Hemorrhage and Coagulopathy in the Critically Ill

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KEYWORDS

- Hemorrhage • Coagulopathy • Transfusion • Resuscitation
- Massive transfusion protocol

KEY POINTS

- Hemorrhage and coagulopathy in the critically ill, if not intervened upon early, can precipitate a vicious cycle of hypothermia and acidosis that worsens coagulopathy and bleeding.
- Transfusion medicine has come a long way since its origin in 1665, but still has a long way to go.
- Coagulopathy may be induced by trauma, acute blood loss, medications, resuscitation with blood products or crystalloid devoid of coagulation factors, or hypothermia.
- Recent oral anticoagulants complicate coagulopathy and present a new dilemma for treatment, not responding to traditional reversal agents.

INTRODUCTION

The first successful blood transfusion was performed by physician Richard Lower on dogs in 1665 and the first accounts of mass casualties and lifesaving blood transfusions was during World War I.[1] Military transfusion practice continues to influence civilian protocols in emergency and trauma medicine. This is so vital because trauma is a major health issue worldwide and is responsible for more than 5 million deaths annually, projected to be more than 8 million by 2020.[2] Uncontrolled hemorrhage is responsible for 40% of all deaths in trauma. The development of coagulopathy in the setting of hemorrhage occurs frequently and confounds our ability to restore normal

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physiology. In an emergency setting, coagulopathy may be a direct result of trauma, ongoing bleeding, traumatic brain injury, upper or lower gastric hemorrhage, cirrhosis, medication effects, or multifactorial. Consumption of clotting factors, hemodilution, acidosis, hypoperfusion, and hypothermia are causes of hypocoagulability that occur within the first 24 hours of admission. One third of patients presenting to the emergency department are coagulopathic, which in turn increases mortality and morbidity.[3]

THE COAGULATION CASCADE

To discuss hemorrhage and coagulopathy, the coagulation cascade must be reviewed (Fig. 1). The endothelium normally promotes blood fluidity unless there is an intimal injury. Coagulation is promoted at the site of injury and the response is contained by a series of procoagulating and anticoagulating interactions. Tissue factor is expressed on the surface of injured adventitial vascular walls, after activation by local cytokines. Tissue factor binds to activated factor VII (FVIIa) then activates FX; FX and is the major activator of the extrinsic pathway. The intrinsic pathway is a series of proteolytic reactions, which culminate to activate FIX. The intrinsic and extrinsic pathways converge at the level of FX, the common pathway (see Fig. 1).

MONITORING OF COAGULATION

Clotting tests routinely performed in the emergency setting include partial thromboplastin time (PTT), prothrombin time (PT), International Normalized Ratio (INR). Fibrinogen, fibrin split products, and D-dimer are other measured factors that can alter bleeding. PTT is an indicator of the efficacy of both the intrinsic and the common pathway, and is used to monitor therapeutic levels of heparin. PT is a measure of the extrinsic pathway. The INR is a ratio of the PT and the normal mean PT, and

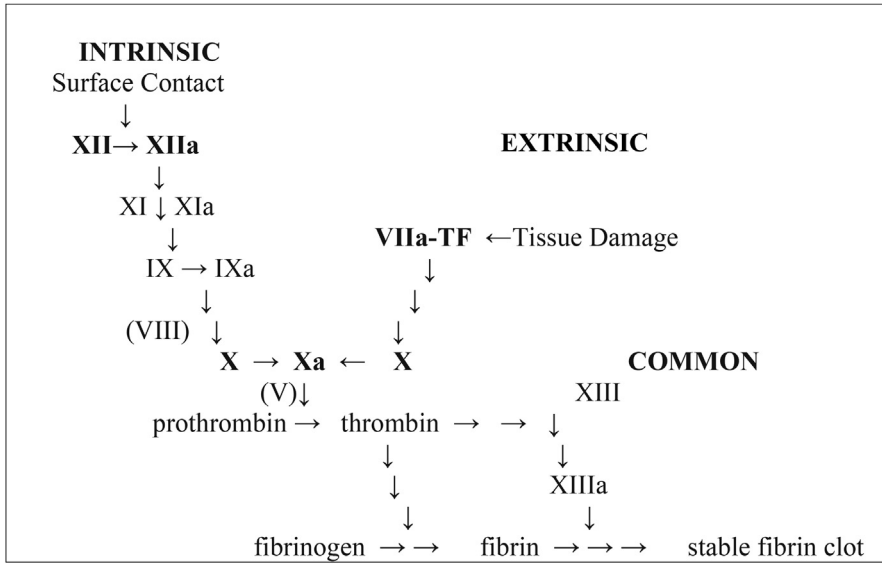

Fig. 1. The coagulation cascade. (*Adapted from* The Classical Blood Coagulation Pathway by Dr Graham Beards under the Creative Commons Attribution-Share Alike 3.0 Unported license.)