TEG: The ABCs of Implementing Thromboelastography in a Trauma Center

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Neuro/Critical Care CNS

Mission Hospital

Learning Objectives

- Implement TEG into a Trauma Center
- Describe the normal/abnormal dynamic clotting parameters of Thromboelastography (TEG) and propose treatment using an algorithm
- Strategize treatment options involving actual cases of hemorrhage

Disclosure Statement

- Bader
  - Board of Directors: Secretary
    - Neurocritical Care Society
  - Honorarium
    - Bard
    - Integra
  - Medical Advisory Board
    - Brain Trauma Foundation and Neuroptics
  - Scientific Advisory Board
    - Cerebrotech
  - Stock options
    - Neuoptics and Cerebrotech

Hemorrhage States

- Trauma
  - Traumatic Brain Injury
- Intracranial Hemorrhage
  - ICH
  - SAH
- GI Bleeding
- Liver disease/disorders
- OB Hemorrhage
- Ruptured vessels

Trauma Injury, Hemorrhage, & TBI

- Trauma/Injury is the 2nd leading cause of death globally
  - 40% of mortality associated with injury due to uncontrollable hemorrhage
- 1/3 of severely injured trauma patients sustain Trauma Induced coagulopathy (TIC)
  - Poorly understood mechanisms
  - Several theories
- Coagulopathy of TBI (CTBI) is a component of TIC
  - Multiple theories contribute to early platelet dysfunction
  - Correlation between severity of TBI and platelet dysfunction

Coagulopathy of TBI (CTBI)

- Presence of CTBI ranges 10-97% in ROL due to many factors

11. Harhangi, B.S. Kompanje, E.J., Leebeek, F.W., and Maas, A.I. (2008). Coagulation disorders after traumatic brain injury. Acta Neurochir. (Wien) 150, 165–175.

  - Heterogeneity of patients, types of lab tests, timing of tests, and lack of clear defined consensus to define CTBI
  - Associated with poor outcomes
  - Blunt TBI: coagulopathy increases mortality (50% vs 17.3%) compared to no coagulopathy
  - Factors increase risk include GCS≤8, ISS≥16, hypotension on admit, cerebral edema, SAH, shift

Traumatic Brain Injury-Associated Coagulopathy

Jianning Zhang,[1] Rongcai Jiang,[1] Li Liu,[1] Timothy Watkins,[2,3] Fangyi Zhang,[4] and Jing-fei Dong[1–3]

JOURNAL OF NEUROTRAUMA 29:2597–2605 (November 20, 2012)

DOI: 10.1089/neu.2012.2348

Coagulopathy of TBI (CTBI)

- Platelets & Platelet Activating Factor Theories
  - TBI may result in platelet hyperactivity
  - Platelet activating factor (PAF) induces aggregation and contributes to hypoxia-induced breakdown of the BBB
  - Tissue Factor normally not exposed to circulating blood volume…in TBI brain tissue (rich in TF) & platelets (breakdown) release TF in response to the injury and other cellular dynamics

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Exhausted Platelet Dysfunction

- BBB disruption releasing TF (Castellino et al 2014)
  - Qualitatively different form that found in most tissues (unexposed to soluble clotting factors –unsaturated by factor VII)
  - Liberation of free TF into circulation, provokes TF binding to VIIa on a massive scale
    - Results in stimulation of thrombin production in the initiation phase
    - Flood of TF –generated thrombin results in platelet exhaustive syndrome
    - Large numbers of circulating platelets **exist in a refractory state**
      - Leads to Platelet inhibition at the ADP receptor site (Davis et al 2013)
      - Platelets incapable of stimulation and cannot form a stable thrombus through usual pathways
      - Platelet count usually normal (Davis et al 2013)
    - No evidence of fibrinolysis (Davis et al 2013)

J Trauma Acute Care Surg. 2014 May ; 76(5): 1169–1176. doi:10.1097/TA.0000000000000216.

Traumatic brain injury causes platelet adenosine diphosphate and arachidonic acid receptor inhibition independent of hemorrhagic shock in humans and rats

METHODS—We used thrombelastography with platelet mapping as a measure of platelet function, assessing the degree of inhibition of the adenosine diphosphate (ADP) and arachidonic acid (AA) receptor pathways. First, we studied the early effect of TBI on platelet inhibition by performing thrombelastography with platelet mapping on rats. We then conducted an analysis of admission blood samples from trauma patients with isolated head injury (n = 70). Patients in shock or on clopidogrel or aspirin were excluded.

RESULTS—In rats, ADP receptor inhibition at 15 minutes after injury was 77.6% ± 6.7% versus 39.0% ± 5.3% for controls ($p < 0.0001$). Humans with severe TBI (Glasgow Coma Scale [GCS] score ≤ 8) showed an increase in ADP receptor inhibition at 93.1% (interquartile range [IQR], 44.8–98.3%; n = 29) compared with 56.5% (IQR, 35–79.1%; n = 41) in milder TBI and 15.5% (IQR, 13.2–29.1%) in controls ($p = 0.0014$ and $p < 0.0001$, respectively). No patient had significant hypotension or acidosis. Parallel trends were noted in AA receptor inhibition.

CONCLUSION—Platelet ADP and AA receptor inhibition is a prominent early feature of CTBI in humans and rats and is linked to the severity of brain injury in patients with isolated head trauma. This phenomenon is observed in the absence of hemorrhagic shock or multisystem injury. Thus, TBI alone is shown to be sufficient to induce a profound platelet dysfunction. (*J Trauma Acute Care Surg*. 2014;76: 1169–1176.

Rats c TBI – TEGPM
Human TBI 70 Pts
TEGPM done
Rats: ADP inhibition within 15 min
Humans: ADP inhibition 93% in TBI

Francis J. Castellino, PhD, Michael P. Chapman, MD, Deborah L. Donahue, Scott Thomas, MD, Ernest E. Moore, MD, Max V. Wohlauer, MD, Braxton Fritz, Robert Yount, MD, Victoria Ploplis, PhD, Patrick Davis, Edward Evans, and Mark Walsh, MD

J Trauma Acute Care Surg. 2014 May ; 76(5): 1169–1176. doi:10.1097/TA.0000000000000216.

WHY TEG?

Assessing Coagulopathy

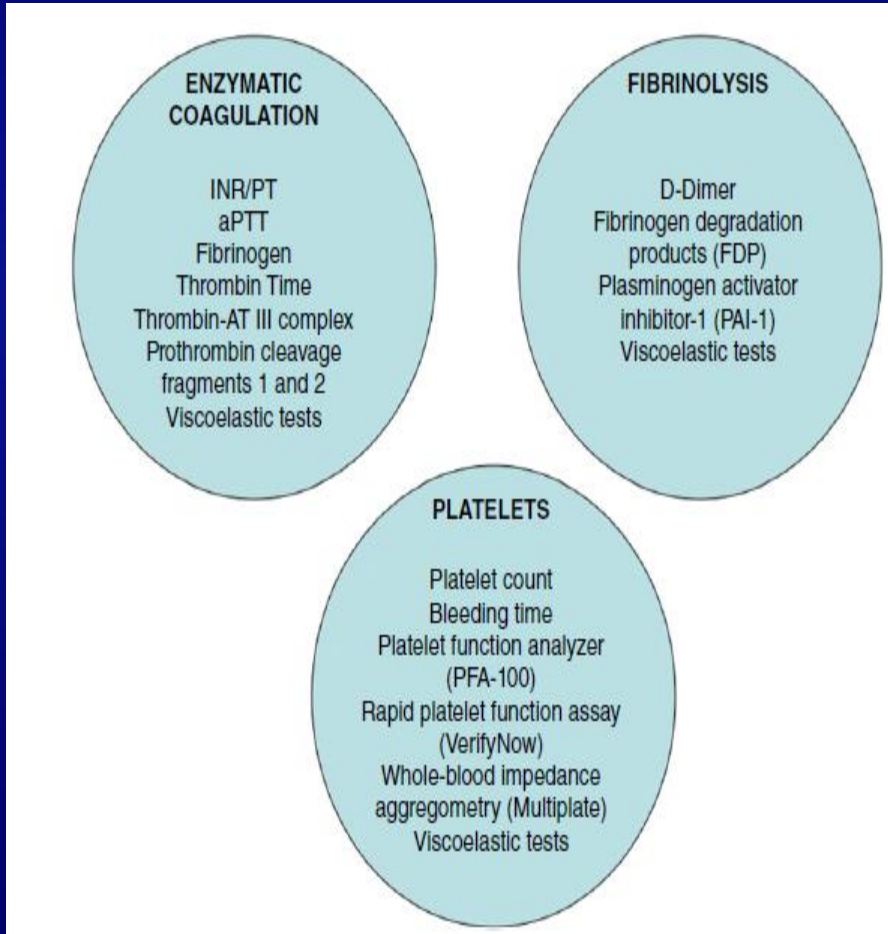


Fig. 2. Laboratory tests currently available to assess the coagulopathy after TBI (modified from[11]).

Coagulopathy after traumatic brain injury: incidence, pathogenesis, and treatment options

Marc Maegele

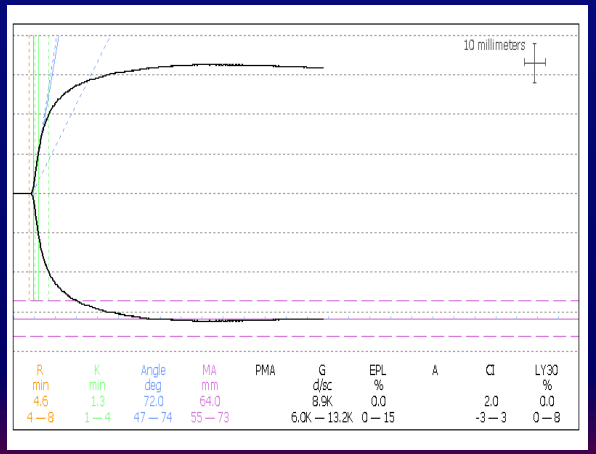
Measuring TIC and CTBI
Value of Viscoelastic Analysis

- Viscoelastical Hemostatic Assays (VHAs) tests that reflect the new understanding of hemostasis
  - Initiation – Amplification –Propagation
  - TEG and ROTEM
- VHAs assess properties of coagulation in whole blood
  - Can differentiate between low fibrinogen and reduced platelet function as the cause of impaired clot strength as well as systemic hyperfibrinolysis
  - Clinical value of VHA is corroborated by > 30 clinical studies on patients with massive hemorrhage—
    - Demonstrates Superiority over conventional coagulation tests

Hemostasis Monitoring:

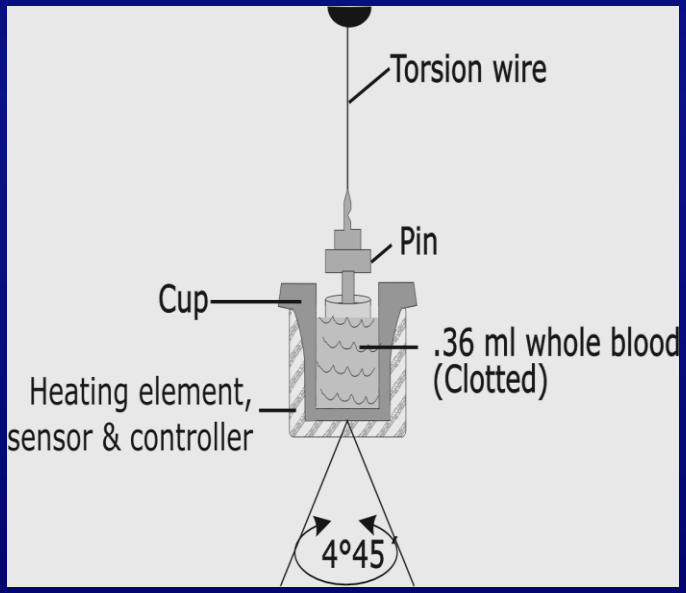
TEG Hemostasis System

- Whole blood test
- Measures hemostasis
  - Clot initiation through clot lysis
  - Net effect of components
- TEG system
  - Laboratory based
  - Point of care
  - Remote, can be networked
  - Flexible to institution



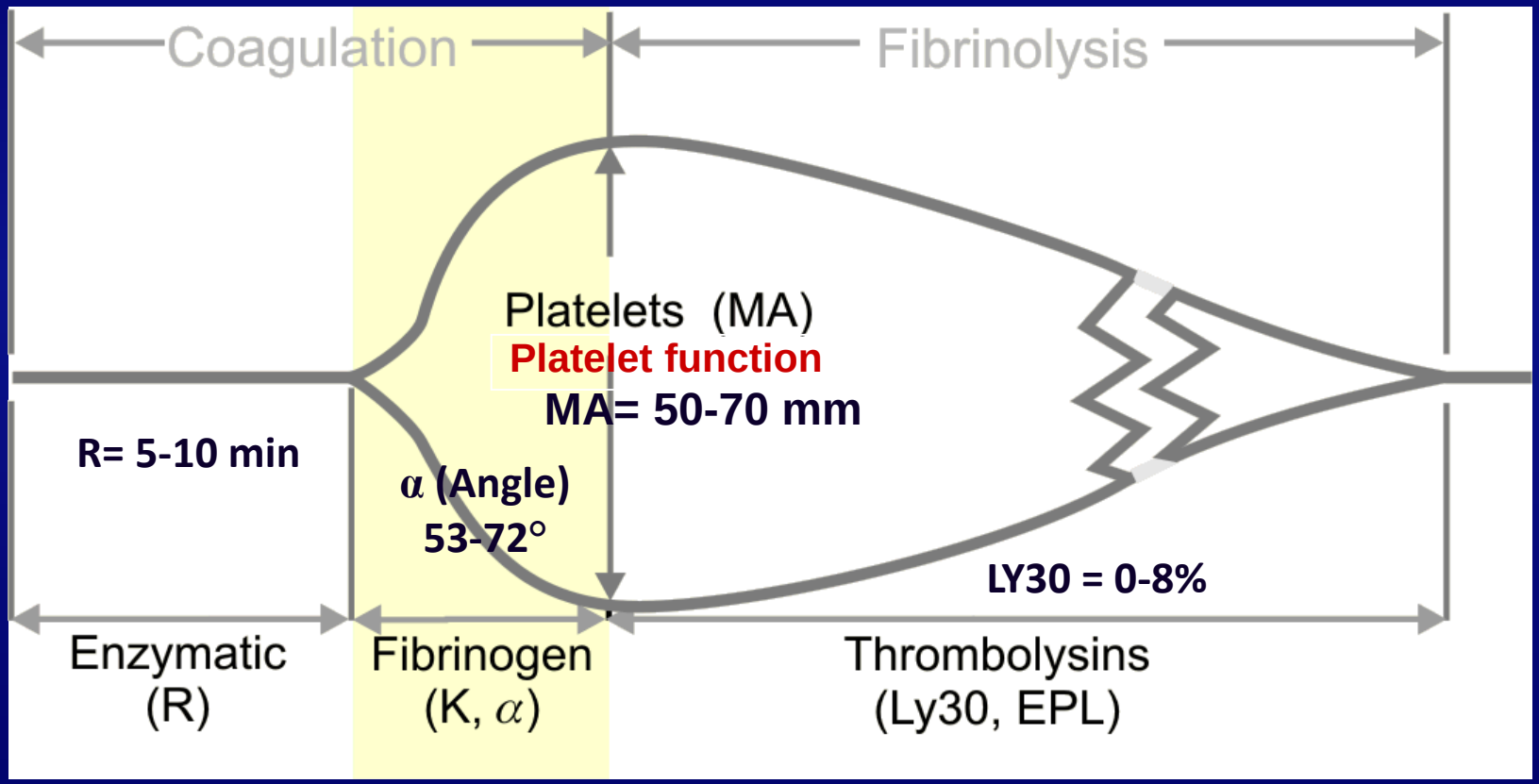
TEG Technology:

How It Works



- Cup oscillates
- Pin is attached to a torsion wire
- Clot binds pin to cup
- Degree of pin movement is a function of clot kinetics
- Magnitude of pin motion is a function of the mechanical properties of the clot
- System generates a hemostasis profile
  - From initial formation to lysis

TEG Parameter Summary

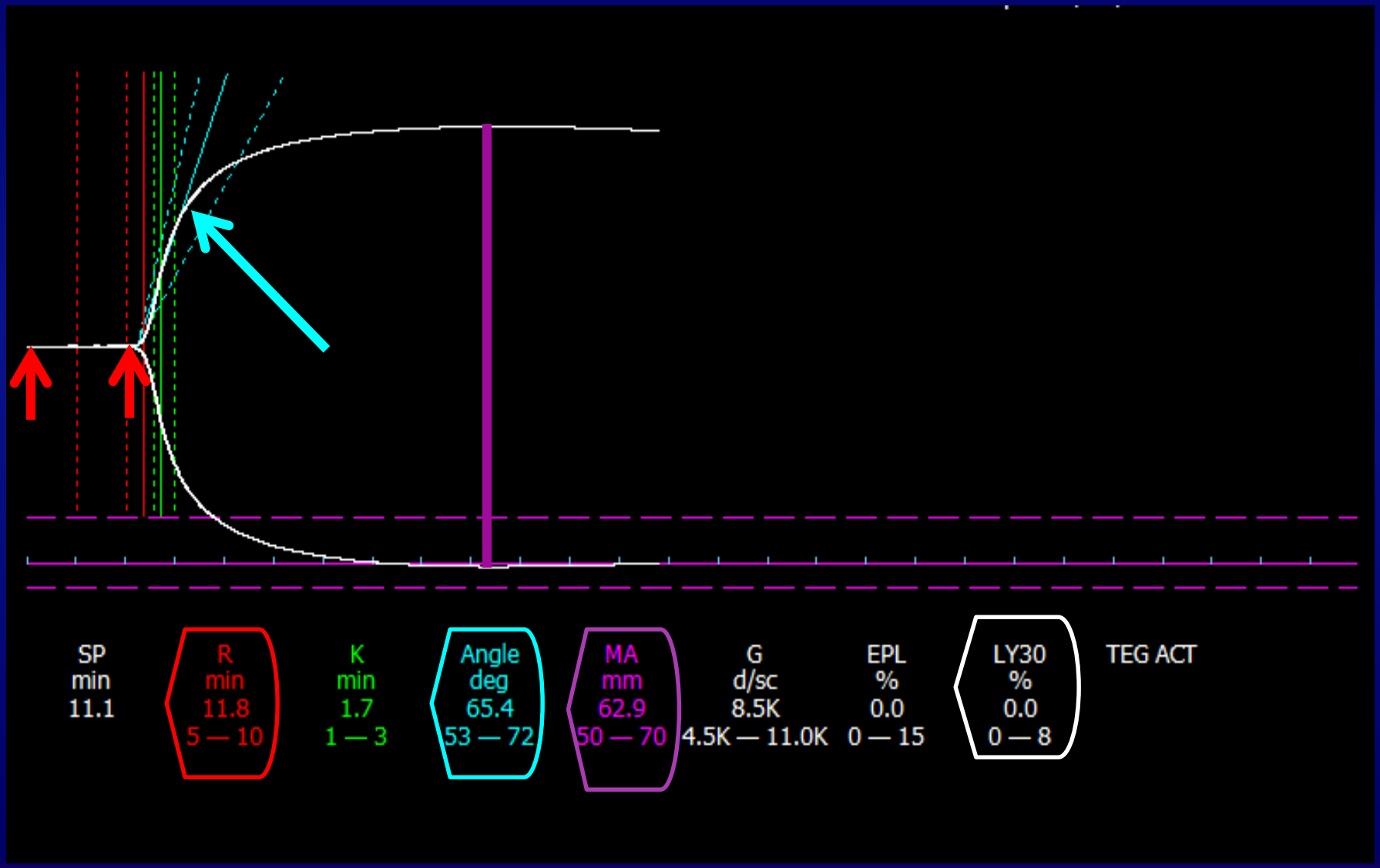


Clotting time

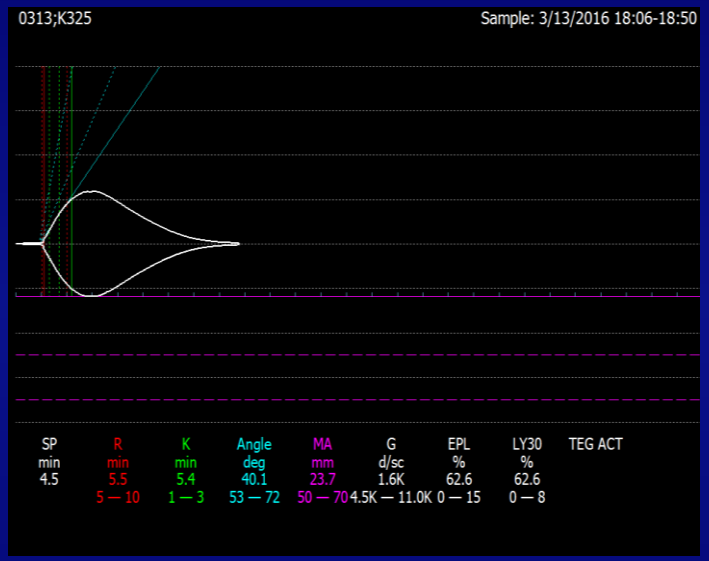
Clot kinetics

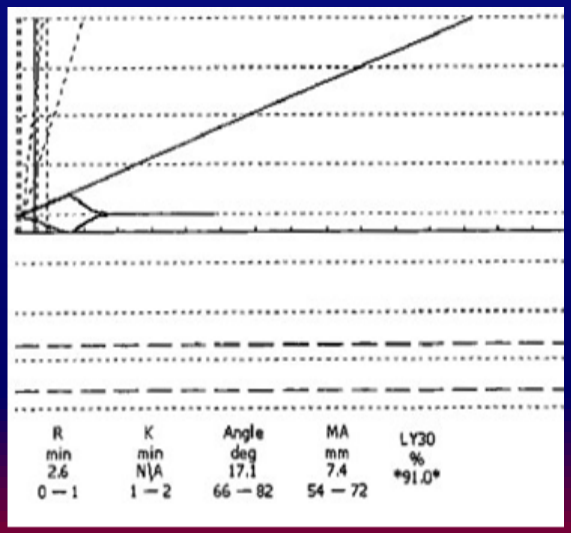
Clot stability
Clot breakdown

Standard TEG Tracing



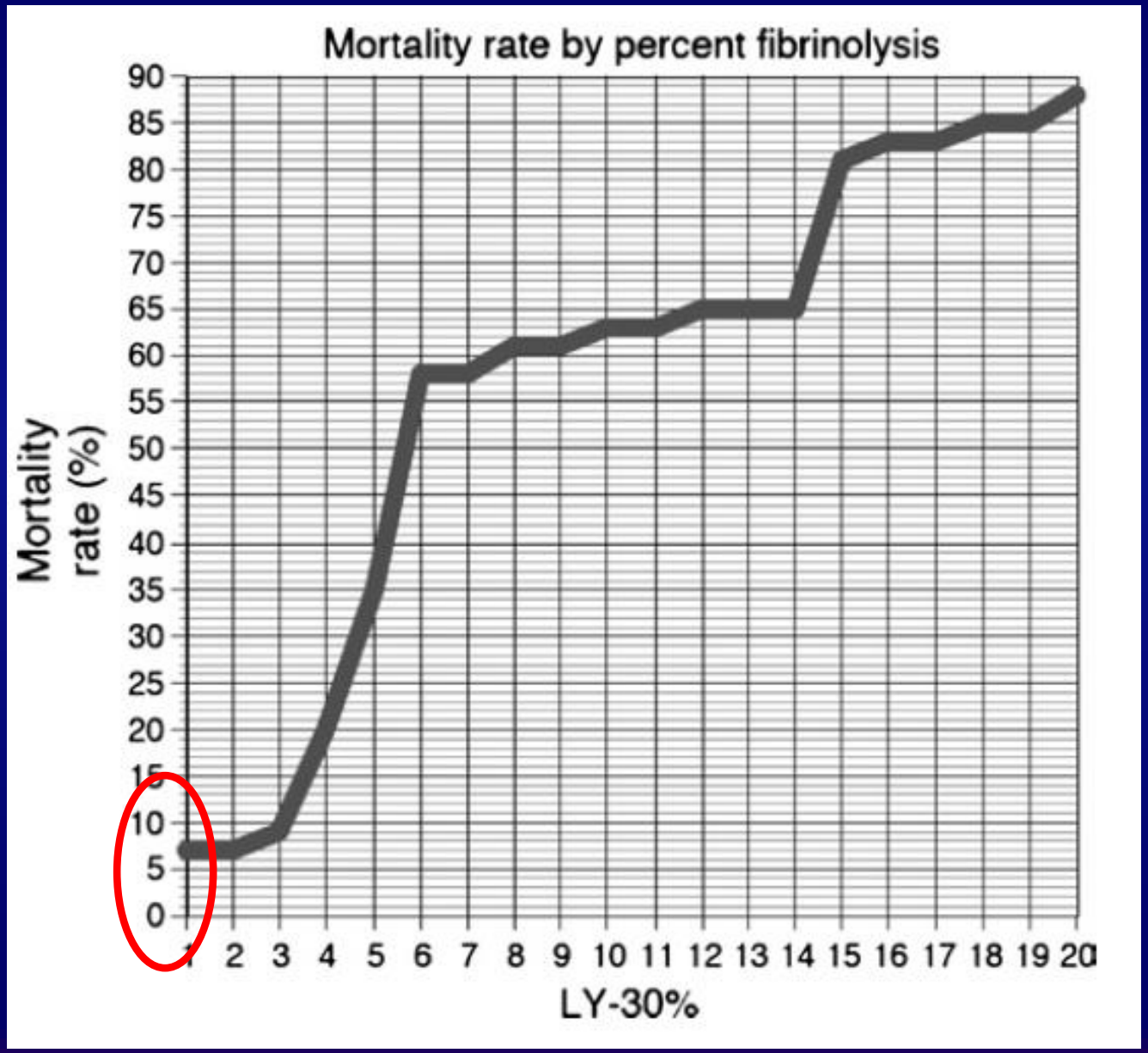
Fibrinolytic Abnormalities
LY30 Parameter: Primary Fibrinolysis





When fibrinolysis is greater than the rate of clot formation, or when it causes the breakdown of new clots, bleeding typically occurs. This condition is primary fibrinolysis and is identified with the TEG analyzer by an LY30 value of greater than 7.5% (or EPL > 15%), **combined with a CI value of less than or equal to 1.0**.

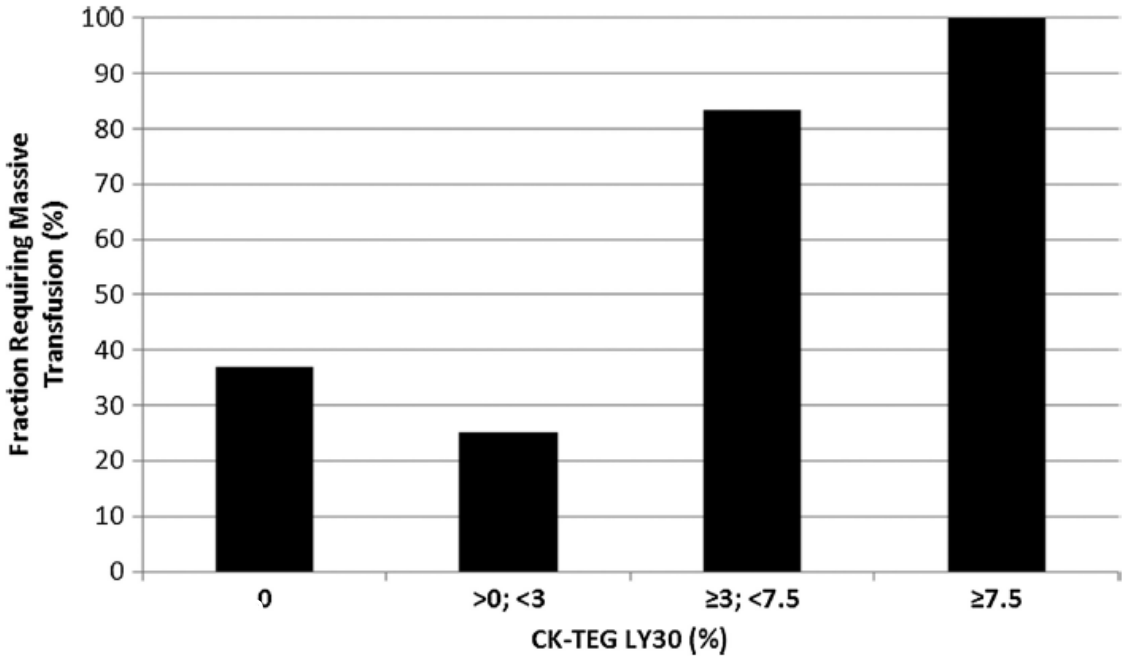
- Possible causes:
  - Excessive rate of fibrinolysis
- Possible etiologies:
  - High levels of tPA
- Common treatments:
  - Anti-fibrinolytic agent



Courtesy: Dr. Bryan Cotton

Fibrinolysis greater than 3% is the critical value for initiation of antifibrinolytic therapy

Michael P. Chapman, MD, Ernest E. Moore, MD, Christopher R. Ramos, MD, Arsen Ghasabyan, MPH, CCRC, Jeffrey N. Harr, MD, MPH, Theresa L. Chin, MD, John R. Stringham, MD, Angela Sauaia, MD, PhD, Christopher C. Silliman, MD, PhD, *and* **Anirban Banerjee, PhD,** *Aurora, Colorado*



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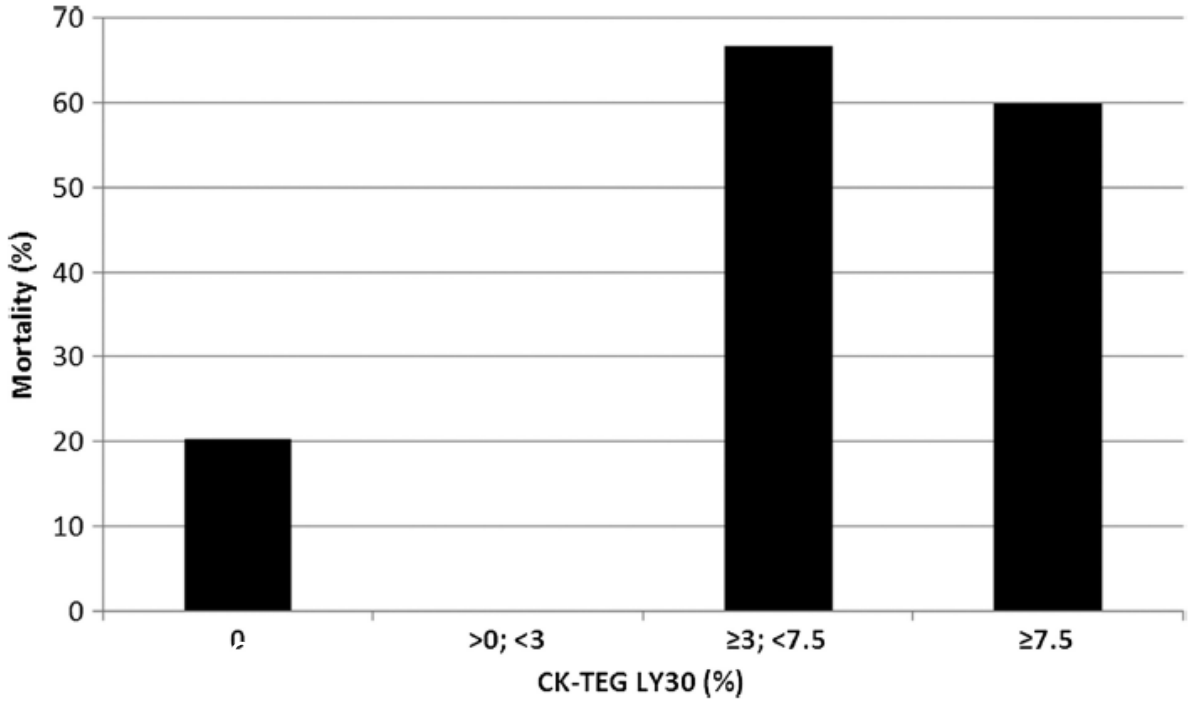


WHAT ABOUT RAPID TEG?

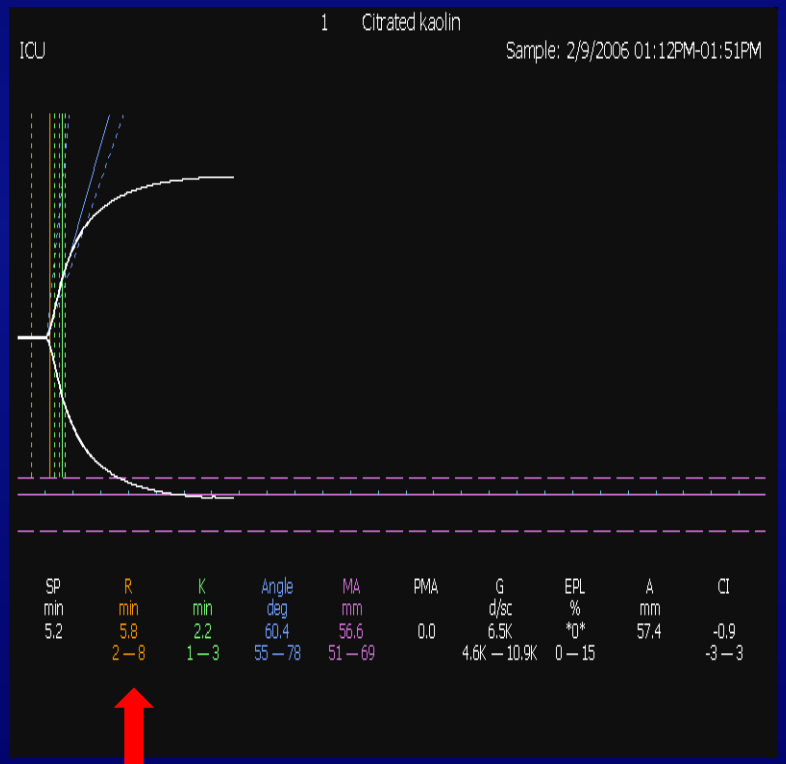
Rapid TEG

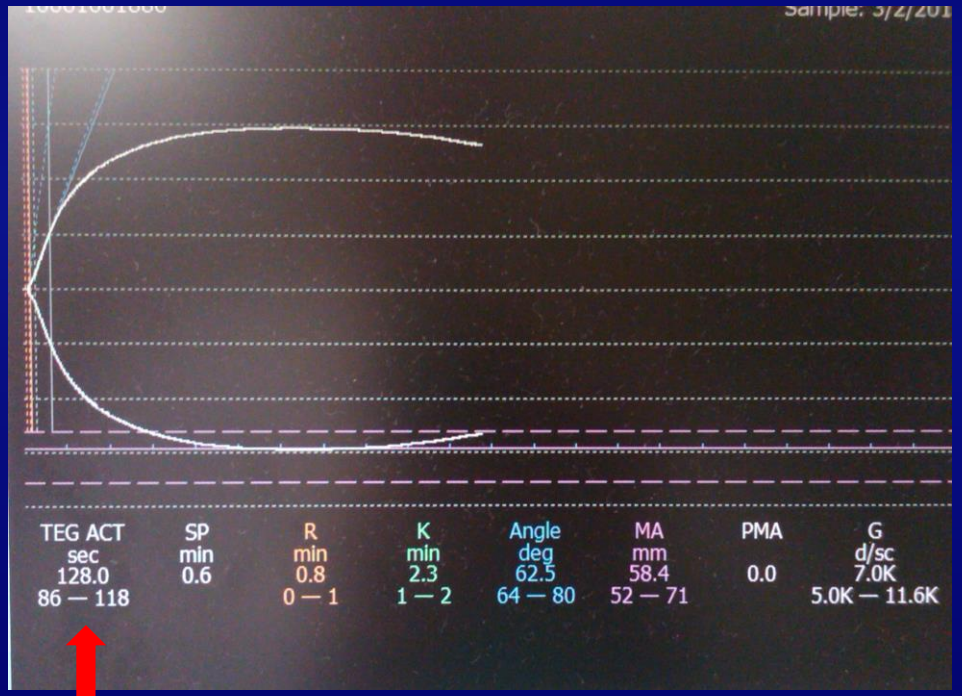
- Jeger and colleagues evaluated Rapid TEG
- r-TEG utilizes tissue factor in addition to kaolin for activation of the clotting cascade
- 20 trauma patients: r-TEG results available < 20 min. vs. > 30 min. for TEG, PT/PTT

  Jeger V et al J Trauma 2009

Courtesy: Dr. Bryan Cotton

Normal TEG vs **r-TEG**





Courtesy: Dr. Bryan Cotton

Rapid TEG predicting coagulopathy

TABLE 4. Multivariate Logistic Regression Model Predicting MT (≥10 Units PRBC) in the First 6 h

	Odds Ratio	95% CI	*p*
ACT >128 s	5.15	1.361–19.494	0.016
Age (yr)	1.00	0.974–1.043	0.636
Male gender	0.56	0.139–2.319	0.431
Blunt mechanism of injury	0.43	0.128–1.473	0.475
White race	0.65	0.353–0.1.233	0.191
ED systolic blood pressure	0.99	0.970–1.011	0.378
ED heart rate	0.98	0.968–1.017	0.548
Positive FAST examination	1.59	0.466–5.148	0.181

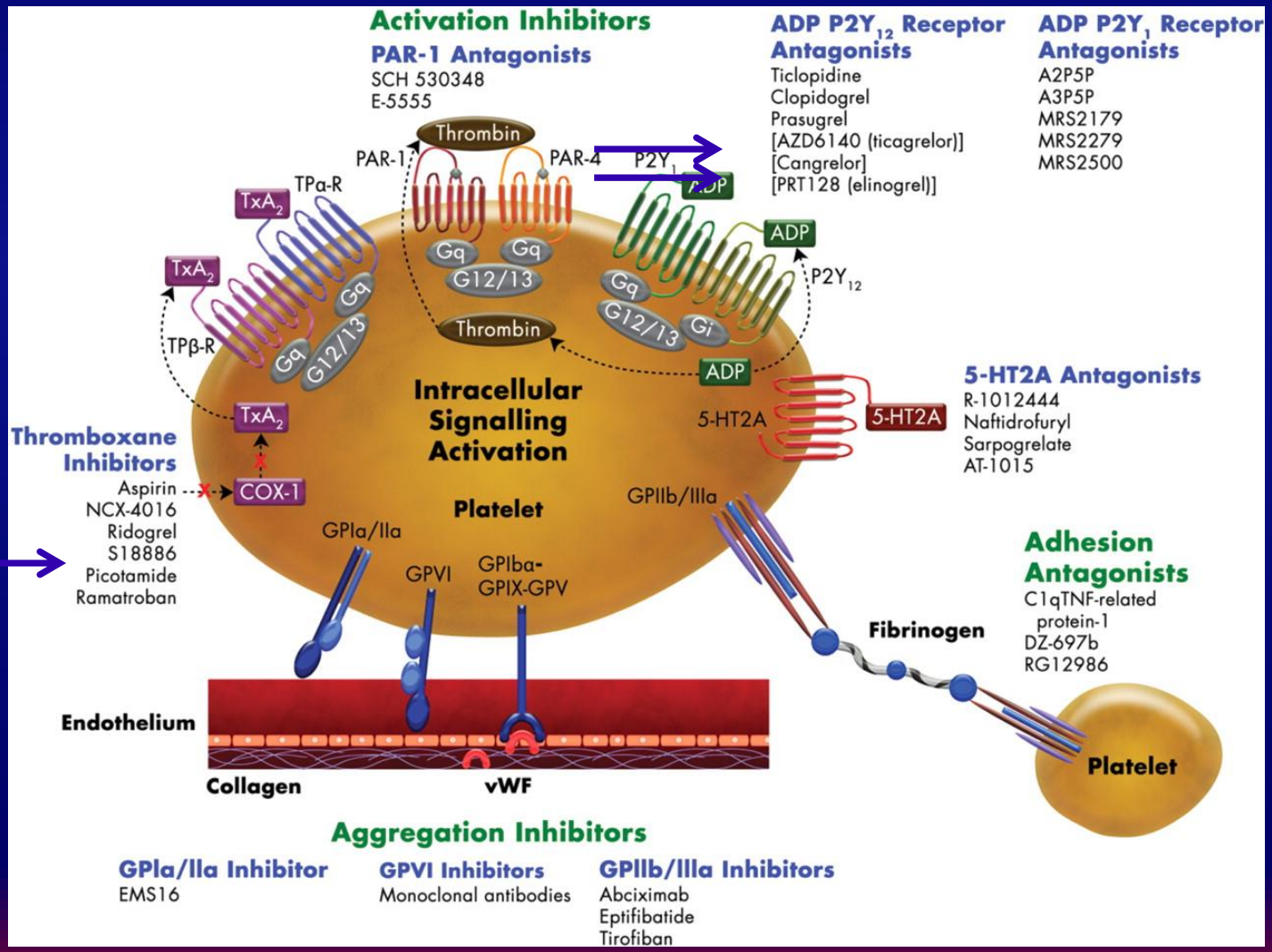
r-TEG predicting NO blood

TABLE 5. Multivariate Logistic Regression Model Predicting No PRBC Transfusions in the First 6 h

	Odds Ratio	95% CI	*p*
ACT <105 s	1.85	1.069–3.185	0.028
Age (yr)	0.99	0.978–1.007	0.340
Male gender	0.88	0.486–1.606	0.686
Blunt mechanism of injury	1.56	0.868–2.835	0.136
White race	0.60	0.355–1.037	0.068
ED systolic blood pressure	1.00	0.985–1.027	0.558
ED heart rate	0.98	0.976–0.998	0.020
Positive FAST examination	0.62	0.313–1.238	0.117

Courtesy: Dr. Bryan Cotton

Platelets


Activation Inhibitors
PAR-1 Antagonists
SCH 530348
E-5555
ADP P2Y12 Receptor Antagonists
Ticlopidine
Clopidogrel
Prasugrel
[AZD6140 (ticagrelor)]
[Cangrelor]
[PRT128 (elinogrel)]
ADP P2Y1 Receptor Antagonists
A2P5P
A3P5P
MRS2179
MRS2279
MRS2500
Thrombin
PAR-1
PAR-4
P2Y1
ADP
TPα-R
TxA2
Gq
G12/13
Gi
P2Y12
TPβ-R
Intracellular Signalling Activation
5-HT2A
5-HT2A Antagonists
R-1012444
Naftidrofuryl
Sarpogrelate
AT-1015
Thromboxane Inhibitors
Aspirin
NCX-4016
Ridogrel
S18886
Picotamide
Ramatroban
COX-1
Platelet
GPIIb/IIIa
GPIa/IIa
GPVI
GPIba-GPIX-GPV
Adhesion Antagonists
C1qTNF-related protein-1
DZ-697b
RG12986
Fibrinogen
Endothelium
Collagen
vWF
Platelet
Aggregation Inhibitors
GPIa/IIa Inhibitor
EMS16
GPVI Inhibitors
Monoclonal antibodies
GPIIb/IIIa Inhibitors
Abciximab
Eptifibatide
Tirofiban

Measuring Platelet Dysfunction

Device	Technique	Antiplatelet Medication Detection	Unit
PFA 100	Cessation of blood flow by occlusion of aperture through platelet plug	Aspirin GpIIb/IIIa antagonists	Closure Time
Multiplate	Altered electrical impedance through platelet aggregation	Aspirin Thienopyridines GpIIb/IIIa antagonists	Aggregation Unit
Verify Now	Altered light transmission through platelet aggregation	Aspirin Thienopyridines GpIIb/IIIa antagonists	Aspirin Reaction unit $P2Y_{12}$ reaction unit Platelet aggregation unit
TEG-PM	Platelet effects on clot strength	Aspirin Thienopyridines GpIIb/IIIa antagonists	% Platelet inhibition Measured levels of clot stregth MA_{ADP} and MA_{AA}

255

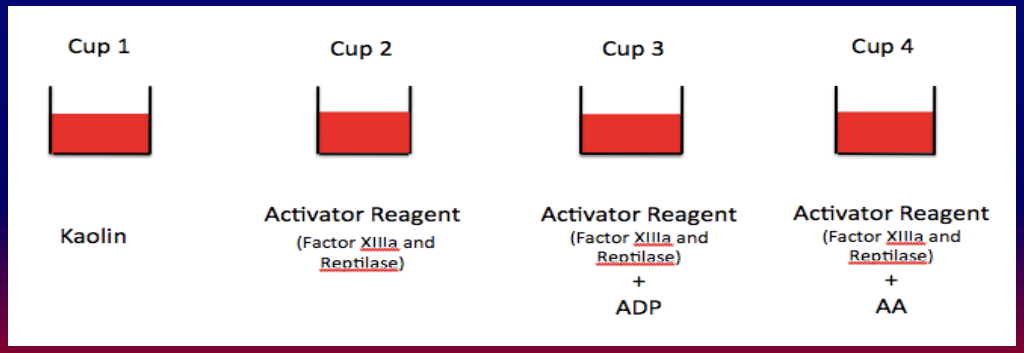
Point of care coagulation testing in neurosurgery

Christopher Beynon *, Andreas W. Unterberg, Oliver W. Sakowitz

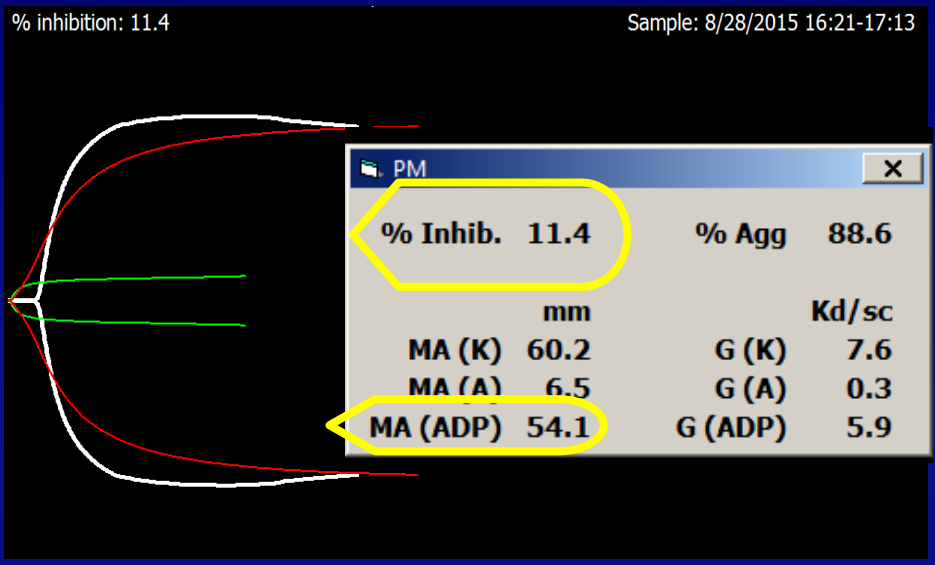
C. Beynon et al. / Journal of Clinical Neuroscience 22 (2015) 252–257

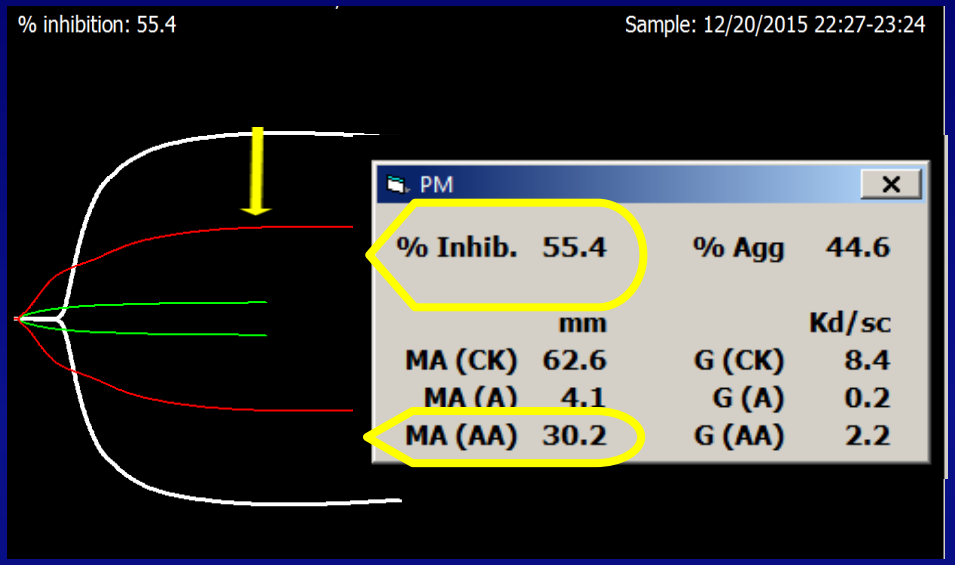
TEG with Platelet Mapping

- Platelet function is analyzed using the TEG/PM assay.
  - Four individual samples of 360 µL of whole blood are placed into separate specialized cups from blue-capped collection tubes. Next, 10 µL of the prepared activator solution, comprised of reptilase, factor XIIIa, and phospholipids are added to three of the cups
  - CUP 1: Kaolin (TEG Tracing – $MA_{Thrombin}$)
  - CUP 2: MA Fibrin (MA_{Fibrin})
  - Cup 3: MA_{ADP} (adenosine diphosphate) (MA_{ADP})
  - Cup 4: MA_{AA} (arachidonic acid) (MA_{AA})



Platelet Mapping Values





MA $_{ADP}$ or MA$_{AA}$ absolute values should be interpreted in correlation with the MA

- **MA $_{ADP}$** > 50 less likely to bleed
- **MA $_{ADP}$** 35-50 moderate chance of bleeding
- **MA $_{ADP}$** < 35 high chance of bleeding

Platelet Mapping: Tx Thresholds

MA $_{ADP\ or\ AA}$ > 50 less likely to bleed

MA $_{ADP\ or\ AA}$ 35-50 mod chance of bleeding

MA $_{ADP\ or\ AA}$ < 35 ↑ chance of bleeding

Critical Value < 35

% Inhibition Platelet Mapping Normal

ADP or AA % Inhibition 0-40%

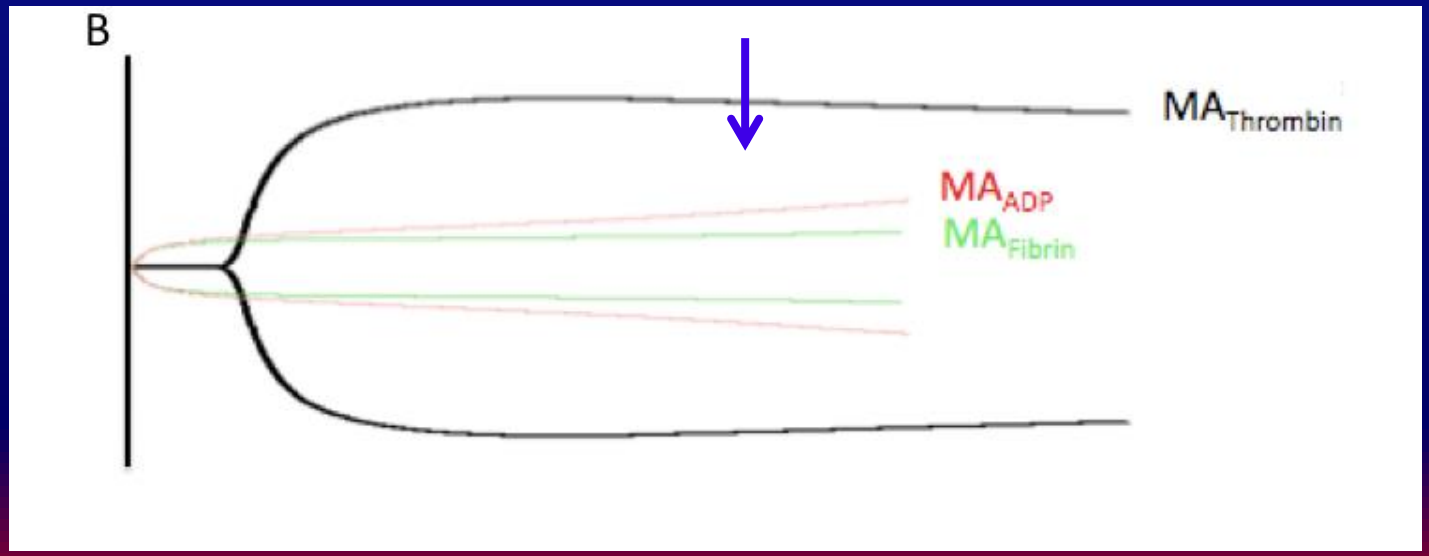
Critical Value > 60%

Inhibited Platelets

Platelets are more inhibited:

TEG/PM tracing with platelet inhibition greater than normal.

A patient can have a normal Platelet count but the platelets are not functioning properly



ABCs of TEG: Where to start?

- Become the Expert or Find a Nurse Colleague who wants to be content expert
  - Review/Read the literature on the use of TEG
  - Attend a lecture on how it is applied
  - Visit a center that has implemented a program
  - Become the expert!
- Find a Physician Champion
  - Get Physician BUY IN from the surgeons, intensivists, and Anesthesia
  - Find a champion from each area of expertise
    - Trauma, Neurosurgery, Neuro Critical Care, Intensivists, ED, Anesthesia
- Make friends with the Lab Manager or Perfusionist

ABCs of TEG: Where to start?

- Budget?
  - Donors are nice!
  - Build it into the budget cycle
- Once purchased: Form a MD team to build a hospital based protocol
  - Start with another hospital based protocol or develop one
  - Gain consensus: 2-3 meetings
- Educate staff
  - Physicians: Bring in physician content expert (national)
  - Nurses: Provide 1 – 1/2 hour lecture on TEG for key staff
- Get BUY IN from the rest of the hospital
  - Engage key nurses from ED/ICU, Laboratory personnel, OR personnel, and IT
- Provide 24/7 support

St. Joseph Health
Mission • Mission Laguna Beach

DEPARTMENT	Collaborative Practice
ORIGINAL DATE CREATED	12/2014
LAST DATE REVISED	9/28/15
ORIGINAL DATE ADOPTED	7/20/15
PROTOCOL APPROVED BY	Linda Johnson

Thromboelastography (TEG) Protocol for Monitoring/Treatment of Coagulation Alterations

I. Responsibility: Executive Director, Critical Care Services

II. Scope: Emergency Department Physicians, General Surgeons, Neurosurgeons, Cardiologists, Critical Care Physicians, Cardiovascular Surgeons, Nurses, Pharmacists, and Laboratory Personnel

III. Key Words: @TEG, @hemorrhage, @neurotrauma, @ life threatening Hemorrhage, @Fibrinolysis

IV. General Description:

Patients presenting to the Emergency Department with trauma may sustain massive bleeding. Trauma induced coagulopathy (TIC) is a complication of severe hemorrhage requiring blood products to reverse the coagulopathy. Patients presenting with severe traumatic brain injury are at risk for refractory intracranial hemorrhage from platelet dysfunction or the concurrent use of anticoagulants or anti-platelet medications. In addition, patients presenting with massive hemorrhage require targeted blood product administration to halt the hemorrhagic processes.

Thromboelastography (TEG, Haemonetics Corporation, Braintree, MA) testing provides a more predictive and accessible point-of-care (POC) measurement of clot formation and strength, platelet function, and fibrinolytic activity that the critical care team needs to guide effective hemostatic therapy for TIC as well as excessive hemorrhage.

IV. Purpose:

1. This protocol will define the assessment, diagnostic workup, and treatment choices to reverse the hemorrhagic state in patients with trauma, severe TBI, uncontrolled bleeding, and/or reversal of anti-coagulants/anti-platelet medications.
2. Define the parameters associated with TEG
3. Define the parameters associated with TEG with platelet mapping.
4. Provide treatment recommendations for abnormal TEG/TEG with Platelet Mapping results.

Table 1: **Algorithm for TEG-Guided Blood Component Therapy**

Note: Gray tracing overlapping diagram represents normal TEG tracing

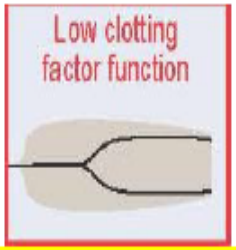
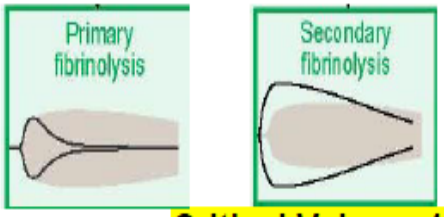
TEG Normal/Abnormal Value	Blood Component Therapy
Normal 5-10 min Prolonged R > 10 min  Critical Value >10 Minutes	**1) Prothrombin Complex Concentrate (PCC) Preferred as #1** 2) Fresh Frozen Plasma (FFP) alternative 3) Protamine **if Heparin** Present 4) Factor VIIa **if Jehovah's Witness**
Normal K 1-3 min **Normal α-angle 53-72 degrees** Prolonged K time and/or reduced α-angle (<53°)  **Critical Value < 53 degrees**	1) Cryoprecipitate and/or 2) FFP

Table 1: **Algorithm for TEG-Guided Blood Component Therapy**

Note: Gray tracing overlapping diagram represents normal TEG tracing

TEG Normal/Abnormal Values	**Blood Component Therapy**
Normal MA 50-70 mm Low MA (< 50 mm)  **Critical Value < 50 mm**	1) Platelets 2) Consider DDAVP if going to OR stat
Normal LY30% 0-8% Elevated LY 30% (≥ 8%)  **Critical Value > 15%**	1) **Primary Fibrinolysis** LY30% > 8% with CI of < 1: **TXA** 2) **Secondary Fibrinolysis** LY30% > 8% with CI of > 3 : **Consider Anticoagulation**

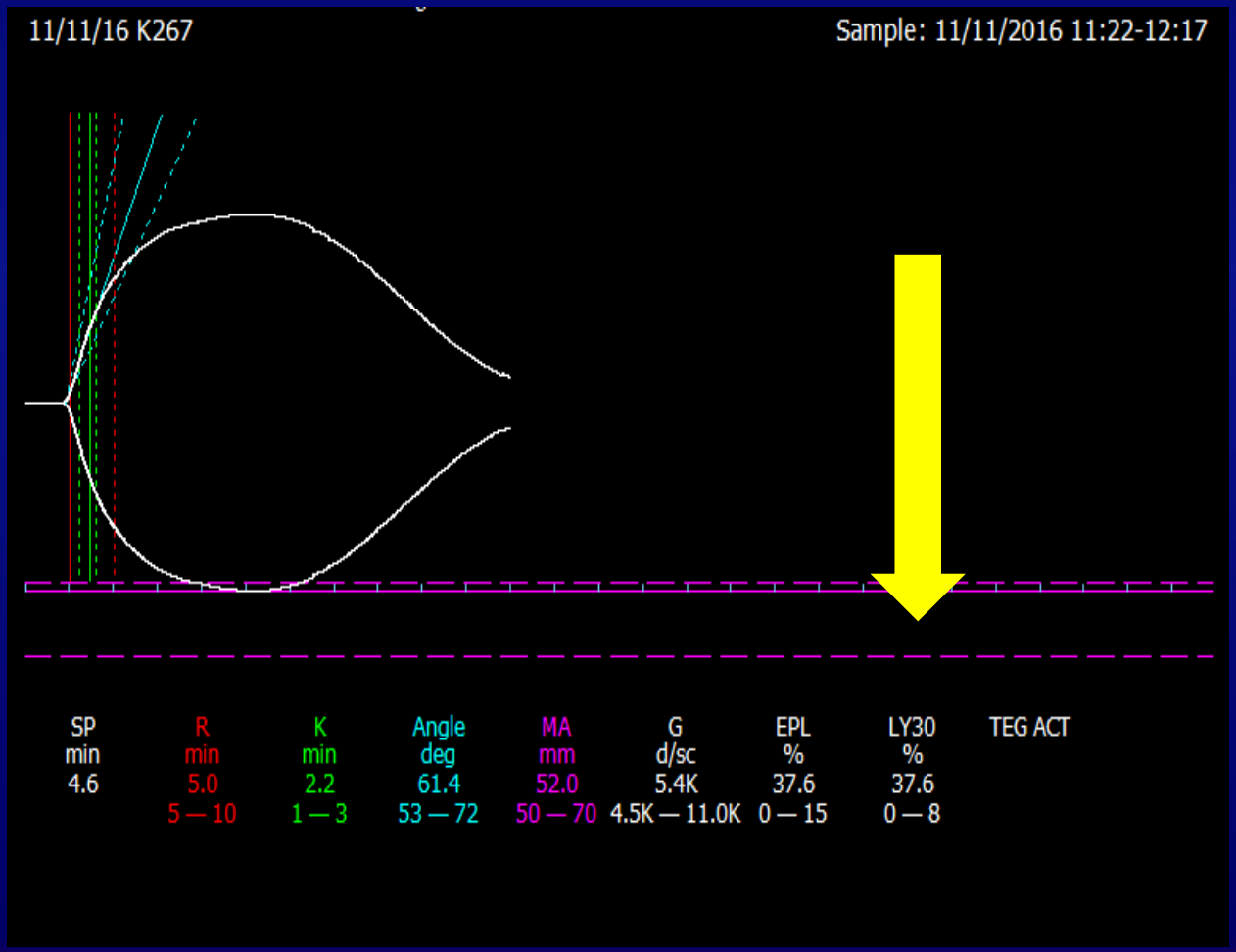
21 year old Male- Ped vs Train

- Red Trauma Alert…1035
  - GCS 1-4-2
  - VS HR 160 BP 92/50 R28
  - O2 saturation 60%
  - Hgb 11.9/Lactate 8.4
  - Diagnostics
    - Left chest pulmonary contusion, fx clavicle, scapulae, Left Rib fractures (1-3, 5, 7, 8-9) hemopneumothorax
    - Pelvic left side rami fractures/acetabular fx, Right sacral fx
    - Facial fractures: R maxillary sinus, right zygoma/nasal fx, orbital emphysema, fx anterior right orbital floor;
    - CT abdomen: active hemorrhage within the left flank, left gluteal region, near the left sacroiliac joint, near the left medial gluteal muscle, suspected mesenteric and retroperitoneal contusions, severe left hydronephrosis from uteropelvic junction obstruction, right adrenal contusion.

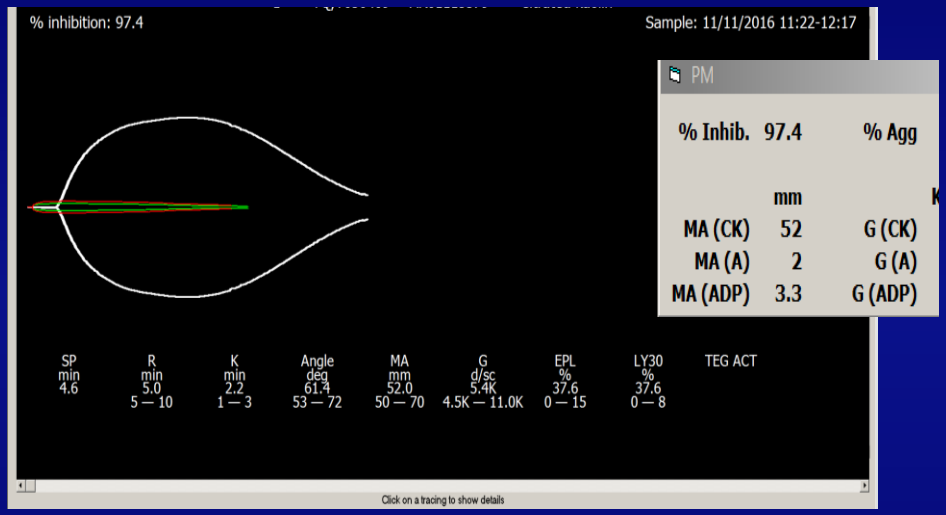
Other injuries

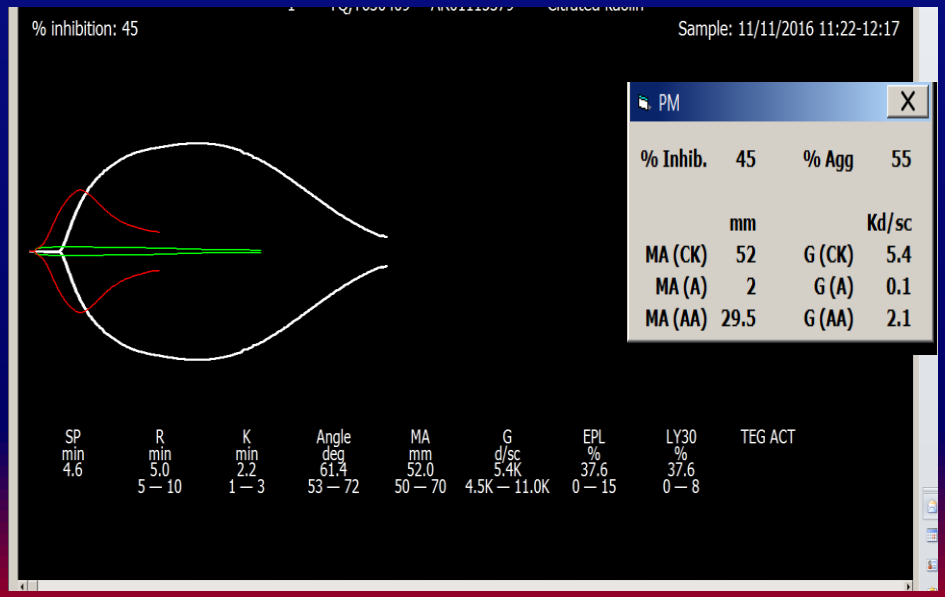
- CT brain: bilateral apical parasagital parenchymal hemorrhages, SDH, contusions, and cerebral edema
  - ICP opening pressure 30s
- Interventional OR: embolization of internal iliac artery

What is this?


11/11/16 K267
Sample: 11/11/2016 11:22-12:17
SP min 4.6
R min 5.0 5 — 10
K min 2.2 1 — 3
Angle deg 61.4 53 — 72
MA mm 52.0 50 — 70
G d/sc 5.4K 4.5K — 11.0K
EPL % 37.6 0 — 15
LY30 % 37.6 0 — 8
TEG ACT

Platelet Mapping





- TXA completed at 1500

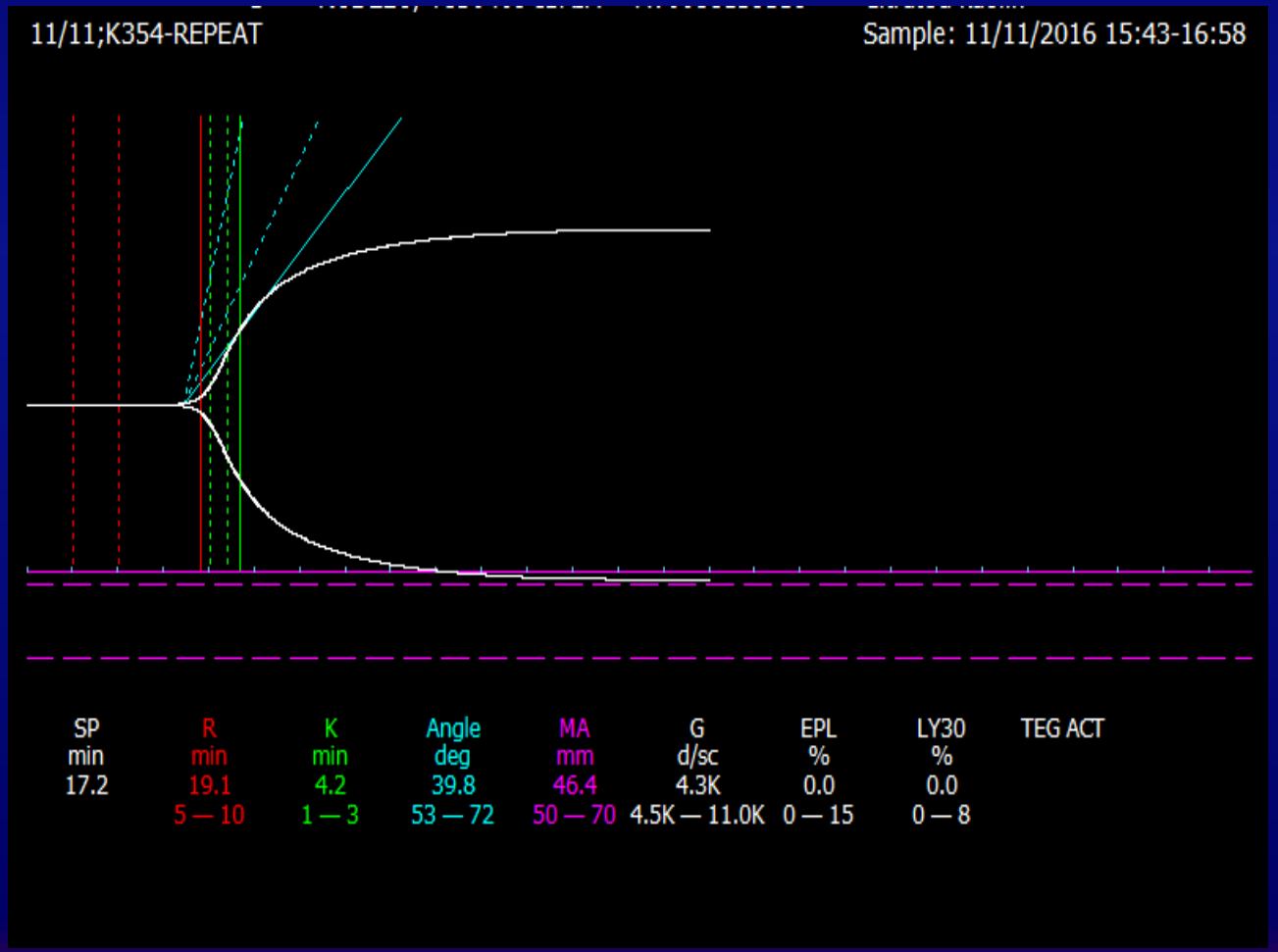
Massive Transfusion Protocol

Blood Product Summary | Blood Bank Tests | Transfusion History

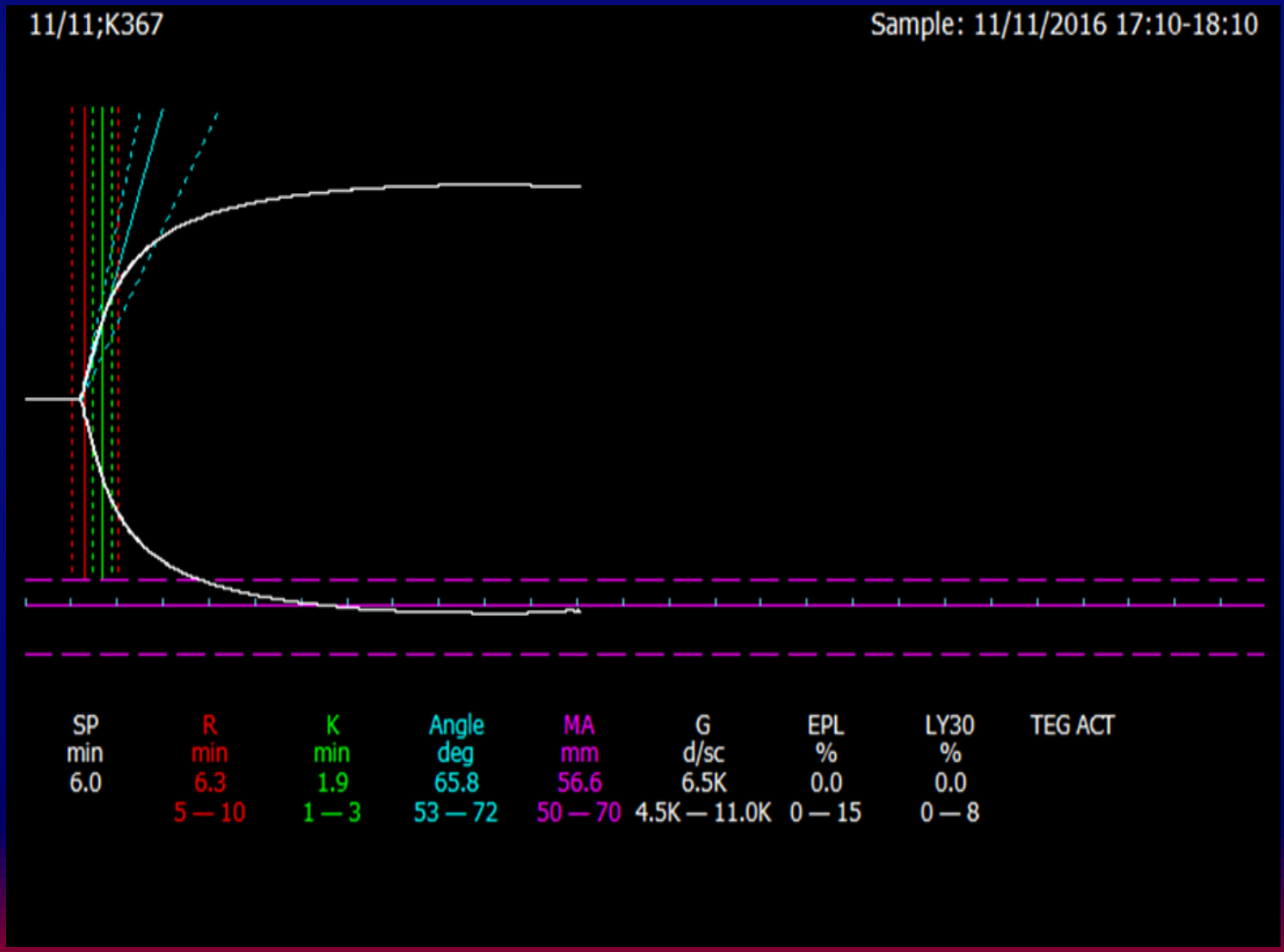
Date	Time	Product	Unit#	Volume	Blood Type	Reaction	Details
11/15/16	22:34	Leuk-reduced RBC	W333416021720	300	OP		
11/12/16	14:16	Leuk-reduced RBC	W333616086328	300	OP		
11/12/16	02:57	Leuk-reduced RBC	W044216566964	300	OP		
11/11/16	15:29	Leuk-reduced RBC	W044216576640	300	OP		
11/11/16	15:29	Leuk-reduced RBC	W044216575831	300	OP		
11/11/16	15:16	Thawed Plasma	W087916203071	304	ON		
11/11/16	15:16	Thawed Plasma	W044216578557	294	OP		
11/11/16	15:16	Pheresis Platelets	W333516020674	235	AN		
11/11/16	15:01	Thawed Plasma	W087916553834	311	OP		
11/11/16	15:00	Thawed Plasma	W087916553838	320	OP		
11/11/16	15:00	Thawed Plasma	W044216575767	339	OP		
11/11/16	15:00	Thawed Plasma	W037916163020	318	OP		
11/11/16	15:00	Pheresis Platelets	W044216584282	232	AP		
11/11/16	15:00	Pheresis Platelets	W044216584276	232	AP		
11/11/16	14:59	Thaw Pooled Cryoprecip	W149516813948	98	OP		
11/11/16	14:59	Thaw Pooled Cryoprecip	W149516813648	104	OP		
11/11/16	14:56	Thawed Plasma	W044216575799	317	OP		
11/11/16	14:56	Thawed Plasma	W044216564971	310	OP		
11/11/16	14:56	Leuk-reduced RBC	W044216579120	300	OP		
11/11/16	14:56	Leuk-reduced RBC	W044216577744	300	OP		
11/11/16	14:45	Leuk-reduced RBC	W044216581346	300	OP		
11/11/16	14:45	Leuk-reduced RBC	W044216579113	300	OP		
11/11/16	14:15	Pheresis Platelets	W051516109819	247	OP		
11/11/16	13:59	Leuk-reduced RBC	W044216577648	300	OP		
11/11/16	13:59	Leuk-reduced RBC	W044216575845	300	OP		
11/11/16	13:59	Leuk-reduced RBC	W044216575819	300	OP		
11/11/16	13:59	Leuk-reduced RBC	W044216574654	300	OP		
11/11/16	13:54	Thawed Plasma	W044216575704	315	OP		
11/11/16	13:54	Thawed Plasma	W044216571975	325	OP		
11/11/16	13:54	Thawed Plasma	W044216571455	333	OP		
11/11/16	13:54	Thawed Plasma	W044216571436	309	OP		
11/11/16	13:54	Thawed Plasma	W037916160722	212	ABP		
11/11/16	13:54	Thawed Pheresis Plasma	W044216584284D	224	ABP		
11/11/16	13:54	Thawed Pheresis Plasma	W044216584284B	249	ABP		
11/11/16	13:54	Thawed Pheresis Plasma	W044216566506B	228	ABP		
11/11/16	13:34	Thawed Plasma	W044216582821	314	OP		
11/11/16	13:34	Thawed Plasma	W044216580524	311	OP		
11/11/16	13:29	Leuk-reduced RBC	W044216578551	300	OP		
11/11/16	13:29	Leuk-reduced RBC	W044216576633	300	OP		
11/11/16	13:29	Irrad/Leuk-reduced RBC	W044216583073	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W333616082943	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W333416022537	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W333416021363	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W333416020632	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W044216578772	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W044216574561	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W044116120399	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W039516024052	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W039516024035	300	OP		
11/11/16	13:00	Leuk-reduced RBC	W039516019671	300	OP		

Post TXA

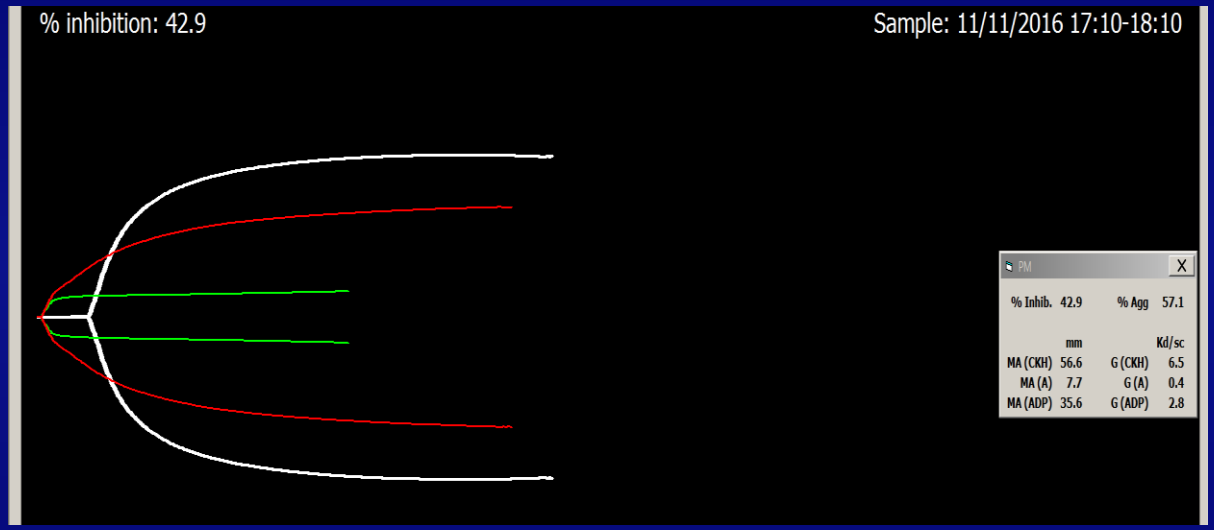
Given
2 units
FFP



Post op 1710


11/11;K367
Sample: 11/11/2016 17:10-18:10
SP min 6.0
R min 6.3 5 — 10
K min 1.9 1 — 3
Angle deg 65.8 53 — 72
MA mm 56.6 50 — 70
G d/sc 6.5K 4.5K — 11.0K
EPL % 0.0 0 — 15
LY30 % 0.0 0 — 8
TEG ACT

Post Op 1710


% inhibition: 42.9
Sample: 11/11/2016 17:10-18:10
PM
X
% Inhib. 42.9
% Agg 57.1
mm
Kd/sc
MA (CKH) 56.6
G (CKH) 6.5
MA (A) 7.7
G (A) 0.4
MA (ADP) 35.6
G (ADP) 2.8


% inhibition: 6.1
Sample: 11/11/2016 17:10-18:10
PM
X
% Inhib. 6.1
% Agg 93.9
mm
Kd/sc
MA (CKH) 56.6
G (CKH) 6.5
MA (A) 7.7
G (A) 0.4
MA (AA) 53.6
G (AA) 5.8

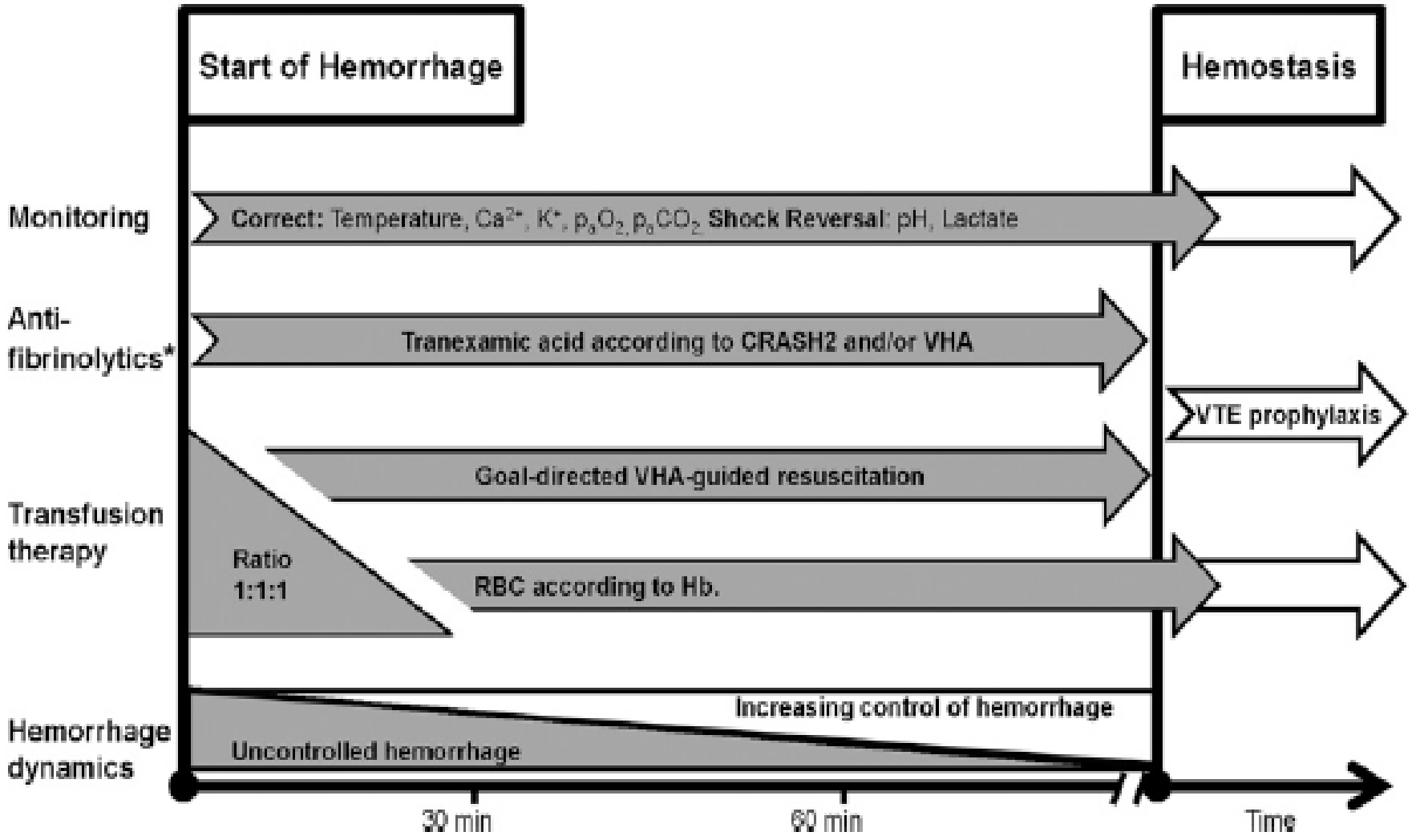
Discussion

Ratio-driven Resuscitation vs Goal-driven Resuscitation

- Use Point of care testing and patient vitals to guide initial care in major trauma
- Look at your patient (bleeding sources, vitals, etc)
- MTP and damage control surgery

- Using TEG in major trauma
  - "Mop up" once patient stabilized
  - Tailor the products to what the patient needs
  - Limitations (length of time to run test)

Conclusion


Start of Hemorrhage
Hemostasis
Monitoring
Correct: Temperature, Ca^{2+}, K^+, p_aO_2, p_aCO_2 Shock Reversal: pH, Lactate
Anti-fibrinolytics*
Tranexamic acid according to CRASH2 and/or VHA
VTE prophylaxis
Goal-directed VHA-guided resuscitation
Transfusion therapy
Ratio 1:1:1
RBC according to Hb.
Increasing control of hemorrhage
Hemorrhage dynamics
Uncontrolled hemorrhage
30 min
60 min
Time

End Points of Resuscitation

- The MTP should be discontinued when there is recognition that further resuscitation is futile.
- The following should be used as guides to cease therapy with blood and blood components in a patient who is (1) not actively bleeding and (2) still in the acute resuscitation phase:
- RBC transfusions for **hemoglobin** ≥10 g/dL
- Plasma transfusion for **prothrombin time (PT)** <18 seconds
- Plasma transfusion for activated **partial thromboplastin time (aPTT)** <35 seconds
- Platelet transfusions for **platelet count** >150 x10^9
- Cryoprecipitate or fibrinogen concentrate for **fibrinogen level** >180 g/L

If standard thrombelastography (TEG®) is available, the following cut-points for transfusion triggers may also be used:

- Plasma for **r-value** >9 minutes
- Plasma and/or cryoprecipitate (fibrinogen concentrate) for **k-time** >4 minutes
- Cryoprecipitate (or fibrinogen concentrate) and/or plasma for **α-angle** <60°
- Platelets for **mA** <55 mm
- Anti-fibrinolytics for **LY30** >7.5 percent