized and necrotic tissue at the bedside. The procedure is quick, cost effective and painless (because of the presence of neuropathy). However, clinicians must

TABLE 3

Risk classification system of the Task Force of the Foot Care Interest Group of the American Diabetes Association[21]

Risk category	Definition	Treatment recommendations	Suggested follow-up
0	No LOPS, no PAD, no deformity	Consider patient education on foot care, including information on appropriate footwear	Annually (by generalist and/or specialist)
1	LOPS ± deformity	Consider prescriptive or accommodative footwear Consider prophylactic surgery if deformity is not able to be safely accommodated in shoes Continue patient education	Every 3–6 months (by generalist or specialist)
2	PAD ± LOPS	Consider the use of accommodative footwear Consider a vascular consultation for combined follow-up	Every 2–3 months (by generalist or specialist)
3	History of ulcer or amputation	Consider patient education on foot care Consider vascular consultation for combined follow-up if PAD present	Every 1–2 months (by specialist)

LOPS = loss of protective sensation; PAD = peripheral arterial disease

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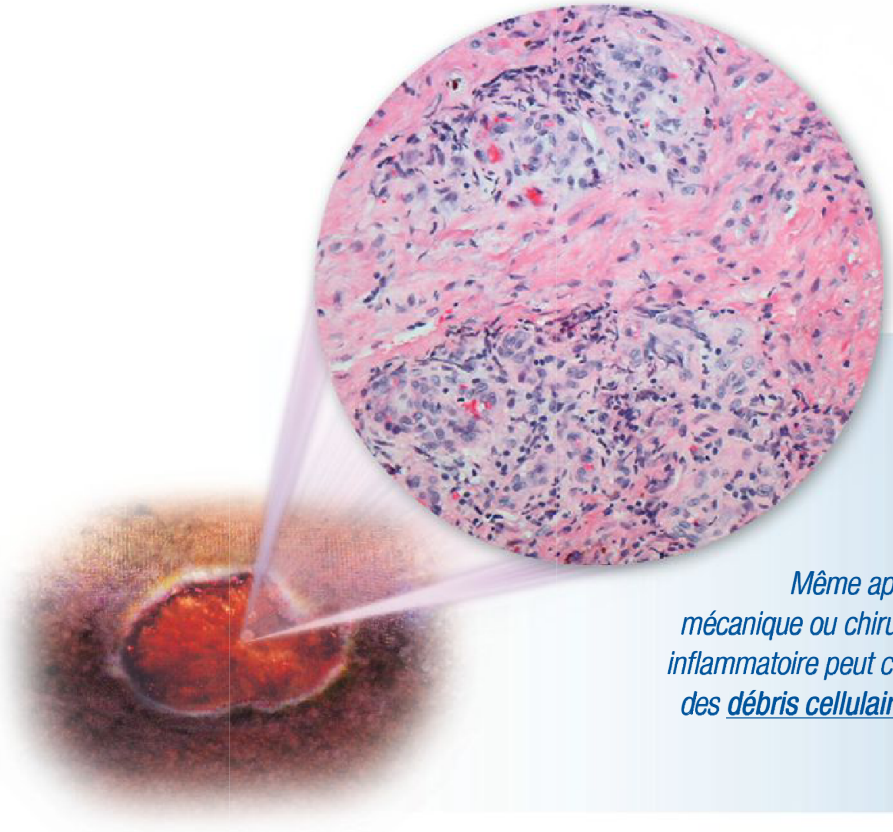

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Occasional slight transient erythema has been noted in surrounding tissue when applied outside the wound. One case of systemic hypersensitivity has been reported after 1 year of treatment with collagenase and cortisone.

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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.

Product monograph available upon request.

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DESCRIPTION : Santyl® (collagénase) onguent est un agent de débridement topique stérile enzymatique qui renferme 250 unités de collagénase par gramme de pétrolatum blanc U.S.P. L'enzyme collagénase est dérivée de la fermentation de *Clostridium histolyticum* possédant le pouvoir unique de digérer de manière sélective le collagène aussi bien naturel que dénaturé qui lie les fibres nécrosées à la surface de la plaie.

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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ensure they have adequate training to perform this procedure, and that the procedure falls within their scope of practice.

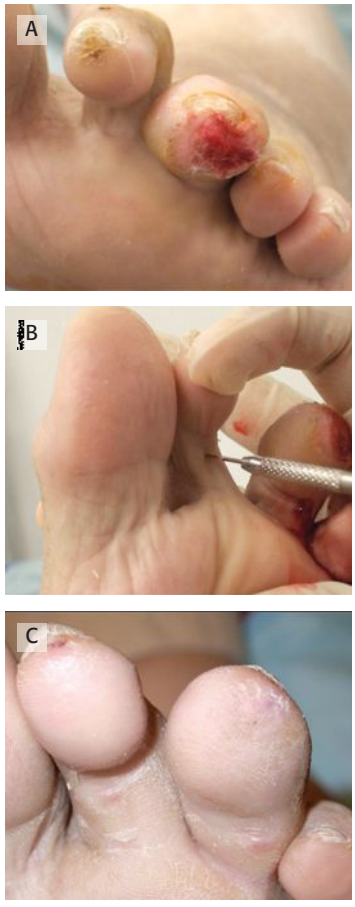
Achilles tendon lengthening

People with diabetes undergo structural changes in the Achilles tendons. These lead to changes in the function of tendons that may contribute to limited dorsiflexion/plantar flexion in the ankle joints. This is called ankle equinus.[23,24] Limited range of motion in the ankle joints may result in early heel rise and prolonged and excessive weight-bearing stress under the metatarsal heads, leading to increased forefoot pressure.[25,26] Others have suggested that the relationship between ankle equinus (dorsiflexion <5 degrees) and forefoot plantar pedal pressure is significant, but that ankle equinus plays only a limited role in causing forefoot pressure.[27,28]

Proponents of ankle equinus as a major factor in forefoot ulceration have shown that Achilles tendon lengthening to treat recurrent forefoot ulcerations increases the range of motion of the ankle joint from 0 to 9 degrees,[27] consequently reducing forefoot pressure. However, after 7 months—when plantar flexor/muscle power returns to preoperative levels, while range of motion in the ankle joint remains at the post-treatment level—plantar pressure returns to the level preoperatively recorded.[29,30]

Achilles tendon lengthening for the treatment of neuropathic ulcers causes a temporary reduction in forefoot pressure associated with changes in plantar flexor power, rather than ankle motion during gait; thus, it appears that the reduction in forefoot pressure is associated with changes in plantar flexor muscle power rather than ankle motion during gait. In addition, a study by Maluf et al. showed that by increasing range of motion in the ankle joint, rear foot pressure remained permanently increased while forefoot pressure was only temporarily decreased.[30] This may lead to a significant complication (i.e. increased risk of heel ulceration), which has been recorded in 15% of patients treated with Achilles tendon lengthening.[29] Thus, there are strong arguments against tendon Achilles lengthening for the treatment of forefoot foot ulcers.

FIGURE 2



Percutaneous flexor tenotomy. (A) Claw toe with apical ulceration. (B) Performing percutaneous flexor tenotomy. (C) Complete healing of the apical ulcer (3 weeks post-operatively).

Digital surgery

Digital surgery (hammer-toe correction, bunionectomy) can reduce bony prominences and ensure a better fit between footwear and the foot, thus preventing ulceration. Such offloading procedures have the greatest benefit in young, healthy, complication-free (i.e. without vascular disease or neuropathy) people with diabetes.[31] In older patients with diminished general health, the risk of complications from the surgery may outweigh the benefits.[32]

Percutaneous flexor tenotomy for claw-toe deformity

Claw-toe deformities are common in those who have had diabetes for more than 10 years.[33] Claw toe is defined as hyperextension of the metatarsophalangeal joint and flexion of the interphalangeal joint. This deformity may cause ulceration at the tip of the toe due to pressure, or at the dorsum of the proximal interphalangeal joint from rubbing against footwear.[34] Percutaneous flexor tenotomy may be performed to release the long flexor tendon of the toe (Figure 2).[35]

A retrospective study from 1 Canadian hospital demonstrated complete healing of all ulcerations and correction of the deformity with percutaneous flexor tenotomy.[36] Of the 34 toes reviewed, all had apical ulcers and 3 were complicated by osteomyelitis. Most ulcers healed within 3 weeks of percutaneous flexor tenotomy and those with osteomyelitis healed within an average of 8 weeks. There were no significant complications and the average length of follow-up was 13 months. Percutaneous flexor tenotomy can be considered an effective method for the management of claw-toe deformity and ulceration.[36]

Silicone injections

A randomized controlled trial injected silicone into diabetic feet at the metatarsal heads to increase tissue thickness and decrease peak plantar pressure. Although this proved effective in the short term, at 24-month follow up the tissue thickness area and plantar pressure had returned to pre-injection levels.[37]

Charcot foot

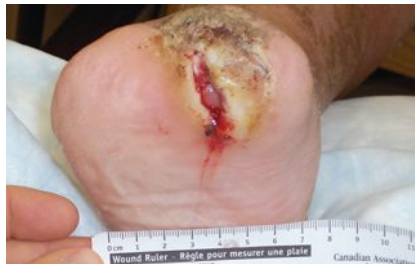
Surgical reconstruction of the Charcot foot is extremely valuable for patients with recurrent ulceration (with or without infection), severe instability or severe deformities that cannot be managed with footwear or braces.[20,38] The goal of this surgery is to stabilize and align the foot, thereby allowing patients to wear shoes and braces.[20] However, the risk of adverse events is high.

Charcot foot surgeries can be challenging and have frequent complications. Proper patient selection is important for limb salvage.[20]

Achilles tendon lengthening with plantar exostectomy

A chronic Charcot neuroarthropathy commonly represents as a rocker-bottom foot, in which the midfoot bones of the arch collapse and subsequently weight bear in areas that are unaccustomed to the high pressures. By combining Achilles tendon lengthening with removal of the bony prominences, a plantigrade foot with reduced plantar foot pressures may be produced.[20,38] A stable foot structure is necessary for this procedure.

FIGURE 3



A transmetatarsal amputation demonstrates pressure distribution issues that can often occur if supportive orthotic devices are not fitted appropriately.

Arthrodesis

Arthrodesis is a process of bony fusion that is carried out after ulcerations have healed.

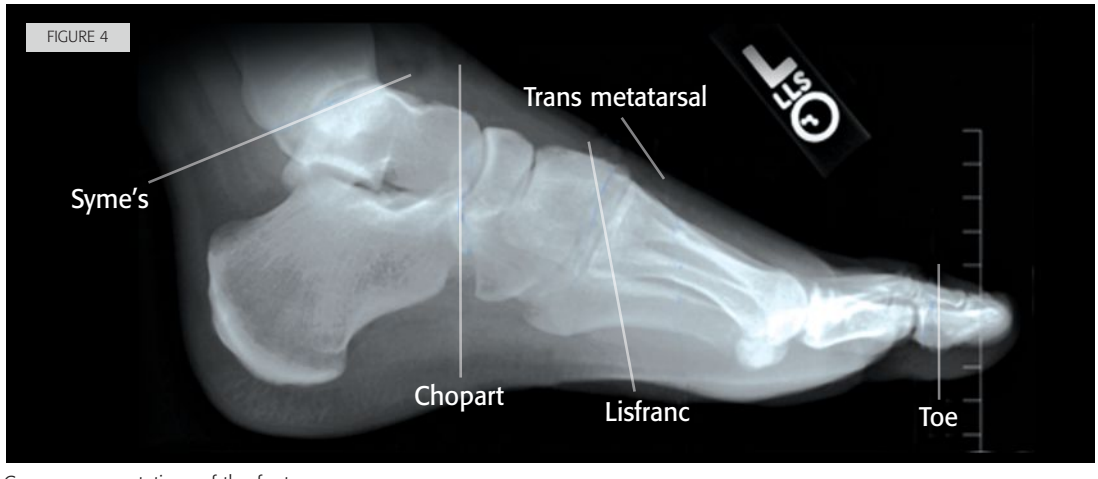
The choice of internal or external fixation depends on the quality of the bone.[20,39]

Amputation

The major indications for therapeutic amputation are trauma, ischemia, malignancy and infection.[40] In chronic wound situations, intractable pain and patient choice weigh heavily in the decision to proceed with amputation once limb salvage options have been exhausted. Surgical removal of portions of a foot or a total below-the-knee amputation may be required when best practice wound therapy is unable to close a foot wound. In these cases, minor amputations may be perceived as victories, as the tissue damage requiring removal will have been restricted.[41]

The surgical goal of amputation is to obtain a balance between wound healing and a functional limb. As with all limb salvage techniques, the arterial supply is directly associated with postoperative outcomes.[42] Adequate viable soft tissue coverage of the surgical wound is imperative to protect and cushion during eventual mobilization of the residual limb. As increasing amounts of the foot are removed, there is a corresponding increase in plantar pressures and instability due to altered biomechanics. These must be supported with appropriate foot wear and customized orthotics (Figure 3).

FIGURE 4



Common amputations of the foot.

TABLE 4

"Amputation cascade" of the foot

Level of amputation	Bony structures involved
Toe(s)	Part or all of the phalanges
Ray(s)	All of the phalanges and part or all of the respective metatarsal
Transmetatarsal	Through all of the metatarsals
Lisfranc	Disarticulation at the tarsometatarsal joints
Chopart	Disarticulation through the talonavicular and calcaneocuboid joints
Syme's	Disarticulation of the talus and tibia/fibula, with retention of the calcaneal fat pad

Although preoperative planning is essential for amputations, the exact level of amputation may be dictated intraoperatively due to sinus tracts, necrotic or infected tissue and skin flap bleeding. Common amputation levels are shown in Figure 4 and Table 4. Lisfranc and Chopart amputations have minimal if any functional benefits over Syme's amputation, despite preserving more of the foot,[43] and frequently result in recurrent ulcerations; in general, these are avoided. Syme's amputation is suitable for patients with diabetes; however, below-the-knee amputation is often favoured due to greater prosthetic options.

Most patients with diabetes and partial foot amputations require foot orthotics at a minimum to prevent further complications. Postoperatively, higher-level amputations are optimally coupled with preprosthetic rehabilitation planning.

The partial loss of a lower limb represents a major change in a person's life, but patients should be encouraged to approach amputation as the beginning of a new phase of life and not as the culmination of previous treatment failures.[44] Pain relief and the removal of a nonfunctional limb may greatly enhance quality of life.

Conclusion

As a component of care, surgery provides an effective method of addressing diabetic foot complications to offload, revascularize, manage infection and, ultimately, improve the quality of life for people with diabetes.

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Vivre avec une plaie chirurgicale…

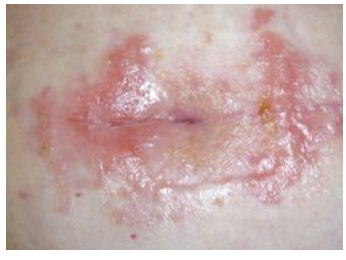
une réalité qui coûte cher à tous, mais qui favorise le développement des connaissances

PAR
Maryse Beaumier
MSc Inf

L'art et la science du traitement des plaies sont aussi vieux que la profession médicale elle-même. Le premier document sur les soins des plaies, un papyrus découvert par Edwin Smith au XIX[e] siècle, date de 3000 ans avant Jésus-Christ[1-3]. Selon Kane[1], les pansements interactifs et bioactifs d'aujourd'hui sont l'aboutissement de l'évolution de la pensée scientifique et de l'expérience du traitement des plaies de guerre entre le Moyen Âge et l'ère moderne. Les soins des plaies sont devenus vraiment omniprésents et importants dans le système de santé[3].

Une plaie est définie comme une rupture de la structure anatomique normale de la peau et de sa fonction[4]. Après une blessure, il est essentiel que l'hémostase soit rapide pour prévenir l'invasion bactérienne et rétablir le plus vite possible la fonction du tissu lésé[5]. Jones et ses collaborateurs ajoutent que la cicatrisation d'une plaie est un processus complexe qui comporte quatre étapes : hémostase, inflammation, granulation et épithélialisation. Une plaie devient chronique quand au moins une des étapes du processus de cicatrisation est inadéquate, trop longue ou inefficace[5].

FIGURE 1
Plaie de fermeture d'iléostomie après 6 semaines



Voici le témoignage d'une femme de 42 ans qui souffre de la maladie de Crohn. Elle a subi une iléostomie temporaire qui lui a causé beaucoup de difficultés et qui a exigé l'intervention de plusieurs professionnels de la santé de divers services. La fermeture de la stomie devait selon le chirurgien être simple : quelques visites aux services courants du CLSC (Centre local de services communautaires) pour des pansements, puis reprise des activités normales en une à trois semaines. Mais les semaines passent, les visites aux services courants se multiplient et, sept semaines plus tard, la plaie exige toujours un nettoyage avec une solution saline à 0,9 % et un pansement (deux compresses de coton maintenues en place au moyen de ruban adhésif chirurgical). Le pansement est changé tous les deux jours. À plusieurs reprises, la patiente fait part au personnel soignant de ses inquiétudes relativement au délai de cicatrisation, mais on lui répond toujours que les choses ne vont pas trop mal et que la plaie devrait se cicatriser sous peu.

« Il est très inquiétant pour le patient de sortir de l'hôpital alors que sa plaie est ouverte (et coule), car il n'y connaît rien. Il faudrait lui expliquer en gros de quelle manière et en combien de temps sa plaie va guérir. Quand on se rend régulièrement aux soins courants du CLSC, on n'est bien sûr pas toujours traité par la même personne : une infirmière nous dit une chose et, deux jours plus tard, une infirmière différente nous dit autre chose. Et quand on lui pose une question, l'infirmière dit souvent qu'elle ne peut pas répondre parce quelle ne connaît pas le dossier, ce qui n'est pas une chose rassurante à entendre de la personne qui vous soigne ce jour-là! Dans mon cas, après six semaines de visites régulières aux soins courants, personne ne trouvait que ce n'était pas normal que ma plaie coule toujours. J'ai dû faire appel à mes contacts pour voir quelqu'un qui connaissait très bien le soin des plaies et, à partir de ce moment, j'ai reçu de bons soins. Ma plaie a fini par guérir. Si j'avais été une personne démunie, âgée ou un peu moins débrouillarde, il aurait probablement fallu beaucoup plus longtemps pour que ma plaie guérisse. »

Maryse Beaumier est professeure en soins de plaies au Département des sciences infirmières à l'Université du Québec à Trois-Rivières et étudiante au doctorat en santé communautaire à l'Université Laval.

La patiente et son conjoint ont suivi leur intuition et consulté une infirmière spécialiste du soin des plaies connue pour se conformer aux pratiques exemplaires. Cette infirmière suit d'aussi près que possible les recommandations sur la prévention et la gestion des plaies chirurgicales ouvertes qui ont été publiées en 2010 par l'Association canadienne du soin des plaies (figure 2)[6].

La première recommandation pour les plaies chirurgicales ouvertes devenues chroniques consiste à procéder à une évaluation holistique afin d'identifier les facteurs susceptibles d'affecter la cicatrisation des plaies chirurgicales au cours des phases préopératoires, intraopératoires et postopératoires[6]. Dans le cas clinique qui nous intéresse, la patiente souffre de la maladie de Crohn et, au fil des années, un psoriasis s'est installé chez elle. Au cours de la dernière année, elle a aussi été traitée par l'infliximab. Elle ne fume pas, est sportive et est en généralement bonne santé. Elle ne prend aucun médicament au moment des soins de la plaie chirurgicale ouverte. La fermeture de stomie s'est déroulée sans complication. Toutefois, depuis le début de la phase postopératoire, soit sept semaines, on ne lui fait que des pansements secs après nettoyage de la plaie avec une solution saline à 0,9 %.

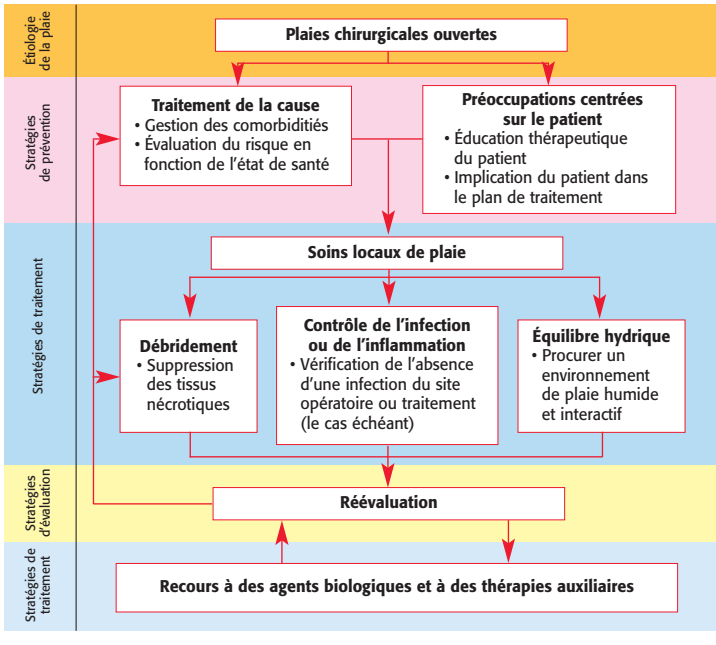
Dans un deuxième temps, on recommande d'élaborer un plan de traitement pour éliminer ou réduire les facteurs susceptibles d'affecter la cicatrisation des plaies chirurgicales au cours des phases préopératoires, intraopératoires et postopératoires[6,7]. Pour cette deuxième recommandation, il va de soi qu'il faut suivre les Recommandations des pratiques exemplaires pour la préparation du lit de la plaie : Mise à jour 2006[7]. En outre, notre patiente s'alimente bien, est active et a un mode de vie sain, ce qui favorise la cicatrisation de la plaie.

Pour l'élaboration des plans de traitement, on recommande en outre d'intégrer le patient, sa famille et/ou la personne dispensant les soins au même titre que les membres de l'équipe soignante[6]. Notre patiente reçoit un appui remarquable de son conjoint. Tous deux ont compris la quatrième recommandation et y souscrivent : éduquer le patient, sa famille et/ou la personne dispensant les soins afin que la cicatrisation des plaies chirurgicales soit optimale[6]. La patiente est très réceptive aux conseils qu'on lui donne et suit à la lettre les recommandations des intervenants. On travaille à contrer l'insécurité et la tendance à l'anxiété de la patiente pour qu'elle reprenne ses activités normales quand l'évolution de la plaie lui permettra de le faire.

Les recommandations 5 à 9 portent sur les soins locaux de la plaie : évaluer la plaie chirurgicale et consigner toute observation selon une approche normalisée; débrider les tissus nécrotiques de la plaie chirurgicale; déterminer l'absence d'une infection du site opératoire ou, le cas échéant, la traiter; maintenir un équilibre hydrique optimal au niveau de la plaie pour favoriser la cicatrisation, en choisissant un pansement conçu pour la cicatrisation des plaies chirurgicales en phase aiguë et chronique; et déterminer l'efficacité des interventions puis aviser si la cicatrisation n'intervient pas dans les délais prévus et évaluer les lèvres de la plaie et le rythme de cicatrisation pour déterminer si l'approche de traitement choisie est optimale[6]. Dans le cas qui nous intéresse, la réépithélialisation a été trop rapide par rapport à la granulation, ce qui a contribué à la formation d'un sérome puis d'un abcès. Il y avait un écoulement verdâtre depuis quatre semaines. La solution saline à 0,9 % et les compresses de coton n'ont aucune propriété antibactérienne. L'ouverture de la plaie était insuffisante pour l'insertion d'une mèche. Le chirurgien a pratiqué une nouvelle incision au site de la plaie, puis on a utilisé un pansement interactif et antibactérien, qui a été remplacé deux fois à trois jours d'intervalle. Un corticostéroïde a été appliqué autour de la plaie pour traiter le psoriasis. À la troisième visite, l'écoulement avait pratiquement cessé, l'épithélialisation avait repris et il y avait une bonne granulation du fond de la

FIGURE 2

Étapes de la prévention et de la gestion des plaies chirurgicales ouvertes[6]



plaie. Comme la cicatrisation optimale de la plaie se fait du fond vers les bords, il faut s'assurer que la plaie demeure suffisamment ouverte jusqu'à ce que la granulation soit complète, d'où l'importance de toujours bien combler les cavités.

Chez notre patiente, l'utilisation de pansements bioactifs et de traitements auxiliaires (recommandation 10) n'a pas été nécessaire en raison du bon potentiel de cicatrisation[6]. Comme la plaie était peu complexe et que le potentiel de cicatrisation était bon, la collaboration entre le médecin et l'infirmière a été suffisante; la recommandation 11 (reconnaître que la cicatrisation des plaies chirurgicales nécessite une approche d'équipe) ne s'applique donc pas à ce cas clinique, mais devrait être privilégiée dans les services de santé[6]. C'est aussi le cas de la dernière recommandation, qui est de mettre en œuvre un programme de surveillance du site opératoire sur plusieurs milieux cliniques[6].

Les soins des plaies chroniques représentent un défi clinique de taille, surtout pour les professionnels des soins à domicile[8]. Les infirmières en santé communautaire sont les travailleurs de la santé que les patients qui ont une plaie voient le plus souvent[9]. Ces infirmières ont des connaissances et des aptitudes variables parce que le soin des plaies est un domaine en émergence. Les infirmières, tout comme les autres membres de l'équipe soignante, y compris le médecin, ne reçoivent aucune formation sur le soin des plaies, ce qui fait que l'écart entre les connaissances et la pratique s'élargit, au détriment des patients. La situation est regrettable, car l'amélioration des connaissances sur le soin des plaies en pratique courante pourrait améliorer la qualité de vie des patients et réduire l'utilisation du système santé et les coûts, ce qui est important compte tenu des pénuries et des restrictions budgétaires actuelles.

Cinq types de facteurs sont susceptibles d'affecter les plaies complexes ou récalcitrantes : les facteurs liés au patient, les facteurs liés à la plaie, les facteurs liés au professionnel de la santé et les facteurs liés aux ressources et au traitement[9]. Dans ce modèle des facteurs prédicteurs de la cicatrisation des plaies de Moffat et Vowden[10], les compétences et le savoir sont des éléments essentiels à la bonne cicatrisation des plaies. Par conséquent, il est maintenant plus qu'urgent

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que le soin des plaies soit intégré au programme de formation des divers professionnels de la santé concernés, surtout les infirmières, étant donné que le soin des plaies est un acte professionnel qui leur est réservé au Québec[11,12].

Le vécu d'une patiente présentant une plaie chirurgicale ouverte confirme tout le travail qu'il y a à faire dans le domaine du soin des plaies. Tous les intervenants n'ont pas toujours les connaissances scientifiques voulues, comme dans beaucoup d'autres domaines. Mais une chose est certaine : il faut tenir compte de ce que disent les patients pour que les soins évoluent et pour prendre conscience que nos interventions, toutes déterminantes qu'elles soient, peuvent être nuisibles si nous manquons de connaissances.

Remerciements

Je remercie très sincèrement madame Luce Langelier de son témoignage. Elle s'est prêtée aux soins avec beaucoup de courage. Son compagnon de vie, Guy Tremblay, mérite aussi une reconnaissance pour ses grandes capacités de soignant.

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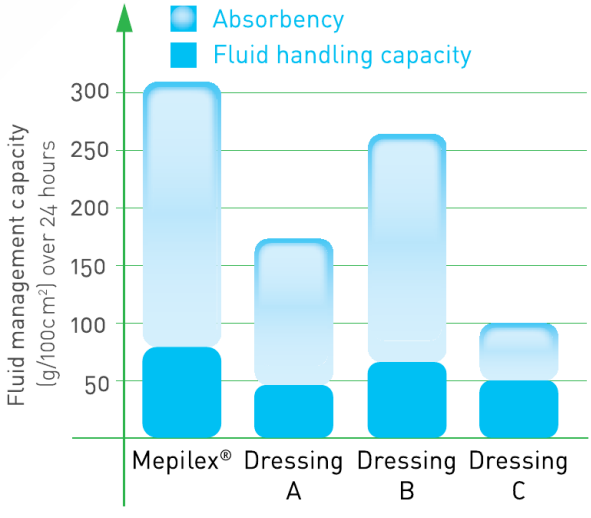

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