

Thromboelastography (TEG) & Its Role in Determining a Patient's Coagulation Status

ALYSSA SONCHAIWANICH, PHARM.D

MEMORIAL HOSPITAL OF SOUTH BEND

A WEBINAR FOR HEALTHTRUST MEMBERS

MAY 22, 2019



Disclosures

- The presenter has no financial relationships with any commercial interests pertinent to this presentation
- This program may contain the mention of drugs, brands, or suppliers presented in a case study or comparative format using evidence-based research. Such examples are intended for educational and informational purposes and should not be perceived as an endorsement of any particular drug, brand, or supplier

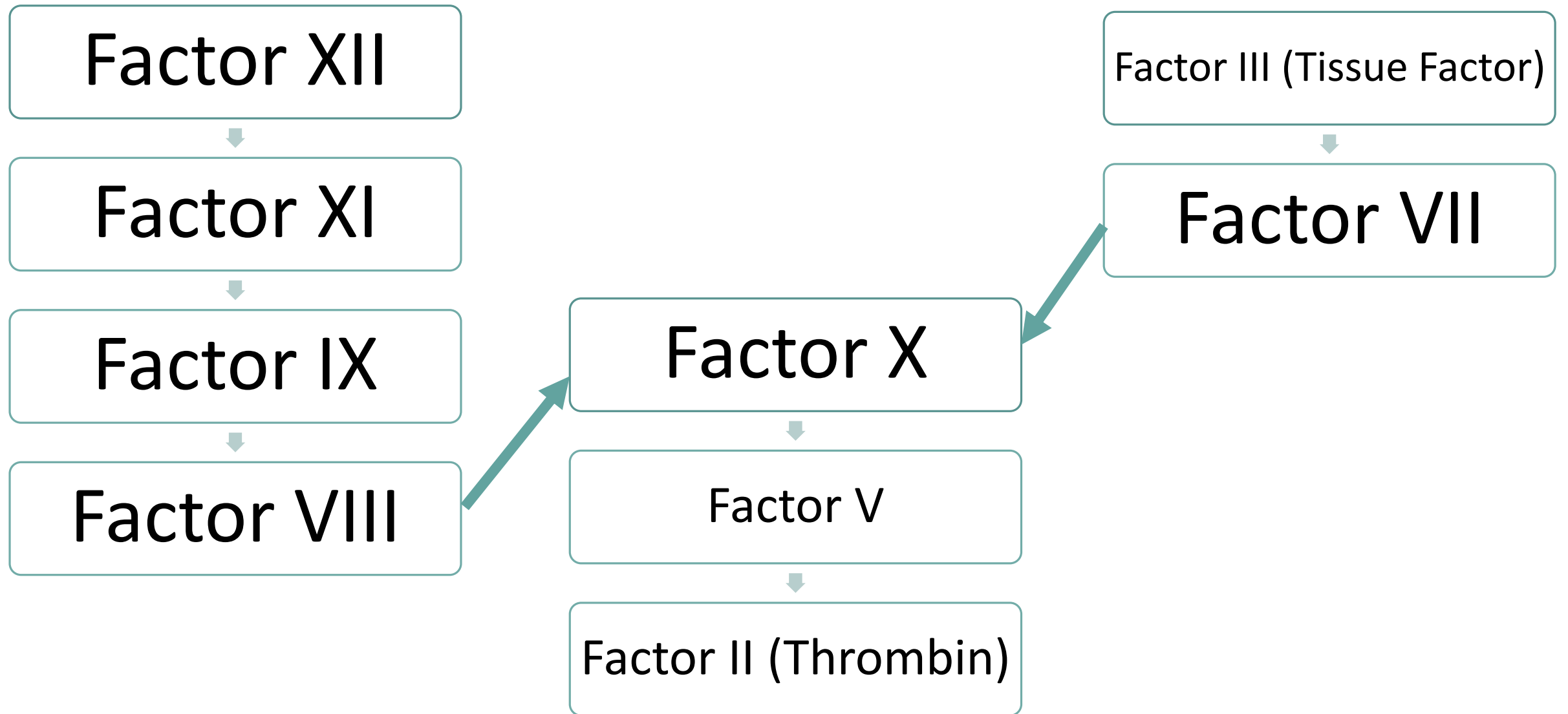
Pharmacist Learning Objectives

At the end of this session, participants should be able to:

- 1. Explain how TEG can be used to determine the coagulation status of a patient**
- 2. Identify key measurements of a TEG and apply it to a TEG analysis**
- 3. Interpret a TEG analysis in order to determine the therapeutic interventions appropriate the patient's coagulation status**

Coagulation Cascade

A BRIEF OVERVIEW



Factor XII



Factor XI



Factor IX



Factor VIII

Intrinsic Pathway

Factor III (Tissue Factor)



Factor VII

Factor X



Factor V



Factor II (Thrombin)

Factor XII



Factor XI



Factor IX



Factor VIII

Intrinsic Pathway



Factor X



Factor V



Factor II (Thrombin)

Factor III (Tissue Factor)



Factor VII



Extrinsic Pathway

Factor XII



Factor XI



Factor IX



Factor VIII

Intrinsic Pathway

Factor III (Tissue Factor)



Factor VII

Extrinsic Pathway

Factor X



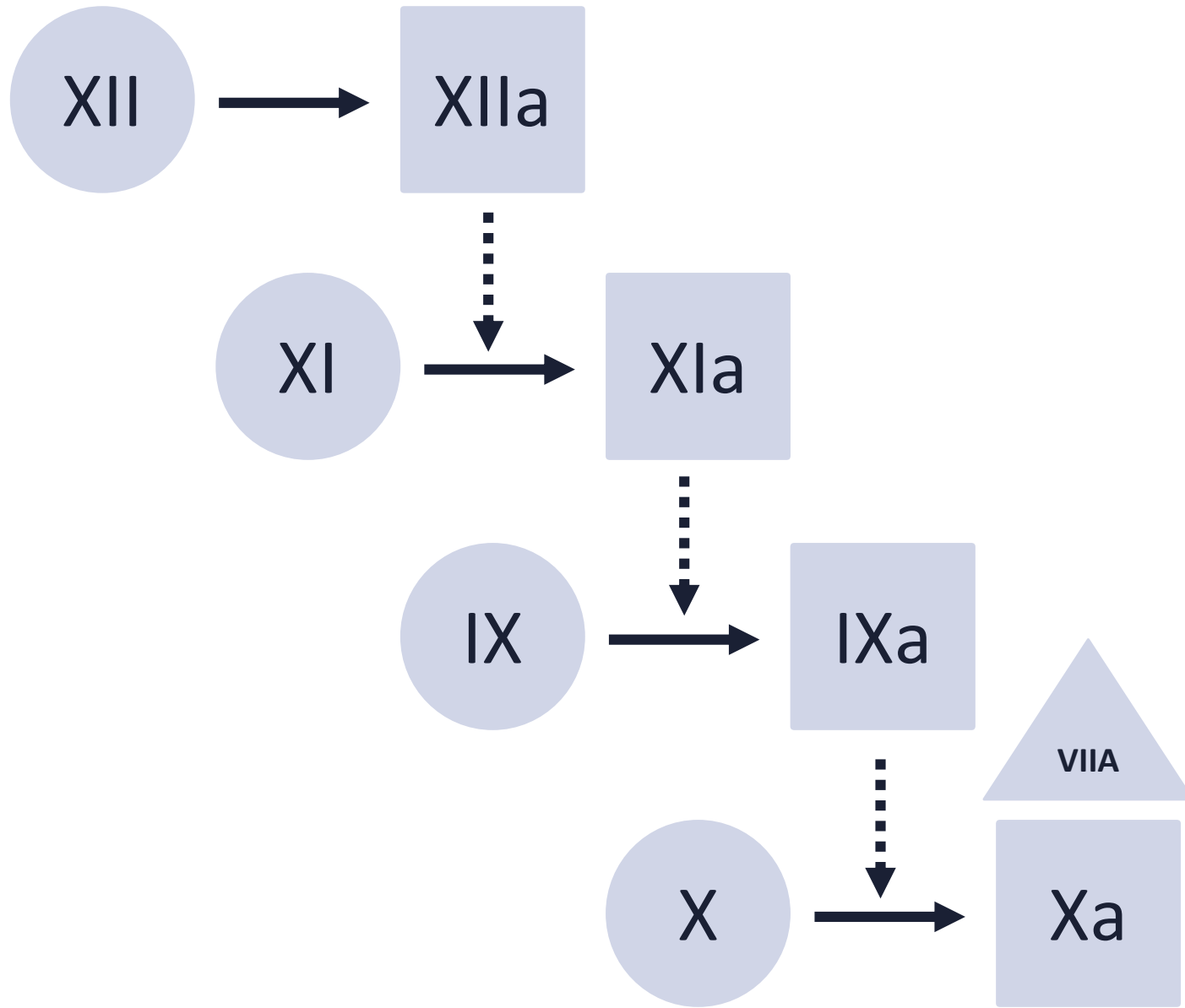
Factor V



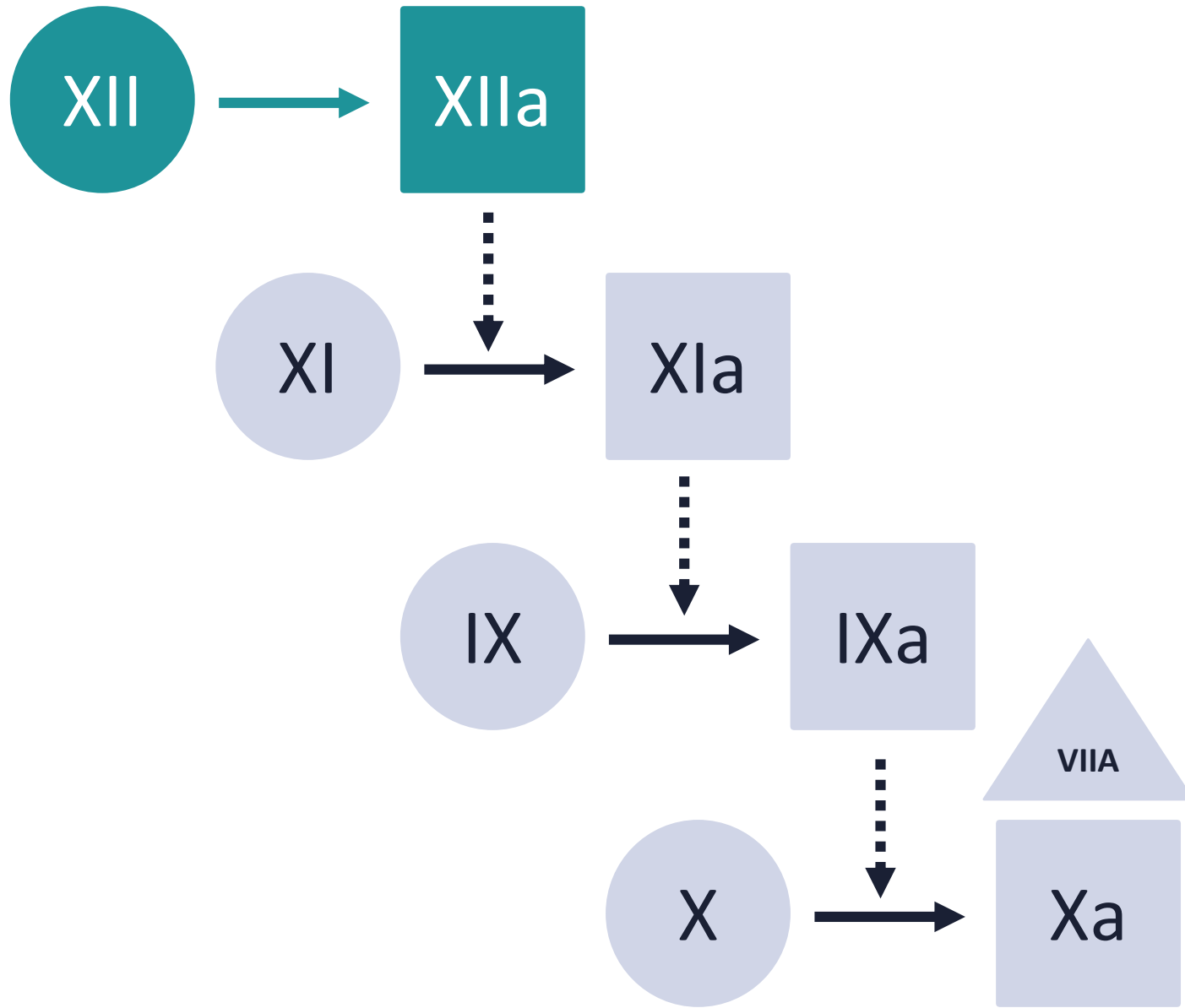
Factor II (Thrombin)

Common Pathway

Intrinsic Pathway

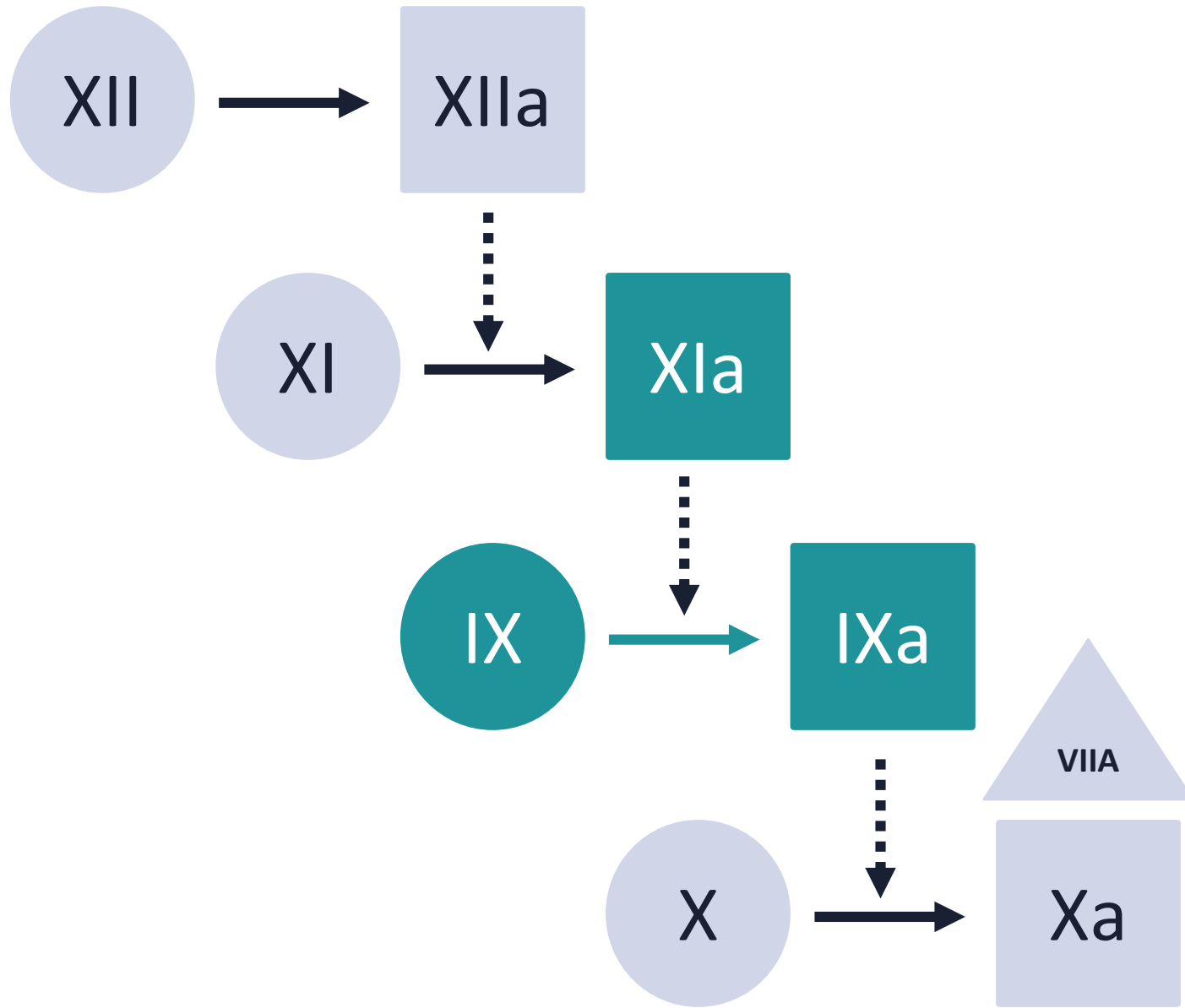


Intrinsic Pathway

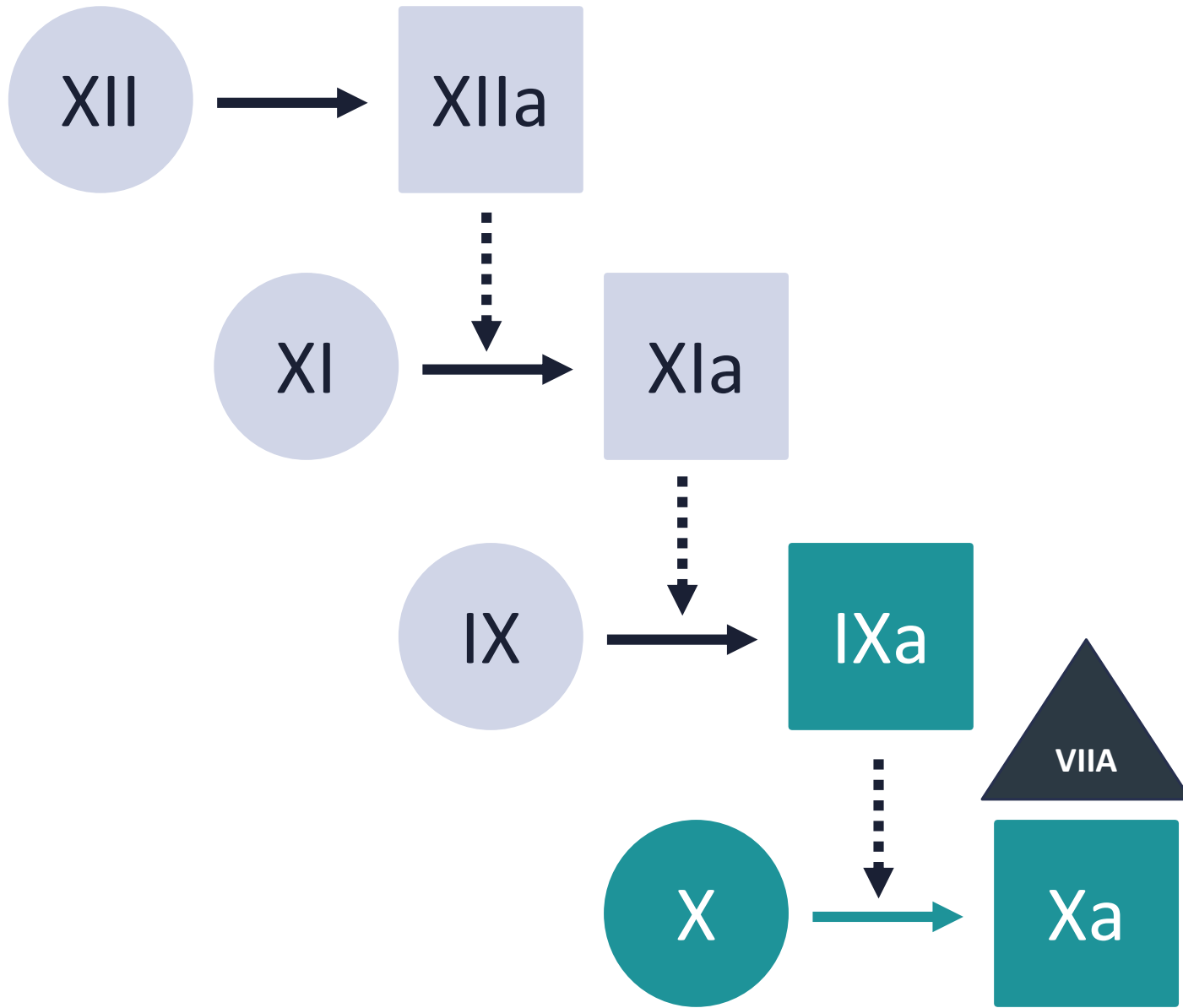


The diagram illustrates the intrinsic pathway of blood coagulation. It begins with Factor XII (a light blue circle) being converted to XIIa (a teal square). XIIa then acts as a catalyst (indicated by a dashed arrow) to convert Factor XI (a teal circle) to XIa (a teal square). XIa then acts as a catalyst (indicated by a dashed arrow) to convert Factor IX (a light blue circle) to IXa (a light blue square). IXa then acts as a catalyst (indicated by a dashed arrow) to convert Factor X (a light blue circle) to Xa (a light blue square). Finally, Xa acts as a catalyst (indicated by a dashed arrow) to convert Factor VII (a light blue triangle) to VIIa (a light blue triangle). The active forms (XIIa, XIa, IXa, Xa, VIIa) are shown in squares, while the inactive forms (XII, XI, IX, X, VII) are shown in circles or triangles.

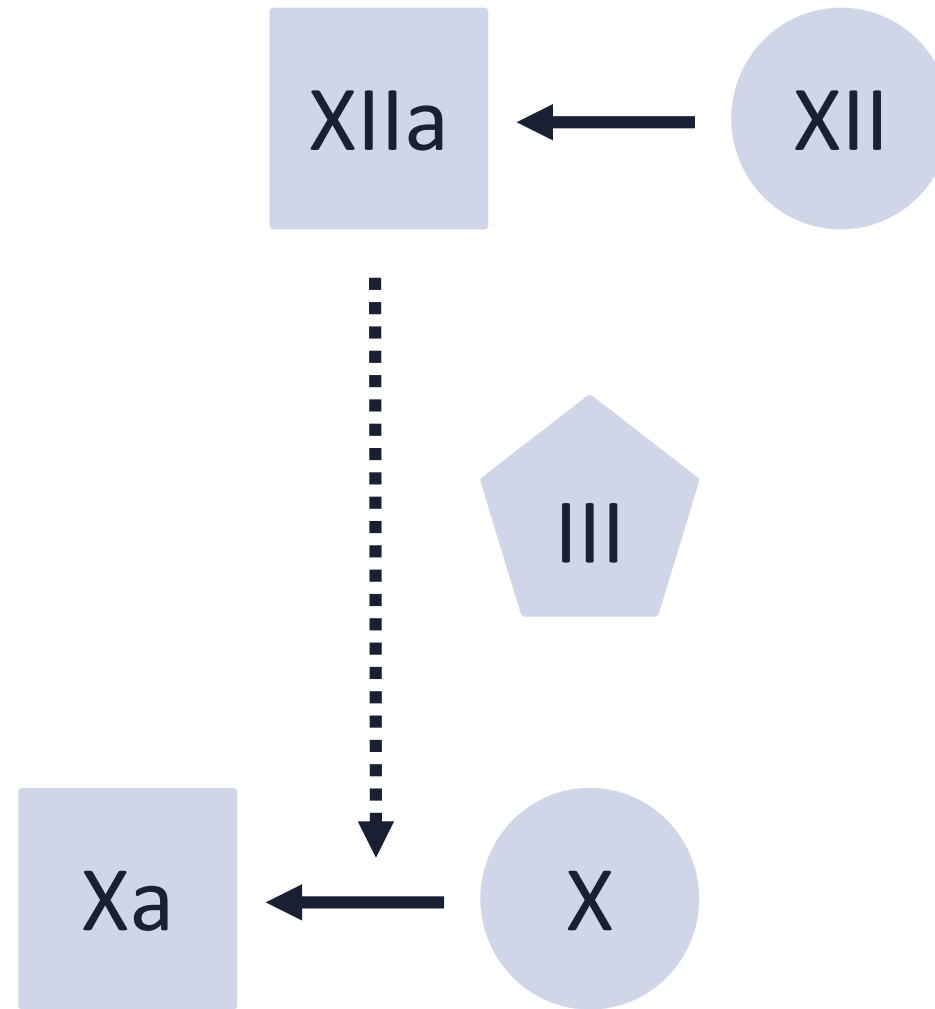
Intrinsic Pathway



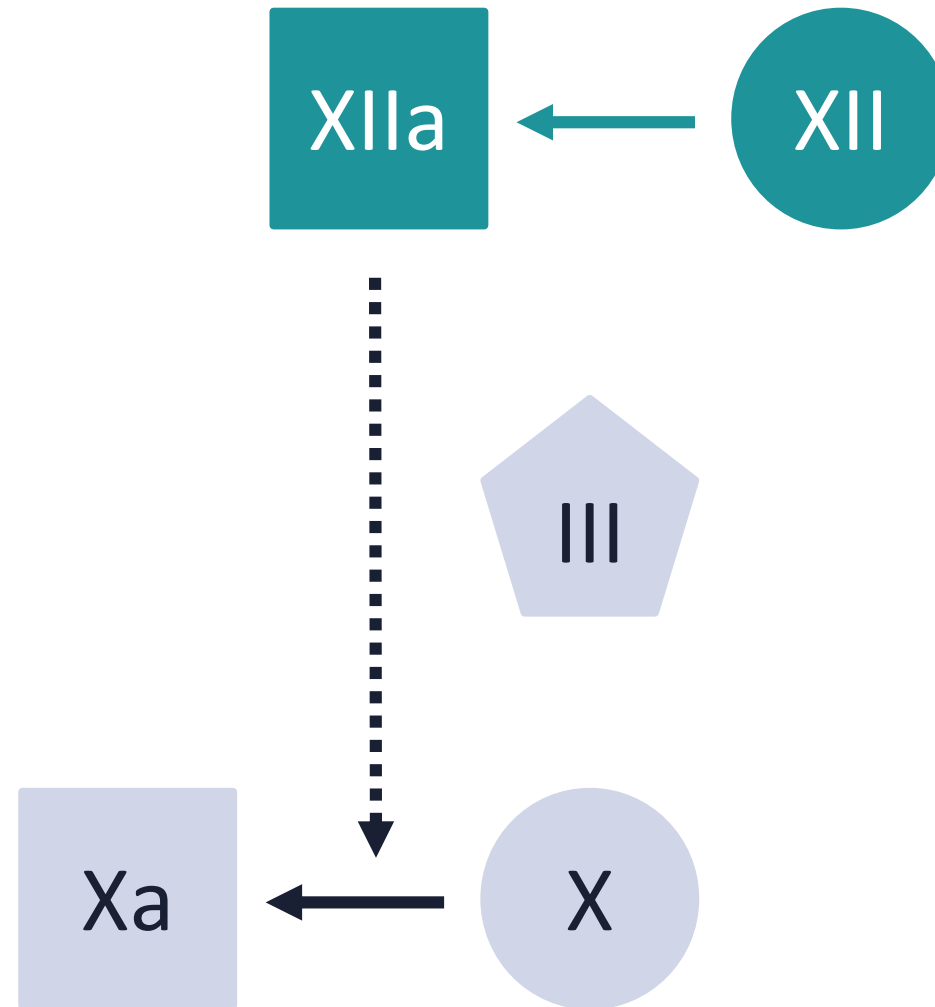
Intrinsic Pathway



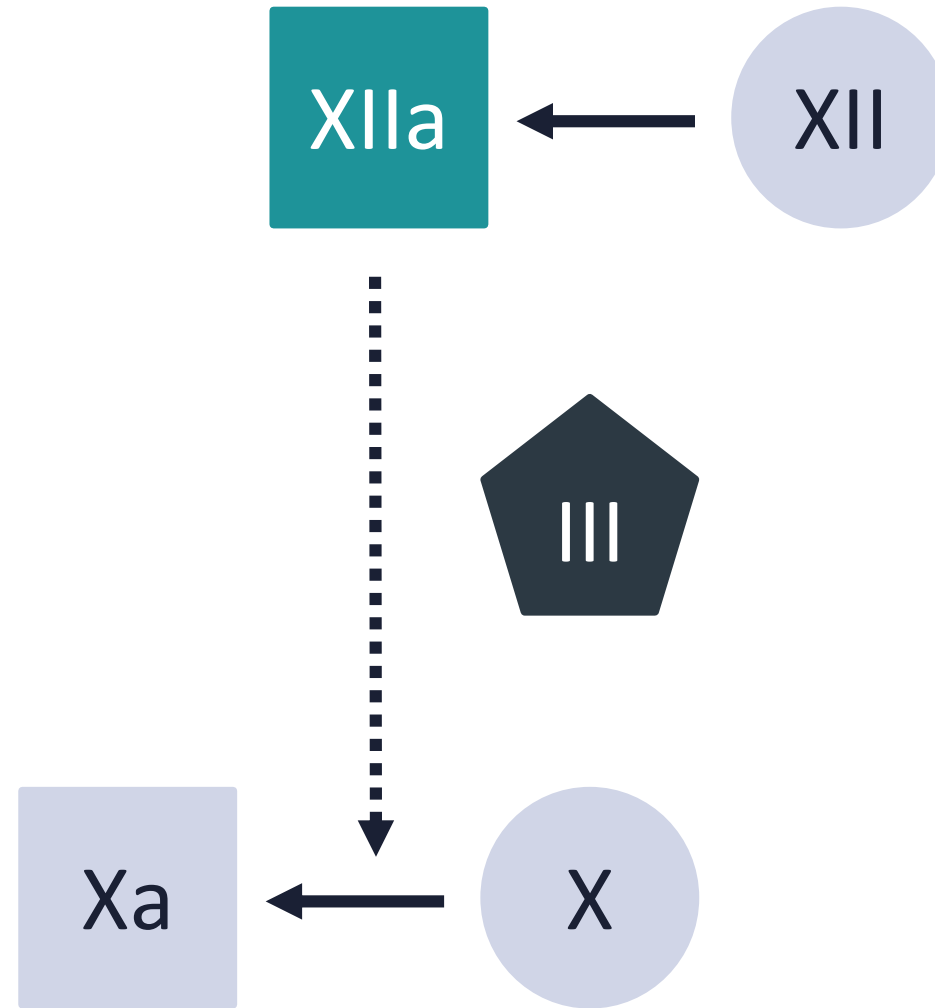
Extrinsic Pathway



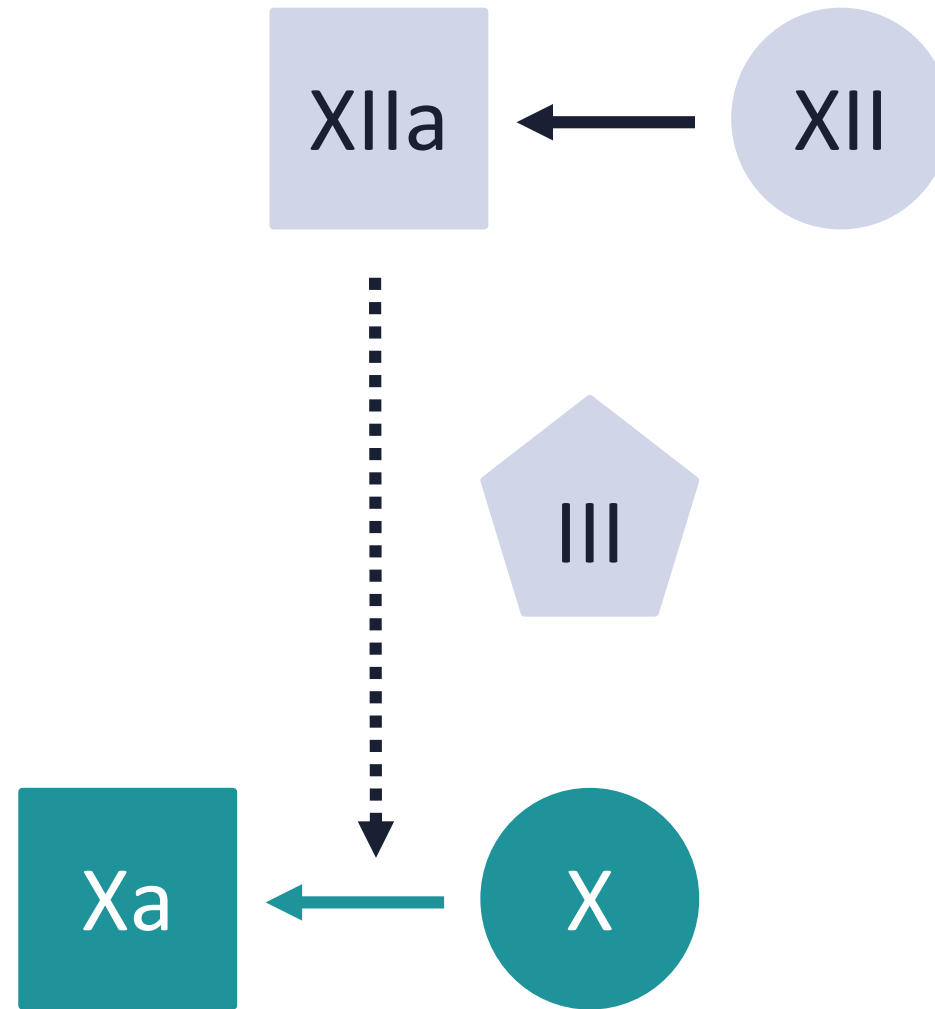
Extrinsic Pathway



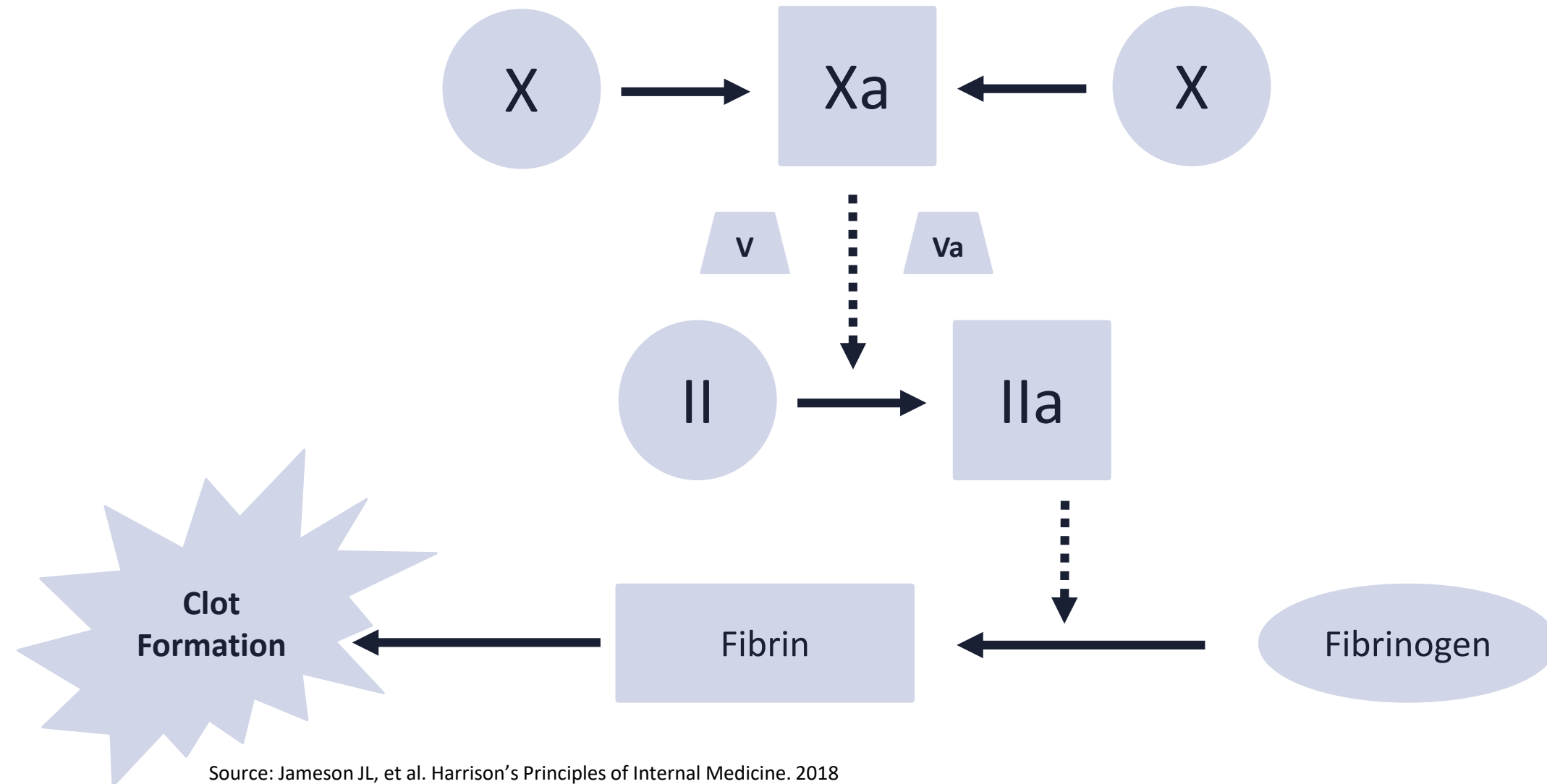
Extrinsic Pathway



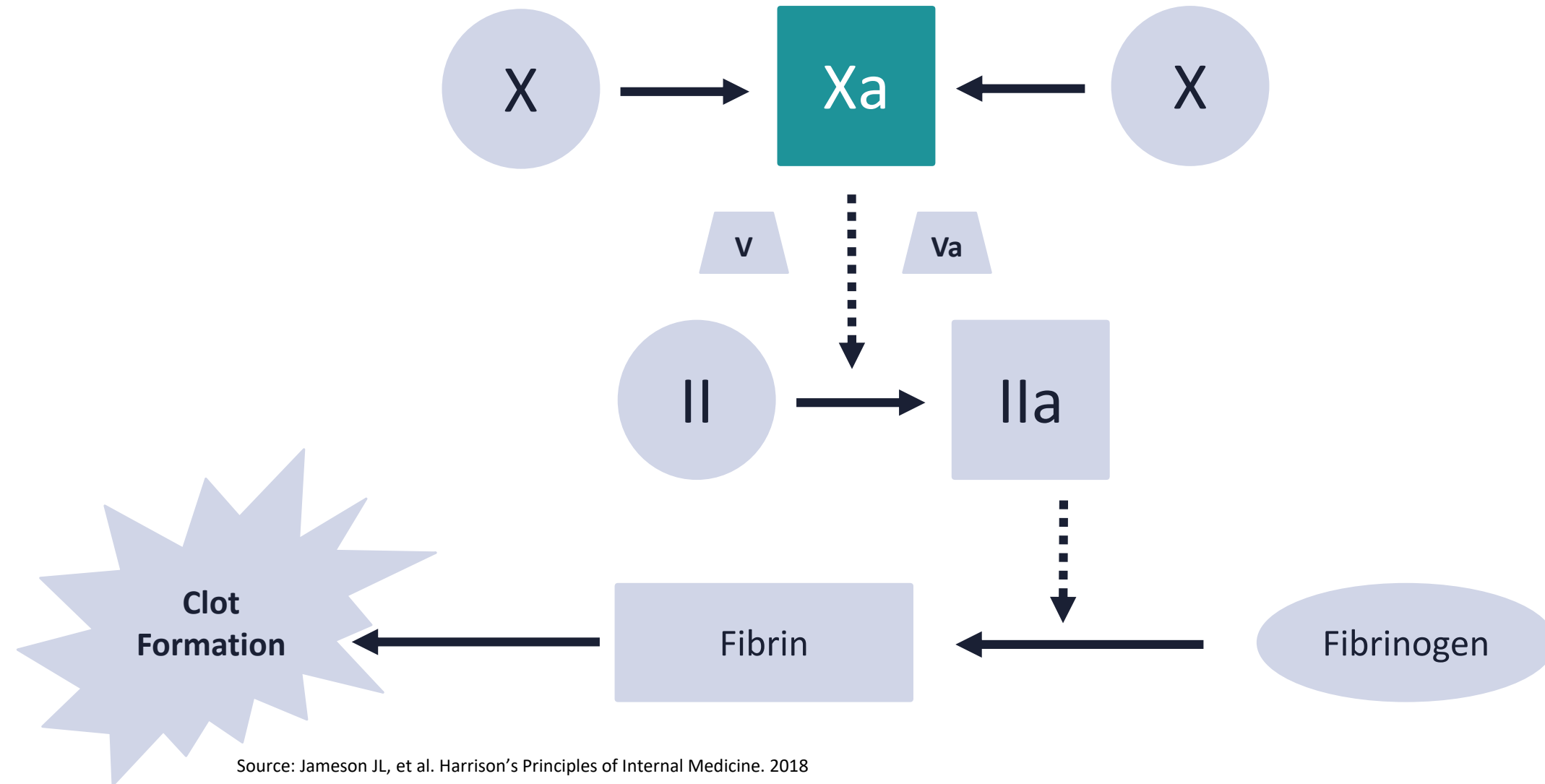
Extrinsic Pathway



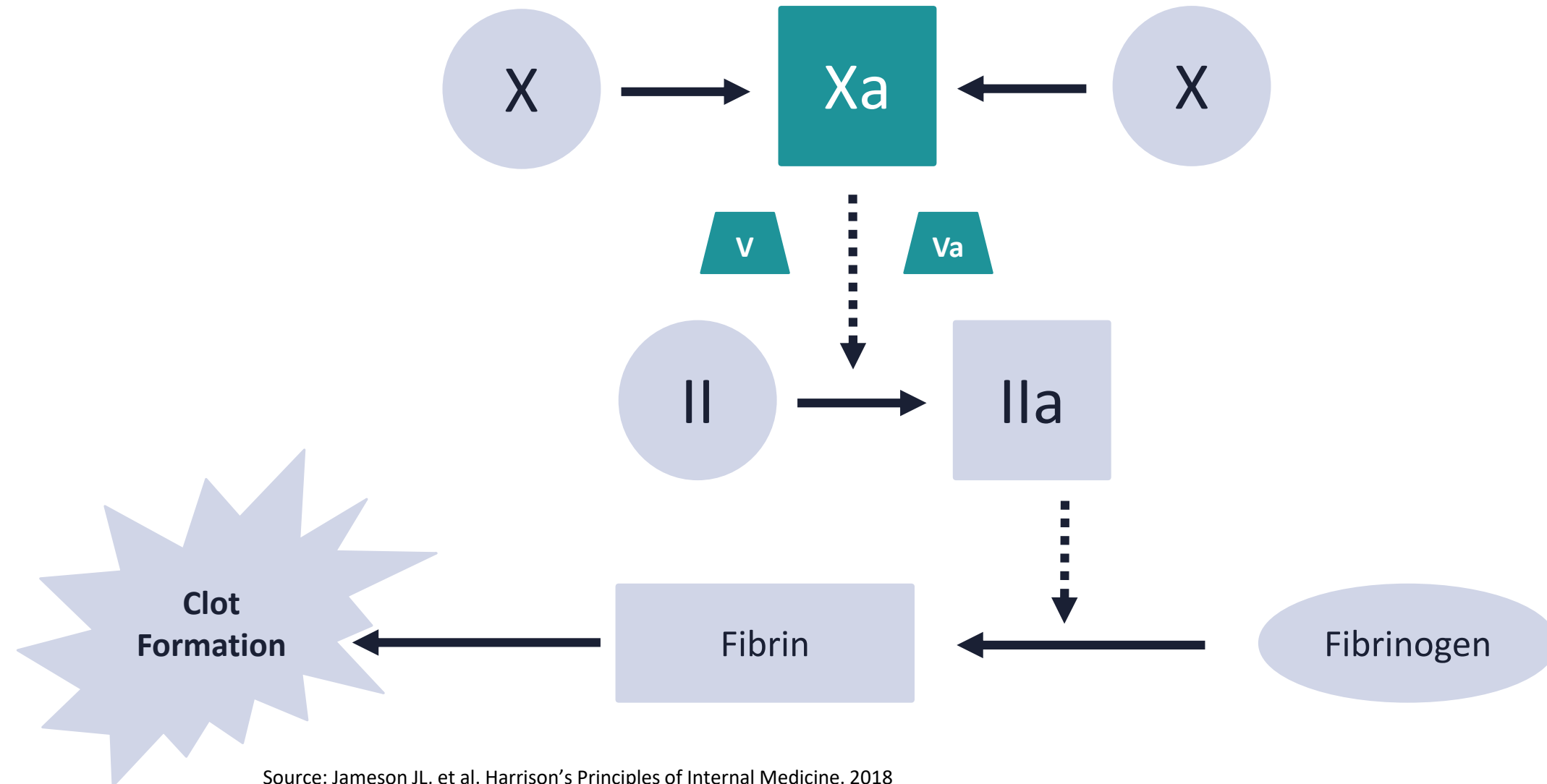
Common Pathway



Common Pathway

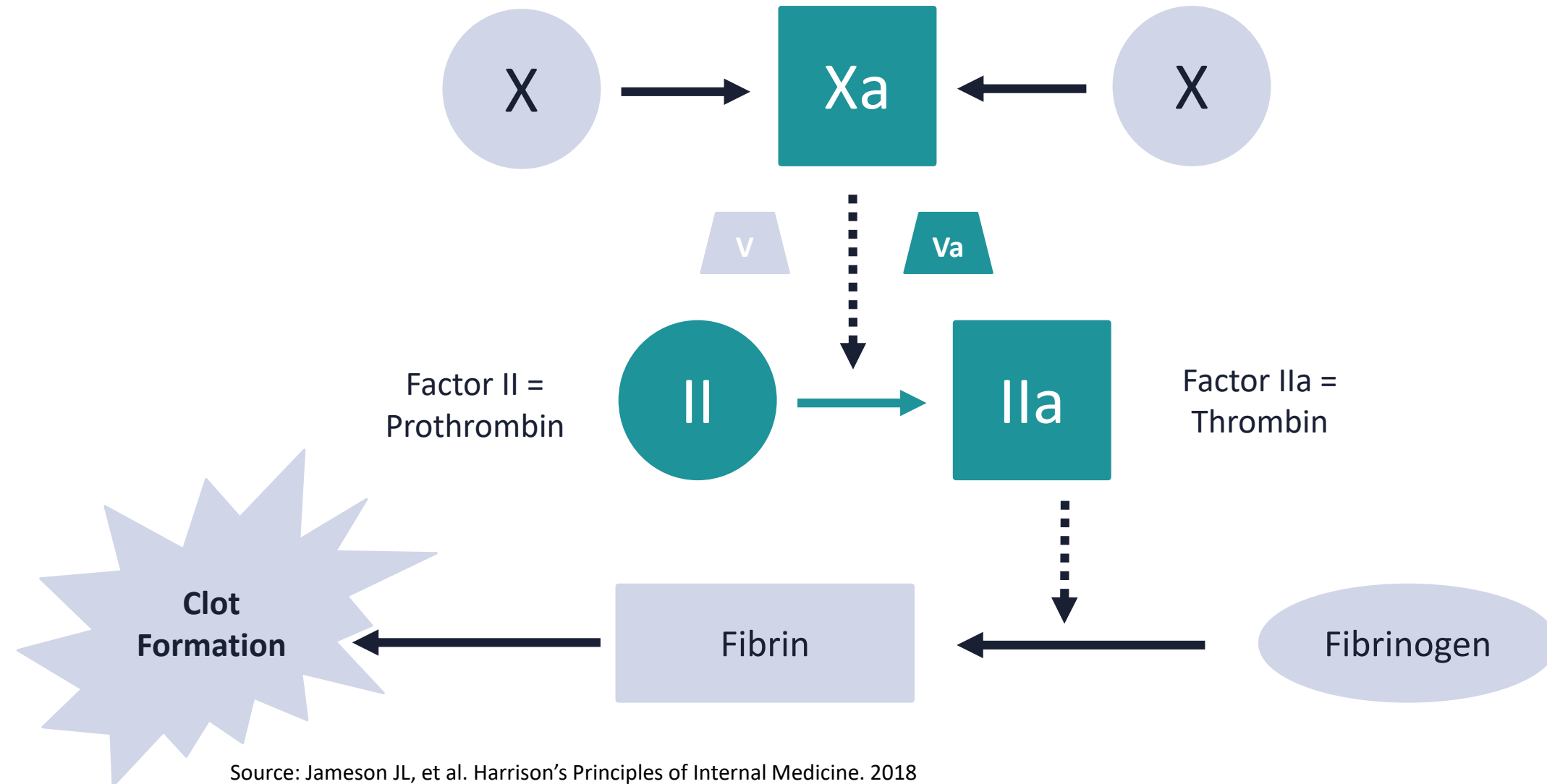


Common Pathway

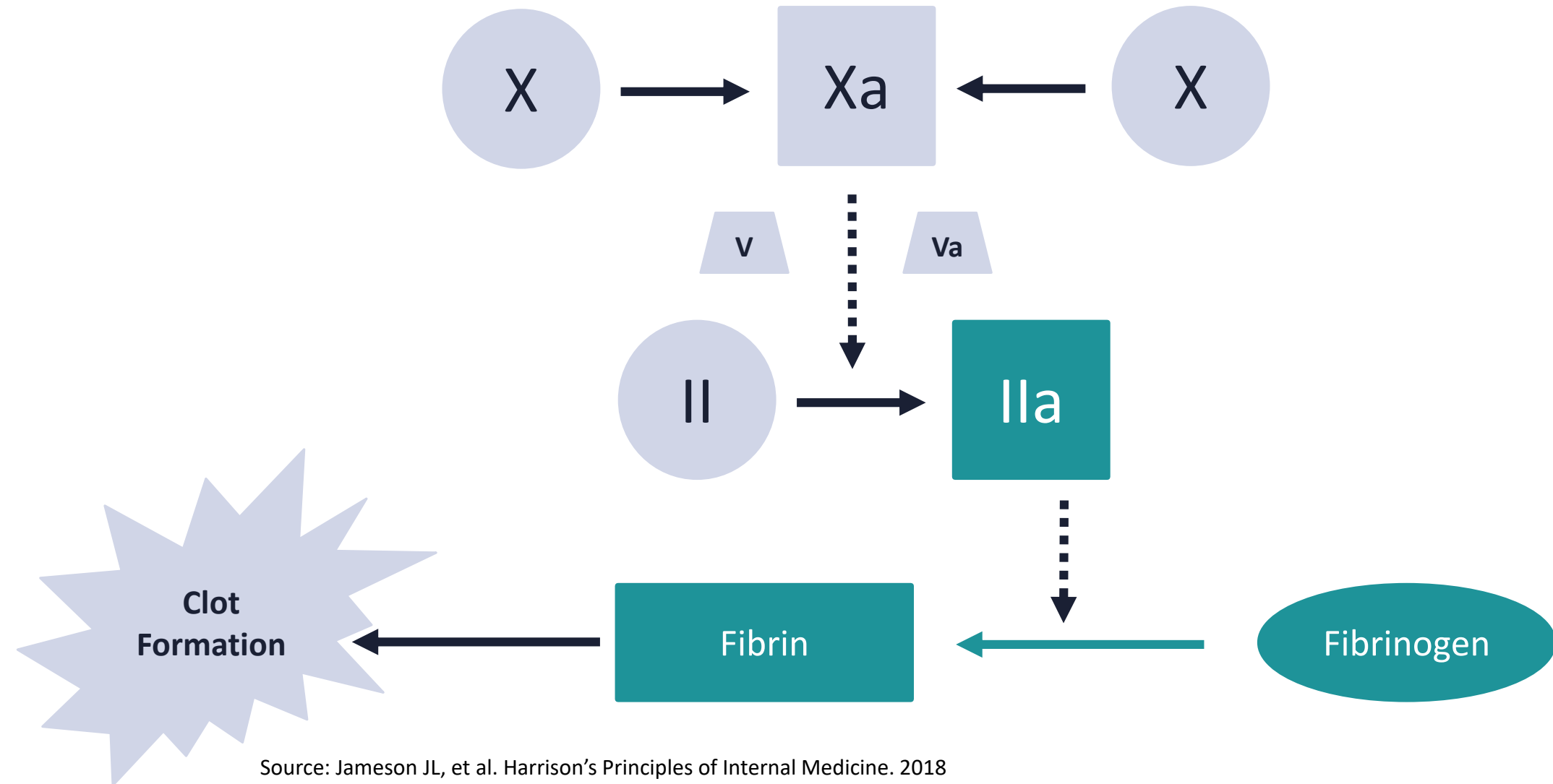


Source: Jameson JL, et al. Harrison's Principles of Internal Medicine. 2018

Common Pathway

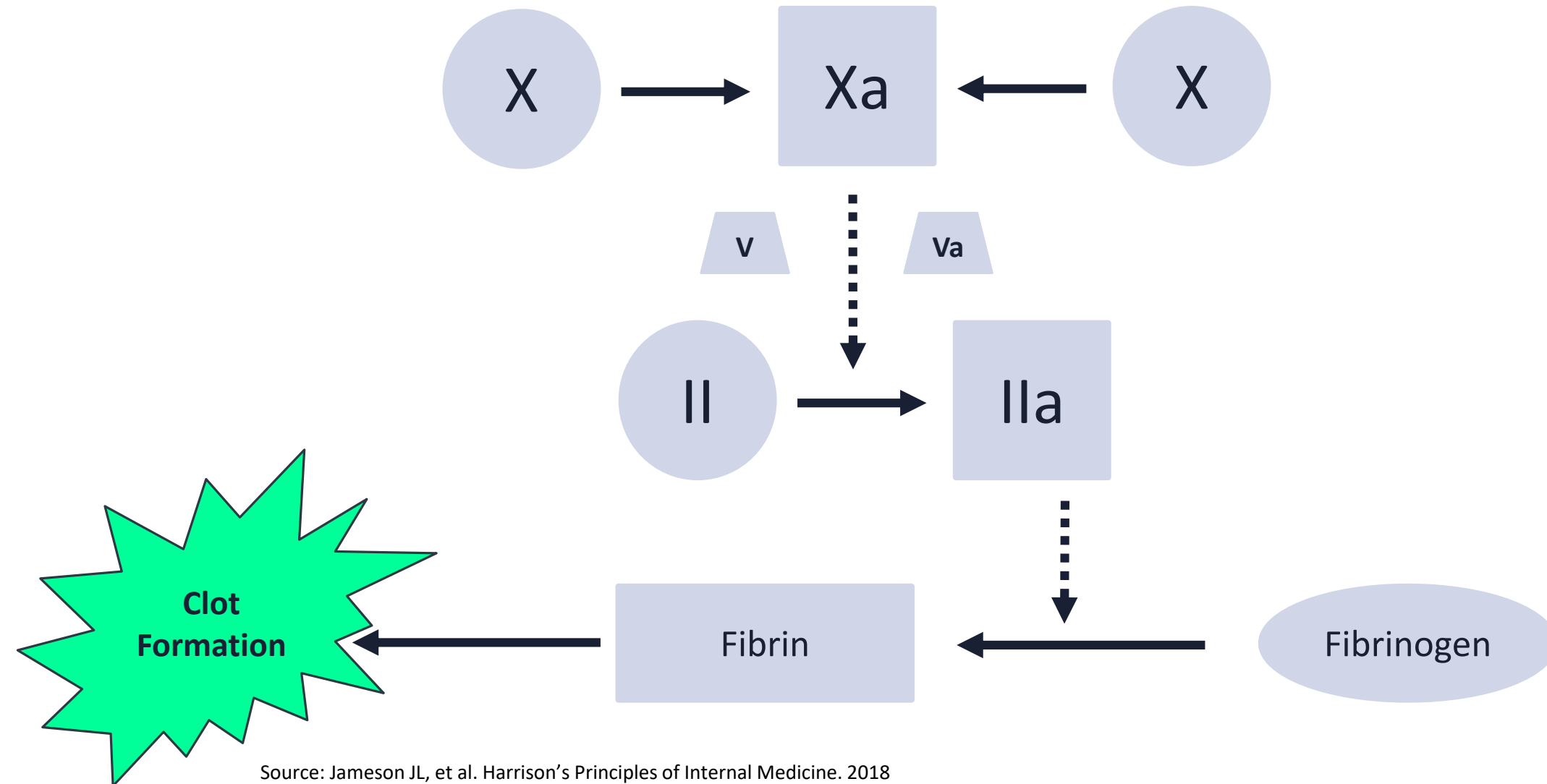


Common Pathway



Source: Jameson JL, et al. Harrison's Principles of Internal Medicine. 2018

Common Pathway



Source: Jameson JL, et al. Harrison's Principles of Internal Medicine. 2018

Thromboelastography (TEG)

AN INTRODUCTION

What is TEG?



How Does It Work?



Source: http://www.haemonetics.com/~link.aspx?_id=F761DCC0EE5248AA853B8C4BB317137A&_z=z

How Does It Work?



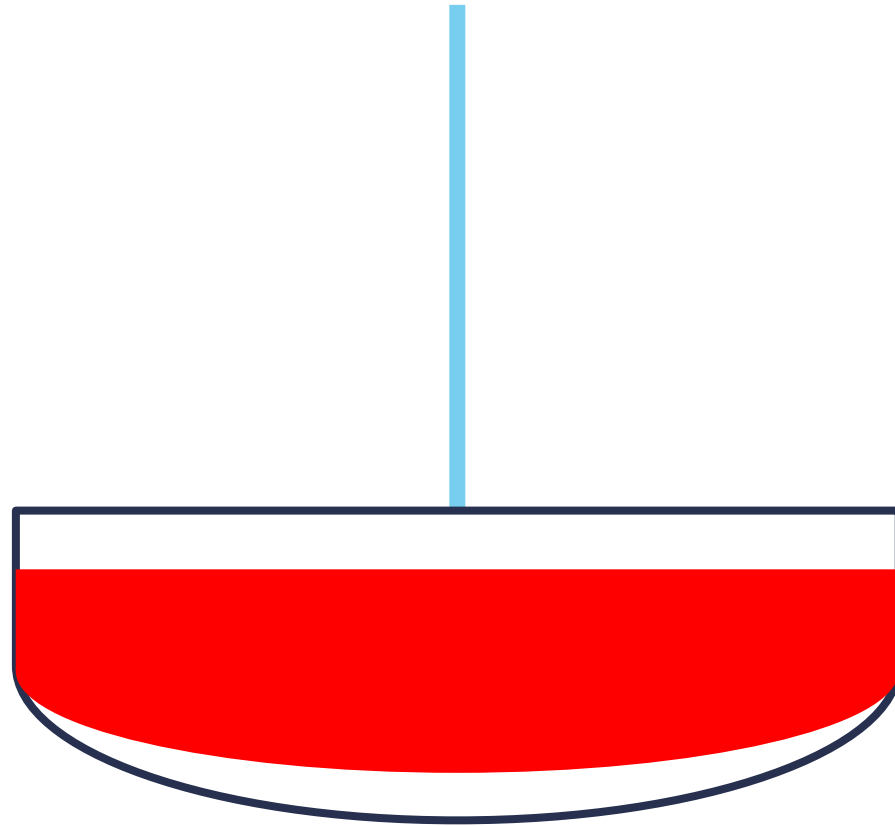
Source: http://www.haemonetics.com/~link.aspx?_id=F761DCC0EE5248AA853B8C4BB317137A&_z=z

How Does It Work?

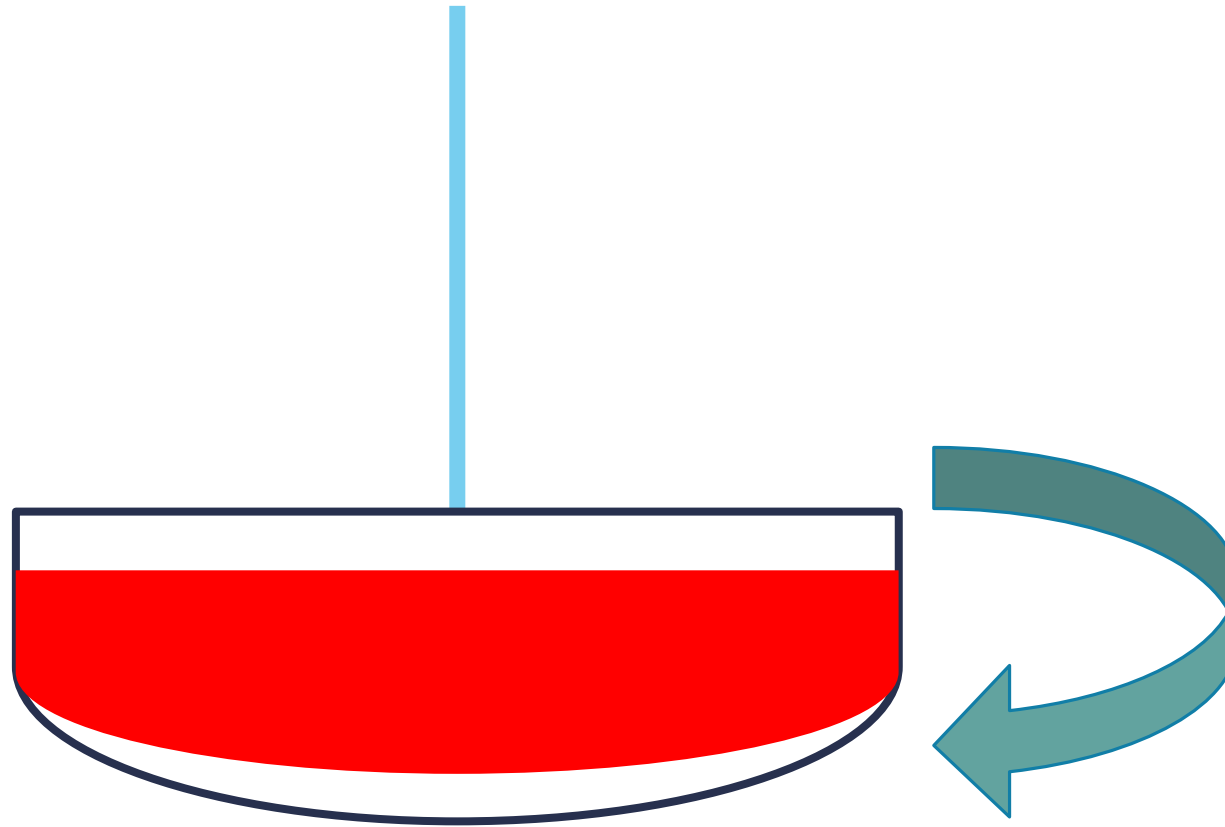


Source: http://www.haemonetics.com/~link.aspx?_id=F761DCC0EE5248AA853B8C4BB317137A&_z=z

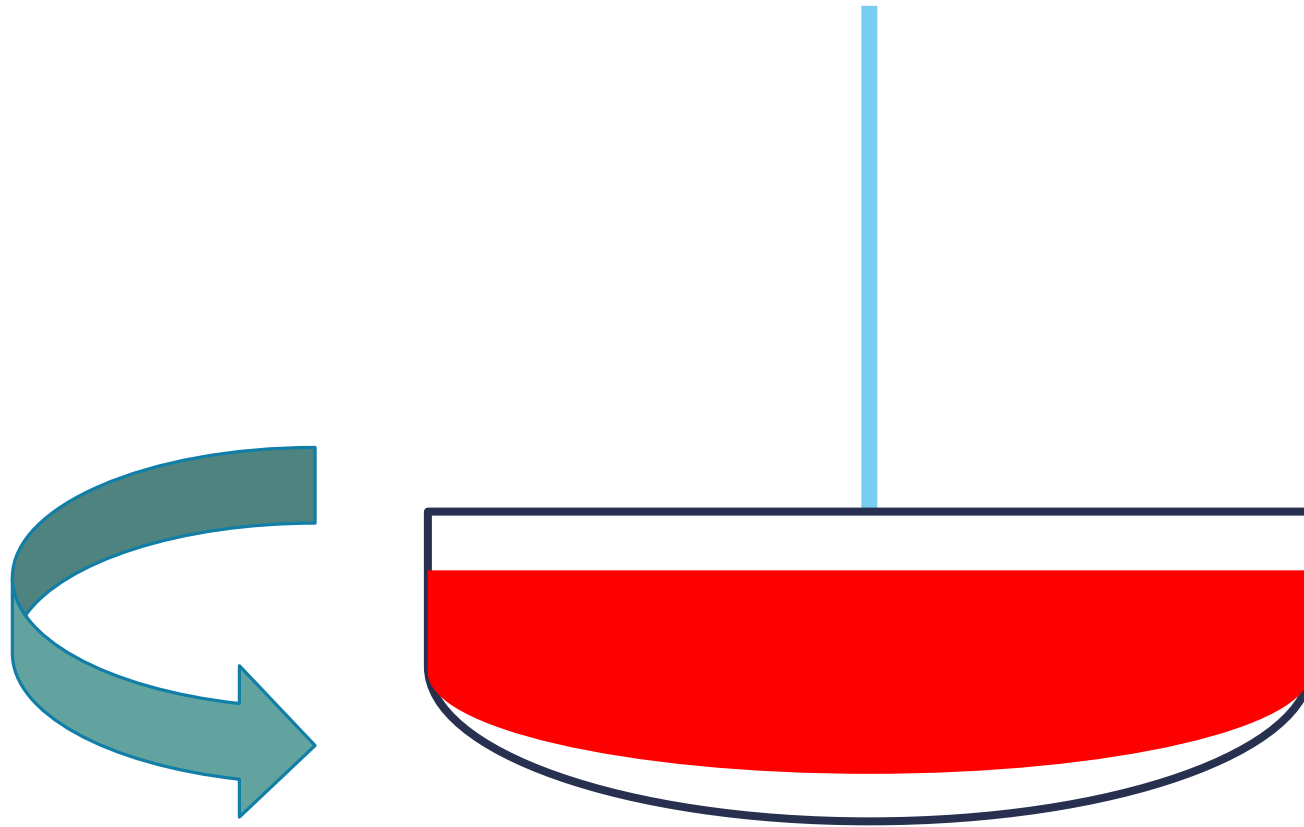
How Does It Work?

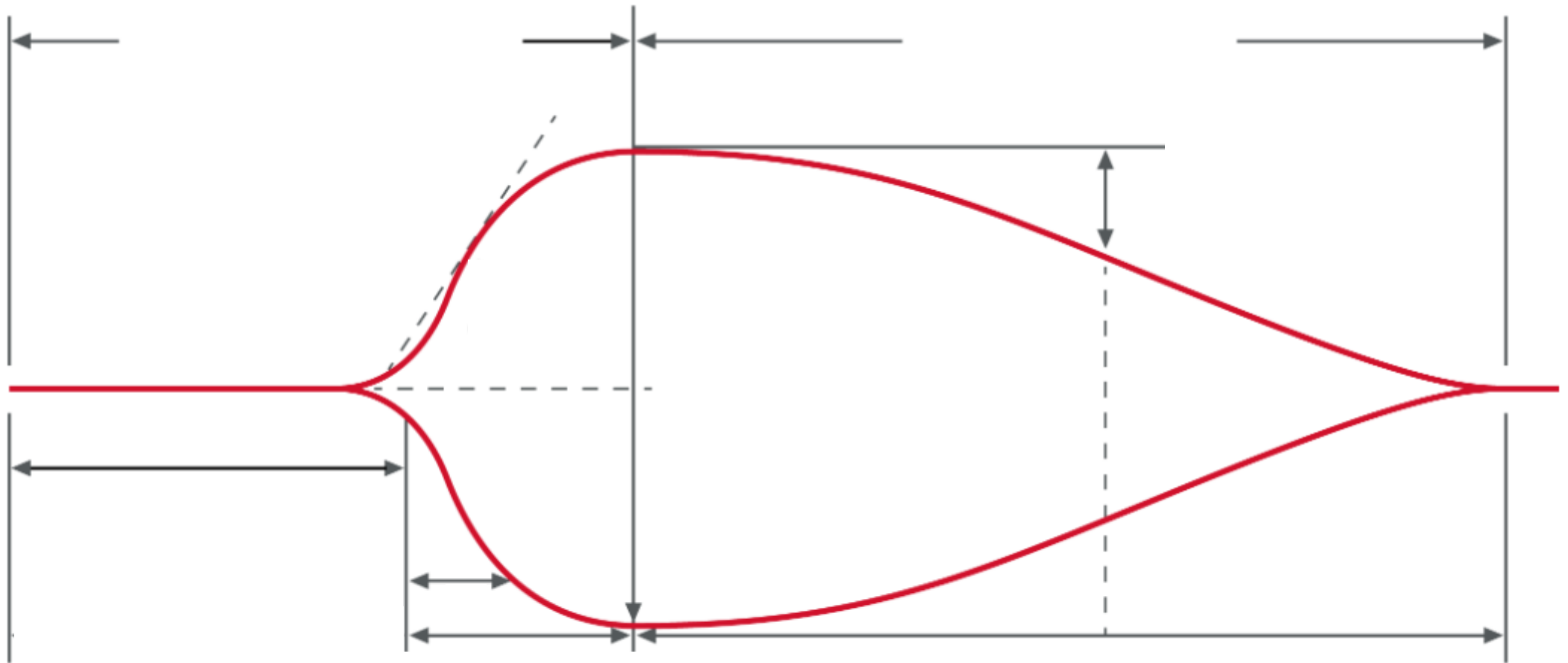


How Does It Work?

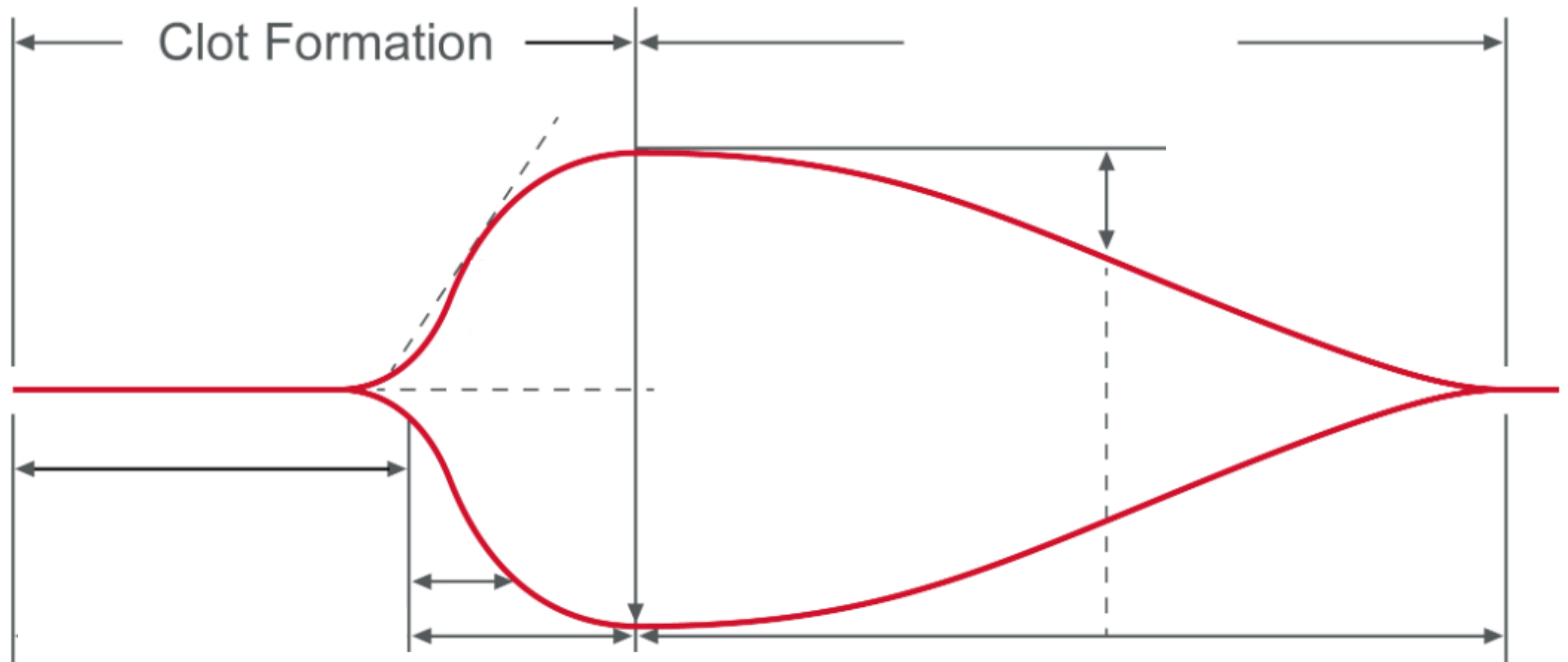


How Does It Work?

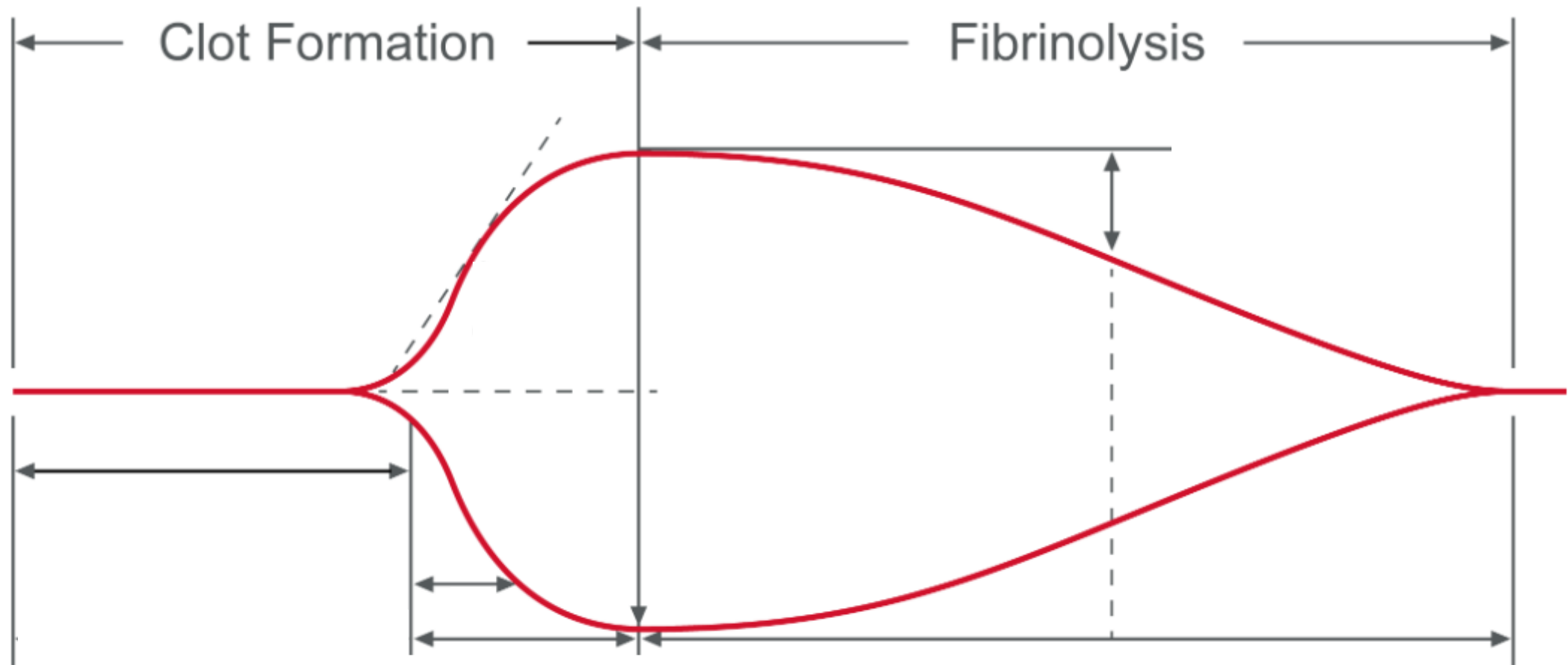




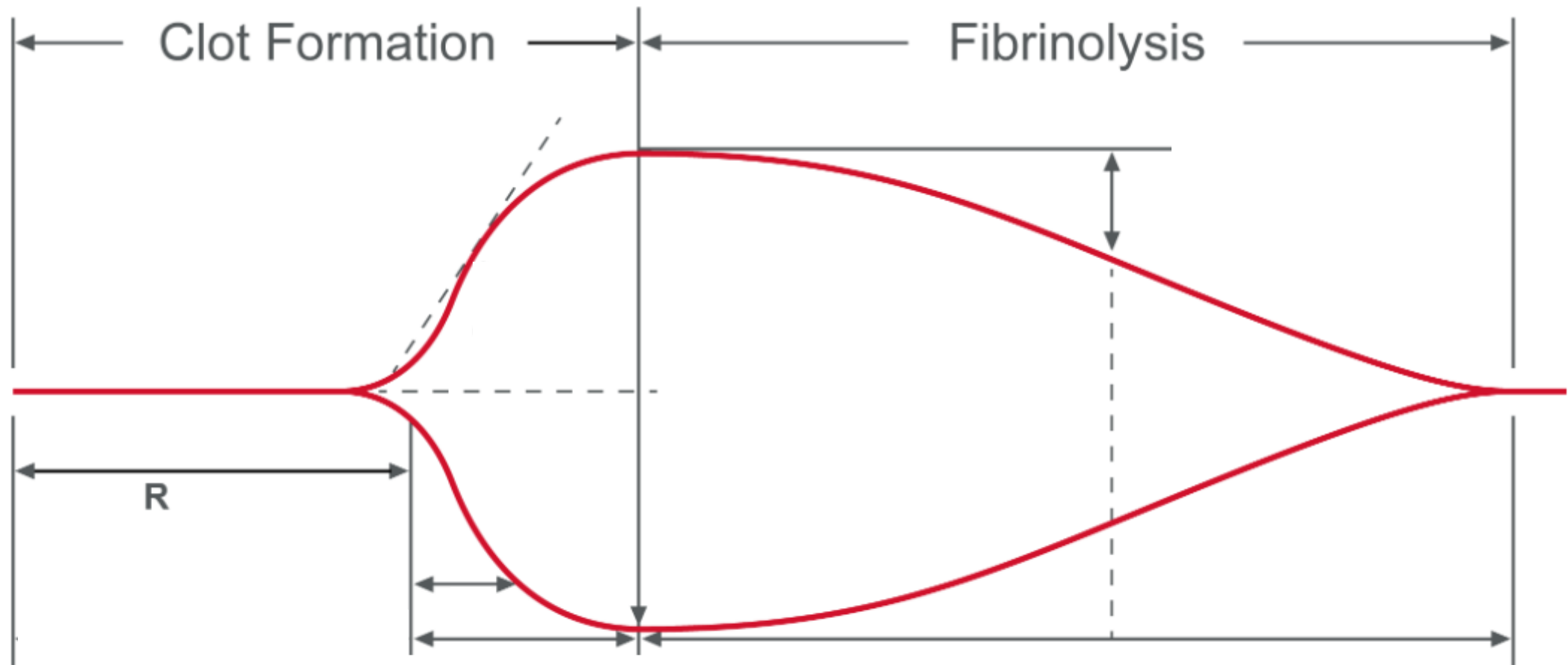
Source: <https://teg.haemonetics.com/en/teg-5000-thrombelastograph>



Source: <https://teg.haemonetics.com/en/teg-5000-thrombelastograph>

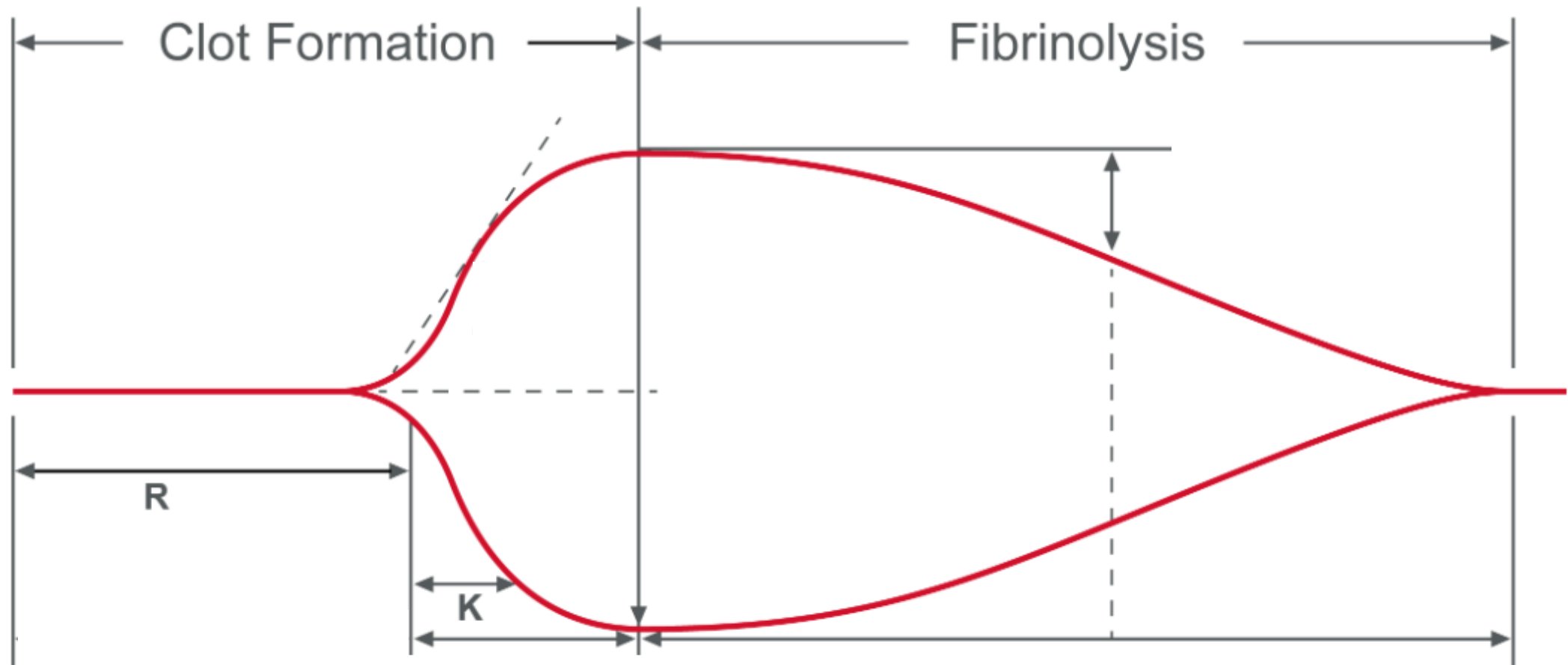


Source: <https://teg.haemonetics.com/en/teg-5000-thrombelastograph>

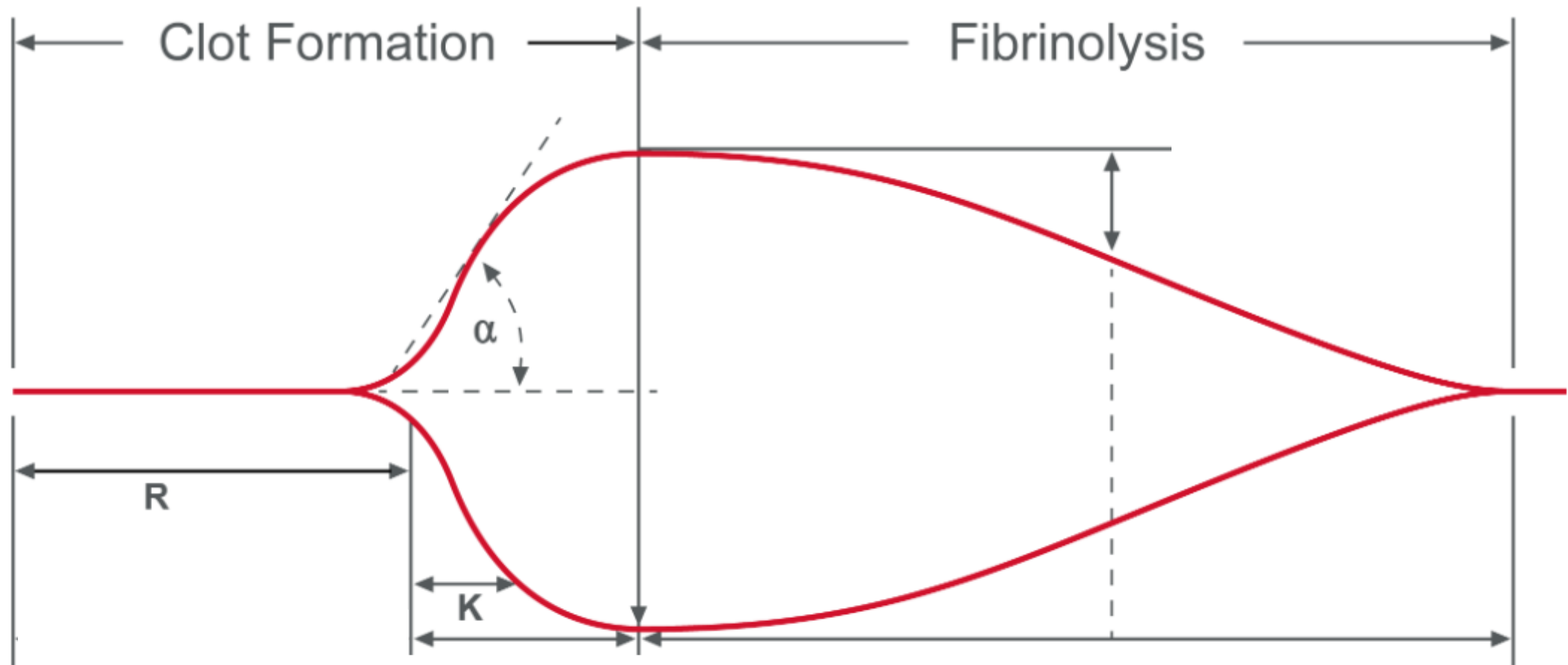


*Clotting Time
(Coagulation Factors)*

Source: <https://teg.haemonetics.com/en/teg-5000-thrombelastograph>

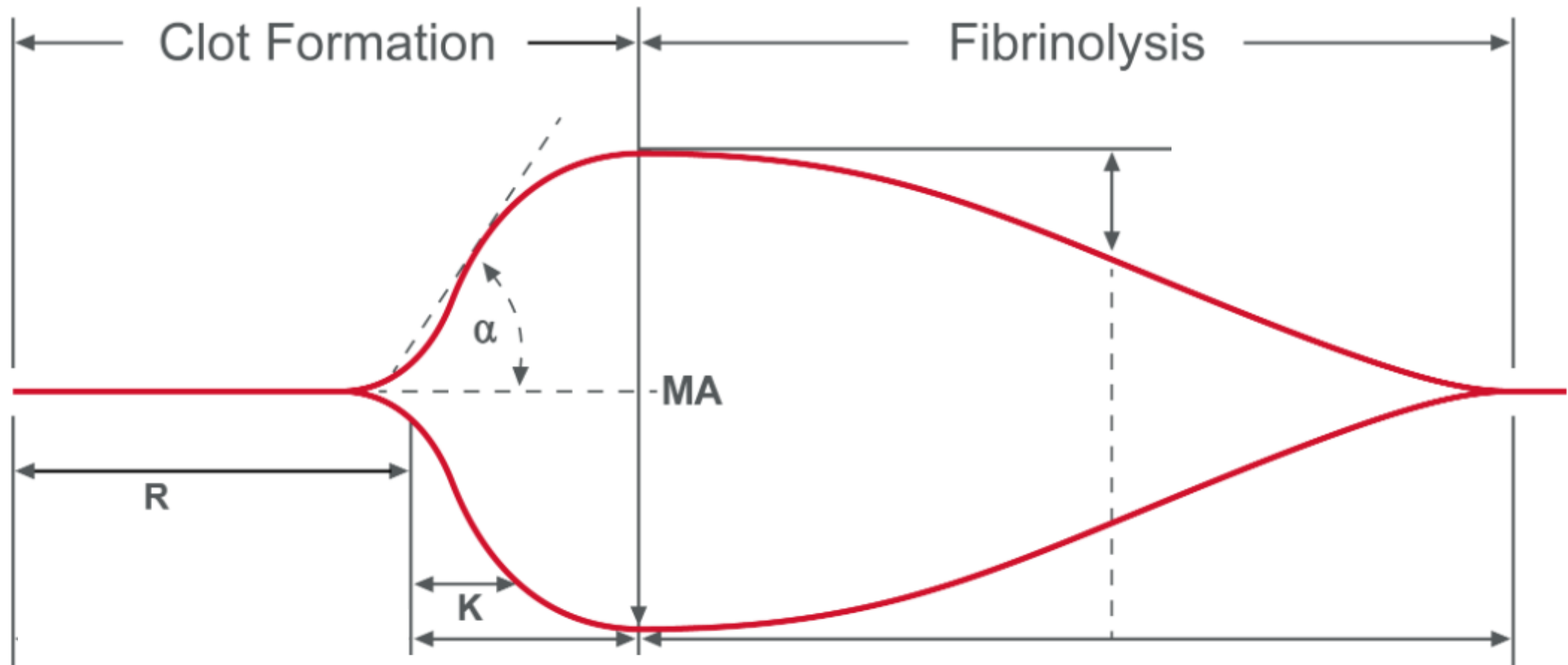


*Clotting Time
(Coagulation Factors)*



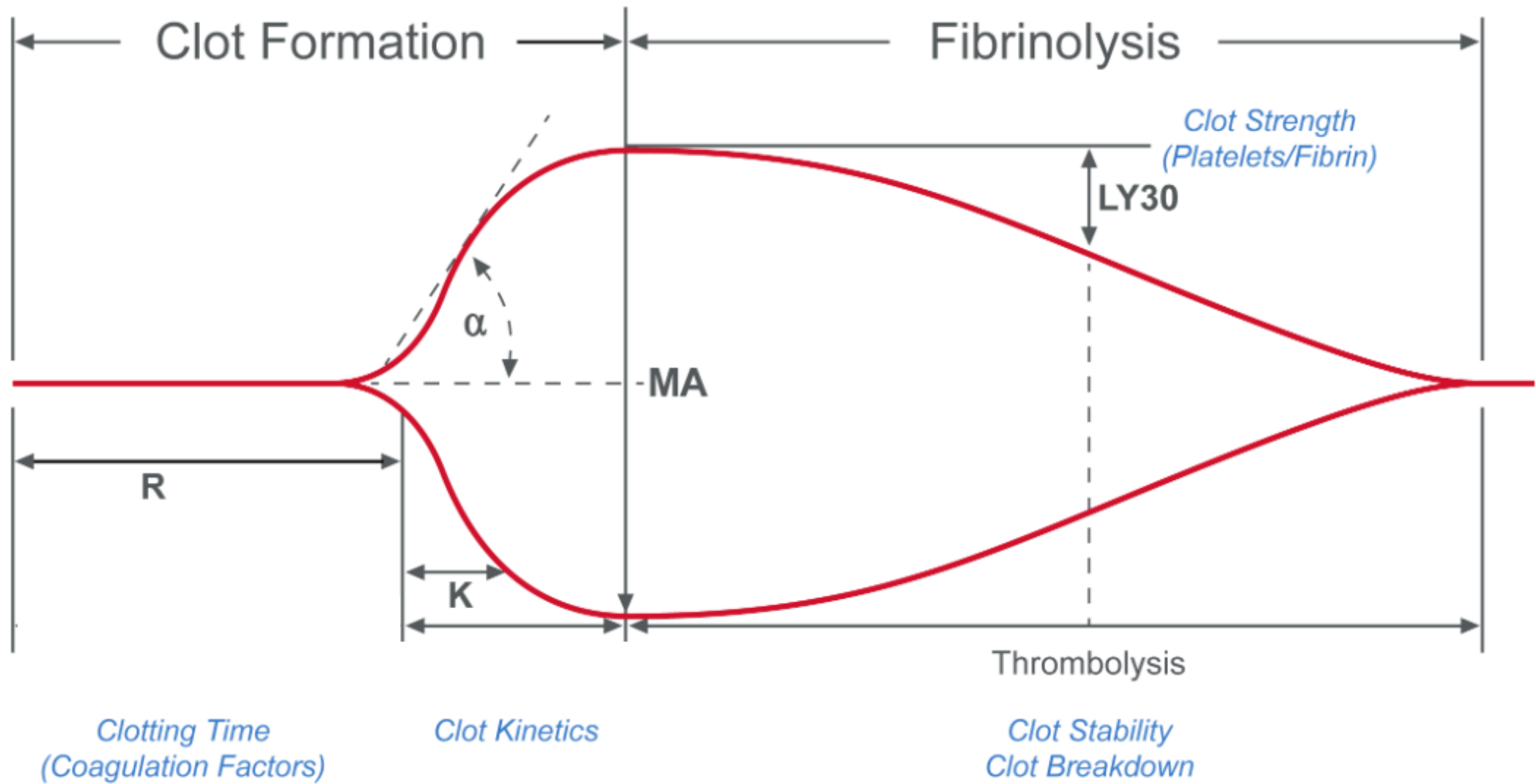
Clotting Time
(Coagulation Factors)

Source: <https://teg.haemonetics.com/en/teg-5000-thrombelastograph>

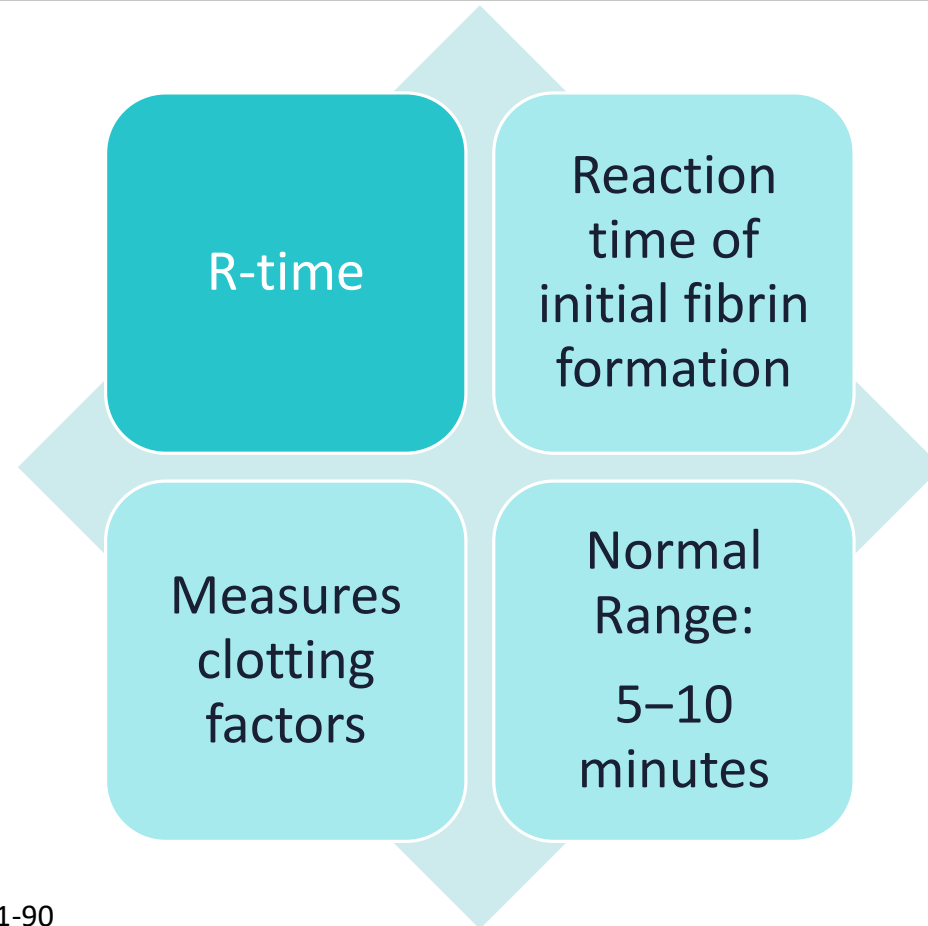


Clotting Time
(Coagulation Factors)

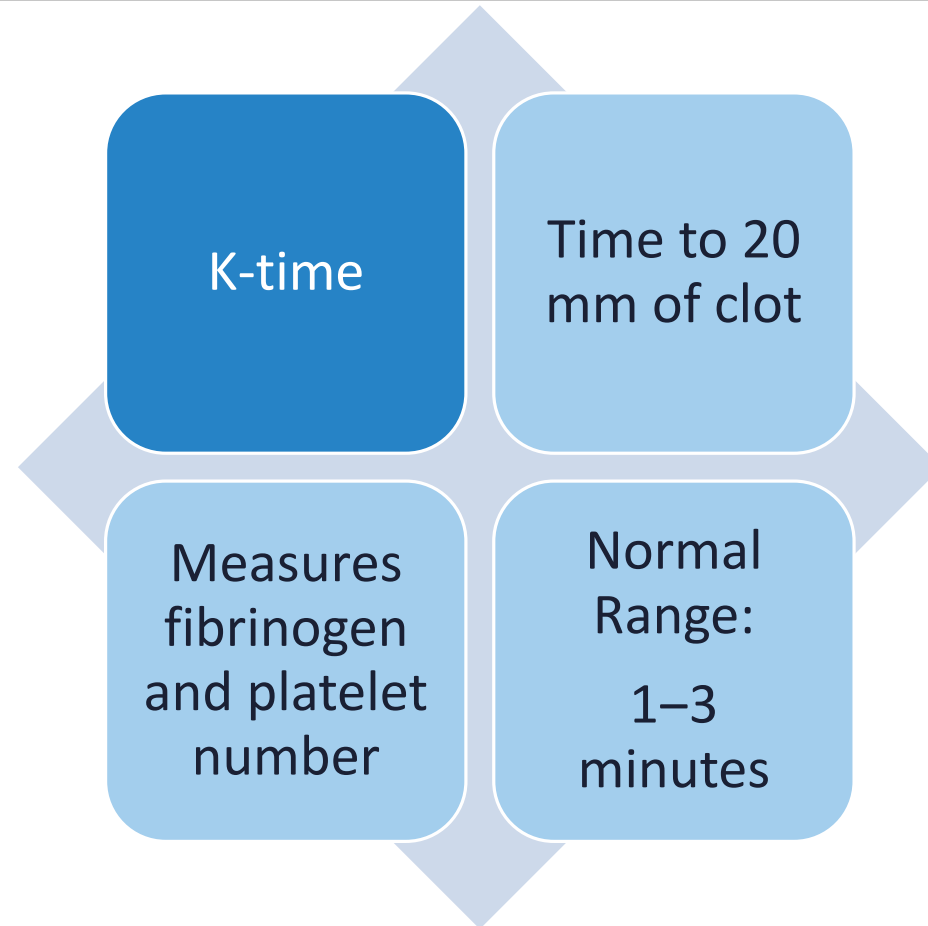
Clot Kinetics



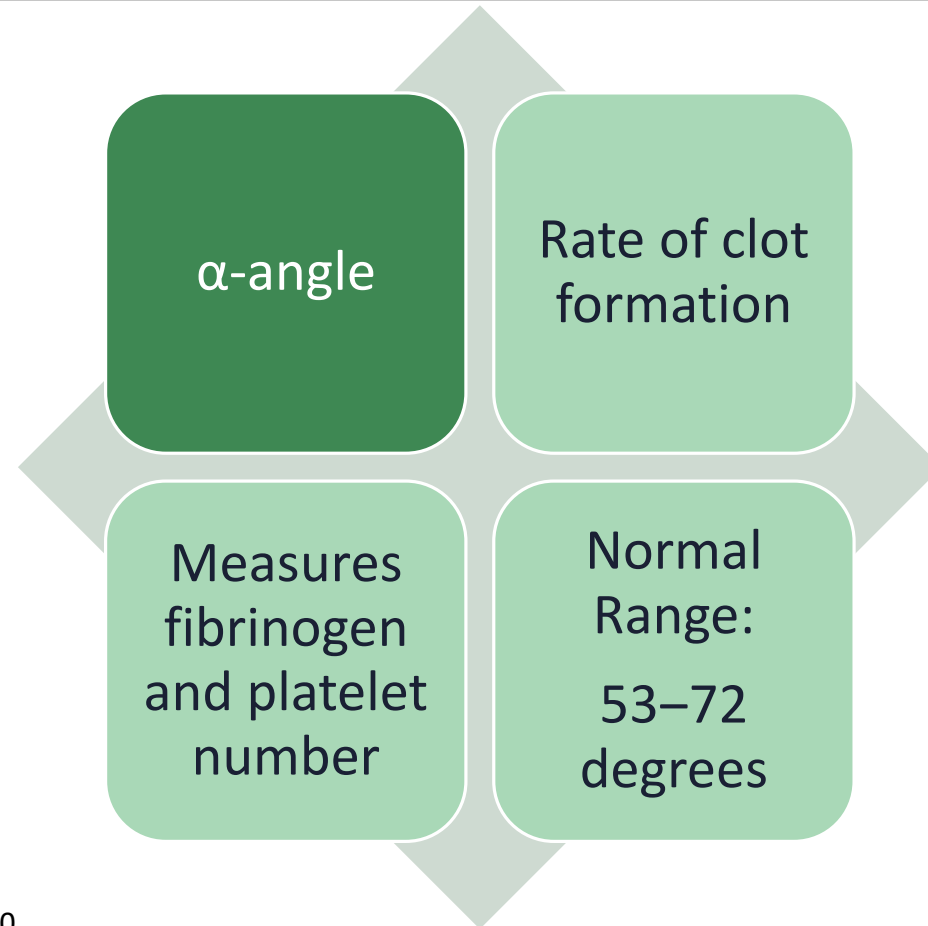
Components of TEG Tracing



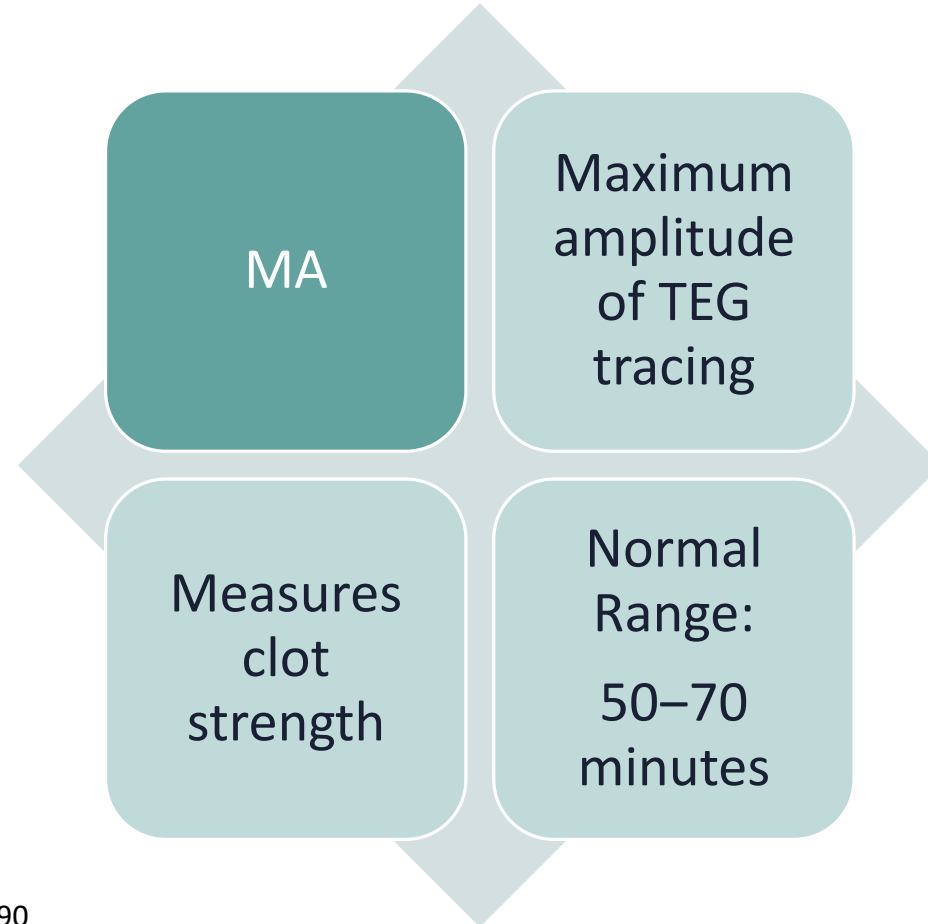
Components of TEG Tracing



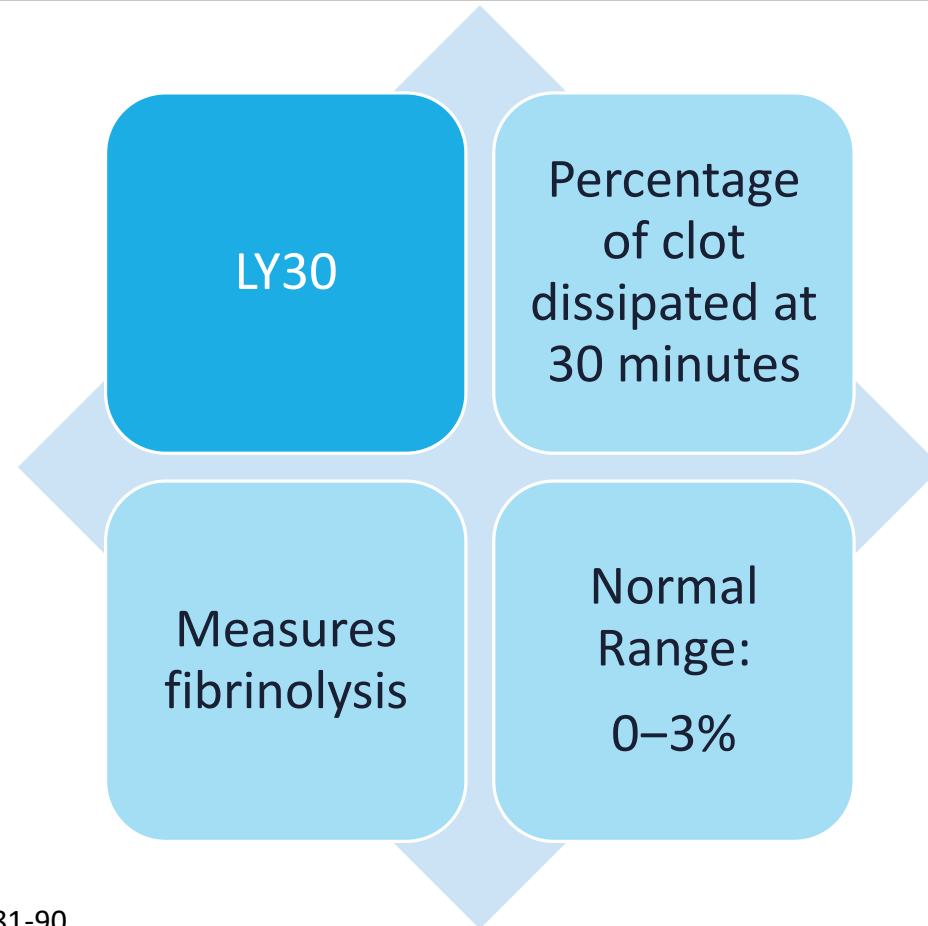
Components of TEG Tracing



Components of TEG Tracing



Components of TEG Tracing



TEG-guided Transfusion

TEG Value	Transfusion Recommendation
R-time >10	Fresh frozen plasma (FFP)
K-time >3	Cryoprecipitate
α -angle <53	Cryoprecipitate +/- platelets
MA <50	Platelets
LY30 >3%	Tranexamic acid (TXA)

Assessment Question #1

Which of the following represents a patient that is in a hypocoagulable state?

- A. ↑ R-time, ↑ MA, ↓ LY30
- B. ↓ R-time, ↑ MA, ↓ LY30
- C. ↓ R-time, ↓ MA, ↑ LY30
- D. ↑ R-time, ↓ MA, ↑ LY30

Assessment Response #1

Which of the following represents a patient that is in a hypocoagulable state?

- A. ↑ R-time, ↑ MA, ↓ LY30
- B. ↓ R-time, ↑ MA, ↓ LY30
- C. ↓ R-time, ↓ MA, ↑ LY30
- D. ↑ R-time, ↓ MA, ↑ LY30

The Role of TEGs

The Role of TEGs



The Role of TEGs

Hemostatic
Monitoring in Liver
Disease

The Role of TEGs

Hemostatic
Monitoring in Liver
Disease

TEG-guided
Transfusion in
Cardiac Surgery

The Role of TEGs

Hemostatic
Monitoring in Liver
Disease

TEG-guided
Transfusion in
Cardiac Surgery

Goal-Directed
Transfusion
Strategy in Trauma
Patients



The Role of TEGs

Hemostatic
Monitoring in Liver
Disease

TEG-guided
Transfusion in
Cardiac Surgery

Goal-Directed
Transfusion
Strategy in Trauma
Patients

Monitoring of
Direct Oral
Anticoagulants
(DOACs)

Hemostatic Monitoring in Liver Disease

Liver Disease & Hemostatic Function

Procoagulant and Anticoagulant Proteins

- Decreased synthesis of factors II, VII, IX, X
- Decreased synthesis of protein C and S

Platelet Defects

- Decreased platelet adherence

Hyperfibrinolysis

- Decreased fibrinogen concentrations

Minimal Effects of Liver Injury on Hemostasis Assessed by TEG

Bleeding diathesis
based on elevated
INR

Assess overall
hemostasis

Prospective study
of 51 patients

Mean INR was 3.4

Mean TEG
parameters were
normal

Normal
coagulation
parameters
maintained

TEG-Guided Transfusion in Cardiac Surgery

Cardiac Surgery & Post-Operative Bleeding

Use of Anticoagulants

- Heparin and extracorporeal circulation: prevent coagulation

Extracorporeal Circuit Associated Trauma

- Decreased platelet number and function

Hypothermia

- Further impairs platelet function

Changes in Transfusion Therapy After Institution of Blood Management Program

Impact of
coagulation and
transfusion
management

Retrospective study
of 1,079 patients

Transfusion
requirements, donor
exposure, incidence
of reoperation

Lower incidence of
overall transfusion

Lower incidence of
reoperation

TEG-Guided
transfusion may
decrease transfusion
requirements

Goal-Directed Transfusion in Trauma

Trauma Induced Coagulaopathy (TIC)

Increased Activation of Coagulation

- Uncontrolled coagulation

Coagulation Factor Deficiency

- Coagulation impairment leading to hypocoagulability

Increased fibrinolysis

- Can lead to catastrophic bleeding

TEG-guided Massive Transfusion Protocols (MTP)

TEG-Guided MTP

Fixed Transfusion
Ratio 1:1:1

TEG-Guided MTP:
improved mortality

TEG-guided MTP
vs. Conventional
coagulation assays
(CCA)

TEG-guided MTP
improved mortality
(19% vs. 36%)

CCA: plasma and
platelet units vs.
TEG-guided:
cryoprecipitate

Sources: Taipia NM, et al. J Trauma Acute Care Surg. 2013;74(2):378-385
Gonzalez E, et al. Crit Care Clin. 2017;33(1): 119-134

TEG-guided Characterization of TIC Phenotypes

Hyperfibrinolysis

- Hemorrhagic phenotype

Hypofibrinolysis

- Fibrinolytic shutdown

Physiologic fibrinolysis

Monitoring of DOACs

Direct Oral Anticoagulants (DOACs)

Management Challenges

No Reliable Methods to Detect and Monitor

Limited Antidotes for Reversal

Sources: Iapichon GE, et al. Semin Throm Hemost. 2017;43:423-432

Dias JD, et al. Arch Pathol Lab Med. 2015;139:665-673

Use of TEG for Detection of New Oral Anticoagulants

Utilization of TEG

Monitor and
differentiate
between DOACs

Direct Thrombin
Inhibitors & Factor
Xa Inhibitors

R-time and MA
were prolonged for
apixaban and
dabigatran

Capable of
detecting and
differentiating
DOACS

Valuable tool to
analyze hemostasis
and effectiveness
of reversal

Patient Case

RD is a 18-year old male who was involved in an MVC vs. pedestrian trauma. He was stabilized at an outside hospital and sent to your hospital for further evaluation. The trauma surgeon has the results of the patient's TEG from the outside hospital and asks for your recommendation on transfusion for the patient. The patient's TEG results are below:

R-time: 7

K-time: 1

α -angle:
78

MA: 84

LY30: 10%

Assessment Question #2

What can you conclude from RD's TEG analysis?

- A. His R-time indicates that his time to clot initiation is prolonged
- B. His K-time indicates that his time to 20 mm clot is prolonged
- C. His MA indicates that his clot strength is minimal
- D. His LY30 indicates that he is in a fibrinolytic state

Assessment Response #2

What can you conclude from RD's TEG analysis?

- A. His R-time indicates that his time to clot initiation is prolonged
- B. His K-time indicates that his time to 20 mm clot is prolonged
- C. His MA indicates that his clot strength is minimal
- D. His LY30 indicates that he is in a fibrinolytic state

Assessment Question #3

What pharmacologic therapy would you recommend for RD?

- A. Tranexamic acid
- B. Platelets
- C. Cryoprecipitate
- D. RD does not require any pharmacologic intervention

Assessment Response #3

What pharmacologic therapy would you recommend for RD?

- A. Tranexamic acid
- B. Platelets
- C. Cryoprecipitate
- D. RD does not require any pharmacologic intervention

References

1. Jameson JL, et al. Harrison's Principles of Internal Medicine. Accessed April 29, 2019
2. Luddington RJ. Thromboelastography/thromboelastometry. Clin Lab Haem. 2005;27:81-90
3. Stravitz RT, Lisman T, Luketic VA, et al. Minimal effects of acute liver injury/acute liver failure on hemostasis as assessed by thromboelastography. J Hepatol. 2012;56(1):129-136
4. Spiess BD, Gillies BS, Chandler W, et al. Changes in transfusion therapy and reexploration rate after institution of a blood management program in cardiac surgical patients. J Cardiothoracic Vasc Anesth. 1995;9(2):168-173
5. Kushimoto S, Kudo D, and Kawazoe Y. Acute traumatic coagulopathy and trauma-induced coagulopathy: an overview. J Intensive Care. 2017;5(6):1-7
6. Tapia NM, Chang A, Norman M, et al. TEG-guided resuscitation is superior to standardized MTP resuscitation in massively transfused penetrating trauma patients. J Trauma Acute Care Surg. 2013;74(2):378-385
7. Gonazlez E, Moore EE, Moore HB. Management of trauma induced coagulopathy with thromboelastography. Crit Care Clin. 2017;33(1):119-134
8. Walsh M, Thomas S, Moore E, et al. Tranexamic acid for trauma resuscitation in the United States of America. Semin Thromb Hemost. 2017;43:213-223
9. Iapichino GE, Bianchi P, Ranucci M, et al. Point-of-care coagulation tests monitoring of direct oral anticoagulants and their reversal therapy: state of the art. Semin Thromb Hemost. 2017;43:423-432
10. Dias JD, Norem K, Doorneweerd DD, et al. Use of thromboelastography (TEG) for detection of new oral anticoagulants. Arch Pathol Lab Med. 2015;139:665-673

Thromboelastography (TEG) & Its Role in Determining a Patient's Coagulation Status

Thank You!

Alyssa Sonchaiwanich, PharmD

Memorial Hospital of South Bend

asonchaiwanich@beaconhealthsystem.org