Blood Bank

&BLOODTRANSFUSION

Lecturer Objectives

1. **Historical notes.**
2. **Blood grouping.**
3. **Haemolytic disease of newborn.**
4. **Blood component.**
5. **Autologus blood transfusion**
6. **Blood complications.**
7. **Cross matching.**
8. **Coomb's test.**

Historical notes

- China, 1000 BC The soul was contained in the blood.
- Egyptians bathed in blood for their health.
- Romans was drinking the blood of fallen gladiators to gain strength and vitality and to cure epilepsy.
- The first recorded attempt of a blood transfusion was described by the 15th-century.
- In 1492, Blood of three boys was given to Pope Innocent VIII, who had fallen into a coma. Following orders from a physician, the blood was transferred to his mouth, as the concept of intravenous circulation had not yet been discovered. The three young blood donors, all ten years old, had undertaken this experiment after being promised a ducat each. Unfortunately, the Pope and all three boys died.

- 1665 The first recorded successful blood transfusion occurs in England: Physician Richard Lower keeps dogs alive by transfusion of blood from other dogs.

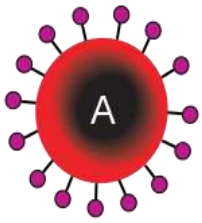
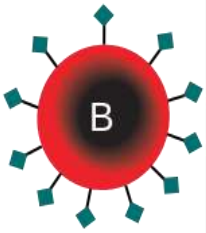
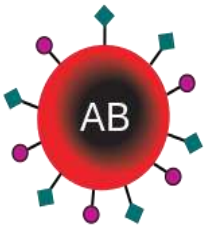
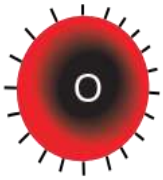
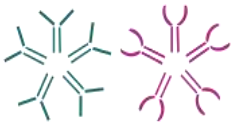
- 1667 Jean-Baptiste Denis in France and Richard Lower in England separately report successful transfusions from lambs to humans.
- Later, animals to humans transfusion becomes prohibited by law because of reactions.
- 1795 In Philadelphia, American physician Philip Syng Physick, performs the first human blood transfusion, although he does not publish this information.
- 1818 James Blundell, a British obstetrician, performs the first successful transfusion of human blood to a patient for the treatment of postpartum hemorrhage. Using the patient's husband as a donor from the husband's arm and, using a syringe, successfully transfuses the wife.

- 1900 Karl Landsteiner, an Austrian physician, discovers the first three human blood groups, A, B, and C. Blood type C was later changed to O. His colleagues Alfred Decastello and Adriano Sturli add AB, the fourth type, in 1902. Landsteiner receives the Nobel Prize for Medicine for this discovery in 1930.
- By 1907, the blood of donors and recipients was routinely tested and matched
- From that time blood transfusion and recent blood banks start to grow and progress until now.

Blood grouping systems

ABO system

- All humans and many other primates can be typed for the ABO blood group. There are four principal types: A, B, AB, and O.
- There are two antigens and two antibodies that are mostly responsible for the ABO types. The specific combination of these four components determines an individual's type in most cases.

	Group A	Group B	Group AB	Group O
Red blood cell type				
Antibodies in Plasma	 Anti-B	 Anti-A	None	 Anti-A and Anti-B
Antigens in Red Blood Cell	 A antigen	 B antigen	 A and B antigens	None

Blood containing red cells with type A antigen on their surface has in its serum (fluid) antibodies against type B red cells. If, in transfusion, type B blood is injected into persons with type A blood, the red cells in the injected blood will be destroyed by the antibodies in the recipient's blood. In the same way, type A red cells will be destroyed by anti-A antibodies in type B blood. Type O blood can be injected into persons with type A, B, or O blood unless there is incompatibility with respect to some other blood group system also present. Persons with type AB blood can receive type A, B, or O blood, as shown in the table.

The ABO and Rh groups in transfusion			
system	**recipient type**	**donor red cell type**	**donor plasma type**
ABO	A	A* or O	A or AB
ABO	B	B or O	B or AB
ABO	O	O only	O, A, B, or AB
ABO	AB	AB*, A*, B, or O	AB
Rh	positive	positive or negative	positive or negative
Rh	negative	negative or positive**, ***	negative or positive**

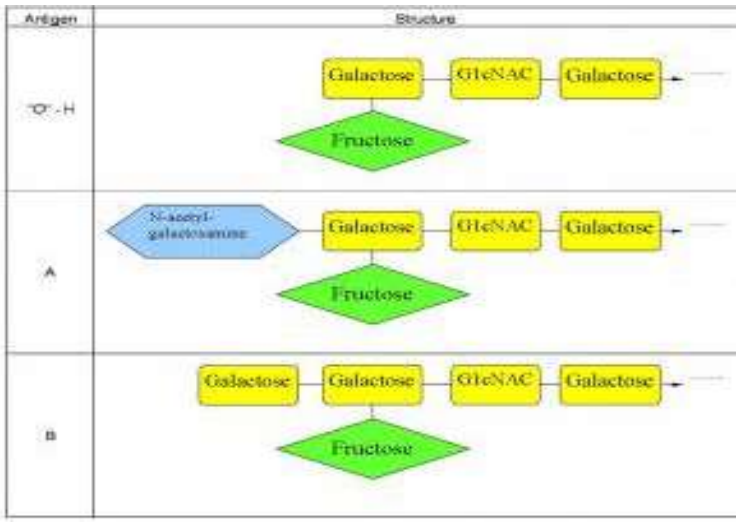
*Not if the patient's serum contains anti-A1 (antibody to common type A red cell in subgroup A patients).

**Not if the patient is a female less than 45 years old (childbearing possible), unless life-threatening hemorrhage is present and transfusion of Rh-positive blood is lifesaving.

***Not if the patient's serum contains anti-D (antibody to positive red cells), except under unusual medical circumstances.

ABO inheritance

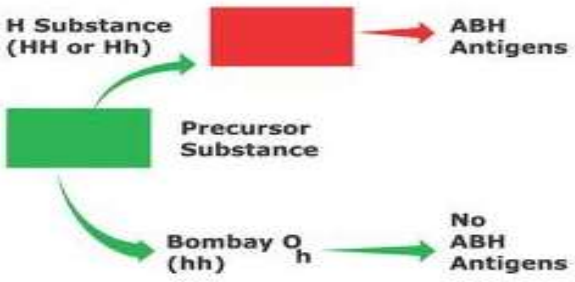
- The ABO blood group system consists of three alleles: two co-dominant A and B alleles, and one silent and recessive O allele.
- Inheritance of the ABO genes follows Mendelian principles. The system is controlled by a single gene located on the terminal portion of the long arm of chromosome 9 (9q34.2.
- This gene encodes a glycosyltransferase enzyme that adds a sugar residue to a carbohydrate structure called the H antigen.



- Therefore, ABO expression is also regulated by the H gene that is responsible for the synthesis of the H antigen substrate, the precursor of A and B antigens.
- The ABH antigens also occur as soluble glycoprotein antigens in secretion that include saliva, tears, breast milk, and seminal fluid. The presence of ABH antigens in the secretion is controlled by a Se gene.
- The **A allele** codes for an enzyme that adds a N-acetyl galactosamine to the subterminal galactose of H antigen, while **the B allele**, which differs from the former by four amino acid changes, codes for an enzyme that adds a D-galactose to the same subterminal galactose. In group AB individuals, both A and B structures are synthesized. The O allele occurs most frequently in modern humans and carries a human-specific inactivating mutation, which produces a non-functional enzyme, thus the H antigen remains without further modification on the surface of the cells.

Bombay blood group

- Bombay blood phenotype was first discovered in Bombay in India by Dr. Y.M. Bhende in1952. Named for the city in which it was first discovered. It describes individuals whose RBC's lack the H antigen, also known as Oh group
- The first person found to have the Bombay phenotype had an interesting blood type that reacted to other blood types in a way never seen before. The serum contained antibodies that reacted with all red blood cells' normal ABO phenotypes. The red blood cells appeared to lack all of the ABO blood group antigens and to have an additional antigen that was previously unknown.
- Individuals with the rare Bombay phenotype (hh) do not express H antigen (also called substance H), the antigen which is present in blood group O. As a result, they cannot make A antigen (also called substance A) or B antigen (substance B) on their red blood cells, whatever alleles they may have of the A and B blood-group genes, because A antigen and B antigen are made from H antigen. For this reason people
- who have Bombay phenotype can donate red blood cells to any member of the ABO blood group system (unless some other blood factor gene, such as Rh, is incompatible), but they cannot receive blood from any member of the ABO blood group system (which always contains one or more of A, B or H antigens), but only from other people who have Bombay phenotype.
- Receiving blood which contains an antigen which has never been in the patient's own blood causes an immune reaction due to the immune system of a hypothetical receiver producing immunoglobulins not only against antigen A and B, but also against H antigen.
- It is very important, in order to avoid any complications during a blood transfusion, to detect Bombay phenotype individuals, because the usual tests for ABO blood group system would show them as group O. Since Anti-H immunoglobulins can activate the complement cascade, it will lead to the lysis of red blood cells while they are still in the circulation, provoking an acute hemolytic transfusion reaction. This, of course, cannot be prevented unless the lab technologist that is involved is aware of the existence of the Bombay blood group and has the means to test for it.



RH system

- Discovered in 1940 after work on Rhesus monkeys
- The 2nd most important after ABO in the crossmatch test
- ABO system and has become known for its role in hemolytic The Rhesus system is much more complex than the anemia of newborn.
- Rh antibodies do not occur naturally but only develop due to allo-immunization by previous transfusion or pregnancy.
- Rh inheritance is controlled by 3 closely linked loci on each chromosome of a homologous pair
- Each locus has its own set of alleles which are: Dd , Cc , and Ee .
- The D gene is dominant to the d gene, but Cc and Ee are co-dominant.
- Depending on the presence of D antigen, individuals are
- **Classified as:**
    1. **Rh positive (85%).**
    2. **Rh negative (15%).**

General notes

- Blood group O is universal donor
- Blood group AB is universal recipient
- Group A can donate group A & AB and can receive blood from group A and group O
- Group B can donate group B & AB and receive blood from group B and group O

		Donor							
		O-	O+	B-	B+	A-	A+	AB-	AB+
Recipient	AB+	🩸	🩸	🩸	🩸	🩸	🩸	🩸	🩸
	AB-	🩸		🩸		🩸		🩸	
	A+	🩸	🩸			🩸	🩸		
	A-	🩸				🩸			
	B+	🩸	🩸	🩸	🩸				
	B-	🩸		🩸					
	O+	🩸	🩸						
	O-	🩸							

Cross matching

Cross Matching is a procedure performed prior to a blood transfusion to determine whether donor blood is compatible (or incompatible) with recipient blood. Compatibility is determined through matching of different blood group systems, the most important of which are the ABO and Rh system, and/or by directly testing for the presence of antibodies against a sample of donor tissues or blood..

Purpose

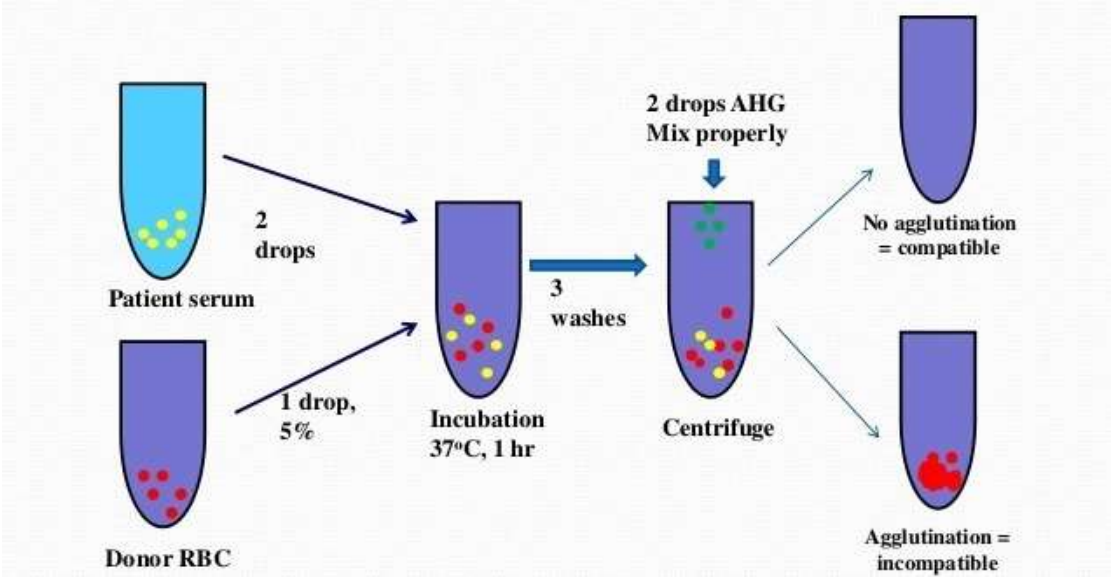
The crossmatch will detect the following:

1. Most recipient antibodies directed against antigens on the donor red blood cells.
2. Major errors in ABO grouping, labeling, and identification of donors and recipients.

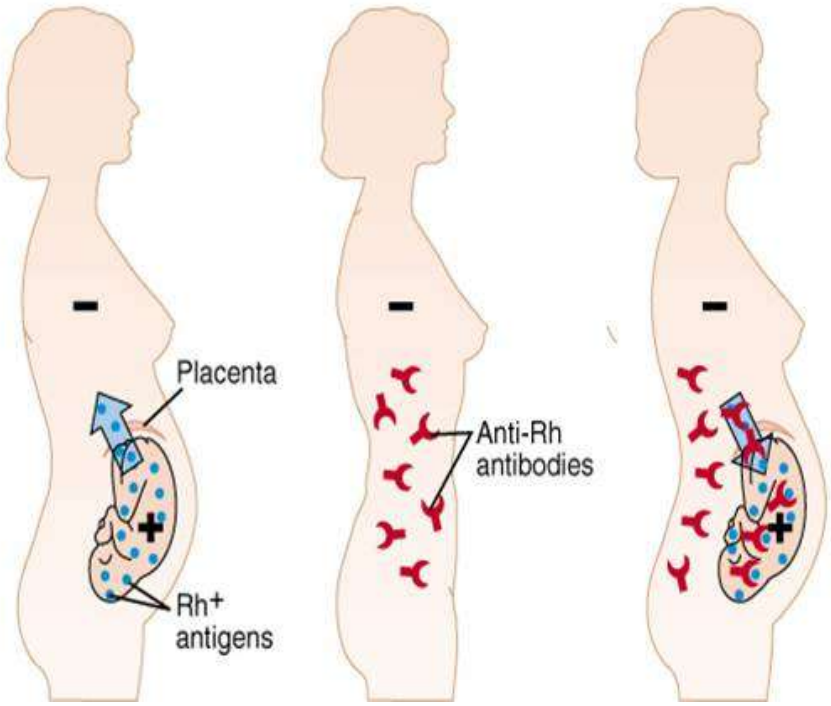
Principle

Cross-matching will detect incompatibilities between the donor and recipient that will not be evident on blood typing. There are two types of cross-matches: Major cross-match and Minor cross-match.

The **major crossmatch** involves testing the patient's serum with donor cells to determine whether the patient has an antibody which may cause a hemolytic transfusion reaction or decreased cell survival of donor cells. This is the most important cross-match.
The **minor crossmatch** involves testing the patients cells with donor plasma to determine whether there is an antibody in the donor's plasma directed against an antigen on the patient's cells.



HAEMOLYTIC DISEASE OF NEWBORN

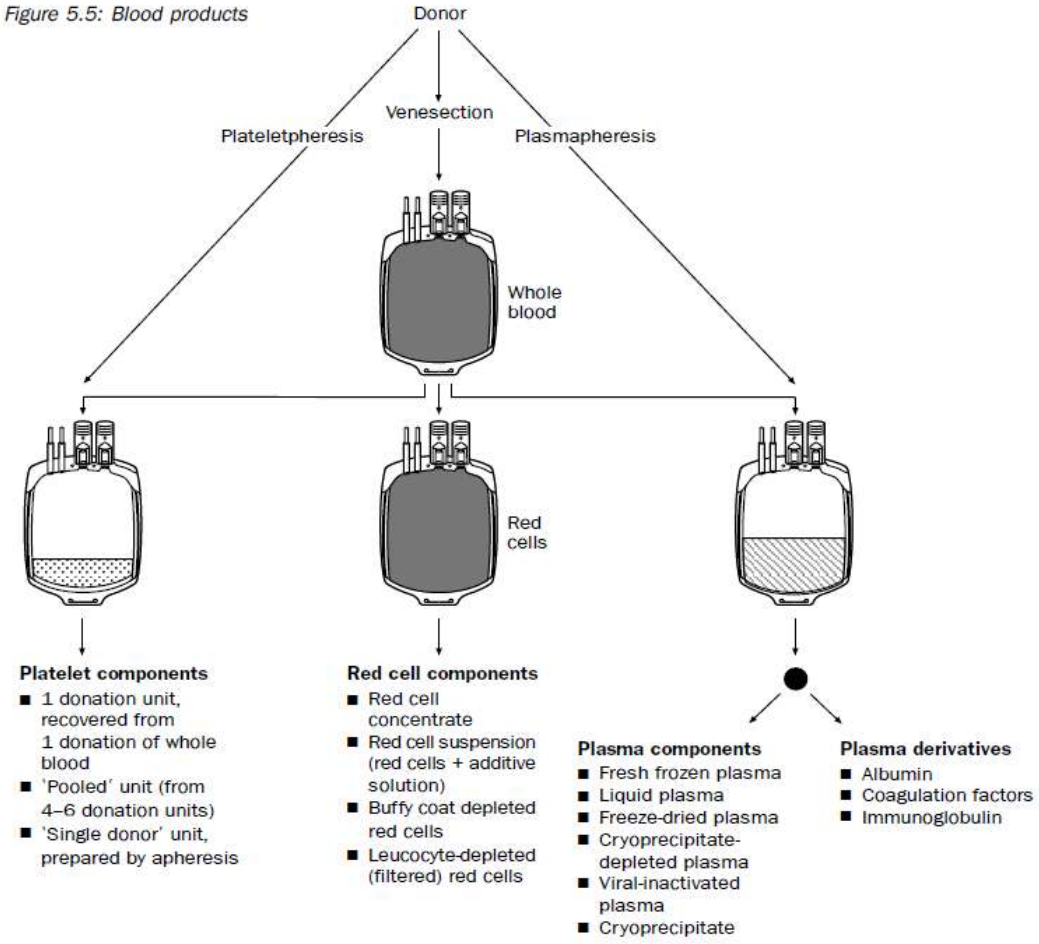


- **When an Rh D -** negative woman has a pregnancy with an Rh D positive fetus.
- Rh D - positive fetal red cells cross into the maternal circulation (especially at parturition and during the third trimester) and sensitize the mother to form anti-D.
- Anti - D crosses the placenta to the fetus during the next pregnancy with an RhD - positive fetus, coats the fetal red cells and results in destruction of these cells causing HAEMOLYTIC anaemia and jaundice.
- The main aim of management is to prevent anti - D antibody formation in Rh D - negative mothers.
- This can be achieved by the administration of small amounts of anti - D antibody which ‘ mop up ’ and destroy Rh D - positive fetal red cells before they can sensitize the immune system of the mother to produce anti – D

Blood component

Whole blood is now rarely used for transfusion. Blood component therapy makes clinical sense as most patients require a specific element of blood, such as red cells or platelets, and the dose can then be optimised. Each component is stored under ideal conditions (e.g. red cells must be refrigerated, platelets must not) and the use of precious blood donations becomes more efficient.

Figure 5.5: Blood products



WHOLE BLOOD (CPD-Adenine-1)

Data for a 450 ml (10%) donation volume. Whole blood may contain an alternative anticoagulant/additive solution.

Description	■ Up to 510 ml total volume (this volume may vary in accordance with local policies) ■ 450 ml donor blood ■ 63 ml anticoagulant ■ Haemoglobin approximately 12 g/ml ■ Haematocrit 35 (45%) ■ No functional platelets ■ No labile coagulation factors (V and VIII)
Unit of issue	1 donation, also referred to as a 'unit' or 'pack'
Infection risk	Not sterilized, so is capable of transmitting any agent present in cells or plasma which has not been detected by routine screening for transfusion-transmissible infections, including: ■ HIV-1 and HIV-2 ■ Hepatitis B and C ■ Other hepatitis viruses ■ Syphilis ■ Malaria ■ Chagas disease
Storage	■ Between +2°C and +6°C in an approved blood bank refrigerator, ideally fitted with a temperature chart and alarm ■ May be stored up to 35 days if collected in a suitable anticoagulant such as citrate phosphate dextrose with added adenine (CPDA-1) (see Figure 5.2) ■ During storage at +2°C to +6°C, changes in composition occur resulting from red cell metabolism (see Figure 5.3)
Indications	■ Red cell replacement in acute blood loss with hypovolaemia ■ Exchange transfusion ■ Patients needing red cell transfusions where red cell concentrates or suspensions are not available
Contraindications	Risk of volume overload in patients with: ■ Chronic anaemia ■ Incipient cardiac failure
Administration	■ Must be ABO and Rh compatible with the recipient ■ Complete transfusion within 4 hours of commencement ■ Never add medication to a unit of blood

RED CELL CONCENTRATE

May also be called 'packed red cells', 'concentrated red cells' or 'plasma-reduced blood'.

Description	■ 150–200 ml red cells from which most of the plasma has been removed ■ Haemoglobin approximately 20 g/100 ml (not less than 45 g per unit) ■ Haematocrit 55–75%
Unit of issue	1 donation
Infection risk	Same as whole blood
Storage	Same as whole blood
Indications	■ Replacement of red cells in anaemic patients ■ Use with crystalloid replacement fluids or colloid solution in acute blood loss
Administration	■ Same as whole blood ■ To improve transfusion flow, normal saline (50–100 ml) may be added using a Y-pattern infusion set

PLATELET CONCENTRATES (collected by plateletpheresis)

Description	■ Volume 150–300 ml ■ Platelet content 150–500 x 10^9, equivalent to 3–10 single donations ■ Platelet content, volume of plasma and leucocyte contamination depend on the collection procedure
Unit of issue	1 pack containing platelet concentrates collected by a cell separator device from a single donor
Infection risk	Same as whole blood
Storage	Up to 24 hours at 20°C–24°C (with agitation) unless collected using a blood cold chain system validated for longer storage periods; do not store at 4°C
Indications	■ Platelet concentrates collected by apheresis are, generally, equivalent to the same dose of platelet concentrates prepared from whole blood ■ If a specially typed, compatible donor is required for the patient, several doses may be obtained from the selected donor
Dosage	1 pack of platelet concentrate collected from a single donor by apheresis is usually equivalent to 1 therapeutic dose
Administration	Same as for recovered donor platelets, but ABO compatibility is more important: high titre counts of A or B in the donor plasma used to suspend the platelets may cause haemolysis of recipient's red cells

PLATELET CONCENTRATES (prepared from whole blood donations)

Description	Single donor unit in a volume of 50–60 ml of plasma should contain: ■ At least 55 x 10^9 platelets ■ <1.2 x 10^9 red cells ■ <0.12 x 10^9 leucocytes
Unit of issue	May be supplied as either: ■ Single donor unit: platelets prepared from one donation ■ Pooled unit: platelets prepared from 4 to 6 donor units 'pooled' into one pack to contain an adult dose of at least 240 x 10^9 platelets
Infection risk	■ Same as whole blood, but a normal adult dose involves between 4 and 6 donor exposures ■ Bacterial contamination affects about 1% of pooled units
Storage	■ 20°C–24°C (with agitation) for up to 5 days in specialized platelet packs, although some centres use ordinary plastic packs which restrict storage to 72 hours ■ Longer storage increases the risk of bacterial proliferation and septicaemia in the recipient
Indications	■ Treatment of bleeding due to: — Thrombocytopenia — Platelet function defects ■ Prevention of bleeding due to thrombocytopenia, such as in bone marrow failure
Contraindications	■ Not generally indicated for prophylaxis of bleeding in surgical patients, unless known to have significant pre-operative platelet deficiency ■ Not indicated in: — Idiopathic autoimmune thrombocytopenic purpura (ITP) — Thrombotic thrombocytopenic purpura (TTP) — Untreated disseminated intravascular coagulation (DIC) — Thrombocytopenia associated with septicaemia, until treatment has commenced or in cases of hypersplenism
Dosage	■ 1 unit of platelet concentrate/10 kg body weight: in a 60 or 70 kg adult, 4–6 single donor units containing at least 240 x 10^9 platelets should raise the platelet count by 20–40 x 10^9/L ■ Increment will be less if there is: — Splenomegaly — Disseminated intravascular coagulation — Septicaemia
Administration	■ After pooling, platelet concentrates should be infused as soon as possible, generally within 4 hours, because of the risk of bacterial proliferation ■ Must not be refrigerated before infusion as this reduces platelet function ■ 4–6 units of platelet concentrates (which may be supplied pooled) should be infused through a fresh standard blood administration set ■ Special platelet infusion sets are not required

	■ Platelet concentrates should be infused over about 30 minutes ■ Platelet concentrates prepared from Rh D positive donors should not be given to a Rh D negative potential child-bearing female ■ Platelet concentrates that are ABO compatible should be given whenever possible
Complications	Febrile non-haemolytic and allergic urticarial reactions are not uncommon, especially in patients receiving multiple transfusions. For management, see Section 7: *Adverse Effects of Transfusion*

FRESH FROZEN PLASMA

Description	■ Pack containing the plasma separated from one whole blood donation within 6 hours of collection and then rapidly frozen to –25°C or colder ■ Contains normal plasma levels of stable clotting factors, albumin and immunoglobulin ■ Factor VIII level at least 70% of normal fresh plasma level
Unit of issue	■ Usual volume of pack is 200–300 ml ■ Smaller volume packs may be available for children
Infection risk	■ If untreated, same as whole blood ■ Very low risk if treated with methylene blue/ultraviolet light inactivation (see virus 'inactivated' plasma)
Storage	At –25°C or colder for up to 1 year
Indications	■ Replacement of multiple coagulation factor deficiencies, e.g.: — Liver disease — Warfarin anticoagulant overdose — Depletion of coagulation factors in patients receiving large volume transfusions. ■ Disseminated intravascular coagulation (DIC) ■ Thrombotic thrombocytopenic purpura (TTP)
Dosage	Initial dose of 15 ml/kg
Administration	■ Must normally be ABO compatible to avoid risk of haemolysis in recipient ■ No crossmatching needed ■ Before use, should be thawed in water which is between 30°C and 37°C. Higher temperatures will destroy clotting factors and proteins ■ Once thawed, should be stored in a refrigerator at 2°C–6°C ■ Infuse using a standard blood infusion set as soon as possible after thawing ■ Labile coagulation factors rapidly degrade; use within 6 hours of thawing
Precautions	■ Acute allergic reactions are not uncommon, especially with rapid infusions ■ Severe life-threatening anaphylactic reactions occasionally occur ■ Hypovolaemia alone is *not* an indication for use

CRYOPRECIPITATE

Description	■ Prepared from fresh frozen plasma by collecting the precipitate formed during controlled thawing and resuspending it in 10–20 ml plasma ■ Contains about half of the Factor VIII and fibrinogen in the donated whole blood: e.g. Factor VIII: 80–100 i.u./pack; fibrinogen: 150–300 mg/pack
Unit of issue	Usually supplied as a single donor pack or a pack of 6 or more single donor units that have been pooled
Infection risk	As for plasma, but a normal adult dose involves at least 6 donor exposures
Storage	At –25°C or colder for up to 1 year
Indications	■ As an alternative to Factor VIII concentrate in the treatment of inherited deficiencies of: — Von Willebrand Factor (von Willebrand's disease) — Factor VIII (haemophilia A) — Factor XIII ■ As a source of fibrinogen in acquired coagulopathies: e.g. disseminated intravascular coagulation (DIC)
Administration	■ If possible, use ABO-compatible product ■ No compatibility testing is needed ■ After thawing, infuse as soon as possible through a standard blood administration set ■ Must be infused within 6 hours of thawing

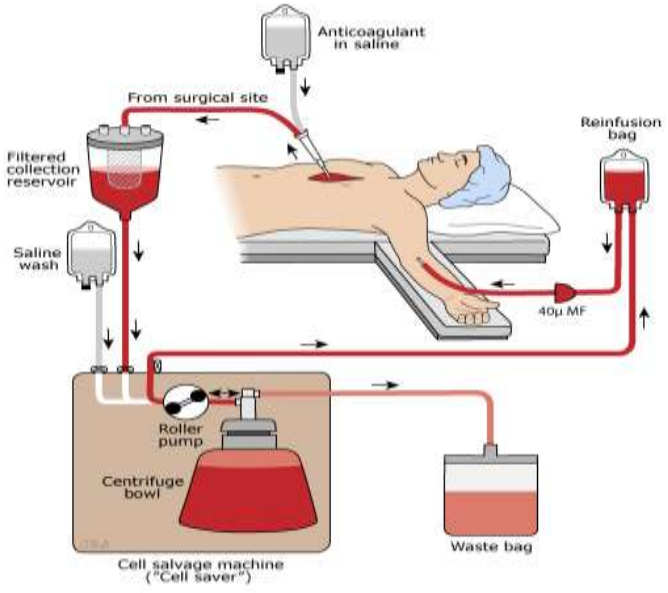
Autologous donation and transfusion

Anxiety over acquired immune deficiency syndrome (AIDS) and other infections has increased thedemand for autotransfusion.

There are three ways of administration of an autologous transfusion:

1. **Predeposit**
   Blood is taken from the potential recipient in the weeks immediately prior to elective surgery.
2. **Haemodilution**
   Blood is removed immediately prior to surgery once the patient has been anaesthetized and then reinfused at the end of the operation.
3. **Salvage**
   Blood lost during the operation is collected during heavy blood loss and then reinfused.

× Autotransfusion is the **safest form** of transfusion with regard to transmission of **viral** disease though it has a higher risk of bacterial contamination and of clerical errors.
× The individual involved must be fit enough to donate blood and the predicted operative replacement transfusion should be **2-4 units.** So**larger replacement** transfusions would require blood to be collected over a longer period and red cells stored in the **frozen state**, which is both labor **intensive and expensive**.
× The high cost and initial restriction of its use to patients undergoing elective surgery means that it will benefit only a minor proportion of the total number of blood recipients.
× Preoperative autotransfusion is largely reserved for those patients with multiple antibodies can find available matched blood.



Transfusion reaction

Acute complications of transfusion

Acute transfusion reactions occur during or shortly after (within 24 hours) the transfusion. They can be broadly classified in the following three categories according to their severity and the appropriate clinical response.

Category 1: Mild reactions

- Mild hypersensitivity: allergic, urticarial reactions

Category 2: Moderately severe reactions

- Moderate–severe hypersensitivity (severe urticarial reactions)
- Febrile non-haemolytic reactions:
  - — Antibodies to white cells, platelets
  - — Antibodies to proteins, including IgA
- Possible bacterial contamination (early signs)
- Pyrogens

Category 3: Life-threatening reactions

- Acute intravascular haemolysis
- Bacterial contamination and septic shock
- Fluid overload
- Anaphylactic reactions
- Transfusion-associated lung injury

Delayed complications of transfusion

Delayed complications of transfusion essentially fall into two categories.

Transfusion-transmitted infections

- HIV-1 and HIV-2
- HTLV-I and II
- Viral hepatitis B and C
- Syphilis
- Chagas disease
- Malaria
- Cytomegalovirus

Other rare transfusion-transmissible infections include:

- Human parvovirus B19
- Brucellosis
- Epstein-Barr virus
- Toxoplasmosis
- Infectious mononucleosis
- Lymes disease.

The possibility has been raised that blood could transmit a new variant of Creutzfeldt-Jakob disease (CJD). Initially labelled 'new variant CJD' (nvCJD), it is now known as 'variant CJD' (vCJD). It is the subject of intense investigation, but there is no evidence of transfusion-associated transmission of CJD or its variant. (See WHO *Weekly Epidemiological Record*, 1998, 73: 6–7).

Other delayed complications of transfusion

Other delayed complications of transfusion which occur days, months or even years after the transfusion has been completed, include:

- Delayed haemolytic reaction
- Post-transfusion purpura
- Graft-vs-host disease
- Iron overload (in patients who receive repeated transfusions)

CATEGORY	SIGNS	SYMPTOMS	POSSIBLE CAUSE
CATEGORY 1: MILD	▪ Localized cutaneous reactions: — Urticaria — Rash	▪ Pruritus (itching)	▪ Hypersensitivity (mild)
CATEGORY 2: MODERATELY SEVERE	▪ Flushing ▪ Urticaria ▪ Rigors ▪ Fever ▪ Restlessness ▪ Tachycardia	▪ Anxiety ▪ Pruritus (itching) ▪ Palpitations ▪ Mild dyspnoea ▪ Headache	▪ Hypersensitivity (moderate–severe) ▪ Febrile non-haemolytic transfusion reactions: — Antibodies to white blood cells, platelets — Antibodies to proteins, including IgA ▪ Possible contamination with pyrogens and/or bacteria
CATEGORY 3: LIFE-THREATENING	▪ Rigors ▪ Fever ▪ Restlessness ▪ Hypotension (fall of ≥20% in systolic BP) ▪ Tachycardia (rise of ≥20% in heart rate) ▪ Haemoglobinuria (red urine) ▪ Unexplained bleeding (DIC)	▪ Anxiety ▪ Chest pain ▪ Pain near infusion site ▪ Respiratory distress/shortness of breath ▪ Loin/back pain ▪ Headache ▪ Dyspnoea	▪ Acute intravascular haemolysis ▪ Bacterial contamination and septic shock ▪ Fluid overload ▪ Anaphylaxis ▪ Transfusion-associated lung injury

Note: If an acute transfusion reaction occurs, first check the blood pack labels and the patient's identity. If there is any discrepancy, stop the transfusion immediately and consult the blood bank.

In an unconscious or anaesthetized patient, hypotension and uncontrolled bleeding may be the only signs of an incompatible transfusion.

In a conscious patient undergoing a severe haemolytic transfusion reaction, signs and symptoms may appear very quickly – within minutes of infusing only 5–10 ml of blood. Close observation at the start of the infusion of each unit is essential.

IMMEDIATE MANAGEMENT

CATEGORY 1: MILD

1 Slow the transfusion.

2 Administer antihistamine IM (e.g. chlorpheniramine 0.1 mg/kg or equivalent).

3 If no clinical improvement within 30 minutes or if signs and symptoms worsen, **treat as Category 2**.

CATEGORY 2: MODERATELY SEVERE

1 Stop the transfusion. Replace the infusion set and keep IV line open with normal saline.

2 Notify the doctor responsible for the patient and the blood bank immediately.

3 Send blood unit with infusion set, freshly collected urine and new blood samples (1 clotted and 1 anticoagulated) from vein opposite infusion site with appropriate request form to blood bank and laboratory for investigations.

4 Administer antihistamine IM (e.g. chlorpheniramine 0.1 mg/kg or equivalent) and oral or rectal antipyretic (e.g. paracetamol 10 mg/kg: 500 mg – 1 g in adults). Avoid aspirin in thrombocytopenic patients.

5 Give IV corticosteroids and bronchodilators if there are anaphylactoid features (e.g. broncospasm, stridor).

6 Collect urine for next 24 hours for evidence of haemolysis and send to laboratory.

7 If clinical improvement, restart transfusion slowly with new blood unit and observe carefully.

8 If no clinical improvement within 15 minutes or if signs and symptoms worsen, **treat as Category 3**.

CATEGORY 3: LIFE-THREATENING

1 Stop the transfusion. Replace the infusion set and keep IV line open with normal saline.

2 Infuse normal saline (initially 20–30 ml/kg) to maintain systolic BP. If hypotensive, give over 5 minutes and elevate patient's legs.

3 Maintain airway and give high flow oxygen by mask.

4 Give adrenaline (as 1:1000 solution) 0.01 mg/kg body weight by slow intramuscular injection.

5 Give IV corticosteroids and bronchodilators if there are anaphylactoid features (e.g. broncospasm, stridor).

6 Give diuretic: e.g. frusemide 1 mg/kg IV or equivalent

7 Notify the doctor responsible for the patient and the blood bank immediately.

8 Send blood unit with infusion set, fresh urine sample and new blood samples (1 clotted and 1 anticoagulated) from vein opposite infusion site with appropriate request form to blood bank and laboratory for investigations.

9 Check a fresh urine specimen visually for signs of haemoglobinuria (red or pink urine).

10 Start a 24-hour urine collection and fluid balance chart and record all intake and output. Maintain fluid balance.

11 Assess for bleeding from puncture sites or wounds. If there is clinical or laboratory evidence of DIC (see Section 9.11), give platelets (adult: 5–6 units) **and** either cryoprecipitate (adult: 12 units) or fresh frozen plasma (adult: 3 units). Use virally-inactivated plasma coagulation products, wherever possible.

12 Reassess. If hypotensive:
- Give further saline 20–30 ml/kg over 5 minutes
- Give inotrope, if available.

13 If urine output falling or laboratory evidence of acute renal failure (rising K^+, urea, creatinine):
- Maintain fluid balance accurately
- Give further frusemide
- Consider dopamine infusion, if available
- Seek expert help: the patient may need renal dialysis.

14 If bacteraemia is suspected (rigors, fever, collapse, no evidence of a haemolytic reaction), start broad-spectrum antibiotics IV, to cover *pseudomonas* and gram positives.

TYPE OF DRUG	RELEVANT EFFECTS	EXAMPLES		NOTES
		Name	Route and dosage	
Intravenous replacement fluid	Expand blood volume See Section 4.2	Normal saline	If patient hypotensive, 20–30 ml/kg over 5 minutes	Avoid colloid solutions
Antipyretic	Reduce fever and inflammatory response	Paracetemol	Oral or rectal 10 mg/kg	Avoid aspirin-containing products if patient has low platelet count
Antihistamine	Inhibits histamine mediated responses	Chlorphen-iramine	Intramuscular or IV 0.1 mg/kg	
Bronchodilator	Inhibits immune mediated bronchospasm	Adrenaline	0.01 mg/kg (as 1: 1000 solution) by slow intramuscular injection	Dose may be repeated every 10 minutes according to blood pressure and pulse until improvement occurs
		Consider salbutamol	By nebuliser	
		Aminophylline	5 mg/kg	
Inotrope	Increases myocardial contractility	Dopamine	IV infusion 1 microgm/kg/minute	▪ Dopamine in low doses induces vasodilation and improves renal perfusion ▪ Doses above 5 microgms/kg/minute cause vaso-constriction and worsen heart failure
		Dobutamine	IV infusion 1–10 microgm/kg/ minute	
Diuretic	Inhibits fluid reabsorption from ascending loop of Henle	Frusemide	Slow IV injection 1 mg/kg	

INVESTIGATING ACUTE TRANSFUSION REACTIONS

1 Immediately report all acute transfusion reactions, with the exception of mild hypersensitivity (Category 1), to the doctor responsible for the patient and to the blood bank that supplied the blood.

If you suspect the patient is having a severe life-threatening reaction, seek help immediately from the duty anaesthetist, emergency team or whoever is available and skilled to assist.

2 Record the following information on the patient's notes:

- Type of transfusion reaction
- Length of time after the start of transfusion that the reaction occurred
- Volume, type and pack numbers of the blood products transfused.

3 Take the following samples and send them to the blood bank for laboratory investigations:

- Immediate post-transfusion blood samples (1 clotted and 1 anticoagulated: EDTA/Sequestrene) from the vein opposite the infusion site for:
  - — Full blood count
  - — Coagulation screen
  - — Direct antiglobulin test
  - — Urea
  - — Creatinine
  - — Electrolytes
- Blood culture in a special blood culture bottle
- Blood unit and giving-set containing red cell and plasma residues from the transfused donor blood
- First specimen of the patient's urine following the reaction.

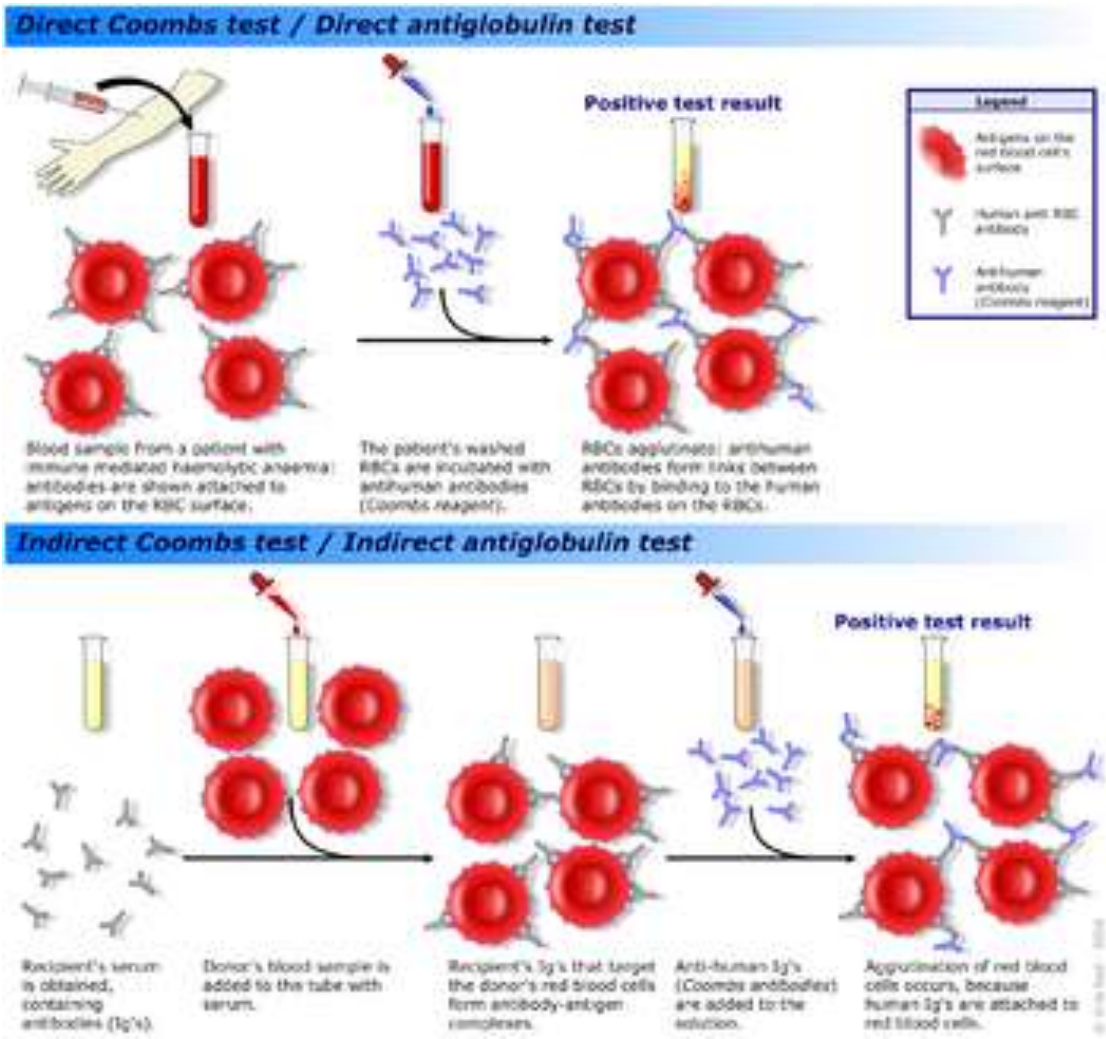
4 Complete a transfusion reaction report form.

5 After the initial investigation of the reaction, send the following to the blood bank for laboratory investigations:

- Blood samples (1 clotted and 1 anticoagulated: EDTA/Sequestrene) taken from the vein opposite the infusion site 12 hours and 24 hours after the start of the reaction
- Patient's 24-hour urine sample.

6 Record the results of the investigations in the patient's records for future follow-up, if required.

Coomb;s test



References: WHO guidelines of Clinical application of blood bank .