WOUND HEALING

DEPT. PATOLOGI VETERINER
FKH UNAIR 2018

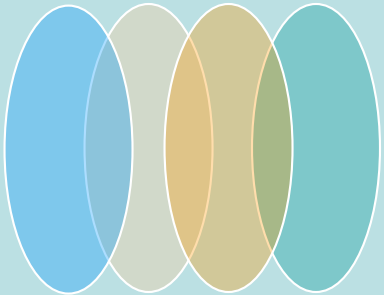
- Healing Process occur almost immediately After wound develop
- Classically wound healing devine in four overlap phase :

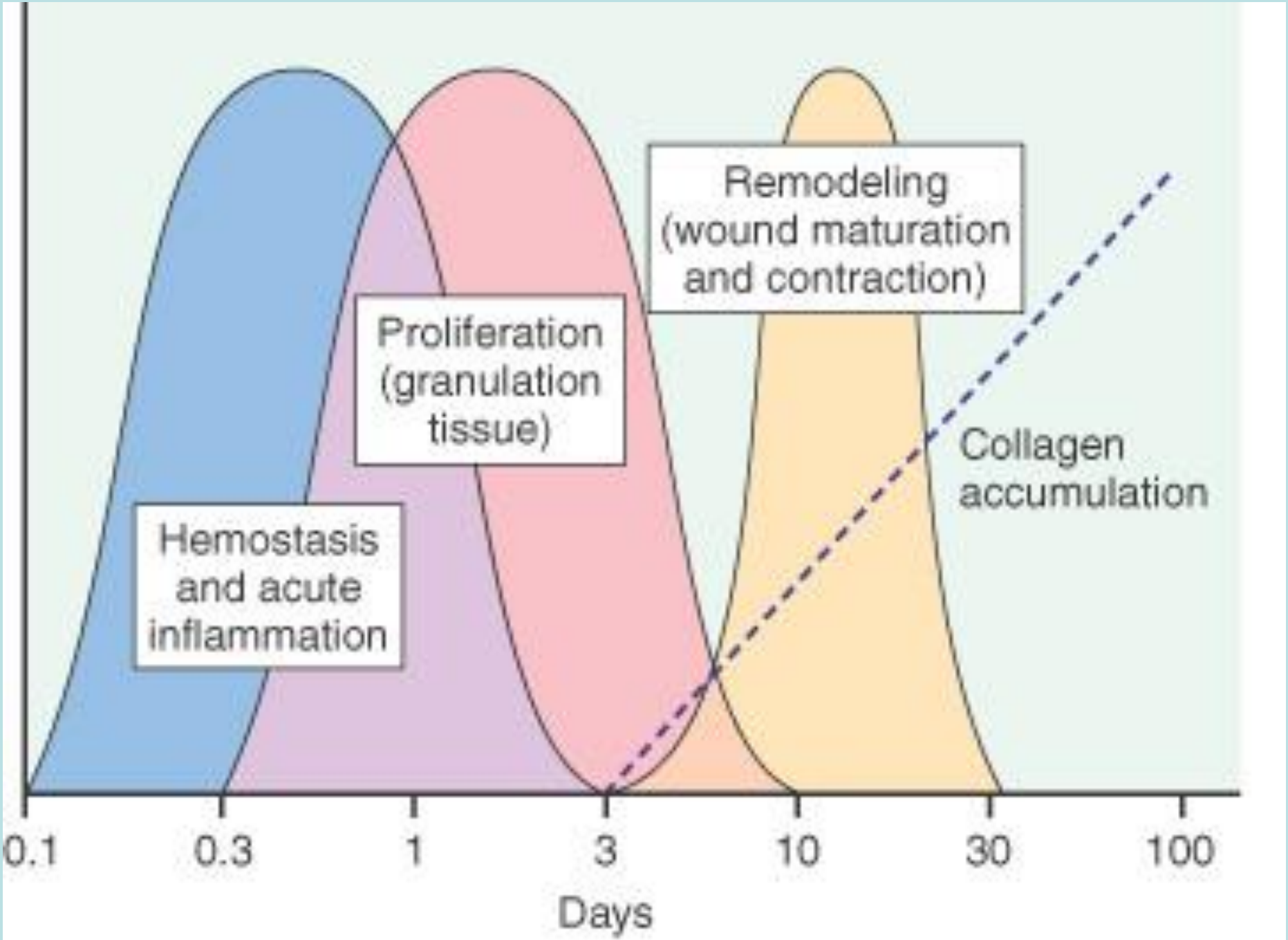
(1) Hemostasis

(2) Acute Inflamation

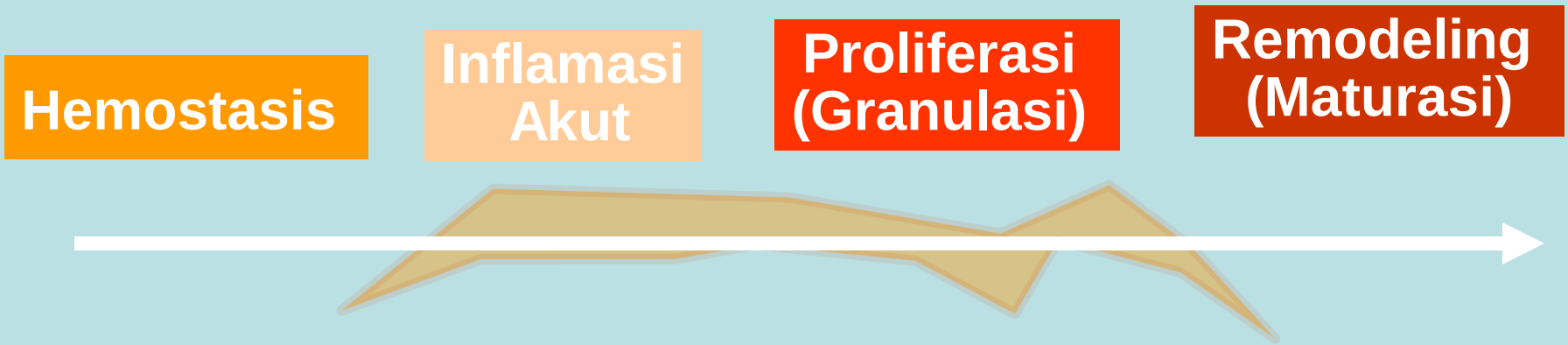
(3) Granulation

(4) Remodeling (Maturation / Contraction)



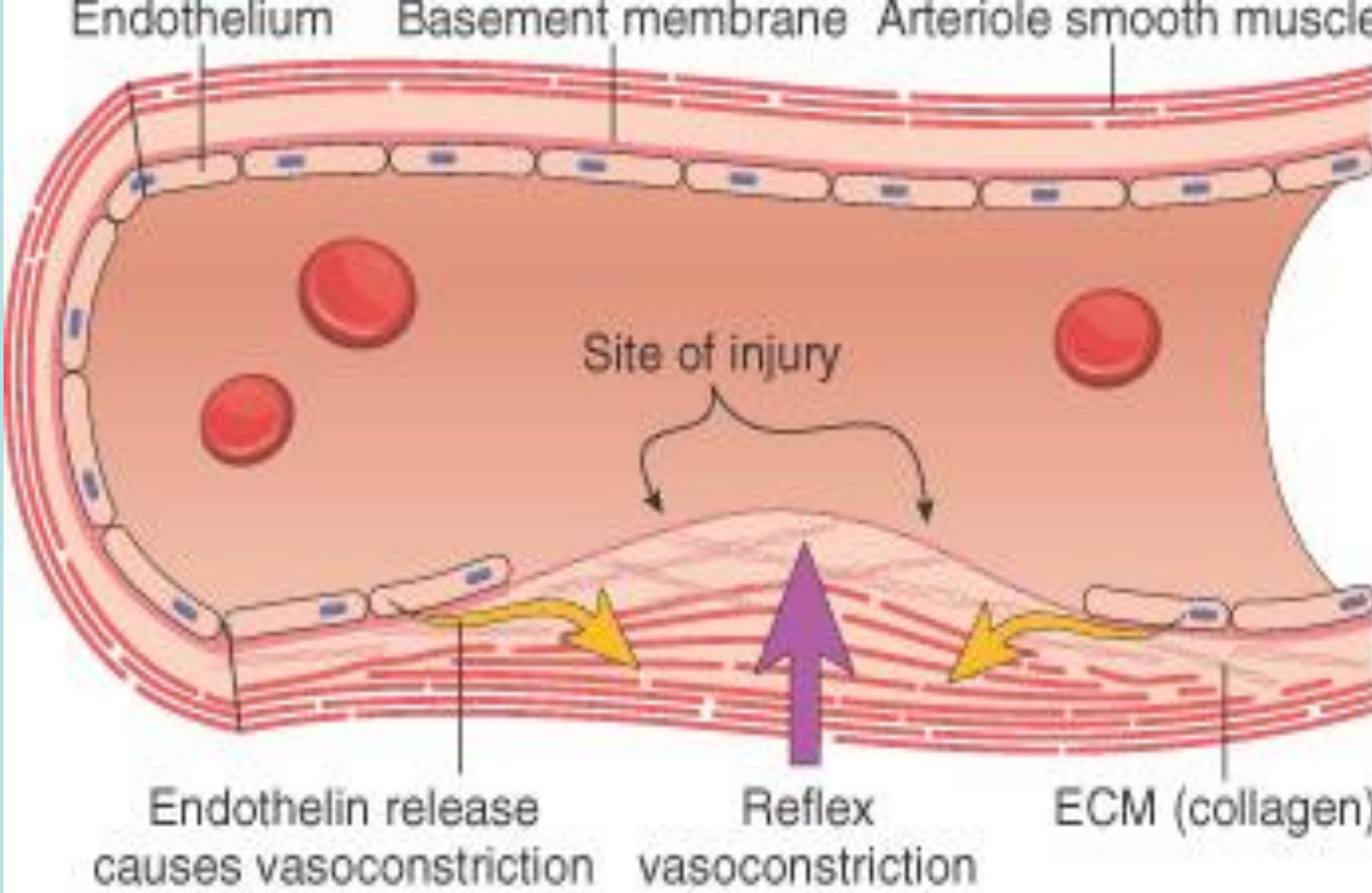

Remodeling
(wound maturation
and contraction)
Proliferation
(granulation
tissue)
Collagen
accumulation
Hemostasis
and acute
inflammation
0.1
0.3
1
3
10
30
100
Days

- The Phases of wound healing Occur in sequence with different time

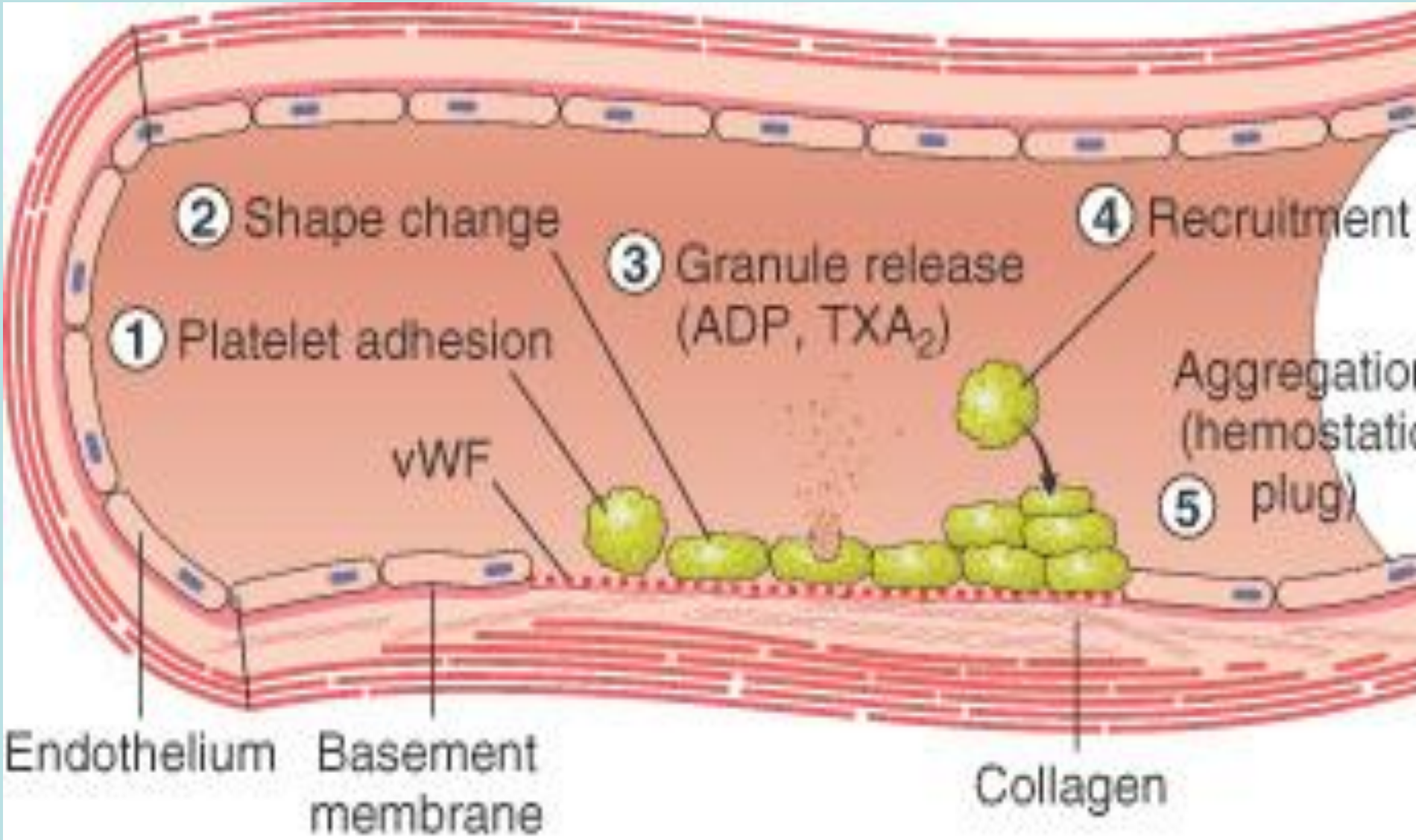


Sequences of Hemostasis

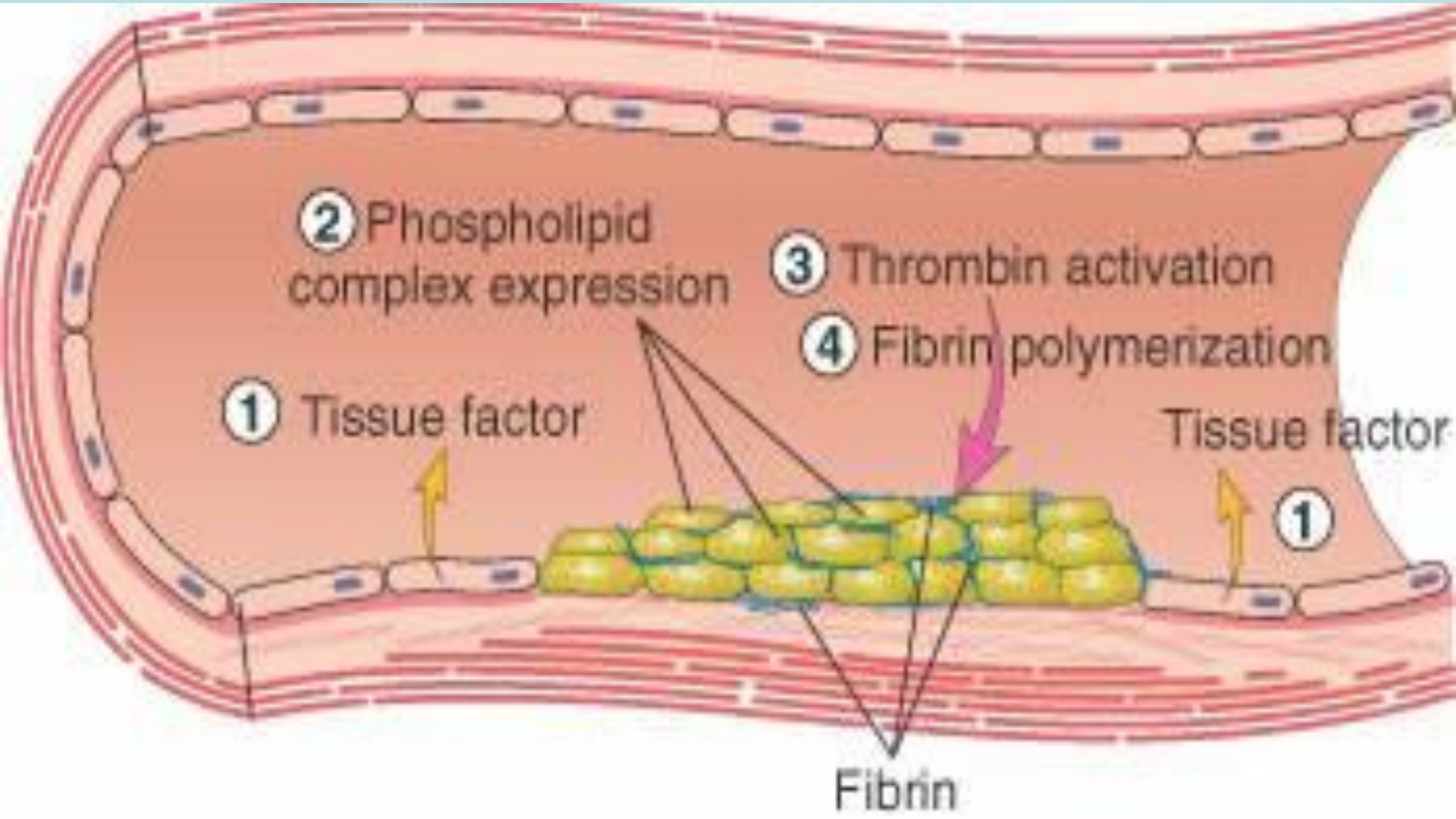
1. injury → induce vascular and platelet to release neurogenic mediators following vasoconstriction immediately after damage.
2. Narrowing of the vessel lumen allow opposing endothelial surface into contact each other to reduce blood loss
3. pletelets adhere to exposed subendothelial matrix of collagen, fibro nectin, and other glycoproteins and proteoglycans
4. local activated endothelium release von Willebrand`s factor that cause more platelet adhere
5. Platelets within the aggregate secrete the content of their dense bodies and α-granuls and produce substances such as thromboxane to accelerate hemostasis
6. ADP-ase released from dense granules triggers the binding of fibrinogen to platelets receptor GpIIb-IIIa, resulting in th formation of bridges that link platelets into a **loose aggregate → dense plug**
7. Mild injury → could be recovered just by platelet alone (plug)
   severe injury → exposed collagen and plug promote to secondary hemostasis



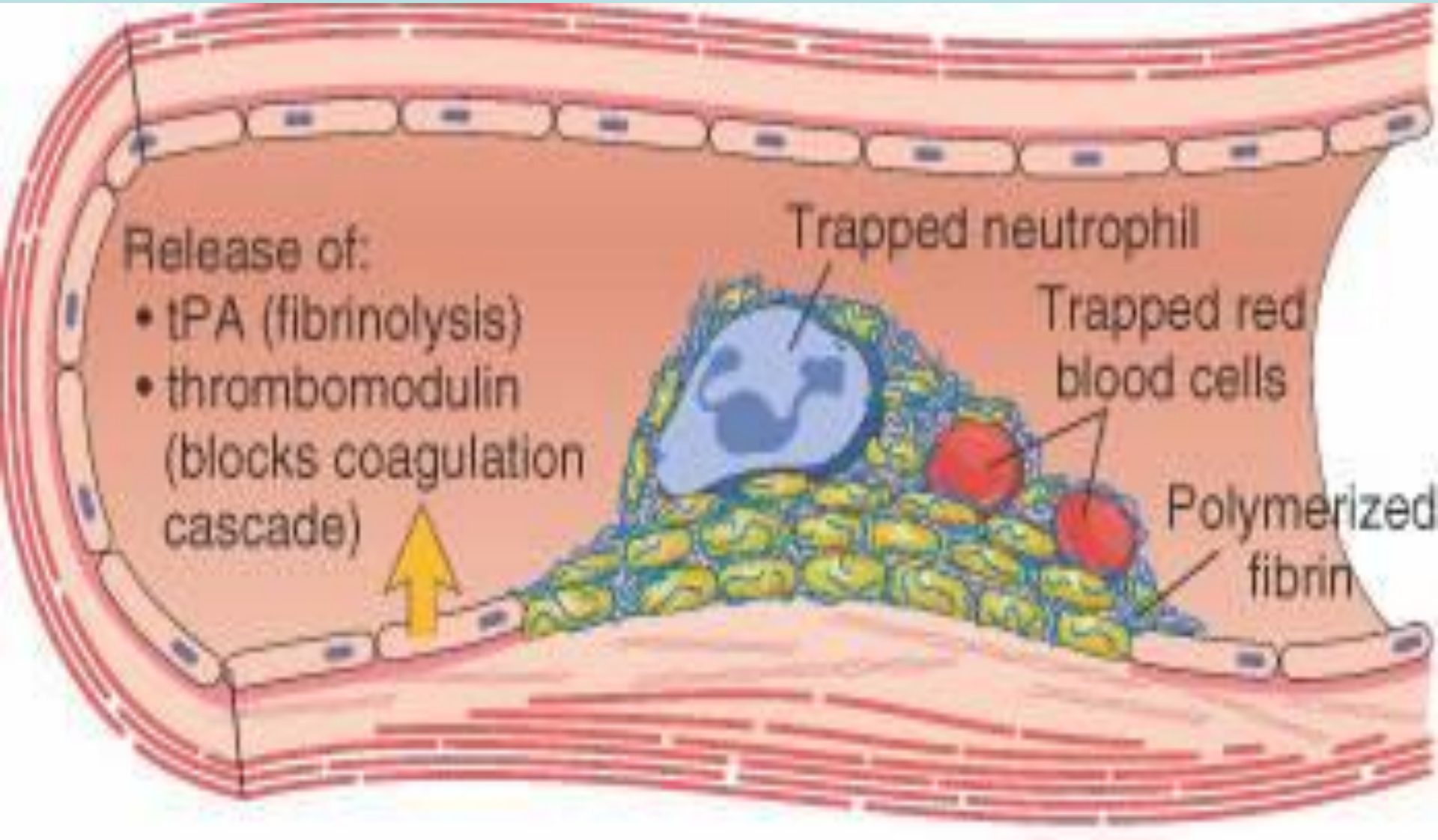
After injury, local neurohumoral factors induce a transient vasocontriction



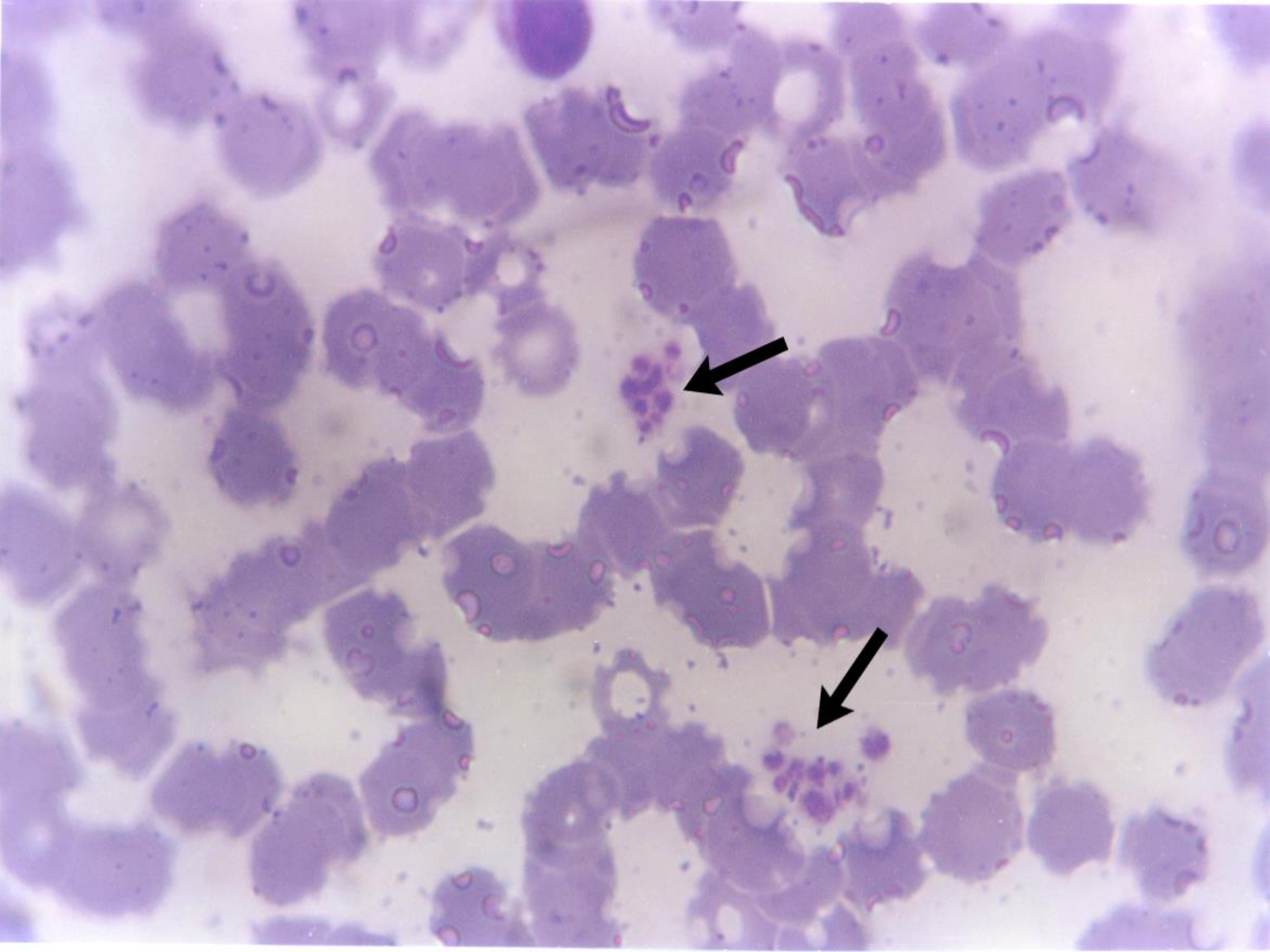
Platelets adhere to ECM via *von Willebrand`s factors (vWF)* and activated (Change shape and granule release). Released ADP and Thomboxane`s (TXA2) lead to further platelets aggregation to form primary hemostatic plug



Local activation of coagulation cascade (involving TF and platelets phospholipid) result in fibrin polymerization, cementing the platelets into a definitiv of secondary hemostatic plug



Counter-regulatory mechanism, such as releas of tissue plasminogn (tPA) (fibrinoyitic) and Throbomodulin, limit the hemostatic procss to site of injury



Inflamation Acute Phase

- Initiate in 6-7 post hemostasis phase and disappear in 24 hour post trauma
- This Phase could be prolong to 3-4 days in infected wound

- These phases known as “Cardinal Sign”

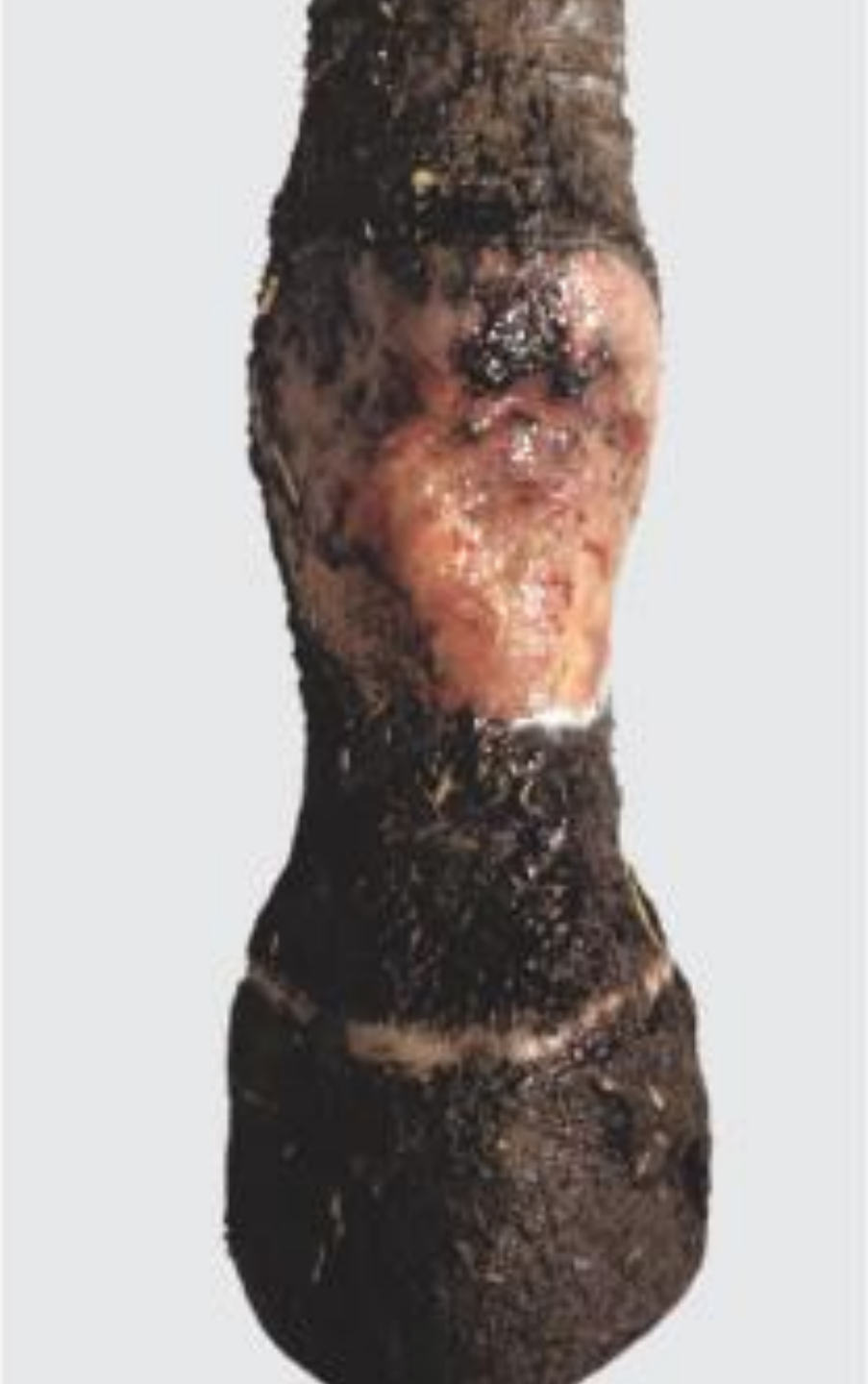
Heat (calor); Redness (rubor); Swelling (tumor); Pain (dolor)
Loss of function (fungtio laesa)

- Inflamation cell marginated on vessel wall, and provoke debris cleaning and tissue proliferation

Granulatin Phase

- It Occur 4 days post trauma and could be prolong in 3-4 weeks depent on wide of wound

- This phase characterized with Angiogenesis, fibroplasia and epitheial renewel



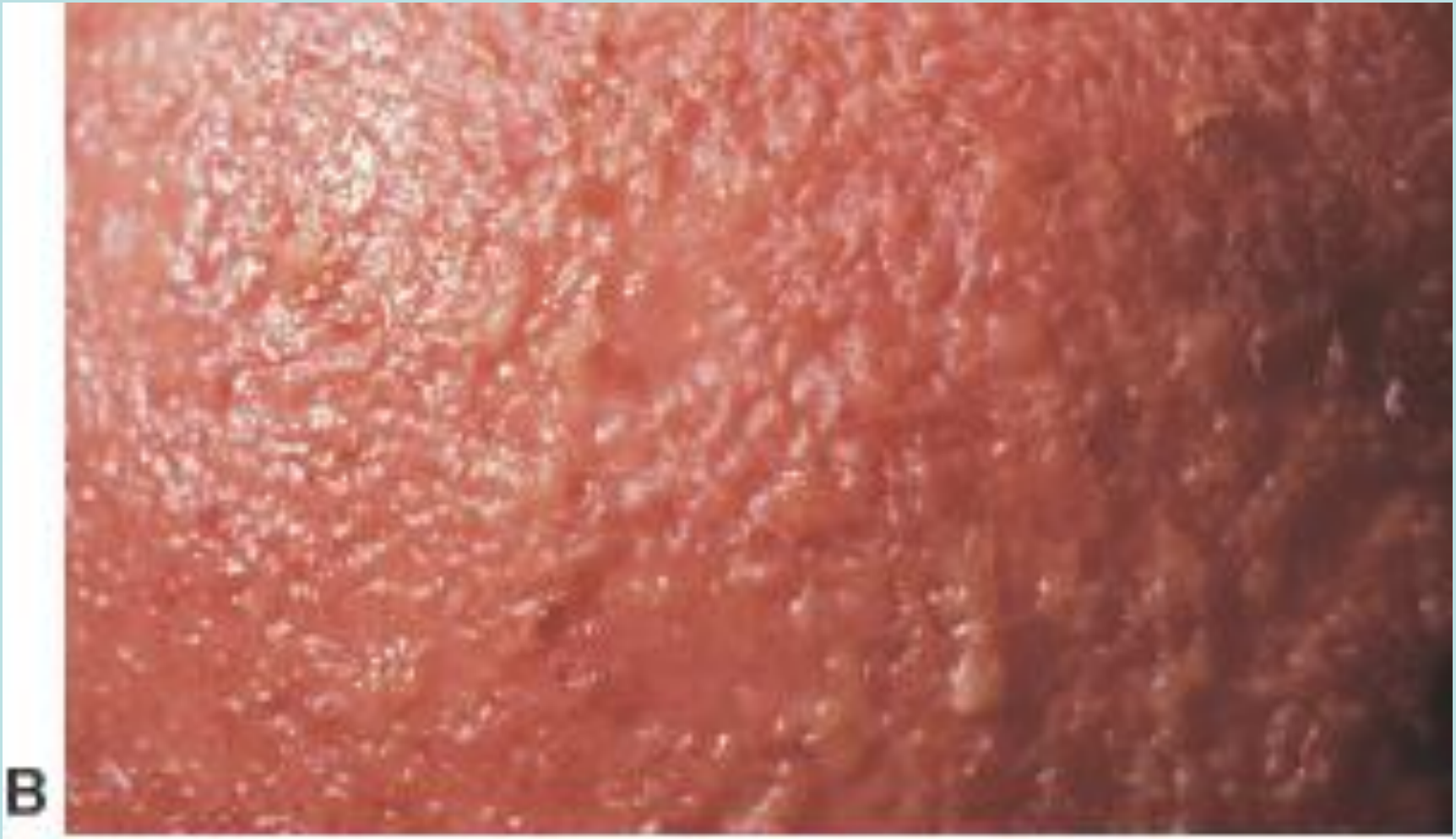


Fig. 4-27B Granulation tissue, nonhealing ulcer, skin, distal limb, horse. A, In the bed of the ulcer, there is extensive fibrosis and *granulation tissue*. **B.** Gross photograph of the

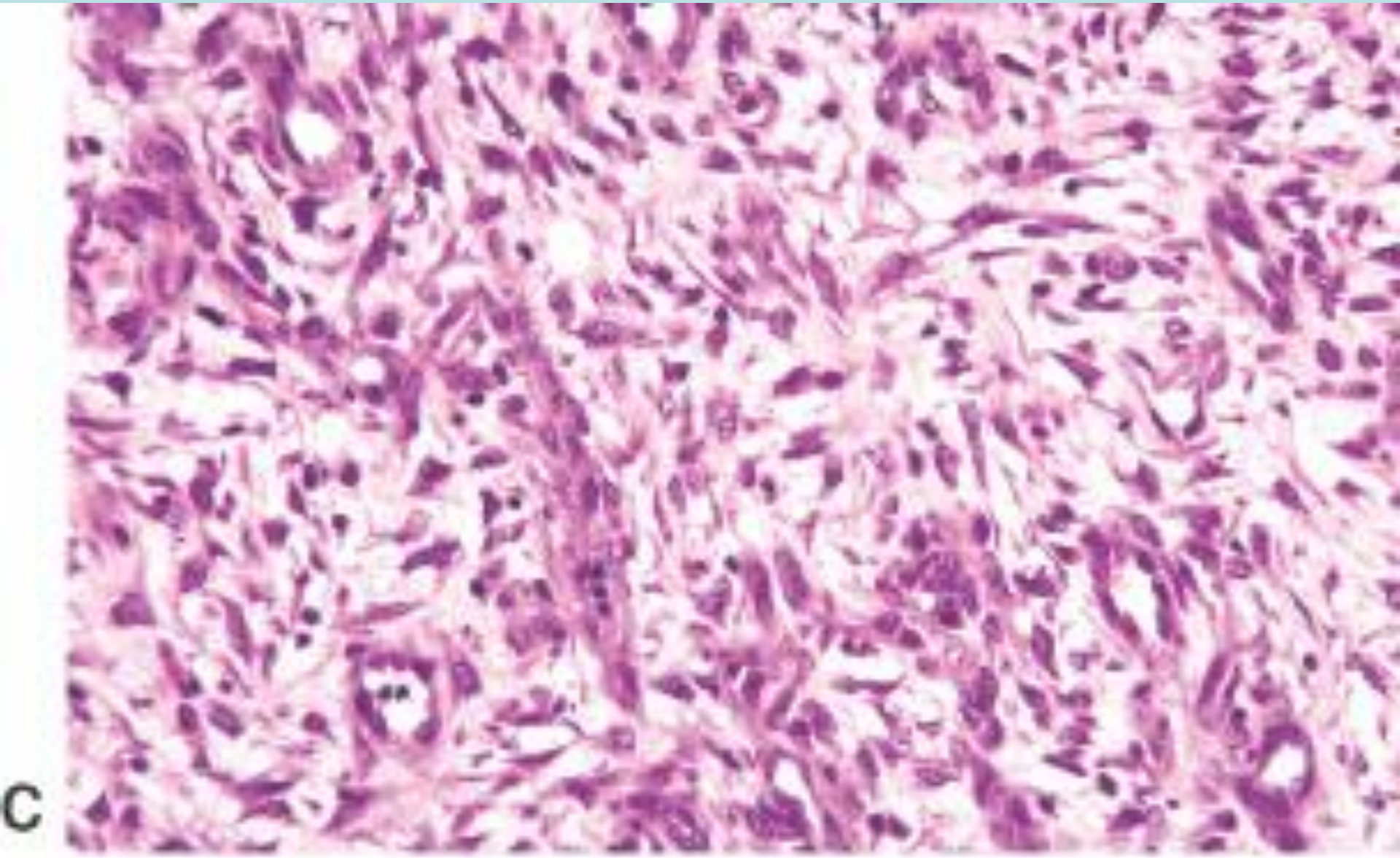


Fig. 4-27C Granulation tissue, nonhealing

Remodeling Phase (Maturation

- Occur in 3 - 4 weeks until 2 year depent on Tisseue characteristic (muscle healing relatively faster than bone healing or nervous System healing)

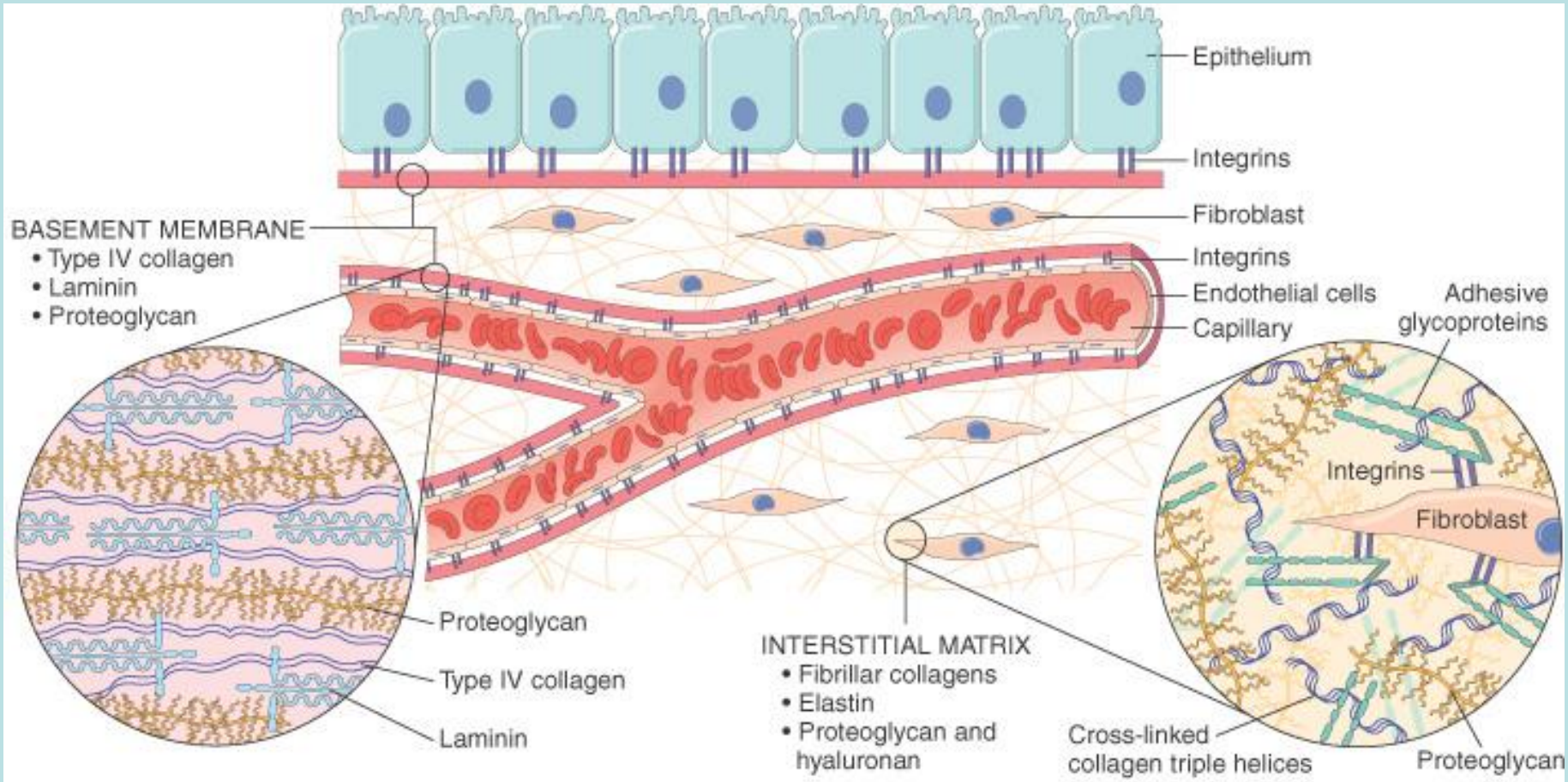


Fig. 4-19 Extracellular matrix (ECM). Major components of the ECM include collagens, proteoglycans, and adhesive glycoproteins. Both epithelial and mesenchymal cells (e.g., fibroblasts) interact with ECM via integrins. To simplify the diagram, many ECM components (e.g., elastin, fibrillin, hyaluronan, and syndecan) are not included.

(From Kumar V, Abbas A, Fausto N: Robbins & Cotran pathologic basis of disease, *ed 7, Philadelphia, 2005, Saunders.)*

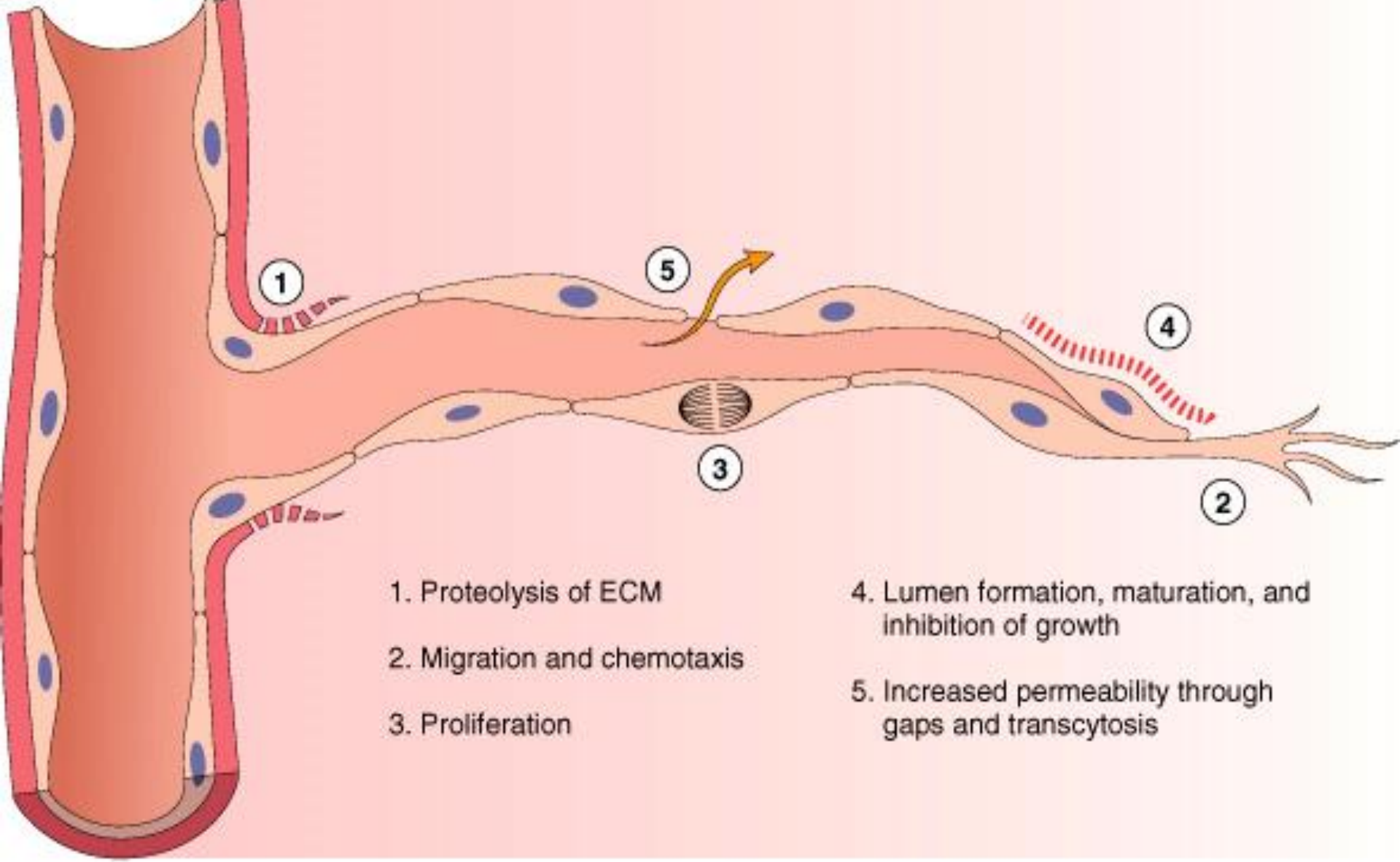


Fig. 4-30 Steps in the process of angiogenesis. *ECM,* Extracellular matrix.

(Modified from Motamed K, Sage EH: Kidney *Int 51:1383, 1997.)*

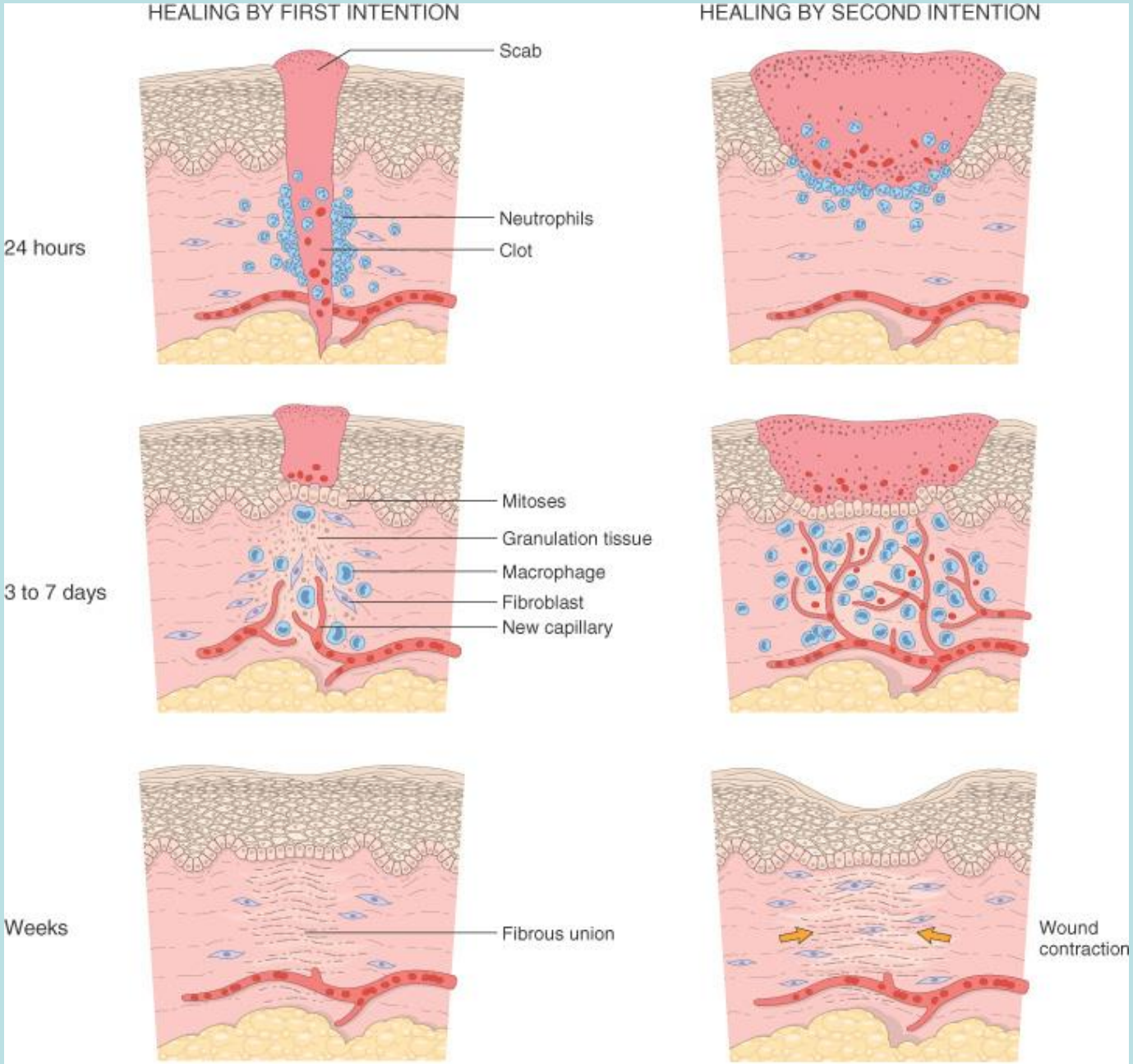


Fig. 4-20 Wound healing. Steps in wound healing by first intention (*left*) and second intention (*right*). Note large amounts of granulation tissue and wound contraction in healing by second intention.

(From Kumar V, Abbas A, Fausto N: Robbins & Cotran pathologic basis of disease, *ed 7, Philadelphia, 2005, Saunders.)*

THANKS