

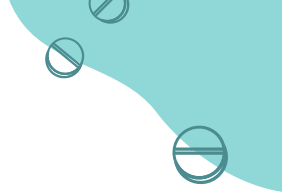
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Color index:

- Main Text
- **IMPORTANT**
- *Girls' slides only*
- *Boys' slides only*
- Extra Info
- Drs Notes

Haemostasis 2

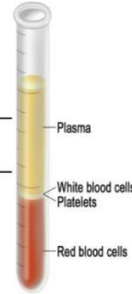


Hemostatic Mechanisms:

- Vessel wall
- Platelet
- **Blood coagulation**
- Fibrinolytic system

Clotting Factors

Factors	Names
I	Fibrinogen
II	Prothrombin
III	Thromboplastin
IV	Calcium
V	Labile factor
VII	Stable factor
VIII	Antihemophilic factor A
IX	Antihemophilic factor B
X	Stuart-Power factor
XI	Plasma thromboplastin antecedent (PTA)
XII	Hagman factor
XIII	Fibrin stabilizing factors



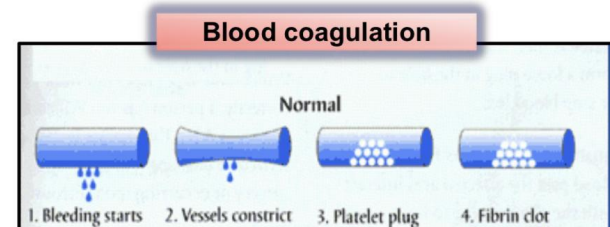
Circulate in plasma in inactive state

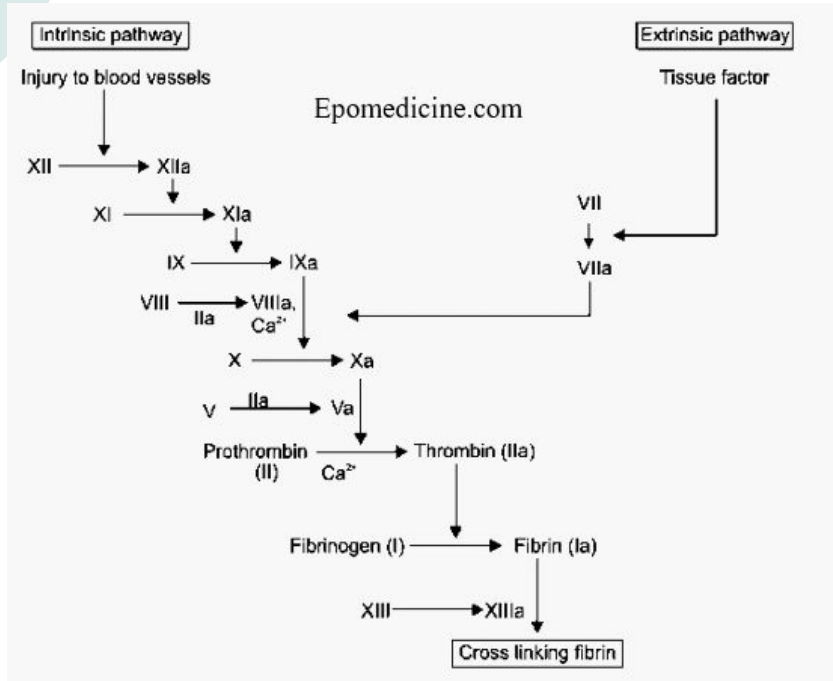
Role in Calcium ions in clotting:

No Ca^{++} means no clotting.

Blood samples are prevented from clotting by adding:

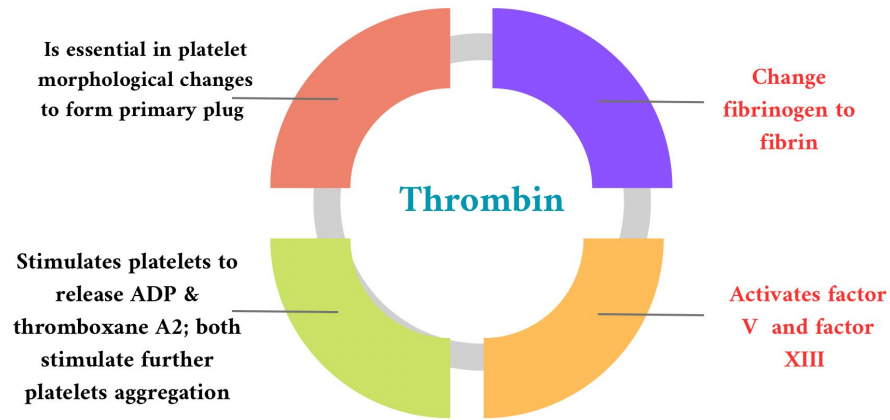
Citrate ions \rightarrow Deionization of Ca^{++}
 Oxalate ions \rightarrow ppt the Ca^{++}



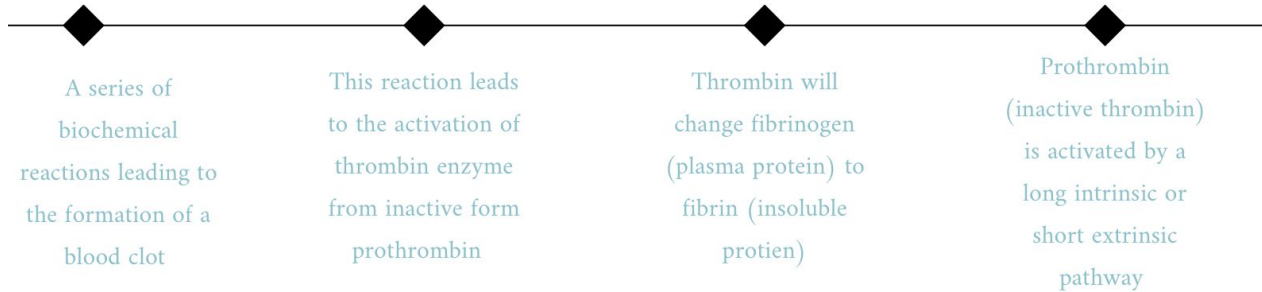


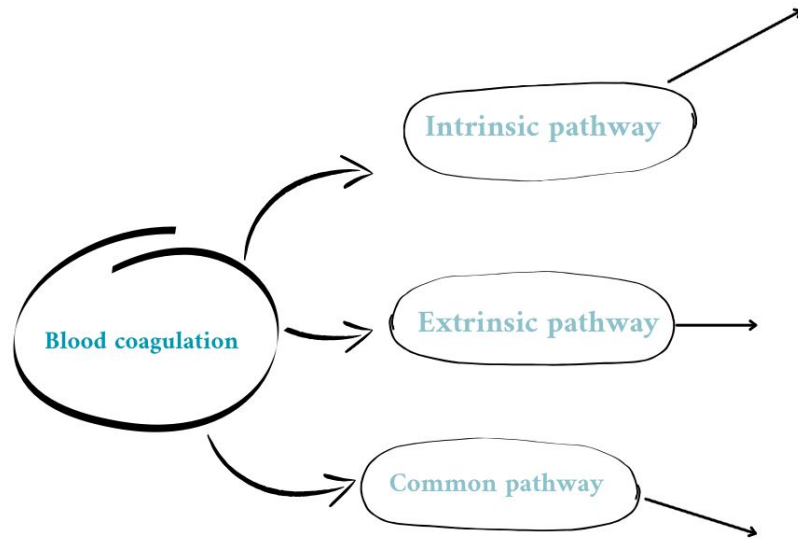
Intrinsic pathway	Extrinsic pathway
Initiated by injury to the blood vessels	Initiated by trauma to the tissue
Both activated by factor x	





Blood coagulation





- The trigger is the activation of factor XII by contact with foreign surfac, injured, blood vessel and glass.
 - Activated factor (XIIa) -> activate XI.
 - XI -> activate IX.
 - IXa + VIII + platelet phospholipid + ca -> activate X .
 - All clotting factor present in the blood.
-
- Triggered by material released from damaged tissue (tissue thromboplastin).
 - tissue thrpmoplastin + VII + Ca -> activate X.
 - triggered by tissue factor.
-
- Xa + V + PF3 +Ca (prothrombin activator) it is a proteolytic enzyme activate prothrombin-> thrombin.
 - Thrombin act on fibrinogen-> insoluble thread like fibrin.
 - Factor XIII + Ca -> strong fibrin (strong clot).

In male slide only

	Extrinsic pathway	Intrinsic pathway
Duration	Rapid	Slow
-	Weaker than the intrinsic pathway	More extensive. Forms more fibrin threads
Start by	Factor III (Thromboplastin or tissue factor)	Factor XII (contact factor)
Occurs	Only invivo	Both invivo and invitro
Tested by	Prothrombin time (PT)	Activated partial thromboplastin time (APTT)



Regulation of coagulation:

Antithrombin(**Heparin**)

- Inhibit thrombin and other enzymes

Protein C

- Degrade activated factor V and VIII

Protein S

- Cofactor for protein C

Tissue factor pathway inhibitor(TFPI)

- Inhibit the extrinsic system by inhibiting factor VIIa

Smooth vascular endothelium

Thus there is no activation of factor XII or platelets

The fibrinolytic system



Fibrinolytic System:



Definition:

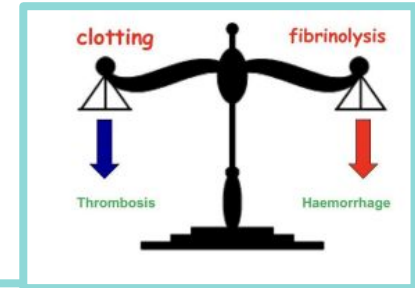
it is the breakdown of **fibrin** by naturally occurring enzyme **plasmin** therefore prevent intravascular blocking.

- 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**.

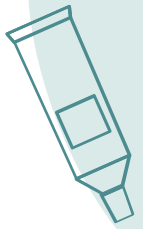
Plasmin is controlled by: -

- . Antiplasmin from the liver.
- . Plasminogen Activator Inhibitor (PAI).

Uses of Plasmin: - Tissue Plasminogen Activator (t-PA) used to activate plasminogen to dissolve coronary clots. There is balance between clotting and fibrinolysis: - Excess clotting → blocking of Blood Vessel
Excess fibrinolysis → tendency for bleeding



Hemostatic function tests :



	Test for	Prolonged in
Bleeding time	Platelets function	Thrombocytopenia Thrombocytoasthenia
Coagulation (clotting time)	Coagulation cascade	All disorders of coagulation (Hemophilia – Vitamin K deficiency -
Prothrombin time (PT)	Extrinsic pathway	Abnormalities of the extrinsic pathway (Vitamin K deficiency)
Activated partial prothrombin time (APTT)	Intrinsic pathway	Abnormalities of the intrinsic pathway (Hemophilia)



Bleeding disorders:

Vitamin K
deficiency

Prothrombin, **FVII, FIX, & FX** require **vitamin K** for their synthesis

Platelet defect

deficiency in number or defect in function.
(**thrombocytopenia**)

Coagulation factors defect

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

Liver disease

almost all coagulation factors are synthesized in the liver.(E.g.hepatitis).

X-linked disease (genetic disorder)
Affects males, females are only carriers. -
Increase bleeding tendency.
- It has (3) types

Hemophilia

(A): Classic hemophilia, **85%cases, due to VII deficiency**

(B): **15% cases, due to IX deficiency**

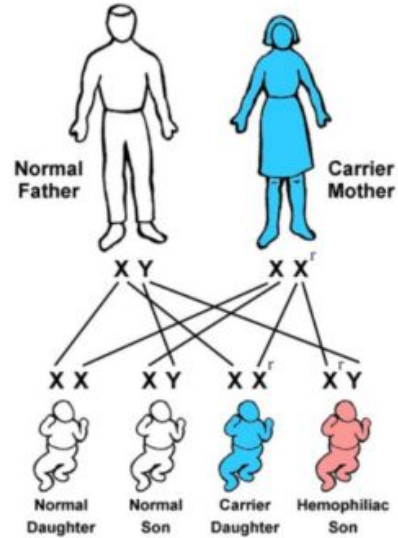
© due to absence of **factor XI**
● Clinical features: Easy bruising, massive bleeding after trauma or operation, hemorrhages in joints





Male slides only

Inheritance of hemophilia :





THROMBOCYTOPENIA



PLT count upto 50,000 ul ❖ Less than 10,000 → Fatal

❖ Etiology:

A) Decreased production

1. Aplastic anemia
2. Leukemia
3. Drugs
4. Infections (HIV, Measles)

B) Increased destruction

1. ITP.
2. Drugs.
3. Infections

❖ Clinical Features:

1. Easy bruisability
2. Epistaxis
3. Gum bleeding
4. Hemorrhage after minor trauma.
5. Petechiae/Ecchymosis.

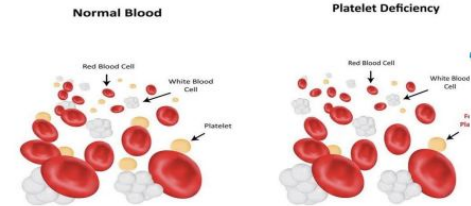
❖ Diagnosis:

1. Platelets decrease.
2. Blood trauma increase

Clinical note: The VWF* has a dual role in hemostasis:

First: it promotes platelet adhesion to thrombogenic surfaces as well as platelet to- platelet cohesion during thrombus formation.

second: it is the carrier for FVIII in plasma. FVIII acts as a cofactor to accelerate the activation of factor X by activated factor IX in the coagulation






Anticoagulants :

Heparin:

- Liver, lungs, masts cells and basophils.
 - Direct antithrombin.
 - Prevent the conversion of prothrombin to thrombin.
- Injection only.
- 6-8 Hours. Warfarin

- 
- Almost all coagulation factors are synthesis
In the liver .
 - Suppresses the synthesis of prothrombin, FVII, FIX and FX (Vitamin K dependent factors).
 - Orally.
 - 48 Hours





MCQs

1- Thrombin activates factors:

A- I , V , IX , XI

B- I , X , XI , XIII

C- III , IX , VII

D- I , V , VIII ,XIII

2- Both intrinsic pathways activate clotting factor number:

A- X

B-XII

C- VII

D- A & C

3- Wil change fibrinogen to fibrin:

A- Thrombin

B- Serotonin

C- Calcium

D- TXA2



3-A
2-A
1-D



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