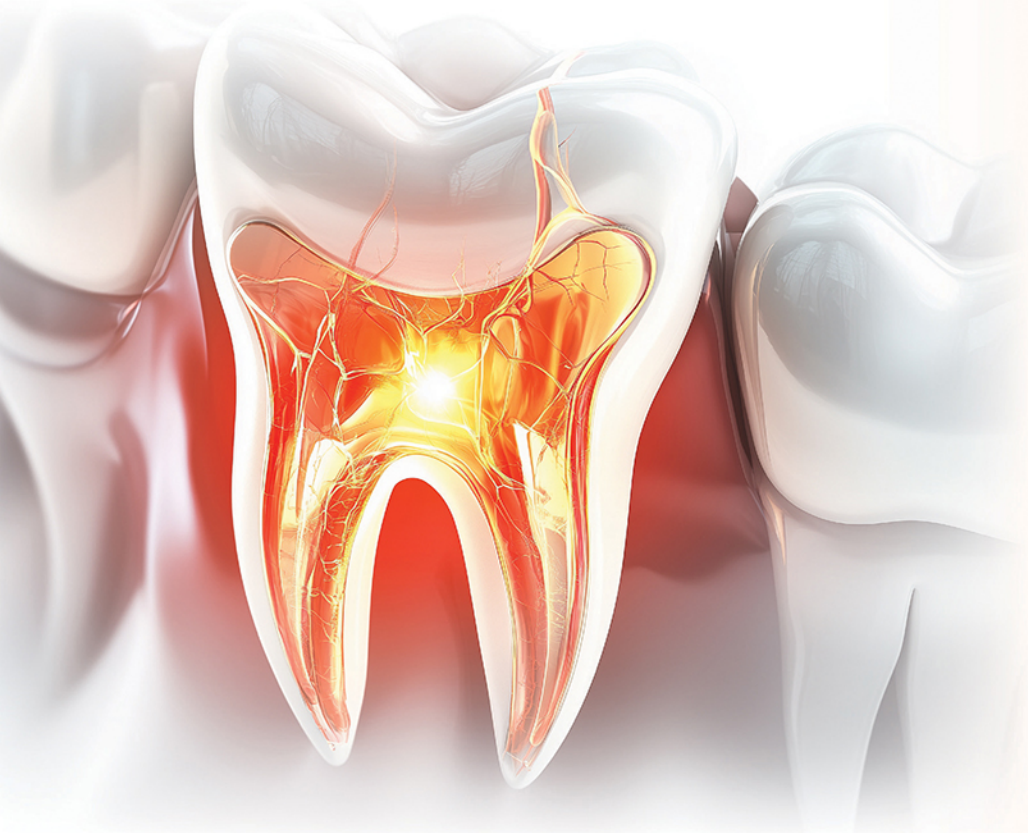
4th Edition

Essentials of PHYSIOLOGY for Dental Students



Dr. Wise
Innovative & Genius
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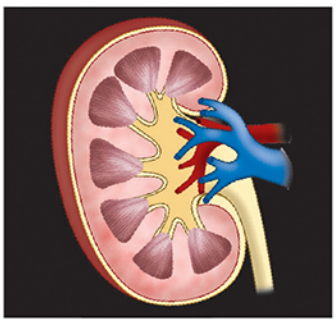
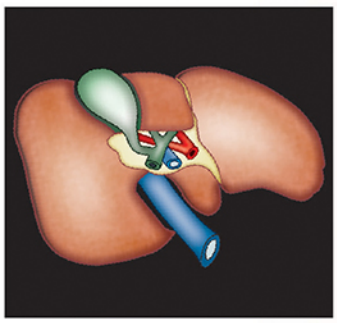
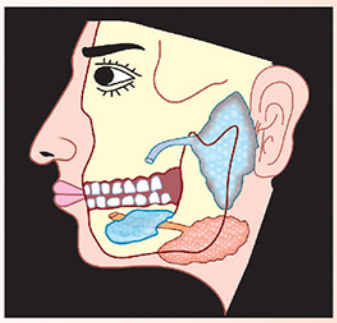
HIGHLIGHTS

- In accordance with DCI curriculum
- Up to date with essential pathophysiological facts
- Simple tables and figures for better student understanding
- Numerous boxes on applied physiology
- Important questions at the end of each section
- Core concept building book







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Prema Sembulingam

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18 CHAPTER

Coagulation of Blood

CHAPTER OUTLINE

DEFINITION AND CLOTTING FACTORS

Blood coagulation or **blood clotting** is defined as a process during which blood loses its fluidity and becomes a jelly-like mass few minutes after it is shed out or collected in a container.

FACTORS INVOLVED IN BLOOD CLOTTING

Coagulation of blood occurs through a series of reactions due to activation of a group of substances. The substances necessary for clotting are called **clotting factors**. Thirteen clotting factors are identified and listed in **Table 18.1**.

Clotting factors were named after the scientists who discovered them or as per their activity except factor IX. Christmas factor (factor IX) was named after the patient in whom it was discovered.

MECHANISM OF BLOOD CLOTTING

ENZYME CASCADE THEORY

Most of the clotting factors are proteins in the form of enzymes. Normally, all factors are present in the form of inactive **proenzymes** which must be activated into enzymes to enforce blood clotting. It is carried out by series of **proenzyme-enzyme conversion reactions**. First one of the series is converted into an active enzyme that activates the second one, which activates the third one; this continues till the final active enzyme thrombin is formed.

Enzyme cascade theory explains how various reactions involved in the conversion of proenzymes to active enzymes take place in the form of a cascade. **Cascade** refers to a process that occurs through a series of steps, each step initiating the next, until the final step is reached.

Stages of Blood Clotting

*Blood clotting occurs in **three stages**:*

I. Formation of prothrombin activator.
II. Conversion of prothrombin into thrombin.
III. Conversion of fibrinogen into fibrin.

TABLE 18.1: Clotting factors.

Factors	Name
I	Fibrinogen
II	Prothrombin
III	Thromboplastin (tissue factor)
IV	Calcium
V	Labile factor (proaccelerin or accelerator globulin)
VI	Presence has not been proved
VII	Stable factor
VIII	Antihemophilic factor (antihemophilic globulin)
IX	Christmas factor
X	Stuart-Prower factor
XI	Plasma thromboplastin antecedent
XII	Hageman factor (contact factor)
XIII	Fibrin-stabilizing factor (fibrinase)

STAGE I : FORMATION OF PROTHROMBIN ACTIVATOR

Blood clotting commences with the formation of a substance called prothrombin activator. Formation of prothrombin is initiated by substances produced either within blood itself or outside the blood.

Formation of prothrombin activator occurs through **two pathways**:

A. Intrinsic pathway.
B. Extrinsic pathway.

Intrinsic Pathway for Formation of Prothrombin Activator

In intrinsic pathway, formation of prothrombin activator is initiated by platelets, which are within the blood itself **(Fig. 18.1)**.

Sequence of intrinsic pathway

1. During injury, blood vessel is ruptured. Endothelium is damaged and **collagen** beneath the endothelium is exposed.
2. When factor XII (**Hageman factor**) comes in contact with collagen, it is converted into activated factor XII in the presence of **kallikrein** and **HMW kininogen** (high molecular weight kininogen).
3. Activated factor XII converts factor XI into activated factor XI in the presence of HMW kininogen.
4. Activated factor XI activates factor IX in the presence of factor IV (calcium).
5. Activated factor IX activates factor X in the presence of factor VIII and calcium.
6. When platelet have contact with collagen of damaged blood vessel, it gets activated and releases phospholipids.
7. Now, the activated factor X reacts with platelet phospholipid and factor V to form prothrombin activator. This needs presence of calcium ions.
8. Factor V is also activated by positive feedback effect of thrombin (see below).

Extrinsic Pathway for Formation of Prothrombin Activator

In this pathway, formation of prothrombin activator is initiated by tissue thromboplastin which is formed from the injured tissues.

Sequence of events in extrinsic pathway

1. Tissues that are damaged during injury release factor III, i.e., **tissue thromboplastin**. Thromboplastin contains proteins, phospholipid and glycoprotein, which act as proteolytic enzymes.
2. Glycoprotein and phospholipid components of thromboplastin convert factor X into activated factor X, in the presence of factor VII.
3. Activated factor X reacts with factor V and phospholipid component of tissue thromboplastin to form prothrombin activator. This reaction requires the presence of calcium ions.

STAGE II: CONVERSION OF PROTHROMBIN INTO THROMBIN

Blood clotting is all about thrombin formation. Once thrombin is formed, it leads to clot formation.

Sequence of Events in Stage II

1. Prothrombin activator that is formed in intrinsic and extrinsic pathways converts prothrombin into thrombin in the presence of calcium ions (factor IV).
2. Once formed thrombin initiates the formation of more thrombin molecules. The initially formed thrombin activates factor V. Factor V in turn accelerates formation of both extrinsic and intrinsic prothrombin activator which converts prothrombin into thrombin. This effect of thrombin is called **positive feedback effect (Fig. 18.1)**.

STAGE III: CONVERSION OF FIBRINOGEN INTO FIBRIN

Final stage of blood clotting involves the conversion of fibrinogen into fibrin by thrombin.

Sequence of Events in Stage III

1. Thrombin converts fibrinogen into **activated fibrinogen** which is called **fibrin monomer**.
2. Fibrin monomer polymerizes with other monomer molecules and form loosely arranged strands of fibrin.
3. Later the **loose fibrin strands** are modified into dense and **tight fibrin threads** by **fibrin-stabilizing factor** (factor XIII) in the presence of calcium ions **(Fig. 18.1)**. All the tight fibrin threads are aggregated to form a meshwork of **stable clot**.

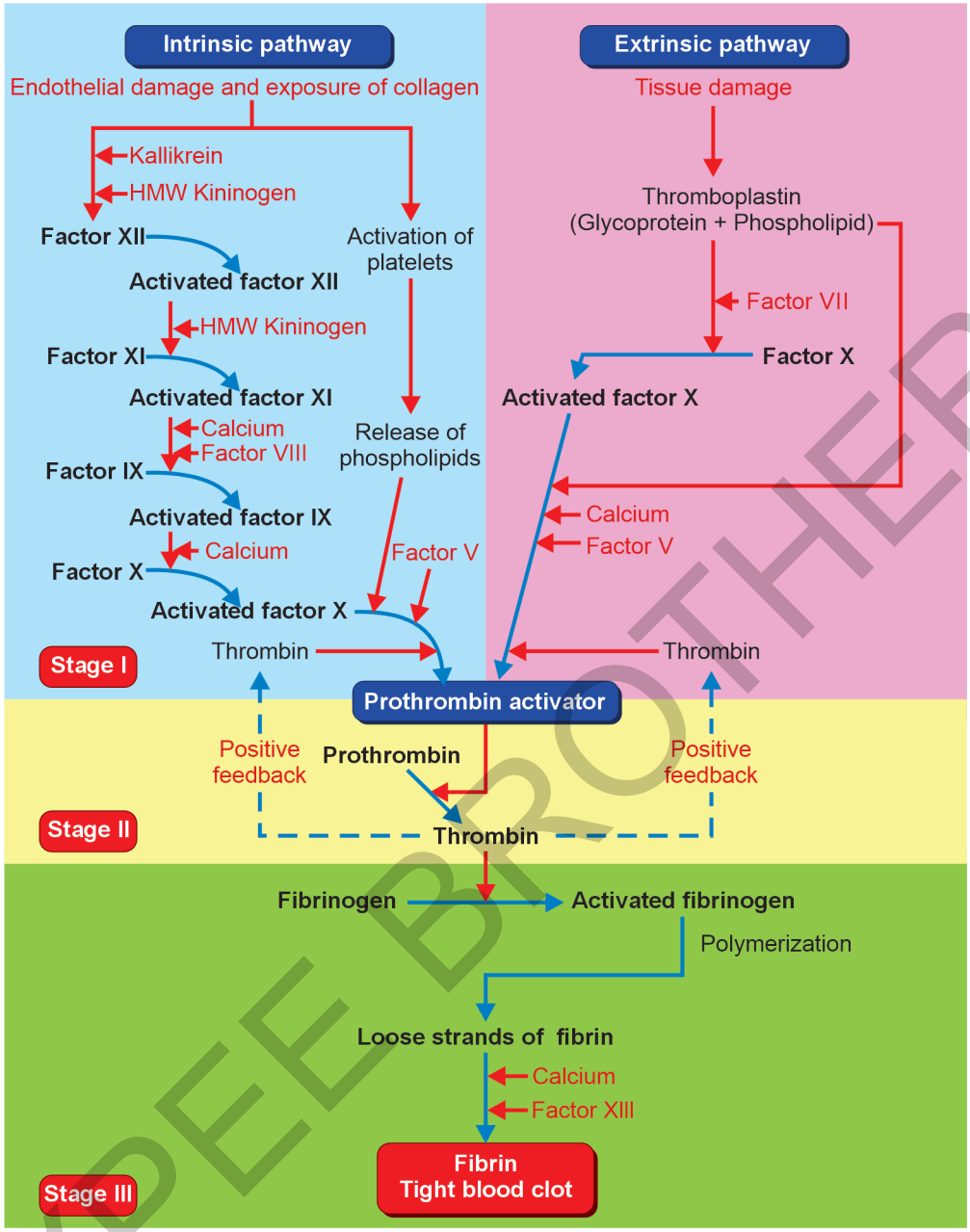

FIGURE 18.1: Stages of blood coagulation.
HMW = High molecular weight.

BLOOD CLOT

DEFINITION AND COMPOSITION OF BLOOD CLOT

Blood clot is defined as the mass of coagulated blood which contains RBCs, WBCs and platelets entrapped in fibrin meshwork **(Fig. 18.2)**.

RBCs and WBCs are not necessary for clotting process. However, when clot is formed, these cells are trapped in it along with platelets. Entrapped RBCs are responsible for red color of clot.

External blood clot called **scab** adheres to the opening of damaged blood vessel and prevents blood loss.

CLOT RETRACTION

Clot retraction is a process which involves contraction of blood clot 30 to 45 minutes after formation and oozing of **serum** out of clot. Contractile proteins namely, actin, myosin and **thrombosthenin** in cytoplasm of platelets are responsible for clot retraction.

FIBRINOLYSIS

Fibrinolysis is a process that results in breakdown and dissolution of blood clot inside the blood vessel. It helps to remove the clot from lumen of blood vessel. Fibrinolysis requires a substance called **plasmin** or **fibrinolysin**.

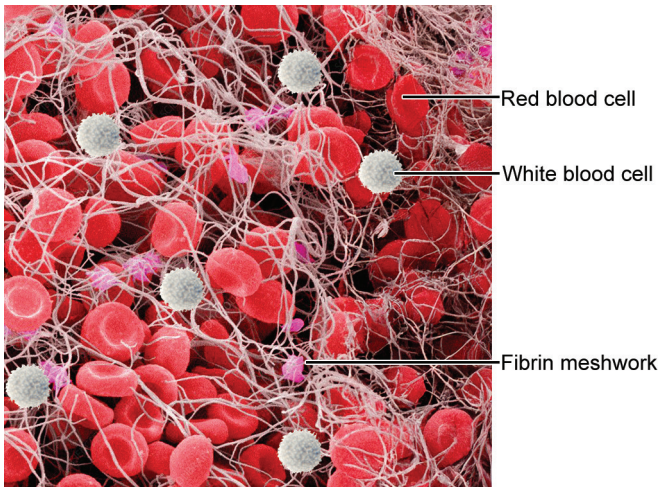

FIGURE 18.2: Blood clot.

Formation of Plasmin

Plasmin is formed from inactivated glycoprotein called **plasminogen**. Plasminogen is synthesized in liver and is incorporated with other proteins in the blood clot. Plasminogen is converted into plasmin by tissue **plasminogen activator (t-PA),** lysosomal enzymes and thrombin. The t-PA and lysosomal enzymes are released from damaged tissues and damaged endothelium. Thrombin is derived from blood. The t-PA is always inhibited by a substance called **t-PA inhibitor.** It is also inhibited by factors V and VIII.

Besides t-PA, there is another plasminogen activator called **urokinase plasminogen activator (u-PA).** It is derived from blood.

Sequence of Events Involved in Activation of Plasminogen

1. During intravascular clotting, endothelium of the blood vessel secretes a **thrombin-binding protein** called **thrombomodulin.** It is secreted by endothelium of all the blood vessels, except minute vessels of brain.
2. Thrombomodulin combines with thrombin and forms a **thrombomodulin-thrombin complex**.
3. Thrombomodulin-thrombin complex activates **protein C**.
4. Activated protein C inactivates factor V and VIII in the presence of a cofactor called **protein S.**
5. Protein C also inactivates the t-PA inhibitor.
6. Now, t-PA becomes active.
7. Activated t-PA and lysosomal enzymes activate plasminogen to form plasmin. Plasminogen is also activated by thrombin and u-PA **(Fig. 18.3)**.

Significance of Lysis of Clot

In vital organs, particularly heart, blood clot obstructs the minute blood vessel leading to **myocardial infarction**. Lysis of blood clot allows reopening of affected blood vessels and prevents the development of infarction.

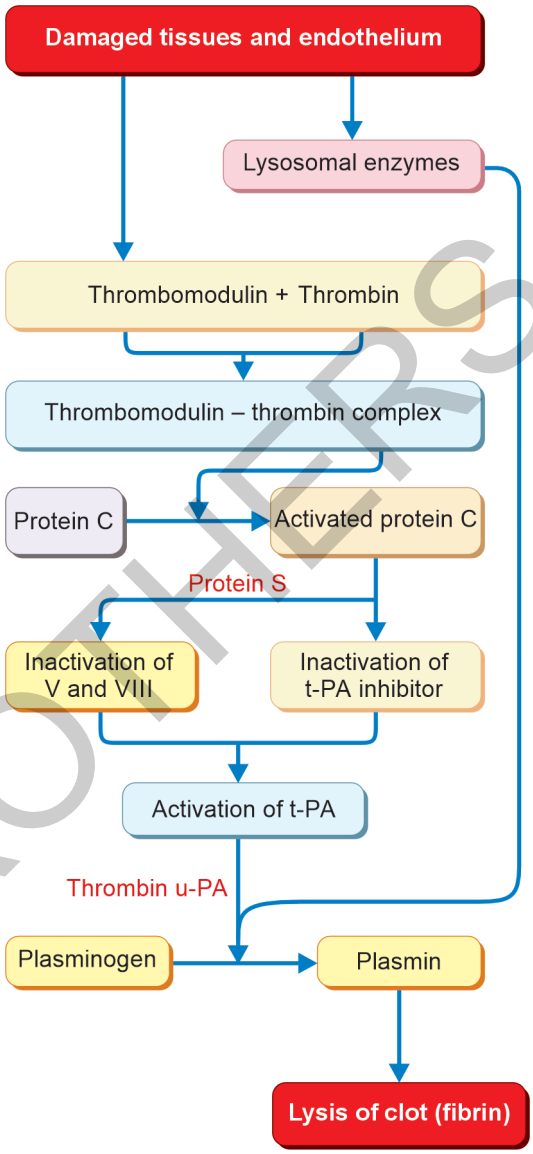

FIGURE 18.3: Fibrinolysis.
t-PA = Tissue plasminogen activator, u-PA = Urokinase plasminogen activator.

Fibrinolytic enzymes such as **streptokinase** are used for lysis of blood clot, during the treatment in early stages of myocardial infarction.

■ ANTICLOTTING MECHANISM IN BODY

In physiological conditions, intravascular clotting does not occur. It is because of presence of some physico-chemical factors in the body.

Factors Preventing Blood Clotting during Circulation

1. **Smooth surface** of endothelium of blood vessels prevents activation of clotting factors.
2. **Glycocalyx layer** on inner surface of endothelium repels platelets and clotting factors and thereby initiation of blood clotting is prevented.
3. **Continuous flow of blood** does not allow aggregation of platelets and prevents blood clotting.

4. Presence of **natural anticoagulants** in blood:
   i. Heparin: It is the natural anticoagulant that is produced by the liver (see below for its mechanism of action).
   ii. Protein C: Activated by **thrombomodulin-thrombin complex.** Activated protein C along with its cofactor protein S inactivates Factor V and Factor VIII. Inactivation of these two clotting factors prevents clot formation.

ANTICOAGULANTS

Anticoagulants are the substances, which prevent or postpone coagulation of blood.

*Anticoagulants are of **three types**:*

A. Anticoagulants used to prevent blood clotting inside the body: **In vivo anticoagulants**.
B. Anticoagulants used to prevent clotting of blood that is collected from the body: **In vitro anticoagulants**.
C. Anticoagulants used to prevent blood clotting both in vivo and in vitro.

Commonly used anticoagulants are explained below.

I. HEPARIN

Heparin is a naturally produced anticoagulant in the body. It is produced by **mast cells**, the wandering cells situated immediately outside the capillaries in many tissues or organs that contain more connective tissue. Mast cells are abundant in liver and lungs. **Basophils** also secrete heparin.

Heparin is a conjugated polysaccharide. Commercial heparin is prepared from liver and other organs of animals. Commercial preparation is available in liquid form or dry form as sodium, calcium, ammonium or lithium salts.

Mechanism of Action of Heparin

Heparin:

1. Prevents blood clotting by its **anti-thrombin activity**. It directly suppresses the activity of thrombin.
2. Combines with antithrombin III present in circulation and removes thrombin from circulation.
3. Activates antithrombin III.
4. Inactivates the active form of other clotting factors like IX, X, XI and XII **(Fig. 18.4)**.

Uses of Heparin

Heparin is used as an anticoagulant both in vivo and in vitro.

Clinical Use

Intravenous injection of heparin (0.5 to 1 mg/kg body weight) postpones clotting for 3 to 4 hours (until it is destroyed by the enzyme heparinase). So, it is widely used as an anticoagulant in clinical practice.

Heparin is used for the following purposes:

1. To prevent intravascular blood clotting during **surgery**.

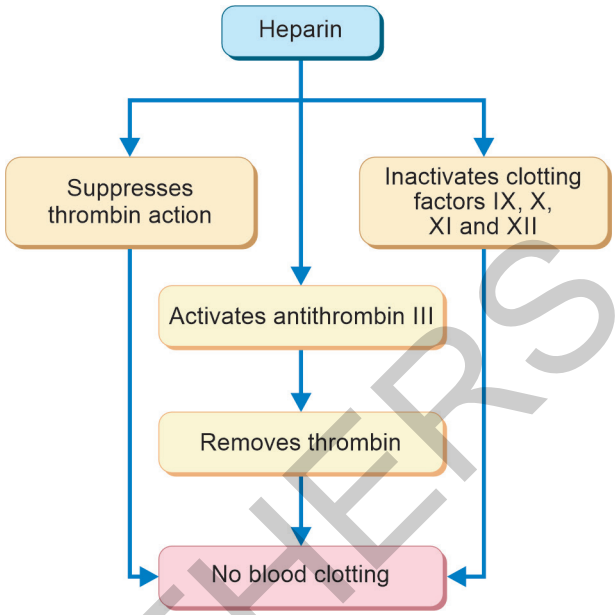

FIGURE 18.4: Mechanisms of action of heparin.

2. During dialysis when blood is passed through **artificial kidney**.
3. During cardiac surgery, that involves passing the blood through **heart lung machine**.
4. To preserve the blood before transfusion.

Use in Laboratory

Heparin is also used as anticoagulant in vitro while collecting blood for various investigations. Heparin is the most expensive anticoagulant.

II. COUMARIN DERIVATIVES

Dicoumarol and warfarin are the derivatives of coumarin.

Mechanism of Action

Coumarin derivatives prevent blood clotting by inhibiting the action of vitamin K. Vitamin K is essential for the formation of various clotting factors namely, II, VII, IX and X.

Uses

Dicoumarol and warfarin are the commonly used **oral anticoagulants** (in vivo).

III. EDTA

Ethylenediaminetetraacetic acid (EDTA) is a strong anticoagulant. It is available in two forms namely disodium salt (Na_2 EDTA) and tripotassium salt (K_3 EDTA).

Mechanism of Action of EDTA

EDTA prevents blood clotting by removing calcium from blood.

Uses of EDTA

EDTA is used as an anticoagulant both in vivo and in vitro:

1. It is administered intravenously in cases of **lead poisoning** (in vivo).

2. It is also used as an anticoagulant in the laboratory (in vitro).

IV. OXALATE COMPOUNDS

Oxalate compounds prevent coagulation by forming calcium oxalate, which is precipitated later. Thus, oxalate compounds reduce the blood calcium level.

A mixture of ammonium oxalate and potassium oxalate in the ratio of 3:2 is used. Each salt is an anticoagulant by itself. But potassium oxalate alone causes shrinkage of RBCs. Ammonium oxalate alone causes swelling of RBCs. But together, both the substances do not alter cellular activity.

Mechanism of Action of Oxalate Compounds

Oxalate combines with calcium and forms insoluble calcium oxalate. Thus, oxalate removes calcium from blood and lack of calcium prevents coagulation.

Uses of Oxalate Compounds

Oxalate compounds are used as in vitro anticoagulants. Oxalate is poisonous so it cannot be used in vivo.

V. CITRATES

Sodium, ammonium and potassium citrates are used as anticoagulants.

Mechanism of Action of Citrates

Citrate combines with calcium in blood to form insoluble calcium citrate. Like oxalate, citrate also removes calcium from blood and prevents coagulation.

Uses of Citrates

Citrate is used as an anticoagulant both in vivo and in vitro:

1. Used to store blood in **blood bank**. It is available in two forms:
   a. **Acid citrate dextrose** (ACD).
   b. **Citrate phosphate dextrose** (CPD).
2. Used in laboratory in vitro for RBC and platelet counts.

VI. OTHER SUBSTANCES, WHICH PREVENT BLOOD CLOTTING

Peptone, proteins from venom of copper-head snake and hirudin (from leech) are the known anticoagulants.

PHYSICAL METHODS TO PREVENT BLOOD CLOTTING

Coagulation of blood is postponed or prevented by the following physical methods:

1. COLD

Reducing the temperature to about 5°C postpones coagulation of blood.

2. COLLECTING BLOOD IN A CONTAINER WITH SMOOTH SURFACE

Collecting the blood in a container with smooth surface like a **silicon-coated container** prevents clotting. Smooth surface inhibits the activation of factor XII and platelets. So, the formation of prothrombin activator is prevented.

PROCOAGULANTS

Procoagulants or **hemostatic agents** are the substances, which accelerate process of blood coagulation.

Procoagulants are:

1. ***Thrombin***: Thrombin is sprayed upon the bleeding surface to arrest bleeding by hastening blood clotting.
2. ***Snake venom***: Venom of some snakes (vipers, cobras and rattle snakes) contains proteolytic enzymes which enhance blood clotting by activating the clotting factors.
3. ***Extracts of lungs and thymus***: Extract obtained from lungs and thymus has thromboplastin, which causes rapid blood coagulation.
4. ***Sodium or calcium alginate***: Sodium or calcium alginate enhances blood clotting process by activating Hageman factor.
5. ***Oxidized cellulose***: This causes clotting of blood by activating Hageman factor.

TESTS FOR BLOOD CLOTTING

Blood clotting tests are used to diagnose blood disorders. Some tests are also used to monitor the patients treated with anticoagulant drugs such as heparin and warfarin.

***Six tests** are available for blood clotting*:

1. BLEEDING TIME

Bleeding time is the time interval from oozing of blood after a cut or injury till arrest of bleeding. Usually, it is determined by **Duke method** using blotting paper or filter paper method.

Normal bleeding time: 3 to 6 minutes.

It is prolonged in **purpura**.

2. CLOTTING TIME

Clotting time is the time interval from oozing of blood after a cut or injury till the formation of clot. It is usually determined by capillary tube method.

Normal clotting time: 3 to 8 minutes.

And it is prolonged in **hemophilia**.

3. PROTHROMBIN TIME

It is the time taken by blood to clot after adding tissue thromboplastin to it. Blood is collected and oxalated so that, calcium is precipitated and prothrombin is not converted into thrombin. Thus, the blood clotting is prevented.

Then a large quantity of tissue thromboplastin with calcium is added to this blood. Calcium nullifies the effect of oxalate. The tissue thromboplastin activates pro-thrombin and blood clotting occurs.

During this procedure, the time taken by blood to clot after adding tissue thromboplastin is determined.

Prothrombin time indicates the total quantity of prothrombin present in blood.

Normal prothrombin time: 10 to 12 seconds.

It is prolonged in deficiency of prothrombin and other factors such as factors I, V, VII and X. However, it is normal in hemophilia.

4. PARTIAL PROTHROMBIN TIME

Partial prothrombin time (PPT) or activated prothrombin time is the time taken for blood to clot after adding an activator such as phospholipid, along with calcium to it. It is also called activated partial prothrombin time (APPT). This test is useful in monitoring the patients taking anticoagulant drugs.

It is carried out by observing clotting time after adding phospholipid, a **surface activator** and calcium to a patient's plasma. Phospholipid serves as **platelet substitute.** Commonly used surface activator is **kaolin.**

Normal partial prothrombin time: 30 to 45 seconds.

It is prolonged in **heparin or warfarin therapy** (since heparin and warfarin inhibit clotting) and deficiency or inhibition of factors II, V, VIII, IX, X, XI and XII.

5. INTERNATIONAL NORMALIZED RATIO

International normalized ratio (INR) is the rating of a patient's PT when compared to an average. It measures extrinsic clotting pathway system.

INR is useful in monitoring impact of anticoagulant drugs such as warfarin and to adjust the dosage of anticoagulants. Patients with atrial fibrillation are usually treated with warfarin to protect against blood clot, which may cause strokes. These patients should have regular blood tests to know their INR in order to adjust warfarin dosage.

Blood takes longer time to clot if INR is higher. Normal INR is about 1. In patients taking anticoagulant therapy for atrial fibrillation,

Normal INR: Should be between 2 and 3.

For patients with heart valve disorders, INR should be between 3 and 4. But, INR greater than 4 indicates that blood is clotting too slowly and there is a risk of uncontrolled blood clotting.

6. THROMBIN TIME

Thrombin time (TT) is the time taken for the blood to clot after adding thrombin to it. It is done to investigate the presence of heparin in plasma or to detect fibrinogen abnormalities. This test involves observation of clotting time after adding thrombin to patient's blood.

Normal thrombin time: 12 to 20 seconds.

It is prolonged in heparin therapy and during dysfibrinogenemia (abnormal function of fibrinogen with normal fibrinogen level).

APPLIED PHYSIOLOGY

I. THROMBOSIS

Thrombosis or **intravascular blood clotting** refers to coagulation of blood inside the blood vessels. Normally, blood does not clot in the blood vessel because of some factors which are already explained. But some abnormal conditions can cause thrombosis.

Causes of Thrombosis

1. **Injury to blood vessels**: Damage of endothelial lining of blood vessels during infection or mechanical obstruction initiates thrombosis.
2. **Roughened endothelial lining**: During infection, damage or arteriosclerosis, endothelium becomes rough and initiates clotting.
3. **Sluggishness of blood flow**: Decreased rate of blood flow causes aggregation of platelets and formation of thrombus. Slowness of blood flow occurs during reduced cardiac action, hypotension, low metabolic rate, prolonged confinement to bed and immobility of limbs.
4. **Agglutination of RBCs**: Agglutination of RBCs occurs by foreign antigens or toxic substances. It leads to thrombosis.
5. **Toxic thrombosis**: This occurs due to the action of chemical poisons like **arsenic compounds**, mercury, poisonous mushrooms and snake venom.
6. **Congenital absence of protein C**: This causes thrombosis and death in infancy.

Complications of Thrombosis

1. *Thrombus*

During thrombosis, lumen of blood vessels is occluded. The solid mass of platelets, red cells and/or clot, which obstructs the blood vessel, is called thrombus. Thrombus formed due to agglutination of RBC is called **agglutinative thrombus**.

2. *Embolism and embolus*

Embolism is a process during which thrombus or part of it is detached and carried in bloodstream and occludes the small blood vessels resulting in arrests of blood flow to any organ or region of the body.

Embolus is a thrombus or part of it, which arrests the blood flow. Obstruction of blood flow by embolism is common in lungs (**pulmonary embolism**), brain (**cerebral embolism**) and heart (**coronary embolism**).

3. *Ischemia*

Insufficient blood supply to an organ or area of body by the obstruction of blood vessels is called ischemia. Ischemia results in tissue damage because of **hypoxia** (lack of oxygen). Ischemia also causes discomfort, pain and tissue death. Death of body tissue is called necrosis.

4. *Necrosis and infarction*

Necrosis is a general term that refers to tissue death caused by loss of blood supply, injury, infection, inflammation, physical agents or chemical substances.

Infarction means the tissue death due to loss of blood supply. Loss blood supply is usually caused by occlusion of an artery by thrombus or embolus and sometimes by atherosclerosis (Chapter 51). The area of tissue that undergoes infarction is called infarct. Infarction commonly occurs in heart, brain, lungs, kidneys and spleen.

II. DISSEMINATED INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation (DIC) is a rare condition characterized by abnormal blood clotting in all the blood vessels throughout the body. It is caused by infection, inflammation or cancer.

DIC is a serious condition and often results in development of **multiple organ dysfunction syndrome (MODS).**

III. BLEEDING DISORDERS

Bleeding disorders are the diseases characterized by prolonged bleeding time or clotting time.

*Bleeding disorders are of **three types***:

1. *Hemophilia*

Hemophilia is a group of sex-linked inherited blood disorders characterized by prolonged clotting time. However, bleeding time is normal in hemophilia. In this disorder, males are affected and females are the carriers.

Because of prolonged clotting time, even a mild trauma causes excess bleeding which can lead to death. Damage of skin while falling or extraction of a tooth may cause excess bleeding for few weeks. Easy bruising and hemorrhage in muscles and joints are also common in this disease.

Cause for hemophilia

Lack of prothrombin activator is cause for hemophilia. Formation of prothrombin activator is affected due to the deficiency of factor VIII, IX or XI.

Types of hemophilia

Depending upon deficiency of the factor involved, hemophilia is classified into **three types**:

i. Hemophilia A or **classic hemophilia** that is due to the deficiency of factor VIII. 85% of people with hemophilia are affected by hemophilia A.
ii. Hemophilia B or **Christmas disease** which is due to the deficiency of factor IX. 15% of people with hemophilia are affected by hemophilia B.
iii. Hemophilia C which is due to the deficiency of factor XI. It is a very rare blood disorder.

2. *Purpura*

It is a disorder characterized by prolonged bleeding time. However, clotting time is normal. Characteristic feature of this disease is spontaneous bleeding under the skin from ruptured capillaries. It causes small tiny hemorrhagic spots under the skin which are called **purpuric spots** (purple colored patch-like appearance). That is why this disease is called purpura.

Types and causes of purpura

i. **Thrombocytopenic purpura** which is due to deficiency of platelets (thrombocytopenia). In bone marrow disease, platelet production is affected leading to deficiency of platelets.
ii. **Idiopathic thrombocytopenic purpura** that is due to some unknown cause. It is believed that platelet count decreases due to the development of antibodies against platelets, which occurs after blood transfusion.
iii. **Thrombasthenic purpura** which is due to structural or functional abnormality of platelets. However, the platelet count is normal. It is characterized by normal clotting time, normal or prolonged bleeding time, but defective clot retraction.

3. *von Willebrand Disease*

von Willebrand disease is a bleeding disorder characterized by excess bleeding even with a mild injury. It is due to inherited deficiency of **von Willebrand factor** which is a protein secreted by endothelium of damaged blood vessels and platelets. This protein is responsible for adherence of platelets to endothelium of blood vessels during hemostasis after an injury. It is also responsible for the survival and maintenance of factor VIII in plasma.

Deficiency of von Willebrand factor suppresses platelet adhesion. It also causes deficiency of factor VIII. This results in excess bleeding which resembles the bleeding that occurs during platelet dysfunction or hemophilia.

Essentials of PHYSIOLOGY for Dental Students

Salient Features

- Fulfils the requirements of the dental students in understanding the complex concepts of physiology
- Fully updated with pathophysiological facts in accordance with modern curriculum of dental science
- Tables and figures formatted in new attractive style will help students to understand the contents easily
- Enriched with many boxes containing important facts on applied physiology
- Model Questions given at the end of each section will benefit the students to assess themselves and approach the subject with clear idea
- Formatted in a unique style that enables the readers to understand core concepts of physiology

K Sembulingam PhD is a renowned person in the field of Physiology. He has a long and vast experience of teaching Physiology to undergraduate and postgraduate medical students in various institutions in India and abroad. He had served MR Medical College, Kalaburagi, Karnataka, India; Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India; School of Health Sciences, Universiti Sains Malaysia, Kelantan, Malaysia; Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry, India; Sri Manakula Vinayagar Medical College and Hospital, Puducherry, India; Shri Sathya Sai Medical College and Research Institute, Nellikuppam, Tamil Nadu, India; and Madha Medical College and Research Institute, Chennai, Tamil Nadu, India. Currently, he is working as Visiting Professor in Advanced Physiology at Datta Meghe Medical College, Nagpur, Maharashtra, India and Datta Meghe Institute of Higher Education and Research (Deemed to be University), Wardha, Maharashtra, India.

In addition to this textbook, he has published *Essentials of Medical Physiology*, *Essentials of Physiology for Dental Students* and *Review in Medical Physiology*, which is like a pocketbook for quick revision before the examination. His interest and knowledge in research are commendable. He has published many papers in indexed scientific journals. He was the guide and supervisor for many PhD and postgraduate students in India and Malaysia. His fields of interest in research are Neuroscience, Stress physiology, Antistressor effects of indigenous plants, Fertility control, and Evaluation of autonomic functional status.

Prema Sembulingam PhD started her career as Lecturer in MR Medical College, Kalaburagi, Karnataka, India and step-by-step gained a long and vast experience of teaching Physiology to undergraduate and postgraduate medical students by serving in various institutions in India and abroad. She worked as Professor of Physiology in Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India; School of Health Sciences, Universiti Sains Malaysia, Kelantan, Malaysia; Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry, India; Sathyabama University Dental College and Hospital, Chennai, Tamil Nadu, India; Sri Manakula Vinayagar Medical College and Hospital, Puducherry, India; Shri Sathya Sai Medical College and Research Institute, Nellikuppam, Tamil Nadu, India; and Madha Medical College and Research Institute, Chennai, Tamil Nadu, India.

She has co-authored all the books with Dr K Sembulingam. Her research experience is also commendable with many publications to her credit in indexed scientific journals. Her major field of interest in research is evaluation of autonomic functional status. Her other fields of interest are Neuroscience, Stress physiology and Antistressor effects of indigenous plants.

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