patients, such as treatment of perioperative anaemia or perioperative autologous blood salvage.\textsuperscript{10}

\textbf{Declaration of interest}

None declared.

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\textbf{Antifibrinolytics in subarachnoid haemorrhage}

Editor—Dr Ortmann and colleagues\textsuperscript{1} comprehensively review the evidence for antifibrinolytics across a spectrum of anaesthetic specialities. Concluding the section on subarachnoid haemorrhage (SAH), they state ‘there is a place for antifibrinolytic therapy as prophylaxis for early re-bleeding in subarachnoid haemorrhage’. We contend that the situation is more controversial than Ortmann and colleagues suggest.

Recent systematic reviews on antifibrinolytics in SAH are equivocal. A recent meta-analysis found evidence of improved functional outcome with short-term use (i.e. \textless 72 h post-ictus);\textsuperscript{2} but, the most recent Cochrane review found a reduction in re-bleeding was not accompanied by improved mortality or morbidity.\textsuperscript{3} Recent European guidelines reflect the equivocal evidence base, upholding the decade-old consensus that the benefit of antifibrinolytics in aneurysmal SAH is outweighed by the increased risk of delayed cerebral ischaemia.\textsuperscript{4} There have been false dawns in SAH management before—notably with supplementary magnesium, which ultimately ended with MASH-2\textsuperscript{5}—and enthusiasm for antifibrinolytics in major trauma has been tempered by post hoc analysis showing that administration more than 3 h post-injury is associated with excess mortality.\textsuperscript{6} This, combined with the increased risk of cerebral ischaemia\textsuperscript{5} and thromboembolism\textsuperscript{7} associated with antifibrinolytics in SAH, suggests that further research is needed to clarify their exact role in managing SAH patients, particularly with relation to timing.

Furthermore, the definitive prevention of re-bleeding is aneurysmal coiling or clipping (with an aneurysmal re-bleeding rate under 1\% at 6 yr post-intervention).\textsuperscript{8} Any benefit from antifibrinolytics in SAH is likely to be as a temporary measure until the offending aneurysm is secured. Quality improvement is better coming from greater availability of interventional radiology and prompt return from a drug that is far from risk-free.

\textbf{Declaration of interest}

None declared.

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Antifibrinolitics and current anaesthetic

Editor—We read with interest the article by Ortmann and colleagues.1 One aim of the highly informative and well-written review article was to enhance further discussion regarding the use of antifibrinolytic agents.

One important area not mentioned in the article on the use of antifibrinolytic agents is the effective role these agents have in major oncological surgeries. During the resection of retroperitoneal sarcomas, for example, massive intra- and postoperative blood loss is not uncommon. Such patients often possess multiple risk factors for haemorrhage; these include tumour factors modulating the fibrinolytic process, the effects of chemotherapeutic agents, and the presence of anticoagulant drugs.2 These together with the complexity of surgical resection undertaken and vascularity of the tumour present a clinical scenario where antifibrinolytics can be effectively used. It is our view that antifibrinolytic agents are an important intervention not to be overlooked.

Although antifibrinolitics have very little inherent prothrombotic property, we would be grateful if the authors could share their experience on the use of antifibrinolitics in patients who have an indwelling inferior venacaval filter.

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Use of antifibrinolitics in liver transplantation

Editor—We read with interest Dr Ortmann and colleagues’ review on the role of antifibrinolitics.3 With regard to liver transplantation, it was highlighted that more than three-quarters of patients undergoing this procedure develop perioperative hyperfibrinolysis, and prophylactic antifibrinolytic agents should be considered.1 This quoted incidence of fibrinolysis was from a prospective observational study of 23 patients in Bordeaux based on a reduction in euglobulin lysis time (ELT) >50% in 18 patients (78.3%).2 Only two patients (8.7%) had a ROTEM demonstrating a typical hyperfibrinolysis trace. The use of antifibrinolitics in instances of bleeding was not emphasized.

Perioperative bleeding in liver transplantation is often multifactorial. Point-of-care coagulation testing allows early detection of hyperfibrinolysis in the bleeding patient and informs clinicians about the individual patient’s requirement for antifibrinolytic drugs.3 These agents may increase the risks of vascular occlusion of the graft and other thrombotic events.4 While this was not demonstrated in a meta-analysis published in 2007, its limitations were numerous and the authors clearly state a large prospective randomized trial with thromboembolic events as the primary endpoint would be preferable to determine actual risk.5

In our unit, we have just completed a review of ROTEM data for 181 consecutive liver transplant procedures and, in a similar fashion to Roulet and colleagues in Bordeaux, assessed coagulation at five fixed time-points during surgery. Our data showed that 12 of 181 patients (6.7%) developed fibrinolysis during liver transplantation. All of these cases were treated successfully with tranexamic acid. We therefore suggest that routine administration of antifibrinolitics is not indicated, given its low incidence and success of treatment when identified.

In view of the potential for thrombotic complications, we reserve the use of antifibrinolytic agents to those patients who are bleeding and have a ROTEM demonstrating a typical hyperfibrinolysis trace.

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