SPECIAL SECTION: CROSS-SECTIONAL INTERVENTIONS

Thromboelastography: a review for radiologists and implications on periprocedural bleeding risk

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Abstract

Thromboelastography (TEG) and rotational thromboelastometry are emerging technologies that are gaining increasing acceptance in the medical field to evaluate the coagulation status of patients on an individual level by assessing dynamic clot formation. TEG has been proven to reduce blood product use as well as improve patient outcomes in a variety of medical settings, including trauma and surgery due to the expedited nature of the test as well as the ability to determine specific deficiencies present in whole blood that are otherwise undetectable with traditional coagulation studies. Currently, no guidelines or recommendations are in place for the utilization of TEG in interventional or diagnostic radiology although access to TEG has become increasingly common in recent years. This manuscript presents a review of prior literature on the technical aspects of TEG as well as its use in various fields and explains the normal TEG-tracing parameters. Common hemodynamic abnormalities and their effect on the TEG tracing are illustrated, and the appropriate treatments for each abnormality are briefly mentioned. TEG has the potential to be a useful tool for determining the hemodynamic state of patients in both interventional and diagnostic radiology, and further research is needed to determine the value of these tests in the periprocedural setting.

Keywords Thromboelastography · Hemorrhage · Biopsy · Interventional radiology

Introduction

Safely performing minimally invasive image-guided interventions on patients receiving anticoagulation and antiplatelet therapy require meticulous and dynamic management to minimize bleeding and coagulopathy-related adverse outcomes in the periprocedural period. There is a wide range of different bleeding disorders, compounded by specific clinical settings (i.e., cirrhosis, heart disease, renal failure), each having its own set of nuances for specific periprocedural management guidelines. Criteria for determining periprocedural strategies in these patients during image-guided procedures are well described in the Society for Interventional Radiology Consensus Guidelines for the Periprocedural Management of Thrombotic and Bleeding Risk in Patients Undergoing Percutaneous Image-Guided Interventions—Parts I and II published in 2019 (SIR 2019) [1, 2]. Traditional laboratory values used to assess bleeding risk are prothrombin time (PT), activated partial thromboplastin time (PTT), and International Normalized Ratio (INR), along with secondary tests such as thrombin time, fibrinogen assay, D-Dimer, and platelet count. However, despite not necessarily a component of routine laboratory testing, thromboelastography (TEG) and rotational thromboelastometry (ROTEM) have emerged as useful tools to quickly assess bleeding and/or thrombotic risk and etiology in numerous different clinical and periprocedural scenarios. As a result, the European Society of Anaesthesiology (ESA) has begun recommending the use of TEG as a means of identifying and treating bleeding in the perioperative setting with additional recommendations being made by the Eastern Association for the Surgery of Trauma regarding TEG use in trauma surgery with emerging evidence of benefits in cardiovascular and transplant surgeries [3–7].

TEG and ROTEM are viscoelastic tests that provide a unique, comprehensive assessment of the integrity of a patient's coagulation system. These tests measure the

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dynamic process of clot formation in its entirety, taking into account not only the presence of the necessary clotting components for thrombus formation such as plasma, clotting factors, platelets, and blood cells but also the availability and functionality of these components using methods that more closely mimic in vivo clot formation when compared to legacy blood tests. TEG uses an oscillating cylindrical container of whole blood in which a small pin is submersed to process real-time viscoelastic properties of clot formation and clot lysis via a torsion wire (Table 1). ROTEM evolved from TEG and differs in that the suspended pin oscillates instead of the container of blood [8]. While many authors have advocated the benefits of TEG compared to traditional coagulation tests in a variety of clinical settings [8–11], neither TEG nor ROTEM has been validated to assess bleeding risk in non-bleeding patients to guide blood component therapy, and their value in the preprocedural workup for an interventional radiologic procedure is not yet known [12–14]. As these tools continue gaining attention as viable indicators of coagulation status, radiologists who perform image-guided procedures would benefit from having knowledge of the current and potential applications of TEG and ROTEM. The purpose of this review is to provide an overview of TEG including the benefits of TEG when compared to legacy blood tests, the current use-cases of TEG, and a brief explanation of how the specific parameters of the test are generated. A normal TEG tracing as well as common abnormal tracings will also be explained, and the viability of adding TEG to the field of radiology will be discussed.

Benefits of TEG compared to other coagulation parameters

Traditional coagulation studies such as PT, aPTT, INR, platelet count, fibrinogen concentration, D-Dimer, and bleeding time are useful for the clinical diagnosis of a coagulopathy or potential thrombotic state, as well as monitoring anticoagulation therapy, and treating episodes of bleeding. However, these tests do have limitations, such as increased turnaround time in the setting of acute bleeding, and their inability to provide a complete picture of hemostasis due to their lack of adequate representation of certain coagulation factors (i.e., Factor XIII), platelet function, and the fibrinolytic system. For instance, platelet function is influenced by substances such as non-steroidal anti-inflammatory drugs (NSAIDs), antiplatelet medicines, and alcohol, as well as disease states such as malignancy and uremia. Testing for platelet concentration, which is part of a routine complete blood count, does not assess platelet function. TEG, however, does give a visual and numeric representation of platelet function by quantifying the kinetics and strength of

clot formation itself which reflects not only platelet quantity abnormalities but also problems with platelet function.
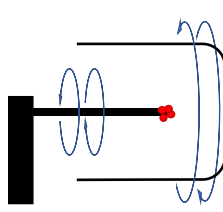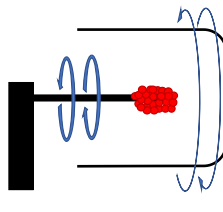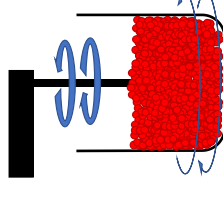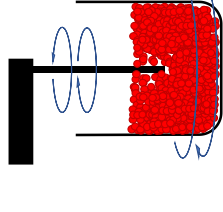
One particularly significant difference between traditional coagulation studies and TEG is in the setting of liver disease. Since most of the coagulation factors assessed with PT, aPTT, INR, etc. are synthesized in the liver, these coagulation studies often have abnormal results. Recent revisions of the 2019 SIR guidelines have included parameters for patients with chronic liver disease that reflects the rebalanced coagulation system in these patients [2]. However, in patients with chronic liver disease, TEG offers additional information about the exact hemostatic condition of each of these patients and may provide more useful information over standard preprocedural laboratory tests [15]. Further benefits of TEG include the ability to monitor clot fibrinolysis directly. In general, other coagulation assays are unable to adequately represent clot lysis [16]. As a result, TEG is unique in its ability to measure a state of hyperfibrinolysis which has been associated with increased mortality but can be treated when identified in a timely manner [17]. Another key difference between traditional coagulation tests and TEG is the turnaround time to receive test results. For instance, turnaround time for PT, INR, aPTT, and platelet count is approximately 45 min to 1 h in a standard hospital laboratory due to logistic restraints. Comparatively, TEG can be measured in real-time with actionable data generated in as little as 5 min in some cases [18].

Current applications of TEG

Due to expedited nature of TEG analysis, TEG is most commonly utilized in settings where hemorrhagic blood loss is common such as cardiothoracic surgery, postpartum hemorrhage, and trauma [19–21]. In addition to the speed in which the test results can be obtained, TEG results are able to guide blood product selection. TEG has been associated with reduced volume of transfusion of various blood products including red blood cells (RBCs), fresh frozen plasma (FFP), and platelets in addition to other benefits including decreased bleeding volume, increased cost savings, lower mortality rates, and reduced reoperation rates [4, 21–23].

As TEG has become more widely available, the technology has been implemented in a variety of areas. In the critical care setting, TEG has been used to predict thrombotic risk and transfusion requirements [24]. For oncologic patients with septic shock, TEG has been shown to be predictive of venous thromboembolism (VTE) due to its ability to generate real-time assessment of in vitro clot formation [25]. In the assessment of critically ill neonates, TEG has been shown to be predictive of bleeding events which could lead to more optimized blood product utilization which is complicated by the dynamic hemostatic status present in

Table 1 TEG-tracing parameters and causes of abnormal tracings

	Start (T0)	R	K & α	MA	Ly30
	Cup heated to 37°C; oscillates left to right for 4°45' at a rate of 6 cycles per minute	Time from T0 to 2 mm amplitude	K: Time from R to 20 mm amplitude α-angle: angle from midline of tracing to intersection of line at amplitude = 20 mm	Maximum amplitude (MA) of TEG curve corresponds to time of maximal amplitude (TMA)	Lysis of blood indicated by the percentage change in amplitude from MA to amplitude at 30 min after TMA (measures fibrinolysis)
Measured coagulation factor		Clotting factors and anticoagulants	Fibrinogen	Platelets	Fibrinolysis
Causes of abnormalities		Increased: anticoagulants, hypocoagulable state Decreased: hypercoagulable state, DIC	Increased K & Decreased α-angle: thrombocytopenia, platelet inhibitor, hypocoagulable state, low fibrinogen Decreased K and Increased α-angle: hypercoagulable state, DIC	Increased: hypercoagulable state, DIC Decreased: fibrinolysis, thrombocytopenia, platelet inhibitor	Increased: fibrinolysis, DIC
Treatment		Increased: Fresh Frozen Plasma, Reversal of Anticoagulants Decreased: treat underlying cause	Increased: Cryoprecipitate Decreased: treat underlying cause	Increased: treat underlying cause Decreased: Platelets or DDAVP	Increased: Tranexamic Acid, Aminocaproic Acid, other lysine analogs

DIC disseminated intravascular coagulation, *DDAVP* 1-deamino-8-D-arginine vasopressin

critically ill neonates [26]. With some cases of COVID-19 leading to coagulopathies in a subset of patients, TEG has also been used to predict thrombotic complications and to guide patient management in this patient cohort [27, 28].

Interpretation of TEG and implications on bleeding risk

The interpretation of TEG is typically dependent on the measurement of five values, reaction time (R), clot kinetics (K), clot strengthening (α-angle), maximum strength of the clot which corresponds to the maximum amplitude (MA), and percentage of clot lysis 30 min after maximum amplitude (Ly30) [29]. Importantly, values may fluctuate based on manufacturer and/or reagent used for the assay [22]. To ensure adequate interpretation, the normal limits for the particular TEG used must be established. An example of a normal TEG graph with corresponding labels is shown in Table 1.

Thrombus formation occurs via a complex, multistep process that includes three phases: initiation, amplification, and propagation [30]. The initiation phase involves a multitude of coagulation factors interacting via tissue factor which leads to the amplification phase, defined by the activation of platelets and other coagulation factors. Ultimately, the activation of factor X on the platelet surface begins the propagation phase where a complex of factor X and V collectively convert factor II (prothrombin) to thrombin. The newly formed thrombin subsequently acts on fibrinogen to form fibrin which is the component of the thrombus which provides additional structural integrity to the clot by crosslinking the platelets, forming a fibrin-platelet mesh. As illustrated in Table 1, the TEG tracing reflects the integrity of these processes in whole blood.

The TEG tracing is generated by submersing a pin coupled to a torsion wire into a cup of whole blood after which various reagents are added to cause clot formation [8, 9]. The cup of whole blood is rotated about its central axis in an oscillating motion from 4° to 45°, while the pin initially remains still. As the clot begins to form on both the cup and the pin, the pin begins to move with the cup as the clot strengthens which is recorded by a torsion wire connected to the pin.

The time from when the reagent is added until the amplitude of the pin rotating about its axis has reached 2 mm is recorded as the reaction time (R) [22]. The reaction time corresponds to the formation of the platelet plug which is dependent upon the availability and functionality of coagulation factors as well as various enzymatic reactions require for thrombus formation. Typical R values range from 4 to 10 min depending on manufacturer and reagent used for the specific TEG system. A prolonged R value may indicate low factor levels or may indicate that the individual has an inhibitor present which can disrupt thrombus formation. Other conditions that can cause prolonged R values include patients on warfarin, as well as those with hemophilia, although there have been mixed findings associated with the R value and its reliability with warfarin assessment, showing no correlation with INR but has been shown to be useful with treating patients on unfractionated heparin [29, 31–33]. Treatment for a prolonged R time is typically fresh frozen plasma (FFP) which contains a high concentration of all essential coagulation factors required for clot formation [33, 34].

After the initial formation of the platelet plug, amplification occurs. The TEG tracing reflects this amplification via two measurements, the kinetic parameter (K) and the α-angle. The K value is recorded as the time that elapses between reaching 2 mm of amplitude and 20 mm of amplitude [8]. The K value is most commonly representative of the availability of fibrinogen to facilitate thrombus formation [29]. The α-angle is a measurement of the angle from midline of the graph to the tangent of the curve at an amplitude of 20 mm. A steeper angle indicates a hypercoagulable state while an angle below normal limits is reflective of thrombocytopenia or hypofibrinogenemia. Factor deficiencies and thrombocytopenia can also increase the K value and lower the α-angle as a result of slowed clot proliferation [22]. In general, a low K or α-angle can be corrected with fibrinogen or cryoprecipitate which contains a high concentration of fibrinogen [33, 35].

The maximum amplitude (MA) of the graph is also measured from the TEG tracing and reflects the strength of the clot which is dependent on a multitude of factors including fibrinogen levels, platelet count, and platelet function. The MA is increased in hypercoagulable states and decreased in thrombocytopenia, platelet dysfunction, and hypofibrinogenemia. Treatment for elevated MA revolves around treating the underlying condition while transfusion of platelets or administration of desmopressin (DDAVP) is typically the preferred treatment for decreased MA [29].

After reaching the MA, the adhesion between the clot and the pin begins to breakdown, allowing the pin to slip within the clot, reducing the rotation of the pin which in turn results in a reduction in the amplitude of the TEG tracing [29]. The percentage of lysis at 30 min (Ly30) is measured as the percent change in amplitude of the TEG tracing 30 min after reaching the MA. The Ly30 reflects fibrinolysis of the clot which is mediated by plasmin. Some TEG tracings also record the percentage of amplitude lost at 60 min (Ly60), but many TEG tracings do not run to completion as the utility of the test is gained through the aforementioned parameters which occur early within the testing process [22]. States of hyperfibrinolysis indicated by an increased Ly30 can be treated with tranexamic acid or antifibrinolytic agents [36].

Table 2 Schematic of common TEG tracings with associated parameters

Pathology	Tracings	R	K	α	MA	Ly30
Normal		–	–	–	–	–
Hypercoagulable state		↓	↓	↑	↑	–
Hypocoagulable state		↑	↑	↓	↓	↑
Antiplatelet pharmaceutical		↑	↑	↓	–	–
DIC (early)		↓	↓	↑	↑	↑
DIC (late)		↑	↑	↓	↓	↑
Thrombocytopenia		–	↑	↓	↓	–
Fibrinolysis		–	–	–	↓	↑
Low fibrinogen		–	↑	↓	–	–

Figures not to scale and do not represent a TEG tracing in its entirety but are meant to accurately depict expected features of actual tracings

DIC disseminated intravascular coagulation

Coagulation index (CI), a comprehensive indicator of the hemodynamics of the blood, can be calculated using the parameters produced from the TEG [29]. The normal values for the CI range between − 3.0 and + 3.0, which represents three standard deviations from the mean of zero. A commonly used formula for calculating that the CI is shown below which includes four parameters from the TEG tracing (R, K, MA, and α-angle). Values greater than + 3.0 or less than − 3.0 indicate a hypercoagulable or coagulopathic state, respectively. While effective as a single numerical value that provides an insight into the hemodynamic properties of the patient, CI is seldomly used in practice, and as a result, the significance of the CI remains largely unvalidated.

Examples of a normal TEG tracing as well as some common abnormalities are illustrated in Table 2. While some electronic health record (EHR) systems provide only the values of the aforementioned numerical parameters from a TEG tracing rather than the tracing itself, some basic knowledge of a normal TEG tracing and common abnormal variants may be beneficial for the rapid identification of hemostatic abnormalities both in interventional and diagnostic radiology. The ability to identify the differences between hypercoagulable and hypocoagulable states based on the tracing may

be sufficient for the vast majority of applications in radiology; however, other common etiologies are also presented.

Conclusions and future directions

Given the knowledge gap among radiologists who may have been trained prior to TEG becoming a more prevalent hemodynamic assessment tool, continued awareness, and education on the topic is essential to providing safe and effective patient care. As TEG continues to be validated in various settings, further research is needed in its potential applications in image-guided procedures. Given the relative novelty of TEG and its role in hemodynamic assessment, tools that enable radiologists to consider and interpret that TEG results may facilitate a more widespread and rapid acceptance. Potential future iterations of periprocedural anticoagulation guidelines, including future revisions to SIR guidelines, may consider adding additional recommendations for the incorporation of TEG into perioperative and transfusion management.

Declarations

References

1. Davidson JC, Rahim S, Hanks SE, Patel IJ, Tam AL, Walker TG, et al. Society of Interventional Radiology Consensus Guidelines for the Periprocedural Management of Thrombotic and Bleeding Risk in Patients Undergoing Percutaneous Image-Guided Interventions-Part I: Review of Anticoagulation Agents and Clinical Considerations: Endorsed by the Canadian Association for Interventional Radiology and the Cardiovascular and Interventional Radiological Society of Europe. J Vasc Interv Radiol. 2019;30(8):1155-67. https://doi.org/10.1016/j.jvir.2019.04.016.
2. Patel IJ, Rahim S, Davidson JC, Hanks SE, Tam AL, Walker TG, et al. Society of Interventional Radiology Consensus Guidelines for the Periprocedural Management of Thrombotic and Bleeding Risk in Patients Undergoing Percutaneous Image-Guided Interventions-Part II: Recommendations: Endorsed by the Canadian Association for Interventional Radiology and the Cardiovascular and Interventional Radiological Society of Europe. J Vasc Interv Radiol. 2019;30(8):1168-84.e1. https://doi.org/10.1016/j.jvir.2019.04.017.
3. Kozek-Langenecker SA, Ahmed AB, Afshari A, Albaladejo P, Aldecoa C, Barauskas G, et al. Management of severe perioperative bleeding: guidelines from the European Society of Anaesthesiology: First update 2016. Eur J Anaesthesiol. 2017;34(6):332-95. https://doi.org/10.1097/EJA.0000000000000630.
4. Bugaev N, Como JJ, Golani G, Freeman JJ, Sawhney JS, Vatsaas CJ, et al. Thromboelastography and rotational thromboelastometry in bleeding patients with coagulopathy: Practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2020;89(6):999-1017. https://doi.org/10.1097/TA.0000000000002944.
5. Meco M, Montisci A, Giustiniano E, Greco M, Pappalardo F, Mammana L, et al. Viscoelastic Blood Tests Use in Adult Cardiac Surgery: Meta-Analysis, Meta-Regression, and Trial Sequential Analysis. J Cardiothorac Vasc Anesth. 2020;34(1):119-27. https://doi.org/10.1053/j.jvca.2019.06.030.
6. Fleming K, Redfern RE, March RL, Bobulski N, Kuehne M, Chen JT, et al. TEG-Directed Transfusion in Complex Cardiac Surgery: Impact on Blood Product Usage. J Extra Corpor Technol. 2017;49(4):283-90.
7. Lawson PJ, Moore HB, Moore EE, Stettler GR, Pshak TJ, Kam I, et al. Preoperative thrombelastography maximum amplitude predicts massive transfusion in liver transplantation. J Surg Res. 2017;220:171-5. https://doi.org/10.1016/j.jss.2017.05.115.
8. Whiting D, DiNardo JA. TEG and ROTEM: technology and clinical applications. Am J Hematol. 2014;89(2):228-32. https://doi.org/10.1002/ajh.23599.
9. Mallett SV. Clinical Utility of Viscoelastic Tests of Coagulation (TEG/ROTEM) in Patients with Liver Disease and during Liver Transplantation. Semin Thromb Hemost. 2015;41(5):527-37. https://doi.org/10.1055/s-0035-1550434.
10. Afshari A, Wikkelso A, Brok J, Moller AM, Wetterslev J. Thrombelastography (TEG) or thromboelastometry (ROTEM) to monitor haemotherapy versus usual care in patients with massive transfusion. Cochrane Database Syst Rev. 2011(3):CD007871. https://doi.org/10.1002/14651858.CD007871.pub2.
11. Baksaas-Aasen K, Van Dieren S, Balvers K, Juffermans NP, Naess PA, Rourke C, et al. Data-driven Development of ROTEM and TEG Algorithms for the Management of Trauma Hemorrhage: A Prospective Observational Multicenter Study. Ann Surg. 2019;270(6):1178-85. https://doi.org/10.1097/SLA.0000000000002825.
12. De Pietri L, Bianchini M, Montalti R, De Maria N, Di Maira T, Begliomini B, et al. Thrombelastography-guided blood product use before invasive procedures in cirrhosis with severe coagulopathy: A randomized, controlled trial. Hepatology. 2016;63(2):566-73. https://doi.org/10.1002/hep.28148.
13. Adler M, Ivic S, Bodmer NS, Ten Cate H, Bachmann LM, Wuillemin WA, et al. Thromboelastometry and Thrombelastography Analysis under Normal Physiological Conditions - Systematic Review. Transfus Med Hemother. 2017;44(2):78-83. https://doi.org/10.1159/000464297.
14. Chitlur M, Lusher J. Standardization of thromboelastography: values and challenges. Semin Thromb Hemost. 2010;36(7):707-11. https://doi.org/10.1055/s-0030-1265287.
15. Lisman T, Porte RJ. Rebalanced hemostasis in patients with liver disease: evidence and clinical consequences. Blood. 2010;116(6):878-85. https://doi.org/10.1182/blood-2010-02-261891.
16. Levrat A, Gros A, Rugeri L, Inaba K, Floccard B, Negrier C, et al. Evaluation of rotation thrombelastography for the diagnosis of hyperfibrinolysis in trauma patients. Br J Anaesth. 2008;100(6):792-7. https://doi.org/10.1093/bja/aen083.
17. Raza I, Davenport R, Rourke C, Platton S, Manson J, Spoors C, et al. The incidence and magnitude of fibrinolytic activation in trauma patients. J Thromb Haemost. 2013;11(2):307-14. https://doi.org/10.1111/jth.12078.
18. Barrett CD, Moore HB, Vigneshwar N, Dhara S, Chandler J, Chapman MP, et al. Plasmin thrombelastography rapidly identifies trauma patients at risk for massive transfusion, mortality, and hyperfibrinolysis: A diagnostic tool to resolve an international debate on tranexamic acid? J Trauma Acute Care Surg. 2020;89(6):991-8. https://doi.org/10.1097/TA.0000000000002941.

19. Wikkelso A, Wetterslev J, Moller AM, Afshari A. Thromboelastography (TEG) or rotational thromboelastometry (ROTEM) to monitor haemostatic treatment in bleeding patients: a systematic review with meta-analysis and trial sequential analysis. Anaesthesia. 2017;72(4):519-31. https://doi.org/10.1111/anae.13765.

20. Fahrendorff M, Oliveri RS, Johansson PI. The use of viscoelastic haemostatic assays in goal-directing treatment with allogeneic blood products - A systematic review and meta-analysis. Scand J Trauma Resusc Emerg Med. 2017;25(1):39. https://doi.org/10.1186/s13049-017-0378-9.

21. Wikkelso A, Wetterslev J, Moller AM, Afshari A. Thromboelastography (TEG) or thromboelastometry (ROTEM) to monitor haemostatic treatment versus usual care in adults or children with bleeding. Cochrane Database Syst Rev. 2016;8):CD007871. https://doi.org/10.1002/14651858.CD007871.pub3.

22. Schmidt AE, Israel AK, Refaai MA. The Utility of Thromboelastography to Guide Blood Product Transfusion. Am J Clin Pathol. 2019;152(4):407-22. https://doi.org/10.1093/ajcp/aqz074.

23. Bolliger D, Tanaka KA. Point-of-Care Coagulation Testing in Cardiac Surgery. Semin Thromb Hemost. 2017;43(4):386-96. https://doi.org/10.1055/s-0037-1599153.

24. Harahsheh Y, Duff OC, Ho KM. Thromboelastography Predicts Thromboembolism in Critically Ill Coagulopathic Patients. Crit Care Med. 2019;47(6):826-32. https://doi.org/10.1097/CCM.0000000000003730.

25. Liu J, Wang N, Chen Y, Lu R, Ye X. Thrombelastography coagulation index may be a predictor of venous thromboembolism in gynecological oncology patients. J Obstet Gynaecol Res. 2017;43(1):202-10. https://doi.org/10.1111/jog.13154.

26. Katsaras G, Sokou R, Tsantes AG, Piovani D, Bonovas S, Konstantinidi A, et al. The use of thromboelastography (TEG) and rotational thromboelastometry (ROTEM) in neonates: a systematic review. Eur J Pediatr. 2021;180(12):3455-70. https://doi.org/10.1007/s00431-021-04154-4.

27. Hartmann J, Ergang A, Mason D, Dias JD. The Role of TEG Analysis in Patients with COVID-19-Associated Coagulopathy: A Systematic Review. Diagnostics (Basel). 2021;11(2). https://doi.org/10.3390/diagnostics11020172.

28. Laubscher GJ, Lourens PJ, Venter C, Kell DB, Pretorius E. TEG((R)), Microclot and Platelet Mapping for Guiding Early Management of Severe COVID-19 Coagulopathy. J Clin Med. 2021;10(22). https://doi.org/10.3390/jcm10225381.

29. Shaydakov ME, Sigmon DF, Blebea J. Thromboelastography. StatPearls. Treasure Island (FL)2022.

30. Hoffman M, Monroe DM, 3rd. A cell-based model of hemostasis. Thromb Haemost. 2001;85(6):958-65.

31. Dunham CM, Rabel C, Hileman BM, Schiraldi J, Chance EA, Shima MT, et al. TEG(R) and RapidTEG(R) are unreliable for detecting warfarin-coagulopathy: a prospective cohort study. Thromb J. 2014;12(1):4. https://doi.org/10.1186/1477-9560-12-4.

32. Schmidt DE, Holmstrom M, Majeed A, Naslin D, Wallen H, Agren A. Detection of elevated INR by thromboelastometry and thromboelastography in warfarin treated patients and healthy controls. Thromb Res. 2015;135(5):1007-11. https://doi.org/10.1016/j.thromres.2015.02.022.

33. Walsh M, Fritz S, Hake D, Son M, Greve S, Jbara M, et al. Targeted Thromboelastographic (TEG) Blood Component and Pharmacologic Hemostatic Therapy in Traumatic and Acquired Coagulopathy. Curr Drug Targets. 2016;17(8):954-70. https://doi.org/10.2174/1389450117666160310153211.

34. Hashim YM, Dhillon NK, Rottler NP, Ghoulian J, Barmparas G, Ley EJ. Correcting Coagulopathy With Fresh Frozen Plasma in the Surgical Intensive Care Unit: How Much Do We Need to Transfuse? Am Surg. 2021:31348211023412. https://doi.org/10.1177/00031348211023412.

35. Harr JN, Moore EE, Ghasabyan A, Chin TL, Sauaia A, Banerjee A, et al. Functional fibrinogen assay indicates that fibrinogen is critical in correcting abnormal clot strength following trauma. Shock. 2013;39(1):45-9. https://doi.org/10.1097/SHK.0b013e3182787122.

36. Chapman MP, Moore EE, Ramos CR, Ghasabyan A, Harr JN, Chin TL, et al. Fibrinolysis greater than 3% is the critical value for initiation of antifibrinolytic therapy. J Trauma Acute Care Surg. 2013;75(6):961-7; discussion 7. https://doi.org/10.1097/TA.0b013e3182aa9c9f.