

Excessive Scarring

Hypertrophic scars • Keloids • Proud flesh • Contractures

Tissue repair and its pathologic deviations

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Scope: Excessive scarring and abnormal repair

- Excess matrix deposition may produce raised scars that alter skin contour.
- Excess granulation tissue may obstruct epithelial resurfacing.
- Excess wound contraction may deform tissue and limit joint movement.

**Excess
collagen**

Raised scars

**Excess
granulation**

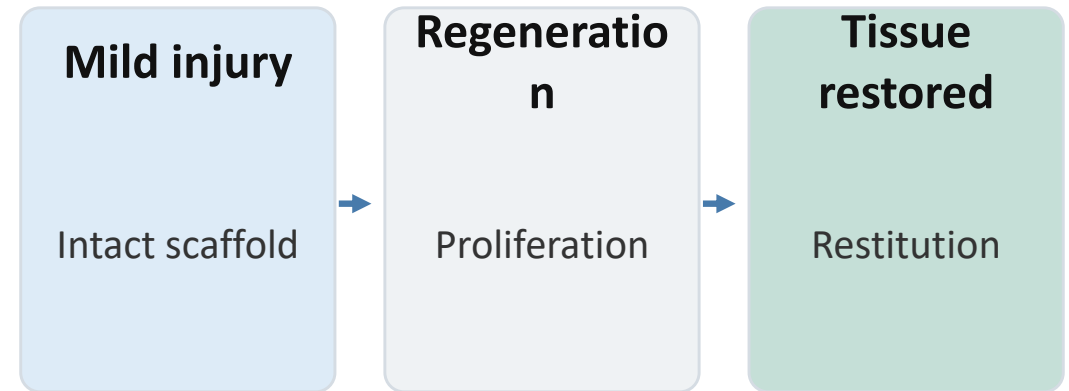
Proud flesh

**Excess
contraction**

Contracture

Normal repair must be understood first

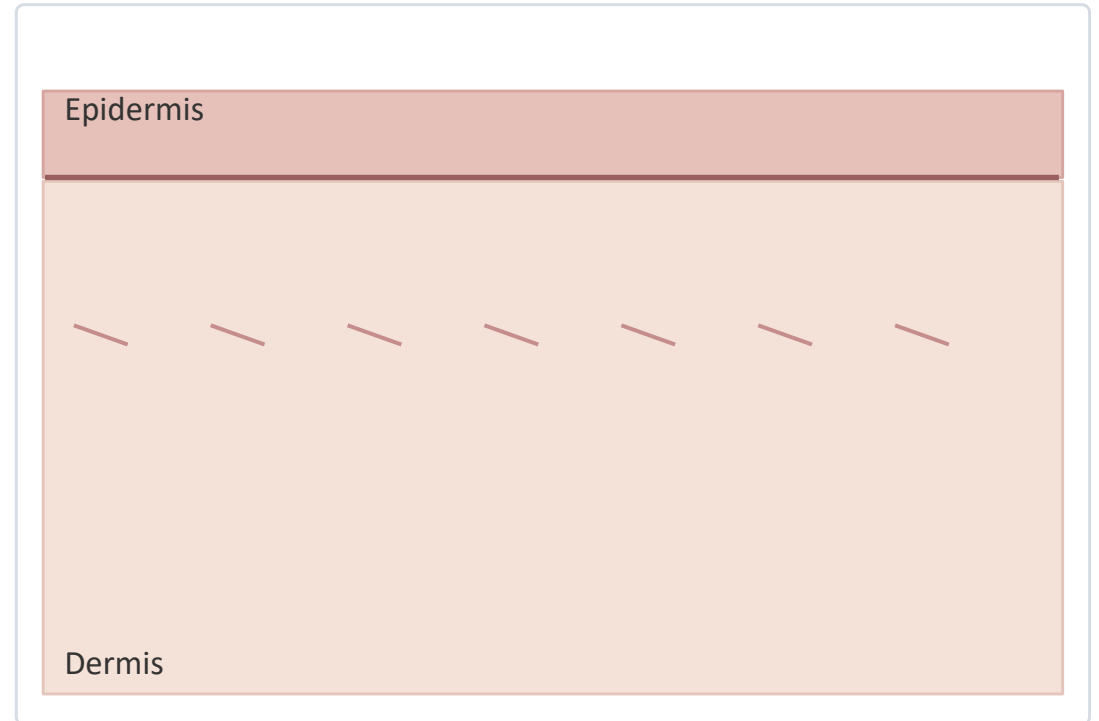
- Tissue repair replaces or restores structures after injury.
- Regeneration restores cells when the supporting framework remains suitable.
- Scarring deposits connective tissue when full restoration is not possible.



More severe injury → connective-tissue scar

Why some wounds heal with a scar

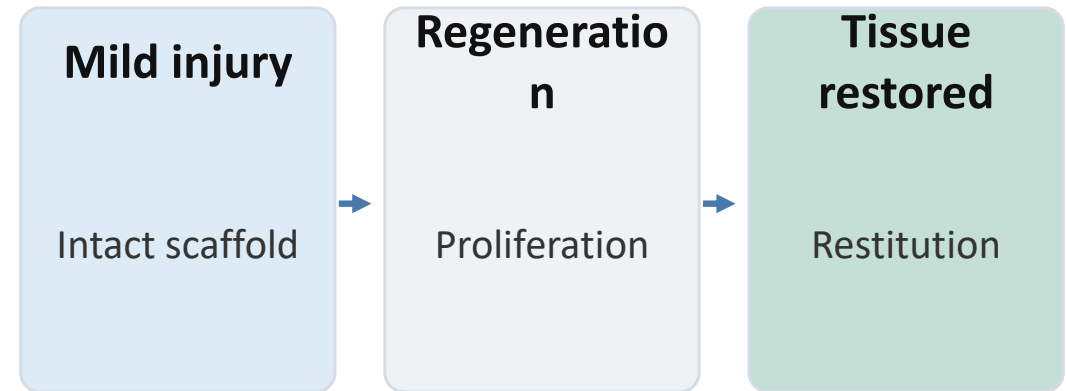
- Deeper injury can damage the connective-tissue framework.
- Loss of a supporting scaffold makes complete restitution difficult.
- Collagen-rich connective tissue provides mechanical continuity, not full regeneration.



Schematic

Repair and regeneration are different outcomes

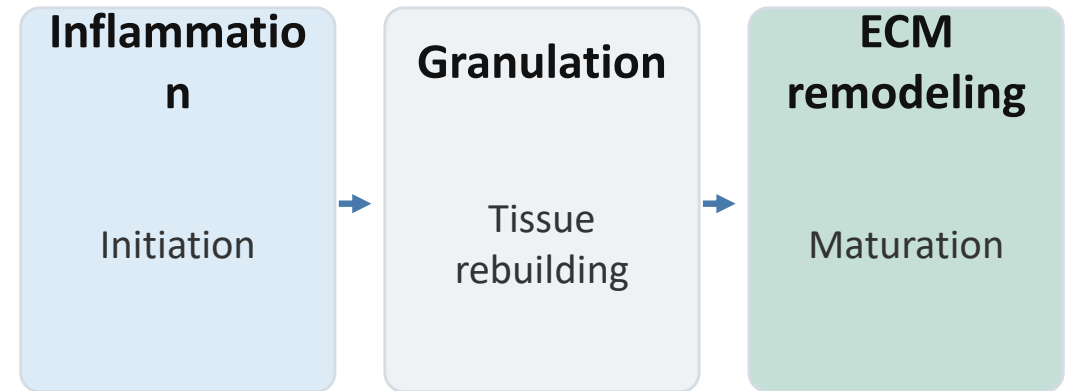
- Regeneration restores components of the original tissue.
- Scar formation patches defects with collagen-rich connective tissue.
- A wound may use both processes in varying proportions.



Order shown conceptually; Stages overlap

Three major phases of cutaneous healing

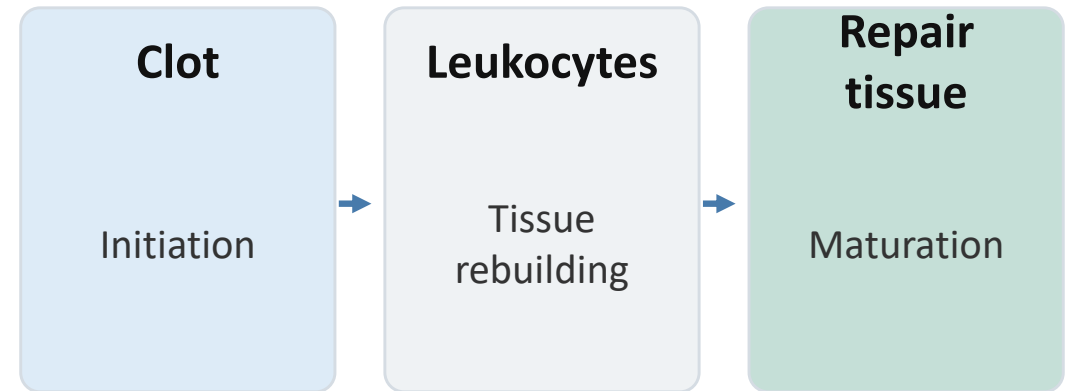
- Inflammation clears injured material and initiates the repair response.
- Granulation tissue brings cells, vessels and extracellular matrix into the defect.
- Extracellular-matrix remodeling changes the early repair tissue into a scar.



Order shown conceptually; Stages overlap

A clot initiates structural repair

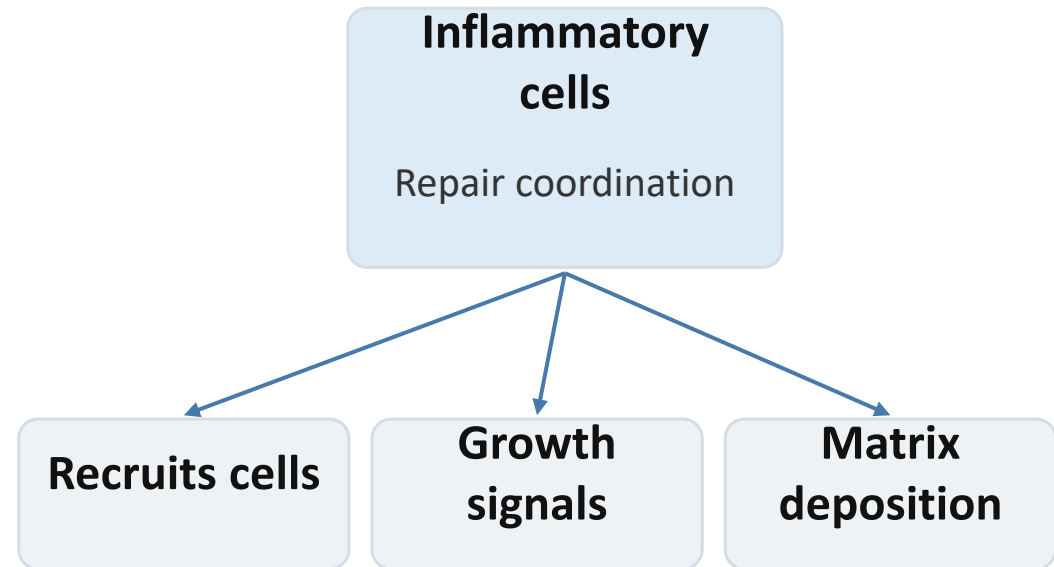
- A platelet-rich hemostatic plug forms within minutes of injury.
- It controls bleeding and provides an initial provisional scaffold.
- Subsequent inflammatory and reparative cells enter the injured region.



Order shown conceptually; Stages overlap

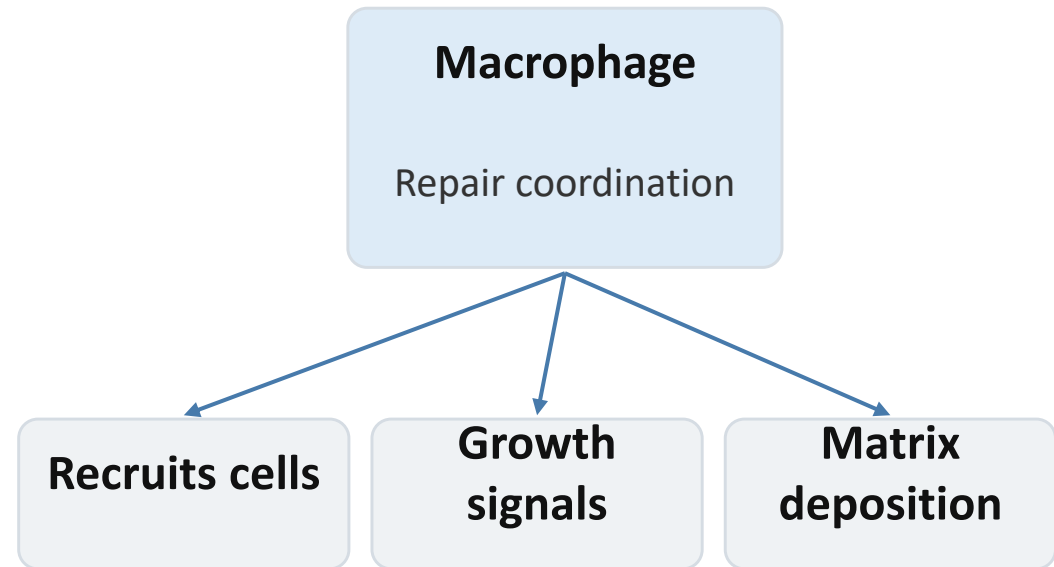
Inflammatory cells prepare the wound

- Neutrophils appear early at wound margins.
- Monocytes and macrophages subsequently become important.
- Removal of the offending cause precedes stable tissue reconstruction.



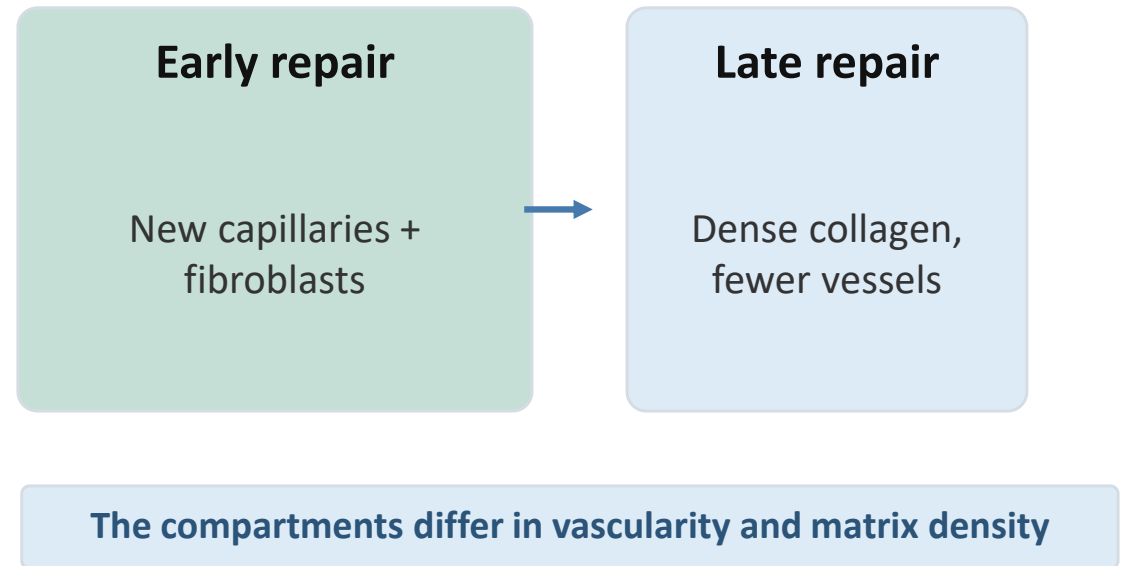
Macrophages coordinate repair

- Macrophages help eliminate injurious material.
- They produce mediators that recruit and activate repair-associated cells.
- Alternatively activated macrophages contribute to the shift toward connective-tissue deposition.



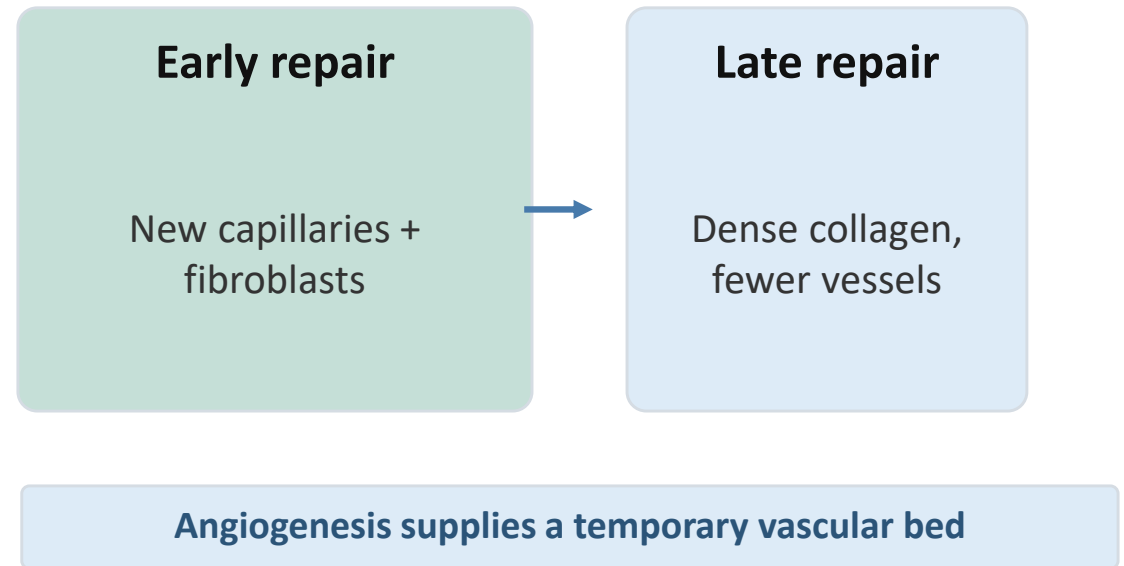
Granulation tissue is an active repair compartment

- New capillaries, fibroblasts and loose matrix characterize granulation tissue.
- Edema and inflammatory cells can still be present.
- It is distinct from the later dense, relatively avascular scar.



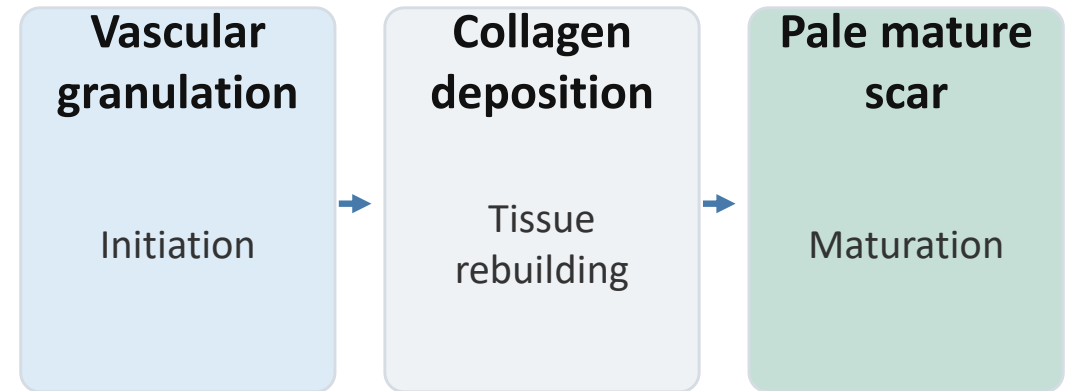
Angiogenesis supplies the healing wound

- New capillaries sprout from existing vessels.
- Endothelial migration and proliferation precede maturation.
- Pericyte recruitment and basement-membrane deposition stabilize vessels.



Granulation tissue evolves into a mature scar

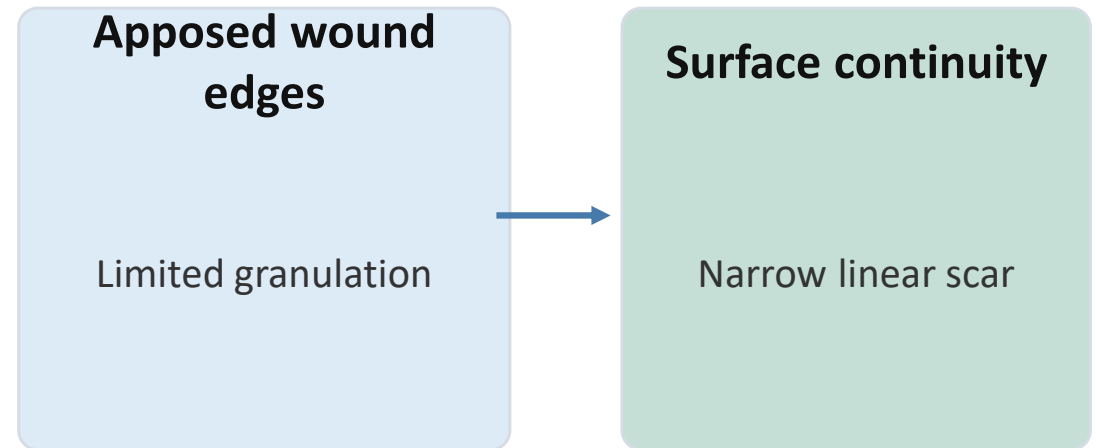
- Early granulation tissue is vascular and cellular.
- Progressive collagen deposition strengthens the healing site.
- With maturation, many vessels regress and dense connective tissue predominates.



Order shown conceptually; Stages overlap

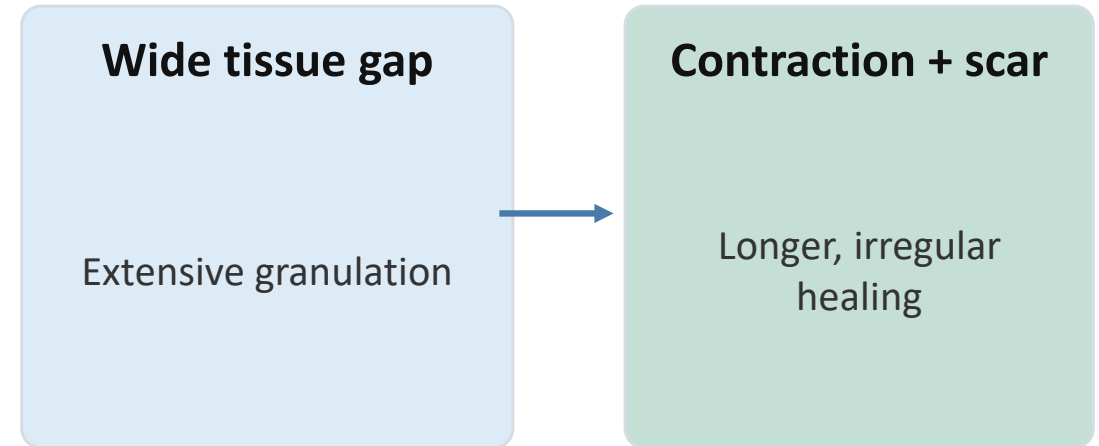
Primary intention limits the tissue defect

- Well-apposed clean incisions leave a small gap.
- Inflammation and granulation-tissue formation are comparatively limited.
- A neat linear scar generally results.



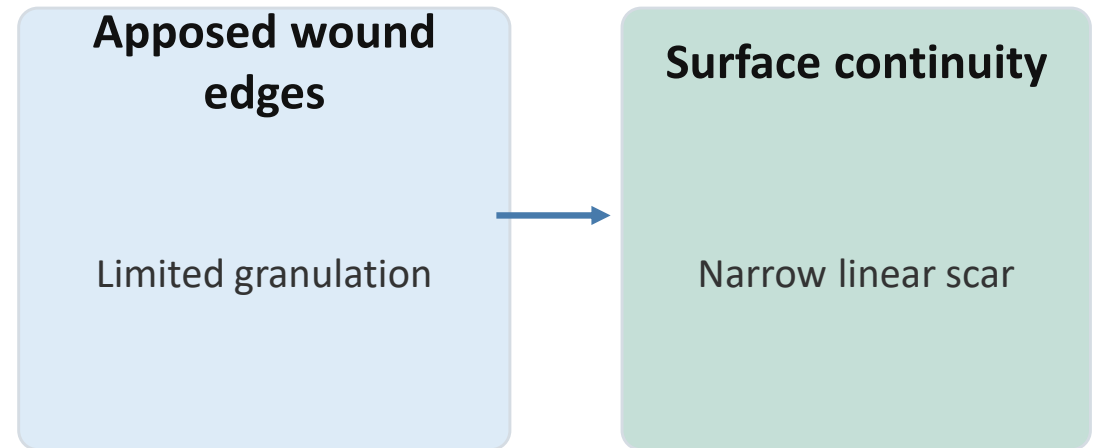
Secondary intention involves a larger deficit

- Large or irregular wounds need more tissue replacement.
- Inflammation and granulation tissue are more extensive.
- Wound contraction and irregular scarring become more prominent.



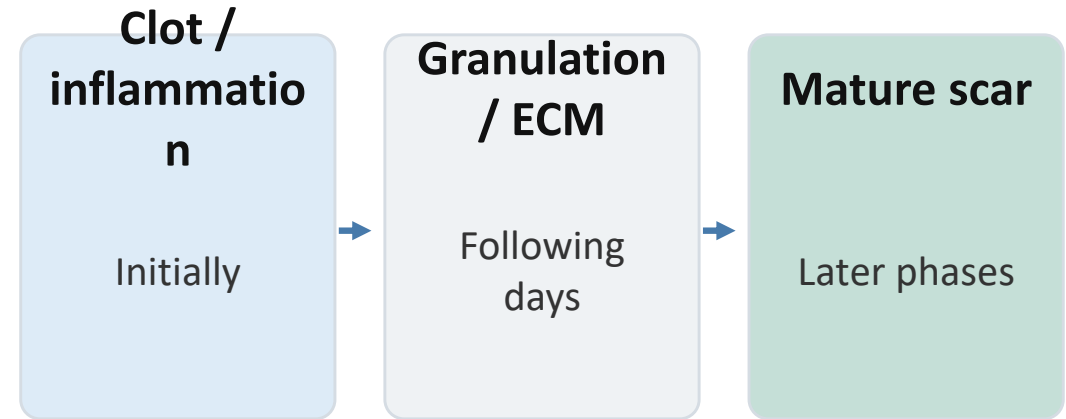
Primary versus secondary intention

- Wound dimensions govern how much tissue must be replaced.
- Secondary healing generally entails more contraction.
- These are healing patterns, not diagnoses of hypertrophic scarring.



Events evolve over days to weeks

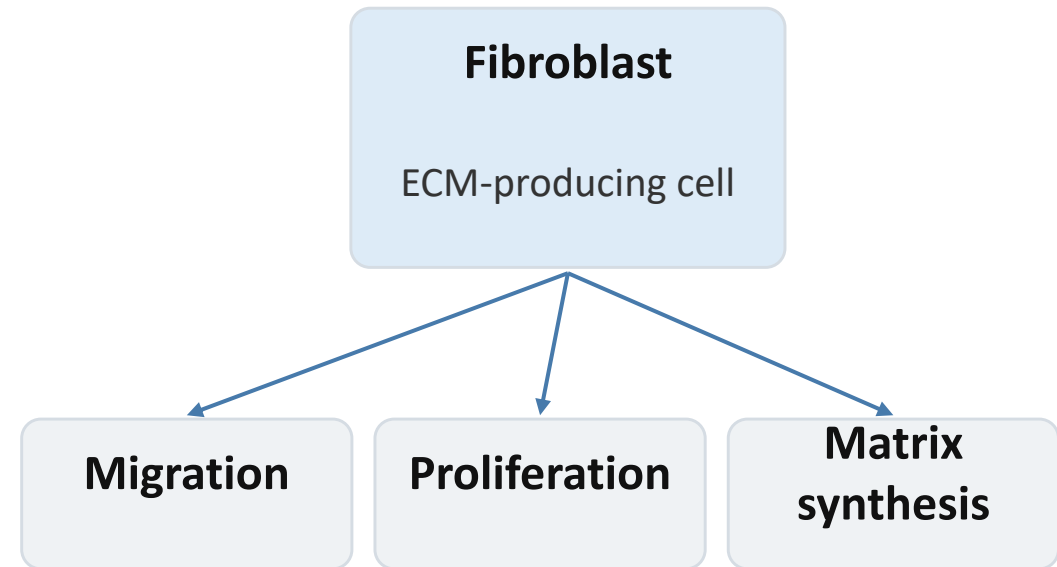
- Initially, a clot and inflammatory cells dominate.
- Granulation tissue and collagen deposition emerge over the following days.
- Later remodeling turns the repair tissue into a mature scar.



Order shown conceptually; Stages overlap

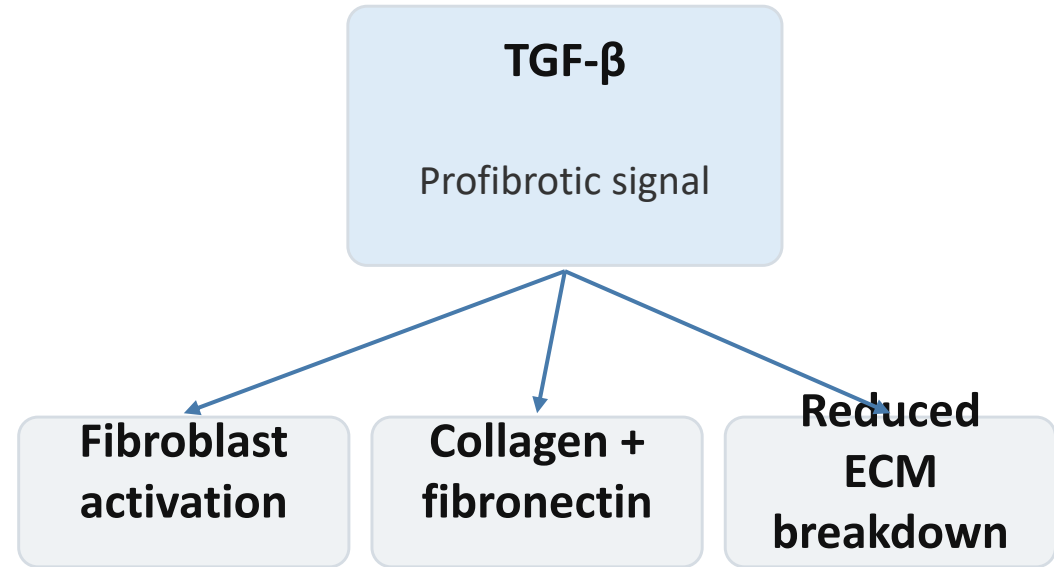
Fibroblasts create the fibrous component

- Fibroblasts migrate and proliferate within the repair site.
- They synthesize collagen and other extracellular-matrix proteins.
- Their continued synthetic activity is central to scar formation.



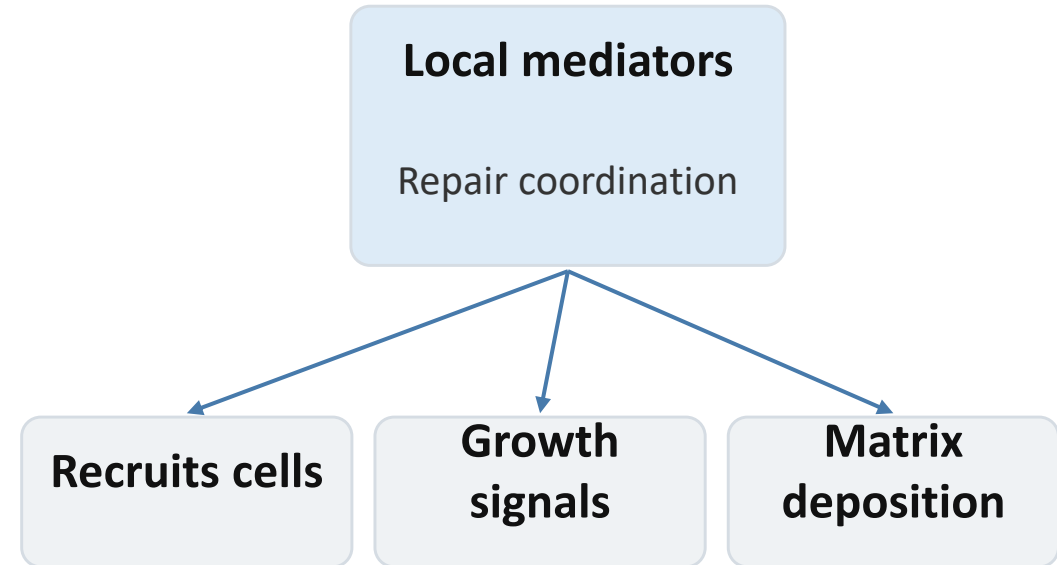
TGF- β is a principal fibrogenic signal

- TGF- β enhances fibroblast migration and proliferation.
- It promotes collagen and fibronectin synthesis.
- It also limits extracellular-matrix degradation by inhibiting metalloproteinases.



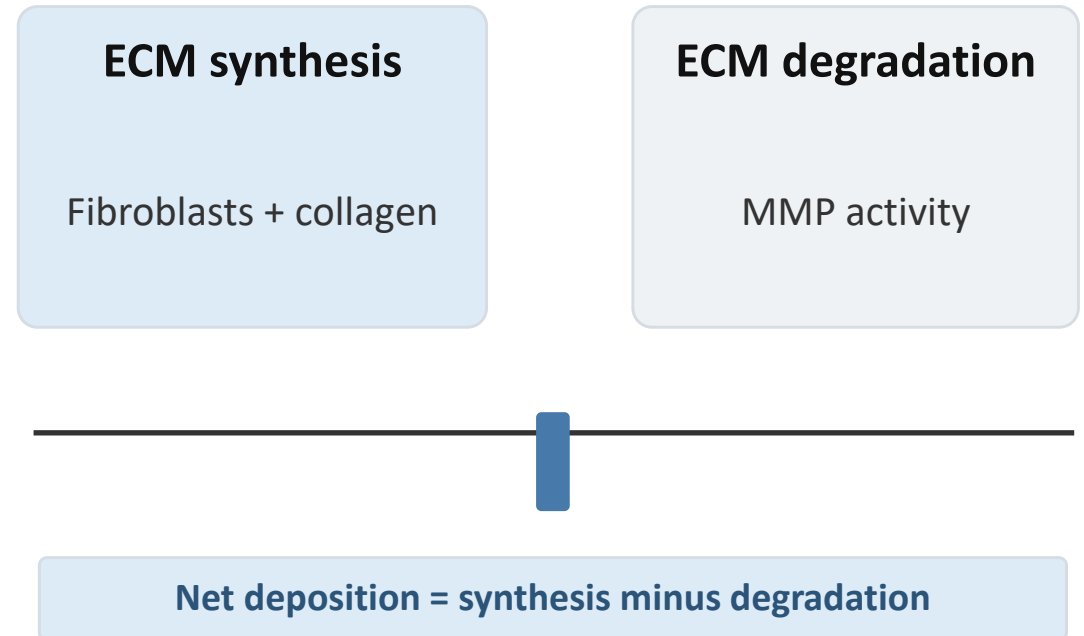
Several signals cooperate in fibroblast recruitment

- PDGF and FGF-2 contribute to migration and proliferation during repair.
- Activated inflammatory cells provide a local source of growth factors.
- The integrated repair response depends on cell–matrix interactions.



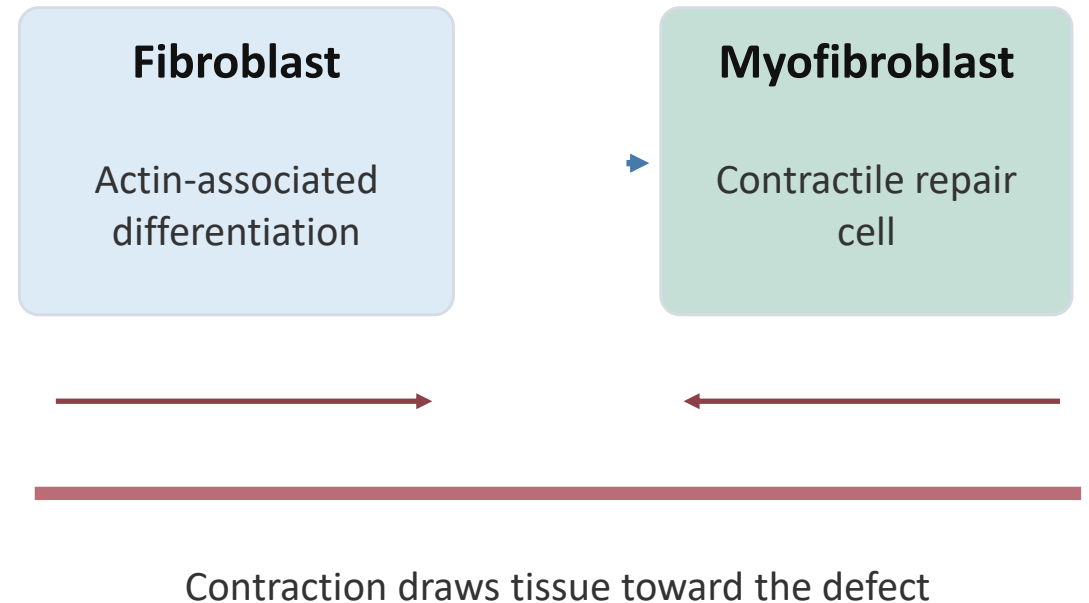
Collagen deposition builds wound strength

- Collagen accumulation contributes substantially to tensile stability.
- Fibroblasts become more synthetically active as healing progresses.
- Dense scar tissue is mechanically useful but is not normal skin architecture.



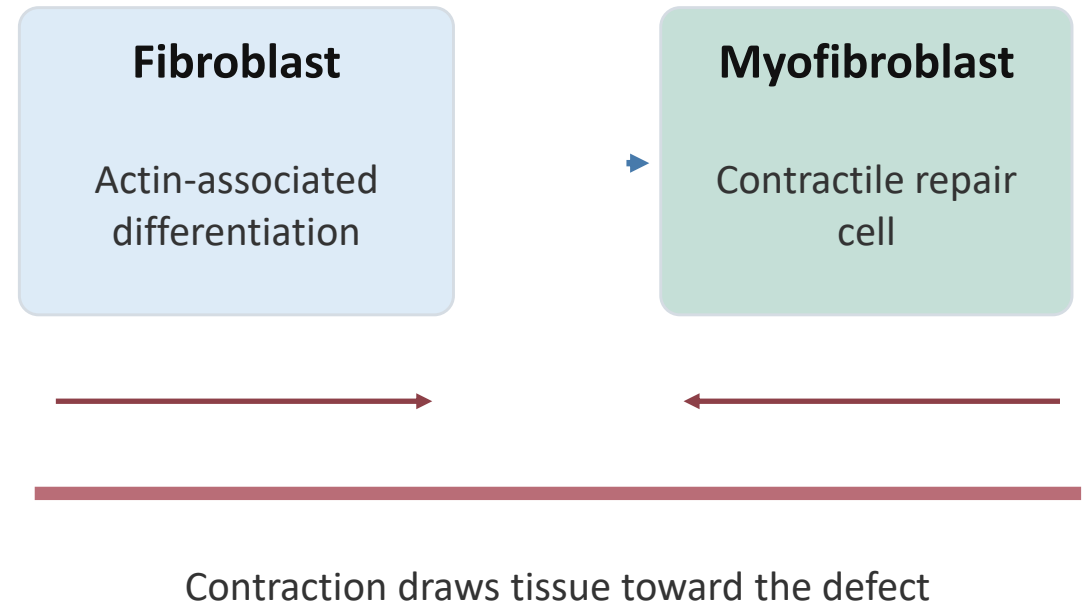
Myofibroblasts link matrix production and contraction

- Some fibroblasts acquire smooth-muscle-like actin features.
- These differentiated cells are called myofibroblasts.
- They contribute to contraction of the scar during healing.



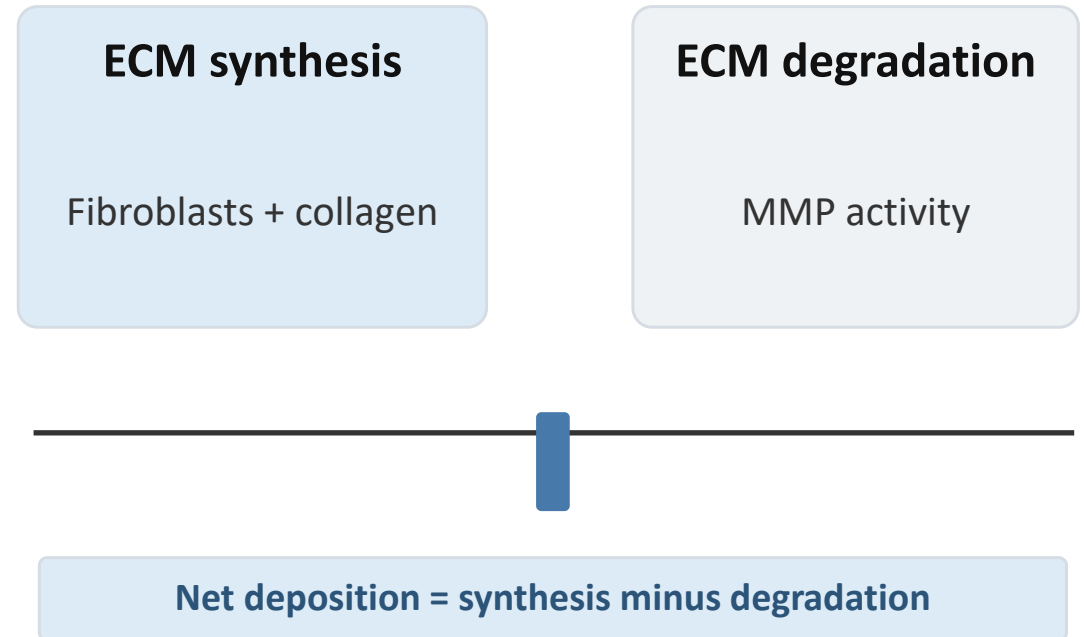
Normal wound contraction is adaptive

- Contraction reduces the size of an open wound.
- Myofibroblast-driven forces act within the repair tissue.
- The same mechanism can become harmful when excessive.



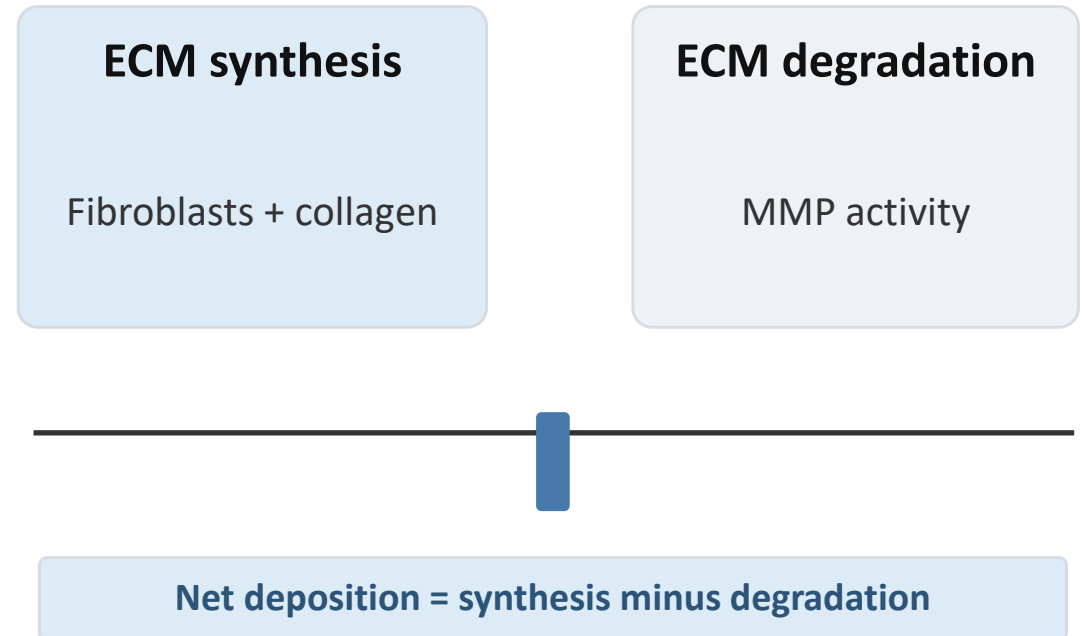
Matrix degradation is necessary for remodeling

- Scar maturation is not solely an accumulation process.
- Matrix metalloproteinases (MMPs) degrade components of extracellular matrix.
- Remodeling depends on regulated deposition and removal.



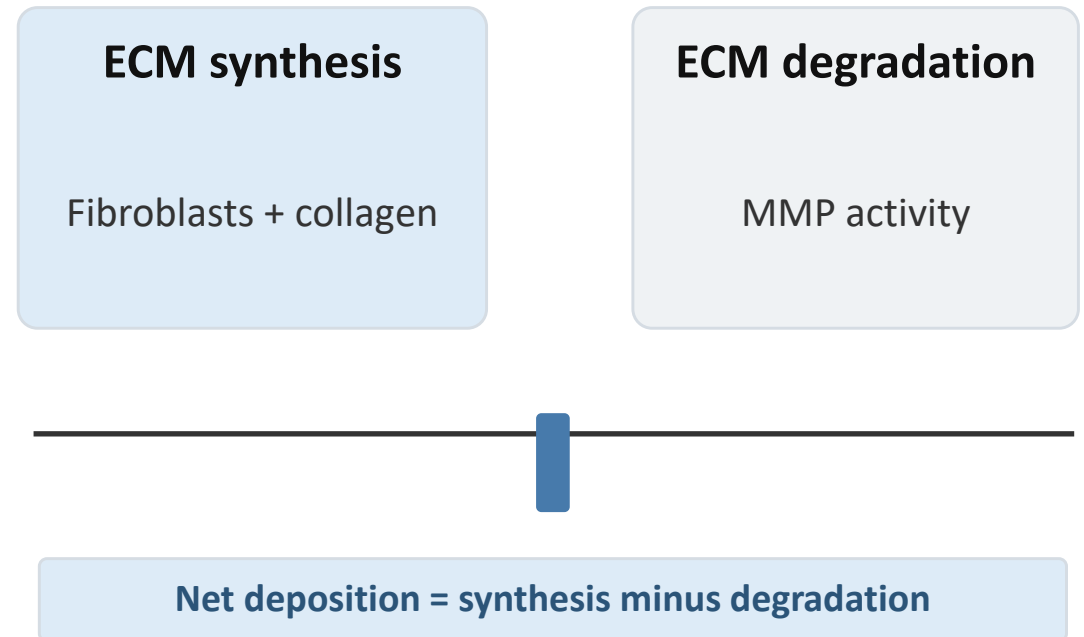
MMPs remodel collagen and matrix

- MMPs include enzymes that degrade collagen and other matrix proteins.
- They are generated as inactive precursors requiring activation.
- Their activity is localized and tightly controlled during repair.



TIMPs restrain matrix breakdown

- Tissue inhibitors of metalloproteinases (TIMPs) inhibit active MMPs.
- They protect deposited matrix from uncontrolled degradation.
- Net scar remodeling reflects interplay between synthesis and removal.



Excessive scarring reflects abnormal repair output

- Too much deposited collagen can elevate the scar above skin level.
- Overabundant granulation tissue can prevent epithelial closure.
- An excessive contractile response creates deforming contractures.

**Excess
collagen**

Raised scars

**Excess
granulation**

Proud flesh

**Excess
contraction**

Contracture

Classify the clinically relevant deviations

- Hypertrophic scar: Raised collagen-rich scar with a tendency to regress.
- Keloid: Scar growth outside the original wound boundary without regression.
- Proud flesh and contracture reflect other exaggerated components of healing.

**Excess
collagen**

Raised scars

**Excess
granulation**

Proud flesh

**Excess
contraction**

Contracture

Hypertrophic scar: A collagen-rich raised lesion

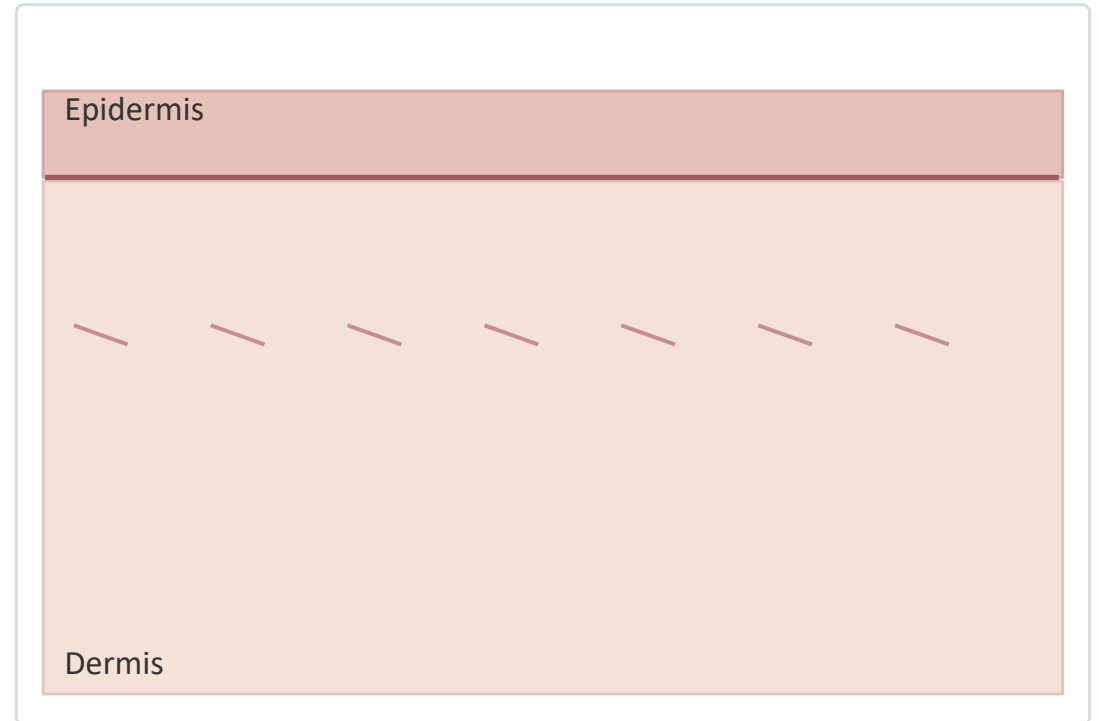
- The scar projects above the surrounding skin surface.
- Excess collagen underlies the abnormal contour.
- The process occurs in the setting of cutaneous wound repair.



Schematic

Hypertrophic scars follow deeper skin injury

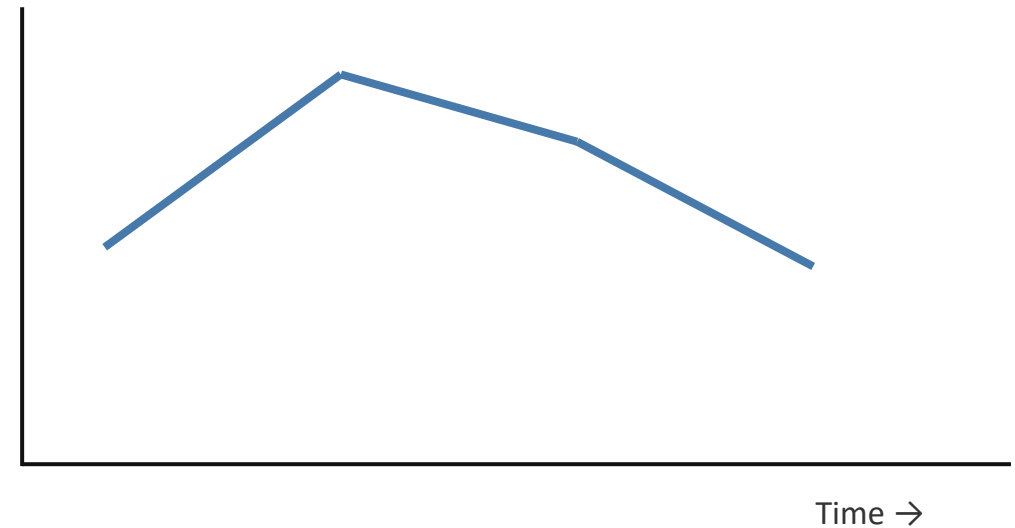
- Thermal injury is a typical antecedent.
- Traumatic injury involving the deep dermis is also characteristic.
- The depth and extent of dermal damage help explain increased repair demand.



Schematic

Hypertrophic scars can enlarge early

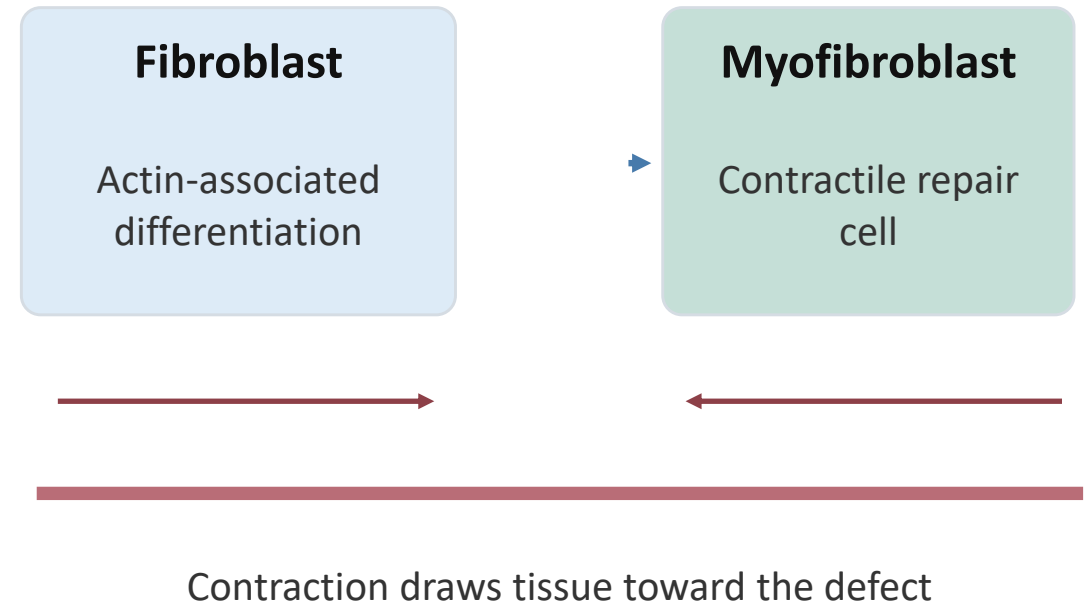
- These scars as often growing rapidly.
- The early reparative lesion may be especially cellular and contractile.
- Growth rate alone does not distinguish a hypertrophic scar from a keloid.



Qualitative schematic; No quantitative curve is implied

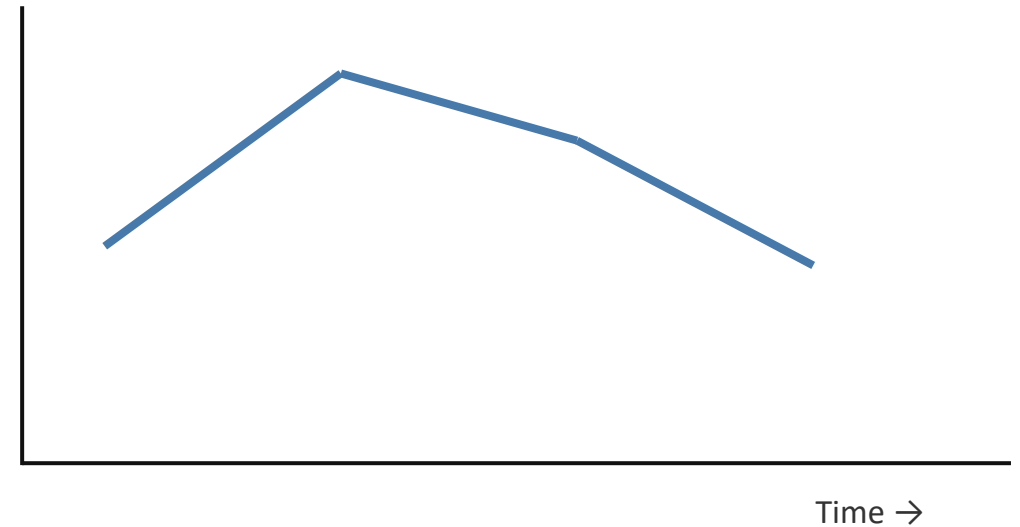
Myofibroblasts are prominent in hypertrophic scars

- Hypertrophic scars often contain abundant myofibroblasts.
- Their presence links the lesion to the contractile repair response.
- The cell population helps explain why scar contraction can accompany collagen deposition.



Regression is a key hypertrophic-scar behavior

- Hypertrophic scars tend to regress over several months.
- Their natural history contrasts with the non-regressing keloid.
- Time course should be considered alongside anatomic boundaries.



Qualitative schematic; No quantitative curve is implied

Morphologic interpretation: Hypertrophic scars

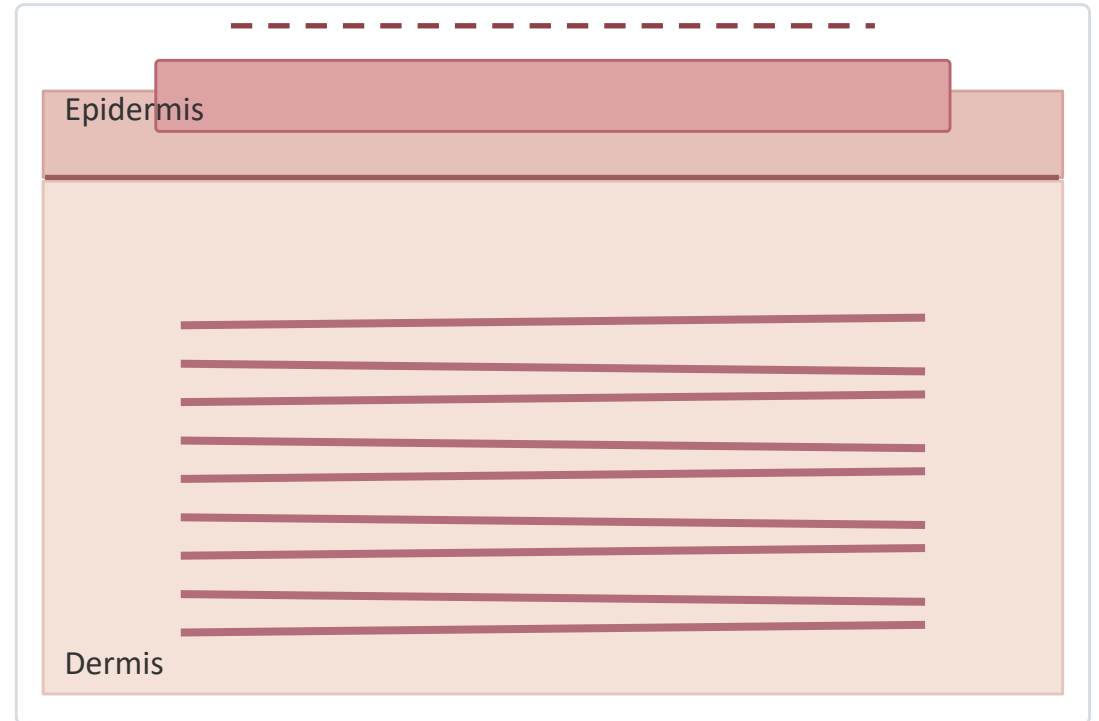
- The key source-described finding is excessive collagen forming a raised scar.
- The lesion remains linked to the site of prior dermal injury.
- A single static image cannot establish whether subsequent regression occurs.



Schematic

Keloid: Extension beyond the initial wound

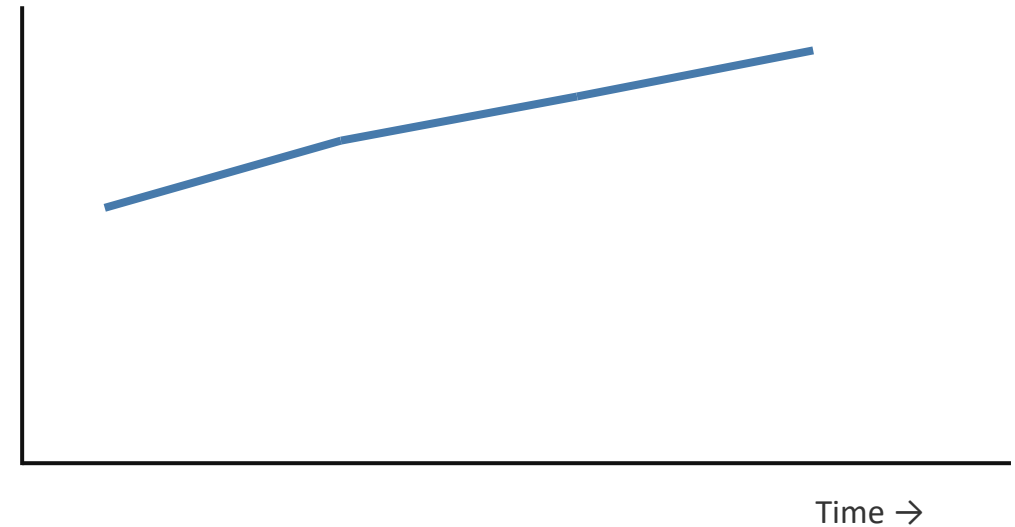
- A keloid continues past the boundaries of the original injury.
- The growth is scar tissue rather than ordinary epithelial spread.
- An accurate wound history makes the boundary criterion interpretable.



Schematic

Keloids persist rather than regress

- Keloids as scars that do not regress.
- Persistence separates them from the usual hypertrophic-scar trajectory.
- A raised scar is not automatically a keloid without these features.



Qualitative schematic; No quantitative curve is implied

Keloid formation shows individual predisposition

- An apparent individual predisposition.
- Predisposition is a clinical observation, not a quantified inherited risk in this source.
- Do not infer a specific gene or inheritance pattern from the assigned text.

**Observed
susceptibility**

Clinical
interpretation

**Not a gene
test**

Clinical
interpretation

**Clinical
history**

Clinical
interpretation

Clinical appearance varies with skin tone

- Keloids may be erythematous on lighter skin tones.
- They may be hyperpigmented on darker skin tones.
- Color alone is insufficient: Extension and persistence define the lesion.

Erythematous

Clinical
interpretation

Hyperpigmented

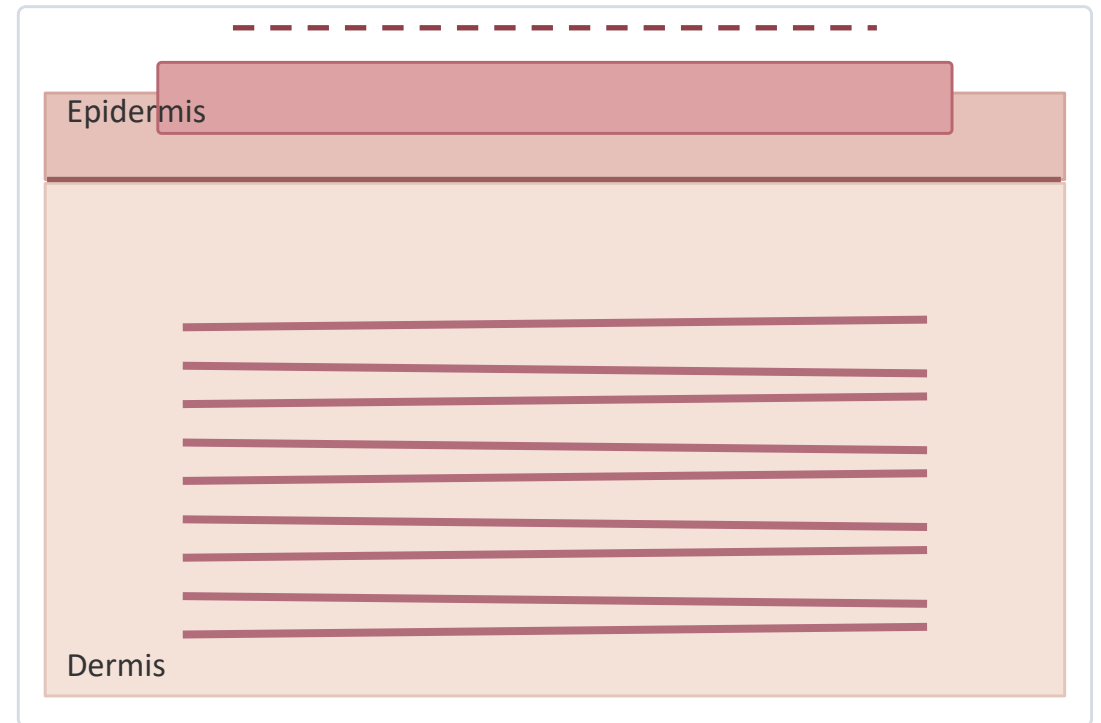
Clinical
interpretation

Boundary matters

Clinical
interpretation

Keloid histology: Abundant dermal connective tissue

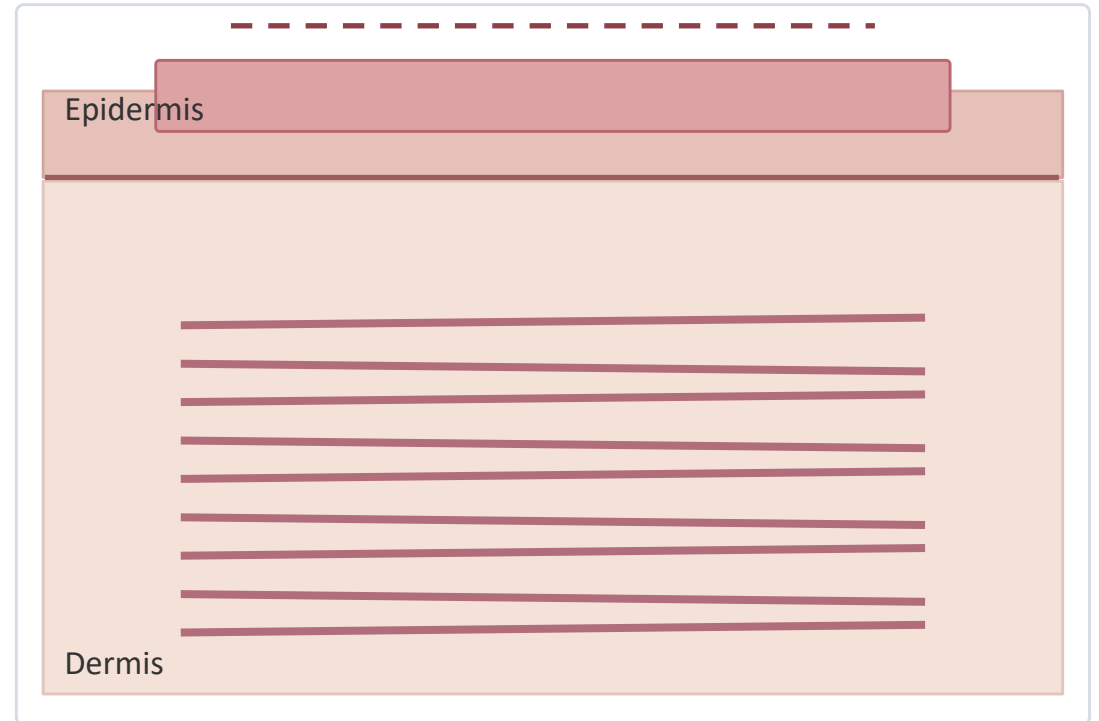
- The textbook figure emphasizes thick connective tissue deposited in the dermis.
- Histology supports an excessive collagen-deposition process.
- Boundary and natural-history information remain clinical rather than purely microscopic.



Schematic

Anatomic interpretation of excess dermal collagen

- Epidermis lies above the collagen-containing dermis.
- An excessive dermal collagen mass changes surface elevation.
- The schematic represents architecture, not an authentic H&E section.



Schematic

Hypertrophic scars versus keloids

- Both are excessive scarring responses with raised collagen-rich tissue.
- Keloid extension beyond original wound boundaries is decisive.
- Regression over months favors the hypertrophic-scar pathway.

Hypertrophic scar	Keloid
Usually within wound	Extends beyond wound
Tends to regress	Does not regress
Raised collagen scar	Raised collagen scar

Boundary is a spatial diagnostic concept

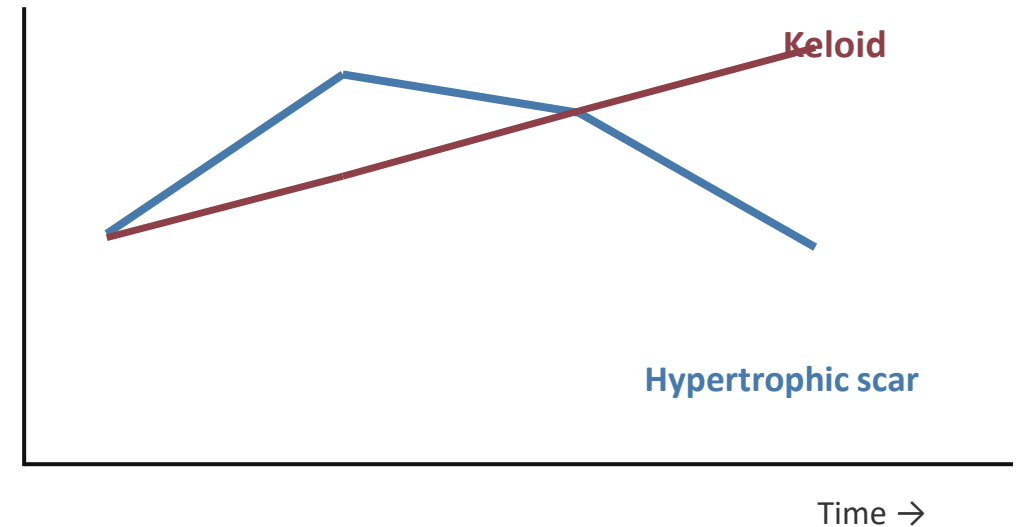
- Record the extent of the original injury if available.
- Compare the eventual scar perimeter with that original outline.
- Outward extension is the textbook distinction for keloid.



Dashed idea: Initial wound region vs subsequent scar outline

Evolution over time adds diagnostic information

- An enlarging early scar may be hypertrophic or keloidal.
- A tendency to regress supports hypertrophic scar.
- Persistent growth beyond the injury supports keloid.



Qualitative schematic; No quantitative curve is implied

Color and elevation are not enough

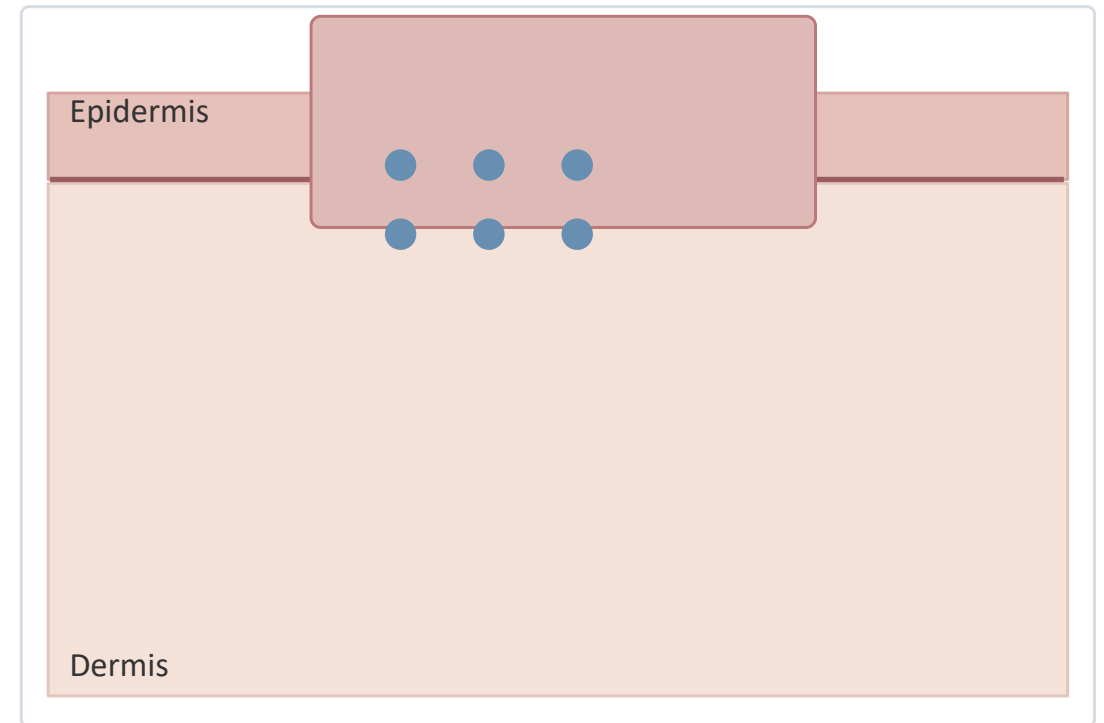
- Erythema and hyperpigmentation describe appearance, not biological category.
- Elevation can occur in either collagen-rich scar type.
- The joint assessment of boundary and trajectory is more discriminating.



Dashed idea: Initial wound region vs subsequent scar outline

Exuberant granulation tissue is a different problem

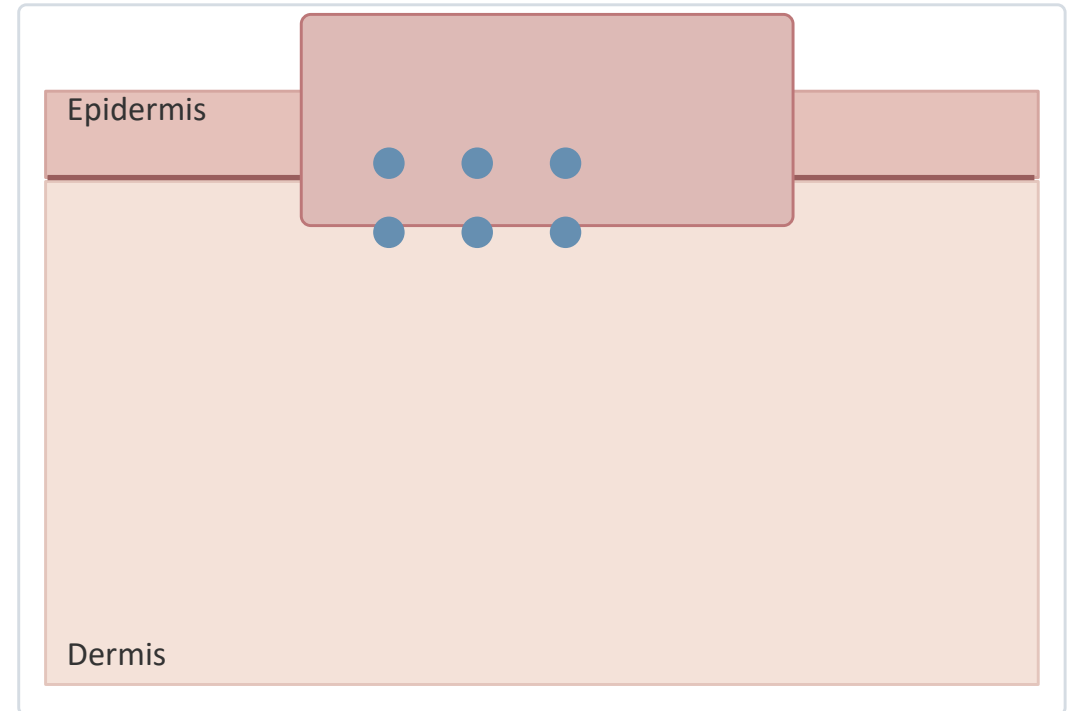
- This lesion consists of excessive granulation tissue, not simply dense mature scar.
- The tissue protrudes above the adjacent skin level.
- The traditional clinical term is proud flesh.



Schematic

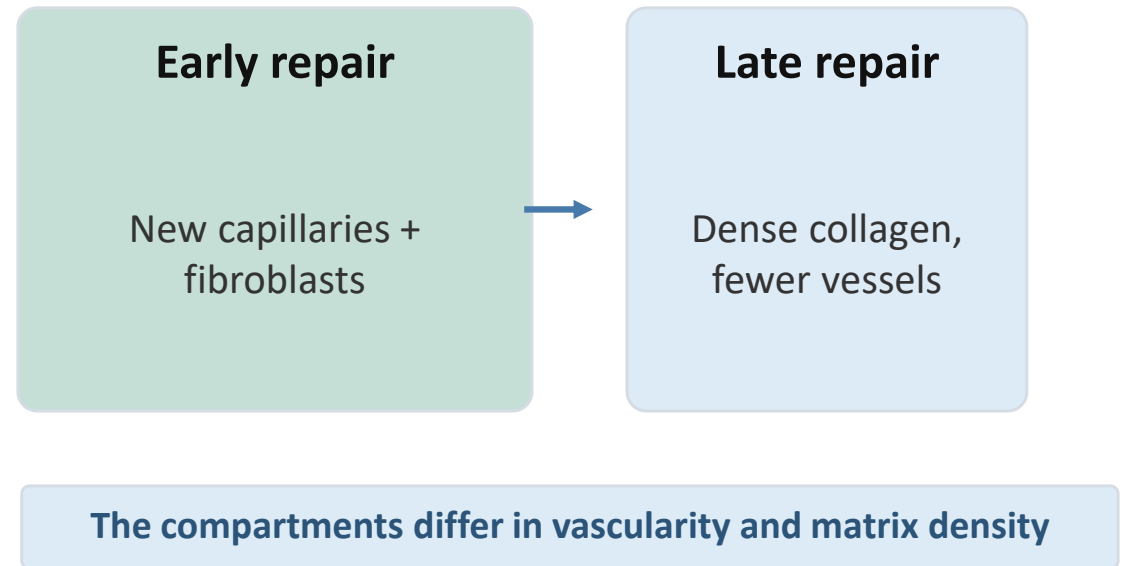
Proud flesh blocks epithelial resurfacing

- Elevated granulation tissue obstructs re-epithelialization.
- Epithelial cells cannot restore an uninterrupted surface across the raised defect.
- Thus excessive repair tissue can paradoxically delay closure.



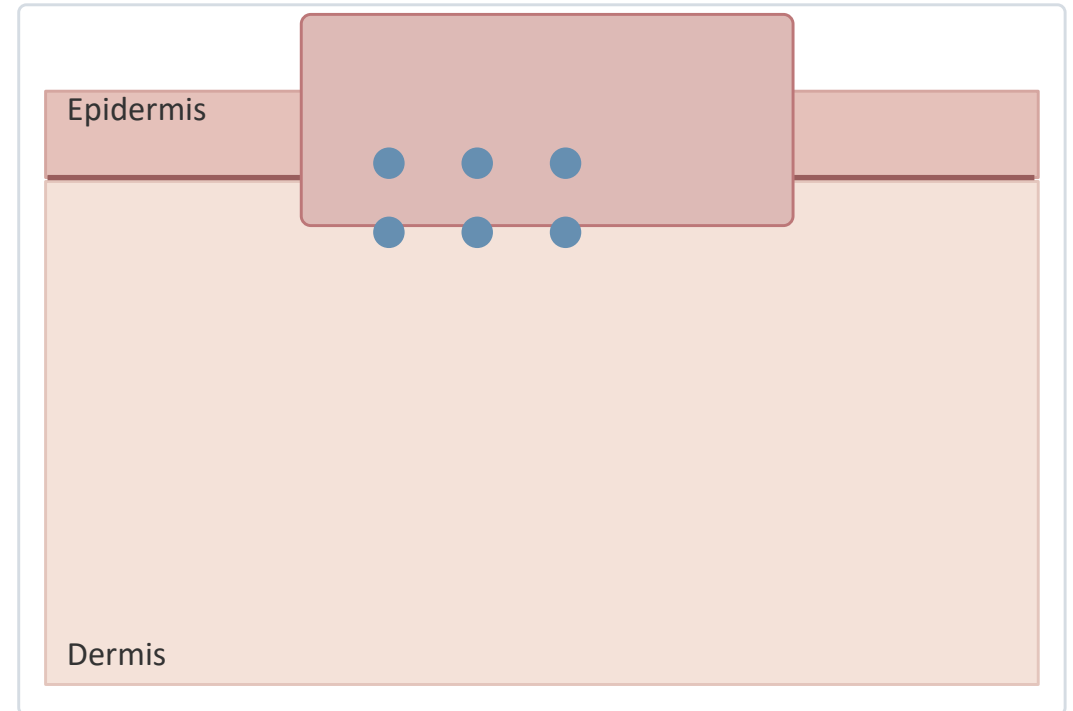
Distinguish granulation tissue from mature scar

- Granulation tissue is capillary-rich and relatively loose.
- A mature scar is denser and substantially less vascular.
- Proud flesh is primarily overproduction of the former compartment.



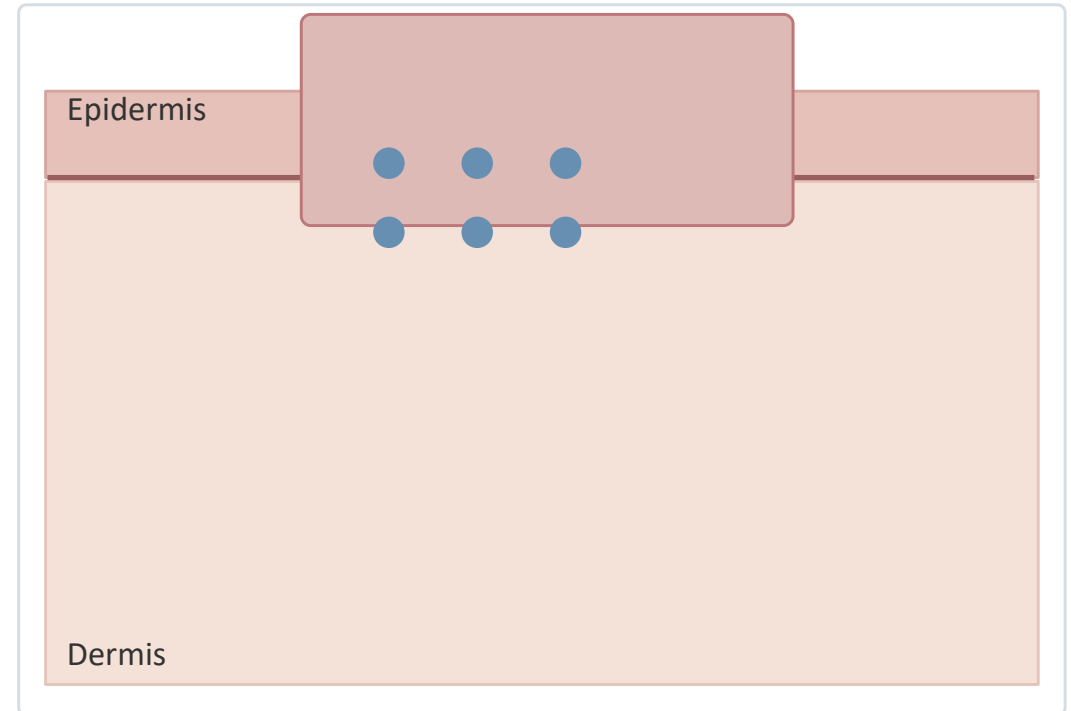
Proud flesh: Intervention targets the obstruction

- Cautery or surgical excision of excess granulation tissue.
- The intended result is removal of the tissue blocking epithelial continuity.
- These approaches are source-described principles, not a comprehensive treatment guideline.



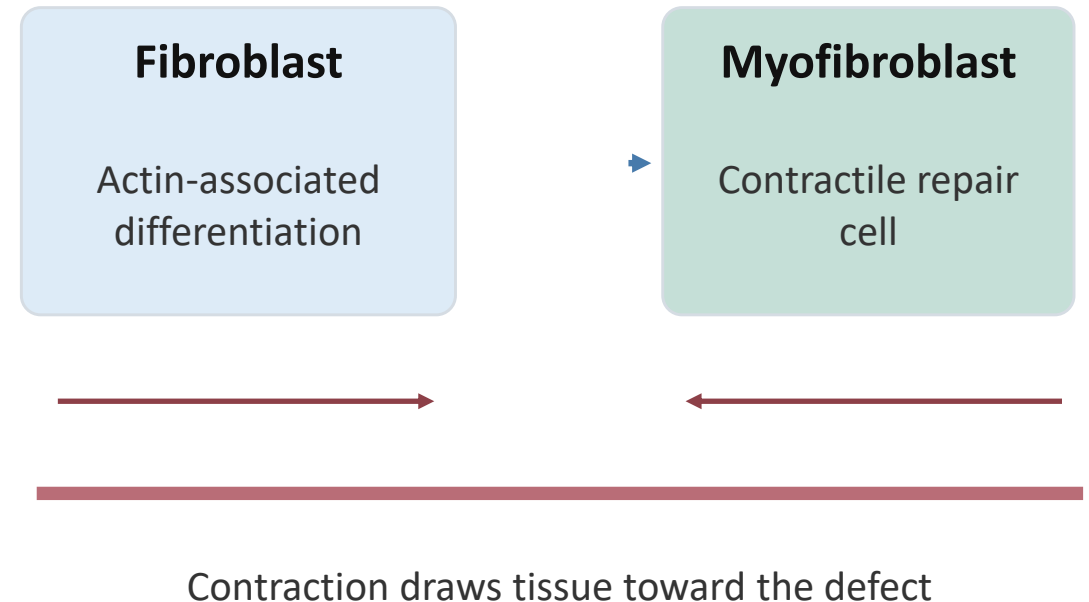
Epithelial repair and connective-tissue repair must align

- Surface closure requires epithelial continuity.
- Underlying granulation tissue supports regeneration when properly organized.
- Excess granulation illustrates how one overactive repair component disrupts another.



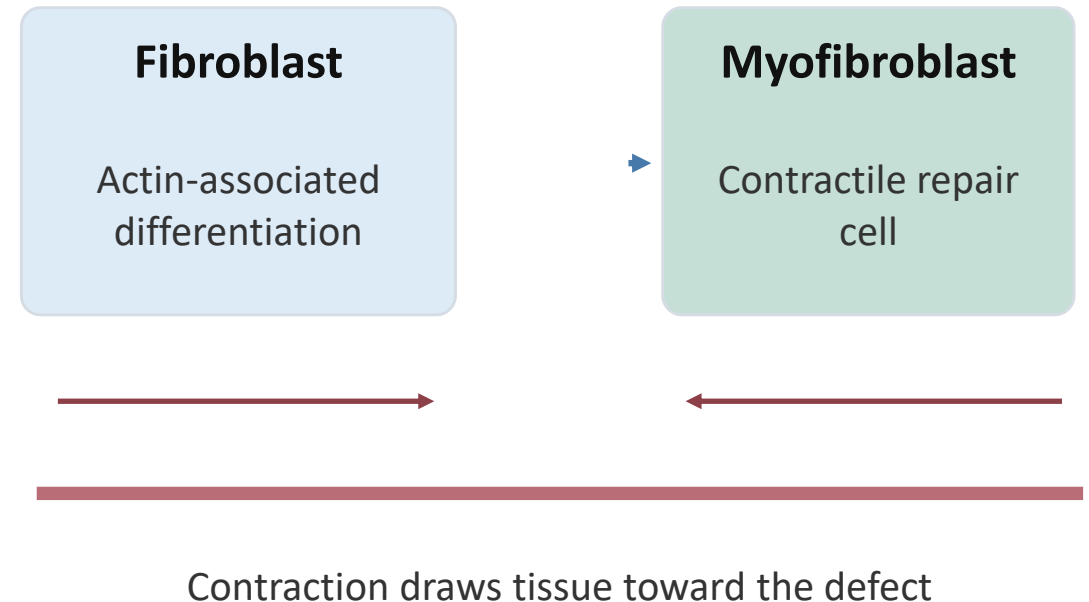
Wound contraction is normally useful

- Reducing wound dimensions helps close tissue deficits.
- Secondary-intention wounds show more contraction than small incisions.
- Normal contraction must be distinguished from deforming contracture.



Contracture is excessive contraction

- An exaggerated decrease in wound dimensions distorts adjacent tissue.
- The deformity can persist after the surface is closed.
- Contracture is a functional and geometric complication of repair.



Certain sites are especially susceptible

- Palms are cited as contracture-prone sites.
- Soles are also vulnerable.
- The anterior thorax is another recognized location.

Palms

Grip / flexion
surfaces

Soles

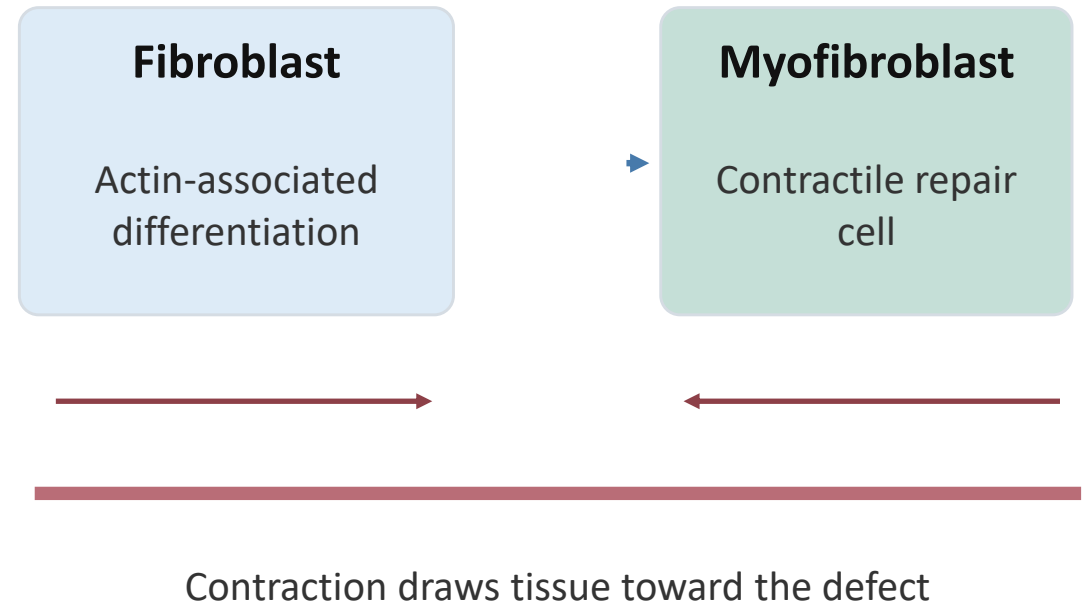
Weight-bearing
surface

Anterior thorax

Anterior chest
region

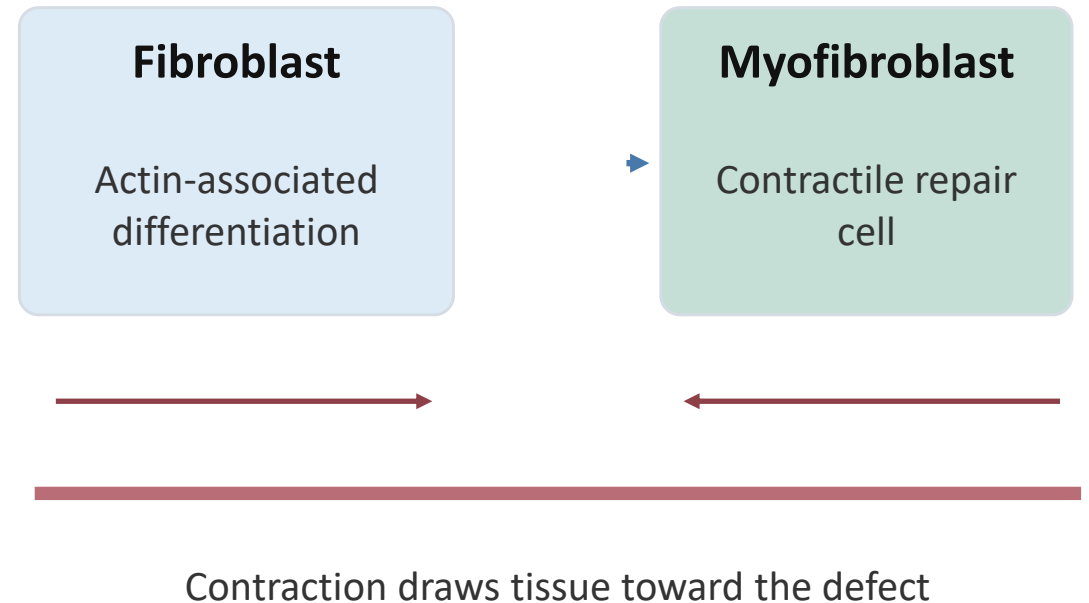
Severe burns can lead to disabling contractures

- Burn injury may extend through deep dermal structures.
- Healing with marked contraction can alter joint movement.
- The important outcome is impaired function, not just scar appearance.



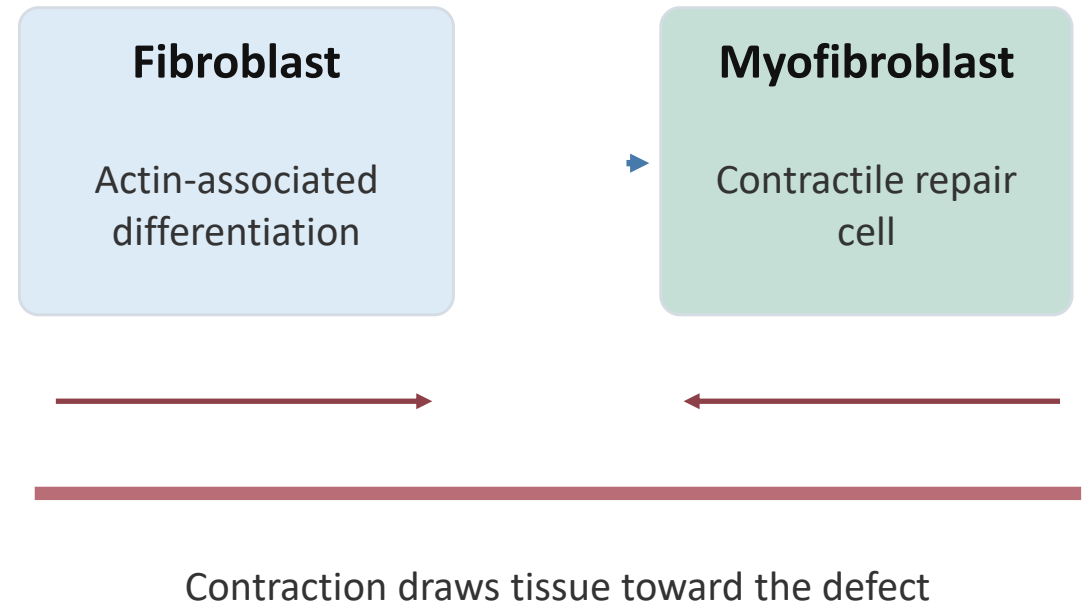
Contracture distorts the surrounding tissue

- Contraction exerts forces on tissue bordering the wound.
- In a mobile region, deformation may restrict a normal range of motion.
- The schematic displays a mechanical concept, not patient anatomy.



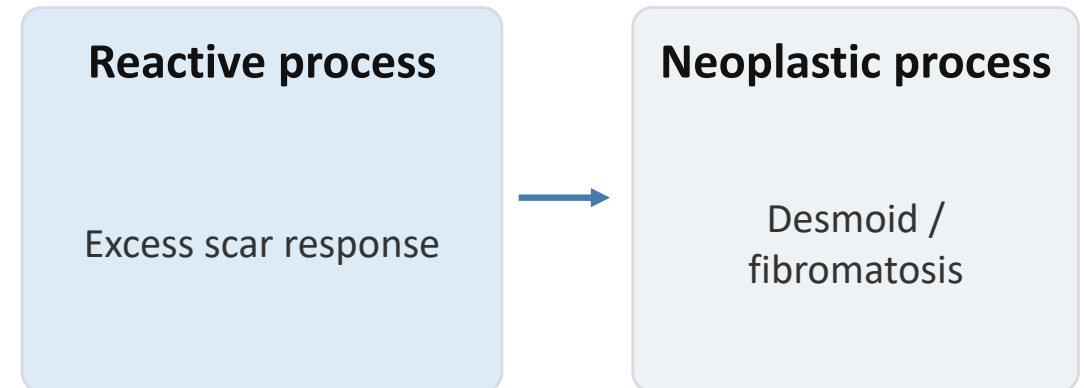
Hypertrophic scar and contracture may coexist

- Myofibroblasts contribute to normal scar contraction.
- Hypertrophic scars often contain many myofibroblasts.
- A contracture is classified by deformation and functional effect, not simply by scar height.



Rare fibroblastic proliferation can follow injury

- Some incisional scars or traumatic wounds are followed by exuberant connective-tissue-cell proliferation.
- These proliferations may recur after excision.
- This discussion leads to desmoid-type aggressive fibromatosis.



Desmoid is not simply another keloid

- Aggressive fibromatosis (desmoid) is classified as a benign connective-tissue neoplasm.
- Its nature differs from a collagen-rich reactive scar.
- The source refers detailed tumor classification to a separate chapter.

Scar	Desmoid
Reactive scar	Benign connective-tissue neoplasm
Excess repair product	Fibroblastic proliferation
Wound-healing deviation	Desmoid / aggressive fibromatosis

An integrated map of abnormal wound repair

- Excess collagen produces raised fibrous scars.
- Excess granulation tissue interrupts epithelial closure.
- Excess contraction deforms tissues; Fibroblastic neoplasia is a separate consideration.

**Excess
collagen**

Raised scars

**Excess
granulation**

Proud flesh

**Excess
contraction**

Contracture

Apply the boundary–trajectory distinction

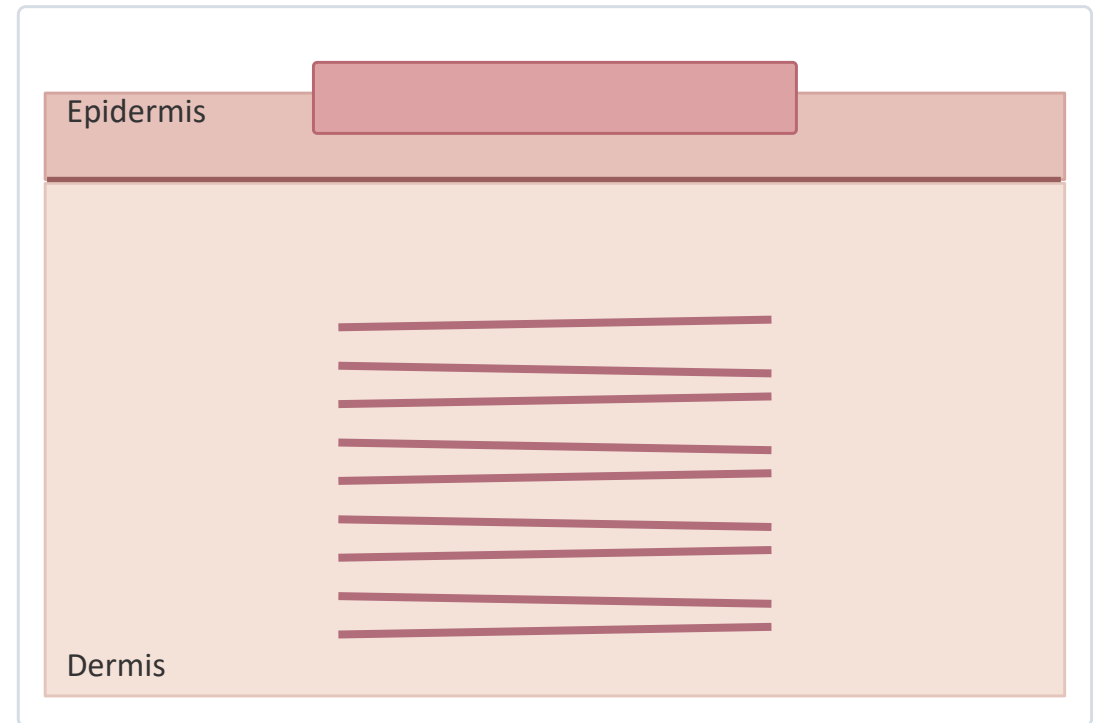
- Ask whether the lesion extends beyond the former wound.
- Ask whether it regresses over subsequent months.
- Do not use skin color alone to decide between hypertrophic scar and keloid.



Dashed idea: Initial wound region vs subsequent scar outline

Case: Raised scar after deep dermal injury

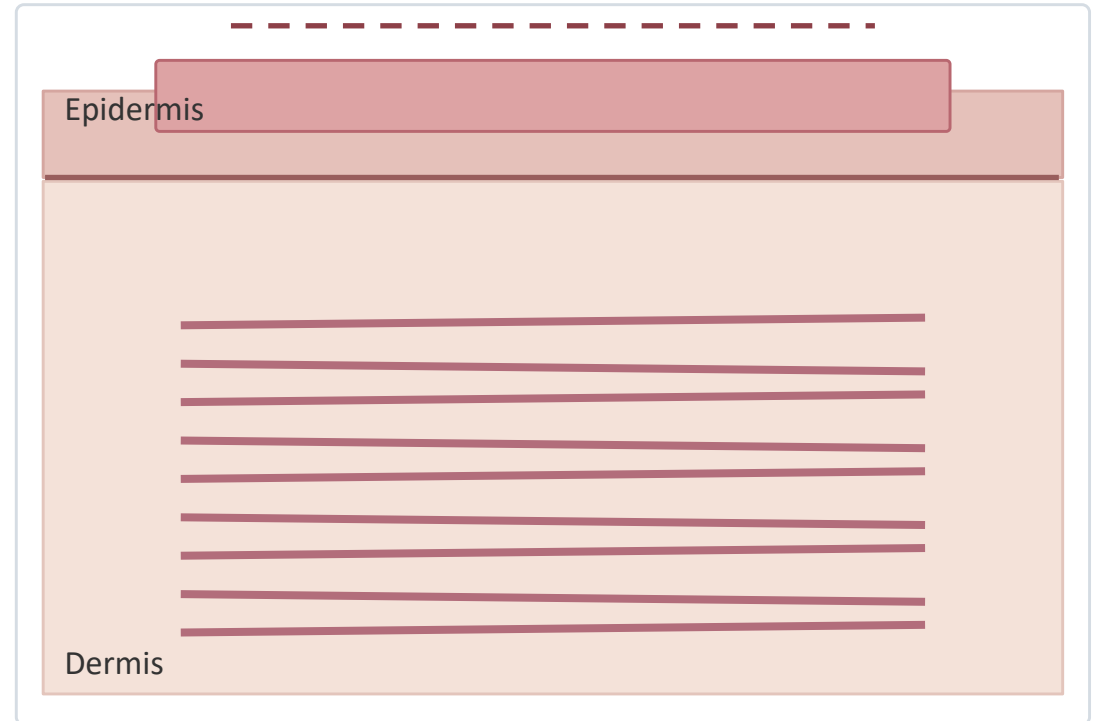
- A hypothetical thermal injury heals with an elevated collagen-rich scar.
- The lesion stays within the injured region and later becomes less prominent.
- Interpretation: This trajectory is most consistent with a hypertrophic scar.



Schematic

Case: Persistent scar growth past a wound

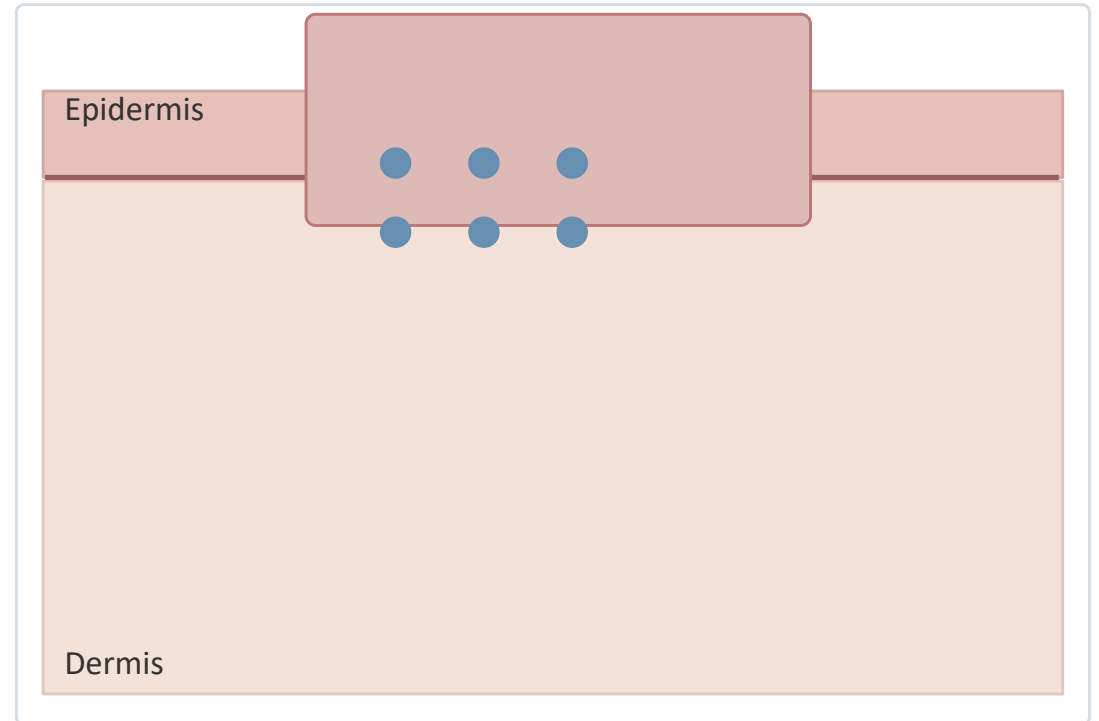
- A hypothetical healed incision develops a raised scar beyond the original edges.
- The lesion remains prominent rather than regressing.
- Interpretation: The defining behavior supports a keloid.



Schematic

Case: Healing surface blocked by raised tissue

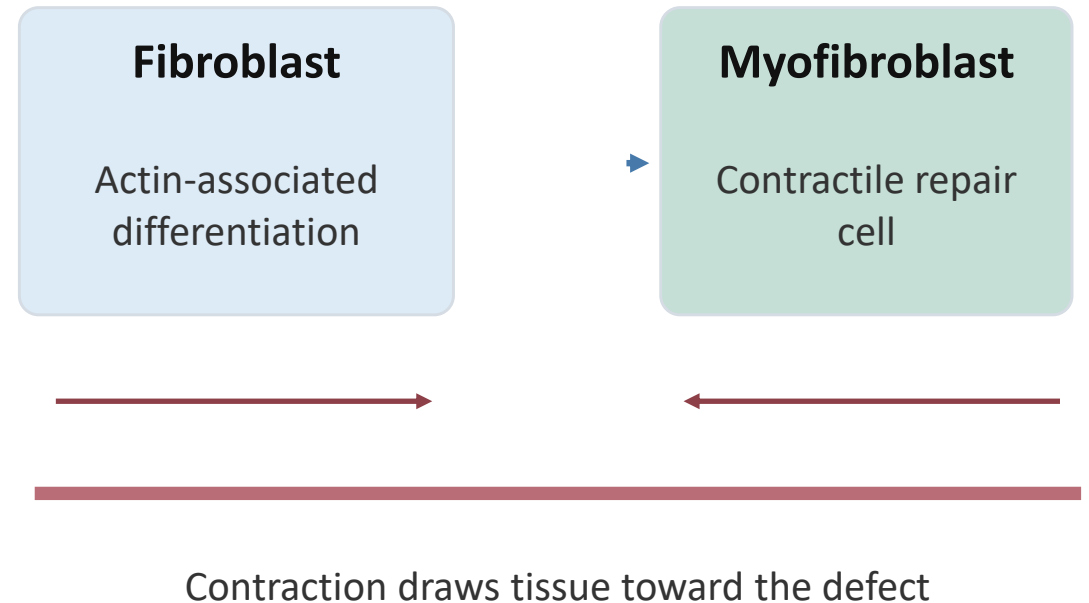
- A hypothetical wound develops a protruding mass of granulation tissue.
- Surface epithelial continuity remains incomplete across that prominence.
- Interpretation: Exuberant granulation tissue (proud flesh).



Schematic

Case: Impaired movement after major burn

- A hypothetical severe burn is followed by tightening across a joint.
- The healed area deforms surrounding tissues and limits movement.
- Interpretation: Deforming wound contracture.



Four abnormalities, four dominant features

- Hypertrophic scar: Raised lesion and usual regression over months.
- Keloid: Beyond wound margins and persistent.
- Proud flesh: Raised granulation blocks epithelium; Contracture: Distortion from excessive contraction.

**Excess
collagen**

Raised scars

**Excess
granulation**

Proud flesh

**Excess
contraction**

Contracture

Questions to check understanding

- Which finding most strongly separates keloids from hypertrophic scars?
- Why can excessive granulation tissue delay complete epithelial closure?
- Which cell type links collagen-rich repair to contraction?

Core concept

Repair is normally regulated in space and time

Core teaching references

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 3: Tissue Repair; Repair by Scarring; Healing of Skin Wounds; Excessive Scarring.
- The figures here are original editable schematics, not reproductions of the textbook images.

Core concept

Repair is normally regulated in space and time