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Influence of fluid therapy on the haemostatic system of intensive care patients

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Haemostatic alterations associated with the use of fluids are related to non-specific dilutional effects and colloid-specific effects, such as acquired von Willebrand syndrome, inhibition of platelet function and fibrin polymerization. Judging by currently available evidence, dextran, hetastarch and pentastarch have a more pronounced impact than tetrastarch, gelatin and albumin. In patients with hypocoagulability, tetrastarch appears to be a suitable volume expander due to its high safety index and volume efficacy. Gelatins have lower inhibitory effects on clot strength compared with tetrastarch, but their volume efficacy is also lower. Dextrans are potent anticoagulants with a high risk for adverse reactions. Albumin has negligible effects on haemostasis, but low volume efficacy and costs limit the use of a blood product as a routine volume replacement fluid. To avoid potential acidosis-induced changes in haemostasis, plasma-adapted carrier solutions may be used instead of saline-based solutions.

Intensive care patients may have various coagulopathies depending on the underlying disease. This article will focus on critically ill patients with the need for fluid therapy and volume expansion, such as in sepsis, trauma and postoperatively after major surgery. In this group of patients, crystalloid and colloid fluids are required either for acute restoration and stabilization of the haemodynamic condition in situations of relative or absolute fluid deficits, or for long-term maintenance of intravascular volume and improved tissue perfusion. Among the colloids, three synthetic colloidal solutions [hydroxyethyl

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starch (HES), gelatin and dextran] and one endogenous colloid (albumin) exist. Chemistry of the fluids is described elsewhere in this issue. Understanding of direction and magnitude of fluid-dependent influences on the haemostatic system is necessary for decision making in fluid management.

Targets for anticoagulant side-effects

Haemostatic alterations are related to non-specific dilutional effects (induced by all fluids) and specific effects of colloidal macromolecules. Specific effects of synthetic colloids have been reviewed elsewhere[1–3] based on the useful evidence derived from extensive in-vitro trials, animal studies and clinical studies in many types of major surgery. In this chapter, this information is updated and extrapolated to critical illness in the absence of firm evidence in this group of patients. Targets for anticoagulant effects are summarized in Fig. 1.

Primary haemostasis: decrease in platelet function

Physicochemical differences were found to be important for the platelet-inhibiting properties of particular HES preparations. Slowly degradable HES solutions has been found to prolong closure times in the Platelet Function Analyzer (PFA)-100[4,5], while rapidely degradable HES had minimal specific effects, if any, in healthy volunteers.[5] Rapidly degradable HES did not decrease platelet aggregation[6], while slowly degradable HES impaired platelet aggregation.[7] Slowly degradable HES induces cellular abnormalities with decreased agonist-induced expression and activation of platelet surface glycoprotein (GP) IIb-IIIa.[4,5] Slowly degradable HES molecules do not appear to exert their inhibitory effect

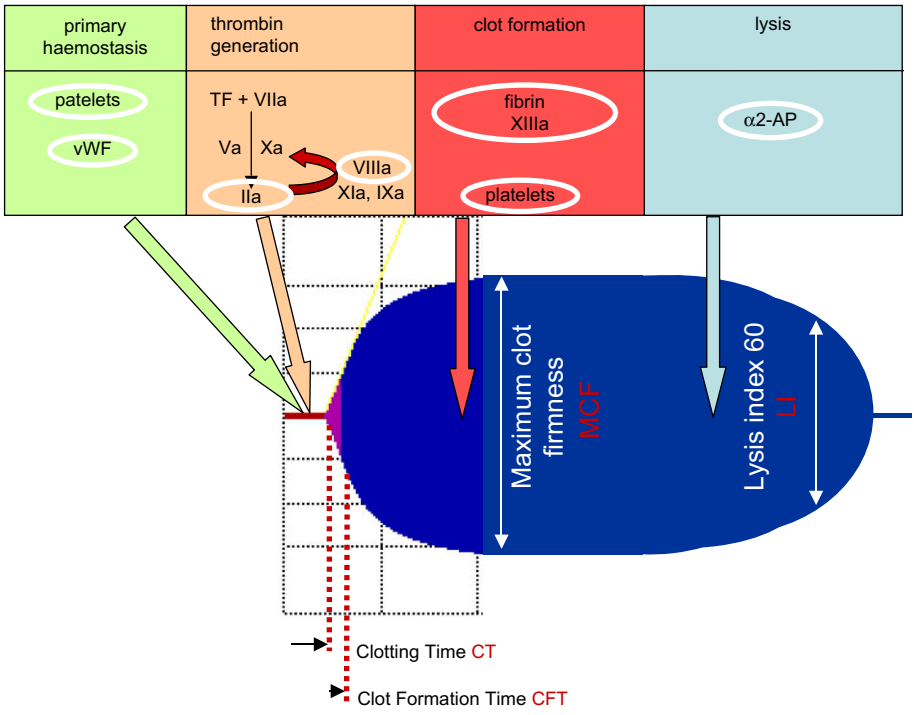

Fig. 1. Thrombelastometric trace and parameters. Clotting time (CT in s), clot formation time (CFT in s), maximum clot firmness (MCF in mm) and lysis index at 60 min (LI in %). White circles indicate targets for anticoagulant effects of synthetic colloids (dextran > slowly degradable hydroxyethyl starch > rapidly degradable hydroxyethyl starch = gelatin). vWF, von Willebrand factor; TF, tissue factor; t-PA, tissue plasminogen activator; α2-AP, alpha 2-antiplasmin.