AANA Journal Course

Update for Nurse Anesthetists

Thromboelastography: Clinical Application, Interpretation, and Transfusion Management

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The coagulation cascade is a dynamic process dependent on many factors. It involves interaction between primary hemostasis, platelet clot formation, secondary hemostasis, thrombin generation, and fibrinolysis. The assessment of this process is particularly important in the surgical patient to properly manage hemostatic issues. Traditionally, coagulation tests used to guide transfusion management have included platelet count, activated partial thromboplastin time, prothrombin time, international normalized ratio, and activated clotting time, among others. Although these tests provide the practitioner with valuable information, they lack the ability to measure platelet function. The ability to measure whole blood coagulation, including platelet function, and not just the number of platelets, can be critical when a healthcare provider is determining what products are appropriate for a particular patient during surgery. One possible solution to this deficit in traditional coagulation monitoring is thromboelastography. Thromboelastography provides a more complete picture of coagulation status, taking into account more factors involved in the clotting process, including platelet function and temperature.

**Objectives**
At the completion of this course, the reader should be able to:
1. Identify current clinical applications of thromboelastography.
2. Interpret a thromboelastography tracing.
3. Compare thromboelastography parameters to common coagulation profiles.
4. Identify basic transfusion management based on a thromboelastography tracing.

**Introduction**
The coagulation cascade is a dynamic process dependent on many factors. It involves interaction between primary hemostasis, platelet clot formation, secondary hemostasis, thrombin generation, and fibrinolysis. The assessment of this process is particularly important in the surgical patient to properly assess patient coagulation assessment, manage hemostatic therapy and transfusion in trauma and perioperative care, and assess bleeding in hemophilic patients. Complex surgical patients may require targeted hemostatic therapy using blood products and hemostasis-altering medications. Traditionally, coagulation tests used to guide transfusion management have included platelet count, activated partial thromboplastin time (aPTT), prothrombin time (PT), international normalized ratio, and activated clotting time (ACT), among others.

Although these tests provide the practitioner with valuable information, they lack the ability to measure platelet function. However, some argue that screening for coagulation abnormalities and application of hemostatic interventions based on classical coagulation tests such as PT and aPTT are of limited value in perioperative and acutely ill patients. Also, the ability to measure whole blood coagulation, including platelet function, and not just the number of platelets, can be critical when a healthcare provider is determining what products are appropriate for a particular patient during surgery, or promptly determining when a patient might need to return to surgery for surgical hemostasis when a thromboelastography (TEG) assay is normal.

One possible solution to this deficit in traditional co-

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agulation monitoring is TEG. Monitoring with TEG provides a global assessment of hemostatic function and clot formation. Dr. Helmut Hartet initially developed TEG in 1947 for use specifically in research; more recently, it has become a tool to properly identify and treat coagulation abnormalities. Thromboelastography provides a more complete picture of coagulation status, taking into account more factors involved in the clotting process, including platelet function and temperature.

Because TEG is particularly sensitive to changes in fibrin polymerization and platelet count, it is most useful for early detection of trauma and surgery related dilutional coagulopathy in which plasma fibrinogen and platelets fall rapidly. In addition, TEG is valuable in guiding the use of cryoprecipitate or purified fibrinogen concentrate and potentially platelet transfusion. Watson et al. write: “Using TEG, clinicians may be able to optimize targeted transfusion therapies with specific coagulation factor(s) instead of empirically administering multiple components with potentially hazardous effects.”

Thromboelastography Methods
The 2 main components of the TEG machine are a cup and a pin. Whole blood is mixed with the activating agent kaolin as well as calcium. The cup then oscillates around the pin slowly, at a rate of 6 times per minute, to mimic natural blood flow in vivo and activate the clotting cascade. As the clot forms, the torque between the cup and pin is transduced and measured, creating a curve (Figure). As the clot breaks down and torque decreases, the tracing converges to represent this. The different parameters of the curve are then measured to assess current coagulation status.

Initial results from TEG are available within minutes and include the reaction time, or R time, which is the time to initial clot formation, and the α angle or K value, which represents clot kinetics. It may take as long as 30 to 60 minutes until all TEG values are obtained. One study demonstrated that early rapid TEG values (k time and r value) are available within 5 minutes, late rapid TEG values (maximum amplitude [MA] and α angle) within 15 minutes, and conventional coagulation testing within 48 minutes (P < .001). The ACT, r value, and k time showed strong correlation with PT, international normalized ratio, and partial thromboplastin time (all r > 0.70; P < .001), whereas MA (r = -0.49) and α angle (r = 0.40) correlated with platelet count (both P < .001). Normal values based on the activator used for the test can be found in Table 1.

Thromboelastography Interpretation
Of the 4 types of TEG assays available, the most common is the rapid TEG, and it is the assay referred to in this review. The use of an activator in rapid TEG standardizes the TEG test and speeds up the rate at which clotting takes place, thus making results available more quickly.

The first measurement of note is the reaction time (R time). This is the time interval from the start of the test to the initial detection of the clot (see Figure). This value is roughly equivalent to the PT that provides a measurement of the extrinsic clotting pathway and the aPTT that provides a measurement of the intrinsic clotting pathway in standard coagulation assays. Similarly, the R time provides information about clotting factor deficiencies or possible heparin therapy. A major advantage of TEG over the aPTT is the ability to add the reagent heparinase to monitor patients currently receiving heparin therapy for other possible clotting deficiencies. Samples with TEG are taken concurrently; one is exposed to heparinase and the other is not. Comparisons of the TEG measurements alert the provider to possible clotting factor deficiencies or residual heparin.

The clot strength is measured by 2 variables in TEG. The K value represents clot kinetics and measures the interval between the R time and the time when the clot reaches 20 mm. The α angle is another measurement of clot kinetics and measures a line tangent to the slope of the curve during clot formation (see Figure). Both these parameters depend mostly on fibrinogen levels. This can help identify states of hyper- or hypocoagulopathies (Table 2).

Maximum amplitude is a measurement of maximum clot strength and provides information on both fibrinogen and platelet function (see Figure). Current coagulation profiles include platelet count but generally do not take into account platelet function. Disruptions in the endothelium cause the exposure of collagen and von Willebrand factor. Platelets then adhere to these and release substances that cause platelet aggregation. Fibrinogen then binds to glycoprotein IIb/IIIa receptors, causing further platelet aggregation and formation of a platelet plug. Platelet function is an integral part of clot formation during surgery and an alteration in function
can disrupt the ultimate formation of a clot.4,8

The last major TEG parameter is the LY30, which measures the percent of clot lysis 30 minutes after the MA is achieved (see Figure). This measurement is most useful for patients undergoing thrombolytic drug therapy or during more advanced stages of disseminated intravascular coagulation. This can be observed by rapid curve convergence.4,8

Thromboelastography curves represent the coagulation status at the time of blood draw. During changes in coagulation, such as during active hemorrhage or periods of massive transfusion, this status can change rapidly. Thromboelastography offers the ability to monitor multiple consecutive samples all at one time.4,8

**Thromboelastography Clinical Applications**

- **Cardiopulmonary Bypass.** Extracorporeal circulation during cardiopulmonary bypass in the surgical patient has long been known to cause coagulation disturbances.12 Cardiopulmonary bypass disrupts the hemostatic system in a number of ways, including hemodilution of procoagulants and platelets, a reduction in coagulation factors due to the interaction of blood with the surface of the bypass circuit, the use of heparin, and altered temperature.12 Hemodilution of clotting components occurs mainly due to the large amount of crystalloid and colloid solutions used to prime the bypass circuit.12 Exposure of heparinized circulating blood to the bypass circuit as well as the surgical wound activates both the intrinsic and extrinsic clotting cascade, triggering a prothrombotic reaction. Heparin is administered in large doses, but it cannot prevent this thrombin formation; heparin can only reduce the thrombin after it has already been produced. This constant formation of thrombin leads to what is called a consumptive coagulopathy due to the exhaustion of clotting factors.11 Hypothermia is known to cause multiple coagulation abnormalities leading to increased bleeding. Low body temperature blocks thromboxane synthesis, which results in decreased platelet aggregation.13

Traditionally, coagulation is monitored using the ACT

<table>
<thead>
<tr>
<th>Test (activator)</th>
<th>R (s)</th>
<th>K (s)</th>
<th>Angle α (°)</th>
<th>MA (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid TEG (tissue activator)</td>
<td>78-110</td>
<td>30-120</td>
<td>68-82</td>
<td>54-72</td>
</tr>
<tr>
<td>kaoTEG (kaolin)</td>
<td>180-480</td>
<td>60-180</td>
<td>55-78</td>
<td>51-69</td>
</tr>
</tbody>
</table>

**Table 1. Normal Thromboelastography Values Based on Activator Used**

Abbreviations: MA, maximum amplitude; TEG, thromboelastography.

<table>
<thead>
<tr>
<th>Coagulation status</th>
<th>TEG tracing</th>
<th>R value</th>
<th>K and α value</th>
<th>Maximum amplitude</th>
<th>Treatment algorithm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal hemostasis</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Attain surgical hemostasis using sutures</td>
</tr>
<tr>
<td>Hemodilution or clotting factor deficiency</td>
<td>High</td>
<td>Low or normal</td>
<td>Low or normal</td>
<td>Administer fresh frozen plasma if indicated</td>
<td></td>
</tr>
<tr>
<td>Fibrinogen deficiency</td>
<td>Normal or high</td>
<td>Low</td>
<td>Low or normal</td>
<td>Administer cryoprecipitate</td>
<td></td>
</tr>
<tr>
<td>Low or dysfunctional platelets</td>
<td>Normal</td>
<td>Normal</td>
<td>Low</td>
<td>Administer platelets</td>
<td></td>
</tr>
<tr>
<td>Primary fibrinolysis</td>
<td>Normal</td>
<td>Normal</td>
<td>Low</td>
<td>Administer antifibrinolitics or tranexamic acid as indicated</td>
<td></td>
</tr>
<tr>
<td>Secondary fibrinolysis:</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>Treat disseminated intravascular coagulopathy</td>
<td></td>
</tr>
<tr>
<td>hypercoagulopathy with fibrinolysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombosis</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>Administer anticoagulant indicated</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2. Common Clotting Disorders, Thromboelastography (TEG) Tracing Example, Characteristic Values, and Treatments**
throughout cardiopulmonary bypass surgery. Research has demonstrated ACT to be less accurate than TEG or aPTT in this setting.13 Activated clotting time has many disadvantages, including the inability to monitor a true heparin level as well as the impact of temperature and hemodilution on the ACT results.14 The manner in which TEG is monitored is believed to be analogous to in vivo coagulation because it takes into account the temperature as well as the impact of platelets on hemostasis. In addition, TEG is superior to ACT in this setting because of the unique ability to isolate heparin in the TEG sample using heparinase. This allows the practitioner to monitor underlying coagulation issues, not mistaking a bleeding disorder for heparin therapy.15 The use of a TEG-guided algorithm during cardiac surgery has been shown to reduce the number of blood products transfused as well as the number of patients requiring transfusion during cardiac surgery.2

• Liver Surgery. The liver is the major source for the production of clotting factors. Patients with chronic liver disease have defects in coagulation, making them a population requiring targeted transfusion therapy. Generally, it is understood that patients with chronic liver disease have a lack of clotting factors and are therefore hypocoagulable; however, these patients can also exhibit systemic hypercoagulability because of the concurrent lack of natural anticoagulants.16 Because of their clotting issues, patients with chronic liver disease require quick assessment of coagulation abnormalities and targeted use of blood products for treatment of these abnormalities. Thromboelastography provides a more complete assessment of coagulation abnormalities and has been shown to reduce the amount of blood products utilized in this patient population, although it has not been demonstrated that this decrease in blood product utilization has an impact on long-term morbidity and mortality.17 Thromboelastography-guided transfusion therapy has demonstrated applicability not only to liver surgery but also to chronic liver disease, liver cancer, and pancreatic cancer patient populations.18

• Trauma Surgery. Coagulopathy resulting from traumatic injury is a common occurrence requiring rapid intervention. Coagulopathies are associated with a high rate of mortality in the trauma patient; this is especially true within the first 24 hours after the trauma incident.19 There are multiple mechanisms that contribute to the coagulopathies observed in trauma. The first resuscitative efforts after a trauma are generally crystalloid and colloidal fluids that do not contain clotting factors, leading to hemodilutional coagulopathies.20

Another cause of traumatic coagulopathy is rapid consumption of clotting factors similar to the consumption of clotting factors observed in patients undergoing cardiopulmonary bypass. An additional method of coagulopathy involves the activation of protein C due to hypoperfusion from hemorrhage during a traumatic injury. Activated protein C affects coagulation in 2 ways. Protein C inhibits clotting factors V and VIII, decreasing total thrombin formation. It also decreases the inhibition of tissue plasminogen activator, which causes plasminogen to convert to plasmin faster, leading to fibrinolysis.20 This combination rapidly leads to coagulopathy and fibrinolysis following a trauma.20,21

Traditional coagulation tests are of limited value in this population of patients because of the inability to detect clot strength and the length of time required to obtain results. For patients with trauma, TEG offers benefits because of the rapid return of results, the power to measure clot strength, and the ability to monitor the hyperfibrinolysis commonly found in this population.21 A systematic review of the literature found that targeted use of blood products and clotting factors guided by TEG reduced the overall incidence of morbidity and mortality in this population of patients.22

• Obstetrics. Obstetrics is another patient population for whom TEG can improve monitoring of coagulation status. Women exhibit many coagulopathies during pregnancy. These range from the normal physiologic changes causing a hypercoagulable state to the coagulopathy exhibited during hemolysis, elevated liver enzyme levels, and low platelet (HELLP) syndrome.23 The coagulation status of these patients is particularly important for the anesthetist because of the risk of epidural hematoma with neuraxial blockade. Although the incidence of epidural hematoma in the general obstetric population of 1 in 168,000 is relatively low, several case studies and cohort studies suggest a higher incidence in the coagulopathic patient.24,25

Traditionally, the major factor determining the safety of neuraxial anesthesia for the laboring parturient has been the platelet count.20 The platelet count alone is rarely responsible for marked changes in coagulation in this population.27 Thromboelastography is being used successfully to predict morbidity following neuraxial anesthesia. A recent study found that parturients with a low platelet count (56,000 × 10^3/μL), but normal TEG could safely receive a neuraxial anesthetic.28 Additionally, TEG more accurately identifies patients with hypercoagulopathies and can help guide the dosing of low-molecular-weight heparin in these patients.23

Thromboelastography-Guided Transfusion Management

Impaired hemostasis and coagulopathies are known to cause serious conditions associated with morbidity and mortality during major surgery.2,12,17-21 Poorly guided transfusion therapy during massive transfusion protocols can cause or worsen existing coagulopathies.29 In addition to worsening coagulopathies, blood transfusions are associated with transfusion reactions, transfusion-related acute lung injury (TRALI), and transfusion-related immunodilution (TRIM) among many other issues.30 Multiple studies
have found that using a transfusion algorithm based on a TEG tracing decreases the amount of blood product transfused.\(^\text{17,31,32}\) This not only decreases the risks associated with blood transfusions but also decreases total treatment cost and strain on blood bank resources.\(^\text{17,30}\)

An alteration in R time represents alterations in hemostatic clotting factors. If this value is elevated, it can signify a deficiency in clotting factors, hemodilution, or increased endogenous heparin production.\(^\text{31}\) Excessive heparin may be present if R time is prolonged in a normal sample, but may be normal in a sample with heparinase. A prolonged R time in a sample with heparinase may indicate hemodilution or clotting factor deficiencies, and this value could indicate a need for transfusion of fresh frozen plasma.\(^\text{11}\) On the other hand, a shortened R time can indicate a hypercoagulopathy warranting the use of an anticoagulant (see Table 2).\(^\text{20}\)

Alterations in the rate of clot growth, as evidenced by changes in the K value or angle, show the clot growth kinetics. A low value can indicate a deficiency in fibrinogen and may reveal a need for cryoprecipitate. Similarly to the R time, a high value may represent a hypercoagulable state in which an anticoagulant may be applicable (see Table 2).\(^\text{20}\)

The MA value represents the ultimate strength of the clot formed by fibrin and platelet bonding. A low MA value is indicative of low clot strength, which can be caused by decreased fibrinogen levels, low platelet counts, or decreased platelet function.\(^\text{20}\) Paired with a low K value, this could be a sign of the need for cryoprecipitate. This parameter becomes most important when paired with a platelet count. Administration of platelets may be avoided with a low platelet count but normal platelet function as indicated by a normal MA value. Conversely, treatment with platelets may be indicated for patients with a low MA value, or low platelet function, and normal platelet count.\(^\text{11}\) A high MA value may indicate the need for an anticoagulant (see Table 2).\(^\text{20}\)

**Conclusion**

Thromboelastography, although not a new science, is gaining ground as a monitor of coagulation status in many different surgical areas. It provides a quick global assessment of hemostasis more similar to in vivo hemostasis than traditional coagulation profiles.\(^\text{4}\) Rapid interpretation by anesthetists can help identify coagulopathies sooner and guide transfusion management more accurately.\(^\text{2,4,12,17,20,31,32}\) This results in less blood product utilization, which can improve long-term patient outcomes as well as help decrease overall surgical cost.\(^\text{17,31,32}\)

**REFERENCES**


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