How Wounds Heal:
A Guide for the Wound-care Novice

BY Christine Pearson

Christine Pearson, RN, IIWCC, is a wound clinician for Vancouver Coastal Health and has worked in community nursing for the last 25 years. She develops and presents wound education sessions in post-secondary institutions and provides consultation on difficult wounds for physicians, community nurses, student nurses, long-term-care facilities, street clinics and jails.

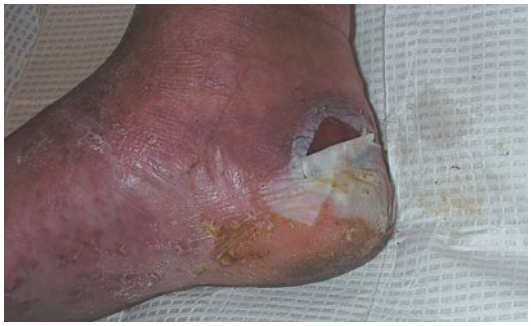
This article covers the basics of how wounds heal and how you can help healing progress in a timely fashion. Words highlighted in red are defined in the glossary at the end of the article. The References and Suggested Reading lists will help you continue your understanding of wound healing.

Although this article is focused on the wound, it is important that readers always remember to look at the whole patient and address patient-centred concerns (see Figure 1); there is a person attached to the wound. Be sure to get a good patient history; assess nutrition, medications, lifestyle (e.g., smoking), pain; and determine adequate blood supply, patient's goals, etc.[1]

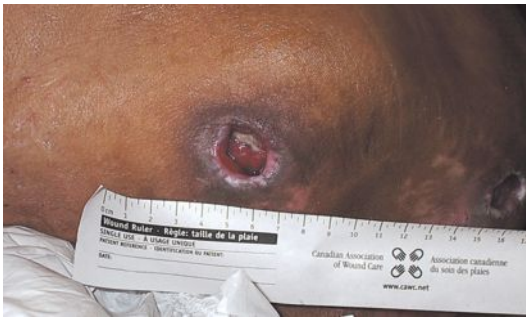
Types of Wounds

The vast majority of wounds are considered *acute* and heal quickly for most people. However, some wounds that do not heal quickly become *chronic* for a variety of reasons. It is important to understand what processes need to occur for a wound to heal so we can identify the obstacles that are hindering healing and implement strategies to overcome them.

When someone has a superficial, or partial-thickness, wound, the wound will heal through the creation of new epithelium from the edge of the wound, as well as from the hair follicles, sweat glands and sebaceous glands, to cover the damage.



Partial thickness

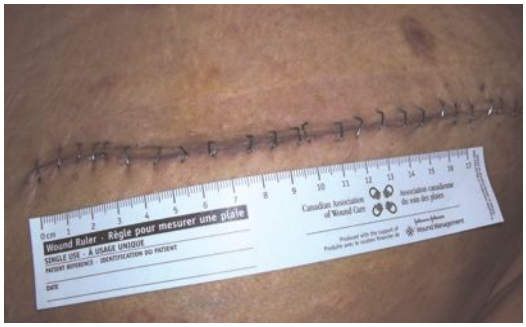


Full thickness

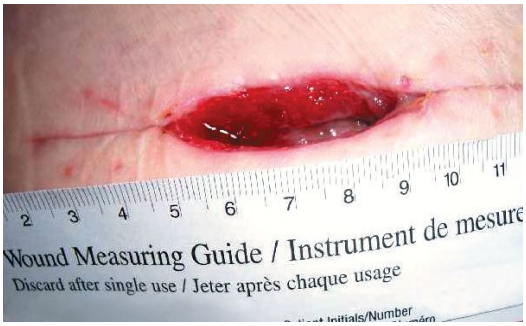
When someone has a deeper, or full-thickness, wound, it isn't as simple. Humans are not like newts. If you cut off a newt's tail, it grows a new one. If we lose significant tissue, we grow granulation tissue to fill the damaged area, thereby losing most of the original components of the dermis such as nerve endings, sebaceous glands and hair follicles.

If the wound edges are sutured or stapled together, it is called *primary intention* or *primary closure*.

If the wound cannot be closed for some reason (e.g., infection), then it is left open to heal by *secondary intention* or *secondary closure*.



Primary intention or closure



Secondary intention or closure

The Phases of Healing

For a wound to heal it must go through certain overlapping phases. These three phases of wound healing are the *inflammatory, proliferative* and *maturation phases*.[2]

The moment a wound occurs, whatever the cause (be it falling off a bike or having surgery), the inflammatory phase starts as the blood vessels constrict and blood cells called platelets form a clot to stop the bleeding (hemostasis). For the next few days, white blood cells called neutrophils and other white blood cells called macrophages migrate to the wound to remove bacteria and start the clean-up of debris (autolytic debridement). Macrophages also send out signals to bring other needed cells, chemicals, proteins and growth factors to the injury site. With all these new cells and activity happening at the wound site, you will find increased redness, heat, swelling, pain and drainage. For the first three to four days this is the normal, natural healing process—not signs of clinical infection. People who mistake this for infection tend to want to needlessly apply topical antibiotics that can lead to antibiotic resistance.

The *proliferative*, or *granulation*, phase occurs over approximately the next two to three weeks (or longer for large wounds). Cells called fibroblasts start off the reconstruction by laying down new collagen fibres and stimulating the growth of new blood vessels *(angiogenesis)*. This highly vascular substance, called granulation tissue, fills the wound. The edges of the wound start contracting and new epithelial cells close the wound if the conditions are just right. A scar has now replaced the open wound.

At this point, we say the wound is *closed*, not healed. The randomly placed collagen that was used to close the wound is not very strong, so during the *maturation phase* it is replaced with a stronger collagen that is laid in a more organized fashion. The scar softens, flattens, and changes colour. This process takes from six months to two years to complete. At the end of the phase the wound is finally healed, but the resulting scar tissue is only about 80 per cent as strong as the original skin.

Factors Affecting Healing

An *acute wound* is one that heals following the timely reparative process described above. A *chronic wound* is one that gets "stuck" in one of the phases and needs help to progress. The most common reasons for a wound becoming chronic are infection or heavy colonization; lack of oxygen delivery to the tissues; presence of debris, slough, or necrotic tissue; repeated trauma or pressure to the area; systemic issues such as diabetes, malnutrition, dehydration or immunodeficiency; and certain medications. You need to identify the reason why the wound is stuck and correct the hindrance where possible.

You can improve healing by optimizing the conditions in the wound. This process is called wound bed preparation (see Figure 1).

The first step in wound bed preparation is to treat or remove the cause of the wound. For instance, if the cause of the leg ulcer is venous insufficiency the patient may need compression wraps or stockings to improve the venous flow, thus reducing the edema.

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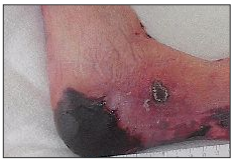
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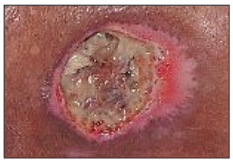

Slough

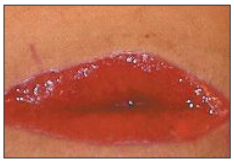
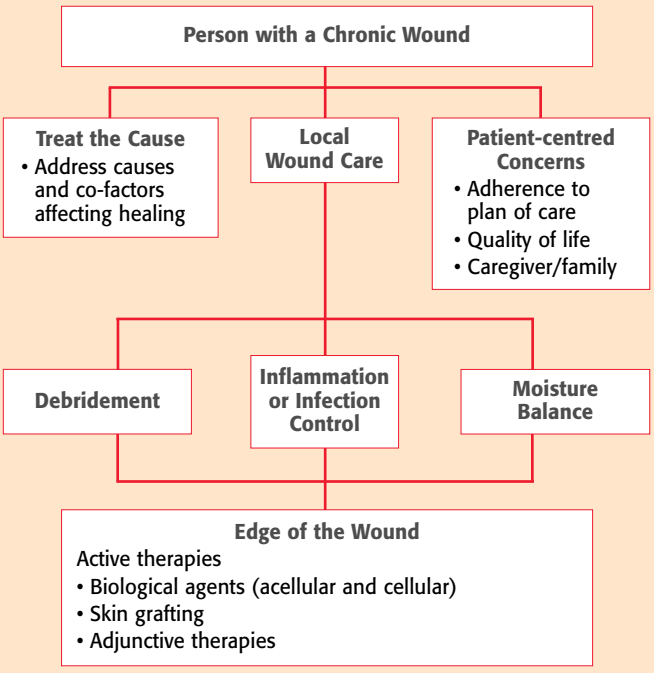

Granulation

FIGURE 1

Preparing the Wound Bed



Sibbald RG, Orsted HL, Schultz GS, et al.[1]

TABLE 1

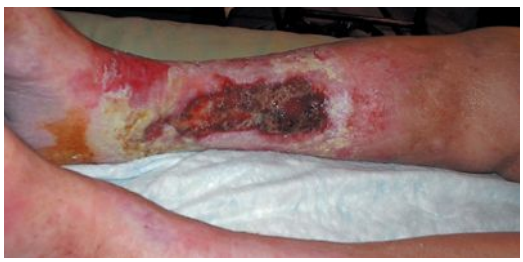
Methods of Debridement

Type	When to use	Advantage	Disadvantage
Surgical • done by a physician, usually in an OR with anesthetic	• grossly contaminated wounds • for large, chronic or non-healing ulcers • large areas of necrosis • exposed vital structure • to prepare for skin grafts or flaps	• most expedient and effective • fast and selective • removes all tissue down to bleeding, which kick-starts the inflammatory phase • reduces wound contamination • faster healing	• painful • difficult to find someone to do it • costly • sterile procedure should be used
Sharp • done by a qualified clinician	• when there is loose slough or eschar • vital structures can be identified	• removes excess dead tissue • fast and selective	• may cause bleeding • does not remove all dead tissue
Autolytic • using products that allow moisture balance in the wound	• painful wounds	• selective and inexpensive • relatively painless • versatile • requires little skill	• slow • not useful with severe infection • can be odorous
Mechanical • pressure irrigation <16psi • wiping with moist gauze (Wet-to-dry dressings are not recommended because they are painful and not selective)[3]	• wounds with moderate to large amounts of debris	• inexpensive	• nonselective • may damage healthy tissue • healing delayed by repeated trauma • usually painful • time-consuming and messy for staff • pressurized irrigation can drive bacteria into deeper tissues
Biological • maggots	• wounds with moderate to large amounts of debris	• fast and selective • removes dead tissue and bacteria • stimulates granulation	• some patients will not want this method • maggots sometimes escape • moderate cost
Enzymatic • none available	• wounds with moderate amount of debris • if type of enzyme matches the type of debris	• selective • moderately fast	• no product available in Canada at this time • moderate cost

The second step is to determine the need for local wound care by assessing the wound bed for debris, infection and moisture balance. The wound bed may have debris, slough or necrotic material. Healthy granulation tissue is needed for optimal healing.

If it is appropriate based on the clinical assessment of the patient (e.g., the patient does not have a bleeding disorder or is on anticoagulants), and there is sufficient blood flow to the area for healing, then this debris needs to be removed (debrided) or healing will be delayed. There are several debridement options available (see Table 1).

Moisture balance in the wound is vital. If the wound is allowed to dry, then the cells are dry; a dry cell is a dead cell, and it cannot be brought back to life. The body will have to grow all new cells to replace the dead ones, therefore slowing healing. In contrast, if there is too much moisture or drainage in the wound it will be like a gushing flood and wash out many of the necessary cells, proteins and enzymes from the wound bed and damage the surrounding skin (maceration).



Maceration

Examine the **edge of the wound** to determine the edge effect. If it is healthy and attached to the wound base, new epithelium will likely spread across the moist wound bed to close the wound. If the edge is unhealthy,

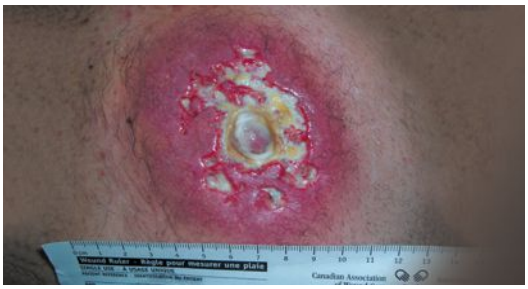
rolled, unattached, scarred, calloused, or macerated, you are unlikely to see epithelialization occurring.[4]

Choosing the appropriate dressing at the appropriate time will allow you to maintain an appropriate moisture balance. For instance, when a wound is heavily exudating you need dressings that are very absorbent such as foams, alginates and combination dressings. As the wound improves and the drainage decreases, you must change the type of dressing used or you could dehydrate the wound. When there is just the right amount of drainage you can maintain the moisture balance with hydrocolloids, acrylics or transparent dressings.

If there is too little moisture in the wound you may be using too absorbent a dressing or a dressing that allows too much moisture to escape such as gauze. Adjust your dressing choice. If that doesn't work, consider using an amorphous gel that will add moisture to the wound. Note, however, that gels can also cause maceration if too much is used.

Check your facility's wound product formulary to familiarize yourself with your product options. Find out what each product is made of (the form) and what it can do for the wound (the function), so you can make the best-informed choice for optimal healing.[5]

A wound with too much bacteria will cause a competition for available oxygen and nutrients: the bacteria will win and the wound will lose. All chronic wounds are colonized with bacteria, and we know that the wound actually needs some bacteria present for optimal healing. Again, we need the right balance—in this case, *bacterial balance*. If the wound is heavily colonized, wound healing will slow or stop. Thorough flushing of the wound with a non-toxic cleanser, such as normal saline, will help reduce the number of bacteria. There are also many topical antimicrobial dressings available that do not promote antibiotic resistance (e.g., cadexomer iodine and silver dressings). Many of the over-the-counter and prescription antibiotic creams and ointments do promote antibiotic resistance. If the bacteria win the competition and have taken over the wound, you have *clinical infection* (increased redness, heat, drainage, purulence, odour, pain, slough and size) and the wound and the host will deteriorate further. This is when systemic antibiotics are needed.



Wound infection related to antibiotic resistance

Over the last few years there has been an increased interest in wounds and healing, which has prompted more clinical research into how wounds heal. Some articles have shown that the wound also needs *chemical balance*. Acute wounds have been found to have many different kinds and large amounts of proteins—called growth factors—that stimulate growth, and not many destroyer cells (matrix metalloproteases). Chronic wounds have been found to have the opposite conditions: few kinds and small amounts of growth factors and too many destroyer cells. To address these conditions and get closer to chemical balance in the wound, products are available that may help (some examples are oxidized regenerated cellulose with collagen dressing, porcine intestinal submucosa dressing and growth factor gels).[6]

continued on page 18

Glossary

Antibiotic resistance: Occurs when bacteria have mutated and are no longer susceptible to the effects of a particular antibiotic.

Collagen: An insoluble fibrous protein.

Epithelial cells: Some of the cells needed to form new skin.

Fibroblast: A cell that secretes proteins and collagen to form a matrix of connective tissue.

Full-thickness wound: Loss of dermis with its blood vessels, sebaceous glands, hair follicles, nerves and possibly deeper tissues; wounds that heal by filling with granulation or scar tissue.

Growth factors: Substances that promote cellular growth

Maceration: A condition that occurs when the outer layer of the epidermis is exposed too long to moisture and separates from the lower layer; the skin appears white and/or wrinkled.

Macrophages: A phagocytic (cell-debris-eating) tissue cell of the immune system.

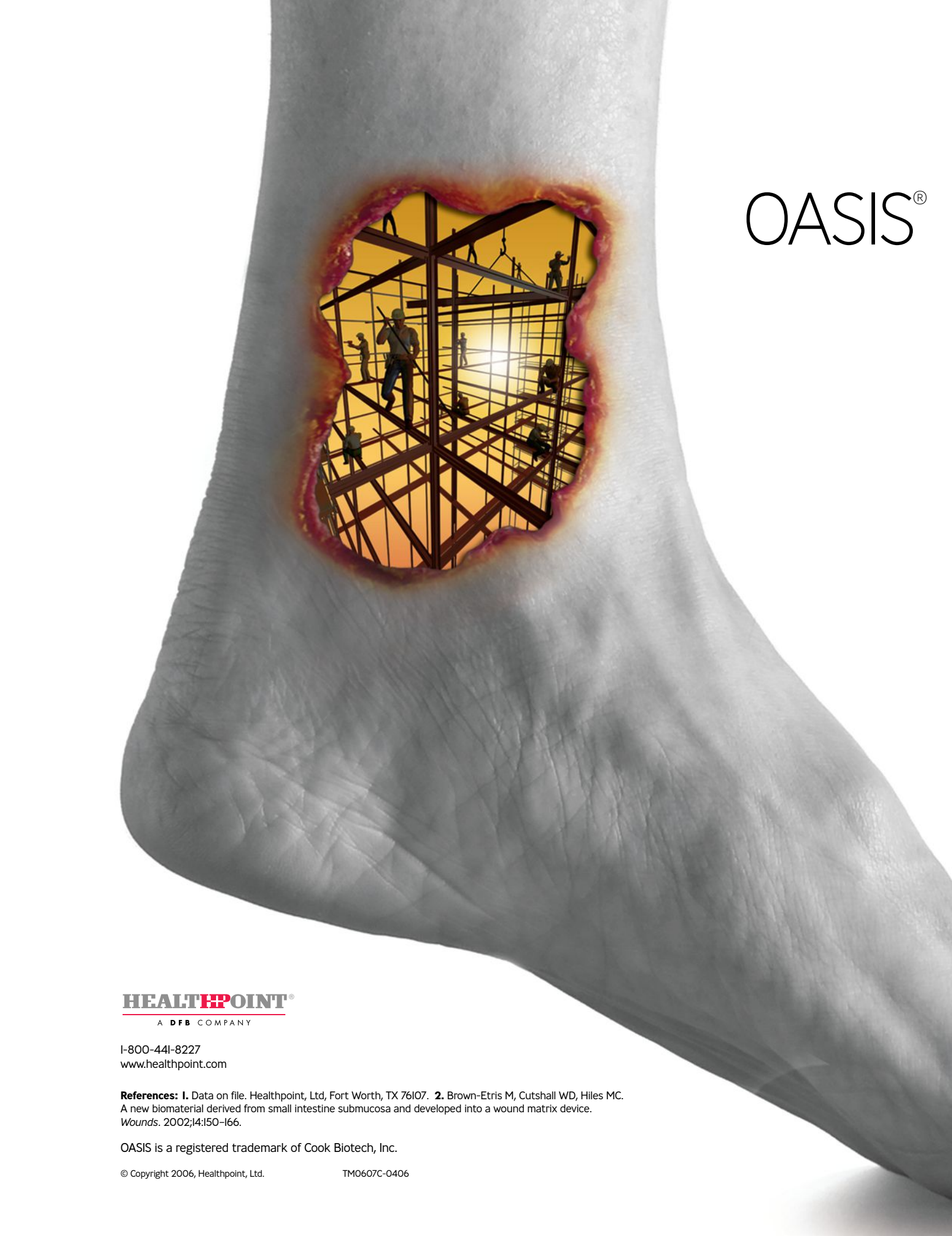
Matrix metalloproteases: Enzymes that break down the protein that holds cells in place.

Neutrophil: A phagocytic white blood cell.

Partial-thickness wound: Loss of epidermis and maybe superficial dermis; wounds that heal by growing new epidermis over the area.

Platelets: A minute disk that is released from the bone marrow into the blood: it assists in blood clotting by sticking to other platelets and to damaged epithelium.

Venous insufficiency: An impairment to the blood flow returning from the feet to the heart.



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References: 1. Data on file. Healthpoint, Ltd, Fort Worth, TX 76107. **2.** Brown-Etris M, Cutshall WD, Hiles MC. A new biomaterial derived from small intestine submucosa and developed into a wound matrix device. *Wounds*. 2002;14:150–166.

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