INTRODUCTION

The possible role of calcium in the pathogenesis of essential hypertension had recieved increased attention. Some studies had suggested a possitive correlation between serum total calcium levels and the height of blood pressure (Kesteloot and Geboers, 1982). However, other reports had raised the opposite possibility, that an actual deficiency of calcium may somehow be causal (McCarron, 1982 and Villar et al., 1987).

Belizan et al., (1983b), in a study on 36 normotensive primigravidae with normal pregnancy had found that the calcium-supplemented group had significantly lower diastolic blood pressure than the control group between the twentieth and the twenty fourth weeks of gestation. However, no difference was observed at term.

Belizan and Villar, (1980), had found that normal women with low calcium intake have an increase in mean arterial blood pressure that predisposes them to the development of pregnancy-induced hypertension (PIH) during the last part of gestation.

The results of some recent clinical trials have suggested that daily calcium supplementation may be associated with a decreased risk of PIH (Kawasaki et al., 1985 and Villar et al., 1987), although this association has not been demonstrated by others (Marya et al., 1987). Lopez Jarmillo et al., (1989) found that calcium supplementation was associated with a significantly decreased risk of PIH with 4.1% developing PIH in the treatment group versus 27.9% in the placebo group. Treatment was associated with a decrease in both systolic and diastolic blood pressure over the course of pregnancy.

It is important to determine whether an alteration in calcium metabolism occurs in pregnancies complicated by PIH and to determine the value of calcium supplementation on blood pressure.