



Viscoelastic hemostatic assays in the management of the pediatric trauma patient



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ABSTRACT

Viscoelastic hemostatic assays (VHA), such as TEG and ROTEM, are whole blood tests that depict functional coagulation both numerically and graphically. The development of rapid VHA technology, which allows for the first data points to result within minutes of test initiation, has increased the utility of these tests in the treatment of trauma patients. Both adult and pediatric centers have integrated VHAs into trauma resuscitation and transfusion protocols. Literature regarding the use of VHAs for injured children is limited. Here, we discuss the mechanics and interpretation of VHAs as well as the use of VHAs in data-driven resuscitation of pediatric trauma patients. Novel research on fibrinolysis states after injury as well as hypercoagulable state diagnosed with VHAs are presented.

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Viscoelastic assays in trauma

Introduction

Viscoelastic hemostatic assays (VHA) are whole blood tests that are a functional measure of clot formation and degradation. There are two primary instruments that perform this type of testing: thromboelastography¹ (TEG, Haemonetics[®]) and thromboelastometry² (ROTEM, Tem International GmbH[®]). Our center utilizes and is most familiar with TEG; however, both are applied in a similar manner and are relevant for the following discussion. In the following text, TEG and ROTEM will refer to the specific test, and viscoelastic hemostatic assay (VHA) will be used when discussing general aspects pertaining to both tests.

As functional assays, VHAs supplement existing conventional coagulation tests (CCTs). CCTs, including international normalized ratio (INR), prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen and platelet counts, do have value in the workup and management of trauma patients. Acute traumatic coagulopathy, as defined by an admission INR ≥ 1.3 , is common in children (incidence 20–30%)^{3–10} and is associated with increased mortality and functional disability.^{5,6,8} However, children are unique from adults in that the incidence of TBI is greater and hemorrhagic shock is less prevalent.¹¹ Even in the

setting of an elevated INR, children are not commonly clinically coagulopathic, and there is little evidence to suggest a true increased risk of bleeding with invasive interventions.¹² Further, research has shown that blood product transfusion does not normalize INR or reduce mortality in severely injured children.¹³ While INR is a valuable measure for outcome prognostication, it does not consistently represent a treatment target for most patients.

VHAs have the potential to identify a point of intervention in the subset of children who do have a functional coagulopathy. VHAs capture the contribution of the endothelium and cellular components of blood, monitor all phases of coagulation, measure fibrinolysis and are functional tests that depict component activity rather than static counts, and can provide information on hypercoagulability. In studies comparing INR and TEG, patients with an elevated INR do not necessarily have functional coagulopathy based on TEG values.^{14,15} VHAs, therefore, can be used to distinguish between children with an elevated INR associated with the body's response to severe injury versus those with a true bleeding tendency.

TEG and ROTEM are by no means a new technology; however, their use in trauma has relatively recently gained popularity within the trauma community. It is currently mandated by the American College of Surgeons for VHAs to be available in all level 1 and level 2 trauma centers, although in surveys of trauma center practices only 9–18% of sites reported routinely incorporating point-of-care VHAs into MTP policies.^{16,17}

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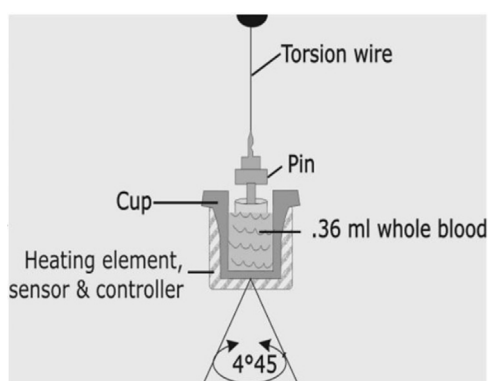


Fig. 1. Mechanics of TEG.¹

Logistics and test mechanics

In our institution, the laboratory technician receives notification of level 1 patient alerts via pager and begins preparation of the reagents prior to patient arrival so as to minimize time to test results. Reagents required are kaolin for standard TEG, and kaolin plus tissue factor for rapid TEG. These require 10–15 min to come to room temperature prior to mixing with the specimen. Blood samples are collected in citrated tubes after an initial 5–10 cc waste and sent to the central laboratory via the pneumatic tube system. Specimens are processed according to manufacturer's recommendations by designated TEG technicians using TEG 5000 Hemostatic Analyzers (Haemonetics[®]). All samples are run immediately upon receipt per hospital protocol for trauma patients; however, there is a 2 h shelf life allowable for specimens in citrated tubes. Integrity of the specimen in the tube system, two sizes of vacutainer tubes (1.0 and 1.8 mL) in use at our facility and the manufacturers' normal ranges were validated in our pediatric population prior to implementation of rapid thromboelastography as standard of care in our hospital.

The laboratory computers are connected to the hospital network and results are available in real time via a remote-viewing software platform. Results can, therefore, be accessed from anywhere in the hospital. This is of particular value in trauma patients who potentially pass quickly through many locations during their initial evaluation (trauma bay, radiology suite, interventional radiology, operating room, and intensive care unit). Further, the ability to visualize the tracing and results as the test is ongoing allows early and aggressive targeted resuscitation.

VHA results include numeric values and a visual representation of a patient's coagulation status. To perform TEG testing, a metal pin suspended by a torsion wire is immersed in the whole blood sample in a cup (Figure 1). The cup is then rotated back and forth around the fixed pin. Once blood starts to clot, fibrin strands are formed and the blood thickens which changes the movement of the cup around the pin. After a clot has formed and subsequently is breaking down, the thinning of the blood in the cup will again change the movement sensed by the pin. These changes are detected by a computer and are converted into a tracing via a

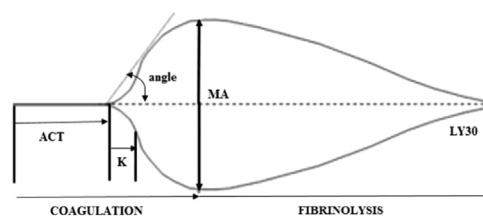


Fig. 2. TEG parameters and tracing.

software program that reflects the entire process of clot formation and ultimate dissolution. ROTEM employs a similar design: the pin is connected to an optical detector and suspended in the sample cup. The pin is rotated and once blood starts to clot, fibrin strands deflect the pin and the tracing is generated.

Test Parameters

Both TEG and ROTEM measure clot initiation, clot kinetics, clot strength, and fibrinolysis (Table 1). VHA parameters correspond with abnormalities in these processes. For instance, TEG parameters and primary contributing components include activated clotting time (ACT)/reaction time (R value) – clotting factors; K value and alpha angle (α) – fibrin cross-linking; maximum amplitude (MA) – fibrinogen and platelets; and lysis at 30 min (LY30) – fibrinolysis. While both have good clinical predictive value, studies of the two tests side by side show weak or no association preventing a direct comparison.^{18,19} Identification of abnormalities in these parameters permits transfusion of products that address a patient's specific blood product need.

Test interpretation

Each part of the tracing depicts the function of a different component or contributor to the clotting cascade (Figure 2). In a standard TEG, the R value reflects clotting factors and is the time until formation of the first measurable clot (amplitude of 2 mm); an abnormality would be treated with plasma. In rapid TEG, the R value is replaced by the ACT value, which results in seconds as opposed to minutes. The remainder of the values is the same for both standard and rapid TEG tests. K is the time from the beginning of clot formation until a fixed level of clot strength (amplitude of 20 mm). The alpha angle is the slope of the tracing between the R/ACT and K values; these reflect fibrinogen function and an abnormality would be treated with cryoprecipitate. MA primarily reflects platelet function and is the highest vertical amplitude of the TEG tracing; an abnormality would be treated with platelets. And finally, the LY30 reflects the degree of fibrinolysis and is the breakdown of the clot 30 min after MA is reached; an abnormality would be treated with an antifibrinolytic agent such as tranexamic acid (TXA). The American College of Surgeons TQIP Massive Transfusion in Trauma Guidelines recommends transfusion triggers as shown in Table 2.

VHA-guided resuscitation relies upon serial testing (test-treat-test-treat strategy). The VHA is sent and the first round of product transfusion is undertaken, followed by repeat VHA to evaluate for

Table 1
Comparison chart for TEG[®] and ROTEM[®] parameters.

Variable	TEG [®]	ROTEM [®]
Time to first clot formation: 2-mm deflection above baseline	R	CT (clotting time)
Time to standard clot firmness: from 2 mm above baseline to 20 mm above baseline	K	CFT (clot formation time)
Alpha angle (deg.)	α (slope between R and K)	α (angle of tangent at 2-mm amplitude)
Maximum clot strength: maximum height of tracing	MA (maximal amplitude)	MCF (maximal clot firmness)
Fibrinolysis	LY30, LY60	CL30, CL60

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