Trauma-induced coagulopathy

Understand the changing clotting phenotype.

RESUSCITATION / ADULT TRAUMA

Coagulopathy can begin early after tissue injury and shock, before dilution or hypothermia fully develops.

01 Think beyond dilution

Tissue injury and shock disturb endothelial function, platelets, coagulation and fibrinolysis. Hypothermia, acidosis and resuscitation-related dilution can compound this process. Several mechanisms may coexist in one patient.

02 Describe the phenotype

Early bleeding and later thrombosis are different expressions of a dynamic disorder. A normal platelet count does not establish normal platelet function; an isolated clotting time does not describe every mechanism.

03 Connect tests to bleeding

Interpret serial laboratory or viscoelastic results alongside clinical haemorrhage and treatment already given. Identify specific deficits and pre-injury anticoagulant exposure. Use the local haemostatic algorithm rather than treating an isolated number.

04 Reassess after control

Revisit the coagulation picture after haemostasis and resuscitation. The balance between bleeding and thrombosis changes. Communicate ongoing deficits and the plan to review thromboprophylaxis with the receiving team.

WATCH POINT Haemostatic treatment supports source control; it cannot compensate for an uncontrolled anatomical bleeding site.

DISCUSS WITH THE TEAM

Which part of the current clotting abnormality is measured, and which remains uncertain?

READ THE SOURCES

[1] Moore et al. Trauma-induced coagulopathy (2021; corrected 2022), pathophysiology.
[2] European bleeding guideline, 6th ed. (2023), recommendations 2, 9, 13, 23-29, 31.

Educational summary for clinicians. Apply local protocols and patient-specific judgement.