Somato Publications ISSN: XXXX-XXXX

Archives of Hematology and Blood Transfusion

Original Article

Utility of Thromboelastography in Guiding Blood Product Resuscitation During Liver Transplantation

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Received: 26 July 2022; Accepted: 26 October 2022; Published: 15 January 2023.

Citation of this article: Rusnak S, Matuska B, Weisz A (2023) Utility of Thromboelastography in Guiding Blood Product Resuscitation During Liver Transplantation. Arch Hematol Blood Transfus, 5(1): 01-06. DOI: 10.5281/zenodo.22818757

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Abstract

Background: Coagulopathy in end-stage liver disease is a rebalanced, dynamic hemostatic state, not a simple deficiency, and standard coagulation tests correlate poorly with bleeding risk during liver transplantation.

Objective: To synthesize published evidence on thromboelastography and rotational thromboelastometry for guiding blood product resuscitation during liver transplantation.

Methods: Narrative synthesis of transplant-specific randomized trials, before-and-after algorithm studies, and mechanistic coagulation literature.

Findings: Viscoelastic assays track the clot physiology that drives surgical bleeding more closely than the INR, and goal-directed algorithms consistently reduce plasma, platelet and red-cell exposure without increasing bleeding complications, though certainty remains low to moderate.

Conclusions: Phase-anchored TEG or ROTEM sampling before incision, during the anhepatic phase, and after reperfusion supports blood product decisions on a timescale a delayed INR cannot match.

Keywords: thromboelastography; rotational thromboelastometry; liver transplantation; coagulopathy; goal-directed transfusion

1. Introduction

Orthotopic liver transplantation is one of the few operations in which the organ being resected is also the organ that manufactures most of the proteins responsible for stopping the bleeding it causes. A cirrhotic recipient arrives with a liver that has already stopped making adequate factor II, V, VII, IX and X, and the operation then adds portal hypertensive venous collaterals, an anhepatic phase with no hepatic synthetic function at all, and a reperfusion event that releases tissue plasminogen activator and heparinoid substances from the graft into the circulation. For much of the 1980s and 1990s the answer to this problem was empiric: give plasma to a fixed ratio, give platelets below a round threshold, and watch the field. Kang and colleagues changed that early, describing four reproducible stages of coagulation change across the preanhepatic, anhepatic, early post-reperfusion and late post-reperfusion periods using thrombelastography, and showing that a fibrinolytic pattern at reperfusion predicted the need for antifibrinolytic therapy well before the field looked diffusely oozy [1]. An Australian series of the first 75 cases monitored with thrombelastography at Austin Hospital corroborated the same phase-specific pattern [2], establishing viscoelastic testing as a bedside tool for a bleeding phenotype that standard plasma-based assays were never designed to capture.

Conventional coagulation tests — prothrombin time, INR, activated partial thromboplastin time, platelet count and fibrinogen — were developed to detect single-factor deficiencies in hemophilia, not to describe the net hemostatic balance of a complex, multi-factor deficiency state. Cirrhosis lowers procoagulant factors, but it lowers protein C, protein S and antithrombin in roughly parallel measure, and it raises factor VIII while lowering the VWF-cleaving protease ADAMTS13, producing a system that is reset rather than simply depleted [3]. An elevated INR reflects the loss of procoagulant drive without capturing the accompanying loss of anticoagulant drive, and correlates poorly with actual bleeding risk — a disconnect formalized when Tripodi and colleagues showed that thrombin generation in cirrhotic plasma, once thrombomodulin was added to simulate the endothelial anticoagulant surface, was frequently normal despite an INR of 1.5 to 2.5 [4], and extended by showing that the ratio of procoagulant to anticoagulant drivers overlapped healthy controls rather than being uniformly shifted toward hypocoagulability [5]. Steib and colleagues had already shown that preoperative standard coagulation values poorly predicted intraoperative blood loss and transfusion in a French transplant cohort [6]. This narrative synthesis lays out what the transplant-specific literature shows about using viscoelastic testing to guide blood product resuscitation, where that evidence is strong, and where it remains thin.

2. Coagulopathy in end-stage liver disease is not simple deficiency

Lisman and Porte's synthesis of rebalanced hemostasis described a system in which losses of hepatically synthesized procoagulant factors are offset, to a clinically meaningful degree, by parallel losses of protein C, protein S and antithrombin, together with a disproportionate rise in factor VIII and a rise in von Willebrand factor that substitutes, in part, for the low platelet count of portal hypertensive splenic sequestration [3]. Tripodi's thrombin-generation work made the point quantitatively: with thrombomodulin present, cirrhotic samples generated thrombin at rates overlapping healthy controls in a substantial fraction of patients despite an abnormal INR, and a formal procoagulant-to-anticoagulant ratio across cirrhotic patients showed no systematic shift toward net hypocoagulability [5]. Blasi and colleagues extended this to acute-on-chronic liver failure, the population most likely to need urgent transplantation, showing that standard tests and even some viscoelastic parameters correlated only weakly with a global thrombin-generation assessment, and that derangement tracked liver failure grade more closely than INR [7].

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Table 1: Direction of change in selected procoagulant and anticoagulant factors in cirrhosis, illustrating why an isolated INR does not describe net hemostatic balance [3,4,5].

Factor	Role	Change in cirrhosis
Factors II, V, VII, IX, X	Procoagulant	Decreased [3]
Protein C, protein S	Anticoagulant	Decreased, in parallel [3]
Antithrombin	Anticoagulant	Decreased [3]
Factor VIII	Procoagulant	Increased, disproportionately [3]
Von Willebrand factor	Platelet adhesion	Increased, offsetting thrombocytopenia [3]
Thrombin generation (with thrombomodulin)	Net coagulation capacity	Frequently normal despite prolonged INR [4,5]

The corollary, stated plainly by Stravitz, is that transfusing plasma to correct an isolated INR abnormality in a patient who is not clinically bleeding treats a number rather than a hemostatic deficit, and in a portal hypertensive candidate can raise portal pressure and paradoxically increase blood loss [8]. Krzanicki, Sugavanam and Mallett captured the other side directly: serial TEG tracings during transplantation showed a hypercoagulable pattern, with shortened reaction time and increased clot strength, in a substantial minority of intraoperative samples — precisely the patients in whom empiric plasma guided by a prolonged INR would be not merely unnecessary but potentially harmful [9]. Desborough and colleagues' nationwide audit of blood component use in cirrhosis documented wide hospital-to-hospital variation in transfusion before low-bleeding-risk procedures [10]. Coagulopathy in end-stage liver disease is a rebalanced, dynamic state rather than a static deficiency, and any monitoring strategy built around a single plasma assay will misclassify a meaningful share of patients in both directions.

3. Viscoelastic assays versus standard coagulation tests

Viscoelastic testing measures clot development in whole blood over time — initial fibrin polymerization, maximum clot strength, and subsequent breakdown — rather than the time to a fixed fibrin endpoint in citrated, centrifuged plasma. Roullet and colleagues tested this during transplantation, showing that ROTEM's fibrinogen-specific FIBTEM maximum clot firmness and EXTEM clotting time correlated with platelet count and fibrinogen level more closely than standard laboratory turnaround allowed, and that anhepatic-phase ROTEM values predicted the transfusion requirement of the reperfusion phase before it occurred [11]. McNicol's Australian series had already shown TEG tracings tracking the classic reperfusion coagulopathy — falling maximum amplitude and rising clot lysis — in close temporal step with observed surgical bleeding, at a point when a central-laboratory sample would not yet have resulted [2]. Turnaround is not incidental: a citrated INR sample sent during the anhepatic or reperfusion phase, when the coagulation state can shift within minutes, is frequently clinically obsolete by the time it results, whereas a point-of-care device produces an actionable platelet- and fibrinogen-specific tracing within 5 to 10 minutes.

Table 2: What standard plasma-based coagulation tests and whole-blood viscoelastic assays each measure, and their reported turnaround at the bedside during transplantation [11,12,2].

Property	PT/INR, aPTT, platelet count	TEG/ROTEM
Specimen	Citrated, centrifuged plasma	Whole blood
Platelet contribution	Not assessed (count only)	Functional contribution to clot strength [12]
Fibrinogen deficit	Separate, delayed assay needed	Isolated on fibrinogen-specific channel [12]
Fibrinolysis	Not assessed	Directly visualized as clot lysis [1,2]
Typical result turnaround intraoperatively	20 minutes or more, central laboratory	5-10 minutes, bedside [11]

Mallett, Chowdary and Burroughs framed the comparative advantage by what each assay is built to detect: INR responds to the extrinsic and common pathway and is insensitive to platelet number or function, to moderate fibrinogen deficits, and to fibrinolysis, whereas TEG/ROTEM is sensitive to all three and distinguishes a fibrinogen deficit from a platelet deficit

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without a separate, delayed assay [12]. Haas and colleagues' systematic review of viscoelastic-guided bleeding management across critically ill populations concluded that TEG/ROTEM parameters correlated better with clinically relevant coagulopathy than routine screening in most studies reviewed, while noting heterogeneity in assay thresholds and outcome definitions [13]. Pandey and colleagues showed a narrower, concrete application in cirrhotic patients undergoing central venous cannulation, in whom TEG reaction time and maximum amplitude, but not standard PT/INR or platelet count, predicted puncture-site bleeding [14] — consistent with the broader argument that standard tests measure the wrong thing for this population, not merely the right thing imprecisely.

4. Goal-directed algorithms and blood product sparing

The clinical case rests less on correlation than on whether acting on a TEG/ROTEM-guided algorithm changes what gets transfused, and the trial literature is direct here. Wang and colleagues randomized 28 transplant recipients to TEG-guided versus laboratory-guided transfusion and found the TEG-guided group received significantly less intraoperative plasma, without any difference in blood loss or postoperative bleeding [15]. A related retrospective analysis from the same program then examined whether more liberal TEG-derived thresholds increased surgical blood loss and found they did not, arguing the algorithm's benefit came from avoiding unnecessary transfusion rather than under-transfusing at the margin [16].

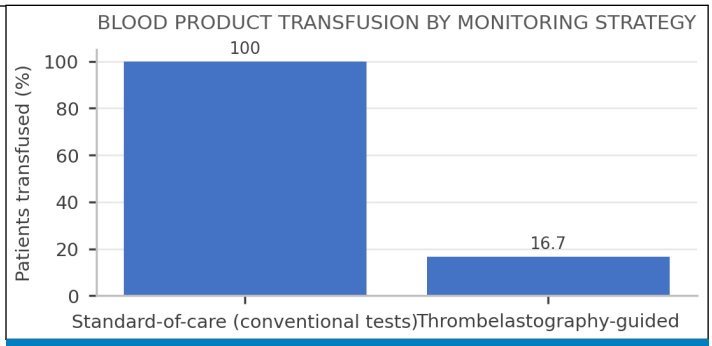

Figure 1: In a randomized trial of 60 cirrhotic patients with severe coagulopathy undergoing invasive procedures, blood product transfusion (fresh frozen plasma and/or platelets) occurred in all patients managed by a standard-of-care protocol keyed to conventional coagulation tests, compared with roughly one in six patients managed by a thrombelastography-guided algorithm, without a difference in bleeding complications between arms [17].

De Pietri and colleagues moved the same logic to the pre-procedural setting, randomizing 60 cirrhotic patients with severe coagulopathy undergoing invasive procedures to thrombelastography-guided transfusion versus a standard protocol keyed to conventional tests. Blood product transfusion occurred in a small minority of the TEG-guided arm compared with essentially all of the standard-protocol arm, with no significant difference in bleeding complications, one of the clearest demonstrations that viscoelastic guidance reduces plasma and platelet exposure without a safety penalty [17]. Roullet and colleagues reported a before-and-after comparison of introducing a ROTEM-based algorithm for transplantation; the algorithm era showed a marked reduction in patients exposed to plasma and a reduction in median red-cell transfusion, persisting after adjustment for MELD score [18]. Leon-Justel and colleagues reported a similar transition to point-of-care thromboelastometry in a Spanish program, with reduced plasma and platelet transfusion and shorter intensive care stay [19], and an earlier report from the same group had shown that adding fibrinogen concentrate to a thromboelastometry-triggered algorithm reduced allogeneic red-cell and plasma exposure [20]. Kirchner and colleagues, describing a large German cohort using factor-concentrate correction triggered by ROTEM rather than fixed-ratio plasma,

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reported substantially reduced allogeneic blood product use versus historical practice, with acceptable rates of postoperative thrombosis [21]. Görlinger and colleagues' cardiac surgery cohort of nearly 3,900 consecutive patients before and after adopting a ROTEM-and-factor-concentrate algorithm lies outside the transplant setting proper, but its scale and consistent direction — a marked reduction in allogeneic transfusion together with lower six-month mortality — is frequently cited in the transplant anesthesia literature as external corroboration that the mechanism generalizes [22].

Wikkelsø and colleagues' Cochrane review of viscoelastic-guided transfusion — pooling cardiac surgery, liver transplantation, trauma and obstetric hemorrhage trials — found a consistent direction of benefit for reduced red-cell, plasma and platelet transfusion, and a signal toward reduced mortality, but rated certainty as low to moderate given trial heterogeneity, modest samples and inconsistent blinding [23]. Donohue and Mallett's review of transfusion-reduction strategies situates the viscoelastic algorithm as one contributor, alongside surgical technique, cell salvage and a lower transfusion trigger, to a multi-decade decline in median red-cell requirement from double digits of units in early series to often fewer than two at contemporary high-volume centers [24]. Disentangling the algorithm's specific contribution from the rest of that trend is difficult from observational before-and-after data, a limitation the primary studies generally acknowledge.

5. Evidence during the anhepatic and reperfusion phases

The physiological case for viscoelastic monitoring is strongest during the two phases in which the coagulation state moves fastest: the anhepatic phase, when synthetic function is entirely absent and dilutional and consumptive coagulopathy compound uninterrupted, and reperfusion, when the graft releases tissue plasminogen activator and heparin-like glycosaminoglycans over minutes. Kang's original description characterized post-reperfusion fibrinolysis as reproducible and visible on the tracing within minutes of reperfusion, and showed that patients with this pattern went on to need antifibrinolytic therapy and had greater blood loss — a prediction unavailable from a standard panel sent before reperfusion and resulted twenty or more minutes later [1]. Krzanicki and colleagues extended the phase-specific picture, showing that some patients moved within the same operation from a hypocoagulable anhepatic tracing to a distinctly hypercoagulable post-reperfusion tracing, a trajectory a clinician anchored to a single preoperative INR could not anticipate, arguing for repeated, phase-triggered sampling rather than one intraoperative measurement [9].

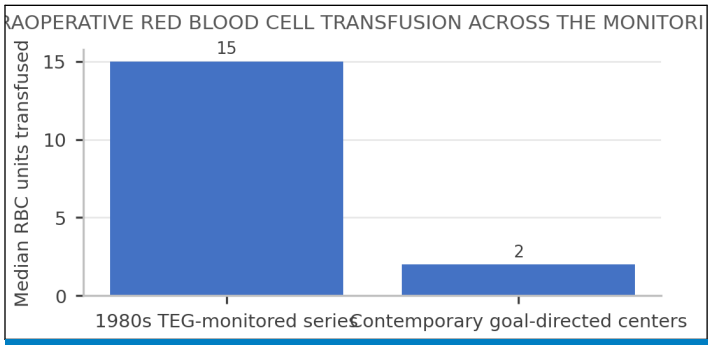

Figure 2: Reported median intraoperative red blood cell transfusion in orthotopic liver transplantation has fallen across the era of phase-anchored, viscoelastic-guided coagulation management, from double-digit unit requirements in early thrombelastography-monitored series to often fewer than two units at contemporary high-volume centers with mature blood-conservation and goal-directed transfusion programs; the two points bracket an illustrative range rather than a single controlled comparison [24,1].

Kirchner and colleagues' factor-concentrate cohort targeted correction specifically to the reperfusion phase, using FIBTEM-triggered fibrinogen concentrate and clotting-time-triggered prothrombin complex concentrate rather than empiric plasma, describing this phase-specific correction as central to the reduction in allogeneic transfusion observed [21]. Roullet's phase-sampled ROTEM data showed anhepatic-phase FIBTEM and EXTEM values predicted the reperfusion phase's

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transfusion burden with useful lead time, supporting anticipatory rather than purely reactive product preparation [11]. Bezinover and colleagues' consensus statement on perioperative coagulation management explicitly recommends phase-anchored sampling — before incision, during the anhepatic phase, and after reperfusion — as the minimum schedule for a goal-directed program, since a single measurement is likely to misrepresent at least one of the operation's three hemostatically distinct phases [25]. Massicotte and colleagues' transfusion-predictor analysis, at a center with aggressive intraoperative monitoring and a restrictive transfusion philosophy, found a substantial proportion of transplants completed without any allogeneic red-cell transfusion, reinforcing that the anhepatic-to-reperfusion window, rather than the preoperative baseline, holds most of the actionable coagulation information in this operation [26].

6. Limitations and implementation barriers

Three limitations recur. First, trial quality is modest: most of the studies cited above enrolled fewer than 100 patients, were single-center, and could not blind the treating team, all reasons the Cochrane reviewers rated overall certainty as low to moderate [23]. Second, TEG and ROTEM are not interchangeable, and thresholds validated for one device and reagent system do not transfer directly to the other; a program adopting a published algorithm must either replicate the source device or independently validate its own thresholds. Third, viscoelastic assays interrogate secondary hemostasis and fibrinolysis but not platelet-endothelial interaction under shear, so a normal tracing does not guarantee absence of a clinically relevant bleeding tendency, and Haas and colleagues noted inconsistency across studies in how "clinically relevant coagulopathy" was defined as the validating outcome [13].

Implementation carries its own barriers apart from the evidence question. Point-of-care devices, staff training, and the parallel availability of factor concentrates that several goal-directed algorithms above use preferentially over plasma require capital investment and, in some jurisdictions, a regulatory and cost pathway not available outside major transplant centers [25]. Desborough and colleagues found wide variation in whether any point-of-care coagulation testing was available at all for cirrhotic patients, suggesting the gap between the literature and routine practice is partly an access problem [10]. Clevenger and Mallett's broader review situates viscoelastic algorithms as one component of a bundle that also includes restrictive transfusion triggers, antifibrinolytic prophylaxis and surgical blood conservation, cautioning against reading reported transfusion reductions as attributable to monitoring technology alone [27]. Finally, most of the transplant-specific trial evidence predates the shift toward sicker, higher-MELD-score candidates at many centers, and whether earlier, lower-acuity cohorts' benefit extends unchanged to today's recipients is an open question.

7. Conclusion

Across four decades of published work, from Kang's original description of phase-specific coagulation change [1] through McNicol's early TEG series [2] to the randomized and before-and-after algorithm trials of the last fifteen years [15,17,18,19], the transplant-specific evidence converges: standard coagulation tests correlate poorly with the rebalanced, dynamic hemostatic state of end-stage liver disease [3,5], viscoelastic assays track the platelet-, fibrinogen- and fibrinolysis-dependent clot physiology that actually drives bleeding in this operation more closely [11,12], and goal-directed algorithms built on viscoelastic triggers reduce exposure to plasma, platelets and, in several series, red cells, without an accompanying increase in bleeding complications [15,17,21,22]. The certainty of that evidence remains lower than the consistency of its direction would suggest, constrained by small single-center trials, device-specific thresholds, and an implementation gap that is as much about access to point-of-care testing and factor concentrates as about

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clinical conviction [23,10,25]. The practical synthesis for a program considering adoption is not that viscoelastic testing replaces clinical judgment or renders standard coagulation tests obsolete, but that phase-anchored TEG or ROTEM sampling — before incision, during the anhepatic phase, and after reperfusion — supplies actionable, whole-blood information on a timescale that matches how fast the coagulation state of this operation moves, in a way a delayed, plasma-based INR structurally cannot.

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