TEG, Trauma and Blood Management

Jerod M. Grove, M.D.
Chief Resident
University of Kansas School of Medicine
21 April 2015
Disclosures:

No financial disclosures
Disclosures:

No industry bias, more familiarity with TEG -ROTEM will not be discussed
Outline

• What is Thromboelastography?
  – History of TEG
  – How it works, what exactly does the tracing mean?
  – Correction of specific defects in the coagulation system

• Trauma and the increasing adoption of TEG
  – Role in Massive Transfusion
  – Increasing role in the ICU, at least at KUMC, Denver Health, UTHSC Houston
  – Treatment of fibrinolysis of acute injury

• Use for blood management
  – Couple of recent cases where TEG makes a difference

• My thoughts about the whole process
History

- Developed by Dr. Hellmut Hartert in Germany in 1948 at University of Heidelberg
  - He eventually took his invention to Denver
  - Used for transplant and cardiac surgery for >50yrs
  - Initially all TEG was performed with whole blood (POC)
  - Eventually it was validated against citrated blood (increased use)
  - Now CaCl₂ is added as well as kaolin (speed up the process)
  - Has rapidly become the topic of discussion at most trauma meetings
    - Correction of “coagulopathy of trauma” or “resuscitation”
    - Increased use for fibrinolysis (ACA, TXA)
    - Slowly taking over coagulation management in “Massive Transfusion”
    - Being used along with platelet mapping to tailor anti-platelet and anticoagulation for cardiac patients
    - To determine hypercoagulable state and determine increased anticoagulation need for those at risk for VTE

- My interest has primarily been in the ongoing management of the acutely injured patient in the ICU
How Does it Work?

Sample placed in oscillating cup at 37°C

A pin is suspended from a torsion wire into blood

Development of fibrin strands couple the motion of the cup to the pin - coupling is directly proportional to clot strength

Increased wire tension is detected by a transducer and a tracing is created

Kaolin can be added to activate intrinsic pathway to give faster results
Complete TEG Tracing (Kaolin)
- Measures time from latency to initiation of fibrin formation
- Time reflects level of clotting factors
- Normal R time on Kaolin TEG is 5-7min
- Plasma transfusion triggers may be based on degree of prolonged R-time
K Time and “alpha” Angle

- K is time to develop certain level of clot strength R time= 0 to level of 20mm (1-3min)
- Angle known as “alpha” is the slope of horizontal line to TEG tracing (NL-53-67 degrees)
  - Speed of fibrin build up and cross linking
- Reflect presence of absence of fibrinogen and may be used as cryoprecipitate transfusion trigger
Maximum Amplitude (MA)

- Direct reflection of maximum dynamic properties of fibrin and platelet bonding
- 80% platelets 20% fibrin (NL- 59-68mm)
- Platelet transfusion triggers may be based on sub-optimal MA
- Measures decrease in amplitude 30min post-MA
- Gives degree of fibrinolysis (NL<7.5%) some trauma studies are treating >2%
- ACA and TXA are being studied for Tx of hyperfibrinolysis in Trauma pts
Important TEG Tracings

- **Normal**: R; K; MA; Angle = Normal
- **Anticoagulants/hemophilia**: Factor Deficiency
  - R; K = Prolonged;
  - MA; Angle = Decreased
- **Platelet Blockers**: Thrombocytopenia/
  - Thrombocytopeny
  - R ~ Normal; K = Prolonged;
  - MA = Decreased
- **Fibrinolysis (UK, SK, or t-PA)**
  - Presence of t-PA
  - R ~ Normal;
  - MA = Continuous decrease
  - LY30 > 7.5%; WBCLI30 < 97.5%;
  - Ly60 > 15.0%; WBCLI60 < 85%
- **Hypercoagulation**: R; K = Decreased;
  - MA; Angle = Increased
- **D.I.C**: Stage 1
  - Hypercoagulable state with secondary fibrinolysis
  - Stage 2
  - Hypocoagulable state
Trauma, Coagulopathy and TEG
Role in Massive Transfusion

- Usually defined as >10 Units of blood product administered in 24 hours

- Cotton et al., as well as Denver group actively working on rTEG use in trauma bay

- Has been shown to be as accurate as traditional coagulation studies, ACT predictor of 1 hour transfusion

- I feel that most accurate predictor is patient evaluation (don’t need a number)

- Even rTEG is too slow for first box of blood

- Still only 3min faster than average coag study return (rTEG), but can provided targeted data once available
Role in Massive Transfusion

- TEG speed will have to be drastically increased to be helpful in the acute management of bleeding pt, it does help to identify underlying bleeding diathesis in the acute setting

- May have use for Dx and Tx of acute fibrinolysis
Evidence of acute fibrinolysis may require anti-fibrinolytic therapy along with blood product administration, may be a major benefit of initial TEG usage.
Increasing Use in Trauma ICU

• Trauma to the body causes alterations in coagulation cascade
  – Initial resuscitation can lead to coagulopathy
  – We still don’t really know appropriate resuscitation technique (currently 1:1:1)
  – Patients with large clot volume (pelvic fractures) may have fibrinolysis
  – Underlying liver and renal disease may alter traditional non whole-blood studies
    • ie- renal failure and uremia effect on Plt function, or ESLD and elevated INR and its contribution to real coagulopathy
Increasing Use in Trauma ICU

• Use of TEG after Acute resuscitation phase
  – Allows for proper transfusion ratios
  – May aid in identification of surgical bleeding
  – Can prevent wasted or unnecessary blood product use
  – Aids in identification of true coagulopathy when traditional Plt count, INT, aPTT may be inaccurate
  – Overall leads to a more targeted approach
    • Now we are using sniper rifles instead of shotguns
Blood Management Using TEG

• Case Study #1
  – 44M involved in highway speed MVC. Large volume hemoperitoneum and massive transfusion. Taken emergently to OR for laparotomy. In OR Bolt was placed for closed head injury. On POD#3 ICP had been normal for 24 hours, but INR was 2.8. Neurosurgery wanted FFP reversal prior to DC.
  • TEG was ordered
Case Study #1

TEG was completely normal despite INR elevation, no FFP infused, bolt pulled, no evidence of bleeding. NET BLOOD PRODUCT SAVED 2-4 UNITS FFP.
Blood Management Using TEG

• Case Study #2
  – 38F with ESLD fall from balcony, admitted with closed head injury. Compressive expansile SDH identified. To OR for decompressive craniotomy. POD#1 Plt count 33, Neurosurgery requests to keep >50K.
  • TEG was ordered
Case Study #2

TEG was completely normal despite low Plt count. No platelets were infused. No evidence of bleeding

NET BLOOD PRODUCT SAVED 1 6pack platelets
Conclusions

• Understanding how to interpret tracings is becoming increasingly important

• TEG is here to stay in Trauma, and likely increases in speed will make it a staple with POC testing in trauma bays nationwide (though not quite yet)

• Ongoing management of the massively transfused pt relies heavily on TEG at most major trauma centers

• Likely changes are coming for management of fibrinolysis, and instead of timing, will be TEG driven

• Appropriate utilization of blood products should be the goal of all involved and TEG can help us to transfuse based on actual clot function not just a number
My Thoughts

- Thromboelastography is not new. It is though a useful weapon in the armamentarium to combat the confusing coagulopathies that are becoming increasingly common in patients. As the presence of ESLD, ESRD and those surviving catastrophic traumatic injuries continues to increase, blood management strategies continue to focus on appropriate usage and preventing over utilization.

  - Appropriate use of TEG can aid in both dilemmas
Sometimes, no matter what we think we know, we are just little kids playing with expensive gadgets.
Questions?