Using TEG in the ED, OR, and ICU

Don H. Van Boerum, MD, FACS

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Trauma ICU, Intermountain Medical Center, Intermountain Healthcare
Salt Lake City, Utah

Objectives:

- Discuss the basics of TEG interpretation
- Describe the role of thromboelastography in the acutely bleeding patient
- Review the concept of TEG-guided resuscitation
TEG - Thromboelastography

Don H. Van Boerum MD FACS
Trauma Surgery
Intermountain Medical Center
Disclosures

I am recommending the use of patient treatment plans, not specific individual products that may be used in patient treatment.

I will be discussing off-label uses of TEG technology.
Objectives

- Explain the Complexities in Traumatic Coagulopathy
- Describe Thromboelastography (TEG)
- Explain Interpretation of TEG Data
- Show How TEG Can be a Useful Tool in Treating Trauma & Other Bleeding Patients
Trauma

- Leading Cause Death 1-44
- 3rd Leading Cause Death All Ages
- Hemorrhagic Shock Cause of Almost 50% in Hospital Trauma Deaths World Wide
- Hemorrhage is Leading Cause of “Preventable” Trauma Related Deaths
- 25% Severely Injured Patients Arrive with Coagulopathy
Bloody Vicious Cycle

- Life-Threatening Trauma
- Blood Loss
- Progressive Systemic Coagulopathy
- Core Hypothermia
- Metabolic Acidosis
- FFP Resistant
- FFP Sensitive
- Acute Endogenous Coagulopathy
- Clotting Factor Deficiencies
- Massive RBC Transfusion
- Iatrogenic Factors
- Cellular Shock
- Tissue Injury
- Preexisting Diseases
Five Phases of Hemostasis

1. Vascular Phase: constriction of vessel to decrease blood flow.
2. Platelet Phase: platelets adhere to injured vessel to form a plug and seal defect.
3. Coagulation Phase: coagulation factors are activated, thrombin burst, fibrin strands combine with platelets to form "clot".
4. Clot Retraction: bleeding stopped; clot retracts, becomes more firm and brings torn edges together.
5. Fibrinolysis: final repair, clot is broken up, blood flows normally.
With blood flow in the body three things can happen ...

And two of them are not good!
Steady State – Pre Injury

Thrombosis

Fibrinolysis
Injury Occurs

Thrombosis  Fibrinolysis
Injury Occurs

Thrombosis  Fibrinolysis
Injury Occurs

- Thrombosis
- Fibrinolysis
Injury - Early

- Thrombosis
- Fibrinolysis
Injury

Thrombosis

Fibrinolysis
Injury - Late

- Thrombosis
- Fibrinolysis
Traumatic Coagulopathy

Clotting Factors
  Consumption
  Warfarin & Newer Poisons
Platelets
  Platelet Dysfunction
  Anti-Platelet Medications
Fibrinolysis
Inherited Coagulopathies
Combinations of the Above
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Do We Currently Manage?
How Does It All Work?

Need Formation of Clot
***But Not Too Much!

Three Interrelated Systems
The Enzymatic System (Factors)

- Clot (Plug)
  - Enzymatic System
    - Fibrinogen
    - Thrombin
    - Fibrin
The Platelet System

- Clot (Plug)
  - Enzymatic System
    - Thrombin
    - Fibrin
  - Platelet System
    - Fibrinogen
    - Activation / Aggregation
    - Adhesion
The Fibrinolytic System

Clot (Plug)

Enzymatic System
- Thrombin
- Fibrinogen

Platelet System
- Fibrin
- Activation / Aggregation
- Adhesion

Lysis (Breakdown)
- Physiologic
- Pathologic: Primary & Secondary
Extrinsic Pathway

F X

F VIIa

Ca²⁺

F VII

F III (Tissue Thromboplastin)

Ca²⁺

F X

Extrinsic Pathway

Tissue/Cell Defect
Extrinsic Pathway

Measured by Prothrombin Time PT

Extrinsic Pathway

- Tissue/Cell Defect
- F VIIa
- Ca²⁺
- F VII
- F III (Tissue Thromboplastin)
- Ca²⁺
- F X
- Ca²⁺
- F Xa

Measured by Prothrombin Time PT
Intrinsic Pathway

Surface Contact
Collagen
FXII activator

F XII \rightarrow F XIIa

F XI \rightarrow F Xla

F IX \rightarrow F IXa

F X \rightarrow F Xa

Ca^{2+}
Intrinsic Pathway

Intrinsic Pathway

Surface Contact
Collagen
FXII activator

F XII → F XIIa

Ca²⁺

F XI → F Xla

Ca²⁺

F IX → F IXa

Ca²⁺

F X → F Xa

Measured by Partial Thromboplastin Time PTT
Common Pathway

- Fibrinogen
- Fibrin monomers
- Fibrin polymers
- Prothrombin II
- Thrombin IIa
- Ca²⁺

Diagram showing the common pathway in blood clotting involving fibrinogen, fibrin monomers, fibrin polymers, prothrombin II, thrombin IIa, and calcium ions (Ca²⁺).
The Coagulation Cascade

**Intrinsic Pathway**
- Surface Contact Collagen FXII activator

- F XII → F XIIa
  - F XI → F Xla
    - Ca²⁺

- F IX → F IXa
  - Ca²⁺
  - F VIII
  - F VIIIa
    - Platelet Factor 3

- F X → F Xa
  - Ca²⁺

**Extrinsic Pathway**
- Tissue/Cell Defect

- F VII → F VIIa
  - Ca²⁺

- F III (Tissue Thromboplastin)

- F X → F Xa
  - Ca²⁺

- Thrombin IIa

- F Va

- F XIIIa

- F XIII

- F XIIIa → F XIII

- Fibrin polymers

- Fibrin monomers

- Fibrinogen

Yellow lines indicate positive feedback loops lost in isolated tests
Traditional Lab Testing

Clot (Plug)

- Enzymatic System
  - Thrombin
  - Fibrinogen
- Platelet System
  - Activation / Aggregation
  - Adhesion
- Lysis (Breakdown)
  - Physiologic
  - Pathologic: Primary & Secondary

Tests:

- PTT
- PT
- INR
- Fibrinogen
- Platelet Count
- Bleeding Time
- D dimer
- PFA
- P2Y12
TEG – What Is It?

Viscoelastic Measurement of Coagulation
“Total Clot” Beginning to Fibrinolysis

Not New Technology
World War II – Hartert 1948
Liver Transplant 1970’s
Cardiac Devices 1990’s
TEG
TEG® Technology
R = Platelet function
G = Clot Strength
EPL, LY30

SP = Split Point, time to first fibrin strands
R = Reaction time to end of thrombin burst
K = fibrin cross-linkage, fibrinogen function
Angle = fibrinogen function
MA = platelet function in mm
G = MA converted to Kdynes/cm²
EPL = Estimated Percent Lysis, clot breakdown
LY30 = Lysis 30 minutes after MA reached
Alphabet Soup

**SP Split Point:** Initial fibrin formation

**R Reaction Time:** Initial clot @ 2mm FACTORS, HEPARIN, WARFARIN, DILUTIONAL

**R-SP Delta:** Think THROMBIN

**Alpha Angle:** Rate clot formation FIBRINOGEN

**K Kinetics:** Rate @ 20mm FIBRINOGEN

**MA Maximum Amplitude:** Clot strength
   PLATELET contribution...number & function

**G:** MA converted to dynes Clot strength

**Net G:** Calculated comparing baseline and result with percent platelet inhibition

**EPL Estimated Percent Lysis:** Represents clot breakdown DIC & HYPERFIBRINOLYSIS
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EPL Estimated Percent Lysis: Represents clot breakdown DIC & HYPERFIBRINOLYSIS
Normal TEG® Tracing

Thrombin-generated MA

Sample: 11/21/2003 10:05AM-10:49AM

<table>
<thead>
<tr>
<th>TEG ACT</th>
<th>SP</th>
<th>R</th>
<th>K</th>
<th>Angle</th>
<th>MA</th>
<th>G</th>
<th>EPL</th>
<th>LY30</th>
<th>A</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>6.2</td>
<td>6.8</td>
<td>2.1</td>
<td>63.7</td>
<td>57.2</td>
<td>6.7K</td>
<td><em>1.4</em></td>
<td><em>0.4</em></td>
<td>57.0</td>
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<tr>
<td></td>
<td>5-10</td>
<td>1-3</td>
<td>53-72</td>
<td>50-70</td>
<td>4.5K-11.0K</td>
<td>0-15</td>
<td>0-8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Hemodilution/Anticoagulants

#### TEG ACT

<table>
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<tr>
<th>SP min</th>
<th>R min</th>
<th>K min</th>
<th>Angle deg</th>
<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.7</td>
<td>10.5</td>
<td>2.2</td>
<td>53 — 72</td>
<td>50 — 70</td>
<td>4.5K — 11.0K</td>
<td>0 — 15</td>
<td>0 — 8</td>
<td>58.2</td>
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Heparin/LMWH

Citrated kaolin

Sample: 6/24/2008 04:56AM-06:00AM

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<th>Angle deg</th>
<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A mm</th>
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<td>12.5</td>
<td>2.5</td>
<td>59.6</td>
<td>69.1</td>
<td>11.2K</td>
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<td>0.0</td>
<td>70.3</td>
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</table>

Citrated Kaolin w/heparinase

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<thead>
<tr>
<th>min</th>
<th>min</th>
<th>min</th>
<th>deg</th>
<th>mm</th>
<th>d/sc</th>
<th>%</th>
<th>%</th>
<th>mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.0</td>
<td>8.8</td>
<td>1.5</td>
<td>69.6</td>
<td>72.1</td>
<td>12.9K</td>
<td>2.2</td>
<td>2.2</td>
<td>67.6</td>
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Factor Deficiency or Non-heparin Anticoagulants (no reversal with heparinase)
Fibrinogen Deficiency/Dysfunction

Sample: 4/26/2001 11:34AM-12:46PM

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<th>K min</th>
<th>Angle deg</th>
<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6.8</td>
<td>8.1</td>
<td>4.8</td>
<td>39.5</td>
<td>56.0</td>
<td>6.4K</td>
<td><em>0</em></td>
<td><em>0</em></td>
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## Decreased Platelet Function

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<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A mm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4.8</td>
<td>5.2</td>
<td>2.4</td>
<td>61.4</td>
<td>45.9</td>
<td>4.2K</td>
<td><em>0.5</em></td>
<td>0.0</td>
<td>45.3</td>
</tr>
<tr>
<td></td>
<td>5 — 10</td>
<td>1 — 3</td>
<td>53 — 72</td>
<td>50 — 70</td>
<td>4.5K — 11.0K</td>
<td>0 — 15</td>
<td>0 — 8</td>
<td></td>
<td></td>
</tr>
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</table>

Sample: 1/31/2005 10:55AM-11:39AM
Enzymatic (Thrombin-Driven) Hypercoagulability
Platelet-Driven Hypercoagulability

Platelet Mapping

Sample: 5/22/2008 04:28PM-05:51PM

TEG ACT
SP min 7.2
R min 7.7
K min 1.0
Angle deg 77.6
MA mm 82.5
G d/sc 23.5K
EPL % 0.0
LY30 % 0.0
A mm 79.5

10 millimeters
Combination Hypercoagulability

Sample: 12/14/2004 12:06PM-01:24PM

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<th>Value</th>
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<tr>
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<td>4.0</td>
</tr>
<tr>
<td>R min</td>
<td>4.4</td>
</tr>
<tr>
<td>K min</td>
<td>0.8</td>
</tr>
<tr>
<td>Angle deg</td>
<td>76.7</td>
</tr>
<tr>
<td>MA mm</td>
<td>76.1</td>
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<tr>
<td>G d/sc</td>
<td>15.9K</td>
</tr>
<tr>
<td>EPL %</td>
<td>3.1</td>
</tr>
<tr>
<td>LY30 %</td>
<td>3.1</td>
</tr>
<tr>
<td>A mm</td>
<td>65.4</td>
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Secondary Fibrinolysis, Stage I DIC

Sample: 3/19/2001 12:10PM-01:18PM

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<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3.0</td>
<td>3.4</td>
<td>1.0</td>
<td>79.0</td>
<td>82.5</td>
<td>23.6K</td>
<td>12.5</td>
<td>12.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 - 9</td>
<td></td>
<td>1 - 3</td>
<td>59 - 74</td>
<td>55 - 74</td>
<td>5.3K - 13.2K</td>
<td>0 - 15</td>
<td>0 - 8</td>
<td></td>
</tr>
</tbody>
</table>
Secondary Fibrinolysis, Stage II
DIC

(factor deficiency)

Sample: 7/7/1998 07:42AM-09:51AM

<table>
<thead>
<tr>
<th>TEG ACT</th>
<th>SP</th>
<th>R</th>
<th>K</th>
<th>Angle</th>
<th>MA</th>
<th>G</th>
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</tr>
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<tbody>
<tr>
<td></td>
<td>min</td>
<td>min</td>
<td>min</td>
<td>deg</td>
<td>mm</td>
<td>d/sc</td>
<td></td>
<td>%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>16.5</td>
<td>20.0</td>
<td>13.2</td>
<td>16.5</td>
<td>38.0</td>
<td>3.1K</td>
<td>0.5*</td>
<td></td>
<td>0—8</td>
</tr>
</tbody>
</table>

10 millimeters
Primary Fibrinolysis - TEG® Tracing

Sample: 12/8/2000 10:03PM-10:50PM

(Primary fibrinolysis)
Pattern Recognition

- Normal
- Anticoagulants/hemophilia
- Platelet Blockers
- Fibrinolysis
- Hypercoagulation
- D.I.C
  - Stage 1
  - Stage 2
Normal TEG® Analysis

**INCLUDES:**
- Heparin
- LMMH
- Coumadin
- Arixtra®
- Pradaxa®
- Xarelto®
- Factor Deficiency
- Fibrinogen Deficiency
- Platelet Function
- Clot Strength
- Lysis
- Surgical Bleeding from Hemostasis
- Hypercoagulability

**EXCLUDES:**
- Plavix®
- Effient®
- Brillinta ®
- Integrilin ®
- Reopro ®
- Aggrastat®
- Pletal®
- Persantine®
- NSAIDs
- ASA
- Pathologic Platelet Inhibition
PlateletMapping® Assay

Monitoring Platelet Inhibition and Baseline Platelet Function
Why PlateletMapping?
Personalized Platelet Therapy

Patient A: 50% platelet inhibition does not provide sufficient reduction of the risk of a thrombotic or ischemic event.

Patient B: 50% platelet inhibition provides antithrombotic protection without risk of bleeding.

Patient C: 50% platelet inhibition increases risk of bleeding.
Meds Requiring Platelet Mapping™

Platelet Inhibition

- GPIIb/IIIa
  - Reopro
  - Integrilin
  - Aggrastat

- ADP
  - Plavix
  - Persantine
  - Effient
  - Ticlid
  - Brilinta

- GPIb
  - ASA

-ASA

Integrilin

Effient

Ticlid

Brilinta
Before Plavix

% inhibition: 15.2

ADP / GPIIB IIIA Platelet Receptor, Functionally
Green Top + fXIII + ADP

% Inhib. 15.2 % Agg. 84.8

mm
MA (CK) 77.5 G (CK) 17.2
MA (A) 18.0 G (A) 1.1
MA (ADP) 68.5 G (ADP) 10.9
After Plavix

% inhibition: 93.5

<table>
<thead>
<tr>
<th></th>
<th>% Inhib</th>
<th>% Agg.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citrated kaolin</td>
<td>93.5</td>
<td>6.5</td>
</tr>
</tbody>
</table>

ADP / GPIIB IIIA Platelet Reception, Functionally

Green Top + fXIII + ADP

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<th>Kd/sc</th>
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<tbody>
<tr>
<td>MA (CK)</td>
<td>78.0</td>
<td>17.7</td>
</tr>
<tr>
<td>MA (A)</td>
<td>18.1</td>
<td>1.2</td>
</tr>
<tr>
<td>MA (ADP)</td>
<td>22.0</td>
<td>1.4</td>
</tr>
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Plavix, Effient, Ticlid, Brilinta, Integrilin, Aggrastat, Reopro, Persantine, Toradol, Pletal, Pathologic inhibition

% Inhibition: 93.5

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<th>Clot strength uninhibited (Can also use CKH, K, KH sample types)</th>
</tr>
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<tbody>
<tr>
<td>ADP</td>
<td>Clot strength with inhibitor effect such as Plavix®, etc. (Can also use AA sample type for inhibitor effect such as aspirin, etc.)</td>
</tr>
<tr>
<td>A</td>
<td>Fibrin effect on clot strength</td>
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</tbody>
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<th>Kd/sc</th>
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<td>G (CK)</td>
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</tr>
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</tr>
</tbody>
</table>
Platelet Mapping Assay
TEG Analysis: Aspirin

% Inhib: 97.7
% Agg.: 2.3

MA (CK): 52.6
G (CK): 5.5
MA (A): 8.7
G (A): 0.4
MA (ADP): 9.7
G (ADP): 0.5
Pathologic Platelet Inhibition

Not drug-induced
May be due to foods or neutraceuticals
May be underlying pathology
May impact platelet activation or aggregation
It is real, and should be considered for risk
Platelet Mapping™ Assay

**HEMOSTASIS**
- Hypercoagulability
- Fibrinolysis
- Surgical Bleeding from Hemostasis
- Factor Deficiency
- Fibrinogen Function
- Platelet Function

**PLATELET MAPPING™ INTERVENTION**

- Shows percent platelet inhibition due to:
  - Interventional Treatment
  - Pathologic Inhibition

**INCLUDES:**

**DRUGS:**
- Heparin
- LMWH
- Coumadin
- Arixtra®
- Pradaxa®
- Xarelto®
- Plavix®
- Effient®
- Ticlid®
- Brilinta®
- Reopro®
- Integrilin®
- Aggrastat®
- ASA
- NSAIDs
Objectives of TEG-Guided Therapy

To express function and pinpoint dysfunction in the hemostasis system

• Reference the appropriate types and amounts of blood products needed to correct bleeding from this dysfunction

• Allow accurate anticoagulation or antiplatelet interventions to reduce thrombotic complications without inappropriate bleeding
Objectives of TEG-Guided Therapy

- To distinguish between anatomical and coagulopathic bleeding
- To distinguish primary from secondary fibrinolysis, including the consumptive phase
- To reduce the use of unnecessary blood products and reduce thrombotic complications
Stab Wound
Aorta, SMA, Liver, Pancreas

<table>
<thead>
<tr>
<th>TEG ACT</th>
<th>SP min</th>
<th>R min</th>
<th>K min</th>
<th>Angle deg</th>
<th>MA mm</th>
<th>G d/sc</th>
<th>EPL %</th>
<th>LY30 %</th>
<th>A mm</th>
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<td>7.2</td>
<td>8.0</td>
<td>3.4</td>
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<td>2.3K</td>
<td>76.7</td>
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<td>0.2</td>
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</table>
Post Op One Hour
Post Op Day 1
Massive Resuscitation

5 Liter Bloodloss
30 PRBC 30 FFP 5 Platelets (8Pk equiv)

Normalized TEG by End of Case

Hypercoagulable in Less Than 24 hours!
Limitations of TEG

• Von Willebrand’s Disease (endothelial adhesion)
• Lupus Anticoagulant
• Protein C & S deficiency
• Factor V Leiden
• HIT
Conclusions

• Traditional Coagulation Tests Alone are Inadequate
• TEG Measures Global Clot Formation
• TEG Identifies the Contribution of Platelets and Fibrinolysis to Hemostasis
• TEG Allows a Targeted Guide to Blood Component Therapy – Reducing Overtransfusion
• TEG May Also be Useful in Guiding VTE Prophylaxis
More Conclusions

• Platelet Mapping Accounts for Platelet Dysfunction from Drugs & Injury
• Early Recognition of Fibrinolysis May Improve Outcomes with Treatment (TXA)
• TEG Guided Therapy will Continue to Evolve with More Studies