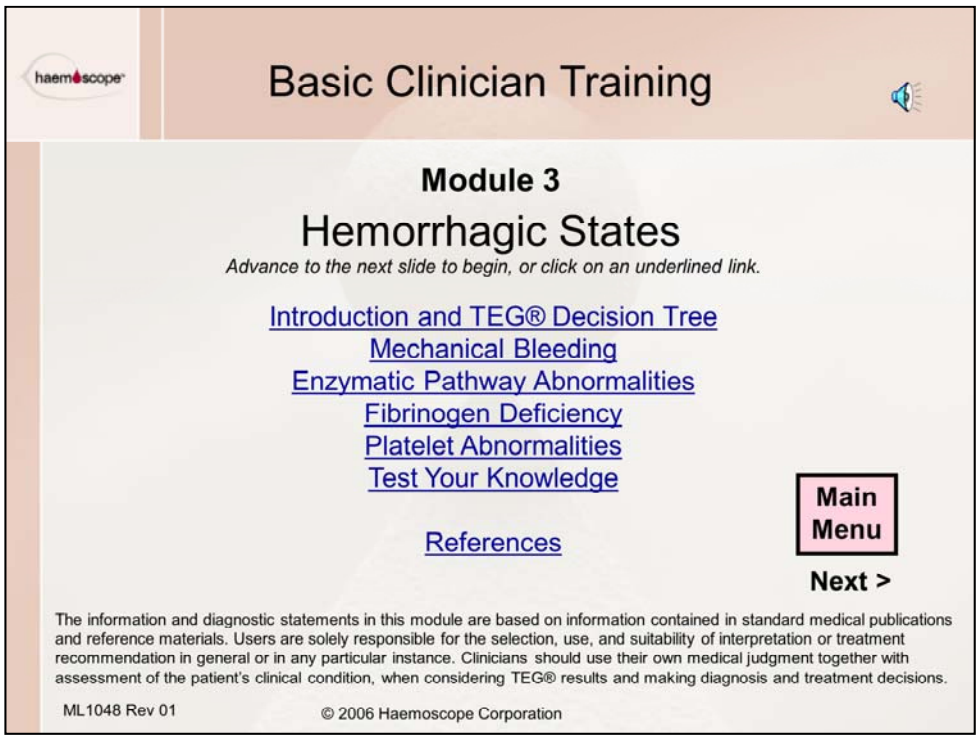
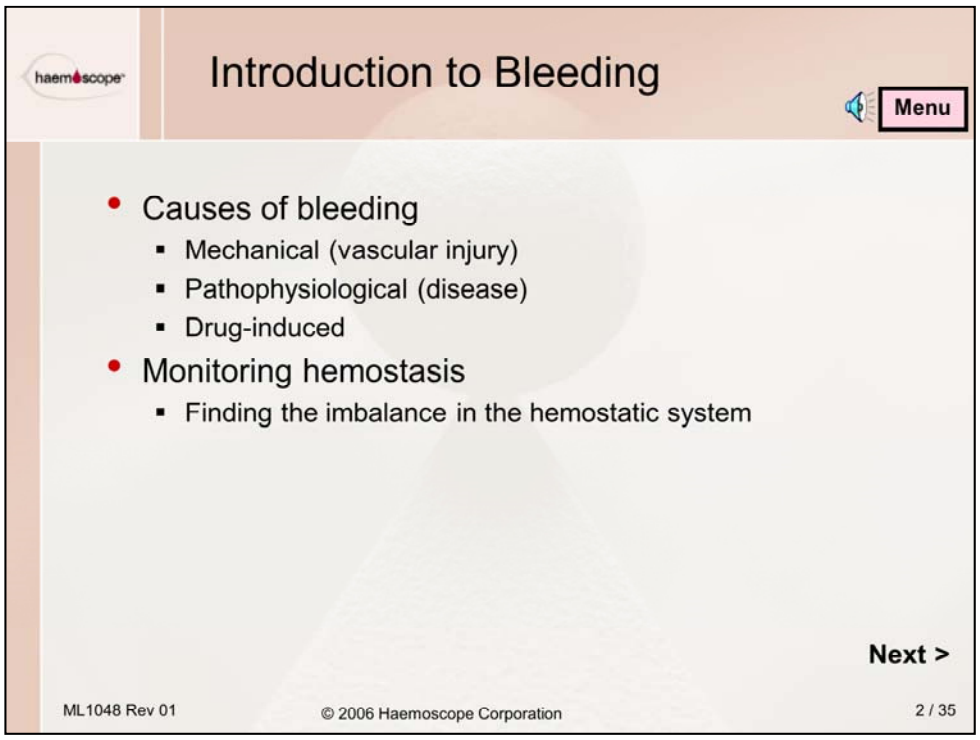


This module covers the interpretation of TEG tracings from bleeding patients, along with a brief overview of the causes of bleeding.

Advance to the next slide to begin the presentation, or click on an underlined link to proceed to a specific topic.



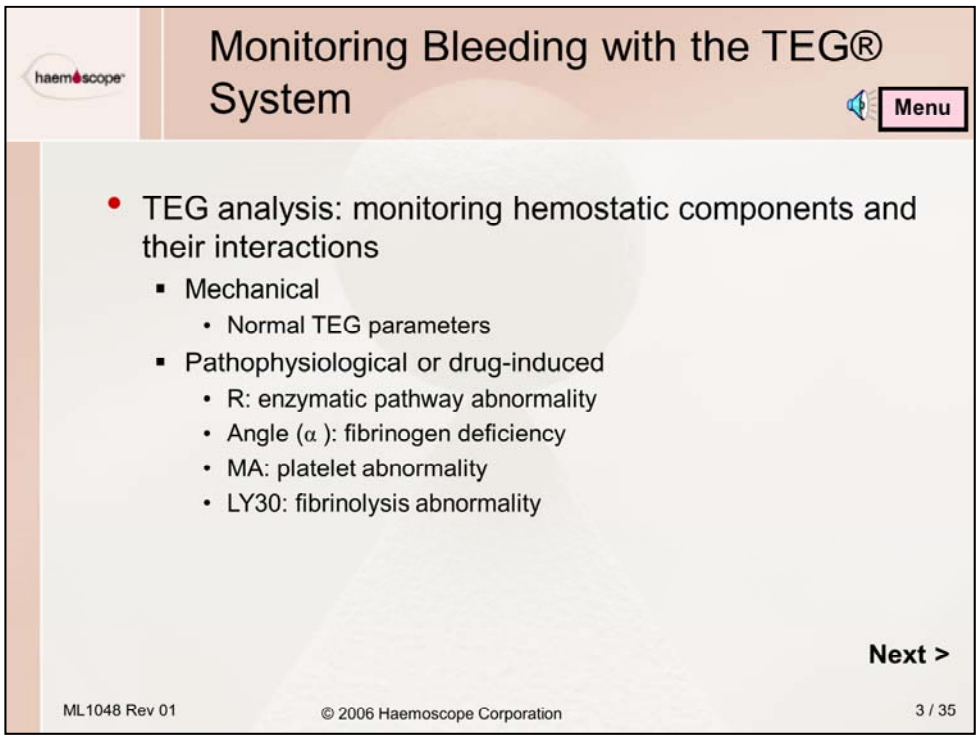
There are many causes of bleeding. The three major categories are mechanical, pathophysiological, and drug-induced.

Mechanical bleeding occurs when there is a disruption in the integrity of the vascular wall. This can be from either trauma or a surgical situation.

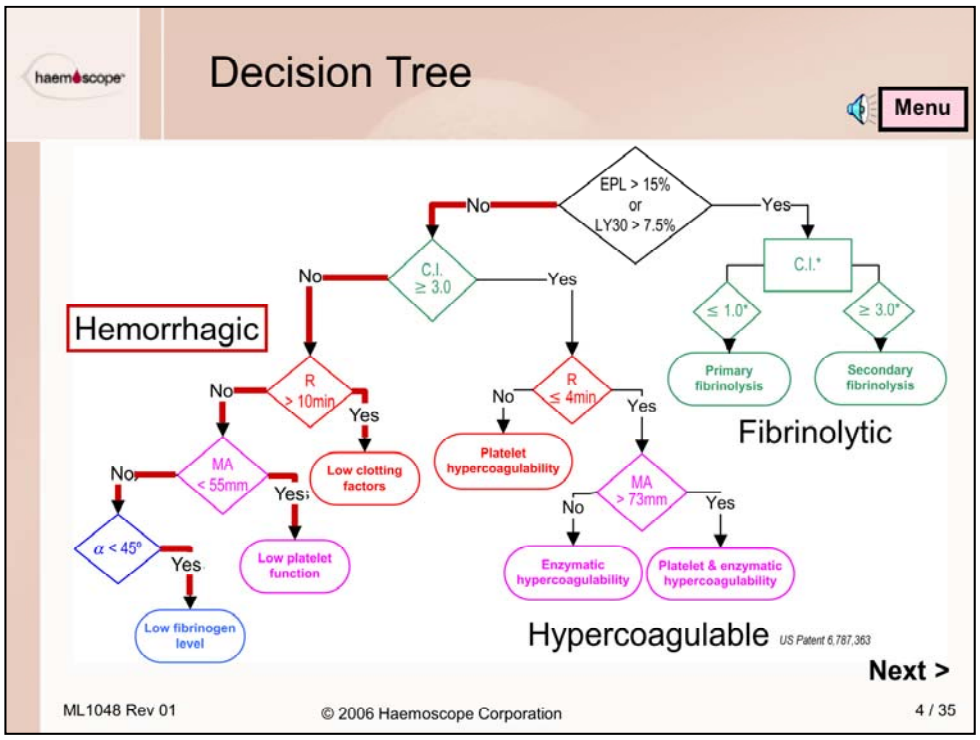
Pathophysiological bleeding occurs when a disease state or genetic condition creates a shift in the hemostatic balance towards the anticoagulant and antithrombotic components. Liver disease and hemophilia are examples; both increase the risk of bleeding.

Drugs can also cause shifts in the hemostatic balance, especially platelet inhibitors and anticoagulants.

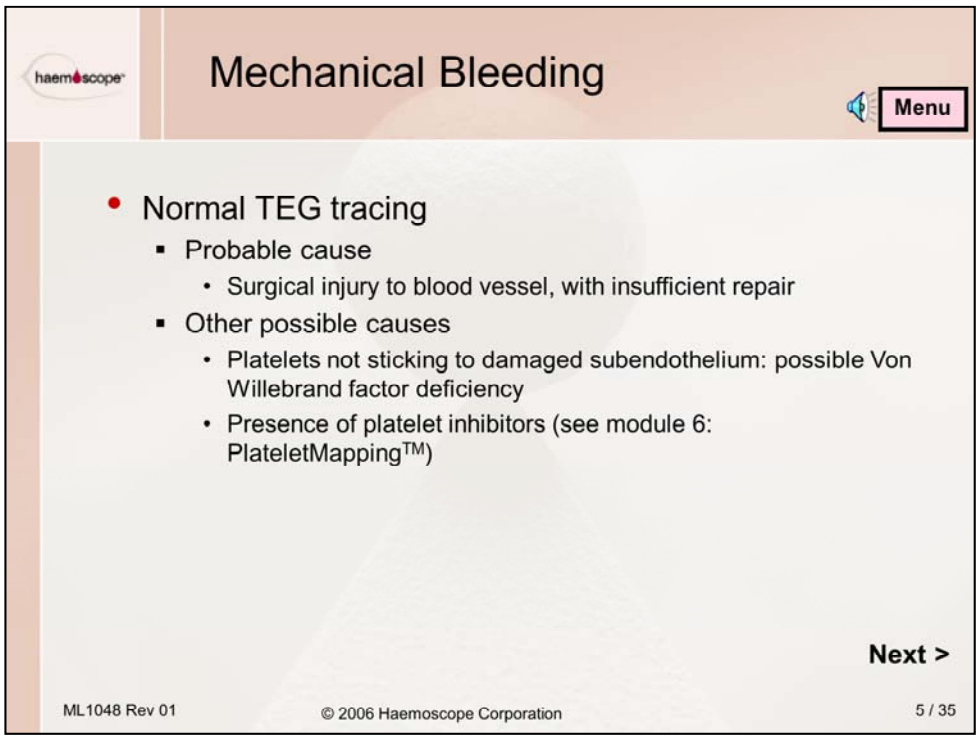
It is often difficult to decide on a specific cause of bleeding even after assessing the severity of vascular injury, disease state, and types of medication. One reason to monitor hemostasis is to determine where the imbalance within the hemostatic system lies and, if possible, to establish the cause of that imbalance. This information, along with an understanding of a patient's history and current status, can help in determining the best treatment options.



The TEG analyzer is used to monitor all categories of bleeding. Mechanical bleeding produces a tracing in which all parameters are normal. Depending on the hemostatic defect, pathophysiological and drug-induced bleeding are indicated by abnormalities in the R, angle, MA, and LY30 values.



The quantitative TEG decision tree is useful for determining where hemostatic defects occur, and it provides information on interpreting tracings that indicate bleeding conditions. In this module, the hemorrhagic branch of the decision tree will be examined.



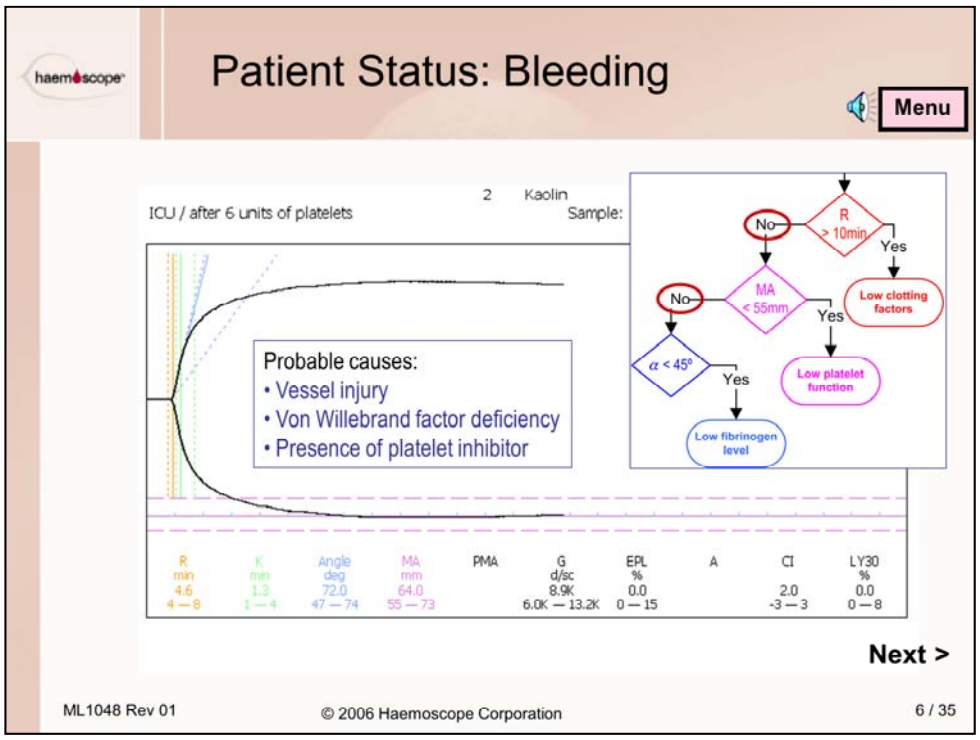
Mechanical bleeding is characterized by a patient who is bleeding, yet whose blood sample produces a normal TEG tracing, with the hemostatic components in balance. Such a tracing suggests that coagulation pathways, platelet s, and fibrinolytic pathways are not responsible. This further suggests that the probable cause is injury to the blood vessel due to trauma, surgical damage, or rupture of the vessel. Repair of the vessel is therefore required to reduce bleeding.

Although vessel injury is typically the primary cause of bleeding when the TEG tracing is normal, there are other conditions that could produce a similar result. In order for platelets to form a platelet plug, they must first adhere to the subendothelium at the site of the injury. Interaction between the platelet GPIb receptor and von Willebrand factor is required for this adhesion. Since the TEG analyzer monitors only interactions in the blood, a von Willebrand factor deficiency or a dysfunction of the platelet GPIb receptor will not be demonstrated; the tracing will be normal, but the patient will be bleeding.

In addition, the presence of platelet inhibitors may not be demonstrated due to maximum generation of thrombin *in vitro*. Large quantities of thrombin can activate platelets and override the inhibition of receptors and activation pathways. Standard TEG tracings may appear normal *in vitro*, while bleeding occurs *in vivo*. More information on how to use the TEG system to measure the effects of platelet inhibitors is available in Module 6.

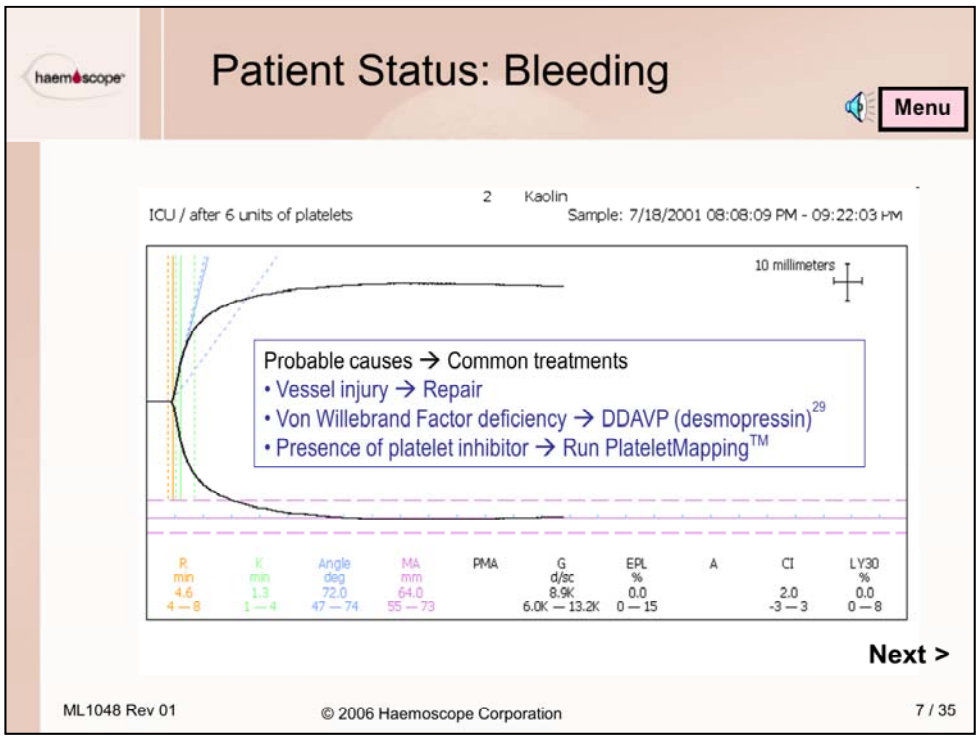
Since a TEG tracing can appear normal under a variety of bleeding conditions, it is necessary to have a thorough knowledge of the patient's history and status when trying to determine the best

treatment option. In other words, TEG analysis does not work alone.



This TEG tracing, although within normal limits, is from a patient who is bleeding. If this were a trauma or surgical patient, such a tracing would suggest vessel injury.

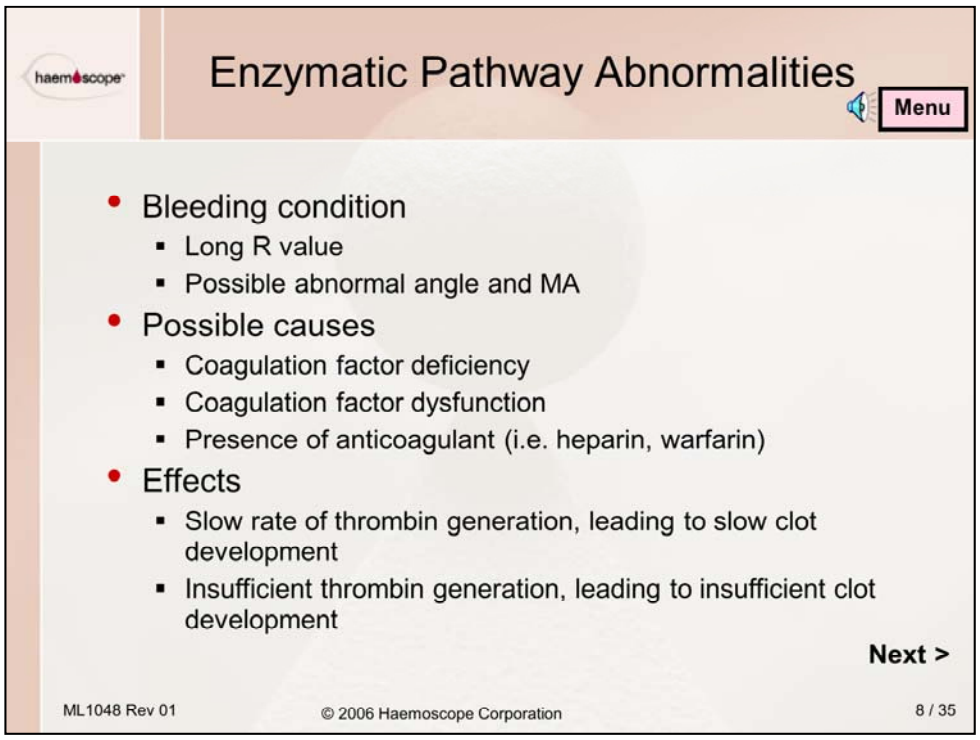
A review of patient history and other laboratory values will provide additional information on the actual cause of bleeding if a specific vessel injury cannot be found.



Once the probable cause of bleeding has been determined, the best treatment can be administered.

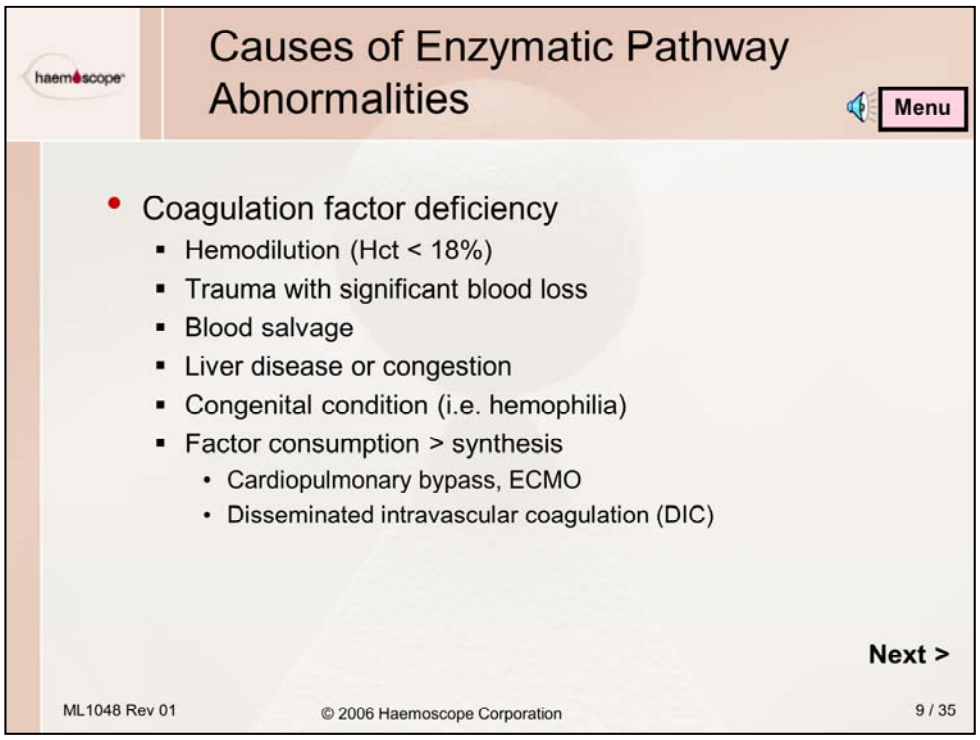
The common treatment for vessel injury is to repair the vessel. If von Willebrand factor deficiency is the probable cause, desmopressin (DDAVP) is commonly administered.[29]

Finally, if the presence of a platelet inhibitor is suspected, it is advisable to run a PlateletMapping assay to determine the extent of inhibition. See Module 6 for more information on this topic.



Enzymatic pathway abnormality can result in a bleeding condition. In such a case, the TEG tracing will have a long R value, and may also have an abnormal angle and MA, depending on the extent of the deficiency.

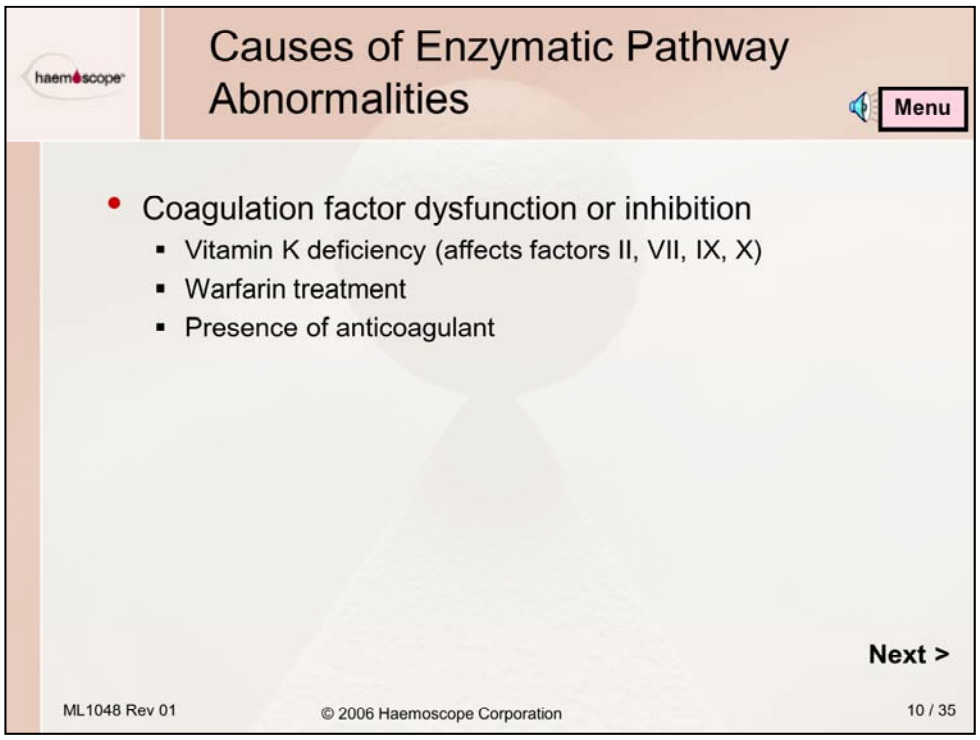
Possible causes of a long R value include coagulation factor deficiency, coagulation factor dysfunction, and the presence of an anticoagulant such as heparin or warfarin. Any of these conditions can cause a slow rate of thrombin generation, leading to slow clot development, or insufficient thrombin generation, leading to insufficient clot development.



Enzymatic pathway abnormalities can be caused by a number of different conditions, including hemodilution, trauma with significant blood loss, and blood salvage. Salvaged blood is blood lost by the patient that is recovered, washed, with only the red blood cells returned. This process leaves only the red blood cells intact; platelets and coagulation factors are lost.

Since many of the coagulation factors are manufactured in the liver, liver disease or congestion can impede factor production, leading to a deficiency. Also, congenital conditions such as hemophilia result in dysfunction of specific coagulation factors, disrupting the progression of the coagulation cascade.

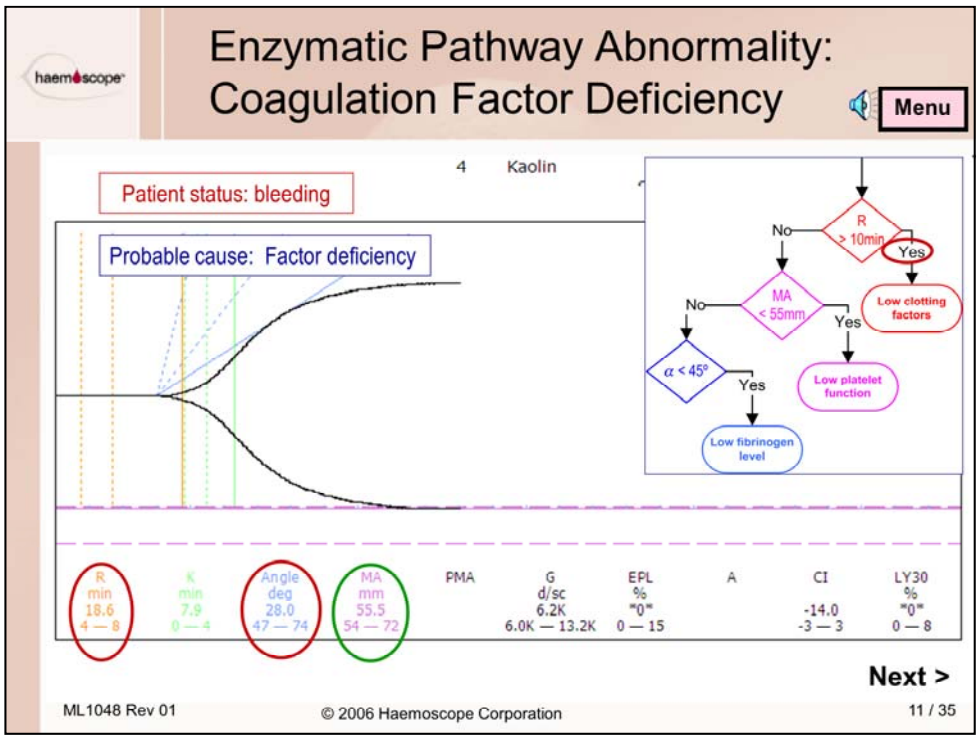
Finally, there are certain conditions in which factor consumption is greater than factor synthesis. These may include cardiopulmonary bypass and extracorporeal membrane oxygenation (ECMO), as well as disseminated intravascular coagulation (DIC).



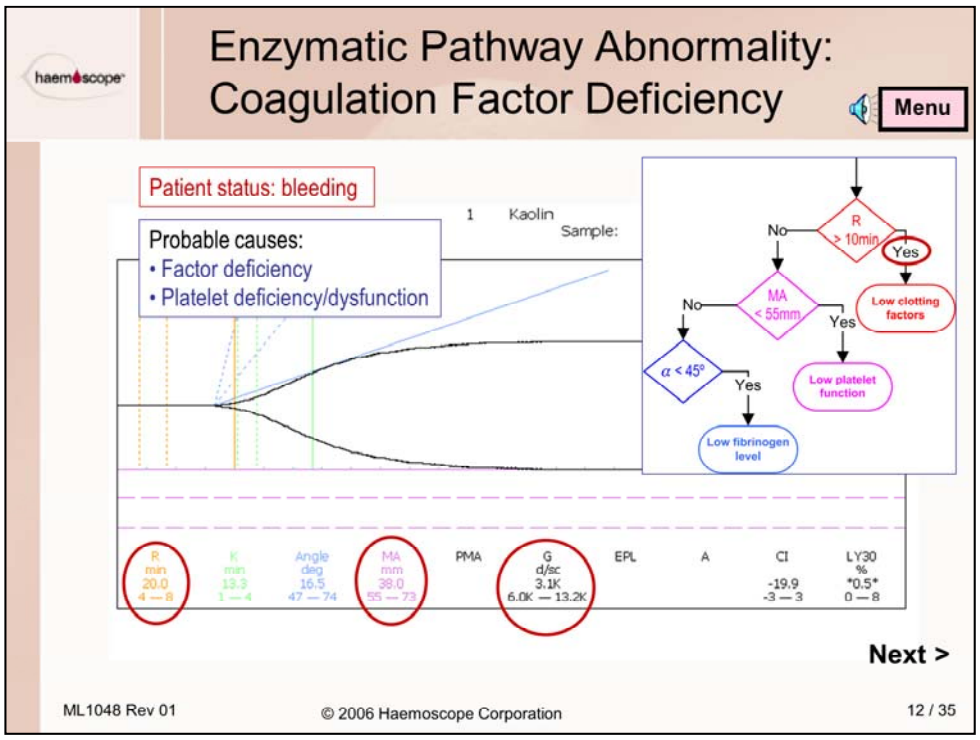
Enzymatic pathway abnormalities can also be caused by coagulation factor dysfunction or inhibition.

Vitamin K is an important cofactor in the synthesis of many coagulation factors (factors II, VII, IX, X); therefore, vitamin K deficiency can cause a general dysfunction or deficiency in these factors. Warfarin treatment, which inhibits incorporation of vitamin K into coagulation factors, results in the synthesis of nonfunctional coagulation factors.

The presence of other anticoagulants such as heparin or thrombin inhibitors hinders coagulation factor function and results in coagulation pathway abnormalities.

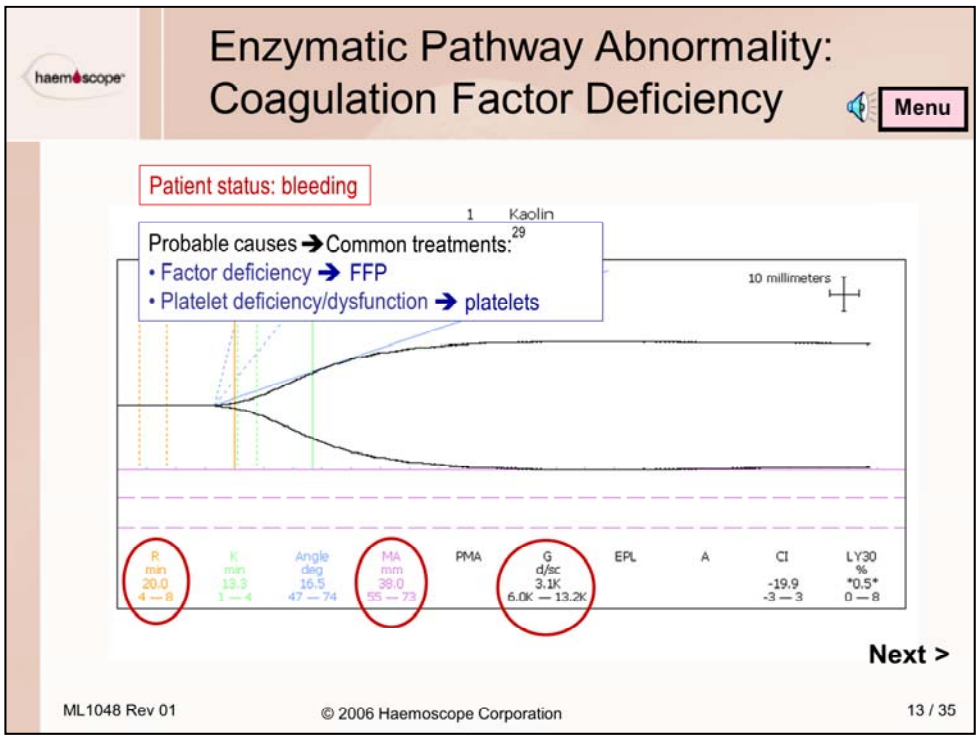


This tracing demonstrates an enzymatic pathway abnormality in a patient who is bleeding. The elongated R and normal MA values suggest factor deficiency or dysfunction. The depressed angle value demonstrates the effect of factor deficiencies on the rate of fibrin-platelet clot formation.



This illustrates another factor deficiency in a bleeding patient, as indicated by the long R value. In this case, the MA and G are low, indicating that the clot is weak. Because of platelet contribution to clot strength, the low MA and G values suggest that a possible platelet deficiency or dysfunction is also contributing to the bleeding state.

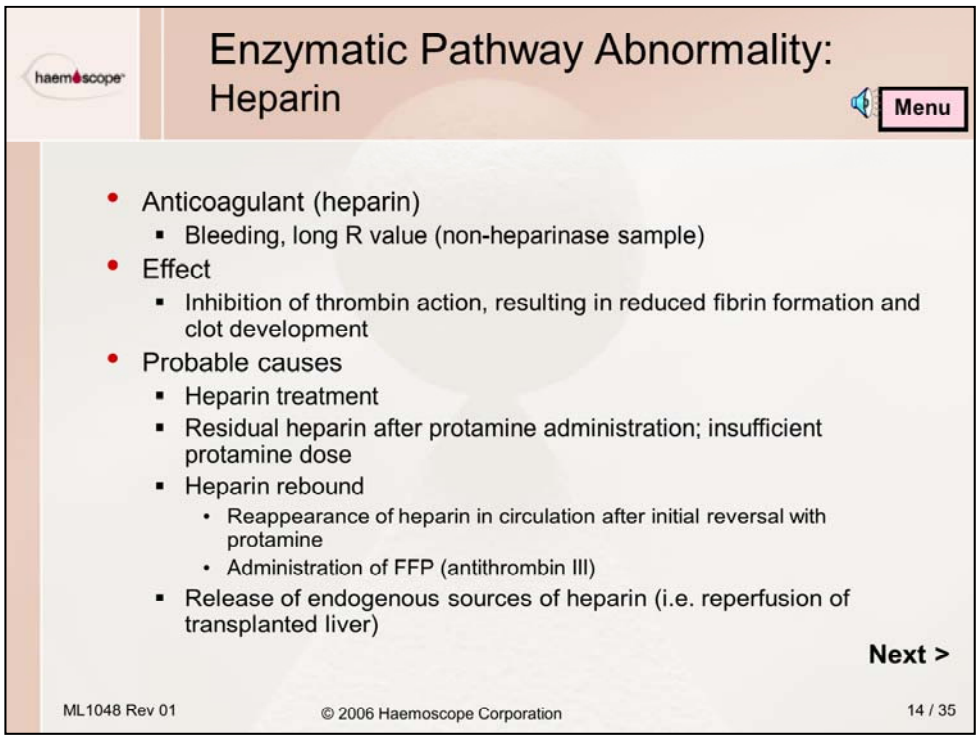
The depressed angle value again reflects the effect of coagulation factor deficiencies on the rate of fibrin-platelet clot formation.



To restore balance to the hemostatic system, the TEG decision tree suggests reducing the R value first. Since this measures the time until formation of a critical mass of thrombin, an elongated R indicates factor deficiency. To increase thrombin generation, the common treatment is fresh frozen plasma, and a normal TEG tracing after treatment with FFP would indicate that the therapy was effective.[29]

After giving FFP, a TEG tracing with a persistent low MA suggests low platelet count or function. If the patient is still bleeding, the common treatment would be a platelet transfusion. [29]

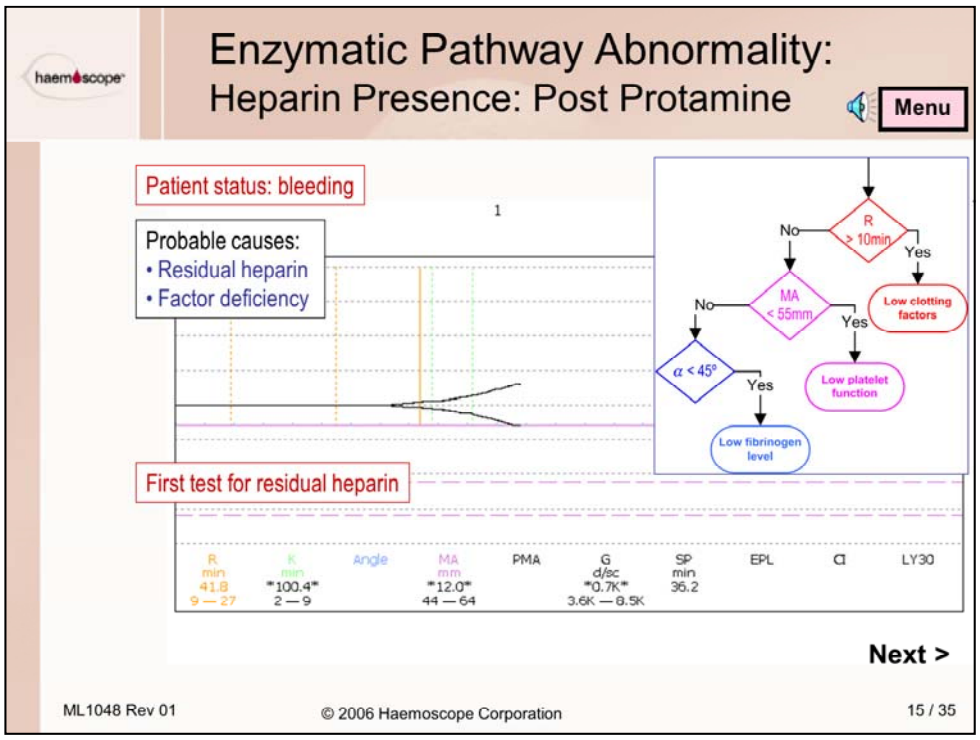
Monitoring bleeding status, along with repeating the TEG analysis after treatment, should provide the information necessary to determine the efficacy of each treatment option.



A common cause of enzymatic pathway abnormalities is the presence of an anticoagulant, especially heparin. In a bleeding patient, this type of abnormality is characterized by a long R value when using a kaolin-activated sample without heparinase. The R value is increased by heparin's inhibitory effect on thrombin and factor Xa, which delays fibrin formation and clot development. Probable causes include heparin treatment, residual heparin after administration of protamine — usually due to an insufficient protamine dose — and heparin rebound.

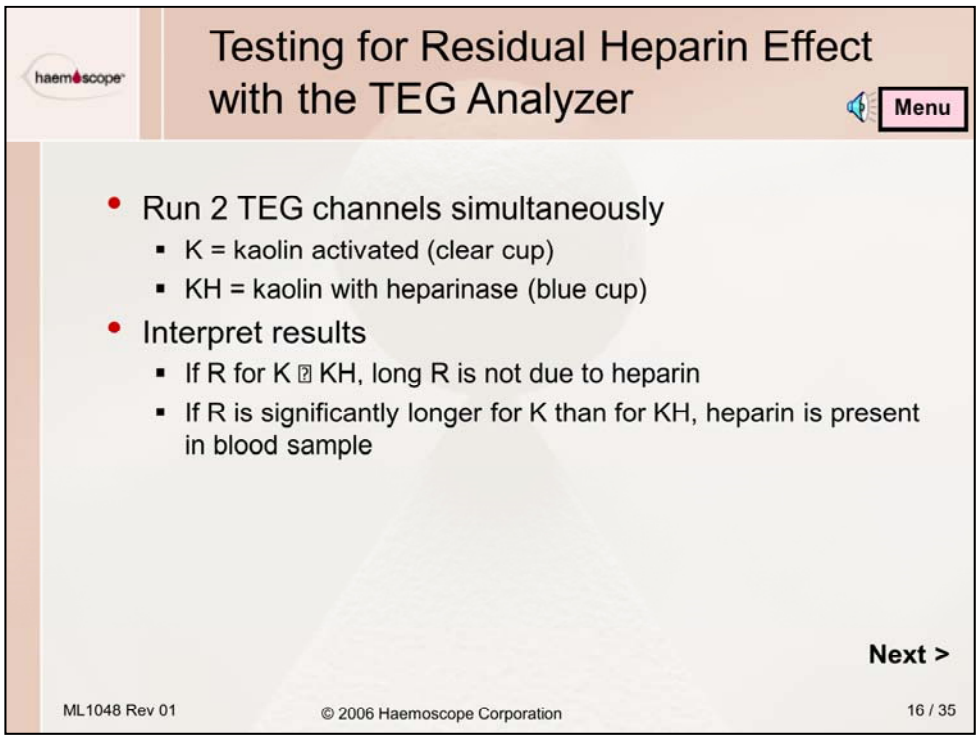
Heparin rebound is the reappearance of heparin in the blood after initial reversal with protamine. It typically appears around an hour after protamine administration, and may be due to differences in the pharmacokinetics of heparin and protamine. Administration of FFP may cause a similar effect due to the presence of antithrombin, which increases heparin activity.

Finally, release of endogenous sources of heparin may cause an increase in the R value, as seen during the reperfusion phase of liver transplantation.



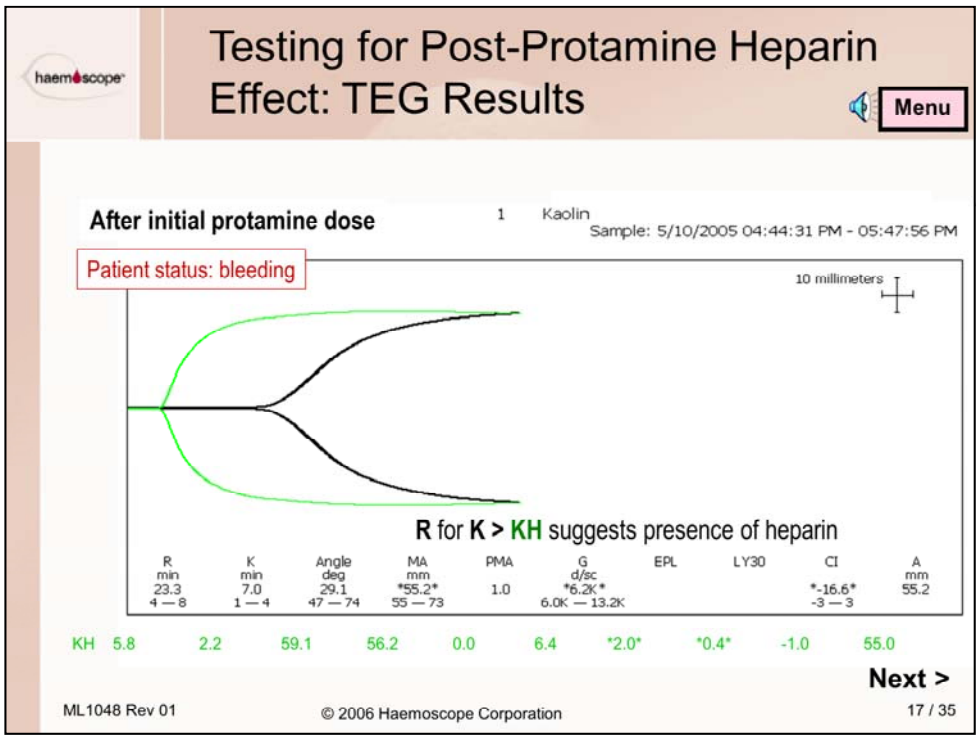
This tracing demonstrates the presence of heparin after administration of protamine. Since the patient is bleeding, the probable cause is either residual heparin or factor deficiency.

Since the sample is kaolin without heparinase, the first step in determining proper treatment is to rule out the presence of heparin.



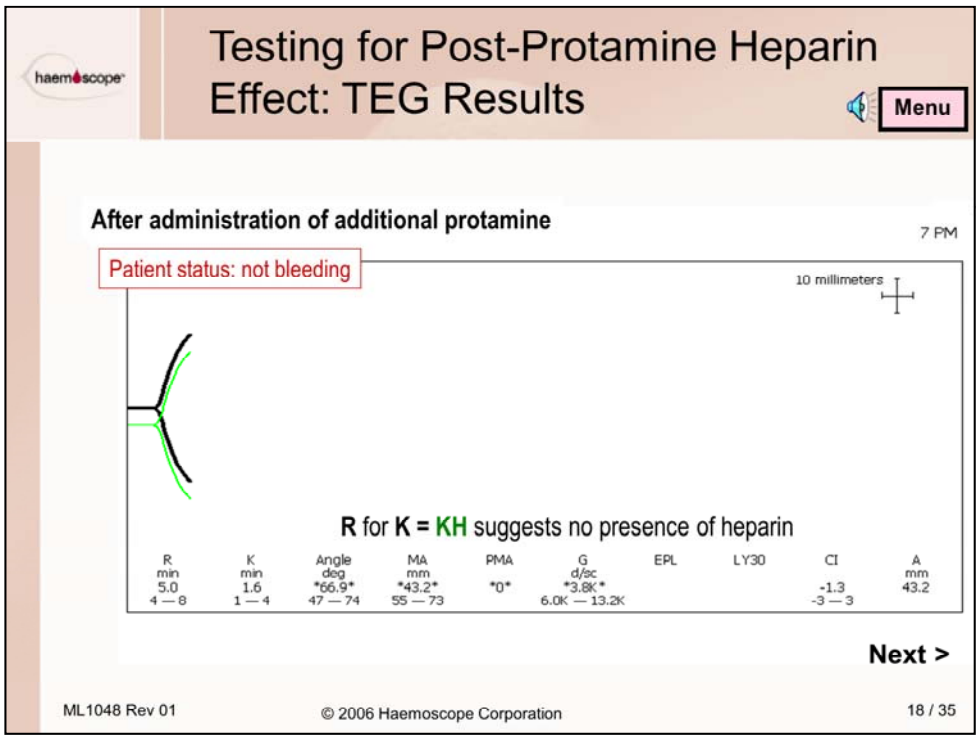
Testing for residual heparin requires running two TEG channels simultaneously — one with kaolin only (K), and one with kaolin and heparinase (KH).

If the R values for both channels are approximately equal, then the long R is not due to the presence of heparin. However, if the R value for the K channel is significantly greater than that for the KH channel, heparin is likely present in the sample, and the administration of more protamine may be required.

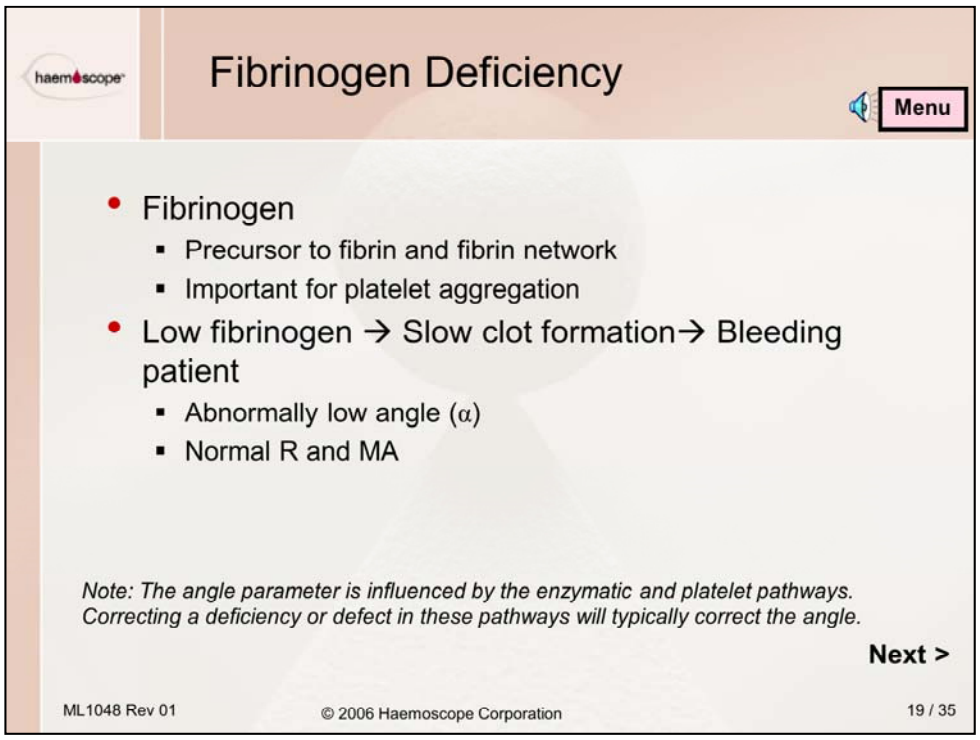


This tracing illustrates a residual heparin effect after administration of protamine.

In this case, the patient is bleeding, and the TEG results suggest the presence of heparin.



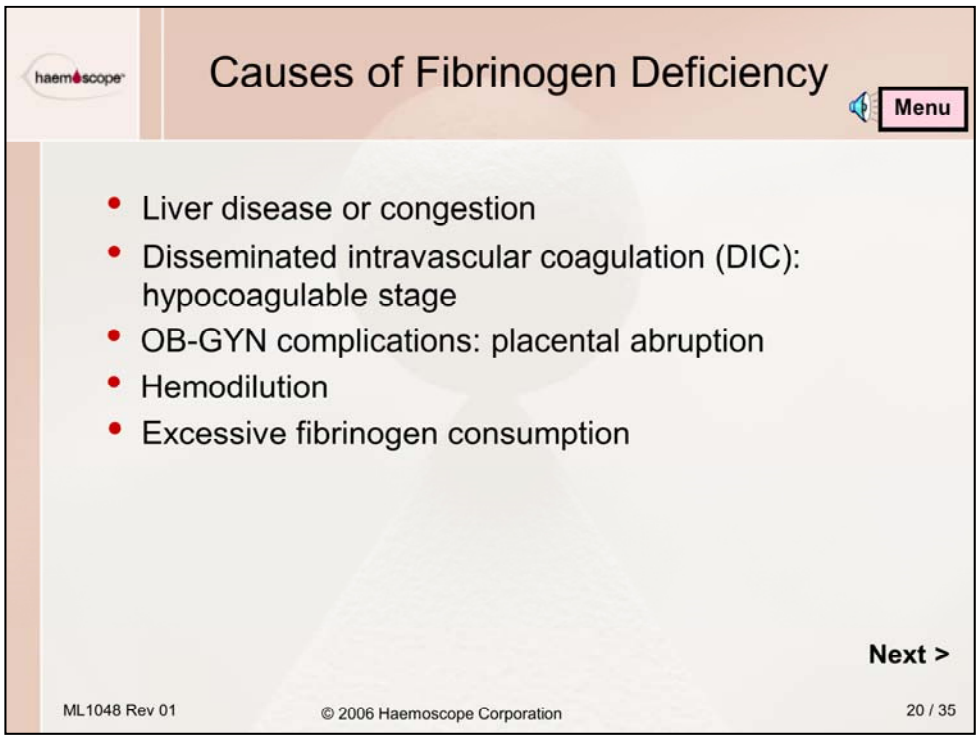
After administration of additional protamine, the patient stopped bleeding. TEG analysis demonstrates that the heparin effect in the kaolin sample has been eliminated.



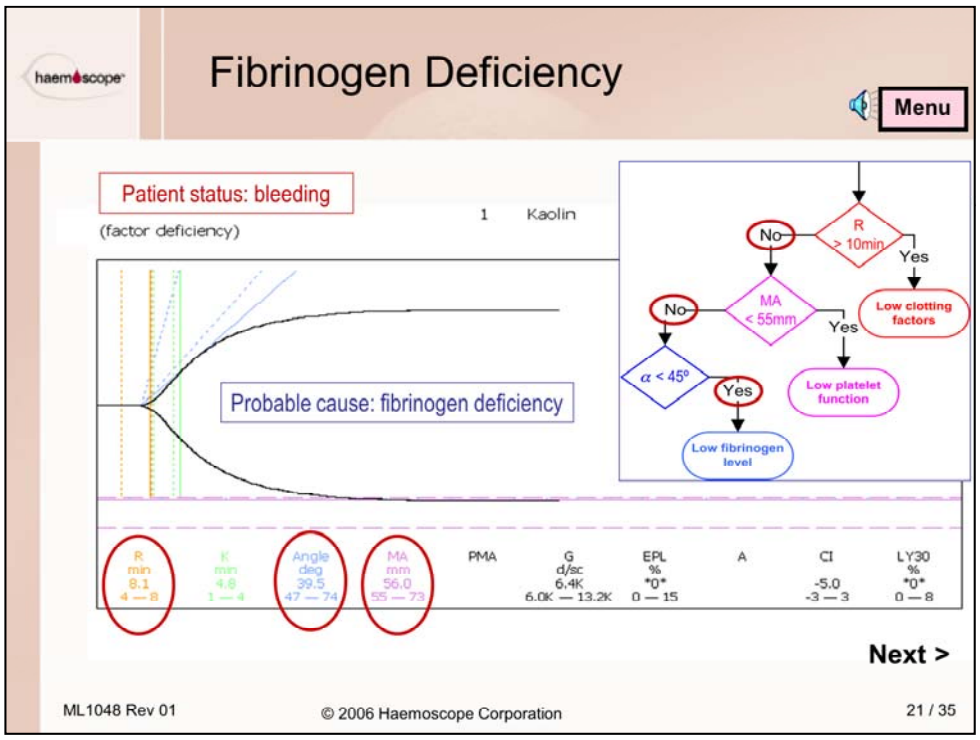
Fibrinogen is a coagulation factor with at least two important roles in hemostasis. First, it is the precursor of fibrin; therefore, sufficient fibrinogen levels are required for the construction of the fibrin network. In addition, it is also the ligand for the platelet GPIIb/IIIa receptor and is important for platelet aggregation; as a result, low fibrinogen levels may cause bleeding due to a slow rate of clot development.

Fibrinogen deficiency is identified in a TEG tracing by an abnormally low angle combined with normal R and MA values.

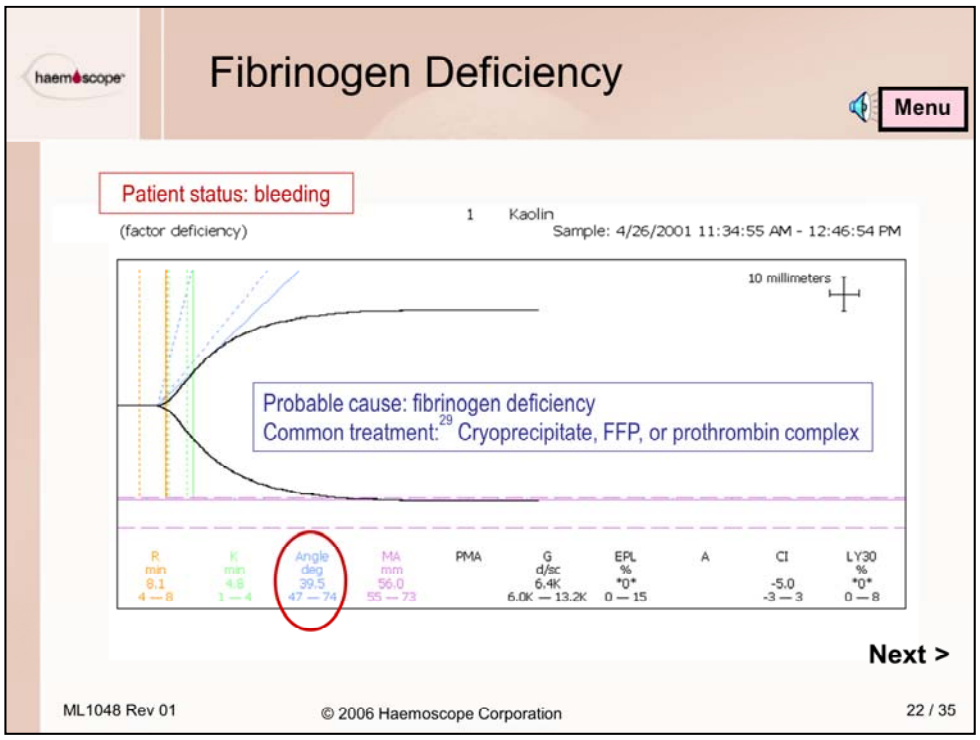
Due to the interaction of fibrinogen in both the enzymatic pathway and platelet aggregation reactions, a low angle is often seen in the presence of enzymatic and platelet defects. In such cases, correction of these defects will typically correct the angle.



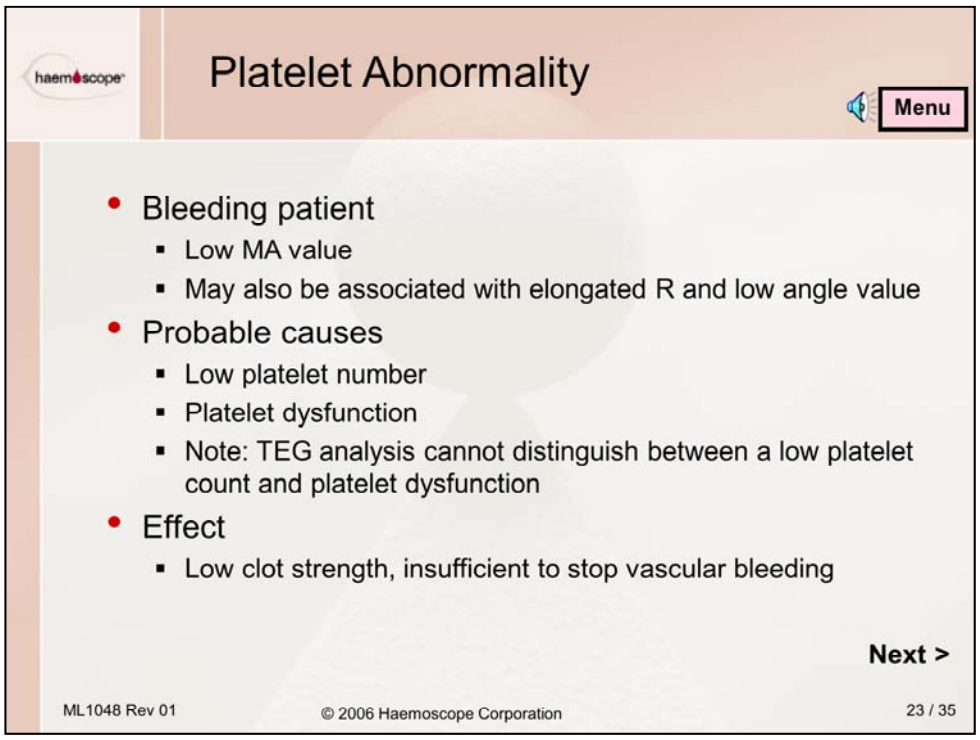
Fibrinogen deficiency can be caused by liver disease or congestion, DIC, OB-GYN complications such as placental abruption, hemodilution, and excessive fibrinogen consumption due to a disease state.



This tracing illustrates fibrinogen deficiency in a bleeding patient. In this case, the angle is abnormally low, but the R and MA values are normal. Such a result suggests an isolated fibrinogen deficiency.



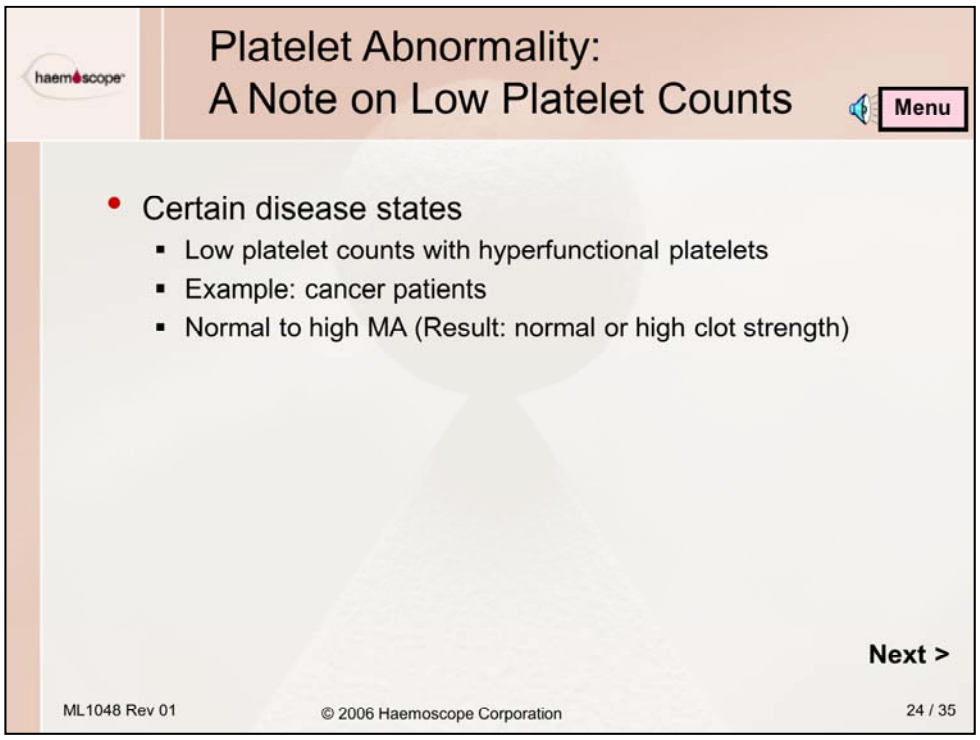
Common treatments for fibrinogen deficiency include cryoprecipitate, FFP, and prothrombin complex.[29] The choice of treatment is determined by product availability and protocols at each institution.



Platelet abnormalities are a common cause of bleeding, especially in cardiac patients. They are characterized by a TEG tracing with an abnormally low MA value.

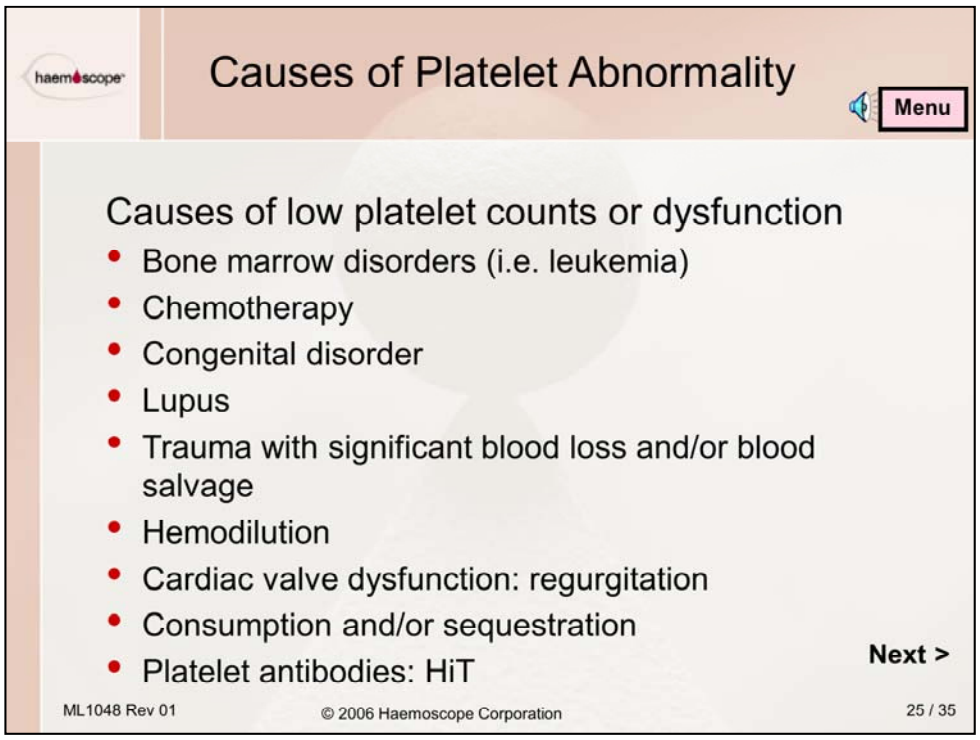
Due to the important role of platelets in thrombin generation, a low MA value may also be associated with an elongated R value and a low angle.

The probable causes of a low MA include low platelet number and platelet dysfunction. Since MA measures clot strength, it cannot distinguish between low platelet number and platelet dysfunction, as both conditions result in low clot strength.



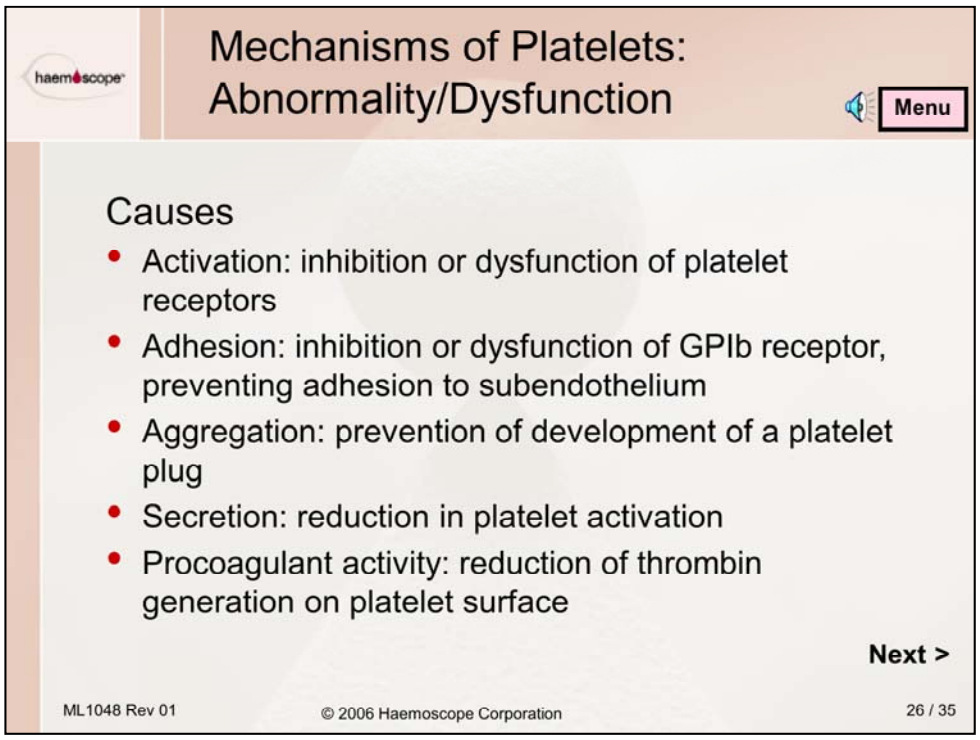
The importance of monitoring platelet function can be seen in conditions where platelet count is low, but the platelets themselves are hyperfunctional.

For example, certain types of cancer patients demonstrate a very low platelet count but a normal to high MA value, suggesting that the platelets are active and able to produce clots.



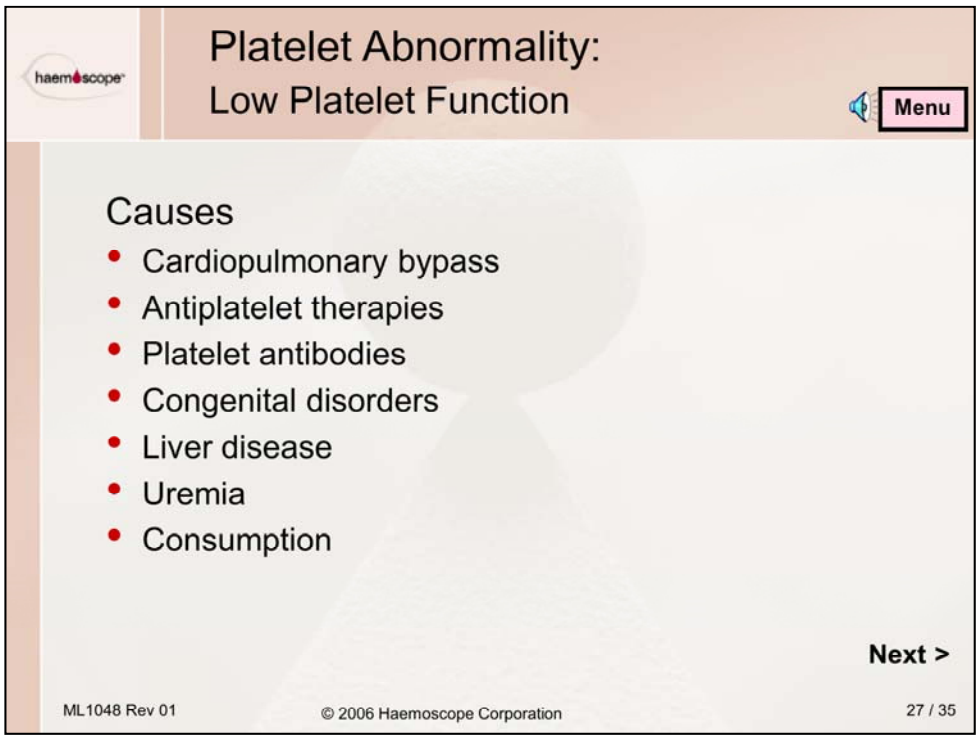
Low platelet count or dysfunction is common in many disease states. It can also be a result of treatment for these disease states.

Causes include bone marrow disorders, chemotherapy, congenital disorders, lupus, trauma with significant blood loss, hemodilution, certain heart valve defects, consumption and sequestration, and the presence of platelet antibodies, as in heparin-induced thrombocytopenia (HiT).



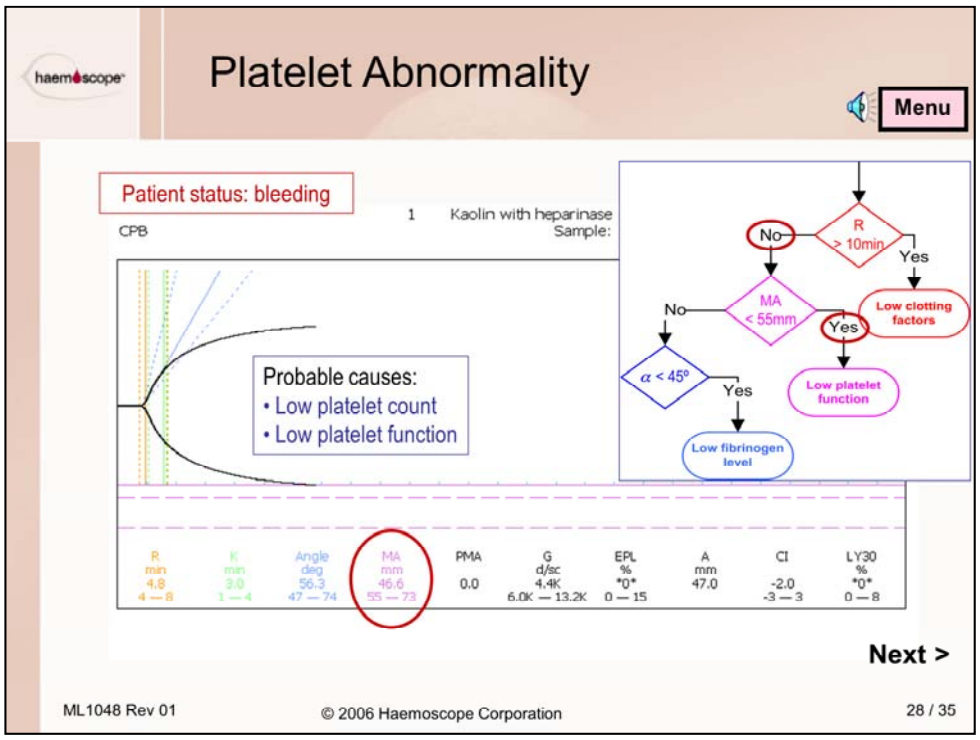
Platelet dysfunction can have several causes. These include defects in activation, adhesion, aggregation, secretion, and procoagulant activity.

- Activation defects are due to inhibition or dysfunction of platelet receptors.
- Adhesion defects are due to flaws involving the GPIb receptor.
- Aggregation defects are due to inhibition or dysfunction of the GPIIb/IIIa receptor.
- Secretion defects are due to inhibition or dysfunction of the secretory pathways, resulting in reduced platelet activation capability.
- Defects in platelet procoagulant activity may reduce overall thrombin generation, thereby slowing or reducing the overall hemostatic response.

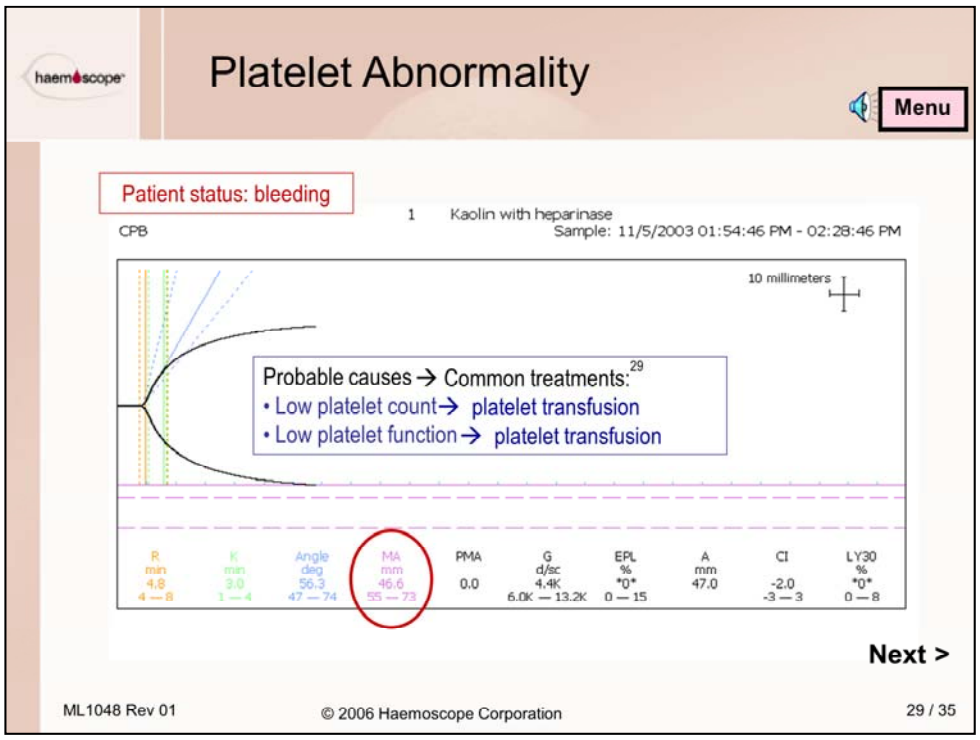


A reduction in platelet function can be caused by cardiopulmonary bypass, antiplatelet therapies, the presence of platelet antibodies, congenital disorders, liver disease, uremia, and consumption.

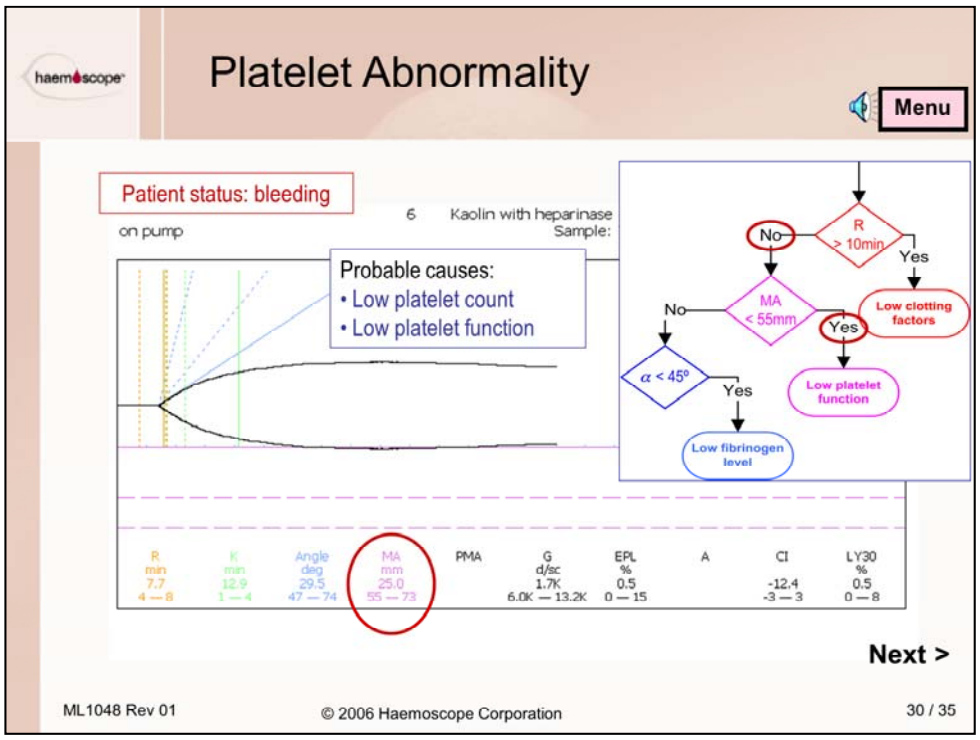
Many of the causes of low platelet function can also be responsible for low platelet counts. Therefore, when monitoring hemostasis, it is important to be able to monitor the overall contribution of platelets to the clotting process, rather than just a number.



Platelet abnormalities are typically identified by a low MA value. In cases where a patient is bleeding and the MA is low, reduced platelet count and/or function should be suspected as the cause.

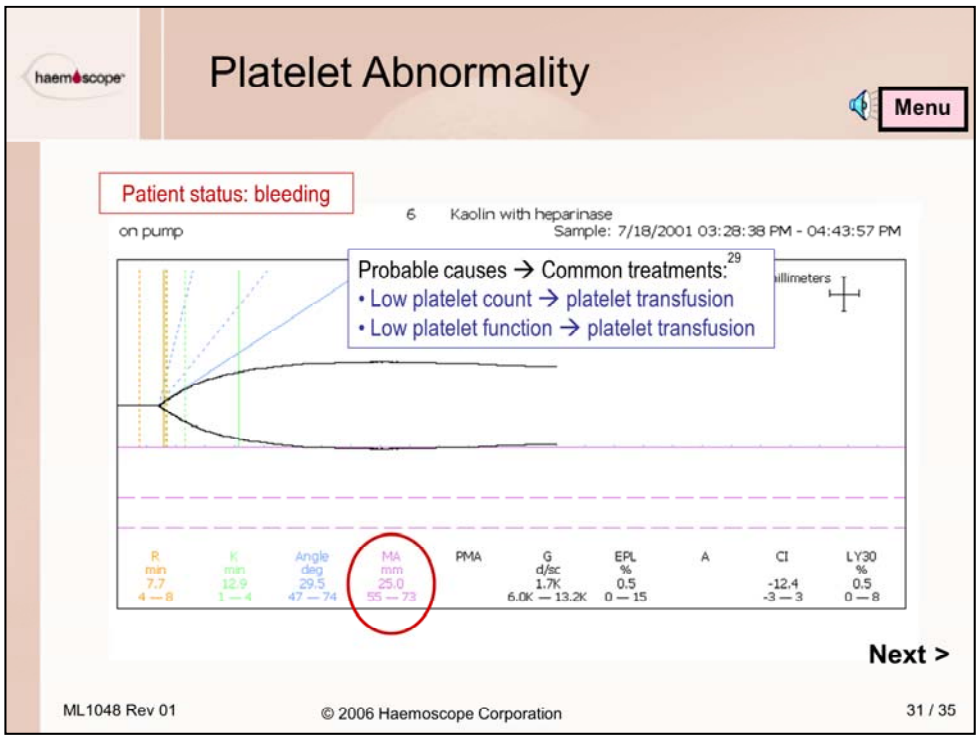


When a patient is bleeding and the TEG tracing shows a low MA, the common treatment is platelet transfusion, irrespective of whether the MA value is due to low platelet number or to low platelet function.[29]



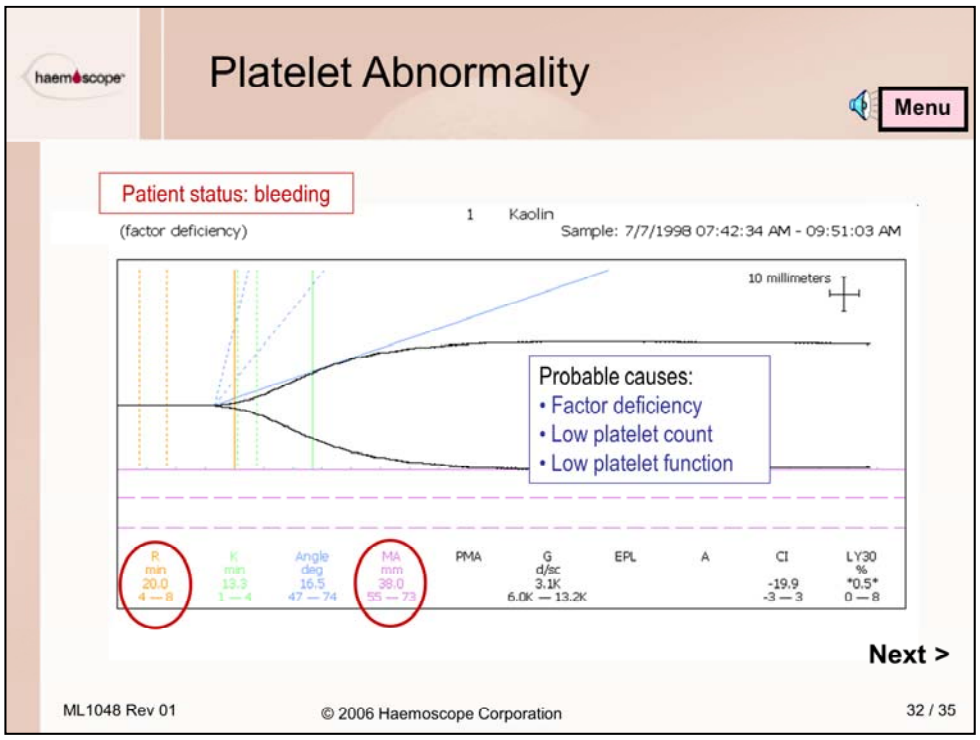
This tracing is from another bleeding patient with a platelet abnormality.

Note that the R value is normal, suggesting that there are sufficient levels of coagulation factors available to make a clot.



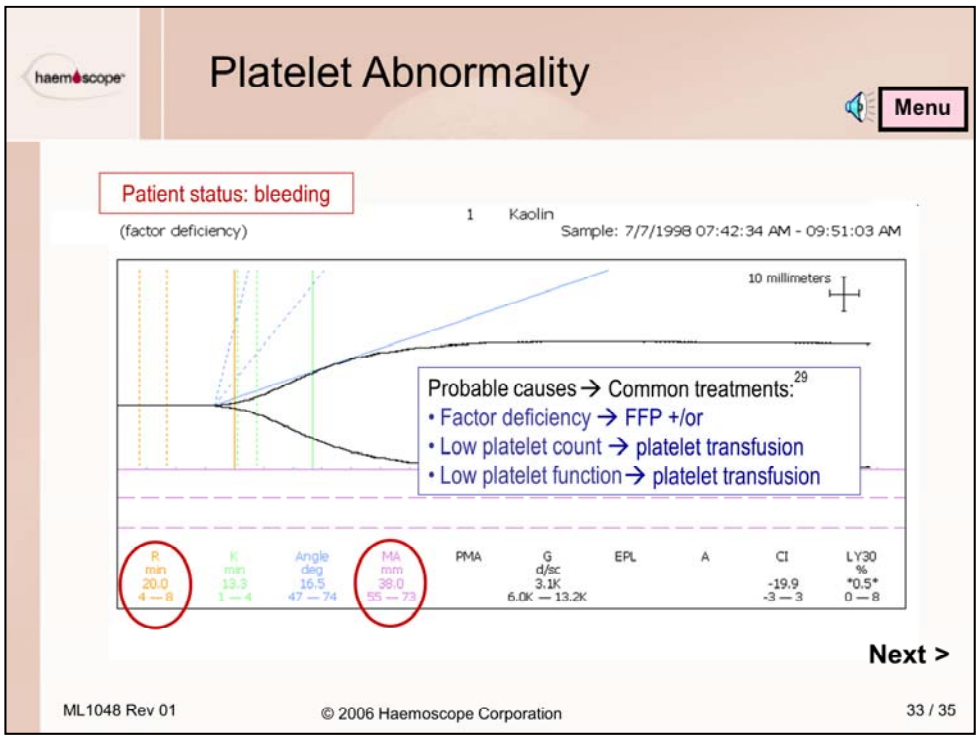
Again, the common treatment for this patient is platelet transfusion.[29]

In this case, multiple transfusions may be required. Observation of bleeding status, combined with additional TEG analysis, will demonstrate the efficacy of each transfusion.



In this tracing, bleeding is probably due to a combination of factors. The long R value suggests a coagulation factor deficiency, while the low MA suggests low platelet count and/or platelet dysfunction.

The TEG decision tree, used as a guide for bringing the hemostatic system back into balance, suggests that the R should be reduced first, then the MA.

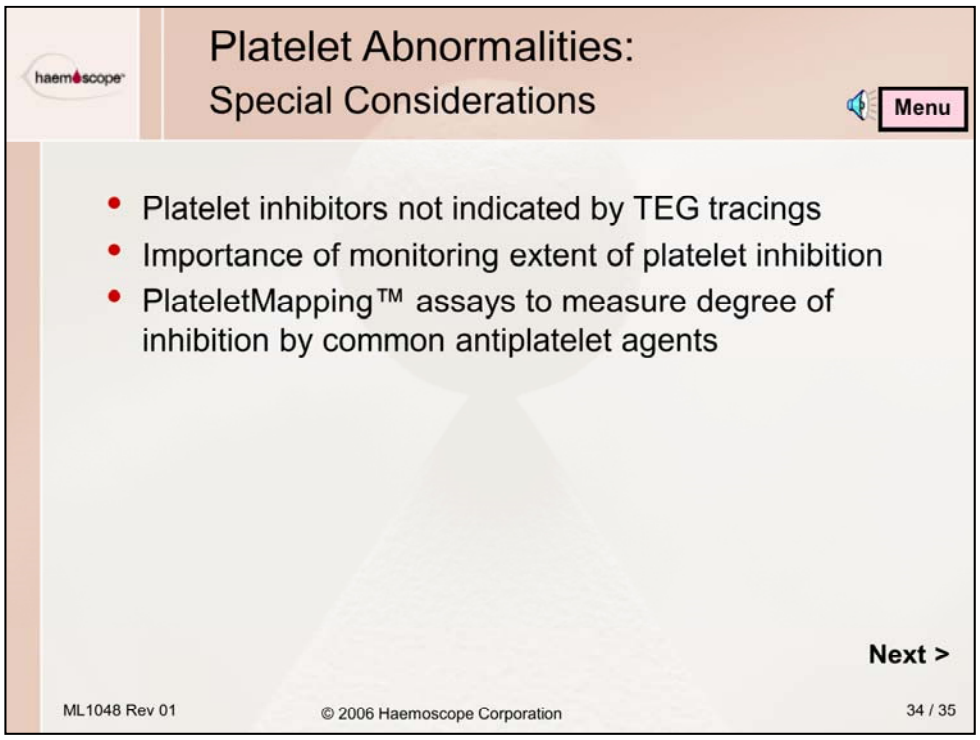


Since bleeding may be caused by a combination of factors, treatment may require a combination of blood components.

The TEG decision tree suggests reducing the R first in order to restore hemostatic balance. Since the elongated R suggests slow thrombin generation and factor deficiency, common treatment is fresh frozen plasma.[29] A normal TEG tracing after FFP treatment indicates the therapy was effective.

After FFP treatment, a TEG tracing with a low MA suggests low platelet count or function. The common treatment is platelet transfusion.[29]

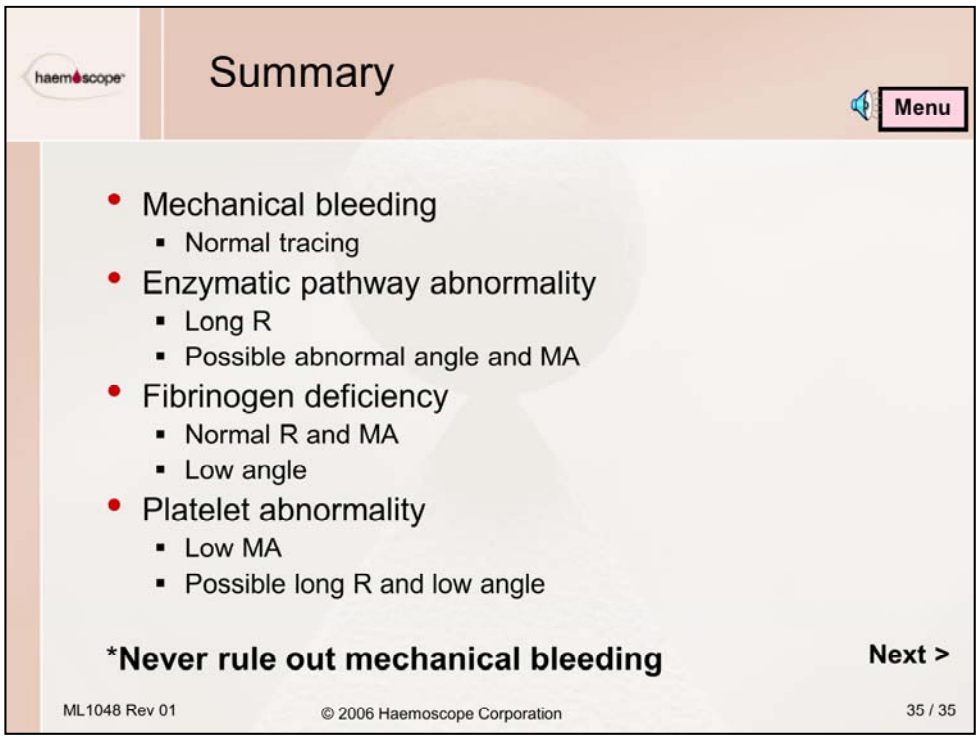
Monitoring bleeding status, along with repeating TEG analysis after treatment, should provide the information necessary to determine the efficacy of each treatment option.



Since thrombin is the most potent of all platelet activators, its presence in a sample will override inhibition at other platelet receptors or activation pathways. Therefore, because of thrombin generation in the *in* vitro environment, the presence of platelet inhibitors will normally not be demonstrated by an *in vitro* TEG test.

In the presence of platelet inhibitors, a standard TEG tracing may be normal, but the patient may still be bleeding. Therefore, due to the prevalence of the use of pharmacological platelet inhibitors, it is important to monitor the extent of platelet inhibition.

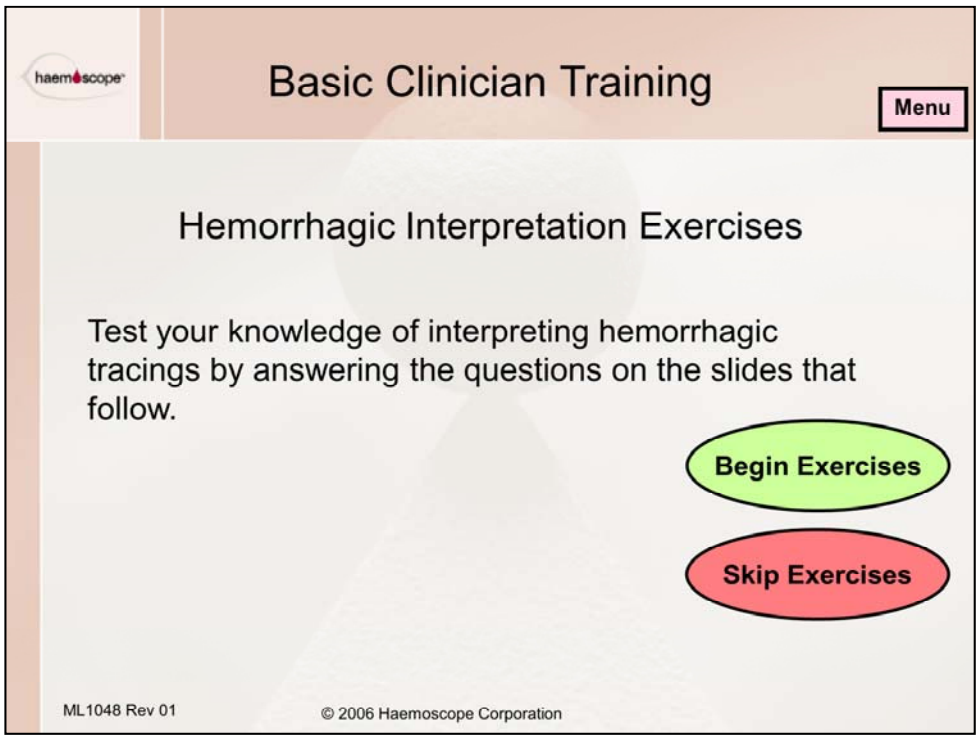
Haemoscope developed the PlateletMapping assay to measure the degree of inhibition by common antiplatelet agents. This allows clinicians to monitor the efficacy and effect of these agents, while also providing a platelet function reference point for each patient. See Module 6 for further information.



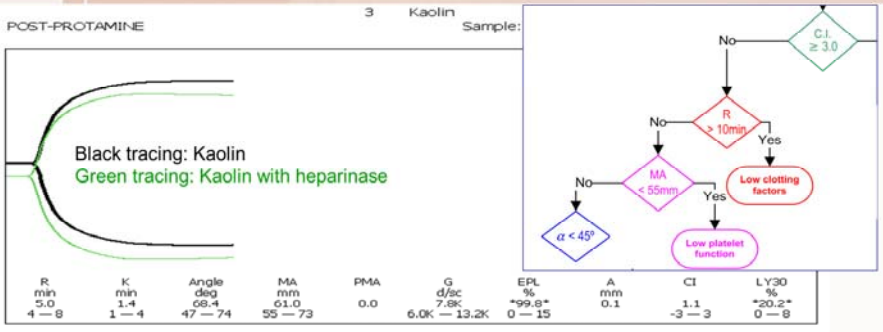
This module on hemorrhagic states has examined causes of bleeding and the TEG tracings resulting from them.

- Mechanical bleeding results in a normal TEG tracing.
- An enzymatic pathway abnormality results in a tracing with a long R value, and possibly an abnormal angle and MA.
- A fibrinogen deficiency results in a tracing with normal R and MA values, but an abnormally low angle.
- A platelet abnormality results in a low MA value, and possibly a long R and low angle.

Although a tracing may suggest an enzymatic pathway abnormality, a fibrinogen deficiency, or a platelet abnormality, mechanical bleeding is always a possible cause of bleeding.


haemoscope
Basic Clinician Training
Menu
Hemorrhagic Interpretation Exercises
Test your knowledge of interpreting hemorrhagic tracings by answering the questions on the slides that follow.
Begin Exercises
Skip Exercises
ML1048 Rev 01
© 2006 Haemoscope Corporation

Exercise 1



Using the TEG decision tree, what is your interpretation of this tracing?
(Select all that apply)

a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal

Has heparin been reversed in this patient? (Yes or No)

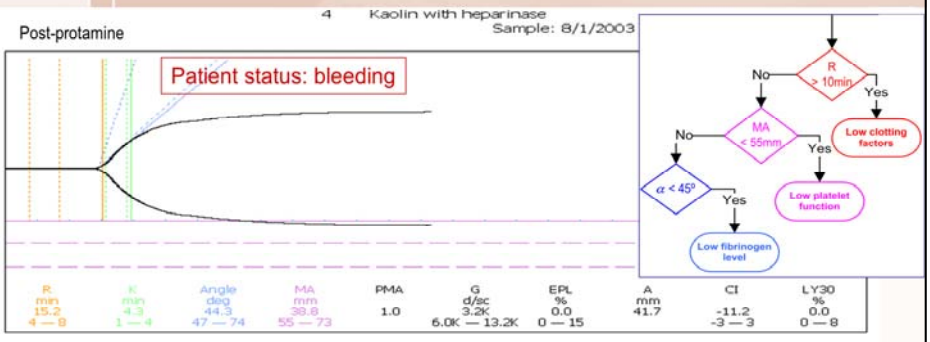
Answer

Next

Menu

ML1048 Rev 01

Exercise 2



Using the TEG decision tree, what is your interpretation of this tracing?
(Select all that apply)

a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal

What treatment(s) would you consider for this patient?

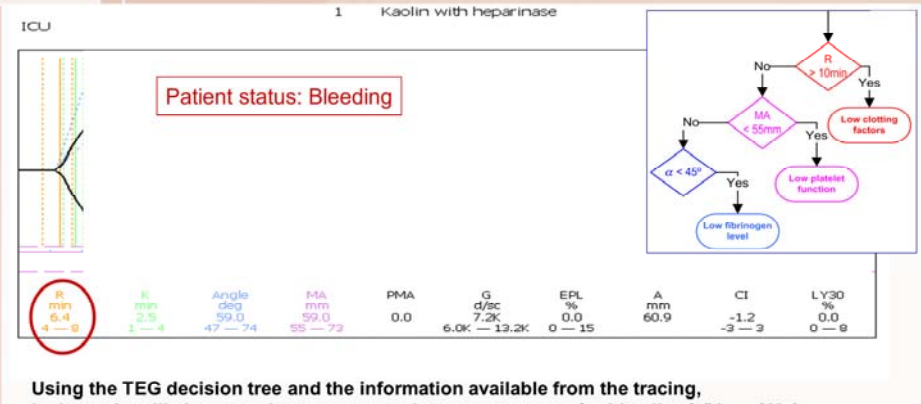
Answer

Next

Menu

ML1048 Rev 01

Exercise 3



Using the TEG decision tree and the information available from the tracing, is the patient likely to require more protamine as a treatment for bleeding? (Yes of No)

Answer

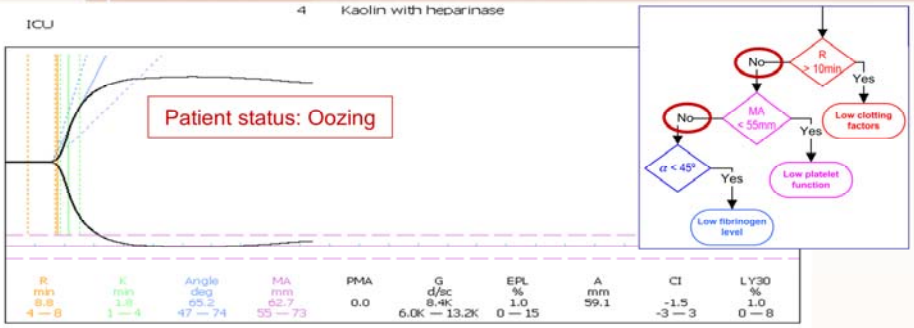
Is the patient likely to require FFP as a treatment for bleeding? (Yes of No)

Next

Menu

ML1048 Rev 01

Exercise 4



Using the TEG decision tree, what is the likely cause(s) of oozing in this patient? (Select all that apply)

a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Surgical bleeding

What treatment(s) would you consider for this patient?

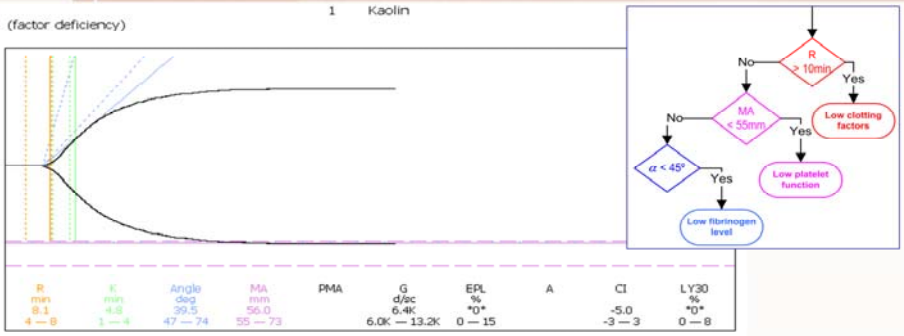
Answer

Next

Menu

ML1048 Rev 01

Exercise 5



Using the TEG decision tree, what is your interpretation of this tracing?
(Select all that apply)

a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal

If this patient is bleeding, what treatment(s) would you consider?

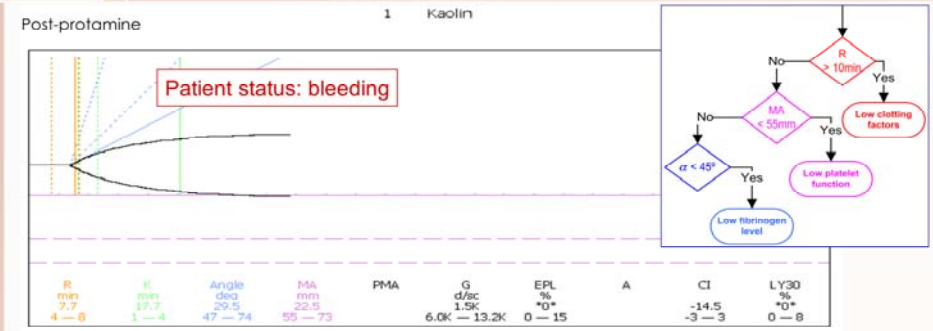
Answer

Next

Menu

ML1048 Rev 01

Exercise 6



Using the TEG decision tree, what is your interpretation of this TEG tracing?
(Select all that apply)

a) Factor deficiency
b) Anticoagulant effect
c) Platelet deficiency or dysfunction
d) Low fibrinogen level
e) Surgical bleeding

What treatment(s) would you consider for this patient?

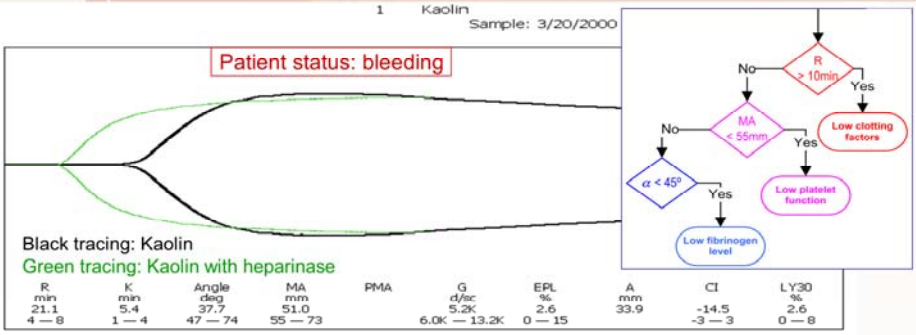
Answer

Next

Menu

ML1048 Rev 01

Exercise 7



Using the TEG decision tree, what is your interpretation of this tracing? (Select all that apply)

a) Factor deficiency
b) Anticoagulant effect
c) Platelet deficiency or dysfunction
d) Low fibrinogen level
e) Surgical bleeding

How would you treat this patient?

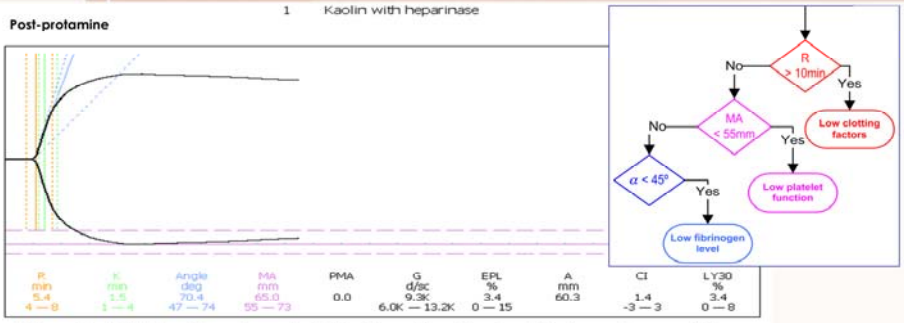
Answer

Next

Menu

ML1048 Rev 01

Exercise 8



This patient is bleeding after administration of protamine. What are the possible causes? (Select all that apply) Identify one treatment consideration for each selection.

a) Surgical bleeding
b) Factor deficiency
c) Residual platelet inhibitor effect
d) Diminished platelet adhesion
e) Anticoagulant effect

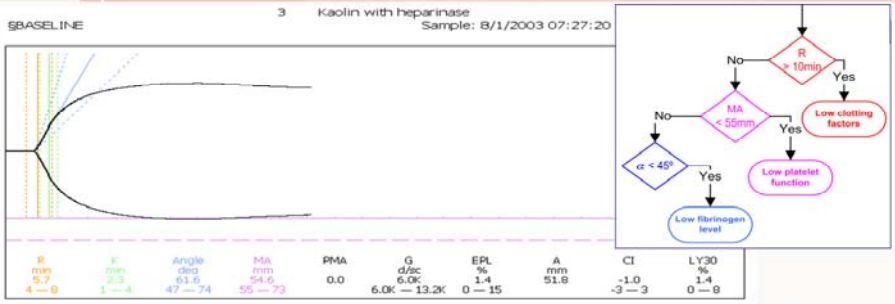
Answer

Next

Menu

ML1048 Rev 01

Exercise 9



This is the baseline TEG tracing (post-induction) of a female patient (70 yr, 54kg) requiring CABG with CPB. She stopped taking aspirin 7 days previously (81 mg/d). Based on this tracing and the patient's history, is she at risk for bleeding post-CPB? (Yes or No)
If so, what will be the most likely cause(s) of bleeding?

a) Surgical bleeding
b) Factor deficiency
c) Platelet deficiency/dysfunction
d) Fibrinolysis
e) Anticoagulant effect

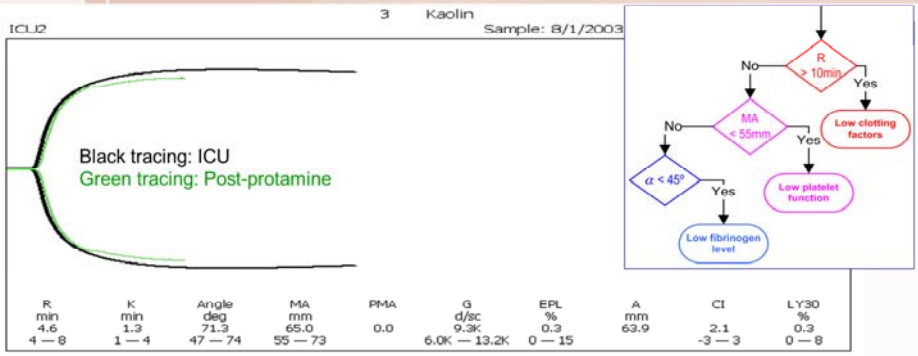
Answer

Next

Menu

ML1048 Rev 01

Exercise 10



This patient began bleeding approximately 1 hr post-op. What is the most likely cause(s) of bleeding?

a) Surgical bleeding
b) Factor deficiency
c) Platelet deficiency/dysfunction
d) Fibrinolysis
e) Anticoagulant effect

How would you treat this patient?

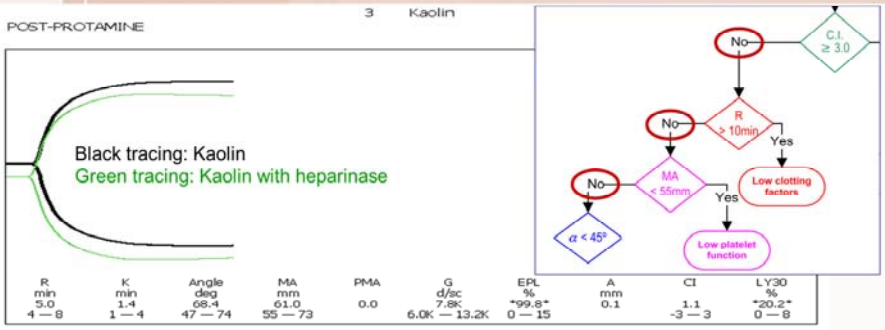
Answer

End

Menu

ML1048 Rev 01

Answer to Exercise 1



Using the TEG decision tree, what is your interpretation of this tracing?
(Select all that apply)

a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal - all parameters are within normal range

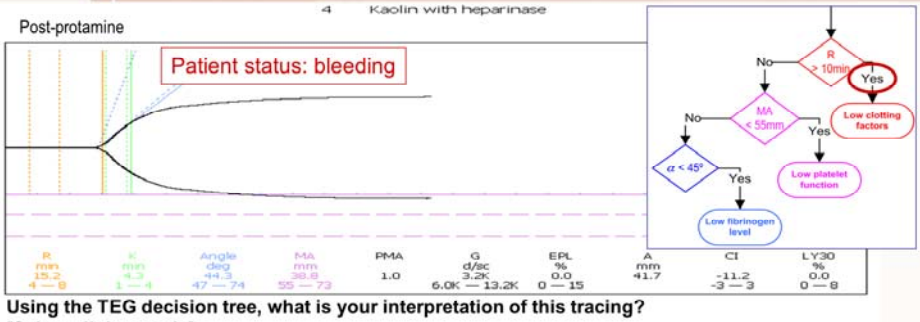
Has heparin been reversed in this patient? Yes. The R values for both samples(kaolin and kaolin with heparinase) are the same, suggesting that the heparin has been reversed.

Next

Menu

ML1048 Rev 01

Answer to Exercise 2



Using the TEG decision tree, what is your interpretation of this tracing?
[Select all that apply]
a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal

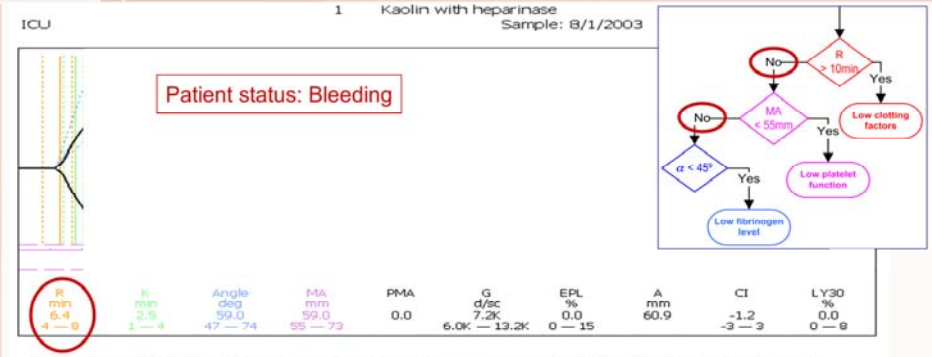
What treatment(s) would you consider for this patient? Common treatment would be in two steps. First, consider treating with FFP to reduce the R value by increasing thrombin generation. Follow this with another TEG analysis. Then, if R value is still elongated and the patient is still bleeding, consider a platelet transfusion.[29]

ML1048 Rev 01

Next

Menu

Answer to Exercise 3



Using the TEG decision tree and the information available from the tracing, is the patient likely to require more protamine as a treatment for bleeding?

No. This tracing is from a kaolin activated sample. The presence of heparin would result in an elongated R value; an R within normal range suggests heparin is not present, so additional protamine is not required.

Is the patient likely to require FFP as a treatment for bleeding?

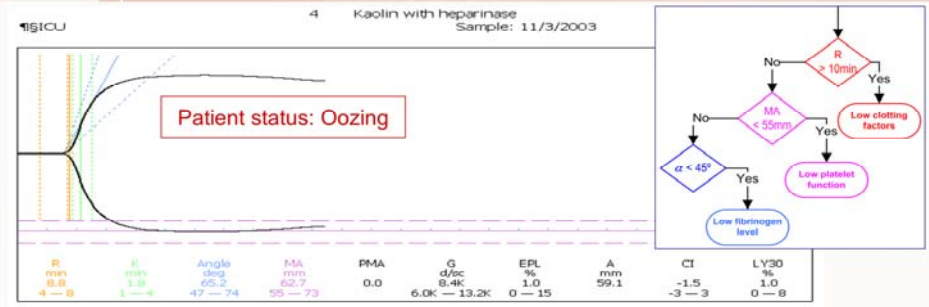
No. An R value within normal range suggests that factor deficiency is not the cause of bleeding.

Next

Menu

ML1048 Rev 01

Answer to Exercise 4



Using the TEG decision tree, what is the likely cause(s) of oozing in this patient? (Select all that apply)

a) Factor deficiency

b) Platelet deficiency or dysfunction

c) Low fibrinogen level

d) Fibrinolysis

e) Surgical bleeding — A slightly elongated R may suggest factor deficiency; however, surgical bleeding cannot be ruled out.

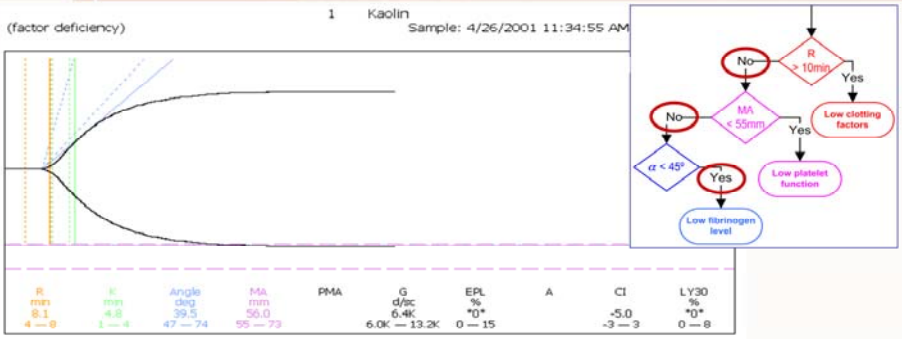
What treatment(s) would you consider for this patient? The R value is less than the trigger point for FFP transfusion. If the patient is oozing, wait an hour, then repeat the TEG analysis. If the R is still elongated and the patient is still oozing, consider treating with FFP.[29]

Next

Menu

ML1048 Rev 01

Answer to Exercise 5



Using the TEG decision tree, what is your interpretation of this tracing?
(Select all that apply)

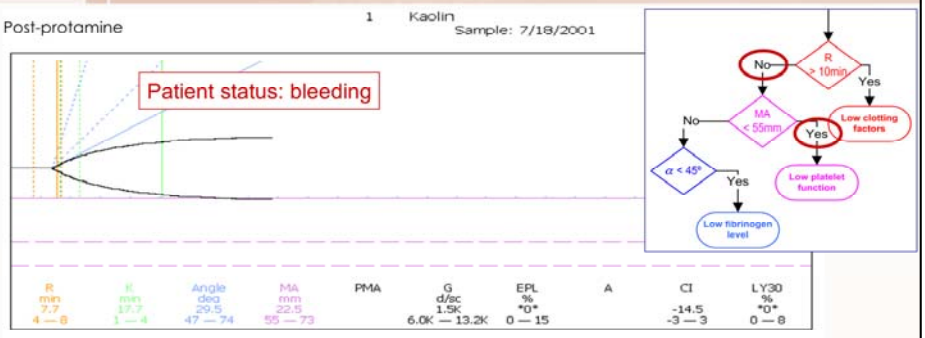
a) Factor deficiency
b) Platelet deficiency or dysfunction
c) Low fibrinogen level
d) Fibrinolysis
e) Normal

If this patient is bleeding, what treatment(s) would you consider?
Cryoprecipitate or FFP are possible treatments.[29]

Next

Menu

Answer to Exercise 6



Using the TEG decision tree, what is your interpretation of this TEG tracing? (Select all that apply)

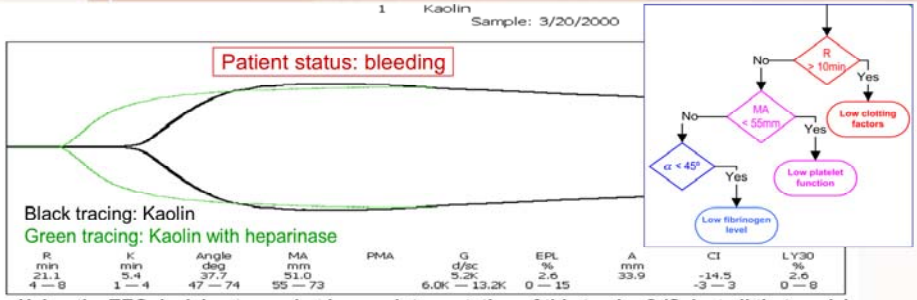
a) Factor deficiency
b) Anticoagulant effect
c) Platelet deficiency or dysfunction
d) Low fibrinogen level
e) Surgical bleeding

What treatment(s) would you consider for this patient? Platelet transfusion is a possible treatment.[29]

Next

Menu

Answer to Exercise 7



Using the TEG decision tree, what is your interpretation of this tracing? (Select all that apply)

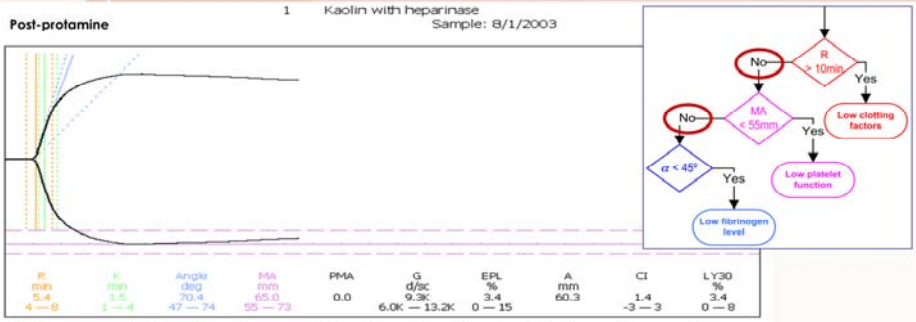
a) Factor deficiency —Cannot rule this out
b) Anticoagulant effect — Incomplete reversal of heparin
c) Platelet deficiency or dysfunction — Cannot rule this out
d) Low fibrinogen level
e) Surgical bleeding

How would you treat this patient? A common treatment protocol would likely include administration of additional protamine to reverse the heparin effect. If the patient continues to bleed, repeat the TEG analysis to determine the probable cause, which most likely is platelet deficiency/dysfunction.[29]

Next
Menu

ML1048 Rev 01

Answer to Exercise 8



This patient is bleeding after administration of protamine. What are the possible causes? (Select all that apply) Identify one treatment consideration for each selection.

a) Surgical bleeding — Locate bleeding site and repair

b) Factor deficiency

c) Residual platelet inhibitor effect — Administer platelet transfusion[29]

d) Diminished platelet adhesion: — Administer DDAVP[29]

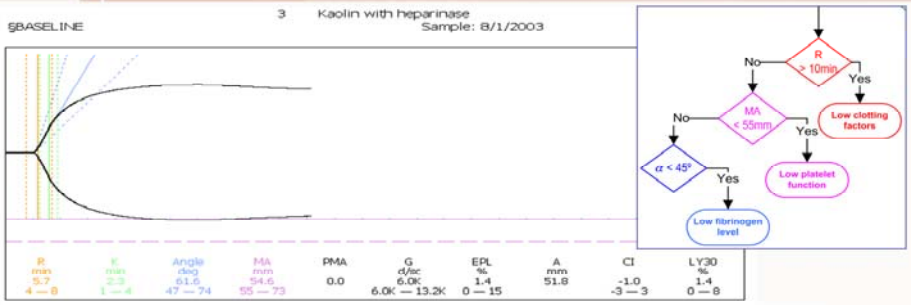
e) Anticoagulant effect — Since this is a KH sample, there is a possibility of residual heparin. Compare R values for the K and KH samples; if they are similar, the cause of bleeding is not residual heparin.

Next

Menu

ML1048 Rev 01

Answer to Exercise 9



This is the baseline TEG tracing (post-induction) of a female patient (70 yr, 54kg) requiring CABG with CPB. She stopped taking aspirin 7 days previously (81 mg/d). Based on this tracing and the patient's history, is she at risk for bleeding post-CPB? Yes
If so, what will be the most likely cause(s) of bleeding?

a) Surgical bleeding

b) Factor deficiency — Cannot be ruled out

c) Platelet deficiency/dysfunction — Since the patient started out with a low platelet count/function (low MA), platelet function may be compromised post-CPB. Consider using Aprotinin to reduce the impact of CPB on the hemostatic system. The results of a TEG® sample taken approximately 30 minutes before the end of CPB should demonstrate the effect of CPB on hemostasis and help determine appropriate treatment if the patient is bleeding post-CPB.[29]

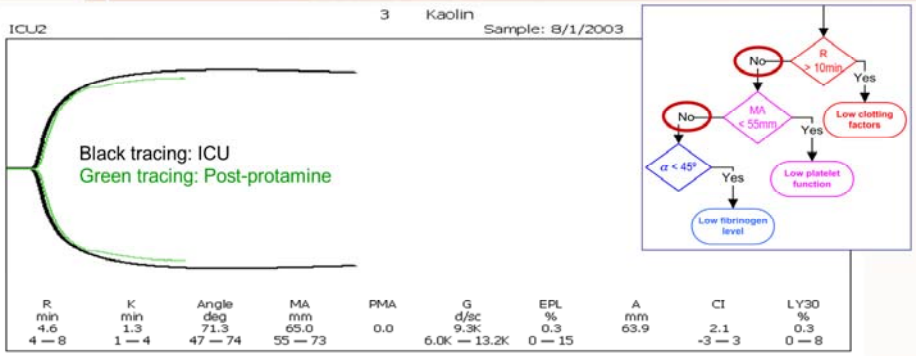
d) Fibrinolysis

e) Anticoagulant effect

Next

Menu

Answer to Exercise 10



This patient began bleeding approximately 1 hr post-op. What is the most likely cause(s) of bleeding?

a) Surgical bleeding

b) Factor deficiency

c) Platelet deficiency/dysfunction

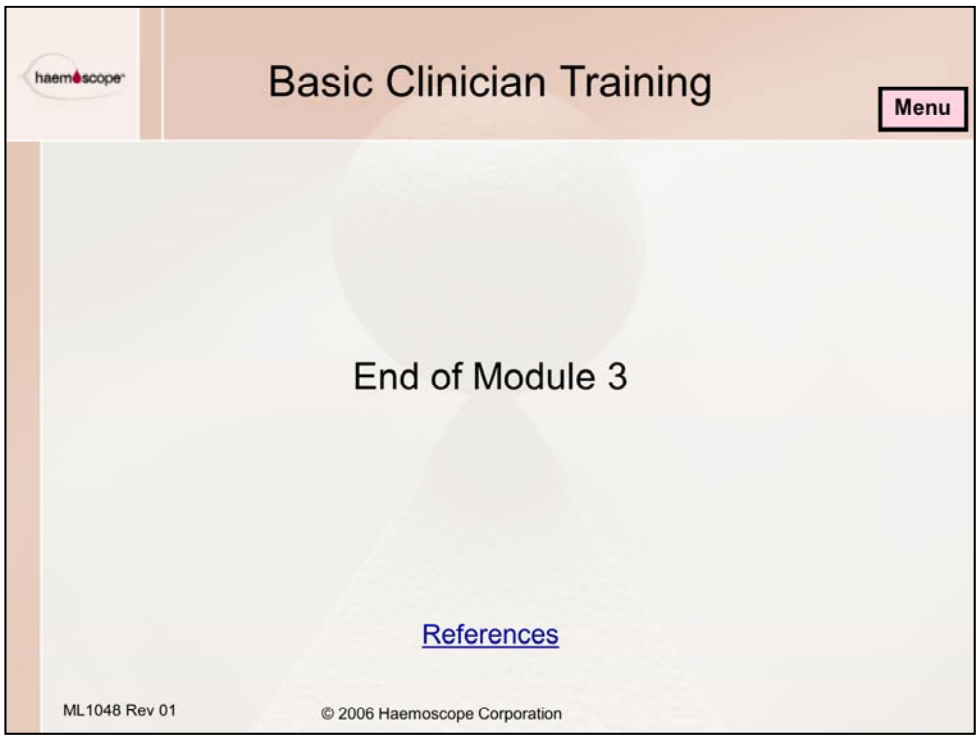
d) Fibrinolysis

e) Anticoagulant effect — Since this is a kaolin sample, heparin is not a contributor

How would you treat this patient? If von Willebrand factor deficiency and antiplatelet drugs have been ruled out, consider returning to the OR for re-exploration.

End

Menu


haemoscope
Basic Clinician Training
Menu
End of Module 3
References
ML1048 Rev 01
© 2006 Haemoscope Corporation