A defect in any step of hemostasis can lead to potentially catastrophic results. The purpose of this article is to review hemostatic physiology, laboratory studies, and management of platelet and coagulation disorders to familiarize the advanced practice RN (APRN) with this often overlooked but critical system. Learning the underlying mechanisms allows for better understanding of the various disease states that can occur in the hematology and oncology settings.

AT A GLANCE

- A single alteration in hemostasis can lead to excess bleeding or thrombosis.
- Laboratory studies, such as screening assays, can be performed to detect coagulopathy, and additional studies may be needed for diagnosis.
- An understanding of the underlying mechanisms of hemostasis is essential for APRNs managing patients with platelet and coagulation disorders.

KEYWORDS
hemostasis; coagulopathy; critical care; hematology; advanced practice

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Hemostatic Disorders

Physiology, diagnostics, and management

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Hemostasis is an intricate system within the vasculature that can be difficult to appreciate until dysfunction occurs. As a response to tissue injury, a cascade of cellular and molecular events occurs to achieve hemostasis. A defect in any of the steps of hemostasis can lead to an impaired response with potentially catastrophic results. This article aims to familiarize oncology advanced practice RNs (APRNs) with the physiology, laboratory studies, and management of platelet and coagulation disorders that disrupt the hemostatic system.

Physiology of Hemostasis

The principal components of hemostasis are the vascular endothelium, platelets, coagulation, and fibrinolysis. The vascular endothelium consists of a monolayer of cells separating blood from the contents of the subendothelium. The vascular endothelium actively prevents unnecessary thrombosis via platelet inhibition, regulation of coagulation factor activity, promotion of fibrinolysis (clot breakdown), and alteration of vascular tone and permeability (Fredenburg & Weitz, 2018). Platelets prevent bleeding after vascular endothelial injury by first adhering to the exposed areas of the endothelium and the subendothelium and then secreting substances that cause subsequent aggregation, clot factor activation, and vasoconstriction. Platelets also change shape, serving as a surface for clotting factor assembly (Fredenburg & Weitz, 2018).

Coagulation and fibrinolysis are opposing systems that are carefully balanced in normal hemostasis (see Figure 1). Coagulation results in thrombin (factor II) generation, which converts fibrinogen to fibrin, the protein needed to create the mesh for a stable clot. There are three enzyme complexes critical to thrombin generation: extrinsic tenase (tissue factor/factor VIIa), intrinsic tenase (factor VIIIa/IXa), and prothrombinase (factor Va/Xa). Initial thrombin generation is mainly mediated by extrinsic tenase. Ongoing thrombin generation is necessary for sustained coagulation and is mediated primarily by intrinsic tenase. Subsequent molecular effects of the extrinsic and intrinsic tenase complexes lead to the formation of the prothrombinase complex. This complex (factor Va/Xa) is essential because it is the only physiologic producer of thrombin. Thrombin is responsible for the cleavage of fibrinogen to fibrin, the protein mesh needed for a stable clot. Termination of coagulation is facilitated by enzymes to ensure that thrombin generation is finite and localized (Fredenburg & Weitz, 2018).

Elimination occurs through the fibrinolytic system and allows for the process of tissue repair. Plasmin is needed for fibrin breakdown and is generated by the enzymes tissue plasminogen activator and urokinase plasminogen activator. Fibrinolysis must also be regulated tightly by enzymes (Brummel-Ziedins & Mann, 2018).

Laboratory Studies

Two common screening assays for the detection of coagulopathy are the activated