ACE inhibitor, an exaggerated effect of angiotensin II as a facilitating mechanism for progressive thrombosis in selected cases with a stimulated renin angiotensin system is an interesting hypothesis. The presentation of such a hypothesis was the intent of our report. Recently, we also had a patient with high plasma renin levels, occluded renal artery and coexisting pulmonary embolus (personal observation). Such a potential interaction between the renin angiotensin system and prothrombotic mechanisms adds to the importance of a tight follow-up of risk patients, and anticoagulation should be considered part of the armamentarium in high risk patients with a stimulated renin angiotensin system.

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Prevention of activation of coagulation during haemodialysis and the use of warfarin

Sir,
I read with interest the well written article in the July 2003 issue entitled ‘Different effects of enoxaparin and unfractionated heparin (UFH) on extrinsic blood coagulation during haemodialysis: a prospective study’ by Naumnik et al. [1]. The authors studied 25 haemodialysis (HD) patients using a single dose of enoxaparin. Of these, 12 were randomly assigned to receive UFH as a bolus dose plus continuous infusion, and 13 were maintained on enoxaparin. All patients were followed prospectively for 12 weeks. Among other parameters, a marker of activated coagulation, prothrombin fragment 1+2 (PF1+2) was measured at the start and after 10 and 180 min of HD. They found that PF1+2 was significantly higher than normal pre-dialysis in all patients. In patients switched to UFH, PF1+2 increased significantly during HD, indicating that plasma PF1+2 levels in six warfarin-free patients remained stable until 180 min of dalteparin-anticoagulated HD, and then increased by a mean of 17% at 240 min compared with baseline [2]. Detailed analysis of the graphically presented data in the elegant Norwegian study (figure 1, middle) indicates that plasma PF1+2 levels in six warfarin-free patients were within normal limits both at the start and during HD.

It is important to know if any of the patients in the study of Naumnik et al. [1] were treated with warfarin since warfarin reduces the blood levels of functional thrombin and therefore also the levels of PF1+2. They stated that criteria for exclusion included application of antiplatelet drugs, but nothing is mentioned regarding warfarin. If some or all of the patients were treated with warfarin, it is not surprising that the levels of PF1+2 did not rise during HD.

Without information concerning warfarin treatment, the conclusion indicated by the authors that enoxaparin ensures a higher anti-thrombotic effect than dalteparin may be disputed.

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Reply

Sir,
We appreciate the important points made by Sagedal in her Letter. In our article we indeed failed to provide vital information on the use of oral anticoagulants [1]. Actually, none of our HD patients was treated with either warfarin or acenocoumarol. Inspired by Sagedal’s final comment that enoxaparin may not be superior to dalteparin for blood anticoagulation during HD, we attentively compared the two reports [1,2]. Detailed analysis of the graphically presented data in the elegant Norwegian study (figure 1, middle) indicates that plasma PF1+2 levels in six warfarin-free patients were within normal limits both at the start and during HD.

However, as we did not perform the PF1+2 measurements at 240 min of the HD session, the potential increase in the activated coagulation marker occurring over the last hour of the procedure could have been missed. Therefore, our previous suggestion that enoxaparin provides a better over-dialytic anticoagulant effect than dalteparin may not be corroborated enough [1]. This, however, does not affect the main finding of our study that enoxaparin is superior to unfractionated heparin (UFH) in this clinical setting [1].