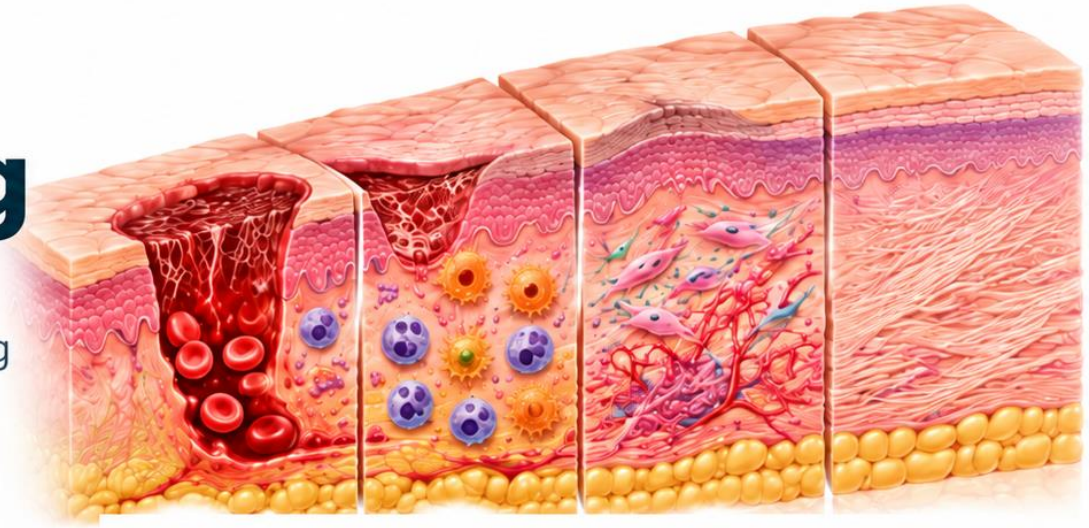
DERMATOLOGY COURSE · 3rd YEAR OF MEDICAL SCHOOL

Cutaneous Wound Healing

Physiological phases, influencing factors, healing modalities and pathological scarring



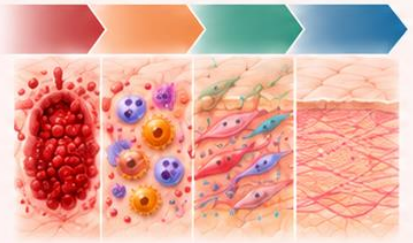
Prof. Ahmed Bouhamidi, MD, PhD
Head of the Department of Dermatology
Military Hospital Avicenne, Marrakech, Morocco

By the end of this course, you will be able to…

1

Describe the physiological phases

Hemostasis, inflammation, proliferation, remodeling: cells, duration, clinical appearance



2

Identify influencing factors

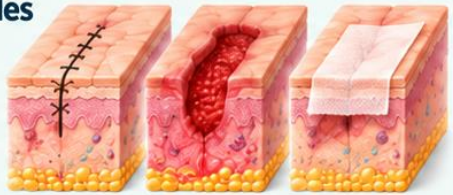
Local and systemic factors: diabetes, smoking, malnutrition, age



3

Distinguish the modes of wound healing

Directed wound healing, primary intention, secondary intention



4

Recognize pathological wound healing

Delayed healing, keloid, hypertrophic scar

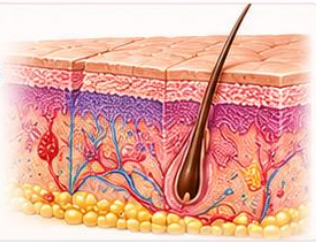


Six key topics to understand cutaneous wound healing

1

1. Introduction and key concepts

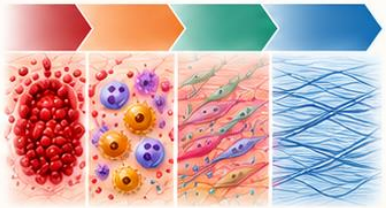
Definitions, skin structure, repair vs regeneration



2

2. Physiological phases

Hemostasis, inflammation, proliferation, remodeling



3

3. Influencing factors

Local and systemic factors: diabetes, smoking, malnutrition, age



4

4. Modes of wound healing

Primary intention, secondary intention, directed wound healing



5

5. Pathological wound healing

Delayed healing, hypertrophic scar, keloid



6

6. Clinical cases and MCQs

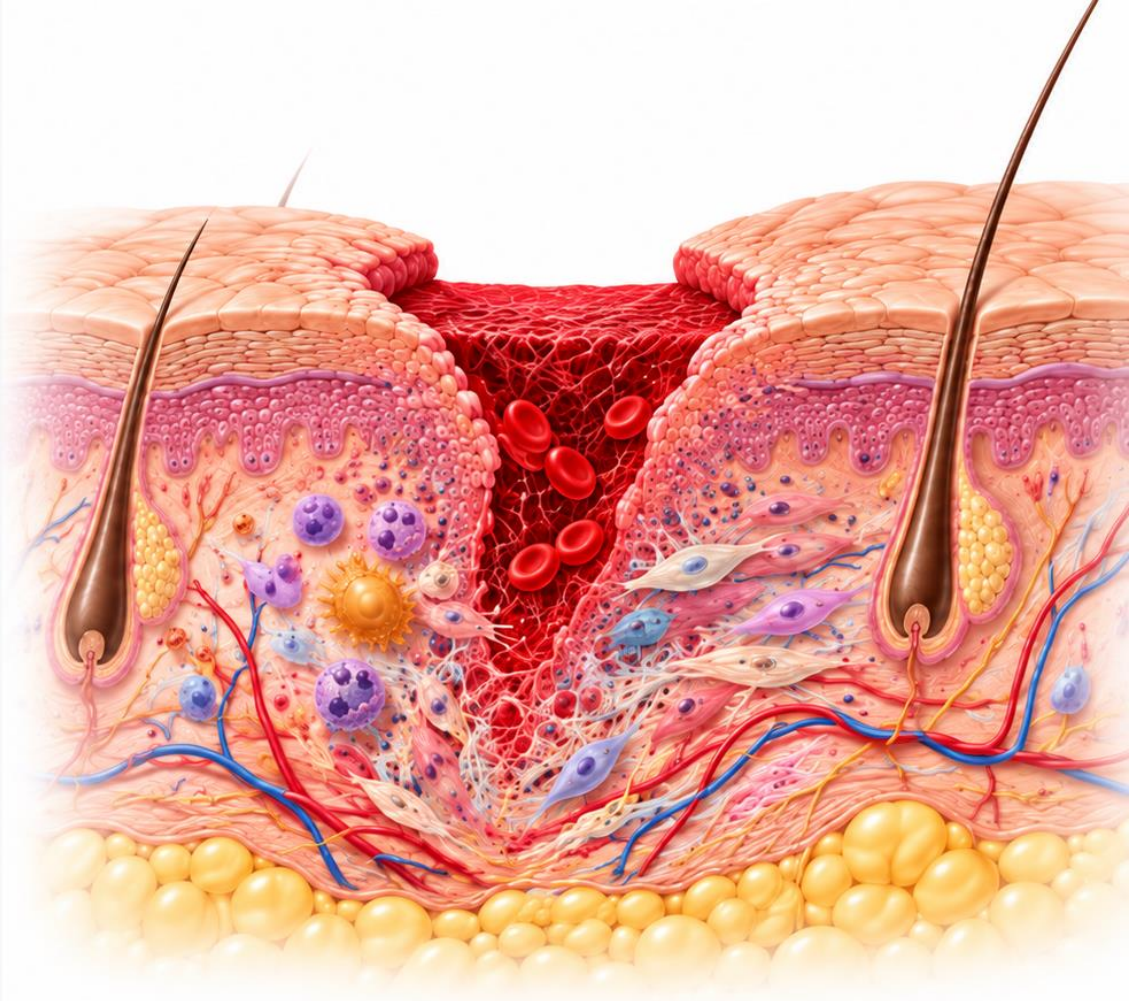
Practical application, self-assessment, key take-home messages



1

Introduction and key concepts

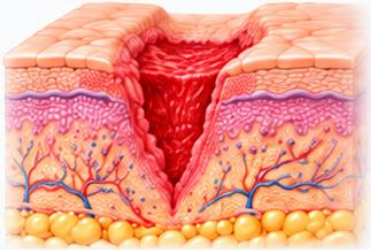
Definitions, skin structure, repair versus regeneration



Essential definitions

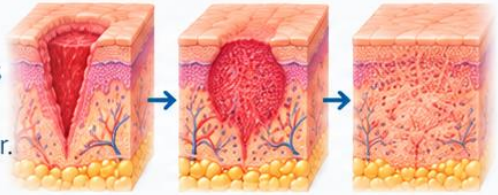
Wound

A disruption of skin continuity, with or without tissue loss, potentially involving the underlying tissues.



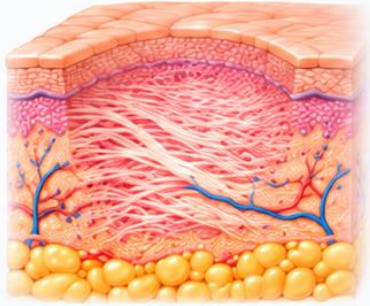
Wound healing

A dynamic biological process of tissue repair that restores the integrity of the skin barrier.



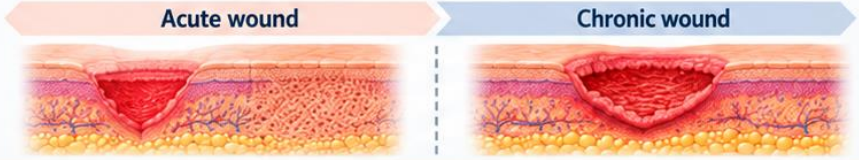
Scar

Fibrous replacement tissue without skin appendages (hair follicles, glands), with lower mechanical strength than healthy skin.



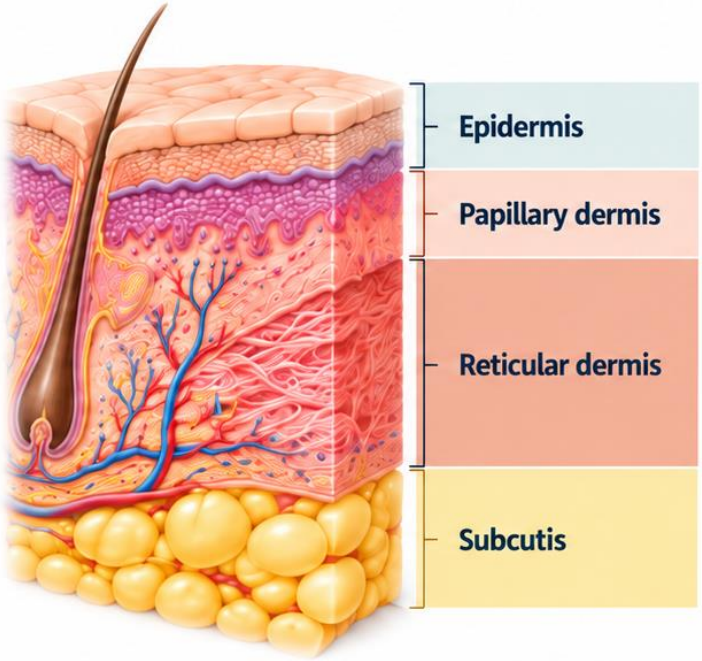
Acute vs chronic wound

Acute: follows the 4 phases within the expected time frame.
Chronic: not healed after 4–6 weeks, depending on the underlying cause (HAS).



TAKE-HOME MESSAGE Wound healing = repair (with fibrosis), not regeneration to the exact original state.

How deep is the wound, and what kind of healing?

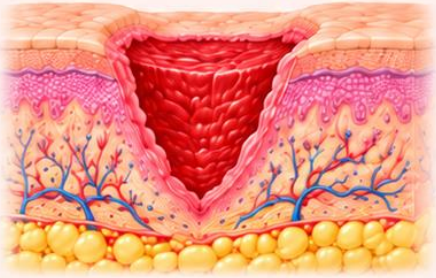


Superficial wound: epidermis and papillary dermis



Re-epithelialization from skin appendages (hair follicles, glands) : rapid healing, usually without a visible scar.

Deep wound: reticular dermis and beyond



Skin appendages are destroyed or scarce: granulation tissue is required and fibrous scar formation is inevitable.

Key players: *keratinocytes (epidermis), fibroblasts and blood vessels (dermis), immune cells.*

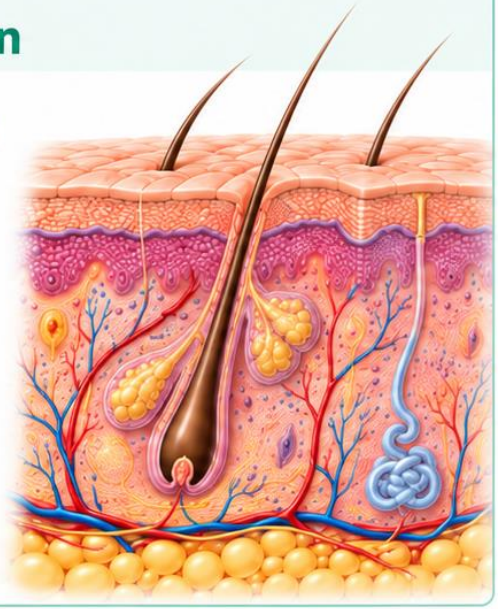
TAKE-HOME MESSAGE The deeper the wound, the longer the healing process and the more visible the scar.

Repair or regeneration ?

Regeneration

Restoration of the injured tissue to its original state, including its appendages and architecture.

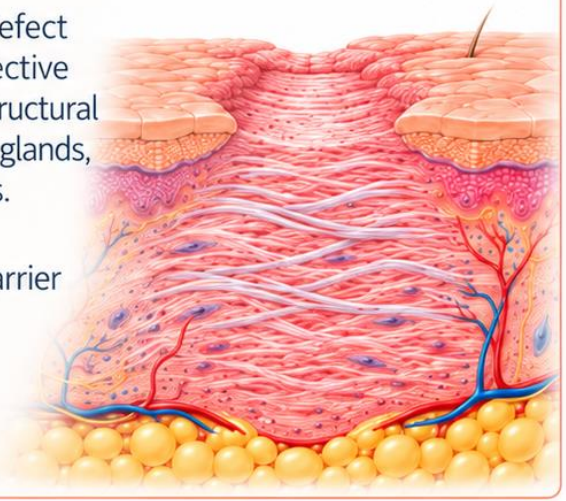
Observed in fetal skin early in gestation and in certain species.



Repair (adult skin)

Rapid closure of the defect by scar-forming connective tissue, at the cost of structural loss: no hair follicles or glands, aligned collagen fibers.

Priority: restore the barrier against infection and water loss.

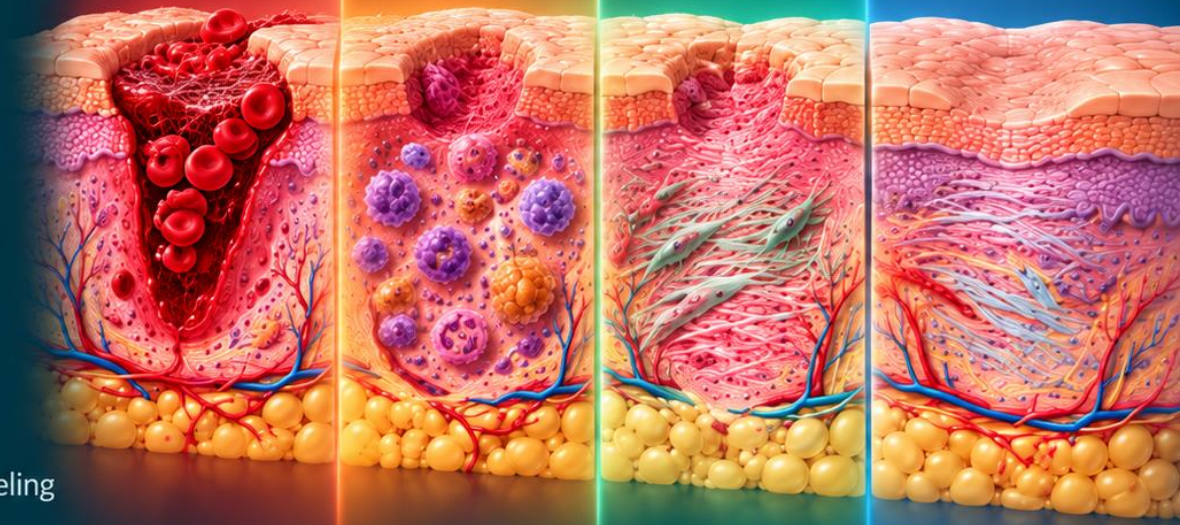


TAKE-HOME MESSAGE In adults, skin heals by repair: the scar is the price of rapid closure.

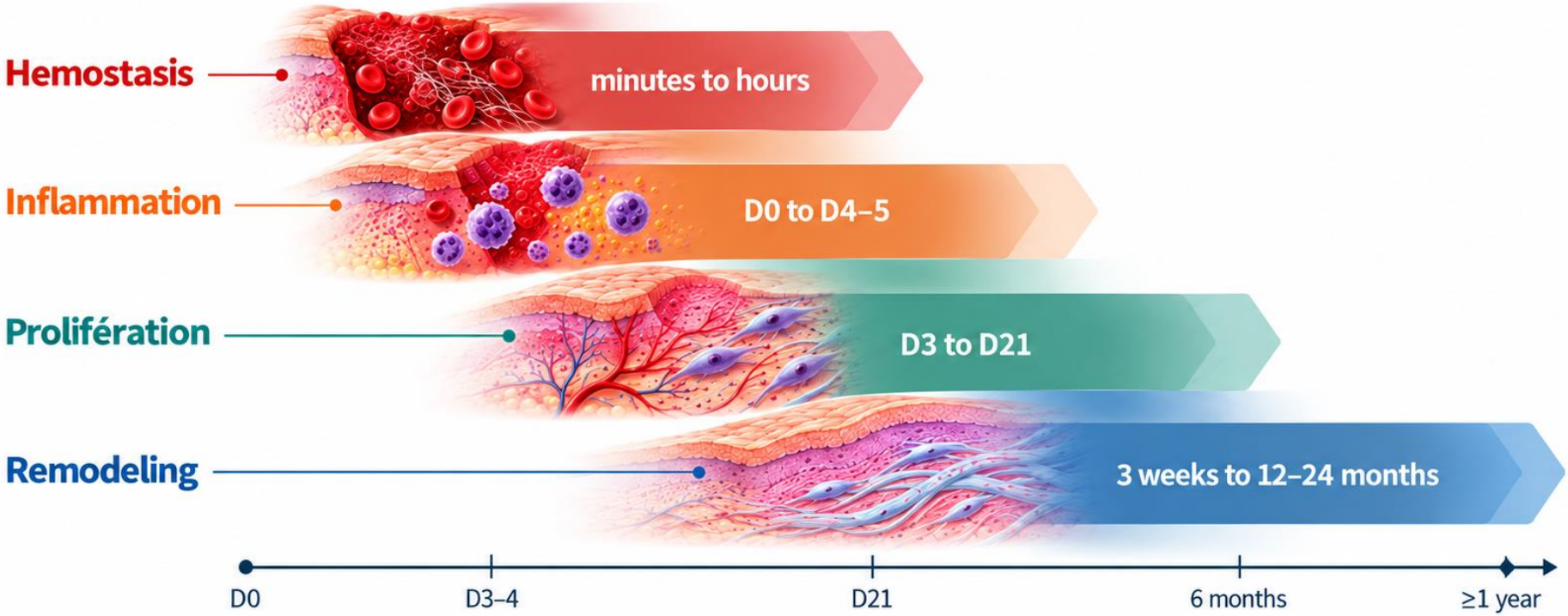
2

Physiological phases

Hemostasis · inflammation · proliferation · remodeling



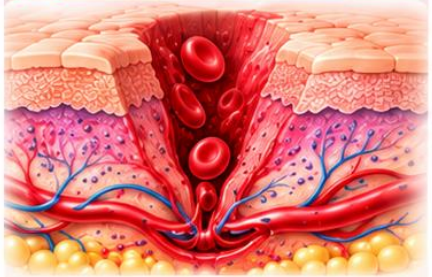
Four overlapping phases



Non-linear scale, indicative durations

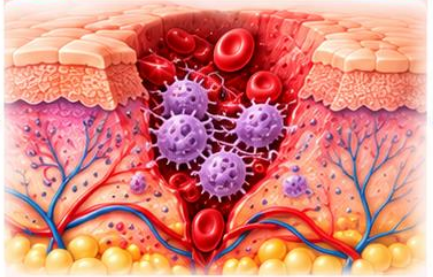
Stop the bleeding, establish the provisional matrix

1 Vasoconstriction



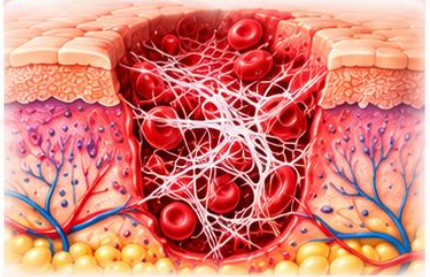
Immediate, a few minutes (endothelin, catecholamines)

2 Platelet aggregation



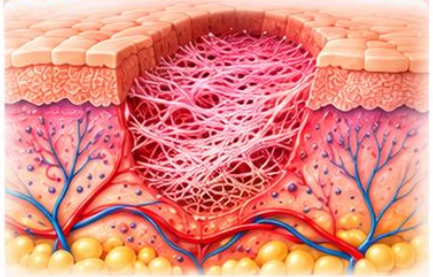
Adhesion to exposed collagen (von Willebrand factor): platelet plug

3 Coagulation



Thrombin: fibrinogen → fibrin

4 Provisional matrix



Fibrin and fibronectin clot: cellular scaffold

Platelets: much more than a plug



Their degranulation releases PDGF, TGF-β, VEGF… which recruit inflammatory cells.

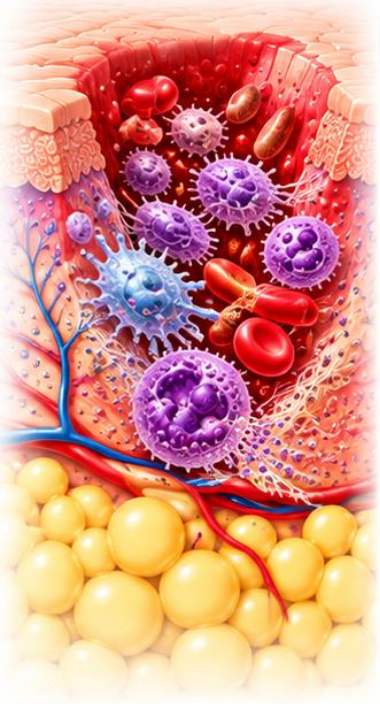
Clinical appearance

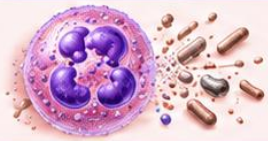
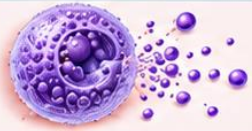


Bleeding stops, crust formation.
After 5–10 min: reactive vasodilation (histamine, kinins).

TAKE-HOME MESSAGE Hemostasis initiates wound healing: the clot is a reservoir of growth factors.

Clean the wound and initiate repair

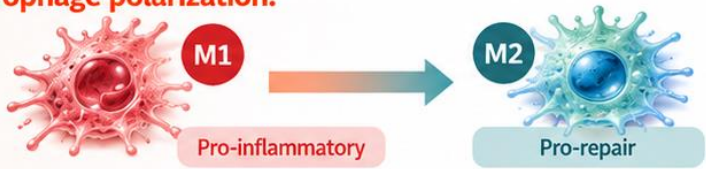


Cell	Arrival	Main role
 **Neutrophils** Polymorphonuclear cells	**First hours,** peak at 24–48 h	Phagocytosis of bacteria and debris, release of proteases and reactive oxygen species (detersion).
 **Monocytes → Macrophages**	**Day 2–3,** then dominate	Phagocytose apoptotic neutrophils; secrete TNF-α, IL-1, IL-6, then TGF-β, VEGF, PDGF (pro-inflammatory → pro-repair).
 **Mast cells**	Early	Histamine: vasodilation and increased vascular permeability.

Local signs:



Macrophage polarization:



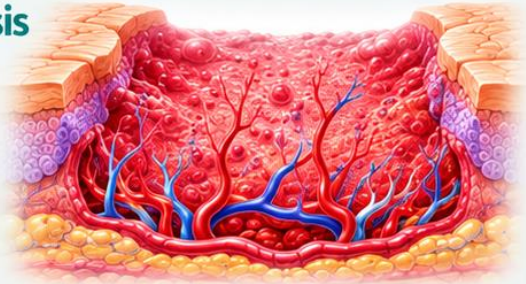
KEY MESSAGE | Inflammation does not block healing if it resolves properly: it is the engine of chronic wounds.

PHASE 3 · PROLIFÉRATION

Fill, cover, contract

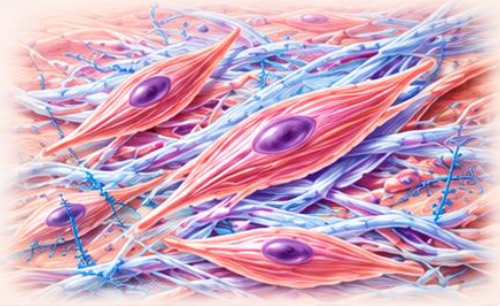
1 Angiogenesis

VEGF, FGF-2, hypoxia : vascular sprouts that irrigate the granulation tissue.



2 Fibroplasia

Fibroblasts: type III collagen, fibronectin, proteoglycans, provisional extracellular matrix.



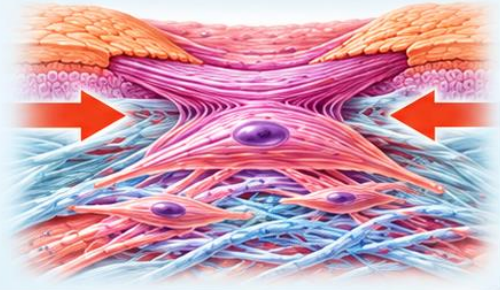
3 Epithelialization

Keratinocytes at the wound edges and skin appendages migrate, proliferate, and cover the wound.



4 Contraction

Myofibroblasts (actin–smooth muscle): draw the wound edges together and reduce the surface area.



KEY MESSAGE | Four simultaneous processes produce granulation tissue.

Assessing the wound bed: healthy vs. concerning granulation tissue

Healthy granulation tissue



- Bright red, granular, moist, and bleeds on contact.
- Regular surface, below the level of the surrounding skin.
- Epithelializing wound edge: thin pink peripheral rim.

Concerning granulation tissue



- **Pale, gray, or friable:** ischemia, infection, malnutrition.
- **Fibrinous, yellowish, necrotic:** debridement required.
- **Hypergranulation:** exuberant granulation tissue rising above the skin level and delaying epithelialization.

Management of hypergranulation: stop occlusive dressings; use an absorbent dressing; topical corticosteroid for 2–3 days (medical supervision); silver nitrate; biopsy if the appearance is suspicious.

KEY MESSAGE | A pale or friable wound bed is an alarm signal: look for ischemia, infection, or malnutrition.

Maturation: from a red scar to a pale scar

≈ 20 %
of final tensile strength at 3 weeks

70–80 %
of normal skin strength: maximum reached between 3 months and 1 year

< 100 %
strength is never completely restored

- Type III collagen is replaced by type I collagen, reorganized into bundles (MMP/TIMP balance).
- Vascular regression and myofibroblast apoptosis.
- Scar initially red and raised (3–6 months), then pale, flat, and supple (12–24 months).
- Photoprotection of the scar during maturation.



KEY MESSAGE | A scar should not be judged before 12–18 months.

Overview of the four phases

Phase	Duration	Key cells	Mediators	Clinical appearance
1 Hemostasis 	Minutes to hours	 Platelets	• Thrombin • Fibrin • PDGF • TGF-β	 Clot, scab
2 Inflammation 	Day 0 to Day 4–5	 Neutrophils, then macrophages	• TNF-α • IL-1 • IL-6	 Redness, warmth, edema, exudate
3 Proliferation 	Day 3 to Day 21	 Fibroblasts, keratinocytes, endothelial cells, myofibroblasts	• VEGF • FGF • EGF • TGF-β	 Red granulation tissue, advancing wound edges
4 Remodeling 	3 weeks to 12–24 months	 Fibroblasts, myofibroblasts (apoptosis)	• MMP • TIMP	 Red scar, then pale and supple

KEY MESSAGE | Hemostasis → inflammation → proliferation → remodeling: any abnormality in one phase disrupts the following phases.

3

Factors influencing wound healing

Local and systemic factors: diabetes, smoking, malnutrition, age.

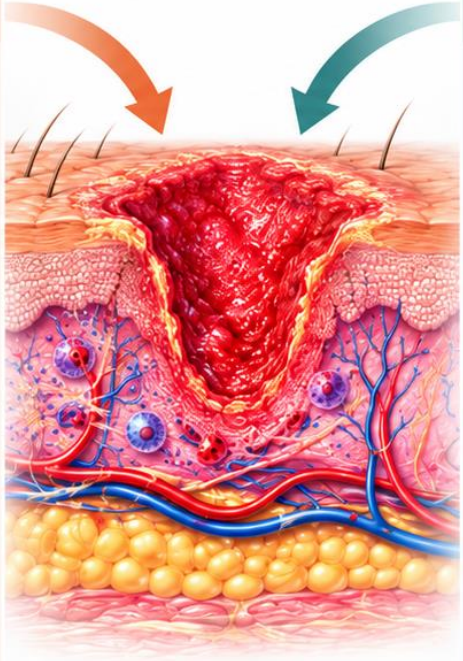


Two families of factors

Local factors

- Infection, critical colonization, biofilm
- Ischemia, tissue hypoxia
- Necrosis, fibrin, foreign body
- Tension, mobility, pressure
- Inappropriate environment: dryness or maceration
- Previous radiotherapy, repeated trauma





Systemic factors

- Age
- Diabetes
- Smoking
- Malnutrition and deficiencies
- Vascular disease, renal or hepatic insufficiency
- Immunosuppression, obesity, medications (corticosteroids…)



KEY MESSAGE | First correct accessible local and systemic causes: no dressing can compensate for untreated underlying factors.

What in the wound impairs healing

Infection and biofilm

Critical colonization maintains inflammation. Warning signs: increasing pain, exudate, odor, delayed healing.



Ischemia, hypoxia

Oxygen is essential for fibroblasts, collagen synthesis and host defense against infection.



Necrosis, foreign bodies

Fibrin, eschar and debris maintain inflammation: debridement is a prerequisite for healing.



Tension and pressure

Excessive tension promotes wound dehiscence and wide scarring; pressure is a cause of pressure ulcers.



Local environment

A moist environment promotes keratinocyte migration, prevents desiccation and maceration, and supports healing.



Previously injured tissue

Irradiated, fibrotic or hypovascularized skin: repeated trauma and manipulations.



KEY MESSAGE | Infection, ischemia, necrosis, and tension: the four local causes to systematically look for.

Diabetes: A Defect at Every Stage

Angiopathy

Microangiopathy and macroangiopathy: tissue hypoxia and impaired perfusion.



Neuropathy

Loss of protective pain sensation: unperceived microtrauma and pressure-related injuries.



Impaired Immunity

Reduced neutrophil chemotaxis and phagocytosis, increasing susceptibility to infection.



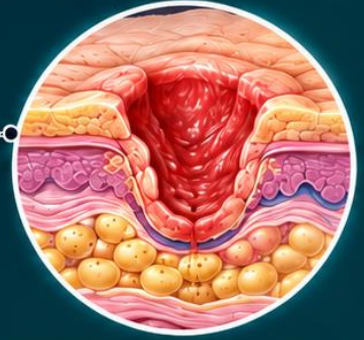
Hyperglycemia

Prolonged inflammation, increased matrix metalloproteinase activity, and protein glycation.

Diabetic Foot Ulcer

Neuropathy + peripheral arterial disease ± infection.

Lifetime risk is estimated at 19–34% among people with diabetes.



Key Takeaway | **Glycemic control, offloading, revascularization, and infection management**: a multidisciplinary approach.

Tobacco: Hypoxia and Cellular Dysfunction

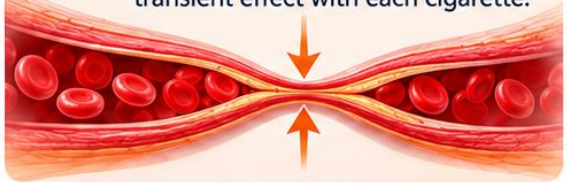
12% vs 2%

Wound infection rate: smokers vs non-smokers (standardized wound studies).



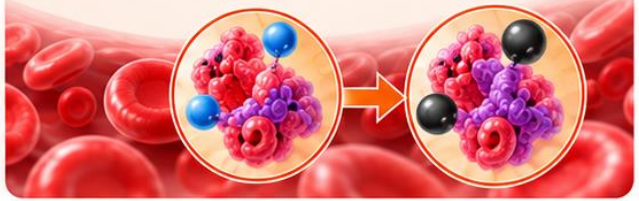
Nicotine

Vasoconstriction: reduced cutaneous perfusion and transient effect with each cigarette.



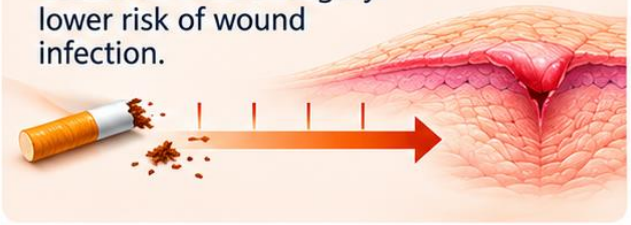
Carbon Monoxide

Carboxyhemoglobin: reduced oxygen delivery to tissues.



≥ 4 weeks

Cessation before surgery: lower risk of wound infection.



Inflammatory Cells

Impaired chemotaxis, migration, and bactericidal activity of neutrophils and macrophages.



Proliferation

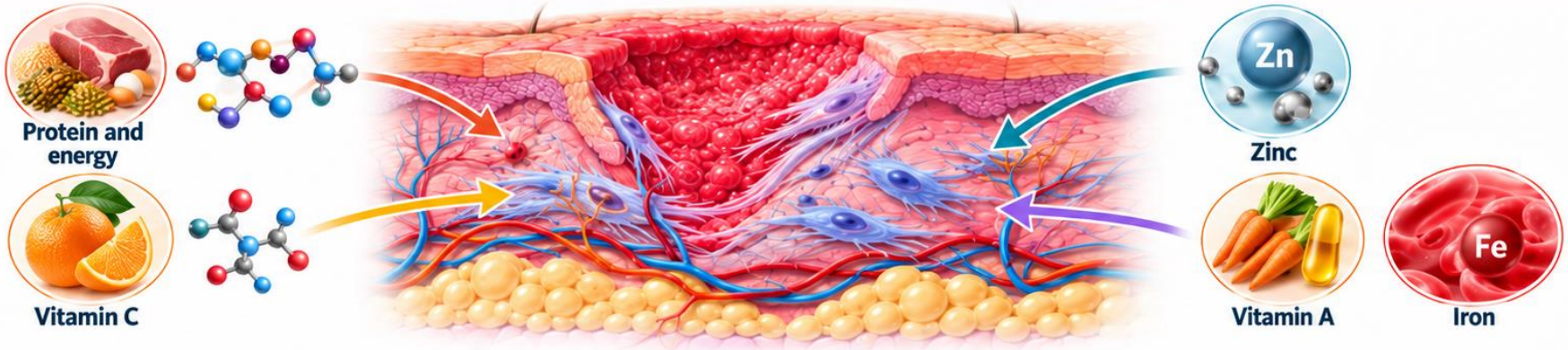
The proliferative response remains impaired even several weeks after cessation.



Key Takeaway | **Scheduled surgery:** stop tobacco at least 4 weeks before and continue abstinence thereafter.

Malnutrition: no building blocks, no repair

Nutrient	Role in wound healing
Protein and energy	Collagen and extracellular matrix synthesis; increased requirements with extensive wounds
Vitamin C	Hydroxylation of collagen proline and lysine (scurvy: impaired wound healing)
Zinc	Enzymatic cofactor; cellular proliferation and epithelialization
Vitamin A, iron	Inflammation and epithelialization (vitamin A); oxygen transport (iron)



Warning signs: weight loss, low BMI, pale granulation tissue, delayed healing, pressure ulcers. Low serum albumin is a marker of severity and should be interpreted with CRP.

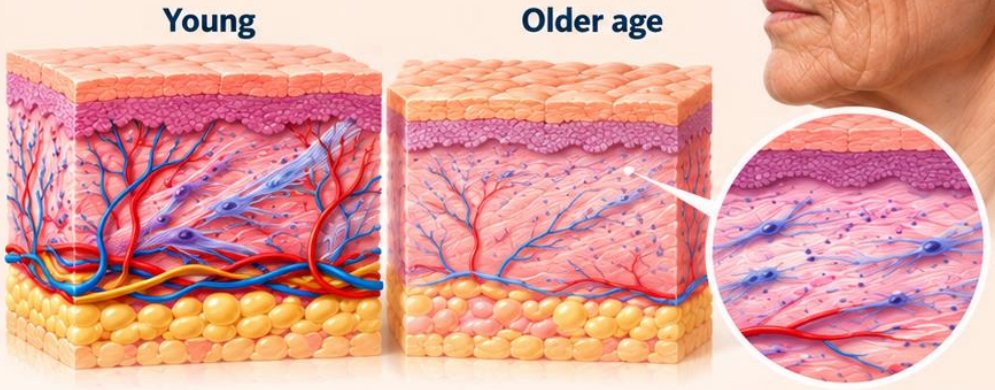
KEY MESSAGE Pressure ulcer in a malnourished or at-risk adult: 30–35 kcal/kg/day and 1.25–1.5 g protein/kg/day (ESPEN/NPIAP/PPPIA 2019).

Age: slower wound healing, not impossible

In older adults

- Delayed and less effective inflammatory response.
- Reduced fibroblast proliferation and angiogenesis.
- Slower epithelialization, thinner dermis, fragile skin.
- Associated comorbidities and polypharmacy.





Practical implications

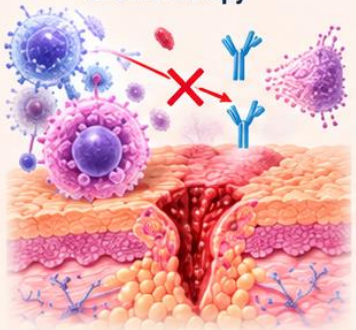
- Acute wounds take longer to heal, with more frequent dehisence.
- Use gentle dressings and adhesives (risk of skin tears).
- Assess for malnutrition, vascular disorders, and medications.
- Hypertrophic scars are less common; keloids occur mainly in younger adults.



KEY MESSAGE | Age slows wound healing, but associated comorbidities are the main contributors.

Other systemic factors to know

1 Systemic corticosteroids, immunosuppressants, chemotherapy



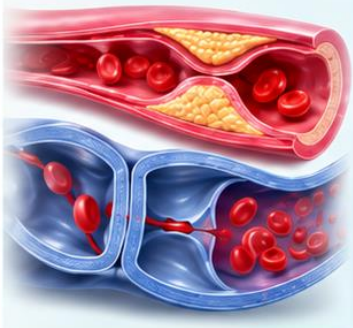
Main mechanism

Impaired inflammation and proliferation, increased infection risk

Management

Reassess the indication, monitor closely

2 Arteriopathy, venous insufficiency



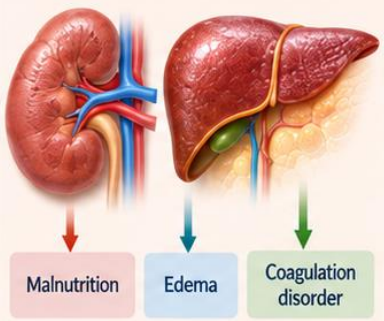
Main mechanism

Hypoxia, venous hypertension

Management

Revascularization, appropriate compression

3 Renal or hepatic failure



Main mechanism

Malnutrition, edema, coagulation disorder

Management

Optimize the underlying disease

4 Obesity, alcohol



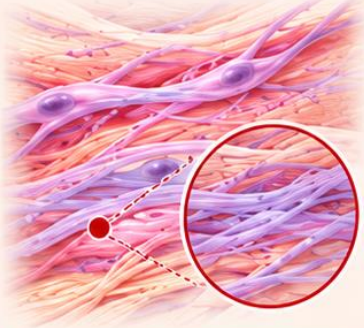
Main mechanism

Tension, hypoperfusion, deficiencies

Management

Weight loss, alcohol cessation

5 Connective tissue disorders (Ehlers-Danlos...)



Main mechanism

Abnormal collagen

Management

Adapted surgical technique

KEY MESSAGE Modifiable factors: smoking, glycemic control, nutrition, medications. Identifying them is a therapeutic intervention.

4

Modes of Wound Healing

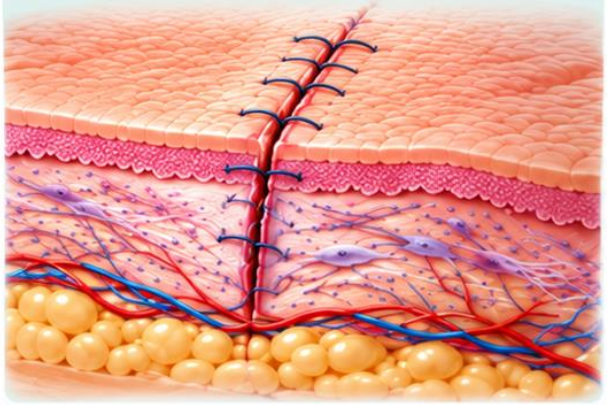
Primary intention • Secondary intention • Secondary healing / directed wound healing



Three ways to close a wound

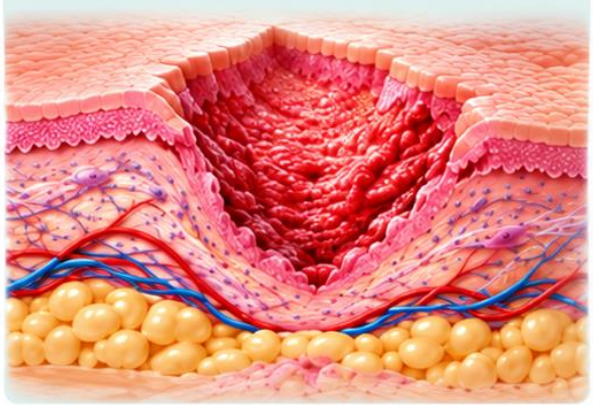
Primary intention

Clean, closely apposed edges by sutures, staples, or adhesive strips.
No tissue loss.
Rapid healing with a fine linear scar.



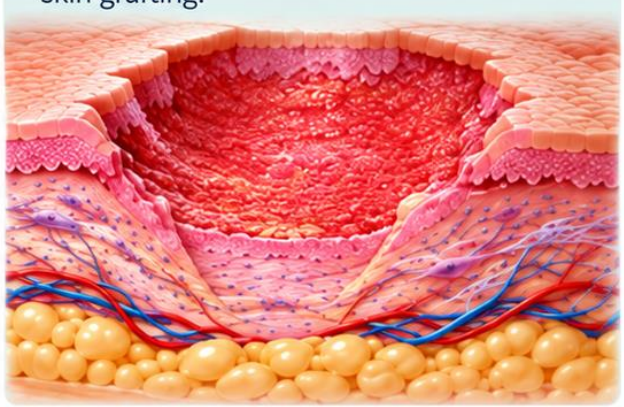
Secondary intention

Separated edges with tissue loss.
Spontaneous filling by granulation tissue, contraction, and epithelialization.
Longer healing time, wider scar.



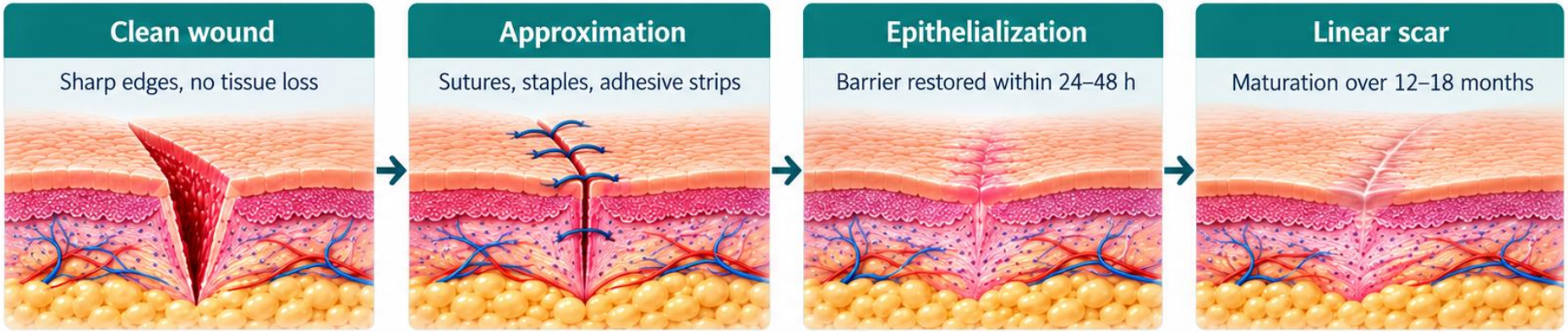
Directed wound healing

Wound intentionally left open.
Stage-adapted care (debridement, granulation, epithelialization).
Dressings, negative-pressure wound therapy, skin grafting.



KEY MESSAGE Directed wound healing is a form of secondary-intention healing that is actively managed.

Approximated wound edges, linear scar



- Indications: clean, recent wound (acceptable delay depends on site and contamination)
- Dirty wound, bite, or old wound: debridement; do not close primarily
- Check tetanus vaccination status

Site	Suture removal (indicative)
Face	5 to 7 days
Scalp	7 to 10 days
Trunk, limbs	10 to 14 days
Joints, sole	14 days or longer

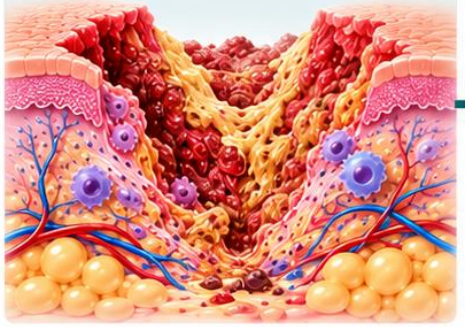
Indicative timings; adapt to tension and local evolution.

KEY MESSAGE A clean wound closed early heals with the finest scar.

Let the wound heal by itself

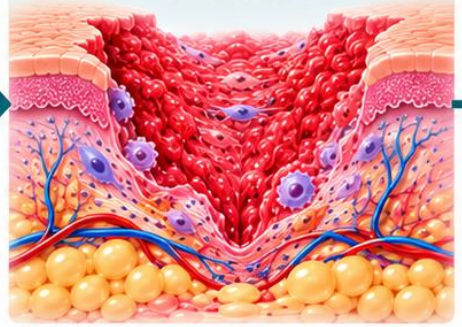
Debridement

Removal of fibrin and debris



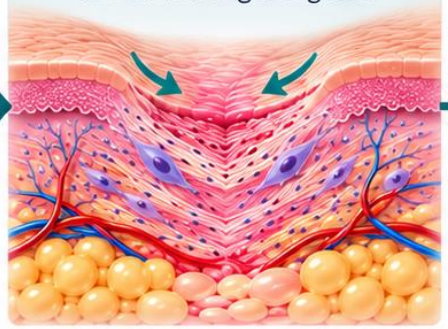
Granulation

Granulation tissue fills the tissue defect



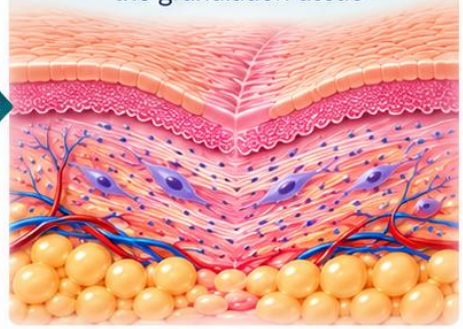
Contraction

Myofibroblasts draw the wound edges together



Epithelialization

Keratinocytes cover the granulation tissue



Indications

Non-suturable tissue loss, infected or contaminated wounds, wounds in concave areas (nose, eye, ear, temple), chronic wounds.

Limitations

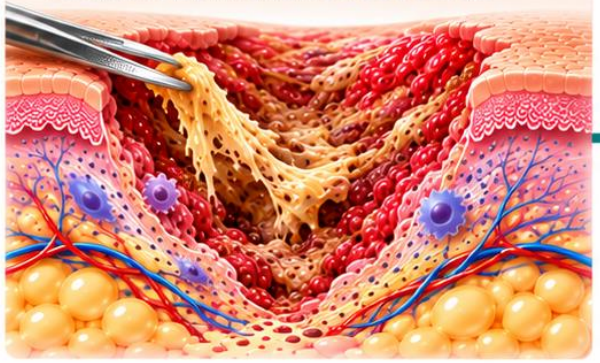
Healing takes several weeks, frequent wound care, wider or contracted scar, risk of hypergranulation (often beneath occlusive dressings).

KEY MESSAGE | Facial concavities often heal very well by secondary intention (Zitelli, 1983).

Wound healing by secondary intention: step by step

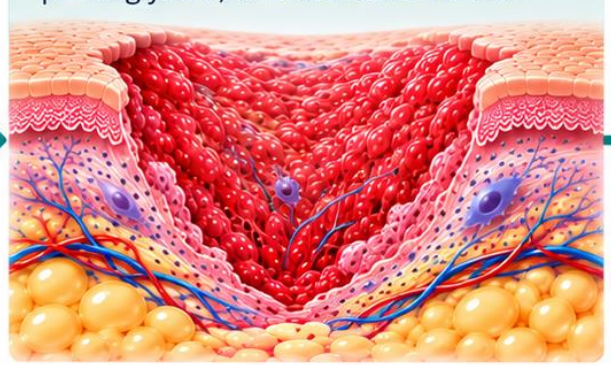
1 Debridement

Mechanical, autolytic (hydrogel, hydrocolloid) or surgical debridement to remove fibrin and necrotic tissue.



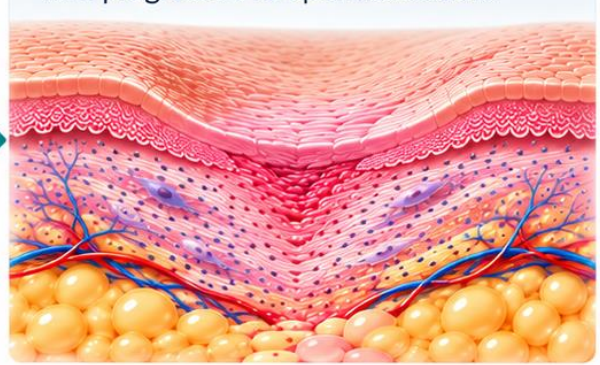
2 Granulation

Granulation tissue fills the defect with extracellular matrix (collagen, proteoglycans) and new blood vessels.



3 Epithelialization

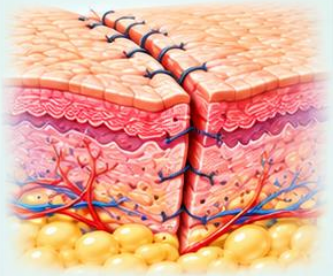
Keratinocytes migrate from the wound edges over the granulation tissue, with progressive re-epithelialization.



- **Principle :** manage the cause, debridement, maintain a moist environment, protect the surrounding skin.
- **Dressing choice:** based on exudate level, wound depth, pain and perilesional skin.
- **Options for complex wounds :** negative pressure wound therapy, skin grafting or dermal substitutes.

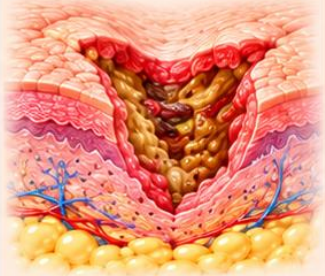
KEY MESSAGE — Secondary intention healing proceeds through debridement, granulation and epithelialization; dressing should be adapted as the wound evolves.

Which healing mode to choose for a wound?



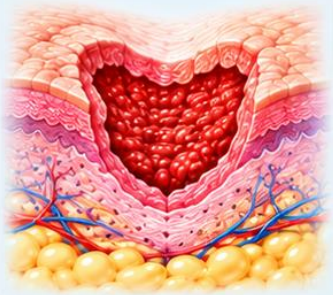
Clean, recent, proper wound

- Sharp edges, no tissue loss
- Closing by sutures: primary intention
- Fast, linear scar



Dirty, contaminated or old wound

- Debridement and irrigation
- Wound left open
- Delayed closure after 3–5 days if clean: tertiary intention



Loss of tissue, concave area

- Healing by secondary intention (directed healing)
- Granulation, contraction and epithelialization



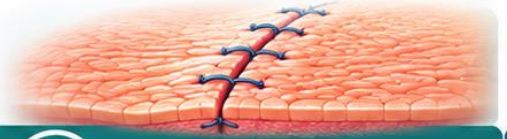
Extensive tissue loss, exposed bone or tendon

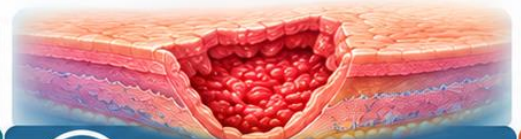
- Reconstructive procedures: skin graft, flap
- Negative pressure wound therapy
- Specialist referral

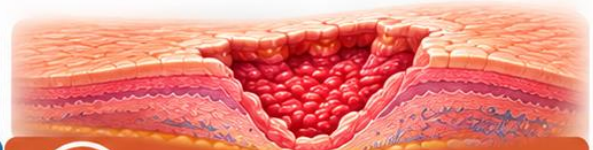
KEY MESSAGE

Choose the healing mode based on the wound characteristics. Assess and manage in all cases: wound assessment, hemostasis, debridement, and tetanus immunization.

Comparative overview







	Primary intention	Secondary intention	Directed wound healing
Wound edges	Approximated	Separated	Separated; wound intentionally left open
Mechanism	Epithelialization across a narrow wound gap	Granulation, contraction, epithelialization	Same mechanisms, actively guided step by step
Duration	Days (maturation 12–18 months)	Weeks	Weeks to months depending on the wound
Scar	Linear, fine	Wider, sometimes contractile	Variable, improved with appropriate management
Indications	Clean, uncomplicated, recent wound	Tissue loss, concave areas, infected wounds	Chronic wounds, tissue loss, wounds after debridement
Care	Sutures, simple wound care	Dressings, monitoring	Stage-adapted dressings, ± negative-pressure wound therapy, ± skin grafting

KEY MESSAGE | Distinguish: approximated edges (primary intention), separated edges (secondary intention), secondary intention actively guided (directed healing).

5 Pathological wound healing

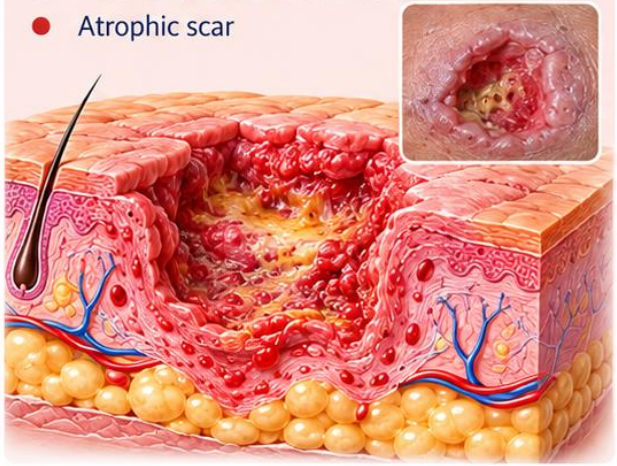
Delayed healing · Hypertrophic scar · Keloid



Deficient or excessive wound healing

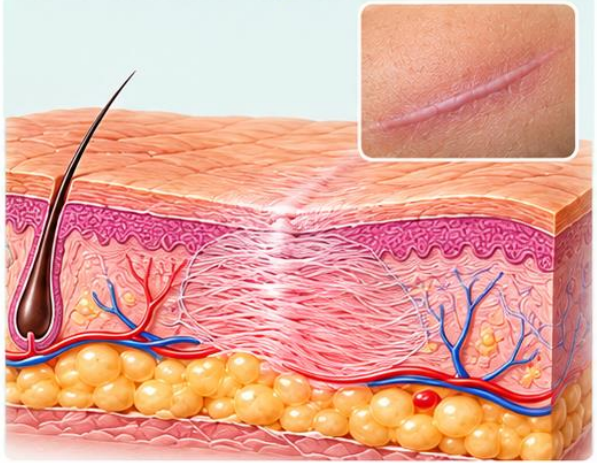
Deficient healing

- Delayed healing and chronic wound
- No healing after 4–6 weeks (e.g. HS)
- Infection, suture dehiscence
- Atrophic scar



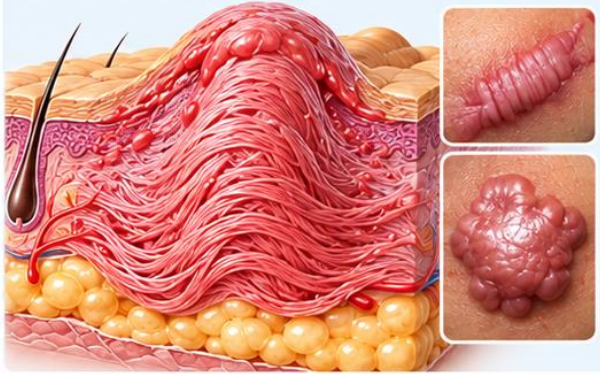
Normal healing

- Normal scar
- Flat, soft, pale scar after 12–18 months
- No functional impairment



Excessive healing

- Hypertrophic scar and keloid
- Excessive fibrosis
- Hypertrophic scar: limited to the wound
- Keloid: extends beyond the wound margins and does not regress
- Also: wider, raised, retractile scar



KEY MESSAGE | **Pathological** healing includes delayed healing, hypertrophic scar, and keloid.

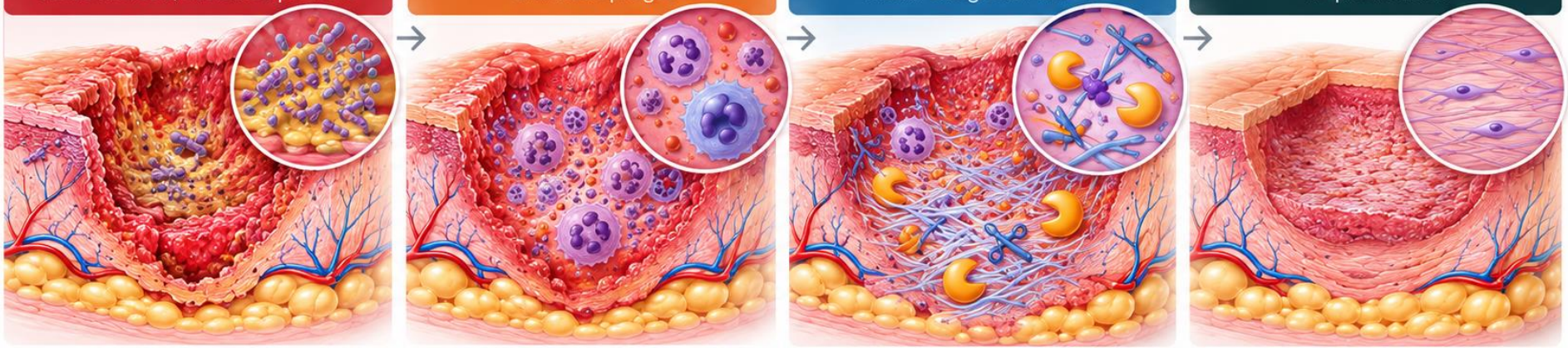
A wound arrested in the inflammatory phase

Persistent aggression
Infection, biofilm, hypoxia, tissue necrosis, sustained pressure

Prolonged inflammation
Persistent activation of neutrophils and macrophages

Excessive proteases
Degradation of the extracellular matrix and growth factors

Stagnant wound
Low cell reactivity, no proliferation



Practical definition

Non-healing wound after 4–6 weeks of evolution depending on the etiology (e.g. HS), despite appropriate care.

Key point

Identify and treat the underlying cause; do not change dressings repeatedly without a rationale.

KEY MESSAGE | A chronic wound corresponds to a healing process arrested in the inflammatory phase.

Identify the underlying cause

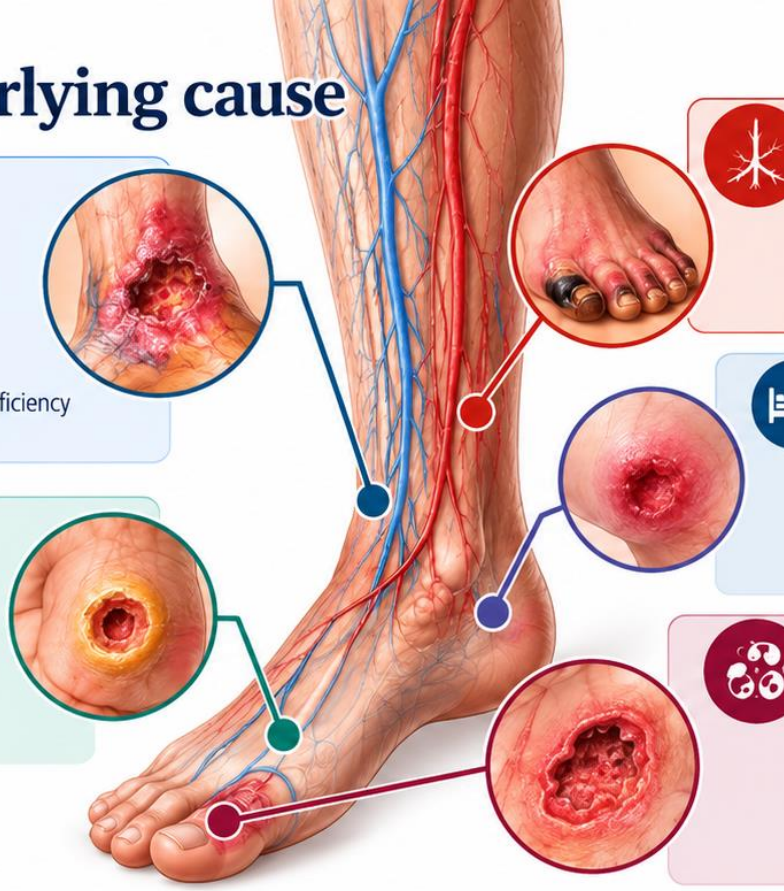
Venous ulcer

- Leg ulcer, often medial malleolus
- Pain usually mild, edema, stasis dermatitis, hyperpigmentation
- Venous Doppler ultrasound, venous insufficiency

Diabetic foot ulcer

- Plantar ulcer, painless (neuropathy)
- Peripheral hyperkeratosis (callus)

Offloading, glycemic control



Arterial ulcer

Distal foot or toes, painful (especially at night)

Cold extremity, absent pulses

ABI, revascularization

Pressure ulcer

Pressure areas: sacrum, heels, trochanters

Immobility, poor support, malnutrition

Relief of pressure, support, nutrition

Unusual cause

Undermined, painful, violaceous borders

Necrosis, suspicious nodule

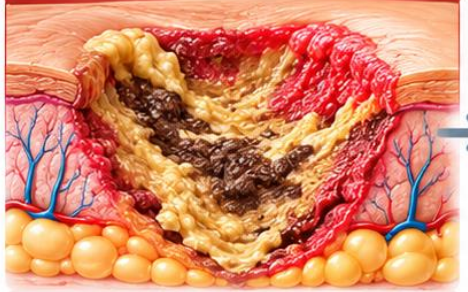
Biopsy (pyoderma gangrenosum, vasculitis, carcinoma)

KEY MESSAGE | An atypical or non-healing wound despite appropriate management requires investigation, including biopsy.

Prepare the wound bed: the TIME concept

T — Non-viable tissue

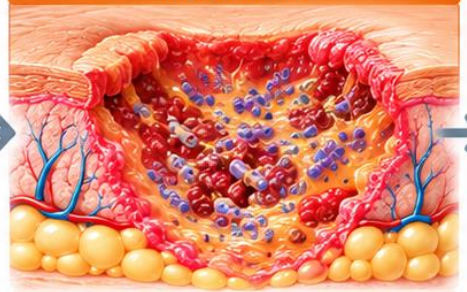
Debridement: mechanical, autolytic, surgical



- Necrosis, fibrin, devitalized tissue
- Barrier to healing
- Appropriate debridement
- Tailored to the patient

I — Infection / Inflammation

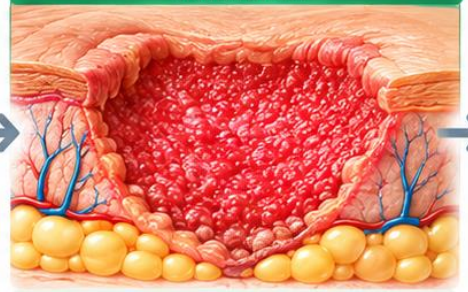
Treat infection and control bacterial load



- Signs of local infection
- Biofilm possible
- Take a sample if needed
- Antiseptics / antibiotics according to context

M — Moisture (balance)

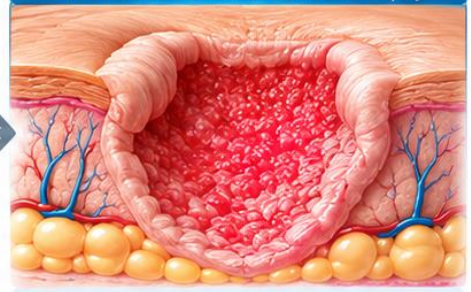
Maintain a balanced exudate environment



- Balanced exudate
- Appropriate dressing
- Prevent maceration
- Avoid desiccation

E — Edge (margins)

Manage wound edges, reassess the cause, consider biopsy



- Thickened, detached edges
- Signs of chronicity
- Reassess the cause (e.g. underlying disease)
- Consider biopsy if atypical or non-healing

Always: treat the underlying cause and correct general factors (nutrition, glycaemia, smoking), reassess after 4 weeks.

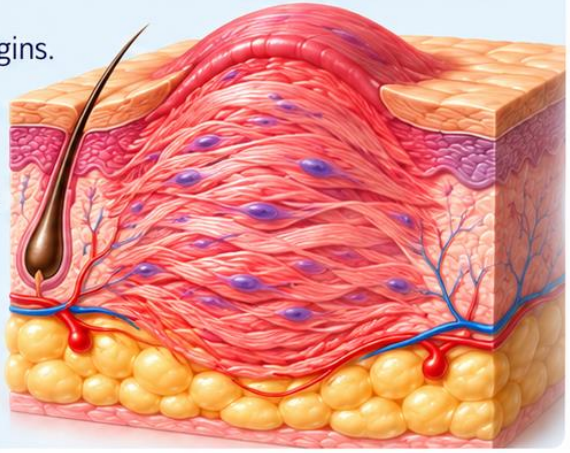
KEY MESSAGE — **TIME: Tissue, Infection, Moisture, Edge.** Without treating the underlying cause, wound bed preparation alone is insufficient.

Red, raised scar, but limited to the wound

Clinical features

- Early appearance, usually 4–6 weeks after the wound.
- Red, firm, raised scar, often pruritic.
- Respects the initial wound margins.
- Common sites: areas of tension, joints, burns.
- Spontaneous regression possible within 1–2 years.





Favouring factors

- Delayed epithelialisation beyond 10–14 days (deep burns).
- Infection, poorly closed wound.
- Excessive tension, unfavourable orientation.







KEY MESSAGE | **Hypertrophic scar:** early, raised, red scar limited to the wound, with potential for regression.

A scar that extends beyond the wound and does not regress

Clinical features

- **Firm, raised, pink to brown** lesion extending beyond the initial **wound**.
- **Progressive growth** with a “crab-like” shape.
- Often **delayed** onset (months to years).
- May be **pruritic, painful,** or sensitive.
- **No spontaneous** regression.



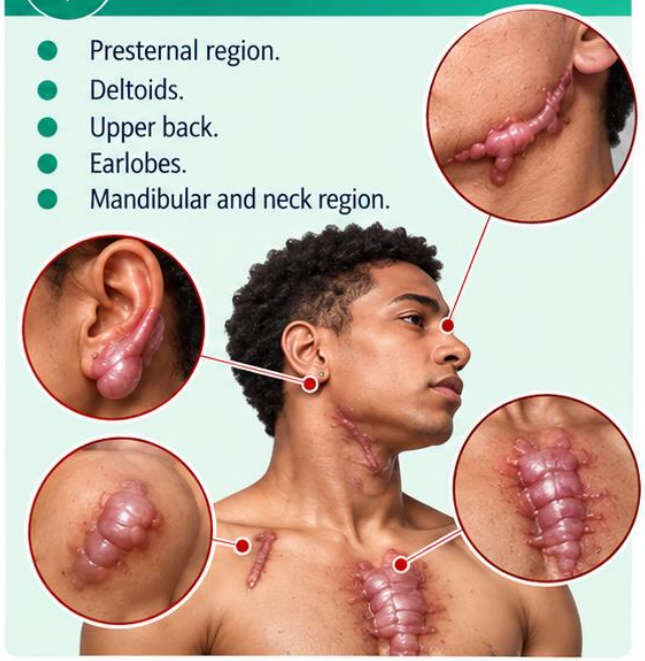
Predisposing factors

- **Darker phototypes** (IV–VI).
- **Family history**.
- **Young adults,** 10–30 years.
- **Skin trauma:** surgery, piercing, acne, varicella, vaccination, burn.



Typical sites

- Presternal region.
- Deltoids.
- Upper back.
- Earlobes.
- Mandibular and neck region.

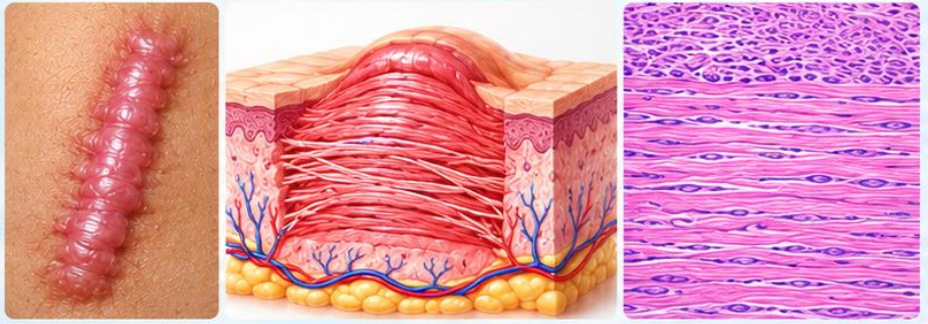


KEY MESSAGE

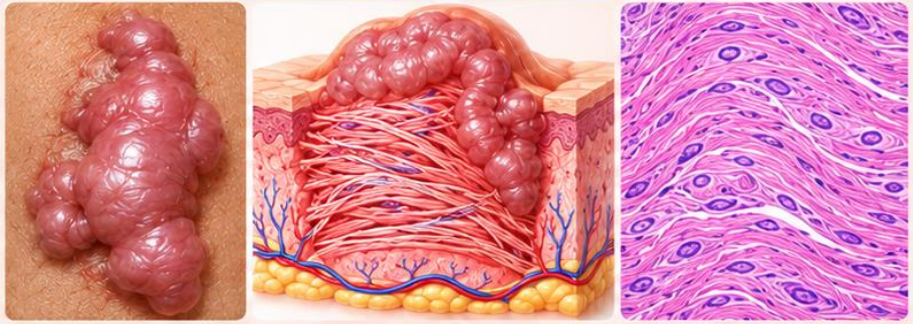
Keloid: extends beyond the initial wound, appears late, and does not regress.
Pathophysiology: chronic inflammation of the reticular dermis and mechanical tension.

Hypertrophic scar or keloid?

Hypertrophic scar
Raised scar within the original wound margin



Keloid
Raised scar extending beyond the original wound margin



Feature	Hypertrophic scar	Keloid
Time of appearance	Early: 4–6 weeks after the wound	Often later: months to years
Limits	Respects the original wound margin	Extends beyond the original wound margin
Evolution	Spontaneous regression possible (1–2 years)	Persistent, progressive, no spontaneous regression
Predisposing factors	All wound types, areas of tension, burns	Darker phototypes, family history, age 10–30 years
Histology	Parallel, well-organized collagen bundles in the dermis	Thick, hyalinized, disorganized collagen bundles
Recurrence after excision alone	Possible if tension persists	Very frequent (45–100%)

KEY MESSAGE | **Differentiate** before any treatment: keloids almost always recur after excision alone.

Prevent and treat: a stepwise strategy

Prevention

Reduce tension, avoid infection, photoprotection, adhesive strips





1st line

Silicone gel or sheet (recommended), pressure therapy, massage









Injections and physical therapies

Intralesional corticosteroids (spaced 4–8 weeks according to published practice), 5-fluorouracil, cryotherapy, laser



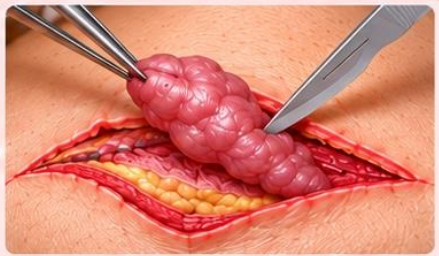






Surgery + adjuvant therapy

Excision should always be combined with adjuvant therapy (corticosteroids, radiotherapy, pressure) for keloids







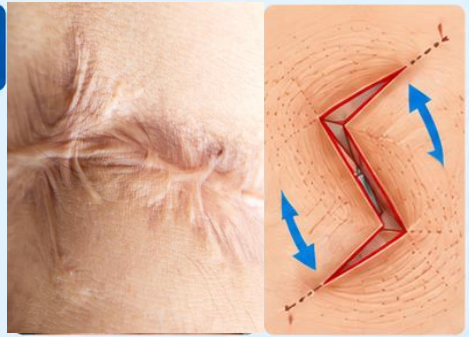


KEY MESSAGE | **Silicone** is the reference non-invasive treatment, for both prevention and treatment.

Other abnormal scars

Contracture (tight scar)

- Burns, flexion creases, functional limitation.
- Physiotherapy, Z-plasty.



Widened scar

- Tension, dehiscence, growth.
- Common on the back and shoulders.



Atrophic scar

- Depressed: sequelae of acne or varicella.
- Collagen deficiency.



Dyschromic scar

- Hypo- or hyperpigmented, especially in darker skin.
- Photoprotection.

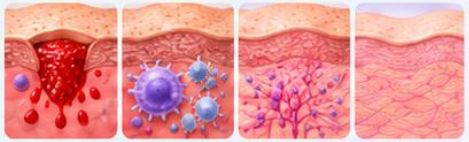


KEY MESSAGE | **Each type of abnormal scar requires specific management:** establish the diagnosis before treatment.

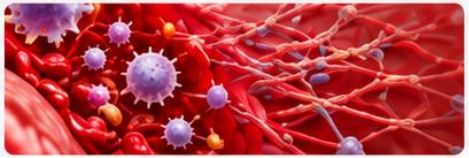
CONCLUSION

Ten key messages

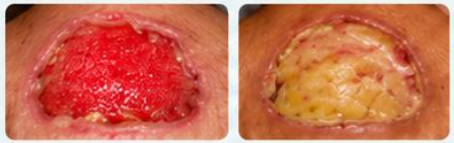
1. **Four overlapping phases:** hemostasis, inflammation (D0–5), proliferation (D3–21), remodeling (up to 12–24 months).



2. The clot is a growth-factor-rich matrix; prolonged inflammation blocks healing.



3. **Healthy granulation**: bright red; pale or friable granulation: ischemia, infection, malnutrition.



4. A scar reaches a maximum of 70–80% of normal skin strength and should be assessed at 12–18 months.



5. **Local factors**: infection, ischemia, necrosis, tension, inappropriate environment.



6. **General factors**: diabetes, smoking (stop ≥ 4 weeks before surgery), malnutrition, age, medications.



7. **Primary intention**: approximated edges; **secondary intention**: tissue loss; **directed healing**: secondary intention managed step by step.



8. **Chronic wound** (> 4–6 weeks): treat the cause; biopsy if atypical.



9. **Hypertrophic scar**: early, limited to the wound, may regress; **keloid**: extends beyond the wound, appears later, predisposition, does not regress.



10. **Silicone is first-line**; excision alone for keloid has a high recurrence rate.



Bibliography (1/2)

1. Singer AJ, Clark RAF. Cutaneous wound healing. **N Engl J Med.** 1999;341(10):738–46.
2. Gurtner GC, Werner S, Barrandon Y, Longaker MT. Wound repair and regeneration. **Nature.** 2008;453(7193):314–21.
3. Eming SA, Martin P, Tomic-Canic M. Wound repair and regeneration: mechanisms, signaling, and translation. **Sci Transl Med.** 2014;6(265):265sr6.
4. Rodrigues M, Kosaric N, Bonham CA, Gurtner GC. Wound healing: a cellular perspective. **Physiol Rev.** 2019;99(1):665–706.
5. Winter GD. Formation of the scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. **Nature.** 1962;193:293–4.
6. Guo S, DiPietro LA. Factors affecting wound healing. **J Dent Res.** 2010;89(3):219–29.
7. Gosain A, DiPietro LA. Aging and wound healing. **World J Surg.** 2004;28(3):321–6.
8. Sørensen LT, Karlsmark T, Gottrup F. Abstinence from smoking reduces incisional wound infection: a randomized controlled trial. **Ann Surg.** 2003;238(1):1–5.
9. Sørensen LT. Wound healing and infection in surgery: the clinical impact of smoking and smoking cessation: a systematic review and meta-analysis. **Arch Surg.** 2012;147(4):373–83.

Bibliography (2/2)

10 Sørensen LT. Wound healing and infection in surgery: the pathophysiological impact of smoking, smoking cessation, and nicotine replacement therapy: a systematic review. **Ann Surg**. 2012;255(6):1069–79.

11 Armstrong DG, Boulton AJM, Bus SA. Diabetic foot ulcers and their recurrence. **N Engl J Med**. 2017;376(24):2367–75.

12 Haesler E, editor. **EPUAP/NPIAP/PPPIA**. Prevention and treatment of pressure ulcers/injuries: clinical practice guideline. The international guideline. 2019.

13 Zitelli JA. Wound healing by secondary intention: a cosmetic appraisal. **J Am Acad Dermatol**. 1983;9(3):407–15.

14 Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: a systematic approach to wound management. **Wound Repair Regen**. 2003;11(Suppl 1):S1–28.

15 Ogawa R. Keloid and hypertrophic scars are the result of chronic inflammation in the reticular dermis. **Int J Mol Sci**. 2017;18(3):606.

16 Mustoe TA, Cooter RD, Gold MH, et al. International clinical recommendations on scar management. **Plast Reconstr Surg**. 2002;110(2):560–71.

17 Monstrey S, Middelkoop E, Vranckx JJ, et al. Updated scar management practical guidelines: non-invasive and invasive measures. **J Plast Reconstr Aesthet Surg**. 2014;67(8):1017–25.

18 Meaume S, Le Pillouer-Prost A, Richert B, Roseeuw D, Vadoud J. Management of scars: updated practical guidelines and use of silicones. **Eur J Dermatol**. 2014;24(4):435–43.

19 Gauglitz GG. Management of keloids and hypertrophic scars: current and emerging options. **Clin Cosmet Investig Dermatol**. 2013;6:103–14.

20 French National Authority for Health (HAS/CNEDiMTS). Dressings: recommended indications and uses. Saint-Denis La Plaine; HAS; April 2011.

Thank you for your attention



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