INDUCTION OF LABOUR VERSUS CONSERVATIVE MANAGEMENT FOR MILD GESTATIONAL HYPERTENSION AT TERM

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE RULES

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CERTIFICATE

This is to certify that the dissertation entitled "**Induction of labour versus conservative management for mild gestational hypertension at term**" is the original work of **Dr. Bhageerathy P.S** towards the M.S Branch II (Obstetrics and Gynaecology) Degree examination of The Tamil Nadu Dr.M.G.R Medical University, Chennai to be held in April 2013

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INTRODUCTION

Hypertensive disorders of pregnancy remain a leading cause of maternal and perinatal morbidity and mortality. Most of the time, it is a pregnancy specific disorder characterised by hypertension with or without proteinuria, sometimes progressing to multi- organ dysfunction with varying clinical features[1].The incidence of hypertensive disorders at term is high, though the incidence of maternal morbidity is low at this gestational age. But the incidence of maternal morbidity is higher remote from term, though the incidence of hypertensive disorders is low at this gestational age.

The term gestational hypertension was first described by Dr. Jack Pritchard for new onset uncomplicated hypertension during pregnancy. The Working Group[2] classifies hypertension complicating pregnancy into gestational hypertension, preeclampsia-eclampsia syndrome, preeclampsia superimposed on chronic hypertension and chronic hypertension which are described in detail later.

In Christian Medical College, Vellore, there are about 1000 to 1200 deliveries per month. Of these, we have about 25-45 patients (2-4 %) with gestational hypertension, 5-12 patients (0.5- 1 %) with mild pre eclampsia, 30-45 patients (2.5-4%) with severe pre eclampsia, 5-10 patients(0.5-0.9%) with eclampsia and 5-10 patients (0.5-0.9%) with chronic hypertension every month.

The definitive treatment for preeclampsia is delivery of the placenta, but the gestational age at delivery has an impact on perinatal outcome[3]. In these high risk women, the maternal and foetal interests are in conflict, with the best interests of the mother dictating delivery, but the best interests of the foetus without

compromise dictating prolongation of pregnancy. The health care providers have to balance the risks and the benefits for the mother and her baby before deciding on the timing for delivery.

The treatment for severe gestational hypertension and preeclampsia at term is induction of labour in most situations. But the management of mild hypertension without any complications at term is not clear. Induction of labour is thought to prevent progression of hypertension and its complications like eclampsia, HELLP syndrome, placental abruption , renal failure etc which can increase the maternal morbidity and even lead to mortality and also perinatal deaths. But induction of labour can increase the instrumental delivery and caesarean section rates which lead to additional morbidity and costs[4].

The advantage of conservative management is that there is less interference with the normal course of pregnancy and more chance of spontaneous onset of labour and normal delivery. Conservative management is only practised in case of mild gestational hypertension as the progression to severe hypertensive disease is less likely and complications are uncommon. There is a 10% risk of progression of hypertension and its complications like eclampsia, abruption, HELLP syndrome, renal failure and stillbirths[5]. Hence it should be practised only in centres with experienced staff and where close maternal and foetal surveillance is possible.

The aim of this study is to provide evidence as to whether induction of labour in women with mild gestational hypertension at term is effective in preventing maternal and foetal complications without increasing caesarean rates and expenditure when compared to conservative management.

AIMS AND OBJECTIVES

1. To study whether induction of labour in women with singleton pregnancy complicated by gestational hypertension at 37 weeks reduces maternal morbidity and mortality when compared to conservative management

2. To study whether this intervention increases instrumental delivery and caesarean section rates.

3. To study whether induction of labour in women with singleton pregnancy complicated by gestational hypertension at 37 weeks reduces neonatal morbidity and mortality when compared to conservative management
4. To study whether induction of labour reduces the expenditure when compared to conservative management as the costs for foetal surveillance and increased number of antenatal visits can be avoided.

REVIEW OF LITERATURE

Hypertensive disorders complicate 5-10 % of all pregnancies[6]. It accounts for 9.1 % and 16.1 % of maternal deaths in developing and developed countries respectively[7]. In Christian Medical College, Vellore, gestational hypertension alone accounts for 2-4 % of all pregnancies. It is found that about 10% of women with gestational hypertension at term progress to preeclampsia and its complications[5].

The high maternal morbidity and mortality associated with hypertensive disorders of pregnancy are due to its progression to severe preeclampsia and its complications like pulmonary oedema , thrombocytopenia , cerebral haemorrhage , hepatic failure , renal failure and abruptio placenta . The foetal and neonatal complications seen are intrauterine growth restriction , preterm birth , stillbirth , admission to NICU and neonatal death .

Classification:

There are different classifications used to categorise hypertension during pregnancy. Organisations like American College of Obstetrics and Gynaecology[8] , The Canadian Hypertensive Society[9] , and The Australian Society for study of Hypertension in Pregnancy[10] have published different diagnostic criteria for diagnosis of hypertension in pregnancy . The currently accepted classification was laid down by the Working Group of the National High Blood Pressure Education Programme 2000.[2]

This Working Group classifies hypertensive disorders of pregnancy into four groups.

1 . Gestational Hypertension

2 . Pre-eclampsia and eclampsia syndrome

3 . Pre-eclampsia syndrome superimposed on chronic hypertension

4 . Chronic hypertension

The significant changes that have been brought out by this group are the following:

1. The presence or absence of oedema is not considered in this classification
2. The rise in systolic and diastolic blood pressure by 30 and 15 mm of Hg from baseline during the course of pregnancy has been removed from the classification
3. Korotkoff sound phase IV which corresponds to its muffling has been replaced by Korotkoff sound phase V which corresponds to its disappearance to define diastolic blood pressure.[11]

Gestational Hypertension:

Systolic blood pressure more than or equal to 140 mm of Hg or diastolic blood pressure more than or equal to 90 mm of Hg without

proteinuria for the first time during pregnancy after 20 weeks of gestation is defined as gestational hypertension .

The blood pressure usually returns to normal by 12 weeks postpartum. If preeclampsia does not develop during pregnancy and blood pressure returns to normal by 12 weeks postpartum , gestational hypertension is then called transient hypertension[12] . Generally , the outcome of pregnancy in women with gestational hypertension is good.

Gestational hypertension is considered severe if there is elevation in systolic blood pressure to 160 mm of Hg or more or diastolic blood pressure to 110 mm of Hg or more.

Pre- eclampsia :

The minimum criteria for diagnosis of preeclampsia is the finding of systolic blood pressure more than or equal to 140 mm of Hg or diastolic blood pressure more than or equal to 90 mm of Hg with proteinuria of 300 mg or more in 24 hours or 1+ or more by the dipstick method[2] .

Preeclampsia is considered severe if the following criteria are present:

1. Systolic blood pressure more than or equal to 160 mm of Hg or diastolic blood pressure more than or equal to 110 mm of Hg.
2. Proteinuria of 3 g or more in 24 hours or 2+ or more by the dipstick method.
3. Serum creatinine more than 1.2 mg/dl or oliguria.

4. Platelet count less than 1,00,000/mm^3
5. Microangiopathic haemolysis resulting in raised LDH.
6. Elevated serum transaminases – SGOT , SGPT
7. Persistent headache or other cerebral or visual disturbance.
8. Persistent epigastric pain.
9. Pulmonary oedema.
10. Foetal growth restriction.

Contrary to the working group of the National High Blood Pressure Education Programme 2000, Sibai advocates that severe preeclampsia should be considered in the absence of proteinuria when gestational hypertension is associated with persistent cerebral symptoms , epigastric or right upper quadrant pain with nausea or vomiting or thrombocytopenia and abnormal liver enzymes[13]. According to Sibai, some women may have atypical preeclampsia with all aspects of the syndrome, but without hypertension or proteinuria or both[14].

Eclampsia:

The onset of convulsions in a women with pre eclampsia that cannot be attributed to other causes is defined as eclampsia. It is characterised by generalised tonic clonic seizures before , during or after labour.

Eclampsia occurs antepartum in 35-45 %, intrapartum in 15-20 % and postpartum in 35-45 % of cases. 10% of women develop eclampsia 48 hours after delivery.[15] 10% of eclampsia occurs without overt proteinuria[16] .

Pre clampsia superimposed on chronic hypertension :

It is defined as the following:

1. New onset proteinuria of 300 mg or more in 24 hours in hypertensive women with no proteinuria before 20 weeks of gestation.
2. A sudden increase in proteinuria or blood pressure or fall in platelet count less than 1,00,000/mm^3 in those women diagnosed with hypertension and proteinuria before 20 weeks of gestation[2] .

Superimposed preeclampsia usually develops earlier in pregnancy than "pure" preeclampsia. Superimposed disease has a tendency to become more severe and is usually associated with foetal growth restriction.

Chronic hypertension:

Systolic blood pressure more than or equal to 140 mm of Hg or diastolic blood pressure more than or equal to 90 mm of Hg before pregnancy or diagnosed before 20 weeks of gestation not attributable to gestational trophoblastic disease is defined as chronic hypertension . Hypertension first diagnosed after 20 weeks of gestation and persistent 12 weeks postpartum is also called chronic hypertension[2].

The incidence of chronic hypertension during pregnancy ranges from 1% to 5% . The risk factors for chronic hypertension are obesity , older age , diabetes mellitus and renal disease[2,17] . Pregnant women with chronic hypertension are at increased risk for superimposed preeclampsia and abruptioplacentae . There is increased perinatal morbidity and mortality due

to prematurity , intrauterine growth restriction , stillbirth , placental abruption and caesarean delivery.[14]

RISK FACTORS FOR PRE-ECLAMPSIA[19,20]:

Genetic factors:

Genetic pre-disposition

Race and ethnicity: more in blacks and Asians

Family history of pre-eclampsia (RR 2.9, 95% CI 1.7 – 4.93)

Pregnancy by ovum donation

Age and parity:

Teenage pregnancy

Age more than 40 years (RR 1.96, 95% CI 1.34 – 2.87)

Long interval between pregnancies

Nulliparity (RR 2.91, 95% CI 1.28 – 6.61)

Though the incidence of preeclampsia in multiparas is less than that of nulliparas, the risk for stillbirths was found to be more in hypertensive multiparas compared to nulliparas.[21]

Partner related factors:

Change of partner

Partner who fathered a pre-eclamptic pregnancy in another woman

Limited sperm exposure

Pregnancy due to donor insemination

Presence of underlying disorders:

Chronic hypertension

Diabetes Mellitus (RR 3.56, 95% CI 2.54 – 4.99)

Renal disease

Obesity (RR 2.47, 95% CI 1.66 – 3.67)

Maternal low birth weight

Polycystic ovarian syndrome

Migraine

Collagen vascular disorders

Uncontrolled hyperthyroidism

Factor V Leiden deficiency, activated protein C deficiency, thrombophilia

Sickle cell disease or trait, other haemoglobinopathies

Antiphospholipid antibodies (RR 9.72 , 95% CI 4.34 -21.75)

Protein-S deficiency and hyperhomocysteinaemia

Women with excessive snoring

Pregnancy related risk factors:

Multiple pregnancy

Hydatidiform mole (RR 2.93, 95% CI 2.04 -4.21)

Hydrops fetalis

Congenital and chromosomal fetal anomalies (trisomy 13, triploidy)

Urinary tract infection

Previous preterm birth

Miscellaneous factors:

Smoking (reduced risk)

Psychological strain and stress at work place

Previous history of preeclampsia (RR 7.19, 95% CI 5.85 - 8.83)

Raised blood pressure at booking (RR 1.38, 95% CI 1.01 – 1.87)

(Diastolic blood pressure more than 80 mm of Hg)

AETIOPATHOGENESIS:

The aetiopathogenesis of the disease is not well understood. However, research in this field is ever-progressing. A fetus is not a requisite for pre-eclampsia , but the presence of chorionic villi is essential. Various theories for the aetiopathogenesis of preeclampsia are based on the observation that gestational hypertensive disorders are more common in women who:

- Are exposed to chorionic villi for the first time
- Are exposed to large amounts of chorionic villi, as with multiple pregnancy or vesicular mole.
- Have prior cardiovascular or renal disease
- Are genetically prone to develop hypertension during pregnancy.

Recently, pre-eclampsia is thought to be a two stage disorder where stage 1 is preclinical and is due to faulty trophoblastic vascular remodelling of uterine arteries that cause hypoxia. Stage 2 is characterised by release of placental factors into maternal circulation causing systemic inflammatory response and endothelial activation leading to the clinical syndrome of pre-eclampsia.[22]

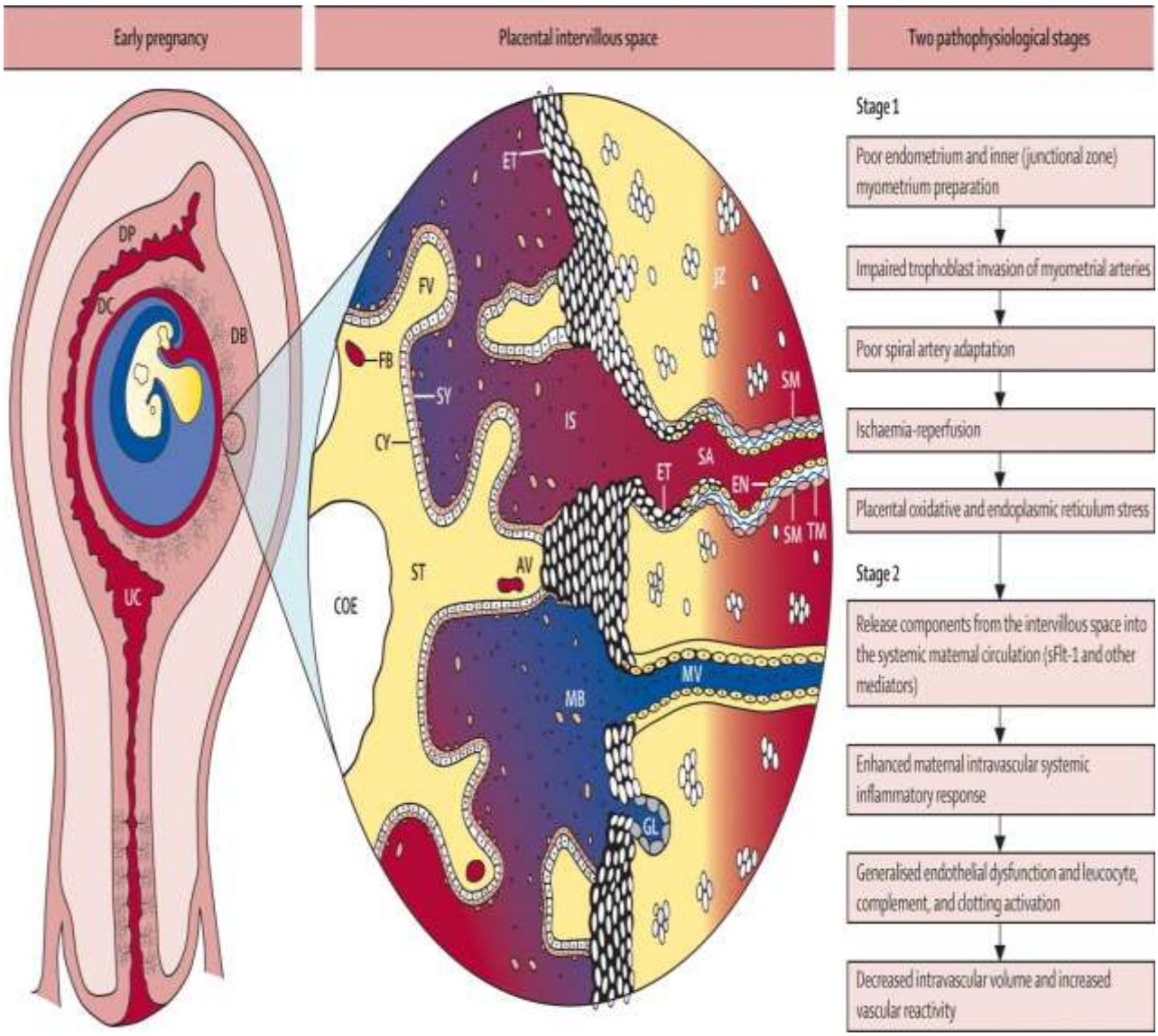


Figure1. Possible pathophysiological process in pre-eclampsia[1]

AV-anchoring villi, COE-coelomic cavity, CY-cytotrophoblast, DB-decidua basalis, DP-decidua parietalis, EN-endothelium,ET-extravillous trophoblast,FB-fetal blood vessel, FV-floating villus, GL-gland, IS-intervillous space, JZ-junctional zone myometrium, MB-maternal blood, MV-maternal vein, SA-spiral artery, SM-smooth muscle, ST-stroma, SY-syncytiotrophoblast, TM-tunica media, UC-uterine cavity. sFlt -1 – soluble form of the vascular endothelial growth factor receptor.

Pathogenesis:

Vasospasm:

The concept of vasospasm was introduced by Volhard (1918). This causes increased resistance leading to hypertension. The decreased blood flow because of vasoconstriction causes ischaemia of the surrounding structures leading to necrosis, haemorrhage and other end organ changes.

Endothelial cell activation:

Certain unknown factors of placental origin are released into maternal circulation which causes activation and dysfunction of the vascular endothelium.

Increased Pressor Response:

These women have increased vascular reactivity to vasopressors like angiotensin II and norepinephrine unlike normal pregnant women. The damaged endothelial cells produce less nitric oxide and secrete procoagulant substances and thereby increase the sensitivity to vasopressors.[23]

Prostaglandins:

The endothelial prostacyclin (PGI2) production is decreased and thromboxane A2 secretion by platelets is increased in women developing preeclampsia. Thus the prostacyclin: thromboxane ratio decreases leading to vasoconstriction.[24]

Nitric oxide:

Nitric oxide is a potent vasodilator synthesized by endothelial cells. In preeclamptics, its synthesis is reduced.

Endothelins:

Endothelin-1 which is a potent vasoconstrictor is secreted in excess by the damaged endothelial cells.[25]

Angiogenic imbalance:

Increased maternal antiangiogenic factors like sFlt-1 and soluble endoglins and decreased angiogenic factors like vascular endothelial growth factor and placental growth factor characterise this disease.

PATHOPHYSIOLOGY:

Cardiovascular system:

Hyperdynamic ventricular function is characteristic of preeclampsia

Blood volume:

Haemoconcentration is the hallmark of the disease

Blood and coagulation:

Thrombocytopenia and haemolysis are common. Coagulopathy occurs only if abruption supervenes.

Endocrine changes:

There are decreased levels of renin, angiotensin II, angiotensin 1-7 and aldosterone when compared to normal pregnancy, but still they remain above non-pregnant values.[26]Vasopressin levels are similar to that of normally pregnant women and secretion of atrial natriuretic peptide is increased.

Fluid and electrolyte change:

Extracellular fluid volume is increased in pregnant women with preeclampsia leading to pathological fluid retention. Electrolyte levels do not differ significantly compared to normal pregnant women.

Kidney:

The renal perfusion and glomerular filtration are reduced. Plasma uric acid and serum creatinine may be elevated. Urine sodium concentration is also elevated. Proteinuria may be present.

Anatomical changes:

In the kidney, glomerular capillary endotheliosis is the characteristic lesion. The endothelial cells are swollen and they block the capillary lumen. There are homogenous subendothelial deposits of proteins and fibrin like material. Sometimes, acute tubular necrosis develops when there is associated haemorrhagic hypotension. Rarely, irreversible renal cortical necrosis develops.

In the liver, periportal haemorrhages and some degree of hepatic infarction may be seen.

In the brain, cortical and subcortical haemorrhages, cerebral oedema, multiple non-haemorrhagic areas of softening, fibrinoid necrosis of the arterial wall and

perivascular microinfarcts may be seen. The most characteristic changes are described recently as posterior reversible encephalopathy syndrome (PRES).[27]

Uteroplacental perfusion is decreased leading to fetal growth restriction and increased perinatal morbidity and mortality. The measurement of uterine artery blood flow using Doppler velocimetry will provide information regarding the diminished uteroplacental perfusion.

NATURAL COURSE OF HYPERTENSION:

Patric Saudan et al did a retrospective analysis and a prospective study on women with gestational hypertension to determine the likelihood of progression from gestational hypertension (GH) to pre-eclampsia(PE)[5].In the retrospective analysis of 416 women who initially presented with GH,15% progressed to PE. In the prospective study of 112 women who initially presented with GH, 26% progressed to PE, with an overall progression of 17%. Approximately, 15-25% of women initially diagnosed with GH developed PE and this is more likely with earlier presentation. Women with gestational hypertension diagnosed after 36 weeks had only about 10% risk of progressing to PE.

Barton et al (2001) studied 748 women with mild gestational hypertension with singleton pregnancy between 24-35 weeks gestation to find out the progression to pre-eclampsia[28]. Antepartum progression to severe preeclampsia occurred in 9.6%. They found that the development of proteinuria was associated with an earlier gestational age at delivery and lower birth weight.

Barton et al (1997) compared maternal and perinatal outcomes of 379 elderly gravidas (more than or equal to 35 years) with that of younger women with mild gestational hypertension .The mean gestational age at delivery, the mean pregnancy prolongation and the mean birth weights were similar in both groups ($p > 0.05$). Though there were no differences in complications like abruption, thrombocytopenia and HELLP syndrome, there were 5 stillbirths among elderly gravidas (p 0.063) which was not statistically significant.[29]

Barton et al (1995) compared maternal and perinatal outcomes of 60 teenage pregnancies with 120 adult controls with mild gestational hypertension remote from term. They found that the mean gestational age at delivery, the mean pregnancy prolongation and mean birth weights were not statistically different (p more than 0.05) and there were no stillbirths, neonatal deaths or eclampsia in either group.[30]

Barton et al evaluated the influence of ethnicity on outcome in a prospective analysis of 1182 patients with gestational hypertension and mild preeclampsia of Caucasian, Hispanic and African American ethnicity. Hispanics had a higher rate of progression to severe preeclampsia and intrauterine growth restriction when compared to Caucasians (p less than 0.05).The rates of progression to HELLP and eclampsia were similar among all groups. African Americans demonstrated lower gestational age at delivery, lower birth weights and higher stillbirth and neonatal death rate.[31]

Intrauterine growth restriction (IUGR) of variable severity is a known foetal complication of hypertensive disorders of pregnancy. Monitoring the patients with IUGR with biweekly non stress test, Biophysical profile and Doppler velocimetry of

foetal umbilical and middle cerebral arteries is important to prevent perinatal deaths and to decide on timing and mode of delivery.

A population based retrospective study in 2004 of 16,000 women with gestational hypertension, severe pre-eclampsia and women with normal blood pressure was done to find out the impact of hypertension on birth weight. The difference in mean birth weight (132.2 gm-174.6 gm) between normotensive women and pre-eclamptic women was not statistically significant. There were no differences in mean birth weights between women with gestational hypertension and those with normal blood pressure.[32]

Xiongx et al (2002), in a retrospective observational study of 97,000 women showed that the differences in mean birth weight between mothers with severe pre-eclampsia and those with normal blood pressure ranged from -54.5 g to 239.5 g for the gestational age of less than or equal to 32 weeks. The birth weights were lower among mothers with pre-eclampsia who delivered at less than 37 weeks. But at term, foetal growth of mothers with preeclampsia was similar to that of babies born to normotensive mothers.[33]

Lau T.K (2005), in a comparative study of 35,000 singleton pregnancies to investigate the impact of hypertensive disorders of pregnancy at term found that the incidence of small for gestational age (SGA) babies (<10^{th} centile) was significantly higher in subjects with pre-eclampsia and eclampsia than the control group. In contrast, there was no significant difference in the incidence of SGA babies in gestational hypertensives when compared to normotensives.[34]

PREDICTION OF PREECLAMPSIA

Currently, there are no screening tests that are reliable, valid and economical. Measurement of various biological, biochemical and biophysical markers implicated in the pathophysiology of preeclampsia have been proposed for predicting the development of preeclampsia.

1. Placental perfusion / vascular resistance related tests:

Provocative pressor tests

They evaluate an increase in blood pressure in response to a stimulus. The roll over test, isometric exercise test and angiotensin II infusion test performed between 28 to 32 weeks are examples. Conde- Agudelo etal (2009) found that all these tests have sensitivities ranging from 55 to 70 percent and specificities of 85%[35].

Uterine artery Doppler velocimetry

The detection of a high resistance index along with persistence of an early diastolic notch is used for predicting preeclampsia. Cooper and Campell studied 977 unselected women between 16 to 24 weeks of gestation and found that in the prediction of preeclampsia, the sensitivity of the method was 25 %, the specificity was 95 %, the positive predictive value was 20% and the negative predictive value was 96%[36].

2. Foetal placental unit endocrine dysfunction

Many serum markers like human chorionic gonadotropin, alpha fetoprotein, estriol, pregnancy associated protein A, inhibin A, activin A, placental protein 13 and corticotropin releasing hormone have been proposed to help predict preeclampsia,

but none of these tests is found to be clinically beneficial for hypertension prediction[35].

3. Renal dysfunction related tests

Serum uric acid :

Hyperuricaemia is one of the earliest laboratory manifestations of preeclampsia. Cnossen and associates (2006) reported a sensitivity of 0 to 55 per cent and specificity of 77 to 95 percent[37].

Microalbuminuria :

Conde-Agudelo and associates (2009) reported a sensitivity of 7 to 90 per cent and specificity of 29 to 97 per cent for this test[35].

4. Endothelial dysfunction and oxidant stress related tests

Fibronectins

These high molecular weight glycoproteins are released from endothelial cells and extracellular matrix following endothelial injury. Leeflang and associates (2007) did not find this test clinically useful to predict preeclampsia following their systematic review[38].

Coagulation activation

Although increase in markers of coagulation activation and thrombocytopenia were analysed in several studies for predicting preeclampsia, the overlap with levels in normotensive pregnant women prevents their predictive use[35].

Oxidative stress

Increased levels of lipid peroxides like malondialdehyde along with decreased antioxidant activity have been studied as markers for predicting preeclampsia. Hyperhomocysteinemia at midpregnancy had a three to four fold risk of

preeclampsia, but these tests are not shown to be clinically useful predictors for preeclampsia [39].

Angiogenic factors

Increase in serum levels of proangiogenic factors like vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) and decrease in levels of antiangiogenic factors like soluble fms-like tyrosine kinase 1 (sFlt-1) and soluble endoglin (s Eng) may serve as predictors for preeclampsia. Though the preliminary results suggest a clinical role for preeclampsia prediction[35], their clinical use is not currently recommended until further studies provide better evidence. A multicentre trial enrolling 12,000 women is being carried out since 2008 by the World Health Organisation to evaluate these factors.

5. Miscellaneous

Free foetal DNA

Holtzgrave and associates (1998) reported that in pregnancies complicated by preeclampsia, foetal-maternal cell trafficking is increased[40]. These free foetal DNA can be detected in maternal plasma using polymerase chain reaction. Conde - Agudalo and associates (2009) reported that free foetal DNA detection and quantification was not useful in preeclampsia prediction[35].

MANAGEMENT:

Pregnancy complicated by gestational hypertension is managed depending upon the severity, gestational age and presence of pre-eclampsia. Given the explosive nature of the disease, both the American College of Obsteticians and Gynecologists (2002a)[41] and the National High Blood Pressure Education Program (NHBPEP) [2] Working Group (2000) recommend more frequent antenatal check-ups, even if pre-eclampsia is only suspected. Increased surveillance allows early recognition of ominous changes in blood pressure, critical laboratory findings and development of clinical signs and symptoms.

According to Friedman and Lindheimer, at present there are no screening tests for predicting preeclampsia that are reliable, valid and economical.[42] Various investigations are available to help diagnose, grade the severity and help in early detection of complications in hypertensive disorders of pregnancy, though none of them can replace good clinical acumen. Lab values are usually unrevealing in cases of gestational hypertension and mild preeclampsia, but may be abnormal in severe disease.

Hospitalisation versus Outpatient Management:

Non-proteinuric hypertension can be monitored and managed in 3 different settings -hospital inpatient, hospital day-care and domiciliary. Several studies have compared these management settings in terms of both clinical and cost-effectiveness.

In a comparative study to evaluate the efficiency of day-care in managing hypertension of pregnancy when compared to in-patient care with prior domiciliary visits, Twaddle and Harper concluded that for most women with non-proteinuric pregnancy hypertension, day-care is the most cost-effective management setting though there is no significant difference in any of the pregnancy outcomes studied.[43]

In a pilot study from Parkland hospital, Horsager et al (1995) randomly assigned 72 nulliparas with gestational hypertension from 27-37 weeks either to continued hospitalisation or to outpatient care. Outpatient management included daily blood pressure monitoring and weight and urine spot protein measurement thrice weekly. A home health nurse visited twice weekly and women were seen twice weekly in clinic. Though perinatal outcomes were similar, women in the home care group developed severe preeclampsia significantly more frequently than hospitalised women (42 versus 25%).[44]

Turnbull and associates (2004) enrolled 395 gestational hypertensives randomly to either day-care or inpatient management. Though there were no neonatal deaths, eclampsia or HELLP syndrome in both groups, general satisfaction favoured day-care. But, costs for either groups were not statistically different.[45]

The basic management goals for any pregnancy complicated with preeclampsia are:

1. Termination of pregnancy with the least possible trauma to mother and baby
2. Delivery of an infant who subsequently thrives
3. Restoration of health to the mother

In most women with preeclampsia, especially those at or near term, all 3 goals are served equally well by induction of labour.

The treatment for severe hypertension is clear. The only definitive treatment is delivery. The management of mild gestational hypertension without any complications at term is not uniform. Induction of labour is thought to prevent progression of hypertension and its complications such as eclampsia, HELLP syndrome, placental abruption, maternal death and foetal distress. Conversely, induction might increase the risk of instrumental delivery and caesarean section and thereby generate additional morbidity and costs.[4]

The HYPITAT trial (induction of labour vs expectant monitoring for gestational hypertension or mild pre eclampsia after 36 weeks) proved that induction of labour is associated with improved maternal outcome and should be advised for women with mild hypertensive disease beyond 37 weeks. 756 patients were allocated to receive induction of labour (n=377 patients) or expectant monitoring (n=379).Of women who were randomized, 117 (31%) allocated to induction of labour developed poor maternal outcome compared with 166 (44%) allocated to expectant monitoring (relative risk 0·71, 95% CI 0·59–0·86, p<0·0001). No cases of maternal or neonatal death or eclampsia were recorded. Furthermore, induction of labour was not associated with increased rates of caesarean delivery or neonatal morbidity.[4, 46]

Donna D Johnson, in a review article commented that one valid concern in HYPITAT trial was to include women at 36 weeks gestation[47]. Moreover, in subgroup analysis, composite maternal morbidity was not found to be improved by labour induction at gestational ages between 36 and 37 weeks.

Tajik and associates (2012) did an exploratory analysis of HYPITAT trial to find out if cervical favourability plays a role in deciding on induction of labour in gestational hypertension and mild preeclampsia[48]. They found that the superiority of induction of labour in preventing complications in women with gestational hypertension and mild preeclampsia at term varied significantly according to cervical favourability. Among women in expectant management arm, the longer the cervix, the higher the risk of developing maternal high risk situations whereas among women in induction arm, cervical length was not associated with an increased risk of developing maternal high risk situations (p=0.03). The beneficial effects of induction of labour in reducing the caesarean section rate were found to be stronger in women with unfavourable cervix.

An economic analysis of induction of labour and expectant monitoring in women with gestational hypertension or pre eclampsia at term showed that induction of labour was 11% cheaper than expectant management[49]. Though HYPITAT has not determined optimum management, yet the UK national institute for health and clinical excellence is using these data to determine clinical practice.[50]

vaan der Tuuk et al analysed the women in the expectant arm of HYPITAT trial to evaluate the predictors of progression to high risk situations in women with gestational hypertension and mild preeclampsia at term[51]. They found that a distinction can be made between women with low risk and women with high risk for progression of the disease. Women who progressed to have complications had younger gestational age at the start of expectant monitoring (p=0.004), were more frequently of non-Caucasian ethnicity (p=0.02), had more severe proteinuria at dipstick (p=0.03), a lower Bishop score at vaginal examination (p=0.02) and had higher systolic (p<0.001) and diastolic (p=0.02) blood pressure at study entry. The

laboratory findings that predicted progression of the disease were lower haemoglobin (p=0.03), lower haematocrit (p=0.04), lower platelet counts (p=0.02) and higher creatinine (p=0.03).

vaan der Tuuk et al (2011) studied the impact of the HYPITAT trial on doctors' behaviour and prevalence of eclampsia in Netherlands[52]. The HYPITAT trial was between October 2005 and March 2008. They identified 43641 women with gestational hypertension and mild preeclampsia beyond 36 weeks before, during and after the trial from Perinatal Registry. There was an increase in induction of labour from 58.3 to 67.1% ($p<0.001$) and decrease in prevalence of eclampsia from 0.85 to 0.19% ($p< 0.001$) before and after the trial.

HYPITAT-II study is on-going in Netherlands where women with gestational hypertension and mild preeclampsia at gestational ages between 34 and 36.6 weeks are randomised to either induction of labour or expectant monitoring. This trial will provide evidence as to whether induction of labour in women with gestational hypertension and mild preeclampsia in late preterm gestations is effective in preventing severe maternal complications without increasing neonatal morbidity.[53]

Schutte et al (2008) audited 27 cases of maternal deaths due to hypertensive disorders of pregnancy from 2000 to 2004 which were reported to Dutch Maternal Mortality Committee and found that in 96% of cases substandard care factors were present. The most frequent substandard care factor at community midwifery level was not testing for urine protein when indicated (41%). Substandard care in hospital settings included insufficient diagnostic testing when indicated (41%), insufficient management of hypertension by obstetricians (85%) , not using magnesium sulphate (67%), inadequate stabilisation before transfer to tertiary care centres(52%) and

failure to consider timely delivery (44%).They recommended education of pregnant women regarding danger signs, proper training of midwives and obstetricians and timely delivery to prevent maternal deaths in hypertensive disorders of pregnancy.[54]

Antihypertensive Therapy for Mild to Moderate Hypertension

The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy suggests antihypertensive therapy when systolic blood pressure is more than or equal to 160 mm of Hg or diastolic blood pressure is more than or equal to 100 mm of Hg[2]. It is believed that severe hypertension (diastolic blood pressure more than or equal to 110 mm of Hg) requires treatment to prevent cardiovascular accident or target organ damage. There is no evidence that antihypertensive therapy is beneficial in mild hypertension, except for a reduction in the rate of progression to severe hypertension. Antihypertensives reduce the risk of developing severe hypertension by half.[55]

Sibai and colleagues (1987a) compared the effectiveness of labetalol and hospitalisation when compared to hospitalisation alone in 200 primigravidas with gestational hypertension between 26 and 35 weeks. No difference was found in terms of mean pregnancy prolongation, gestational age at delivery, caesarean section rates or NICU admissions. But, growth restricted infants were significantly twice as frequent in labetalol arm-19 versus 9 %[56]

Abalos and associates (2007) reviewed 46 randomised trials of active antihypertensive therapy compared with either no treatment or placebo in 4282 women with mild to moderate hypertension. Except for a halving of risk for

developing severe hypertension, there were no benefits from antihypertensive therapy. Furthermore, the expected reduction in clinical outcomes (caesarean sections, preterm delivery, cardiovascular accidents) following a reduction in severe hypertension was not evident. They did not find foetal growth restriction in the treated group.[57]

The Cochrane Review concludes that no single class of antihypertensive agent is better than the other[58]. Prior to initiating therapy, one needs to individualise the drug of choice based on obstetrician's experience, the patient's medical profile and in some situations, patient preference.

The on-going CHIPS trial (Control of Hypertension In Pregnancy Study) will determine the effects of 'less tight' versus 'tight' control of non-severe hypertension on serious maternal and perinatal outcomes.[59]

Prevention of eclampsia

Preeclampsia complicated by generalised tonic clonic convulsions is a rare but serious complication which can be decreased by anticonvulsant therapy. Results from various studies have unequivocally shown that the use of magnesium sulphate for 24 hours is the single most effective anticonvulsant, more than halving the risk of eclampsia when compared to placebo, other anticonvulsants or no therapy (RR 0.41, 95% CI 0.29 to 0.58) [60]. Mild side effects like flushing were common with magnesium sulphate (24%) and no difference in the risk of stillbirth or neonatal death was found (RR 1.04, 95% CI 0.93 to 1.15).

The largest comparative study was the MAGPIE[61] (Magnesium sulphate for prevention of Eclampsia) trial. The NNT to prevent a case of eclampsia varied between the different subgroups within the Magpie trial: 109 (95% CI 72 to 225) for women with mild preeclampsia, 91 (95% CI 63 to 143) for the whole study population, 63 (95%CI 38 to 181) for women with severe preeclampsia and 36 (95% CI 21 to 125) for women thought to have imminent eclampsia. Hence, prophylactic treatment with magnesium sulphate to prevent seizures is indicated in severe preeclampsia and imminent eclampsia, but not necessarily in mild preeclampsia.

Timing of delivery

Delivery of the placenta is the ultimate cure for preeclampsia, though some women may worsen in the immediate postpartum period[62]. In pre-eclamptic women, the foetal and maternal interests are in conflict, with the best interests of mother dictating delivery, but the best interests of the foetus without compromise dictating prolongation of pregnancy. The timing of delivery depends on the balance of the estimated risks of temporising management and immediate delivery.[3]

Two contrasting management approaches are available for preeclampsia: interventionist approach of stabilisation and delivery and temporising approach. Although temporising management was previously termed as ‘expectant management’, this is a misnomer as ‘expectant’ suggests a passive attitude from maternity care givers. Actually, temporising management is active involving close monitoring of the mother and the baby to decide on timing of delivery to prevent complications. Whatever is the approach, these high risk pregnancies should be

managed in tertiary care centres with relevant maternal experience and appropriate neonatal intensive facilities.

Management depending on gestational age:

Gestational age before 24 weeks:

Considering the maternal risks and lack of obvious perinatal benefits, temporising management is not recommended. Termination of pregnancy after counselling is the treatment option.

Gestational age between 24 and 34 weeks:

Careful selection of patients eligible for temporising management should be done. Delivery is indicated when there are maternal end organ complications and doubts about foetal wellbeing. With increasing gestational age, the willingness to run maternal risks decreases and delivering the patient after stabilisation (antihypertensives, steroids for foetal lung maturity as indicated) increases.

Gestational age after 34 weeks:

Considering the good perinatal outcome in this group, interventionist management is preferred. The ongoing HYPITAT-II trial will throw light on the management of late preterm gestations. Delivery is preferred at term after the HYPITAT trial [4].

Mode of delivery

Vaginal delivery is a viable option at term if maternal and foetal conditions allow it, although caesarean rates are nonetheless high (14-19%) [46]. The rates of successful vaginal delivery are about 69% between 32 and 34 weeks and 48% between 28 and 32 weeks. Vaginal delivery with a viable baby is rarely achieved at less than 28 weeks (7%).[63]

Counselling for future pregnancies

Women who have had hypertensive disorders of pregnancy are at increased risk for hypertensive or metabolic complications in future pregnancies. Hjartardottir and associates (2006) studied 511 Icelandic women with gestational hypertension during their first pregnancy and found a 70% recurrence risk for hypertension in the second pregnancy of which 5% had preeclampsia and 16% had chronic hypertension.[64]

Diets low in energy or salt, supplementation of antioxidants ,Vitamin C or E, fish oil, garlic, zinc, selenium, folic acid or magnesium are all found to be ineffective in preventing preeclampsia[65,66,67]. No evidence was found between calcium supplementation and risk reduction of preeclampsia, although supplementation may have some benefits in calcium deficient group[68]. Progesterone, diuretics and antihypertensives have not been found to be useful in reducing the risk of preeclampsia.

Low dose aspirin prophylaxis is thought to correct an imbalance in the ratio of thromboxane A_2 to prostacyclin that is associated with increased vasoreactivity. A meta-analysis from 31 randomised trials showed that aspirin was associated with a

10% reduction in preeclampsia and prematurity and it was found to be safe also[69]. Hence, low dose aspirin can be offered on an individual basis depending upon the women's risk profile.

According to WHO criteria by Wilson and Jungner [70], routine screening of women who had preeclampsia and treatment if they were positive for thrombophilia is not justified, unless in a research setting. Thrombophilia work-up is recommended for those with a personal or family history of thrombosis.

Long term sequelae

Several long term studies have proven that any hypertension during pregnancy is a marker for later cardiovascular morbidity and mortality.

Arnadottir and co-workers (2005) followed up for 50 years, 325 women from Iceland who had hypertension in pregnancy from 1931 to 1947 and found that 60% of hypertensive women compared to only 53% of controls had died. The prevalences among hypertensives and normotensives of ischemic heart disease were 24 versus 15% and that of stroke was 9.5 versus 6.5%.[71]

Lykke and associates (2009a) followed up 780000 nulliparous women who had hypertension complicating pregnancy for 15 years[72]. The incidence of chronic hypertension was 5.2 fold in those with gestational hypertension, 3.5 fold after mild preeclampsia and 6.4 fold after severe preeclampsia. They also reported a significant 3.5 fold increased risk for type 2 diabetes mellitus.

MATERIALS AND METHODS

Study Design: Randomised controlled trial

Intervention and Comparator agent : Induction of labour and Conservative monitoring of mild gestational hypertension at 37 weeks .

Inclusion Criteria:

- Age 18-35 years
- Singleton pregnancy
- Cephalic presentation
- Gestational age 37 – 40 weeks
- Gestational hypertension (Systolic BP between 140 and 159 , Diastolic BP between 90 and 100)
- Urine albumin – trace or nil by dipstick method
- Intact membranes

Exclusion Criteria:

- Pre-eclampsia , Severe Gestational hypertension
- Chronic hypertension , Patients on anti- hypertensives
- Gestational Diabetes Mellitus , Renal disease , Heart disease
- Previous LSCS
- Suspected IUGR (< 2.5 Kg)
- Suspected Fetal distress (AFI < 8 , Non-reassuring Non stress test)
- Foetal anomalies

Method of randomization:

Computer generated randomization codes using the software RALLOC

Method of allocation concealment:

Sealed opaque envelopes

Blinding and masking:

Masking of intervention allocation is not possible

Informed Consent:

Written informed consent was taken in the patient's language before recruiting the patients. The consent form and the patient information sheet are attached as Appendix. Institutional Review Board approval was obtained prior to commencing the study. The study was reviewed in detail by the research and ethics committee and reported as safe to be conducted in pregnant women , provided the mother and the foetus are carefully monitored.

Methods:

Eligible patients presenting to the Obstetric outpatient department (OPD) or labour room of Christian Medical College, Vellore between 37 and 40 weeks from September 2011 to October 2012 with gestational hypertension ,that is, systolic blood pressure of 140 to 159 mm of Hg and diastolic blood pressure of 90 to 100 mm Hg (Korotkoff Phase V) recorded at least twice , four hours apart with nil or trace proteinuria by the dipstick method were included in the study . Ultrasound scan was done to rule out foetal growth restriction or any foetal compromise . After taking a written informed consent , eligible women

were randomized in a 1:1 ratio to receive induction of labour or conservative management.

For those in the induction arm , a per vaginal examination was done to assess the Bishop's score (for favourability of cervix) . If the score was more than or equal to 6, artificial rupture of membranes with or without oxytocin augmentation was done within 12 hours of randomisation . If the score was less than 6 , cervical ripening was done with PGE1 (25 mcg 6^{th} hourly 2 doses) as is the routine for induction of labour in our hospital . A 3^{rd} dose was used if the cervix was still unfavourable following the existing protocol . If cervix was unfavourable after 3 doses , patient may be re-induced after 2-3 days as the situation warrants or as per unit protocol .

For those allocated to the conservative management arm , Pregnancy induced hypertension (PIH) work up which includes platelet count , Serum creatinine, Serum transaminases (SGOT , SGPT),Lactate dehydrogenase(LDH) and blood picture was done . They were advised daily home BP monitoring by a local doctor or nurse who recorded it. Biweekly visits to the outpatient department was advised until they went into spontaneous labour or till 39 weeks and 5 days or till they developed any progression of hypertension . Induction of labour was done if diastolic BP became more than 100 mm Hg , urine albumin became more than or equal to 1+ by dipstick method , if the patient developed signs and symptoms of impending eclampsia , suspected fetal distress , eclampsia or HELLP syndrome or 39 weeks and 5 days as per the induction protocol.

Details of delivery , drugs used , intrapartum and postnatal complications for the mother and the baby were noted .

The primary outcome measured for the mother were maternal mortality and composite maternal morbidity (pre eclampsia , eclampsia HELLP syndrome , pulmonary oedema , renal failure , thromboembolic disease , abruption , need for ICU care and major postpartum haemorrhage) .The primary outcome measured for the baby was perinatal mortality .

The secondary outcomes measured for the mother were the mode of delivery (normal / instrumental / caesarean) , cost analysis , need for anti convulsant , need for anti hypertensives (intra partum / post natal) .The secondary outcomes measured for the baby were neonatal morbidity (5 minute APGAR score less than 7 , cord pH less than 7, admission to neonatal intensive care unit) .

The cost analysis was done only for the direct medical costs. This was done by adding the consultation charges for each OPD visit and the charges for the tests for maternal well-being (PIH work up) and the tests for foetal well-being (Modified Biophysical Score) along with the inpatient medical bill of the mother and the baby for patients on conservative management and the final medical inpatient bill of the mother and the baby at discharge for the patients in the induction group .

Sample Calculation:

Sample Size : 342 (171 in each arm)

Sample size was calculated according to the following formula:

$$N = \frac{Z_{1-\alpha/2}\sqrt{2p(1-p)} + Z_{1-\beta}\sqrt{p_1(1-p_1) + p_2(1-p_2)}}{(p_1-p_2)^2}$$

Error $\alpha = 5\%$

Power $\beta = 80\%$ $\quad p = \frac{p_1+p_2}{2}$

$p_1 = 0.31$

$p_2 = 0.44$

Hypothesis testing for 2 large proportions $H_1 : p_1 < p_2$

Statistical Analysis :

Chi square test was used for test of categorical variables. Student's t test was used for test of maternal morbidity and comparison of continuous variables . Treatment effect is presented as relative risk with 95 % confidence interval. Stratified analysis was done using logistic regression , presenting as odd's ratio for primary outcome .

A p value of less than 0.05 indicates statistical significance.

RESULTS

The calculated sample size was 342 (171 in each arm). But only 100 eligible patients could be recruited in the study (49 patients in the induction arm and 51 patients in the conservative management arm) within a period of 14 months from September 2011 to October 2012. Hence the statistical analysis was done only on 100 patients.

One patient in the induction arm whose Bishop score was unfavourable after 2 doses of misoprostol was discharged and was planned for re induction after 3 days. She was lost to follow up for 2 weeks and was re induced at 39 weeks and 6 days. She was analysed in the induction arm as she was induced once at 37 weeks with the intention to treat.

Some of the baseline characteristics taken at the trial entry were tabulated below:

Table: 1.BASELINE CHARACTERICTICS IN THE INDUCTION GROUP

Induction Group	N	Mean (SD)	Minimum	Maximum
Gestational age at trial entry	49	38.07 (0.82)	37.0	39.4
Maternal Age	49	25.37 (3.91)	19	35
Systolic BP at trial entry	49	143.67 (4.78)	140	156
Diastolic BP at trial entry	49	92.53 (3.82)	90	100

Table: 2 BASELINE CHARACTERISTICS IN THE CONSERVATIVE MANAGEMENT GROUP

Conservative management group	**N**	**Mean (SD)**	**Minimum**	**Maximum**
Gestational age at trial entry	51	37.97 (0.65)	37.0	39.2
Maternal Age	51	25.90 (3.44)	19	32
Systolic BP at trial entry	51	141.96 (3.63)	140	150
Diastolic BP at trial entry	51	91.69 (3.54)	90	100

The average age in both groups was about 25. The gestational age at randomisation was on an average 38 weeks in both arms. The average systolic and diastolic blood pressures also matched to about 142 mm of Hg and 92 mm of Hg respectively in both groups.

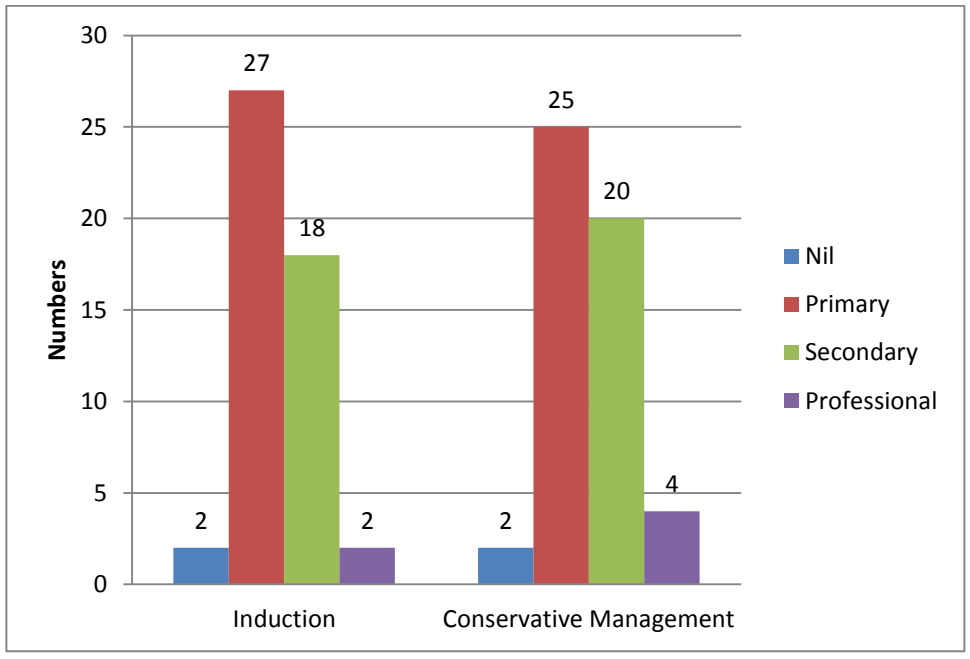
The next baseline characteristic that was analysed was the level of education among the patients in both groups which is tabulated below.

Table 3.

LEVEL OF EDUCATION

Education	Induction group		Conservative Management group		p – Value
	N	%	N	%	
Nil	2	4.1	2	3.9	0.847
Primary	27	55.1	25	49.0	
Secondary	18	36.7	20	39.2	
Professional	2	4.1	4	7.8	

Graph 1. Level of education



The baseline education in both groups was similar.

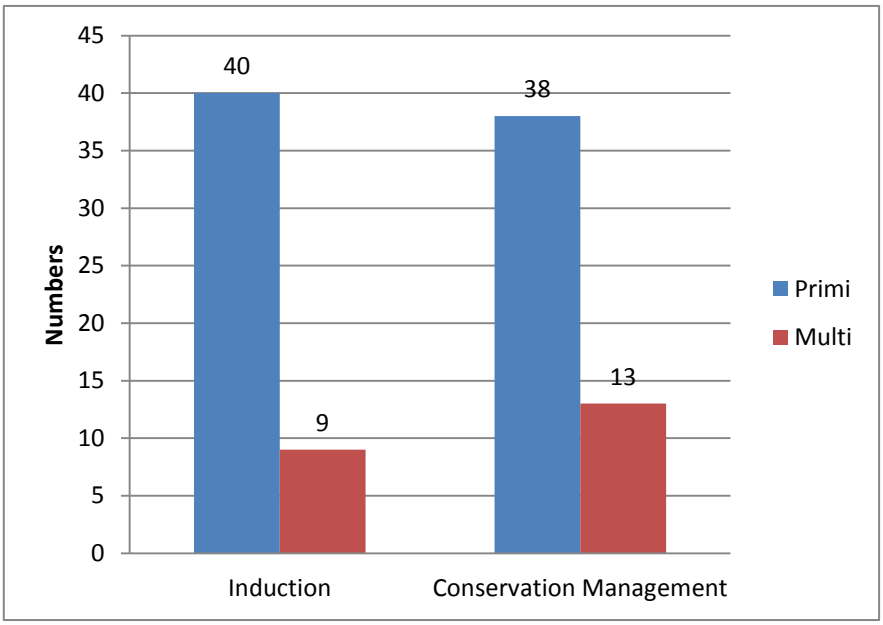
The next baseline characteristic that was analysed was obstetric score among the 2 groups:

Table 4.

OBSTETRIC SCORE

Obstetric Score	Induction group		Conservative Management group		p - Value
	N	%	n	%	
Primi	40	81.6	38	74.5	0.472
Multi	9	18.4	13	25.5	

Graph 2 Obstetric score:



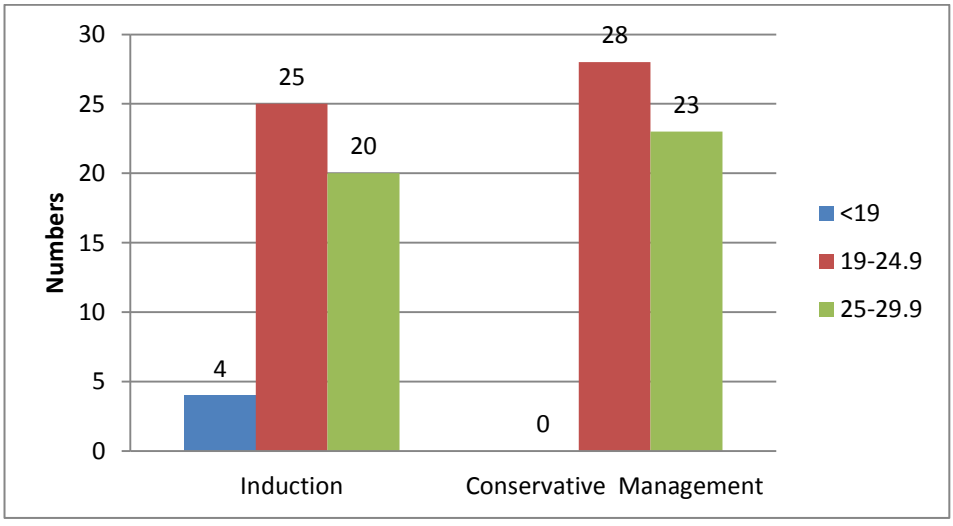
There were 40 primigravidas and 9 multigravidas in the induction arm and 38 primigravidas and 13 multigravidas in the conservative management arm.

The next baseline characteristic that was analysed was the body mass index among the patients in the 2 groups.

Table 5 . BODY MASS INDEX (BMI)

BMI	Induction Group		Conservative Management Group		p - Value
	n	%	n	%	
<19	4	8.2	-	-	0.114
19-24.9	25	51.0	28	54.9	
25-29.9	20	40.8	23	45.1	

Graph 3 . Body Mass Index (BMI)



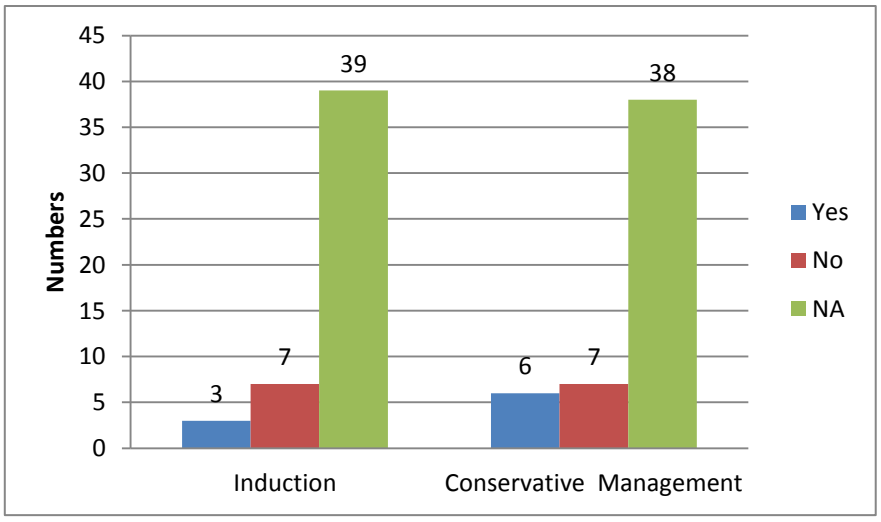
BMI in both groups were similar.

The final baseline characteristic that was analysed was the history of gestational hypertension in previous pregnancy among the patients in both groups.

Table 6. GESTATIONAL HYPERTENSION IN PREVIOUS PREGNANCY

Gestational hypertension in previous pregnancy	Induction Group		Conservative Management Group		p – Value
	n	%	n	%	
Yes	3	6.1	6	11.8	0.615
No	7	14.3	7	13.7	
NA	39	79.6	38	74.5	

Graph 4. Gestational hypertension in previous pregnancy



Both groups had similar number of patients with history of gestational hypertension in previous pregnancy.

Table 7

NUMBER OF OUTPATIENT (OPD) VISITS

OPD visits	**N**	**Mean(SD)**	**Minimum**	**Maximum**
Induction Group	49	1.06 (0.32)	1	3
Conservative management group	51	2.33 (0.79)	1	4

Patients in the induction arm had only 1 OPD visit after randomisation while patients on conservative management had 2 to 3 OPD visits for maternal and foetal surveillance. Only 1 patient in the induction group who was induced at 37 weeks, but was discharged after 2 doses of misoprostol due to unfavourable cervix had to come back for 2 more OPD visits. She was not induced on the 2nd visit according to the study protocol as her blood pressure was normal on that day and she refused induction.

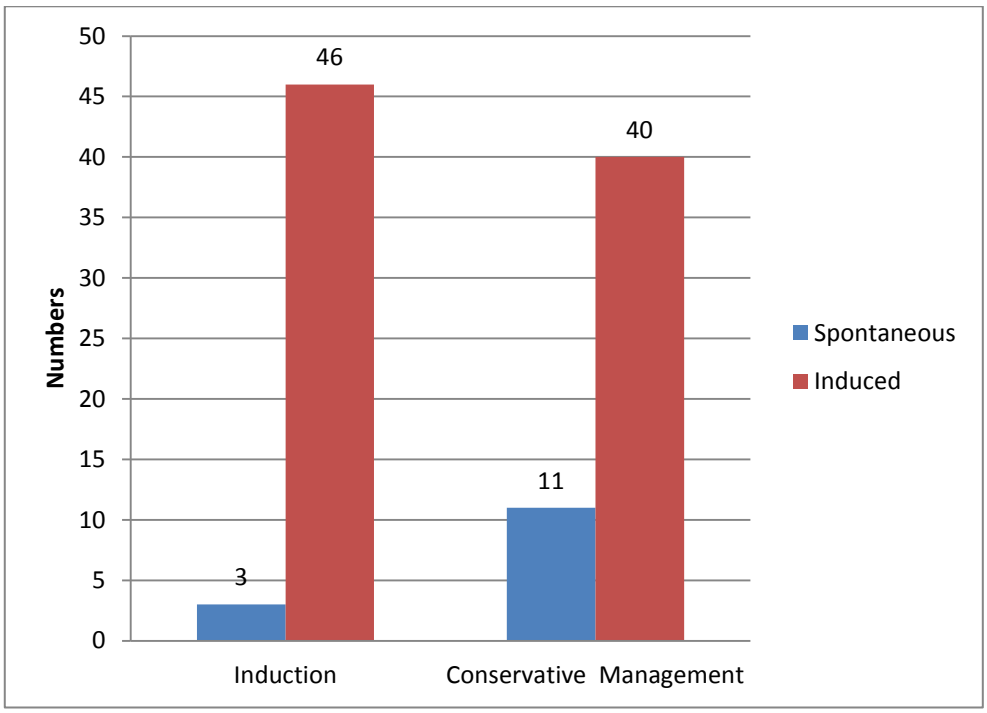
LABOUR CHARACTERISTICS:

Table 8 .

ONSET OF LABOUR:

Onset of labour	Induction group		Conservative Management group		p – Value
	N	%	n	%	
Spontaneous	3	6.1	11	21.6	0.041
Induced	46	93.9	40	78.4	

Graph 5. Onset of labour



There were more patients with spontaneous onset of labour in the conservative management arm when compared to induction arm(11 versus 3) . Three patients in the induction group who had favourable Bishop score went into labour after vaginal examination and did not require oxytocin augmentation.They delivered within 12 hours of vaginal examination.

Though there were more patients with spontaneous onset of labour in the conservative management arm,78.4% of patients in the conservative management arm had to be induced for various reasons (p 0.041). This is statistically significant.

The indications for induction in the conservative management arm were:

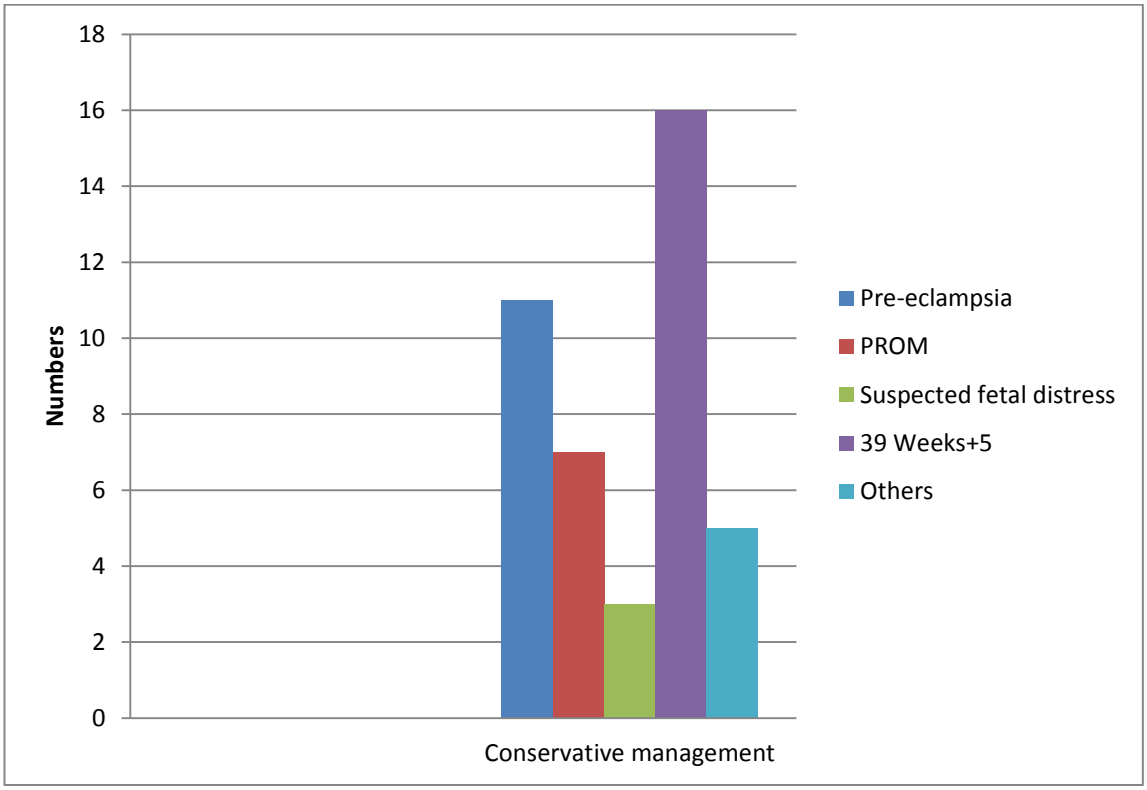
Table 9

INDICATIONS FOR INDUCTION IN THE CONSERVATIVE GROUP:

Indication for induction	**Conservative Management group**	
	n	**%**
Pre-eclampsia	11	26.2
PROM	7	16.7
Suspected foetal distress	3	7.1
39 Weeks + 5 days	16	38.1
Others	5	11.9

Graph 6

Indications for induction in the conservative management group:



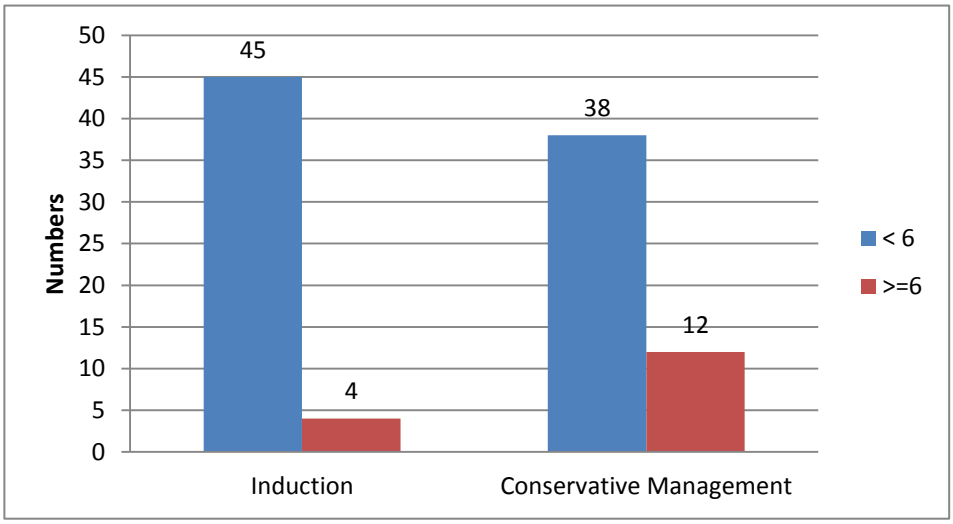
The most common indication for induction of labour in the conservative management group was reaching the gestational age of 39 weeks and 5 days (n=16, 38.1%) followed by progression to preeclampsia (n=11,26.2%) as the next common indication.

Table 10

BISHOP SCORE AT THE ONSET OF INDUCTION OF LABOUR:

Bishop score	Induction group(n=49)		Conservative Management group (n=51)		p – Value
	n	%	N	%	
< 6	45	91.8	38	76.0	0.054
≥ 6	4	8.2	12	24.0	

Graph 7 Bishop score



Patients on conservative management were found to have a more favourable Bishop score at the onset of induction of labour. This was statistically significant (p 0.054).

Table 11 ANALYSIS OF OTHER CONTINUOUS VARIABLES:

Group	Variables	N	Mean (SD)	Minimum	Maximum
Induction group	Rupture of membranes to delivery(hours)	49	9.78(4.26)	1	18
	Gestational age at delivery(weeks)	49	38.24	37.1	40
	Number of days of hospital stay	49	5.33(1.87)	3	12
Conservative Management group	Rupture of membranes to delivery(hours)	51	10.39(5.48)	1	26
	Gestational age at delivery(weeks	51	38.98(0.73)	37.2	40
	Number of days of hospital stay	51	4.65(1.35)	2	8

- There was no significant difference in the rupture of membranes to delivery interval between the 2 groups.
- The average gestational age at delivery was 38 weeks in the induction arm and 39 weeks in the conservative management arm.
- There was no statistically significant difference in the number of days of hospital admissions between the 2 groups.

PRIMARY OUTCOMES:

The primary outcome measured for the mother were maternal mortality and composite maternal morbidity (pre eclampsia , eclampsia HELLP syndrome , pulmonary oedema , renal failure , thromboembolic disease , abruption , need for ICU care and major postpartum haemorrhage). The complications encountered in each group were analysed separately and together and are discussed below.

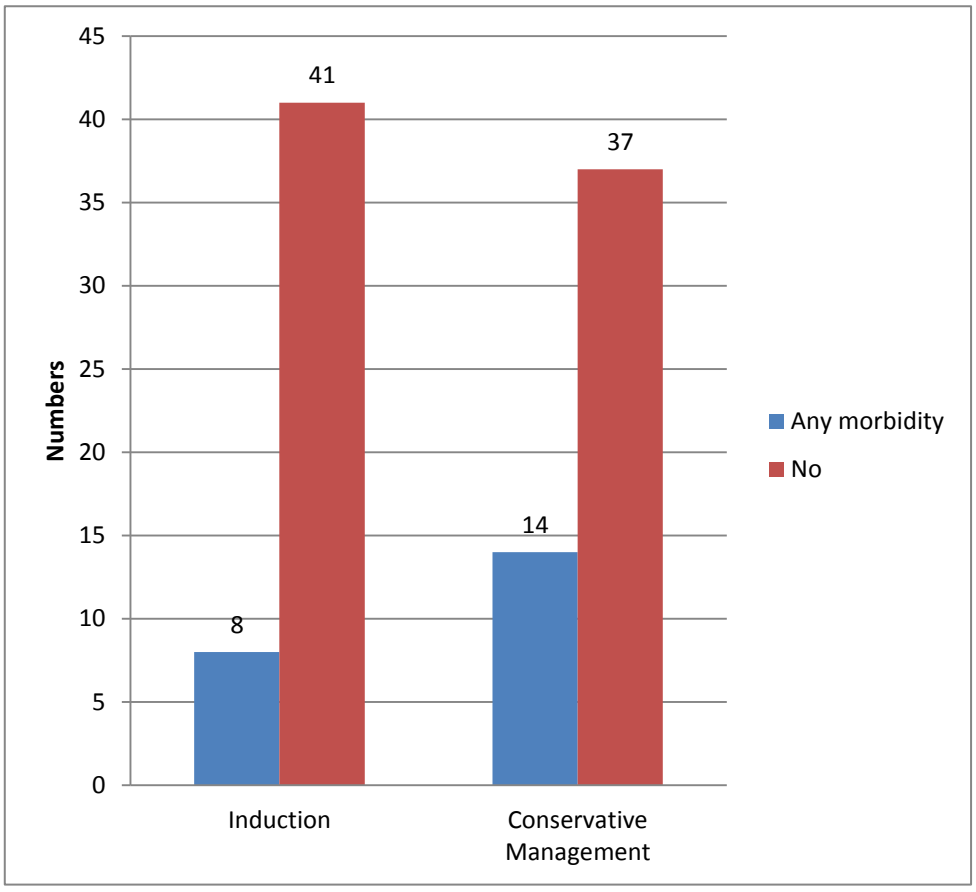
- There was no maternal mortality in both the groups.

Table 12

COMPOSITE MATERNAL MORBIDITY:

Any morbidity	Induction group(n=49)		Conservative Management group(n=51)		p – Value
	n	%	n	%	
Yes	8	16.3	14	27.5	0.230
No	41	83.7	37	72.5	

Graph 8. Composite maternal morbidity



Composite maternal morbidity meant presence of any one of the following complications - pre eclampsia , eclampsia HELLP syndrome , pulmonary oedema , renal failure , thromboembolic disease , abruption , need for ICU care or major postpartum haemorrhage

- There was a slightly increased incidence of composite maternal morbidity in the conservative management arm when compared to induction arm (14 versus 8), though this was not statistically significant (p 0.23).

The lack of statistical significance may be because only 100 patients were analysed whereas 342 patients were required for adequate power.

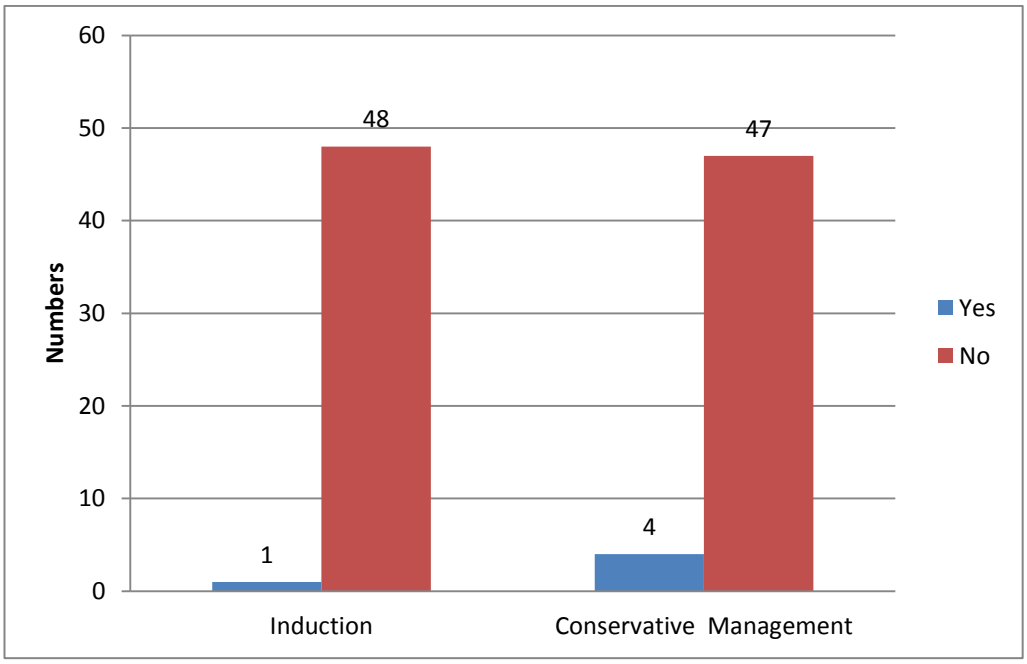
The different variables within the composite maternal morbidity were analysed separately and p value for each variable was calculated.

Table 13

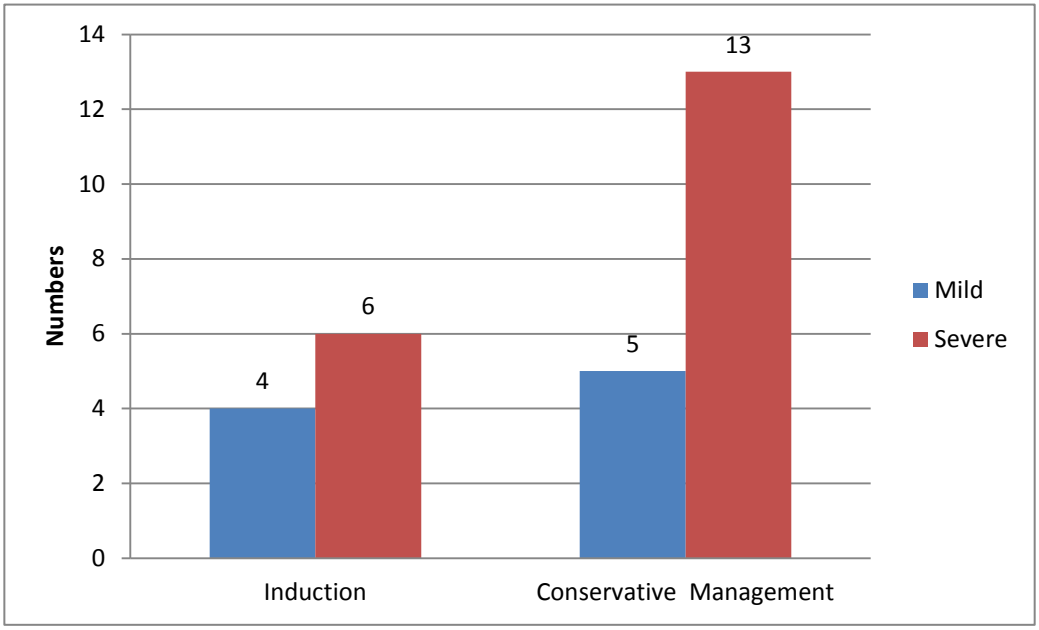
PROGRESSION TO SEVERE GESTATIONAL HYPERTENSION, PREECLAMPSIA AND ECLAMPSIA

Variables	Induction group		Conservative Management group		p – Value
	n	%	N	%	
Severe hypertension					
Yes	1	2.0	4	7.8	0.363
No	48	98.0	47	92.2	
Preeclampsia					
Mild	4	40.0	5	27.8	0.677
Severe	6	60.0	13	72.2	
Eclampsia					
Yes	1	2.0	-	-	0.490
No	48	98.0	51	100.0	

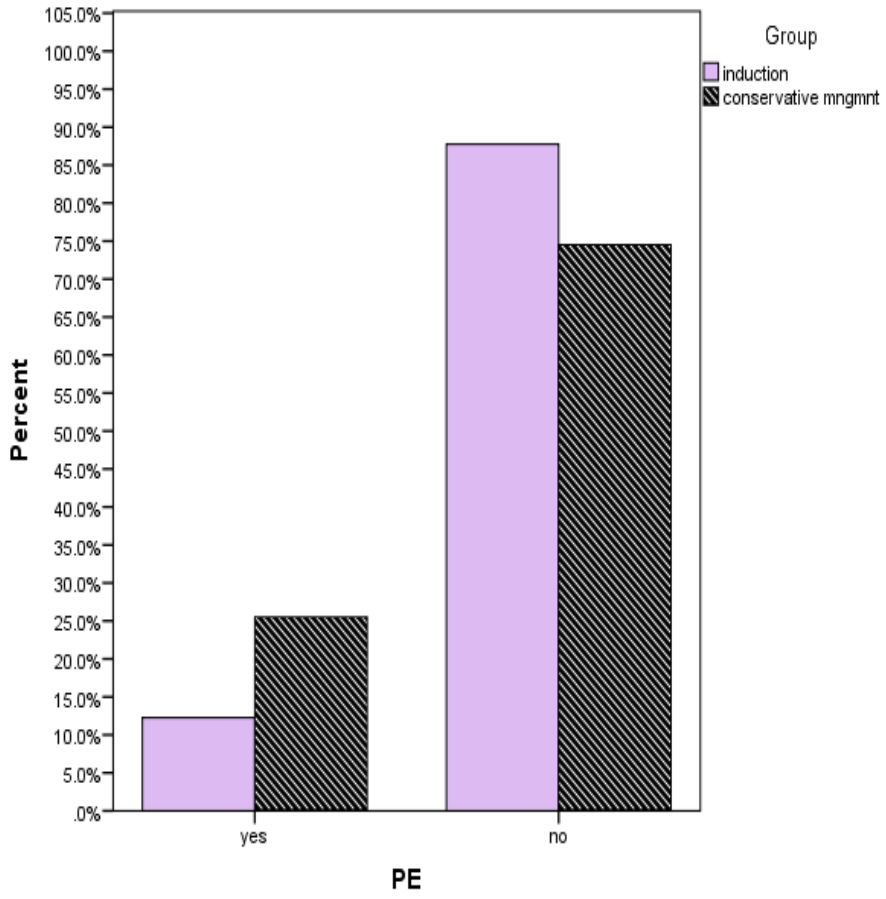
Graph 9. Progression to severe gestational hypertension



Graph 10. Progression to preeclampsia

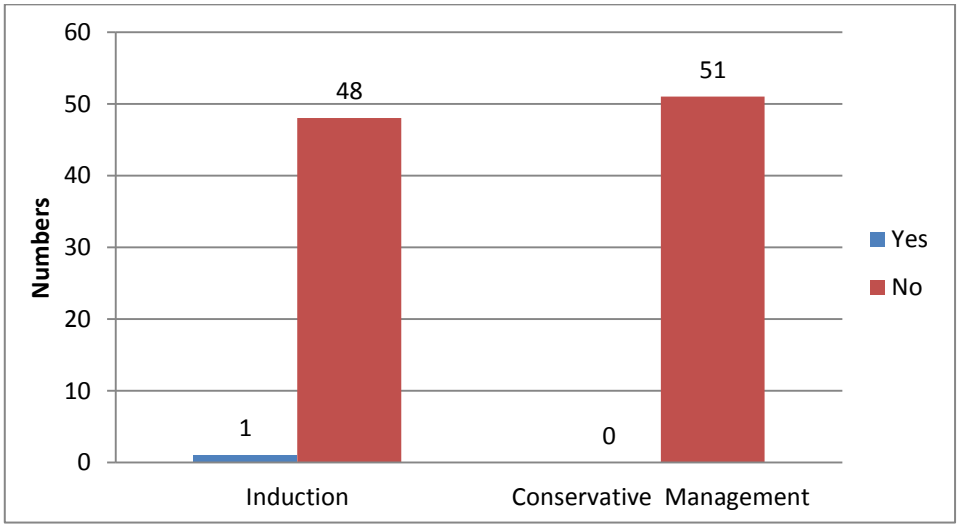


Graph 11. Percentage of progression to preeclampsia(PE)



Graph 12.

Progression to eclampsia



- Only 1 patient in the induction group (2%) while 4 patients in the conservative management group (7.8%) progressed to severe gestational hypertension .But this was not statistically significant(p 0.363)
- A total of 10 patients in the induction arm progressed to pre eclampsia – 4 to mild pre eclampsia and 6 to severe pre eclampsia .In the conservative arm, 18 patients progressed to pre eclampsia - 5 to mild pre eclampsia and 13 to severe pre eclampsia. Though there is an increase in the progression to pre eclampsia in the conservative management group, this is not statistically significant (P 0.677).
- One patient in the induction arm had intrapartum eclampsia while none in the conservative management arm had eclampsia.

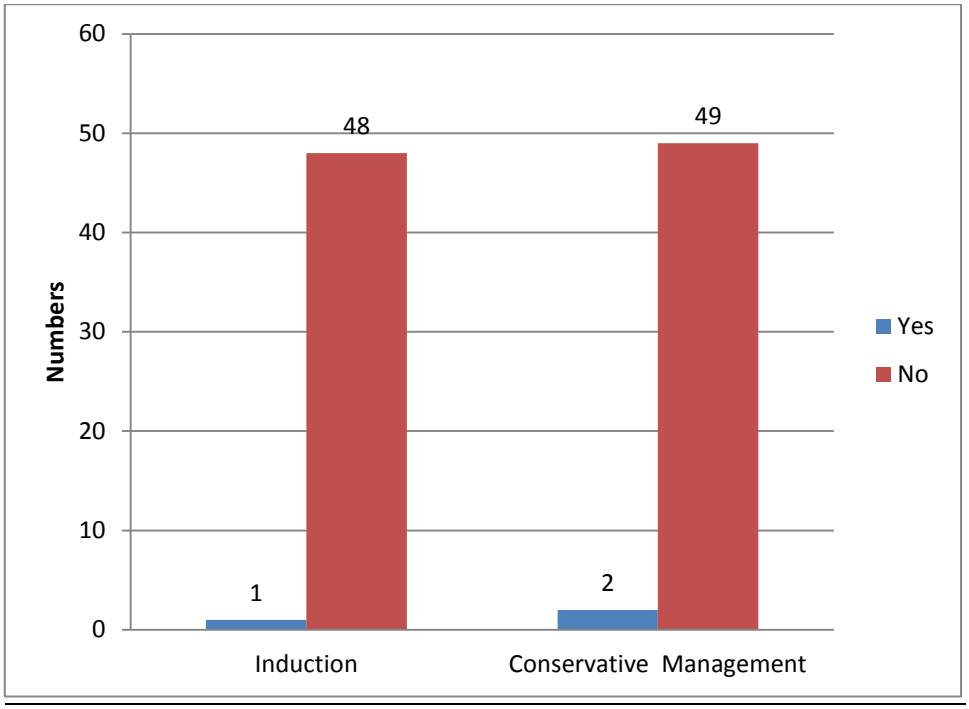
Table 14

POSTPARTUM HAEMORRHAGE (PPH):

PPH	**Induction Group(n=49)**		**Conservative Management Group(n=51)**		**p – Value**
	n	**%**	**n**	**%**	
Yes No	1 48	2.0 98.0	2 49	3.9 96.1	1.000

Graph 13

Postpartum haemorrhage:



There was no statistically significant difference in the incidence of postpartum haemorrhage between the 2 groups. None of the patients in either groups required blood transfusion.

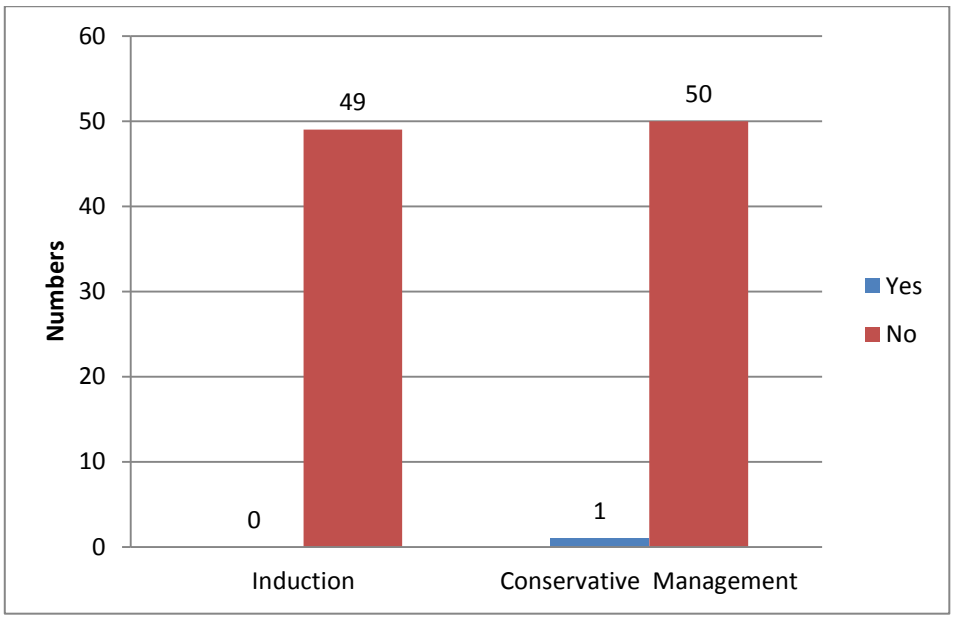
- None of the patients in either group had complications like HELLP syndrome, pulmonary oedema, renal failure or placental abruption.
- There were no patients in either group who required Intensive Care Unit admission.

Table 15

CEREBRAL VENOUS THROMBOSIS:

Thrombosis	Induction group(n=49)		Conservative Management group(n=51)		p – Value
	N	%	n	%	
Yes	-	-	1	2.0	1.000
No	49	100.0	50	98.0	

Graph 14. Cortical VenousThrombosis:



One patient in the conservative management arm who delivered normally and was discharged on 5th postnatal day without any complications got re admitted on her 12th postnatal day with right focal motor seizures followed by right upper and lower limb weakness. Magnetic resonance imaging of her brain showed evidence

of cortical venous thrombosis and she was started on antiepileptics and anticoagulants on which she gradually improved.

SECCONDARY OUTCOMES:

The secondary outcomes measured for the mother were the mode of delivery (normal / instrumental / caesarean) , cost analysis , need for anti convulsant , need for anti hypertensives (intra partum / post natal).

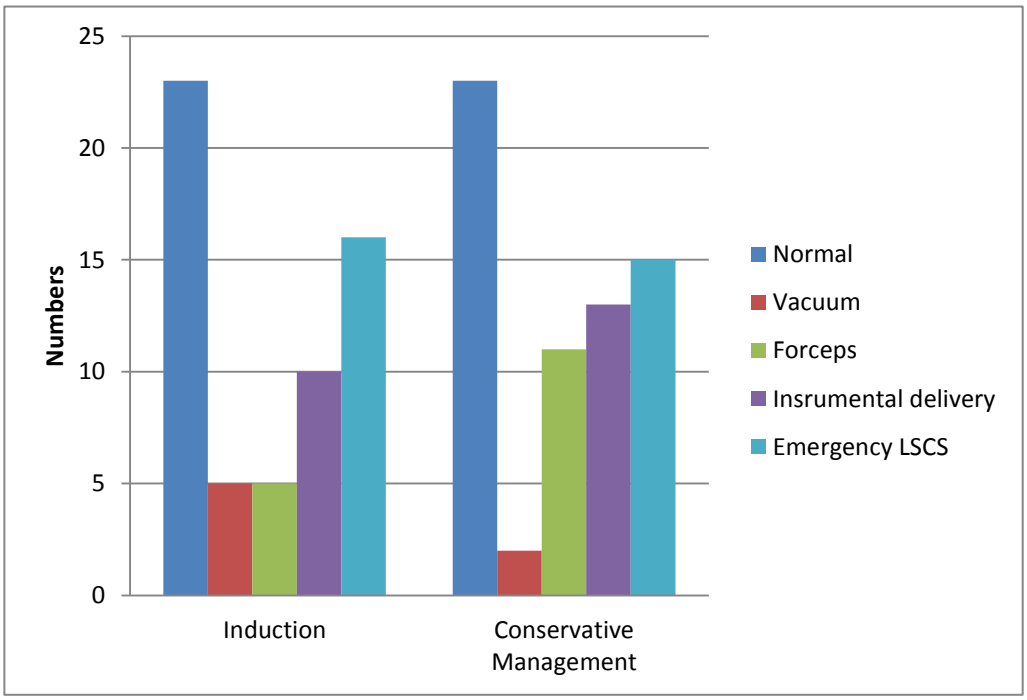
Table 16

MODE OF DELIVERY:

Mode of delivery	**Induction Group(n=49)**		**Conservation Management Group (n=51)**		**p - Value**
	n	**%**	**n**	**%**	
Normal	23	46.9	23	45.1	0.500
Vacuum	5	10.2	2	3.9	
Forceps	5	10.2	11	21.6	
Instrumental delivery(vacuum+ forceps)	10	20.4	13	25.5	0.313
Emergency LSCS	16	32.7	15	29.4	0.306

Graph 15

Mode of delivery:



There was no statistically significant difference in the instrumental delivery and caesarean section rates between the 2 groups (p 0.313 and 0.306 respectively). Induction of labour was not found to increase the caesarean section rate contrary to the popular belief. This was inspite of a much favourable Bishop score in the conservative management group when compared to the induction group (0.054). Though there were more women who went into spontaneous onset of labour in the conservative management group, the caesarean section rate was almost the same in both groups. This may be because of a slightly higher rate of progression of the disease in the conservative management group which predisposed them to caesarean section.

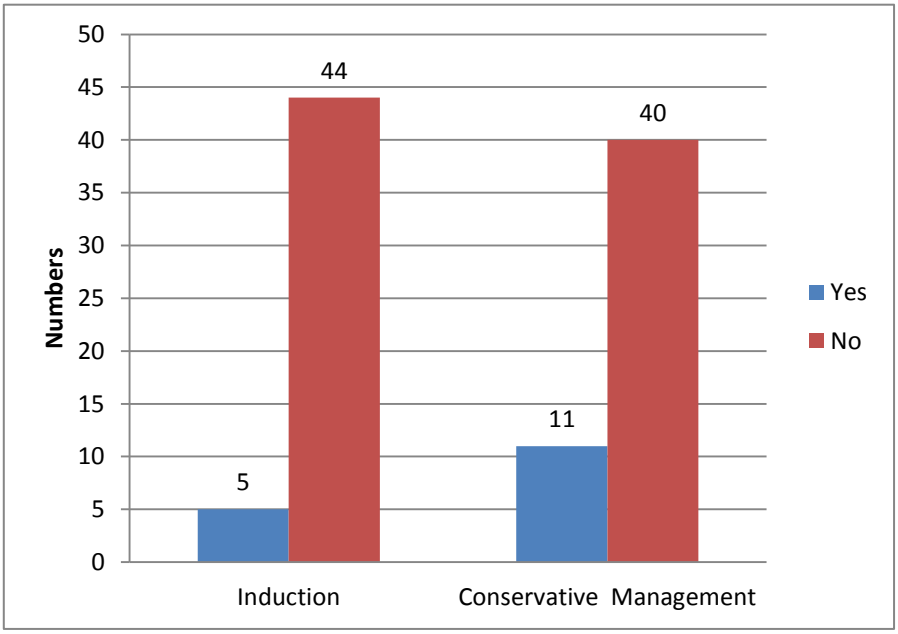
Table 17

USE OF ANTIHYPERTENSIVES:

Variables	Induction group(n=49)		Conservation Management group(n=51)		p - Value
	n	%	N	%	
Antihypertensives					
Yes	5	10.2	11	21.6	0.173
No	44	89.8	40	78.4	
If yes, (n = 5)					
Intrapartum	2	40.0	10	90.9	0.063
Postpartum	3	60.0	1	9.1	

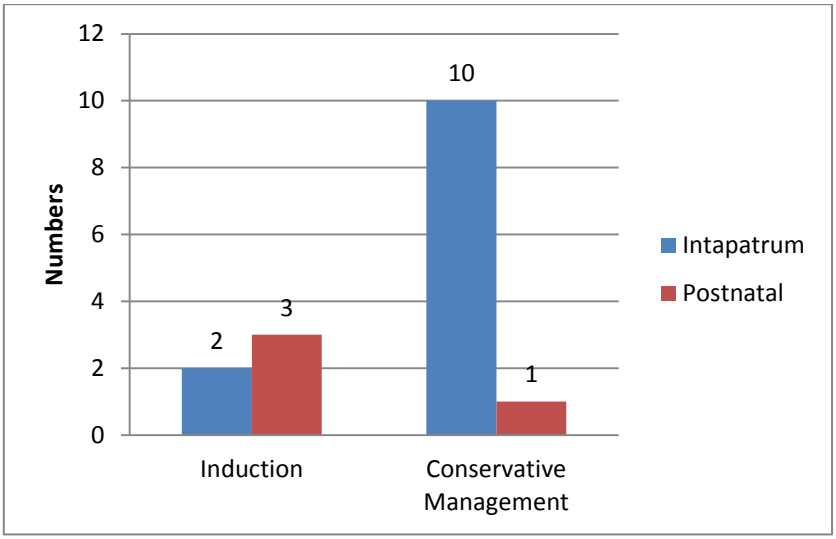
Graph 16

Use of antihypertensives:



Graph 17

Intrapartum versus postpartum use of antihypertensives



Eleven patients in the conservative management group and 5 patients in the induction group required use of antihypertensives. But this was not statistically significant (p 0.173). Patients on conservative management required more intrapartum use of antihypertensives (0.063)

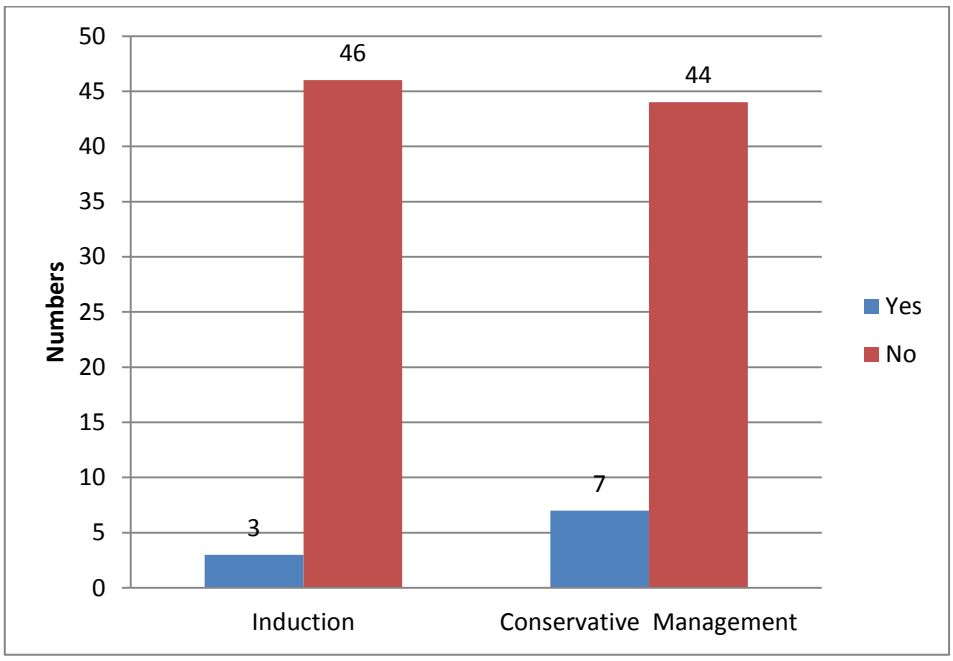
Table 18

USE OF ANTICONVULSANT-MAGNESIUM SULPHATE:

Mgso4	**Induction group(n=49)**		**Conservative Management group(n=51)**		**p – Value**
	N	**%**	**n**	**%**	
Yes No	3 46	6.1 93.9	7 44	13.7 86.3	0.319

Graph 18.

Use of anticonvulsant



There was increased usage of anticonvulsant $MgSO_4$ in the conservative management arm when compared to the induction arm (7 versus 3), though not statistically significant (p 0.319).

NEONATAL OUTCOMES:

The primary outcome measured for the baby was perinatal mortality and the secondary outcomes measured for the baby was neonatal morbidity (5 minute APGAR score less than 7 , cord pH less than 7, admission to neonatal intensive care unit) .

- There were no intrauterine deaths in either group.

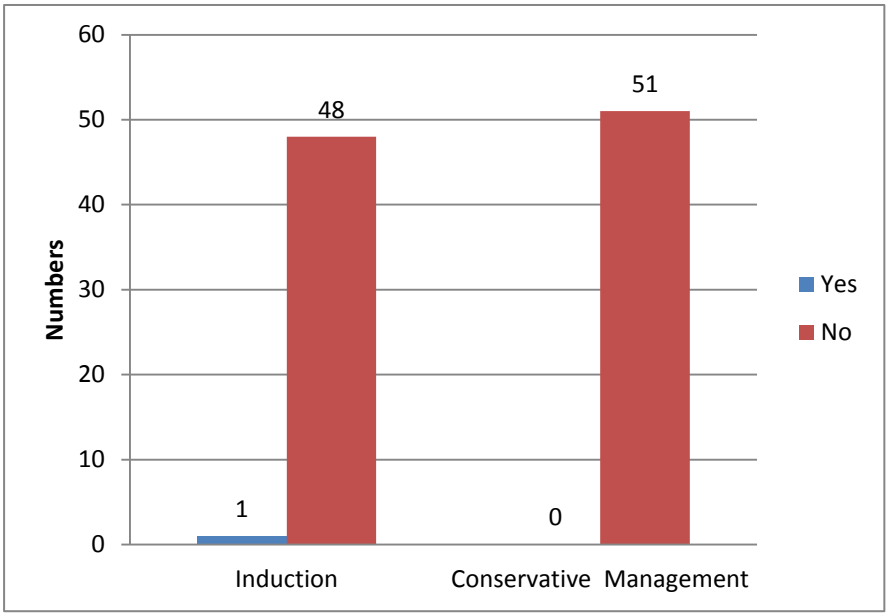
Table 19

NEONATAL OUTCOMES:

Variables	Induction group(n=49)		Conservative Management group(n=51)		p - Value
	n	%	n	%	
Neonatal deaths					
Yes	1	2.0	-	-	0.490
No	48	98.0	51	100.0	
Apgar Score					
7-9	48	98.0	50	98.0	1.000
5-6	1	2.0	1	2.0	
NICU admissions					
Yes	3	6.1	1	2.0	0.357
No	46	93.9	50	98.0	

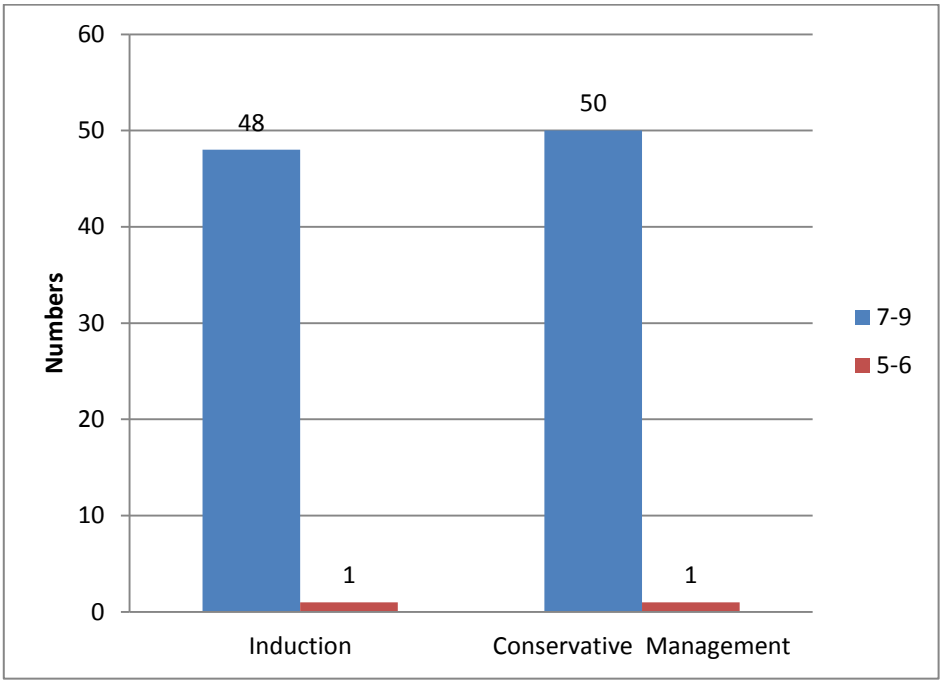
Graph 19

Neonatal deaths



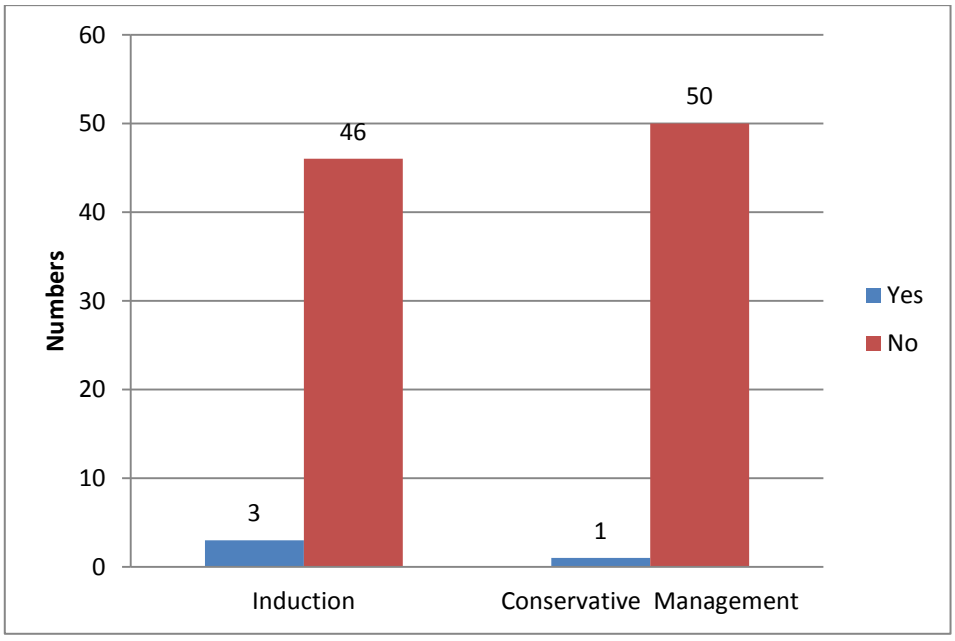
Graph 20

Apgar scores



Graph 21

NICU admissions.



- There was 1 neonatal death in the induction group due to perinatal asphyxia but this was not statistically significant (p 0.490).
- The babies in both groups had no difference in the Apgar scores at birth
- One patient in the induction group had cord pH less than 7 (Cord pH 6.9) and this baby was admitted to NICU and died on 6th postnatal day.
- There was a slightly increased incidence of NICU admissions in the induction arm when compared to conservative management arm (3 versus 1), though not statistically significant (p 0.357).

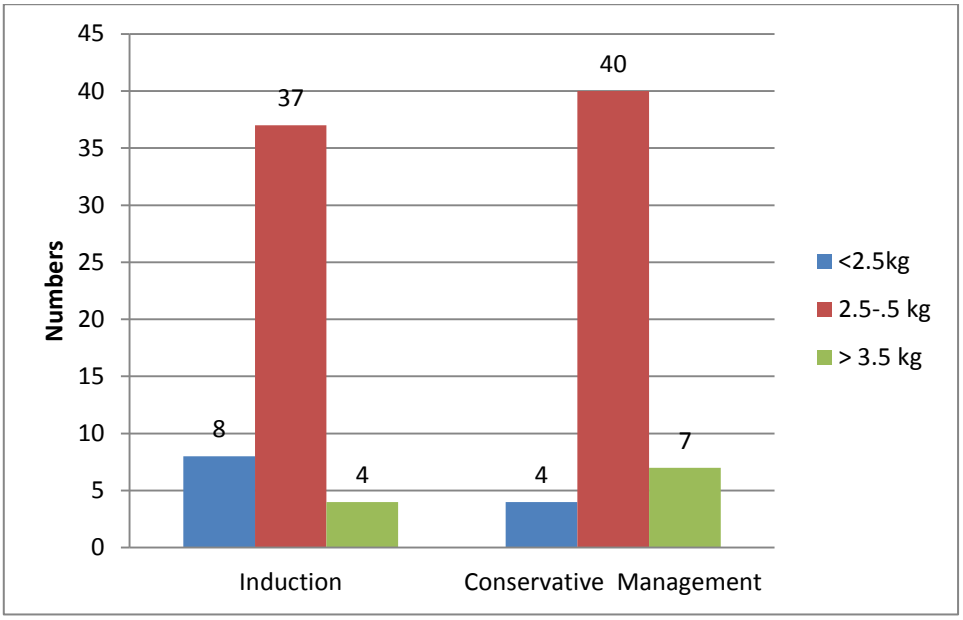
Table 20

BIRTH WEIGHT:

Birth weight	**Induction group(n=49)**		**Conservative Management group(n=52)**		**p – Value**
	n	**%**	**n**	**%**	
< 2.5kg 2.5 – 3.5 kg > 3.5 kg	8 37 4	16.3 75.5 8.2	4 40 7	7.8 78.4 13.7	0.328

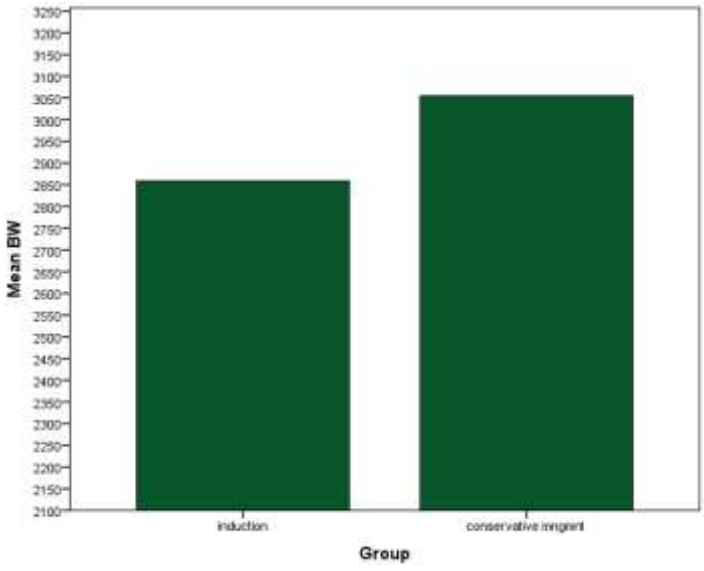
Graph 22

Bir.th weight



Graph 23

Average birth weight



The mean birth weight was 2.8 kg in the induction group and 3 kg in the conservative management group. But there was no statistically significant difference in the incidence of growth restricted babies or big babies between the 2 groups (p 0.328).

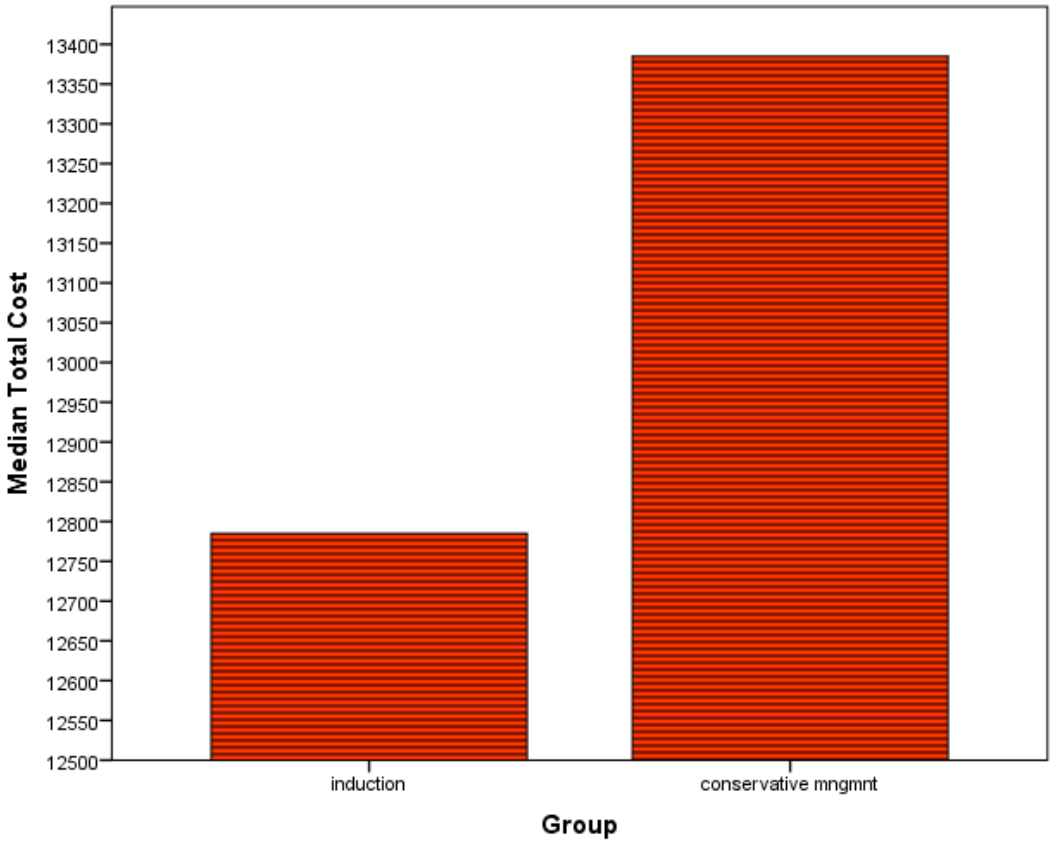
Table 22

COST ANALYSIS (IN INDIAN RUPEES)

Total cost	**n**	**Mean (SD)**	**Median**	**Minimum**	**Maximum**
Induction group	49	17224 (11714.26)	**12785**	8250	75355
Conservative Group	51	15163.31 (5471.37)	**13385**	7970	26280

Graph 25

Cost Analysis



There is a slight increase in the median total cost in the conservative management group by about 600 rupees which may be attributable to the tests for maternal and foetal surveillance, but this is not statistically significant. The mean cost was not taken for statistical analysis because of the error in calculation due to the very high maximum cost in the induction group for 1 patient whose baby had perinatal asphyxia and was admitted in NICU for 6 days.

DISCUSSION

The management of severe gestational hypertension or preeclampsia at term is delivery. But for mild hypertension at term, there is no definite treatment protocol. Only about 10% of patients with gestational hypertension at term progress to preeclampsia and its complications[5]. Though induction of labour is thought to prevent the maternal and neonatal complications in most situations, it might increase the chance for caesarean section. Conversely, in conservative management, there is more chance for spontaneous onset of labour and normal delivery. But because of the 10% risk of progression of the disease, it should be practised only in centres where close maternal and foetal surveillance is possible. Our study aims to find out the optimal treatment option for women with mild gestational hypertension after 37 weeks.

We recruited 100 patients with mild gestational hypertension between 37 weeks and 39 weeks and 5 days for the study. Induction group had 49 patients and conservative management group had 51 patients. Though the calculated sample size based on the results of HYPITAT study for adequate power was 342, we could recruit only 100 eligible patients within a period of 14 months.

The primary outcomes measured for the mother were maternal mortality and composite maternal morbidity. There was no maternal mortality in both the groups. This result was similar to the results of the HYPITAT trial[4].

Composite maternal morbidity included the presence of any one of the

following complications like pre eclampsia , eclampsia HELLP syndrome , pulmonary oedema , renal failure , thromboembolic disease , abruption , need for ICU care or major postpartum haemorrhage. We found that there was a slightly increased incidence of composite maternal morbidity in the conservative management arm when compared to induction arm (14 versus 8), though this was not statistically significant (p 0.23). 44% of the patients allocated to the expectant monitoring arm compared to 31% of patients in the induction group had maternal morbidity in HYPITAT trial ($p < 0.001$). Though our findings showed a similar trend, we did not get the statistical significance because our study was not adequately powered.

One patient in the induction group (2 %) and 4 patients in the conservative management group (7.8%) progressed to severe hypertension (p 0.363), but this was not statistically significant. Twelve percent of patients in the induction group and 26% of patients in the conservative management group progressed to preeclampsia (p 0.677). A study done by Patrick Saudan also found only 10% progression of term gestational hypertension to preeclampsia.[5]There was no statistically significant difference in other maternal morbidities like eclampsia, HELLP syndrome, thrombosis, renal failure, pulmonary oedema, abruption and postpartum haemorrhage.

The secondary outcomes measured for the mother were the mode of delivery (normal / instrumental / caesarean section) , cost analysis , need for anti convulsant , need for anti hypertensives (intra partum / postpartum).

There was no statistically significant difference in the instrumental delivery and caesarean section rates between the 2 groups (p 0.313 and 0.306 respectively).

Though the HYPITAT study showed that fewer caesarean sections were needed in the induction group when compared to expectant monitoring (not statistically significant), we did not get this result. Several non-randomised trials had showed the association of induction of labour with increased number of caesarean sections[73,74]. But we found that induction of labour did not significantly increase the caesarean section rate either. Therefore, we need more evidence to find out the association between labour induction in these high risk women and caesarean section rates.

This result was inspite of a much favourable Bishop score in the conservative management group when compared to the induction group (0.054). Though there were more women who went into spontaneous onset of labour in the conservative management group, the caesarean section rate was almost the same in both groups. This may be because of a slightly higher rate of progression of the disease in the conservative management group which predisposed them to caesarean section.This result is similar to the result of the study done by Tajik and associates in 2012[48].

A significant number of patients (78.4%) in the conservative management arm had to be induced for various reasons (p 0.041).In HYPITAT study also, almost half of the patients in the expectant group had to be induced.

Eleven patients in the conservative management group and 5 patients in the induction group required use of antihypertensives. But this was not statistically significant (p 0.173). Patients on conservative management required more intrapartum use of antihypertensives (0.063). There was also increased usage of anticonvulsant $MgSO_4$ in the conservative management arm when compared to the induction arm (7 versus 3), though not statistically significant (p 0.319).This result was similar to that of HYPITAT trial.

The primary and secondary outcomes for the baby were perinatal mortality and neonatal morbidity respectively. There was no statistically significant difference in the neonatal morbidity and mortality between the 2 groups which was similar to that of HYPITAT study.

The mean birth weight in the induction arm was lower than that in the conservative management arm (2.8 kg versus 3 kg) which was similar to the results from the HYPITAT study. But there was no statistically significant difference in the incidence of growth restricted or big babies between the 2 groups. A population based retrospective study in 2004 in women with gestational hypertension, severe pre-eclampsia and women with normal blood pressure also did not find any significant differences in mean birth weights between women with gestational hypertension and those with normal blood pressure.[32]

The cost analysis showed that induction of labour was cheaper than conservative management, though this was not significant statistically. This result was similar to that of the HYPITAT study data. An economic analysis of induction of labour and expectant monitoring in women with gestational hypertension or pre eclampsia at term done by Mortiary showed that induction of labour was 11% cheaper than expectant management[49]

Thus, the results of the study favour induction of labour for mild gestational hypertension at term. The study has to be continued to reach the calculated sample size for proper statistical significance.

POINTS TO BE NOTED

MATERNAL FACTORS:

1. Patients in the conservative management arm had a more favourable Bishop score at the onset of induction of labour (p 0.054).
2. The average gestational age at delivery was 38 weeks for patients in the induction arm and 39 weeks in patients on conservative management.
3. Though there were more patients with spontaneous onset of labour in the conservative management group, a significant number of patients (78.4%) in the conservative management arm had to be induced for various reasons (p 0.041).
4. There was no maternal mortality in both groups
5. The composite maternal morbidity was slightly more in the conservative management group when compared to the induction group, but this was not statistically significant (p 0.23)
6. Two percent of patients in the induction arm versus 7.8% of in the conservative management arm progressed to severe gestational hypertension, though this is not statistically significant(p 0.363)
7. In the induction arm, 10 patients progressed to pre eclampsia- 4 mild pre eclampsia and 6 severe pre eclampsia. In the conservative management arm 18 progressed to pre eclampsia- 5 mild pre eclampsia and 13 severe pre eclampsia. Though there is a higher rate of progression to pre eclampsia in the conservative group, this is not statistically significant (p 0.677).
8. There was no statistically significant difference in the incidence of eclampsia, postpartum haemorrhage and thrombosis.
9. None of the patients in either group had HELLP syndrome, pulmonary oedema, renal failure or abruptionplacenta .None of the patients required ICU admission.

10. There was no statistically significant difference in the instrumental delivery or caesarean section rates between the 2 groups. Induction of labour was not found to increase the caesarean section rate contrary to the popular belief.
11. There was a slight increase in the use of antihypertensives and magnesium sulphate in the conservative management arm (p 0.173 and 0.319 respectively), but this was not statistically significant.
12. There was no statistically significant difference in the number of days of hospital admissions between the 2 groups.

NEONATAL FACTORS

13. There was no intrauterine death in both groups .
14. There was no statistically significant difference in Apgar scores or neonatal deaths between the 2 groups.
15. A slight increase in NICU admissions was found in the induction arm when compared to the conservative management arm, but this was not statistically significant (p 0.357).
16. The mean birth weight in the induction arm was 2.8 kg and that in the conservative management arm was 3 kg .There was no statistically significant difference in the incidence of growth restricted babies or big babies between the 2 groups.

COST ANALYSIS

17. A slight increase by about 600 rupees in the median total cost was found in the conservative management group when compared to the induction group, but this was not statistically significant. This may be because of the more OPD visits in

the conservative management group and the more tests for maternal and foetal surveillance done in this group.

LIMITATION

The sample size calculated with a power of 80% and error of 5% based on the results of the HYPITAT trial was 342 (171patients in each arm) .But only 100 eligible patients could be recruited in the study (49 patients in the induction arm and 51 patients in the conservative management arm) within a period of 14 months from September 2011 to October 2012.This is the main limitation of this study. The study needs to be continued to attain the calculated sample size for proper statistical significance.

CONCLUSIONS

1. The results of the study showed that induction of labour in women with singleton pregnancy complicated by gestational hypertension at term reduces maternal morbidity when compared to conservative management, though not statistically significant. There was no maternal mortality in either group.
2. There was no statistically significant difference in the instrumental delivery or caesarean section rates between the 2 groups. Induction of labour was not found to increase the caesarean section rate contrary to the popular belief.
3. There was no statistically significant difference in the neonatal morbidity and mortality between the 2 groups
4. Induction of labour reduced the expenditure when compared to conservative management as the costs for foetal surveillance and increased number of antenatal visits can be avoided. A slight increase by about 600 rupees in the median total cost was found in the conservative management group when compared to the induction group, but this was not statistically significant

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ANNEXURE-1

PATIENT INFORMATION SHEET

Christian Medical College, Vellore

Department of Obstetrics and Gynaecology

A randomized trial comparing induction of labour with conservative monitoring for mild gestational hypertension at term

Hypertensive disorders complicate 6-10 % of all pregnancies close to delivery (at term) and can result in complications for the mother and the baby. Delivery of the baby usually reverses the complications brought about by this hypertensive disorder.

Definite guidelines are there for treatment of very high blood pressures during pregnancy. But strong practice variations exist for treatment of women with mild elevation of blood pressure without other complications at term.

Immediate delivery is thought to prevent mainly progression to severe hypertension. Along with progression of the disease there could be severe maternal and neonatal complications like fits, low platelet count, kidney failure, heart failure and fetal distress.

Conversely, immediate delivery might increase the risk of instrumental delivery (forceps and vacuum delivery) and caesarean section in the mother and special care for the baby in the neonatal intensive care due to early delivery. These generate additional morbidity and costs along with complications in subsequent pregnancies.

The aim of this study is to find out whether immediate delivery in women with singleton pregnancy complicated by mild elevation of blood pressure at term prevents adverse pregnancy outcome when compared to conservative management without increasing instrumental delivery and caesarean section rates. You may be enrolled in either group (immediate delivery or conservative management) based on randomization.

Before you agree to participate in this study, an ultrasound scan will be done to know the estimated weight and well being of the baby. If the baby's growth is adequate and if there are no other complications, you will be eligible to be enrolled in the study.

Neither you nor your doctor will have any choice in whether you will be in the immediate delivery group or monitoring group as this will be decided by a computer program; this is like tossing a coin and you have an equal chance of being in either group.

If you are in the immediate delivery group, a vaginal examination will be done to know the favourability of the cervix and the delivery will be planned accordingly. If you are in the other group, initially blood tests will be done and you will be requested to check your BP daily at home or locally whichever is convenient to you till your expected date of delivery. You will be advised to attend the OPD twice a week to monitor BP, urine protein and growth of the baby. Ultrasound scan will be done weekly free of cost to check the well being of the baby. You will be asked to come to the labour room if your diastolic BP is more than 100, if you have headache, blurring of vision, abdominal pain, decreased urine output,any watery discharge, bleeding from

the vagina or if baby's movements are decreased. In any of these situations, your delivery will be planned immediately. Otherwise,you will be monitored till your expected date of delivery.

If you agree to take part in this study, Your antenatal record will be screened and details of socio demographic profile will be recorded. The outcome of the pregnancy will also be recorded in the clinical research form.

No monetary or material incentive will be provided for participation in the study.

The results of this study may be published in a medical journal but you will not be identified by name in any publication or presentation of results. However, your medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your usual treatment at this hospital in any way.

Your participation is important and of immense value as the results of this study will be useful in improving maternal care in the future.

ANNEXURE 2

CONSENT TO TAKE PART IN A CLINICAL TRIAL

Study Title: *Induction of Labour Vs Conservative Management for Mild Gestational Hypertension at term.*

Study Number:

Participant's name:

Date of Birth / Age (in years):

I_____

_____, son/daughter of _____

(Please tick boxes)

I declare that I have read the information sheet provided to me regarding this study and have clarified any doubts that I had. [ ]

I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]

I also understand the usual antenatal care will be provided irrespective of my participation in the study [ ]

I also understand that neither I, nor my doctors, will have any choice or knowledge of whether I will be in the immediate delivery group or the conservative management group [ ]

I understand that I will not receive any financial benefit or compensation in the event of any complication [ ]

I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access [ ]

I understand that my identity will not be revealed in any information released to third parties or published [ ]

I voluntarily agree to take part in this study [ ]

Name:

Signature:

Date:

Name of witness:

Relation to participant:

Date:

ANNEXURE-3

PROFORMA

Case no: Hospital no: Contact no:

Name: Date of enrollment:

LMP: GA at randomization: Group

	IOL	CONS
Education:1)Nil 2)primary 3)secondary 4)professional		
Age:1)18-30 2)31-35		
Obstetric score: 1)primi 2)multi		
BMI: 1)<19 2) 19-24.9 3)25-29.9		
H/O Gestational hypertension in previous pregnancy:1)yes 2)no 3)NA		
No: of OPD visits:1,2,3,4,5,6		

ANTENATAL CHECK-UP	IOL	C-1	2	3	4	5	6
Syst BP:1)<140 2)140-159 3)>/= 160							
Dias BP: 1)<90 2)90-99 3)100-109 4)>/=110							
Urine alb: 1)nil/trace 2)1+ 3)>/=2+							
Headache 1)yes 2)no							
Blurring of vision:1)yes 2) no							
Decreased urine output:1)yes 2)no							
Vomiting :1)yes 2)no							
Modified BPS:1)Normal 2)abnormal 3) NA							

LABOUR:

Spontaneous-1,Induced-2

Gestational age at delivery :37-38 weeks-1,38+1-39 weeks -2,39+1 -40 weeks -3

If induced,indication for induction:Pre-eclampsia-1,Eclampsia-2,PROM-3,suspected fetal distress-4, 39 weeks+5 days-5,others-6

Bishop's score: <6-1,>/=6-2

First Induction date: Gestational age:

Reinduction date: Gestational age:

No: of attempts: 1,2,3

Time from rupture of membranes to delivery: <12 hours-1,12-24 hours-2,>24 hours-3

Mode of delivery: Normal-1,vaccum-2,forceps-3,Emergency LSCS-4, Elective LSCS-5

OUTCOMES

MOTHER:

Progression to severe hypertension:yes-1,no-2	
Pre-eclampsia:yes-1,no-2 If yes,mild-1,severe-2	
Eclampsisia:yes-1,no-2 HELLP syndrome:yes-1,no-2	
Pulmonary oedema:yes-1,no-2	
Renal failure:yes-1,no-2	
Thromboembolic disease:yes-1,no-2	
Placental abruption:yes-1,no-2 If yes,grades-1,2,3	
PPH (>1 litre blood loss):yes-1 no-2 Need for transfusion of blood or blood products:yes-1,no-2	
for ICU Care:yes-1,no-2 If yes,no: of days of ICU care:	
No: of days of hospital stay:<5-1,5 to 10-2,11 to 20-3,>20 days-4	
Use of antihypertensive:1)yes 2)no If yes, intrapartum-1,postnatal-2,both-3	
e of anticonvulsant:1)yes 2)no	
Maternal mortality:yes-1,no-2	

BABY:

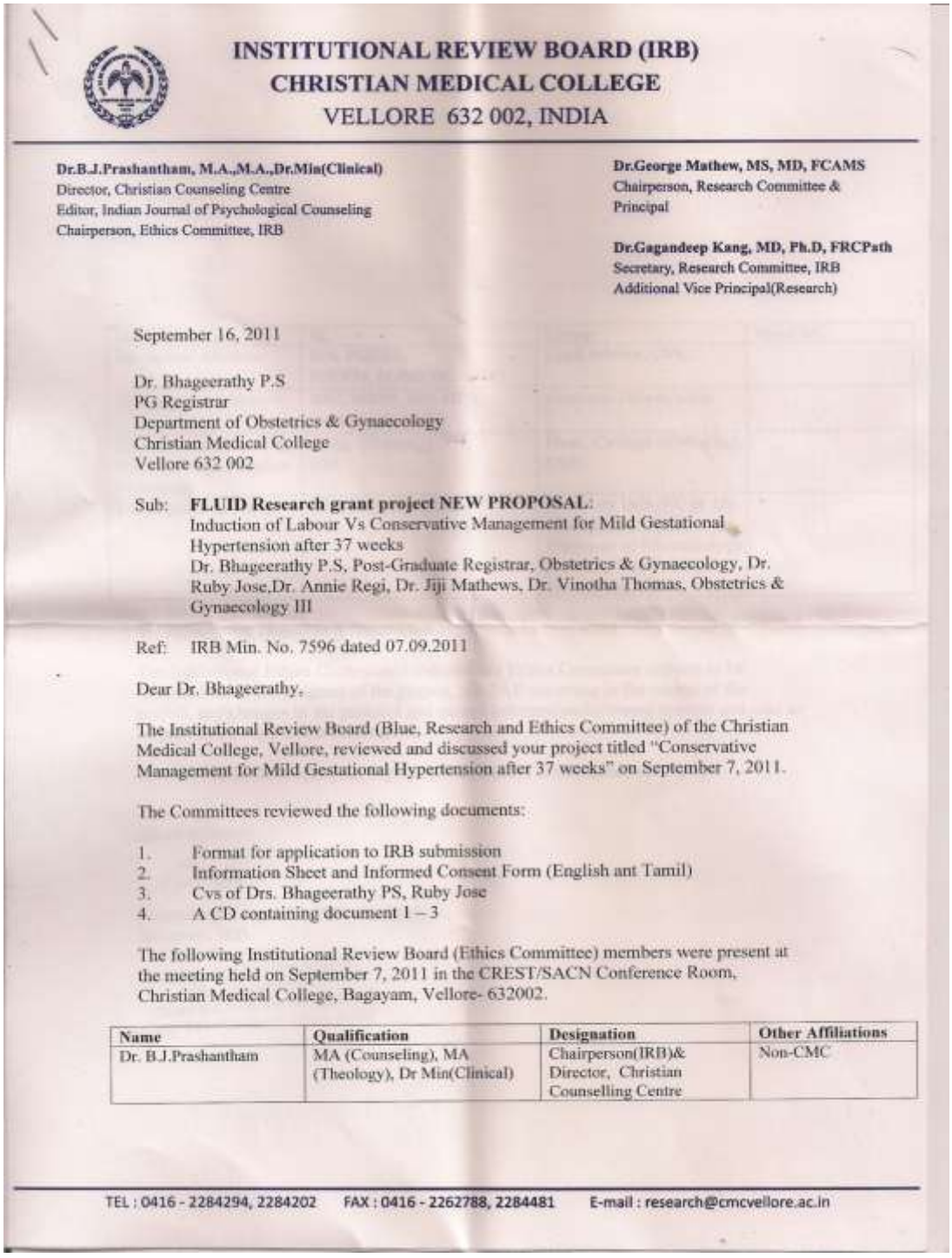
Intra uterine death:yes-1,no-2		
Neonatal death:yes-1,no-2		
Birth weight:<2.5 kg-1,2.5-3.5 kg -2, >3.5 kg -3		
5 minute Apgar score: 7 to9-1,5 to 6-2,1 to 5-3,o-4		
Cord Ph: >/=7.2-1,7 to 7.19-2,<7-3		
Need for ICU care:yes-1,no-2 If yes,no: of days of ICU care:		

COST ANALYSIS-DIRECT MEDICAL COSTS

Consultation charges No: of visits * chage per visit		
Costs for PIH work up		
Modified BPS(160*No: of BPS done)		
Mother's final bill		
Baby's final bill		
Total		

ANNEXURE 4

IINSTITUTIONAL REVIEW BOARD CLEARANCE FORM



INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE
VELLORE 632 002, INDIA

Dr.B.J.Prashantham, M.A.,M.A.,Dr.Min(Clinical)
Director, Christian Counseling Centre
Editor, Indian Journal of Psychological Counseling
Chairperson, Ethics Committee, IRB

Dr.George Mathew, MS, MD, FCAMS
Chairperson, Research Committee &
Principal

Dr.Gagandeep Kang, MD, Ph.D, FRCPath
Secretary, Research Committee, IRB
Additional Vice Principal(Research)

September 16, 2011

Dr. Bhageerathy P.S
PG Registrar
Department of Obstetrics & Gynaecology
Christian Medical College
Vellore 632 002

Sub: **FLUID Research grant project NEW PROPOSAL:**
Induction of Labour Vs Conservative Management for Mild Gestational Hypertension after 37 weeks
Dr. Bhageerathy P.S, Post-Graduate Registrar, Obstetrics & Gynaecology, Dr. Ruby Jose,Dr. Annie Regi, Dr. Jiji Mathews, Dr. Vinotha Thomas, Obstetrics & Gynaecology III

Ref: IRB Min. No. 7596 dated 07.09.2011

Dear Dr. Bhageerathy,

The Institutional Review Board (Blue, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Conservative Management for Mild Gestational Hypertension after 37 weeks" on September 7, 2011.

The Committees reviewed the following documents:

1. Format for application to IRB submission
2. Information Sheet and Informed Consent Form (English ant Tamil)
3. Cvs of Drs. Bhageerathy PS, Ruby Jose
4. A CD containing document 1 – 3

The following Institutional Review Board (Ethics Committee) members were present at the meeting held on September 7, 2011 in the CREST/SACN Conference Room, Christian Medical College, Bagayam, Vellore- 632002.

Name	Qualification	Designation	Other Affiliations
Dr. B.J.Prashantham	MA (Counseling), MA (Theology), Dr Min(Clinical)	Chairperson(IRB)& Director, Christian Counselling Centre	Non-CMC

TEL : 0416 - 2284294, 2284202 FAX : 0416 - 2262788, 2284481 E-mail : research@cmcvellore.ac.in

INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE
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Principal

Dr.Gagandeep Kang, MD, Ph.D, FRCPath
Secretary, Research Committee, IRB
Additional Vice Principal(Research)

Mr. Harikrishnan	BL	Lawyer	Non-CMC
Mr. Samuel Abraham	MA, PGDBA, PGDPM, M.Phil, BL.	Legal Advisor, CMC.	
Dr. Jayaprakash Muliyil	BSC, MBBS, MD, MPH, DrPH(Epid), DMHC	Academic Officer, CMC	
Dr. Vathsala Sadan (on behalf of Mrs. Rosaline Jayakaran)	M.Sc. (Nursing), RN, RM	Dean, College of Nursing, CMC.	
Dr. Gagandeep Kang	MD, PhD, FRCPath.	Secretary IRB (EC)& Dy. Chairperson (IRB), Professor of Microbiology & Addl. Vice Principal (Research), CMC.	

We approve the project to be conducted as presented.

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

A sum of Rs 80,000/- (Rupees Eighty thousand only) is sanctioned for 2 years.

Yours sincerely,

G. Kang

Gagandeep Kang, MD, PhD, FRCPath
Secretary, IRB

Secretary
Institutional Review Board
(Ethics Committee)
Christian Medical College
Vellore - 632 002, Tamil Nadu, India

TEL : 0416 - 2284294, 2284202 FAX : 0416 - 2262788, 2284481 E-mail : research@cmcvellore.ac.in

Name	Hosp No	Date	LMP	GA @ R	Group	Educn
Rabiya Haleem	226925d	9/21/2011	12/25/2010	38.4	1	3
Ameena	041763d	9/22/2011	12/20/2010	39.3	1	2
Lalitha	933673d	9/26/2011	1/8/2011	37.2	2	3
Suganya	043295f	9/29/2011	12/28/2010	39.2	2	2
Priya.S	035024f	9/29/2011	9/29/2011	37	1	3
Annapoorani	932427d	9/22/2011	12/24/2011	37.5	2	4
Samundeeswari	934288d	10/4/2011	1/13/2011	37.5	2	3
Sudha.c	945274d	10/6/2011	1/20/2011	37	1	3
Nirmala	022987f	10/6/2011	1/6/2011	39	1	4
Durga	030813f	9/29/2011	1/4/2011	38.2	2	2
Divya.R	920778D	10/5/2011	1/14/2011	37.5	2	3
Zulekha	029244f	10/10/2011	1/21/2011	37.3	1	2
Sasikala	050455f	10/16/2011	1/28/2011	37.2	1	2
Kalaiyarasi	008943f	10/15/2011	1/23/2011	37.6	2	3
Malarvizhi	957481c	10/14/2011	1/28/2011	37	1	3
Madinabee	547723b	10/10/2011	1/12/2011	38.4	2	2
Assefa	044010f	10/18/2011	1/27/2011	37.4	2	2
Wahida	059303f	10/25/2011	2/7/2011	37.1	1	1
Annapoorani	953706d	10/14/2011	1/26/2011	37.2	2	2
Radha	059403f	10/27/2011	2/10/2011	37.1	1	2
unnamalai 065130f		10/28/2011	2/10/2011	37.1	1	2
Priya.v	851059d	10/31/2011	2/5/2011	38.2	2	3
Priya.l	586535d	11/21/2011	2/20/2011	39.1	2	3
Sreedevi	824822d	11/9/2011	2/12/2011	38.4	1	2
deepa.n	860013c	11/13/2011	2/22/2011	37.5	1	2
Tessina	672396d	11/9/2011	2/20/2011	37.4	2	3
Lakshmi	065305f	4/5/2011	1/5/2011	39.2	1	2
Anandi	978681d	1/4/2012	4/6/2011	39	1	2
Jamuna	790658c	1/3/2012	4/16/2011	37.3	2	2
sivasankari	966672d	12/29/2012	4/2/2011	38.4	2	2
Divisha	095223f	2/16/2012	5/22/2011	39.1	1	3
Devapriya	081887f	2/3/2012	5/15/2011	37.5	2	3
Premila	568210c	2/16/2012	6/2/2011	39.1	1	2
Manjula	871332c	2/19/2012	2/4/2011	37.1	1	2
Monica	032436b	2/3/2012	5/9/2011	38.4	2	2
Kala	147502f	2/27/2012	2/11/2011	37	2	1
Sarunya	143046f	2/20/2012	5/20/2011	39	1	2
Shyamala	024689f	3/1/2012	6/3/2011	38.2	1	3
Ramadevi	989816d	3/1/2012	6/1/2011	39.1	1	4
kalaivani	091800f	2/22/2012	6/2/2011	37.6	2	4
Yasmin	147598f	3/2/2012	6/3/2011	39	1	2
Jothi	891489d	2/25/2012	6/7/2011	37.2	2	2
Ramya	546851d	3/1/2012	6/11/2011	37.1	2	3
Jagadeswari	086763f	3/6/2012	6/15/2011	37.6	1	2

Devi	151707F	3/9/2012	6/14/2011	38.4	2	4
Maheswari	066394f	3/5/2012	6/18/2011	37.1	2	2
Hemalatha	011391F	3/11/2012	6/18/2011	39	1	2
Saranya	966935d	3/13/2012	6/13/2011	39.1	1	3
Manimegalai	022696f	3/16/2012	6/24/2011	38	1	3
Priscilla	765340B	3/15/2012	6/20/2011	38.2	2	3
Kavita	156417F	3/10/2012	6/12/2011	38.5	2	2
Kalpana	163325f	3/16/2012	6/20/2011	38.4	2	3
Asha A	154135F	3/19/2012	6/23/2011	38.4	1	3
Bharani	699787B	3/17/2012	6/20/2011	38.5	2	3
Malliga	042181f	3/21/2012	6/6/2011	37	1	3
Backialakshmi	033638F	3/20/2012	6/23/2012	37.3	1	3
Samundeswari	166577f	4/27/2012	8/7/2011	37.5	2	3
Sangeetha	189306F	4/30/2012	8/9/2011	37.6	1	2
Nishath Anjum	314465c	3/24/2012	7/25/2011	38.5	2	2
Amudha	587331c	4/23/2012	7/29/2011	38.2	2	2
Anitha B	115484f	4/21/2012	8/3/2011	37.2	2	2
Vijayalakshmy	088931f	5/5/2012	8/14/2011	37.6	1	2
Renuka	878615d	5/11/2012	8/16/2011	38.3	1	1
Saraswathy	878148d	5/19/2012	6/12/2011	38.3	1	3
Sara Thilagavathy	702234d	5/24/2012	9/3/2011	37.5	1	3
Dharani akshmi	192850f	5/3/2012	8/5/2011	38.6	2	1
Shabana	190089f	5/12/2012	8/13/2011	38.6	2	3
Malathy	108065f	5/24/2012	9/7/2011	37.1	1	3
Dagary	092523f	5/17/2012	5/19/2011	38.3	2	2
Kavitha	357701c	5/24/2012	9/8/2011	37	2	2
Afsari Banu	190936f	5/31/2012	18/08/2011 (Scan	38.6	1	2
Revathy	921374a	6/2/2012	9/13/2011	37.4	1	2
Rihana	138493f	6/5/2012	9/20/2011	37	2	2
Durga	227017f	6/15/2012	9/16/2011	39	2	2
Vanaja	501949f	7/6/2012	10/13/2011	38.1	1	2
Sumathi	193479f	7/27/2012	10/24/2011	39.4	1	2
Yasmin banu	228290f	8/31/2012	11/20/2011	37.2	1	3
Pushpalatha	135651f	7/28/2012	10/28/2011	39.2	2	2
Ahmedi Begum	176080f	7/16/2012	sedc-28/7/12	38.2	2	2
Lavanya	259615f	8/2/2012	11/10/2011	39	1	2
Vinodhini	122781f	7/28/2012	10/30/2011	38.6	2	3
Deepa	175872f	8/6/2012	11/16/2011	37.5	1	3
Pavani	547294d	7/30/2012	11/1/2011	38.5	2	3
Poornima	187008f	8/8/2012	11/19/2011	37.4	1	3
Sandhya	140221f	8/8/2012	11/19/2011	37.4	1	2
Shanti	259808f	7/30/2012	10/30/2011	38.6	2	2
Zahara	253482f	8/4/2012	11/7/2011	38.5	2	2
anitha	281742f	8/24/2012	11/29/2011	38.3	1	3
Aruna	290102f	9/1/2012	12/5/2011	38.5	2	3
Sargunam	290112f	9/10/2012	12/10/2011	39.2	1	2
Vandarkuzhali	154647f	8/30/2012	12/9/2011	37.6	2	4
Geeta S	659120D	9/18/2012	12/27/2011	38	2	2
Esther Radhika	161792F	9/9/2012	12/16/2011	38.2	1	2
Swati	165729F	9/12/2012	12/5/2011	37.3	2	3

Kalaiselvi	299103f	9/18/2012	12/22/2011	38.5	2	2
Parvathy	281747f	9/19/2012	12/28/2011	37.6	1	2
Padmavathy	303053f	9/8/2012	12/19/2011	37.4	2	3
Meena	321886d	10/4/2012	1/8/2012	38.4	2	3
Wasiya Banu	750105c	10/4/2012	1/19/2012	37.1	2	2
Jothi	245030f	10/8/2012	1/9/2012	39	1	2

Age	Obs score	BMI	GHTN in pr	OPD visits	syst.BP	Dias BP	Urine alb	headache
23	2	2	1	1	140	90	1	2
28	2	3	1	1	140	92	1	2
21	1	3	3	1	140	98	1	2
19	1	2	3	2	142	90	1	2
28	1	3	3	1	150	92	1	2
26	1	3	3	3	140	90	1	2
22	1	3	3	2	150	92	1	1
28	1	3	3	1	142	90	1	2
25	1	2	3	1	140	90	1	2
30	1	3	3	3	140	98	1	2
23	1	3	3	2	140	90	1	2
24	1	3	3	1	150	98	1	2
27	1	3	3	1	140	94	1	2
26	1	3	3	1	140	98	2	2
22	2	3	2	1	142	92	1	2
22	1	2	3	3	140	90	1	2
24	1	3	3	2	140	90	1	2
21	1	3	3	1	144	92	1	2
25	2	3	2	3	140	90	1	2
24	1	2	3	1	140	94	1	2
22	1	2	3	2	150	90	1	2
27	1	2	3	2	146	90	1	2
32	1	3	3	2	140	90	1	2
22	1	3	3	1	150	90	1	2
25	2	3	2	1	140	92	1	2
29	1	3	3	3	140	90	2	2
25	1	1	2	1	140	90	1	2
29	1	2	3	1	140	90	1	2
29	2	2	2	1	150	90	1	1
25	1	2	3	3	140	90	1	2
21	1	2	3	1	140	92	1	2
25	1	3	3	4	142	98	1	2
30	2	3	1	1	156	98	1	2
31	2	2	2	1	140	90	1	2
20	1	2	3	2	140	90	1	2
31	1	2	3	2	148	90	2	2
23	1	2	3	1	150	90	1	2
24	1	3	3	1	140	100	1	2
25	1	3	3	1	140	90	1	2
26	1	3	3	3	140	90	1	2
22	2	2	2	1	140	100	1	2
24	2	3	1	3	140	94	3	2
26	1	2	3	2	150	90	1	2
22	1	1	3	1	140	100	1	2

26	1	2	3	2	150	100	1	2
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23	1	1	3	3	140	90	1	2
23	1	2	3	1	140	90	1	2
23	1	2	3	1	146	90	1	2
30	1	3	3	2	140	90	1	2
21	1	2	3	2	150	90	3	2
27	1	2	3	2	140	100	2	2
22	1	3	3	1	140	90	1	2
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27	1	2	3	1	140	100	1	2
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28	1	3	3	3	140	90	1	2
24	1	3	3	1	140	90	1	2
28	2	2	2	3	142	90	1	2
28	2	2	1	3	140	90	1	2
29	1	2	3	4	140	90	1	2
36	1	2	3	1	150	94	1	2
22	2	1	2	1	140	90	1	2
35	1	2	3	1	140	90	1	2
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25	1	3	3	1	140	90	1	2
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19	1	3	3	2	140	90	1	2
28	1	2	3	1	150	100	1	2
23	1	2	3	3	140	94	1	2
28	2	3	2	1	150	90	1	2
26	1	2	3	4	148	90	1	2
32	1	3	3	2	140	94	1	1
30	1	2	3	1	150	90	1	2
28	1	2	3	2	150	90	1	2

27	2	2	1	2	140	90	3	2
26	1	3	3	1	150	90	1	2
29	2	2	1	4	140	90	1	2
30	2	2	2	1	144	92	1	2
28	2	3	1	1	140	90	1	2
26	1	3	3	1	150	90	1	2

BOV	< UO	Vomiting	Mod BPS	Labor	Indcn in C	GA @ del	BS	1st IOL
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2	2	2	1	2		39.4	1	9/22/2011
2	2	2	1	1		37.3	2	
2	2	2	1	2	5	39.6	2	03/10/2011
2	2	2	1	2		37.1	1	29/09/2011
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1	2	2	1	2	1	38.5	1	10/10/2011
2	2	2	1	2		37.1	1	06/10/2011
2	2	2	1	2		39.1	1	06/10/2011
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2	2	2	1	1	6	38.2	1	10/6/2011
2	2	2	1	2		37.4	1	10/10/2011
2	2	2	1	2		37.2	2	10/16/2011
2	2	2	1	2	1	38.2	1	10/16/2011
2	2	2	1	2		37.1	1	10/14/2011
2	2	2	1	2	5	39.6	1	10/17/2011
2	2	2	1	1	3	38.1	2	
2	2	2	1	2		37.1	1	25/10/2011
2	2	2	2	2	4	39	1	10/25/2011
2	2	2	1	2		37.2	1	10/27/2011
2	2	2	1	2	5	40	1	10/28/2011
2	2	2	1	1		38.5	2	
2	2	2	1	2	5	39.5	1	11/24/2011
2	2	2	1	1		38.5	1	11/9/2011
2	2	2	1	2		37.5	1	11/13/2011
2	2	2	1	2	1	38.1	1	11/13/2011
2	2	2	1	2		39.3	1	1/5/2012
2	2	2	1	2		39.1	1	1/4/2012
2	2	1	1	2	1	37.6	1	1/6/2012
2	2	2	1	2	5	39.4	1	1/5/2012
2	2	2	1	2		39.2	2	2/16/2012
2	2	2	1	2	5	39.5	1	2/16/2012
2	2	2	1	2		39.1	1	2/16/2012
2	2	2	1	2		37.2	1	2/19/2012
2	2	2	1	2	6	39.2	1	2/8/2012
2	2	2	1	2	1	37.2	1	2/29/2012
2	2	2	1	2		39.1	1	2/20/2012
2	2	2	1	2		38.3	1	3/1/2012
2	2	2	1	2		39.2	1	3/1/2012
2	2	2	1	2	3	39.2	1	3/2/2012
2	2	2	1	2		39.1	1	3/2/2012
2	2	2	1	2	1	39	1	3/7/2012
2	2	2	1	1		38.6		
2	2	2	1	2		38	1	3/6/2012

2	2	2	1	2	4	39.2	1	3/13/2012
2	2	2	1	2	1	38.1	1	3/10/2012
2	2	2	1	2		39.1	1	3/11/2012
2	2	2	1	2		39.2	2	3/13/2012
2	2	2	1	2		38.1	1	3/16/2012
2	2	2	1	2	3	38.5	2	3/19/2012
2	2	2	2	2	1	39.5	1	3/16/2012
2	2	2	1	2	1	39.2	1	3/20/2012
2	2	2	1	2		38.5	1	3/19/2012
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2	2	2	1	2		37.1	1	3/21/2012
2	2	2	1	2		37.4	1	3/20/2012
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2	2	2	1	2		38	1	4/30/2012
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2	2	2	1	2	3	39.3	1	4/30/2012
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2	2	2	1	2		38.4	1	5/11/2012
2	2	2	1	2		38.4	1	5/19/2012
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2	2	2	1	2	5	39.6	2	5/10/2012
2	2	2	1	2	5	39.6	2	5/19/2012
2	2	2	1	2		37.2	1	5/24/2012
2	2	2	1	2	1	39.3	1	5/24/2012
2	2	2	1	2	6	37.5	1	5/29/2012
2	2	2	1	2		39	1	5/31/2012
2	2	2	1	2		37.5	1	6/2/2012
2	2	2	1	2	3	38.1	1	6/12/2012
2	2	2	1	2	3	39.4	1	6/18/2012
2	2	2	1	2		38.2	1	7/6/2012
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2	2	2	1	2	5	39.6	1	7/28/2012
2	2	2	1	2		39.1	1	8/3/2012
2	2	2	1	2	5	40	1	8/5/2012
2	2	2	1	2		37.6	1	8/7/2012
2	2	2	1	2	5	39.6	1	8/6/2012
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2	2	2	1	2		39.6	1	8/5/2012
2	2	2	1	2	5	39.6	1	8/12/2012
2	2	2	1	2		38.5	1	8/25/2012
2	2	2	1	2	5	39.6	1	9/8/2012
2	2	2	1	2		39.2	1	9/10/2012
2	2	2	1	2	5	39.5	1	9/11/2012
2	2	2	1	2	6	39.1	1	9/24/2012
2	2	2	1	1		38.3	1	9/9/2012
2	2	2	1	1	3	38.3	1	9/18/2012

2	2	2	1	2	1	39.1	1	9/21/2012
2	2	2	1	1		38	1	9/19/2012
2	2	2	1	1		39.4	2	
2	2	2	1	1		39.2	2	
2	2	2	1	1		37.5	2	
2	2	2	1	2		39	1	10/8/2012

GA	Re-IOL	GA-Re IOL	Attempts	ROM to del	MOD	sev.HTN	PE	Mild/sev PI
38.4			1	4	1.00	2	2	
39.3			1	7	1.00	2	2	
				6	1.00	2	2	
39.5			1	16	4.00	2	2	
37			1	17	3.00	2	2	
				4	1.00	2	2	
38.4			1	13	4.00	1	1	2
37			1	8	4.00	2	1	1
39			1	8	1.00	2	2	
39.2			1	14	4.00	2	2	
38.1			1	12	3.00	2	2	
37.3			1	16	1.00	2	2	
37.2			1	6	1.00	2	2	
38.1			1	10	3.00	2	1	1
37			1	11	1.00	2	2	
39.5			1	10	3.00	2	2	
				10	4.00	2	2	
37.1			1	7	4.00	2	2	
38.6			1	5	1.00	2	2	
37.1			1	8	1.00	2	1	1
37.1	11/16/2011	39.6	2	7	3.00	2	2	
				14	3.00	2	2	
39.4			1	8	4.00	2	2	
38.4			1	12	4.00	2	2	
37.5			1	7	1.00	2	2	
38			1	12	4.00	2	1	1
39.2			1	7	1.00	2	2	
39			1	18	4.00	2	2	
37.5			1	6	1.00	2	1	2
39.4			1	8	4.00	2	2	
39.1			1	8	1.00	2	2	
39.4			1	20	3.00	1	1	2
39.1			1	4	2.00	2	2	
37.1			1	13	1.00	2	2	
39.1			1	8	3.00	2	2	
37.2			1	16	4.00	2	1	2
39			1	18	3.00	2	2	
38.2			1	12	4.00	2	2	
39.1			1	16	4.00	2	2	
39.1			1	20	3.00	2	2	
39			1	7	4.00	2	2	
38.6			1	10	1.00	2	1	2
			2	4	1.00	2	2	
37.6			1	10	1.00	2	2	

39.1	1	12	4.00	2	2	
38	1	20	2.00	1	1	2
39	1	14	1.00	2	2	
39.1	1	16	4.00	2	2	
38	1	10	1.00	2	2	2
38.5	1	10	1.00	2	1	1
39.4	1	12	4.00	1	1	2
39.1	1	12	4.00	2	1	1
38.4	1	18	1.00	2	2	2
		14	1.00	2	2	2
37	1	10	1.00	2	2	2
37.3	1	12	1.00	2	2	2
39.2	1	10	3.00	2	2	2
37.6	1	8	4.00	2	1	2
39.5	1	26	4.00	2	2	2
39.2	1	12	1.00	2	2	2
39.6	1	10	4.00	2	2	2
37.6	1	6	1.00	2	2	
38.3	1	7	1.00	2	2	
38.3	1	6	4.00	2	2	
37.5	1	1	4.00	2	1	1
39.5	1	8	3.00	2	2	
39.6	1	3	1.00	2	2	
37.1	1	10	3.00	2	1	1
39.3	1	12	3.00	2	1	1
37.4	1	8	1.00	2	2	
38.6	1	14	3.00	2	2	
37.4	1	10	2.00	1	1	2
38	1	12	1.00	2	2	
39.3	1	18	1.00	2	2	
38.1	1	12	4.00	2	2	
39.4	1	14	4.00	2	2	
37.2	1	12	4.00	2	2	
39.5	1	12	2.00	2	2	
39.5	1	1	4.00	2	2	
39	1	9	2.00	2	2	
39.6	1	9	3.00	2	2	
37.5	1	8	4.00	2	2	
39.5	1	3	1.00	2	2	
37.4	1	5	1.00	2	2	
37.4	1	7	1.00	2	2	
39.5	1	6	1.00	2	2	
39.5	1	8	1.00	2	2	
38.4	1	6	2.00	2	2	
39.5	1	6	1.00	2	2	
39.2	1	2	1.00	2	2	
39.4	1	15	1.00	2	2	
39	1	7	1.00	2	2	
38.2	1	12	2.00	2	2	
38.2	1	23	1.00	2	2	

39.1	1	4	4.00	2	1	2
37.6	1	14	4.00	2	2	
		5	1.00	2	2	
		3	1.00	2	2	
		3	1.00	2	2	
38.6	1	5	1.00	2	2	

eclampsia	HELLP	Pulm.oed	RF	Throm.	Abruption	gr.abr	PPH	Transfusior
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ICU-Mothe	ICU-No	hosp days	antihyper	intrapar/pc	Mgso4	M mort	IUD	ND
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2		4	2		2	2	2	2
2		4	2		2	2	2	2
2		3	1	2	2	2	2	2
2		4	1	1	1	2	2	2
2		9	2		2	2	2	2
2		4	2		2	2	2	2
2		6	2		2	2	2	2
2		7	2		2	2	2	2
2		8	2		2	2	2	2
2		5	1	1	1	2	2	2
2		4	2		2	2	2	2
2		4	2		2	2	2	2

2	7	2		2	2	2	2
2	6	1	1	1	2	2	2
2	3	2		2	2	2	2
2	7	2		2	2	2	2
2	7	2		2	2	2	2
2	5	2		2	2	2	2
2	6	1	1	1	2	2	2
2	6	1	1	2	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	6	2		2	2	2	2
2	8	2		2	2	2	2
2	4	2		2	2	2	2
2	5	1	2	1	1	2	2
2	5	2		2	2	2	2
2	5	2		2	2	2	2
2	3	2		2	2	2	2
2	3	2		2	2	2	2
2	5	2		2	2	2	2
2	7	2		2	2	2	1
2	12	2		2	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	7	2		2	2	2	2
2	3	2		2	2	2	2
2	5	1	1	2	2	2	2
2	7	2		2	2	2	2
2	5	1	1	1	2	2	2
2	4	2		2	2	2	2
2	6	2		2	2	2	2
2	6	2		2	2	2	2
2	4	2		2	2	2	2
2	6	2		2	2	2	2
2	4	2		2	2	2	2
2	5	2		2	2	2	2
2	5	2		2	2	2	2
2	6	2		2	2	2	2
2	7	1	1	1	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	3	2		2	2	2	2
2	7	2		2	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	3	1	2	2	2	2	2
2	4	2		2	2	2	2
2	4	2		2	2	2	2
2	3	2		2	2	2	2
2	5	2		2	2	2	2

2	6	1	1	1	2	2	2
2	8	2		2	2	2	2
2	3	1	1	2	2	2	2
2	4	2		2	2	2	2
2	2	2		2	2	2	2
2	5	2		2	2	2	2

BW	APGAR	Cord ph	NICU	NICU-No	sex	total cost	
2780	1	1	2		2	8250	
3100	1	1	2		1	10113	
3320	1	1	2		1	8315	
3521	1	1	2		1	20021	ARREST OF DIL
2800	1	1	2		1	11650	
3020	1	1	2		2	12932	
2940	1	1	2		2	21206	failed IOL
2380	1	1	2		2	15978	unfav cervix
2660	1	1	2		1	16211	
3140	1	1	2		1	18146	,150/106
2800	1	1	2		2	17,350	
2600	1	1	2		2	11980	
2740	1	1	2		1	10905	
3780	1	1	2		1	10850	
3210	1	1	2		2	9132	
2900	1	1	2		2	13220	
3160	1	1	2		1	18993	NRFS
2280	1	1	2		2	16537	NRFS
2800	1	1	2		2	8110	
3520	1	1	2		2	8728	
2700	1	1	2		2	16,745	lost to follow up in betw
3800	1	1	2		2	13290	
3180	1	1	2		1	26280	
4100	1	1	2		1	18307	
3010	1	1	2		1	11040	
2920	1	1	2		1	20835	failed IOL
2530	1	1	2		1	20988	
3300	1	1	2		2	20375	NRFS
3005	1	1	2		1	8935	
3380	1	1	2		1	24781	nrfs
2960	1	1	2		1	8876	
2800	1	1	2		2	17461	3rd degree tear
3030	1	1	2		2	10553	
2370	1	1	2		1	9043	
3200	1	1	2		2	10942	
2480	1	1	2		1	21414	
2520	1	1	2		1	18395	
2680	1	1	2		2	19625	failed iol
2740	1	1	2		1	20815	NRFS
3060	1	1	2		1	16065	
2940	1	1	2		1	27903	Chorioamnionitis with N
2100	1	1	2		2	10755	neuro d-12-CVT-9D-ant
2550	1	1	2		2	10327	
3000	1	1	2		1	11274	3 deg PT

2960	1	1	2		1	24772	failed iol
3090	1	1	2		1	13385	failed iol
2420	1	1	2		2	9387	
2780	1	1	1		1	25992	arrest of dilatation
2180	1	1	2		2	12905	
3300	1	1	2		2	17212	
2140	1	1	2		2	25320	failed iol
2940	1	1	2		1	25199	
2480	1	1	2		2	9540	
3300	1	1	2		2	9525	
2500	1	1	2		2	11415	
3300	1	1	2		1	12765	
2800	1	1	2		1	11382	
2820	1	1	1		1	25355	nrfs
2890	1	1	2		2	22843	failed iol
2630	1	1	2		2	11750	
2560	2 (5)	2 (7.16)	2		1	20967	nrfs
2530	1	1	2		2	8430	4th degree tear
2780	1	1	2		2	9755	
3570	2 (4, 6)	3 (6.9)	1	6	1	75355	nrfs
2620	1	1	2		1	39006	cord prolapse, puerpera
3470	1	1	2		2	10145	
2610	1	1	2		1	10556	
2720	1	1	2		2	24666	
3380	1	1	2		1	9685	
3460	1	1	2		1	11750	
2980	1	1	2		2	12263	
3230	1	1	2		1	12128	
2760	1	1	2		1	9666	
2860	1	1	2		1	15656	
2900	1	1	2		1	22334	failed iol
2841	1	1	2		2	22337	failed iol
3080	1	1	2		1	25993	failed iol
3580	1	1	2		1	11475	NRFS
2520	1	1	2		2	23742	NRFS
3500	1	1	2		1	14100	NRFS
3520	1	1	2		2	14042	prolonged 2nd stage
2620	1	1	2		2	47540	APE in early labour
4030	1	1	2		1	14280	
3040	1	1	2		2	11298	
2880	1	1	2		2	8850	
2600	1	1	2		2	11755	
3750	1	1	2		1	14275	fever
2380	1	1	2		2	13025	
3270	1	1	2		2	13853	
2410	1	1	2		2	9600	
3400	1	1	2		2	12427	
3230	1	1	2		1	10055	
3620	1	1	2		1	9315	
3110	1	2	1	1	1	15445	apgar 2/7/10

2460	1	1	2	2	23819	NRFS
3460	1	1	2	1	24462	phototherapy
2800	1	1	2	1	10155	
3012	1	1	2	2	9995	
3500	1	1	2	2	7970	
2500	1	1	2	1	12785	

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IRFS
iepileptic and warf

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ABSTRACT

TITLE OF THE ABSTRACT :

Induction of labour versus conservative management for mild gestational hypertension at term

DEPARTMENT :

Obstetrics and Gynaecology, Christian Medical College, Vellore

NAME OF THE CANDIDATE :

Dr. Bhageerathy. P.S

DEGREE AND SUBJECT :

MD Branch II – Obstetrics and Gynaecology

NAME OF THE GUIDE :

Dr. Ruby Jose

OBJECTIVES: Describe the objectives of your study (maximum 30 words)

To study whether induction of labour in term pregnant women with gestational hypertension reduces maternal and neonatal morbidity, mortality and expenditure compared to conservative management without increasing caesarean section rates.

METHODS: Explain the clinical and statistical methods used (maximum 100 words)

Eligible patients presenting to the Obstetric outpatient department of Christian Medical College, Vellore between 37 and 40 weeks from September 2011 to October 2012 with gestational hypertension (systolic blood pressure of 140 to 159 mm of Hg and diastolic blood pressure of 90 to 100 mm Hg) are randomised in 1:1 ratio to either induction of labour or conservative management after taking consent. Patients in the conservative management arm are monitored biweekly for any maternal or fetal compromise

till 39 weeks and 5 days when they are induced. Women in the induction group were induced within 12 hours of randomisation. Details of delivery, any maternal or neonatal complications were recorded and analysed using Chi Square test for categorical variables and Student's t test for comparison of continuous variables. A p value of less than 0.05 denoted statistical significance.

RESULTS: Summarise the findings and conclusions of your study (maximum 90 words)

1. Though a higher rate of progression to severe hypertension and preeclampsia in the conservative group was noticed, this was not statistically significant.
2. There was no statistically significant difference in the incidence of complications like eclampsia, postpartum haemorrhage,thrombosis,HELLP syndrome,pulmonary oedema,renal failure, abruptionplacenta or maternal mortality .
3. There was no statistically significant difference in caesarean section rates between the 2 groups.
4. There was no statistically significant difference in neonatal morbidity and mortality.
5. A slight increase in the median total cost was found in the conservative management group.

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E-mail	drbhagee@gmail.com
Submission time	17-Dec-2012 01:29AM
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First 100 words of your submission

INDUCTION OF LABOUR VERSUS CONSERVATIVE MANAGEMENT FOR MILD GESTATIONAL HYPERTENSION AT TERM A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE RULES AND REGULATIONS FOR THE MD BRANCH II (OBSTETRICS AND GYNAECOLOGY) DEGREE EXAMINATION OF THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY TO BE HELD IN APRIL 2013 CERTIFICATE This is to certify that the dissertation entitled “Induction of labour versus conservative management for mild gestational hypertension at term” is the original work of Dr. Bhageerathy P.S towards the M.D Branch II (Obstetrics and Gynaecology) Degree examination of The Tamil Nadu Dr.M.G.R Medical University, Chennai to be held in April 2013 Signatures Guide: Head of the...