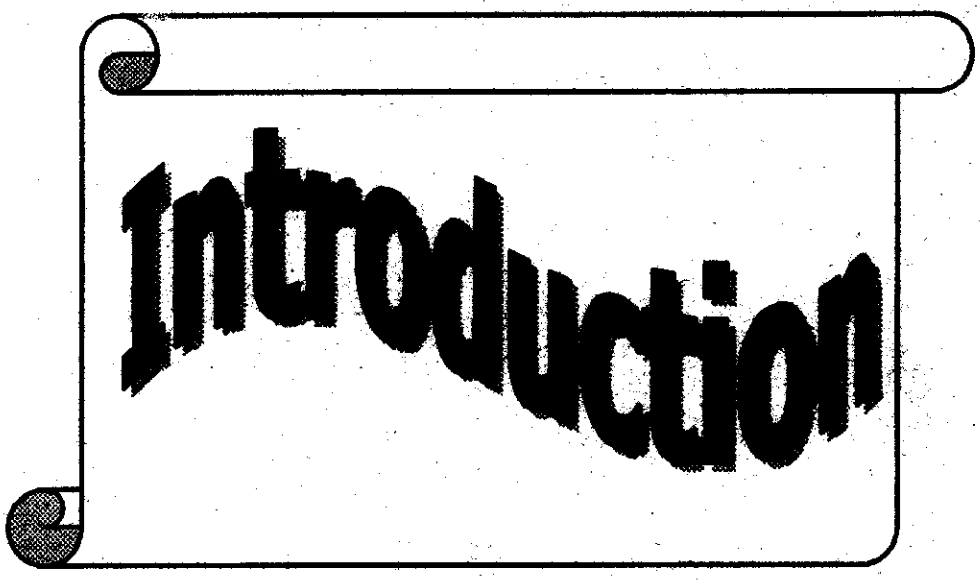
Introduction

INTRODUCTION

Pre-eclampsia is a fascinating disease, the aetiology of which has remained elusive for centuries. (Fried man; 1988).

Many theories were claimed to be a cause of pre-eclampsia; Cooper and Liston (1979) examined the possibility that susceptibility to pre-eclampsia is dependent upon a single recessive gene.

Dietary deficiency especially for calcium has been suspected as a cause of pre - eclampsia [Belizan & et al (1988), Marcoux & et al 1991].

Endothelins are claimed in the causation of pre-eclampsia where higher levels were reported in pre - eclamptic women (Clark; 1992, Nova; 1991).

Prostaglandins are implicated in the causation of pre - eclampsia. Prostacyclin (PGI_2); the vasodilating factor may account for haemo-dynamic changes in pregnancy while increase of "Thromboxane A_2" the vaso-constrictor, and platelet aggregating factor is seen in pre - eclampsia (Friedman, 1988).

Some studies have indicated a decreased urinary out put of stable prostacyclin metabolites in pregnancy Induced

Hypertension which suggests that impaired prostacyclin production may also be involved in the aetiology.

Recently Kaaja et al (1995) found that the metabolic changes "hypertriglyceridaemia - Hyperinsulinaemia - low H.D.L_2 cholesterol, hyperuricemia" in P.I.H. resemble the main features of "Insulin resistance syndrome". This may result in endothelial cell dysfunction as evidenced by PGI_2 supprersion (Kaaja, 1995).

An interesting analogy of the concept of insulin resistance can by observed in P.I.H. (Kaaja, 1995).

It has been Suggested that insulin resistance might be Common aetiologic factor causing hyperinsulinaemia, hypertension, hypertriglyceridaemia and low serum LDL, a cluster of risk factors for coronary artery disease also designated "Syndrome X" (Reaven, 1988)