Physiology Team 439

MED439
KING SAUD UNIVERSITY

Revised & Approved
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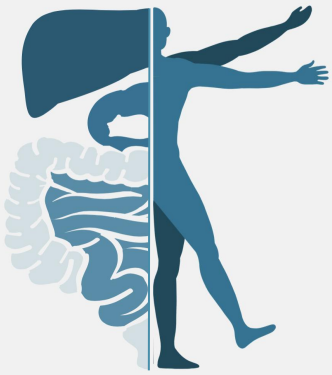


Platelet structure and functions

Objectives:

- Describe formation and development of platelets.
- Understand platelet normal ultrastructure
- Describe the functions of different platelets organelles and surface receptors
- Describe the mechanisms of platelet functions
- Relate membrane receptors and granule content to normal function in hemostasis and bleeding (platelet) disorders

Color index:

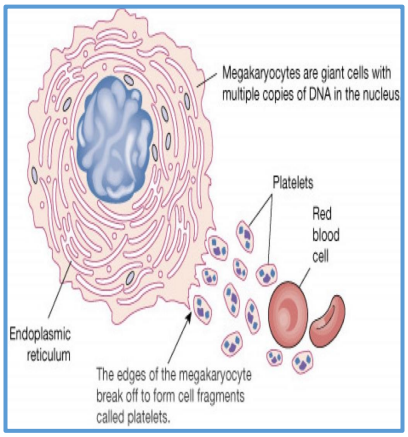
- Important.
- Girls slide only.
- Boys slide only.
- Dr's note.
- Extra information.

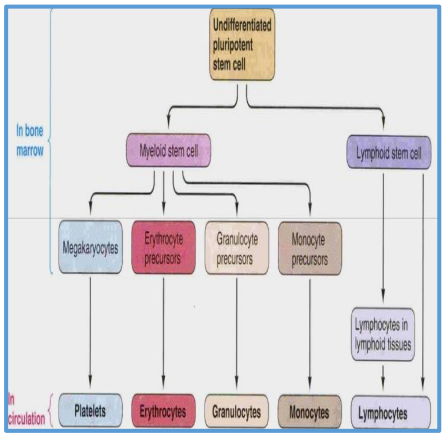
Editing File

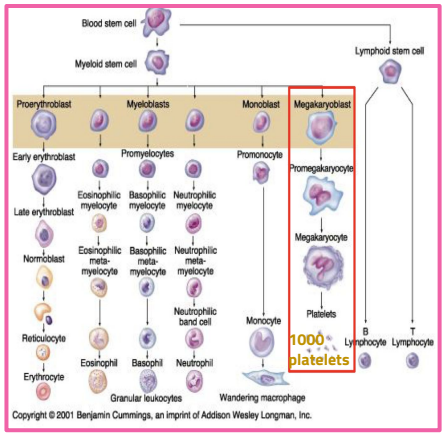
Platelets

Formation

- What is difference between platelets and other blood cells ?
  - size: platelet is smaller than RBC.
  - amount: platelets are less than RBCs.
- What is difference between platelets and other blood cells in terms of function?
  -RBC = blood
  -WBC= مناعة
  -platelets = خلية متحولة تتغير وتتحول لخلية مختلفة من الخواص ولكن على درجة عالية من التنظيم
- Platelets Formation (Thrombopoiesis)
- Regulation of thrombopoiesis by Thrombombopoietin produced from liver, target ? Work on megakaryocytes by increase it, and increase number of platelets produced from each megakaryocytes.
- Site of formation: Bone marrow
- Steps: Stem cell → Megakaryoblast → Megakaryocyte → Platelets
- Formed by fragmentation from Megakaryocytes







- نعرف هذه الخطوات بالمحددة بالأحمر، و megakaryocyte الخلية الكبيرة تنقسم لأجزاء صغيرة وتعطينا platelets، لكن ما تتحول.

Thrombocyte

- Anuclear and discoid cell (resting) → spherical when activated
- Shape: minute round or oval discs
- Platelet count = 150 x10^3-300x10^3/ml
  150,000 – 300,000/ microlitrer
- Size: 1.5–3.0 µm in diameter
  تقريبًا ٣-٤ لكل RBCs
- Life span: 7–10 days
- Location: Sequestered in the spleen; 80% in the blood, and 20% in the spleen

ايش الفائدة ؟
في حالات الطوارئ فيه platelets في ال circulation لكن نحتاج زيادة وما فيه وقت للتصنيع، يصير جاهز ومصنع في spleen

- hypersplenism may lead to low platelet counts.
- Contractile, adhesive, cell fragments
- Store coagulation factors & enzymes
- Surface binding antigens glycoproteins

Functional Characteristics

Doctor Q: What are the main functional characteristics of platelets?

- **Motile**: Actin and myosin molecules
- **Active**: Endoplasmic reticulum, Golgi apparatus & mitochondria
- **Enzymes system**: such as for synthesis of prostaglandins
- Granules (α and δ):
  - **Alpha Granules:** Coag factors (eg: fibrinogen, vWF), PDGF, Chemokines
  - **Dense or δ Granules**: ADP, Ca+, Serotonin

Platelet Ultrastructure

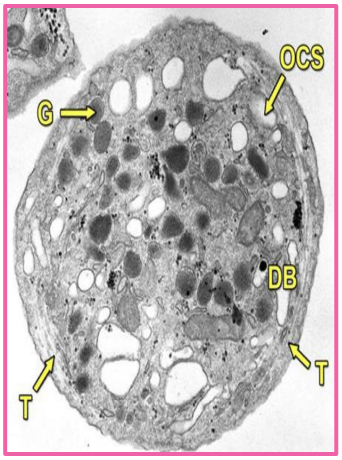
1. **Mitochondria**
2. **Microtubules**
3. **Open canalicular system**
4. **Alpha Granules**:
   - von Willebrand Factor
   - Fibrinogen
   - Chemokines (PF4,etc.)
   - Thrombospondin
   - P-selectin
   - Platelets-derived growth factor

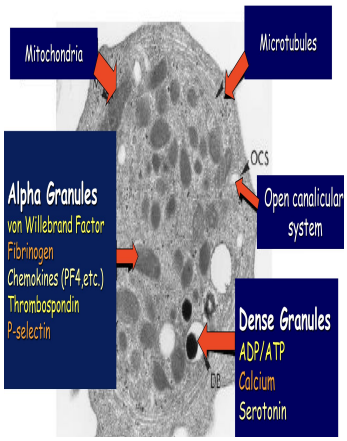
يستخدمونها أطباء الجلدية والتجميل لبلازما الوجه

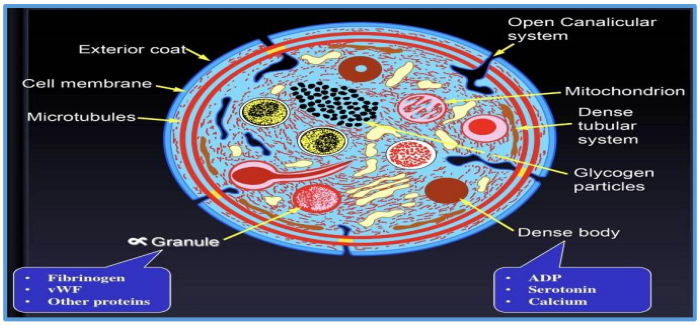
والأماكن الأخرى للنظارة وما الى ذلك، فيستخدمون

مكونات ال platelets.

5. **Dense Granules**:
   - ADP/ATP
   - Calcium
   - Serotonin







Explanation for the picture below:

1. Cell membrane cover with exterior coat (glycoproteins).
Increase surface area to close opening

2. Disc shape, why?

عندها خاصية معينة واللي هي microtubules، عبارة عن tubules تعطي دعامة لل platelets حتى تحتفظ بشكلها disc shape.

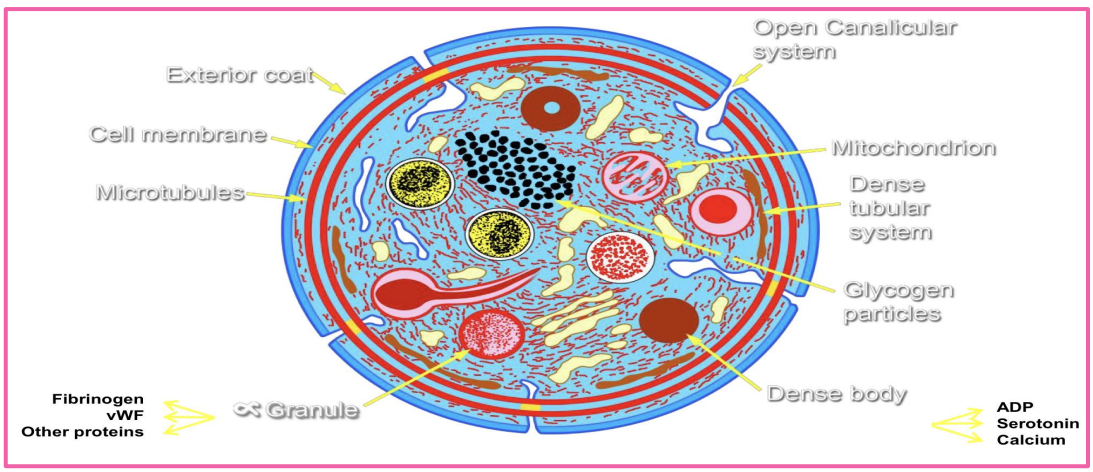
3. No nucleus inside the cytoplasm. Also, it has opening / invaginations to inside the cell membrane called open canalicular system. Function:

- تتحول وتغير شكلها وتطلع لها invagenation ، تطلع للخارج وفائدتها (Increase surface area to close opening).
- any stimulus from blood reach to the cells by these opening.

- اي content داخل platelets يطلع برا عن طريقها.
فهذا system عبارة عن قنوات متصلة ببعض.

4. Has many organelles : mitochondria and glycogen particles. Also, 2 important granules, dense body (4-8 in each cell + غامقة) and alpha granules (40 - 80 in each cell + فاتحة).

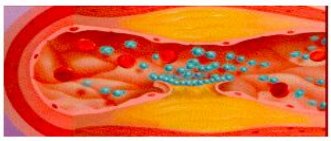
يعني platelets عبارة عن خلية تحمل اكياس تحوي مواد مهمة.



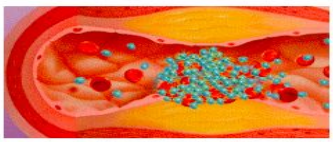
Platelet Function*

1. Adhesion
2. Activation
3. Aggregation
4. Secretion

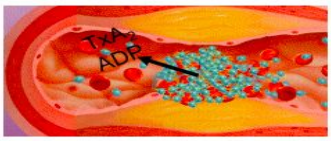
Adhesion



Activation



Aggregation



Secretion

Explanation for the figure above:

1-Adhesion:

بالأحوال الطبيعية ال platelets حذرة وخاملة بال circulation لكن مترقبة فما فيه اي attraction بينها وبين endothelium cell بالعكس تطردها repel.
لكن تحد ال endothelial layer يوجد collagen، وال platelets عندها جاذبية كبيرة لل collagen لكن مين حاجز هم عن بعض ويمنع platlets to be attracted to collagen?
لكن لما يحدث endothelial injury ينكشف ال collagen تجي ال collagen" platelets and attracted to the sub endothelial layer!
طيب ترتبط platelets مع ال collagen عن طريقتين :

Directly : by GP 1a - 6 receptor
Indirectly :vWF عن طريق وسيط > which is in the blood circulation, when endothelium get injured> get out of it and bind platelets with collagen> we call this prosecco adhesion.

بعد ال adhesion تبدأ المرحلة الأخرى وتتحول ل active cell، ايش معناها؟
Change its shape to globular in shape >(open canalicular system) وتطلع لها اذرع > called **protrogens**.
زي الجيب حق الملابس فيه حيز لجوا، لكن لما تطلعينه يصير بارز،
and once the platelets is outside, it becomes sticky

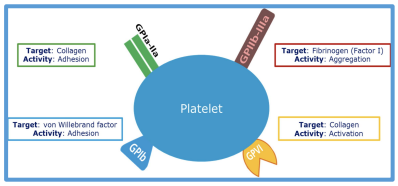
2-Aggregation: Biochemical reaction > (receptors) ال تطلع > become sticky > another platelets will come and interact with it, this interaction we call it aggregation

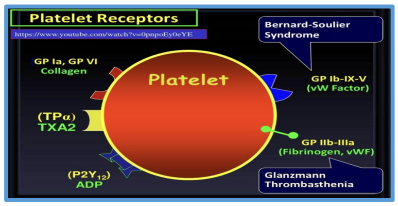
3-Secretion:

مو قلنا ان ال platelets تحمل أكياس جوا؟محملة بمواد مهمة ADP منها، بعد ال activation
Wil secret and release dens body and alpha granules by : actin and myosin contraction squeeze the platelets> تطلع مكوناتها .
Tubules will help them.

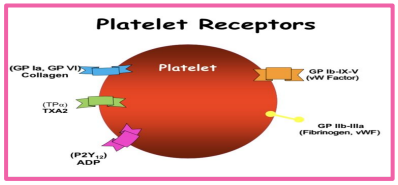
Platelet Receptors

Receptor	Target (for binding with)	Activity
GP Ia-IIa, GP VI Glycoprotein 1a, glycoprotein 6	Collagen	Adhesion
GP Ib - IX - V Glycoprotein 1b-9-5	Von willebrand Factor (vWF)	Adhesion
TPα	Thromboxane A2 (TXA2)	-
$P2Y_{12}$	ADP	-
GP IIb-IIIa Glycoprotein 2b-3a	Fibrinogen, vWF	Aggregative





Doctor: remember the receptors related to diseases (IX-V and IIb-IIIa)



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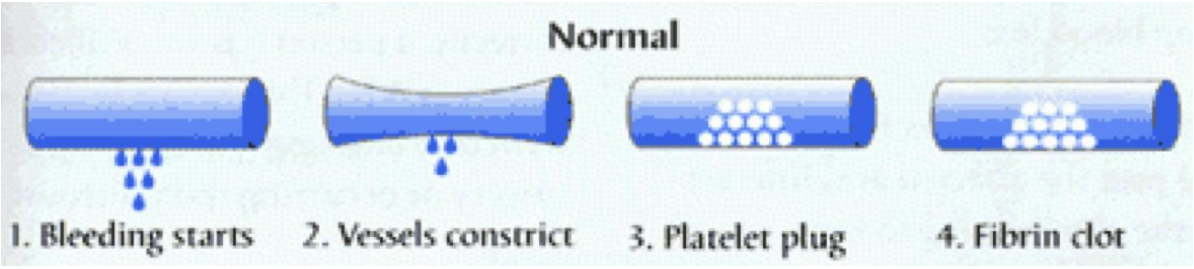
Hemostasis

Phases:	Hemostatic Mechanisms:
1-Vascular phase 2-Platelet phase 3-Coagulation Phase 4-Fibrinolytic phase	1-Vessel wall 2-Platelet 3-Blood coagulation 4-Fibrinolytic system

Hemostasis: Stop bleeding/ spontaneous arrest of bleeding

الانسان الطبيعي في كل يوم يصير له injury لل capillaries and small blood vessel ، ما نشعر فيه لانه يتصلح ب platelets ، زي ما قلنا خلية متحولة منظمة تسكر injury بدون ما نشعر. ولما يكون جرح خارجي ويكون في نزيفا تلعب platelets دور فيه.



Platelet Activation

Doctor Q: what are the 4 steps of platelet activation?. He said 4 since "shape change" isn't in the boys slides

Adhesion → Shape Change → Aggregation (Needs Fibrinogen) → Release Reaction → Clot Retraction

لازم تفرقون بين شيئين function and mechanism :

- function : to form the primary hemostatic plug, how? By the mechanism

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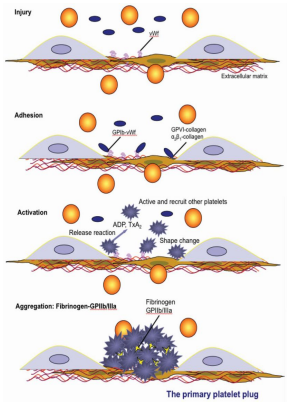
Platelet haemostatic plug formation

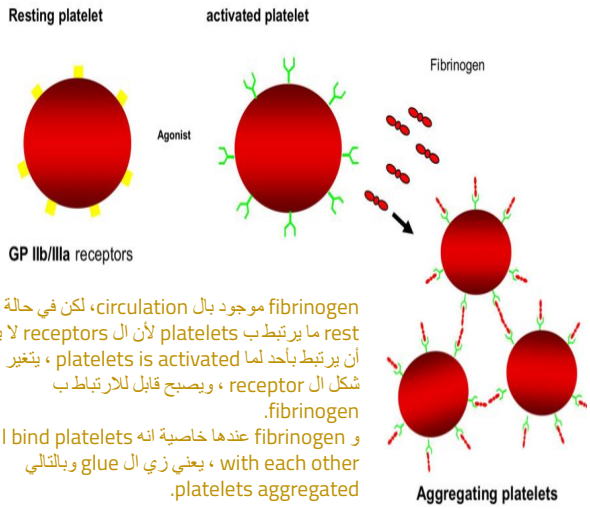
- Platelets activated by adhesion
- Extend projections to make contact with each other
- Release: thromboxane A2, serotonin & ADP >>> activating other platelets
- Serotonin & thromboxane A2 are vasoconstrictors decreasing blood flow through the injured vessel.
- ADP causes stickiness and enhances aggregation



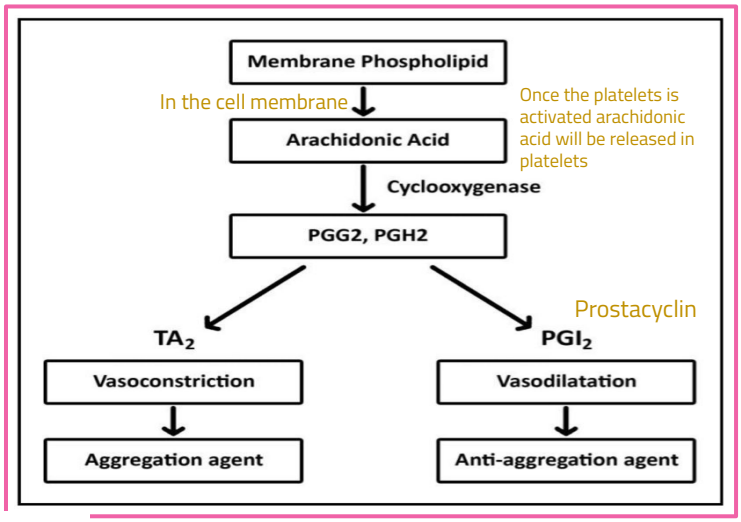
Platelet Aggregation

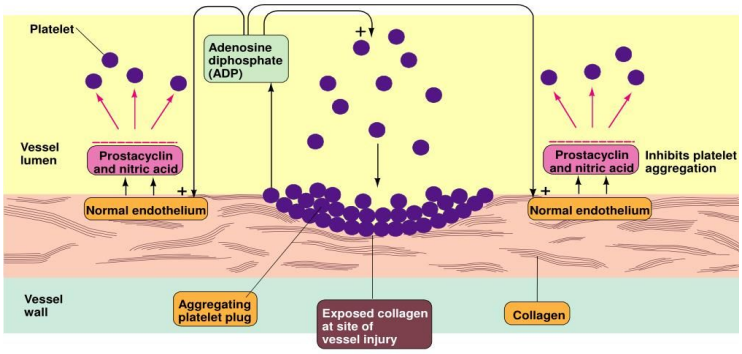
Platelet Aggregation

Fibrinogen is needed to join platelets to each other via platelet fibrinogen receptors



fibrinogen موجود بال circulation، لكن في حالة rest ما يرتبط ب platelets لأن ال receptors لا يقبل أن يرتبط بأحد لما platelets is activated ، يتغير شكل ال receptor ، ويصبح قابل للارتباط ب fibrinogen.
و fibrinogen عندها خاصية انه will bind platelets with each other ، يعني زي ال glue وبالتالي platelets aggregated.





Normal (intact) endothelium secrets

- Prostacyclin(PGI2)
- NO

Inhibit aggregation

- ADP phosphatase:

Any activated platelets release ADP and TXA2> activate more platelets > aggregation> primary hemostatic plug > example of +ive feedback.

Secretions of Activated Platelets

Doctor Qs: -Name 3 platelet secretion and their functions
-Which platelet secretion is inhibited by aspirin?

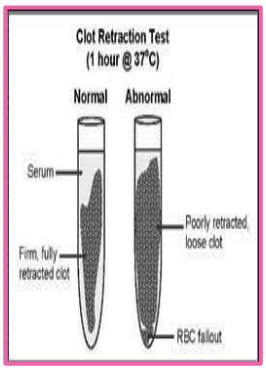
Secretion	Effect
ADP ADP and TXA2 are very important to activate platelets	Adhesion
5HT	Vasoconstriction
Platelet phospholipid (PF3)	Clot formation
Thromboxane A2 (TXA2) ❖ is a prostaglandin formed from arachidonic acid ❖ Inhibited by aspirin (inhibit the TXA2, so prevent vasoconstriction and aggregation)	❖ Vasoconstriction ❖ Platelet aggregation

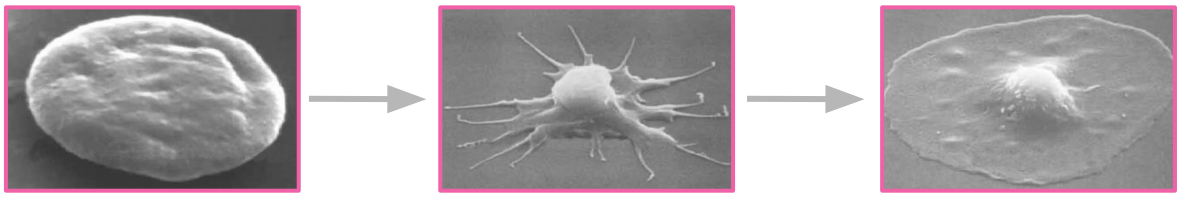
Clot Retraction

Platelet Retraction (clot retraction)

- Myosin and actin filaments in platelets are stimulated to contract during aggregation further reinforcing the plug and help release of granule contents
- When clot retracts (contracts), it expresses most of the fluid from the clot within 20-60 min called → Serum
- Serum cannot clot
- Role of platelets in clot formation & retraction→ they are contractile.
- **Fate of Clot**:

Lysis or Fibrous tissue Formation (platelet derived growth factor)





Inactivated platelets — Activated platelets — Activated platelets
بمراحل متقدمة تسمى spread platelets

Role of Platelet in blood Coagulation*

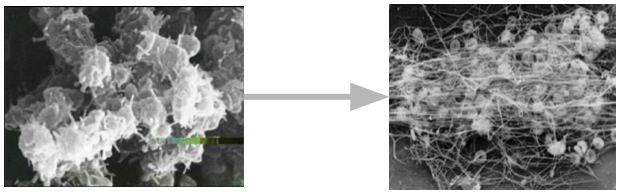
(The cell based model of blood coagulation)

Platelet function: Maintenance of vascular integrity

1- Initial arrest of bleeding by platelet plug formation (picture A)

2- Stabilization of hemostatic plug by contributing to fibrin formation (Picture B)

An adequate number and function of platelets is essential to participate optimally in hemostasis. مهم جدًا

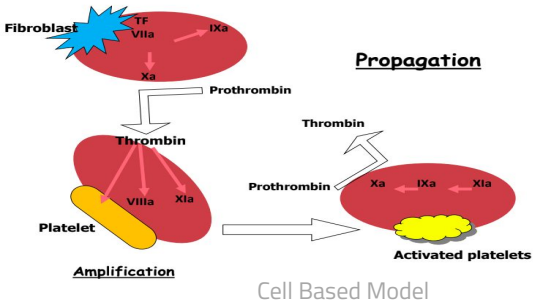


Picture A — Picture B

لأن Plug is weak > لو احد حركها يرجع النزيف مرة ثانية، فلازم تصير اقوى، كيف ؟

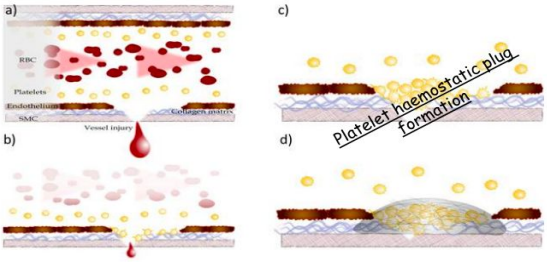
By fibrin formation

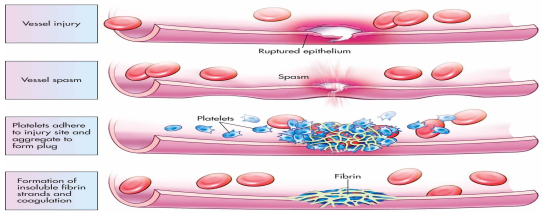
واللي هو زي الشبكة تحاوط plug وتخلي ال platlets كلها بمكان واحد، يعني زي الاسمنت يثبت الاحجار.



Cell Based Model

Coagulation reaction > ما يصير في(bloodstream) > So, platelets provide a surface for this reaction. After platelets activation and cell membrane will provide receptors for reaction to occur.





Extra

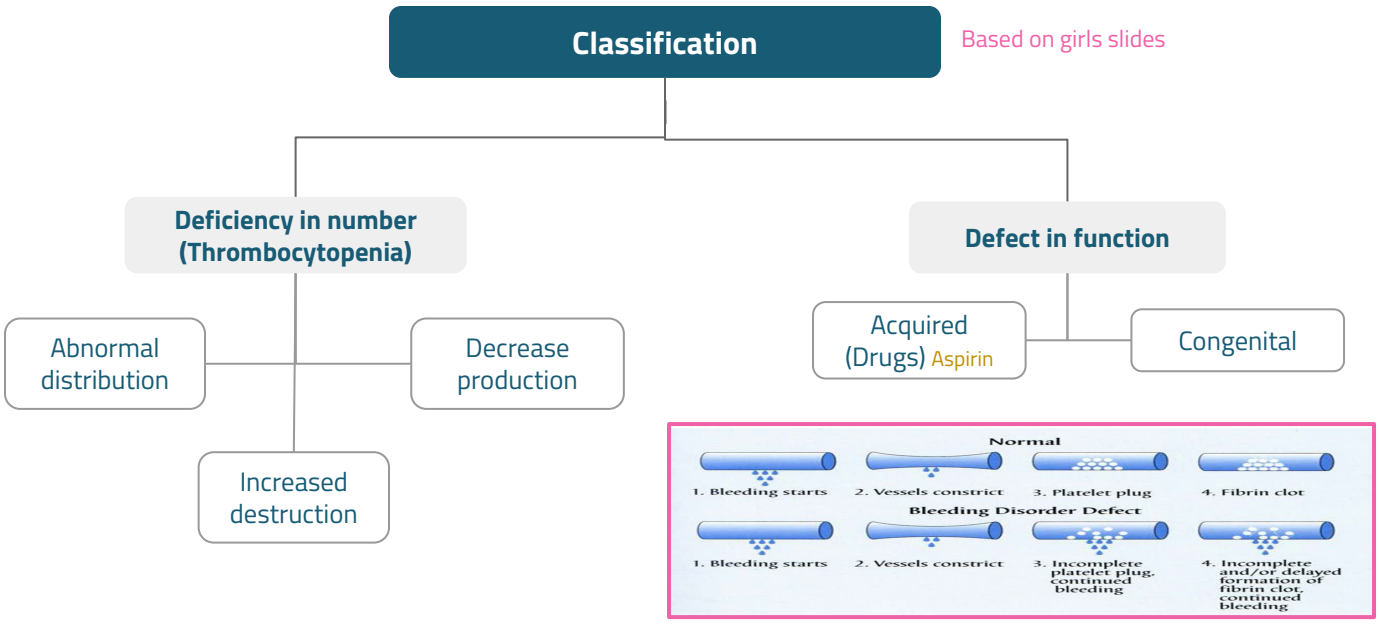
Platelet Activation-Summary*

1	Platelets are activated when brought into contact with collagen exposed when the endothelial blood vessel lining is damaged.
2	Activated platelets release a number of different coagulation and platelet activating factors.
3	Transport of negatively charged phospholipids to the platelet surface; provide a catalytic surface for coagulation cascade to occur.
4	Platelets adhesion receptors (integrins): Platelets adhere to each other via adhesion receptors forming a hemostatic plug with fibrin.
5	Myosin and actin filaments in platelets are stimulated to contract during aggregation further reinforcing the plug and help release of granule contents.
6	GPIIb/IIIa: the most common platelet adhesion receptor for fibrinogen and von Willebrand factor (vWF).

Physiological Factors Affecting Platelet Count*

1. Increase in injury
2. Increase with adrenaline.
3. Increase with hypoxia.
4. Age: decrease in newborn.
5. Menstrual cycle: decrease prior menstruation and increase after it.
6. Decrease in pregnancy
7. Decrease with smoking
8. Decrease with nutritional deficiencies. Eg: vitamin B12, folic acid and iron.

Bleeding Disorders



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1-Thrombocytopenia

highlighted مطالبين فقط بال examples, غير مطالبين بباقي الأمثلة

Type	Decreased Production	Increased destruction	Abnormal distribution
Causes	Various anemias		Splenomegaly with sequestration in the spleen
	Leukemia or lymphoma	Autoimmune diseases: **Idiopathic (immune) thrombocytopenic purpura** -Pregnancy: about 5% of pregnant women develop mild decrease Thrombotic thrombocytopenic purpura	
	Cancer treatments such as radiation or **chemotherapy**	Surgery: man-made heart valves, blood vessel grafts, bypass machines	
	Medications : diuretics, chloramphenicol	Medications :quinine, antibiotics containing sulfa, Dilantin®, vancomycin, rifampin, heparin-induced thrombocytopenia	
	Infections (Viruses): chickenpox, mumps, Epstein-Barr, parvovirus, AIDS	Infections: septicemia	
	Toxic chemicals	Disseminated intravascular coagulation	
	Alcohol in excess		
	Genetic conditions: Wiskott-Aldrich, May-Hegglin.		

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Pseudothrombocytopenia

Pseudothrombocytopenia: It is a relatively uncommon phenomenon caused by in vitro agglutination of platelets The phenomenon occurs when the anticoagulant used while testing the blood sample causes clumping of platelets.
As a result of platelet clumping, platelet counts reported by automated counters may be much lower than the actual count in the blood because these devices cannot differentiate platelet clumps from individual cells leading to the misdiagnosis of thrombocytopenia

Causes of Pseudothrombocytopenia:
-Partial clotting of specimen
-EDTA-platelet clumping
-Platelet satellitism around WBCs
-Cold agglutinins
-Giant platelets

2-Congenital platelet disorders

highlighted examples مطالبين فقط بال, غير مطالبين بباقي الأمثلة (female dr)

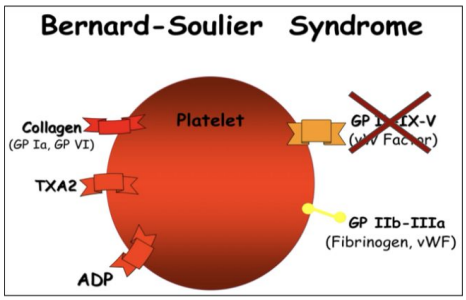
Disorder	Adhesion	Aggregation	Granules	Production	Primary Secretion	Cytoskeleton
Syndrome	**Bernard-Soulier**	**Glanzmann thrombasthenia**	Grey Platelet Syndrome. (α granule deficiency.	Congenital amegakaryocytic thrombocytopenia	Receptor defects (TXA2, collagen ADP, epinephrine)	Wiskott-Aldrich syndrome
			Storage Pool deficiency.	MYH9 related disorders.		
			Hermansky-Pudlak syndrome	Thrombocytopenia with absent radii (TAR).		
			Chediak-Higashi syndrome.	Paris-Trousseau/ Jacobsen.		

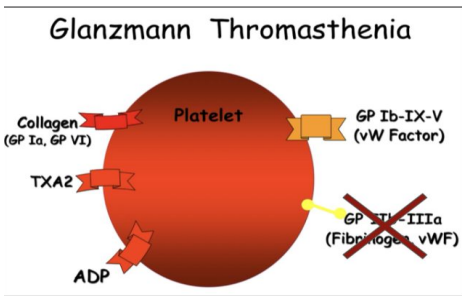
Bernard-Soulier Syndrome

In Bernard-Soulier Syndrome, there is deficiency of vW factor receptors causing defects in adhesion.
No GP 1b-9-5 receptor > (adhesion)مشكلة في ال

Glanzmann Thromasthenia

In Glanzmann Thrombasthenia, there is deficiency of fibrinogen receptors causing defects in aggregation.
No GP 2b-3a receptor > (aggregation)مشكلة في ال





Laboratory Testing of Platelet Function

How to investigate for a platelet disorder?
by Laboratory Testing of platelet function

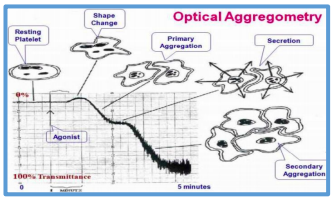
Laboratory tests include:

1. Peripheral smear (Blood Smear) and platelet count (& shape)
2. Electron-microscopy ultrastructure of platelets
3. Bleeding time **(Duke method)**
   If Prolonged = platelets dysfunction
   بدون ما يقول وش هي بالضبط
4. Platelet Function Analyzer (PFA-100) (Automated) replacing bleeding time
5. Flow-cytometry
6. Granule release products
   More detailed and more specific test
7. Platelet Aggregation
   Gold standard test for platelets aggregation
   نشوف المشكلة بأي receptor، نجيب الدم ونعمل له centrifuge ونفصل ال RBCs عن ال (plasma
   agonist مصنعة ونحطها بالجهاز ، نجيب (platelets rich plasma
   stimulate the platelets.
   الفكرة الرئيسية نعمل activation لل platelets:
   -normal > activation and form plug.
   -abnormal > no plug formation.


Bleeding time test


PFA


Platelet Aggregometry



Platelet Aggregation

Bleeding time: is a medical test that measures how fast small blood vessels in the skin stop bleeding. The bleeding time test is used to evaluate how well a person's blood is clotting. The test evaluates how long it takes the vessels cut to constrict and how long it takes for platelets in the blood to seal off the hole
Duke method: the patient is pricked with a special needle or lancet, preferably on the earlobe or fingertip, after having been swabbed with alcohol. The prick is about 3–4 mm deep. The patient then wipes the blood every 30 seconds with a filter paper. The test ceases when bleeding ceases. The usual time is about 2–5 minutes.
Platelet Function Analyzer: It performs an in vitro test of platelet plug formation.
Flow cytometry: is a technique used to detect and measure physical and chemical characteristics of a population of cells or particles. In this process, a sample containing cells or particles is suspended in a fluid and injected into the flow cytometer instrument. The sample is focused to ideally flow one cell at a time through a laser beam, where the light scattered is characteristic to the cells and their components.

Lab Tests In Bleeding and Clotting

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Test	Normal Value	Importance
PLATELET COUNT	100,000 - 400,000 CELLS/MM³	Thrombocytopenia
PLATELET FUNCTIONS	Normal Aggregation	Thrombocytopathy (normal count) [Congenital or Acquired...Aspirin]
BLEEDING TIME (BT)	2-8 MINUTES	Bleeding disorders
PROTHROMBIN TIME (PT)	10-15 SECS	Measures Effectiveness of the Extrinsic Pathway
PARTIAL THROMBOPLASTIN TIME (PTT)	25-40 SECS	Measures Effectiveness of the Intrinsic Pathway
THROMBIN TIME (TT) $INR = \left(\frac{PT_{test}}{PT_{normal}}\right)^{ISI}$	9-13 SECS	A Measure of Fibrinolytic Pathway Time for Thrombin To Convert Fibrinogen ► Fibrin

Platelet Aggregation in Platelet Rich Plasma (PRP)

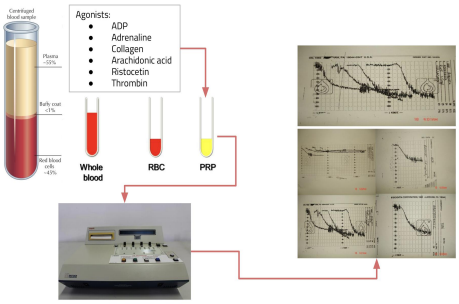
By Platelet Aggregation Method: It provides information on time course of platelets activation.

Agonists:

- ADP
- Adrenaline
- Collagen
- Arachidonic acid
- Thrombin
- Ristocetin→ need vWF > (aggregation) بدونه ما يعطيني

Need GP 2b-3a receptor to make aggregation

(Reference ranges need to be determined for each agonist)



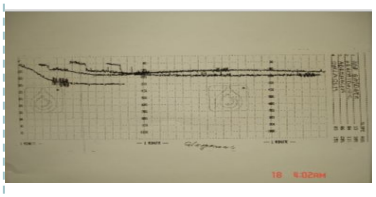


Figure A

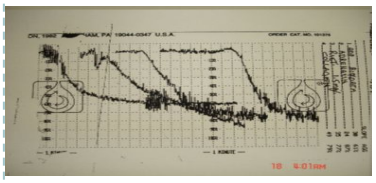


Figure B

Figure A:

لو عطاني خط مستقيم بعد ما حطيت ال agonist=

Low or No activation and aggregation of platelets. Which is abnormal.

Figure B:

لو عطاني curve بعد ما حطيت agonist= معناها ال receptor موجود ، يعني فيه aggregation

لما يحصل activation ينزل ال curve ، وكلما زادت ال amplitude of the wave، زاد ال aggregation.
وهذا الطبيعي.

معناها لما حطيت ADP and arachiodonic acid < aggregation

ما استجاب لل ADP> = No GP 2b-3a
ما استجاب لل arachiodonic acid < عند اللي يأخذون aspirin

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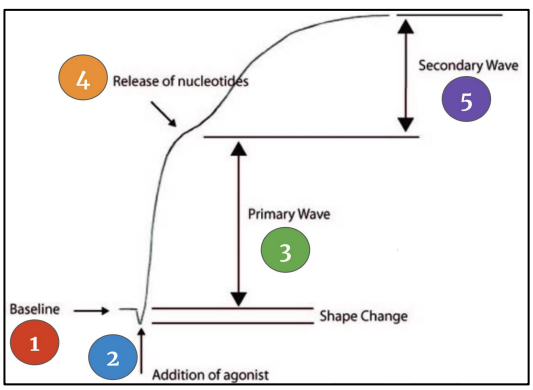
Classic Biphasic Aggregation

1- Baseline.
2- Addition of an agonist.
(this results in a change in platelet
change and hence a drop in the baseline absorbance)
3- Primary wave aggregation.
4- Release of nucleotides.
5- Secondary wave aggregation.

(Adrenaline and low dose ADP classically give a biphasic aggregation)



Aggregometry Results and Diagnosis

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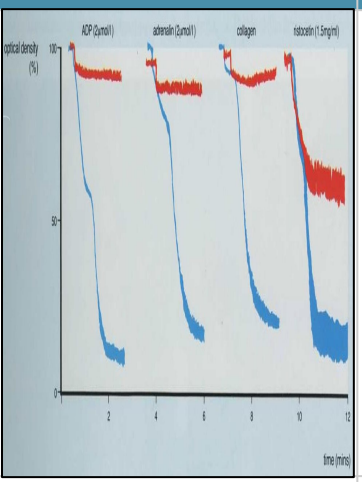
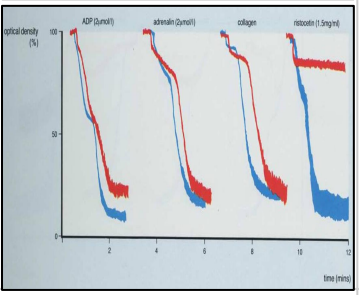
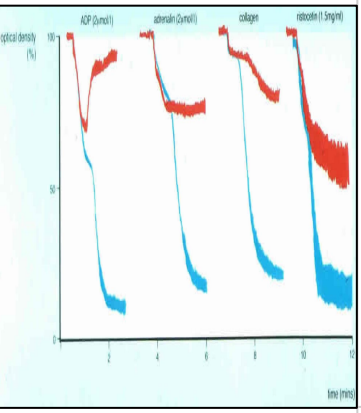
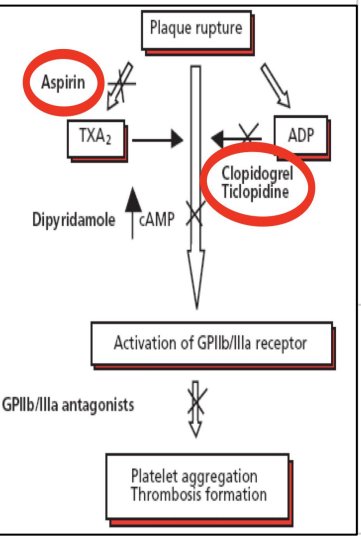
Figure	Characteristic of findings on LTA	Diagnosis
	Absent or markedly impaired aggregation to all agonists except ristocetin.[and this is not complete] (Ristocetin-induced agglutination shows only primary wave aggregation, aggregation cannot occur because fibrinogen cannot bind) There is no aggregation with ADP, adrenaline or collagen. [Remember, platelet agglutination with Ristocetin occurs independently of Fibrinogen.]	• Glanzmann's thrombasthenia • Afibrinogenemia
	Absent or markedly reduced platelet agglutination with Ristocetin.	• Bernard soulier syndrome • Von willebrand disease
	Primary(first) wave aggregation only with ADP, Adrenaline and collagen and only partial agglutination with ristocetin The picture is clearly different from the two traces above 1) or 2): the results suggest a failure of granule release suggesting failure of granule release or a deficiency of platelet granules.	• Platelet storage pool Disorder. • Platelet release defects (defect in nucleotide release)
	Absent aggregation to Arachidonic acid. Primary wave aggregation only with ADP. Decreased or absent aggregation with collagen.	Aspirin (or defects on COX pathway) Aspirin inhibits platelet cyclooxygenase by irreversible acetylation, thereby preventing the formation of thromboxane A2, which is powerful stimulant of platelet aggregation.
	Absent aggregation with ADP	Clopidogrel (ADP inhibitor) Clopidogrel a thienopyridine, acts by inhibiting adenosine receptors, which inhibits the early step of platelet activation.

Summary*

Platelets are cell fragments derived from megakaryocyte in the bone marrow.

Platelets play a pivotal role in haemostasis

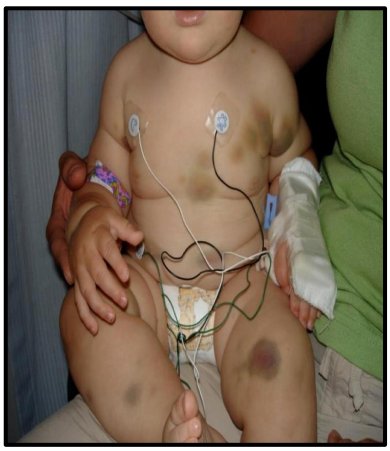
By arresting bleeding from an injured blood vessels

Bleeding can result from: Platelet defects acquired or congenital

Platelet function tests are used to detect abnormal platelet function.

Case Study (From Slides)

A 7 years old girl complaining of severe bruising since birth and if she had injury she would bleed for days. She had epistaxis which lasted for days.
Her mother said "she just bruise more easily than her older sister".



Investigation:

- **CBC:** RBC, WBC, Platelets. (All normal)
- **Platelet morphology**: normal.
- **Aggregometry**: Absent platelet aggregation in response to ADP, collagen, thrombin, and epinephrine. (GP2b-3a)معناها ما عندها بما انه اعطانا خط مستقيم 1 figure >

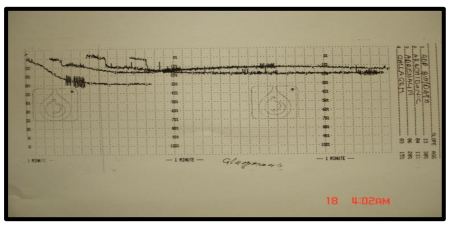


Figure 1: we give her ADP, collagen, thrombin, and epinephrine.

لكن لما عملنا Ristocetin طلع فيه aggregation > لأن ال curve نزل figure 2، وهو يعتمد على vWF ، فيطلع المرض اللي عندها Glanzmann's Thrombasthenia

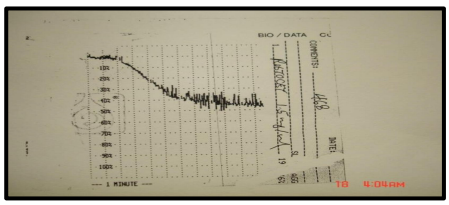


Figure 2: we give her Ristocetin.

If Ristocetin is abnormal = خط مستقيم
والباقي يعطوننا curve > مشكلة في ال adhesion

Diagnosis

Glanzmann's Thrombasthenia
(Defects in aggregation)

MCQ & SAQ:

Q1: The regulation of Platelet production is done by:

A. Thrombopoietin
B.Thrombin
C. Fibrin
D. A&B

Q2: Thrombocytopenia due to increased destruction:

A. HIV
B. Pregnancy
C. Chemotherapy
D. Leukemia

Q3: von Willebrand Factor is released from:

A. Dense Granules
B. Alpha Granules
C. Open canalicular system
D. Both A&B

Q4: Normal Platelets Count:

A. (100-200) x103/ml
B. (200-400)) x103/ml
C. (15-30)) x103/ml
D. (150-300) x103/ml

Q5: Platelet phospholipid (PF3) effect is:

A. Adhesion
B. Clot formation
C. Vasoconstriction
D. Aggregation

Q6: Which of the following is not a normally secreted from the endothelium

A. NO
B. ADP phosphate
C. Prostacyclin(PGI2)
D. Collagen

answer key:
1:A
2:B
3:B
4:D
5:B
6:D

1-What are the phases of Hemostasis?

2-A teenage boy with numerous nosebleeds was referred to a physician for evaluation prior to a minor surgery. His prothrombin time(PT) was 11 secs (11-15sec normal), partial thromboplastin time(PTT) was 58 secs (25-40sec normal), and bleeding time was 6.5 min (2-7 min normal). Which of the following is most likely abnormal in this young man?

3-How do platelets get activated? (Stages)

4-Mention 2 vasoconstrictors that will decrease blood flow through the injured vessel

A1: 1-Vascular phase 2-Platelet phase 3-Coagulation phase 4-Fibrinolytic phase

A2: Intrinsic pathway

A3: adhesion-shape change-aggression-release reaction-clot retraction

A4: serotonin-thromboxane A2

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