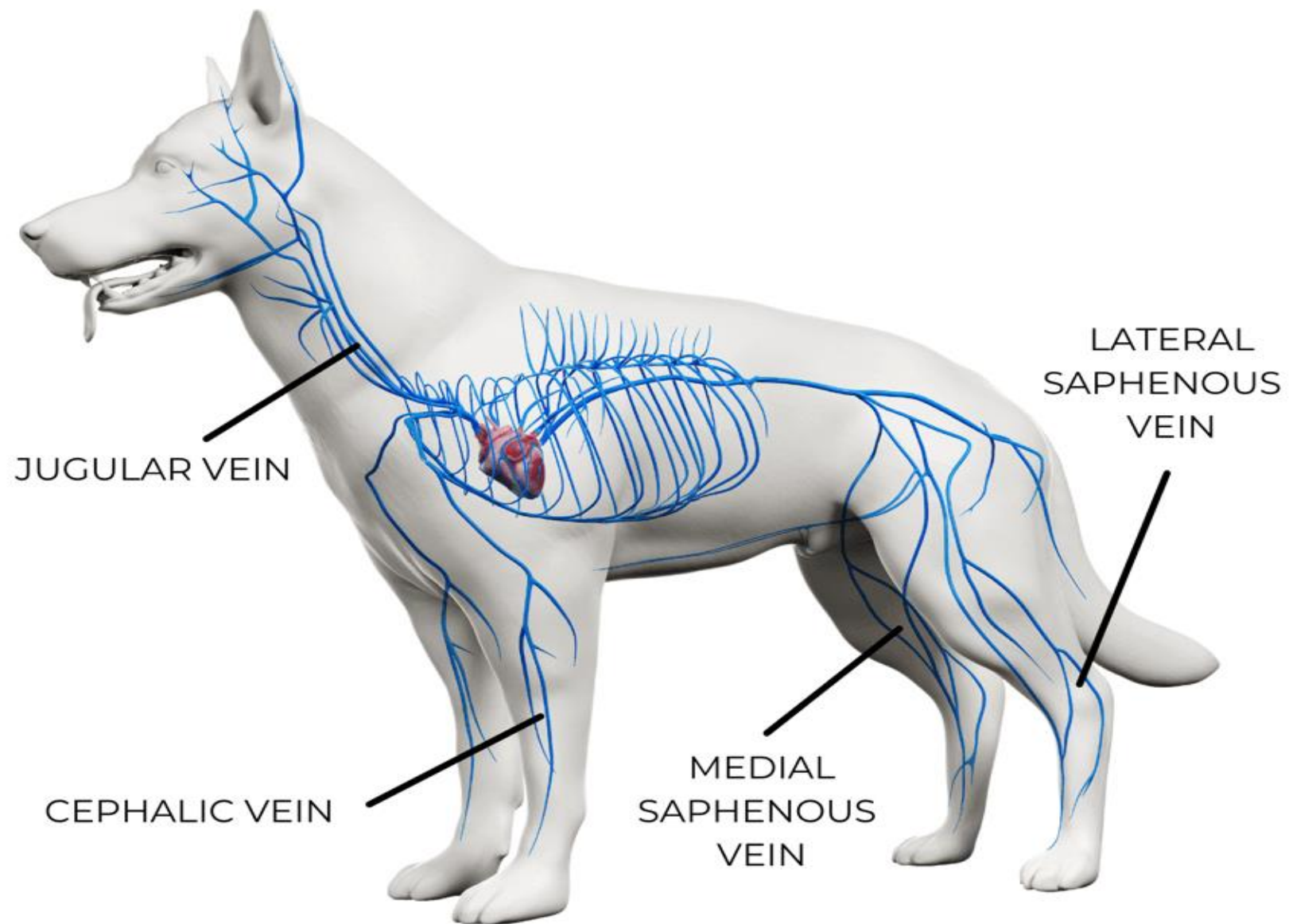
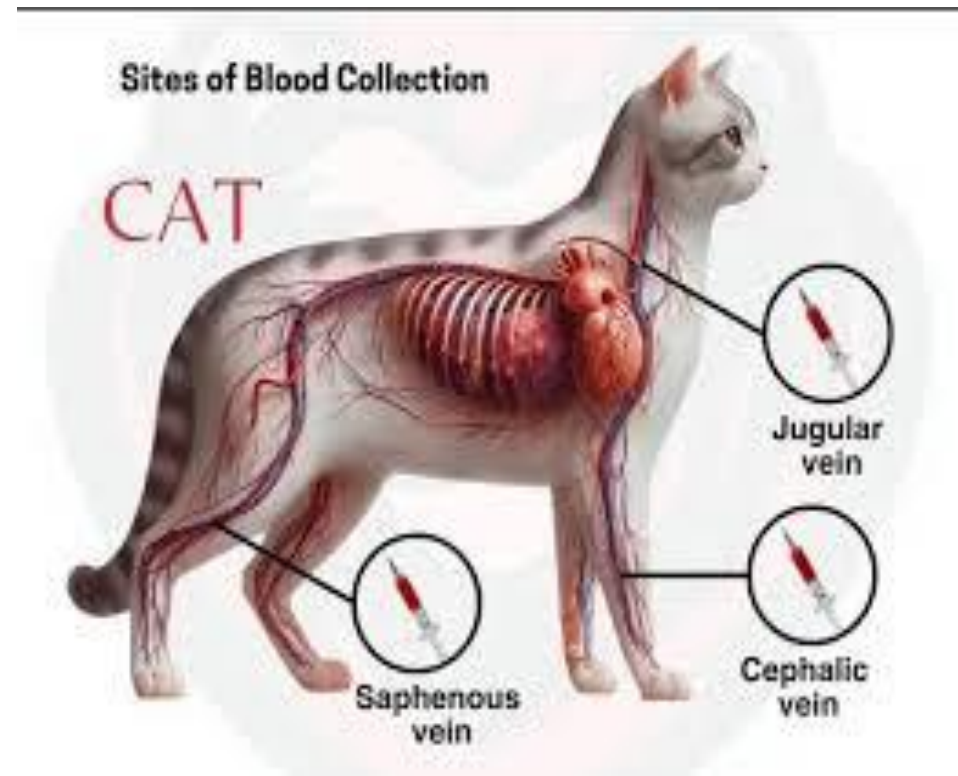


Complete Blood Count(CBC)
Full Blood Count (FBC)
Full Haemogram(FHG)

The **CBC** indicates, the counts of white Blood Cells(**WBC**), Red Blood Cells(**RBC**), Platelets(**Plt**), the concentration of Hemoglobin(**Hgb**), and the Hematocrit (**Hct**), the RBC indices, which indicate the average size and Hemoglobin content of **RBC** and a differential WBC, which counts the different types of **WBC**.



Sites of Blood Collection in Animals

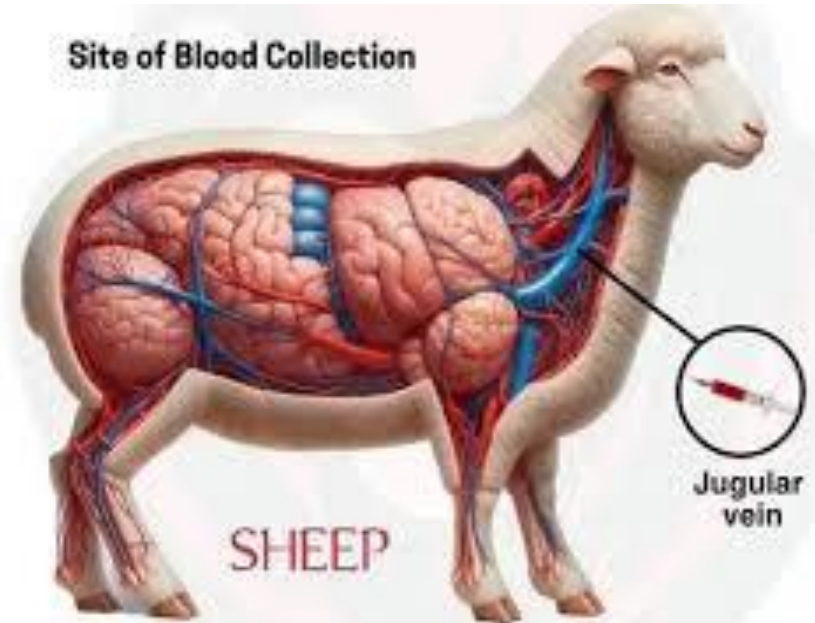


Sites of Blood Collection in Animals

Site of Blood Collection



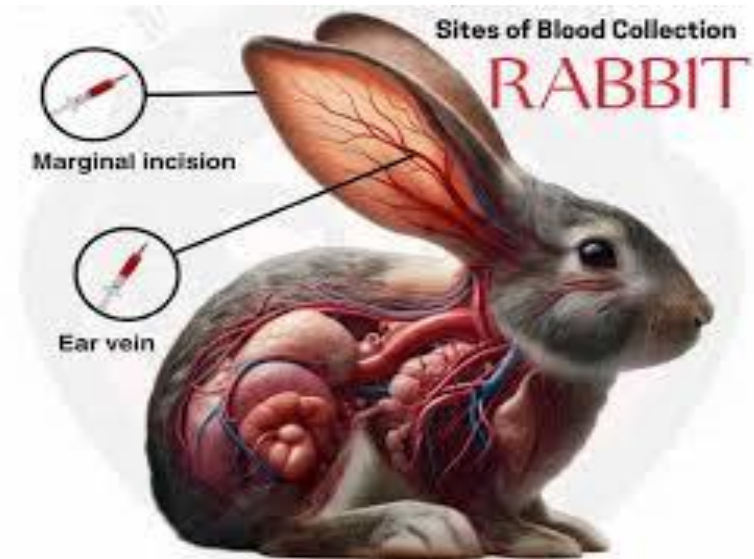
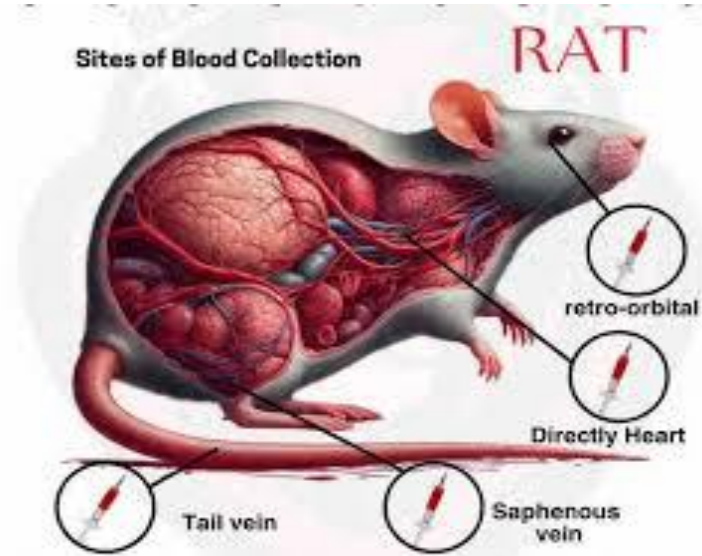
Site of Blood Collection



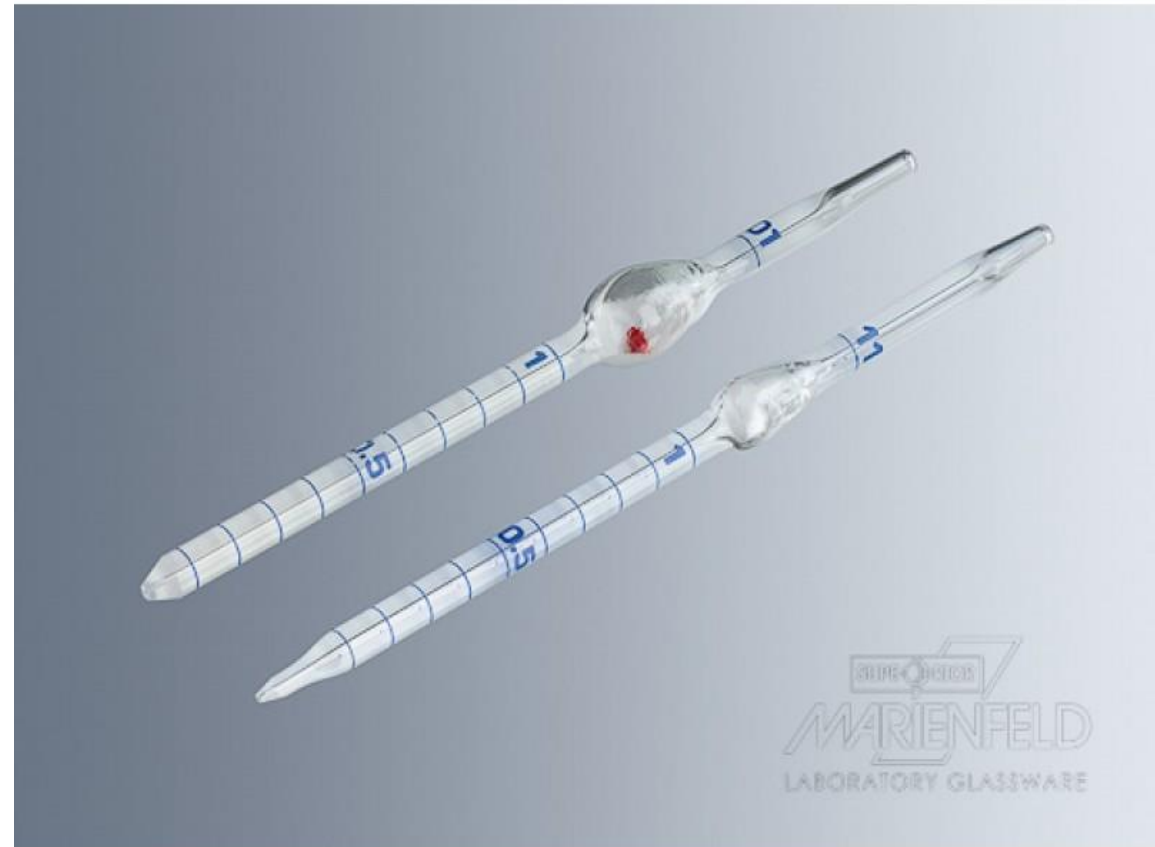
Sites of Blood Collection in Animals



Sites of Blood Collection in Animals



Blood diluting pipettes



Blood diluting pipettes

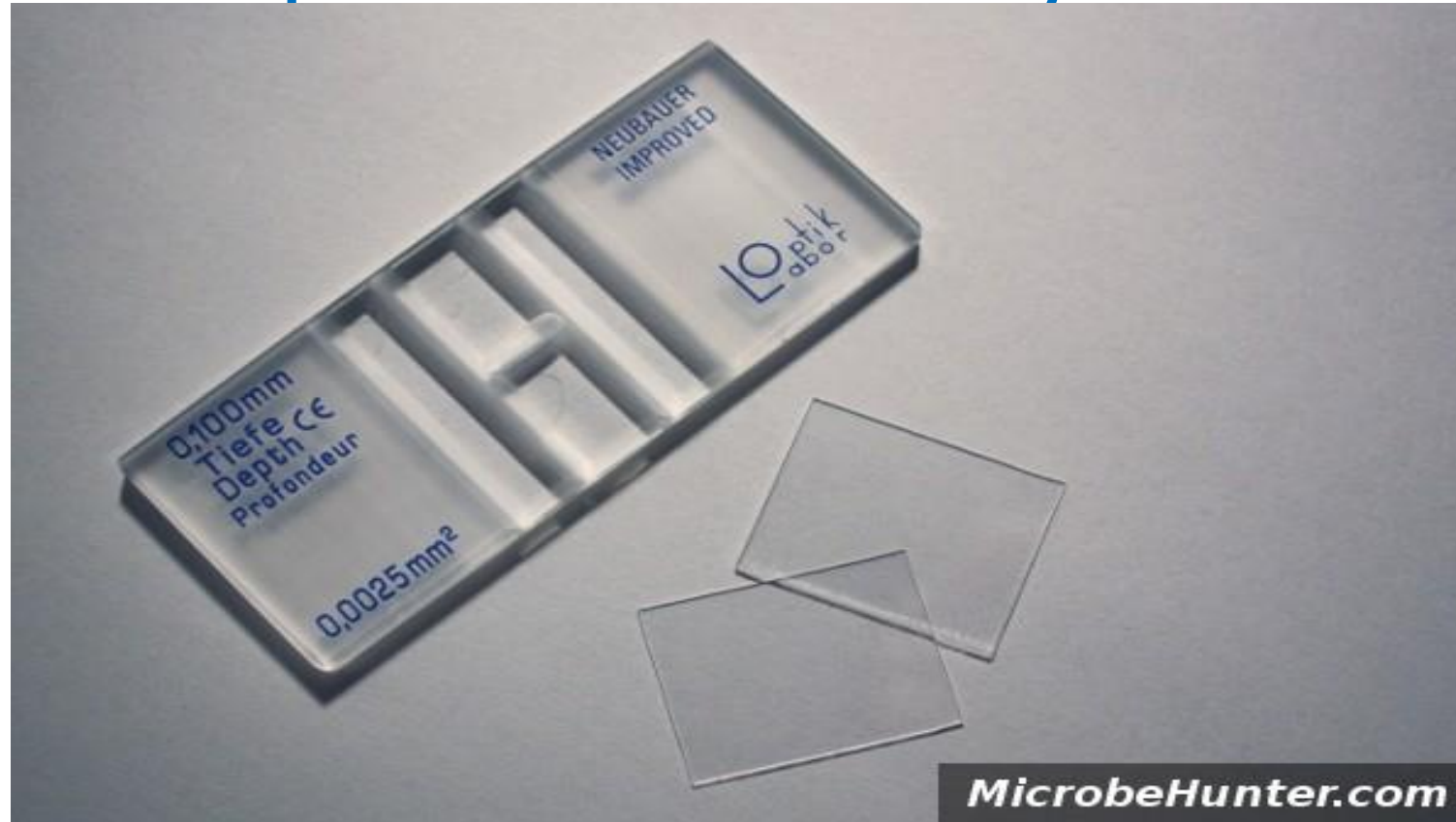


Blood diluting fluids

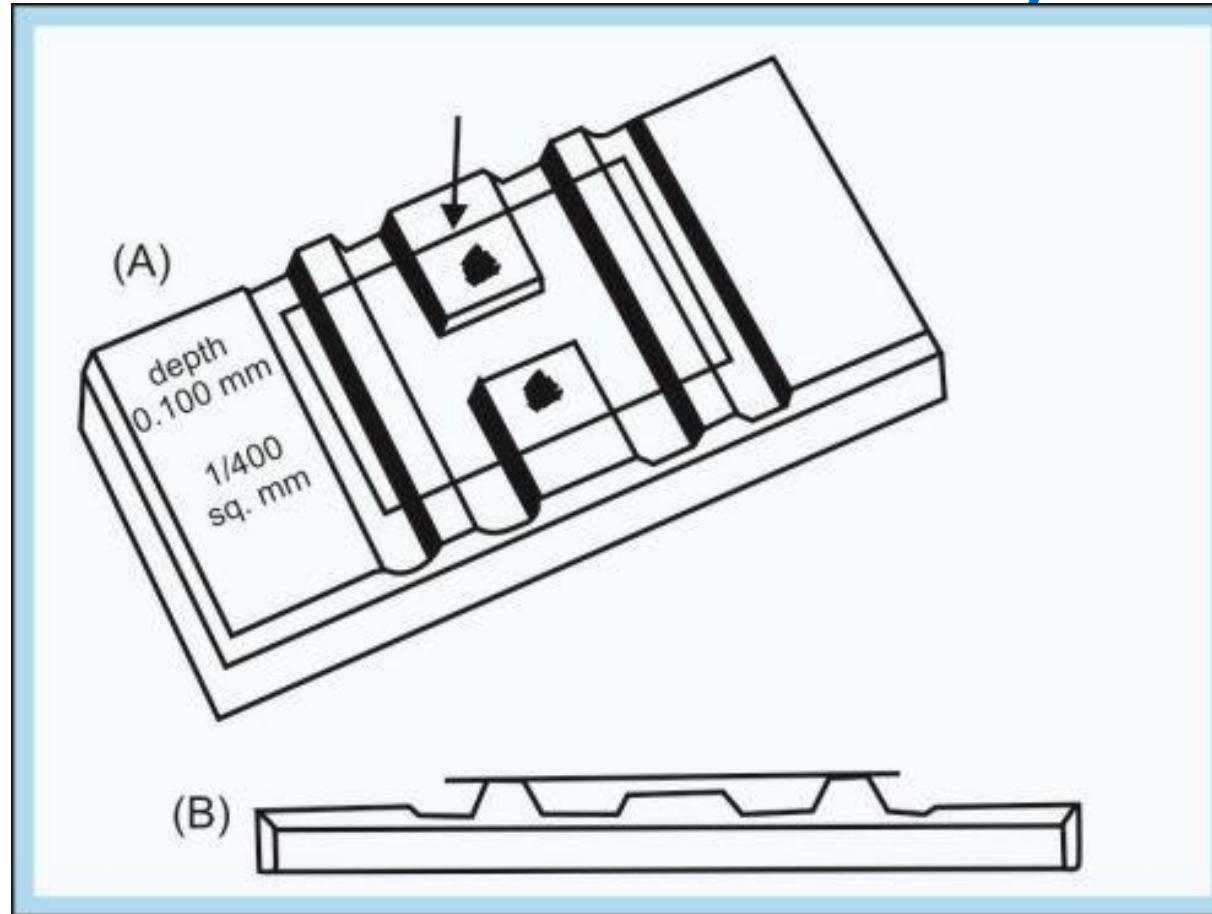


Hemocytometer

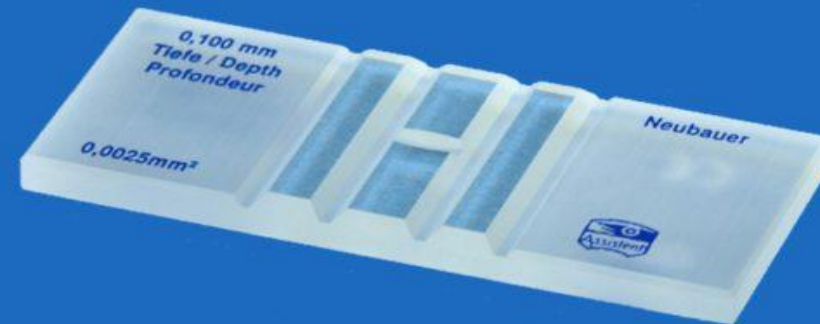
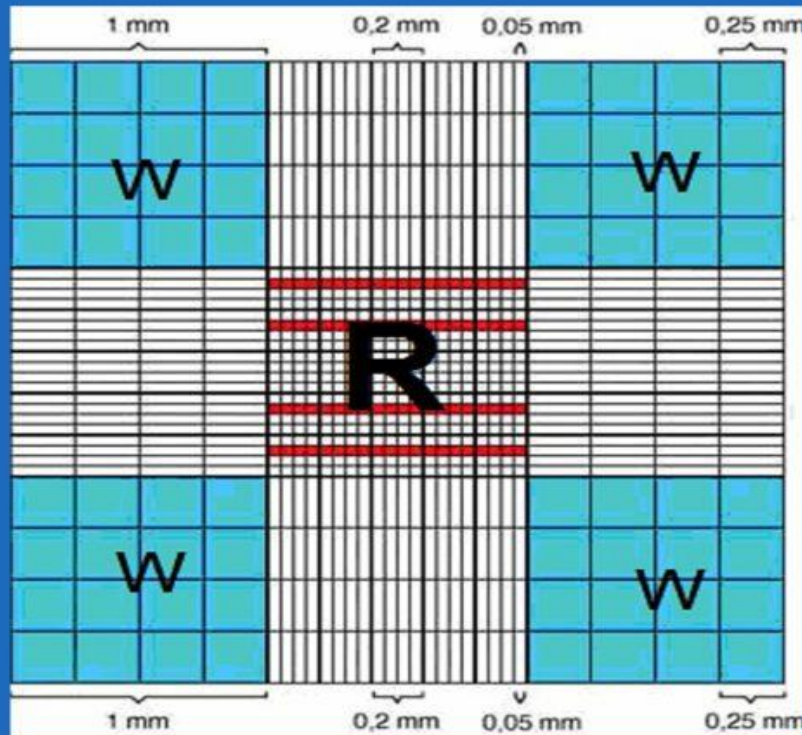
Improved Neubauer Hemocytometer



Improved Neubauer Hemocytometer

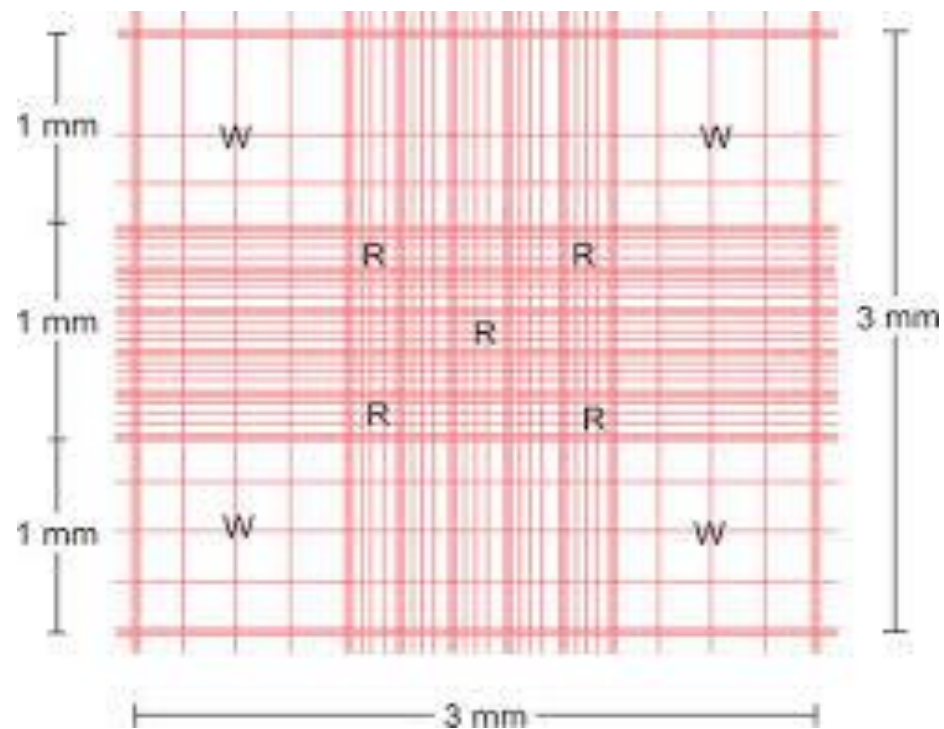
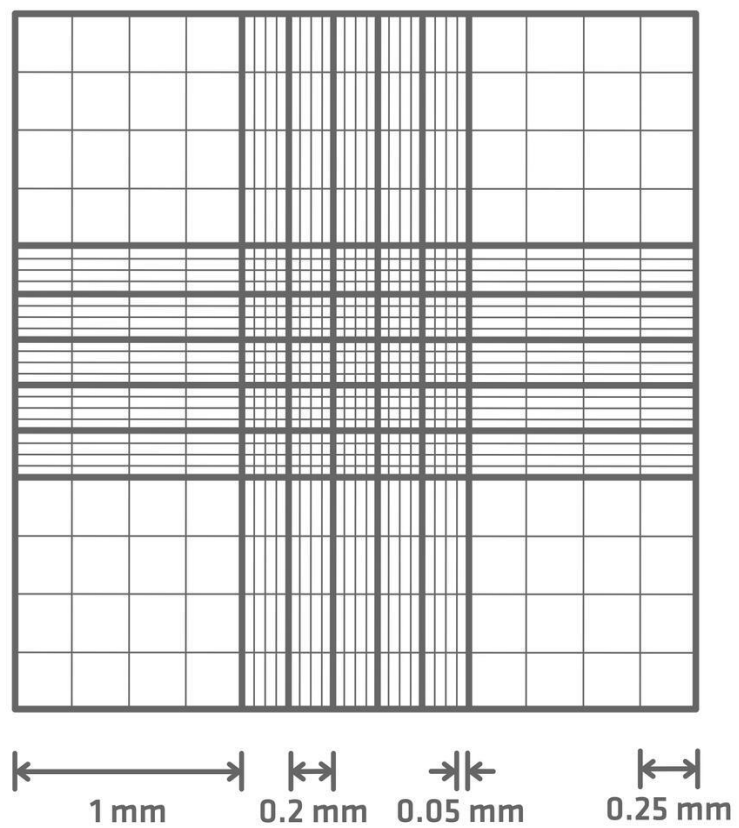


Haemocytometer - Improved Neubauer's chamber

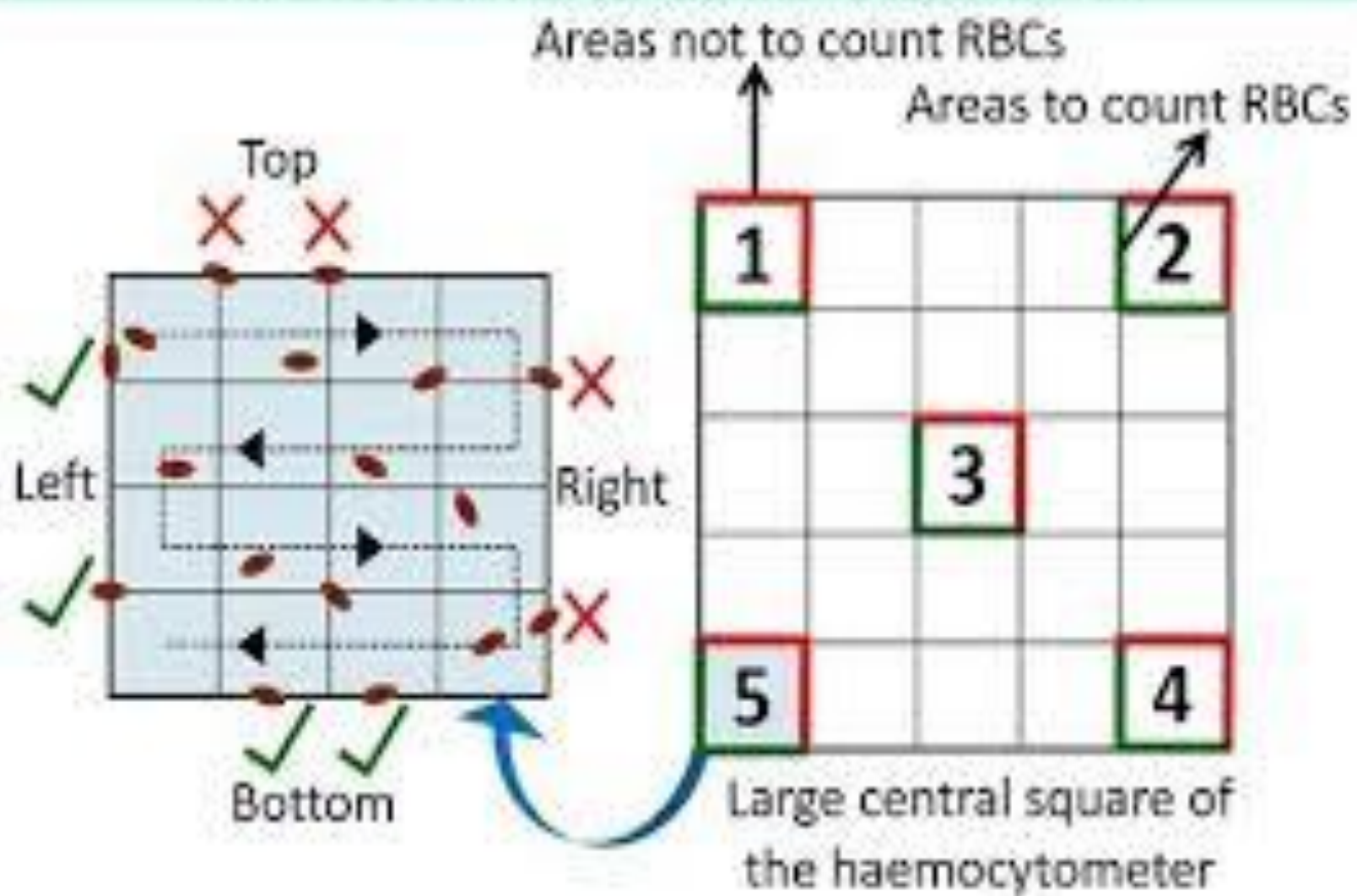


<http://mbbsstudystuff.com/>

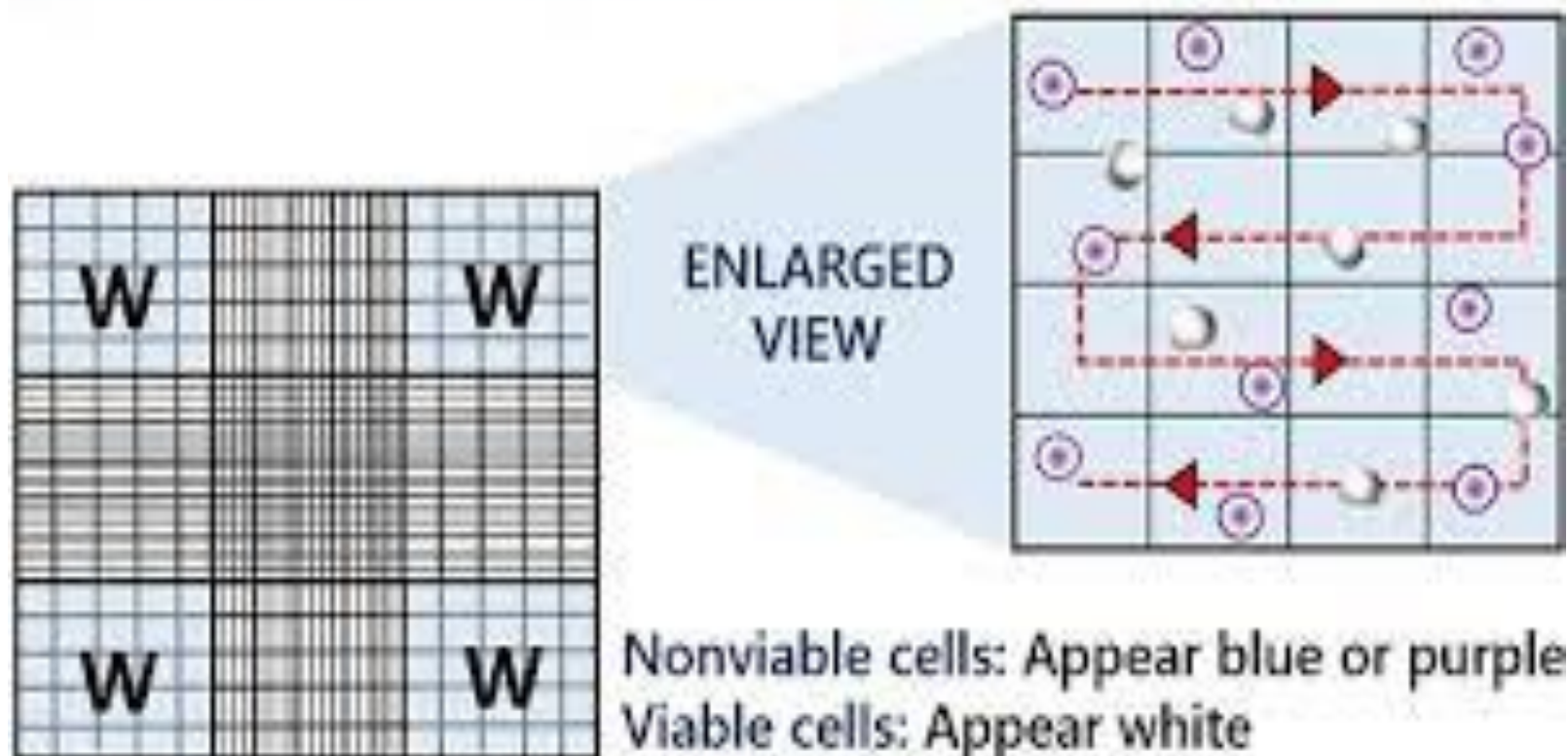
Improved Neubauer chamber counting grid



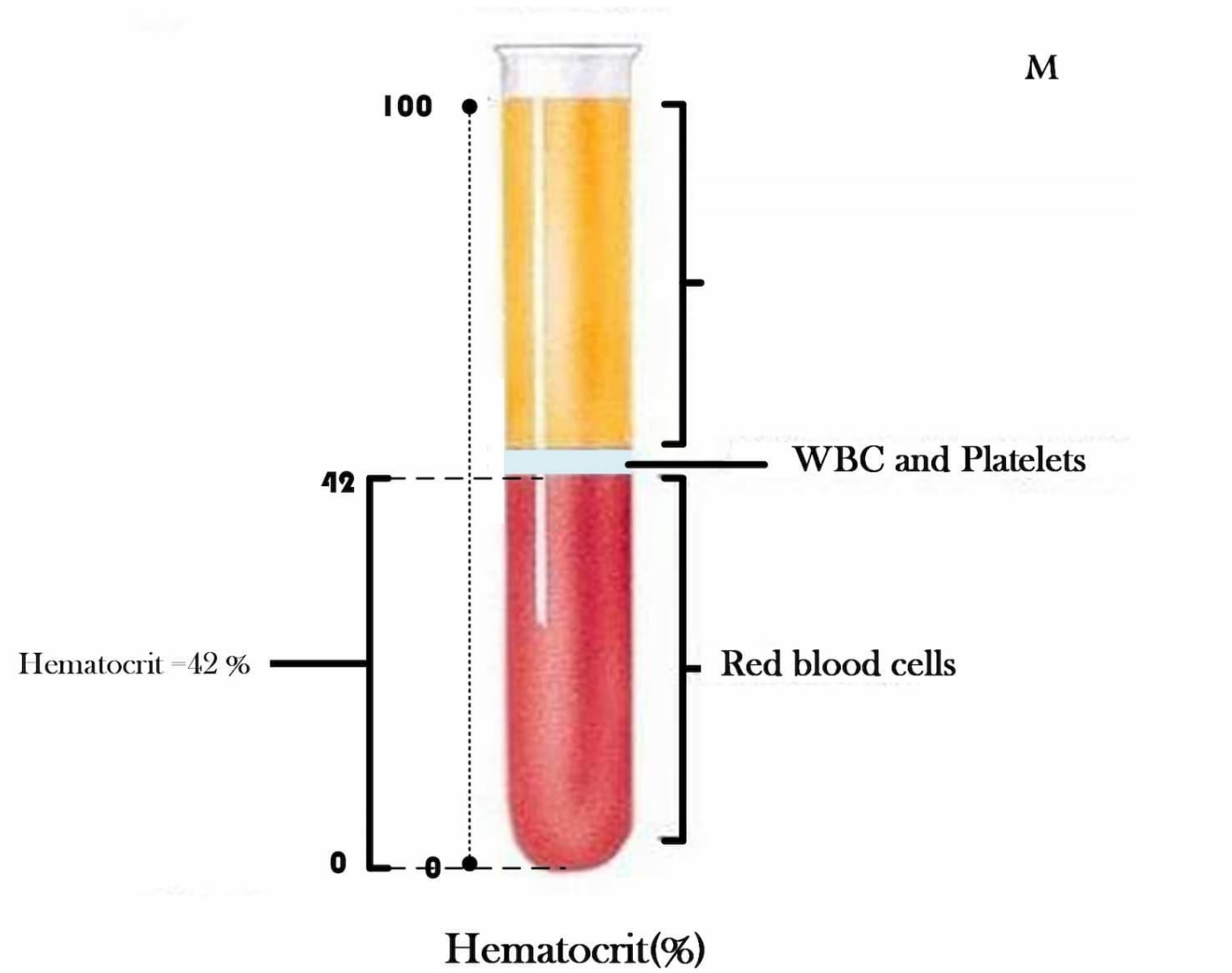
RBC Count Under Microscope



WBC Count Under Microscope







Plasma

- water
- proteins
- glucose
- hormones
- ...

Buffy coat

- leukocytes
- and platelets

Hematocrit

- red blood cells



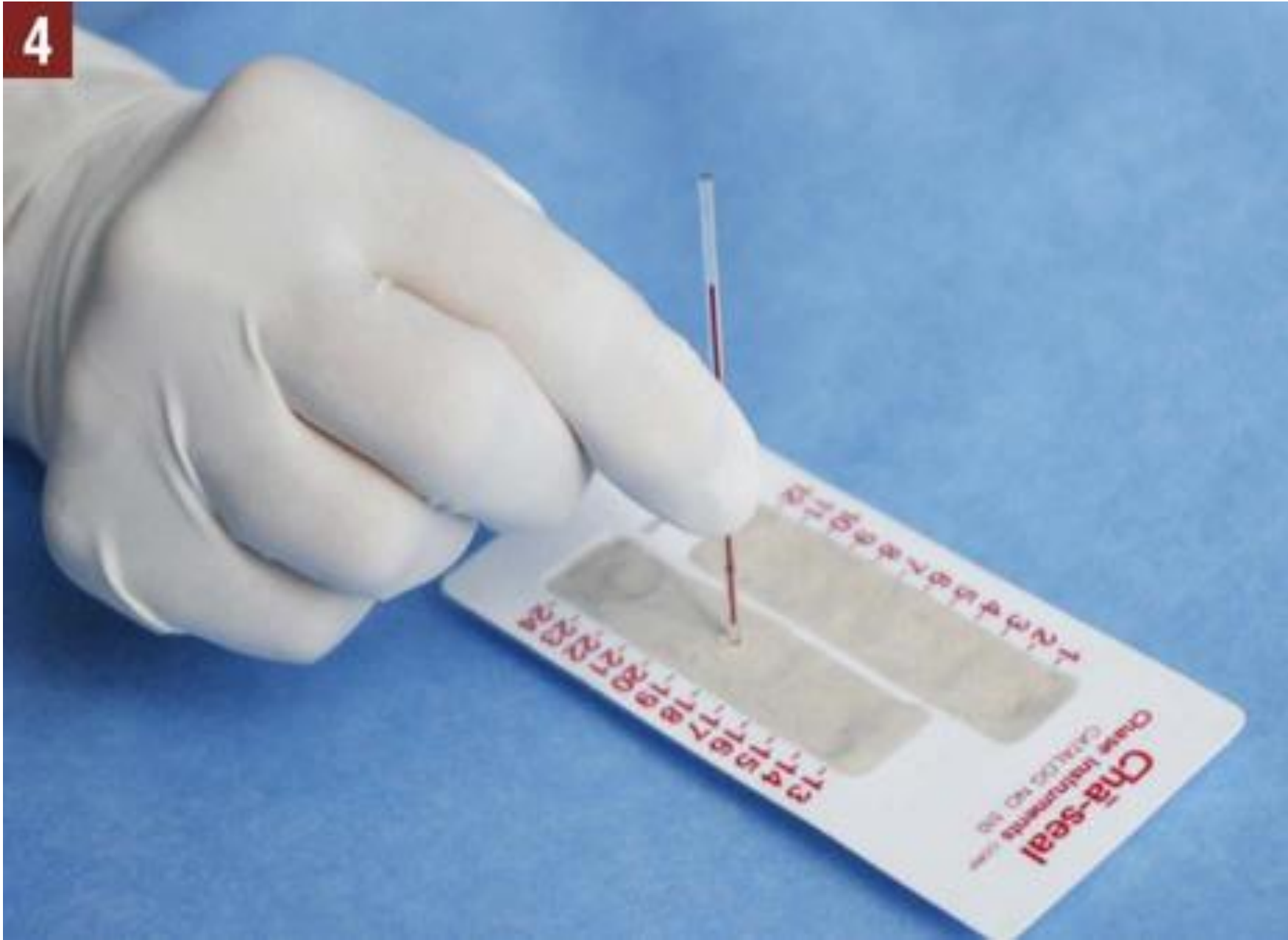
Normal Blood

Anemia

Polycythemia











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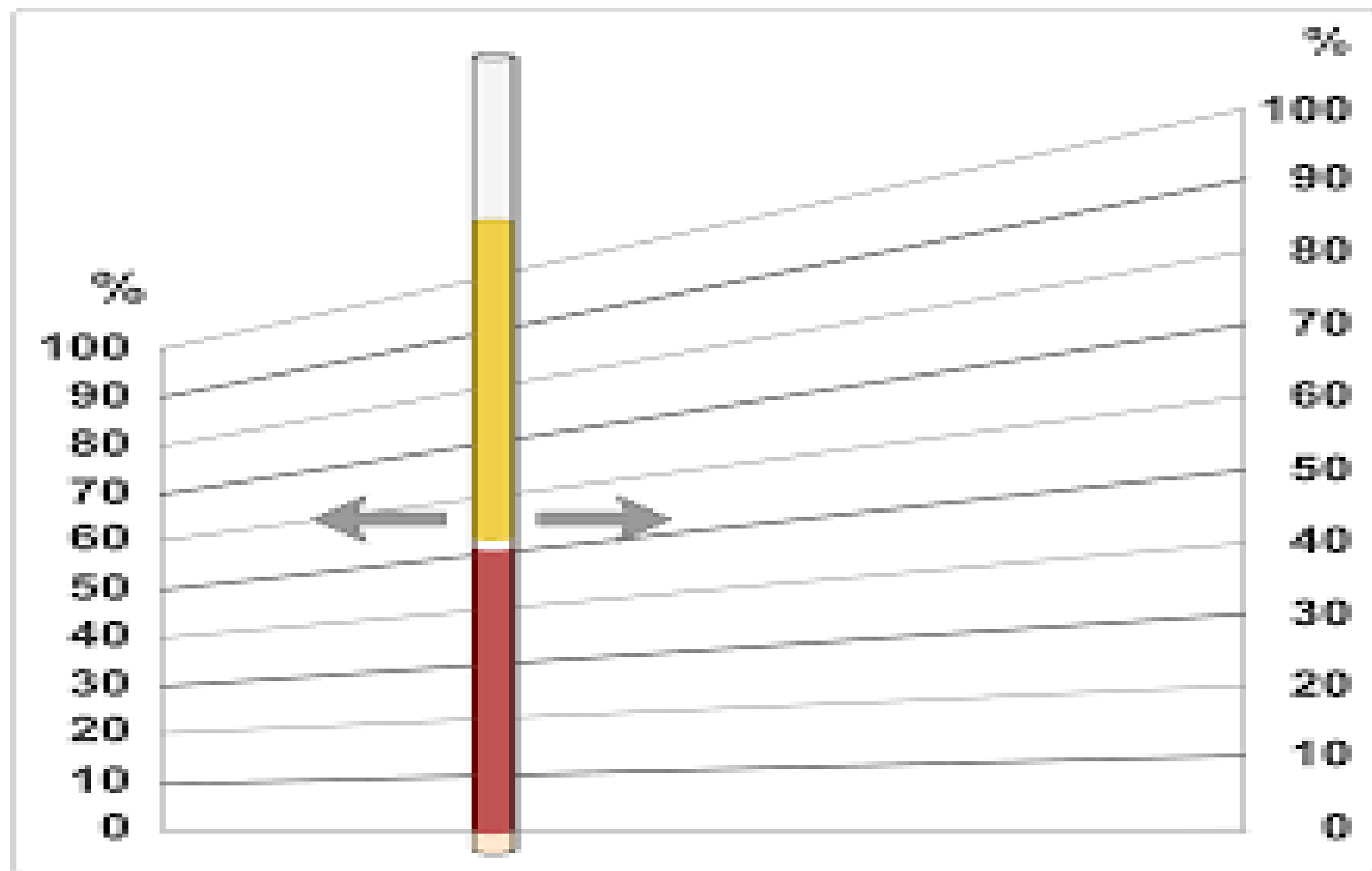
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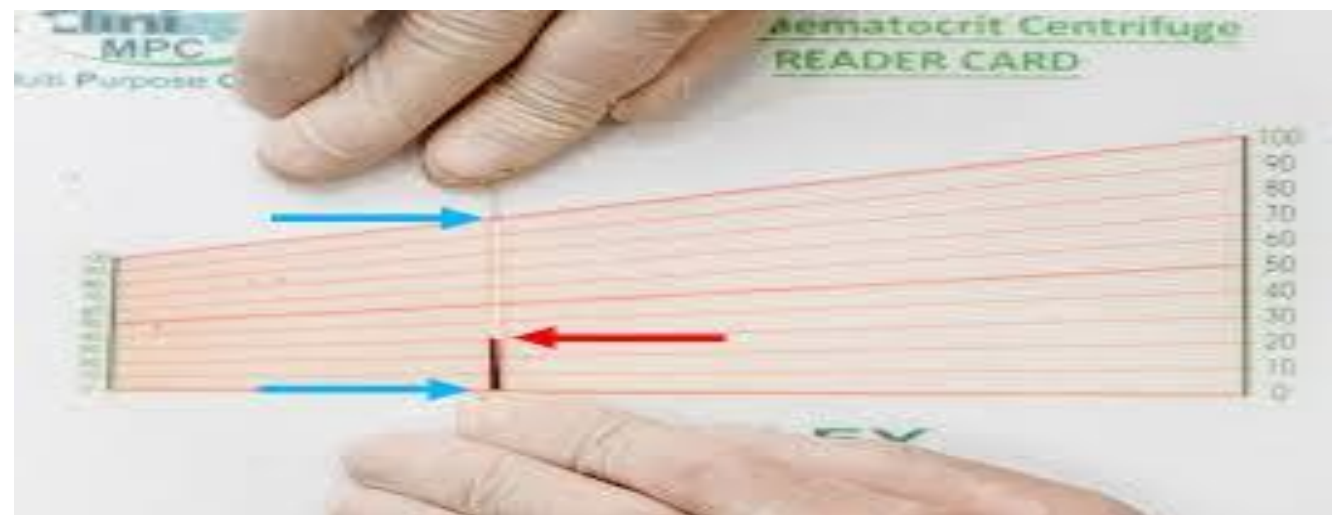


TNM SHOP

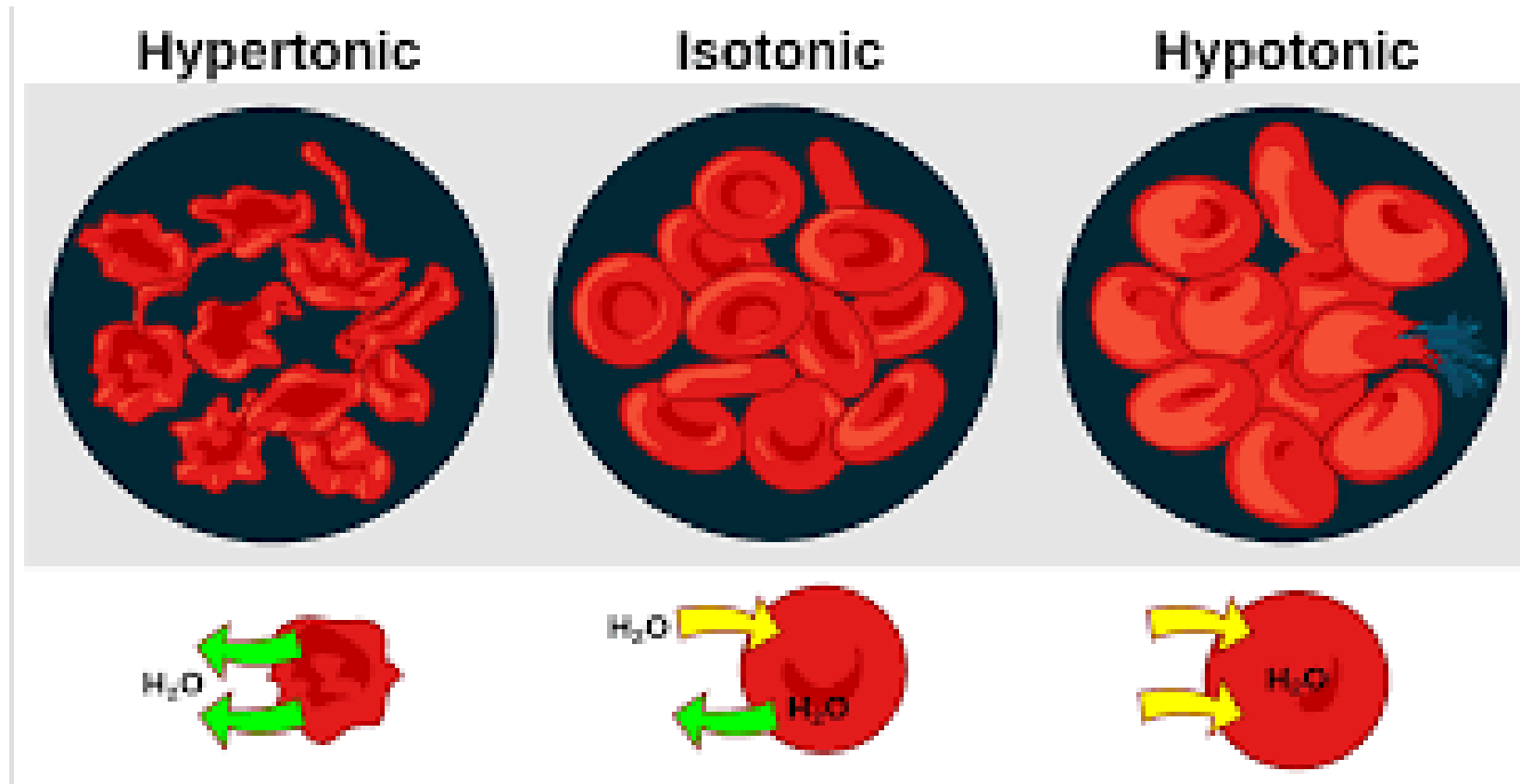


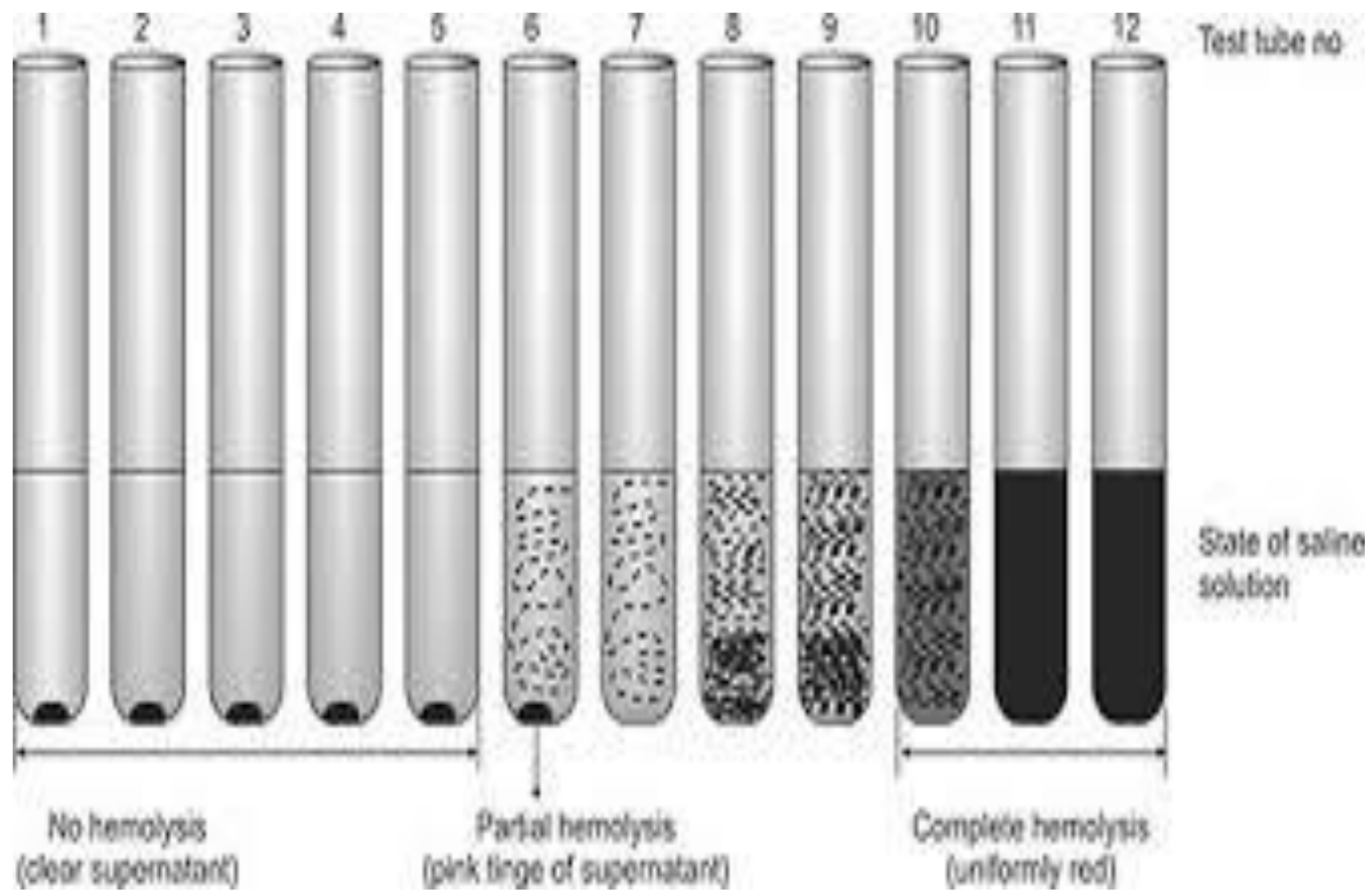
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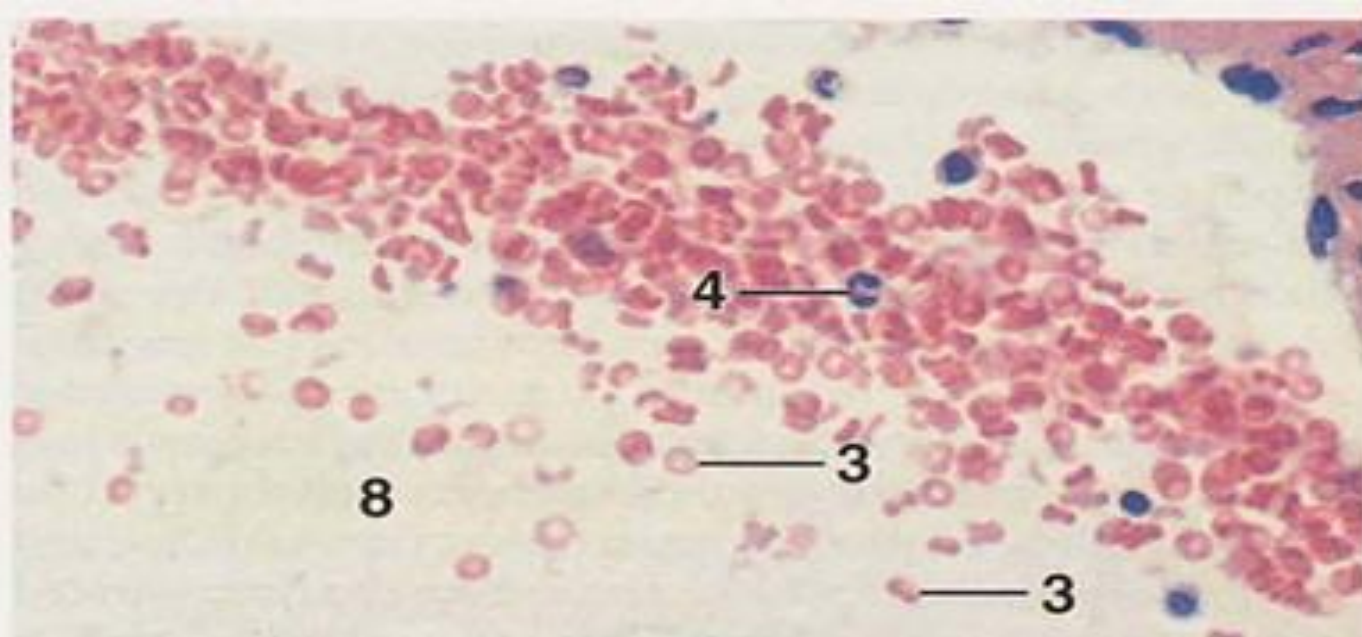


Erythrocyte Fragility(Osmotic Hemolysis)





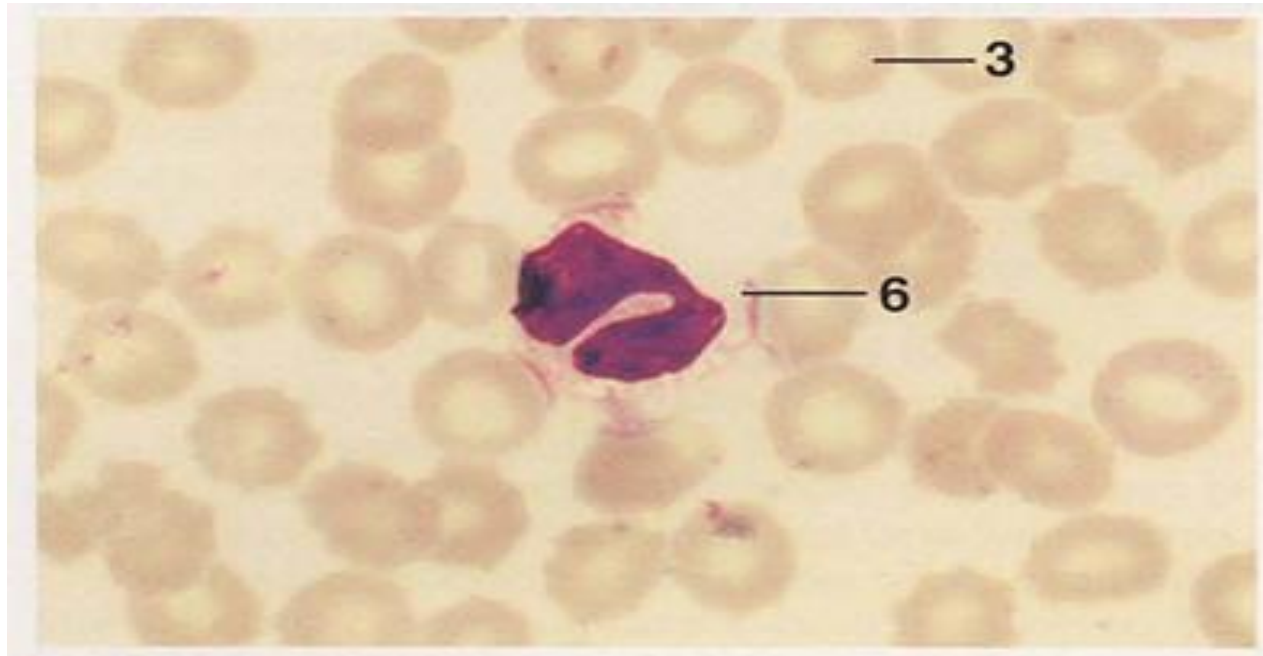
Blood Cells in Cat



KEY

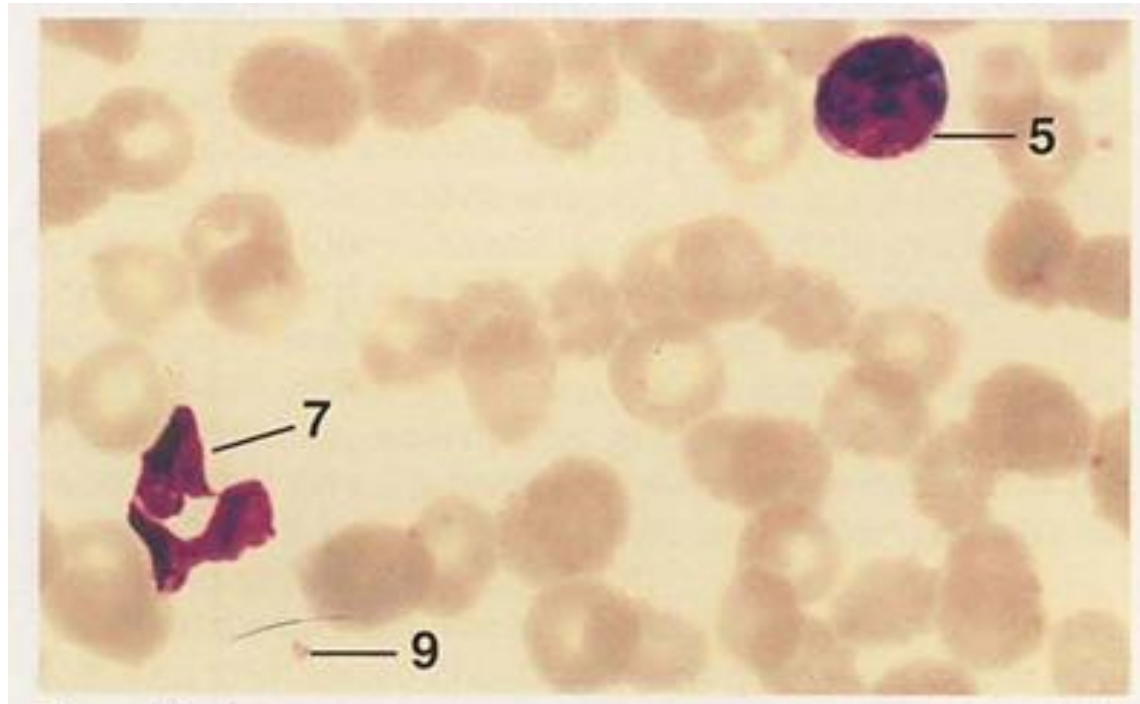
- | | |
|----------------------|------------------------|
| 1. Basophil | 7. Neutrophil |
| 2. Eosinophil | 8. Plasma |
| 3. Erythrocyte | 9. Platelet |
| 4. Leukocyte | 10. Smudged eosinophil |
| 5. Lymphocyte, small | 11. Stain precipitate |
| 6. Monocyte | |

Blood Cells in Dog



- | | |
|----------------------|------------------------|
| 1. Basophil | 7. Neutrophil |
| 2. Eosinophil | 8. Plasma |
| 3. Erythrocyte | 9. Platelet |
| 4. Leukocyte | 10. Smudged eosinophil |
| 5. Lymphocyte, small | 11. Stain precipitate |
| 6. Monocyte | |

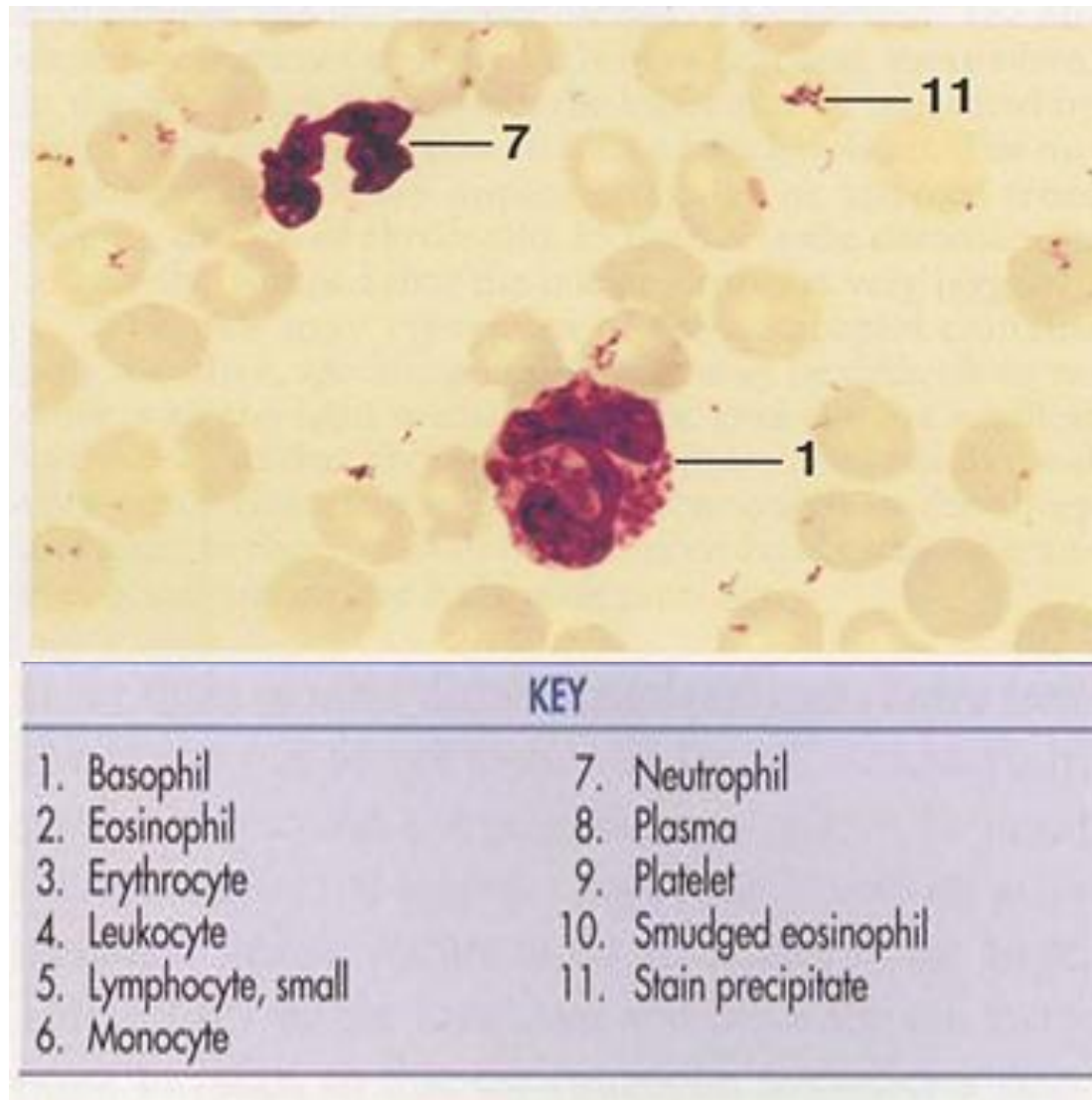
Blood Cells in Dog



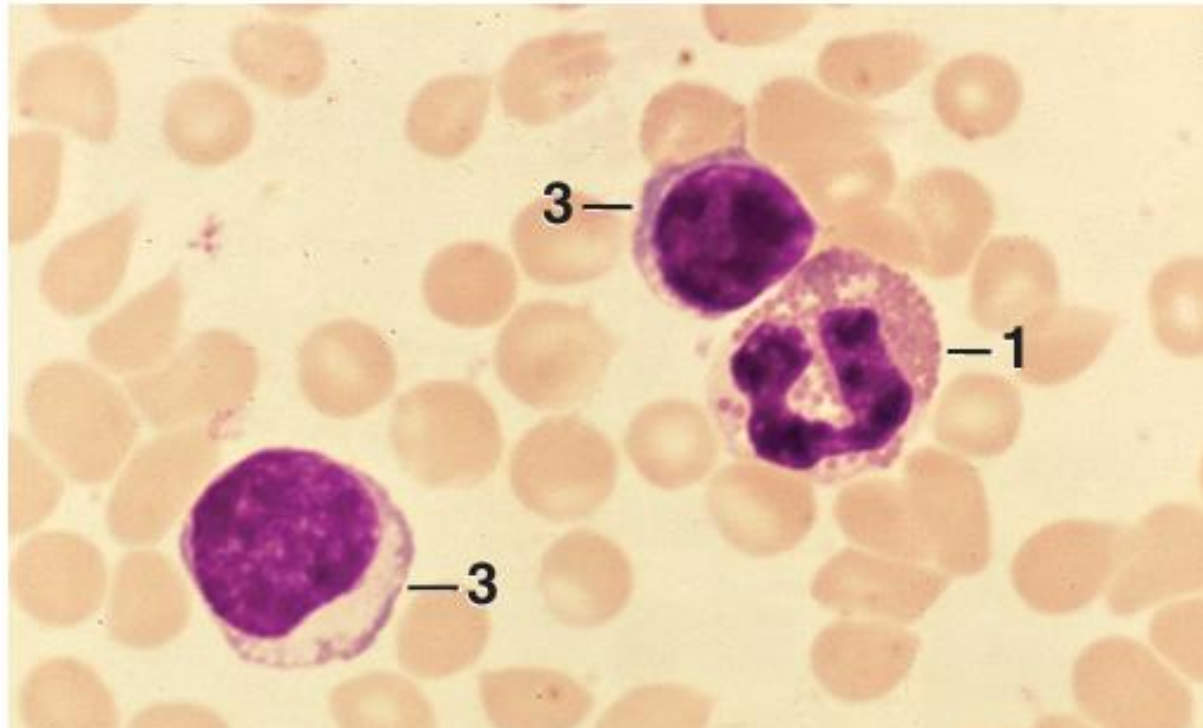
KEY

- | | |
|----------------------|------------------------|
| 1. Basophil | 7. Neutrophil |
| 2. Eosinophil | 8. Plasma |
| 3. Erythrocyte | 9. Platelet |
| 4. Leukocyte | 10. Smudged eosinophil |
| 5. Lymphocyte, small | 11. Stain precipitate |
| 6. Monocyte | |

Blood Cells in Dog



Blood Cells in Cow

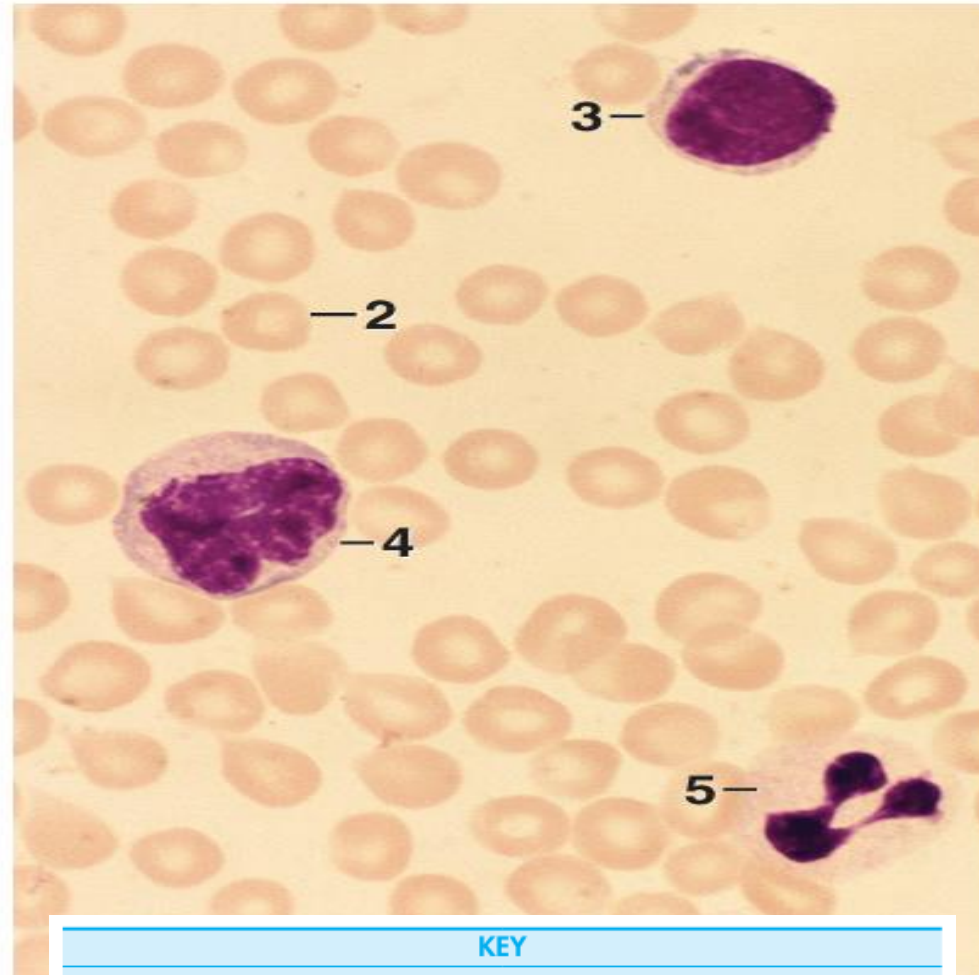


KEY

- 1. Eosinophil
- 2. Erythrocyte
- 3. Lymphocyte

- 4. Monocyte
- 5. Neutrophil

Blood Cells in Cow



KEY

- | | |
|----------------|---------------|
| 1. Eosinophil | 4. Monocyte |
| 2. Erythrocyte | 5. Neutrophil |
| 3. Lymphocyte | |

Blood Cells in Horse(buffy coat)

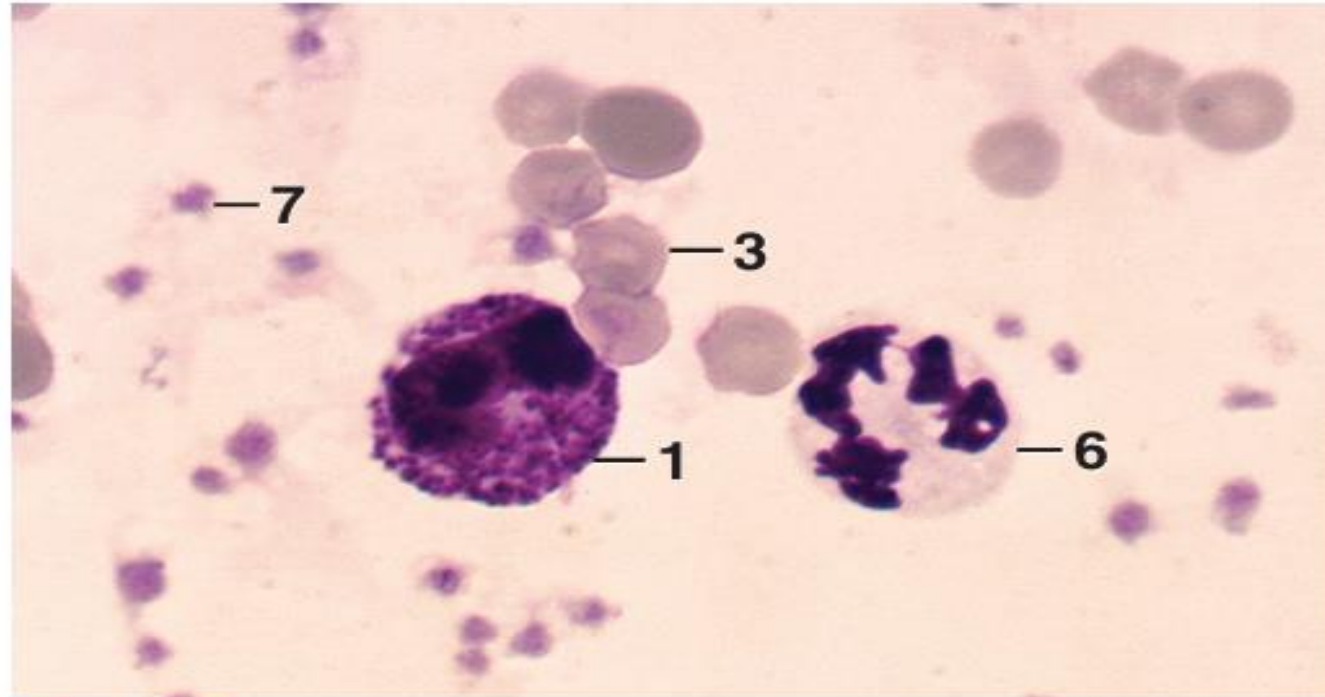


Figure 6.18

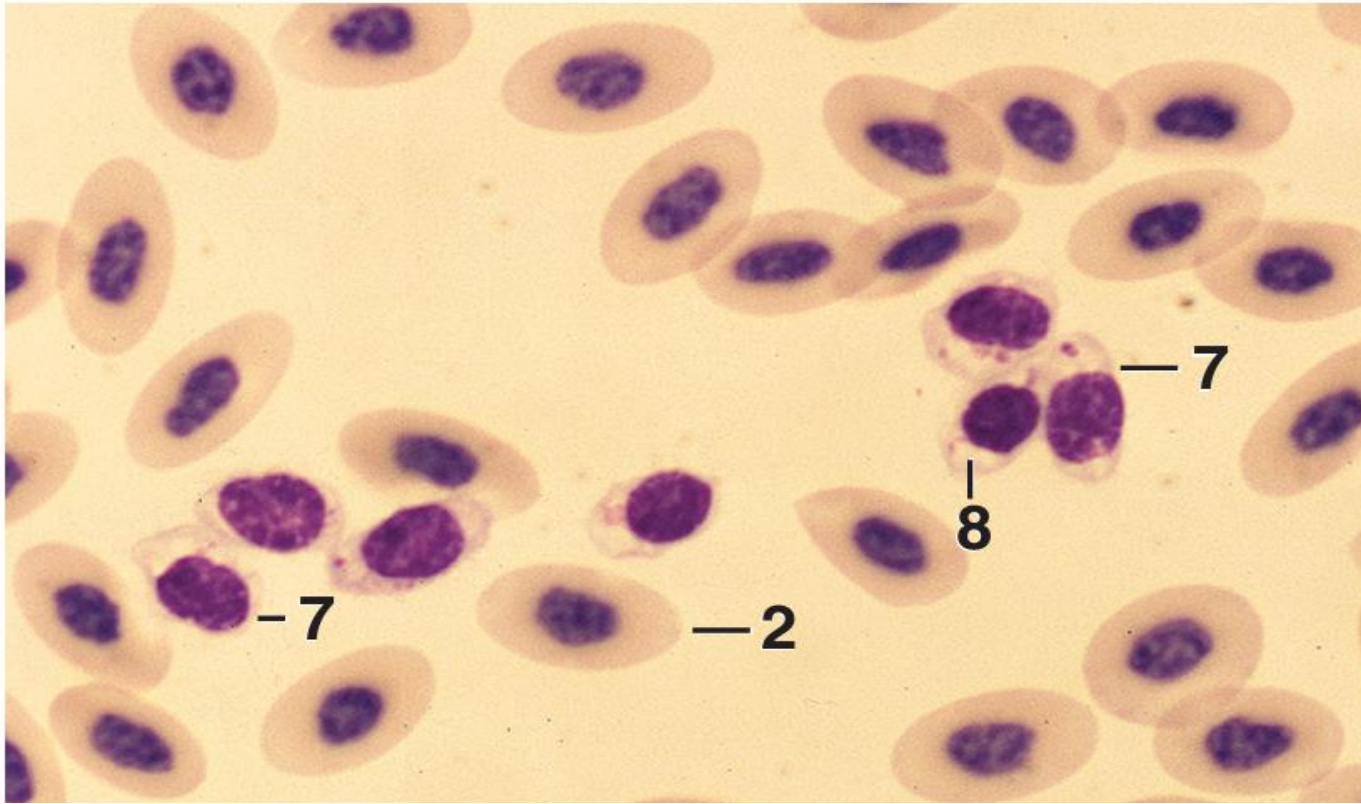
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KEY

1. Basophil
2. Eosinophil
3. Erythrocyte
4. Lymphocyte

5. Monocyte
6. Neutrophil
7. Platelet

Blood Cells in Chicken

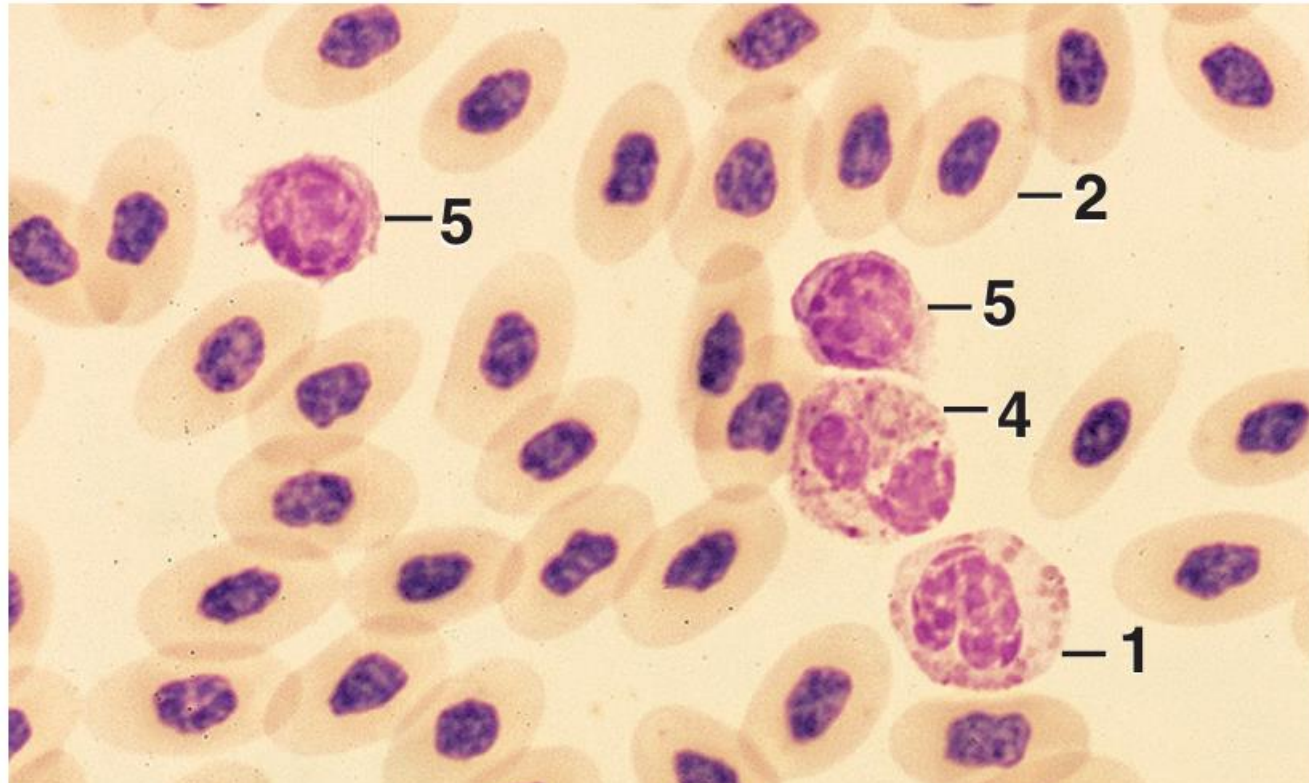


KEY

1. Eosinophil
2. Erythrocyte
3. Granulocyte
4. Heterophil

5. Lymphocyte
6. Monocyte
7. Thrombocyte
8. Vacuole

Blood Cells in Chicken



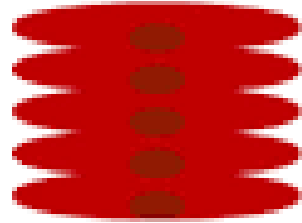
KEY

- 1. Eosinophil
- 2. Erythrocyte
- 3. Granulocyte
- 4. Heterophil

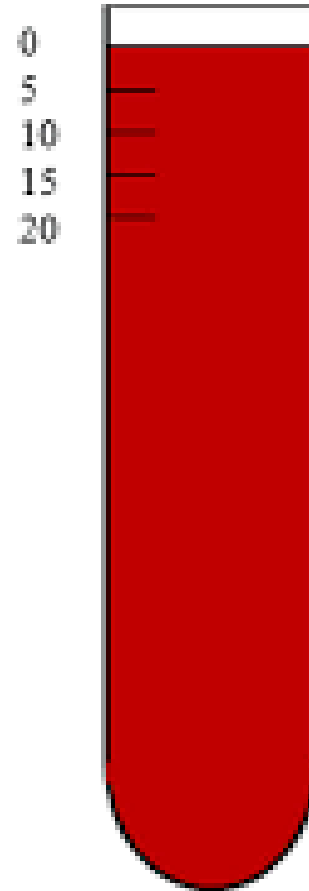
- 5. Lymphocyte
- 6. Monocyte
- 7. Thrombocyte
- 8. Vacuole

Erythrocyte Sedimentation Rate(ESR)

A



B



C



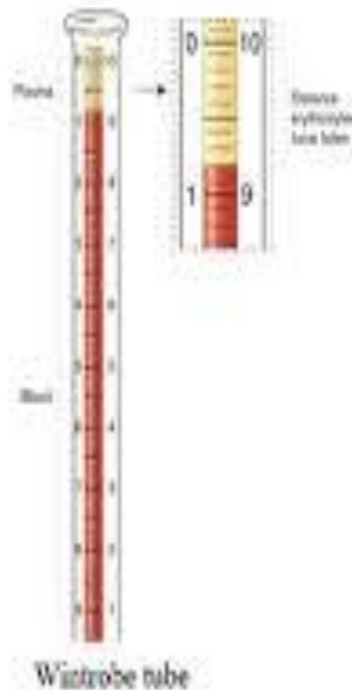
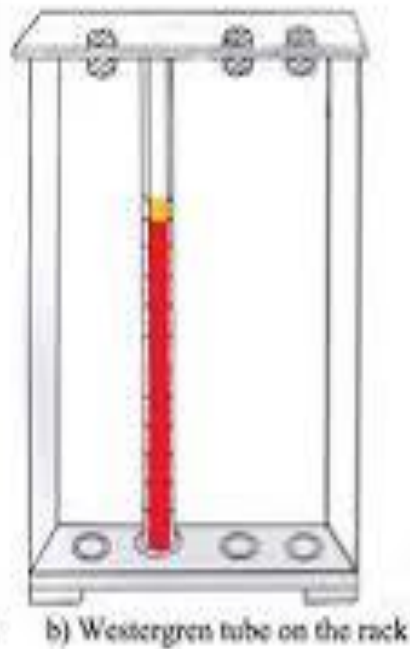
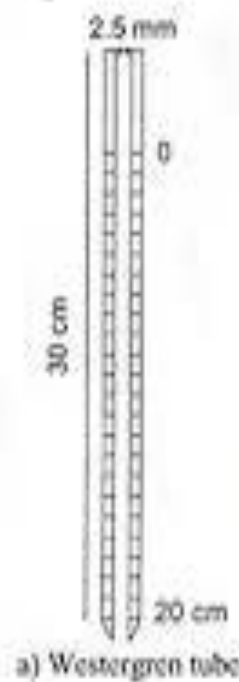
Erythrocyte Sedimentation Rate (ESR)

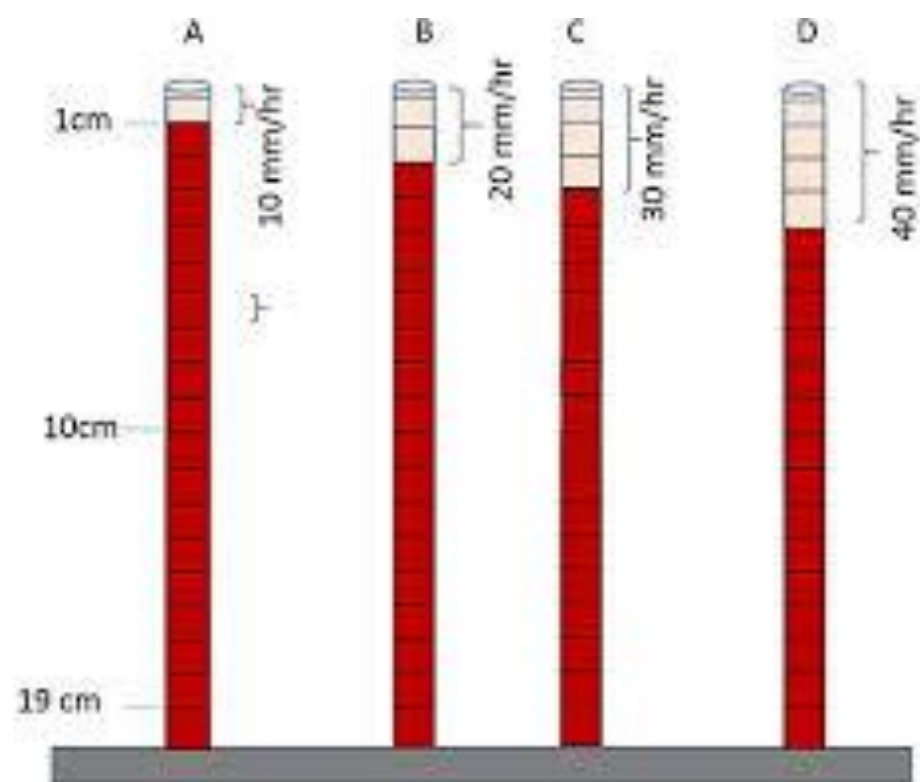


1hr



The distance, in mm, the RBC fall in 1 hr is the Sed Rate





Erythrocyte sedimentation rate

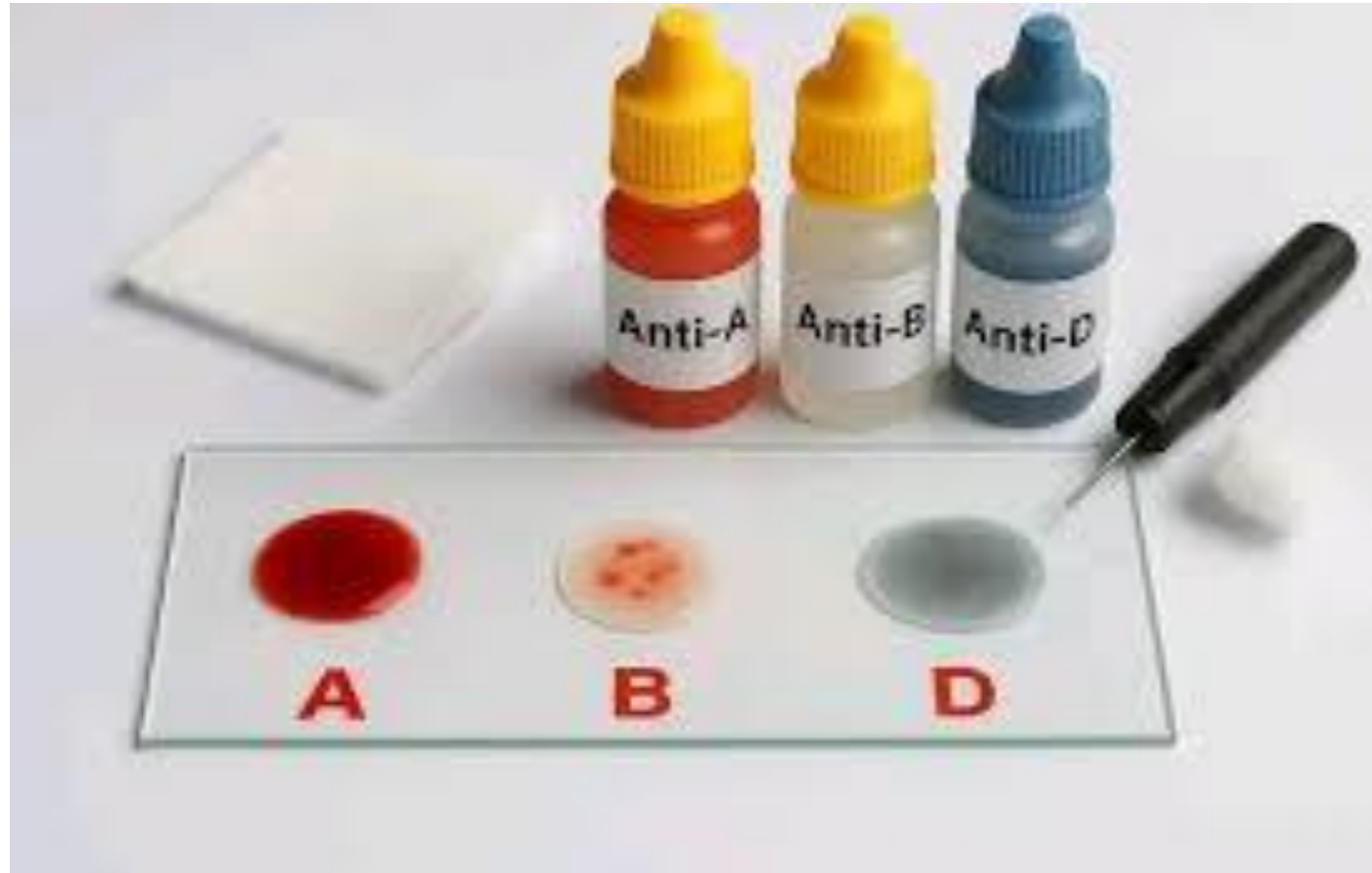


Blood Typing Test

Table 36-1 Blood Types With Their Genotypes and Constituent Agglutinogens and Agglutinins

Genotypes	Blood Types	Agglutinogens	Agglutinins
OO	O	–	Anti-A and Anti-B
OA or AA	A	A	Anti-B
OB or BB	B	B	Anti-A
AB	AB	A and B	–

Blood Typing Test



Blood Typing Test



Blood Typing Test

Table 36-2 Blood Typing: Agglutination of Cells of Different Blood Types With Anti-A or Anti-B Agglutinins in the Sera

Red Blood Cell Types	Sera	
	Anti-A	Anti-B
O	–	–
A	+	–
B	–	+
AB	+	+

Hemostasis & Blood coagulation

The term *hemostasis* means prevention of blood loss. Whenever a vessel is severed or ruptured, hemostasis is achieved by several mechanisms: (1) vascular constriction; (2) formation of a platelet plug; (3) formation of a blood clot as a result of blood coagulation; and (4) eventual growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.

Hemostasis & Blood coagulation

VASCULAR CONSTRICTION

Immediately after a blood vessel has been cut or ruptured, the trauma to the vessel wall causes smooth muscle in the wall to contract; this instantaneously reduces the flow of blood from the ruptured vessel. The contraction results from the following: (1) local myogenic spasm; (2) local autacoid factors from the traumatized tissues, vascular endothelium, and blood platelets; and (3) nervous reflexes. The nervous reflexes are initiated by pain nerve impulses or other sensory impulses that originate from the traumatized vessel or nearby tissues. However, even more vasoconstriction probably results from local *myogenic contraction* of the blood vessels initiated by direct damage to the vascular wall. And, for the smaller vessels, the platelets are responsible for much of the vasoconstriction by releasing a vasoconstrictor substance, *thromboxane A₂*.

The more severely a vessel is traumatized, the greater the degree of vascular spasm. The spasm can last for many minutes or even hours, during which time the processes of platelet plugging and blood coagulation can take place.

Hemostasis & Blood coagulation

FORMATION OF THE PLATELET PLUG

If the cut in the blood vessel is very small—many very small vascular holes develop throughout the body each day—the cut is often sealed by a *platelet plug* rather than by a blood clot. To understand this process, it is important that we first discuss the nature of platelets themselves.

Hemostasis & Blood coagulation

Platelets have many functional characteristics of whole cells, even though they do not have nuclei and cannot reproduce. In their cytoplasm are the following: (1) *actin* and *myosin molecules*, which are contractile proteins similar to those found in muscle cells, and still another contractile protein, *thrombosthenin*, that can cause the platelets to contract; (2) residuals of both the *endoplasmic reticulum* and *Golgi apparatus* that synthesize various enzymes and especially store large quantities of calcium ions; (3) mitochondria and enzyme systems that are capable of forming *adenosine triphosphate* (ATP) and *adenosine diphosphate* (ADP); (4) enzyme systems that synthesize *prostaglandins*, which are local hormones that cause many vascular and other local tissue reactions; (5) an important protein called *fibrin-stabilizing factor*, which we discuss later in relation to blood coagulation; and (6) a *growth factor* that causes vascular endothelial cells, vascular smooth muscle cells, and fibroblasts to multiply and grow, thus causing cellular growth that eventually helps repair damaged vascular walls.

Hemostasis & Blood coagulation

- On the platelet cell membrane surface is a coat of *glycoproteins* that repulses adherence to normal endothelium and yet causes adherence to *injured* areas of the vessel wall, especially to injured endothelial cells and even more so to any exposed collagen from deep within the vessel wall. In addition, the platelet membrane contains large amounts of *phospholipids* that activate multiple stages in the blood-clotting process, as discussed later.

Hemostasis & Blood coagulation

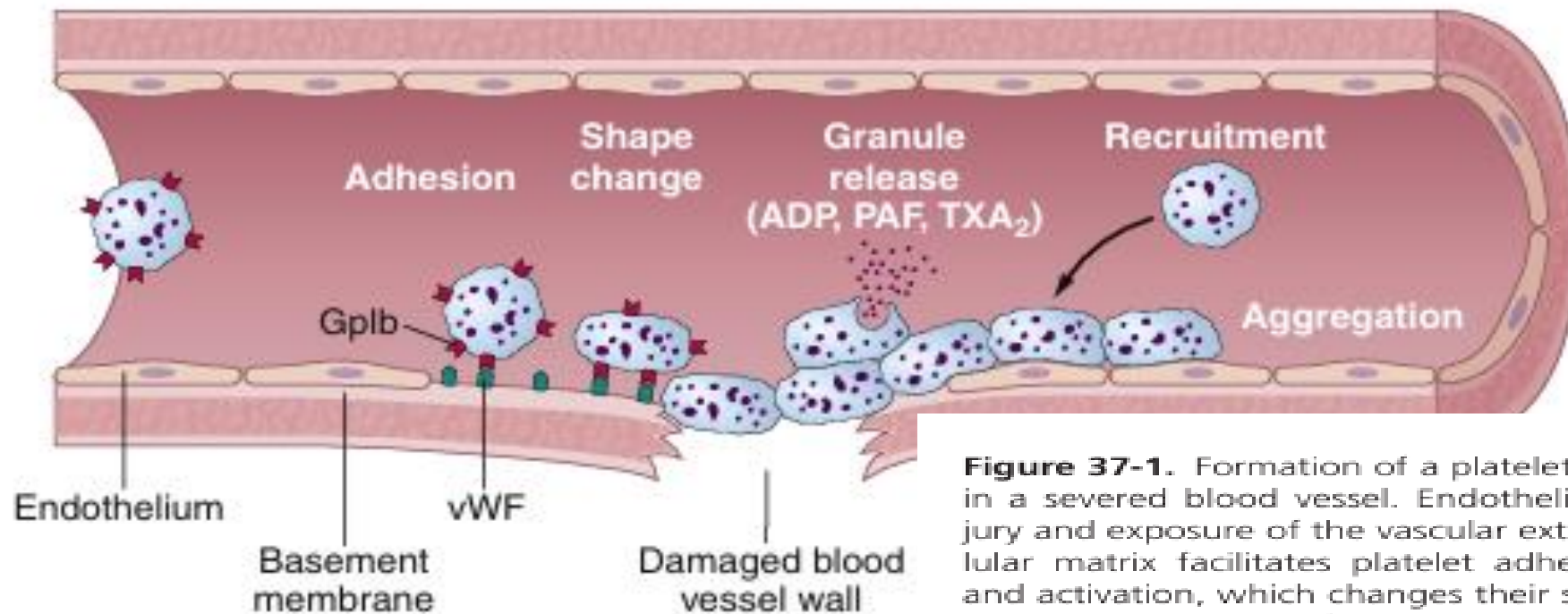


Figure 37-1. Formation of a platelet plug in a severed blood vessel. Endothelial injury and exposure of the vascular extracellular matrix facilitates platelet adhesions and activation, which changes their shape and causes release of adenosine diphosphate (ADP), thromboxane A₂ (TXA₂), and platelet-activating factor (PAF). These platelet-secreted factors recruit additional platelets (aggregation) to form a hemostatic plug. Von Willebrand factor (vWF) serves as an adhesion bridge between sub-endothelial collagen and the glycoprotein Ib (GpIb) platelet receptor.

Hemostasis & Blood coagulation

MECHANISM OF BLOOD COAGULATION

GENERAL MECHANISM

More than 50 important substances that cause or affect blood coagulation have been found in the blood and in the tissues—some that promote coagulation, called *procoagulants*, and others that inhibit coagulation, called *anticoagulants*. Whether blood will coagulate depends on the balance between these two groups of substances. In the blood stream, the anticoagulants normally predominate, so the blood does not coagulate while it is circulating

in the blood vessels. However, when a vessel is ruptured, procoagulants from the area of tissue damage become activated and override the anticoagulants, and then a clot does develop.

Hemostasis & Blood coagulation

Table 37-1 Clotting Factors in Blood and Their Synonyms^a

Clotting Factor	Synonym(s)
Fibrinogen	Factor I
Prothrombin	Factor II
Tissue factor	Factor III; tissue thromboplastin
Calcium	Factor IV
Factor V	Proaccelerin; labile factor; Ac-globulin (Ac-G)
Factor VII	Serum prothrombin conversion accelerator (SPCA); proconvertin; stable factor
Factor VIII	Antihemophilic factor (AHF); antihemophilic globulin (AHG); antihemophilic factor A
Factor IX	Plasma thromboplastin component (PTC); Christmas factor; antihemophilic factor B
Factor X	Stuart factor; Stuart-Prower factor
Factor XI	Plasma thromboplastin antecedent (PTA); antihemophilic factor C
Factor XII	Hageman factor
Factor XIII	Fibrin-stabilizing factor
Prekallikrein	Fletcher factor
High-molecular-weight kininogen	Fitzgerald factor; high-molecular-weight kininogen (HMWK)
Platelets	

Hemostasis & Blood coagulation

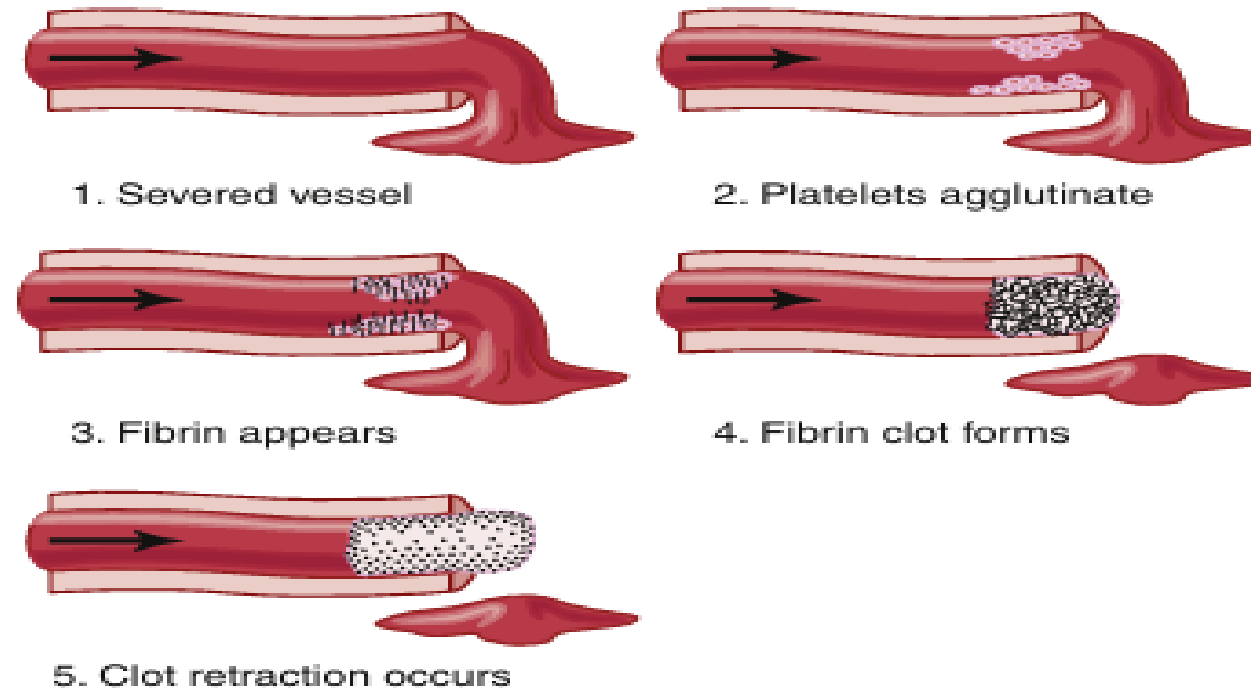
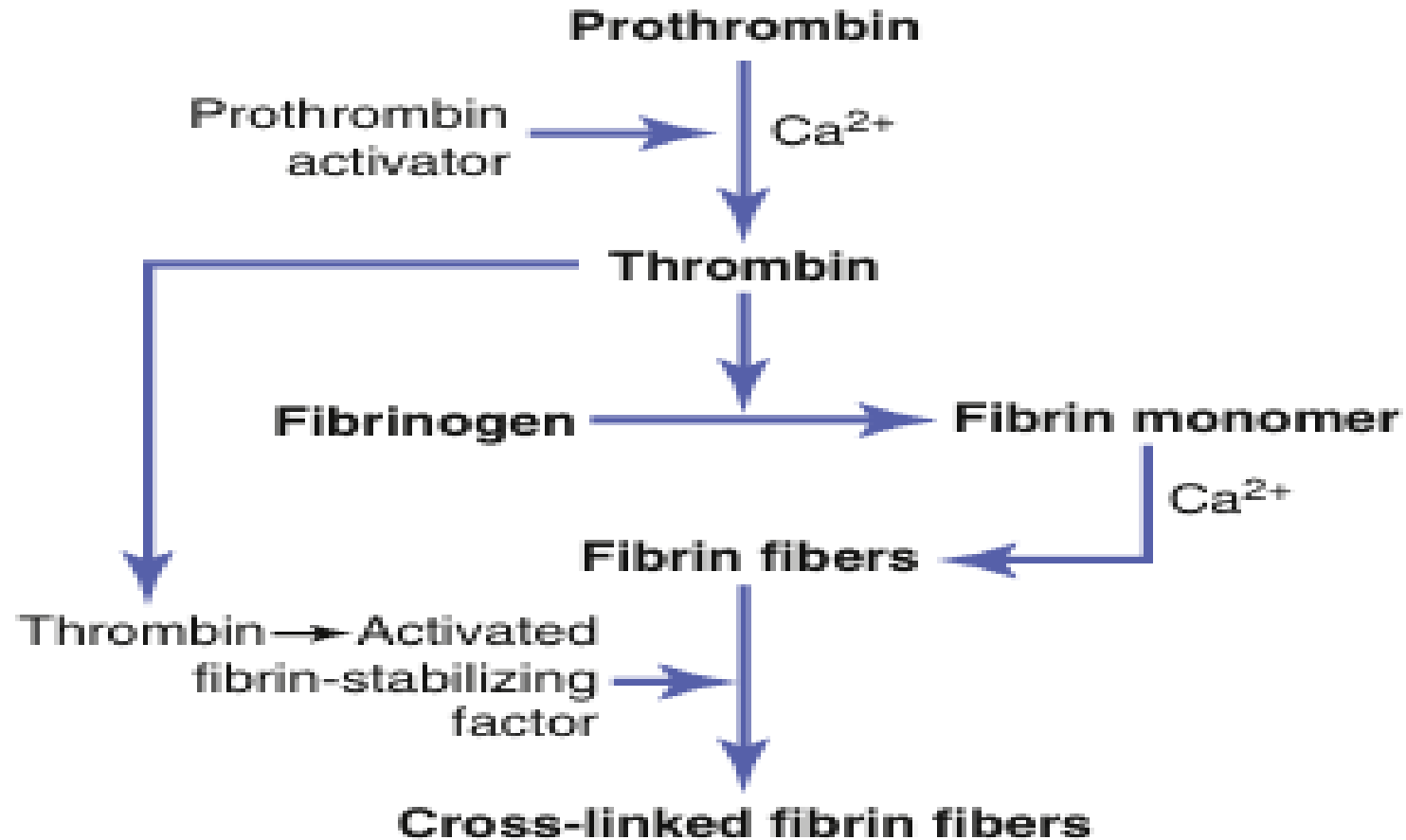


Figure 37-2. Clotting process in a traumatized blood vessel. (Modified from Seegers WH: *Hemostatic Agents*. Springfield, IL: Charles C Thomas, 1948.)

Hemostasis & Blood coagulation



Hemostasis & Blood coagulation

Clot Retraction and Expression of Serum. Within a few minutes after a clot is formed, it begins to contract and usually expresses most of the fluid from the clot within 20 to 60 minutes. The fluid expressed is called *serum* because all its fibrinogen and most of the other clotting factors have been removed; in this way, serum differs from plasma and cannot clot because it lacks these factors.

Hemostasis & Blood coagulation

Extrinsic Pathway for Initiating Clotting

The extrinsic pathway for initiating the formation of prothrombin activator begins with a traumatized vascular wall or traumatized extravascular tissues that come in contact with the blood. This condition leads to the following steps, as shown in **Figure 37-4** and **Figure 37-5**:

Hemostasis & Blood coagulation

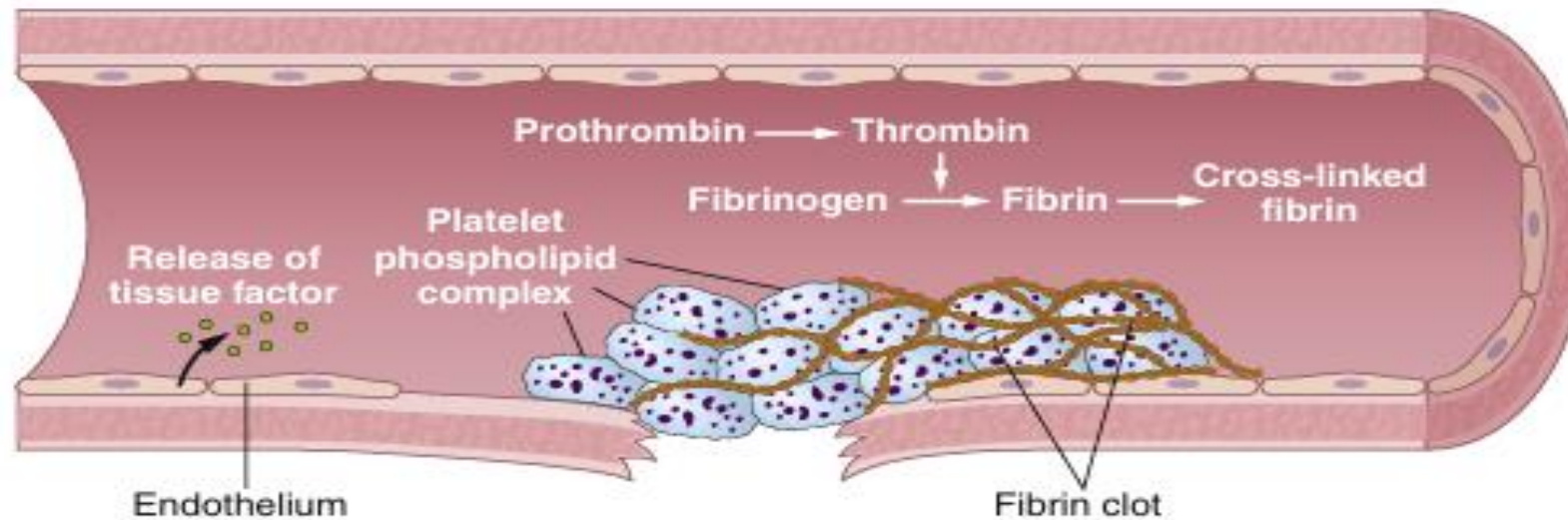


Figure 37-4. Coagulation cascade after vascular injury. Exposure of blood to the vascular wall causes release of tissue factor (also called factor III or thromboplastin) from endothelial cells, phospholipid expression, activation of thrombin, which then acts on fibrinogen to form fibrin, and fibrin polymerization to form a meshwork that stabilizes the platelet plug.

Hemostasis & Blood coagulation

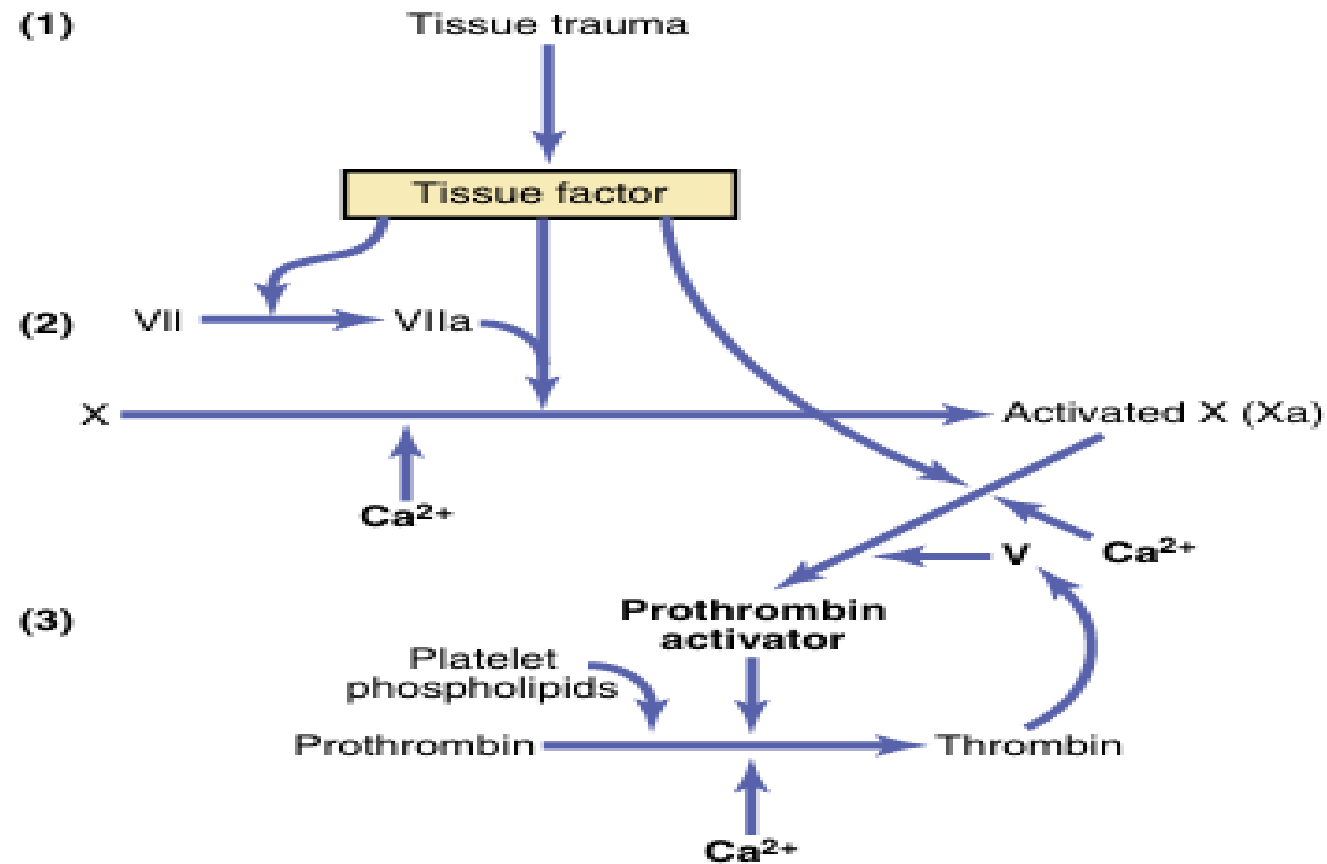


Figure 37-5. Extrinsic pathway for initiating blood clotting.

Hemostasis & Blood coagulation

Intrinsic Pathway for Initiating Clotting

The second mechanism for initiating formation of prothrombin activator, and therefore for initiating clotting, *begins with trauma to the blood or exposure of the blood to collagen* from a traumatized blood vessel wall. Then the process continues through the series of cascading reactions shown in **Figure 37-6**.

Hemostasis & Blood coagulation

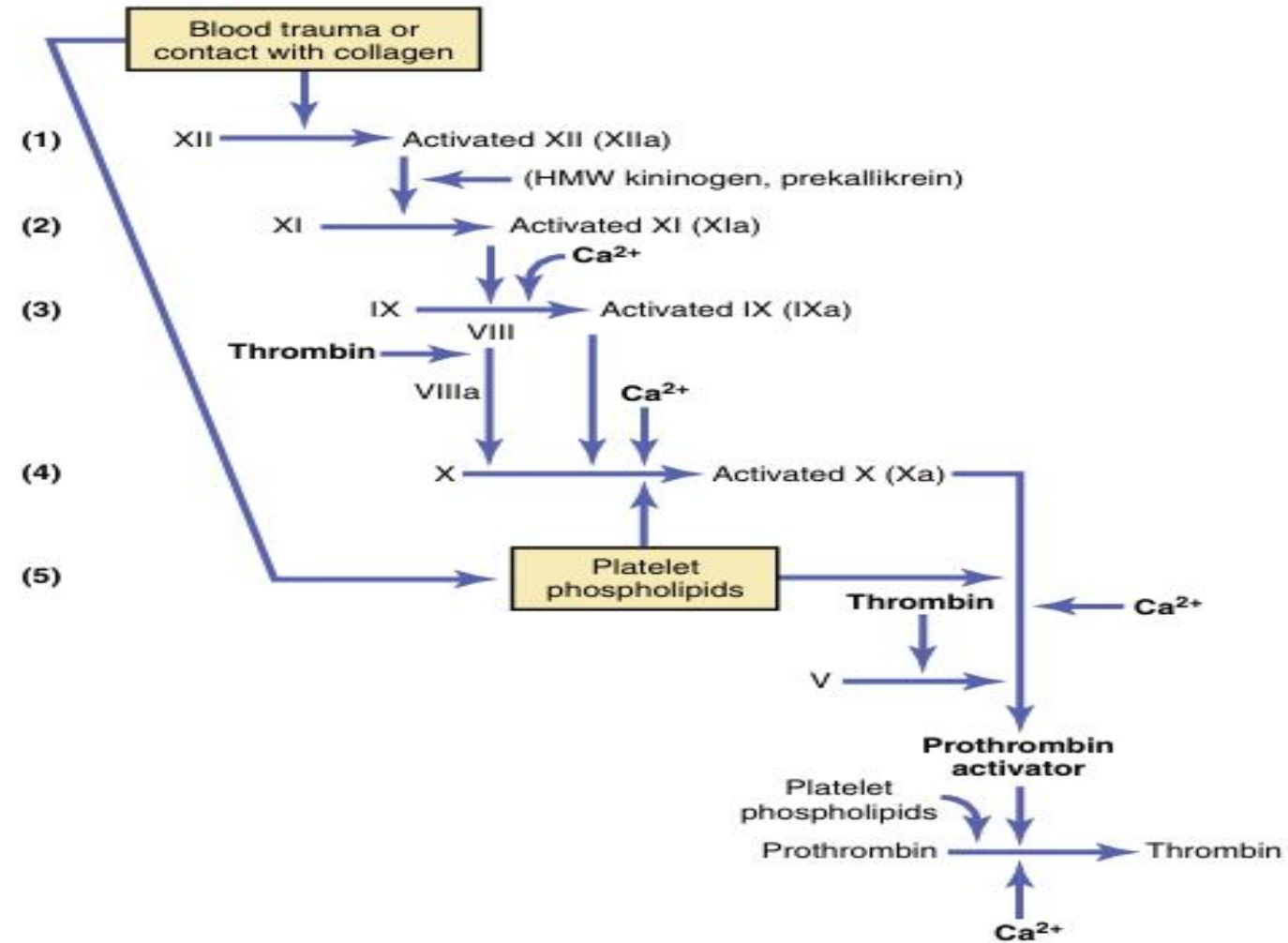


Figure 37-6. Intrinsic pathway for initiating blood clotting. HMW, High-molecular weight.