Saving Lives at Speed

Exploring Haemostasis @ Ground Level

Thursday 28 May 2026: NZ College of Critical Care Nurses.

Suzi Rishworth, Transfusion Nurse Specialist, New Zealand Blood Service -Dunedin

Opening Karakia

Kia tau ngā manaakitanga o te wāhi ngaro

ki runga, ki tēnā, ki tēnā o tātou

Kia mahea te hua mākihikihi

kia toi te kupu, toi te mana, toi te aroha, toi te reo

Kia tūturu, kia whakamaua, kia tīna! Tīna!

Hui e, Tāiki e!

Let the strength and life force of our ancestors

Be with each and every one of us

Freeing our path from obstruction

So that our words, spiritual power, love, and language are upheld;

Permanently fixed, established and understood!

Forward together!

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NZCCN
28 May 2026

suzi.rishworth@nzblood.co.nz

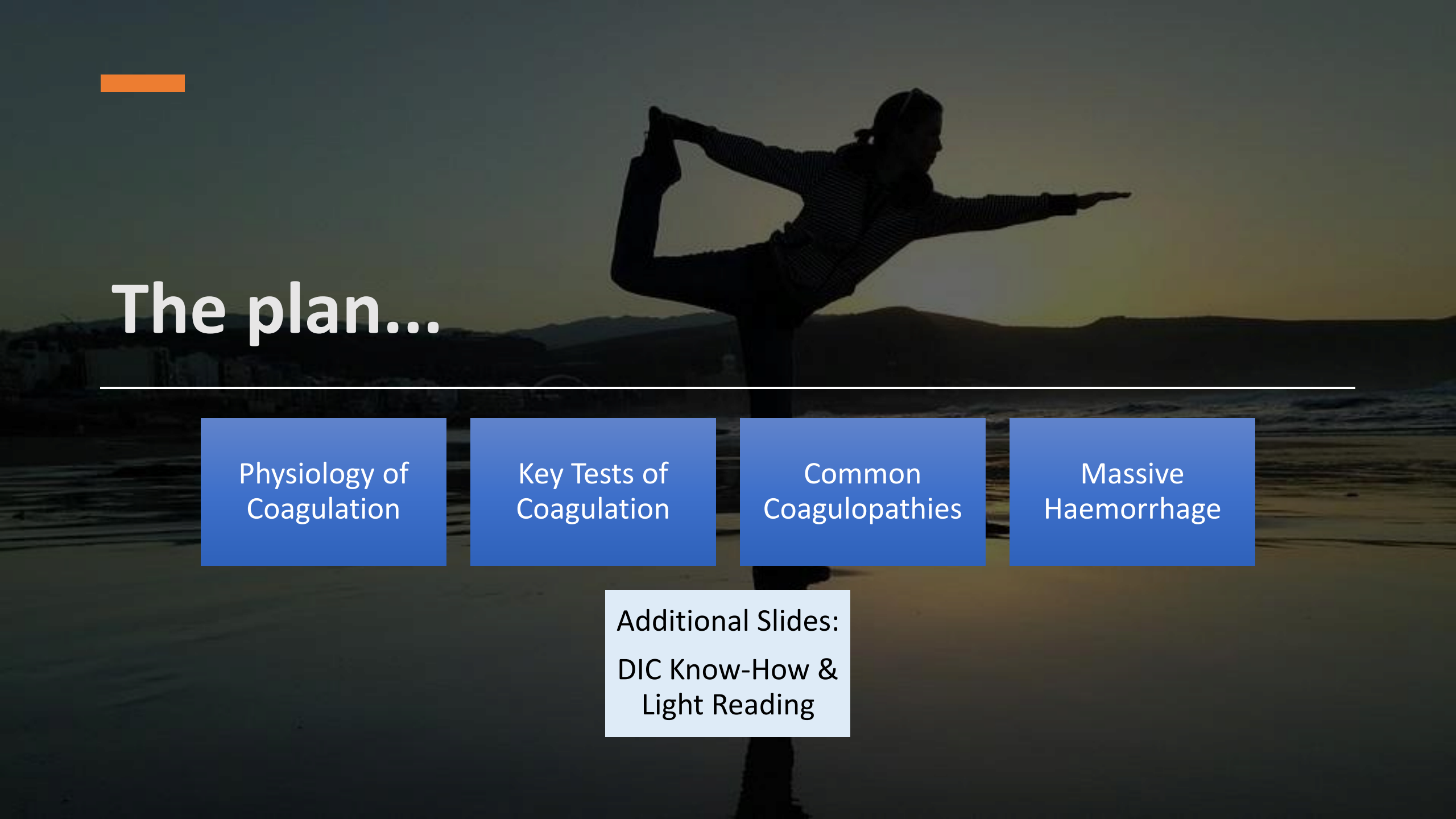
Acknowledgements:
Mobile Health Group team
Alicia Osland, NZCCN


Sphere of Influence
How do we apply & translate what we know

Balancing Act

Team-Work

Context



The plan...

- Physiology of Coagulation
- Key Tests of Coagulation
- Common Coagulopathies
- Massive Haemorrhage

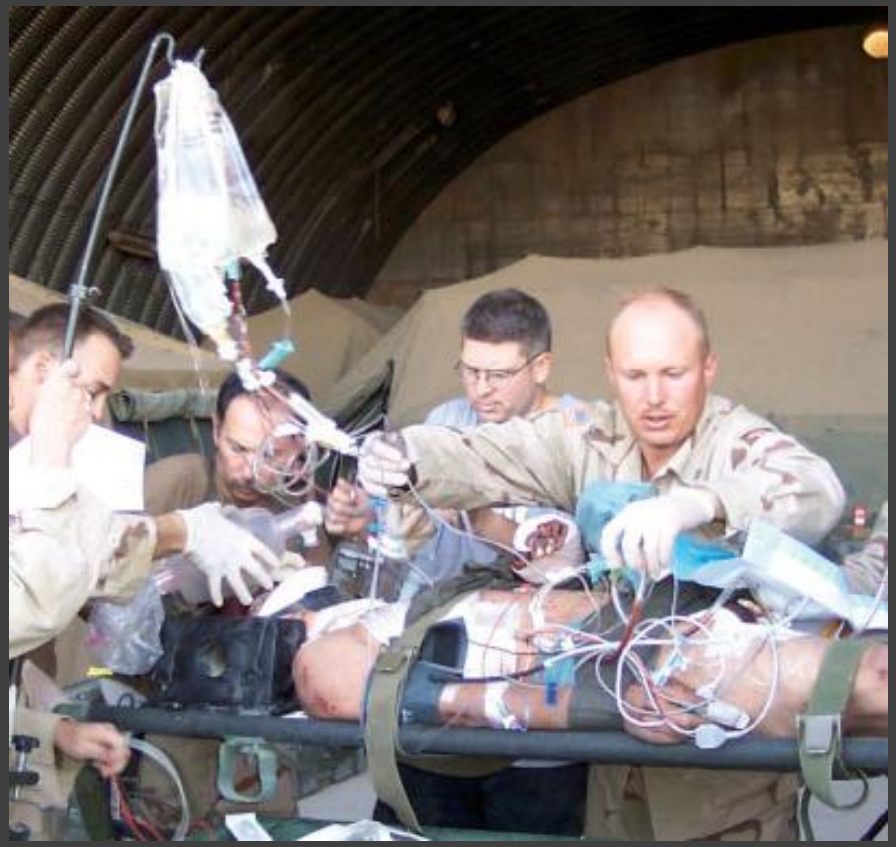
Additional Slides:

DIC Know-How & Light Reading

Definitions

Haemostasis

Critical & normal sequential process to stop bleeding and prevent blood loss following vascular injury



Coagulopathy

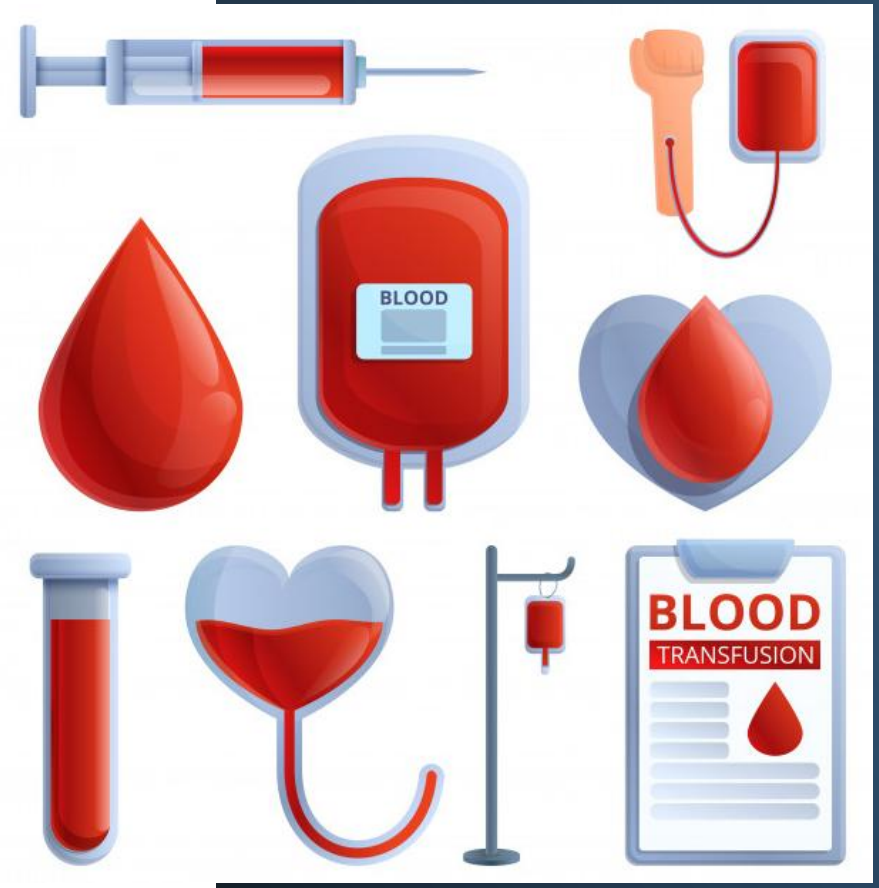
A pathologic condition that affects the ability of the blood to coagulate | clot

Mosby's Medical Dictionary, 8th edition. © 2009, Elsevier.

Blood Transfusion

Process of transferring blood into another's circulation,

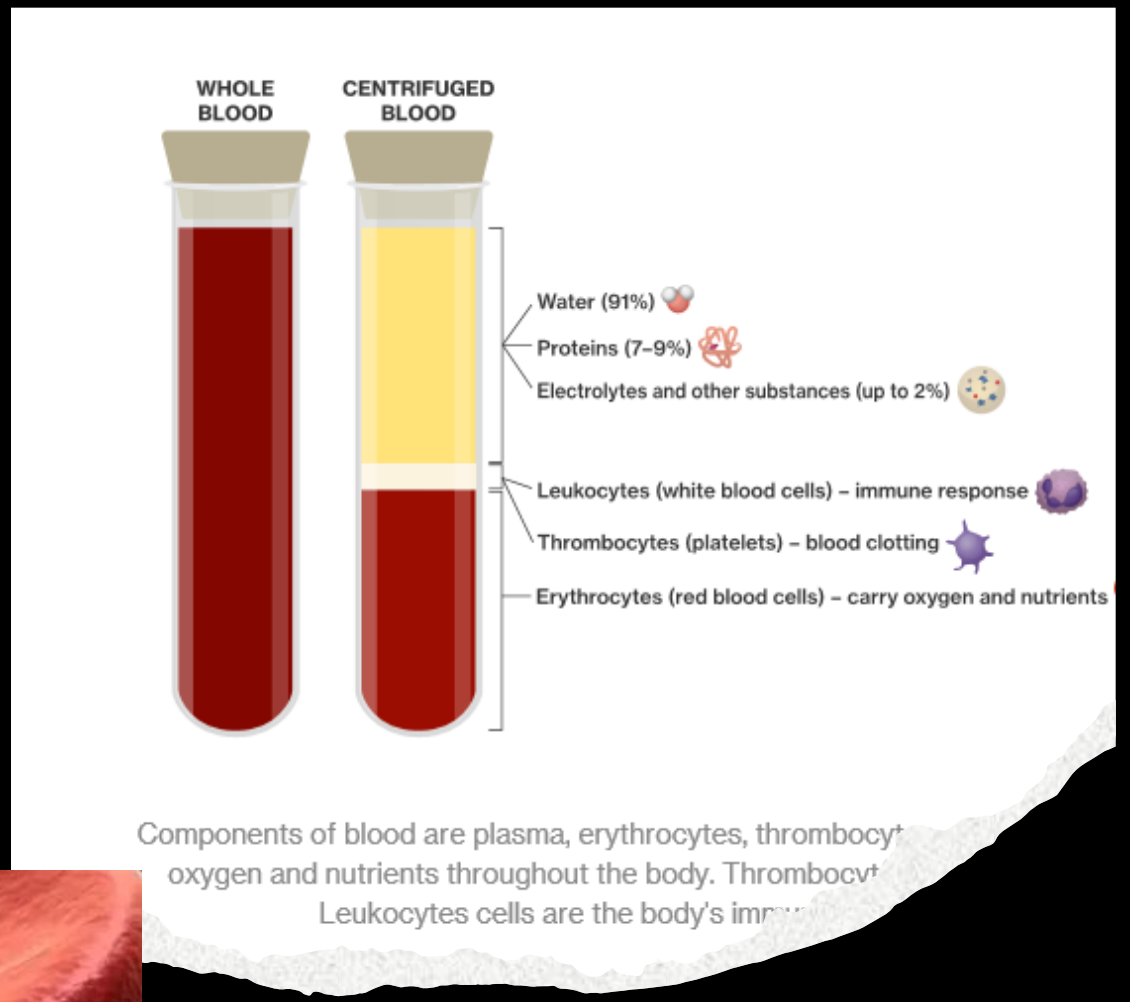
to replace and resupply the properties and functions lost



Modern Transfusion

Utilises and maximizes the various properties of whole blood sustainably & effectively

It is targeted and personalized medicine



https://www.coursehero.com/sg/anatomy-and-physiology/blood-composition/

Transfusion is underpinned by

Patient Safety

Te haumaru o te tūroro

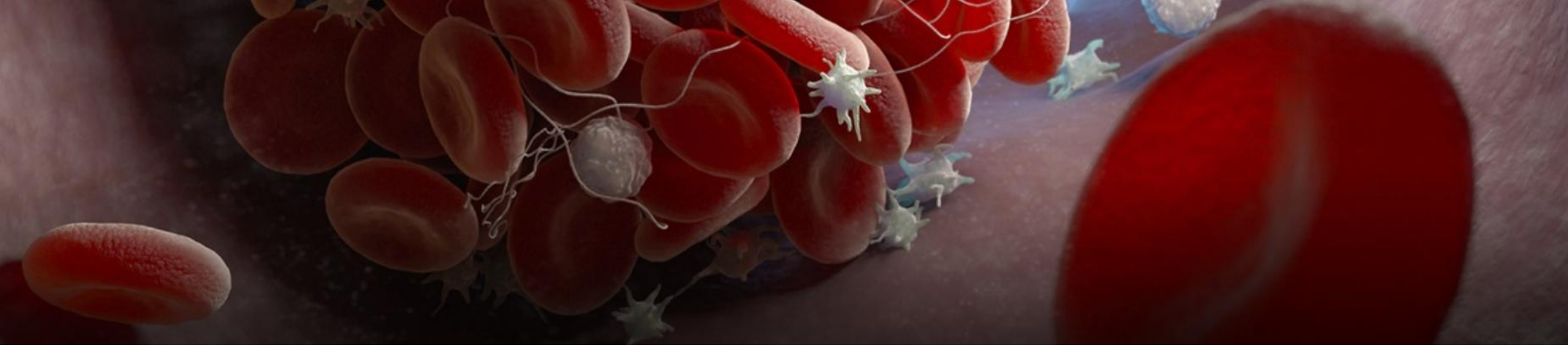
Traceability

Te whai i te whai

Custodianship

Kaitiakitanga

Fundamentals of Haemostasis



Haemostasis- the major forces

Tissue Injury exposes collagen

1. Vasoconstriction

2. Platelet Activation

3. Coagulation Cascade Activation

4. Fibrinolysis

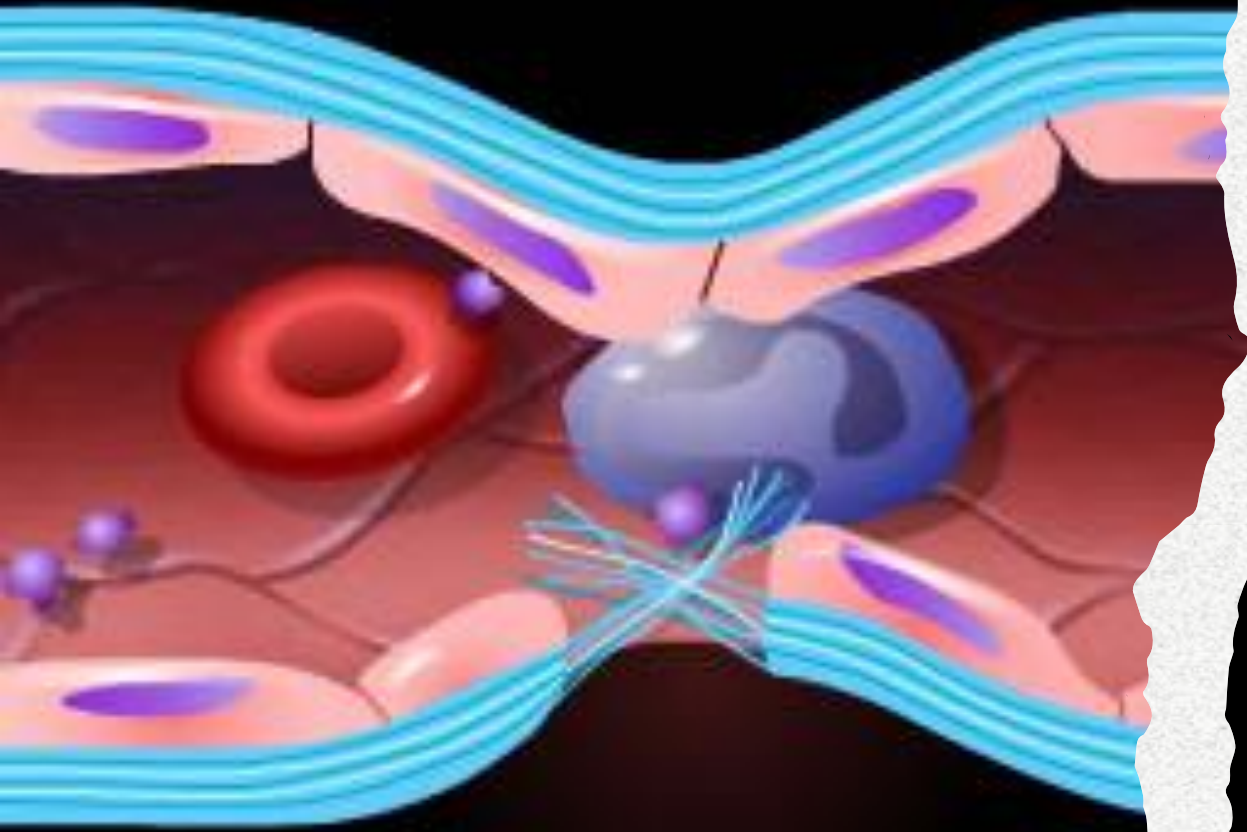
Primary Haemostasis

Secondary Haemostasis

Image. Johns Hopkins Medicine. 2024.

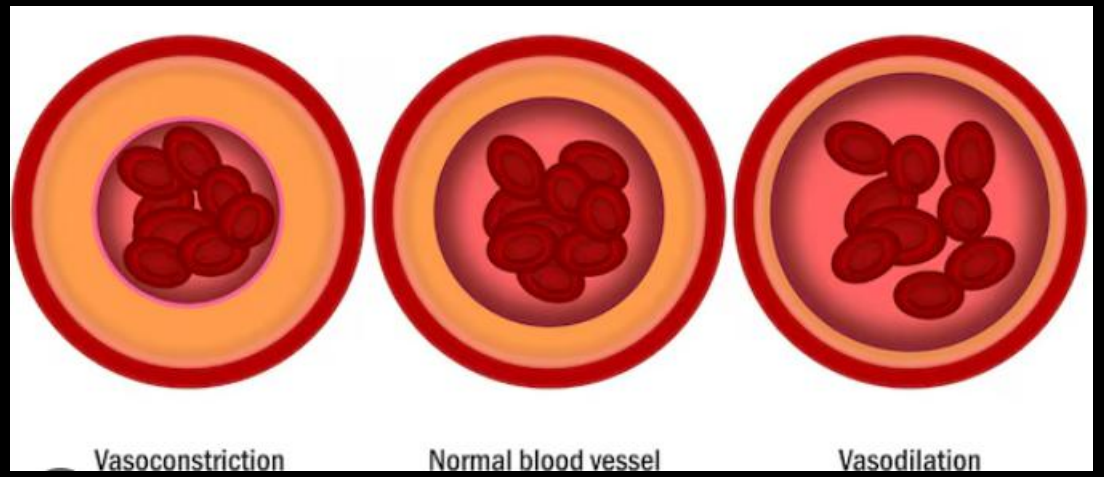
Trigger → tissue injury → exposes collagen

1. Vasoconstriction = Primary Haemostasis



Tissue damage causes local vasoconstriction to decrease blood flow to the area

Neural response



1; slows the bleed, temporary measure

2. Platelet Activation = Primary Haemostasis

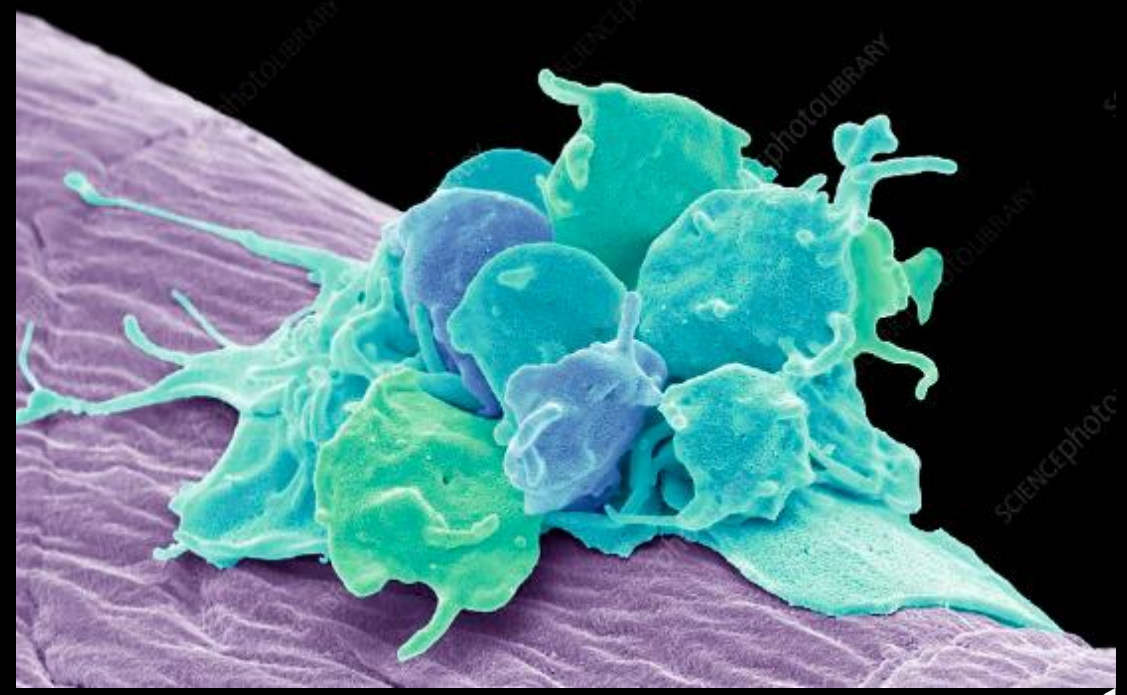
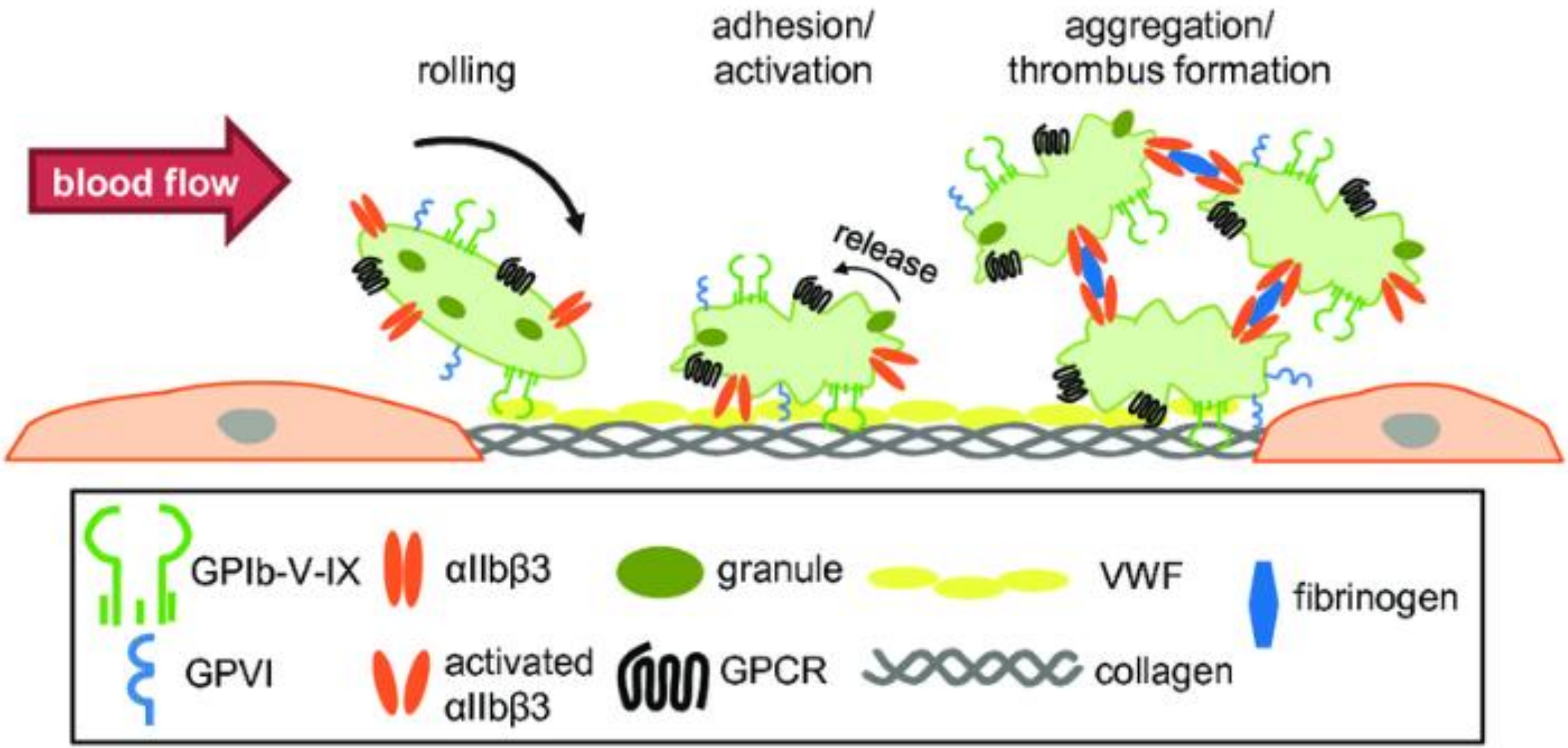


Image Credit (magnification x 7000 @ 10cm wide)
STEVE GSCHMEISSNER / SCIENCE PHOTO LIBRARY

Damage to the vessel wall exposes the **sub-endothelium collagen** which **stimulates platelet activation**

- platelets adhere to von Willebrand factor (vWF)
- platelets activate & recruit more help (release reaction)
- platelets aggregate to form a soft, friable platelet plug



Mechanism of platelet adhesion and thrombus formation at sites of... | Download Scientific Diagram (researchgate.net)

Exposure of sub-endothelial collagen + Platelet Rolling

↓

Platelet Adhesion via vWF

↓

Platelet Release Reaction: Activation

Thromboxane A^2 ↙ | ↓ ADP, Thromboxane A^2 | ↘ Platelet phospholipid

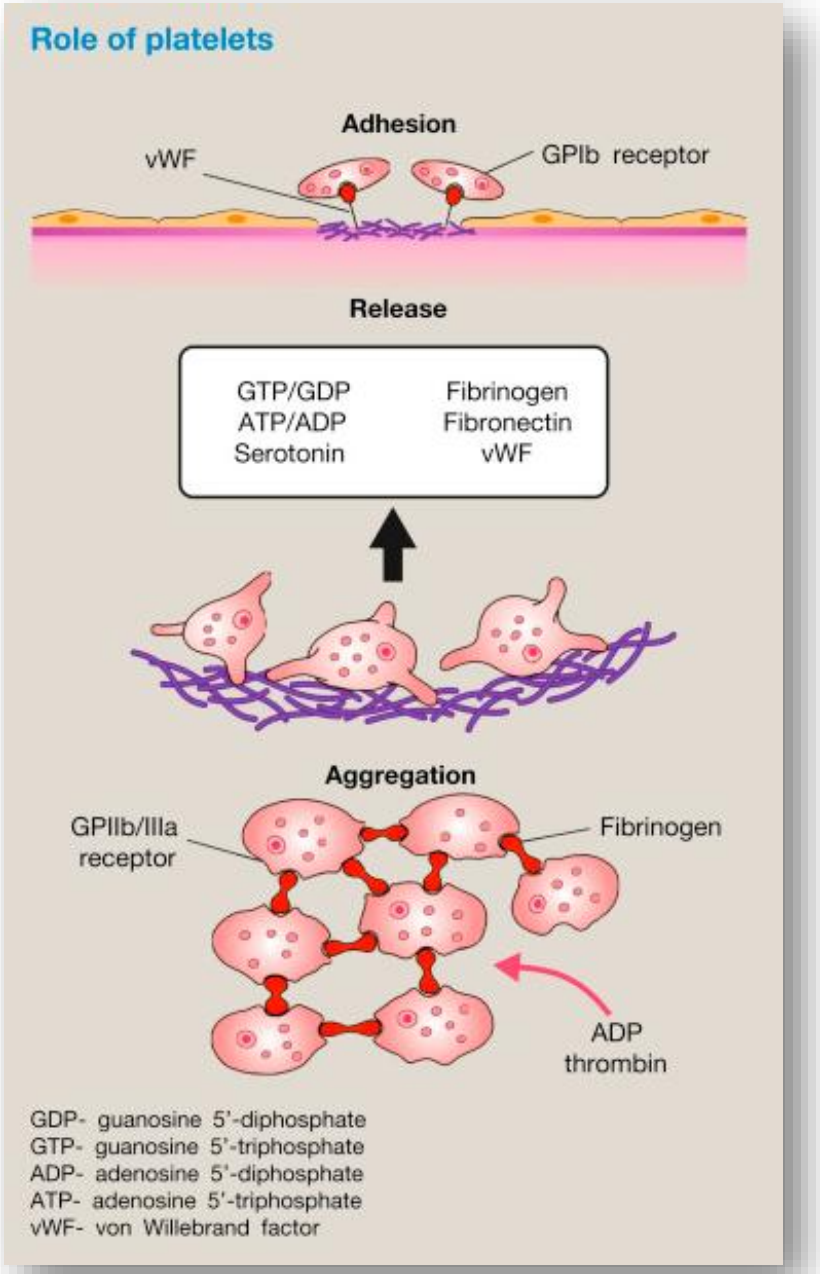
Increased Vasoconstriction

Further clumping/aggregation

Clotting cascade enhanced

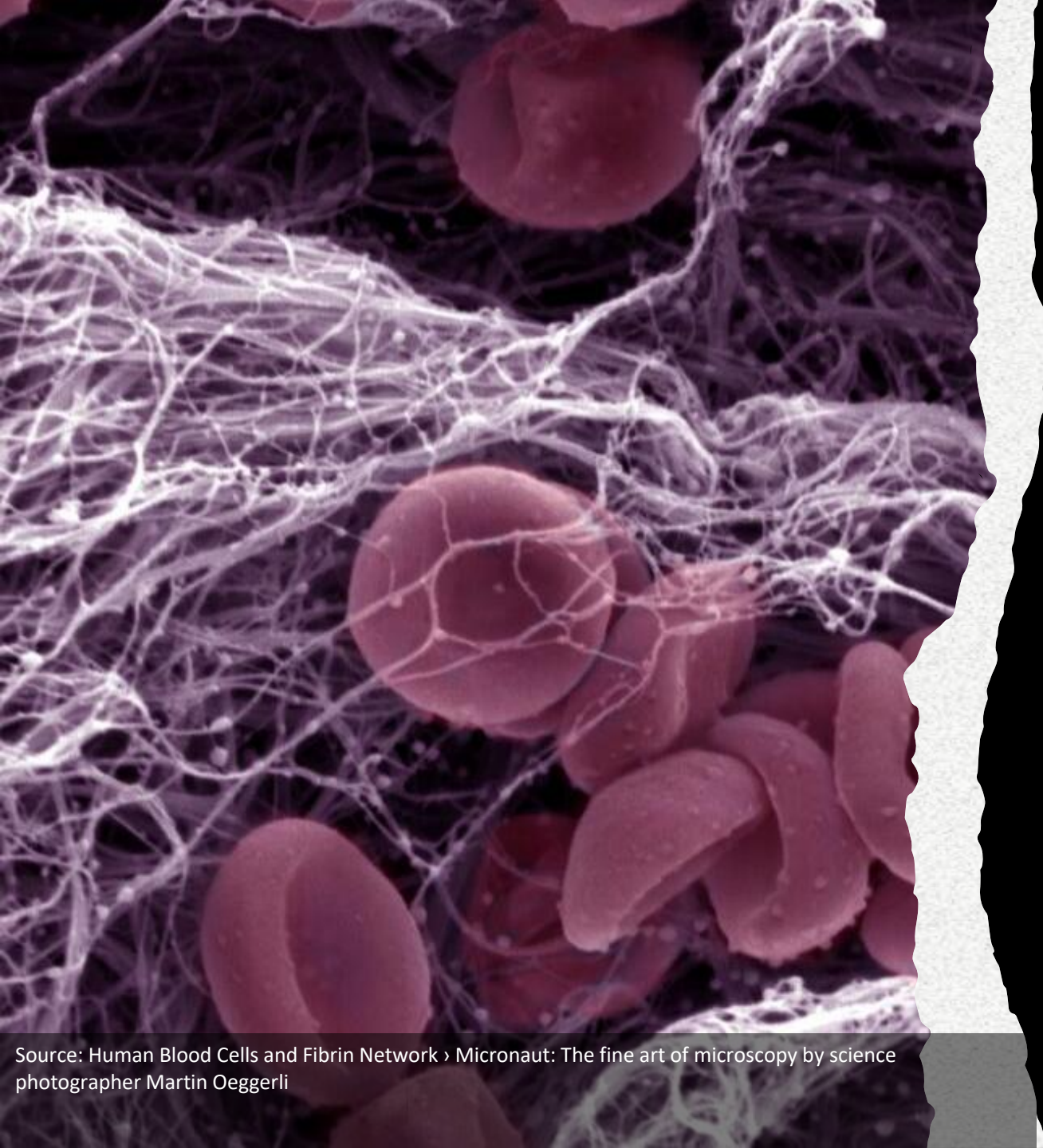
↓

Clot stabilized



https://www.sciencedirect.com/science/article/abs/pii/S1472029915002702

2. Forms the plug, not the final action



Source: Human Blood Cells and Fibrin Network › Micronaut: The fine art of microscopy by science photographer Martin Oeggerli

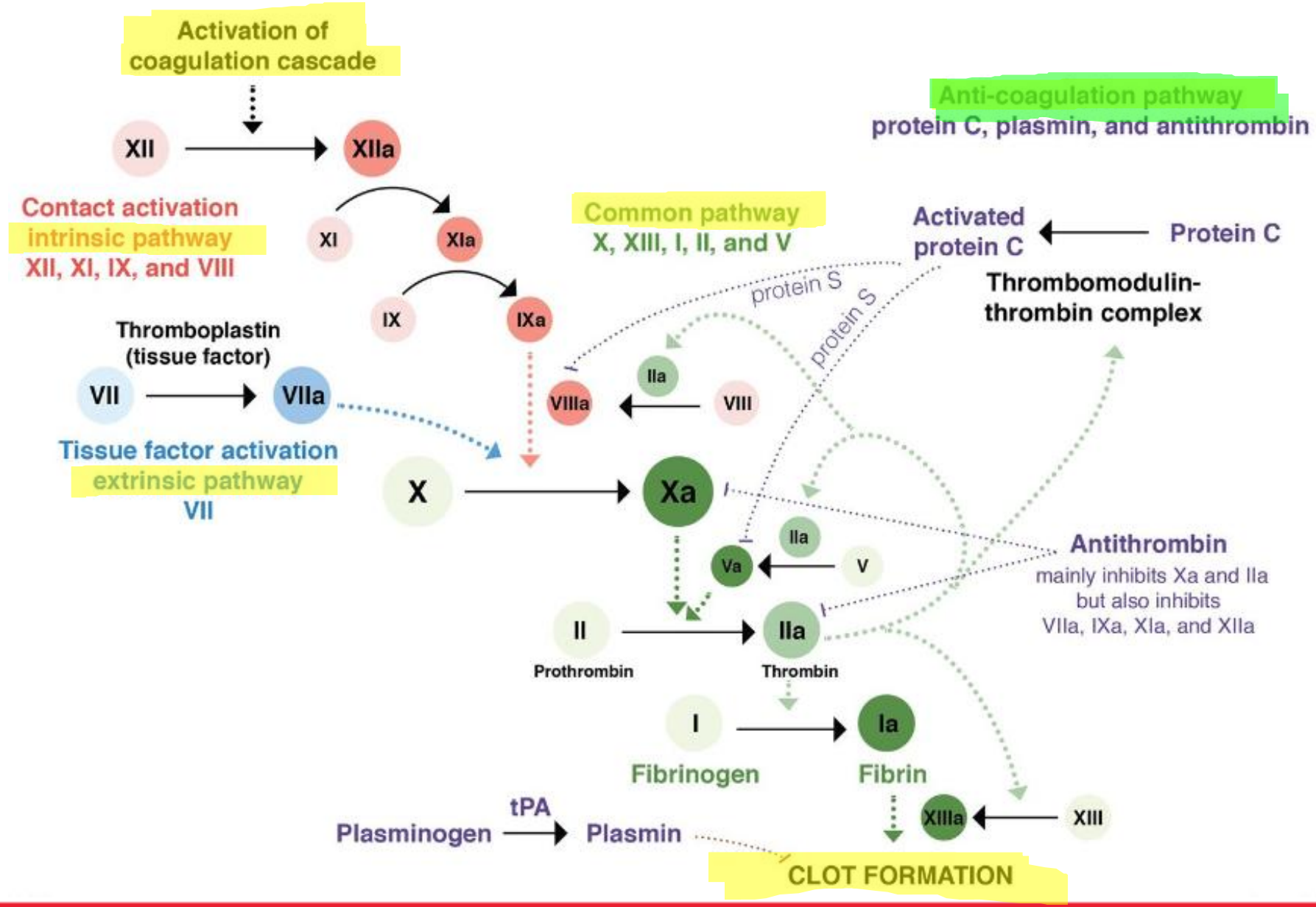
3.Coagulation Cascade Activation = Secondary Haemostasis

Tissue factor released by injured cells triggers the coagulation pathway

Complex cascade of sequential activation of circulating coagulation factors to form fibrin net

Coagulation Cascade

Accelerators
(pro-coagulants)
&
Brakes
(anti-coagulants)



FACTOR	SYNONYM
I	Fibrinogen
II	Prothrombin
III	Tissue factor, thromboplastin
IV	Calcium
V	Proaccelerin, labile factor
VI	—
VII	Proconvertin, stable factor
VIII	Antihemophilic factor
IX	Christmas factor
X	Stuart-Prower factor
XI	Plasma thromboplastin antecedent
XII	Hageman factor
XIII	Fibrin-stabilizing factor, transglutaminase

12 factors, whom identify as Roman

Calcium (FIV | factor 4) required to activate factors → II, VII, IX, X*
a lack of calcium directly impacts the cascade, (consider MTP/MHPs)

*Factors 2, 7, 9, 10 are the **vitamin K dependent factors**,
aka, the **prothrombin complex factors**

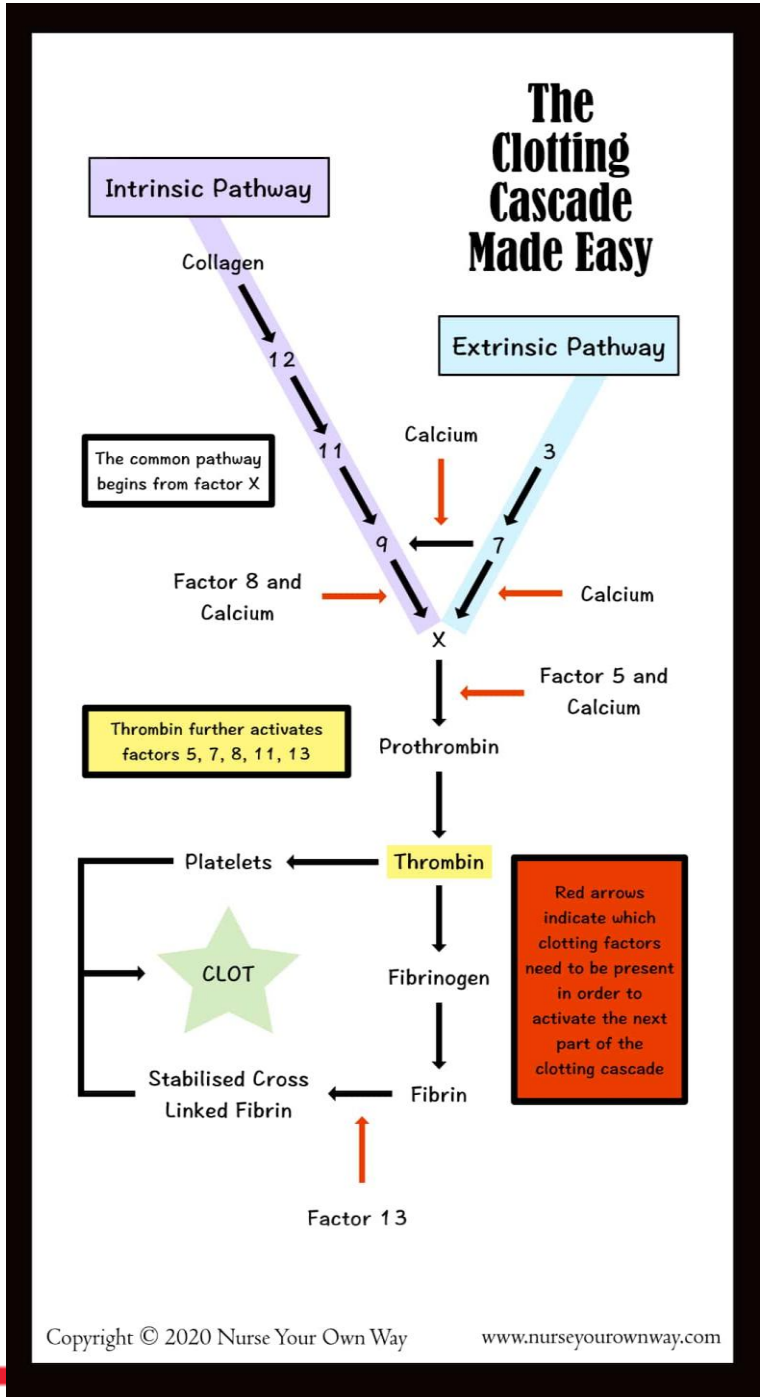
Factor X (10) is the crossroads of the two paths

Prothrombin (FII or 2) activates **Thrombin (FIIa or 2a)**

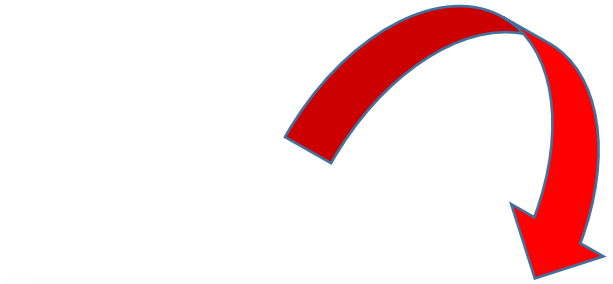
Thrombin (FIIa) further activates factors:

- V (5), VII (7), VIII (8), XI (11), XIII (13)

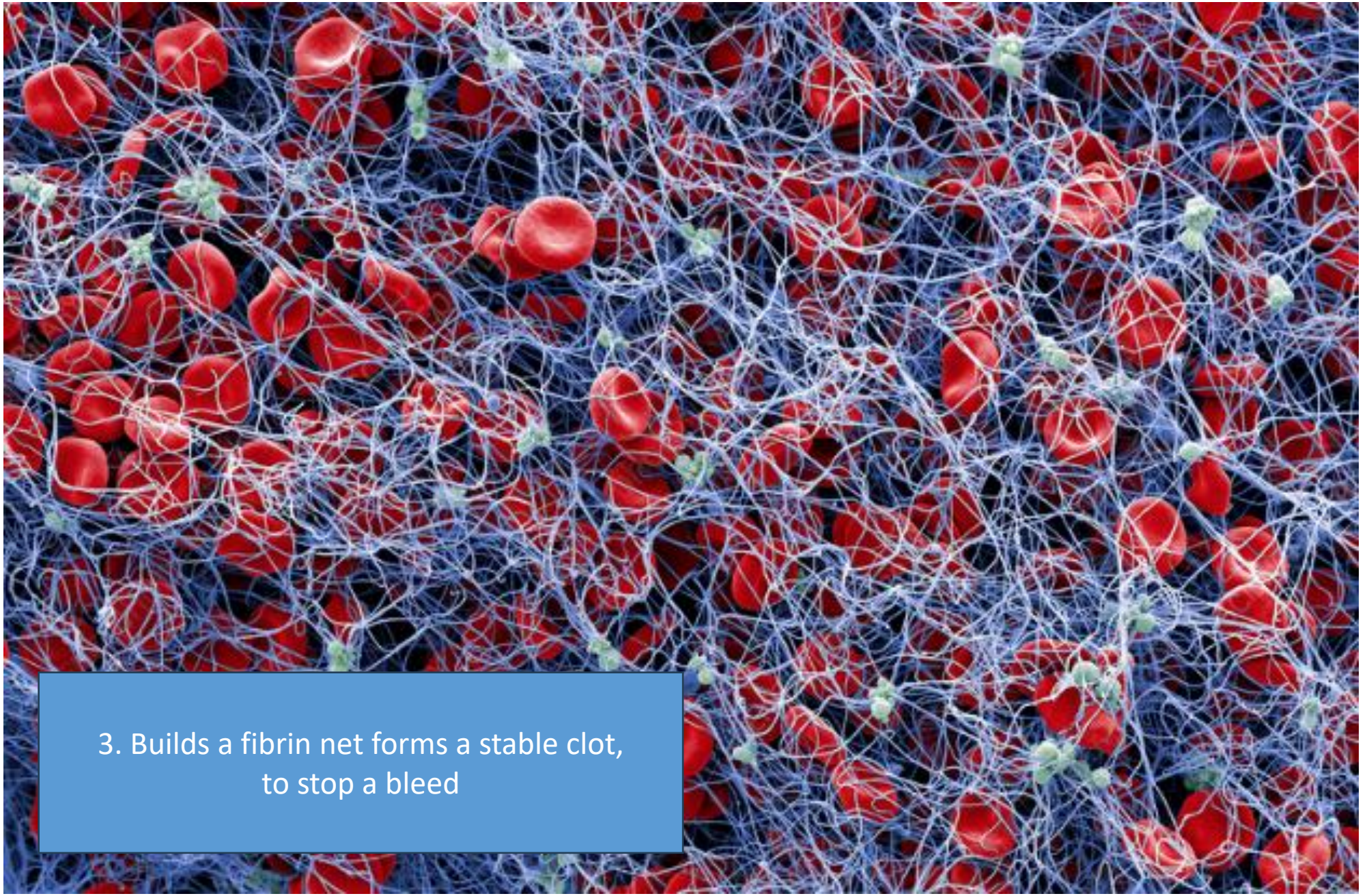
Fibrinogen (FI or 1) activates **Fibrin (FIa), crosslinks form, form a net**



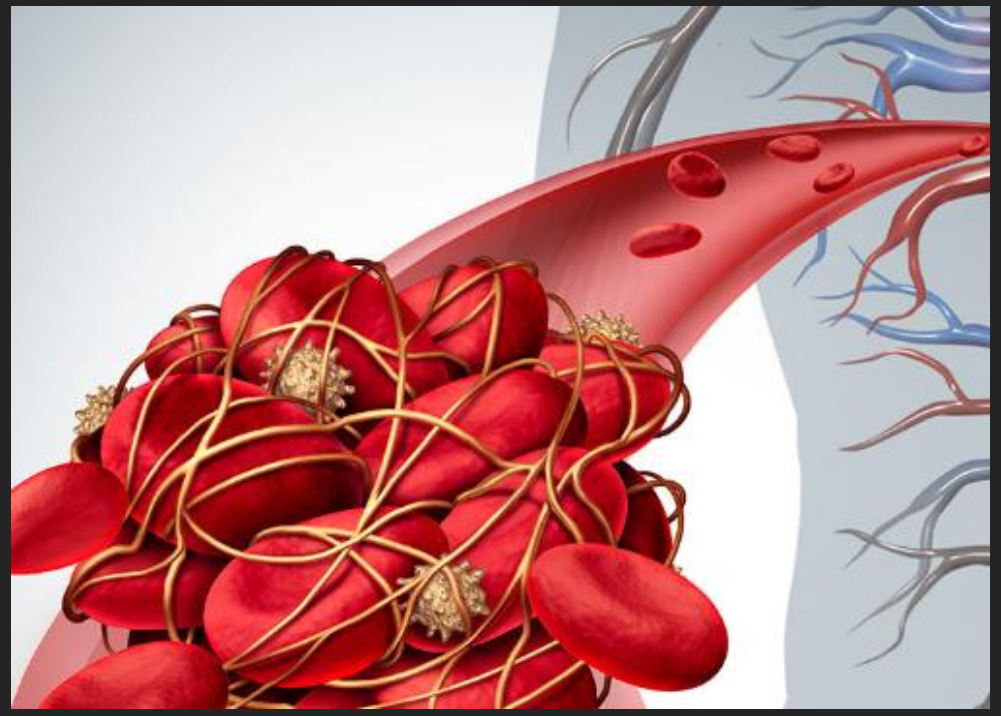
Coagulation factors activated to produce pieces of **fibrin**



Strands of **fibrin** form a **fibrin net** to consolidate the **primary platelet plug**

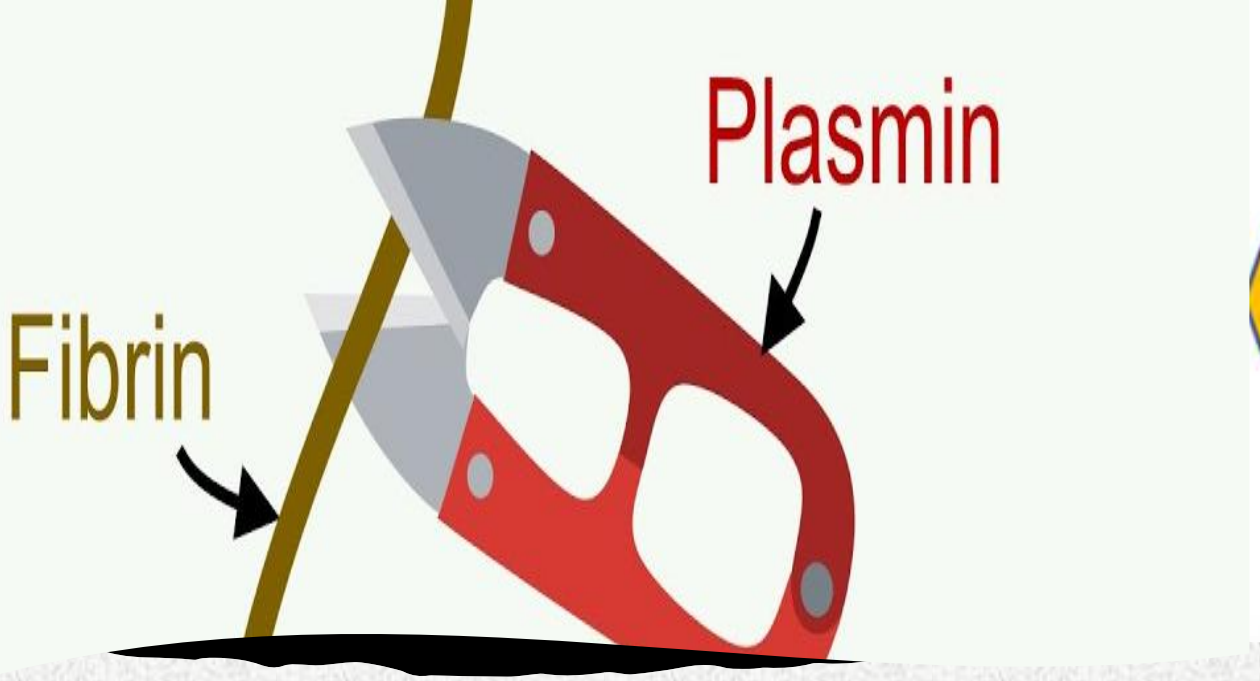


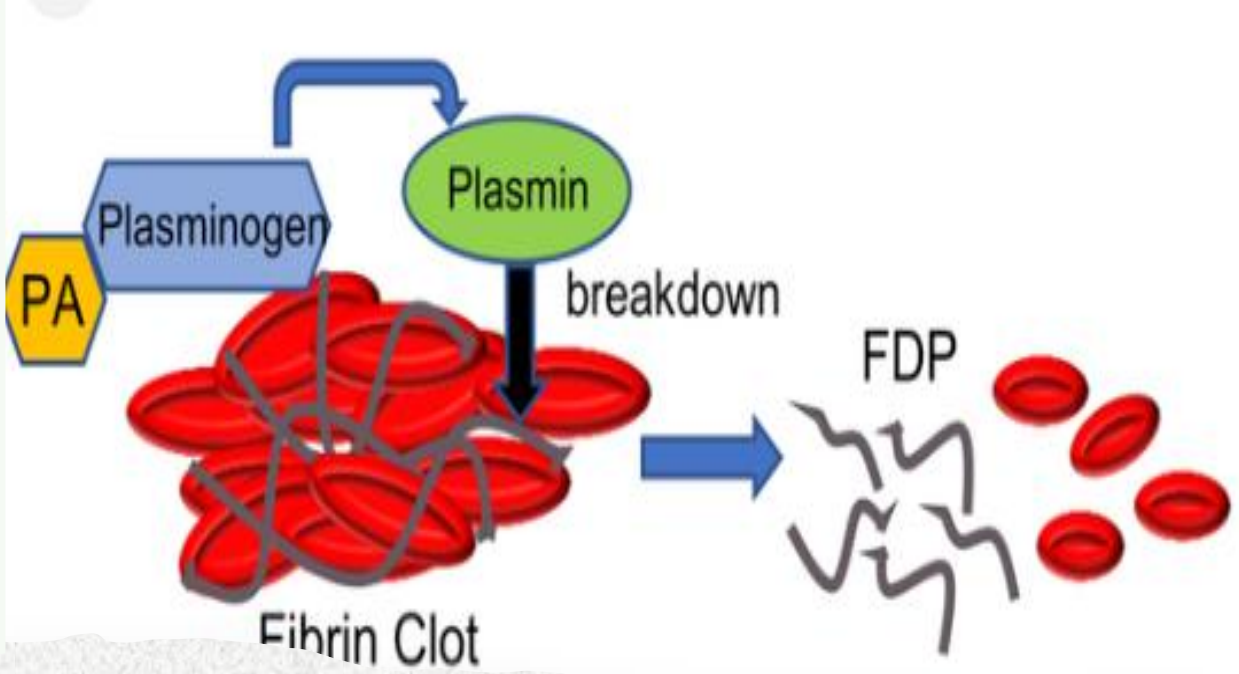
Activated Platelets and RBC in a Fibrin Mesh, [coloured scanning electron micrograph (SEM)]



Balancing act,

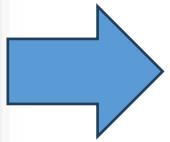
halt clot to emboli





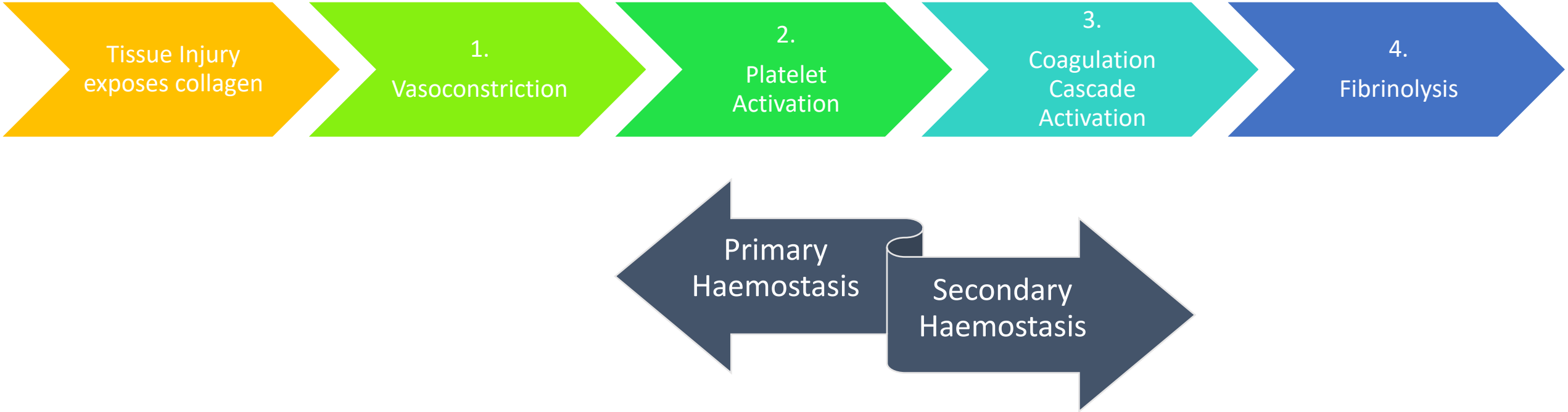
4. Fibrinolysis

- **t-PA** is secreted by wound endothelium
- **t-PA converts plasminogen** (bound in the fibrin net) to **plasmin**
- **Plasmin** cuts the cross-links in the **fibrin-net**
- D-Dimers & fibrin(ogen) degradation products are released into the circulation
- Phagocytes clear the remainder of the clot



4, cleans up the clot

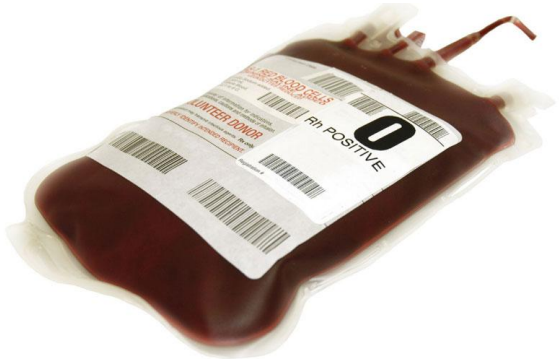
Haemostasis- the major forces



In clinical practice

Give 'GOLD'
for haemostasis

Give 'RED'
for tissue oxygenation



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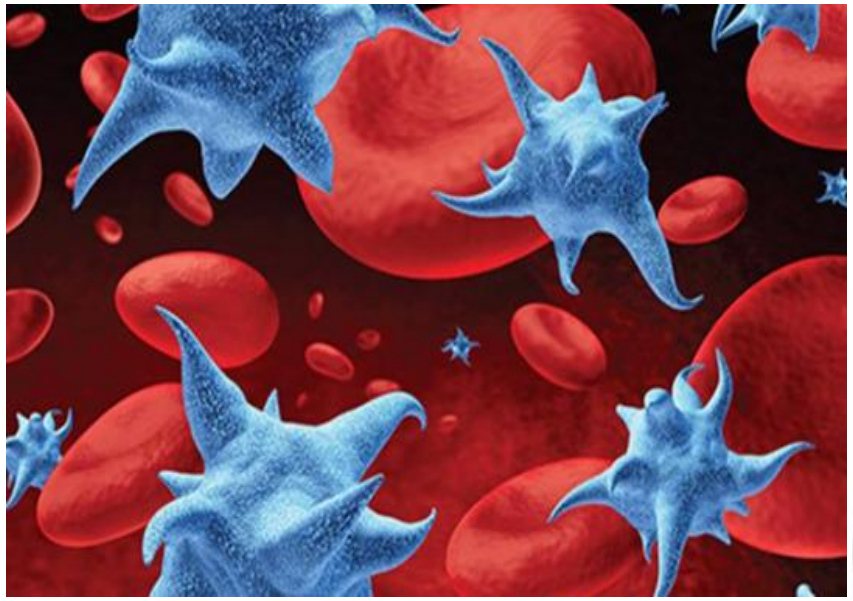
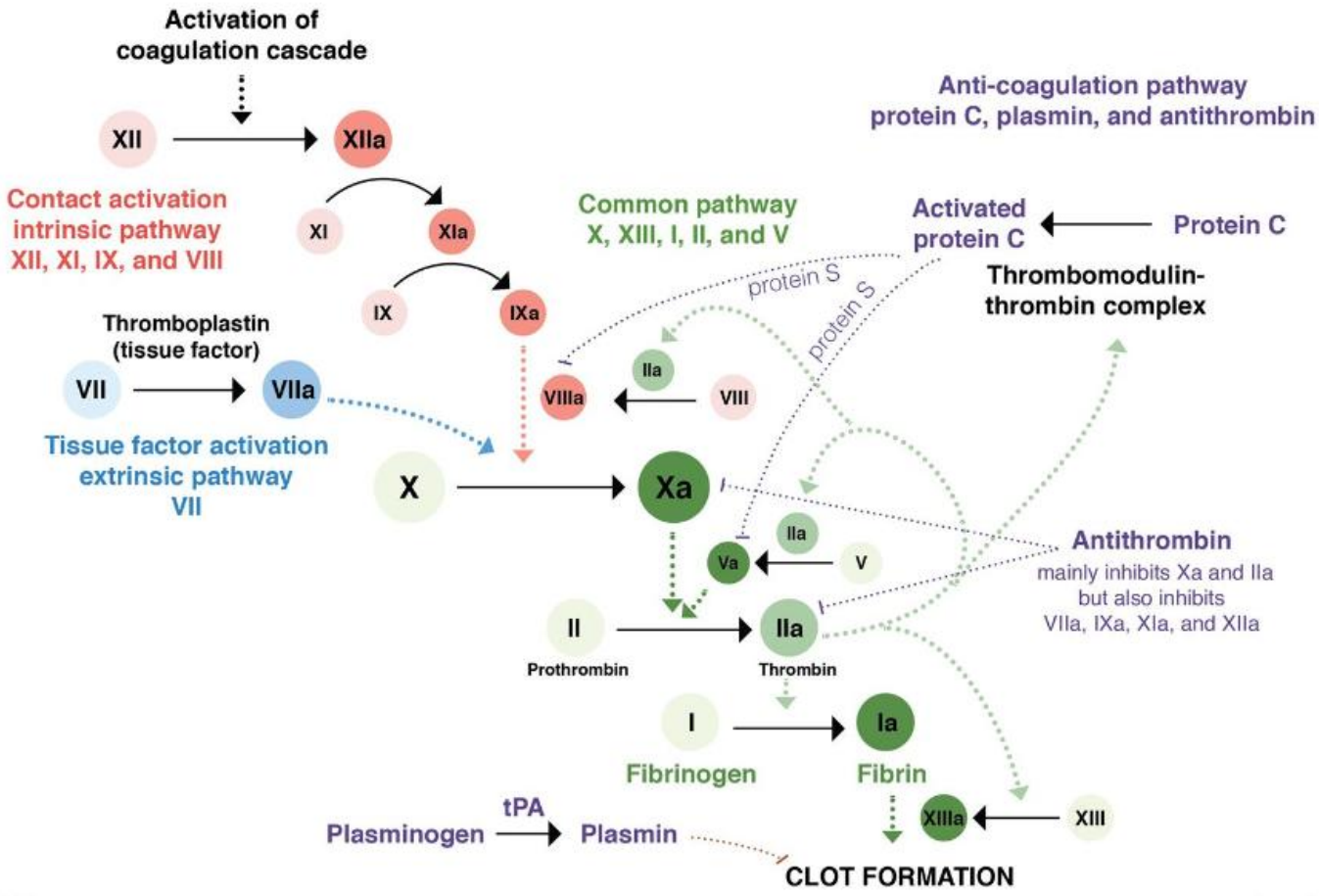
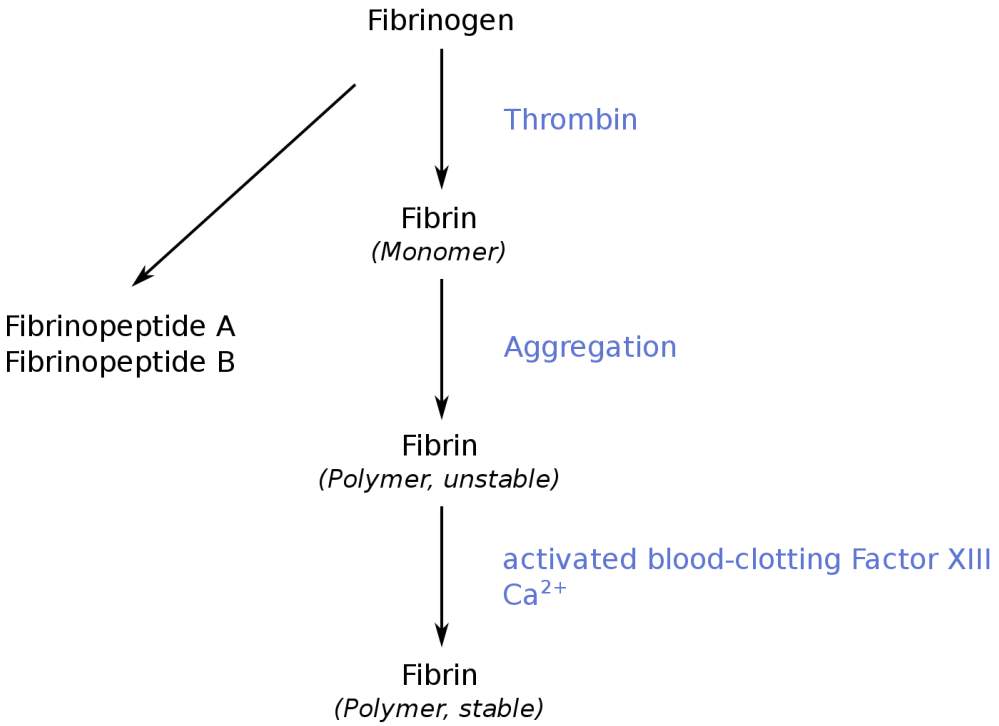


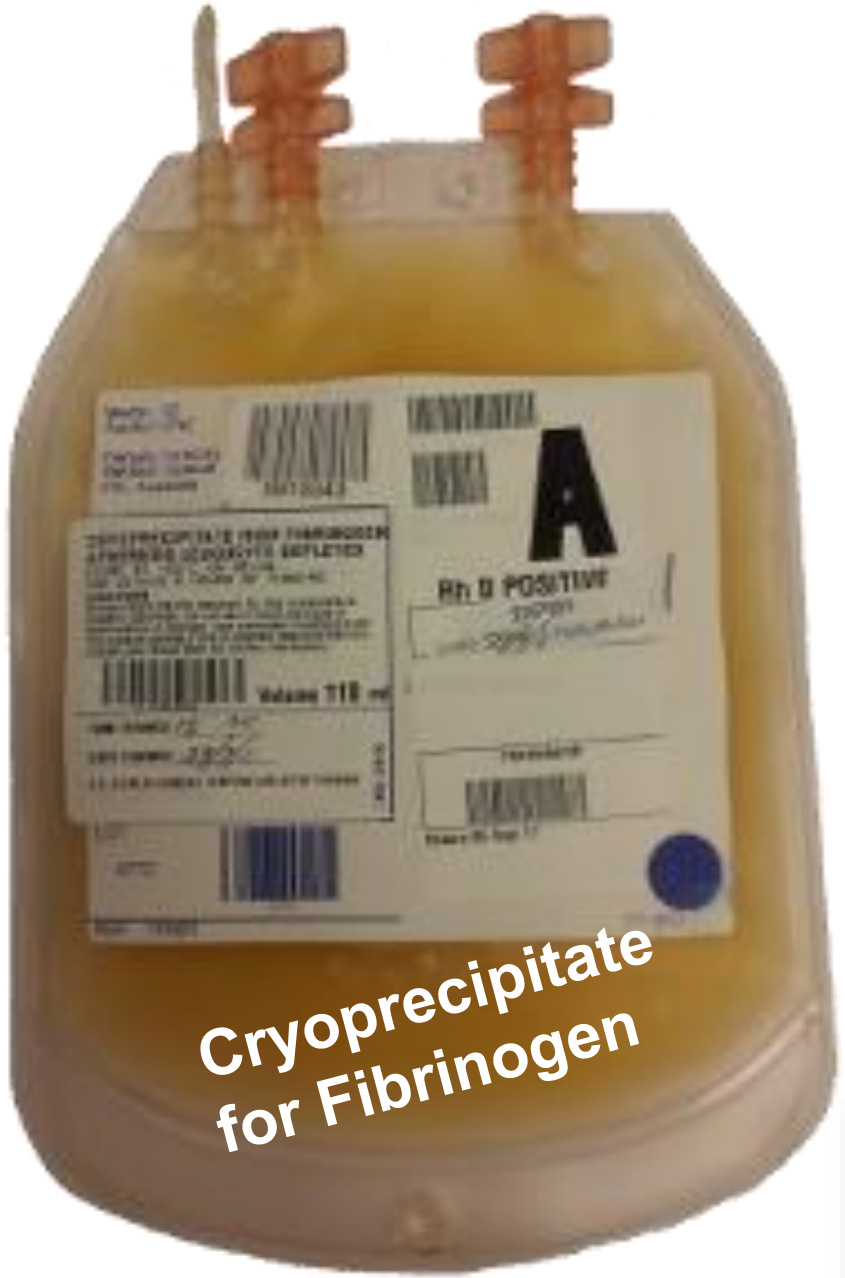
Thrombocytopenia

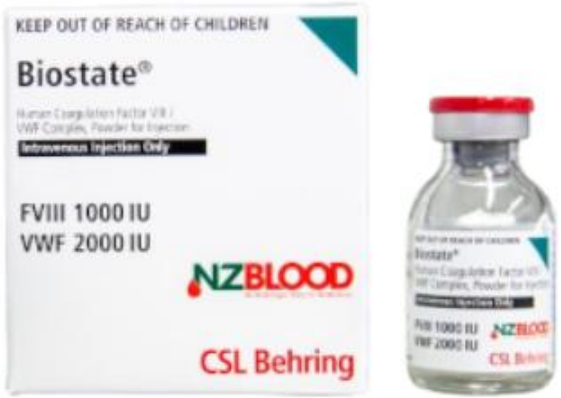
Platelet dysfunction

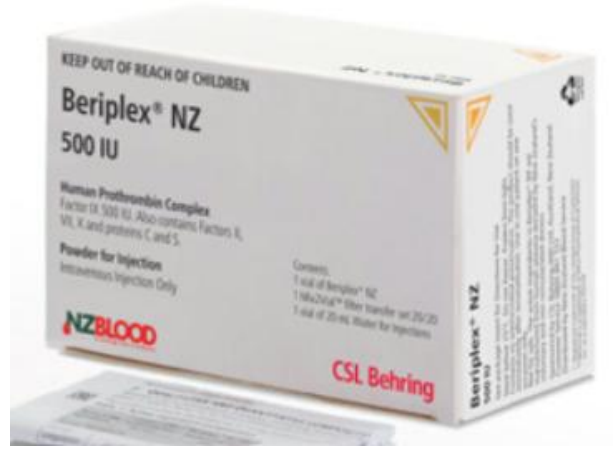
Coagulation Cascade




Fibrinogen
Thrombin
Fibrin
(Monomer)
Fibrinopeptide A
Fibrinopeptide B
Aggregation
Fibrin
(Polymer, unstable)
activated blood-clotting Factor XIII
Ca2+
Fibrin
(Polymer, stable)


A
Rh D POSITIVE
Cryoprecipitate
for Fibrinogen





Prepare at room temp
Swirl don't shake
Don't rely on muscle memory, follow the steps
Administered as IV injection – no mixers!
Blood traceability is needed

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Testing Coagulation

Haemostasis Assessment

Context!

Coagulation screen

provides insight into haemostatic function

Consider in context with full blood count and blood film (platelet number and appearance)

Tested by activated partial thromboplastin time

Tested by prothrombin time

Intrinsic pathway

Surface contact

XII → XIIa

XI → XIa

IX → IXa

VIIIa
Ca^{2+}
PF3

Extrinsic pathway

TF

VIIa ← VII

TF

X → Xa ← X

Activated protein C (with protein S) deactivates Va and VIIIa

Va
Ca^{2+}
PF3

Antithrombin III deactivates Xa and thrombin

Thrombomodulin–thrombin complex activates protein C

Prothrombin (II) → Thrombin (IIa)

Thrombomodulin binds and inactivates thrombin

Fibrinogen (I) → Fibrinogen (Ia)

XIIIa

Cross-linked fibrin clot

Tested by the thrombin time

Final common pathway

Prothrombin Time (PT) & INR

Essentially a test of the **efficiency** of the extrinsic path in addition to factors V, X, prothrombin and fibrinogen

- Blue top (citrate)
- Reference range **7.6 – 10 seconds**

Hoffbrand, et al, 2019

= the PT value varies across labs, dependent on the reagents / test kits they use

To give **consistent PT** from laboratory to laboratory the WHO instituted the **International normalized ratio** (INR)

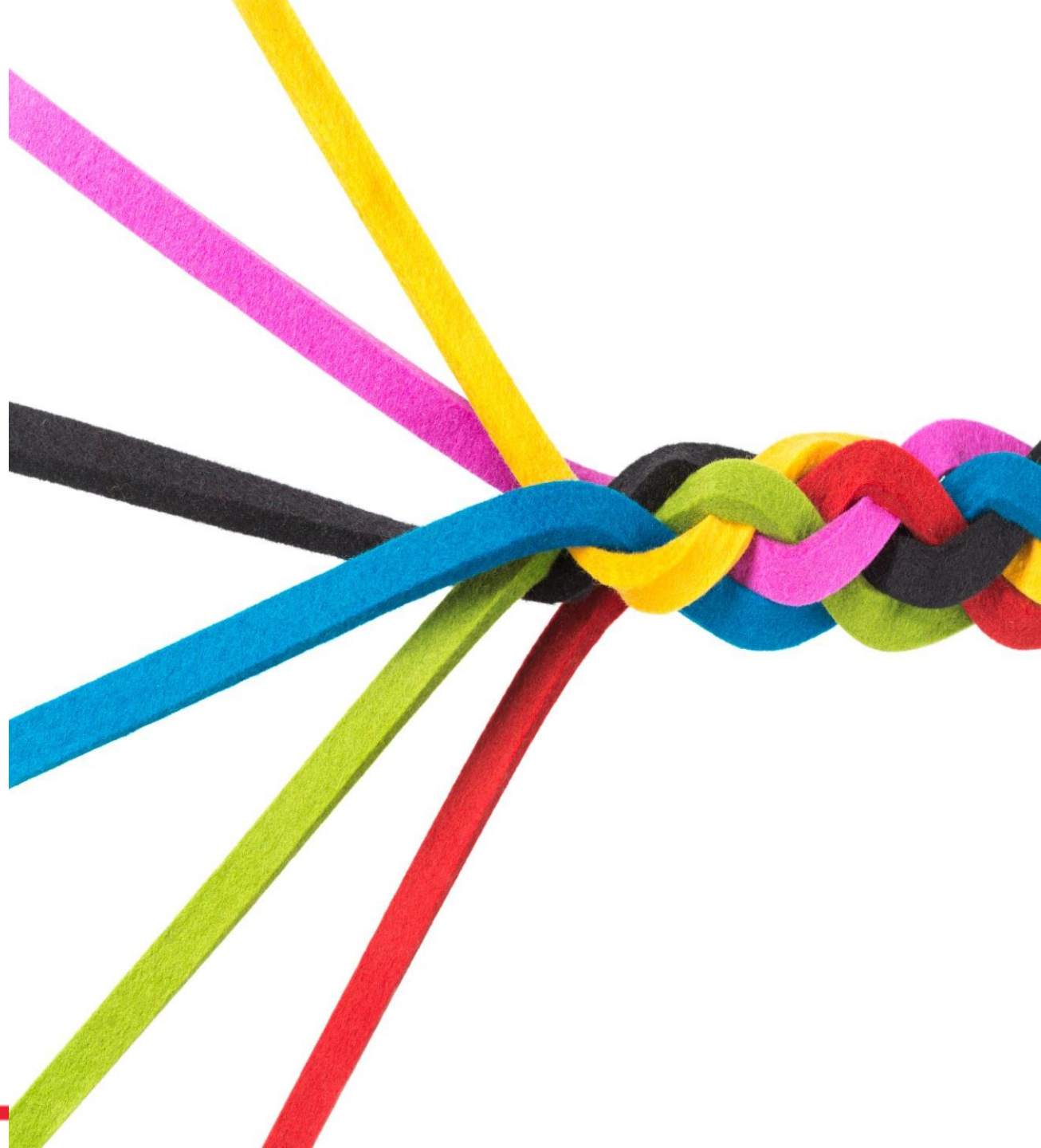
- **Normal INR** = 1

PR= prothrombin ratio

Some labs use the PR, not measured in time but is a ratio of the persons PT against the labs normal value for a PT, this varies by lab

Where do you see it?

In the national MHP as a value



APTT (Activated Partial Thromboplastin Time)

Measures overall efficiency of the intrinsic path (factors VIII, IX, XI, XII) & factors X, V, prothrombin, & fibrinogen

- Blue top (citrate)
- Reference range 29.0 – 41 seconds

Hoffbrand, et al, 2019

TCT or TT (Thrombin Clotting Time)

Measures rate of converting fibrinogen to fibrin

- Blue top (citrate)
- Reference range 11 – 18 seconds

Hoffbrand, et al, 2019

Fibrinogen

Measures the **amount of fibrinogen** (Factor I) **circulating** in the blood

- Normal plasma **fibrinogen level range** of **2.0–4.5 g/L**
- The **critical** plasma **fibrinogen level** in haemorrhage is approximately 1 g/L
- 🚩 obstetric haemorrhage, alarm at 2 g/L

D-Dimers
FDPs

Measures **fibrinolysis**

- **D-Dimers** are released into the circulation when **cross-linked fibrin** is broken down by plasmin

- **FDPs** [fibrin(ogen) degradation products] are parts of the **non-cross-linked fibrin** or **fibrinogen** molecules that have been broken down

Normal coag. function

Slowed coag. function

Common causes of abnormal coagulation tests

INR | PT | PR

- Warfarin, liver disease, vit k deficiency, DIC

APTT

- Heparin, haemophilia, vWD, DIC, liver disease

TT

- DIC, liver disease, heparin

The APTT is used commonly for routine monitoring of heparin

But, the ACT (activated clotting time) is a rapid point of care test in specialized scenarios requiring large heparin doses, to determine how well heparinised a patient may be

Clinical setting: cardiac bypass surgery, angioplasty, dialysis.

Measured in seconds, noting the longer the time the higher the degree of anticoagulation

Reference range

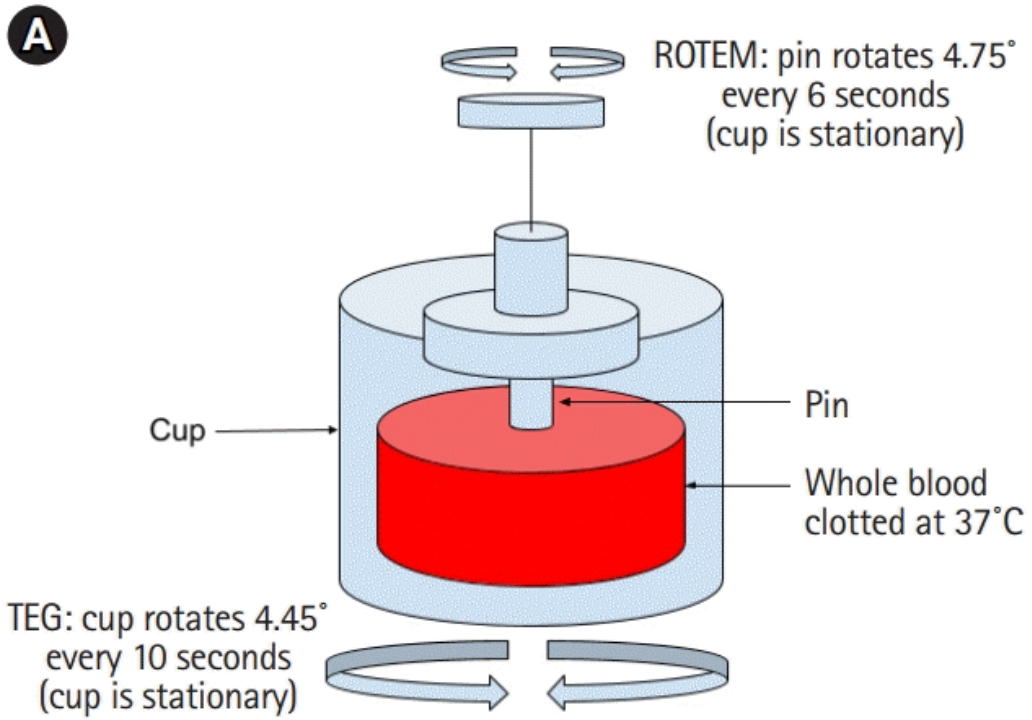
70- 120 seconds (normal, no heparin)

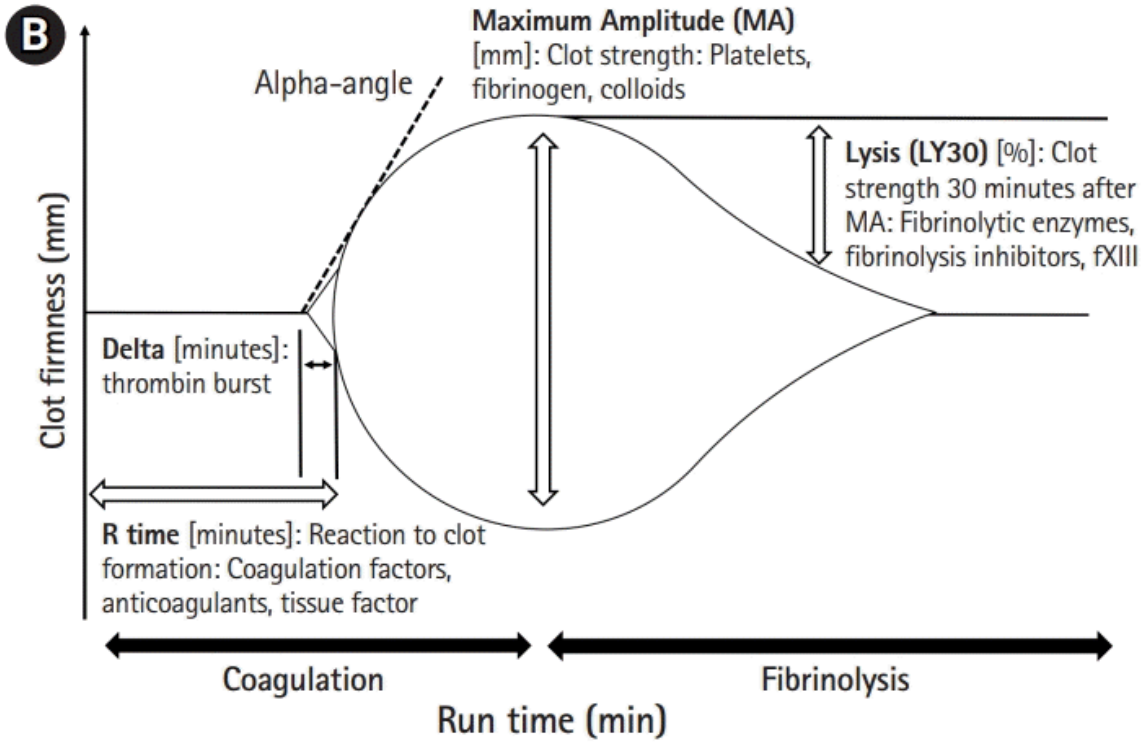
180-240 seconds (time to clot with heparin / therapeutic)

Uni Rochester, 2023

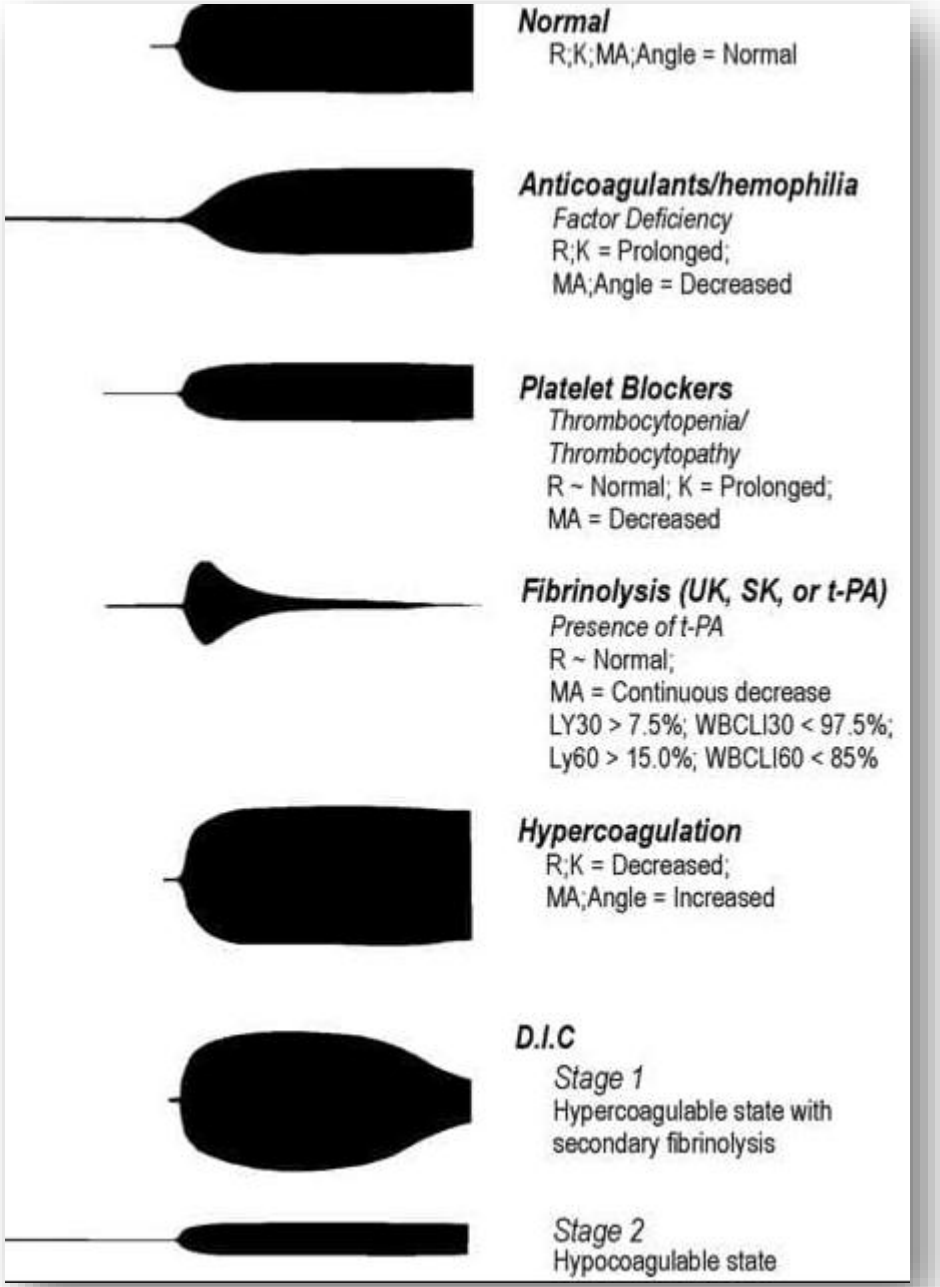
Other Tests?

- Bleeding time: test for abnormal platelet function
  - Controlled incision and observed time to stop bleeding
- Tests of platelet function
  - special requests (blue top)
- Specialized clotting factor assay
  - special requests, lab specific
- TEG (thrombo-elastography) or ROTEM
  - hospital specific to access, require know-how to interpret





https://www.e-jnc.org/upload//thumbnails/jnc-190086f1.gif



Normal
R;K;MA;Angle = Normal

Anticoagulants/hemophilia
Factor Deficiency
R;K = Prolonged;
MA;Angle = Decreased

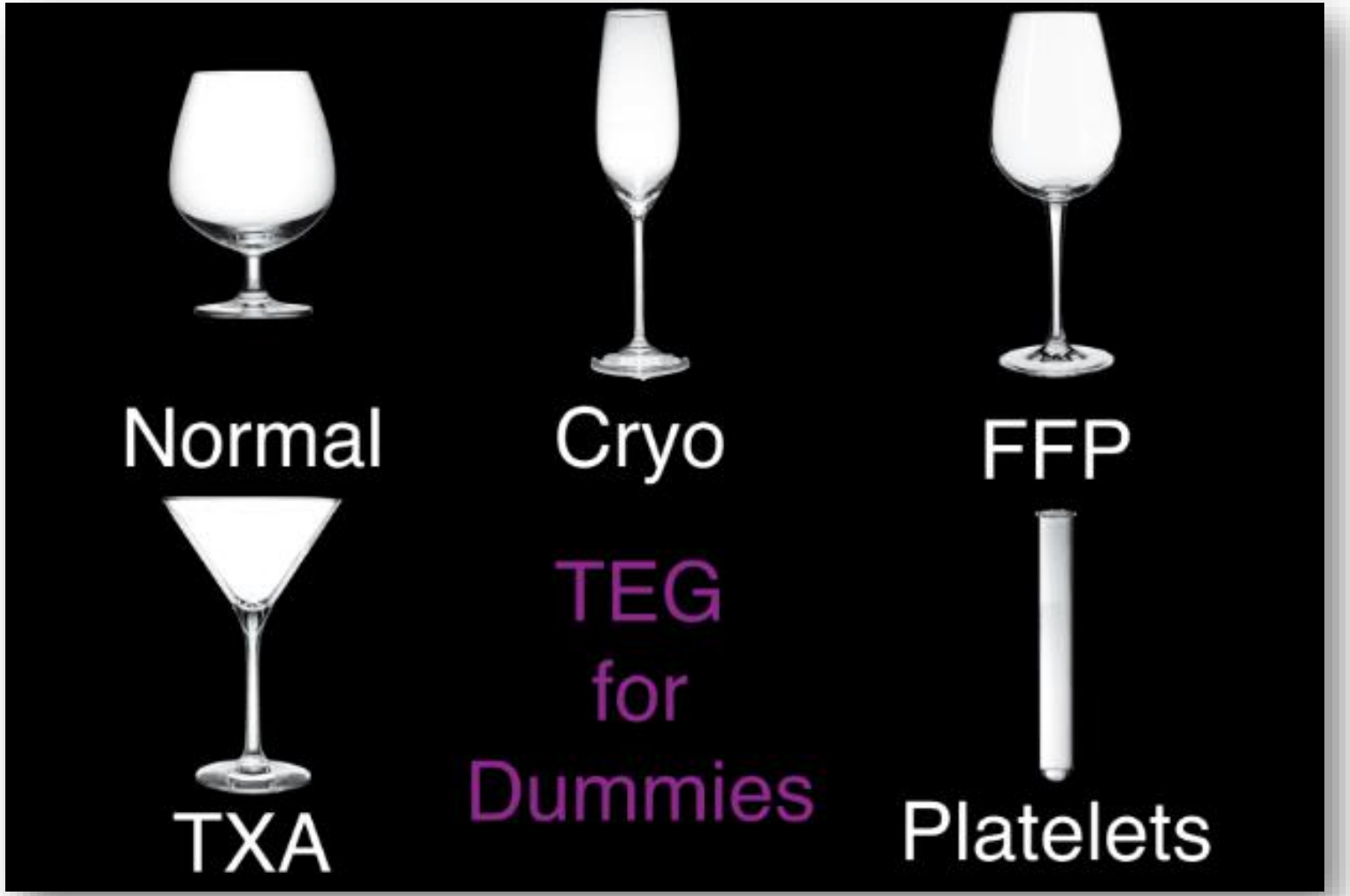
Platelet Blockers
Thrombocytopenia/
Thrombocytopathy
R ~ Normal; K = Prolonged;
MA = Decreased

Fibrinolysis (UK, SK, or t-PA)
Presence of t-PA
R ~ Normal;
MA = Continuous decrease
LY30 > 7.5%; WBCLI30 < 97.5%;
Ly60 > 15.0%; WBCLI60 < 85%

Hypercoagulation
R;K = Decreased;
MA;Angle = Increased

D.I.C
Stage 1
Hypercoagulable state with
secondary fibrinolysis

Stage 2
Hypocoagulable state



Massive Haemorrhage & assorted coagulopathies

People who:

lose more than 30% of their blood volume

and have

rapid on-going blood loss

are at an increased risk of

haemorrhagic shock

and

coagulopathy

with

life-threatening consequences

Know the EBV Rule context: 30%

Estimated Blood Volume (EBV) in mL = Weight (kg) × Blood Volume Factor (mL/kg)

Estimated Blood Volume (EBV) in L = EBV (mL) ÷ 1000

Population Group	Blood Volume Factor (mL/kg)	Typical Range
Adult Male	75	70-80
Adult Female	65	60-70
Child (1-12 years)	80	75-80
Infant (1-12 months)	85	85-90
Neonate (0-28 days)	95	90-100
Preterm Infant	105	100-110

- 70kg male: 5.75 L
- 3kg neonate: 285 mL

[Estimated Blood Volume (EBV) Calculator | Lab Tests Guide]

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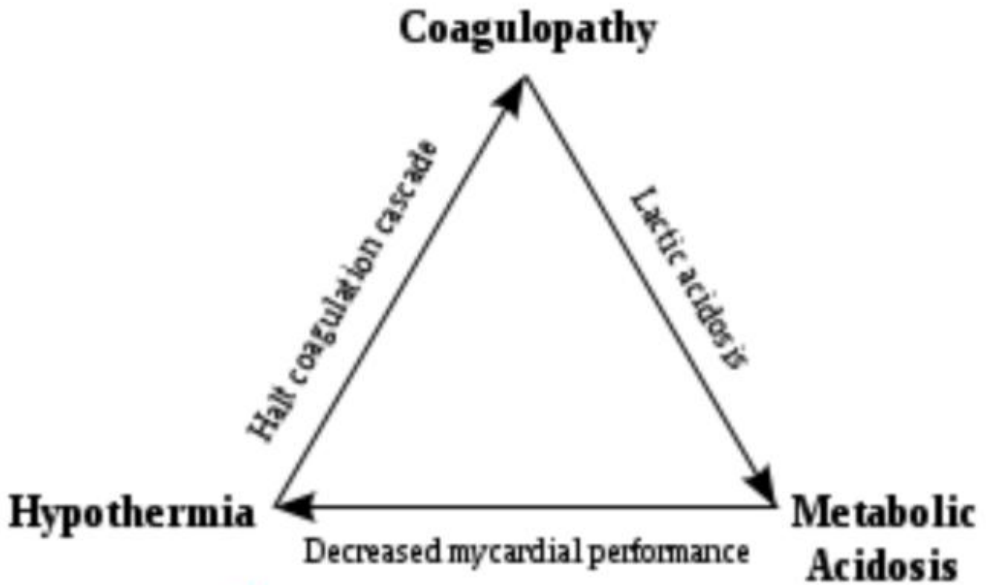
Advanced Trauma Life Support (ATLS)

Classifications of Hypovolaemic Shock

Clinical class of shock	Amount of blood lost		Blood pressure		Pulse rate	Respiratory rate	Extremities	Mental state
	Volume	Percentage	Systolic	Diastolic				
Class I	~750 ml	<15%	Normal	Normal	Normal	Normal	Normal	Alert
Class II	800 – 1500 ml	15–30%	Normal	↑	100 –120	Normal	Pale	Anxious or aggressive
Class III	1500 – 2000 ml	30–40%	↓	↓	~120+	↑~ 20/min	Pale	Anxious, aggressive or drowsy
Class IV	>2000 ml	>40%	↓↓	↓↓	120+ & thready	↑↑> 20/min	Ashen & cold	Drowsy, confused or unconscious

American College of Surgeons (2008). *Atls, Advanced Trauma Life Support Program for Doctors*. Amer College of Surgeons.

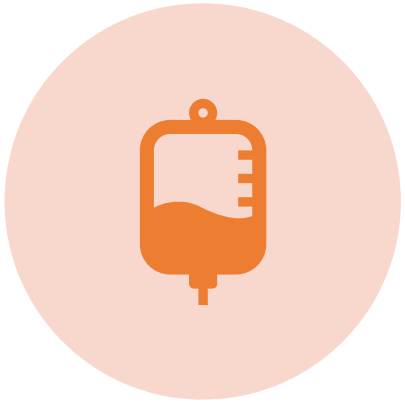
Guard against



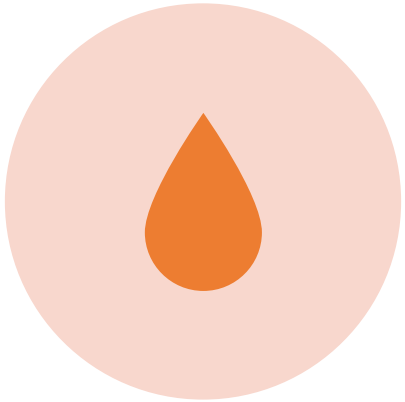
Lethal Triad

https://www.jems.com/patient-care/trauma-s-lethal-triad-hypothermia-acidos/

Stop the TRIAD

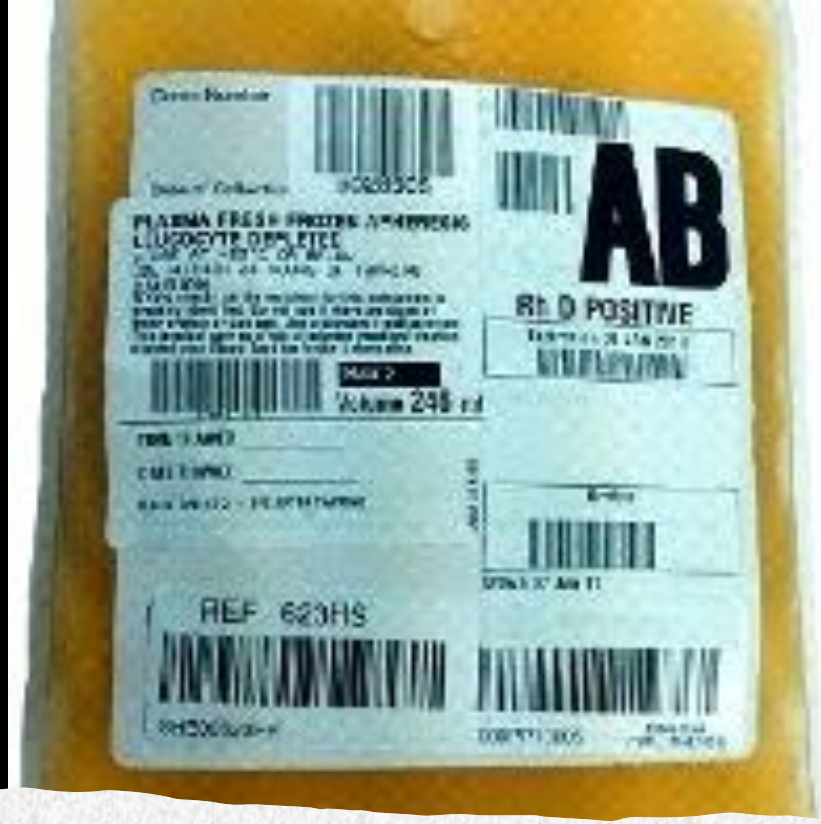


RED CELLS
TO MITIGATE ACIDOSIS



PLASMA & PLATELETS
TO CORRECT COAGULOPATHY
(& RESTRICT CRYSTALLOIDS)

WARM PATIENT & FLUIDS TO
COMBAT HYPOTHERMIA
(& HASTEN SURGICAL REPAIR)



Maximizing the power of red and gold;

(You may also need the massive haemorrhage pathway - MHP)

MHPs rapidly respond to blood loss, their ‘recipe’ is underpinned by haemostasis

Massive haemorrhage + shock

Initiate STAT PACK

Assess

Activate MHP

Haemostasis achieved → STOP

A ‘code sign’ to notify your local BB of a massive haemorrhage

The process to notify your BB can vary across Aotearoa – find out how you activate locally

MHPs provide an ingredient list to best suit (not tailored)

The pathway ‘recipes’ change according to:

- the type of bleed (adult) or
- weight | EBV of child (paed)

Dunedin Adult Massive Haemorrhage Pathway

Massive Bleeding PLUS **Shock Signs** or **HR > 120** or **SBP < 90**

Initiate STAT PACK + send urgent group & screen to Blood Bank (6 mL pink top → hand write ID details)

CODE CRIMSON Call	STANDARD* MHP	OBSTETRIC MOB Call
Via VINA +/- 777	Medical or Surgical Bleeding	Via 777
2g Tranexamic Acid	**1g Tranexamic Acid**	**1g Tranexamic Acid**
administer IV (0.5 mL/min)	administer IV (0.5 mL/min)	administer IV (0.5 mL/min)
Blood Bank auto-release	☎ Dial 777, state your emergency:	Blood Bank auto-release
CRIMSON STAT PACK	**'STAT PACK'** + NHI, name, location	**MOB STAT PACK**
(2 RBC + 2 FFP)	(2 RBC)	(2 RBC)
	* ICU Chest Re-opening → Blood Bank auto-release	

Re-Assess Ongoing, uncontrolled Massive Bleeding & Shock?

Activate MHP ☎ **Dial 777**, state your emergency:

'MHP – crimson or **standard** or **obstetric'**

Provide patient NHI, name, location & name of the MHP guardian

Crimson MHP Box 1	Standard MHP Box 1	Obstetric MHP Box 1
(starts @ **Box 2**)	2 RBC, 2 FFP	2 RBC, 3 Cryo

1st Box is auto-released

Call Blood Bank →
- To **request 2nd Box** on
- Boxes 2 & 3 alternate

Dial 777 →
- If patient location changes
- To **stop the MHP** when bleeding slows (then start targeted transfusion)

MHP Bloods#

Repeat every 30 minutes

Blood gas; iCa^{2+}; FBC; Coags + Fibrinogen

#Awanui lab form in MHP Box
- If available use TEG*

Box 2
4 RBC, 4 FFP, 3 Cryo

Administer 1g of calcium^ with each Box

Box 3
4 RBC, 4 FFP, 1 Platelet

Adjunct Medications

Calcium & Tranexamic Acid
- Located on crash trolley
- Don't co-mix with blood

^ Calcium 1g →
- 10 mL $CaCl_2$ 10% **or**
- 30 mL Ca^{2+} Gluconate 10%

Obstetric Haemorrhage

1. **Manage the 4 T's** (tone, trauma, tissue, thrombin)
2. If significant, ongoing bleeding @ 30 minutes, **administer additional 1g tranexamic acid**

Coagulation Targets	If target not met ⇒ give additional:
PR < 1.5 | **APTT** < 40	4 units FFP
Fibrinogen > 2 g / L	3 units Cryoprecipitate
Platelets > 75 x 10^9/ L	1 unit Platelets
Ionised Ca > 1.1 mmol / L	1g Calcium

Contact Numbers

Blood Bank: 03 470 9369 or ext. 59369

MHP Guardian: 027 591 0235

Dunedin Paediatric Massive Haemorrhage Pathway

Massive Bleeding PLUS signs of Shock or Coagulopathy

INITIATE **Dial 777**, state your emergency: "Paediatric Stat Pack"
Provide NHI, name, gender, weight & location

SEND Group & Screen to Blood Bank (if 6 mL pink-top: minimum volume = 1 mL → Hand-write ID details)

CONSIDER Tranexamic Acid (15 mg/kg to maximum 1g)

Paediatric Stat Pack: 2 RBC (1 RBC if < 10 kg)

Transfuse 10 mL/kg **OR** 1 unit RBC if > 30 kg - then **reassess**

Ongoing bleeding or shock then transfuse 10 mL/kg **OR** 1 unit if > 30 kg

REASSESS Ongoing Massive Bleeding or Shock?

ACTIVATE **Dial 777**, state your emergency:
"Paediatric MHP – alpha, bravo, charlie or standard"
Provide patient NHI, name, weight, location & MHP guardian's name

ALPHA 0 - 10 kg	BRAVO 11 - 20 kg	CHARLIE 21 – 45 kg	Adult Standard MHP > 45 kg
ALPHA BOX 1 RBC, 1 FFP, 1 Platelet & 1 Cryoprecipitate	**BRAVO BOX 1** 1 RBC & 1 FFP	**CHARLIE BOX 1** 2 RBC & 2 FFP	**STANDARD BOX 1** 2 RBC & 2 FFP
Draw via a blood filter & transfuse in a 10 mL/kg sequence, as follows:	**With each Box, give 0.3 mL/kg Calcium gluconate 10%**		With each Box, give 10mL Ca. chloride , OR 30 mL Ca. gluconate
First Round RBC ⇒ FFP ⇒ RBC ⇒ Cryoprecipitate ⇒ **Then give** 0.45 mL/kg Calcium gluconate 10%	**BRAVO BOX 2** 1 RBC 1 FFP 1 Cryoprecipitate	**CHARLIE BOX 2** 2 RBC 1 FFP 2 Cryoprecipitate	**STANDARD BOX 2** 4 RBC 4 FFP 3 Cryoprecipitate
Second Round RBC ⇒ FFP ⇒ RBC ⇒ Platelet ⇒ **Then give** 0.45 mL/kg Calcium gluconate 10%	**BRAVO BOX 3** 1 RBC 1 FFP 150 mL Platelets	**CHARLIE BOX 3** 2 RBC 2 FFP 1 Platelet	**STANDARD BOX 3** 4 RBC 4 FFP 1 Platelet
Repeat, until bleeding slows, then **Stop MHP via 777**	**Alternate Box 2 & 3,** until bleeding slows, then **Stop MHP via 777**	**Alternate Box 2 & 3,** until bleeding slows, then **Stop MHP via 777**	**Alternate Box 2 & 3,** until bleeding slows, then **Stop MHP via 777**

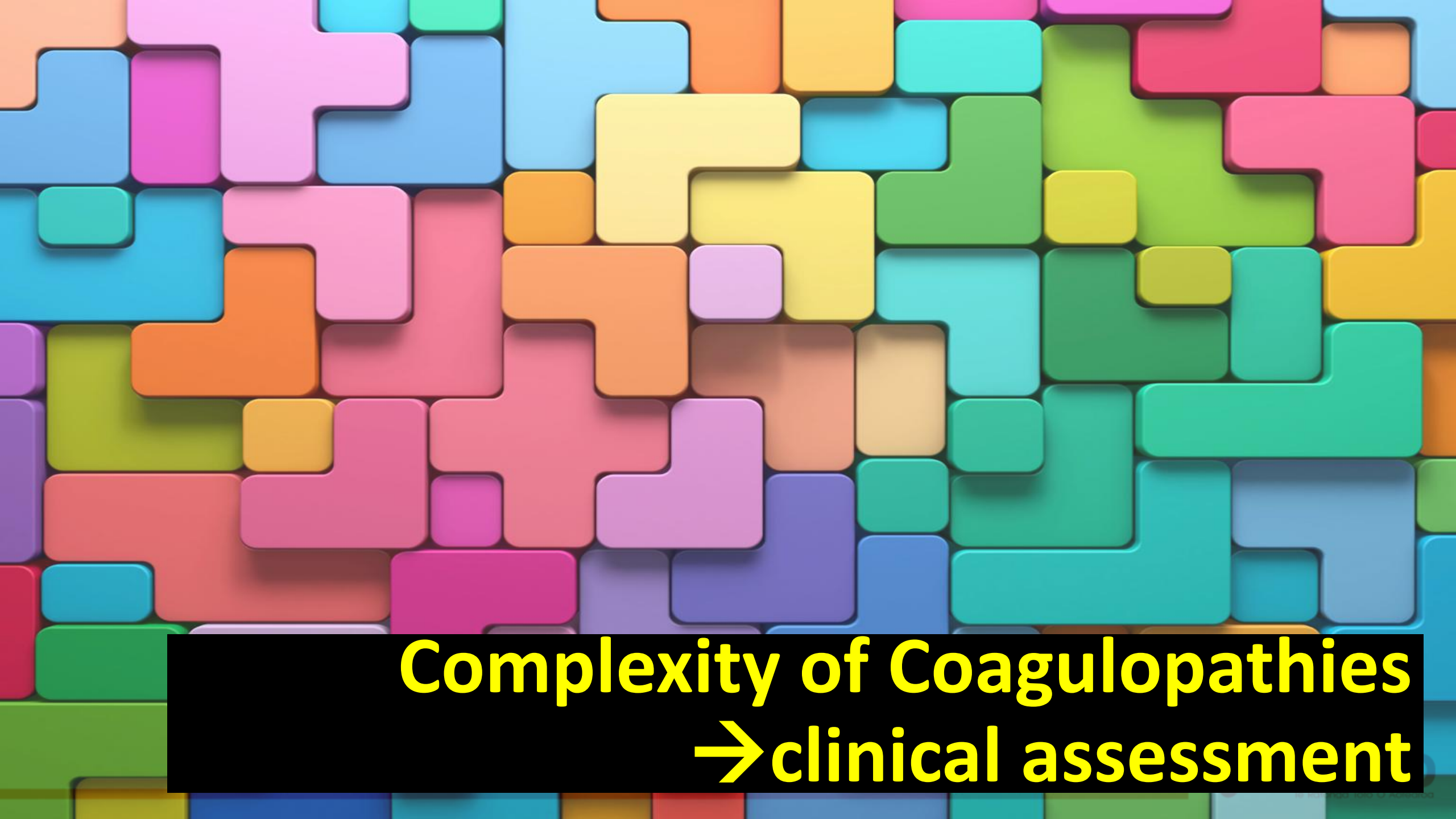
Coagulation Targets	If target not met ⇒ give additional:	Bloods ⇒ repeat every 30 minutes
PR < 1.5 | APTT < 40	FFP → 20 mL / kg	Blood Gas (include K+/Ca++)
Fibrinogen > 1 g / L	Cryoprecipitate → 5 mL / kg	FBC
Platelets > 75 x 10^9/ L	Platelets → 10 mL / kg	Coags (including Fibrinogen)
Ionised Ca > 1.1 mmol / L	Calcium gluconate 10% → 0.3 mL / kg	TEG* if available

Box One is auto-released

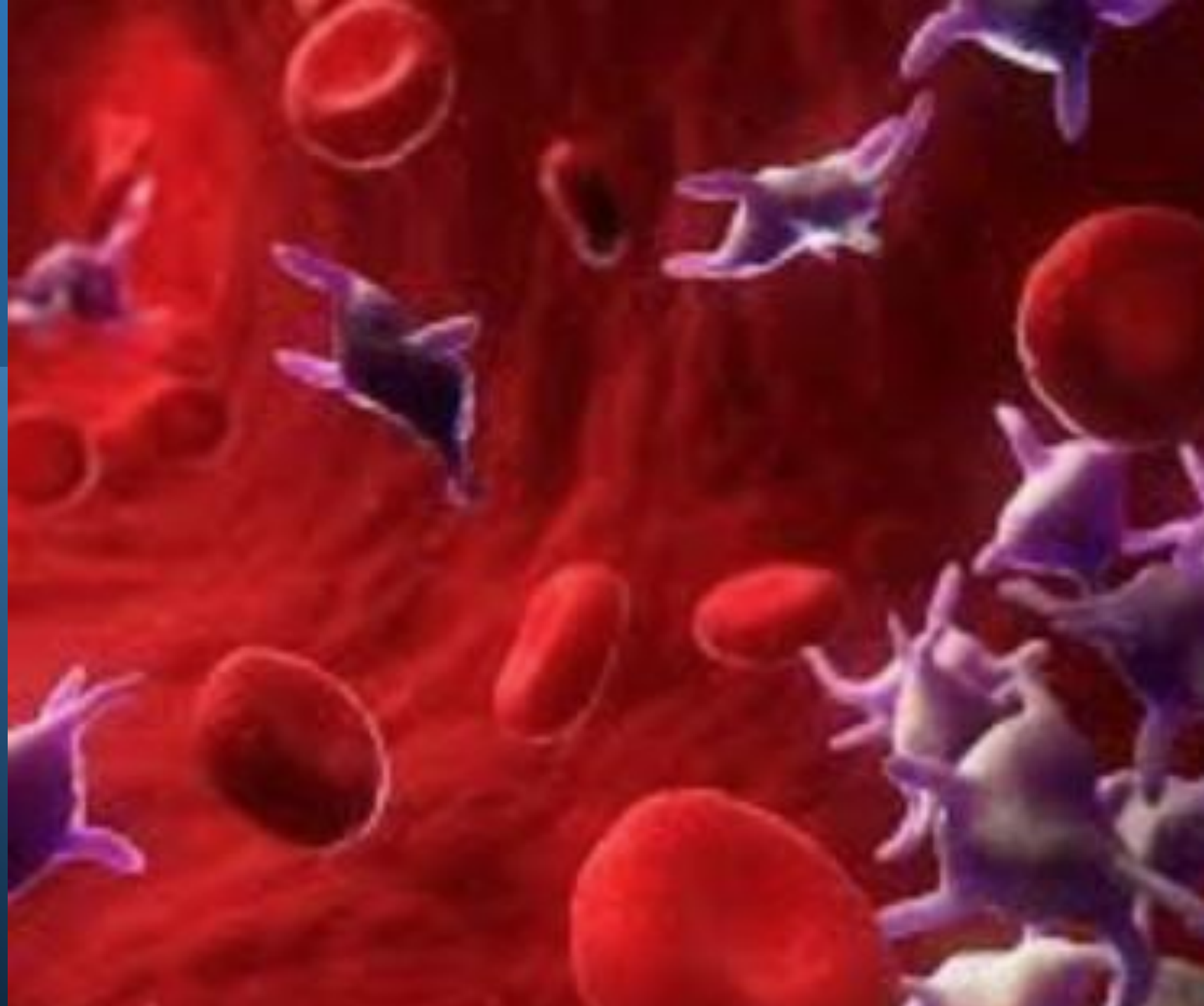
☎ Call BB + 470 9369 to request Box Two onwards

☎ MHP Cell = 027 591 0235

Dunedin Hospital examples

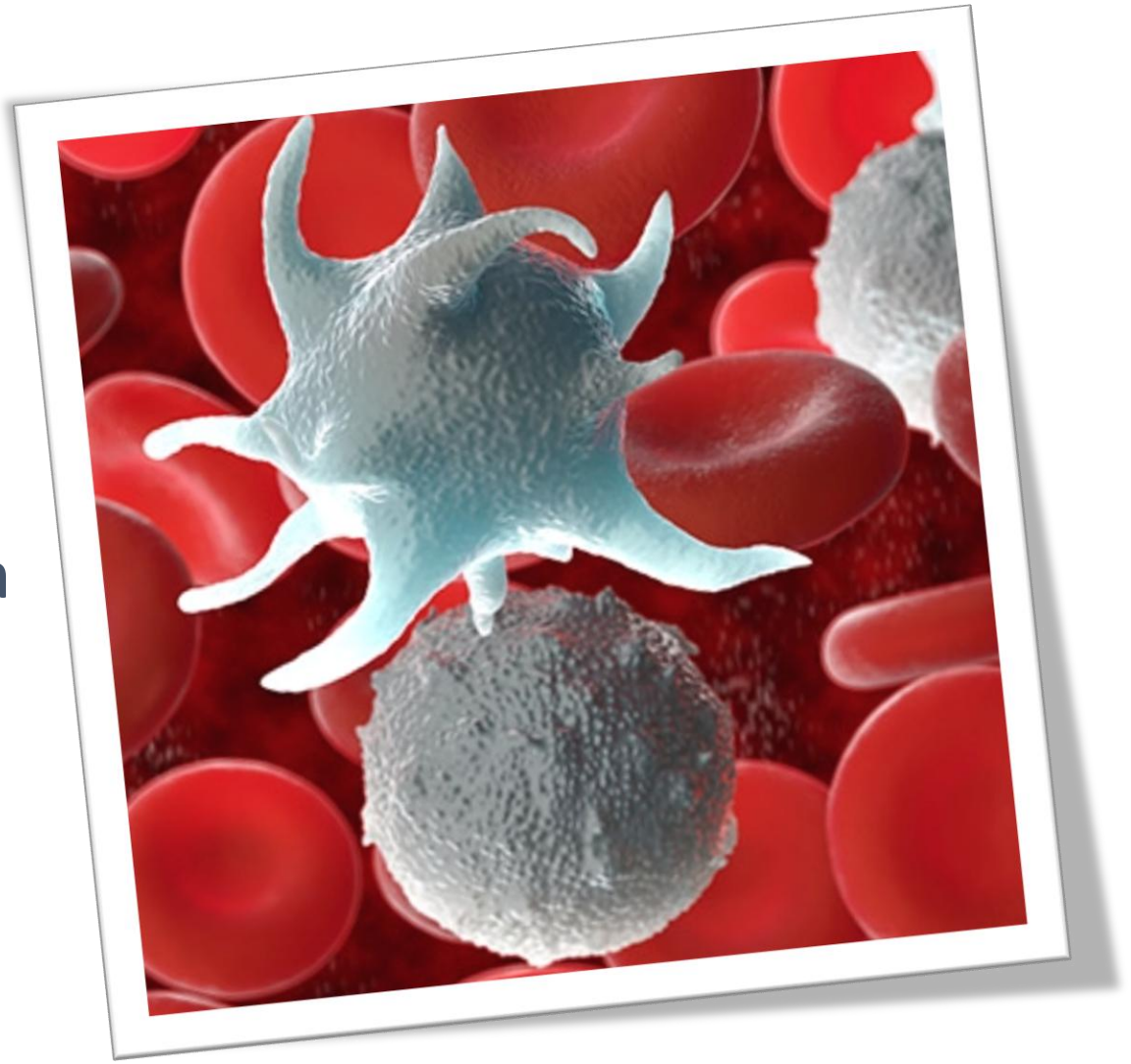

Complexity of Coagulopathies
→clinical assessment

Platelet-related Coagulopathy



Thrombocytopenia

- Failure of bone marrow e.g. AML, AA
- Shortened life span e.g. ITP, drugs, DIC
- Sequestration e.g. CABG, hypersplenism
- Dilution e.g. massive transfusion



Platelet Dysfunction

Consider if there are clinical symptoms & signs of thrombocytopenia in the presence of a normal or moderately reduced platelet count

- Inherited, e.g. vWD
- Acquired, e.g. medications (DAPT), OTC (ST Johns Wort), foods (garlic, chocolate), cRF, CABG

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Induced Coagulopathy

Dilutional Coagulopathy

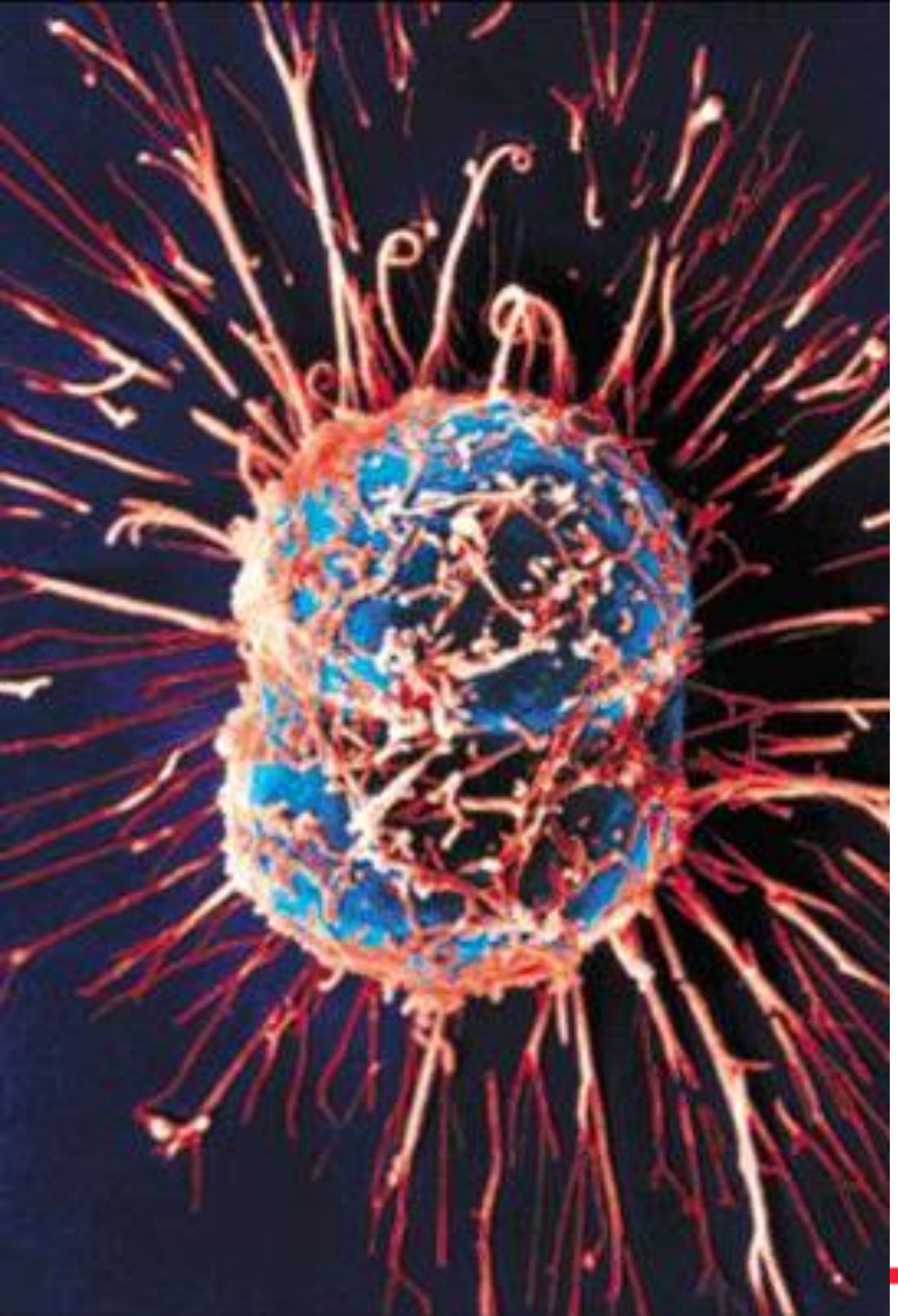
Multiple transfusion of red cells or crystalloid/colloid volume depletes available platelets and coagulation factors

- Red cell transfusion replaces red cells only

Increases the destruction of platelets & coagulation factors

Hypothermia

Decreases platelet function & coagulation



Neoplasia

- Malignancy induced thrombocytopenia (e.g. leukaemia)
- Treatment (chemo) induced thrombocytopenia

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Drug induced coagulopathy

Drugs that interfere with haemostasis

Those that alter **platelet function** e.g. aspirin, NSAID

Those that **interfere with coagulation** e.g. warfarin, heparin

*Those that increase the rate of **breakdown of stabilized fibrin** e.g. t-PA, urokinase.*

*Those that **stabilize fibrin**, e.g. TXA*

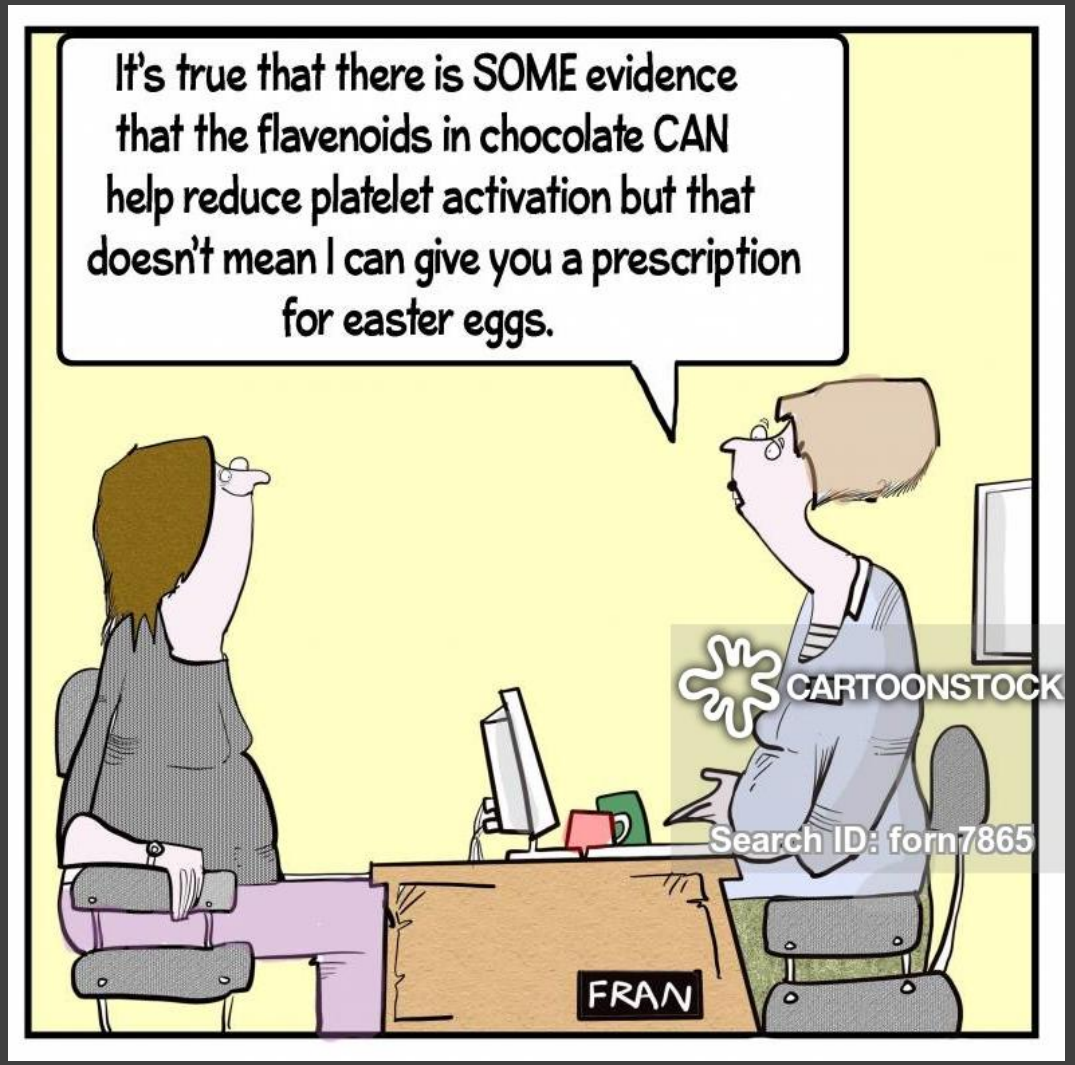
Anti-Platelet Agents!

Aspirin

- prevents the formation of thromboxane by binding irreversibly to the enzyme required

NSAIDs

Chocolate, garlic…..

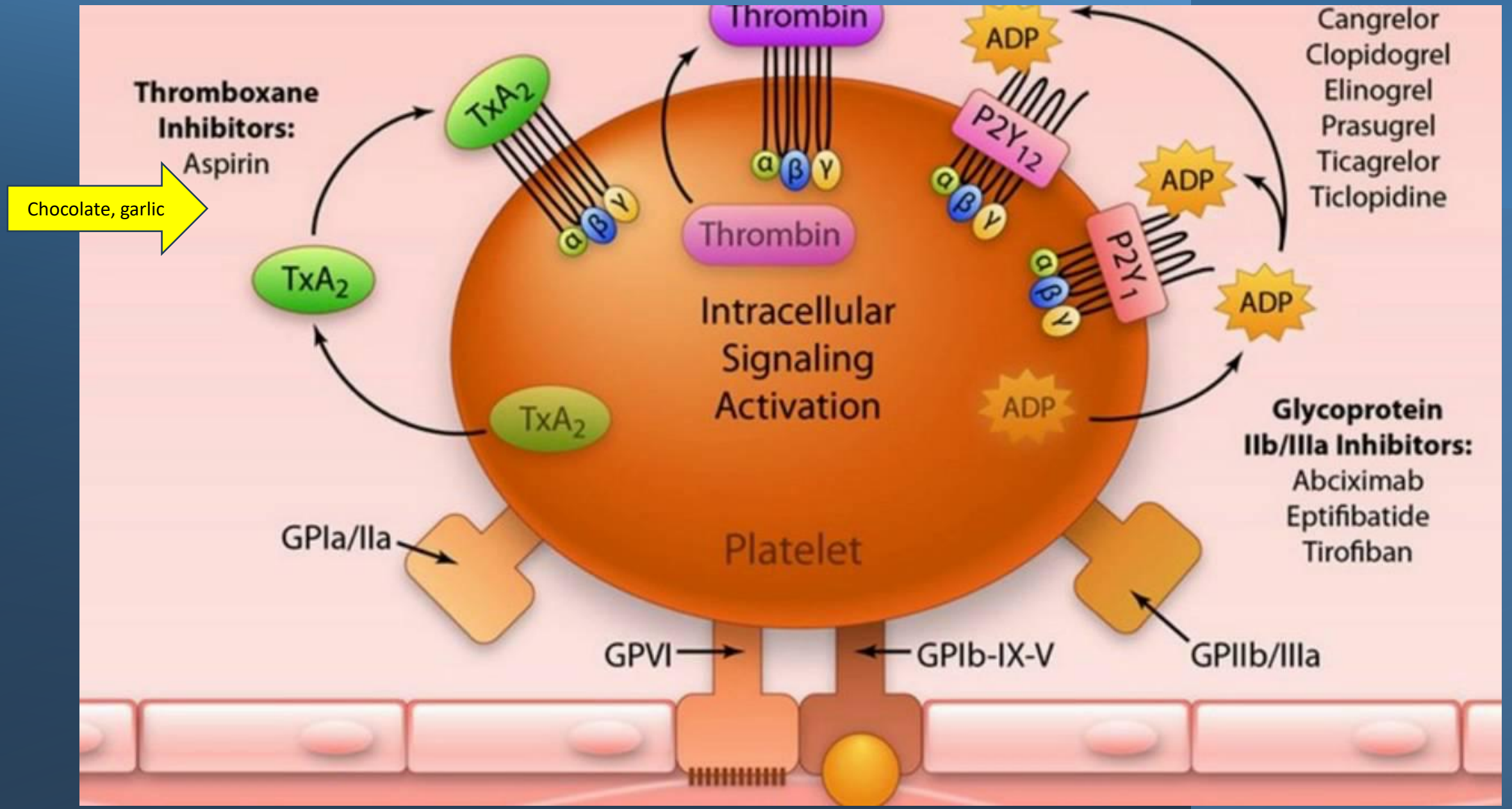


Some drugs **inhibit the action of thromboxane and ADP** on platelets

E.g. Ticlopidine, clopidogrel & dipyridimole (enhances aspirin)

DAPT- dual anti-platelet therapy
- Two anti-platelet medications combined

- Commonly combines aspirin with a P2Y12 inhibitor (clopidogrel, ticagrelor)


Thromboxane Inhibitors:
Aspirin
Chocolate, garlic
TxA2
TxA2
TxA2
Thrombin
Thrombin
ADP
P2Y12
ADP
P2Y1
ADP
ADP
Cangrelor
Clopidogrel
Elinogrel
Prasugrel
Ticagrelor
Ticlopidine
Intracellular
Signaling
Activation
Platelet
Glycoprotein
IIb/IIIa Inhibitors:
Abciximab
Eptifibatide
Tirofiban
GPIa/IIa
GPVI
GPIb-IX-V
GPIIb/IIIa

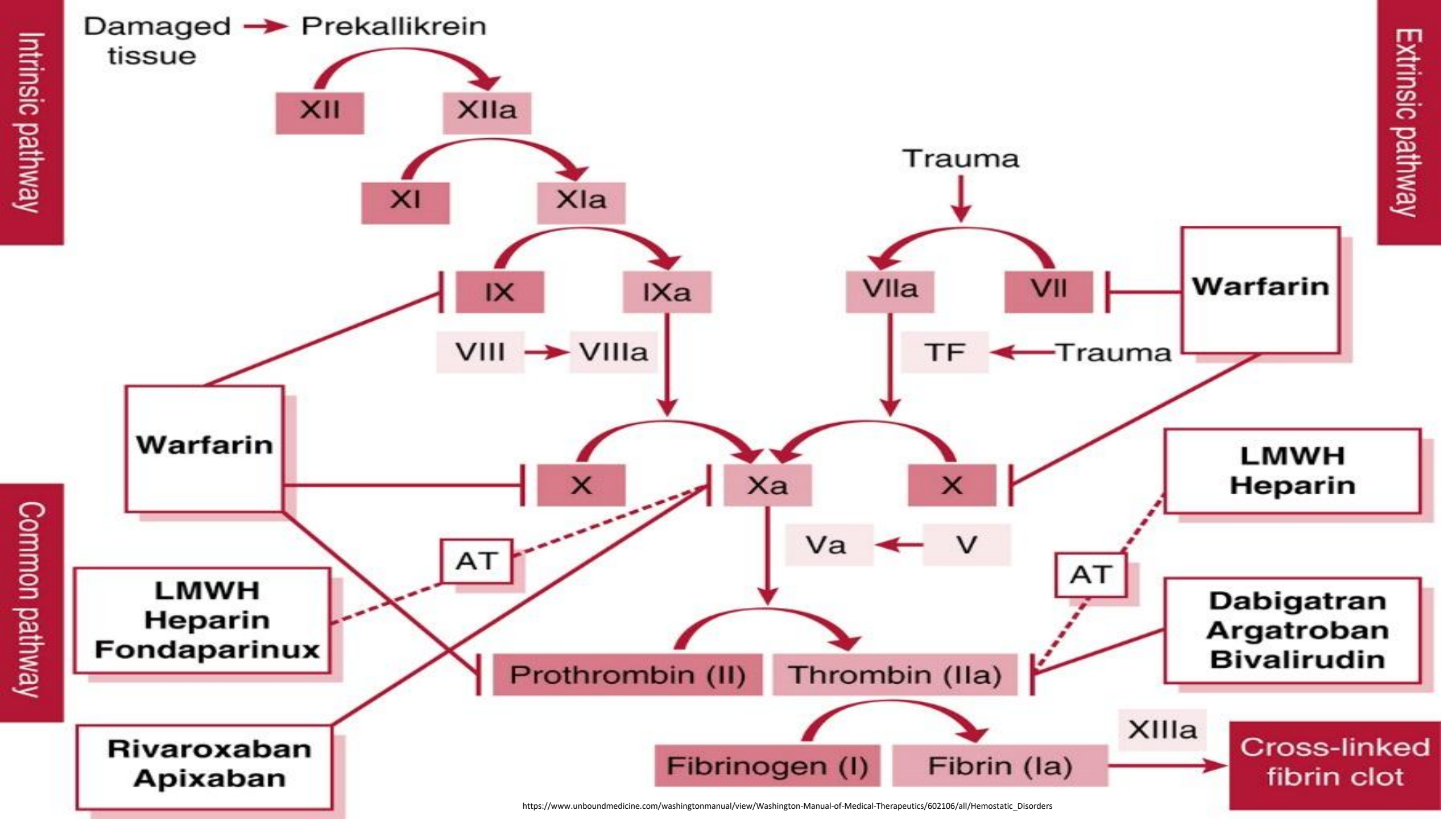
Anticoagulants

Oral

- Warfarin- vitamin K antagonist (VKA)
- Dabigatran – direct thrombin inhibitor (a DOAC)
- Rivaroxaban – direct FXa inhibitor (a DOAC)

IV

- (UF) Heparin – potentiates antithrombin III & anti FX



https://www.unboundmedicine.com/washingtonmanual/view/Washington-Manual-of-Medical-Therapeutics/602106/all/Hemostatic_Disorders

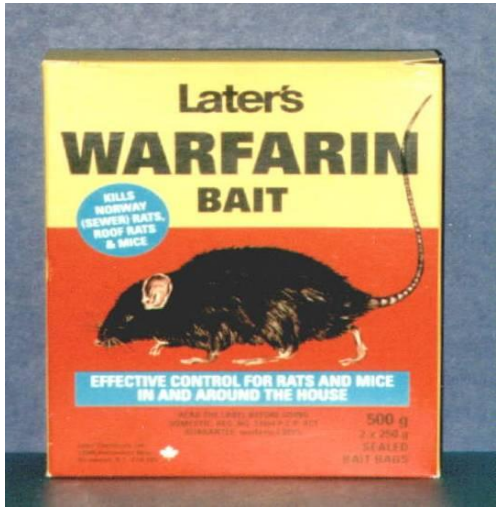
Anti-coagulants (courtesy Dunedin Pharmacy)

Anticoagulant	Onset & duration of action	Pre-operative	Renal impairment	Antidote/reversal
Warfarin	Onset: 36-72 hours Duration: 48-96 hours	Varies, usually stop warfarin 5-7 days prior, may need to bridge w enoxaparin	No adjustment needed	Vitamin K 4FPCC : Beriplex NZ
Dabigatran	Onset: 30 minutes Duration: 24-36 hours	Stop 1-2 days before surgery	Contraindicated if CrCl<30mL/min, reduce dose if CrCl 30-50mL/min	Idarucizumab (Praxbind)
Rivaroxaban	Onset: within 30 minutes Duration: 24 hours	Stop 1-2 days before surgery	Contraindicated if CrCl<15mL/min, see monograph for CrCl 15-50mL/min	Andexanet alfa (FDA approved - not on NZ schedule) 4FPCC= second line option

Warfarin coagulopathy

Vitamin K antagonist (VKA)

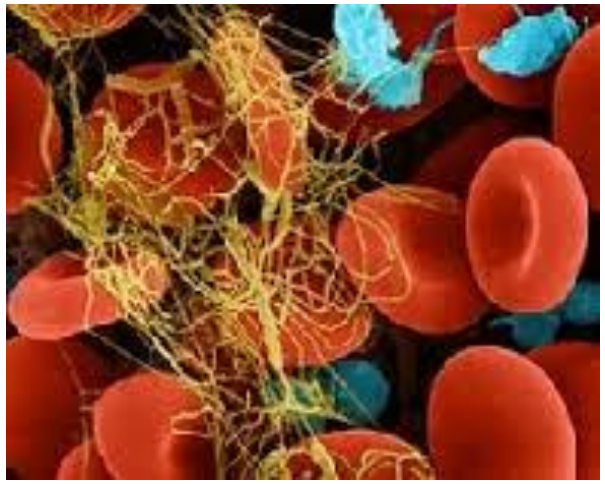
First introduced into commercial use in 1948, and licensed from medicinal use in the US in 1954



https://www.e-jnc.org/m/journal/view.php?number=306

Why is Vit K important?

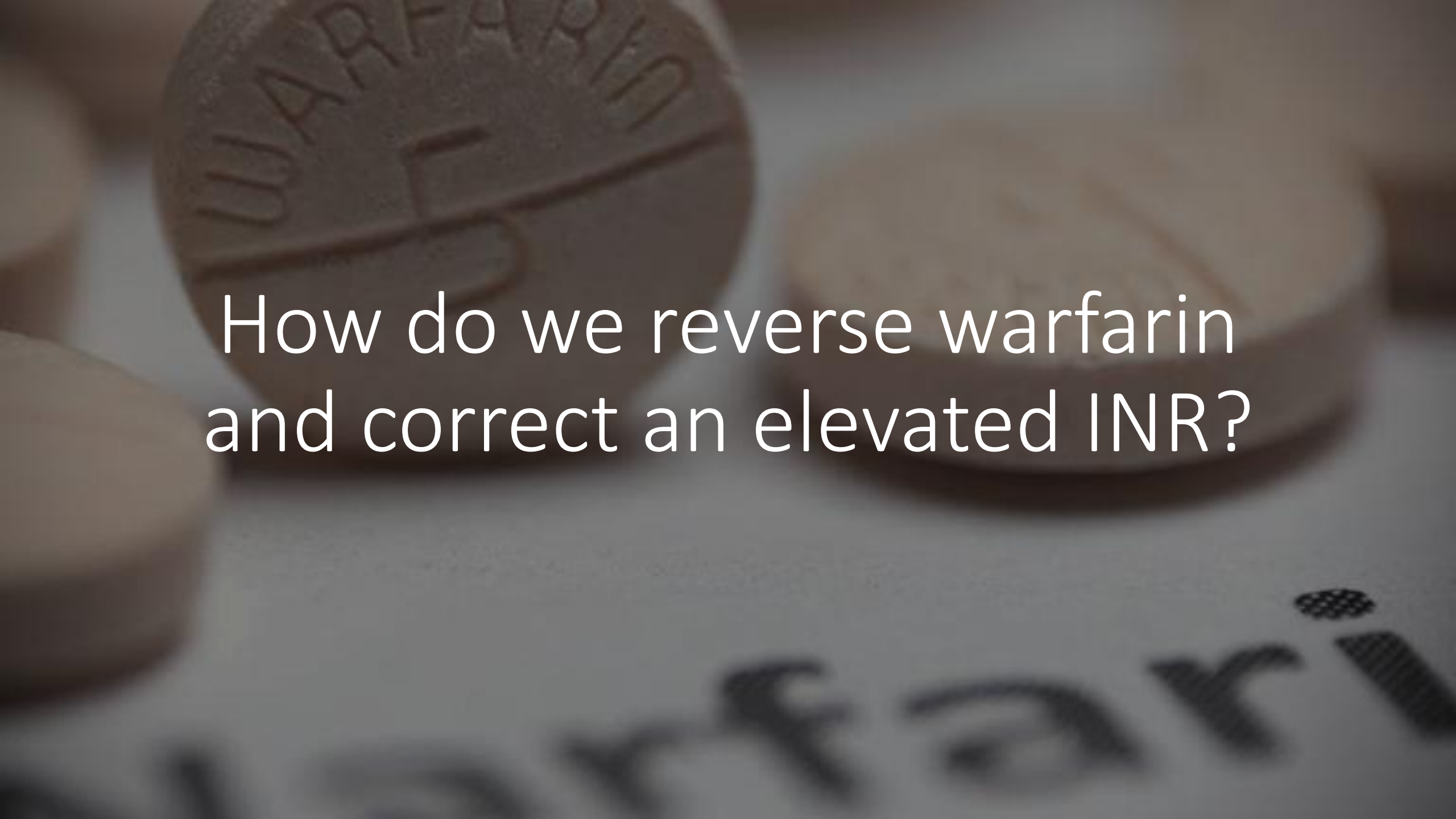
Vitamin K used by liver as a co-enzyme in the manufacture of coagulation factors, specifically factors II, VII, IX & X



Warfarin affects the 'manufacture' of the prothrombin complex, vit K dependent, coagulation factors

→Slows fibrin net development

Multiple factors affect warfarin (diet, medications, age, liver function, gut absorption – with sudden changes transforming 'therapeutic' to 'toxic'



How do we reverse warfarin and correct an elevated INR?

⊕ Routine reversal

Healthcare Professionals

Anticoagulant Reversal

A prothrombin complex concentrate (PCC) contains proteins that are sourced from donated plasma. It is manufactured by a process called fractionation. A PCC is used for a variety of conditions including reversal of anticoagulants such as warfarin or rivaroxaban (a DOAC) in acute bleeding and emergency surgery.

Healthcare Professionals / Anticoagulant Reversal

⊕ Acute warfarin reversal → 4FPCC

✓ Human product → all Transfusion & Traceability rules apply

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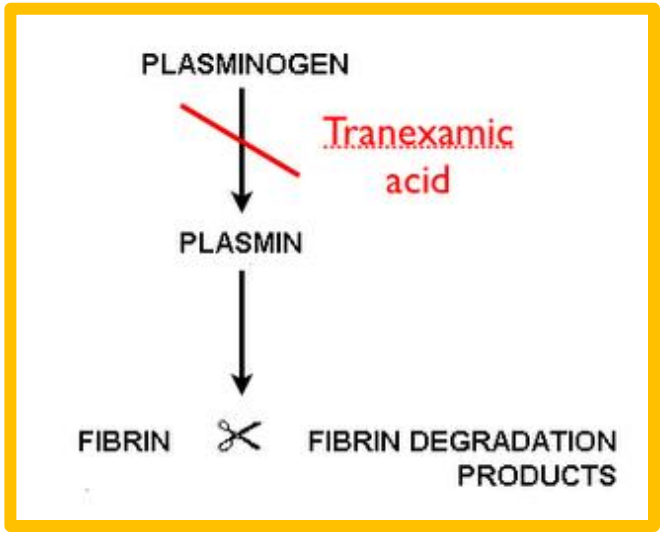
Tranexamic acid (TXA)

anti-fibrinolytic agent which blocks fibrinolysis (enzymatic clot breakdown)

binds to fibrinogen and stops plasminogen binding and activators (e.g. t-PA)



Randomized Controlled Trial > Health Technol Assess. 2013 Mar;17(10):1-79.
doi: 10.3310/hta17100.

The CRASH-2 trial: a randomised controlled trial and economic evaluation of the effects of tranexamic acid on death, vascular occlusive events and transfusion requirement in bleeding trauma patients

I Roberts [1], H Shakur, T Coats, B Hunt, E Balogun, L Barnetson, L Cook, T Kawahara, P Perel, D Prieto-Merino, M Ramos, J Cairns, C Guerriero

Affiliations + expand

PMID: 23477634 PMCID: PMC4780956 DOI: 10.3310/hta17100

Intensive Care Med (2021) 47:14–27
https://doi.org/10.1007/s00134-020-06279-w

SYSTEMATIC REVIEW

Efficacy and safety of tranexamic acid in acute traumatic brain injury: a systematic review and meta-analysis of randomized-controlled trials

Kumait Al Lawati[1,2,3], Sameer Sharif[1,2*], Said Al Maqbali[1,3], Hussein Al Rimawi[1], Andrew Petrosoniak[4], Emilie P. Belley-Cote[2,5], Sunjay V. Sharma[2,6], Justin Morgenstern[7], Shannon M. Fernando[8,9], Julian J. Owen[1,2], Michelle Zeller[10], David Quinlan[1], Waleed Alhazzani[2,11] and Bram Rochwerg[2,11]

THE LANCET

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ARTICLES · Volume 394, Issue 10210, P1713-1723, November 09, 2019 · *Open Access* Download Full Issue

Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH-3): a randomised, placebo-controlled trial

The CRASH-3 trial collaborators †

J Clin Med. 2021 Mar 3;10(5):1030. doi: 10.3390/jcm10051030

Efficacy and Safety of Tranexamic Acid in Emergency Trauma: A Systematic Review and Meta-Analysis

Mahdi Al-Jeabory [1], Lukasz Szarpak [2,*], Kecskes Attila [3], Michael Simpson [4], Adam Smereka [5], Aleksandra Gasecka [6,7], Wojciech Wieczorek [8], Michal Pruc [1], Maciej Koselak [9], Wladyslaw Gawel [10], Igor Checinski [11], Milosz J Jaguszewski [12], Krzysztof J Filipiak [6]

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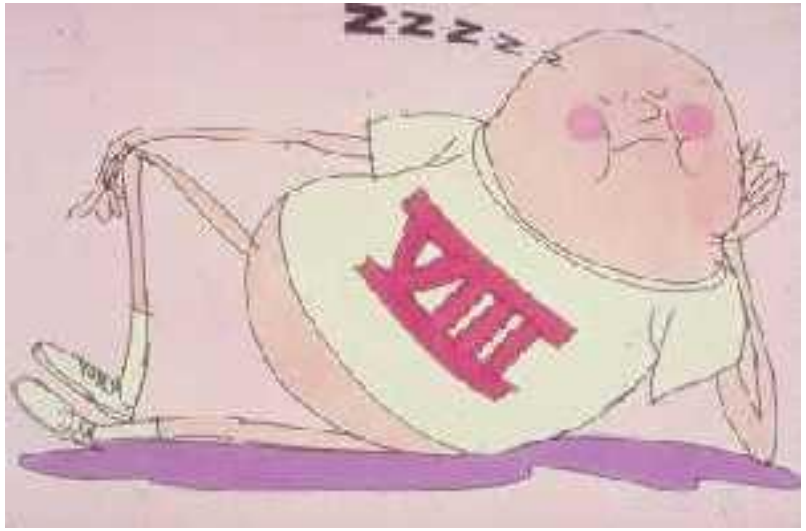
Genetic coagulopathy

Haemophilia



Bleeding disorder in which there is a deficiency or lack of:

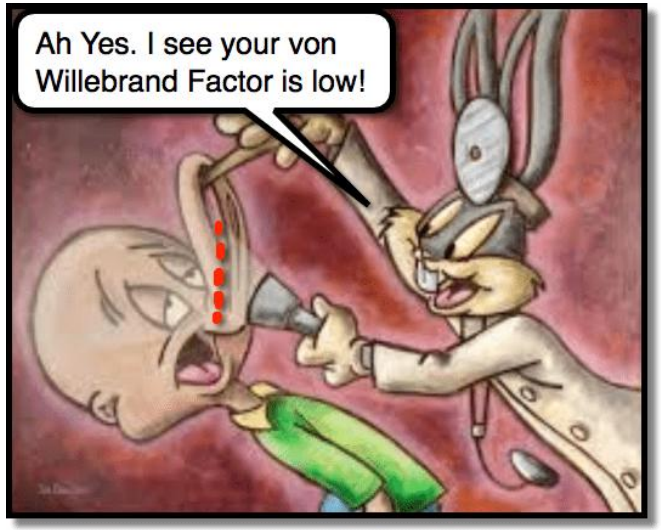
- factor VIII (Haemophilia A)
- factor IX (Haemophilia B or Christmas disease)

Von Willebrand Disease

Deficiency | dysfunctional von Willebrand Factor (vWF) impairs platelet actions during a bleed

clinical picture: slow “oozing” bleeding, especially mucosa, bruising tendency

In addition, FVIII circulates bound to vWF



The plan...

- Physiology of Coagulation
- Key Tests of Coagulation
- Common Coagulopathies
- Massive Haemorrhage

Additional Slides:

DIC Know-How & Light Reading

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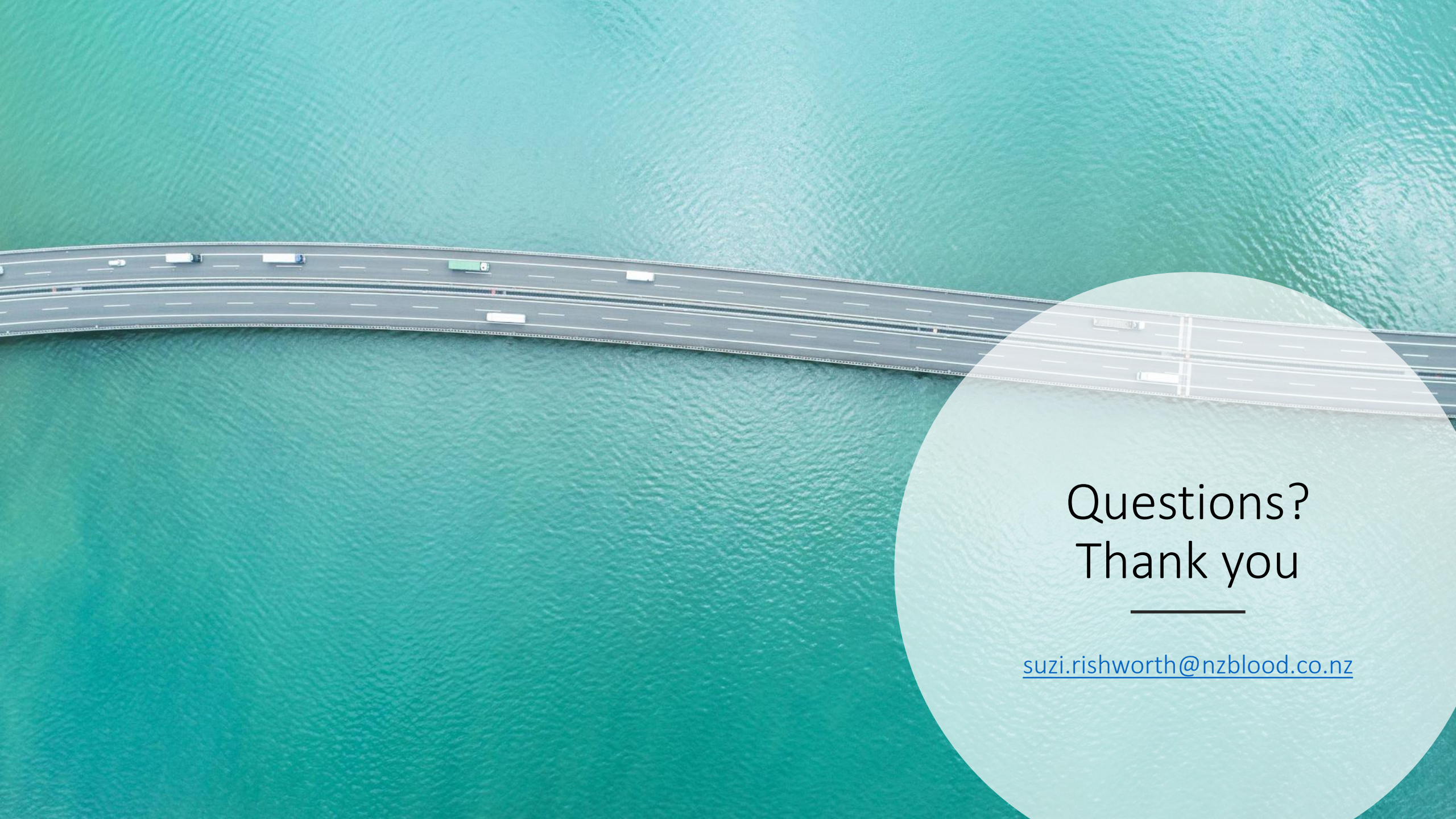


Questions?
Thank you

suzi.rishworth@nzblood.co.nz

Additional Know-How

Using your haemostasis knowledge to understand DIC

DIC

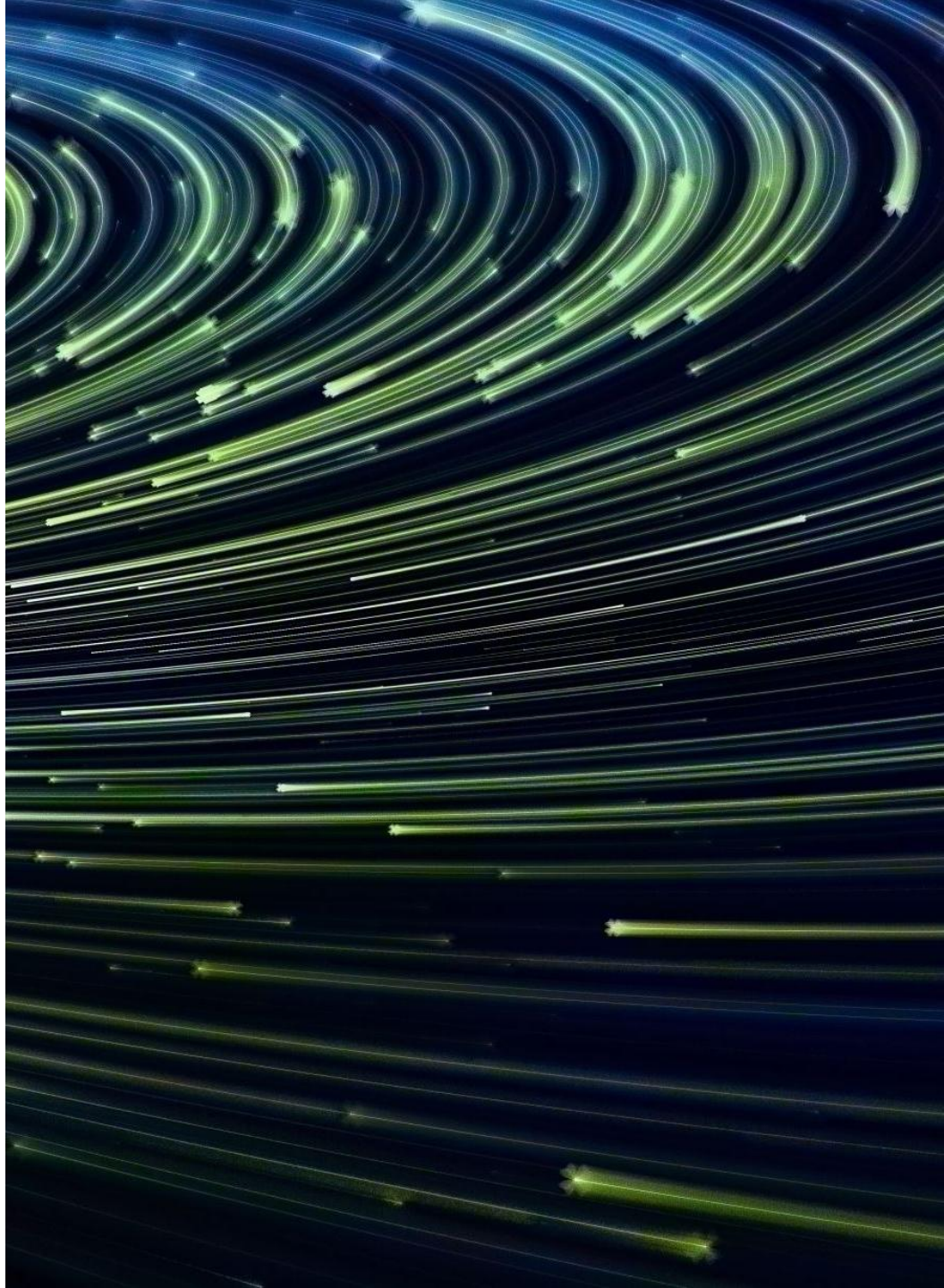
What is DIC?

- Death is Coming
- Drapery Importing Company
- Disseminated Intravascular Coagulopathy
- Drunk in Charge

Disseminated intravascular coagulation (DIC)

a complex clinical syndrome where “chaos” rules; activation of coagulants & platelets occurs abnormally throughout (or parts of) the vasculature

widespread intravascular deposition of fibrin with the consumption of coagulation factors and platelets



Causes of Acute DIC, include:

Endotoxin: gram negative bacteria

Severe persistent shock causing widespread tissue injury

Malignancy- disseminated mucin secreting adenocarcinoma or acute promyelocytic leukaemia

Obstetric emergencies: e.g. amniotic fluid embolism

https://onlinelibrary.wiley.com/doi/full/10.1111/jth.13174

Hypersensitivity: e.g. anaphylaxis

Venom-induced consumptive coagulopathy, (VICC) , varying clinical opinion if true DIC or if VICC a differing entity. Venom activates the coag cascade

https://pubmed.ncbi.nlm.nih.gov/25556574/

Types

Acute DIC:

- associated with fulminant (i.e. sudden in onset, rapid in course) haemorrhagic or thrombotic syndrome

Chronic DIC:

- subtle, slow & evidence of coagulation activation

Intravascular haemostatic response:

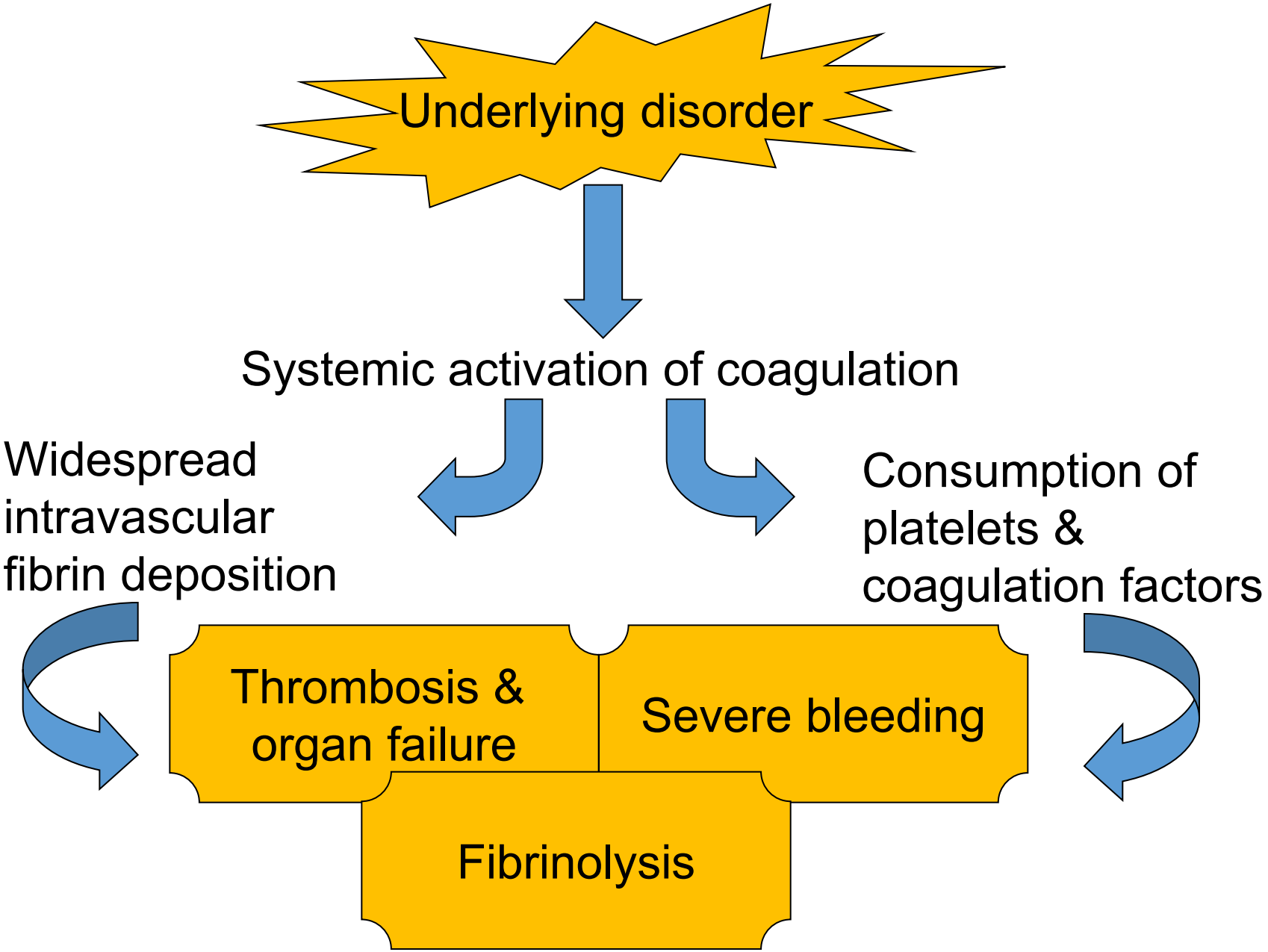
- Explosive generation of thrombin → fibrin deposits in microcirculation & depletes factors

- Circulating fibrin → forms complexes with fibrinogen → forms dysfunctional fibrin net

Intense fibrinolysis stimulated → in the presence of fibrin & thrombi on vascular walls

Fibrinolysis → further depletes fibrinogen, prothrombin, FV & FVIII

Thrombin → causes widespread platelet aggregation/deposits → depletes circulating platelets



Source. M Levi, et al.2001
Molecular Mechanisms of DIC

Clinical Features

Dominated by bleeding

⇒ oozing wounds & venepuncture sites

⇒ may also be generalised : including gastrointestinal, oropharynx, lung

Because→ dysfunctional fibrin net, depleted platelets & coagulation factors

Normal haemostasis is failing

less frequently, microthrombi may cause:

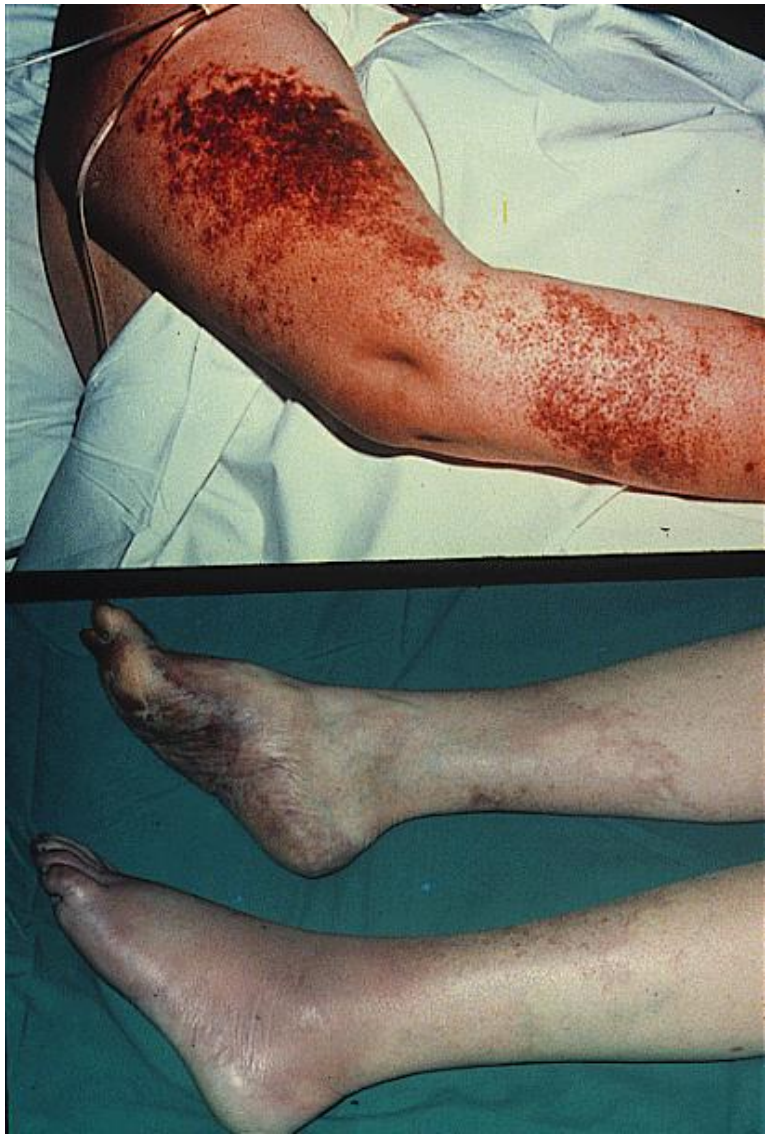
→ skin lesions, renal failure, gangrene, cerebral ischaemia, irreversible organ damage

Skin necrosis secondary to micro-thrombi/DIC

1.indurated & confluent purpura of arm

2. peripheral gangrene with swelling and discolouration

Source: Hoffbrand, et al, 2001.



Diagnosis of DIC

Depends on lab demonstration of:

Falling coagulation factor/platelet levels in a patient with an associated disease known to cause DIC

Evidence of fibrinolysis: very high levels of FDP's & / or D-dimers

Lab findings:

- Reduced/falling platelet count
- Falling fibrinogen
- Rising FDP's and D-dimers (shows fibrin breakdown)

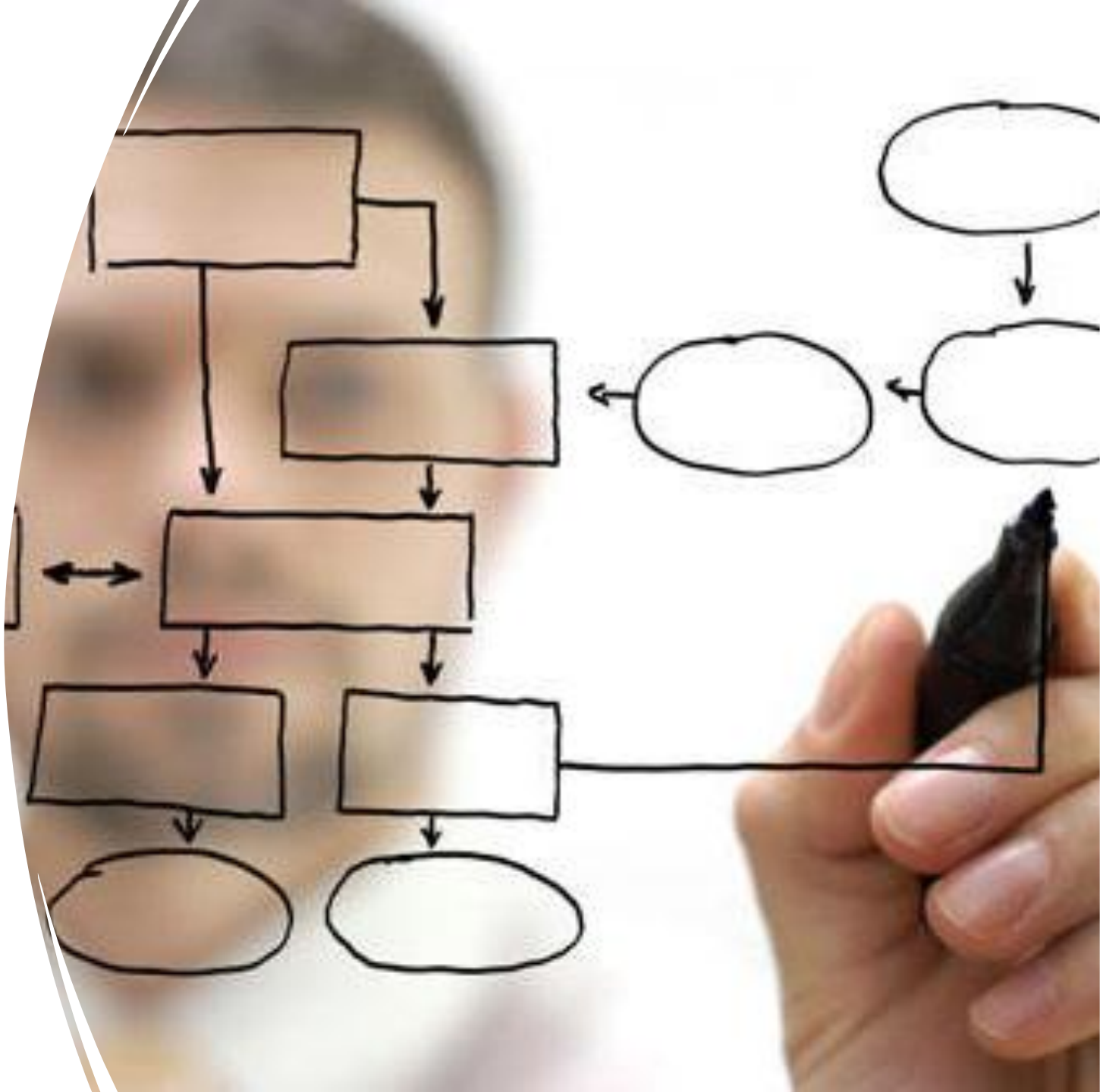
- Later, prolonged INR & APTT

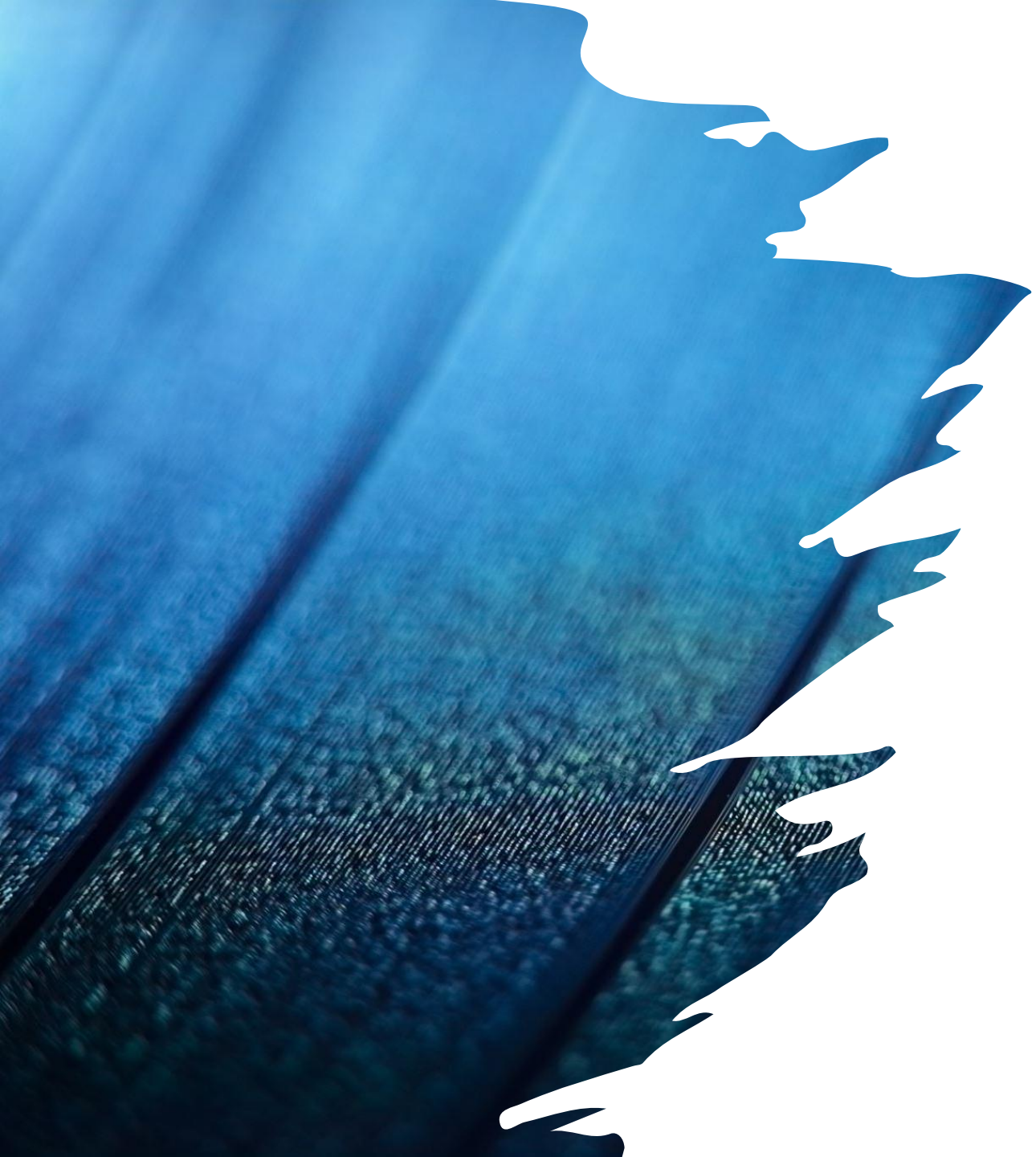
Scoring System for DIC		
Parameter	**Result**	**Score**
Platelet count	>100K <100K <50K	0 1 2
D-dimer	<1mcg/ml 1.0-5.0 mcg/ml > 5.0 mcg/ml	0 2 3
PTT	<3sec >3sec >6sec	0 1 2
Fibrinogen	>100 mg/dl <100 mg/dl	0 1
Score ≥ 5 is indicative of overt DIC with increasing scores correlating with higher mortality.		

https://www.mdcalc.com/isth-criteria-disseminated-intravascular-coagulation-dic#why-use

DIC Treatment

1. Cornerstone: Identify & treat underlying cause





2. Supportive care:

Administer blood components guided by bleeding & lab results

- Platelet replacement
- FFP to replace coagulation factors
- Cryoprecipitate to replace fibrinogen
- Red cells for anaemia from bleeding

- NOTE:
  - Heparin & antiplatelet drugs to inhibit coagulation not usually indicated because can aggravate bleeding
  - Antifibrinolytics, e.g. Tranexamic acid, usually contraindicated because of thrombotic risk
  - Protein C may have some clinical application.

Protein C in DIC ?

Interacts on FV & FVIII.

Essentially it is an anticoagulant, breaking down activated FV & FVIII.

Trials:

variable success/little consensus

Very high cost.

Case by case, specialist request.

Management of DIC

1

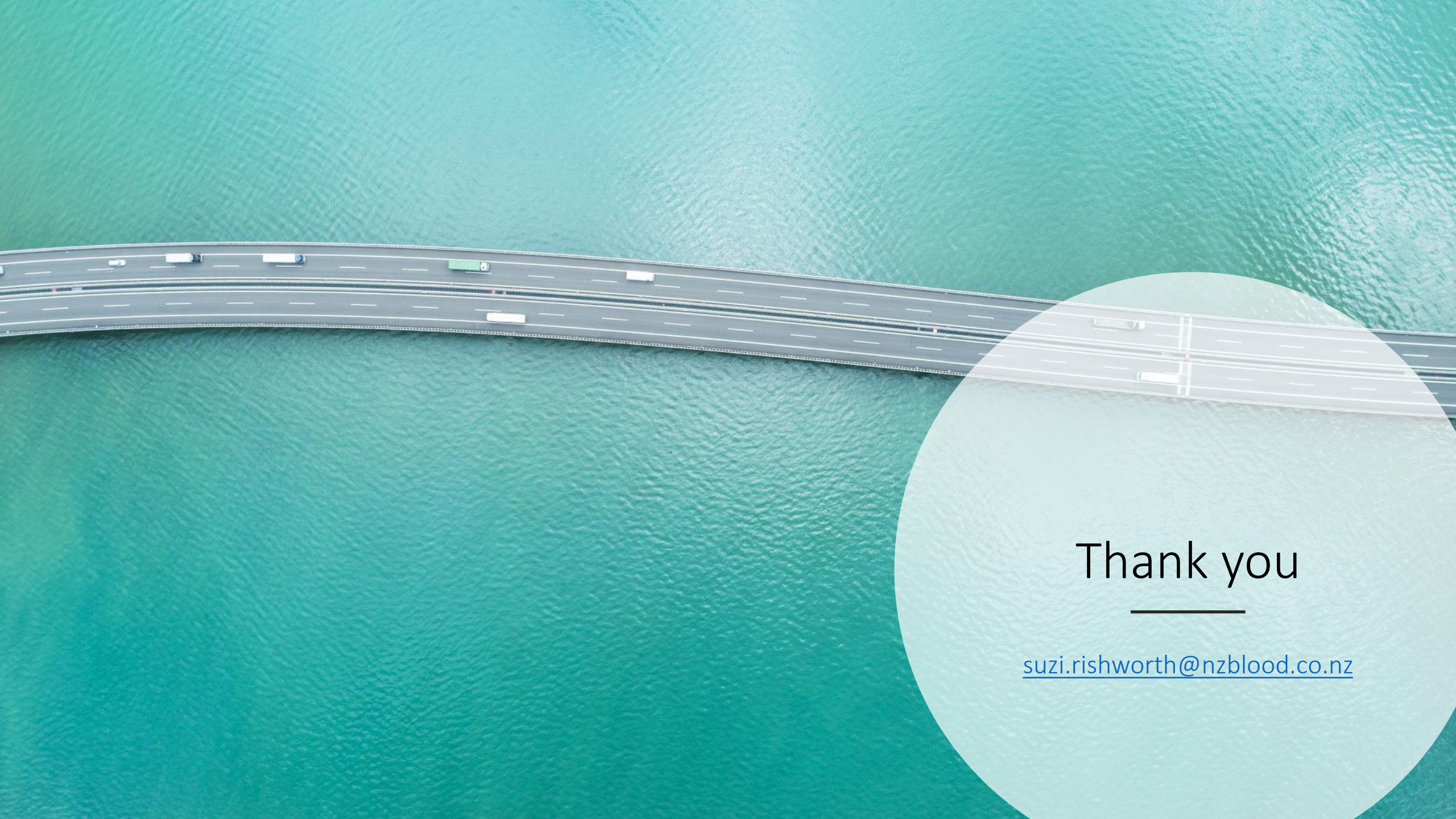
Diagnose accurately so that appropriate treatment to stop the DIC

2

Evaluate severity of DIC

3

If bleeding: appropriate blood components & specialist consultation



Thank you

suzi.rishworth@nzblood.co.nz

Light Reading Supplements

Transfusion medicine

Transfusion medicine handbook

The Transfusion Medicine Handbook is designed to assist hospital staff and other health professionals in modern Transfusion Medicine Practice.

[Transfusion Medicine Handbook | New Zealand Blood Service]

BJHaem
BRITISH JOURNAL OF HAEMATOLOGY

British Society for Haematology
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GUIDELINE | Free Access

Haematological management of major haemorrhage: a British Society for Haematology Guideline

Simon J. Stanworth, Kerry Dowling, Nikki Curry, Heidi Doughty, Beverley J. Hunt, Laura Fraser, Shruthi Narayan, Juliet Smith, Ian Sullivan, Laura Green,
The Transfusion Task Force of the British Society for Haematology

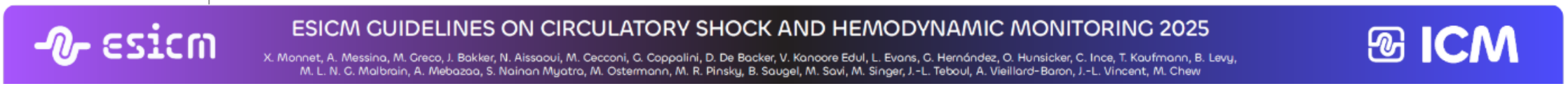
First published: 10 June 2022 | https://doi.org/10.1111/bjh.18275 | VIEW METRICS

Haematological management of major haemorrhage

[New ESICM Guideline on Circulatory Shock and Haemodynamic Monitoring - ESICM]

New ESICM Guideline on Circulatory Shock and Haemodynamic Monitoring

Last updated : 19/11/2025 - 27414 views



Hemorrhagic Shock

Nicholas Hooper; Tyler J. Armstrong.

Author Information

Last Update: July 13, 2021.

Continuing Education Activity

Go to:

https://www.ncbi.nlm.nih.gov/books/NBK470382/

[Patient Blood Management Guidelines | National Blood Authority]

NATIONAL BLOOD AUTHORITY AUSTRALIA

Blood Products | Supply System | Clinical Guidance | Data and Research | Patient Information

Patient Blood Management Guidelines

NZBLOOD Te Ratonga Toto O Aotearoa

- https://onlinelibrary.wiley.com/doi/full/10.1111/bjh.16776

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Guideline | Open Access

Guidelines on the laboratory aspects of assays used in haemostasis and thrombosis

Peter Baker, Sean Platton, Claire Gibson, Elaine Gray, Ian Jennings, Paul Murphy, Mike Laffan, On behalf of British Society for Haematology, Haemostasis and Thrombosis Task Force

First published: 14 June 2020 | https://doi.org/10.1111/bjh.16776 | Citations: 3

THANZ
Thrombosis & Haemostasis society of Australia and New Zealand

https://www.thanz.org.au/resources/thanz-guidelines

Position statement

Updated recommendations for warfarin reversal in the setting of four-factor prothrombin complex concentrate

Danielle Robinson[1], James McFadyen[2], Eileen Merriman[3], Tan Chee Wee[4], Ross Baker[5], Huyen Tran[1,2]

doi:10.1111/imj.70103

POSITION PAPER

2025 Guidelines for direct oral anticoagulants: a practical guidance on the prescription, laboratory testing, peri-operative and bleeding management

Huyen A. Tran [1,2] Eileen Merriman [3] Ross Baker,[4] Jennifer Curnow,[5] Laura Young,[6] Chee Wee Tan,[7] Simon McRae[8] and Sanjeev D. Chunilal[9]

[1]Haemostasis Thrombosis Unit, The Alfred Hospital, [2]Australian Centre for Blood Diseases, Monash University, and [9]Haematology Department, Monash Medical Centre, Melbourne, Victoria, [4]Haematology Department, Perth Blood Institute, Perth, Western Australia, [5]Haematology Department, Westmead Hospital, Sydney, New South Wales, [7]Haematology Department, Royal Adelaide Hospital, Adelaide, South Australia, [8]Department of Haematology, Launceston General Hospital, Launceston, Tasmania, Australia, and [3]Haematology Department, Royal North Shore Hospital, and [6]Haematology Department, Auckland City Hospital, Auckland, New Zealand

Key words
direct oral anticoagulant, major bleeding, reversal agent, perioperative anticoagulation.

Correspondence
Huyen A. Tran, Australian Centre for Blood Diseases, Monash University, Melbourne, Vic. 3800, Australia.
Email: huyen.tran@monash.edu

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Abstract

Direct oral anticoagulants (DOACs) are widely prescribed to prevent and treat venous and arterial thromboembolism, supported by published evidence, and are preferred over warfarin in many guidelines. Although the risk of major bleeding, in particular intracranial haemorrhage (ICH), is decreased with DOACs, gastrointestinal bleeding is increased with some DOACs, and the case fatality rate of bleeding remains high. Therefore, it is important to (i) prescribe DOACs appropriately, (ii) have strategies to manage major bleeding including the use of specific reversal agents and (iii) interrupt and resume DOACs for procedures. The main recommendations are as follows: (i) Select the appropriate dose of DOAC according to indications and consider patient factors to minimise bleeding risks; (ii) DOACs do not require routine laboratory testing; (iii) for life-threatening uncontrollable bleeding, specific agents

2025 Guidelines for direct oral anticoagulants: a practical guidance on the prescription, laboratory testing, peri-operative and bleeding management

BSH, 2023

The British Society of Interventional Radiology

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IR PROCEDURE BLEEDING RISK GUIDANCE

PRE-ASSESSMENT SCREENING

All patients, not on anti-thrombotic therapy, can be initially assessed using the HEMSTOP questionnaire below (each question scores 1 for yes):

- Have you ever consulted a doctor or received treatment for prolonged or unusual bleeding (such as nosebleeds, minor wounds)?
- Do you experience bruises/haematomas larger than 2 cm without trauma or severe bruising after minor trauma?
- After a tooth extraction, have you ever experienced prolonged bleeding requiring medical/dental consultation?
- Have you experienced excessive bleeding during or after surgery?
- Is there anyone in your family who suffers from a bleeding disorder (such as haemophilia or von Willebrand disease)?
- Have you ever consulted a doctor or received treatment for heavy or prolonged menstrual periods (contraceptive pill, iron etc.)?
- Did you experience prolonged or excessive bleeding after delivery?

If < 2 positive responses:	**LOW RISK PROCEDURES:** No coagulation screen or FBC required **MODERATE/HIGH RISK PROCEDURES:** No coagulation screening required; FBC only
If ≥ 2 positive responses:	Perform coagulation screen (FBC, PT, APTT, Clauss fibrinogen assay) and discuss with haematologist prior to procedure

BLEEDING RISK STRATIFICATION FOR COMMON IR PROCEDURES

LOW RISK INTERVENTIONS	MODERATE RISK INTERVENTIONS	HIGH RISK INTERVENTIONS
Basic venous interventions (IVC filter insert/removal)	Arterial interventions (≤ 6F)	Arterial interventions (≥ 7F)
Superficial interventions/ biopsies (excluding liver/renal)	Embolisation (TACE/UAE/PAE)	Aortic stent grafting
GI tract stenting	Venous/dialysis access interventions	Tumour ablation
MSK interventions	Tunnel line insertions	PCNL/renal biopsy/nephrostomy
US guided drainages		TIPSS/TJ liver biopsy
Catheter exchange/removal		Liver biopsy/biliary intervention

PRE-PROCEDURAL BLOOD PARAMETERS REQUIREMENTS

LOW RISK INTERVENTIONS	MODERATE RISK INTERVENTIONS	HIGH RISK INTERVENTIONS
No procedure specific laboratory tests	Hb: > 70 g/L Plts: > 50 x 10^9/L If on vit K antagonist INR: < 2.0	Hb: > 70 g/L Plts: > 50 x 10^9/L If on vit K antagonist INR: < 1.5

LIVER DISEASE*

Consider correction if: Fibrinogen: < 1.2 g/L Plts: < 50 x 10^9/L Haematocrit < 25%

* Neither PT nor INR correlate well with bleeding risk in patients with liver disease

PRE-PROCEDURAL ANTI-THROMBOTIC MEDICATION INSTRUCTIONS*

***CONSIDERATIONS:**
1. Cardiac stents and stroke or thrombosis within 3 months: consult appropriate clinical team
2. Patients on dual antiplatelet therapy, ticagrelor or prasugrel: follow local Trust policy or consult appropriate specialist
3. Follow local Trust policy for referral to bridging clinic
4. Bleeding and thrombosis risks should be discussed as part of the consent process

HEPARINS: Low Risk Procedures

	Hold duration prior to procedure	Suggest restart time following procedure
Unfractionated Heparin	2-4 h	6 h
LMWH (prophylactic)	12 h	6-12 h
LMWH (therapeutic)	1 day	6-12 h

HEPARINS: Moderate/High Risk Procedures

	Hold duration prior to procedure	Suggest restart time following procedure
Unfractionated Heparin	4 h	12-48 h
LMWH (prophylactic)	12 h	1 day
LMWH (therapeutic)	1 day	1-3 days

Vitamin K Antagonists: Low Risk Procedures | INR < 2.0 on day of procedure

	Hold duration prior to procedure	Suggest restart time following procedure
Warfarin/Acenocoumarol	2-3 days	Evening

Vitamin K Antagonists: Moderate/High Risk Procedures | INR < 1.5 on day of procedure

	Hold duration prior to procedure	Suggest restart time following procedure
Warfarin/Acenocoumarol	5 days	12-24 h

Thrombin Inhibitors: Low Risk Procedures (as per PAUSE protocol)

	Hold duration prior to procedure	Suggest restart time following procedure
Dabigatran	1 day if eGFR > 50 2 days if eGFR < 50	1 day
Argatroban	2-4 h	6 h

Thrombin Inhibitors: Moderate/High Risk Procedures (as per PAUSE protocol)

	Hold duration prior to procedure	Suggest restart time following procedure
Dabigatran	2 days if eGFR > 50 4 days if eGFR < 50	2-3 days
Argatroban	4 h	6 h

Factor Xa Inhibitors: Low Risk Procedures (as per PAUSE protocol)

	Hold duration prior to procedure	Suggest restart time following procedure
Apixaban/Rivaroxaban/Endoxaban	Omit 1 day prior	Restart after 1 day
Fondaparinux (prophylactic)	1 day	6 h
Fondaparinux (therapeutic)	2 days	6 h

Factor Xa Inhibitors: Moderate/High Risk Procedures (as per PAUSE protocol)

	Hold duration prior to procedure	Suggest restart time following procedure
Apixaban/Rivaroxaban/Endoxaban	Omit 2 days prior	Restart after 2-3 days
Fondaparinux (prophylactic)	1 day	12-24 h
Fondaparinux (therapeutic)	2 days	12-24 h

Aspirin & ADP Receptor Inhibitors: Low Risk Procedures

	Hold duration prior to procedure	Suggest restart time following procedure
Aspirin/Clopidogrel/Ticagrelor/Prasugrel	Does not need to be stopped	N/A

Aspirin & ADP Receptor Inhibitors: Moderate/High Risk Procedures

	Hold duration prior to procedure	Suggest restart time following procedure
Aspirin (low dose monotherapy)	Does not need to be stopped	N/A
Clopidogrel	VASCULAR: Operators discretion NON-VASCULAR: 7 days	VASCULAR: Operators discretion NON-VASCULAR: 1 day
Ticagrelor/Prasugrel	7 days	1 day

Authors: Clare Bent and Raj Das on behalf of the BSIR Safety and Quality Committee, Keith Gomez and Will Lester on behalf of the BSH Haemostasis and Thrombosis Task Force.
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NZ_National_Guidelines_for_the_Management_of_Haemophilia.pdf