Understanding and interpreting the TEG

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OBJECTIVES
After this educational activity, participants should be able to:
1. Describe the mechanisms of measurement of the thromboelastogram and related devices

2. Define the need for point of care testing in the perioperative environment to include bleeding and thrombosis

3. Describe a rational approach to hemostasis testing and design algorithms to test the bleeding patient or the patient preparing for surgery on anti-platelet medication
**CASE PRESENTATION**

68 year old male with ascending aorta/proximal arch aneurysm presented for repair under deep hypothermic circulatory arrest. His chief complaint upon admission was substernal chest pain so he was started on heparin drip before being ruled out for an acute MI. His medical history was significant for uncontrolled hypertension, non-insulin dependent diabetes, coronary artery disease s/p CABG X2, peripheral vascular disease and COPD.

Medications: Aspirin, plavix, metoprolol, glucophage, spiriva, and albuterol.

**Imaging**

Chest CT: Aneurysmal dilatation of the distal ascending aorta and proximal arch.

EKG: Normal sinus rhythm, no abnormalities

CXR: Wide mediastinum

TTE: Normal LV systolic function, stage I diastolic dysfunction, normal RV size and function, no significant valvular disease, ascending aorta and arch aneurysm 5.5cm in maximal dimension

**Labs**

CMP: Na+=142, K+=4.1, Cl-=106, HCO3-=24, BUN=12, Cr=0.8, Glc=188

CBC: WBC=11.2, H/H=12/37, Plt=250,000

Coags: PT=15 INR=1.2 PTT=48

ACT=165

**Baseline TEG**

Heparinase and non-heparinase samples

![TEG Graph](image)

A. Non-heparinase

MA=48mm  R=12min  Apha= 40°

B. Heparinase

MA=47mm  R=7min  Apha=42°

**Normal Values**

R= 4-8min  
Apha= 47-74°

K= 0-4min  
MA= 54-72mm  
LY30= 0-8%

**Questions**

1. Is the non-heparinase TEG shown above normal?
2. If abnormal, what is responsible for this tracing?
3. Does this TEG affect your post CPB management of the patient?
4. Can the TEG be utilized to evaluate the effect of plavix and aspirin on this patient's hemostasis competency?
CASE CONTINUATION
The patient was brought to the operating room, STD ASA monitors and left radial arterial line placed followed by induction of general anesthesia with midazolam, lidocaine, etomidate, fentanyl and pancuronium. A right internal jugular cordis with PA catheter were placed post induction. After chest preparation, median sternotomy was made followed by anticoagulation with 400U/kg of heparin, right axillary and right atrial cannulation. After Cardiopulmonary bypass was initiated, the patient was cooled to 20 degrees followed by circulatory arrest. Epsilon aminocaproic acid (EACA) was given after heparinization (70mg/kg bolus over 30 minutes followed by an infusion of 30mg/kg/hour).

QUESTIONS
1. What is the effect of heparin on the thromboelastogram?
2. Why was EACA infused in this patient and what is its effect on the thromboelastogram?
3. What are the effects of cardiopulmonary bypass and hypothermia on the coagulation system and thromboelastogram?

CASE CONTINUATION
The ascending aorta and hemiarch were replaced followed by reimplantation of the inominate and left common carotid arteries. Total duration of DHCA and CPB were 35 minutes and 2 hours respectively. The patient was rewarmed to 37°C prior to separation from cardiopulmonary bypass. His post CPB ACT was 380. A TEG is performed 10 minutes after heparin reversal with protamine (1 mg Protamine: 100U Heparin), and repeat ACT=180

A. Non-heparinase  B. Heparinase
R= 22min                     R= 10min
Apha= 68°                   Apha= 66
K= 5min                   K= 4.9min
MA= 45mm                   MA= 46mm
LY30=1%                   LY30= 0%

QUESTIONS
1. How much protamine should be given to reverse the heparin?
2. What is hepcon? Should hepcon be checked in patients prior to protamine dosing?
3. What effect does overdosing protamine have on the TEG?
4. What coagulopathy is indicated by the post CPB TEGs (Heparinase and non-heparinase)?
5. Is heparin solely responsible for this ACT value and the prolonged R-time?
CASE CONTINUATION
After reversing heparin with extra protamine, his ACT=124. In spite of warming the patient actively, his temperature slowly drifts to 35.2°C and he starts bleeding from multiple surgical sites in the thoracic cavity. Repeat ACT is 198 and the following TEG is obtained. The surgeon requests an extra 100mg of protamine to help stop the bleeding.

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\begin{align*}
R &= 20\text{min} \\
\text{Alpha} &= 40° \\
K &= 6\text{min} \\
\text{MA} &= 36\text{mm} \\
\text{LY30} &= 4% \\
\end{align*}
\]

QUESTIONS
1. Would extra protamine help stop the bleeding given the information above?
2. What is the cause of the patient’s coagulopathy?
3. What should be done to treat this coagulopathy?

CASE CONTINUATION
After the treatment above the bleeding subsides but an arterial blood gas obtained prior to chest closure shows a hematocrit of 18. 4 units of PRBCs are given which brings the Hct to 30. 10 minutes later the patient starts bleeding again. Coagulation tests sent after discontinuing CPB 60 minutes ago are as follows:

PT=18  PTT=40  INR=1.6  Fibrinogen=95  Platelet=150,000

TEG shows R-time=10min, prolonged K-time=8min, Alpha angle of 38 degrees, MA=49, with normal LY30=1%  
ACT=185

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\begin{align*}
R &= 7\text{min} \\
\text{K-time} &= 3 \\
\text{Alpha angle} &= 72° \text{ degrees} \\
\text{MA} &= 52\text{mm} \\
\text{LY30} &= 15% \\
\end{align*}
\]

QUESTIONS
1. What is the cause of bleeding?
2. How would you correct the coagulopathy?
3. What is the cause of the elevated ACT value?

CASE CONTINUATION
After this treatment, hemostasis is maintained, the chest closed and the patient transported to the ICU. Two hours later, the chest tube output gradually increases, and a repeat TEG is shown below.

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\begin{align*}
R &= 7\text{min}, \text{K-time}=3, \text{Alpha angle of } 72° \text{ degrees, } \text{MA}=52\text{mm} \text{, with } \text{LY30}=15% \\
\end{align*}
\]
QUESTIONS
1. What is the cause of bleeding and treatment options?
2. Is this treatment different from other forms of this condition?

DISCUSSION
Cardiac surgery is associated with coagulopathy due to iatrogenic reasons from the use of anticoagulants like heparin, mild hypothermia, and the use of an extracorporeal circuit leading to consumption of coagulation factors and platelet dysfunction. Post cardiopulmonary bypass coagulopathy and massive blood transfusion with its associated complications like TRALI, transfusion reactions and infection etc also worsen patient outcomes.13,5 The use of lab tests like PT/PTT/INR to guide transfusion in this dynamic perioperative setting is useful but not feasible because the lab results always lag behind the clinical setting due to the time involved in performing these tests, and as a result most clinicians transfuse blood products without any scientific algorithm.

Point-of-care tests like the activated clotting time (ACT) and thromboelastogram (TEG) are quick to perform, cheap, reproducible, portable, and thus help diagnose and treat coagulation disorders quicker in the perioperative setting.14 A post CPB TEG with and without heparinase for example guides the clinician in treating coagulopathy due to heparin effect quicker than ordering a PTT. TEG guided transfusion has been shown to decrease the use of blood products in both adult and pediatric cardiac surgical procedures and improve patient outcomes.3,4,8,9

The TEG is a point of care test which measures in vitro the visco-elastic properties of whole blood and is useful in diagnosing coagulation disorders and guiding both pharmacologic treatment and blood transfusion.11 Its usefulness in the perioperative setting has been shown in multiple studies in cardiac surgical and liver transplantation patients.3,4,8,9,11 The TEG has also been shown to be helpful in guiding the use of rFVIIa in patients with refractory hemorrhage after cardiac surgery, and in predicting thrombotic complications in the perioperative period16

The TEG provides numerical date and a graphical representation of the different stages of thrombogenesis from the initial formation and stabilization of clot, to possible fibrinolysis over a time period.2 Unlike other coagulation lab studies which assess separate entities of coagulation, it assesses the whole coagulation cascade and helps the clinician decide on the best treatment option.10

TEG-platelet mapping (TEG-PM) is another useful tool in the perioperative setting that helps the clinician identify the degree of platelet inhibition by antiplatelet agents like Plavix and Aspirin.17 In-vivo, platelet activation which is mediated by the GP IIb/IIIa receptor involves the ADP or TXA2 receptor but the standard TEG depends on direct thrombin activation of the GP IIb/IIIa receptor, limiting the identification of patients with platelet inhibition from ASA/Plavix.13,17 The TEG-PM utilizes exogenous ADP and arachidonic acid to assess the degree of platelet inhibition by plavix and aspirin. This is extremely important in cardiac surgical patients who are frequently on these agents and as a result a normal platelet count on CBC or TEG MA is less indicative of the degree of platelet dysfunction.
When performing a TEG, the coagulation cascade is activated with a reagent like tissue factor or kaolin. These reagents help speed up the initiation of thrombin formation. Without activation, the TEG takes about an hour for completion.


The TEG assesses coagulation profile with five main parameters, the R-time, K-time, Alpha angle, MA, and LY30.

**R (Reaction time)**
Is the time from initiation of the test to clot formation, and can be prolonged with clotting factor deficiency or anticoagulants like heparin. Hypercoagulable states on the contrary shorten the R value. A Prolonged R time hence indicates clotting factor deficiency or iatrogenic anticoagulation (heparin effect), and is best treated with FFP or protamine.

**Alpha angle:**
Angle of divergence of the curve from the baseline, measures the rate of clot formation (fibrin formation and platelet cross-linking). It is more comprehensive than K. A low angle is primarily due to hypofibrinogenemia, but can also be due to thrombocytopenia, or thrombocytopathy. Alpha angles less than 45 °C reflect low levels of fibrinogen or platelets and the best treatment option is transfusing Cryoprecipitate or platelets.

**K-time:**
Represents the period from initial clot formation(R) until an amplitude of 20mm is reached. Low levels of fibrinogen prolong the K-time and vice versa. Platelet count/function also affects the K-time in a similar fashion. Both K and alpha angle are measures of clot kinetics. Prolonged K and low alpha angle reflect fibrinogen deficiency.

**MA:**
Is a measure of clot strength, and reflects platelet strengthening of the fibrin plug. It is affected mainly by platelet count/function, fibrinogen levels, and factor VIII levels. MA values between 46-54min reflect mild platelet dysfunction and can be treated with DDAVP to increase VWF, and factor VIII levels. In patients with coagulopathy and low MAs, if the MA remains low after adequate platelet transfusion, Cryo or FFP should be transfused to increase fibrinogen levels.

**LY30:**
Measures the rate of amplitude reduction 30 minutes after the MA is reached, and represents ultimate clot strength. LY30 > 7.5-8% represents fibrinolysis. If the TEG is normal but
the patient keeps bleeding, then a surgical cause or Von Willebrand factor deficiency may be the cause.

The TEG is a very cheap test, which is quick to perform, reproducible, and easy to interpret. Its usefulness in the management of cardiac surgical patient is outlined above and should be incorporated into the perioperative management of these complex patients.

REFERENCES