Introduction:
Previous studies indicate vitamin K-deficiency to be common in critically ill patients without overt bleeding. The aim of this study was to evaluate the effect of intravenously given fytomenadion on routine coagulation status, activities of vitamin K-dependent coagulation factors, thrombin generation and thromboelastography.

Methods:
Critically ill patients with prolonged prothrombin time (PT) – Owren (PT-INR) >1.2 were included during office hours. Routine coagulation status (Owren PT, Quick PT), activity of coagulation factor (F) II, FVII, FIX, FX, protein C, protein S, thrombin generation (TGA) and thromboelastography (ROTEM), were measured before and 24 hours after 10 mg fytomenadion had been given intravenously. The exclusion criteria were on-going treatment with warfarin- or NOACs, hepatocellular cancer, liver resection <6 months, known coagulopathy or treatment with fytomenadion <36 hours.

Results:
27 patients were included. A significant decrease of Owren PT and Quick PT was demonstrated 24 hours after given fytomenadion (p<0.001), Figure 1. The activity of FII, FVII, FIX and FX was increased (p<0.01, p<0.01, p<0.01, p<0.001). The activity of protein C, protein S and TGA were unchanged. No changes were demonstrated in ROTEM, except for maximal clot formation (MCF), indicating increased clot strength. The sensitivity analysis demonstrated that patients with higher Owren PT>1.3 at baseline showed a stronger response to fytomenadion, including increased TGA.

Conclusion:
PT decreases and the activity of FII, FVII, FIX and FX increases 24 hours after intravenously given fytomenadion in critically ill patients. In patients with more pronounced PT increase at baseline, these effects were even stronger and also included increased TGA.
Figure 1. PT before and 24 hours after the administration of 10 mg fytomenadion given intravenously. Box-plots with min-max whiskers. PT= prothrombin time.

***p<0.001.