Introduction Hemostasis: Tourniquet Test & Bleeding Time

Hematology-Immunology System
Faculty of Medicine – Universitas Padjadjaran
Hemostasis

A series of reactions that function to stop bleeding, maintaining blood in a fluid state and confined to the circulatory system

During hemostasis, three phases occur in rapid sequence

- Vascular spasms – immediate vasoconstriction in response to injury
- Platelet plug formation
- Coagulation (blood clotting)
Hemostasis: A delicate balance

hemostasis/thrombosis
hemorheology

prothrombotic

Normal

endogeneous fibrinolysis

antithrombotic

BLEEDING

THROMBOSIS
The function of hemostasis

- To prevent blood loss from severed vessels
- To stop bleeding
- To prevent thrombosis
These phases are not separated but rather interconnected and related.

- **VASCULAR PHASE**
- **PLATELET PHASE**
- **COAGULATION PHASE**
- **FIBRINOLYTIC PHASE**
HEMOSTASIS

- **Primary Hemostasis**
  - Blood vessel contraction
  - Platelet plug formation

- **Secondary Hemostasis**
  - Activation of coagulation cascade
  - Deposition & stabilization of fibrin

- **Tertiary Hemostasis (fibrinolysis)**
  - Dissolution of fibrin clot
  - Dependent on plasminogen activation
Primary Hemostasis
(platelet plug)

Secondary Hemostasis
(Hemostatic Plug)
Primary Hemostasis: Vascular phase

When a blood vessel is damaged, vasoconstriction reduces blood flow to injured area

- Spasm of smooth muscle in blood vessels
- Vasoconstrictive substances (thromboxane A$_2$)
- Nervous reflexes
Platelets adhere to the damaged surface and form a temporary plug.
Plug Formation

resting platelets

platelet adhesion

shape change

release

aggregation

fibrin

coaagulation
SECONDARY HEMOSTASIS: Coagulation Cascade

- A set of reactions in which blood is transformed from a liquid to a gel (clot) to stabilize and reinforce the weak platelet plug by fibrin

- Coagulation follows intrinsic and extrinsic pathways

- 3 main steps (Coagulation Phases):
  - formation of prothrombin activator
  - conversion of prothrombin into thrombin
  - conversion of fibrinogen to fibrin
### Table 19.4 Coagulation Factors

<table>
<thead>
<tr>
<th>Number*</th>
<th>Name(s)</th>
<th>Source</th>
<th>Pathway(s) of Activation</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV</td>
<td>Calcium ions (Ca(^{2+})).</td>
<td>Diet, bones, and platelets.</td>
<td>All.</td>
</tr>
<tr>
<td>V</td>
<td>Proaccelerin, labile factor, or accelerator globulin (AcG).</td>
<td>Liver and platelets.</td>
<td>Extrinsic and intrinsic.</td>
</tr>
<tr>
<td>VII</td>
<td>Serum prothrombin conversion accelerator (SPCA), stable factor, or proconvertin.</td>
<td>Liver.</td>
<td>Extrinsic.</td>
</tr>
<tr>
<td>VIII</td>
<td>Antihemophilic factor (AHF), antihemophilic factor A, or antihemophilic globulin (AHG).</td>
<td>Platelets and endothelial cells.</td>
<td>Intrinsic.</td>
</tr>
<tr>
<td>IX</td>
<td>Christmas factor, plasma thromboplastin component (PTC), or antihemophilic factor B.</td>
<td>Liver.</td>
<td>Intrinsic.</td>
</tr>
<tr>
<td>X</td>
<td>Stuart factor, Prower factor, or thrombokinase.</td>
<td>Liver.</td>
<td>Extrinsic and intrinsic.</td>
</tr>
<tr>
<td>XI</td>
<td>Plasma thromboplastin antecedent (PTA) or antihemophilic factor C.</td>
<td>Liver.</td>
<td>Intrinsic.</td>
</tr>
<tr>
<td>XII</td>
<td>Hageman factor, glass factor, contact factor, or antihemophilic factor D.</td>
<td>Liver.</td>
<td>Intrinsic.</td>
</tr>
</tbody>
</table>
Initiated by trauma to the vascular wall

Extrinsic Pathway

Tissue damage

Prothrombin activator

Prothrombin

Fibrinogen

Fibrin

Thrombin

VIIa

Ca

PF$_3$

Xa

V

Tissue factor

VII
Intrinsic Pathway

Only uses components found within blood itself or exposure of the blood to collagen

XII
  \[ \text{Contact} \]

XI → IX → VIII → X

Prothrombin activator

Ca\(\text{PF}_3\) → Xa

V

Prothrombin

Fibrinogen

Thrombin

Fibrin
Intrinsic pathway:
- XIIa
  - XIa
    - IXa
      - VIIIa
        - Xa
          - Ca
            - PF$_3$
              - Prothrombin activator
                - Prothrombin
                  - Fibrin

Extrinsic Pathway:
- TF
  - VIIa
    - Xa
      - V
        - Prothrombin activator
          - Prothrombin
            - Thrombin
              - Fibrinogen
Intrinsic pathway:
- XIIa
- XIa
- IXa
- VIIIa

Extrinsic Pathway:
- TF
- VIIa

Prothrombin

Xa

VIIIa

Va

V

Fibrinogen

Thrombin stimulates formation of fibrin stabilizing factor (XIII), factor V and factor VIII

Fibrin

Soft clot

Hard clot
Screening assays in hemostasis:
1. Patients without any signs/symptoms → preoperative
2. Monitoring of anticoagulant therapy
3. Disseminated Intravascular Coagulation
4. Thrombophilia
5. Inhibitor (Lupus Anticoagulant, Anti Phospholipid Antibody)
<table>
<thead>
<tr>
<th></th>
<th>Hemostasis Screening Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tourniquet Test</td>
</tr>
<tr>
<td>2</td>
<td>Bleeding Time</td>
</tr>
<tr>
<td>3</td>
<td>Clotting Time</td>
</tr>
<tr>
<td>4</td>
<td>Clot Retraction</td>
</tr>
<tr>
<td>5</td>
<td>Platelet Count*</td>
</tr>
<tr>
<td>6</td>
<td>PT*</td>
</tr>
<tr>
<td>7</td>
<td>APTT*</td>
</tr>
<tr>
<td>8</td>
<td>TT*</td>
</tr>
<tr>
<td>9</td>
<td>Fibrinogen*</td>
</tr>
<tr>
<td>10</td>
<td>Euglobulin Clot Lysis Test</td>
</tr>
<tr>
<td>11</td>
<td>D-Dimer</td>
</tr>
</tbody>
</table>

Thrombelastography
TORNIQUET TEST

= RUMPEL LEEDE TEST
= HESS TEST

Wait for 5 min

Systolic/diastolic

100 mmHg

PETECHIAE

: ≥ 10 → ABNORMAL

Rough capillary fragility evaluation

ABNORMAL:
- TROMBOCYTOPENIA
- PURPURA
- VON WILLEBRAND DISEASE
1. **DUKE METHOD**: earlobe

- Clean with 70% alcohol
- Puncture with special blood lancet

- Touch blood drop with filter every 30 seconds
- BT = number of blood drops in filter paper X 30 seconds
  - If BT > 10 min → stop the test

**N**: 1 - 3 min
Thank You!