

Wound healing I

General Principles of Surgery

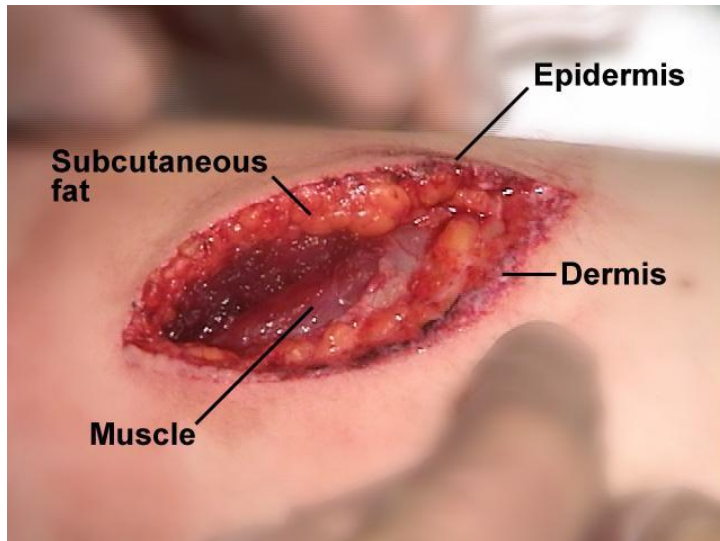
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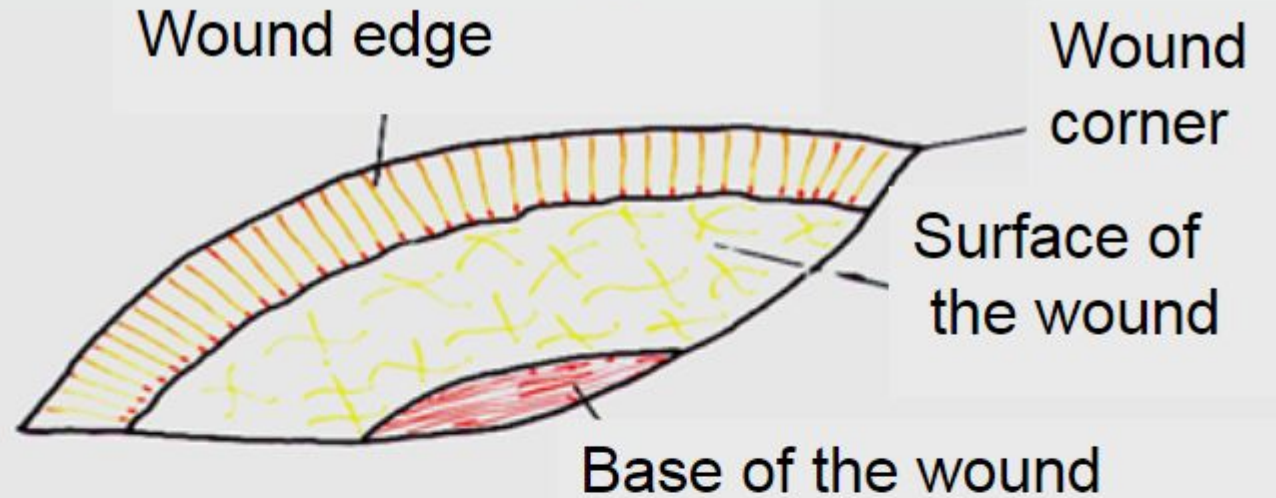
<https://sites.google.com/newvision.ge/bkobalava>



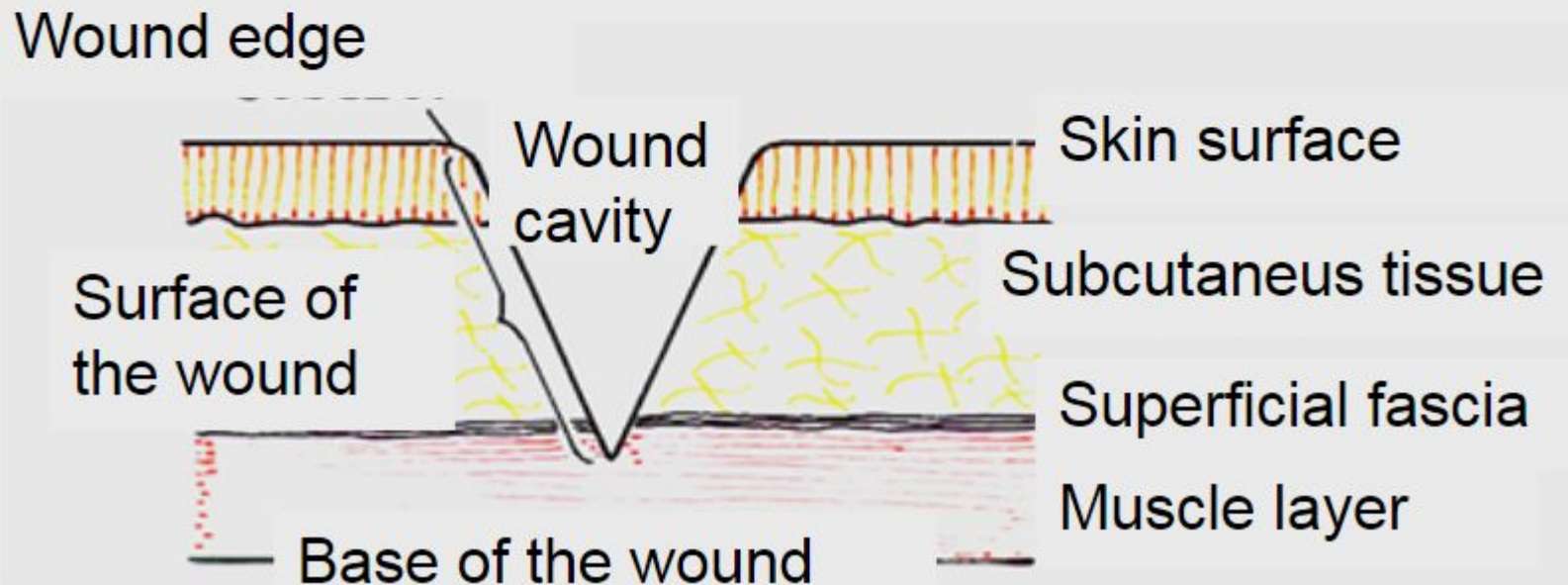
- A break in the normal continuity of a tissue.
- A wound occurs when the integrity of any tissue is compromised (e.g. skin breaks, muscle tears, burns, or bone fractures).



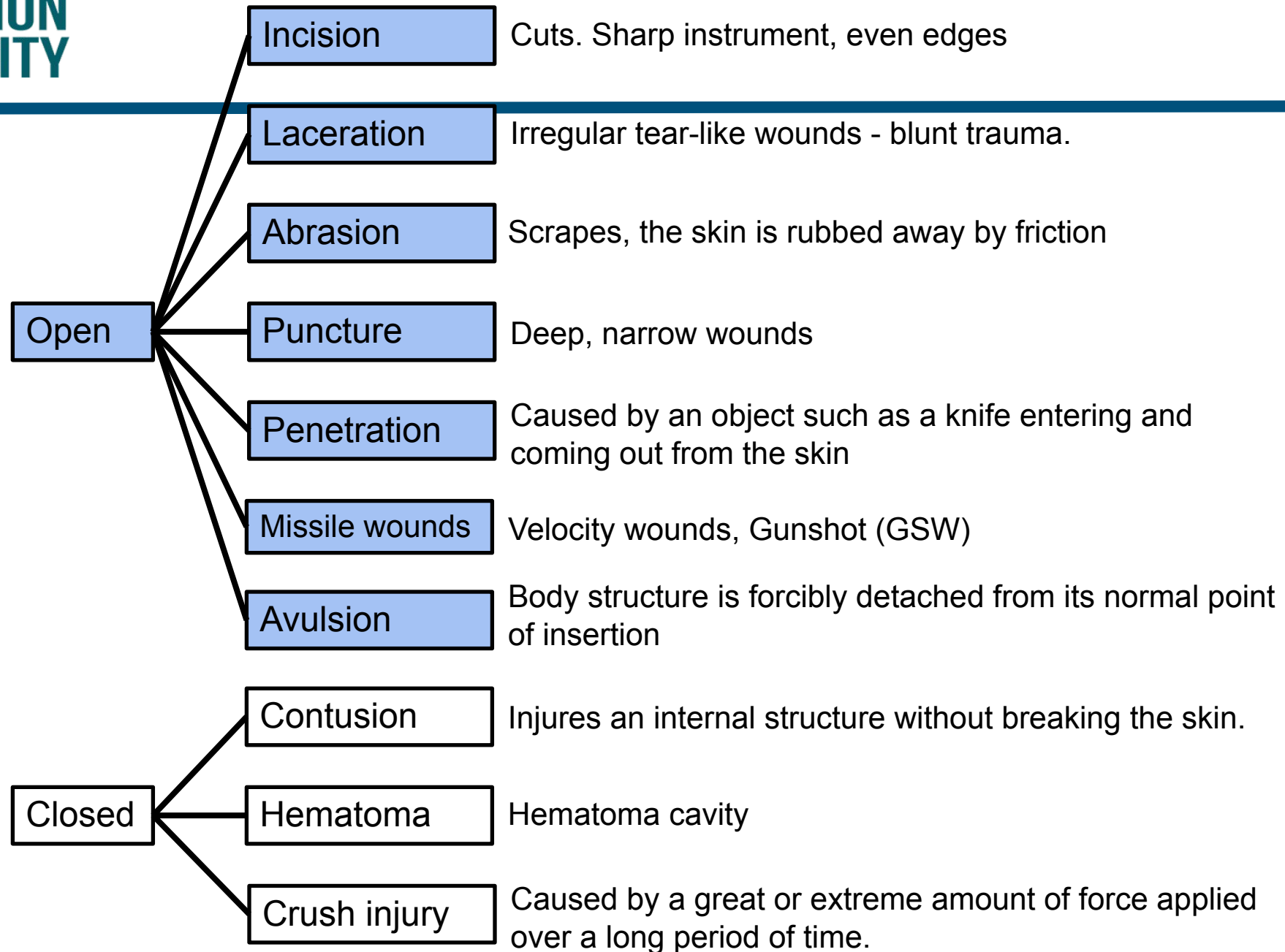
WOUND DEFINITION



Cross section of a simple wound



- MECHANICAL (See in next slide)
- CHEMICAL
 - Acid
 - Base
- Caused by RADIATION
- THERMAL
 - Burning
 - Freezing
- BITE



WOUND CLASSIFICATION

I. Clean:

- - Uninfected, no inflammation
- - Visceral organs not entered
- - Closed primarily
- - Infection rate <3%

II. Clean-contaminated:

- - Resp, Upper GI, biliary GU tracts entered, controlled
- - No unusual contamination
- - Infection rate <10%

III: Contaminated:

- - Open, fresh, accidental wounds
- - Major break in sterile technique
- - Gross Spillage from GI tract
- - Acute non purulent inflammation
- Examples: Inflamed appy, bile spillage in chole, diverticulitis, Rectal surgery, penetrating wounds
- - Infection rate >30%

IV: Dirty:

- - Old traumatic wounds, devitalized tissue
- - Existing infection or perforation
- - Organisms present BEFORE procedure
- Examples: Abscess I&D, perforated bowel, peritonitis, wound debridement, positive cultures pre-op

Factors affecting wound healing

LOCAL	GENERAL
Foreign body, infection, ischemia, arterial or venous insufficiency, neoplasia, radiotherapy, neuropathy, iatrogenic stress e.g. rough tissue handling.	Systemic factors: nutritional deficiency, diabetes, ageing, liver disease, steroids, immunosuppressants, neoplasia, chemotherapy. Congenital conditions: Ehlers Danlos, epidermolysis bulosa, cutis laxa, etc. fastbleep))

Acute and chronic wounds

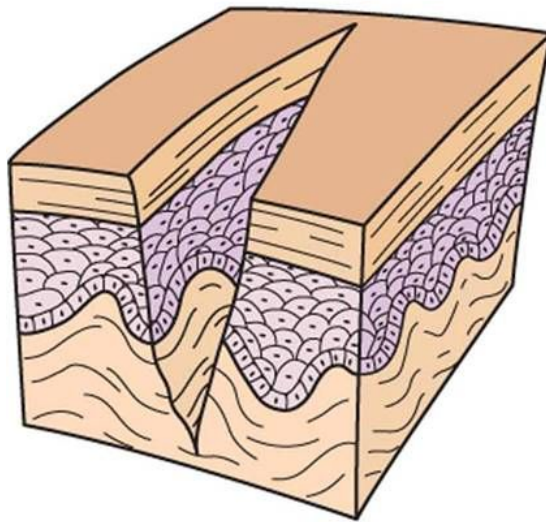
	Acute Wound	Chronic Wound
Definition	Occurred in last 4-6 weeks.	Present for longer than 6 weeks. Caused by endogenous mechanisms related to a predisposing condition or risk factors (diabetes, obesity, smoking, AIDS, chemotherapy) which eventually compromises dermal and epidermal tissue structures.
Examples	Surgical wounds, bites, burns, abrasions, traumatic wounds.	Leg/foot ulcers and pressure sores – likely from vascular insufficiency or neuropathy.

MAJOR TYPES OF CHRONIC WOUNDS

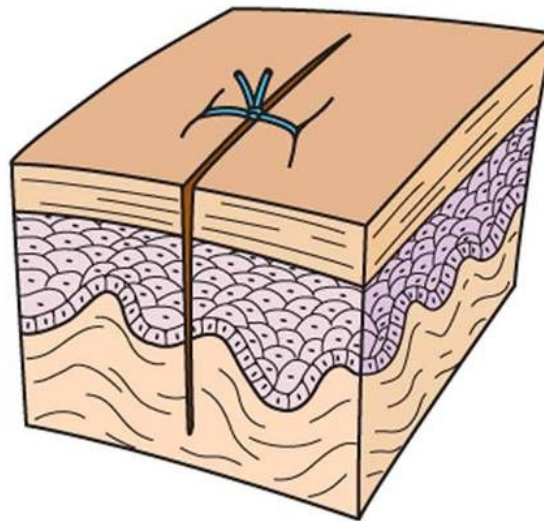
Vascular	Venous ulcers	Venous insufficiency, thrombosis, varicosis
	Arterial ulcers	Macroangiopathy, atherosclerosis, arterial insufficiency
Diabetic ulcers		Neuropathy, microangiopathy, hyperglycemia
Pressure ulcers		Immobility, excessive pressure

Primary intention

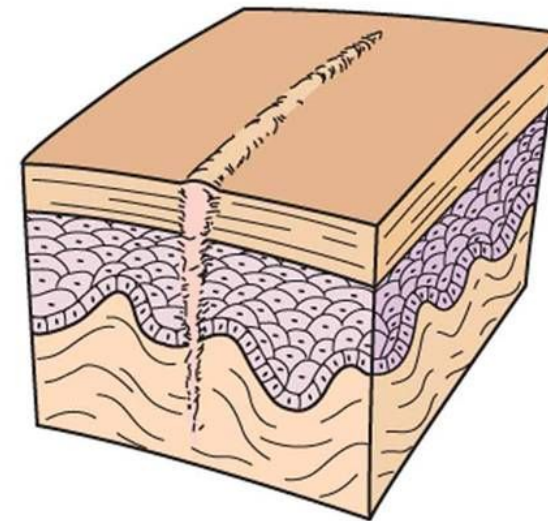
- Occurs when:
 - The edges are clean and held together with ligatures
 - There is little gap to bridge Healing
- Healing properties (When uncomplicated)
 - Occurs quickly
 - Rapid ingrowth of wound healing cells (macrophages, fibroblasts, etc.)
 - Restoration of the gap by a small amount of scar tissue.
- soundly united within 2 weeks
- Dense scar tissue is laid down within 1 month



Clean incision



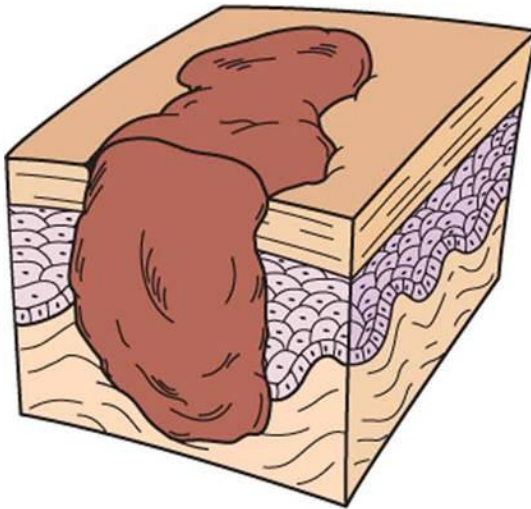
Early suture



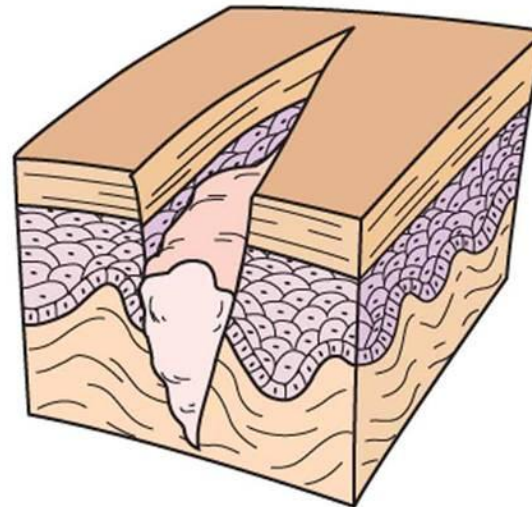
“Hairline” scar

Secondary intention

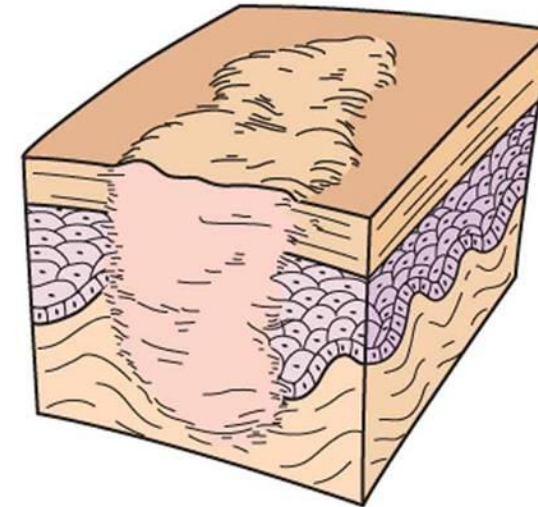
- Occurs when:
 - The edges are separated
 - The gap can not be directly bridged
 - Extensive epithelial loss
 - Severe contamination
 - Significant subepithelial tissue damage
- Healing properties
 - Occurs slowly
 - Granulation; healing from the bottom towards the surface
 - Restoration of the gap by a small amount of scar tissue.
- Scarring
- Wound contracture



Gaping wound
with blood clot



Granulation tissue
fills in wound

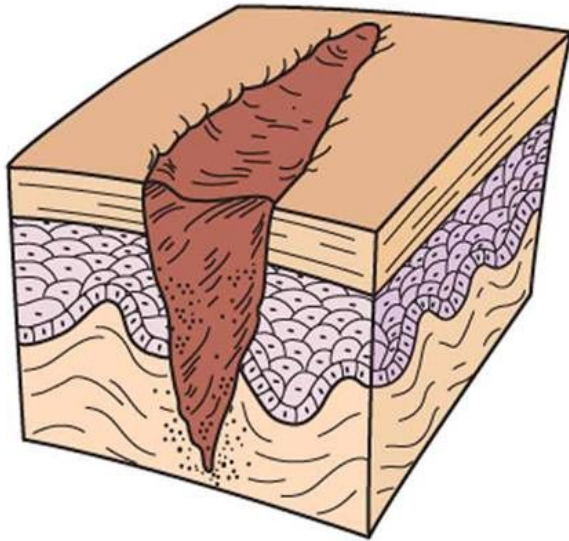


Large scar

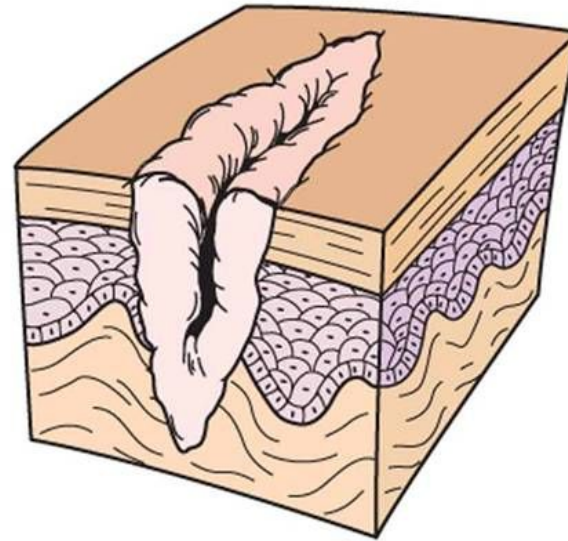
Differences between primary and secondary healing

Feature	Primary healing	Secondary healing
Cleanness	Clean	Unclean
Infection	Generally uninfected	May be infected
Margins	Surgically clean	Irregular
Healing	Scanty granulation tissue	Granulation tissue fill the gap
Healing period	Short	long
Healing direction	Direct healing	From the bottom to the edge
Outcome	Neat linear scar	Contracted irregular wound

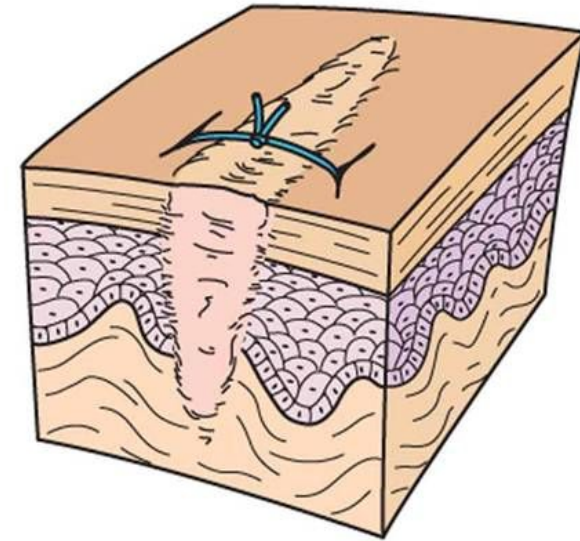
Tertiary intention



Contaminated wound

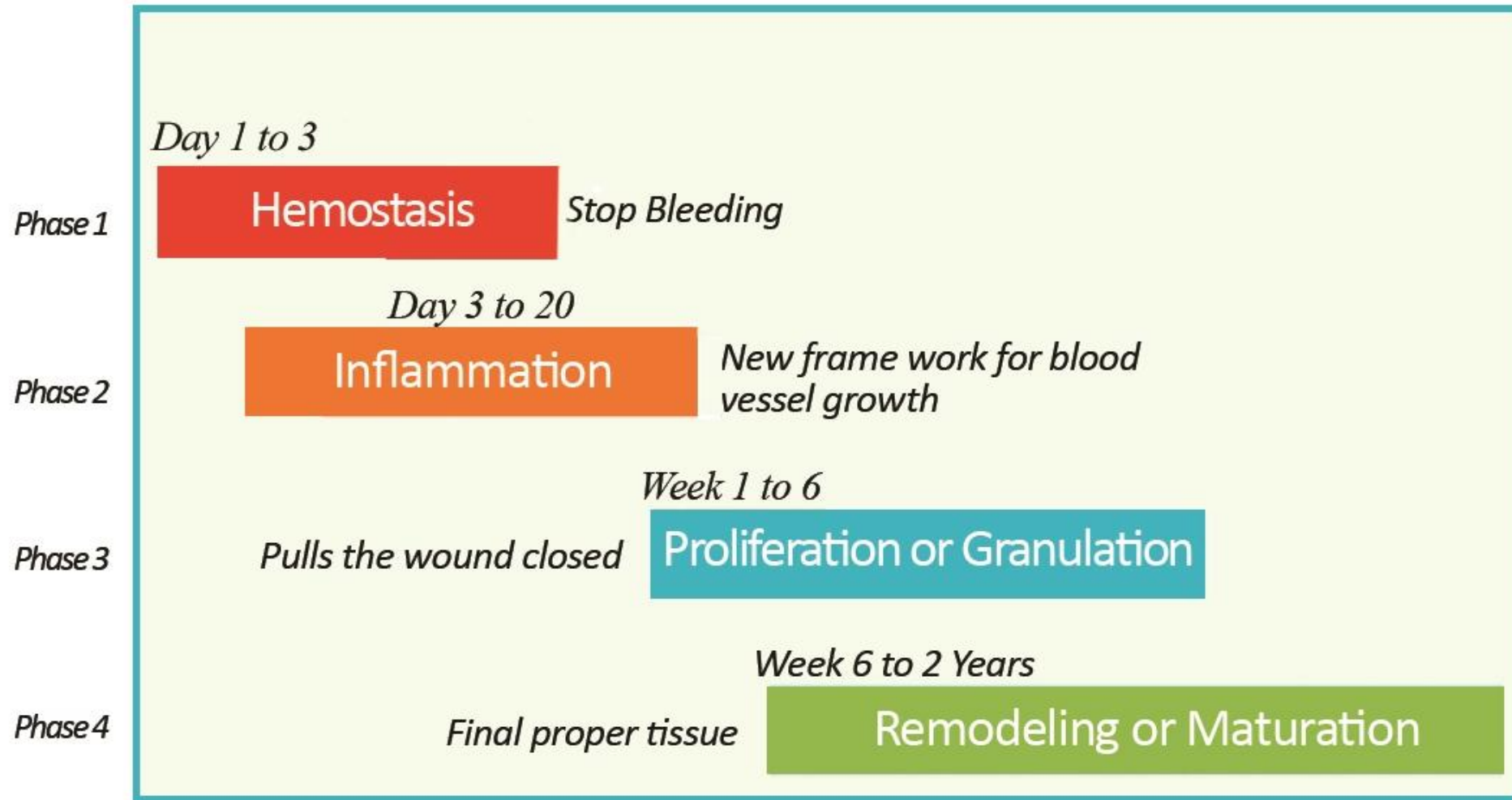


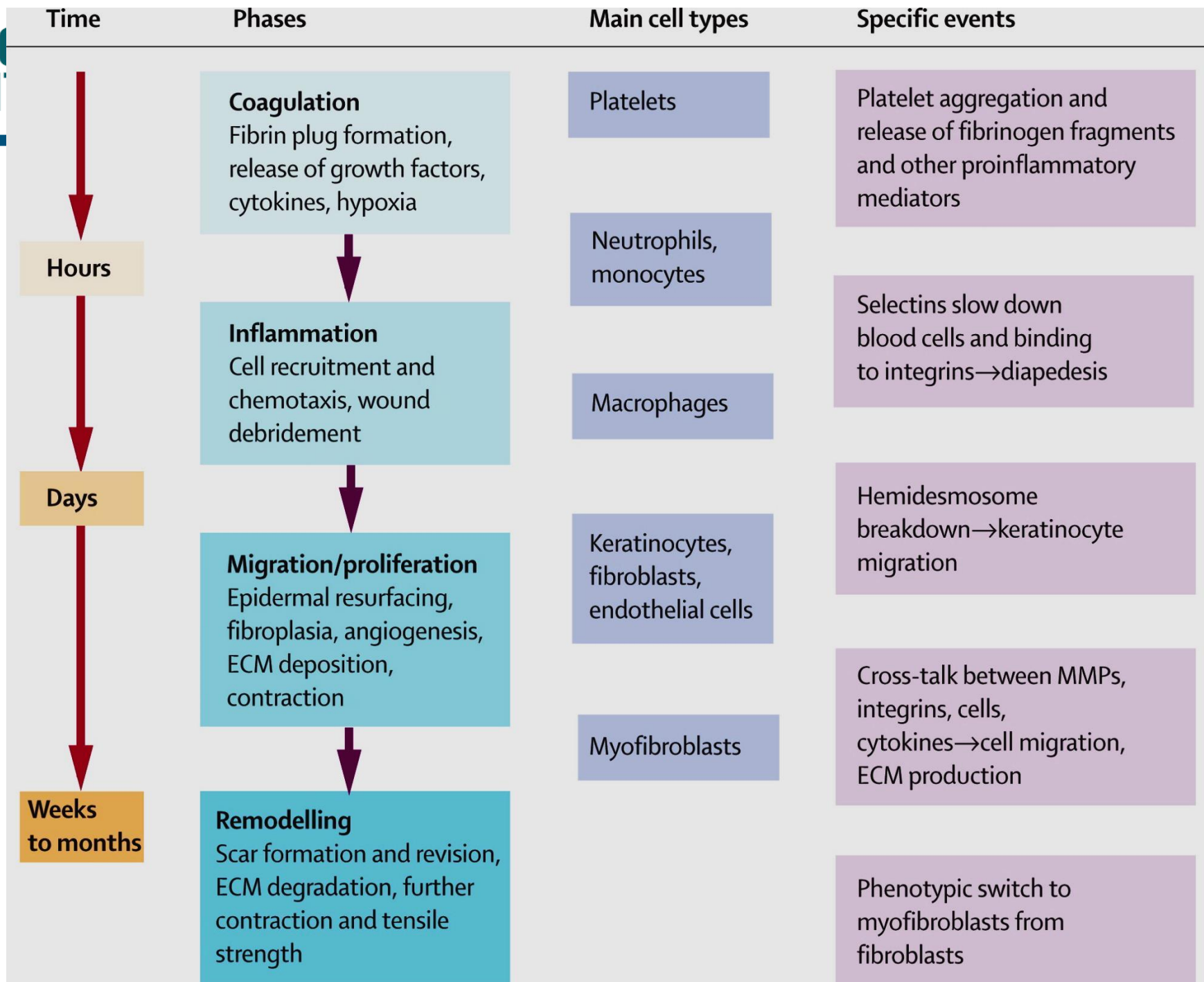
Granulation tissue

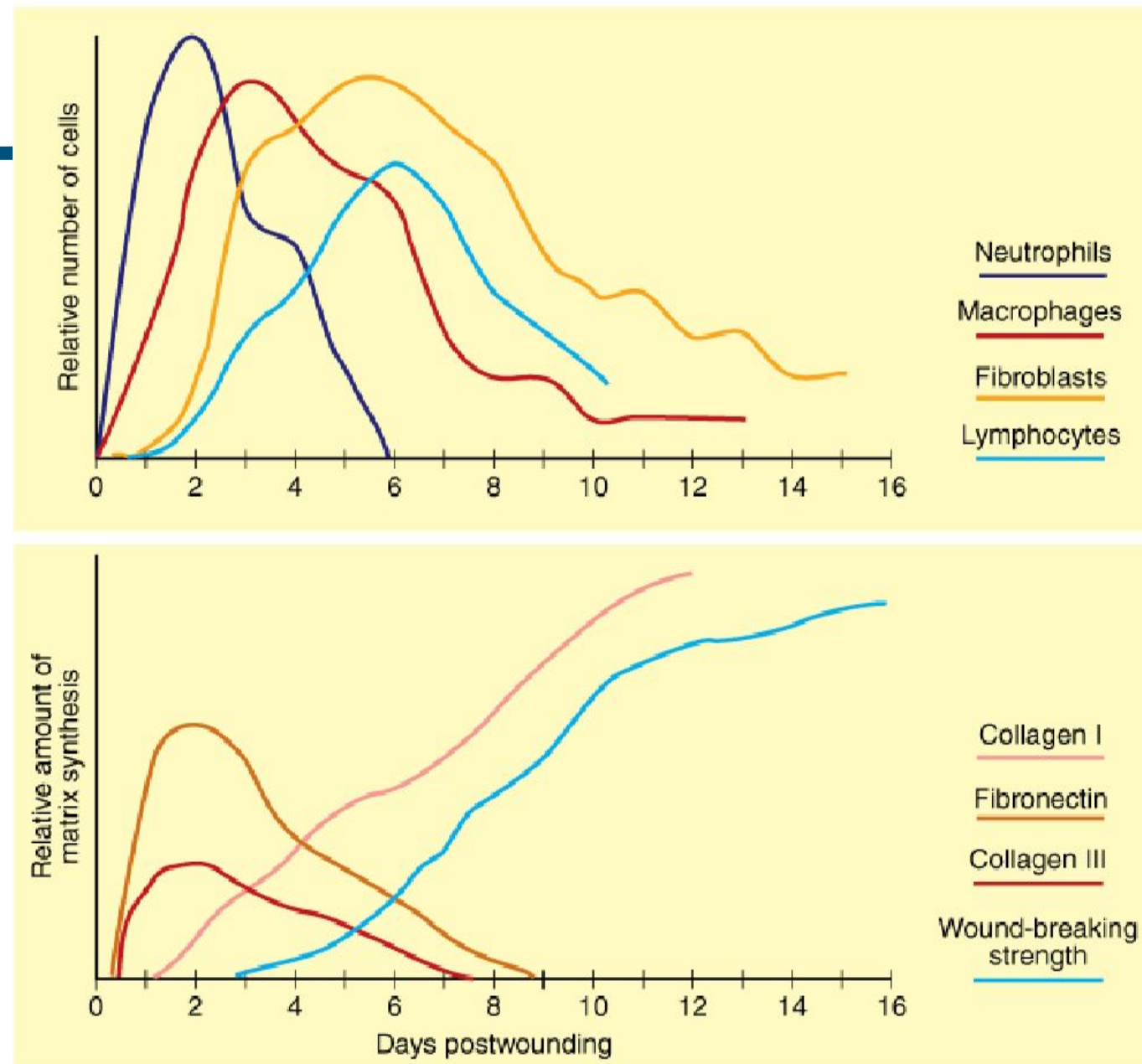


Closure with
wide scar

4 Phases of wound healing







Source: Brunicaudi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE: *Schwartz's Principles of Surgery, 9th Edition*: <http://www.accessmedicine.com>
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Coagulation/Inflammatory Phase

Platelets adhere to damaged blood vessels

Hemostatic reaction

Platelets release growth factors & cytokines

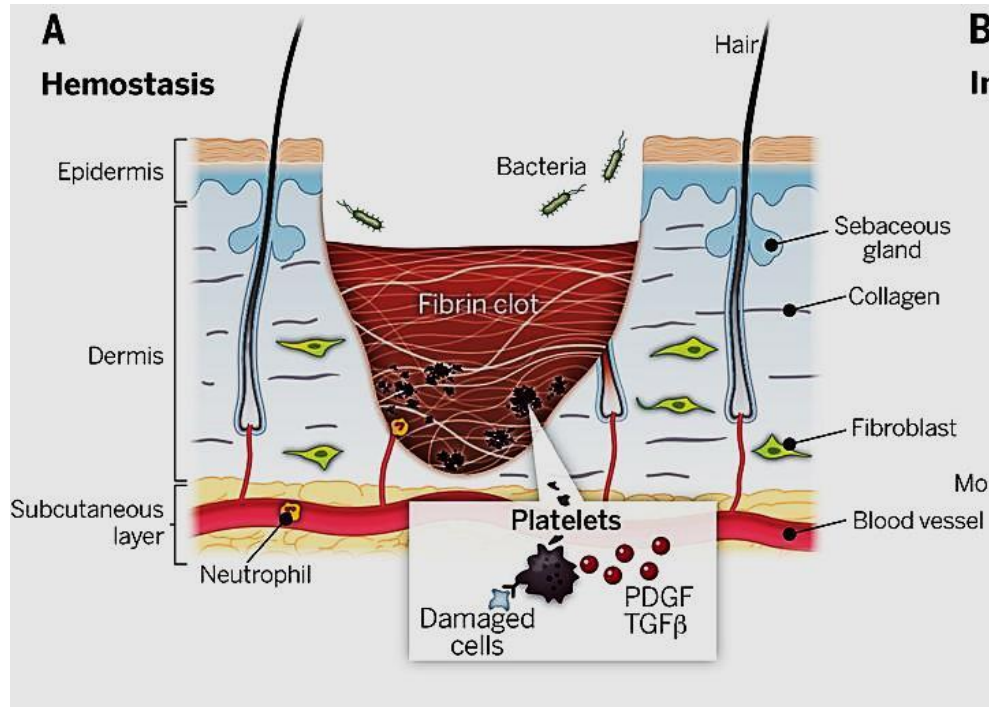
- **Platelet-derived growth factor**
- **Transforming growth factors A1 and 2**

Neutrophils, and Macrophages

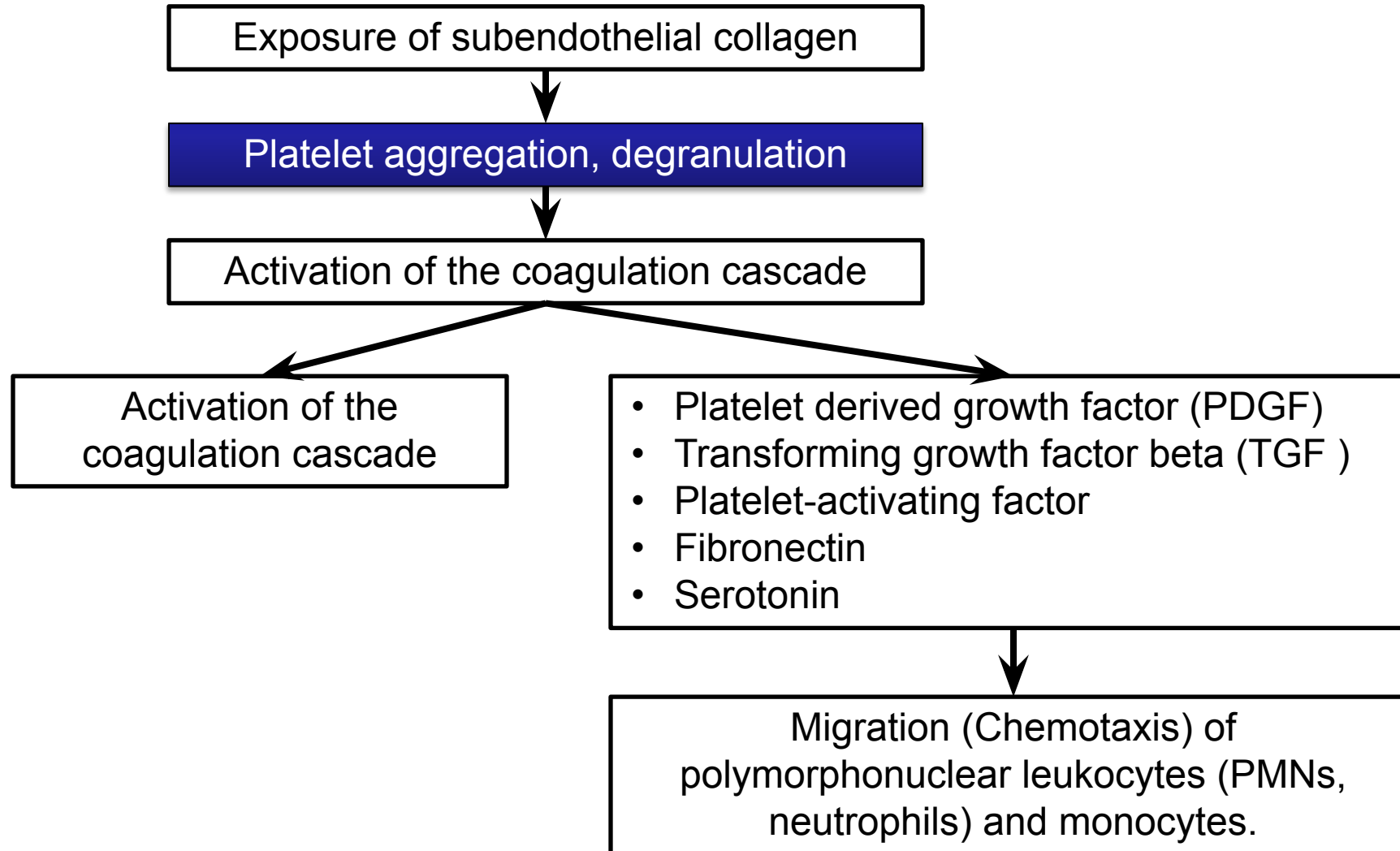
Reactive oxygen species that are antimicrobial

Proteases that clear the wound of foreign bodies and bacteria

Apoptosis of inflammatory cells, which occurs gradually within a few days after wounding

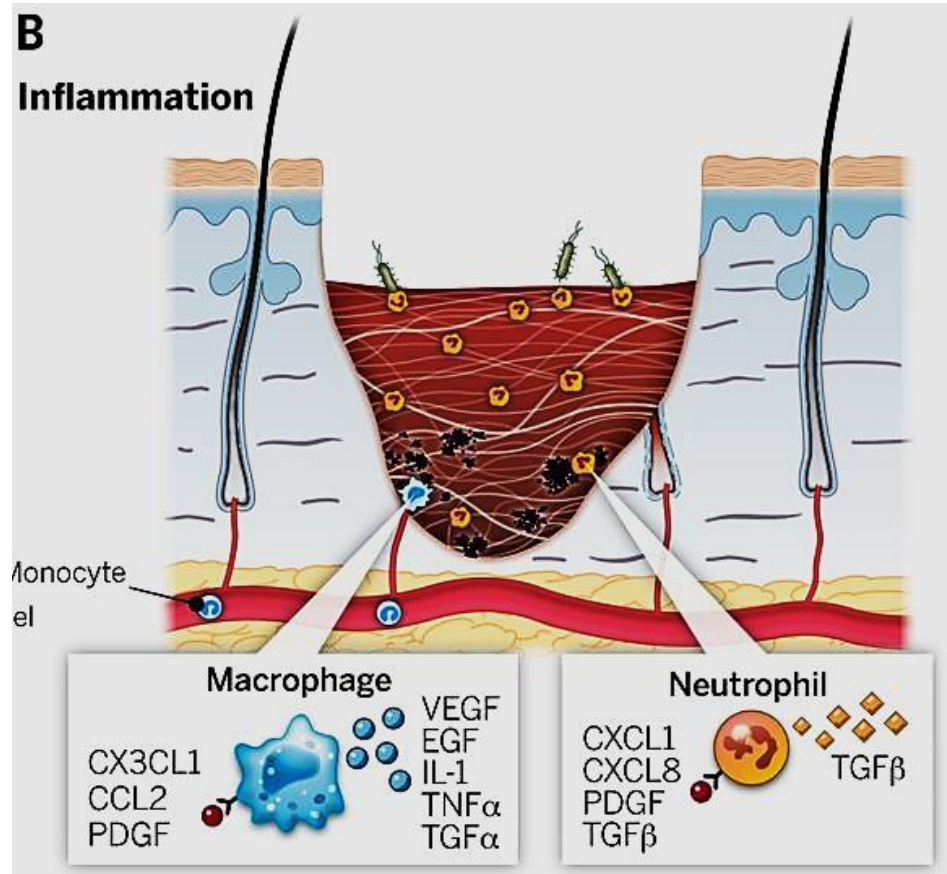


Coagulation/Inflammatory Phase

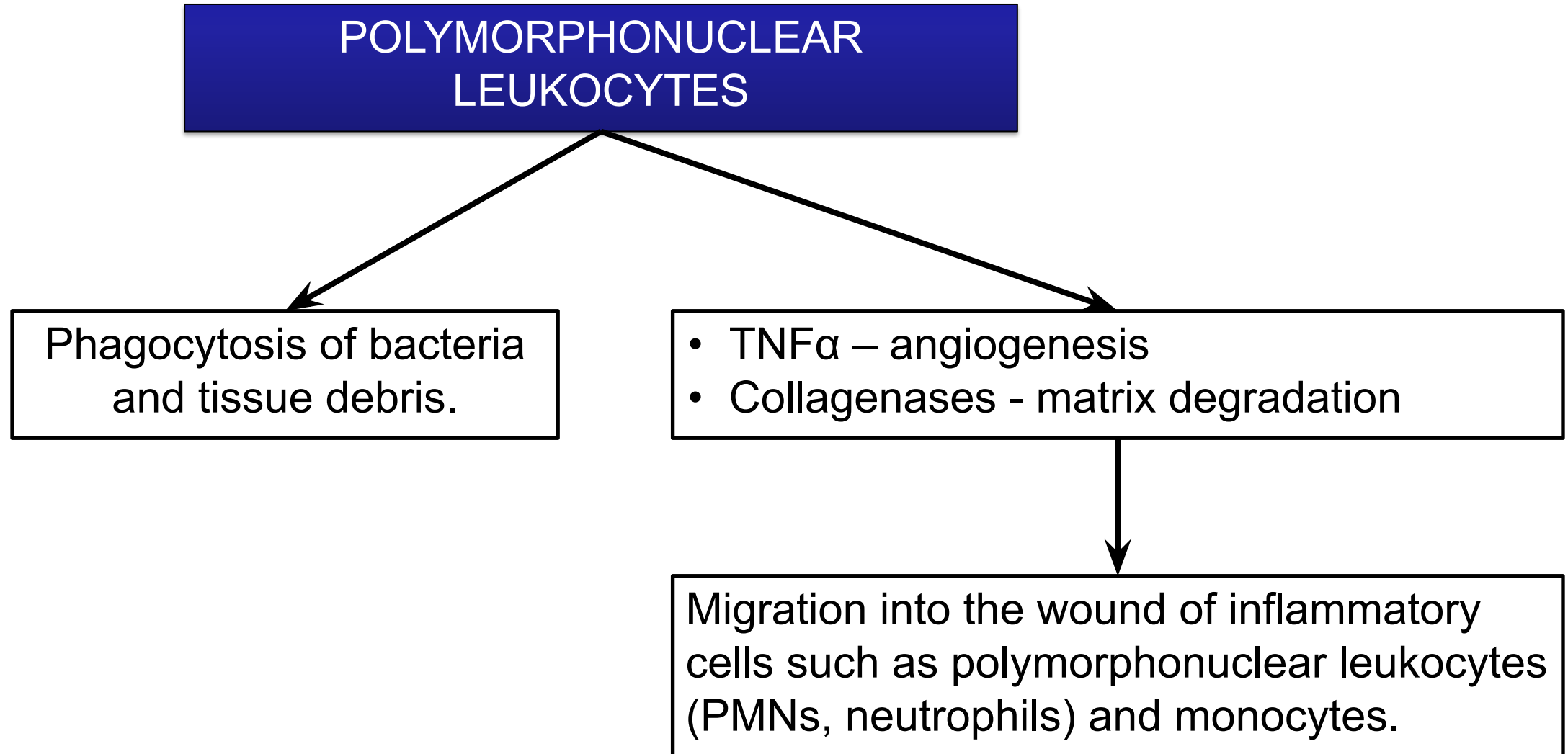


Starts immediately, lasts 3-5 days

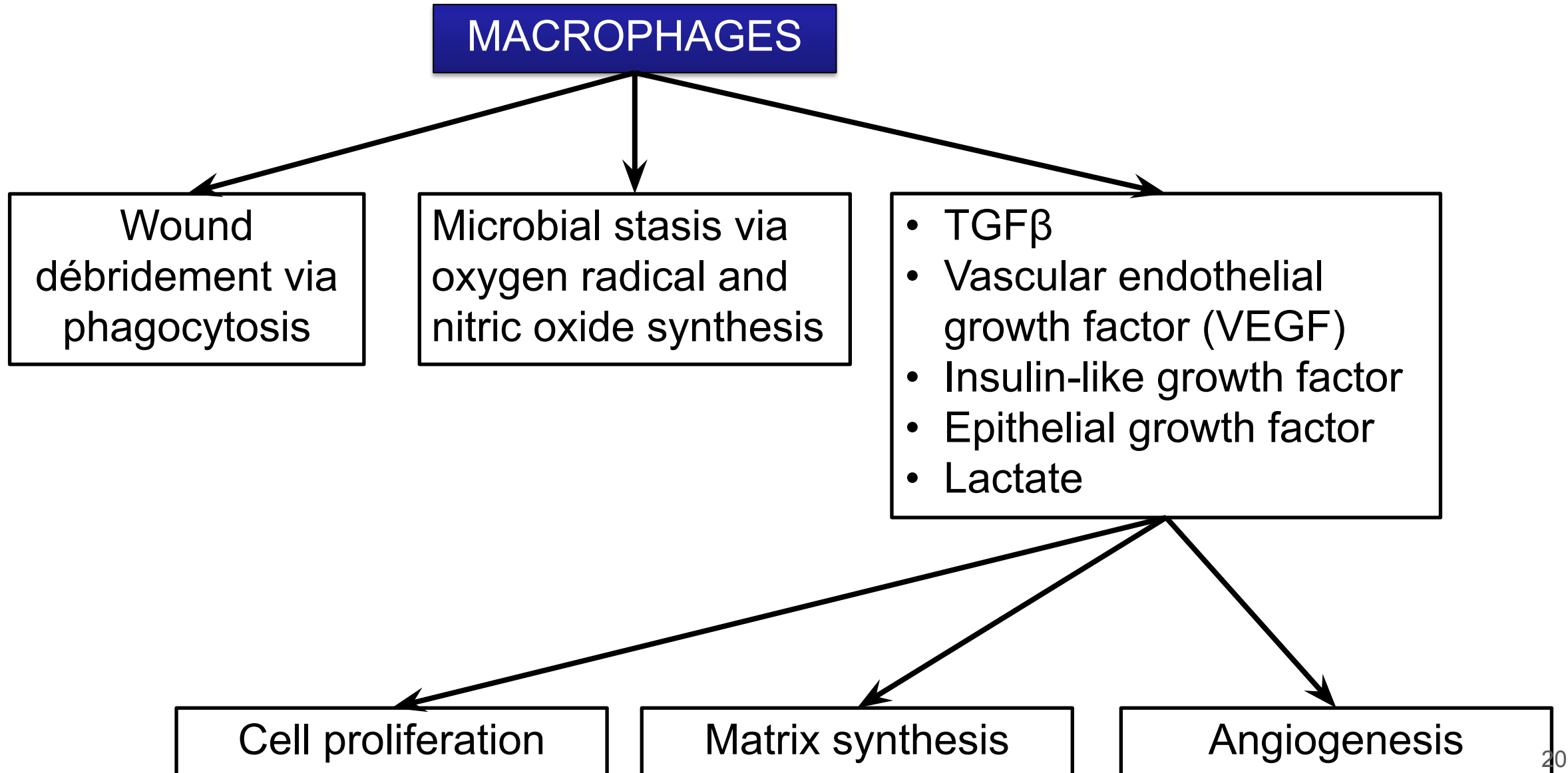
1. Vasoconstriction
2. Blood clot formation
3. Platelets aggregation
4. Platelets degranulation
5. Vasodilation
6. Chemotaxis
 - a. Leucocytes
 - b. Neutrophils
 - c. Macrophages



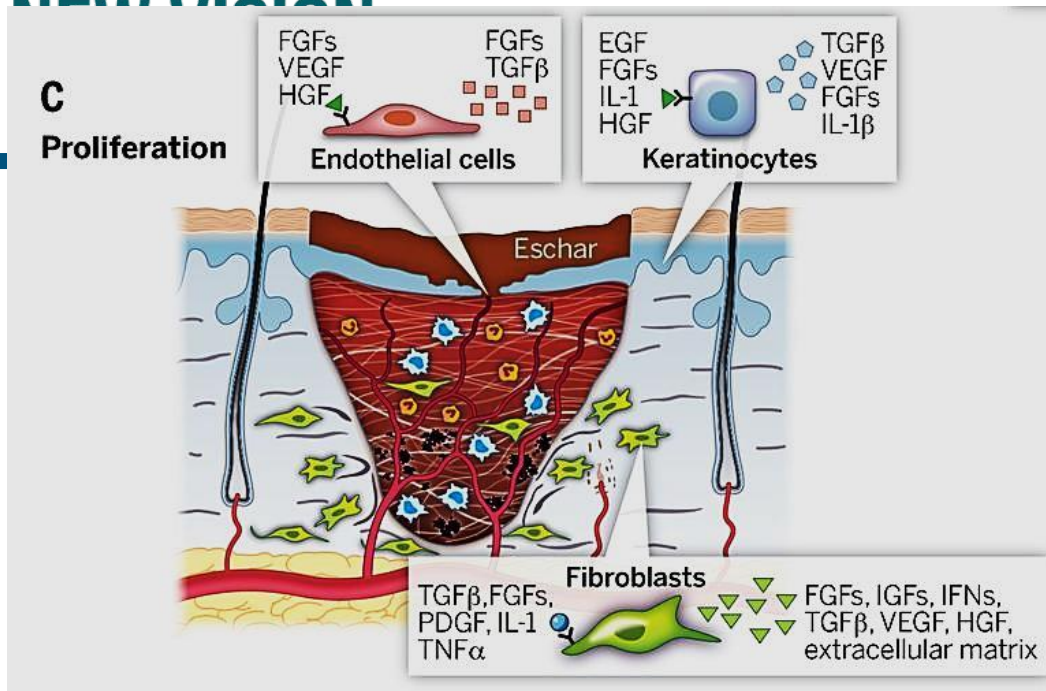
Coagulation/Inflammatory Phase



Coagulation/Inflammatory Phase

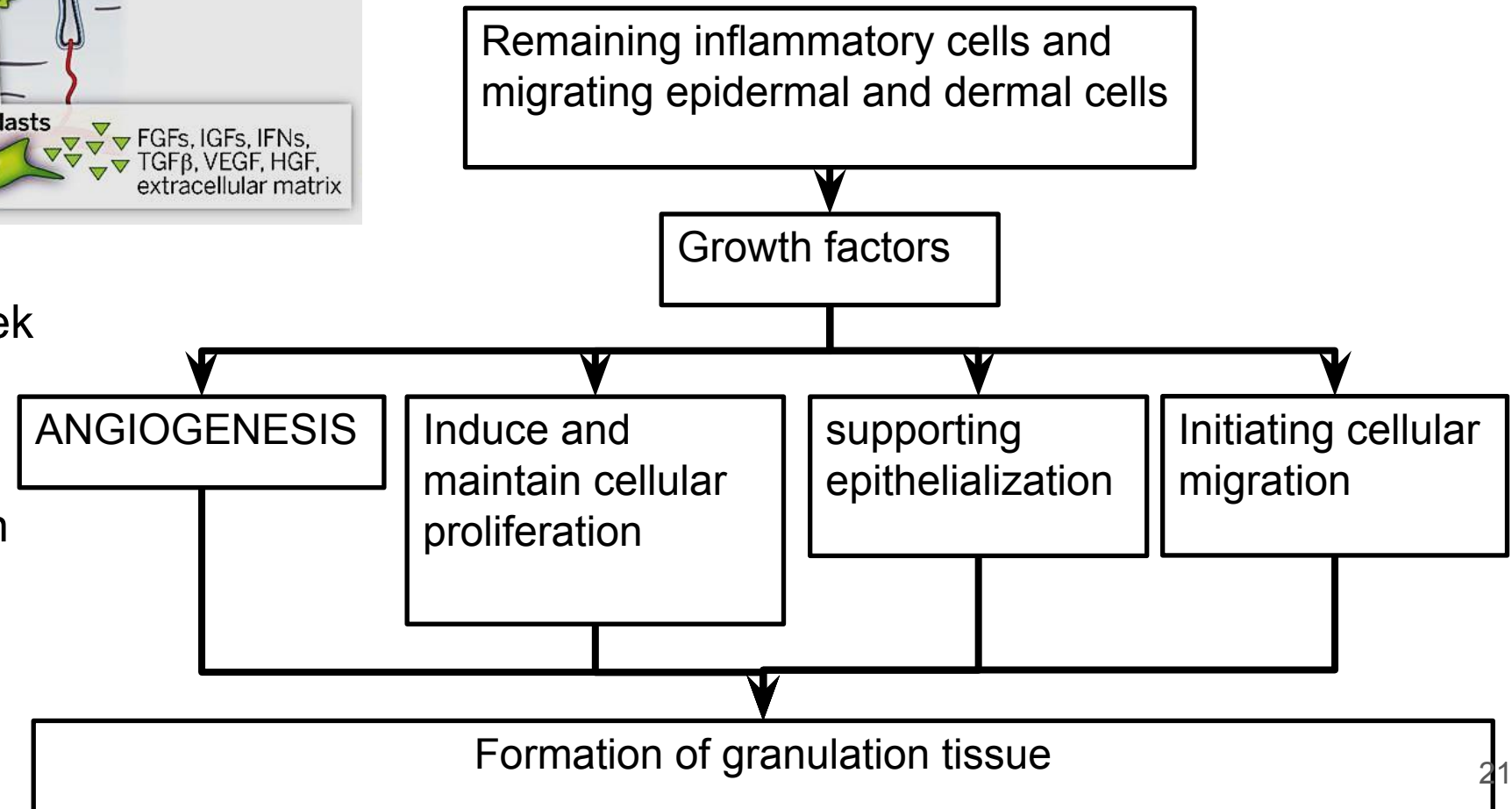


Proliferative phase

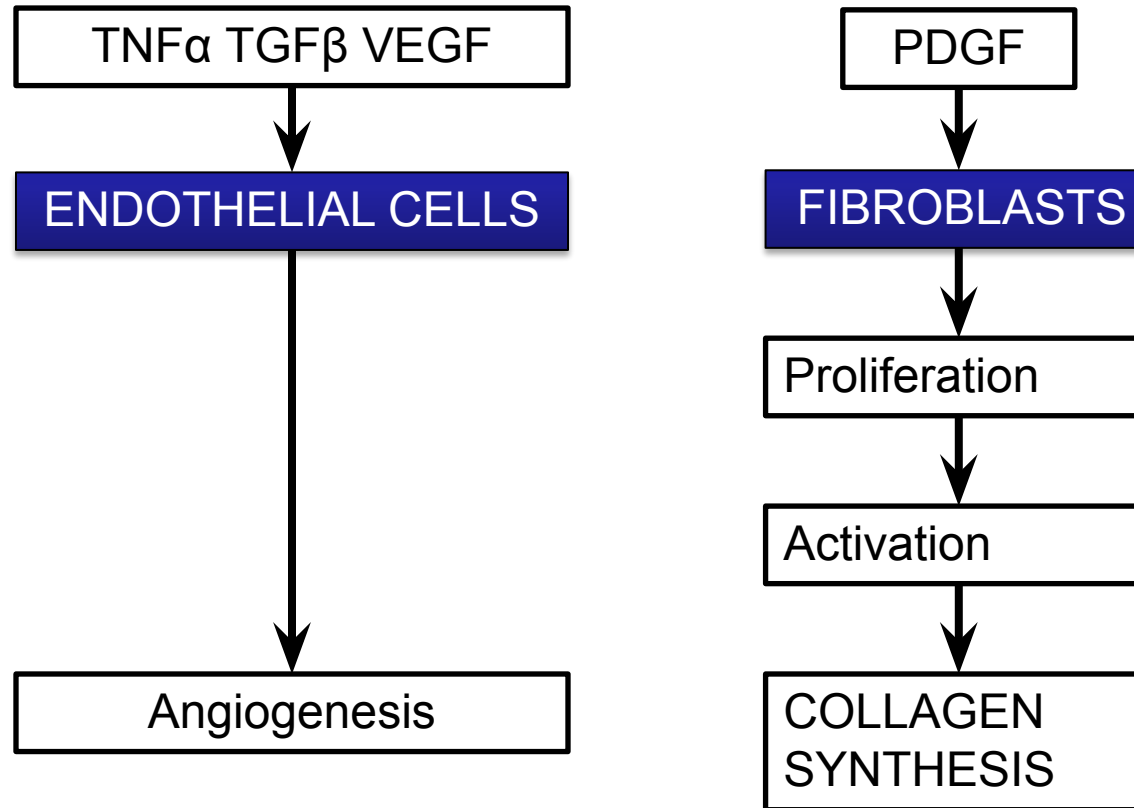


Starts 3rd day, lasts for 3rd week

1. Angiogenesis
2. Fibroblasts migration
3. Formulation of granulation tissue
4. Epithelization



Coagulation/Inflammatory Phase



Angiogenesis

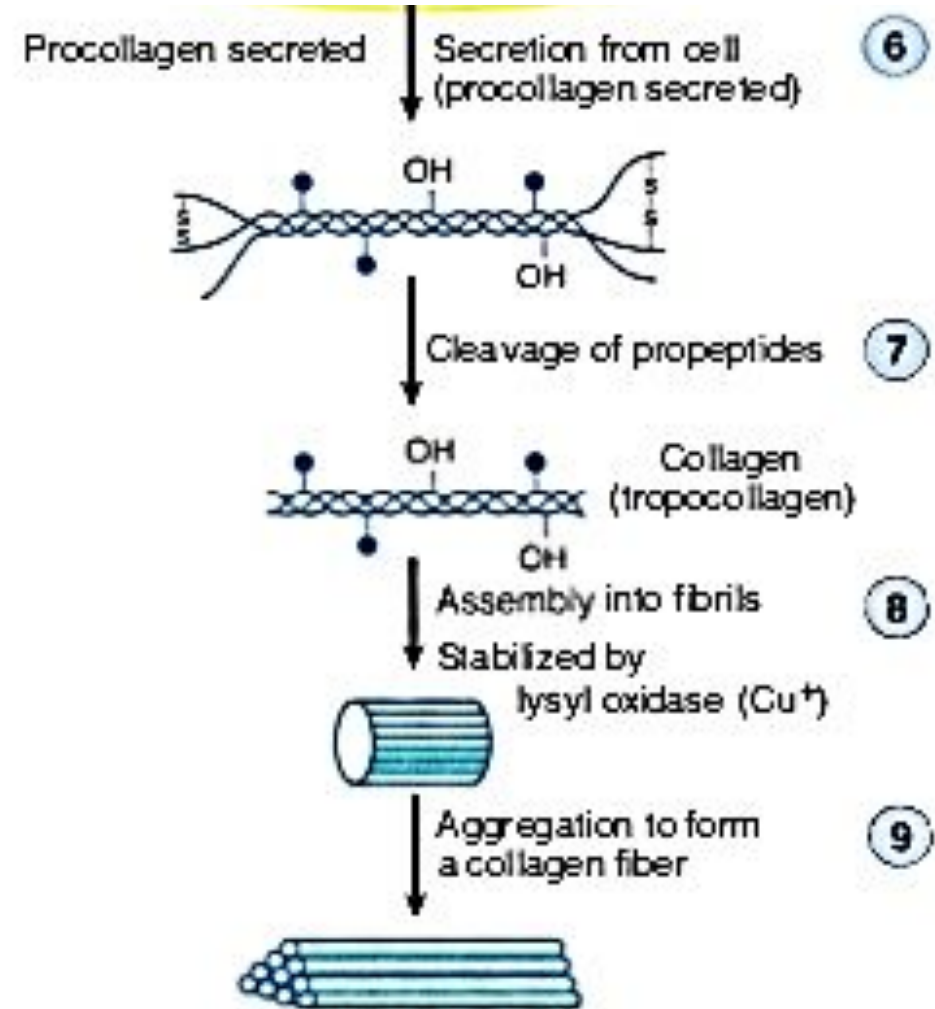
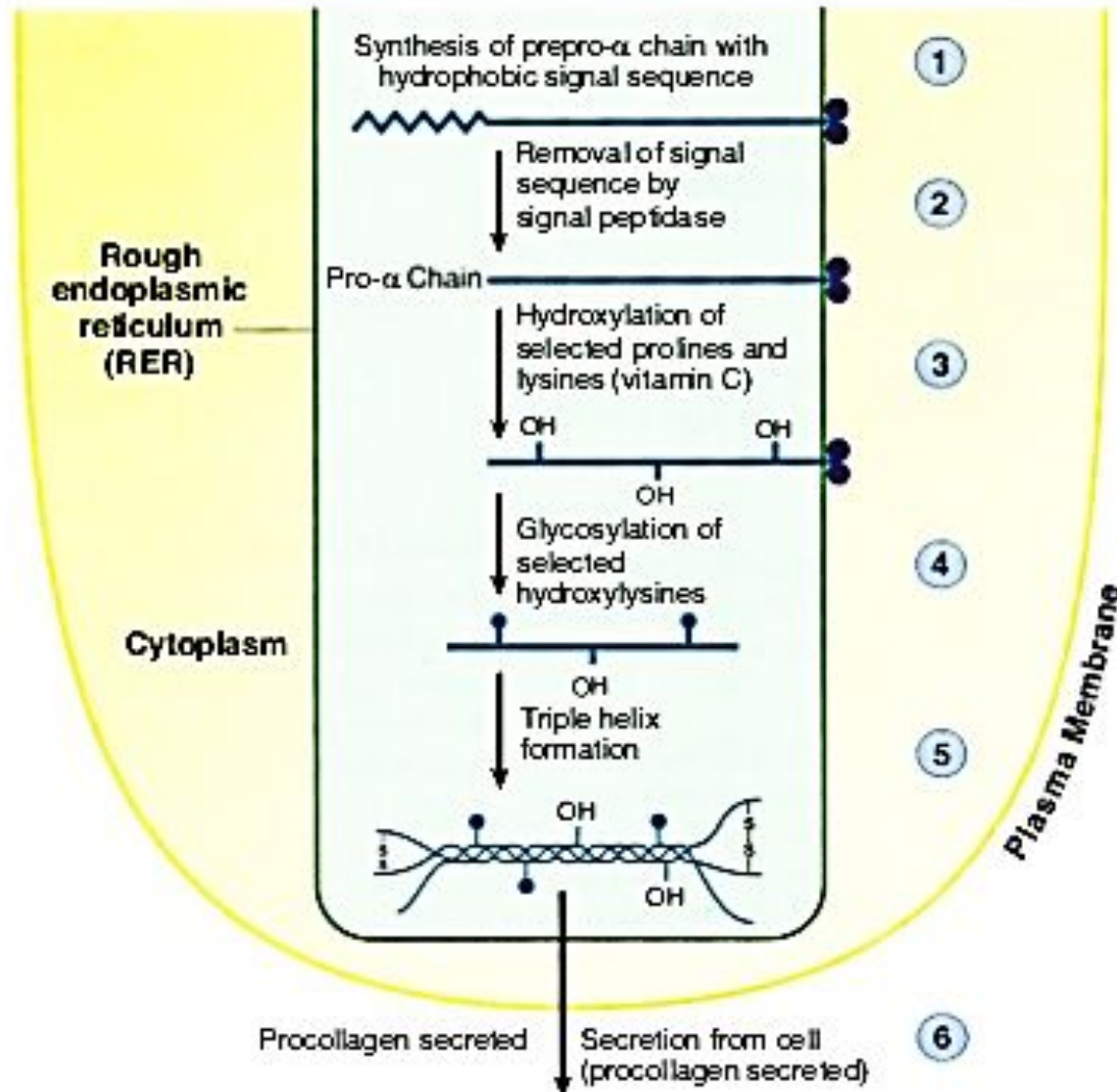
Production of proangiogenic factors:

- Vascular endothelial growth factor
- Fibroblast growth factor 2
- Platelet-derived growth factor

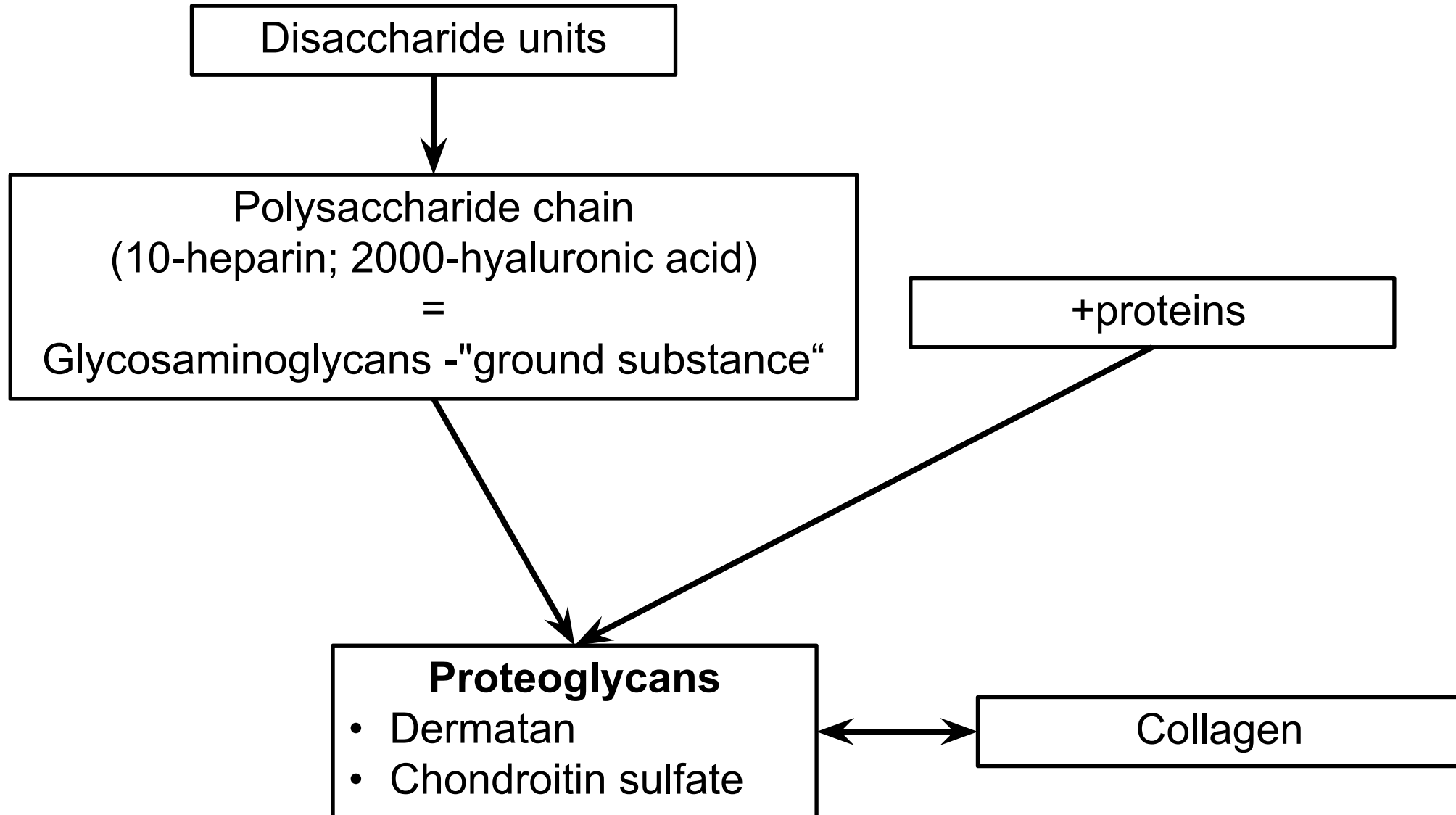
Endothelial cells degrade basement membrane, migrate toward the wound site, proliferate, and form cell-cell contacts and eventually new blood vessels.

Endothelial progenitor cells are also required for wound revascularization. Normally, EPCs reside in the bone marrow, mobilization is mediated by nitric oxide, Vascular endothelial growth factor, and matrix metalloproteinases, stomal cell–derived factor 1, insulinlike growth factor (IGF).

Collagen synthesis



Extracellular matrix



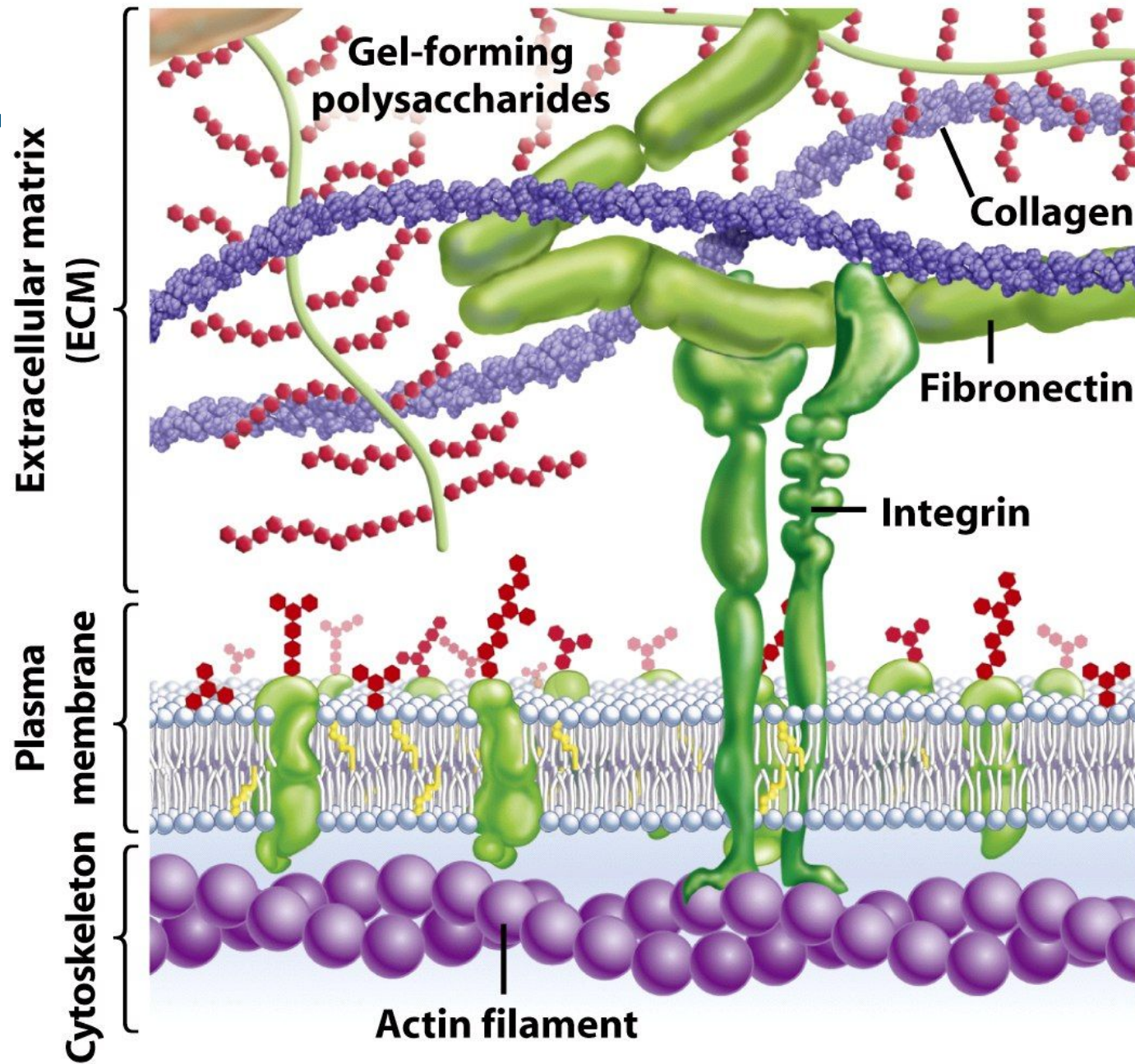
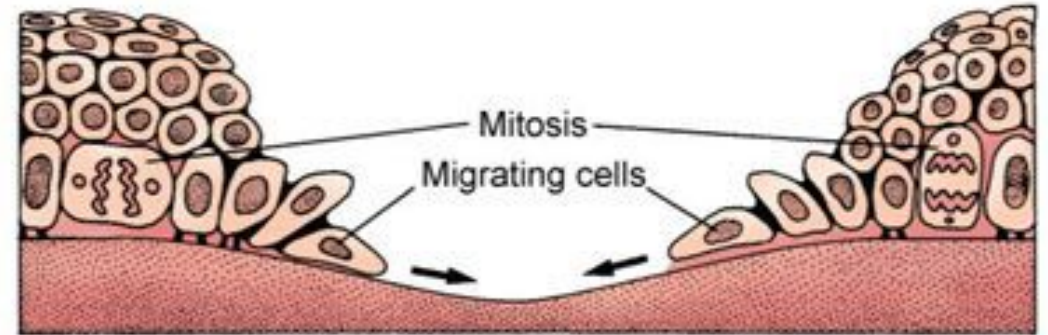
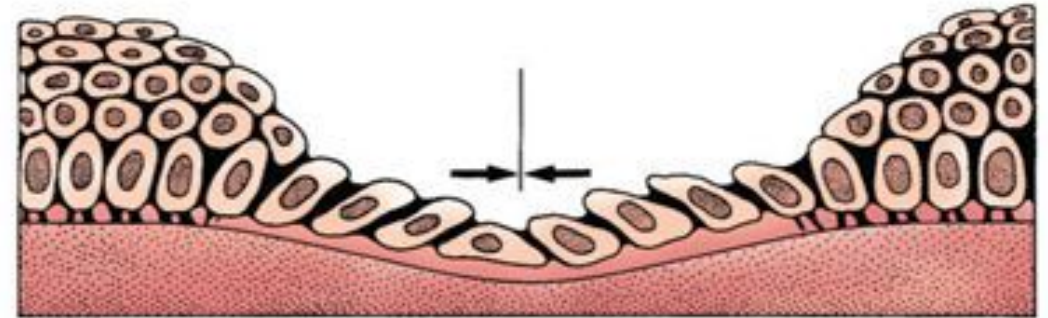


Figure 8-4 Biological Science, 2/e

Re-Epithelization



Skin cells at the edge of the wound begin multiplying and migrate toward the center of the wound.



Once advancing epidermal cells from the opposite sides of the wound meet, migration halts.

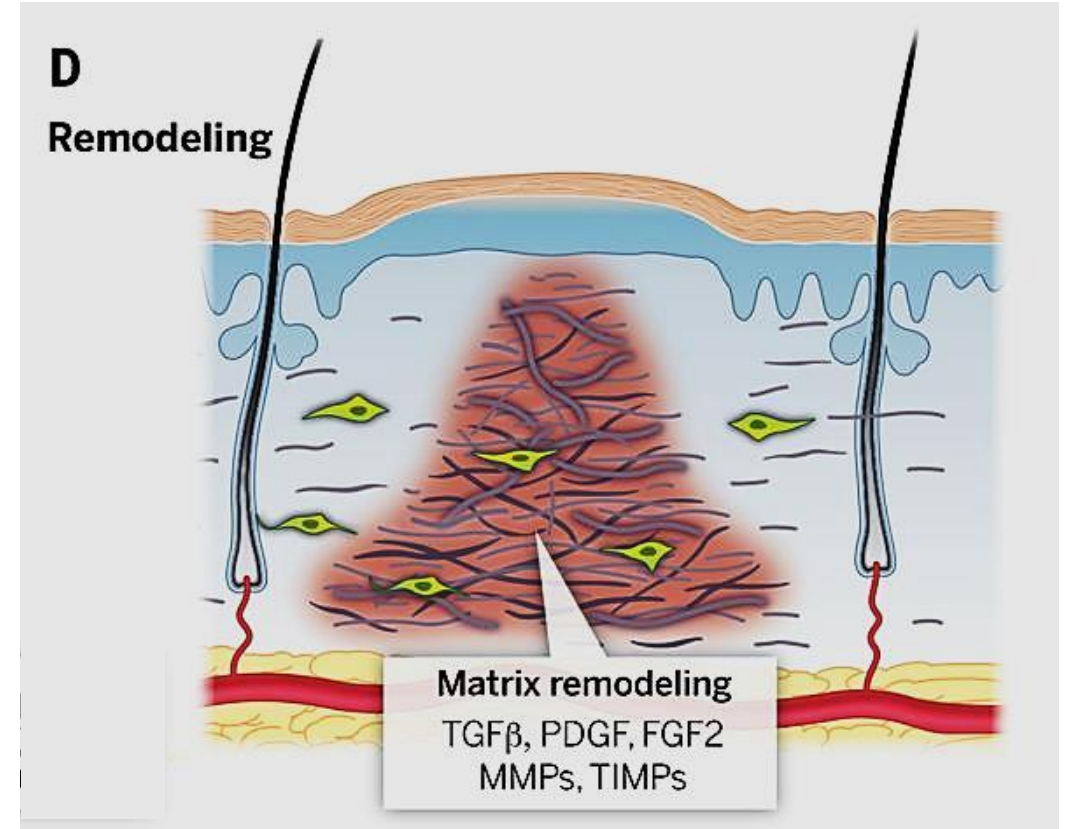


Epithelial cells continue to divide until the thickness of the new skin layer approaches normal.

Remodeling phase

Starts from weeks to years

- The fibroblasts start to disappear
- **Collagen type III is gradually replaced by stronger type I collagen**
- Type III collagen is broken down by matrix metalloproteinases
- The tensile strength of the scar tissue gradually increase
- Mature, avascular, and acellular scar formation



Understanding wound-healing phases

The three phases of wound healing—inflammatory, proliferative, and remodeling—overlap. During each phase, key physiologic events occur and specific nutrients play a vital role in healing.

Phase, onset, and duration	Key events	Essential nutrients
Inflammatory phase Starts at time of injury and lasts 4 to 6 days	• Hemostasis • Release of pro-inflammatory cytokines • Vasodilation • Phagocytosis of bacteria by neutrophils • Removal of debris and growth-factor secretion by macrophages	• Vitamins A, C, and K • Iron • Zinc • Protein • Fat • Carbohydrates
Proliferative phase Starts 3 or 4 days after injury and lasts 2 to 3 weeks	• Fibroblast proliferation • Angiogenesis • Collagen synthesis • Collagen deposition and cross-linking • Development of granulation tissue and extracellular matrix • Epithelialization • Onset of wound closure by myofibroblasts	• Vitamins A and C • Iron • Copper • Zinc • Manganese • Protein • Carbohydrates
Maturation/remodeling phase Starts about 21 days after injury and lasts up to 2 years	• Collagen maturation and stabilization • Increased tensile strength • Scar-tissue formation	• Vitamins A and C • Zinc • Protein

- Ehlers-Danlos syndrome
- Marfan syndrome
- Osteogenesis imperfecta
- Epidermolysis bullosa
- Acrodermatitis enteropathica

Ehlers-Danlos Syndrome

Genetic connective tissue disorder

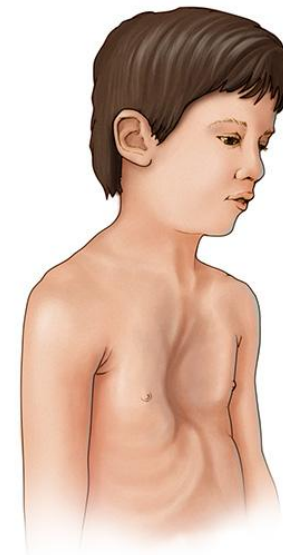
- Loose joints
- Stretchy skin
- Abnormal scar formation
- Easy bruising
- Recurrent hernias
- GI bleeding, hiatal hernia, intestinal diverticulae, rectal prolapse.
- Aneurysms, varicosities, arteriovenous fistulas



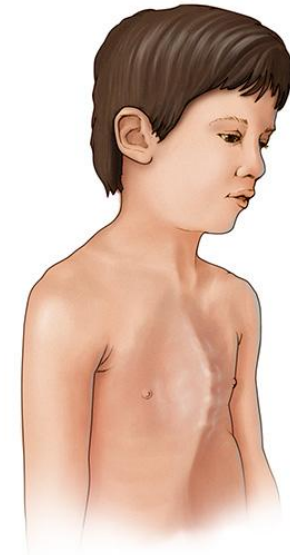
Marfan syndrome

The genetic defect is in an extracellular protein, fibrillin

- Tall stature, arachnodactyly, lax ligaments, myopia, scoliosis, pectus excavatum.
- Aneurysm of the ascending aorta
- Hernias.



Indented breastbone
(pectus excavatum)



Protruding breastbone
(pectus carinatum)

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Osteogenesis imperfecta

Brittle bone disease

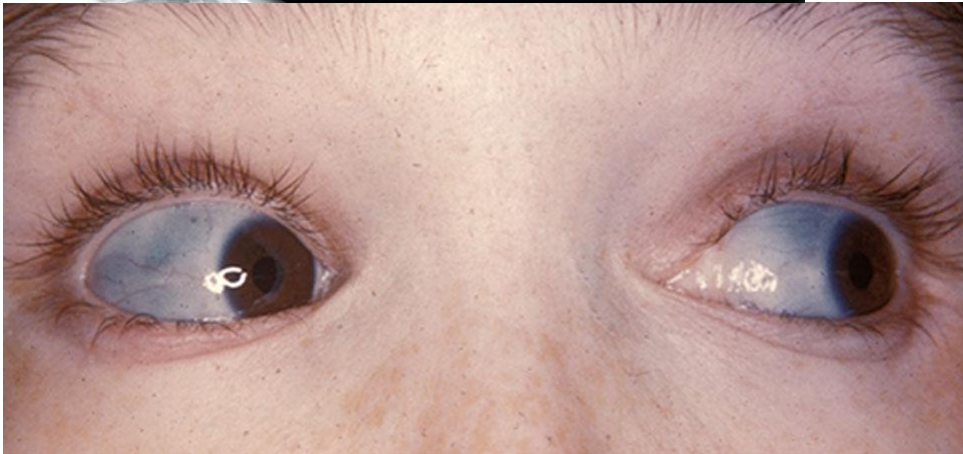
Mutation in type I collagen

Bones fracture easily under minimal stress,
osteopenia

Hernias

Ligament and joint laxity

Dermal thinning and increased bruisability.

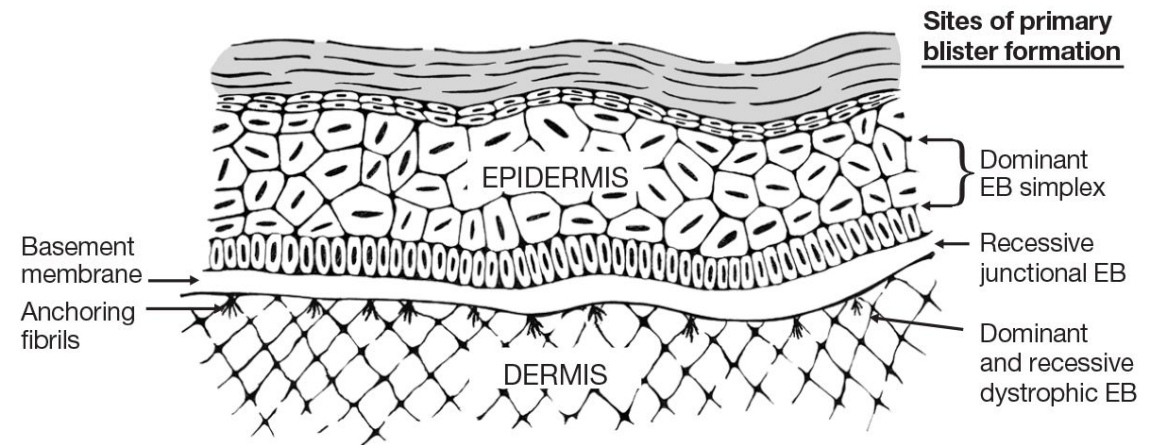


Epidermolysis Bullosa



Impairment in tissue adhesion within the epidermis, basement membrane, or dermis, resulting in tissue separation and blistering with minimal trauma.

Skin Structure



Source: Adapted from Dystrophic Epidermolysis Bullosa Research Association of America (DeBRA).

Acrodermatitis Enteropathica



Inability to absorb
sufficient zinc from
breast milk or food



Zinc deficiency



Impaired granulation
tissue formation



**Erythematous
pustular dermatitis
around the bodily
orifices**

