## THE IMPACT OF MEAN-ARTERIAL PRESSURE IN THE MIDDLE TRIMESTER UPON OUTCOME OF PREGNANCY

by

Manju Gita Mishra,\* M.B.B.S., D.G.O., M.S. (Pat)

and

D. SINGH,\*\* M.S., F.R.C.O.G. (Lond)

Relationship of hypertension with perinatal loss has been well recognised. Since the precise etiology is not yet established, treatment would be of greater value if one could predict which patient would develop pre-eclampsia and then only the prophylactic management can atleast effectively lower its ill-effects. There is general agreement about the lowering of blood pressure during the mid-trimester. Clinical attention, however, always have been focussed upon the behaviour of blood pressure in the third trimester of pregnancy. Not much stress has been laid to trace back any indication of such adverse pressure behaviours as early as 5th or 6th months. We undertook this study to observe the level of blood pressure in mid trimester of pregnancy and tried to correlate it with pressure rises in 3rd trimester, stillbirth or growth retardation. We also tried to observe the effect of socio-economic levels on the incidence of preeclamptic toxaemia and growth retardation.

The impact which a given blood pressure has upon circulatory dynamics is

probably best expressed by utilizing mean arterial pressure (M.A.P.). This also has been the advantage of working with a single figure which may be readily derived from the systolic and diastolic readings. The most commonly used formula is that advocated by Burton:

$$M.A.P. = \frac{S + 2D}{3}$$

which may be converted to a single formula for the clinical use namely M.A.P. is equal to diastolic pressure plus one third of the pulse pressure.

The blood pressure usually shows drop in the 5th or 6th month averaging about 100/60, 110/70 or even 96/60, which if simplified to mean arterial pressure comes about 75, 85 or so on. The patient who did not show any fall or whose M.A.P. was above 85 were specially observed for their pregnancy outcome and subsequent changes in blood pressure.

## Material and Methods

The study includes 1125 women whose pregnancy terminated in single live or stillbirths. One or more blood pressure readings were taken during 1st, 2nd and 3rd trimesters and labelled as M.A.P. I, II and III. These patients include hospital as well as private patients who had atleast two antenatal check-up before delivery.

<sup>\*</sup>Registrar, Department of Obstetrics & Gynaecology.

<sup>\*\*</sup>Associate Professor of Obstetrics & Gynaecology, Patna Medical College Hospital, Patna.

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The M.A.P. was noted and the outcome of pregnancy was observed in all these patients.

TABLE I

Number of Upper Middle Class and Low Income

Group Patients

Total No. of patients	Upper and Middle class (Group A)	Low income group (Group B)
1125	395	730

The bulk of our patients consisted of low income group. The upper income group patients were mainly private patients.

Out of 395 patients belonging to group A, 300 patients had their M.A.P. 85 mm. or below, only 18.7 per cent showed higher reading than 85 mm. of Hg. blood pressure. M.A.P. was higher than 95 in 21 patients in this category.

In group B, 483 patients out of 730 had

M.A.P. below 85 mm. of Hg. and as many as 247 patients showed higher reading. Eighty-six patients had blood pressure higher than 95 mm. Hg.

This shows that the patients of low socio-economic group are more prone for hypertension in pregnancy and show higher level of mid-trimester M.A.P.

The incidence of toxaemia was significantly higher in the patients showing M.A.P. higher than 85 in group A as well as group B. The incidence rose steeply when M.A.P. II was above 95 mm. Hg.; 14.3 per cent in group A and 19.76 per cent of group B.

The point to stress is that potential toxaemic patients can be picked up in the mid pregnancy and if followed carefully incidence of pre-eclamptic toxaemia can be reduced and its ill-effect can be minimised. In the absence of a proper specific treatment of toxaemia this is the best we can achieve.

TABLE II Incidence of M.A.P. II rise

M.A.P. II	Group A		Group B	
	No. of patients	Percentage	No. of patients	Percentage
75-84	300	75.94	483	66.96
85-94	74	18.73	161	22.05
Above	21	5.31	86	11.98
Total	395		730	

TABLE III
Incidence of Diagnosed Pre-eclampsia

M.A.P.	Group A		Group B	
	No. of patients	Percentage	No. of patients	Percentage
75-84	2 20 20	0.66	6	1.24
85-94	3	4.0	11	6.83
Above 95	3	14.3	17	19.76

TABLE IV
Incidence of Small for Gestational Infants

M.A.P.	Gr	Group A		Group B	
	No. of cases	Percentage	No. of patients	Percentage	
75-84	12	4.0	23	4.75	
85-94	6	8.1	19	11.8	
Above 95	2	9.52	16	18.6	

There were only 20 cases of small for dates babies weighing less than 5 lbs, but more than 37 weeks of gestation. This includes the patients of hypertension, pre-eclamptic toxaemia and unexplained causes. Correct assessment of growth retardation was not so easy in group B where most of the women due to illiteracy could not be sure of their dates. So an overlaping of prematurity is possible. The interesting point to note is the rise in number of growth-retarded babies with rise of M.A.P. and again the per-

centage is higher in low socio-economic group than the higher group.

With each 5 mm. of Hg. rise there was a progressive increase in perinatal mortality rate. In group A perinatal mortality was 14.3 per cent when M.A.P. was more than 95, whereas in group B perintal mortality was 19.7 per cent. Thus the perinatal mortality is higher in low socio-economic group than the higher class for the same pressure level (Tables V and VI).

TABLE V
Incidence of Perinatal Death with rise in M.A.P. II

M.A.P.	Group A		Group B	
	No. of patients	Percentage	No. of patients	Percentage
75-84	5	1.6	15	3.1
85-94	6	7.1	15	9.31
Above 95	3	14.3	17	19.76

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Groups	M.A.P. II	No. of patients	Diagnosed pre-eclamptic toxaemia	Small for gestational infants	Perinatal death
Group A	75-84	300	2 (0.66%)	12 (4.0%)	5 (1.6%)
	85-94	74	3 (4.0%)	6 (8.1%)	6 (7.1%)
	Above 95	21	3 (14.5%)	2 (9.52%)	3 (14.3%)
Group B	75-84	483	6 (1.24%)	23 (4.75%)	15 (3.1%)
	85-94	161	11 (6.83%)	19 (11.8%)	15 (9.3%)
	Above 95	86	17 (19.76%)	16 (18.6%)	17 (19.76%)

It is seen that with rise in the mean arterial pressure there is significant increase in the diagnosed pre-eclamptic toxaemia, intrauterine foetal growth retardation and a progressive increase in the perinatal mortality rate.

## Comment

Browne as early as 1958 observed significant rise in perinatal death rate from 37/1000 when M.A.P. was below 87 to 65/1000 when mean pressure was above 107. Fallis and Langford (1963) studied 113 young primigravida and reported that 82 per cent of those developing toxaemia had mean pressure of 90 or more in second trimester. They observed that higher pressures in second trimester are associated with an increased incidence of toxaemia later and high perinatal loss.

Friedman (1976) made an ertensive analysis of blood pressure trends and noted that perinatal mortality rate increased when pressure exceeded 125/75 a level far below the conventional level of 140/90. Simplification of these values to M.A.P. coincides with the suggestion that perinatal mortality rate increase sharply when M.A.P. rises above 90 mm. mid trimester or 105 mm. in third trimester. Page and Christianson (1976) made a prospective study of 14833 white and black gravidas in an effort to study the impact of mid trimester pressure readings over the pregnancy outcome in relation to development of toxaemia and foetal growth and survival. The M.A.P. exhibited during 5th and 6th months of pregnancy was found to be of great significance. With each 5 mm. Hg. rise in M.A.P. there was a progressive rise in perinatal mortality rate, incidence of toxaemia and intrauterine growth retardation. Our observation confirms the work of Page et al (1976) that the level of hypertension at which its effect occur is much lower and much earlier than we have previously throught. The uteroplacental blood flow seems to be adversely affected in these cases to cause deleterious effect on the foetus.

Coming to the conclusion it is seen that with each 5 mm. Hg. rise in the mean arterial pressure (M.A.P.) there is progressive increase in the perinatal mortality rate, where M.A.P. is 90 mm. Hg or more there is significant increase in the diagnosed pre-eclamptic toxaemia, intrauterine foetal growth retardation. All these events may be due to an impaired utero-placental circulation as sequelae of elevated blood pressure. In all the categories these untoward events are more marked in low socio-economic group than in higher socio-economic group. Women who have an average M.A.P. of 90 or more during the 5th and 6th months should be considered in a high risk category. This is the group where an efficient and frequent antenatal care, and foetal monitoring from 28th week onward by repeated oestriol estrimation and oxytocin challenge tests can minimise all the ill-effects. Maturity of the foetus in such cases may be done by L.S. ratio for inducing labour.

We admit our limitation that we can not cure hypertensive disorders, we can only minimise its ill-effect by advising extra rest and sedation.

Lastly this also opens up further field of research work on the relationship of utero-placental blood flow and maternal hypertension. The biochemical and enzymatic studies should be done from earlier onwards that we did before.

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