



Basic Clinician Training



Module 2
TEG® Technology

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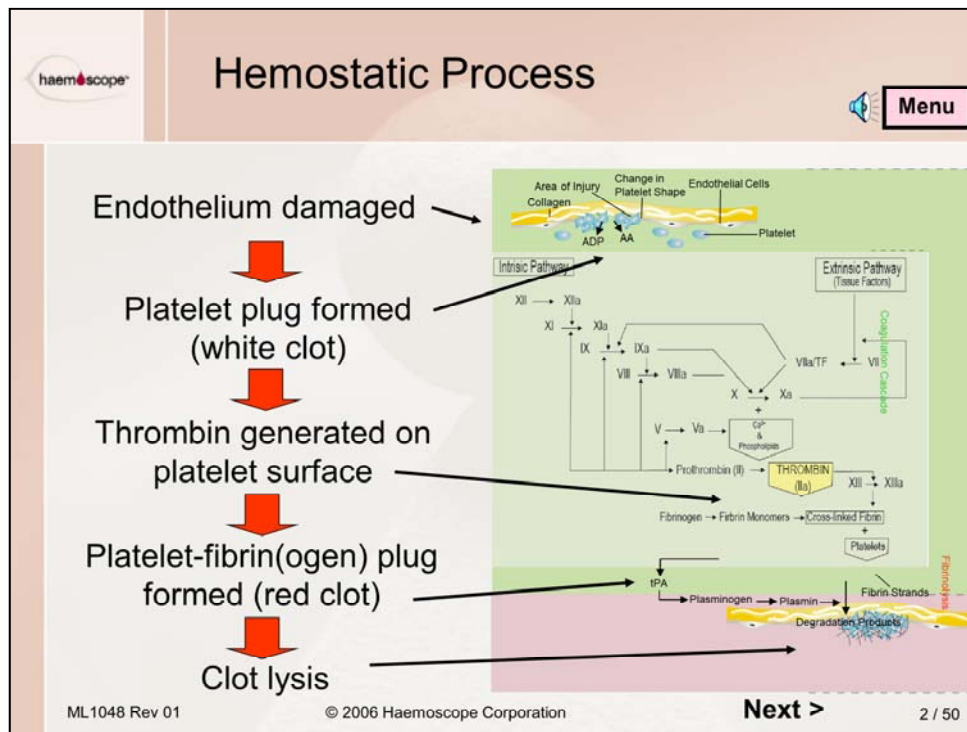
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The information and diagnostic statements in this module are based on information contained in standard medical publications and reference materials. Users are solely responsible for the selection, use, and suitability of interpretation or treatment recommendation in general or in any particular instance. Clinicians should use their own medical judgment together with assessment of the patient's clinical condition, when considering TEG® results and making diagnosis and treatment decisions.

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This module discusses monitoring hemostasis with the TEG analyzer and shows how TEG analysis reflects the cell based model of hemostasis. For more information on the cell based model and hemostasis in general, see Module 1.

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The hemostatic process typically begins with endothelial damage. Next, platelets adhere to the site of damage, forming a platelet plug, or white clot. Thrombin generation follows. This is a pivotal point in hemostasis, and occurs on the surfaces of platelets and tissue factor bearing cells.

Thrombin amplifies further thrombin generation by activating factor XI in the intrinsic pathway. This leads to an explosive generation of thrombin. It also activates factor XIII, which allows fibrin to cross-link, further increasing the strength of the platelet-fibrin plug.

Finally, thrombin activates platelets, leading to further platelet adhesion and aggregation. In addition, it converts fibrinogen to fibrin, which forms a strong platelet-fibrin(ogen) plug, or red clot.

Clot lysis actually begins while the clot forms, limiting the size of the clot and preventing obstruction of blood flow. Lysis is also involved in the final stage of the healing process, when the clot is completely removed from the surface of the endothelium.

The slide is titled "Routine Coagulation Tests" and features the Haemoscope logo in the top left corner. A "Menu" button with a speaker icon is in the top right. The main content is a bulleted list:

- Traditional lab tests
 - PT, aPTT
 - Platelet number/count
 - D-DIMER, FDP
- Based on cascade model of coagulation
 - Measure protein interaction in plasma (thromboplastin)
 - Exclude cellular contributions (platelets, monocytes, etc.)
 - Determine adequacy of coagulation factor levels
- Use static endpoints
 - Ignore altered thrombin generation
 - Ignore cellular elements
 - Ignore overall clot structure

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Steps of the hemostatic process can be monitored with traditional lab tests. These include:

- PT and aPTT for coagulation pathway function
- Platelet number
- D-DIMERS or fibrin degradation products (FDP) for clot lysis

PT and aPTT are plasma-based coagulation tests commonly used to monitor hemostasis and are based on the cascade model of hemostasis. This cascade model provides a good representation of the hemostatic processes observed in the laboratory, where components are isolated and the processes occur in plasma.

However, PT and aPTT determine only if coagulation factors are present in levels adequate for clot formation. They do not consider the role of cells or the contribution of local vascular and tissue conditions. Therefore, they do not measure the impact of platelets and platelet activation on thrombin generation.

Furthermore, these plasma-based assays use static endpoints such as fibrin formation. As a result, they do not measure the impact of altered thrombin generation on platelet function and overall clot structure. Also, PT and aPTT monitor only the first 5% of total thrombin generation.

Bottom line: PT and aPTT are isolated, static tests that monitor only a small part of the entire hemostatic process.



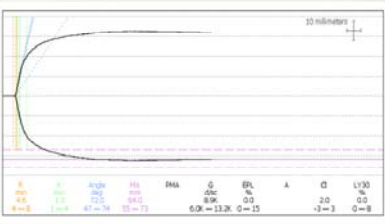
Hemostasis Monitoring: TEG Hemostasis System



Menu



- Whole blood test
- Measures hemostasis
 - Clot initiation through clot lysis
 - Net effect of components
- TEG system
 - Laboratory based
 - Point of care
 - Remote, can be networked
 - Flexible to institution needs





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The TEG® hemostasis system monitors hemostasis using whole blood and is able to measure the balance of the hemostatic components through all phases, from clot initiation through clot lysis. Because it uses whole blood, it demonstrates the net effect of all the hemostatic components including coagulation factors, platelets, and other cellular elements.

The TEG system is considered a moderately complex test by the U.S. FDA and configures well in laboratory based situations. Because no processing is required to run a test, and because of its relatively small footprint, the TEG analyzer can also be used as a point of care monitoring device.

The TEG software can be networked, allowing for use in locations remote from the patient. The placement of the analyzer depends on the needs and requirements of the institution.



The TEG Analyzer: Description

 Menu

- Reflects balance of the hemostatic system
- Measures the *contributions* and *interactions* of hemostatic components during the clotting process
- Uses *activated* blood to maximize thrombin generation and platelet activation in an *in vitro* environment
 - Measures the hemostatic potential of the blood at a given point in time under conditions of maximum thrombin generation

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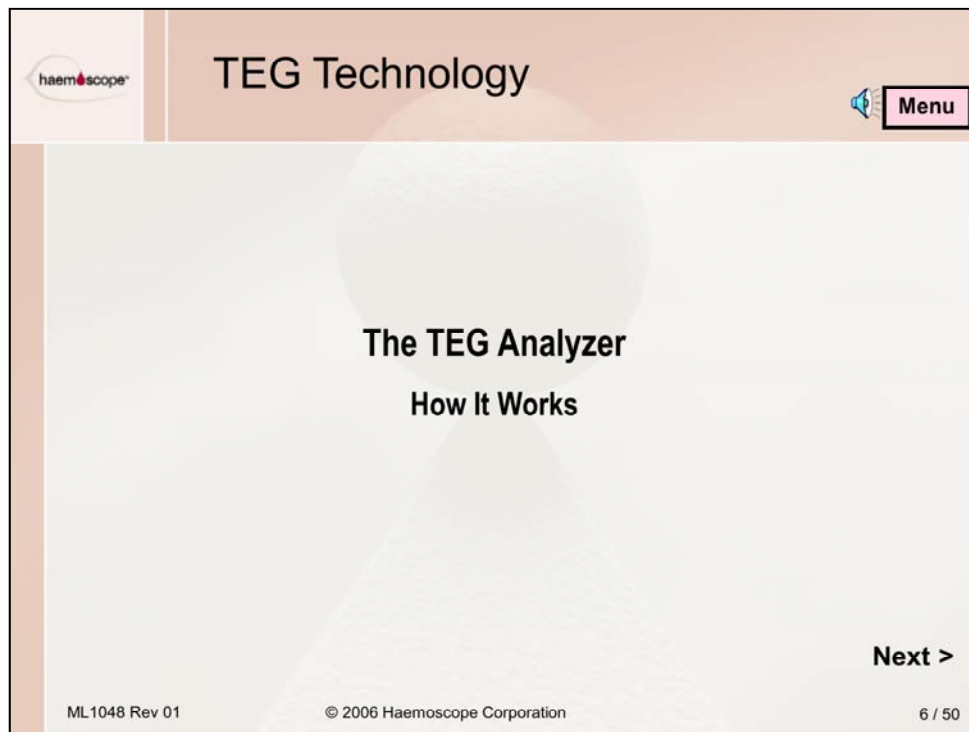
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Analysis of TEG results helps the clinician look at the balance of the hemostatic system. The TEG analyzer demonstrates both the contributions and the interactions of the blood-borne hemostatic components during the clotting process. It actually monitors the shear elasticity of clotting blood — or in other words, the mechanical properties of the developing clot.

The whole blood sample is activated *in vitro* to maximize thrombin generation, and thus platelet activation. As a result, the TEG analyzer demonstrates the hemostatic potential of the blood at a given point in time. This simulates *in vivo* clot formation under conditions of explosive thrombin generation.

However, the anticoagulant effects of the many endothelium-derived factors on the hemostatic process are not measured by the TEG system, or by any other *in vitro* test.

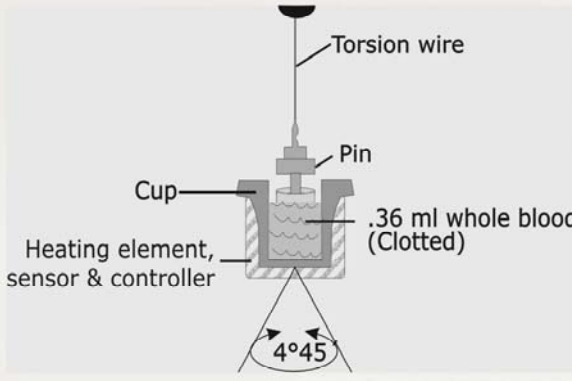


This section explains how the TEG analyzer works. The analyzer monitors the dynamic changes in the hemostasis of a sample with respect to time.



TEG Technology: How It Works


Menu



- Cup oscillates
- Pin is attached to a torsion wire
- Clot binds pin to cup
- Magnitude of pin motion is a function of the mechanical properties of the clot
- System generates a hemostasis profile
 - From initial formation to lysis

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The TEG analyzer has a sample cup that constantly oscillates at a set speed through an arc of $4^{\circ}45'$; each oscillation lasts ten seconds. A whole blood sample of $360\ \mu\text{l}$ is placed into the cup, and a stationary pin attached to a torsion wire is immersed in the blood. When fibrin first forms, it begins to bind the cup and pin, causing the pin to oscillate in phase with the cup.

The torque of the rotating cup is transmitted to the immersed pin only after fibrin or fibrin-platelet bonding has linked the cup and pin together. The strength of these fibrin-platelet bonds affects the magnitude of the pin motion.

The magnitude of the output is directly related to the strength of the forming clot. As the clot retracts or lyses, the bonds between the cup and pin are broken, and the transfer of cup motion is diminished. The movement of the pin is converted by a mechanical-electrical transducer into an electrical signal, which can be monitored by a computer.

The movement of the pin generates a hemostasis profile, which is a measure of the time it takes for the first fibrin strand to form, the kinetics of clot formation, the strength of the clot (in shear elasticity units of dyn/cm^2), and the dissolution of clot.



Utility of TEG Analysis

 Menu

- Demonstrates all phases of hemostasis
 - Initial fibrin formation
 - Platelet-fibrin(ogen) plug construction
 - Clot lysis
- Identifies imbalances in the hemostatic system
 - Risk of bleeding
 - Risk of thrombotic event

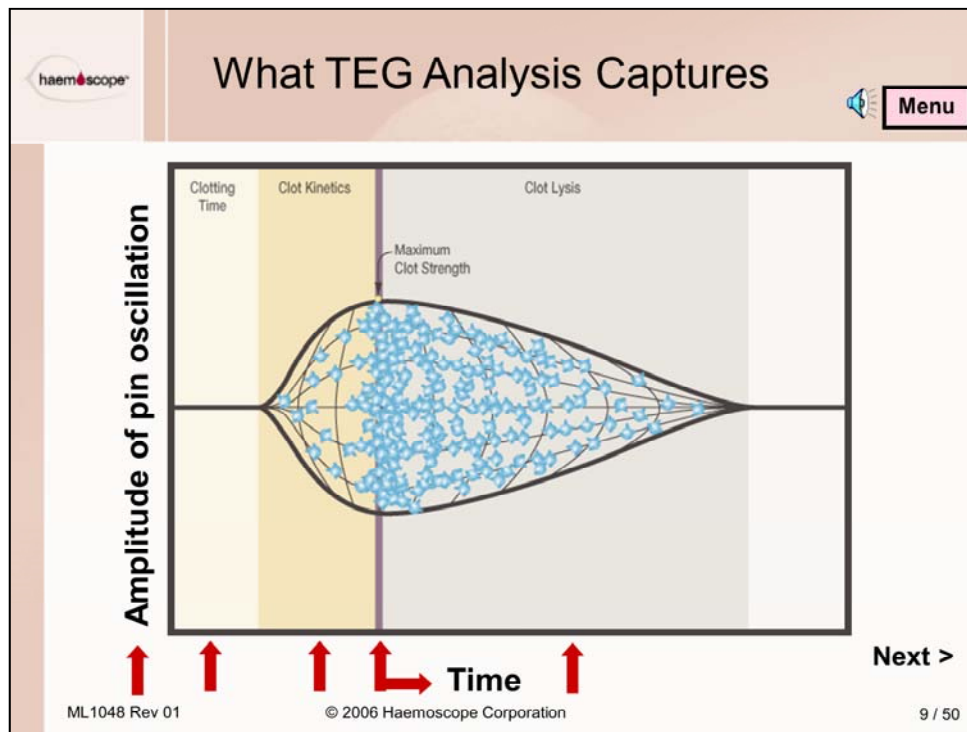
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TEG results demonstrate all phases of hemostasis, from initial fibrin formation to platelet-fibrin(ogen) plug construction through clot lysis. A TEG analysis can also identify imbalances within the hemostatic system, which can help to stratify the risk of bleeding or a thrombotic event.



TEG analysis monitors the shear elasticity, or mechanical properties, of the developing clot. These mechanical properties influence the movement of the pin, which in turn creates the TEG hemostasis profile. The Y-axis of the profile demonstrates the amplitude of pin motion in millimeters, and the X-axis demonstrates the time in minutes.

The hemostasis profile demonstrates all phases of hemostasis:

- Initial fibrin formation, which is clotting time
- Development of the fibrin and fibrin-platelet clot which is clot kinetics
- Attainment of maximum clot strength
- Clot breakdown, which is lysis

The time to initial fibrin formation reflects the involvement of the coagulation factors and pathways in the generation of thrombin and in subsequent fibrin formation.



Basic Clinician Training

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TEG Parameters

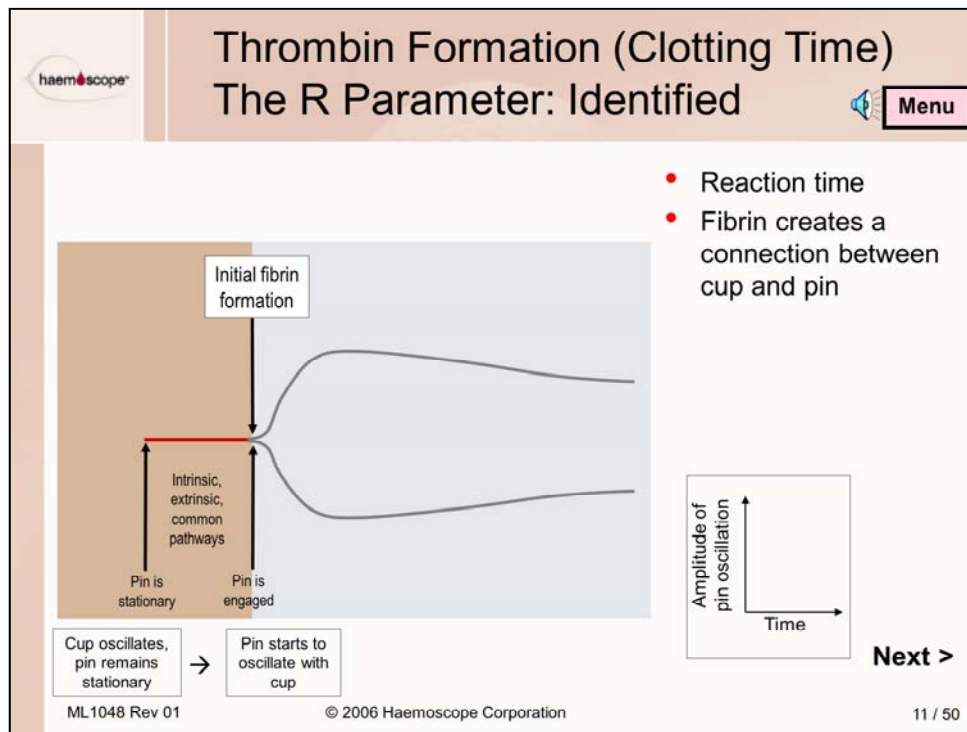
Identification Definition

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This section identifies and defines the TEG parameters commonly used for the interpretation of hemostatic abnormalities.



The R parameter represents clotting time. R, or reaction time, is identified as the time from the start of the test, when the pin is stationary, to the time of initial fibrin formation, when fibrin creates a connection between the surface of the cup and the surface of the pin, causing the pin to begin oscillating.

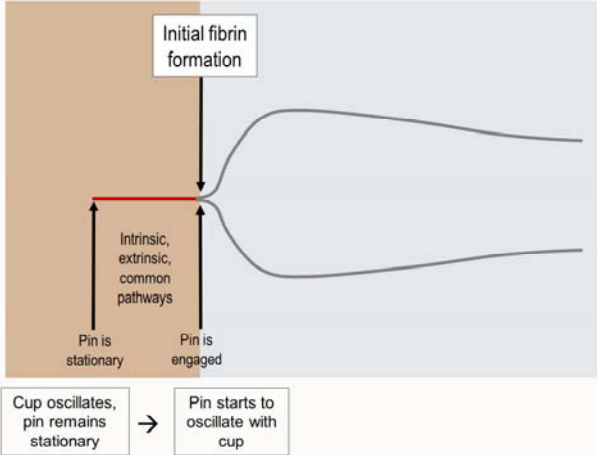


Thrombin Formation

The R Parameter: Defined



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- Time until initial fibrin formation
- Expression of enzymatic reaction function (i.e. the ability to generate thrombin and fibrin)

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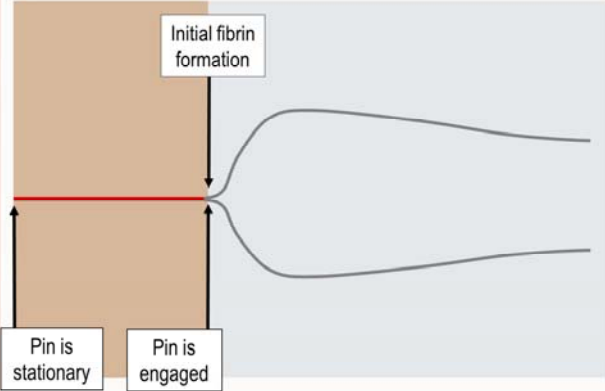
The R parameter designates the time until initial fibrin formation, sufficient to engage the pin. The R value reflects the ability of the coagulation pathways — or the series of enzymatic coagulation reactions — to generate thrombin, which in turn cleaves fibrinogen into fibrin.



Thrombin Formation Abnormalities

The R Parameter: Elongated R


Menu



- Possible causes of imbalance:
 - Slow enzymatic reaction
- Possible etiologies:
 - Factor deficiency/dysfunction
 - Residual heparin
- Common treatments:
 - FFP²⁹
 - Protamine²⁹

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An elongated R parameter indicates an imbalance in the coagulation pathways, causing a delay in the generation of thrombin or fibrin, typically resulting in bleeding. Possible causes include:

- The slowing of one or all of the enzymatic coagulation reactions
- A possible dysfunction in the activity of thrombin, or in the fibrin and fibrinogen molecules themselves

Possible etiologies include:

- Deficiencies in coagulation factors
- A dysfunction in a particular coagulation factor, limiting the rate of the entire enzymatic process

Another possibility is the presence of a coagulation factor inhibitor such as heparin. Heparin inhibits the activity of thrombin and factor Xa, thus reducing the rate of thrombin generation and the ability of thrombin to reach critical mass for cleaving fibrinogen into fibrin.

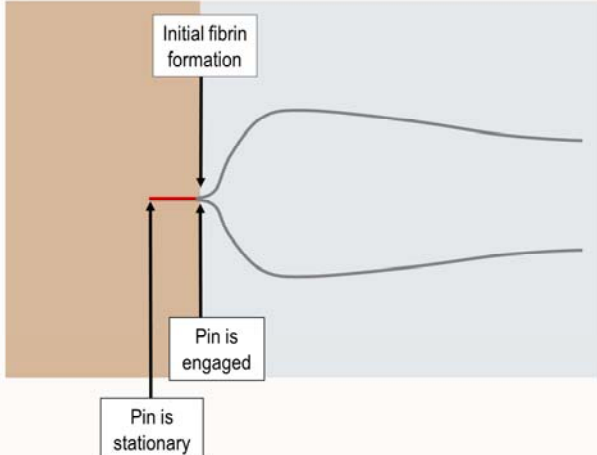
Treatment for an elongated R value depends on the cause. General factor deficiencies are commonly treated with fresh frozen plasma (FFP), whereas the presence of heparin is commonly treated with protamine, which neutralizes its effect.²⁹



Thrombin Formation Abnormalities

The R Parameter: Short R


Menu



- Possible causes of imbalance:
 - Over-stimulated enzymatic reaction
 - Fast fibrin formation
- Possible etiologies:
 - Enzymatic hypercoagulability
- Common treatments:
 - Anticoagulant³⁰

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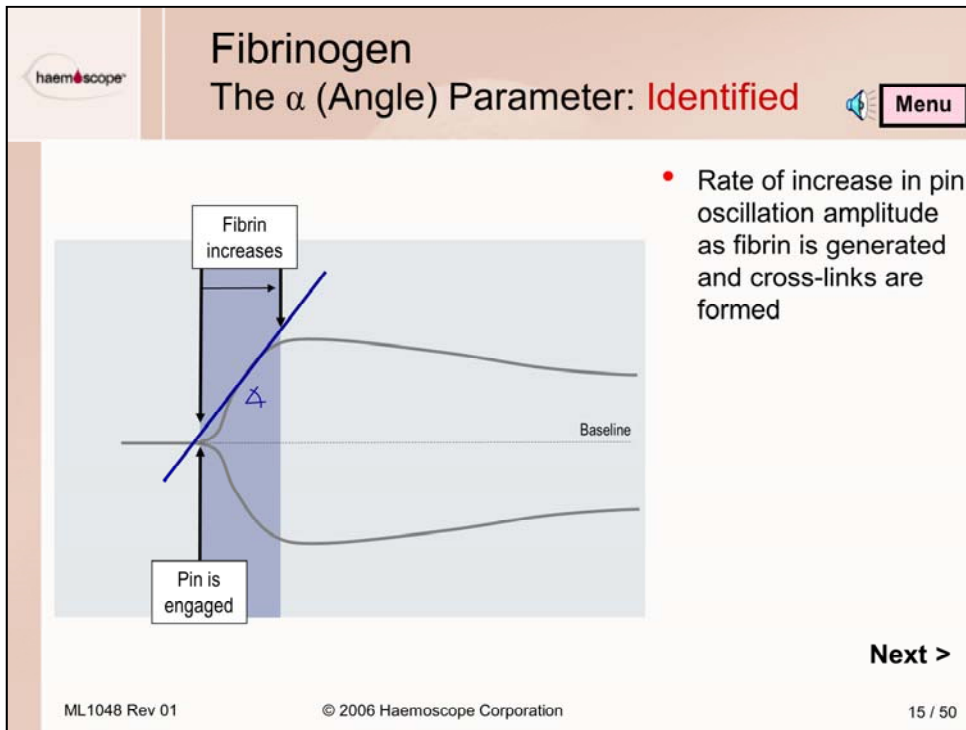
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A short R value is also indicative of an imbalance in the hemostatic system, one that may result in the inappropriate formation of a fibrin clot that could impede blood flow. Possible causes include:

- Accelerated enzymatic reactions, resulting in rapid generation of thrombin
- Rapid or excessive fibrin generation

The cause of a short R value is commonly enzymatic hypercoagulability, which could be due to the loss of one or more of the feedback control mechanisms in the hemostatic system.

The common treatment for a short R value is an anticoagulant such as heparin, low molecular weight heparin (LMWH), warfarin, or a direct thrombin inhibitor.³⁰



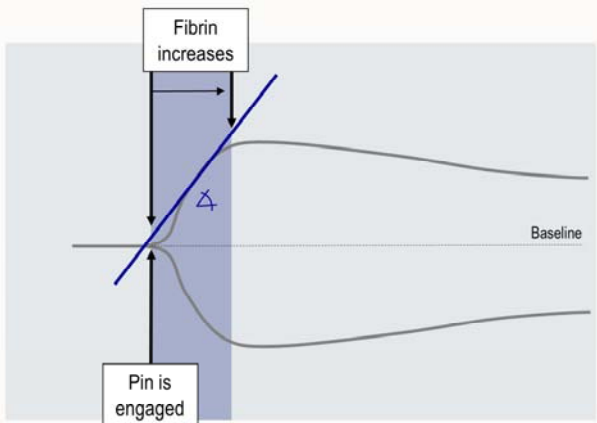
The angle or alpha (α) parameter is a kinetic measurement of clot formation that represents the rate of increase in pin oscillation amplitude due to fibrin generation and fibrin cross-linking.



Fibrinogen

The α (Angle) Parameter: **Defined**


Menu



- Kinetics of clot formation
 - Rate of thrombin generation
 - Conversion of fibrinogen → fibrin
 - Interactions among fibrinogen, fibrin, and platelets
 - Cellular contributions

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The angle represents the kinetics of clot formation. This includes:

- The rate of thrombin generation
- The conversion of fibrinogen to fibrin
- The interactions among fibrinogen, fibrin, and platelets
- The cellular contributions to clot formation

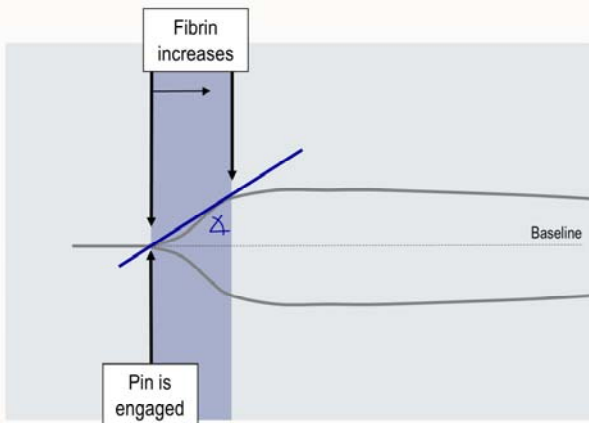
The faster the rate of fibrin generation, the greater the increase in pin oscillation amplitude, and the larger the angle.



Fibrinogen Abnormalities

The α (Angle) Parameter: **Low α**


Menu



- Possible causes of imbalance:
 - Slow rate of fibrin formation
- Possible etiologies:
 - Low fibrinogen levels or function
 - Insufficient rate/amount of thrombin generation
 - Platelet deficiency/dysfunction
- Common treatments:
 - FFP²⁹
 - Cryoprecipitate²⁹

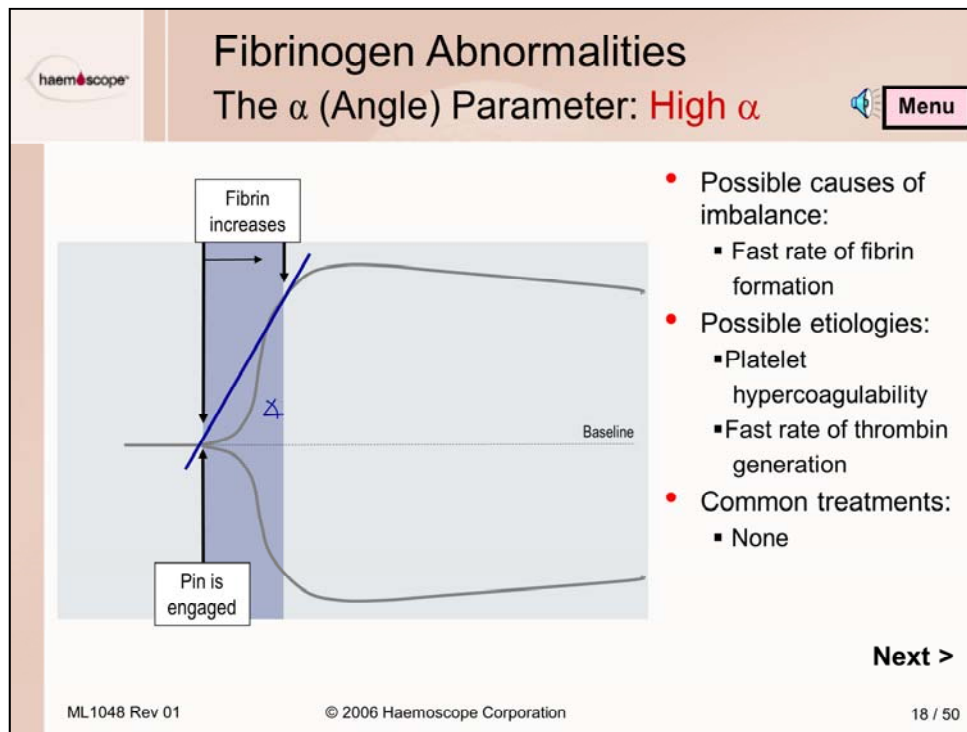
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Abnormalities in the angle parameter typically represent an imbalance in fibrinogen levels and in the interaction among fibrin, fibrinogen, and platelets. A low angle suggests a slow rate of fibrin formation, which could lead to bleeding.

Possible causes of slow fibrin formation include:

- Low fibrinogen levels or function
- An insufficient rate or amount of thrombin generation to sustain fibrin formation
- Platelet deficiency or dysfunction

The common treatments for a low angle depend on the cause and the degree of bleeding. An isolated low angle with normal R and MA values is indicative of low fibrinogen levels, a condition commonly treated with FFP or cryoprecipitate.²⁹



A high angle suggests a rapid rate of fibrin formation. This is usually indicative of platelet hypercoagulability or a rapid rate of thrombin generation, which can lead to thrombosis.

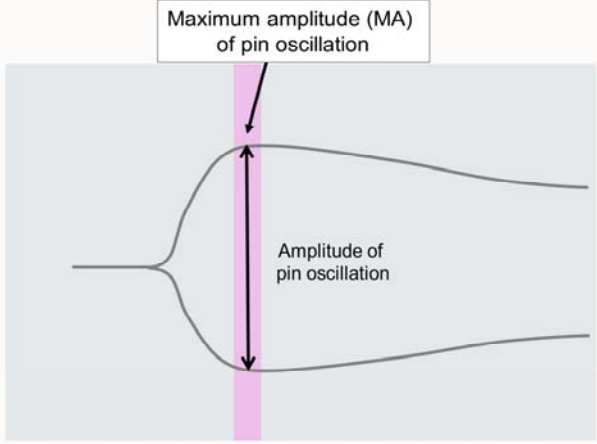
Since a high angle is the result of an imbalance in other phases of the hemostatic process, there is no specific common treatment for it. Possibilities for reducing the angle includes anticoagulation and/or platelet inhibition therapy.



Platelet Function

The MA Parameter: Defined


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- Maximum amplitude
- Clot strength = 80% platelets + 20% fibrinogen
- Platelet function influences thrombin generation and fibrin formation → relationship between R, α , and MA

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The maximum amplitude, or MA parameter, represents clot strength; the stronger the clot, the greater the amplitude of pin oscillation.

The major contributors to clot strength are platelets (80-90%) and fibrinogen (10-20%), which binds the platelets together. The MA parameter represents the function of platelets present in the sample.

Since platelet number and function influence thrombin generation, an abnormality in the MA value may also be accompanied by slightly abnormal R and angle values.

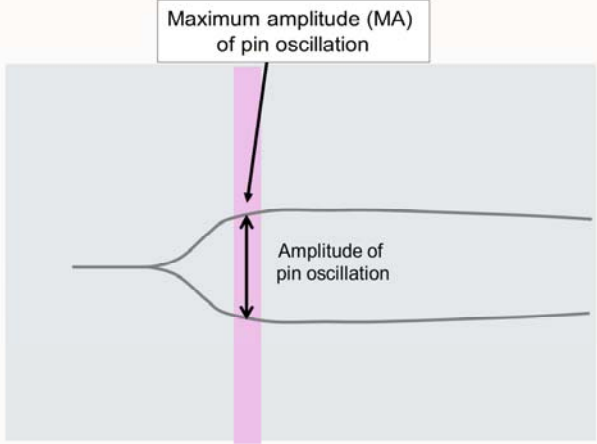


Platelet Function Abnormalities

The MA Parameter: Low MA



Menu



- Possible causes:
 - Insufficient platelet-fibrin(ogen) clot formation
- Possible etiologies:
 - Poor platelet function
 - Low platelet count
 - Low fibrinogen levels or function
- Common treatments:²⁹
 - Platelet transfusion

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Abnormalities in the MA value represent an imbalance in the hemostatic system, and are typically associated with platelet function because of the 80% contribution by platelets to clot strength. A low MA value is indicative of insufficient platelet-fibrin(ogen) clot formation due to poor platelet function, low platelet count, or low fibrinogen levels or function, all of which can cause bleeding.

A low MA value does not differentiate between low platelet number and platelet dysfunction. A low platelet number, combined with normal or hyperfunctional platelets, could result in a normal or high MA value. A high platelet count with dysfunctional platelets could result in a low MA and be associated with bleeding.

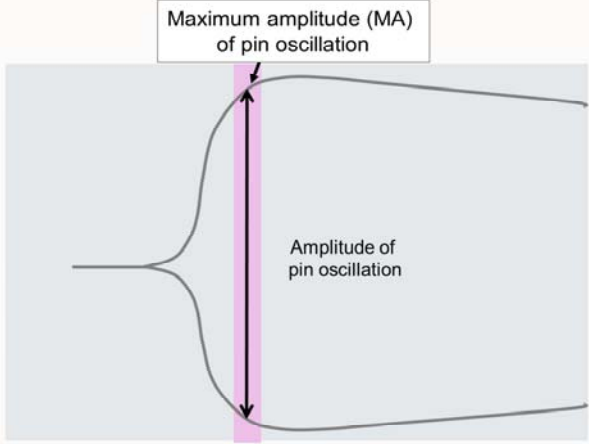
The most common treatment of a low MA in a bleeding patient is transfusion of platelets.²⁹ The amount of platelets required to reverse bleeding depends on the magnitude of the abnormality and on the patient's overall status.



Platelet Function Abnormalities

The MA Parameter: High MA


Menu



Maximum amplitude (MA)
of pin oscillation

Amplitude of
pin oscillation

- Possible causes:
 - Excessive platelet activity
- Possible etiologies:
 - Platelet hypercoagulability³⁰
- Common treatments:³⁰
 - Antiplatelet agents
 - Note: Should be monitored for efficacy and/or resistance (See Module 6: PlateletMapping™)

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A high MA suggests excessive platelet activity due to platelet hypercoagulability or high platelet count. Patients with an abnormally high MA are at higher risk of a thrombotic event. [Gurbel et al. "Platelet Reactivity in Patients and Recurrent Events Post-Stenting" (*J Am Coll Cardiol* 2005; 46: 1820-1826); McGrath DJ et al. "Thrombelastography Maximum Amplitude Predicts Postoperative Thrombotic Complications Including Myocardial Infarction" (*Anesth Analg* 2005; 100: 1576-83)]

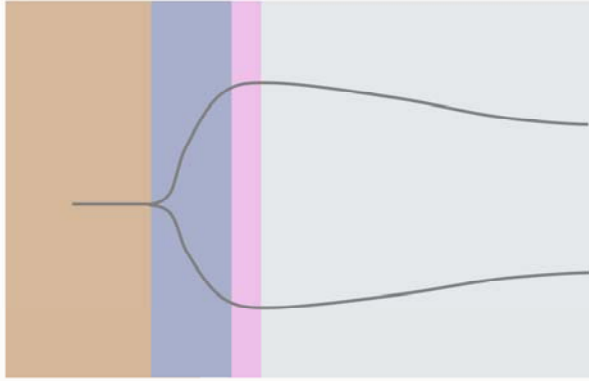
The most common treatment for platelet hypercoagulability is an antiplatelet agent, such as aspirin or clopidogrel.³⁰ The standard TEG test does not measure the effects of these antiplatelet agents; however, the PlateletMapping assays have been developed specifically to monitor these effects. See Module 6 for more details.



Coagulation Index

The CI Parameter: **Defined**

 **Menu**



- Global index of hemostatic status
- Linear combination of kinetic parameters of clot development and strength (R, K, angle, MA)
 - CI > +3.0:
hypercoagulable
 - CI < -3.0:
hypocoagulable

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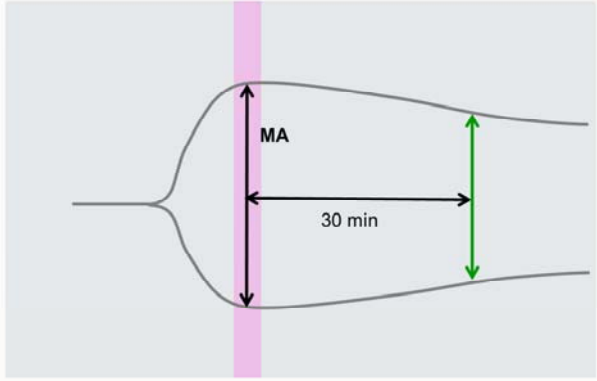
The coagulation index, or CI value, provides an indication of the global hemostatic state of a patient. It is derived from a linear combination of the kinetic parameters of clot development (R, K, angle) and clot strength (MA).

A CI value greater than 3.0 suggests a hypercoagulable state and a higher risk of thrombotic events. On the other hand, a CI value less than -3.0 suggests a hypocoagulable state and a higher risk of bleeding.



Fibrinolysis: LY30 and EPL

LY30 and EPL Parameters: **Identified**  [Menu](#)



The diagram shows a typical fibrinogen curve. A vertical pink line marks the point where Maximum Amplitude (MA) is reached. A horizontal arrow labeled '30 min' indicates the time interval after MA for measuring LY30 and EPL. A green double-headed vertical arrow indicates the change in amplitude over this 30-minute period.

- LY30 is the percent decrease in amplitude of pin oscillation 30 minutes after MA is reached
- Estimated percent lysis (EPL) is the estimated rate of change in amplitude after MA is reached

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The final phase of hemostasis, clot breakdown or fibrinolysis, is represented by two parameters, LY30 and EPL.

The LY30 value indicates the percent decrease in the amplitude of pin oscillation (i.e. clot strength) 30 minutes after MA is attained.

The EPL, or estimated percent lysis value, estimates the rate of change in amplitude after MA is reached. The EPL value estimates the rate of overall clot breakdown.

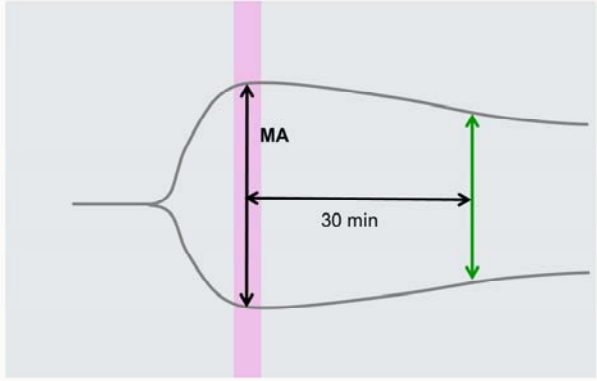


Fibrinolysis: LY30 and EPL

LY30 and EPL Parameters: **Defined**

 **Menu**

- Reduction in amplitude of pin oscillation is a function of clot strength, which depends on extent of fibrinolysis



The diagram illustrates a blood clot on a light blue background. A vertical pink line is positioned on the left side of the clot. A horizontal black arrow labeled '30 min' indicates the time interval from the pink line to the right edge of the clot. A vertical black double-headed arrow labeled 'MA' indicates the maximum amplitude of the clot. A green vertical double-headed arrow is shown on the right edge of the clot, representing the reduction in amplitude of pin oscillation.

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Once the clot has formed, clot strength depends on the extent of clot breakdown or fibrinolysis. Therefore, reduction in the amplitude of pin oscillation depends on reduction in clot strength.



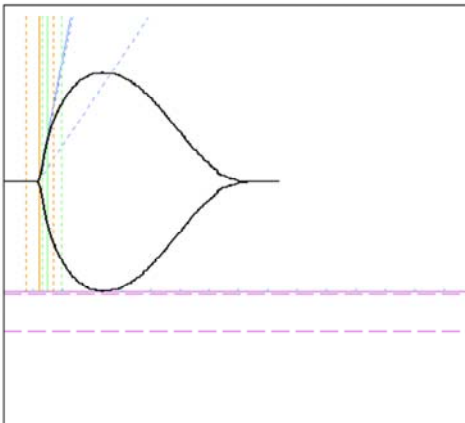
Fibrinolytic Abnormalities

LY30 Parameter: **Primary Fibrinolysis**

Menu

(primary fibrinolysis)

4 Kaolin



- Possible causes:
 - Excessive rate of fibrinolysis
- Possible etiologies:
 - High levels of tPA
- Common treatments:²⁹
 - Antifibrinolytic agent

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Fibrinolysis is an important part of hemostasis because fibrinolysis limits the amount of clot formation and removes the clot during the healing process. When fibrinolysis is greater than the rate of clot formation, or when it causes the breakdown of new clots, bleeding typically occurs. This condition is primary fibrinolysis and is identified with the TEG analyzer by an LY30 value of greater than 7.5% (or EPL > 15%), combined with a CI value of less than or equal to 1.0.

The common cause of primary fibrinolysis is high levels of tissue plasminogen activator (tPA). tPA is released by the vascular endothelial cells and in turn generates high levels of circulating plasmin. Since primary fibrinolysis is due to pathological levels of circulating plasmin, the most common treatment is an antifibrinolytic agent.²⁹

It is important to distinguish between primary and secondary fibrinolysis, since treatments are very different. Incorrect treatment can be fatal.



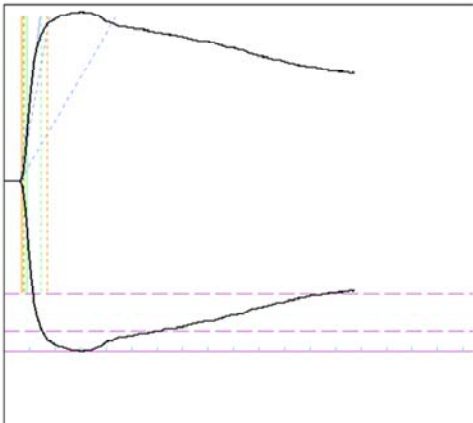
Fibrinolytic Abnormalities

LY30 Parameter: **Secondary Fibrinolysis**


Menu

3 Kaolin

§(secondary fibrinolysis)



DIC = disseminated intravascular coagulation

- Possible causes:
 - Rapid rate of clot formation/break-down
- Possible etiologies:
 - Microvascular hypercoagulability (i.e. DIC)

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Secondary fibrinolysis is also caused by an excessive rate of clot breakdown. However, in this case, it is activated by excessive clot formation and is actually a protective mechanism to ensure that inappropriate clotting does not impede blood flow. Secondary fibrinolysis commonly occurs in the first phase of sepsis, or in disseminated intravascular coagulation.

With the TEG analyzer, secondary fibrinolysis is identified as a LY30 value of greater than 7.5% (EPL > 15%), combined with a CI value of greater than 3.0.

A common cause of secondary fibrinolysis is microvascular hypercoagulability, a condition in which clots form in the microvasculature, possibly due to a dysfunction in the antithrombotic properties of the vascular endothelium. A systemic inflammatory response may also initiate secondary fibrinolysis via platelet activation or the coagulation pathways.



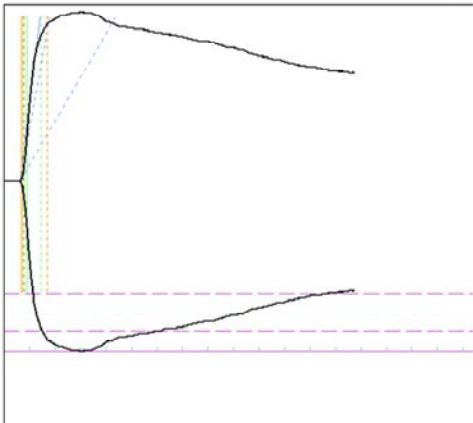
Fibrinolytic Abnormalities

LY30 Parameter: **Secondary Fibrinolysis**


Menu

3 Kaolin

S(secondary fibrinolysis)



DIC = disseminated intravascular coagulation

- Possible causes:
 - Rapid rate of clot formation/break-down
- Possible etiologies:
 - Microvascular hypercoagulability (i.e. DIC)
- Common treatments:³¹
 - Anticoagulant

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In either case, a hypercoagulable state is initiated, which in turn activates fibrinolysis. This fibrinolytic response attempts to keep the blood vessels clear of clots. If the cause is not reversed, the cycle of clot formation and clot breakdown will continue until the coagulation factors are consumed, or until the platelets can no longer support coagulation reactions.

Since the cause of secondary fibrinolysis is hypercoagulability, a common treatment is an anticoagulant and/or an antiplatelet drug. Treatment of this condition with an antifibrinolytic agent could inhibit an important protective mechanism and increase the probability of an ischemic event.³¹

Misclassifying secondary fibrinolysis as primary fibrinolysis can be fatal.



Clot Strength: The G Parameter


Menu

- Representation of clot strength and overall platelet function
 - G = shear elastic modulus strength (dyn/cm²)
 - $G = (5000 \cdot MA) / (100 - MA)$
- Relationship between clot strength and platelet function
 - MA = linear relationship between clot strength and platelet function
 - G = exponential relationship between clot strength and platelet function
 - More sensitive to changes in platelet function

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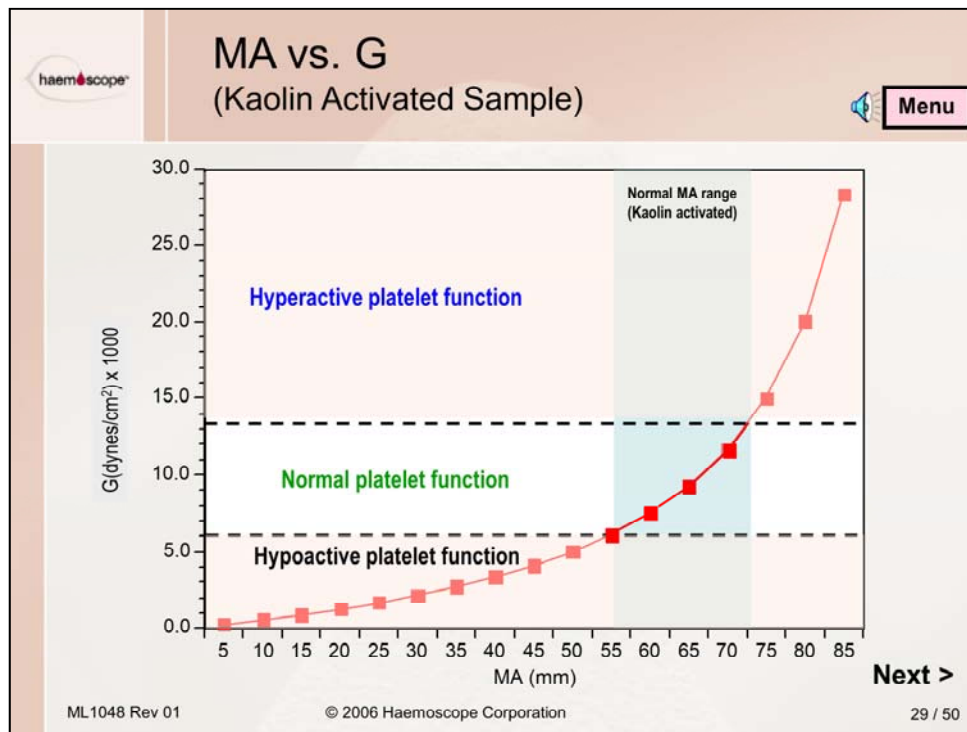
The G parameter provides another perspective on clot strength, and thus on overall platelet function. It is a transformation of the MA value, defining the shear elastic modulus strength of the clot, and is measured in units of dyn/cm².

G is calculated from MA using the equation:

$$G = (5000 \cdot MA) / (100 - MA)$$

Both MA and G represent the relationship between clot strength and platelet function. MA represents a linear relationship based on distance, whereas G displays it exponentially in the form of dyn/cm².

The exponential expression, G, is more sensitive to changes in platelet function than MA at higher MA values. For example, a change in MA from 50 to 67 mm (34% increase) represents a more than two-fold or 200% increase in the G value (5,000 to 10,200 dyn/cm²).



This is a plot of G versus MA showing the exponential increase of G relative to MA.



TEG Parameter Summary: Definitions


[Menu](#)

Clotting Time	R	The latency period from the time that the blood was placed in the TEG® analyzer until initial fibrin formation. Represents enzymatic reaction.
Clot Kinetics	K	A measure of the speed to reach 20 mm amplitude. Represents clot kinetics.
	Alpha	A measure of the rapidity of fibrin build-up and cross-linking (clot strengthening). Represents fibrinogen level.
Clot Strength	MA	A direct function of the maximum dynamic properties of fibrin and platelet bonding via GPIIb/IIIa. Represents maximum platelet function.
	G	A transformation of MA into dyn/cm ² .
Coagulation Index	CI	A linear combination of R, K, alpha, MA.
Clot Stability	LY30	A measure of the rate of amplitude reduction 30 min. after MA.
	EPL	Estimates % lysis based on amplitude reduction after MA.

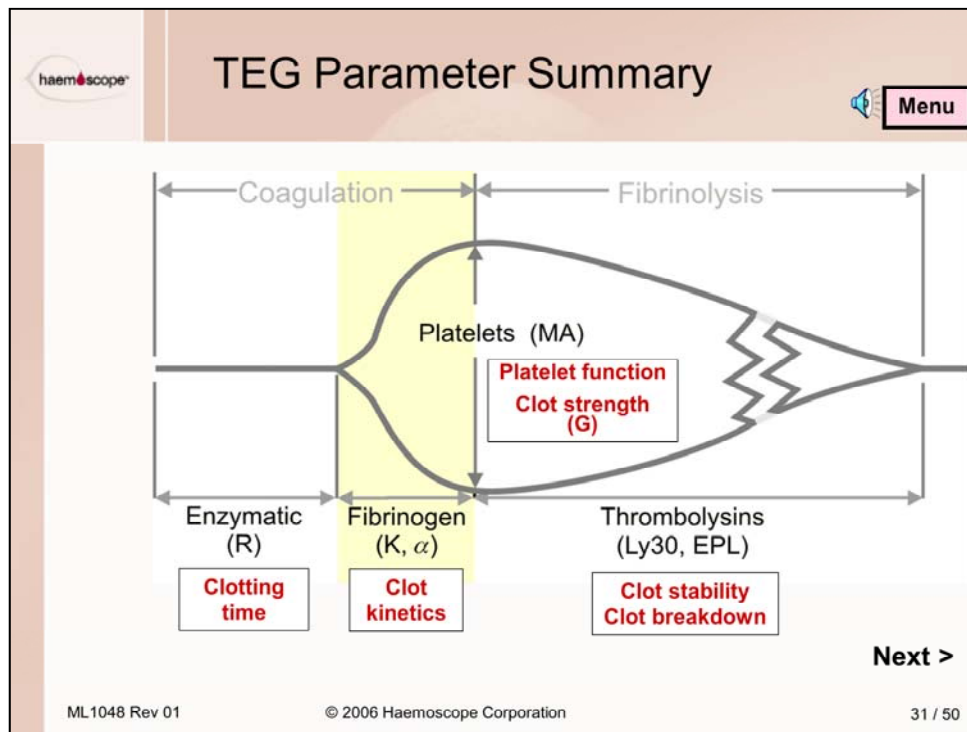
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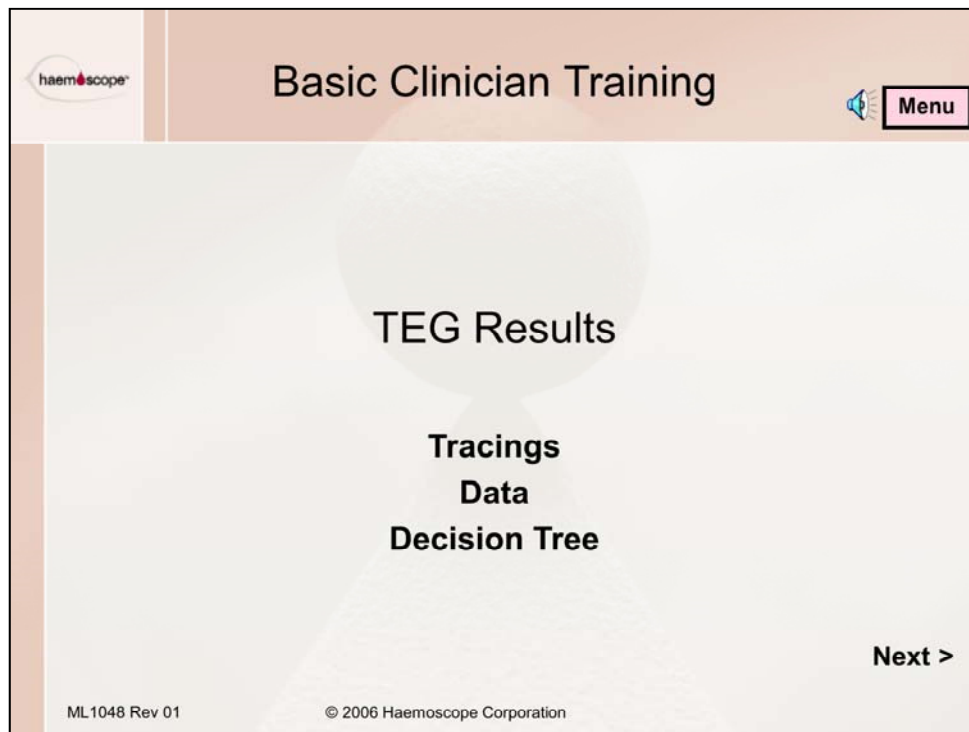
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This table summarizes the common TEG parameters for clotting time, clot kinetics, clot coagulation strength, overall coagulation index, and clot stability.

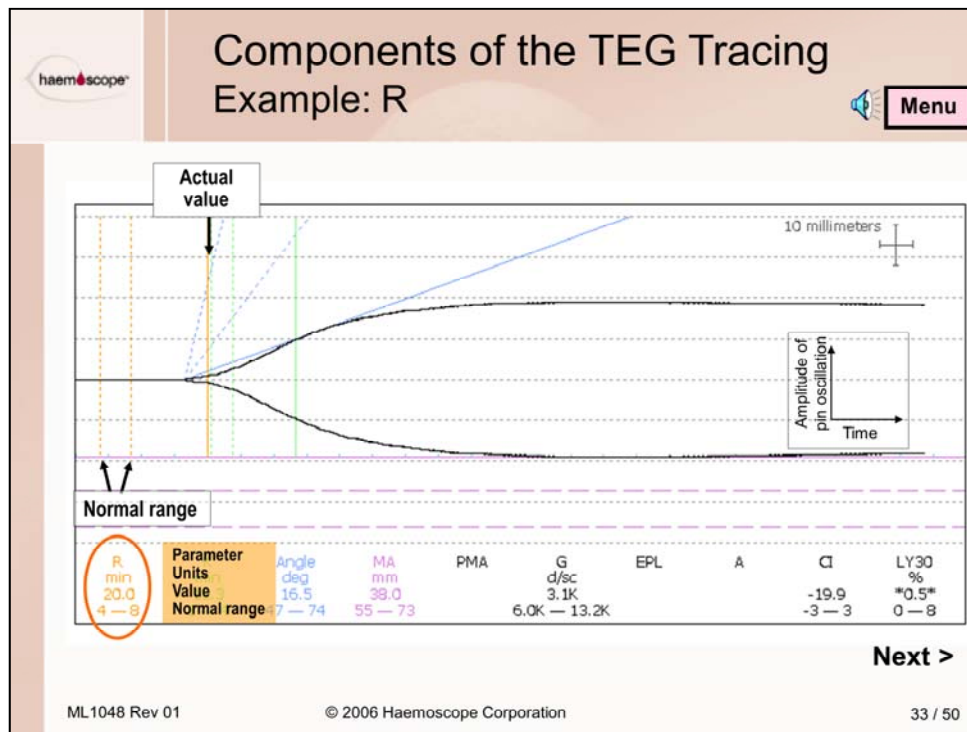


The relationship between the TEG parameters and the hemostatic components is demonstrated above. To summarize:

- The R value represents clotting time, and is associated with enzymatic reactions.
- The angle (α) and K values represent the rate of clot build-up, and indicate deficiencies in fibrinogen or in the interactions between the enzymatic pathways and platelet function.
- MA and G values represent clot strength, and mainly indicate platelet function (80-90%); fibrinogen also contributes (10-20%).
- LY30 and EPL values represent the rate of clot breakdown; they are related to the activity of thrombolysins such as tPA and plasmin.
- The CI value provides an indication of the global hemostatic state, reflecting both clot development and clot strength. It is a combination of R, K, angle, and MA.



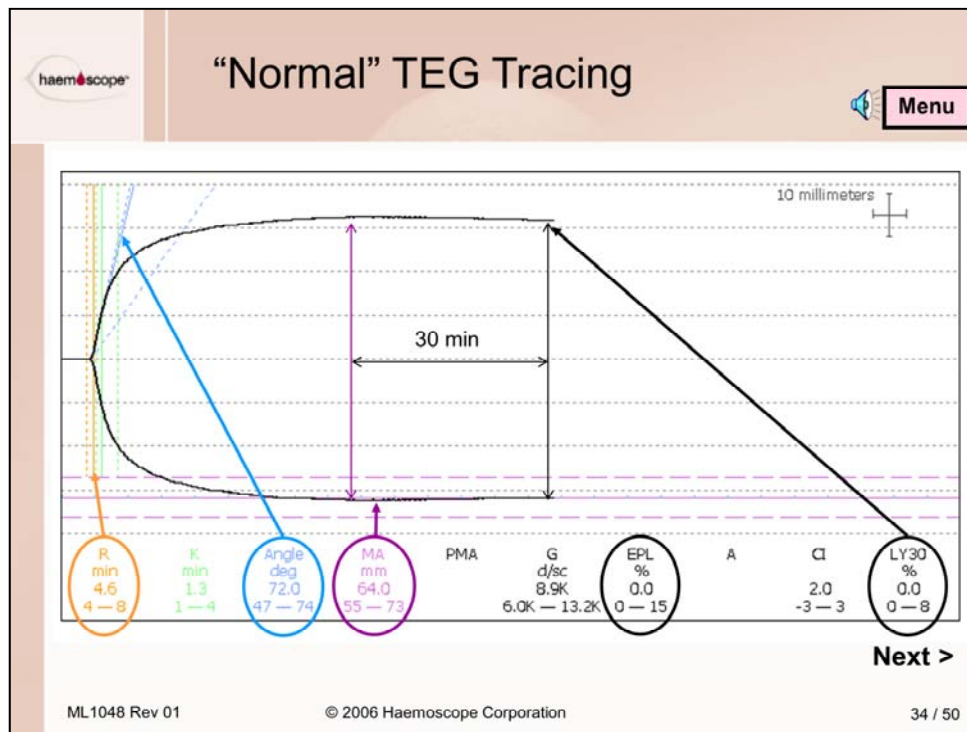
This section examines TEG tracings and data, giving examples of normal and abnormal results. It also explains the TEG decision tree.



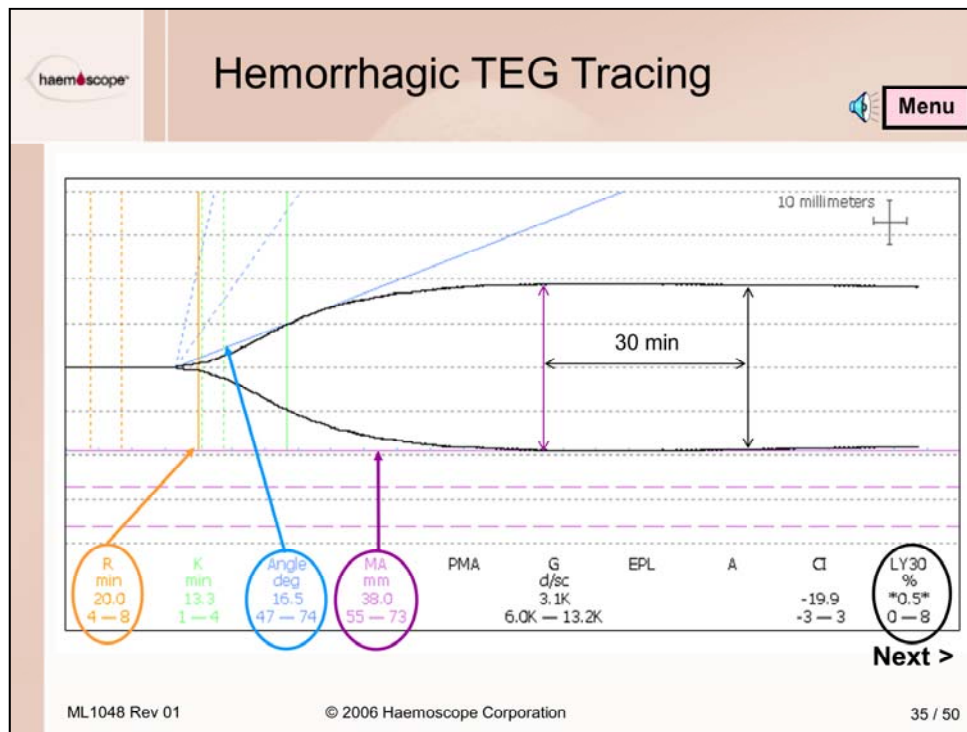
This is a representative TEG tracing. The numerical values of the parameters are along the bottom. Each parameter is designated with the letters previously described, as well as with the units of measurement for each.

Also associated with each parameter are the actual value and the normal range relative to a given sample type. For example, a kaolin activated sample will have a different normal range than a citrated kaolin activated sample.

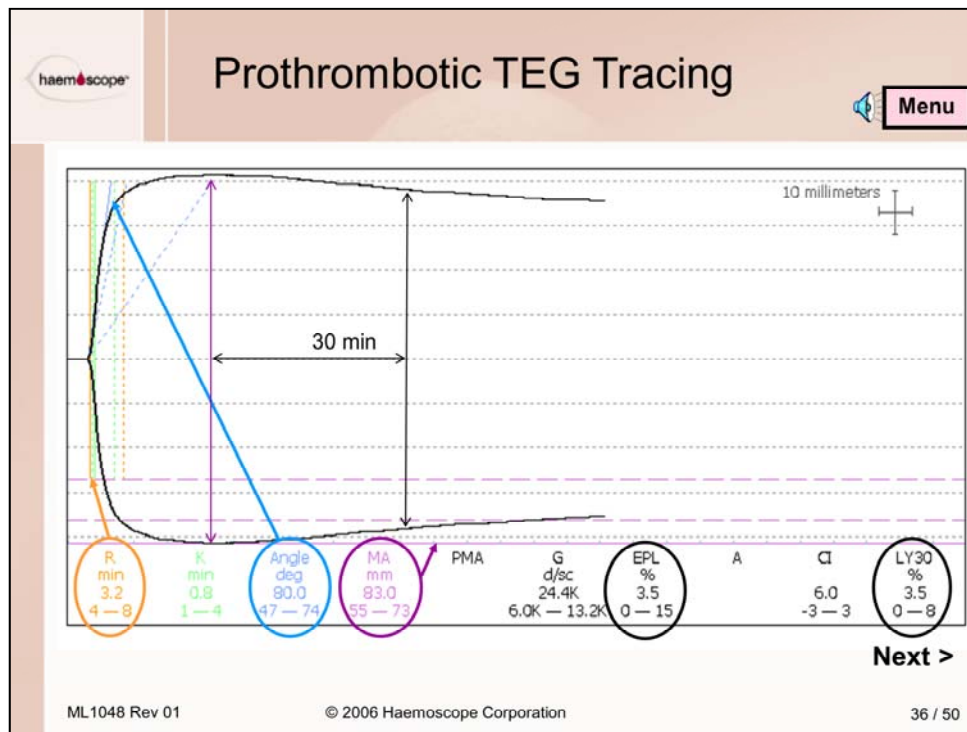
The normal range is also identified by dashed lines, which are color coordinated for each parameter. The actual value is designated by a solid line.



This example represents a normal TEG tracing, since all parameter values fall within their normal ranges.



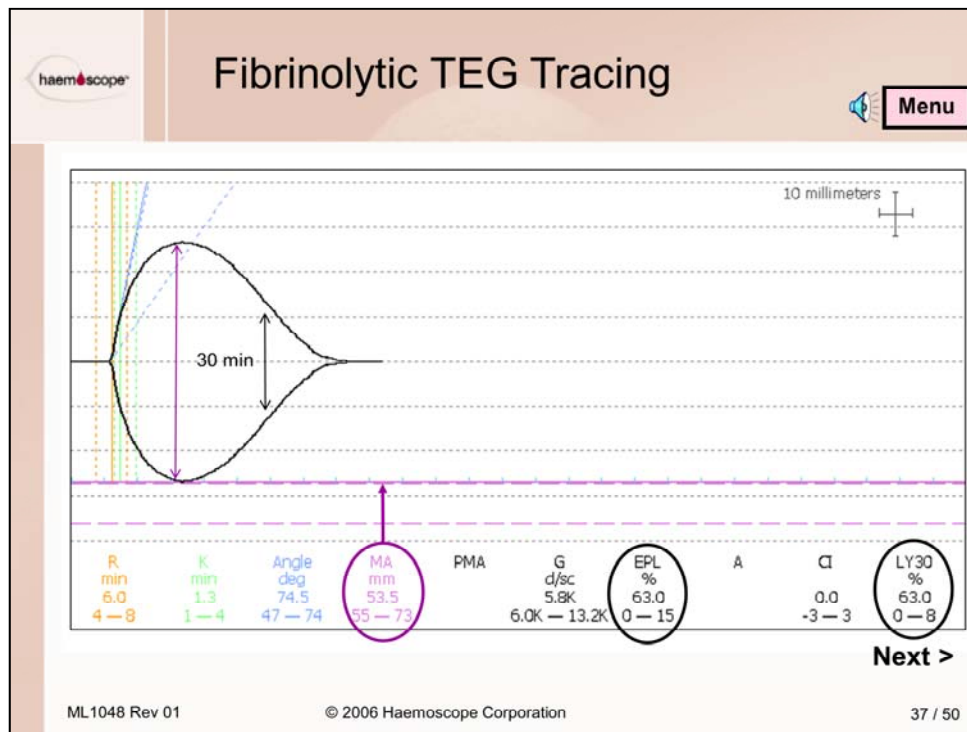
This is an example of a hemorrhagic TEG tracing. The R, angle, and MA values are all outside normal ranges. If this patient were bleeding, the likely cause would be a combination of enzymatic pathway and platelet dysfunction.



This is an example of a prothrombotic, or hypercoagulable, TEG tracing. Since the R, angle, CI, MA, and G values are all outside normal ranges, the prothrombotic state is likely due to a combination of enzymatic and platelet hyperactivity or hypercoagulability.

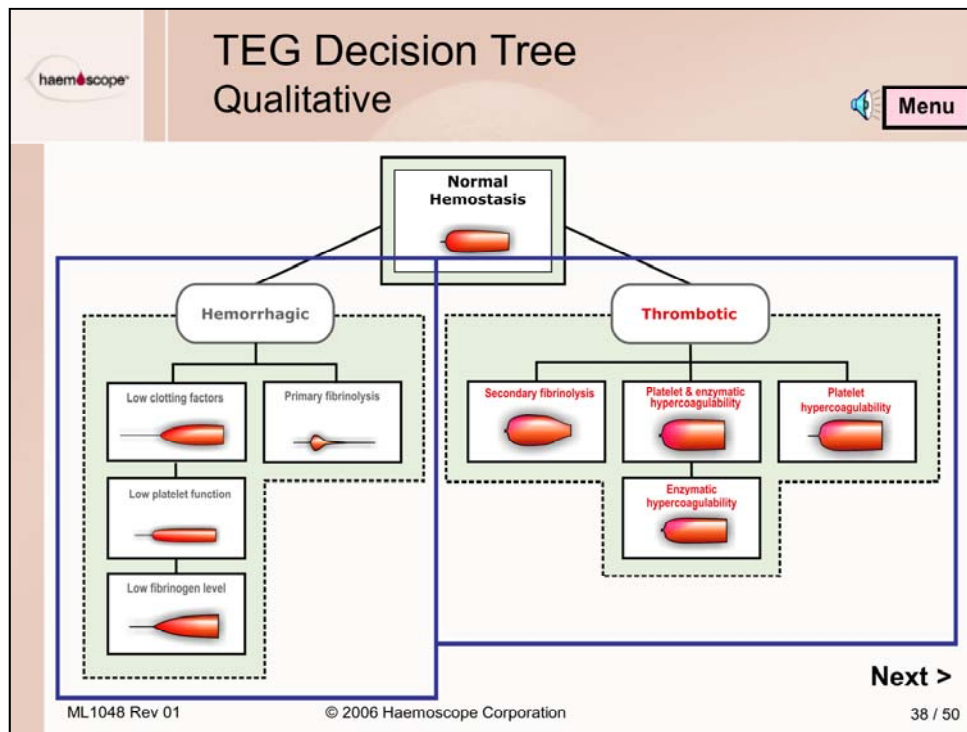
Note that the CI is 6.0, and the G is approximately double the upper limit of normal.

Although LY30 and EPL demonstrate a decrease in amplitude, the decrease is within normal ranges. Thus, the degree of fibrinolysis is normal.



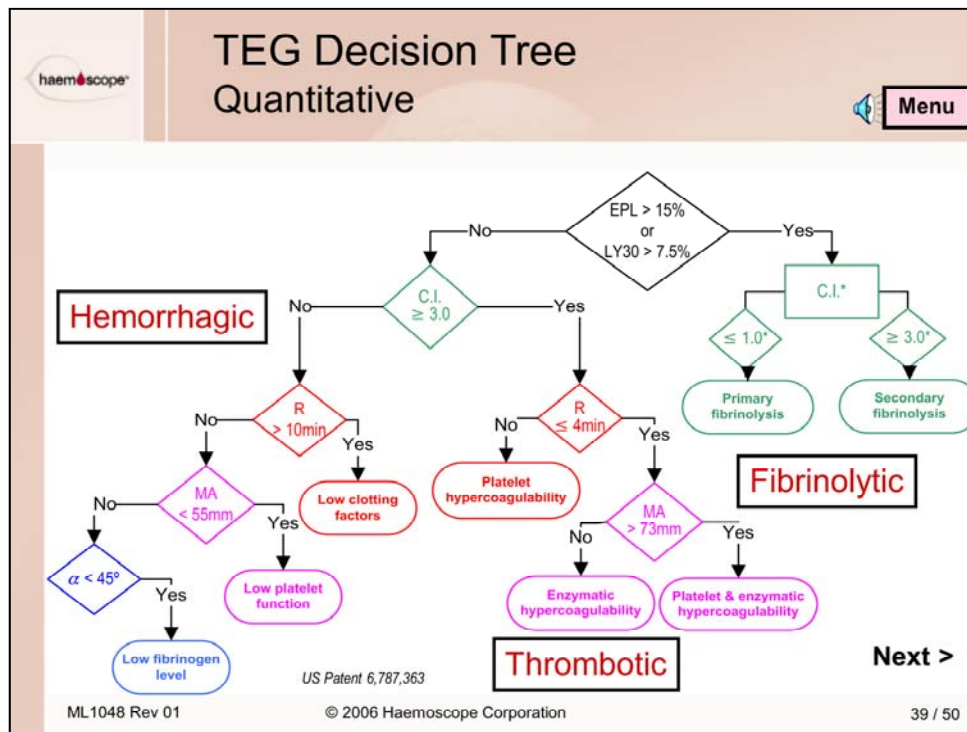
This is an example of a fibrinolytic TEG tracing. Both the LY30 and EPL values exceed normal ranges.

Since the CI value is less than 1.0, the correct interpretation is primary fibrinolysis. The low MA value suggests that fibrinolysis may be limiting overall clot formation due to an excess of tPA and plasmin.



The qualitative TEG decision tree demonstrates the general shape of tracings for different hemostatic conditions.

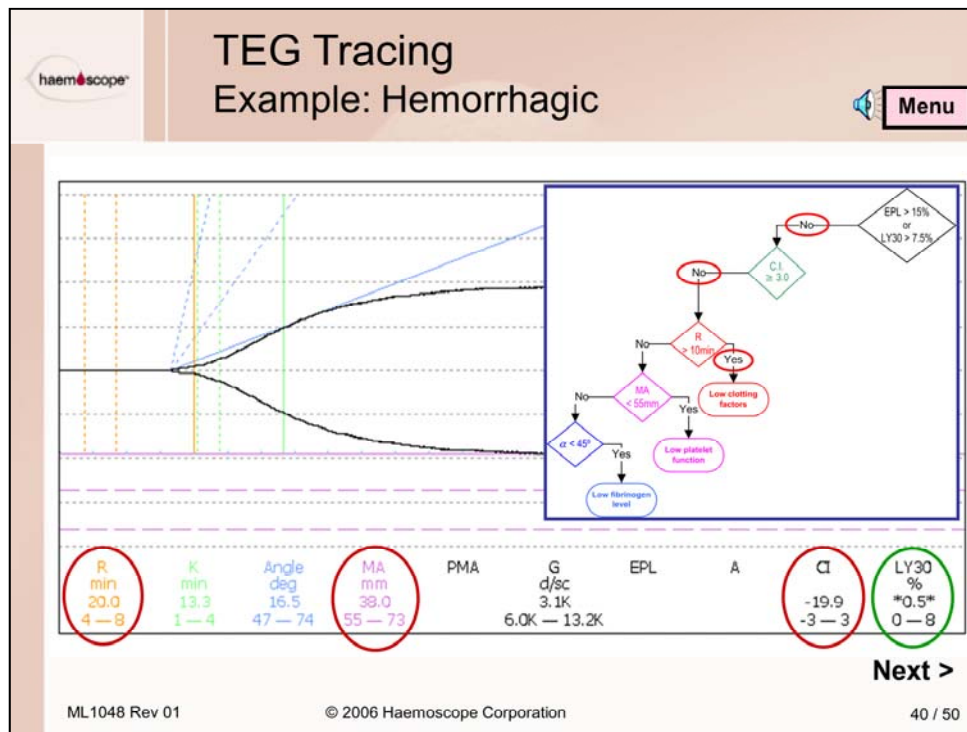
The decision tree is divided into two categories, hemorrhagic and thrombotic. The subdivisions represent different causes of each hemostatic state.



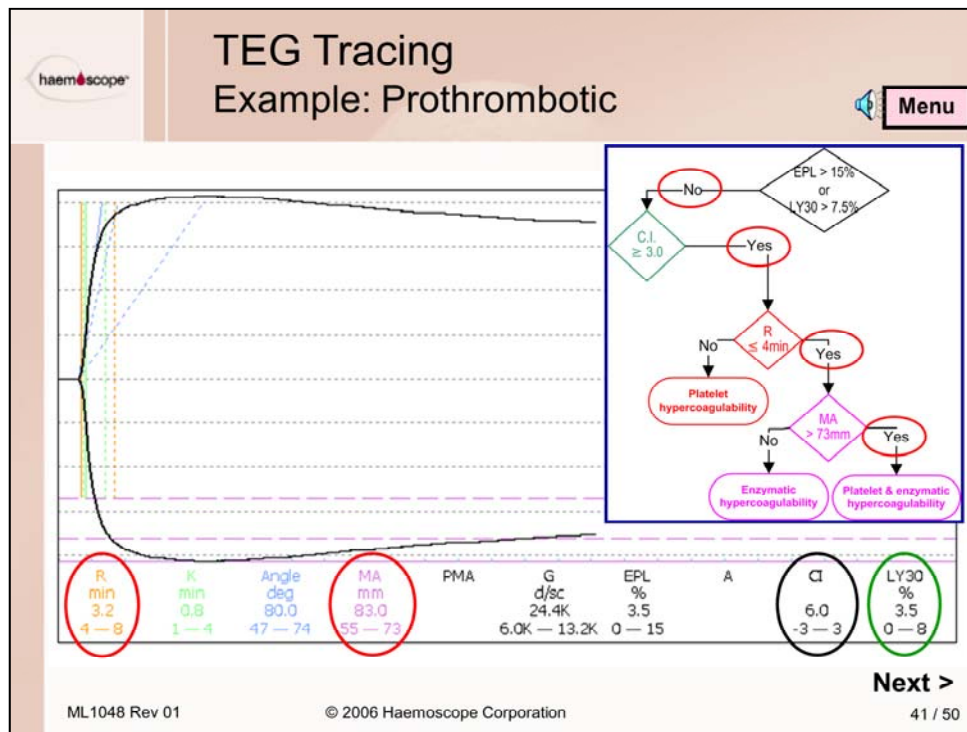
The quantitative TEG decision tree uses the values of the main TEG parameters to determine potential causes for a given hemostatic state.

There are three categories of hemostatic conditions: hemorrhagic, thrombotic, and fibrinolytic. The subdivisions provide more specific causes for each condition.

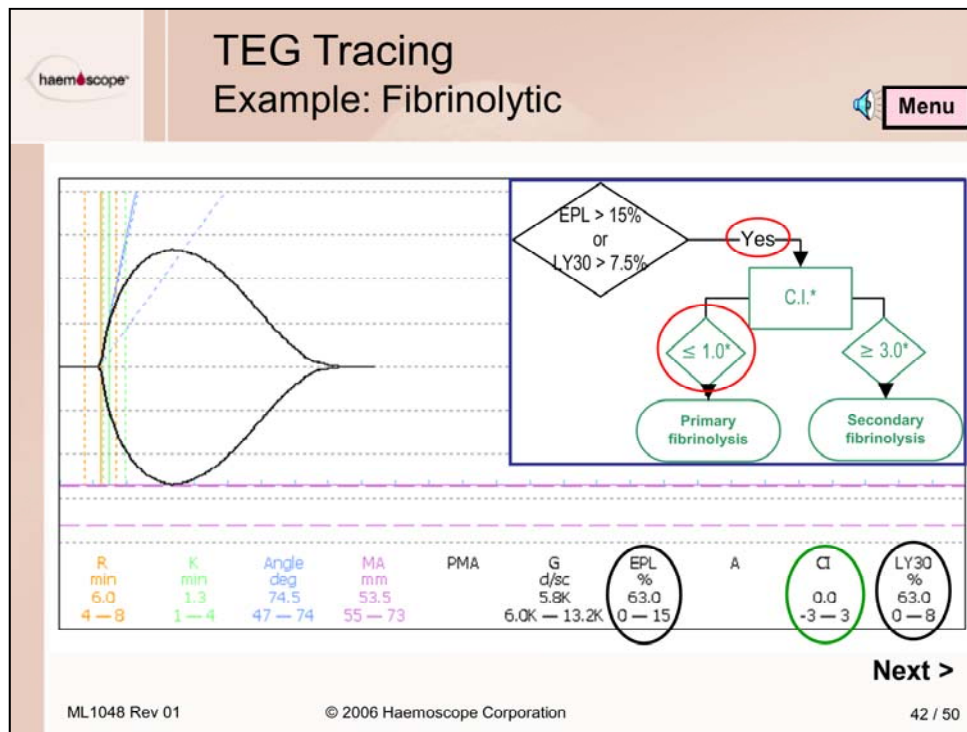
This quantitative decision tree is a useful tool for novice users of the TEG analyzer. The following slides demonstrate how to use it.



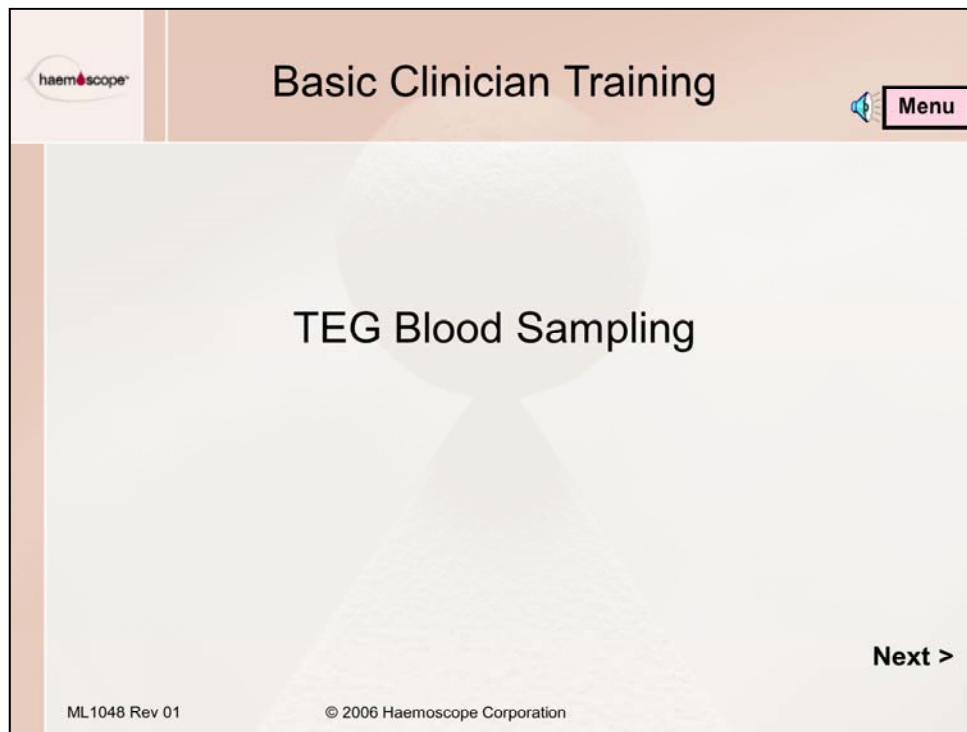
This is an example of a hemorrhagic TEG tracing. LY30, CI, and R values suggest that bleeding is due in part to low clotting factors. The low MA also suggests possible platelet dysfunction.



This is an example of a prothrombotic TEG tracing. LY30 and CI values lead to the prothrombotic part of the decision tree. R and MA values suggest that the cause of the prothrombotic state is a combination of enzymatic and platelet hyperactivity.



This is an example of a fibrinolytic TEG tracing. In this case, LY30 and EPL values both exceed the normal range. The CI value of less than 1.0 suggests primary fibrinolysis.



This section discusses TEG blood sampling.



TEG Blood Sampling

 Menu

- Blood samples
 - Arterial or venous
 - Samples should be consistent

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Patient blood samples can be either arterial or venous. However, since there may be some differences in clotting between arterial and venous blood, it is recommended that for any given patient, the blood sample be consistently one or the other.



TEG Blood Sampling Native

 Menu

- Non-modified blood samples
 - Assayed 4 minutes
 - TEG software based upon assay at 4 minutes

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Native blood is defined as non-modified blood. Native samples are assayed at four minutes after drawing the sample. Normal values in the TEG analytical software are based upon assay at four minutes.



TEG Blood Sampling
Modified

 Menu

- Activator
 - Reduces variability
 - Reduces running time
 - Maximizes thrombin generation
- Kaolin
 - Activates intrinsic pathway
 - Used for normal TEG analysis
- Tissue factor
 - Specifically activates extrinsic pathway

Next >

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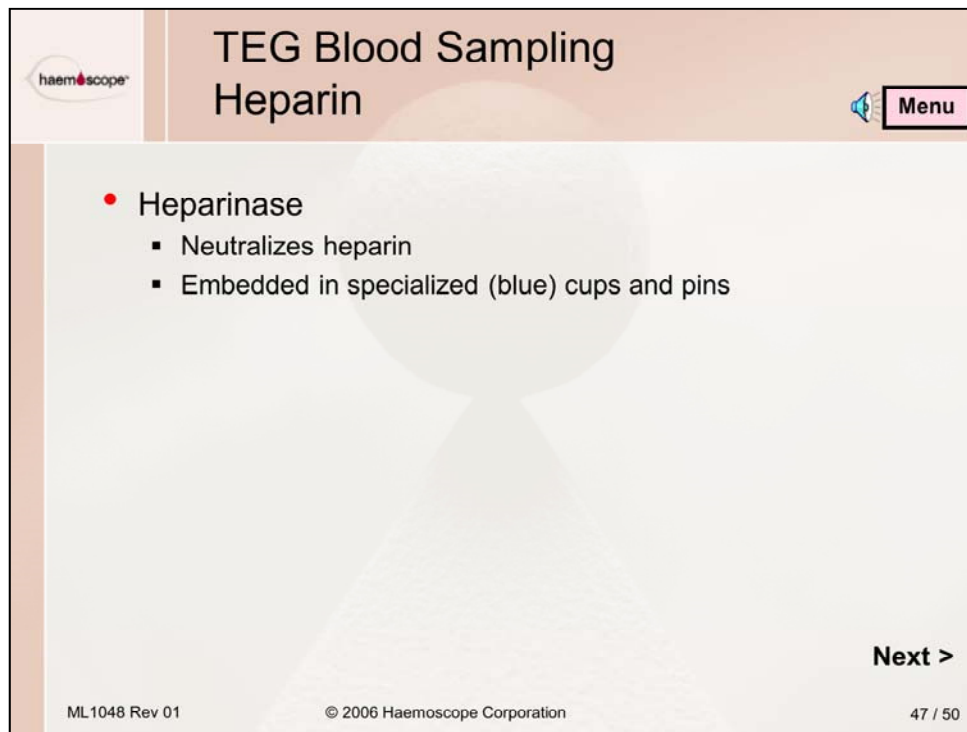
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Whole blood samples can be modified by the addition of reagents to the sample cup or by the use of treated sample cups. It is recommended that **all** blood samples be activated. Activators are used to reduce both variabilities and running time of native whole blood samples. Since a critical mass of thrombin must be generated to cleave fibrinogen into fibrin, activation maximizes thrombin generation and speeds up the entire hemostatic process.

Kaolin is the primary activator used for TEG analysis. It activates the intrinsic pathway, which leads to thrombin formation and subsequent clot formation *in vitro*.

Tissue factor is also available as an activator. It specifically activates the extrinsic pathway, thus simulating the initiation of coagulation *in vivo*.

A presentation slide titled "TEG Blood Sampling Heparin". The slide features a light orange header with the "haemoscope" logo on the left and a "Menu" button on the right. The main content area is white with a large, faint background image of a person in a white lab coat. The text on the slide is as follows:

**TEG Blood Sampling
Heparin**

- **Heparinase**
 - Neutralizes heparin
 - Embedded in specialized (blue) cups and pins

Next >

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Heparin may be present in the blood of patients undergoing anticoagulation. If heparin is known or suspected to be in the blood sample, heparinase must be used to neutralize its effect. Failure to neutralize the heparin will result in a lack of significant clot formation (see Module 3).

Heparinase is embedded in specialized (blue) cups and pins. These should be used any time a blood sample may have been exposed to heparin .



TEG Blood Sampling Citrated

 **Menu**

- Citrated tubes are used
- Recalcified before analysis
- Standardize time between blood draw and running test
- Specific platelet activators are required to demonstrate effect of antiplatelet agents

Next >

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Citrated blood samples are used when there will be a delay in the time between drawing the sample from the patient and running the test on the TEG analyzer.

Citrated samples are drawn into citrated tubes (3.2% sodium citrate) for transport to the TEG analyzer, typically located in the laboratory. The blood must then be recalcified with calcium chloride before running the analysis. The time between drawing the blood and running the test should be standardized (e.g. thirty minutes after the draw) to eliminate any artifacts due to calcium transients within the platelets, blood cells, and other living cells.

Standard TEG analysis does not measure the effect of antiplatelet drugs. Specific platelet activators are required to demonstrate the effect of antiplatelet agents on clot formation. More information on these activators is found in Module 6, PlateletMapping Assays.



Sample Type Designations


Menu

Whole blood + kaolin

Sample type	Conditions	Time to run sample	Sample prep
K (kaolin activated)	No anticoagulation	< 4 min	Clear cup & pin
KH (kaolin + heparinase)	With heparin	< 4 min	Blue cup & pin (coated with heparinase)
CK (citrate + kaolin)	With citrate	< 120 min	Add calcium chloride Clear cup and pin
CKH (citrate + kaolin + heparinase)	With citrate and heparin	< 120 min	Add calcium chloride Blue cup & pin

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This table provides designations, definitions, and conditions of the different sample types. As shown, all blood samples are activated with kaolin. The use of heparinase depends on whether or not the patient is on heparin at the time of the blood draw.

Citrated samples are recommended for all blood samples that will require more than four minutes before being run. Citrated samples should be run at a consistent delay time throughout the institution, for example 15 minutes.

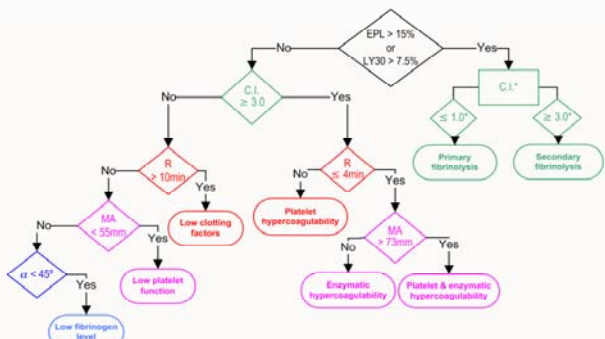


Summary



Menu

- The TEG technology measures the complex balance between hemorrhagic and thrombotic systems.
- The decision tree is a tool to identify coagulopathies and guide therapy in a standardized way.



```

graph TD
    A{EPL > 15%  
or  
LY30 > 7.5%} -- No --> B{CI ≥ 3.0}
    A -- Yes --> C{CI*}
    B -- No --> D{R > 10min}
    B -- Yes --> E{R ≤ 4min}
    C -- ≤ 1.0* --> F(Primary fibrinolysis)
    C -- ≥ 3.0* --> G(Secondary fibrinolysis)
    D -- No --> H{MA < 55mm}
    D -- Yes --> I(Low clotting factors)
    E -- No --> J{Platelet hypercoagulability}
    E -- Yes --> K{MA > 73mm}
    H -- No --> L{α < 45°}
    H -- Yes --> M(Low platelet function)
    L -- Yes --> N(Low fibrinogen level)
    K -- No --> O(Enzymatic hypercoagulability)
    K -- Yes --> P(Platelet & enzymatic hypercoagulability)
    
```

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In summary, the TEG technology measures the complex balance of the components of hemostasis at all phases of the hemostatic process. It functions well in a laboratory based environment, but may also be used in point of care situations, since samples do not require processing; they can be prepared to suit the needs of the institution or hospital.

The parameters generated from the TEG tracing relate to the hemostatic process:

- R for time to the initial clot
- Alpha and K for rate of clot build-up
- MA and G for clot strength
- LY30 and EPL for clot breakdown
- CI for the global hemostatic state

The values of these parameters aid the clinician in determining the type and magnitude of hemostatic abnormalities.

The TEG decision tree can be an invaluable tool for identifying coagulopathies and guiding therapy in a personalized, yet standardized manner. This helps clinicians deliver the appropriate treatment for each patient, thus improving patient care.



Basic Clinician Training

Menu

TEG Parameters Hemostasis Monitoring

Test your knowledge of TEG parameters and hemostasis monitoring by answering the questions on the slides that follow.

Begin Exercises

Skip Exercises

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Exercise 1: TEG Parameters

The R value represents which of the following phases of hemostasis?

- a. Platelet adhesion
- b. Activation of coagulation pathways and initial fibrin formation
- c. Buildup of platelet-fibrin(ogen) interactions
- d. Completion of platelet-fibrin(ogen) buildup
- e. Clot lysis

[Answer](#)[Next](#)[Menu](#)

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Exercise 2: TEG Parameters

Select the TEG parameters that demonstrate kinetic properties of clot formation. (Select all that apply)

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 3: TEG Parameters

The rate of clot strength buildup is demonstrated by which of the following TEG parameters?

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 4: TEG Parameters

Which of the following TEG parameters will best demonstrate the need for coagulation factors (e.g. FFP)?

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 5: TEG Parameters

Clot strength is dependent upon which of these hemostatic components?

- a. 100% platelets
- b. 80% platelets, 20% fibrin
- c. 50% platelets, 50% fibrin
- d. 20% platelets, 80% fibrin
- e. 100% fibrin

[Answer](#)[Next](#)[Menu](#)

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Exercise 6: TEG Parameters

Which of the following TEG parameters demonstrate a structural property of the clot?
(Select all that apply)

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 7: TEG Parameters

Because the TEG is a whole blood hemostasis monitor, a low MA demonstrating low platelet function may also influence which of the following TEG parameters?
(Select all that apply)

- a. R
- b. Angle (α)
- c. LY30
- d. CI
- e. None of the above

[Answer](#)[Next](#)[Menu](#)

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Exercise 8: TEG Parameters

Clot stability is determined by which of the following TEG parameters?

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 9: TEG Parameters

Which of the following reagents should be used to provide the information necessary to determine if heparin is the cause of bleeding in a patient?

- a. R value: Kaolin with heparinase
- b. R value: Kaolin vs. Kaolin with heparinase
- c. MA value: Kaolin with heparinase
- d. MA value: Kaolin vs. kaolin with heparinase

[Answer](#)[Next](#)[Menu](#)

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Exercise 10: TEG Parameters

Which of the following parameters provides an indication of the global coagulation status of a patient?

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

[Answer](#)[Next](#)[Menu](#)

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Exercise 11: TEG Parameters

Which of the following statements are true regarding the PT and aPTT tests? (select all that apply)

- a. Measure coagulation factor interaction in solution
- b. Measure platelet contribution to thrombin generation
- c. Measure the influence of thrombin generation on platelet function
- d. Use fibrin formation as an end point

[Answer](#)[Next](#)[Menu](#)

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Exercise 12: TEG Parameters

The TEG analyzer can monitor all phases of hemostasis except which of the following? (select all that apply)

- a. Initial fibrin formation
- b. Fibrin-platelet plug construction
- c. Platelet adhesion
- d. Clot lysis

Answer

End

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Answers to Exercise 1: TEG Parameters

The R value represents which of the following phases of hemostasis?

- a. Platelet adhesion
- b. Activation of coagulation pathways and initial fibrin formation**
- c. Buildup of platelet-fibrin(ogen) interactions
- d. Completion of platelet-fibrin(ogen) buildup
- e. Clot lysis

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Answers to Exercise 2: TEG Parameters

Select the TEG parameters that demonstrate kinetic properties of clot formation. (select all that apply)

- a. R
- b. Angle (α)**
- c. MA
- d. LY30
- e. CI

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Answers to Exercise 3: TEG Parameters

The rate of clot strength buildup is demonstrated by which of the following TEG parameters?

- a. R
- b. Angle (α)**
- c. MA
- d. LY30
- e. CI

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Answers to Exercise 4: TEG Parameters

Which of the following TEG parameters will best demonstrate the need for coagulation factors (e.g. FFP)?

- a. R**
- b. Angle (α)
- c. MA
- d. LY30
- e. CI

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Answers to Exercise 5: TEG Parameters

Clot strength is dependent upon which of these hemostatic components?

- a. 100% platelets
- b. 80% platelets, 20% fibrin**
- c. 50% platelets, 50% fibrin
- d. 20% platelets, 80% fibrin
- e. 100% fibrin

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Answers to Exercise 6: TEG Parameters

Which of the following TEG parameters demonstrate a structural property of the clot?
(select all that apply)

- a. R
- b. Angle (α)
- c. MA (demonstrates maximum clot strength)**
- d. LY30 (demonstrates clot breakdown or the structural stability of the clot)**
- e. CI

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Answers to Exercise 7: TEG Parameters

Because the TEG is a whole blood hemostasis monitor, a low MA demonstrating low platelet function may also influence which of the following TEG parameters? (select all that apply)

- a. **R** – Thrombin generation occurs mainly on the surface of platelets; therefore, a defect in platelet function may slow the rate of thrombin generation and fibrin formation.
- b. **Angle (α)** – A defect in platelet function may slow the rate of formation of platelet-fibrin interactions, thereby slowing the rate of clot buildup.
- c. LY30
- d. **CI**
- e. None of the above

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Answers to Exercise 8: TEG Parameters

Clot stability is determined by which of the following TEG parameters?

- a. R
- b. Angle (α)
- c. MA
- d. LY30**
- e. CI

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Answers to Exercise 9: TEG Parameters

Which of the following reagents should be used to provide the information necessary to determine if heparin is the cause of bleeding in a patient?

- a. R value: Kaolin with heparinase
- b. R value: Kaolin vs. Kaolin with heparinase**
- c. MA value: Kaolin with heparinase
- d. MA value: Kaolin vs. kaolin with heparinase

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Answers to Exercise 10: TEG Parameters

Which of the following parameters provides an indication of the global coagulation status of a patient?

- a. R
- b. Angle (α)
- c. MA
- d. LY30
- e. **CI** (Coagulation Index — a linear combination of the R, K, angle, and MA)

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Answers to Exercise 11: TEG Parameters

Which of the following statements are true regarding the PT and aPTT tests? (select all that apply)

- a. Measure coagulation factor interaction in solution**
- b. Measure platelet contribution to thrombin generation
- c. Measure the influence of thrombin generation on platelet function
- d. Use fibrin formation as an end point**

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Answers to Exercise 12: TEG Parameters

The TEG analyzer can monitor all phases of hemostasis except which of the following? (select all that apply)

- a. Initial fibrin formation
- b. Fibrin-platelet plug construction
- c. Platelet adhesion** — this is a vascular mediated event that occurs *in vivo*, but not *in vitro*
- d. Clot lysis

End

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Basic Clinician Training

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End of Module 2

[References](#)

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