

II. TESTS FOR HEMOSTASIS, CLOTTING TIME (CT) (Coagulation Time; Capillary Blood Clotting Time; Wright's Capillary Glass Tube Method) and Platelet count:

PROCEDURE:

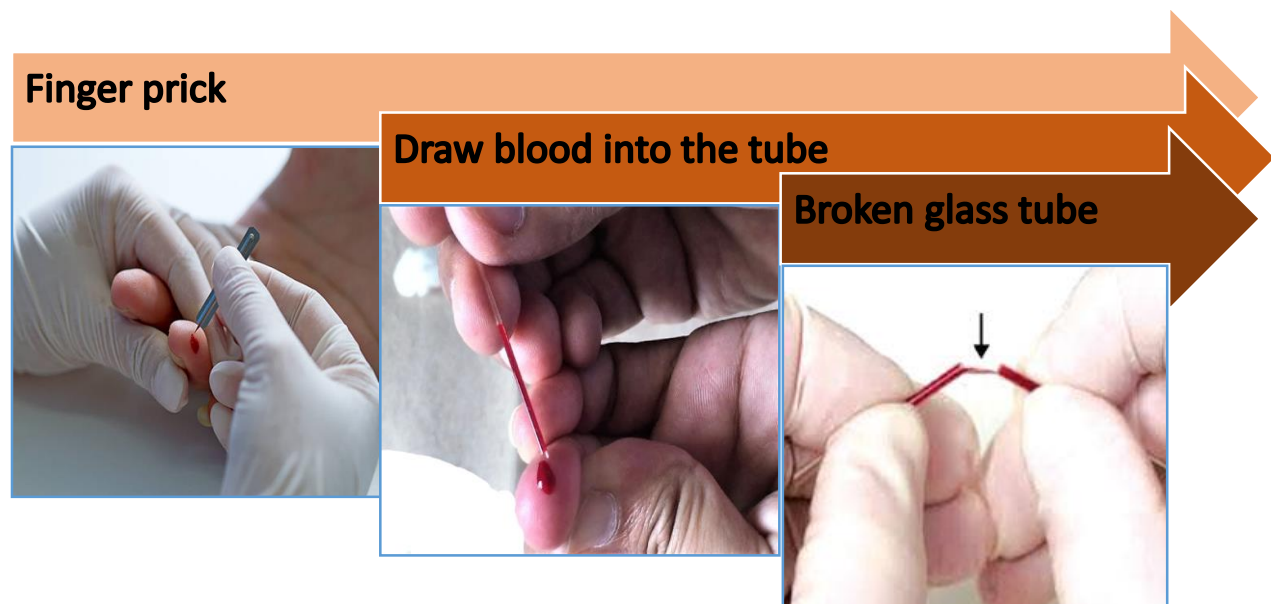
Via using Sodium heparinized (red tip), or Plain, Non-heparinized (blue tip) capillary tubes:

1. Allow a large drop to form. Now dip one end of the capillary tube in the blood; the blood rises into the tube by capillary action. This can be enhanced by keeping its open end.
2. Note the time when blood starts to enter the tube. This is the zero time.
3. Gently break off 1 cm bits of the glass tube from one end, at intervals of 30 seconds, and look for the formation of fibrin threads between the broken ends "rope formation".

* Normal clotting time = 3–6 minutes. The CT is prolonged in hemophilia and other clotting disorders because thrombin cannot normally be generated.

Clotting time depends on:

- I. Nature of contact surface; siliconized surface would prolong the CT.
- II. Presence or absence of clotting factors.
- III. Temperature: Low temperature may prolong the CT.



OBSERVATIONS:

- ❖ Connective tissue diseases: Some of these diseases may be associated with purpuric bleeding. Note in all cases of purpura due to vessel wall defects the platelet counts are normal, but BT is prolonged and the capillary fragility test is positive.
- ❖ Variations in platelet count under physiological conditions are uncommon. Increased counts may be seen after severe exercise, and sometimes at high altitudes. Decreased counts, near the lower side of the normal, may be seen in newborns and females during menstruation.

*** Pathological variations in platelet count:**

Thrombocytopenia: The term refers to a decreased count of platelets. It may be due to:

A. Decreased production

1. Bone marrow injury/depression/failure: Drugs (sulpha, chloramphenicol, cytotoxic drugs); irradiation, acute septic fevers, toxemias, and aplastic anemia.
2. Bone marrow invasion: By leukemia, and secondary deposits of malignant disease.
3. Periodic thrombocytopenic purpura (purpura hemorrhagica): Cause not known.

B. Increased destruction (i.e decreased survival time).

1. Drugs: Thiazides, quinine, ethanol, estrogens, methyldopa, quinidine.
2. Immune thrombocytopenic purpura (ITP): Autoimmune destruction of platelets. May be idiopathic, or associated with some disease, e.g. AIDS.
3. Sequestration in the spleen: There is increased trapping and/or destruction by the enlarged spleen.
4. Disseminated intravascular coagulation (DIC): Platelets are depleted and coagulation factors consumed during widespread clotting, e.g. severe infection (especially meningococcal, pneumococcal), severe and extensive burns, trauma (crush injuries), mismatched transfusion, and retained dead fetus. There may be severe bleeding in some cases.
5. Hemorrhage with extensive transfusion.

Thrombocytosis: The term refers to an increase in platelet count.

A. Primary thrombocytosis (thrombocythemia: count > 800,000/mm³). It is a myeloproliferative disease involving megakaryocytes. Bleeding and thrombosis may occur.

B. Secondary (or reactive) thrombocytosis: count > 500,000/mm³). This condition occurs after the removal of the spleen or after severe hemorrhage.