Developmental hemostasis: consequences for clinical practice

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Hemostasis is a complex process leading to the formation of a blood clot at the site of vessel injury while simultaneously preventing excessive clotting. In the 1980's Maureen Andrew was the first to introduce the term "developmental hemostasis" to describe the maturation of the hemostasis system from fetal to adult system. Significant differences exist in the physiology of primary hemostasis as well as secondary hemostasis and fibrinolysis in particularly fetal and neonatal life compared to adult life. The reasons for these differences are unclear and may be related to factors not associated with blood coagulation. In healthy neonates these differences are functionally balanced, as they do not cause an increased tendency to bleeding or thrombosis. However, developmental hemostasis may have consequences for both the diagnosis and management of hemorrhagic or thrombotic events, especially in neonates.
Diagnosis of hemostatic disorders in neonates may be difficult as result of age-related changes in the coagulation system. Therefore, age-, analyzer- and reagent-specific reference ranges should always be used to avoid overdiagnosis and misdiagnosis.

Developmental hemostasis may be of importance in management of neonatal hemostatic disorders, for example in neonatal transfusion medicine. Regardless of reduced platelet function, bleeding time is shorter in neonates, probably as result of increased levels of von Willebrand Factor and high hematocrit. As in vitro studies revealed shorter closure times in neonatal blood transfused with adult platelets, adult platelets transfusion may increase the thrombosis risk, especially in preterm neonates. Furthermore, developmental hemostasis may influence the management of anticoagulation therapy in young children with thrombosis. Antithrombin levels are decreased in neonates, which can effect heparin treatment. Neonates need higher dosages per kilogram of all types of anticoagulants than adults. Therapeutic levels of anti-Xa are difficult to obtain. Despite the delay in reaching therapeutic anti-FXa levels, thrombus resolution or no thrombus progression does occur. Age-specific guidelines for management of neonatal hemostasis is, therefore, warranted.