A hemosztázis…

- *kórélettana és diagnosztikája* -

- Dr. Tánczos Krisztián -

Aneszteziológiai és Intenzív Terápiás Intézet

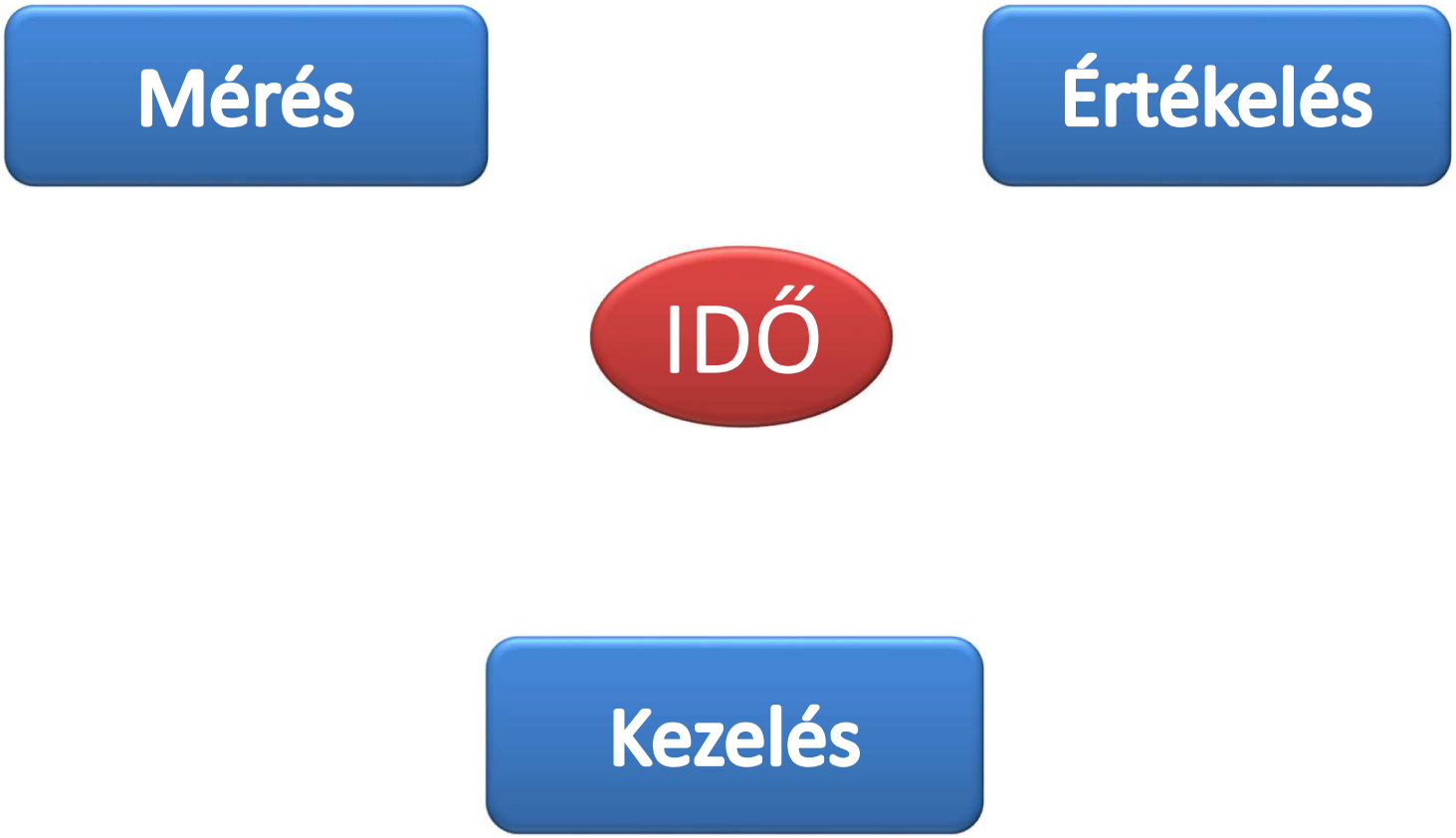
XVI. Magyar Sürgősségi Orvostani Kongresszus

MSOTKE - MOT közös tudományos rendezvénye

Siófok, 2017. november 16-18.

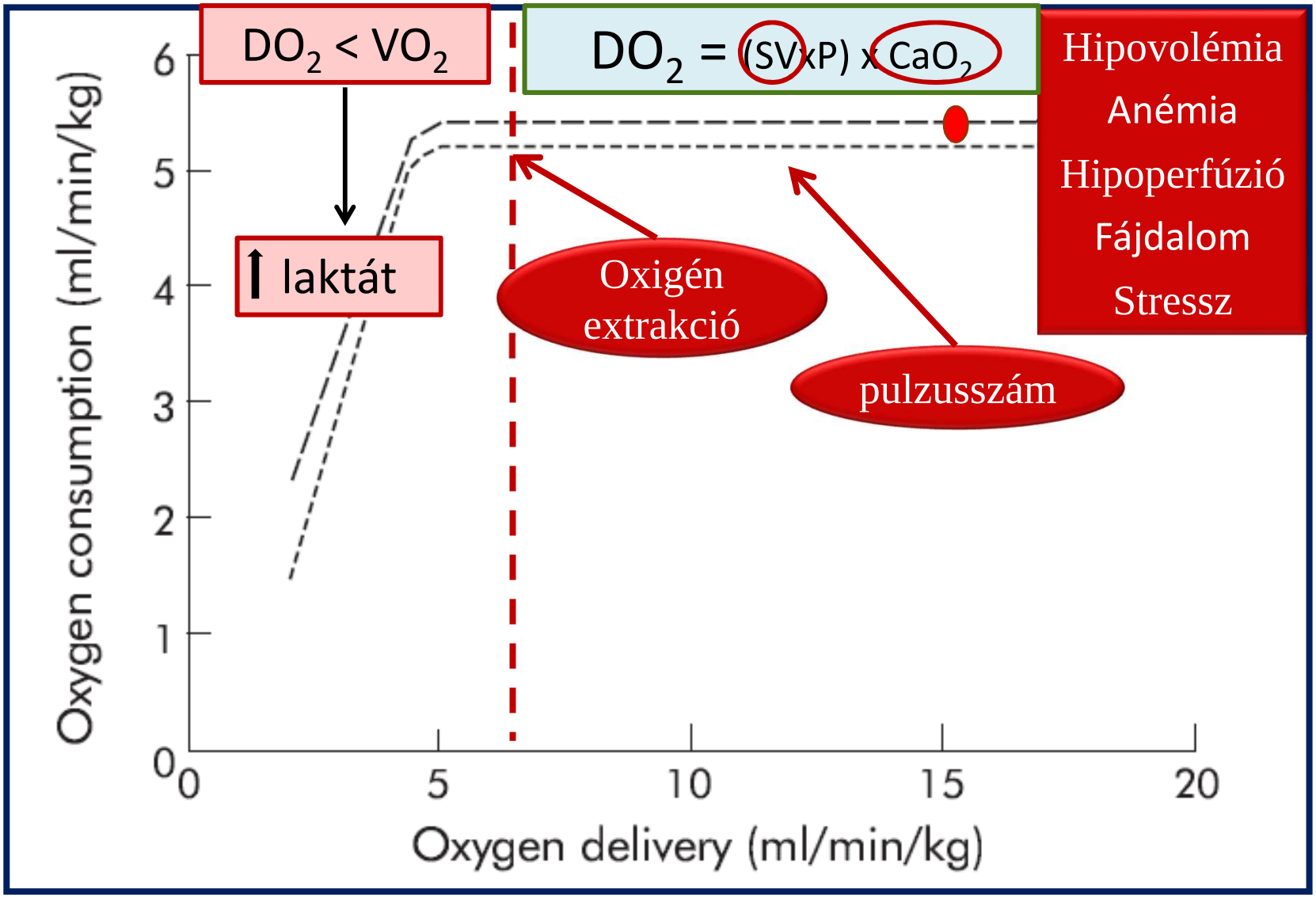
Dinamikus szemlélet

- kritikus állapot -



Súlyos vérzés = oxigénadósság

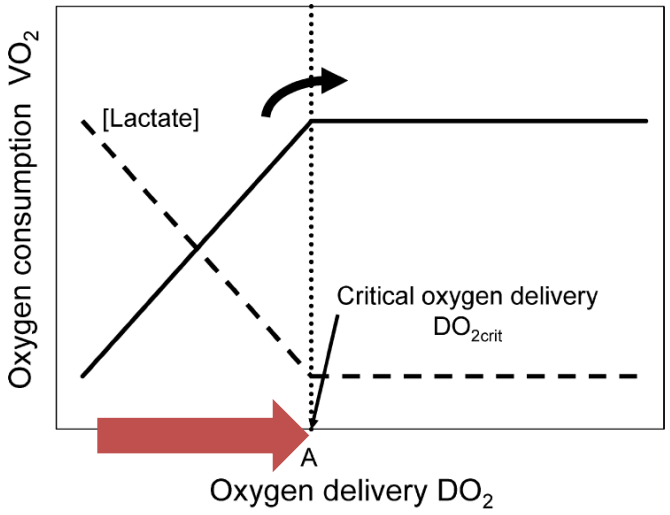
Vincent JL et al. *Int. Care Med* 1990; 16:145–148

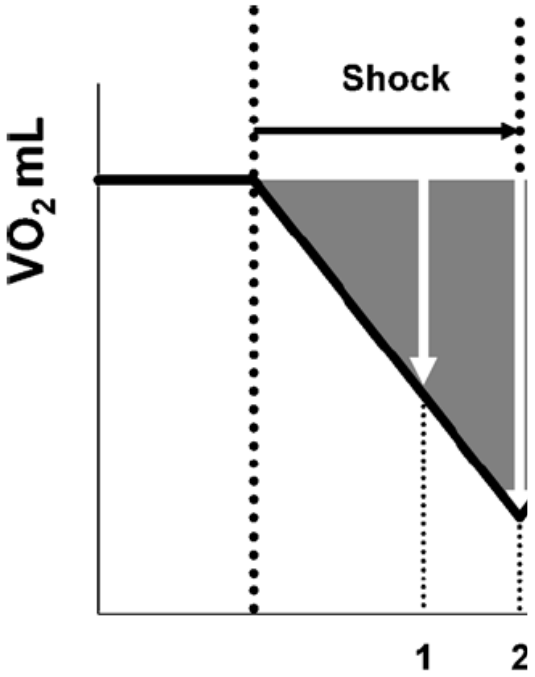


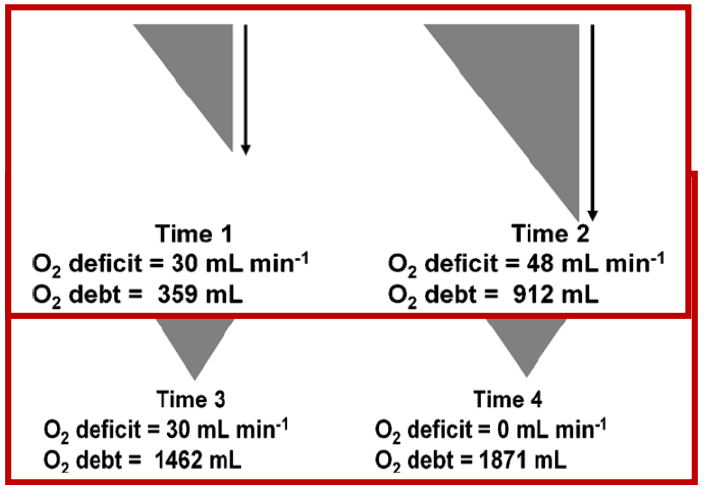
l. 33, No. 2, pp. 113–122, 2010

ASSES EGIES

Robert Wayne in R. Ward*†‡§
Departments of *Em ginia Commonwealth
University nonwealth







Review Article

SHOCK, Vol. 33, No. 2, pp. 113–122, 2010

ASSESSING SHOCK RESUSCITATION STRATEGIES BY OXYGEN DEBT REPAYMENT

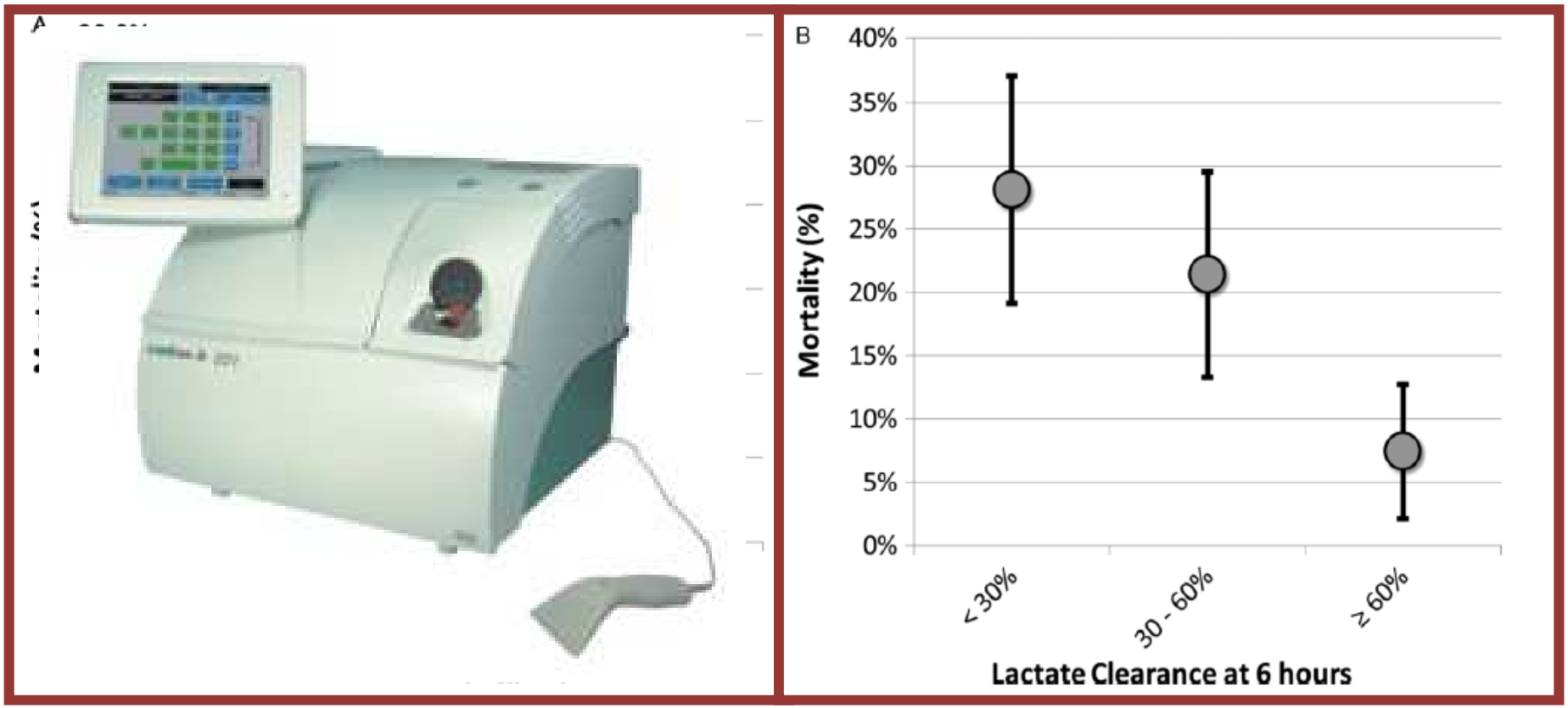
Robert Wayne Barbee,*†§ Penny S. Reynolds,*§ and Kevin R. Ward*†‡§

*Departments of *Emergency Medicine, †Physiology, ‡Biochemistry, and §Virginia Commonwealth University Reanimation Engineering Shock Center, Virginia Commonwealth University Medical Center, Richmond, Virginia*

Lactate clearance as a predictor of mortality in trauma patients

Stephen R. Odom, MD, Michael D. Howell, MD, MPH, George S. Silva, BA, Victoria M. Nielsen, Alok Gupta, MD, Nathan I. Shapiro, MD, MPH, *and* Daniel Talmor, MD, MPH, *Boston, Massachusetts*

J Trauma Acute Care Surg. 2013;74:999-1004



Review Article

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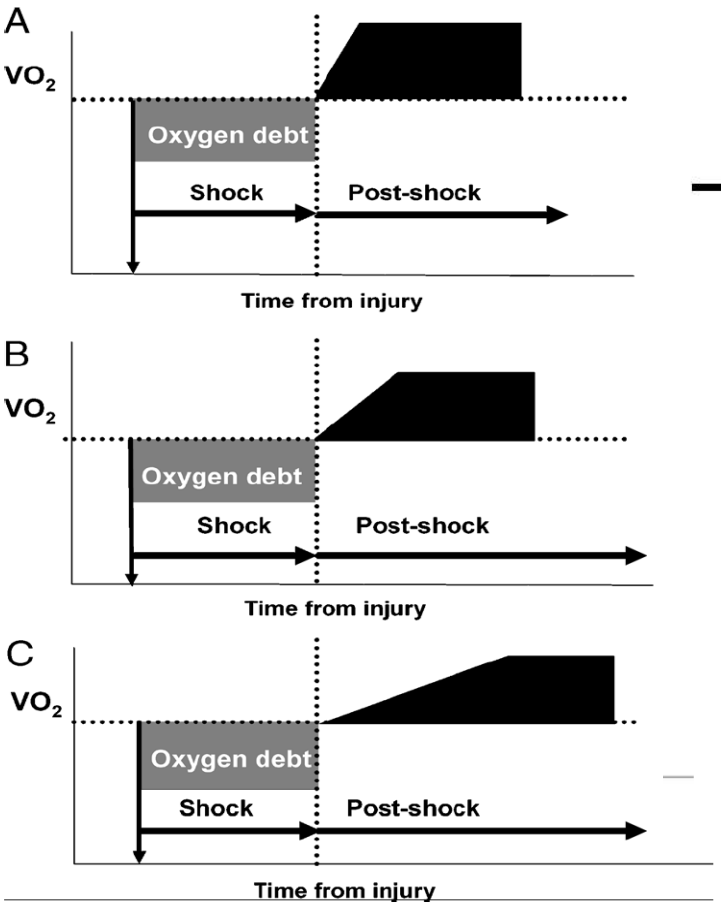
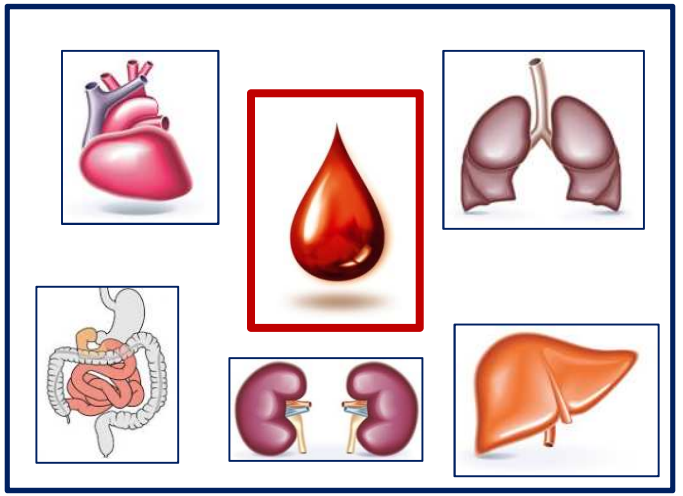


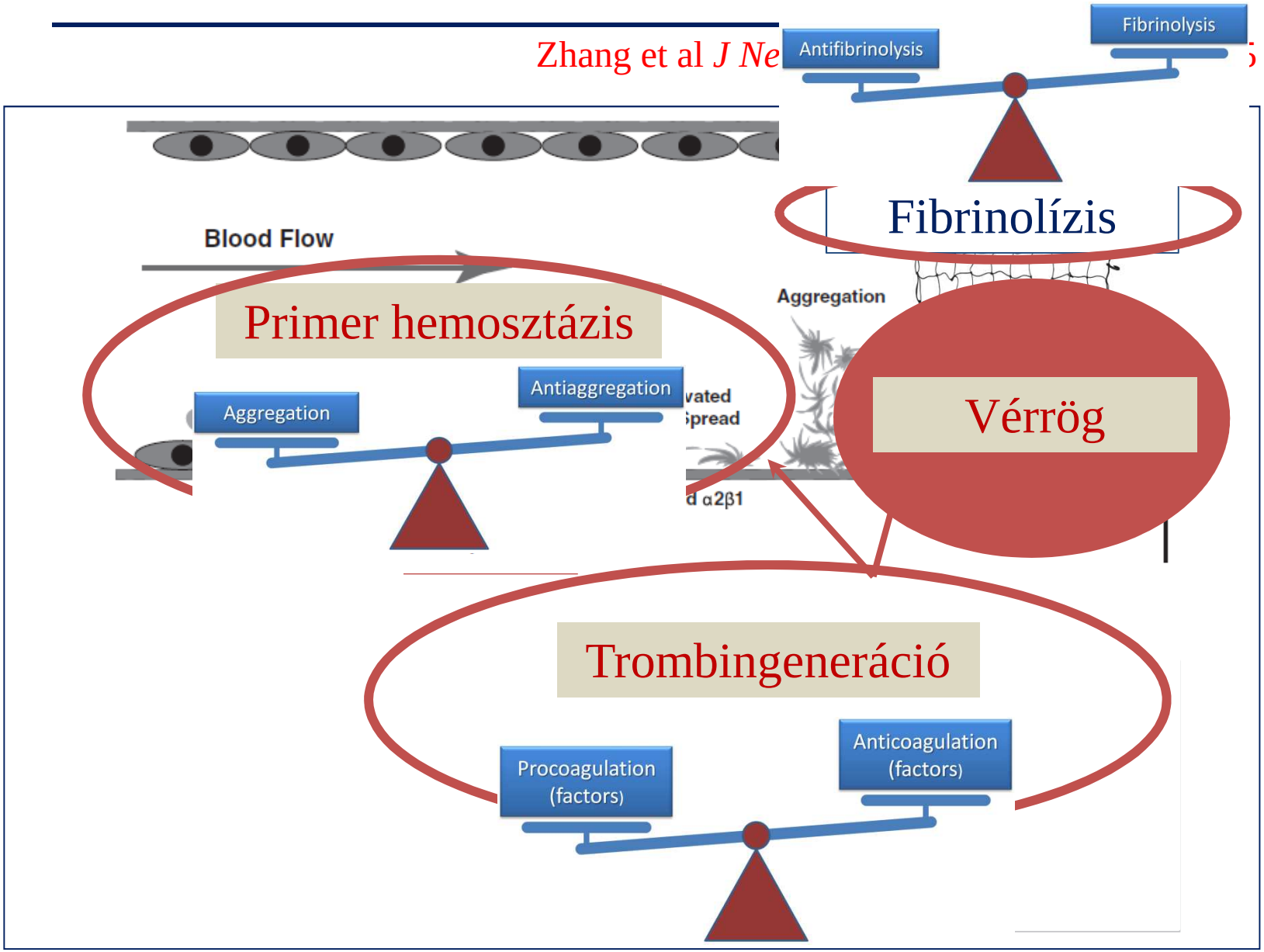
Szervelégtelenség

„VÉRELÉGTELENSÉG”



A véralvadás alapjai…

Zhang et al *J Ne*



Szöveti sérülés
(Trauma, műtét)

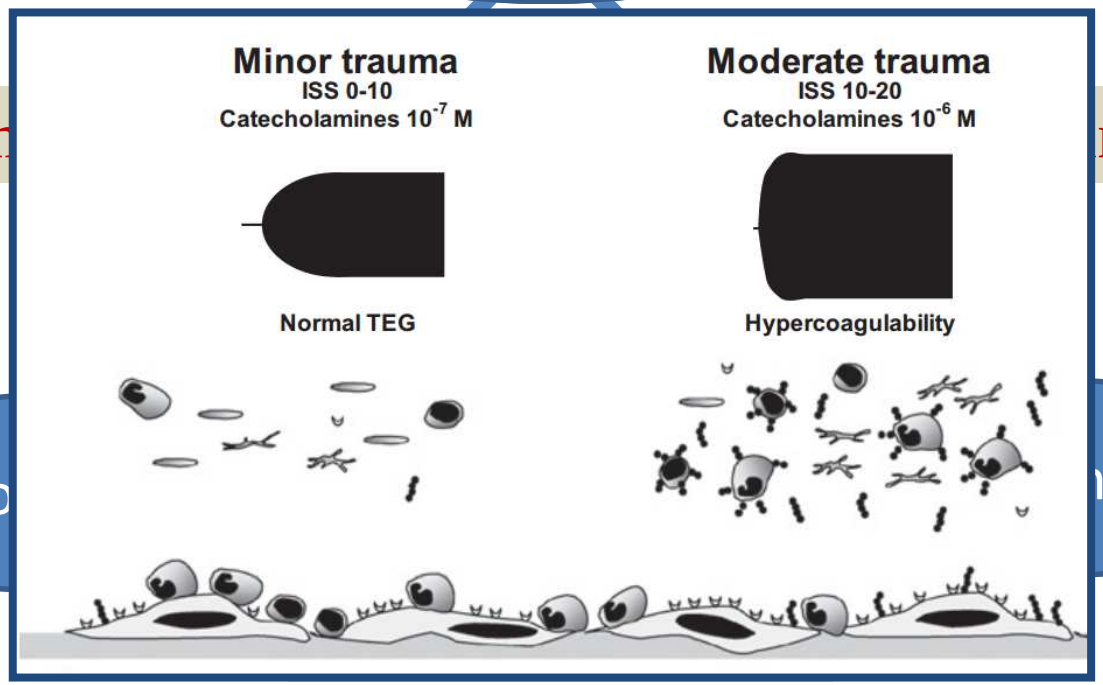
Inflammáció
(fertőzés, égés..)

Katekolamin

Endothél
aktiváció

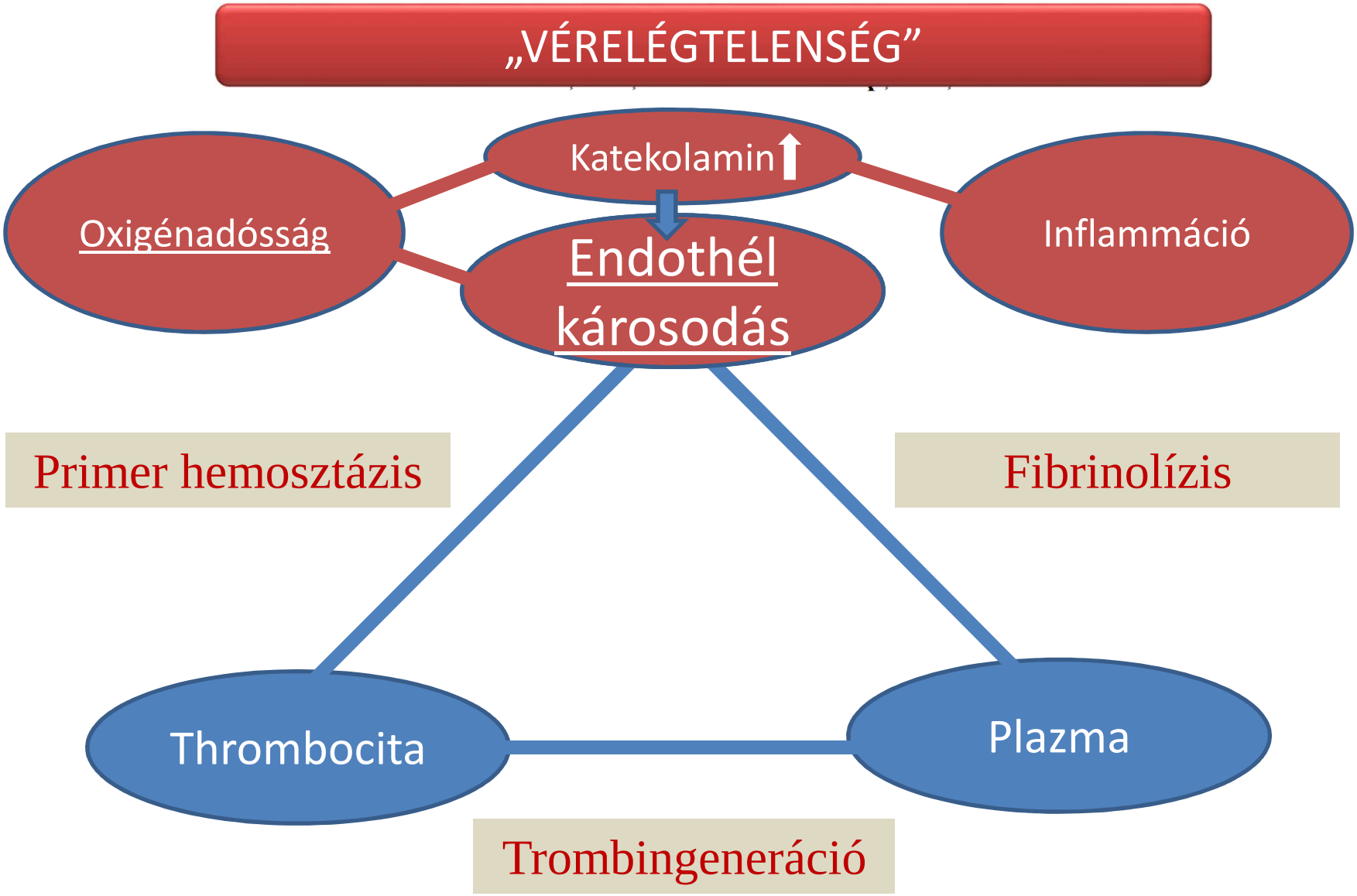
Primer hem nolízis

Thro na



Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

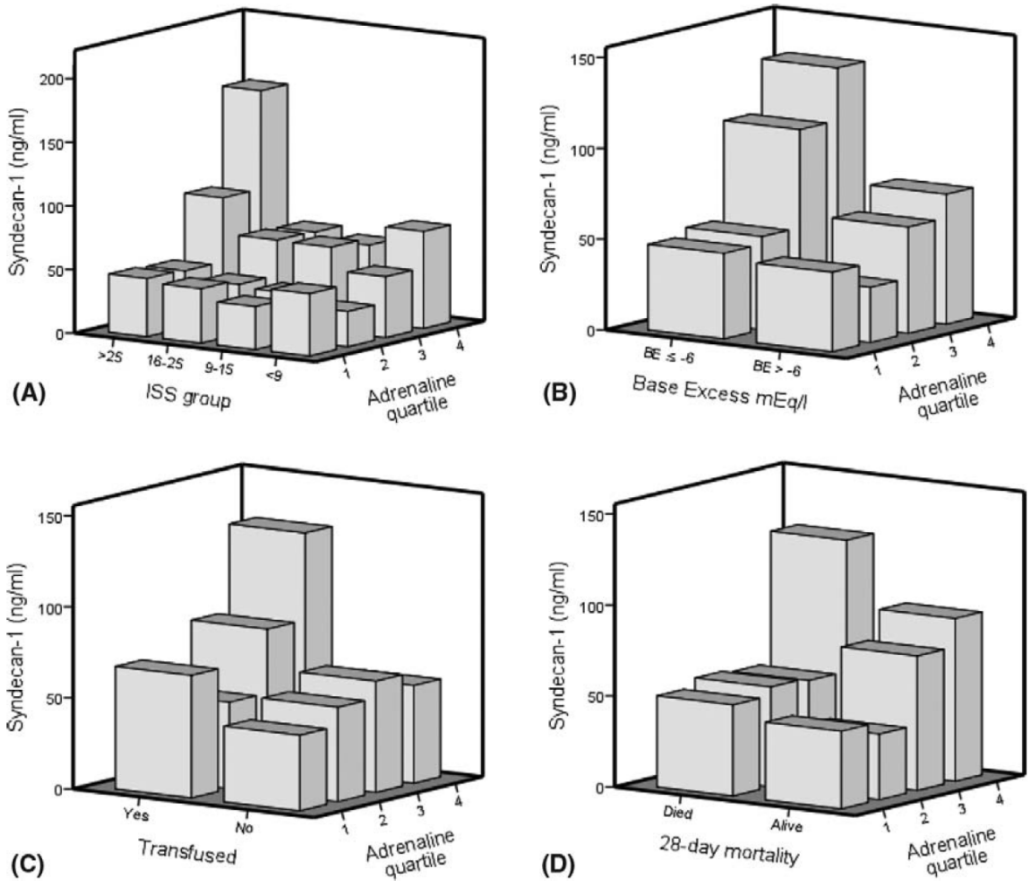
J Trauma Acute Care Surg. 2017;82:6,S41-49



Traumatic Endotheliopathy: A Prospective Observational Study of 424 Severely Injured Patients

Pär I. Johansson, MD, DMSc, MPA,† Hanne H. Henriksen, BSc,* Jakob Stensballe, MD, PhD,*‡ Mikkel Gybel-Brask, MD,* Jessica C. Cardenas, PhD,† Lisa A. Baer, BSc,† Bryan A. Cotton, MD, MPH,† John B. Holcomb, MD,† Charles E. Wade, PhD,† and Sisse R. Ostrowski, MD, PhD, DMSc**

(*Ann Surg* 2017;265:597–603)

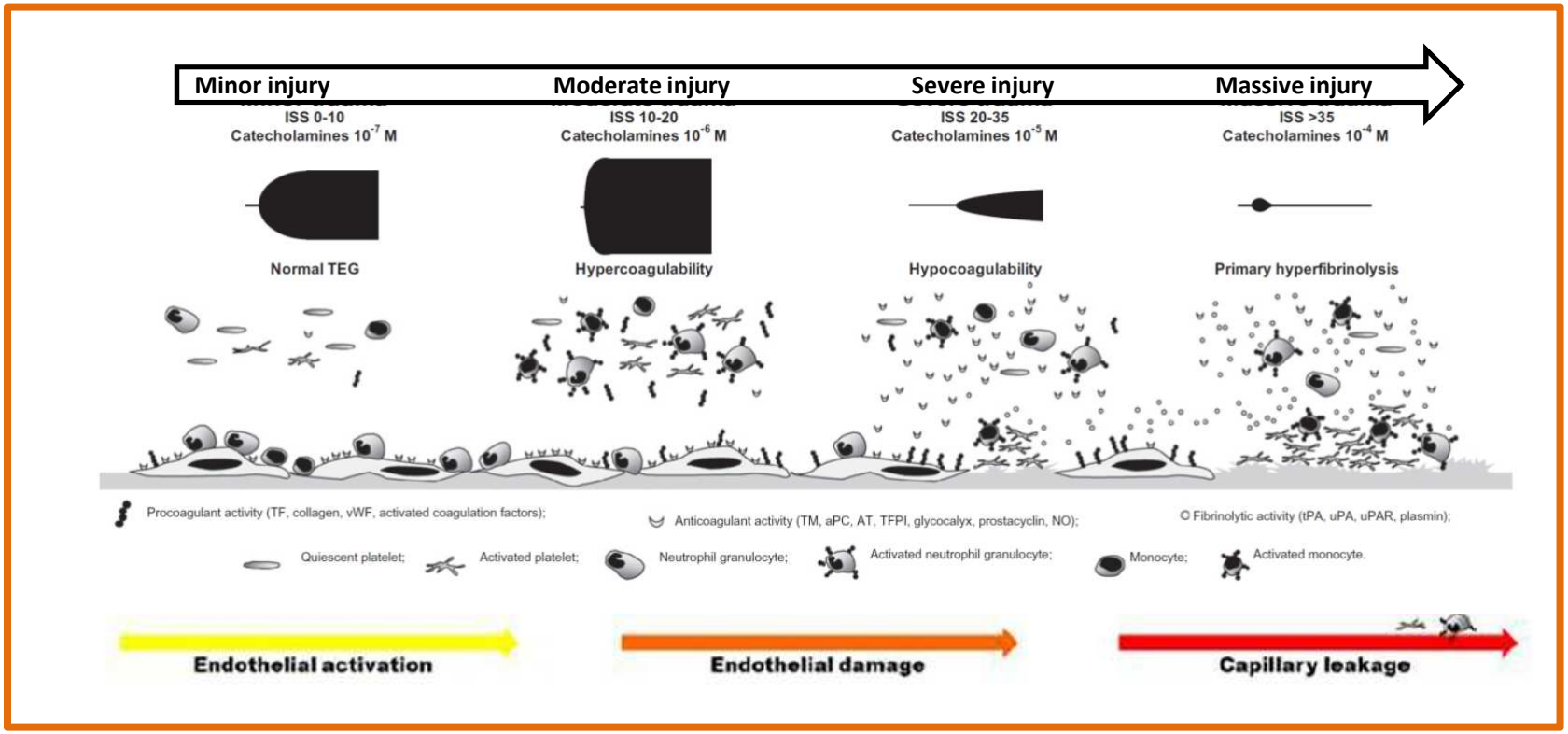


REVIEW Open Access

Shock induced endotheliopathy (SHINE) in acute critical illness - a unifying pathophysiologic mechanism

PärIngemar Johansson[1,2,3*], Jakob Stensballe[1,4] and SisseRye Ostrowski[1]

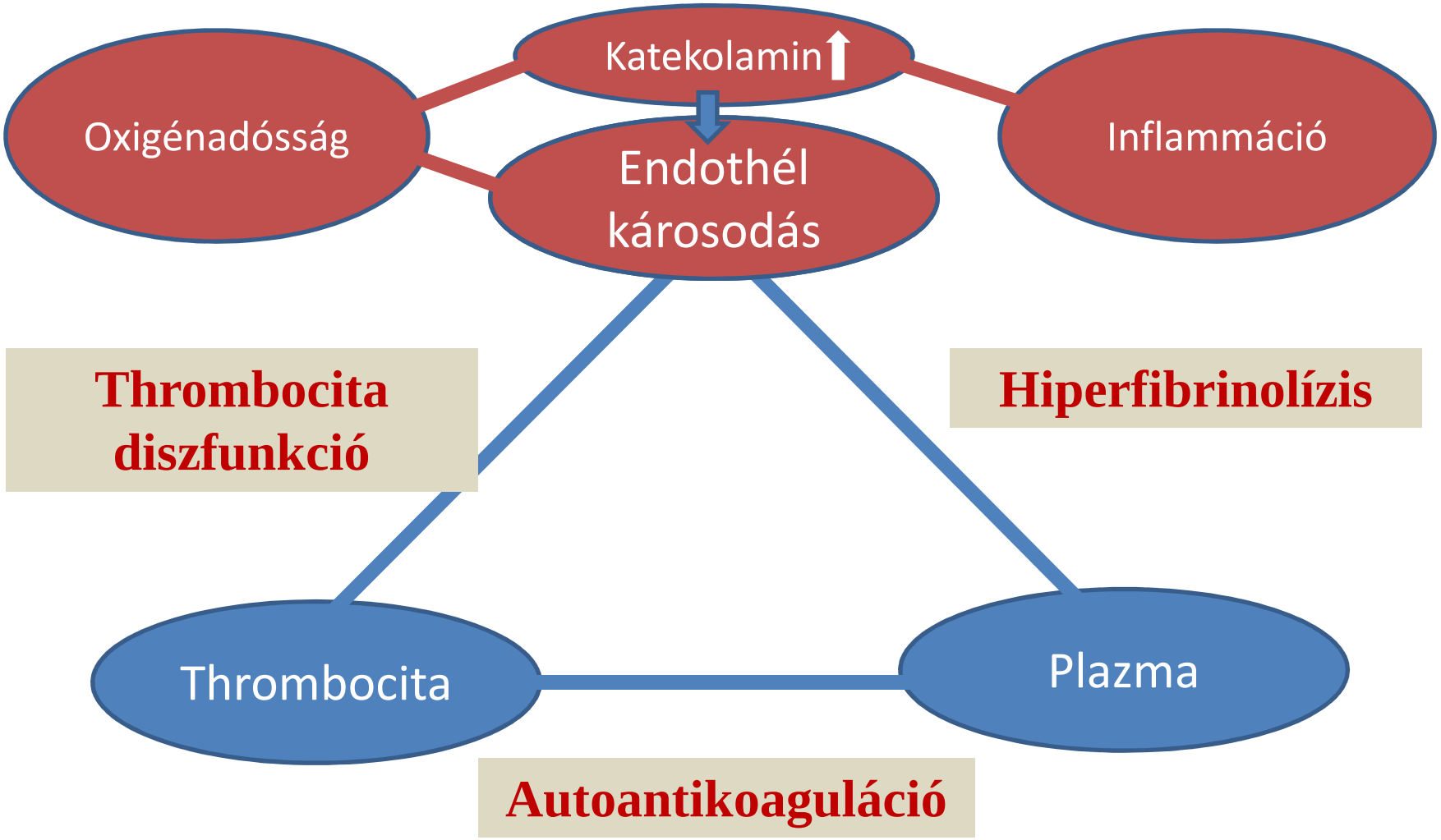
Critical Care (2017) 21:25 DOI 10.1186/s13054-017-1605-5



Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

J Trauma Acute Care Surg. 2017;82:6,S41-49

Nathan J. White, MD, MS, Kevin R. Ward, MD, Shibani Pati, MD, PhD, Geir Strandenes, MD, *and* Andrew P. Cap, MD, PhD



Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

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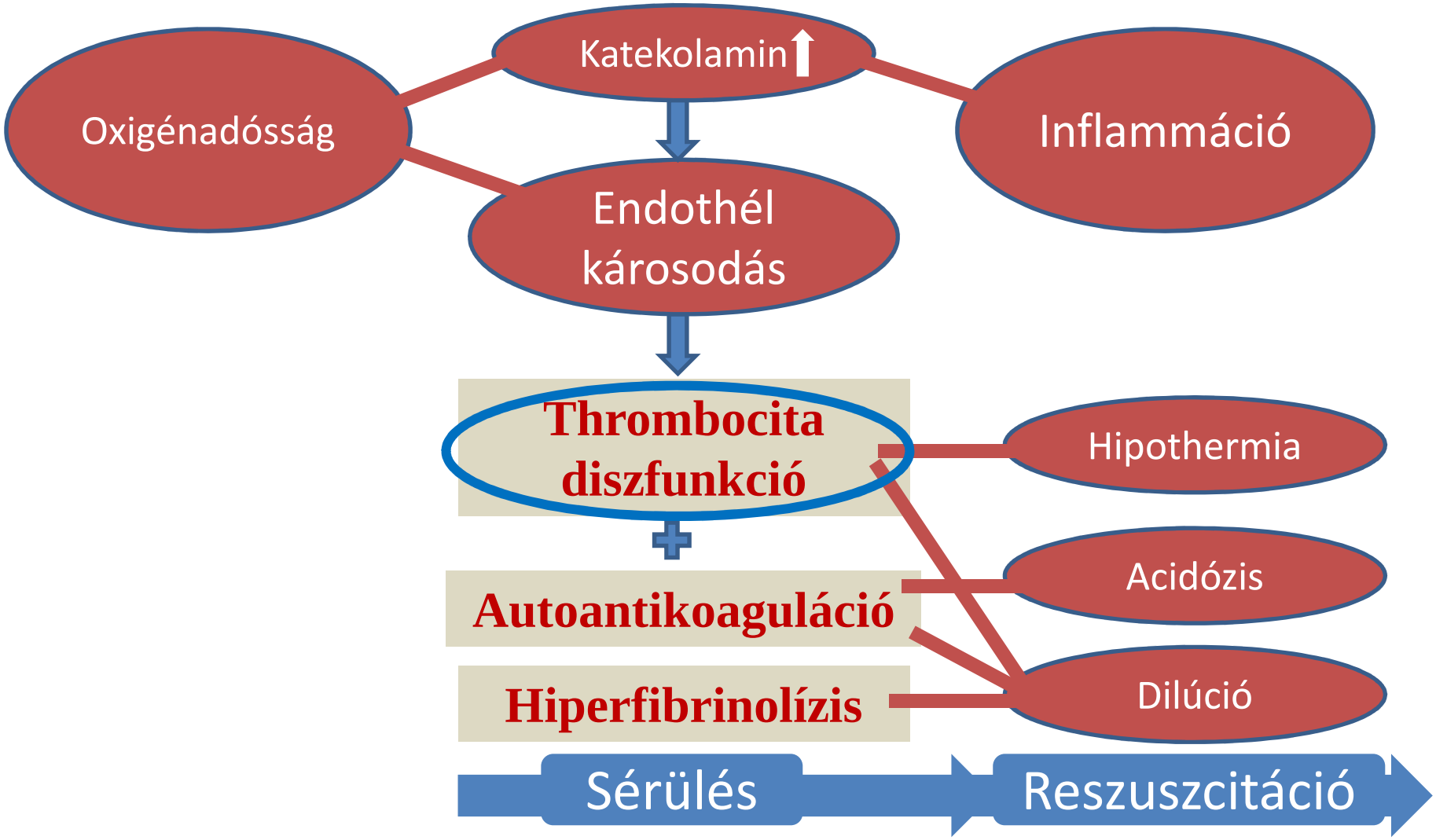
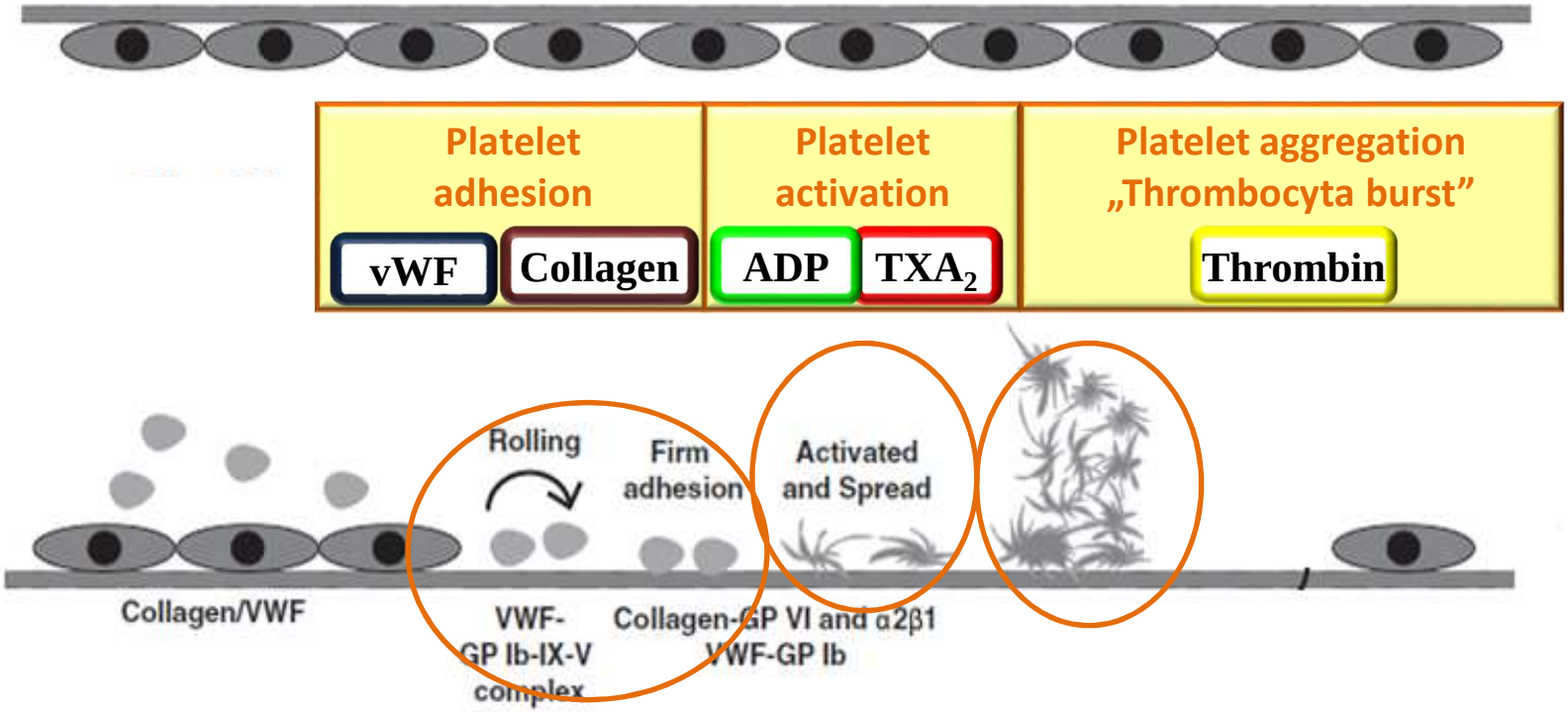
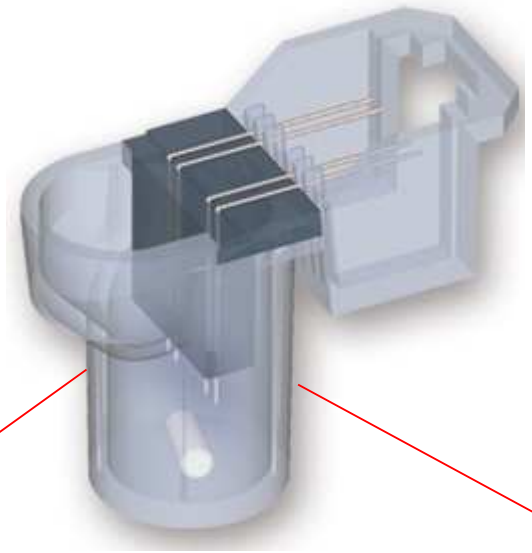


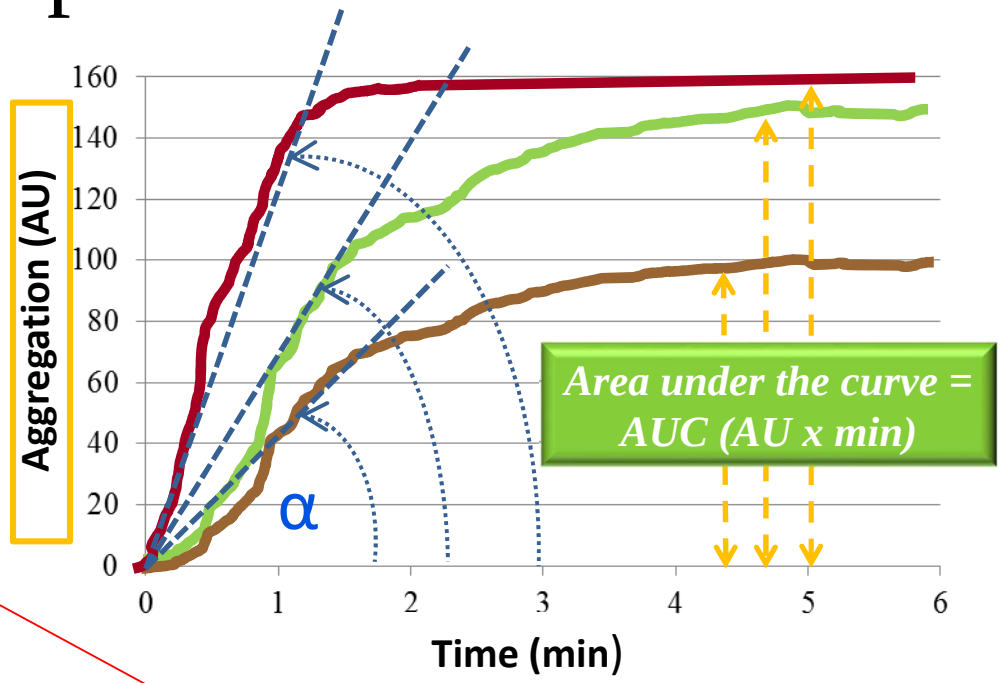
Thrombocitafunkció

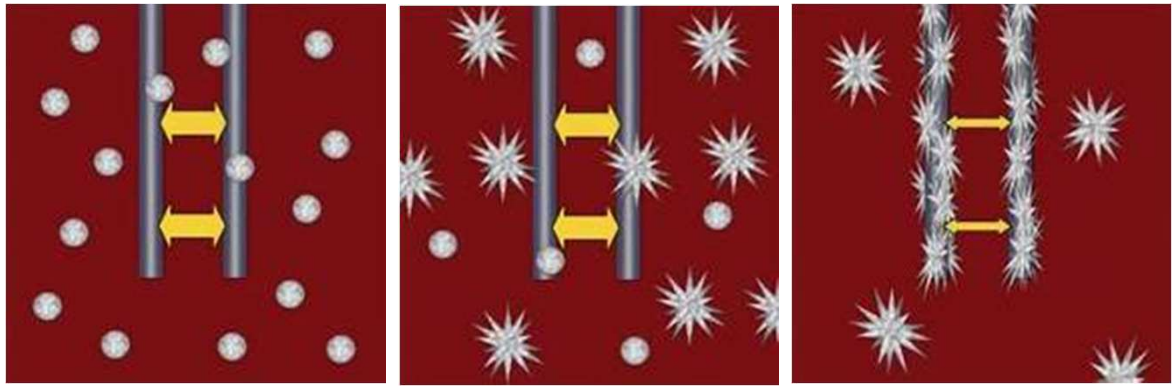
Zhang et al *J Neurotrauma* 2012; 29:2597–2605

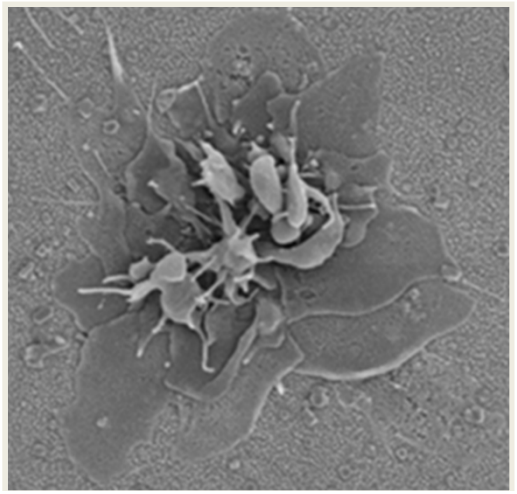


Multiple Electrode Aggregometry (MEA)
Multiplate®



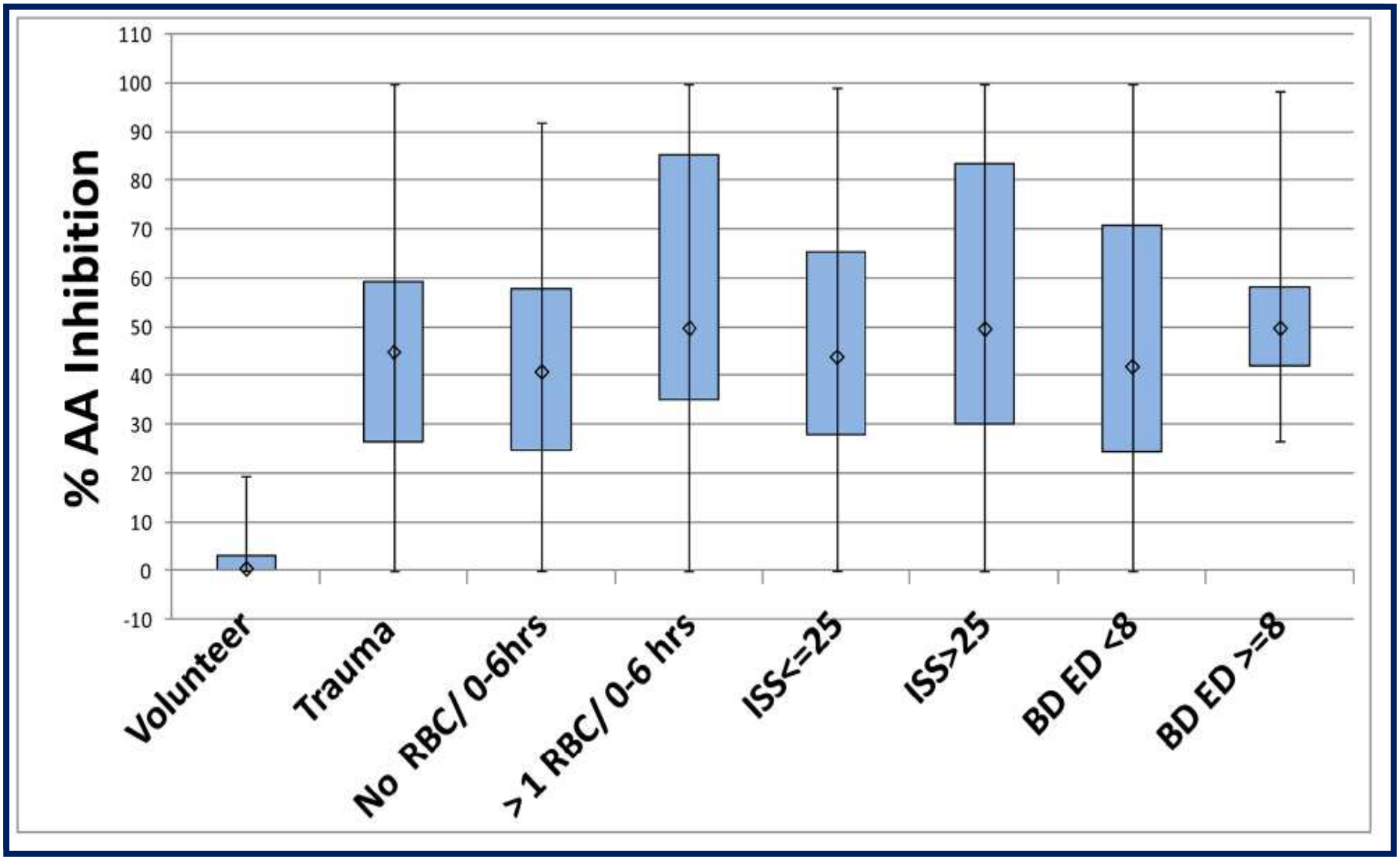




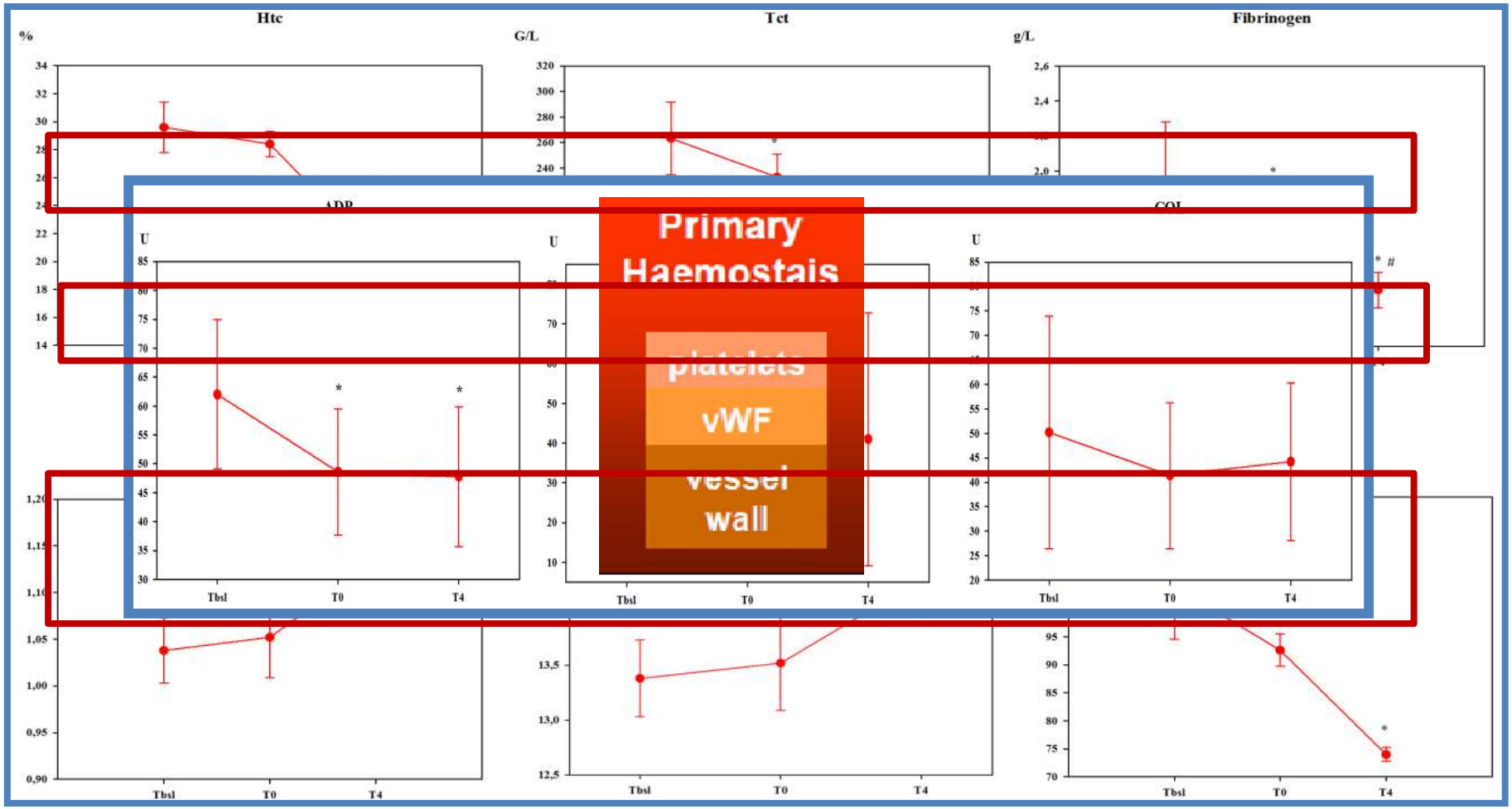


Early Platelet Dysfunction: An Unrecognized Role in the Acute Coagulopathy of Trauma

Wohlauer et al. J Am Coll Surg. 2012; 214(5): 739–746



Mérsékelt hipoperfúzió esetén….!



ORIGINAL ARTICLE

Sirajuddin et al. *J Trauma Acute Care Surg. 2016;81: 328–332.*

Inhibition of platelet function is common following even minor injury

Sarah Sirajuddin, MD, Carrie Valdez, MD, Louis DePalma, MD, Patrick Maluso, MD, Rishi Singhal, Mary Schroeder, MD, *and* **Babak Sarani, MD**, *Washington, District of Columbia*

METHODS: A retrospective study of adult trauma patients evaluated at a Level I trauma center from August 2013 to September 2014 was conducted. Platelet function was assessed using TEG-PM. Patients on any anticoagulant or antiplatelet medication were excluded. Patients were divided into those with and without radiographically evident traumatic brain injury (TBI). Demographic variables, Injury Severity Score (ISS), injury pattern, laboratory test results, and mortality were abstracted. Statistical comparisons were made using the Student's *t* test or Mann-Whitney U-test.

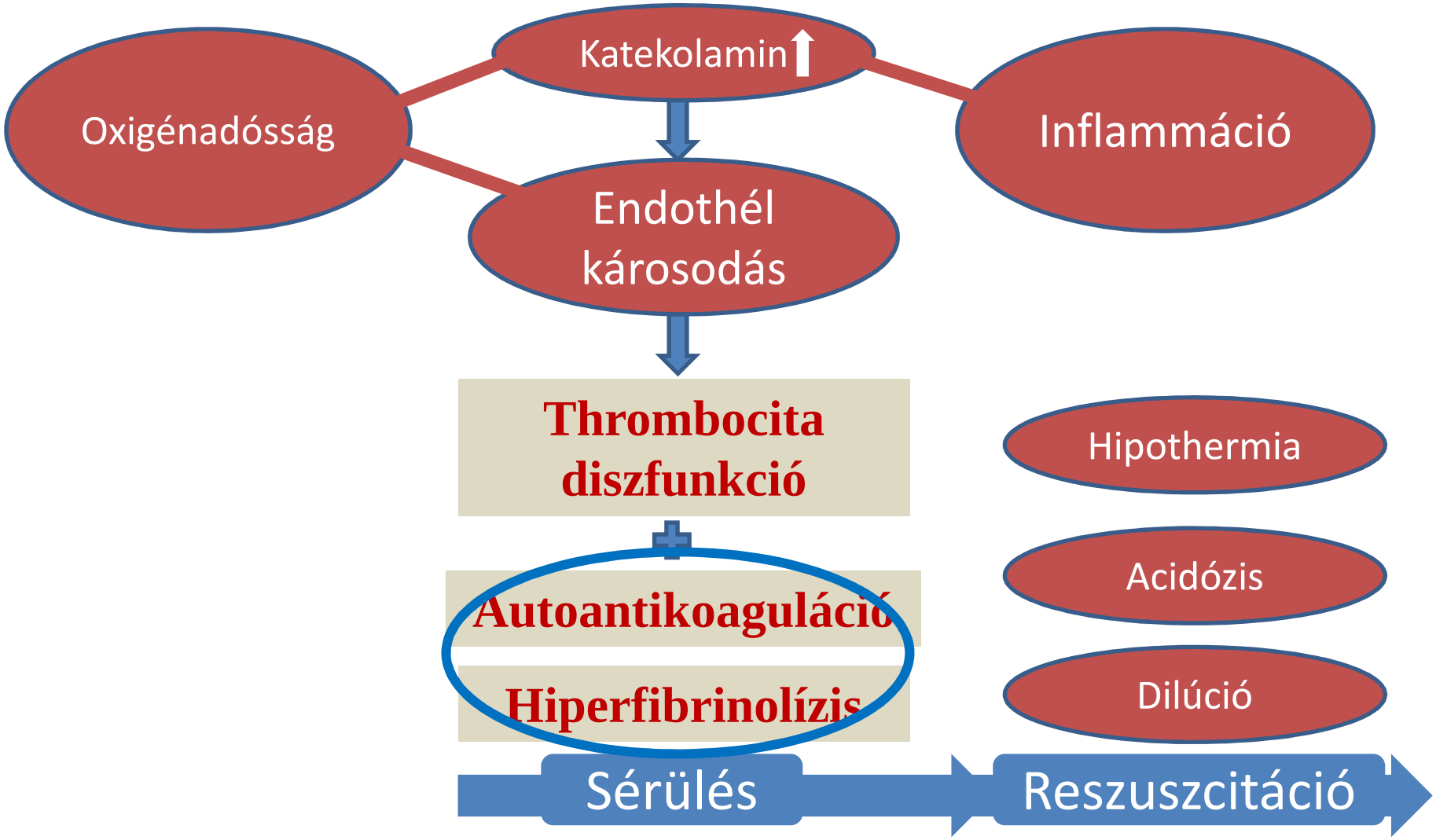
RESULTS: The study includes 459 patients, 92% following blunt injury. Median ISS was 5. Patients with TBI (n = 102) were significantly older (median age, 54 years vs. 35 years), were more severely injured (median ISS, 10 vs. 4), had a longer stay and higher mortality (9% vs. 0.3%). Maximal potential clot strength was normal in all cohorts, but the arachidonic acid and adenosine diphosphate pathways were significantly inhibited (30% ± 26% and 58% ± 27%, respectively). There was no correlation between TEG-PM values and ISS, length of stay, or mortality. There was no difference in the TBI cohort. There were no significant differences in TEG-PM parameters in those with an ISS greater than 14. There was no significant change in TEG-PM following platelet transfusion.

CONCLUSION: Marked platelet inhibition is common following minor injury. Whereas the clinical significance of this finding remains unknown, the results of this study should be factored in the overall resuscitative strategy. (*J Trauma Acute Care Surg.* 2016;81: 328–332.

Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

J Trauma Acute Care Surg. 2017;82:6,S41-49

Nathan J. White, MD, MS, Kevin R. Ward, MD, Shibani Pati, MD, PhD, Geir Strandenes, MD, *and* Andrew P. Cap, MD, PhD

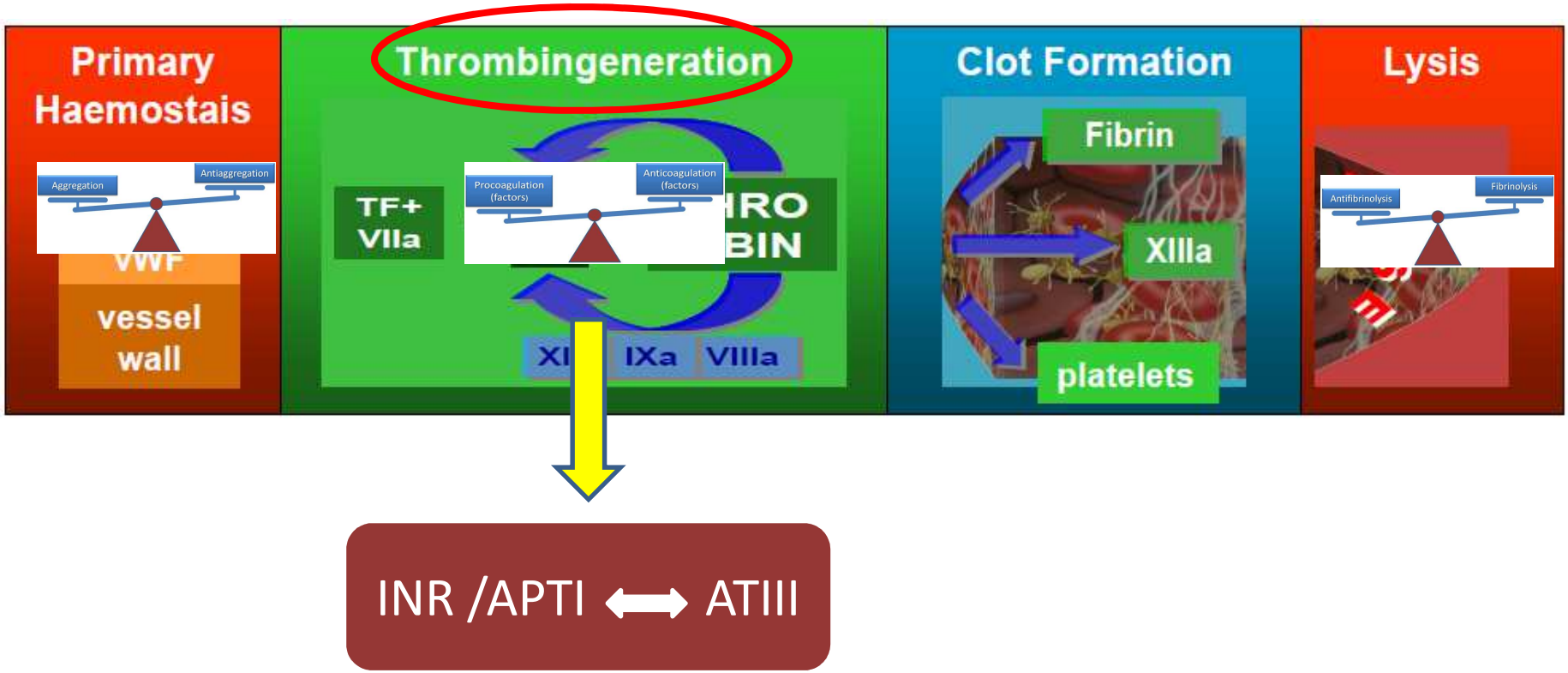


Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

Nathan J. White, MD, MS, Kevin R. Ward, MD, Shibani Pati, MD, PhD, Geir Strandenes, MD, *and* Andrew P. Cap, MD, PhD

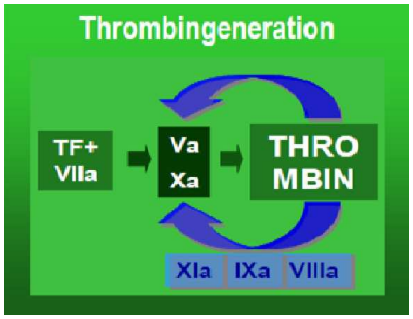
Konvencionális tesztek

Bolliger D et al Transfusion Medicine Reviews 2012; 26: 1-13

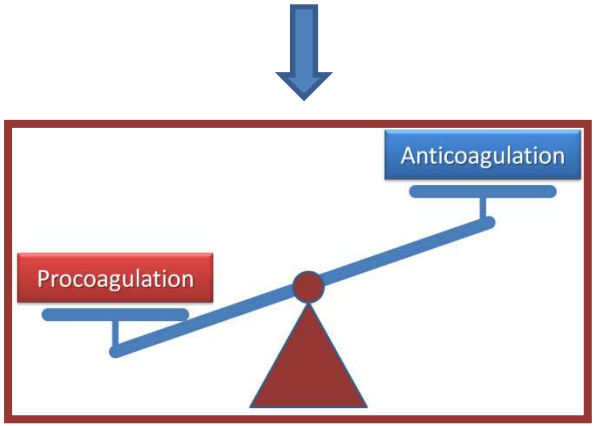


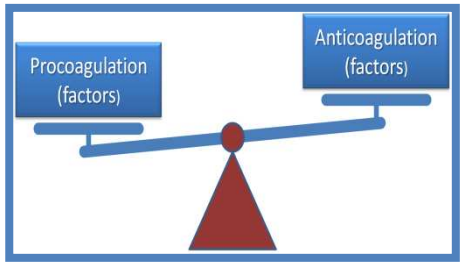
INR = 2.5 ?

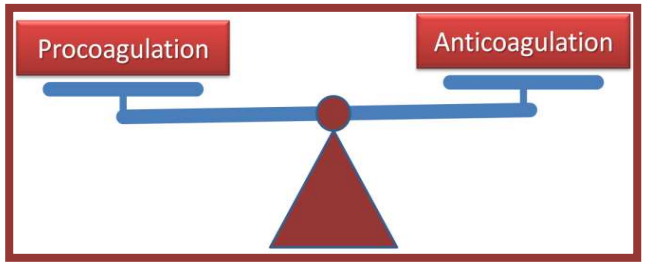
Orális antikoagulánst (warfarint) szedő beteg



Kompenzált májcirrózisban szenvedő beteg (Child B)







Jobban vérzik

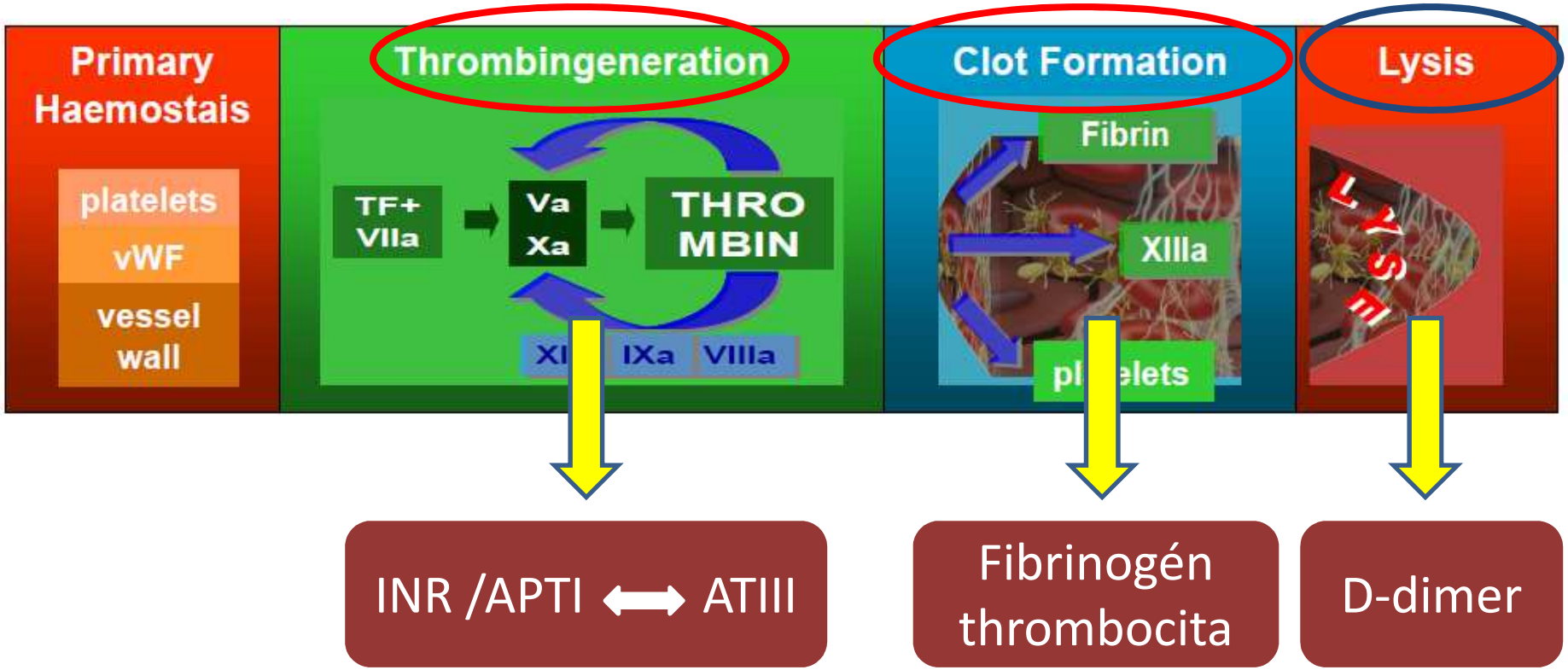
PCC terápia (FFP)
(koponyasérült !!)

Nem vérzik jobban,
DE: nincs tartaléka

FFP + PCC terápia

Konvencionális tesztek

Bolliger D et al Transfusion Medicine Reviews 2012; 26: 1-13



Preoperatí

v időszak

Folyadékre
(2+2 E
Nincs szervel

gázcsere
kvő
in igény

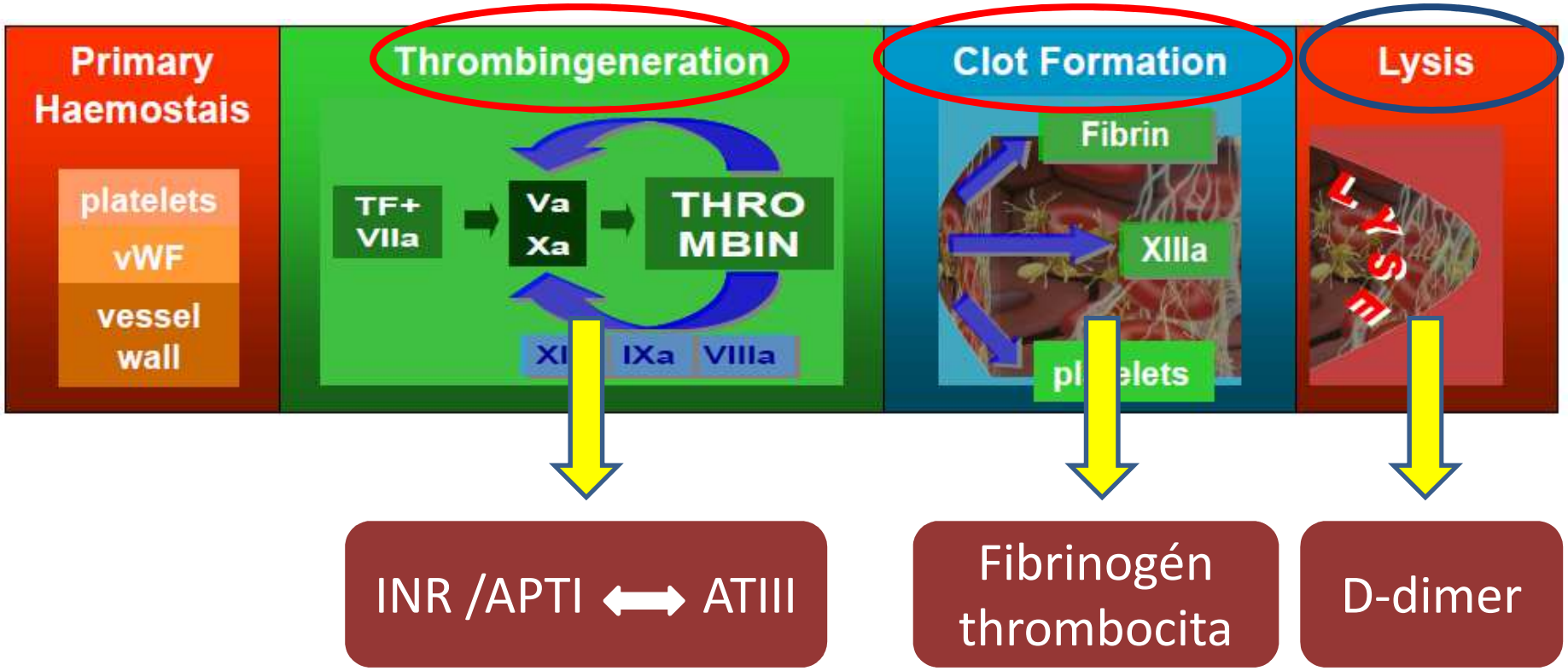
Labor paraméter	
INR	
APTI	
ATIII	
TI	
Fibrinogén	
D-Dimer	1.54 ug/ml
Tct	107 G/l

Tranexámsav
Fibrinogén + FFP

érték
1.38
49.5 s
64 %
19.2 s
1.99 g/l
D-Dimer	10.54 ug/ml
Tct	99 G/l

Konvencionális tesztek

Bolliger D et al Transfusion Medicine Reviews 2012; 26: 1-13



Gyors – Dinamikus ?

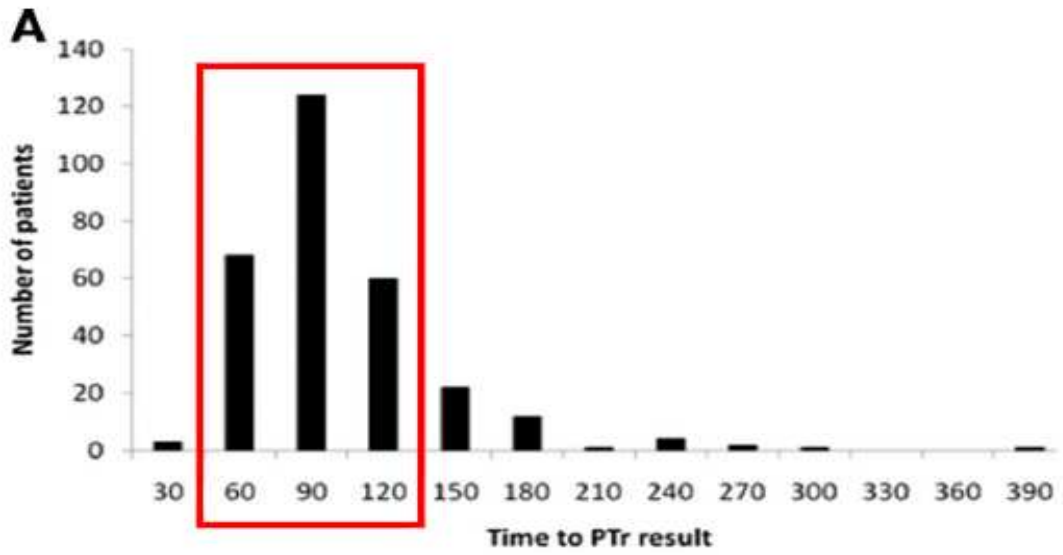
Konvencionális tesztek

Functional definition and characterization of acute traumatic coagulopathy

Crit Care Med 2011 Vol. 39, No. 12

Ross Davenport, BSc, MD, MRCS; Joanna Manson, MD, MRCS; Henry De'Ath, MD, MRCS; Sean Platton, MSc, CSci, FIBMS; Amy Coates, BSc; Shubha Allard, MD, FRCP, FRCPath; Daniel Hart, MD; Rupert Pearse, MD, FRCA; K. John Pasi, PhD, FRCP, FRCPath, FRCPCH; Peter MacCallum, MD, FRCP, FRCPath; Simon Stanworth, DPhil, MRCP, FRCPath; Karim Brohi, FRCA, FRCS

Main Results: **Three hundred patients were included in the study. Laboratory prothrombin time results were available at a median of 78 (62–103) mins.**



Optimális alvadási teszt:

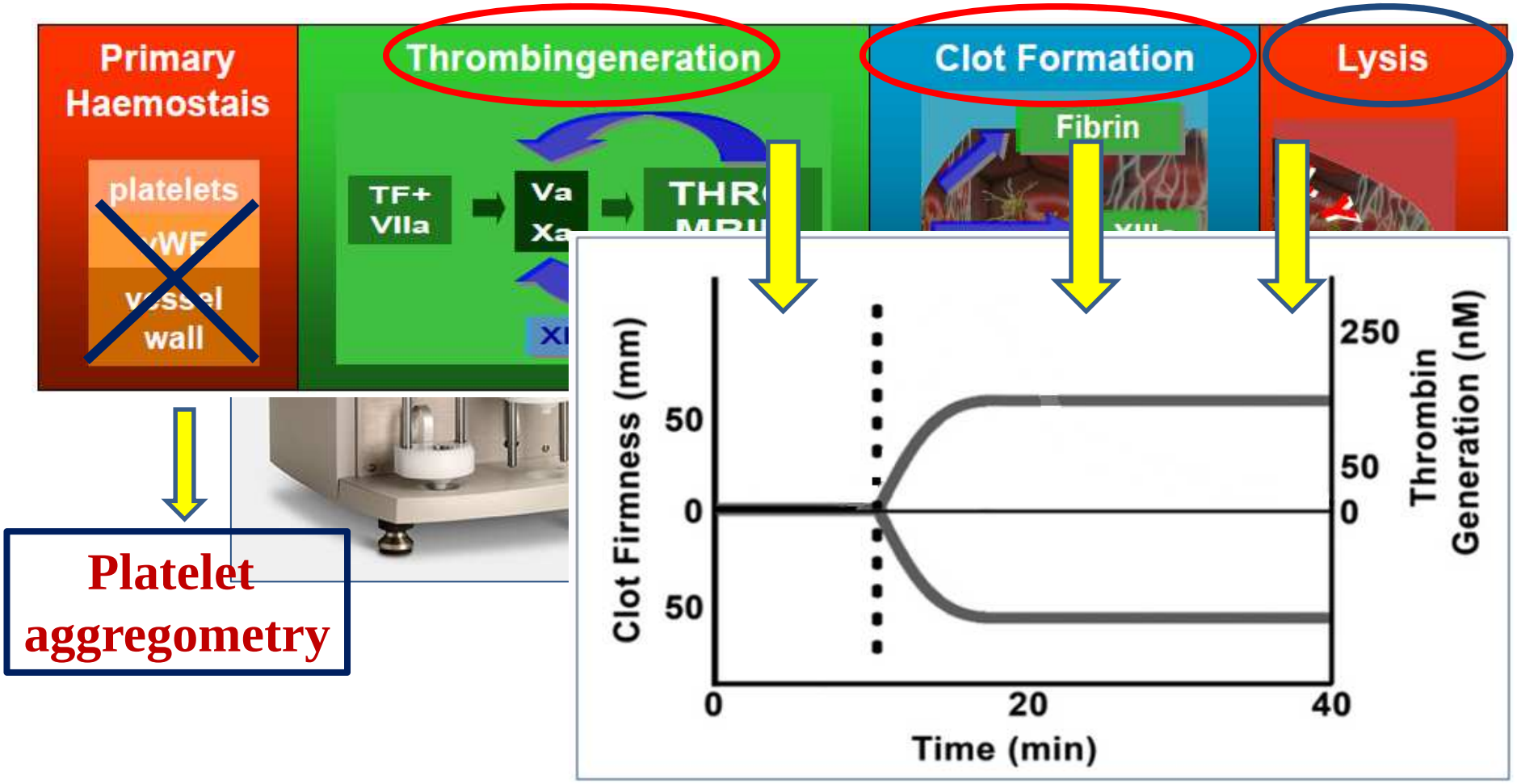
- súlyos vérzés esetén -

Gyors – Teljes vér - Dinamikus

Viszkoelasztikus tesztek (TEG®, ROTEM®)

Bolliger D et al Transfusion Medicine Reviews 2012; 26: 1-13

Fast – Whole Blood - Dynamic



CURRENT OPINION

Hemotherapy algorithm for the management of trauma-induced coagulopathy: the German and European perspective

Curr Opin Anesthesiol 2016, 29:000–000

Marc Maegele[a,b], *Giuseppe Nardi*[c], *and Herbert Schöchl*[d]

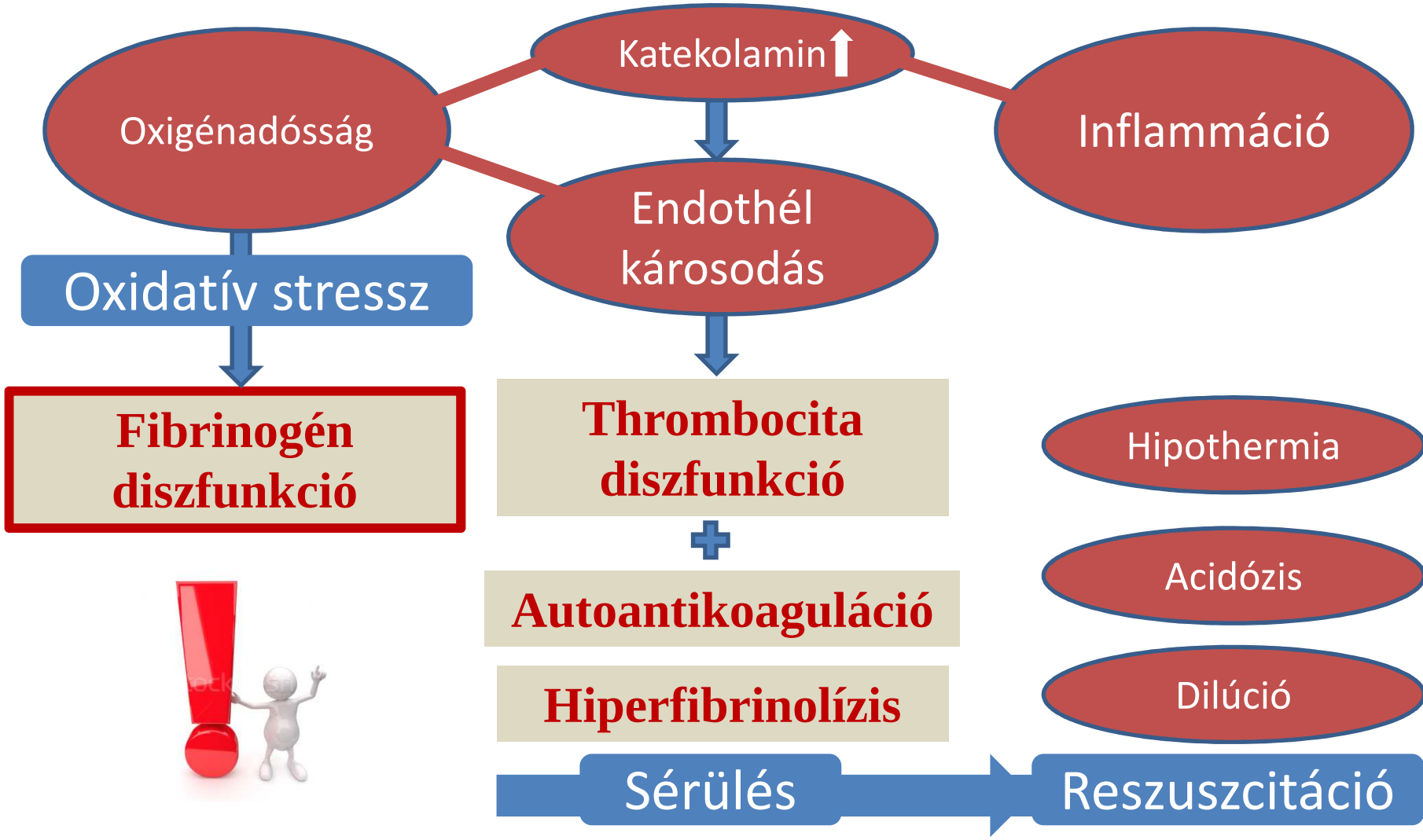
ROTEM-based algorithm for the use of hemostatic agents and blood products during early trauma care

Recommendation	Interpretation
	EXTEM  A10 < 45 mm (A5 < 35 mm) or MCF < 55 mm <u>and</u> **FIBTEM**  A10 < 10 mm (A5 < 9 mm) or MCF < 12 mm

Hemorrhagic blood failure: Oxygen debt, coagulopathy, and endothelial damage

J Trauma Acute Care Surg. 2017;82:6,S41-49

Nathan J. White, MD, MS, Kevin R. Ward, MD, Shibani Pati, MD, PhD,
Geir Strandenes, MD, *and* Andrew P. Cap, MD, PhD

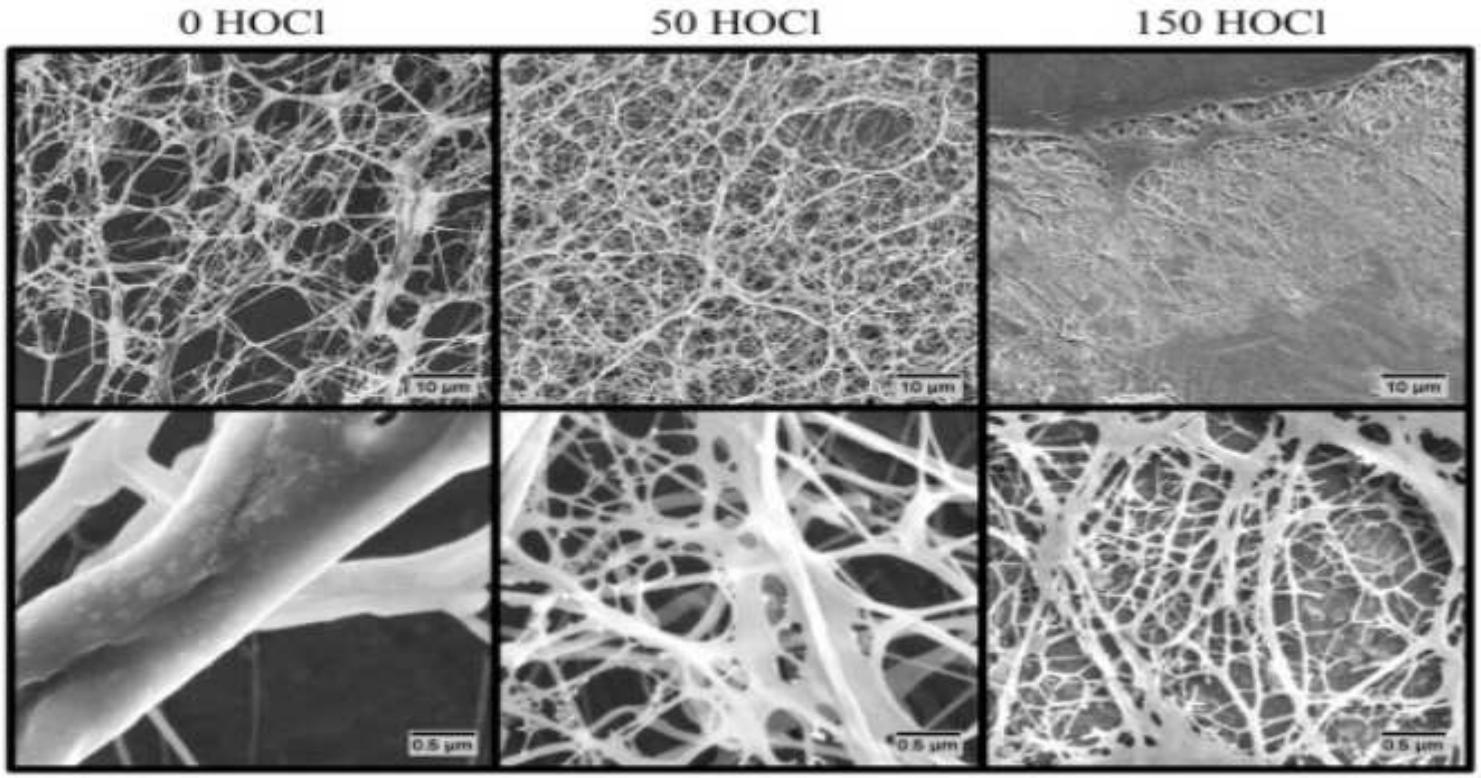


OPEN ACCESS Freely available online

January 2014 | Volume 9 | Issue 1 | PLOS ONE

Structural Effects of Methionine Oxidation on Isolated Subdomains of Human Fibrin D and αC Regions

Patrick R. Burney[1], Nathan White[2], Jim Pfaendtner[1]*

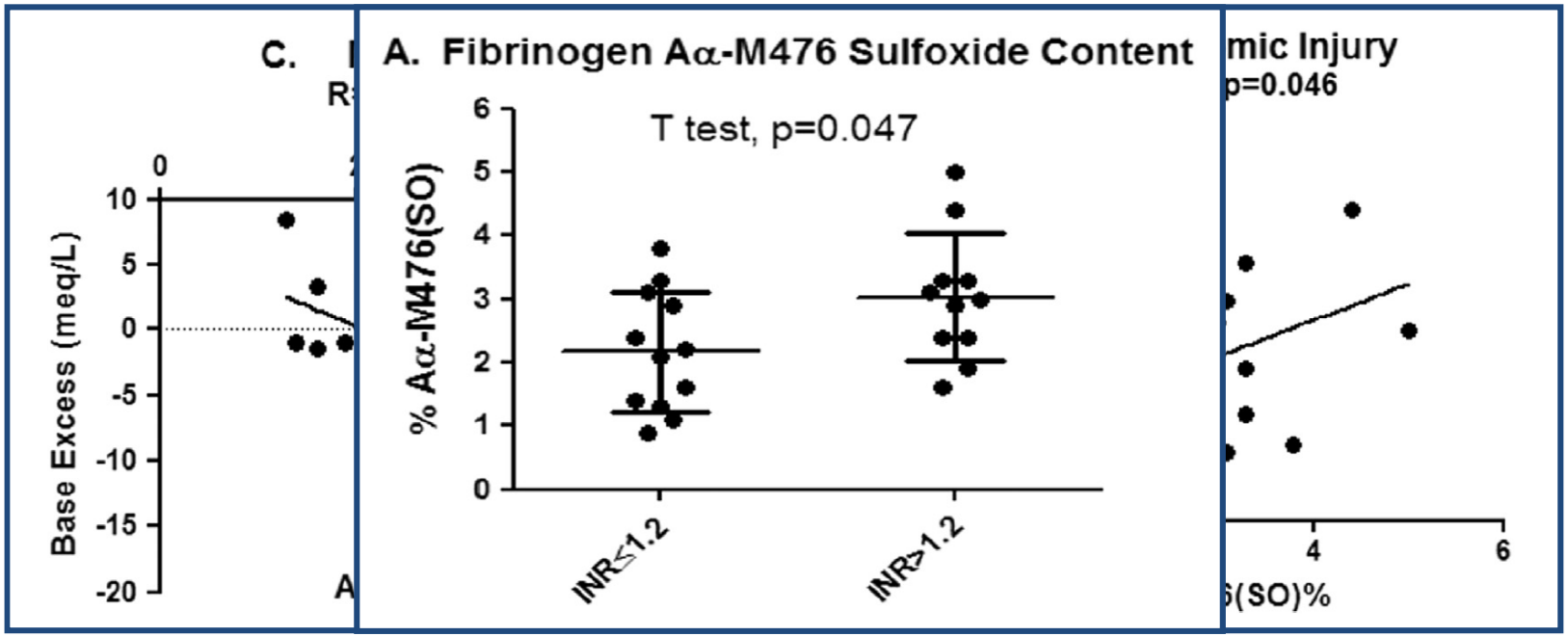


Weigandt et al. Biophysical Journal Volume (2012):2399–2407

Post-translational oxidative modification of fibrinogen is associated with coagulopathy after traumatic injury

Free Radical Biology and Medicine 96 (2016) 181–189

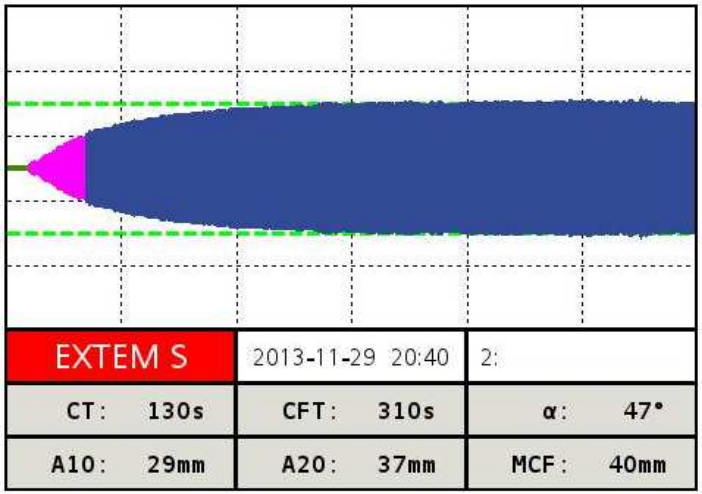
Nathan J. White [a,b,*], Yi Wang [b], Xiaoyun Fu [b], Jessica C. Cardenas [c], Erika J. Martin [d], Donald F. Brophy [d], Charles E. Wade [c], Xu Wang [a], Alexander E. St. John [a], Esther B. Lim [a], Susan A. Stern [a], Kevin R. Ward [e], José A. López [b], Dominic Chung [b]

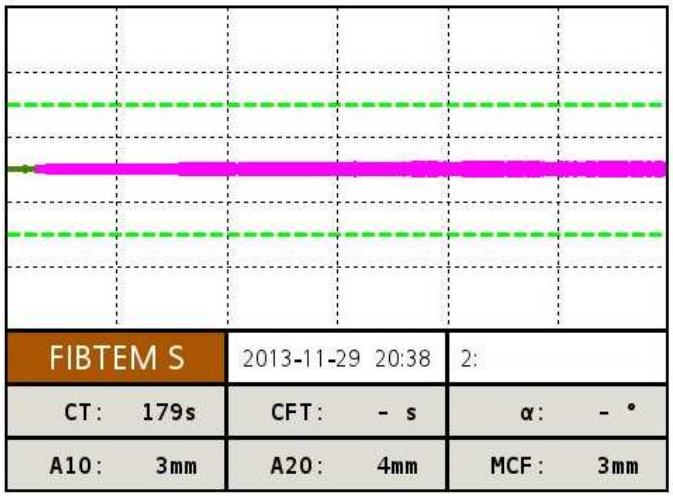


Traumás diszfibrinogenémia

Konvencionális teszt
fibrinogén szint: 2.2 g/l

Viszkoelasztikus teszt

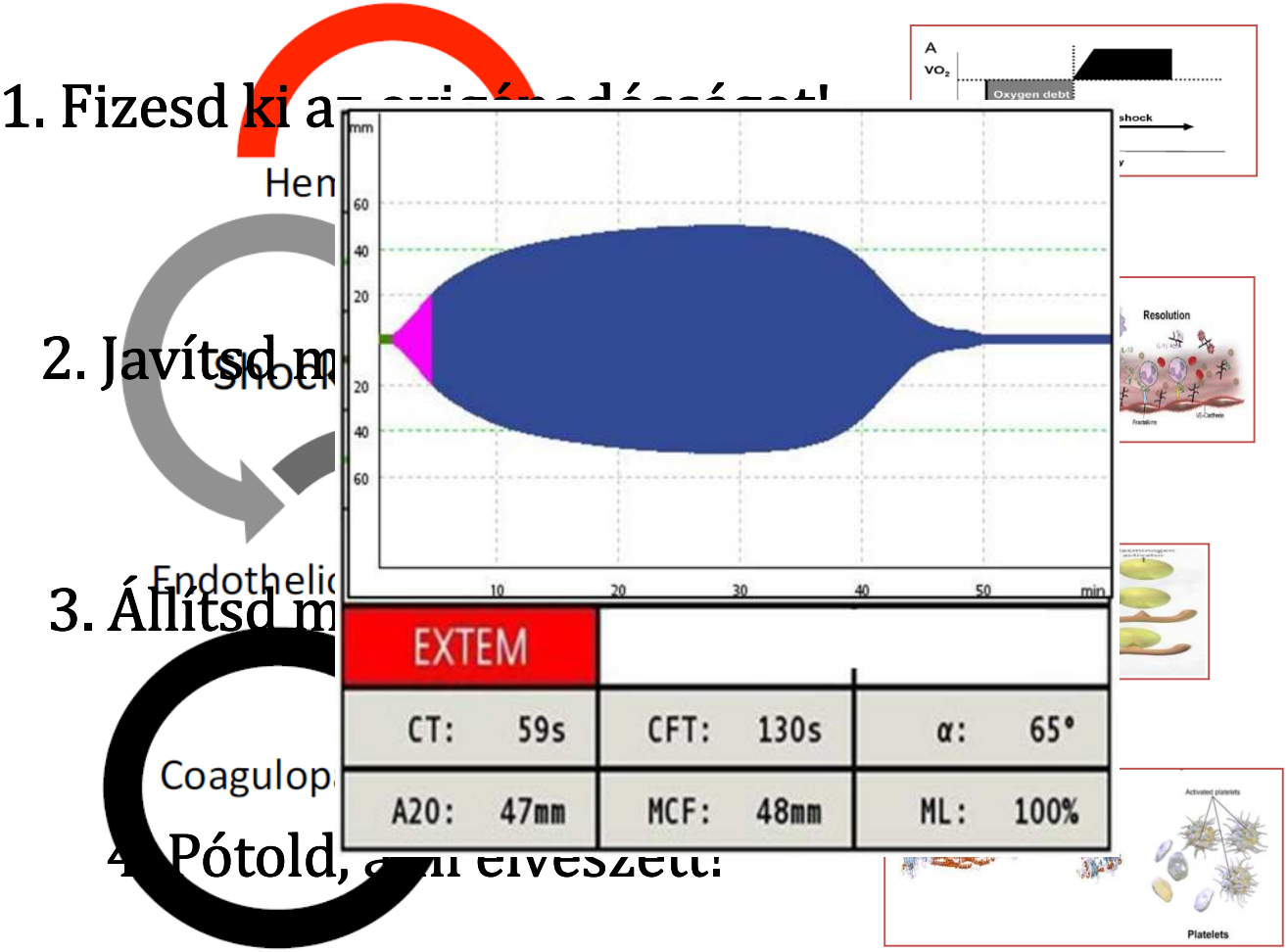




„Vérelégtelenség”

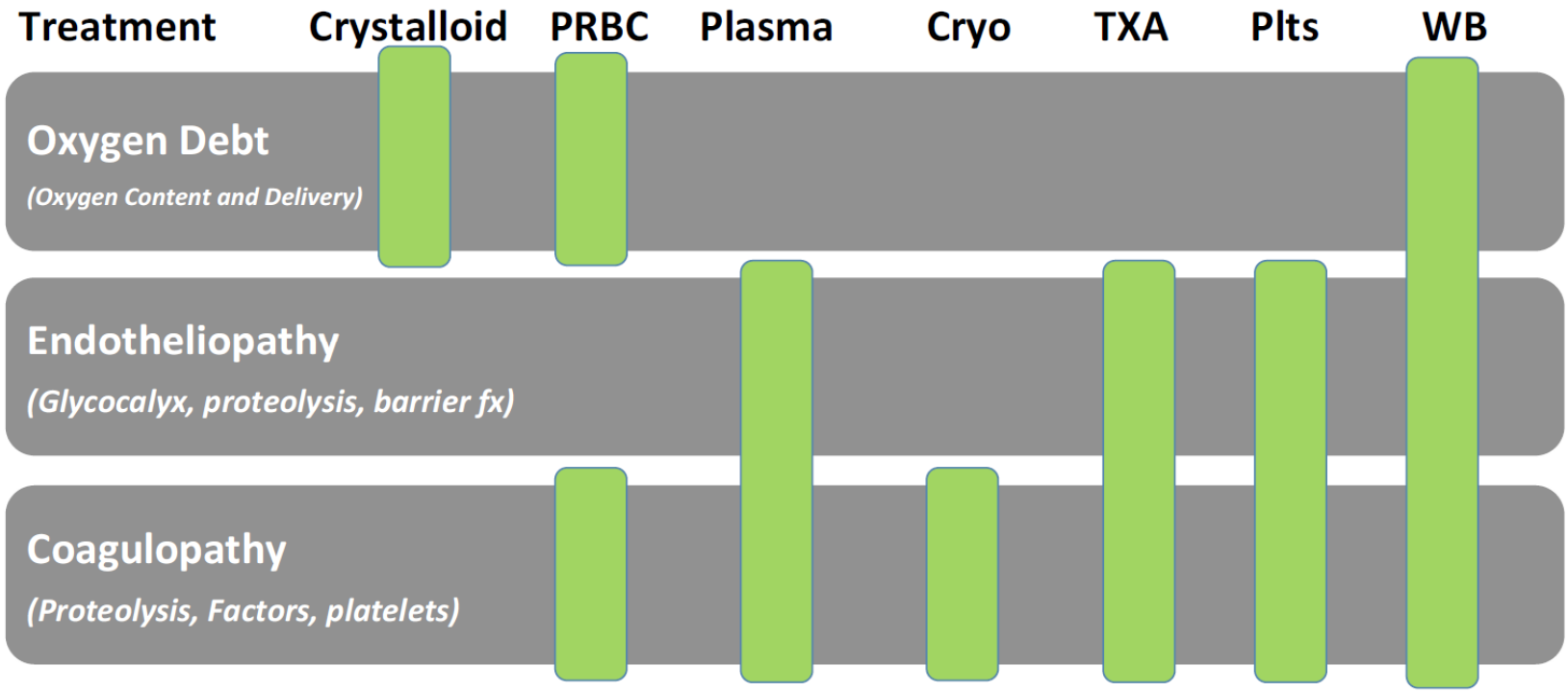
White NJ et al, J Trauma Acute Care Surg 2017;82, S41-49

1. Fizesd ki a
2. Javítsd m
3. Állítsd m
4. Pótold, a



„Vérelégtelenség”

White NJ et al, J Trauma Acute Care Surg 2017;82, S41-49



Kezelési stratégia súlyosan vérző betegeknél

Várható vvt. konc. szükséglet: > 4E/4 óra + hemodinamikai instabilitás +/- fennálló vérzés

Traumás eredet (súlyos mellkasi, hasi, medence sérülés és/vagy többszörös hosszú csöves csont törés)
Nem traumás eredet (major szülészeti/gasztrointesztinális/sebészeti vérzés)

Dinamikusan működő rendszer !

Laboratórium / Vérellátó Szolgálat

- a súlyosan vérző beteg mintái prioritást élveznek
- felkészül a nagyobb számú laborvizsgálat elvégzésére
- felkészül a várhatóan nagyobb mennyiségű vérkészítményigény kielégítésére
- a transzporthoz szükséges idő minimalizálása

Transzfúziológus / Hematológus

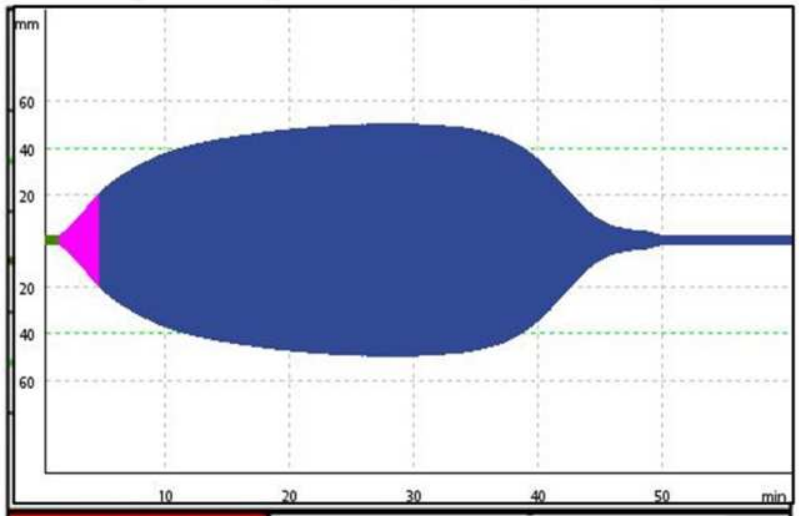
- állandó kapcsolatot tart fenn az ellátást végző klinikusokkal
- értékeli a laboratóriumi vizsgálatok eredményeit
- segít a hemosztázis rendezésében

Ellátást végző Klinikusok

- definitív vérzéscsillapítás

igen

Vérzés ?

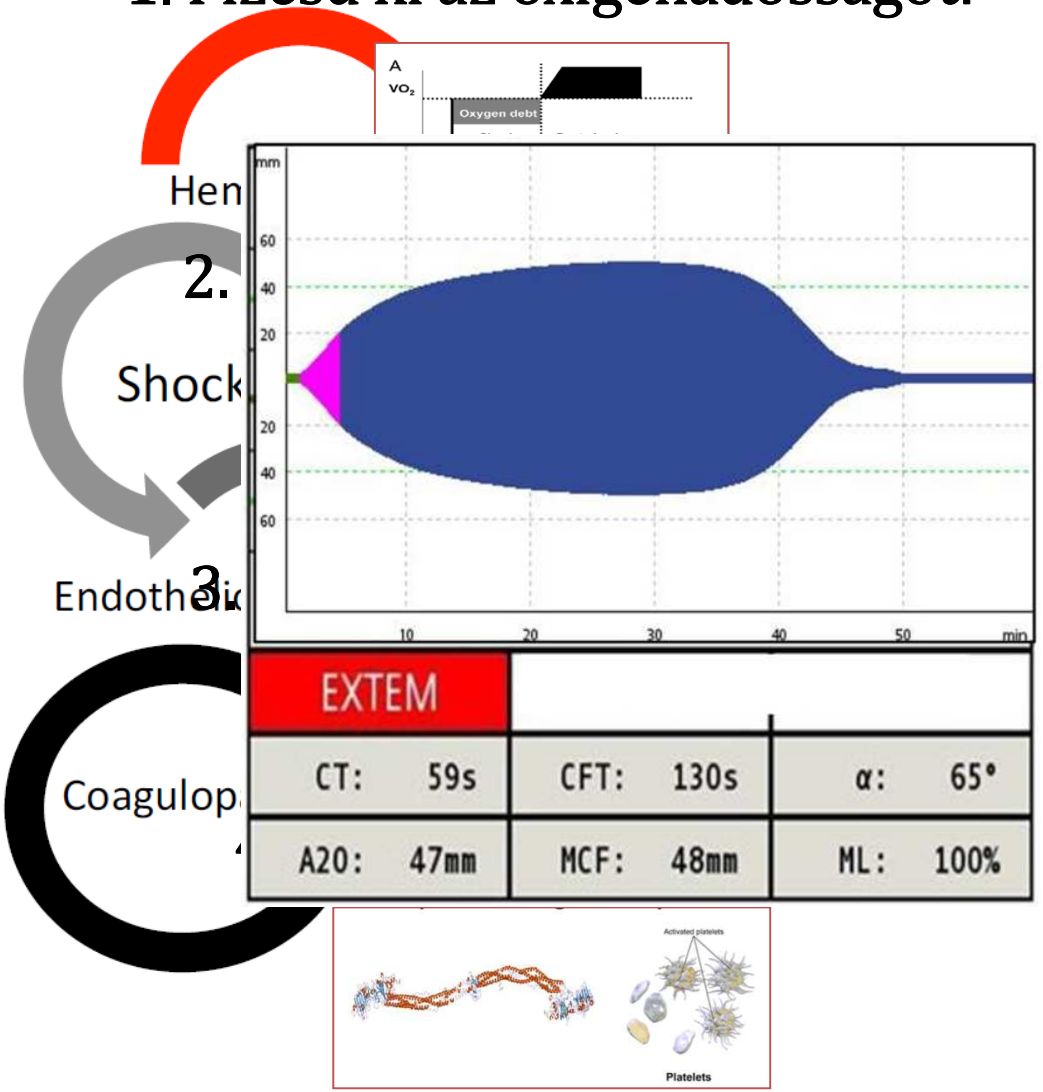


EXTEM		
CT: 59s	CFT: 130s	α: 65°
A20: 47mm	MCF: 48mm	ML: 100%

„Vérelégtelenség”

White NJ et al, J Trauma Acute Care Surg 2017;82, S41-49

1. Fizesd ki az oxigénadósságot!



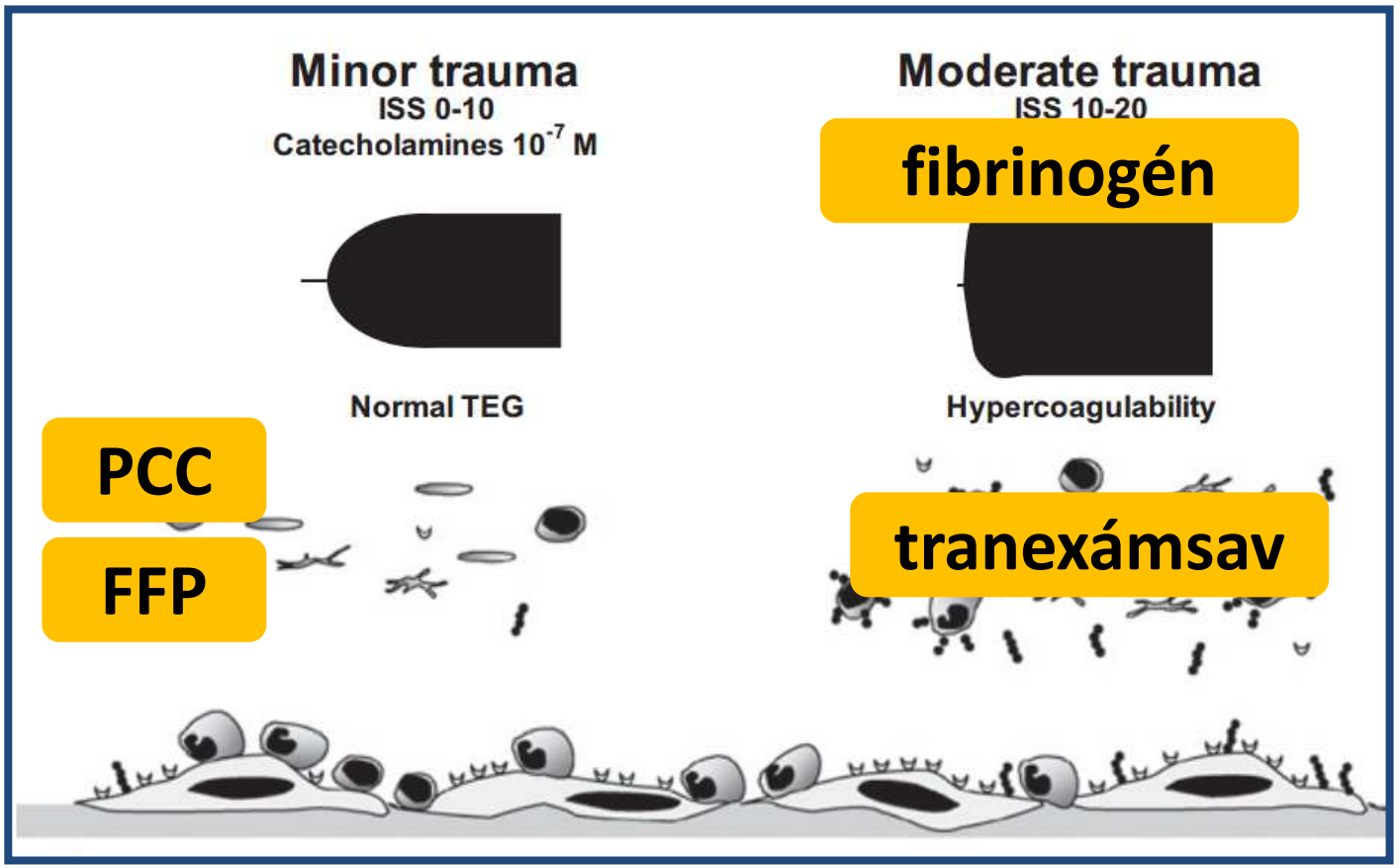
Review Article

blood

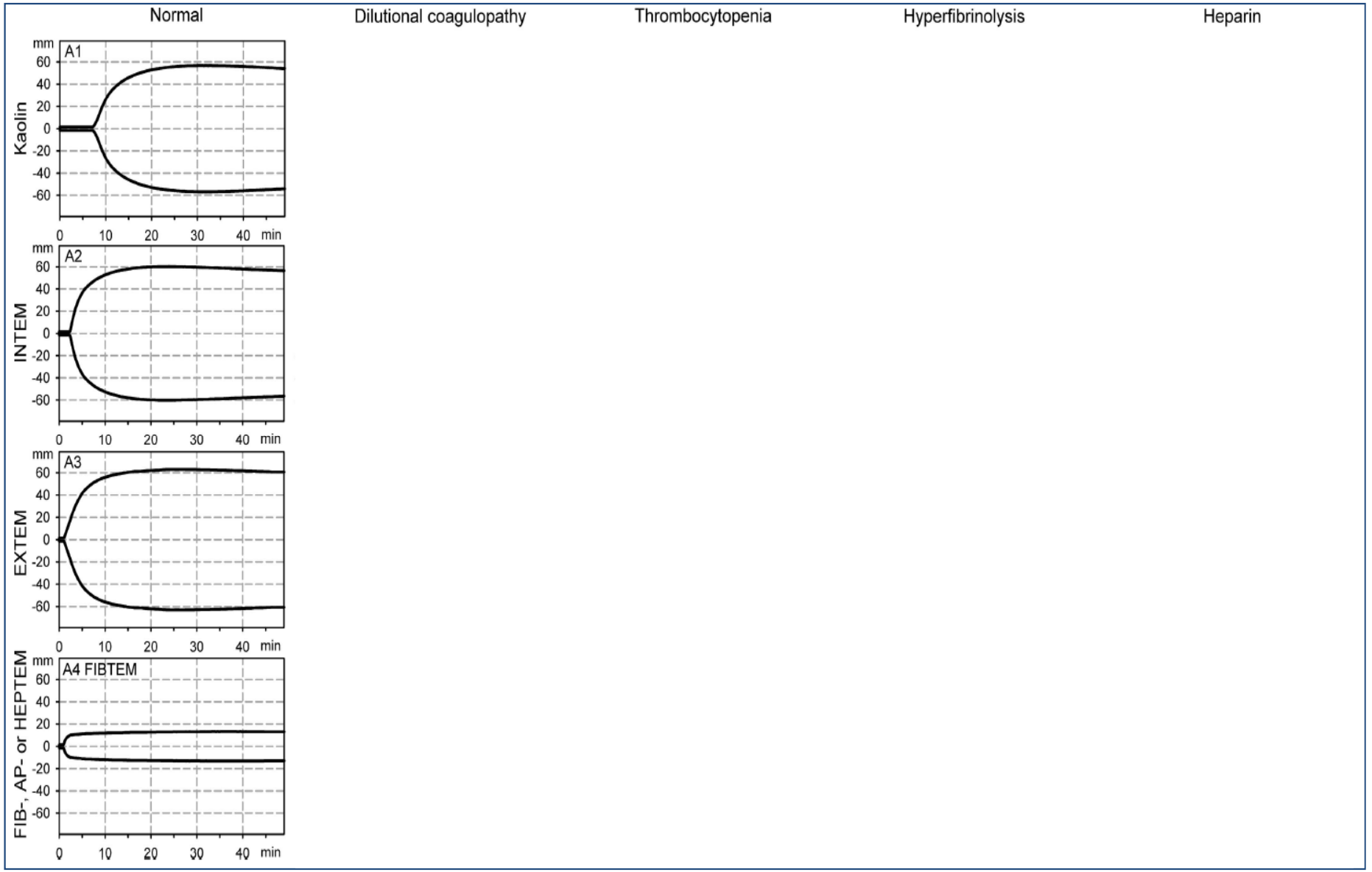
BLOOD, 25 AUGUST 2016 • VOLUME 128, NUMBER 8

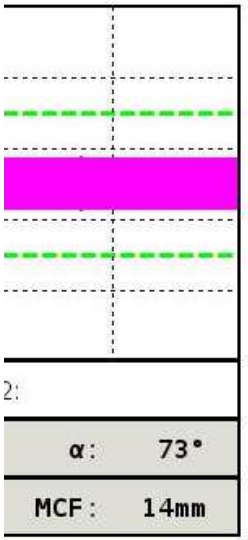
Advances in the understanding of trauma-induced coagulopathy

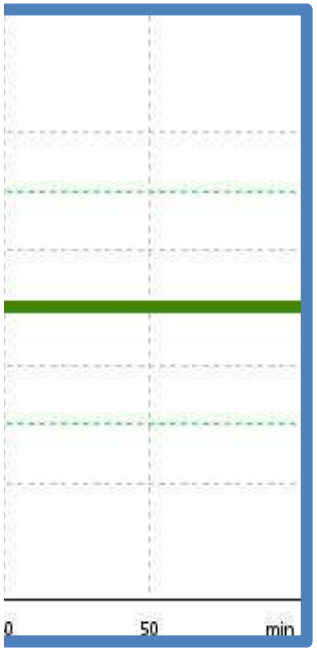
Ronald Chang,[1,2] Jessica C. Cardenas,[1,2] Charles E. Wade,[1,2] and John B. Holcomb[1,2]



TEG/ROTEM curves…




α: 73°
MCF: 14mm


0
50
min

Regular Article

BLOOD, 16 APRIL 2015 • VOLUME 125, NUMBER 16

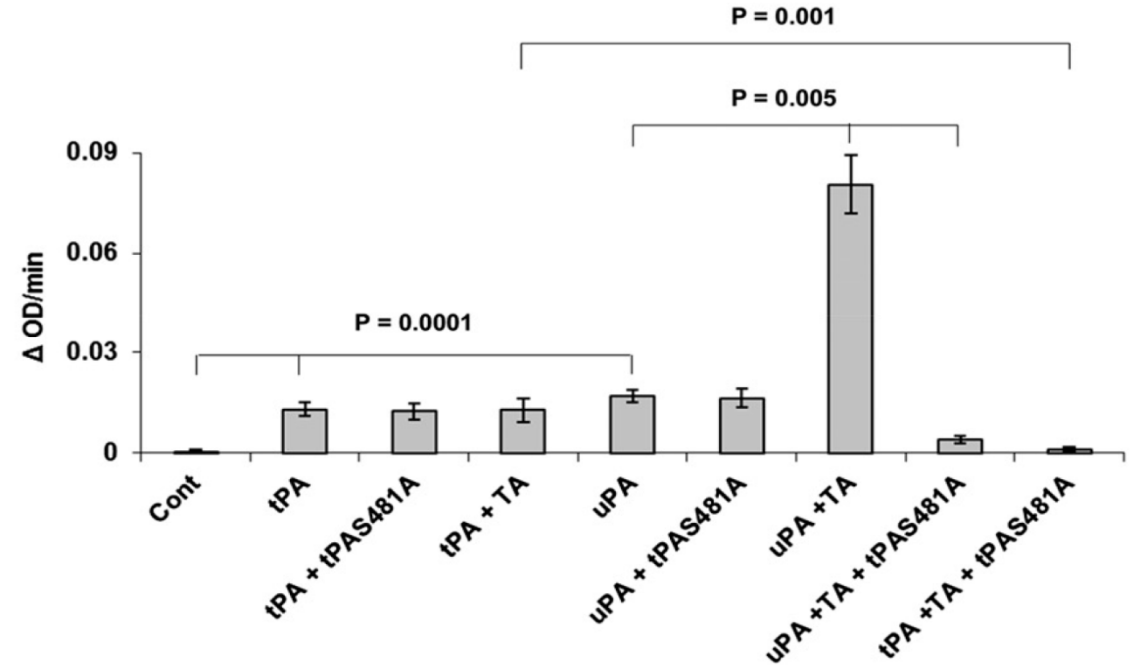
THROMBOSIS AND HEMOSTASIS

Endogenous plasminogen activators mediate progressive intracerebral hemorrhage after traumatic brain injury in mice

Nuha Hijazi,[1] Rami Abu Fanne,[1] Rinat Abramovitch,[2] Serge Yarovoi,[3] Muhamed Higazi,[1] Suhair Abdeen,[1] Maamon Basheer,[1] Emad Maraga,[1] Douglas B. Cines,[3] and Abd Al-Roof Higazi[1,3]

Key Points

- Closed head trauma sequentially releases tPA followed by uPA from injured brain.
- Increased uPA is responsible for delayed intracerebral hemorrhage, which is prevented by a tPA variant that inhibits uPA activity.



Acute Fibrinolysis Shutdown after Injury Occurs Frequently and Increases Mortality: A Multicenter Evaluation of 2,540 Severely Injured Patients

Hunter B Moore, MD, Ernest E Moore, MD, FACS, Ioannis N Liras, MD, Eduardo Gonzalez, MD, John A Harvin, FACS, MD, John B Holcomb, MD, FACS, Angela Sauaia, MD, PhD, Bryan A Cotton, MD, MPH, FACS

Vol. 222, No. 4, April 2016

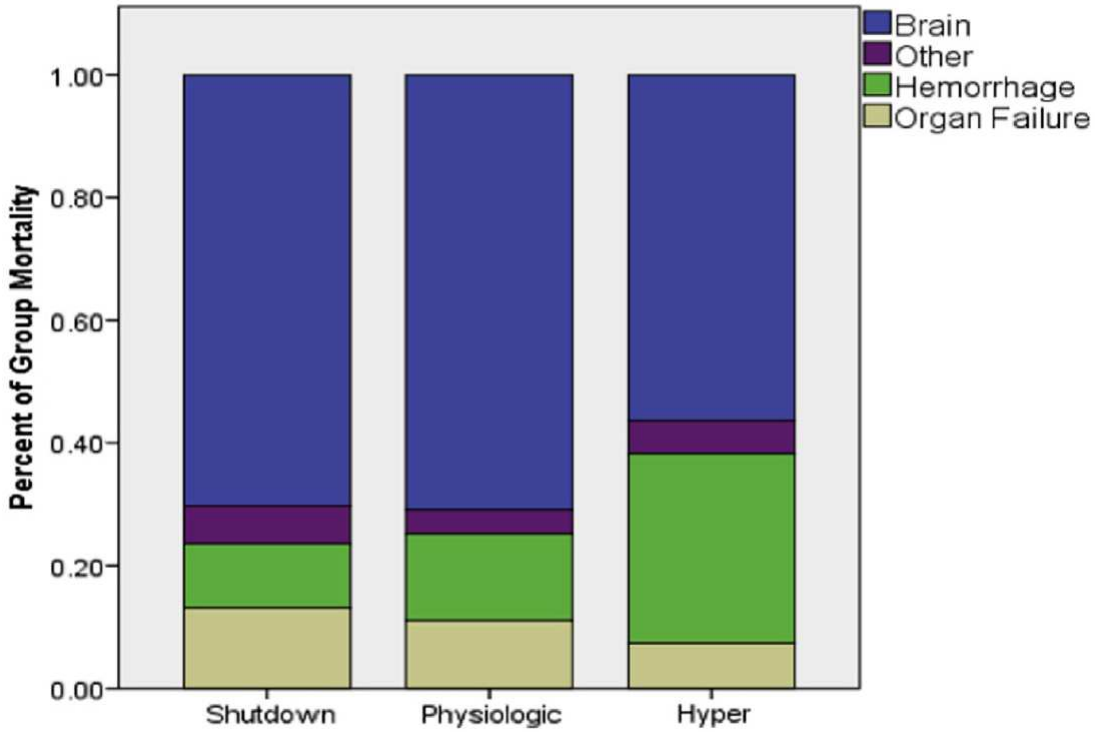
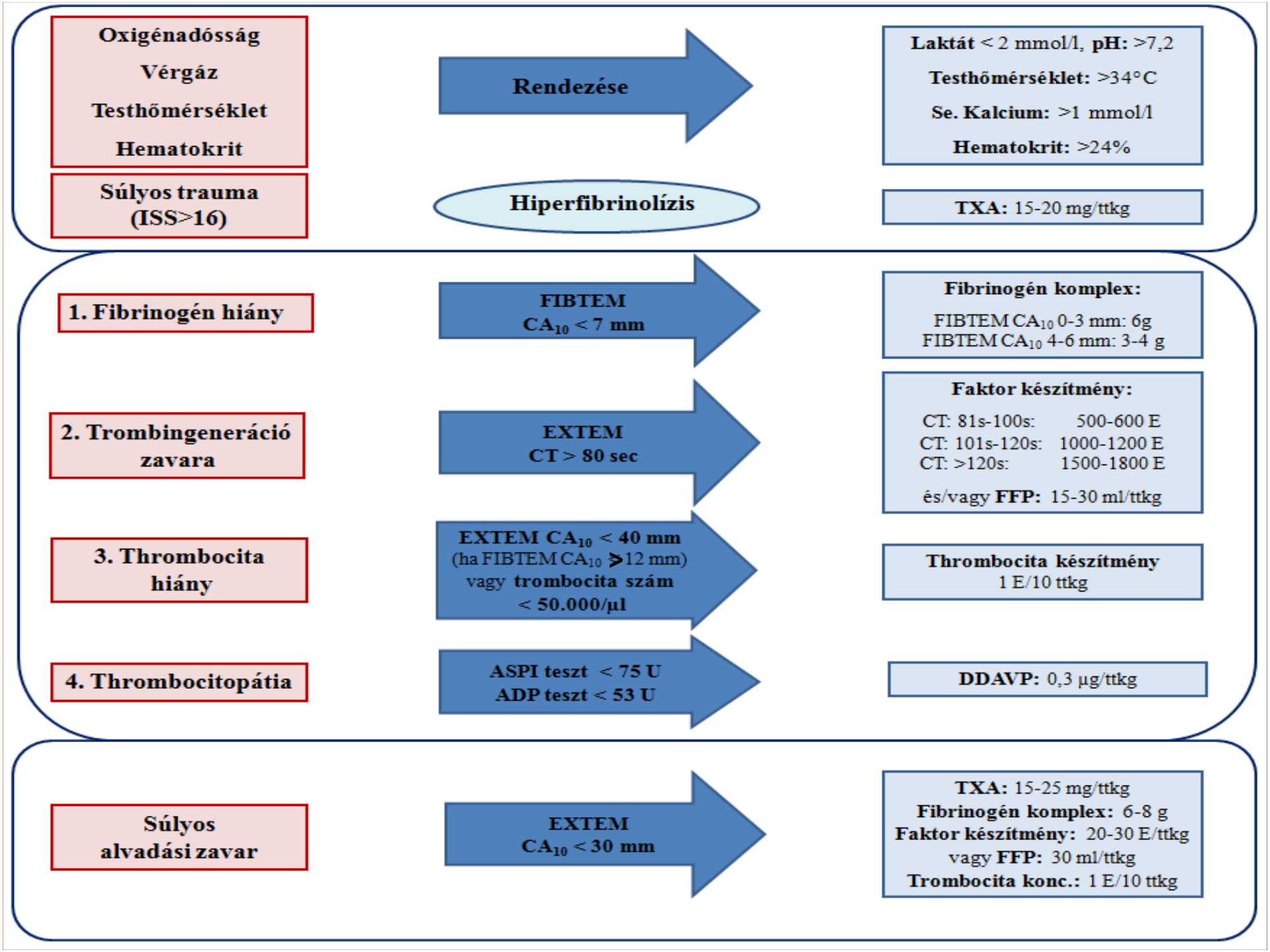


Figure 3. Differences in causes of mortality between phenotypes.


Oxigénadósság
Vérgáz
Testhőmérséklet
Hematokrit
Rendezése
Laktát < 2 mmol/l, pH: >7,2
Testhőmérséklet: >34°C
Se. Kalcium: >1 mmol/l
Hematokrit: >24%
Súlyos trauma (ISS>16)
Hiperfibrinolízis
TXA: 15-20 mg/ttkg
1. Fibrinogén hiány
FIBTEM CA10 < 7 mm
Fibrinogén komplex:
FIBTEM CA10 0-3 mm: 6g
FIBTEM CA10 4-6 mm: 3-4 g
2. Trombingeneráció zavara
EXTEM CT > 80 sec
Faktor készítmény:
CT: 81s-100s: 500-600 E
CT: 101s-120s: 1000-1200 E
CT: >120s: 1500-1800 E
és/vagy FFP: 15-30 ml/ttkg
3. Thrombocita hiány
EXTEM CA10 < 40 mm
(ha FIBTEM CA10 ≥12 mm)
vagy trombocita szám
< 50.000/µl
Thrombocita készítmény
1 E/10 ttkg
4. Thrombocitopátia
ASPI teszt < 75 U
ADP teszt < 53 U
DDAVP: 0,3 µg/ttkg
Súlyos alvadási zavar
EXTEM CA10 < 30 mm
TXA: 15-25 mg/ttkg
Fibrinogén komplex: 6-8 g
Faktor készítmény: 20-30 E/ttkg
vagy FFP: 30 ml/ttkg
Trombocita konc.: 1 E/10 ttkg

POCT diagnosztika (ACT, INR)

Aktivált alvadási idő (ACT)

(Hattersley, 1966)

- az aPTT analógja

  aPTT: citrátos (heparinos) vér + Ca + **kaolin**

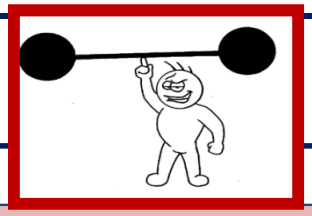
  ACT: natív (heparinos) vér + **kaolin** (+ testhő)

- Normál értéke 90-120 s (CPB:400-480 s)

Prothrombin idő (INR)

Teszt	Normál érték	Pozitív próba oka lehet pl.
Prothrombin idő (PT, Quick v. recalcificatiós idő) Mivel a vizsgálandó plasma thromboplastinnal és phospholipiddel inkubált, Ca++ adása után az alvadási folyamat „végigszalad” az extrinsic és a közös úton.	· 12 s · 100 % (prothrombin százalékos aktivitás) · 1 (INR=beteg PT / ref.PT)	Exstrinsic alvadási trigger rendszer zavara, kumarin terápia

TEG® vs. ROTEM®

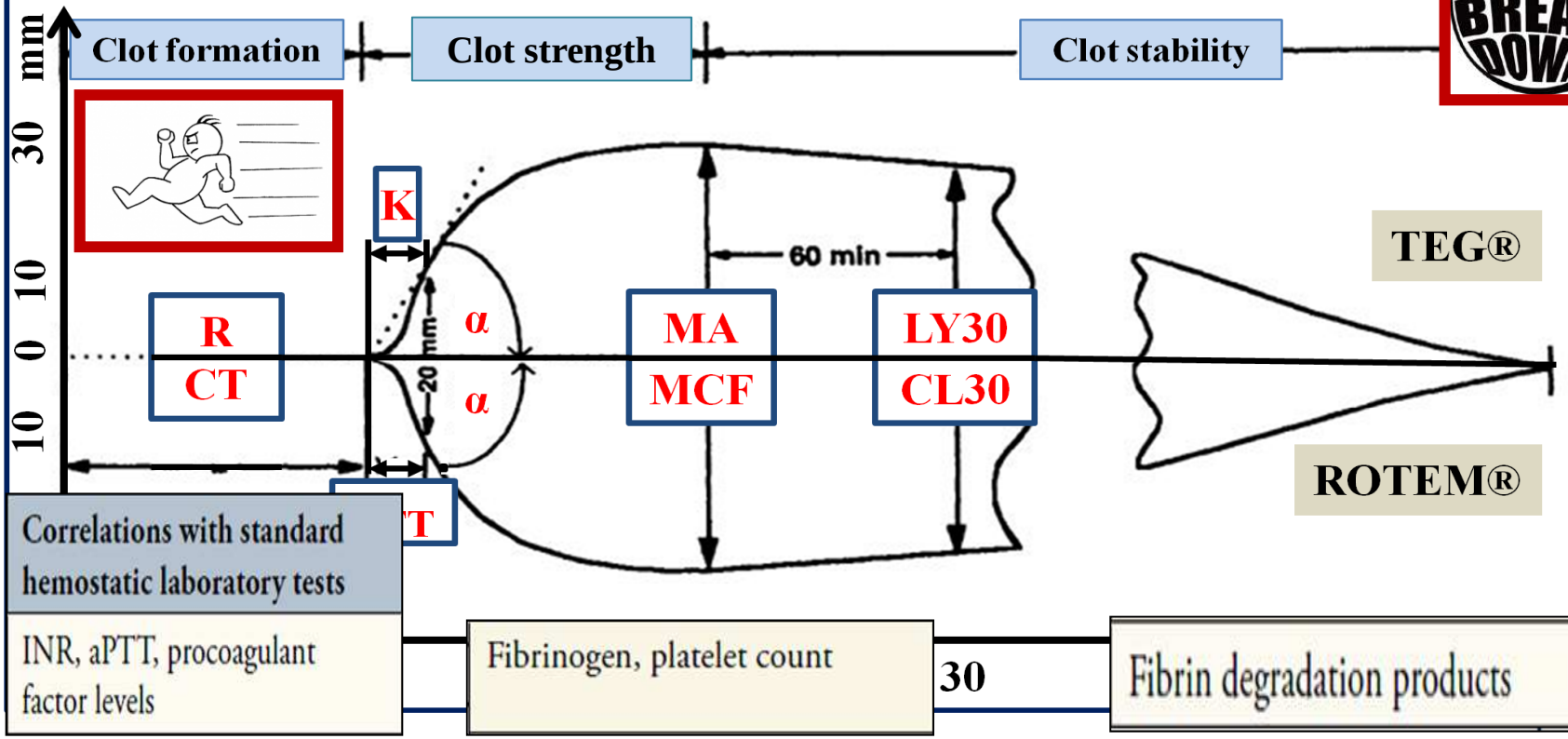


Balance in Pro- and Anticoagulation Factors

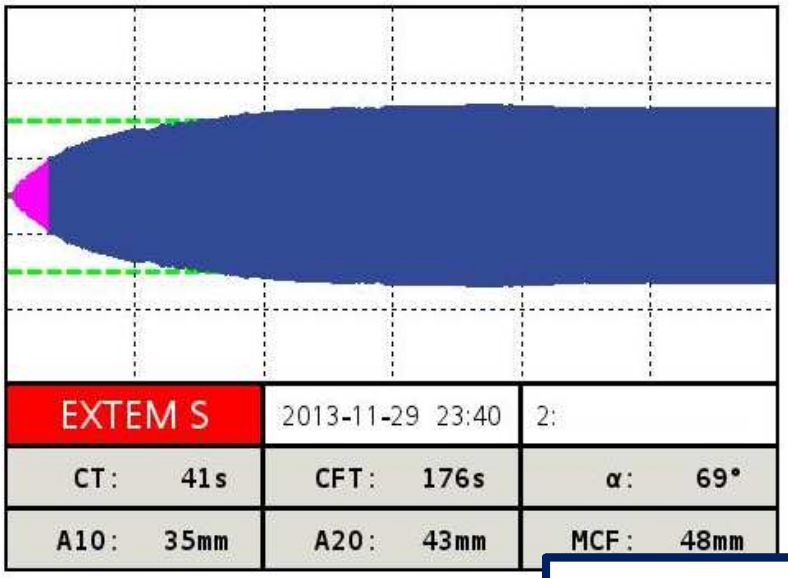
Platelets and Fibrin mesh

Balance in Pro- and Antifibrinolysis

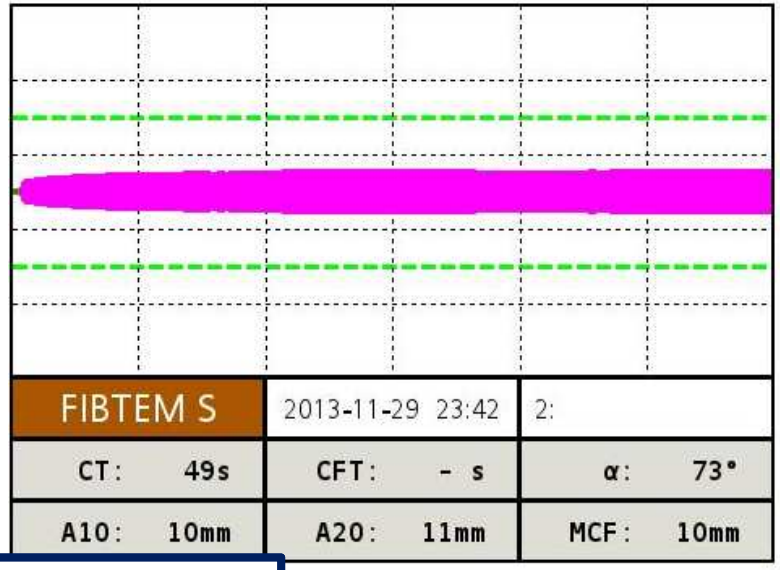
Clot formation | **Clot strength** | **Clot stability**



Stravicz RT et al Gastroent & Hepat 2012; 8: 513-520

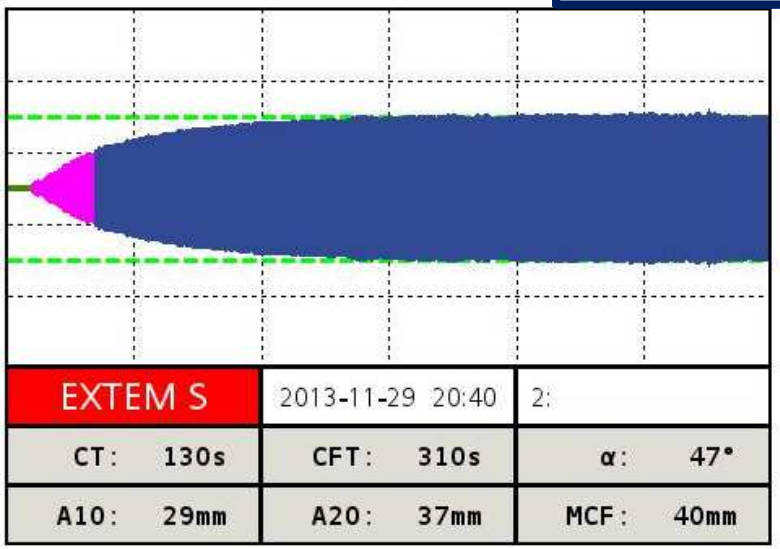


EXTEM S	2013-11-29 23:40	2:
CT: 41s	CFT: 176s	α: 69°
A10: 35mm	A20: 43mm	MCF: 48mm

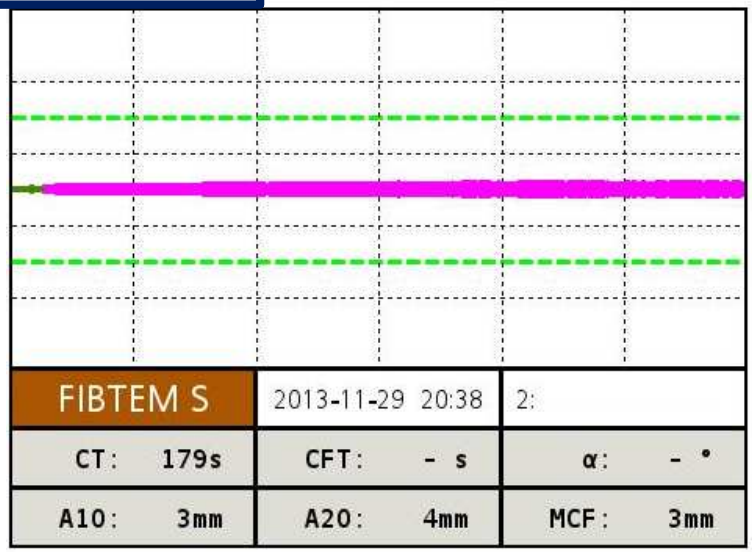


FIBTEM S	2013-11-29 23:42	2:
CT: 49s	CFT: - s	α: 73°
A10: 10mm	A20: 11mm	MCF: 10mm

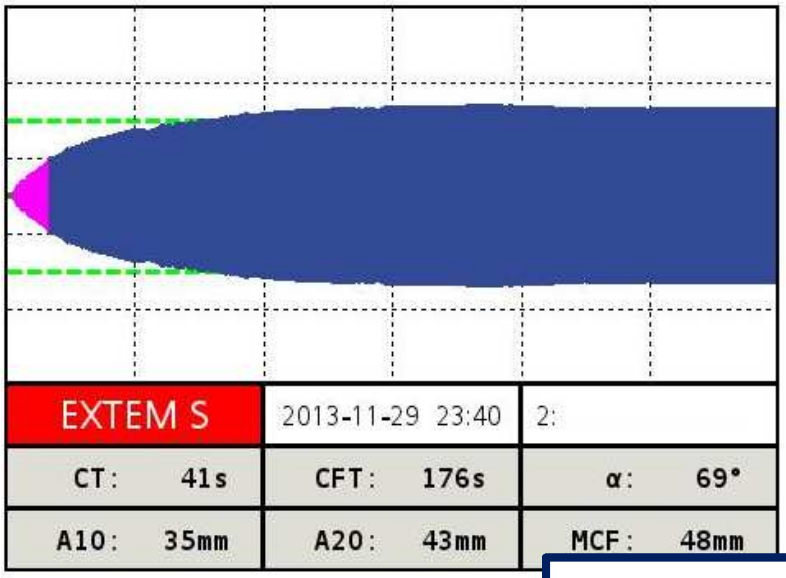
2 E vvt



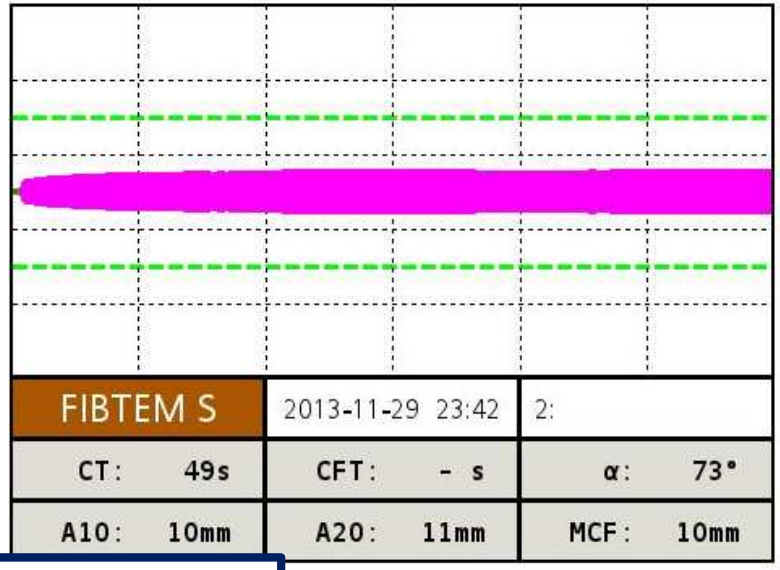
EXTEM S	2013-11-29 20:40	2:
CT: 130s	CFT: 310s	α: 47°
A10: 29mm	A20: 37mm	MCF: 40mm



FIBTEM S	2013-11-29 20:38	2:
CT: 179s	CFT: - s	α: - °
A10: 3mm	A20: 4mm	MCF: 3mm

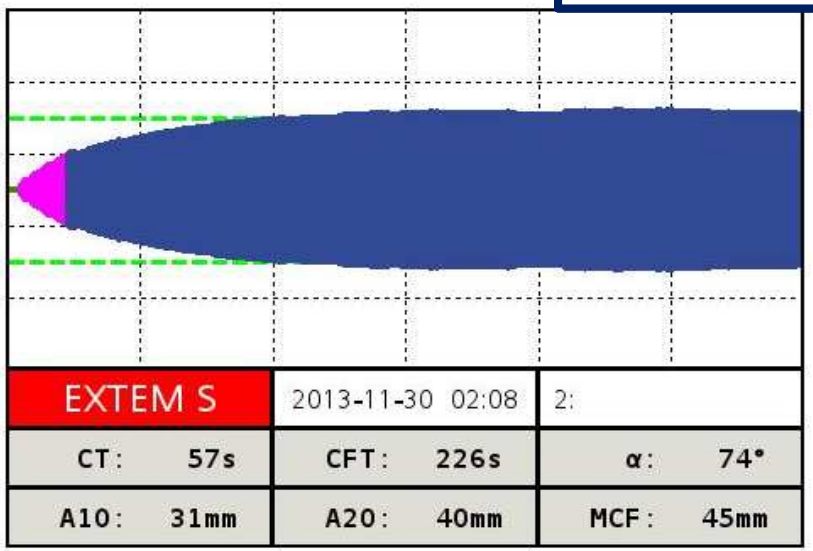


EXTEM S	2013-11-29 23:40	2:
CT: 41s	CFT: 176s	α: 69°
A10: 35mm	A20: 43mm	MCF: 48mm

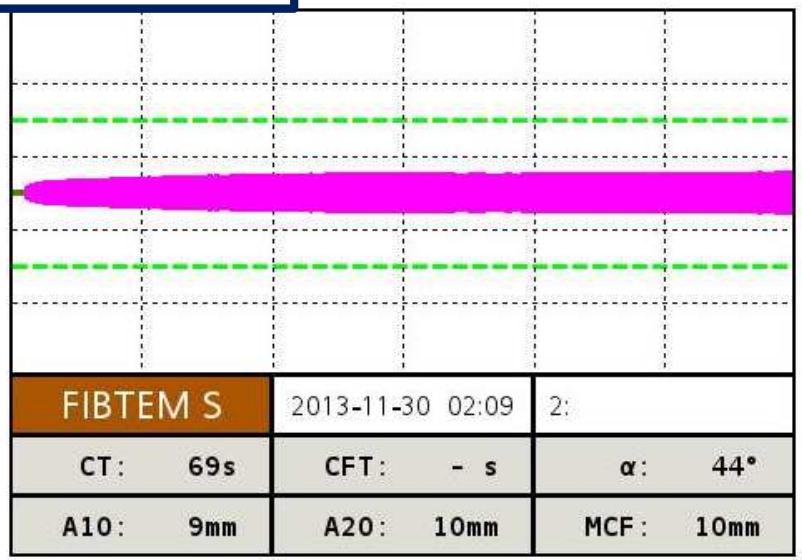


FIBTEM S	2013-11-29 23:42	2:
CT: 49s	CFT: - s	α: 73°
A10: 10mm	A20: 11mm	MCF: 10mm

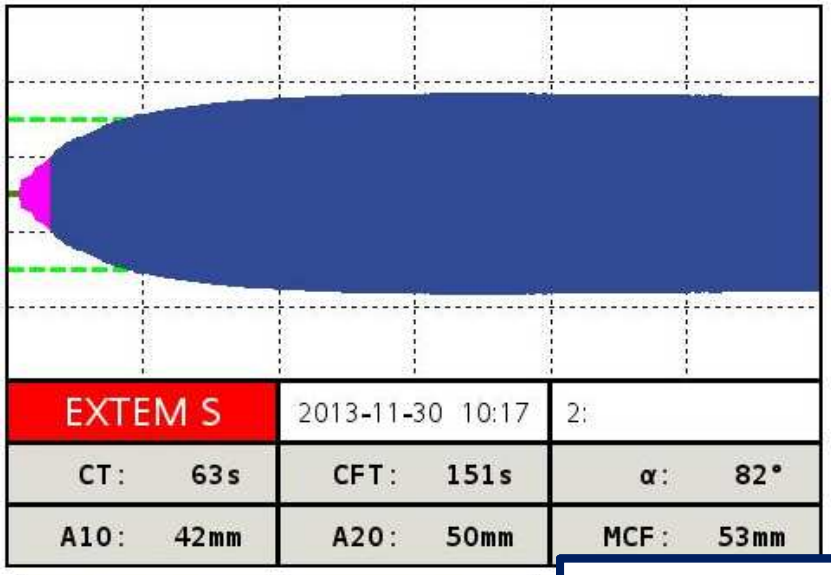
2 E vvt

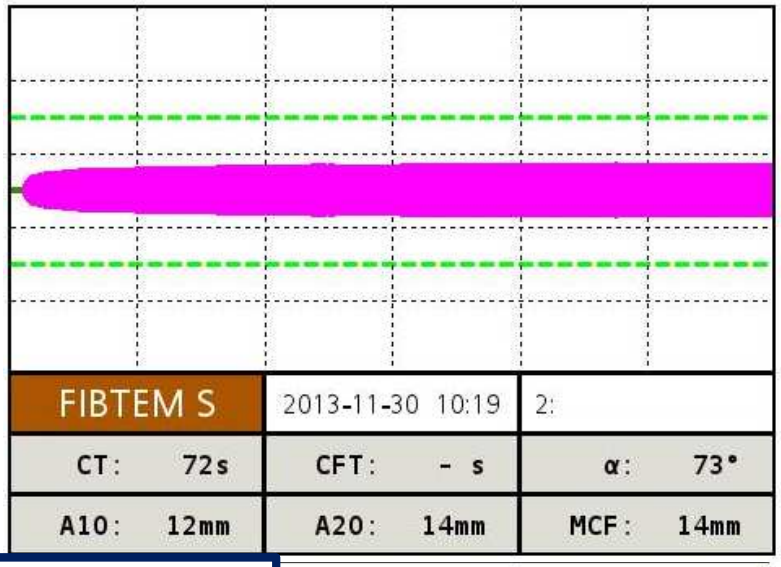


EXTEM S	2013-11-30 02:08	2:
CT: 57s	CFT: 226s	α: 74°
A10: 31mm	A20: 40mm	MCF: 45mm

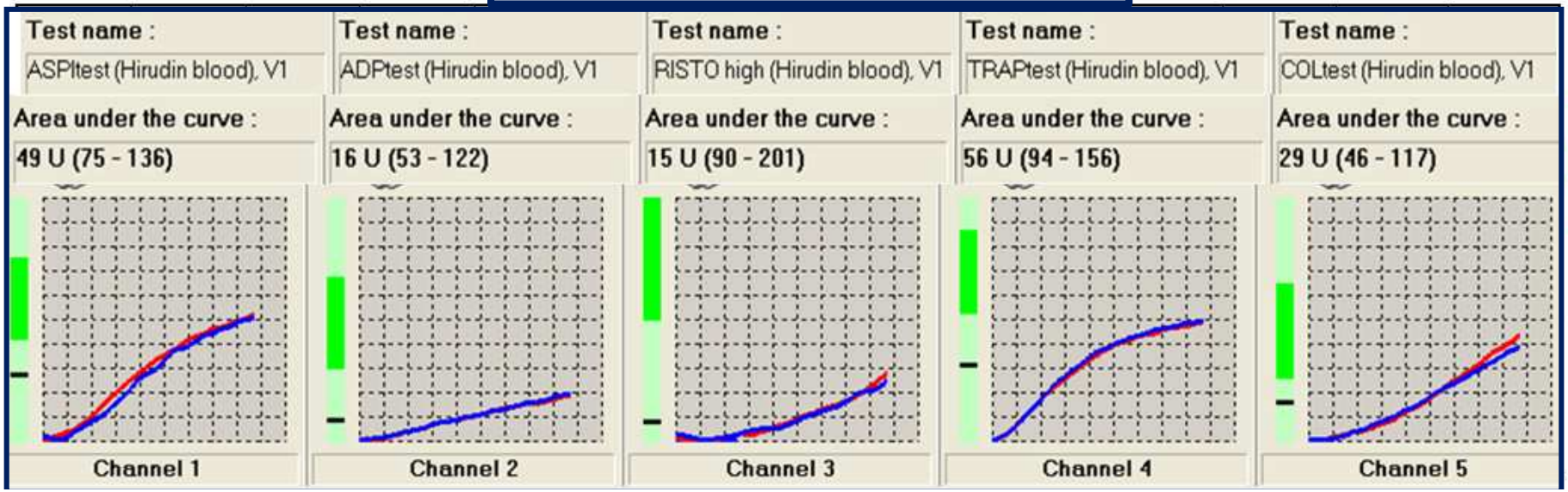


FIBTEM S	2013-11-30 02:09	2:
CT: 69s	CFT: - s	α: 44°
A10: 9mm	A20: 10mm	MCF: 10mm





2 E vvt
1 gr fibrinogén
8 E Tct
24 mikrogr DDAVP



Esetismertetés

- 46 éves ffi, autóbaleset, stabil vitális paraméterek, GCS 15

- Diagnosztika: Hasi UH (3-4 cm szabad hasi folyadék)

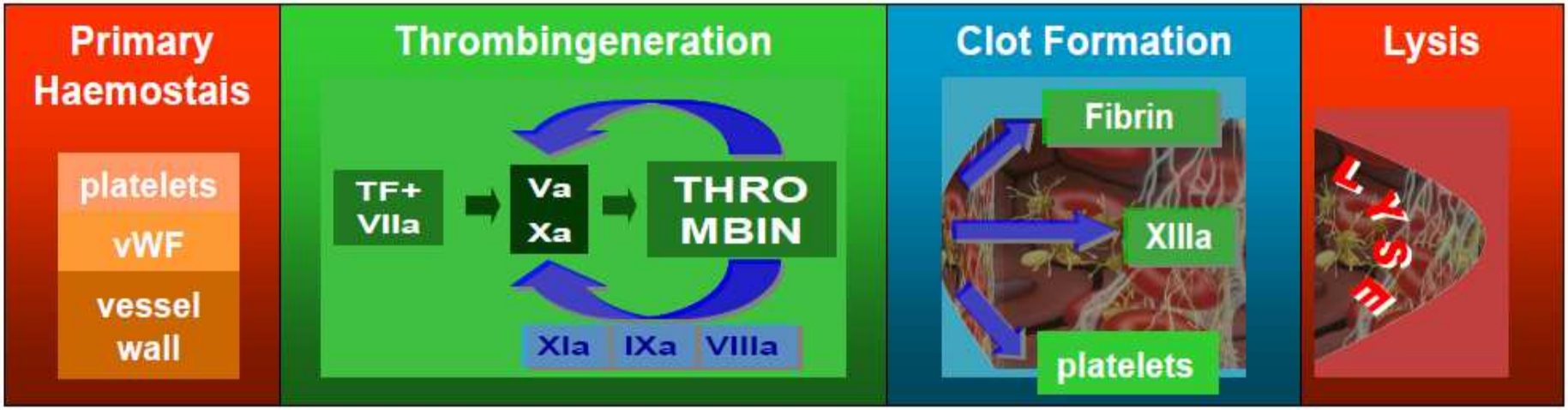
- Anamnézis: vaszkulárisan dekompenzált májcirrózis

(korábban: thr: 40-60G/L, INR: 1.51)

Esetismertetés

- Obszerváció Traumatológiai osztályon (23:00)
- 05:54 - Periarrest állapot-reanimáció (6 perc)
- ROSC és felvétel az ITO-ra
- Elsődleges ellátás

A mindennapokban…

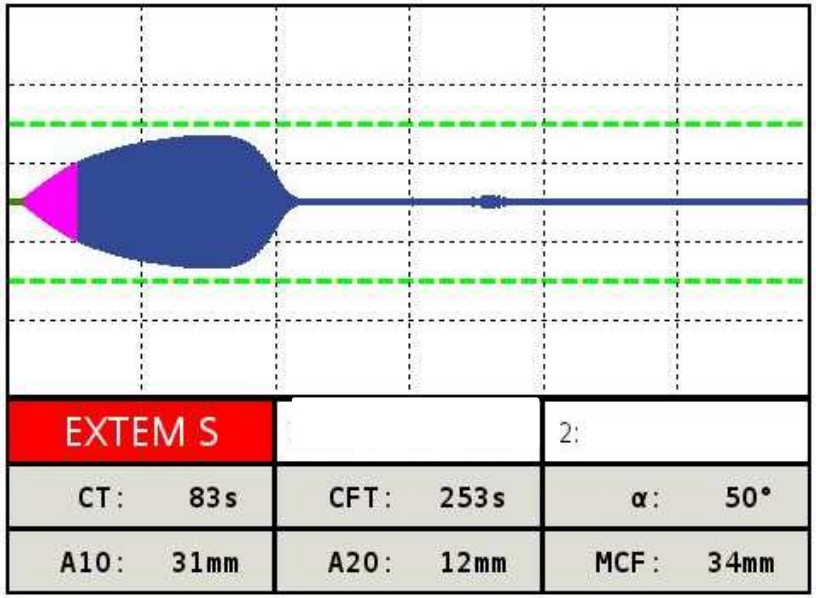


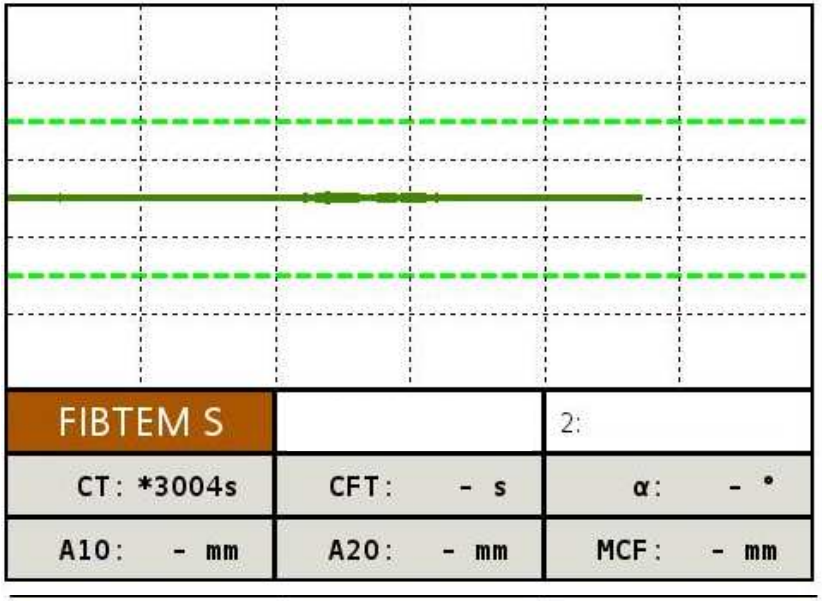
Dr. Dietmar Fries: Basics of coagulation 2013; CSL Behring Symposium

Noradrenalin: 30 mcg/min
P: 115/min
RR: 106/32 Hgmm
Hőm. 33.1 C

	06:32 v	06:35 a
Hg (g/dL)	4.5	4.5
Hct (%)	15	15
pH	6.7	6.8
PCO2 (Hgmm)	71.4	67.7
PO2 (Hgmm)	25	151
BE (mmol/L)	-23.2	-22.8
cHCO3 (mmol/L)	9.4	8.9
SO2 (%)	19	90
Glu (mmol/L)	1.9	1.9
Na (mmol/L)	148	149
K (mmol/L)	5.4	5.3
Ca (mmol/L)	0.75	0.74
Lac (mmol/L)	+++	+++

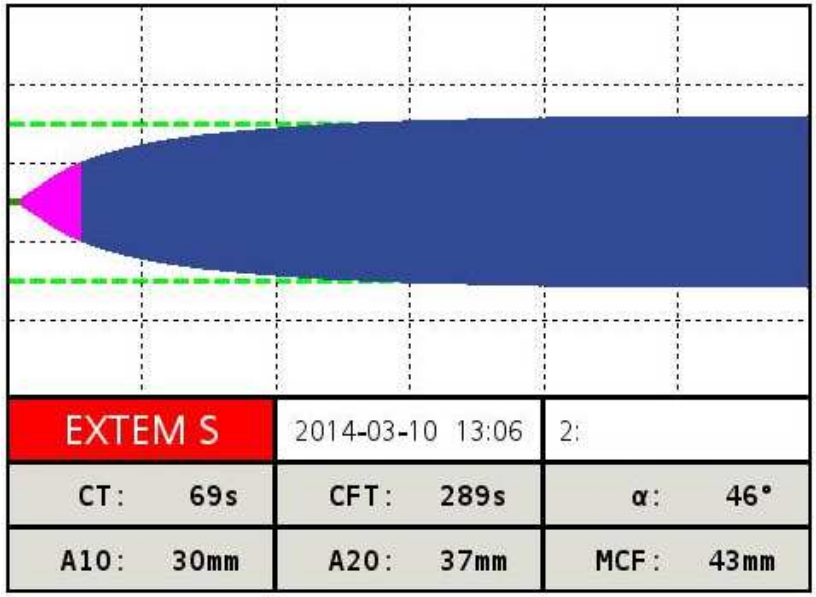
Véralvadás szempontjából probléma:

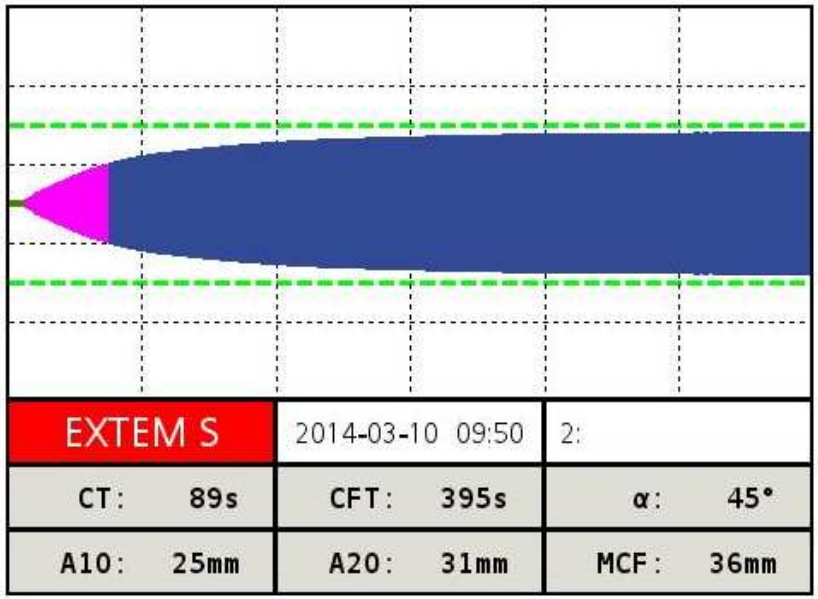




CT: máj körüli vérzés → **Műtét!** - De mikor?

3E vvt, NA, 60 mmol HCO_3, 3g Ca, melegítés, 40g Glu,
2g Exacyl, 3g Fibrinogén,
4E FFP, 1000 E PPC,
24 mcg DDAVP

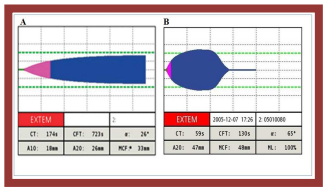


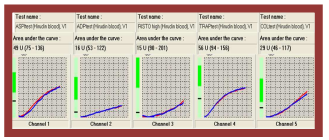


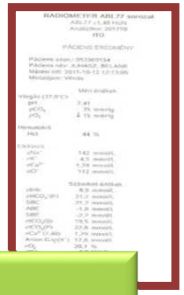
8E vvt, NA, 100 mmol HCO_3, melegítés,
8E FFP, 500 PCC,
24E Tct,

Dinamikus koagulációs menedzsment

Mérés



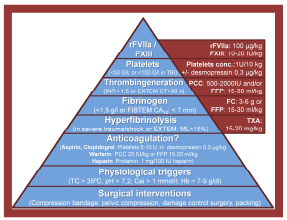




Értékelés

Dinamikus, individualizált kezelés !

Kezelés



Take Home Message

Complex coagulopathy and Time

<u>Viscoelastic test</u>
(CT, CFT, α, MCF, ML)
+
<u>Platelet aggregometry</u>

⟷

<u>Conventional test</u>
(I., II., III.,…XI., XII.,XIII,
PT, INR, aPTT, AT III,
fibrinogen, platelets count
FDP, D-dimer…)

Meybohm *et al. Critical Care* 2013, **17**:218
http://ccforum.com/content/17/2/218

CRITICAL CARE

REVIEW

Point-of-care coagulation management in intensive care medicine

Patrick Meybohm, Kai Zacharowski*, Christian F Weber

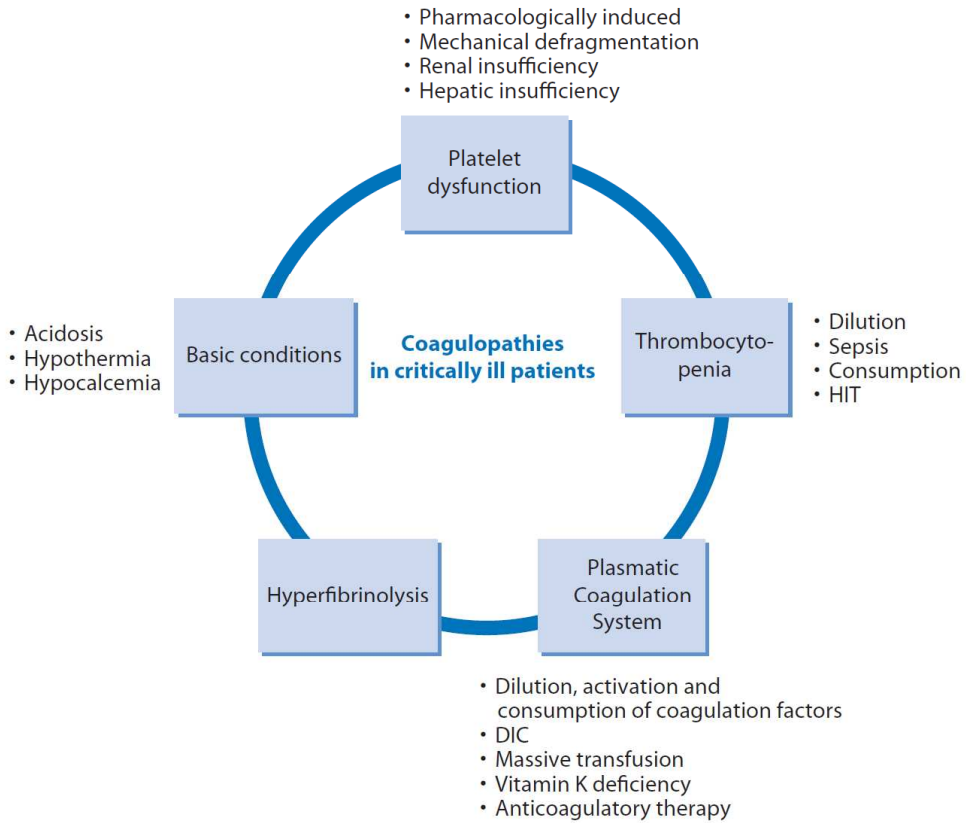
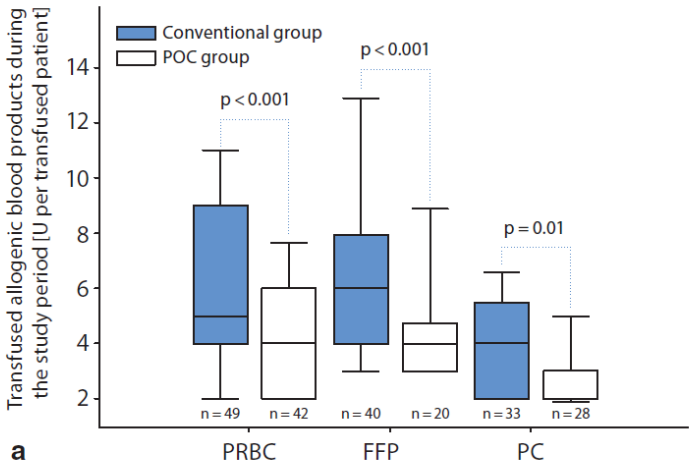
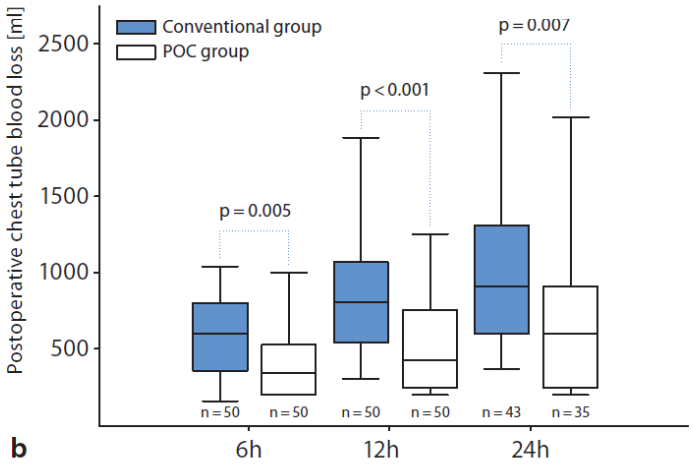
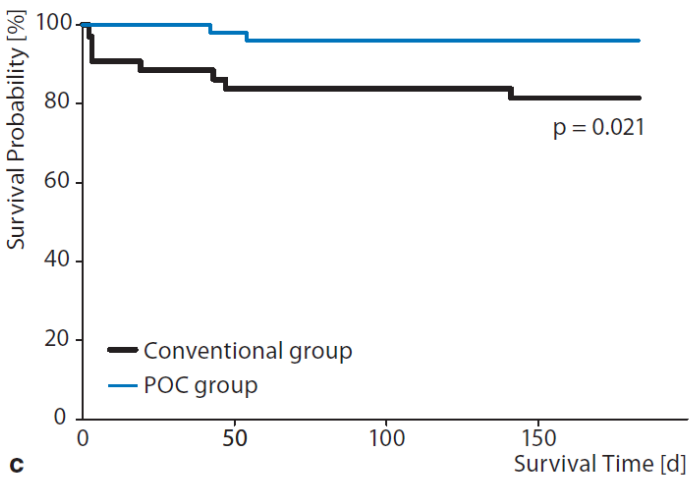


Figure 1. Overview of coagulopathies typically present in critically ill patients. DIC: disseminated intravascular coagulopathy; HIT: heparin-induced thrombocytopenia.





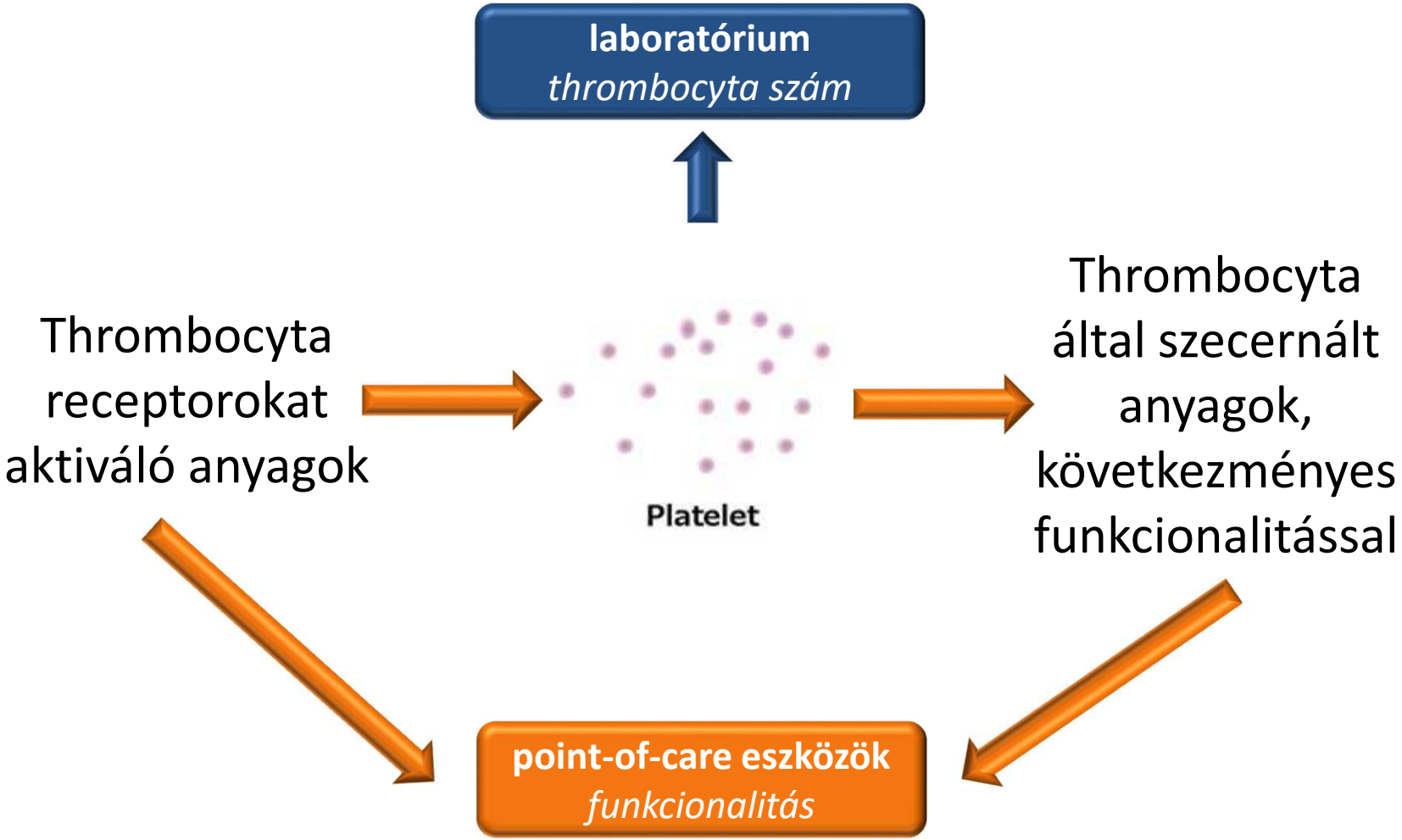


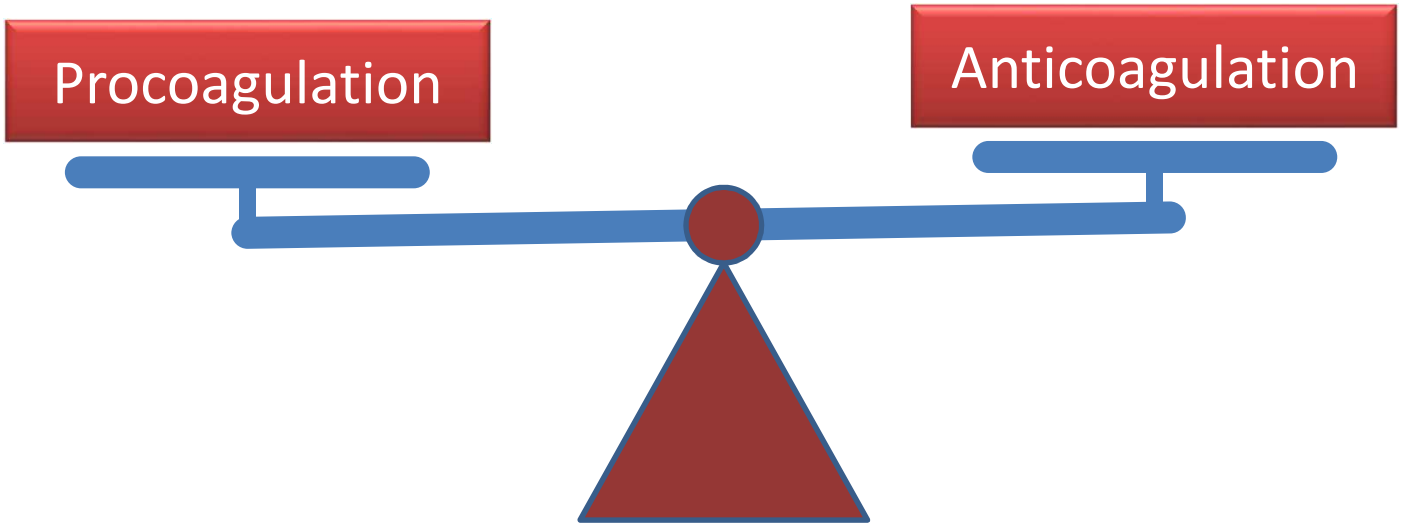
	Conventional group	POC group	p-value
Postoperative PaO_2/FiO_2–indices			
At ICU admission	323 (201/402) (n–50)	355 (282/460) (n–50)	0.069
2 h after ICU admission	326 (219/393) (n=50)	338 (277/423) (n=50)	0.087
4 h after ICU admission	309 (256/373) (n=50)	358 (300/407) (n=50)	0.015
12 h after ICU admission	299 (222/371) (n=48)	398 (328/467) (n=45)	< 0.001
24 h after ICU admission	228 (137/312) (n=23)	327 (259/468) (n=17)	0.009
Time of mechanical ventilation [min]	827 (440/2835) (n=50)	316 (230/513) (n=50)	< 0.001
ICU period [h]	24 (20/87) (n=50)	21 (18/31) (n=50)	0.019
Hospitalization period [d]	12 (9/23) (n=50)	12 (9/22) (n=50)	0.718

d

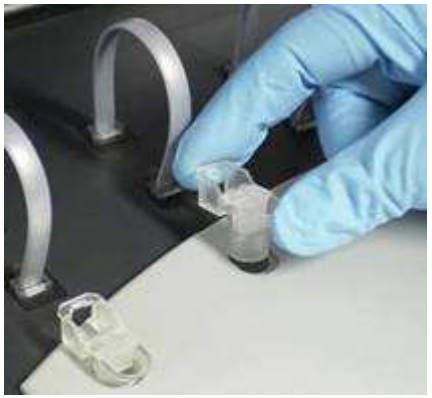
Figure 2. Primary and secondary outcomes of the first prospective randomized study in complex cardiac surgery including viscoelastic and aggregometric measures into a point-of-care (POC)-based algorithm for hemotherapy as proof-of-concept [13]. (a) Packed red blood cells (PRBC), fresh frozen plasma (FFP) and platelet concentrate (PC) transfusion rate; **(b)** postoperative chest tube blood loss during the first 24 h after admission to the intensive care unit; **(c)** mortality during a 6-month follow up observation period; and **(d)** ventilation time, PaO_2/FiO_2 – index, and length of stay in the intensive care unit and hospital. From [13] with permission.

Thrombocyta szám ≠ funkció




Procoagulation
Anticoagulation

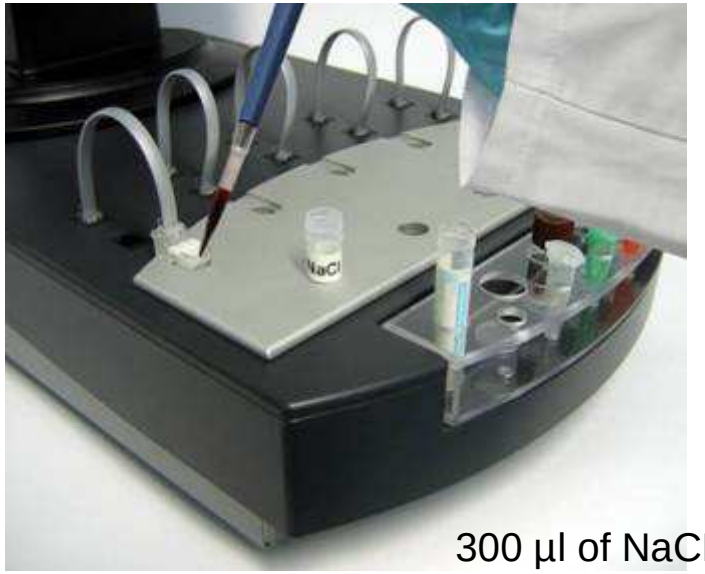
A mérés…



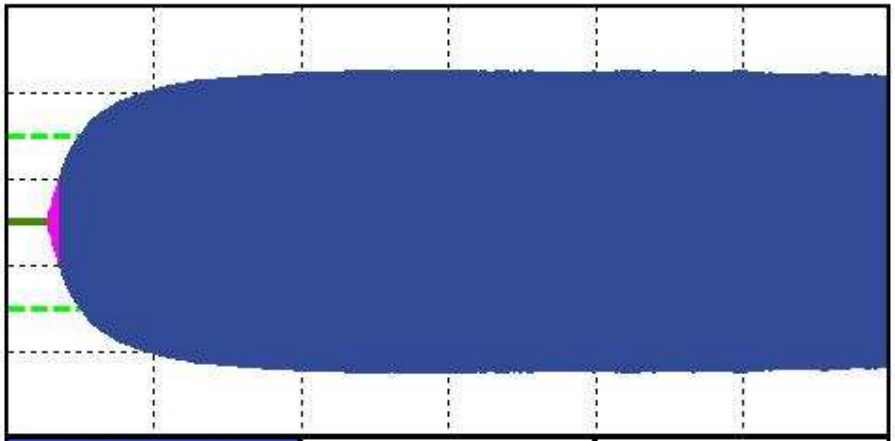
Hirudin vagy
Heparin 40 perc

Aktivátor + 6 perc

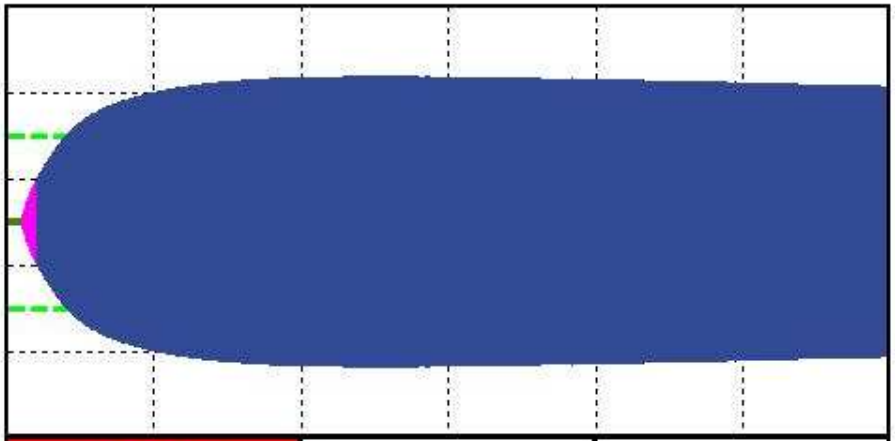
3 perc



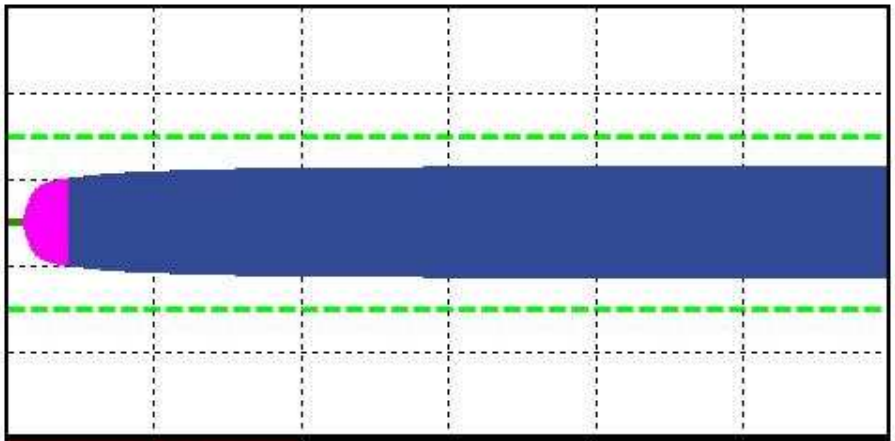
300 µl of NaCL
+ 300 µl vér



INTEM S	2013-12-06 14:47	2:
CT: 180s	CFT: 51s	α: 80°
A10: 64mm	A20: 69mm	MCF: 70mm



EXTEM S	2013-12-06 14:49	2:
CT: 70s	CFT: 67s	α: 79°
A10: 60mm	A20: 66mm	MCF: 67mm



FIBTEM S	2013-12-06 14:51	2:
CT: 78s	CFT: 192s	α: 80°
A10: 23mm	A20: 25mm	MCF: 25mm

TEG® vs. ROTEM®

Solomon C et al BJA 2012; 109 (6): 851–63

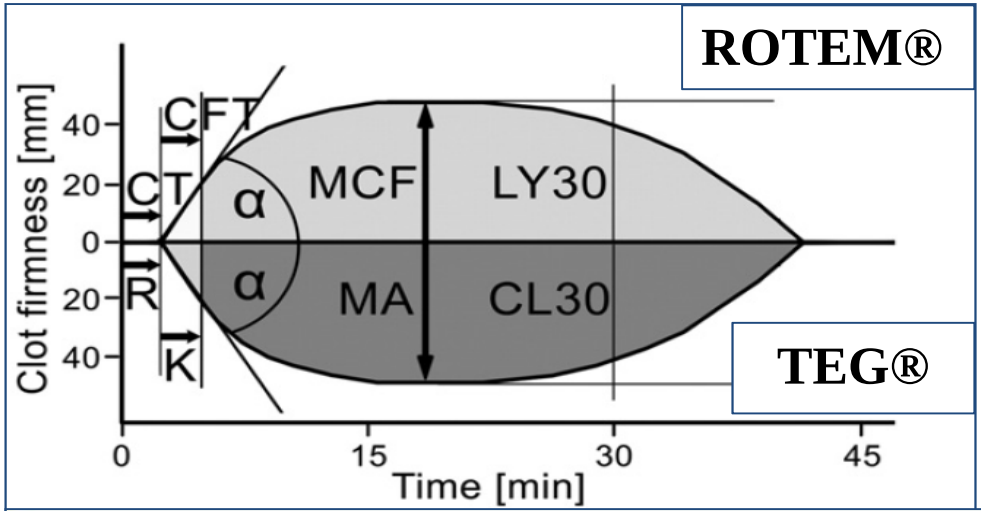


Table 1 Parameters recordable using TEG® and ROTEM®-based tests. *G=(5000×MA)/(1

Parameter recorded	TEG® value	ROTEM® value	Correlations with standard hemostatic laboratory tests
Coagulation initiation	*r* (reaction time)	CT (clotting time)	INR, aPTT, procoagulant factor levels
Clot formation	*k*	CFT (clot formation time)	
	α° (alpha angle)	α° (alpha angle)	Fibrinogen, platelet count
Clot strength/quality		A5, A10, A15, etc.	
	MA (maximum amplitude)	MCF (maximum clot firmness)	Fibrinogen, platelet count
	G (clot rigidity)	MCE (maximum clot elasticity)	
Clot lysis	LY30 (lysis)	LI30 (lysis index)	Fibrinogen, platelet count
		MI (maximum lysis)	Fibrin degradation products

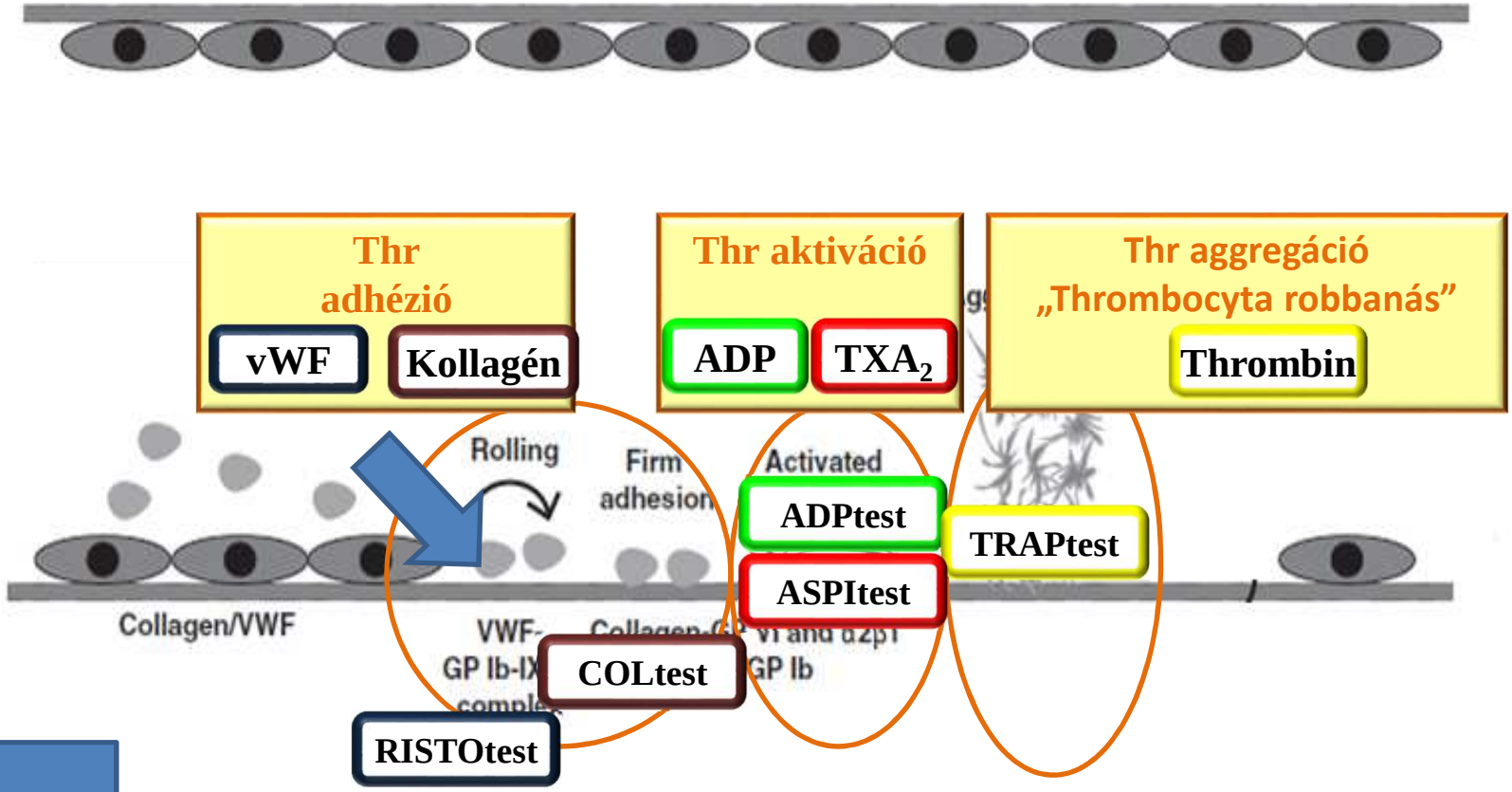
Stravicz RT et al Gastroent & Hepat 2012; 8: 513-520

Activators…

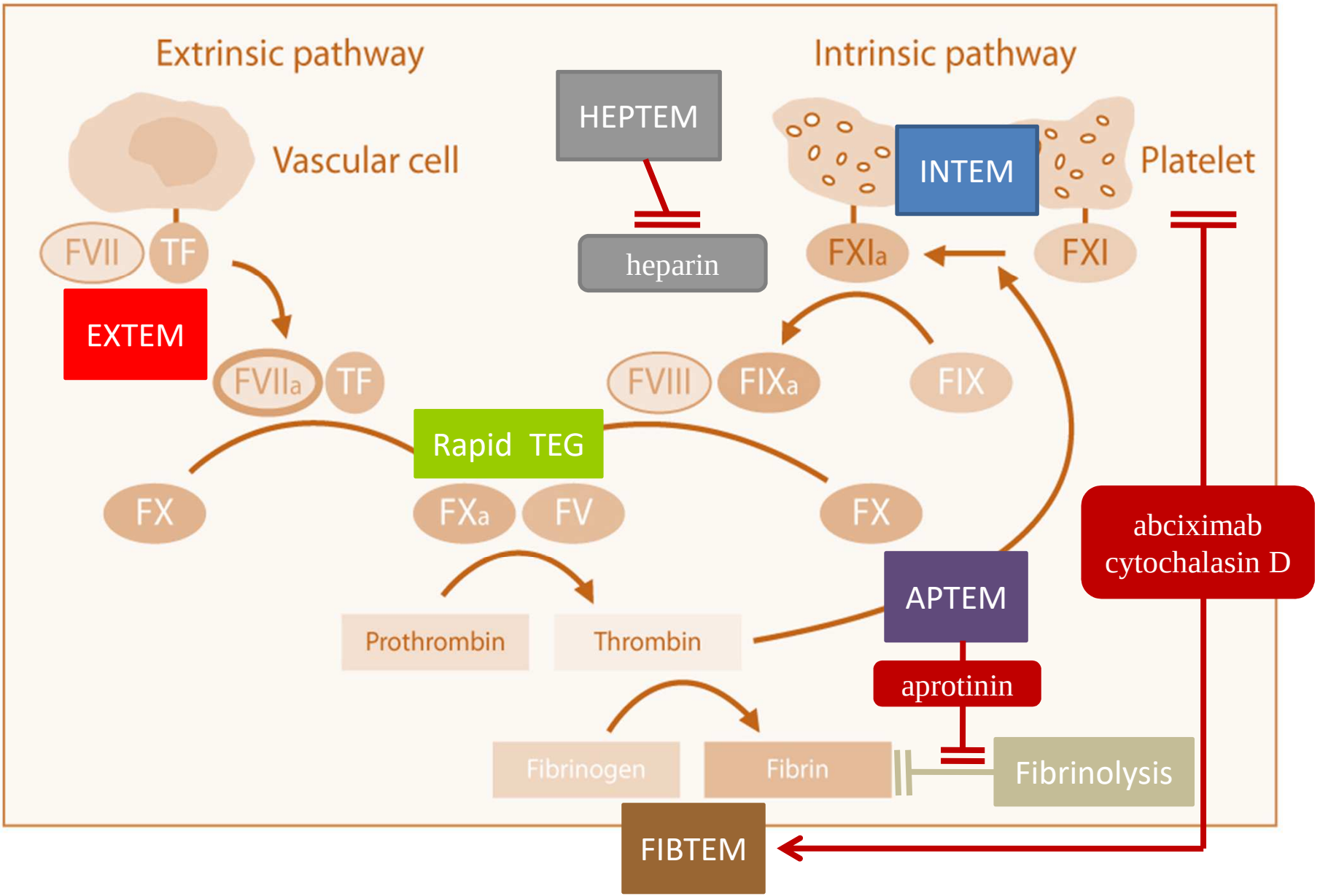
	Activation	Sensitive	Insensitive
RISTO test	**Ristocetin**: vWF dependens platelet activation via GpIb receptor	Bernard-Soulier syndroma, severe vWD, aspirin	
COL test	**Collagen:** platelet activation via GpIV and α2β1 receptor	aspirin, IIb/IIIa antagonists	clopidogrel, vWF
ASPI test	**arachidon acid:** activation via COX - TXA_2 pathway	aspirin, IIb/IIIa antagonist	clopidogrel, vWF
ADP test	**ADP**: ligand of platelet's ADP receptors	clopidogrel, IIb/IIIa antagonist	aspirin, vWF
TRAP test	**TRAP-6** :thrombin receptor activating peptide	IIb/IIIa antagonista	vWF, aspirin, clopidogrel

Platelet aggregometry (Multiplate®)

Zhang et al *J Neurotrauma* 2012; 29:2597–2605



kirni


Extrinsic pathway
Intrinsic pathway
HEPTEM
Vascular cell
INTEM
Platelet
heparin
FVII
TF
FXIa
FXI
EXTEM
FVIIa
TF
FVIII
FIXa
FIX
Rapid TEG
FX
FXa
FV
FX
abciximab
cytochalasin D
APTEM
Prothrombin
Thrombin
aprotinin
Fibrinogen
Fibrin
Fibrinolysis
FIBTEM