

Bleeding, Hemostasis, Transfusion

Emergency Surgery

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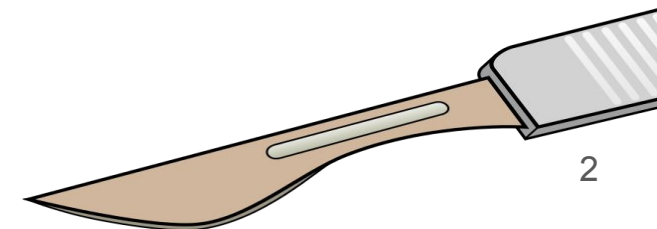
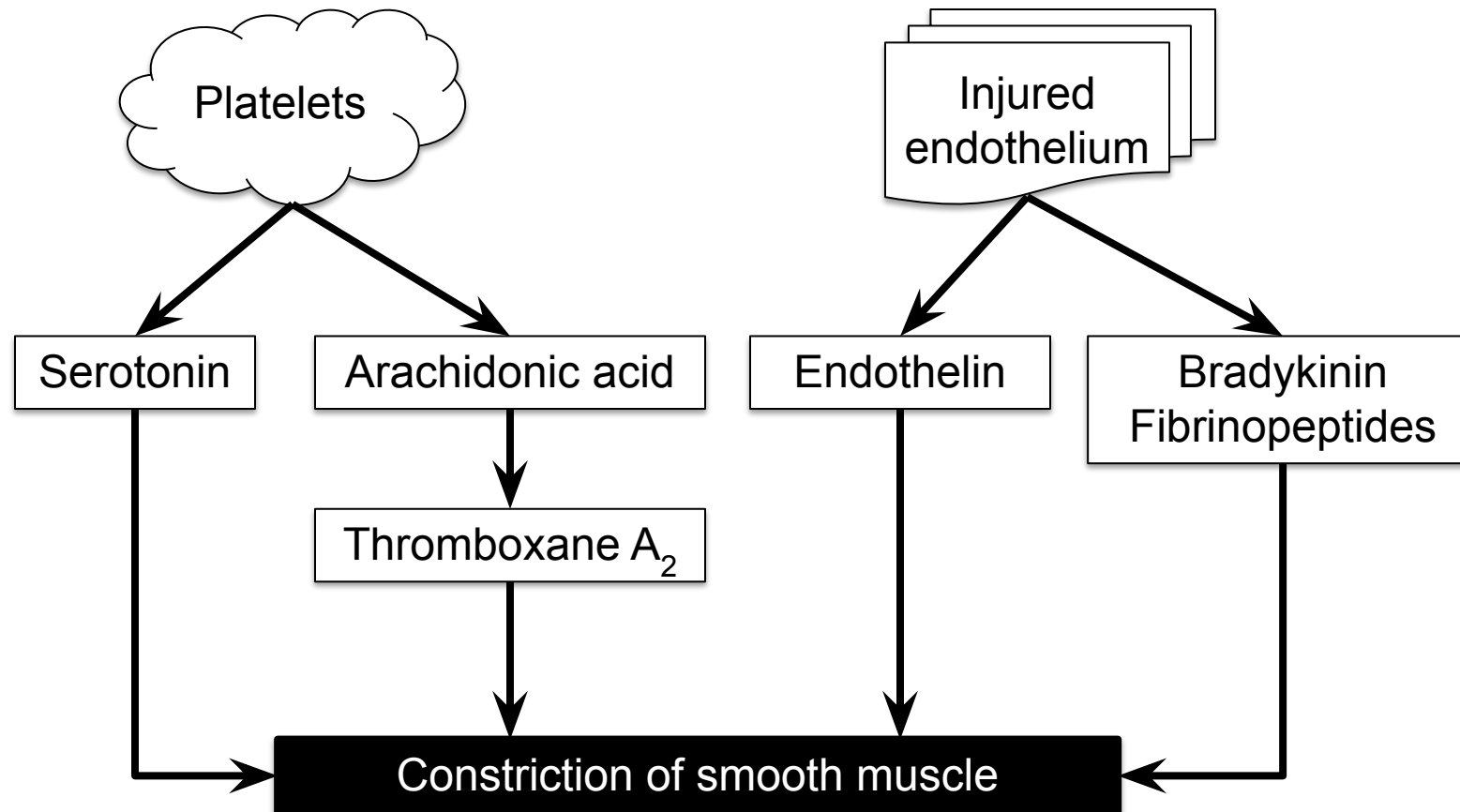


1) Vascular
constriction

2) Platelet
plug formation

3) Fibrin plug
formation

4) Fibrinolysis



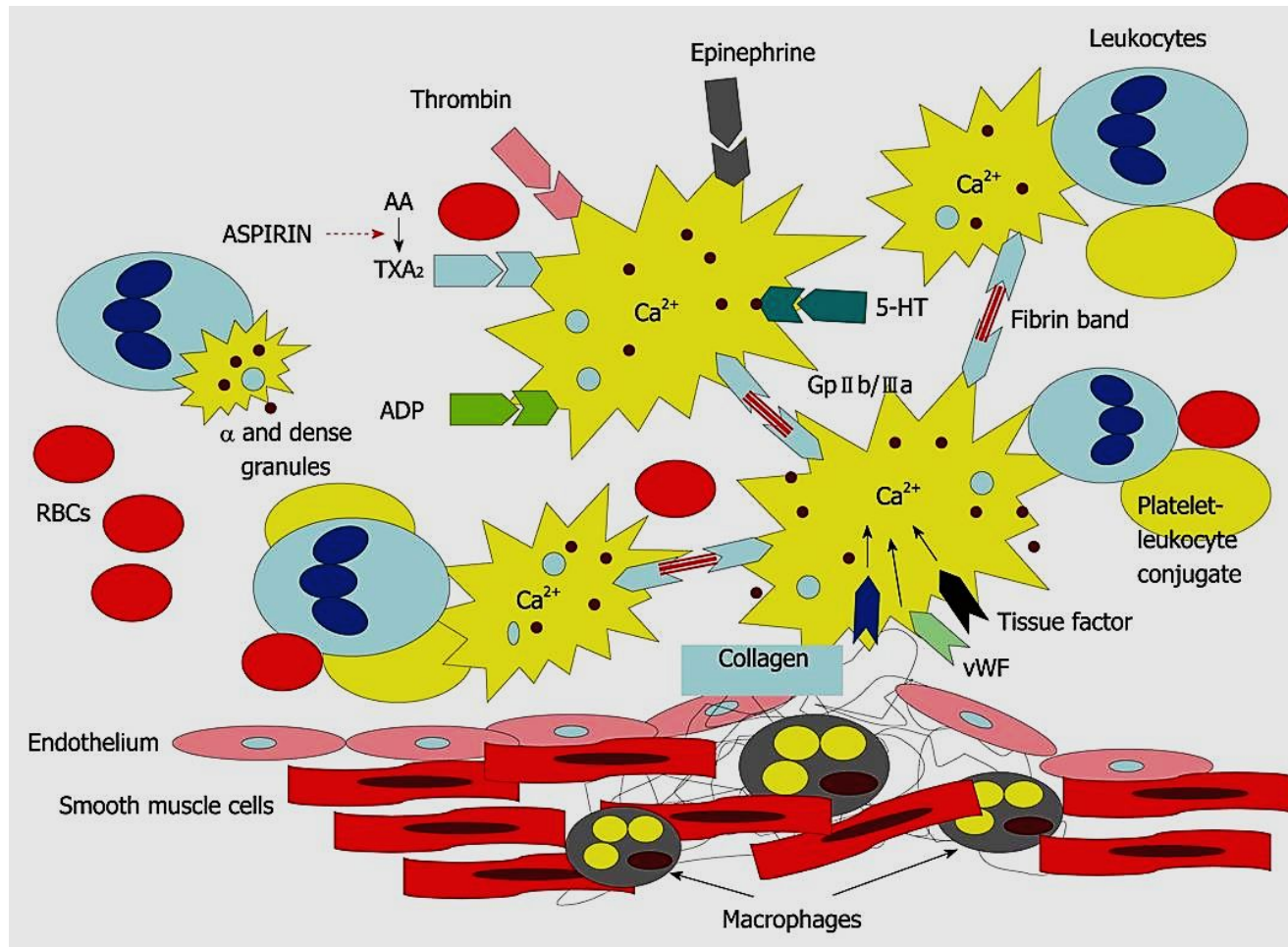
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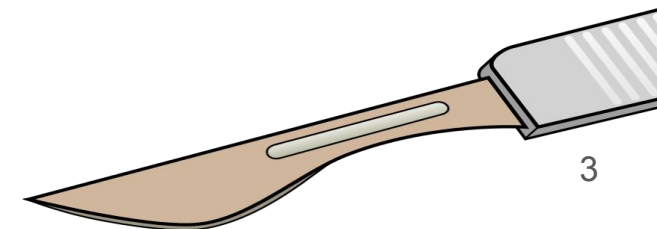
4. Fibrinolysis

First wave of platelet aggregation = PRIMARY HEMOSTASIS



von Willebrand's
disease

Heparin does not
interfere with this
reaction, and thus
hemostasis can occur
in the heparinized
patients.

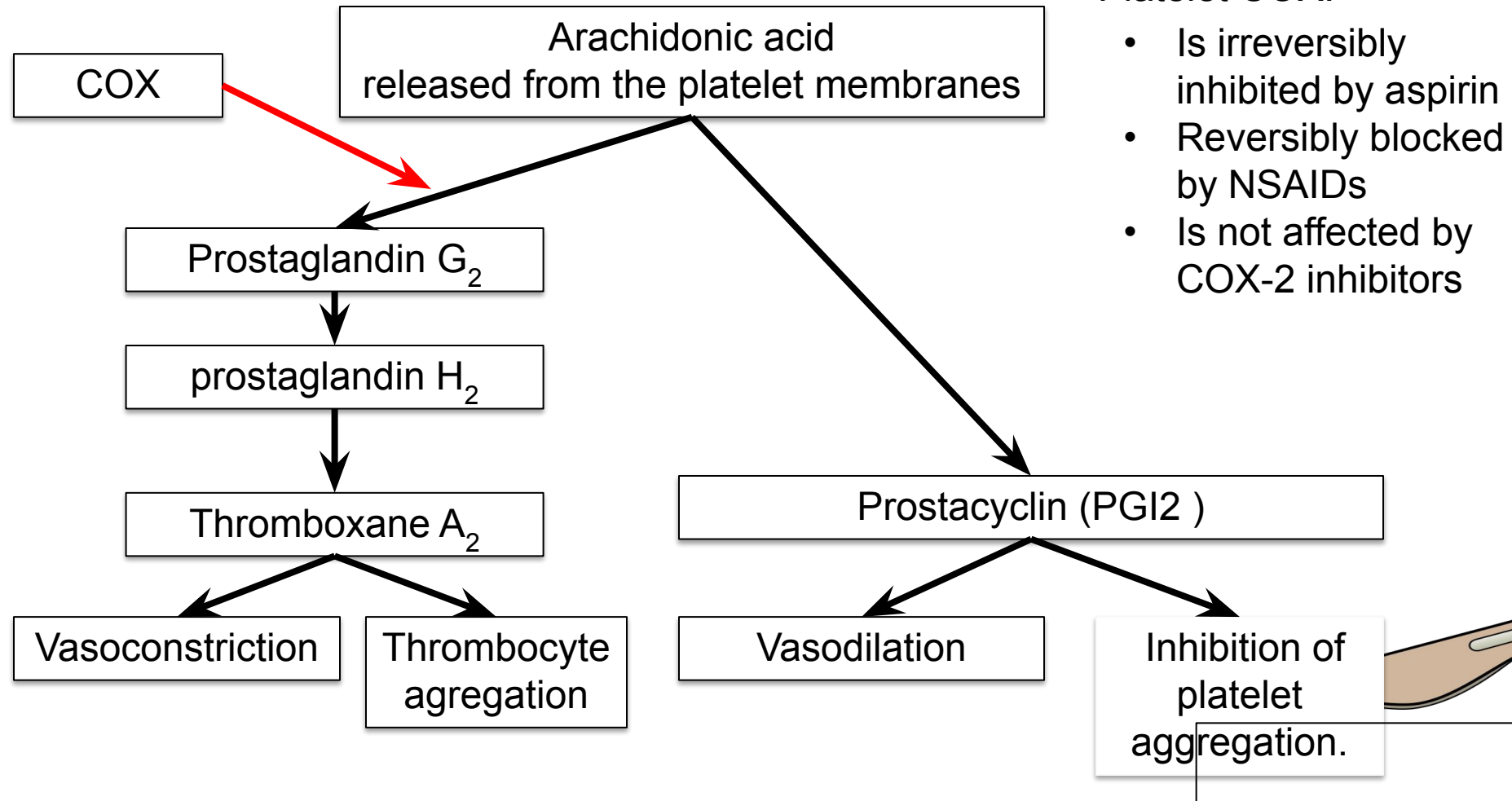


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Is inhibited by:

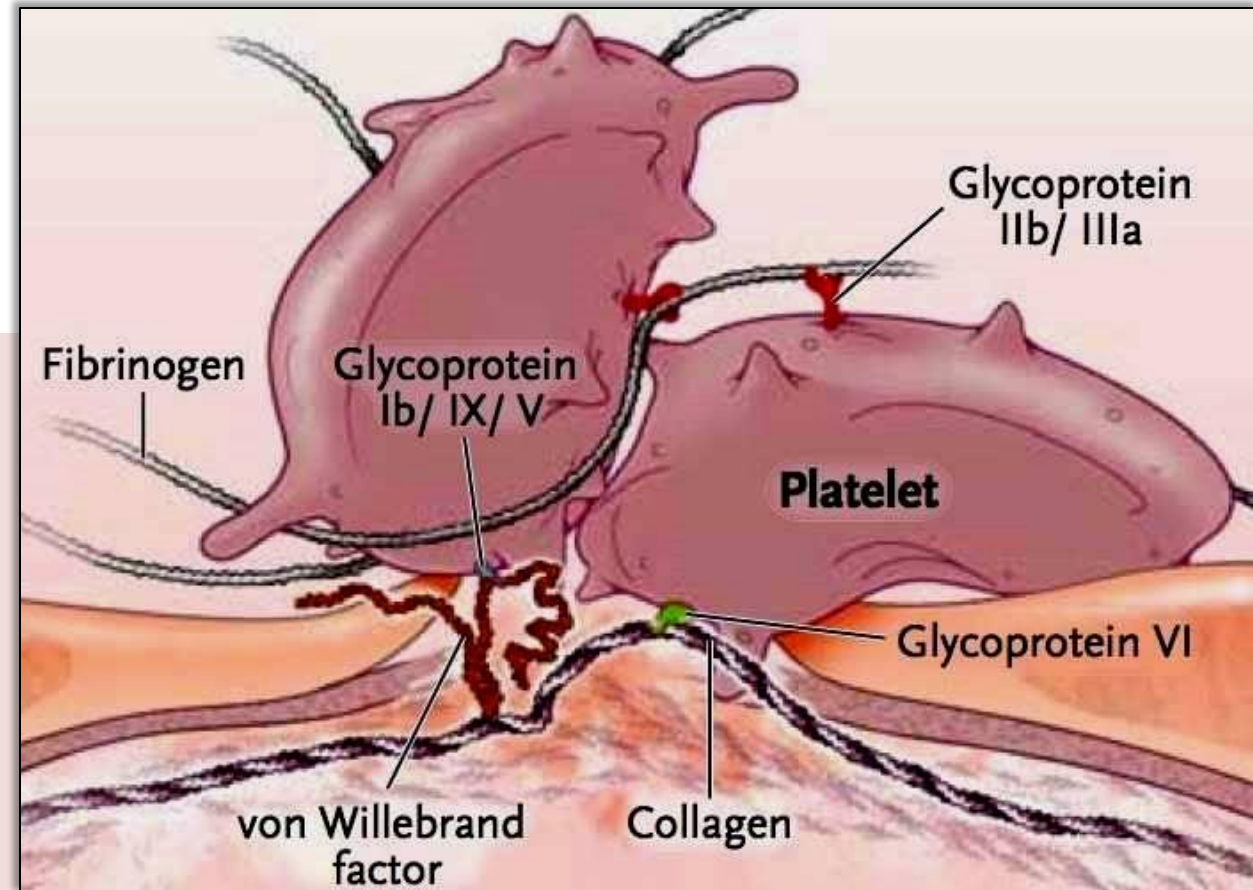
Aspirin and NSAIDs
Cyclic adenosine
monophosphate (cAMP)
Nitric oxide

Bleeding time

**Fibrinogen is bridge for
the glycoprotein IIb/IIIa**

- ADP
- Serotonin
- TXA_2
- α -granule proteins
(thrombospondin)
- α -thromboglobulin
- Platelet factor 4 =
heparin antagonist

Second wave of platelet aggregation

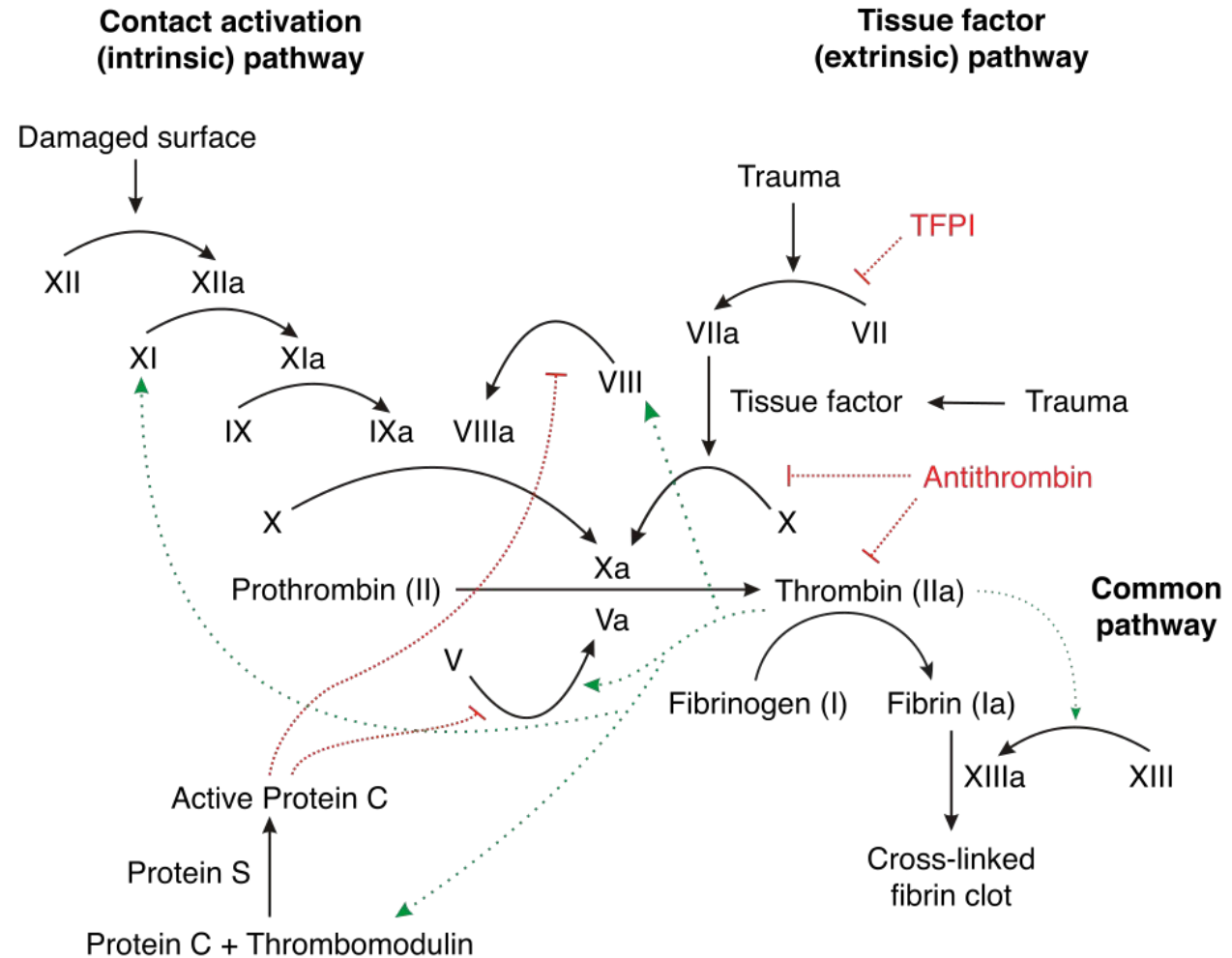


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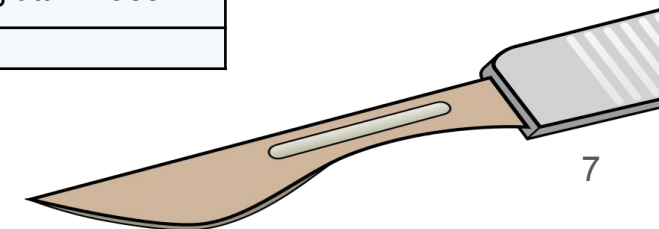
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PreferredName	Synonym	Role inModernCoagulationCascadeModel
Factor I	Fibrinogen	–
Factor II	Prothrombin	Prethrombin
Tissuefactor (formerlyknown asfactor III)	Tissue factor	Tissuethromboplastin
Calcium (formerly known as factor IV)	Calcium	Ca ²⁺
Factor V	Proaccelerin	Labile factor, acceleratorglobulin (AcG)
Factor VII	Proconvertin	Stabile factor, serumprothrombinconversionaccelerator,autoprotein
FactorVIII	Antihemophilic factor (AHF)	Antihemophilicglobulin (AHG), antihemophilicfactor A, plateletcofactor 1
Factor IX	Plasma thromboplastin component(PTC)	Christmas factor, antihemophilicfactor B, plateletcofactor 2
Factor X	Stuart-Prowerfactor	AutoproteinIII, thrombokinase
Factor XI	Plasma thromboplastin antecedent(PTA)	Antihemophilic factor C
Factor XII	Hagemanfactor	Glass factor, contact factor
FactorXIII	Fibrin-stabilizingfactor (FSF)	Laki-Lorandfactor (LLF), fibrinase, plasma transglutaminase
Von Willebrand factor	von Willebrandfactor	vWF



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All of the clotting factors except VIII are produced in the liver. Factor VIII is manufactured in the endothelial cells.

Factors II, VII, IX, and X are vitamin K dependent clotting factors.

Without adequate vitamin K, we see prolonged PT/INR and PTT/APTT values.

Vitamin K deficiency:

- Fat soluble

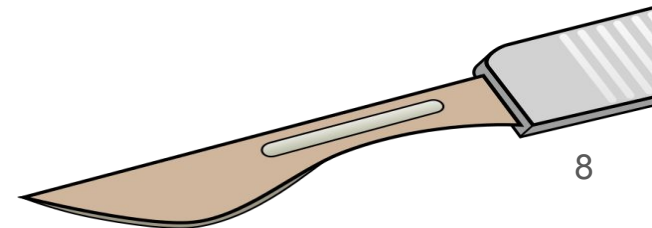
- Low bile production

- Inadequate vitamin K intake

- Chronic small intestinal disease

can all result in chronic vitamin K and prolonged PT/INR and PTT/APTT values.

Factor VII has the shortest half-life compared to the other factors.



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INTRINSIC PATHWAY

- Damaged vessel wall
- Slow
- aPTT
- Heparin

12

11

9

8

EXTRINSIC PATHWAY

- Damaged tissue
- Quick
- PT / INR
- Warfarin

Tissue
thromboplastin

7

COMMON PATHWAY

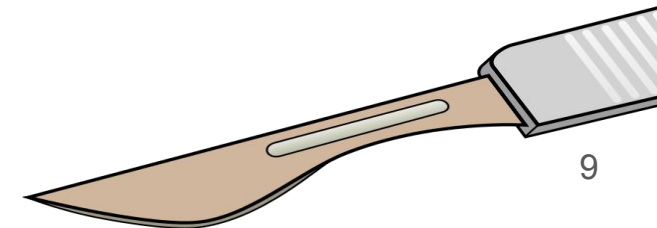
10+5+Ca⁺⁺

2

13

1

COAGULATION

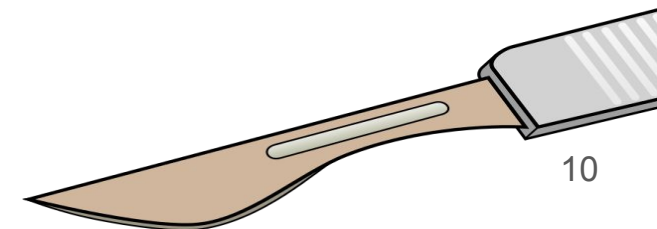
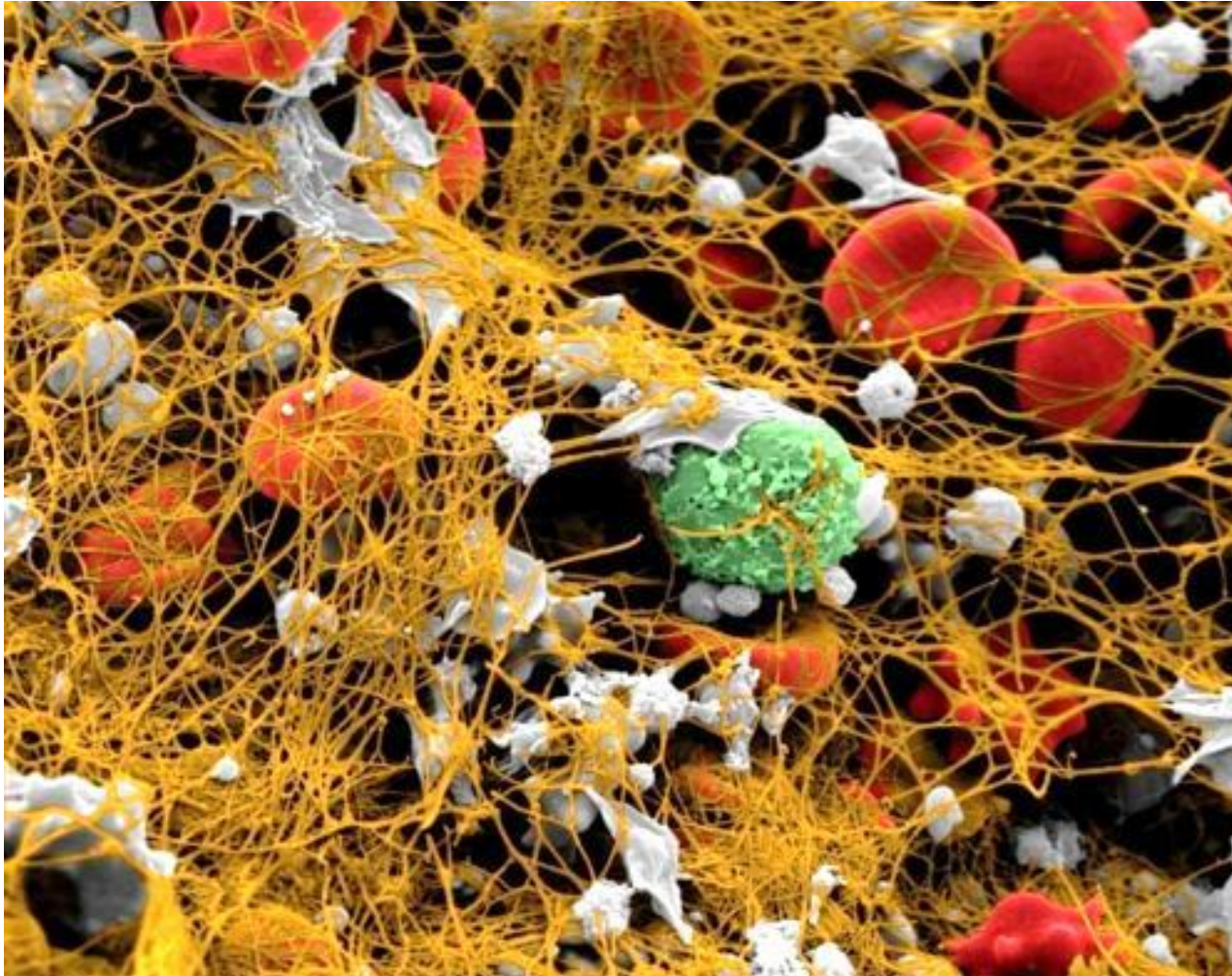


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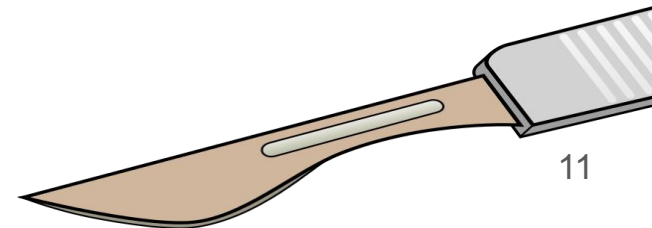
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Regulation of coagulation

- Prevention of propagation of the clot beyond the site of injury:
 - Feedback inhibition on the coagulation cascade
 - Mechanisms of fibrinolysis
- Repair of the injured vessel with deposition of connective tissue.



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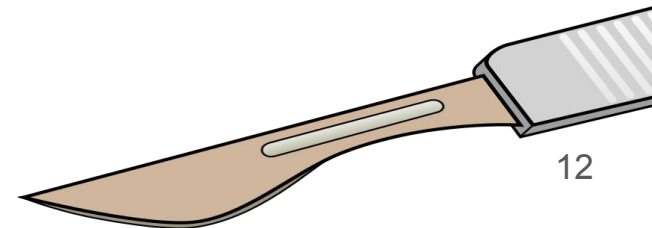
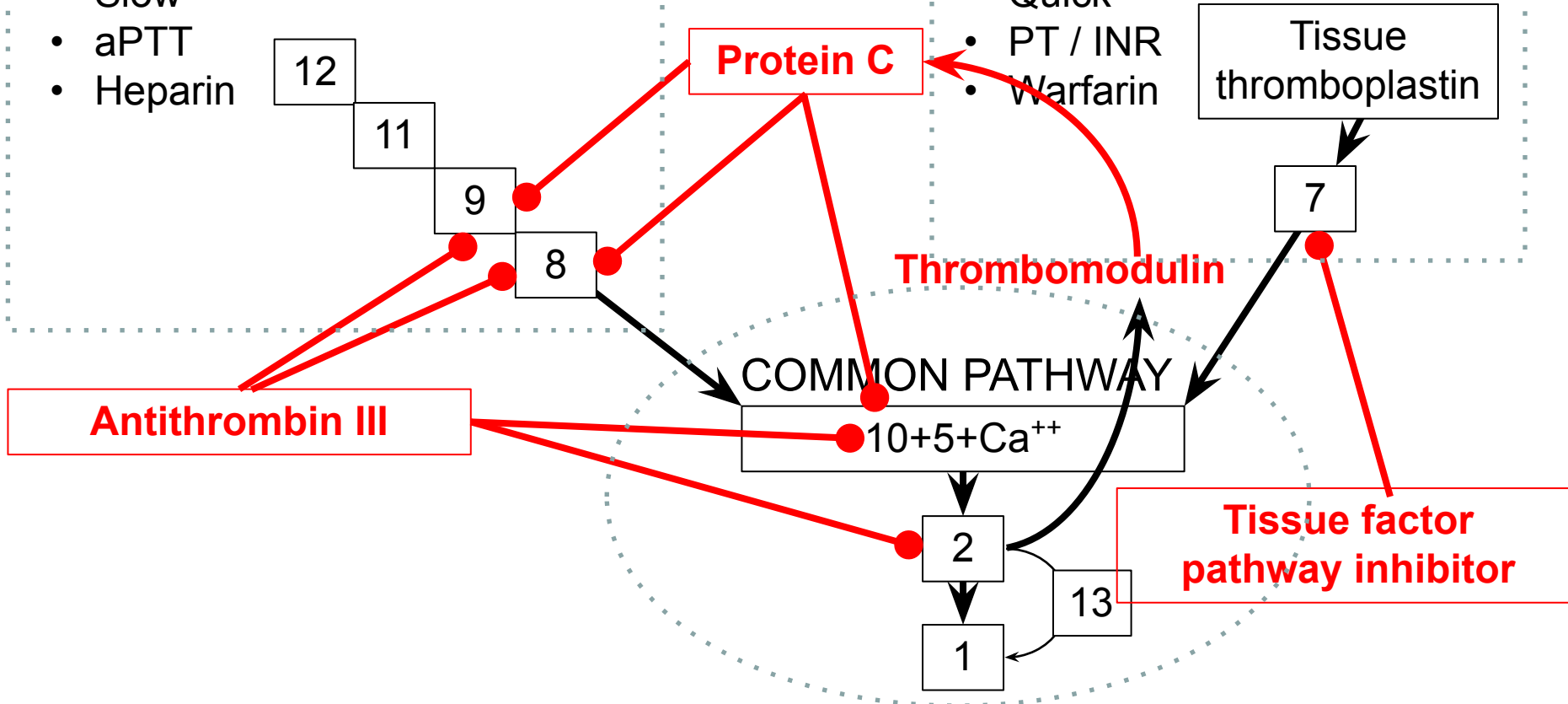
4. Fibrinolysis

INTRINSIC PATHWAY

- Damaged vessel wall
- Slow
- aPTT
- Heparin

EXTRINSIC PATHWAY

- Damaged tissue
- Quick
- PT / INR
- Warfarin

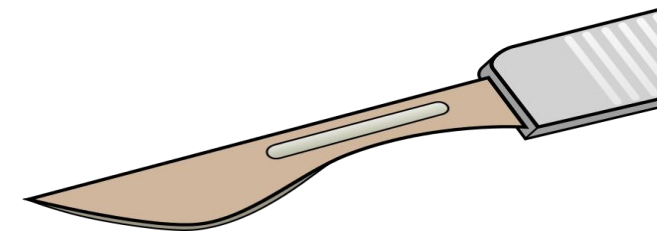
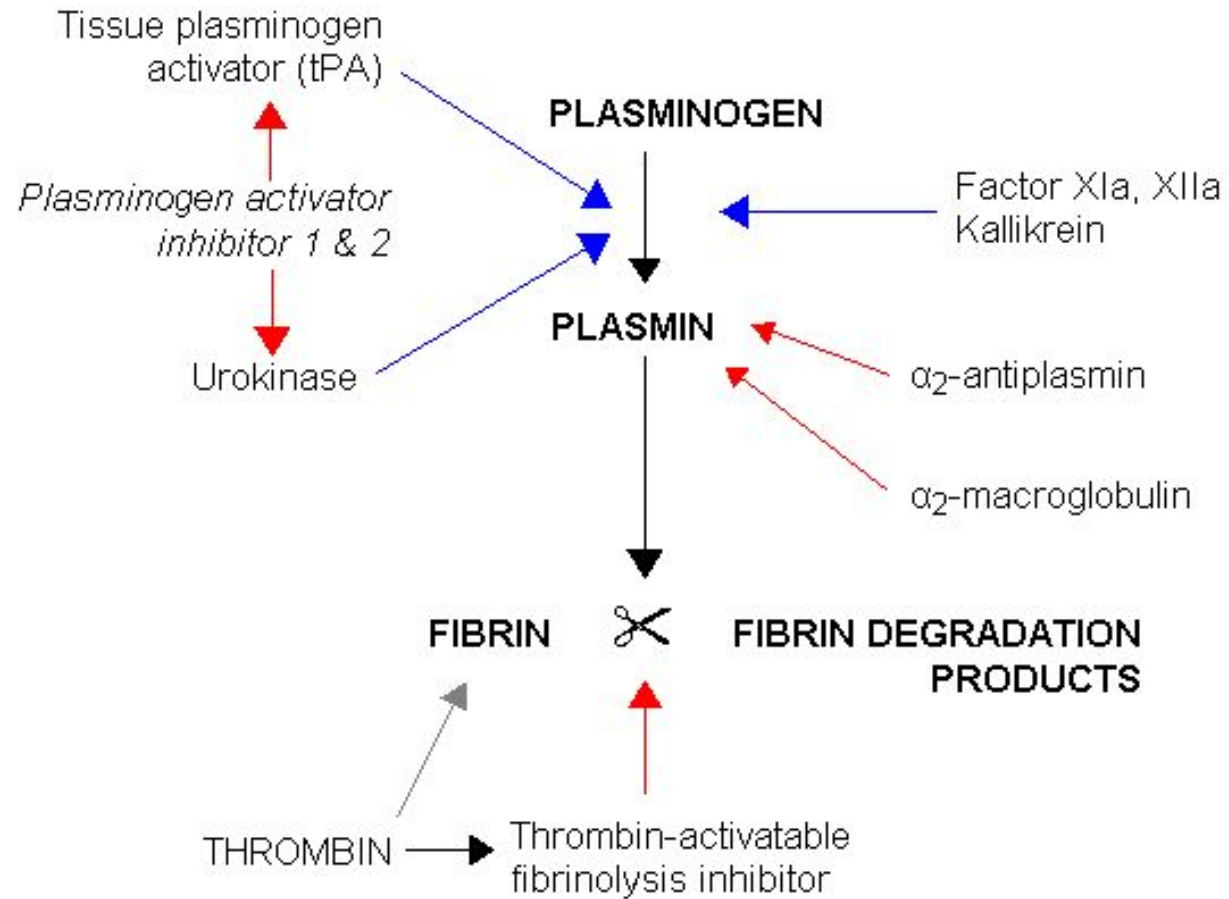


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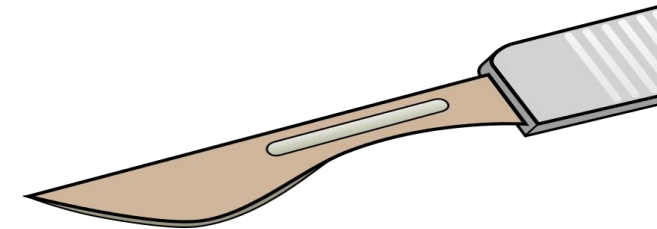
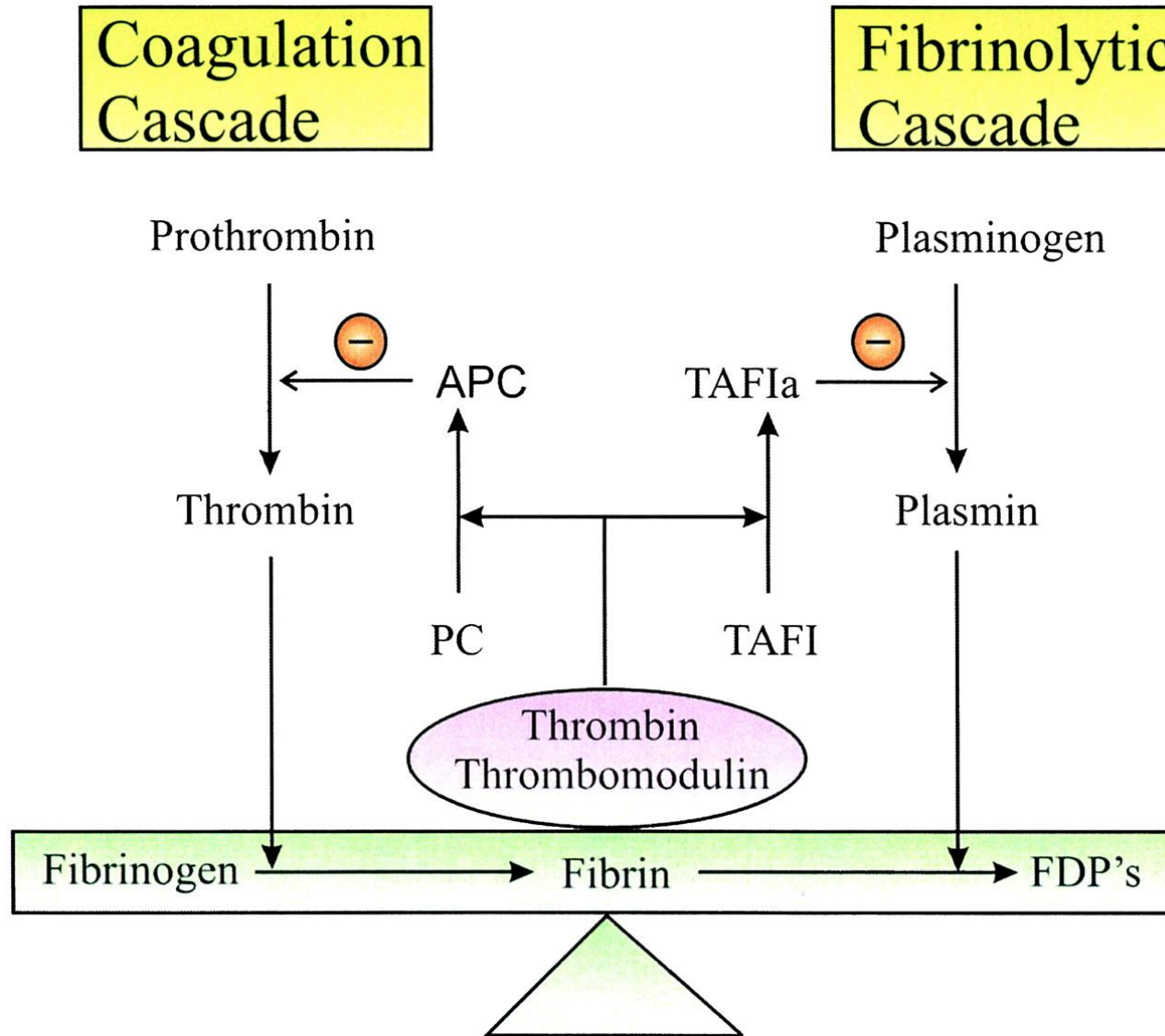
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Equilibrium



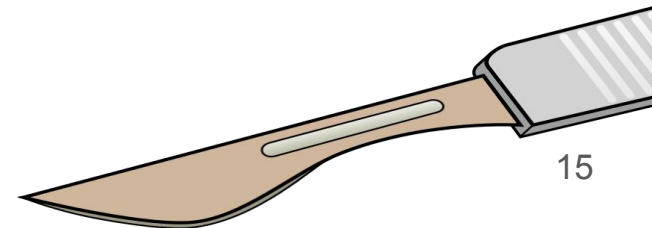
Coagulation Factor Deficiencies

- Factor VIII deficiency
 - Hemophilia A
 - Willebrand's disease
- Factor IX deficiency
 - Hemophilia B
- Factor XI deficiency
 - Hemophilia C

8 9 11

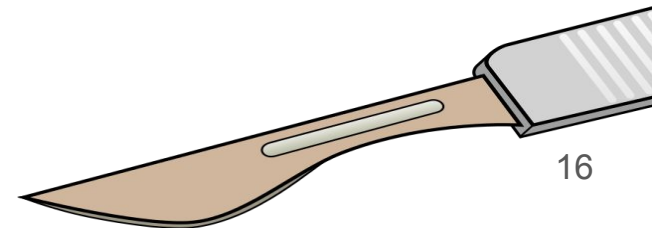
Plasma factor levels:

- <1% of normal - severe
 - Spontaneous bleeds into joints, intramuscular, retroperitoneal hematomas, and GI, genitourinary and retropharyngeal bleeding. Intracranial bleeding and bleeding from the tongue or lingual frenulum.
- 1÷5% - moderate
 - less spontaneous bleeding but are likely to bleed severely after trauma or surgery.
- >5% - mild disease
 - Minor bleeding after major trauma or surgery



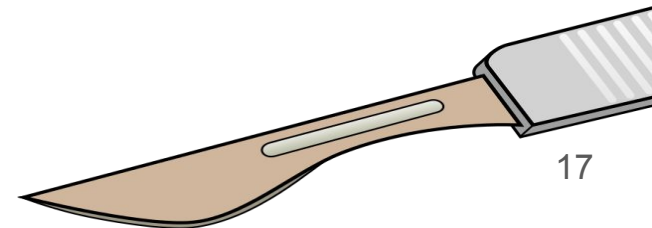
von Willebrand's disease

- Most common congenital bleeding disorder. Autosomal dominant disorder.
- Low level of vWF, a large glycoprotein responsible for carrying factor VIII and platelet adhesion→Low levels of factor VIII.
- Easy bruising, mucosal bleeding, menorrhagia.
 - Type I - partial quantitative deficiency.
 - Type II - qualitative defect
 - Type III is total deficiency
- Treatment:
 - Concentrate of vWF and VIII.
 - Desmopressin acetate →of the vWF from endothelial cells.
 - Type III patients are usually unresponsive



Factor XI deficiency

- Autosomal recessive (hemophilia C)
- Ashkenazi Jewish population.
- Bleeding may occur after surgery, trauma, or invasive procedures, rarely spontaneously.
- Treatment:
 - Fresh-frozen plasma
 - Recombinant factor VIIIa
 - Desmopressin acetate



INHERITED DEFICIENCIES OF FACTORS II, V, AND X

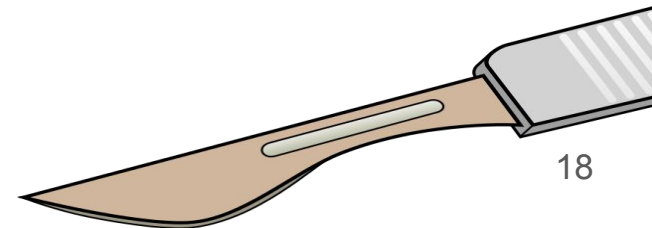
- Rare, autosomal recessive
- Treated with FFP

CONGENITAL FACTOR XIII DEFICIENCY

- Rare, autosomal recessive
- Bleeding typically is delayed, because clots susceptible to fibrinolysis.
- Umbilical stump bleeding, intracranial bleeding, spontaneous abortion,
- FFP, cryoprecipitate, factor XIII concentrate

INHERITED FACTOR VII DEFICIENCY

- Rare, autosomal recessive
- Easy bruising, epistaxis or oral mucosal bleeding.
- Postoperative bleeding
- Treatment is with FFP or recombinant factor VIIa.
- The half-life VIIa ~ 2 hours→Frequent infusions.



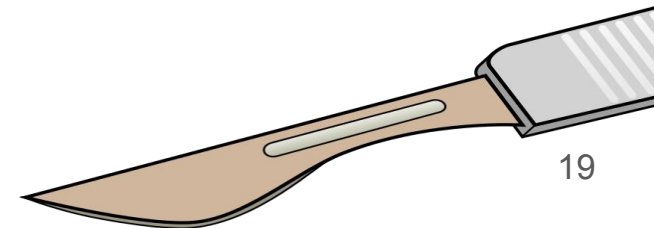
Platelet Functional Defects

Surface protein abnormalities:

- Glanzmann thrombasthenia
 - Rare, autosomal recessive
 - Glycoprotein IIb/IIIa complex is either lacking or dysfunctional
 - Faulty platelet aggregation and subsequent bleeding.
- Bernard-Soulier syndrome.
 - Defect in the glycoprotein Ib/IX/V receptor for vWF
 - Transfusion of normal platelets.

Intrinsic platelet defect - storage pool disease.

- Dense granules [storage sites for ADP, adenosine triphosphate (ATP), Ca^{2+} , and inorganic phosphate] deficiency - Bleeding is variable.
- An isolated defect of the α -granules - gray platelet syndrome
- Both dense and α -granules - more severe bleeding disorder.
 - Desmopressin acetate $\rightarrow$ high levels of vWF compensate deficit
 - Platelet transfusion



Acquired Platelet Disorders

1) QUANTITATIVE DISORDERS

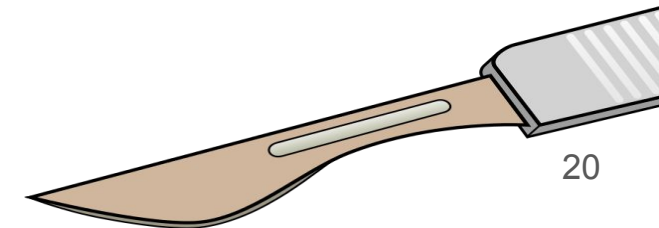
- a) Failure of production: related to impairment of bone marrow function
 - i) Leukemia
 - ii) Myeloproliferative disorders
 - iii) Vitamin B12 or folate deficiency
 - iv) Chemotherapy or radiation therapy
 - v) Acute alcohol intoxication
 - vi) Viral infections
- b) Decreased survival
 - i) Immune-mediated disorders
 - (1) Idiopathic thrombocytopenia
 - (2) Heparin-induced thrombocytopenia
 - (3) Autoimmune disorders or B-cell malignancies
 - (4) Secondary thrombocytopenia
 - ii) Disseminated intravascular coagulation
 - iii) Disorders related to platelet thrombi
 - (1) Thrombocytopenic purpura
 - (2) Hemolytic uremic syndrome

c) Sequestration

- i) Portal hypertension
- ii) Sarcoid
- iii) Lymphoma
- iv) Gaucher's disease

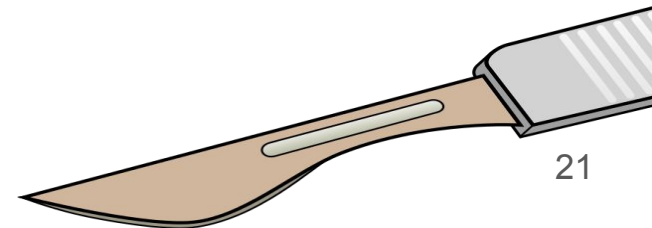
2) QUALITATIVE DISORDERS

- c) Massive transfusion
- d) Therapeutic administration of platelet inhibitors
- e) Disease states
 - i) Myeloproliferative disorders
 - ii) Monoclonal gammopathies
 - iii) Liver disease



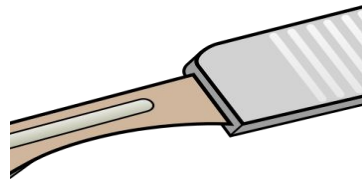
Idiopathic thrombocytopenic purpura

- Primary immune thrombocytopenia
- Children: Acute, after virus infection;
- Adults: Gradual in onset, chronic, and has no identifiable cause



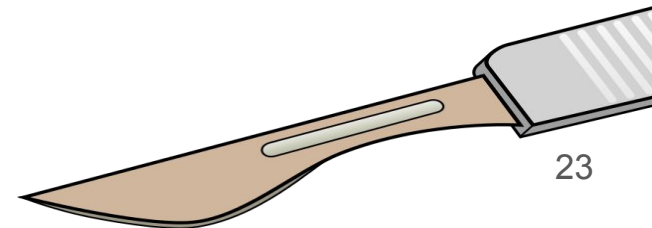
Management of Idiopathic Thrombocytopenic Purpura in Adults

- 1) First Line
 - a) Corticosteroids.
 - b) IV immunoglobulin.
 - c) Anti-D immunoglobulin: Active only in Rh-positive patients before splenectomy.
- 2) Second Line
 - a) Splenectomy: Treatment failure may be due to retained accessory splenic tissue.
- 3) Third Line. Maintain the platelet count $>20-30 \times 10^9/L$
 - a) Rituximab, an anti-CD20 monoclonal antibody: Acts by eliminating B cells.
 - b) Alternative medications producing mixed results and a limited response: Danazol, cyclosporine A, dapsone, azathioprine, and vinca alkaloids.
 - c) Thrombopoietic agents: A new class of drugs, still in clinical trials include AMG531 and eltrombopag.

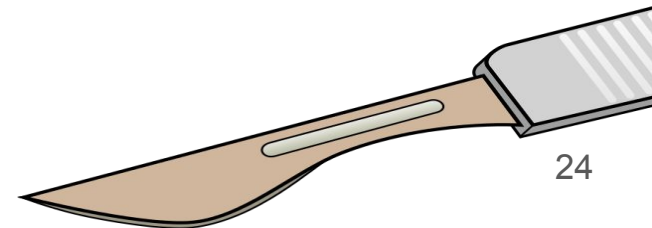


Heparin-induced thrombocytopenia

- Drug-induced immune thrombocytopenia. antibodies against PF4
- 17% - unfractionated heparin and 8% of LMW heparin develop antibodies against PF4 without clinical HIT.
- High incidence of arterial or venous thrombosis
- Importantly, the absence of thrombocytopenia in these patients does not preclude the diagnosis of HIT.
- Diagnosis:
 - Serotonin release assay
 - Enzyme-linked immunosorbent assay (ELISA)
- Treatment:
 - Stop heparin and begin an alternative anticoagulant (thrombin inhibitors).



- One unit of platelet concentrate increases the circulating platelet count by approximately 10,000/ μ L



Disseminated intravascular coagulation (DIC)

- Central nervous system injuries with embolization of brain matter;
- Fractures with embolization of bone marrow;
- Amniotic fluid embolization;
- Malignancy;
- Organ injury (severe pancreatitis);
- Liver failure;
- Vascular abnormalities (aneurysms);
- Snakebites;
- Illicit drugs;
- Transfusion reactions;
- Transplant rejection;
- Sepsis;

Excessive thrombin generation

Consumption and depletion of coagulation factors and platelets

Diffuse bleeding

Thrombocytopenia, $\uparrow$ PT, $\downarrow$ fibrinogen, $\uparrow$ Fibrin markers (fibrin degradation products, D-dimer, soluble fibrin monomers).

Treatment:

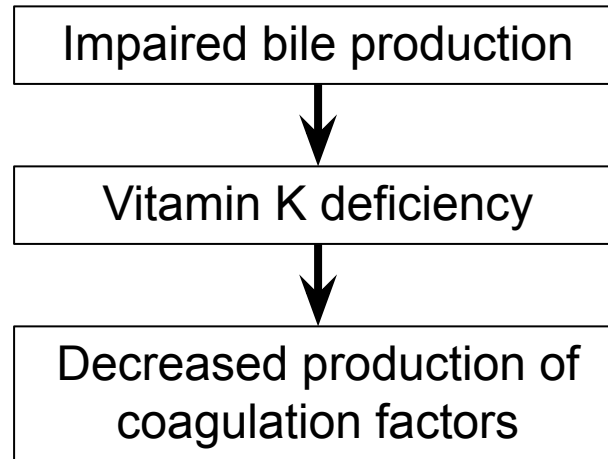
- Relieving causative problem;
- FFP;
- Microthrombi $\rightarrow$ heparin.

Coagulopathy of Liver Disease

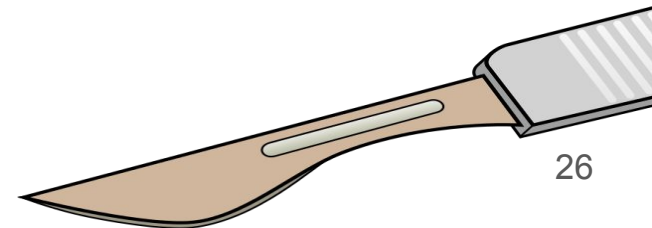
- Most coagulation factors are synthesized in liver → ↑PT, ↑INR;
- Portal hypertension → Splenomegaly → Hypersplenism → Thrombocytopenia
- Platelet transfusions - effect lasts only several hours.
- Administration of interleukin-11 – Significant side effects.
- Splenectomy or splenic embolization or transjugular intrahepatic portosystemic shunt (TIPS)

▼
Reduced splenic blood flow can
reduce portal vein flow

▼
Portal vein thrombosis.

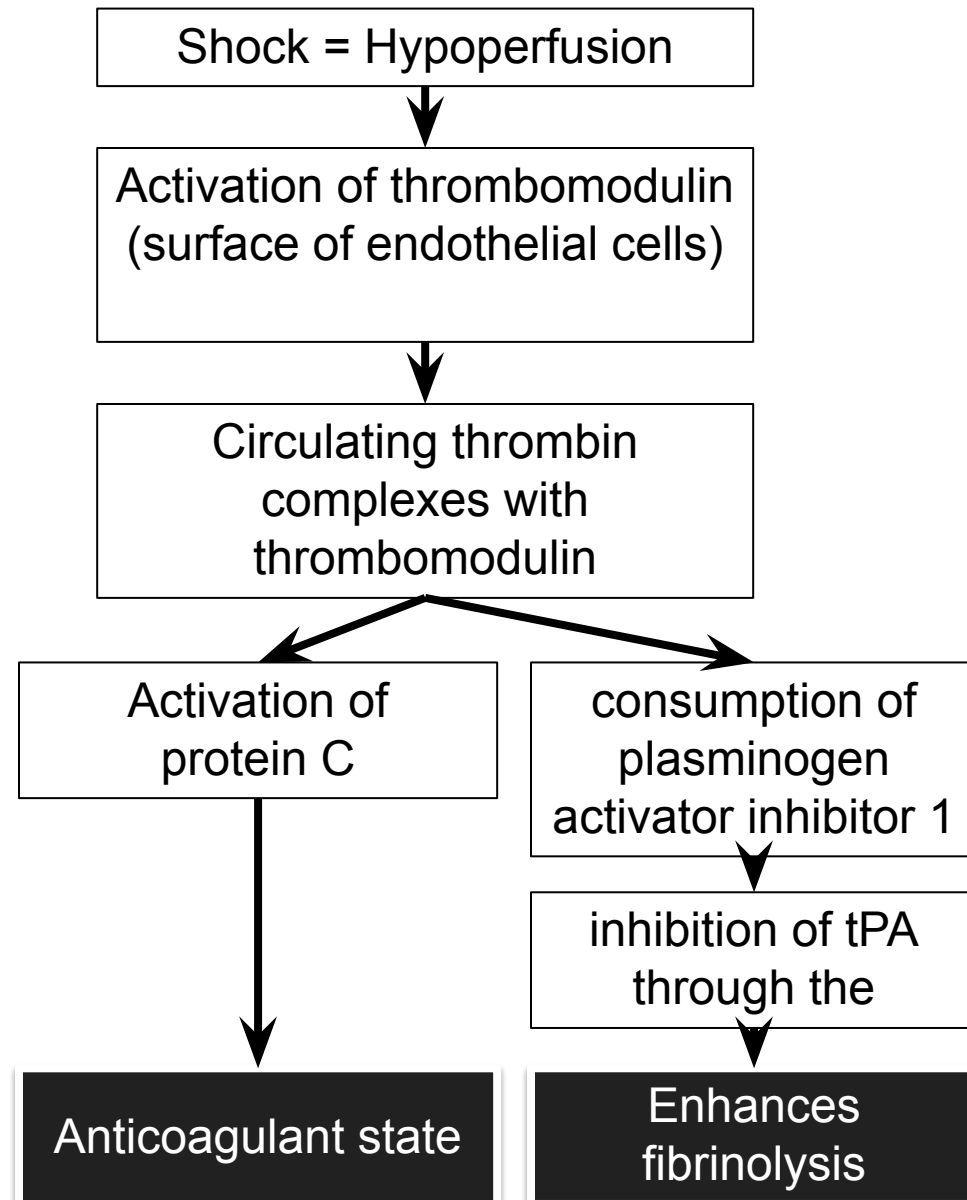


FFP
Vitamin K



Coagulopathy of Trauma

- Acidosis
- Hypothermia
- Dilution of coagulation factors.
- Only patients in shock are coagulopathic
- Shock → activation of anticoagulant and fibrinolytic pathways → coagulopathy



Anticoagulation and Bleeding

HEPARIN	WARFARIN	RIVAROXABAN (Xarelto®)
Direct anticoagulant	Indirect anticoagulant	Direct anticoagulant
Enhancing Antithrombin III	Vitamin K antagonist	Factor Xa inhibitor
SC injection, IV infusion	Oral	The novel oral anticoagulants (NOACs)
aPTT control (for unfractionated heparin)	INR control	No lab control required
For low molecular weight heparin lab control not required.	Interaction of the P-450 system:	
Antidote - Protamine sulfate	Antidote - Vitamin K	Antidote - Recombinant factor Xa

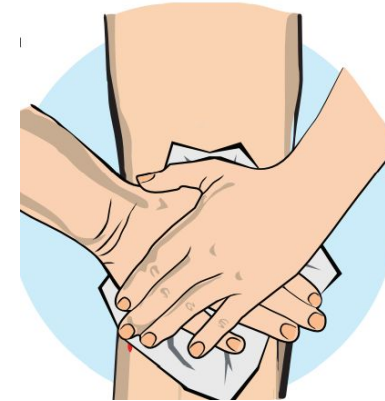
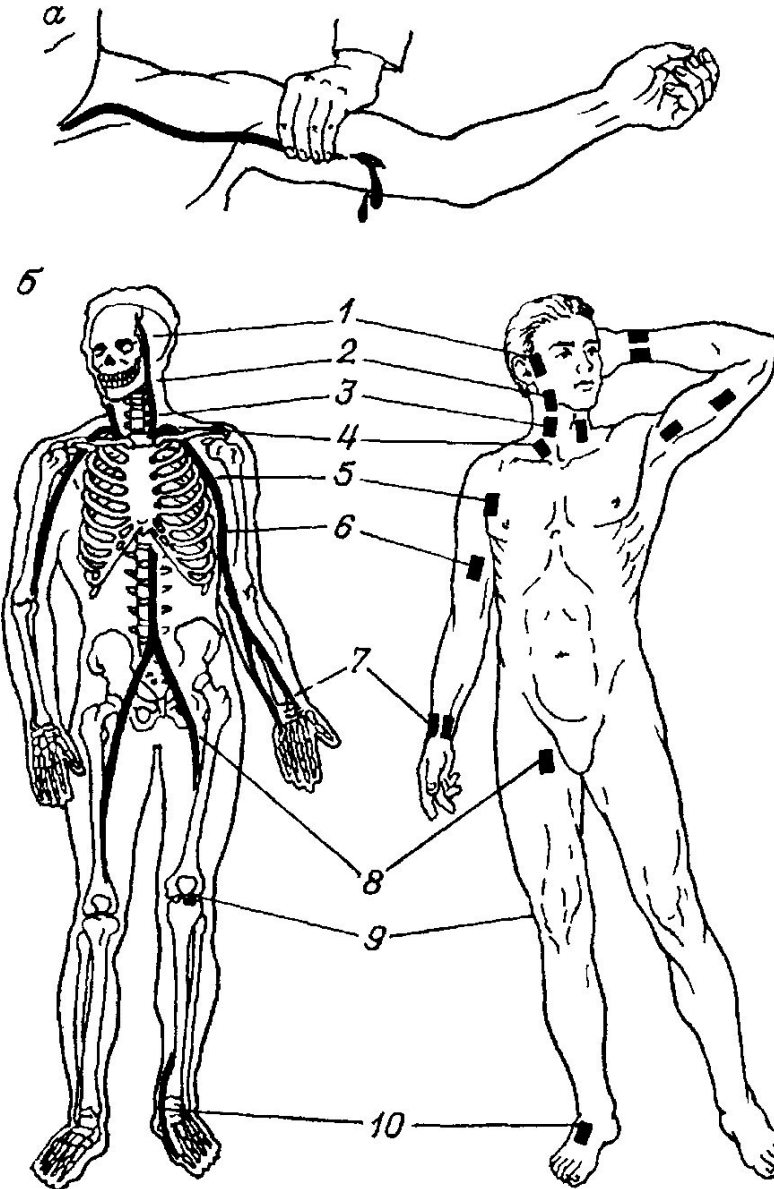
Hematuria, soft tissue bleeding, intracerebral bleeding, skin necrosis, bleeding into the abdominal cavity, intramural bowel hematoma, rectus sheath hematomas

- aPTT is <1.3 times the control value in a patient receiving heparin
 - INR is <1.5 in a patient taking warfarin
- reversal of anticoagulation therapy may not be necessary.
Exception: central nervous system or eye surgery.

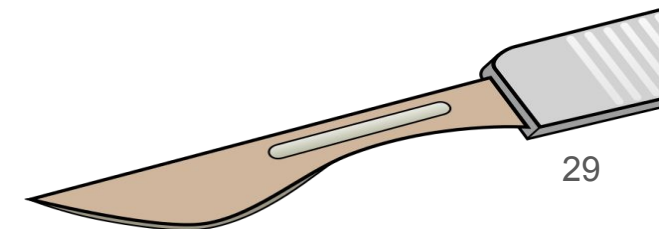
TEMPORARY HEMOSTASIS

Mechanical Procedure

- Limb elevation
- Direct pressure over a bleeding site
- Digital pressure over artery
- Extremity tourniquet
- Wound packing
- Vascular clamp



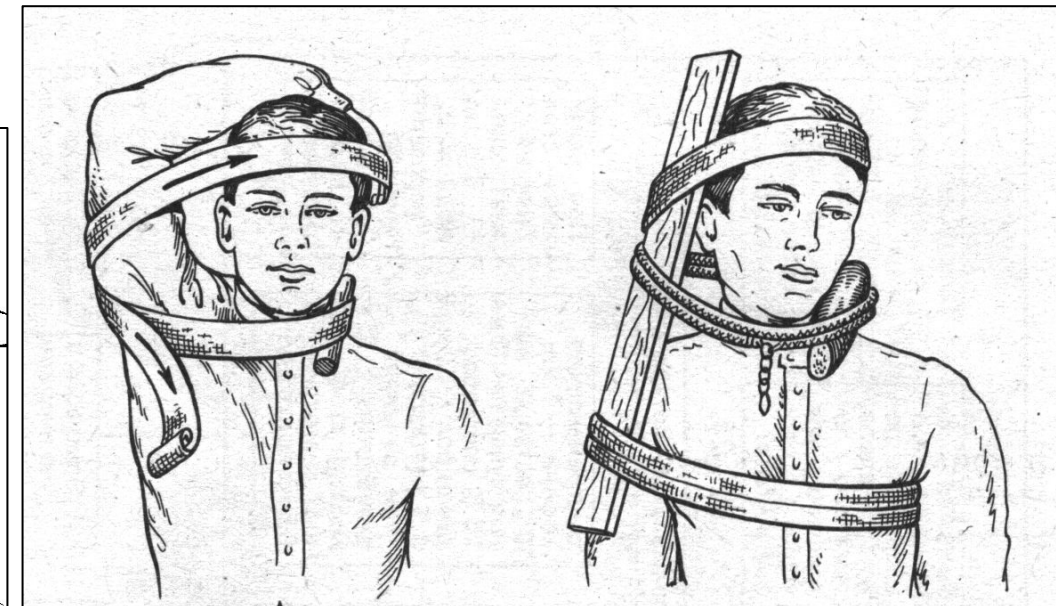
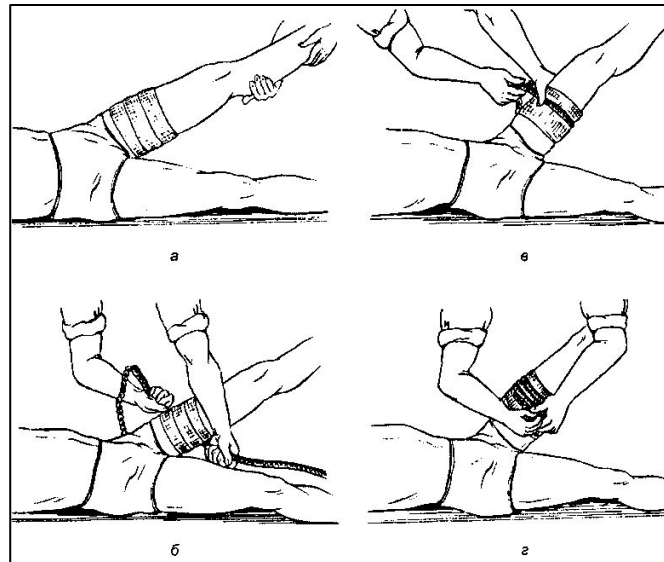
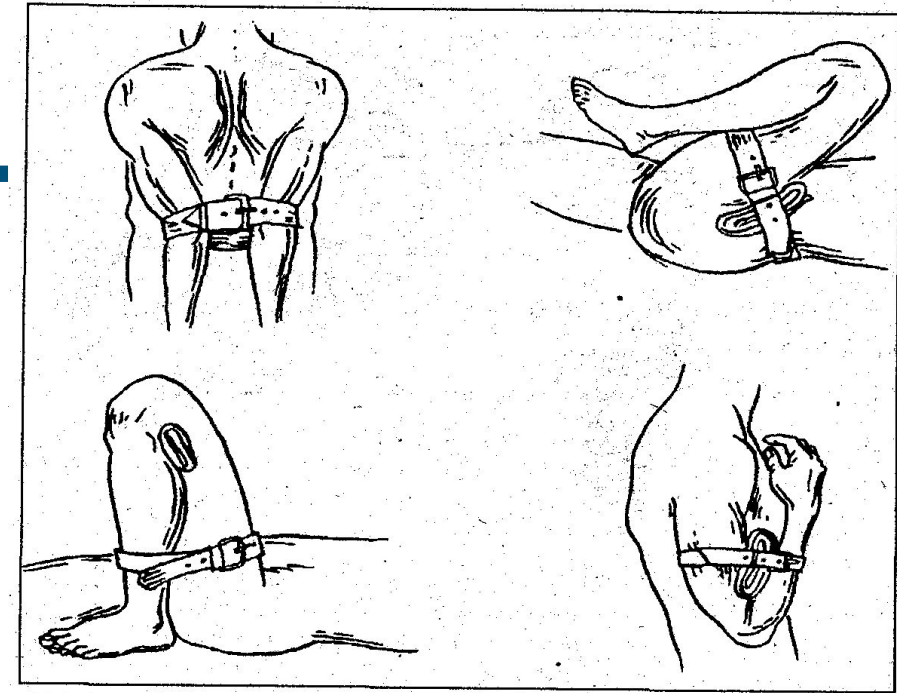
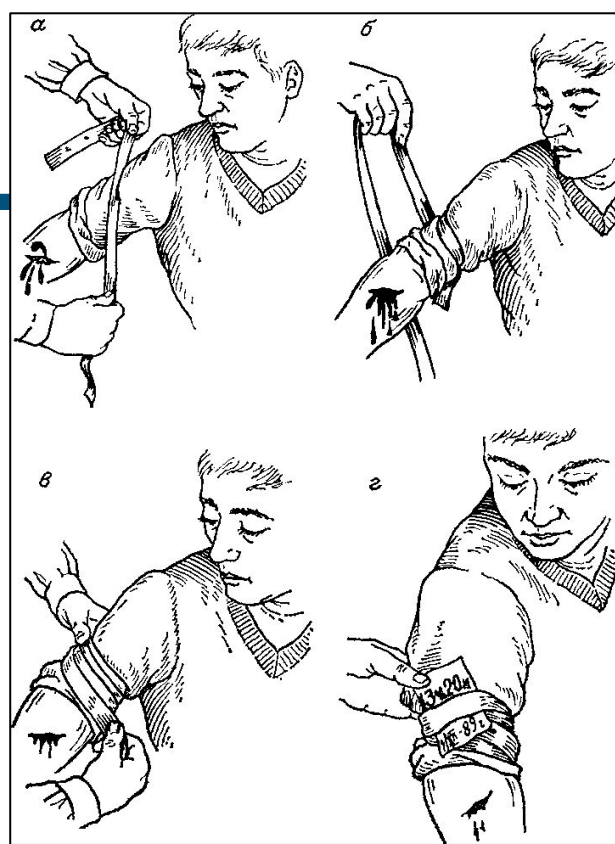
Direct pressure



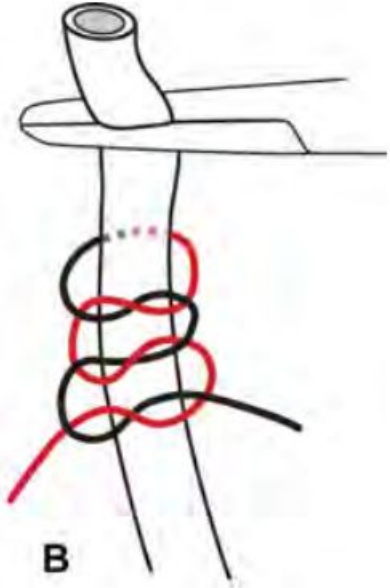
Extremity tourniquet

1. Elevate limb.
2. Apply tourniquet over clothing, 5-7 cm above the wound.
3. Apply tourniquet over the limb segment with single bone (arm or thigh)
4. Tighten the tourniquet until the bleeding stops or peripheral pulse disappears
5. Attach note with tourniquet application time
6. Send the patient for definite hemostasis.

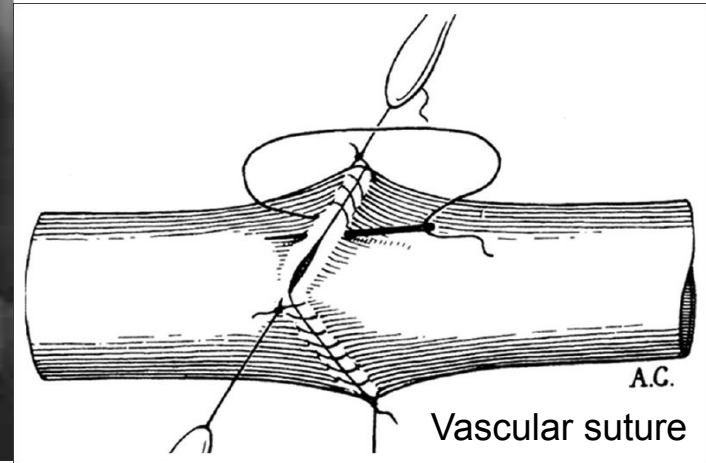
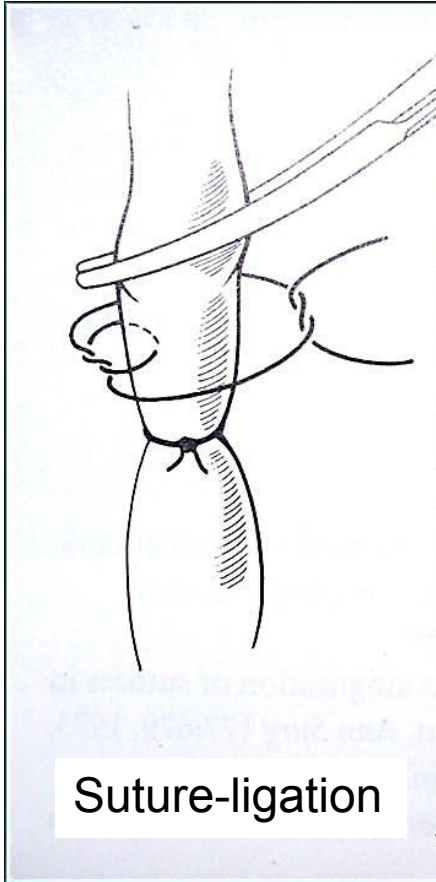
- *Tourniquet can be painful.*
- *Loose tourniquet can increase hemorrhage!*
- *Too tight tourniquet can crush tissues!*
- *Delay in tourniquet removal can cause gangrene!*



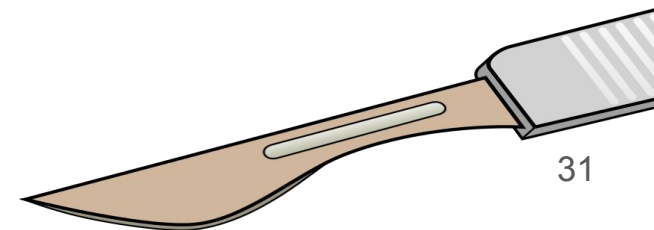
Definite hemostasis



Clamping and ligation



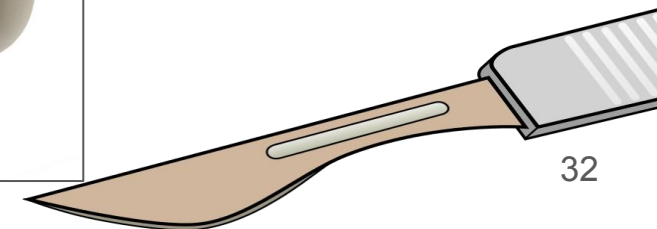
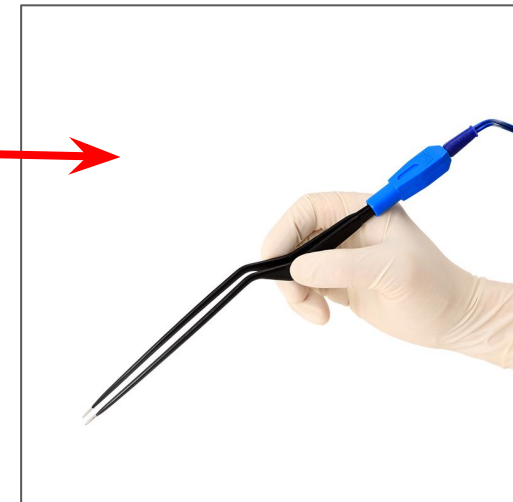
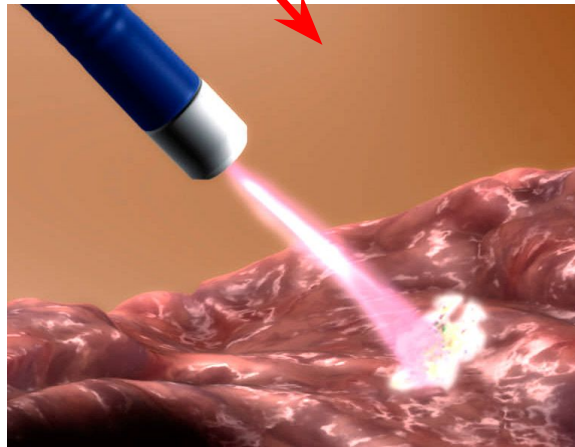
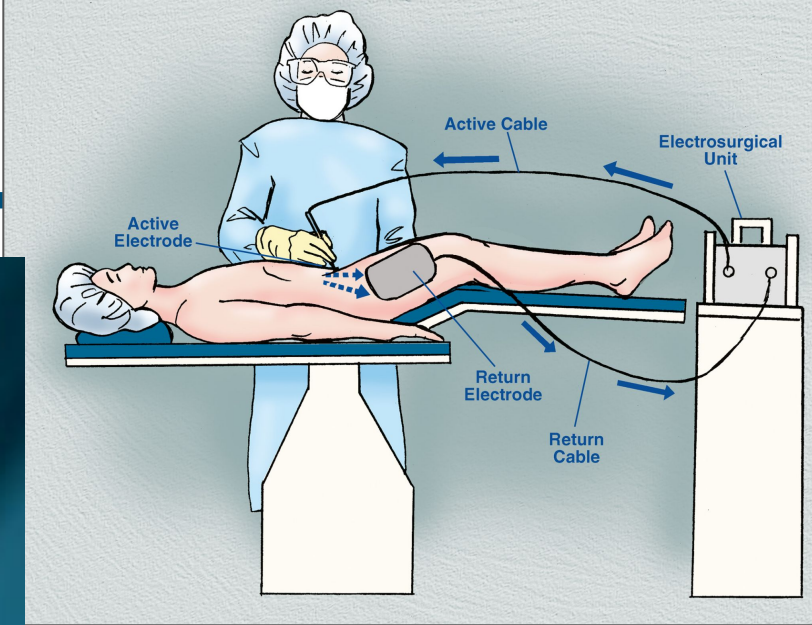
Vascular reconstruction with synthetic graft.



LOCAL HEMOSTASIS

Thermal Agents

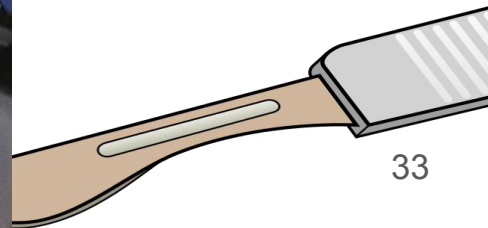
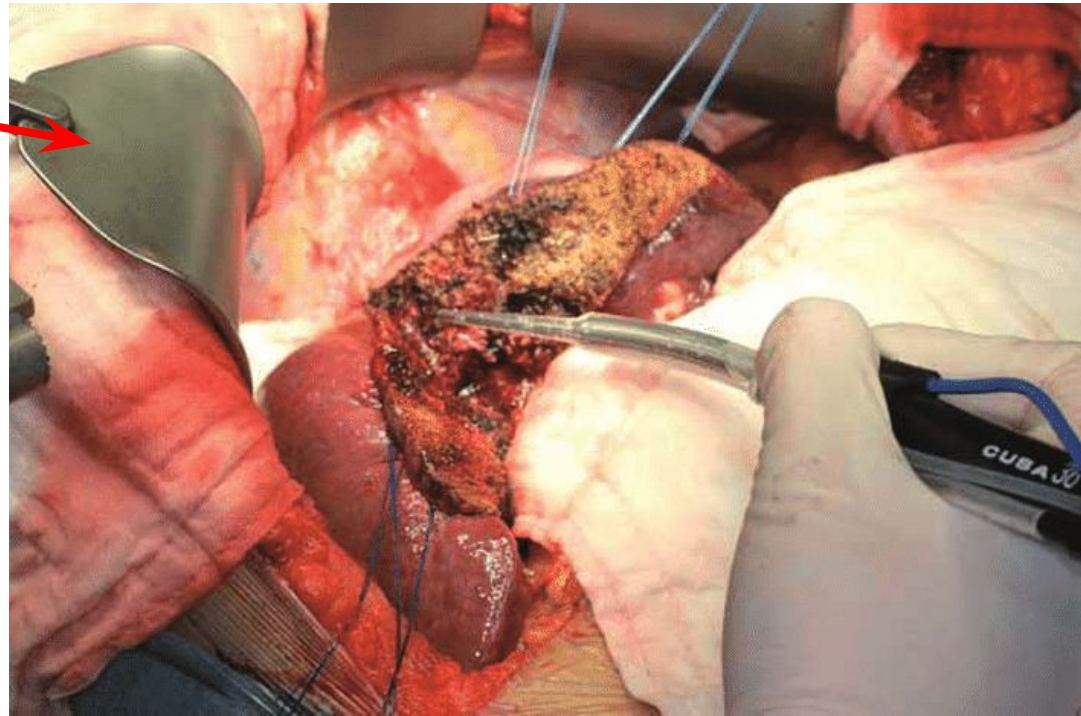
- Electrocautery - alternating current
 - ⇒ Heat ⇒ denaturation of protein
 - hazard of explosion
 - Monopolar
 - Bipolar
 - Bipolar vessel sealing (LigaSure™)
- Argon beam coagulator



LOCAL HEMOSTASIS

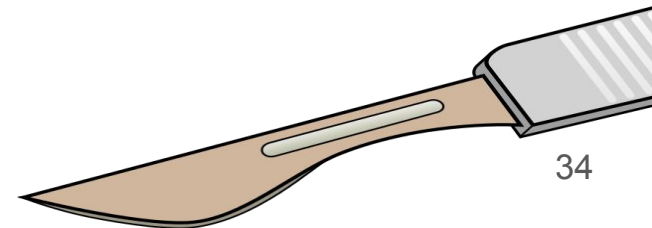
Thermal Agents

- Laser
- Radiofrequency energy
- Harmonic scalpel (Ultrasound)
- Cavitron ultrasonic suction aspirator (CUSA)



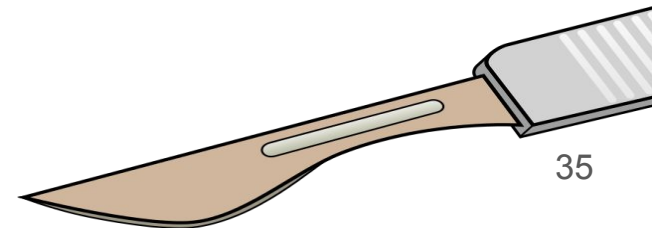
Topical Hemostatic Agents

- Oxidized cellulose
 - Surgicel
 - Low pH
 - Bactericidal
- Gelatin sponge
 - Gelfoam/Surgifoam
 - Neutral pH
 - Good carrier
- Microfibrillar collagen
 - Avitene
- Biodegradable matrix
- Absorb blood
- Activate platelets
- Induce coagulation
- Bovine thrombin
 - Semi-pure cow thrombin
 - Activates platelets and fibrin
 - Immunologic effects
- FloSeal
 - Bovine (now human) thrombin-gelatin sponge mix
 - Easy to use
- CoSeal
 - Polyethylene glycol glue
 - No obvious biologic effect/immunology



Topical Hemostatic Agents

- Fibrin sealants: Tisseel, Crosseal, Evaseal
 - Human thrombin fibrinogen mix
 - Some contain an antifibrinolytic (bovine aprotinin and TMA)
 - Hard to mix
 - Better sealant than hemostat
- BioGlue
 - Bovine albumin and glutaraldehyde
 - Easy to use
 - Good for aortic dissection
 - Direct contact harmful to exposed nerves and cardiac conduction tissue¹
 - Intraluminal fragments of glue may embolize^{1,2}
 - Can leak through suture-line needle holes¹
 - Fatal right ventricular infarction after embolism reported²

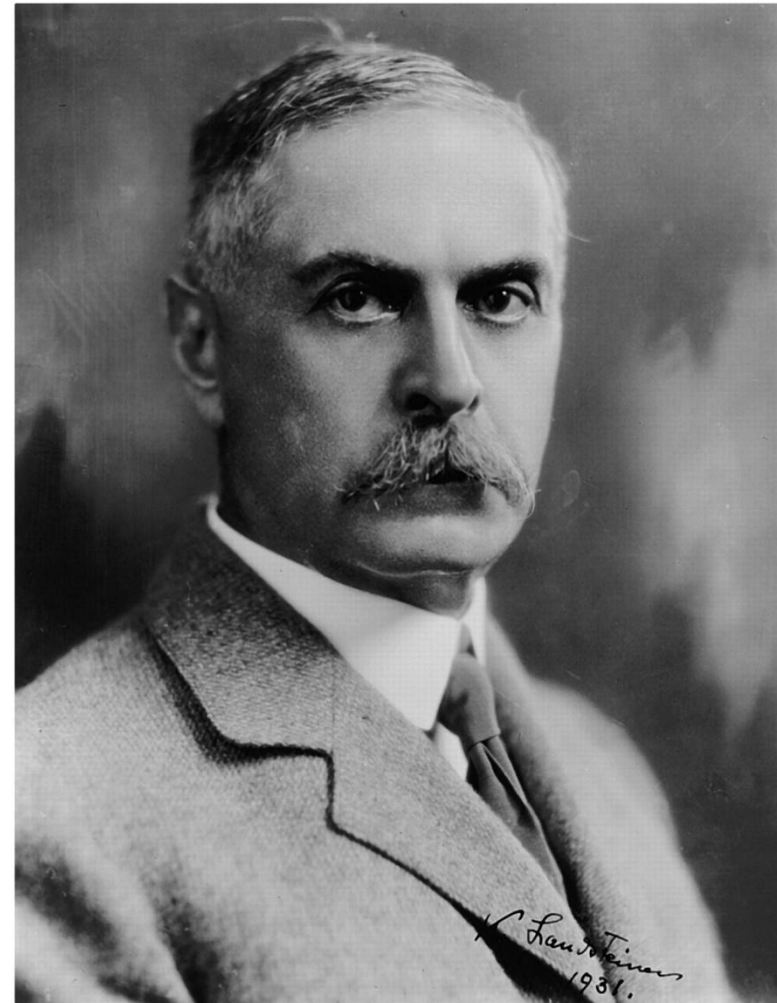


Human blood replacement therapy

Dr. Karl Landsteiner - A, B, and O groups in 1900.

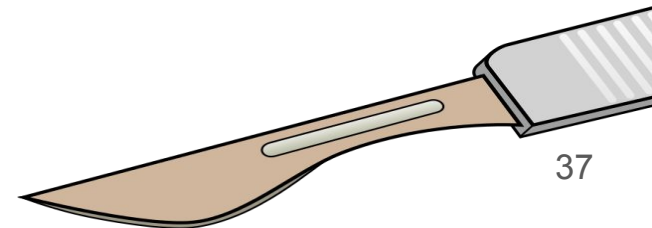
In 1939 Dr. Philip Levine and Dr. Rufus Stetson - Rh grouping.

Whole blood was considered the standard in transfusion until the late 1970s, when goal-directed component therapy began.



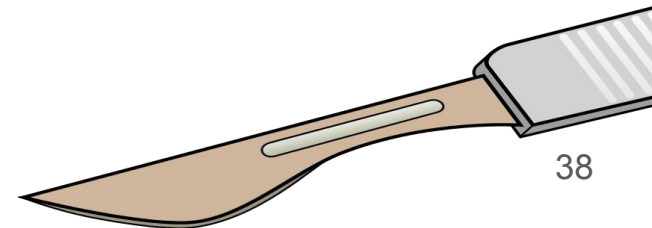
Hemotransfusion

- A, B, O, and Rh groups
 - Cross-matching between donors' red blood cells and recipients' sera;
 - Rh-negative recipients should receive transfusions only of Rh-negative blood;
 - Rh-positive blood should not be transfused to Rh-negative females who are of childbearing age;
 - In emergency situations, type O-negative blood may be transfused to all recipients.
 - Cross-matching should always be performed before the administration of dextran, because it interferes with the typing procedure.
-
- Banked whole blood, once the gold standard, is rarely available.
 - Fresh (24h) whole blood may be used in patients with trauma-associated coagulopathy in the combat situation.



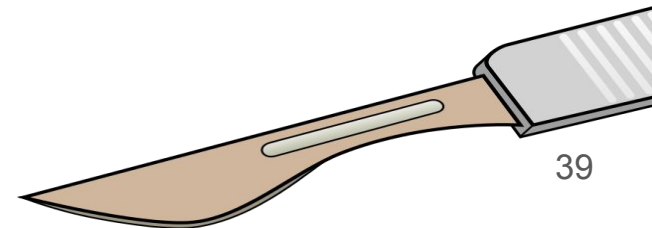
Red Blood cells

- Packed red blood cells are the product of choice for most clinical situations.
- Leukocyte-reduced and leukocyte-reduced/washed red blood cell
 - Filtration that removes approximately 99.9% of the white blood cells and most of the platelets.
 - Prevents almost all febrile, nonhemolytic transfusion reactions (fever and/or rigors), alloimmunization to HLA class I antigens, and platelet transfusion refractoriness as well as cytomegalovirus transmission.
 - It is the standard red blood cell transfusion.



- Platelet concentrate
 - shelf life of platelets is 120 hours from time of donation.
 - One unit - approximately 50mL.
 - Prevention of HLA alloimmunization can be achieved by leukocyte reduction through filtration.
- Fresh-frozen plasma (FFP) prepared from freshly donated blood;
- Human polymerized hemoglobin (PolyHeme) - artificial oxygen carrier
 - No crossmatch needed
 - No viral infections
 - Prolonged periods of storage.
 - Has not yet been approved for use in patients.

-





PRC	FFP	Plts	Cryo	PCC
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Red Cell Concentrates

Dose - in the absence of active bleeding, use the minimum number of units required to achieve a target Hb. Consider the size of the patient; assume an increment of 10g/L per unit for an average 70 kg adult.

R1 Acute Bleeding

Acute blood loss with haemodynamic instability. After normovolaemia has been achieved/maintained, frequent measurement of Hb (including by near patient testing) should be used to guide the use of red cell transfusion – see suggested thresholds below.

R2 Hb < 70g/L stable patient

Acute anaemia.
Use an Hb threshold of 70g/L and a target Hb of 70-90g/L to guide red cell transfusion. Follow local/ specific protocols for indications such as post cardiac surgery, traumatic brain injury, acute cerebral ischaemia.

R3 Hb < 80g/L if cardiovascular disease

Use an Hb threshold of 80g/L and a target Hb of 80-100g/L.

R4 Chronic transfusion dependent anaemia

Transfuse to maintain an Hb which prevents symptoms.
Suggest an Hb threshold of 80g/L initially and adjust as required. Haemoglobinopathy patients require individualised Hb thresholds depending on age and diagnosis.

R5 Radiotherapy maintain Hb > 110g/L

There is limited evidence for maintaining an Hb of 110g/L in patients receiving radiotherapy for cervical and possibly other tumours.



PRC	FFP	Plts	Cryo	PCC
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Fresh frozen plasma

Dose - 15 ml/kg body weight, often equivalent to 4 units in adults.

F1 Major haemorrhage

Early infusion of FFP is recommended in a ratio of 1 unit FFP:1 unit red cells for trauma and at least 1 unit FFP: 2 units red cells in other major haemorrhage settings. Once bleeding is under control, FFP use should be guided by timely tests for coagulation as indicated below.

F2 PT Ratio / INR >1.5 with bleeding

Clinically significant bleeding without major haemorrhage. FFP required if coagulopathy. Aim for a PT and APTT ratio of ≤ 1.5 .

F3 PT Ratio / INR >1.5 and pre-procedure

Prophylactic use when coagulation results are abnormal e.g. disseminated intravascular coagulation and invasive procedure is planned with risk of clinically significant bleeding.

F4 Liver disease with PT Ratio/INR >2 and pre-procedure

FFP should not be routinely administered to non-bleeding patients or before invasive procedures when the PT ratio/ INR is ≤ 2 .

F5 TTP / plasma exchange.

F6 Replacement of single coagulation factor.



PRC	FFP	Plts	Cryo	PCC
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Platelet concentrates

Dose – for prophylaxis, do not routinely transfuse more than 1 adult therapeutic dose.

Prior to invasive procedure or to treat bleeding, consider the size of the patient, previous increments and the target count.

Prophylactic platelet transfusion

- **P1** Plt $< 10 \times 10^9/L$ reversible bone marrow failure

Note: Not indicated in chronic bone marrow failure

- **P2** Plt 10-20 $\times 10^9/L$ sepsis / haemostatic abnormality

- **P3** To prevent bleeding associated with invasive procedures

Note: Transfusion prior to a bone marrow biopsy is not required

- **P3a** Plt $< 20 \times 10^9/L$ central venous line

- **P3b** Plt $< 40 \times 10^9/L$ pre lumbar puncture / spinal anaesthesia

- **P3c** Plt $< 50 \times 10^9/L$ pre liver biopsy / major surgery

- **P3d** Plt $< 80 \times 10^9/L$ epidural anaesthesia

- **P3e** Plt $< 100 \times 10^9/L$ pre critical site surgery e.g. CNS

Therapeutic use to treat bleeding (WHO bleeding grade 2 or above)

- **P4a** Major haemorrhage Plt $< 50 \times 10^9/L$

- **P4b** Critical site bleeding (e.g. CNS / traumatic brain injury) Plt $< 100 \times 10^9/L$

- **P4c** Clinically significant bleeding Plt $< 30 \times 10^9/L$

Specific clinical conditions

Table 1. Indications for Transfusion of Plasma Products

<i>Indication</i>	<i>Associated condition/additional information</i>
International Normalized Ratio > 1.6	Inherited deficiency of single clotting factors with no virus-safe or recombinant factor available—anticoagulant factors II, V, X, or XI Prevent active bleeding in patient on anticoagulant therapy before a procedure Active bleeding
Emergent reversal of warfarin (Coumadin)	Major or intracranial hemorrhage Prophylactic transfusion in a surgical procedure that cannot be delayed
Acute disseminated intravascular coagulopathy	With active bleeding and correction of underlying condition
Microvascular bleeding during massive transfusion	≥ 1 blood volume (replacing approximately 5,000 mL in an adult who weighs 155.56 lb [70 kg])
Replacement fluid for apheresis in thrombotic microangiopathies	Thrombotic thrombocytopenic purpura; hemolytic uremic syndrome
Hereditary angioedema	When C1 esterase inhibitor is unavailable ⁹

Information from references 7 through 9.

Table 4. Indications for Transfusion of Cryoprecipitate

Adults	Neonates (continued)
Hemorrhage after cardiac surgery	Anticoagulant factor XIII deficiency
Massive hemorrhage or transfusion	Congenital dysfibrinogenemia ¹²
Surgical bleeding	Congenital fibrinogen deficiency
Neonates	von Willebrand disease*
Anticoagulant factor VIII deficiency*	

**—Use when recombinant factors are not available.*

Information from references 12 and 15.

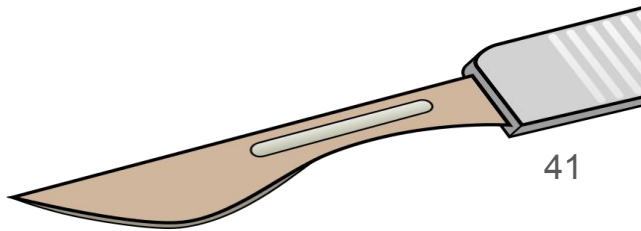


Table 2. Indications for Transfusion of Platelets in Adults

<i>Prophylactic transfusion indications</i>	<i>Platelet count ($\times 10^3$ per μL)</i>
Major surgery or invasive procedure, no active bleeding	≤ 50
Ocular surgery or neurosurgery, no active bleeding	≤ 100
Surgery with active bleeding	< 50 (usually) > 100 (rarely)
Stable, nonbleeding	< 10
Stable, nonbleeding, and body temperature $> 100.4^\circ\text{F}$ (38°C) or undergoing invasive procedure	< 20

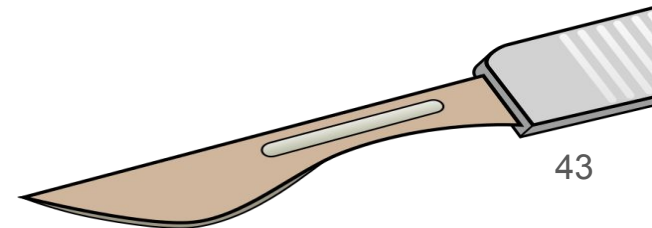
Information from reference 9.

Acute blood loss

- Anemia (Hb & Hct are misleading)
 - Transfusion Trigger – $\text{Hb} \leq 7 \text{ g/dL}$
- Hypovolemia:

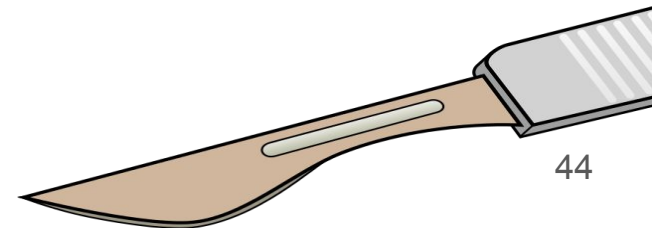
Hemorrhage	Blood loss		
Class I	<750 mL	<15%	Minor effects
Class II	750-1500 mL	15-30%	Tachycardia and decreased pulse pressure but, normal BP
Class III	1500-2000 mL	30-40%	Tachycardia, tachypnea, hypotension, oliguria, and changes in mental status
Class IV	>2000mL	>40%	Life-threatening

- blood loss of up to 1000 mL can be compensated by the body's solution.



Damage control resuscitation

- Old strategy:
 - Start resuscitation with crystalloid
 - Followed by packed red blood cells
 - Transfusion of units of plasma or platelets
 -



Damage control resuscitation

A. Initial Transfusion of Red Blood Cells (RBCs):

1. Notify Blood Bank immediately of urgent need for RBCs.

O Negative Uncrossmatched (available immediately).

As soon as possible switch to O-negative for females and O-positive for males

Type-Specific Uncrossmatched (available in approximately 5–10 minutes)

Completely Crossmatched (available in approximately 40 minutes)

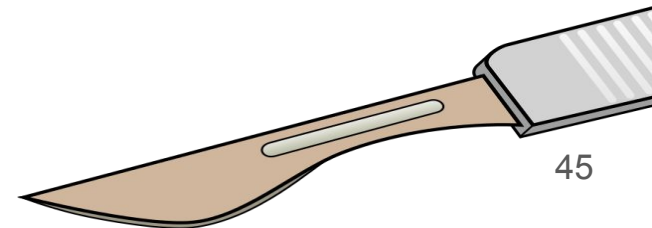
2. A blood sample must be sent to Blood Bank for a Type & Cross.
3. The Emergency Release of Blood form must be completed. If the blood type is not known and blood is needed immediately, O Negative RBCs should be issued.
4. RBCs will be transfused in the standard fashion. All patients must be identified (name and number) prior to transfusion.
5. Patients who are unstable or receive 1–2 RBCs and do not rapidly respond should be considered candidates for the massive transfusion (MT) guideline.

- CBC
- INR, fibrinogen
- pH and/or Base deficit
- TEG, where available

B. Adult Massive Transfusion Guideline:

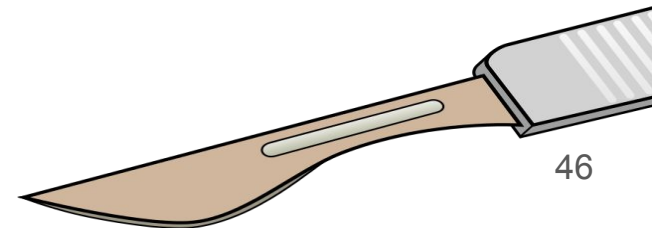
Keep platelet counts >100,000/L Cryoprecipitate
If fibrinogen ≤100 mg/dL, give 20 units of cryoprecipitate

1. The **Massive Transfusion Guideline (MTG)** should be initiated as soon as it is anticipated that a patient will require massive transfusion (≥10 U RBCs in 24 hours). The Blood Bank should strive to deliver plasma, platelets, and RBCs in a 1:1:1 ratio. To be effective and minimize further dilutional coagulopathy, the 1:1:1 ratio must be initiated early, ideally with the first 2 units of transfused RBCs. Crystalloid infusion should be minimized.



Complications of Transfusion

- Transfusion-related events - 10%; <0.5% are serious.
- Febrile nonhemolytic reactions
 - Cytokines
 - Use of leukocyte-reduced blood products
 - Pretreatment with acetaminophen
- Bacterial contamination
 - Fever, chills, tachycardia, and hypotension, and GI symptoms (abdominal cramps, vomiting, and diarrhea), hemoglobinemia, hemoglobinuria, and disseminated intravascular coagulation.
 - Discontinue, blood cultured. oxygen, antibiotics

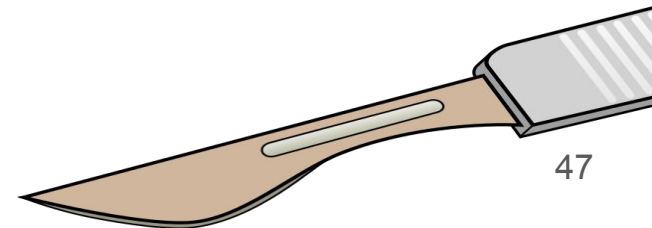


Complications of Transfusion

- Allergic reactions
 - Anaphylactic shock (rare)
 - rash, urticaria, and fever.
 - Treatment and prophylaxis
 - Antihistamines.
 - Epinephrine or steroids
- Respiratory compromise
 - Transfusion-associated circulatory overload
 - Central venous pressure monitoring
 - Diuresis, slowing the rate of blood administration

Transfusion-related acute lung injury (TRALI)

- Pulmonary edema related to transfusion
- Any plasma-containing blood product.
- Dyspnea and associated hypoxemia
- Anti-HLA or anti-human neutrophil antigen antibodies
- Discontinuation and provision of pulmonary support



Complications of Transfusion

Acute hemolytic reactions

- ABO-incompatible blood
- Fatal in up to 6%
- Immediate intravascular destruction of red blood cells and consequent hemoglobinemia and hemoglobinuria.
- DIC can be initiated
- Acute renal insufficiency, tubular necrosis

Delayed hemolytic transfusion reactions

- 2 to 10 days
- Extravascular hemolysis, mild anemia, and indirect hyperbilirubinemia.
- Has a low antibody titer
- Reactions of reticuloendothelial system to non-ABO antigens
- Pain at the site of transfusion, facial flushing, and back and chest pain. Fever, respiratory distress, hypotension, and tachycardia. Diffuse bleeding and hypotension.
- Hemoglobinuria
- Transfusion should be stopped immediately. Urine output, adequate hydration

