NEWS FROM THE PIT

Arizona Poison and Drug Information Center

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Everyone Wins with TEG

By Allison Chiakmakis, BNS, RN, AMB-BC

Hello! My name is Allison Chiakmakis, I am the TEG Clinical Specialist in Arizona and the author for this newsletter. The purpose of this newsletter is purely educational, but I would like to disclose that I am an employee of Haemonetics. This letter does not reflect the opinions of my employer, but more so offers an overview of the TEG system and its advantages. If you would like additional information beyond what is shared in this letter, please contact me directly.

Despite healthcare innovation and advancements over the years, 60,000 Americans still die every year from hemorrhage. Additionally, over 100,000 Americans die each year from blood clots. Why is this?

Blood is complex, and in order to maintain hemostasis, our bodies require an intricate balance between bleeding and clotting. The responsibility of blood is to provide oxygen and nutrients to all our organs, especially the vital ones. The slightest blood imbalance (or deficit) could result in a life-threatening situation leaving the organs starved for blood. So it's safe to say that as healthcare providers, it is important to understand how to manage these critical situations. Interestingly enough, when dealing with an imbalanced hemostatic system, there is not a standardized methodology that all practitioners follow.

NEWSLETTER HIGHLIGHTS

Everyone Wins with TEG

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The Conventional Approach

Everyone is familiar with the traditional plasma-based coagulation tests: the PT/INR and PTT. Plasma based tests evaluate the traditional coagulation cascade, but they do not reflect the contemporary understanding of cell-based coagulation. The PT/INR was created solely to manage patients on Warfarin. The PTT looks at how long it takes for initial blood clot formation. The PTT does not offer any insight as to what follows the initial clot formation, nor does it shed light on thrombotic risk. Platelet counts and fibrinogen levels are utilized to determine a quantitative analysis relative to clot strength. What the blood counts are missing is the ability to qualitatively assess how well those components are functioning. So, the question remains: why are these tests considered the gold standard for managing hemostasis, when that was not what they were intended to do? Nor are these tests capable of fully capturing the complexity of the hemostatic system.

Fortunately, a viscoelastic test has been developed that evaluates a patient's comprehensive coagulation status: Thromboelastography (TEG). TEG takes the coagulation cascade and the cell-based model of coagulation into account, and it was made specifically for managing the coagulopathic patient. In a hemorrhagic situation (*think trauma/MTP, postoperative bleeding*) TEG provides the clinician with quick and actionable results that report exactly which product to give. TEG can also identify hypercoagulable patients before an adverse event occurs (*think DVT, PE*). This information allows the clinician to individualize treatment for the critically ill patient. Not only does TEG deliver rapid, actionable results, but it allows the clinician to individualize patient care rather than taking a one size fits all approach.

When managing a bleeding patient, four blood product options are available to give: Packed Red Blood Cells (PRBCs), Fresh Frozen Plasma (FFP), Platelets, and Cryoprecipitate (Cryo). There are also an array of pharmaceuticals that can be given as a substitute for some blood products (ex: Kcentra for FFP). TEG evaluates the need for FFP, Platelets, or Cryo. Hemoglobin levels remain the gold standard for transfusing RBCs. Each product above does something entirely different, so giving the wrong product will not help the patient and can even worsen the coagulopathy. For example, giving RBCs when clotting factors are needed. Additional RBCs can further dilute the clotting factors, worsening the coagulopathy. It is also well established that patients who receive blood products are at risk for transfusion reactions, infections, and even have higher instances of mortality. Obtaining a TEG sample will help the clinicians decide exactly which product is necessary; and maybe even sway the clinician to consider another intervention besides a transfusion.

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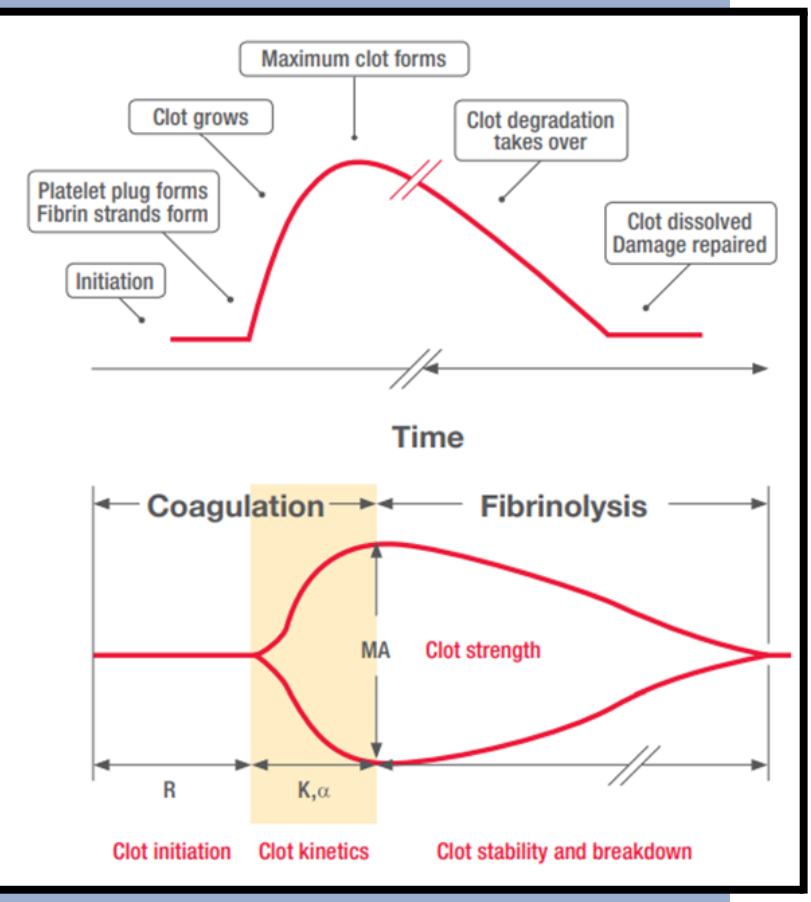

What is TEG and how does it work?

TEG is a whole blood test that evaluates the life cycle of a blood clot. Essentially, TEG is evaluating clot strength over time. To start the test, whole blood is taken from a patient and mixed with reagents that stimulate the clotting process. As a blood clot forms, three different aspects of the clotting process are measured. The first analysis is the rate of clot formation, measured in minutes (R-time). Then, the strength of the clot is measured in millimeters (MA). And finally, the stability of the clot is measured as a percentage (LY30). TEG differs drastically compared to conventional tests because it provides a comprehensive picture of the patient's hemostatic status by providing qualitative analysis opposed to quantitative analysis.

Traditionally, viscoelastic testing platforms have utilized "cup and pin technology". With the new TEG 6s, an advanced concept, resonant frequency, is being utilized. Resonant frequency produces faster results and the results are more precise and reproducible. Here's how it works: the blood sample is mixed with reagents that stimulate blood clot formation. As the clot is forming, the sample is exposed to a range of frequencies. The frequency that causes the greatest sample motion is detected, plotted, and transformed into a TEG tracing.

Let's Breakdown the Measured Parameters

Reaction time (R-time) is measured in minutes and evaluates how long it takes for the initial blood clot to form. If the patient is taking too long to clot, then they are at higher risk for uncontrolled bleeding events (or maybe they're already bleeding). In a scenario where a bleeding patient has a prolonged R-time, the clinician should consider transfusing clotting factors (FFP). Adversely, if clots are forming quite quickly, they may be at risk for an ischemic event.

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Maximum Amplitude (MA) is measured in millimeters and evaluates the strength of the blood clot. Clot formation relies heavily on both platelets and fibrin contribution; therefore, both components need to be adequate in order to create a clot with enough strength to achieve hemostasis. If the patient's platelets or fibrin are not working properly to build a strong clot, they will form weak blood clots. In order to correct the weak blood clots, platelet or cryo transfusions may be in order. Conversely, if a patient is forming stronger clots than necessary (think COVID), they may also be at risk for an adverse ischemic event (think pulmonary embolism, stroke, myocardial infarction, deep vein thrombosis).

Lysis after 30 minutes (LY30) is measured as a percent. LY30 measures how quickly the blood clot is being broken down. If blood clots are immediately being destroyed as soon as they are created (think primary fibrinolysis), then they will not do a satisfactory job in achieving hemostasis. Clot lysis can also happen slowly over time, for which TEG can identify this before it becomes problematic (think Secondary Fibrinolysis, ECMO, Consumptive DIC). In patients with primary hyperfibrinolysis, an anti-fibrinolytic may be needed. Patients in DIC may require adjustments in anticoagulation.

TEG Cartridges

The TEG 6s system is a cartridge-based analyzer, with each cartridge offering multiple tests. There are three different TEG 6s testing cartridges options currently available: Global Hemostasis, Global Hemostasis with Lysis, and PlateletMapping.

Global Hemostasis: Four tests in one cartridge

1. Citrated Kaolin (CK): Kaolin is the reagent used to stimulate the clotting process. Kaolin activates the intrinsic pathway by stimulating thrombin. The CK assay produces the R-time.
2. Citrated Kaolin with Heparinase (CKH): contains kaolin and heparinase. This test is the same as the CK, with the addition of the heparinase. CKH neutralizes all unfractionated heparin (UFH) or low molecular weight heparin (LMWH), which enables the clinician to see underlying clotting function *while* a patient is anticoagulated (with UFH or LMWH). The CKH assay will also produce the R-time.
3. Citrated Rapid TEG (CRT): contains both kaolin and tissue factor to activate the intrinsic and extrinsic pathway. As stated in the name, by simultaneously activating both pathways, we get a more "rapid" result compared to the CK test. This is utilized to determine total clot strength: MA. It is important to remember that total clot strength includes both platelet and fibrin contribution. The CFF will help determine which product is needed, if total clot strength is low.
4. Citrated Function Fibrinogen (CFF): is extrinsically activated with tissue factor and contains ReoPro. ReoPro is an anti-platelet drug that suppresses all platelet function in the sample. This allows us to evaluate clot strength MA in regards to fibrin contribution only. By comparing your CRT MA and CFF MA, a clinician can determine if the patient is in need of Platelets, Cryo or a combo of both.

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Global Hemostasis with Lysis: Three tests in one cartridge

1. Citrated Kaolin (CK): Kaolin is the reagent used to stimulate the clotting process. Kaolin activates the intrinsic pathway by stimulating thrombin. The CK assay produces the R-time and the LY30.
2. Citrated Rapid TEG (CRT): contains both kaolin and tissue factor to activate the intrinsic and extrinsic pathway. As stated in the name, by simultaneously activating both pathways, we get a more "rapid" result compared to the CK test. This is utilized to determine total clot strength: MA. It is important to remember that total clot strength includes both platelet and fibrin contribution. The CFF will help determine which product is needed, if total clot strength is low.
3. Citrated Function Fibrinogen (CFF): is extrinsically activated with tissue factor and contains ReoPro. ReoPro is an anti-platelet drug that suppresses all platelet function in the sample. This allows us to evaluate clot strength MA in regards to fibrin contribution only. By comparing your CRT MA and CFF MA, a clinician can determine if the patient is in need of Platelets, Cryo or a combo of both.

TEG PlateletMapping

All viscoelastic testing platforms (not just TEG) are blind to platelet inhibition. This occurs because viscoelastic tests are thrombin generated, and thrombin has the ability to overpower inhibited platelets. What does that mean? It means that if a patient's platelets are inhibited (ex: on daily Plavix/Aspirin), the GH/GHL tests will not reveal that the patient's clots are weak (low MA). This is where PlateletMapping comes into play. PlateletMapping removes thrombin from the equation so that true platelet function/dysfunction can be uncovered. PlateletMapping evaluates clot strength (MA) only. The adenosine diphosphate (ADP) platelet receptor and the arachidonic acid pathway (AA) are isolated to determine clot strength at those specific sites. Drug effects from P2Y12 inhibitors (ex: Plavix, Effient, Brilinta) can be seen on the ADP assay, whereas NSAIDs (ex: Aspirin, Ibuprofen) are seen on the AA assay. In theory, if a patient was therapeutic on dual antiplatelet therapy (Plavix and Aspirin) they would be forming weak MAs on the ADP and AA assays. It is also important to note that other substances can cause platelet inhibition such as diet, dietary supplements, genetics, or pathologic abnormalities.

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PlateletMapping: Four tests in one cartridge

1. Heparinized Kaolin with Heparinase (HKH): contains kaolin and heparinase as reagents. The HKH will yield a MA that represents full platelet function. This assay will activate all platelet receptors and form a clot with maximum strength, regardless if the patient is on anti-platelet therapy. This process is essentially what is happening on the GH/GHL, hence the inability to identify dysfunction. The HKH MA acts as an indicator of the patient's MA maximum potential.
2. Activator F (ActF): contains a concoction of reagents which include Reptilase, factor Xlll and ReoPro. The goal on the ActF channel is to create a fibrin only clot without the use of thrombin or platelets. The ActF produces a MA that represents the strength of a fibrin only clot (very similar to CFF), which represents MA with complete platelet inhibition.
3. Adenosine Diphosphate (ADP): contains ActF and ADP. In the ADP assay, the platelets are exposed to ADP to determine how they react. If the patient forms a clot at the ADP site similar to the HKH MA, then they are minimally inhibited (if at all). If a clot forms that is similar to the ActF MA, then they are experiencing platelet inhibition. TEG software will calculate the percentage of platelet inhibition and provide a value for the clinician.
4. Arachidonic Acid (AA): contains ActF and AA. In the AA assay, the platelets are exposed to AA to determine how they react. If the patient forms a clot at the AA site similar to the HKH MA, then they are minimally inhibited (if at all). If a clot forms that is similar to the ActF MA, then they are experiencing platelet inhibition. TEG software will calculate the percentage of platelet inhibition and provide a value for the clinician.

The HKH and the ActF provide the clinician with two MA values
HKH MA = maximum platelet function & ActF MA = no platelet function
Now, let's determine where the patient falls on that spectrum- are they closer to FULL potential (HKH) or are they platelets fully inhibited (ActF).

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Clear as mud? I'll be the first to admit that TEG takes time and commitment to achieve understanding. But once understood, TEG has the ability to transform and individualize patient care. It is also important to remember that utilizing TEG in your practice does not throw out everything you already know. TEG is simply another tool to add to the belt. The ultimate goal with TEG is to provide the right blood product to the patient at the right time. Studies indicate this will improve patient care, while also saving the hospital money and resources. Everybody wins with TEG!

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