Elevated blood pressure during pregnancy is a challenging clinical problem for which the approach to evaluation and treatment differs substantially from that employed in non pregnant patients.

Despite recent major increases in the quantity and sophistication of scientific literature, elevated blood pressure, continues to be a major worldwide cause of maternal and perinatal mortality.

When drug therapy is considered for hypertensive gravidas one must be aware of the likely benefits to mother and fetus but also of potential acute and long term adverse effects on the offspring.

In the present study we are discussing the efficacy of methyldopa and nifedipine and their maternal and fetal safety.

The present study was conducted on 50 patients of pregnancy induced hypertension admitted in the Department of Gynaec and obstet, M. I. B Medical College, Jhansi.

In the present study the majority of patients were in the age group of 21-30 yrs 80 percent in methyl dopa group and 68 percent in nifedipine group followed by 11 percent cases of 31-40 yrs in both the groups.

In our study there was equal distribution of parity cases as 50 percent were primigravida and 50 percent were multigravida.

According to Willms (1993) PIH most often affects nulliparous women. With advancing age, the incidence of chronic hypertension increases and the patient is at great risk of pregnancy aggravated hypertension or super imposed preclampsia. Thus women at either end of reproductive age are considered more susceptible to develop PIH.
The incidence is influenced by parity. From 1983 through 1986, 139 of 49,992 women who delivered at Parkland hospital, Texas, were identified as having PIH or aggravated hypertension. Almost 70% were multiparous but only half of these had proteinuria and thus preeclampsia according to Chesley.

Our observation is contrary to usual the dictum that PIH is a disease of primigravida. We could not exclude pregnancy aggravated hypertension or chronic hypertension from pregnancy-induced hypertension because we could not elicit the history of prepregnancy high blood pressure.

So these case overlapped in our series of cases. Hence for the better result, prolonged follow-up and examination of the patients in non pregnant state is necessary.

Distribution of cases according to the period of gestation was almost equal in both the groups.

Maximum number of patients were of 32-35 of gestation out of which 56 percent were in methyl dopa group 64 percent in nifedipine group. 10% cases were below 32 weeks of gestation and only 30% developed PIH after 35 weeks.

Among 50 patients recruited for drug therapy 36 (72%) were found to have mild PIH and 14 (28%) were found to have severe PIH.

In mild PIH all the patients responded to methyl dopa and nifedipine. 14 in nifedipine and 22 in methyldopa group.

None of the cases responded to single drug in methyldopa group while
al the cases gave response when the drug was combined with atenolol

Majority of cases of severe PIH responded to a single drug in nifedipine group 6(54.5%) while 5 (45.5%) cases responded when combined with atenolol

There was a significant and almost similar fall in mean systolic B P and in mean diastolic B P in both groups, fall being greatest in first 24 hours followed by a more gradual and sustained fall over the next 7 days till B P fell to near normal levels

Five patient of severe PIH of nifedipine group and three patients of severe PIH of methyldopa group did not respond to antihypertensive therapy

The mean pre treatment B P in methyldopa was 158 ± 17 31 systolic and 103 8 ± 7 52 mmHg and post treatment it was 139 44 ± 8 1 mmHg systolic and 92 08 ± 4 1mmHg diastolic. The decrease in systolic and diastolic B P was significant

Similarly the mean pretreatment B P in nifedipine group was 170 32 ± 25 mmHg systolic and 102 ± 12 72 systolic and 86 24 ± 18 36 diastolic

This is comparable to the study of Contantine and Beevers

Amongst majority of methyldopa group 22 (88%) cases improvement was observed in edema whereas deterioration was observed in cases (12%) As regards nifedipine group amongst 21(84%) cases improvement was observed whereas deterioration was observed in 4 (16%) cases

Proteinuria improved in 43(86%) of cases as detected by dextrostix paper from +++ down to trace. This improvement of proteinuria was almost equal in both methyldopa and nifedipine groups. The results were similar to the study by Redman

In contrast three patients in nifedipine group and one patient in
methyldopa group showed deterioration of proteinuria

Serum creatinine and uric acid concentrations were measured at the time of admission and biweekly thereafter.

The mean serum creatinine concentration in methyldopa group was 88 ± 27 mg% before treatment and 79 ± 29 mg% after treatment and the decrease was not statistically significant.

Similarly, the mean serum creatinine concentration in nifedipine group was 88 ± 19 mg% before treatment and 85 ± 34 mg% after treatment and this decrease was not statistically significant.

The mean serum uric acid concentration in methyldopa group before and after treatment was 686 ± 414 and 392 ± 105 mg% followed by nifedipine group 556 ± 108 and 414 ± 167 mg% this decrease was highly significant statistically.

Dunlop and Donaldson (1977) observed that significant changes in renal tubular function occur in PIH. Plasma urate levels which fall in second trimester of normal pregnancy return to normal non-pregnant level at term.

In PIH serum uric acid level are raised and such changes may be observed even before the appearance of hypertension (Redman et al. 1976)

Increase in the serum uric acid level have been observed by many workers in India. The serum uric acid levels have a prognostic value. There is a close relationship between the uric acid level and the severity of hypertension (P.K. Devi 1989). Redman, Beiler and Bonnar 1976 have suggested that serum uric acid is a useful prognostic index for the development of severe PIH.

In majority of cases of mild PIH the period of termination of gestation
was >36 wk in 19 (86.4%) cases of methyldopa group whereas in 13 (93%) patients of nifedipine group duration of pregnancy was prolonged beyond 36 weeks.

In severe PIH seven patients continued their pregnancy for more than 36 weeks. While in methyldopa group only one patient was able to continue her pregnancy beyond 36 weeks.

The mean prolongation of pregnancy with methyldopa was 22.68 ± 12.6 days while with nifedipine it was 24.92 ± 10.8 days.

The results are comparable to the study of Kaur et al. The mean duration of treatment was 17.28 ± 11.58 days in methyldopa group followed by 12.5 ± 6.17 days in nifedipine group. No maternal death was reported in our study which was comparable.

The pregnancies were terminated due to an inadequate response to anti-hypertensive drugs.

Regarding the mode of termination of pregnancy in mild PIH, 16 patients had vaginal delivery in methyldopa group followed by 12 patients of nifedipine group having vaginal delivery.

In severe PIH, the mode of termination of pregnancy in methyldopa group was that 2 patients had LSCS & one had vaginal delivery where as in nifedipine group seven had LSCS due to uncontrolled BP, failure to respond to therapy and non-responsiveness to combination anti-hypertensives.

Majority of cases had LSCS due to uncontrolled Blood pressure 7 (41.17%) in both the groups followed by fetal distress 5 (29.4%).

However there was no distinct difference in the duration of therapy and mean duration of labour between the two groups.
The prenatal outcome was as such.

The mean birth weight of babies in methyldopa group was 2 55 ± 86 kg while in nifedipine group it was 2 62 ± 1 44 kg which is definitely better than the birth weight in methyldopa group but this increase in birth weight was not found to be statistically significant.

In methyldopa group 16 babies (66.6%) were appropriate for gestational age and 8 (33%) were IUGR while in nifedipine group 16 babies (69%) were IUGR and 16 (50%) were appropriate for gestational age. These results were comparable with the study of Redman and Ounsted.

There were two intrauterine deaths in nifedipine group and one intrauterine death in methyldopa group. Apgar score was comparable in both the groups. Failure of the fetus to grow or diminution of amniotic fluid volume as estimated clinically or by sonography are ominous signs of fetal jeopardy.

In our series of 50 patients 15 had IUGR diagnosed clinically and by ultrasonography and were terminated by LSCS, out of which 8 (32%) were in methyldopa group followed by 7 (28%) in nifedipine group.

There was one neonatal death each in methyldopa and nifedipine groups. Regarding neonatal complications 4 (16%) babies in methyldopa group had birth anoxia followed by 3 (12%) babies in nifedipine group.

Jaundice occurred in 2 (8%) cases of methyldopa group and 3 (12%) cases of nifedipine group. There were 4 (16%) admissions to the NICU in methyldopa group followed by 3 (12%) admissions in nifedipine group.

The mean duration of stay in NICU was 5.8 ± 2.2 days in methyldopa group followed by 3.75 ± 1.70 days in nifedipine group.
There were no report of septicemia and aspiration pneumonia in our study.

This is comparable to the study by Vaneet et al 1996 who reported a better perinatal outcome in neonates whose mothers received nifedipine (mean stay 3.6 SD 2.41) for treatment of pregnancy-induced hypertension as compared to those who received methyldopa (mean stay 5.6, SD 2.36) thus the neonates in nifedipine group needed to stay in NICU for a shorter period compared to methyldopa group.

There were no perinatal deaths in their study where as in our study there was one perinatal death each in methyldopa and nifedipine group.

Our results are also comparable to the study by Jayawardana who reported the mean birth weight of 2.015 ± 0.957 kg in nifedipine group and 1.922 ± 0.660 kg in methyldopa group. In their study of 126 patients the Apgar score was better in methyldopa group in comparison to nifedipine group.