69 CONGRESO NACIONAL
SOCIEDAD ESPAÑOLA DE FARMACIA HOSPITALARIA

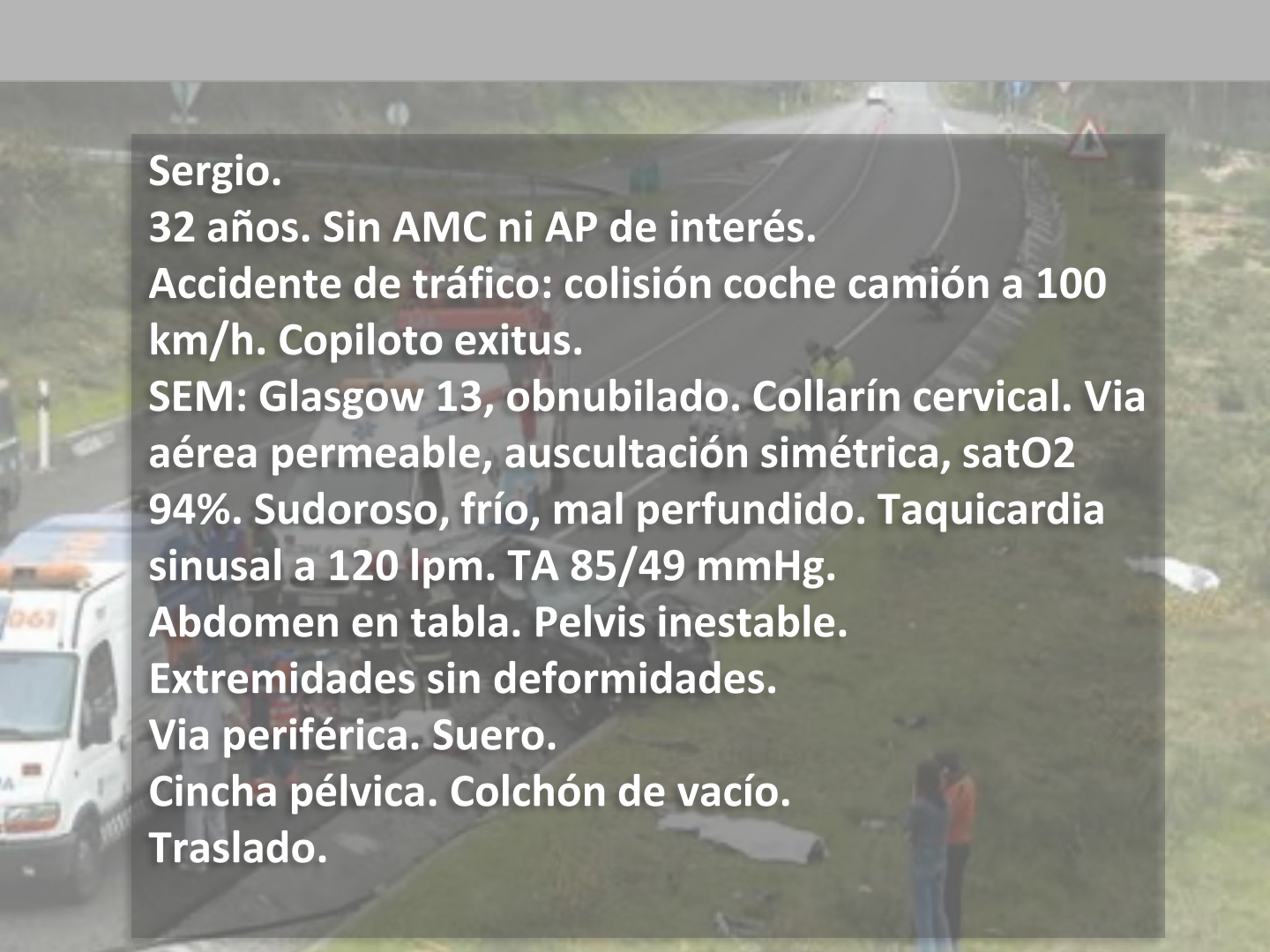
A CORUÑA
17-19 OCT 24

POLITRAUMATIZADO CON SHOCK HEMORRÁGICO

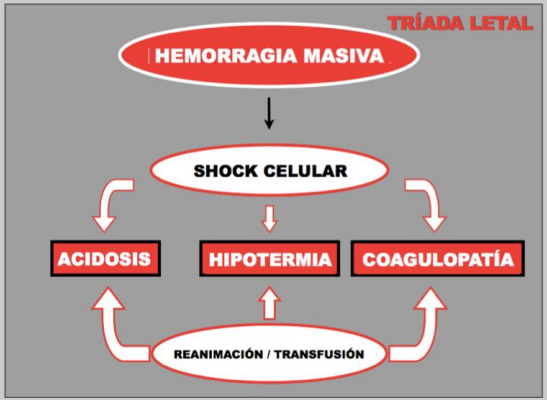
...buscando cómplices.

MARTA BARQUERO.
Anestesiología y Reanimación.

sefh
Sociedad Española de Farmacia Hospitalaria



Sergio.
32 años. Sin AMC ni AP de interés.
Accidente de tráfico: colisión coche camión a 100 km/h. Copiloto exitus.
SEM: Glasgow 13, obnubilado. Collarín cervical. Via aérea permeable, auscultación simétrica, satO2 94%. Sudoroso, frío, mal perfundido. Taquicardia sinusal a 120 lpm. TA 85/49 mmHg.
Abdomen en tabla. Pelvis inestable.
Extremidades sin deformidades.
Via periférica. Suero.
Cincha pélvica. Colchón de vacío.
Traslado.



Punto de partida/

La tríada letal

Acute Traumatic Coagulopathy

Karim Brohi, BSc, FRCS, FRCA, Jasmin Singh, MB, BS, BSc, Mischa Heron, MRCP, FFAEM, and Timothy Coats, MD, FRCS, FFAEM

Background: Traumatic coagulopathy is thought to be caused primarily by fluid administration and hypothermia.

Methods: A retrospective study was performed to determine whether coagulopathy resulting from the injury itself is a clinically important entity in severely injured patients.

Results: One thousand eight hundred sixty-seven consecutive trauma patients were reviewed, of whom 1,088 had full data sets. Median Injury Severity Score was 20, and 57.7% had an Injury Severity Score > 15; 24.4% of patients had a significant coagulopathy. Patients with an acute coagulopathy had significantly higher mortality (46.0% vs. 10.9%; χ^2, $p < 0.001$). The incidence of coagulopathy increased with severity of injury, but was not related to the volume of intravenous fluid administered ($r^2 = 0.25$, $p < 0.001$).

Conclusion: There is a common and clinically important acute traumatic coagulopathy that is not related to fluid administration. This is a marker of injury severity and is related to mortality. A coagulation screen is an important early test in severely injured patients.

Key Words: Traumatic coagulopathy, Hypothermia, Fluid administration.

J Trauma. 2003;54:1127–1130.

- ¼ parte de los pacientes politraumáticos con shock hemorrágico estan coagulopáticos (TP/TTPa>1.5)
- A peor ISS mayor coagulopatía
- Los pacientes coagulopáticos presentan mayor mortalidad, FMO y estancia hospitalaria.

Trauma Induced Coagulopathy (TIC)

CONSUMO: consumo de plaquetas y factores a nivel de las lesiones.

PÉRDIDA DE SANGRE

ACIDOSIS: alteración de la función de los factores de coagulación.

HIPOTERMIA: alteración de la función plaquetar.

COAGULOPATÍA DILUCIONAL: dilución de los factores debido al aporte de volumen.

ACoTS (Acute Coagulopathy of Trauma Shock): Mecanismo endógeno descrito en el paciente PPT grave secundario a un estado de hipoperfusión + importante lesión tisular, esto genera un estado de ANTICOAGULACIÓN + HIPERFIBRINOLISIS.

TIC

Trauma Induced Coagulopathy

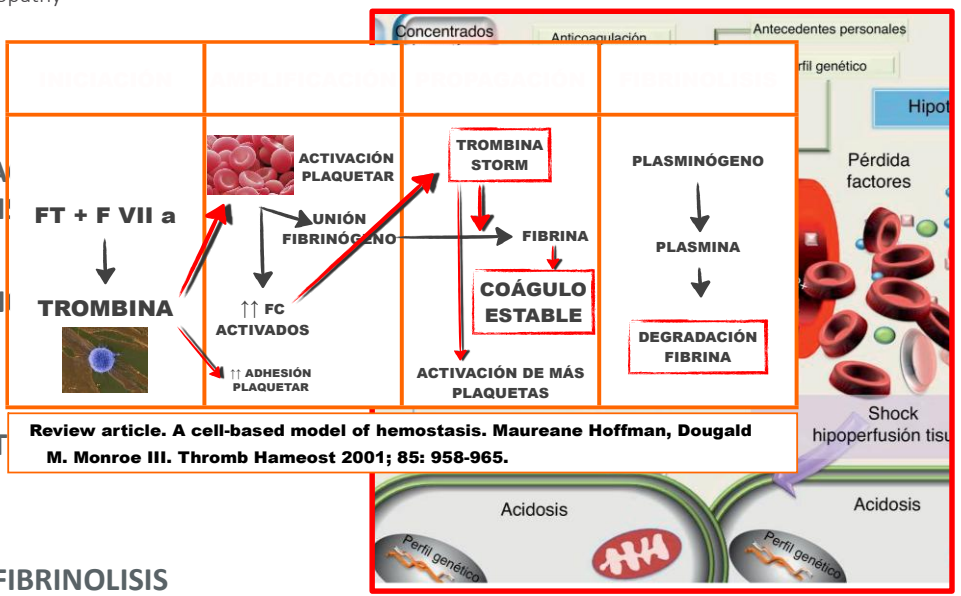
RESULTADO

ALTERA
SÍNTESI

DISFUN

DÉFICIT

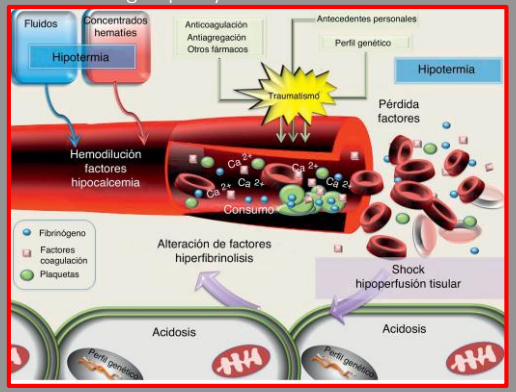
HIPERFIBRINOLISIS



Review article. A cell-based model of hemostasis. Maureane Hoffman, Dougald M. Monroe III. Thromb Hameost 2001; 85: 958-965.

TIC

Trauma Induces Coagulopathy



- Acute Traumatic Coagulopahy. Brohi K, Singh J, Heron M, Coats T. J Trauma. 2003;54:1127-1130.
- Acute coagulopathy of trauma: mechanism, identification and effect. Brohi K, Cohen M, Davenport R. Curr Opin Crit Care 13:680-685.

"We now know, from our work and the work of others, that our understanding of the disease processes was wrong, that our resuscitation goals were wrong and that our treatment was too little, too late." K. Brohi

TRATAMIENTO

⇩

DAMAGE CONTROL RESUSCITATION

1. Control **PRECOZ** de la hemorragia
2. Resucitación **HIPOTENSIVA**
3. Sueroterapia **RESTRICTIVA**
4. Transfusión precoz de hemocomponentes con ratio (CH:PFC:CP) ELEVADA

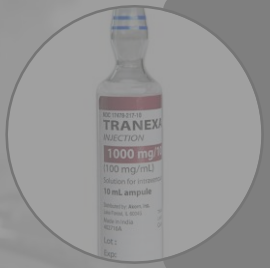
Table 1. Recent trials investigating fibrinogen replacement in severe trauma

Trial	Intervention	Setting	Sample size	Primary outcome	Results
CRYOSTAT-1	Early Cryo vs. Standard MHP	Trauma Unit	43	Time to delivery of SI	85% received Cryo < 90 min Time to delivery Cryo 60 min
E-FIT-1	FC vs. Placebo	Trauma Unit	40	Time to delivery of SI	69% received SI in < 45 min
FEISTY-Pilot	FC vs. Cryo	Trauma Unit	100	Time to delivery of SI	Time to FC 29 min Time to Cryo 60 min
FlinTIC	FC vs. Placebo	Pre-Hospital	53	Clot stability - ROTEM	FIBTEM ↑ in FC Arm FIBTEM ↓ in placebo arm
FiiRST-1	FC vs. Placebo	Trauma Unit	50	Time to delivery of SI	95% received SI in < 60 min
PRooF-iTH	FC vs. Placebo	Trauma Unit	40	Clot stability - TEG	Not reported

Pragmatic, Randomized Optimal Platelet and Plasma Ratios

DAMAGE CONTROL RESUSCITATION

PROTOCOLOS DE TRANSFUSIÓN MASIVA



ÁCIDO TRANEXÁMICO

FIBRINÓGENO

TÉCNICAS VISCOELÁSTICAS

DAMAGE CONTROL RESUSCITATION

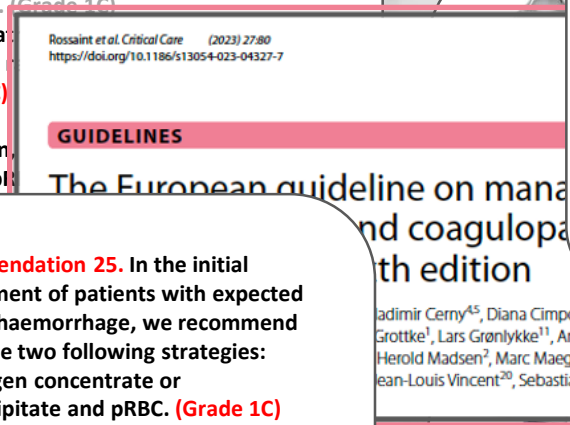

Critical Care

GUIDELINES Open Access

The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition

Recommendation 25. In the initial management of patients with expected massive haemorrhage, we recommend one of the two following strategies:
*Fibrinogen concentrate or cryoprecipitate and pRBC. (Grade 1C)
*FFP or pa
FFP/pRBC
(Grade 1C)

In addition
platelet/pl


Rossaint et al. Critical Care (2023) 27:80
https://doi.org/10.1186/s13054-023-04327-7

GUIDELINES

The European guideline on mana
nd coagulopa
th edition

adimir Cerny[4,5], Diana Cimpoesu[6], Nicola Curry[7,8],
Grottke[1], Lars Grønlykke[11], Anatole Harrois[9], Beverley J. Hunt[12],
Herold Madsen[2], Marc Maegele[13],
ean-Louis Vincent[20], Sebastian

Recommendation 23. We recommend that tranexamic acid be administered to the trauma patient who is bleeding or at risk of significant haemorrhage as soon as possible, if feasible en route to the hospital, and within 3h after injury at a loading dose of 1 g infused over 10 min, followed by an intravenous infusion of 1 g over 8h (Grade 1A)
We recommend that the administration of TXA not await results from a viscoelastic assessment (grade 1B)

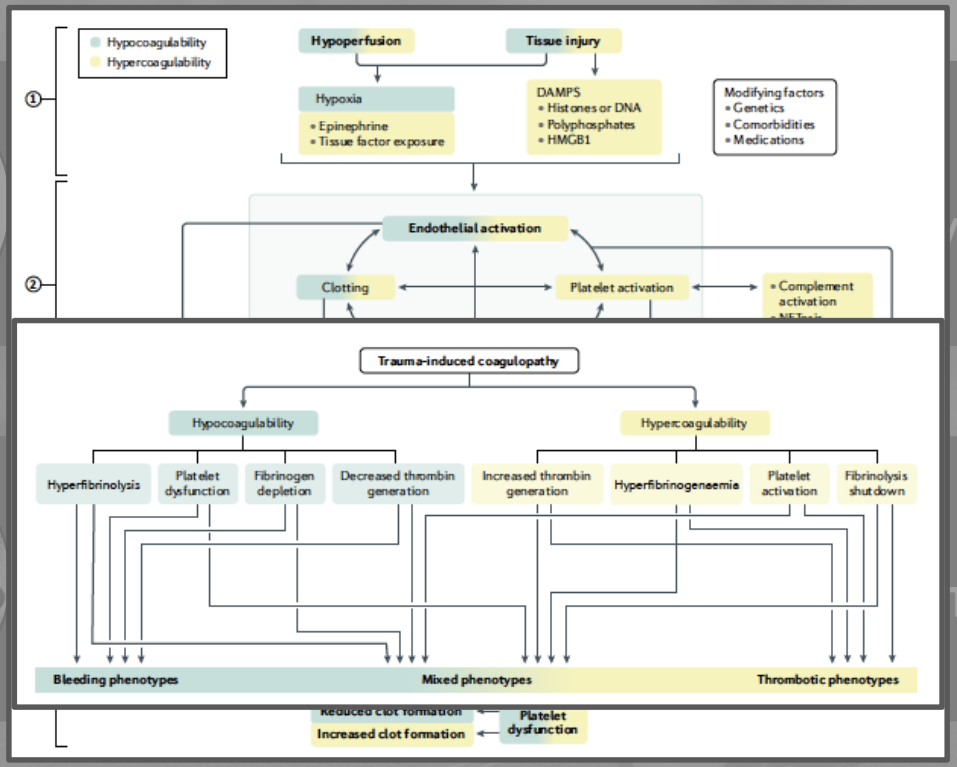
Recommendation 25. In the initial management of patients with expected massive haemorrhage, we recommend one of the two following strategies:
*Fibrinogen concentrate or cryoprecipitate and pRBC. (Grade 1C)
*FFP or pathogen-inactivated-FFP in a FFP/pRBC ratio of at least 1:2 as needed (1C)

Recommendation 24. We recommend that monitoring and measures to support coagulation be initiated immediately upon hospital admission (grade 1B)

Recommendation 26. We recommend that resuscitation measures be continued using a goal-directed strategy, guided by standard laboratory coagulation values and/or viscoelastic methods (Grade 1B)

Recommendation 29. We recommend treatment with fibrinogen concentrate or cryoprecipitate if major bleeding is accompained by hypofibrinogenemia (viscoelastic signs of a functional deficit or a plasma fibrinogen level of less than 1.5g/l) (Grade 1C)

DAMAGE CONTROL RESUSCITATION. Controversias



Trauma-induced coagulopathy. Moore, Hoffman, Schöchl, Hunt et al. Nature reviews. 2021

DAMAGE CONTROL RESUSCITATION. Controversias

- Metodología PROMMT/PROPPR
- Tratamiento empírico y agresivo
- Mejora en la síntesis de trombina?
- Dilución del fibrinógeno

ÁCIDO TRANEXÁMICO

FIBRINÓGENO

TÉCNICAS VISCOELÁSTICAS

DAMAGE CONTROL RESUSCITATION. Controversias

- Metodología PROMMT/PROPPR
- Tratamiento empírico y agresivo
- Mejora en la síntesis de trombina?
- Dilución del fibrinógeno

- Metodología CRASH-II
- Fibrinolysis shutdown

FIBRINÓGENO

TÉCNICAS VISCOELÁSTICAS

DAMAGE CONTROL RESUSCITATION. Controversias

- Metodología PROMMT/PROPPR
- Tratamiento empírico y agresivo
- Mejora en la síntesis de trombina?
- Dilución del fibrinógeno

- Metodología CRASH-II
- Fibrinolysis shutdown

- Falta de evidencia
- Fibrinógeno vs crioprecipitado
- Momento ideal, empírico?
- Trombosis?

TÉCNICAS VISCOELÁSTICAS

DAMAGE CONTROL RESUSCITATION. Controversias

- Metodología PROMMT/PROPPR
- Tratamiento empírico y agresivo
- Mejora en la síntesis de trombina?
- Dilución del fibrinógeno

- Metodología CRASH-II
- Fibrinolysis shutdown

- Falta de evidencia
- Fibrinógeno vs crioprecipitado
- Momento ideal, empírico?
- Trombosis?

- Falta de evidencia
- Hemostasia primaria
- Curva de aprendizaje
- Algoritmos no validados

PROTOCOLOS TRANSFUSIÓN MASIVA

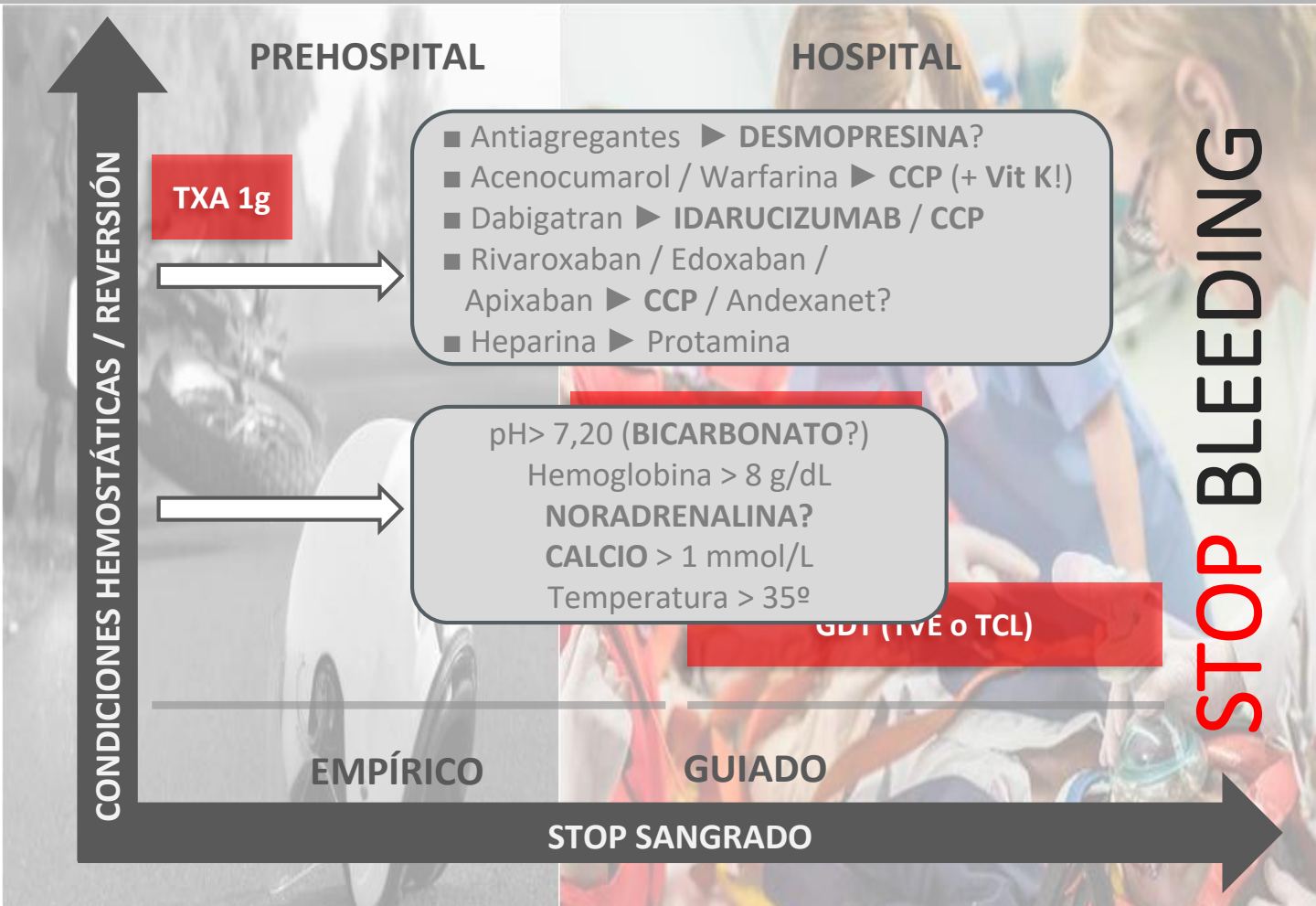
ÁCIDO TRANEXÁMICO

TICAS

Parámetro	Intervención
Déficit de otros FC	rFVIIa FVIII / FXIII
Trombocitopenia	Plaquetas
Defecto en la generación de trombina	CCP (o PFC)
Déficit de fibrinógeno	Concentrado de Fibrinógeno / crioprecipitado
Hiperfibrinolisis	Ácido tranexámico
Tratamientos previos	Aspirina? Clopidogrel? Warfarina? Heparina?
Condiciones hemostáticas	Establecer condiciones óptimas: Tc > 35ºC; pH 7,2; Ca iónico > 1mmol/L; Hb > 8g / dL
Sangrado	Detener el sangrado (cirugía, arteriografía, ...)

CCP:Concentrado de complejo protrombínico; PFC: Plasma fresco congelado; Tc Temperatura central; Hb: Hemoglobina

REANIMACIÓN HEMOSTÁTICA



TÉCNICAS VISCOELÁSTICAS: CÓMO FUNCIONAN?

PPT CON SOSPECHA DE COAGULOPATÍA

Paciente PPT inestable con importante lesión tisular.

MUESTRA DE SANGRE CON CITRATO

Es necesaria la obtención de una muestra de sangre en un tubo con citrato.

REACTIVO ESPECÍFICO

Vamos a usar un reactivo que contiene un activador de la coagulación.

CUBETA

Se pone en contacto una muestra de sangre con el reactivo, y la mezcla se deposita en una cubeta.

VARIEDAD DE REACTIVOS

Existen varios reactivos con propiedades específicas, vamos a obtener gráficos similares pero con interpretación variable segun el reactivo. La combinación de todos ellos nos va a proporcionar la máxima iinformación sobre la coagulación.

GRÁFICO

El cambio de resistencia detectado por el pistón se representa en un gráfico que nos informa de las propiedades viscoelásticas del coágulo.

MECANISMO

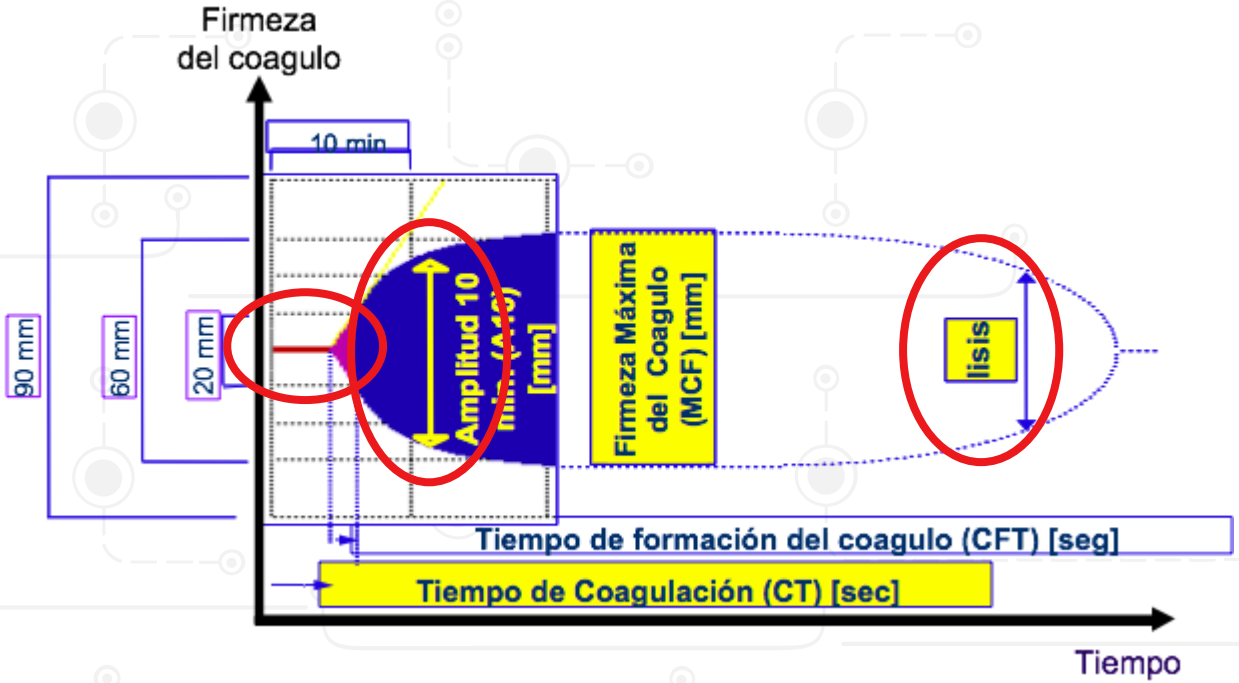
La sangre empieza a coagular debido a que se ha puesto en contacto con un reactivo activador de la coagulación.A medida que la sangre coagula el pistón detcta mayor resistencia dentro de la muestra.

PISTÓN

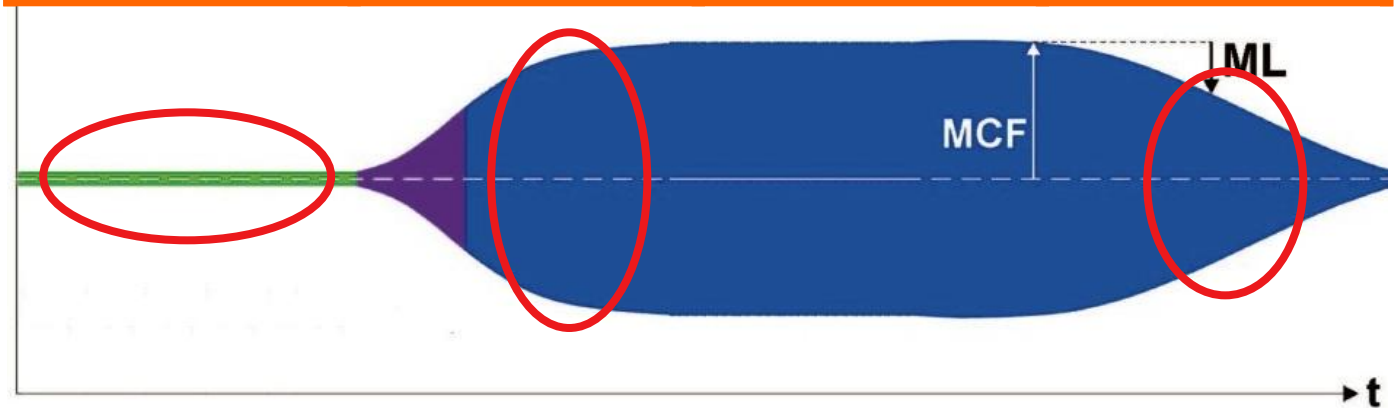
La mezcla se pone en contacto con un pistón que ejerce una fuerza rotatoria en la cubeta.

TÉCNICAS VISCOELÁSTICAS: CÓMO FUNCIONAN?

TÉCNICAS VISCOELÁSTICAS: INTERPRETACIÓN



INICIACIÓN	AMPLIFICACIÓN	PROPAGACIÓN	FIBRINOLISIS
FT + F VII a → TROMBINA 	 ACTIVACIÓN PLAQUETAR UNIÓN FIBRINÓGENO ↑↑ FC ACTIVADOS ↑↑ ADHESIÓN PLAQUETAR	TROMBINA STORM FIBRINA COÁGULO ESTABLE ACTIVACIÓN DE MÁS PLAQUETAS	PLASMINÓGENO → PLASMINA → DEGRADACIÓN FIBRINA



ORIGINAL

Viscoelastic haemostatic assay augmented protocols for major trauma haemorrhage (ITACTIC): a randomized, controlled trial

K. Baksaas-Aasen[1], L. S. Gall[2], J. Stensballe[3], N. P. Juffermans[4], N. Curry[5], M. Maegele[6], A. Brooks[7], C. Rourke[2], S. Gillespie[2], J. Murphy[8], R. Maroni[8], P. Vulliamy[2], H. H. Henriksen[3], K. Holst Pedersen[3], K. M. Kolstadbraaten[1], M. R. Wirtz[4], D. J. B. Kleinveld[4], N. Schäfer[6], S. Chinna[7], R. A. Davenport[2], P. A. Naess[1], J. C. Goslings[4], S. Eaglestone[2], S. Stanworth[5,9], P. I. Johansson[3], C. Gaarder[1] and K. Brohi[2*]

Abstract

Purpose: Contemporary trauma resuscitation prioritizes control of bleeding and uses major haemorrhage protocols (MHPs) to prevent and treat coagulopathy. We aimed to determine whether augmenting MHPs with Viscoelastic Haemostatic Assays (VHA) would improve outcomes compared to Conventional Coagulation Tests (CCTs).

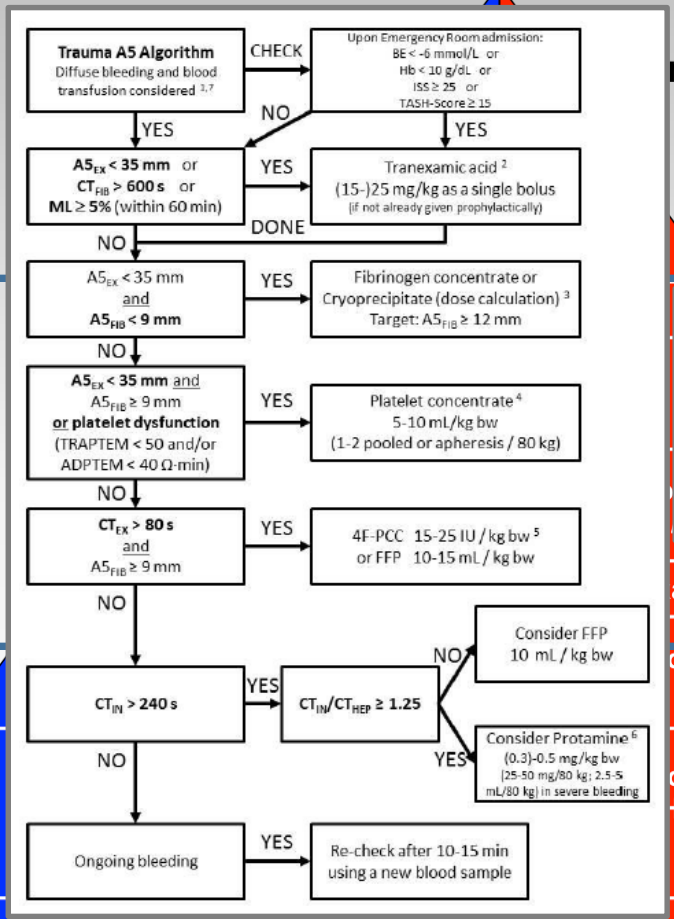
Methods: This was a multi-centre, randomized controlled trial comparing outcomes in trauma patients who received empiric MHPs, augmented by either VHA or CCT-guided interventions. Primary outcome was the proportion of subjects who, at 24 h after injury, were alive and free of massive transfusion (10 or more red cell transfusions). Secondary outcomes included 28-day mortality. Pre-specified subgroups included patients with severe traumatic brain injury (TBI).

Results: Of 396 patients in the intention to treat analysis, 201 were allocated to VHA and 195 to CCT-guided therapy. At 24 h, there was no difference in the proportion of patients who were alive and free of massive transfusion (VHA: 67%, CCT: 64%, OR 1.15, 95% CI 0.76–1.73). 28-day mortality was not different overall (VHA: 25%, CCT: 28%, OR 0.84, 95% CI 0.54–1.31), nor were there differences in other secondary outcomes or serious adverse events. In pre-specified subgroups, there were no differences in primary outcomes. In the pre-specified subgroup of 74 patients with TBI, 64% were alive and free of massive transfusion at 24 h compared to 46% in the CCT arm (OR 2.12, 95% CI 0.84–5.34).

Conclusion: There was no difference in overall outcomes between VHA- and CCT-augmented-major haemorrhage protocols.

Keywords: Trauma, Haemorrhage, Coagulopathy, Thrombelastography, Thromboelastometry

TRATAMIENTO DE LA COAGULOPATÍA GUIADA POR OBJETIVOS



The role of evidence-based algorithms for rotational thromboelastometry-guided bleeding management. *Görlinger K. Korean J Anesthesiol 2019.*

rvención

ámico

opidogrel? Warfarina?

ondiciones óptimas:
H 7,2; Ca iónico > 1mmol/L;

Sangrado — Detener el sangrado (cirugía, arteriografía, ...)

CCP:Concentrado de complejo protrombínico; PFC: Plasma fresco congelado; Tc Temperatura central; Hb: Hemoglobina

TRAT

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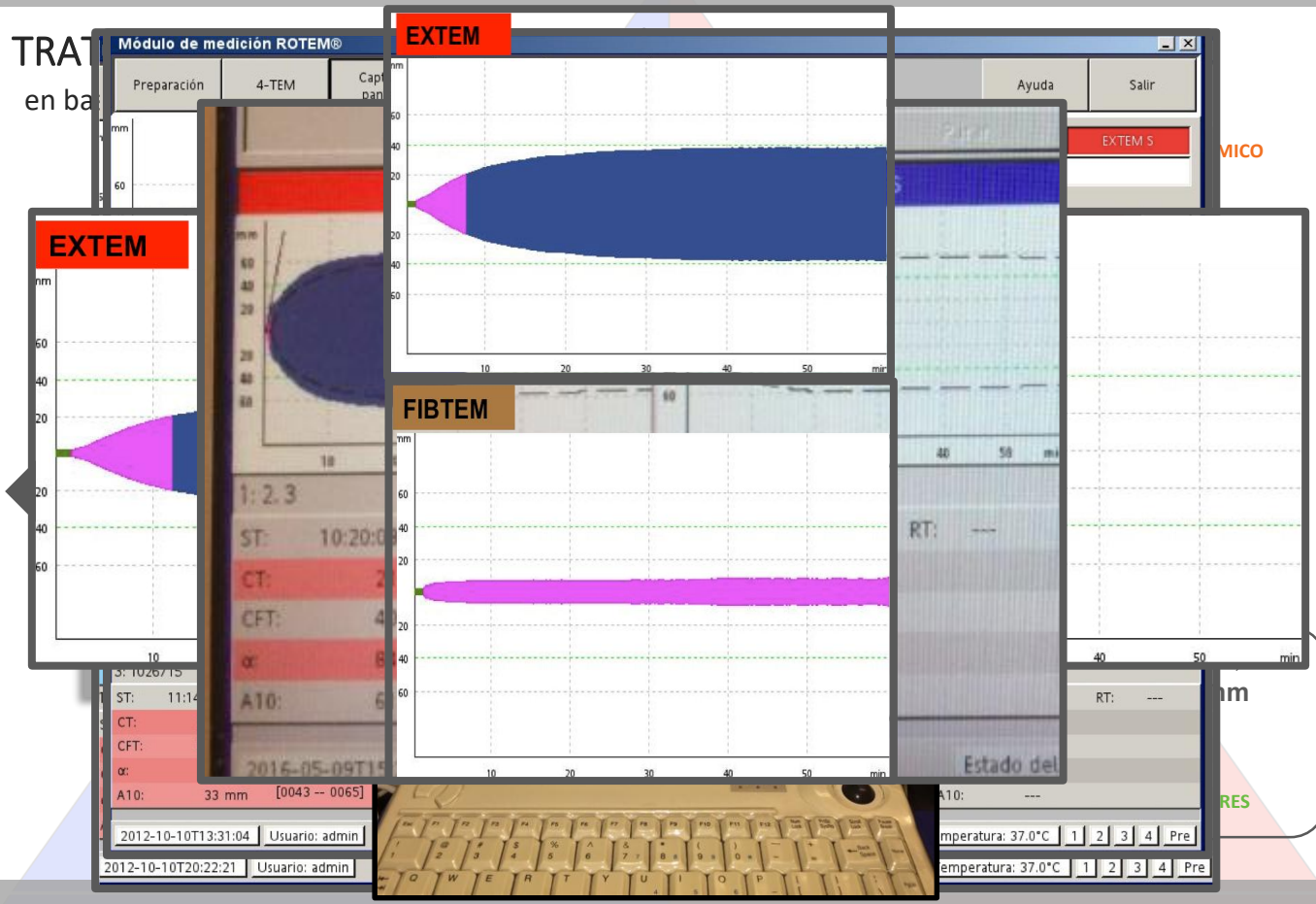


MICO

RES

Sangrado | Detener el sangrado (cirugía, arteriografía, ...)

REANIMACIÓN HEMOSTÁTICA (la alternativa?)

REANIMACIÓN HEMOSTÁTICA (la alternativa?)

REANIMACIÓN HEMOSTÁTICA (la alternativa?)

European Journal of Trauma and Emergency Surgery (2022) 48:763–774
https://doi.org/10.1007/s00068-020-01563-2

REVIEW ARTICLE

Variations and obstacles in the use of coagulation factor concentrates for major trauma bleeding across Europe: outcomes from a European expert meeting

Vladimir Černý[1] · Marc Maegele[2] · Vanessa Agustini[3] · Dietmar Fries[4] · Santiago R. Leal-Noval[5] · Gábor Nardai[6] · Giuseppe Nardi[7] · Anders Östlund[8] · Herbert Schöchl[9]

Received: 26 May 2020 / Accepted: 19 November 2020 / Published online: 5 January 2021

- We suggest a simple definition of TIC.
- We propose a simple set of criteria to guide when to administer an MTP in the majority of clinical trauma settings. However,
- Immediate admi
- We suggest that C, ie upon hospital admission
- We suggest that **hypofibrinogener**
- **Impaired thromb** **ated bleeding management**, as t buting factors, such as hypobrinogenemia and hyperfibrinolysis, should be managed first and the severity/risk of ongoing bleeding determined, before PCC administration.
- We believe a **step-wise approach** to the treatment for trauma-related bleeding allows for individualised therapy, and avoids overtreatment and unnecessary allogenic transfusion.

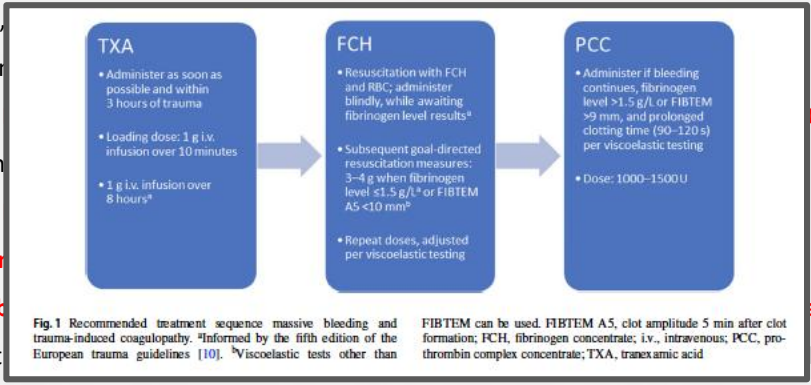
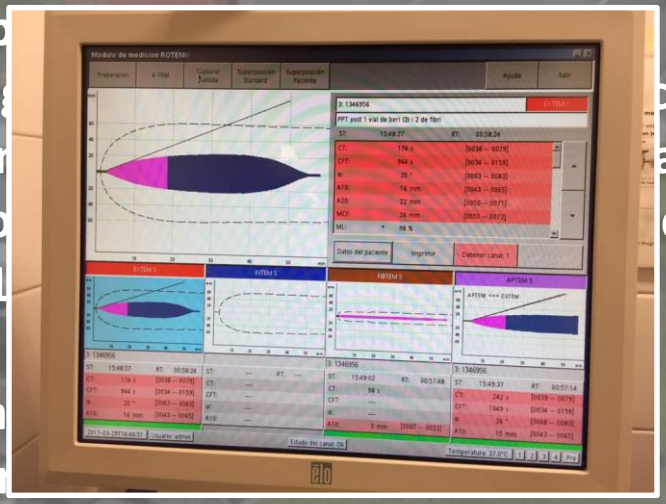


Fig. 1 Recommended treatment sequence massive bleeding and trauma-induced coagulopathy. [a]Informed by the fifth edition of the European trauma guidelines [10]. [b]Viscoelastic tests other than FIBTEM can be used. FIBTEM A5, clot amplitude 5 min after clot formation; FCH, fibrinogen concentrate; i.v., intravenous; PCC, prothrombin complex concentrate; TXA, tranexamic acid

Sergio.

32 años. Sin AMC ni AP de interés.

Accidente de tráfico: colisión coche camión a 100 km/h. Cop



SEM: Glas cervical. Via aérea perr a, satO2 94%. Sudo quicardia sinusal a 1

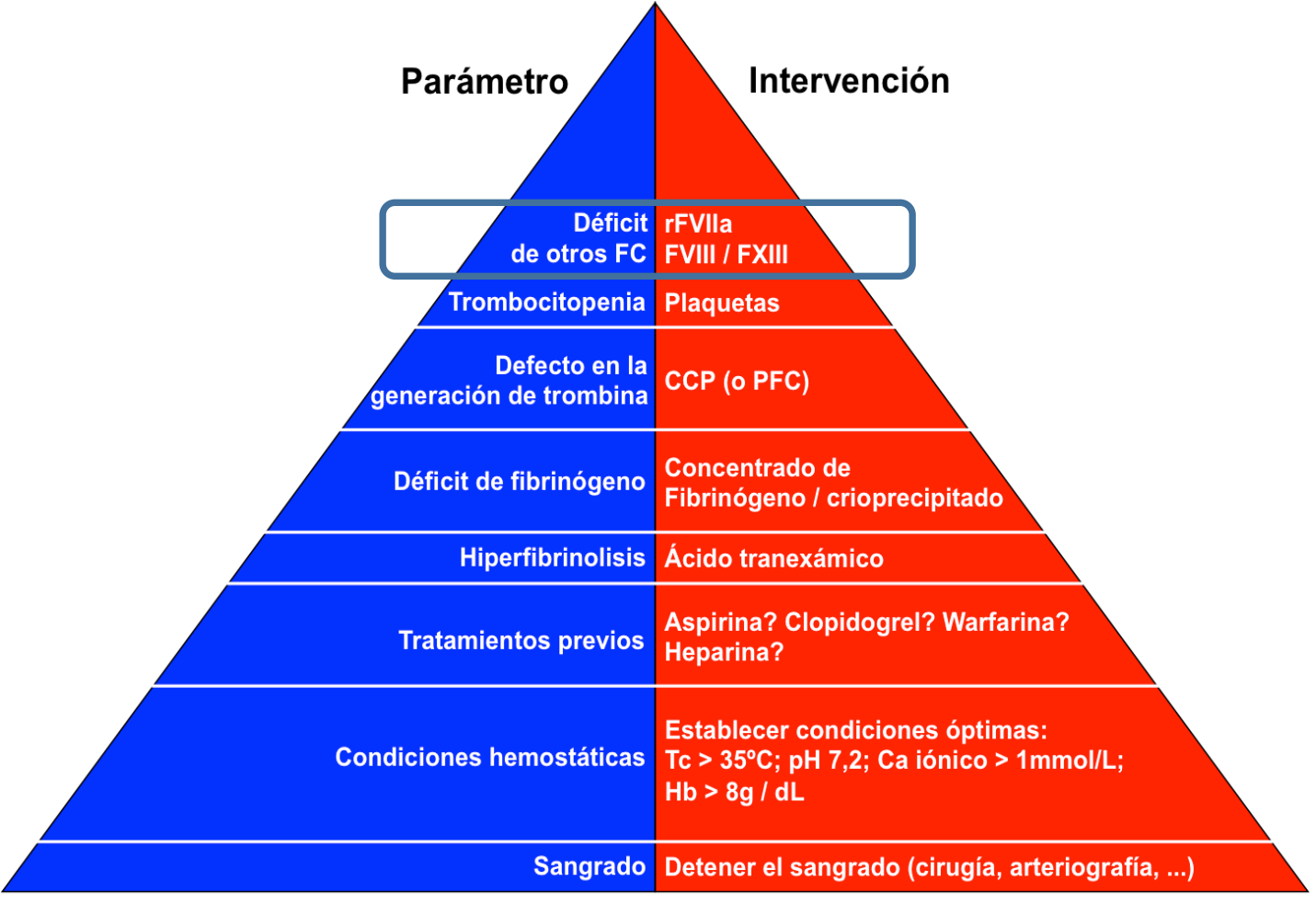
Abdomen

Extremida

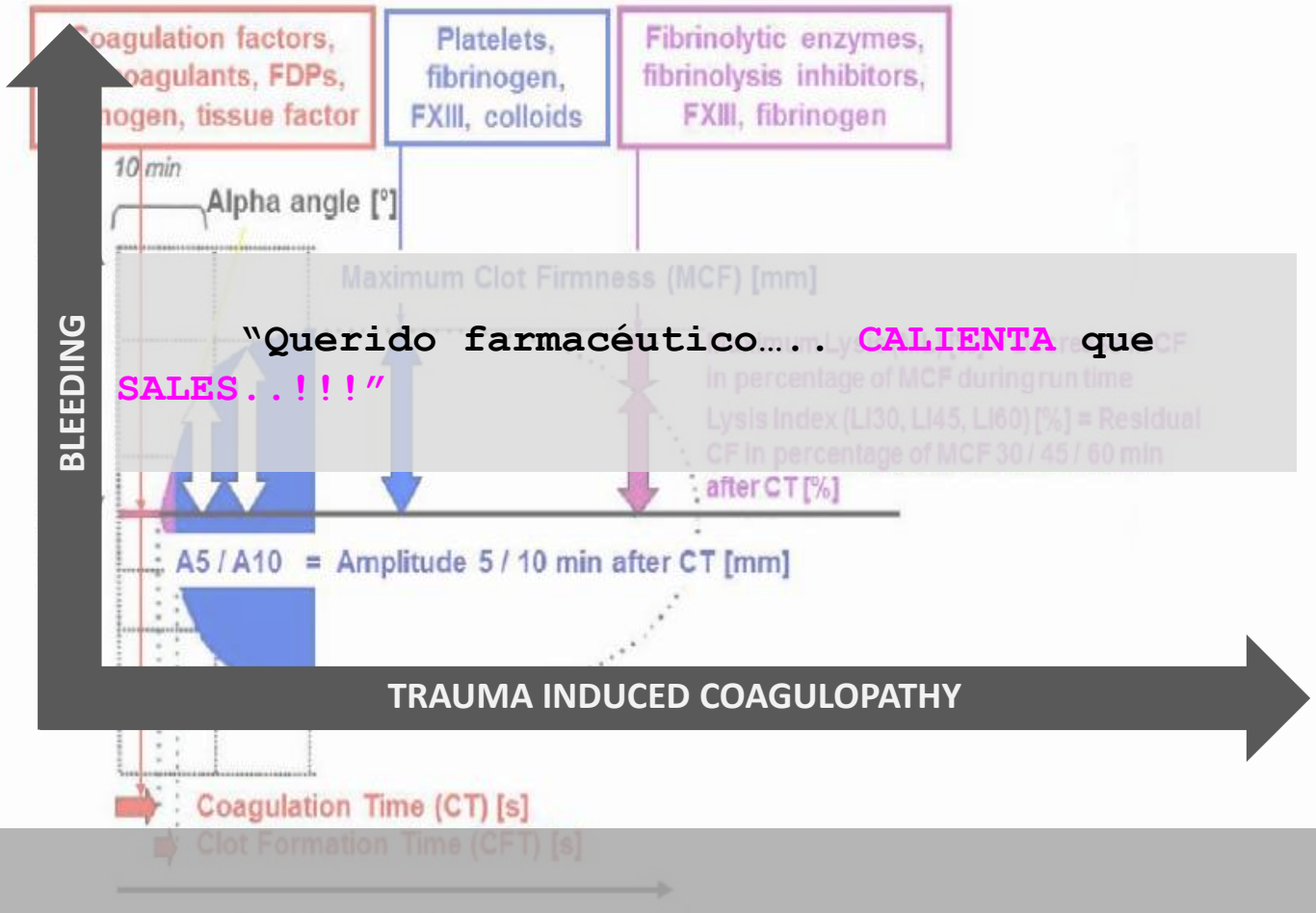
Via periféi

Cincha pélvica. Colchón de vacío.

Traslado.



CCP:Concentrado de complejo protrombínico; PFC: Plasma fresco congelado; Tc Temperatura central; Hb: Hemoglobina



"Querido farmacéutico….. CALIENTA que SALES..!!!"

ABRAZANDO LA EXCELENCIA · CUIDANDO EN TODAS LAS DIRECCIONES

FARMACIA 360º

A CORUÑA
17-19 OCT 24

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SOCIEDAD ESPAÑOLA DE FARMACIA HOSPITALARIA

Marta_barquero@hotmail.com

sefh Sociedad Española de Farmacia Hospitalaria