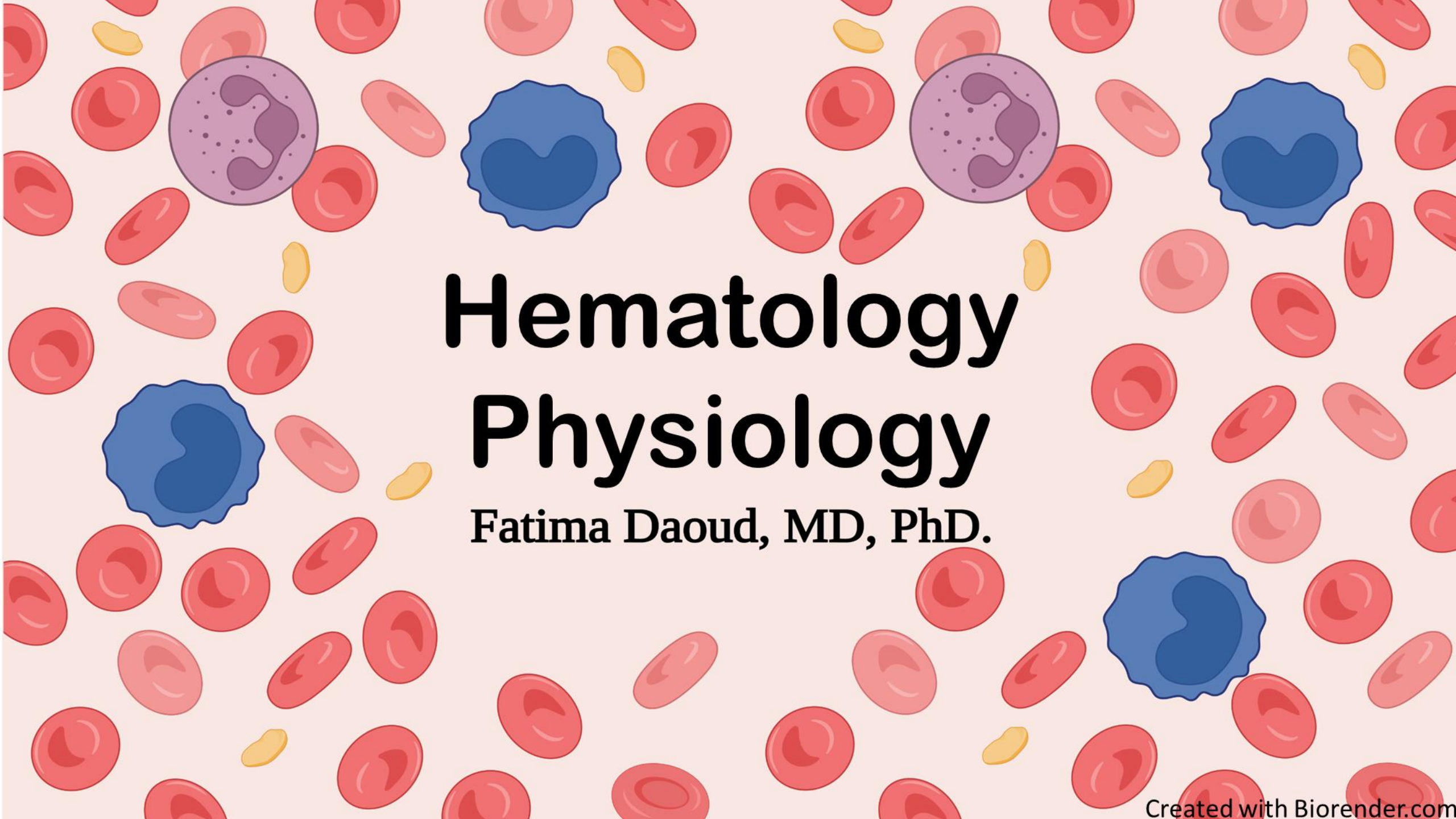
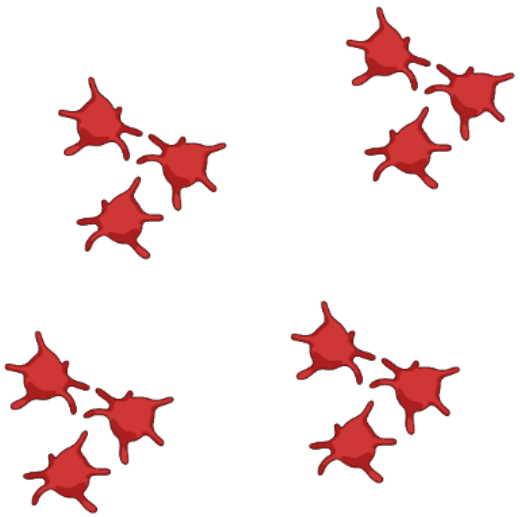


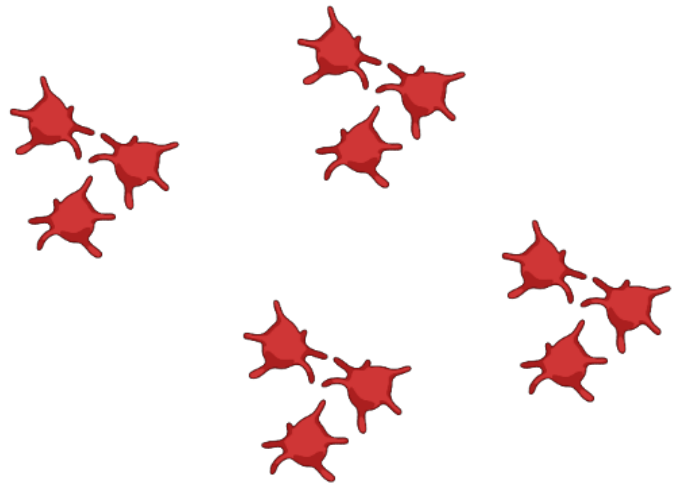
Hematology Physiology

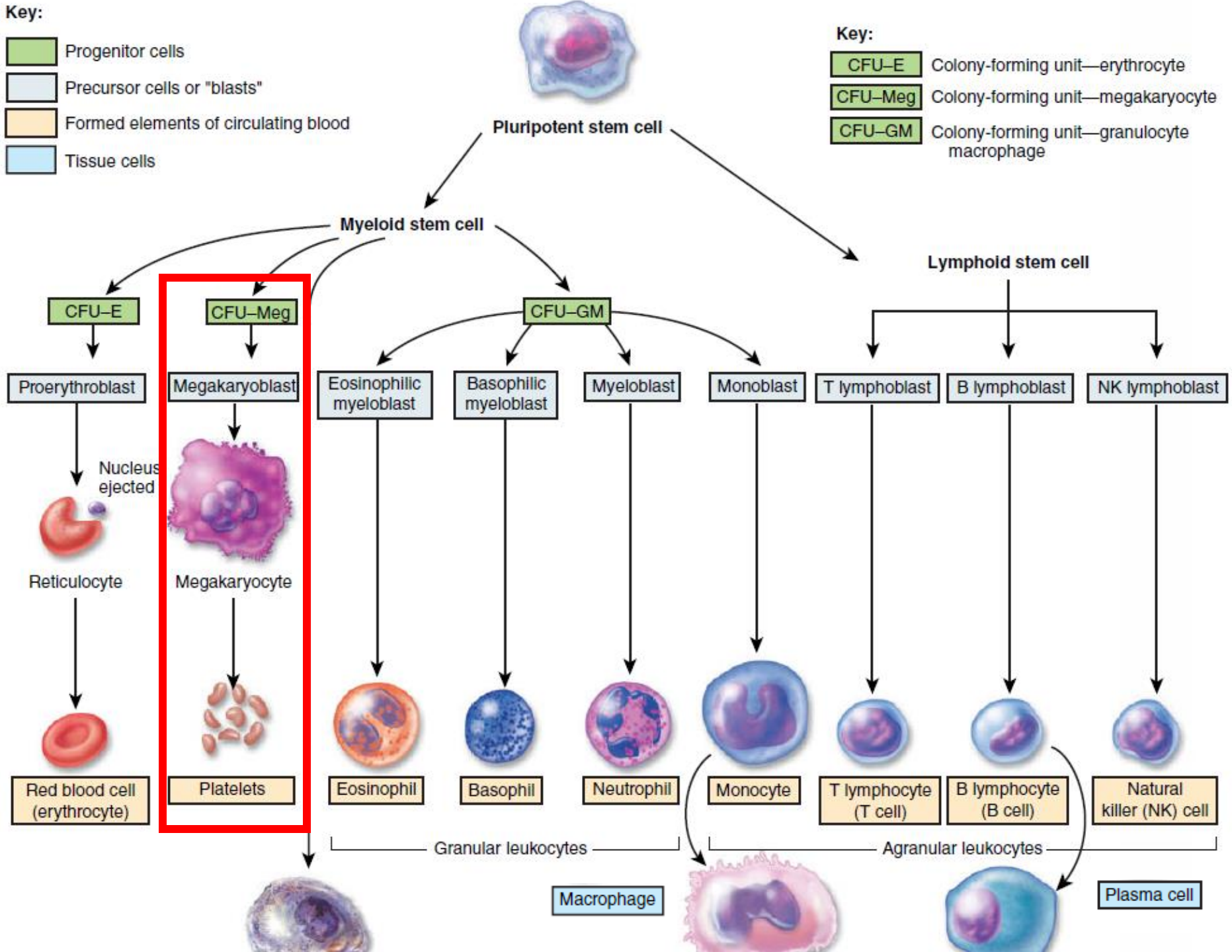
Fatima Daoud, MD, PhD.

Lecture 4 slides with DR NOTES
Lynn Alhamaideh



Platelets (Thrombocytes)




Key:
Progenitor cells
Precursor cells or "blasts"
Formed elements of circulating blood
Tissue cells
Key:
CFU–E Colony-forming unit—erythrocyte
CFU–Meg Colony-forming unit—megakaryocyte
CFU–GM Colony-forming unit—granulocyte macrophage
Pluripotent stem cell
Myeloid stem cell
Lymphoid stem cell
CFU–E
CFU–Meg
CFU–GM
Proerythroblast
Megakaryoblast
Eosinophilic myeloblast
Basophilic myeloblast
Myeloblast
Monoblast
T lymphoblast
B lymphoblast
NK lymphoblast
Nucleus ejected
Reticulocyte
Megakaryocyte
Red blood cell (erythrocyte)
Platelets
Eosinophil
Basophil
Neutrophil
Monocyte
T lymphocyte (T cell)
B lymphocyte (B cell)
Natural killer (NK) cell
Granular leukocytes
Agranular leukocytes
Macrophage
Plasma cell

Note slide 3 //Platelets are formed in the bone marrow
from megakaryocytes which are extremely large cells of the hematopoietic series in the marrow.

Platelets Overview

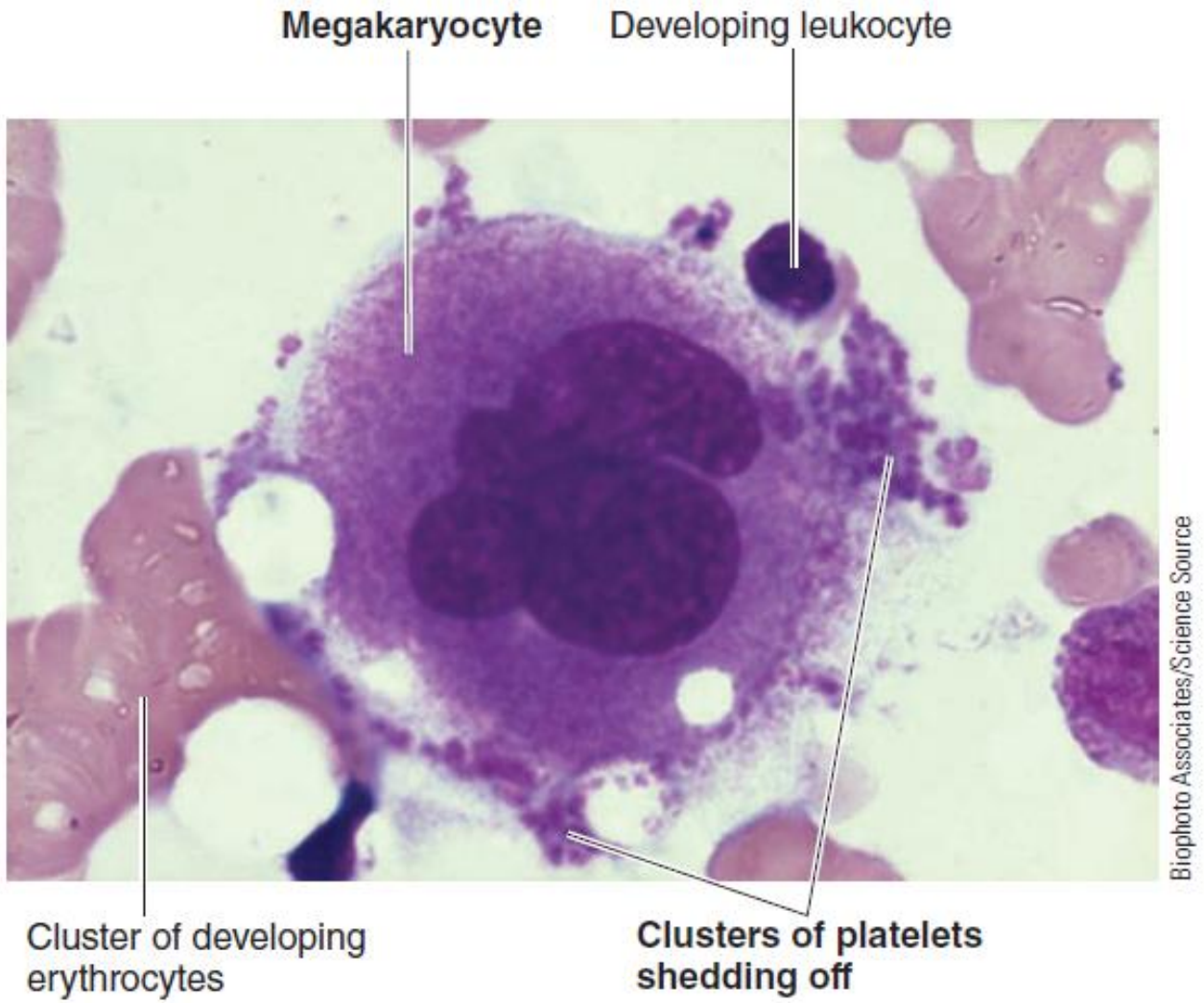
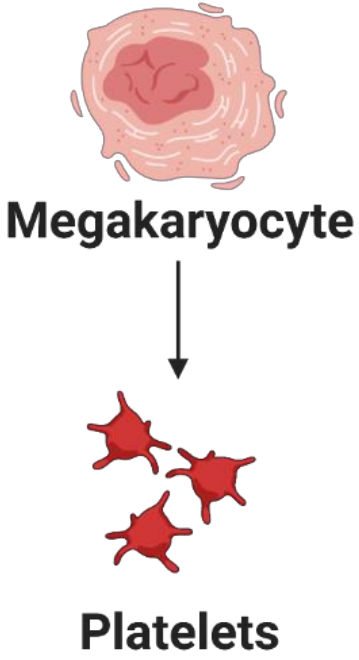


Figure 11-10 A megakaryocyte forming platelets.

Platelets Characteristics

- The normal concentration of platelets in the blood is between 150,000 and 450,000/ µL.
- Half-life in the blood of 8 to 12 days.
- Eliminated from the circulation mainly by the tissue macrophage (Spleen).

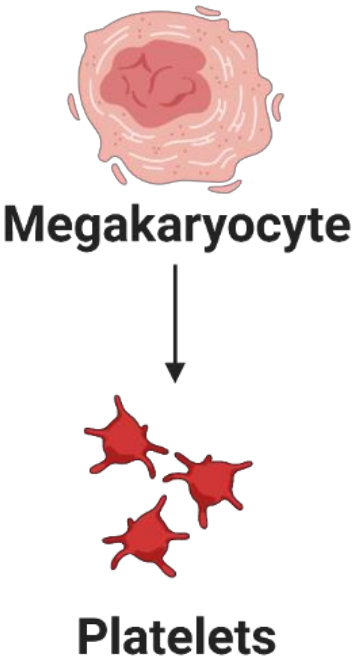


Platelets Cytoplasm

- They do not have nuclei.

- However they contain:

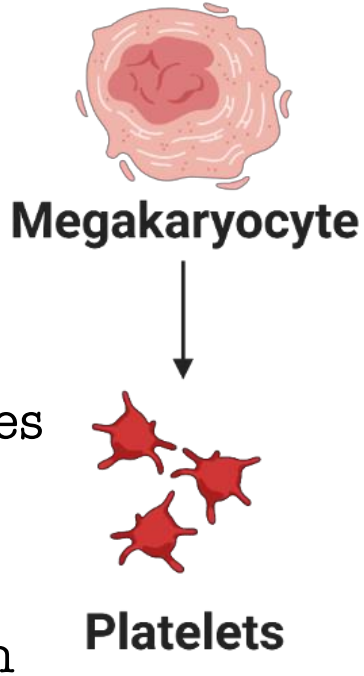
1. Contractile proteins: actin, myosin, and thrombosthenin.
2. Residuals of both the endoplasmic reticulum and the golgi apparatus.
3. Mitochondria (ATP).
4. Enzyme systems that synthesize prostaglandins.
5. Fibrin-stabilizing factor.
6. Vascular endothelial cells, vascular smooth muscle cells, and fibroblasts growth factors.



Platelets membrane

- There is a coat of glycoproteins that repulses adherence to normal endothelium and yet causes adherence to injured areas of the vessel wall.

- Contains large amounts of phospholipids that activate multiple stages in the blood-clotting process.



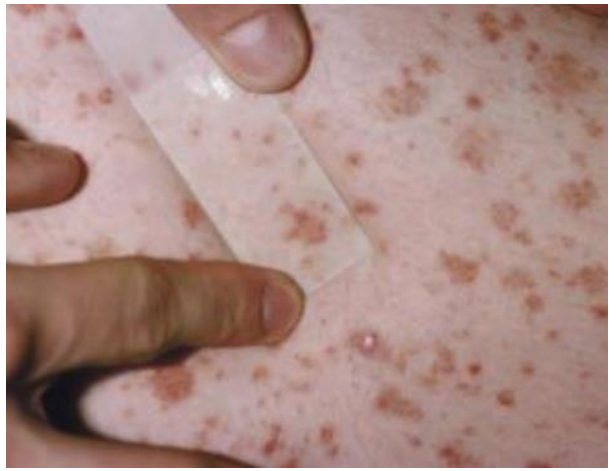
Note // * * If the cut in the blood vessel is very small—many very small vascular holes develop throughout the body each day—the cut is often sealed by a platelet plug rather than by a blood clot.

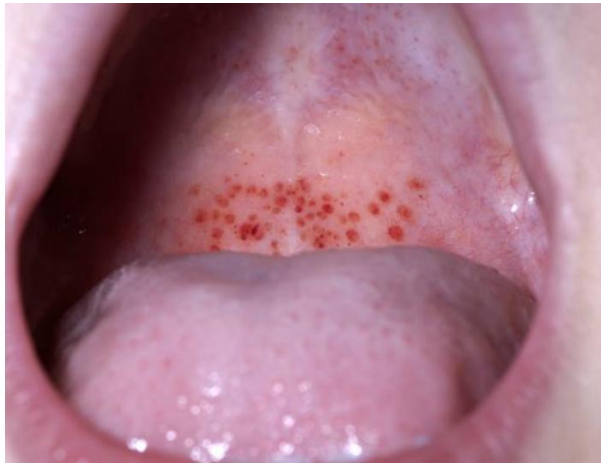
* * Literally thousands of small hemorrhagic areas develop each day under the skin (petechiae, which appear as purple or red dots on the skin) and throughout the internal tissues of a person who has few blood platelets. This phenomenon does not occur in persons with normal numbers of platelets.

Platelets Thrombocytopenia

- Cutaneous and mucosal bleeding
- Easily bruising
- Petechiae
- Increased bleeding time

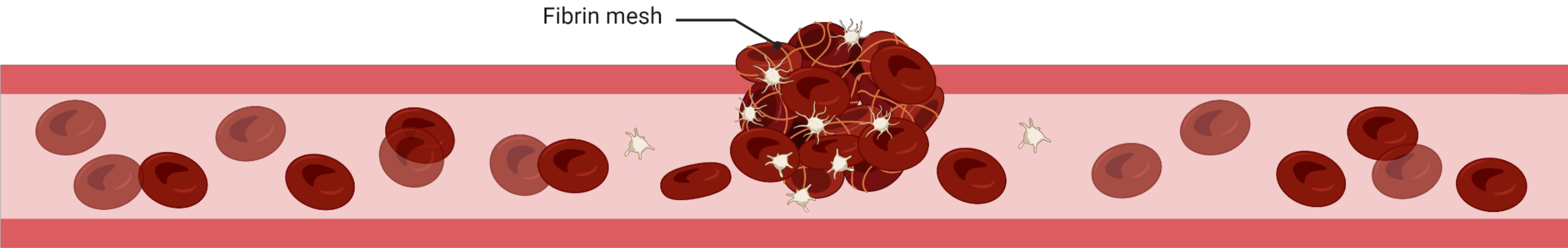
Thrombocytopenia



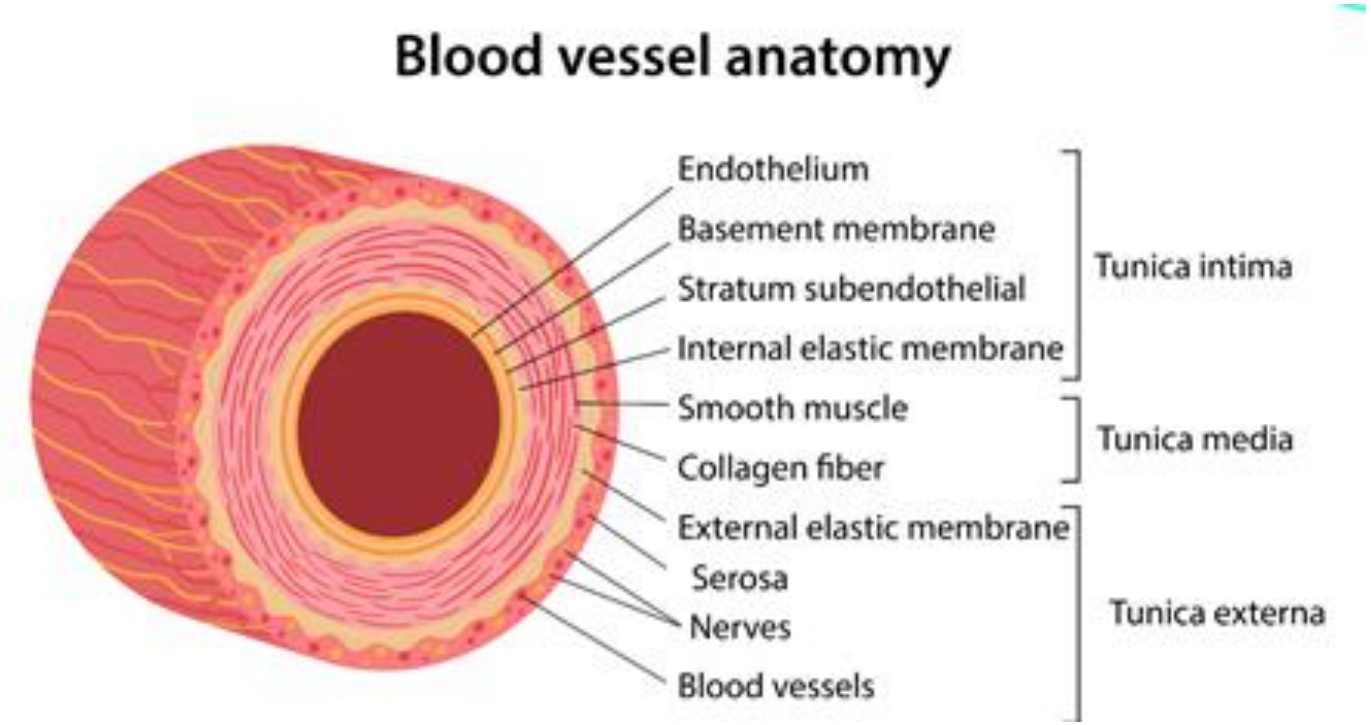


@MSD manual, pediatric UCI

Hemostasis



Hemostasis > Blood vessel anatomy

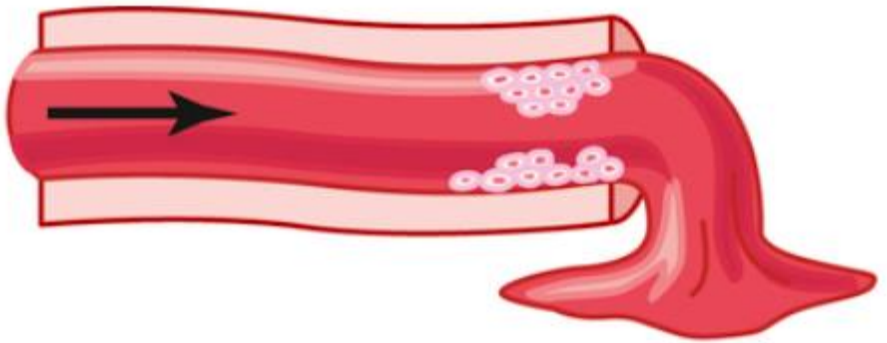


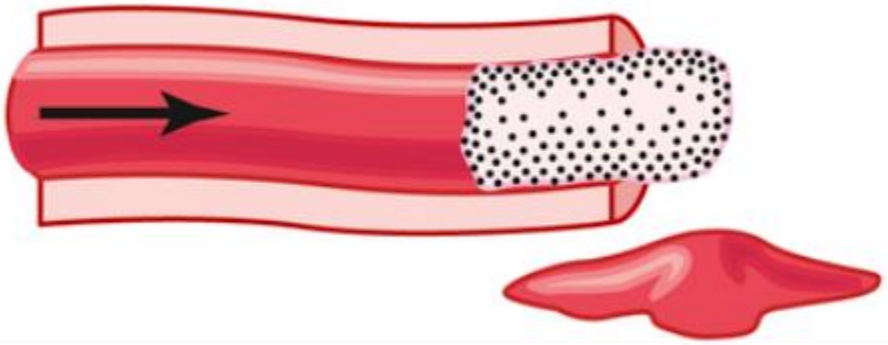
Hemostasis

Events of hemostasis

Hemostasis means prevention of blood loss and includes the following events:

(1) Vascular constriction.
(2) Formation of a platelet plug.
(3) Formation of a blood clot as a result of blood coagulation.
(4) Growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.





Note/** For bleeding to take place from a vessel, a break must be present in the vessel wall and the pressure inside must be greater than the pressure outside the vessel to force blood out through the defect.

** The small capillaries, arterioles, and venules are often ruptured by minor traumas of everyday life; such traumas are the most common source of bleeding, although we often are not even aware that any damage has taken place> The body's inherent hemostatic mechanisms normally are adequate to seal defects and stop blood loss through these small microcirculatory vessels.

** The rarer occurrence of bleeding from medium to large vessels usually cannot be stopped by hemostatic mechanisms alone. Bleeding from a severed artery is more profuse and therefore more dangerous than venous bleeding, because the outward driving pressure is greater in arteries (that is, arterial blood pressure is higher than venous pressure).

** First aid measures for a severed artery include applying external pressure to the wound that is greater than the arterial pressure to temporarily halt bleeding until the torn vessel can be surgically closed. Hemorrhage from a severed vein can often be stopped simply by elevating the bleeding body part to reduce gravity's effects on pressure in the vein. If the accompanying drop in venous pressure is not enough to stop bleeding, mild external compression is usually adequate.

Hemostasis 1. Vascular constriction

- Immediately → smooth muscle contraction.
- Reduces the flow of blood from the ruptured vessel.
- The contraction results from the following:

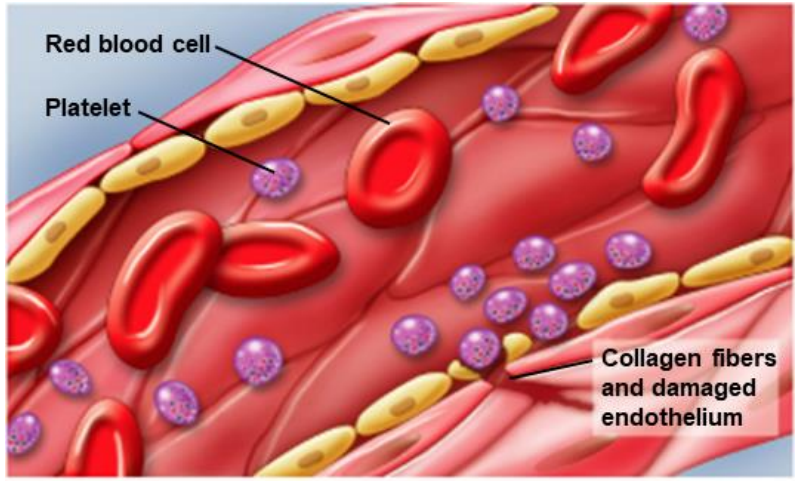
(1) local myogenic spasm

(2) local autacoid factors from the traumatized tissues, vascular endothelium, and blood platelets.

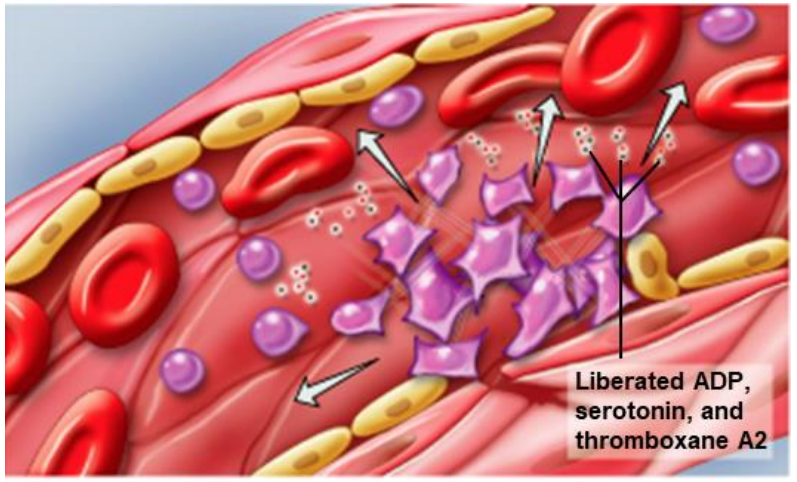
(3) nervous reflexes.

- The spasm can last for many minutes or even hours, during which time the processes of platelet plugging and blood coagulation can take place.

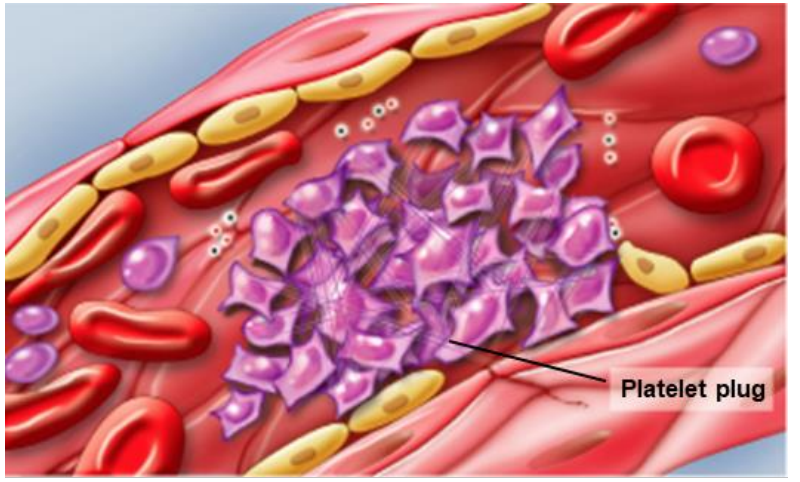
Hemostasis 2. Platelet plug formation



1 Platelet adhesion



2 Platelet release reaction



3 Platelet aggregation

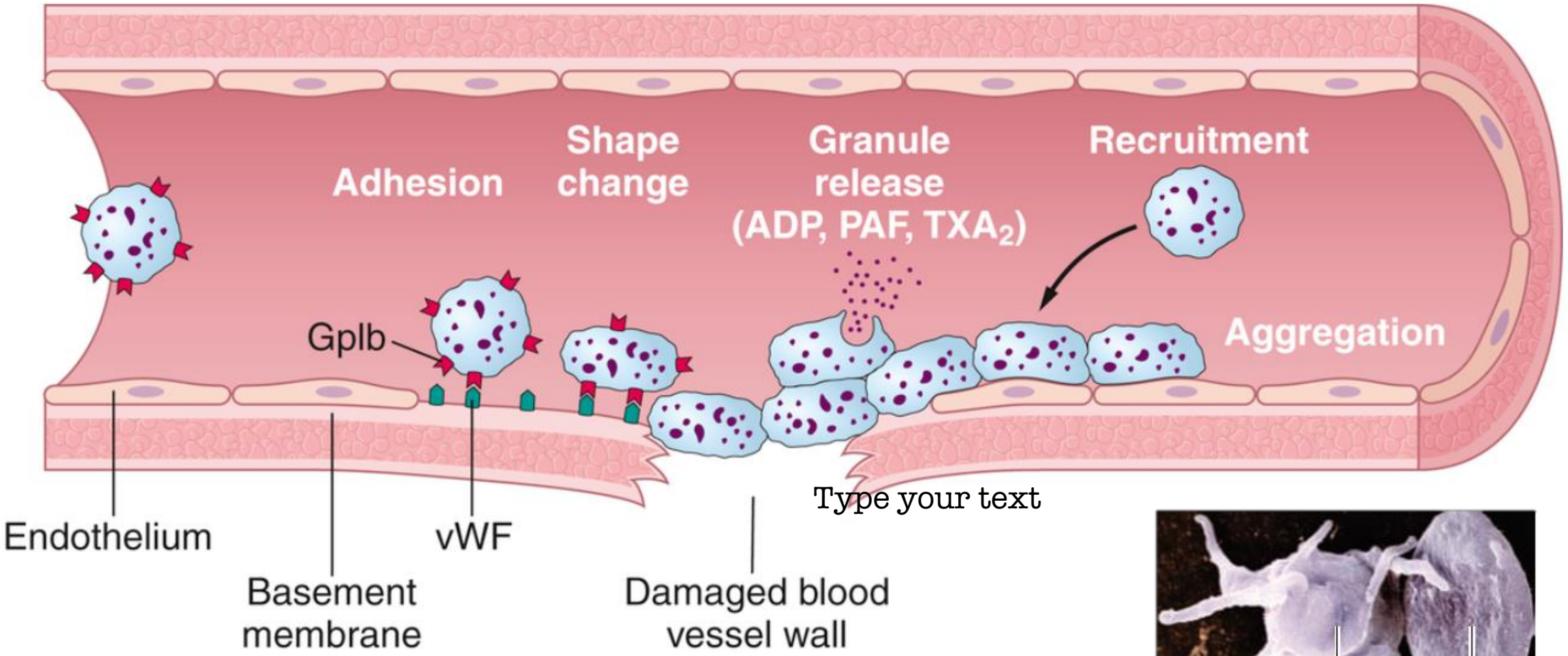
Note/●Adhesion – The deposition of platelets on the subendothelial matrix

●Secretion – The release of platelet granule proteins (Form numerous irradiating pseudopods) (They secrete large quantities of ADP and thromboxane A2 > to activate nearby platelets.)

●Aggregation – Platelet-platelet cohesion

Hemostasis

2. Platelet plug formation

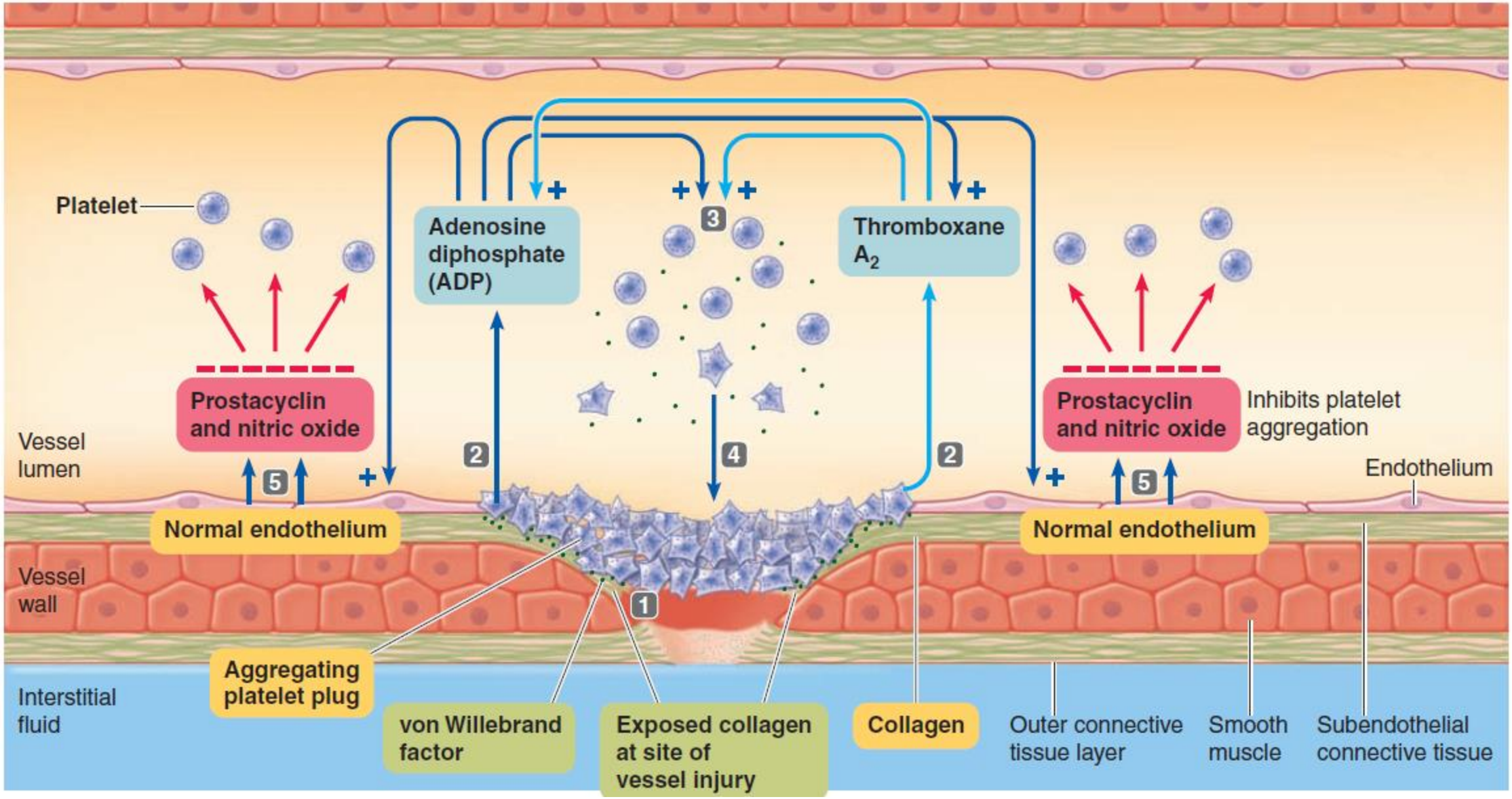


Type your text

Note /* * Platelets normally do not adhere to the smooth endothelial lining of blood vessels, but they do stick to damaged vessels.
* * When the endothelial lining is disrupted because of vessel injury, von Willebrand factor (vWF), a plasma protein secreted by megakaryocytes, platelets, and endothelial cells and always present in the plasma, adheres to the exposed collagen. Thus vWF serves as a bridge between platelets and the injured vessel wall

Hemostasis

Control of platelet plug



Hemostasis 2. Platelet plug formation

The aggregated platelet plug not only **physically seals** the break in the vessel but also performs three other important roles:

(1) The actin–myosin complex within the aggregated platelets **contracts** to **compact and strengthen** the plug.

(2) The platelet plug releases several powerful **vasoconstrictors** that induce **profound** constriction.

(3) The platelet plug releases other chemicals that **enhance blood coagulation.**

References

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