# COMPARISON OF EFFICACY OF LABETALOL VERSUS ALPHA METHYL DOPA IN THE MANAGEMENT OF PRE ECLAMPSIA

*Dissertation submitted to*

**The Tamil Nadu Dr. M.G.R. Medical University**

*in partial fulfilment for the award of the Degree of*

**M.D. (OBSTETRICS AND GYNECOLOGY)**

**BRANCH-II**


**THE TAMIL NADU Dr. M. G. R. MEDICAL UNIVERSITY**

**INSTITUTE OF SOCIAL OBSTETRICS,**

**GOVT KASTURBA GANDHI HOSPITAL,**

**MADRAS MEDICAL COLLEGE & HOSPITAL.**

**APRIL 2012**

# BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **" COMPARISON OF EFFICACY OF LABETALOL VERSUS ALPHA METHYL DOPA IN THE MANAGEMENT OF PRE ECLAMPSIA "** is the bonafide work done by **Dr. PREETHI. B,** Post Graduate in Obstetrics and Gynaecology under my over all supervision and guidance in the Institute of Social Obstetrics, Kasturba Gandhi Hospital, Madras Medical College Chennai, in partial fulfillment of the requirements of The Tamil Nadu Dr.M.G.R.Medical University for the award of M.D DEGREE in Obstetrics and Gynaecology BRANCH - II.

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## INSTITUTIONAL ETHICS COMMITTEE
## MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
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### CERTIFICATE OF APPROVAL

To
Dr. Preethi .B
PG in MDOG
KGH/ Madras Medical College, Chennai -3

Dear Dr. Preethi .B

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the project / proposal / clinical trail entitled " Compare the efficacy of oral labetolol with routinely used regimen of oral anti hypertensive tablet alpha methyl dopa in management of pre eclampsia" No. 04102010.

The following members of Ethics Committee were present in the meeting held on 22.10.2010 conducted at Madras Medical College, Chennai -3.

| | |
|---|---|
| 1. Prof. S.K. Rajan, MD | -- Chairperson |
| 2. Prof. J. Mohanasundaram, MD,Ph.D,DNB<br>Dean, Madras Medical College, Chennai -3 | -- Deputy Chairman |
| 3. Prof. A. Sundaram, MD<br>Vice Principal , MMC, Chennai -3 | -- Member Secretary |
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| 7. Prof. Md. Ali, MD, DM<br>Professor & Head ,,Dept. of MGE, MMC, Ch-3 | -- Member |
| 8. Thiru. S. Govindasamy BA.BL | -- Lawyer |
| 9. Tmt. Arnold Soulina | -- Social Scientist |

We approve the Proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

## CONTENTS

# INTRODUCTION

Pre Eclampsia is a multi-system disorder of unknown etiology, unique to pregnancy characterized by occurrence of Gestational Hypertension along with proteinuria after the 20th week of pregnancy in a previously normotensive and non-protienuric patient. Gestational Hypertension is defined as Systolic blood pressure of 140 mm of Hg or more and Diastolic blood pressure of 90 mm of Hg or more on two occasions, measured at least 6 hours apart but within 7 days. Proteinuria is defined as excretion of 0.3 g or more of protein in a 24 hour urine sample or >1+ on dipstick in a random sample after excluding urinary tract infection.

Pre Eclampsia complicates 2-8% of pregnancies. Pre Eclampsia can affect virtually every organ system in the body and is a major cause of maternal and perinatal mortality and morbidity. Pre Eclampsia, when not controlled or left untreated can lead to catastrophes like Eclampsia, Abruptio placenta, HELLP syndrome, fetal growth restriction, and intrauterine fetal death. Though the definitive treatment of Pre Eclampsia is termination of pregnancy, aggressive treatment is necessary to ameliorate the disease progression in order to carry on the pregnancy till adequate fetal maturity is obtained.

Oral anti hypertensive drugs have a major role in the management of Pre Eclampsia. A comparison is made here between Labetalol and the commonly used drug Alpha methyl dopa in the management of Pre Eclampsia.

# REVIEW OF LITERATURE

Preeclampsia, a disorder with genetic and immunological components, occurs in approximately 3% of patients – a figure mirrored worldwide. It is possible that unmanaged preeclampsia could proceed to Eclampsia in a large proportion of cases.

Though Eclampsia was known for centuries and attempts were made to find out the cause of Eclampsia, the concept of preeclampsia was discovered much later. It was only in the 19th century, the link between Eclampsia and preeclampsia was made. Rayer's landmark contribution (1839-1841) provided evidence for renal involvement with the observation of protein in the urine of pregnant, edematous women. Lever (1843), of Guy’s Hospital in London, published a paper on a series of cases of puerperal convulsions and reported finding proteinuria in Eclampsia and observed the disappearance of proteinuria after delivery of the child. Lever‘s work led to the belief that Eclampsia was a renal disease, a form of nephritis.

Blood pressure measurements began in 1910, which was when preeclampsia became distinguished from Eclampsia.

Preeclampsia and Eclampsia still account for 20% of maternal deaths worldwide; the current annual worldwide mortality can be estimated to be about 150, 000 women. The principal causes of death in the UK remain pulmonary complications and cerebral haemorrhage. It became clear earlier on in the 20th century that Eclampsia was a preventable disease and some of the

improvement seen during this time has been due to the development of antenatal care with the early recognition of signs of preeclampsia and immediate treatment.

Methyldopa and labetalol have for many years been primary agents used for control of blood pressure in pregnancy.

Mahmoud TZ, Bjornsson S, Calder AA (1993) prospectively studied the effects of oral labetalol therapy in patients with moderate to severe pregnancy induced hypertension (PIH). The outcome variables were blood pressure control, effect on umbilical artery flow velocity waveforms (UAFVW) and fetal outcome. Forty-two patients were recruited, all had moderate to severe PIH. The mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) on entry were 154 +/- 7 mmHg and 104 +/- 5 mmHg, respectively. All had significant proteinuria. After 1 week on labetalol therapy, 85% of patients had their blood pressure controlled. The reduction in both SBP and DBP was statistically significant. There were no significant changes in UAFVW, Resistance Index (RI), uric acid or platelets. The mean birth weight was 2712 +/- 609 g. No perinatal mortality occurred in this study. Labetalol is an effective drug in controlling blood pressure and does not adversely affect the UAFVW. No neonatal problems were attributed directly to the drug. Fetal outcome was satisfactory despite the 12 fetuses that were growth-retarded. Labetalol allows safe prolongation of pregnancies complicated by PIH.

Richards et al., (1979) Prichard et al., (1975) established that the dual alpha- and beta-adrenoceptor blocking properties of labetalol in normal subjects and

hypertensive patients lead to a similar pattern of circulatory effects. Following acute intravenous administration in the supine position, significant reductions in blood pressure occur without a fall in heart rate or cardiac output. After continuous oral administration to patients, small reductions in resting heart rate are usually found (Lund-Johansen, 1979), although individual changes are influenced by the degree of resting sympathetic drive. Marked reductions in resting heart rate after labetalol may follow intravenous administration (Cumming et al., 1979; Marx & Reid, 1979). Cardiac output at rest does not usually change much after labetalol treatment (Koch, 1976; Edwards & Raftery, 1976; Mehta & Cohn, 1977).

Mabie and coworkers (1987) compared bolus intravenous labetalol with intravenous hydralazine in the acute treatment of severe hypertension. They found that labetalol had a quicker onset of action and did not result in reflex tachycardia. In terms of fetal effects, blood pressure reduction with labetalol does not result in fetal distress (Mahmoud and co-workers, 1993) unlike acute blood pressure reduction with hydralazine where 15% of fetuses will exhibit distress frequently requiring immediate cesarean section for delivery. Studies of uteroplacental blood flow indicate that no decrease in perfusion occurs despite the reduced maternal blood pressure Lunell and co-workers, 1982). As with all beta-blockers, labetalol has been associated with hypoglycemia, bradycardia and hypotension but neonatal outcome is uniformly good and ACOG currently recommends labetalol as one of the first line antihypertensive medications for preeclampsia (ACOG, 1996).

Labetalol has some interesting, and potentially, important non-antihypertensive effects that may be beneficial in preeclampsia. Among these are an anti-platelet aggregation action, a thromboxane reducing effect (Greer and co-workers, 1985), and a fetal lung maturation accelerating influence (Micheal and co-workers, 1980).

A prospective placebo controlled study done in Nottingham (1992) included 144 women (86 primigravidae) who developed PIH after 20 weeks gestation. They were treated with either oral labetalol up to 600 mg daily or placebo. Main outcomes measured included the number of days spent as an antenatal inpatient; the development of proteinuria; the perceived need for induction of labour or elective caesarean section; and gestation age at delivery. According to the study, Labetalol significantly lowered the blood pressure and reduced the incidence of proteinuria. However, neither the number of days spent as an antenatal inpatient, nor the perceived need for induction of delivery or elective caesarean section, nor the gestation age at delivery differed significantly between the two treatment groups. The placebo treated early-onset group seemed to have more patients with severe hypertension (> 150/110 mmHg) and a greater requirement for additional antihypertensive therapy prior to labour than the group treated with labetalol.

## PRE ECLAMPSIA

Preeclampsia is best described as pregnancy-specific syndrome that can affect virtually every organ system. Although preeclampsia is much more than

simply gestational hypertension with proteinuria, appearance of proteinuria remains an important objective diagnostic criterion. Proteinuria is defined by 24-hour urinary protein excretion exceeding 300 mg, a urinary protein: creatinine ratio of >= 0.3 or persistent 30 mg/dl (1+ dipstick) in random urine samples (Lindheimer and colleagues, 2008 a). Some women may have atypical preeclampsia with all aspects of the syndrome, but without hypertension or proteinuria, or both (Sibai and Stella, 2009).

## Eclampsia

The onset of generalized convulsions in a woman with preeclampsia that cannot be attributed to epilepsy or other causes is termed Eclampsia. Eclampsia is a preventable condition. Adequate prenatal care, early detection of preeclampsia and its adequate control can prevent Eclampsia; hence the need to treat preeclampsia aggressively.

## Indicators of Severity of Preeclampsia

Pre Eclampsia is divided into mild and severe. Severe Pre Eclampsia is characterized by one or more of the following:

1. Severe hypertension (BP 160/110 mm of Hg or more)
2. Proteinuria (>5 g/24 hours or 3+ or more on random samples)
3. Oliguria (<500 ml /24 hours)
4. Elevated serum creatinine level
5. Pulmonary oedema
6. Thrombocytopenia (<1, 00,000 /cumm)

7. Microangiopathic hemolysis
8. Elevated liver enzymes
9. Symptoms of end-organ involvement like headache, blurring of vision and epigastric pain
10. Fetal growth restriction.
11. HELLP Syndrome

## RISK FACTORS

1. Age – Pre Eclampsia is more likely to occur at both extremes of age, but is greatest in women younger than 20 years of age.
2. Parity – Pre Eclampsia is believed to be a disease of primigravidae. The incidence of pre Eclampsia in multiparous women is lower than primipara women.
3. Obstetric factors – Pre Eclampsia occurs most commonly in pregnancies associated with a large placenta, such as multiple pregnancies, molar pregnancies and hydrops fetalis. Hydramnios and history of Pre Eclampsia in previous pregnancy are also predisposing factors.
4. Pre existing medical disorders – like Hypertension, Gestational Diabetes, Renal disease, Obesity, Thrombophilias (both inherited and acquired) such as Antiphospholipid antibody syndrome, Factor 5 Leiden deficiency, Activated protein C resistance, and hyperhomocysteinaemia.

5. Family history – Women with Pre Eclampsia are more likely to have family history of female relatives (mothers or sisters) affected by preeclampsia.

## AETIOPATHOGENESIS

The exact etiology of Pre Eclampsia remains unknown. Several theories have been proposed over the years. As Boyd stated Pre Eclampsia remains “die krankheit der theorien” – the disease of theories. Any satisfactory theory concerning the etiology and pathogenesis of Pre Eclampsia must account for the observation that Pre Eclampsia is more likely to develop in women who:

- Are exposed to chorionic villi for the first time
- Are exposed to a superabundance of chorionic villi, as with twins or hydatidiform mole
- Have preexisting renal or cardiovascular disease
- Are genetically predisposed to hypertension developing during pregnancy.

Thus, presence of a fetus is not a requisite for preeclampsia, although chorionic villi are essential. The chorionic villi need not be located within the uterus. Worley and associates (2008) reported a 30-percent incidence of preeclampsia in women with an extrauterine pregnancy exceeding 18 weeks gestation.

## Preeclampsia as a Two- Stage Disorder

Observations that abnormal interfaces between maternal, paternal and fetal tissues may cause preeclampsia have led to hypothesis that the syndrome is a two-stage disorder. According to Redman and colleagues (2009), stage 1 is caused by faulty endovascular trophoblastic remodeling that downstream causes the stage 2 clinical syndrome. Importantly, stage 2 is susceptible to modification by preexisting maternal conditions that include cardiac or renal disease, diabetes, obesity, or hereditary influences. Thus, although such compartmentalization is helpful to classify the syndrome for research purposes, preeclampsia is clinically a continuum of worsening disease.

## ETIOLOGY

Instead of being simply ‘one disease,’ preeclampsia appears to be a culmination of factors that likely involves a number of maternal, paternal and fetal factors. Those currently considered important include:

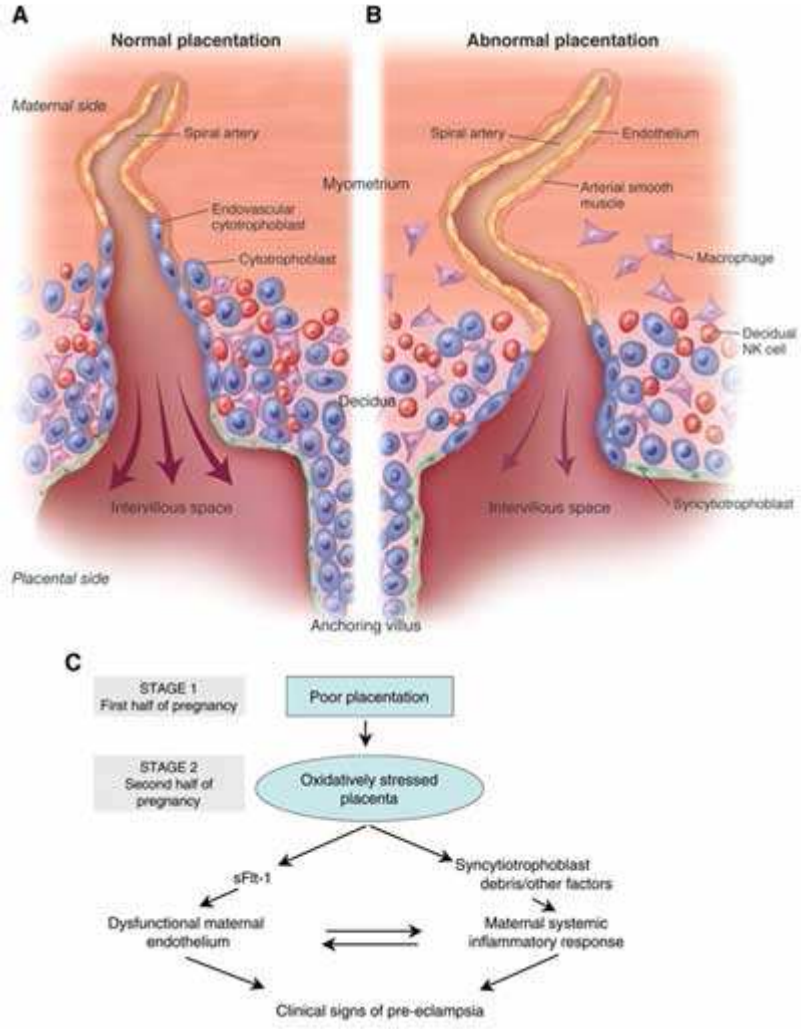
1. Placental implantation with abnormal trophoblastic invasion of uterine vessels
2. Immunological maladaptive tolerance maternal, paternal (placental) and fetal tissues
3. Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy
4. Genetic factors including inherited predisposing genes as well as epigenetic influences.

## Abnormal Trophoblastic Invasion

The inciting organ in Pre Eclampsia is essentially the placenta. Abnormal placentation, in particular lack of dilatation of the uterine spiral arteries leading to placental ischaemia, seems to be the common denominator in the genesis of Pre Eclampsia. Optimal placental development in normal pregnancy involves a process of controlled trophoblastic invasion extending from the deciduas up to the inner third of myometrium. The endothelial lining and the muscular layer of the spiral arteries are disrupted and replaced by cytotrophoblasts, in turn converting the small caliber muscular arteries into large capacity low resistance spaces. The process commences around 10-12 weeks and is completed by 18-20 weeks. In women destined to develop Pre Eclampsia, there is incomplete trophoblastic invasion. With such shallow invasion, decidual vessels, but not myometrial vessels become lined with endovascular trophoblasts. The deeper myometrial arteries do not lose their endothelial lining and musculoelastic tissue. Thus, the mean external diameter of these vessels is only half that of vessels in normal placentas (Fisher and colleagues, 2009), resulting in reduced uteroplacental blood flow.

The ensuing placental ischaemia and hypoxia leads to an aberrant expression of genes which encode for certain cytokines and vasoactive molecules, inciting a systemic inflammatory response that contributes to the pathophysiology of Pre Eclampsia. Using electron microscopy, De Wolf and co-workers (1980) examined arteries taken from the implantation site. They

reported that early preeclamptic changes included endothelial damage, insudation of plasma constituents into vessel wall, proliferation of myointimal cells and medial necrosis. Lipid first accumulated in myointimal cells and then within macrophages. Such lipid –laden cells and associated findings were referred to as atherosis by Hertig (1945). Typically the vessels affected by atherosis develop aneurismal dilatation (Khong, 1991).




## Immunological factors

Loss of maternal immune tolerance to paternally derived placental and fetal antigens, or perhaps its dysregulation, is another theory cited to account

for preeclampsia syndrome. The histological changes at the maternal-placental interface in preeclampsia are suggestive of acute graft rejection (Labrrere, 1988). Tolerance dysregulation might also explain an increased risk when the paternal antigenic load is increased, that is, with two sets of paternal chromosomes – a “double dose”. For example, women with molar pregnancies have a high incidence of early-onset preeclampsia.

Redman and colleagues (2009) recently reviewed the possible role of immune maladaptation in the pathophysiology of preeclampsia. Early in a pregnancy destined to be preeclamptic, extravillous trophoblasts express reduced amounts of immunosuppressive human leukocyte antigen G (HLA-G). This may contribute to defective placental vascularisation in stage 1.

## Endothelial Cell Dysfunction

Endothelial cell dysfunction in Pre Eclampsia occurs in response to placental factors released secondary to ischaemic changes, setting a cascade of events in motion. Briefly, cytokine such as tumour necrosis factor (TNF-alpha) and the interleukins (IL) may contribute to the oxidative stress associated with preeclampsia. This is characterized by reactive oxygen species and free radicals that lead to formation of self propagating lipid peroxides (Manten and associates, 2005). These in turn generate highly toxic radicals that injure endothelial cells, modify their nitric oxide production, and interfere with prostaglandin balance. Other consequences of oxidative stress include

activation of microvascular coagulation manifest by thrombocytopenia and increased capillary permeability manifest by oedema and proteinuria.

## Genetic factors

Preeclampsia is a multifactorial, polygenic disorder. In their comprehensive review, Ward and Lindheimer (2009) cite an incident risk for preeclampsia of 20-40 percent for daughters of preeclamptic mothers; 11-37 percent for sisters of preeclamptic women; and 22-47 percent in twin studies. From their recent review, Ward and Lindheimer (2009) found that more than 70 genes have been studied for their possible association with preeclampsia. This hereditary predisposition is likely the result of hundreds of inherited genes. From their recent review, Ward and Lindheimer (2009) found that more than 70 genes have been studied for their possible association with preeclampsia.

# PATHOGENESIS

### Endothelial Cell Activation

During the past two decades, endothelial cell activation has become the centerpiece in understanding the pathogenesis of preeclampsia. Unknown factors – likely placental in origin – are secreted into the placental circulation and provoke activation and dysfunction of the vascular endothelium. The clinical syndrome of preeclampsia is thought to result from these widespread endothelial cell changes. Grundmann and associates (2008) have reported that

circulating endothelial cell – CEC – levels are significantly elevated fourfold in the peripheral blood of preeclamptic women. Intact endothelium has anticoagulant properties, and endothelial cells blunt the response of vascular smooth muscle to pressor agents by releasing nitric oxide. Damaged and activated endothelial cells may produce less nitric oxide and increase sensitivity to vasopressors and secrete substances that promote coagulation.

## Prostaglandins

A number of prostanoids are thought to be central to the pathophysiology of preeclampsia syndrome. Specifically, the blunted pressor response in normal pregnancy is atleast partially mediated by endothelial prostaglandin synthesis. Compared with normal pregnancy, endothelial prostacyclins (PGI2) production is decreased in preeclampsia, thromboxane A2 secretion by platelets is increased and the prostacyclin: thromboxane A2 ratio decreases. These actions are mediated by phospholipase A2. The net result favors increased sensitivity to infused angiotensin 2, and ultimately, vasoconstriction. Chavarria and co-workers (2003) have provided that these changes are apparent as early as 22 weeks in women who later develop preeclampsia.

### Endothelins

These 21-amino acid peptides are potent vasoconstrictors, and endothelin-1 is the primary isoform produced by human endothelium. Plasma

ET-1 levels are increased in normotensive pregnancies, but women with preeclampsia have even higher levels. According to Taylor and Roberts (1999), endothelins arise from systemic endothelial activation and not from placental source.

### Angiogenic and Antiangiogenic Proteins

Angiogenic imbalance in preeclampsia is used to describe excessive amounts of antiangiogenic factors that are hypothesized to be stimulated at the uteroplacental interface by worsening hypoxia. Trophoblastic tissue of women destined to develop preeclampsia overproduces atleast two antiangiogenic peptides that enter the maternal circulation:

1. Soluble Fms-like tyrosine kinase 1 (sFlt-1)
2. Soluble endoglin (sEng)

## PATHOPHYSIOLOGY

**Placenta -** Placentae from preeclamptic pregnancies have shown an increased incidence of infarcts, haematomas, congested chorionic villi, proliferative endarteritis and degeneration, although the changes are not specific to preeclampsia. Microscopy reveals increased syncitial knots, cytotrophoblastic cellular proliferation, fibrinoid necrosis, endothelial proliferation and calcified and hyalinised villous spots.

**Kidney –** Preeclampsia appears to have a characteristic renal lesion termed as ‘glomerular capillary endotheliosis’. The glomeruli are diffusely enlarged and avascular. Electron microscopy reveals endothelial cell hyperplasia and exudation of foamy macrophages, lymphocytes and polymorphonuclear leukocytes within the capillary lumen and mesangium. The capillary walls show hypertrophied endothelial cells, subendothelial fibrinoid and granular deposits and interposition of mesangial cells resulting in reduced glomerular filtration. However, these lesions resolve completely after pregnancy.

**Liver –** The pathological changes in the liver include periportal haemorrhages, ischaemic lesions and fibrin deposition. Liver damage may vary from mild hepatocellular necrosis to severe lung injury with marked increase in liver enzymes, subcapsular rupture and rarely even liver rupture.

**Brain –** The brain may show cortical and subcortical petechial hemorrhages, subcortical oedema, multiple areas of softening throughout the brain and gross intracerebral haemorrhage in the basal ganglia or pons, often with rupture into the ventricles. The classical microscopic lesions consist of fibrinoid necrosis of the arterial wall and perivascular microinfarcts and haemorrhages.

**Other organs –** Subendocardial petechial haemorrhages may be present in the myocardium. The lungs may demonstrate pulmonary oedema. Haemorrhages and necrosis of the adrenal glands are likely to occur.

## COMPLICATIONS

### Maternal

1. Eclampsia
2. HELLP syndrome
3. Abruption placenta
4. Acute left ventricular failure with pulmonary oedema
5. Acute renal failure
6. Cerebrovascular accidents

### Fetal

1. Intrauterine death
2. Intrauterine growth restriction
3. Prematurity
4. Antepartum and intrapartum asphyxia

## MANAGEMENT

The only definitive cure of preeclampsia is termination of pregnancy. Hence, management is directed towards early detection and amelioration of its progression, the goal being to prolong the pregnancy to achieve fetal maturity and prevent maternal complications. Management is determined by the period of gestation and the maturity and growth of the foetus. If the pregnancy is 37 weeks or more, elective induction of labour should be performed. When hypertension develops earlier, the aim is to continue pregnancy upto 37 weeks

if possible, with close maternal and fetal monitoring to ensure adequate growth and maturity of the baby.

The management of severe preeclampsia at various gestational ages is as follows:

<=24 weeks - stabilize the patient and terminate pregnancy

25 – 33 weeks - expectant management with intensive maternal and fetal surveillance. Deliver if maternal or fetal indication

>= 34 weeks - stabilize the patient, strict fetal surveillance and deliver

Antihypertensive therapy plays a very important role in preeclampsia. Antihypertensive agents reduce the blood pressure and thereby allow continuation of pregnancy and reduce the need to terminate the pregnancy prematurely. The commonly used antihypertensives include

**Methyldopa:**

Methyldopa is a centrally acting agent (alpha-adrenergic receptor blocker) and remains the drug of first choice for treating hypertension in pregnancy. It has been the most frequently assessed antihypertensive in randomized trial and has the longest safety track record. Treatment does not seem to have adverse effects on utero-placental or on foetal haemodynamics. Long term use has not been associated with fetal or neonatal problems and there are safety data for children exposed in utero up to 7.5 years follow up available, the children exhibited intelligence and cognitive development similar

to control subjects. Dose of methyldopa is 250 mg (thrice or four times daily), not exceeding 3 gm/day. Side effects are consequence of central alpha-2 agonism or decrease peripheral sympathetic tone and include decreased mental alertness and impaired sleep leading to fatigue and depression in some patients, increased liver enzymes (in 5% cases) and a positive coomb's test (rare).

**Labetalol:**

Labetalol is an adrenergic receptor blocking agent possessing both alpha-1 (Post synaptic) and betą receptor blocking activity (4 times more potent action on beta-receptors than on alphą receptors). It lowers blood pressure by partially blocking alpha-adrenoceptors in the peripheral arterioles, thus causing vasodilatation and resulting reduction of peripheral resistance. At the same time, blocking of beta-adrenoceptors in the myocardium prevents reflex tachycardia and subsequent elevation of blood pressure. Recommended initial dose 100 mg twice daily, maximum dose is 2,400 mg daily. The peak effects of single oral doses of labetalol occur within 2 to 4 hours. The duration of effect depends upon dose, lasting at least 8 hours following single oral doses of 100 mg and more than 12 hours following single oral doses of 300 mg. The maximum, steady-state blood pressure response upon oral, twice-a-day dosing occurs within 24 to 72 hours. The metabolism of labetalol is mainly through conjugation to glucuronide metabolites. These metabolites are present in plasma and are excreted in the urine, and via the bile, into the faeces. Adverse effects are consequence of alphą receptor block and include fatigue, lethargy,

and exercise intolerance due to alpha 2 blocking effect in skeletal muscle vasculature, peripheral vasoconstriction, sleep disturbances, and bronchospasm. Labetalol should not be used in women with asthma or having history of obstructive airway diseases, uncontrolled CCF, 2nd or 3rd degree AV block, severe peripheral arterial disease and hepatic impairment.

**Nifedipine:**

Nifedipine is a calcium channel blocker, used in preeclampsia as first-line therapy or in combination with other antihypertensives. Nifedipine is available as 5 mg and 10 mg tablets. The dose is 10-20 mg three or four times daily, with a maximum dose of 120 mg/ day.

**Hydralazine:**

Hydralazine acts directly on the arterial smooth muscle to cause vasodilatation. It is given orally in a dose of 25 to 100 mg three or four times daily with a maximum daily dose 300 mg.

# AIM OF THE STUDY

This study compares the efficacy of oral Labetalol versus oral Alpha methyl dopa in the management of Pre Eclampsia in terms of reducing the blood pressure, need for labour induction, mean birth weight, APGAR score and rate of neonatal admissions.

# MATERIALS AND METHODS

## STUDY DESIGN:

Prospective case control study

## SETTINGS:

This randomized prospective comparative study was conducted at Institute of Social Obstetrics and Govt. Kasturba Gandhi Hospital for Women and Children, Triplicane, Chennai, on hundred patients, diagnosed as preeclampsia and admitted in the Eclampsia ward.

## DURATION OF STUDY:

From September 2010 to August 2011

## METHODOLOGY:

The patients included in this study were assigned to two groups at random of 50 patients in each group.

## GROUP 1:

Tablet Alpha methyl dopa (Aldomet) 250 mg was given thrice daily.

## GROUP 2:

Tablet Labetalol 100 mg was given twice daily.

**INCLUSION CRITERIA**

All patients with Gestational Hypertension (more than 20 weeks of gestation till term)

- Systolic Blood Pressure of 140 mm of Hg or more
- Diastolic Blood Pressure of 90 mm of Hg or more
- Proteinuria ( 0.3 g in 24 hours or more/ 1+ dipstick or more)

**EXCLUSION CRITERIA**

- Chronic Hypertension
- Renal Disease
- Liver Disease
- Bronchial Asthma
- GDM
- Cardiac Disease
- Imminent symptoms :
    - ❖ Headache
    - ❖ Blurring
    - ❖ Epigastric pain
    - ❖ Oliguria(<500 mg/24 hrs)

- Complications –
    - ❖ Acute LVF
    - ❖ Coagulation failure
    - ❖ Intracerebral Hemorrhage
    - ❖ HELLP Syndrome
- Eclampsia

## PROCEDURE

Informed consent was obtained from these patients before administration of the drugs. Blood Pressure was recorded every 12th hourly. The treatment was continued till delivery if the blood pressure is controlled. If the blood pressure was not controlled within 48 hours, the dose of drugs was doubled. Blood pressure was measured by a mercury sphygmomanometer over the right arm in the sitting position after a period of rest for 15 minutes. Korotkoff phase 5 was used to define diastolic blood pressure. Proteinuria was detected using the sulphasalicylic acid test. The period of study was 1 year. The change in BP after 48 hours, need for induction, and mode of termination of pregnancy, birth weight, Apgar score and neonatal admissions were recorded.

# RESULTS AND ANALYSIS

This study was commenced with 100 women and the outcome was analyzed using various parameters. The results were subjected to statistical analysis using the t test and chi square test.

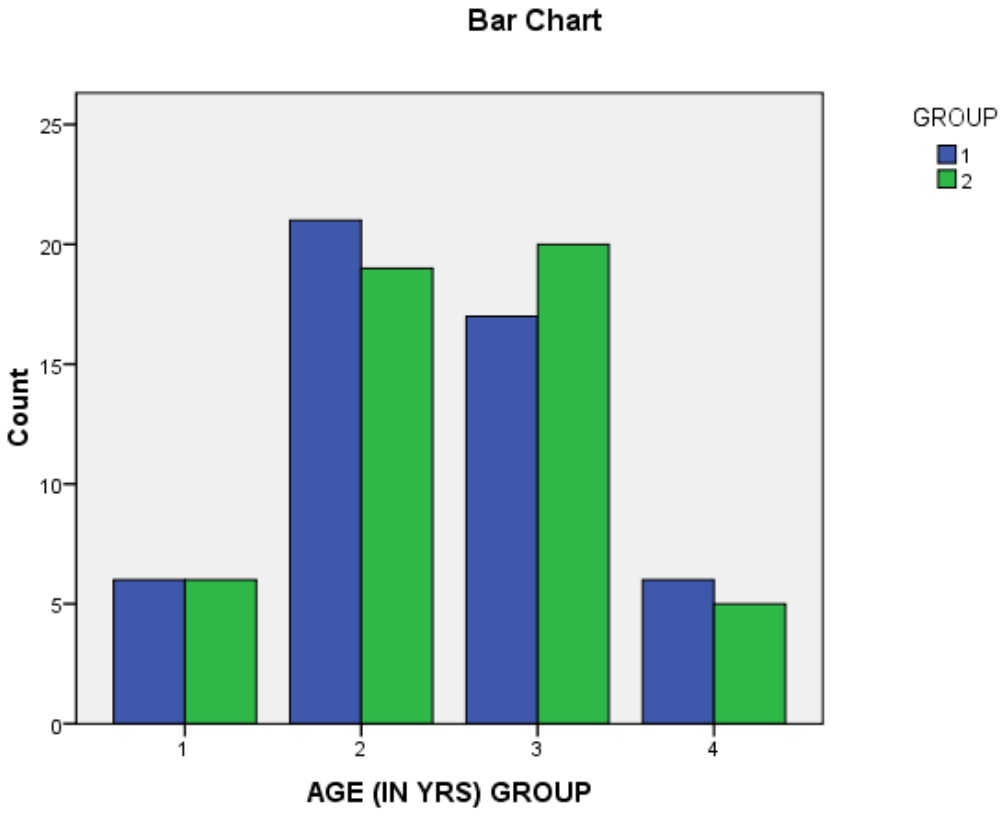
**AGE GROUP:**

**TABLE 1:**

| AGE IN YEARS | LEGEND | | GROUP 1 ALPHA METHYL DOPA | GROUP 2 LABETALOL | Total |
|---|---|---|---|---|---|
| < 20 | 1 | Count | 6 | 6 | 12 |
| | | % within GROUP | 12.0% | 12.0% | 12.0% |
| 21-25 | 2 | Count | 21 | 19 | 40 |
| | | % within GROUP | 42.0% | 38.0% | 40.0% |
| 26-30 | 3 | Count | 17 | 20 | 37 |
| | | % within GROUP | 34.0% | 40.0% | 37.0% |
| >30 | 4 | Count | 6 | 5 | 11 |
| | | % within GROUP | 12.0% | 10.0% | 11.0% |
| | Total | Count | 50 | 50 | 100 |
| | | % within GROUP | 100.0% | 100.0% | 100.0% |

The above table shows the prevalence of preeclampsia in each age group.

Most of the patients in both Groups were in the age group of 21-25 yrs. 42% of cases in Group 1 and 38% of cases in Group 2 were in the age group of 21-25 yrs. However, this was not statistically significant (p value = 0.933)


Bar Chart
GROUP
1
2
Count
25
20
15
10
5
0
1
2
3
4
AGE (IN YRS) GROUP

.

**TABLE 2:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| AGE (IN YRS) | 1 | 50 | 25.50 | 3.808 | .539 |
| | 2 | 50 | 25.82 | 4.443 | .628 |

The above table shows the mean ages of the patients in both the Groups. The difference between the mean ages between the two Groups is not statistically significant (p value = 0.700).

**PARITY:**

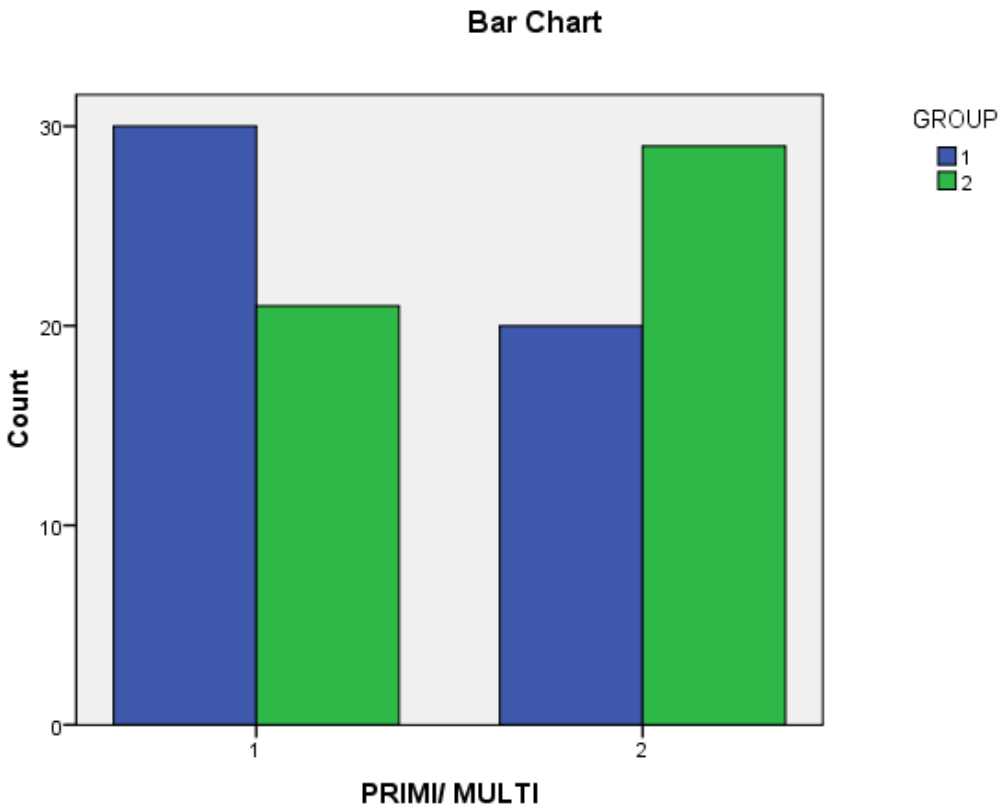
**TABLE 3:**

| PARITY | LEGEND | | GROUP 1 ALPHA METHYL DOPA | GROUP 2 LABETALOL | Total |
|---|---|---|---|---|---|
| PRIMI | 1 | Count | 30 | 21 | 51 |
| | | % within GROUP | 60.0% | 42.0% | 51.0% |
| MULTI | 2 | Count | 20 | 29 | 49 |
| | | % within GROUP | 40.0% | 58.0% | 49.0% |
| | Total | Count | 50 | 50 | 100 |
| | | % within GROUP | 100.0% | 100.0% | 100.0% |

p value = 0.072

60% of women in Group 1 and 40% of women in Group 2 were primigravidae.

40% of women in Group 1 and 58% of women in Group 2 were multigravidae.


Bar Chart
GROUP
1
2
Count
30
20
10
0
1
2
PRIMI/ MULTI

## GESTATIONAL AGE:

**TABLE 4:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| GESTATION AGE (IN WEEKS) | 1 | 50 | 37.90 | 1.930 | .273 |
| | 2 | 50 | 37.94 | 1.596 | .226 |

The above table shows the mean gestational age in both the Groups. The difference between the mean gestational ages between the two Groups is not statistically significant (p value = 0.910).

## BODY MASS INDEX:

**TABLE 5:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| BODY MASS INDEX | 1 | 50 | 27.0076 | 3.28910 | .46515 |
| | 2 | 50 | 27.3086 | 3.76136 | .53194 |

The above table shows the mean BMI in both the Groups. The difference between the mean BMI between the two groups is not significant (p value = 0.671).

## BLOOD PRESSURE:

**TABLE 6:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| SYSTOLIC BLOOD PRESSURE IN MM OF HG | 1 | 50 | 150.60 | 8.668 | 1.226 |
| | 2 | 50 | 150.20 | 8.204 | 1.160 |
| DIASTOLIC BLOOD PRESSURE IN MM OF HG | 1 | 50 | 103.40 | 4.785 | .677 |
| | 2 | 50 | 102.40 | 4.314 | .610 |

The above table shows the mean systolic and diastolic blood pressures in both the Groups. There is no statistically significant difference between the mean systolic blood pressures between the two Groups (p value = 0.813). Also there is no statistically significant difference between the mean diastolic blood pressures between the two Groups (p value =0.275)

## BP AFTER 48 HOURS:

**TABLE 7:**

| BP AFTER 48 HRS | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| SYSTOLIC BLOOD PRESSURE IN MM OF HG | 1 | 50 | 146.20 | 8.303 | 1.174 |
| | 2 | 50 | 144.60 | 8.621 | 1.219 |
| DIASTOLIC BLOOD PRESSURE IN MM OF HG | 1 | 50 | 95.00 | 7.354 | 1.040 |
| | 2 | 50 | 91.20 | 6.273 | .887 |

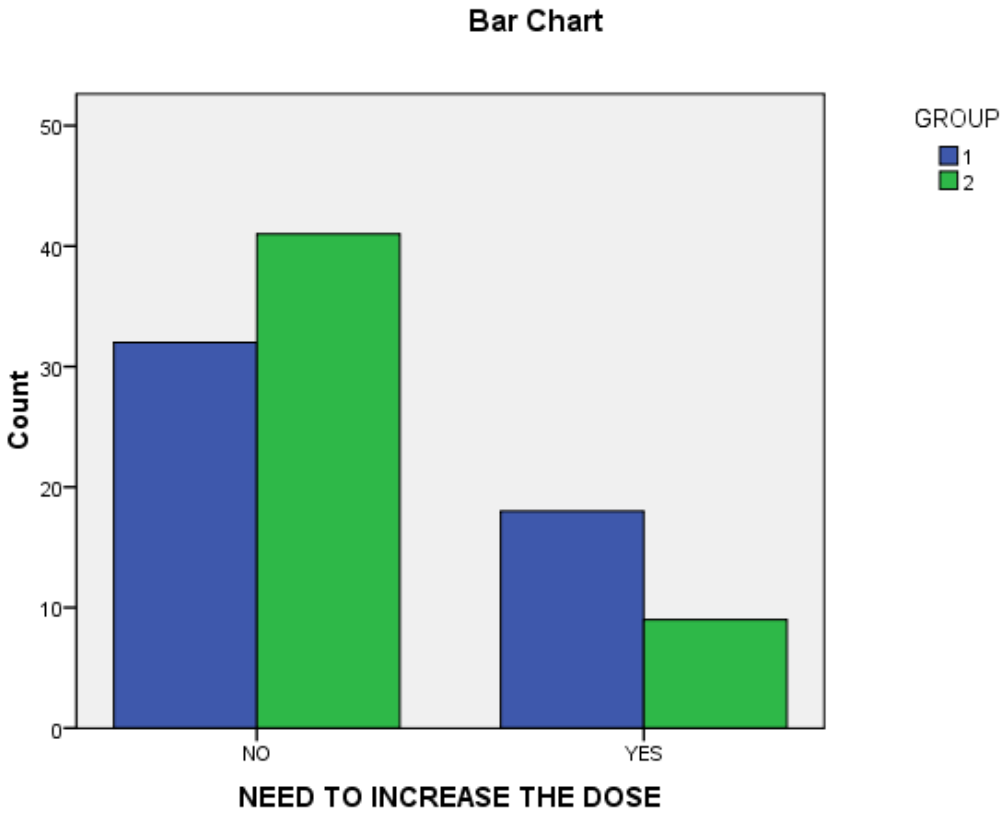
The above table shows the mean systolic and diastolic blood pressures in both the Groups 48 hrs after administration of the drug. There is no statistically significant difference between the mean systolic blood pressures between the two Groups (p value = 0.347). However, there is a statistically significant difference between the mean diastolic blood pressures between the two Groups (p value =0.007)

## NEED TO INCREASE THE DOSE:

**TABLE 8:**

| NEED TO INCREASE THE DOSE | | GROUP 1 ALPHA METHYL DOPA | GROUP 2 LABETALOL | |
|---|---|---|---|---|
| | | | | Total |
| NO | Count | 32 | 41 | 73 |
| | % within GROUP | 64.0% | 82.0% | 73.0% |
| YES | Count | 18 | 9 | 27 |
| | % within GROUP | 36.0% | 18.0% | 27.0% |
| Total | Count | 50 | 50 | 100 |
| | % within GROUP | 100.0% | 100.0% | 100.0% |

This table shows the need to increase the dose after 48 hrs in both the groups. 36% of the cases in Group 1 needed an increase in the dose when compared to 18% in Group 2. There is a statistically significant need to increase the dose after 48 hrs in Group 1 when compared to Group 2 (p value = 0.043).

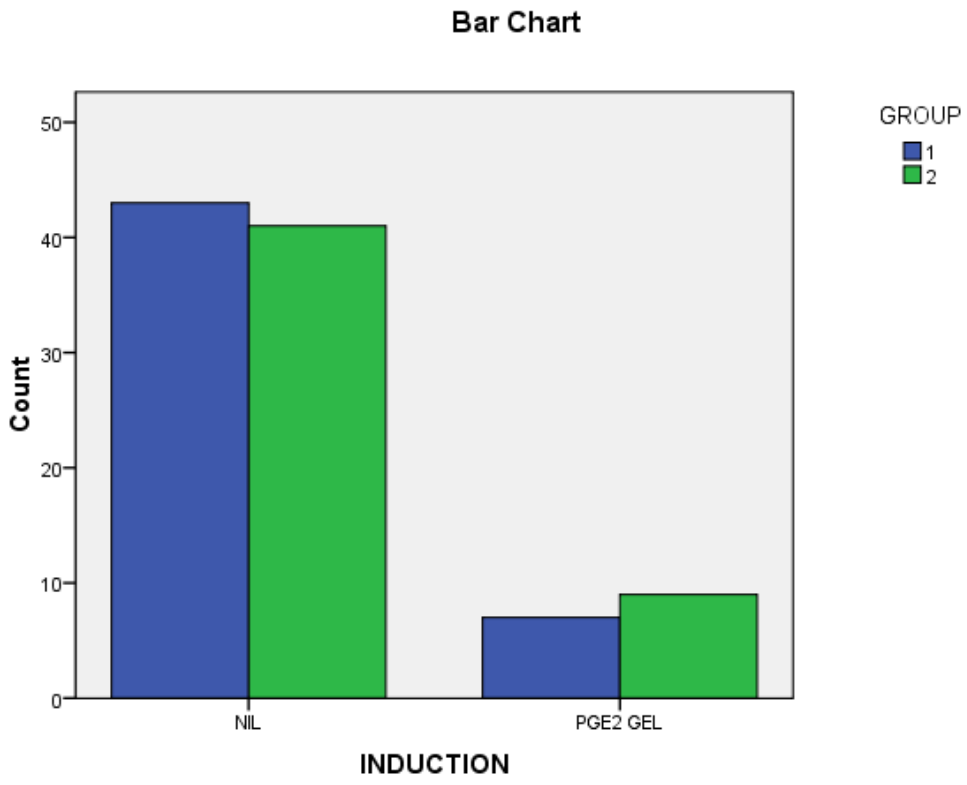

Bar Chart
GROUP
1
2
Count
50
40
30
20
10
0
NO
YES
NEED TO INCREASE THE DOSE

## NEED FOR INDUCTION:

**TABLE 9:**

| INDUCTION | | GROUP 1 ALPHA METHYL DOPA | GROUP 2 LABETALOL | Total |
|---|---|---|---|---|
| NIL | Count | 43 | 41 | 84 |
| | % within GROUP | 86.0% | 82.0% | 84.0% |
| PGE2 GEL | Count | 7 | 9 | 16 |
| | % within GROUP | 14.0% | 18.0% | 16.0% |
| Total | Count | 50 | 50 | 100 |
| | % within GROUP | 100.0% | 100.0% | 100.0% |

The above table shows the need for induction in both the Groups. 14% in Group 1 and 18% in Group 2 were induced with PGE2 gel. However, this is not statistically significant (0.585)


Bar Chart
GROUP
1
2
Count
50
40
30
20
10
0
NIL
PGE2 GEL
INDUCTION

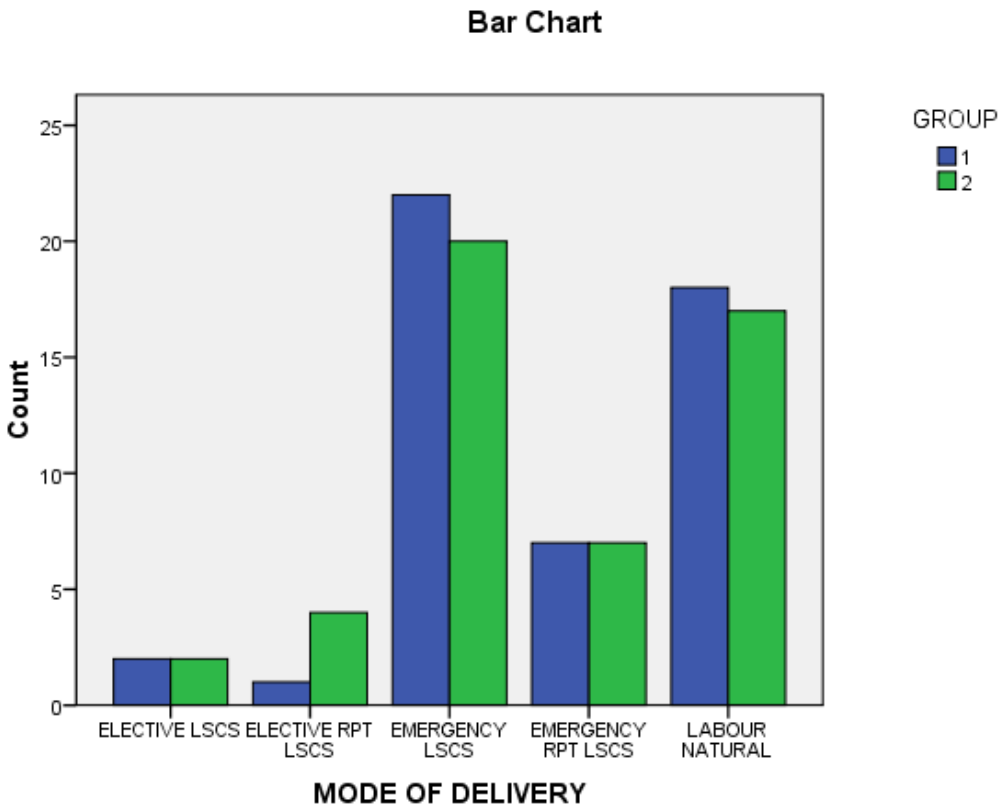
## MODE OF DELIVERY:

### TABLE 10:

| MODE OF DELIVERY | | GROUP 1 ALPHA METHYL DOPA | | Total |
|---|---|---|---|---|
| | | | GROUP 2 LABETALOL | |
| ELECTIVE LSCS | Count | 2 | 2 | 4 |
| | % within GROUP | 4.0% | 4.0% | 4.0% |
| ELECTIVE RPT LSCS | Count | 1 | 4 | 5 |
| | % within GROUP | 2.0% | 8.0% | 5.0% |
| EMERGENCY LSCS | Count | 22 | 20 | 42 |
| | % within GROUP | 44.0% | 40.0% | 42.0% |
| EMERGENCY RPT LSCS | Count | 7 | 7 | 14 |
| | % within GROUP | 14.0% | 14.0% | 14.0% |
| LABOUR NATURAL | Count | 18 | 17 | 35 |
| | % within GROUP | 36.0% | 34.0% | 35.0% |
| Total | Count | 50 | 50 | 100 |
| | % within GROUP | 100.0% | 100.0% | 100.0% |

p value = 0.750

The above table shows the mode of delivery in both the Groups. 44% of cases in Group 1 and 40% of cases in Group 2 underwent Emergency LSCS. 36% of cases in Group 1 and 34% of cases in Group 2 were delivered by Labour natural.


Bar Chart
Count
25
20
15
10
5
0
ELECTIVE LSCS
ELECTIVE RPT LSCS
EMERGENCY LSCS
EMERGENCY RPT LSCS
LABOUR NATURAL
MODE OF DELIVERY
GROUP
1
2

.

## BP AT THE TIME OF DELIVERY:

**TABLE 11:**

| BP AT THE TIME OF DELIVERY | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| SYSTOLIC BP IN MM OF HG | 1 | 50 | 143.00 | 9.530 | 1.348 |
| | 2 | 50 | 139.40 | 9.982 | 1.412 |
| DIASTOLIC BP IN MM OF HG | 1 | 50 | 91.60 | 7.918 | 1.120 |
| | 2 | 50 | 89.40 | 6.197 | .876 |

The above table shows the mean systolic and diastolic blood pressures in both the Groups at the time of delivery. There is no statistically significant difference between the mean systolic blood pressures between the two Groups (p value = 0.068). Also there is no statistically significant difference between the mean diastolic blood pressures between the two Groups (p value =0.125)

## BIRTH WEIGHT:

**TABLE 12:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| BIRTH WEIGHT | 1 | 50 | 2.67736 | .604872 | .085542 |
| | 2 | 50 | 3.11936 | .611578 | .086490 |

The above table shows the mean birth weight in both the Groups. The difference between the mean birth weights between the two groups is 442 g, which is statistically significant (p value = 0.000 < 0.01).

## APGAR:

**TABLE 13:**

| | GROUP | N | Mean | Std. Deviation | Std. Error Mean |
|---|---|---|---|---|---|
| APGAR | 1 | 50 | 8.24 | 1.318 | .186 |
| | 2 | 50 | 8.64 | 1.064 | .151 |

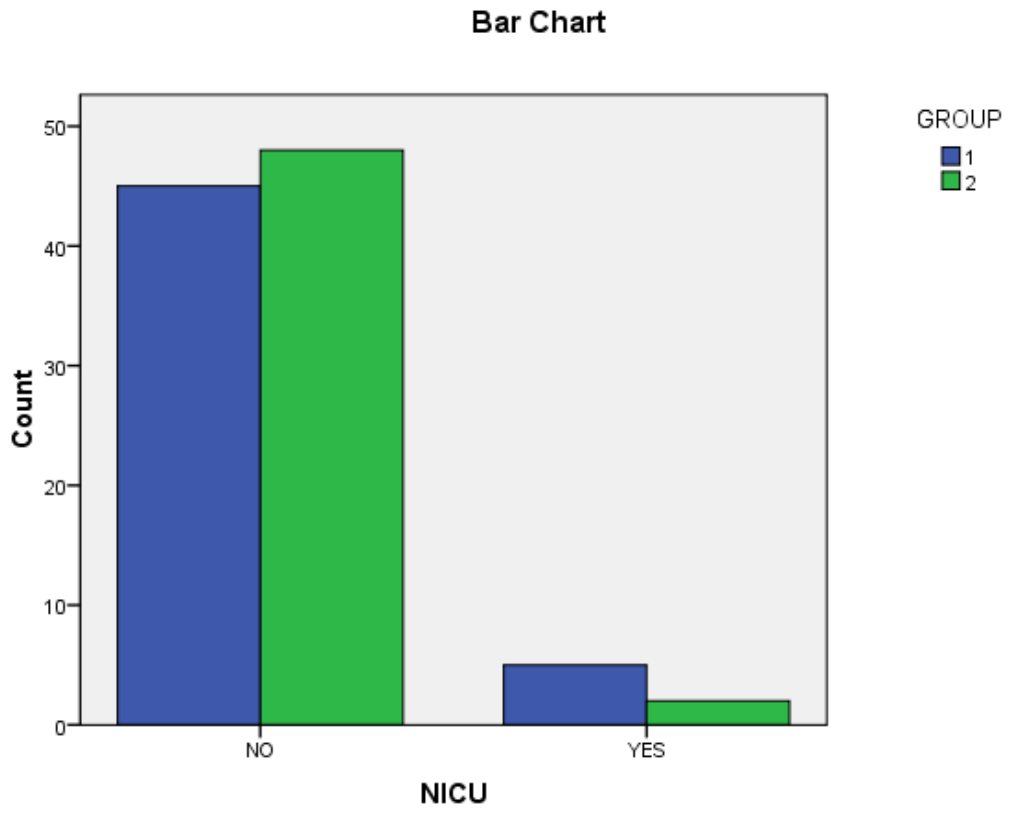
The above table shows the mean APGAR scores in both the Groups. The difference between the mean APGAR score between two groups is 0.4, which is not statistically significant (p value = 0.090)

**NICU ADMISSION:**

**TABLE 14:**

| NICU ADMISSION | | GROUP 1 ALPHA METHYL DOPA | GROUP 2 LABETALOL | TOTAL |
|---|---|---|---|---|
| | | | | |
| NO | Count | 45 | 48 | 93 |
| | % within GROUP | 90.0% | 96.0% | 93.0% |
| | % of Total | 45.0% | 48.0% | 93.0% |
| YES | Count | 5 | 2 | 7 |
| | % within GROUP | 10.0% | 4.0% | 7.0% |
| | % of Total | 5.0% | 2.0% | 7.0% |
| Total | Count | 50 | 50 | 100 |
| | % within GROUP | 100.0% | 100.0% | 100.0% |
| | % of Total | 50.0% | 50.0% | 100.0% |

The above table shows the need for NICU admission in both the Groups. 10% of babies delivered in Group 1 and 4% of babies delivered in Group 2 needed NICU admission. However, this difference is not statistically significant (p value = 0.240).


Bar Chart
GROUP
1
2
Count
50
40
30
20
10
0
NO
YES
NICU

.

# DISCUSSION

This randomized prospective study compares the efficacy of oral labetalol versus oral alpha methyl dopa in the management of preeclampsia.

Preeclampsia is an important cause of maternal mortality and perinatal mortality and morbidity. Oral antihypertensive drugs have played a major role in controlling the disease progression, preventing Eclampsia and other dreaded complications, prolonging pregnancy, and reducing fetal pre maturity.

Though methyl dopa has been used routinely because of its safety profile, several controlled trials have suggested labetalol to be a better drug in controlling hypertension with the least side effects.

A prospective study was carried out at City Hospital, Nottingham, UK in 1979. Nineteen patients with Pregnancy Induced Hypertension whose Mean arterial pressure were >103.3 mm of Hg were selected. They were randomly allocated to two groups. They were given either Labetalol 400 mg or Alpha methyl dopa 750 mg daily. This dose was doubled 3 days later if satisfactory BP control had not occurred. Significant falls in BP only occurred in the group treated with labetalol, and daily BP control was better in this group. There was a higher incidence of spontaneous labour in the labetalol group and a significant difference in the Bishop score of the cervix between the two groups. There were no apparent detrimental effects on the foetus antenatally, during labour or postpartum.

In our study, the initial daily dose of labetalol was 200 mg and the initial daily dose of alpha methyl dopa was 750 mg. The dose was increased after 48 hrs if satisfactory BP control had not occurred. Statistically significant fall occurred only in the diastolic blood pressure in the labetalol group after 48 hrs ($p = 0.007$). There was no statistically significant difference between the need for PGE2 induction between alpha methyl dopa and labetalol groups. 86% of cases in alpha methyl dopa group and 82% of cases in labetalol group went in for spontaneous labour. Only 5% of babies born in alpha methyl dopa group and 2% of babies born in labetalol group required NICU admission. This difference was also not statistically significant (p value = 0.240)

A prospective study (2005) was carried out at Al-Jahra Hospital, Jahra, Kuwait to assess the efficacy and safety of labetalol compared with methyldopa in the management of mild and moderate cases of pregnancy-induced hypertension (PIH). One hundred four primigravidae with PIH were randomly allocated to receive either labetalol (group A) or methyldopa (group B). The dose of the drugs was doubled every 48 h to maintain a mean arterial blood pressure ≤103.6 mmHg. Ten patients in group B (18.5%) developed significant proteinuria whereas none developed proteinuria in group A. Labetalol was quicker and more efficient at controlling blood pressure, having a beneficial effect on renal functions and causing fewer side effects compared with methyldopa. The rate of induction of labor and rate of caesarean section for uncontrolled PIH was less in group A (48% and 1%, respectively) compared with group B (63.0% and 5.6%, respectively). Moreover a higher Bishop score

at induction of labor was noticed in group A. Conclusions: Labetalol is better tolerated than methyldopa, gives more efficient control of blood pressure, and may have a ripening effect on the uterine cervix.

In our study, 44% of cases in alpha methyl dopa group and 40% of cases in labetalol group delivered by Emergency LSCS; 36% of cases in alpha methyl dopa group and 34% of cases in labetalol group delivered by Labour Natural.

In a Randomized controlled trial (1988), labetalol was compared with methyldopa in a randomized controlled trial involving 176 pregnant women with mild to moderate hypertension. Diastolic blood pressure below 86 mmHg was obtained in a similar proportion of women given labetalol or methyldopa. Intrauterine death occurred in four women treated with methyldopa, and the one neonatal death on day 1 occurred in the labetalol group. The average birth weight and the proportion of preterm or small-for-gestational-age babies were similar in both groups. Heart rate, blood pressure, blood glucose, respiratory rate, and Silverman score of the babies did not differ between the two treatment groups, whether the comparison was made for all the infants or only for those that were preterm or small-for-gestational-age. These data indicate that maternal beta blockade with labetalol is as safe as methyldopa for the fetus and the newborn.

In our study, there were no reports of intrauterine deaths. There was a statistically significant increase in the mean birth weight in the labetalol group

when compared to alpha methyl dopa group (3.11 kg and 2.67 kg respectively, p value = 0.00)

# SUMMARY


- This is a study comparing the efficacy of oral Labetalol and Alpha methyl dopa in the management of preeclampsia, carried out in ISO KGH.
- The time duration of the study was from September 2010 to August 2011.
- 100 patients diagnosed as preeclampsia and admitted to ISO KGH were included in this study.
- Inclusion criteria included those who came under the definition of preeclampsia.
- Exclusion criteria included those who had pre-existing medical disorders, those who had features of imminent Eclampsia, Eclampsia and those who developed complications of preeclampsia like acute left ventricular failure and HELLP syndrome.
- These 100 patients were assigned to two groups at random of 50 patients in each group. Group 1 was started on tablet Alpha methyl dopa 250 mg thrice daily and Group 2 was started on tablet Labetalol 100 mg twice daily. Blood Pressure and proteinuria was recorded every 12th hourly.
- The treatment was continued till delivery if the blood pressure is controlled. If the blood pressure was not controlled within 48 hours, the dose of drugs was doubled.
- The relationship of age, parity, gestational age and body mass index to prevalence of preeclampsia in both the groups has been analyzed. Also

the fall in BP after 48 hrs, need to increase the dose of the drugs, need for labour induction, method of delivery, blood pressure at the time of delivery, birth weight, APGAR score and neonatal admissions in each group has been analyzed.

# CONCLUSION

- This is a study comparing the efficacy of Labetalol and Alpha methyl dopa in the management of preeclampsia, in which 50 patients were started on oral Labetalol and 50 patients were started on oral Alpha methyl dopa.
- Significant fall in the diastolic blood pressure after 48 hrs occurred only in the labetalol group ($p = 0.007$).
- In the Alpha methyl dopa group, there was a significant need to increase the dose of the drug after 48 hrs ($p = 0.043$)
- There appears to be no significant difference in the rate of induction between the two groups ($p = 0.585$)
- The mean birth weight was significantly higher ($p = 0.00$) in the labetalol group (3.11 kg) compared to the alpha methyl dopa group (2.67 kg).
- There was no significant difference in the APGAR scores ($p = 0.090$) and rate of neonatal admissions ($p = 0.240$) in both the groups.

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# PROFORMA

NAME:

AGE:

IP NO:

ADDRESS:

OBSTETRIC SCORE:

GESTATION AGE:

HEIGHT:

WEIGHT:

BODY MASS INDEX:

BLOOD PRESSURE AT THE TIME OF ADMISSION:

URINE ALBUMIN AT THE TIME OF ADMISSION:

DRUG ADMINISTERED: Alpha methyl dopa / Labetalol

BLOOD PRESSURE AFTER 48 HRS:

URINE ALBUMIN AFTER 48 HRS:

NEED TO INCREASE THE DOSE: Yes / No

INDUCTION WITH PGE2 GEL: Yes / No

MODE OF DELIVERY:

1. Elective LSCS
2. Elective Repeat LSCS
3. Emergency LSCS
4. Emergency Repeat LSCS
5. Labour Natural

BP AT THE TIME OF DELIVERY:

URINE ALBUMIN AT THE TIME OF DELIVERY:

BIRTH WEIGHT:

APGAR SCORE:

NICU ADMISSION: Yes / No

# Master Chart

| S.NO | GROUP | NAME | AGE (IN YRS) | OBSTETRIC SCORE | GESTATION AGE (IN WEEKS) | HEIGHT (IN CM) | WEIGHT (IN KG) | BODY MASS INDEX | BLOOD PRESSURE (IN MM OF HG) | URINE ALBUMIN | DRUG GIVEN | BP AFTER 48 HRS | URINE ALBUMIN AFTER 48 HRS | NEED TO INCREASE THE DOSE | INDUCTION | BP AT THE TIME OF DELIVERY | URINE ALBUMIN AT DELIVERY | MODE OF DELIVERY | BIRTH WEIGHT | APGAR | NICU |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | ROSEMARY | 35 | G3P1L1A1 | 38 | 151 | 64 | 28.06 | 160/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | PGE2 GEL | 140/90 | NIL | LABOUR NATURAL | 2.92 | 9 | NO |
| 2 | 1 | RAJESWARI | 24 | PRIMI | 37 | 148 | 62 | 28.3 | 150/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 140/80 | NIL | EMERGENCY LSCS | 2.25 | 9 | NO |
| 3 | 1 | SATYA | 27 | G2P1L1 | 36 | 166 | 58 | 21.04 | 160/110 | 3+ | ALPHA METHYL DOPA | 150/110 | 1+ | YES | NIL | 150/100 | NIL | LABOUR NATURAL | 2 | 6 | YES |
| 4 | 1 | SARASWATHY | 24 | PRIMI | 38 | 149 | 70 | 31.53 | 160/100 | 1+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY LSCS | 3.25 | 8 | NO |
| 5 | 1 | KALPANA | 28 | PRIMI | 39 | 144 | 80 | 38.58 | 140/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 2.92 | 9 | NO |
| 6 | 1 | DEVI | 29 | G2P1L1 | 40 | 148 | 71 | 32.41 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/100 | NIL | YES | NIL | 130/80 | NIL | LABOUR NATURAL | 2.1 | 9 | NO |
| 7 | 1 | SARASWATHY | 26 | PRIMI | 37 | 156 | 75 | 30.81 | 150/110 | 2+ | ALPHA METHYL DOPA | 160/100 | 2+ | YES | NIL | 160/110 | 1+ | EMERGENCY LSCS | 2.56 | 9 | NO |
| 8 | 1 | BHUVANESHWARI | 28 | PRIMI | 38 | 151 | 60 | 26.31 | 140/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 2.025 | 7 | NO |
| 9 | 1 | ANURADHA | 20 | PRIMI | 39 | 150 | 54 | 24 | 160/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 140/80 | NIL | LABOUR NATURAL | 2.39 | 8 | NO |
| 10 | 1 | UMA MAHESHWARI | 29 | G2P1L1 | 38 | 157 | 58 | 23.53 | 160/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | ELECTIVE RPT LSCS | 3.135 | 8 | NO |
| 11 | 1 | JENITHA | 21 | PRIMI | 38 | 149 | 53 | 23.87 | 150/110 | 2+ | ALPHA METHYL DOPA | 150/100 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY LSCS | 3.24 | 8 | NO |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12 | 1 | DEVAKI | 32 | G3A2 | 38 | 152 | 69 | 29.86 | 140/100 | 1+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | ELECTIVE LSCS | 2.53 | 9 | NO |
| 13 | 1 | HAJIRA | 27 | PRIMI | 37 | 166 | 80 | 29.03 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 120/80 | NIL | EMERGENCY LSCS | 2.31 | 9 | NO |
| 14 | 1 | AARIFA | 22 | PRIMI | 36 | 159 | 70 | 27.68 | 160/110 | 3+ | ALPHA METHYL DOPA | 150/100 | 1+ | YES | PGE2 GEL | 150/100 | NIL | LABOUR NATURAL | 2 | 9 | NO |
| 15 | 1 | NISHANTHI | 23 | PRIMI | 39 | 142 | 56 | 27.77 | 140/100 | 2+ | ALPHA METHYL DOPA | 130/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 2.21 | 8 | NO |
| 16 | 1 | CHITRA | 25 | PRIMI | 40 | 148 | 54 | 24.65 | 150/100 | 1+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | PGE2 GEL | 150/80 | NIL | EMERGENCY LSCS | 2.75 | 9 | NO |
| 17 | 1 | ANANDHI | 25 | PRIMI | 38 | 149 | 70 | 31.53 | 150/110 | 2+ | ALPHA METHYL DOPA | 150/110 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY LSCS | 2.163 | 7 | NO |
| 18 | 1 | ZARINA BEGUM | 30 | G3P1L1 A1 | 39 | 167 | 78 | 27.96 | 140/110 | 2+ | ALPHA METHYL DOPA | 150/100 | 1+ | YES | NIL | 140/90 | NIL | EMERGENCY LSCS | 2.81 | 9 | NO |
| 19 | 1 | NANCY | 25 | PRIMI | 40 | 164 | 59 | 21.93 | 140/100 | 1+ | ALPHA METHYL DOPA | 130/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 3.201 | 9 | NO |
| 20 | 1 | ROSEMATHY | 20 | PRIMI | 39 | 168 | 60 | 21.25 | 140/110 | 2+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 3.332 | 9 | NO |
| 21 | 1 | VASANTHI | 29 | G2P1L1 | 39 | 156 | 60 | 24.65 | 140/100 | 1+ | ALPHA METHYL DOPA | 140/100 | NIL | YES | NIL | 140/90 | NIL | EMERGENCY RPT LSCS | 3.102 | 9 | NO |
| 22 | 1 | USAINA | 24 | G2P1L1 | 37 | 163 | 59 | 22.2 | 150/100 | 1+ | ALPHA METHYL DOPA | 150/100 | NIL | YES | NIL | 150/90 | NIL | EMERGENCY RPT LSCS | 2.81 | 9 | NO |
| 23 | 1 | SANGEETHA | 26 | PRIMI | 37 | 157 | 60 | 24.34 | 150/110 | 2+ | ALPHA METHYL DOPA | 150/100 | 1+ | YES | NIL | 150/90 | NIL | EMERGENCY LSCS | 3.225 | 8 | NO |
| 24 | 1 | MINNALKODI | 32 | G3P1L1 A1 | 37 | 158 | 61 | 24.43 | 160/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | LABOUR NATURAL | 3.615 | 9 | NO |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 25 | 1 | RASHEELA BEE | 29 | G3P1L1 A1 | 38 | 166 | 64 | 23.22 | 160/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY RPT LSCS | 3.402 | 9 | NO |
| 26 | 1 | SANGEETHA | 23 | PRIMI | 40 | 154 | 68 | 28.67 | 140/100 | 1+ | ALPHA METHYL DOPA | 130/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 3.155 | 9 | NO |
| 27 | 1 | MANJULA | 22 | PRIMI | 39 | 152 | 60 | 25.97 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/100 | 1+ | YES | NIL | 140/90 | NIL | EMERGENCY LSCS | 2.275 | 9 | NO |
| 28 | 1 | INDIRAJI | 25 | G3A2 | 37 | 149 | 54 | 24.32 | 140/100 | 2+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/90 | NIL | ELECTIVE LSCS | 2.85 | 9 | NO |
| 29 | 1 | SHAILAJA | 19 | PRIMI | 40 | 141 | 58 | 29.17 | 140/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/80 | NIL | LABOUR NATURAL | 3.075 | 8 | NO |
| 30 | 1 | MAHESWARI | 32 | G3P2L1 | 37 | 161 | 64 | 24.69 | 150/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY RPT LSCS | 2.235 | 9 | NO |
| 31 | 1 | PERIYANAYAKI | 29 | G2P1L0 | 36 | 160 | 67 | 26.17 | 150/110 | 2+ | ALPHA METHYL DOPA | 150/110 | 2+ | YES | NIL | 150/110 | 1+ | EMERGENCY LSCS | 2.4 | 8 | NO |
| 32 | 1 | PREMA | 23 | PRIMI | 40 | 158 | 69 | 27.64 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | PGE2 GEL | 140/90 | NIL | EMERGENCY LSCS | 2.695 | 9 | NO |
| 33 | 1 | NANDHINI | 20 | PRIMI | 32 | 162 | 68 | 25.91 | 150/110 | 2+ | ALPHA METHYL DOPA | 160/110 | 2+ | YES | NIL | 150/110 | 3+ | LABOUR NATURAL | 0.89 | 4 | YES |
| 34 | 1 | CHITRA | 20 | PRIMI | 38 | 163 | 77 | 28.98 | 160/110 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | LABOUR NATURAL | 2.968 | 9 | NO |
| 35 | 1 | PRINCY | 28 | G2P1L1 | 39 | 166 | 62 | 22.5 | 170/110 | 3+ | ALPHA METHYL DOPA | 160/110 | 4+ | YES | NIL | 160/100 | 3+ | EMERGENCY LSCS | 1.67 | 5 | YES |
| 36 | 1 | HEMALATHA | 26 | G2P1L1 | 37 | 165 | 74 | 27.18 | 160/110 | 3+ | ALPHA METHYL DOPA | 160/100 | 3+ | YES | NIL | 150/100 | 1+ | LABOUR NATURAL | 2.651 | 9 | NO |
| 37 | 1 | MOHANA | 24 | PRIMI | 40 | 154 | 66 | 27.83 | 140/100 | 1+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 120/80 | NIL | LABOUR NATURAL | 2.84 | 9 | NO |

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| 38 | 1 | MUMTAZ | 31 | G2P1L1 | 38 | 161 | 68 | 26.23 | 150/110 | 2+ | ALPHA METHYL DOPA | 150/110 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY RPT LSCS | 3.35 | 9 | NO |
| 39 | 1 | DHANALAKSHMI | 28 | G2P1L1 | 39 | 156 | 64 | 26.3 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY RPT LSCS | 2.963 | 9 | NO |
| 40 | 1 | SHARMILA | 23 | PRIMI | 39 | 149 | 62 | 27.93 | 160/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | PGE2 GEL | 140/90 | NIL | EMERGENCY LSCS | 3.562 | 9 | NO |
| 41 | 1 | KAVITHA | 24 | PRIMI | 40 | 148 | 66 | 30.13 | 150/100 | 2+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 2.969 | 8 | NO |
| 42 | 1 | GAYATHRI | 26 | PRIMI | 38 | 150 | 57 | 25.33 | 150/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY LSCS | 2.659 | 9 | NO |
| 43 | 1 | SHAHINA | 20 | PRIMI | 38 | 146 | 56 | 26.27 | 150/100 | 1+ | ALPHA METHYL DOPA | 140/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 2.106 | 8 | NO |
| 44 | 1 | PRIYA | 25 | PRIMI | 39 | 160 | 72 | 28.13 | 140/100 | 1+ | ALPHA METHYL DOPA | 130/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 2.352 | 7 | NO |
| 45 | 1 | VIJAYALAKSHMI | 31 | G2P1L1 | 38 | 155 | 66 | 27.47 | 150/100 | 1+ | ALPHA METHYL DOPA | 150/100 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY RPT LSCS | 3.265 | 8 | NO |
| 46 | 1 | HEMAVATHY | 22 | PRIMI | 34 | 149 | 70 | 31.53 | 170/110 | 3+ | ALPHA METHYL DOPA | 160/90 | NIL | NO | PGE2 GEL | 160/90 | NIL | EMERGENCY LSCS | 1.986 | 5 | YES |
| 47 | 1 | KARPAGAM | 25 | G2P1L1 | 39 | 165 | 84 | 30.85 | 150/110 | 3+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 3.456 | 9 | NO |
| 48 | 1 | SAIBUNISHA | 26 | G3P2L1 | 30 | 152 | 60 | 25.97 | 170/110 | 3+ | ALPHA METHYL DOPA | 160/110 | 3+ | YES | PGE2 GEL | 150/110 | 4+ | LABOUR NATURAL | 0.98 | 4 | YES |
| 49 | 1 | SUMATHY | 22 | PRIMI | | 149 | 65 | 29.28 | 150/100 | 2+ | ALPHA METHYL DOPA | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGGENCY LSCS | 3.111 | 9 | NO |
| 50 | 1 | KAMATCHI | 21 | PRIMI | 39 | 154 | 64 | 26.99 | 140/100 | 1+ | ALPHA METHYL DOPA | 130/90 | NIL | NO | NIL | 130/80 | NIL | LABOUR NATURAL | 3.155 | 9 | NO |

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| 51 | 2 | SUMATHY | 28 | PRIMI | 37 | 154 | 88 | 37.1 | 150/100 | 1+ | LABETALOL | 150/100 | 1+ | YES | NIL | 140/90 | NIL | ELECTIVE LSCS | 2.21 | 9 | NO |
| 52 | 2 | MANJULA | 33 | G3A2 | 37 | 160 | 56 | 21.87 | 150/100 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 150/90 | NIL | ELECTIVE LSCS | 3.5 | 9 | NO |
| 53 | 2 | SHANTHI | 32 | G2P1L0 | 38 | 155 | 53 | 22.06 | 140/100 | 2+ | LABETALOL | 140/90 | NIL | NO | NIL | 130/80 | NIL | EMERGENCY LSCS | 2.85 | 9 | NO |
| 54 | 2 | BEULA | 28 | G2P1L1 | 30 | 155 | 62 | 25.8 | 160/110 | 4+ | LABETALOL | 160/100 | 4+ | YES | PGE2 GEL | 160/100 | 3+ | LABOUR NATURAL | 1.214 | 3 | YES |
| 55 | 2 | VENNILA | 27 | G2P1L1 | 38 | 158 | 69 | 27.63 | 140/100 | 1+ | LABETALOL | 130/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 2.85 | 9 | NO |
| 56 | 2 | JAMEELA BANU | 22 | G2P1L1 | 38 | 166 | 63 | 22.86 | 150/100 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 150/90 | NIL | ELECTIVE RPT LSCS | 2.8 | 9 | NO |
| 57 | 2 | VIJAYALAXMI | 26 | PRIMI | 40 | 160 | 80 | 31.25 | 140/100 | 1+ | LABETALOL | 130/80 | NIL | NO | PGE2 GEL | 130/80 | NIL | EMERGENCY LSCS | 2.79 | 9 | NO |
| 58 | 2 | AMMU | 24 | PRIMI | 38 | 156 | 70 | 28.76 | 150/100 | 1+ | LABETALOL | 140/90 | NIL | NO | PGE2 GEL | 140/90 | NIL | LABOUR NATURAL | 2.355 | 9 | NO |
| 59 | 2 | JOTHI | 36 | G2P1L1 | 38 | 157 | 65 | 26.37 | 150/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 3.13 | 9 | NO |
| 60 | 2 | THENMOZHI | 29 | PRIMI | 39 | 147 | 84 | 38.87 | 150/100 | 1+ | LABETALOL | 140/90 | NIL | NO | PGE2 GEL | 130/90 | NIL | EMERGENCY LSCS | 3 | 9 | NO |
| 61 | 2 | VARALAXMI | 26 | PRIMI | 37 | 154 | 78 | 32.88 | 140/100 | 1+ | LABETALOL | 130/80 | NIL | NO | NIL | 120/80 | NIL | LABOUR NATURAL | 2.305 | 8 | NO |
| 62 | 2 | MADHUMITHA | 22 | PRIMI | 38 | 142 | 60 | 29.75 | 140/110 | 2+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 4.07 | 8 | NO |
| 63 | 2 | NEELAVATHY | 24 | G2A1 | 39 | 153 | 66 | 28.19 | 160/100 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 3.7 | 9 | NO |
| 64 | 2 | DHANALAXMI | 20 | PRIMI | 37 | 155 | 63 | 26.22 | 150/100 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 2.523 | 9 | NO |
| 65 | 2 | DEVI | 29 | G2P1L1 | 38 | 160 | 89 | 34.76 | 150/100 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 120/80 | NIL | LABOUR NATURAL | 3.325 | 9 | NO |
| 66 | 2 | KAMATCHI | 29 | G3P2L1 | 37 | 148 | 54 | 24.65 | 150/110 | 2+ | LABETALOL | 150/100 | 2+ | YES | PGE2 GEL | 140/100 | 1+ | EMERGENCY LSCS | 3.75 | 9 | NO |
| 67 | 2 | SAMUNDESHWARI | 19 | PRIMI | 38 | 155 | 55 | 22.89 | 140/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 120/80 | NIL | EMERGENCY LSCS | 3.78 | 9 | NO |
| 68 | 2 | INDARANI | 26 | G2P1L1 | 38 | 163 | 59 | 22.2 | 150/100 | 1+ | LABETALOL | 140/80 | NIL | NO | NIL | 140/80 | NIL | EMERGENCY RPT LSCS | 3.05 | 7 | NO |
| 69 | 2 | PITCHAIAMMAL | 22 | G2P1L0 | 38 | 154 | 68 | 28.67 | 140/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 130/90 | NIL | ELECTIVE RPT LSCS | 4.015 | 9 | NO |

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| 70 | 2 | SUMATHY | 36 | G3P1L1 A1 | 39 | 153 | 56 | 23.92 | 150/100 | 1+ | LABETALOL | 150/100 | NIL | YES | NIL | 140/90 | NIL | EMERGENCY RPT LSCS | 3.455 | 9 | NO |
| 71 | 2 | FAIZ JAHAN | 28 | G2P1L1 | 37 | 149 | 71 | 31.98 | 150/100 | 2+ | LABETALOL | 150/100 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY RPT LSCS | 3.37 | 9 | NO |
| 72 | 2 | KAVITHA | 24 | PRIMI | 40 | 159 | 66 | 26.1 | 150/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/80 | NIL | EMERGENCY LSCS | 3.625 | 9 | NO |
| 73 | 2 | REBECCA | 21 | PRIMI | 36 | 144 | 56 | 27 | 160/110 | 3+ | LABETALOL | 150/90 | NIL | NO | PGE2 GEL | 150/90 | NIL | LABOUR NATURAL | 2.78 | 9 | NO |
| 74 | 2 | SEETHA | 29 | G2P1L1 | 38 | 166 | 66 | 23.99 | 170/110 | 4+ | LABETALOL | 160/110 | 3+ | YES | PGE2 GEL | 160/100 | 2+ | EMERGENCY LSCS | 2.955 | 9 | NO |
| 75 | 2 | BHAVANI | 28 | PRIMI | 39 | 147 | 66 | 30.54 | 140/100 | 1+ | LABETALOL | 130/90 | NIL | NO | NIL | 120/80 | NIL | EMERGENCY LSCS | 3.22 | 9 | NO |
| 76 | 2 | CHITRA | 25 | PRIMI | 38 | 148 | 59 | 26.93 | 150/110 | 2+ | LABETALOL | 150/100 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY LSCS | 3.19 | 9 | NO |
| 77 | 2 | VANESHREE | 25 | G3P1L1 A1 | 38 | 167 | 68 | 24.38 | 150/110 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/80 | NIL | ELECTIVE RPT LSCS | 4.125 | 9 | NO |
| 78 | 2 | SABEERA BEGUM | 22 | PRIMI | 40 | 155 | 66 | 27.47 | 140/100 | 1+ | LABETALOL | 130/90 | NIL | NO | PGE2 GEL | 130/90 | NIL | EMERGENCY LSCS | 2.955 | 8 | NO |
| 79 | 2 | NITHYAVATHI | 29 | G2P1L1 | 38 | 164 | 64 | 23.79 | 150/100 | 2+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/80 | NIL | LABOUR NATURAL | 2.69 | 8 | NO |
| 80 | 2 | SASIKALA | 22 | G2P1L1 | 39 | 168 | 78 | 27.63 | 150/110 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 150/100 | NIL | LABOUR NATURAL | 3.27 | 9 | NO |
| 81 | 2 | ARIFA BEE | 24 | G2A1 | 39 | 157 | 60 | 24.34 | 140/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 3.711 | 9 | NO |
| 82 | 2 | KOTTEESWARI | 30 | PRIMI | 37 | 153 | 57 | 24.34 | 160/110 | 3+ | LABETALOL | 160/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY LSCS | 4 | 9 | NO |
| 83 | 2 | TAMILSELVI | 29 | G3P1L1 A1 | 38 | 165 | 79 | 29.02 | 140/100 | 1+ | LABETALOL | 140/80 | NIL | NO | NIL | 140/90 | NIL | ELECTIVE RPT LSCS | 3.25 | 9 | NO |
| 84 | 2 | SHANAZ BEGUM | 18 | PRIMI | 39 | 151 | 59 | 25.88 | 140/100 | 1+ | LABETALOL | 130/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 3.285 | 9 | NO |
| 85 | 2 | RAJESWARI | 27 | G2P1L1 | 37 | 151 | 71 | 31.14 | 160/100 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY RPT LSCS | 2.925 | 9 | NO |
| 86 | 2 | MANIMEGALAI | 36 | G4P1L1 A2 | 39 | 161 | 65 | 25.08 | 150/110 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 3.25 | 8 | NO |
| 87 | 2 | SAINA | 19 | PRIMI | 40 | 144 | 59 | 28.45 | 160/100 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 3.152 | 8 | NO |

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| 88 | 2 | PRABAVATHY | 24 | PRIMI | 37 | 152 | 73 | 31.6 | 160/100 | 2+ | LABETALOL | 150/90 | NIL | NO | PGE2 GEL | 150/90 | NIL | LABOUR NATURAL | 2.97 | 9 | NO |
| 89 | 2 | ALMAS | 21 | PRIMI | 38 | 160 | 70 | 27.34 | 160/100 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY LSCS | 3.845 | 9 | NO |
| 90 | 2 | KOKILA | 25 | PRIMI | 38 | 155 | 59 | 24.56 | 160/100 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 3.2 | 9 | NO |
| 91 | 2 | MEHERNISHA | 30 | G2P1L1 | 40 | 162 | 66 | 25.15 | 140/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 3.1 | 9 | NO |
| 92 | 2 | MAHALAKSHMI | 29 | G3P1L1 A1 | 38 | 152 | 60 | 25.97 | 160/100 | 1+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY RPT LSCS | 3.212 | 9 | NO |
| 93 | 2 | KALAIARASI | 29 | G2P1L1 | 39 | 164 | 65 | 24.17 | 140/100 | 2+ | LABETALOL | 140/80 | NIL | NO | NIL | 130/80 | NIL | LABOUR NATURAL | 2.563 | 9 | NO |
| 94 | 2 | SEETHA | 24 | PRIMI | 34 | 165 | 60 | 22.04 | 160/100 | 2+ | LABETALOL | 160/100 | 2+ | YES | NIL | 150/100 | 3+ | LABOUR NATURAL | 1.243 | 5 | YES |
| 95 | 2 | SAIRA BANU | 19 | PRIMI | 39 | 145 | 58 | 27.59 | 150/100 | 2+ | LABETALOL | 140/90 | NIL | NO | NIL | 130/90 | NIL | EMERGENCY LSCS | 2.856 | 9 | NO |
| 96 | 2 | SATYA | 23 | G2A1 | 38 | 153 | 68 | 29.05 | 140/100 | 1+ | LABETALOL | 130/90 | NIL | NO | NIL | 130/90 | NIL | LABOUR NATURAL | 2.658 | 9 | NO |
| 97 | 2 | SHAKIRA | 20 | G2A1 | 37 | 150 | 70 | 31.11 | 160/110 | 3+ | LABETALOL | 160/110 | 1+ | YES | NIL | 150/100 | NIL | EMERGENCY RPT LSCS | 3.985 | 9 | NO |
| 98 | 2 | PARVATHY | 26 | G2P1L1 | | 163 | 78 | 29.36 | 160/110 | 2+ | LABETALOL | 150/90 | NIL | NO | NIL | 140/90 | NIL | LABOUR NATURAL | 3.252 | 9 | NO |
| 99 | 2 | UMADEVI | 23 | G3P1L1 A1 | 39 | 159 | 64 | 25.32 | 150/100 | 1+ | LABETALOL | 140/90 | NIL | NO | NIL | 150/90 | NIL | EMERGENCY RPT LSCS | 3.673 | 9 | NO |
| 100 | 2 | RAHIMA | 24 | G2P1L1 | | 154 | 65 | 27.41 | 160/100 | 2+ | LABETALOL | 140/90 | NIL | NO | NIL | 140/90 | NIL | EMERGENCY LSCS | 2.986 | 9 | NO |