TESTS OF HEMOSTASIS

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Hemostasis

Haemostasis means prevention of blood loss from blood vessels.

Bleeding is stopped by several mechanisms, which are:

- 1. Local vasoconstriction
- 2. Formation of platelet plug
- 3. Blood coagulation

1. Local vasoconstriction

Immediately after injury, constriction of an injured arteriole or small artery may be so marked that its lumen is obliterated. The vasoconstriction is probably due to:

- Liberation of serotonin and other vasoconstrictors from platelets.
- Local myogenic contraction of the blood vessel.

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2. Formation of platelet plug

Platelets adhere to damaged endothelium to form a platelet plug (*primary hemostasis*). Platelets adhere to the collagen fibers of a wound, then release chemical messengers such as adenosine diphosphate (ADP), serotonin and thromboxane A₂, causing more platelets to stick to the area, release their contents, and enhance vascular spasms. As more chemicals are released more platelets stick and release their chemicals; creating a platelet plug.

3. Blood coagulation

Blood coagulation is dependent upon a number of factors, which interact to produce the prothrombin conversion factor, which then convert prothrombin to thrombin and the later convert the fibrinogen to fibrin, which appears to be important in stabilizing the platelet thrombus. Fibrin mesh helps hold the plug in place. Red and white blood cells become caught up in the fibrin mesh which causes the clot to become even stronger. This step of coagulation is referred to as **secondary hemostasis**

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Why is platelet adhesion clinically important?

Platelet adhesion is required for primary hemostasis. Platelets do not adhere to healthy endothelium. Intact endothelial cells secrete antithrombotic substances such as *prostacyclin* (*PGI*), a prostaglandin, vasodilator, and platelet inhibitor. Platelets are also repelled by the negatively charged surface of intact endothelium.

Tests of hemostasis

- 1. Whole blood coagulation time (Clotting time)
- 2. Bleeding time
- 3. Prothrombin time (PT)
- 4. Thrombin time (TT)
- 5. Platelet count (Thrombocytes)

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1. Whole blood coagulation time (Clotting time)

It is the time that elapses from the start of bleeding till the formation of clot.

2. Bleeding time

It is the time needed for bleeding to stop when small injury of skin occurs.

3. Prothrombin time (PT)

It is the time required for clotting to take place in citrated plasma to which optimum amount of thromboplastin and calcium ions have been added. It is prolonged with severe liver disease or vitamin K deficiency.

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4. Thrombin time (TT)

This is the time taken for fibrin to be formed after the addition of thrombin to citrated plasma sample. It is a measure of the amount of fibrinogen present and the ability to convert it to fibrin. A prolonged thrombin time is found with a deficiency of fibrinogen or with difficulty in the production of fibrinogen or the presence of inhibitors such as heparin.

5. Platelet count (Thrombocytes)

Platelets in mammals are fragments that contain small pink-red granules. Shed into the blood from megakaryocytes in bone marrow

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Methods of counting

1. Direct method

- a. Haemocytometer.
- b. Automatic blood cell counter.
- 2. Indirect method: blood film.

The platelets per oil immersion field on a stained blood smear are counted and compared with the number of red or white cells. For example, the number of platelets per 100 white blood cells multiplied by the total white count is an estimate of the platelet count. Another method is to simply count the number of platelets per oil immersion field where one /oil is equivalent to 15,000/ul.

HEMORRHAGIC DISORDERS

- I. Abnormalities of blood platelets
 - a. Thrombocytopenia
 - b. Thrombocytosis
 - c. Thrombocythaemia
 - d. Thrombocytopathia
- II. Haemorrhagic disorders due to defect in the clotting mechanisms

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a. Thrombocytopenia

- Aplasia of the bone marrow.
- Bacterial and viral infections (Bacteremia and hepatitis C)
- Abnormal loss of blood platelets in peripheral circulation (autoimmune disease, severe hemorrhage).
- Sequestration of platelets (Hepatomegaly and splenomegaly)

b. Thrombocytosis

Increase the number of circulating blood platelets, may be transient such as occurring after trauma or during disease process.

c. Thrombocythaemia

Means persistent increase in circulating blood platelets.

Causes:

- Megakaryoblastic tumors.
- Over production of thrombopoietin from the kidney.

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d. Thrombocytopathia

Platelet normal in number but abnormal in function.

These platelets may have a normal or abnormal appearance, they may be:

- Defective in adhesiveness.
- Defective in aggregation.

Laboratory findings:

- Hemorrhagic diathesis.
- Normal levels of blood coagulation factors.
- Normal platelet count.
- ☐ Prolonged bleeding time.

II. Haemorrhagic disorders due to defect in the clotting mechanisms

 An increase in the clotting time, with normal values for the bleeding time and platelet count, indicates existence of haemophilia i. e. a deficiency of one or more of the factors necessary for normal coagulation.

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- An increase in both the clotting time and bleeding time (usually associated with normal level of platelets) most probably indicates a deficiency of prothrombin (Hypoprothrombinemia). This is usually due to either:
- Liver disease as hepatic toxins, hepatitis or obstructive iaundice.
- Warfarin poisoning.
- Vitamin K deficiency.
 - a. Inadequate diet from excessive antibiotics either as medicaments or in feed, which interfere with bacterial synthesis of vitamin K.
 - b. Deficiency of bile, which interfere with absorption of vitamin K.

Summary of changes in some diseases causing increased bleeding tendency

Test	Hypoprothrom -binaemia	Thromb- cytopenia	Haemophilia	Traumatic
Clotting time	Increased	Usually normal	Increased	Normal
Bleeding time	Increased	Increased	Normal	Normal
Thrombocyte count	Normal	Decreased	Normal	Increased
Prothrombin time	Increased	Normal	Usually normal	Normal

