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We appreciate the thoughtful comments and analyses provided by Meli et al (1). Despite clear differences in baseline hemoglobin, anticoagulation therapy, and disease in their cohort compared with ours, they identified comparable lower hemoglobin-faster/stronger viscoelastic hemostatic assay (VHA) tracing relationships. However, their identified relationships were separately influenced by platelet counts and fibrinogen concentrations.

Due to the absence of fibrinogen data and exclusion of patients with thrombocytopenia, we were unable to assess the impact of these variables (2). Thus, the findings from Meli et al (1) provide perspective suggesting hemoglobin-VHA tracing artifacts are seen when platelet counts and fibrinogen concentrations are stable/elevated. However, the full nature of these observations requires clarification as both low and high platelet counts had different impacts on their hemoglobin-VHA tracing relationships. It is additionally unclear whether platelet/fibrinogen function, which is not equivalent to mere counts, impact hemoglobin-VHA relationships. Furthermore, it is unknown whether confounders unique to their cohort: liberal RBC transfusions, anticoagulation, underlying disease, and the extracorporeal membrane oxygenation (ECMO) circuit itself influenced their observations. ECMO patients are particularly vulnerable to encountering complex thromboinflammatory responses, even independent to the ECMO circuit itself (3). Thus, different etiologies of anemia (hemolysis, blood loss, anemia of inflammation) and dynamic changes in coagulation factors, platelets, and fibrinogen occur unique to the patient's hospitalization course. Subsequently, it is unclear whether analyzing serial laboratory data points among and between patients as independent data points will miss heterogeneous disease processes that may differentially impact these pre-analytical factors (i.e., do hemolytic anemia, bleeding, anemia of inflammation have similar impacts on VHAs?).

Even with these unknowns, we agree with the statement by Meli et al (1) that our findings may not be uniformly identified in all patient populations, especially in trauma patients who are actively losing whole blood volume (concomitant loss of RBCs, plasma, platelets). However, it is worth mentioning that VHA testing in trauma often occurs after empiric, balanced resuscitation begins, whether it be whole blood or 1:1:1 RBC:platelet:plasma transfusion protocols. Thus, these baseline and follow-up VHA results may reflect a more "repleted/stable" platelet/fibrinogen state. Assessments of how hemoglobin relates to VHA tracings, while accounting for fibrinogen/platelet counts (and function), will be informative to identify whether hemoglobin or other factors are relevant preanalytical factors to consider in trauma patients.

Outside of trauma, VHAs are becoming more widely implemented in assessments of coagulopathy for the critically ill. In these scenarios, hemoglobin changes relate to critical illness (or other processes) rather than whole blood volume loss, which may not be matched by proportional losses of fibrinogen or platelets. Yet, the observations by Meli et al (1) suggest that fibrinogen and platelets have a relevant influence on hemoglobin-VHA tracing relationships even without active hemorrhage. As VHAs become more widely implemented in ICUs with heterogeneous patient populations, there is a need to highlight findings such as these to further understand and effectively implement VHAs across patient populations. This may be a call for larger studies and cross-collaborations between clinical and laboratory medicine specialists using these tests to "shed a greater light" on optimal diagnostic approaches for critically ill patients vulnerable to the impacts of coagulopathy.

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