3. Thrombocytes (Platelets)

small, irregular shape cell fragments

2-4 µm diameter

usually 250,000 – 500,000/mm³

no gender differences

short life span: ~10 days

**Formation**

formed in marrow, lungs and spleen by fragmentation of large cells (=megakaryocyte)

their production is controlled by thrombopoietin

play important role in hemostasis and blood clotting

**Hemostasis**

stoppage of blood flow

include:

- **vascular spasm** reduces blood loss
- **platelet plug** 1-5 seconds after injury
  platelets become sticky

<table>
<thead>
<tr>
<th>platelets swell</th>
<th>develop spiky processes</th>
<th>become sticky → adhere tenaciously</th>
<th>degranulate → release serotonin &amp; thromboxane</th>
<th>→ enhance vascular spasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>aggregating agens attract more platelets</td>
<td></td>
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prostaglandins may be involved
**Blood Clotting**

if injury is extensive clotting cascade is initiated

mechanism must be rapid to stop bleeding

\[ \rightarrow \text{involve over 30 different chemicals} \]

\[ \rightarrow \text{each is activated in a rapid sequence} = \text{cascade (positive feedback)} \]

1. trigger: rough spot in lining of blood vessel
   slow blood flow
   (also, bedridden)

2. clumps of platelets adhere to site (1-2sec)

3. platelets release serotonin and thromboxane
   \[ \rightarrow \text{constricts blood vessels at site of injury} \]

4. platelets and damaged tissues release chemical
   (=thromboplastin, = prothrombin activator)

5. prothrombin (an inactive albumin)
   \[ \rightarrow \text{becomes thrombin} \]

6. thrombin converts fibrinogen to fibrin
   (fibrinogen – soluble protein)
   (fibrin – insoluble protein)
   
   fibrin is a protein forming fine threads that tangle together forming a clot

**clot retraction**

30-60 minutes

draws edges of clot together

**fibrinolysis**

\[ = \text{clot dissolution} \]

\[ \text{occurs continuously} \]

\[ \text{plasmins & fibrolysin} = \text{clot busters} \]

**Thrombocyte Disorders**

1. **Spontaneous Clotting**

   body has mechanism that prevent spontaneous clotting without vessel damage:
- normal lining of vessels is smooth
  → platelets do not adhere

- blood also contains antithrombins
  → inactivate thrombin
    eg. heparin (a natural blood constituent)

sometimes clots are triggered by internal factors

two conditions favor clots:
  1. rough spots on blood vessels
     atherosclerosis may trigger clotting
  
  2. abnormally slow flow of blood
     bedridden or immobilized patients

these may be caused by:
  atherosclerosis
  severe burns
  inflammation
  slow flow

thrombus – a fixed persistent clot
embolism – a traveling clot

2. Bleeding Disorders (=Hemophilias)
   inability of blood to clot in normal amount of time
   may be caused by
     decreased # of platelets
     liver disease
     inability to form various clotting factors

prothrombin and fibrinogen are produced in liver
require vitamin K (absorbed from intestine)
vitamin K requires bile to be digested and absorbed

if bile ducts become obstructed results in vitamin K deficiency
  → liver can't produce prothrombin at normal rate

  eg. factor VII
    comprises 83% of cases
  eg. factor X
    a sex linked condition