Transfusion Complications

Rudiments of Blood Transfusion
for IV. grade medical students

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Problems of Blood Transfusion

Technical problems

Harvay (1628) Circulatory

Devising of instruments, **problems of infections**

Hustin, Lewisohn (1914) Hemostasis

Blood collection in bottle (1940)

Serolgical incompatibility

Landsteiner (1900) **ABO blood group**

Wiener (1940) Rh blood group

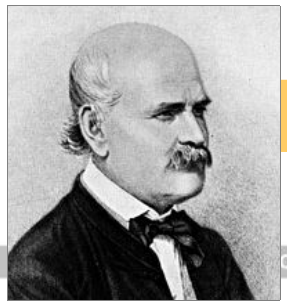
Other blood groups

Bacterial and viral contamination

Semmelweis (1847)

Sterile closed blood collection bag system (1963)

Virus inactivation of blood products

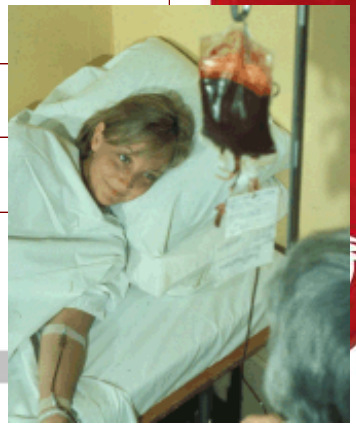


Ignác Fülöp **Semmelweis** (Hungary)

Figure 2. Blundell's Gravitator in use. Arguably the most famous image of transfusion's history in nineteenth-century Britain (from James Blundell, "Observations on Transfusion of Blood, with a Description of his Gravitator," The Lancet 2 [1828–29]: 321–24).

The most important symptoms of transfusion complications:

- hemolysis, hemoglobinuria
- fever, rigor, chills
- shortness of breath, dyspnoea
- hypotension, hypertension, tachycardia
- pain, malaise
- skin rash, angioedema
- preshock



Transfusion reactions can develop early or late after transfusion

I. Incompatibility

Immunisation, immune reactions

II. Properties of blood products

quality, quantity, administration technics

III. Pathogen agents

transmission of pathogens (virii, bacteria, protozoa)

I. Immune Complications

complications	causes
I. *In vivo antigen-antibody reactions*	
1. Hemolysis Immediate, intravasal (IgM)	**Antibodies against Red Cell antigens**
Late, majority of extravasal (IgG)	
2. Post-transfusion purpura	**Antibodies against Platelet antigen / Anti-HPA-1a or HLA class I /**
3. TRALI	**Antibodies against Granulocyte antigens / HLA or anti-HNA /**
4. Allergy, anaphylaxis	**Antibodies against Plasma Protein antigens**
II. *Immune cells in vivo effects*	
5. TA-GVDH	**Viable donor lymphocytes**
6. Immunomodulation	**Difference in white blood cells HLA antigens**

1. HAEMOLYTIC TRANSFUSION COMPLICATIONS due to blood group incompatibility

IgM antibodies

I. Intravascular haemolysis (within 24 hours)

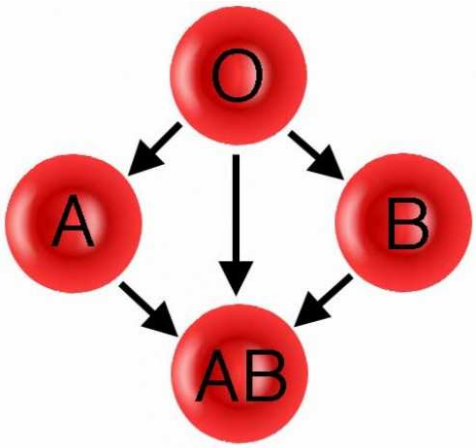
1. Antigen - antibody binding → **complement (C ') activation** - lysis
2. Ag-A + C komplex → activation of phagocytes
3. Release of inflammatory mediators and cytokines

The factors involved in the development of hemolysis:

1 ABO incompatibility - the presence, titer, termal amplitude of IgM antibodies
2 The volume of foreign blood (20 ml)
3 The blood group antigen type
4 Actual Complement rate and regeneration rate
5 The Ag-Ab-complenent 'complex formation

Donor	Recipiens	Mortality %
A	**O**	61
B/AB	O	20
A/AB	B	9
B	A	4,6
O plasma	A/AB	4,6
B plasma	AB	0,8

RBC ABO compatibility



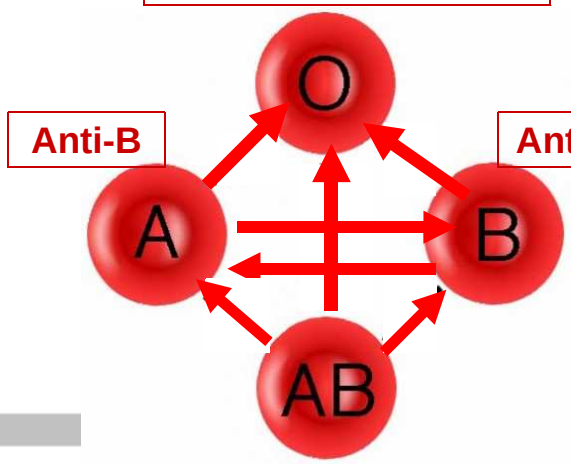
anti-A, anti-B present

ABO incompatibility

!

Clerical errors:

Patient identification
ABO testing



In vivo effects of antigen-antibody reaction:

1. Neuroendocrine response

Immune Complex - **activation of Hagemann factor** (F XII) - Bradykinin
Hypotension - catecholamines, epinephrine
Vasoconstriction (kidneys, intestines, lungs, skin)

damage of tissue oxygenation, kidney damage

2. Complement activation

C3-C5 (anaphylatoxins) release - mast cell and basophil degranulation
histamine release - eosinophil degranulation
platelet aggregation, release of hydrolytic enzymes from neutrophils
mast cell and basophil degranulation
cytokine release(TNF, IL-8, MCP, etc.) from monocytes

fever, hypotension, bronchospasm

3. Blood clotting activation

Hageman factor activation due to- Ag-Ab-C ' complex and RBC stroma
DIC - intravascular thrombus formation
- utilisation of Clotting factors and platelets
- Increased fibrinolysis

bleeding, shock

Symptoms:

- Chills and fever
- Hypotension
- Back Pain
- Tight chest pain
- Suffocation, cyanosis
- Fullness of neck veins
- **Burning and itching pain running along in the infused vein**
- Anxiety
- Renal impairment: oliguria, anuria (36%)
- Unusual bleeding (DIC!) (10%)
- Shock

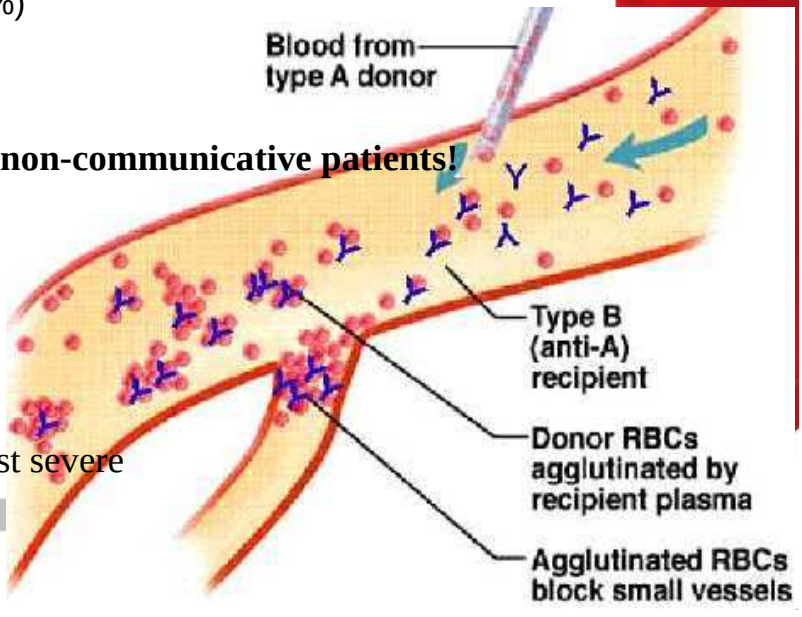
Laboratory findings:

• 1. haemoglobinaemia (Hb binding capacity of haptoglobin!)
• 2 LDH increase
• 3 hyperbilirubinemia
• 4 haptoglobin decrease
• 5 Urea, creatinine increased in patients with renal impairment
• haemoglobinuria

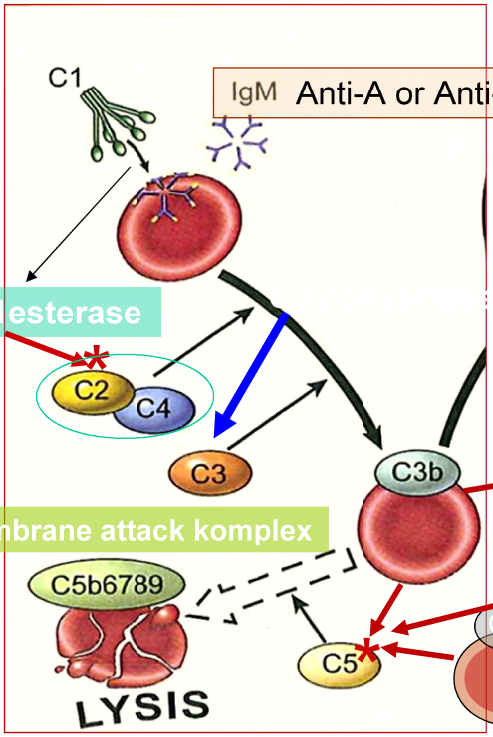
Symptoms in anesthetized, unconscious, non-communicative patients!

- diffuse bleeding in the surgical area
- hypotension

It could be caused by adminisrtation of
10 -15 ml incompatible blood
- ABO incompatibility is usually the most severe



Intravascular haemolysis



IBCT: Incorrect blood component transfused
ADU: Avoidable, delayed or undertransfusion
HSE: Handling & storage errors
Anti-D: Anti-D errors

ACUT STAGE

Figure 2: Cases reviewed in 2012

HSE	316	19.2%
ADU	145	8.8%
Anti-D	313	19.0%
IBCT	252	15.3%
62.3 %		
TA-GvHD	1	0.1%
TTI	3	0.2%
PTP	1	0.1%
CS	11	0.7%
UCT	8	0.5%
TAD	19	1.2%
TACO	82	5.0%
TRALI	11	0.7%
ALLO	69	4.2%
HTR	42	2.6%
ATR	372	22.6%
TOTAL	**1645**	**100.0%**

Treatment:

- **transfusion should be stopped immediately**
- At-Ag-reaction should be braked with Steroid
- **antishock terapy** – electrolytes, plasma substitutes - albumin
- restoration of tissue **oxygenation** - selected blood transfusion
- **Renal impairment management** - diuretics - hemodialysis (10-15%)
- **Fluid balance maintenance** – loss and intake rate
- **Metabolism recovery** - hyponatremia, hyperkalemia
- **DIC treatment**
- Exchange transfusion (in the first 12-24 hours)

Tasks:

- Check **data**
- **Consultation**
  - **Laboratory** tests – blood groups, serological investigation of complications, urinanalysis, free hemoglobin, renal function tests, coagulation tests, LDH, Hp
- **Sepsis** investigation
- Continue **monitoring** of patient

II. Delayed extravascular hemolysis (5-10 days after transfusion)

- mostly occurs as a result of secondary immunization **IgG antibody**

The antigen - antibody reaction consequences:

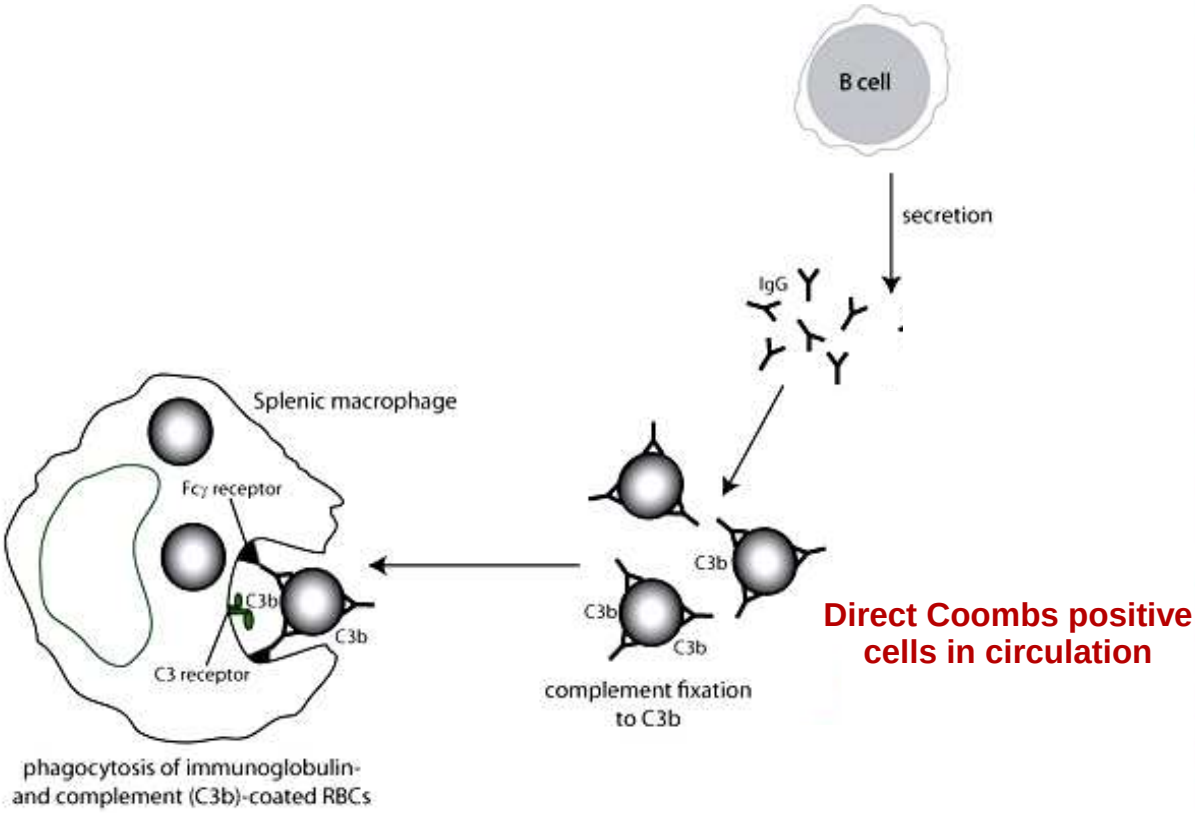
1 **C 'activation-**depends on subclasses of IgG antibody (**IgG3, IgG1,** IgG2, IgG4)
2 **Extravascular lysis** - Immune Complex - macrophage activation
3 Phagocytosis – fragmentation - lysis - **release of cytokines** (IL-1, IL-6,TNF,IL-8)
- ADCC (antibody dependent cellular cytotoxicity)

Influencing factors:

- The actual amount of the antibody
- The individual immunoglobulin synthesis rate
  - The current saturation of the phagocytic cell receptors
- The blood group antigen type
  - The amount of transfused incompatible blood

rarely fatal

Extravascular haemolysis



Direct Coombs positive cells in circulation

Symptoms: (from 24 hours to 3 weeks)

- Fever
- **Ineffectiveness of transfusion**
- Hemolysis, hemoglobin decrease, icterus, hemoglobinuria
- Hypotension
- Renal impairment (6%) - treatment necessary only for these cases

- May be asymptomatic - Late serological transfusion reaction

Laboratory findings:

- **Positive Direct Coombs** – antibody-coated red blood cells
- Antibody appearance or sudden increase

A history of previous immunizations

Therapy:

- **generally not necessary**
- close monitoring

The antibodies involved in hemolytic transfusion reactions and types of hemolytic transfusion reactions

Blood group system	AHTR (intravascular)	Delayed HTR (extravascular)	
ABO,H	A,B,H		
Rh		***all types***	34,4%
Kell	K	K,k,Kp^{a+b},Js^{a+b}	13,3%
Kidd	Jk^{a}	Jk^{a+b+3}	30,0%
Duffy		Fy^{a+b}	14,4%
MNS		M,S,s,U	4,4%
Lutheran		Lu^{b}	
Lewis	Le^{a}		
Vel		Vel	other 3,3%
Colton		Co^{a+b}	
Dombrock		Do^{a+b}	

Other acute intravasal hemolysis

Immune hemolysis

- ABO incompatible **plasma** transfusions
- **AIHA** patients transfusion
- **Cold agglutinin** disease

Non-immune haemolysis

- Red blood cell **enzyme defects**
- **Infections**
- **Drugs**
- **Diseases associated with hemolysis**
  (PNH, microangiopathic hemolytic anemia)
- **Haemolytic blood transfusion**

2. FEBRILE REACTIONS

- **Haemolysis** - blood group incompatibility
  - bacterial contamination (endotoxin, cell debris)
- **No Haemolysis - NHFTR** – **non haemolytic febrile transfusion reactions**
  - **Infection** (malaria)
  - **Other transfusion independent reason**

1. Non haemolytic febrile transfusion reactions

Cause: white blood cell content of blood products – **cytokine effect**

Symptoms: fever (during or after transfusion temperature increases **≥1.5 ℃)**
flushing
tachycardia
shaking, chills

Occurrence: (6.8%) RBC products – to immunized patients
(37.5%) platelet products – to non-immunized patients

common complication - 0,5 - 6%

1. NHFTR – CAUSING FACTORS:

- antibodies in the recipient serum
  - HLA antibodies
  - Anti-granulocyte antibodies
  - Anti-platelet antibodies
- stored PLT products
  - destroyed granulocytes

Ag-At-C' complex

↓

macrophag activation in recipient

↓

Release of pyrogens

(TNF-α, IL-1, IL-6)

Treatment:

- mild: interrupt the transfusion - antipyretic –
- severe: - antipyretic - differential diagnosis !

Unit causing complications should not be administered.!

Prevention: - **removal of white blood cells before blood product storage** (removal buffy coat, filtration)

2. PTP – post transfusion purpura

Anti-platelet antibody

acut complication – **one weak after transfusion**

Prior immunization - especially women

Cause: 80-90% **anti-HPA-1a** other: anti- HPA-1b, -3a, -4a, -5b

Symptoms:
- bleeding -severe thrombocytopenia -!
- fever - NHFTR (+ anti-HLA antibodies)

Differential diagnosis: ITP, drug induced trombocytopenia, TTP, DIC

Treatment:
- immediately!
- high-dose IVIG (2g/ kg bw for 2-5 days)
- steroid
- plasma exchange
- blood products (RBC or PLT) only from antigen negative donor!

After PLT administration both administered and own PLT destruction occur! Reasons: donor HPA-1a antigen or recipient Ag-Ab complex binding to the recipient's platele or cross-reactive antibody production

3. TRALI - transfusion related acut lung injury

severe acute reaction within 6 hours

Anti-granulocyte antibody

Cause: - anti – granulocyte antibodies (HLA/HNA)

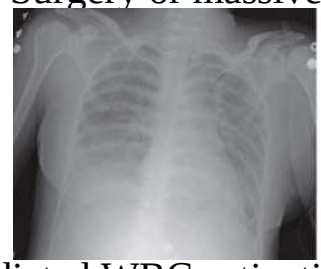
- often in blood products (*multipara women plasma*)
- rarely in recipient's serum

Symptoms:

- Dyspnea (respiratory distress)
- Severe hypoxia, cyanosis, hypotension
- Severe bilateral pulmonary edema
- Fever

Predisposing factors:

- Active infection
- Cytokine therapy
- Surgery or massive transfusion



Factors responsible for developing TRALI

Neutrophyl activation

- Ab-Ag komplex – leucoembolus – C'mediated WBC activation
  – pulmonar endothelial damage
- leukocyte activation in blood components during storage

Therapy: - respiratory support immediately – **mechanical ventilation**

- steroid

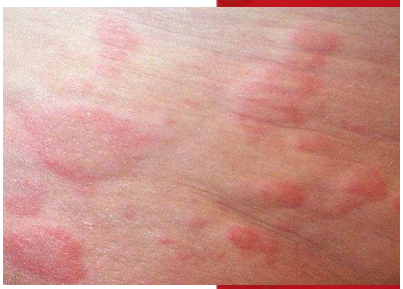
4. Allergy, anaphylaxis: - acut reation /may be life threatening

Etiology: **antibody against donor blood proteins**/ IgA content!
transfusing of allergens nutrients, drugs (Aspirin, ACE inhibitor)
passive transfer of **IgE** (to drugs, food), or complement

Symptoms:

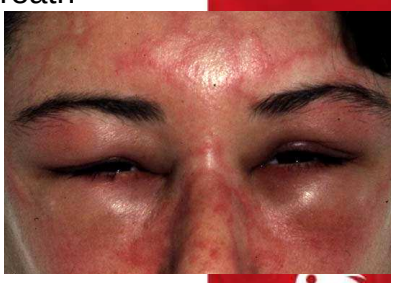
Mild reactions - malaise
- Itchy, burning red spots / neck, thorax /
- local urticaria
- Low-grade fever, fever

The transfusion could be continued after treatment



Sever reactions - Swollen mucosa / laryngeal edema - shortness of breath
- Anaphylactic shock

The transfusion should be stoped



Treatment: - antipyretic, fluid replacement
- antihistamines, Ca- preparations
- Steroids (Cortisone, Prednisolone)
- Epinephrine (Adrenaline)

Prophylaxis: - IgA-free blood to IgA deficient patient
- No (or IgA deficient) plasma transfusion
- **washed blood products**

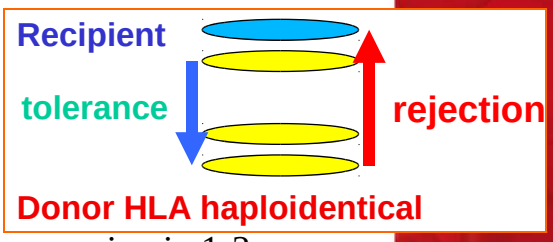
5. TA-Graft versus host reaction

few cases, high mortality >90%

complex immune process which is caused by immunocompetent donor lymphocytes against immunocompromised or immunocompetent recipient

Etiology: transfusion of haploidentical blood products

blood transfusion from relatives



Symptoms: Fever, rash, liver dysfunction, diarrhea commencing in 1-2 weeks post-transfusion followed by pancytopenia later

Risk factors: Any condition with impaired cellular immunity, or not developed immunological competence / premature babies and newborns/

- transplantation, leukaemia, lymphoma

- intrauterin transfusion, exchange transfusion, extracorporeal circulation

Therapy: **Largely ineffective**
immunosuppressive therapy, high dose steroids?

Prevention: For patients at risk (e.g., imunocompromised patients), it is critical to **irradiate cellular blood components** (RBC and platelets).

6. Transfusion-related immunomodulation (TRIM)

Transient immunosuppression

Etiology: **Allogeneic leucocyte-containing RBC transfusions**

the presence of foreign HLA class II. antigens
(the role of HLA DR 3 is suspected)

Cellular effects:

Decreased *T helper reaction*
Increased *T cell suppressor activity*
Increased *B cell antibody production*
Impaired NK cell function
Defective antigen presentation

Clinical signs:

reduced graft rejection
decreased recidive in Crohn's patients
increased risk of cancer recurrence
increased postoperative infection rate
potential risk of tumorous disease in adult age

Prophylaxis: **leucodepletion of blood products** in question

II. Early non immune complications

Complication	Etiology
Heart failure	volume overload / Whole blood, FFP /
High fever and shock	bacterial infection
hypothermia	Too rapid administration of cold blood / Massive transfusion /
Hemolysis	physical or chemical damage of the the blood administered
air embolism	Transfusion uder uncontrolled high pressure or priming
Hypocalcemia	**Massive transfusion of citrate-containing blood products / plasma ! /**
Hyperkalemia	massive transfusion of **old blood**

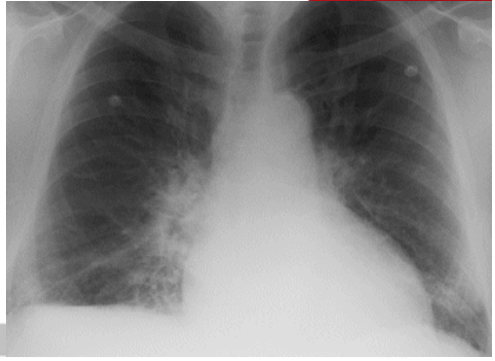
1. Transfusion-related circulatory overload (TACO)

may develop within 1 to 2 hours of transfusion

Symptoms: acut pulmonary oedema
(dispnoea, cyanosis, head ache, hypertension, heart failure)

Frequency: about 1% children and elderly patients
cardiac and/or pulmonary decompensation
chronic anemia (plasma)
chronic renal failure

Ethiology: - high volume transfusion (whole blood, plasma)
- high (20-25%) concentration albumin infusion
- rapid or massive transfusion



Therapy: Stop transfusion immediately
upright position , diuretics, oxygen

Prevention:

Slow rate transfusion!

Normal urine output 0,5 – 2 ml/min

2. Massive transfusion syndrome

Mortality about 60%

Transfusion of severe shock patients
(10-15 U blood in 24 hours or replacement of 1 blood volume(TBV))

Symptoms:
bleeding - dilution and consumption of platelets and clotting factors(DIC)
severe hypoxia in tissues

Multiplex complications: Coagulation, biochemistry (hypocalcaemia, hyperkalaemia), acid base abnormality, hypothermia

Therapy: fluid replacement, blood (fresh warmed blood!), cardiac support

3. Cold blood transfusion

Decrease in tissue oxygenation

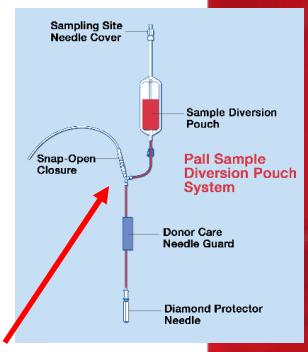
Symptoms: ventricular arrhythmias
impaired blood coagulation
worsen of hypokalcemia and hyperkalaemia symptoms
peripheral vasoconstriction
increased calorie need

Prevention: Use of blood warmer

4. Transfusion of infected blood: rare

/ mortality of 80-100% /

Sources of infection:
- **donor** arm or donor granulocytes
- poor **venipuncture** technique - foamy blood
- **storage** temperature, inappropriate storage
- **opened** blood bag, not cleaned **water bath**



Signs in blood product:
hemolysis, clots,cloudy plasma - white-gray precipitate, bacterial or fungal colonies on surface

Prevention: donor skin desinfection, removal of first aliquot of donor blood
good product collecting and manufacturing (**closed system**!)
controlled blood product storage
opened products management to appropriate standards

Symptoms: fever, chills, RR decrease, **severe rapid shock**, DIC, **intavascular hemolysis**, heart, liver, kidney failure

Treatment: **stop transfusion** immediately
shock therapy, resuscitation
i.v. broad-spectrum antibiotics

Bacteriological examination

blood culture test of blood product and patient blood samples

5. Transfusion of haemolytic blood:

several liters of old stored blood conteins harmful amount of hemoglobin

- large amounts of Hb appears as a cylinder in renal tubular causing renal failure
  / Renal disease patients, shock, dehydration /

Reasons for the development of hemolysis in blood product:

- **Outdated RBCs**
- **Drugs** or infusion solutions mixing with blood product.
- **Thermal effects** - Heat or freezing (temperature above 38°C)
- **Bacterial** contamination
- **Mechanical** damage - shaking, harsh handling and transport
  (Thin needle, artificial heart valves, extracorporeal circulation, high pressure transfusion, etc.).

Prevention: - high quality blood products
- considering of transfusion indication

Treatment: - **remove Hb** / infusions, diuretics /
- Urine alkalinisation
- **desferroxamin**

6. Air embolism:

very rare since using plastc blood bags
the foamy blood is transferred into right ventricle

Causes:

- inadequate priming of transfusion set
- transfusion with overpressure

Symptoms: Cough • Dyspnea • Chest pain • Shock

Prevention: the appropriate use of technology

Treatment: : ➢ Laying the patient on the **left side**
- Rhythmic **compression of the chest**
- Suction of the frothy blood with catheter
- resuscitation

7. Citrate intoxication

massive transfusion with plasma
Infants, patients with heart disease or liver disease

Symptoms: - Neuromuscular disorders / **tetany**
- Cardiac arrhythmia

Treatment: **Ca support**

8. Transfusion of hyperkalemic blood:

High risk in hyperkalemic conditions / uremia, heart disease, massive transfusion, acidosis / or in infants

Symptoms: arrhythmia, cardiac arrest

Prevention:
- exchange transfusion with blood less than 7 days
- massive blood transfusion with blood less than 10 days
- RBC washing
- use of in-line potassium filters

Treatment:
- 10% NaCl, NaHCO3 or Ca composition
- Hypertonic glucose / + **insulin**/
- Ion exchange resin / Resonium /
- **Dialysis**, hemofiltration

III. Infection transmission

Non immune late transfusion complications

Complications	*Causes*
Hemosiderosis	**Politransfusion / > 100 U vvs /**
Hepatitis	**HBV /±DELTA/, HCV, HGV/?/, HAV, HEV,CMV**
AIDS	**HIV-I, HIV -II / after years? /**
CLL /adult T-cell/	**HTLV -I**
TSP tropical spasticus paraparesis	**HTLV-II (human T lymphotrope virus)**
Zoonosis	**Malaria, kala-azar, babesiosis**
Syphilis	**Treponema Pallida**
Aplastic anaemia	**Parvovírus B 19**
Fetal damage	**CMV**

1. Hemosiderosis:

accumulation of iron in organs

1U blood transfusion - 200 mg iron intake

Cause: 50 - 100 U RBC transfusion
transfusion of large amount hemolyzed blood

Symptoms: RES – organs failure - heart, liver, endocrin organs

bronze skin, liver cirrhosis, heart failure

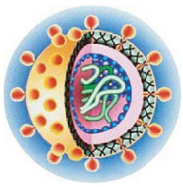
Treatment:

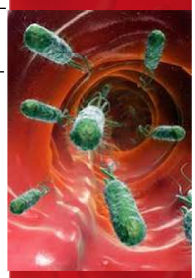
chelation therapy - iron removal
desferoxamine, deferiprone, deferasirox

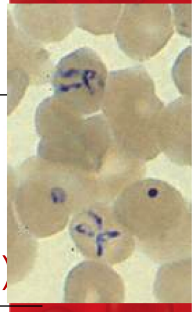
exchange transfusion
phlebotomy

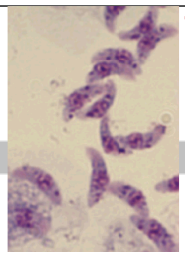
2. Transmissible pathogenic agents with the different blood fractions

Blood Fraction	Pathogens		
	Virus	**Bacteria**	**Protozoa**
Plasma	Hepatitis A virus Hepatitis B virus Hepatitis C virus Hepatitis D virus Hepatitis G virus[1] HIV Parvovirus B 19 (Prions)	*Treponema pallidum (syphilis)*	
Red blood cells			Plasmodium (malaria) Babesia microti (babesiosis)
White blood cells	HIV I and II Epstein Barr vírus Cytomegalovirus virii as with plasma		Toxoplasma gondii (toxoplasmosis)









Risks of transfusion-transmissible infection

Agent and testing standard	Window period	Estimate of residual risk 'per unit' (a)
HIV (antibody + NAT)	5.6 days	Less than 1 in 1 million
HCV (antibody + NAT)	3.1 days	Less than 1 in 1 million
HBV (HBsAg + NAT)	23.9 days	Approximately 1 in 538,000
HTLV 1 & 2 (antibody)	51 days	Less than 1 in 1 million
vCJD [No testing]		Possible
Malaria (antibody)	7–14 days	Less than 1 in 1 million

Australian data

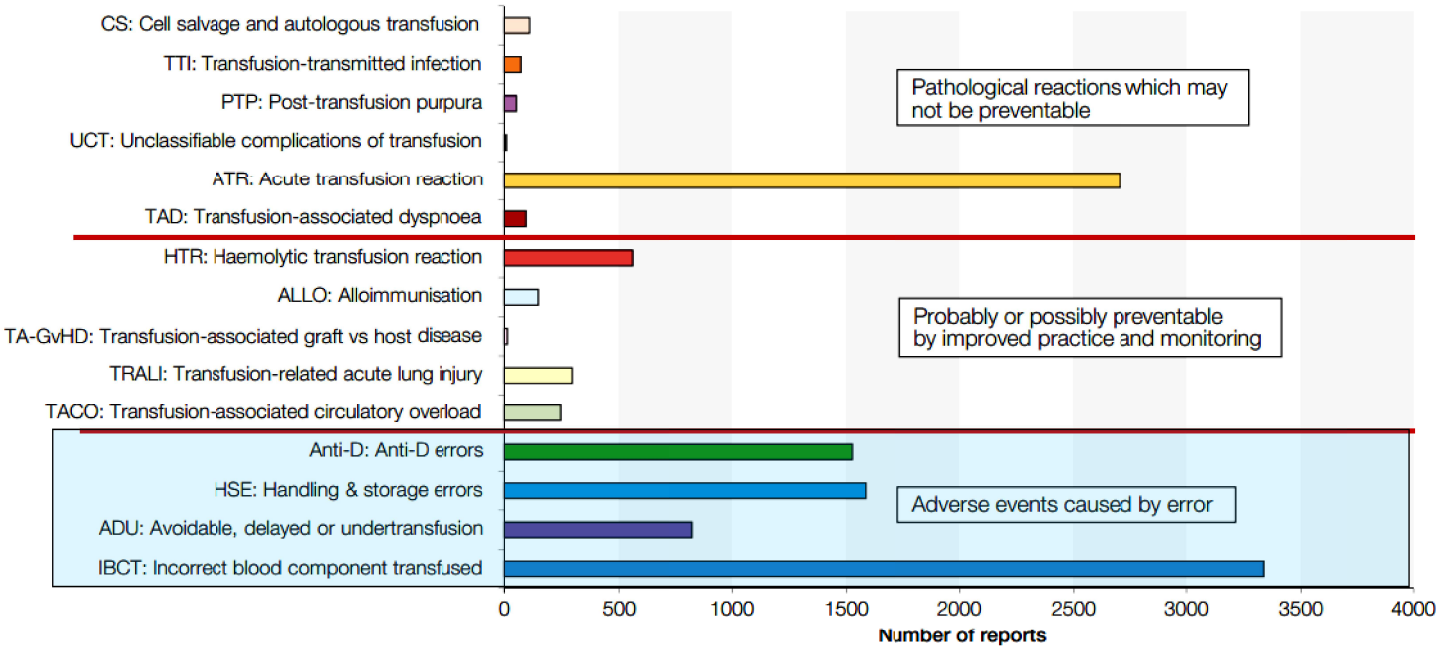
Estimated risk of infection from transfusion in the UK

(Public Health England, 2013) and risk of major morbidity or death (all causes) from transfusion based on SHOT data for 2012 (Bolton-Maggs *et al*, 2013a).

Category	Risk per million donations [95% confidence interval] for viral infections, and per million components issued for SHOT data	Reciprocal expression of same risks, 1 per number of components issued
Major morbidity	46·7 (all causes)	1 in 21 413
Death	3·1 (all causes)	1 in 322 580
Hepatitis B	0·76 [0·22–1·61]	1 in 1·3 million
Hepatitis C	0·036 [0·015–0·07]	1 in 28 million
HIV	0·15 [0·09–0·32]	1 in 6·7 million

Reported transfusion adverse events

Figure 1: Cumulative data for SHOT categories 1996/7–2012 (n=11,570)



CONTACT DETAILS
SHOT Office, Manchester Blood Centre,

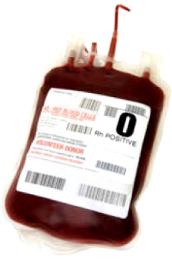
Transfusion related death

reported to the FDA 2008–2012 (US Food and Drug Administration, 2013).

Complication	Total (*n*)	%
Transfusion-related acute lung injury	74	37
Haemolytic transfusion reactions (non-ABO)	31	16
Haemolytic transfusion reactions (ABO)	22	11
Microbial infection	21	11
Transfusion-associated circulatory overload	35	18
Anaphylaxis	12	6
Other	3	1
	198	100

Estimated risk per transfused blood components

HIV	1 in 1 467 000
HCV	1 in 1,149,000
HBV	1 in 282,000 to 1 in 357,000
Haemolysis (death)	1 in 1 250 000



Transmissible pathogens in the stored donor's blood

Sepsis by bacteria transfer :

Endotoxin formation is during storage!

RBC transfusion:

Yersinia enterocolitica (51%) **+ 4C°**
Pseudomonas fluorescens (26,5%)
Treponema pallidum (4,1%)
Pseudomonas putida (4,1%

PLT transfusion: (storage: **+20 C°**)

Staphylococcus epidermidis (25%)
Salmonella coholerae-suis (13,5%)
Serratia marcescens (9,6%)
Staphylococcus aureus (5,8%)
Bacillus cereus (5,8%)
Streptococcus viridans (3,8%)

Virus transmission:

Problems: **- new mutants and new virii - expansion of vector-borne diseases –** dengue fever, chikungunya, WNV

- Screening tests do not detect fresh infection

- Virus inactivation procedures are at experimental state for labile blood products or not available for all countries

- prions

Hemovigilance

is a "quality process" which aims to improve quality and increase safety of blood transfusion, by surveying all activities of the blood transfusion chain, from donors to recipients. Haemovigilance means a set of organised surveillance procedures relating to serious adverse or unexpected events or reactions in donors or recipients, and the epidemiological follow-up of donors incluing obligation of adverse events reporting.

Deaths due to transfusion complications

USA: 1511/ 19 230 000 /year = 7 / 100 000

Hungary: 1 / 100 000 transfusion

Reporting institution? Transfused blood?

Distribution of transfusion complications

Cause of complications	SHOT (n=169)	Pécsi RVK (n=134)
Wrong blood group	47%	59%
Acut transfusion reaction	13%	18%
Late transfusion reaction	13%	16%
PTP	1%	0,8%
GVHD	1%	0
TRALI (or respiratory symptoms)	7%	6%

Summary: Types of transfusion complications

Immediate complications

Within 10 – 15 minutes

ABO – incompatibility
Anaphilaxis
Air embolism

Late complications

1 – 7 after transfusion

Delayed immunohemolysis
Immunisation
Immunodeficiency
TA-Graft versus host disease
Hemosiderosis (months, years)

Early complications

Within 1 – 24 hours

Allergy
Febrile non-hemolytic complications
Haemolytic complications of immunised patients
Haemolytic complication of anesthetized patients
Circulatory overload
Citrate intoxication
Endotoxin shock
Hypothermia
Coagulation disorders
Trombembolia

Weeks, month, years after transfusion

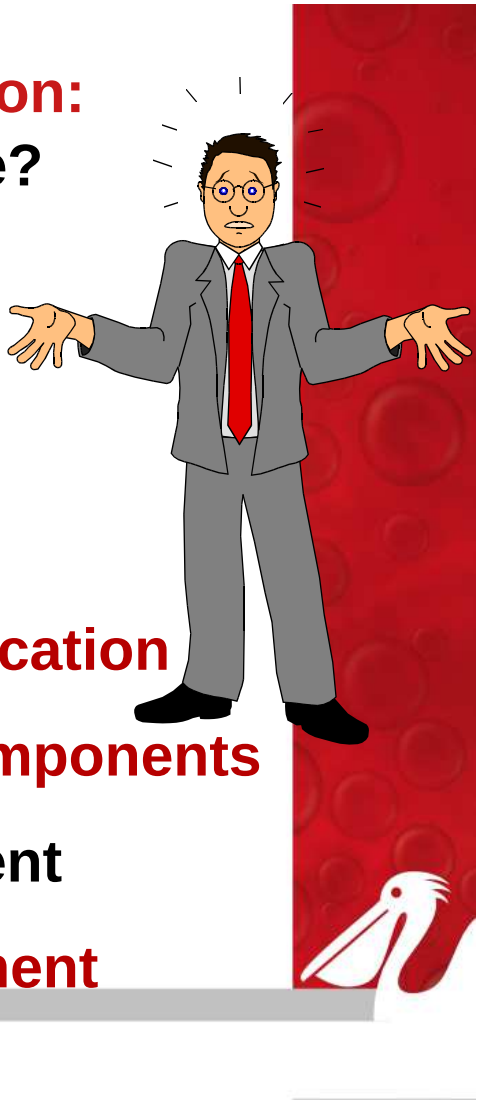
Transmission of pathogens

Hepatitis (B,C stb.) CMV HIV and other **virii** (EBV, Parvovísus B19 ….)
Lues and other **bakteria**
Malaria, babesiosis and other **protozoa**

The basic principles of transfusion:

HOW to transfuse?

Blood cannot be manufactured – it can only come from generous donors. The blood availability is limited and there are many risks of blood transfusion.



You should transfuse blood

1 never unnecessarily

2 if there is an appropriate indication

3 only the necessary blood components

4 effective amount of component

5 with prudent blood management