Thromboelastography (‘TEG’) and rotational thromboelastometry (‘ROTEM’) are used increasingly in cardiac and liver surgery to evaluate clotting factor deficiencies and platelet dysfunction. Both monitor viscoelastic properties of whole blood as it is induced to clot in a low-shear environment (resembling sluggish venous flow) and provide valuable information on haemostasis from initiation to fibrinolysis.

Our case illustrates the limitations that routine clotting tests have in predicting the risk of haemorrhage and we postulate that had results of TEG or ROTEM been available the extent of the coagulation defect might have been determined more expeditiously. We believe that there may be an increasing role for such point of care testing in this setting.

Conflict of interest
None declared.

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Comparison between RapidTEG® and conventional thromboelastography in cardiac surgery patients

Editor—Coagulopathy after cardiopulmonary bypass (CPB) remains a complex issue in terms of monitoring and treatment. Point-of-care testing with thromboelastography (TEG) has shown to have a positive predictive value of 87–89% and a negative predictive value of 92% for postoperative haemorrhage. Since it takes 15–20 min to obtain complete conventional TEG results, a more rapid modification, Rapid-TEG®, was developed, which incorporates tissue factor to the kaolin-activated thromboelastogram. This double activation accelerates both the intrinsic and extrinsic coagulation pathways. We designed a study to assess if the data obtained from RapidTEG® concerning clot strength correlate with similar data from conventional TEG.

In 24 adult cardiac surgery patients, three samples were obtained from each patient, one before (baseline) and two after CPB (one immediately after protamine and one 10 min later). All samples underwent simultaneous analysis with RapidTEG® and conventional TEG. R- and K-times, α-angles, and MA amplitudes were compared. Pre- and post-CPB platelet counts were recorded as well. Data are reported as mean (±SD) or median (inter-quartile range). The Wilcoxon rank-sum test was used for intergroup comparisons. The Spearman correlation (ρ) and locally weighted regression analysis were used to examine the association between different TEG measurement methods. Bias and limits of agreement were investigated by the Bland–Altman analysis. A P-value of <0.05 was considered statistically significant.

MA amplitudes decreased significantly with both TEG methods during CPB. With conventional TEG, from baseline median 70.0 (66.4–72.5) to 59.4 (57.4–66.5) mm (P<0.0002) in sample 1 and to 59.3 (56.9–65.1) mm (P<0.0001) in sample 2. With RapidTEG®, from 67.2 (62.4–69.6) to 60.8 (57.3–63.8) mm (P<0.0001) in sample 1 and to 62.0 (57.2–66.1) mm (P<0.01) in sample 2.

Of all the TEG variables studied, only MA amplitudes demonstrated a significant correlation between conventional and RapidTEG®. The Bland–Altman analysis showed minimal bias for baseline values (4.5%) and Protamine-2 (−1.6%), with small 95% limits of agreements (within 20%). More variations in MA values were noted at the time point of Protamine-1 (Fig. 1) Median platelet count decreased significantly from 238 (190–277) × 10^9 litre⁻¹ pre-CPB to 142 (108–170) × 10^9 litre⁻¹ post-CPB (P<0.0001).

The results show that there is a significant correlation for MA magnitude data obtained with RapidTEG® compared with conventional TEG, providing a reliable indication of maximal clot strength. The correlation was better 10 min after heparin reversal. MA values are reported to be mainly dependent on platelet numbers and on fibrinogen levels. The relationship between platelet numbers and MA values was confirmed in our study. In contrast, there was no correlation for α-angles, representing the speed of clot formation, including platelet activity. Earlier studies have shown that these values are strongly depending on concentrations of fibrinogen and FXIII. They are apparently significantly affected by RapidTEG®.

These findings are important, since impaired haemostasis after CPB is considered to be partly due to platelet dysfunction, which has a multifactorial underlying mechanism including preoperative medication, fibrinolysis, receptor defect, contact activation, and hypothermia.
RapidTEG® allows more rapid detection of impairment of clot strength due to low platelet numbers. Larger studies, preferably multicentre and prospective, have to be performed to assess if RapidTEG® may contribute to the reduction of transfusion requirements.

**Conflict of interest**

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