

BLOOD GROUPS AND BLOOD TRANSFUSION

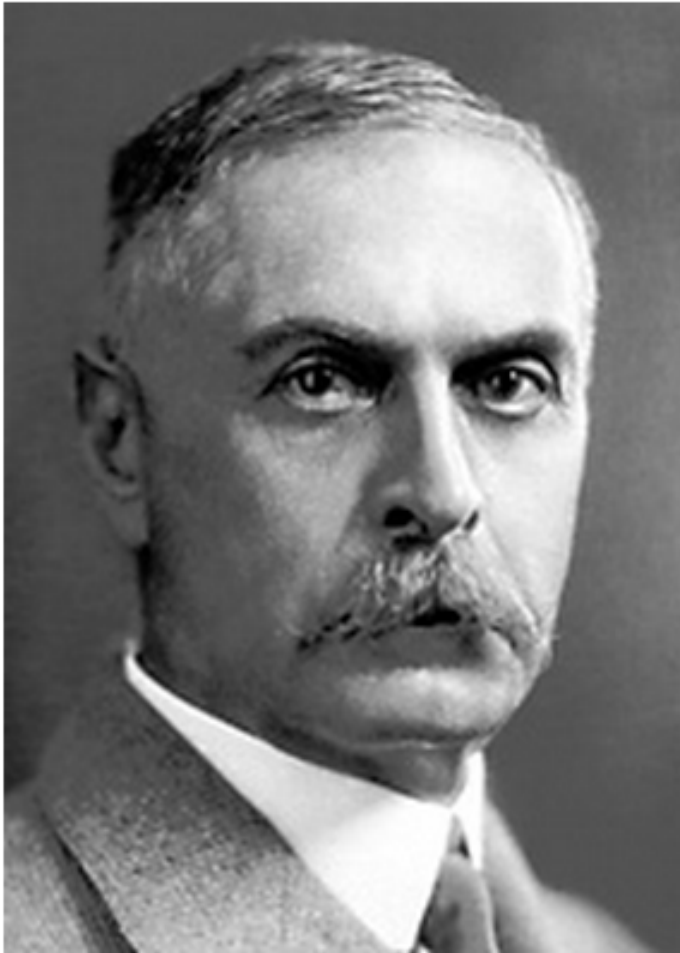
Prof. Shahid Habib

OBJECTIVES

At the end of this lecture you should be able to:

- 1. Describe ABO blood groups types.**
- 2. Recognize Agglutinin in plasma.**
- 3. Describe genetic inheritance of Blood groups.**
- 4. Recognize transfusion reactions.**
- 5. Describe Rhesus blood groups.**
- 6. Describe causes of hemolytic disease of the newborn.**

BLOOD GROUPS



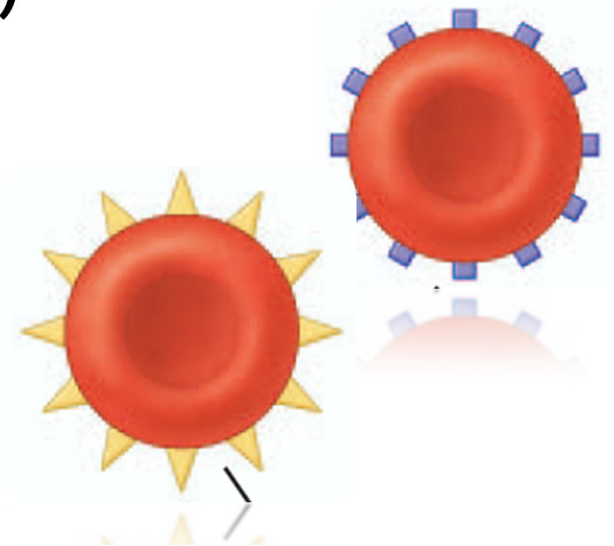
KARL LANDSTEINER
(1886-1943)

- Discovered ABO Blood group system in 1901
- Discovered Rh factor in 1930 along with Alexander S. Wiener
- Noble prize in Physiology or Medicine in 1930

Austrian scientist

BLOOD GROUP SYSTEMS

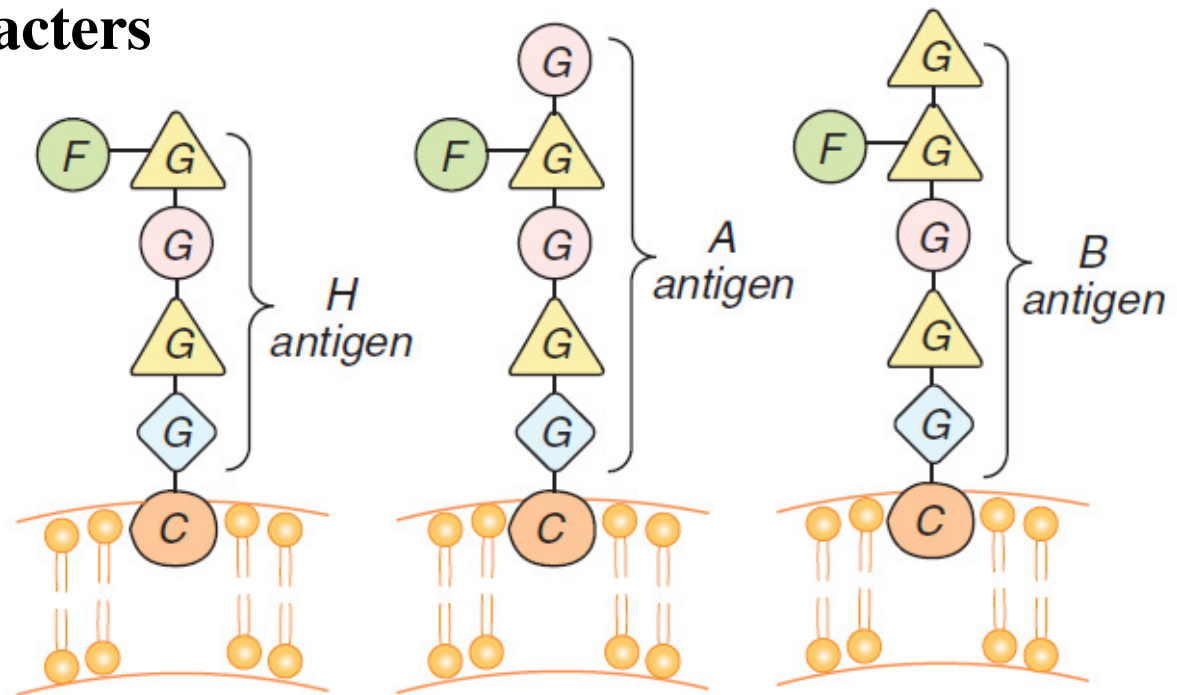
- **Blood Groups are determined by: Antigens** (glycoproteins, complex oligosaccharides that differ in their terminal sugar) **present on the surface of RBCs**
- About **20 blood group systems** are known (Eg. ABO System, Rh-System MNS System, Kell System, Lewis System, Duffy, Lutheran, KIDD)
- **Two are common**
 - ABO blood group system
 - Rh (Rhesus) blood group system



Are inherited characters

An H gene codes for a fucose transferase that adds a terminal fucose, forming the **H antigen** that is the foundation upon all blood types are formed

A and B genes code for enzymes that add a sugar to the H antigen



(F) = fucose

(G) = N-acetylgalactosamine

(G) = glucose

(G) = galactose

(C) = ceramide

(lipid bilayer) = lipid bilayer

FIGURE 31-9 Antigens of the ABO system on the surface of red blood cells.

The ABO system:

- Depends on whether the red cells contain one, both or neither of the two blood antigens: A and B
- Four main ABO groups: **A, B, AB, O**

Locus of alleles responsible of ABO system is on long arm of chromosome 9 while Rh locus is on chromosome 1

Rhesus (Rh) Blood Group is Determined by:

- Presence or absence of the Rhesus antigen (D) on the surface of RBC:
 - –Presence of D (individual is Rh+ve) [85%]
 - –Absence of D (individual is Rh–ve) [15%]
- Rhesus antigens:
 - Dd, Cc, Ee
 - Clinically most important is D



Landsteiner and Weiner in 1940 discovered other antigens in human R.B.Cs and named rhesus antigens or Rh-antigen because the same antigens are present in Rhesus monkey.

Antibodies in ABO & Rh Systems

- Anti-A & Anti-B are: **naturally** occurring antibodies.
 - Not present at birth, appear 8 weeks after birth.
 - Triggered by A & B antigens in food and bacteria.
-
- Anti-D antibody (agglutinin):
 - Is not naturally-occurring and are **Acquired** by:
 - Transfusion of Rh-ve individual with Rh+ve blood.
 - Rh-ve pregnancy with Rh+ve faetus.

Landsteiner Law (1900) If an agglutinogen is present on the RBC of an individual, the corresponding agglutinin must be absent in the plasma of that individual and vice-versa. This law is only applicable to ABO blood grouping system.

TABLE 31-3 Summary of ABO system.

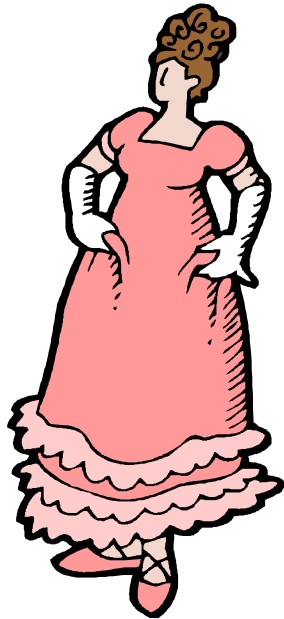
Blood Type	Agglutinins in Plasma	Frequency in United States %	Plasma Agglutinates Red Cells of Type:
O	Anti-A, anti-B	45	A, B, AB
A	Anti-B	41	B, AB
B	Anti-A	10	A, AB
AB	None	4	None

Antibodies in Serum are also known as AGGLUTININS

Inheritance of ABO system

- The **ABO gene** locus is located on the **chromosome 9**
- **A** and **B** blood groups are **dominant** over the **O** blood group
- **A** and **B** group genes are **co-dominant**
- Each person has **two copies of genes** coding for their ABO blood group (one maternal and one paternal in origin)

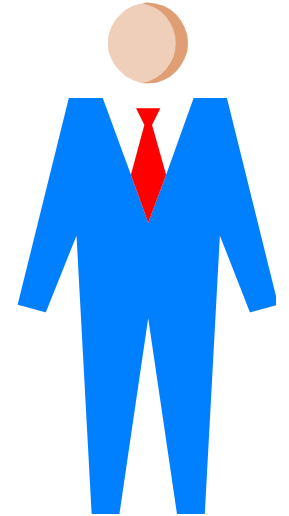
AUTOSOMAL CHROMOSOME



MOTHER



The alleles for Blood group are in the same place on the chromosome 9. However the genes have a different code giving the different blood group



FATHER

one alleles from Mother and one from Father.

GENETIC DETERMINATION OF AGGLUTINOGENS

Parent Allele	A	B	O
A	AA	AB	AO
B	AB	BB	BO
O	AO	BO	OO

GENETIC DETERMINATION OF AGGLUTINOGENS

Blood Group	Antigens on RBCs	Antibodies in Serum	Genotypes
A	A	Anti-B	AA or AO
B	B	Anti-A	BB or BO
AB	A and B	Neither	AB
O	Neither	Anti-A and Anti-B	OO

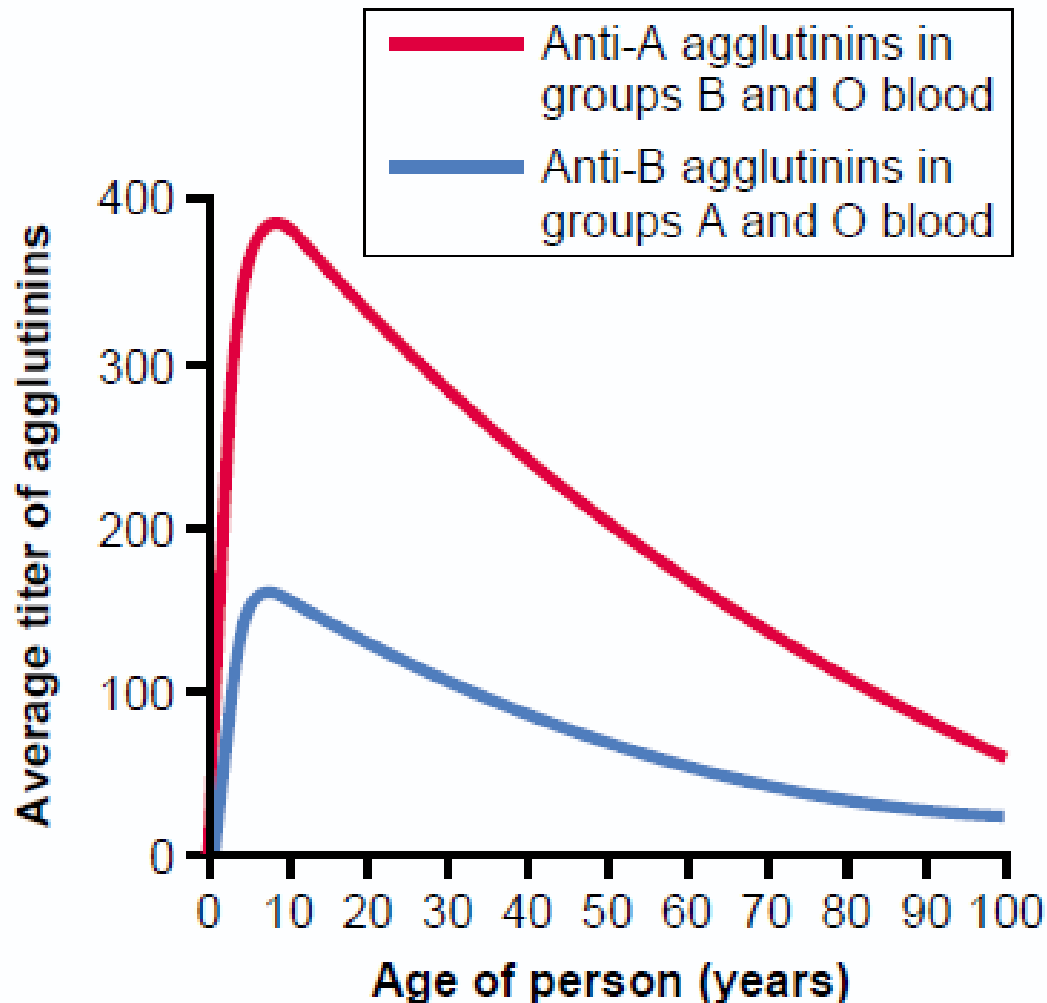
Antibodies in Serum are also known as AGGLUTININS

AGGLUTININS / ANTIBODIES

- Immunoglobulins Are proteins which appear in the plasma or body fluids in response to administration of antigens
 - IgM & IgG

anti-A or anti-B antibodies are of the IgM class (large molecules) and these do not cross the placenta
Rh antibodies are IgG type & can cross placenta

TITER OF ANTI-A& B AGGLUTININS AT DIFFERENT AGES



Two to 8 months after birth, an infant begins to produce agglutinins.

A maximum titer is usually reached at 8 to 10 years of age, and this gradually declines throughout the remaining years of life.

THE Rh IMMUNE RESPONSE

- **Formation of anti-Rh agglutinins**

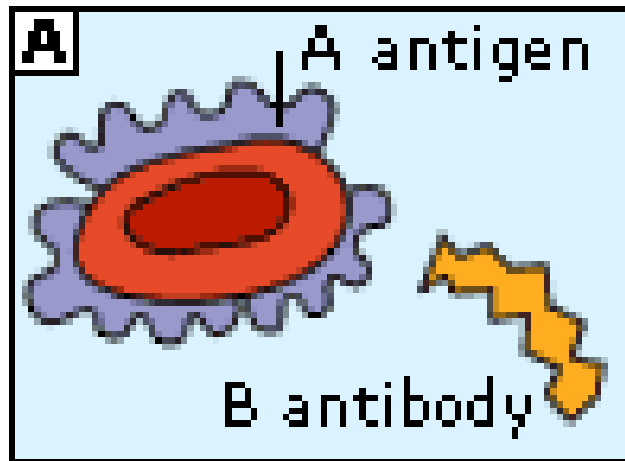
If Rh +ve blood transfused to



Rh -ve person

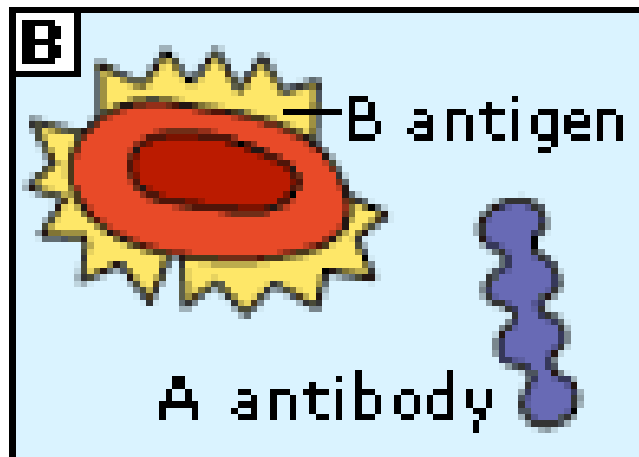


Anti-Rh agglutinins develop slowly (2-4 months)
Once produced they persist for years & can produce
serious reactions during 2nd transfusion



Blood group A

If you belong to the blood group A, you have A antigens on the surface of your RBCs and B antibodies in your blood plasma.



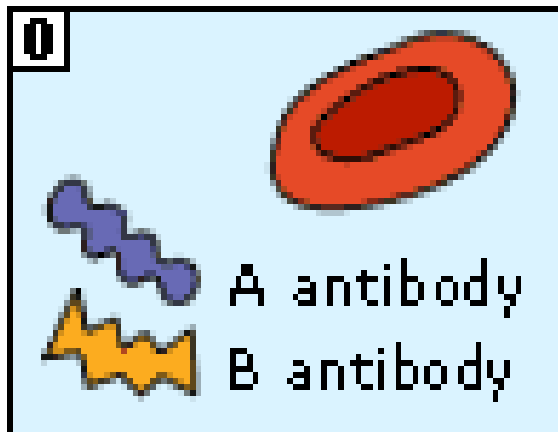
Blood group B

If you belong to the blood group B, you have B antigens on the surface of your RBCs and A antibodies in your blood plasma.



Blood group AB

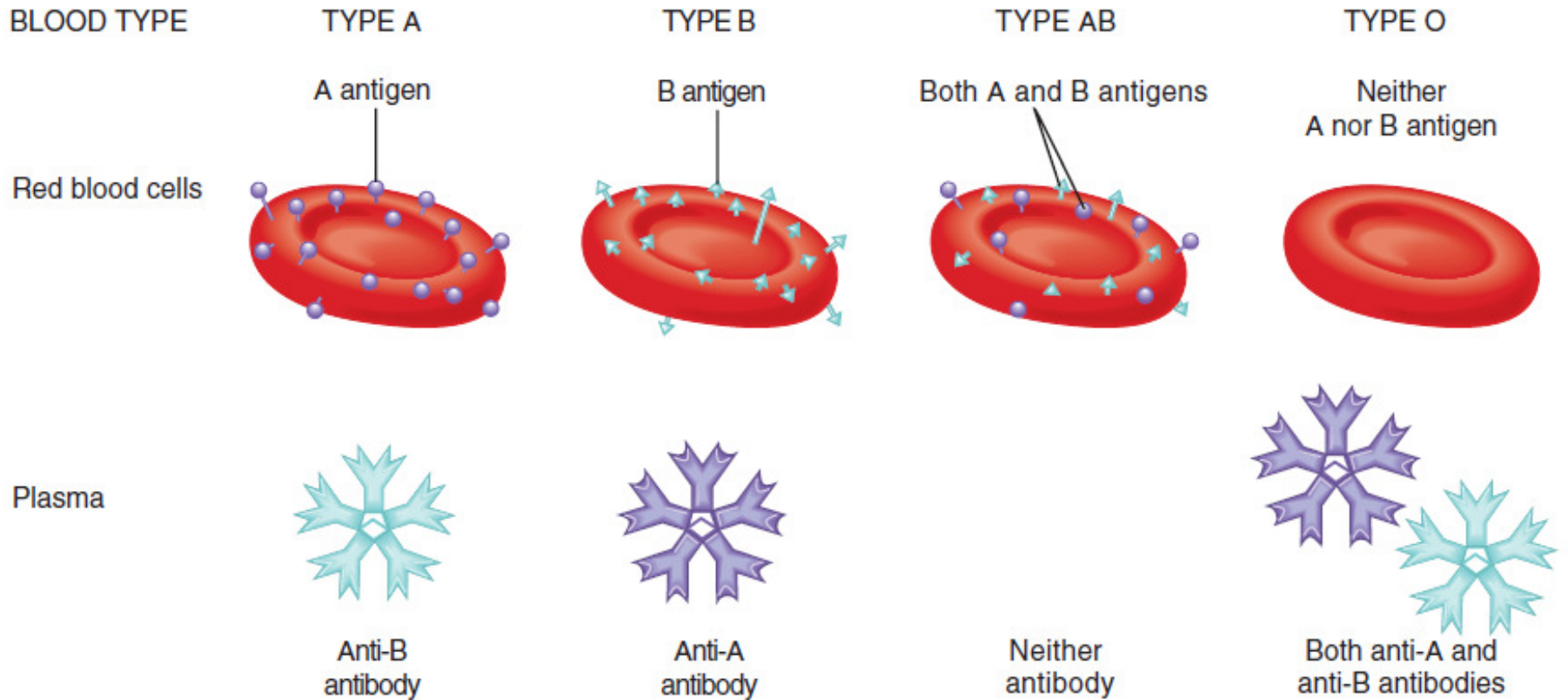
If you belong to the blood group AB, you have both A and B antigens on the surface of your RBCs and no A or B antibodies at all in your blood plasma.

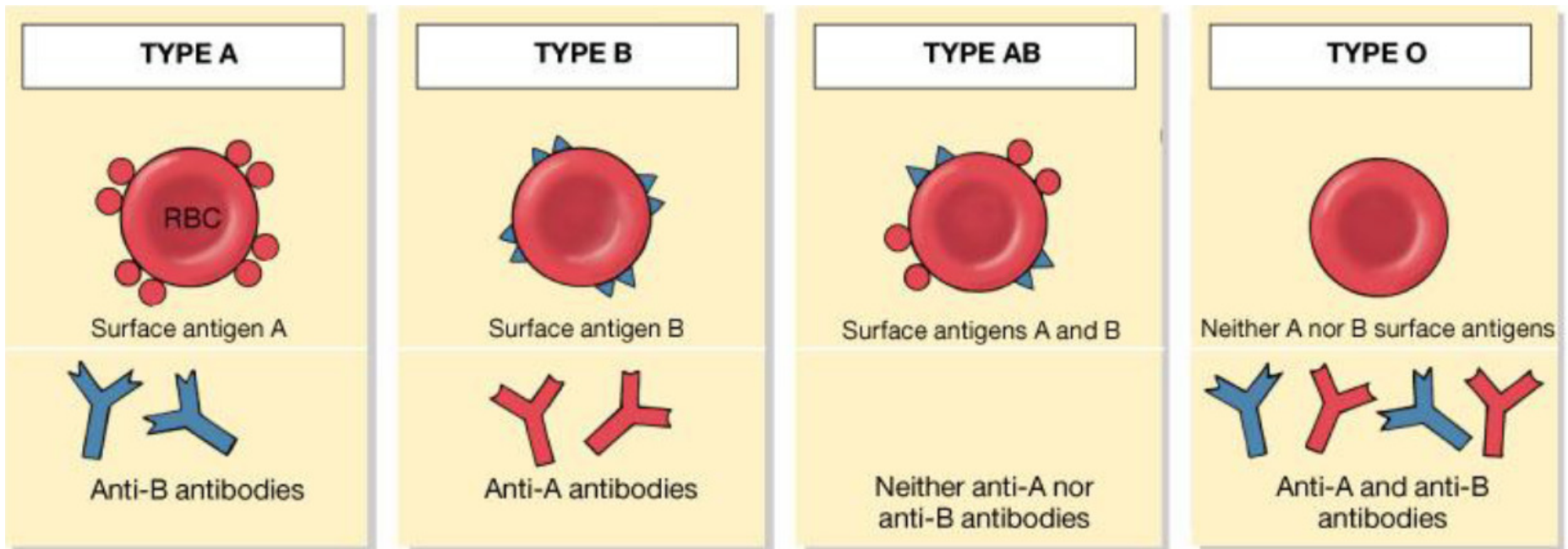


Blood group O

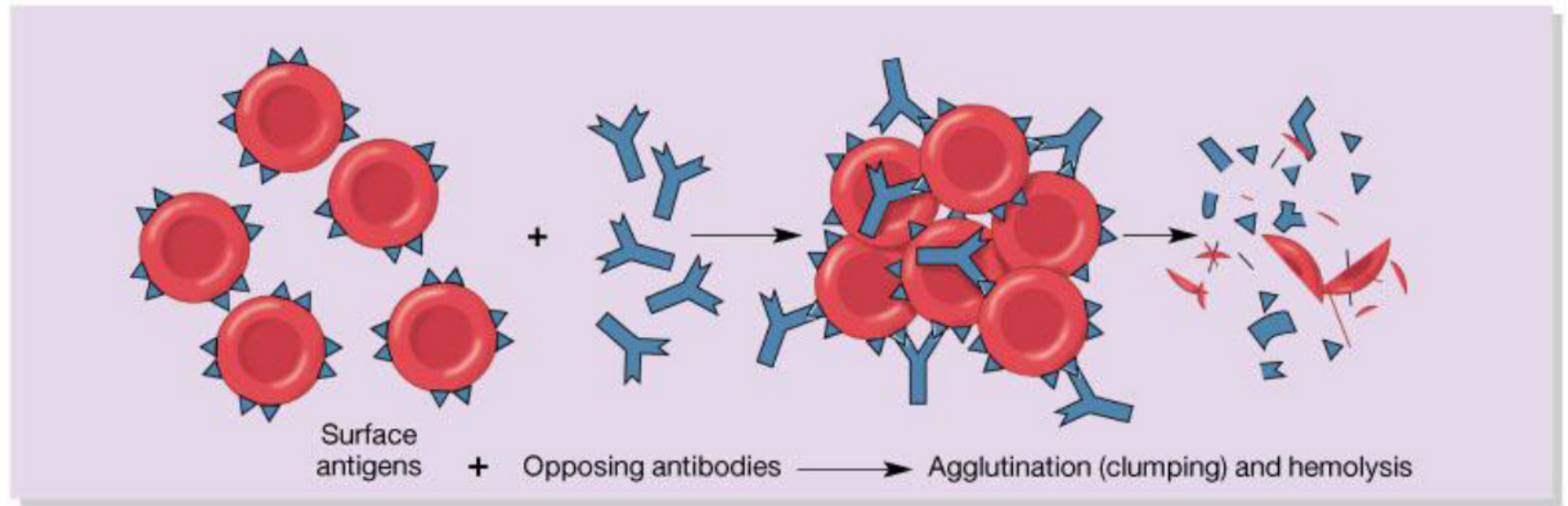
If you belong to the blood group O (null), you have neither A or B antigens on the surface of your RBCs but you have both A and B antibodies in your blood plasma.

BLOOD GROUP TYPES





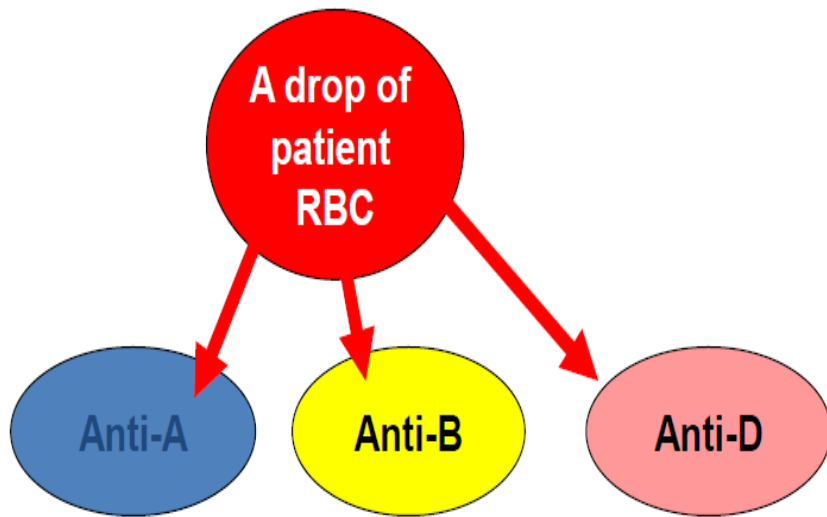
(a)



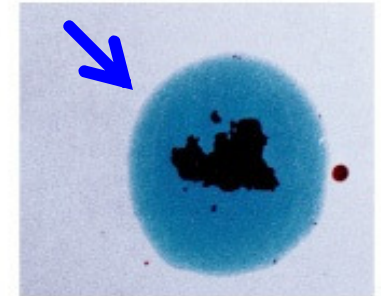
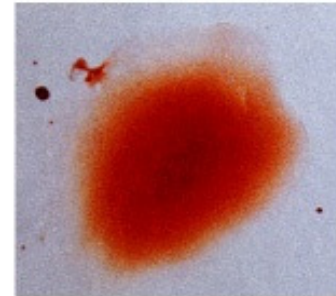
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BLOOD TYPING

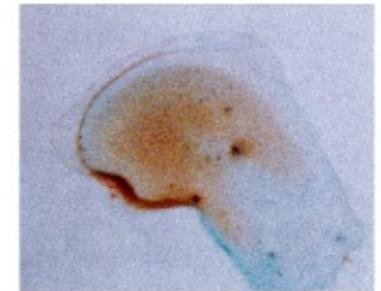
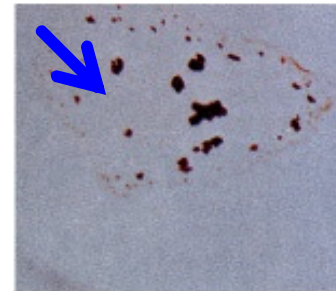
Anti-D serum determines Rh+ and Rh-



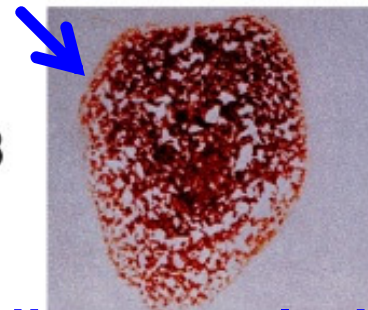
Type A



Type B



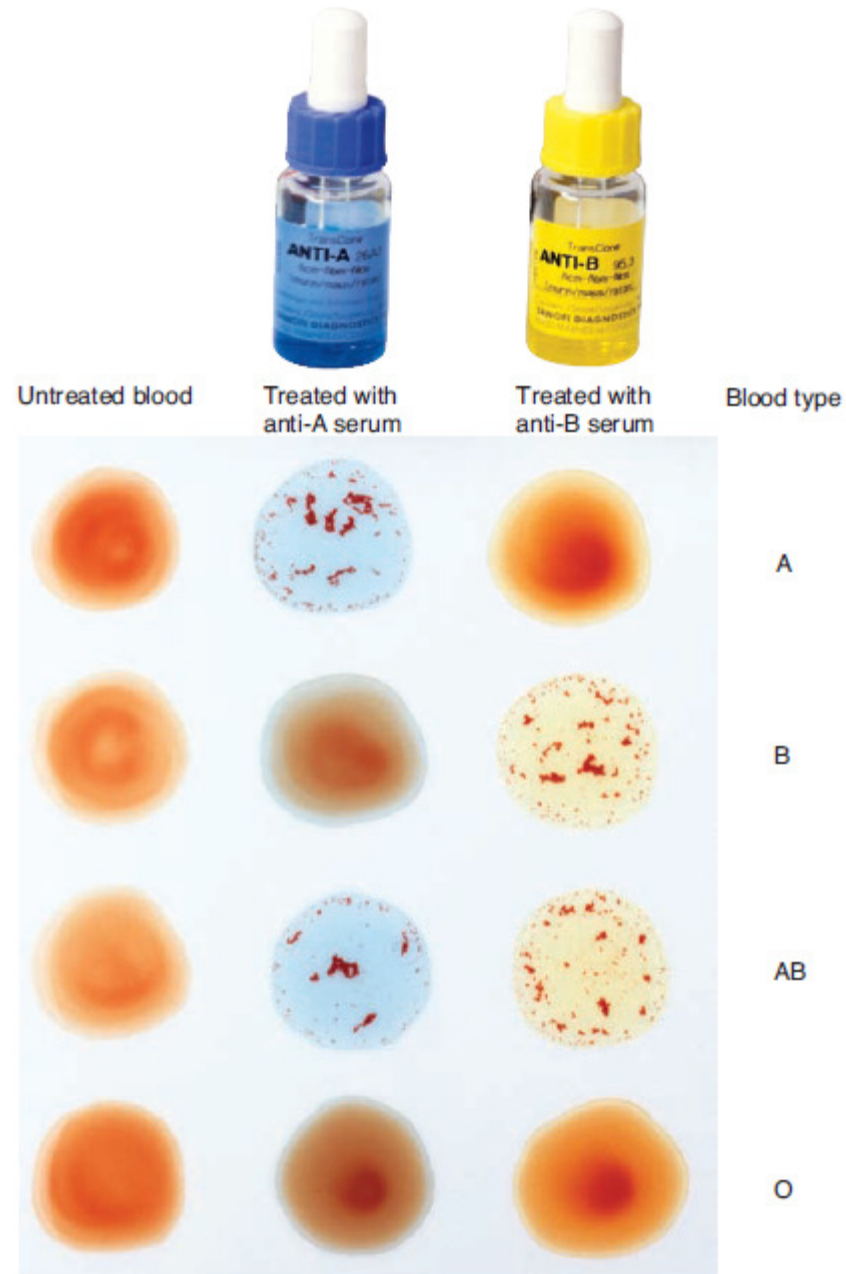
Type AB



Blue arrows indicate agglutination

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Detremination of blood groups



Importance of blood groups

- In Blood transfusion
- In preventing hemolytic disease (Rh incompatibility)
- In paternity disputes
- In medicolegal cases
- In knowing susceptibility to disease
 - Group O- duodenal cancer
 - Group A- Carcinoma of stomach, pancreas & salivary glands

Agglutination in transfusion reaction

- If a patient of blood group A transfused with blood of group B
- The anti-B in plasma will agglutinate the transfused group B cells:
- Outcome:
 - The clumped cells plug small blood vessels (kidney shut down).
 - Sometimes immediate hemolysis.

Agglutination in transfusion reaction

- If a person with blood group A transfused with blood of group B (contains anti-A in plasma)
- The anti-A in plasma of recipient blood group B will agglutinate the transfused cell (A)
- The clumped cells plug small blood vessels
- Sometimes causes immediate hemolysis
 - **Transfusion reaction**

Rh incompatibility between mother and fetus

- Mother is Rh-ve and first baby is Rh+ve:
- At delivery
 - Fetal Rh+ RBC cross to maternal blood
 - The mother will develop Anti-D after delivery.
 - First child escapes & is safe

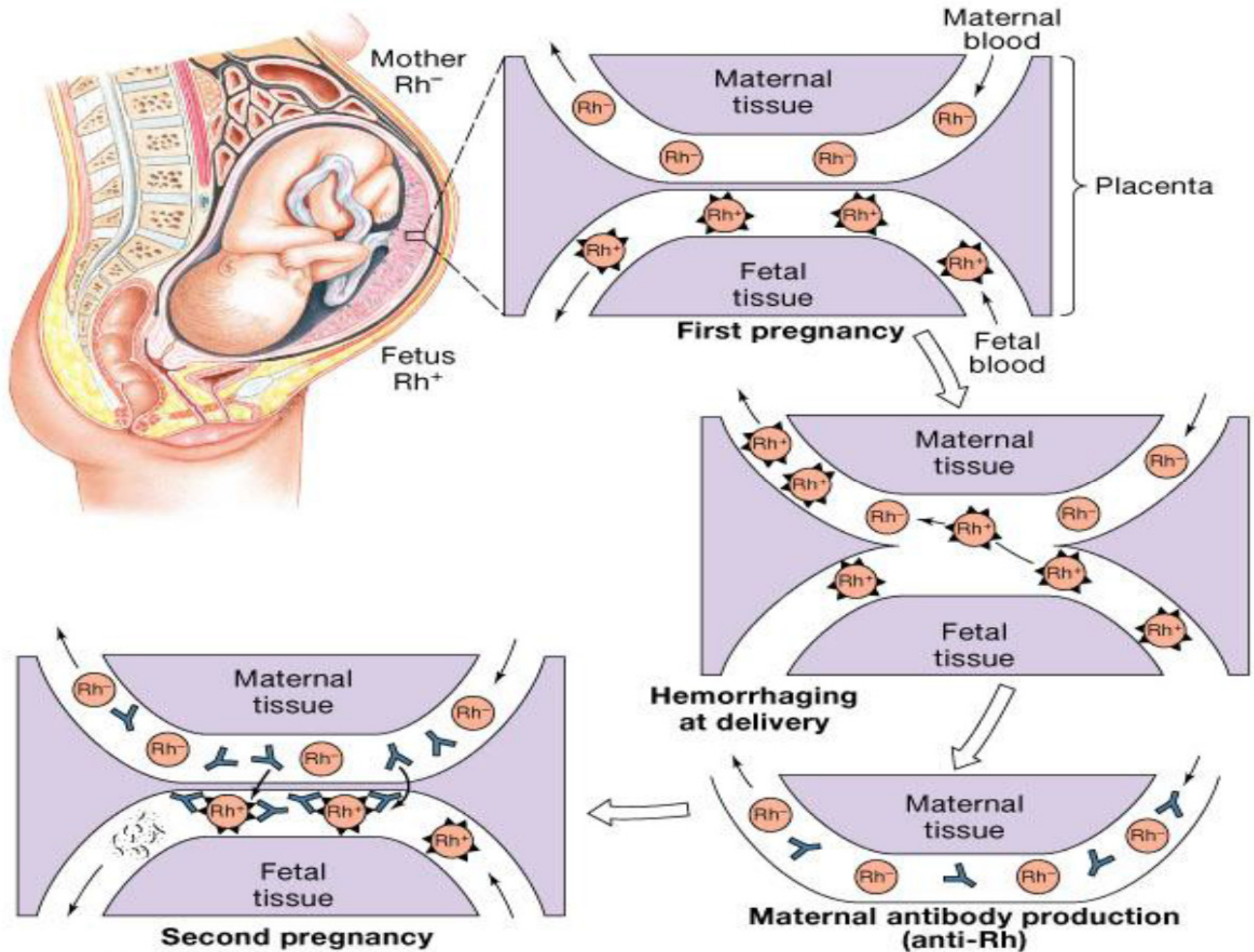
If the mother was transfused before with Rh+ve blood before, first child will also be affected

Rh incompatibility between mother and fetus

- Second fetus
 - If Rh+ve
 - Anti-D crosses placenta and destroys fetal Rh+ RBC

Outcome?

Hemolytic Disease of the newborn



ERYTHROBLASTOSIS FETALIS / HEMOLYTIC DISEASE OF NEW BORN

1. Hemolytic anemia:

- If severe:
- treated with exchange transfusion:
- Replace baby blood with Rh-ve RBC (several times)

2. Hydrops fetalis (death in utero)

Prevention:

Injecting the mother with anti-D immediately after 1st childbirth
Antenatal (during pregnancy) prophylaxis

ERYTHROBLASTOSIS FETALIS

- Prophylaxis

- Anti-D injection is given I/M to the mother

- Prevalence of Disease

- 1st Pregnancy ----- 0%
- 2nd Pregnancy ----- 3%
- 3rd Pregnancy ----- 10%

ERYTHROBLASTOSIS FETALIS

■ Etiology

- Exposure of the mother to the fetal RBC antigens
- Production of antibodies against foreign antigens in maternal circulation
- Maternal resensitization in the subsequent pregnancy
- Free passage of antibodies through the placenta
- Fetal red cell hemolysis

ERYTHROBLASTOSIS FETALIS

- Clinical Features
 - Anemia
 - Jaundice
 - Hepatosplenomegaly
 - Generalized edema
 - Immature RBC in circulation
 - Kernicterus

ERYTHROBLASTOSIS FETALIS

■ Diagnosis

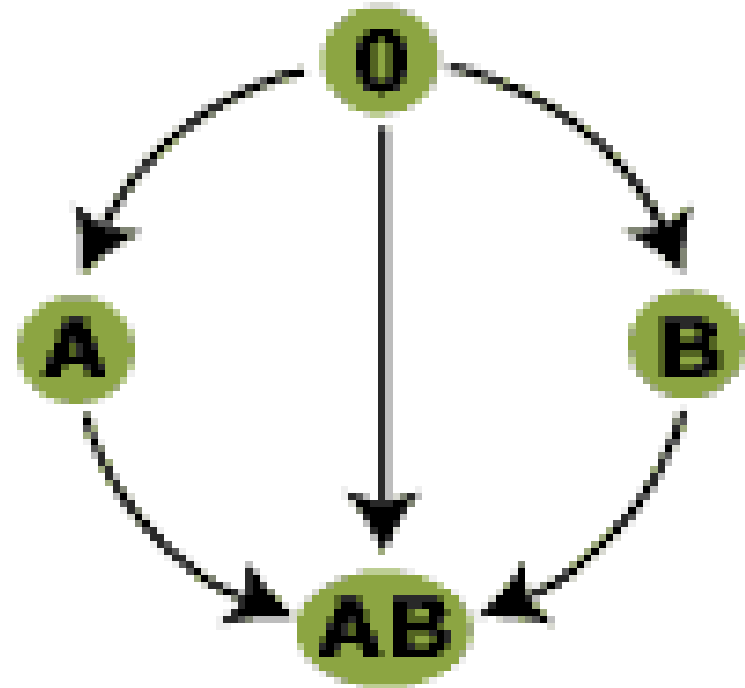
- Rapidly rising Rh antibody level in the mother during pregnancy
- Amniotic fluid showing high levels of bilirubin
- +ve coombs test on fetal cord blood

■ Treatment

- Exchange transfusion
- Phototherapy
- Drugs

DONORS AND RECIPIENTS

- People with blood group o are called "universal donors" and people with blood group AB are called "universal receivers."

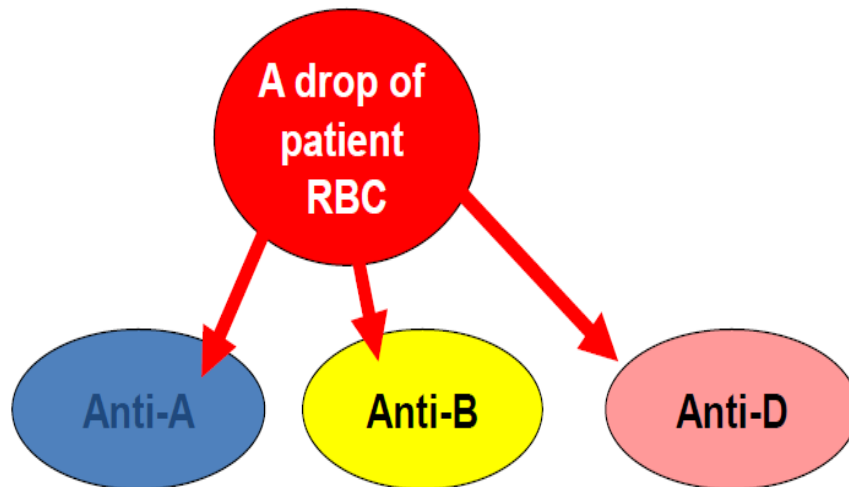


If You Have You Can Receive

O+	O+	O-		
O-	O-			
A+	A+	A-	O+	O-
A-	A-	O-		
B+	B+	B-	O+	O-
B-	B-	O-		
AB+	AB+	AB-	O+	O-
	A+	A-	B+	B-
AB-	AB-	O-	A-	B-

Blood tests before transfusion

- 1. Blood group type of patient (recipient)
- 2. Cross-matching



**2. Cross-matching:
donor cells
+
recipients (patient)
serum**

**mixing of donor's cells with
recipient's plasma**

DONORS AND RECIPIENTS

Blood Group	Antigens	Antibodies	Can give Blood to	Can receive blood from
AB	A and B	None	AB	AB, A, B and O
A	A	B	A and AB	A and O
B	B	A	B and AB	B and O
O	None	A and B	AB, A, B and O	O

Complications of blood transfusion

- 1. Immune reaction: Incompatible blood transfusion leading to immediate or delayed reaction, fever, haemolysis, allergic reaction**
- 2. Transmission of diseases; malaria, syphilis, viral hepatitis & Aids**
- 3. Iron overload due to multi-transfusion in case of sickle cell anemia and thalassemia.**

TRANSFUSION REACTIONS

■ 2 Types

■ Immediate

- Agglutination
- Hemolysis
- Fever
- Allergic reaction
- Acute renal shutdown
 - Renal vasoconstriction
 - Circulatory shock
 - Tubular blockage

■ Delayed

- Sensitization
- Thrombo Phlebitis

BLOOD TRANSFUSION

- **Indications**
 - When blood is lost as a whole or as a result of haemorrhage or surgery
 - Diseases
 - Anemias



TESTS DONE BEFORE TRANSFUSION

- **Blood grouping**
 - **ABO & Rh grouping of donor and recipient**
 - **Cross matching**
 - **Screening of Donor Plasma**

THANK YOU