INTRODUCTION

Pregnancy - induced hypertension is a high - risk problem for mother and fetus. Fortunately the incidence is only 5% of all pregnancies. It is also primarily a disease of primigravidas and pregnant women at the extreme ends of the age scale (Pritchard et al., 1985). Data have been presented by Weinstein, (1982) that described what may be a separate entity called the HELLP syndrome in severely preeclamptic patients. This syndrome is characterized by hemolysis, elevated liver enzymes and low platelets. Pregnancies complicated by severe preeclampsia - eclampsia and HELLP syndrome are associated with perinatal mortality (Killam et al., 1975; Thiagarajah et al., 1984; Weinstein, 1985), considerable reduction in platelet count, prothrombin - proconvertin (PP), fibrinogen content, factor V as well as prolonged partial thromboplastin time (PTT), quick (prothrombin) time and thrombin time. Also some degree of intravascular coagulation had taken place in infants of pre-eclamptic mothers (Nielson, 1969).

Protein C is a vitamin K dependent plasma protein, (Stenflo, 1976) which in its activated form functions as a potent anticoagulant (Kisiel, 1979). Activated protein C degrades coagulation factors Va and VII by limited proteolysis (Marlar et al., 1981) and may also induce fibrinolytic activity in plasma (Comp and Esmon, 1981). Its role in the normal hemostatic balance was discovered when patients with a familial proneness to recurrent venous thrombosis were found to have low plasma concentration of protein C (36 - 62% of normal) (Griffin et al., 1981; Broekmans

et al., 1983; Pabinger - Fasching et al., 1983). Severe homozygous protein C deficiency was subsequently detected in infants whose protein C concentrations were very low or undetectable and who died of massive venous thrombosis or purpura fulminans (Branson et al., 1983; Estelles et al., 1984; Seligsohn et al., 1984; Sills et al., 1984; Yuen et al., 1986).

Protein C and prothrombin are both vitamin K-dependent proteins, though protein C is an inhibitor of blood coagulation while prothrombin is a procoagulant. Normally there is a delicate balance between procoagulants and inhibitors. Either excess of procoagulants or lack of inhibitors would promote thrombotic tendencies, while lack of procoagulants or excess of inhibitor would induce bleeding. Some coagulation factors (e.g. prothrombin, and factor VII, IX and X) are low in preterm infants thus predisposing them to bleeding (Manco - Johnson et al., 1988).