INTRODUCTION
Hypertension is one of the commonest complications of pregnancy and is one of the commonest causes of maternal and fetal mortality and morbidity in India. The maternal mortality rate in India is 300 - 400 per lac live births and PIH is responsible for 15 2% of total maternal mortality. Pregnancy can induce hypertension in normotensive women or aggravate already existing hypertension. Generalised oedema or proteinuria or both may accompany pregnancy induced or aggravated hypertension.

PIH causes considerable increase in maternal morbidity rate in terms of chronic hypertension among multiparous women having eclampsia. The prevalence of diabetes, developing many years after affection is 2.5 times the expected rate in primiparous and about four times in multiparous eclamptic women. Pre-eclampsia can affect blood glucose control up to 8% of cases and perinatal mortality can be doubled.

PIH also contributes to a great extent towards fetal wastage. Das et al observed that there was a sudden rise in perinatal mortality where blood pressure exceeded 180/110 mmHg, albuminuria exceeded 5 gm / 24 hr urine and blood urea exceeded 40 mg / dl. The large toll of maternal and perinatal death due to hypertension, induced or aggravated by pregnancy are often preventable with careful antenatal supervision and appropriate therapy.

Pregnancy-induced hypertension is divided into three categories:

1. Hypertension alone
2. Pre-eclampsia
3. Eclampsia

The diagnosis of preeclampsia is based on the development of hypertension plus proteinuria or generalised edema or both. The blood pressure is 140/90 mmHg or greater or there has been an increase of 30 mmHg systolic or 15 mmHg diastolic over baseline values on at least two occasions six or
more hours apart

This commonly used classification includes oedema which occurs in about 80% of all pregnant women and is frequently found in normal pregnancy. So hypertension and proteinuria are the definitive signs of pre eclampsia. However, these definitive signs are not only arbitrary but also nonspecific.

Pre eclampsia has been defined as mild when not accompanied by significant proteinuria and blood pressure does not exceed 160 mmHg systolic or 110 mmHg diastolic. It has been defined as severe when blood pressure exceeds 160 mmHg or if proteinuria is ++ or greater on albumin or when there are associated symptoms like headache, visual disturbances, upper abdominal pain, oliguria or signs of retinal haemorrhage, and pulmonary edema.

Eclampsia is characterised by signs of pre eclampsia plus convulsion that are induced by pregnancy induced hypertension. The cause of pre eclampsia is not known. The presence of trophoblast is essential, while a fetus is not as pre eclampsia can develop with hydatidiform mole. Pre eclampsia must therefore be associated with either an abnormality of trophoblast itself or of the maternal adaptation to trophoblast. Pre eclampsia is associated with an increase in the peripheral vascular resistance while cardiac output is maintained resulting in hypertension.

Women with pre eclampsia have reduced plasma volume compared to normal control. A reduced plasma volume has been proposed as the cause of growth retardation of fetus and other manifestations of pre eclampsia. Utero placental blood flow is decreased in pre eclampsia and the more severe the pre eclampsia the greater the compromise.

It the present state of knowledge the management of all hypertensive and proteinuric disorders of pregnancy is essentially dependent upon the risk
to the mother, the risk to the fetus, the duration of pregnancy and associated factors such as age, parity and previous obstetric history

Uptil now prevention of PIH has not been possible Both maternal and fetal results would be better if management of preeclampsia is started in early stage The manifestations of preeclampsia are usually treated with bed rest, sedative and anti hypertenives

In recent years a variety of drugs which lower the blood pressure have been described in literature and many of these are being used to treat hypertension during pregnancy These agents have potential adverse effects both to the mother and fetus How ever it is clear from studies that mother gets benefit from such therapy, the benefit for the fetus have been less obvious

Recent studies (Gallery et al 1979 Micheal and Potter 1982, Redman et al 1976, Rubin et al 1983 ) have demonstrated a diminution of incidence of respiratory distress syndrome, improved fetal growth, a reduction in deterioration of hypertension with proteinuria and few mid trimester abortions The ideal drug for treating hypertension during pregnancy would be one which reverses the pathophysiological features of hypertension without adverse effect to mother and fetus At present there is no agent which fulfills the criteria

Methyldopa is the time honoured antihypertensive and it is widely used in the treatment of PIH. But it is having some disadvantages eg., it takes 12 - 24 hr for adequate therapeutic response and a large dose may be required which can cause oliguria but proteinuria remains unchanged It crosses the placental barrier and accumulates in amniotic fluid and it causes psychic depression, orthostatic hypotension, sodium and water retention Because of these disadvantage necessity is felt for better antihypertensive which can over come the above disadvantages with additional benefit

Nifedipine is - a dihydropyridine calcium channel blocker with potent vasodilator property It causes vasodilation in human pregnant uterine vessels
as well as in foetal placental vessels (Maigaard et al 1984, 1986) ie it at least maintains and possibly increases uteroplacental perfusion. Found to be effective in controlling blood pressure without any maternal and foetal adverse effect reported to improve renal function.

To enunciate the above facts, the present study was conducted. Using calcium channel blocker nifedipine and alpha methyl dopa in the drug treatment of PIH.