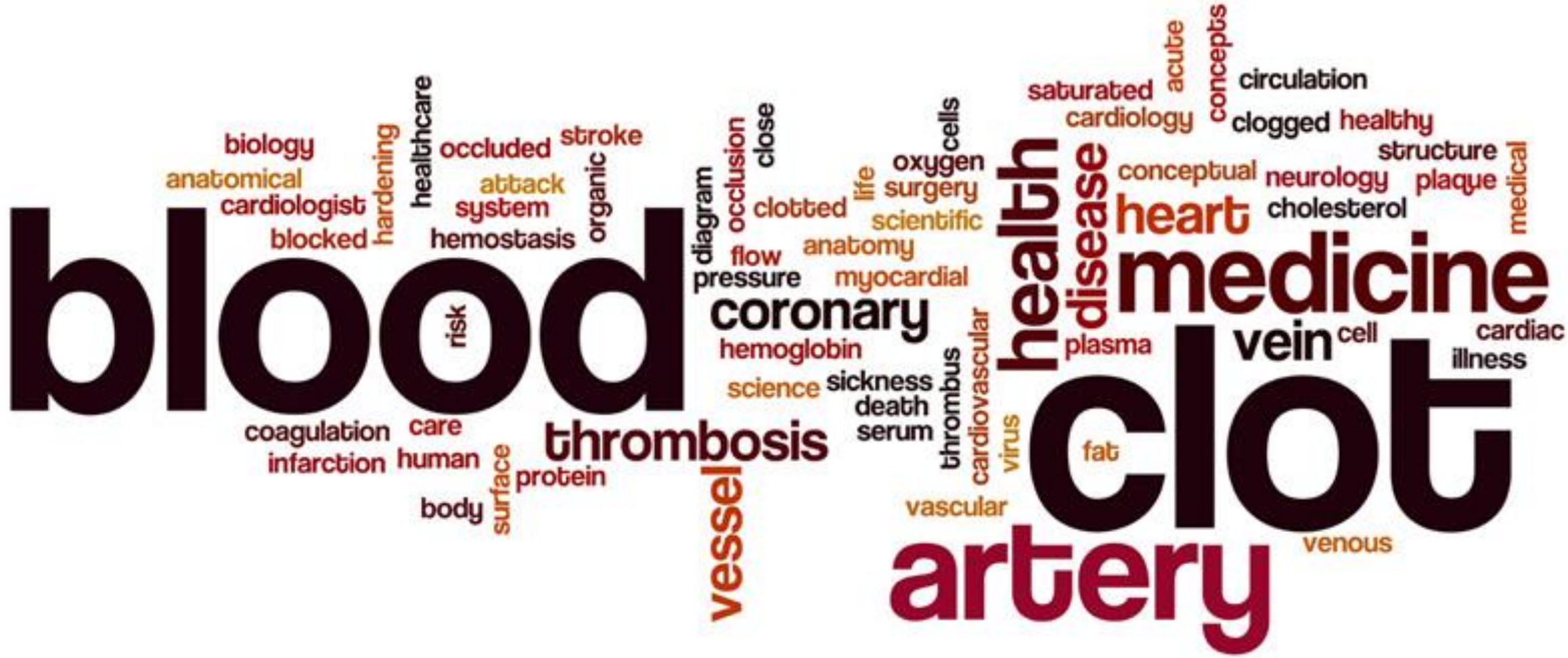
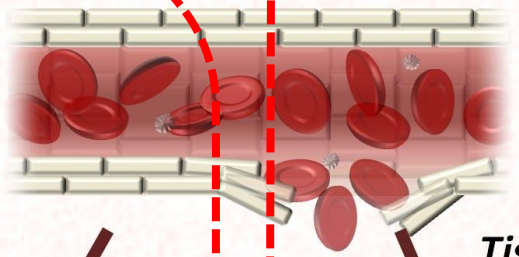
PERIOPERATIVE COAGULATION MONITORING

R2 Thanitthi Thiparporn
R2 Ajana Trisukhon

Supervisor Aj.Wiriya Maisat


blood
clot
artery
medicine
heart
health
disease
vein
thrombosis
coronary
vessel
risk
coagulation
infarction
care
human
body
surface
protein
thrombus
cardiovascular
virus
vascular
fat
plasma
cell
cardiac
illness
sickness
death
serum
science
hemoglobin
myocardial
pressure
flow
anatomy
diagram
occlusion
close
clotted
life
surgery
scientific
oxygen
cells
saturated
cardiology
acute
concepts
circulation
clogged
healthy
structure
conceptual
neurology
plaque
medical
cholesterol
venous
biology
anatomical
cardiologist
blocked
hardening
healthcare
occluded
attack
system
hemostasis
stroke
organic

Major Components of Hemostasis



Vascular Injury

Collagen

Tissue factor

Vasoconstriction

Platelet Activation

Coagulation Cascade

Antithrombotic Control Mechanisms

vWF, fibrinogen

Thrombin

Platelet Plug

Fibrinogen → *Fibrin*

Blood Clot

Plasmin

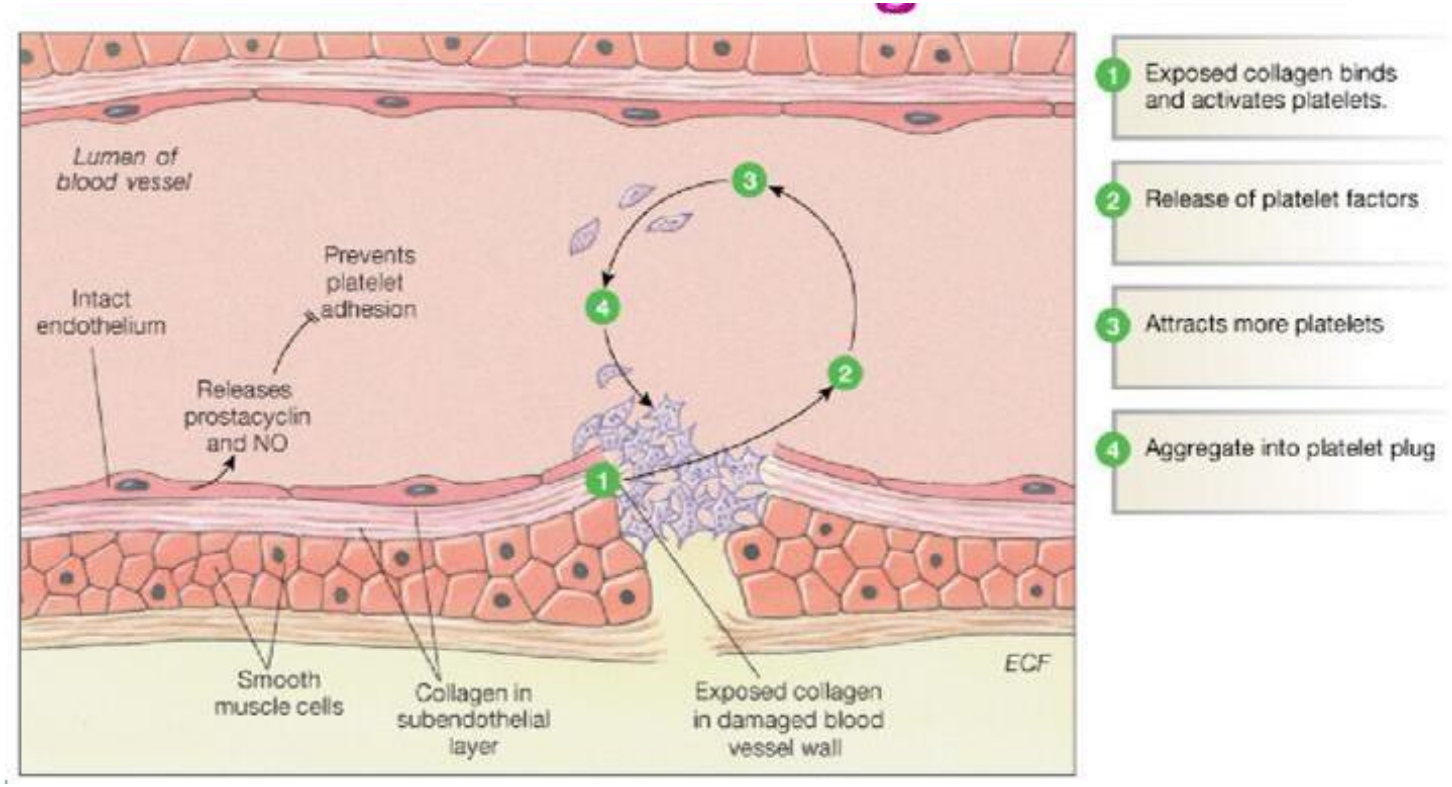
Fibrinolysis & Clot Degradation

Primary Hemostasis

Secondary Hemostasis

Primary hemostasis

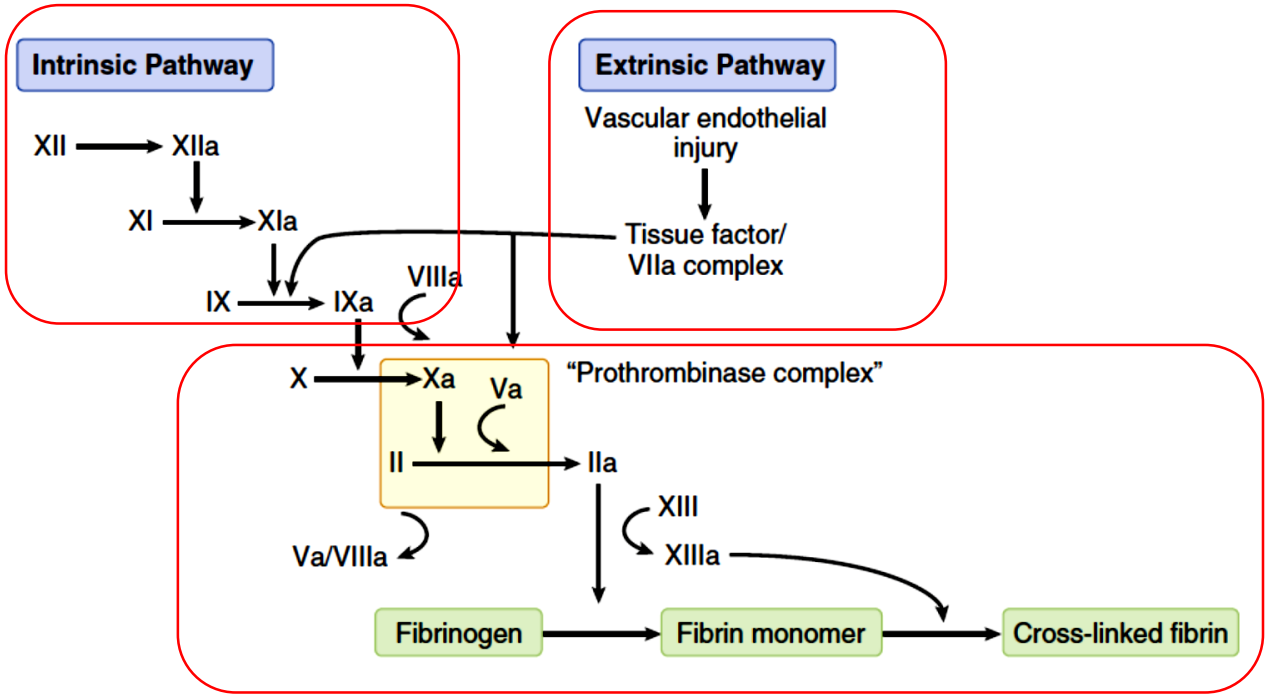
- **Vascular endothelium**
  - Vasoconstriction : local tissue factor , nervous system
- **Platelet Plug**
  - Platelet Adhesion
  - Platelet Activation
  - Platelet Aggregation
  - Platelet Plug Formation



Secondary Hemostasis

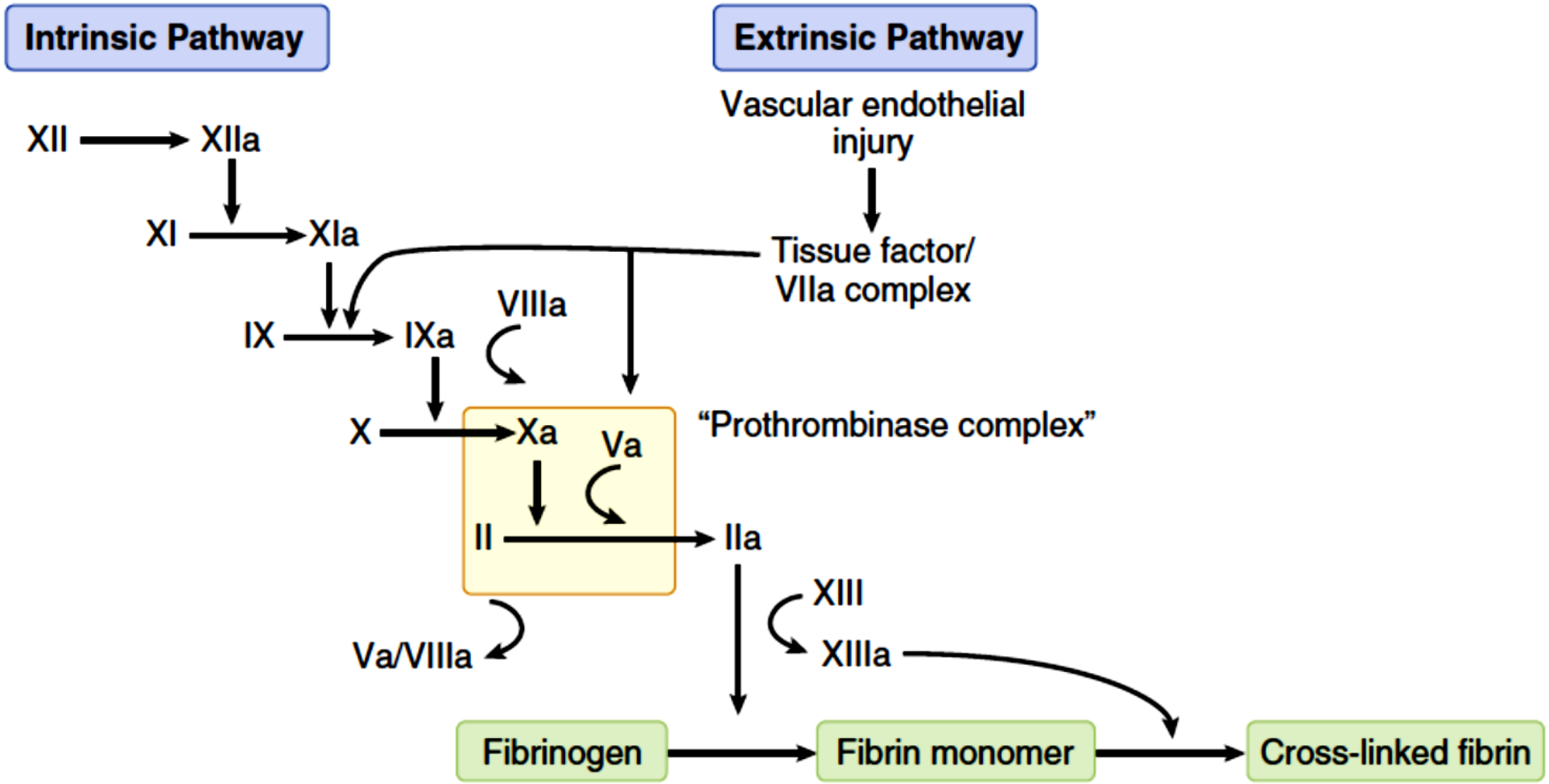
Cascade / Waterfall Model

- **Activation of coagulation system**
  - Extrinsic pathway
  - Intrinsic pathway
  - Common pathway



Secondary Hemostasis

Cascade / Waterfall Model



Secondary Hemostasis

Review Article

A Cell-based Model of Hemostasis

Maureane Hoffman, Dougald M. Monroe III

Pathology and Laboratory Medicine Service, Durham VA and Duke University Medical Centers, Durham, NC, USA, and Division of Hematology/Oncology, Department of Medicine, The University of North Carolina, Chapel Hill, NC, USA

Secondary Hemostasis

- Current concept : ***Cell-based model of hemostasis***
- *Hoffman and Monroe 2001*

- Hemostatic process 3 phases
  - Initiation
  - Amplification
  - Propagation (thrombin burst)

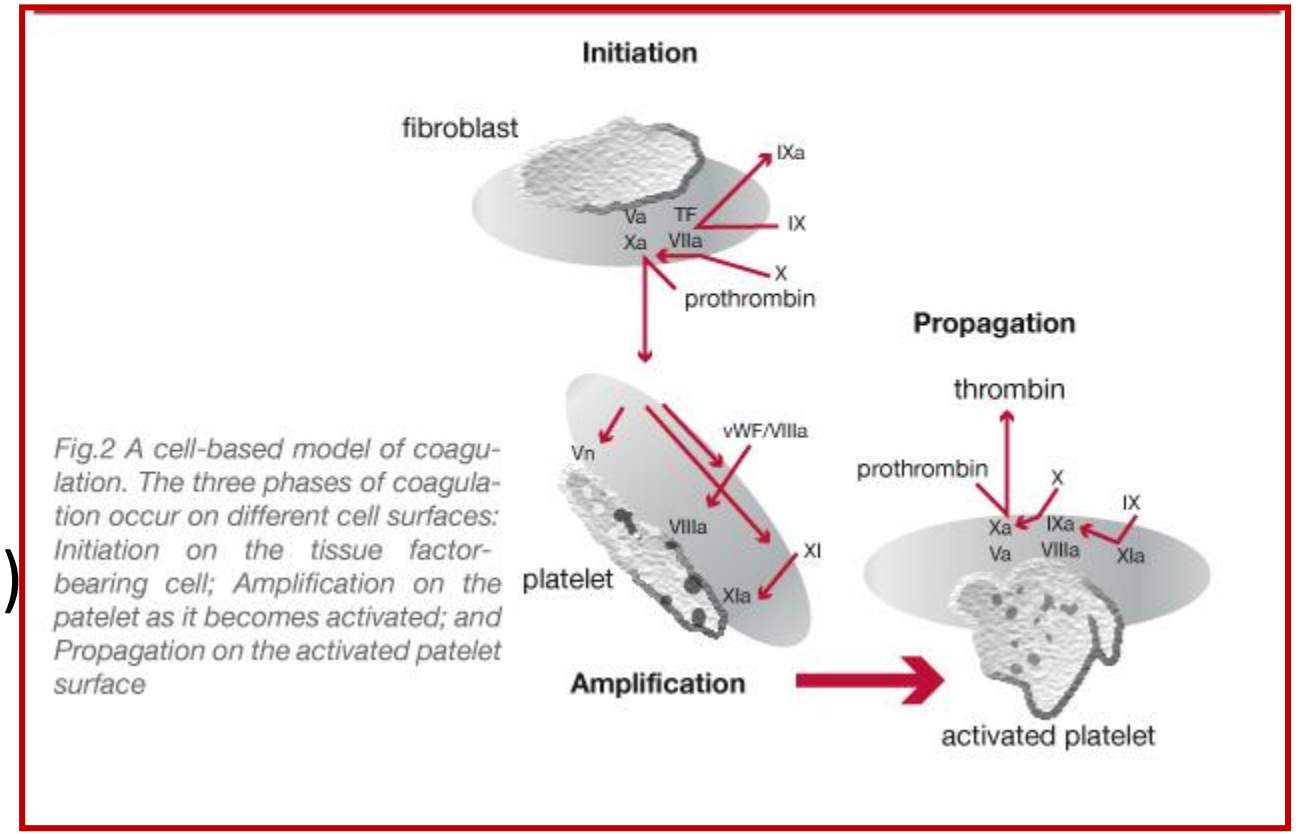
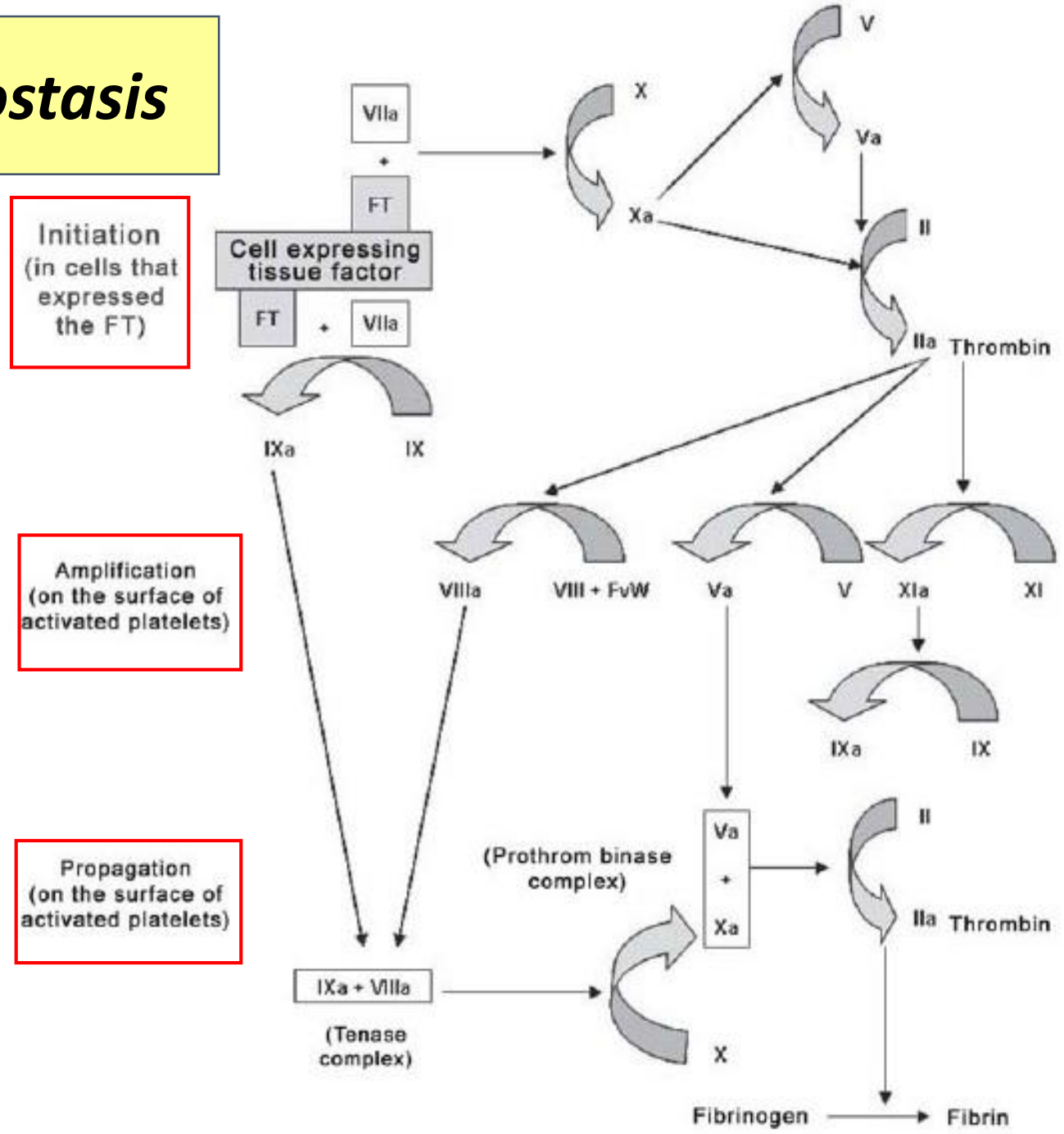
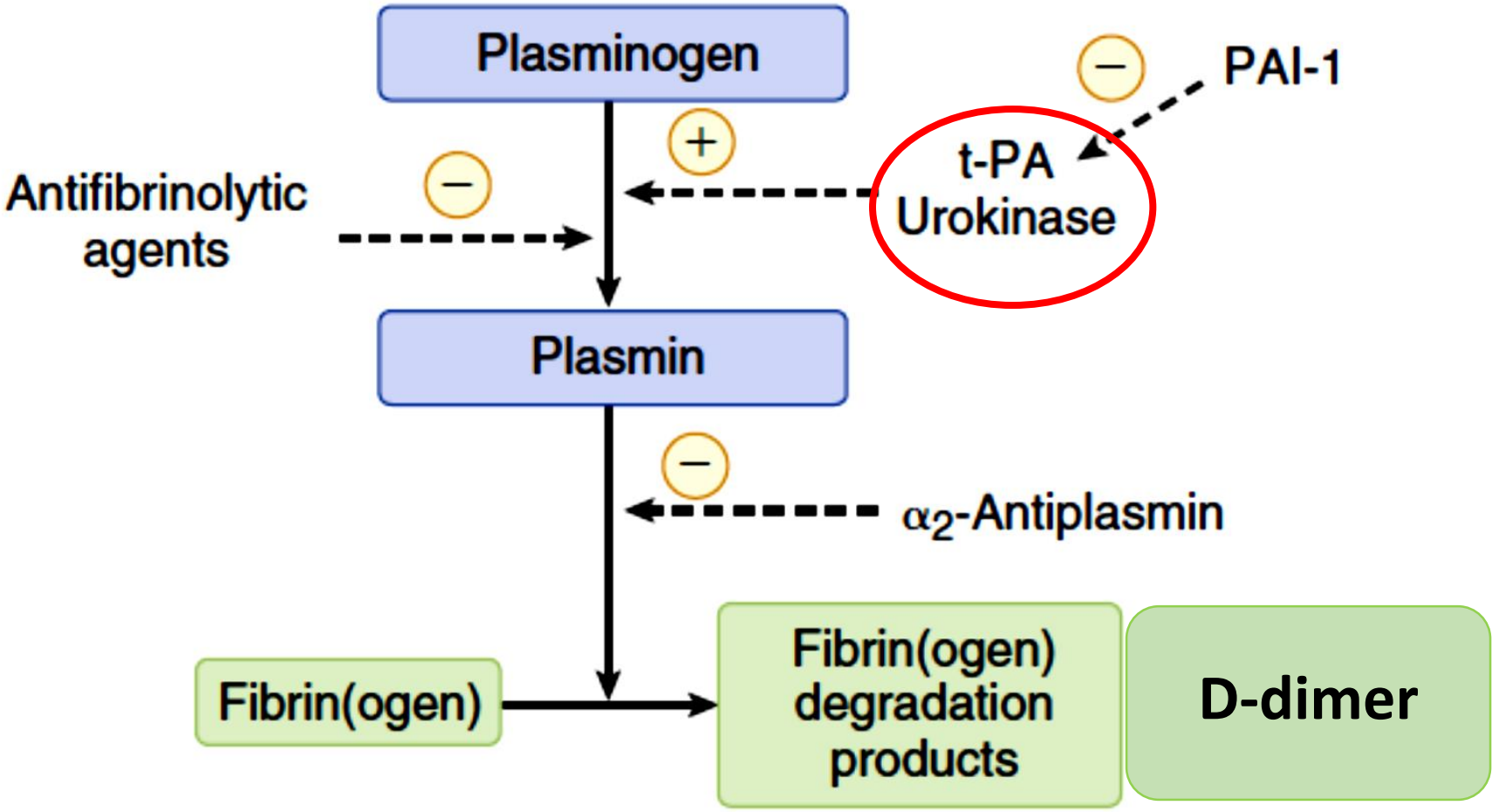


Fig.2 A cell-based model of coagulation. The three phases of coagulation occur on different cell surfaces: Initiation on the tissue factor-bearing cell; Amplification on the patelet as it becomes activated; and Propagation on the activated patelet surface

Cell-based model of hemostasis



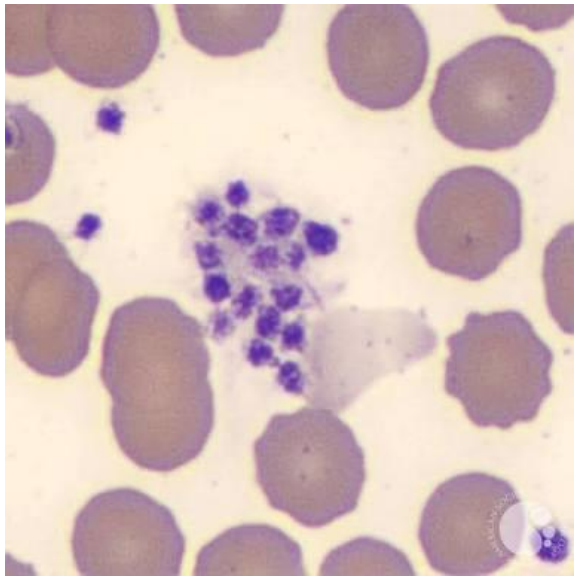
Fibrinolytic Pathway



PERIOPERATIVE
Coagulation monitoring

Platelet count

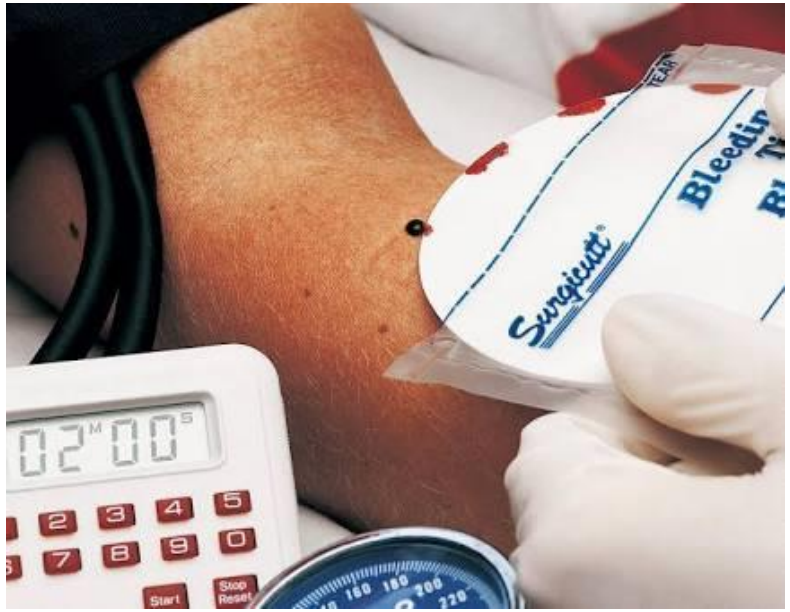
- Primary hemostasis
- Reflect quantitative of platelet
- Normal range : 150,000-450,000
- Turn around time : 1 hour



Bleeding time

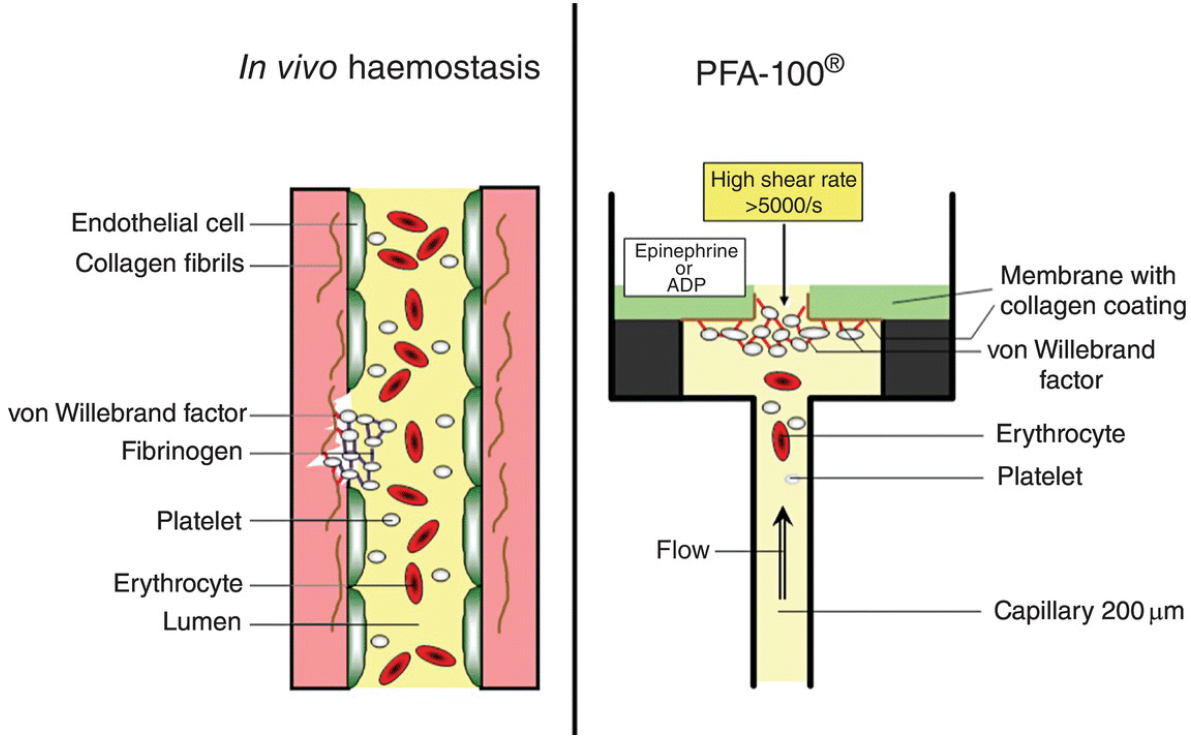
Out of Use

- Assess ***platelet function***
- Making a puncture and monitoring time for bleeding stop
- Normal: 2-10 minutes at anterior forearm
- Delicate , experienced operator
- Prolongation:
  - thrombocytopenia
  - hypofibrinogenemia
  - severe anemia(Hct<30%)
  - vWD



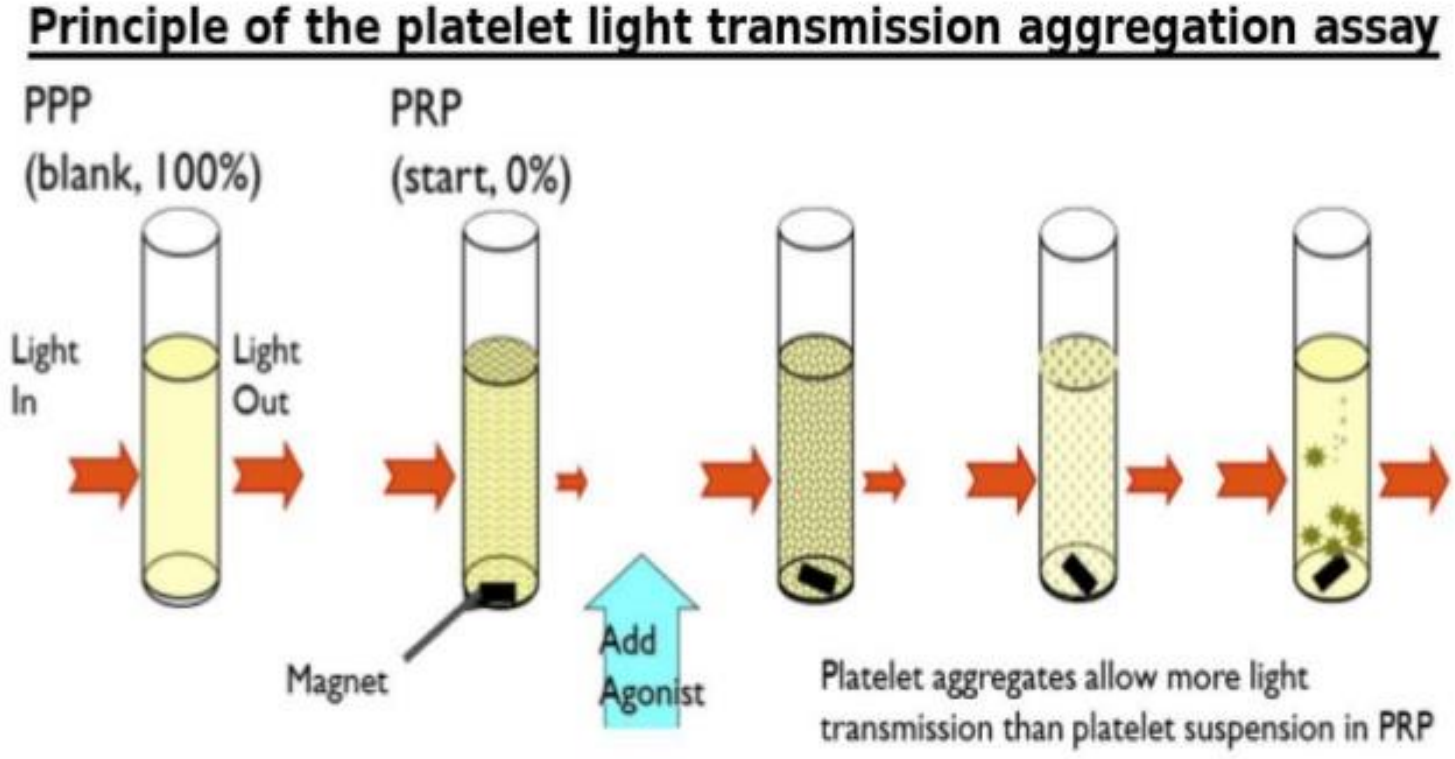
Platelet function analyzer

- PFA-100 , PFA-200 , Siemens
- Congenital and Acquired platelet dysfunction



Light transmission platelet aggregometry

- Platelet aggregation assays
- Congenital and Acquired qualitative platelet disorder



Activated Partial Thromboplastin Time (aPTT)

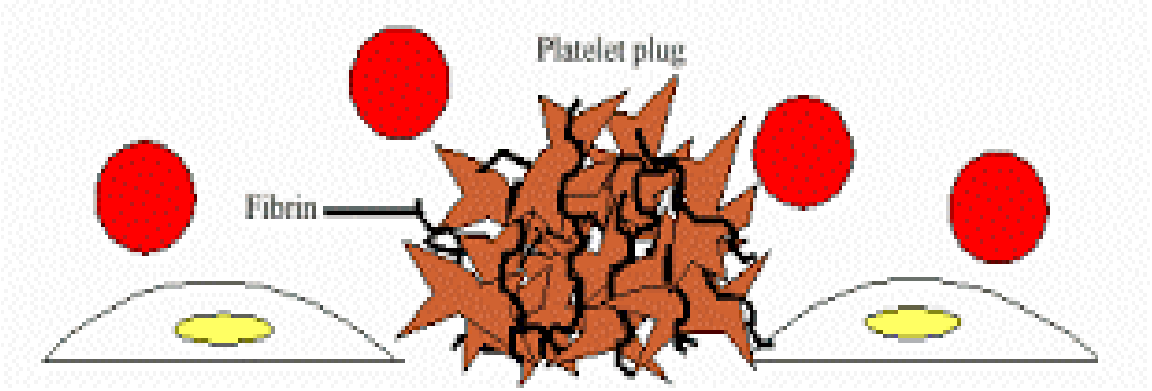
- Intrinsic & Common pathway
- Activate intrinsic pathway by celite, kaolin, silica
- Detection at factor concentration below 30%-40% of normal
- Turn around time : 90 minutes

Prothrombin Time (PT)

- Extrinsic & Common pathway
- Activate intrinsic pathway by Ca&tissue thromboplastin
- Turn around time : 90 minutes

Fibrinogen level

- **Fibrinogen is converted into fibrin** to stabilize clot
- Normal value : 200-400 mg/dL
- Turn around time : 90 mins



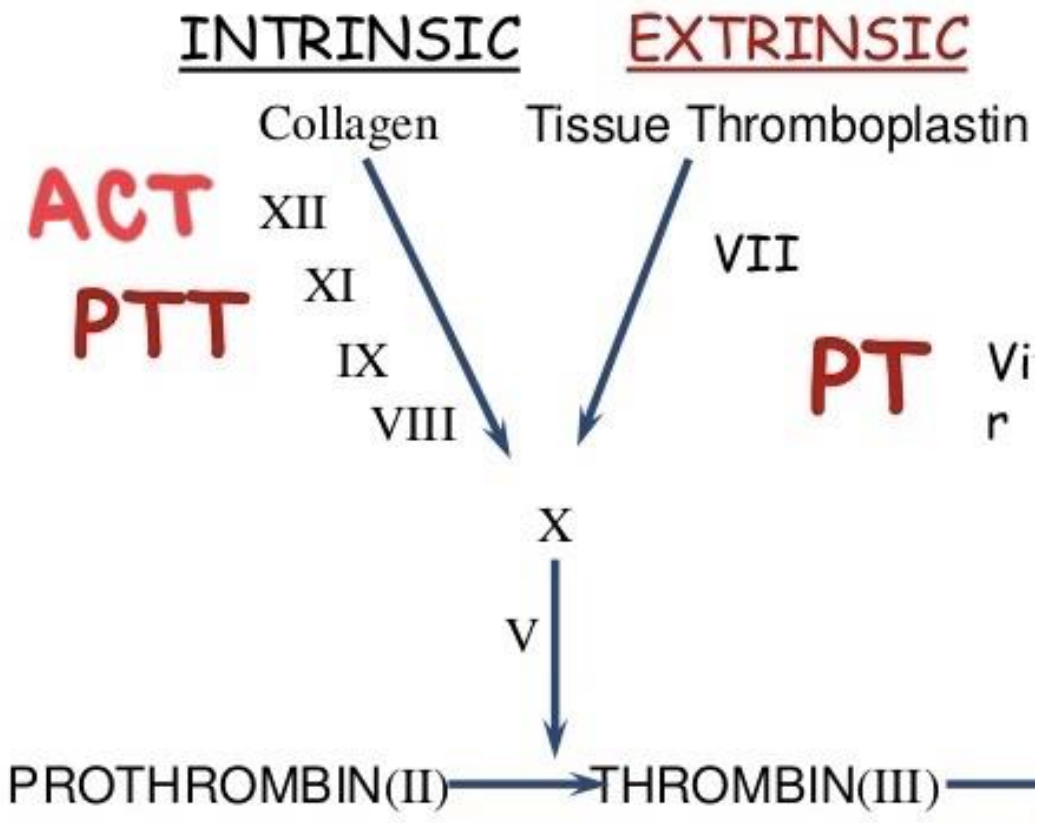
POINT-OF-CARE COAGULATION TEST

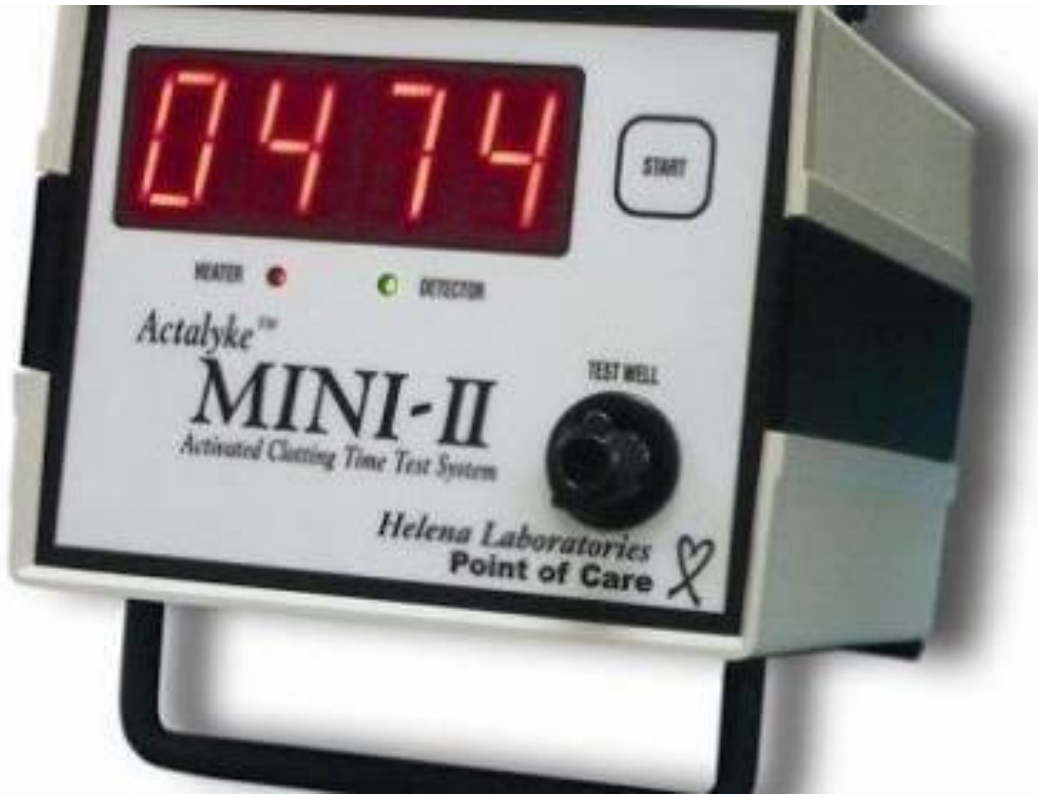
POINT OF CARE COAGULATION TEST

- ACT
- TEG
- ROTEM

Activated Clotting Time

- Activation of coagulation via the *intrinsic [Factor XII] pathway*
- Monitor the anticoagulant effect of heparin
- Clinical application
  - Cardiopulmonary bypass surgery
  - ECMO support
  - Catheterization laboratory
  - Angiography intervention




0474
START
HEATER
DETECTION
Actalyke™
MINI-II
Activated Clotting Time Test System
TEST WELL
Helena Laboratories
Point of Care


Magnets


ACT Plus™
BIOHAZARD
1
Clot Time
2
Main Menu
QC
Medtronic
Stop Key
Cancel Key
Enter Key
Clear Key

Activated Clotting Time

- Limitation

  Lack of sensitivity at low heparin concentration

TABLE 67-3 CLINICAL VARIABLES THAT CAN AFFECT THE ACTIVATED CLOTTING TIME

Hemodilution	Prolong ACT
Hypothermia	
Thrombocytopenia	
Platelet inhibitors	
Platelet lysis	Shorten ACT

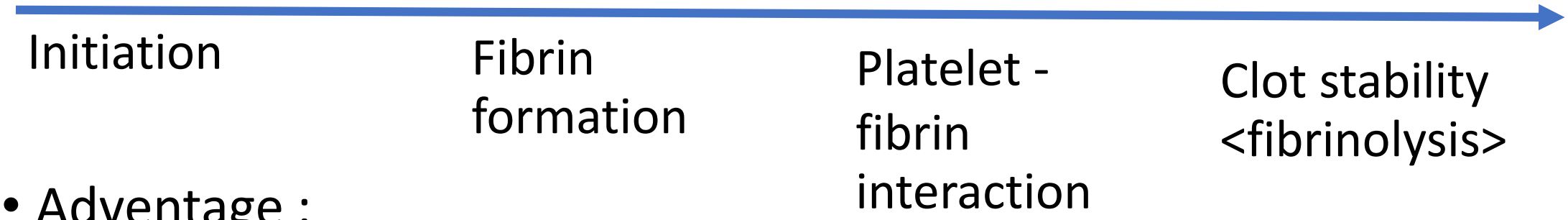
Chapter 62 patient blood management Miller's anesthesia 8th edition

- 67-year-old woman was diagnosed severe AR from IE s/p MVR AVR on warfarrin
- Preoperative lab : INR 1.7
- Operation Redo AVR
- Post CPB:
  - Bleeding from surgical field despite 2 rounds of blood components

ThromboElastoGraphy

ROtational ThromboElastoMetry

- Examines entire of hemostasis.



- Adventage :
  - Real-time analyse of clot formation and dissolusion
  - Use whole blood & Fast turnaround
  - Guide for specific transfusion → Reduce blood product transfusions

American Journal of Hematology, Vol. 89, No. 2, February 2014 doi:10.1002/ajh.23599

Fahrendorff *et al. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine* (2017) 25:39
DOI 10.1186/s13049-017-0378-9

Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine

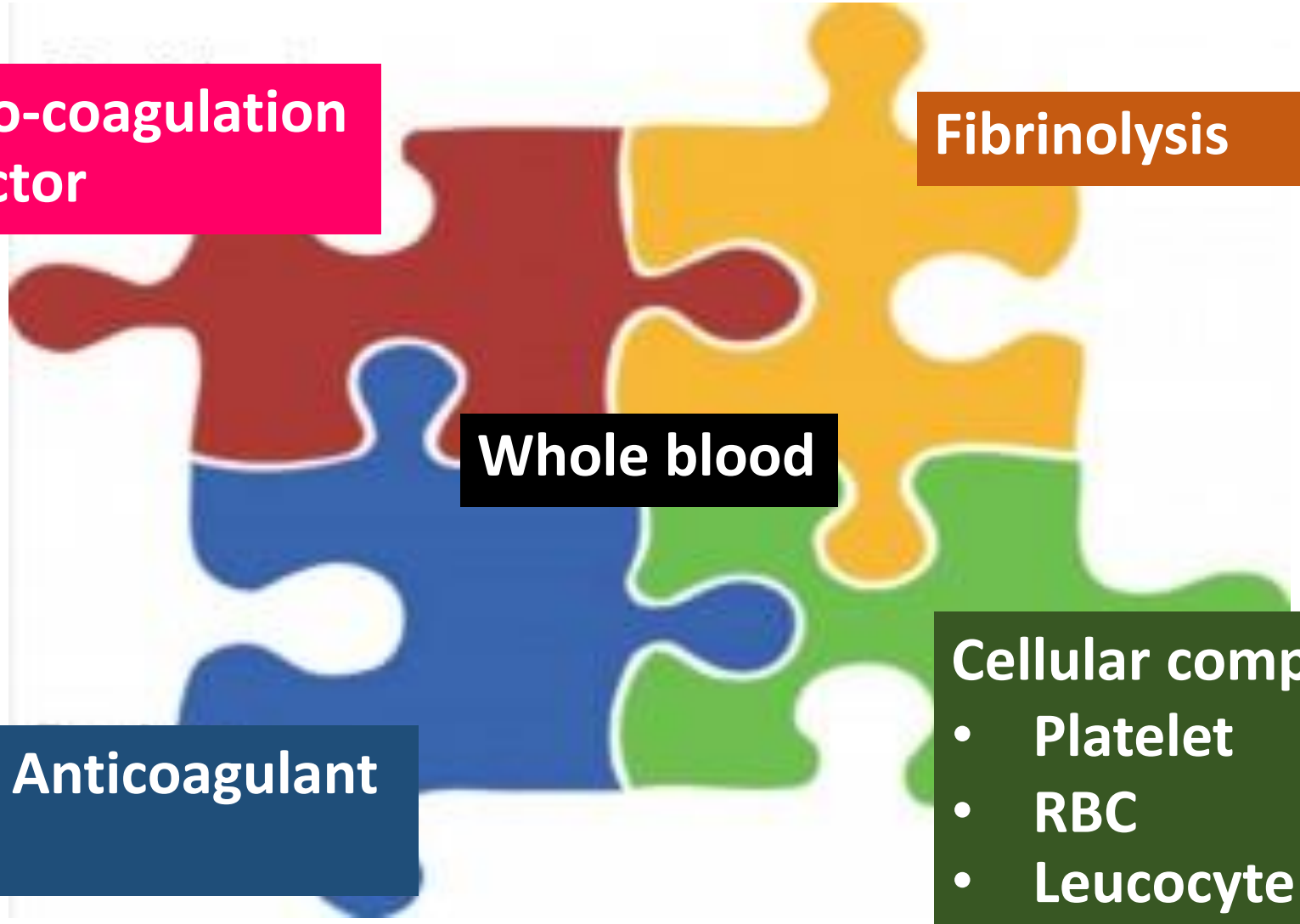
REVIEW Open Access

The use of viscoelastic haemostatic assays in goal-directing treatment with allogeneic blood products – A systematic review and meta-analysis

Mathilde Fahrendorff[1*], Roberto S. Oliveri[1] and Pär I. Johansson[1,2,3]

- The amount of transfused RBC FFP and bleeding volume was found to be significant reduce in VHA-guide group

Hemostasis



Pro-coagulation factor

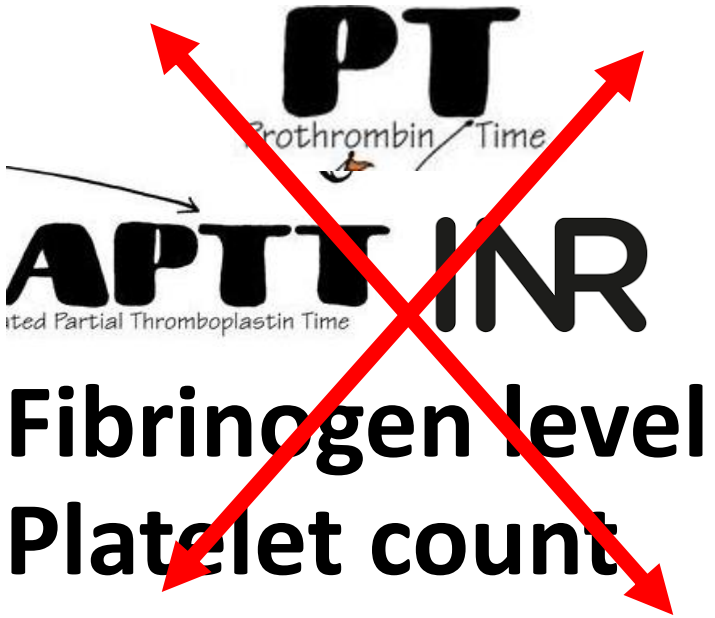
Fibrinolysis

Whole blood

Anticoagulant

Cellular component

- **Platelet**
- **RBC**
- **Leucocyte**



Fibrinogen level

Platelet count

TEG®

Confident care **starts** with the **complete picture**

Thromboelastography <TEG>

- CLINICAL USES
  - Cardiac surgery
  - Liver transplantation
  - Major trauma
  - Major obstetric hemorrhage.

American Journal of Hematology, Vol. 89, No. 2, February 2014 doi:10.1002/ajh.23599

Thromboelastography <TEG>

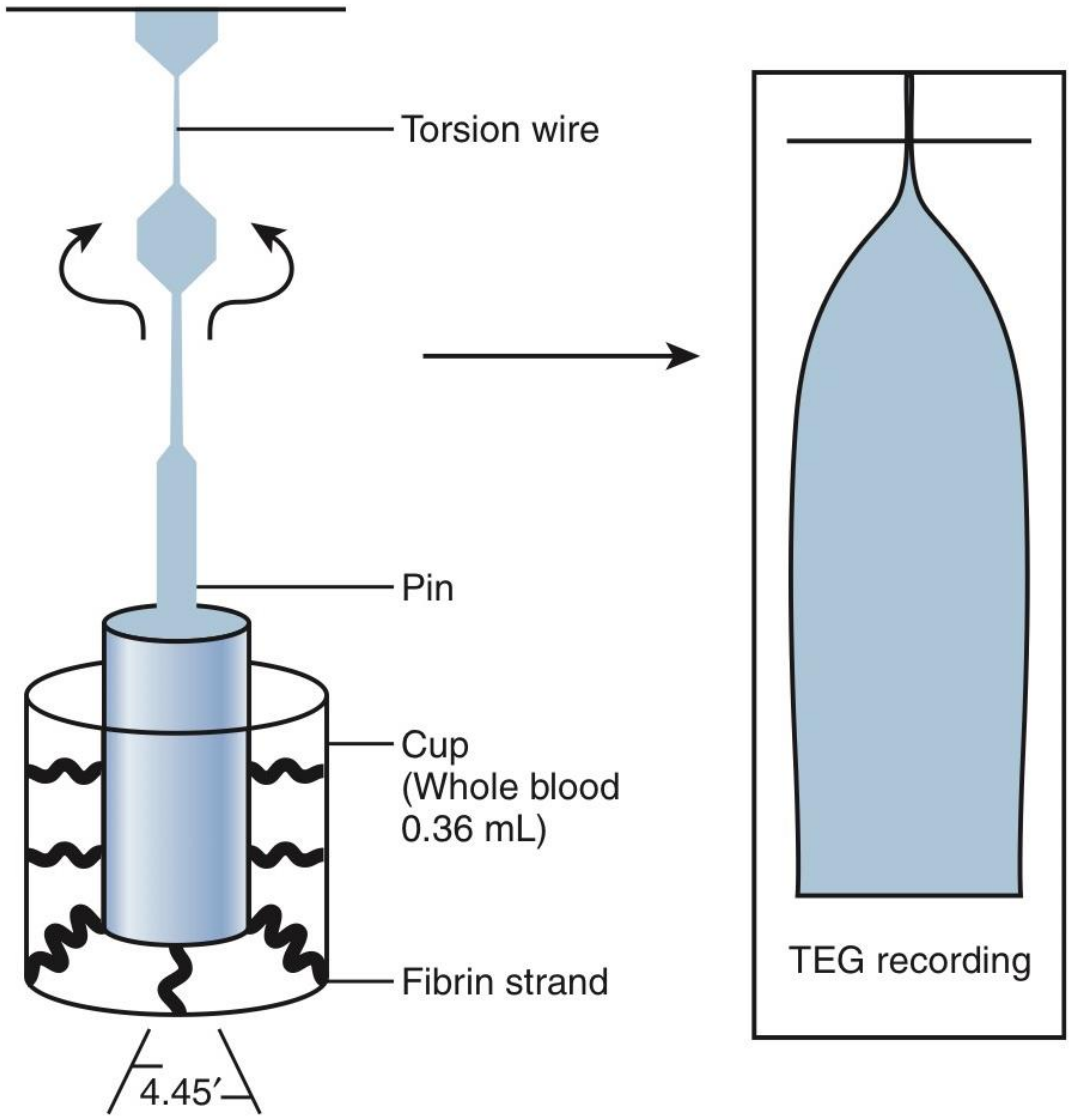
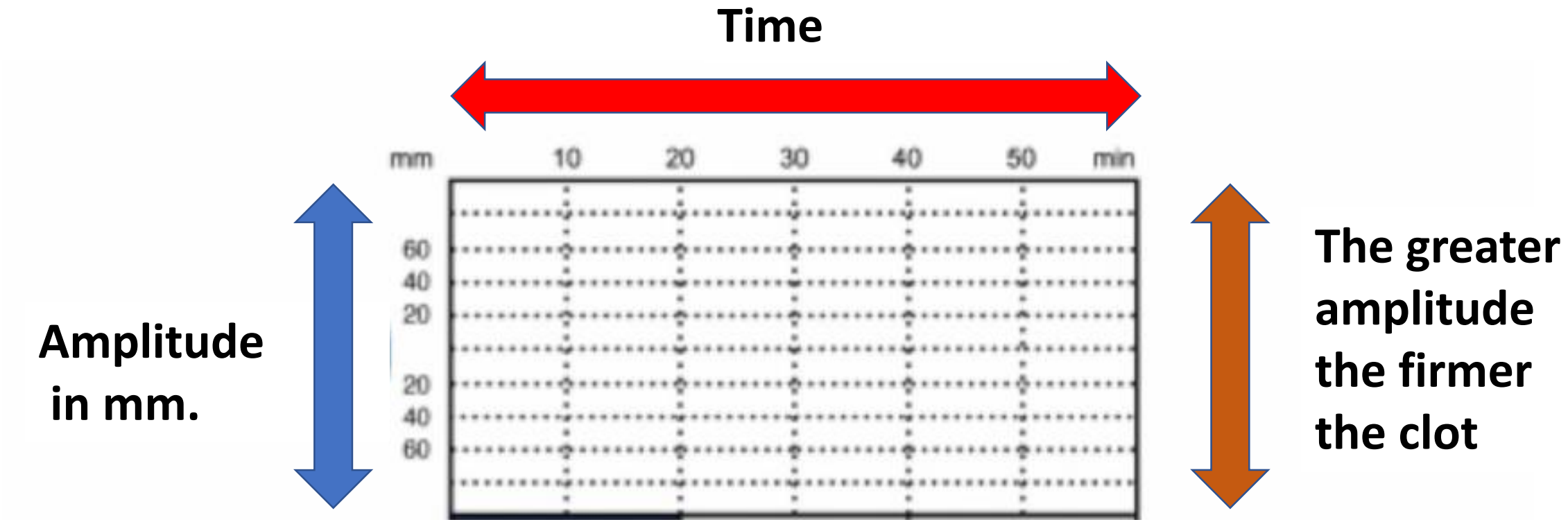


Figure 17-16 Schematic diagram of the thromboelastograph (TE instrumentation *(left)* and a sample tracing *(right).* A whole-bloc sample is placed into the cup into which a plastic pin is suspende This plastic pin is attached to a torsion wire that is coupled to an amp fier and recorder. *(From Mallett SV, Cox DJA: Thromboelastograph* Br J Anaesth *69:307–313, 1992.)*

Kaplan's cardiac anesthesia 6th edition

TEMogram


Time
mm
10
20
30
40
50
min
60
40
20
20
40
60
Amplitude in mm.
The greater amplitude the firmer the clot

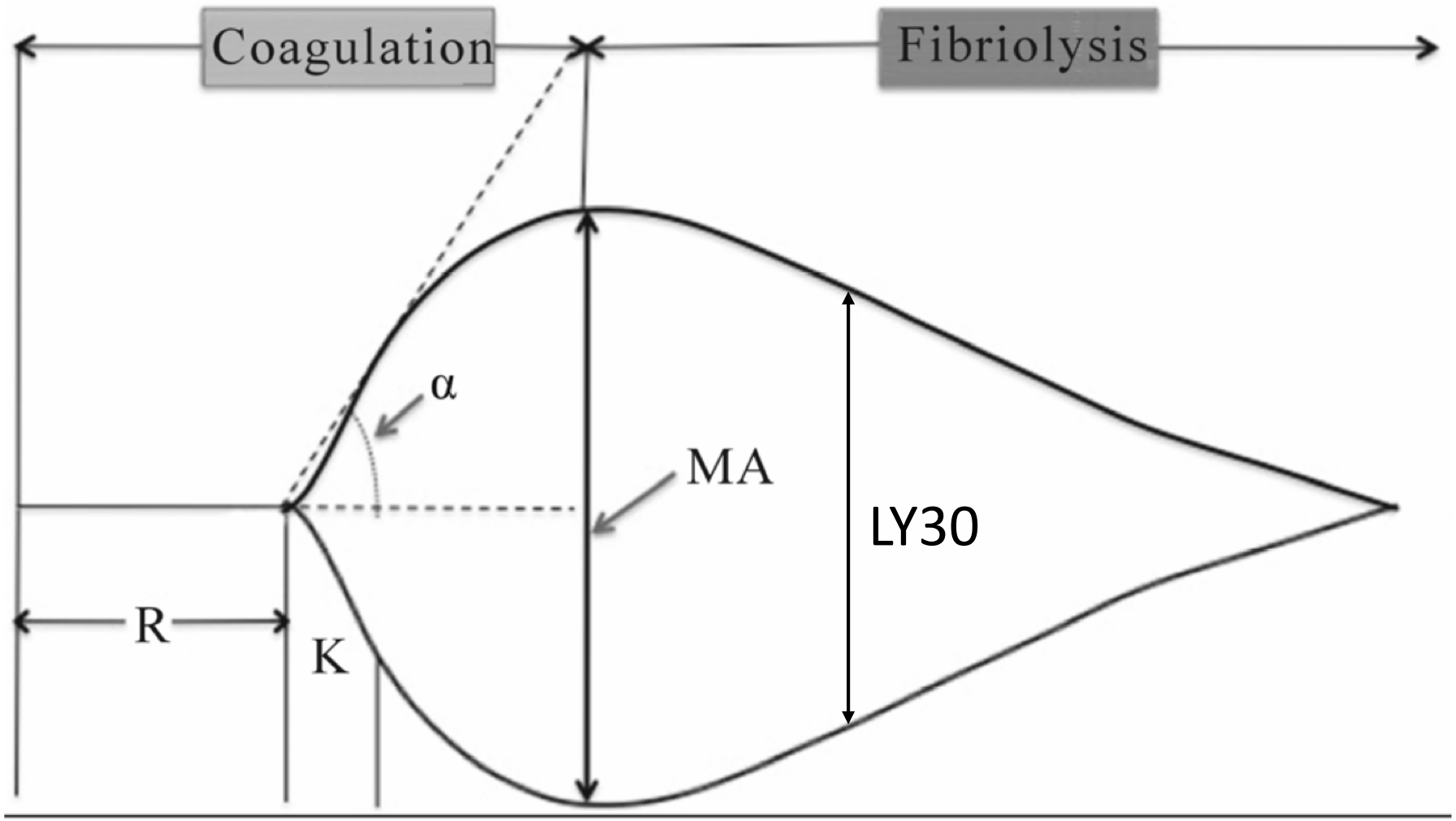
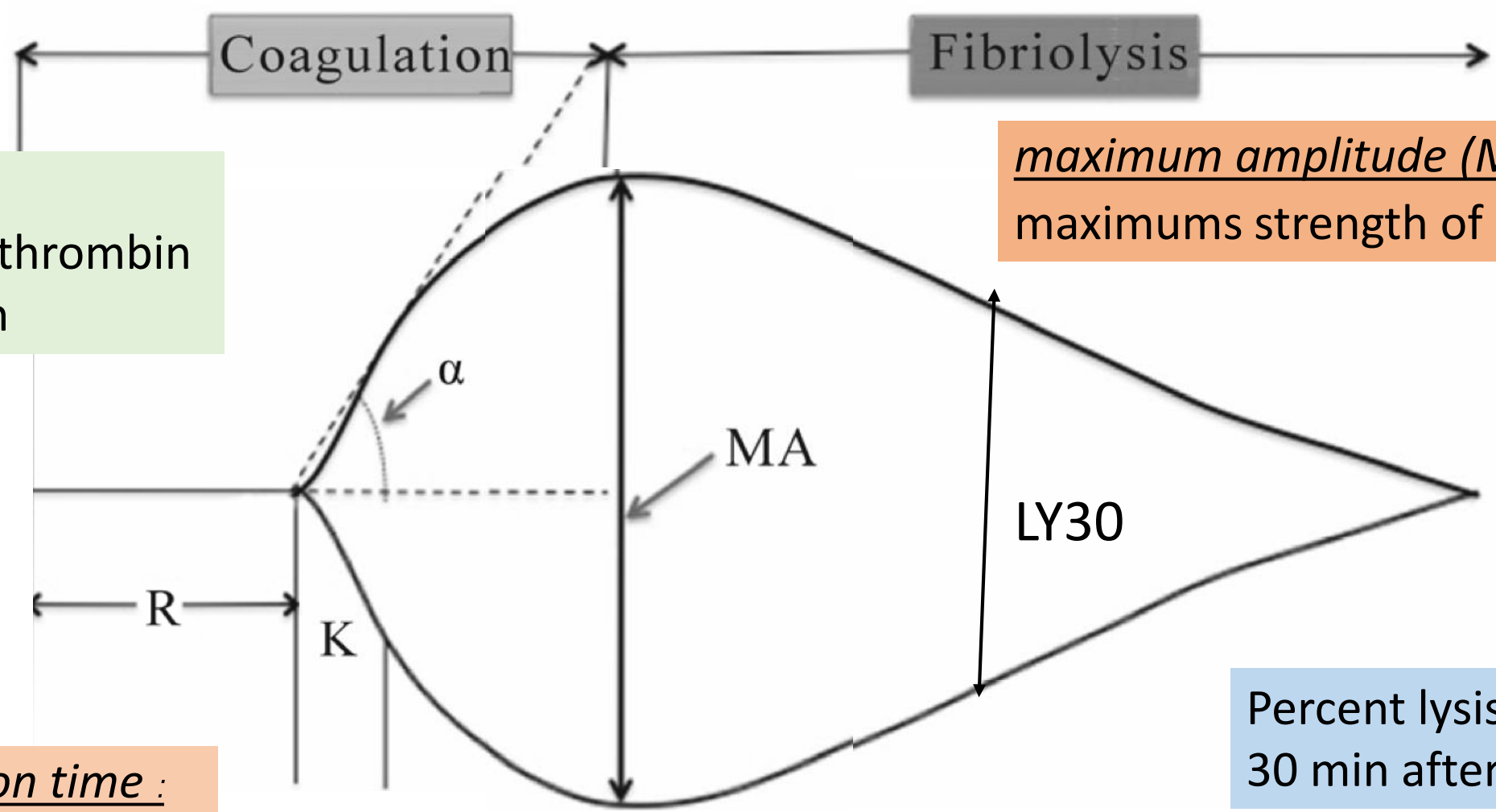


Figure 1. Normal TEG tracing, depicting rate of formation and degradation of clot, as well as the MA, R, *α* and K.



Angle:
Speed of thrombin formation

maximum amplitude (MA) :
maximums strength of clot

Reaction time :
1st significant clot formation

clot kinetics :
speed of clot formation

Percent lysis in 30 min after MA

Point-of-care coagulation testing
Continuing Education in Anaesthesia Critical Care & Pain, Volume 13, Issue 1, 1 February 2013, Pages 12–16

Thromboelastography <TEG>

- Initial interpretation

R time : Reflects coagulation factor level

K & α Angle : Reflects fibrinogen activity

MA. : Reflects platelets function and fibrinogen activity

LY 30 : Reflects clot stability or fibrinolysis

Point-of-care coagulation testing
Continuing Education in Anaesthesia Critical Care & Pain,
Volume 13, Issue 1, 1 February 2013, Pages 12–16

Table 2: Suggested TEG-guided transfusion

TEG Value	Transfuse
R time > 10	FFP
K time > 3	cryoprecipitate
α angle < 53	cryoprecipitate +/- platelets
MA < 50	platelets
LY30 > 3%	tranexamic acid

Point-of-care coagulation testing
Continuing Education in Anaesthesia Critical Care & Pain,
Volume 13, Issue 1, 1 February 2013, Pages 12–16

Normal
R; *K*; MA; Angel: normal

Anticoagulants/haemophilia
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%

Conventional and near-patient tests of coagulation
Continuing Education in Anaesthesia Critical Care & Pain, Volume 13, Issue 1, 1 February 2013, Pages 12–16

Thromboelastography <TEG>

- **Limitation** :

Inability to detect impairment in platelet function induced by anti-platelet agents

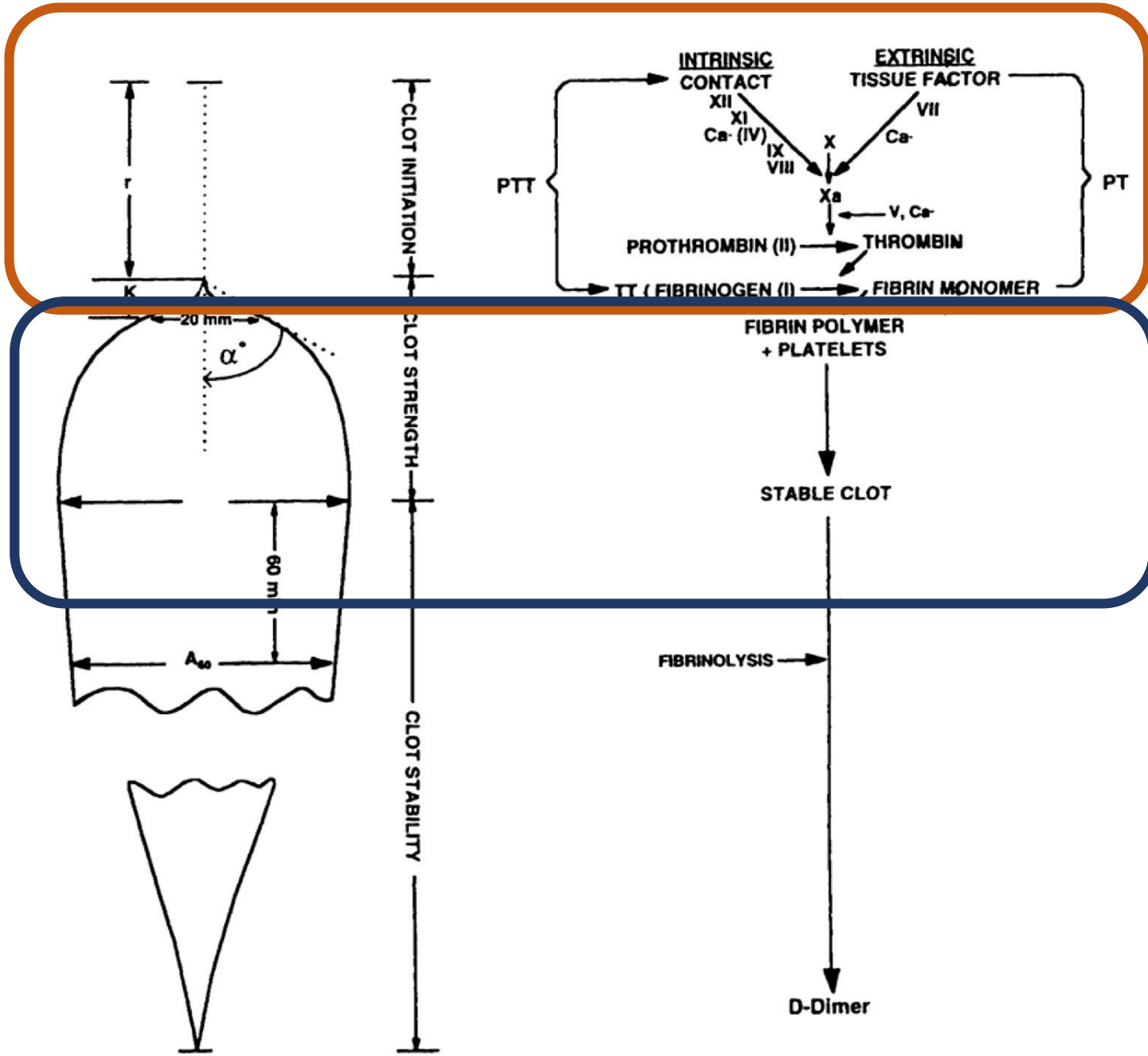
Poor ability to detect condition affect platelet adhesion e.g.von Willebrand's disease

Point-of-care coagulation testing
Continuing Education in Anaesthesia Critical Care & Pain,
Volume 13, Issue 1, 1 February 2013, Pages 12–16

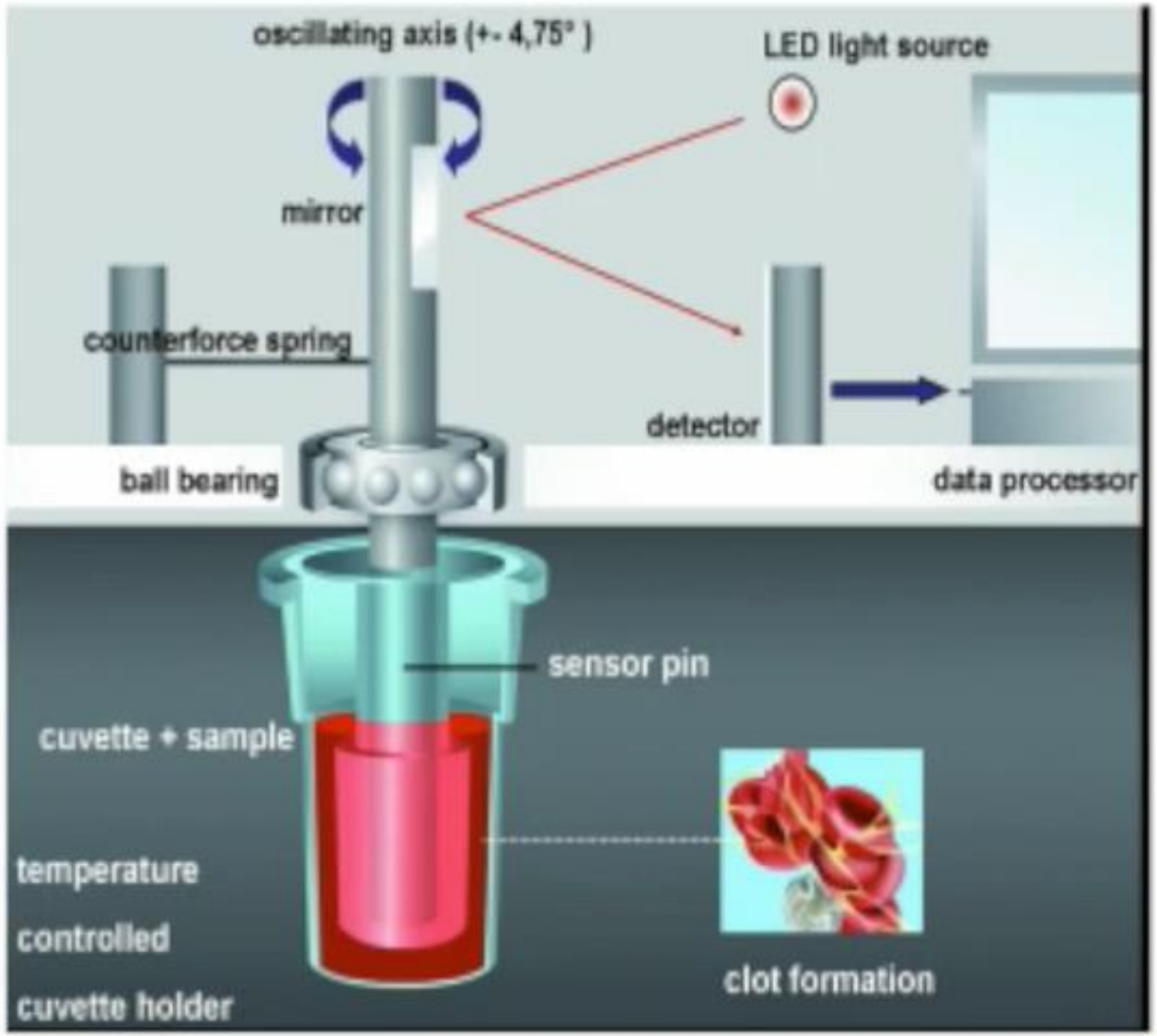
Limitation : TEG

Prolong R

Narrow amplitude



Rotational thromboelastometry<ROTEM>

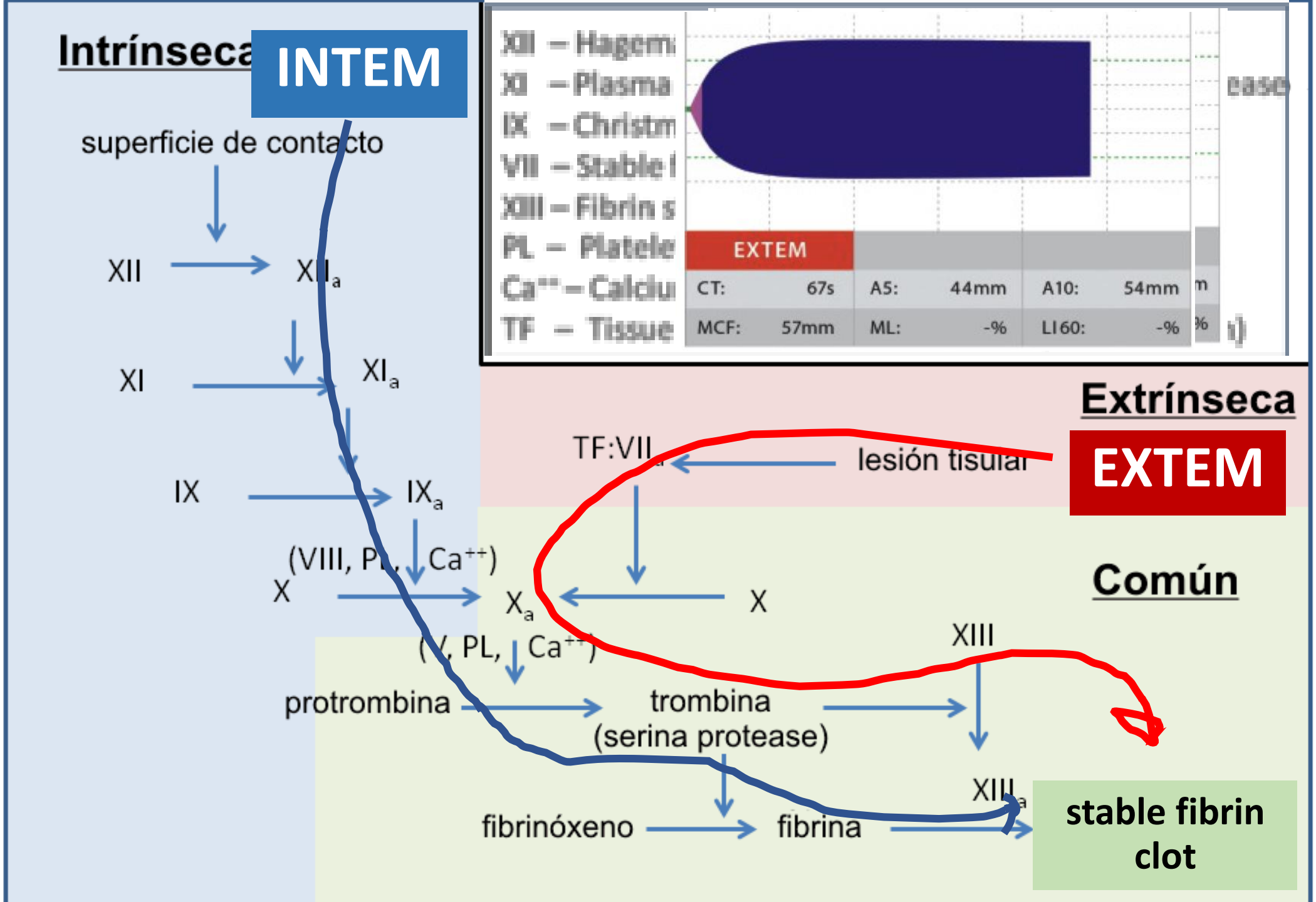


ROTEM's unique shaft spring and ball bearing technology provides for high level of precision and sensitivity of clot formation

ROTEM ASSAY

- **INTEM**
  - HEPTEM

- **EXTEM**
  - FIBTEM
  - APTEM

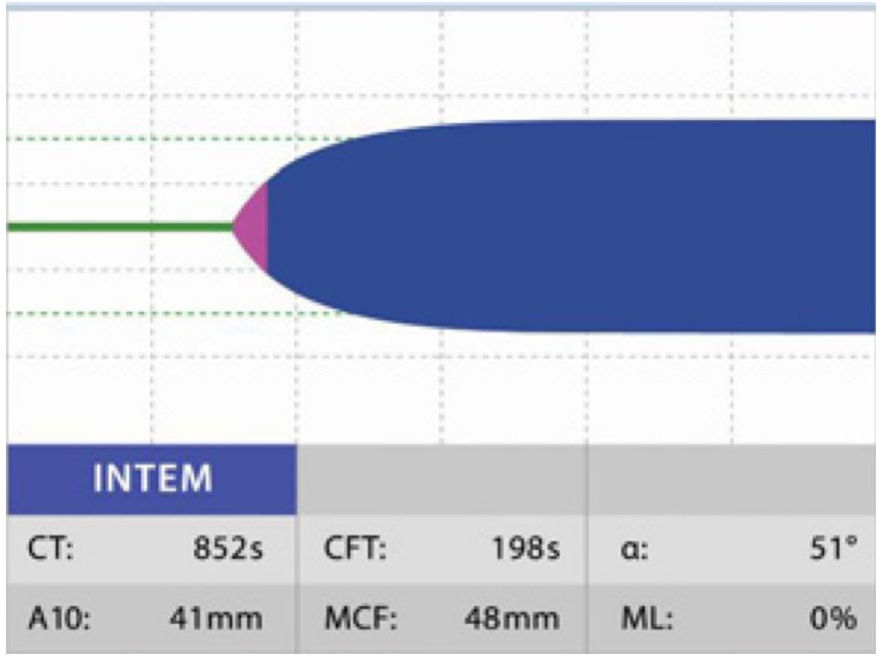

Intrínseca
INTEM
superficie de contacto
XII
XIIa
XI
XIa
IX
IXa
(VIII, PL, Ca++)
X
Xa
(V, PL, Ca++)
protrombina
trombina
(serina protease)
fibrinógeno
fibrina
XIIIa
XIII
Extrínseca
EXTEM
TF:VII
lesión tisular
X
Común
stable fibrin clot
EXTEM
CT: 67s
A5: 44mm
A10: 54mm
MCF: 57mm
ML: -%
LI60: -%

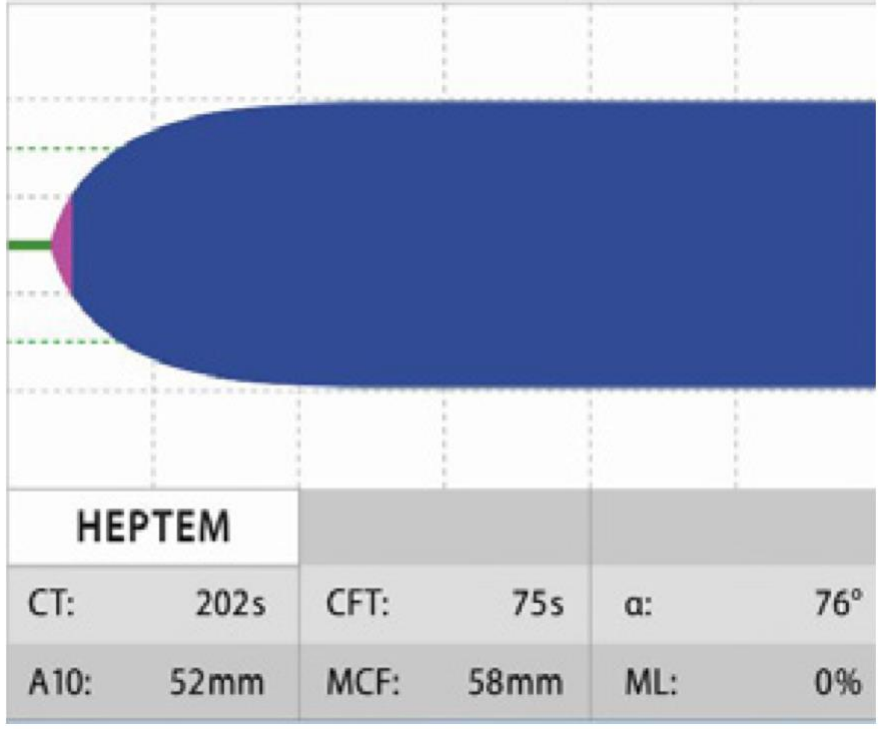
ROTEM assay : HEPTEM

➡ ***Residual heparinization***

Activation as in INTEM with the addition of heparinase

HEPTEM compared to INTEM

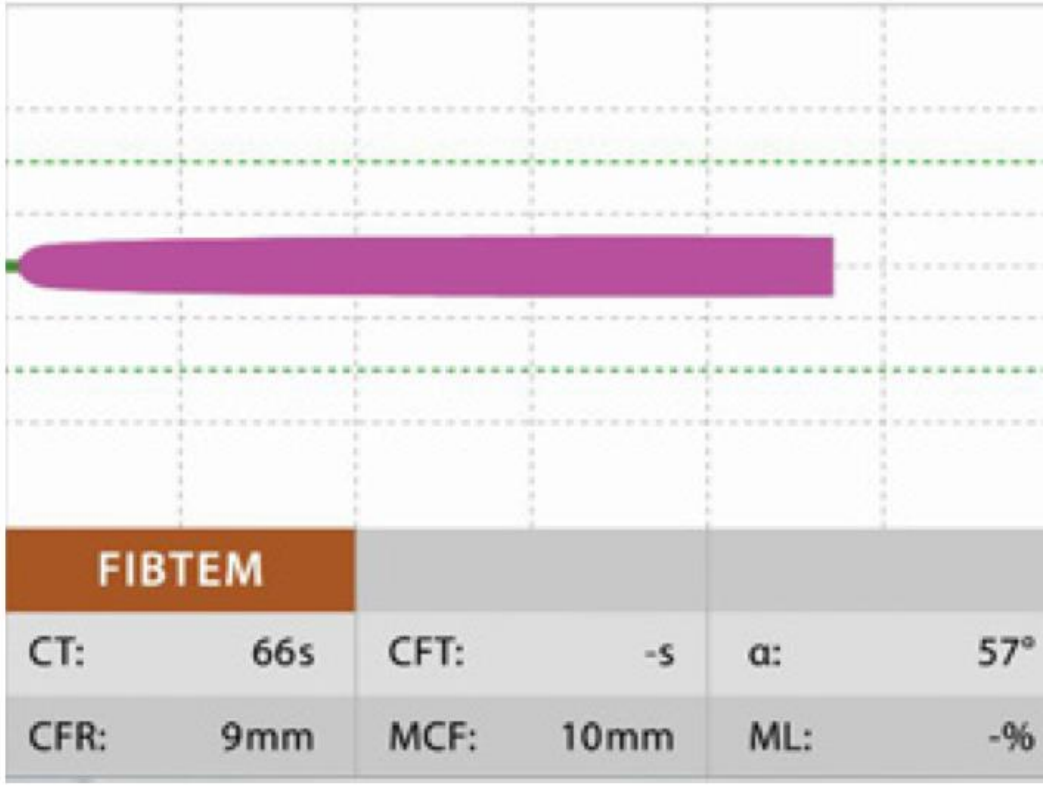




ROTEM assay : FIBTEM

➡ ***Fibrinogen level & Fibrin net polymerization***

Activation as in EXTEM with the addition of platelet blocking substance

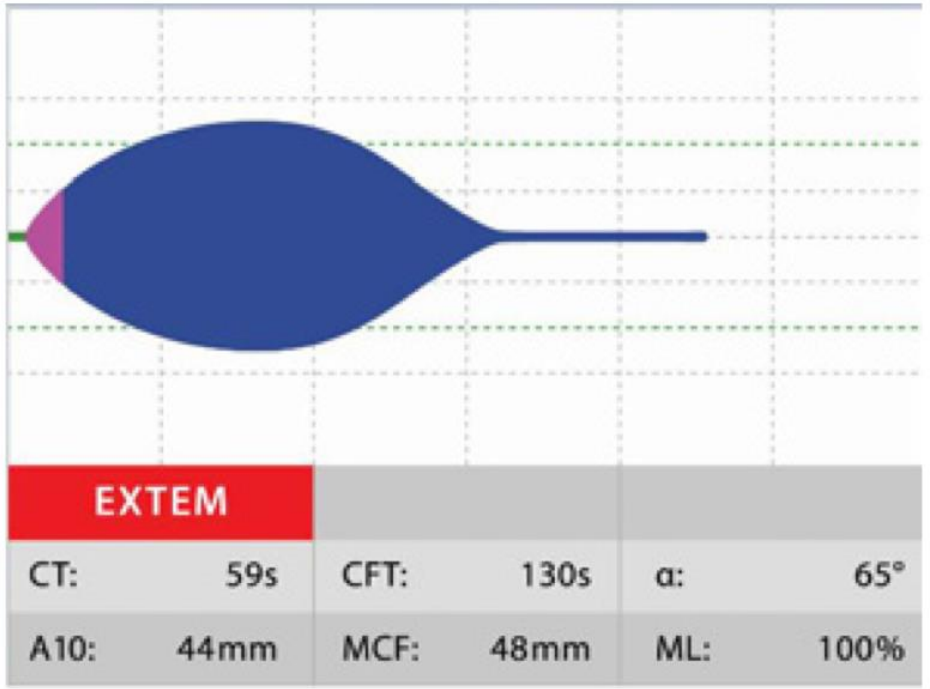


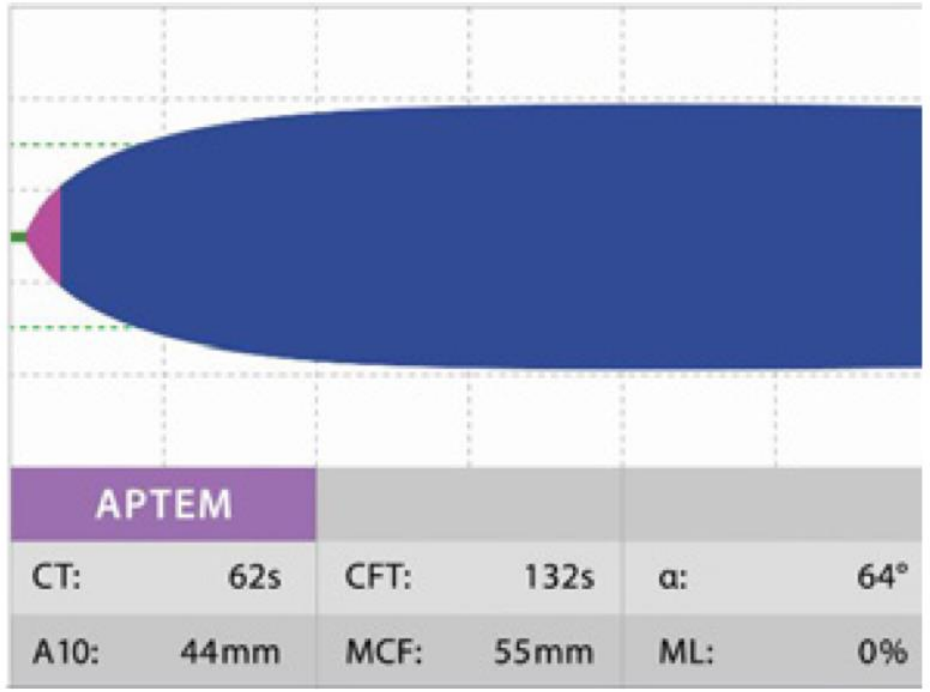
ROTEM assay : APTEM

➡ ***Hyperfibrinolysis***

Activation as in EXTEM with the addition fibrinolysis inhibitors

APTEM compared with EXTEM





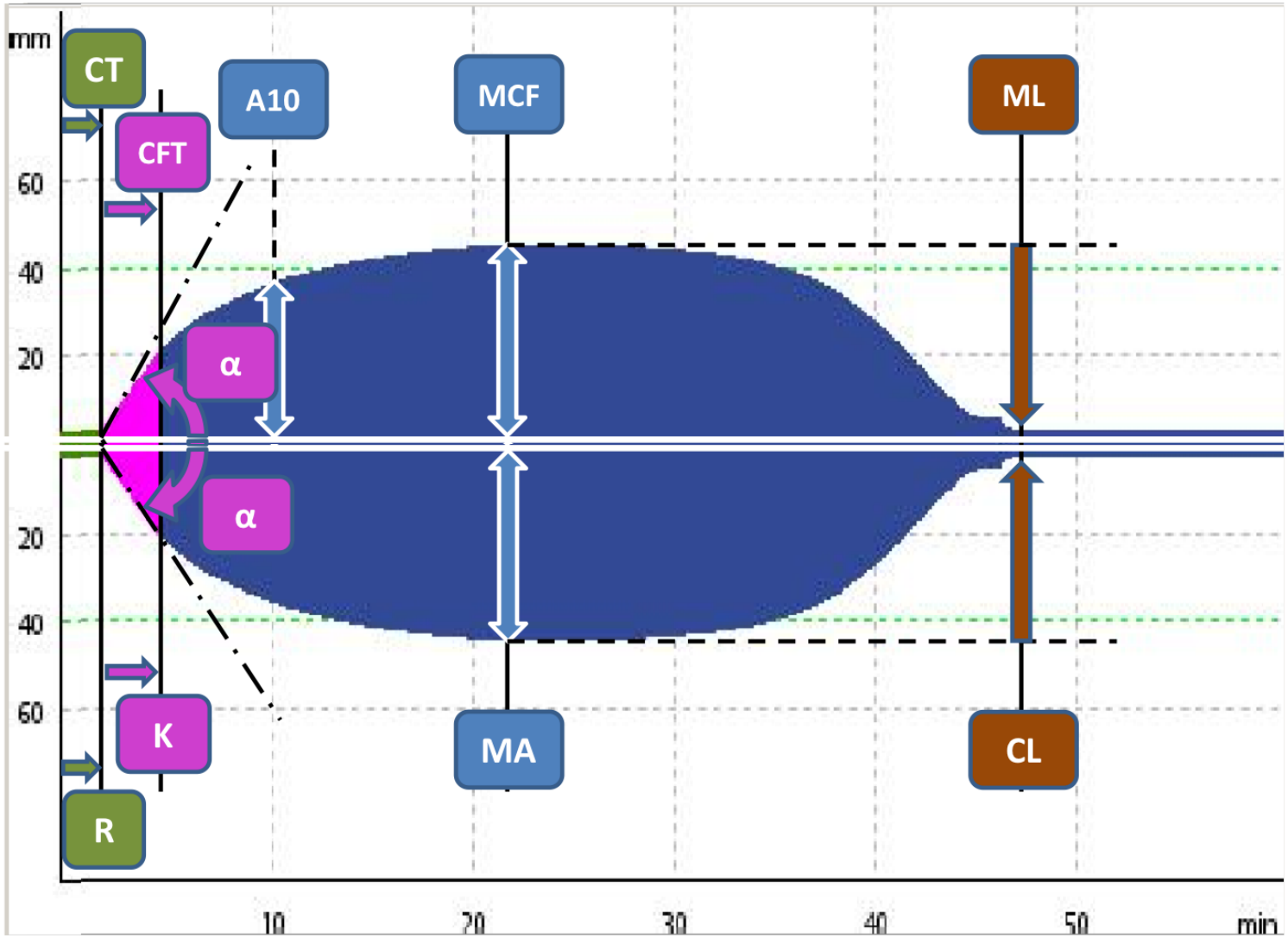
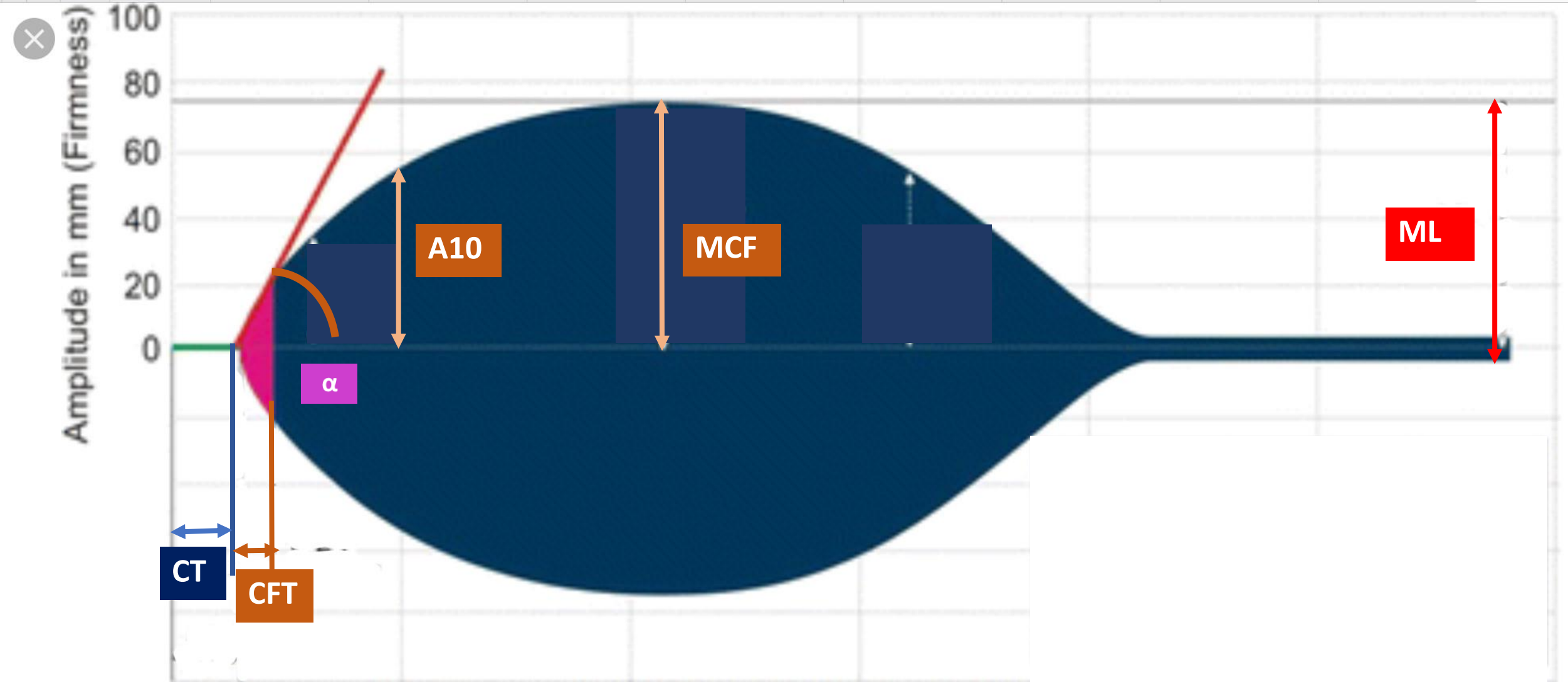
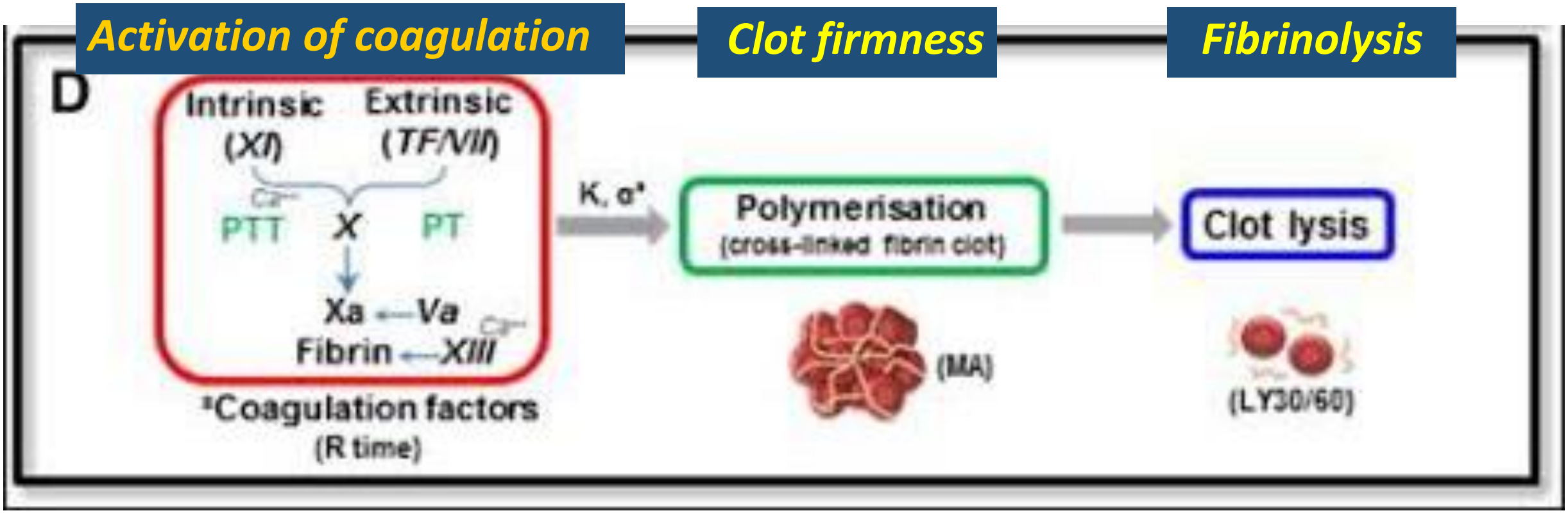
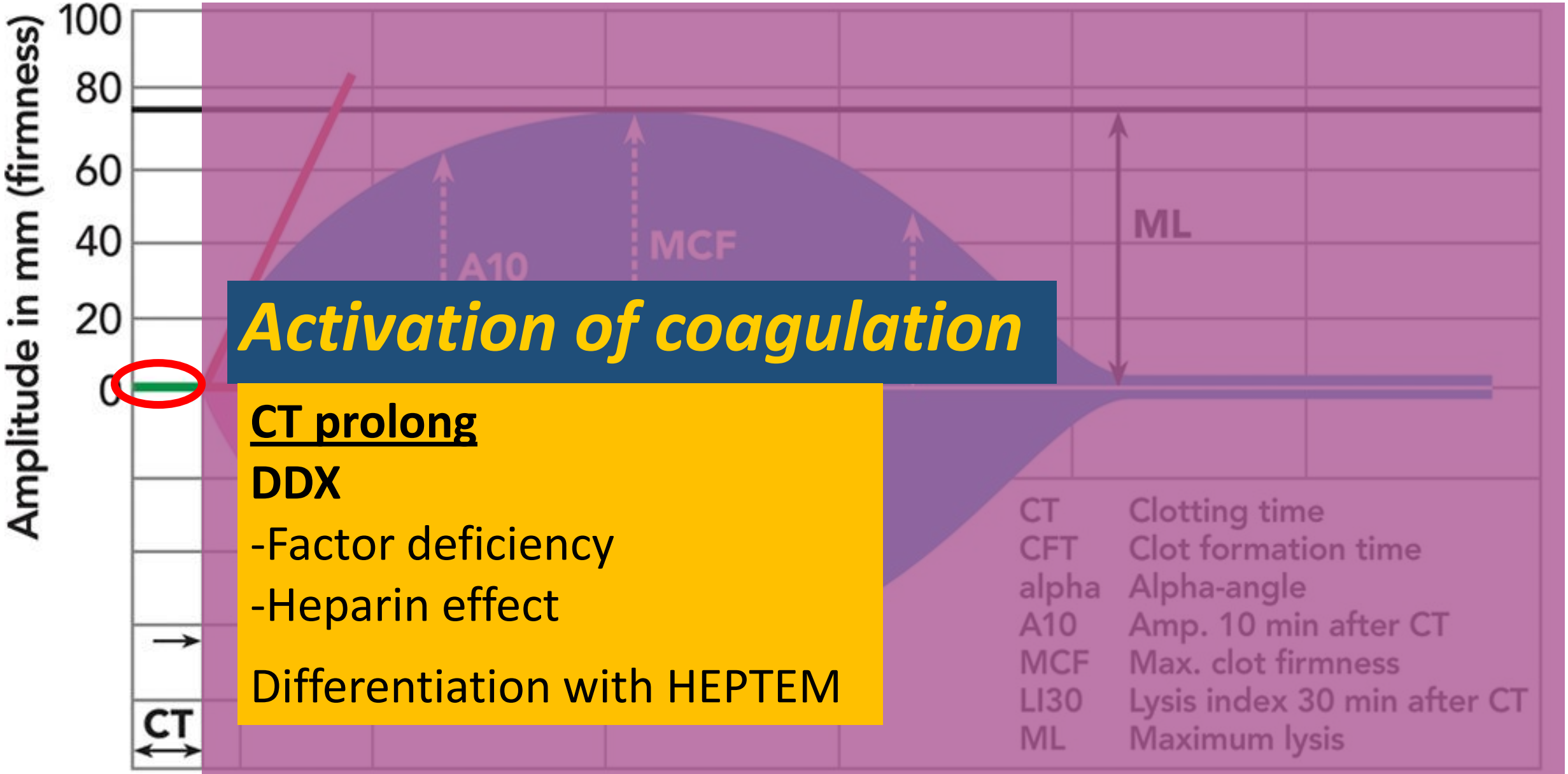


FIGURE 2 | The typical tracings of ROTEM® (upper panel) and TEG® devices (lower panel) with the most prominent parameters of both methods with the comparison (see also Table 2).


Amplitude in mm (Firmness)
100
80
60
40
20
0
A10
MCF
ML
α
CT
CFT

ROTEM : INTERPRETATION





Activation of coagulation

<u>CT prolong</u>

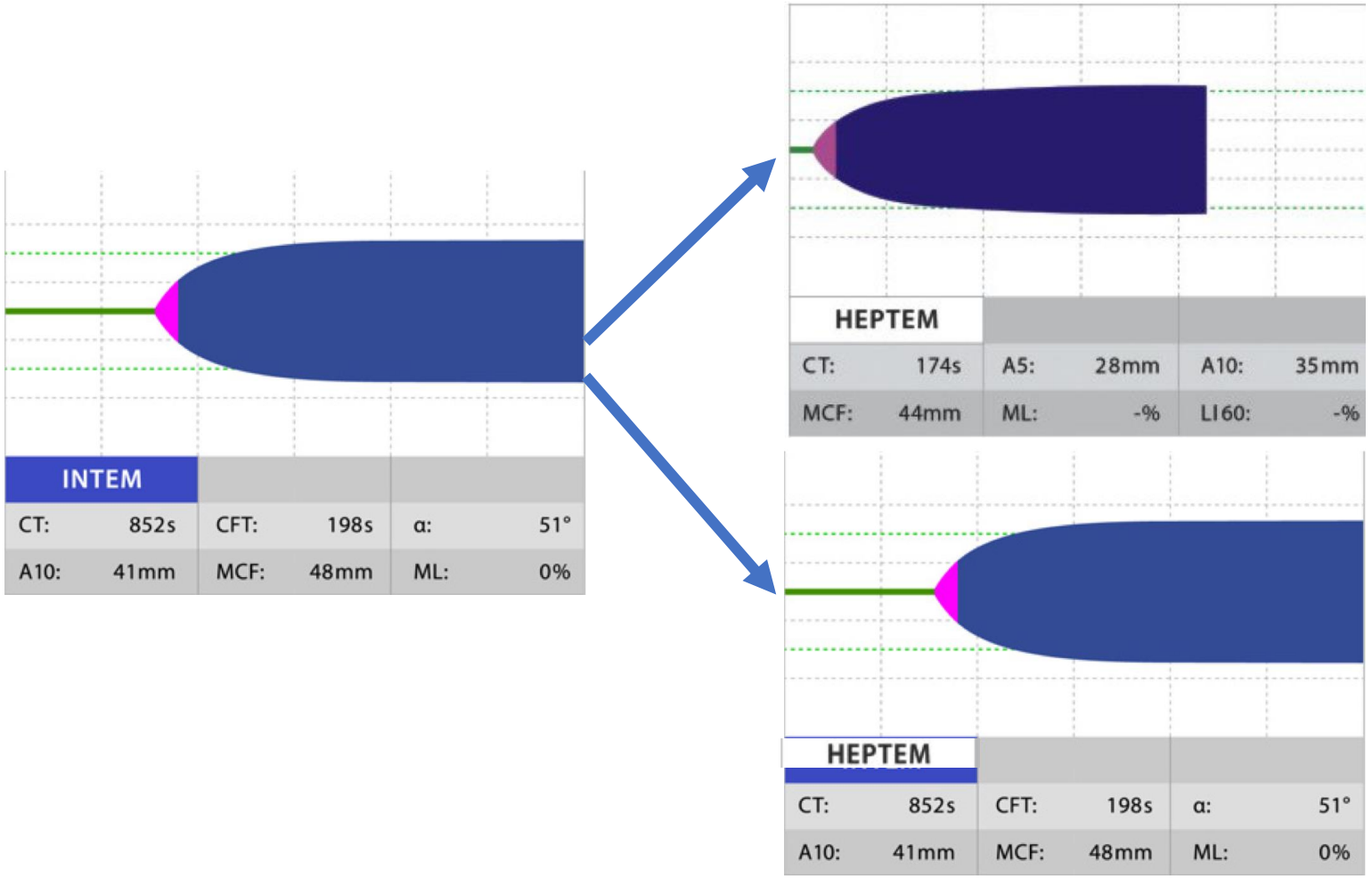
DDX

-Factor deficiency

-Heparin effect

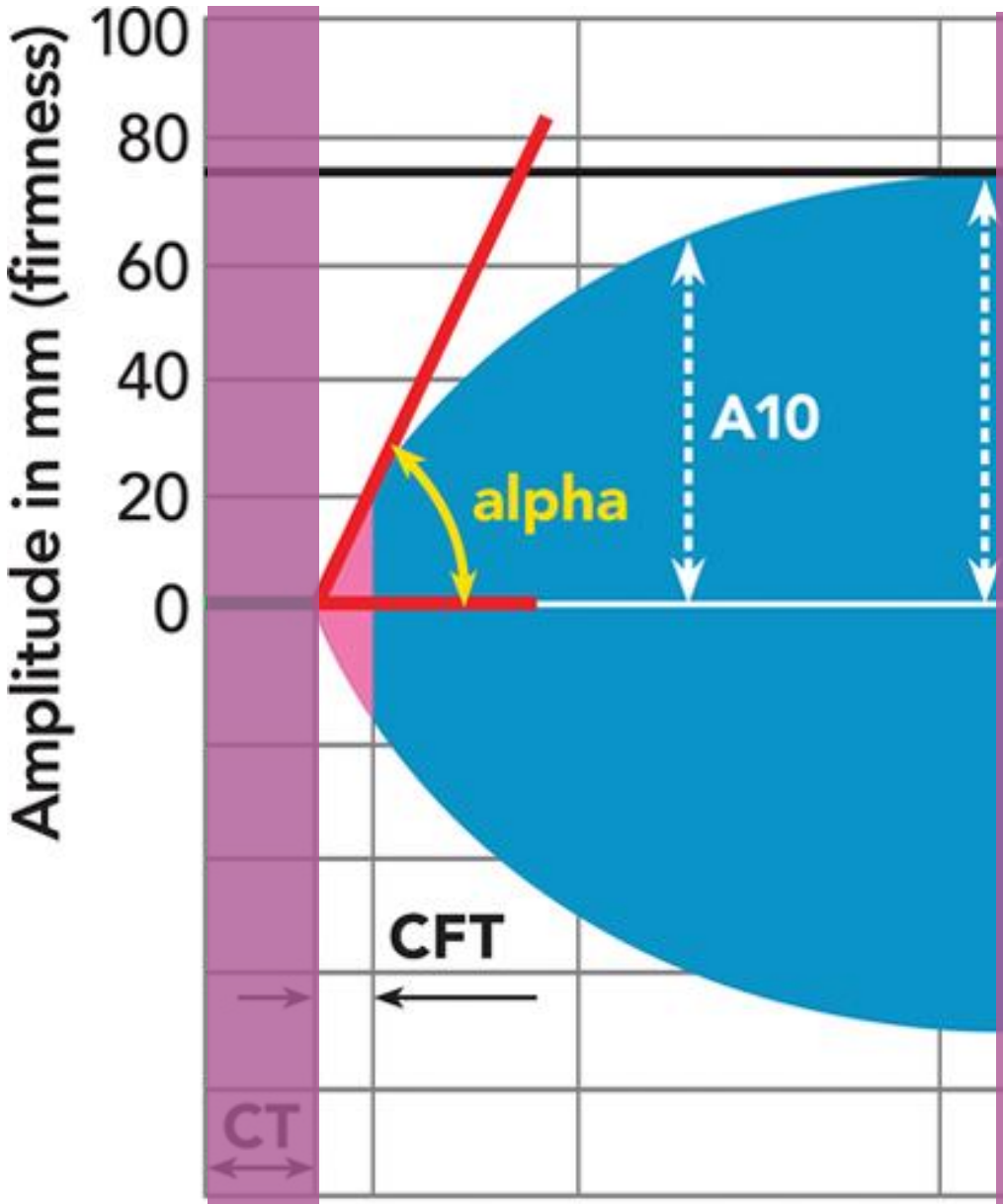
Differentiation with HEPTEM

HEPTEM compare with INTEM



INTEM CT long
HEPTEM CT normalization
—> Heparin effect

INTEM CT long
HEPTEM CT also long
—> No Heparin effect
—> Factor deficiency



Clot firmness

<u>Narrow A10 , MCF</u>

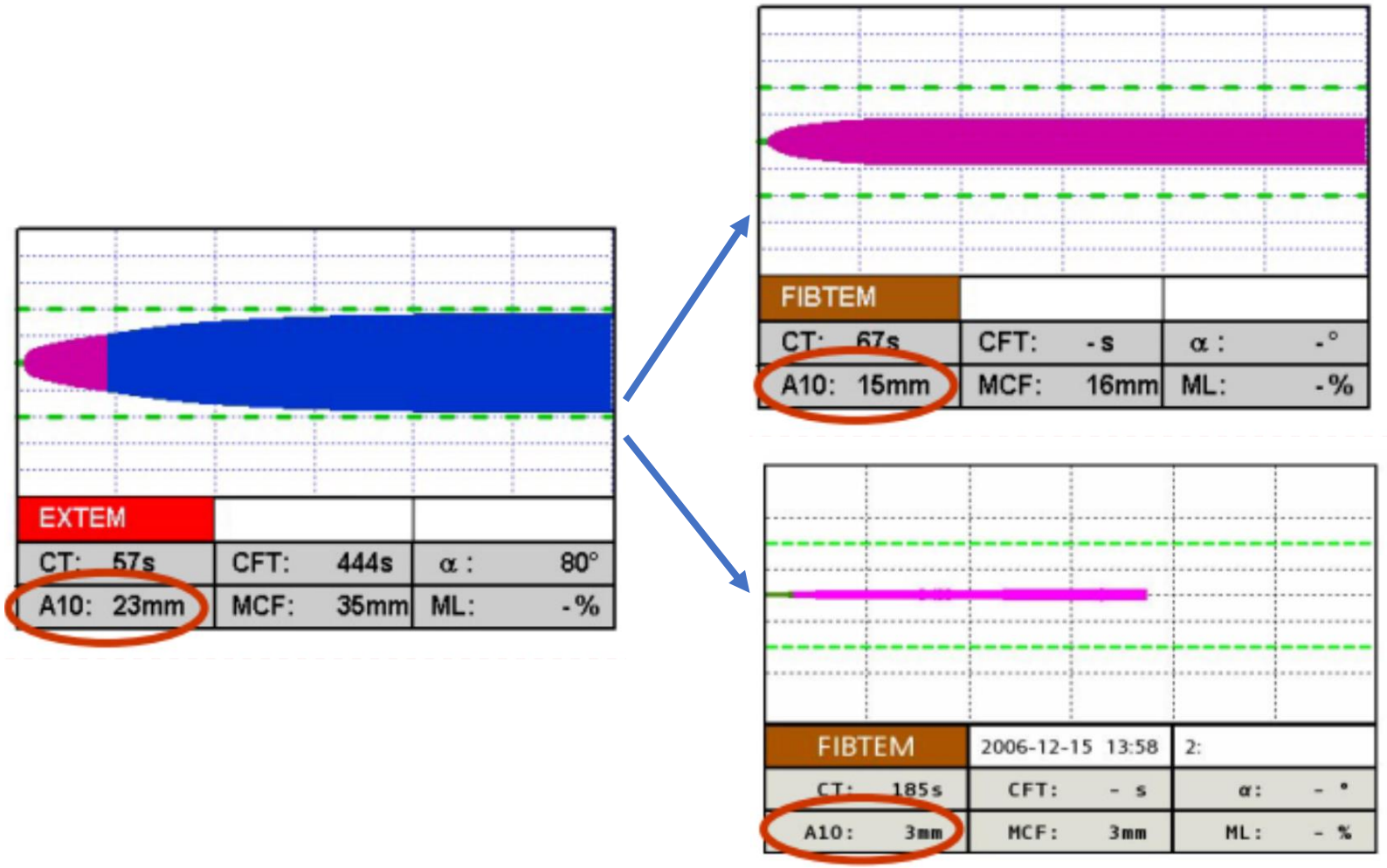
DDX

-Platelets disorder

-Fibrinogen disorder

differentiation with FIBTEM

FIBTEM compare with EXTEM



EXTEM amplitude low
FIBTEM amplitude normal
—> fibrinogen normal
—> platelets deficiency

EXTEM amplitude low
FIBTEM amplitude low
—> fibrinogen deficiency

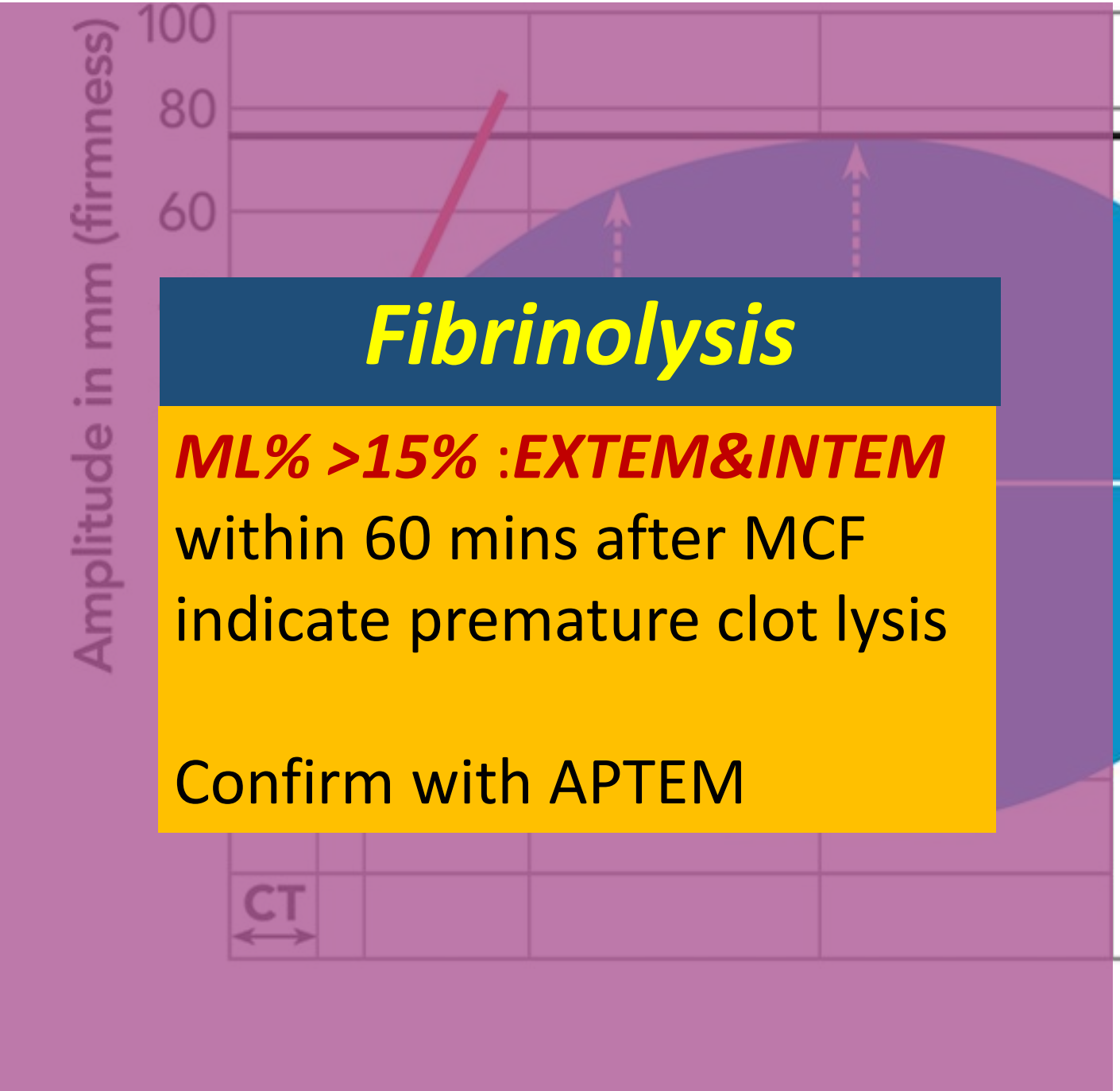
Fibrinolysis

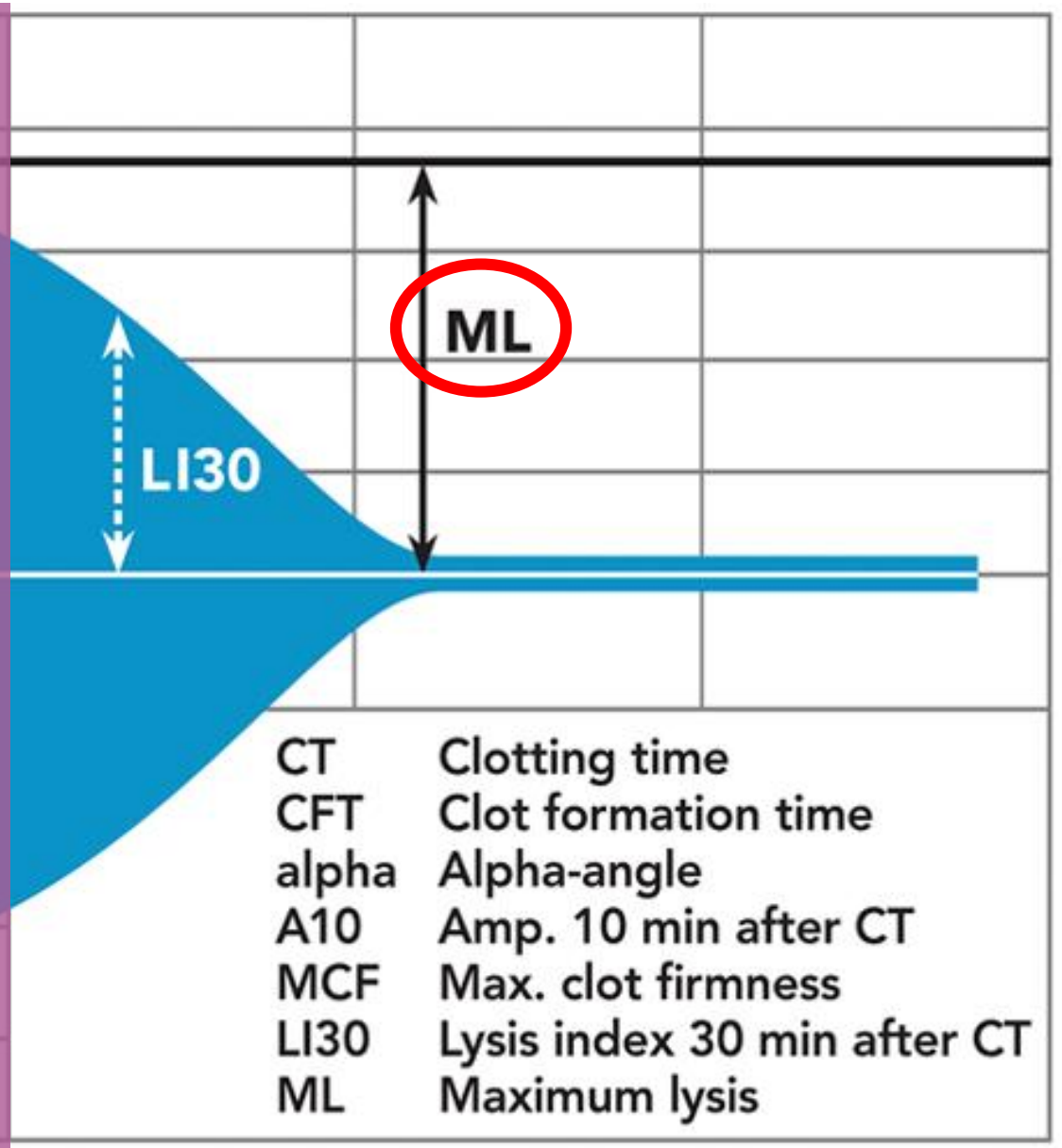
ML% >15% **:*EXTEM&INTEM***

within 60 mins after MCF

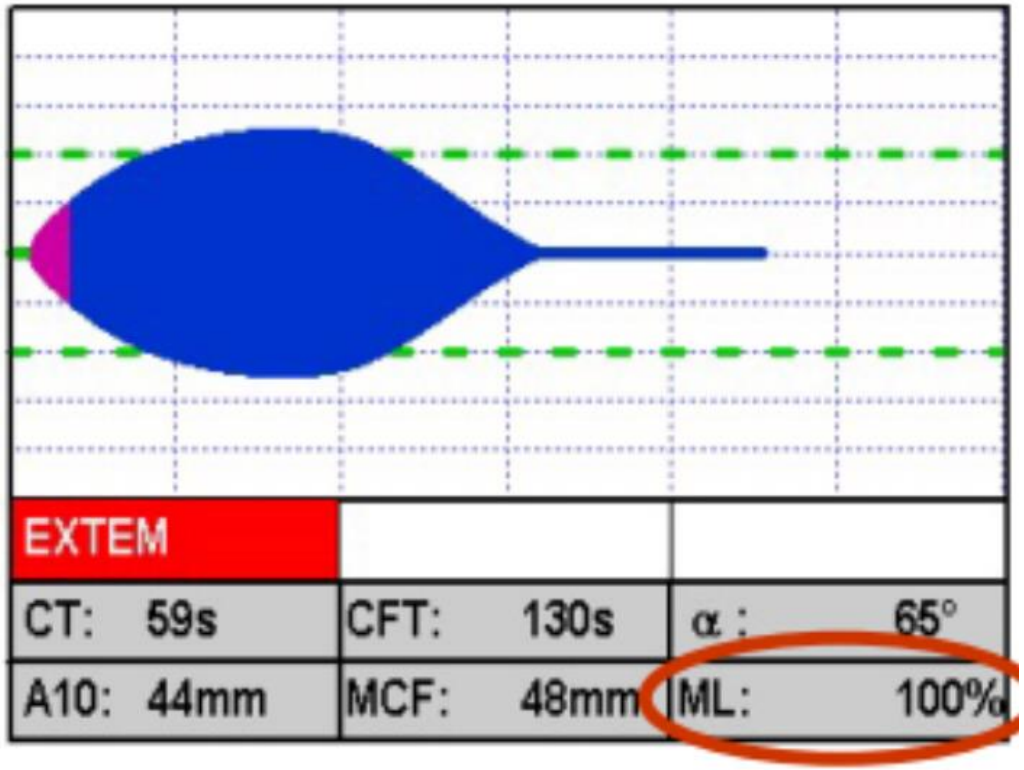
indicate premature clot lysis

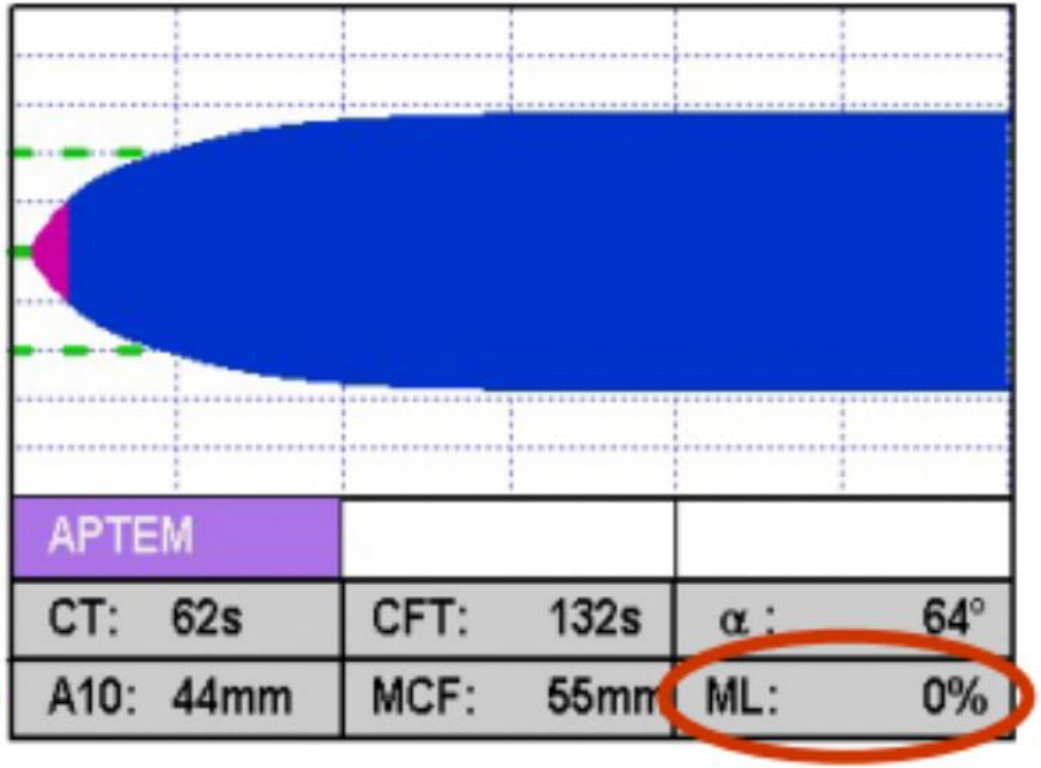
Confirm with APTEM





APTEM compare with EXTEM





EXTEM: hyperfibrinolysis. ML >100% APTEM: fibrinolysis inhibited. ML <15%

—> Hyperfibrinolysis

ROTEM

- **Limitation** :

Inability to detect impairment in platelet function induced by anti-platelet agents

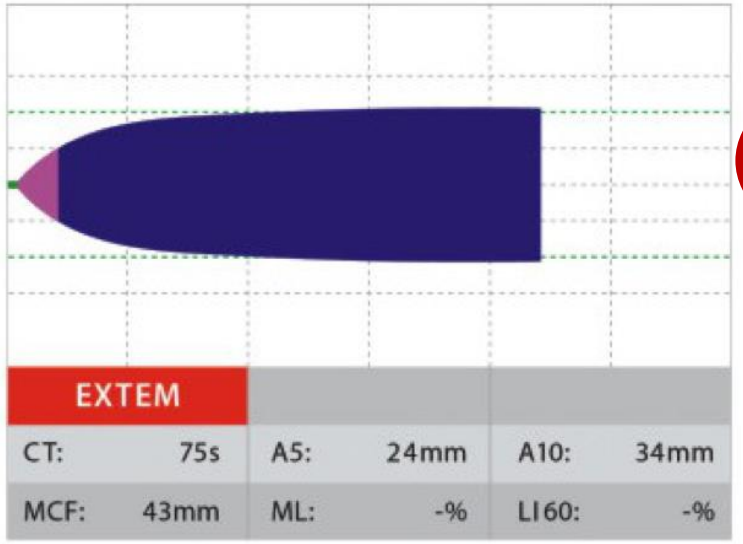
Poor ability to detect condition affect platelet adhesion e.g.von Willebrand's disease

Point-of-care coagulation testing
Continuing Education in Anaesthesia Critical Care & Pain,
Volume 13, Issue 1, 1 February 2013, Pages 12–16

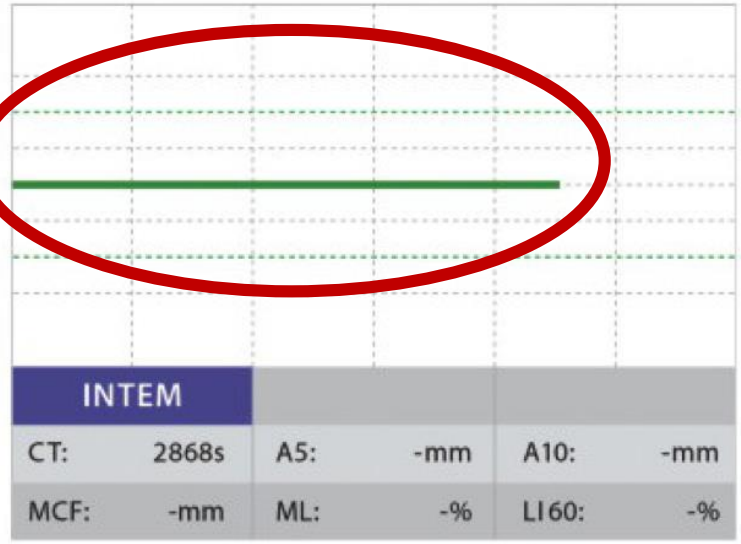

QUIZ

- 67-year-old woman was diagnosed severe AR from IE s/p MVR AVR on warfarrin
- Preoperative lab : INR 1.7
- Operation Redo AVR
- Post CPB:
  - Bleeding from surgical field despite 2 rounds of blood components

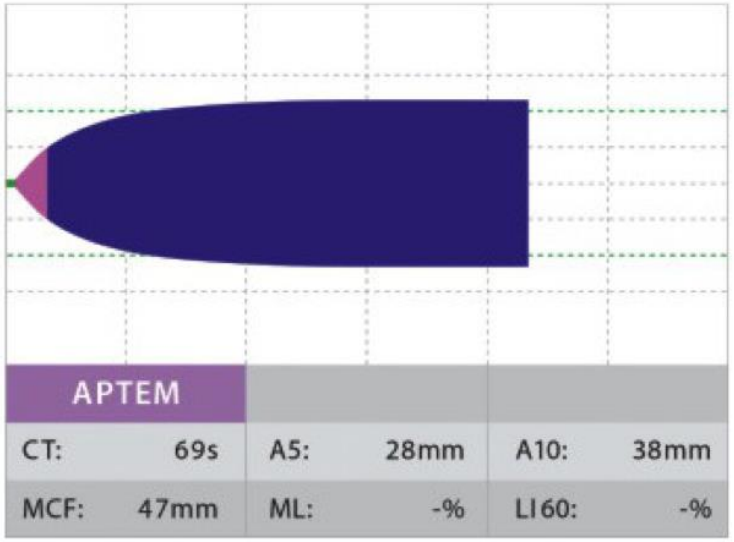
Analyze the ROTEM result



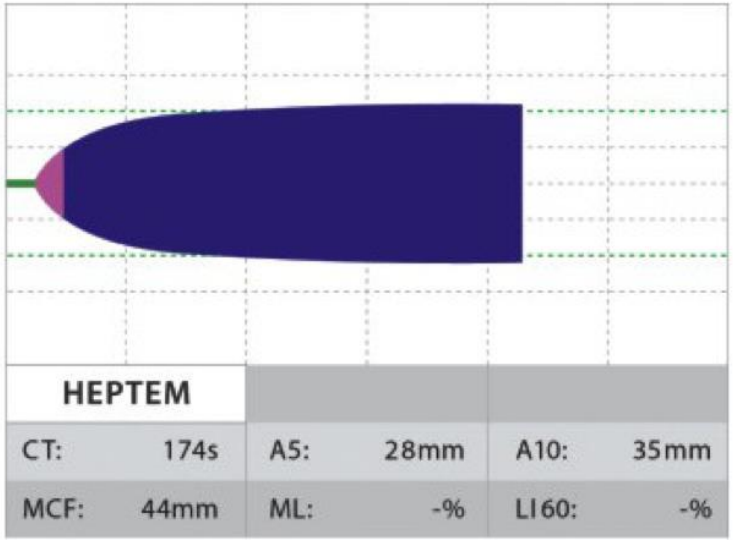
EXTEM					
CT:	75s	A5:	24mm	A10:	34mm
MCF:	43mm	ML:	-%	LI60:	-%



INTEM					
CT:	2868s	A5:	-mm	A10:	-mm
MCF:	-mm	ML:	-%	LI60:	-%



APTEM					
CT:	69s	A5:	28mm	A10:	38mm
MCF:	47mm	ML:	-%	LI60:	-%



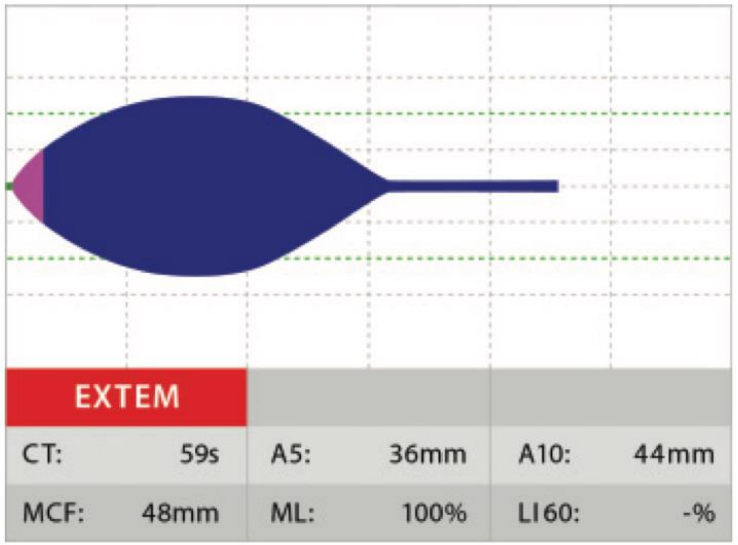
HEPTEM					
CT:	174s	A5:	28mm	A10:	35mm
MCF:	44mm	ML:	-%	LI60:	-%

After Protamine neutralization

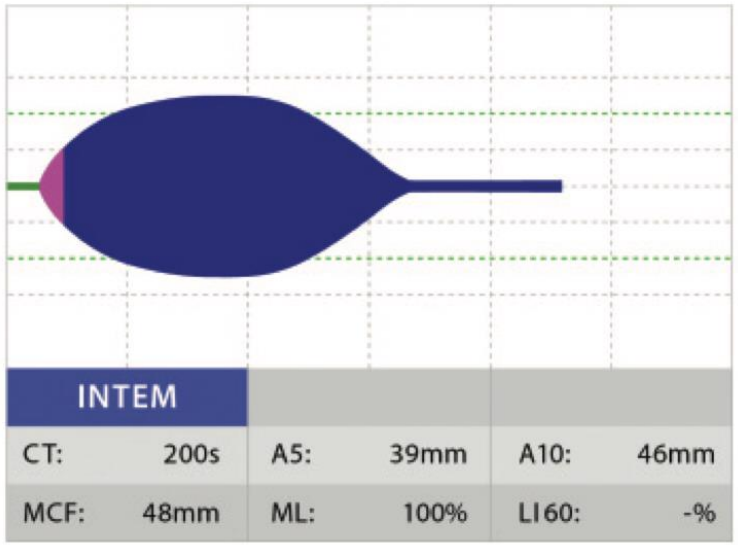
- Clots in surgical field & Operation success

- After 6 hr Continuous bleeding from the drain!!!!!

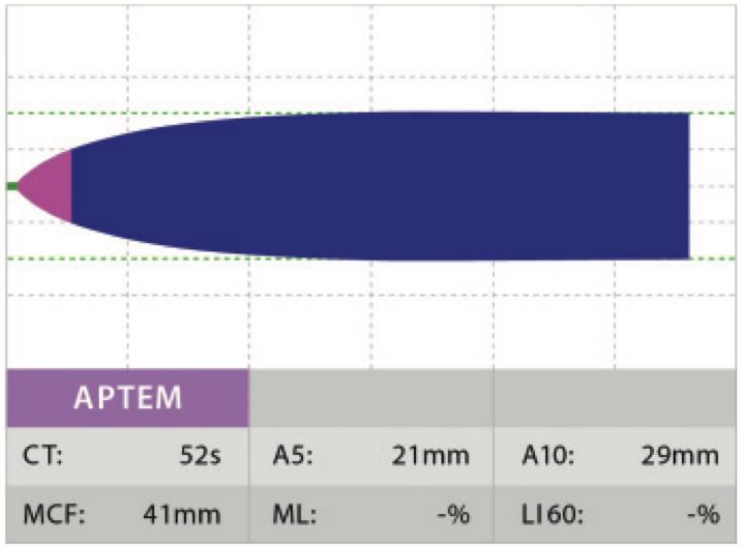
Analyze the ROTEM result at ICU



EXTEM					
CT:	59s	A5:	36mm	A10:	44mm
MCF:	48mm	ML:	100%	LI60:	-%

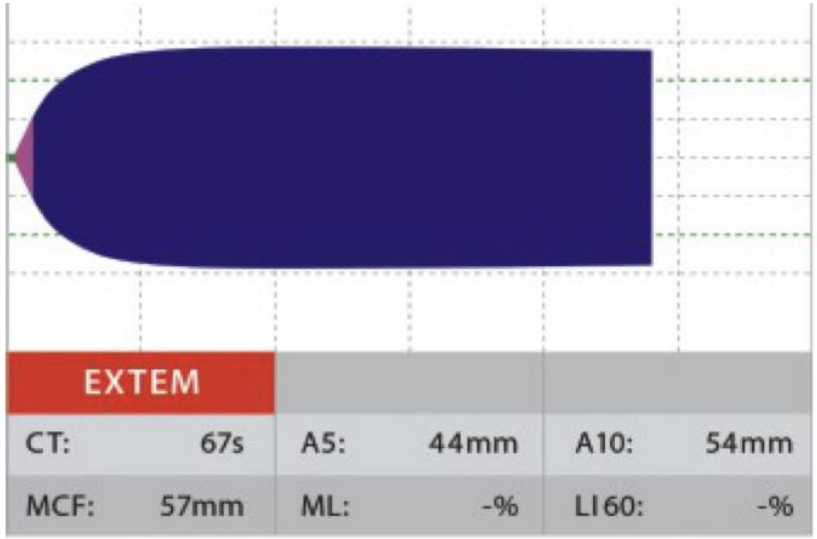


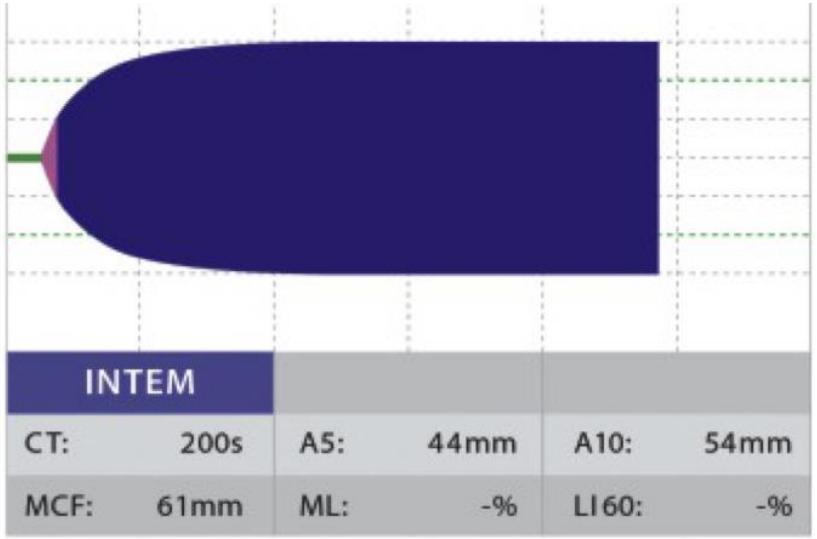
INTEM					
CT:	200s	A5:	39mm	A10:	46mm
MCF:	48mm	ML:	100%	LI60:	-%

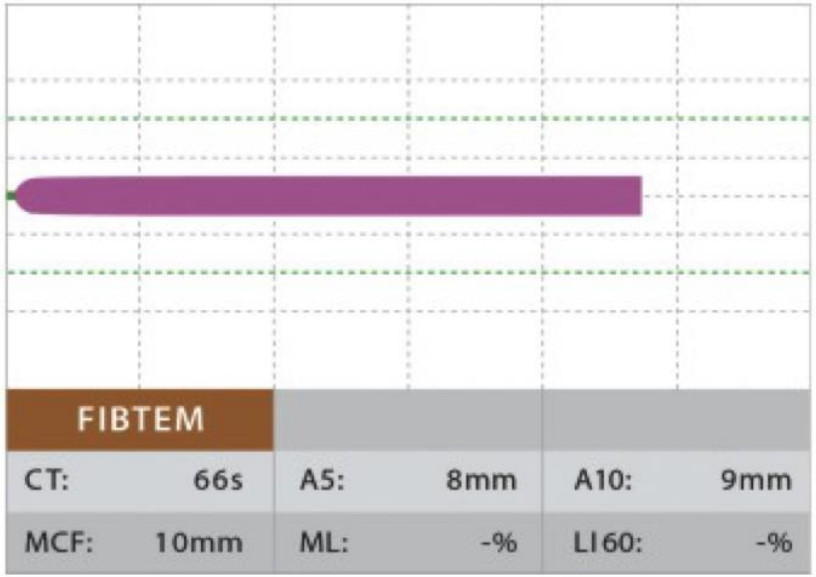


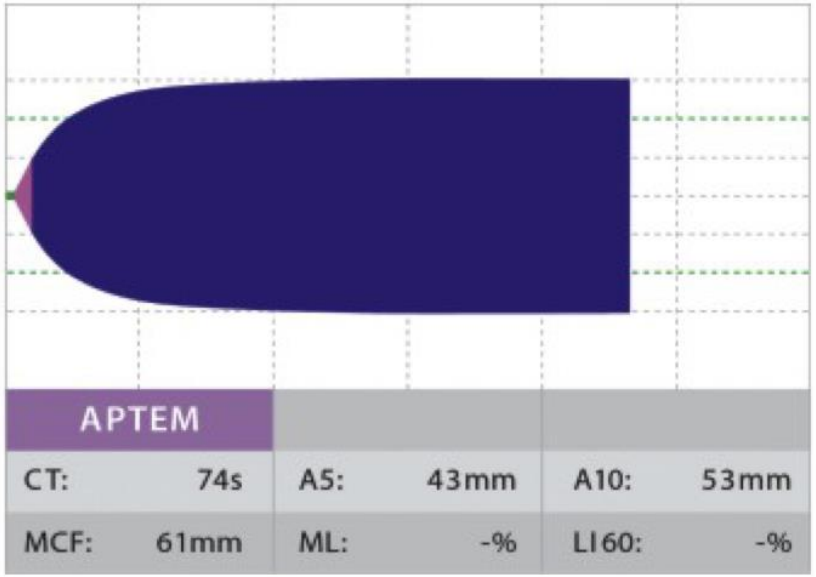
APTEM					
CT:	52s	A5:	21mm	A10:	29mm
MCF:	41mm	ML:	-%	LI60:	-%

After Transamine 1 g IV → *persistent bleeding*









- Set OR Stop bleeddddddddddddddddd


ไม่
เลือดข้นคนจาง