A small proportion (4%) need an aggressive approach to transfusion.

Systems that include fibrinogen substrates seem to improve outcome.

The challenge is to develop systems that deliver fibrinogen rapidly enough to these patients.

TEG and ROTEM then can help us direct fibrinogen and drugs to the patients that need it.

Unless we include POC monitoring of coagulopathy we risk replacing exsanguination with thrombosis.
No Transfusion → Focused Tx → DCR

- 10 units RBC in 4 hrs
- 10 units RBC in 24 hrs
Table 1. Investigations to be performed during massive transfusions

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Target value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin; haematocrit</td>
<td>10 g/dl; 0.32</td>
</tr>
<tr>
<td>Platelet count</td>
<td>&gt; 50 × 10⁹/l</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>&lt; 1.5 × control</td>
</tr>
<tr>
<td>Partial thromboplastin time</td>
<td>&lt; 1.5 × control</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>&gt; 0.8 g/l</td>
</tr>
</tbody>
</table>

BSCH guidelines for massive transfusion 1998
Thromboelastography

- measures viscoelastic properties
- incorporates input from clotting, platelets and fibrinolysis
- dynamic
- rapid results
APPARATUS
Point-of-care testing Measurement of coagulation

J. Hirsch, T. Wendt, P. Kuhly and W. Schaffartzik

Can RapidTEG Accelerate the Search for Coagulopathies in the Patient With Multiple Injuries?

Victor Jeger, MS, Heinz Zimmermann, MD, and Aristomenis E. Eustathopoulos, MD

Hypothetical: Early recognition of coagulopathy may improve the care of patients with multiple injuries. Rapid thromboelastography (Rapid-TEG) is a real time variant of thromboelastography (TEG), in which coagulation is initiated by the addition of protein factor. The time of coagulation and the time of measurement were compared for two variants of TEG—Rapid-TEG and conventional TEG, in which coagulation was initiated with kaolin. The measurements were performed on blood samples from 20 patients with multiple injuries. The Rapid-TEG results were also compared with conventional measurements of blood coagulation. The mean time for the Rapid-TEG test was 9.2 ± 3.1 minutes (mean ± SD), in comparison with 20.0 ± 4.3 minutes for kaolin TEG and 34.1 ± 18.5 minutes for conventional coagulation tests. The mean time for the Rapid-TEG test was 9.2 ± 3.1 minutes, in comparison with 41.5 ± 3.6 minutes for kaolin TEG and 64.9 ± 18.3 for conventional coagulation tests—measured from admission of the patient to the resuscitation bay until the results were available. There were significant correlations between the Rapid-TEG results and those from kaolin TEG and conventional coagulation tests. Rapid-TEG is the most rapid available test for providing reliable information on coagulopathy in patients with multiple injuries. This has implications for improving patient care.

Key Words: Thromboelastography, Traumatic coagulopathy, Multiple injuries, tissue factor.

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HEAD-TO-HEAD
The TEG® vs the ROTEM® thromboelastometry systems

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Chelsea and Westminster Hospital, London, UK

Does Thromboelastography Predict Postoperative Thromboembolic Events? A Systematic Review of the Literature

Yue Dai, MB, MSc*
Anna Lee, PhD*
Lester A. H. Critchley, MD*
Paul F. White, PhD, MD*

BACKGROUND: Since thromboelastography (TEG) can detect hypercoagulable states, it is a potentially useful test for predicting postoperative thromboembolic complications. Therefore, we performed a systematic review of the literature to evaluate the accuracy of TEG in predicting postoperative thromboembolic events.

METHODS: PubMed and EMBASE electronic databases were searched by two independent investigators to identify prospective studies involving adult patients undergoing operative procedures in which a TEG test was performed preoperatively and outcomes were measured by reference standards. The quality of included studies was assessed and measures of diagnostic test accuracy were...
Does TEG *predict* these coagulation changes in haemorrhagic and shocked trauma patients?
Table 6: Prediction of transfusion by CCT, Rapid TEG*, and Kaolin TEG*.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Cut-offs</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>AUC</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>&gt;1.2</td>
<td>38%</td>
<td>88%</td>
<td>57%</td>
<td>77%</td>
<td>73%</td>
</tr>
<tr>
<td>INR</td>
<td>&gt;1.5</td>
<td>19%</td>
<td>96%</td>
<td>67%</td>
<td>74%</td>
<td>73%</td>
</tr>
<tr>
<td>aPTT (sec)</td>
<td>&gt;60.0</td>
<td>5%</td>
<td>98%</td>
<td>50%</td>
<td>69%</td>
<td>74%</td>
</tr>
<tr>
<td>Fibrinogen (g/L)</td>
<td>&lt;3.0</td>
<td>90%</td>
<td>48%</td>
<td>43%</td>
<td>92%</td>
<td>74%</td>
</tr>
<tr>
<td>Thrombin time [sec]</td>
<td>&gt;13.2</td>
<td>48%</td>
<td>73%</td>
<td>45%</td>
<td>75%</td>
<td>53%</td>
</tr>
<tr>
<td>Rapid K (min)</td>
<td>&gt;1.8</td>
<td>68%</td>
<td>78%</td>
<td>61%</td>
<td>83%</td>
<td>79%</td>
</tr>
<tr>
<td>Kaolin K (min)</td>
<td>&gt;1.7</td>
<td>68%</td>
<td>59%</td>
<td>46%</td>
<td>78%</td>
<td>67%</td>
</tr>
<tr>
<td>Rapid α-Angle (deg)</td>
<td>&lt;74.7</td>
<td>84%</td>
<td>57%</td>
<td>49%</td>
<td>88%</td>
<td>77%</td>
</tr>
<tr>
<td>Kaolin α-Angle (deg)</td>
<td>&lt;58.5</td>
<td>72%</td>
<td>61%</td>
<td>47%</td>
<td>82%</td>
<td>66%</td>
</tr>
<tr>
<td>Rapid MA (mm)</td>
<td>&lt;59.6</td>
<td>68%</td>
<td>80%</td>
<td>63%</td>
<td>83%</td>
<td>75%</td>
</tr>
<tr>
<td>Kaolin MA (mm)</td>
<td>&lt;58.4</td>
<td>56%</td>
<td>88%</td>
<td>70%</td>
<td>80%</td>
<td>70%</td>
</tr>
<tr>
<td>Rapid TMA (min)</td>
<td>&gt;17.3</td>
<td>76%</td>
<td>57%</td>
<td>46%</td>
<td>83%</td>
<td>69%</td>
</tr>
<tr>
<td>Kaolin TMA (min)</td>
<td>&gt;24.7</td>
<td>64%</td>
<td>63%</td>
<td>46%</td>
<td>78%</td>
<td>58%</td>
</tr>
<tr>
<td>Rapid G (d/sc)</td>
<td>&lt;7374</td>
<td>68%</td>
<td>78%</td>
<td>61%</td>
<td>83%</td>
<td>73%</td>
</tr>
<tr>
<td>Kaolin G (d/sc)</td>
<td>&lt;7073</td>
<td>56%</td>
<td>88%</td>
<td>70%</td>
<td>80%</td>
<td>70%</td>
</tr>
</tbody>
</table>

Combined indicators:

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Cut-offs</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>AUC</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Angle + Heart Rate</td>
<td>Rapid α-Angle (deg)</td>
<td>&lt;75</td>
<td>84%</td>
<td>75%</td>
<td>62%</td>
<td>90%</td>
</tr>
<tr>
<td></td>
<td>Heart Rate (bpm)</td>
<td>&gt;75</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>α-Angle + Hct</td>
<td>Rapid α-Angle (deg)</td>
<td>&lt;75</td>
<td>88%</td>
<td>73%</td>
<td>61%</td>
<td>93%</td>
</tr>
<tr>
<td></td>
<td>Hct (%)</td>
<td>&lt;41</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Cut-offs determined by the data.

Jeger et al Scientific World Journal 2012 p 821794
Can the TEG explain the coagulation changes in haemorrhagic and shocked trauma patients?
Trauma Induced Coagulopathy

- Usually has an adequate Thrombin burst
- Fibrinogen levels are reduced
- Fibrin laydown and cross-bridging is impaired
- Fibrinolysis is increased
Can TEG direct product treatment in haemorrhagic and shocked trauma patients?
The place of TEG?

Appendix 1  Thrombelastography (TEG) treatment algorithm for patients with ongoing bleeding

<table>
<thead>
<tr>
<th>TEG Parameter</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>R 11–14 min</td>
<td>2 x FFP or 10 ml/kg</td>
</tr>
<tr>
<td>R &gt; 14 min</td>
<td>4 x FFP or 20 ml/kg</td>
</tr>
<tr>
<td>MA 46–50 mm</td>
<td>1 platelet concentrate</td>
</tr>
<tr>
<td>MA &lt; 46 mm</td>
<td>2 platelet concentrates</td>
</tr>
<tr>
<td>Angle &lt; 52</td>
<td>2 x FFP or fibrinogen</td>
</tr>
<tr>
<td>Ly30 &gt; 8%</td>
<td>Antifibrinolytics</td>
</tr>
</tbody>
</table>

R, R-time, minutes; MA, maximum amplitude; Ly30, lysis in percent 30 min after MA is reached; FFP, fresh-frozen plasma.
One platelet concentrate pooled from the buffy-coat from four donors.

Johanson Vox Sanguinis 96 111-118
TEG ALGORITHM FOR MANAGEMENT OF BLEEDING PATIENTS

Kaolin TEG

- **R time 10-14**
  - **FACTOR DEFICIENCY**
    - Give 2 units FFP
  - **Heparin effect possible?**
    - Check Heparinase TEG
  - **Difference in R time > 2min**
    - Give Protamine 25-50mg

- **R time >14**
  - **FACTOR DEFICIENCY**
    - Give 4 units FFP

- **MA <49 or \( \alpha <52 \)**
  - Measure Functional Fibrinogen

- **Ly30 >8**
  - **FIBRINOLYSIS**
    - Tx A 10-20mg/kg

- **MA <49 FFMa <7**
  - **LOW FIBRINOGEN**
    - Give 4u Cryo (or 4g Fibrinogen)

- **MA <49 FFMa 7-14**
  - **LOW FIBRINOGEN**
    - Give 2u Cryo (or 2g Fibrinogen)

- **MA 45-49 FFMa >14**
  - **LOW PLATELETS**
    - Give 1 unit platelets

- **MA <45 FFMa >14**
  - **LOW PLATELETS**
    - Give 2 units platelets
Most of the benefit in TEG in improving blood management outside trauma is in:

- **Stopping** blood product use when it isn’t required
- **Targeting** specific product use when a defect exists (as opposed to a reduced conc of something)
ADHB Adult Code Crimson MTP

Team Leader Responsibilities
- Team leader should be a trauma team member
- Notify Coag Lab and send Coag requests on the Labplus Urgent form (orange border)
- Activate protocol by ringing Blood Bank (ext 24015) and say "I am activating the "Code Crimson MTP"
- Call for each box as required
- Make a decision to cease MTP and contact Blood Bank

Blood Bank Responsibilities
- Ensure X-match sample processed ASAP after O-neg release
- Notify NZBS Medical Officer after issuing MTP Box Four
- Thaw next box in advance and await request
- Ensure supply of platelets

Contacts
- Blood Bank - Ext 24015
- Coagulation Lab - Ext 22069
- Level 8 Anaesthetist - 021 496 374

Massive bleeding (ABC Score ≥ 2/4)
- Consider Tranexamic acid 1g
- Ensure delivery of X-match specimen to Blood Bank
- Give 3 Units O-neg or type specific RBC
- Ring Blood Bank to Activate Code Crimson MTP

REQUEST, DELIVER AND TRANSFUSE AS BELOW:

- **MTP BOX ONE**
  - 2U RBC and 4G Fibrinogen concentrate

- **MTP BOX TWO**
  - 4RBC
  - 4FFP
  - 3U Cryoprecipitate

- **MTP BOX THREE**
  - 4RBC
  - 4FFP
  - 1U Platelets

- **MTP BOX FOUR**
  - 4RBC
  - 4FFP
  - 3U Cryoprecipitate

and alternate 3 & 4...

- Check Coags / TEG / Platelets / FBC
- ABGs / Ca++
- Check TEG/Coags / Platelets / FBC
- ABGs / Ca++
- Check TEG/Coags / Platelets / FBC
- ABGs / Ca++
- Repeat every 30 min

Additional treatment thresholds
- if PR >1.5 or APTT >40 consider additional 4 units FFP
- if fibrinogen <1g/L consider additional 3U Cryoprecipitate
- if platelets <75x10^9/L consider additional one pack platelets
- if ionized Ca++ <1mmol/L give 10mls Calcium
Goal-directed coagulation management of major trauma patients using thromboelastometry (ROTEM®)-guided administration of fibrinogen concentrate and prothrombin complex concentrate

Herbert Schöchl1, Ulrike Niemeyer1, Georg Heller1, Wolfgang Voelckel1, Caila Jambor1, Gisela Schärbert1, Silbyle Krzak-Langenecker1 and Cristina Solomon2

Abstract

Introduction: The appropriate strategy for trauma-induced coagulopathy management is under debate. We report the treatment of major trauma using mainly coagulation factor concentrates.

Methods: This retrospective analysis included trauma patients who received ≥ 5 units of red blood cell concentrate within 24 hours. Coagulation management was guided by thromboelastometry (ROTEM). Fibrinogen concentrate was given as first-line haemostatic therapy when maximum clot firmness (MCF) measured by FIBTEM ( fibrin-based test) was < 10 cm. Prothrombin complex concentrate (PCC) was given in case of recent coumarin intake or clotting time measured by extrinsic activation test (EXTEM) > 1.5 times normal. Lack of improvement in EXTEM MCF after fibrinogen concentrate administration was an indication for platelet concentrate. The observed mortality was compared with the mortality predicted by the trauma injury severity score (TRISS) and by the revised injury severity classification (RISC) score.

Results: Of 131 patients included, 128 received fibrinogen concentrate as first-line therapy, 98 additionally received PCC, while 3 patients with recent coumarin intake received only PCC. Twelve patients received FFP and 29 received platelet concentrate. The observed mortality was 24.4%, lower than the TRISS mortality of 33.7% (P = 0.032) and the RISC mortality of 28.7% (P = 0.025). After excluding 17 patients with traumatic brain injury, the difference in mortality was 14% observed versus 27.8% predicted by TRISS (P = 0.0018) and 24.3% predicted by RISC (P = 0.014).

Conclusions: ROTEM-guided haemostatic therapy, with fibrinogen concentrate as first-line therapy and additional PCC, was goal-directed and fast. A favourable survival rate was observed. Prospective, randomized trials to investigate this therapeutic alternative further appear warranted.
Algorithm for treating bleeding in patients with trauma-induced coagulopathy

Temperature
- Optimize preconditions
- Temperature > 34°C
- pH > 7.2
- Calcium >1 mmol/L
- Hematocrit > 24%

Severe trauma (ISS > 16) and/or severe shock
- TXA 15-20 mg/kg BW

Run ROTEM (EXTEM, INTEM, FIBTEM, APTEM)*

1. Focus on: hyperfibrinolysis
   - EXTEM CT > APTEM CT
   - Treat fibrinolysis
     - TXA 15-20 mg/kg BW*

2. Focus on: fibrin deficit
   - FIBTEM CA10 < 7 mm
   - Increase FIBTEM CA10 to 10-12 mm
     - Fibrinogen concentrate 3-6 g
     - (Cryoprecipitate, FFP)

3. Focus on: thrombin generation deficit
   - EXTEM CT > 60 sec
     - EXTEM CT > APTEM CT
   - Treat coagulation factor deficiency
     - PCC 20 U/kg BW (FFP)

4. Focus on: platelet deficit
   - EXTEM CA10 < 40 mm
     - (with FIBTEM CA10 > 10 mm and platelet count <50,000/µL)
   - Increase platelet count to ≥50,000/µL
     - Platelet concentrate

Severe clot deficiency
- EXTEM CA10 < 30 mm
- Treat immediately
- TXA 15-20 mg/kg BW
  - Fibrinogen concentrate 6-8 and
  - PCC 20-30 U/kg BW
  - (cryoprecipitate, FFP [high dose])
  - Platelet concentrate (increase platelet count to ≥50,000/µL)

ROTEM may also identify:

Potential heparin exposure (e.g., cell-saver blood)
- HEPTEM CT < INTEM CT
- Treat heparin effect
  - Protamine 1000-2000 U

Clot instability not related to hyperfibrinolysis
- EXTEM ML > 15% and APTEM ML > 15%
- Consider Factor XIII 100 U
### Sources of Fibrinogen

<table>
<thead>
<tr>
<th></th>
<th>Fibrinogen Content</th>
<th>Thaw Time Delay</th>
<th>Factors Present</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FFP</strong></td>
<td>1:6G/L</td>
<td>30 min. + Transport</td>
<td>Fibrinogen (I) II, VII, IX, X, XII [V &amp; VIII 65% (N)]</td>
<td>Weak TACO TRALI immunomodulation</td>
</tr>
<tr>
<td><strong>Cryoprecipitate</strong></td>
<td>2.5G/150ml</td>
<td>30 min. + Transport</td>
<td>Fibrinogen (I) vWF, VIII XIII fibronectin</td>
<td>Availability no viral inactivation</td>
</tr>
<tr>
<td><strong>Fibrinogen Concentrate</strong></td>
<td>1.0G/100ml</td>
<td>NIL</td>
<td>Fibrinogen (I)</td>
<td>Cost 20% greater than cryoprecipitate</td>
</tr>
</tbody>
</table>
Electron microscopic scan of a ×2000 magnified blood clot.

- **fibTEM > 10mm**
- **Fibrinogen > 1.5-2.0 g/L**

65% haemodiluted

Post fibrinogen administration
TEG is a more intuitive coagulation test for replacement of factors in bleeding

It has the potential to focus on better prediction of TIC

It has the potential to increase our understanding of TIC

It may be the tool to move us to MTP 2.0