THE RESULTS OF THROMBOELASTOGRAPHY: DOES IT FIT WITH LABORATORY TESTING ?

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The use of thromboelastography (TEG) to monitor whole-blood coagulation was first described by Hartert in 1948. This technique provided information on the initiation of coagulation, propagation kinetics, fibrin-platelet interaction, clot firmness and fibrinolysis. By miniaturization of the technology for the thromboelastograph (TEG®; Haemoscope Corporation, IL, USA) and the use of standardized reagents containing activator for the modified rotation thrombelastogram analyzer (ROTEM®; Pentapharm, Munich, Germany), the bedside use of TEG has been developed.

Using standardized reagents, some TEG parameters have been demonstrated to be correlated to laboratory testing.
We have shown that in trauma patients, the clot firmness is correlated to protrombin time using a tissue factor containing reagent, and to fibrinogen using a reagent containing tissue factor and platelet antagonist.

In comparison with laboratory testing, in trauma patients or during orthotopic liver transplantation (OLT), thromboelastography could detect earlier and more quickly (≤ 15 minutes) a hyperfibrinolysis. The test would be of interest to guide which patients may benefit from treatment in association with antifibrinolytic agents.
Using heparinase containing reagent, thromboelastography could contribute to the assessment of coagulopathy versus the heparin effects in cardiac surgery or in OLT

ROTEM® may also be useful in the management of patients with severe FXIII deficiency particularly in patients receiving prophylaxis. Current chromogenic methods lack accuracy for low FXIII levels. In our experience, ROTEM® was able to detect viscoelastic changes of fibrin clot in whole blood samples with low FXIII levels (< 15 UI/dl) and has been successfully used to monitor FXIII replacement therapy.

In the laboratory the technology has been applied to areas where conventional testing is inadequate. Recently, thromboelastography has been evaluated in the monitoring of replacement therapy in rare bleeding disorders especially with by-passing agents, given to the absence of effective laboratory methods for monitoring these treatments. Using a reagent containing a very low concentration of TF, only an increase of the initiation phase is observed without any change of clot firmness. This would be, at least partly explained by the large interindividual variation of TEG patterns. Moreover, this interindividual variation was also observed in haemophilia patients with inhibitor before and after treatment with rFVIIa. Similarly, TEG could not be a method to monitor treatment by rFVIIa in patients with Glanzmann thrombasthenia.