HEMOSTASIS, SURGICAL BLEEDING AND TRANSFUSION

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Classification

Haemorrhage in the surgical patient can be classified into 3 main categories:

- **Primary bleeding** – bleeding that occurs within the intra-operative period
  - This should be resolved during the operation, with any major haemorrhages recorded in the operative notes and the patient monitored closely post-operatively
- **Reactive bleeding** – occurs within 24 hours of operation
  - Most cases of reactive haemorrhage are from a ligature that slips or a missed vessel; these vessels can often be missed intraoperatively due to intraoperative hypotension and vasoconstriction, meaning only once the blood pressure normalises post-operatively will this bleeding occur
- **Secondary bleeding** – occurs 7-10 days post-operatively
  - Secondary haemorrhage is often due to erosion of a vessel from a spreading infection, such as when a heavily contaminated wound is closed primarily

Clinical Features and Assessment

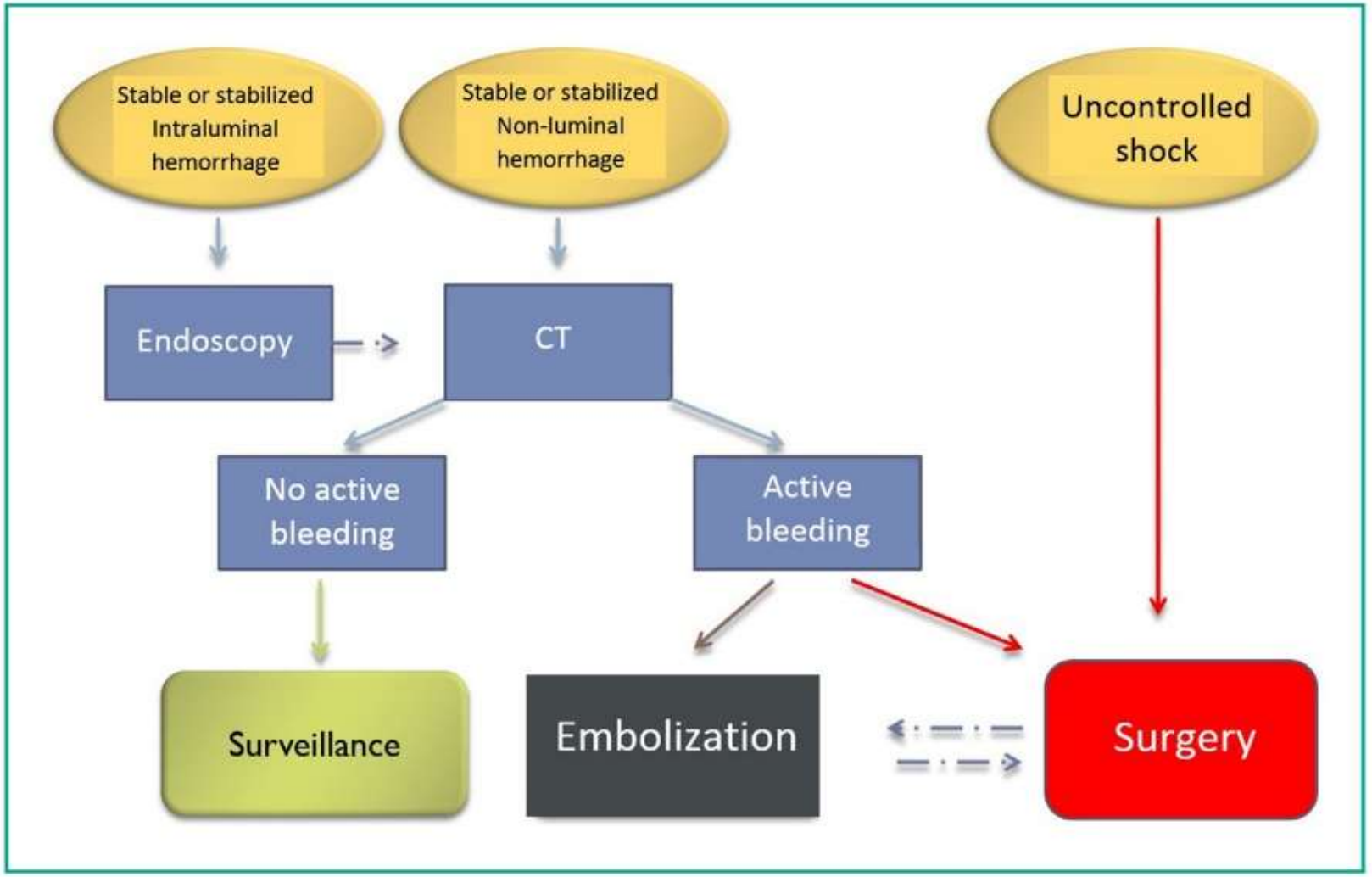
Clinical features of haemorrhagic shock* include **tachycardia**, **dizziness**, **agitation**, a **raised respiratory rate**, or a **decreased urine output**. Any external bleeding from a wound or drain will also be evident.

Examination of the patient should include a **thorough exposure** looking for bleeding, followed by systematic palpation of the surgical area looking for **swelling**, **discoloration**, **disproportionate tenderness**, and any peritonism (in abdominal cases). Review the **observations** and assess any **degree of shock** (see Table 1).

**Hypotension is often a late sign, it is important to not assume a patient is not bleeding just because their blood pressure is normal*

	Class I	Class II	Class III	Class IV
Blood Loss (ml)	<750ml	750-1500ml	1500-2000	>2000
Blood Loss (%)	<15%	15-30%	30-40%	>40%
Heart Rate	<100	100-120	120-140	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Respiratory Rate	14-20	20-30	30-40	>40
Urine Output (mL/hr)	>30	20-30	5-20	<5

Table 1 – Classification of Haemorrhagic Shock



Algorithm of patient management for postoperative hemorrhage.

Role of diagnostic radiology

A diagnosis of bleeding is suggested by a poor clinical condition that may include :

a state of shock, a decrease in hemoglobin and/or the presence of blood in the drain

Multidetector computed tomography (**MDCT**) is **the first-line imaging test** for the diagnosis of abdominal bleeding,postoperative or otherwis

Computed tomography features:

hemoperitoneum and hemoretroperitoneum are characterized by the visualization of a spontaneously high-attenuation mass or effusion

high-attenuated blood may be masked after contrast administration.

A non-enhanced sequence is therefore essential for detection of this entity

*__hematoma__ is seen as spontaneously high-attenuation round or oval shaped collection of blood. As with blood effusion

Figure 2. Comparison between sequence before (a) and after (b) iodinated contrast material administration for a cephalic post-pancreatectomy hemorrhage; spontaneous high attenuation is more visible on unenhanced images (arrow).

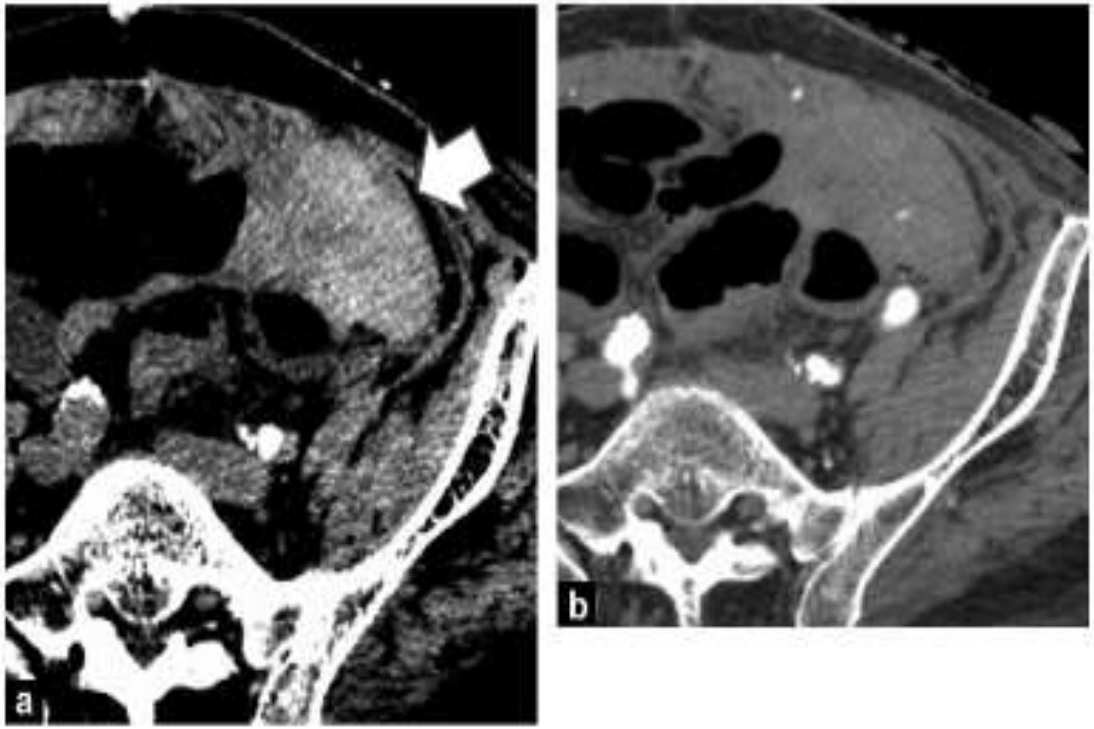


Figure 1. a: non-enhanced axial sequence showing a hematoma in the left iliac fossa following sigmoid surgery (arrow); b: the same sequence following contrast material administration which masks the high-attenuation. Note extravasation of the contrast material in the hematoma (active bleeding).

Computed tomography features:

- **the sentinel clot sign** refers to the fact that clotted blood can be found close to the area of active bleeding allowing localization of this area. This sign is not always seen

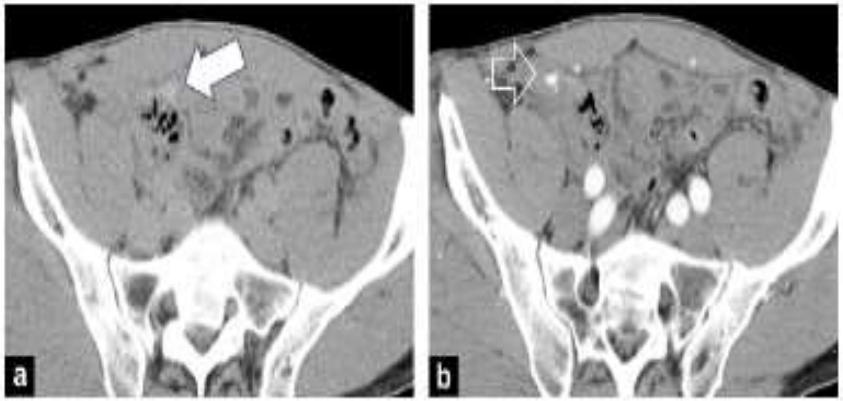


Figure 3. Postoperative bleeding from the small intestine during colon surgery: a: spontaneous high attenuation of small sentinel clot suggesting the origin of the bleeding (arrow); b following iodinated contrast material injection, visualization of small area of active bleeding (arrow).

extravasation of contrast material, also called blush, is a sign of active bleeding

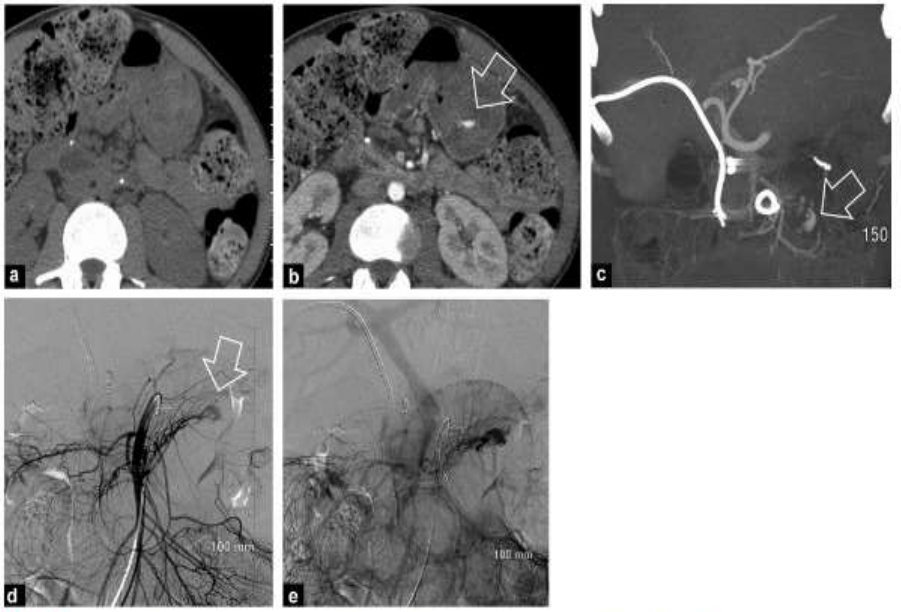


Figure 5. 27-year-old man who underwent a partial gastrectomy for a stenosing antropyloric ulcer. The patient presented with hypovolemic shock and endoluminal bleeding visible on MDCT (a: non-enhanced and b: following contrast material administration, arrow): c: MIP reconstruction showing the source of bleeding on a branch of the superior mesenteric artery (arrow). Arteriography confirms bleeding (d: arterial phase sequence with clearly visible blush; e: venous phase shows extravasation of contrast material).

Figure 4. The same patient as in Fig. 1. Extravasation of contrast material is clearly visible on arterial (a), portal (b) and delayed (c) phase sequences. Extravasation (arrows) extends over time, signaling active bleeding.

The role of interventional radiology

interventional radiology **is first-line technique for the management** of postoperative complication .

In this context, additional surgery in the early postoperative period is associated with significant mortality, especially following hepatectomy or pancreatectomy.

interventional radiology must be proposed as the first-line treatment .

embolization of the gastroduodenal artery has been shown to reduce the risk of re-bleeding in case of gastrointestinal bleeding that is not visible on arteriography(but has been confirmed by MDCT, endoscopy or based on clinical signs)

- finally, and more recently, in case of a bleeding ulcer in a context of intensive care, preventive occlusion of the gastroduodenal artery following successful endoscopic hemostasis reduces the rate of recurrence

Take-home messages

- Postoperative hemorrhage is always a serious event.
- The first-line technique for the investigation of digestive luminal hemorrhage is endoscopy.
- The first-line imaging technique for the investigation of non-luminal hemorrhage is MDCT including unenhanced, arterial, portal and delayed imaging phases.
- Embolization should be discussed as the first-line treatment if treatment is needed.

Bleeding

physiologic mechanism that stops bleeding after injury to the vasculature

- 1ry +2ry hemostasis

Local hemostasis Methods

- Mechanical
- Thermal
- Topical

HEMOSTASIS

Complex process whose function is to limit blood loss from an injured vessel

4 Major Events

- Vascular Constriction
- Platelet Plug Formation
- Fibrin Formation
- Fibrinolysis

Hemostasis

Vascular injury

Collagen

Tissue factor

Platelet activation

Coagulation cascade

Antithrombotic mechanisms

vWF, fibrinogen

Platelet adhesion, release and aggregation

Thrombin

Fibrinogen → Fibrin

Plasmin

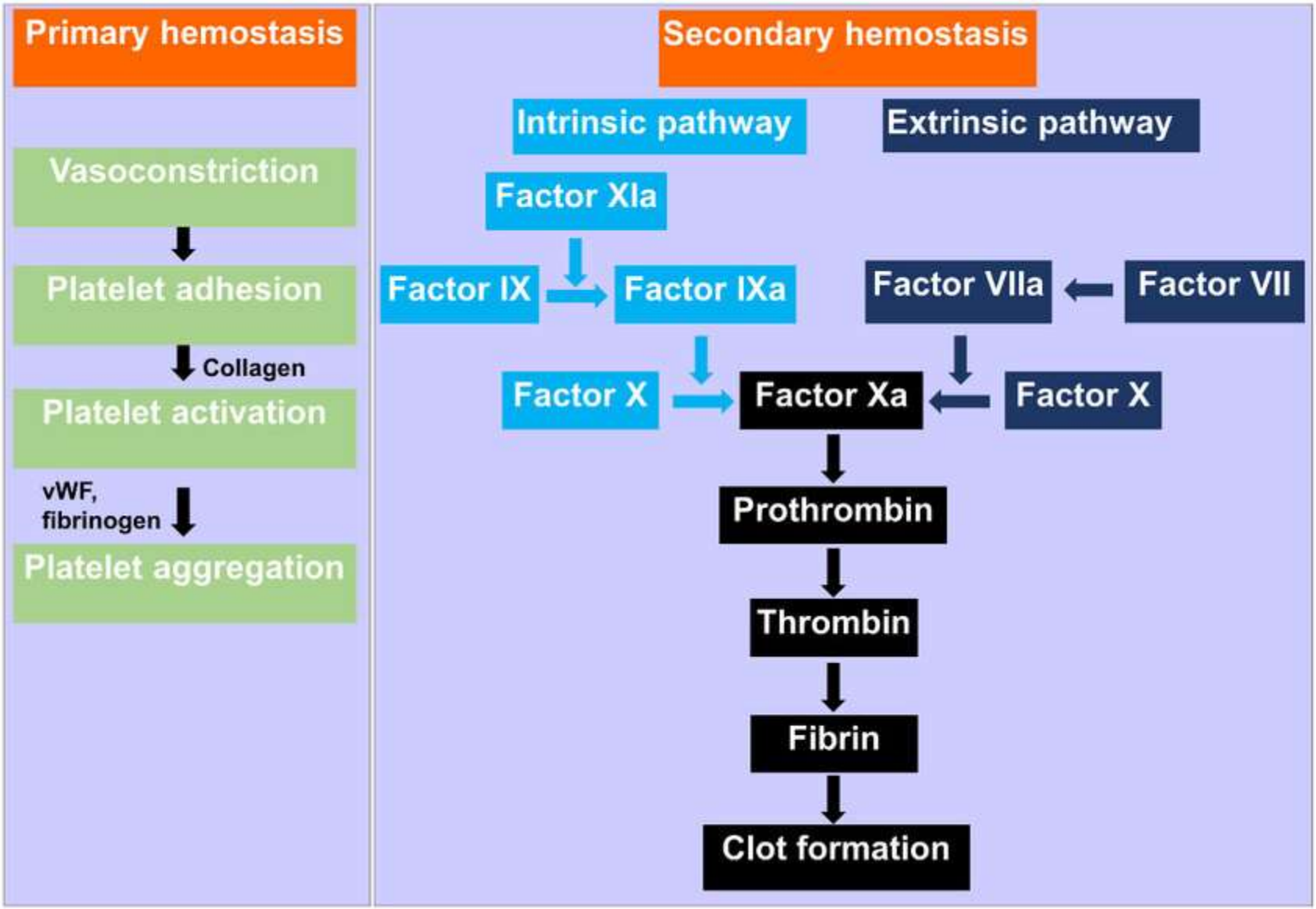
Fibrinolysis, clot dissolution

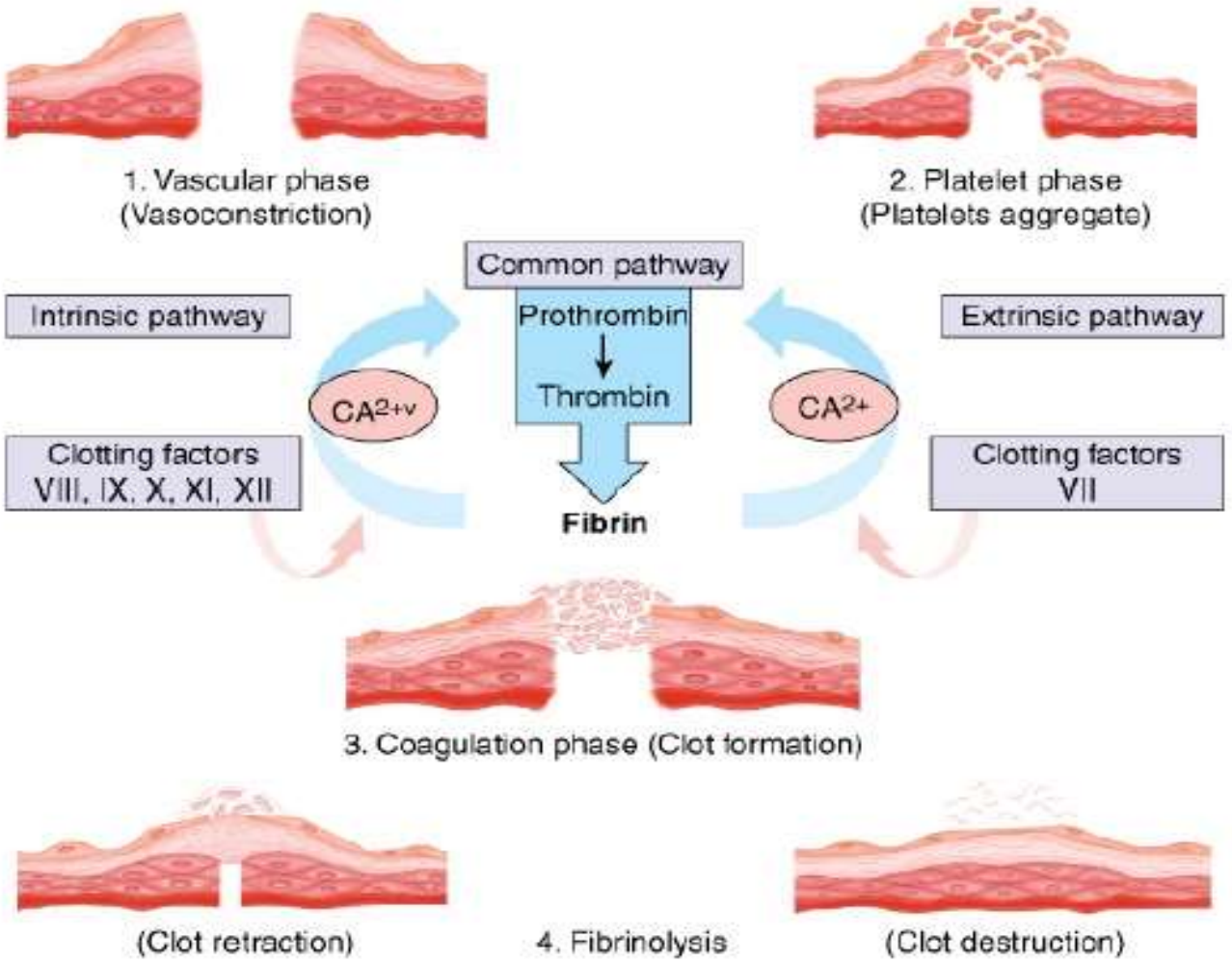
Platelet plug

Blood clot

Primary hemostasis

Coagulation


Primary hemostasis
Vasoconstriction
Platelet adhesion
Collagen
Platelet activation
vWF, fibrinogen
Platelet aggregation
Secondary hemostasis
Intrinsic pathway
Extrinsic pathway
Factor XIa
Factor IX
Factor IXa
Factor VIIa
Factor VII
Factor X
Factor Xa
Factor X
Prothrombin
Thrombin
Fibrin
Clot formation



Biology of hemostasis. The four physiologic processes that interrelate to limit blood loss from an injured vessel are illustrated and include vascular constriction, platelet plug formation, fibrin clot formation, and fibrinolysis.

Initial response to vascular injury

THROMBOXANE A2 (TXA2) : produced locally at site of injury via release of arachidonic acid from platelet membranes and is a potent constrictor of smooth muscle

ENDOTHELIN : synthesized by injured endothelium

SEROTONIN : released during platelet aggregation

BRADYKININ & FIBRINOPEPTIDES : vasoconstrictors also involved in coagulation cascade

→ Extent of vasoconstriction varies with degree of vessel injury

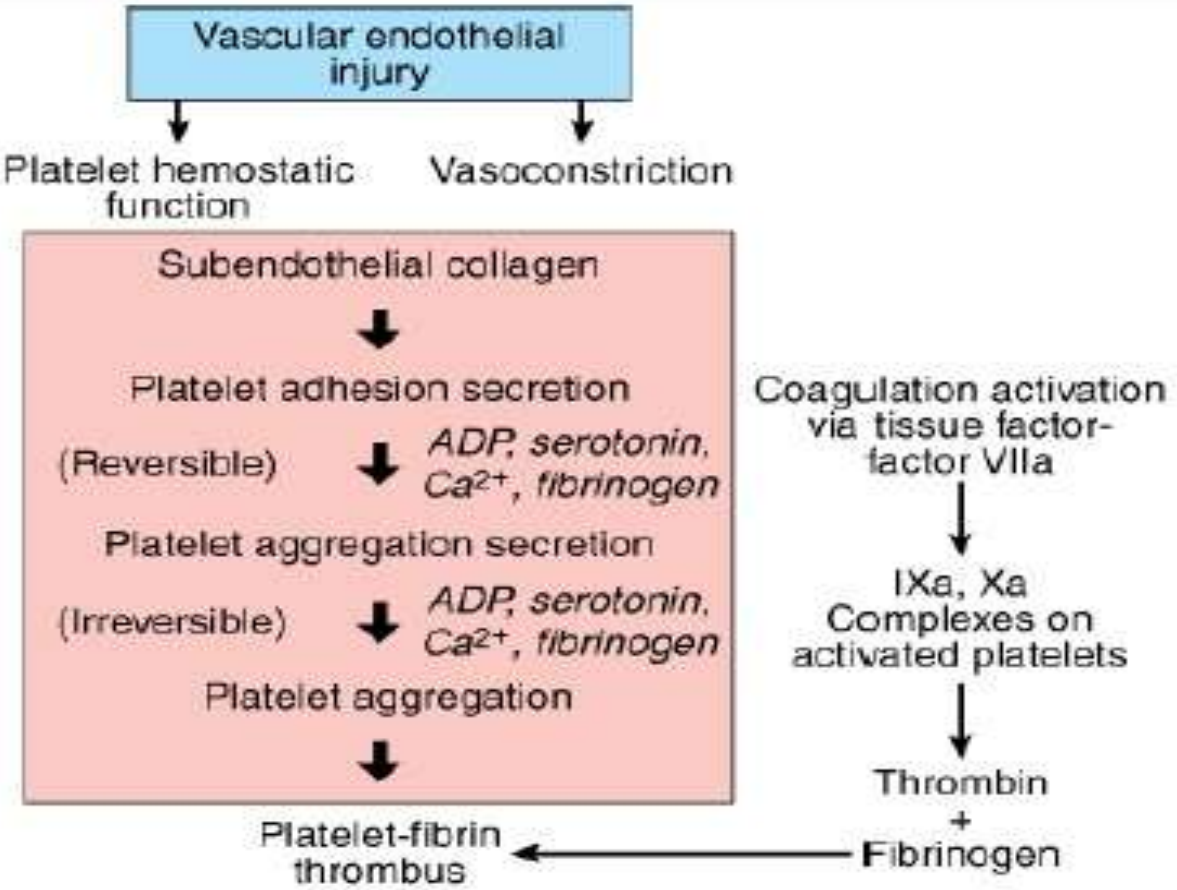
- **PLATELETS** : anucleate fragments of megakaryocytes (150T – 400T/uL)

  - hemostatic plug and contribute to thrombin formation

- Injury to the intimal layer → exposes subendothelial collagen →platelets adhere → **PLATELET ADHESION** → recruits other platelets from the circulating blood to seal the disrupted vessel → **PRIMARY HEMOSTASIS**

Vascular Constriction | Platelet Plug Formation | Fibrin Formation | Fibrinolysis

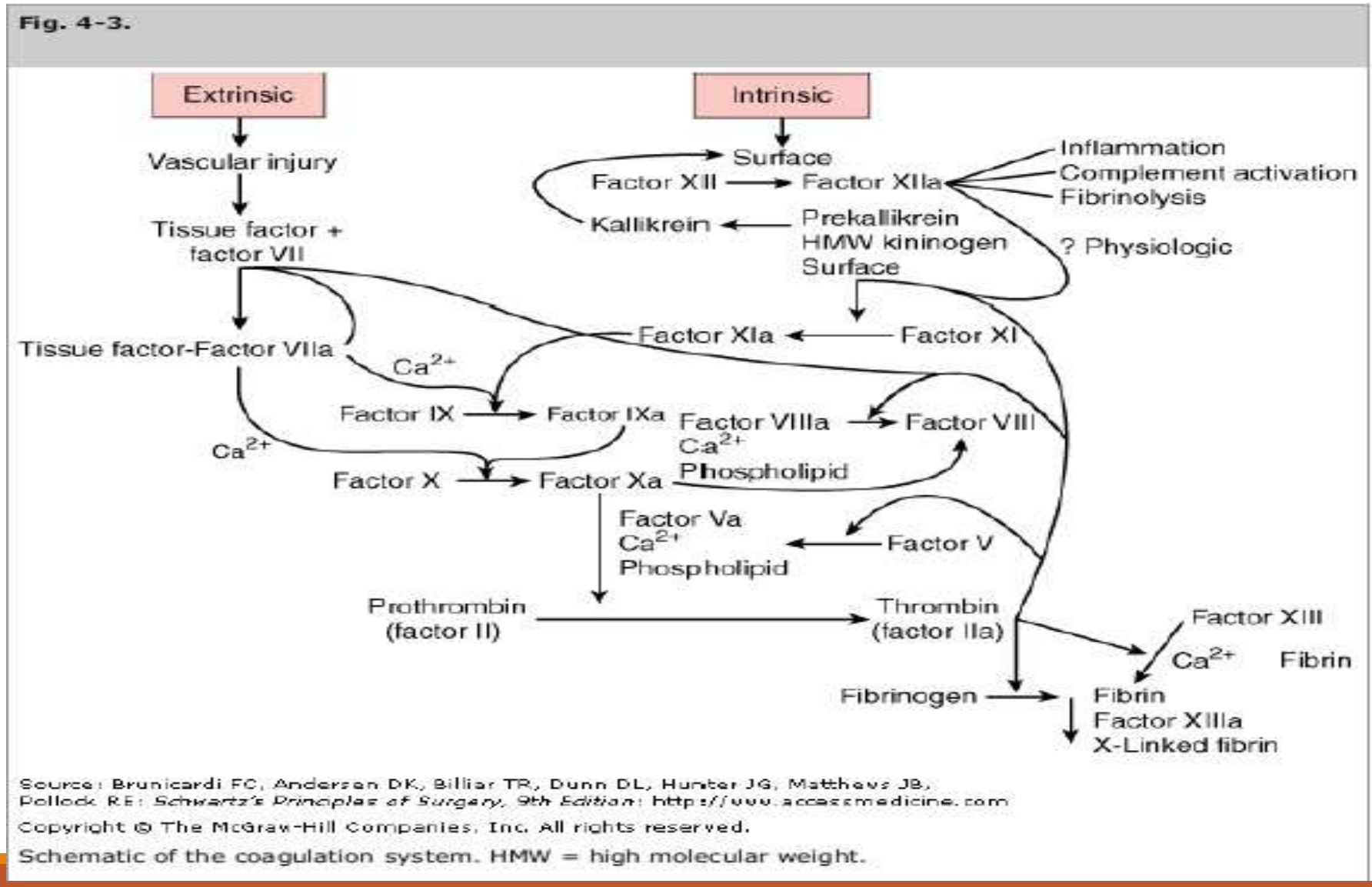
Fig. 4-2.



Schematic of platelet activation and thrombus function. ADP = adenosine diphosphate.

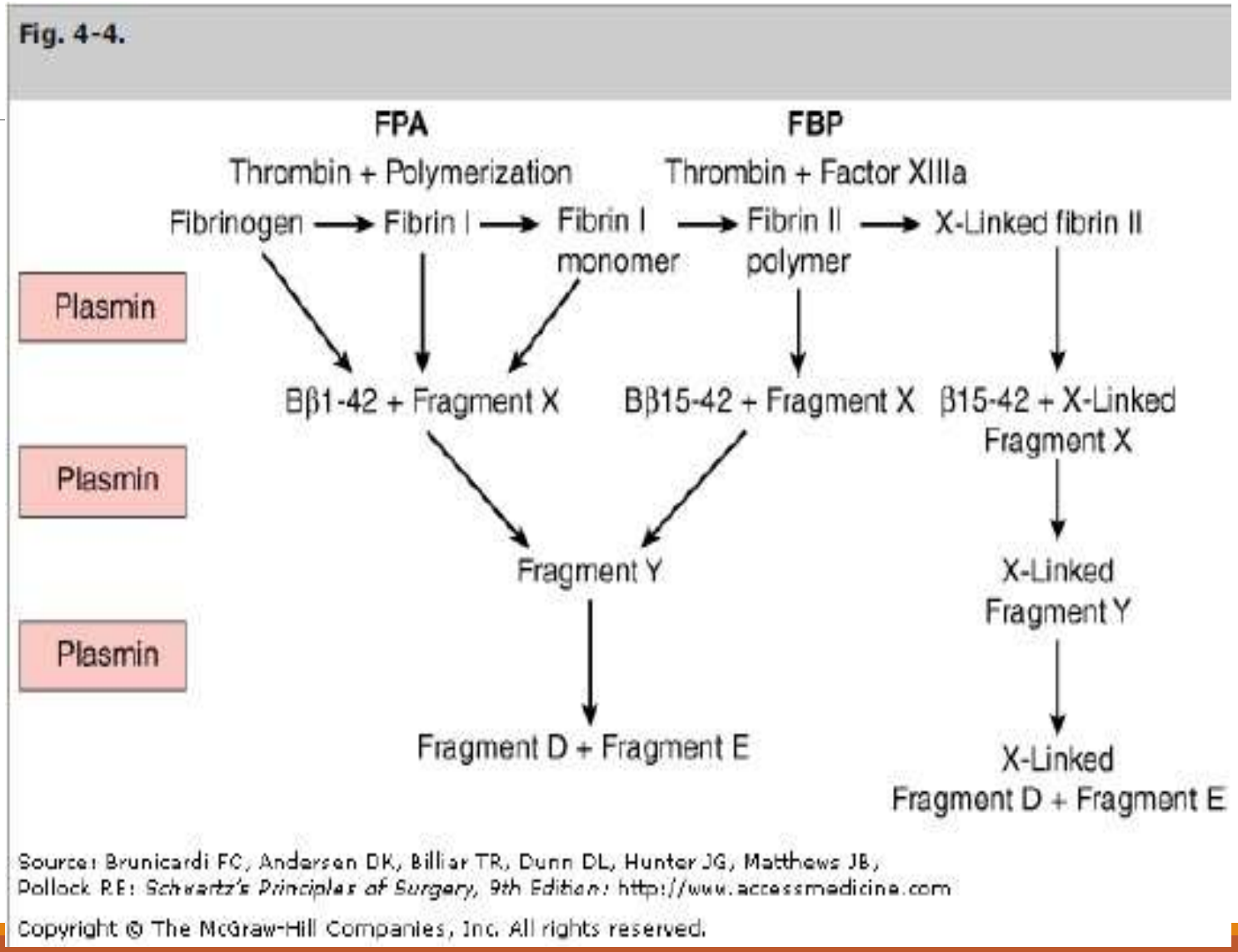
I fibrinogen
II prothrombin
V proaccelerin, accelerator globulin, labile factor
VII proconvertin, serum prothrombin conversion accelerator, stable factor
VIII antihemophilic factor, antihemophilic globulin
IX Christmas factor, plasma thromboplastin component
X Stuart-Prower factor
XI plasma thromboplastin antecedent
XII Hageman factor
XIII fibrinase, fibrin-stabilizing factor

Vascular Constriction → Platelet Plug Formation → Fibrin Formation → Fibrinolysis



Schematic of the coagulation system. HMW = high molecular weight.

Vascular Constriction → Platelet Plug Formation → Fibrin Formation → **Fibrinolysis**



PT = prothrombin time

- II,V, VII, X
- Fibrinogen

aPTT = activated partial thromboplastin time

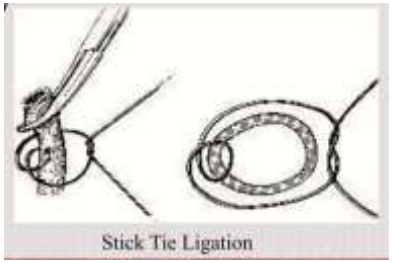
- XII, High molecular weight kininogen, Prekallikrein, XI, IX, VII, X, V , II and Fibrinogen

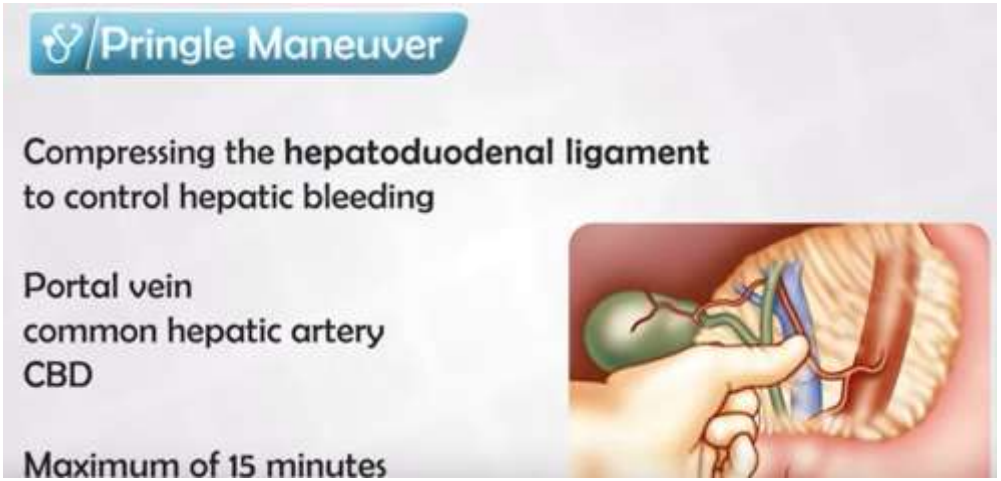
LOCAL HEMOSTASIS

GOAL IS TO PREVENT FURTHER BLOOD LOSS FROM A DISRUPTED VESSEL VIA DIRECT CLOSURE OF THE BLOOD VESSEL WALL DEFECT

MECHANICAL PROCEDURES

1. Direct pressure or by packs
2. Extremity tourniquet to occlude a major vessel proximal to bleeding site
3. Pringle maneuver for liver bleeding
4. Simple ligation for small vessels





THERMAL AGENTS

Heat denatures CHON → coagulation of large areas of tissue

Cautery

Amplitude setting must be high enough to produce prompt coagulation but not so high as to set up an arc between the tissue & cautery tip

TOPICAL HEMOSTATIC AGENTS

Ideal topical hemostatic agent has:

- Significant hemostatic action
- Shows minimal tissue reactivity
- Nonantigenic
- Provides ease of sterilization
- Low cost
- Tailored to specific needs

Table 4-6 Common Hemostatic Agents

Hemostatic Agent	Manufacturer	Cost	Comments
Thrombin Products			
Floseal	Baxter	$1500 per 6 pack/5 mL	Disseminated intravascular coagulation may result from intravascular exposure. Solution soaked in gauze or injected over wound bed, forming attachment.
Thrombostat	Parke-Davis	$56–60/5000–10,000 vial	
Thrombin-JMI	King Pharmaceuticals	$285/10,000 units	
Fibrin Sealant			
Tisseel	Baxter	$135/2 mL	Useful in skin grafts or anticoagulated patients. Crosseal contains no aprotinin, reduces anaphylaxis risk.
Crosseal	Johnson & Johnson	$100–150/1 mL	
Gelatin Agents			
Gelfoam	Pfizer	$90/1 g	Forms hydrated meshwork to promote clotting. Can swell. May cause granulomatous reaction.
Surgifoam	Johnson & Johnson	$8–14/gelatin square	

TRANSFUSION

HUMAN BLOOD REPLACEMENT THERAPY

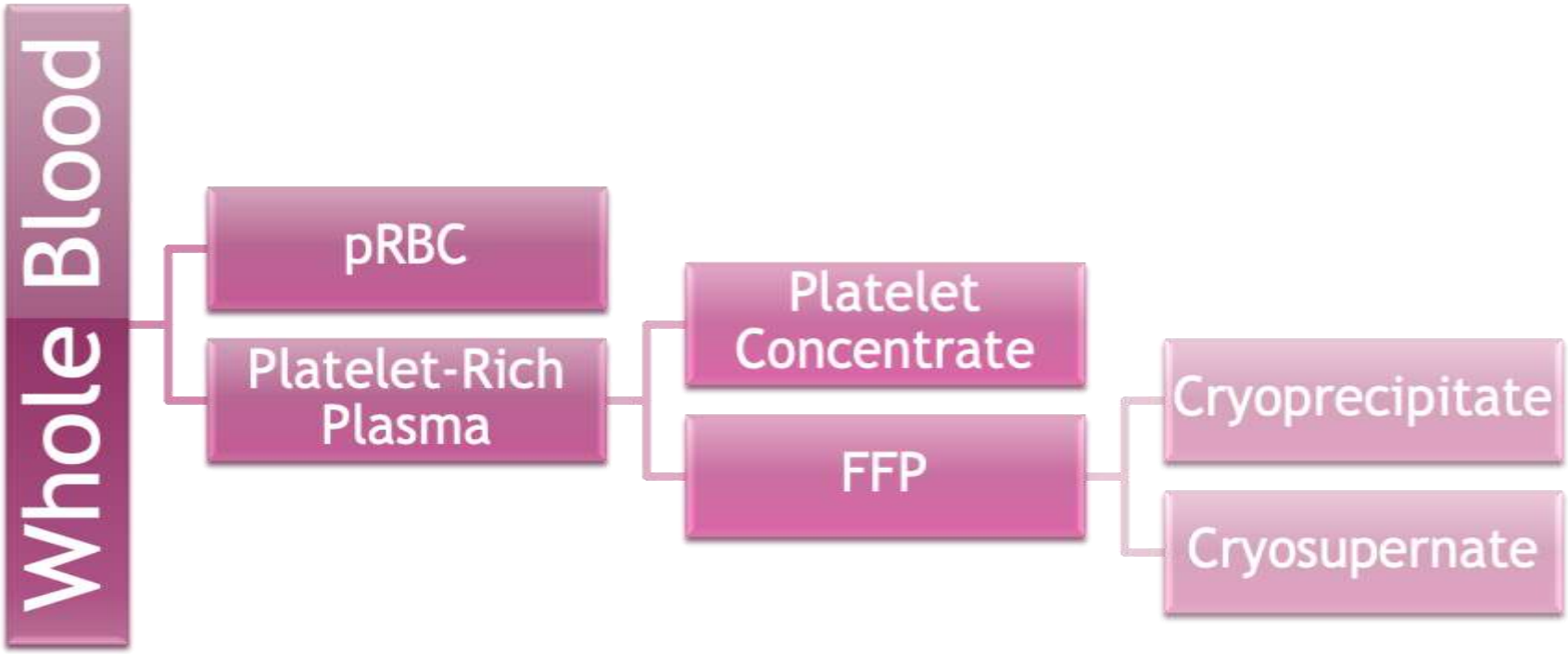
TYPING AND CROSS-MATCHING

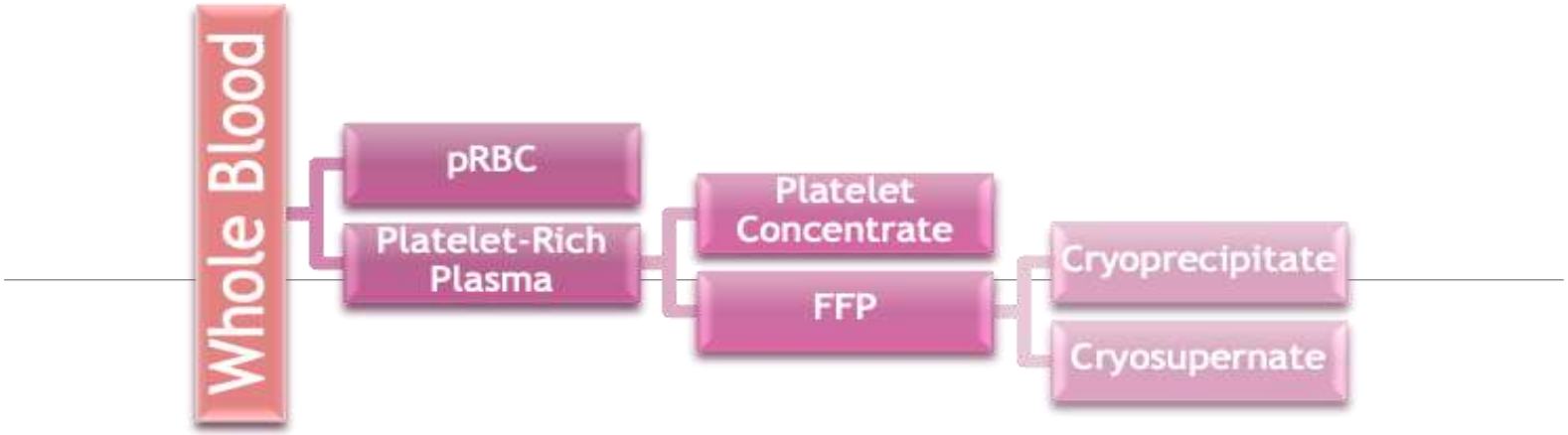
"O-" Blood

- Emergency situations
- > 4 U transfused → increased risk of hemolysis

Blood donors:

- Hgb > 11 g/dL or if Hct > 34%


Whole Blood
pRBC
Platelet-Rich Plasma
Platelet Concentrate
FFP
Cryoprecipitate
Cryosupernate

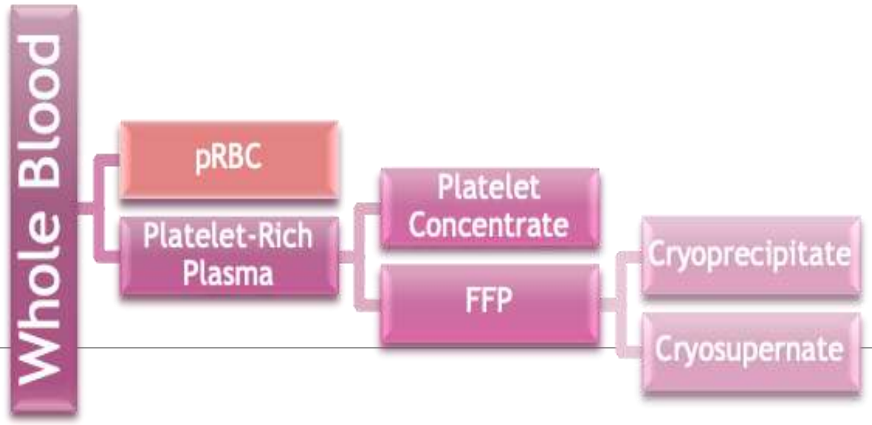


Banked whole blood

- Shelf life: 6 weeks

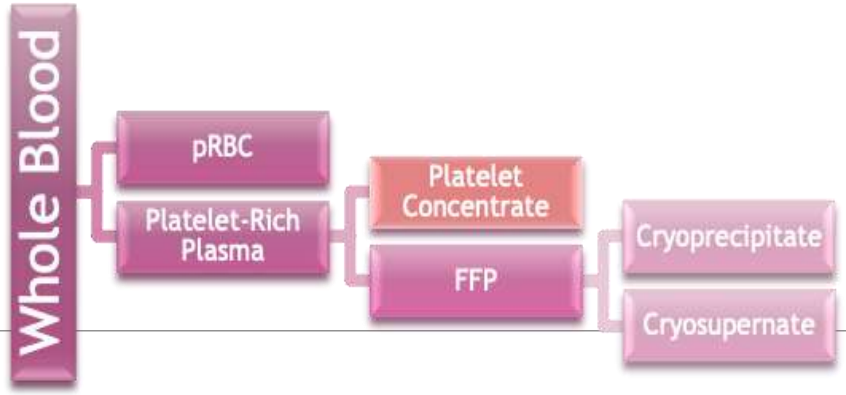
Fresh whole blood

- blood that is administered within 24 hours of its donation

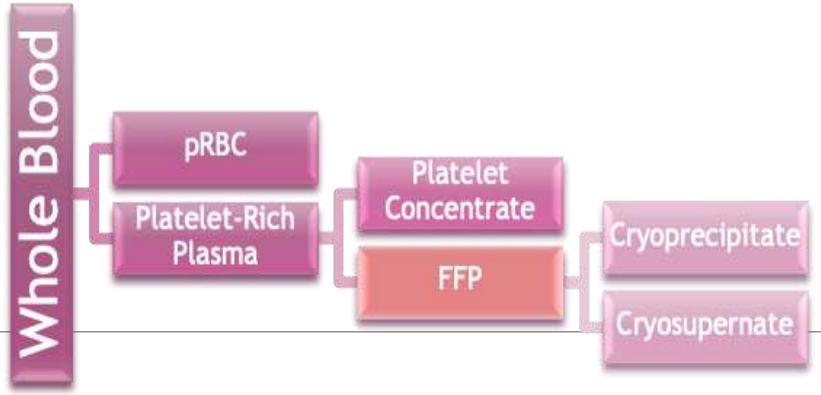


- Usual product of choice
- Shelf-life same as WB
- Leukocyte-reduced or Washed RBCs
  - prepared by filtration that removes approximately 99.9% of the white blood cells and most of the platelets (leukocyte-reduced red blood cells), and if necessary, by additional saline washing (leukocyte-reduced/washed red blood cells).

- For:
  - Chronic anemias, bone marrow & liver failure
  - Pre-op and post-op surgery
  - CHF, uremia



- Shelf-life: 120 hrs after donation
- 1 U per 10kg BW (1 U = 50 mL = 5.5 x 10^10)
- Apheresis = 4.4 x 10^10
- For:
  - Massive blood loss
  - Nonbleeding: plt < 20T (chemo, tumor invasion)
  - Pre-op/invasive procedure: plt < 50T
  - Documented bleeding: plt < 50T
  - Documented abnormal plt function

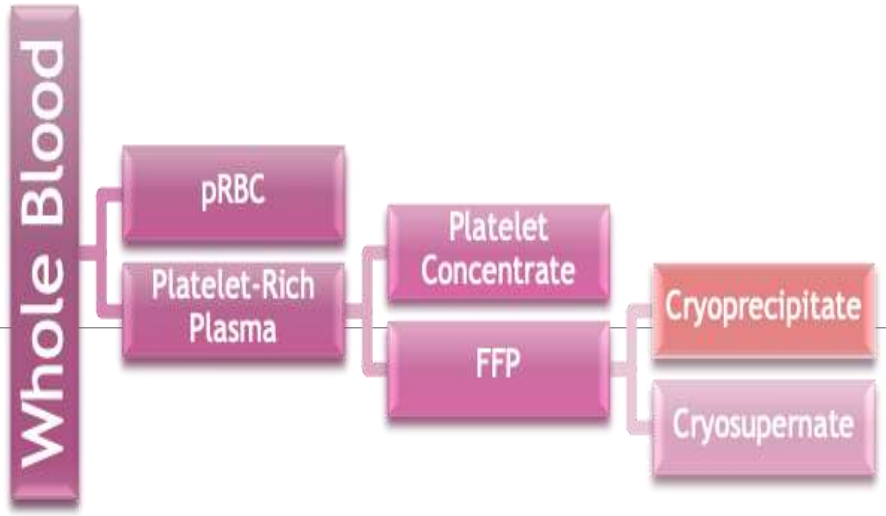


Usual source of Vit K-dependent factors

Only source of Factor V

For:

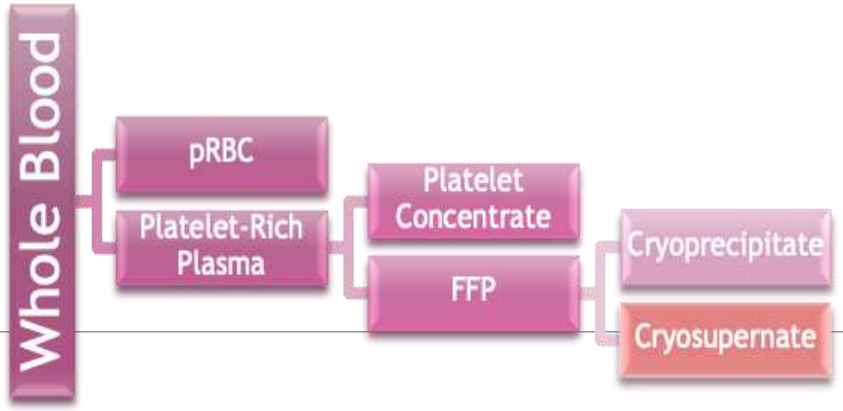
- Liver disease, Warfarin overdose
- DIC, TTP
- ↓CF d/t large volume transfusions



fibronectin, fibrinogen, CF VIII & XIII

For:

- Hemophilia A
- ↓fibrinogen, factor XIII
- Bleeding related to renal failure



Cryo-poor plasma, cryoprecipitate depleted

Factors II, VII, IX, XI

For:

- Hemophilia B
- Liver disease

INDICATIONS FOR BLOOD TRANSFUSION

Improvement in oxygen-carrying capacity

Treatment of anemia

- Hgb approaching 9 g/dL

Volume replacement

- Blood loss can be evaluated by estimation in wound, drapes, sponges, suctioned

INDICATIONS FOR BLOOD TRANSFUSION

COMPLICATIONS OF TRANSFUSION

TRANSFUSION REACTIONS

Reaction	Acute (mins-hrs)	Delayed (days-yrs)
Immune mediated	Hemolytic Febrile non-hemolytic Urticarial Anaphylactic	Alloimmune Hemolytic Post-transfusion purpura
Non-immune mediated	Hemolytic Circulatory Metabolic Embolic	Infectious Metabolic iron overload

TRANSFUSION REACTIONS

Symptoms in an **AWAKE** patient:

- Pain at site of transfusion
- Facial flushing
- Back & chest pain
- Fever
- Respiratory distress
- Hypotension
- tachycardia

TRANSFUSION REACTIONS

- Symptoms in an **ANESTHETIZED** patient:
  - Diffuse bleeding
  - Hypotension
- Laboratory criteria:
  - Hemoglobinuria, hemoglobinemia
  - Serologic findings
  - Positive Coomb's test
  - Jaundice
  - Low levels of haptoglobins

NONHEMOLYTIC REACTIONS

FEBRILE NONHEMOLYTIC REACTION

- Increase in temp >1°C associated with a transfusion (1% of transfusions)

Bacterial contamination of infused blood

- Yersinia enterocolitica, Pseudomonas
- Emergency!
  - Discontinue transfusion ASAP
  - Oxygen, adrenergic blocking agents, antibiotics

ALLERGIC REACTIONS

1% of all transfusions

Mild rash, urticaria & fever within 60-90 minutes of the start of transfusion

Treatment:

- Mild: Antihistamines
- Severe: Steroids or epinephrine

RESPIRATORY COMPLICATIONS

Associated with transfusion-associated circulatory overload

Occur with rapid infusion of blood, plasma expanders, crystalloids esp in older patients with heart disease

Rise in venous pressure, dyspnea, cough, rales in LLF

RESPIRATORY COMPLICATIONS

- **TRANSFUSION-RELATED ACUTE LUNG INJURY (TRALI)**
  - Noncardiogenic pulmonary edema related to transfusion
  - Occur with administration of any plasma-containing blood product
  - Fever, rigors, bilateral pulmonary infiltrates on CXR
  - Within 1-2 hrs after onset of transfiusion (before 6 hrs)

HEMOLYTIC REACTIONS

ACUTE HEMOLYTIC REACTION

- Administration of ABO-incompatible blood
- Fatal in 6% of cases
- Technical or clerical errors
- Administration of blood of the wrong blood type

HEMOLYTIC REACTIONS

IMMEDIATE HEMOLYTIC REACTION

- Intravascular destruction of RBCs & consequent hemoglobinemia & hemoglobinuria
- Acute renal insufficiency d/t toxicity associated with free Hgb in the plasma → tubular necrosis & precipitation of Hgb in tubules

HEMOLYTIC REACTIONS

- **DELAYED HEMOLYTIC REACTION**
- Occur 2-10 days after transfusion
- Extravascular hemolysis, mild anemia, indirect hyperbilirubinemia
- Low antibody titer at time of transfusion but titer increases after transfusion d/t anamnestic response

DISEASE TRANSMISSION

Malaria (P. malariae) , Chagas’ disease, brucellosis, syphilis.

TESTS OF HEMOSTASIS & BLOOD COAGULATION

Careful review of the patient's history

- Abnormal bleeding/bruising, drug use

Platelet count

- Plt > 1M/uL → Bleeding o r thrombotic complications
- Major surgical procedures: <100T/uL
- Minor surgical procedures: <50T/uL
- Spontaneous bleeding: <20T/uL

TESTS OF HEMOSTASIS & BLOOD COAGULATION

PT	aPTT	INR	BT
For: Vit K def; warfarin therapy	For: heparin therapy	Measured PT divided by Control PT N: 2-3	Ivy test; Bleeding must stop in 7mins
VII	XII		
	HMWK		
	Prekallikrein		
	XI		
	IX		
	VIII		
X			
V			
II (Prothrombin)			
Fibrinogen			

Thank you