



SNS COLLEGE OF ALLIED HEALTH SCIENCES
SNS Kalvi Nagar, Coimbatore - 35
Affiliated to Dr MGR Medical University, Chennai



DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE
TECHNOLOGY

COURSE NAME: CPB & Perfusion Technology – II

TOPIC : Haematological Monitoring



HEMATOLOGICAL EFFECTS



CPB induces so many hematological problems;

- Exposure of patient's blood to the **circuit** & systemic heparinization
- **Dilution** of priming solutions
- Unneutralized **heparin** is a cause for post op bleeding
- Pt's with **pre-existing disease** that compromise coagulation or those on certain medicines such as aspirin



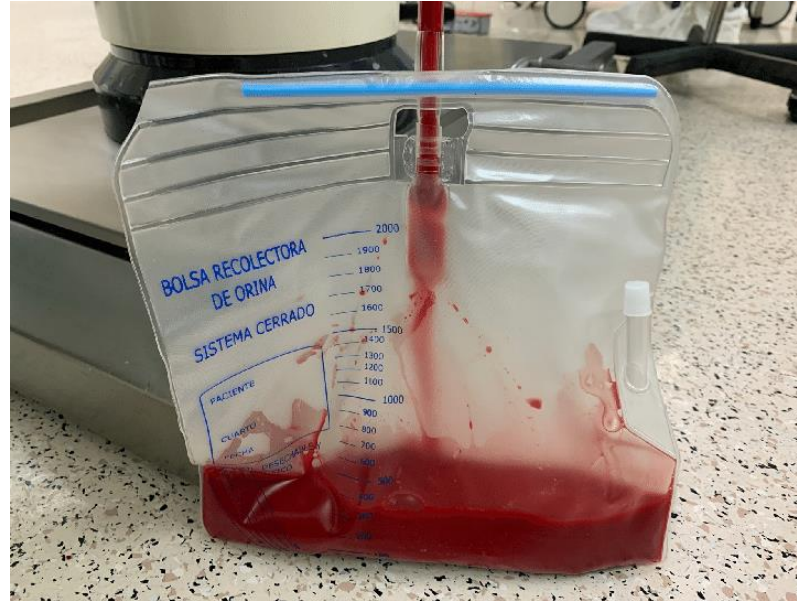


REASONS FOR USING BLOOD INTO THE CIRCUIT



- Cyanotic patient's
- Anemic patient's
- Pediatric patient's
- Hypovolemic patient's

- **Haematuria:** mild to moderate haemolysis may lead to haematuria.



Cyanotic Heart Defects

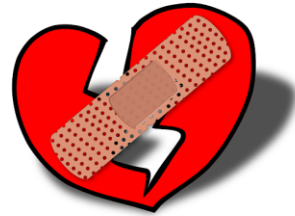
Truncus Arteriosus

Transposition of Great Arteries

Tricuspid Atresia

Tetralogy of Fallot

Total Anomalous Pulmonary Venous Return



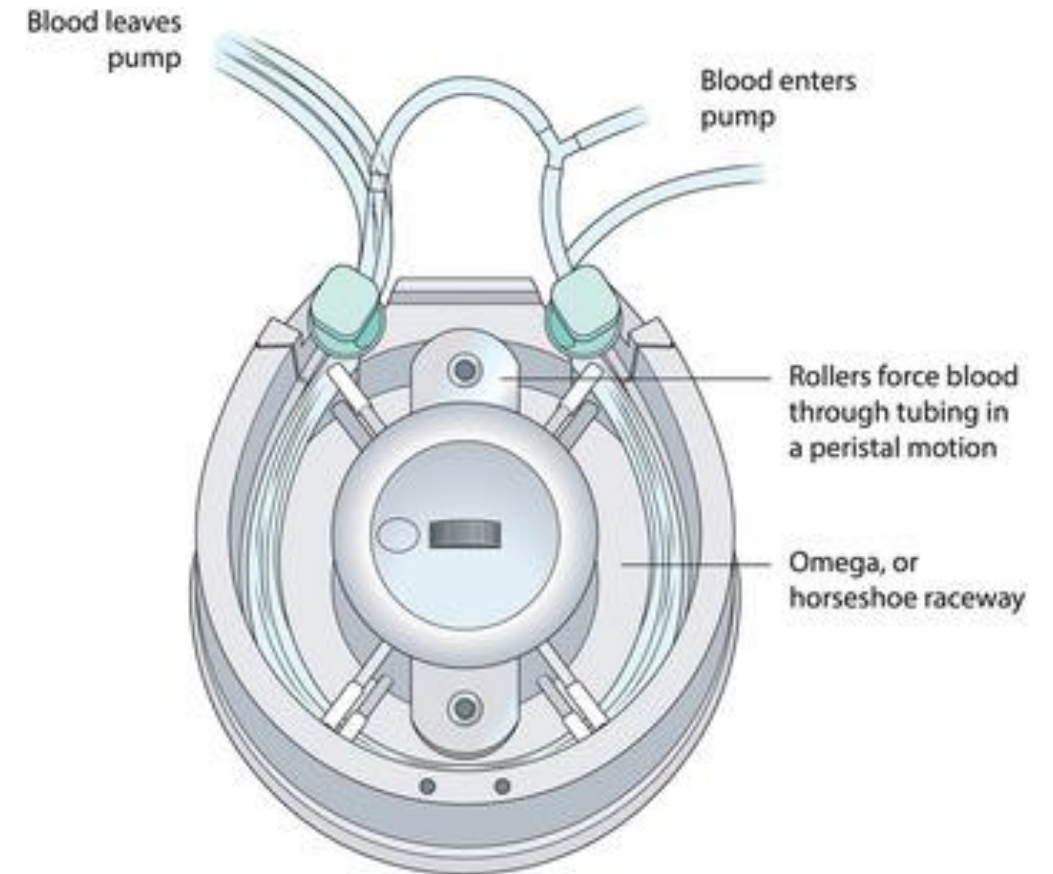


COMPLICATIONS ENCOUNTERED



Haemolysis

- RBCs are exposed to stronger sheer stress force
- Old blood usage
- Higher flows
- Tight occlusion
- More no. Of connectors
- High haematocrit





COMPLICATIONS ENCOUNTERED



Haemoglobinuria:

- Presence of **free Hb** in the urine which make the urine dark.
- It is the sign of abnormal condition such as bleeding & paroxysmal nocturnal haemoglobinuria
- Often associated with **haemolytic anaemia.**
- Causes:- **Sickle cell anaemia, transfusion reaction, pyelonephritis, acute glomerular nephritis.**

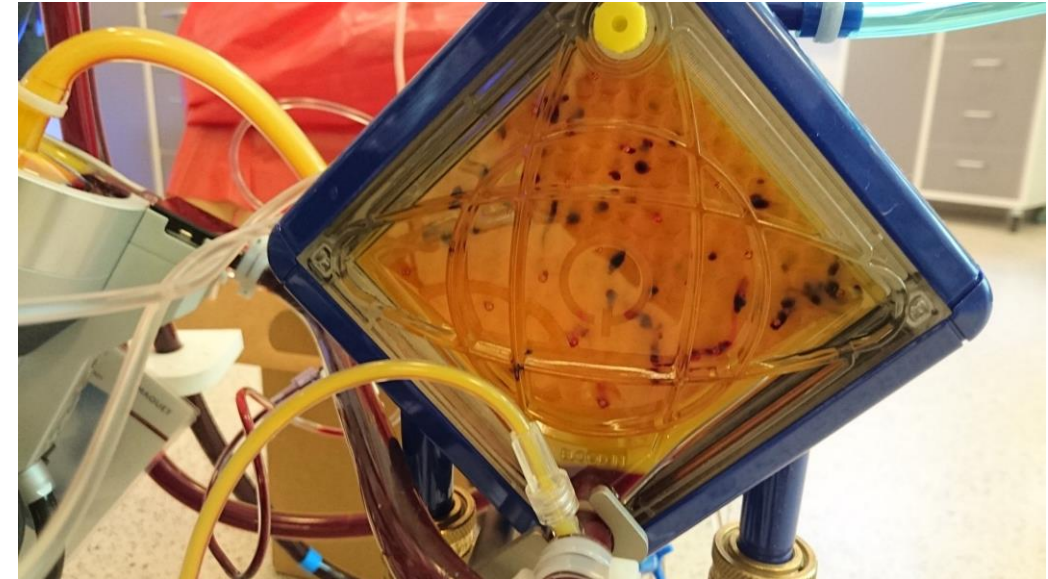




OTHER COMPLICATIONS

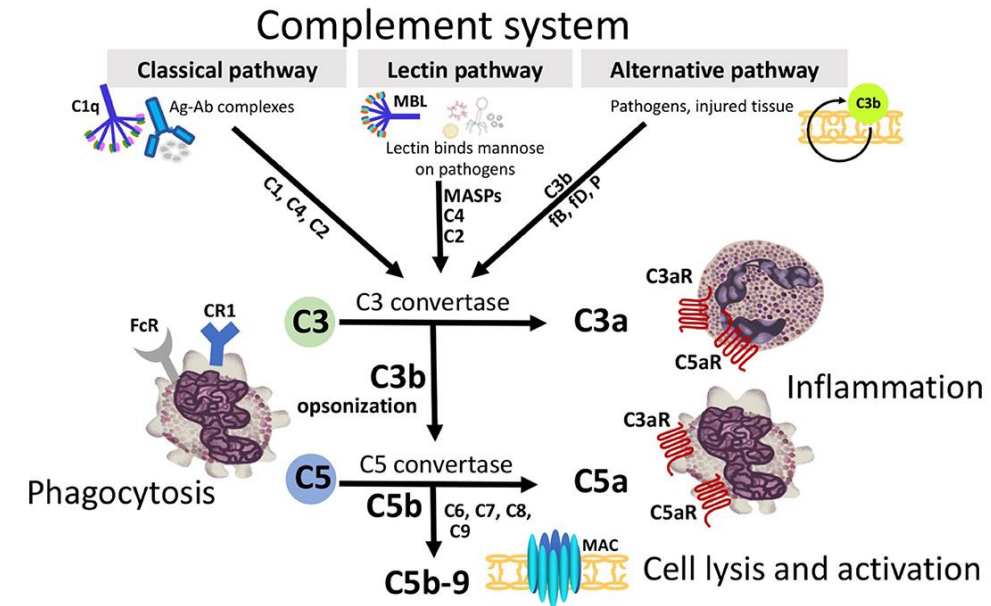


- **DIC:** occurs post bypass. coagulation factors of the patient become activated and bleeding occurs systemically.
- **Acid base imbalance:** hypocalcemia, hyperkalemia, hypomagnesemia
- Citrate toxicity
- **Oxygenator clotting:** due to blood priming or blood transfusion will activates the clotting factors.
- Improper heparinization of oxygenator
- Use of **un coated oxygenators**



COMPLIMENT & INFLAMMATORY RESPONSE

- **Contact activation** of blood by ECC triggers a series of amplification cascades mediated by proteolytic enzymes especially serine protease.
- It results in the activation of compliment, especially **C5 anaphylatoxin**.
- C5 is a potent bioactive molecule that mediates acute inflammatory response, which has spasmogenic & leukocyte activating properties to cause degranulation & **release of O₂ free radicals**.
- Compliment exposed neutrophils are stimulated to adhere to the surfaces & margination of BV & **leuko embolism**.
- Compliment activation & neutrophil arachidonic acid metabolites causes **increased vascular permeability with capillary leak**.

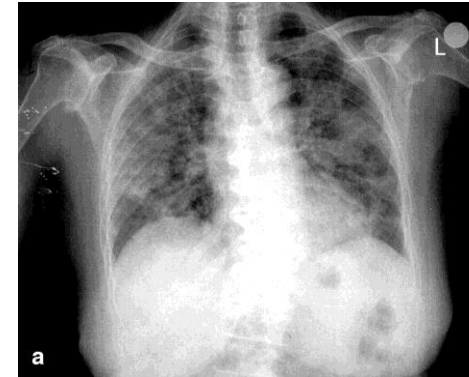




TRANSFUSION RELATED LUNG INJURY



- Reaction occurs when **donor plasma** contain antibodies that cause damage to the cells of lung.
- This usually occurs within **6 hours of receiving blood.**
- TRLI characterize by the acute onset of **non cardiogenic pulmonary edema.**
- TRLI is due to the presence of **leukocyte antibodies** in transfused plasma.
- Leuko-agglutination and pooling of granulocytes in the recipient lung may occur with release of leukocyte granules and resulting to injury to cell membrane endothelial surface and potentially to **lung parenchyma.**
- It results in **dyspnea, hypoxemia, hypotension**





TRANSFUSION RELATED RENAL INJURY



- Pt's with renal disease, cardiac surgery, CPB present difficulty primarily related to their **inability to excrete k+** & tendency to become fluid overloaded during peri-op period.
- Renal failure usually caused by **mismatched blood.**
- RBCs undergo **irreversible morphological & biochemical changes** during storage.
- As a result after transfusion they can promote a pro-inflammatory state, **impair tissue oxygen delivery** & exacerbate tissue oxidative stress.
- This in turn can cause AKI in susceptible pt's undergoing CPB such as those with pre existing kidney dysfunction & anemia





ASSESSMENT - I

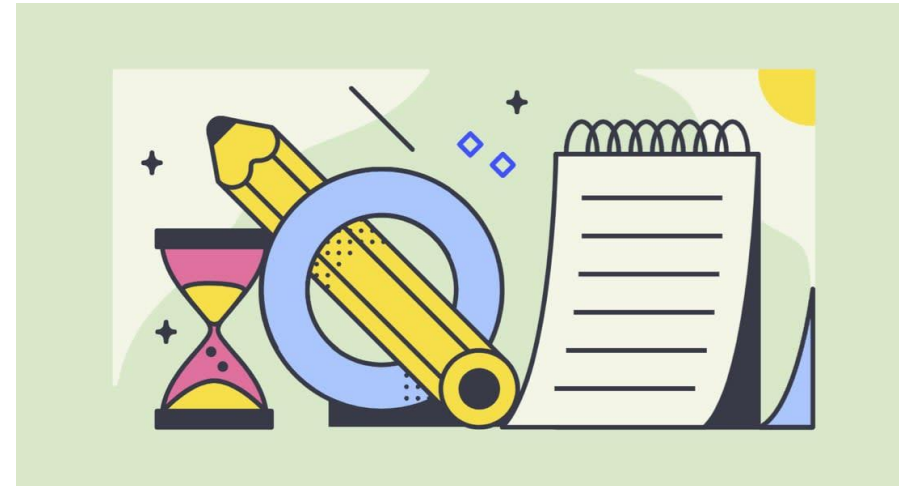


Causes of Hemolysis ?

What are the Anaphylatoxins?

What is TRALI?

When Hematuria Occurs?





JEHOVAH'S WITNESS



- This patient's who **refuse the blood transfusion** & reduce hemodilution with out use of banked blood.
- **Avoidance of homologous blood** transfusion is the goal when ever the CPB is used with this patient.
- **For this pt's we can go for :** low prime membrane oxygenator circuit, cell salvage, reinfusion of shed mediastinal blood, antegrade & retrograde autologous prime, use blood substituite.



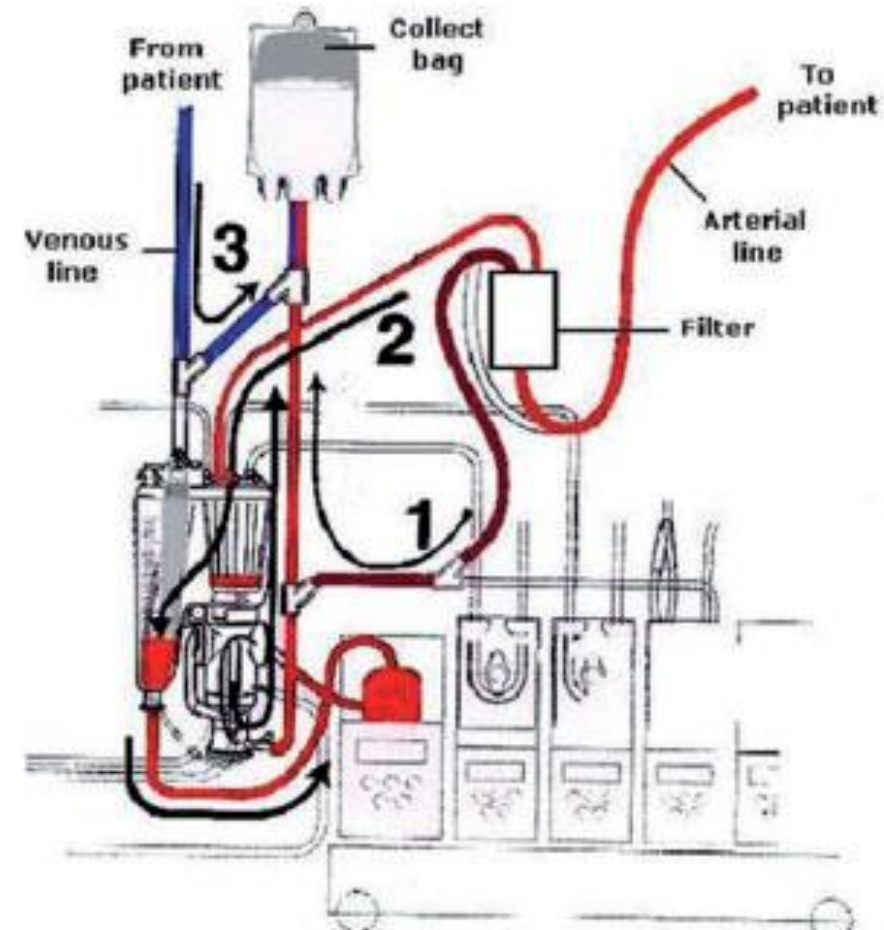


ANTEGRADE AUTOLOGUS PRIMING



This method replaces part of CPB prime volume with the pt's own blood thus reducing degree of hemodilution.

AAP utilizes **partial filling of venous reservoir** with pt's own blood from the venous limb of the CPB circuit on initiation of CPB, but before institution of CPB flows through the oxygenator & arterial limb of circuit



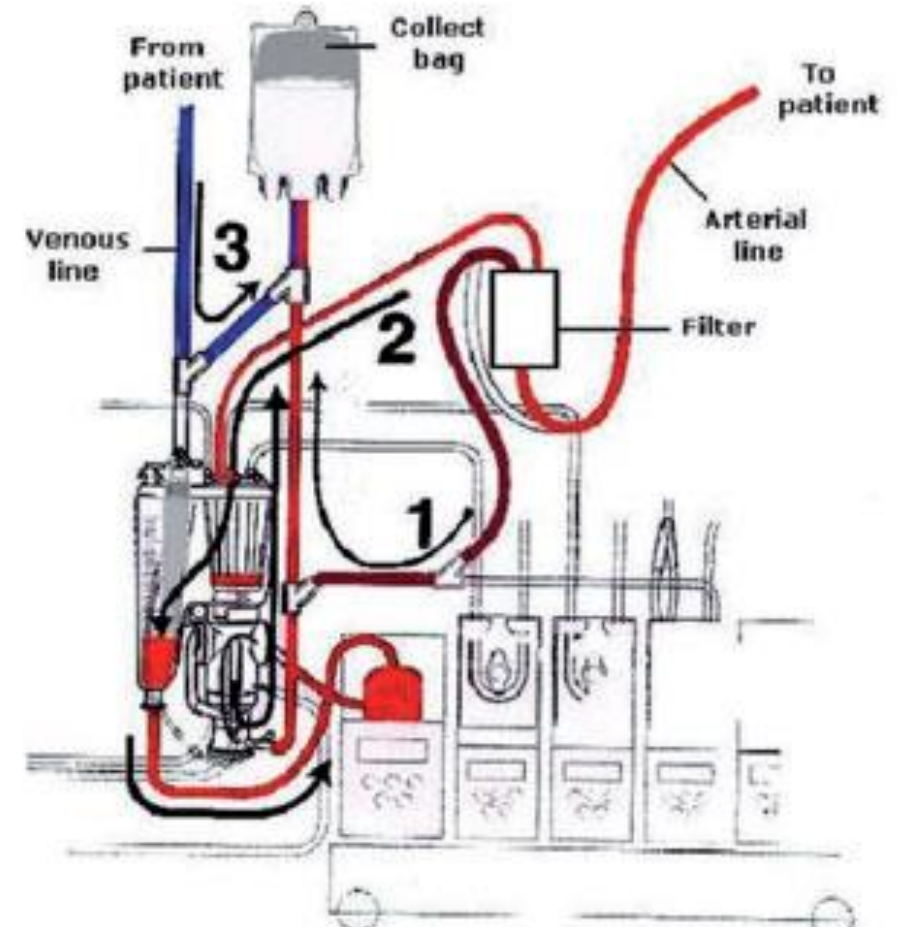


RETROGRADE AUTOLOGOUS PRIMING



It utilizes the retrograde filling of venous reservoir **via arterial limb of CPB circuit**, just prior to the initiation of CPB, displacing the crystalloid prime volume in the arterial line tubing, AF and oxygenator and so partially filling with pt's own blood.

Both the methods reduces the volume of crystalloid in the prime by replacing it with **400 to 500 ml** of patient's own blood.





BLOOD SUBSTITUTE



- It is also known as **artificial blood or blood surrogates**
- The substance **used to mimic** and full fill some function of blood.
- It aims to provide a **alternative to blood** and blood product transfusion.
- **Perfluorocarbons** are blood substitute in CPB.
- PFCs have a potential to **increase o2 content** of the perfusate and thus increase the capacity of HLM to deliver o2 to the body during CPB.
- **O2 solubility** of PFC increase significantly at low temperature
- Avoid risk of disease transmission & immunosuppression
- Address concern of **jehovah's witness**

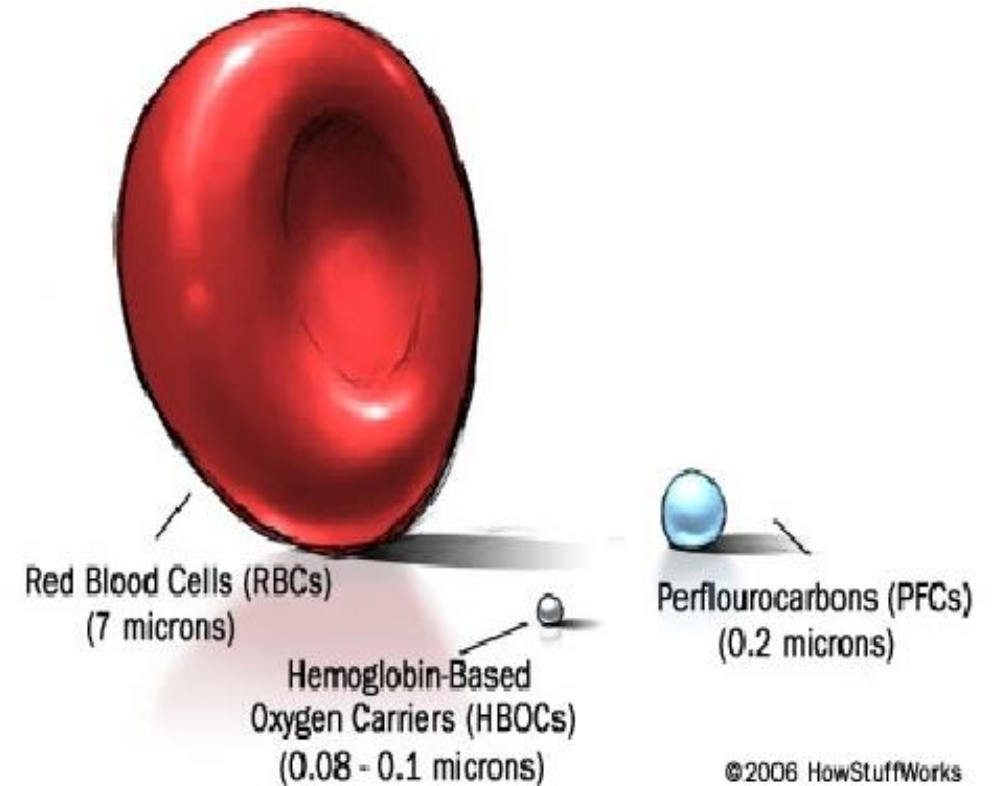




BLOOD SUBSTITUTE

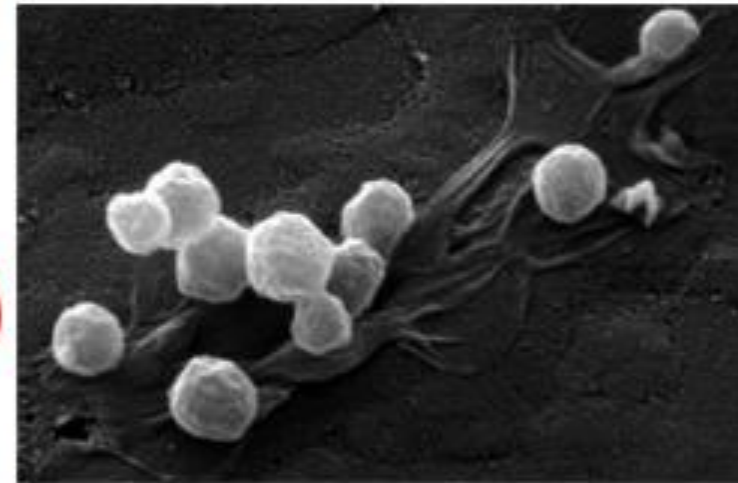
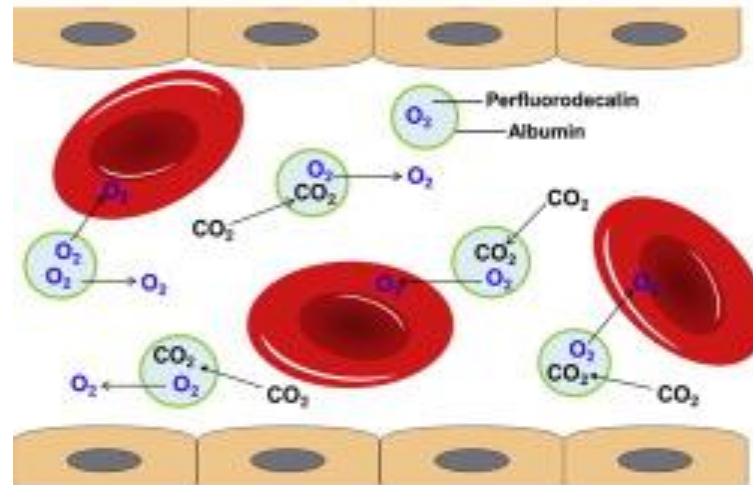


- PFCs to the pump prime solution may allow larger volumes of blood to be with drawn from the patient immediately prior to bypass for transfusion after bypass.
- **Lowering acceptable HCT** during CPB.
- O₂ solubility of PFC increase significantly at **low temperature**
- Avoid risk of **disease transmission** & immuno-suppression
- Address concern of **Jehovah's witness**



ASSESSMENT – II

- What is Autologous Priming?
- Types of Autologous Priming
- What is Perfluorocarbons?
- Why Perfluorocarbons is not used clinically?





CRYSTALLOID PRIME



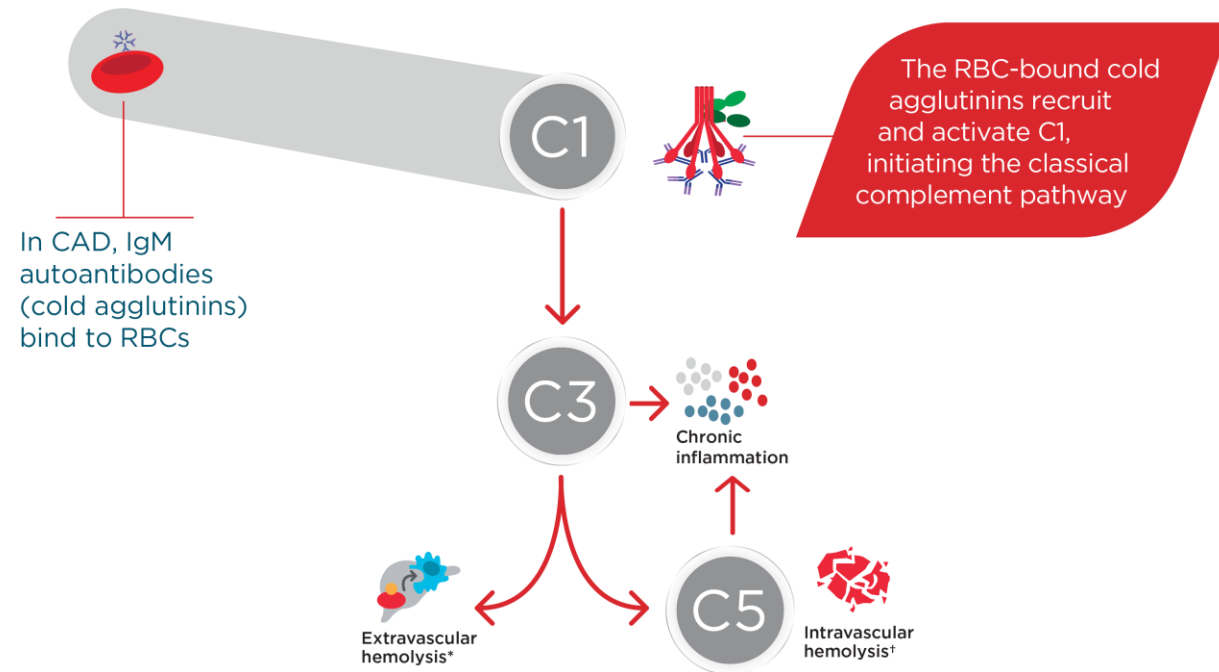
Solution composed of electrolyte & water & the principle component is Na+Adv

- Used to hemodilute the patient
 - Volume expander
 - Mimics plasma electrolytes
 - Expands interstitial volume
 - Reduces the oncotic pressure
- example: RL, PLASMALYTE, NS, MANNITOL.



COLD AGGLUTINININS

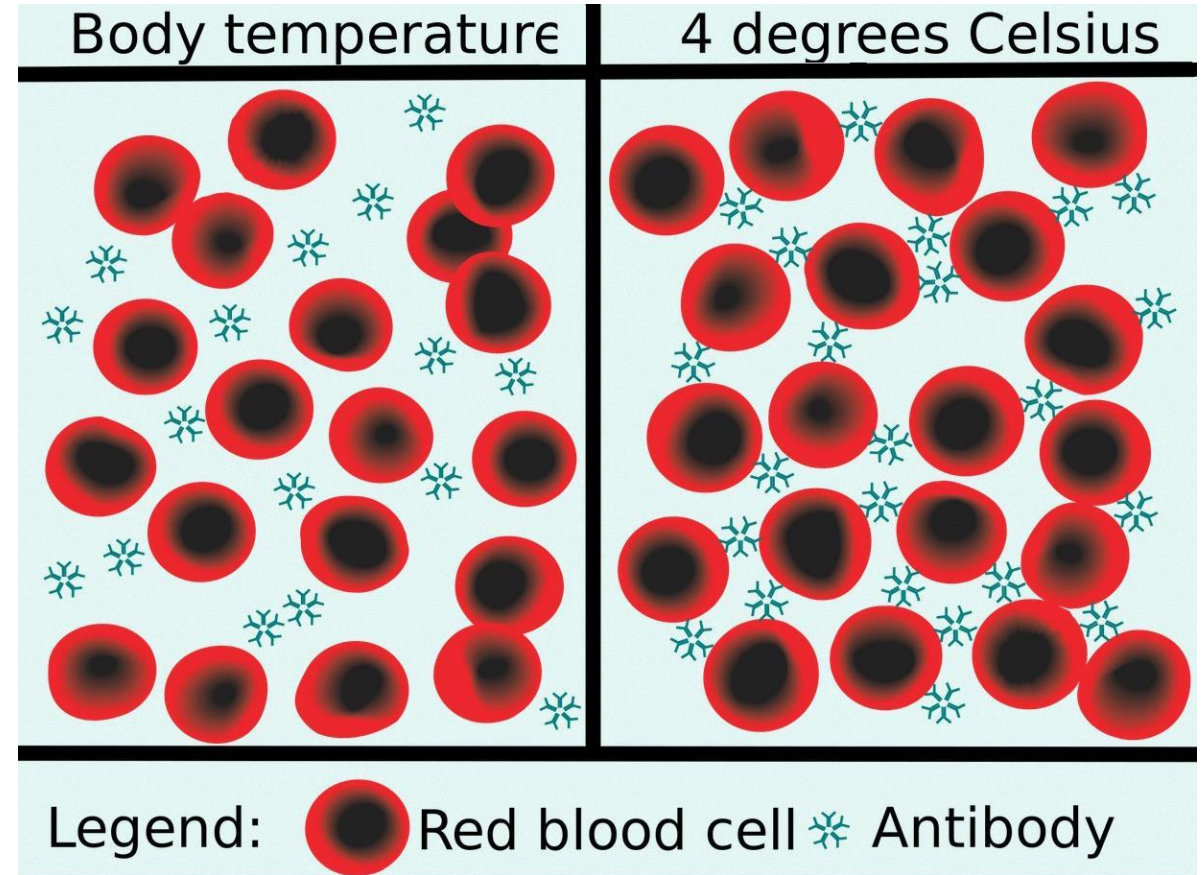
- Cold agglutinin disease is a rare type of **autoimmune hemolytic anemia** in which the body's immune system mistakenly attacks and destroys its own red blood cells
- This may cause complement activation & RBCs to clump at low temp and haemolyzed.
- **Immunoglobulin M (IgM) autoantibodies** to red blood cells
- **It does not occur if patient kept at warm temp.**



MANAGEMENT OF COLD AGGLUTINININS

MANAGEMENT IN CPB:

- Patient should be kept warm before, during, after bypass.
- A heating table on the or table
- **Normothermic bypass with warm prime**
- CP should be started warm to flush the coronaries and then switched over to cold after 200 to 300 ml
- For proper myocardial protection continuous Retrograde CP given with intermittent Antegrade CP
- A warm infusion of CP just before ACC release.





SICKLE CELL ANEMIA



- It is one of a group of inherited disorders
- Abnormal red cell that is crescent shaped caused by **HbS - an abnormal Hb.**
- The cells move slowly, clump together and hemolysed.
- It inhibits the ability of hemoglobin in red blood cells to carry oxygen
- In environment of **low oxygen and low temperature** the sickling takes places.
- Patient's have blood clots, anemia, occluded blood vessels, pain, shortness of breath and convulsions.



Normal Red Blood Cell

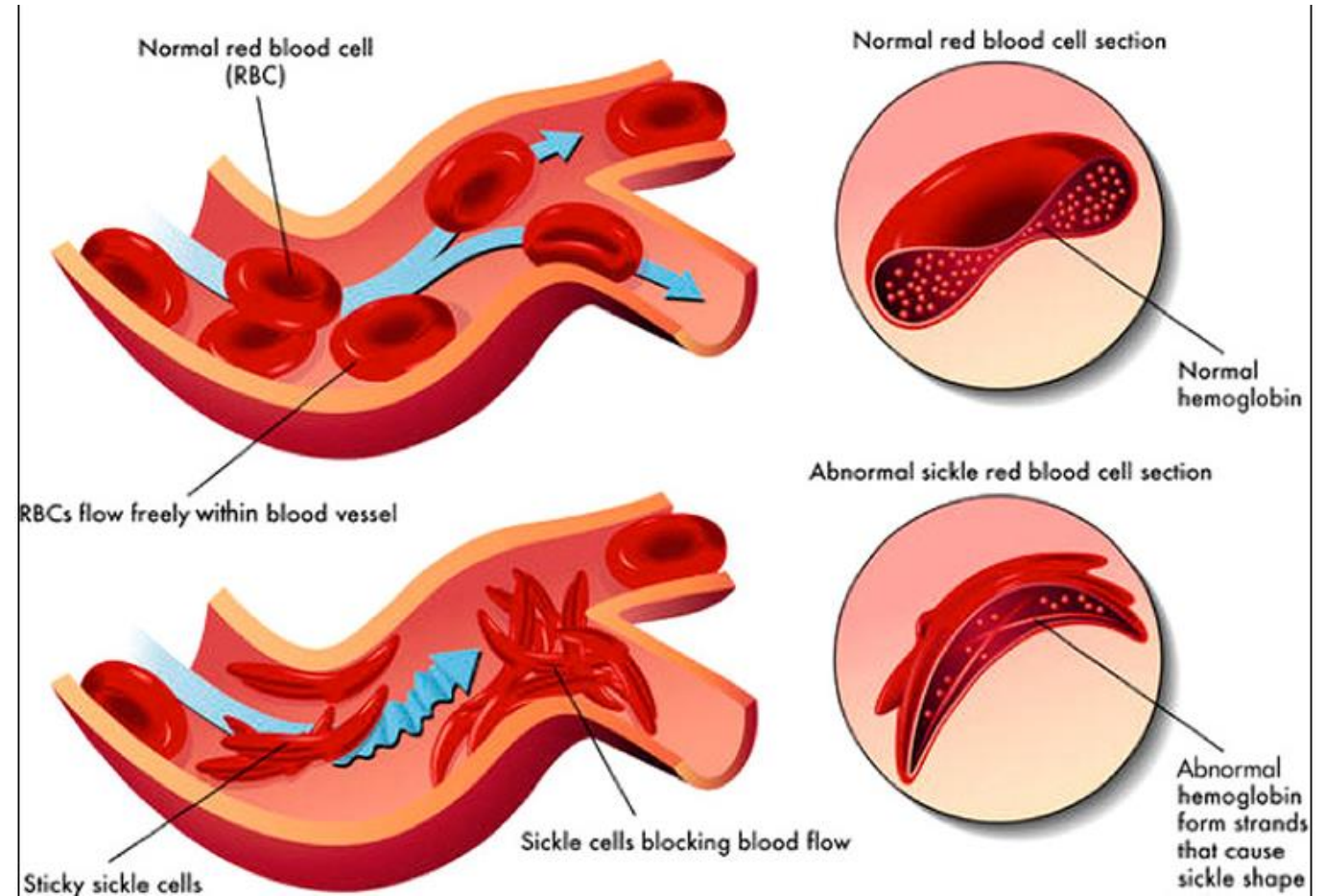


Sickle Cell

SICKLE CELL ANEMIA

Results of Intravascular Sickling:

- Vaso-occlusion and Infarction
- Partially or completely blocked blood vessels supplying various organs and tissues
- Intravascular thrombi
- Hemolysis
- Dysfunction
- Permanent tissue or organ damage





MANAGEMENT OF SICKLE CELL ANAEMIA



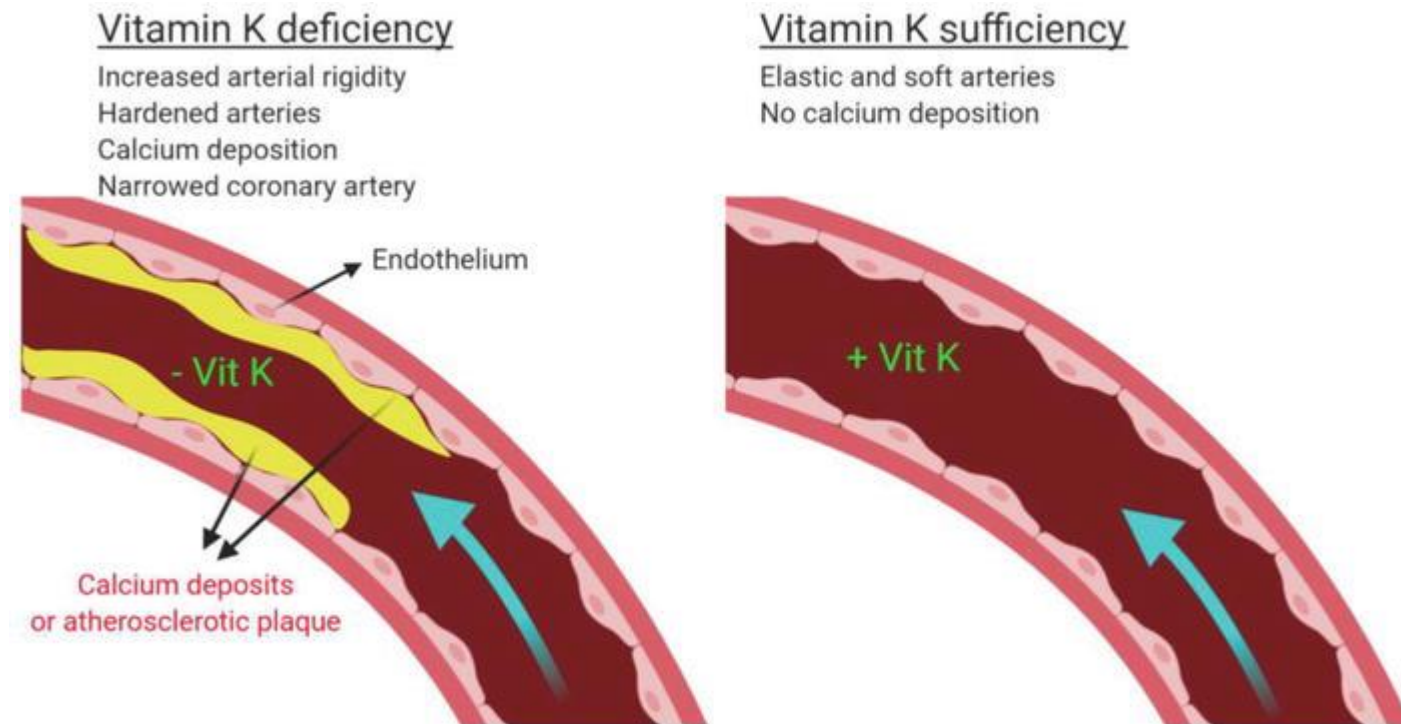
MANAGEMENT:

- Keep the **oxygen % high** and not to cool the pt.
- Maintain the **Po2 (300)**. Keep O2 Saturations **>97%**
- Prevent acidosis and keep blood flow high.
- Add NaHCO₃ if needed and keep **pH = 7.5**
- Use **warm cp or crystalloid cp** that is first infused warm and then cold.
- **Hypothermia should be minimized**, not below 30 degree Celsius
- The use of **vasodilators** to keep vessel open to prevent blockage.
- Maintain adequate level of anaesthesia
- Since pt's with this disease experience sickling at % drop below 85%, it necessitates removing the blood before bypass begins and replacing with either whole blood or adequate combination of components **[packed RBC, FFP, colloids & crystalloid]**.

VITAMIN K DEFICIENCY

Vit K deficiency: Encountered on open heart surgeries.

- Vitamin K is a **fat-soluble vitamin**
- Its function is in **blood coagulation.**
- Regulating calcium level and inhibit vascular calcification





VITAMIN K DEFICIENCY – MANAGEMENT



Causes of Vitamin K Deficiency:

- Due to hemodilution & occlusion etc., **coagulation factors are damaged** or decreased
- vitamin k deficiency leads to bleeding & post operatively leads to cardiac tamponade & DIC.

Treatment: replace blood components like FFP cryoprecipitates platelets, antifibrinolytics & packed RBCs.





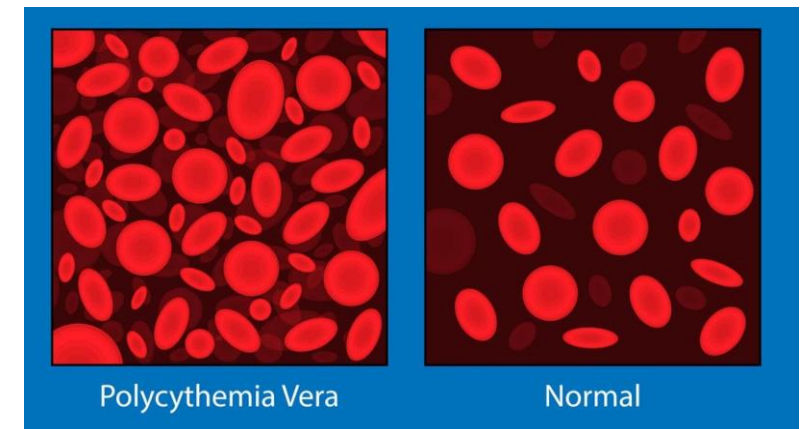
POLYCYTHEMIA



- **Polycythemia:** abnormal increase in the no. of RBCs.

Types of Polycythemia

- Primary Polycythemia – Abnormal bone marrow synthesis RBC
- Secondary Polycythemia – Increased Erythropoietin production in response to chronic hypoxia
- Relative Polycythemia – Increase in number of RBC, caused by loss of plasma volume
- Stress Polycythemia – Because of low volume of plasma seen in anxious persons





POLYCYTHEMIA MANAGEMENT



- A high hematocrit level in patients with erythrocytosis is linked with **increased blood viscosity and increased risk of thromboembolism.**
- It is necessary to adequately lower the hematocrit level before performing a high-risk surgery.
- **Phlebotomy** can be performed as a management procedure once a week prior to surgery to lower the hematocrit level to below 45% as a means to significantly reduce the blood viscosity.
- **Intraoperative hemodilution** is performed by drawing a specific amount of blood and replacing the same amount with colloid solution

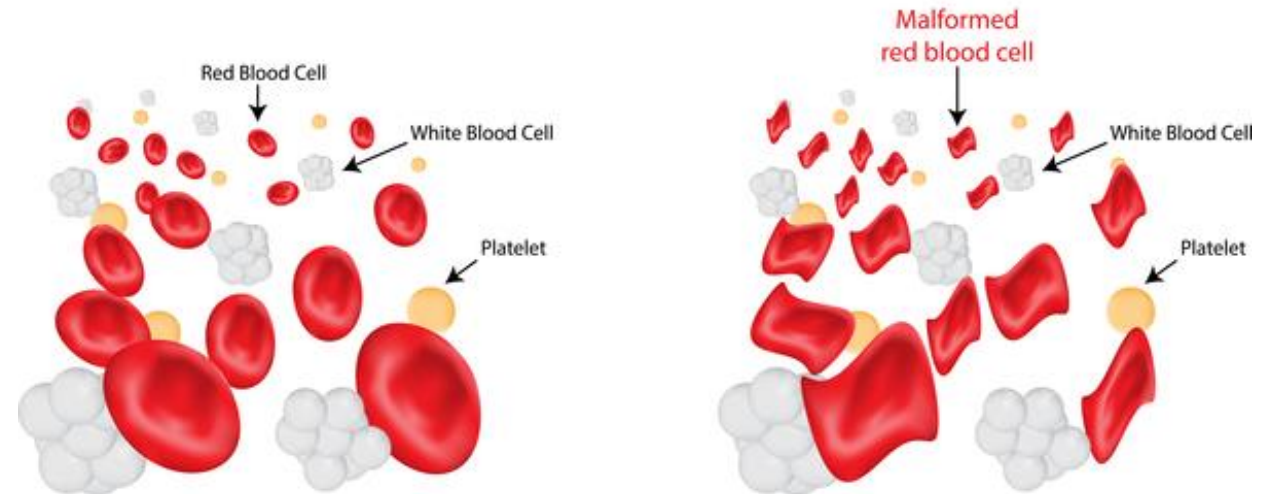


THALASSEMIA

- A blood disorder involving lower-than-normal amounts of an **oxygen-carrying protein**.
- RBCs are microcytic, hypochromic & short lived.
- Increased hemolysis should not occur in bypass.
- It is mainly caused by insufficient haemoglobin synthesis
- Thalassemia major is called as Cooley's Anaemia – RBC destroy occurs in large amount with splenomegaly, requires more transfusion

Management:

- Blood Transfusion is required (Packed RBC)
- Reduction of hemolysis during bypass





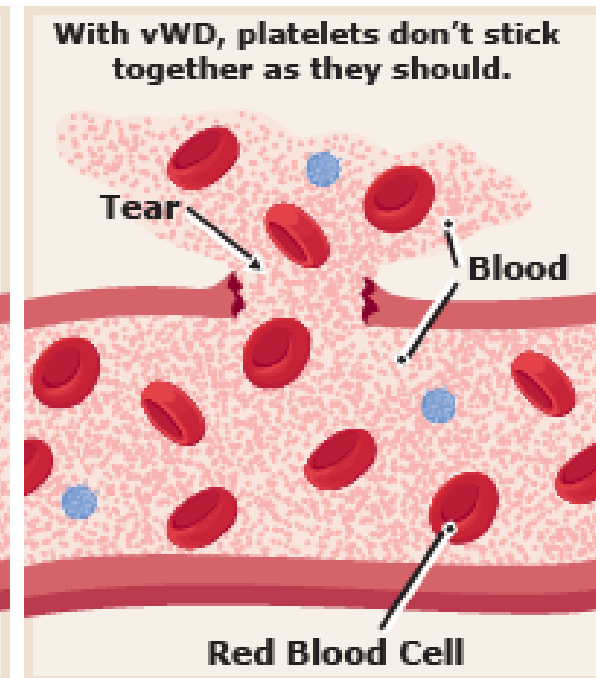
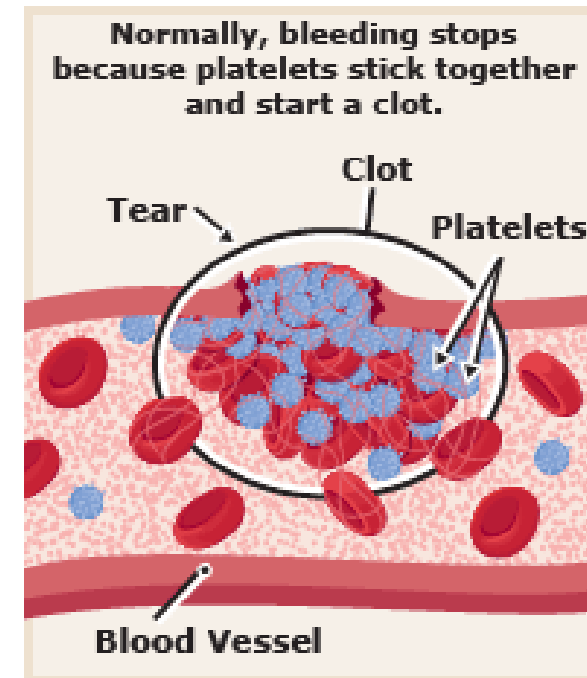
VON WILLEBRAND'S DISEASES



- It is the most common inherited bleeding disorder.
- Blood doesn't clot properly in this disorder
- Von Willebrand's is a carrier protein that facilitates platelet adhesion.

Management:

The Management strategies includes infusion of 10 bags of **cryoprecipitate** after coming off bypass.





OTHER BLOOD DISORDERS

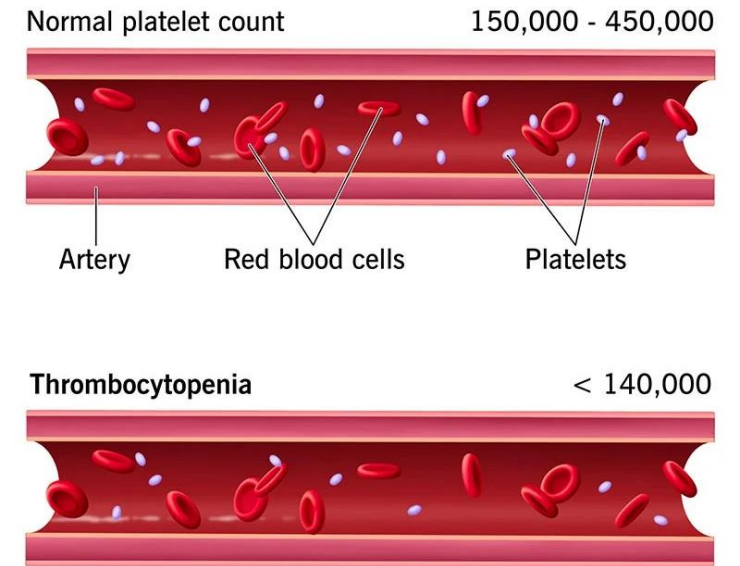


Thrombocytopenia: As a result of decreases bone marrow production, splenomegaly, liver diseases, as a result of drugs, foreign body contact (CPB, IABP, Cardiac valves) and due to heparinisation.

Can be treated by **addition of FFP.**

Disseminated Intravascular Coagulation (DIC) – Life threatening bleeding disorder caused by clotting factor exhaustion. Clot occurs throughout the body and decrease in the factor causes uncontrollable bleeding.

Can be treated by **administration of FFP, Cryoprecipitate, Platelets and Packed RBC**





TREATMENT FOR HEMATOLOGICAL PROBLEMS IN CPB



- **Proper selection** of tubing and prime
- Add only required amount of blood with **proper heparinization**
- **Incorporate hemofilter** to reduce the immune response
- **Incorporate arterial filter**
- **Proper occlusion setting** , reduces hemolysis.





CONTROL POST-OP BLEEDING



Excessive bleeding in the postop period should be controlled:

Greater than 200ml / hr for 4 hrs

Greater than 300ml / 3 hrs

Greater than 400ml / 2 hrs

Greater than 500ml / 1 hr

Bleeding with hemodynamic stability

Management:

- **Increase Positive End Expiratory Pressure (PEEP)**
- **Blood Pressure Control**
- **Additional Protamine Administration**
- **Administer Desmopressin**
- **Blood Product Administration**





USES OF BLOOD PRODUCTS



Packed Red Blood Cells	Reduced HCT, Requiring Transfusion
Whole Blood	Massive Blood Loss
Platelets	Thrombocytopenia
Fresh Frozen Plasma	Clotting Factor Deficiency
Cryoprecipitate	Hemophilia, Von Willebrand's Disease, Factor VIII Deficiency
Albumin	Volume Expander, Decreased Colloid Osmotic Pressure and Hypoproteinemia
Leukocyte Poor RBC	To prevent reactions from leukocyte antibodies and organ transplant patients



ASSESSMENT – III



- What is Sickle cell anemia?
- What is the management for Von Willebrand's disease?
- What is DIC?
- When should we administer Albumin?



THANK YOU



References:

Manual of Clinical Perfusion – ED BRYAN

CPB – Principles and Techniques – Mohamed Barham