Hemostasis

Red: very important.
Green: only found in males' slides.
Purple: only found in females' slides.
Gray: notes.

Physiology Team 436 – Foundation block lecture 12

Lecture: If work is intended for initial studying.
Review: If work is intended for revision.

Objectives

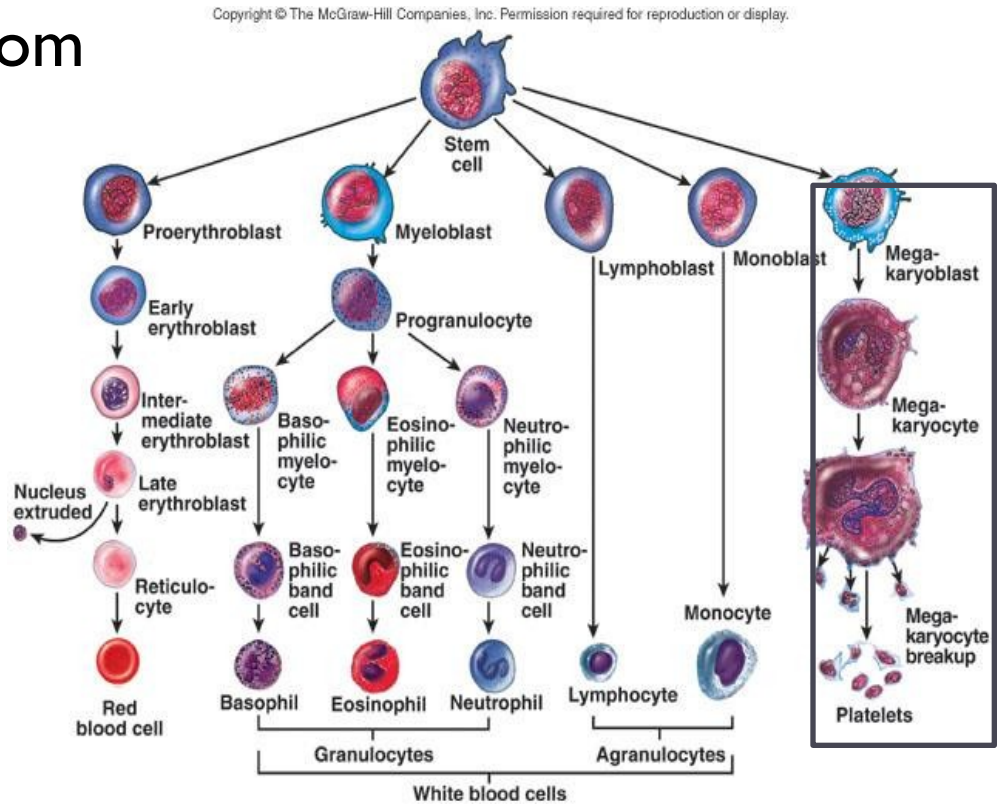
- Recognize different stages of hemostasis.
- Describe formation and development of platelets.
- Explain the role of platelets in hemostasis.
- Recognize different clotting factors & cascade of clotting.
- Describe the intrinsic, extrinsic and common pathways.
- Recognize the role of thrombin in coagulation.
- Explain the process of fibrinolysis and function of plasmin.

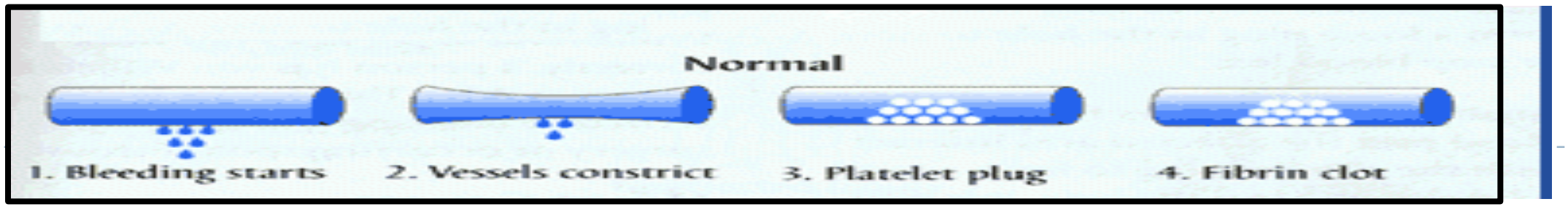
Hemostasis

- It is the spontaneous arrest (stop) of bleeding from ruptured blood vessels.

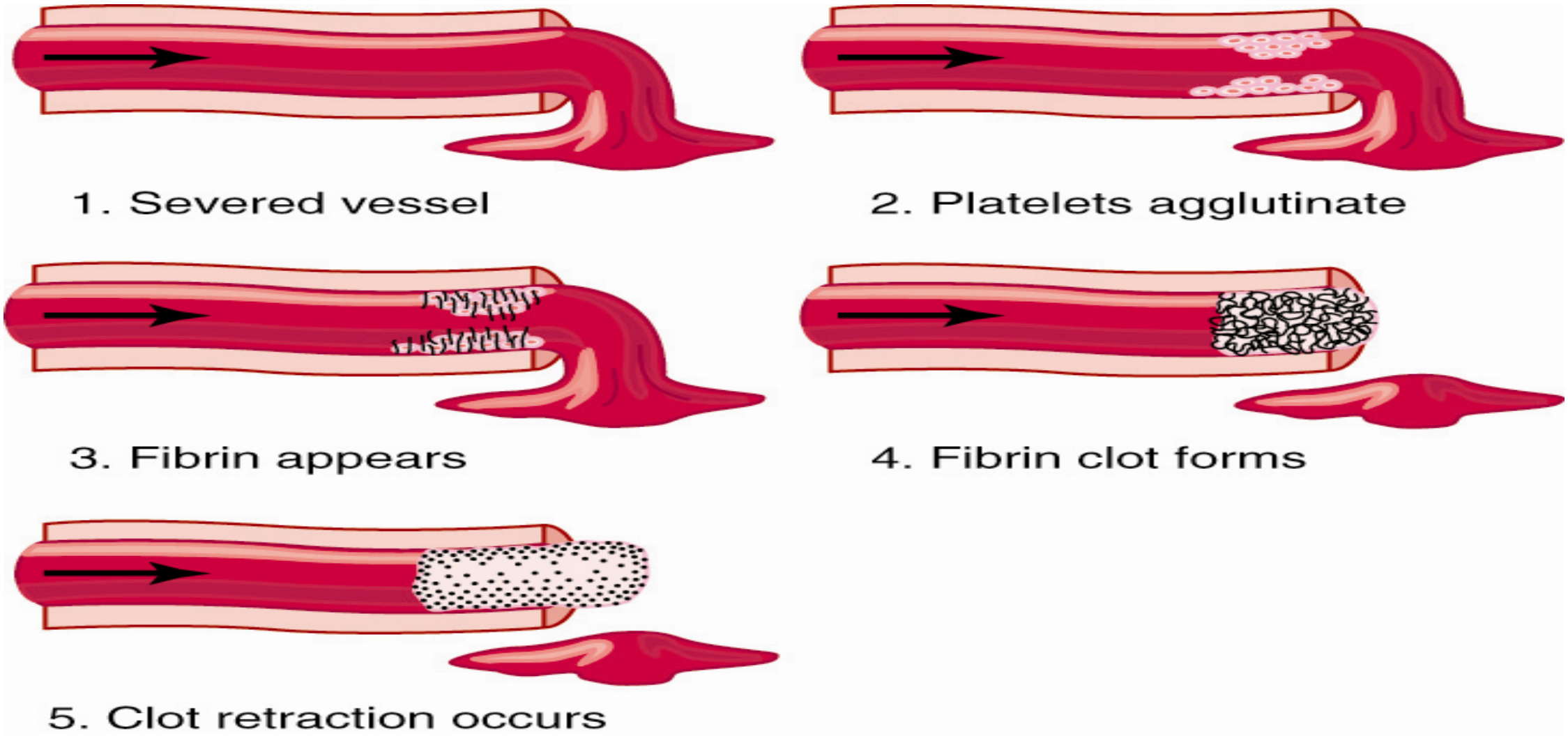
- Its mechanism (steps of function):

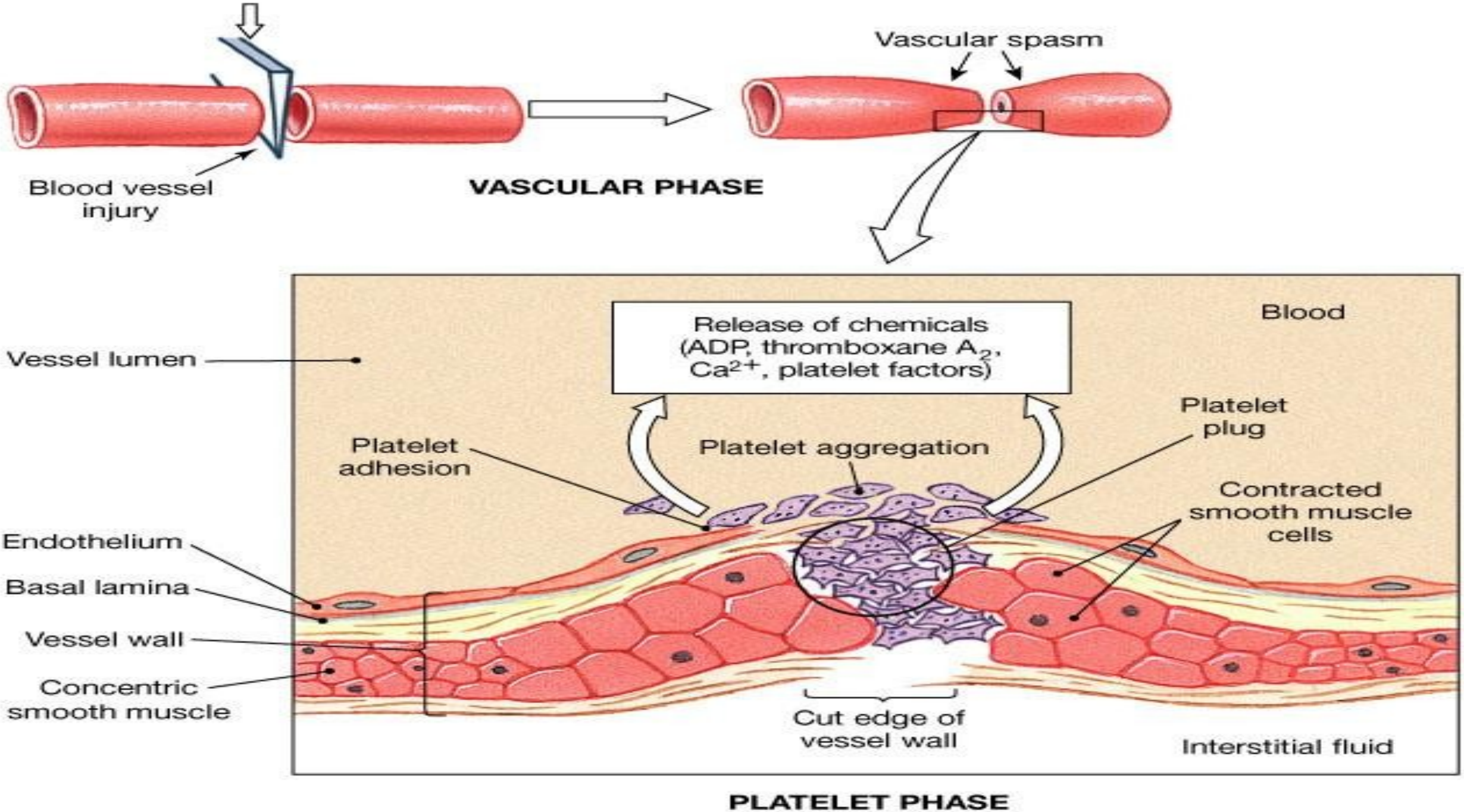
1) **Vessel wall (Vascular spasm).**
2) **Formation of platelet plug.**
3) **Blood coagulation & clot retraction.**
4) **Fibrinolytic system (Fibrinolysis).**





Steps of Hemostasis





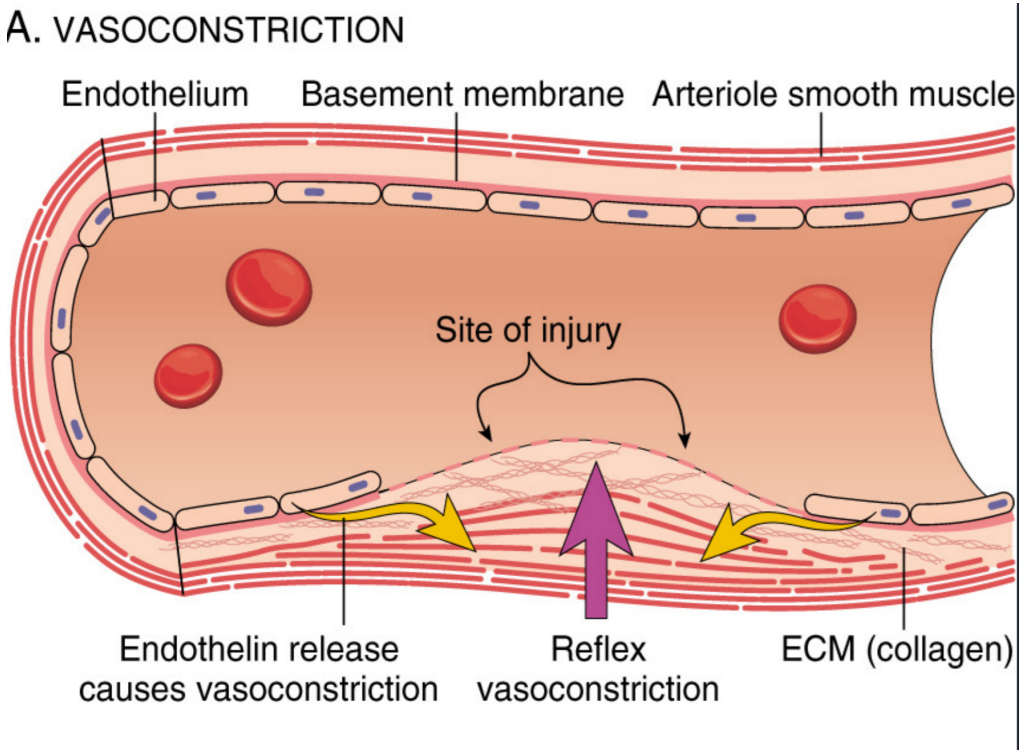
Copyright © 2004 Pearson Education, Inc., publishing as Benjamin Cummings.

Hemostatic Mechanism

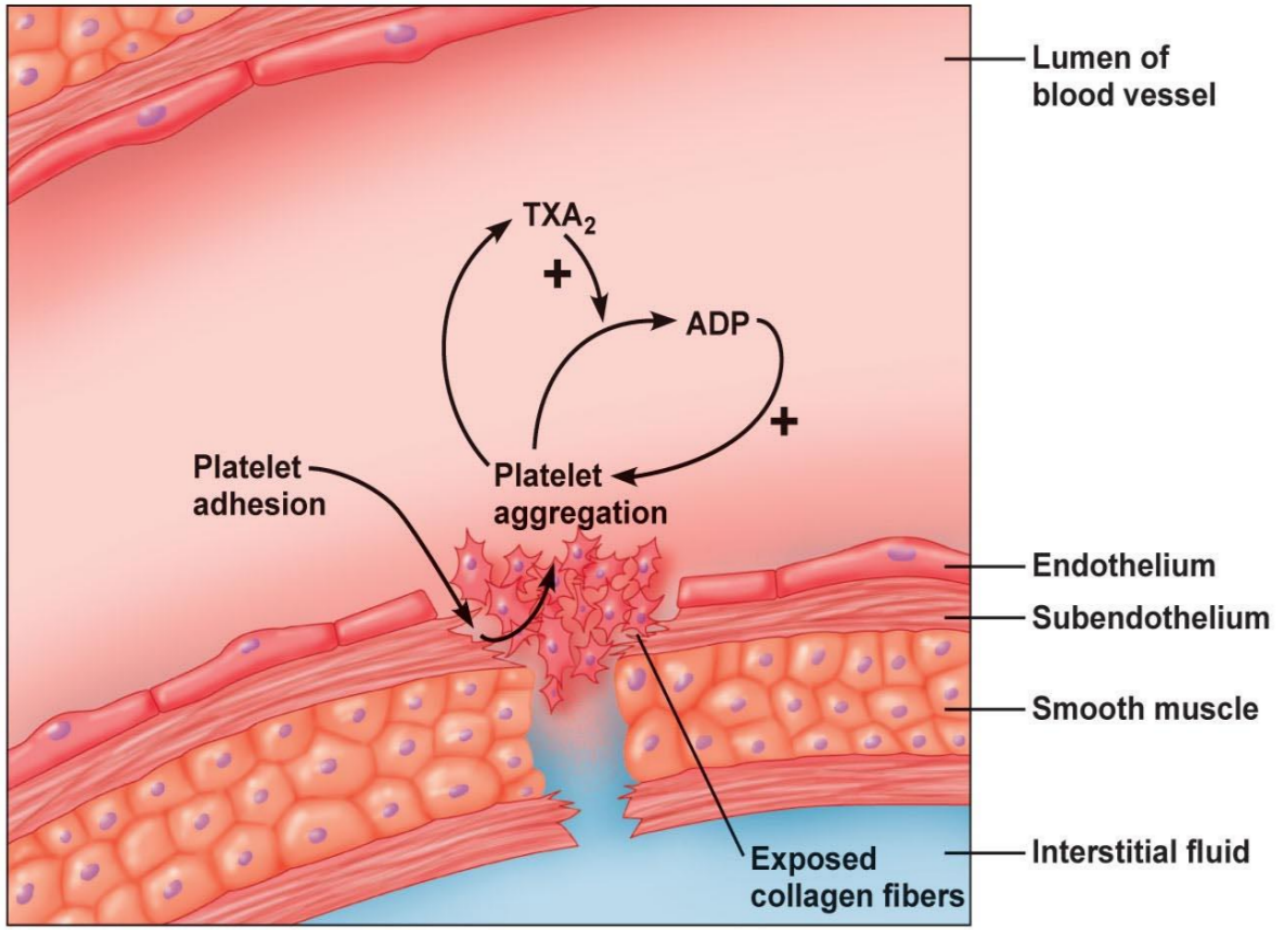
1-Vessel wall.

- Immediately after injury there is a localized **Vasoconstriction** (vascular spasm, in order to decrease blood flow)
- **Mechanism of vasoconstriction (causative Factor):**
  - Local myogenic spasm (systemic release of adrenaline).
  - Local release of Thromboxane A2 [TXA2] & 5HT" serotonin" by platelets. (humoral factors)
  - Nervous factors "stimulation for nerve impulses + nerve reflex is vasocontraction". (reflex action involves simple nervous pathway)
- **Importance:**

Crushing injuries > intense spasm > no lethal loss of blood.

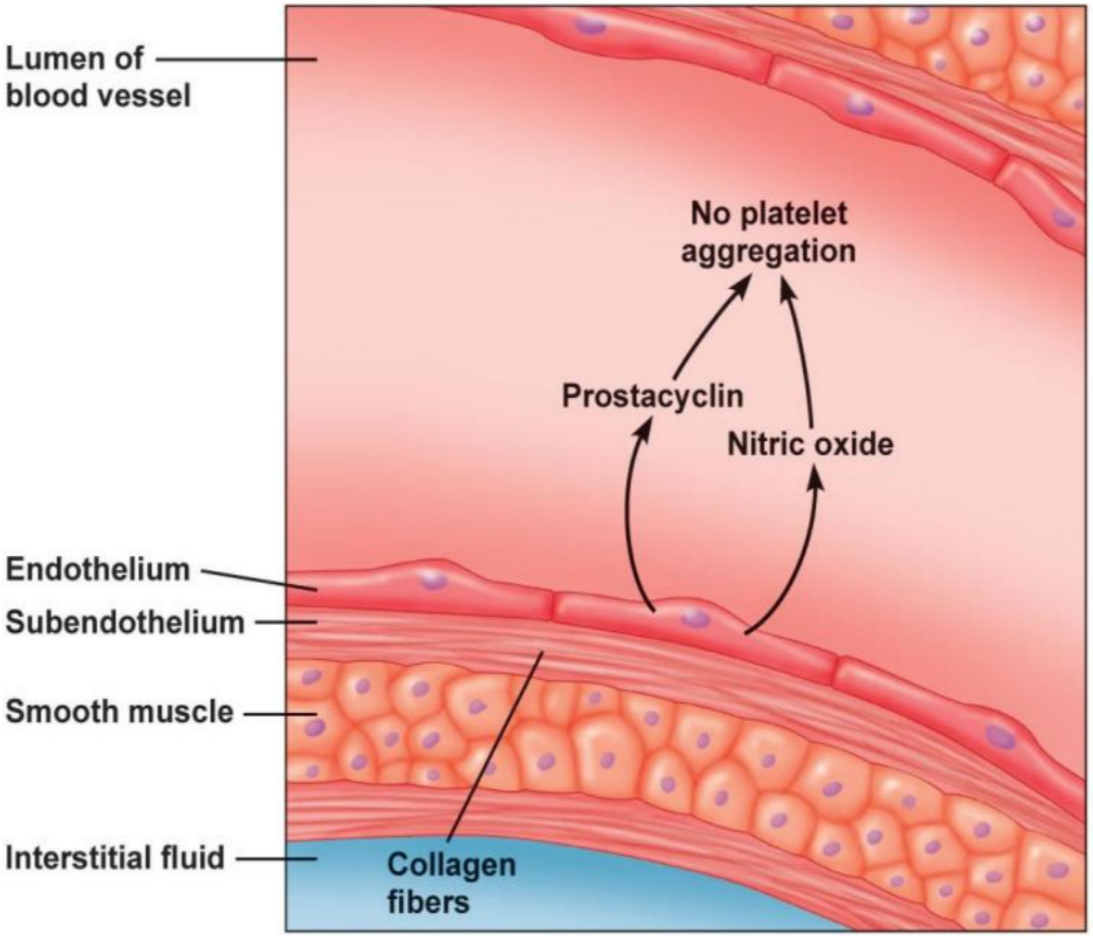


Notes:
Causative Factor: meaning that it is "a cause"
Vasoconstriction is produced by vascular smooth muscle cells, and is the blood vessels first response to injury.
The damaged vessels will constrict (vasoconstrict) which reduces the amount of blood flow through the area and limits the amount of blood loss.
Myogenic contraction refers to a contraction initiated by the myocyte cell. (muscle cell)
Platelets release cytoplasmic granules which contain, ADP, 5HT (serotonin) and thromboxane A2 (TXA2), all of which, increase the effect of vasoconstriction. *will be explained in the next slides*



(a) Damaged blood vessel endothelium

© 2011 Pearson Education, Inc.



(b) Normal blood vessel endothelium

© 2011 Pearson Education, Inc.

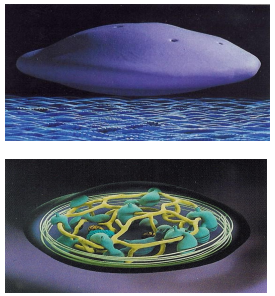
2-Formation of platelet plug (primary hemostasis)

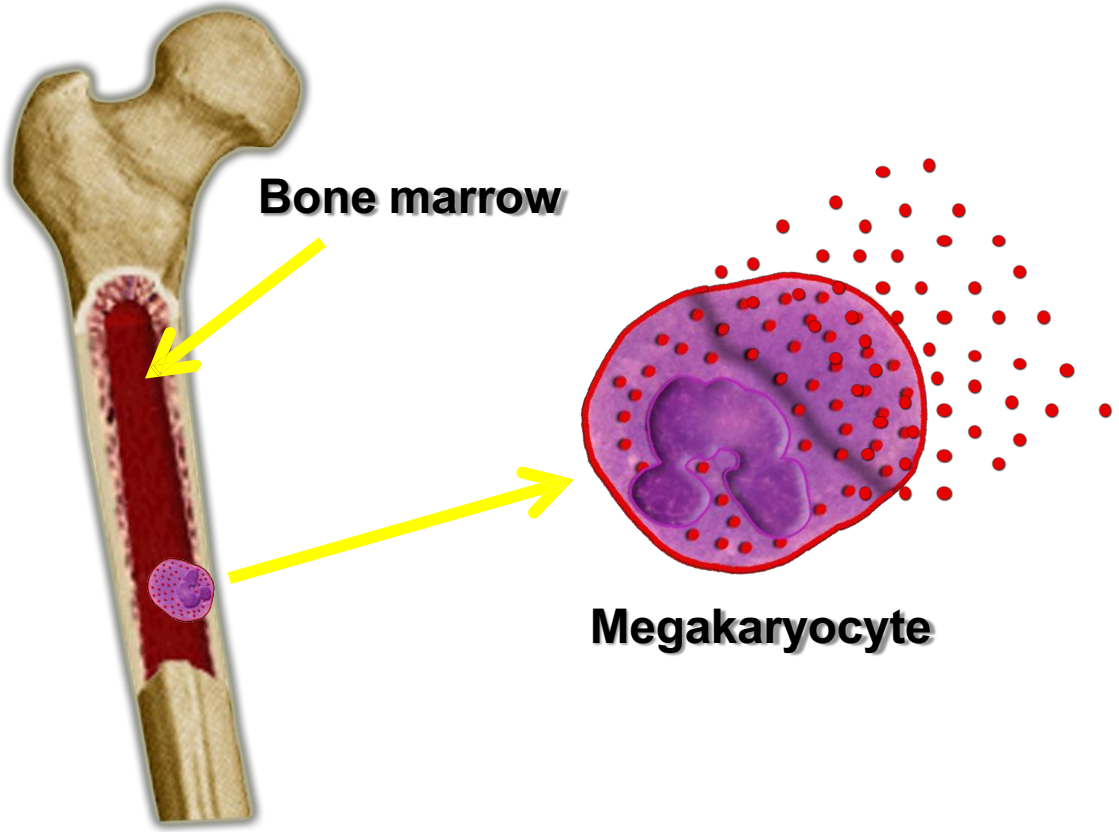
- Platelets are small "biconvex" disc shaped cells.
- (Remember: RBCs are biconcave discs)
- **Platelet count**: $150x10^3$ – $300x10^3$/ml.
- **Life span**: 8 – 12 days.
- Contain high calcium content & rich in ADP.
- Active cells contain contractile protein.
- Thrombocytes are fragments of megakaryocytes in the bone marrow.
- Regulation of thrombipoiesis is done by thrombopoietin.
- **Site of formation**: bone marrow, steps:

Stem cell > Megakaryoblast > **Megakaryocyte** > Platelets.

- Importance: enough to stop bleeding from small vascular damage.

Platelet shape:

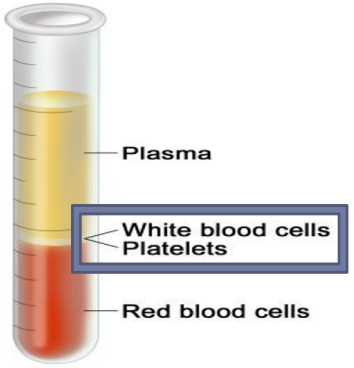


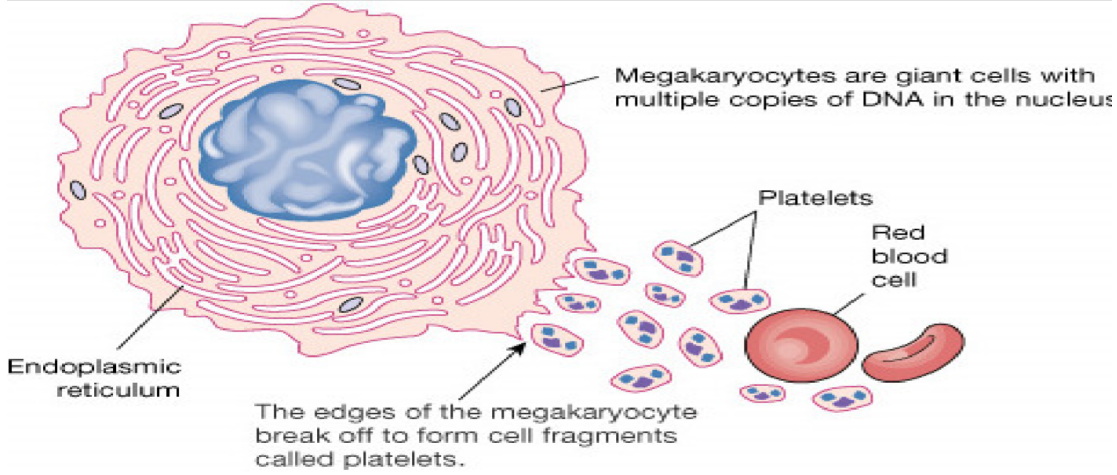


زي Nucleus مافيها الـ RBCs بس فيها Enzyme

It also has pores that enable movement of substances from granules to circulation.

Remember: Regulation of Erythropoiesis by Erythropoietin (Erythropoietin is formed in kidney & liver , while Thrombopoietin in liver)





Platelet Cont.

Platelet Charactersitics:

shape: minute round or oval discs
size: 1-4 um in diameter
location: 80% in blood & 20% in spleen

Contractile, adhesive, cell fragments.

Store coagulation factors & enzymes

Surface Binding sites: **GP Ib** (Glyco Protein Ib or 1b on cell membrane of platelets) is a component of **GP Ib-IX-V complex** that functions as a receptor of vW factor.

FUNCTIONAL CHARACTERISTICS:

- Motile: Actin And Myosin Molecules (for muscle contraction)
- Active: Endoplasmic Reticulum, Golgi Apparatus & Mitochondria
- Enzymes Systems For Synthesis Of Prostaglandins
- Granules

Postglandins: any of a group of compounds with varying hormone-like effects

Platelet Receptors:

-**GP Ia, GP VI** (adhesion to Collagen)
-**GP Ib-IX-V** (receptor for vW Factor)
-**TPα** (for TXA2)
-**GP IIb-IIIa** (for Fibrinogen, vW Factor)
-**P2Y12** (for ADP)

Dense or δ granules contain:

- Serotonin
- ADP
- Ca++

α granules contain:

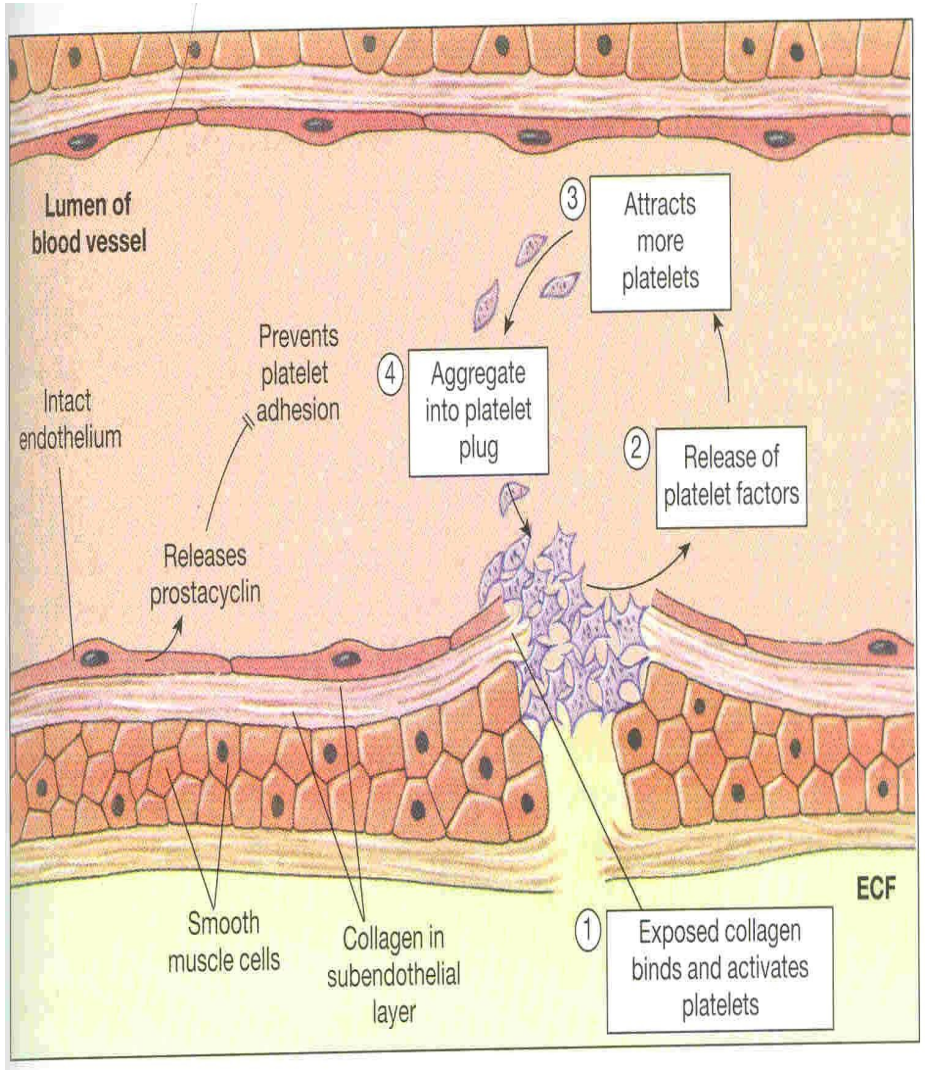
- Coagulation Factors

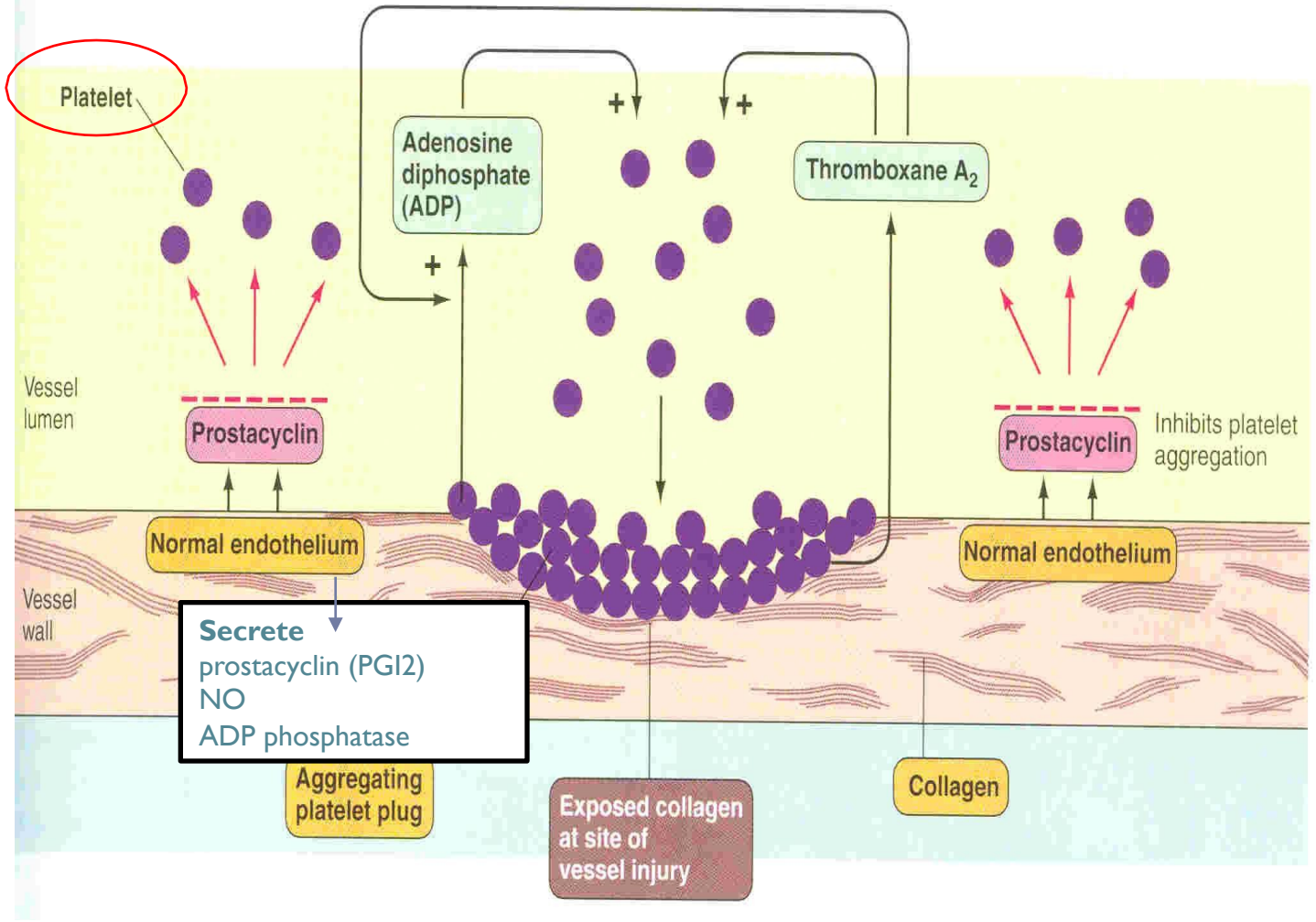
Fibrinogen

- PDGF (Platelet-derived growth factor)

Platelet Plug Formation

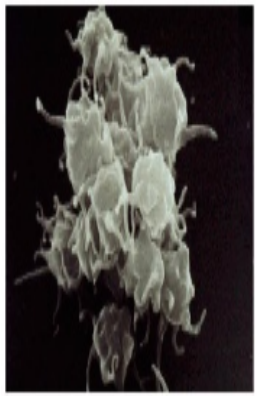
(Importance → enough to stop bleeding from small vascular damage.)

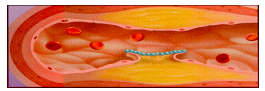


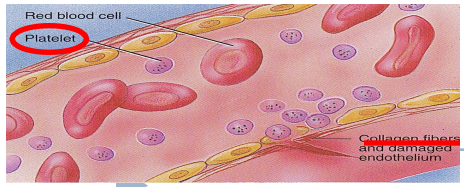


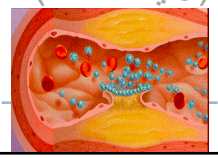
Intact (normal) endothelium secret **prostacyclin** and **NO** which inhibit **aggregation**.

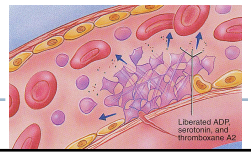
Platelet Function:
Begins With Platelet (Thrombocytes) Activation:

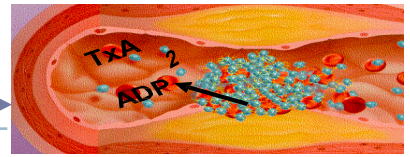
Platelet adhesion	Shape change	Activation and Aggregation*	Release reaction/Secretion	Clot retraction*
○ Exposed collagen attracts platelets, ○ Platelets stick to exposed collagen (to the sub endothelial layer) (Von WilleBrand Factor) released from the damaged endothelial cells in vessel wall or underlying damaged endothelial cells in vessel wall.	 Resting platelets  **Activated Platelets*** Activated platelets	○ Activated platelets stick together and activate new platelets to form a mass called a **platelet plug.** ○ Plug reinforced مثبّت by fibrin threads formed during clotting process.** ○ *Platelet aggregation: The clumping together of platelets in the blood. **خيوط تسوي زي الشبكة تمسكها عشان يوقف الدم (زي السداده)	○ Activated platelets (activated by adhesion) extend projections (protrusions) to make contact with each other and release **Serotonin, ADP (activating other platelets) & thromboxane A2 (TXA2).** ○ Serotonin & TXA2 are vasoconstrictors decreasing blood flow through the injured vessel. ○ 1. ADP & TXA2: increase the **stickiness** of platelets ○ 2. **aggregation** is increased. ○ 3. cut vessel is **plugged.**	○ Myosin and actin filaments in platelets are **stimulated to contract** during aggregation further **reinforcing** the plug and **help release of granule contents.** ○ *Clot retraction: is the shrinking of a clot over a number of days.











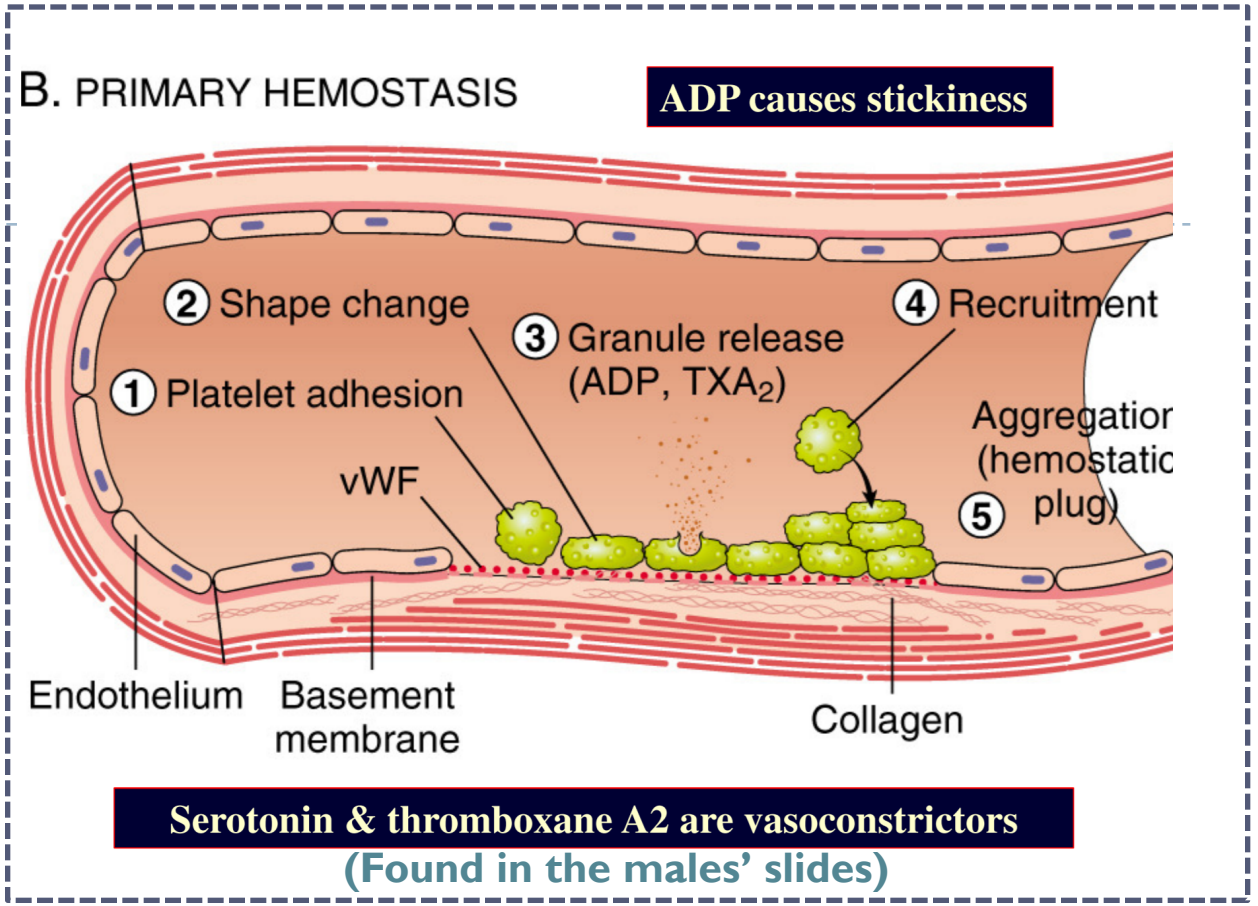
Video ;- https://www.youtube.com/watch?v=9QVTHDM90io

Activated Platelets (during shape change)

- When platelets are activated they secrete:

1) **5HT** > causes vasoconstriction.
2) **ADP (Adenosine Di-Phosphate) >** stickiness
3) **Platelet phospholipid (PF3)** > clot formation.
4) **Thromboxane A2 (TXA2)** > a prostaglandin formed from arachidonic acid, and it is inhibited by aspirin. (its function is vasoconstriction and platelet aggregation)

يمنع تكون الجلطات لمقدرته على جعل الدم في حالة سائلة



Further explanation of the process: platelets adhere to damaged endothelium to form a platelet plug (*primary hemostasis*) and then degranulate Platelets play one of the biggest roles in the hemostatic process. When platelets come across the injured endothelium cells, they change shape, release granules and ultimately become 'sticky'. Platelets express certain receptors, some of which are used for the adhesion of platelets. When platelets are activated, they express receptors that interact with other platelets, producing aggregation and adhesion. Platelets release cytoplasmic granules such as (ADP), serorotin and thromboxane A2. Adenosine diphosphate (ADP) attracts more platelets to the affected area, serotonin is a vasoconstrictor and thromboxane A2 assists in platelet aggregation, vasoconstriction and degranulation. More chemicals are released more platelets stick and release their chemicals; creating a platelet plug and continuing the process in a positive feedback loop. Platelets alone are responsible for stopping the bleeding of unnoticed wear and tear of our skin on a daily basis. This is referred to as primary hemostasis

Hemostatic Mechanism (cont.)

1 - I	6 - VI	11 - XI
2 - II	7 - VII	12 - XII
3 - III	8 - VIII	13 - XIII
4 - IV	9 - IX	14 - XIV
5 - V	10 - X	15 - XV

3- Blood Coagulation: secondary hemostasis

- **CLOT** is a meshwork of fibrin fibers running in all directions entrapping blood cells, platelets and plasma.
- **Blood clotting** is the transformation of blood (soluble **fibrinogen**) from a liquid into a solid gel form (insoluble **fibrin** strands) this fibrin will strengthen the previous Platelet Plug.

- **Clotting Cascade Pathways**: Intrinsic and Extrinsic
- Begins to develop in

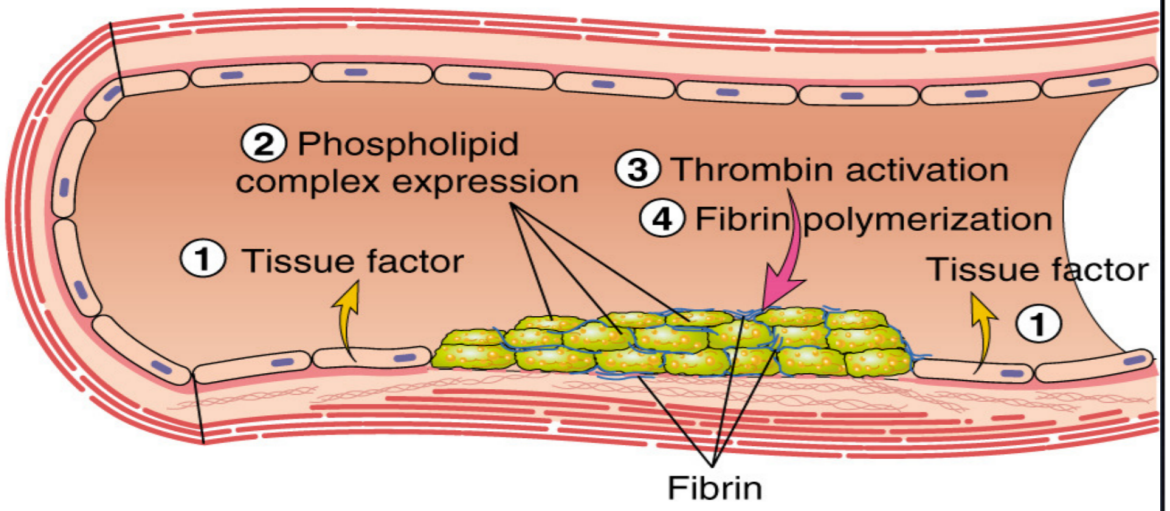
15-20 sec → MAJOR/SEVERE TRAUMA

1-2 min → MINOR TRAUMA

Factors	Names
I (1)	Fibrinogen
II (2)	Prothrombin
III (3)	Thromboplastin
IV (4)	Calcium
V (5)	Labile factor
VII (7)	Stable factor
VIII (8)	Antihemophilic factor A
IX (9)	Antihemophilic factor B
X (10)	Stuart-Power factor
XI (11)	Plasma thromboplastin antecedent (PTA)
XII (12)	Hagman factor
XIII (13)	Fibrin stablizing factors

Clotting Factors
Circulate in plasma in inactive state
*most of them synthesized by the liver

C. SECONDARY HEMOSTASIS



D. THROMBUS AND ANTITHROMBOTIC EVENTS

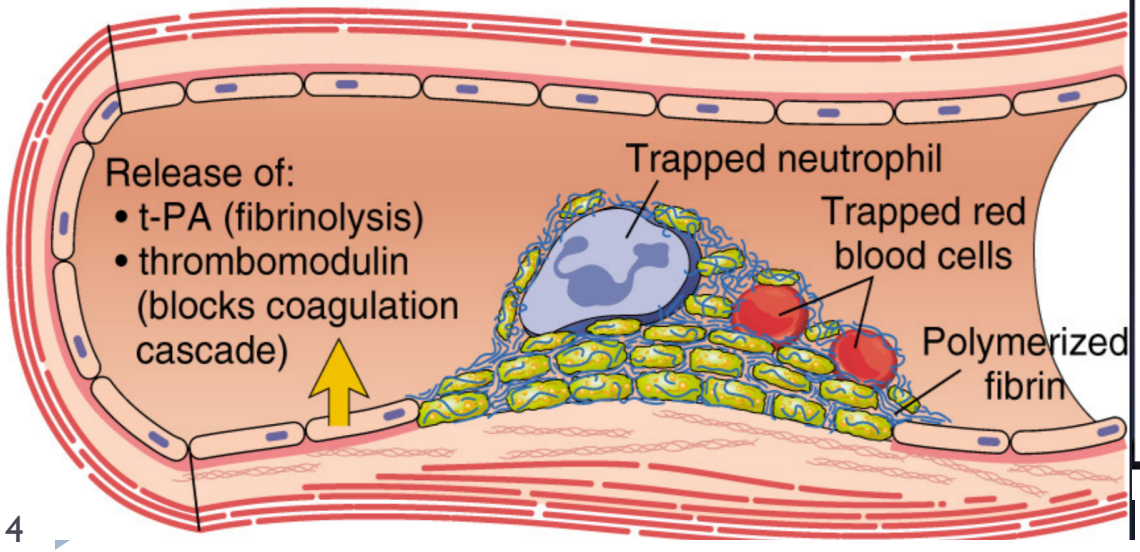


TABLE 31–5 System for naming blood-clotting factors.

Factor[a]	Names
I	Fibrinogen
II	Prothrombin
III	Thromboplastin
IV	Calcium
V	Proaccelerin, labile factor, accelerator globulin
VII	Proconvertin, SPCA, stable factor
VIII	Antihemophilic factor (AHF), antihemophilic factor A, antihemophilic globulin (AHG)
IX	Plasma thromboplastic component (PTC), Christmas factor, antihemophilic factor B
X	Stuart–Prower factor
XI	Plasma thromboplastin antecedent (PTA), antihemophilic factor C
XII	Hageman factor, glass factor
XIII	Fibrin-stabilizing factor, Laki–Lorand factor
HMW-K	High-molecular-weight kininogen, Fitzgerald factor
Pre-Ka	Prekallikrein, Fletcher factor
Ka	Kallikrein
PL	Platelet phospholipid

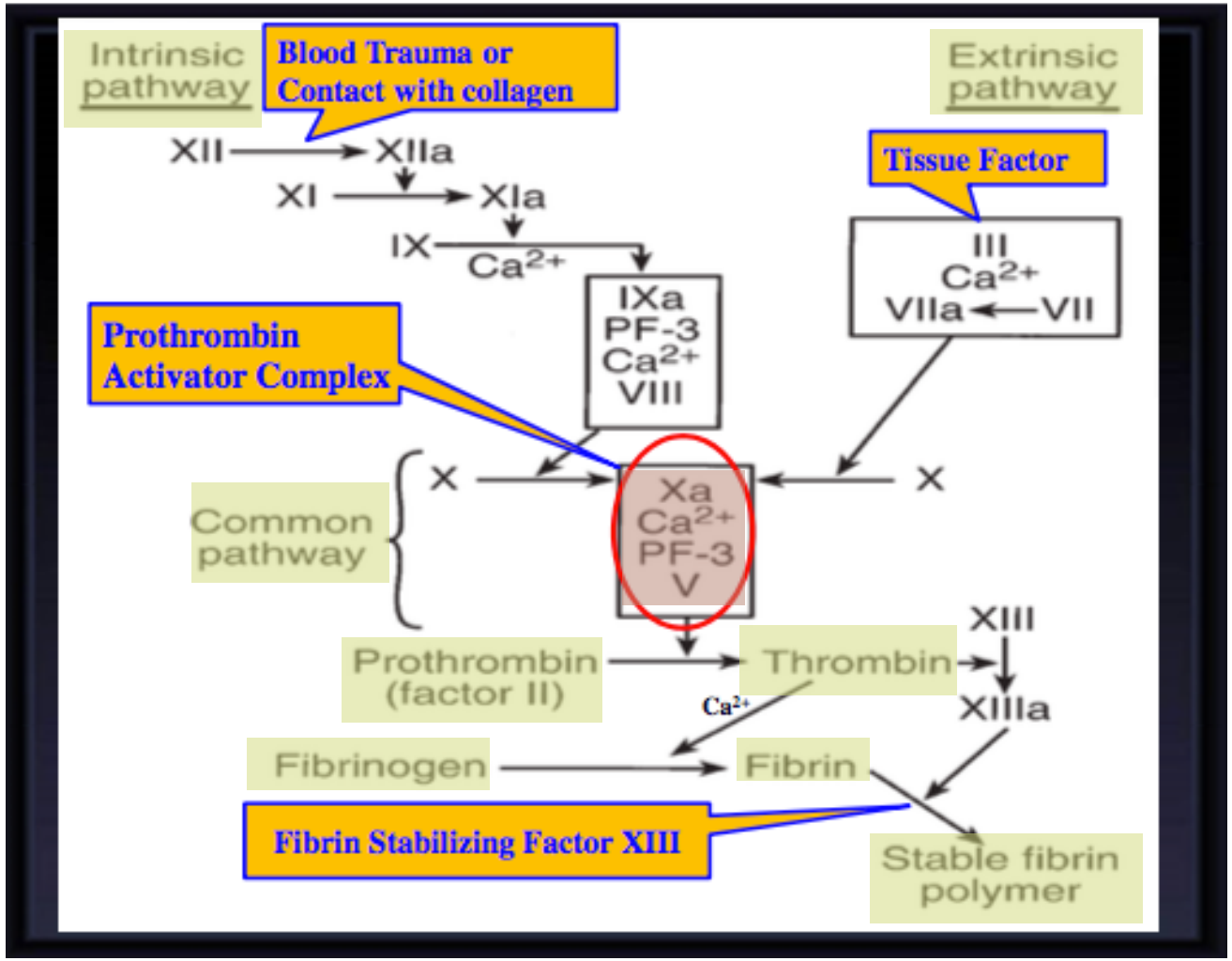
[a]Factor VI is not a separate entity and has been dropped.

Found in the males' slides

Blood coagulation (clot formation) mechanism (steps)

- A series of biochemical reactions leading to the formation of a blood clot
- **1. Formation of Prothrombin activator complex (Xa+Ca+PF-3+V)** by Extrinsic & Intrinsic Pathways → leading to **Common Pathway**.
- **2.** This reaction leads to the activation of thrombin enzyme from inactive form prothrombin **(Conversion of prothrombin** (clotting factor II) **into thrombin)**
- Prothrombin (inactive thrombin) is activated by a long intrinsic or short extrinsic pathways
- **3.** Thrombin will change fibrinogen (plasma protein) to fibrin (insoluble protein)

  (Conversion of fibrinogen into fibrin)
- **4. Fibrin converts to stable fibrin polymer**



Clotting Cascade

Found in the males' slides

Explanation:

1- يتنشط الـ Coagulation system

2- الـ Prothrombin (inactive form) يتنشط عن طريق Intrinsic or short الى ايش ؟ الى Extrinsic pathways

Thrombin (active form enzyme)

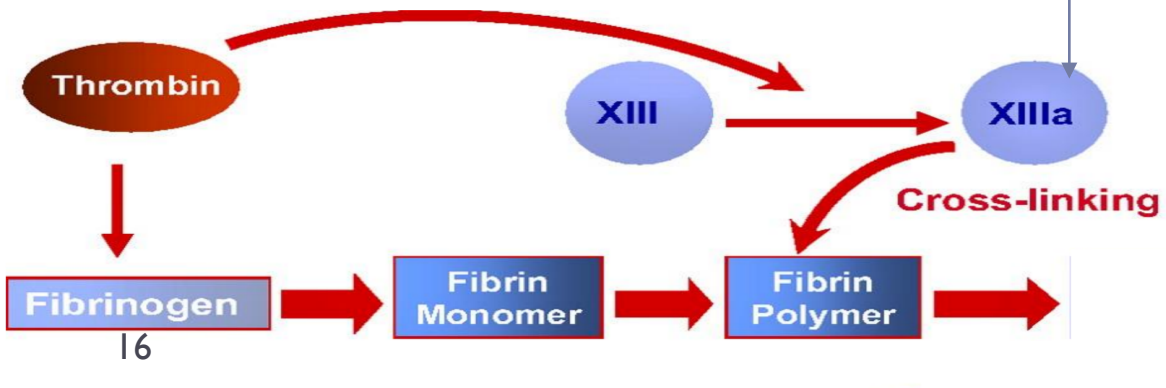
3- يجي الـ Thrombin يخلي الـ Fibrogen (plasma protien+soluble in plasma) الموجود بالدم يتحول الى

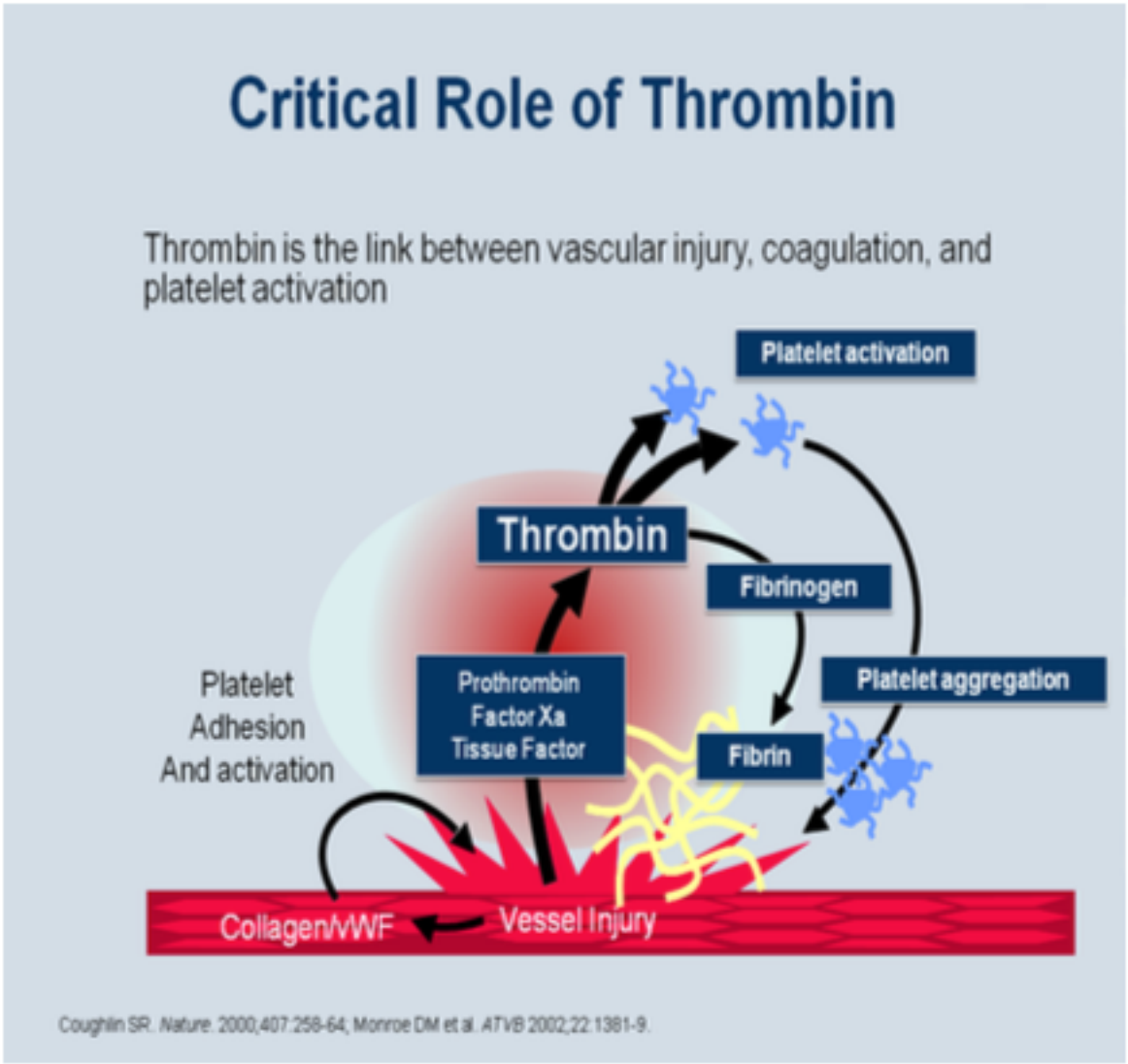
Fibrin (insoluble protein)

4- Blood clot

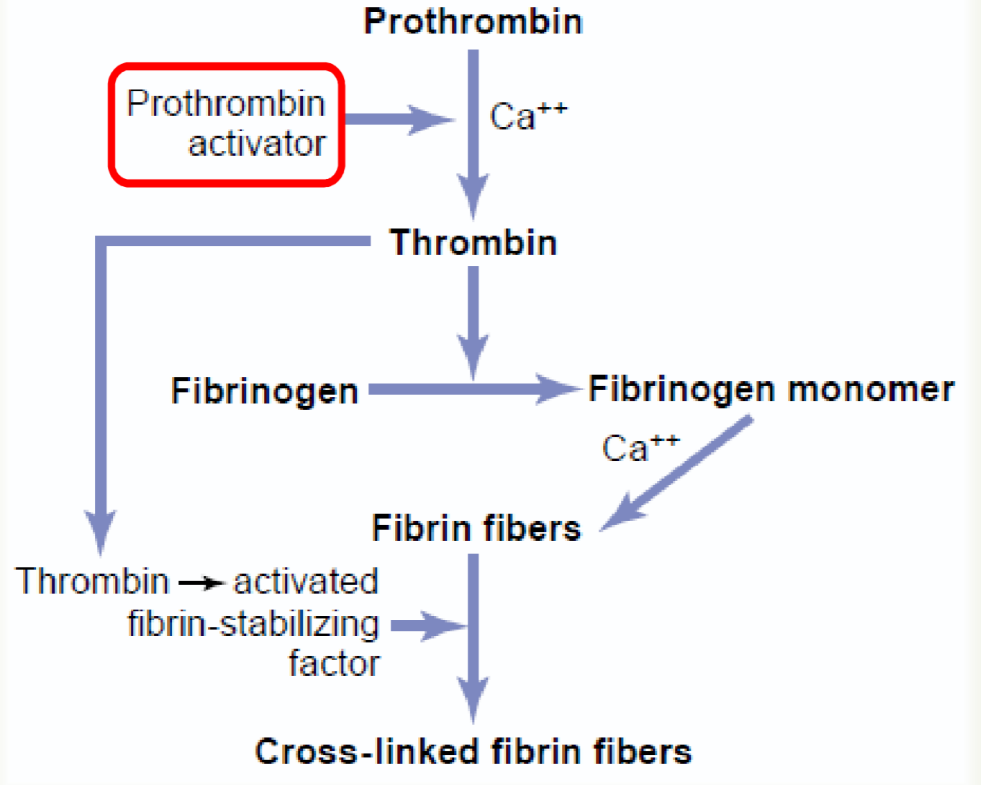
Thrombin

- Thrombin changes **fibrinogen to fibrin**
- Activates **factor V** (proaccelerin or labile factor) and XIII (fibrin stabilizing meshwork)
- Thrombin is essential in platelet morphological changes to form primary plug.
- Thrombin stimulates platelets to release **ADP & thromboxane A2**; both stimulate further platelets aggregation.
- Explanation: As part of its activity in the coagulation cascade, thrombin also promotes platelet activation and aggregation via activation of receptors on the cell membrane of the



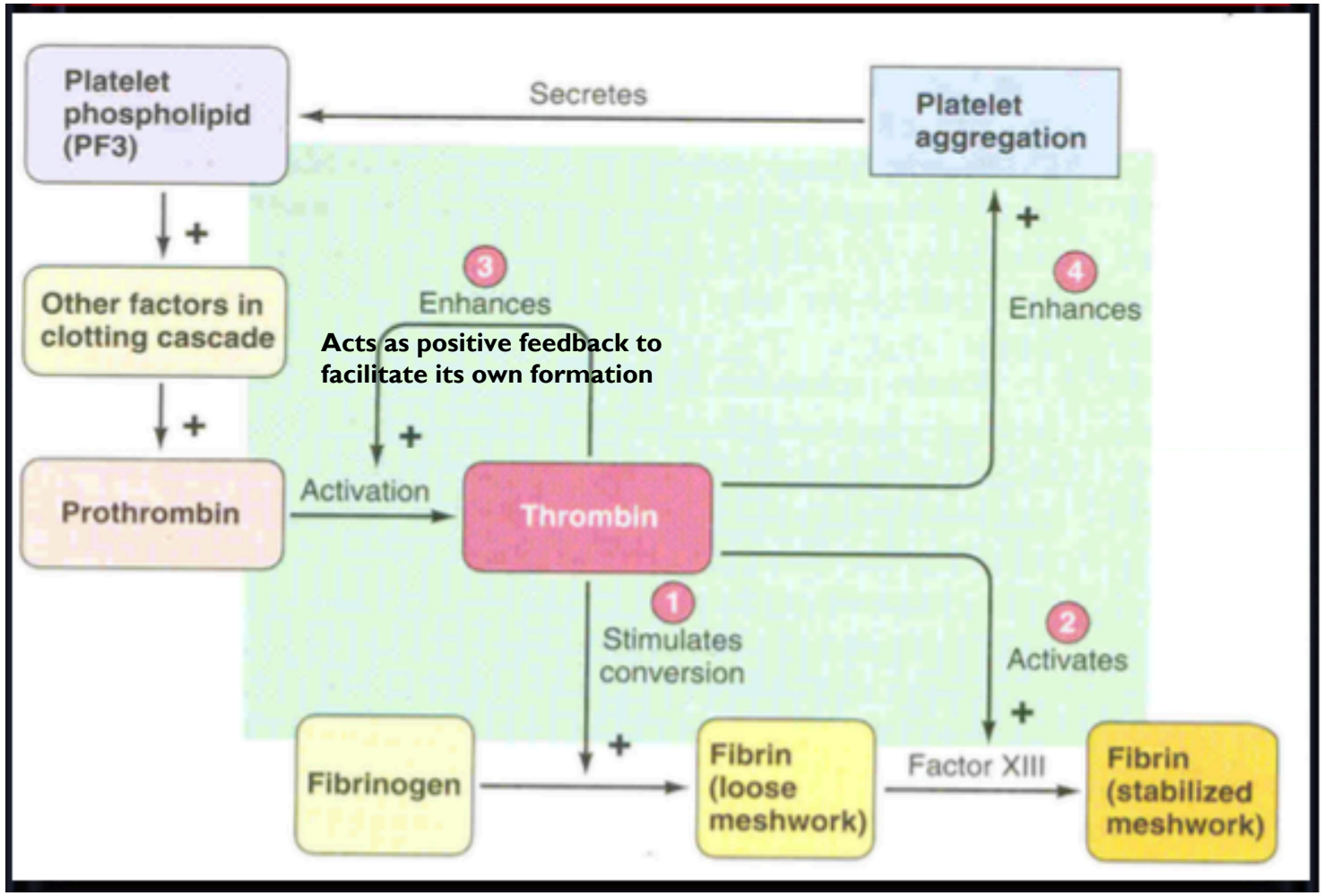


ACTION OF THROMBIN ON FIBRONOGEN TO FORM FIBRIN



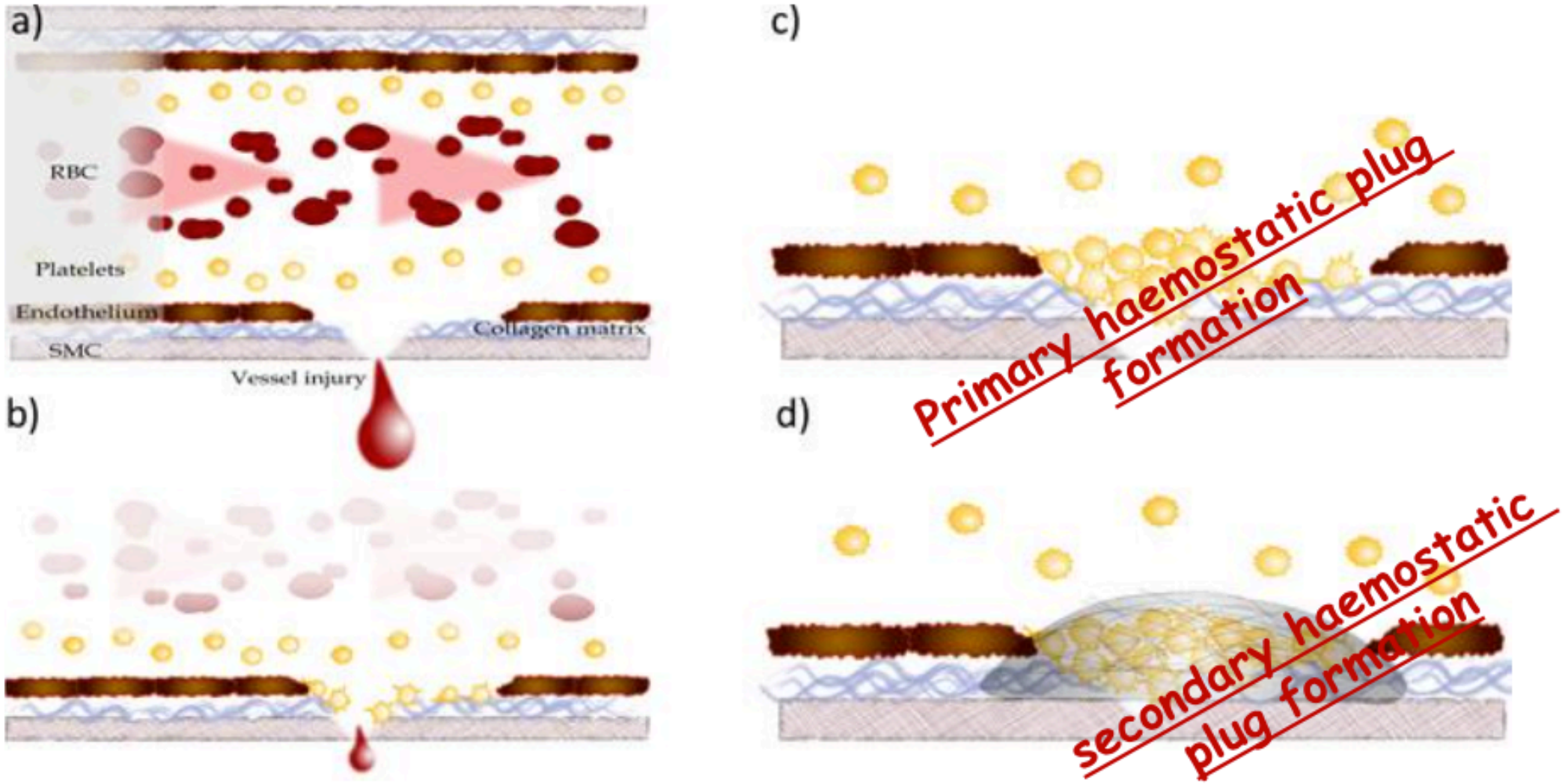
Found in the males' slides

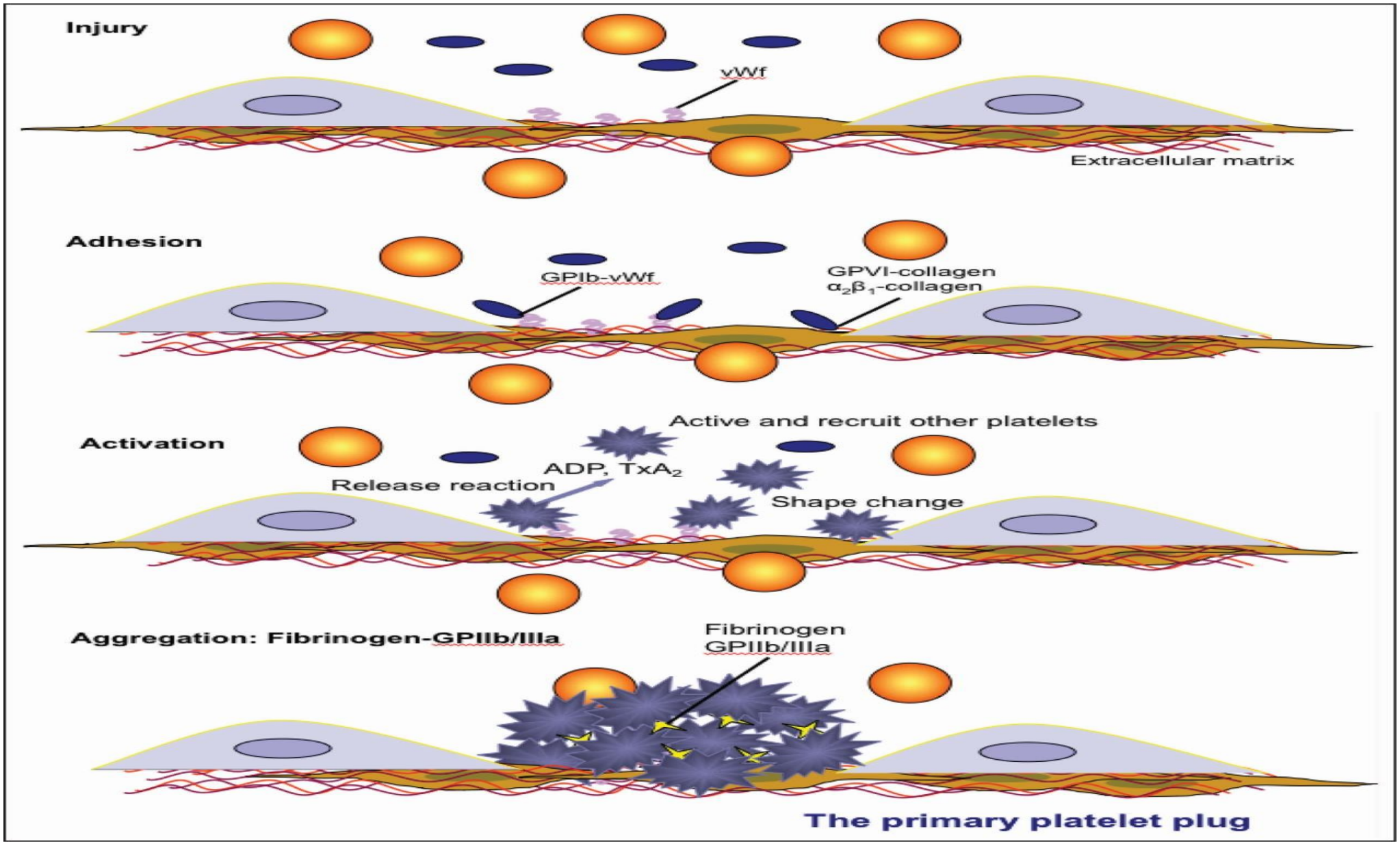
ROLES OF THROMBIN IN HEMOSTASIS



Platelet Haemostatic Plug Formation

Platelet plug formation → primary blood coagulation → secondary



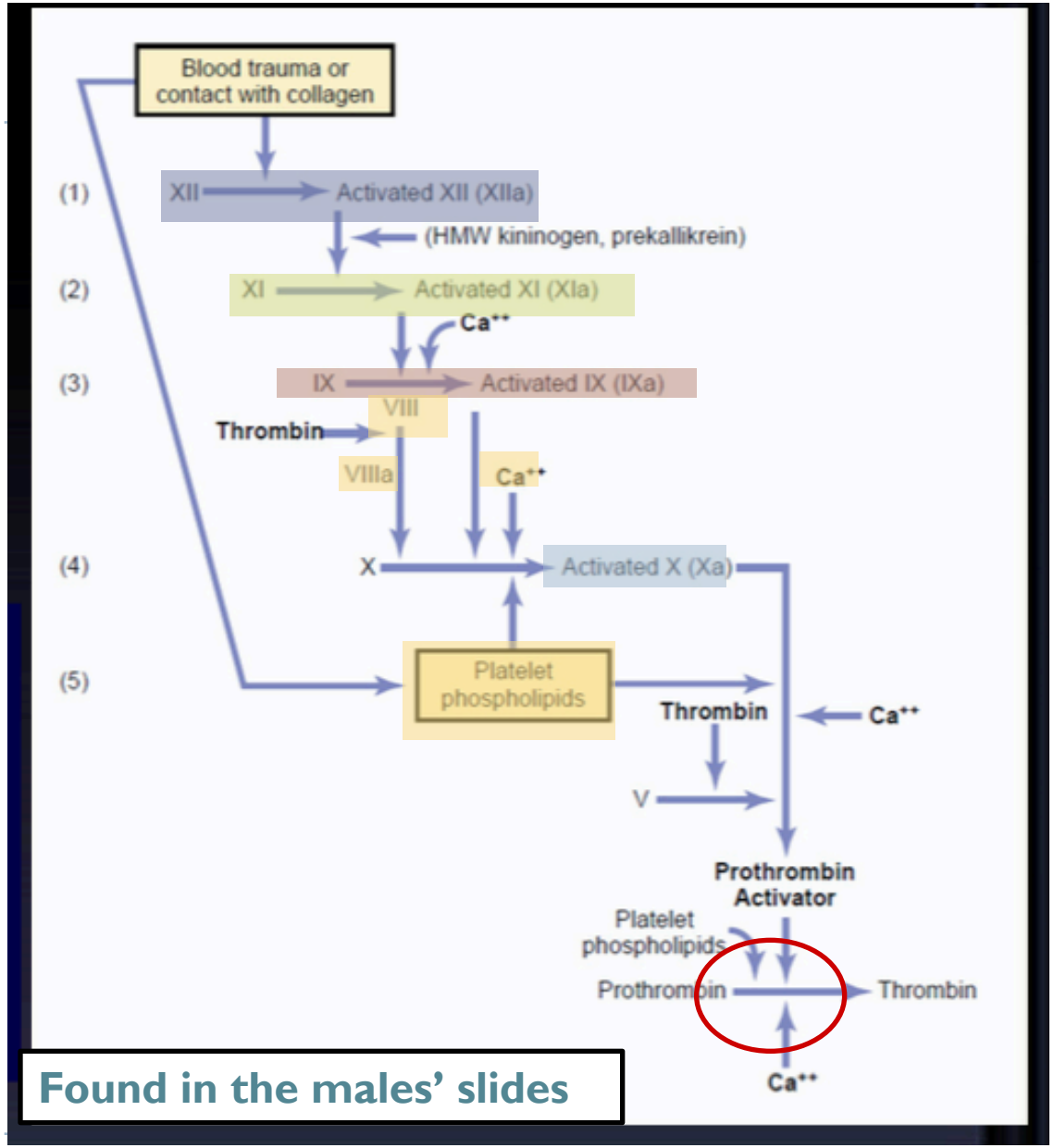

Injury
vWf
Extracellular matrix
Adhesion
GPIb-vWf
GPVI-collagen
$\alpha_2\beta_1$-collagen
Activation
Active and recruit other platelets
ADP, TxA_2
Release reaction
Shape change
Aggregation: Fibrinogen-GPIIb/IIIa
Fibrinogen
GPIIb/IIIa
The primary platelet plug

Intrinsic pathway
(intrinsic mechanism for initiating clotting)

- The **trigger** is the activation of factor XII (12) (Hagman factor). This occurs when blood comes in contact with foreign surface (different from normal) such as glass, injured blood vessel and exposed collagen or endothelium.
- Activated factor (**XIIa**) will activate **XI** (11)
- **XIa** will activate **IX** (9)
- **IXa + VIII (8) + platelet phospholipid + Ca** activate **X**
- Following this step the pathway **is common for both**

Trauma to the blood itself or exposure
of the blood to collagen (from a traumatized blood vessel wall), foreign surface/glass

Note: In the blood coagulation pathway, **thrombin** acts to **convert** factor XI to Xia (activated form) and VIII to VIIIa, V to Va, fibrinogen to fibrin, and XIIIt o XIIIa.



Colours are for your understanding ONLY. ☺

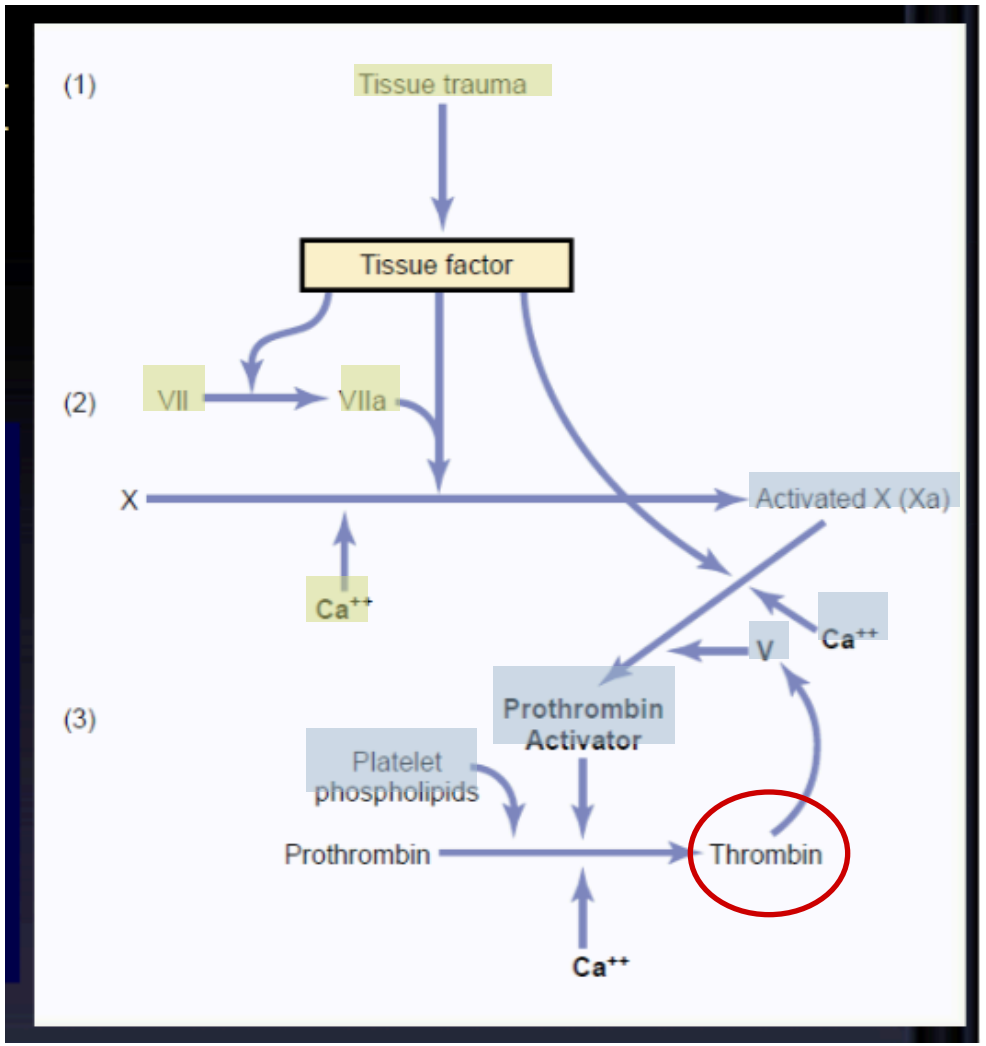
Extrinsic pathway

- • Triggered by material released from damaged tissues (**tissue thromboplastin**)
- • tissue thromboplastin + VII (7) + Ca → activate X **Common pathway**
- • Xa + V +PF3 + Ca (prothrombin activator) it is a

proteolytic enzyme* **activate** prothrombin to change into **thrombin**.

Xa: main factor. V: enhances its activity.

- Thrombin act on fibrinogen and changes it into insoluble thread like fibrin
- Factor **XIII** (13) (fibrin stabilizing factor) + Ca → strong fibrin (strong clot)
- TF or tissue thromboplastin** includes phospholipids from the membranes of the tissue plus a lipoprotein complex that functions mainly as a proteolytic enzyme



. *noun: protease: enzymes that breaks down protein. (Protein here is thromboplastin it broken down.
**Thromboplastin is a plasma protein catalyzes conversion of prothrombin to thrombin

Colours are for your understanding ONLY. ☺

- Activation of Blood Coagulation: (2 ways)
- Intrinsic Pathway (also known as contact activating pathway): all clotting factors present in the blood. (initiated by injury to the blood vessel)
- Extrinsic Pathway (also known as tissue factor pathway): triggered by tissue factor (initiated by trauma to tissue)
- Both activate **Final Common Pathway**

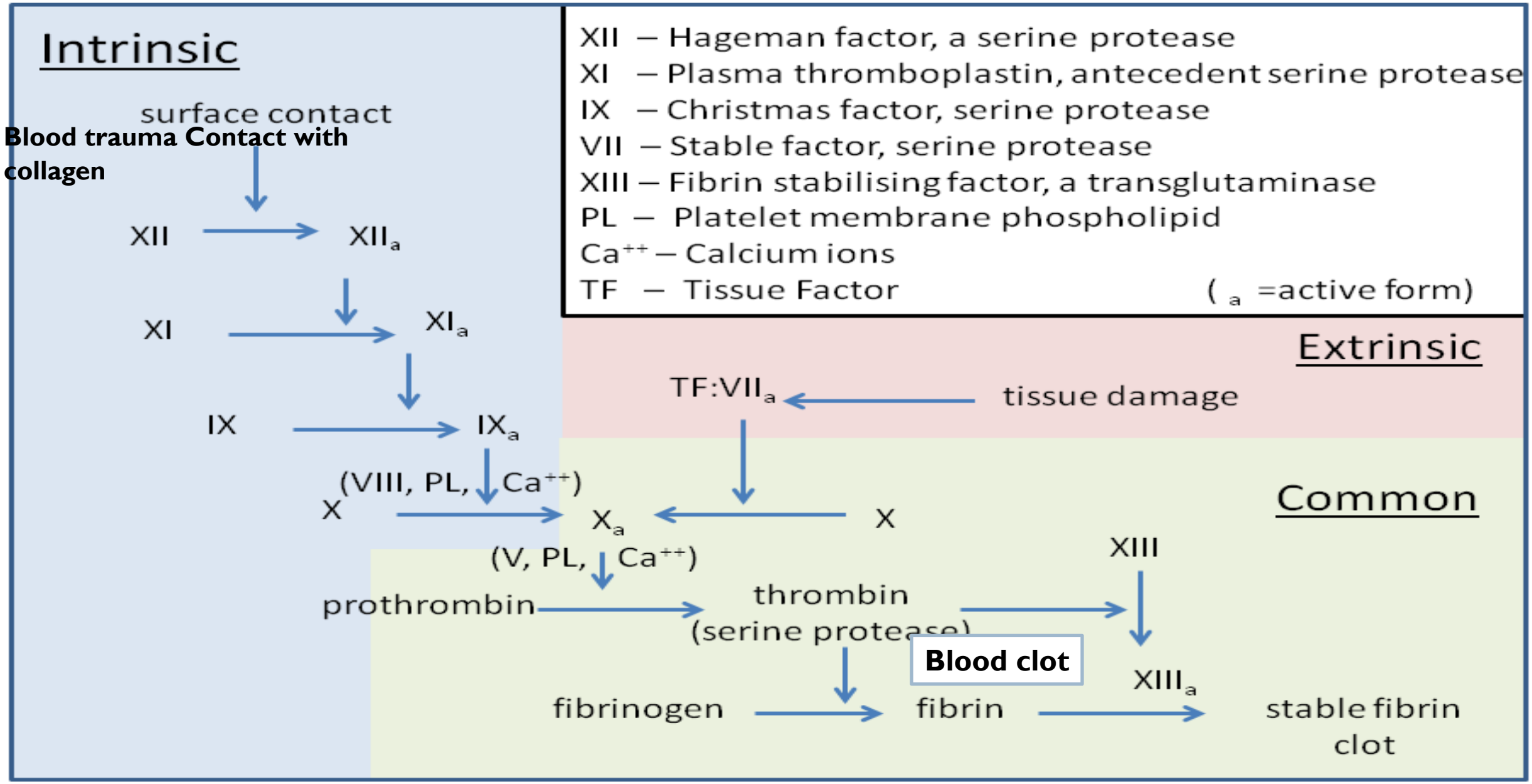
مـــهم

	Intrinsic	Extrinsic
الاختلاف	LONGER	FASTER&SHORTER
التشابه	Both will be stimulated at the SAME TIME. Both will activate (Factor 10) and the result is the formation of Thrombin which then converts Fibrinogen into Fibrin.	

P* = phospholipid from platelets

Intrinsic Pathway	Extrinsic pathway
Contact activation ↓ XII → XIIa ↓ XI → XIa ↓ IX → IXa ↓VIII, Ca,P* X → Xa	Tissue factor ↓ ↓ VII,Ca Xa ← X
Xa ↓ ↓ V, Ca, P* Prothrombin (II) → Thrombin (IIa) ↓ Fibrinogen (I) → Fibrin (soluble) ↓ XIII, Ca insoluble fibrin	

The three pathways that makeup the classical blood coagulation pathway



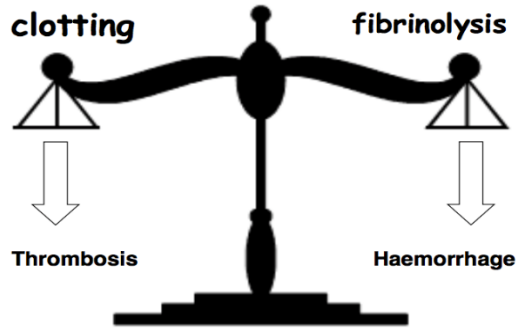
Hemostatic mechanism (cont.)

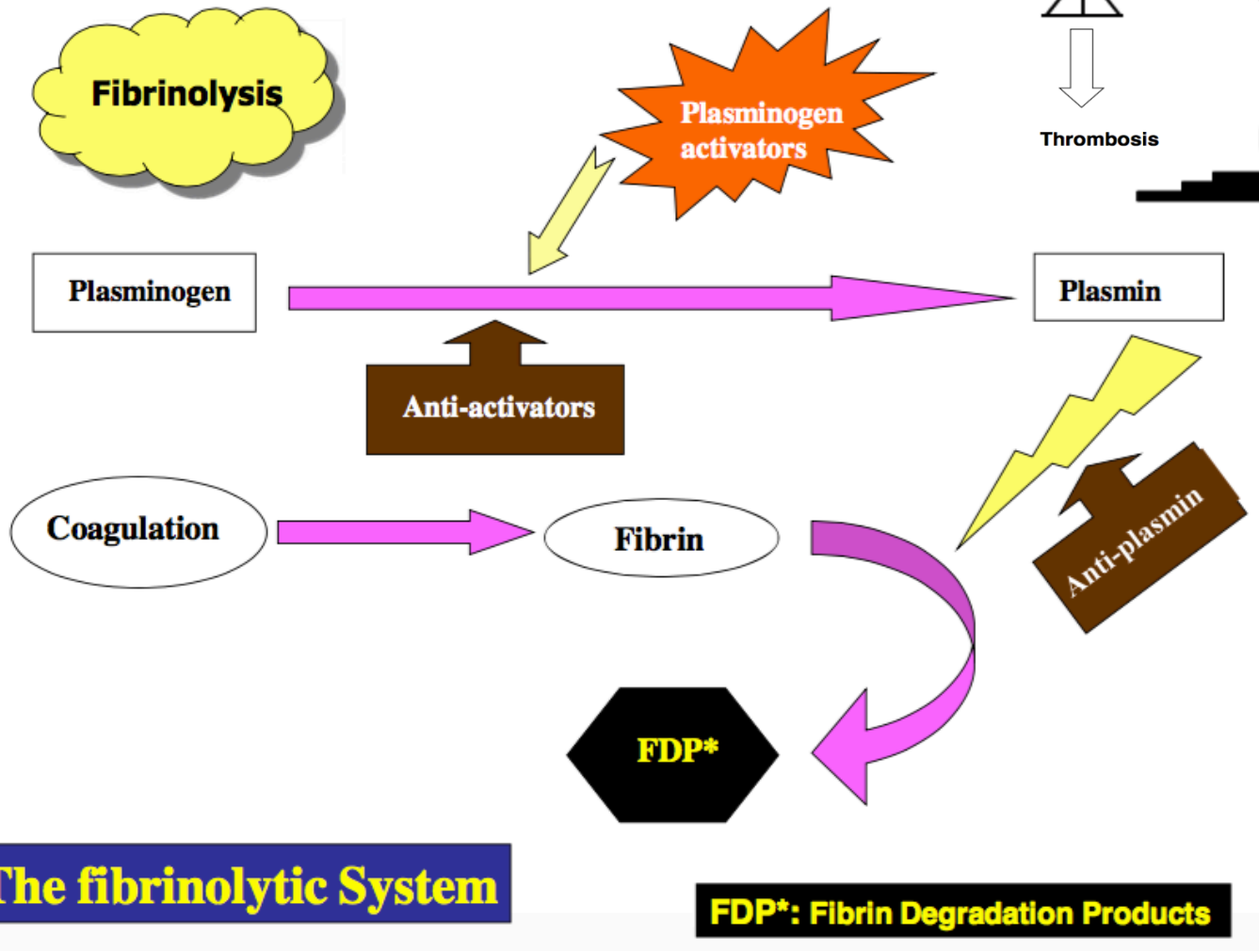
- **4- Fibrinolytic system (Fibrinolysis)**

Formed blood clot can either become **fibrous** or **dissolve**

• Fibrinolysis (dissolving) = **Break down of fibrin** by naturally occurring enzyme **plasmin** therefore prevent intravascular blocking.
• **There is balance between clotting and fibrinolysis**

Examples of imbalance ☹
– Excess clotting → blocking of Blood Vessels
– Excess fibrinolysis → tendency for bleeding

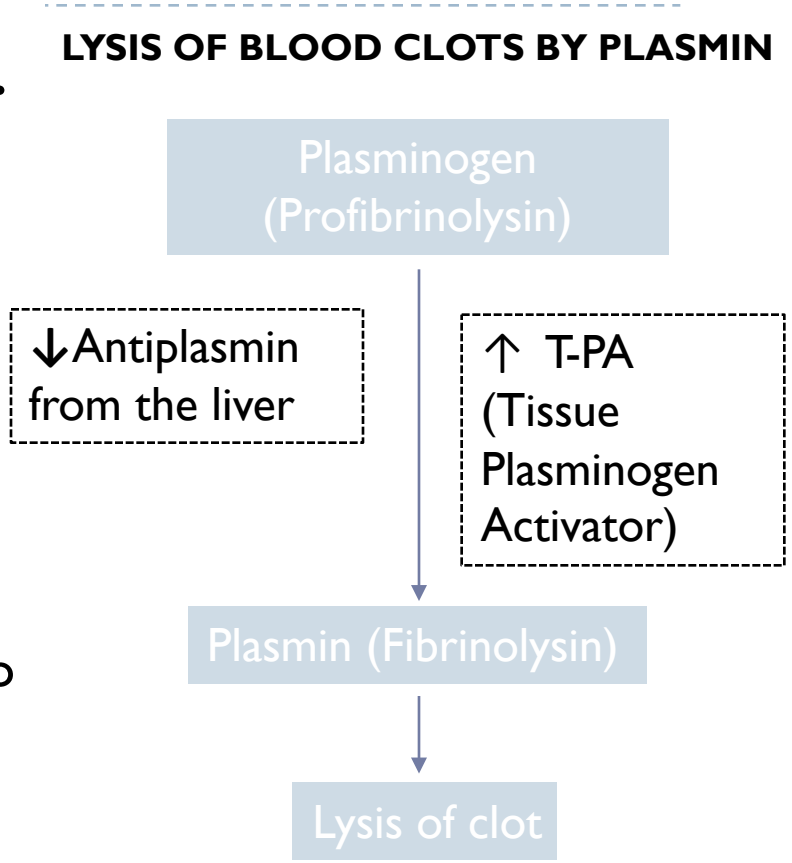


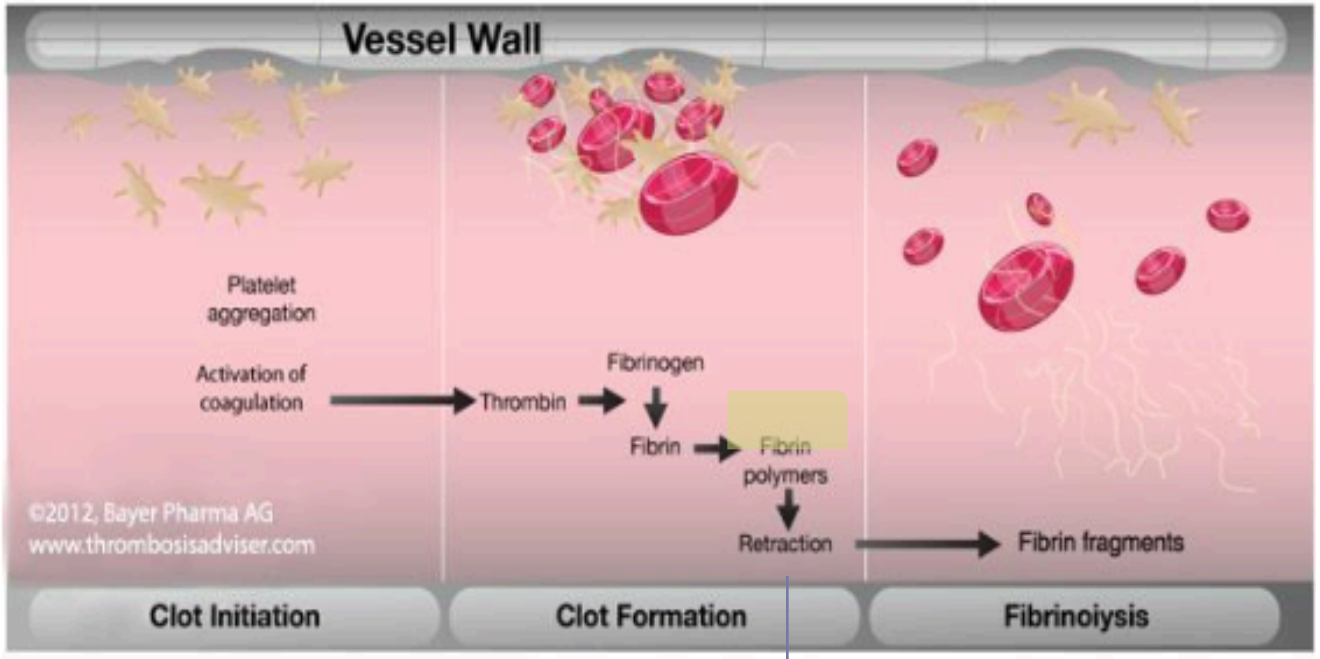


Plasmin

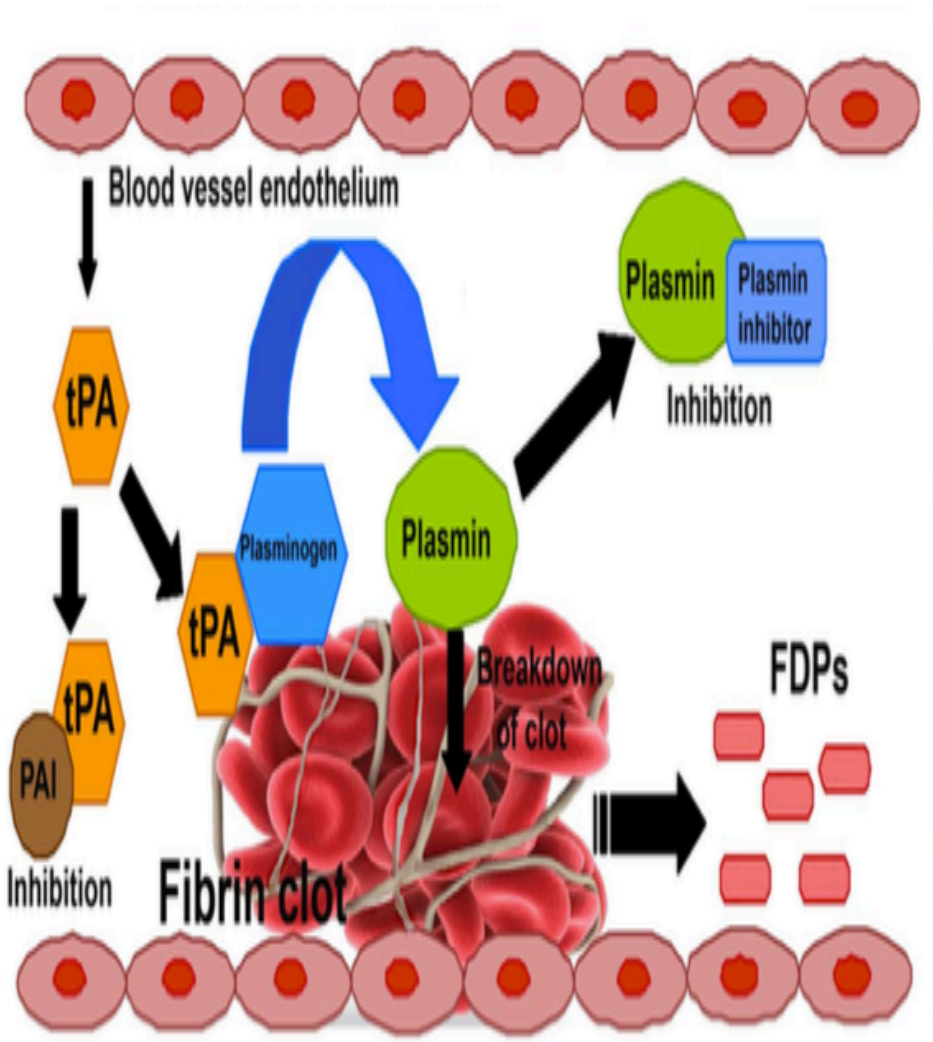
- **Plasmin** is present in the blood in inactive form **plasminogen**
- Plasmin is activated by **tissue plasminogen activators (t-PA) in blood.**
- Plasmin digest intra & extra vascular deposit of Fibrin → **fibrin degradation products (FDP)***
- **Unwanted** ☹ effect of plasmin is the **digestion of clotting factors**

- • **Plasmin is controlled by:**
  – **Plasminogen-Activator Inhibitor (PAI)** – Anti-plasmin from the liver. (inhibits the activation of plasminogen)
- – **Tissue Plasminogen Activator (t-PA)** used to activate plasminogen to dissolve coronary and cerebral clots (activates the activation of plasminogen)
- *As a cut heals, the clotting slows down. Eventually the clot is broken down and dissolved by plasmin. When the clot and fibrin net dissolve, fragments of protein are released into the body. These fragments are fibrin degradation products or FDPs.





We'll talk about retraction in the next slides.



Bleeding disorders (coagulation defects)

Excessive bleeding can result from:

- **Platelet defects:**

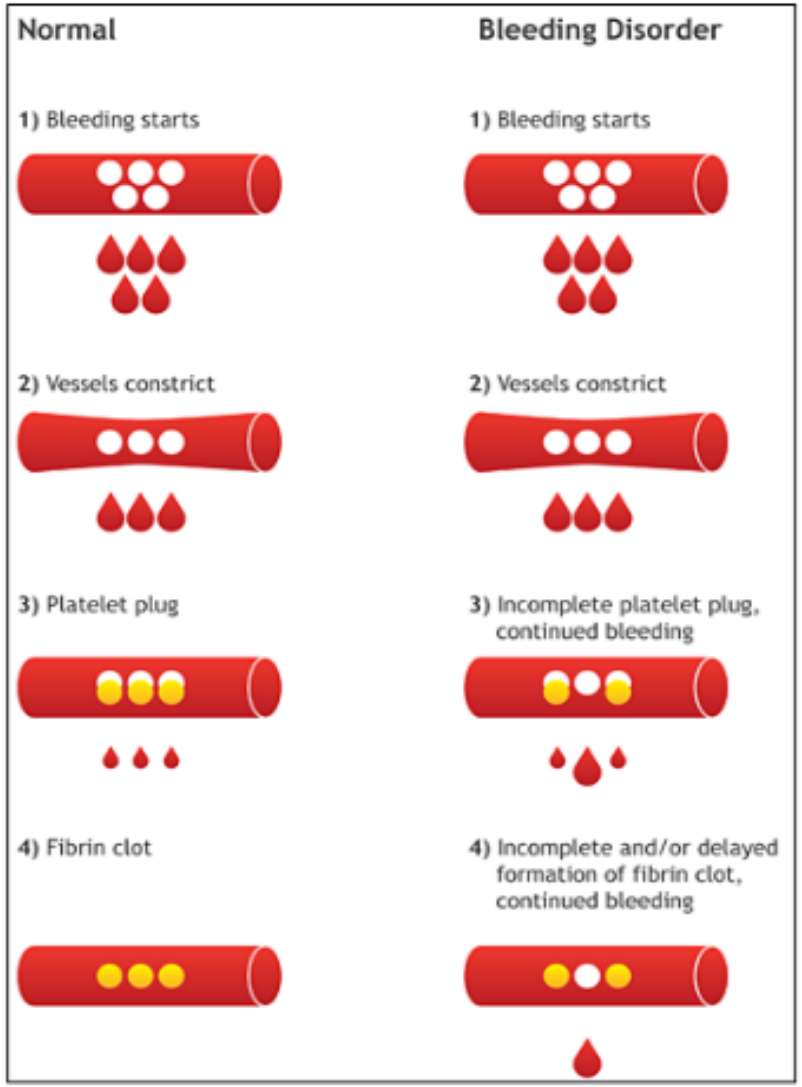
deficiency in number (thrombocytopenia) or defect in function.

- **Coagulation factors defect:**

Deficiency in coagulation factors (e.g. hemophilia).

- **Thrombocytopenia**
  - Low number of platelets

- **Vitamin K deficiency.**



- **Hemophilia**:
  (To be discussed later –slide#31)

- **Vitamin K deficiency**

Prothrombin, Factor VII, Factor IX, & Factor X require vitamin K for their synthesis.

Hepatic (Liver) Disease

(Almost all coagulation factors are synthesized in the liver.)

e.g. Hepatitis, Cirrhosis

- Decreased formation of clotting factors
- Increased clotting time

Only found in males' slides.

CLOT RETRACTION.

When clot retracts (contracts), it expresses most of the fluid (serum) out from the clot within 20-60 min.

Serum cannot clot.

Role of platelets in clot formation & retraction: they are **contractile** (capable of or producing contraction.)

Vitamin K: helps blood clotting by combining with oxygen to activate clotting cascade.

Coagulation inhibition

ROLE OF CALCIUM IONS IN CLOTTING

No Ca++ → No Clotting (Needed in many steps)

Blood samples are prevented from clotting by:

Citrate ions → Deionization of Ca++
Oxalate ions → Precipitate (creation of a solid from solution) of the Ca++

Heparin molecule: is not active by itself but increases the effect of **antithrombin** 100–1000-fold, with the **added effect** of removing activated factors XII, XI, X and IX. (Inhibition of thrombin)

Warfarin (anticoagulant): decrease production of Factors VII, IX and X by liver. (vitamin K-dependent factors) -> Vitamin K antagonist.
EDTA → chelates (binds) calcium ions

NATURAL INTRAVASCULAR ANTICOAGULANTS

- **1. Endothelial Surface Factors**
- The smoothness of endothelial cell surface. (which prevents contact activation of the intrinsic clotting system)
- Glycocalyx Layers (which repels clotting factors and platelets)
- Thrombomodulin protein:
- binds to thrombin
- thrombomodulin-thrombin complex activates a plasma protein, protein C, that acts as an anticoagulant by inactivating Factors V and VIII
- Increases the formation of plasmin

- **2. Anti-thrombin action of <u>Fibrin</u> and Antithrombin III**
- 85-90 % Thrombin binds with Fibrin
- 10-15 % Thrombin binds with Antithrombin III
- **Antithrombin III is a circulating protease blocking clot factors** (It blocks your blood clotting mechanism by turning off the major clotting protein "thrombin.")

3. Heparin

is a highly negatively (-) charged conjugated polysaccharide

Function: as anticoagulant by increasing the effectiveness of Antithrombin III.

Produced by:

1. Mast cells
2. Basophil cells

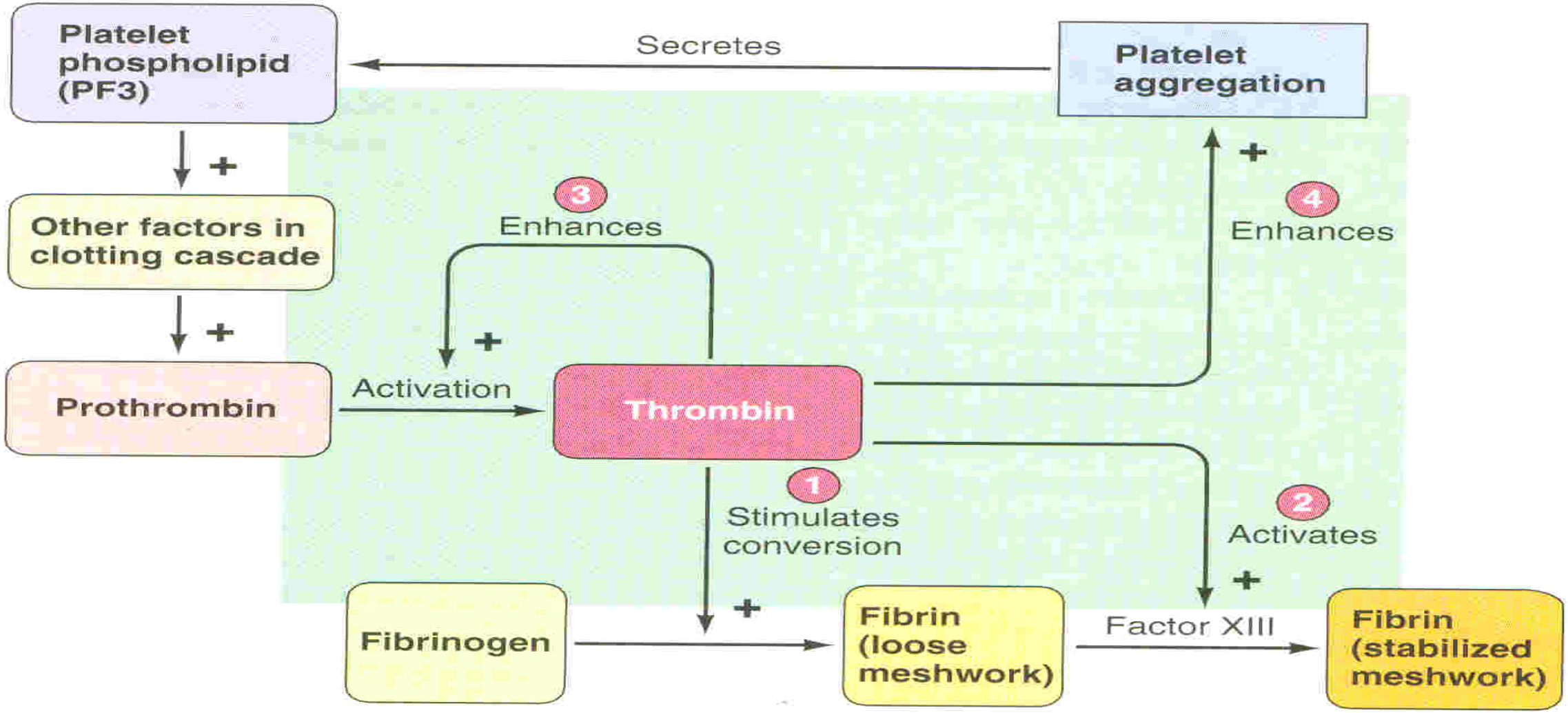
Most widely used anticoagulant clinically e.g. in stroke

4. Alpha2 – Macrogobulin

Acts as a binding agent for several coagulation factors.

Only found in males' slides.

Role of Thrombin in Hemostasis



Only found in males' slides.

THROMBOCYTOPENIA

Diagnosis:

Disorder in which there is a decrease of platelets (PLT or thrombocytes.)

- Platelet count up to 50,000 ul→ thrombocytopenia that requires emergency care.
- Bleeding time increases.
- Less than 10,000 → Fatal.

ETIOLOGY:

Decreased production of platelets due to:

- Aplastic anemia
- Leukemia
- Drugs
- Infections (HIV, Measles)

Increased destruction due to:

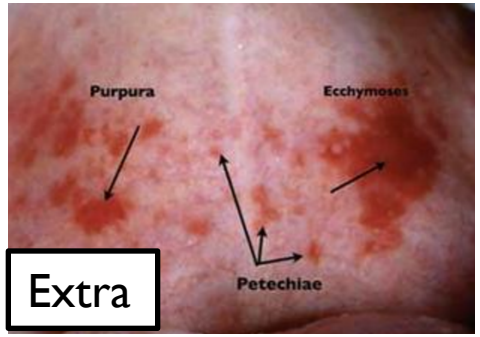
ITP (immune thrombocytopenia purpura)
Drugs
Infections

Clinical Features

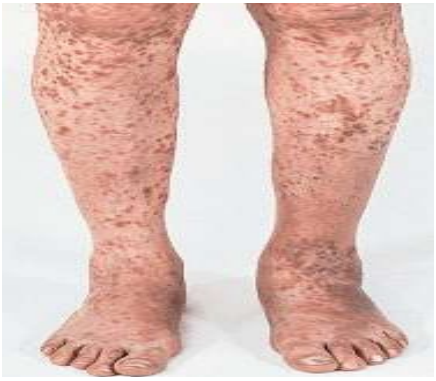
- Easy brusability
- Epistaxis
- Gum bleeding
- Hemorrhage after minor trauma
- Petechiae/Ecchumosis

Rx

- Rx of the underlying cause
- PLT concentrates
- Fresh whole blood transfusion
- Splenectomy



Extra



Hemophilia

(Rare bleeding disorder in which the blood does not clot normally)

- ↑ bleeding tendency.
- Genetic disorders X-linked disorder.
- Transmitted by female chromosome as recessive trait.
- Occurs exclusively (affects) in males
- Females are carriers

Clinical features:

Easy bruising, massive bleeding after trauma or operation, hemorrhages in joints.

-Rx

Hemophilia A: (Classic)

85% of cases

Deficiency (and therefore we inject) of factor VIII

(hemophilia A , 1/10,000)

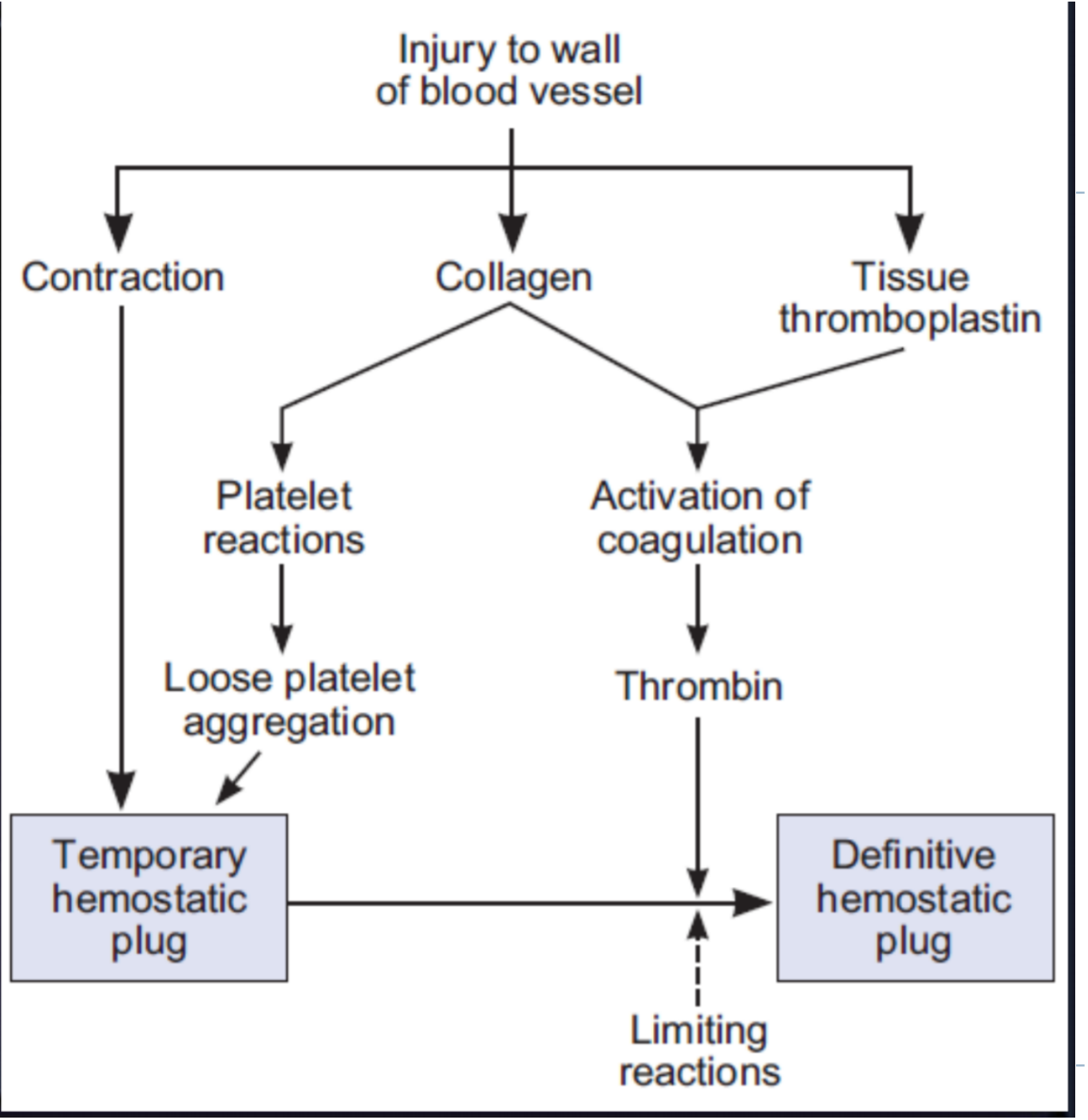
Hemophilia B:

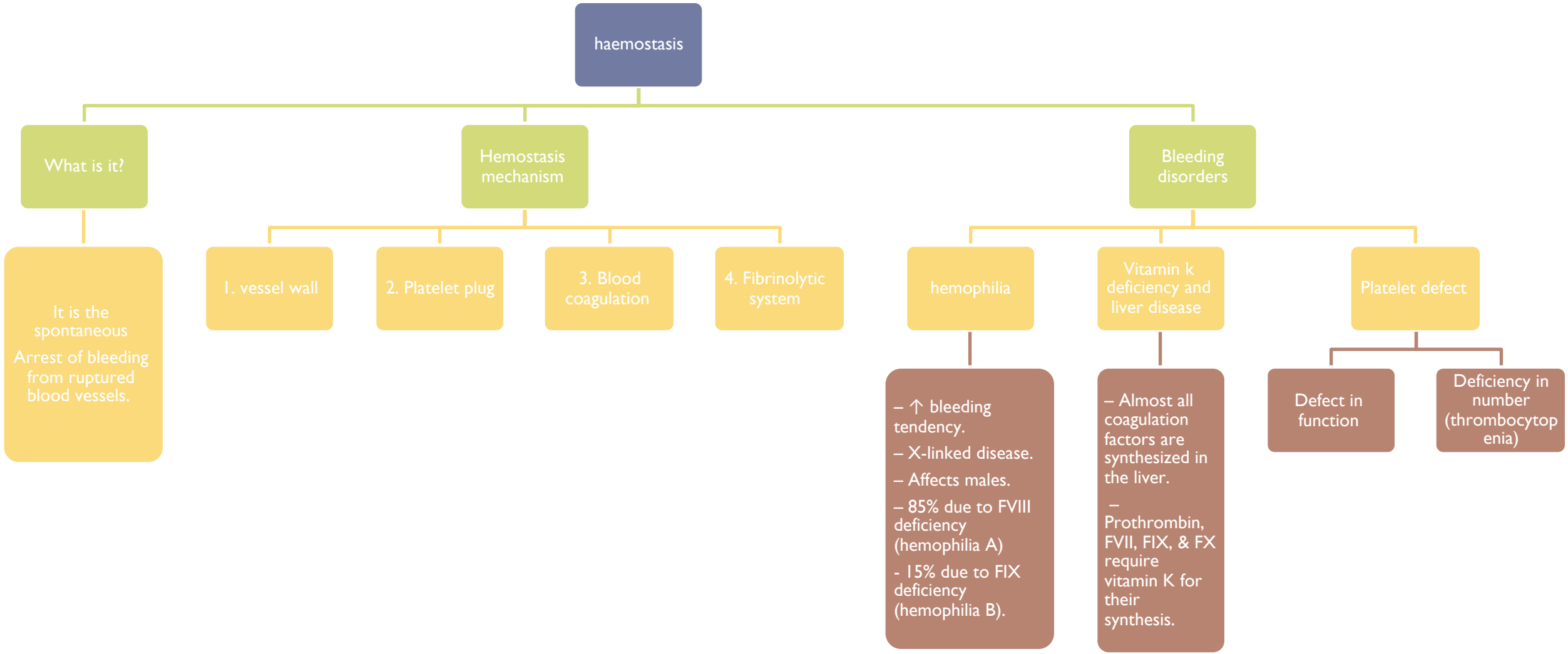
15% of cases

Deficiency (and therefore injection) of factor IX

(hemophilia B, 1/100,000)

Summary of reactions involved in hemostasis.




haemostasis
What is it?
It is the spontaneous
Arrest of bleeding from ruptured blood vessels.
Hemostasis mechanism
1. vessel wall
2. Platelet plug
3. Blood coagulation
4. Fibrinolytic system
Bleeding disorders
hemophilia
– ↑ bleeding tendency.
– X-linked disease.
– Affects males.
– 85% due to FVIII deficiency (hemophilia A)
- 15% due to FIX deficiency (hemophilia B).
Vitamin k deficiency and liver disease
– Almost all coagulation factors are synthesized in the liver.
– Prothrombin, FVII, FIX, & FX require vitamin K for their synthesis.
Platelet defect
Defect in function
Deficiency in number (thrombocytopenia)

Hemostasis Mechanism

1. vessel wall

- Components of blood vessel
  - Endothelial cells
  - Smooth muscle
  - Collagen
- Localized vasoconstriction
  - It happen immediately after injury.
- Mechanism
  - 2. Nervous factors
  - 3. Local release of thromboxane A2 and 5HT by platelets
  - 1. Release of adrenaline → To help vessel contraction

2. Platelets (thrombocytes)

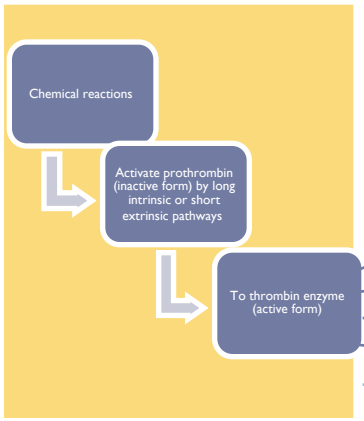
- Structure and shape
  - Biconvex disc
  - Life span 8-12days
  - Do not have nucleus
  - Contain high calcium & rich in ADP.
- What is it ?
  - It is the fragments of megakaryocytes
- Platelet **Receptors**
  - -GP Ia, GP VI Collagen
  - -GP Ib-IX-V (vW Factor)
  - -(TPα) TXA2
  - -GP IIb-IIIa (Fibrinogen, vWF)
  - -(P2Y12) ADP
- Regulation of platelets
  - By thrombo p-oietin which is formed in liver
- function
  - Adhesion
  - Activation
  - Aggregation
  - Secretion
- Steps of formation
  1. Stem cell
  2. megakayoblast
  3. megakaryocyte
  4. platelets
- Site of formation
  - In bone morrow

Hemostasis Mechanism

3. Blood coagulation(clot formation)

What is it

- it is a series of chemical reactions leading to the formation of a blood clot.



- change fibrogen to fibrin
- Activate factor V "5"
- Stimulate platelets to release ADP and thromboxane A2
- Essential in platelet morphological changes to form primary plug

Clotting factors

Circulate in plasma in inactive state

I (1) Fibrinoge

II (2) Prothrombin

VIII (8) Antihemophilic factor A

IX (9) Antihemophilic factor B

XII (12) Hagman factor

XIII (13) Fibrin stablizing factors

Coagulation pathways

Extrinsic pathway

tissue thromboplastin + VII + Ca will activate X

Intrinsic pathway

activation of factor XII "12" (XIIa) will activate XI

- XIa will activate IX
- IXa + VIII + platelet phospholipid + Ca activate X

Common pathway :

- Xa + V +PF3 + Ca will activate prothrombin to give thrombin enzyme
- Thrombin act on fibrinogen and change it to give fibrin (insoluble thread)
- Factor XIII + Ca will make the fibrin strong (strong clot)

4. Fibrinolytic system (fibrinolysis)

What is it?

It is the break down of fibrin by naturally occurring enzyme plasmin therefore prevent intravascular blocking

Steps of fibrinolysis

- Plasmin is present in the blood in inactive form plasminogen
- Plasmin is activated by tissue plasminogen activators (t-PA)
- Plasmin digest intra & extra vascular deposit of Fibrin to give fibrin degradation products (FDP)

Online Quiz

https://www.onlinequizcreator.com/hemostasis/quiz-221751

Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

The Physiology 436 Team:

Lina Alwakeel
Rana Barasain
Heba Alnasser
Munira Aldofayan
Sara Alshamrani
Sundus Alhawamda
Ruba Ali
Rehab Alanazi
Norah Alshabib
Nouf Alaqeeli
Buthaina Almajed
Alaa Alaqeel

Fahad Al Fayez
Ibrahim Al Deeri
Hassan Al Shammari
Abdullah Al Otaibi
Abdullah Al Subhi
Ali Al Subaei
Omar Al Babteen
Foad Fathi
Faisal Al Fawaz
Muhammad Al Aayed
Muhammad Al Mutlaq
Nasser Abu Dujeen
Waleed Al Asqah

Team Leaders:

Qaiss Almuhaideb
Lulwah Alshiha

Contact us:

Physiology436@gmail.com
@Physiology436