CLOTTING DISORDERS

DEPT. OF ORAL PATHOLOGY

Learning Objectives

At the end of the lecture student should be able to-

- Describe mechanism of haemostasis
- Enlist Clotting disorders
- Describe clinical & laboratory assessment

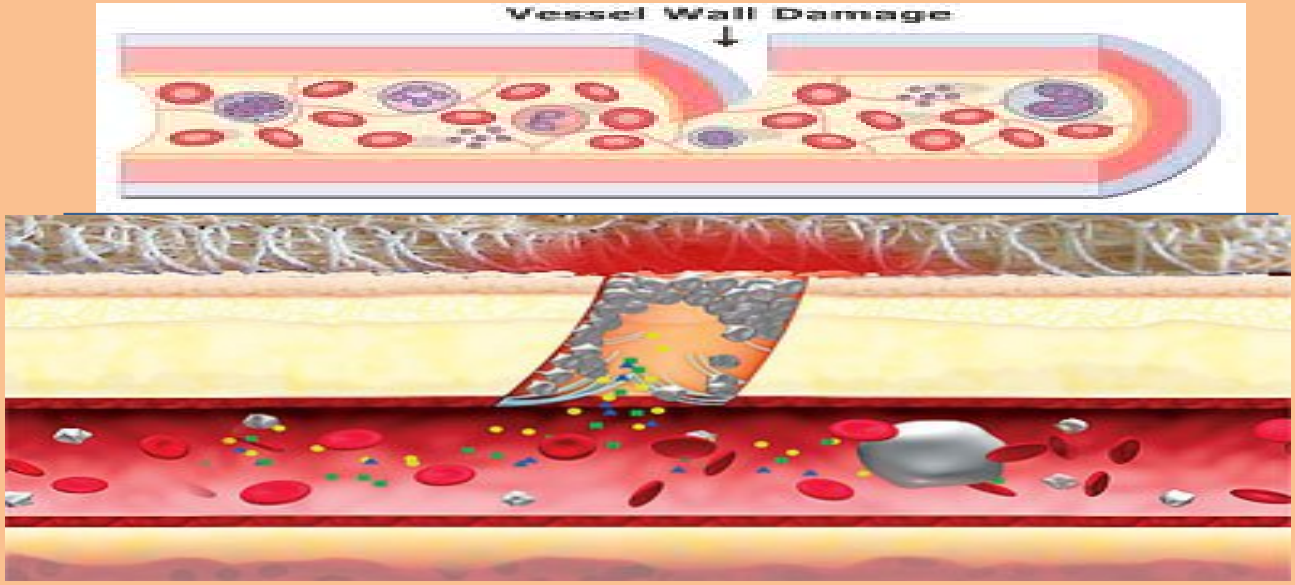
- Dental procedures resulting i
  bleeding can have serious consequences in a pt. having bleeding disorder……
  severe hemorrhage or even death.

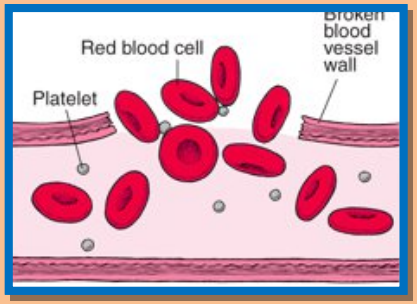
BASIC MECHANISM OF HAEMOSTASIS

- Vascular phase.
- Platelet phase.
- Coagulation phase.
- Fibrinolytic phase.(rate limiting step)

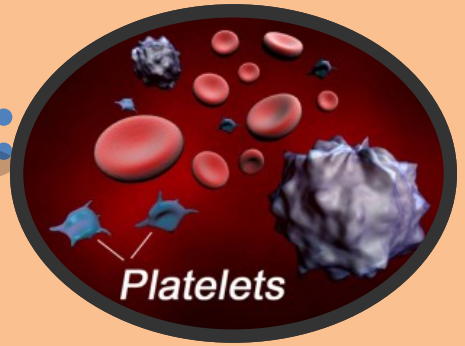
VASCULAR PHASE:

- After tissue injury ⟶ immediate vasoconstriction occurs.
- Serotonin,histamine,PG's etc causes vasoconstriction of the micro vascular bed.



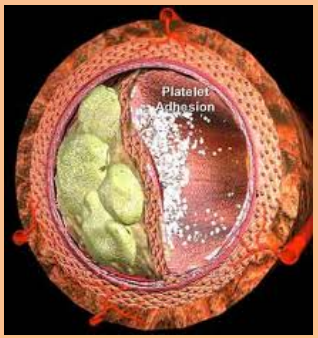


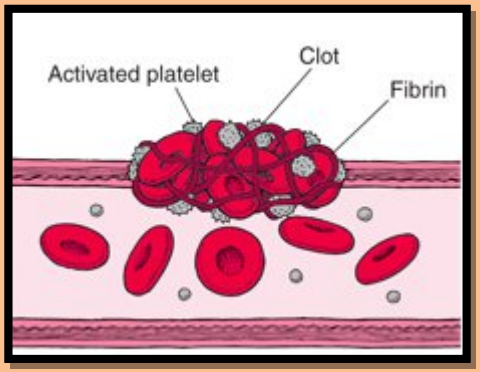
PLATELET PHASE:



Circulating blood platelets are activated → Aggregates → Primary vascular plug(es blood loss from small blood vessels & capillaries)

Adheres to exposed basement membrane.

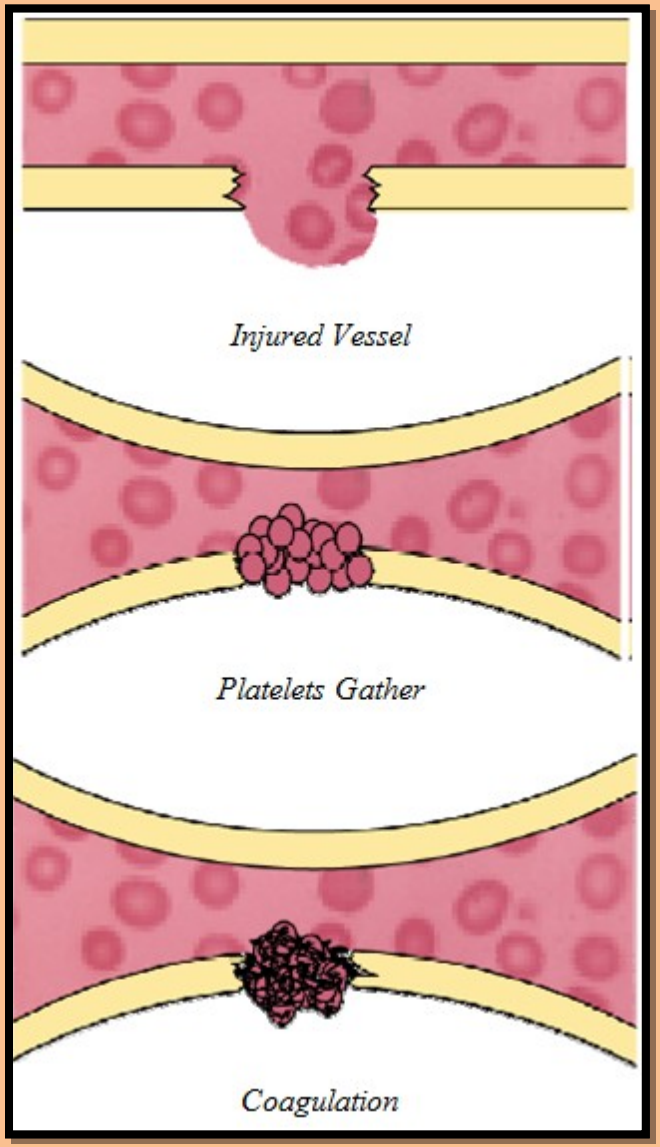


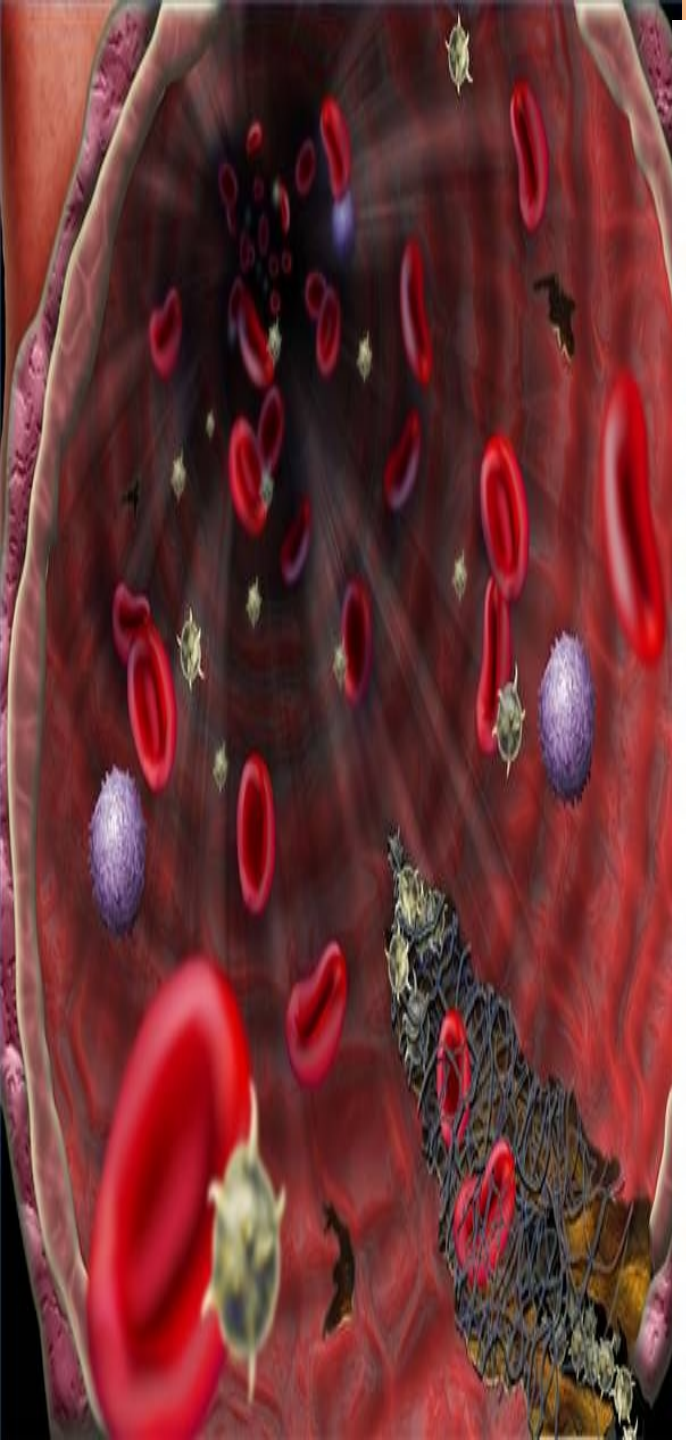


COAGULATION PHASE:

- Generation of THROMBIN and FIBRIN.
- INVOLVES VARIOUS PROTEINS: Fibrinogen,prothrombin,FS-V,VII,IX,X,XI,XII & XIII.--------- Vitamin K dependant-FS-II,VII, IX & X.
- Involves 3 separate pathways → INTRINSIC PATHWAY, EXTRINSIC PATHWAY, COMMON PATHWAY.
- FIBRIN polymerizes to a gel → stabilizes the platelet plug.

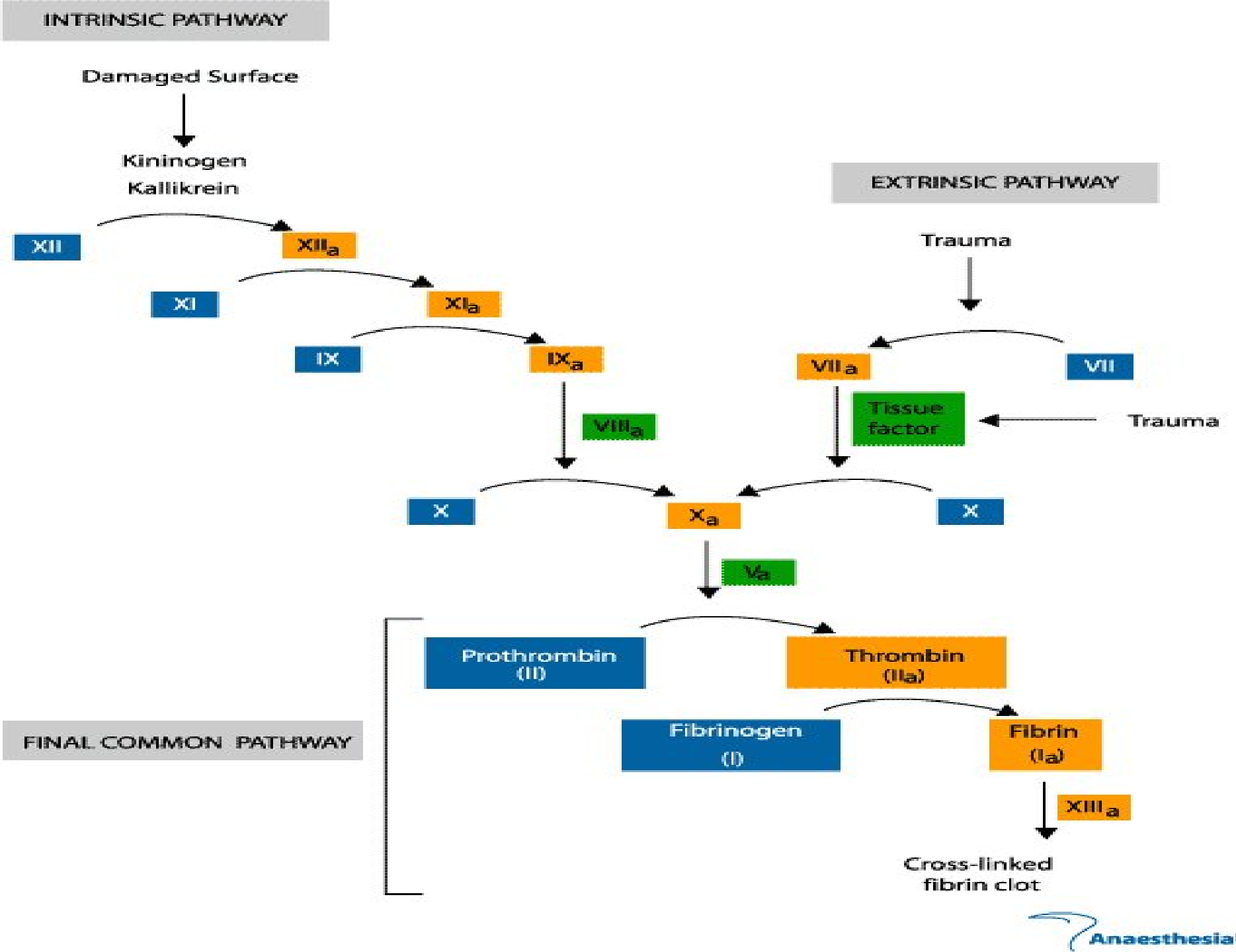
- 2 theories:::
- **Prothrombin to thrombin & fibrinogen to fibrin conversion system** (MARKOWITZ----1903)
- **CASCADE / WATERFALL theory**(1964)-the coagulation mechanism results in a final explosive change of a liquid to a gel.

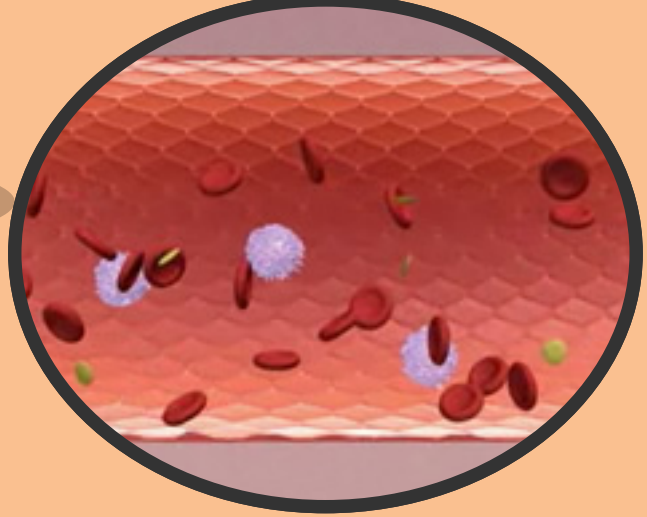

Injured Vessel
Platelets Gather
Coagulation



CLOTTING FACTORS AND THEIR SYNONYMS

Clotting Factor	Synonym
Fibrinogen	Factor I
Prothrombin	Factor II
Tissue Thromboplastin	Factor III; Tissue factor
Calcium	Factor IV
Factor V	Proaccelerin; Labile factor
Factor VII	Serum prothrombin conversion accelerator; proconvertin
Factor VIII	Antihemophilic factor; AHF
Factor IX	Plasma thromboplastin component
Factor X	Stuart-Prower factor
Factor XI	Plasma thromboplastin antecedent
Factor XII	Hageman factor
Factor XIII	Fibrin-stabilizing factor
Platelets	


INTRINSIC PATHWAY
Damaged Surface
Kininogen
Kallikrein
XII
XIIa
XI
XIa
IX
IXa
VIIIa
EXTRINSIC PATHWAY
Trauma
VII a
VII
Tissue factor
Trauma
X
Xa
X
Va
Prothrombin (II)
Thrombin (IIa)
Fibrinogen (I)
Fibrin (Ia)
XIII a
Cross-linked fibrin clot
FINAL COMMON PATHWAY
AnaesthesiaUK



FIBRINOLYTIC PHASE:

- Propagation of the clot is limited by **fibrinolysis.**
- Tissue plasminogen activator(tPA) released from the endothelial cells converts **PLASMINOGEN** to **PLASMIN.**
- Plasmin degrades fibrinogen & fibrin to fibrin degradation products **[FDPs]** .

CLINICAL
&
LABORATARY FINDINGS

CLINICAL FEATURES

- Bleeding from superficial cuts & scratches.
- Delayed bleeding.
- Spontaneous gingival bleeding.
- Petechiae.
- Ecchymoses.
- Epistaxis.
- Deep dissecting hematomas.
- Hemarthroses.

CLINICAL LABORATARY TESTS

- Help to
  Identify deficiency of required elements
  Dysfunction of the phases of coagulation

Platelet count
Bleeding time
PFA-100 CT
PT/INR
aPTT
TT
FDPs
Factor assays
Tests of capillary fragility

PLATELET COUNT:

- Normal-150,000 to 450,000/mm3
- If < 50,000/mm3

Hemorrhagic stroke
Surgical/traumatic hemorrhage
etc. may occur.

- In such cases platel[illegible]ary.
- **Bleeding time{ 1-6 mins}-modified Ivy's test.**

PT & INR

- **Normal-11 to 30 secs**
- **its now commonly reported with its INR.**

INR Intro.by WHO(1983):itz the ratio of PT that adjusts for the sensitivity of the thromboplastin reagants,such that normal coagulation profile is reported as an INR of 1.0

INR (International Normalized Ratio) Test

$$INR = \left[\frac{\text{Patient PT}}{\text{Control PT}} \right] {}^{*}ISI$$

Normal INR: 0.9 - 1.2

- INR: 2.0 - 3.0 : Therapeutic Range with Coumadin
- INR under 2.0 is associated with minimal bleeding
- INR of 3 - 4.5: Associated with excessive bleeding
- INR is checked every 4-6 weeks

*International Sensitivity Index of Thromboplastin

USES of PT/INR:

- **Evaluates extrinsic coagulation system.**
- **Measures the presence/absence of Fs-I,II,V,VII & X.**
- **Reduction of vit K dependant Fs-I,II,VII & X.**

Activated partial thromboplastin time:

- Considered normal if the control aPTT & test aPTT are within 10 secs of each other.
- Control aPTT = 15-35secs.
- Itz altered in hemophilias A & B. and with the use of heparin.

THROMBIN TIME:

- Normal-9 to 13 secs.
- Measure the activity of heparin,FDPs,other para proteins that inhibit conversion of fibrinogen to fibrin.

FACTOR ASSAYS:

- **IDENTIFY FACTOR DEFICIENCIES.**

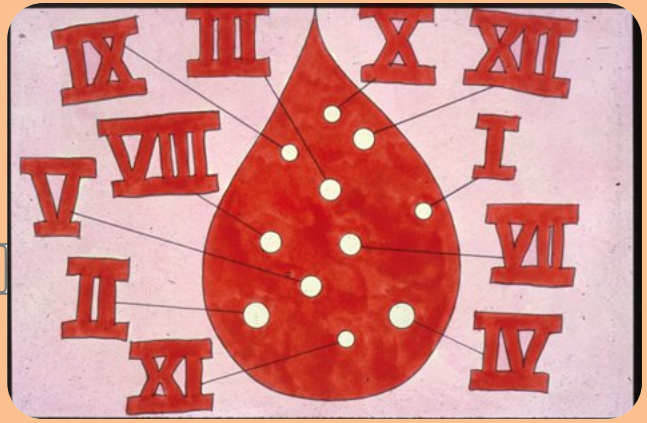
CLASSIFICATION OF BLEEDING DISORDERS:

Vessel wall disorders.

Platelet disorders.

Coagulation disorders.

CONGENITAL COAGULOPATHI



HEMOPHILIA A

HEMOPHILIA B

FACTOR XI DEFICIENCY

FACTOR XII „

FACTOR X „

FACTOR V „

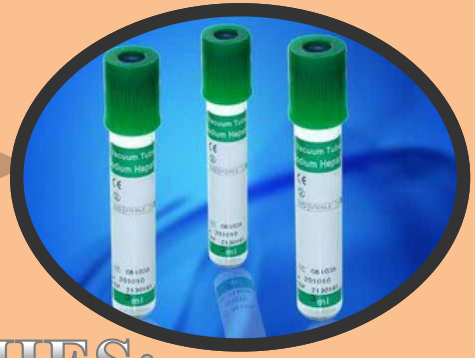
FACTOR XIII & I DEFICIENCIES.

VON WILLEBRAND'S DISEASE.

ANTI COAGULANT RELATED COAGULOPATHIES:

Heparin

Coumarin.



DISEASE RELATED COAGULOPATHIES:

Liver disease

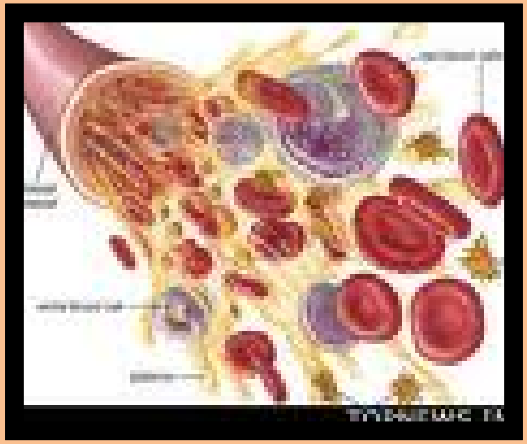
Vitamin K deficiency

DIC

Fibrinolytic disorders.

COAGULATION DISORDERS

- **CONGENITAL--** HEMOPHILIA A
  HEMOPHILIA B
  F XI DEFICIENCY
  F XII ,,
  F X ,,
  F V ,,
  F XIII & I ,,
  VON-WILLEBRAND'S DISEASE.



ACQUIRED- .Secondary to drugs (Heparin , Coumarin) or disease process(Liver disease, Vit K deficiency ,DIC).

SUMMARY

- Mechanism of haemostasis
- Clotting disorders
- Clinical & laboratory assessment

References

- Basic Pathology. Kumar, Cortan, Robbin. sixth edition.
- Shafers Oral Pathology.
- Basics of hematology. Kwathilkar.3rd edition.
- Neville Oral Pathology

THANK YOU