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THROMBOELASTOGRAPHY (TEG) IN TRAUMA

Evidence Based Medicine Guideline

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SUMMARY
Thromboelastography (TEG) is a whole-blood coagulation test that has been shown to decrease blood product use and mortality when used to evaluate the coagulation cascade and guide administration of blood products during hemostatic resuscitation in the trauma setting.

RECOMMENDATIONS

Level 1

- **None**

Level 2

- **TEG may be used to screen patients for coagulopathy in the following situations:**
  - **Blunt or penetrating trauma patients who arrive in hemorrhagic shock**
  - **Patients receiving massive transfusion protocol to evaluate for discontinuation or guided product therapy**
  - **Clinical suspicion for hemorrhage or coagulopathy**

Level 3

- **TEG may be used to guide blood product administration in bleeding patients as follows:**
  - **TEG-ACT > 128 or R-time > 10 → transfuse fresh frozen plasma**
  - **K-time > 3 or ∝ angle < 53 → transfuse cryoprecipitate**
  - **MA < 50 → transfuse platelets**
  - **LY30 > 3% → administer tranexamic acid**
- **TEG should be repeated as needed to guide blood product administration until the patient's coagulopathy is corrected.**

INTRODUCTION

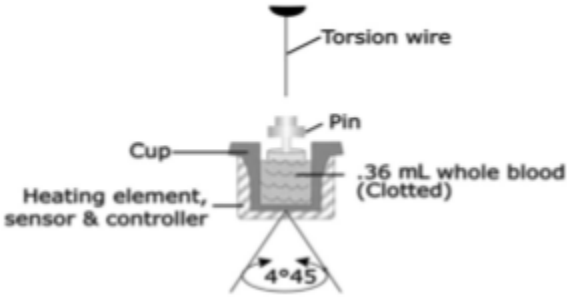
TEG was developed in 1948 by Hellmut Hartert in Heidelberg, Germany as a test to detect clotting factor deficiencies (1). It was not until 1967, when Hardaway used TEG to describe the coagulation changes seen in combat casualties suffering hemorrhagic shock in Vietnam. Its availability in the United States was limited until the 1980s, when the addition of disposable components, tissue activators, and computerized software made the test more practical, reproducible, and timely. TEG is designed to assist the clinician in identifying coagulopathy as demonstrated by deranged clot formation in a whole blood sample. If a coagulopathy is identified, the TEG results will point to the specific therapy to treat it, as identified by the abnormalities in the clot tracing.

LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

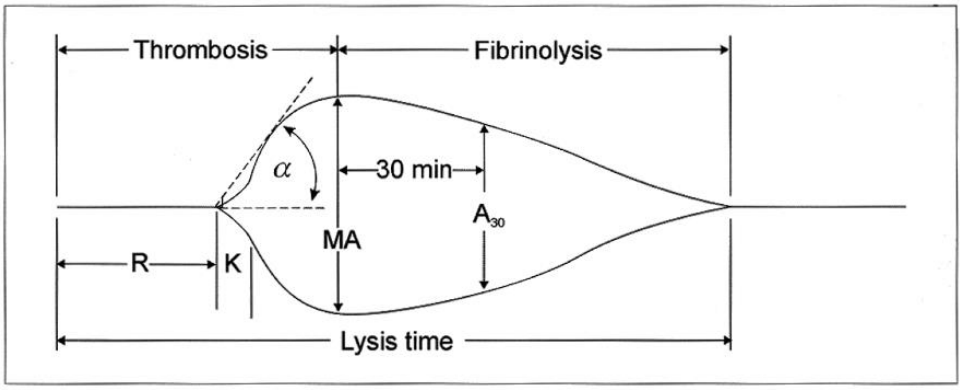
To perform a TEG, an aliquot of blood is placed into a cup that has been pre-warmed to 37°C. A pin is immersed in the sample and attached by a wire to a transducer (Figure 1). The cup rotates around the pin. As clot begins to form, the pin and cup are joined by formation of the clot causing the pin and cup to rotate together. This change in tension is detected by a transducer, which produces a graphical plot of clot strength on the Y axis versus time on the X axis. Different activators may be used to induce the clotting cascade. Conventional TEG is activated with kaolin, which initiates the intrinsic pathway, resulting in the “reaction time” or “R-time” that is analogous to aPTT and INR. This denotes the amount of time it takes the blood to begin forming a clot. It is indicative of the initiation phase of clotting factor activation. Rapid TEG (r-TEG) utilizes tissue factor (TF) in addition to kaolin, activating both the intrinsic and extrinsic pathways and producing the activated clotting time or TEG-ACT.

Figure 1: The TEG mechanism



The next data point encountered is known as the "kinetic time" or “K-time”. The slope of the curve caused by that movement is called the "alpha angle". These two values represent the rate at which the clot strengthens and are most representative of thrombin cleaving available fibrinogen and fibrin. The maximum height of the curve is called the “maximum amplitude”, or MA. This is a result of maximal fibrin–platelet interaction. The termination stage begins with fibrinolytic dissolution of the fibrin–platelet bond between the pin and the cup. The percentage return to baseline of the total MA at 30 minutes is the “lysis at 30 minutes”, or “LY 30”.

Figure 2: TEG tracing with associated data points



TEG is thus a test of whole blood coagulation that assesses multiple points in the clotting cascade and can identify a variety of coagulation deficits. Computer software is available that allows the tracing to be displayed electronically, as the test is performed, at remote locations such as an operating room or trauma bay. Initially results are typically available within 15 minutes with the final tracing becoming available within 45-60 minutes.

LITERATURE REVIEW

The widespread use of TEG in the United States began in the 1980's as an important tool during orthotopic liver transplantation (2). Liver transplantation often requires massive blood transfusion due to blood loss and the complex coagulopathies that occur during the anhepatic and post-reperfusion stages. TEG-guided transfusion algorithms were shown to greatly reduce the requirements for blood product transfusion (2). By the 1990's, TEG was being applied to cardiac surgery which consumes 20% of all blood products in the United States (3). Ten percent of patients have excessive bleeding following cardiopulmonary bypass, 50% of these secondary to coagulopathy. This may occur due to inadequate heparin reversal or the effects of the cardiopulmonary bypass circuit on platelets. The alpha angle was shown to be strongly correlated with total blood loss. Speiss et al., in an analysis of 1079 coronary artery bypass graft (CABG) patients, demonstrated that a TEG-guided algorithm was shown to decrease overall peri-operative transfusion requirements with the number of re-explorations for bleeding decreasing from 5.7% to 1.5% (3). Ak et al. randomized 224 CABG patients to a clinician-directed transfusion strategy or a TEG-guided algorithm (4). Overall, there was no difference in blood loss, packed red blood cells transfused, or clinical outcome. However, the TEG group received fewer transfusions of platelets, fresh frozen plasma (FFP), and tranexamic acid and had significant cost savings.

With its ability to rapidly detect coagulopathies and provide guidance for blood product transfusion, TEG has been applied to the resuscitation of traumatically injured patients. In the setting of massive hemorrhage, the body rapidly activates coagulation pathways to prevent ongoing blood loss. However, a delicate balance must occur to prevent a prothrombotic state, which has complications such as deep venous thrombosis and pulmonary embolism. This results in the so-called "coagulopathy of trauma". Studies have shown that as many as 25% of severely injured trauma patients are coagulopathic upon admission to the emergency department (5-8). In those injured enough to require massive transfusion protocol (MTP), that number has been shown to reach as high as 75% (7).

Cohen et al. examined 203 trauma patients with serial prothrombin time (PT), partial thromboplastin time (PTT), Factor Va, Factor VIIIa, protein C, tissue plasminogen activator (t-PA), and D-dimer levels at arrival, 6 hours, 12 hours, and 24 hours (9). Patients with tissue hypoperfusion and severe traumatic injury showed strong activation of protein C which was associated with coagulopathy. Furthermore, elevated activated protein C levels were significantly associated with organ failure, infection, and death.

The PRospective Observational Multicenter Major Trauma Transfusion (PROMMTT) study evaluated 1,198 trauma patients of whom 41.6% were coagulopathic (10). While also implicating protein C, depletion of Factors I, II, V, VII, VIII, IX, and X was additionally identified in these patients.

Trauma surgeons have traditionally employed conventional coagulation tests (CCTs) to evaluate trauma patients for coagulopathy. These include PT, PTT, and International Normalized Ratio (INR), tests that were developed to guide coumadin and heparin therapy as well as test for clotting factor deficiencies. Platelet count is useful in determining the number of circulating platelets but does not assess platelet function. TEG has thus emerged as a test that than can provide an accurate and real-time picture of whole blood coagulation in the bleeding trauma patient. In one of the first major studies in trauma, only TEG and Injury Severity Score (ISS) were found to be predictive of blood transfusion in this population (11).

In a porcine model, Martini et al. examined PT, PTT, activated clotting time (ACT), and TEG after induced hypothermia and hemorrhagic shock (12). Hypothermia was found to inhibit clotting times and clotting rate whereas hemorrhage impaired clot strength. Combining hypothermia with hemorrhage impaired all these clotting parameters. PT and PTT were not sensitive whereas ACT was not specific in detecting coagulation defects. Only TEG differentiated the mechanism related to clotting abnormalities, leading the authors to conclude that TEG allows better focused treatment of coagulopathy associated with hypothermia and hemorrhagic shock.

Lier et al. performed a systematic search of topics of trauma and coagulation as well as therapeutic options. The acute coagulopathy of trauma impaired survival, correlated independently with an eight-fold increase in mortality within 24 hours, and quadrupled total mortality. They concluded that CCTs are weak predictors of bleeding tendencies in the critically ill (13).

Holcomb et al. evaluated a series of 1974 consecutive trauma patients at a Level 1 Trauma Center with a median ISS of 17, 25% of whom presented in shock and 28% of whom were transfused (14). After controlling for age, mechanism, Revised Trauma Score, base excess, and hemoglobin, the authors found that R-time predicted red

blood cell transfusion and α angle predicted massive transfusion better than PT or PTT. The α angle was superior to fibrinogen in predicting plasma transfusion, and the MA was superior to platelet count in predicting platelet need. These correlations improved for transfused, shocked, or head injured patients. The authors argue that there is little functional information in the CCTs, and the turnaround time is slow in situations where minutes matter.

Schöchl et al. performed a retrospective literature review regarding point-of-care coagulation testing as a means to guide treatment decisions in trauma (15). Conventional plasma-based coagulation testing, such as PT/INR and aPTT failed to fully assess the clotting process. They argue TEG provides a rapid and dynamic bedside assessment of the initiation and kinetics of clot formation, firmness, and breakdown, unlike CCTs, which do not provide information on these parameters. They caution that although TEG-guided transfusion can reduce the amount of bleeding, the use of viscoelastic tests did not reduce morbidity or mortality.

Da Luz et al. performed a descriptive systematic review considering the diagnostic performance of TEG compared to CCT. Current CCTs have limited utility to diagnose early trauma coagulopathies and direct their treatment. The study lacked randomized control trials. They concluded that there is limited, but rapidly growing observational evidence on the use of TEG in trauma (16).

As a test of whole blood coagulation, TEG can be used to guide blood product administration. Using remote tracing software in the emergency department, this can allow rapid decision making early in a patient's resuscitation. In a study of 272 trauma patients, Cotton's group was able to view R- time and K- time within 5 minutes, and α angle and MA within 15 minutes (17). Conventional coagulation tests were not available for 48 minutes. R time and K time were found to strongly correlate with PT/INR and PTT, whereas MA and α angle were found to strongly correlate with platelet count. Controlling for demographics and emergency department vital signs, R time best predicted the need for massive transfusion in the first 6 hours. These findings were echoed in a recent study of pediatric trauma (18).

Tapia et al. evaluated their use of a TEG-guided massive transfusion algorithm before switching to the now common 1:1:1 (pRBC:FFP:platelet) massive transfusion protocol (MTP) (19). TEG-guided resuscitation was equivalent to standardized MTP in patients receiving 6 or more units of pRBC's. However, MTP worsened mortality in penetrating trauma patients receiving 10 or more units pRBC's. TEG-directed therapy seemed to provide optimal ratios through laboratory data rather than a pre-prescribed, ratio-based transfusion practice. Thus, utilization of TEG in conjunction with MTP may help elucidate those patients requiring more aggressive resuscitation because of their unique physiologic response to injury and, inversely, avoid unnecessary transfusion of products when they are not needed.

In a prospective, pragmatic, randomized trial performed at a Level 1 Trauma Center, Gonzalez et al. compared the use of CCT and TEG to direct the resuscitation of severely injured patients (20). Twenty-eight-day survival, the primary outcome, was significantly higher in the TEG group. The amounts of administered crystalloid and RBC were similar between the two groups. The TEG group had significant improvement in survival at 28 days and at 6 hours from injury, while using less plasma and platelets in the early phase of resuscitation compared with the CCT group. They found an MTP goal, directed by TEG, resulted in a survival benefit compared with CCT-guided transfusion. This resulted from less hemorrhagic deaths and less early deaths occurring in the TEG group. MTP based on CCT led to more plasma and platelets transfused in the early phase and more cryoprecipitate transfusion overall. Although the survival benefit was attributable to the first 6 hours from emergency department arrival, survivors in the TEG-guided MTP group also benefited from more ICU-free and ventilator-free days.

An Eastern Association for the Surgery of Trauma (EAST) Practice Management Guideline by Bugaev and colleagues conditionally recommended using TEG-guided transfusions over CCT in adult trauma and surgical patients, and in patients with critical illness (21). This is a conditional recommendation due to the low grade of evidence in the body of literature. Clearly, more research needs to be carried out for this potentially promising method of guided resuscitation.

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Table 1: TEG values and interpretation

TEG Value	Normal	Description	Measures
TEG-ACT (rapid)	86 - 118 sec	“Activated clotting time” to initial fibrin formation	clotting factors (extrinsic/intrinsic pathways)
R time (conventional)	3.0 – 8.0 min	“Reaction time” to initial fibrin formation	clotting factors (intrinsic pathway)
K time	1.0 - 3.0 min	“Kinetic time” for fibrin cross linkage to reach 20 mm clot strength	fibrinogen, platelet number
α angle	55.0 - 78.0 degrees	Angle from baseline to slope of tracing that represents clot formation	fibrinogen, platelet number
MA	51.0 – 69 .0 mm	Maximum amplitude of tracing	platelet number and function
G value	5.3 - 12.4 dynes/cm^2	Calculated value of clot strength	entire coagulation cascade
LY 30	0 - 3%	Clot lysis at 30 minutes following MA	fibrinolysis

Table 2: Suggested TEG-guided transfusion

TEG Value	Transfuse
TEG-ACT > 128	FFP
R time > 10	FFP
K time > 3	cryoprecipitate
α angle < 53	cryoprecipitate +/- platelets
MA < 50	platelets
LY30 > 3%	tranexamic acid

MASSIVE TRANSFUSION PROTOCOL