BLOOD GROUP

Multiplicity of Antigen in Blood cells:-

At least 30 commonly occuring and hundreds of other rare antigens have found on surface of human blood cells.

Two perticular types are:-

(a) O-A-B System

(b) Rh System

O-A-B Blood type:-

→ Two antigen type A & type B, occur on the surface of the RBC.

→ These antigens (agglutinogens) cause most blood transfusion reactions

Landsteiner's laws:-

(1) It States that if an agglutinogen is present on RBC. the corresponding agglutinin must be absent from plasma.

(2) If an agglutinogen is absent on RBC, the corresponding agglutinogen must be present in plasma It has exceptions

* ABO blood group follows co-dominance.
* Relative frequencies of blood types:-

A- 41%
B- 9%
AB- 3%

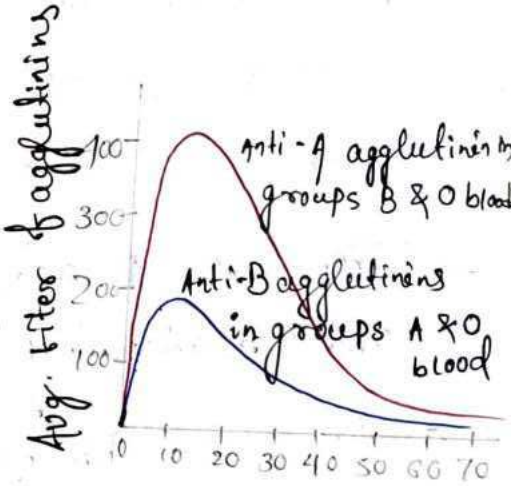
Population	A	B	AB	O
Indian	23	33	7	37
Asian	25	25	5	45
Europeans	42	9	3	46

* All Indians in peru (S.A.) have only 'O' blood group.

Genotypes	Blood types	Agglutinogens	Agglutinins
OO	O	–	anti-A, anti-B
OA or AA	A	A	Anti-B
OB or BB	B	B	Anti-A
AB	AB	A & B	–

Titer of Agglutinins :-

1. Immediately after birth: quantity of agglutinogens in plasma in almost zero.
2. 2-8 months: agglutinins begin to form in body.
3. 8-10 days of age: Max titer



Origin of agglutinins :-

→ The agglutinins are gamma globulins, and they are produced by bone marrow and lymph gland cells.

→ Most of them are IgM & IgG.

→ In A-B-O system, the plasma agglutinins responsible for causing transfusion reactions develop spontaneously whereas in Rh system spontaneous agglutinins almost never occures.

→ The person must first be massively exposed to an Rh antigen, to cause any significant transfusion reaction.

Rh antigen :-

→ 6 common type of Rh antigen. each of which is called Rh factor.

→ Degignated as C, D, E, c, d & e.

* The person who has 'C' antigen does not have 'c' antigen but the person missing 'C' antigen, will have 'c' antigen. These same is true for D-d & E-e antigen.

→ The type D antigen is widely Prevalent is population and considerably more antigenic than the other Rh-antigen.

→ The firm Rh factor (+)/(-) refer to Rh (D) antigen only.

→ Anyone who has this type of antigen is said to be Rh(+) and a person who does not have type D is said to be Rh (-)

→ In India, approx 94.61% ⇒ Rh (+)
About 85% of white people ⇒ Rh (+)

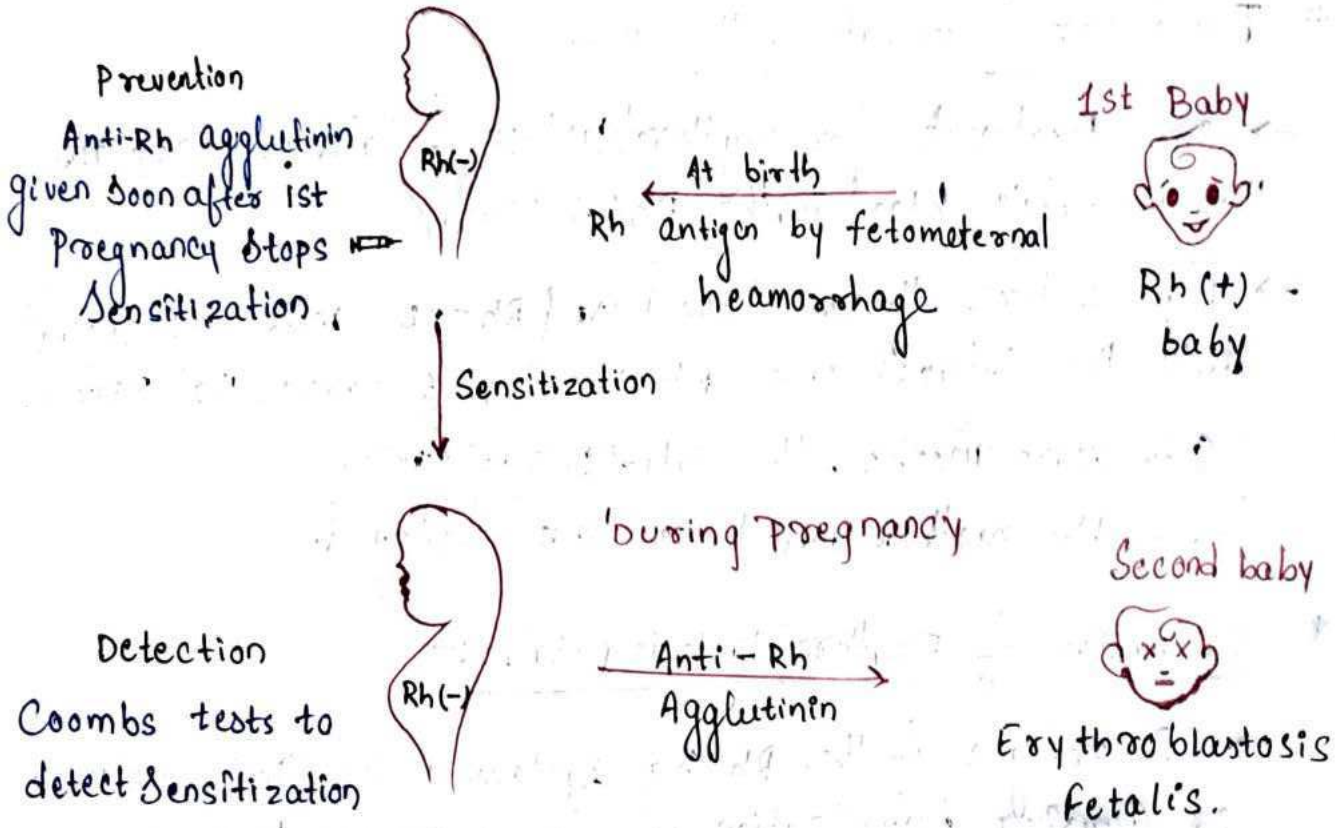
→ Exposure of Rh (-) entity Rh (+) blood will start anti-Rh agglutinins ~~will starts~~ anti formation.

→ Maximum concentration will reach in about 2-4 months later.

→ Rh reaction is a a delayed tranfusion reaction on first time.

Erythroblastosis fetalis :-

→ If mother was Rh (-) earlier sensitized to Rh antigen, (by any means) then first child can be affected too.



Every next child will have ↑ed chance of erythroblastosis fetalis. 0 % of 1st child will have e. fetalis
3 % of 2nd child.
10 % of 3rd child.

Clinical Manifestations:-

1. Liver & Spleen enlarged.
2. Severe Anemia
3. Permanent damage to motheur area of brain.
4. Hydrops fetalis (edema)

* Kernicterus

→ Is a bilirubin.

→ Includes brain dysfunction. Although the severe anemia of erythroblastosis fetalis is usually the cause of death.

→ Because of precipitation of bilirubin in the neuronal cells causing destruction of many, a condition called kernicterus.

* Treatment of neonates with E.F:-

→ One treatment for erythroblastosis fetalis is to replace the neonate's blood with Rh(-) blood.

→ By the time these transfused Rh-ve cells are replaced with the infant's own Rh(+) cells. A process that requires 6 or more weeks, the anti-Rh agglutinins that had come from the mother will have been destroyed.

* Prevention of Erythroblastosis Fetalis:-

→ D antigen of the Rh bg System is the Primary cell in causing imunization of an Rh(-) mother to an Rh(+) fetus.

→ The anti-D antibody is also administrated to Rh(-) women. which prevents Sensitization of the mother to the D-antigen.

Transfusion Reactions :-

Type	Cause	Clinical Feature
Hymolytic		
Immediate	ABO incompatibility	Ranges from fever, chills to shock, renal failure
Delayed	Rh incompatibility, secondary or delayed response to RBC antigen	Recurrent anemia, may be fever.
Non-Hymolytic		
Febrile Rxn	Contamination of stored blood with endotoxin or due to cytokines that are released on storage	Fever & Chills

BLOOD Storage :-

→ About 450 ml of blood is collected from a vein (usually antecubital vein)

→ Collected in flexible plastic bag, which already has Chemical in it.

① Sodium citrate : Binds to Calcium and prevents clotting

② Phosphate : To buffer the pH and also provide Source of Phosphate.

③ Dextrose : To provide energy Source.

④ Adenine : To provide the Substrate for ATP Synthesis.

→ Can be Stored for upto 35 days at 4°C.

Changes due to prolonged Storage :-

* Effects of Storage of blood on RBCs.

Structural changes

→ RBCs tending to lose their shape & becoming Spherical
→ Loss of membrane flexibility – Not deformable and likely to be removed quickly from circulation.
→ Loss of membrane stability.

Biochemical changes

→ Decreased glycolysis.
→ Reduced glutathione.
→ Depletion of ATP and adenine.

→ Granulocytes lose their phagocytic and bactericidal proper within 4-6 hrs.
→ Platelets could become nonfunctional with 36-48 hours
→ potassium levels inc. due to loss of potassium from RBCs into plasma.