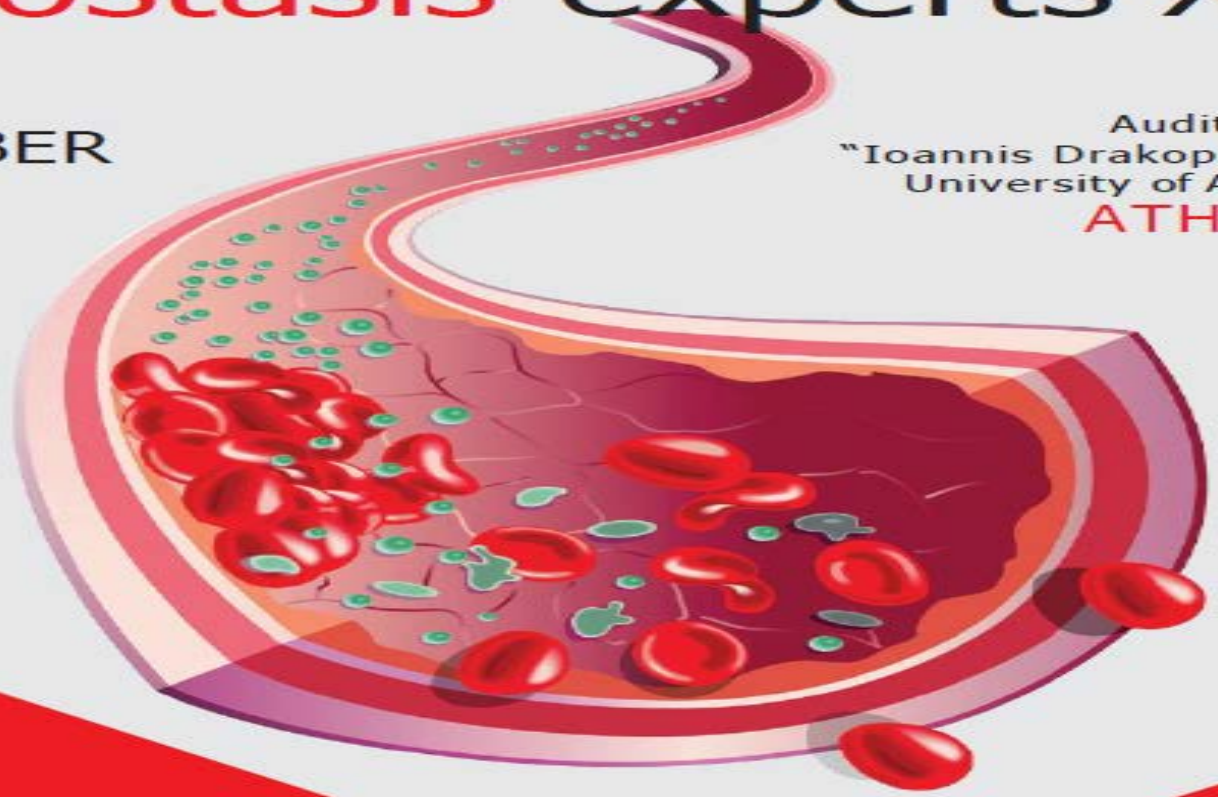
Under the Auspices
National and Kapodistrian
University of Athens Medical School

Organised
National and Kapodistrian University of Athens -
Aretaieion Hospital - Blood Bank
MSc Thrombosis - Haemorrage -
Blood Transfusion Medicine

SCIENTIFIC
PROGRAM

Meet the Hemostasis experts X



8 OCTOBER
2026

Auditorium
"Ioannis Drakopoulos"
University of Athens
ATHENS

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ

Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών

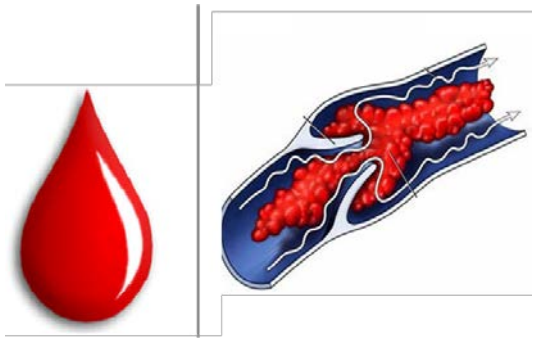
ΙΔΡΥΘΕΝ ΤΟ 1837

Μεταπτυχιακό Πρόγραμμα Σπουδών

«Θρόμβωση – Αιμορραγία - Ιατρική των μεταγγίσεων»

MSc

“Thrombosis- Bleeding -Transfusion Medicine”



https://tbtm-msc.gr

HELLENIC REPUBLIC
National and Kapodistrian University of Athens

MSc
"Thrombosis- Bleeding -Transfusion Medicine"

Established in 2009 (ΦΕΚ 1908_07/09/2009)

External Evaluation in 2014

Accreditation in 2018 (ΦΕΚ 3285_08/08/2018)

External Evaluation in 2023

Aim of the MSC "Thrombosis-Bleeding-Transfusion Medicine"

The scientific field of hemostasis disorders and transfusion therapy has greatly expanded during the last two decades. It is of interest to practically all specialties of medicine and related basic sciences (biochemistry, biology, etc.), as well as scope for all the above scientists and other health professionals (nurses, medical laboratory technologists, etc.).

- The M Sc aims at providing high-level postgraduate education in the scientific field of understanding and studying the mechanism of Hemostasis and its disorders.
- It aims to educate the principles of transfusion therapy and its applications at the clinical and laboratory level.
- The MSc aims to prepare scientists for further studies or research by giving them the theoretical and practical background for carrying out an autonomous research project, while at the same time for strengthening interdisciplinary synergies for the benefit of the cognitive research it serves.

- MSC graduates are provided with the theoretical background and laboratory skills so that they are able to participate in:
- the staff of Hematology Laboratories, Blood Donation Units, Surgical, Pathology, Cardiology, Anesthesiology Clinics, Intensive Care Units of the public and private sector as well as Research Laboratories and Competent Authorities (as defined by national and European legislation),
- positions that require special expertise and knowledge in the laboratory investigation, clinical decision making and treatment of disorders of Hemostasis and Transfusion Therapy
- in university and college as teaching staff
- preparation , writing, submission and publication of research papers ain Greek and international conferences and scientific journals
- further academic projects such as doctoral theses
- competitive national and international research projects.

Steering Committee of
MSc
«Thrombosis- Bleeding -Transfusion Medicine»

Valsami S.

Hematologist, Professor, School of Medicine, National and Kapodistrian University of Athens, Blood Transfusion Unit, Aretaieio Hospital

Klonaris C.

Vascular Surgeon, Professor of Medicine, University of Athens/ A' Surgery Clinic ''Laiko'' Hospital, Athens, Greece

Matsota P.

Anesthetist, Professor of Medical School of University of Athens, B' Anaesthesiology Clinic ''Attikon'' Hospital, Athens, Greece

Politou M.

MD, PhD, Professor of Laboratory Hematology-Immunohematology, Head of Hematology Lab and Blood Bank, "Aretaion" Hospital, Athens, Greece

Terpos E.

MD, PhD, Professor of Hematology in the Department of Clinical Therapeutics in the University of Athens, School of Medicine, Athens, Greece

Eligible students

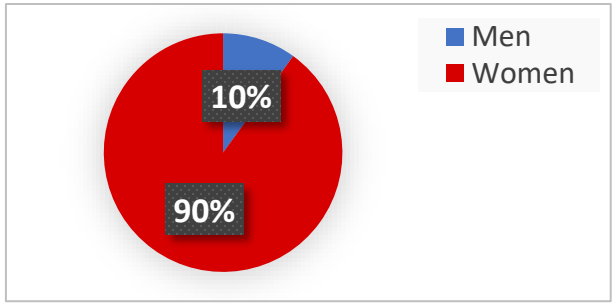
University graduates of the Departments of :

- Medicine
- Dentistry
- Pharmacy,
- Nursing,
- Biology,
- Molecular Biology
- Veterinary Medicine,
- Biochemistry,
- Medical Laboratory Technology,

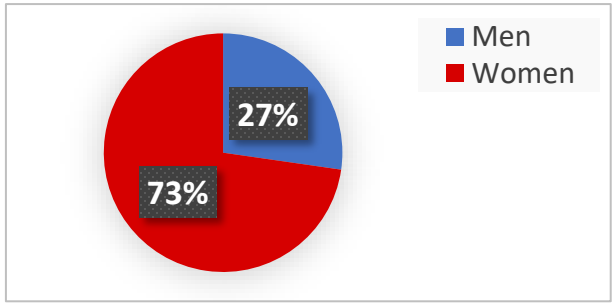
The maximum number of students admitted to the Master's Program is set at thirty (30) in total.

Current Students N=42

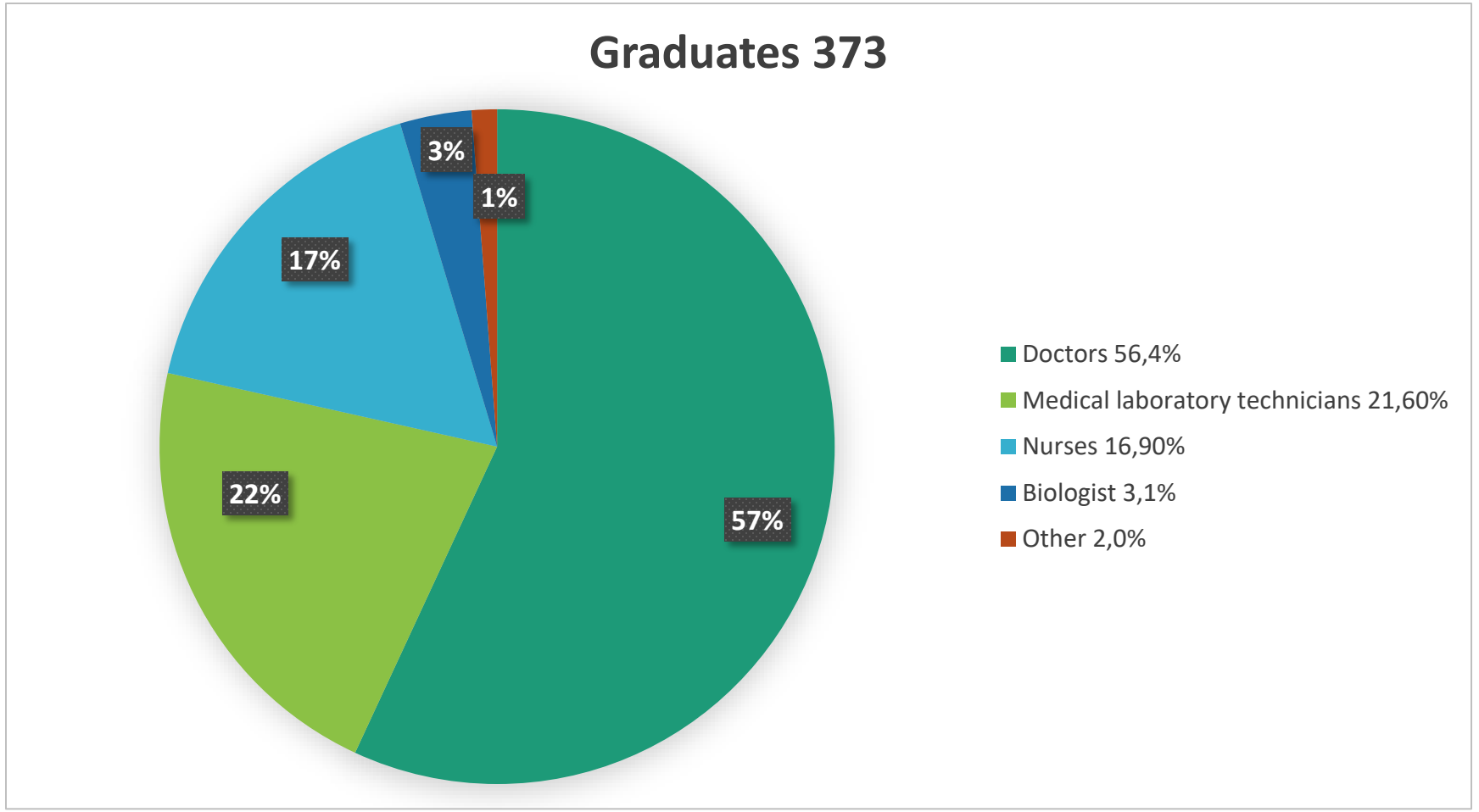
Students B → Female: 18 Male: 2

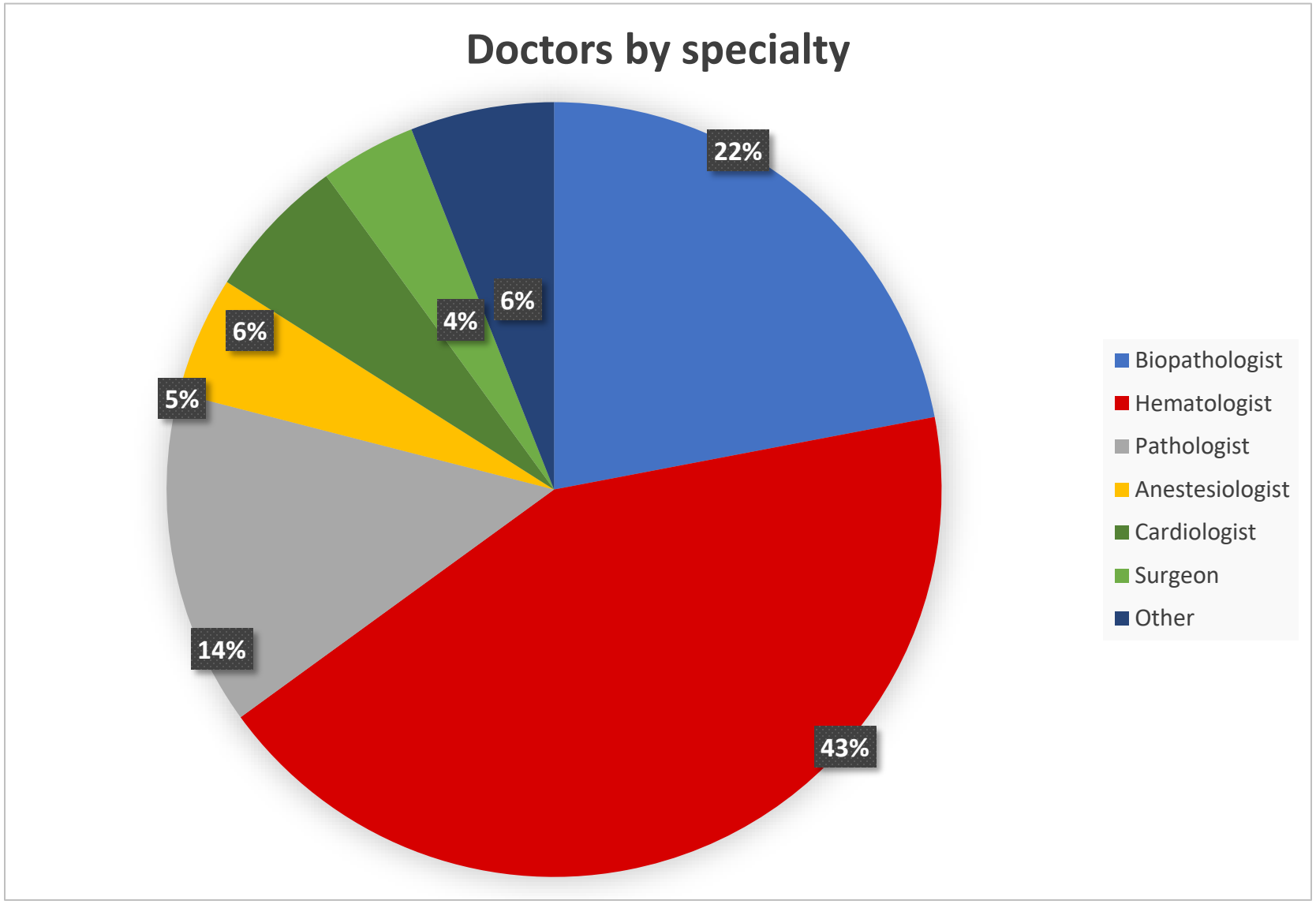


Students D → Female: 16 Male: 6



MSc «Thrombosis- Bleeding -Transfusion Medicine»




Doctors by specialty
22%
43%
14%
5%
6%
4%
6%
Biopathologist
Hematologist
Pathologist
Anestesiologist
Cardiologist
Surgeon
Other

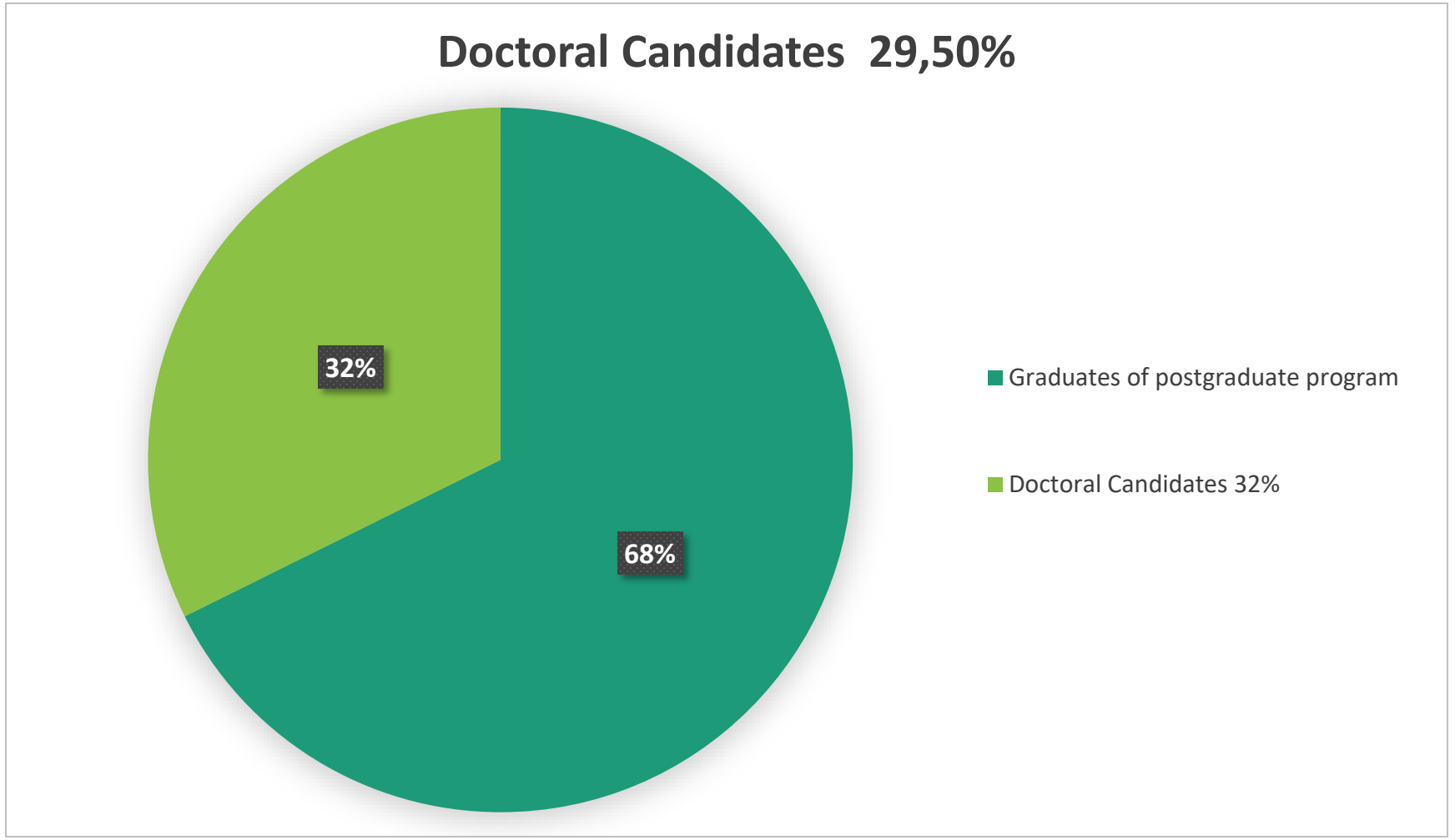
Student-centered learning

Accessibility to people with disabilities

Promotion of equal representation of the sexes

- Inclusivity

MSc «Thrombosis- Bleeding -Transfusion Medicine»



The duration of study at MSC leading to the award of a Master's Degree is completed in four (4) academic semesters

The course schedule is structured as follows:

A΄ semester

Principles of Immunology, Biochemistry, Molecular Biology and Genetics of Hemostasis - **ECTS 9** (5 hours per week)

Methodology of research and practical applications on bleeding diathesis - **ECTS 7** (4 hours per week)

Laboratory investigation of hemorrhagic disorders-Laboratory exercise – **ECTS 7** (4 hours per week)

Evidence based Decision Making in the clinical management of bleeding - **ECTS 7** (2 hours per week)

B΄ semester

Methodology and practical applications of thrombosis research - **ECTS 6** (5 hours per week)

Anticoagulant Treatment: pharmacology, indications, management, side effects – **ECTS 7** (3 hours per week)

Thrombotic complications in clinical entities – **ECTS 10** (5 hours per week)

Research Methodology II & Biostatistics,Study and presentation of recent clinical and basic research articles **- ECTS 7** (2 hours per week)

C΄ semester

Molecular biology and immunology of transfusion - **ECTS 6** (2 hours per week)

Blood and transfusable blood products: preparation, indications, side effects – **ECTS 7** (3 hours per week)

Cell therapies-Alternatives to blood transfusion – **ECTS 4** (3 hours per week)

Legal, administrative and financial framework of Blood donation &Transfusion. The Greek, European and International data - **ECTS 6** (2 hours per week)

Bleeding management algorithms -Laboratory exercise **- ECTS 7** (5 hours per week)

D΄ semester

Master's thesis – **ECTS 30**

List of Published Papers

1.Ισαακίδου Ελίνα, Μ.Πολίτου, Ιακωβίδου Νικολέττα, Κόλλια Μαρία, **Μούγιου Βασιλική**, Σώκου Ροζέτα, Μπούτσικου Μαρία, Ηλιοδρομίτη Ζωή, Μπούτσικου Θεοδώρα, Βαλσάμη Σερένα
Μελέτη της λειτουργικότητας των αιμοπεταλίων με τη μέθοδο platelet function assay (pfa-100) σε νεογνά με περιγεννητική υποξία / ασφυξία.
Πανελλήνιο Νεογνολογικό Συνέδριο 2018.
2**. Α. Χαϊκάλη**, Δ. Αγορίτσα, Β. Καβαλάρη, Μ.Πολίτου , Κ. Σταμούλης
Νέα μετάλλαξη στο γονίδιο rhce που κωδικοποιεί τον d- - φαινότυπο.
Πανελλήνιο Συνέδριο Μεταγγισιοθεραπείας 2019.
3., **Θ. Κουτσούρη Ζούλας Δ, Χαικάλη Ν, Μαραγκάκη Λ, Φλεσσιοπούλου Ι,** Μαυράκη,Μεγάλου Α, Σ. Βαλσάμη, Σταμούλης Κ, Μ.Πολίτου
Μοριακή τυποποίηση του RhD σε RhD αρνητικά δείγματα αιμοδοτών με θετική έμμεση δοκιμασία αντισφαιρινικού ορού με το μονοκλωνικό μίγμα NOVACLONETM Anti-D IgM + IgG στον αυτόματο αναλυτή GALILEO NEO.
Πανελλήνιο Συνέδριο Μεταγγισιοθεραπείας 2019.
4. **Φ. Ρουσσίνου,** Σ. Βαλσάμη, Θ. Κασσή, Δ. Κορακάκης **, Θ. Κουτσούρη, Σ. Ντουένας**, Β. Βάσσιος, Γ. Σαλαβέρης, Β. Κασιδόκωστα, Α. Βασιλακοπούλου, Α. Σίσκος, Β. Φλώρου, Ν. Πάστρα, Δ. Λαχανά, Β. Κλιάφα, Κ. Τζερεμέ, Γ. Μουστάκας, Θ. Πιτταράς, Μ.Πολίτου
Θετική άμεση coombs νεογνών - ο ρόλος της ΑΒΟ ασυμβατότητας.
11ο Πανελλήνιο Συνέδριο Μεταγγισιοθεραπείας, Αθήνα 5-7 Απριλίου 2019 (Προφορική Ανακοίνωση).
5. Μ.Πολίτου, **Φ.Κοντοτέζα,** Α.Πουλιάκης, Δ.Κορακάκης,Θ.Κασσή, **Π.Φόρτσα**, Β.Βάσσιος, **Φ.Ρουσσίνου** Σ.Βαλσάμη, Κ.Σταμούλης
Επίδραση παρατεταμένης αποθήκευσης στα ποιοτικά χαρακτηριστικά ανακτώμενου πλάσματος : υπάρχει όριο λήξης ;
30ο Πανελλήνιο Αιματολογικό Συνέδριο, 01-04 Νοεμβρίου 2019.
6. Ι. Παπασωτηρίου, Σ. Ζάλιου, Β. Τσαουσης, **Μ. Δημοπούλου**, Π. Φλέβάρης, Κ. Λαρίσση, **Β. Κομνηνάκα**, Χ. Ποζιόπουλος, Μ.Πολίτου, Ε. Τέρπος, Ε. Βοσκαρίδου
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30ο Πανελλήνιο Αιματολογικό Συνέδριο, 07-10 Νοεμβρίου 2019.
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1.T.Bagratuni, E. Kastritis, M Roussou1, D. Christoulas, E.Kostouros1, M.Politou, E. Terpos and MA. Dimopoulos,
Prospective Evaluation of the Risk of Thromboembolic Complications in Patients Treated with Lenalidomide-based Therapy and Genetic Associations
53rd ASH ,2011.

2. M M.Politou, V Kapsimali, **V Komninaka**, S Valsami, E Voskaridou.
Role of regulatory t cells in beta thalassemia .
18th Congress of the European Hematology Association, Stockholm, Swedena,13-16 June 2013.

3.Ersi Voskaridou, Katerina Larrisi, M.Politou, **Veroniki Komninaka**, Filia Apostolakou, Chrysanthi Skevaki, Maria Giannaki, Ioannis Papassotiriou and Evangelos Terpos
Increased Von Willebrand Factor and High Circulating Placental Growth Factor Correlate with Inflammation and Iron Overload in Patients with Compound Heterozygous Sickle Cell and Beta-Thalassemia .
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29.Effect of prolonged storage on quality characteristics of recovered plasma: Is there an expiration date?

Marianna Politou 1 **, Ifigeneia Vasiliki Kontoteza** 1 , Abraham Pouliakis 1 , Georgios Dryllis 1 , **Panagiota Fortsa** 1 , Serena Valsami 1 , Konstantinos Stamoulis 2

PMID: 36120965 DOI: 10.1111/trf.17115

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31. Haemostatic abnormalities or defects in greek patients with gaucher disease

Author(s): **VERONIKI KOMNINAKA**, Konstantina Repa, Theodoros Marinakis, Abraham Pouliakis, Dimitrios Korakakis, Theodora Kati , **Dimitrios Tsokanas**, Marianna Politou

(Abstract release date: 05/17/18) EHA Library. Komninaka V. 06/16/2018; 215366; PS1044

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PMCID: PMC9492943

PMID: 36160789

Primary hemostasis in fetal growth restricted neonates studied via PFA-100 in cord blood samples

Maria Kollia, 1 , † Nicoletta Iacovidou, 1 , † Zoi Iliodromiti, 1 Abraham Pouliakis, 2 Rozeta Sokou, 1 **Vasiliki Mougiou**, 1 Maria Boutsikou, 1 Marianna Politou, 3 Theodora Boutsikou, 1 and Serena Valsamicorresponding author 3 ,

33. Laboratory assessment of the anticoagulant activity of dabigatran

Elias Kyriakou 1 , Ignatios Ikonomidis 2 , **Dimitrios Stylos** 1 , Stefanos Bonovas 3 , Ioannis Papadakis 2 , Georgios K Nikolopoulos 4 , Styliani Kokoris 1 , Dimitrios Kalantzis 1 , Christine Economopoulou 1 , Petros Kopterides 5 , John Lekakis 2 , Argirios E Tsantes 6

Affiliations

PMID: 25525048 DOI: 10.1177/1076029614564209

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M Spartalis 1 , E Tzatzaki, E Spartalis, C Damaskos, A Athanasiou, D Moris, M Politou

Affiliations

PMID: 29131238

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Georgios Boutsikas 1 , Evangelos Terpos, Athanasios Markopoulos, Athanasios Papatheodorou, Angeliki Stefanou, George Georgiou, Zacharoula Galani, **Veroniki Komninaka**, Vasileios Telonis, Ioannis Anagnostopoulos, Lila Dimitrakopoulou, Konstantinos Anargyrou, Konstantinos Tsionos, Dimitrios Christoulas, **Maria Gonianaki**, Theophanis Giannikos, Styliani Kokoris, Kostas Konstantopoulos, John Meletis, Maria K Angelopoulou, Anthi Travlou, Theodoros P Vassilakopoulos

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PMID: 28952223

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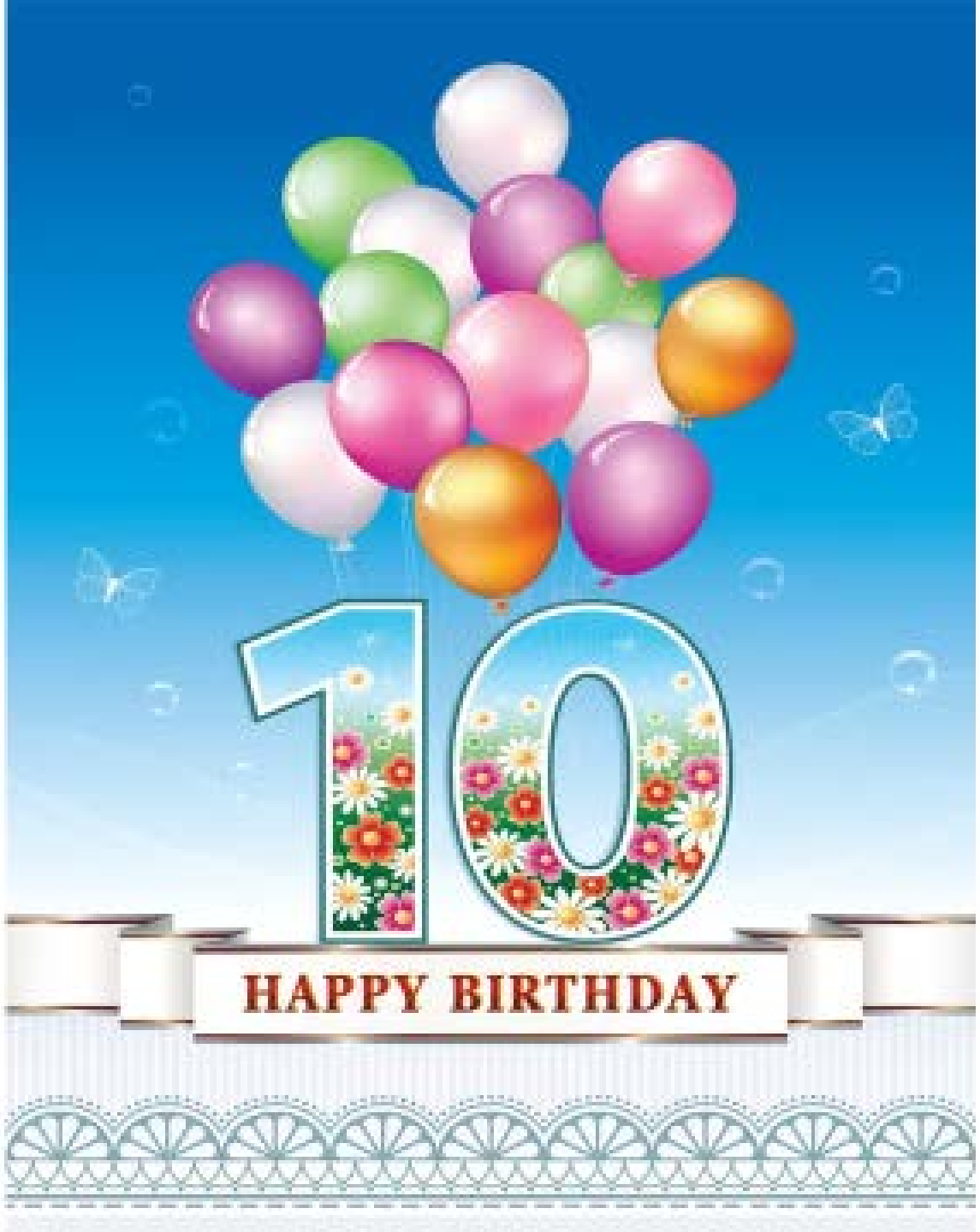
COLLABORATIONS

Transfusion Units of:

- IIBEAA
- EKEA
- Medical school of APT university
- Aretaieion Hospital
- Athens Medical Group
- Attica General Hospital KAT
- Attikon University Hospital
- General Children's Hospital "Aghia Sofia"
- General Hospital “Evaggelismos”

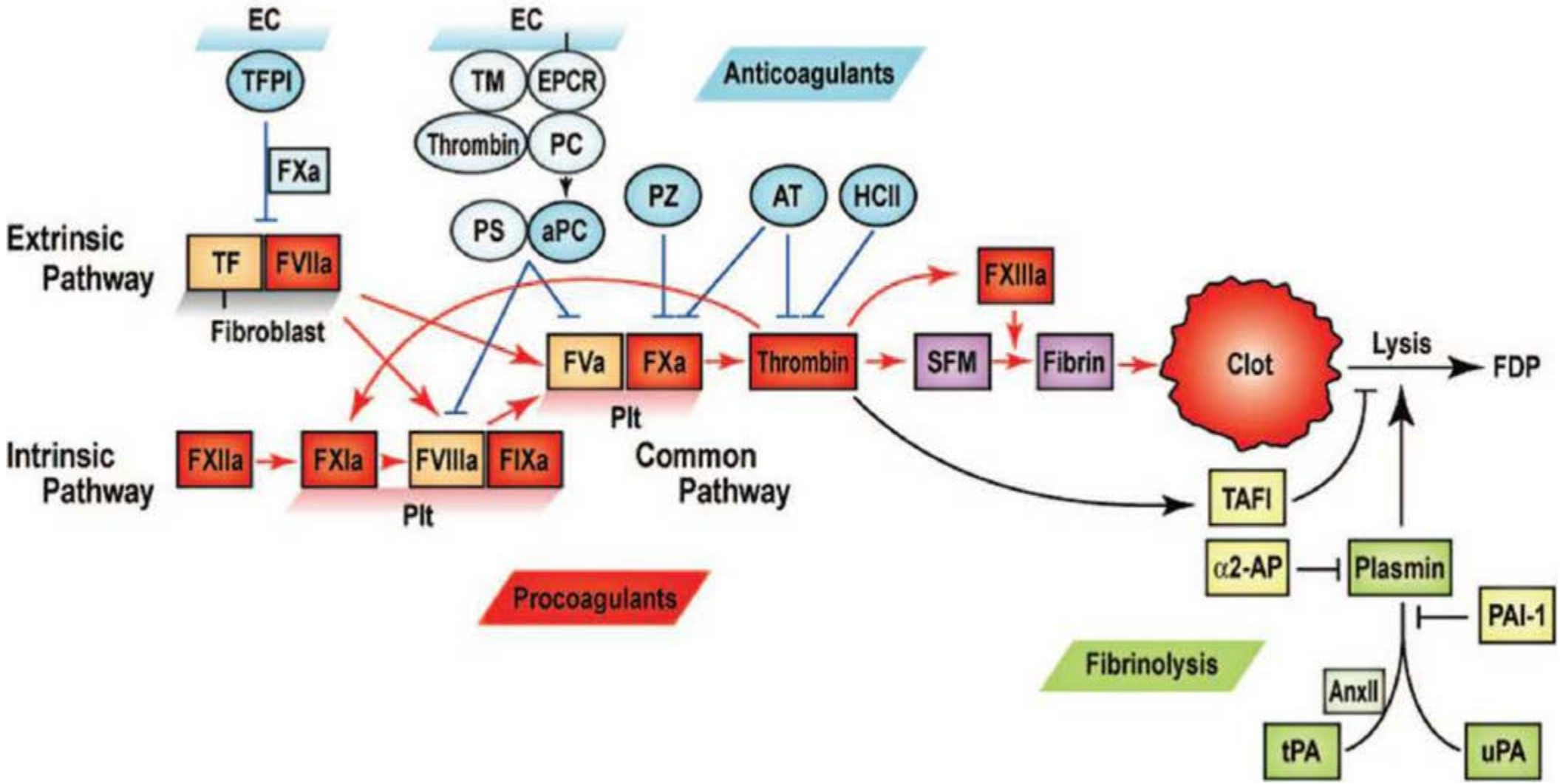
- General Hospital of Irakleion "Venizeleio - Pananeio"
- General Hospital of Nikea
- General Oncological Hospital of Kiffisia
- General University Hospital of Patras
- Geniko Periferiako Nosokomio Papageorgiou
- Ippokrateio General Hospital of Athens
- Ippokrateio General Hospital of Thessaloniki
- Konstantopoulio General Hospital
- Laiko General Hospital of Athens
- Onassis Cardiac Surgery Center
- General Hospital Hellenic Institute Against Cancer “Agios Savvas”

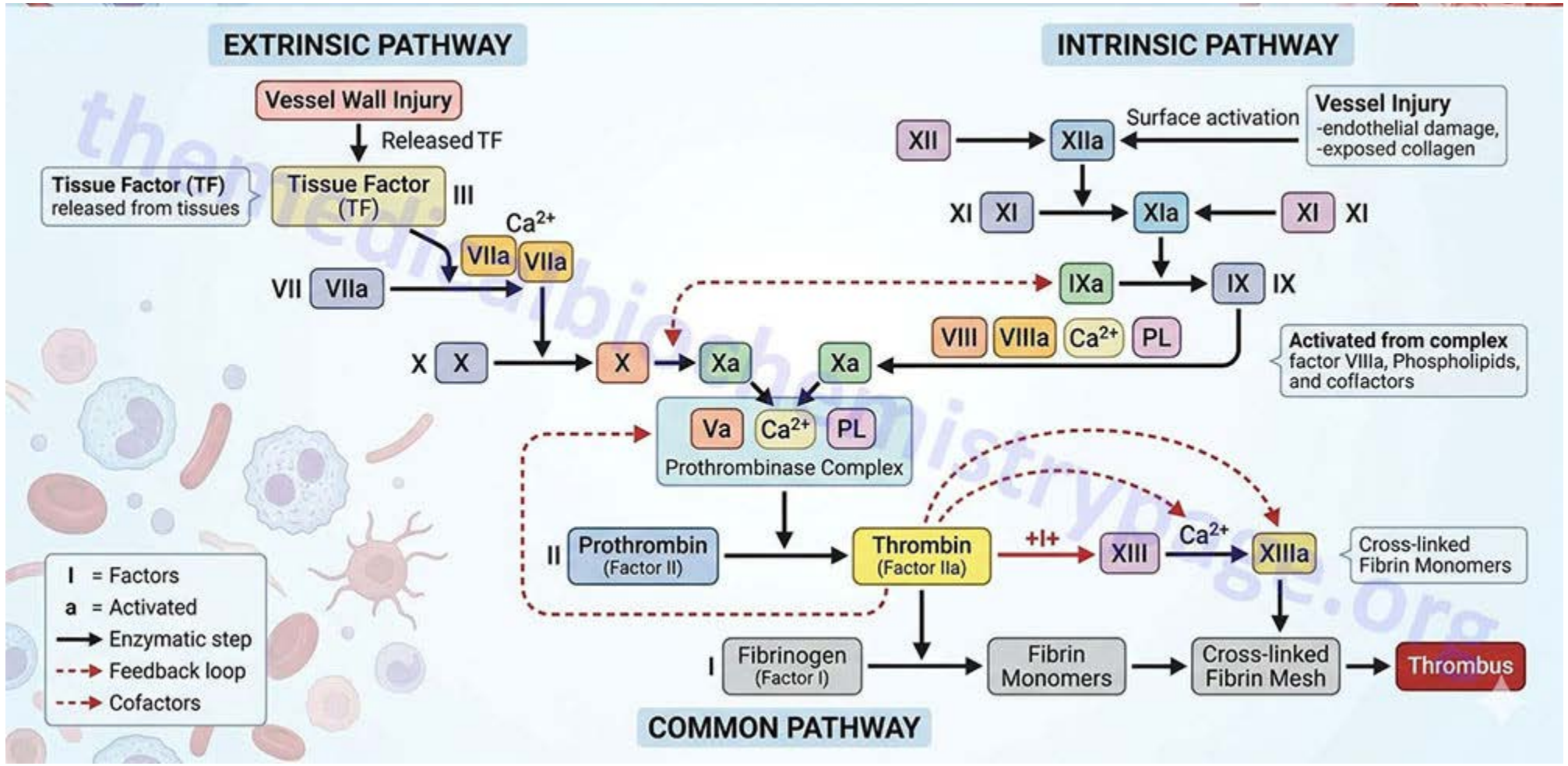

10
HAPPY BIRTHDAY

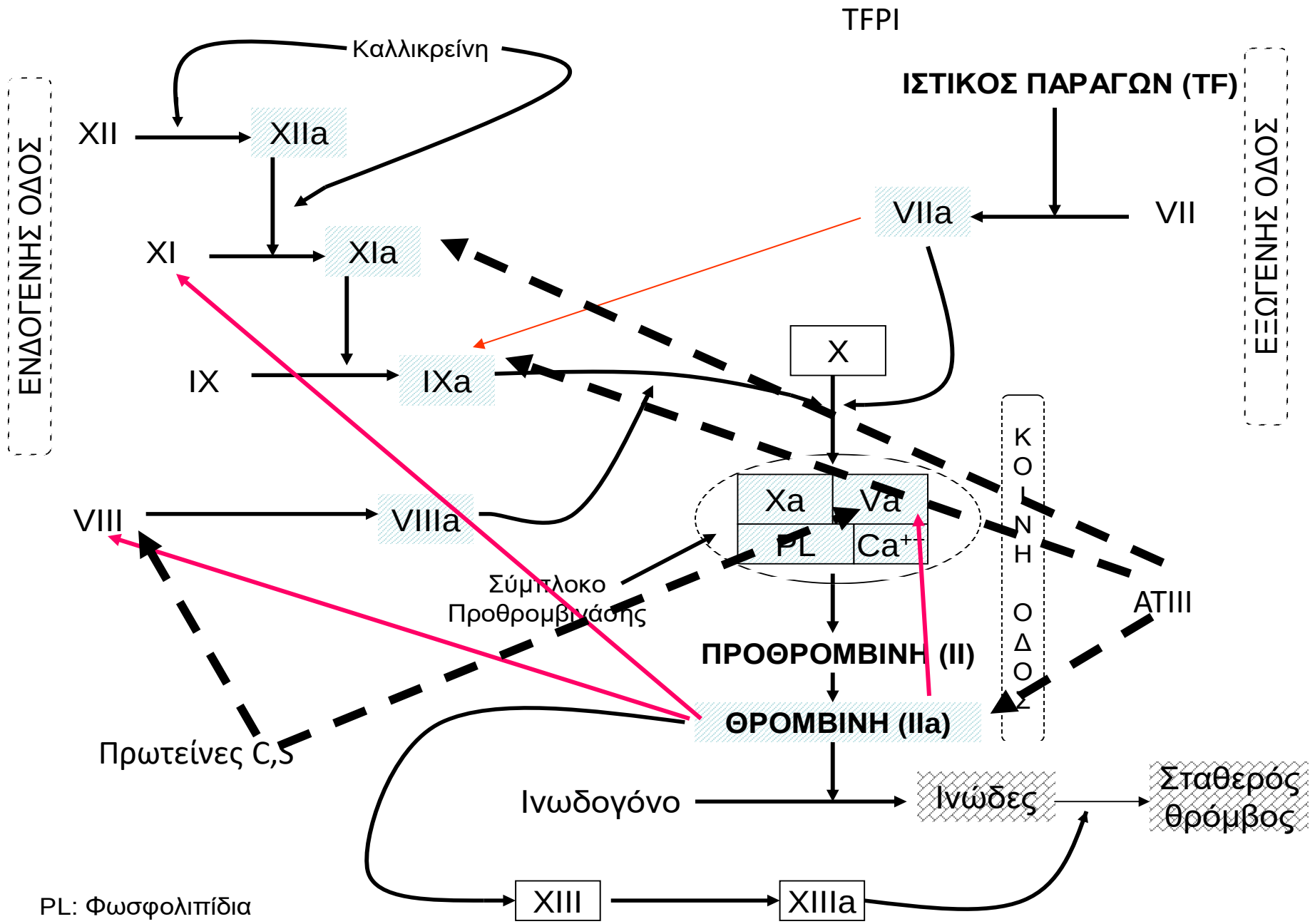
Brain Regulation of Thrombosis and Hemostasis

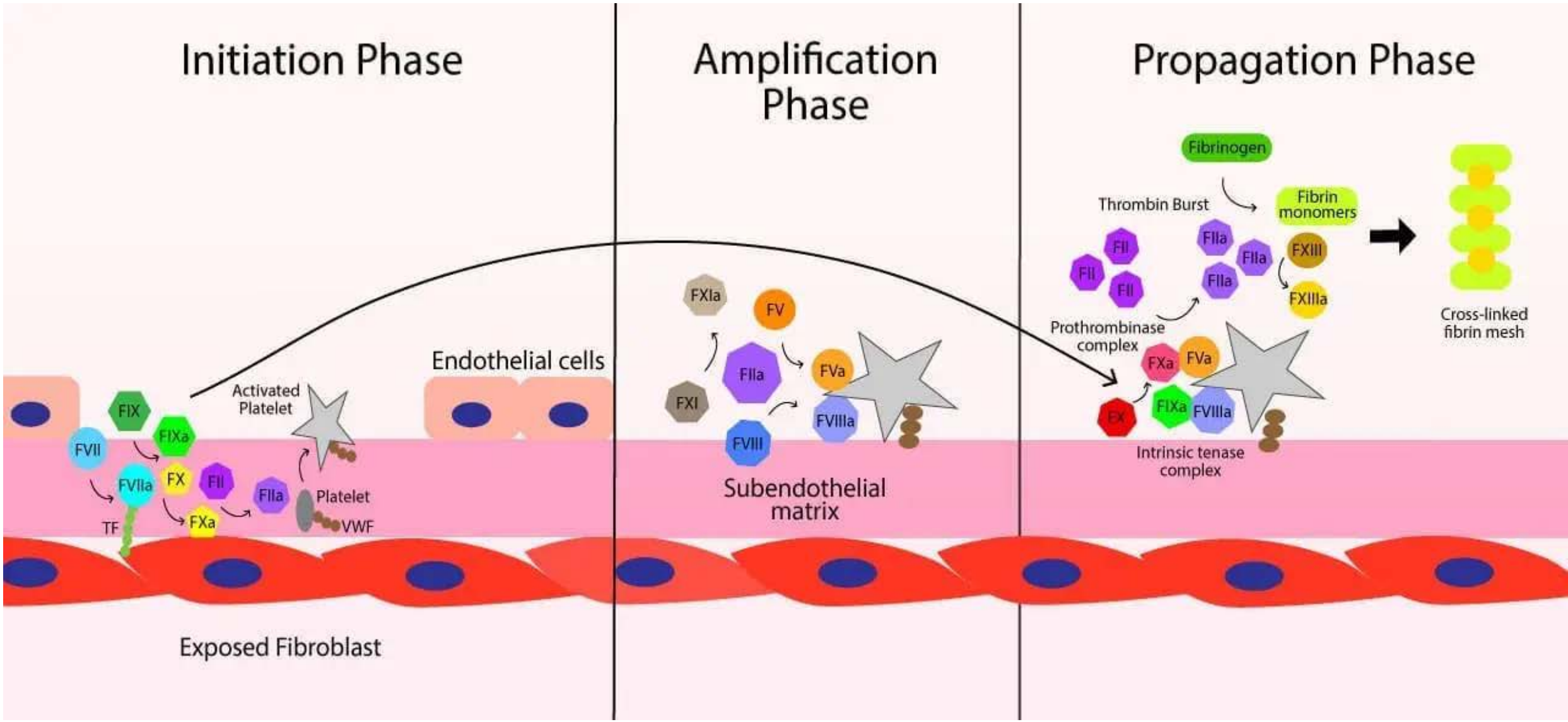
P

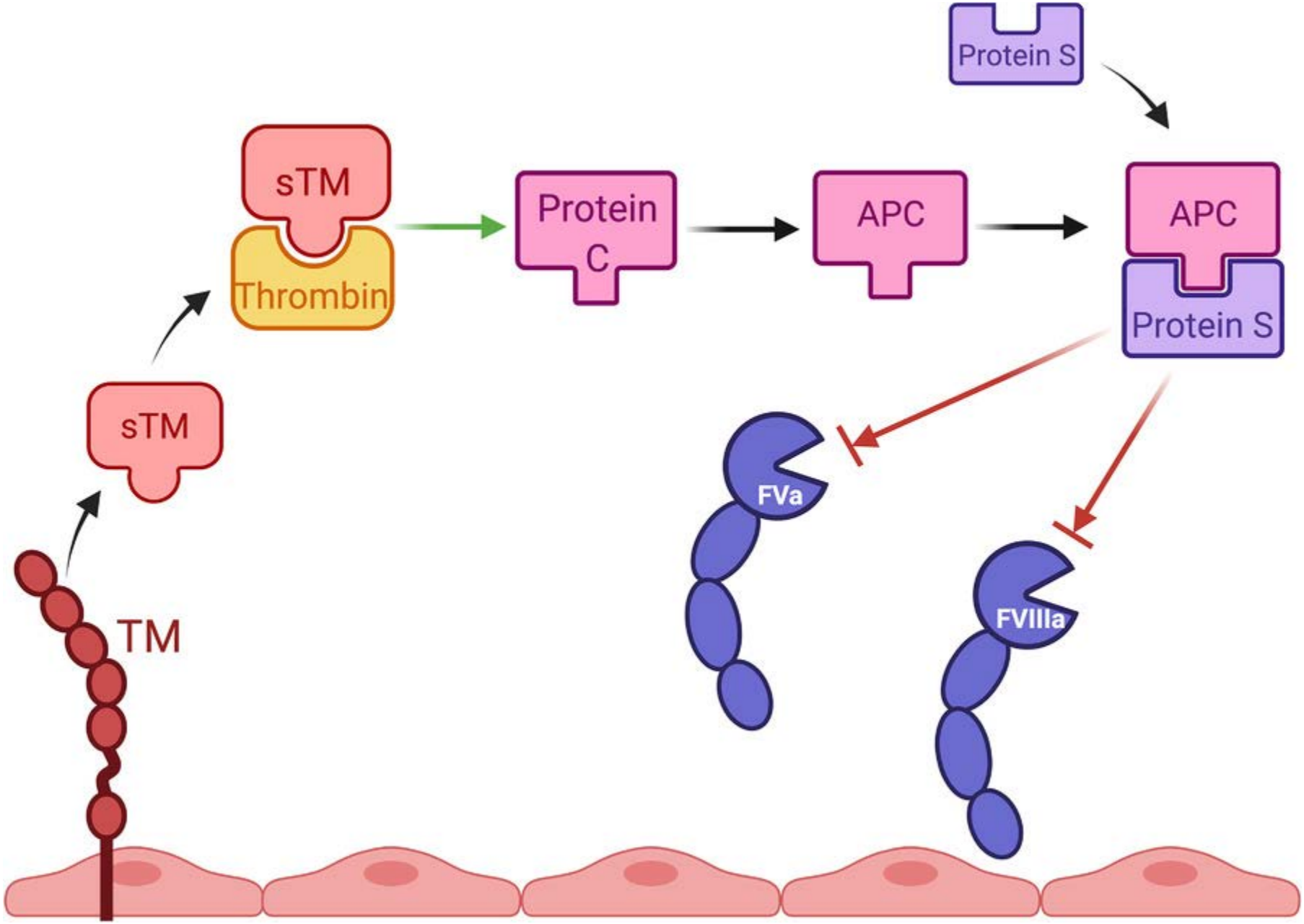
Source: Fisher MJ. Stroke. 2013;44:3275–3285.

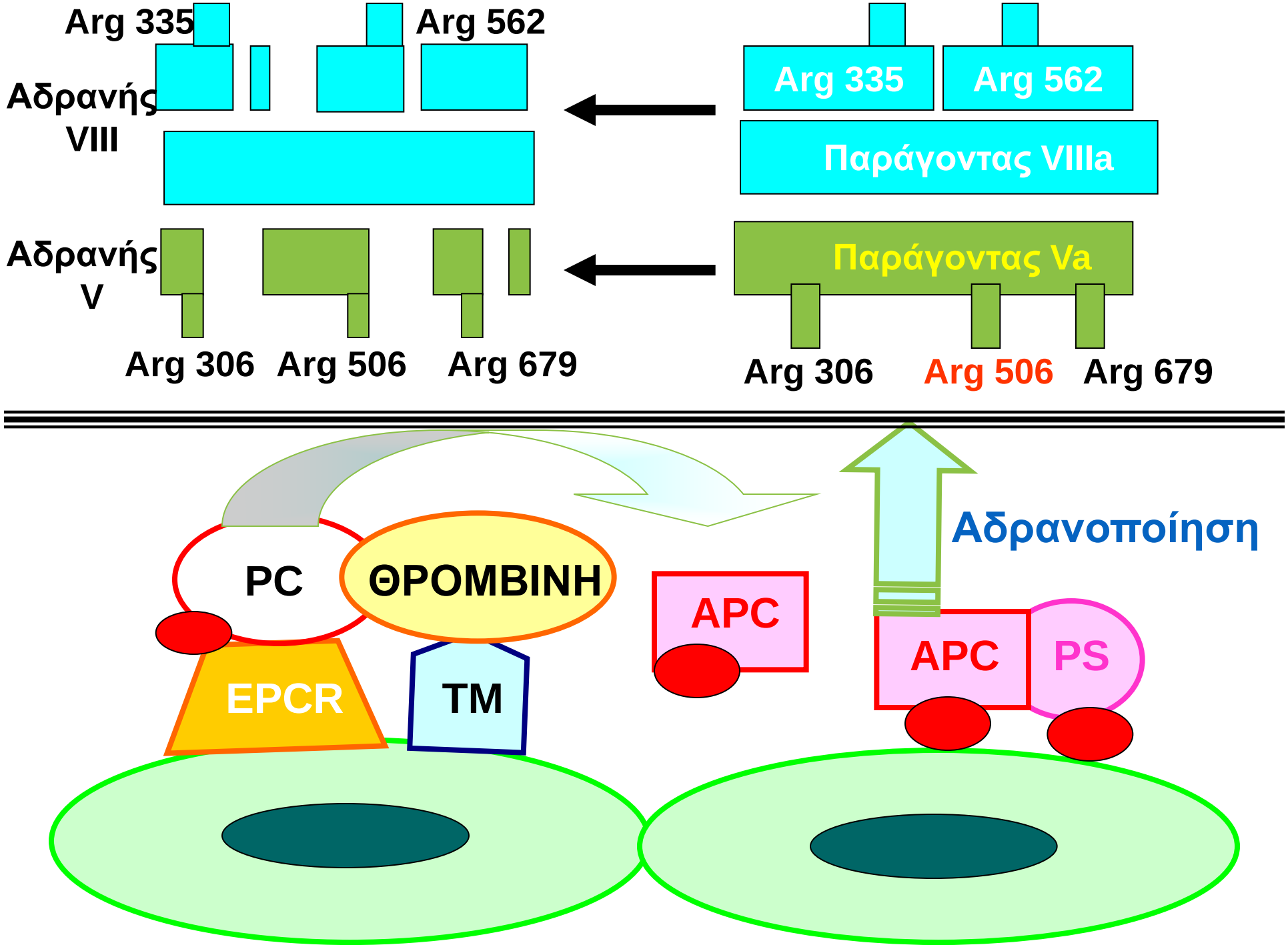

EC
TFPI
FXa
Extrinsic Pathway
TF
FVIIa
Fibroblast
EC
TM
EPCR
Thrombin
PC
PS
aPC
Anticoagulants
PZ
AT
HCII
FXIIIa
FVa
FXa
Thrombin
SFM
Fibrin
Clot
Lysis
FDP
Plt
Intrinsic Pathway
FXIIa
FXIa
FVIIIa
FIXa
Plt
Common Pathway
TAFI
α2-AP
Plasmin
PAI-1
Procoagulants
Fibrinolysis
AnxII
tPA
uPA


EXTRINSIC PATHWAY
Vessel Wall Injury
Released TF
Tissue Factor (TF) released from tissues
Tissue Factor (TF)
III
Ca2+
VIIa
VIIa
VII
VIIa
X
X
X
Xa
Xa
INTRINSIC PATHWAY
XII
XIIa
Surface activation
Vessel Injury
-endothelial damage,
-exposed collagen
XI
XI
XIa
XI
XI
IXa
IX
IX
VIII
VIIIa
Ca2+
PL
Activated from complex
factor VIIIa, Phospholipids, and coffactors
Va
Ca2+
PL
Prothrombinase Complex
II
Prothrombin (Factor II)
Thrombin (Factor IIa)
+I+
XIII
Ca2+
XIIIa
Cross-linked Fibrin Monomers
I
Fibrinogen (Factor I)
Fibrin Monomers
Cross-linked Fibrin Mesh
Thrombus
COMMON PATHWAY
I = Factors
a = Activated
Enzymatic step
Feedback loop
Cofactors

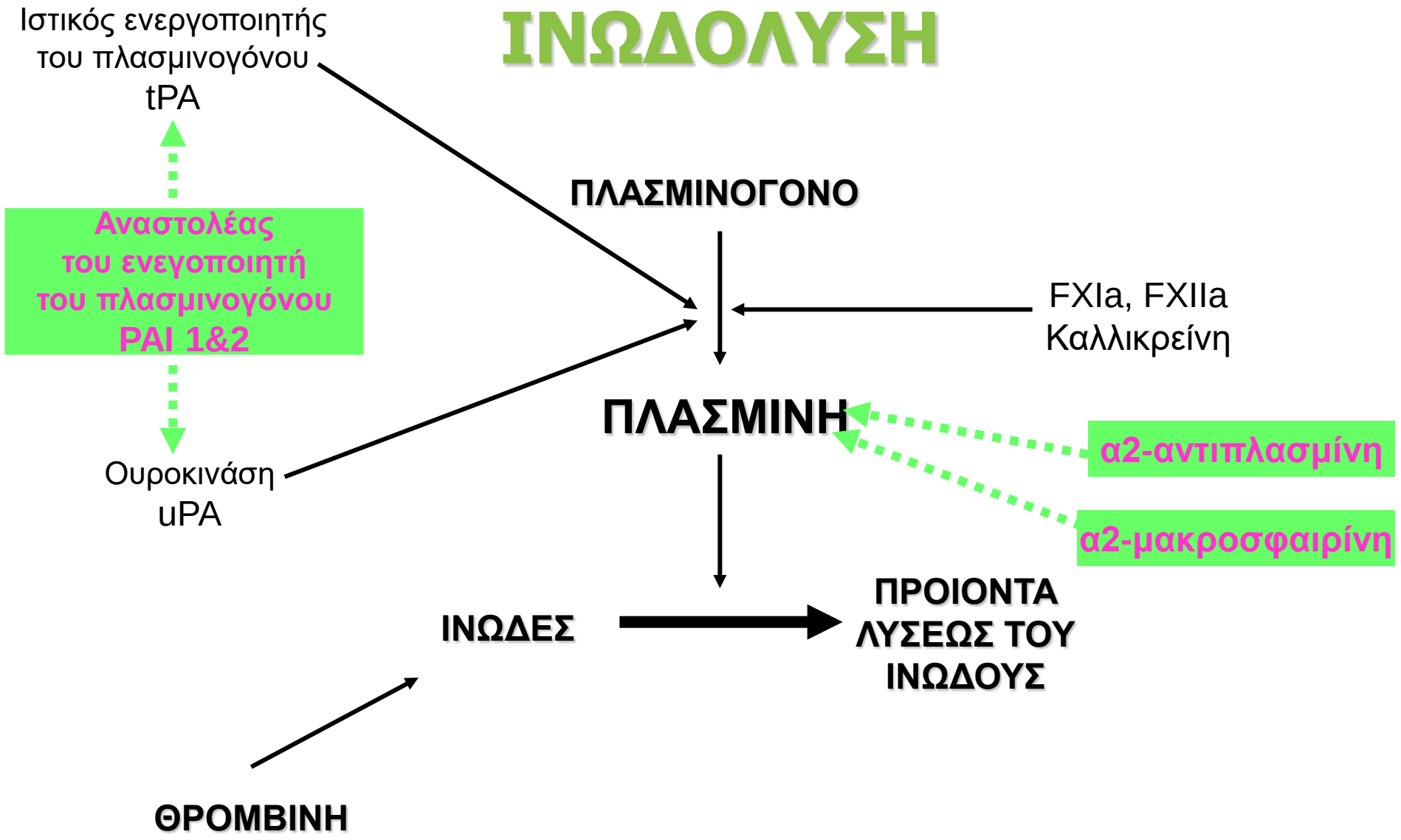

TFPI
Καλλικρείνη
ΙΣΤΙΚΟΣ ΠΑΡΑΓΩΝ (TF)
ΕΝΔΟΓΕΝΗΣ ΟΔΟΣ
ΕΞΩΓΕΝΗΣ ΟΔΟΣ
XII
XIIa
XI
XIa
IX
IXa
VIIa
VII
X
VIII
VIIIa
Xa
Va
PL
Ca++
ΚΟΙΝΗ ΟΔΟΣ
Σύμπλοκο Προθρομβινάσης
ATIII
ΠΡΟΘΡΟΜΒΙΝΗ (II)
ΘΡΟΜΒΙΝΗ (IIa)
Πρωτείνες C,S
Ινωδογόνο
Ινώδες
Σταθερός θρόμβος
XIII
XIIIa
PL: Φωσφολιπίδια

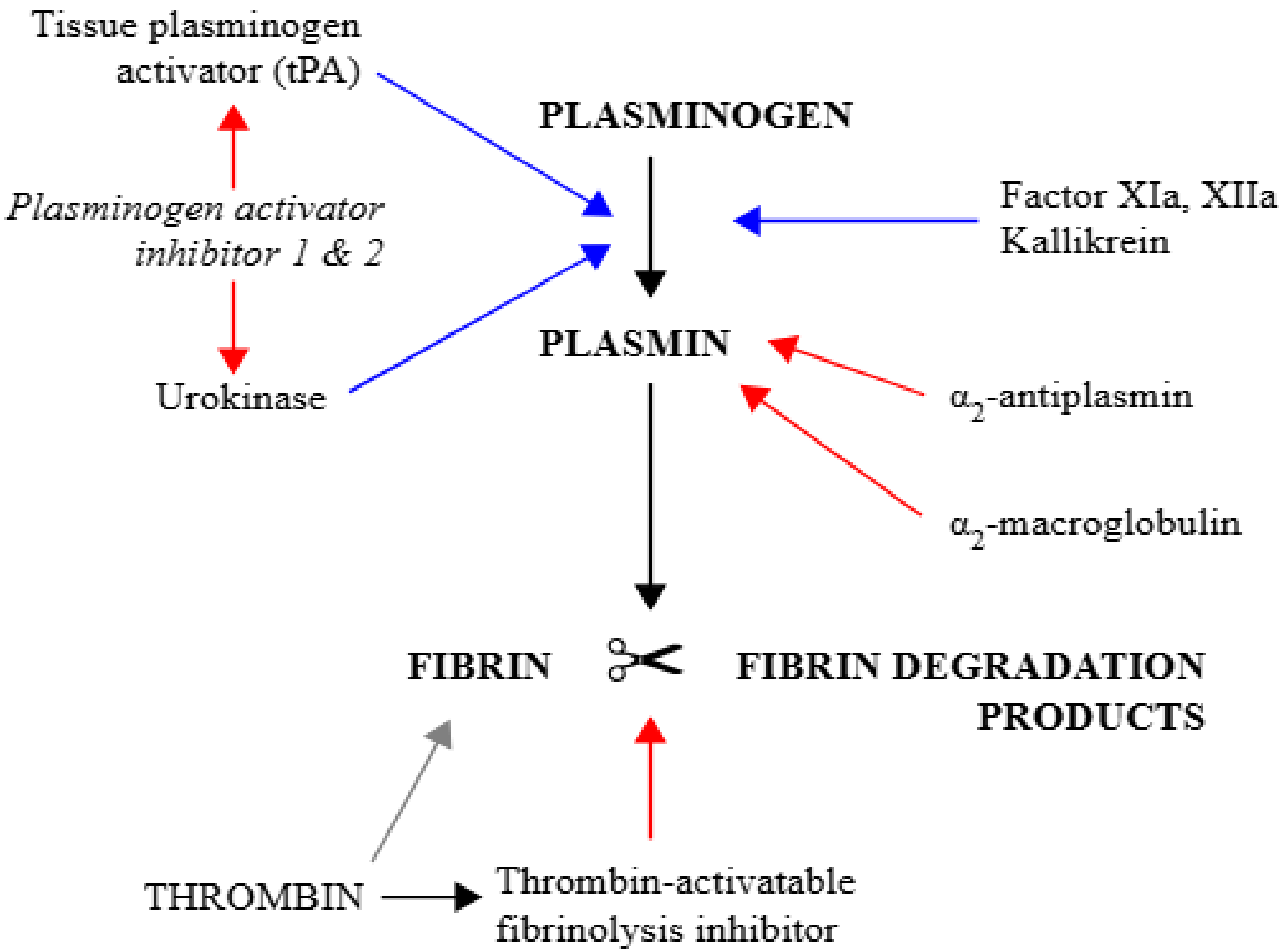

Initiation Phase
Amplification Phase
Propagation Phase
Endothelial cells
Activated Platelet
FIX
FIXa
FVII
FVIIa
FX
FII
FIIa
TF
FXa
Platelet
VWF
Exposed Fibroblast
FXIa
FV
FIIa
FVa
FXI
FVIIIa
FVIII
Subendothelial matrix
Fibrinogen
Thrombin Burst
Fibrin monomers
FII
FII
FII
FIIa
FIIa
FIIa
FXIII
FXIIIa
Prothrombinase complex
FXa
FVa
FX
FIXa
FVIIIa
Intrinsic tenase complex
Cross-linked fibrin mesh


Protein S
sTM
Thrombin
Protein C
APC
APC
Protein S
sTM
FVa
FVIIIa
TM

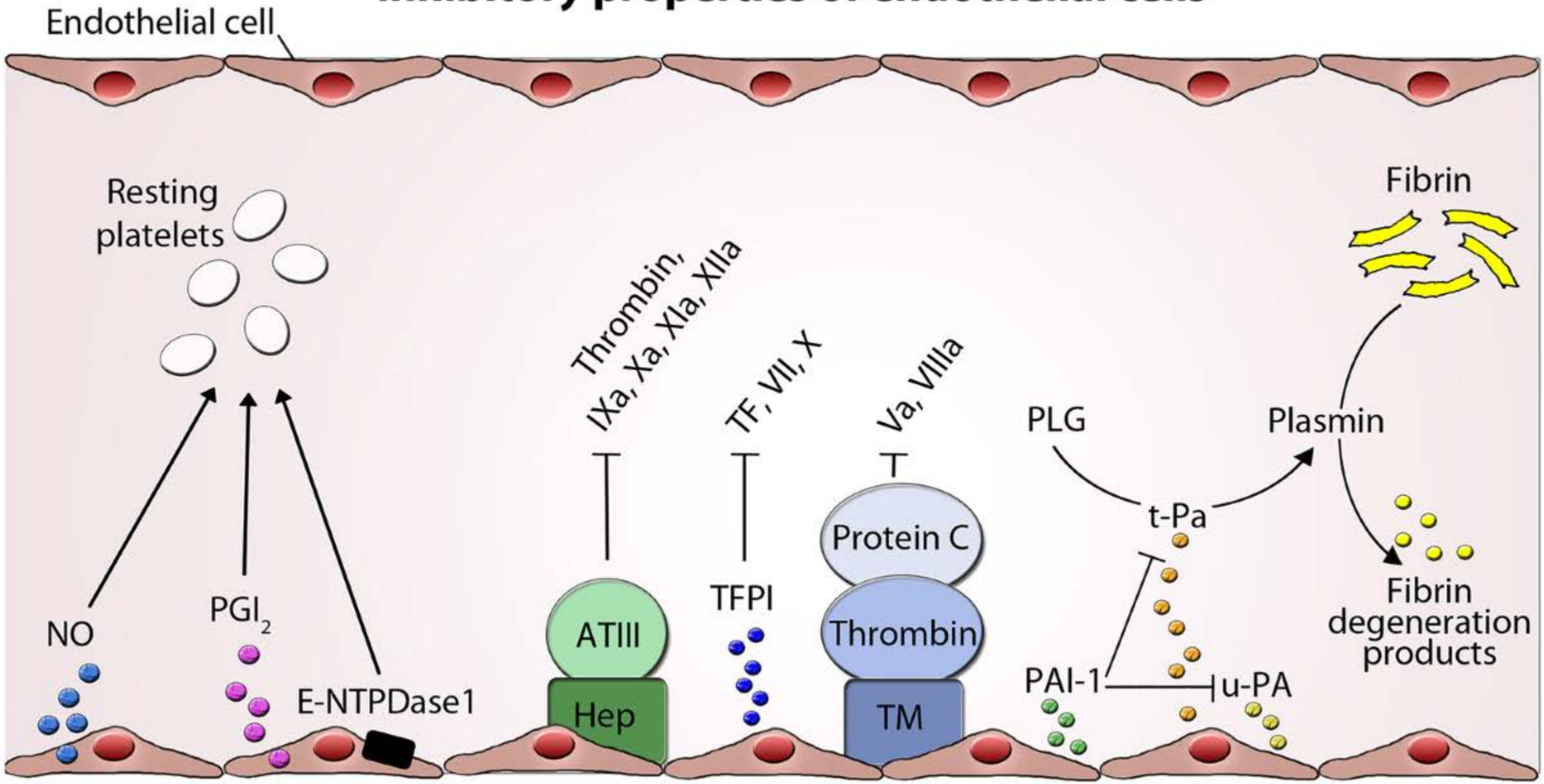

Arg 335
Arg 562
Αδρανής VIII
Arg 335
Arg 562
Παράγοντας VIIIa
Αδρανής V
Παράγοντας Va
Arg 306
Arg 506
Arg 679
Arg 306
Arg 506
Arg 679
PC
ΘΡΟΜΒΙΝΗ
EPCR
TM
APC
APC
PS
Αδρανοποίηση

ΙΝΩΔΟΛΥΣΗ


Ιστικός ενεργοποιητής του πλασμινογόνου
tPA
Αναστολέας του ενεργοποιητή του πλασμινογόνου
PAI 1&2
Ουροκινάση
uPA
ΠΛΑΣΜΙΝΟΓΟΝΟ
FXIa, FXIIa
Καλλικρείνη
ΠΛΑΣΜΙΝΗ
α2-αντιπλασμίνη
α2-μακροσφαιρίνη
ΙΝΩΔΕΣ
ΠΡΟΙΟΝΤΑ ΛΥΣΕΩΣ ΤΟΥ ΙΝΩΔΟΥΣ
ΘΡΟΜΒΙΝΗ


Tissue plasminogen activator (tPA)
PLASMINOGEN
Plasminogen activator inhibitor 1 & 2
Factor XIa, XIIa Kallikrein
PLASMIN
Urokinase
α2-antiplasmin
α2-macroglobulin
FIBRIN
FIBRIN DEGRADATION PRODUCTS
THROMBIN
Thrombin-activatable fibrinolysis inhibitor

Inhibitory properties of endothelial cells



The 4-Component Framework

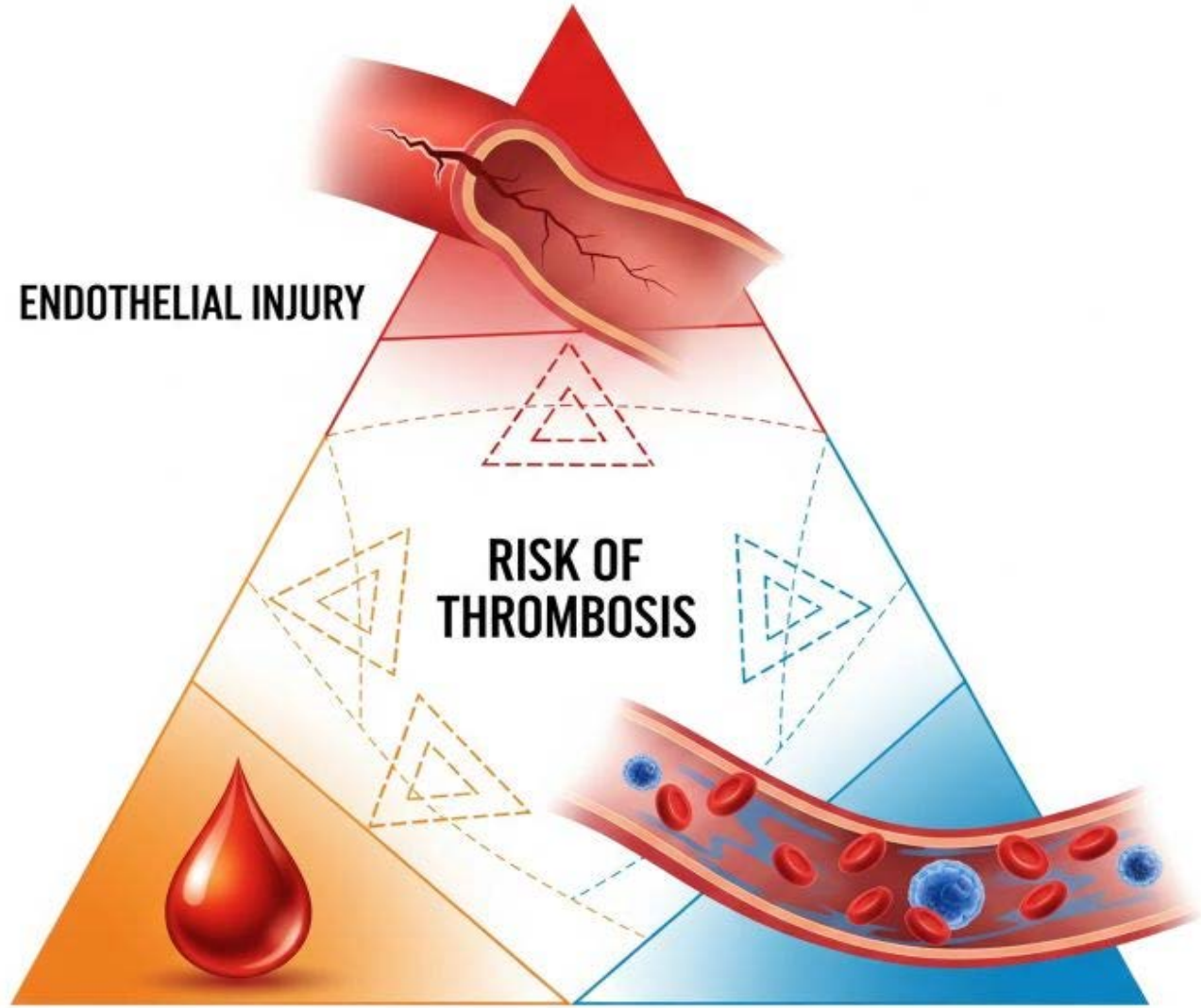
Hepatic Factors (The Liver)• Role: Synthesizes the majority of circulating plasma coagulation factors (such as Fibrinogen, Prothrombin, Factors V, VII, IX, X, XI, and XIII) as well as natural anticoagulants (Antithrombin, Protein C, and Protein S Bone

Marrow–Derived Hematopoietic Cells• Role: Provides cellular effectors of primary hemostasis and inflammation, primarily platelets and monocytes.

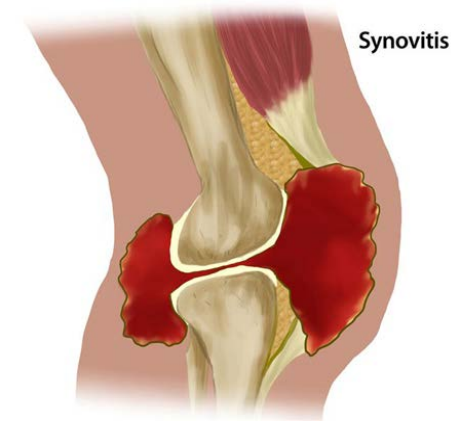
The Endothelium (Lining of Blood Vessels)• Role: Acts as an active regulatory interface that normally maintains a non-thrombotic surface

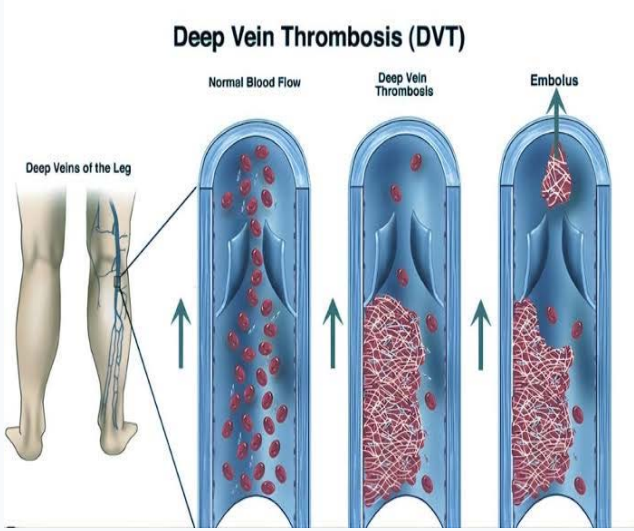
The Vascular Tree (Vessels and Hemodynamics)• Role: Manages local blood flow, shear stress, and immediate vascular tone

VIRCHOW'S TRIAD



Organ-specific hemostasis: beyond the classic cascade





Focal events despite systemic perturbation

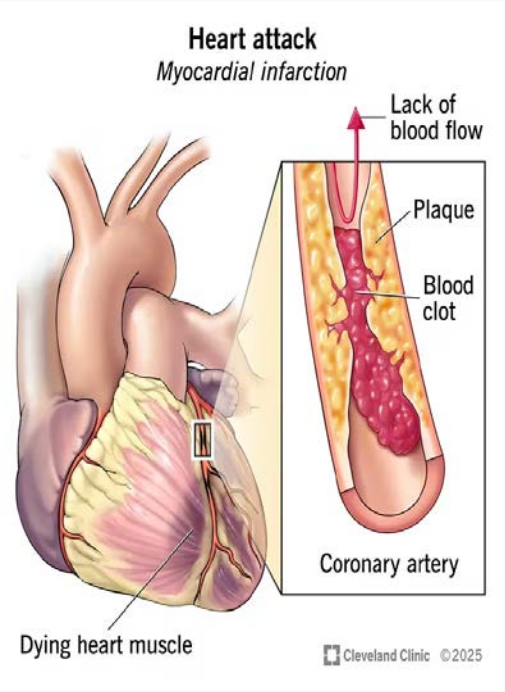
Systemic thrombophilia or bleeding tendency does not distribute uniformly across organs.

Vascular-bed heterogeneity

EPCR, TFPI, vWF, NO/prostacyclin and fibrinolytic proteins vary by vessel type and organ.

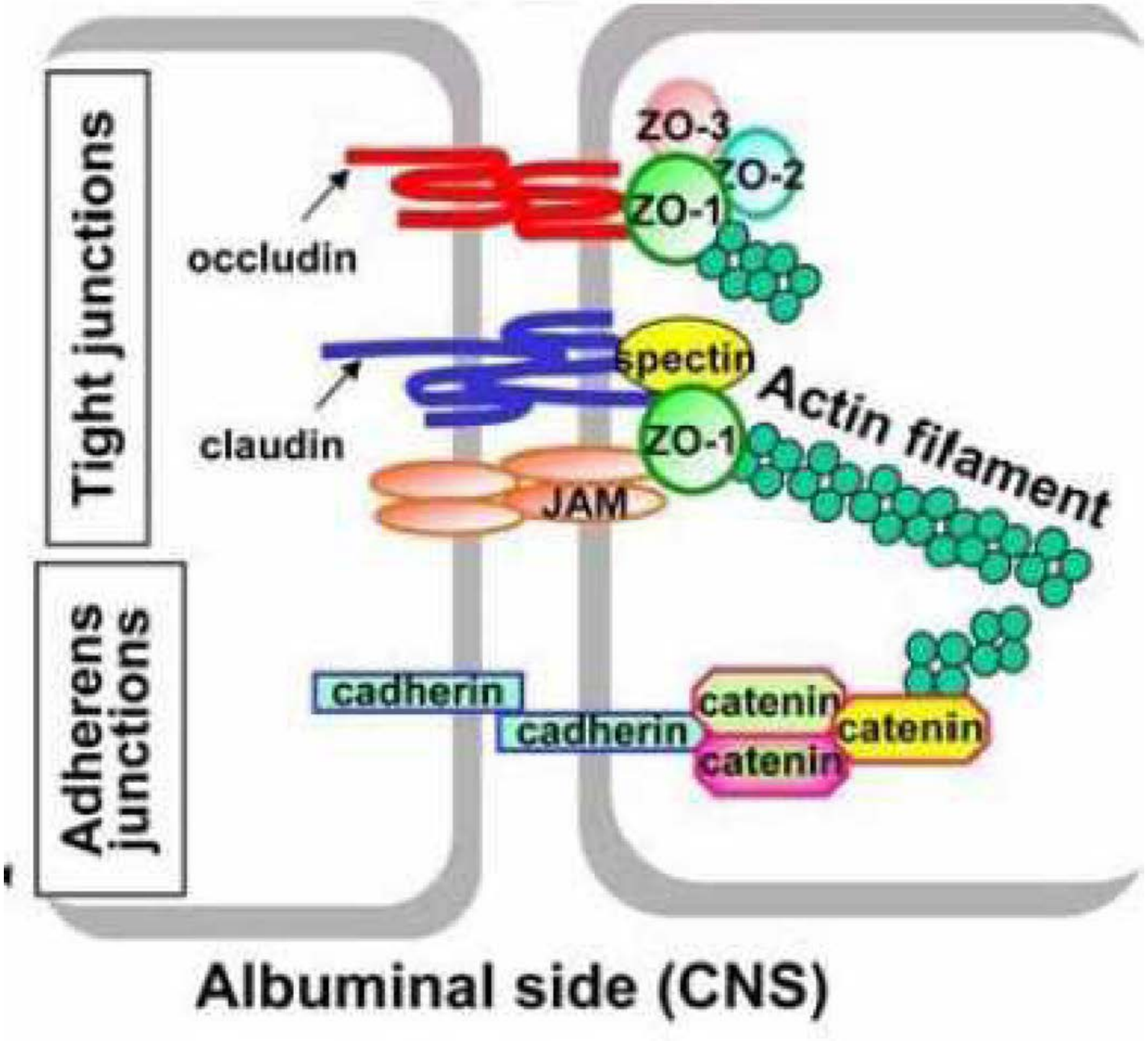
Clinical examples

Venous thrombosis, warfarin skin necrosis, erythromelalgia, hemophilic joint/muscle bleeding.



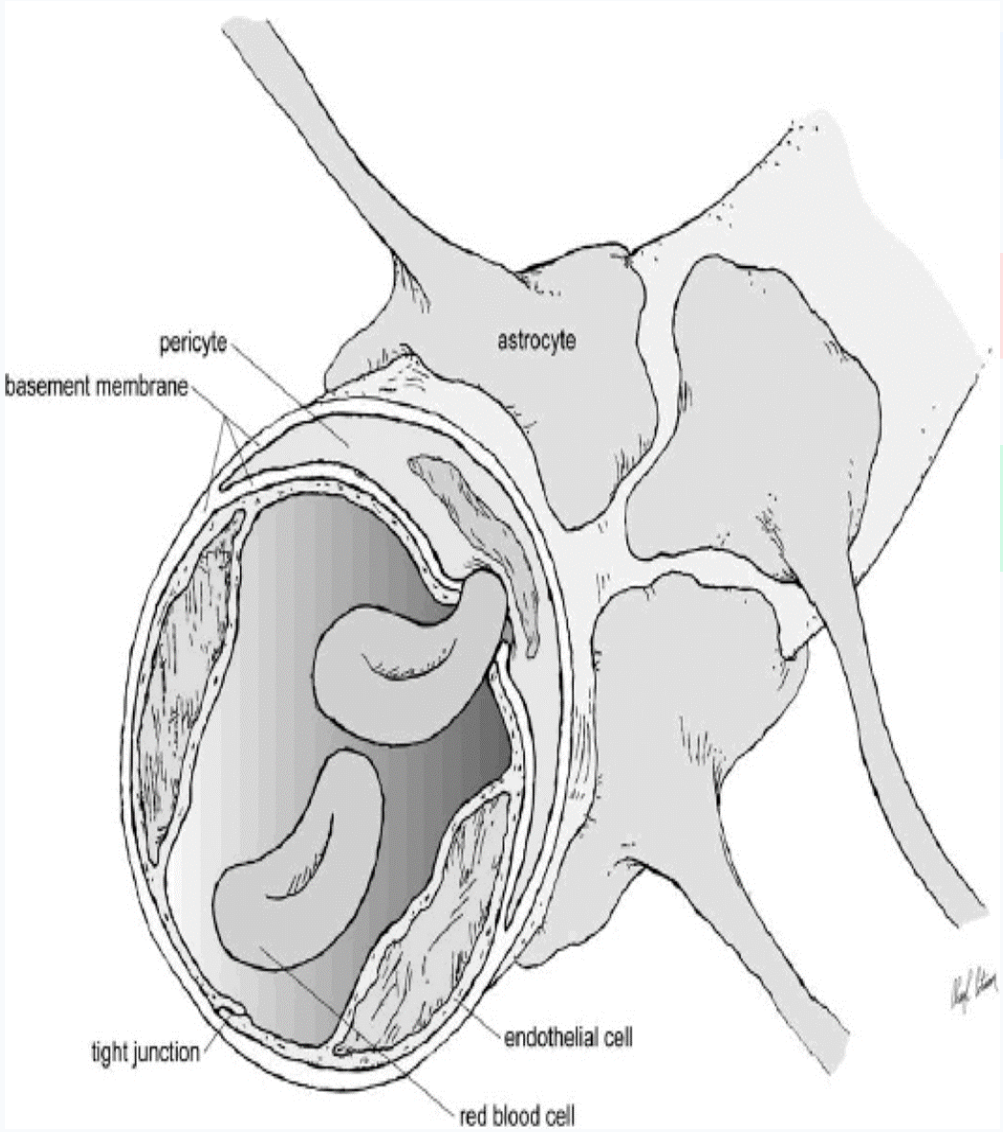
Organ-specific regulation explains why the “same” systemic coagulation change produces different vascular outcomes in different tissues

Fisher 2013; Aird 2007; Rosenberg & Aird 1999.



Brain capillaries
Endothelial tight junctions, with transmembrane molecules occludin and claudin interacting with actin cytoskeleton and zonula occludens proteins including ZO-1. (From Kim JH, Kim JH, Park JA, Lee SW, Kim WJ, Yu YS, Kim KW. Blood-neural barrier: intercellular communication at glio-vascular interface. J Biochem Mol Biol. 2006;39:339–345

Brain-specific hemostasis resides in the neurovascular unit



Structural defense

Tight junctions + adherens junctions strengthen capillary integrity and limit RBC escape.

Gatekeeper cells

Pericytes localize opposite tight junctions; astrocytes provide trophic and tissue-factor–rich support.

Clinical implication

BBB integrity is a hemostatic determinant, not merely a drug-delivery barrier

A pericyte, opposite capillary tight junction, prevents red blood cell exit while tissue-factor expressing astrocytes provide additional protection against hemorrhage.

Fisher 2013, Figures 2–3; Liu et al. 2012.

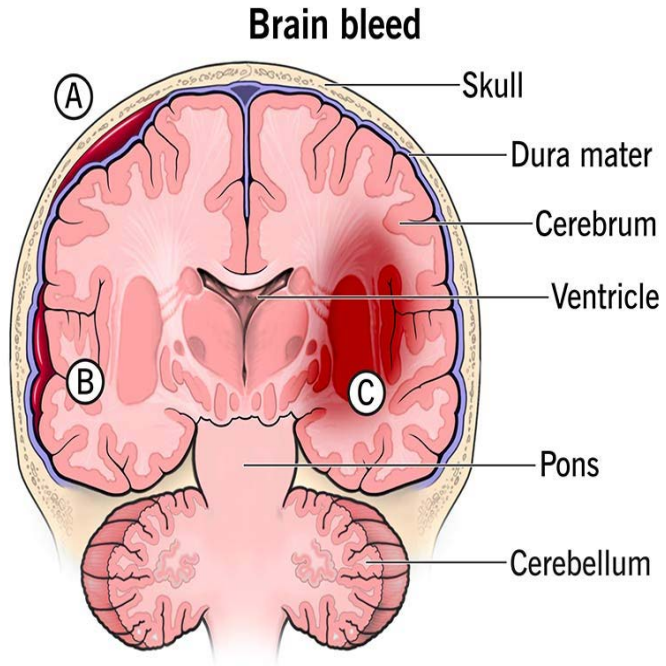
Molecular phenotype: pro-hemostatic, relatively anti-fibrinolytic

Structural Component	**Expression Level/Consequence**
Tight Junctions	Added barrier to RBC vascular escape
Pericytes	Enhance BBB and provide additional structural barrier to RBC escape
Functional Component	
Thrombomodulin	Restricted expression at BBB
Tissue Plasminogen Activator	Restricted expression at BBB
Plasminogen Activator Inhibitor-1	Enhanced expression at BBB
Tissue Factor	Enhanced expression (by astrocytes) at BBB
Tissue Factor Pathway Inhibitor	Restricted expression at BBB
Heparan Sulfate Proteoglycans	Restricted expression at BBB

The brain prioritizes protection against hemorrhage

through architecture
plus local molecular expression.

The brain's microvasculature appears less dependent on classic antithrombotic/fibrinolytic pathways and more dependent on barrier integrity and local tissue factor



Therapeutic challenge

Ischemic compartment

Clinical stroke, TIA, silent infarcts, lacunes, white matter disease.

Coexistence is common and changes the treatment question

Hemorrhagic compartment

Intracerebral hemorrhage, cerebral microbleeds, cortical superficial siderosis.

Hematologist

How do we prevent thrombosis without accelerating local microvascular hemorrhage?

Neurologist

Which lesion phenotype is driving risk: thrombotic, embolic, hypertensive, amyloid, or mixed?

coagulation intervention" in stroke is never only a blood intervention
it is also an intervention in the neurovascular unit.

Fisher 2013; Greenberg 2022 AHA/ASA ICH Guideline.

Coagulation network viewed as local control circuitry



Negative regulators

Thrombomodulin–protein C, TFPI, antithrombin–HSPG and fibrinolysis prevent uncontrolled propagation.

Cellular surfaces

Platelets, monocytes and endothelium provide catalytic surfaces and determine where thrombin is generated.

Brain translation

The BBB modifies this circuit by restricting anticoagulant and fibrinolytic expression while reinforcing hemostatic barriers.

the cascade is not only plasma biochemistry — it is vascular-bed biology

Fisher 2013; Mackman 2005.

Questions

Molecular aging of BBB hemostasis

Which age-dependent changes alter thrombomodulin, TFPI, HSPG, tPA/PAI-1 and tissue factor?

Biomarker–MRI correlations

Can plasma, endothelial, platelet or inflammatory markers predict bleeding/thrombosis burden/progression?

Better models

Animal models need to reproduce diffuse microbleeds, microinfarcts and BBB dysfunction reliably.

Vessel-wall therapies

Evaluate endothelial/BBB protection as a co-primary target alongside platelet/coagulation inhibition.

Fisher 2013; Chea et al. 2024 review.

Take-home messages

1. The brain has a specialized hemostatic system embedded in the BBB and neurovascular unit.

2. This system appears biased toward hemorrhage prevention through tight junctions, pericytes, astrocytic tissue factor and constrained fibrinolysis.

3. **treat the patient’s hemostatic system — not only the coagulation test**

4

Synthesis based on Fisher 2013 and contemporary ICH guidance.

Selected references

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6. Wardlaw JM, et al. European Stroke Organisation guideline on cerebral small vessel disease, part 2. *Eur Stroke J. 2024.*
7. Chea M, et al. The hemostatic system in chronic brain diseases: a new avenue for therapeutic targets. *Thromb Res. 2024.*

Note: This deck is for education and conference discussion; clinical decisions require patient-specific neurologic, hematologic and imaging assessment.

Thank you