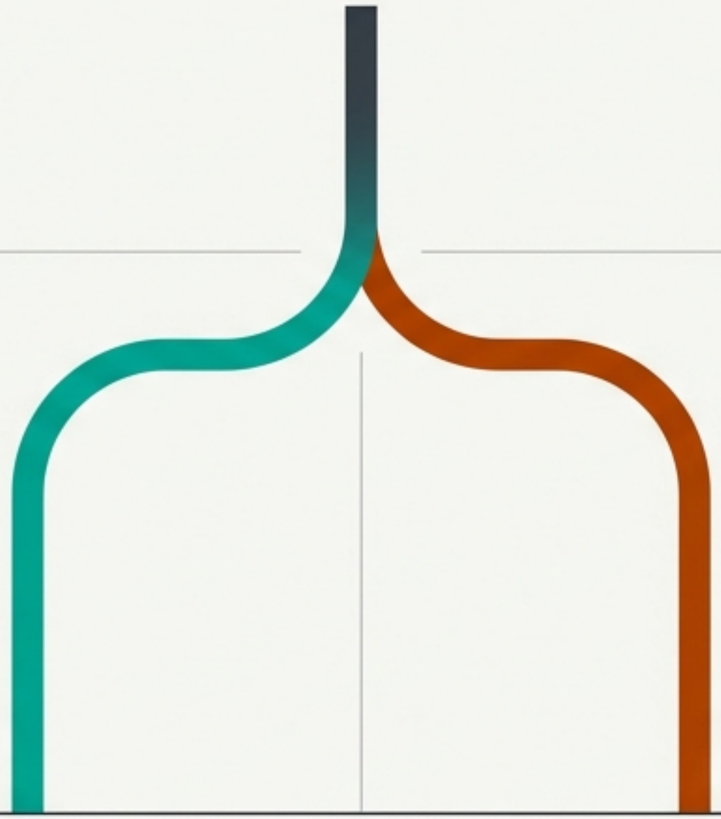
INJURY

THE BIOLOGICAL DECISION MATRIX

Principles of Tissue Regeneration and Wound Repair



PATH A: REGENERATION

Restoration of native architecture.

PATH B: REPAIR

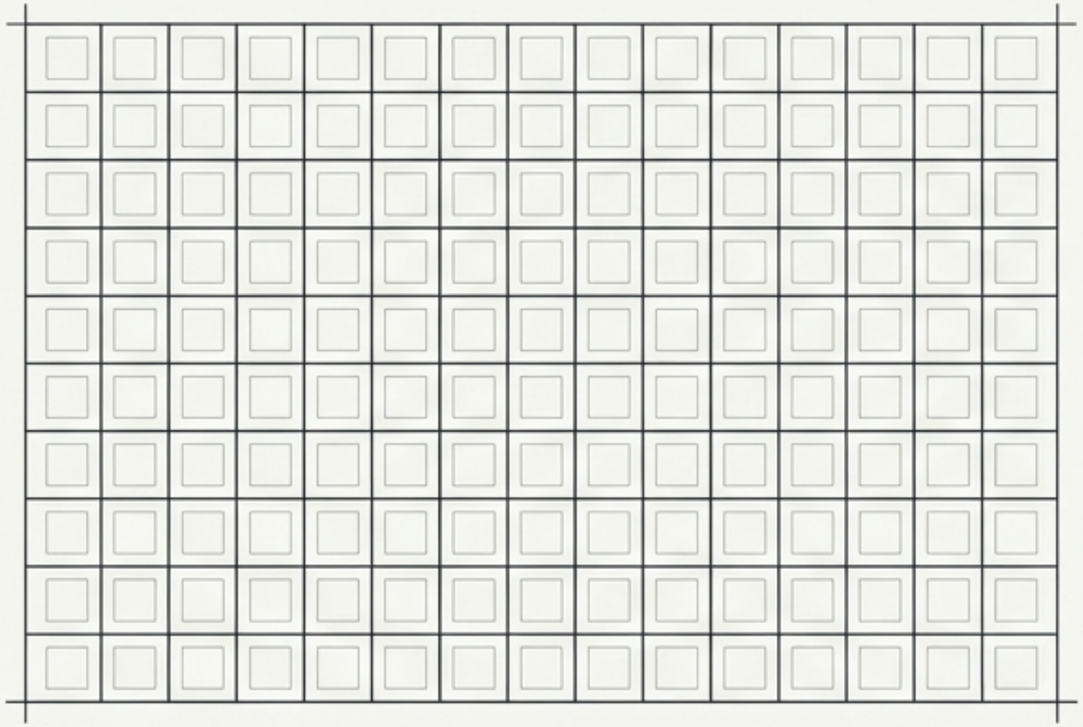
Formation of fibrous scar.

A CLINICAL REVIEW OF HEALING MECHANISMS.

THE TWO OUTCOMES OF INFLAMMATION

TRIGGER: INFLAMMATION

REGENERATION



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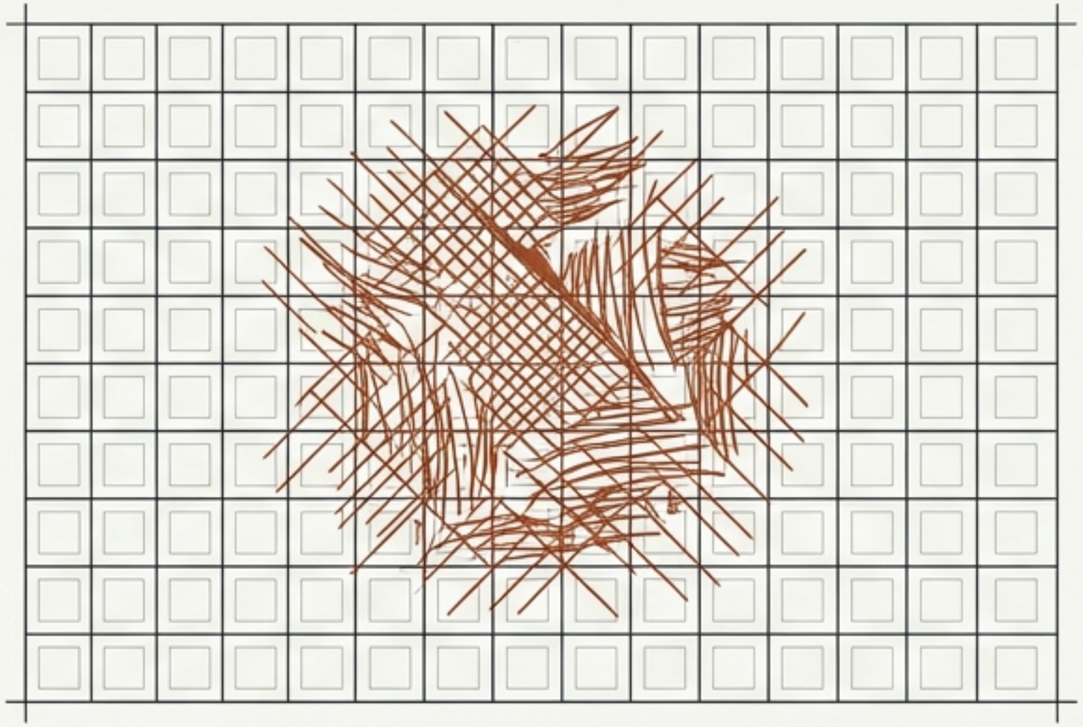
Replacement with native tissue.
Dependent on regenerative capacity.

REPAIR



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Replacement with fibrous scar.
Occurs when stem cells are lost.

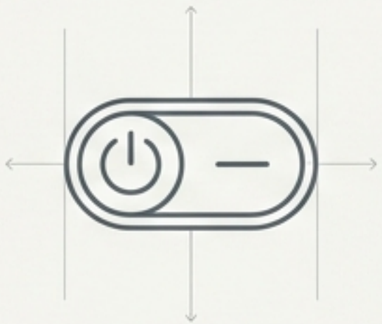
CLASSIFYING REGENERATIVE CAPACITY

LABILE

Stem cells continuously cycle.

Examples:

- Skin (Basal Layer)
- Bowel (Mucosal Crypts)
- Bone Marrow



STABLE

Quiescent (G0 Phase). Re-enter cycle only if stimulated.

Examples:

- Liver (Compensatory Hyperplasia)
- Kidney Tubules

PERMANENT

Zero regenerative potential. Defaults to scar.

Examples:

- Myocardium
- Skeletal Muscle
- Neurons

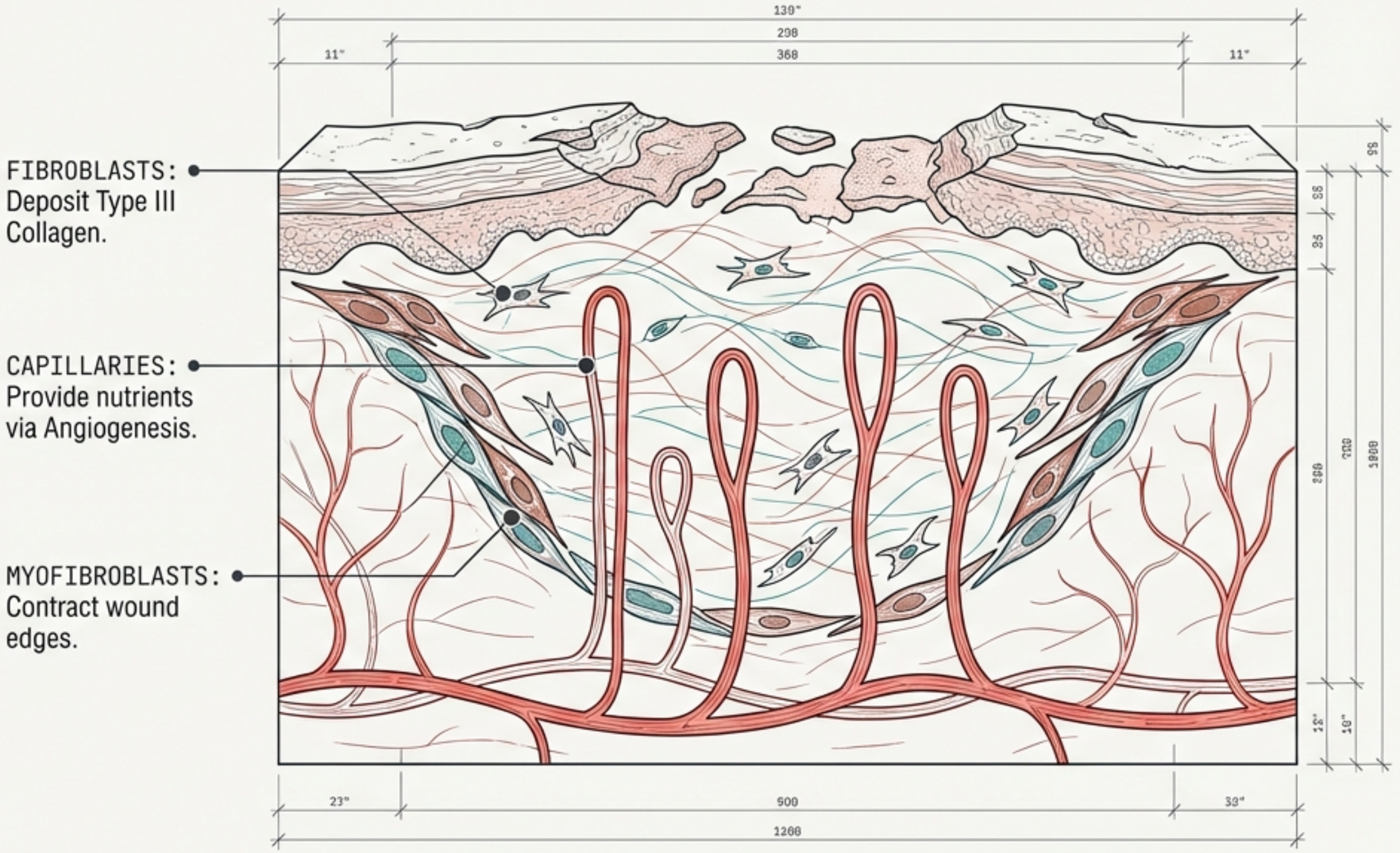
CLINICAL CORRELATION : Damage to Permanent Tissue (e.g., Myocardial Infarction) inevitably leads to Repair (Scarring).

THE ANATOMY OF GRANULATION TISSUE

Definition:
The temporary scaffolding of repair.

Characteristics:
Pink, soft, granular appearance.

Composition:
Type III Collagen (Pliable).



REMODELING: THE COLLAGEN SWITCH

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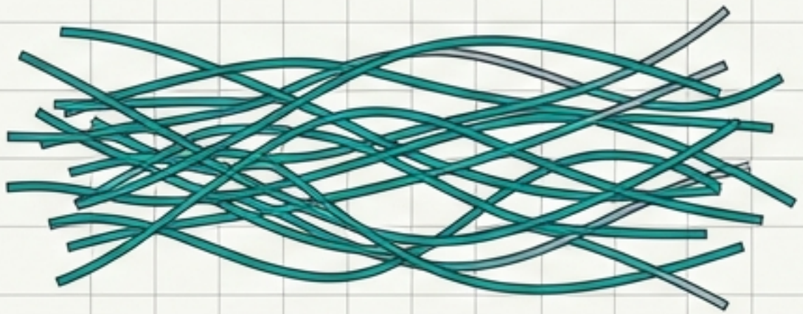
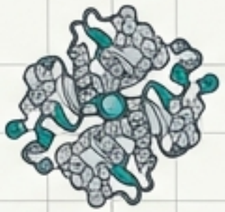
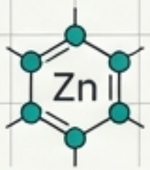
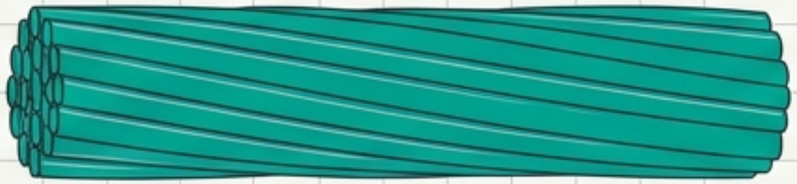




COLLAGENASE



ZINC COFACTOR



TYPE III COLLAGEN

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Pliable / Granulation Tissue / Keloids

TYPE I COLLAGEN

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High Tensile Strength / Skin / Bone / Tendons

Maturation requires the replacement of temporary Type III collagen with permanent Type I collagen. This enzymatic process is Zinc-dependent.

MOLECULAR MEDIATORS OF HEALING

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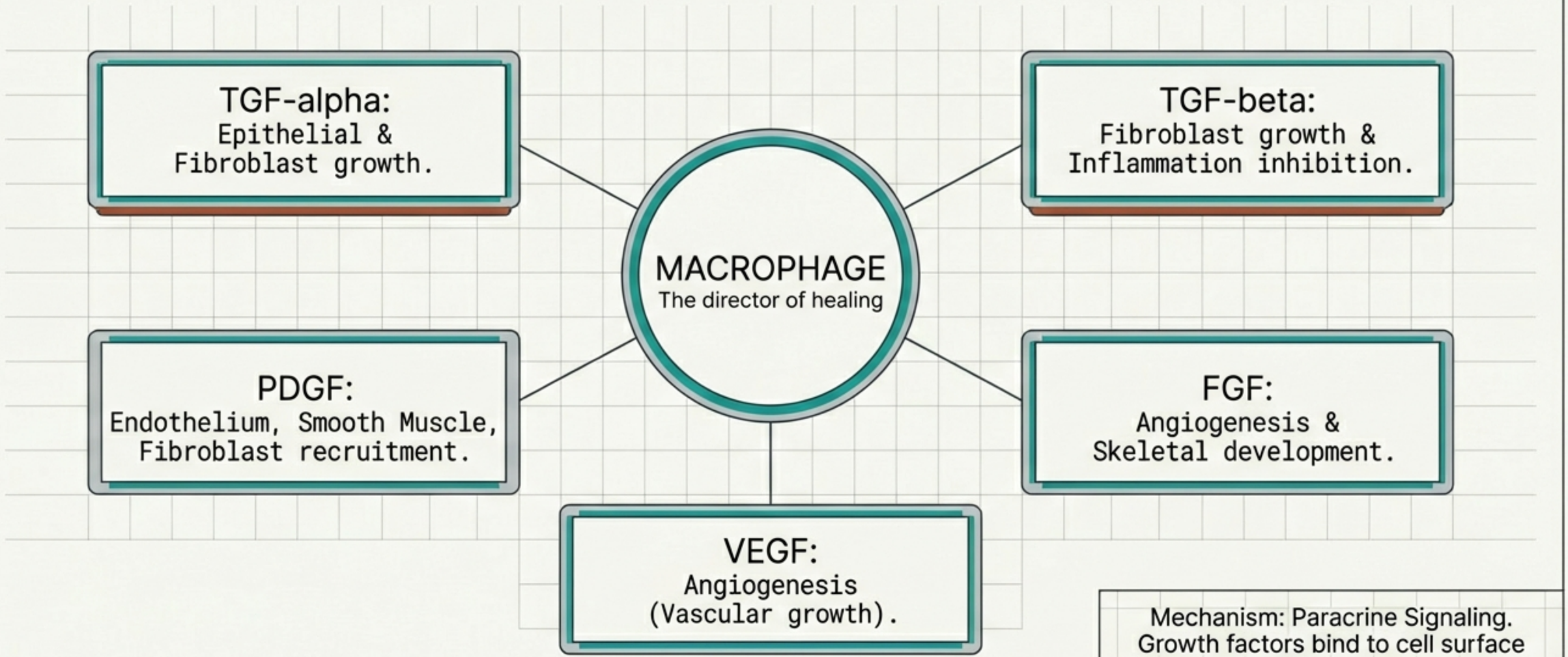

Mechanism: Paracrine Signaling. Growth factors bind to cell surface receptors to trigger gene expression.

CLINICAL PATHWAYS: PRIMARY VS. SECONDARY INTENTION

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PRIMARY INTENTION

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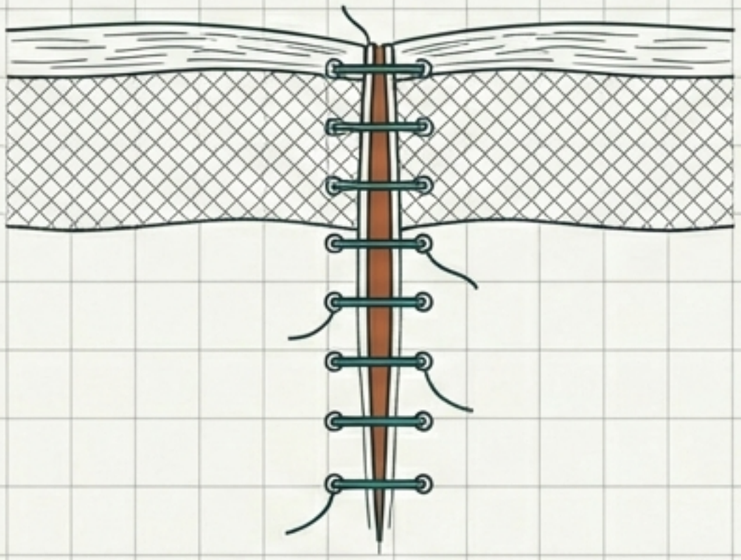

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Edges approximated (Sutured).
Minimal scar.

SECONDARY INTENTION

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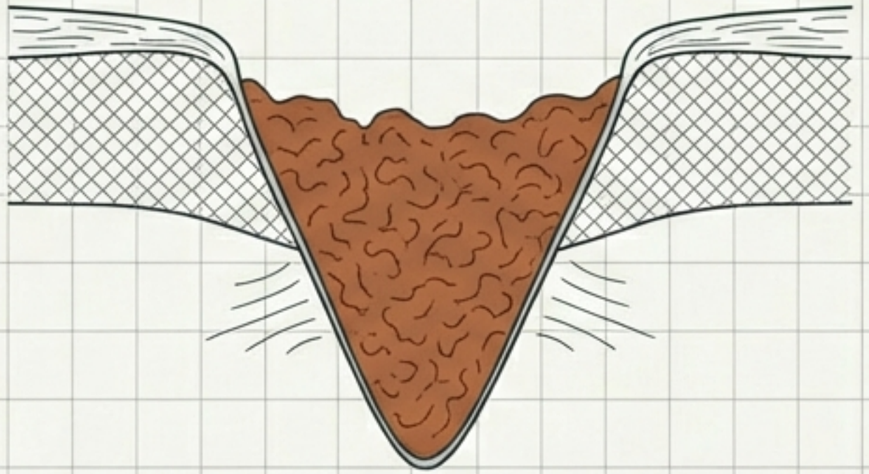

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Edges not approximated.
Granulation fills defect.
Myofibroblast contraction required.

THE BIOCHEMISTRY OF DELAYED HEALING

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VITAMIN C

Proline + Lysine + VITAMIN C → Hydroxylated Residues

Mechanism: Hydroxylation is required for collagen cross-linking.

COPPER

Lysyl Oxidase + COPPER → Stable Cross-links

Mechanism: Stabilizes lysine and hydroxylysine bonds.

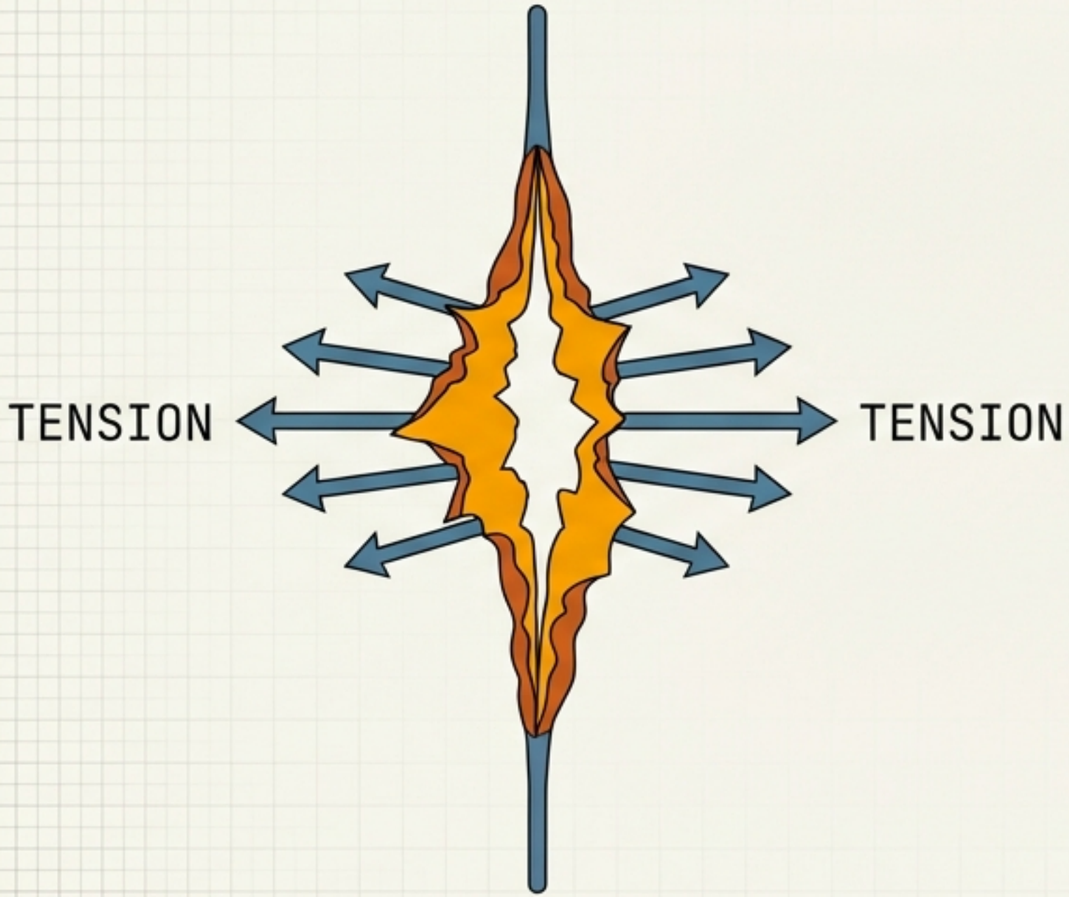
ZINC

Collagenase + ZINC → Type I Replacement

Mechanism: Required to replace weak Type III collagen with strong Type I.

Most common cause of delayed healing: Infection (*S. aureus*).

MECHANICAL FAILURE: DEHISCENCE



RUPTURE OF A WOUND

Occurs when tension exceeds the tensile strength of the healing tissue.

Clinical Setting: Most common after abdominal surgery.

Contributing Factors: Coughing, vomiting, or infection increase mechanical stress.

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ABERRANT HEALING: HYPERTROPHIC VS. KELOID

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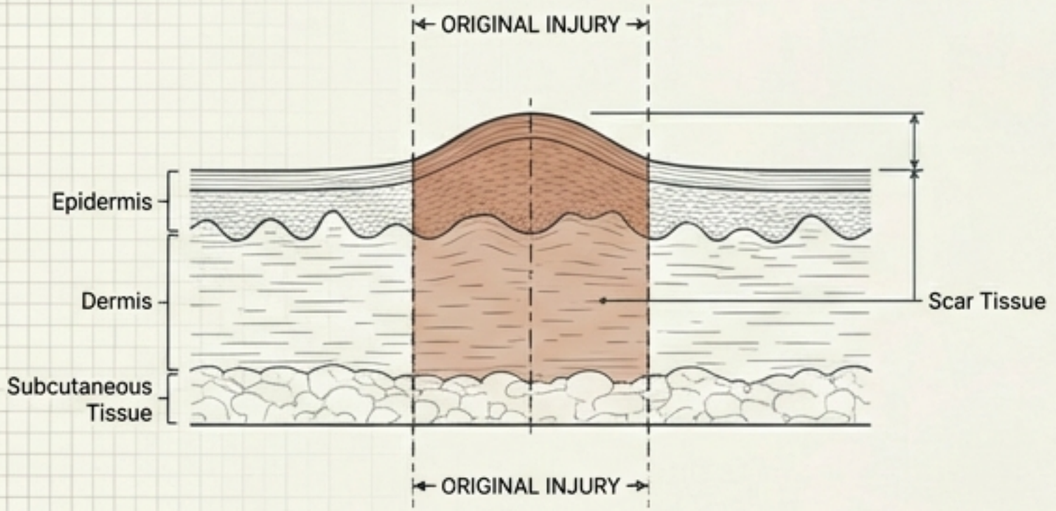
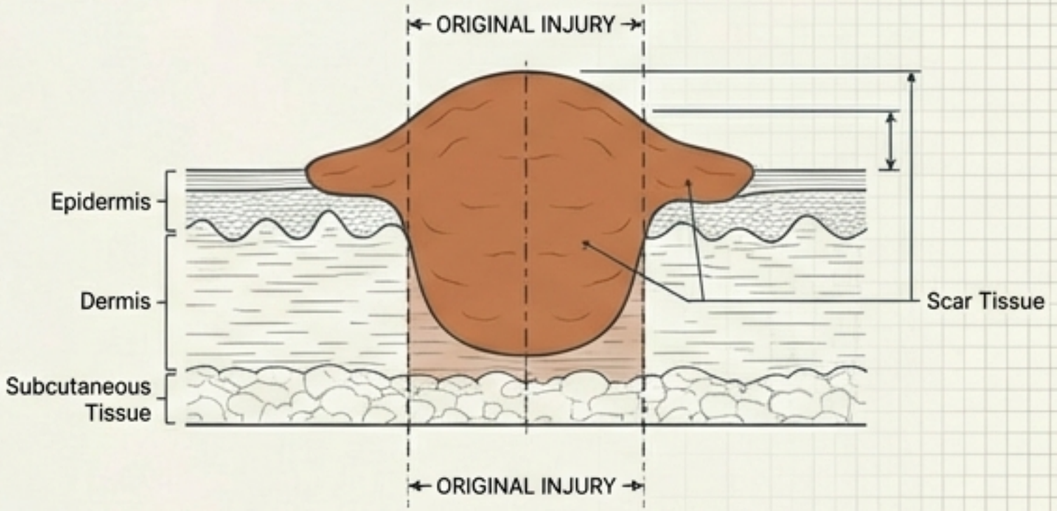
HYPERTROPHIC SCAR



- **Scope:** Localized to wound.
- **Collagen:** Type I (Primary).

KELOID



- **Scope:** Out of proportion to wound.
- **Collagen:** Excess Type III.
- **Demographics:** Genetic predisposition (African American).
- **Sites:** Earlobes, face, upper extremities.

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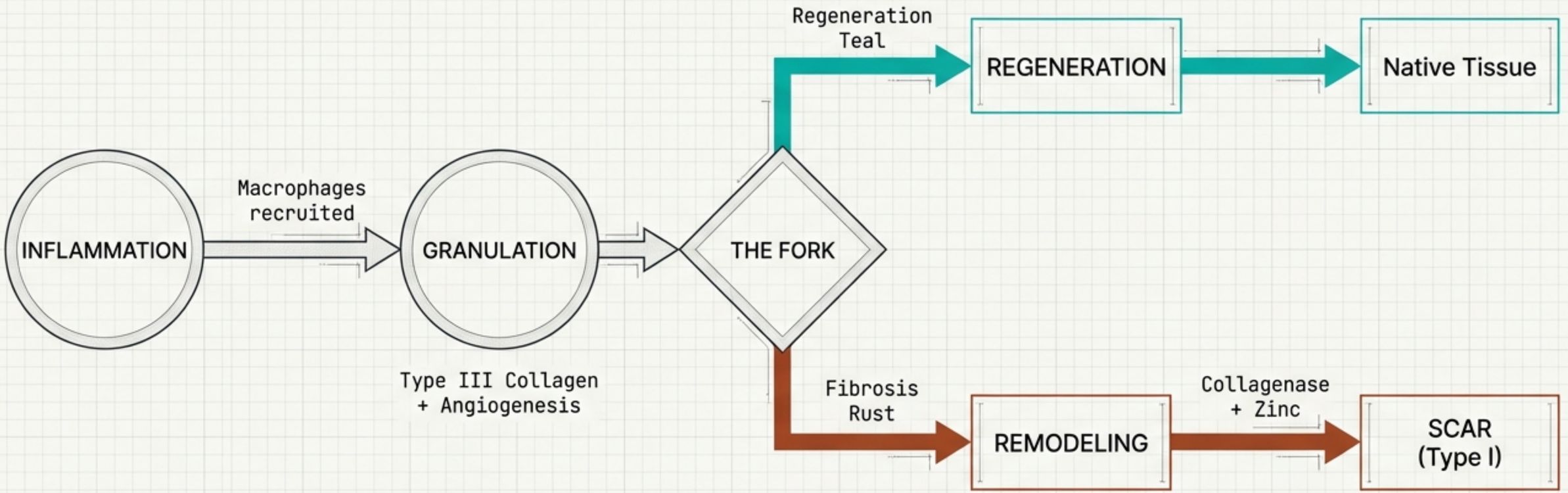
SUMMARY OF THE HEALING LIFECYCLE



Outcome is determined by: 1. Tissue Capacity (Labile vs. Permanent) and 2. Molecular Building Blocks (Vit C, Cu, Zn)