

Wound healing in 20 questions

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1 What are the different phases of normal wound healing?

The healing of acute traumatic or surgical skin wounds involves a complex and unique process. To understand the general principles of wound healing, it is easier to approach the topic by distinguishing the different successive phases, although keeping in mind that they overlap in time and space (*Figure 1*) [1, 2]. It is also important to understand that these different physiological stages are disrupted in chronic wounds subject to delayed healing.



Figure 1. Diagram of physiological healing.

The first phase following wound formation is **haemostasis** and the formation of a temporary matrix. This phase consists of a succession of cellular and molecular mechanisms that allow not only coagulation to reduce bleeding but also involve multiple cytokines and growth factors that promote collagen synthesis, angiogenesis and even re-epithelialisation.

This is followed by an **inflammation phase** that begins with a primary inflammation stage, involving the recruitment of neutrophils, followed by a secondary inflammation stage during which monocytes are transformed into macrophages. In the first few days, neutrophils play a major role by combating bacterial aggression, helping to break down necrotic tissue and attracting other inflammatory cells. Approximately three days after the injury, macrophages become the primary cells of healing, through their ability to destroy cellular debris and synthesise numerous growth factors that stimulate the next proliferation phase.

Damaged tissue is rebuilt during the **proliferation** phase, along with re-epithelialisation and the formation of the vascular network. This crucial stage takes places during the first few weeks and results in the formation of granulation tissue. Re-epithelialisation is achieved by keratinocyte proliferation from the margins of the injury, which have the ability to migrate within the forming matrix, and by epidermal stem cells located in the "bulge" area of hair follicles (*Figure 2*). Physiological proliferation is achieved through a balance between degradation and synthesis of the extracellular matrix. In pathological scars, this balance becomes imbalanced in favour of degradation, in cases of delayed healing, and in favour of synthesis, in keloid and hypertrophic scars.

1 What are the different phases of normal wound healing?



Figure 2. Skin and stem cells.

Finally, during the **maturation phase** a series of changes occur to components of the extracellular matrix starting about three weeks after the injury and lasting for up to two years, which is why the "final" appearance of a scar can only be assessed after this time. Collagen reorganises and remodels itself according to the traction stresses placed on the wound but it will never have the same intertwining appearance as that of uninjured skin. In addition, sweat glands and hair follicles are not reconstructed, which is why healed skin is never identical to the original skin. Finally, since melanocytic proliferation occurs at a later stage, the scar will initially appear paler than the rest of the skin.

2 What are the main factors involved in delayed wound healing?

With all wounds, especially chronic wounds, factors that may cause delayed healing must be taken into consideration. Multiple factors can be involved including medicinal (see question 3), nutritional, vascular, infectious, metabolic, psychiatric, mechanical, etc., factors.

These factors are divided into **general factors** and **local factors**, and their management is key to achieve satisfactory healing (*Table 1*).

General factors	Local factors
 Malnutrition Diabetes Arterial disease Venous insufficiency Anaemia Kidney failure Immune deficiencies Medicines (see question no. 3) Smoking 	 Location of the wound (opposite joints, etc.) Local care protocol Application of allergenic topical products Infection Maceration Presence of foreign bodies Quality and type of sutures

Table 1. Factors in delayed healing.

3 Which medicinal products affect wound healing?

Some medicinal products, due to their mechanism of action, interact with some of the physiological factors involved in wound healing and may slow down the process. Such medicines include:

- cancer drugs which by altering the capacity for cell division and by their immunosuppressive properties, can slow down the healing process;
- **systemic corticosteroid therapy** which at high doses inhibits fibroblast proliferation, collagen synthesis and epithelialisation;
- **immunosuppressants**, **non-steroidal anti-inflammatory drugs**, which, due to their immunosuppressive properties, can also slow down the healing process.

Anticoagulants may alter the haemostasis phase of healing but do not cause delayed healing.

4 What is the impact of topical corticosteroids on wound healing?

Topical corticosteroids play a key role in the treatment of hypergranulating wounds (*Figure 3*). They reduce exuberant granulation tissue and its epidermalization, which is otherwise impossible for mechanical reasons. Although often feared by practitioners, they do not cause infectious complications in chronic wounds and can be a valuable aid.

They do delay healing, unlike systemic corticosteroids.

However, at high doses and with prolonged use (e.g. in the treatment of autoimmune bullous dermatoses), they can cause **skin atrophy**, resulting in skin tears at the slightest trauma.



Figure 3. Hyper-granulation ulcer requiring the application of topical corticosteroids.

5 Is there a difference between wound healing in acute and chronic wounds?

A **chronic wound** is a wound that takes a prolonged time to heal (*Table 2*). A wound is considered as chronic after it has lasted for six weeks. The three most common types of chronic wounds are leg ulcers (*Figure 4*), pressure sores (*Figure 5*) and foot wounds in diabetic patients, also known as plantar sores (*Figure 6*) [3]. Caring for such wounds is a major public health issue costing almost \in 1 billion each year in France.

	Acute wound	Chronic wound
Process	Traumatic	Pathological
Healing time	\leq 6 weeks	> 6 weeks
Delayed healing factors	Few or none	Many
Bacterial colonisation	Rare	Frequent
Objectives	Strong, functional and aesthetic scar	Solid, functional, aesthetic scar, if possible. Act on factors delaying healing

Table 2. Differences between acute and chronic wounds.



Figure 4. Leg ulcer.



Figure 5. Necrotic pressure sores of the heel and outer foot.



Figure 6. Foot wound in a diabetic patient.

The most common **acute wounds** are post-traumatic wounds (dermabrasion, skin tears, cuts and bites), burns and post-operative wounds (*Table 2*). When they occur in a patient with no risk factors for delayed healing, healing is usually straightforward. The main problem is the aesthetic appearance but also the risk of progression into a pathological (keloid or hypertrophic) scar.

Although the wound-healing phases are identical in all types of wound, the duration of each phase is different, with a longer proliferation and maturation phase in chronic wounds, which is the reason for the very different scar healing process between these two types of wounds.

6 How should you clean wounds?

Treating a wound should involve "clean" actions, i.e. wearing personal protective equipment (surgical masks, gloves), although it does not need to be sterile.

The first step in the management of a wound is **cleaning it with soap and water**. This cleaning process should be taught to everyone and it is never contraindicated in any wound. The aim is to remove foreign bodies, coagulated blood and non-adherent necrotic tissue from the wound, thus limiting as far as possible the risk of infection. The use of sterile isotonic saline compared to tap water has not been shown to offer any extra benefit.

The use of antiseptics plays no role in the treatment of chronic wounds. Dressings can therefore be applied directly after washing with soap and water. Antiseptics can also be harmful due to the risk of allergic contact dermatitis or irritant dermatitis, which can delay healing.

After washing with soap and water, antiseptic does not need to be applied in acute wounds treated early and not requiring any invasive procedure. However, antiseptics do have a role to play in invasive procedures such as suturing [4]. Many classes of antiseptics are available such as betadine (povidone-iodine), which comes in several forms, and should be used as the first line of defence. Iodine products, chlorhexidine and their derivatives should not be used in infants aged under 1 month and should only be used for short times and in limited applications in infants between the ages of 1 and 30 months.

Finally, in common with any wound it is essential to check the tetanus vaccination status of the patient.

7 What are the specific features of wound healing in children?

The ability of a child to synthesise tissue is important for development and growth. Wound healing is rarely a problem in paediatrics, except in very specific situations. However, the risk of progression into a **hypertrophic scar** due to fibroblastic overproduction is higher than in adults.

In addition, such pathological scars may be modified by growth, justifying prolonged and increased scar monitoring in children and adolescents (particularly in the case of burns).

8 What are the specific features of wound healing in the elderly?

Faced with an ageing population, practitioners are increasingly confronted with issue of wound healing in elderly patients.

With age, local inflammatory response is diminished, proliferation is impaired by slow keratinocyte turnover, and tissue remodelling is characterised by a lack of metalloproteinase inhibition and thus excess of collagen destruction [5]. All these **changes** have direct consequences on the healing process, making it slower and of poorer quality than in younger subjects.

Apart from these negative aspects, wound healing in the elderly offers certain advantages. Due to lower inflammatory reactions, pathological healing such as hypertrophic and/or keloid scars are rarely observed. In addition, in the case of skin removal, particularly in cancer cases involving a significant loss of substance at times, skin laxity generally allows easy suturing, without tension, producing satisfactory aesthetic results.

Finally, the **comorbidities** frequent in this population, especially malnutrition, venous and arterial insufficiency, but also multiple medications are all elements that often make healing complex and difficult.

9 How to manage wound healing in cancer patients?

For several reasons wound healing in cancer patients is often complex. **Cancer chemotherapies**, as well as **systemic corticosteroid therapy** often prescribed in these cases, may cause of delayed healing. **Progressive cancer** is also often associated with malnutrition to a greater or lesser extent, which can also delay healing. In cancer patients, acute or chronic wounds will therefore generally take longer to heal than in a patient without progressive cancer.

Some cancer treatments can also cause wounds, such as radiation dermatitis (similar to a burn), but also hydroxycarbamide (Hydréa[®]), which is indicated for myeloproliferative syndromes and causes leg ulcers, in particular. In such cases, wound healing relates directly to the continuation or cessation of the cancer treatment.

Finally, it is advisable to consider **skin cancer** (melanoma, basal cell carcinoma, squamous cell carcinoma, etc.) in the presence of any wound that is refractory to healing, particularly if it is haemorrhagic and bulging. In such cases, it is advisable to be aware of this issue when taking the history, as patients often mention a traumatic origin which can mislead the practitioner.

10 What are the specific features of wound healing in patients with burn injuries?

It is estimated that 400,000 people receive a burn injury in France each year (of which more than a third are children). Thus it is important that practitioners are able to accurately assess the wound. This assessment allows the patient to be referred to a specialised unit, if necessary, and be offered a suitable treatment to achieve optimal healing.

The depth of the burns corresponds to the anatomical level affected, and a distinction is made between:

- **superficial burns** (1st degree and 2nd degree burns): medical treatment and healing without after-effects;
- **deep burns** (2nd degree and 3rd degree): management generally requires the intervention of a plastic surgeon, and healing will involve after-effects (*Figure 7* and *Table 3*).

In any burn injury, the wound should first be cooled down under running water for a prolonged time (about ten minutes). Pain management, which is often the primary concern, is essential. In common with all wounds, it is important to check the tetanus vaccination status of the patient.

Superficial burns should be treated by:

- washing with soap and water;
- excision of blisters (except on palms of the hand and soles of the feet);
- daily tulle-gras dressing.



Figure 7. Classification of burns in degrees.

Table 3. Differences in the clinical appearance of the burn depending on the degree.

	Superficial burns		Deep burns	
	1 st degree	2 nd degree superficial	2 nd degree burns	3 rd degree
Blisters	Absent	Present	Present	Absent
Colour	Red	Bright red	Pinkish white	From white to black
Average healing time	5 days	< 2 weeks	> 3 weeks	No scarring
After-effects	No	No	Yes	Yes

Healing normally occurs in under two weeks. If this is not the case, the patient should be referred to a specialist centre. Determining the depth of a burn is not always easy and the initial assessment may be incorrect and explain the delay in healing. For this reason, in second-degree burns, a reassessment after 24-48 hours is always required.

Deep burns should always be treated in a specialised burns centre for rapid flattening and grafting.

11 What dressing should be used according to the appearance of the wound?

The choice of dressing will depend on the appearance of the wound and therefore its assessment by the practitioner. A very simple **colour classification of wounds** is used (*Tables 4* and *5*):

- "red" corresponds to granulation;
- "yellow" corresponds to fibrin;
- "black" corresponds to necrosis.

The percentage of each "colour" should be assessed to determine the type of dressing required and amount of exudate.

Dressings should be associated with specific measures when treating chronic wounds (see questions 17-19).

	Necrosis	Cleaning	Granulation	Epidermalization	Exudate
		CO.			
Hydrocolloid					+ to ++
Hydrocellular					+ to ++
Hydrogel					0
Alginate					+++ Haemostatic infection
Interface, tulle					0 to +

Table 4. Colour classification of wounds and types of dressingsto use.

Table 5. Main primary of	dressings (and	examples of	products)
according to the stage	and appearan	ce of the wou	nd.

Dressing	Necrosis	Fibrin	Granulation	Epithelialisation
Hydrogel (Purilon [®] , Hydrotac [®] transparent)	+	+		
Highly absorbent fibres (Aquacel [®] extra, Urgoclean [®])		+	+	
Hydrocolloid (Duoderm [®] Comfeel [®])		+	+	+
Hydrocellular (Mepilex®, Biatain [®])		+	+	+
Alginate (Algosteril [®] , Biatain [®] Alginate)		+	+	
Interface (Urgotul [®] , Mepitel [®])			+	+
Tulle gras (Jelonet®)				+

12 What role do dermocosmetics play in wound healing?

Some **dermocosmetic products** may be beneficial during the early phase of healing and should be used in the days after the formation of the acute wound. Such products are often enriched with copper and zinc (antibacterial action) but can also include various healing molecules.

Patients often use cosmetic products whose purpose is to hide and improve the aesthetic appearance of scars. They are also well tolerated and **can significantly improve the aesthetic appearance thus** enhancing the quality of life of patients. Some products offer enhanced covering properties compared to conventional products, have a thick texture and are available as compact or fluid foundation products. Patient associations, such as burns associations, offer training in the best way of concealing scars using these products.

13 Is there a role for alternative therapies in wound healing?

Many so-called "alternative" therapies exist for wound healing, some of which date back to ancient times.

Honey, which was already used in ancient Egypt, has antibacterial and immunomodulatory properties [6]. It can be used in several forms (waxes, natural oils, etc.) and is marketed by various pharmaceutical companies. To date, however, no studies of sufficient quality have been conducted to establish with certainty the efficacy of honey.

A number of **essential oils** are available offering anti-infectious, antiinflammatory, analgesic and deodorising properties. Such products are currently used in France in geriatrics and oncology, as diffusion or massage products to soothe, relax and facilitate sleep. However, no studies have been conducted evaluating their effectiveness on wounds. Moreover, many cases of adverse skin reactions, especially allergic reactions, have been described, meaning that their use in wound healing should be avoided.

Sterilised **maggots** and greenfly larvae can be applied directly to chronic wounds to clean up fibrinonecrotic tissue through the secretion of proteolytic enzymes. Clinical trials have demonstrated their effectiveness in the initial management of certain chronic wounds, especially venous leg ulcers [7]. They have been used in some centres for more than 20 years and have been considered as a medicinal product since 2004.

14 What advice should be given to patients to achieve an optimal scar (postoperative and post-trauma)?

The three major components of pathological scar prevention in the months following wound closure are prevention of tension, hydration/ occlusion and compression.

Wounds where the maximum tension is exerted are wounds with edges placed under stress as well as wounds located in the deltoid and presternal region. For scars located in **areas of tension**, it is recommended applying adhesive strips or tapes along the scar for three months after complete healing. Thin hydrocolloid dressings (which are more adherent) can be used as an alternative in children.

Using **emollients** and maintaining moisture through **silicone** strips or gels are recommended, as well as the use of sunscreen (factor 50+) or mechanical protection (clothing, dressings), until the scar has matured, which usually takes one year.

Compression through the use of customised clothing should be reserved for the initial phase of extensive scarring, particularly in burn patients, and should be prescribed by specialists.

It is recommended reassessing patients between 4-8 weeks after an acute wound (trauma or surgery) and between 6 weeks and 3 months for hypertrophic-scar patients, to allow them to benefit from medical compression devices and, if these devices are insufficient, to receive an injection of corticosteroids into the scar.

15 What is a pathological scar and why does it develop?

Healing as described above represents the physiological model of normal healing. In some cases, however, when healing does not occur normally, this is referred to as a **pathological scar** which can result from either delayed healing (leading to a chronic wound) or an excess scarring process (*Figure 8*).



Figure 8. Ideal, insufficient, excessive healing.

There are two clinical forms of pathological scar healing: hypertrophic scars and keloid scars, which have an identical initial phase of healing called hypertrophic. However, their progression differs: simple hypertrophic scars progress favourably over 12-18 months, usually in the form of a larger scar. While keloid scars do not tend to improve spontaneously.

Due to excessive scarring, a **hypertrophic scar** is thick, erythematous and often pruritic (*Figure 9*). Unlike keloid scars, however, it is limited to the area of injury and does not extend beyond it.



Figure 9. Hypertrophic scar.

Keloid scars are characterised by excessive activity during wound healing, resulting in thick, hyalinised, disorganised and excessive collagen fibres (*Figure 10*) [8]. Thus the extracellular matrix is abundant, while cellularity is low. The appearance is described as being like "crab claws", extending beyond the initial scar bed unlike hypertrophic scars.



Figure 10. Keloid scars.

We still do not really understand the mechanism that causes keloid scarring. Several hypotheses have been suggested such as prolonged activation of the proliferative phase of fibroblasts, decreased apoptosis, an increase in metalloproteinase inhibitors and lower collagenase activity.

Several risk factors for the occurrence of these scars have been identified such as having a dark skin type, hormonal factors (occurs more frequently during pregnancy and puberty), orientation of the wound in relation to skin tension lines and location of the wound (*Figure 11*).



Figure 11. Location of keloid scars. Frequent in red and rare in yellow.

16 How do you treat pathological scars?

Patients with pathological scars (keloid or hypertrophic) should be referred to a dermatologist or plastic surgeon.

The treatment of **keloid scars** is complex and varied and includes intrascar corticosteroid injections (triamcinolone), cryotherapy, lasers, cryosurgery, radiotherapy, etc. (*Table 6*).

Silicone, which is easily accessible and can be prescribed by any practitioner, can be beneficial for the initial management of pathological scars. Thus, silicone should be offered to the patient as a first step before referring them to a specialist. Silicone comes in various forms such as patches, gel, spray, etc., and has an occlusive effect favouring hyperhydration of the corneal layer.

Table 6. Summary of treatments for pathological scars.



17 How do you treat and heal a leg ulcer?

Leg ulcers affect 1% of the general population and 5% of patients over the age of 80 years (HAS 2006). They are most commonly caused by **venous insufficiency** but can also be caused by **arterial problems** due to lower limb arteriopathy, or are of mixed origin, combining venous insufficiency and lower limb arteriopathy. Venous ulcers may be the result of venous failure due to leaky valves (leading to varicose ulcers) or may occur after phlebitis, causing deep venous insufficiency triggering venous ulcers that are more difficult to heal.

Treating an ulcer will depend on its origin thus it is essential to determine the nature of the ulcer involved (*Table 7*). With any lower-limb ulcer, it is advisable to palpate the peripheral pulses, and their absence or reduction indicates a potential ulcer of arterial origin. Another central element is the measurement of the **systolic pressure index** (SPI). This measurement, calculated using a hand-held Doppler and a manual pressure cuff, is the ratio between ankle systolic pressure/arm systolic pressure. SPI normally ranges between 0.9 and 1.3. When this figure is above 1.3 and the leg arteries cannot be compressed, this is referred to as "mediacalcosis" (i.e. media calcification of arterial vessels). SPI below 0.9 indicates the presence of lower limb arteriopathy, which is considered to be a critical stage of ischaemia if the SPI is below 0.5 [9].

If doctors do not have the appropriate equipment in their surgery, they can ask the radiologist to measure the SPI when performing the arterial Doppler ultrasound.

	Peripheral pulses	Present	Reduced or even completely absent
	Peri-ulcerous skin	Lower limb oedema, ochre dermatitis, varicose veins	Pale, hairless, cold
	Appearance of the ulcer	Lower third of leg, irregular contours, oozing, shallow	Instep, heel, foot, sharp edges, deep, sometimes necrotic
	Pain	Little or no pain	Very painful, aggravated by supine positions, intermittent claudication
	Location	Women Thromboem- bolic history, multiple pregnancies, varicose vein surgery	Men Cardiovascular risk factors
•	Frequency	60-80% of ulcers	10% of ulcers
		Venous ulcer	Arterial ulcer

Table 7. Major differences between venous and arterial ulcers.

A vascular assessment is important as:

- in venous ulcers, treatment is based on venous compression [10];
- in arterial ulcers, compression is contraindicated (*Figure 12*). An arterial Doppler ultrasound of the lower limbs should be performed and the patient referred to a vascular surgeon.



Figure 12. Deep and necrotic arterial ulcers.

18 How do you treat and heal a pressure sore?

A pressure sore is a skin lesion of ischaemic origin caused by compression between soft tissue, a hard surface (e.g. floor or mattress) and a bony projection (*Figure 13*). By definition, such sores develop in **areas of pressure** such as on the heels, and on the lower part of the back and hips (e.g. sacrum and ischium). Many factors trigger pressure sores including malnutrition, poor vascular condition (especially in heel pressure sores with lower limb arteriopathy), and progressive cancer.

Healing pressure sores is dependent on the management of these **triggering factors** and offloading pressure from the wound. Healing can be slow in patients with multiple co-morbidities, and more rapid in young subjects in an acute setting (e.g. prolonged immobilisation due to voluntary drug intake).

Pressure can be offloaded using a range of devices such as special mattress, mattress topper, pressure-relieving/offloading boots, etc.



Figure 13. Pressure sore in the sacral region.

19 How do you treat and heal a foot wound in a diabetic patient?

Foot wounds in diabetic patients are caused by multiple factors including neuropathy, lower limb arteriopathy, increased susceptibility of diabetic patients to infections, etc. (*Figure 14*). This type of wound can become chronic for three main reasons: sustained pressure, superinfection of the wound and underlying ischaemia, resulting in delayed healing or even worsening of the wound. They may also be further complicated by **osteoarticular infection** with the risk of amputation. It is essential to explain to patients the risks involved in the management of such wounds as they are not always painful and the patient may not be aware of the seriousness of the situation.



Figure 14. Heel wound in a diabetic patient.

The key element of the care is **the offloading of pressure from the wound**. It must also be complete and permanent, making compliance often difficult, especially as healing is often lengthy. Several offloading techniques are available such as offloading shoes (*Figure 15*), removable plaster cast boots, etc.

In addition, new wound-healing booster dressings are now available and have been shown to be effective in neuro-ischaemic foot wounds in diabetic patients [11].



Figure 15. Offloading shoe for a forefoot wound in a diabetic patient.

20 What is the relationship between wound healing and mental health?

It is important to assess the consequences of a scar on the **psychological experience** of the patient. Minimising this impact can be tempting, especially in cases involving post-cancer surgery or scarring after a road-traffic accident. The vital nature of the surgery having caused the scar may overshadow the possibly unsightly appearance of the scar, making any request from the patient appear unjustified.

Quality of life is therefore directly dependent on the condition of the skin as perceived by the patient and his or her family. The **Dermato-logy Life Quality Index** (DLQI) is the most commonly used score to assess the impact of the skin problem on quality of life and can be applied to scars [12]. The **Patient and Observer Scar Assessment Scale** (POSAS) was developed specifically for scar assessment, and its main advantage is that it takes into account both the perception of the patient and that of the clinician.

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- How do you treat pathological scars?
- How do you treat and heal a leg ulcer, pressure sore or foot
 wound in a diabetic patient?

From the process involved to treatment and day-to-day management, Dr Hester Colboc answers essential questions about wound healing.

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