Review of Thromboelastography (TEG): Medical and Surgical Applications

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Clinical Medicine Insights: Circulatory, Respiratory and Pulmonary Medicine
Volume 17: 1–8

DOI: 10.1177/29768675231208426

ABSTRACT: Thromboelastography (TEG) is a laboratory assay utilized to evaluate hemostatic properties of blood, identify coagulopathy, and guide blood product administration. While the clinical use of TEG started in the care of surgical patients, the assay has now been incorporated more routinely in the care of the medical patient as well. In this review, we explore the evolution of TEG from the historical perspective of its inception to the current state of the art of the assay. The TEG procedure and its measurements are illustrated along with a table that summarizes recommendations from across the medical and surgical literature. After each section, we review salient learning points to provide the busy clinician with information that can be immediately integrated at the bedside. We conclude with a series of summary questions to check for comprehension and direct the reader to additional resources to improve their knowledge of TEG.

KEYWORDS: Hemorrhage, coagulation, anticoagulants, antiplatelets, fibrinolysis

RECEIVED: December 13, 2022. **ACCEPTED:** September 22, 2023.

TYPE: Review

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article.

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Introduction

"In the Babylonian Talmud is the story of the four sisters of Sepphoris who, some 2000 years ago, went to see Rabban Simeon ben Gamaliel with a rather alarming story- three of their sons had bled to death at circumcision. They wanted to know whether the fourth son should be circumcised. The Rabban gave a rather wise reply which, in a nutshell, was "no." He said furthermore, "there are some whose blood is loose and others in whom it coagulates." Quote from E.J. Walter Bowie, MD.[1]

Hemorrhage is a major cause of morbidity and mortality worldwide and is often complicated by or incited by coagulopathy. Thromboelastography (TEG) is a laboratory assay that measures the viscoelastic properties of whole blood clotting. TEG can identify patients who have coagulopathy and can guide blood product transfusions. The integration of TEG with traditional coagulation testing in trauma, cardiac, and liver surgical patient management has had beneficial effects on patient outcomes. TEG has been less well studied in non-surgical bleeding patients but has the potential to aid in coagulopathic and bleeding medical patients.

Although it has only recently gained broader acceptance, TEG has been in use for over 60 years since it was developed in 1948 by Helmut Hartert at the University of Heidelberg.[2] TEG experienced limited use in Europe, and several publications in Italy described its usefulness in identifying hemorrhagic disorders in the 1950s.[3,4] TEG gained clinical importance in the 1960s with the advent of liver transplantation. The first liver transplants were performed by Thomas Starzl in 1963. Kurt von Kaulla was a hematologist at the University of Colorado who used TEG to identify coagulation deficits in uremic patients.[5] Von Kaulla was present during the first liver transplant on a 3-year-old boy with biliary atresia. The boy suffered uncontrollable hemorrhage after his liver had been removed and died on the table. Serial TEGs as well as coagulation factor levels were monitored throughout the operation. TEG tracings showed extreme fibrinolytic activity that was only reversed with aminocaproic acid and even then showed persistent hypocoagulability.[6] From that point on, TEG was used during all of Starzl's liver transplants to identify coagulopathy and to guide transfusions and administration of hemostatic agents. In 1963, Starzl included the management of coagulopathy in his methods for liver transplantation: "The overall clotting process was monitored by serial thromboelastograms, as described by von Kaulla. These provide continuous mechano-optical recordings of the onset and progress of fibrin formation and fibrinolysis, insight into speed and kinetics of coagulation being afforded thereby as well as information on the final firmness of the clot."[7] This utilization of TEG shows it has had clinical importance for over 50 years.

The available literature uses different terms when discussing similar concepts, so the term TEG in this text will be used to refer to rotational viscoelastic testing in general. TEG more specifically refers to one testing platform within a group of tests measuring dynamic viscoelastic changes in whole blood during coagulation.[8] More general terms used when referring to this group include viscoelastic method (VEM) or viscoelastic hemostatic assay (VHA). The differences between available assays will be described in this text. When discussing trials, the specific assay used will be described in more detail.

Explanation of the TEG Procedure and Measured Values

TEG is an assay to evaluate the viscoelastic properties of whole blood as clots form and dissolve. The assay involves placing a

sample of blood in a rotating cup with a pin suspended within the blood. The pin is connected to a torsion wire and a mechanical-electrical transducer. Because the sample is collected in blood draw tubes containing sodium citrate to prevent coagulation during handling and transportation to the laboratory, an activator must be used to initiate clotting. The blood sample is warmed to a temperature of 37°C, and calcium is added to overcome the citrate. The simplest tests do not add any additional substrates as activators, and the test will proceed. Simultaneous to the activators being added, the cup starts rotating, and as the blood coagulates, increased rotational forces are transferred from the cup through the forming clot to the pin and are converted to electric signals. These signals are graphed using a computer into a plot of clot strength versus time, as demonstrated in Figure 1.[9] The values generated by the TEG are the reaction time (R time), kinetic time (K time), alpha angle (α), maximum amplitude (MA), and lysis at 30 min (Ly30). The R time refers to the time from the start of the assay to the point where clot strength reaches 2 mm amplitude.[10] This corresponds to the time from initiation of the coagulation cascade to the generation of fibrin and clot propagation. Factors increasing R time include factor deficiencies and anticoagulants. K time refers to the time starting at R time to the point where clot strength reaches an amplitude of 20 mm. The alpha angle (α) is formed by the x-axis and the slope between points R and where K intersects the TEG curve. The value of α is inversely proportional to K. Both K and alpha measure the rapidity of fibrin formation and cross-linking.[11] MA is the maximum value that the clot strength tracing reaches. MA is determined most by platelet number and function and, to a lesser extent, fibrinogen and other coagulation factor concentrations. Ly30 is the percent decrease in the clot strength 30 min after achieving max amplitude. Increased Ly30 indicates hyperfibrinolysis.[12,13]

TEG is one form of VHA measuring rotational forces transmitted through whole blood as it clots, and the other widely used platform is known as rotational thromboelastometry (ROTEM). ROTEM refers to the platform where the pin rotates and forces exerted on the cup are recorded. Both tests measure the firmness of the clot by similar physical derivations and result in similar graphs of clot strength over time. The results from TEG cannot be directly interchanged with those of ROTEM, and manufacturers for each product have specific normal ranges for test results. The ROTEM variable called clotting time (CT) is analogous to reaction time (R) in TEG. The ROTEM clot formation time (CFT) is analogous to the kinetic time (K). The alpha angle retains its name and significance across both platforms; however, in TEG, the angle is measured between the x-axis at R and a line drawn between the R and K points on the curve, whereas in ROTEM, the angle is measured between the x-axis and a line drawn tangential to the clot strength curve. Maximum clot firmness (MCF) is analogous to MA. The percentage of clot lysis at 30 min (Ly30) after MCF can be called LI30 in ROTEM versus Ly30 or CL30 in TEG.

TEG values can be affected by coagulation factor deficiencies or impaired function, medications including anticoagulants, platelet count or platelet function, and changes in

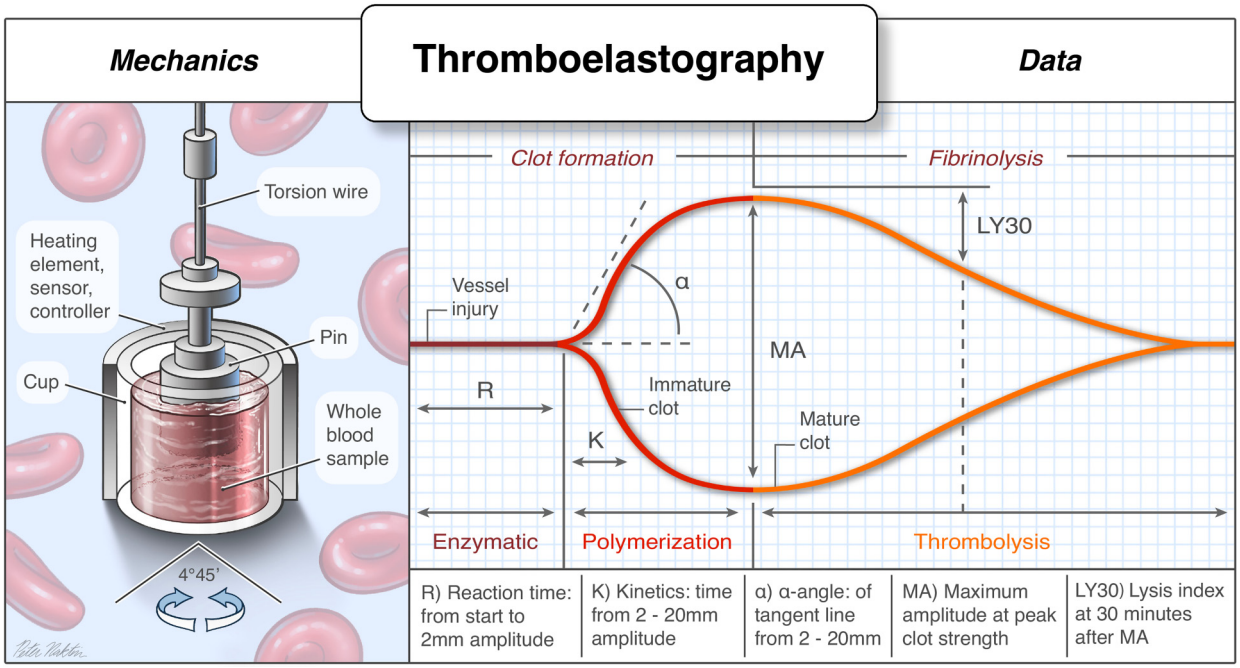

Figure 1. A diagram of the thromboelastography apparatus and the tracing produced by the assay with key values generated from the plot. Printed with permission of © 2022 Augusta University and Peter Naktin.

fibrinolysis.[13] The following examples of clinical situations cause abnormal TEG values and will help in understanding TEG and its potential uses. R-time will be prolonged in patients with significant bleeding and coagulation factor depletion as well as in patients undergoing anticoagulation therapy (especially with inhibition of intrinsic and common pathways). The alpha angle will be decreased in patients with hypofibrinogenemia, such as in severe trauma, hemorrhage, after thrombolytic administration, diffuse intravascular coagulation, and some end-stage liver disease patients. Alpha angle may also be decreased in dysfibrinogenemia either congenital or acquired, for example, in ESLD or some autoimmune disorders. MA will be decreased in thrombocytopenia; however, the platelet level at which MA becomes abnormal can be variable and may remain normal until there is severe thrombocytopenia. MA is also affected by fibrinogen levels, especially in trauma patients, and is also affected by antiplatelet therapy. Ly30 is normally less than 3% but will be higher in hyperfibrinolysis that may be encountered in severe trauma or with thrombolytic therapy. Of note, two important causes for coagulopathy that may not be identified and result in "false" negative testing are hypocalcemia and hypothermia. Because calcium is used to activate clotting and blood samples are heated to 37°C, the in vitro conditions may not accurately approximate the abnormalities in these populations.[14,15]

To add another layer of understanding in particular coagulation profiles, there are additional substrates that may be added at the same time as activators. The commercially available TEG 6 s analyzer includes multiple channels to run the following tests concurrently. The citrated kaolin (CK) channel uses kaolin to activate clotting in the blood sample via the intrinsic pathway. The Citrated Kaolin heparinase (CKH) channel includes heparinase, and if the R time is significantly shorter with CKH compared to the R obtained with the CK channel, then iatrogenic heparin may cause the observed coagulopathy. Citrated rapid TEG (CRT) uses tissue factor in the activation of clotting via the extrinsic pathway to shorten clot initiation and more rapidly give MA values within approximately 10 min. Citrated functional fibrinogen (CFF) uses a GP IIb/IIIa platelet inhibitor in the assay to restrict platelet activity, and when used in conjunction with TEG CK, CFF can assess the contribution of platelets versus fibrinogen to clot strength.[16] ROTEM has available tests to specifically assess extrinsic and intrinsic pathways (EXTEM and INTEM), the contribution of dysfibrinogenemia to clot strength (FIBTEM compared to EXTEM), fibrinolysis (APTEM compared to EXTEM), and the contribution of heparin to clot initiation time (HEPTEM compared to INTEM).

Based on the known causes for abnormal TEG values, more selective blood product transfusions and medications may be administered. In general, prolonged R requires plasma to replace clotting factors, decreased α requires cryoprecipitate to replace fibrinogen, decreased MA requires platelets or DDAVP, and increased Ly30 requires tranexamic acid or aminocaproic acid. Table 1 shows TEG-based transfusion protocols for Kang and Wang in transplant patients, Redfern in cardiac surgery patients, and Kumar in patients with GI bleeding and cirrhosis. The following sections will further discuss the development of TEG use in medical and surgical practice.

Learning Points:

- TEG measures the viscoelastic properties of blood during coagulation. More simply, it measures how quickly blood clots as well as the adhesiveness and stability of clots.

Table 1. Comparison of recommended interventions for TEG abnormalities across the medical literature.

Parameter	Treatment Values	Treatment Protocol
R (TEG)	R > 15 min[17]	2 units of fresh frozen plasma(FFP)
	R > 10 min[18]	FFP titrated over repeat TEG assays
	R < 4 min[19]	Anticoagulation
	R: 11-14 min[19]	2 units FFP
	R > 14 min[19]	4 units FFP
	R > 10 min[20]	FFP 10 mL/kg IBW
ACT (RapidTEG w/Tissue Factor)	Initial ACT > 111-139s[21]	2 units FFP
	Initial ACT > 140s[21]	2 units FFP + 10-pack cryoprecipitate (Cryo) + 1 unit platelet apheresis
	Subsequent ACT > 110s[21]	2 units FFP
α-angle (TEG)	α < 45°[17]	6 units Cryo
	α < 45°[18]	5 units Cryo
	α < 45°[19]	0.06 units/kg Cryo
	α < 45°[20]	5 units Cryo
α-angle (RapidTEG)	α < 63°[21]	10-pack Cryo
MA (TEG)	MA < 40 mm[17]	10 units Platelets
	MA < 55 mm[18]	1 unit platelet apheresis
	MA > 73 mm[19]	Antiplatelet therapy
	MA: 46-54 mm[19]	0.3 μg/kg DDAVP
	MA: 41-45 mm[19]	1 unit platelet apheresis
	MA ≤ 40 mm[19]	2 units of platelet apheresis
	MA < 55 mm[20]	1 unit platelet apheresis
MA (RapidTEG)	MA < 55 mm[21]	1 unit platelet apheresis
Ly30 (RapidTEG)	Ly30 ≥ 7.5% (later reduced to ≥3%)[21]	Tranexamic acid 1 g IV

- The reaction time (R) measures the time from activation of the coagulation cascade to the point when the clot forms.
- Kinetic time (K) measures the time from R to the time when clot strength is 20 mm.
- Alpha angle (α) is the angle formed between the line of the x-axis and a line from the point where clotting starts (R, 2 mm) to the point on the clot strength curve where the K time and 20 mm meet
- Maximum amplitude (MA) is the greatest value of clot strength achieved during the assay, measured from the X-axis unidirectionally to the highest point on the curve.
- Lysis at 30 min (Ly30) is the percent decrease in clot strength from MA at a point 30 min after the time MA is achieved.
- Prolonged R time can be due to coagulation factor deficiency/dysfunction and anticoagulants.
- Reduced α-angle and increased K time may be caused by platelet deficiency or platelet aggregation dysfunction and fibrinogen deficiency or dysfunction
- Decreased MA may be caused by severe platelet deficiency, antiplatelet therapies, or severely disturbed coagulation factor/fibrin formation.
- Increased Ly30 may be due to hyperfibrinolysis, such as in some cases of trauma or surgery, liver failure, and fibrinolytic medication use.

TEG Use in Surgical Specialities

As discussed earlier, transplant surgery has used TEG since at least 1963. After Starzl moved to the University of Pittsburgh, he continued to utilize TEG intraoperatively. In 1985, anesthesiologists YooGoo Kang and Starzl published the first trial comparing intraoperative hemostatic monitoring of patients with TEG and TEG-guided blood product replacement to nonmonitored patients undergoing liver transplant.[17] They found a 33% decrease in blood and infusion volume among TEG-monitored patients.[17] There have been multiple trials since then comparing TEG-monitored and nonmonitored liver transplant patients, including a randomized control trial by Shenchih Wang in Taiwan in 2010, all showing decreased blood product utilization with equivalent patient outcomes.[18,22–24]

Other surgical specialties that have used TEG extensively are cardiac surgery and trauma surgery. Cardiac surgery in particular requires close monitoring of coagulation in its patients since intraoperative hypothermia and cardiopulmonary bypass induce significant hemostatic changes.[25] In the United States, cardiac surgery consumes 20% of the available blood supply. In some case series, 83% of these patients are transfused during the perioperative period.[26] The first randomized controlled trial in cardiac surgery patients comparing TEG-guided transfusions versus usual care was published by Andrew Westbrook in 2009 at the Alfred Hospital in Australia.[27] They found a 58.8% reduction in total blood product usage and a trend toward improved short-term outcomes in the TEG group.[27] A large prepost intervention trial in 2017 by Moront, Fleming, and Redfern including 681 patients showed that mean units of blood products decreased by 40%, decreased risk of reoperation, reduced postoperative length of stay (LOS), and decreased 6-month mortality.[19,28]

Trauma surgery is another specialty utilizing TEG to identify coagulopathy and guide therapy. Trauma is the leading cause of death worldwide for people aged 15-49.[29] Hemorrhage is the cause of death in up to 89% of major vascular trauma patients, and half of those deaths occur after control of major bleeding sites.[30] Trauma surgeons point to the concept of the "Bloody Viscous Cycle" or "Lethal triad" as the most dangerous clinical challenge for patients after initial injury. The lethal triad refers to coagulopathy, acidosis, and hypothermia, which are present in many trauma patients as a result of hemorrhagic shock and then iatrogenic worsening of the situation with resuscitation, including cold crystalloids that further dilute coagulation factors and worsen hypothermia and acidosis. The presence of coagulopathy on presentation in trauma significantly increases the risk of mortality.[31] Primary fibrinolysis has been noted in trauma, and TEG has been used to identify patients with this clinical condition in trauma.[32] TEG identified that hypocoagulability was correlated with the injury severity score and the need for massive transfusion.[31,32] TEG has been used to identify hyperfibrinolysis occurring in trauma.[33] Eduardo Gonzalez et al published the first randomized controlled trial including trauma patients meeting criteria for massive transfusion protocol comparing resuscitation based on TEG versus routine coagulation tests in 2016. He found that TEG-guided therapy improved survival and decreased plasma and platelet transfusions.[34] The same group defined optimal transfusion thresholds for rapid thromboelastography parameters.[21]

Learning Points:

- In liver transplantation, transfusions based on TEG decreased blood product usage without worsening clinical outcomes.
- In cardiac and trauma surgery, TEG-guided transfusion protocols have shown decreased blood product utilization and improved outcomes, including decreased mortality.

TEG Use in Medical Specialities

TEG has only recently become more common in medicine subspecialties. The most promising area for study has been in cirrhosis. In cirrhosis, there are multiple factors leading to hypocoagulability, including deficiency in all coagulation factors except factor VIII (which is made in endothelium), thrombocytopenia is common, and 30%-46% of cirrhotics have increased fibrinolysis due to decreased clearance of tissue plasminogen activator (tPA). There are also factors leading to hypercoagulability: decreased concentrations of Protein C and

Protein S, decreased ADAMTS-13, decreased clearance of vWF, and increased plasminogen activator inhibitor-1 (PAI-1).[35] Because of these alterations in hemostasis, cirrhosis conveys an increased risk of thrombosis despite an elevation in PT and liver cirrhosis, especially when compensated is not associated with an increased risk of periprocedural bleeding.[36–39] Portal hypertension and progression of liver failure are the most important factors leading to those clinical events. Armando Tripodi has published extensively on the coagulopathy of cirrhosis and has demonstrated that thromboelastography gives a closer representation of the hemostatic state in cirrhotics.[40,41] His research has found very little relationship between PT/INR and R-time. TEG values are normal in compensated cirrhosis, and R, K, and α become significantly more hypocoagulable in acute infection.[42] Clinically, TEG has been used in cirrhotic patients with elevated PT planning to undergo procedures. In one study, cirrhotic patients who did not demonstrate hypocoagulability were not exposed to blood products. Withholding blood products did not affect clinical outcomes, including bleeding complications. When transfusing to a goal INR < 1.8 and platelet count over 50,000/μL, 100% of cirrhotics were transfused before procedures. When transfusing to goal R-time less than 40 min and max amplitude over 30 mm, only 16.7% of patients were transfused.[43,44] Regarding gastrointestinal hemorrhage and the utility of thromboelastography, hypocoagulability on TEG has been associated with an increased risk of rebleeding after endoscopic intervention.[45] An important point to make here is varix development, and the risk of upper gastrointestinal bleeding is unrelated to coagulopathy.[46] Taking these studies together, it is clear now that prolonged PT and mild or moderate thrombocytopenia are not worthwhile indications for blood product transfusion in nonbleeding cirrhotics.

Hypercoagulability can be identified by TEG using the same results from the assay used to identify hypocoagulability. Decreased R, increased α, increased MA, and decreased Ly30 compared to normal ranges may indicate hypercoagulability. An increased risk for venous thromboembolism has been identified in trauma patients with hypercoagulable TEG.[47] Another population with developing evidence for TEG use identifying hypercoagulability is COVID-19.

During the COVID-19 pandemic, the prevalence of venous and arterial thrombotic events in patients diagnosed with COVID-19 was observed to be higher than expected.[48,49] In European and Chinese cohorts, 20%-30% of patients had clinical thrombotic events, most often venous thromboembolism, but there are also reports of increased ischemic stroke and myocardial infarction. Fibrin microthrombi were found in 60% of COVID-19 autopsies. The utility of TEG in identifying COVID-19-associated coagulopathy and guiding thromboprophylaxis and anticoagulation has been studied with mixed results. Some investigators have found hypocoagulable profiles (decreased MA) to be associated with VTE, possibly from consumptive coagulopathy.[50] Other investigators have found decreased Ly30, indicating hypofibrinolysis as an accurate predictor of VTE, but these findings have been challenged by more recent articles.[51]

The use of TEG and viscoelastic testing for coagulation, in general, is an expanding area for study and already shows promise in guiding transfusions in bleeding surgical and some nonsurgical patients. In the future, TEG may have more evidence to support its application in understanding hypercoagulability and the clinical implications of TEG-guided treatment for hypercoagulability.

Learning Points:

- In cirrhosis, the INR reflects poor hepatic synthetic function but does not accurately diagnose hypocoagulability.
- Cirrhotic patients in preprocedural settings who were transfused with FFP to correct R time and platelets to correct MA received markedly less FFP and platelets versus those who were transfused with commonly used safety cutoff levels of INR and platelet levels.

TEG in GI Bleeding

An important question that has not been answered is whether TEG-guided transfusions in gastrointestinal bleeds, particularly in cirrhotic patients, would be beneficial over the usual standard of care.[52–54] There have been retrospective reviews and case series describing the use of TEG in gastrointestinal bleeding cirrhotic patients (Predicting post endoscopy rebleeding vs correcting coagulopathy).[45,46,54] In 2019, Manoj Kumar and his group in New Delhi, India, authored the first randomized controlled trial comparing TEG-guided transfusions versus standard of care in nonvariceal upper gastrointestinal bleeding cirrhotic patients.[20] They found that significantly fewer blood products were used in the TEG group. In the standard of care group, 87.2% were transfused with all three of FFP, cryoprecipitate, and platelets versus only 26.5% of the TEG-guided arms received all three. All of the patients in the standard of care arm received at least one of the aforementioned blood products, while 14.3% of patients in the TEG-guided arm received no FFP, platelets, or cryoprecipitate. The other major endpoints included mortality, failure to control bleeding, and failure to prevent rebleeding, and there were no significant differences.[52] This trial included 96 patients and showed a trend toward improved mortality and bleeding control that needs to be explored further with larger trials.

Learning Points:

- In nonvariceal upper GI bleeds in the setting of cirrhosis, TEG-guided transfusions resulted in significantly less blood product utilization versus standard of care and no difference in clinical outcomes.

Future Directions in TEG Application

The indications for TEG may soon advance beyond the evaluation of bleeding patients. It has been studied in other areas of medicine where PT/INR has been insufficient in providing adequate information on coagulation status. Diffuse intravascular coagulation (DIC) is a common complication of sepsis and septic shock. Hypocoagulability in two or more TEG values has been compared to the International Society for Hemostasis and Thrombosis DIC scoring system for overt DIC. In patients with two or more hypocoagulable TEG values, the sensitivity and specificity for overt DIC were 92% and 81%, respectively.[53] There are no assays approved for clinical use in the United States for assessing the hemostatic effect of NOAC medications. R times have been identified that have high sensitivity and specificity for identifying therapeutic serum levels of NOACs.[55] There has even been a case series using TEG to identify coagulation status in patients with antiphospholipid syndrome.[56] Patients with positive lupus anticoagulant, anti-cardiolipin antibodies, or beta-2 glycoprotein-1 antibodies have baseline prolonged PT and PTT, but this is unreliable in determining thrombotic or hemorrhagic risk. TEG values were most often normal in a case series despite elevated PTT.[56]

TEG use in postpartum hemorrhage (PPH) is an emerging area of interest with some parallels to non-traumatic hemorrgage.[57] Patients receiving point-of-care viseoelastic testing in the setting of postpartum hemorrhage have shown fewer transfusions of platelets, FFP, and red blood cells along with lower incidence of postoperative ICU admissions and cesarean hysterectomy.[58] Other retrospective cohort studies have shown that in PPH, TEG provides rapid detection of hypofibrinogenemia and thrombocytopenia to allow rapid identification of clotting disorders. Observational studies show point-of-care viseoelastic testing can reduce the amount of FFP given during postpartum hemorrhage without hemostatic impairment.[59] A barrier to usage has been cited as lack of larger studies and few randomized controlled trials in PPH. Newer systematic reviews clarify that there is a growing body of literature in this area for both TEG and ROTEM to guide transfusions as well as clarify the hypercoabulable changes after pregnancy.[60]

The understanding of coagulation and coagulopathy has been a major accomplishment over the last century, and TEG is a subject with growing interest in the medical literature. The contributions of surgical and anesthesia physicians in this field have shown that TEG provides information regarding the coagulation profile that routine coagulation studies cannot and transfusions of fresh frozen plasma, cryoprecipitate, and platelets may be avoided in many patients. The volume of research with nonsurgical or nontrauma patients is still limited. An area of interest is assessing the coagulation profile of cirrhotic patients where prolonged prothrombin time does correlate with severity of liver disease but does not independently predict bleeding risk. Larger studies are needed to determine whether TEG may be used in other populations with coagulopathy.

Questions:

1. Prothrombin time, PT, is strongly correlated with reaction time, R, in patients with cirrhosis. True or False.

   Answer: False. Prothrombin time in cirrhosis is not an accurate measure of hypocoagulability in liver failure. Elevated PT is an indicator of the degree of synthetic dysfunction in liver disease, but patients may be hyper or hypocoagulable. There was no significant correlation of PT with R time in cirrhotic patients.[61]
2. A 20-year-old male had hemophilia A, factor VIII deficiency, and no other medical history. He currently takes no medical therapy. He has not had any recent bleeding, and the cell count and differential appear normal. True or false, one or more TEG parameters would likely be abnormal in this patient.

   Answer: True. R, or reaction time, measures the time from activation of the coagulation cascade to the initiation of fibrin crosslinking and clot formation. In hemophilia, the deficiency in Factor VIII will delay the coagulation cascade, and Factor VII activity has a correlation with R time.[62]
3. Tranexamic acid administration would be expected to reverse abnormal maximum amplitude in a coagulopathic patient. True or False?

   Answer: False. Tranexamic acid (TXA) and ϵ-aminocaproic acid (EACA) inhibit the activation of plasminogen to plasmin, thereby inhibiting fibrinolysis. The lysis at 30 min, Ly30, measures the amount of fibrinolysis based on the percent of clot strength lost 30 min after the maximum amplitude is achieved. TXA would be expected to decrease Ly30. Any effects on MA would be unpredictable.
4. Prior to undergoing procedures, cirrhotic patients transfused to correct INR received more blood products than those transfused to correct TEG parameters. True of False?

   Answer: True. In a trial by De Pietri, cirrhotic patients undergoing invasive procedures transfused to INR < 1.8 were more often transfused and had more blood product volume compared to those transfused to a goal R time < 40 min and MA >30 mm.[43] There were no differences between the groups in terms of complications.
5. TEG-guided transfusions in cirrhotic patients with nonvariceal upper GI leads to better mortality versus standard of care. True or False?

   Answer: False. The only current study in cirrhotic patients with upper GI bleeds by Kumar in India found that decreased blood products were transfused with TEG-guided therapy.[20] Complications, including failure to control bleeding, rebleeding, and mortality, were

similar between the groups. The populations with evidence at this time showing the superiority of TEG-guided transfusion strategies regarding improved mortality are cardiac surgery and trauma surgery.

Suggestions for further reading:

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Acknowledgments

We would like to thank Dr Shaheen Islam, Dr Rabih Bechara, Dr Thomas Dillard, and Dr Nicola Zetola in the Division of Pulmonary, Critical Care, and Sleep Medicine at the Medical College of Georgia for their review of the manuscript and support.

Author Contribution(s)

TP Whitton: Conceptualization; Methodology; Writing – original draft; Writing – review & editing.
WJ Healy: Conceptualization; Methodology; Resources; Supervision; Writing – original draft; Writing – review & editing.

Competing Interests

Dr Healy and Dr Whitton have no direct or indirect conflicts of interest to disclose.

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