Thrombelastography is a ‘near patient’ test of coagulation. It is easy to perform and can provide information on a patient’s coagulation status within 30 min. Despite more than 25 years of clinical experience, however, several basic questions relating thromboelastograph (TEG) parameters to standard coagulation tests remain unanswered, and the value of the TEG is established only in the setting of orthotopic liver transplantation and cardiopulmonary bypass surgery. This review will focus on the principles and practise of the TEG, and data supporting the current accepted uses. Potential future uses will also be discussed including evaluation of hypercoagulable states, and investigating the mechanism of coagulopathies due to drugs or disease that standard tests have failed to unravel. Blood Coagul Fibrinolysis 12:327–337 © 2001 Lippincott Williams & Wilkins.

Keywords: thrombelastography, ‘near patient’ monitoring, review

Introduction

Thrombelastography was developed by Hartert in Heidelberg, Germany, during World War II as a research tool [1]. Its entry into clinical practice was pioneered by Kang in the setting of liver transplantation at Pittsburgh, USA more than 25 years later [2] and, more recently, it has been evaluated in cardiac surgery [3]. Both of these operations are performed against a background of multiple potential causes for haemostatic derangement and are often associated with massive blood loss. Transfusion of blood products may lead to a worsening coagulopathy and can be associated with immunological complications and the transmission of infection. Furthermore, transfusion in these operations may be costly and place a burden on a region’s blood supply. Strategies to decrease perioperative blood usage are therefore considered to be beneficial from the point of view of both the patient and the transfusion service.

In the clinical management of an acutely bleeding patient, it can be difficult to unravel the relative contributions of surgical haemostasis versus endothelial injury, platelet dysfunction, abnormalities of the coagulation proteases or their inhibitors and excessive fibrinolysis. In particular, there are no rapid standardized laboratory tests that can reliably monitor platelet function or fibrinolysis.

Thrombelastography poses many theoretical advantages to standard available laboratory tests. First, it is a ‘near patient’ test that is easy to perform and can yield results within 30 min. By measuring various parameters, it is able to yield information relating to the cumulative effect of several components of coagulation at a given time point. Its use has therefore been established to guide blood product administration in patients undergoing orthoptic liver transplantation (OLT) and cardiopulmonary bypass (CPB), both of which are associated with multifactorial coagulopathies and require rapid assessment of platelet function, fibrinolysis and overall coagulation status.

At the Royal Free Hospital, where the thromboelastograph (TEG) has been used as an adjunct to liver transplantation since the 1980s, it is also used in the surgical and intensive care settings to guide blood component administration. Potential future uses of the TEG include evaluation of hypercoagulable states, and investigating the mechanism of
coagulopathies due to drugs or disease that standard tests have failed to unravel.

This review will focus on the principles and practise of the TEG®, and data supporting the currently accepted uses. Further potential uses will also be discussed.

**Principles of thrombelastography**

Thrombelastography gives a graphic representation of aspects of clot formation and lysis. It is performed on a small quantity (0.35 ml) of whole blood that is placed in a heated (37°C) cup. The cup oscillates 4° 45' in either direction every 4.5 s. A pin is suspended in the cup of blood from a torsion wire that is mechanically or electrically transduced to a chart recorder or computer monitor, respectively. Initially, when no clot exists, the motion of the cup does not affect the pin and a straight-line trace is recorded. As the blood in the cup clots, however, the motion of the rotating cup is transmitted to the pin.

Four parameters are routinely measured, as indicated in Figure 1. The 'r time' (reaction time) is the latency time from placing blood in the cup until the clot starts to form (taken as reaching a TEG® tracing amplitude of 2 mm). The 'K time' is arbitrarily assigned as the time between the TEG® trace reaching 2 mm and going up to 20 mm. The alpha angle is a slope drawn from the slope of the TEG® tracing from the r to the K value. The maximum amplitude (MA) is the greatest vertical amplitude of the TEG® trace. The whole blood clot lysis index is the amplitude 60 min after the MA is achieved (A60) as a percentage of MA. Alternatively, the LY30 measures the rate of amplitude reduction 30 min after MA. A modern TEG® 5000 machine is shown in Figure 2 together with a normal trace generated by computerized software.

**Significance of the parameters measured**

The r time, K time and alpha angle assess factors involved between contact activation and fibrin formation, such as the function of circulating plasma clotting factors and their inhibitors and platelet function. The MA is taken to represent the ultimate strength of the clot, and the subsequent reduction in amplitude indicates the efficacy of fibrinolysis.

**Variations on the TEG®**

Coagulation in vitro is usually initiated by the addition of calcium to citratted plasma. For TEG® analysis, use of native whole blood is more reliable [4]. If rapid information is required, then the sample can be activated with celite, thrombin or tissue factor. The effect of heparin, either exogenous or endogenous, can be evaluated by placing the blood samples into cups that have been coated with the enzyme heparinase. This is an enzyme isolated from Flavobacterium heparinum and, unlike protamine, it antagonizes the effects of heparin without itself affecting TEG® variables [5,6]. Heparinase cups used in parallel with non-coated cups can be used to either demonstrate the presence of heparin or to monitor coagulation in the presence of full heparinization.

**Liver transplantation**

Bleeding is a common complication of OLT, and patients who have excessive blood loss during the procedure have high rates of morbidity and mortality postoperatively [7,8]. In addition to surgical causes for bleeding, derangement of haemostatic factors may contribute since the liver is the major site of synthesis of coagulation factors. In addition to factor deficiencies, dysfibrinogenaemia and enhanced fibrinolysis may occur in patients with liver
disease. Furthermore, the platelet count may be low due to inadequate marrow production, sequestration associated with hypersplenism or consumption by low grades of disseminated intravascular coagulation (DIC). Inadequate vitamin K absorption may occur as a result of failure of bile salt secretion into the intestine.

When a patient’s liver is removed (the anhepatic phase) fibrinolysis increases, and this can be visualized on a TEG® trace as a reduction in the MA (Fig. 3). The increased fibrinolysis is partly due to increased plasma levels of tissue plasminogen activator (tPA) [9,10], which would normally be cleared from the blood by the liver. Fibrinolytic activity may be further increased by low levels of alpha-2 antiplasmin, the main physiological inhibitor of plasmin that is synthesized in the liver [9].

![Figure 2](image1.png)

**Figure 2.** Thrombelastograph® 5000 coagulation analyser (courtesy Medicell Ltd.) and a normal trace generated by computerized software (courtesy of Dr Sue Mallet, Royal Free Hospital). Parameters are given together with a normal range in parentheses. MA, Maximum amplitude.

![Figure 3](image2.png)

**Figure 3.** Progressive fibrinolysis is represented by a progressive reduction in the maximum amplitude (thrombelastograph trace courtesy of Dr Sue Mallet, Royal Free Hospital).

![Figure 4](image3.png)

**Figure 4.** This two-channel, two-colour thrombelastograph trace was generated by simultaneous recordings from a standard cup and a heparinase-coated cup at the reperfusion phase of orthoptic liver transplantation (courtesy of Dr Sue Mallet, Royal Free Hospital). The native sample (blue trace) initially generates a straight-line trace indicating the reperfusion coagulopathy. The improvement that occurs when engraftment occurs is indicated by a widening in the amplitude of the trace. Comparison with the trace generated from the heparinized cup (red trace) enables the contribution of heparin to the coagulopathy to be assessed. The reduced maximum amplitude (MA) seen in the red trace from the heparinase cup is indicative of a persistent underlying coagulopathy in this patient, unrelated to heparin administration.
On reperfusion of the grafted liver, there is a further deterioration in coagulation. This is related to endothelial cell injury in the donor organ leading to further release of plasminogen activators and heparin. Exogenous heparin from the donor liver may also contribute to the coagulopathy since it is standard practice in the UK to administer 300 U/kg heparin to all donors prior to organ harvesting. In practise, the reperfusion coagulopathy resolves as graft function improves. The contribution of heparin to the reperfusion coagulopathy of OLT has been elegantly demonstrated using TEG® analysis [11,12]. A straight-line native TEG® trace seen immediately after reperfusion is partially corrected in a trace from a heparinized sample (Fig. 4). Administration of intravenous protamine restores the native thrombelastography trace to that of the heparinase-treated sample [11].

Administration of whole blood or blood components (platelets, fresh frozen plasma (FFP) and cryoprecipitate) is almost invariable during OLT. In standard haematology practise, administration of such products are recommended on the basis of the platelet count, the prothrombin time (PT), the activated partial thromboplastin time (aPTT) and the fibrinogen level. These standard coagulation tests, however, have been criticized for being too slow to be clinically useful perioperatively and, in 1985, Kang et al. [2] pioneered the use of the TEG® in monitoring haemostasis for OLT. They used the following TEG®-guided algorithm to guide blood product replacement therapy in 66 patients undergoing OLT: an r time > 15 min was treated with 2 U FFP, a MA < 40 mm was treated with 10 U platelets. Six units of cryoprecipitate were given if these did not improve coagulation or when the alpha angle was less than 45°. The blood requirements of these patients were compared with a historical control group who had received OLT without TEG® monitoring. They found that the TEG®-monitored group received more platelets and cryoprecipitate but 33% less blood and fluid infusion overall.

Pharmacological methods of reducing blood loss in OLT include decreasing fibrinolysis, neutralizing heparin or using agents with procoagulant activity. These methods are potentially hazardous, however, since their injudicious use could be associated with thrombosis or DIC. The TEG® has been of particular value in this setting because it allows rapid assessment of the effect of a pharmacological intervention, and it also allows assessment of agents in vitro prior to administration. The following have been monitored by TEG® agents in the context of OLT: tranexamic acid [13,14], epsilon aminocaproic acid (EACA) [15], aprotinin [16], desmopressin [17], and conjugated oestrogens [18].

Cardiac surgery
Bleeding after cardiac surgery is a major cause of post-operative morbidity [19] and is usually due to inadequate surgical haemostasis. Several defects in coagulation have been described, however, that may contribute to the increased risk of bleeding in such patients. Heparin is used to prevent blood clotting on contact with the surface of the extracorporeal circuit and this may, on occasion, be excessive or inadequately neutralized. The hypothermia that is induced perioperatively to reduce neuronal damage leads to a functional platelet impairment that is proportional to the degree of hypothermia. The number of platelets and their function are also altered by cellular trauma induced by oxygenators, roller pumps and pump suction devices. Fibrinolysis is induced by the interaction of blood with foreign surfaces of the extracorporeal circuit [20]. In the past, the bleeding diathesis was attributed mainly to platelet defects, but the success of high-dose aprotinin [21] and tranexamic acid in reducing perioperative bleeding suggests that hyperfibrinolysis is a major contributor to the bleeding diathesis.

The use of the TEG® in cardiac surgery has been threefold. First, the TEG® has been assessed in its ability to identify patients at increased risk of bleeding during or after bypass. Second, TEG® monitoring can contribute to the assessment of surgical versus haemostatic bleeding postoperatively, and influence whether a bleeding patient returns to theatre for surgical exploration. Third, a TEG®-related algorithm can be used to guide the administration of blood products during cardiopulmonary bypass.

The value of TEG® measurements in assessing the risk of postoperative bleeding has been assessed pre-, during and post-CPB. Preoperative assessment of haemostatic parameters has not been successful in identifying patients who will bleed excessively. A prospective evaluation of 60 patients undergoing CPB correlated the bleeding time, PT, platelet count and TEG® with intra-operative blood loss [22]. This study demonstrated that all components of the TEG® failed to predict blood loss. The bleeding time, platelet count and PT, however, could be modelled to predict blood loss.

Conversely, post-bypass TEG® parameters have been shown by several authors to correlate with the risk of post-operative bleeding. Spiess et al. [3]
monitored 38 patients undergoing cardiac surgery with the TEG® and a coagulation profile comprising a PT, aPTT, fibrinogen and platelet count. Postoperatively, chest tube drainage was assessed for 24 h. Their results indicated that the postoperative TEG® was a significantly better predictor of postoperative bleeding than the standard coagulation profile, having an accuracy rate of 87% compared with 51%.

Tuman et al. [23] evaluated 42 adult patients before and after bypass. Nine of these bled excessively and, in these patients, three postoperative TEG® variables (α, MA and Aₖ₀) were significantly altered compared with either the preoperative readings or with the parameters of non-bleeding patients.

Mongen and Hosking [24] showed that a low MA (< 50 mm) postoperatively was associated with significantly more mediastinal chest tube drainage and increased administration of blood products. The accuracy of the TEG® in predicting blood loss was lower than that quoted by Spiess et al., however, at 78%. This group also demonstrated that, in patients identified by the TEG® as being at risk of postoperative bleeding, intervention by administration of desmopressin could reduce mediastinal chest drainage.

Essell et al. [25] measured the r and K times, the alpha angle, MA and Aₖ₀ in 36 patients before and after bypass, eight of whom had at least one postoperative TEG® parameter indicative of hypocoagulability. Again, the patients with abnormal TEG® parameters had a significantly higher postoperative blood loss than the patients (n = 27) who had a normal postoperative TEG®. These authors also concluded that the post-bypass TEG® was of value in influencing whether a bleeding patient returned to theatre for surgical exploration, since a normal TEG® trace in this setting made surgical bleeding more probable.

The value of the TEG® in guiding blood transfusion perioperatively was highlighted by Spiess et al. in 1995 [26]. They published a retrospective analysis of blood transfusion requirements, blood donor exposure and re-exploration for haemorrhage in patients who either had (n = 591) or had not (n = 488) been evaluated perioperatively by TEG®.

No rigid transfusion protocol had been enforced in these patients but if the TEG® was abnormal postoperatively then efforts were made to normalize the TEG® with 'appropriate' therapy. If bleeding continued with a normal TEG®, then the patient was returned to the operating room. This study demonstrated a significantly lower incidence of overall transfusion (78.5 versus 86.3%), median donor exposure (6 versus 8) and mediastinal re-exploration for haemorrhage (1.5 versus 5.7%) in patients monitored by TEG®.

More recently, Shore Lesserson et al. [27] evaluated the use of a TEG®-related transfusion algorithm to guide administration of blood products following protamine administration during cardiac surgery. One hundred and five patients were randomized to either a TEG®-related algorithm or a non-TEG®-based transfusion policy during cardiac surgery. In both cases, 10 U cryoprecipitate were transfused if the fibrinogen level was less than 100 mg/dl and the patient was bleeding. The TEG® management algorithm additionally included the following:

1. 50 mg protamine was given if the heparinase-modified TEG® r time was less than one-half of the non-heparinase r time;
2. 6 U platelets were transfused if the platelet count was less than 100 000/μl and the TEG® MA < 45 mm;
3. if bleeding persisted and the r time was > 20 mm, 2 U FFP were given;
4. if bleeding persisted and the LY₃₀ was > 7.5% (i.e. evidence of fibrinolysis), additional antifibrinolytic therapy (10 g EACA) was given at the discretion of the physicians caring for the patient.

In the control group, additional protamine was given if the activated coagulation time exceeded baseline by 15%, 6 U platelets were given if the platelet

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Figure 5. A thrombelastograph trace from a patient with primary biliary cirrhosis demonstrating an increased alpha angle and high maximum amplitude (MA) indicative of hypercoagulability (courtesy Dr Sue Mallet, Royal Free Hospital).
count was less than 100, and 2 U FFP was given if the PT was greater than 150% of the control. If bleeding persisted, then an additional bolus of antifibrinolytic therapy (10 g EACA) was given at the discretion of the physicians caring for the patient.

Mediastinal tube drainage was assessed every 6 h for 2 days and was not statistically different 6, 12 or 24 h postoperatively. There was, however, a significant decrease in the number of patients receiving FFP and platelets postoperatively in the TEG®-monitored group and in the total volume of FFP administered to the latter group.

Future possible uses of the TEG®

Screening for hypercoagulability

Standard laboratory tests that may indicate hypercoagulability and a tendency to form thromboses include a high platelet count, high levels of coagulation factors VII, VIII, IX, X, and XI or fibrinogen, low levels of protein S, protein C, antithrombin, and the presence of a lupus anticoagulant. TEG® indicators of hypercoagulability are a short $r$ time, a large MA or an alpha angle that is large and rapidly increasing (Fig. 5). Such parameters have been noted with increased frequency, in a variety of patient groups who are considered to be at increased risk of thrombosis, e.g. in pregnancy and post-partum [28–30], in patients with primary biliary cirrhosis and primary sclerosing cholangitis [31], following general anaesthesia [32,33], and in patients with morbid obesity [34]. The clinical relevance of such information is unclear, however, since there is little data available to indicate that the TEG® may be of value in discriminating patients who will develop thromboses from those who will not.

One prospective study measuring preoperative TEG® parameters and equating this to the development of DVT following abdominal surgery has been reported [35]. Postoperative thrombosis was detected with the 125I-fibrinogen uptake test (FUT) performed on days 1, 3, 5 and 7. Of 100 elective surgical patients, 50 received low-dose subcutaneous heparin. There was no correlation between a hypercoagulable state detected by TEG® and a positive FUT. When subjects who were not receiving heparin were evaluated on their own, however, the MA became predictive of deep vein thrombosis (DVT) development, with a sensitivity of 72.2% and a specificity of 69%. While the predictive value of the TEG® is higher than some other systems reported for general surgery, a role for the TEG® in reducing the number of postoperative DVTs or the number of patients exposed unnecessarily to prophylactic heparin has yet to be established.

There have been preliminary investigations into the relationship between hypercoagulable TEG®s and prothrombotic screening tests. Ben Ari et al. [31] found abnormal levels of proteins C, protein S or antithrombin or activated protein C resistance in 6/22 patients with hypercoagulable TEG® parameters. This result is hard to interpret since there were no data to indicate which patients subsequently developed thromboses.

Preliminary data from the Royal Free Hospital suggests that the TEG® may be of value in screening patients who are being investigated for a suspected prothrombotic state. Twenty-five consecutive patients were evaluated by TEG® in addition to a standard thrombotic screen consisting of the following: FBC, PT, aPTT, fibrinogen, thrombin time, dilute Russell’s viper venom time, protein C, protein S, factor VIII, activated protein C resistance, plasminogen activator inhibitor-1, plasminogen, antithrombin, anticardiolipin antibodies and genotype analysis for factor V Leiden and prothrombin 20210 mutation. Seven of seven patients with a normal TEG® had a normal thrombotic screen, and 10/15 patients with a hypercoagulable or borderline TEG® had an abnormality in their thrombotic screen [36]. Further data is needed, however, before we can recommend that TEG® screening precedes laboratory evaluation for thrombosis.

Evaluating the effects of pharmacological agents on coagulation

Antiplatelet drugs. The TEG® has been used by several groups to investigate the effect of aspirin, in the hope of obtaining clinically useful data to indicate risk of bleeding. However, following ingestion of low-dose aspirin, TEG® parameters remain in the normal range in both pregnant and non-pregnant subjects. Mallett and Platt showed that there was no difference in the $r$ value and MA of 25 healthy subjects following ingestion of 150 mg aspirin daily for 14 days [37]. Orlikowski et al. [38] similarly found the TEG® $r$ time, $K$ time and MA to remain in the normal range in nine pregnant women ($>18$ weeks gestation) receiving 75 mg aspirin a day, using 41 healthy antenatal women ($>18$ weeks gestation) as control subjects. Data relating to the effect of ingestion of larger doses of aspirin (600 mg) on TEG® parameters is less conclusive. Although one study [39] indicated sig-
nificant prolongation of the \( r \) and \( K \) times 2 h after ingestion of 600 mg aspirin, subsequent data on pregnant \((n = 12)\) and non-pregnant \((n = 8)\) subjects has shown unaltered \( r \) and \( K \) times and MA 6 h after ingestion of 600 mg aspirin despite significant prolongation of the bleeding time \([40]\).

The TEG® has also been used to monitor the effects of newer antiplatelet drugs; for example, c7E3Fab \([41,42]\) and WEB2086 \([43]\). These drugs have a narrow therapeutic window, and there is considerable inter-individual variability in response to them. The TEG® has been evaluated with a view to optimizing their dosage and minimizing the risk of bleeding.

**Drugs affecting fibrinolysis.** Fibrinolysis is readily demonstrated on a TEG® trace by a prolonged \( r \) time and a reduction in amplitude of the MA and \( A_{60} \) (Figs 1B,3). The effect of antifibrinolytic drugs can be assessed in vitro or in vivo using the TEG®, and this has been performed for several agents in the context of cardiac surgery as already discussed.

Agents that promote fibrinolysis can also be assessed using the TEG®. Summari et al. \([44]\) used the TEG® to quantify and compare the fibrinolytic effects of a variety of plasminogen activators in vitro. The TEG® is also used clinically in some centres to monitor thrombolytic therapy such as rtPA \([45]\).

**Heparin.** The value of the TEG® in assessing the effect of heparin on blood coagulation has already been discussed. The TEG® has also been used to measure anticoagulation resulting from the administration of low molecular weight heparins, \([46]\). This group of anticoagulants has several effects on the coagulation cascade, which vary from one formulation to another. Where monitoring is required, anti-activated factor X (anti-FXa) assays are recommended; however, this assay is not always readily available. Data examining the relationship between TEG® analysis and anti-FXa levels perioperatively in patients receiving enoxaparin has indicated a correlation between the \( r \) time and anti-FXa levels. Further studies are needed to confirm this and demonstrate clinical benefit.

**Obstetric practise**

Assessment of haemostasis is particularly relevant to obstetric practise because thrombosis and haemorrhage remain two of the commonest causes of maternal mortality \([47]\). Changes in the TEG® during normal pregnancy and in pre-eclamptic subjects have been reported. As already indicated, studies in healthy pregnant subjects indicate hypercoagulable parameters with reduced \( r \) and \( K \) times, and increased MA and \( \alpha \) angles \([28–30]\). These changes persist into the post-partum period \([30]\). In patients with pre-eclampsia, a significantly lower MA has been shown in severe cases where the platelet count is also low, while in patients with mild pre-eclampsia the TEG® is unaltered from that of healthy parturients \([48]\).

The TEG® is used in some departments to guide the administration of regional anaesthesia in situations where the parturient is considered to be at increased risk of haemorrhage, e.g. with a borderline low platelet count \((50–100)\). There is little data on which to base this practice, however. Although there is some data to suggest that the MA is related to the platelet count (see later), there is insufficient data to indicate that a normal MA in the face of a low platelet count should provide grounds for reassurance that a patient will not bleed excessively. A single case report describes a woman with immune thrombocytopenia (ITP) whose platelet count was 64 and TEG® was normal, in whom regional anaesthesia was administered successfully \([49]\).

Preliminary data suggests a possible role for the TEG® in the investigation of women with poor obstetric histories \([50]\). In a study of 205 non-pregnant women with a history of recurrent (three to nine) miscarriages, the MA was significantly higher than in a group of normal non-pregnant parous women. Furthermore, prospective evaluation of the TEG® in a group of 32 non-pregnant women indicated significantly higher MA readings in 10 women who subsequently conceived and miscarried compared with 22 women who had a live birth.

**Evaluation of mechanisms underlying clinically abnormal haemostasis**

The TEG® has been used to evaluate the a putative role for fibrinolysis in the postoperative bleeding that occurs following transurethral prostatectomy. Both systemic fibrinolysis and local fibrinolysis at the prostate bed have been hypothesized to play a role, and a possible role for antifibrinolytics has been entertained. Bell et al. monitored 30 patients undergoing prostate surgery by TEG®, and demonstrated hypercoagulable traces from 3 h post-operatively until 10–14 days later \([33]\). There was no evidence of fibrinolysis, and the authors concluded that the use of antifibrinolytics in these patients could be detrimental.

TEG® analysis of heparinized samples has also been useful in situations where the presence of heparin interferes with assessment of an additional
Identifying patients at risk of bleeding

A role for post-CPB TEG® to predict patients at risk of bleeding following cardiac surgery has already been discussed. Preliminary data also suggests a role for the TEG® predicting risk of bleeding in trauma patients. Kauffmann et al. [51] used the TEG® in the initial assessment of 69 trauma patients, the majority of whom (45/69) had hypercoagulable TEG®s. Seven of 69 were hypocoagulable, however, and this group had a higher incidence of blood transfusion within the first 24 h. Logistic regression analysis showed that the TEG® was predictive of early transfusion.

Comparison of TEG® to standard coagulation tests

Data comparing TEG® parameters with standard coagulation tests is extremely variable. Zuckerman et al. used three statistical methods to evaluate the relationship between TEG® parameters and the following standard tests: haematocrit, platelet count, PT, aPTT, fibrinogen and fibrin degradation products (FDPs) [52]. They investigated 141 normal controls and 121 patients with cancer. An association of fibrinogen with the MA was seen in both study groups but a relationship with the platelet count and the MA was seen only among cancer patients. There was no relationship between the PT, aPTT and FDPs with any of the TEG® variables in this study.

Kang et al. [2] used linear regression to evaluate the relationship between TEG® variables and a standard test profile consisting of PT, aPTT, thrombin time (TT), reptilase time, factors I, II, V, VII, VIII, IX, X, XI, and XII, FDP, euglobulin lysis time (ELT) and platelet count. In general, correlation was poor, but significant associations were noted between the MA and both platelet and fibrinogen level, the r time and aPTT, and the whole blood clot lysis time and the ELT. The relationship of platelet count and the MA was further investigated by measuring the MA and platelet count following infusion of platelets. Ten units of platelets led to an increment of 40 (± 31) x 10^9/l and an increase in the MA of 13.2 mm. Infusion of 6 U cryoprecipitate led to an increased fibrinogen of 37 mg% and a reduction of 5.7 s on the aPTT. Corresponding changes in the TEG® were a decreased r time (1.2 min) and an increased alpha angle. The effect on the MA was not described.

Orlikowski et al. [48] used Pearson’s correlation coefficient analysis to assess the correlation between TEG® variables and standard tests (platelets, fibrinogen, FDPs, PT, aPTT, TT, factors V and VIII, protein C, ATIII and bleeding time) in women with eclampsia and pre-eclampsia. They demonstrated an association between the MA and platelet count that became linear when the platelet count was less than 100. However, only seven patients in this study had platelet counts in this range.

Although the strongest association to emerge is that between the MA and the platelet count, even here data are inconsistent. Spiess et al. [3], for example, failed to demonstrate such an association. They used linear correlation analysis to investigate the association of TEG® variables with PT, aPTT, fibrinogen and platelets in 38 patients undergoing cardiac surgery. No association was found between the platelet count or fibrinogen level with the MA in these patients, either before or after bypass surgery. The only standard test to show a significant correlation with any TEG® variables was the aPTT. Pre-bypass data showed a positive association with the r time and a negative correlation with the alpha angle, MA and A₆₀. Post-bypass, however, only the A₆₀ and aPTT showed a positive association. Similarly, Dorman et al. [22] and Ben Ari et al. [31] failed to detect a significant association between MA and platelet or fibrinogen count in groups of patients undergoing cardiac surgery (n = 60) or with liver disease and hypercoagulable TEG® traces (n = 22), respectively. The former group did note a correlation between the PT and the alpha angle, however.

More recently, Oshita et al. [53] attempted to resolve the question of the relationship of the MA and platelet count by an in vitro study on blood samples from six preoperative patients undergoing elective surgery. By mixing each patients’ platelet-rich and platelet-poor plasma, they created six samples of plasma each with a different concentration of platelets for each patient. These samples were then evaluated by TEG® and statistical analysis was performed by linear regression and analysis of variance. This group demonstrated a linear association between the MA and log_{10} platelet count.

The inconsistencies of other studies are hard to explain but it may relate to the small sample sizes and the sensitivity of TEG® to aspects of testing technique and sampling [54].

Although the TEG® is widely considered to be a good indicator of platelet function, there have been underlying coagulopathy. This has already been discussed in the context of OLT.
few studies formally evaluating the relationship. An abstract by Tuman et al. [55] indicated a significant positive correlation between the alpha angle, MA and A\textsubscript{60} values with the aggregation responses to adenosine diphosphate and collagen. This has not been followed up by more detailed reports, however. McNulty et al. [56] investigated the TEG\textsuperscript{®} changes that occurred when blood samples were spiked with either fresh or cryodisrupted platelets in vitro. In both cases, there was a significant increase in MA and alpha angle but there was no significant difference between the two groups. These disrupted platelets would still provide a phospholipid surface and thus contribute to the maintenance of haemostasis; however, they would be unable to adhere or aggregate. While it is possible that the similarity of the two traces may have reflected disruption of the apparently normal platelets, this result suggests that the TEG\textsuperscript{®} is not a reliable indicator of platelet function. This is consistent with the studies already discussed that have shown the failure of the TEG\textsuperscript{®} to detect an aspirin effect, even in situations where the bleeding time is prolonged.

**Summary**

The TEG\textsuperscript{®} provides a rapid, inexpensive assessment of coagulation at the patients bedside. Correlation of TEG\textsuperscript{®} parameters with standard blood tests is poor; however, TEG\textsuperscript{®} monitoring is of established benefit to guide blood product and drug administration in OLT. Furthermore, TEG\textsuperscript{®} monitoring in cardiac surgery has been shown to reduce transfusion requirements. Further roles for TEG\textsuperscript{®} monitoring have yet to be established, pending prospective trials that link TEG\textsuperscript{®} variables to clinical outcome.

**References**


