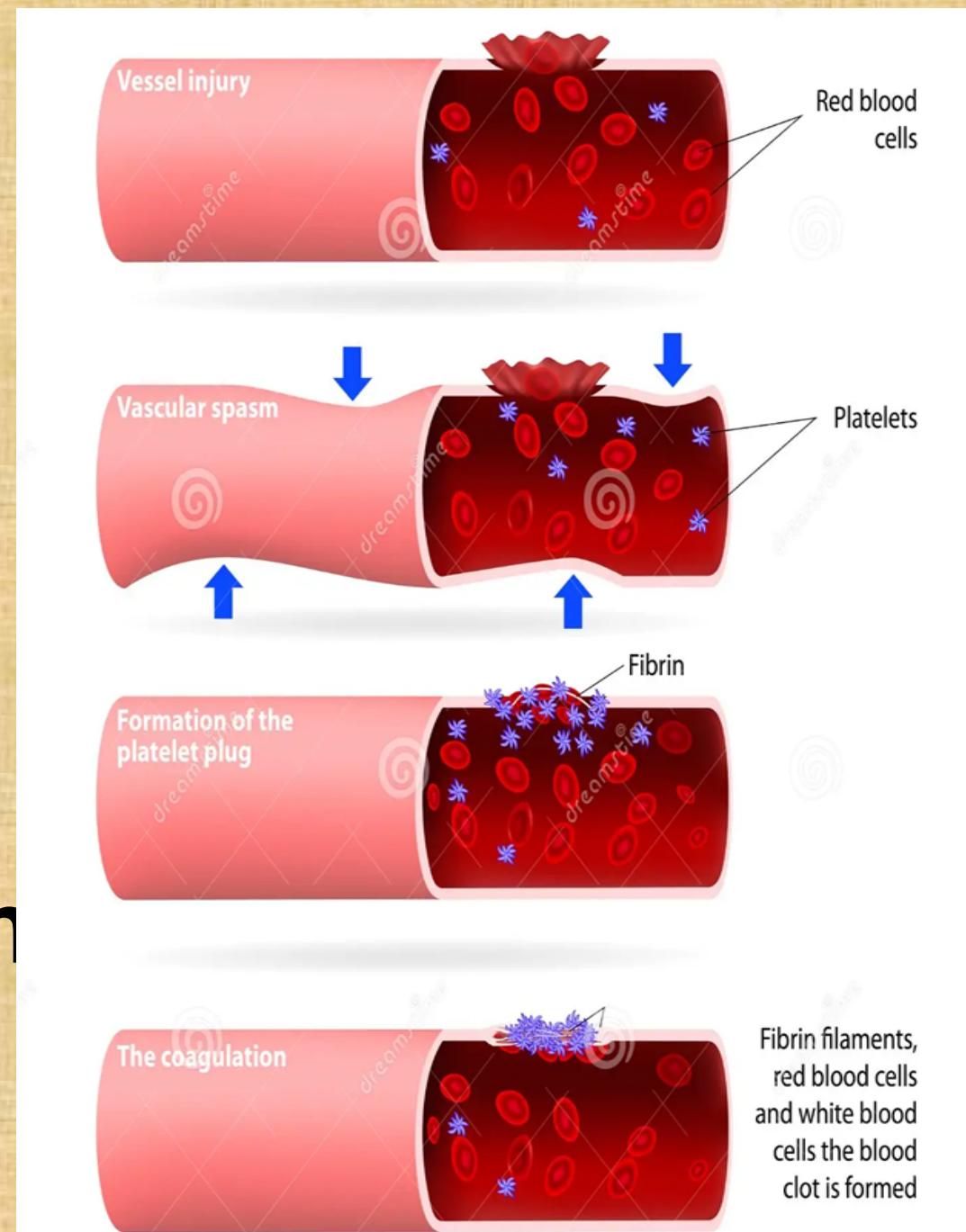


Hemostasis

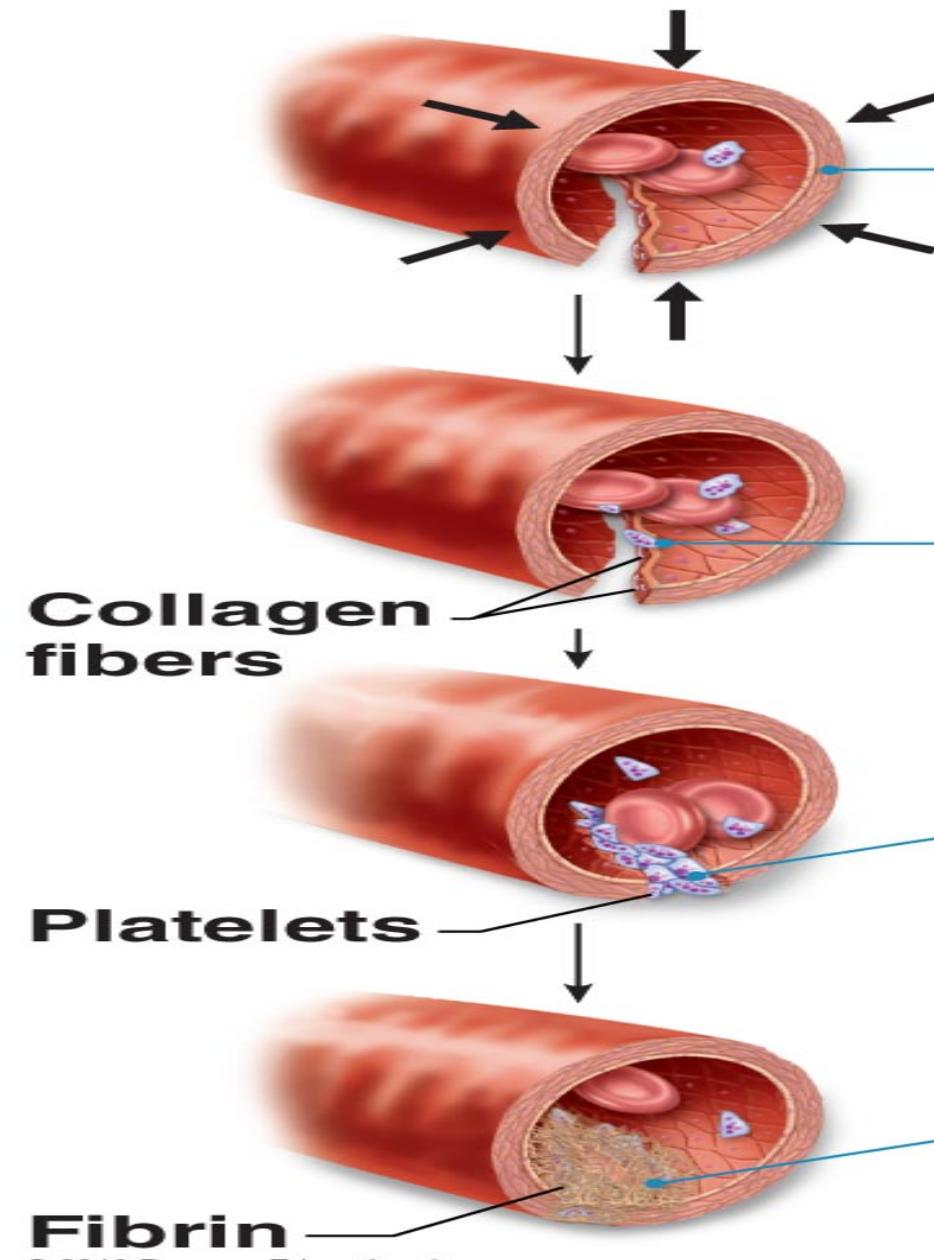
Stoppage of bleeding after injury

Hemostasis is done by:

- 1- Vascular spasm (vasoconstriction).
- 2- Platelet plug formation.
- 3- Blood clot formation



Hemostasis



Step ① Vascular spasm

- Smooth muscle contracts, causing vasoconstriction.

Step ② Platelet plug formation

- Injury to lining of vessel exposes collagen fibers; platelets adhere.

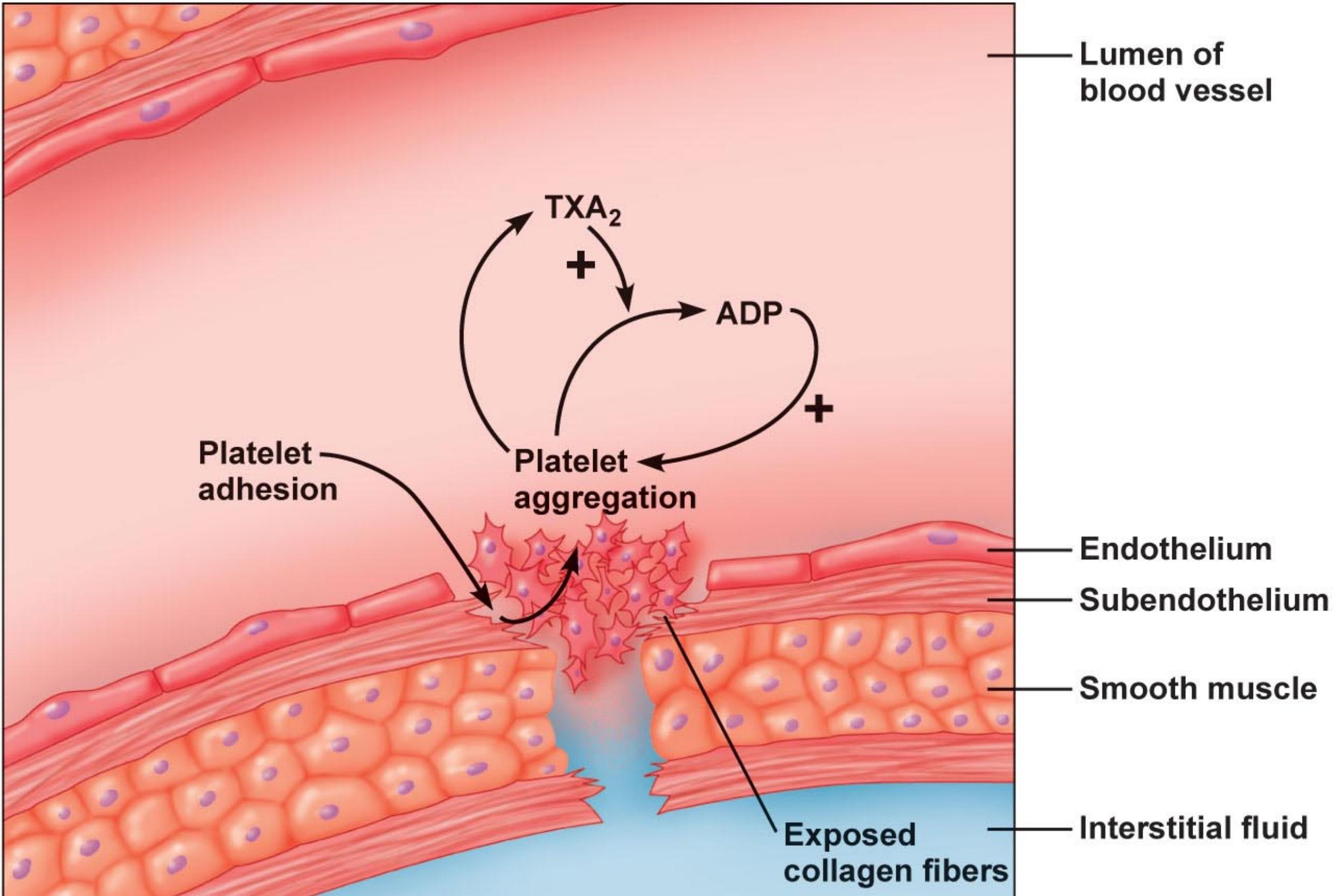
- Platelets release chemicals that make nearby platelets sticky; platelet plug forms.

Step ③ Coagulation

- Fibrin forms a mesh that traps red blood cells and platelets, forming the clot.

1- Vasoconstriction of the injured vessel:

- Nervous reflexes initiated by the pain of injury.
- Myogenic contraction of the injured vessel as a direct effect of trauma.
- Release of serotonin, ADP and thromboxane A₂ from platelets.



(a) Damaged blood vessel endothelium

Steps of platelet plug formation:

1- Platelet adhesion

As a result of injury, the sub endothelial collagen fibers are exposed and platelets adhere to collagen (**through glycoprotein receptors**).

Such adhesion is potentiated by von-Willebrand factor.

2- Platelet activation

Binding of platelets to collagen initiates **platelet activation**.

The activated platelets swell and discharge their contents of granules which include **ADP** which stimulate aggregation .



3- Platelet release

The contents of the **dense and alpha granules** are released.

These granules include **ADP, serotonin, Ca⁺⁺, coagulation factors, platelet derived growth factor and platelet activation factor.**

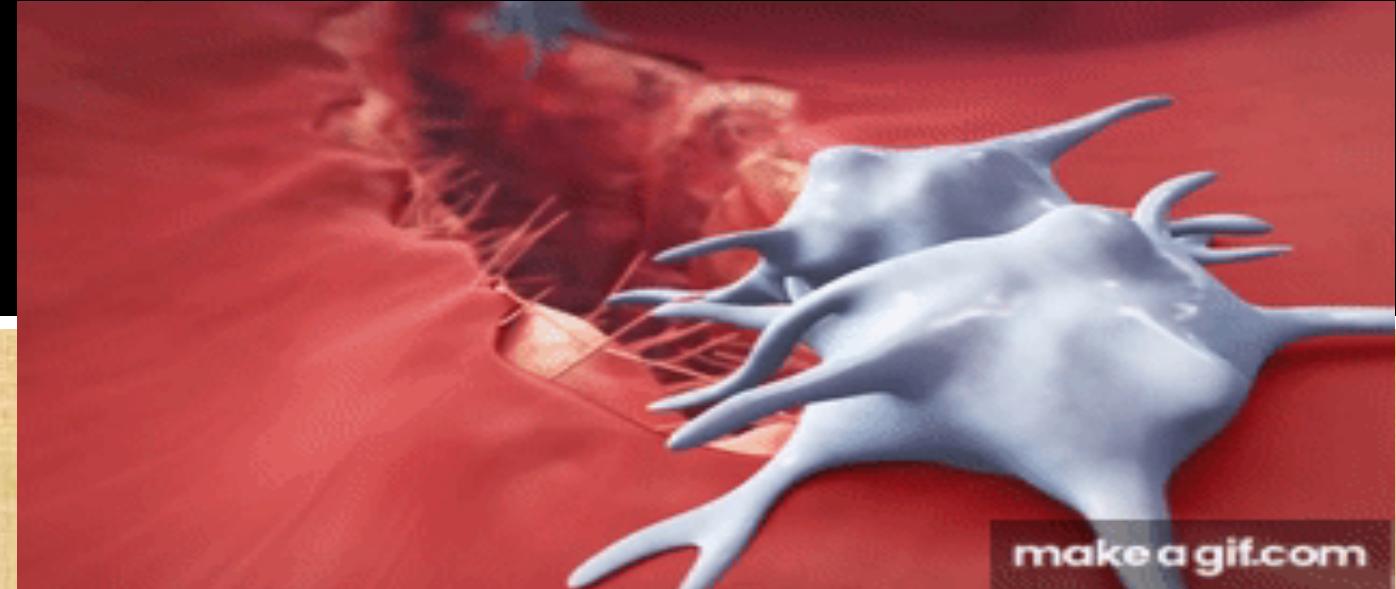
Serotonin which produce vasoconstriction

Thromboxane A2 is synthesized then released from activated platelets which produce vasoconstriction and aggregation.

4- Platelet aggregation

The released **ADP, thromboxane A₂**, causes more platelets to aggregate at the site of vascular injury. This will leads to release of **more ADP**, which in turn activates more and more platelets formation of loose platelet plug which can usually stop bleeding from small wounds.

5- Platelet fusion

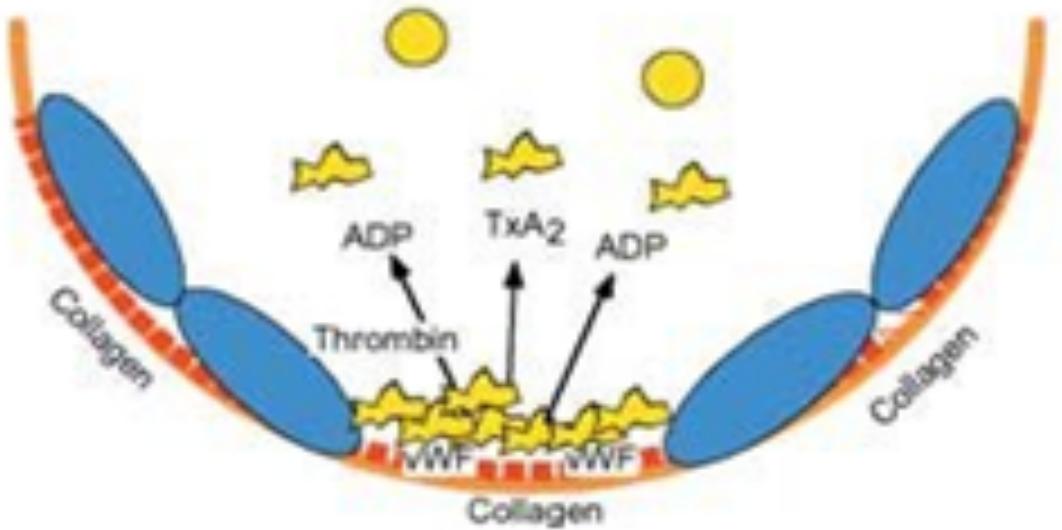
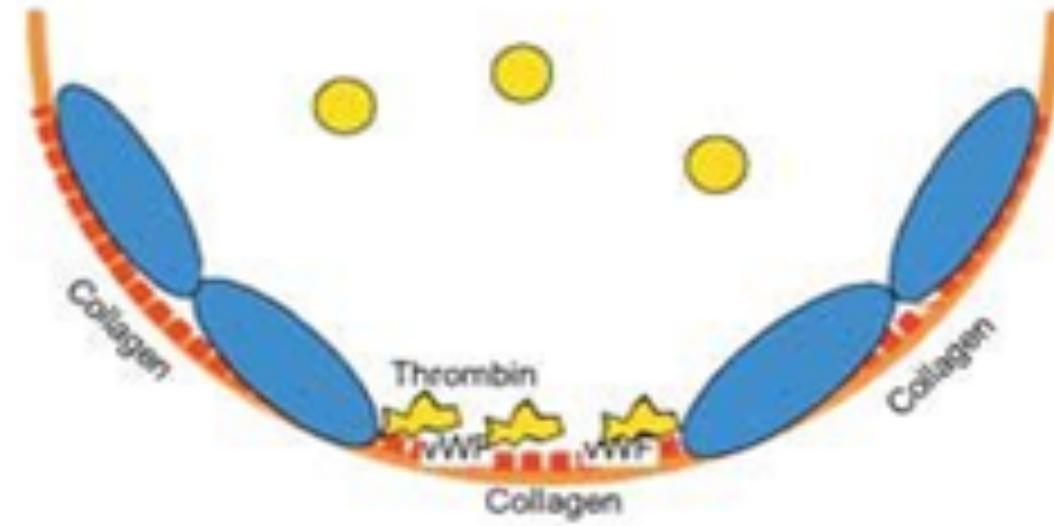
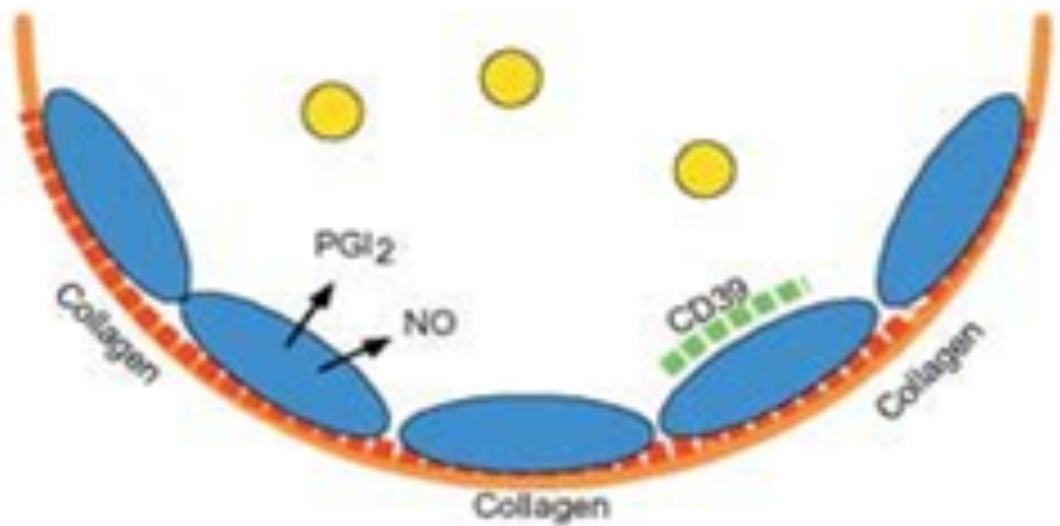


makeagif.com

Irreversible fusion of aggregated platelets at the site of injury is caused by:

- a- High concentration of ADP.**
- b- Thrombin.**
- c- Enzymes liberated during the platelets release reaction.**

Platelet Plug Formation



3-Formation of blood clot (coagulation):

- Blood clotting is a process by which the soluble fibrinogen in the plasma is polymerized into insoluble thread called fibrin. The formed clot is formed of fibrin threads entrapped blood cells and platelets.
- (formation of insoluble fibrin threads).
- Formation of blood clot needs **clotting factors** which are plasma proteins synthesized mostly by the liver except factor III (tissue thromboplastin), factor IV (Ca^{++}), Von-Willebrand factor (a protein synthesized by the platelets and vascular endothelium) and factor XIII (which synthesized in platelets).
- They are present in inactive forms (pro-enzymes) during rest, which could be activated during the process of blood coagulation.

Blood clotting mechanisms

- Clotting factors in the plasma are inactive.

Clotting mechanisms

- Start by activation of clotting factors.
- Ends by formation of fibrin clot.

Fibrin formation occur through

2 pathways

1] Extrinsic pathway

2] Intrinsic pathway

within 15 seconds, with tissue damage

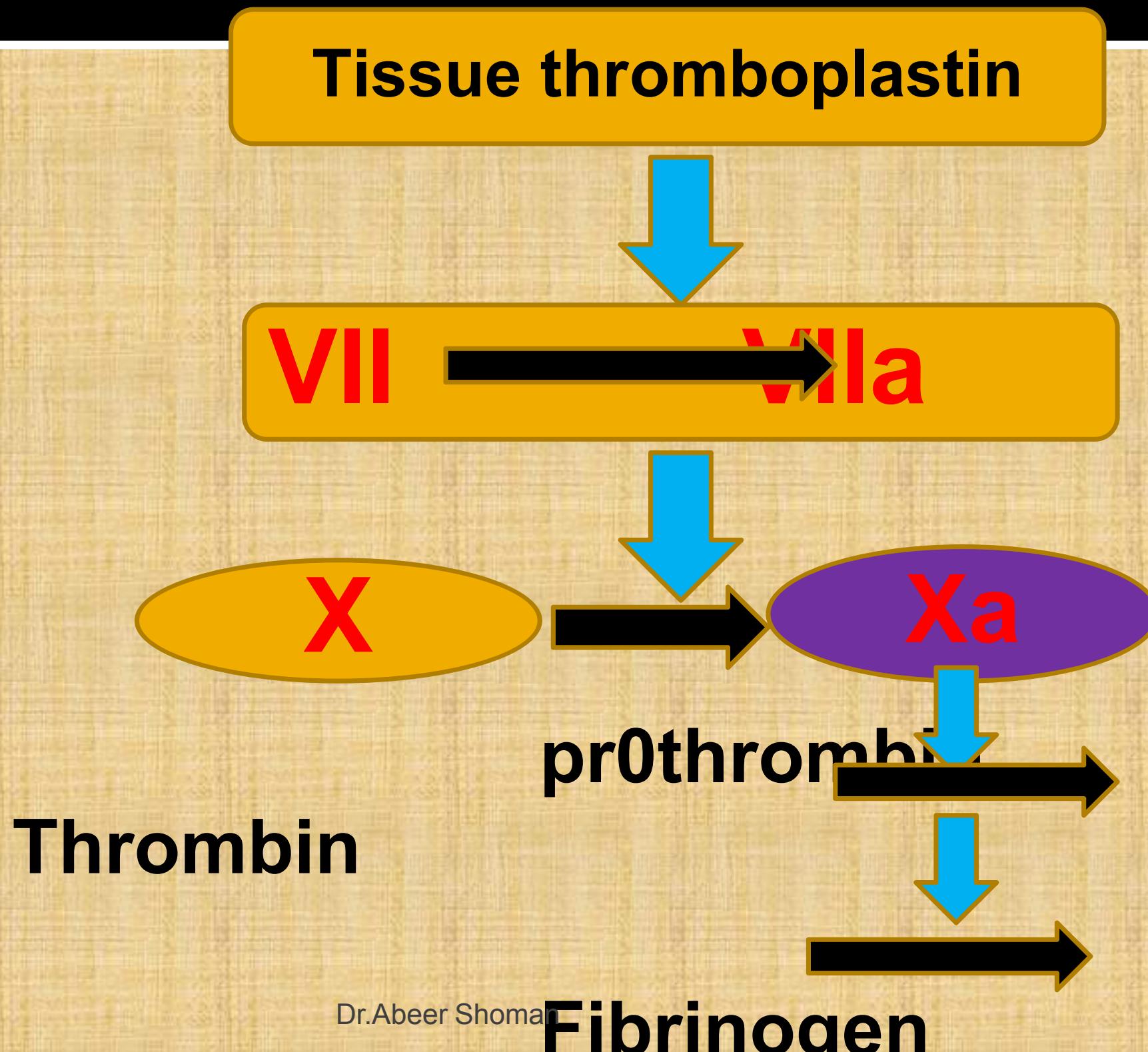
.

**within few minutes,
without tissue damage
ex in test tube**

The clotting factors are:

Name	Factor	Name	Factor
Fibrinogen	I	Antihemophilic A	VIII
Prothrombin	II	Antihemophilic B (Christmas factor)	IX
Thromboplastin	III	Stuart-Prower factor	X
Calcium	IV	Antihemophilic C (Plasma thromboplastin)	XI
Proaccelerin (labile factor)	V	Hagman factor (contact factor)	XII
Proconvertin(stable factor)	VII	Fibrin stabilizing factor	XIII

1- Extrinsic pathway



Extrinsic pathway

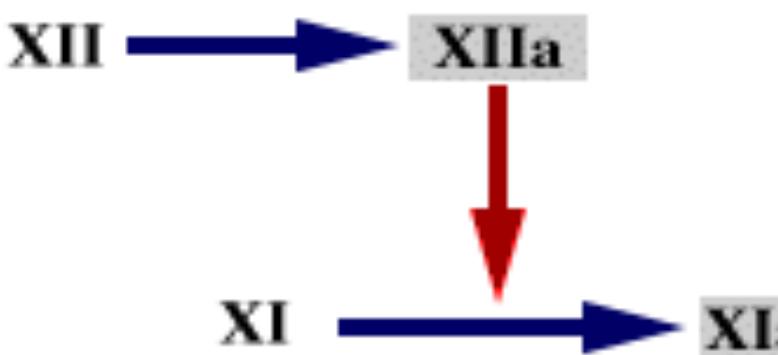
1. Extrinsic path way start by release of **thromboplastin** from injured tissue.
2. Thromboplastin activates **factor VII**.
3. Factor VII active activate **factor X**.
4. Factor X active convert prothrombin to **thrombin** in presence of **Ca**, platelets and **factor V**.
5. Thrombin converts **fibrinogen** to **fibrin monomer**.
6. Fibrin monomer converted to **fibrin clot** by **active factor XIII and Ca**.

Intrinsic pathway

1. **Collagen fiber (in vivo)** or negative charge wet surface(**invitro**)activate **factor XII** this activation is helped by high molecular weight kininogen and plasma kallikrein.
2. Active factor XII activate **factor XI**.
3. Active factor XI activate **factor IX**.
4. Active **IX**, **active VIII**, Ca and platelets form complex activate **factor X**.
5. Factor X active convert prothrombin to **thrombin** in presence of Ca, platelet and factor V.
6. Thrombin converts **fibrinogen** to **fibrin monomer**.
7. Fibrin monomer converted to **fibrin clot** by **active factor XIII and Ca**.

Coagulation Cascade and the use of Heparin

Intrinsic System



XIII

Extrinsic System

VII

Thrombin

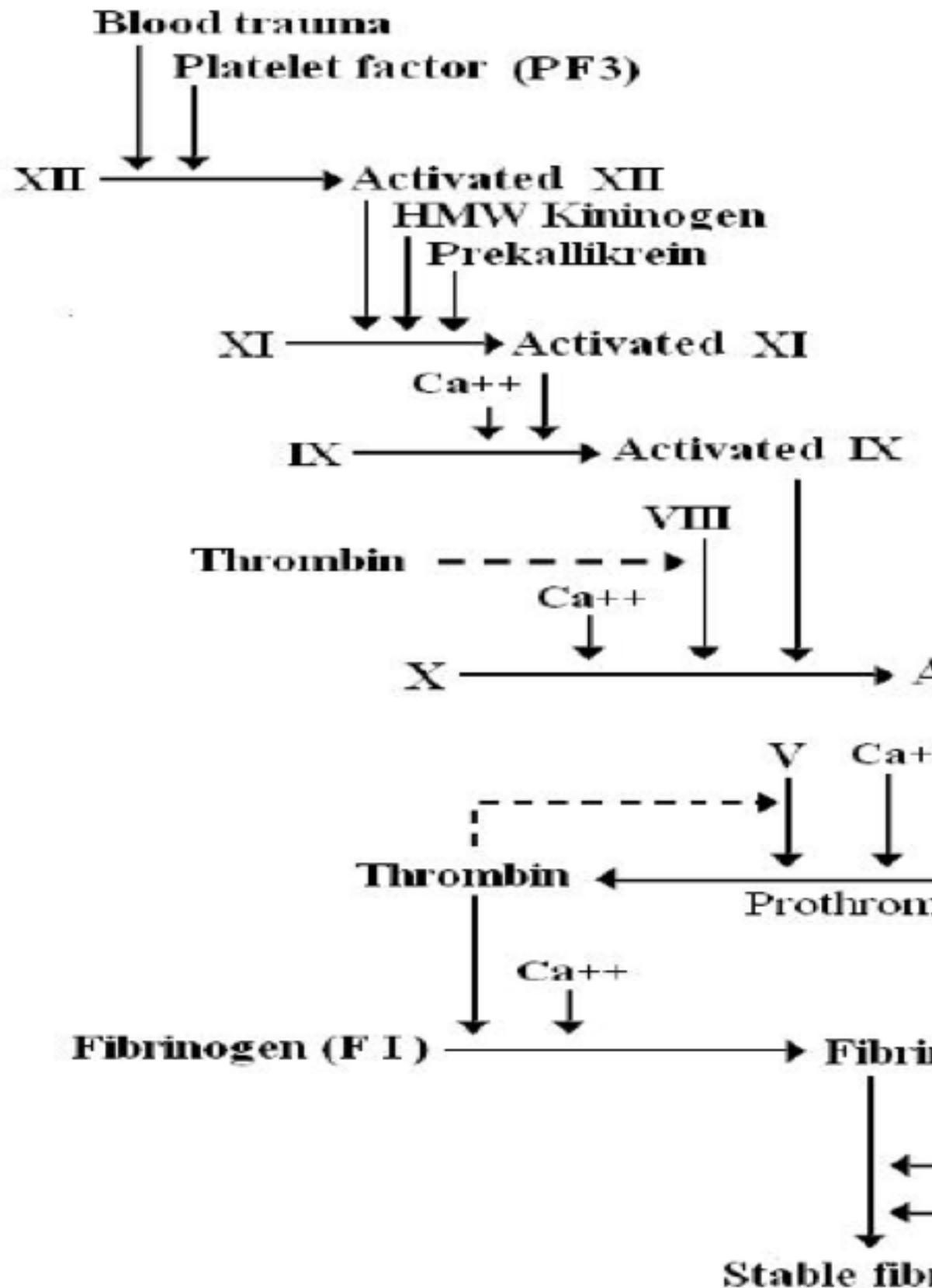
Fibrinogen

Fibrin

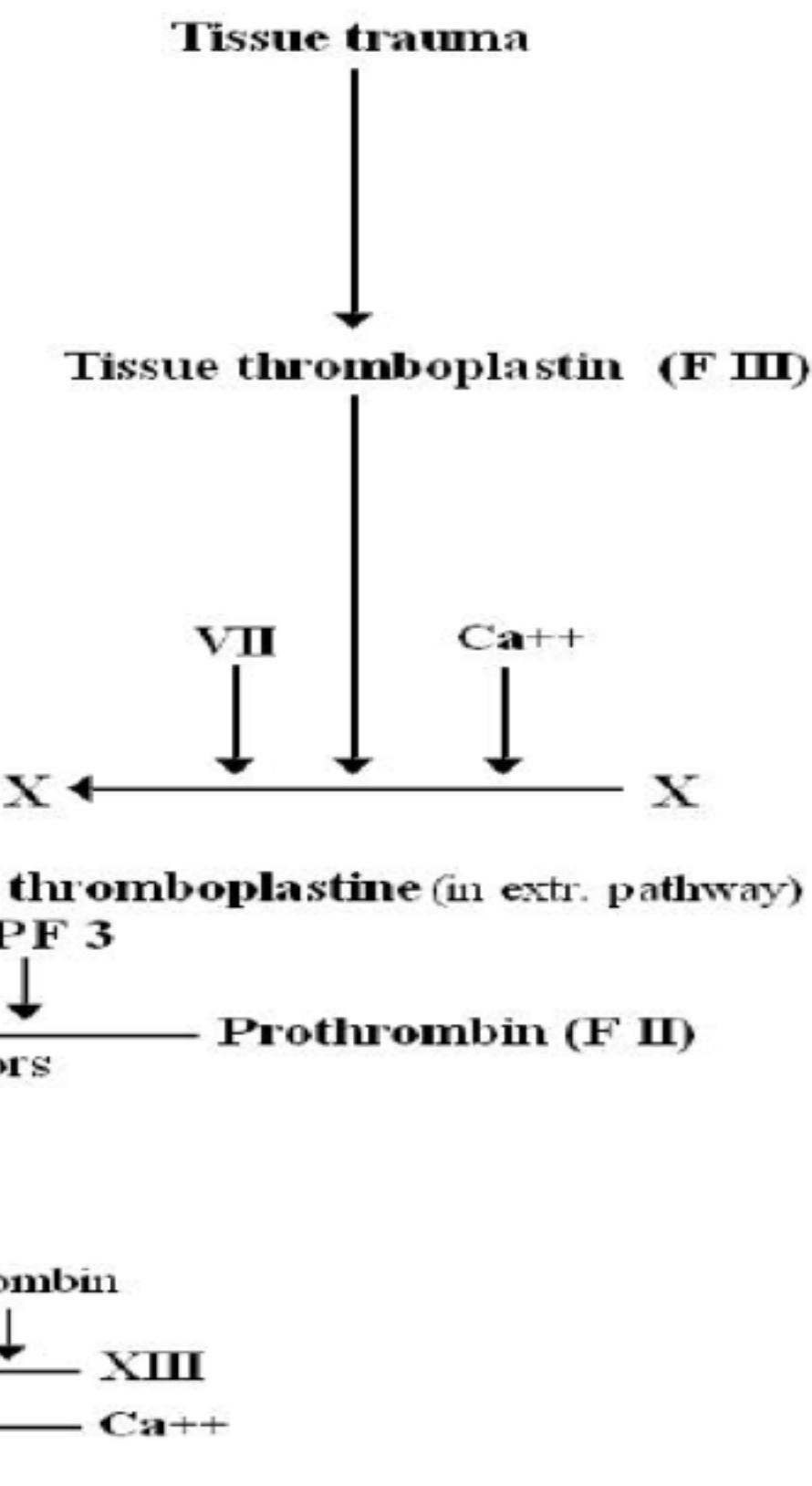
Stable
fibrin clot

- inactivated by heparin

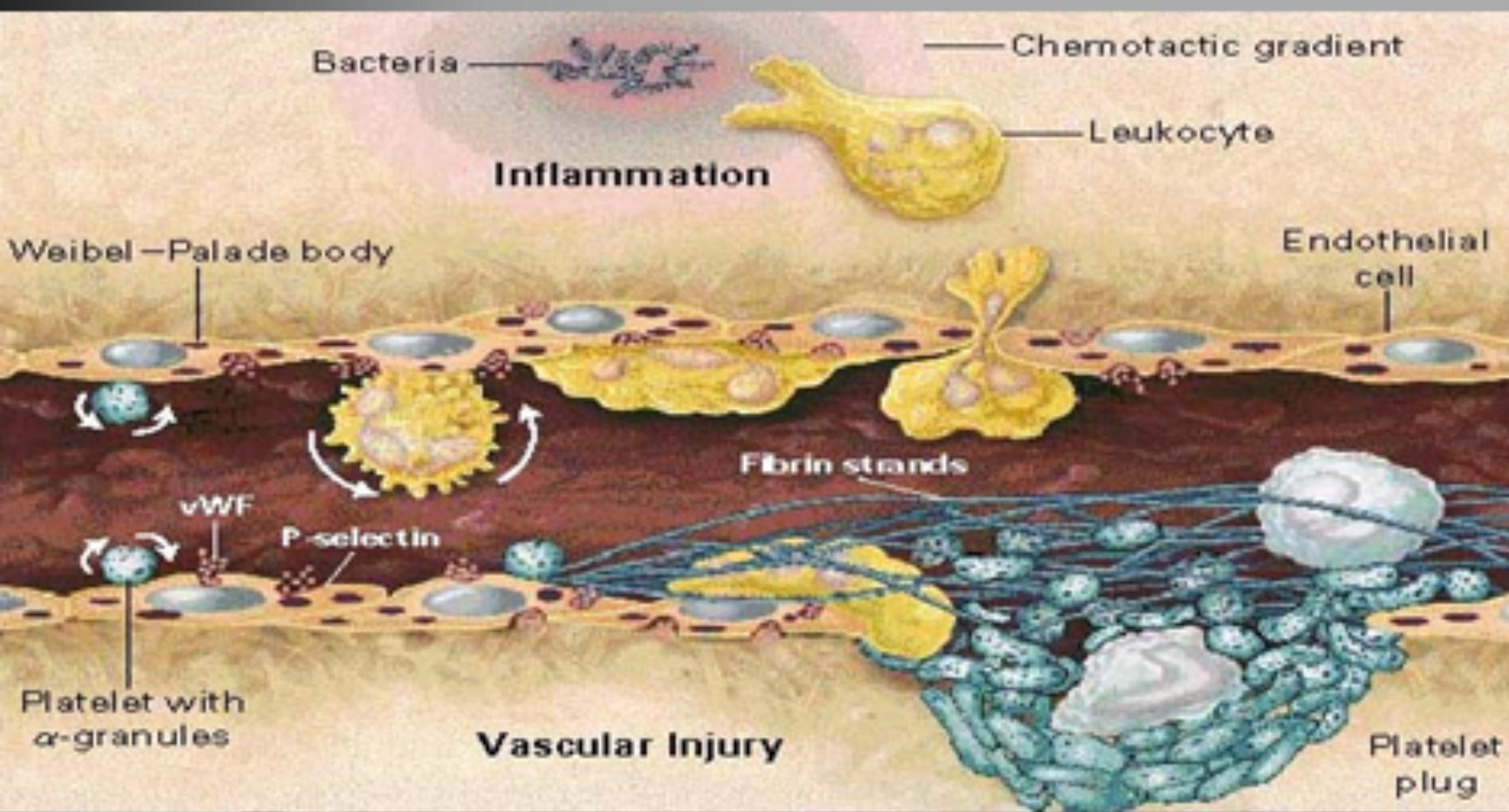
Intrinsic (platelet) pathway



Extrinsic (tissue) pathway



Blood clot



Important notes:

Important notes:

- 1- When blood vessel ruptures, blood clotting is initiated by both systems simultaneously.
- 2- The extrinsic system is very rapid (15 sec) & very extensive, while the intrinsic is slower (1-6 min).
- 3- Ca^{++} is required for promotion of all steps except the first 2 steps of the intrinsic way. Ca^{++} level rarely falls to level that affect clotting.
- 4- There is a link between intrinsic & extrinsic pathway: activated factor VII(extrinsic) & factor IX (intrinsic) activates factor X (common pathway).
- 5- Vitamin K is important for activation of factors II, VII, IX and X.

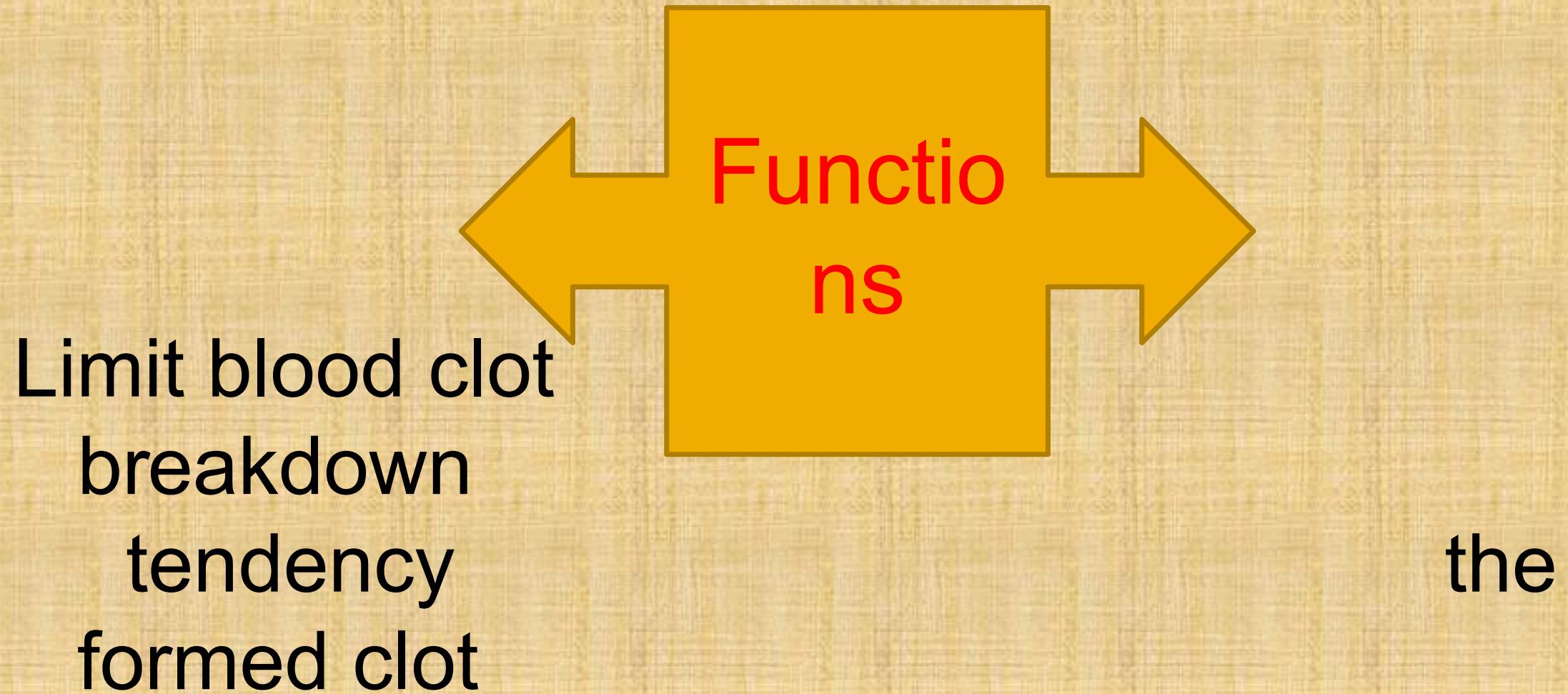
Role of Ca⁺⁺ in blood clotting

- Ca⁺⁺ is an essential catalyst in all reactions of blood clotting except the first 2 steps.
- In vivo reduction of blood Ca⁺⁺ to levels that stop blood clotting is incompatible with life because clotting stops only when Ca⁺⁺ level is severely decreased (to about 4 mg %) and such level cannot be reached clinically since death would occur when the Ca⁺⁺ level drops below 7 mg% due to tetany.

:Serum

- It is the **squeezed plasma** remaining after clot retraction.
- It is plasma **minus fibrin, factor V and VIII.**
- It contains **excess serotonin** due to breakdown of platelets.

Anti-clotting mechanisms



Anti-clotting mechanisms

GENERAL

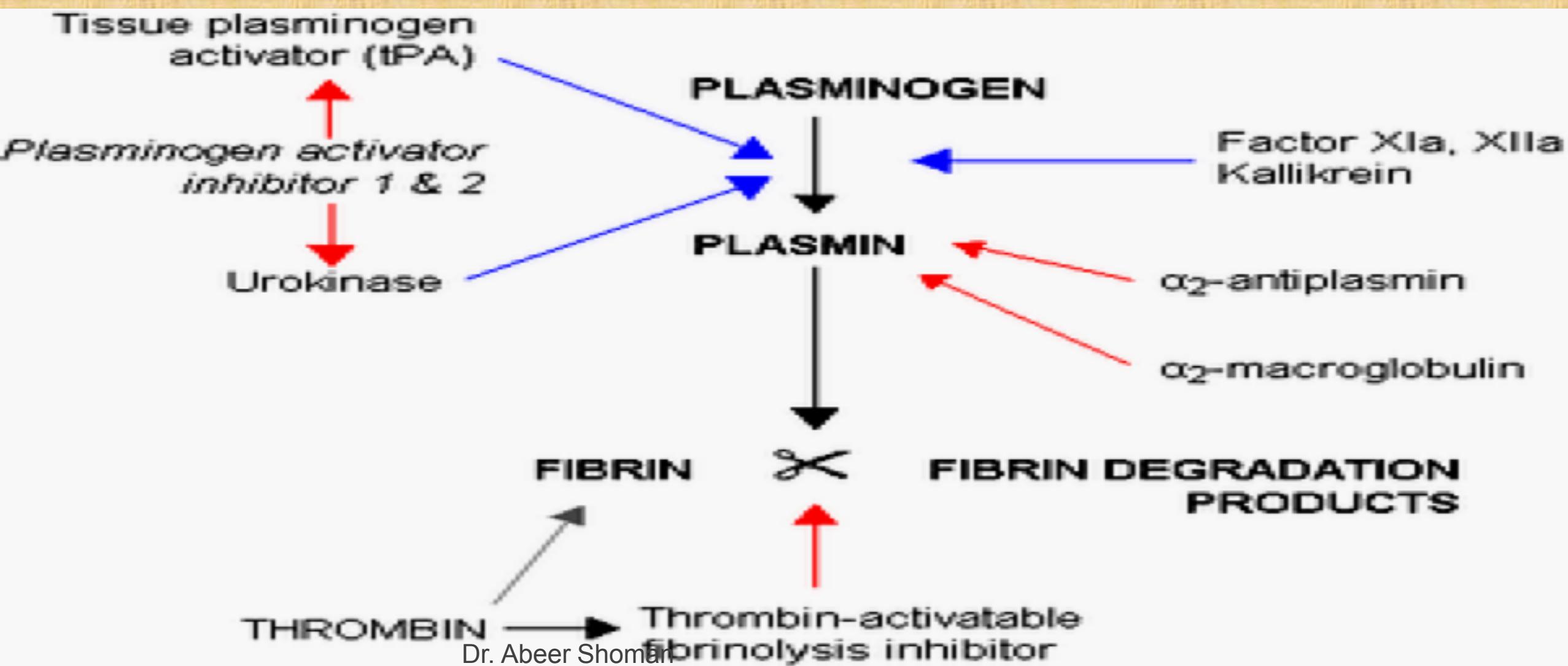
- 1-Smooth endothelium
- 2-Liver inactivates C.F

SPECIFIC

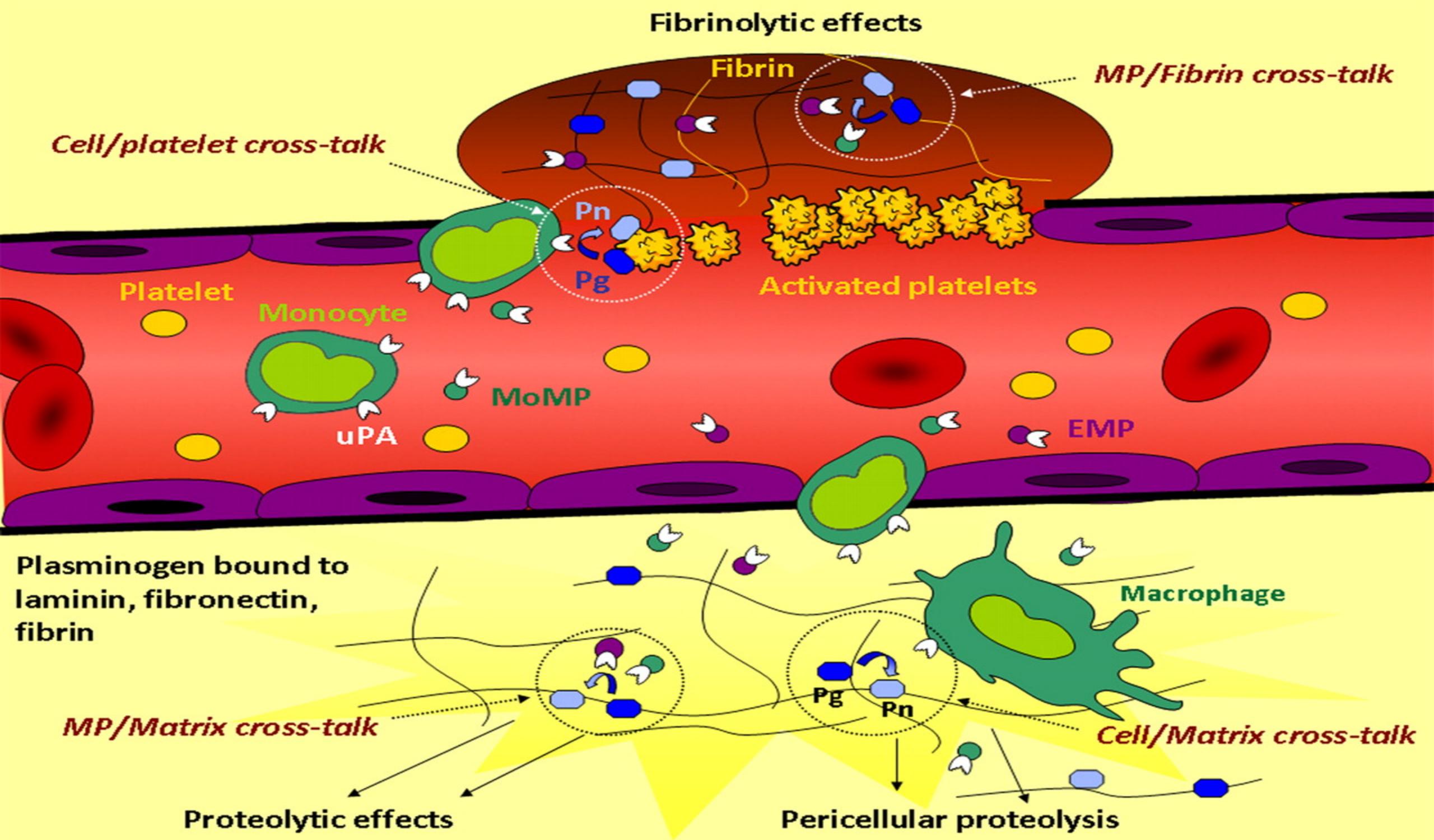
- 1-Antithrombin III
(IX, X, XI, XII)
- 2-prostacyclins
- 3-fibrinolytic system
- 4-protein C, protein S (V, VIII)
Increase plasmin

Fibrinolysis: breakdown and removal of blood clot

Is formed from its **inactive precursor plasminogen** by the action of **tissue plasminogen activator (TPA)**. Plasmin lyses fibrin and fibrinogen into **fibrinogen degradation products (FDP)** which inhibit thrombin



Fibrinolysis



Endothelial cell

Thrombomodulin

Thrombin

Protein C

Activated protein C (APC)

+ Protein S

VIIIa → **Inactive VIIIa**

Va → **Inactive Va**

**Inactivates inhibitor of
tissue plasminogen activator (t-PA)**

Plasminogen → **Plasmin**

**Thrombin
t-PA, u-PA**

Lyses fibrin

Anti-coagulants

IN VITRO

IN VIVO

- 1] Oxalate
- Heparin
- 2] Citrate
- Dicumarol
- 3] Silicon
- 4] Heparin

- 1]
- 2]

Heparin	Dicumarol	
Basophil, mast, liver injection	plant	1-Source
Rapid, short	oral	2-Intake
Sulphated mucopolysaccharide	Slow, long	3-Oncet, duration
In vivo ,vitro	As vitamin K	4- Chemistry
1] Antithrombin III 2] prevent factor IX 3]clearing action→it increases lipase enzyme which clears blood from lipids after meals.	In Vivo >Inhibit vit K >decrease formation of (II,VII,IX, X)	5-Site of action 6-Actions
Protamine sulphate	Vit. K	7-Antidote

Abnormalities of Hemostasis

Vit.K
deficiency

Hemophilia

Purpura

Thrombo-
embolism

D.I.C.

Abnormalities of Hemostasis

Vitamin K
deficiency



Vitamin K deficiency

Vitamin K is important for synthesis of factors II, VII, IX & X by liver

- It is synthesized by bacterial flora of intestine (it is advisable to delay circumcision one month after birth).
- Its deficiency leads to deficiency of factors II, VII, IX & X.

-Causes of its deficiency:

a- Sterility of intestine as in:

- 1- Newly born infants.
- 2- Long treatment with antibiotics

b- Decrease absorption as in:

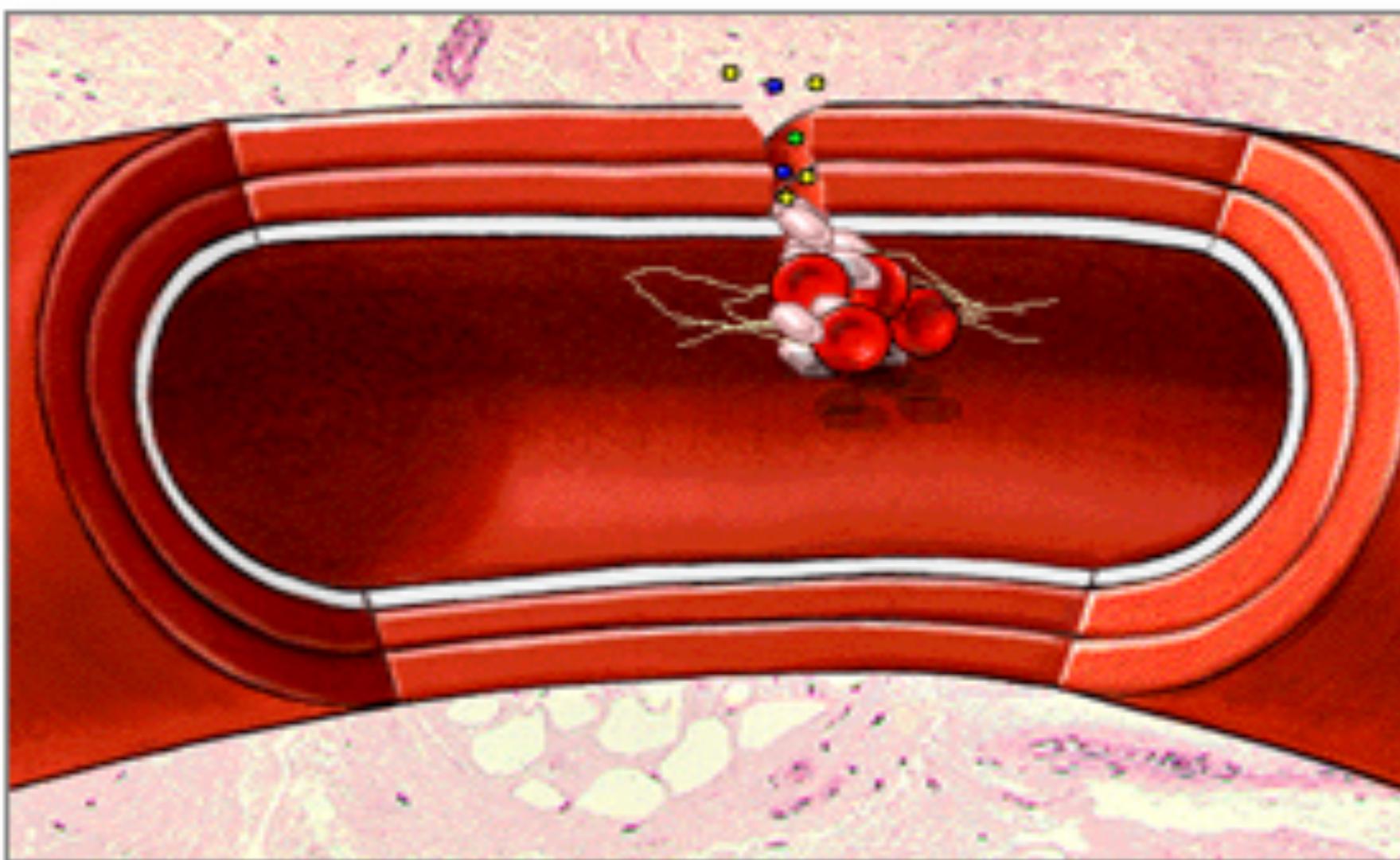
- 1- Obstructive jaundice
- 2- Fat malabsorption because vit K is fat soluble vitamin

a- Liver diseases.

d- Anticoagulants: which act by competitive inhibition with vit. K

Vitamin K

Vitamin K benefits blood clotting



Recommended daily allowance for adults:

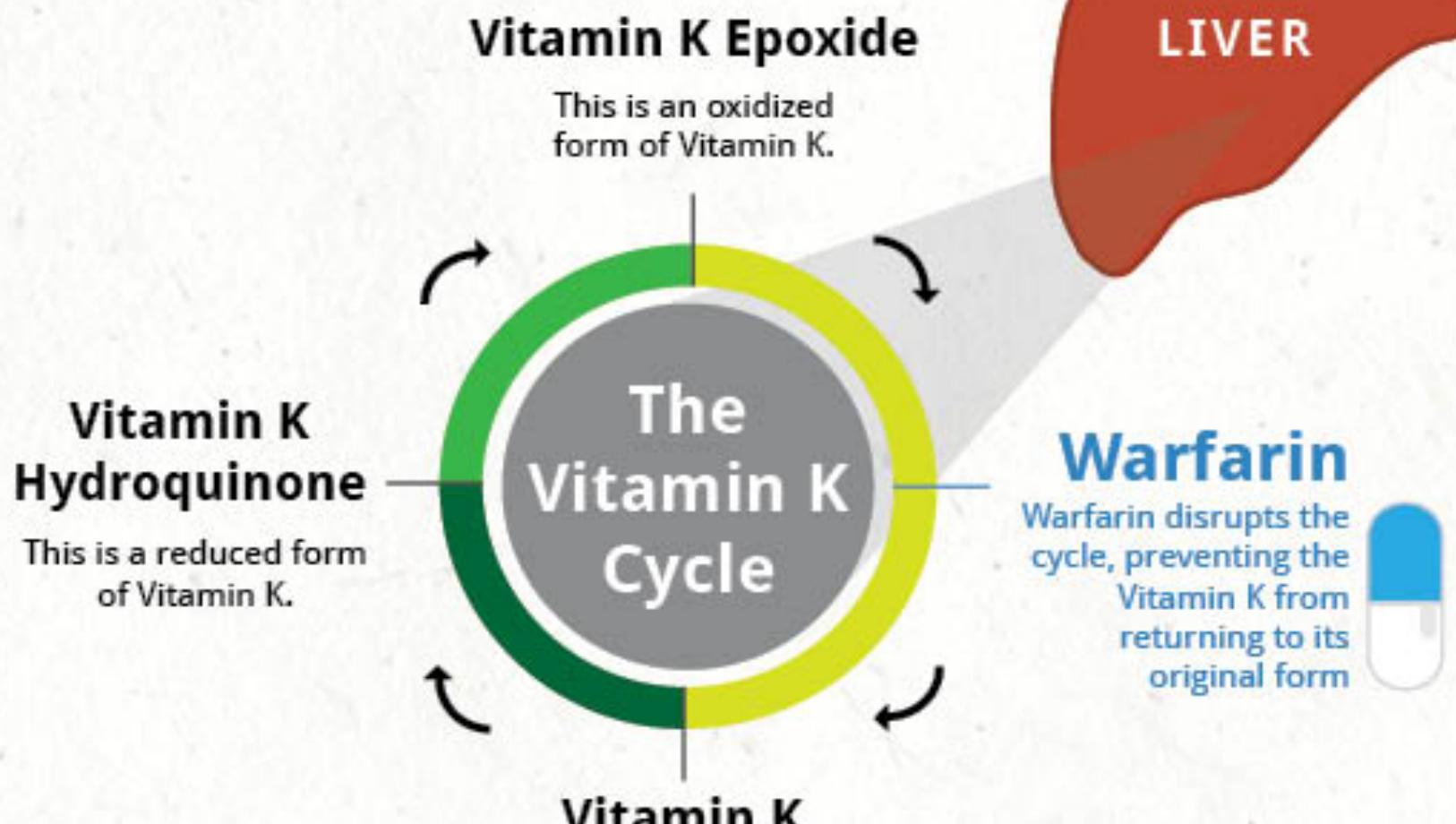
120 µg men
90 µg women

Fat-soluble



Vitamin K

Vitamin K taken from foods, usually leafy green vegetables, is absorbed in the intestinal tract and circulated through the body into the liver.



INRTracker
www.inrtracker.com

VITAMIN K BENEFITS, SOURCES & DEFICIENCY

Vitamin K is not a single nutrient, but the name given to a group of vitamins of similar composition. The two main groups of vitamin K that occur naturally are vitamin K1 and K2. K1 is found in many vegetables and K2 is produced by bacteria. Vitamin K is known as the clotting vitamin, because without it blood would not clot.

FOOD SOURCES OF VITAMIN K

Green leafy vegetables, such as kale, spinach, turnip greens, collards, Swiss chard, mustard greens, parsley, romaine, and green leaf lettuce, Brussels sprouts, broccoli, cauliflower, and cabbage.



VITAMIN K DEFICIENCY

Deficiency is rare as vitamin K is widely available from the diet and is also provided by gut bacteria. Thus, deficiency is generally secondary to conditions such as malabsorption or impaired gut synthesis. Newborn babies up to six weeks old have low levels of vitamin K, therefore, it is usual to give all newborn infants prophylactic vitamin K.

RDAC: 90 mg | Water-soluble Vitamin

BENEFICIAL FOR

- ✓ Blood clotting
- ✓ Bone health and Calcification
- ✓ Antioxidant
- ✓ Anti-inflammatory
- ✓ Brain function

While most
think I like
this cuz it
makes me
stronger, I
actually love
it for it's
Vitamin K!



A photograph of a person's arm and shoulder. The person is wearing a dark blue sleeveless shirt. A large, dark redbruise or hematoma is visible on the upper arm, extending from the shoulder area down towards the elbow. The background is a dark, textured surface.

Hemophilia

(2) Hemophilia:

(2)Hemophilia:

Hereditary, congenital, sex linked recessive disease carried by female, transmitted always to male (carried on X chromosome).

- It is 3 types:
 - Hemophilia A due to deficiency of factor VIII (85 % of cases)
 - Hemophilia B due to deficiency of factor IX (10 % of cases)
 - Hemophilia C due to deficiency of factor XI (5 % of cases).
- It is characterized by severe prolonged bleeding on mild trauma.

HEMOPHILIA

(Inherited Blood Disorder
Factor VIII, Classic, or Type A)

- No Cure
- Avoid Injury & Meds That Promote Bleeding
- Good Nutrition
- Good Dental Hygiene
- IV Administration Of Deficient Clotting Factor



Parents



+



Father
(with hemophilia)
XY

Mother
(carrier for
hemophilia gene)
XX



Son
(without
hemophilia)
XY



Daughter
(carrier for
hemophilia gene)
XX



Son
(has
hemophilia)
XY



Daughter
(has
hemophilia)
XX

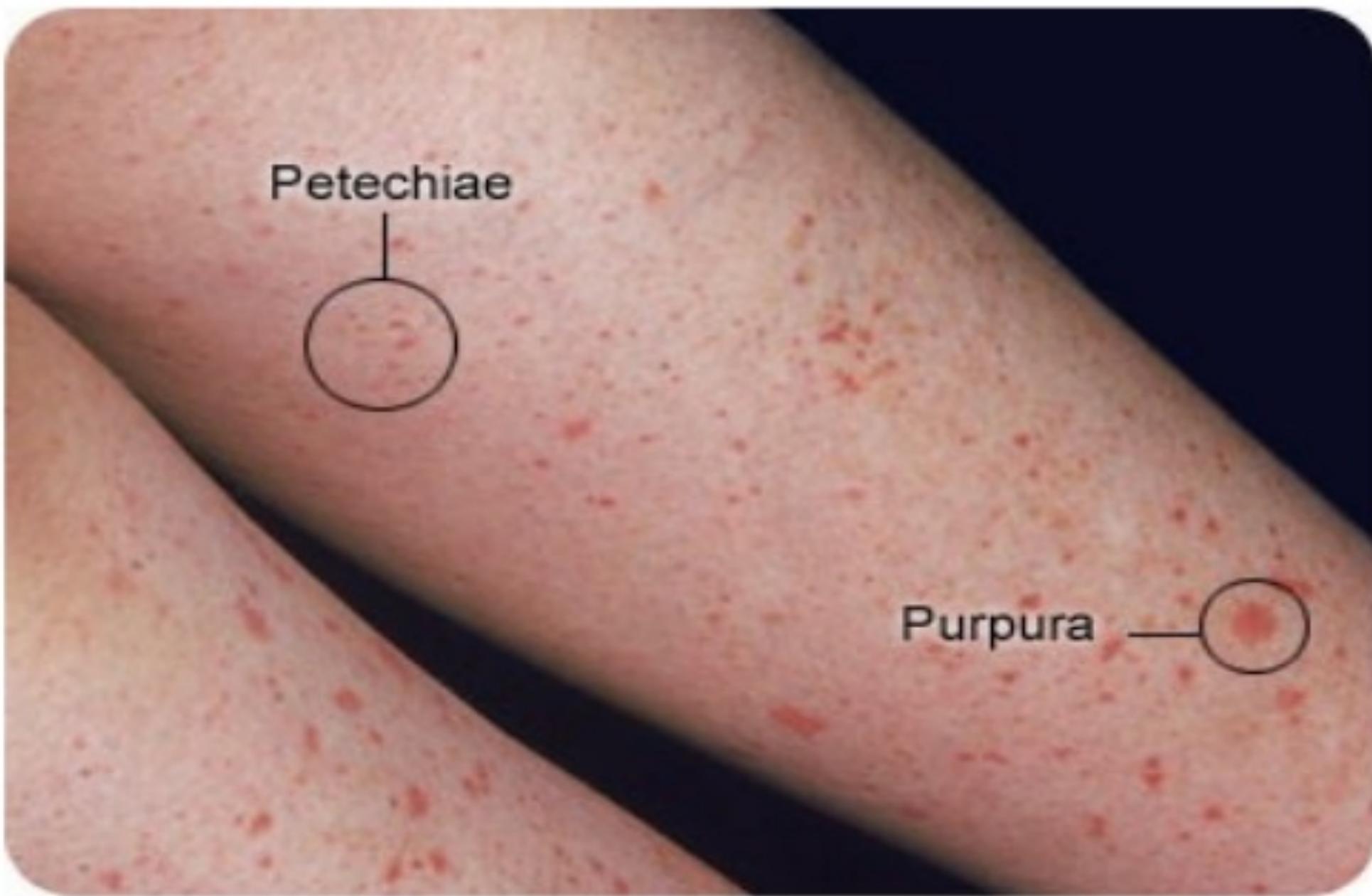
Children

Purpura



(3) Thrombocytopenic purpura:

- Characterized by subcutaneous hemorrhages which are called petichae
- It is due to decrease platelets number (bleeding occurs if it is below $50000/\text{mm}^3$).
- Bleeding time is prolonged.

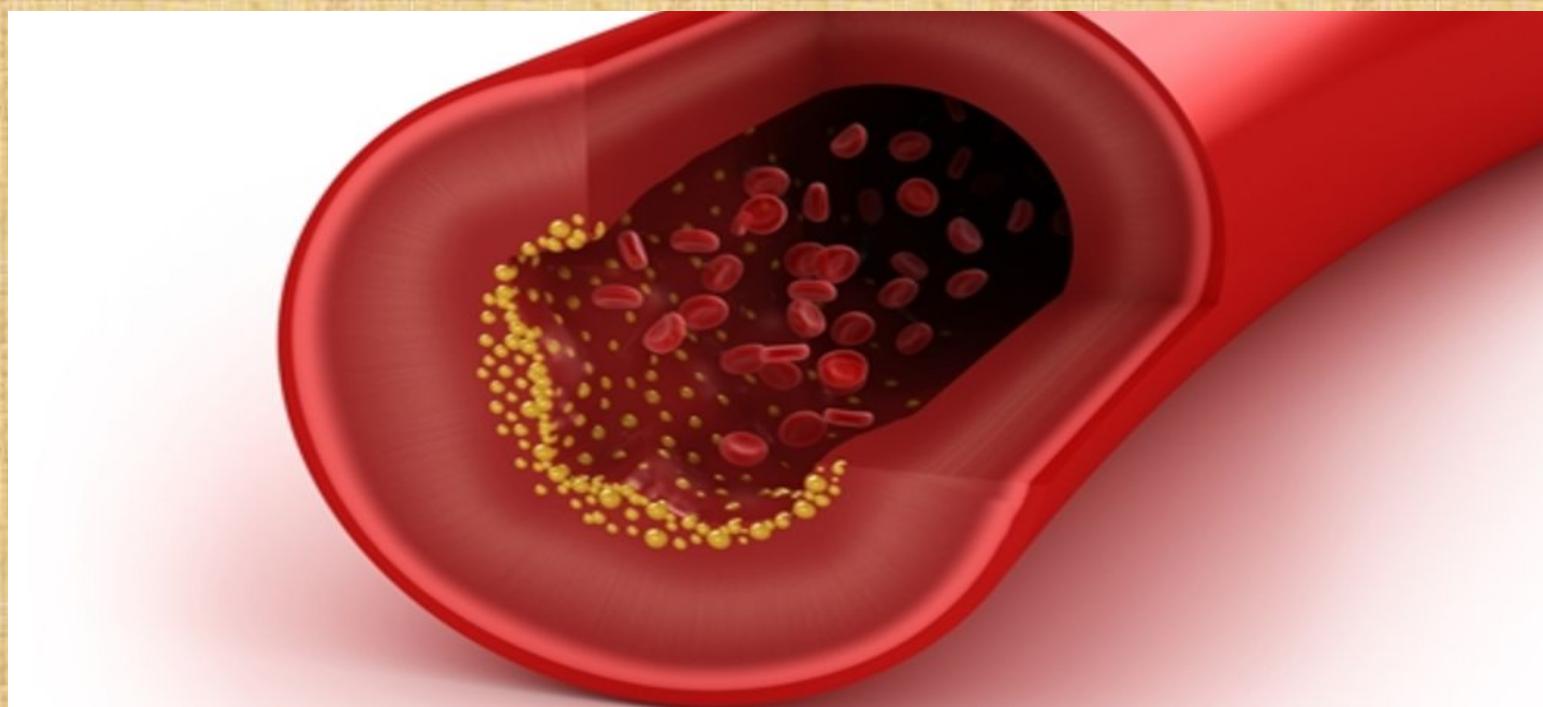
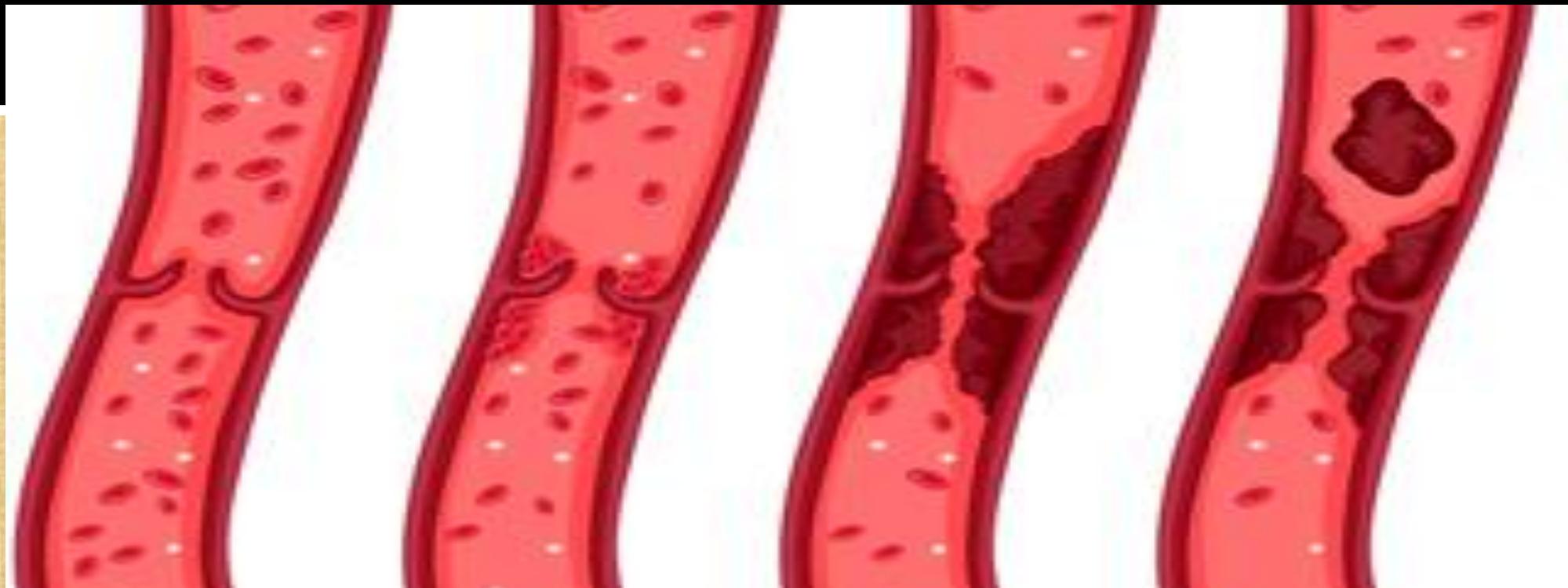


Petechiae	1-3mm
Purpura	3mm-10 mm
Ecchymosis	>10mm



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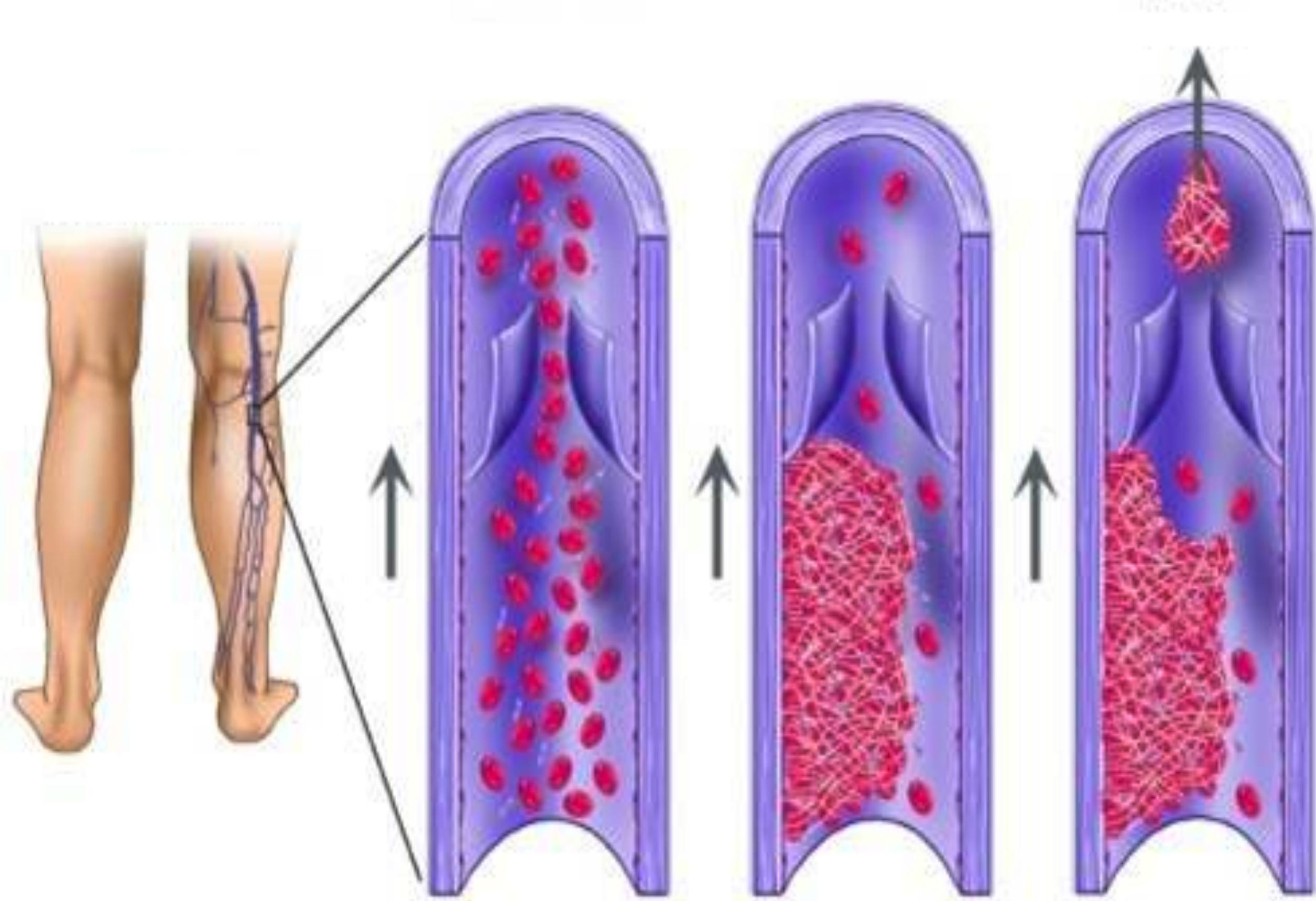
Thromboembolism



(4)Thromboembolism:

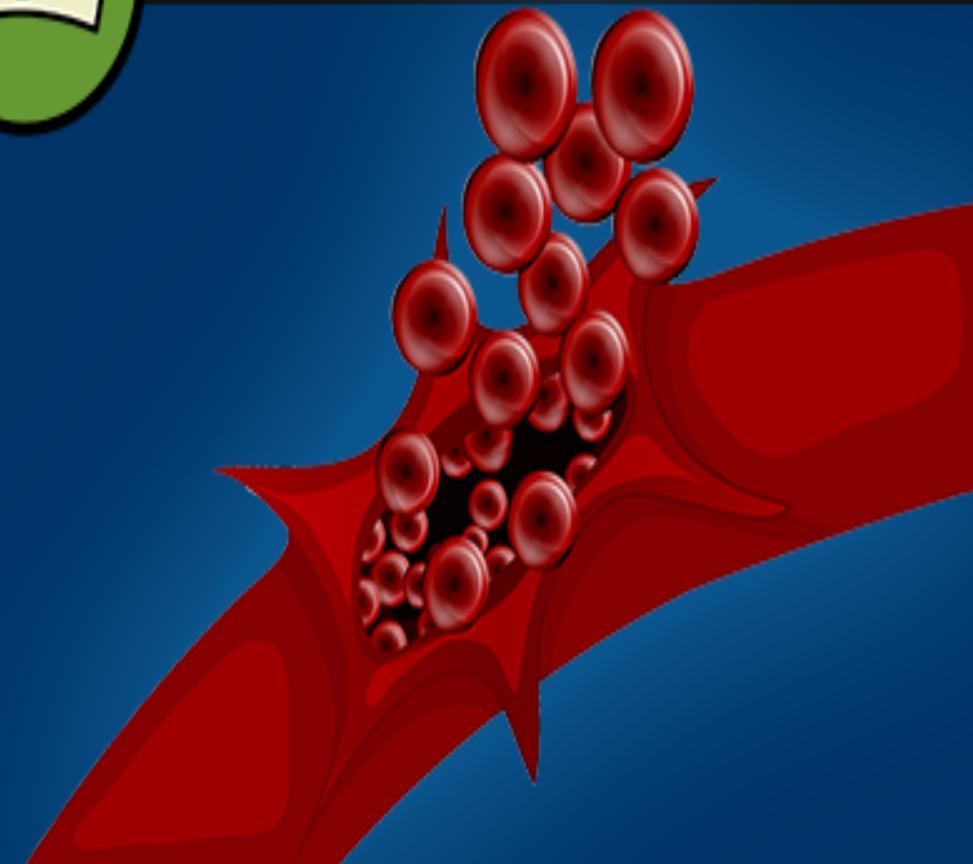
Clot is formed inside blood vessels as in
atherosclerosis & after operation.

(



5) Disseminated intravascular clotting(DIC):

Excessive bleeding & clot formation which may occur in intrauterine fetal death, repeated blood transfusion or repeated renal dialysis.



Disseminated Intravascular Coagulation

NURSING CARE PLANS

Nurseslabs

DISSEMINATED INTRAVASCULAR COAGULATION



•ADAM

