INTRODUCTION
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High blood pressure in association with pregnancy has long been regarded as an ominous sign. Kaplan et al (1962) reviewed chronic renal disease and hypertension associated with pregnancy advised that therapeutic abortion should be carried out if the blood pressure rises to 160/100 mm Hg. Fiarley and Kincaid-Smith (1968) concluded that morbidity and mortality rates mainly related to the fetus, were increased in the presence of hypertension.

Nettles and Flanign (1968) found that documented evidence of high blood pressure before the 20th week of gestation implied the existence of hypertension prior to the pregnancy.

Most significant finding associated with hypertensive pregnancy is the increase in fetal loss. The British perinatal morbidity surgery control week (Butlet and Benham, 1963) recorded both the total number of deliveries in one week taking place after the 28th week of pregnancy and the subsequent fate of the fetus. In all there were 16994 singleton pregnancies and 593 fetal deaths. Mortality figures from three consecutive months were also recorded for comparison.

In all these singleton pregnancies in both primiparous and multiparous women, the overall incidence
of raised maternal blood pressure was 27.5%. The hypertension was graded into three groups according to diastolic blood pressure.

Group I patients (mild hypertension) comprised of 17.4% of pregnancies and suffered only a small rise in blood pressure but nevertheless a diastolic reading above 90 mm Hg.

Group II (moderate hypertension) was associated with a higher rate of perinatal mortality.

Group III (severe hypertension) had highest death rate of all.

Group I patients (10.1%) pregnancies were complicated by a diastolic blood pressure of 100 mm Hg or more and fetal mortality in these patients amounted to an overall perinatal mortality rate of 0.66% representing about one fifth of the total fetal mortality. Fetal mortality rate associated with maternal hypertension was 1.2%.

The reports on confidential enquiries into maternal deaths in England and Wales, first started in 1952 have shown a steady decline in the overall number of maternal deaths. There was an even faster decrease in maternal deaths associated with hypertension during 1962-69 (DHSS, 1972).

The decline in maternal mortality was largely due to improved social conditions and better
antenatal care. In a group of over 70 hypertensive pregnancies the perinatal mortality rate was found to be 20% (Jockes et al, 1976).

The changes in blood pressure that occur during normal pregnancy have been well documented by Mac Gillivray et al (1969). They observed that both systolic and diastolic blood pressure fell to their lowest between the 16th and the 24th weeks. The fall in diastolic blood pressure was greater than that in systolic and both values tended to return to normal at term.

Posture is another important factor in interpreting blood pressure levels in pregnancy. After the 30th week of gestation approximately 10% of all normal pregnant women can develop a profound fall in blood pressure if allowed to remain supine for more than a few minutes.

Wright (1962) showed this to be due to a lowered venous return when the uterus compresses the inferior vena cava while the patient was lying on her back. Kerr et al (1964) using contrast radiography immediately before and after delivery showed inferior vena caval obstruction in 10 of 12 patients studied during late pregnancy. The levels of 139 mm Hg systolic and 89 mm Hg diastolic excluding the time of parturition, have been chosen as the upper limit of normal. mainly on
the groups that a rise in perinatal mortality is seen even if higher levels are found only on a single occasion.

Prevention and treatment of pregnancy induced hypertension have learned in the last 10 years. High blood pressure complicates approximately 10% of all pregnancies. Pre-eclampsia, the association of hypertension, proteinuria and oedema, accounts for more than 50% of all the hypertensive disorders of pregnancy and is a major cause of fetal and maternal morbidity and mortality.

Distinguishing between pre-eclampsia and other causes of hypertension on clinical grounds can be difficult because of the lack of specific tests for differential diagnosis. Increased vascular resistance has been claimed as the primary cause of pre-eclampsia, however, a variable hemodynamic profile with relatively high cardiac output, normal filling pressures and in appropriately high systemic vascular resistances is now reported for investigation by most investigators. Imbalance between vasodilator and vasoconstrictor eicosanoids may account for platelet activation and increased responsiveness to pressor peptides. Altered prostacyclin(PGI₂) to thromboxane A₂(TXA₂) ratio in maternal uteroplacental vascular bed may favour local platelet activation and vasoconstriction contributing to placental insufficiency and fetal distress. Recent
evidence seems to suggest that fetal umbilical placental circulation may be the site of the primary vascular injury.

Hypertension complicates approximately 10% of all pregnancies and accounts for 20% of all maternal deaths. Blood pressure normally decreases in peripheral vascular resistance, reaches its lowest point in the second trimester and then gradually increases to or near pregravid levels at term. Normal pregnant women develop vascular resistance to the pressor effect of angiotensin II, which is precociously lost in women who develop gestational hypertension.

Prostaglandins are involved in the development of this vascular refractoriness. An acute and reversible lesion defined "Glomerular endotheliosis" as the basic pathologic pattern of pre-eclamptic neuropathy although gestational hypertension can be super-imposed on undiagnosed essential hypertension or any of a variety of renal diseases.

Treatment of pregnancy induced hypertension is important because it can adversely influence the health and life of both mother and baby. Therapy is beneficial in terms of immediate pregnancy outcome and is not harmful to the child prevention rather than cure should be the aim in managing hypertensive diseases during pregnancy.
The primary goal when treating gestational hypertension is successful termination of the pregnancy with the least trauma to mother and fetus.

Hypertension in pregnancy treated as :-

1. No restriction, bed rest.

2. Elucidation of some of the mechanisms responsible for blood pressure elevation in pregnancy has permitted therapy to be based on more rational principles. The decreased arterial reactivity encountered in normotensive pregnancy is mediated by prostaglandins, preventive therapy using low dose aspirin is an option to prevent development of proteinuria in pre-existing hypertension and provide prophylaxis, against pregnancy induced hypertension.

3. Antihypertensive therapy using sympathetic inhibition with either methyldopa or alpha and beta adrenoceptor blockade.

Vasodilation with hydralazine, calcium entry blockers (Nifedipine) I/V labetalol or diazoxide is primarily used in severe hypertensive patients.

The use of orally administered Nifedipine in severely hypertensive women is associated with encouraging results.

4. When blood pressure levels greater than 170/110 mm Hg needs antihypertensive therapy for maternal safety,
it remains to be proven to what extent foetal growth and welfare can be improved in women with diastolic pressure levels 85-110 mm Hg when adrenoceptor blocking agents are used for blood pressure control. Therapy improved -
- foetal growth.
- prevention of proteinuria.
- Prevention of respiratory distress syndrome.

5. During long term antihypertensive therapy treatment with pindolol yielded better foetal growth than therapy with atenolol.

The disease and its therapy induced special problems with regard to anaesthetic management and care of the newborn infant. In the anaesthetic management of the pre-eclamptic patients drugs that are excreted unchanged by the kidney or that may accentuate liver dysfunction should be avoided. Marked fluctuation in blood pressure should be prevented. Correction of circulating blood volume, anemia and electrolyte disorders should be stored prior to the onset of anaesthesia. If magnesium has been used in large doses and oliguria occurs magnesium toxicity may develop has been used in large doses and oliguria occurs. Magnesium toxicity may develop since the magnesium ions is largely excreted through the kidney. Magnesium toxicity presents as loss of all reflexes coupled with a progressive fall
in blood pressure and respiratory depression and is reversed by I/V administration of a calcium salts.

In general anaesthesia patient should also be infused with a balanced electrolyte solution.
Following pre-oxygenation, a sleep dose of an I/V induction a drug is followed by endotracheal intubation and maintenance with inhalation agents.

Hypertension in pregnancy causes high fetal mortality rate. The increased attention now given to the presence of hypertension in pregnancy has reduced the risk of serious maternal complications to a very low level. To obtain the best results in terms of the mother and especially of the fetus frequent and careful assessment of both is required during the antenatal period. Where this has been possible a reduction in fetal mortality has been obtained.