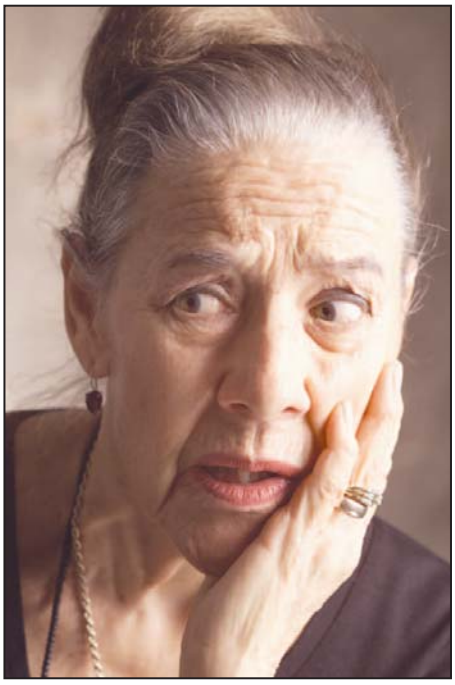
Step-by-Step Guide to Wound Care



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Treating chronic wounds requires a lot of the clinician's time and can be very costly. Therefore, it is important to establish a rigorous treatment strategy to achieve quick healing.

A chronic wound is defined as a loss of substance or breakage of the skin that does not heal spontaneously. In practice, this means wounds that have not healed after six weeks of treatment. Patients often have these wounds for several months before they seek medical attention. There are three main types of chronic wounds: venous ulcers, diabetic ulcers, and pressure ulcers. Mixed ulcers also occur in patients suffering from both arterial and venous problems.

Samantha's wound

Samantha, 72, consults you about a wound on the distal third of her right leg, just above the medial malleolus. The wound measures 4x6 cm and it has been developing for six months. The extremity is edematous. The wound is superficial, well demarcated, and not very painful. There is no granulation tissue and the wound does not seem to be healing. How should you treat this wound?

In this article:

What delays wound healing?

Normally, wounds heal according to a synchronized process consisting of three successive phases.[1] First, in the inflammation phase, hemostasis is immediately followed by neutrophils moving into the wound. Then the macrophages arrive as a reinforcement to help reduce infection. The macrophages also give the start signal for the proliferation of fibroblasts and deposit of collagen.

Table 1
Factors that delay wound healing

Local factors	
Reduction in growth factors	
Edema – ischemia	
Low oxygen tension	
Infection	
Regional factors	
Arterial insufficiency	
Venous insufficiency	
Sensory, motor, or autonomic neuropathy	
Systemic factors	
Metabolic disorders (*e.g.*, hyperglycemia)	
Malnutrition (nutritional deficiencies)	

The second stage of healing is the proliferation phase, marked by the appearance of granulation tissue and angiogenesis.

The final remodelling phase involves the formation of a new epithelium and the marshalling of collagen fibres to increase tensile strength. In chronic wounds, the healing process is interrupted in the proliferation phase, where healing stagnates for a long time. The presence of inflammatory cells may even damage healthy tissues. Three types of factors delay wound healing (Table 1).

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What are the general treatment principles?

The first step

The first step is to make an accurate etiologic diagnosis. The clinician must determine if it is a venous, arterial, or pressure ulcer. The medical history and physical examination will give an idea about the origin of the wound. Its location and dimensions, the quantity and type of exudate, the presence or absence of granulation tissue, signs of infection, necrotic areas, edema, and the condition of the area around the wound are all indicators that will help identify the cause and determine the treatment.

It is essential to treat any underlying medical problems. Optimal control of diabetes and nutritional status is necessary. You must evaluate the possibility of stopping or reducing certain toxic drugs. You must make a detailed examination of the vascularization and neurologic status of the affected extremity.

After a detailed examination, you can start to treat the wound. The basic treatment is the same, regardless of the type of chronic wound: debride the devitalized tissues, clean the wound, control infection, and cover the wound with a moist interactive dressing.

Use one of the following four techniques for debridement: surgical, autolytic, enzymatic, or mechanical.[2] If there is a lot of necrotic tissue, or if intervention is urgent, the surgical approach using scissors and a scalpel is the most appropriate. Autolytic debridement is promoted by modern moist interactive dressings. In a moist environment, phagocytic cells and proteolytic enzymes in the wound clear the dead tissue by liquefaction. Macrophages can then digest this soluble tissue. It is a slower process, but often very appropriate for this type of wound. Enzymatic debridement is another option if there is a moderate amount of necrosis. However, the area around the wound must be protected. Wet-

to-dry mechanical debridement should be avoided because it is not selective and delays healing by destroying the newly formed granulation tissue.

The second step

Next, cleanse the wound with saline (NaCl, 0.9%). Most antiseptics are cytotoxic and should be avoided. Certain signs indicate an infection. A change in the exudate is often an early sign of infection. The exudate may become more copious, purulent, or foul-smelling. Other signs, such as edema, pain, local heat, and fever, indicate an infection. In these cases, it is essential to start antibiotic treatment. Most often, a topical antibiotic will not be enough; you will need to use a systemic antibiotic.

Finally, you must cover the wound with a dressing. Certain rules apply to the dressing's application. Because of the numerous dressings on the market, we need to take a logical, structured approach to avoid falling into the trap of trying something just because it is new.

Modern dressings have been developed based on the principle of moist interactive healing. The first study on this subject, published by Winter in 1962, and confirmed many times since, determined that chronic wounds heal faster in a moist environment than in a dry environment.[3] Hence, many dressings are developed to maintain moisture over the wound (Table 2).

Because the ideal dressing does not exist, certain criteria are used to select the right dressing at the right time. First, it is contraindicated to put an occlusive dressing on an infected wound. We choose a dressing based on its absorption ability to keep the wound moist and the skin around it dry. It is quite common to have to change the type of dressing during treatment. It is impossible to memorize all the dressings on the market, but more useful to remember the categories of dressing. When a particular dressing no longer meets the treatment plan requirements, we can change the category to achieve the desired objectives (Table 3).

Table 2
The ideal dressing

Absorbs the exudate and necrosis
Maintains a high degree of moisture
Is impermeable to liquids
Provides thermal insulation
Protects against infection
Controls odours
Is non-toxic and non-allergenic
Is easy to use

Table 3
Main categories of moist interactive dressing*

Absorbent dressings	Foam (Allevyn®, Lyofoam®, Aquacel®)
Calcium alginate	Algoderm®, Kaltostat®, Melgisorb®, Algisite®
Hydrocolloids	Comfeel®, Duoderm®, RepliCare®, Tegasorb®
Hydrogels	Intrasite gel®, Duoderm gel®
Polimer film dressings	OpSite®, Tegaderm®

*In decreasing order of absorption

Treatments for different types of wounds

Depending on the wound etiology, additional steps, which are just as important as the basic treatment, must be taken.

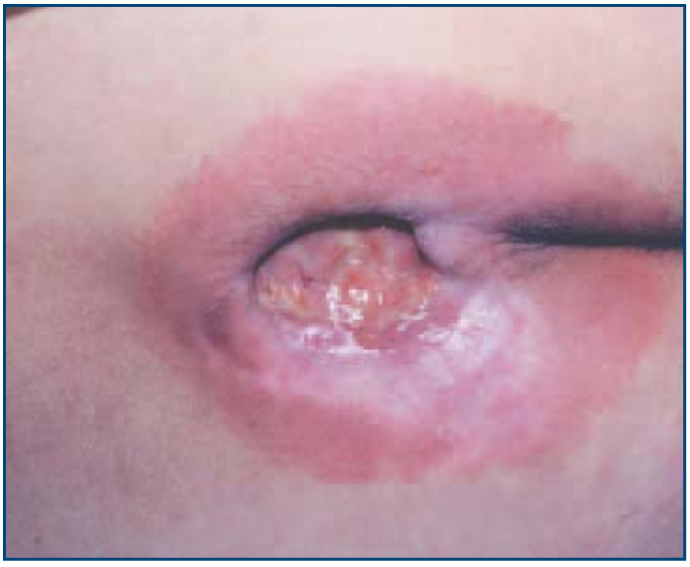

Figure 1. Pressure ulcer on the sacrum.

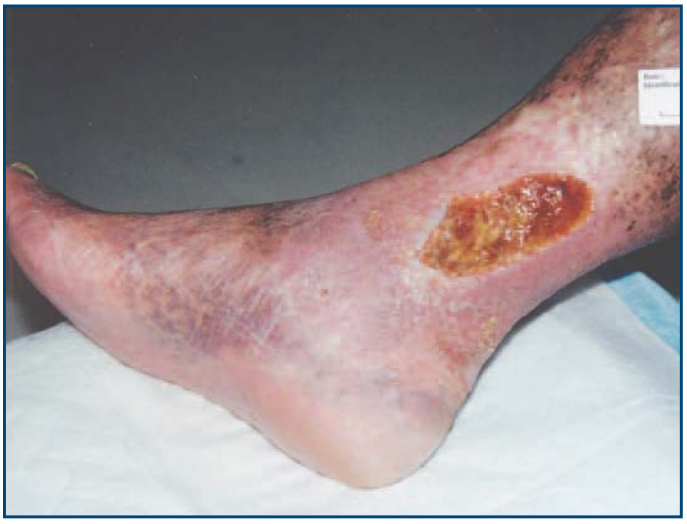

Figure 2. Venous ulcer.

Table 4
Ankle brachial index (ABI)

$$ABI = \frac{\text{Ankle systolic pressure}}{\text{Brachial systolic pressure}}$$

Interpretation	
0.9-1.00	Normal
0.75-0.9	Moderate arterial disease
0.5-0.75	Severe arterial disease
< 0.5	Serious risk of ischemia
< 0.6	Compression therapy contraindicated

Pressure ulcers

Pressure ulcers are caused by pressure on bony protuberances. In about 95% of cases, these wounds are found on the lower part of the body, *i.e.*, on the sacrum, trochanters, malleoli, or heels.[4] Screening patients is essential and can be done using simple tools, such as the Braden scale.[5] Reducing local pressure on the bony protuberances is the basis of treatment. Bedridden patients are turned every two hours. Distribution of body weight can be modified by using cushions, pillows, or orthoses. The head of the bed should not be raised more than 30°, as this would increase the risk of slipping and shearing the skin. Once these measures are well-established, you can apply a moist interactive dressing on the wound (Figure 1).

Venous ulcers

In the case of venous ulcers, the etiologic mechanism is venous hypertension in the lower extremities, which can cause edema, stasis, and ulcers. Thus, the cornerstone of treatment consists of combating the venous hypertension and edema by compressing the lower extremities with multi-layer bandages (*e.g.*, Profore® or SurePress™). These bandages hasten the healing of venous ulcers. Before using them, however, you must make sure there is good arterial circulation.[6] To do so, determine the ankle brachial index (ABI) (Table 4).

An index > 0.6 means that compression can be used without danger. Under the compression bandage, cover the wound with a moist interactive dressing. If there is no infection, this treatment is repeated until the wound heals. Support hose can be worn to prevent recurrences (Figure 2).

Diabetic ulcers

With the aging of the population, the incidence of diabetes and diabetic complications

is increasing. Diabetic ulcers mainly develop on the plantar aspect of the foot, over the head of the metatarsal bone or heel (Figure 3).

Various phenomena contribute to the development of ulcers in patients with diabetes, including sensory, motor, and autonomic neuropathies. Hyperglycemia delays healing. Hyperkeratosis in the area around the ulcer aggravates the problem. Sensory neuropathy can be evaluated with the Semmes-Weinstein monofilament (Figure 4).[7]

If the arterial circulation allows it, the first thing to do is debride the wound surgically. The hyperkeratosis must be completely removed to avoid additional local pressure. It may be necessary to wear an orthosis, cast, or an adapted shoe to reduce the weight on the wound. Strict control of glycemia and cigarette smoking is essential to prevent recurrences. A distal wound on the toe should make us suspect arterial insufficiency (Figure 5).

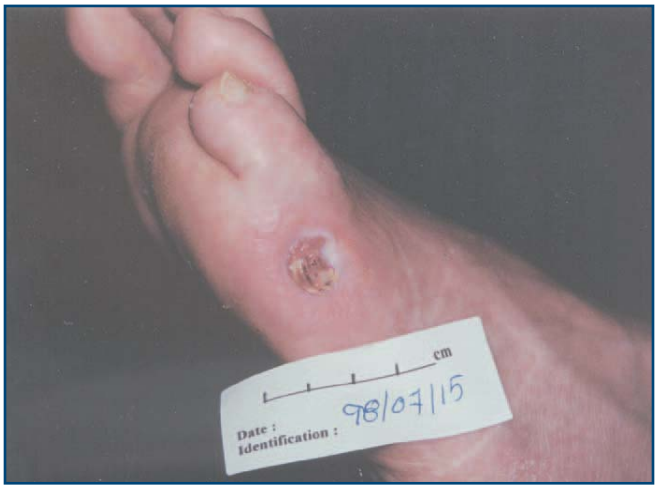

Figure 3. Diabetic ulcer.

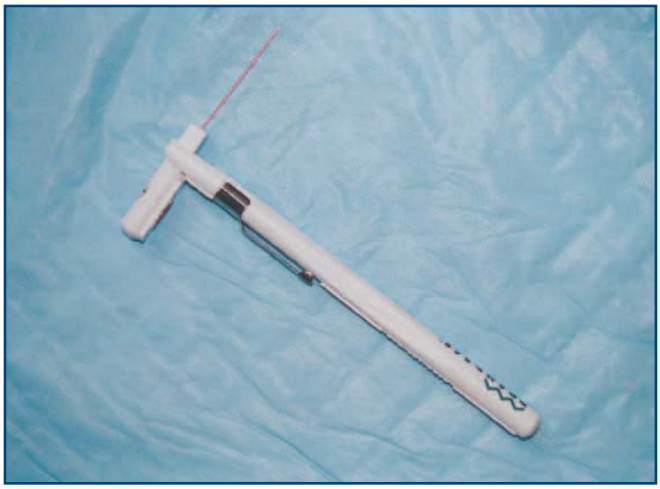

Figure 4. Semmes-Weinstein 10 g monofilament.

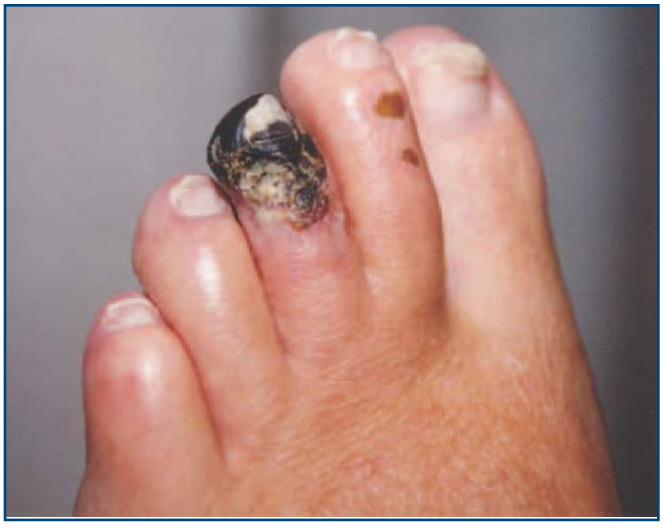

Figure 5. Wound secondary to arterial insufficiency.

Hopelessly chronic wound...

Some wounds do not heal despite the best efforts of the caregiving team. In this case, the clinician

Take-home message

- Treatment in a moist environment hastens the healing of chronic wounds.
- The choice of the type of dressing should be based on the amount of exudate.
- Besides the basic treatment of the wound, an etiologic treatment should be applied.
- An ankle brachial index > 0.6 means that compression bandages can be used to treat venous ulcers.

Wound Healing

Web sites:

1. http://www.ahcpr.gov/clinic/cpgonline.htm
   Very interesting site where you can find: Treatment of Pressure Ulcers, Clinical Guideline Number 15, AHCPR Publication No. 95-0652: December 1994
2. http://www3.sympatico.ca/pm.roy/ (French)
3. http://www.woundheal.com/healing/clinicalPrintVersion.htm

must reassess all the steps described above. Is the diagnosis accurate? Could there be an underlying neoplasy? Is there any excessive local pressure on the wound? Is the arterial circulation sufficient? Have we excluded an infection? Is the debridement adequate? Is the wound being treated in a moist environment? The answers to all these questions should help resolve the problem. CME

References

1. Hunt TK, Hopf H, Hussain Z: Physiology of wound healing. Adv Skin Wound Care 2000; 13(2Suppl):6-11.
2. Singhal A, Reis ED, Kerstein MD: Options for nonsurgical debridement of necrotic wounds. Adv Skin Wound Care 2001; 14(2):96-103.
3. Winter G: Formation of a scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. Nature 1962; 293:193-4.
4. Thomas D: Prevention and management of pressure ulcers. Rev Clin Gerontol 2001; 11:115-30.
5. Braden B, Bergstrom N: Clinical utility of the Braden scale for predicting pressure sore risk. Decubitus 1989; 2(3):44-51.
6. Kunimoto B, Cooling M, Gulliver W, et al: Best practices for the prevention and treatment of venous leg ulcer. Ostomy Wound Manage 2001; 47(2):34-50.
7. Hauck E: Diabetic and general foot care. The Canadian Journal of CME 2001; 13(2):77-85.

Suggested Readings

1. Sibbald RG, Williamson D, Orsted H, et al: Preparing the wound bed—Debridement, bacterial balance and moisture balance. Ostomy Wound Manage 2000; 46(11):14-35.
2. Mégie MF, Tremblay L: De la toile d'araignée au génie tissulaire. Le Médecin du Québec 1999;34 (9):47-53.
3. Ferris FD, Krasner DL, Sibbald RG: Twelve toolkits for successful wound care. Canadian Association of Wound Care, 1999.