Preface

Editorial Compilation XV

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Semin Thromb Hemost

Welcome to the latest issue of *Seminars in Thrombosis and Hemostasis (STH)* published under the "banner" of "Editorial Compilation," this being the 15th such issue (►**Table 1**). Historically, *STH* is a theme-driven publication; however, ongoing opportunities emerge to disseminate wide-ranging contributions of current interest or controversy, and which do not straightforwardly suit an ongoing themed issue. We also require a medium for enabling publication of accepted peer-reviewed "unsolicited" manuscripts, as well as contributions from our Eberhard F. Mammen Young Investigator Award winners (see ►**Table 2** for previous Editorials related to the Eberhard F. Mammen awards). As is now standard for this compilation series, the current issue contains a mixture of articles that comprise the above elements, as well as broadly fitting within the standard themes of "thrombosis" and "bleeding."

This issue begins with several manuscripts still focused on COVID-19 (coronavirus disease 2019) or its potential aftermath, a condition colloquially called "long-COVID," and otherwise known as "post-acute sequelae of COVID-19 (PASC)."[1–3] Long-COVID represents a heterogeneous clinical syndrome characterized by a pathologic continuum of signs, symptoms, and also laboratory/radiologic abnormalities that may persist for a long time after recovering from an acute SARS-CoV-2 (severe acute respiratory syndrome-coronavirus disease 2) infection. Among the various components of this post-viral condition, the risk of venous thromboembolism (VTE) in patients hospitalized for COVID-19 remains considerably higher after discharge, especially in the earlier period (i.e., within the first 6–12 months), in older individuals, in men, in patients with longer hospital stays and more aggressive clinical management (e.g., mechanical ventilation and/or intensive care), when thromboprophylaxis is not used, and in those with a persistent pro-thrombotic state.[4] As also previously noted, STH had previously published COVID-19-related manuscripts in a series of four issues around the broad theme of "maintaining hemostasis and preventing thrombosis in COVID-19," with the final such issue published in early 2023.[5] As the international emergency for COVID-19 has been withdrawn by the World Health Organization, and we now return to "business as usual," we are no longer publishing COVID-19 themed issues, and instead any COVID-19-related material accepted for publication will now appear in the other compilation themes in progress, such as this Editorial Compilation series.

For the current compilation issue, we start with the contribution from Iba and colleagues.[1] As already noted, unexplained sustained fatigue, cognitive disturbance, and muscle ache/weakness are reported in patients who have recovered from an acute COVID-19 infection. This adverse condition, termed long-COVID or PASC, has a prevalence estimated to be around 10 to 20% of convalescent patients. Although the pathophysiology of PASC has been studied, the

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Table 1 Past STH issues in the series "Editorial Compilation"

1. Favaloro EJ, Lippi G. Editorial Compilation I. Semin Thromb Hemost 2016 Feb;42(1):5–8.
2. Favaloro EJ, Lippi G. Editorial Compilation II. Semin Thromb Hemost 2016 Sep;42(6):599–602.
3. Favaloro EJ, Lippi G. Editorial Compilation III. Semin Thromb Hemost 2017 Feb;43(1):4–7.
4. Favaloro EJ, Lippi G. Editorial Compilation IV. Semin Thromb Hemost 2017 Sep;43(6):549–552.
5. Favaloro EJ, Lippi G. Editorial Compilation V. Semin Thromb Hemost 2018 Apr;44(3):193–196.
6. Favaloro EJ, Lippi G. Editorial Compilation VI. Semin Thromb Hemost 2019 Feb;45(1):5–9.
7. Favaloro EJ, Lippi G. Editorial Compilation VII. Semin Thromb Hemost 2019 Jul;45(5):429–432.
8. Favaloro EJ, Lippi G. Editorial Compilation VIII. Semin Thromb Hemost 2020 Jun;46(4):393–397.
9. Favaloro EJ, Lippi G. Editorial Compilation IX. Semin Thromb Hemost 2021 Feb;47(1):6–10.
10. Favaloro EJ, Lippi G. Editorial Compilation X. Semin Thromb Hemost 2021 Oct 47(7):754–758.
11. Favaloro EJ, Lippi G. Editorial Compilation XI. Semin Thromb Hemost 2022 Mar;48(2):127–131.
12. Favaloro EJ, Pasalic L, Lippi G. Editorial Compilation XII. Semin Thromb Hemost 2022 Jul;48(5):497–501.
13. Favaloro EJ, Pasalic L, Lippi G. Editorial Compilation XIII. Semin Thromb Hemost 2023 Jul;49(5):427–432
14. Favaloro EJ, Pasalic L, Lippi G. Editorial Compilation XIV. Semin Thromb Hemost 2024 Mar; 50(2):151–156.

Table 2 Past STH editorials related to Eberhard F. Mammen award announcements

1. Favaloro EJ. Welcome to a Special Issue of Seminars in Thrombosis and Hemostasis—The Closing Issue for 2008. Semin Thromb Hemost 2008;34:693–696
2. Favaloro EJ. A Tribute to Eberhard F. Mammen, M.D. (1930–2008). Semin Thromb Hemost 2008;34:703–708
3. Favaloro EJ. Welcome to the first issue of Seminars in Thrombosis and Hemostasis for 2009. Semin Thromb Hemost 2009;35:1–2.
4. Favaloro EJ. Winners of the Inaugural Eberhard F. Mammen Award for Most Popular Article. Semin Thromb Hemost 2009;35:587–590
5. Favaloro EJ. Editorial. 2009 Eberhard F. Mammen Young Investigator Award Winners. Semin Thromb Hemost 2010;36:469–470
6. Favaloro EJ. Winners of the 2010 Eberhard F. Mammen Award for Most Popular Article during 2008–2009. Semin Thromb Hemost 2010;36(7):685–92.
7. Favaloro EJ. 2011 Eberhard F. Mammen award announcements. Semin Thromb Hemost 2011;37(5):431–9.
8. Favaloro EJ. 2012 Eberhard F. Mammen award announcements. Semin Thromb Hemost 2012;38:425–32.
9. Favaloro EJ. 2013 Eberhard F. Mammen award announcements. Semin Thromb Hemost 2013;39:567–74.
10. Favaloro EJ. 2014 Eberhard F. Mammen award announcements: Part I - most popular articles. Semin Thromb Hemost 2014;40(4):407–12.
11. Favaloro EJ. 2014 Eberhard F. Mammen Award Announcements: Part II - Young Investigator Awards. Semin Thromb Hemost 2014;40(7):718–23.
12. Favaloro EJ. 2015 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2015;41(7):673–9.
13. Favaloro EJ. 2015 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2015;41(8):809–15.
14. Favaloro EJ. 2016 Eberhard F. Mammen Award Announcements: Part I - Most Popular Articles. Semin Thromb Hemost 2016;42(4):325–30.
15. Favaloro EJ. 2016 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2017;43(3):235–241.
16. Favaloro EJ. 2017 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2017;43(4):357–363.
17. Favaloro EJ. 2017 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2018;44(2):81–88.
18. Favaloro EJ. 2018 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2018;44(3):185–192.

Table 2 (*Continued*)

19. Favaloro EJ. 2018 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2019;45(2):123–129.
20. Favaloro EJ. 2019 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2019;45(3):215–224.
21. Favaloro EJ. 2019 Eberhard F. Mammen Award Announcements: Part II—Young Investigator Awards. Semin Thromb Hemost 2020;46(2):105–113
22. Favaloro EJ. 2020 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2020;46(4):383–392.
23. Favaloro EJ. 2020 Eberhard F. Mammen Award Announcements: Part II—Young Investigator Awards. Semin Thromb Hemost 2021;46(3): 229–237.
24. Favaloro EJ. 2021 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2021 Jul;47(5):467–476.
25. Favaloro EJ. 2021 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2022 Apr;48(3):265–273.
26. Favaloro EJ. 2022 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2022 Jul;48(5):502–513.
27. Favaloro EJ. 2023 Eberhard F. Mammen Award Announcements: Part I-Most Popular Articles. Semin Thromb Hemost 2023 Jul;49(5):417–426.
28. Favaloro EJ. 2022 Eberhard F. Mammen Award Announcements: Part II-Young Investigator Awards. Semin Thromb Hemost 2023 Nov;49(8):775–782

exact mechanism remains obscure. Microclots in circulation can represent one of the possible causes of PASC. Although hypercoagulability and thrombosis are critical mechanisms of acute COVID-19, recent studies have reported that thromboinflammation continues in some patients, even after the virus has apparently cleared from the usual sites of sampling (i.e., oro- and/or naso-pharynx). Accordingly, viral spike proteins and even viral RNA can be detected months after patients have recovered, findings that may be responsible for persistent thromboinflammation and development of microclots. Despite this theory, long-term results of anticoagulation, antiplatelet therapy, and vascular endothelial protection are inconsistent, and could not always show beneficial treatment effects. In summary, PASC reflects a heterogeneous condition, and microclots cannot explain all the presenting symptoms. The authors conclude that after clarification of the pathomechanisms of each symptom, a symptom- or biomarker-based stratified approach should be considered for future studies.

Next, Pretorius and Kell provide an extensive Commentary on the topic of microclots, as potentially seen in both acute COVID-19 and in long-COVID.[2] Microscopy imaging has enabled health workers to appreciate the presence of fibrin(ogen) amyloid ("fibrinaloid") microclots in a range of chronic, inflammatory diseases. Microclots may also be induced by a variety of purified substances, often at very low concentrations. These molecules include bacterial inflammagens, serum amyloid A, and the S1 fraction of the spike protein of SARS-CoV-2. Here, the authors explore which of the properties of these microclots might be used to contribute to differential clinical diagnoses and prognoses of the various diseases with which they may be associated. Such properties include distributions in their size and number before and after the addition of exogenous thrombin, their spectral properties, the diameter of the fibers of which they are made, their resistance to proteolysis by various proteases, their cross-seeding ability, and the concentration-dependence of their ability to bind small molecules including fluorogenic amyloid stains. The authors believe that measuring these microclot parameters, together with microscopy imaging itself, along with methodologies such as proteomics and imaging flow cytometry, as well as more conventional assays, including those for cytokines, might open up the possibility of more focused use of these microclot properties in generative methods for a future where personalized medicine will be a standard procedure in all clotting pathology disease diagnoses.

This issue of STH continues with a systematic review from Dorgalaleh et al, on congenital bleeding disorders (CBDs) and COVID-19.[3] As already noted, hypercoagulability is a prominent feature of COVID-19 and can lead to fatal consequences. Although the impact of COVID-19 on several disorders is well-established, its effect on CBDs is not well-documented. To address this ambiguity, the authors conducted a systematic review on the available studies to determine the impact of COVID-19, and vaccination aimed to prevent COVID-19, on patients with CBDs. Of 31 included studies, 12 case series including 770 patients with CBD and COVID-19 were further analyzed. The majority of patients had hemophilia A ($n = 352$, ~46%) or hemophilia B ($n = 74$, ~10%), while the remaining had von Willebrand disease ($n = 43$, 5.6%) or rare bleeding disorders ($n = 15$, ~2%). A total of 25 deaths (3.2%) and 22 intensive care unit admissions (2.8%) were recorded. Bleeding complications were reported in the majority of the 12-case series ($n = 7$, 58.3%) and also in most of the identified case reports ($n = 8$, ~57%), while thrombotic complications

were only reported in two studies (16.6%). The mortality rate ranged from 0% in five studies to 5.7%, and the rate of hospitalization ranged from 0 to 40%. Bleeding complications were reported in a range of 0 to 81%, while the thrombotic complications rate in one study was 6.9%. Depending on the study, the hospitalization rate ranged from 0 to 40%. Vaccination was reported in five case series, which included 821 patients with CBDs with the majority having hemophilia A ($n = 479$; 67.2%) and hemophilia B ($n = 85$; ~12%). The most frequently reported side effects were myalgia (6.5%), flu-like symptoms (4.8%), fever (4.7%), and headache (4%), similar to those seen in the general population. The authors conclude that COVID-19 in patients with CBDs appears to provoke thrombotic complications and frequent bleeding events, as well as a higher rate of hospitalization, which may be partially due to the increased risk of bleeding events. Although it seems that patients with CBD have lower mortality rates, further studies are necessary to fully understand this aspect, especially considering comorbidities and low number of available studies.

Next, we turn our attention away from COVID-19, and return to general topics related to thrombosis and hemostasis. First, Gelbenegger et al provide a review on disseminated intravascular coagulation (DIC) in anaphylaxis.[6] Anaphylaxis is a life-threatening condition that involves severe cutaneous, respiratory, and cardiovascular symptoms, while DIC is an acquired, widespread activation of coagulation that can be caused by infectious (e.g., sepsis) and noninfectious conditions. The onset of DIC following anaphylaxis is not commonly known and information regarding the pathomechanism linking anaphylaxis to DIC is scarce. Further, demographic and clinical data in anaphylaxis-induced DIC are currently missing. Triggered by a case of anaphylaxis-induced DIC that seamlessly transitioned to lethal sepsis-induced DIC, the authors aimed to characterize the patient population affected by anaphylaxis-induced DIC by performing a review of existing literature. Overall mortality of the identified patient cohort ($n = 30$) was considerably high (i.e., 50%). All patients that died either suffered a bleeding or a thrombotic event. The majority of patients ($n = 25/30$; 83%) had bleeding events; thrombotic events were only reported in nonsurvivors ($n = 9/15$ or 60% of nonsurvivors; vs. $n = 0/15$ in survivors; $p < 0.001$). Nonsurvivors of anaphylaxis-induced DIC were on average 25 years older than survivors ($p = 0.068$). In conclusion, the authors comment that DIC can complicate anaphylaxis, and is expected to contribute to poor microvascular perfusion after anaphylaxis. Particularly, elderly patients with a known cardiovascular disease and patients who develop thrombotic events are susceptible to lethal outcome. As a rare and largely uncharacterized disease entity, further research is needed to investigate the link between DIC and anaphylaxis and to potentially identify better treatment strategies.

Gyawali et al then review whole blood viscosity and cerebral blood flow in acute ischemic stroke.[7] Existing effective treatments for ischemic stroke restore blood supply to the ischemic region using thrombolysis or mechanical removal of clot. However, it is increasingly recognized that successful removal of occlusive thrombus from the large artery—recanalization—may not always be accompanied by successful restoration of blood flow to the downstream tissues—reperfusion. Ultimately, brain tissue survival depends on cerebral perfusion, and a functioning microcirculation. Because the capillary diameter is often equal to or smaller than an erythrocyte, microcirculation is largely dependent on erythrocyte rheological (hemorheological) factors such as whole blood viscosity. Several studies in the past have demonstrated elevated whole blood viscosity in stroke compared with healthy controls. Also, elevated whole blood viscosity has shown to be an independent risk factor for stroke. Elevated whole blood viscosity leads to endothelial dysfunction, decreases nitric oxide-dependent flow-mediated vasodilation, and promotes hemostatic alterations/thrombosis, all leading to microcirculation sludging. Compromised microcirculation further leads to decreased cerebral perfusion. Hence, modulating whole blood viscosity through pharmacological agents might be beneficial to improve cerebral perfusion in stroke. This review discusses the effect of elevated whole blood viscosity on endothelial function, hemostatic alterations, and thrombosis leading to reduced cerebral perfusion in stroke.

A review on lupus anticoagulant-hypoprothrombinemia syndrome (LAHPS) follows from Wang et al.[8] LAHPS is a rare, acquired coagulopathy syndrome. Here, the authors aim to summarize the clinical features of LAHPS to improve the understanding of the disease. Stimulated by a local case of a child with LAHPS, the clinical data of 52 patients with LAHPS were retrieved through PubMed from 2019 to 2023. These cases were analyzed and their clinical characteristics summarized. Overall, 56.6% of LAHPS patients were female, the median age at onset was 13.0 years (range 1.2–85 years), and the median activity of factor II was 18% (range 0.1–69%). A total percentage of 64.2% of LAHPS patients experienced hemorrhage, with 29.4% having multi-site hemorrhage, and 20.6% experiencing both nonsevere and severe hemorrhage. Most of the reported cases were secondary to autoimmune diseases (60.6%), followed by infections (33.3%). Corticosteroids were administered to 79.3% of patients with hemorrhage, and 90.6% of patients with LAHPS showed improvement. The authors conclude that LAHPS is most commonly observed in female patients, particularly those under 18 years of age. Furthermore, LAHPS is characterized by hemorrhage, occurring at various sites and with varying degrees of severity, but the majority of patients improve with appropriate treatment and management.

Next is a Commentary from the Guest Editors,[9] regarding the newly published classification criteria for the antiphospholipid syndrome (APS),[10] further identifying that these are not the same as diagnostic criteria for APS. The aim of the latest consensus "guidelines" outlining revised classification criteria for APS was to develop new APS classification criteria with high specificity to be used in observational studies and trials, and was jointly supported by the American College of Rheumatology (ACR) and the European Alliance of Associations for Rheumatology (EULAR). Prior classification of APS, for identification of homogeneous research cohorts, was based on the Sapporo criteria published in 1999,[11] as revised in Sydney in 2006.[12] The 2023 ACR/EULAR APS classification

criteria include an entry criterion of at least one positive antiphospholipid (aPL) test within 3 years of identification of an aPL-associated clinical criterion, followed by additive weighted criteria (score range 1–7 points each) clustered into six clinical domains (macrovascular VTE, macrovascular arterial thrombosis, microvascular, obstetric, cardiac valve, and hematologic) and two laboratory domains (lupus anticoagulant functional coagulation assays, and solid-phase enzyme-linked immunosorbent assays (ELISA) for IgG/IgM aCL and/or aβ2GPI).[10] Patients accumulating at least three points each from the clinical and laboratory domains are *classified* as having APS. The distinction with "diagnostic criteria" is that clinicians are able to "diagnose" APS using the "classification" criteria (both clinical and laboratory), but that diagnosis of APS is not *restricted* to the items present in the "classification criteria." In other words, the "classification" criteria establish a finite list of clinical and laboratory parameters that can be used to identify some "definite" APS manifestation for inclusion in future studies, but a broader list of both clinical and laboratory criteria is available to help diagnose APS.

An exploration into the role of platelets in rheumatic chronic autoimmune inflammatory diseases follows, from the team of Péč et al.[13] As our readership well knows, platelets are essential in maintaining blood homeostasis and they also regulate several inflammatory processes. Platelets constantly interact with immune cells, have immunoregulatory functions, and can affect, through immunologically active substances, endothelium, leukocytes, and other immune response components. On the other side of the coin, inflammatory and immune processes can activate platelets, which might be significant in autoimmune disease progression and arising complications. Thus, considering this interplay, targeting platelet activity may represent a new approach to treatment of autoimmune diseases. This review aims to highlight the role of platelets in the pathogenic mechanisms of the most frequent chronic autoimmune inflammatory diseases, to identify gaps in current knowledge and provide potential new targets for medical interventions.

Next, Sokou et al[14] provide a systematic review of bleeding scoring systems in neonates. The review aims to summarize data on current hemorrhage prediction models and evaluate their potential for generalized application in the neonatal population. The authors searched the electronic databases of PubMed and Scopus, up to September 20, 2023, for studies that focused on development and/or validation of a prediction model for bleeding risk in neonates, and described the process of model building. Nineteen studies fulfilled the inclusion criteria, and 18 bleeding risk prediction models in the neonatal population were identified, 4 of which were internally validated, 1 temporally and 1 externally validated. The existing prediction models for neonatal hemorrhage are mostly based on clinical variables and do not consider the clinical course and hemostatic profile of the neonates. Most studies aimed at predicting the risk of intraventricular hemorrhage (IVH) reflecting the fact that IVH is the most frequent and serious bleeding complication in preterm neonates. A justification for the study sample size for developing the prediction model was given only by one study. Prediction and stratification of risk of hemorrhage in neonates is yet to be optimized. To this end, the authors indicate that qualitative standards for model development need to be further improved, and that assessment of the risk of bleeding, incorporating platelet count, coagulation parameters, and a set of relevant clinical variables is crucial. Large, rigorous, collaborative cohort studies are also warranted to develop a robust prediction model to inform the need for transfusion, which is a fundamental step toward personalized transfusion therapy in neonates.

The last review in this issue of STH is by Capece et al,[15] as led by a previous 2022 Young Investigator Awardee (Lauren G. Poole).[16] Acute liver injury (ALI) represents the development of reduced liver function in patients without pre-existing liver disease, and can result from a wide range of causes, such as viral or bacterial infection, autoimmune disease, or adverse reaction to prescription and over-the-counter medications. ALI patients present with a complex coagulopathy, characterized by both hypercoagulable and hypocoagulable features. Similarly, ALI patients display a profound dysregulation of the fibrinolytic system, with the vast majority of patients presenting with a hypofibrinolytic phenotype. Decades of research in experimental ALI in mice suggests that fibrinolytic proteins, including plasmin(ogen), plasminogen activators, fibrinolysis inhibitors, and fibrin(ogen) can contribute to initial hepatotoxicity and/or stimulate liver repair. This review summarizes major experimental findings regarding the role of fibrinolytic factors in ALI from the last approximately 30 years and identifies unanswered questions, as well as highlighting areas for future research.

As usual for these nonthematic issues of STH, the issue continues with some correspondence. First, Franchini et al discuss recommendations on the use of COVID-19 convalescent plasma to treat immunocompromised patients.[17] Next, Chen and colleagues describe a case of parturient with hereditary thrombotic thrombocytopenic purpura and a novel variant in the gene for ADAMTS13 (a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13).[18] We finalize the standard issue format where we began, with discussion of COVID-19—with Fan at al describing a case of post-COVID-19 large vessel vasculopathy in a previously healthy young male.[19]

And yet, the content described above does not end this issue of STH. As we are celebrating 50 years of publishing, each issue of 2024 will also republish a key paper from the past, and also contain invited Commentaries discussing the historical paper in the context of what we know today. In the first issue of 2024, STH republished the first paper that STH ever published, on the molecular structure of fibrinogen,[20] with an accompanying Commentary from Marguerite Neerman-Arbez and Alessandro Casini.[21] In the second issue of STH, we republished a manuscript that provided the first ever description of an instrument called the Platelet Function Analyzer (PFA)-100,[22,23] with an accompanying Commentary from the Current Editor in Chief of STH.[24] Then in the third issue of STH for 2024, we republished a historical review on the skin bleeding time,[25,26] with an accompanying Commentary from Anetta Undas.[27]

In the current issue of STH, we are republishing one of the most highly cited papers from the Founding Editor in Chief of STH, Eberhard F. Mammen, and colleagues,[28,29] and an accompanying Commentary from one of the original co-authors, Robert C. Gosselin.[30] The original republished paper was the fourth most highly cited manuscript from STH of all time according to Web of Science,[31] and this republication completes the circle around the PFA-100, starting from the original description,[22–24] in part reflecting that the instrument and the associated test output, the closure time, reflected a kind of in vitro replacement to the more invasive skin bleeding time.[25–27] This also reflects on the historical elements around the activities of the Founding Editor in Chief of STH, and those of his successor.[24,31]

We once again thank all the authors to this latest issue of "Editorial Compilations" for their original and comprehensive contributions, and we hope our readership enjoys this latest instalment in this series.

Conflict of Interest
None declared.

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