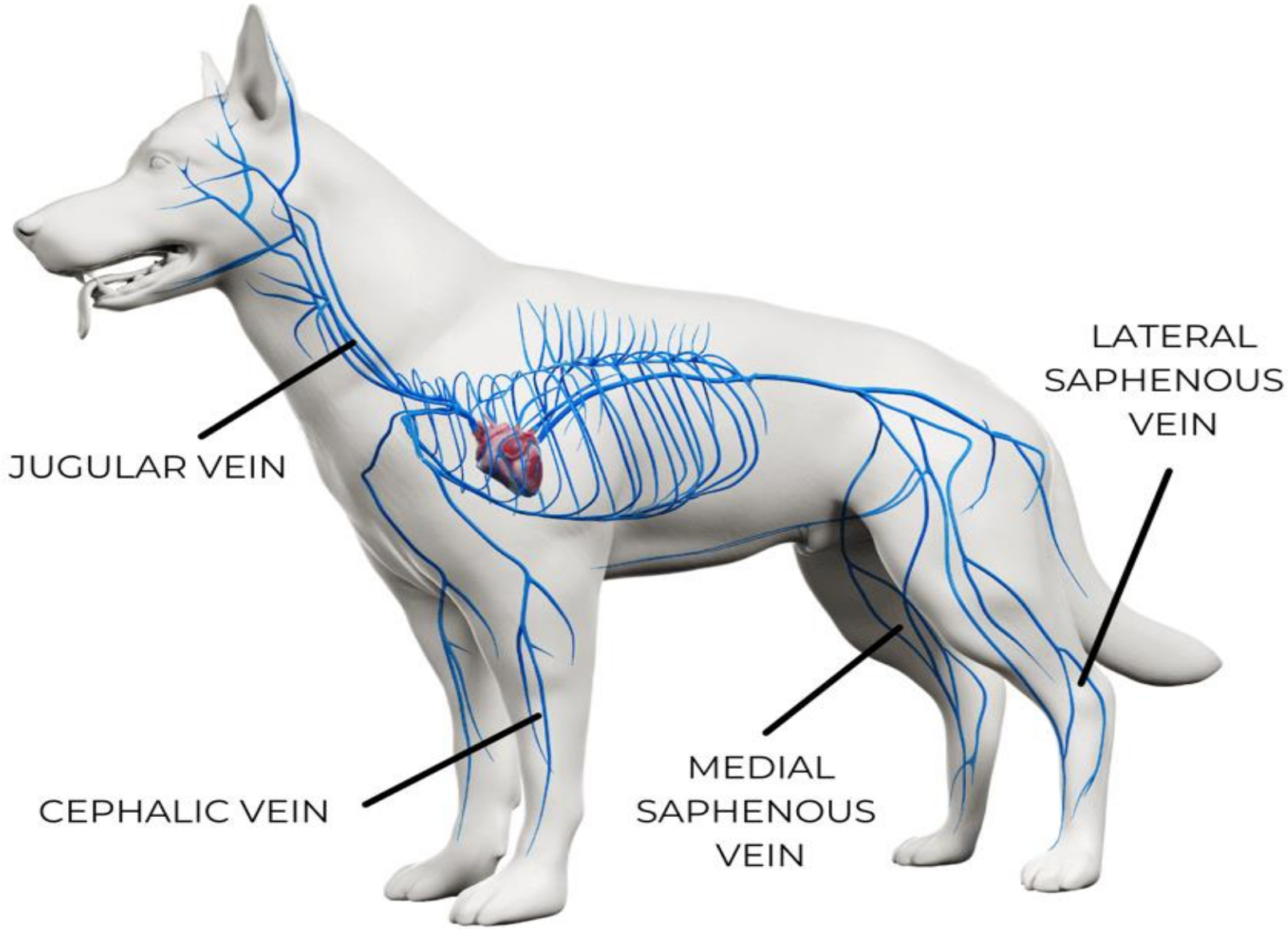
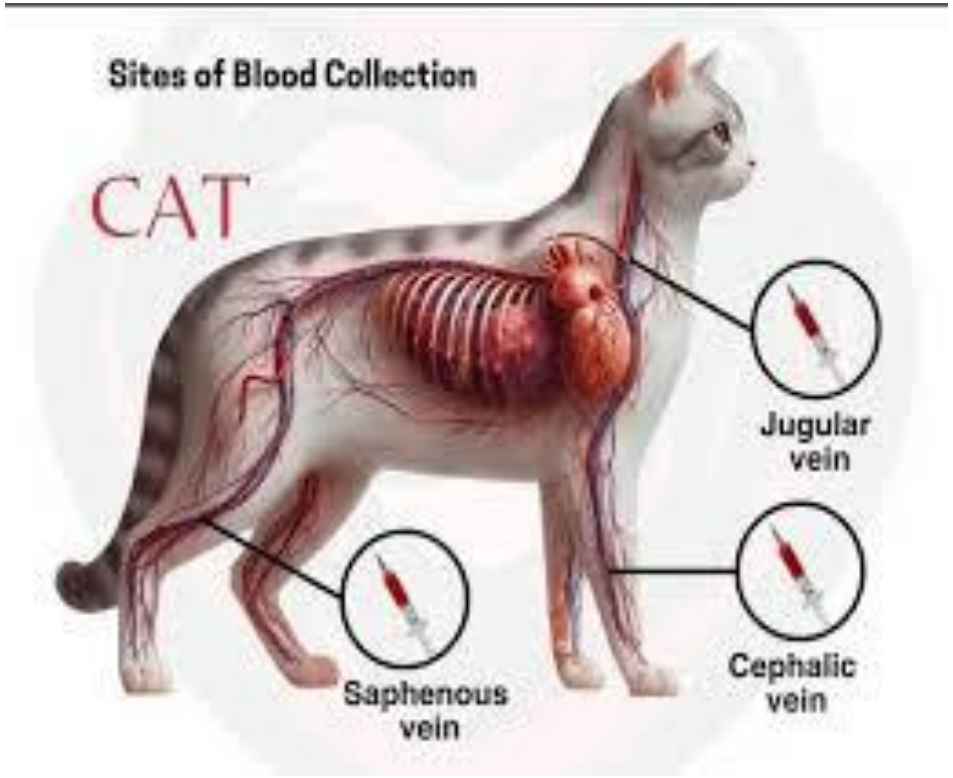
Complete Blood Count(CBC)
Full Blood Count (FBC)
Full Haemogram(FHG)

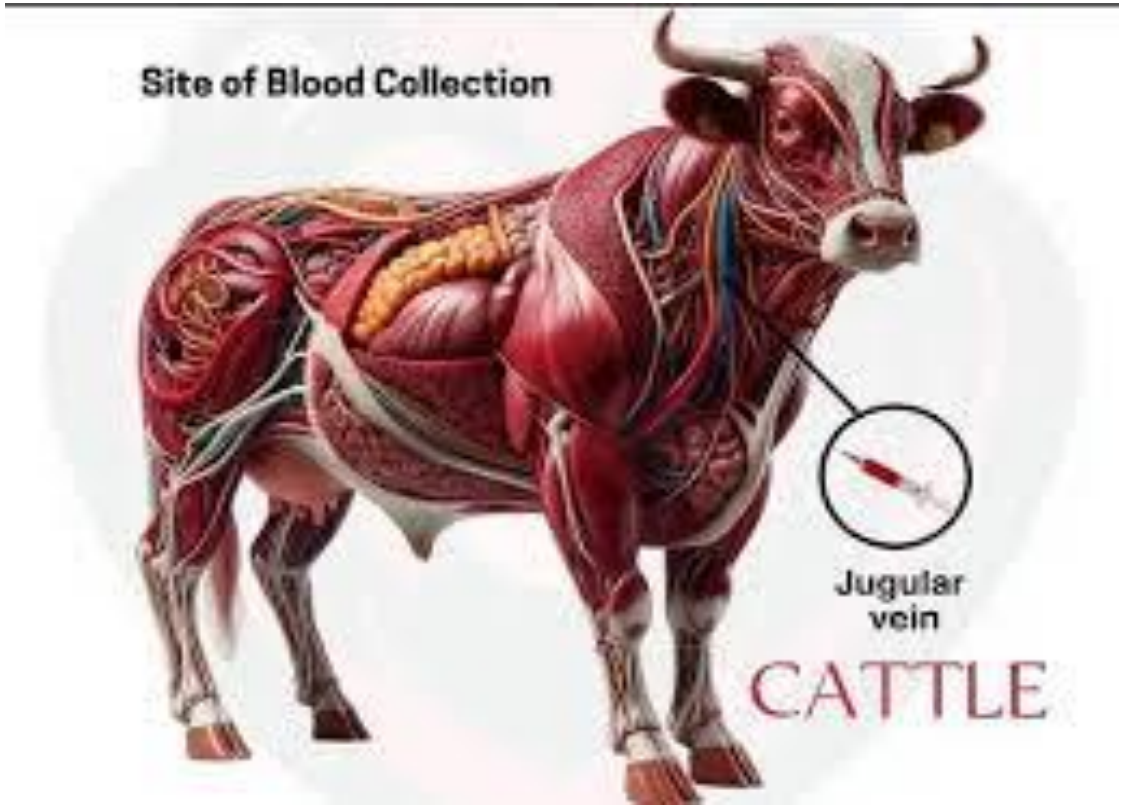
The *CBC* indicates, the counts of *white Blood Cells*(*WBC*), *Red Blood Cells*(*RBC*), *Platelets*(*Plt*), the concentration of Hemoglobin(*Hgb*),and the *Hematocrit* (*Hct*), the *RBC indices*, which indicate the average size and Hemoglobin content of *RBC* and a *differential* *WBC*, which counts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

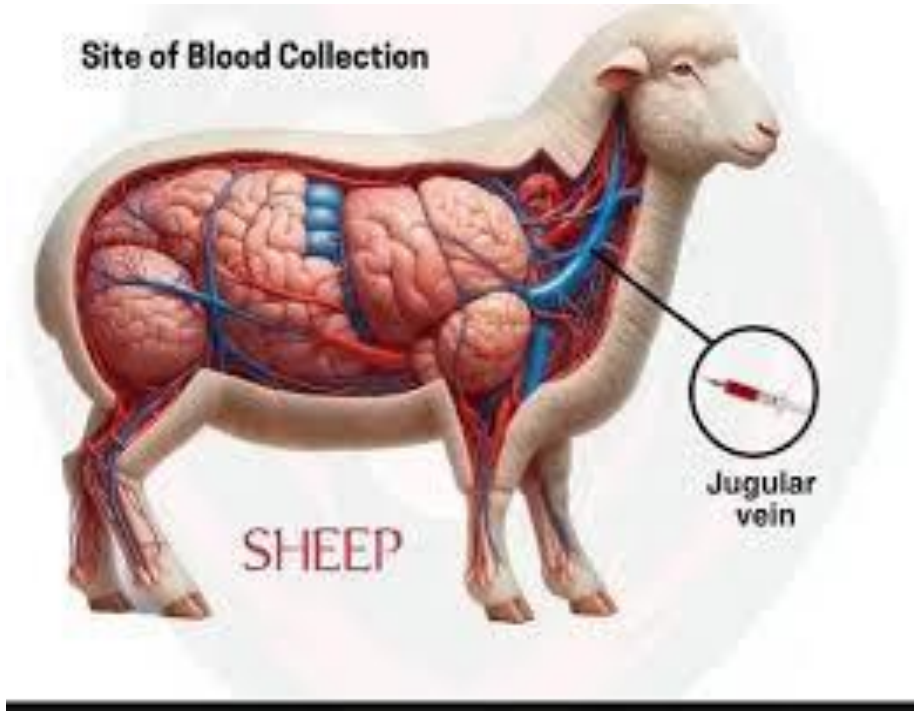

JUGULAR VEIN
LATERAL SAPHENOUS VEIN
CEPHALIC VEIN
MEDIAL SAPHENOUS VEIN

Sites of Blood Collection in Animals

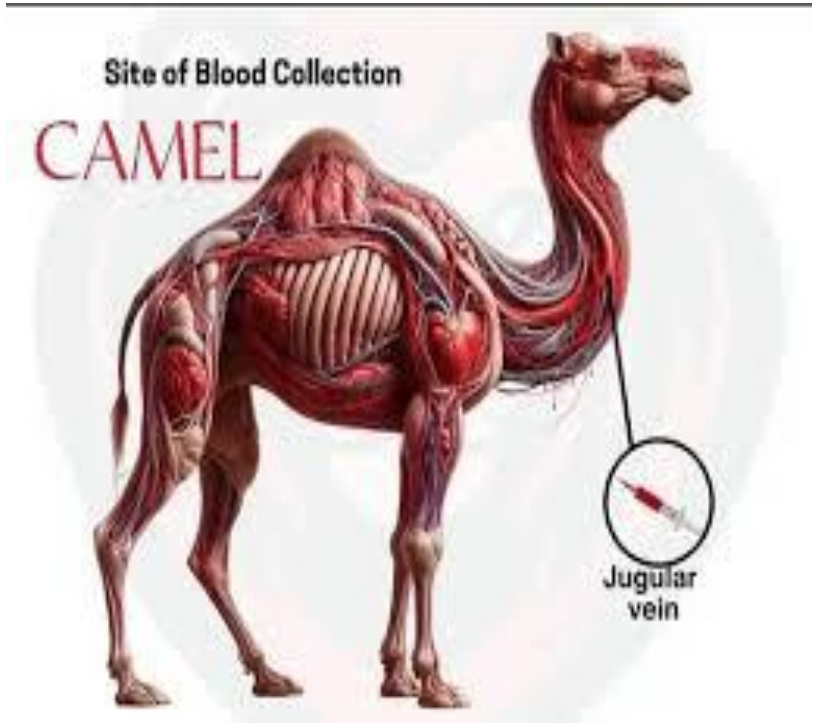


Sites of Blood Collection in Animals

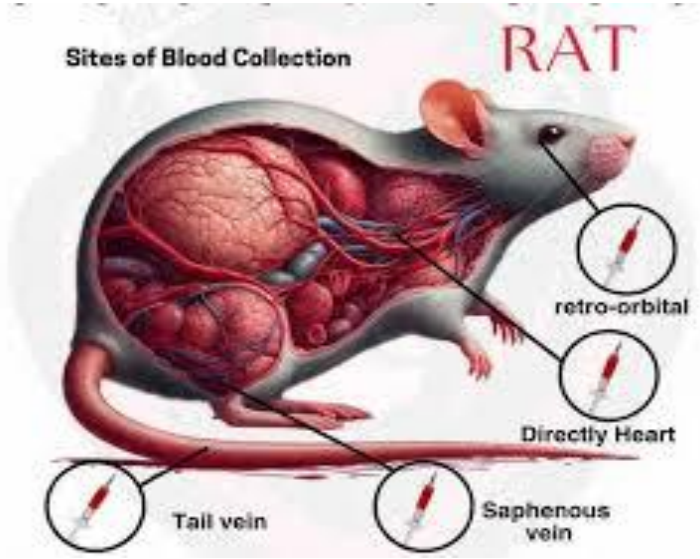


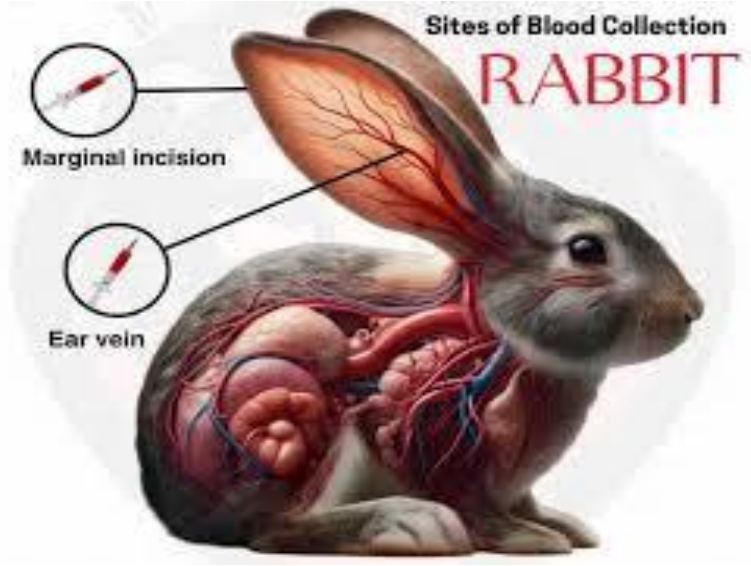


Sites of Blood Collection in Animals

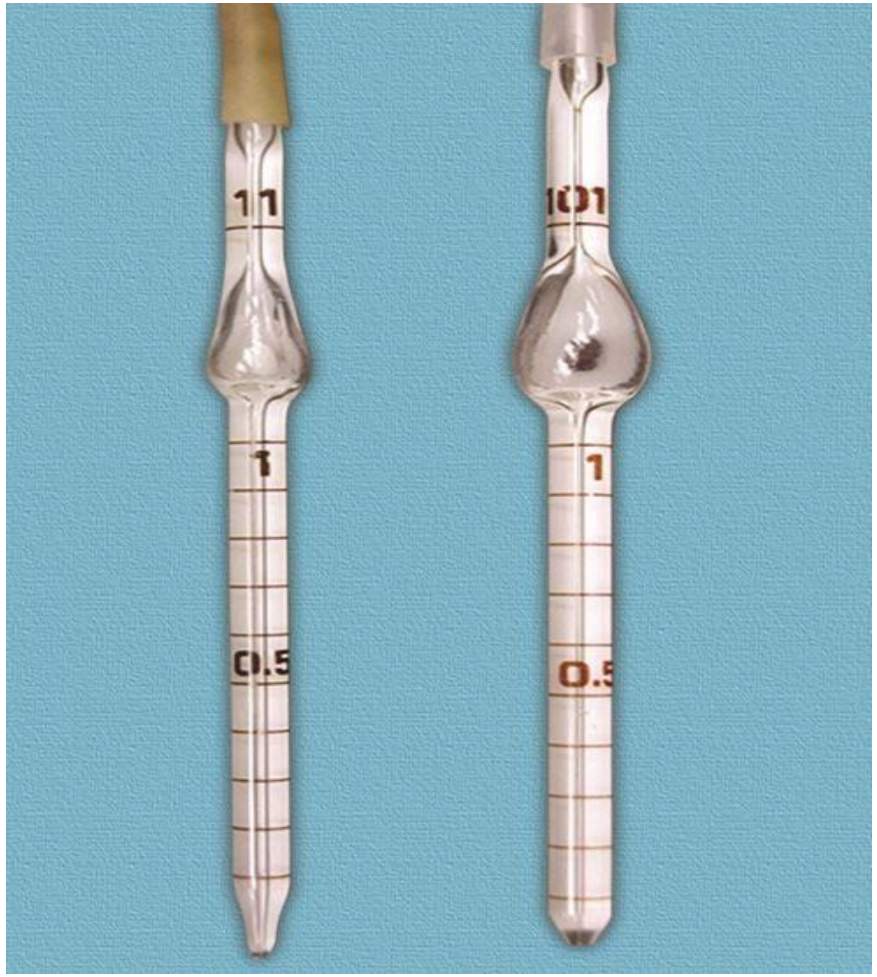


Sites of Blood Collection in Animals





Blood diluting pipettes

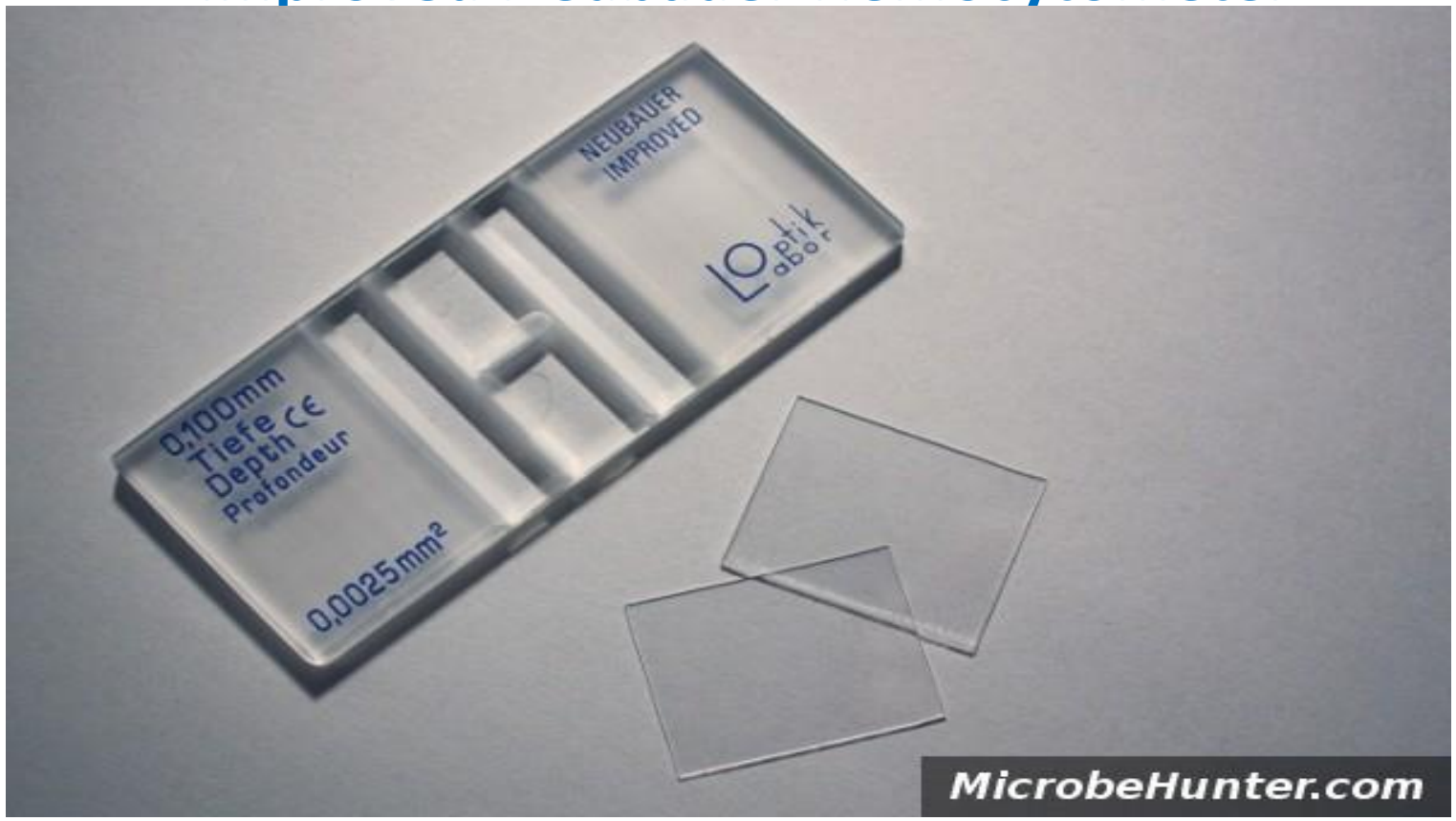


Blood diluting pipettes

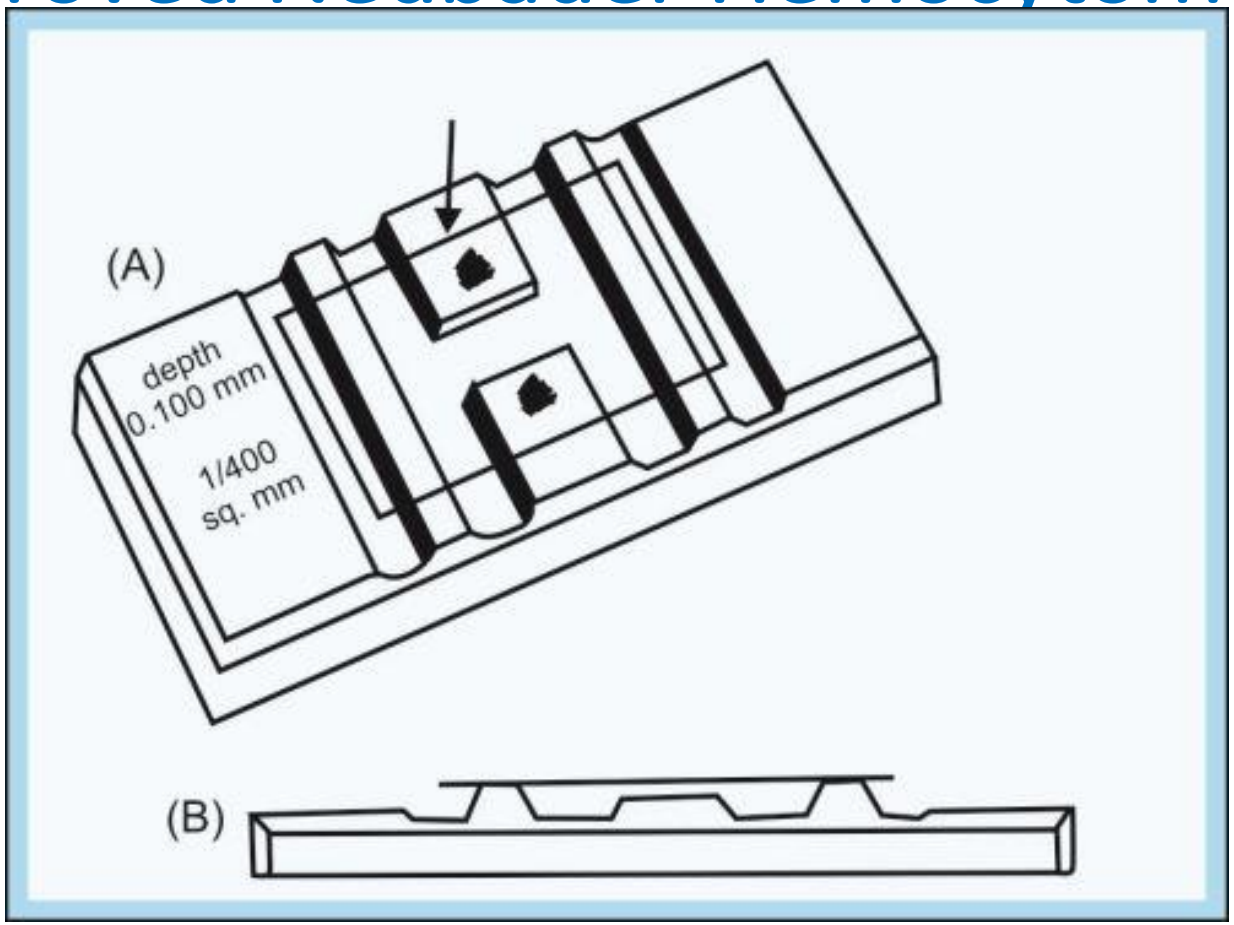
Blood diluting fluids

Hemocytometer

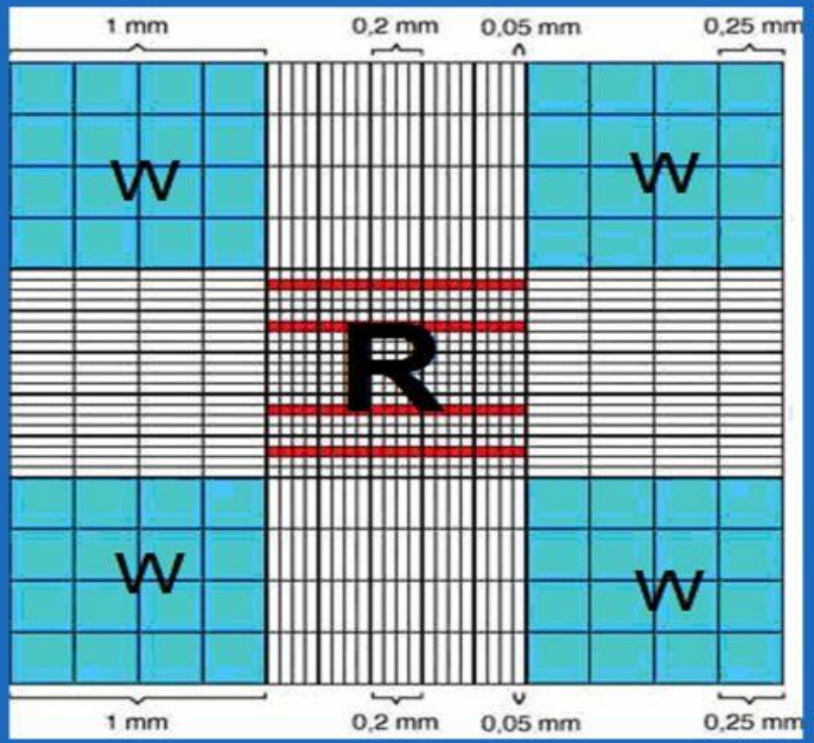
Improved Neubauer Hemocytometer



Improved Neubauer Hemocytometer

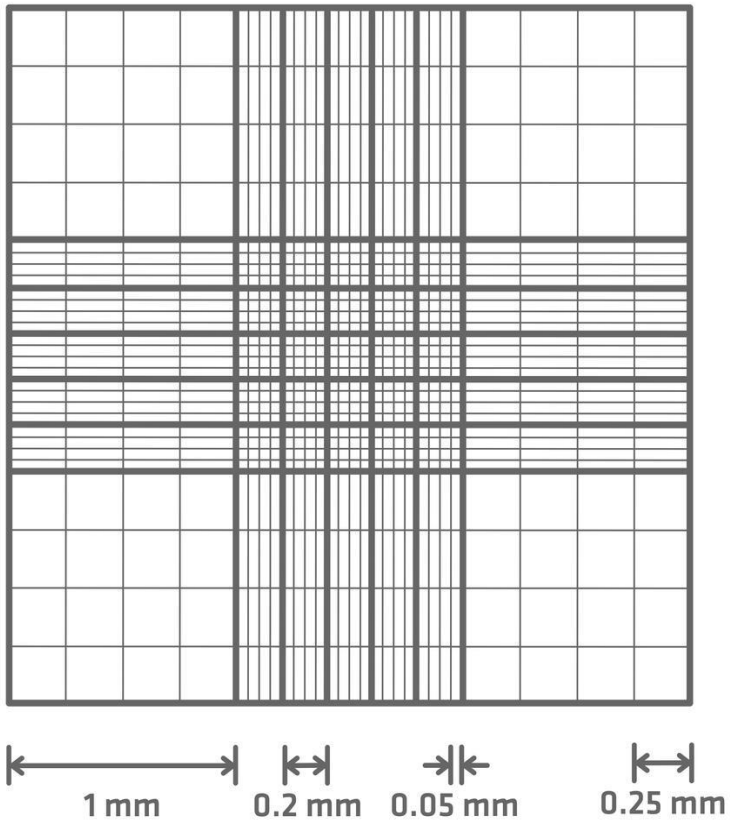


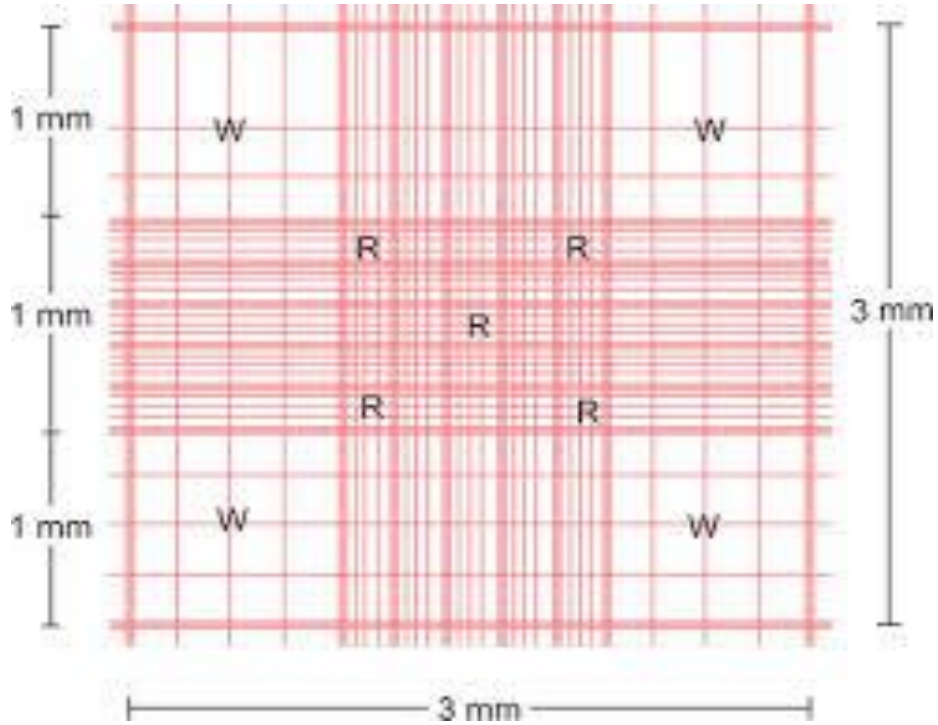
Haemocytometer - Improved neubauer's chamber



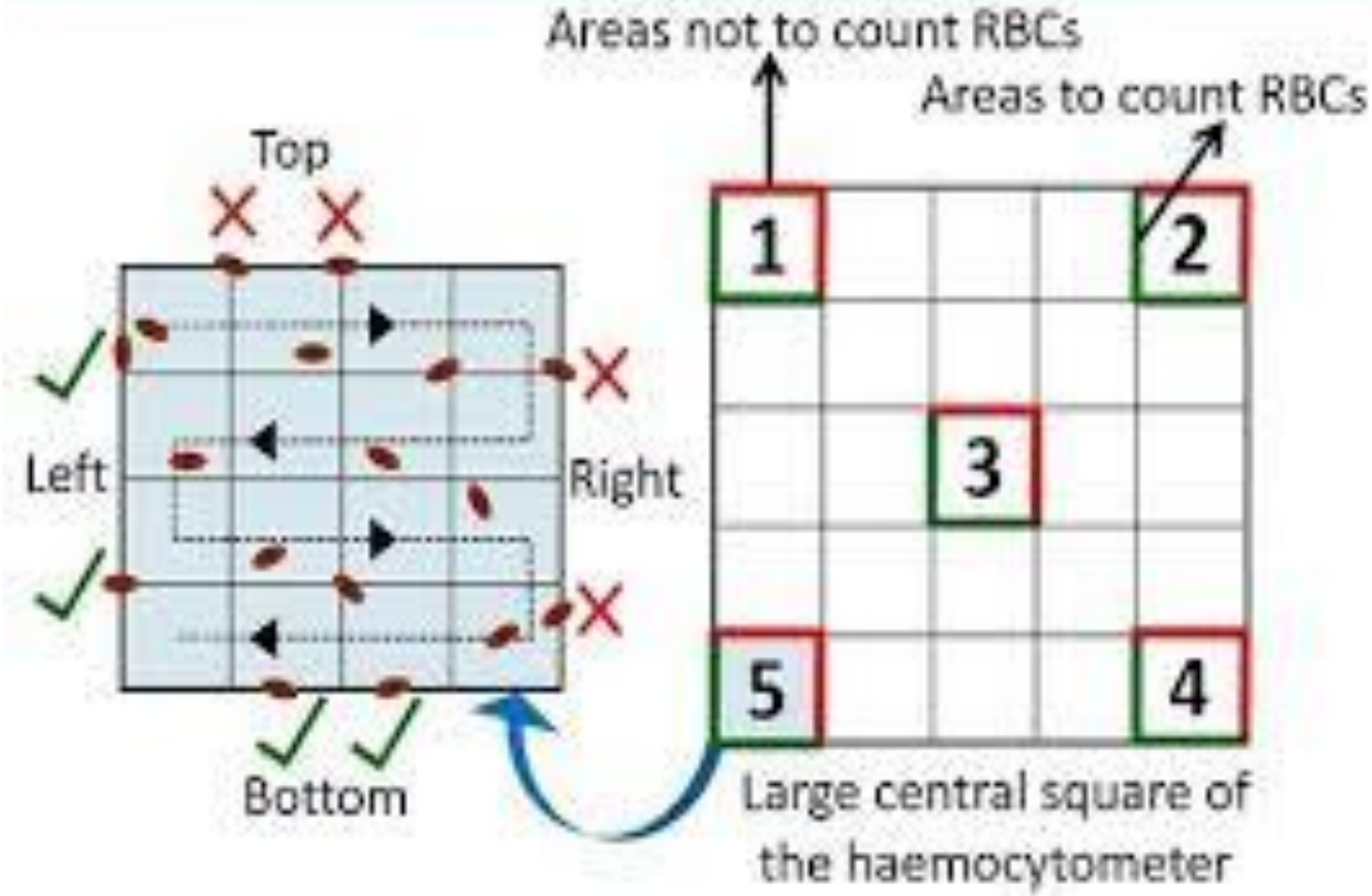
http://mbbsstudystuff.com/

Improved Neubauer chamber counting grid

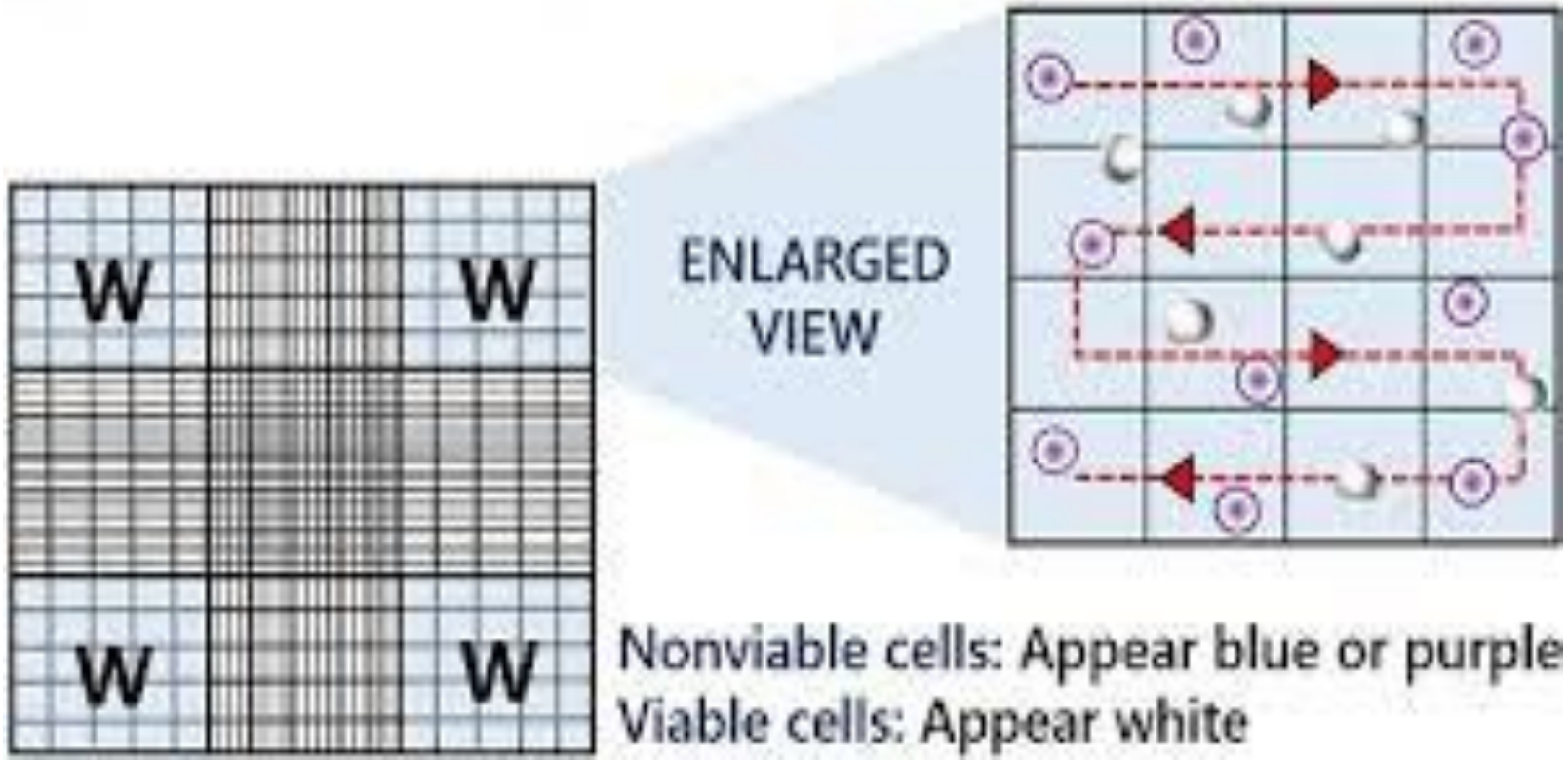




RBC Count Under Microscope

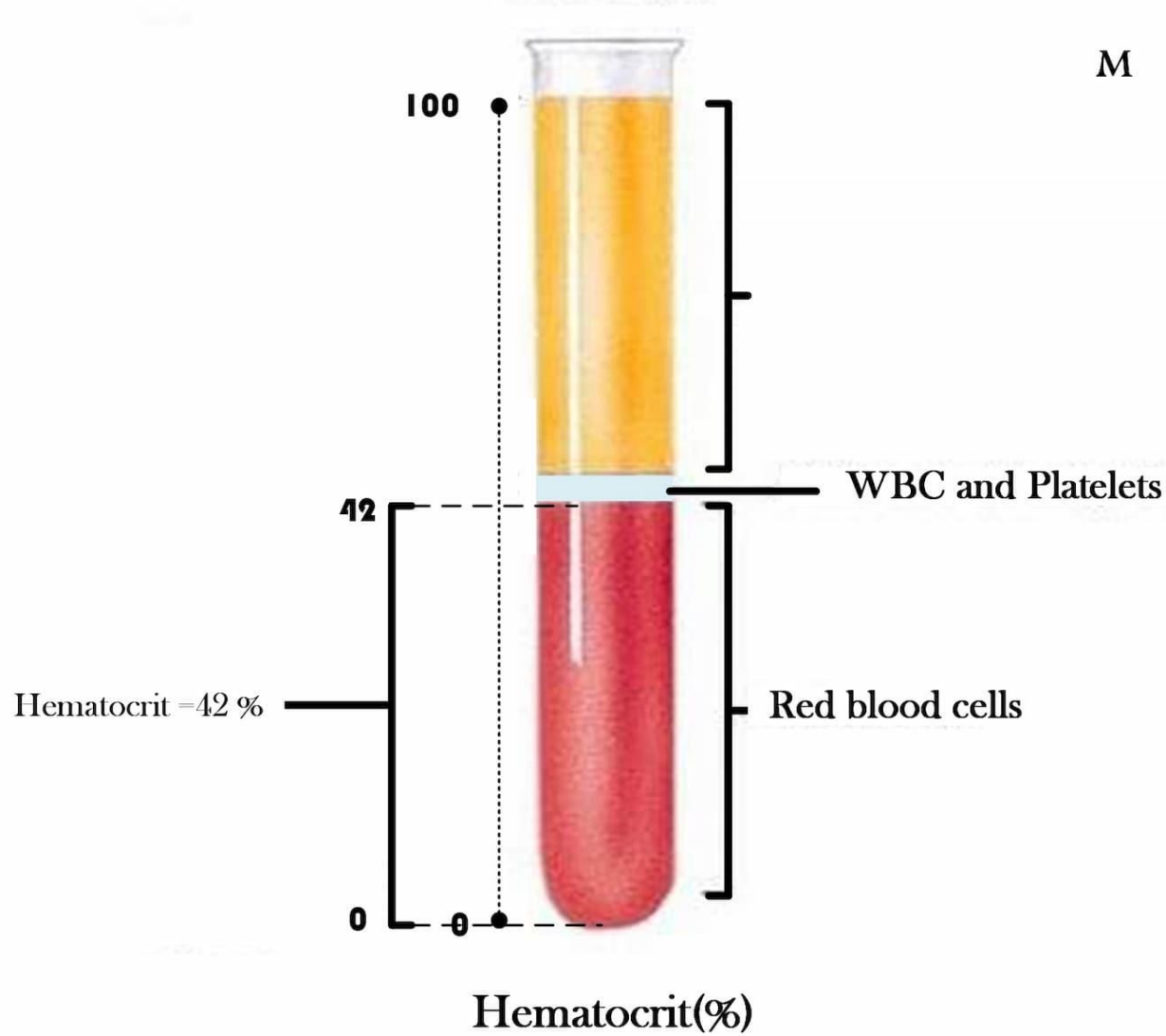


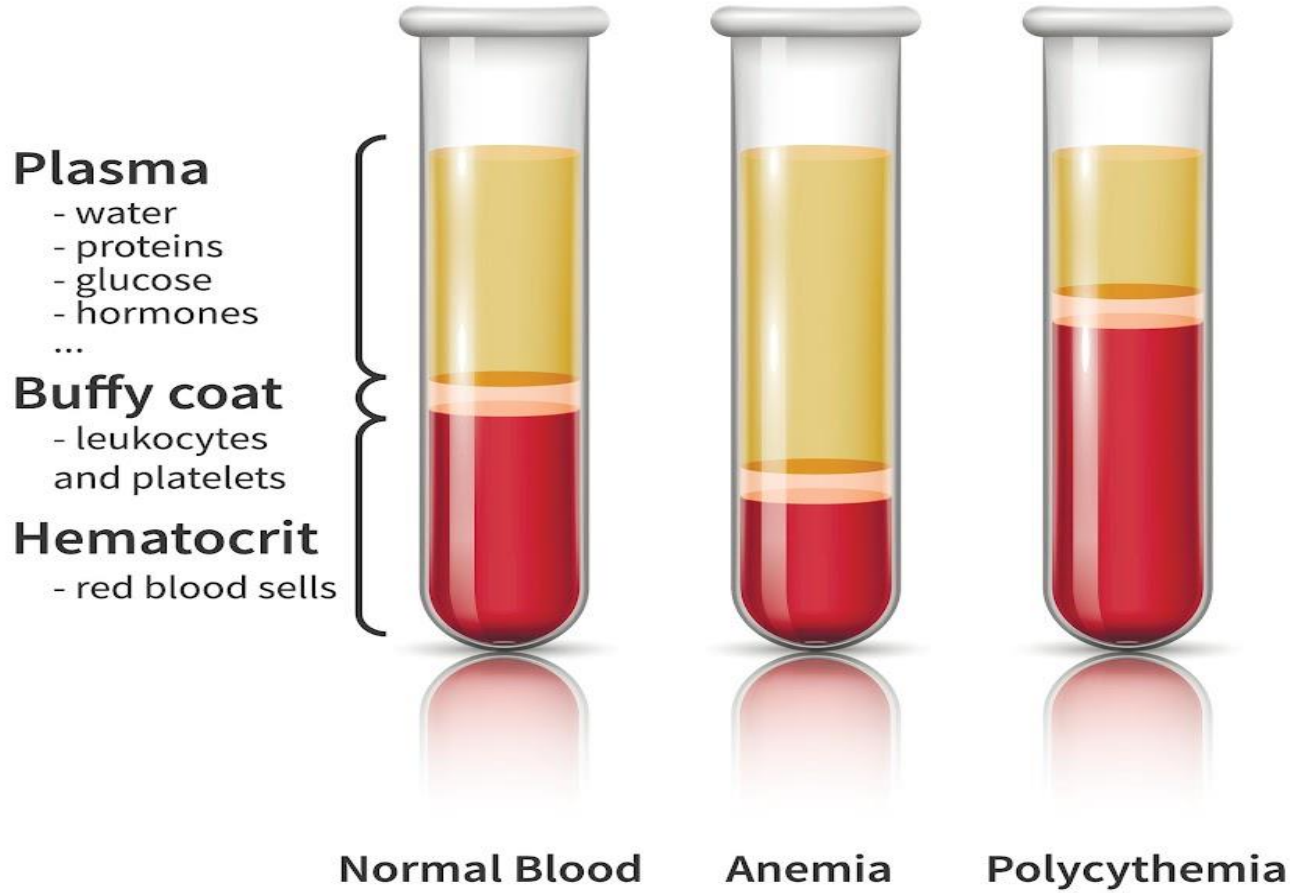
WBC Count Under Microscope



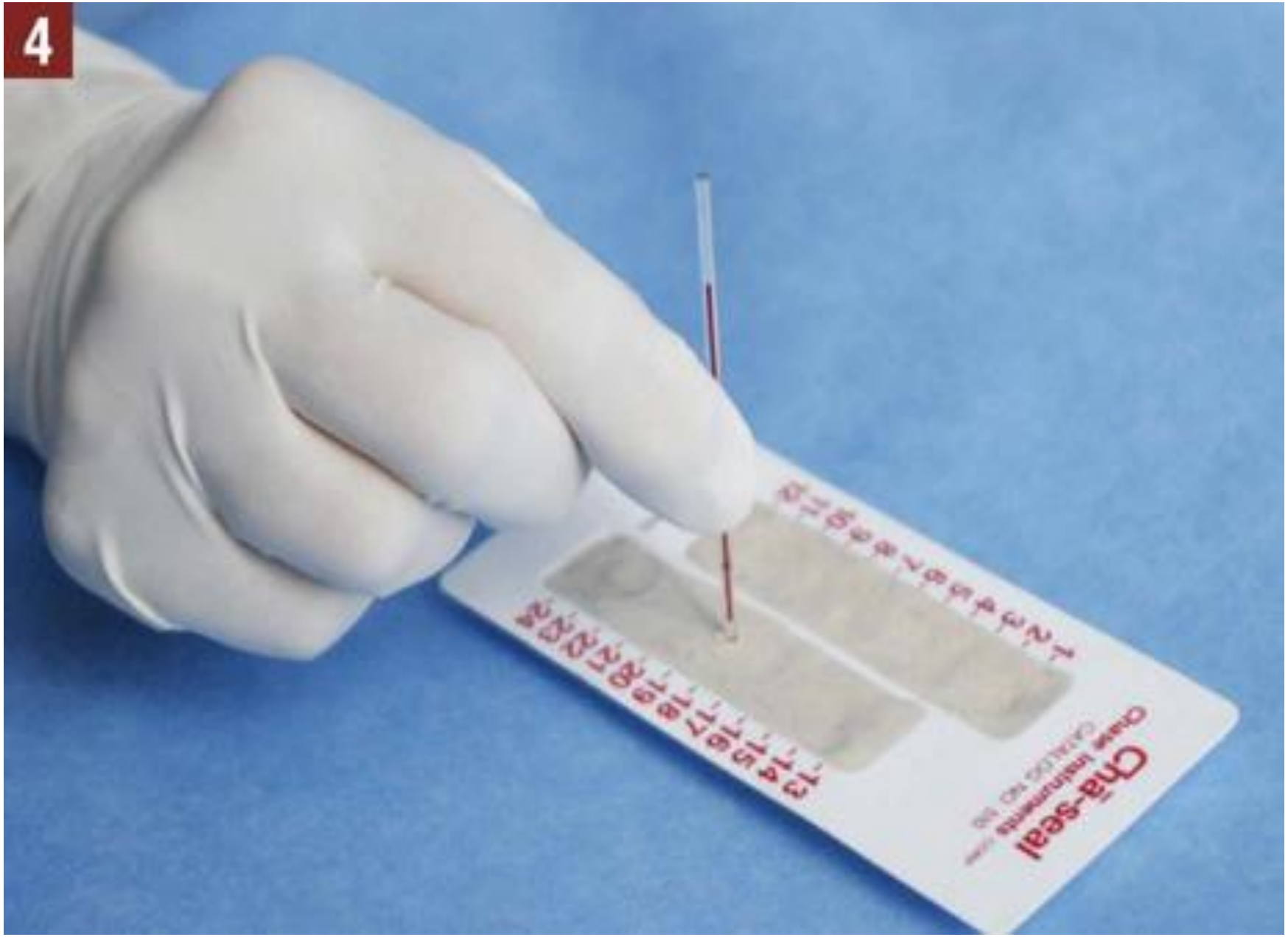
Nonviable cells: Appear blue or purple
Viable cells: Appear white


Hematocrit
smartlabs
NOW


M
100
42
0
0
WBC and Platelets
Red blood cells
Hematocrit =42 %
Hematocrit(%)


Plasma
- water
- proteins
- glucose
- hormones
...
Buffy coat
- leukocytes
and platelets
Hematocrit
- red blood sells
Normal Blood
Anemia
Polycythemia


100 tubes
Capillary
For Micro-
Sodium-
75mm
I.D.1.1-1.2
O.D 1.5-1.6
ISO9001
100 tubes
Capillary
For Micro-
Plain,Non-
75mm
I.D.1.1-1.2
O.D 1.5-1.6
ISO9001


4
Chase Instruments
CHASE-SEAL

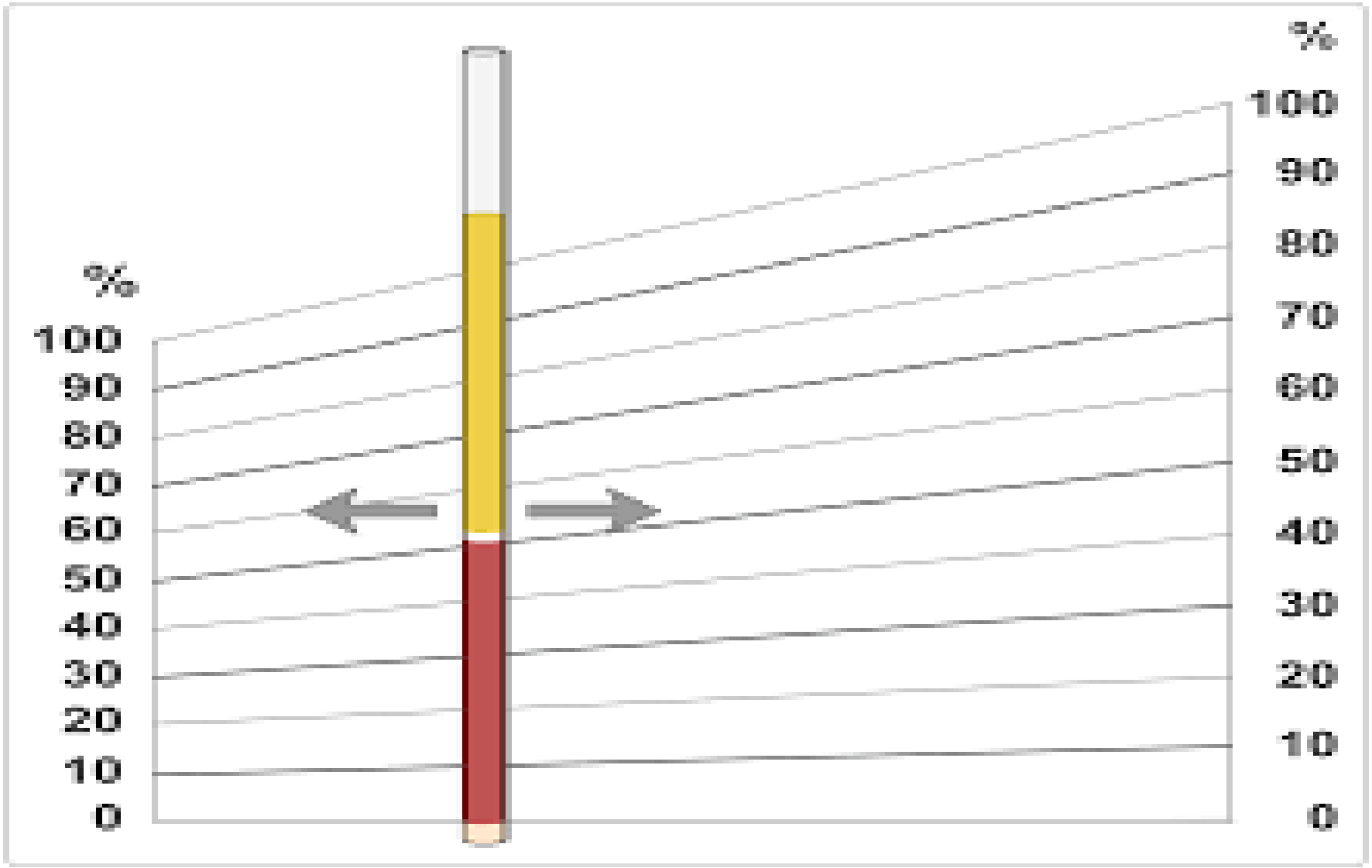

Item 410E
Hematocrit Centrifuge
BRAKE
TIME

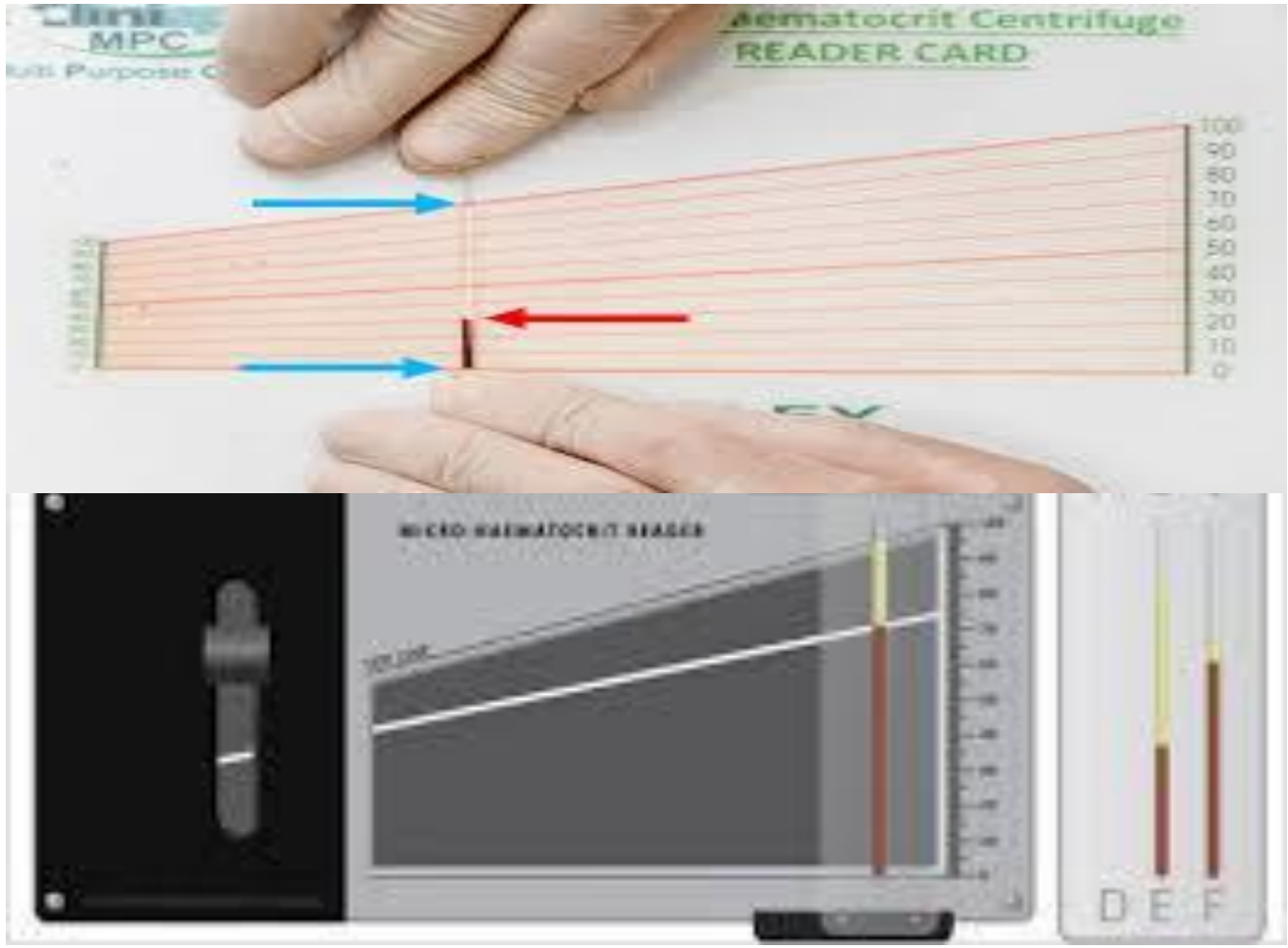

shutterstock.com · 659883262


shutterstock.com · 1335538127

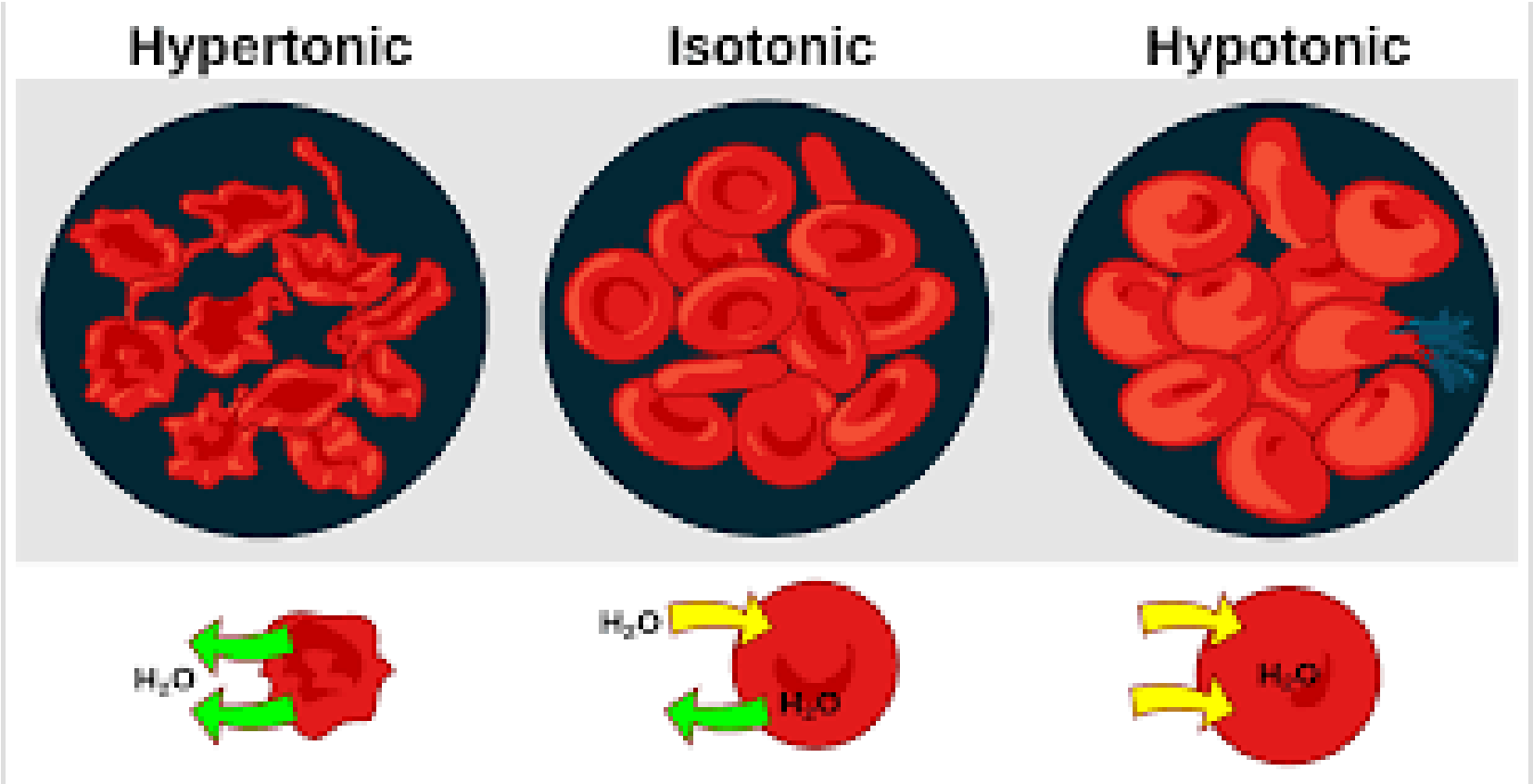
TNM SHOP

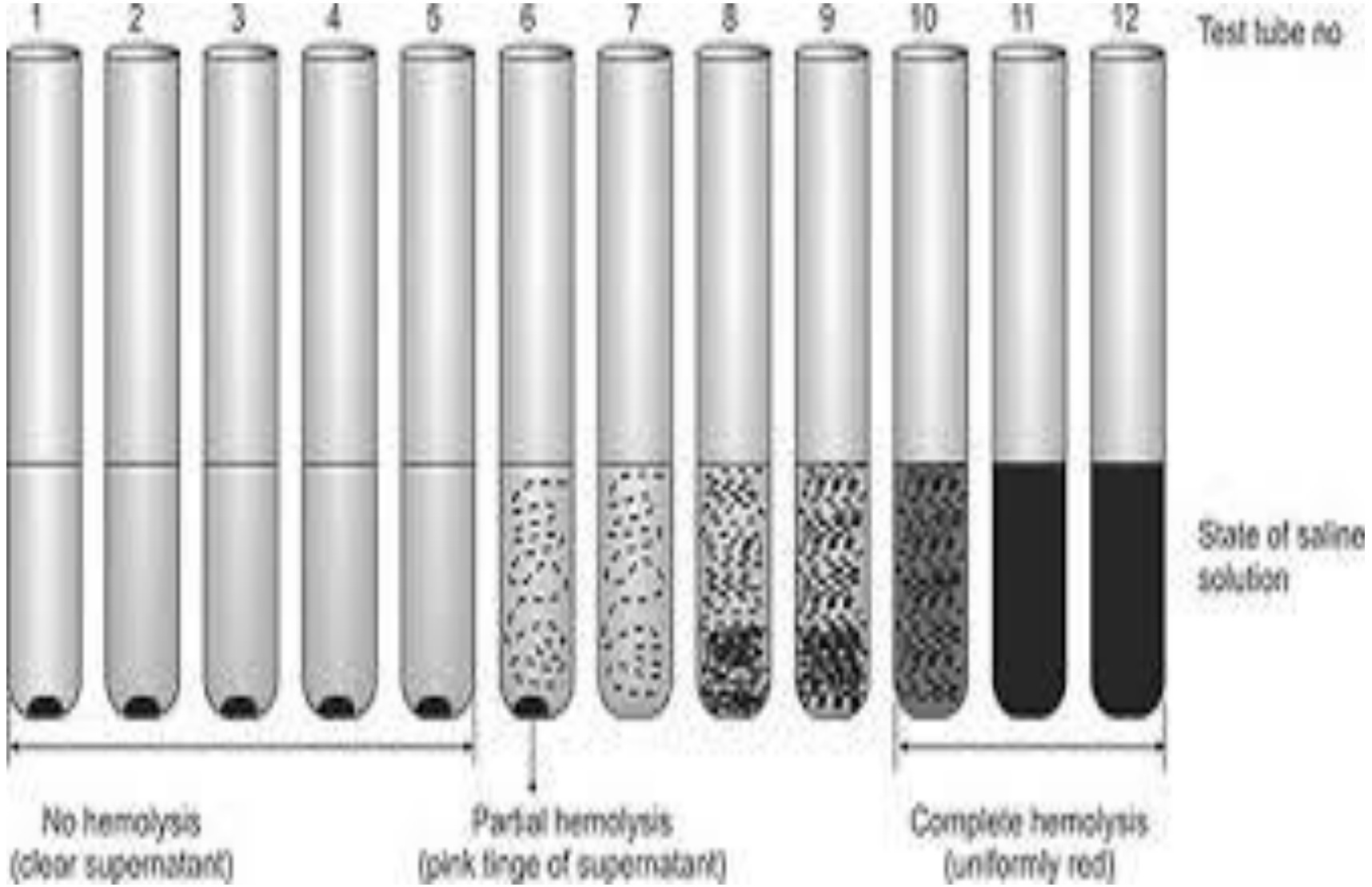
ส่งฟรี

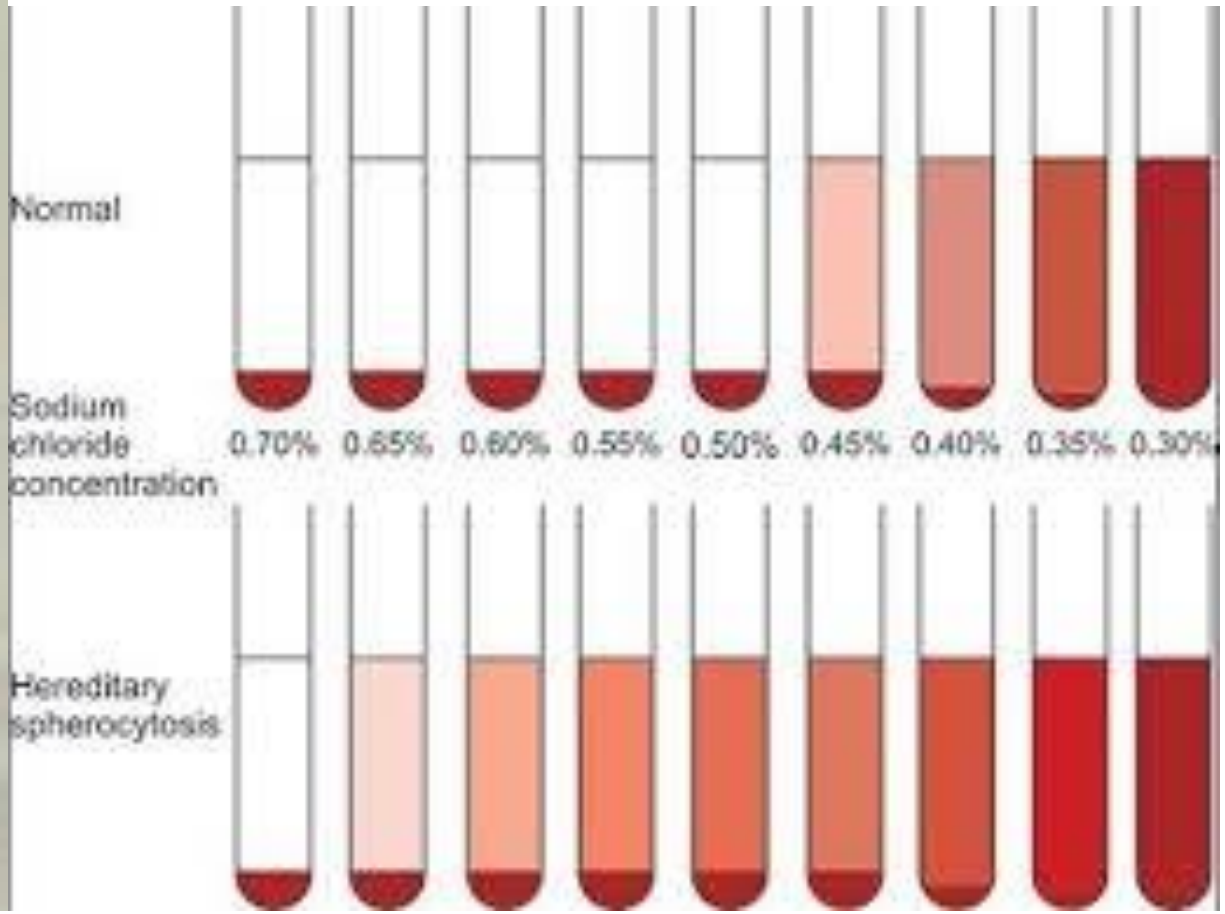

%
100
90
80
70
60
50
40
30
20
10
0
%
100
90
80
70
60
50
40
30
20
10
0


MPC
Hematocrit Centrifuge
READER CARD
100
90
80
70
60
50
40
30
20
10
0
D E F

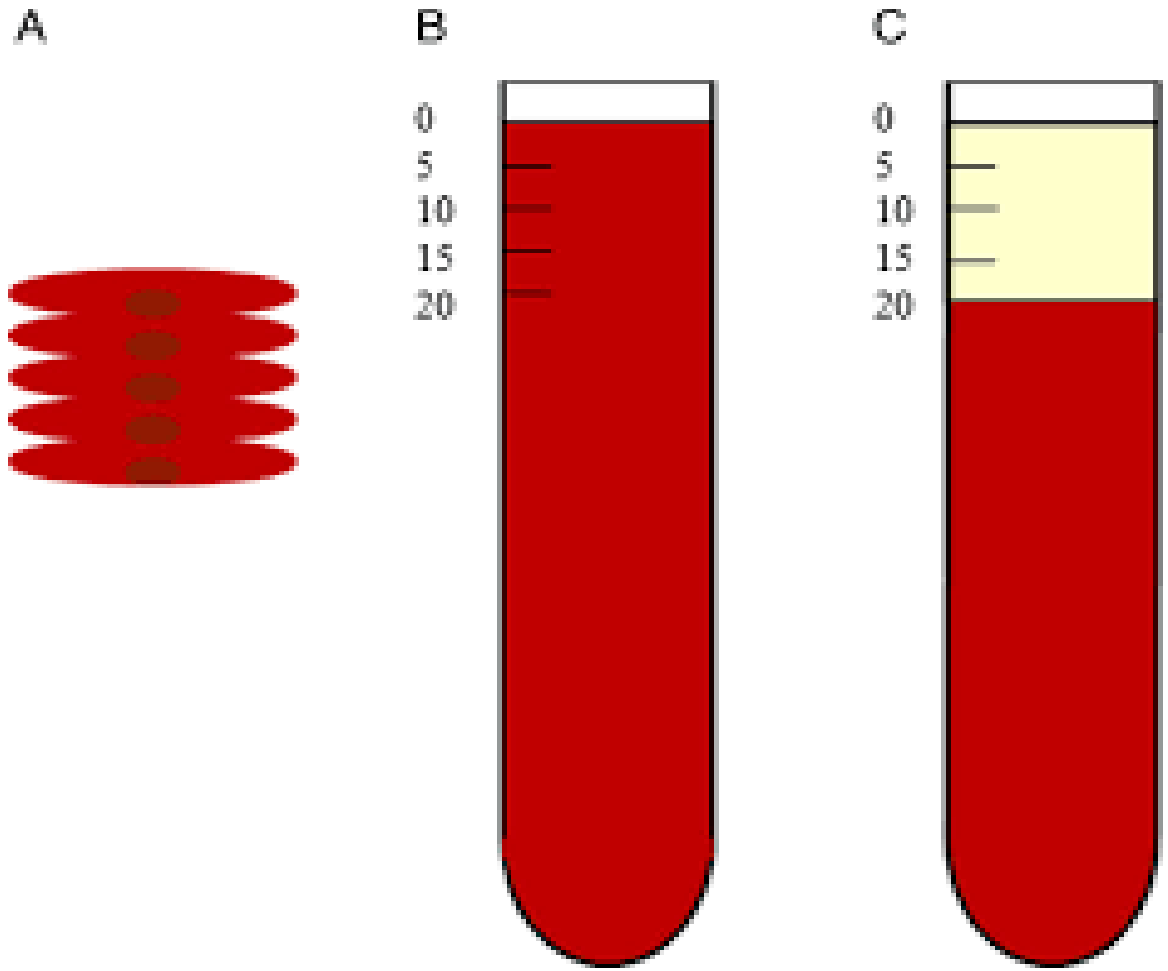
Erythrocyte Fragility(Osmotic Hemolysis)



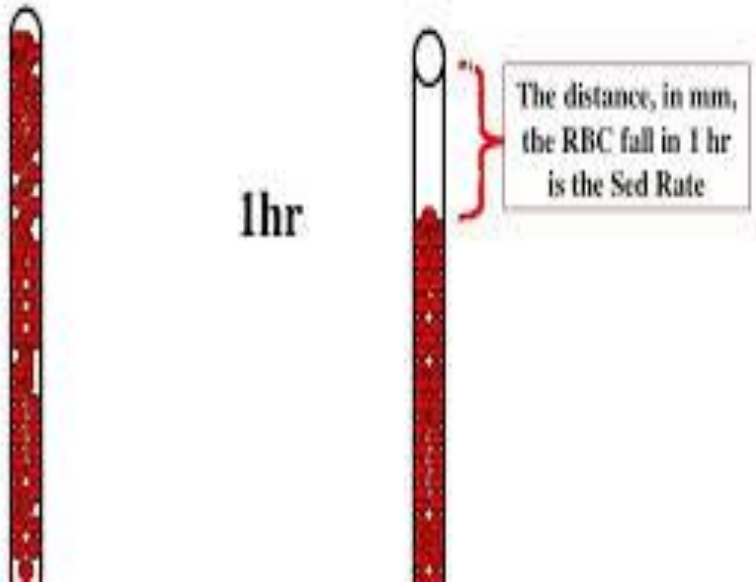

1
2
3
4
5
6
7
8
9
10
11
12
Test tube no
State of saline solution
No hemolysis
(clear supernatant)
Partial hemolysis
(pink tinge of supernatant)
Complete hemolysis
(uniformly red)

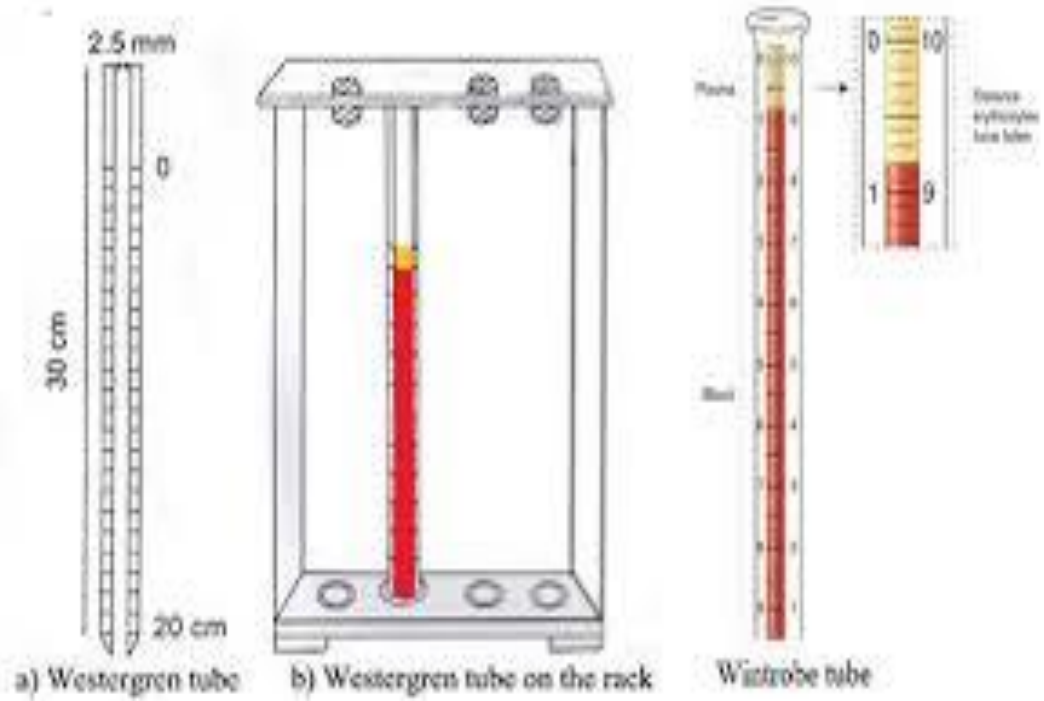

Normal
Sodium chloride concentration
0.70%
0.65%
0.60%
0.55%
0.50%
0.45%
0.40%
0.35%
0.30%
Hereditary spherocytosis

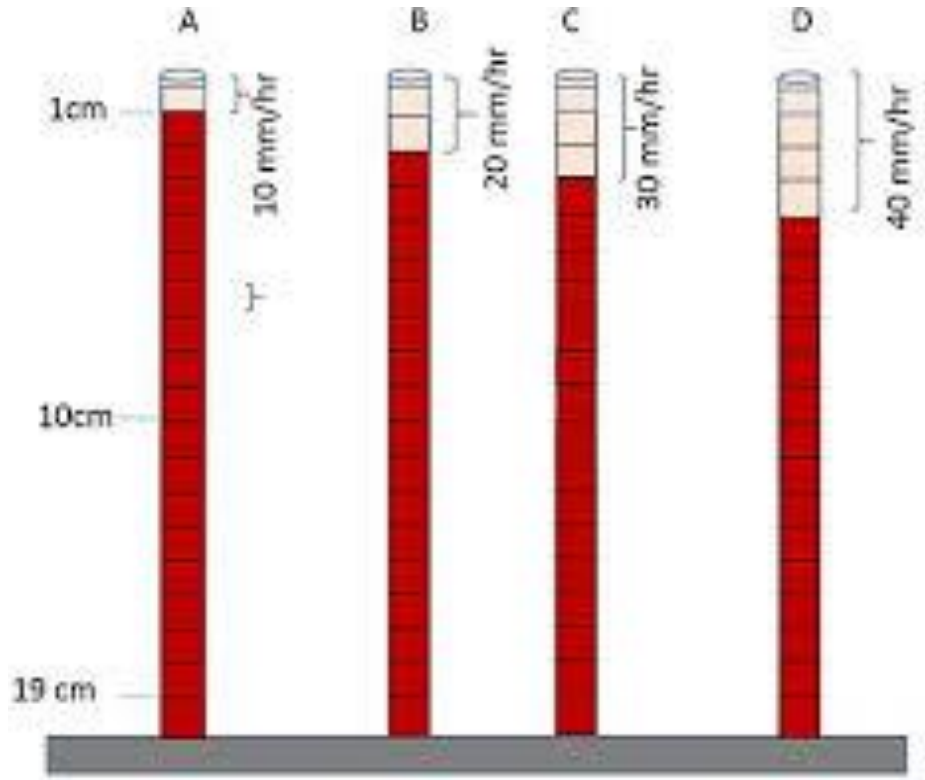
Erythrocyte Sedimentation Rate(ESR)



Erythrocyte Sedimentation Rate (ESR)







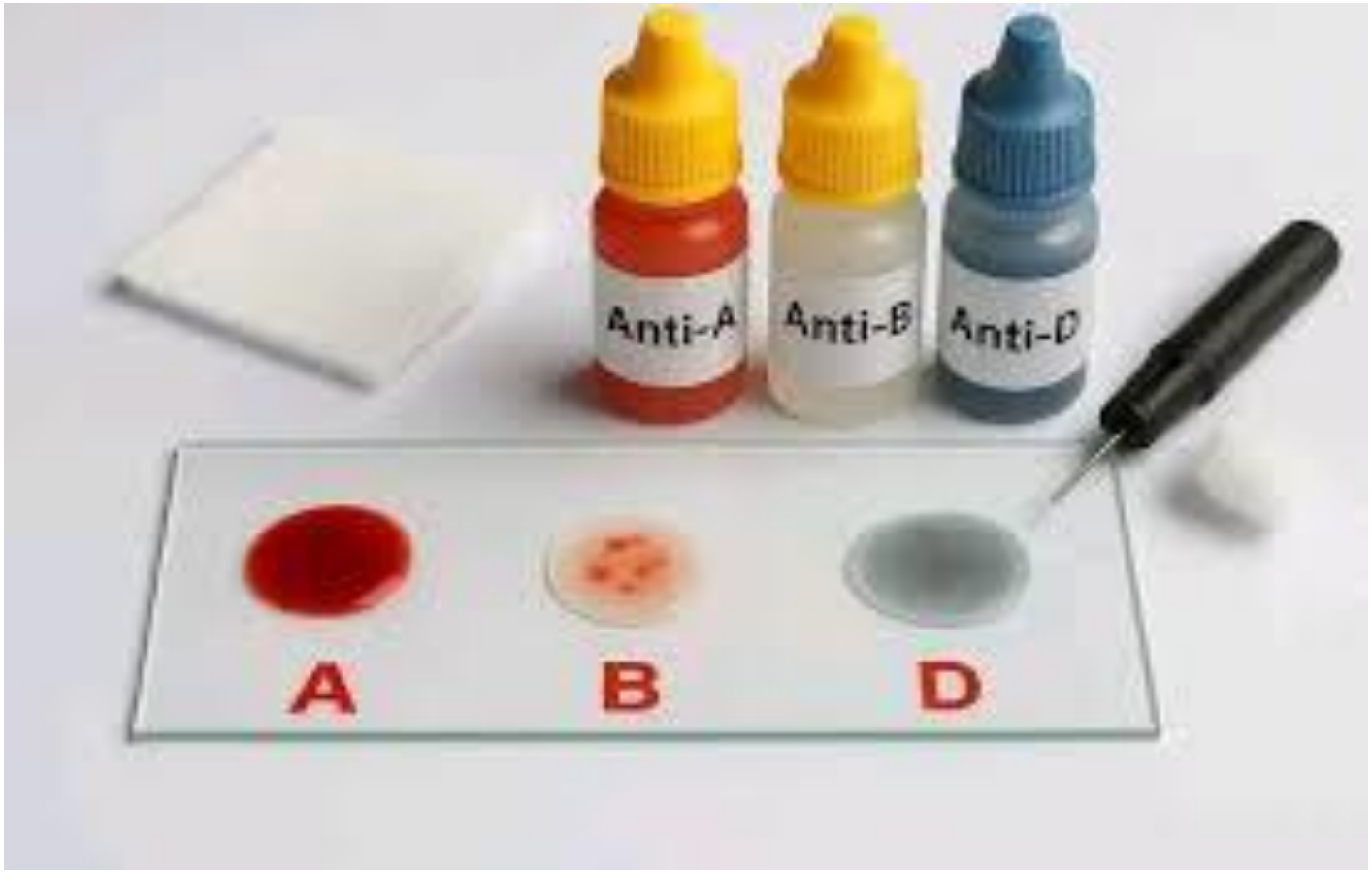
Erythrocyte sedimentation rate

Blood Typing Test

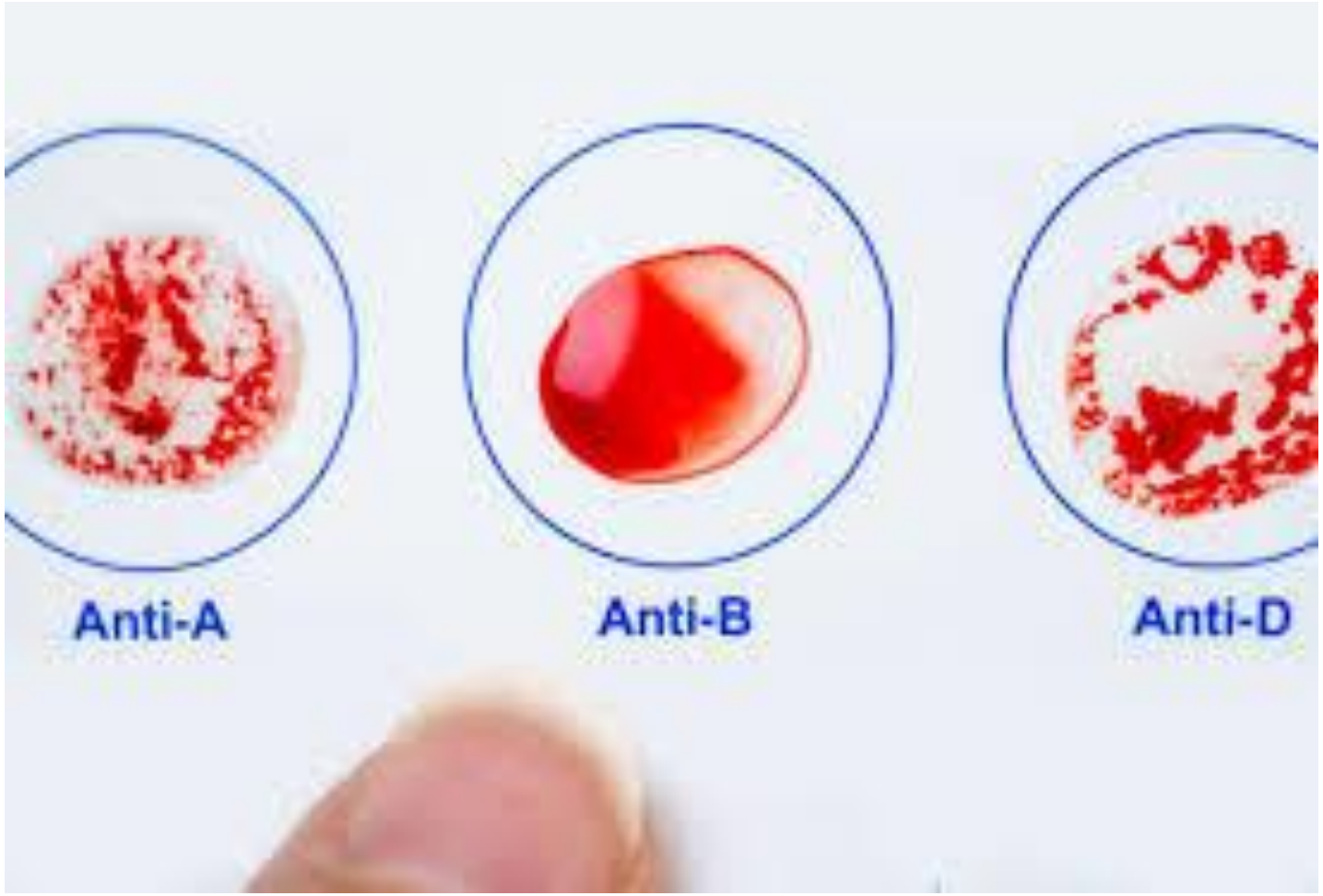
Table 36-1 Blood Types With Their Genotypes and Constituent Agglutinogens and Agglutinins

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

	Sera	
Red Blood Cell Types	**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* A_2.

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

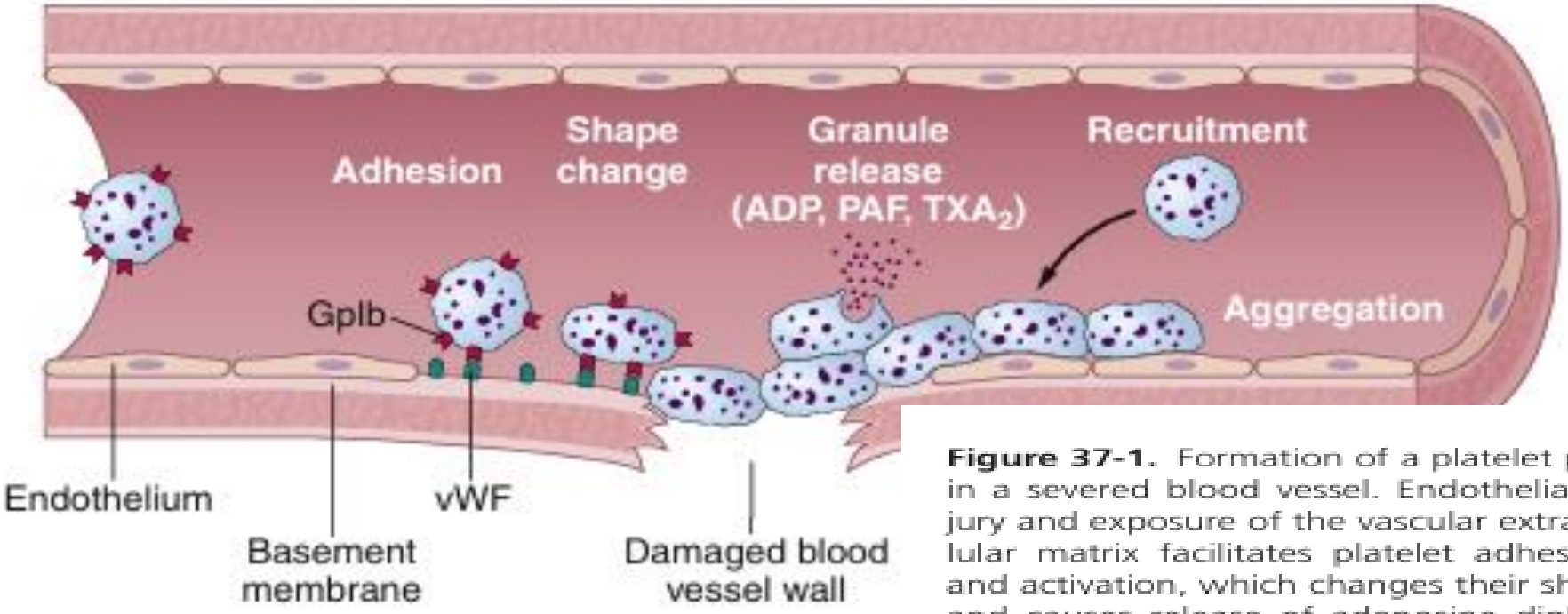


Figure 37-1. Formation of a platelet plug in a severed blood vessel. Endothelial injury and exposure of the vascular extracellular matrix facilitates platelet adhesions and activation, which changes their shape and causes release of adenosine diphosphate (ADP), thromboxane A_2 (T_XA_2), and platelet-activating 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 subendothelial 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

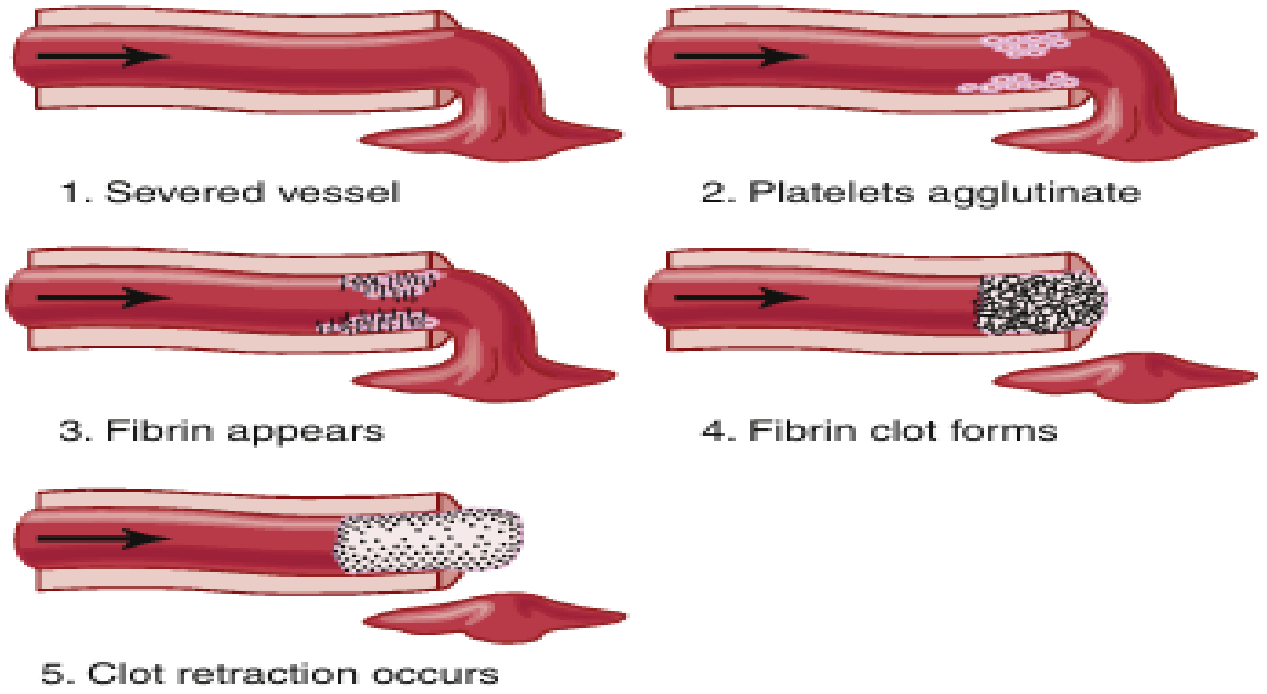
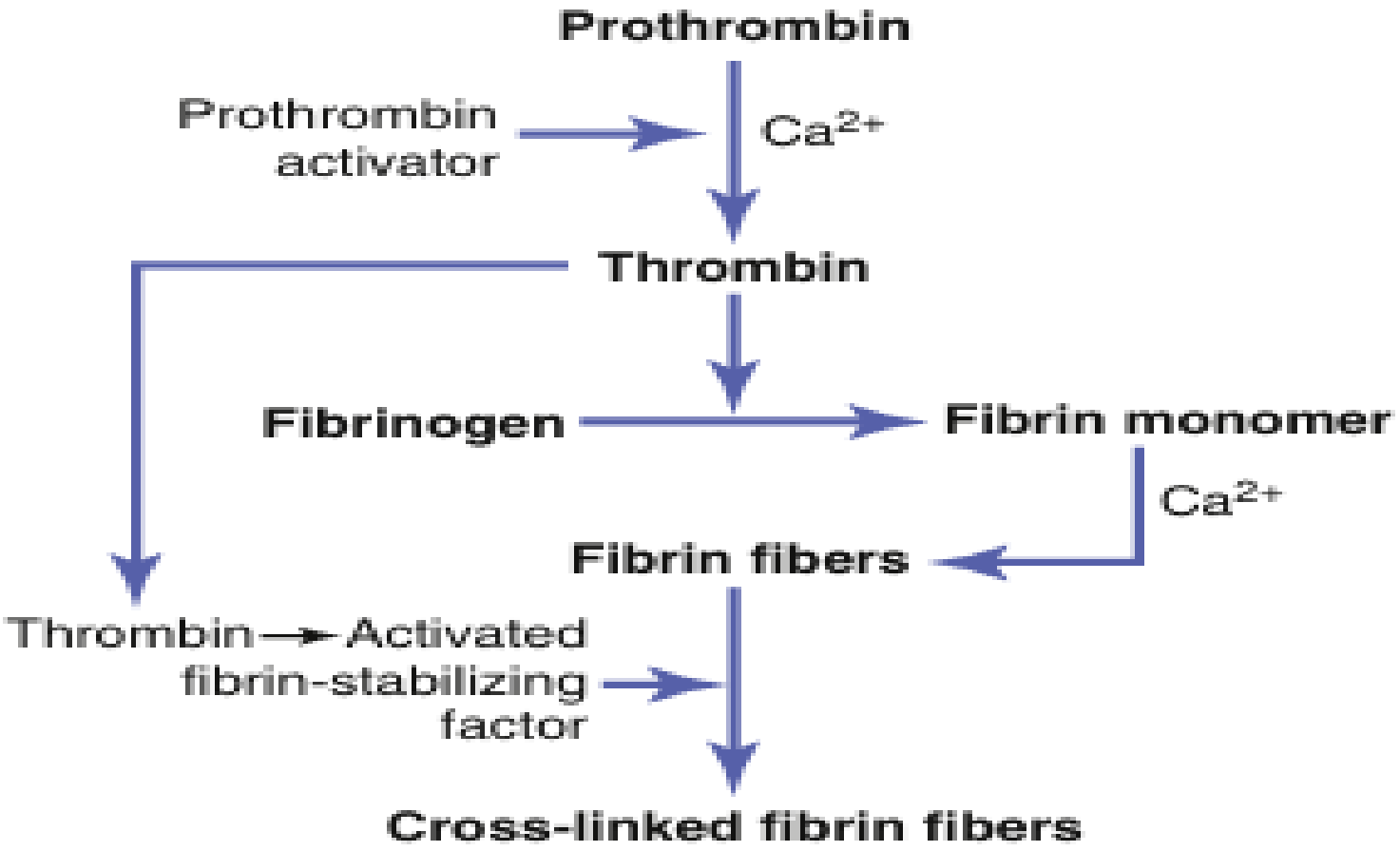


Figure 37-2. Clotting process in a traumatized blood vessel. *(Modified from Seegers WH: Hemostatic Agents. Springfield, IL: Charles C Thomas, 1948.)*

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

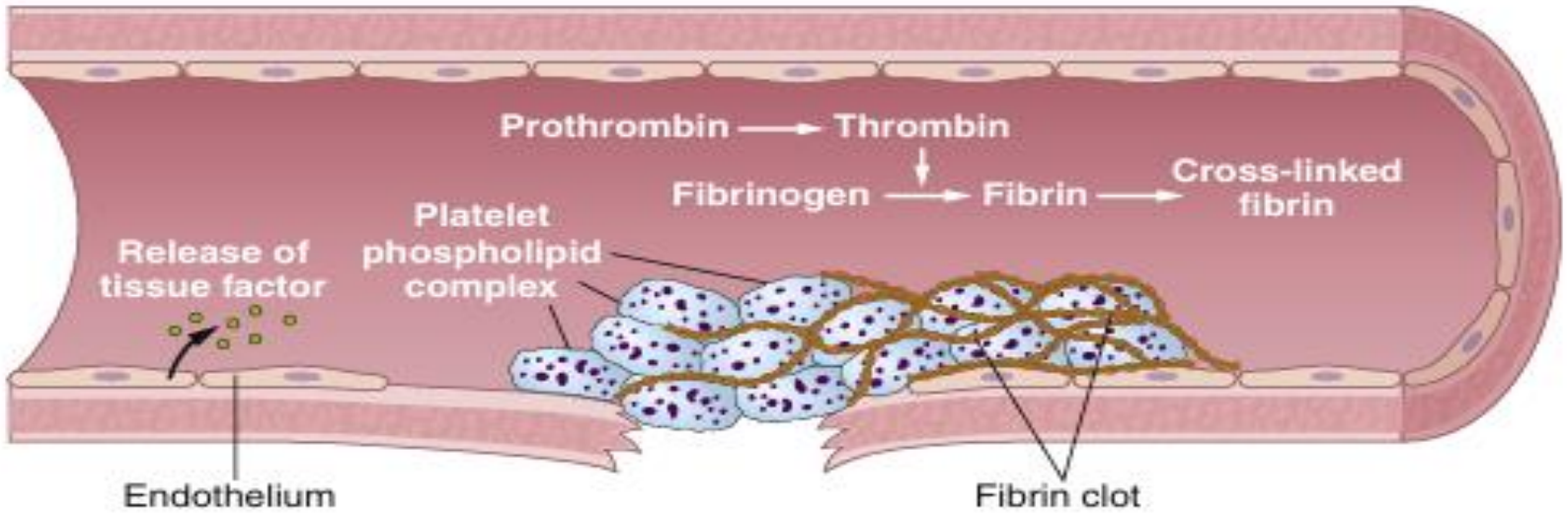


Figure 37-4. Coagulation cascade after vascular injury. Exposure of blood to the vascular wall causes release of tissue factor (also called factor III or thromboplastin) from endothelial cells, phospholipid expression, activation of thrombin, which then acts on fibrinogen to form fibrin, and fibrin polymerization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

Hemostasis & Blood coagulation

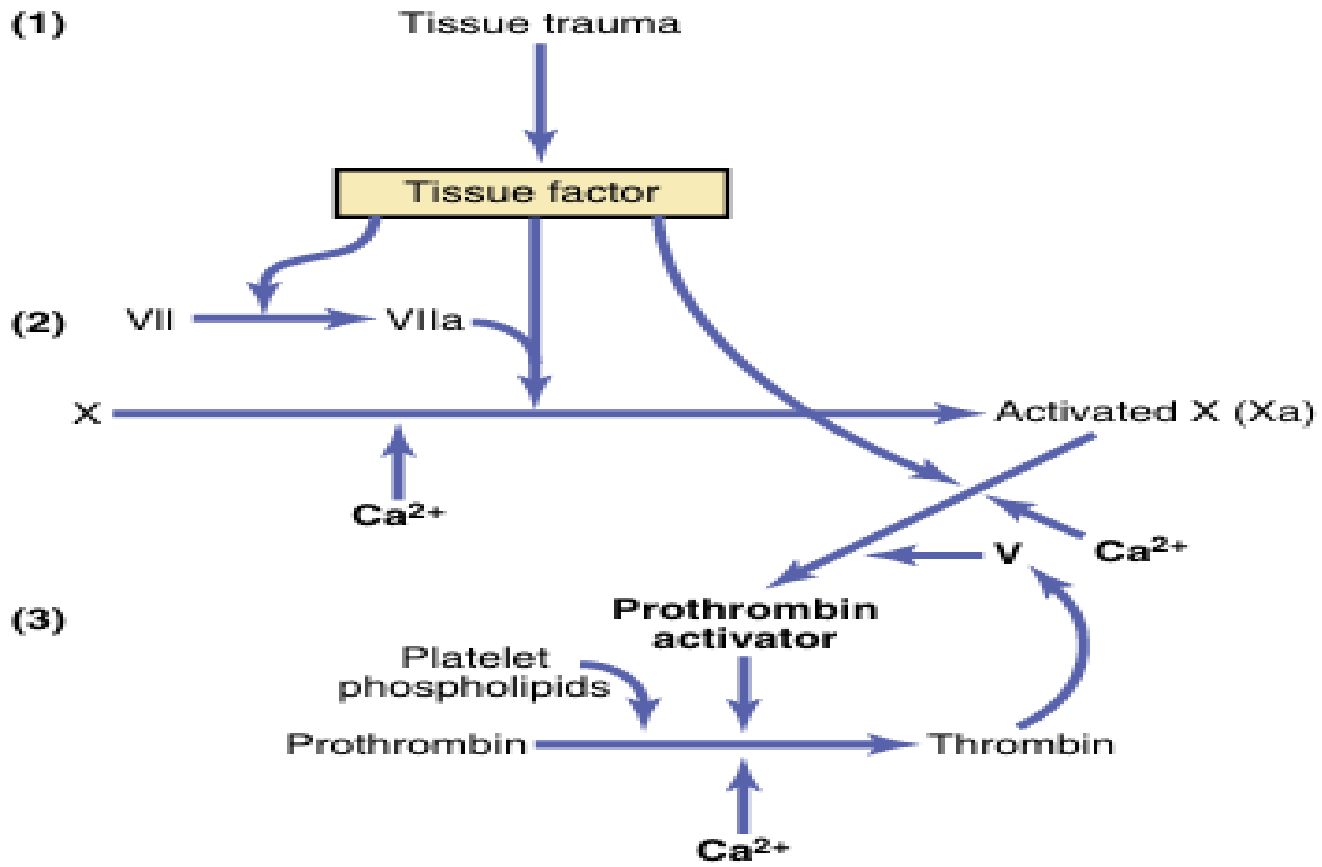


Figure 37-5. Extrinsic pathway for initiating blood clotting.

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

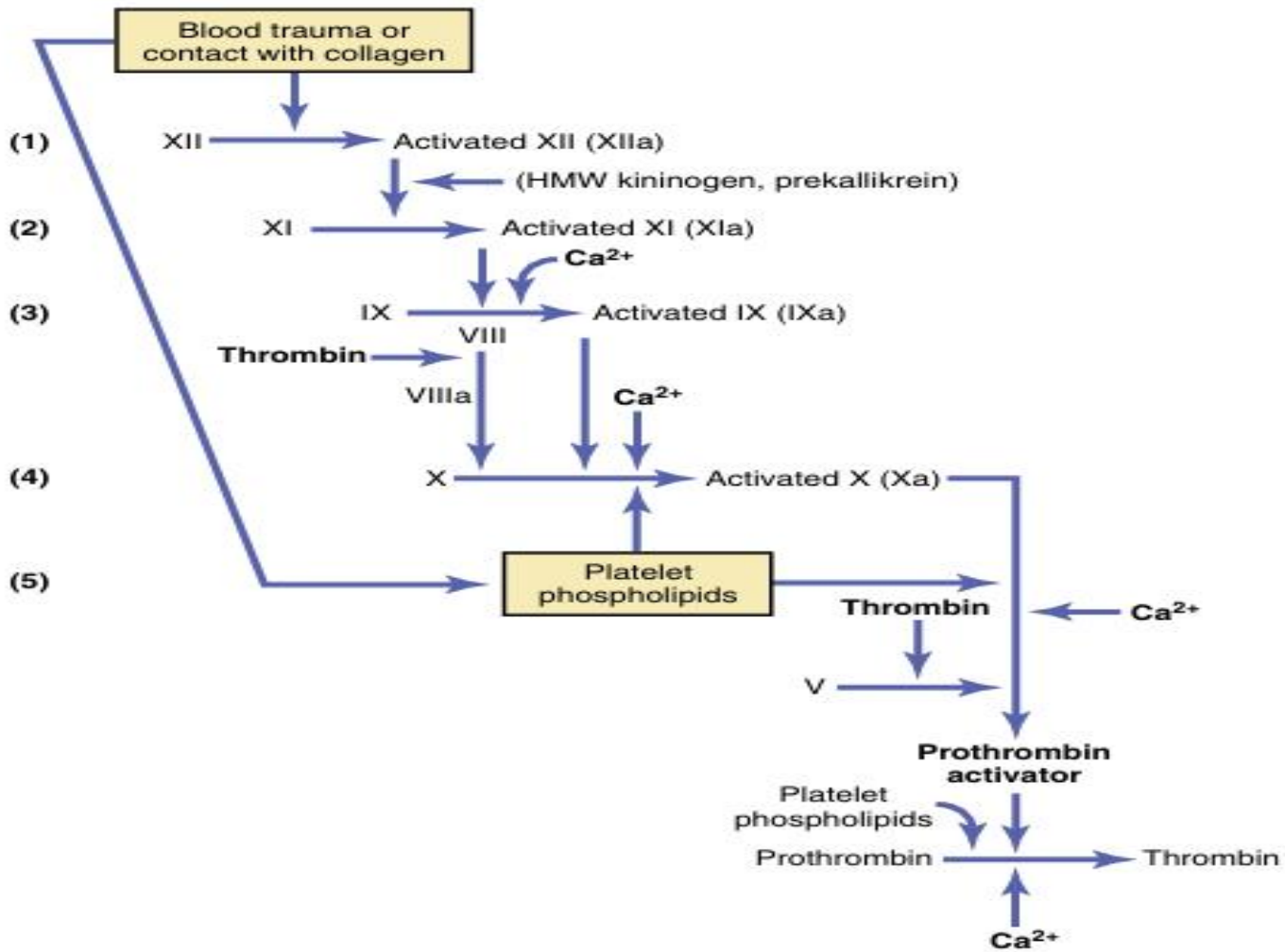


Figure 37-6. Intrinsic pathway for initiating blood clotting. HMW, High-molecular weight.