Intravenous literature: Ryan, M.L., Thorson, C.M., King, D.R., Van Haren, R.M., Manning, R.J., Andrews, D.M., Livingstone, A.S. and Proctor, K.G. (2012) Insertion of central venous catheters induces a hypercoagulable state. The Journal of Trauma and Acute Care Surgery. 73(2), p.385-90.

Abstract:

BACKGROUND:Â Central venous catheters (CVCs) increase the risk of venous thromboembolism. We have previously demonstrated that pulmonary artery catheters are associated with a hypercoagulable state in an animal model and in patients. The purpose of this study is to determine whether the insertion of a CVC is associated with a similar response.

METHODS:Â Animal: 7F femoral artery catheters were placed in healthy anesthetized swine (N = 16). Serial arterial blood samples were drawn immediately before and after an 8.5F jugular vein CVC and then for 3 hours after CVC removal. Samples were analyzed using kaolin-activated thromboelastography (TEG) at precisely 2 minutes. Human: An institutional review board-approved prospective observational trial was conducted, with informed consent, in patients with critical illness (N = 8) at a Level I trauma center. Blood was drawn from indwelling arterial catheters immediately before and 60 minutes after CVC insertion. Samples were stored in sodium citrate for 15 minutes before TEG. Routine and special coagulation tests were performed on stored samples in the hospital pathology laboratory.

RESULTS:Â Insertion of a CVC decreased TEG clotting time (R) by 55% in swine and by 29% in humans ($p < 0.001$ and 0.019, respectively). Initial clot formation time (K) was reduced by 41% in swine and by 36% in humans ($p = 0.003$ and 0.019). Fibrin cross-linking (Î±) was accelerated by 28% in swine and by 17% in humans ($p = 0.007$ and 0.896), but overall clot strength (maximum amplitude) was not affected. There was no change in routine or special coagulation factors, including von Willebrand factor, antithrombin III, prothrombin time, international normalized ratio, or activated partial thromboplastin time. In animals, the hypercoagulable TEG response was persistent for 3 hours after CVC removal and was prevented by pretreatment with enoxaparin ($n = 4$) but not heparin ($n = 2$).

CONCLUSION:Â In healthy swine and patients with critical illness, a systemic hypercoagulable state occurred after CVC insertion, and this may partially account for an increased risk of venous thromboembolism. However, because the sample size was small and not powered to detect changes in coagulation proteins, no inferences can be made about the mechanism for the hypercoagulable response.