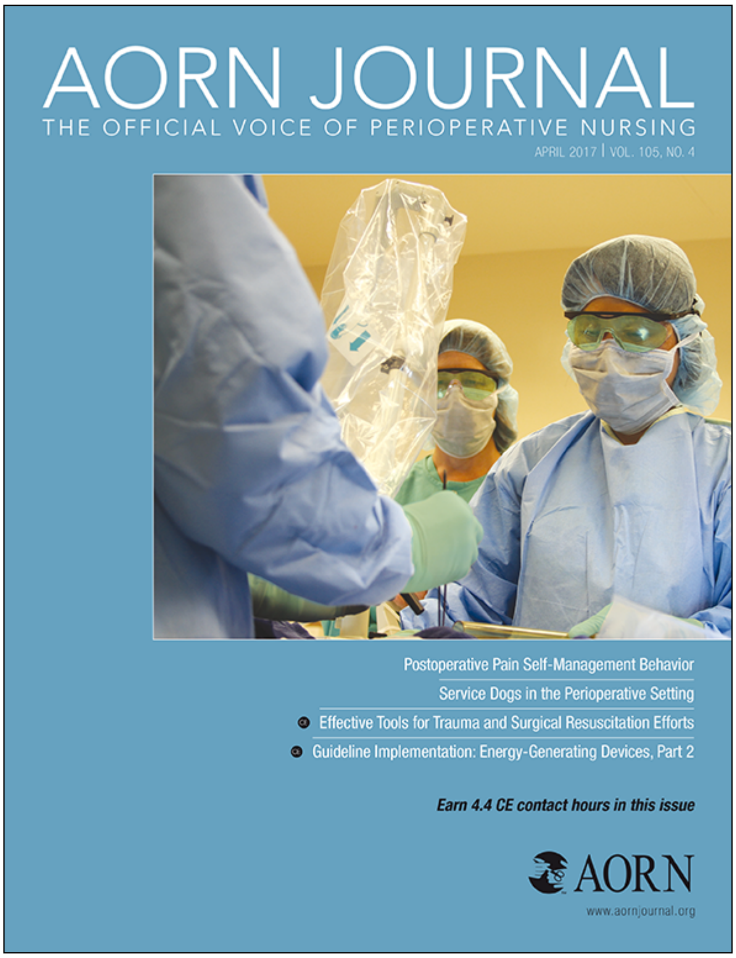
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To provide the learner with knowledge of best practices related to the use of viscoelastic studies, such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM), to manage patients with trauma-induced coagulopathy (TIC) in the OR.

Objectives

1. Discuss the importance of effectively managing hemorrhage and TIC in the OR.
2. Describe the pathophysiology of TIC.
3. Explain the benefits of using TEG and ROTEM technology in the OR.
4. Compare and contrast TEG and ROTEM technology.

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The behavioral objectives for this program were created by Kristi Van Anderson, BSN, RN, CNOR, clinical editor, with consultation from Susan Bakewell, MS, RN-BC, director, Perioperative Education. Ms Van Anderson and Ms Bakewell have no declared affiliations that could be perceived as posing potential conflicts of interest in the publication of this article.

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Viscoelastic Studies: Effective Tools for Trauma and Surgical Resuscitation Efforts 2.1 CE www.aornjournal.org/content/cme

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ABSTRACT

Trauma-induced coagulopathy (TIC) is an abrupt disruption of all hemostatic components of coagulation resulting from severe tissue injury and hypoperfusion. The effective management of TIC has remained elusive to clinicians using traditional laboratory methods, challenging efforts to improve outcomes related to uncontrolled bleeding. Recent initiatives have aimed to reduce TIC-associated morbidity and mortality, further invoking trauma experts to explore innovative modalities in the field of viscoelastic studies, such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM). These tests are able to guide proper blood product administration more effectively during trauma and surgical resuscitation compared with conventional laboratory tests. Although TEG and ROTEM are similar tests, inherent differences in their features produce variation in output results. This article calls on the perioperative clinician to evaluate TEG and ROTEM tests and consider their implementation based on the benefits of their application to clinical practice. *AORN J* 105 *(April 2017) 370-383. Published by Elsevier, Inc, on behalf of AORN, Inc. http://dx.doi.org/10.1016/j.aorn.2017.01.013*

Key words: *viscoelastic studies, trauma-induced coagulopathy, thromboelastography, rotational thromboelastometry, massive transfusion.*

In the United States, trauma accounts for 41 million visits to the emergency department and an average of 2.3 million hospital admissions per year. Of these, approximately 192,000 traumatic injuries result in death,[1] and 30% to 40% of deaths from trauma can be attributed to hemorrhage.[2] Approximately one-quarter to one-third of trauma patients also exhibit trauma-induced coagulopathy (TIC), making them approximately eight times more likely to die within a 24-hour period.[3] Trauma-induced coagulopathy is an abrupt disruption of all hemostatic components of coagulation resulting from severe tissue injury and hypoperfusion.[4] Despite enhanced clinical awareness, hemorrhage continues to be the number-one preventable cause of death after injury[5] and remains a major complication during and after surgery.[6] This situation necessitates that traditional practices in the management of TIC undergo a clinical process transformation.

Traditional laboratory studies that assess coagulation, such as prothrombin time, international normalized ratio, partial thromboplastin time, and activated partial thromboplastin time, inaccurately describe the complex nature of TIC[7] and are limited in their ability to measure clot strength.[8] Clot strength is the best measure of hemostatic capacity and represents the interaction between platelets and fibrinogen, both of which are determinants of stable clot formation.[9] Viscoelastic point-of-care studies, such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM), assess the tensile force of whole blood to provide a quick estimate of thrombin generation, platelet function, and clot

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dissolution by fibrinolysis.[10] Viscoelastic studies are most commonly used in emergency and OR settings[11] and are considered critical to improving trauma resuscitation efforts because of their unparalleled capabilities for providing clot strength feedback in real time. The aim of this article is to introduce the perioperative clinician to two state-of-the-art technologies with the potential to improve patient outcomes in the OR through early detection and treatment of TIC.

RECENT INITIATIVES AND A CALL TO ACTION

A multidisciplinary team of trauma experts developed the STOP the Bleeding Campaign,[12] which specifies a set of guidelines addressing traumatic coagulopathies that aim to reduce mortality rates stemming from exsanguination within the first 24 hours of hospitalization.[13] The premise behind the STOP the Bleeding Campaign is to actively search, treat, observe, and prevent (STOP). The clinician should

- search for patients at risk for bleeding,
- treat patients who are bleeding,
- observe the response to treatment, and
- prevent secondary bleeding and subsequent coagulopathies.

Proponents of the STOP the Bleeding Campaign encourage clinicians to embrace evidence-based principles for the management of exsanguination.[13]

An additional driving force supporting improvements in hemostatic interventions is damage control resuscitation (DCR). The DCR paradigm has been disseminated throughout the trauma community and emphasizes the importance of the early control of hemorrhage and limiting ongoing blood loss by refining blood-transfusion strategies. It provides a systematic approach for managing a bleeding patient from the prehospital setting through the OR and intensive care unit.[14] Both the STOP and DCR initiatives prompt clinicians to augment their traditional practices with cutting-edge methods that have the potential to mitigate the consequences of TIC.

At the 2016 John Pryor Memorial PennTRAC Conference, which is hosted by the University of Pennsylvania and focuses on trauma and critical care topics,[15] trauma experts concluded that there is still much work to be done in control of exsanguination. Raising awareness was one of the primary objectives of this conference, but actively advocating for more effective and efficient modalities that reduce traumatic coagulopathies was the backbone of the discussion. Bleeding-related complications and blood transfusions exhaust many hospital services and resources[5] and, on a greater scale, have serious implications related to length of stay and total hospital costs. Patients with bleeding-related complications spend an average of 2.7 more days in the intensive care unit and incur costs between $2,805 and $17,279 higher than patients without bleeding-related complications, depending on the type of surgery. Trauma surgery costs related to bleeding have been estimated to be as high as $38,628 per patient compared with other routine procedures.[16]

The translation of evidence-based findings requires both a multidisciplinary approach and solicitation of clinicians who are versed in catalyzing positive change. Efforts to address bleeding-associated consequences align well with AORN's value of promoting cutting-edge innovations to enhance safety and optimal outcomes for surgical patients.[17] Given the role that perioperative nurses play in influencing hemostatic practices in the OR, they make excellent agents for championing initiatives that reduce complications and decrease costs related to intraoperative bleeding.[18]

MECHANISMS OF TIC

The pathways of coagulation are complex; however, it is essential that perioperative clinicians gain a basic understanding of the pathophysiologic characteristics of TIC. Literature findings indicate that a prothrombin time ratio greater than 1.2 is suggestive of coagulopathy and is often used as a marker for detection of TIC.[4,19-21] Trauma-induced coagulopathy is characterized by massive thrombin formation, fibrinogen and platelet depletion, and fibrinolysis.[4,19,21]

Massive Thrombin Generation

Trauma-induced coagulopathy follows the orchestrated clotting processes of the extrinsic pathway of the coagulation cascade (Figure 1).[22] Each step is sequential and interdependent. After external injury, the subendothelial lining exposes tissue factor to factor VII, generating an activated complex that subsequently activates factor X (the major protein responsible for the conversion of prothrombin to thrombin). Thrombin, "the master regulator of the clotting cascade,"[23] is responsible for strengthening fibrin clots, enhancing platelet aggregation, activating factors V and VIII through positive feedback, and accelerating protein C activity 1,000-fold.[4] The effect that massive thrombin generation has on the coagulation process is rather perplexing. Davenport[21] suggests that under conditions of hypoperfusion (ie, hypoxia, acidosis, hypothermia), thrombin may be diverted from fibrin generation to produce activated protein C, which aids in premature anticoagulation, becoming a major precipitant to development of TIC.

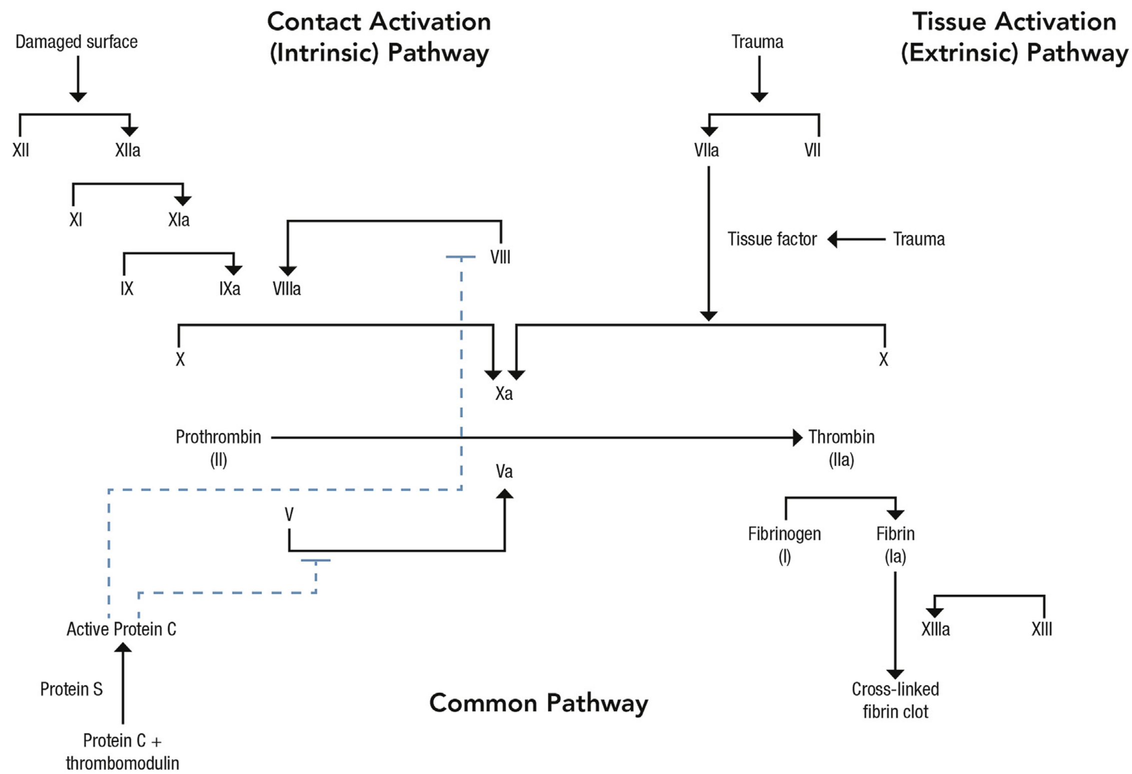

Figure 1. The coagulation cascade. *Adapted from Coagulation. MedRevise. http://medrevise.co.uk/index.php?title=Coagulation. Updated June 1, 2010. Accessed November 22, 2016.*

Depletion of Platelets and Fibrinogen

The exact pathophysiology of fibrinogen and platelet depletion in TIC remains unclear;[4,19,21] however, research implicates the activation of protein C as the primary source for coagulopathies in severe trauma.[4,21] The exponential increase in protein C activity during massive thrombin generation reduces fibrin generation through the inhibition of clotting factors V and VIII and platelet aggregation through the inhibition of clotting factors V and X.[4,21] As a consequence, TIC becomes more profound because of limited fibrin clot substrate and platelet-binding capacity.[5]

Activation of Fibrinolysis

Researchers have hypothesized that the activation of fibrinolysis stems from the consumption of plasminogen activator inhibitor-1 by protein C. Under this hypothesis, diminished quantities of plasminogen activator-1 enhance the unopposed fibrinolytic processes of tissue plasminogen activator, the protein that is involved in breaking down blood clots.[4] This process is further exacerbated when surges of epinephrine, vasopressin, and thrombin trigger the additional release of tissue plasminogen activator after tissue injury.[19] The CRASH-2 (Clinical Randomisation of an Antifibrinolytic in Significant Haemorrhage) trial validates the detrimental effects of fibrinolysis in trauma by demonstrating a significant reduction in overall mortality in adult trauma patients with the administration of tranexamic acid, an inhibitor of plasminogen conversion to plasmin.[24]

Managing TIC rests on the clinician's ability to identify and correct the disruptions that occur in specific pathways of the clotting cascade and to do so promptly. Traditional laboratory coagulation studies (eg, prothrombin time, international normalized ratio) are useful in measuring clotting-factor deficiencies; however, they are unable to detect massive thrombin generation, the contribution of platelets to hemostasis, or the evolution of fibrinolysis.[19] Although the mechanisms of TIC remain elusive to clinicians using traditional laboratory methods, they may capture its derivatives with TEG and ROTEM technology.

DISSEMINATED INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation is similar to TIC and is often covered in the trauma literature. It is characterized by a consumption of platelets and coagulation factors that result from the activation of tissue-factor–dependent pathways and incompetent anticoagulant mechanisms. Trauma, hypoxia, ischemia, inflammation, and endothelial cell damage can all lead to the release of tissue factors. Trauma-induced coagulopathy and disseminated intravascular coagulation share many similarities; however, they differ in that protein C levels surge exponentially in TIC, whereas they remain low in disseminated intravascular coagulation. Despite the subtle difference, both conditions can lead to severe bleeding and could be managed adequately with viscoelastic studies.[25]

VISCOELASTIC STUDIES

Improving management of TIC relies on the rapid quantification and qualitative depiction of coagulation processes. The expeditious and multifaceted assessment of hemostatic processes by point-of-care viscoelastic studies has increased in popularity throughout trauma and surgical settings around the world.[26] Robust transfusion protocols that allow for early hemostatic resuscitation are vital to the management of TIC and may be attributed to reductions in rates of mortality as high as 26%.[27] It is in early recognition and management of TIC that viscoelastic studies have earned their place as effective tools in trauma and surgical resuscitation efforts.

At present, two viscoelastic products dominate the market: TEG 5000[28] and ROTEM.[29] In the United States, clinicians use TEG primarily, but ROTEM is more often used in Europe and Canada.[10] The primary hardware difference between the two technologies concerns the movement of the cup (which holds the whole blood sample) and the sensor pin (which measures clot strength). The TEG cup rotates while the pin is suspended freely in the cup, whereas the ROTEM pin rotates while the cup remains stationary. However, both tests use controlled, low-shear movements to analyze whole blood and capture changes in torque to transduce them into a visual tracing that represents clot strength.[28,30-32] The sensor pin measures the tensile force produced by the interaction of activated platelet receptors and polymerizing fibrin during generation and fibrinolysis of thrombin.[11] Although the mechanical principles of TEG and ROTEM are similar, the hardware and activators or inhibitors of each produce different output values and reference ranges with results that cannot be used interchangeably.[10]

TEG AND ROTEM INTERCHANGEABILITY

The interchangeability of TEG and ROTEM results is limited because of the heterogeneity of their process methods. Each assay contains a variety of activators and inhibitors that produce variations in TEG and ROTEM graphic tracings. These dissimilarities are generally responsible for the differences in their treatment algorithms.[33] The use of different reagents to complete TEG (eg, tissue factor kaolin, sodium citrate)[34] and ROTEM (eg, recombinant tissue factor, cytochalasin)[35] testing derives from the two distinct models of hemostasis used to develop the two technologies. Analysis using TEG is centered on a cell-based model of hemostasis, whereas ROTEM is based on an intrinsic and extrinsic pathway model.[36] Although different nomenclatures are used for interpretation of each test, the parameters for interpreting TEG and ROTEM tracings are essentially the same. Both products analyze

- clotting time (ie, time to reach a 2-mm clot amplitude),
- clot kinetics (ie, time from a 2-mm to 20-mm clot amplitude),
- alpha angle (ie, the slope between clotting time and clot kinetics),
- amplitude (at a fixed time),
- maximum clot strength, and
- clot lysis (at a fixed time).[37]

Respectively, the nomenclatures used to interpret TEG and ROTEM tracings are reaction time and clotting time in the clotting time phase, kinetics and clot formation time in the clot kinetics phase, maximum amplitude and maximum clot firmness in the maximum clot strength phase, and lysis and lysis index in the clot lysis phase (Figure 2).[33] Trained clinicians performing trauma and surgical resuscitation can interpret these results to capitalize on individualized, goal-directed coagulation therapy (Table 1).[7]

Before acquiring either TEG or ROTEM equipment, perioperative leaders should compare both TEG and ROTEM parameters thoroughly for similarities in the diagnosis, transfusion guidance, and prognosis associated with TIC. A 2012 systematic review evaluating 25 quantitative studies suggested that maximum amplitude (TEG) and maximum clot firmness (ROTEM) measurements are statistically similar in their measurement and association with platelet counts, partial thromboplastin time, blood transfusion, and mortality. In addition, similarities were identified between TEG clot lysis and ROTEM maximum lysis in diagnosing excessive fibrinolysis and mortality.[37]

Despite limited interchangeability between TEG and ROTEM results, both methodologies may be independently useful in transfusion guidance and in the early diagnosis of TIC, specifically related to hypocoagulability, hypercoagulability, platelet dysfunction, and hyperfibrinolysis.[38] One could argue that TEG and ROTEM systems are analogous to Macintosh versus Microsoft Windows personal computers; they offer the same utility but with the use of different software and, to some extent, language. Table 2 summarizes the pros and cons of each system.[39]

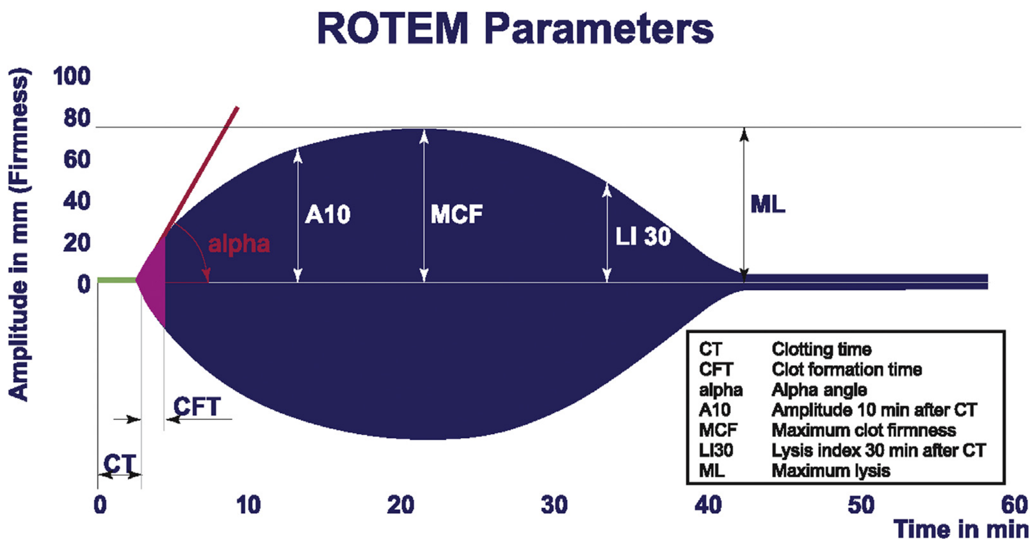

Figure 2. Parameters for interpreting ROTEM tracings. Parameters for interpreting TEG tracings are similar; however, nomenclature varies between the two systems. TEG = thromboelastography; ROTEM = rotational thromboelastometry. *Adapted with permission from TEM Systems, Inc, Durham, NC.* ***Editor's notes:*** *For Figures 2 through 4, TEG is a registered trademark of the Haemoscope Corporation, Braintree, MA. ROTEM is a registered trademark of CA Casyso AG c/o Caspar Stürm, Muttenz, Switzerland.*

APPLICATION TO CLINICAL PRACTICE

Balanced administration of blood products is the core of current resuscitation strategies; it reduces the severity of dilutional coagulopathy by preventing unnecessary fresh frozen plasma (FFP), platelet (PLT), and packed red blood cell (PRBC) transfusions. Although research has demonstrated improved outcomes with the implementation of massive transfusion protocols, they are often initiated by providers after massive amounts of PRBCs have already been infused.[14] Clinicians have developed scoring systems to lessen these effects; however, they often require time-consuming and complicated algorithms.[40] Recent studies have supported the

Table 1. TEG and ROTEM Parameters for Guiding Transfusion

	TEG	ROTEM	Clinical Utility
Time from test initiation to 2 mm above baseline	R	CT	Prolongation may indicate a deficiency of coagulation factors or presence of anticoagulants
Time from 2 mm above baseline to 20 mm above baseline	K	CFT	Representative of the kinetics of clot formation; can be early indicators of clot deficiency or hypercoagulability
Alpha angle	α	α	Prolongation suggests platelet dysfunction or deficiency and fibrinogen deficiencies; shortening may indicate hypercoagulability
Amplitude at time X	A30, A60, etc	A5, A10, etc	Clot strength at given time during the analysis; values at these times are often used as "transfusion triggers"
Clot lysis at time X	LY30, LY60	LI30, LI45, LI60, etc	Indication of fibrinolysis and potential need for antifibrinolytics

TEG = thromboelastography; ROTEM = rotational thromboelastometry; R = reaction time; CT = clotting time; K = kinetics; CFT = clot formation time; A = amplitude; LY = lysis; LI = lysis index.

Editor's notes: *For Tables 1 through 3, TEG is a registered trademark of the Haemoscope Corporation, Braintree, MA. ROTEM is a registered trademark of CA Casyso AG c/o Caspar Stürm, Muttenz, Switzerland.*

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Table 2. Pros and Cons of TEG and ROTEM[1]

	TEG	ROTEM
Pros	• Less expensive than ROTEM • Small and lightweight • Extensively researched • Uses Microsoft Windows software • Detects effects of low-molecular-weight heparin	• Screen monitor and processor are integrated • More resistant to vibration than TEG • Automatic pipetting • Easy step-by-step on-screen instructions • Can run up to 4 assays at one time
Cons	• Can only run 2 assays at one time • Requires manual pipetting • Results may be affected by vibration or physical disturbance such as bumping, natural disasters, or combat environments susceptible to blast effects	• Bulky appearance • Uses Linux software, which may be unfamiliar to users • Cannot examine the effect of low-molecular-weight heparin

TEG = thromboelastography; ROTEM = rotational thromboelastometry.
Editor's notes: *Microsoft Windows is a registered trademark of Microsoft Corporation, Redmond, WA. Linux is a registered trademark of Linus Torvalds, The Linux Foundation, San Francisco, CA.*
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1. *Jackson GN, Ashpole KJ, Yentis SM. The TEG vs the ROTEM thromboelastography/thromboelastometry systems.* Anaesthesia. *2009;64(2): 212-215.*

transfusion ratio of 1:1:1 (FFP:PLT:PRBC), but statistical significance in preventing mortality compared with other resuscitation strategies has not yet been established.[5,14] This lack of a significant relationship between massive transfusion protocols and mortality has shifted expert attention toward goal-directed therapies that may be individualized to the patient's actual needs,[7] thus improving the overall chance of survival.[41]

It is with goal-directed therapies that TEG and ROTEM have their greatest value. Using goal-directed therapies, clinicians can use the results of viscoelastic studies to help determine which blood products are most appropriate to administer to an individual patient rather than administering blood products based on a fixed ratio of FFP:PLT:PRBC.[7] For example, if TEG or ROTEM results indicate that clotting time is prolonged, the trained clinician who is using a TEG- or ROTEM-based algorithm knows to administer FFP; if the alpha angle is too low when interpreting TEG tracings, the trained clinician knows to administer cryoprecipitate (Figure 3).[5,42] One study compared the use of a TEG-guided massive transfusion protocol to one of conventional means (ie, one based on traditional laboratory values) in a randomized clinical trial that included 111 trauma patients; results demonstrated a survival advantage when using TEG compared with conventional means (19% versus 36% mortality, respectively).[5] Similar protocols have also been developed for ROTEM-guided massive transfusion protocols.[42]

Both TEG and ROTEM meet the criterion for use as point-of-care tests in the United States, according to the Clinical Laboratory Improvement Amendments of 1988 database.[43] Meeting this criterion eliminates the need for analysis by specialized laboratory technicians and allows expeditious testing and interpretation by trained clinicians. The 2014 consensus panel on viscoelastic test–based transfusion guidelines for early trauma resuscitation concluded that the optimal location for equipment setup would depend on local factors such as infrastructure and human resources.[10] Members of the panel emphasized, however, that settings such as the OR and emergency department should be considered as processing stations because of the staff members' demonstrated proficiency with point-of-care tests and closed-loop communication with clinical teams.[25]

CASE STUDY

The following case study demonstrates how viscoelastic studies could be used effectively during trauma and surgical resuscitation efforts. Clinicians at a single facility would generally use either TEG or ROTEM for clotting analysis. However, for the purposes of this case study, the use of both TEG and ROTEM technology is demonstrated.

A 34-year-old man arrived in the emergency department after suffering multiple gunshot wounds to his bilateral lower extremities. The emergency team found the patient in profound shock secondary to uncontrolled bleeding. Keeping DCR principles in mind, the team limited administration of fluids to prevent dilutional coagulopathy and instead focused on control of hemorrhage and hemostatic resuscitation. Based on the hospital's viscoelastic testing protocol, the team drew blood for an additional coagulation test tube, along with the

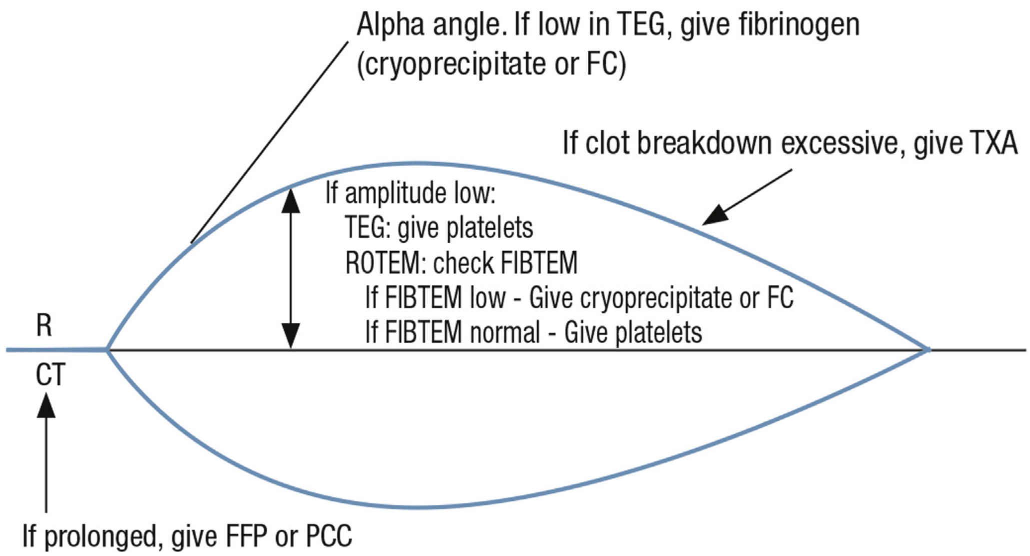

Figure 3. General blood product transfusion guidance based on graphic tracing from TEG or ROTEM analysis. R = reaction time; CT = clotting time; FFP = fresh frozen plasma; PCC = prothrombin complex concentrate; FC = fibrinogen concentrate; TXA = tranexamic acid; FIBTEM = ROTEM assay that assesses fibrinogen status. *Adapted from Abdelfattah K, Cripps MW. Thromboelastography and rotational thromboelastometry use in trauma.* Int J Surg. *2016;33(pt B):196-201, with permission from Elsevier Ltd, Amsterdam, the Netherlands. © 2015 IJS Publishing Group Ltd, Ilford, UK.*

rest of the laboratory studies, and it was immediately processed using TEG and ROTEM for guided therapy. Results were available within 15 minutes of processing time. The graphic tracings displayed a decreased alpha angle in TEG and a reduced maximum clot firmness amplitude in ROTEM; these tracings, in combination with the quantitative results in Table 3, were most characteristic of platelet inhibition and hypofibrinogenemia.

Platelet Inhibition and Hypofibrinogenemia

The patient was transferred to the OR immediately, with direct pressure on his wounds, and was prepared for application of arterioarterial shunts and fixation of bilateral femurs. Based on the results obtained from TEG and ROTEM, the team decided to replace fibrinogen levels by administering cryoprecipitate.

One hour after administering cryoprecipitate, the perioperative team performed follow-up TEG and ROTEM tests in the OR. All values were then found to be within the reference range, with the exception of a prolonged reaction time (52 seconds) for TEG and clotting time (96 seconds) for ROTEM, which was indicative of anticoagulation or factor deficiency. The team attributed these follow-up results to a possible dilutional effect caused by overresuscitation with crystalloid fluids and decided to transfuse the patient with FFP according to protocol. One hour later, the TEG- and ROTEM-guided therapy produced a normal graphic tracing (Figure 4) and quantitative values.

COST AND IMPLEMENTATION OF TEG OR ROTEM

There are subtle mechanical differences between TEG and ROTEM; however, their cost savings and clinical effectiveness are similarly superior to conventional methods. A systematic review comparing the cost-effectiveness of viscoelastic studies with traditional laboratory studies demonstrated a savings of £721 (approximately $916) and £688 (approximately $874) per patient when using TEG and ROTEM, respectively.[32] Similarly, a systematic review conducted in Scotland demonstrated significant annual cost savings in both liver and cardiac surgery totaling £344,183 (approximately $424,790) and £95,507 (approximately $121,401), respectively, when using thromboelastography and thromboelastometry methods compared with standard laboratory tests.[44] These outcomes may be attributed to a more rapid identification of coagulopathies, more prudent use of blood products, and improved prediction of massive transfusion protocol requirements.[42]

Regardless of which viscoelastic study is selected for use, implementation will require a multifaceted approach by

Table 3. Quantitative TEG and ROTEM Values Associated With Case Study

TEG Test		ROTEM Test	
Tissue Factor Kaolin[a]	Results (Reference Range[1])	EXTEM[b]	Results (Reference Range[2])
R	35 s (17-38 s)	CT	76 s (43-82 s)
K	*27 s* (30-118 s)	CFT	*42 s* (48-127 s)
Alpha angle	*55 degrees* (66-82 degrees)	Alpha angle	*48 degrees* (65-80 degrees)
MA	60 mm (54-72 mm)	MCF	*31 mm* (52-70 mm)
		FIBTEM[b]	**Results (Reference Range[3])**
		CT	*185 s* (not determined)
		MCF	*3 mm* (7-24 mm)

NOTE. Values outside the reference range are indicated in italics.

TEG = thromboelastography; ROTEM = rotational thromboelastometry; R = reaction time; K = kinetics; MA = maximum amplitude; CT = clotting time; CFT = clot formation time; MCF = maximum clot firmness.

[a] Tissue factor kaolin is the active reagent used for the TEG test for the purposes of this case study. Multiple TEG active reagents exist; reference ranges vary depending on the active reagent used.[1]

[b] EXTEM is the ROTEM assay that assesses extrinsic pathway clot formation, fibrin polymerization, and fibrinolysis. FIBTEM is the ROTEM assay that assesses fibrinogen status. Multiple ROTEM assays exist; EXTEM and FIBTEM were chosen for the purposes of this case study. Reference ranges vary depending on the assay used.[4]

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2. *Instructions for use: ex-tem. Tem Innovations GmbH.* https://www.rotem-usa.com/wp-content/uploads/2015/12/ex-tem-VER-0015-USA.pdf. *Accessed December 16, 2016.*
3. *Instructions for use: fib-tem. Tem Innovations GmbH.* https://www.rotem-usa.com/wp-content/uploads/2015/12/fib-tem-VER-0014-USA.pdf. *Accessed December 16, 2016.*
4. *Reagents. ROTEM.* https://www.rotem-usa.com/products/reagents/. *Accessed December 16, 2016.*

members of the perioperative, trauma, and critical-care communities. Perioperative nurses often encounter situations that warrant the use of viscoelastic studies. This makes the perioperative department the ideal environment in which to advocate for use of TEG and ROTEM. The process could begin by introducing the innovative topic at various organizational leadership meetings involving directors of surgical services, medical services, nursing services, laboratory services, and administration. Perioperative clinicians may become actively involved in piloting quality improvement initiatives and in developing evidence-based protocols in collaboration with emergency care providers, surgeons, anesthesia care providers, intensivists, perfusionists, and laboratory scientists.

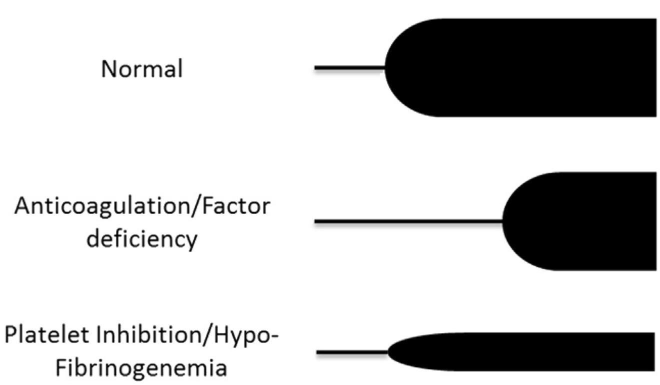

Figure 4. Graphic tracings of coagulation status that are associated with the case study presented. Shapes of graphic tracings are similar whether the clinician uses TEG or ROTEM to analyze coagulation. *Adapted from Abdelfattah K, Cripps MW. Thromboelastography and rotational thromboelastometry use in trauma.* Int J Surg. *2016;33(pt B):196-201, with permission from Elsevier Ltd, Amsterdam, the Netherlands. © 2015 IJS Publishing Group Ltd, Ilford, UK.*

CONCLUSION

Despite recent advances in resuscitative strategies, the effective management of TIC is restricted by the prolonged turnaround time and the limited clot strength assessment of traditional laboratory studies. Point-of-care TEG and ROTEM testing may better guide the transfusion of blood products for trauma patients by providing both quantitative and qualitative feedback in real time, thus avoiding consequences associated with empiric treatment (eg, lung injury, circulation overload, infections).[45]

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Continuing Education: Viscoelastic Studies: Effective Tools for Trauma and Surgical Resuscitation Efforts 2.1 CE www.aornjournal.org/content/cme

PURPOSE/GOAL

To provide the learner with knowledge of best practices related to the use of viscoelastic studies, such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM), to manage patients with trauma-induced coagulopathy (TIC) in the OR.

OBJECTIVES

1. Discuss the importance of effectively managing hemorrhage and TIC in the OR.
2. Describe the pathophysiology of TIC.
3. Explain the benefits of using TEG and ROTEM technology in the OR.
4. Compare and contrast TEG and ROTEM technology.

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QUESTIONS

1. Approximately one-quarter to one-third of trauma patients exhibit _______, making them approximately eight times more likely to die within a 24-hour period.
   a. disseminated intravascular coagulation
   b. trauma-induced coagulopathy
   c. myocardial infarction
   d. traumatic brain injury

2. Viscoelastic studies are considered critical to improving trauma resuscitation efforts because of their unparalleled capabilities for providing clot strength feedback in real time.
   a. true
   b. false

3. Patients with bleeding-related complications spend an average of _______ more days in the intensive care unit than patients without bleeding-related complications.
   a. 2.7
   b. 3.2
   c. 3
   d. 2.4

4. Trauma-induced coagulopathy is characterized by
   1. platelet depletion.
   2. massive thrombin formation.
   3. fibrinogen depletion.
   4. fibrinolysis.

   a. 1 and 3
   b. 2 and 4
   c. 1, 2, and 4
   d. 1, 2, 3, and 4

5. Research implicates the activation of ________ as the primary source for coagulopathies in severe trauma.
   a. fibrinogen
   b. thrombin
   c. protein C
   d. tissue factor

6. The primary hardware difference between TEG and ROTEM technologies concerns the movement of the _______ and the _______.
   a. sensor pin; screen
   b. cup; scanner
   c. cup; sensor pin
   d. screen; scanner

7. The parameters for interpreting TEG and ROTEM tracings are essentially the same. Both products analyze
   1. clot kinetics.
   2. alpha angle.
   3. clotting time.
   4. maximum clot strength.
   5. clot lysis.
   6. sample viscosity.

   a. 1, 3, and 4 b. 2, 4, and 5
   c. 1, 2, 3, 4, and 5 d. 1, 2, 3, 4, 5, and 6

8. Both TEG and ROTEM are useful in transfusion guidance and in the early diagnosis of TIC, specifically related to
   1. hypercoagulability.
   2. hypocoagulability.
   3. platelet dysfunction.
   4. hyperfibrinolysis.

   a. 1 and 3 b. 2 and 4
   c. 1, 2, and 4 d. 1, 2, 3, and 4

9. Using goal-directed therapies, clinicians can use the results of viscoelastic studies to help determine which blood products are most appropriate to administer to an individual patient rather than administering blood products based on a fixed ratio of FFP:PLT: PRBC.

   a. true b. false

10. Both TEG and ROTEM are
    1. interpreted by trained clinicians.
    2. only analyzed by specialized laboratory technicians.
    3. point-of-care tests.

    a. 1 and 2 b. 1 and 3
    c. 2 and 3 d. 1, 2, and 3

LEARNER EVALUATION

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OBJECTIVES

To what extent were the following objectives of this continuing education program achieved?

1. Discuss the importance of effectively managing hemorrhage and TIC in the OR.
   Low 1. 2. 3. 4. 5. High

2. Describe the pathophysiology of TIC.
   Low 1. 2. 3. 4. 5. High

3. Explain the benefits of using TEG and ROTEM technology in the OR.
   Low 1. 2. 3. 4. 5. High

4. Compare and contrast TEG and ROTEM technology.
   Low 1. 2. 3. 4. 5. High

CONTENT

5. To what extent did this article increase your knowledge of the subject matter?
   Low 1. 2. 3. 4. 5. High

6. To what extent were your individual objectives met?
   Low 1. 2. 3. 4. 5. High

7. Will you be able to use the information from this article in your work setting?
   1. Yes 2. No

8. Will you change your practice as a result of reading this article? (If yes, answer question #8A. If no, answer question #8B.)

8A. How will you change your practice? (*Select all that apply*)
   1. I will provide education to my team regarding why change is needed.
   2. I will work with management to change/implement a policy and procedure.
   3. I will plan an informational meeting with physicians to seek their input and acceptance of the need for change.
   4. I will implement change and evaluate the effect of the change at regular intervals until the change is incorporated as best practice.
   5. Other: ________________________________

8B. If you will not change your practice as a result of reading this article, why? (*Select all that apply*)
   1. The content of the article is not relevant to my practice.
   2. I do not have enough time to teach others about the purpose of the needed change.
   3. I do not have management support to make a change.
   4. Other: ________________________________