The evaluation of coagulation disorders in critical patients is traditionally done on the basis of activated partial thromboplastin time (aPTT) and the normalized prothrombin time using the International Normalized Ratio (INR). These give us information about the time for the clot formation after the addition of exogenous reagents. The aPTT was designed to determine any Factor VII, IX and XI deficiencies, while the PT is used in the clinic to detect any Factor II, V and X deficiencies to follow warfarin-treated patients.1

These tests must be carefully interpreted since they do not measure the in vivo activity, and neglect the evaluation of the vascular and platelet interaction that could be promoting bleeding and hence may overestimate the homeostasis, particularly in patients with hypothermia.2 Usually, these tests are accompanied by platelet count but no information is provided as to the role of the adhesion and platelet aggregation tests that also have some limitations due to the poor sensitivity to detect the effect of low doses of antiplatelet aggregation agents.

The new approach of coagulation activation is based on the concomitant interaction of the two traditional pathways with platelets and other cells such as monocytes and fibroblasts, that results in an understanding of the viscoelastic properties of blood as described by Dr. Kang et al., for the management of coagulopathy during liver transplantation and extracorporeal circulation in cardiac surgery. This has been possible with the use of thromboelastography that evaluates the different phases of coagulation, starting with the Reaction Time corresponding to the generation of thromboplastin and reflects the intrinsic system function, particularly of Factors XII, XI and VIII, followed by the Coagulation time that measures the rate at which a solid clot develops as a result of the action of the intrinsic system, platelets and fibrinogen, followed by the Evaluation of Fibrinolysis. Other measures are the alpha angle that represents the velocity at which a solid clot develops; the maximum amplitude that would be the largest amplitude of the clot and is a function of the clot’s elasticity; and, finally, the Lysis Index that is the clot percentage that has presented fibrinolysis in thirty minutes.3–5

The thromboelastography exam can identify specific disorders such as the decrease in coagulation factors and/or platelet alterations and may show the relationship between platelets, fibrinogen and coagulation proteins. Furthermore, it identifies the dilutional coagulopathy evoked by volume exchange in trauma patients, in addition to hypothermia and extravascular disseminated coagulation due to the activation of clotting and the use of factors with the end result of a decreased procoagulation capacity. All of these factors are reviewed by Dr. Gálvez and Dr. Cortés in their review “Nuevos conceptos en la fisiología de la hemostasia y su correlación con la coagulopatía asociada al trauma”6 that provides a detailed explanation of the physiological mechanisms that participate in hemostasis, with a focus on the usefulness of thromboelastography as a diagnostic method with proven benefits in surgical cardiovascular and liver transplant patients, by enabling a comprehensive assessment of the most frequent coagulation disorders and

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guiding in a more objective and rational fashion the use of hemoderivatives. 7

However, the role of thromboelastography in the diagnosis and treatment of coagulation abnormalities in the septic patient is yet to be established, 8, 9 since clearly, most coagulation disorders in these patients are secondary to the systemic activation of the coagulation pathways by infectious agents and cytokines (TNF-α, IL-1) that stimulate the release of tissue factor from the monocytes and the endothelium when the latter is impaired. Thus, the approach to deal with these abnormalities is to correct the primary disorder that triggers the cascade of events. 10

Consequently, it is absolutely compelling to assess the available evidence on the benefit of thromboelastography in other critical patients whose bleeding is not secondary to liver transplantation, trauma or obstetric hemorrhage, based on previously asked questions, so as to select the articles in accordance with an explicit process that screens the relevant articles to answer the questions asked and hence avoid biases in selecting the articles.

**Conflicts of interest**

The author has no conflicts of interest to declare.

**REFERENCES**


