Hemostasis

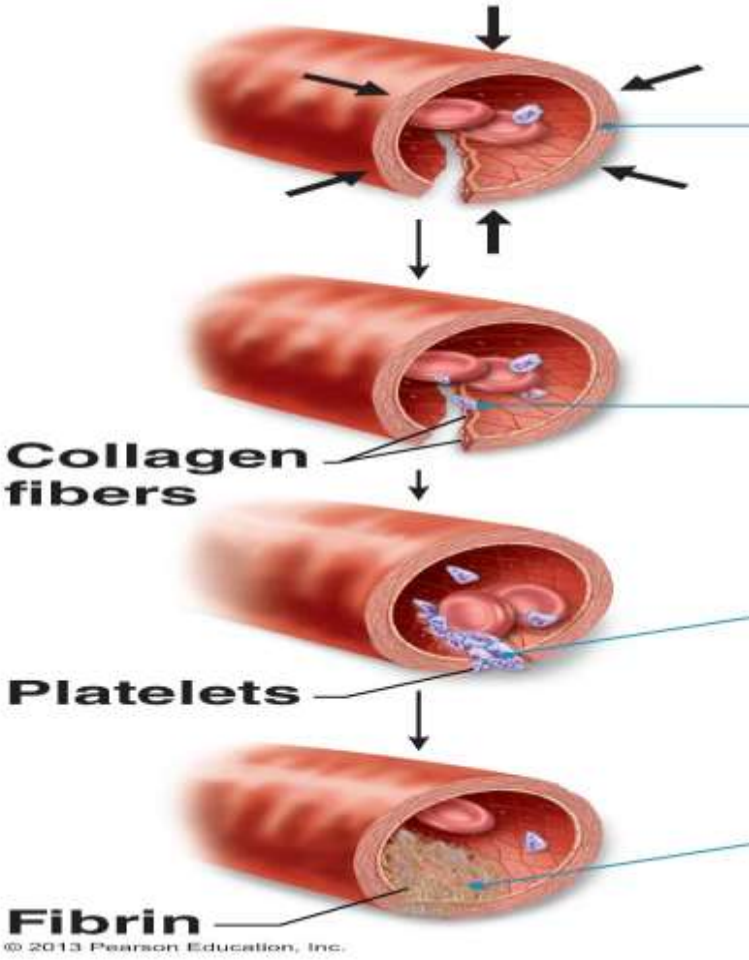
Stoppage of bleeding after injury

Dr.Abeer Shoman

Hemostasis is done by:

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

Hemostasis



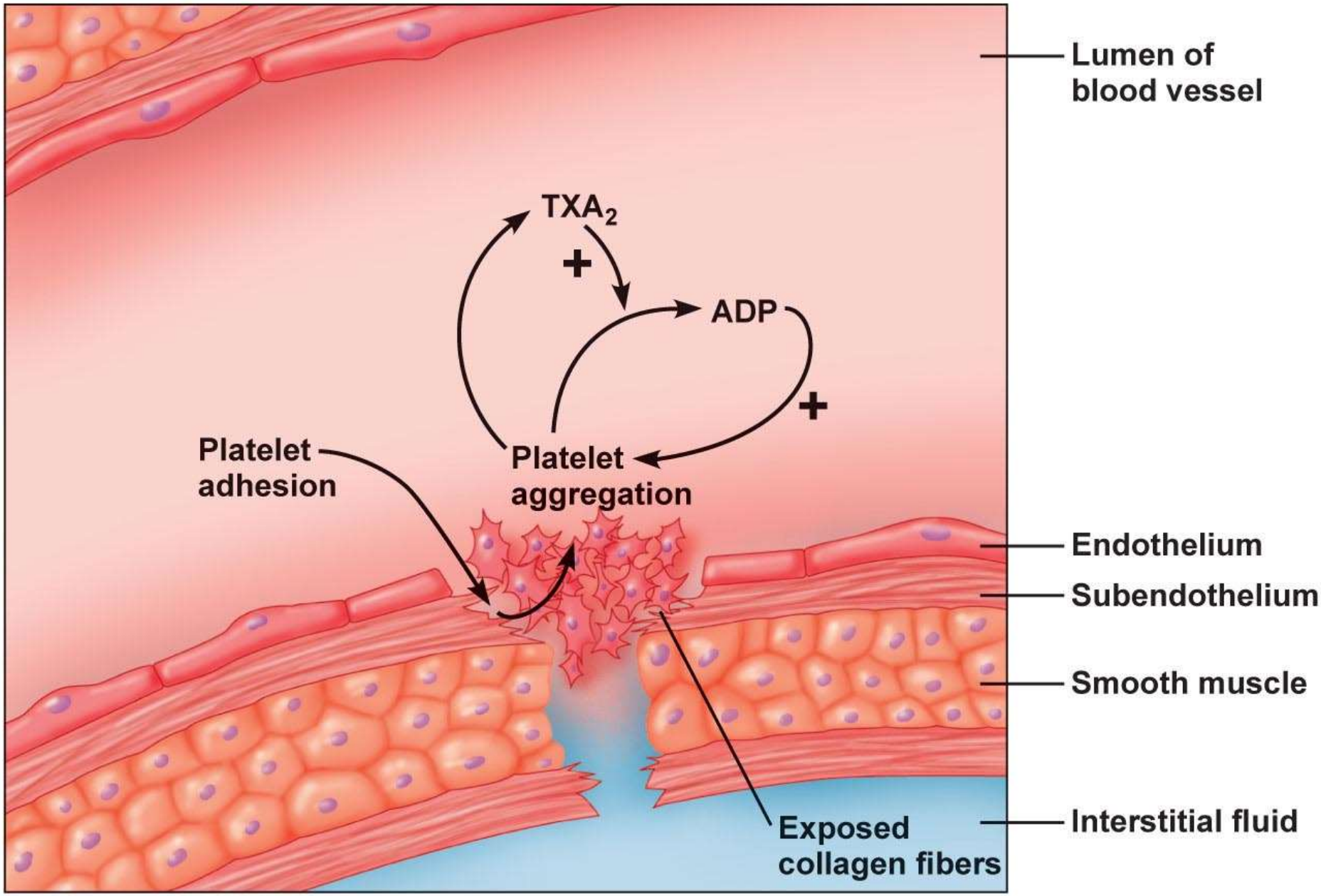
Step ① Vascular spasm
- Smooth muscle contracts, causing vasoconstriction.

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 A_2 from platelets.



(a) Damaged blood vessel endothelium

© 2011 Pearson Education, Inc.

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.

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.

Thromboxane A_2 is synthesized then released from activated platelets.

4- Platelet aggregation

The released ADP, thromboxane A_2, 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

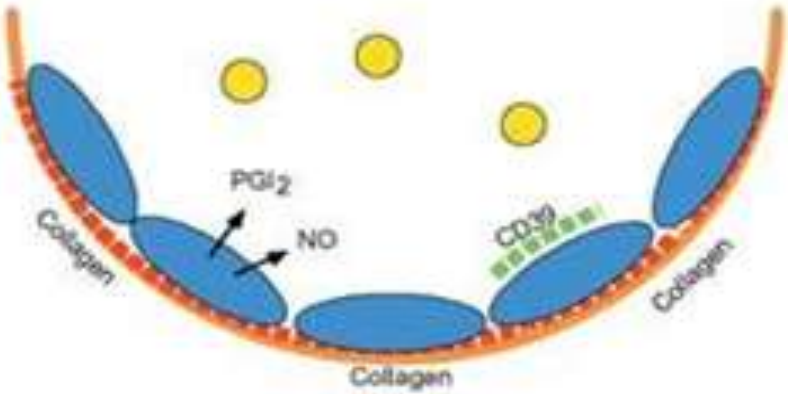
Irreversible fusion of aggregated platelets at the site of injury is caused by:

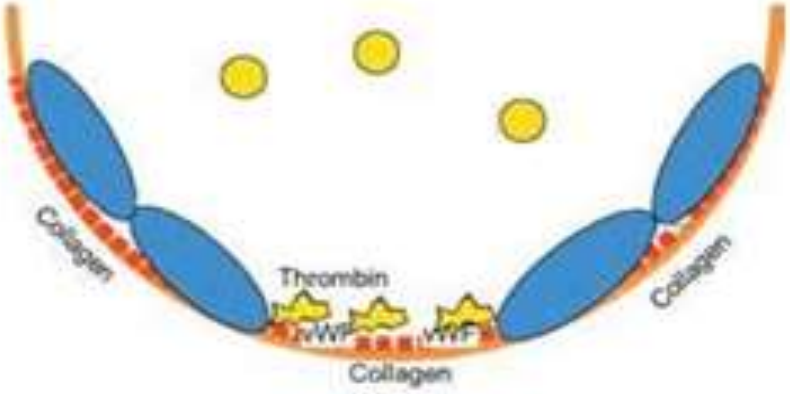
a- High concentration of ADP.

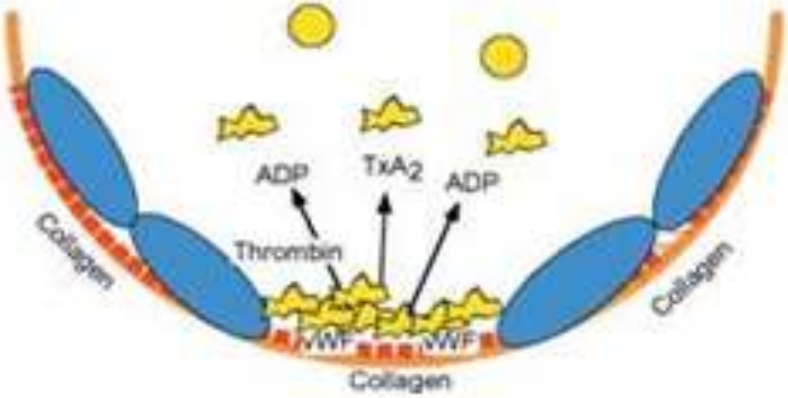
b- Thrombin.

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 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

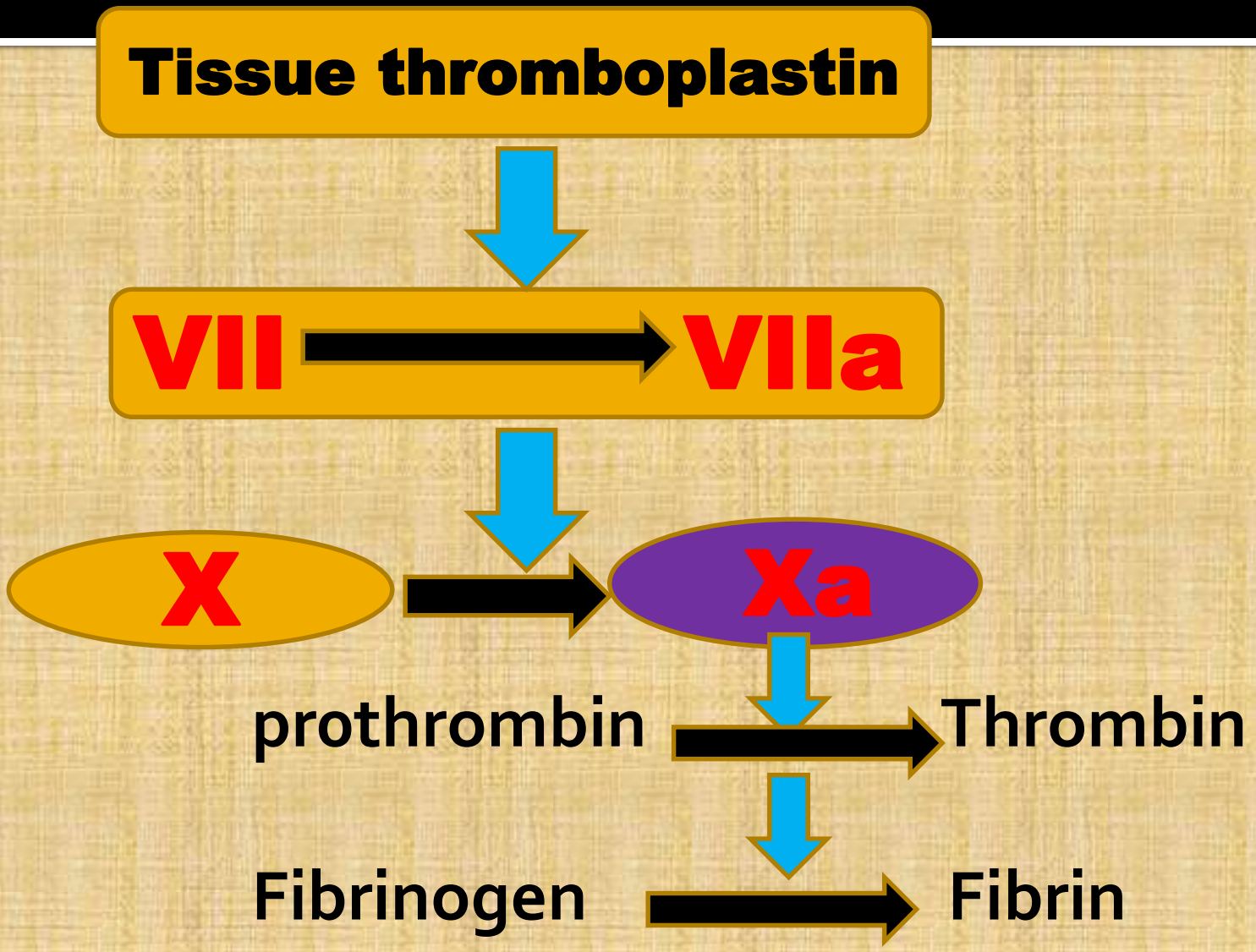
1] Extrinsic pathway

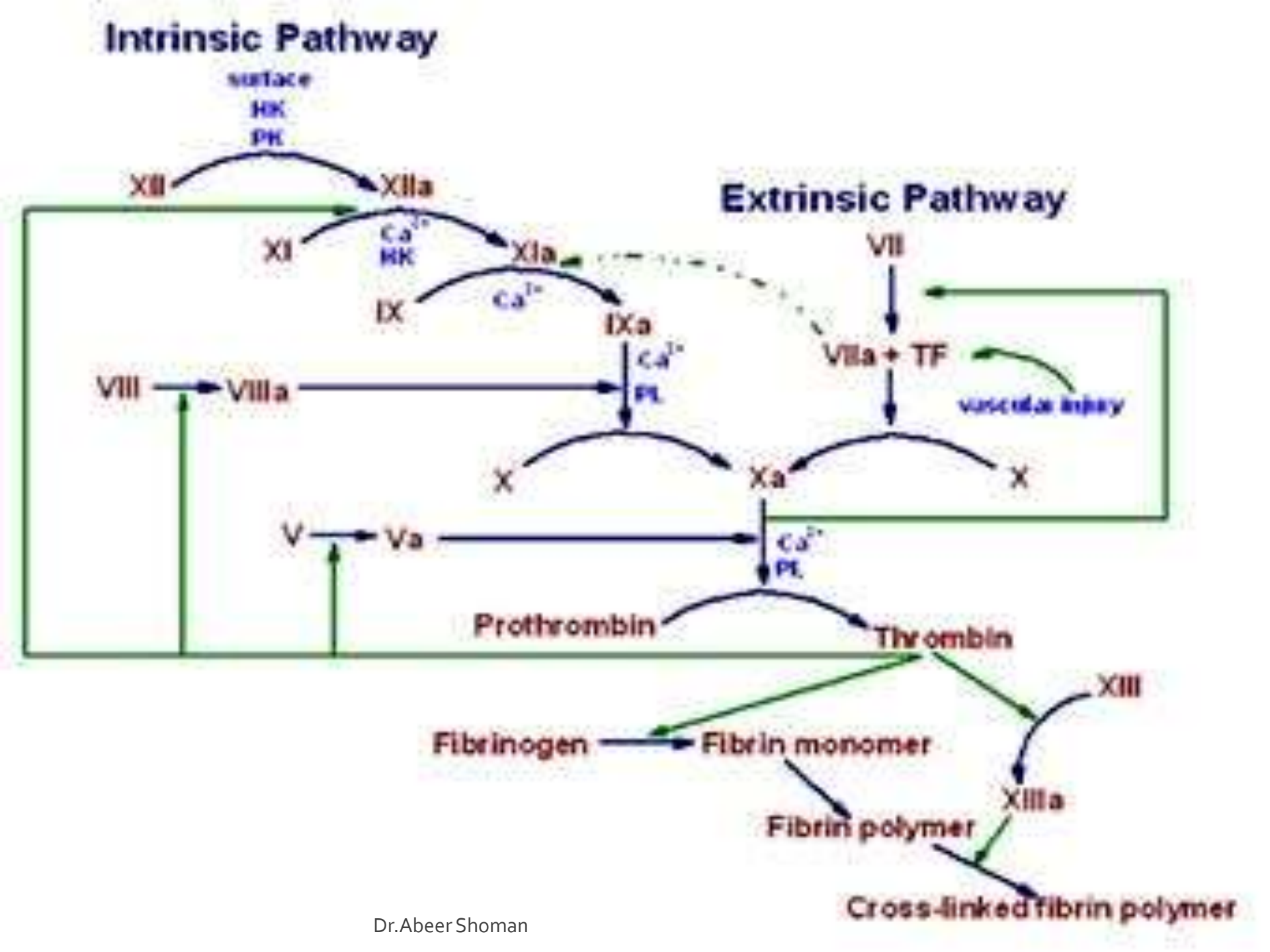
2] Intrinsic pathway

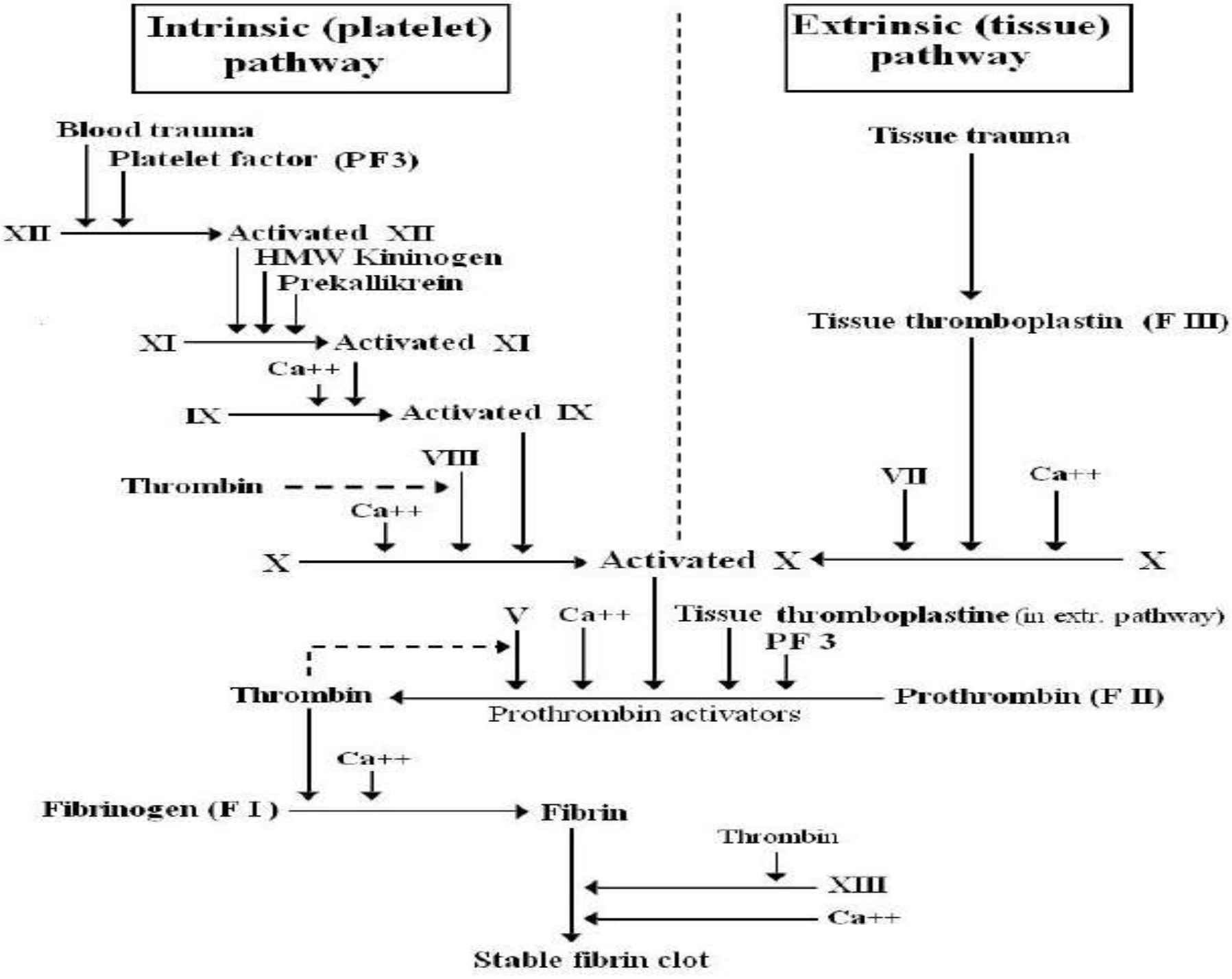
The clotting factors are:

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

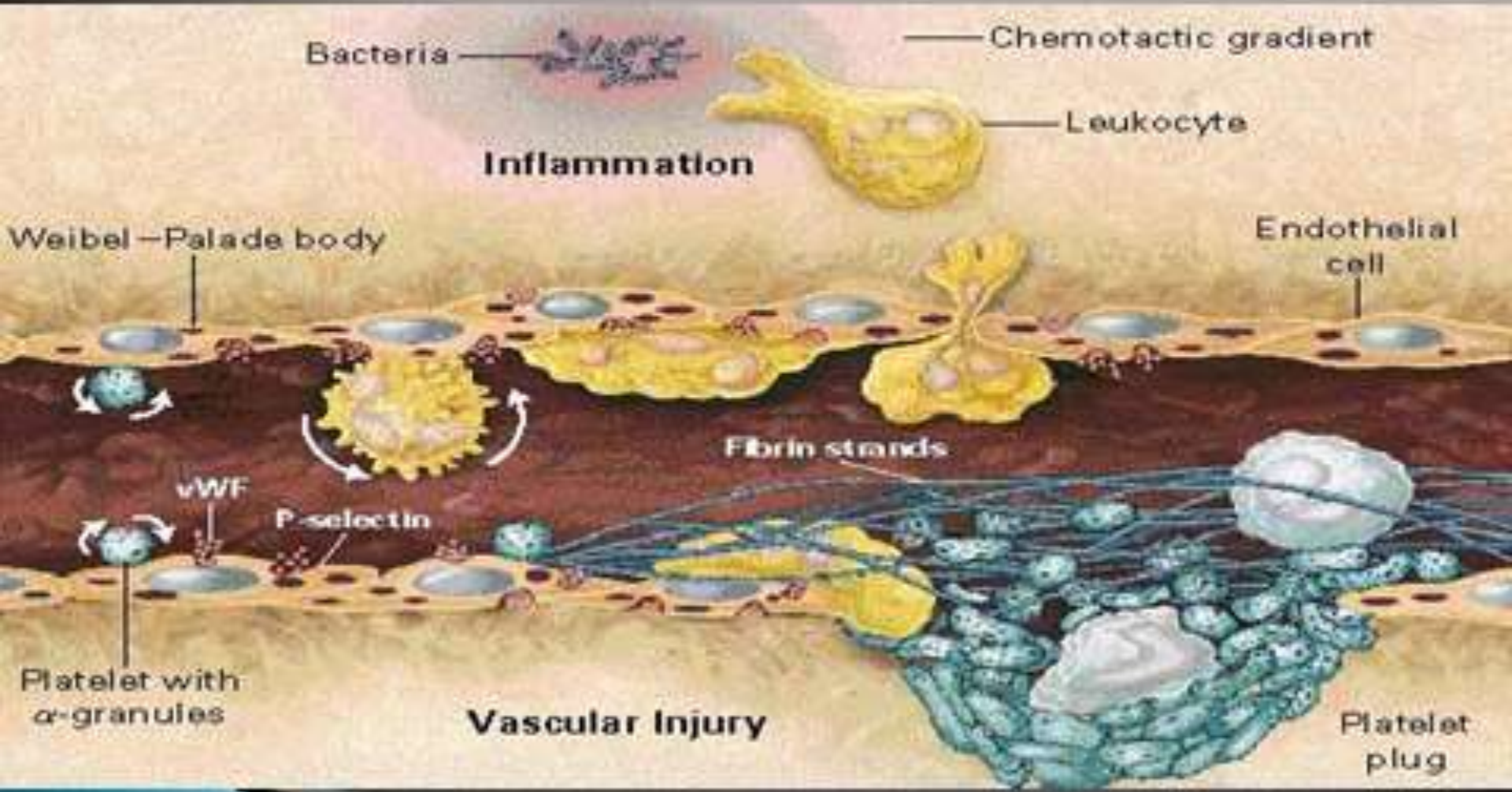
1- Extrinsic pathway




Intrinsic Pathway
surface
HK
PK
XII
XIIa
XI
XIa
IX
IXa
VIII
VIIIa
X
Xa
V
Va
Prothrombin
Thrombin
Fibrinogen
Fibrin monomer
Fibrin polymer
XIII
XIIIa
Cross-linked fibrin polymer
Extrinsic Pathway
VII
VIIa + TF
vascular injury


Intrinsic (platelet) pathway
Extrinsic (tissue) pathway
Blood trauma
Platelet factor (PF3)
Tissue trauma
XII
Activated XII
HMW Kininogen
Prekallikrein
Tissue thromboplastin (F III)
XI
Activated XI
Ca++
IX
Activated IX
VIII
Thrombin
Ca++
VII
Ca++
X
Activated X
X
V
Ca++
Tissue thromboplastine (in extr. pathway)
PF 3
Thrombin
Prothrombin (F II)
Prothrombin activators
Ca++
Fibrinogen (F I)
Fibrin
Thrombin
XIII
Ca++
Stable fibrin clot

Blood clot



Dr. Abeer Shoman

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 stop only when Ca^{++} level is severely decreased (to about 4 mg %) and such level cannot 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

3- Heparin

SPECIFIC

1-Antithrombin III (IX,X,XI,XII)

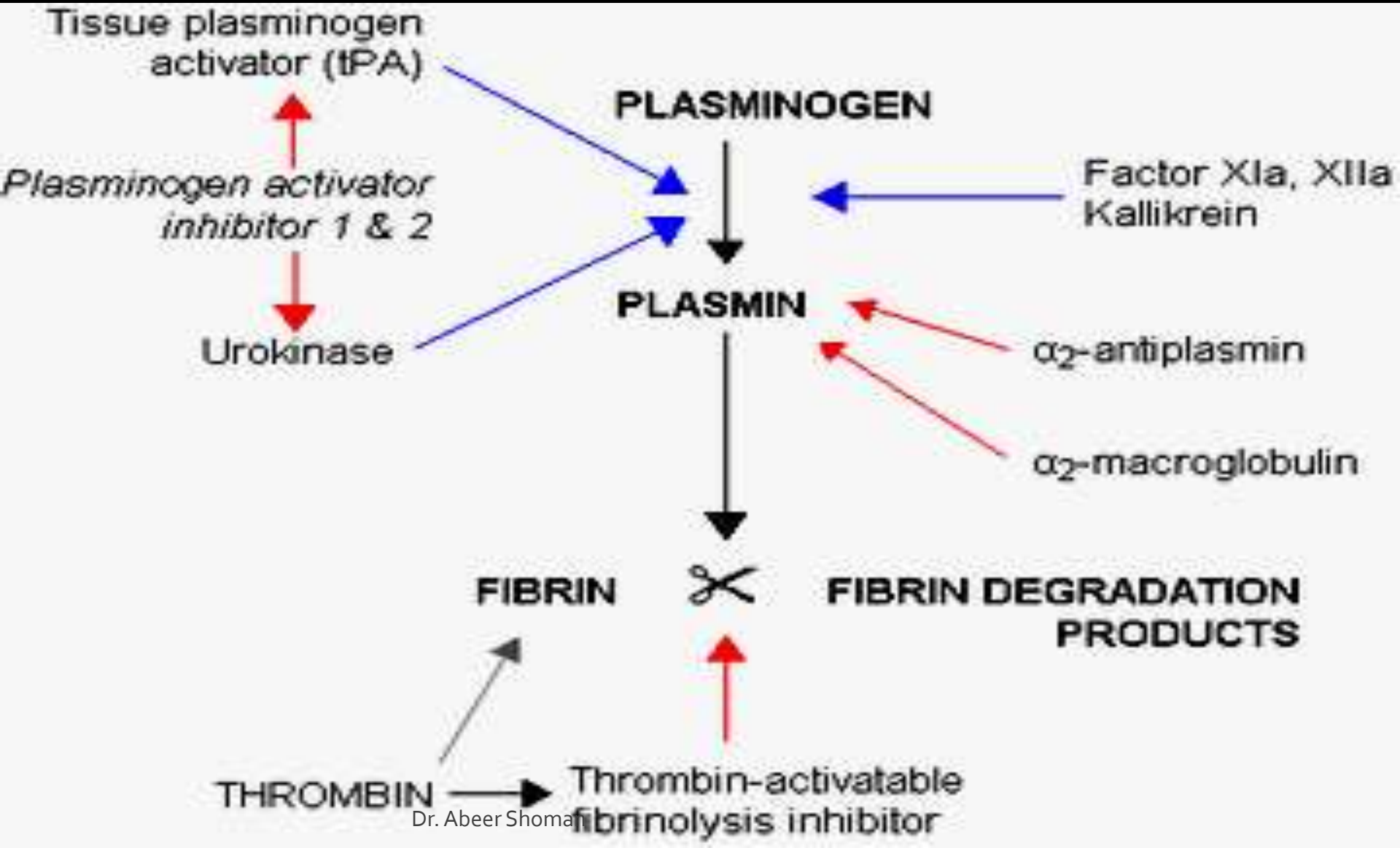
2-prostacyclins

3-fibrinolytic system

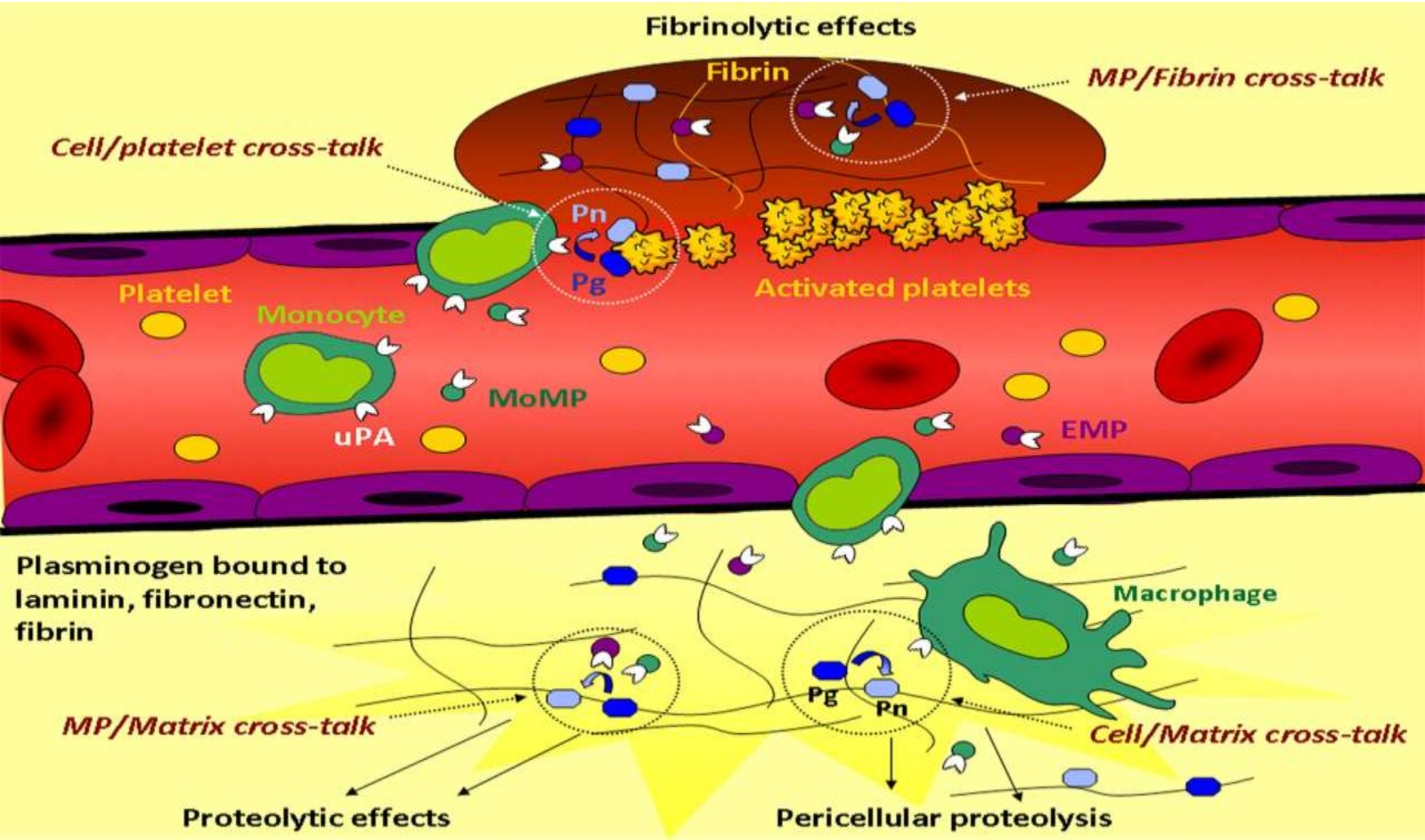
4- protein C, protein S (V, VIII)

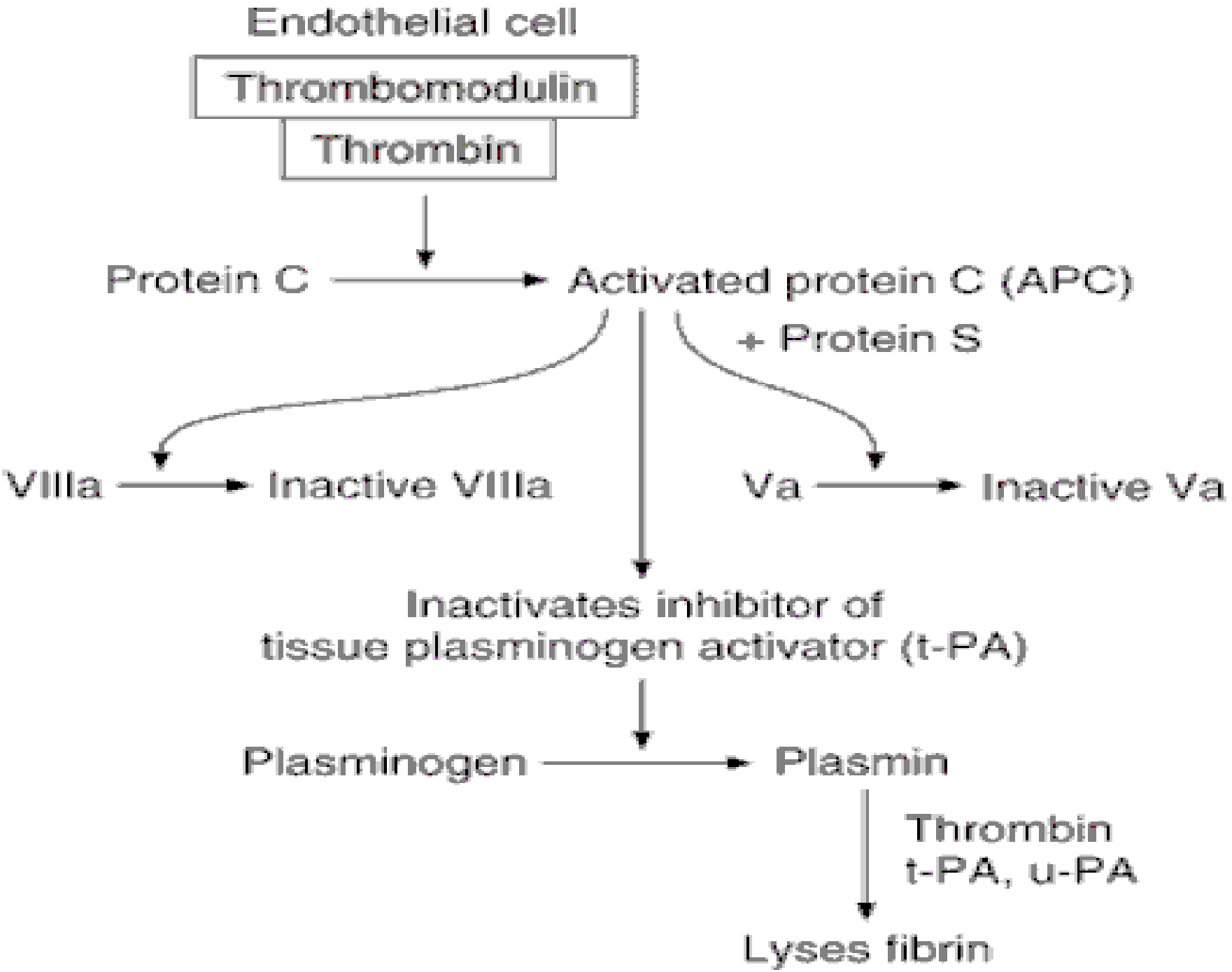
Increase plasmin formation

Fibrinolysis



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

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

IN VIVO

1] Heparin
2] Dicumarol

	Dicumarol	Heparin
1-Source	plant	Basophil, mast, liver
2-Intake	oral	injection
3-Oncet, duration	Slow, long	Rapid, short
4- Chemistry	As vitamin K	Sulphated mucopolysaccaride
5-Site of action	In Vivo	In vivo ,vitro
6-Actions	Inhibit vit K (II,VII,IX, X)	1] Antithrombin III 2] -- factor IX 3]↑lipase enzyme
7-Antidote	Vit. K	Protamine sulphate 1%

Dr.Abeer Shoman

Abnormalities of Hemostasis

Vit.K deficiency

Hemophilia

Purpura

Thrombo-embolism

D.I.C.

Abnormalities of Hemostasis

Vitamin K deficiency

-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

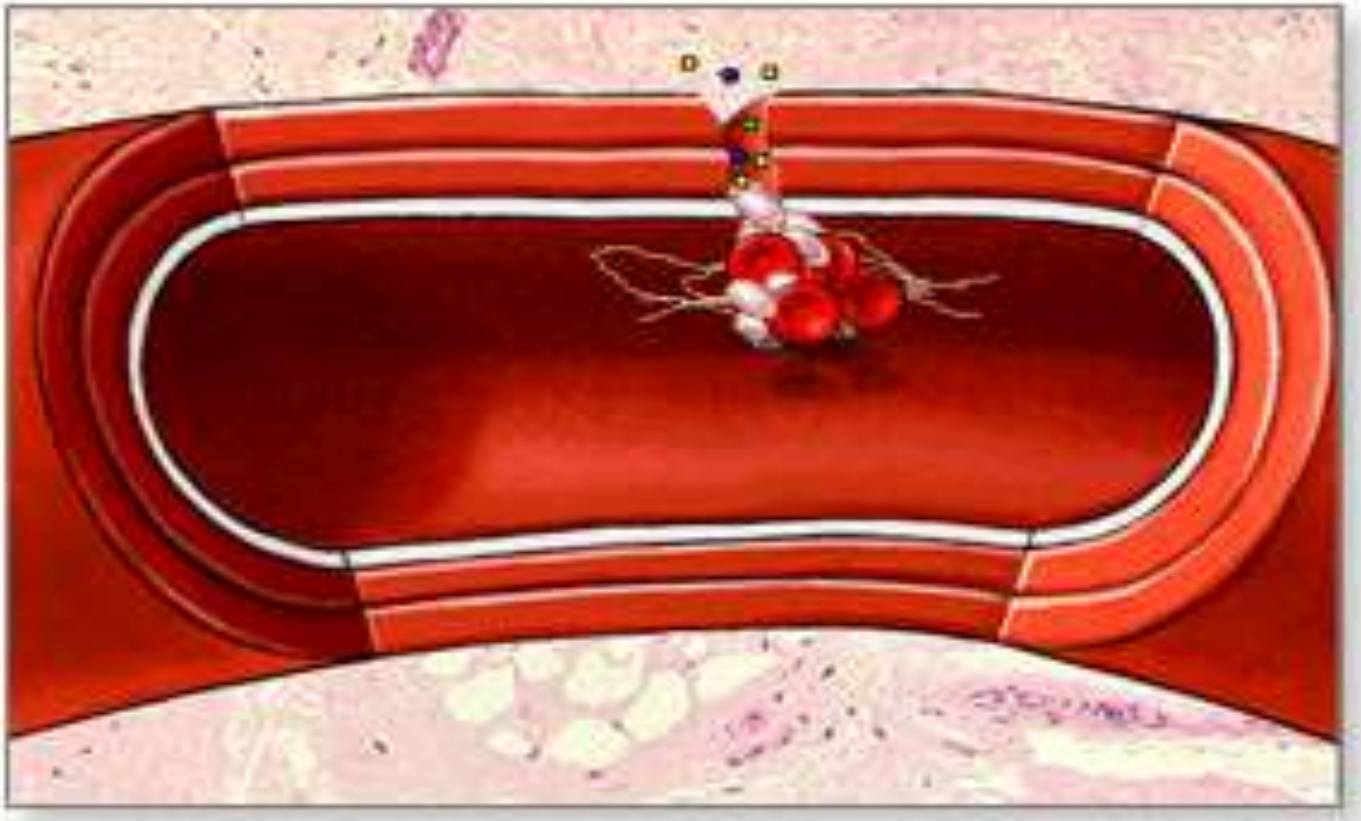
2- Fat malabsorption because vit K is fat soluble vitamin

a- Liver diseases.

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

Vitamin K

Vitamin K benefits blood clotting

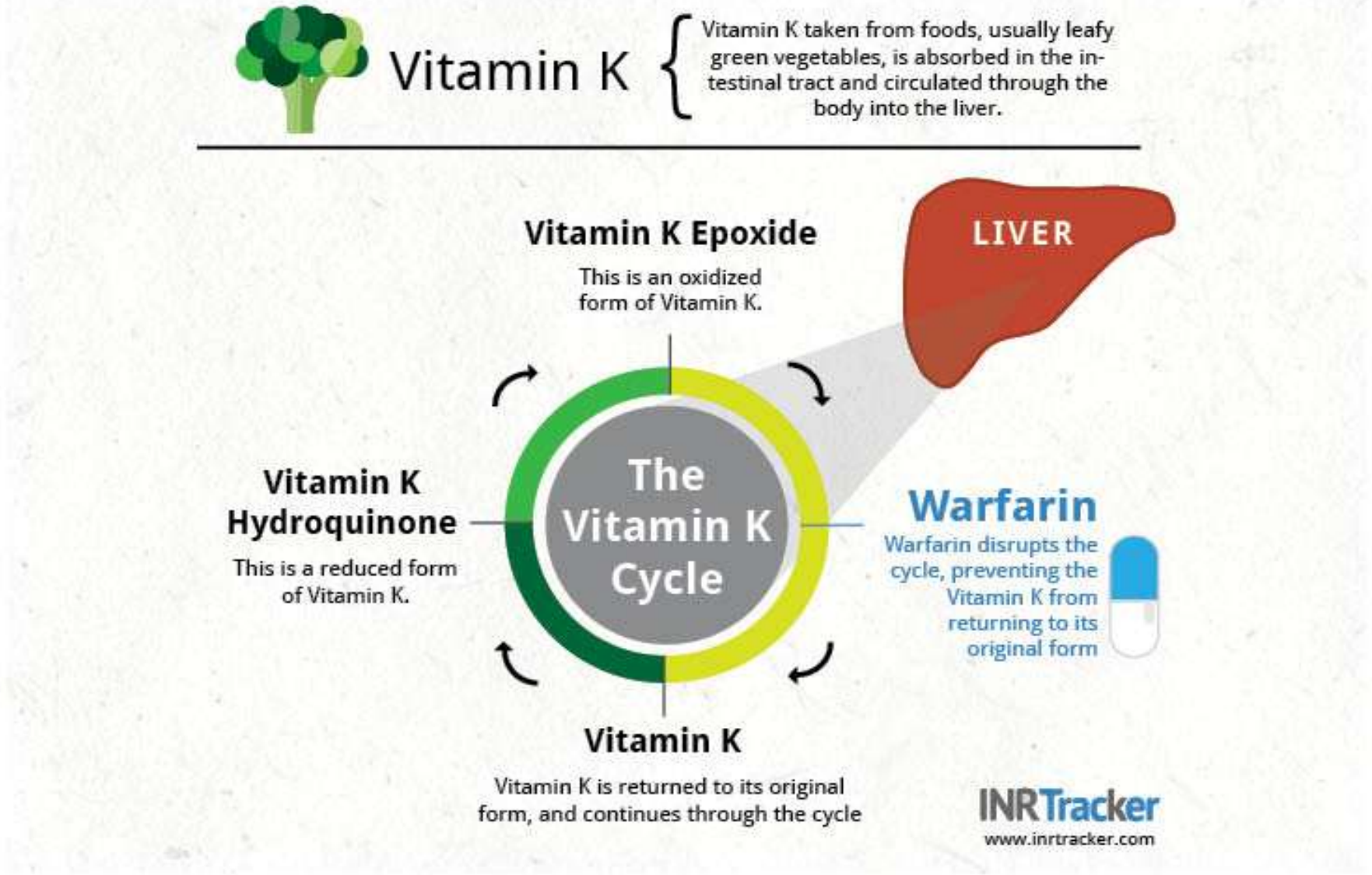


Recommended daily allowance for adults:

120 µg men
90 µg women

Fat-soluble

A.D.A.M.


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.
Vitamin K Epoxide
This is an oxidized form of Vitamin K.
LIVER
Vitamin K Hydroquinone
This is a reduced form of Vitamin K.
The Vitamin K Cycle
Warfarin
Warfarin disrupts the cycle, preventing the Vitamin K from returning to its original form
Vitamin K
Vitamin K is returned to its original form, and continues through the cycle
INRTracker
www.inrtracker.com

VITAMIN K BENEFITS, SOURCES & DEFICENCY

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.

RDA: 90 mg | Water-soluble Vitamin

BENEFICIAL FOR

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

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 DEFICENCY

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, is it usual to give all newborn infants prophylactic vitamin K.

SUPERHERBALFOODS herbal supplements for a healthy life

www.superherbalfoods.com


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

Dr. Abeer Shoman

(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

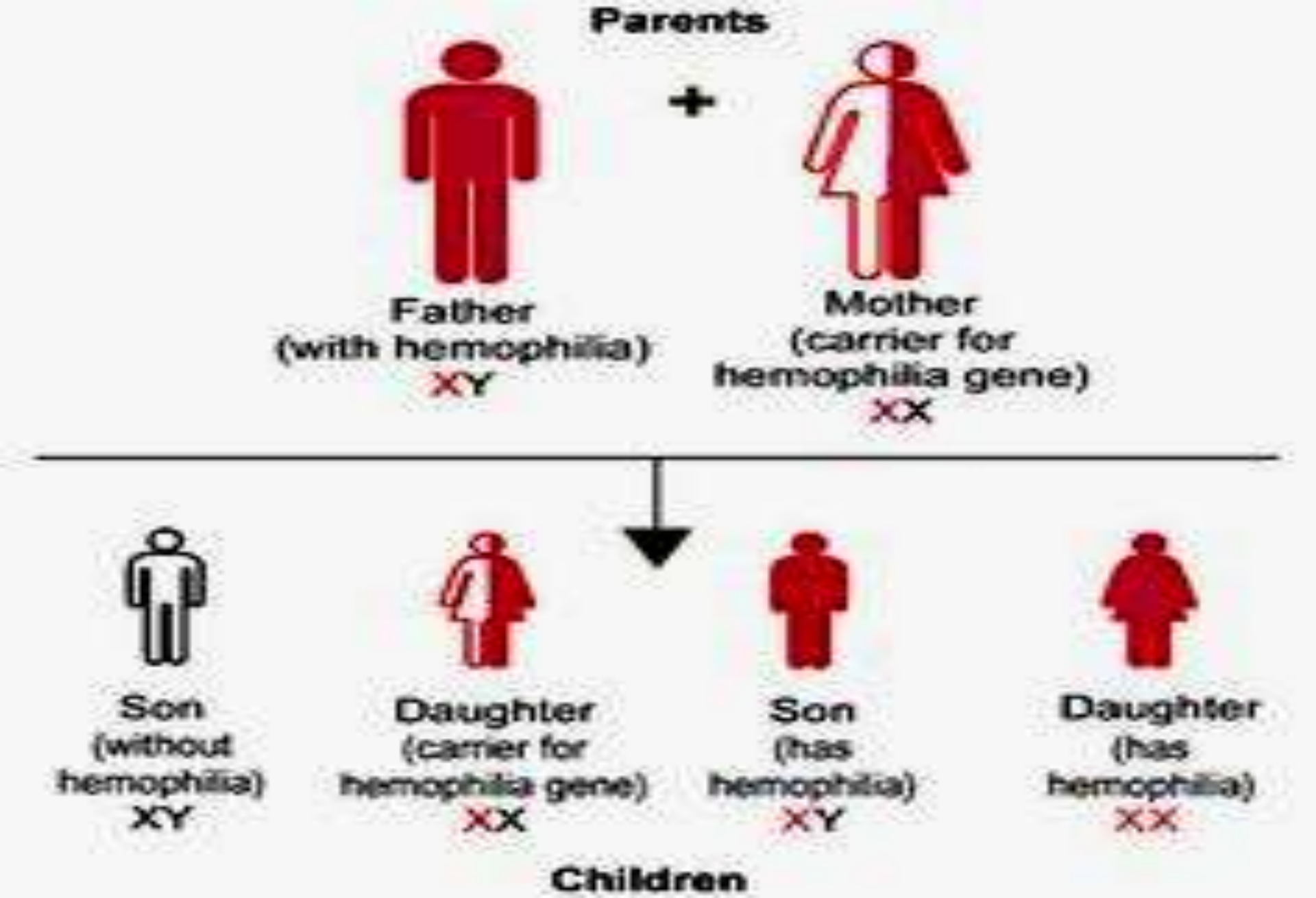
Intracranial Hemorrhage

Prolonged Nosebleeds

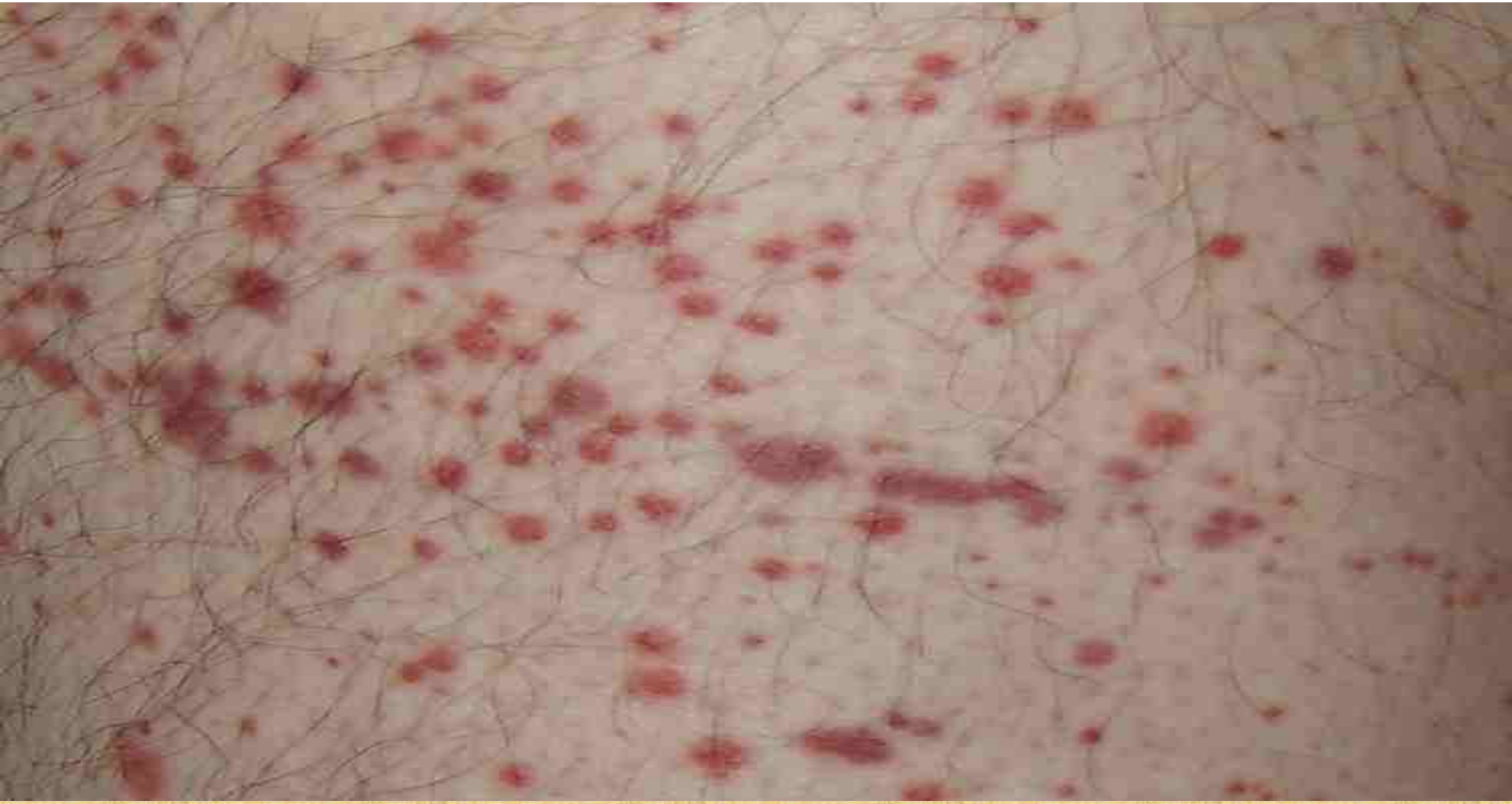
Bruises Easily

Warm, Painful, Swollen Joints With ↓ Movement

GI Hemorrhage

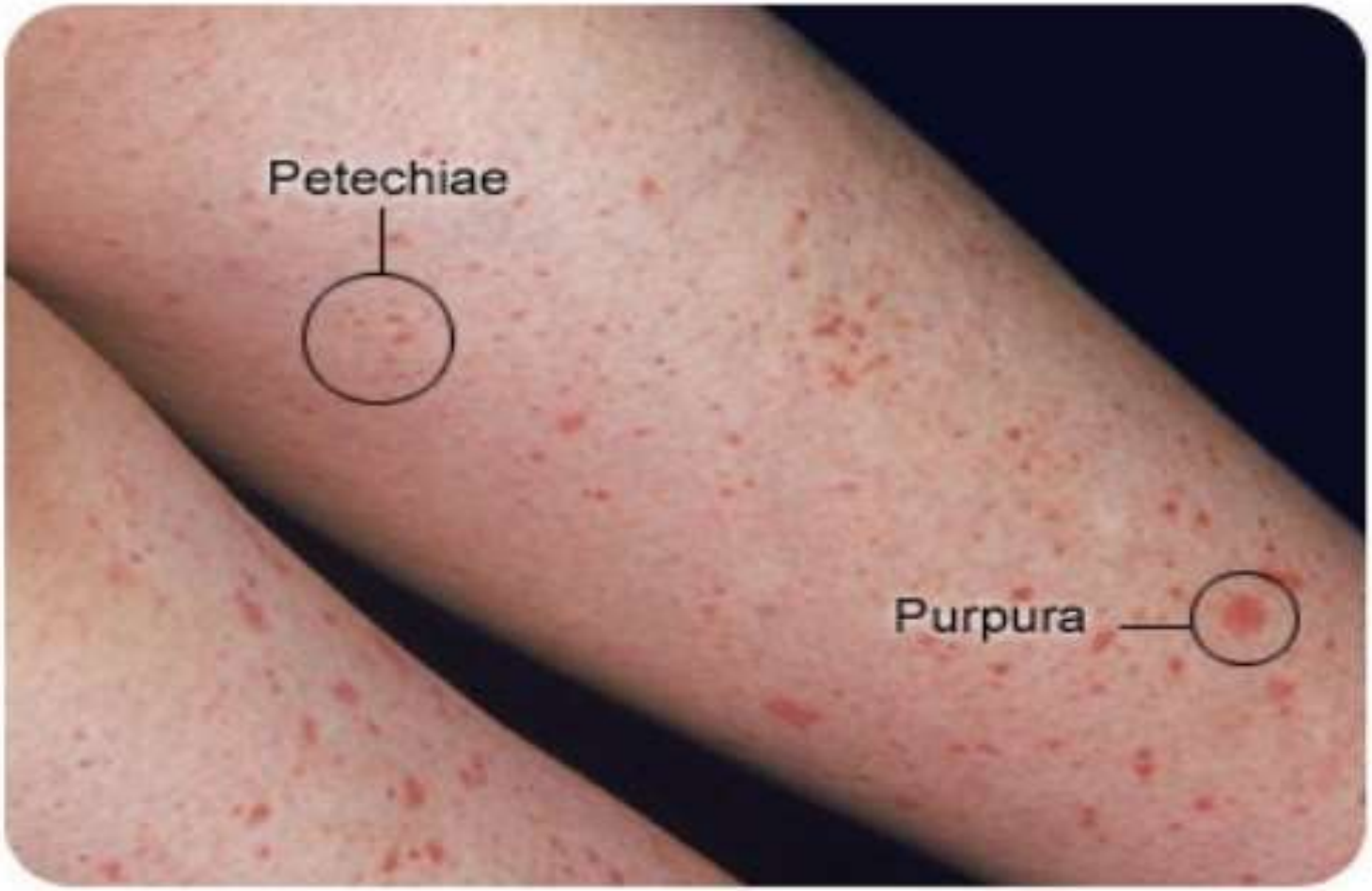

Parents
+
Father
(with hemophilia)
XY
Mother
(carrier for
hemophilia gene)
XX
Son
(without
hemophilia)
XY
Daughter
(carrier for
hemophilia gene)
XX
Son
(has
hemophilia)
XY
Daughter
(has
hemophilia)
XX
Children

Purpura

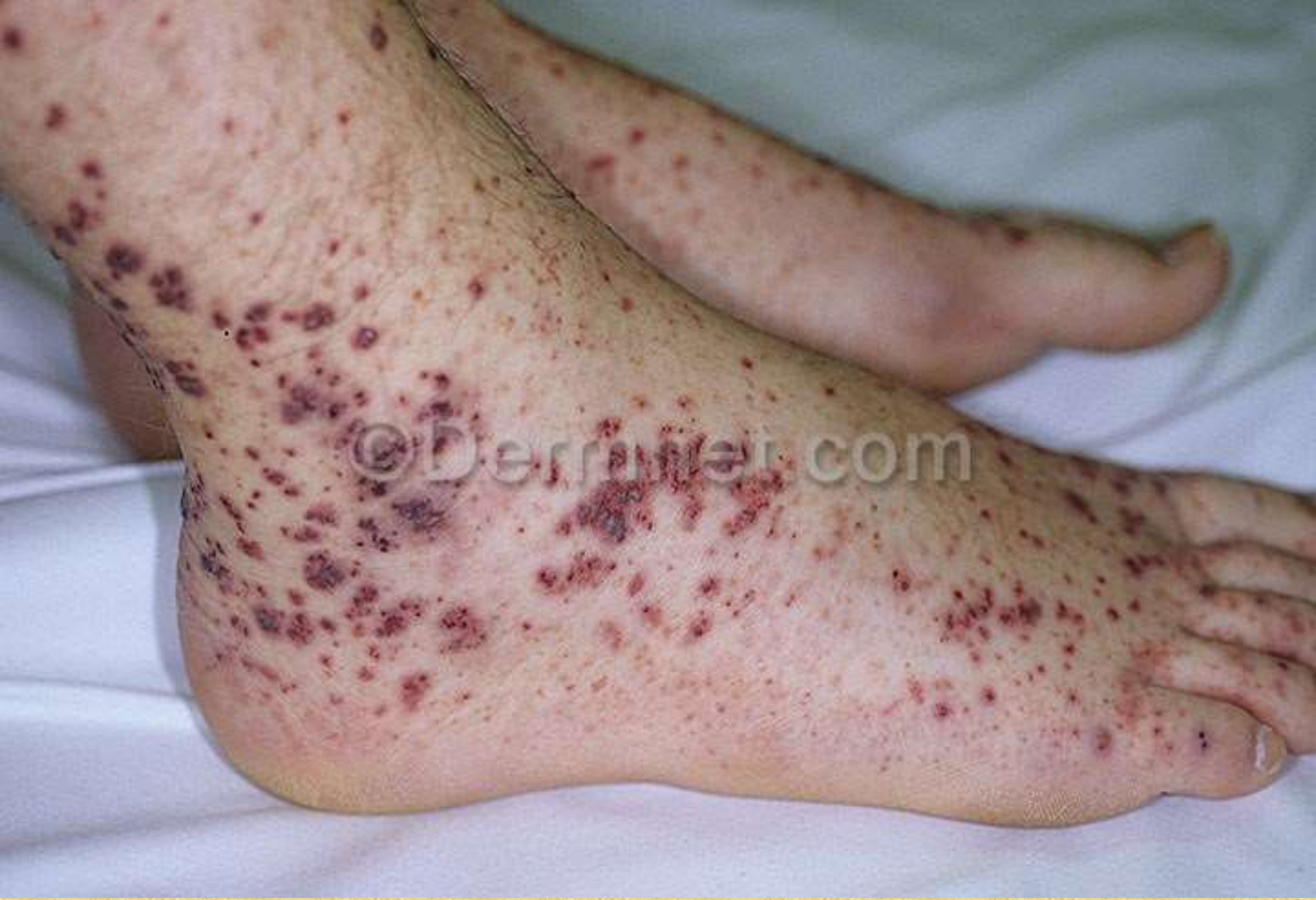


(3) Thrombocytopenic purpura:

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

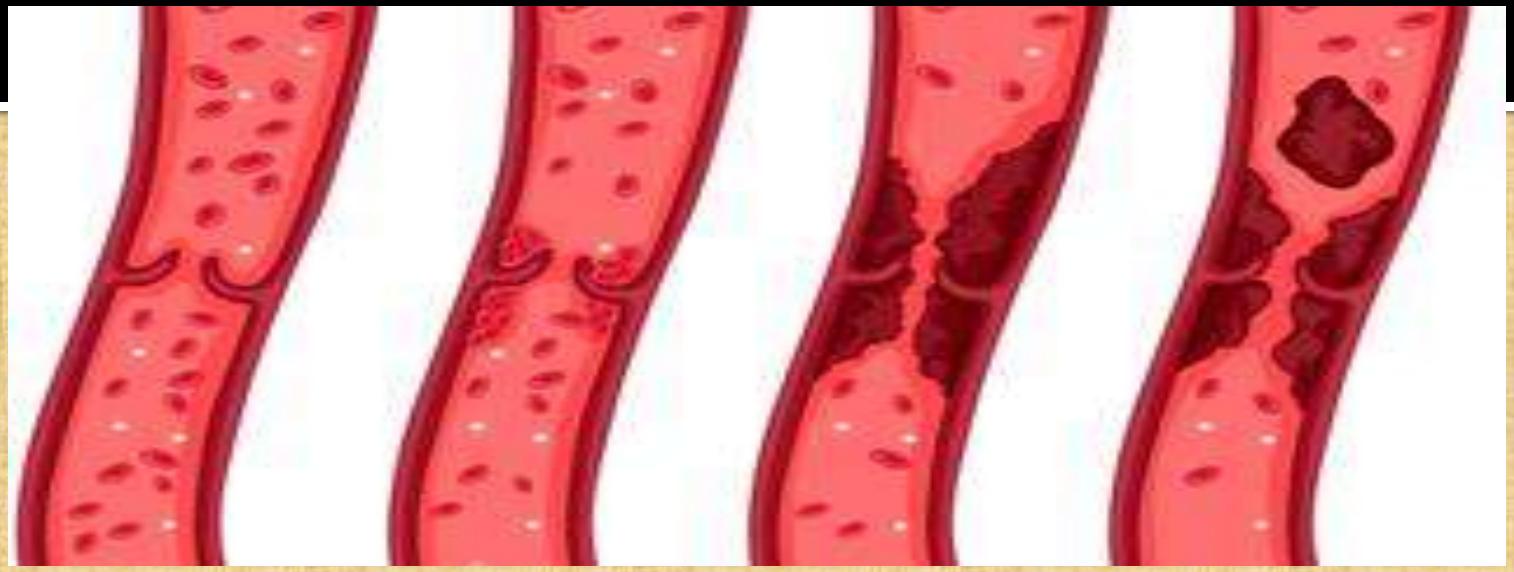


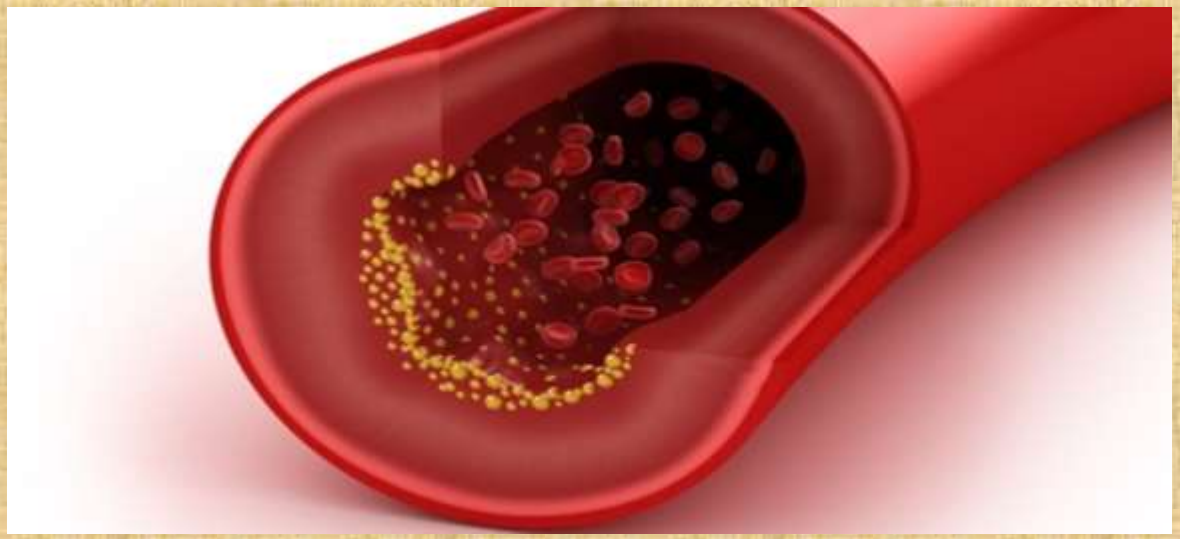
Petechiae	1-3mm
Purpura	3mm-10 mm
Ecchymosis	>10mm



Dr. Abeer Shoman

Thromboembolism

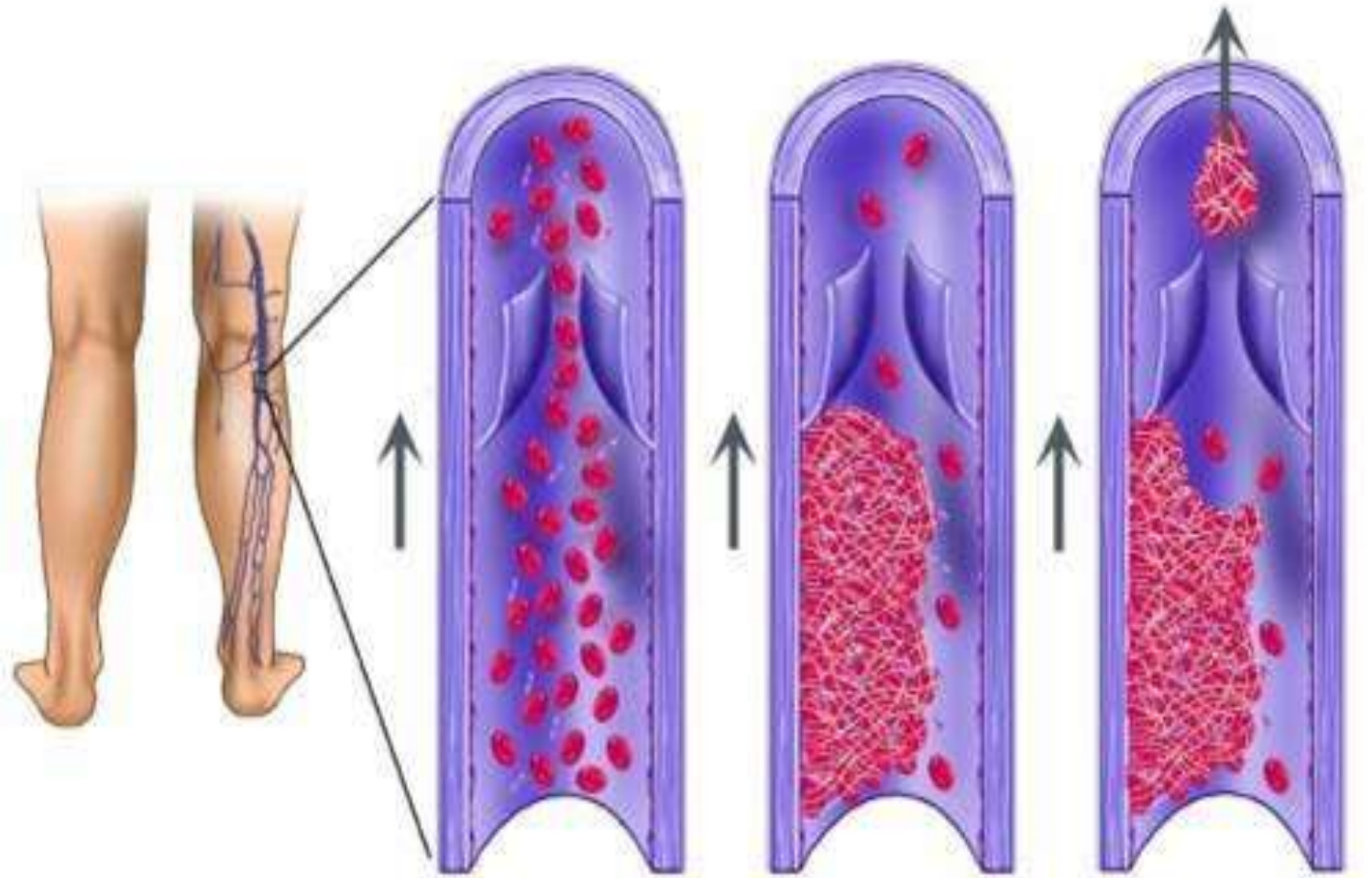




(4)Thromboembolism:

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

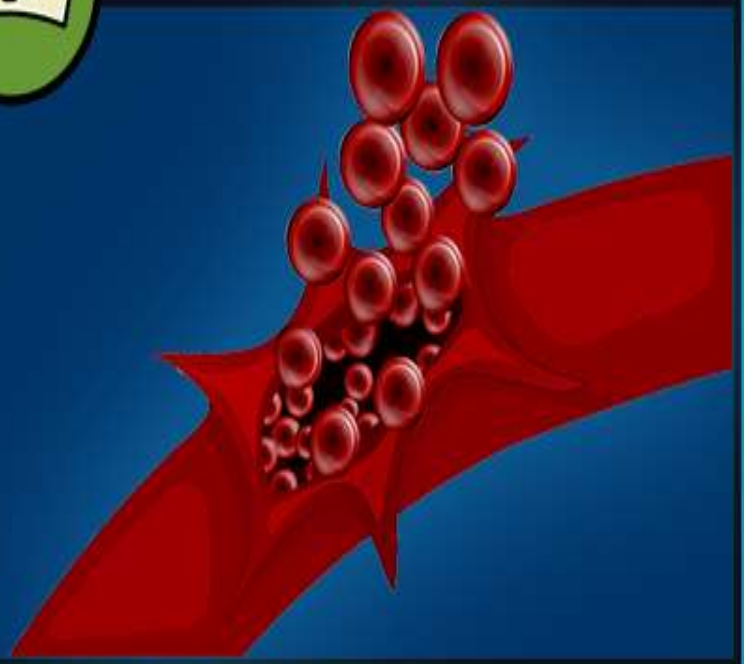
(



5) Disseminated intravascular clotting(DIC):

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

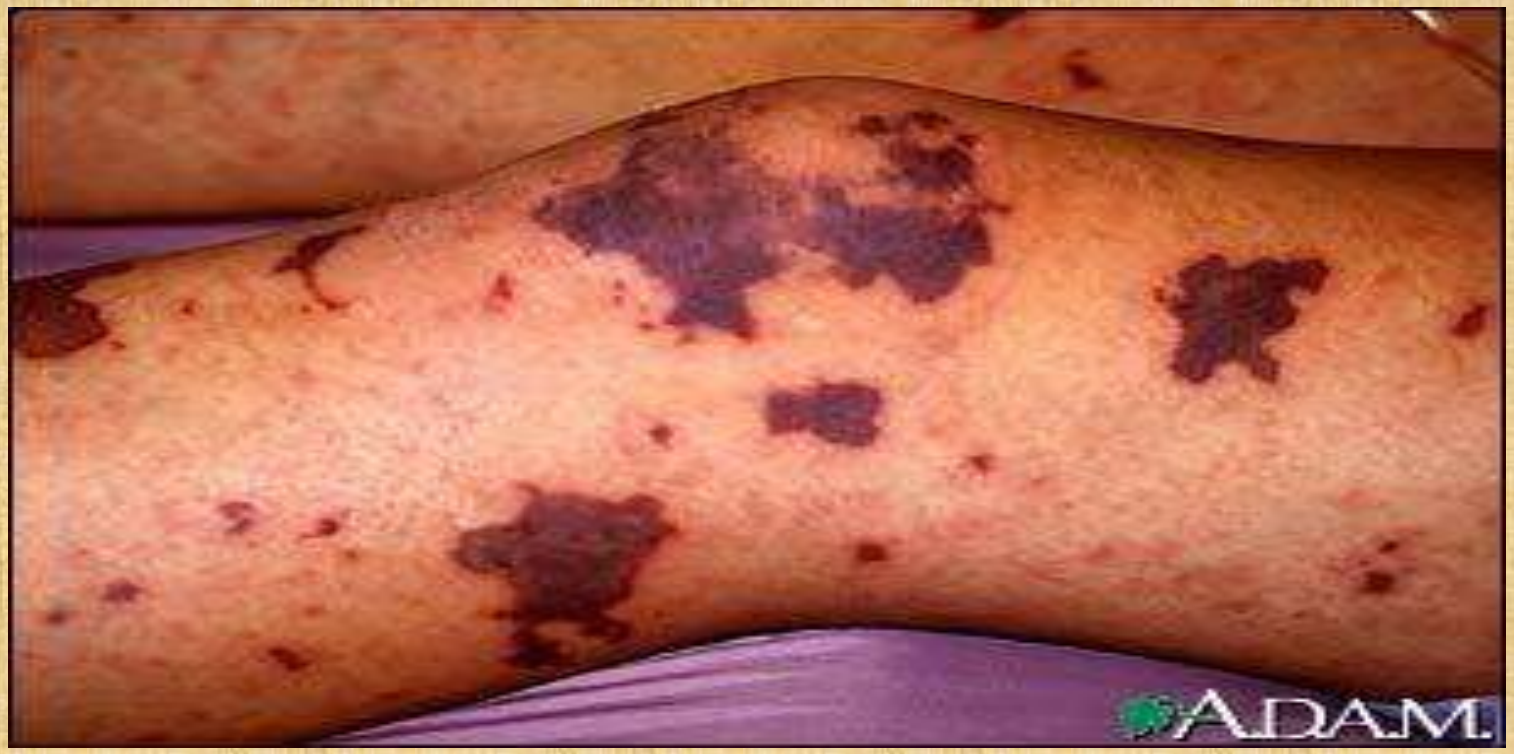
Disseminated Intravascular Coagulation



NURSING CARE PLANS

Nurseslabs

Disseminated Intravascular Coagulation



Dr. Abeer Shoman


Thank You