INTRODUCTION

Renin is a proteolytic enzyme produced in the kidney uterus and chorion which acts on a substrate of alpha-$_2$ globulin to produce the decapeptide angiotensin I to produce the octapeptide angiotensin II. This substance is a potent Vasoconstrictor which is rapidly removed by angiotensinase.

It plays an important part in maintenance of blood pressure and the release of aldosterone which in turn affects sodium and water balance. Development of the fetal renin angiotensin system may also play a role in the mechanism of maternal blood pressure control (Symonds, 1979).

In 1986, Bing and Faarup demonstrated high concentrations of a renin-like enzyme in rabbit myometrium. Gorden et al., (1987) demonstrated, in the same species, that this uterine renin could be released into the peripheral circulation in nephrectomized animals by reducing uterine blood flow. Skinner, et al., (1988), showed very high concentrations of renin in human chorion, and it has been suggested that chorion may provide the source of amniotic fluid renin.

In normal pregnancy, a two-to three fold increase occurs in plasma renin activity, renin concentration and plasma angiotensin II. In pregnancy hypertension, there is general agreement that plasma renin

activity and renin concentration are suppressed due to decreased renin release by the Kidney secondary to sodium and water retention (Weir et al., 1983). However, the key may lie primarily not in angiotensin II levels but in vascular sensitivity, since sensitivity to infused angiotensin II is reduced in normal pregnancy but is increased in pregnancy hypertension (Hibbard, 1988).

The studies of Valloton et al. (1992) have shown that cord blood levels of plasma renin activity and plasma A II are generally higher in cord venous and cord arterial blood than in maternal blood. The cord blood levels in infants born to primigravidae with hypertension showed that the levels of plasma A II were highest in the cord venous circulation whereas the cord arterial levels were similar in normotensive and hypertensive women (Pipkin and Symonds, 1987). This may be an evidence that the placenta is either a site of A II synthesis or the site of converting enzyme activity acting on fetal angiotensin I.