Introduction

Exsanguination is still a major cause of death in severely injured patients [1]. Trauma-associated bleeding diathesis, overt at admission to the trauma unit, correlates with the severity of trauma and mortality [2, 3]. Sufficient hemostatic management is critical to the successful resuscitation of the severely injured patient, second in importance only to adequate ventilation. Despite intense efforts to elucidate the pathomechanism and control the process, trauma-associated coagulopathy remains a challenge in the treatment of trauma patients. In this light, monitoring of hemostasis should confirm and specify the clinical diagnosis of bleeding diathesis, guide goal-directed therapy, and possibly predict consecutive transfusion requirements at admission. The present chapter reviews routine laboratory tests and viscoelastic point-of-care hemostasis monitoring as a means of hemostasis monitoring in the emergency setting, as well as relevant pathomechanisms, and therapeutic approaches.

Pathomechanisms of Trauma-associated Coagulopathy

In massively bleeding patients, coagulopathy is complex in origin, correlating with the severity of injury [3, 4]. Figure 1 summarizes the multiple pathomechanisms leading to trauma-associated coagulopathy. Uncontrolled bleeding initially leads to
loss of coagulation factors and platelets [5, 6]. Trauma-induced exposure of the thromboplastin-rich subendothelial tissue to flowing blood induces the activation of coagulation [7] which may trigger consumptive coagulopathy [8–10]. The majority of blunt trauma and brain injury victims are hypercoagulable early after trauma with tissue trauma being the key stimulus for coagulation [8, 11, 12]. In hypocoagulable patients, however, the remaining procoagulatory potential is reduced by dilution during fluid resuscitation required to restore intravascular volume and to maintain hemodynamic stability. The degree of dilutional coagulopathy also depends on the type of fluid used: Hydroxyethyl starch solutions, gelatins, and dextrans impair platelet function, inhibit fibrin polymerization, and induce an acquired von Willebrand syndrome to varying degrees, depending on the physicochemical characteristics of the colloidal solution [13]. Resuscitation with hypertonic saline appears to aggravate bleeding by its potent anticoagulatory and anti-platelet effects [14], while other hypertonic solutions (glycine, glucose, sorbitol) exhibit a significantly reduced impairment. Additionally, massive transfusion inevitably leads to coagulopathy, although dilution often is not an issue until more than one blood volume (10–12 units packed red blood cells [RBCs]) is given. Tissue injury in trauma may lead to the exposure of tissue plasminogen activator resulting in hyperfibrinolysis if the delicate balance between coagulation and fibrinolysis is lost [15]. Coagulopathy is confounded by hypothermia, acidosis, and pre-existing disorders: Trauma patients are prone to hypothermia, which slows down enzymatic reactions [16], modifies platelet function [17], and stimulates fibrinolysis. Acidosis worsens fibrin polymerization and strengthening of the clot [18]. In a study including both blunt and penetrating injuries, the vicious cycle induced by severity of tissue injury (Injury Severity Score [ISS] > 25), progressive core hypothermia (<34°C), and ongoing cellular shock (pH < 7.0 and low arterial blood pressure) predicted life-threatening coagulopathy in massively transfused patients [10]. Since trauma is not restricted to previously healthy people, the increasing number of patients taking oral anticoagulants and platelet-inhibiting drugs poses a rapidly increasing problem [19]. Patients with inherited coagulation defects may exsanguinate with trauma unless specific factor replacement is provided. Low hematocrit (<30%) and low ionized calcium (after massive packed RBC transfusions containing citrate) further aggravate bleeding diathesis. This vicious cycle of trauma-associated coagulopathy results in: 1) a defect in clot firmness due to fibrinogen deficiency and thrombocytopenia, 2) impaired clot stability due to hyperfibrinolysis and factor XIII deficiency (being a late phenomenon), and 3) prolonged clot generation due to various coagulation factor deficiencies. The coagulopathy causes uncontrolled bleeding requiring massive transfusion, which is commonly defined as the replacement of one blood volume over a period of 24 h or transfusion of at least 4 units of packed RBC within 1 h.

**Diagnosis of Trauma-associated Coagulopathy**

Due to the complex nature of hemorrhage in emergency medicine, physicians require coagulation monitoring strategies sensitive to all these major possible pathomechanisms. Initially, a bleeding history should be assessed from the patient if conscious or from accompanying relatives. The clinical examination in trauma-associated coagulopathy may reveal bleeding from mucosal lesions and serosal surfaces, as well as prolonged bleeding at catheter insertion sites and wounds in the absence of a surgically correctable bleeding site. Finally, coagulation tests are required to verify