COMPARATIVE ANALYSIS OF SAFETY,EFFICACY AND FETOMATERNAL OUTCOME FOLLOWING INDUCTION OF LABOUR WITH MIFEPRISTONE VS INTRACERVICAL DINOPROSTONE GEL.

Dissertation submitted to

In partial fulfillment of the requirements for the degree of

M.S BRANCH II

OBSTETRICS AND GYNAECOLOGY

THANJAVUR MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

May 2018

CERTIFICATE

This is to certify that the dissertation entitled "**COMPARATIVE ANALYSIS OF SAFETY,EFFICACY AND FETOMATERNAL OUTCOME FOLLOWING INDUCTION OF LABOUR WITH MIFEPRISTONE VS INTRACERVICAL DINOPROSTONE GEL**." is the bonafide original work of **Dr. A.SARANYA** under the guidance of **Prof. Dr. S.PRADEEBA MD,** HOD, Department of Obstetrics and Gynecology, T.M.C.H. Thanjavur in partial fulfillment of the requirements for the degree of M.S branch II Obstetrics and Gynecology examination of the Tamilnadu Dr. M.G.R Medical University to be held in May 2018. The period of study was from Aug 2016 to July 2017.

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COMPARATIVE ANALYSIS OF SAFETY, EFFICACY AND FETOMATERNAL OUTCOME FOLLOWING INDUCTION OF LABOUR WITH MIFEPRISTONE VERSUS INTRACERVICAL DINOPROSTONE GEL

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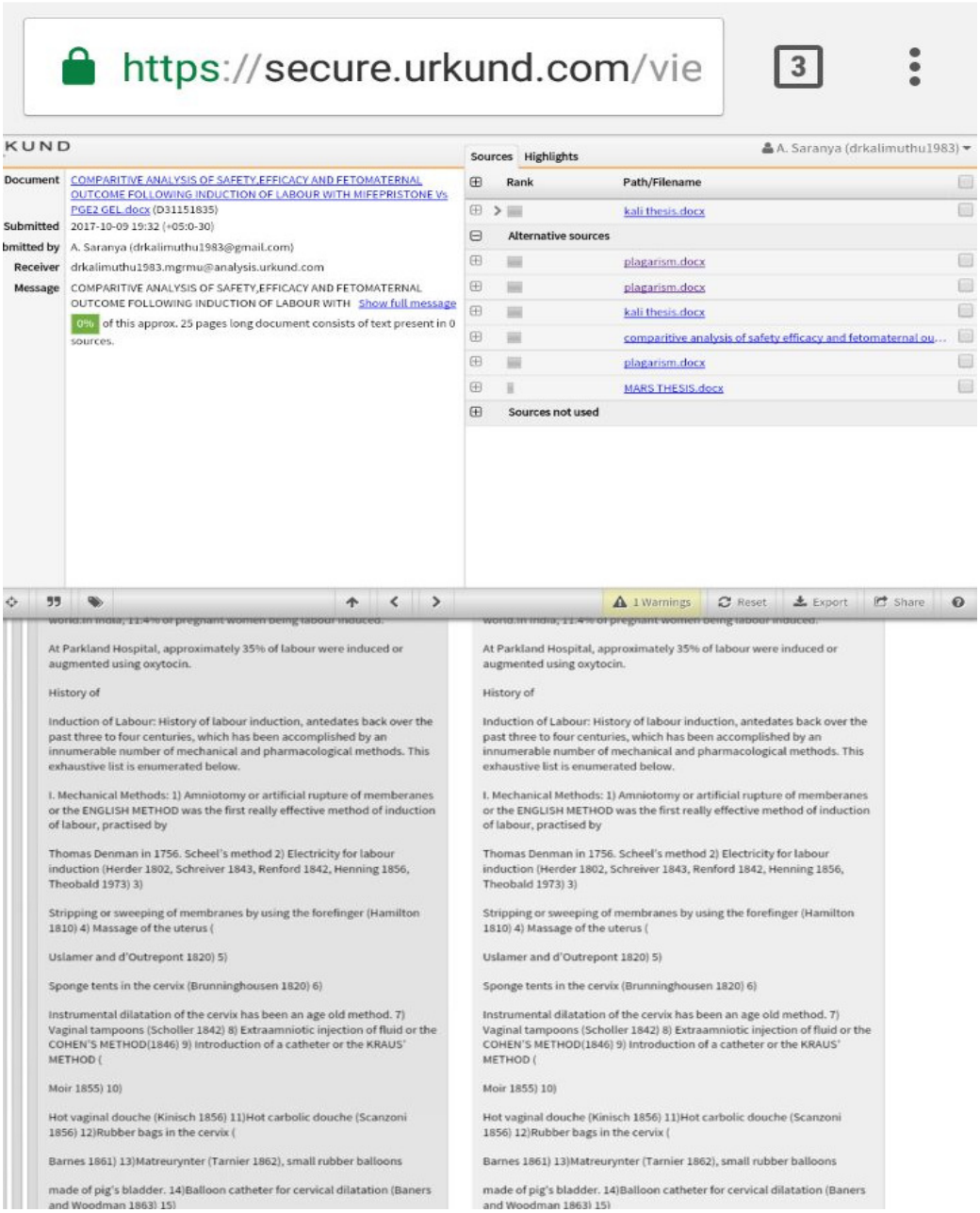

At Parkland Hospital, approximately 35% of labour were induced or augmented using oxytocin.
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CERTIFICATE – II

This is to certify that this dissertation work titled **COMPARATIVE ANALYSIS OF SAFETY, EFFICACY AND FETOMATERNAL OUTCOME FOLLOWING INDUCTION OF LABOUR WITH MIFEPRISTONE VERSUS INTRACERVICAL DINOPROSTONE GEL** of the candidate **DR.SARANYA.A** with registration Number **221516203** for the award of the degree of in the branch of M.S Obstetrics & Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from Introduction to conclusion pages and result shows 0 percentage of plagiarism in the dissertation.

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DECLARATION

I solemnly declare that this dissertation titled "**COMPARATIVE ANALYSIS OF SAFETY,EFFICACY AND FETOMATERNAL OUTCOME FOLLOWING INDUCTION OF LABOUR WITH MIFEPRISTONE VS INTRACERVICAL DINOPROSTONE GEL**" was done by me at department of Obstetrics And Gynaecology ,Thanjavur Medical College during the year 2015-2018 under the guidance and supervision of **Prof.Dr.S.Pradeeba MD;OG.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical university towards the partial fulfillment of requirements for the award of M.D degree in Obstetrics &Gynaecology (Branch-II) .

Dr. A.SARANYA

ACKNOWLEDGEMENT

I am extremely thankful to **Prof. Dr..Jayakumar M.Ch** Dean Government Thanjavur medical College and Hospital, Thanjavur who has granted permission to do this study in this institution.

I take this opportunity to express my deepest sense of gratitude to **Prof. Dr S.Pradeeba M.D,(**OG) Head of the Department of obstetrics and Gynecology Thanjavur Medical College, Thanjavur for encouraging me and rendering timely suggestions and guiding me throughout the course of this work. I will be forever indebted to her for her constant support.

I am extremely thankful to **Prof. Dr.R.Raja Rajeshwari,M.D.,D.G.O., D.N.B.,** for her extensive support, advice and guidance in the analysis and successful Completion of this study.

I sincerely thank my co-guide **Dr.V.Thendral M.D(OG).,** Assistant Professor of the Department of Obstetrics and Gynecology for her constant encouragement and guidance throughout my study.

I express my gratitude to all Assistant Professors of our department for their valuable guidance and suggestions that made this work possible.

I wish to thank **Dr.Maheshwaran MD**, for his excellent support in statistical analysis. I wish to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement. Above all my sincere thanks to antenatal mothers of TMCH without whom this study would not have been possible.

LIST OF ABBREVIATION

ACOG	- American College of Obstetrics and Gynecology
RCOG	- Royal College of obstetrics and gynecology
PGE1	- Prostaglandin E1
PGE2	- Prostaglandin E2
PGF2 α	- Prostaglandin F2 alpha
IUD	- Intra uterine death
ARM	- Artificial rupture of membrane
GI - SYMPTOMS	- Gastrointestinal symptoms
PPH	- Post partum haemorrhage.
MAS	- Meconium aspiration syndrome
NICU	- Neonatal intensive care unit
NN Mortality	- Neonatal mortality
PR	- Progesterone receptor

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INTRODUCTION

Human parturition is termed as 'labour' in recognition of the hard work that the parturient as well as the uterine myometrium have to perform in order to deliver the fetus. Labour is refered to the onset of effective uterine contractions leading to progressive effacement and dilatation of the cervix resulting in the expulsion of the fetus, placenta and the membranes.1

According to Turnbull (1976)- "**The spontaneous onset of labour is a robust and effective mechanism…. And should be given to operate on its own. We should only induce labour when we are sure that we can do better**"

The most important decision to be made when considering induction of labour is whether or not the induction is justified. How it is to be achieved, is a secondary decision. Whatever method is chosen to implement a justified decision to induce labour, uterine contractility, maternal and fetal wellbeing should be monitored carefully.

Induction is indicated when the benefits for the mother and fetus outweigh those of continuing the pregnancy and to achieve vaginal delivery, thus avoiding an unnecessary caesarean section 2.The more

common indications include membrane rupture without labor, gestational hypertension, oligohydromnios, nonreassuring fetal staus, postterm pregnancy, various maternal medical conditions such as chronic hypertension and diabetes(ACOG,2013b)[3].

Compromise to maternal longevity, accounts for the majority of indications for induction of labour, while the wide diversity of fetal indications are most often not compromising to their survival or morbidity.

Favourability of the cervix is a need for labour induction. Research in this direction has helped in the development of various methods to 'ripen' the cervix prior to uterine contractions.

The discovery of prostaglandins, and lately the antiprogesterones, have made labour induction at the disposal of the obstetrician, enabling the delivery of the patient as and when required, thus allowing a carefully planned active management, and in bringing down the trauma of a prolonged or protracted labour for the patient, to give her a healthy baby without compromising her health.

AIM OF THE STUDY

1.To study the efficacy of Mifepristone and Dinoprostone as a cervical ripening/priming agent for induction of labour.

2.To study maternal and fetal outcome.

REVIEW OF LITERATURE

Induction of labour:

Induction of labour is one of the most commonly performed interventions in modern obstetrics with upto 20% of pregnant women being labour induced by one or the other.

Induction implies stimulation of contractions before the spontaneous onset of labour,with or without ruptured membranes after the period of viability. When the cervix is closed and uneffaced, labour induction will commence with cervical ripening, a process that generally employs prostaglandins to soften and open the cervix.

Induction rates have been influenced by several reports worldwide, which claimed that an active induction policy, led to substantial reduction in perinatal and maternal morbidity and mortality.

The incidence of induction of labour varies widely in different parts of the world. It is 10-15% in developing countries and 10-25% in the developed world.In India, 11.4% of pregnant women being labour induced.At Parkland Hospital, approximately 35% of labour were induced or augmented using oxytocin.

History of Induction of Labour:

History of labour induction, antedates back over the past three to four centuries, which has been accomplished by an innumerable number of mechanical and pharmacological methods. This exhaustive list is enumerated below.

I. Mechanical Methods:

1) *Amniotomy or artificial rupture of membranes or the ENGLISH METHOD* was the first really effective method of induction of labour, practised by Thomas Denman in 1756. Scheel's method
2) *Electricity for labour induction* (Herder 1802, Schreiver 1843, Renford 1842, Henning 1856, Theobald 1973)
3) *Stripping or sweeping of membranes by using the forefinger* (Hamilton 1810)
4) *Massage of the uterus* (Uslamer and d'Outrepont 1820)
5) *Sponge tents in the cervix* (Brunninghousen 1820)
6) *Instrumental dilatation of the cervix* has been an age old method.
7) *Vaginal tampoons* (Scholler 1842)
8) *Extraamniotic injection of fluid* or the COHEN'S METHOD(1846)

9) *Introduction of a catheter* or the KRAUS' METHOD (Moir 1855)

10) Hot vaginal douche (Kinisch 1856)

11) Hot carbolic douche (Scanzoni 1856)

12) Rubber bags in the cervix (Barnes 1861)

13) Matreurynter (Tarnier 1862), small rubber balloons made of pig's bladder.

14) Balloon catheter for cervical dilatation (Baners and Woodman 1863)

15) Laminaria tent (Wilson 1865)

16) Cervical tampoons (Kehrer 1888)

17) Paracentesis of amniotic fluid usually with injection of irritant Solutions

18) Massage of the breasts (Friedricgh 1939)

19) Extra amniotic saline infusion – EASI (Schreyer 1989)

20) *Hygroscopic cervical dilators* (Kramner 1995, Gilson 1996)

II. Pharmacological Methods:

1. ***Oxytocin*** is the first polypeptide hormone synthesized, which is an important milestone in labour induction. Its discovery won a Nobel

prize for Du Vigneaud in 1953 and the efforts of Turnbull and Anderson (1968) led to its acceptance in routine obstetric practice.

However, it was noted that this method of induction resulted in more postpartum hemorrhage than induction with prostaglandins (Howarts and Botha 2001). When compared to induction with prostaglandins, evidence suggests that oxytocin induction is associated with a lower chance of delivery within 24 hours; there was no difference in the rate of cesarean section. However, subgroup analysis reveals more information showing that:

- "In primigravid women, there is reduction in the number of women satisfied with the method of induction with oxytocin".
- "In women with unfavorable cervix, oxytocin induction is associated with a higher rate of cesarean section".
- "In women with favorable cervix, induction with oxytocin was associated with greater satisfaction".

2. The discovery of Prostaglandins in the 1930's from human semen and its elucidation in the biological role in the parturition process, has revolutionized the process of labour induction and has been the greatest armamentorium in the induction of labour for the present day obstetricians.

Prostaglandins are autocoids detected in almost all tissues and body fluids including lungs, heart, stomach, adrenals, liver, spleen, kidney, central nervous system, uterus, vesical gland and seminal fluid. Named by Von Euler of Sweden in 1935, who extracted it from the seminal vesicle. Sune Bergstrom of Sweden received a Nobel Prize for its synthesis in 1932.

Most protocols recommend the use of intracervical prostaglandin in women with an unfavourable cervix and intact membranes with Bishop's score less than [4].

Meta-analysis by the Clinical Effectiveness Support Group at the Royal College of Obstetricians and Gynecologists showed improved rates of successful vaginal delivery, lower rates of cesarean section and higher levels of maternal satisfaction in women induced with prostaglandin when compared with oxytocin. However, amniotomy and oxytocin infusion are effective in women with a favourable cervix and in areas where resources are limited, the cheapness of this method may outweigh the consideration of maternal satisfaction. Vaginal PGE2 tablets appear to be as effective as gel formulations. In 1992 FDA approved PGE2 (0.5mg intracervically) for cervical ripening and labour induction.

Misoprostol, a synthetic analogue of prostaglandin E1, is less expensive, more stable and easier to store than PGE2. These factors have led to the suggestion that misoprostol will allow the use of prostaglandin for induction in areas of the world that have previously been unable to afford this luxury (E1 Refaey and Jauniaux 1997). In UK and USA, the drug has a licence currently for the treatment of peptic ulcer but has no licence for the induction of labour[28].

Although the manufacturers have indicated that they do not intend to pursue licensing for this purpose, the American College of Obstetricians and Gynecologists has issued statements that misoprostol is a safe and effective drug for the induction of labour when appropriately used (ACOG 1999, 2000)[2]. In the UK the Royal College of Obstetricians has remained more cautious, agreeing that misoprostol is a cheap and effective agent for inducing labour but due to safety concerns, feel that further clinical trials are required prior to recommending it's use in general obstetric practice (RCOG 2001a). Despite lot of clinical use misoprostol is still not approved by Drug controller of India for use in labour induction.

3. Mifepristone or RU 486, an antiprogesterone is a receptor level antagonist, licensed in U.K in July 1991. Mifepristone ,19-norsteroid has great affinity for the progesterone receptor and thus blocks the action of progesterone at cellular level. As fall in the level of progesterone, is considered one of the important events in the onset of spontaneous labour, it therefore seems that this drug may be useful in labour induction[4].

A number of studies have looked at the efficacy of mifepristone in cervical ripening.

"Although more studies are needed, it is found that mifepristone is a safe, efficient and suitable induction agent for initiation of labour in women at term"(Frydman et al1992).

"When compared to placebo, 200mg oral mifepristone increases the chances of spontaneous labour and reduces the need for prostaglandins" (Lelaidier et al 1993)[18].

"200mg Mifepristone was significantly more likely to result in a favorable cervix than placebo"(Elliot et al,1998)

"Mifepristone is a simple and effective treatment for inducing labor in post-term women with an unripe cervix"(Stenlund 1998)

"Mifepristone proved effective for cervical ripening and reduced the time to delivery compared with placebo, but it did not improve the rate of cesarean. The study did not include enough pregnancies to reach conclusions about fetal or neonatal safety"(Giacalone,1998)

"There is reduction in the induction delivery interval when induction is performed with Mifepristone and a trend to a reduction in the rate of cesarean section"(Wing et al 2000)[17].

"Studies suggest that mifepristone is better than placebo in reducing the likelihood of cesarean sections being performed for failed induction of labour, thereby justifying future trials comparing mifepristone with the routine cervical ripening agents currently in use"(Neilson JP, Cochrane Database Sys Rev 2000)

"Studies revealed that mifepristone was well tolerated by the mother and fetus"(Nadia Berkane, Lieve Verstraete 2004) American journal of obstetrics and Gynaecology 2005

"Women who were induced with 200mg mifepristone showed drastic improvement in cervical score within 24-48hours and decreased the cesarean rate in the study group and amount of dose requirement of

augmentation of labour with misoprostol or oxytocin, lesser NICU admission and maternal complication"(Rutuja Athawale,2013)

"It is found that there is significant improvement in mean Bishop's score at the end of 24hours when induction is performed with 200mg oral Mifepristone .This improvement in score indirectly indicates the withdrawal of progesterone support"(Sonali Deshpande,2014)

"The study reveals that oral mifepristone is very safe and an effective drug for preinduction cervical ripening with added advantage of ease of administration, better patient compliance and acceptance , reduced oxytocin requirement , shorter duration of 2^{nd},3^{rd} stages of labor and less blood loss" (Arumugaselvi,2017) International journal of scientific study 2017

These studies in recent literature over the last two decades, shows the efficacy of mifepristone not only in first and second trimester induced medical abortions, but also its use as a safe, orally effective cervical ripening and labour inducing agent.

Other Therapeutic Agents:

Purified Porcine Ovarian Relaxin (1-4mg)

Relaxin has been used both vaginally and intracervically to induce labour but studies have failed to show any benefit when compared to prostaglandin (Kelly 2001)[1].

Hyaluronidase

Hyaluronidase given by cervical injection has been postulated to increase cervical softening by increasing tissue water content. The problems associated with its administration and the lack of evidence of any benefit associated with its use, is such that its use cannot be recommended.(Kavanagh 2001)[1]

Estradiol

Estradiol in tylose gel is not commonly used as an induction agent but has been previously used in the belief that it may stimulate prostaglandin release. There is no evidence to confirm or refuse their efficacy and their use is therefore of historical interest only(Luther 1980).

Indications for Induction of Labour

The indications for induction of labour are, where the benefits to either mother or fetus outweigh those of pregnancy continuation

There are two main types of induction, namely

a) Indicated Induction

b) Elective induction.

A.Indicated Induction 3

Commonly accepted indications

- Gestational hypertension
- Prelabour rupture of membranes
- Chorioamnionitis
- Severe intrauterine growth restriction
- Isoimmunization
- Maternal medical problems

❖ Diabetes mellitus

❖ Renal disease

❖ Lupus

- Fetal demise
- Prolonged pregnancy
- Oligohydramnios

- Abruptio placentae
- Fetal malformations incompatible with life

B. **Elective induction**

Logistic factors such as distance from the hospital or a history of rapid labor and delivery or psychosocial indications may be reasonable indications. But elective induction(without medical or obstetric indications) is generally not recommended because of the increased risks for adverse maternal outcomes and adverse neonatal morbidity.

Contra indications

1. When vaginal delivery is contraindicated-

    (a) Major degrees of cephalo pelvic disproportion

    (b) Previous VVF repair

    (c) Pelvic tumour

    (d) Carcinoma cervix

    (e) Previous uterine surgery disruption

    (f) Active gential herpes infection.

2. Malpresentations.

3. Placental abnormalities like Vasa praevia and Type III and IV Placenta Praevia.

4. Appreciable macrosomia

5. Severe hydrocephalus

6. Non reassuring fetal heart rate

7.Hypersensitivity to cervical ripening agents

Outcome of Induction

Factors influencing the outcome of induction

The process of prelabour cervical softening and dilatation is a part of a continuum, which culminates in spontaneous labour.

The success of any method of induction depends largely on

(1) Parity

(2) The state of cervix at the beginning of induction,

(3)Body mass index<30,

(4)Birthweight <3500grams.

In most centers, modified Bishop score (1964)Burnett is used to assess the favourability of the cervix both prior to and following induction.5 The partogram aids in assessing the progess of labour.

Some definitions, useful for assessing the success or failure of induction are enlisted below.

Successful induction

Successful induction is defined as "Vaginal delivery of an infant in good condition with minimum maternal discomfort and side effects,within a specified framework of time"

Failed induction

Defined by Duff et al (1984), as the failure to enter the active phase of labour, after twelve hours of regular uterine contractions.

Failed induction, is diagnosed when, a patient who was induced, does not deliver vaginally, in the absence of fetal distress, with acute events like abruption or cord prolapse and failure of progress due to cephalopelvic disproportion or malposition and or if the patient has not entered the active phase of labour despite adequate management for twelve hours (Arulkumaran et al 1985).

According to ACOG,SMFM & NICHD,"failed induction should be diagnosed if there is failure to generate regular contractions and cervical change for atleast 24 hours of oxytocin administration with artificial rupture of membranes if feasible,in the absence of fetal heart rate abnormalities".

PROLONGED PREGNANCY

Prolonged pregnancy as endorsed by the International Federation of Gynaecologist and Obstetricians and the American College of Obstetricians and Gynaecologists(2004),is 42 completed weeks (294 days) or more from the first day of the last menstrual period.

Common causes:

1. Wrong dates
2. Primiparity
3. Previous post-term delivery
4. Male fetus
5. Obesity
6. Anencephaly
7. Genetic factors

Problems

1. Chances of fetal distress are higher in prolonged pregnancy
2. Less liquor may predispose to cord compression and fetal distress.
3. Meconium may be frequent problem since all fetuses of this gestational age have a readily activated vagal system.Since the liquor volume may be less, the meconium will become thick and tenacious.
4. Large babies may lead to prolonged labour.

Induction of labour after 41 weeks has been found to decrease perinatal mortality and morbidity, without increasing the maternal risks of cesarean section. SOGC(2013) recommends induction of labour at 41completed weeks, as evidence reveals a decrease in perinatal mortality without increased risk of cesarean section

In a survey done 10years ago, cleary-goldman and associates(2006) reported that 73% of members of the ACOG routinely induced women at 41 weeks.

Methods of Induction

There are only three existing broad approaches in induction of labour practiced in the current obstetric practise. They are:

a) Amniotomy or Artificial rupture of membranes

b) Stripping of membranes or Sweeping of membranes.

c) Use of oxytocic agents

(a) Amniotomy or Artificial rupture of membranes(Surgical induction)

Introduced by Thomas Denman more than 200 years ago, the procedure represents one of the most irrevocable interventions in pregnancy, and more than any other procedure calls for a firm

commitment to delivery within a short time to avoid the risk of maternal and fetal infection.

Amniotomy alone often results in vaginal delivery in most women with good cervical score. However, Patterson in 1971 found that 15% of primigravidas and 22% of multigravidas were not in established labour more than 24 hours after amniotomy. Therefore in current obstetric practice, amniotomy is combined with oxytocin immediately or after a variable interval. After controlled artificial rupture of membranes without dislodging the presenting part amniotic fluid is allowed to drain, colour of liquor and any cord prolapse noted.

There are two types of rupture of membranes – *Low rupture of membranes* (LARM) done by using a kocher's artery forceps and high rupture of membranes or hind water amniotomy done using a Drew-Smythe catheter. Low rupture of membranes is the basic procedure in induction of labour.

Prerequisites

- Vertex presentation
- Cervix must be well applied to the presenting part
- High Bishop score
- No cephalopelvic disproportion.

Mechanism of action

1. Releases endogenous prostaglandin and may result in labour.
2. Intrauterine space decreases progressively following amniotomy, so that the uterine muscles contract more efficiently.

Complications

Are mainly in the form of infection, chorioamnionitis, cord prolapse, premature separation of placenta, injury to the fetus and cervix, constant drainage of liquor amni, fetal anaemia due to unrecognized vasa previa.

(b) Stripping of membranes or Sweeping of membranes

Sweeping or stripping of membranes is an old method of inducing labour described by Hamilton in 1810. Sweeping of membranes involve the digital separation of the membranes from the wall of the cervix and the lower uterine segment and has been widely used for many years in the belief that it reduces the need for formal induction of labour. The procedure of membrane sweeping causes an increase in the levels of prostaglandin F2alpha (McColgin et al 1993).

In a randomized study of 195 women beyond 40 weeks, two-thirds of women undergoing membrane sweeping laboured spontaneously within 72 hours compared to one-third of women in the control group

(Allot and Palmer 1993). A recent meta-analysis concluded that sweeping of membranes prior to term (38-40 weeks) does reduce the frequency of prolonged pregnancy and reduce the need for formal induction of labour from 36 per cent to 21 per cent (Boulvain et al 2001). The same review found no evidence of serious maternal or neonatal morbidity, such as infection associated with the procedure.

Technically, membrane sweeping is not possible in all women (Cammu and Haitsma 1998), usually requiring a cervical score greater than 4. Women undergoing membrane sweeping, more frequently describe discomfort during vaginal examination, vaginal bleeding and contractions not associated with the onset of labour than women not undergoing sweeping (Boulvain et al 1999). This discomfort will not be tolerated by all women hence counselling prior to membrane sweeping is needed.

Although it is presumed to be a formal method of induction, it is still employed by some obstetricians at term, especially when the indication for induction is not strong enough. The forewater is stripped by a gloved index finger passed through the cervical canal. Uterine contractions are frequently established following the procedure resulting from the release of endogenous prostaglandins, and labour is brought about within 3 days.

(c). Oxytocin

Commonly used method of induction.The goal of induction or augmentation is to effect uterine activity sufficient to produce cervical change and fetal descent, while avoiding development of a nonreassuring fetal status. Seitchik and coworkers(1984) found that the uterus contracts within 3 to 5 minutes of beginning an oxytocin infusion and that a plasma steady state is reached in 40 minutes. Response is highly variable and depends on preexisting uterine activity ,cervical status,pregnancy duration and individual biological differences. A small metaanalysis comparing high-dose and low-dose oxytocin regimens were done ,it reported that high –dose regimens were associated with reduced length of labor and ceaseran delivery rate and with a concomitant increased spontaneous vaginal delivery rate(Mori,2011)

CERVICAL RIPENING

Cervical ripening is a process by which the cervix becomes soft, compliant and partially dilated. It is due to a combination of biochemical, endocrine, mechanical and possibly inflammatory events.

Cervix is composed of collagen, smooth muscle and connective tissue 'ground substance' containing glycosaminoglycans. Cervix is

predominantly composed of Types I (66%) and Type III (33%). The firmness of the cervix in the non pregnant state is mainly due to the properties of the collagen fibrils which are bound together in the form of bundles. These bundles in turn are embeded in ground substance consisting of proteoglycans.[7]

In the cervix the main glycosaminoglycan are dermatan sulphate and chondroitin sulphate both of which are highly negatively charged and hydrophobic. Hence they repel water and are responsible for the firmness of the cervix. Towards term the glycosaminoglycan concentration of the cervix alters , the dermatan and chondroitin sulphates are replaced by hyaluronic acid. Hyaluronic acid is hydrophilic and imbibes water. Accumulation of water within the substance of cervix destabilizes the collagen fibrils contributing to cervical ripening. The water content of human cervix increases from 80% in non pregnant state to 86% in late pregnancy.

Collagenase is an enzyme that breaks down collagen types I, II and III and is produced by fibroblasts and leucocytes. Leucocyte elastase is another enzyme that can break down elastin, collagen and proteoglycans. It is produced by macrophages, neutrophils and eosinophils. The levels of both these enzymes are found to increase with advancing gestation and are associated with progressive decline in the

concentration of cervical collagen. Cervical remodelling takes place with advancing gestation.

The mature collagen, which has many crosslinks that are responsible for its tensile strength, is replaced by an immature collagen, which has few crosslinks.

Functionally the newly formed immature collagen is much weaker and is easily broken down during labour.Ganstrom et al (1991) have shown that the insufficient remodelling of collagen during pregnancy is an independent factor that results in labour.

Methods to assess cervical ripening

- Bishop score
- Lange et al score

BishopScore

BISHOP SCORE =..... (total)			Date of Bishop Score: ...../...../.....	
Score	0	1	2	3
Dilation	Closed	1 - 2	3 - 4	5
Length	>4	3 - 4	1 – 2	0
Consistency	Firm	Medium	Soft	–
Position	Posterior	Midline	Anterior	–
Head: station	-3	-2	-1, 0	+1,+2

A Score of 9 conveys a high likelihood for a successful induction.For research purposes a Bishop's score of 4 or less is considered unfavourable cervix and may be an indication for cervical ripening.

Risks of labour induction:

Maternal Risks:

- Failure leading to Cesarean section
- Uterine tachysystole
- Rupture uterus
- Intrauterine infection, Chorioamnionitis
- Amniotic Fluid Embolism
- Precipitate labor , Dysfunctional labor
- Increased risk of operative vaginal delivery
- Increased risk of post partum hemorrhage
- Abruptio Placentae
- APH from undiagnosed placenta praevia
- Water intoxication
- Gastrointestinal side-effects (vomiting,diarrhoea)

Fetal Risks:

- Fetal distress
- Fetal death
- Neonatal sepsis
- Iatrogenic delivery of a preterm infant
- Cord prolapse
- Neonatal jaundice
- Increased risk of birth trauma

Increase in ceasarean section rate:

The risk of ceasarean section increased nearly threefold in primigravid women (11.85% VS 27.9%) and doubled in multigravid women (3.4% vs 8.5%) who are induced compared to those labouring spontaneously(RCOG 2001)

Uterine tachysystole:

It is defined as more than five contractions in 10 minutes, averaged over 30 minutes.Misoprostol was associated with significantly increased risk of tachysystole when compared with PGE2 gel(WING AND Coworkers 1995a,1995b) Induced labour is associated with an increased risk of postpartum hemorrhage.

Prolonged induction is associated with a small increase in the risk of infectious morbidity with an estimated 10% incidence noted after 40hrs of induction(Bahn et al1998).

Oxytocin induction has been reported to increase the risk of neonatal hyperbilirubinemia.

Iatrogenic prematurity occurs inadverently and a review of the gestational age prior to induction is essential.

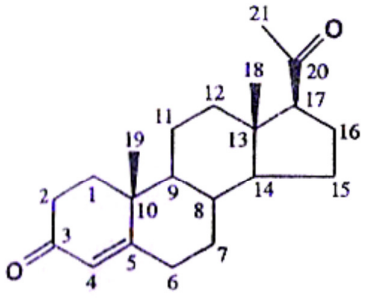
MIFEPRISTONE (RU 486)

Introduction:

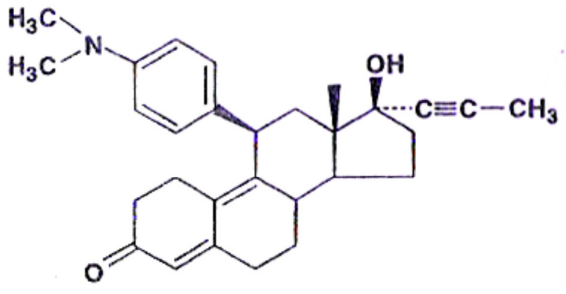
Mifepristone, an antiprogestin, was discovered by Dr.Etienne – Emile Beaulieu of France in 1980. There are two types of antiprogestin,

• Type I -RU486, ZK 112993

• Type II – ZK 98299.



Progesterone

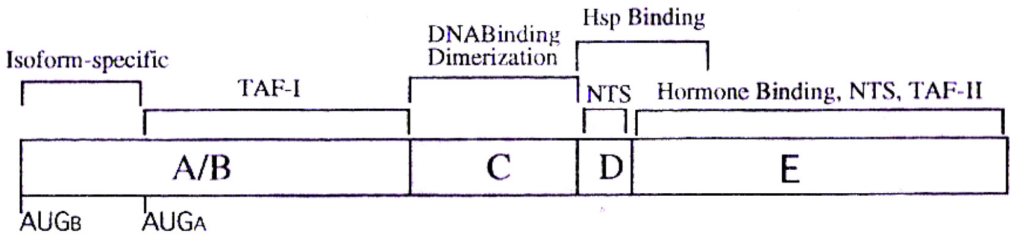


Mifepristone

Structure:

Mifepristone is a 19 nor steroid, chemically referred to as 11 beta-(4- dimethyl amino phenyl)-4, 9-dien-3-one. It is an antiprogestrone, which has a molecular formula of C19H35NO26. Its molecular weight is about 429.6.

The dimethyl amino phenyl side chain at position 11, a hydrophilic moiety, appears to be essential for the antiprogestronic activity. It also has antiglucocorticoid and antiandrogen activity.



The structure of the gene encoding both isoforms (PRA and PRB) of the progesterone receptor includes the location of n-terminal initiation codon for each isoform (AUGB and AUGA)8. The basic structure of this gene is shared by all the members of the steroid, thyroid, vitamin D, retinoic acid and orphan receptor superfamily, with five functional domains: an n-terminal transactivation domain (A/B), a DNA-binding domain (C), a hinge region (D) and a hormone-binding domain (E). Regions important for heat shock protein binding (HSP), nuclear

translocation (NTS) and transcriptional activation (TAF-I, -II) are also indicated.[2]

Mifepristone acts as a competitive receptor antagonist at the progesterone receptor in the presence of progesterone and acts as partial agonist in the absence of progesterone. Mifepristone at doses greater or equal to 1mg/kg antagonize the endometrial and myometrial effects of progesterone. Antiglucocorticoid effect of mifepristone is manifested at doses greater or equal to 5.5mg/kg and antiandrogenic effect in animals is seen with prolonged administration of very high doses of 10-100mg/kg[24]

III. Receptor binding

1) Transactivation domain
2) DNA binding domain
3) Hormone binding domain

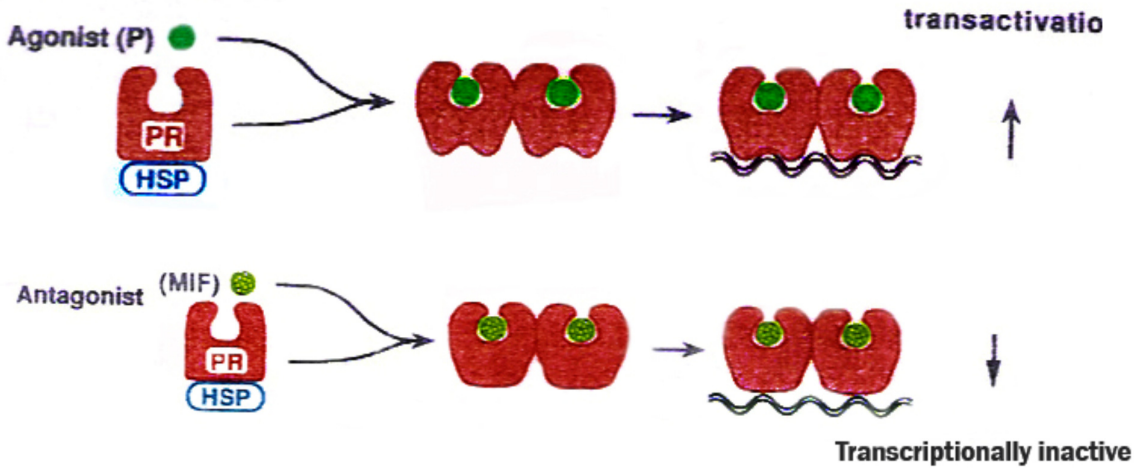
The anti progestin action of mifepristone is mediated by the PR, a ligand activated transcription factor with domains for DNA binding, hormone binding and transactivation. The amino acid glycine at position 722, which is in the hormone-binding domain of the human PR, appears to be critical for mifepristone binding and action. Substitution of glycine

with cysteine in the human PR generates a receptor that no longer binds mifepristone.

Mechanism of action

Progesterone and mifepristone produce a conformational change in the form of the PR that permits it to bind to DNA.

Agonist (Progesterone)



Antagonist (mifepristone)

PR – Progesterone receptor

HSP – Heat shock protein

In the absence of ligand the progesterone receptor is associated with heat shock proteins. Binding of progesterone or mifepristone induces conformational changes resulting in dissociation of HSP and dimerization of PR. The PR complex binds to specific progesterone

response elements in the promoter regions of progesterone responsive genes. Progesterone – PR complex is transcriptionally active resulting in agonistic effects whereas mifepristone – PR complex is not transcriptionally active resulting in antagonistic effects[39].

Under certain circumstances as in the absence of progesterone, mifepristone display progesterone agonistic activity It is related to the existence of two isoforms , PR-A and PR-B. PR-B behaves as a partial agonist in the presence of mifepristone. When PR-A and PR-B are present together the antagonistic effects of PR-A can override the agonistic effects of PR-B. So agonistic or antagonistic action depends on relative expression of PR-A and PR-B in target tissues.

Pharmaco Kinetics

Mifepristone is administered orally .It is readily absorbed. Metabolism is splanchnic circulation which reduces its bioavailability to 40%. Metabolic clearance rate is 0.55l/kg / day. It doesnot bind to cortisol binding globulin or sex steroid binding globulin[31].

Serum mifepristone levels reaches a maximum in one hour after oral administration of single dose ranging from 50 to 800mg. After single dose of 100mg or less the disappearance of mifepristone follows first order kinetics with a half life of 20-25 hours. After higher doses of

200-800mg there is an initial redistribution phase of 6-10 hours followed by a plateau in serum levels for 24 hours or more.

The major excretory pathway is fecal with less than 10% being recovered in urine. Metabolism involves two steps demethylation and hydroxylation. Mifepristone metabolite crosses the placental barrier during second trimester, but the efficacy of placental transfer decreases with advancing pregnancy.[23]

Byrne demonstrated that mifepristone exposure and induced labor were associated with increase in cortisol levels and significant elevation in cortisol levels was observed within 18hours of exposure to mifepristone.

Clinical pharmacology:

Pregnant uterus

Mifepristione stimulates the release of PGEF2α.[33,46,47] The increase in prostaglandin is due to marked reduction in the activity and tissue concentration of prostaglandin dehydrogenase, the key enzyme involved in the control of prostaglandin catabolism by mifepristone21. Mifepristone increases the sensitivity of the myometrium to prostaglandin by increasing the number of gap junctions so that synchronization of uterine muscle contractility occurs. This causes

enhanced electrical activity resulting in opening of voltage dependent calcium channels, which causes calcium influx and thereby muscle contraction.[53]

Mifepristone causes cervical ripening in women undergoing termination of pregnancy. Cervical ripening occurs directly or through the blockage of progesterone receptors49. Mifepristone stimulates the release of nitric oxide and the expression of inducible nitric oxide synthase in the cervical cells . This is one of the mechanism by which mifepristone initiates cervical ripening[52].

Other Uses

1. Termination of early pregnancy

Medical abortion became an option for early abortions in India when in April 2002, the Drugs Controller General approved the use of mifepristone to terminate early pregnancies.

In December 2006, the Drugs Controller General of India granted the permission to manufacture misoprostol and approved its use for gynecological conditions like cervical ripening, prevention of post partum hemorrhage and first trimester abortion with mifepristone50.In India, combination of mifepristone and misoprostol is recommended for termination of early pregnancy up to 49 days/seven weeks from the last

menstrual period (LMP); WHO recommends their use up to 63 days or nine weeks from LMP (WHO, 2003).[13]

2. Contraceptive

Mifepristone, a novel estrogen free contraceptive administered in low doses daily (2 to 10mg), inhibits ovulation, menstruation and significantly suppresses the effect on endometrium.30 Mifepristone as a emergency contraceptive inhibits ovulation, blocks implantation by causing delay in maturation of endometrium and causes regression of the corpus luteum in majority of women when given in middle or late luteal phase.32,43,48 Two randomized trials have compared 600 mg of mifepristone with the Yuzpe regimen. In these trials, single dose of 600mg of mifepristone given within 72 hours of unprotected intercourse was 100 percent effective as an emergency contraceptive.[34]

3. Uterine myoma

On long term basis, mifepristone blocks progesterone dependent growth factors and reduces blood supply to myoma. Hence, Mifepristone can be used in uterine fibroids as an alternative to GnRh anlogues in the preoperative application. If the safety of long term low dose mifepristone is established, perimenopausal women with large, symptomatic fibroid can avoid hysterectomies by using mifepristone till menopause.[41]

4. Endometriosis

Mifepristone through antioxidant property does not allow endometriosis to proliferate. It also preserves follicular phase levels of estradiol. 5mg dose does not stabilize the endometrium and hence needs a dose of 50mg daily. However, use of mifepristone for the treatment of endometriosis requires additional studies.[42]

5. Ovarian Cancer

Mifepristone inhibits ovarian cancer cells growth by inducing G1 cell cycle arrest and blocking G1-S phase transition without causing cell death. This growth arrest is observed by a decline in cyclin – dependent kinase 2 (cdk2) protein level and activity.[44] In 2003, Xu M et al reported that ovarian cancer cells expressed glucocorticoid receptors. Mifepristone may drive its anticancer action by binding to glucocorticoid receptors with an affinity similar to that for progesterone receptors and as an antioxidant to drive G1 arrest through a p53 independent p21. Thus, mifepristone is a single agent potent blocker of ovarian cancer growth, however, the feasibility of using mifepristone to enhance the efficacy of conventional chemotherapy for ovarian cancer requires further investigations.

6. Premenstrual Syndrome

The sex steroid dependency of this disorder has been well established by the absence of PMS in castrated women and women treated with GnRH agonist analogues. Because the main symptom complex occurs in the luteal phase when serum progesterone is at highest level, it was proposed that an antiprogestin, such as mifepristone may be useful in treatment of PMS.[4]

7. Ectopic Pregnancy

The role of antiprogestin in the medical therapy of ectopic pregnancy remains to be clearly defined. Certainly, the timing, dosing, and efficacy of mifepristone treatment in this scenario awaits future studies.

8. Abnormal Uterine Bleeding

It has been suggested by some that antiprogestins may be useful in treatment of dysfunctional uterine bleeding. No clinical experience in this venue has been published. If adenomyosis is the etiology of menorrhagia, it may be expected that treatment with an antiprogestin may be useful.

9. Breast Cancer

It has been observed that estrogen and progesterone in low doses stimulates cancer growth but in high doses both inhibit breast cancer growth. Antiestrogen (Tamoxifen) and antiprogestin produce tumor regression but either agent alone only produces tumor stasis. Tamoxifen down regulates the estrogen receptor but it favors agonists activities and therefore up regulates the progesterone receptor. Mifepristone down regulates both estrogen and progesterone receptors. The finding suggests that tamoxifen can not inhibit the progestin-mediated growth-stimulatory effects. Thus, addition of mifepristone to tamoxifen effectively re-establishes tamoxifen growth inhibition. It has been observed that eventually all advanced breast cancer become hormone independent and increasingly resistant to any subsequent therapy as a result there is limitation in potential utility of antiprogestin and other endocrine therapies for the treatment of advanced disease.

10. Cushing's Syndrome

In 2001, Dwight FM et al reported that extremely ill patient with Cushing's syndrome, treated initially unsuccessfully by a combination of conventional surgical, medical and radiotherapeutic approaches responded extremely well up to 25mg/kg/day, long term mifepristone,

glucocorticoid receptor antagonist therapy. Treatment efficacy was confirmed by the normalization of all biochemical glucocorticoid-sensitive measurements, significant reversal of the patient's heart failure, resolution of the psychotic depression and usual return of his HPA axis to normal.[25]

11. Meningioma

Most meningiomas have no estrogen receptors but have substantial concentrations of progesterone receptors. In patients with unresectable meningiomas, objective response and subjective improvement has been noted.[29]

Contraindication to Mifepristone:

Presence of an intrauterine device (IUD),

Ectopic pregnancy,

Adrenal failure,

Hemorrhagic disorders,

Inherited porphyria,

On anticoagulant therapy,

Long term corticosteroid therapy.

Side Effects

***Short term use*:**

Abdominal pain,

Cramping,

Nausea,

Vomiting,

Headache.

Long term use:

Adrenal insufficiency,

Low serum potassium levels,

A slight increase in serum creatinine levels,

Moderate increase in hepatic enzymes.

The combination of mifepristone plus a prostaglandin has been approved for ending pregnancies up to 49days. The use of mifepristone plus an oral prostaglandin, presumably with fewer side effects,has improved the acceptability of this method for early first-trimester abortion over standard surgical procedure.Mifepristone has also been approved in France for the induction of labour in the event of fetal death Adequate clinical studies have demonstrated the safety and effectiveness

of this drug and these studies support applications to regulatory authorities in other countries[9].

"Cochrane review has justified further trials comparing mifepristone with the routine cervical ripening agents currently used"[53].

PROSTAGLANDINS

Structure

Prostaglandins are biological derivatives of 20 carbon polyunsaturated fatty acids that are released from cell membrane phospholipids. The prostaglandins PGE2 and PGF2 alpha are widely used in obstetric practice.

There are no preformed stores of prostaglandin. They are synthesized locally, in response to appropriate stimulus, at the rates governed by release of arachidonic acid from cell membrane by the action of lysosomal enzyme phospholipase A2, which is said to be the rate limiting step in prostaglandin biosynthesis.

Free arachidonic acid enters the cyclo-oxygenase pathway and converted to prostaglandin, by the enzyme prostaglandin synthase. In pregnant uterus of human being, free arachidonic acid is converted to prostaglandins in chorion leave and decidua vera, by prostaglandin synthetase which is greatest in the amnion.

In the amnion and chorion, PGE2 is formed. In decidua vera, both PGE2 and F2 alpha are formed. The fetal membranes and decidua vera are proved to be the site of synthesis of both arachidonic acid and prostaglandins in amniotic fluid. The half-life of primary prostaglandins is about five minutes while that of the major metabolite is 8 minutes. The lung is the major site of metabolism of prostaglandins, other sites being liver and kidney.

Pharmacological actions

Prostaglandins act on almost every other tissue in the body. Some of the best known actions are

a. Stimulation of smooth muscle leading to either relaxation depending upon the receptors involved
b. Changes in the cervical tissue
c. Inhibition of gastric acid secretion and cytoprotection
d. Inhibition and induction of platelet aggregation
e. Increased vascular permeability
f. Thermoregulation
g. Modification of steroidogenesis in the adrenals and gonads
h. Inhibition of hormone induced lipolysis
i. Release of neurotransmitters in the peripheral nervous system and the potentiation of action of biogenic amines.

However, the most potent action of prostaglandins is their ability to stimulate smooth muscles of the uterus, gut and vasculature. Unlike oxytocin, which is relatively ineffective in early pregnancy prostaglandins, are potent stimulators of uterine myometrium in all stages of pregnancy.

Uses of prostagandins in obstetrics:

a) Induction of abortion,

b) Termination of molar pregnancy,

c) Induction of labour,

d) Acceleration of labour,

e) Management of atonic postpartum haemorrhage.

Muscle physiology consists of three important concepts:

- Phasic contraction,
- Tonic tension,
- Relaxation.

Phasic contraction is intermittent and may last for a short or a long period of time, whereas tonic tension is fairly constant lasting for prolonged periods. At the myometrial cellular level, prostaglandins have been found to induce both phasic contractions as well as tonic tension with superimposed phasic contractions (Chamley and Parkington 1984). In practical terms, they increase both the resting tone of the uterine

myometrium as well as the amplitude and duration of myometrial contractions.

On a molecular level, phasic contractions are due to the influx of sodium ions into the myometrial cell, whereas tonic tension is due the increased availability of intracellular calcium. Both these processes are affected by prostaglandins (Reiner and Marshall 1976).Prostaglandins also induce the formation of gap junctions between the myometrial cells, which help in the development of coordinated myometrial action, giving the advantages of a functional syncytium.

There is also a differential response according to the type of prostaglandins. PGE2 metabolites peak prior to the onset of established labour, whereas PGF2alpha metabolites peak during labour and correlate directly with the duration of labour. PGE2 has a predominant effect on the cervix, whereas PGF2alpha on the myometrium.The United Kingdom's National Institute for Clinical Excellence(NICE) guidelines on the induction of labour recommends that prostaglandin E2 should be used in preference to oxytocin in women with intact membrane regardless of their parity or the ripeness of the cervix.

Contraindications

a. Hypersensitivity to the compounds

b. Bronchial asthma

Advantages

a. It has got powerful oxytocic effects, irrespective of the period of pregnancy.

b. As such it can be used independently especially in induction of abortion with success.

c. It is useful drug not only in induction but also for acceleration of labour.

d. It has no antidiuretic effect.

Disadvantages

a. It is costly

b. Unpleasant side-effects caused by its stimulatory effects on the smooth muscles, which however subside easily due to its rapid metabolism

c. When used as an abortifacient, extensive cervical laceration may occur

d. The hyperactivity of the uterus if occurs during induction may continue for a variable period.

Side effects

a. Nausea, vomiting and diarrhea are common.

b. Cramping pain of uterine origin related to the degree of uterine activity.

c. Unduly forceful uterine contractions.

d. Anaphylaxis.

Oxytocin

The word 'oxytocin' means "Quick birth". The structure of oxytocin was determined by Du vigneaud in 1950.Oxytocin, an octapeptide which is secreted in a pulsatile manner is a neurohormone originating in the hypothalamus and secreted by the posterior lobe of pituitary gland. The half life is 3-5 minutes. The metabolic clearance rate is similar for men, pregnant women and non pregnant women, 20-27 m1/kg/minute. Recent study shows that 40 minutes are required for any particular dose of oxytocin to reach a steady state plasma concentration.3

The sensitivity of uterus to oxytocin increases as pregnancy progresses due to increase in oxytocin receptors in the myometrium and decidua.

Oxytocin has direct stimulatory effects on the myometrium and also stimulates decidual prostaglandin production. The direct effect of oxytocin on myometrium is mediated by polyphosphoinositide hydrolysis with production of inositol phosphates that act as a second messenger and lead to the mobilization of interacellular calcium ion. The principles of current clinical usage of intravenous oxytocin, are based on the classic studies of Turnbull and Anderson (1968).

Oxytocin is known to be a very potent uterotonic, causing uterine contractions in a sensitized uterus. The infusion of oxytocin is relatively ineffective in inducing labour in human pregnancies, except for dose near term. Oxytocin is effective, only in those patients in whom preparation of the uterus for active labour is already completed. The plasma concentration of oxytocin in pregnant women is 2-10mcg/ml.

Advantages

a) Cost effective,
b) Relatively safe,
c) The dose can be adjusted and titrated according to the needs in a particular case,
d) When combined with amniotomy induction delivery interval is very short, labour gets established earlier.

Disadvantages

a) Patient has to be confined to bed,
b) Water intoxication,
c) Rupture of uterus in multigravida,
d) Coronary insufficiency,
e) Incidence of PPH in induced labour is greater.
f) Hyperstimulation, late deceleration of FHR,
g) Fetal hyperbilirubinemia.

SOURCE OF STUDY

The study was carried out in the Department of Obstetrics and Gynaecology, Thanjavur Medical college, Thanjavur,which included 200 uncomplicated prolonged pregnancies with 100 cases in Mifepristone and 100 cases in PGE2gel group during the period of August 2016 to July 2017 .

Study- Case- control study

Inclusion criteria

Uncomplicated prolonged pregnancies(41 completed weeks) with

Adequate liquor

Reactive CTG

No CPD

Pre induction Bishops score<4

Exclusion criteria

Cephalopelvic disproportion

Premature rupture of membranes(PROM)

Oligohydromnios

GHT, GDM and other medical complications

IUGR

IUFD

Method of study

On admission, a detailed history, complete general and obstetric examination is carried out.

Gestational age is assigned as per dating scan in first trimester Vaginal examination is done under strict aseptic precautions and bishops score assessed and CPD ruled out.

Obstetric scan for fetal maturity and Modified Biophysical Profile is done.

Once the inclusion criteria fulfilled , the patient is transferred to the labour ward.

Informed Consent

A detailed written informed consent is obtained from the participant and her relatives.

The following are addressed in the consent form.

Indication for induction of labour,

Drug to be administered with its dosage,

Mode of administration,

Side effect of the drug,

Risks associated with the administration of these drugs and if complications arise alternative mode of termination are all discussed.

Treatment Schedule

Group – I

- 100 pregnant women are given tablet mifepristone 200mg orally on day1.
- They are observed for maternal vitals, uterine activity, bleeding or draining pv and fetal heart rate.
- After the wait period of 24 hours or when the Bishop score is ≥ 6, when the cervical dilatation is > 2cm, or when the membranes ruptured , whichever is earlier, labour is accelerated with oxytocin drip.

Group – II

- 100 pregnant women are instilled endocervical PGE2 gel 0.5mg on day 1.
- They are observed for maternal vitals,uterine activity,bleeding or draining pv and fetal heart rate.
- After the wait period of 6 hours,if required repeat gel at interval of 6hrs max.of 3 doses is kept in 24hours.
- When the Bishop score is ≥ 6, when the cervical dilatation is>2cm, or when the membranes ruptured, whichever is earlier labour is accelerated with oxytocin drip

Monitoring of the patients

Maternal vitals, uterine activity and fetal heart rate .

Partogram is maintained.

If membranes not ruptured ARM is done at 4cm cervical dilatation.

Delivery particulars, duration of each stage of labour, blood loss at third stage of labour are recorded.

In vaginal delivery,Standardized Visual Estimation of Blood Loss method used to assess blood loss. Blood loss estimation is done from the onset of third stage of labour to the end of stoppage of active bleed or upto 1hour post delivery whichever is earlier. Blood was allowed to drain into a fixed container & fixed sized mops were used.

In cesarean delivery,difference between the weight of the dry surgical swabs before use and wet or soaked swabs with blood after use was taken. The weight difference would be nearly the lost blood taking into consideration that 1gram blood will be equal to 1ml blood. Collected blood in the suction bottle would be added to the blood in swabs.

Baby particulars are recorded.

Mother and baby are observed for postnatal complications if any

The efficacy is assessed by the following criteria:

Favourability of Bishop score at 24 hrs.

Duration of I, II and III stage of labour and blood loss.

Drug administration to delivery interval.

The mode of delivery.

Cesarean section rate.

The 5minute Apgar score, neonatal complications and incidence of neonatal mortality.

Maternal complications.

Success of induction is assessed by the following criteria:

Patients who delivered vaginally (including operative vaginal deliveries)

Failure of induction is assessed by the following criteria:

Who has not entered active phase of labour.

Patients who underwent caesarean section

Table 1: Baseline demographic characteristics of the subjects

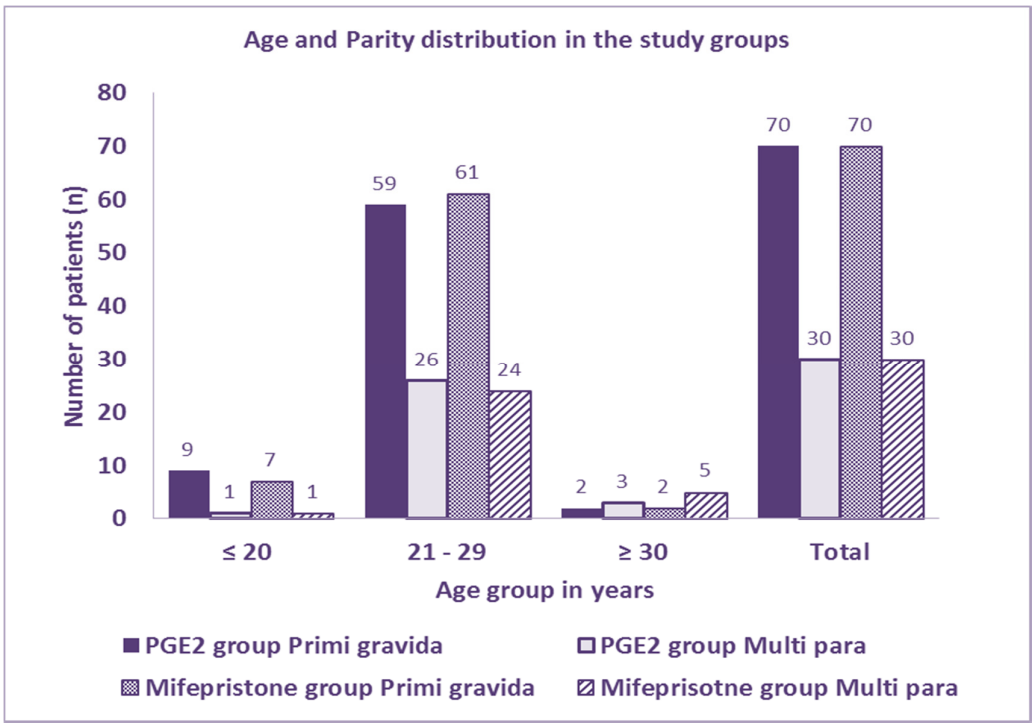
S. No	Parameter	PGE_2 group	Mifepristone Group	P value	Statistical test
1	Number (n)	100	100		
2	Age in years (Mean ± SD)	24.11± 3.16	24.1± 3.05	0.9 (NS)	Student 't' test
3	Socioeconomic status n(%)				
	Class III	11 (11%)	6 (6%)	0.31 (NS)	Chi-Square Test
	Class IV	69 (69%)	82 (82%)	0.047	
	Class V	20 (%)	12 (12%)	0.176 (NS)	
4	Primigravida/ Multigravida	70/30	70/30	0.999(NS)	Chi Square test

Table 2: Age and parity distribution between the PGE2 and mifepristone groups.

S. No	Age in years	PGE_2 group		Mifepristone Group	
		Primigravida	**Multigravida**	**Primigravida**	**Multigravida**
1	≤ 20	9 (9%)	1 (1%)	7 (7%)	1 (1%)
2	21 to 29	59 (59%)	26 (26%)	61 (61%)	24 (24%)
3	≥ 30	2 (2%)	3 (3%)	2 (2%)	5 (5%)
4	Total	70	30	70	30

Age and parity distribution of women included in this study were comparable in both mifepristone and PGE2 gel group.

Figure 1: Age and parity distribution between the PGE2 and mifepristone groups

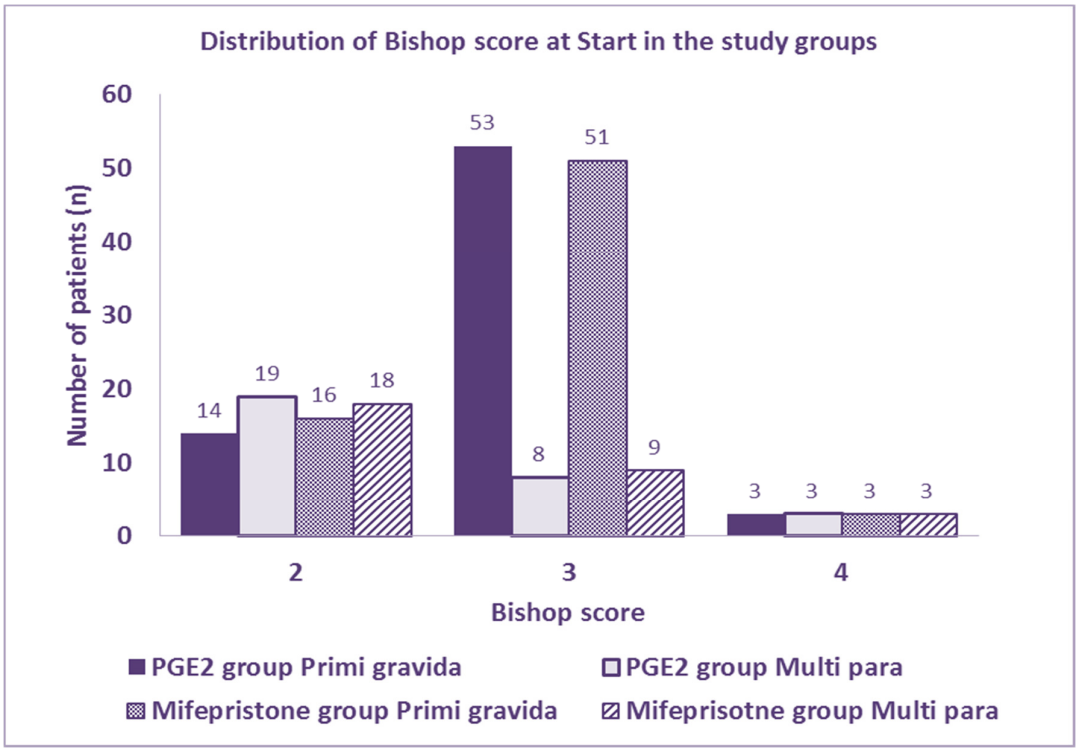


The data are expressed as vertical bar diagram. The height of the bar represents the number of study subjects (n) in each group.

Table 3: Bishop score at the start of study between PGE2 and Mifepristone groups.

S. No	Bishop Score	PGE_2 group		Mifepristone Group	
		Primigravida	Multigravida	Primigravida	Multigravida
1	2	14 (14%)	19 (19%)	16 (16%)	18 (18%)
2	3	53 (53%)	8 (8%)	51(51%)	9(9%)
3	4	3(3%)	3(3%)	3 (3%)	3 (3%)

Figure 2: Bishop score at start between the PGE2 and mifepristone groups



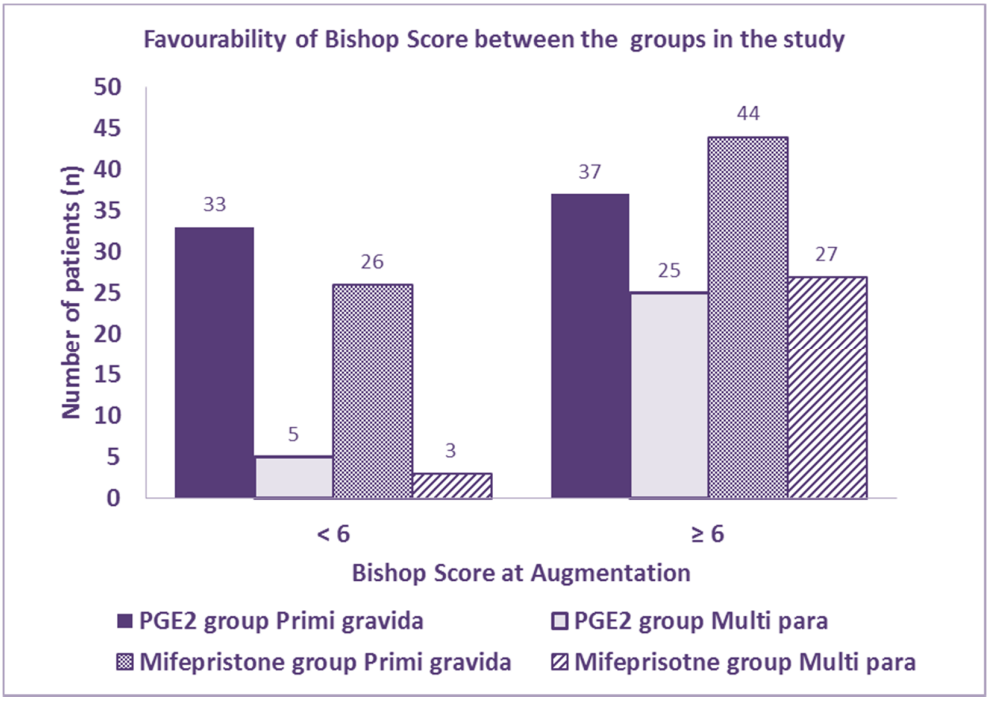
The data are expressed as vertical bar diagram. The height of the bar represents the number of study subjects (n) in each group.

All the mothers in both the groups had initial bishop's score of 2 to 4 before preinduction cervical ripening

Table 4: Favorability of Bishop score between PGE2 and Mifepristone groups.

S. No	Bishop Score	PGE_2 group		Mifepristone Group	
		Primigravida	**Multigravida**	**Primigravida**	**Multigravida**
1	<6	33 (33%)	5 (5%)	26 (26%)	3 (3%)
2	≥ 6	37 (37%)	25 (25%)	44 (44%)	27 (27%)

Figure 3: Bishop score at Augmentation (favorability) between the PGE2 and mifepristone groups



The data are expressed as vertical bar diagram. The height of the bar represents the number of study subjects (n) in each group.

Table 5: Comparison of Bishop Score between the PGE2 and Mifepristone group at various time points

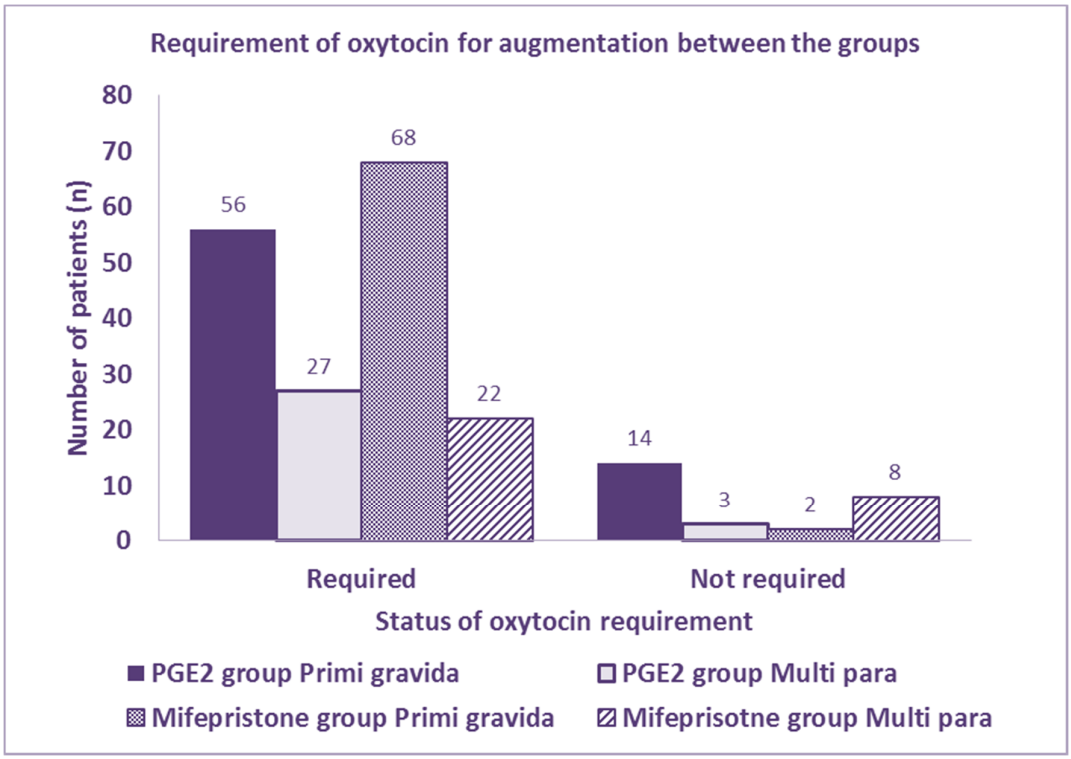
S. No	Bishop Score	PGE_2 group	Mife pristone Group	P value	Statistical test
1	Bishop score at start	2.73 ± 0.56	2.72 ± 0.75	0.9 (NS)	Unpaired t test
2	Bishop score at augmentation	5.37 ± 1.21	5.68 ± 1.23	0.082 (NS)	Mann-Whitney U test
3	Bishop score difference from start to augmentation time	3 ± 1.03	3.1 ± 1.05	0.4 (NS)	Mann-Whitney U test

Data are expressed as mean ± SD. p-value for Bishop's score at start 0.9 which is not significant. p-value for Bishop's score at augmentation 0.082 which is not significant. p-value for Bishop's score difference 0.4 which is not significant.

Table 6: Augmentation with oxytocin between PGE2 and Mifepristone groups.

S. No	Augmentation with oxytocin	PGE_2 group		Mifepristone Group	
		Primigravida	Multipara	Primigravida	Multipara
1	Required	56 (56%)	27 (27%)	68 (68%)	22 (22%)
2	Not required	14 (14%)	3 (3%)	2 (2%)	8 (8%)

Figure 4: Requirement of oxytocin for Augmentation between the PGE2 and mifepristone groups



The data are expressed as vertical bar diagram. The height of the bar represents the number of study subjects (n) in each group.

In the mifepristone group among 68 primigravida who required oxytocin , 53 delivered by labour natural. In PGE2 gel 56 primigravida required oxytocin, 44 delivered by labour natural.

In mifepristone group, 8 multipara not required oxytocin,6 delivered by labour natural.

In PGE2 gel,14 primigravida not required oxytocin,only 3 delivered by labour natural.3 multipara not required oxytocin,3 delivered by labour natural.

Shortest drug administration to delivery interval in mifepristone group was 10 hours and in PGE2 gel group was 8 hours.

In mifepristone and PGE2 group, primigravida and multipara both require oxytocin for vaginal delivery

In PGE2 gel group, 3 primigravida,had 3 repeat PGE2 gel in 24hours among which 2 had vaginal delivery, 17primigravida and 7 multipara had 2 repeat PGE2 gel ,among which 6 had cesarean section, 2 fetal distress and 4 in view of failed induction.

Table 7: Comparison of duration of labour between the PGE2 and Mifepristone group at various stages

S. No	Duration of Labour	PGE_2 group (n=100)	Mifepristone Group (n=100)	P value	Statistical test
1	Stage 1 (in hours)	10.26 ± 2.9	13.8 ± 3.9	<0.0001*	Unpaired 't' test
2	Stage 2 (in minutes)	24.33 ± 8.03	23.3± 8.5	0.312 (NS)	Mann Whitney test
3	Stage 3 (in minutes)	5.48 ± 2.67	4.08 ± 1.27	0.006*	Mann Whitney test
4	DD interval (in hours)	16.48 ± 5.8	21.33 ± 4.5	<0.0001*	Mann Whitney test

Data are expressed as mean ± SD. * indicates $p<0.05$ and hence considered as statistically significant.

Duration of II and III stage of labour were shorter in mifepristone group with the III stage being statistically significant. Duration of I stage is shorter in PGE2 gel group with statistical significance. Drug administration to delivery interval shorter with PGE2 gel group with statistical significance.

Table 8: Comparison of duration of labour between the PGE2 and Mifepristone group at various stages with respect to parity distribution

S. No	Duration of Labour	PGE_2 group (n=100)			Mifepristone Group (n=100)		
		Primi gravida	Multi gravida	P value	Primi gravida	Multi Gravida	P value
1	Stage 1 (in hours)	10.84 ± 2.99	9.23 ± 2.51	0.023*	14.97 ± 3.56	11.63 ± 3.94	0.0002***
2	Stage 2 (in minutes)	26.38 ± 8.23	20.62 ± 6.15	0.002**	25.76 ± 8.85	18.48 ± 5.31	0.0002***
3	Stage 3 (in minutes)	5.5 ± 2.7	5.43 ± 2.65	0.92 (NS)	4.07 ± 1.32	4.09 ± 1.2	0.95 (NS)
4	DD interval (in hours)	17.24 ± 6.28	15.12 ± 4.75	0.13 (NS)	22.6 ± 4.14	18.74 ± 4.3	0.0002***

Data are expressed as mean ± SD. * indicates $p<0.05$ and hence considered as statistically significant.Unpaired 't' test was used to find the statistical significance.

Table 9: Comparison of duration of labour between the PGE2 and Mifepristone group at various stages in primigravida.

S. No	Duration of Labour	PGE_2 group	Mifepristone Group	P value
1	Stage 1 (in hours)	10.84 ± 2.99 (n=47)	14.97 ± 3.56 (n=55)	<0.0001*
2	Stage 2 (in minutes)	26.38 ± 8.23 (n=47)	25.76 ± 8.85 (n=55)	0.717 (NS)
3	Stage 3 (in minutes)	5.5 ± 2.7 (n=60)	4.07 ± 1.32 (n=54)	0.0004*
4	DD interval (in hours)	17.24 ± 6.28 (n=47)	22.6 ± 4.14 (n=55)	<0.0001*

Data are expressed as mean ± SD. * indicates $p<0.05$ and hence considered as statistically significant.Unpaired 't' test was used to find the statistical significance.

Table 10: Comparison of duration of labour between the PGE2 and Mifepristone group at various stages in Multigravida.

S. No	Duration of Labour	PGE_2 group	Mifepristone Group	P value
1	Stage 1 (in hours)	9.23 ± 2.51 (n=26)	11.63 ± 3.94 (n=27)	0.011*
2	Stage 2 (in minutes)	20.62 ± 6.15 (n=26)	18.48 ± 5.31 (n=27)	0.18 (NS)
3	Stage 3 (in minutes)	5.43 ± 2.65 (n=24)	4.09 ± 1.2 (n=26)	0.02*
4	DD interval (in hours)	15.12 ± (n=26)	18.74 ± 4.3 (n=27)	0.005*

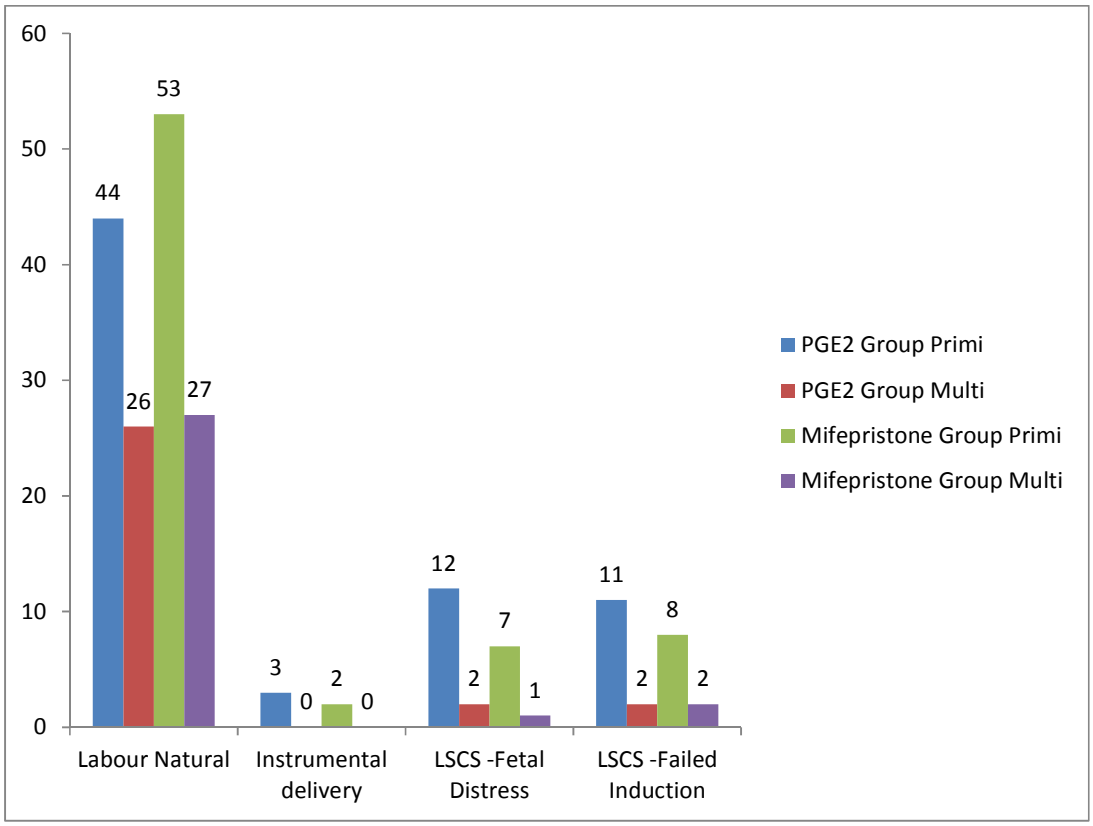
Data are expressed as mean ± SD. * indicates $p<0.05$ and hence considered as statistically significant. Unpaired 't' test was used to find the statistical significance.

Table 11: Mode of delivery between PGE2 and Mifepristone groups.

S. No	Mode of delivery	PGE_2 group (n=100)		Mifepristone Group (n=100)	
		Primigravida	Multigravida	Primigravida	Multigravida
1	Labour naturale	44 (44%)	26 (26%)	53 (53%)	27 (27%)
2	Instrumental delivery	3(3%)		2(2%)	
3	LSCS (Fetal Distress)	12 (12%)	2 (2%)	7 (7%)	1 (1%)
4	LSCS (Failed Induction)	11(11%)	2 (2%)	8 (8%)	2 (2%)

Data are expressed as n(%).

FIGURE 5: Mode of delivery



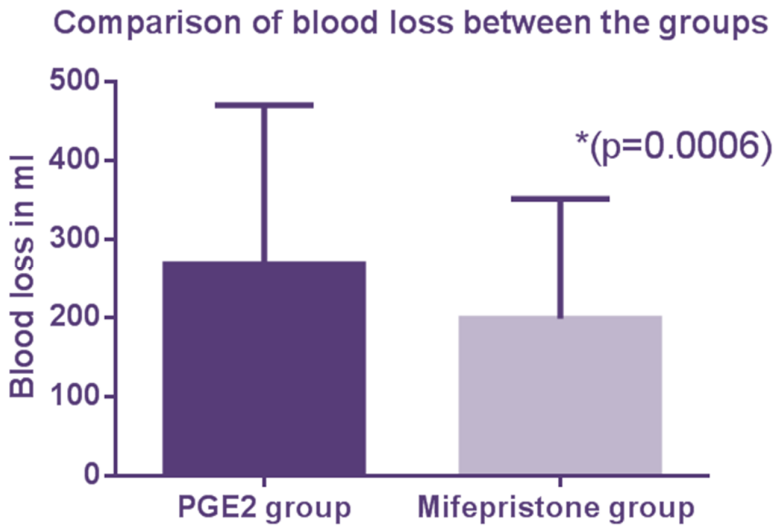
In mifepristone group,55(55%) primigravida 27(27%) multigravida delivered vaginally. 15(15%) primigravida were delivered by cesarean section of which 8(8%) was done for failed induction and 7(7%) for fetal distress, in multigravida 2(2%) was done for failed induction and 1(1%) for fetal distress.

In PGE2 gel group, 47(47%) primigravida 26(26%) multigravida were delivered vaginally. 23(23%) primigravida were delivered by cesarean section of which 11(11%) was done for failed induction and 12(12%) for fetal distress, in multigravida 2(2%) was done for failed induction and 2(2%) for fetal distress

Table 12. Comparison of blood loss between the PGE2 and mifepristone groups

S. No	Group name	Mean	Standard deviation	SEM	P value
1	PGE2 group (in ml)	268.5	201.7	20.17	0.0006 (Mann Whitney Test)
2	Mifepristone group (in ml)	198.5	153.1	15.31	

Figure 6: Comparison of blood loss between the PGE2 and mifepristone groups



Data are expressed as mean with SD. Height of the bar represents the mean and error represents the standard deviation. * indicates $p<0.05$ when compared to the PGE2 group.

P value:0.0006(significant)

Mean blood loss in mifepristone group was less when compared to PGE2 gel group. In PGE2 gel group 1(1%)primigravida had atonic PPH- blood loss of 1100ml which was controlled with uterotonics

Maternal complications like nausea, vomitting and abdominal cramps were similar in both groups except fever was found among 1 primigravida and 1 primigravida had atonic PPH in PGE2 gel group

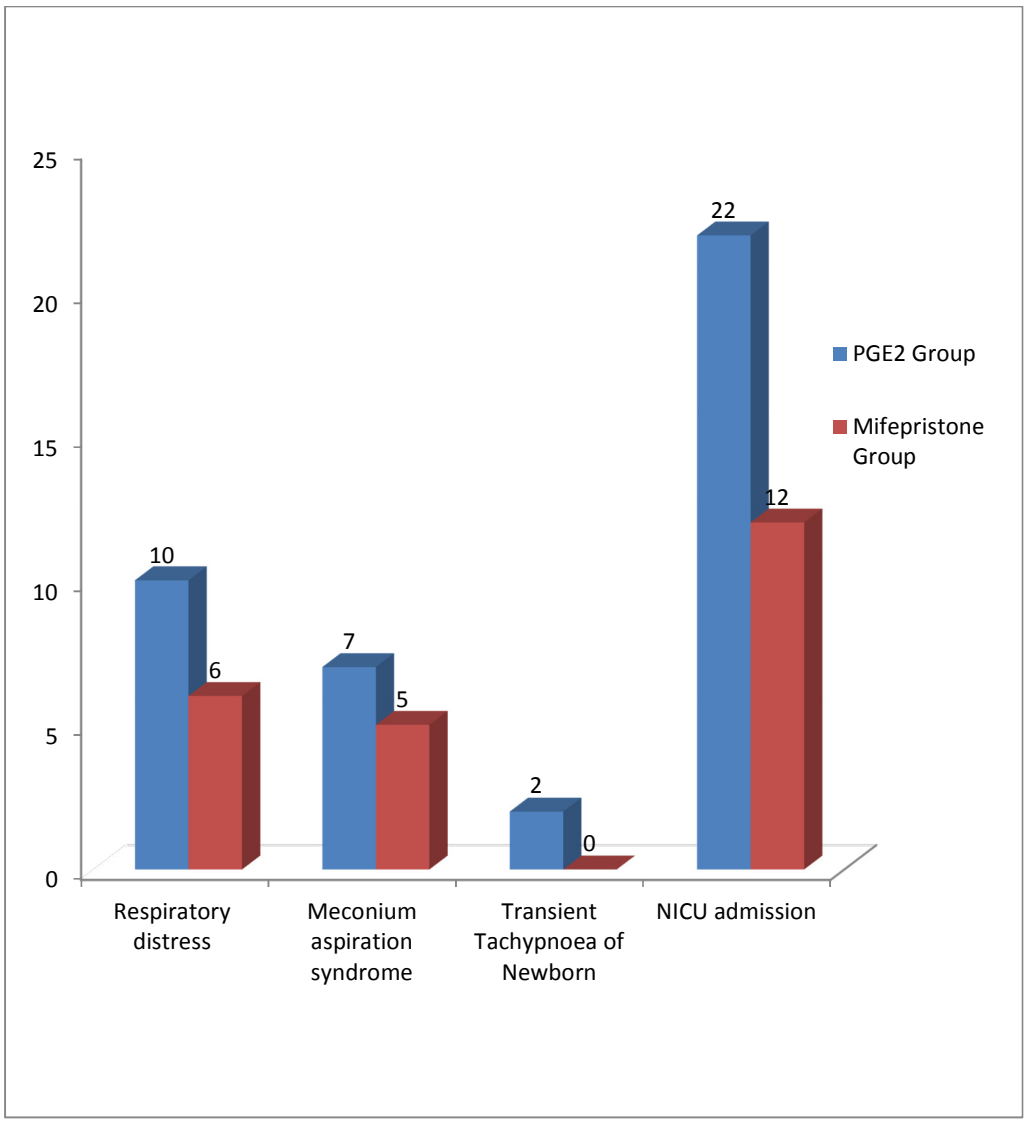
Table 13.Comparison of neonatal complications between PGE2 and mifepristone group.

S. No	Complication/ Indicators	PGE_2 group (n=100)		Mifepristone Group (n=100)	
		Primigravida	Multi gravida	Primi gravida	Multi gravida
1	Respiratory distress	8 (8%)	2 (2%)	5(5%)	1 (1%)
2	Meconium aspiration syndrome	6(6%)	1(1%)	4(4%)	1(1%)
3	Transient tachypnea of newborn	2 (2%)	0	0	0
4	Average Apgar at 1 minute	4.41 ± 0.82	4.8 ± 0.61	4.8 ± 0.69	4.76 ± 0.7
5	Average apgar at 5 minutes	6.67 ± 0.51	6.9 ± 0.30	6.97 ± 0.53	7.1 ± 0.54
6	NICU admission	19/70 (27.14%)	3/30 (10%)	9/70 (12.85%)	3/30 (10%)

NICU admission was 37.14% in PGE2 gel as compared to 22.85% in mifepristone group.In PGE2 gel 2 neonate was found to be low birth weight.But there was no neonatal mortality in both groups.

APGAR score at 1minute and 5 minute were similar in both groups.

Figure 7: Comparison of neonatal complications



200 cases admitted for safe confinement in Thanjavur Medical College, Thanjavur has been recruited for the study. Among this 100cases were induced with PGE2gel intracervically and 100 cases were given Mifepristone 200mg orally.

S.NO	STUDY	YEAR	DOSAGE SCHEDULE	CONTROL	WAIT PERIOD
1	Wing DA at al	2002(180)	200 mg of oral mifepristone dose followed by intravaginal 25 micrograms every 4th hourly or IV oxytocin	Placebo	24Hrs
2	Giacolone et al	1992	400mg of mifepristone as a single oral dose	Placebo	48Hrs
3	Frydam R et al	1992(120)	200mg mifepristone on day 1and 2 followed by augmentation with prostaglandin on day 4	Placebo	4 days
4	Lil et al	1996	150 or 200mg mifepristone in the 1st 2 or 3days & on th day misoprostol was added successively in 100-300 microgram dosage	-	3 days

5	Suh et al	1996(124)	50mg mifepristone 12th hourly for 2days followed by prostaglandin or oxytocin	-	48Hrs
6	Elliot et al	1998(83)	50-200mg mifepristone as a single oral dose	Placebo	24Hrs interval for 72 hours
7	This study	2016	200mg mifepristone as a single oral dose	PGE2 gel	24Hrs

In the present study, we opted for 200mg mifepristone as the tablet is available and the women will get the exact dose without fail. Hapangama and Neilson(2000) reported that there is insufficient evidence to support a particular dose, but a single dose of 200mg mifepristone appears to be the lowest effective dose for cervical ripening.

1.AGE:

In this study,61(61%) primi and 24(24%) multigravida were between the age group of 21-29years in mifepristone group; 59(59%)primi and 26(26%)multigravida were between the age group of 21-29years in PGE2 gel group accounting for 85% in the age group of 21-29years in both groups .

This study correlates with randomized controlled trial conducted by Wing et al of Southern California, Los Angels, Oct 2000 in which 88% of the patients were in the age group of 21-30years.

In this study the mean age in mifepristone group is 24.1years which is also comparable with Kanan Yelikar study,J obstet gynaecol India.2015, where the mean age in study group is 22.98years.

2.GRAVIDITY:

Both primi(70%) and multigravida(30%) were included in the study. In this aspect our study correlates with studies done by Giacalone et al, Department of Obstetrics and Gynaecology, Hospital Arnaud de Villeneuve, University of Montepetlier,Oct 1998.

3.TREATMENT SCHEDULE:

In this study mifepristone given as 200mg single dose orally and observation period of 24 hours similar to the Wing DA etal , Elliot et al study and Kanan Yelikar in which mifepristone were compared with placebo whereas PGE2 gel in this study.

4.BISHOP'S SCORE AT THE START OF THE STUDY:

In our study,patients with bishop's score less than 4 were included in the study group. In this aspect, our study correlates with the study done by Elliot et al, Department of Obstetrics and Gynaecology, University of Edinburh, United Kingdom,1998 in which Bishop's score of 4 less were included in the study group.

In our study, mean Bishop's score at start of the study is 2.72,which is comparable with Kanan Yelikar study, where mean bishop's score at start is 2.02.

5. FAVOURABILITY OF BISHOP'S SCORE AT AUGMENTATION:

In our study, favourable Bishop score of 6 or more at augmentation was seen in 62% in primigravida and 90% in multigravida which was consistent with Frydman et al study, Giacalone et al study, Wing DA et al study and Elliot et al study.

In our study the mean Bishop's score at the end of 24 hours in mifepristone group is 5.68 which is comparable with Kanan Yelikar study where it is 5.04.

6.OXYTOCIN AUGMENTATION:

In our study,in the mifepristone group among 68 primigravida who required oxytocin, 53 delivered by labour natural. In PGE2 gel group 56 primigravida required oxytocin, 44 delivered bylabour natural . In mifepristone group, 8 multipara not required oxytocin, 6 delivered by labour natural, in PGE2 gel, 3 multipara not required oxytocin, 3 delivered by labour natural. In this aspect, our study correlates with the study done by Wing DA et al,2002 in which patient who delivers vaginally needed oxytocin for augmentation when mifepristone had been given.

7.DURATION OF FIRST AND SECOND STAGE OF LABOUR:

In this study, the mean duration of first stage and second stage in primi was 10.84 hours &26.38minutes in PGE2 gel and 14.97 hours &25.76 minutes in Mifepristone group repectively.In multipara, the mean duration of first stage and second stage is 9.23 hours &20.62 minutes in PGE2 gel and 11.63 hours & 18.48 minutes in mifepristone group repectively.These results are consistent with the normal WHO STANDARDS.

8. INDUCTION AND DELIVERY INTERVAL:

In this study, mean induction delivery interval in primi and muti in PGE2 gel was17.24 hours and 15.12 hours respectively, in mifepristone group,in primi and multi was 22.6 hours and 18.74hours.

Parity influenced the likelihood of vaginal delivery.

In this study the mean induction delivery interval in Mifepristone group was 21.33+4 hours which is comparitively less than the the randomized controlled trial conducted by Wing et al, in which mean induction delivery interval was 26.8+11 hours.

In this study 60 (60%) women 37% primigravida and 23% multigravida delivered vaginally within 24 hours and totally 82 (28%) women 55% primigravida and 27% multigravida delivered vaginally within 48 hours which was consistent with Wing DA et al study.

9.MODE OF DELIVERY:

S.NO	STUDY	YEAR	INCIDENCE OF VAGINAL DELIVERY
1.	Giacalone	1998	80.5%
2.	Lil et al	1996	80.88%
3.	Suh et al	1996	22.58%
4.	Wing DA et al	2002	87.5%
5.	This study	2016	82%

In this study vaginal delivery rate was 82% in Mifepristone group (55% primigravida and 27% multigraavida) the results were consistent with above mentioned studies except Suh et al study where the vaginal delivery is only 22.58%.

10.OUTCOME OF INDUCTION:

In this study the success of induction was vaginal delivery. Success rate was 82% in Mifepristone group which was consistent with 87.5% success rate in Wing DA et al study and 80.5% in Giacalone et al study.

LSCS rate was 18% with mifepristone group among which 8% is for Fetal distress and in this aspect our study is consistent with Wing DA et al study.

Our study is comparable with James P Neilson study(Cochrane Database Syst Rev 2009) which concluded that Mifepristone treated women less were likely to undergo cesarean section.

Our study is not comparable with James P Neilson study in which there is more likely chance to have an instrumental delivery whereas in our study it is only 2% of total deliveries.

11.INTRAPARTUM COMPLICATIONS:

In this study intrapartum complications like hypertonus, tachysystole or hyperstimulation were not encountered, which was consistent with Wing DA et al study.

This is in contrast to study conducted by Giacalone et al, Department of Obstetrics and Gynaecology, University of Montpellier, July 2001 in which Mifepristone treated group had higher rates of uterine hyperstimulation and tachysystole.

A total of 11% that is 9% primigravida and 2%multigravida had FHR abnormalities in our study which is consistent with study conducted by Wing et al in which abnormal FHR pattern were found in 18% of the study group.

12.MATERNAL COMPLICATIONS:

In this study,none of our study population had major complications like rupture uterus, chorioamnionitis, postpartum hemorrhage, puerperal sepsis.

A total of 10% study population had minor complications like nausea,vomiting, abdominal cramps in mifepristone group. In this aspect our study is consistent with the study conducted by Stenlund et al, Karolinska Hospital, Stocholm, Oct 1999.

13.NEONATAL COMPLICATIONS:

Meconium passage was encountered in 4% and NICU admission was 22.85% in mifepristone group & meconium passage was 7% and NICU admission was 33.74% in PGE2gel group.. APGAR score at 1minute & 5 minute were similar in both groups. But there was no neonatal mortality in both groups.

In this aspect,our study is consistent with study conducted by Wing DA et al, in which no statistically significant difference in neonatal outcome between mifepristone treated group and control group.

Our study is also comparable with Kanan Yelikar study in which there was no statistically significant difference in perinatal outcomes between two groups.

Mifepristone is administered orally which is very convenient and antenatal mothers can be ambulant when compared to cumbersome PGE2 gel administration which has to be instilled endocervically with strict asepsis by technically skilled personnel and needs observation in left lateral position.

Mifepristone is stored at room temperature whereas PGE2 gel storage needs cold chain maintenance the cost of mifepristone is comparable to PGE2 gel. Further need of oxytocin for augmentation is very much reduced with mifepristone when compared to PGE2 gel.

SUMMARY

The safety and efficacy of oral mifepristone as a preinduction cervical ripening agent is assessed in this study and compared with PGE2 gel. 200 antenatal mothers admitted at Thanjavur Medical College, who needed elective induction, satisfying the inclusion criteria were recruited into two groups and each were given oral mifepristone 200mg or endocervical PGE2 gel 0.5mg for cervical ripening and augmented with oxytocin. This study documents the success of induction, details of parturition, maternal and neonatal outcome.

This study revealed that Mothers in both groups had Bishop score of 2 to 4 at the start of study. 71% (44% primigravida and 27% multigravida) had favourable Bishop score in mifepristone group whereas only 62%(37% primigravida and 25% multigravida) in PGE2 gel group.

- Oxytocin augmentation is needed in 53(77.94%)primigravida in mifepristone group and 44(78.51%)primigravida in PGE2 gel group who delivered vaginally.
- Duration of II and III stage of labour shorter in miferpristone group .

- Cesarean section rate was 18% in mifepristone group whereas 27% in PGE2 gel group.
- Blood loss was less in mifepristone group.
- Neonatal complications and neonatal admissions were lesser in mifepristone group.
- Drug administration to delivery interval shorter with PGE2 group. Maternal complications were similar in both groups.
- The outcome of induction in this study reveals that the mifepristone was successful in 82% in achieving vaginal delivery whereas PGE2 gel was successful in 73%.

CONCLUSION

This study reveals that oral mifepristone is very safe and an effective drug for preinduction cervical ripening. It has an added advantage of ease of administration, better patient compliance and acceptance, shorter duration of II, III stages of labour, less blood loss with an overall success rate of 82%.

The drug has no untoward side effects on uterine contraction and no major maternal complications. This drug has safe nenatal outcome.

This drug is more effective in multigravida when compared to primigravida

Hence mifepristone offers advantages over PGE2 gel which is currently used for preinduction cervical ripening.

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PROFORMA

Name: Age: DOA: DOD:

Address: IP.No L.M.P: E.D.D:

SES:

HISTORY:

- History of Presenting complaints
- Booked Case: Yes/No.
- Obstetric History Gr P L A
- Menstrual History
- Past Medical / Surgical History
- Personal History
- Family History

GENERAL PHYSICAL EXAMINATION

Height Weight BMI

Pallor Pedal Edema

Pulse BP RR Temperature

CVS RS

Breast Thyroid

Per Abdomen – Uterine Size Activity

- Lie Presentation Position

- FHR

Per Speculum

Per Vaginum - Cx Dilatation

Position

Consistency

Effacement

Integrity of membranes

Presentation and Station

Pelvic Assessment

INVESTIGATIONS

1. Hb%

2. Urine-albumin

Sugar

Deposits

3. Bloodgroup&Rh typing

4. Blood-urea

-Sugar

5. Serum creatinine

6. HIV, VDRL, HBSAG

7. Obstetric scan – single, live/dead, fetus

- Cardiac activity&fetal movements
- B.P.D- cms weeks days
- F.L - cms weeks days
- Placenta-fundal anterior/posterior
- Grade maturity
- Liquor adequate/not
- Obvious congenital abnormalities

Bishop score on admission-

Indication for induction:

Date and time of induction-

Bishop score at time of induction-

Drug used for preinduction ripening:

-PGE2 gel/ Mifepristone

Wait period after induction

Bishop score at time of augmentation

Augmentation with oxytocin- yes/no

Dosage needed

DURATION OF LABOUR

First stage (Hrs)

Second stage (Mts)

Third stage (Mts)

NATURE OF DELIVERY

- Labour Natural/labour natural with episiotomy
- Insrumental delivery

  Outlet forceps

  Vacuum delivery
- LSCS Indication

Amount of blood loss at III stage

Drug administration to delivery interval

Complications-Maternal

Nausea/vomiting/diarrhea

Headache/hyperthermia/fever

Abdominal cramps

Chorioamnionitis/endometritis/

puerperal sepsis,

Uterine contraction abnormalities-

Tachysystole/hypertonus/

Hyperstimulation.

Any Treatment Given:

Intrapartum &Fetal Complications

1. Fetal heart rate abnormalities

2. Meconium passage-thin/thick

BABY

Birth weight

Apgar 1’ 5’

Congenital anomalies if any

Neonatal resuscitation

Neonatal admissions

Fetal complications-

-Meconium aspiration syndrome

-Hyperbilirubinemia

-Others

CONSENT FORM

I hereby give consent to participate in the study conducted by DR.SARANYA.A; Postgraduate in the Department of Obstetrics & Gynaecology, Thanjavur Medical College, Thanjavur and to use my personal clinical data and result of induction for the purpose of analysis and to study the safety and efficacy of the drug. I also give consent for any mode of delivery.

Place:

Time: Signature of Participant

KEY TO MASTER CHART

IP No. - In Patient Number

B - Booked

UB – Unbooked

TTN- Transient tachypnoea of newborn

SES-- Socioeconomic status

P- Primigravida

M- Multigravida

PP- Prolonged pregnancy

FHA – Fetal heart rate abnormality

DD interval - Drug administration to delivery interval

B.wt- Birth weight

NICU-Neonatal intensive care unit

LN - Labour natural

FI- Failed induction

FD- Fetal distress

MIFEPPRISTONE

						BISHOPS SCORE				DURATION			
S.NO	**NAME**	**AGE**	**IP.NO**	**BOOKED/ UNBOOKED**	**SES**	**PRIMI/ MULTI**	**AT START**	**AT AUGUMENT ATION**	**AUGUMENTATIO N WITH OXYTOCIN**	**AMOUNT OF OXYTOCIN**	**1ST[hr]**	**2nd[hr]**	**DD interval**
1	chellakili	21	443397	B	IV	P	3	4	YES	5	9	35	26
2	suganya	23	443968	B	IV	P	2	6	YES	5	7	20	23
3	saranya	22	444370	B	V	P	3	6	YES	5	8.3	25	18
4	mariyammal	24	444981	B	V	M	3	8	NO	NIL	6	15	14
5	selvarani	25	444755	B	V	P	3	6	YES	~	~	~	~
6	vidhya	24	444904	B	IV	P	3	8	YES	5	~	~	~
7	padmavathy	23	447081	B	III	M	3	6	YES	2.5	8	20	28
8	gayathri	22	445413	B	IV	P	3	8	YES	5	11	20	25
9	sathya	28	445370	B	IV	M	2	6	YES	5	~	~	~
10	malathy	27	447027	B	IV	P	3	6	YES	5	7.2	15	20
11	kavitha	31	445789	B	V	M	3	8	NO	~	5	17	16
12	vimala	23	456929	B	IV	P	3	4	YES	5	10	40	26
13	chitra	22	456687	B	IV	M	2	6	YES	2.5	7	20	18
14	anbarasi	24	456112	B	IV	M	3	6	YES	2.5	**9**	17	20
15	anitha	22	456127	B	IV	P	3	6	YES	5	11	35	24
16	keerthiga	28	465220	B	IV	M	4	6	YES	2.5	11	20	22
17	ilakiya	24	465328	B	IV	P	2	4	YES	5	16	30	26
18	vinitha	22	465238	B	IV	P	3	6	YES	5	20	40	28
19	abirami	20	465362	B	IV	P	3	4	YES	5	18	50	26
20	rekha	21	465362	B	IV	P	2	4	YES	5	12	20	20
21	rubini	23	465471	B	IV	P	2	4	YES	5	16	30	22
22	manjula	20	465477	B	IV	P	3	6	YES	5	18	20	26
23	roja	23	465319	B	IV	M	3	6	NO	~	14	10	20
24	sathya	20	465603	B	IV	P	4	4	YES	~	~	~	~
25	raniya	26	465303	B	IV	P	3	4	YES	5	16	22	10
26	Sophiya	22	471728	B	IV	P	3	6	YES	2.5	12	10	15

						BISHOPS SCORE					DURATION		
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOKED	SES	PRIMI/ MULTI	AT START	AT AUGUMENT ATION	AUGUMENTATIO N WITH OXYTOCIN	AMOUNT OF OXYTOCIN	1ST[hr]	2nd[hr]	DD interval
27	anikili	24	471726	B	IV	M	2	7	NO		10	15	15
28	sathya	27	471727	B	IV	M	3	4	YES				
29	kowsalya	21	471710	B	IV	P	3	4	YES	5	16	40	28
30	varalakshmi	25	471747	B	IV	P	2	6	YES	5	15	45	26
31	thenmozhi	22	471827	B	IV	P	4	4	YES	5	18	30	25
32	thamarai	22	471713	B	IV	P	3	4	YES	5			
33	sangetha	27	471898	B	IV	P	3	4	YES	5	12	30	28
34	anitha	22	471927	B	IV	M	4	6	YES	2.5	16	15	25
35	selvi	23	471962	B	IV	P	4	6	YES	2.5	16	16	24
36	Meena	27	471552	B	IV	P	3	6	YES	5	18	18	29
37	renganagi	25	471859	B	IV	P	2	6	YES	5	17	25	28
38	bhuveneshi	24	471807	B	IV	P	3	6	YES	5			
39	mariya	30	471987	B	IV	P	3	6	YES	5	16	35	23
40	radhika	21	472145	B	IV	P	3	4	YES	5	12	25	26
41	maheshi	20	472167	B	IV	P	3	5	YES	5	18	30	30
42	Lavanya	20	472167	B	IV	M	2	6	YES	2.5	14	25	25
43	veeramal	21	472154	B	IV	P	2	6	YES	5	16	20	24
44	Dhanam	25	472194	B	IV	P	2	6	YES	5	16	17	26
45	kowsalya	26	472227	B	IV	P	3	6	YES	5	13	20	23
46	Mariyastha	21	472377	B	IV	P	2	4	YES	5			
47	angayarasi	30	472396	B	IV	M	2	6	YES	5	14	25	26
48	maheshi	21	471997	B	IV	M	2	4	YES	2.5			
49	tamilselvi	22	472453	B	IV	P	3	6	YES	5			
50	devika	22	472453	B	IV	P	3	6	YES	5	18	20	26
51	sneha	19	467380	B	V	P	2	4	YES	5	14	25	25
52	Muthu	21	467332	B	V	P	2	6	YES	5	9	15	12
53	sathya	25	467387	b	iv	p	3	8	NO	5	11	18	14
54	lakshmi	32	467383	B	V	M	3	6	NO		10	10	13

						BISHOPS SCORE					DURATION		
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOKED	SES	PRIMI/ MULTI	AT START	AT AUGUMENT ATION	AUGUMENTATIO N WITH OXYTOCIN	AMOUNT OF OXYTOCIN	1ST[hr]	2nd[hr]	DD interval
55	pitchai	24	467400	B	IV	M	4	8	YES	2.5	7	12	11
56	nithya	28	467208	B	V	P	3	4	YES				
57	Pothumpon	23	467389	B	IV	P	3	6	YES	5	14	33	18
58	pavithra	31	46396	B	IV	M	2	6	YES	2.5	10	20	16
59	Pothumpon	23	467389	B	IV	M	2	8	NO		8	15	14
60	anushya	26	466722	B	V	P	3	6	YES	5	13	35	22
61	jeyanthi	24	467354	B	IV	P	3	6	YES	5	16	20	20
62	mariyammal	24	467459	B	IV	P	2	6	YES	5	15	30	23
63	Kaviyarasi	24	467449	B	V	M	2	8	NO		10	10	18
64	bhuveshi	29	467158	B	IV	P	3	6	yes	5	14	30	22
65	ragini	24	466992	B	V	P	3	6	YES	5			
66	ramya	26	464775	B	IV	M	2	6	YES	2.5	10	20	18
67	maha	23	467495	B	IV	P	3	8	YES	5	12	20	17
68	maheshi	22	467487	B	IV	M	2	6	YES	2.5	9	25	17
69	kowsalya	25	467494	B	V	P	3	6	YES	5	16	35	22
70	thennila	29	467335	B	IV	P	2	6	YES	5	15	40	23
71	sundari	28	467224	B	IV	P	3	8	yes	2.5	10	15	18
72	vennila	24	467224	B	IV	P	3	6	YES	5			
73	keerthiga	23	467574	B	IV	M	2	8	NO		8	20	13
74	sathya	29	467362	B	IV	M	3	6	YES	5	16	30	23
75	rengamal	30	467518	B	IV	M	2	8	YES	2.5	12	25	17
76	sathya	28	476676	B	III	P	3	4	YES	5	20	15	24
77	kala	24	476523	B	IV	P	3	6	YES	5	18	20	23
78	tamilselvi	25	476526	B	IV	P	3	6	YES	5	16	20	21
79	uma	22	476771	B	IV	P	2	4	YES	5			
80	pandimena	23	476639	B	IV	P	3	6	YES	5	19	35	24
81	durga	22	476637	B	IV	P	3	4	YES	5	17	20	22
82	parkavi	22	476222	B	IV	M	3	6	YES	2.5	15	25	17

						BISHOPS SCORE					DURATION		
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOKED	SES	PRIMI/ MULTI	AT START	AT AUGUMENT ATION	AUGUMENTATIO N WITH OXYTOCIN	AMOUNT OF OXYTOCIN	1ST[hr]	2nd[hr]	DD interval
83	sudha	28	476217	B	IV	P	3	6	YES	5	20	23	21
84	uma	23	476094	B	IV	P	2	4	YES	5			
85	manjula	28	476314	B	IV	M	2	6	YES	2.5	16	20	18
86	abirami	22	476820	B	III	P	3	6	YES	5	21	35	24
87	Kalai	24	476357	B	IV	P	3	6	YES	5			
88	Soundharya	22	476800	B	IV	P	3	4	YES	5	17	20	22
89	nirmala	29	476980	B	IV	M	2	6	YES	2.5	18	12	21
90	durga	28	476962	B	III	P	3	4	YES	5	22	30	25
91	Parameshi	21	476962	B	III	M	2	6	YES	2.5	15	15	18
92	revathy	30	476909	B	IV	P	3	6	YES	5	16	20	20
93	amutha	22	476173	B	IV	M	2	4	YES	2.5	18	21	22
94	kokila	27	476973	B	IV	P	3	6			14	25	18
95	vinodhini	22	477226	B	IV	P	2	6	YES	5	18	20	21
96	Madhu	19	475647	B	IV	P	3	4	YES	5	15	20	18
97	sathya	26	477174	B	IV	P	3	4	YES	5			
98	vinodhini	22	477183	B	III	P	3	4	YES	5			
99	madhu	20	477015	B	IV	P	3	6	YES	5	19	15	23
100	veeramal	23	477015	B	IV	M	2	6	YES	2.5	18	20	21

			COMPLICATION			APGAR			
S.NO	**MODE OF DELIVERY**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
1	LN	5	150	NIL	NIL	3.1	5	8	NO
2	LN	7	250	NIL	NIL	2.5	7	8	NO
3	LN	3	200	NIL	NIL	3.6	5	7	NO
4	LN	5	250	NAUSEA	FHA	2.7	5	8	YES
5	LSCS(FD)	~	500	~	FHA	3	4	6	NO
6	LSCS(FI)	~	600	~	~	2.6	5	8	NO
7	LN	7	300	NAUSEA	~	2.6	5	7	NO
8	LN	4	100	abd.cramp	~	2.9	7	8	NO
9	LSCS(FI)	~	500	~	~	3.25	4	6	NO
10	LN	5	200	~	~	2.75	7	8	NO
11	LN	6	250	~	~	3	7	8	NO
12	LN	8	200	~	~	4	5	8	NO
13	LN	5	250	~	~	3	7	8	NO
14	LN	7	100	~	~	3.3	5	8	NO
15	LN	5	200	~	~	2.9	5	8	NO
16	LN	10	100	~	~	2.75	5	8	NO
17	LN	10	200	~	~	2.75	5	7	N0
18	LN	8	100	~	~	3.1	5	7	NO
19	Outlet	8	150	~	FHA	3.5	5	7	YES
20	LN	7	150	~	~	2.5	5	7	NO
21	LN	6	100	~	~	2.5	5	7	NO
22	LN	8	100	~	~	2.6	5	7	NO
23	LN	10	100			3	5	7	NO
24	LSCS(FI)		500			2.6	4	6	N0
25	LN	10	100			3	5	7	NO
26	LN	10	150			2.25	4	7	NO

			COMPLICATION			APGAR			
S.NO	MODE OF DELIVERY	3rd STAGE OF LABOUR	BLOOD LOSS	MATERNAL	FETAL	B.WT (kg)	1min	5min	NICU admission
27	LN	8	100			3	5	7	NO
28	LSCS(FI)		500			3	4	6	YES
29	LN	10	100			2.9	5	7	NO
30	VAC DEL	6	200			3	4	6	YES
31	LN	7	100			2.5	5	7	NO
32	LSCS (FD)		500		FHA	2.9	4	6	YES
33	LN	10	100			3	5	7	NO
34	LN	10	100			2.7	5	7	NO
35	LN	8	100			2.5	5	7	NO
36	LN	10	150			2.75	5	7	NO
37	LN	10	150	abd cramps		3.4	5	7	YES
38	LSCS (FI)		500			3.2	5	7	NO
39	LN	10	100			2.5	5	7	NO
40	LN	7	100			3	5	7	NO
41	LN	8	100			3	5	7	NO
42	LN	8	100			3.25	5	7	NO
43	LN	10	100			3.1	5	7	N0
44	LN	10	100			2.75	5	7	NO
45	LN	10	100			3	5	7	NO
46	LSCS(FD)		500		FHA	2.6	3	5	YES
47	LN	10	100			2.6	5	7	NO
48	LSCS(FD)		600		FHA	3.25	4	8	NO
49	LSCS(FI)		500			2.8	5	7	NO
50	LN		100			2.8	5	7	NO
51	LN	4	150			2.6	5	7	NO
52	LN	3	100			2.6	5	7	N0
53	LN	3.5	100			2.75	4	6	NO
54	LN	4	150			2.75	5	7	NO

			COMPLICATION			APGAR			
S.NO	**MODE OF DELIVERY**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
55	LN	3	100	NAUSEA		3.1	5	7	NO
56	LSCS(FI)		600			2.75	5	7	NO
57	LN	4	150			3	4	7	NO
58	LN	3	100			3.2	5	7	NO
59	LN	3	100			3.2	5	7	NO
60	LN	5	150			3.1	4	7	NO
61	LN	5	100			2.5	5	7	NO
62	LN	3	100			2.5	5	7	NO
63	LN	3	150			3	4	7	NO
64	LN	5	100	vomitting		3.5	4	7	NO
65	LSCS(FD)	5	180		FHA	2.9	4	5	YES
66	LN	5	150			2.5	4	7	NO
67	LN	3	100			3	5	7	NO
68	LN	5	100			2.75	4	7	NO
69	LN	3	500			2.5	4	7	NO
70	LN	3	100			3	5	7	N0
71	LN	3	150			3.5	5	7	NO
72	LSCS(FD)		500		FHA	3.5	5	7	YES
73	LN		150			3.2	4	6	YES(FHA)
74	LN		100			3.1	4	7	NO
75	LN		150			2.5	4	7	NO
76	LN	3	100			3.25	5	7	NO
77	LN	2.5	100			2.75	5	7	NO
78	LN	3.5	150			2.75	5	7	NO
79	LSCS(FD)	2	150		FHA	3	3	7	YES
80	LN	3.5	150			2.75	5	7	NO
81	LN	4	100			2.8	5	7	NO
82	LN	3	100			2.9	4	7	N0

			COMPLICATION			APGAR			
S.NO	**MODE OF DELIVERY**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
83	LN	3.5	150	vomit		2.9	5	7	YES
84	LSCS (FI)	3	550		FHA	3	4	7	NO
85	LN	3	150			2.75	5	7	NO
86	LN	3.5	150			2.75	5	7	NO
87	LSCS(FD)	3	500		FHA	2.5	5	7	NO
88	LN	3.5	100			2.75	5	7	NO
89	LN	3	100	VOMIT		2.8	5	7	NO
90	LN	3	150	NAUSEA		3.7	4	7	NO
91	LN	3.5	100			3.5	5	7	NO
92	LN	4	150			2.9	5	7	NO
93	LN	3	100			3	5	7	NO
94	LN	3	100			3	5	7	NO
95	LN	3.5	120			2.7	4	7	NO
96	LN	3	100			3	4	7	NO
97	LSCS(FI)	2.6	500			2.6	4	7	NO
98	LSCS(FI)	3.5	500			3.1	5	7	NO
99	LN	3	150			2.7	5	7	NO
100	LN	3	100	abd cramps		2.7	4	7	NO

PGE2 GEL

						BISHOPS SCORE					DURATION			
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOK	SES	PRIMI/ MULTI	AT START	AT AUGUMEN	2nd/3rd GEL	AUGUMENTATION WITH	AMOUNT OF	1ST[hr]	2nd[mt}	DD interval
1	RAMYA	20	433783	B	IV	P	3	6		Y	5	11	20	28
2	FATHIMA	20	443551	B	IV	P	2	6	2	Y	5	8	18	22
3	KUSHPO	23	444305	B	IV	P	4	4		Y	5			
4	KUSHMA	33	444075	B	IV	M	2	6	2	Y	2.5	7	12	18
5	ESHWARI	29	444283	B	IV	P	3	6		Y	5	9	20	20
6	AKILA	29	444278	B	V	P	3	4	2	Y	5			
7	SAMEERA	23	445306	B	IV	M	2	6		Y	5			
8	KAVITHA	23	444385	B	IV	P	3	6	3	Y	5	8	20	24
9	ILAMATHY	21	444393	B	V	M	2	4		Y	5	7	17	21
10	SARANYA	22	444350	B	IV	P	3	4		Y	5			
11	CHANDRA	21	447365	B	V	M	4	6		Y	5			
12	LATHA	23	466055	B	IV	P	3	6		Y	5	9.3	30	24.2
13	SURYA	21	465787	B	IV	P	3	3		Y	5	9	35	28
14	BRITTA	21	466054	B	IV	P	4	6		Y	5	12	45	28
15	SARANYA	25	466114	B	IV	P	3	6	2	Y	5	14	50	30
16	SABITHA	20	465686	B	IV	P	3	4	2	Y	5			
17	MALAR	24	466213	B	IV	P	2	8		Y	5	12	30	21
18	ANANDHI	23	466233	B	IV	M	2	6		Y	2.5	10	25	14
19	NALINI	28	465306	B	IV	M	4	4	2	Y	2.5			
20	AKILA	25	465937	B	IV	P	4	6		Y	5	11	25	19
21	GAYATHRI	20	466273	B	IV	P	3	6	2	Y	5	11	25	19
22	DEIVA	26	465457	B	IV	M	2	6		Y	2.5	9	20	18
23	SUMATHY	25	466166	B	IV	P	3	6	2	Y	5	16	30	28
24	CHANDRA	21	465876	B	IV	P	3	4	2	Y	5	15	50	25
25	ANANYA	26	466287	B	IV	M	3	4		Y	2.5			
26	SUGANTHI	24	466047	B	IV	M	2	6		Y	2.5	9	25	20
27	BEEVI	25	466338	B	IV	M	4	6				7	20	17
28	MANJULA	26	466252	B	IV	P	3	4			5			
29	MERCY	21	466009	B	V	P	3	4			5			

						BISHOPS SCORE					DURATION			
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOK	SES	PRIMI/ MULTI	AT START	AT AUGUMEN	2nd/3rd GEL	AUGUMENTAT ION WITH	AMOUNT OF	1ST[hr]	2nd[mt}	DD interval
30	RAMYA	18	466347	B	IV	P	2	8		Y	5	8	30	16
31	SASIKALA	29	466262	B	IV	P	3	8		Y	5			
32	AMBIGA	26	466314	B	V	M	2	6		Y	2.5	11	20	22
33	AMBIGA	26	466314	B	V	M	2	6		Y	2.5	11	20	22
34	LAKSHMI	29	466570	B	V	M	2	8	2	Y	2.5	16	25	26
35	MEENA	20	466327	B	V	P	3	4		Y	5	18	20	28
36	RAMYA	24	466390	B	V	P	3	6	2	Y	5	14	20	24
37	SELVA	23	466001	B	IV	P	3	4	2	Y	5			
38	ANBARASI	26	466383	B	IV	M	2	8		Y	2.5	9	25	16
39	NIRMALA	30	466056	B	V	M	2	6		Y	2.5	8	26	16
40	RADHIKA	34	461385	B	IV	P	3	4						
41	VASANTHI	24	466342	B	V	P	2	6	3	Y	2.5	18	20	22
42	SAROJA	20	464977	B	V	M	3	4	2	Y	2.5	15	15	20
43	ABINAYA	23	465868	B	IV	P	3	6		Y	5	12	20	18
44	SUGANYA	20	466470	B	V	P	3	4		Y	5	16	18	22
45	KALA	27	466307	B	IV	M	2	8				8	12	14
46	RADHA	20	466460	B	IV	P	3	4		Y	5	7	15	12
47	THEN	22	466408	B	IV	P	3	4						
48	KASTHURI	23	466674	B	V	M	2	8	2	Y	2.5	13	18	16
49	KALPANA	22	466037	B	IV	P	2	6		Y	5	16	20	26
50	MAHESHI	21	466740	B	V	P	2	4		Y	5			
51	VEERAMAL	22	467386	B	V	P	3	6		Y	5	7	30	12
52	KANCHA	24	466456	B	IV	P	2	6		Y	5	9	25	11
53	KALAI	25	466719	B	IV	P	3	4		Y	5			
54	PRIYA	24	467460	B	V	P	3	6		Y	5	11	20	13
55	LEELA	22	467589	B	V	P	2	6	2	Y	5	14	35	16
56	KASTHURI	25	467525	B	IV	P	2	6		Y		7	35	10
57	KIMTHIGA	26	467512	B	IV	P	3	4	2	Y		12	40	15
58	SUDHA	28	466733	B	V	M	2	6		Y		9	25	12
59	RAMYA	25	466780	B	V	P	3	4	3	Y				

						BISHOPS SCORE					DURATION			
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOK	SES	PRIMI/ MULTI	AT START	AT AUGUMEN	2nd/3rd GEL	AUGUMENTATION WITH	AMOUNT OF	1ST[hr]	2nd[mt}	DD interval
60	DHARSHINI	25	467780	B	IV	M	2	6		Y		7	15	9
61	DEEPA	25	465381	B	IV	P	3	6						
62	SELVI	24	466206	B	IV	P	3	6	2	Y		12	25	14
63	MALATHY	29	467334	B	IV	P	3	4						
64	MANJULA	30	467605	B	IV	M	3	6		Y		7	20	9
65	BHUNESHI	21	467603	B	III	P	2	6		Y		6	19	8
66	VENNILA	26	467610	B	IV	M	2	6		Y		8	11	10
67	UMAPATHI	27	467613	B	IV	M	3	6		Y		12	30	15
68	RANJITHA	24	467337	B	IV	P	3	4		Y		12	30	15
69	GENITA	24	467617	B	IV	M	2	6		Y		6	20	9
70	KANAGA	24	467617	B	IV	P	3	4		Y				
71	RADHA	25	466810	B	IV	P	3	6		Y				
72	RADHIKA	25	467785	B	IV	M	3	4		Y		9	35	13
73	ANANDHI	29	467745	B	IV	P	3	6		Y		7	20	11
74	NANDHINI	25	467756	B	III	M	2	6	2	Y		10	20	14
75	KEERTHI	24	467767	B	IV	P	2	6		Y		6	25	9
76	PUSHPA	26	476850	B	III	P	3	4	2	Y	5			
77	SHEELA	26	476978	B	IV	P	3	6		Y	5	10	25	13
78	MARITA	27	476807	B	IV	P	3	6		Y	5	12	20	14
79	PRIYA	24	476949	B	IV	P	3	4		Y	5	11	25	13
80	AYSHA	21	476997	B	III	P	3	4						
81	SATHYA	26	476987	B	III	P	2	6		Y	5	10	20	14
82	YAZHINI	19	477110	B	III	P	3	4	2	Y	5	11	25	15
83	PRIYA	24	477154	B	IV	P	3	4						
84	JEYANTHI	25	477160	B	IV	P	3	6		Y	2.5	9	15	11
85	MAHA	30	477158	B	III	P	2	6				8	25	10
86	MADHAVI	23	476668	B	IV	P	3	4	2			12	20	14
87	MENAKA	22	477207	B	III	P	3	6		Y	5	11	25	13
88	GAYU	25	477171	B	IV	M	3	6		Y	2.5	9	20	11
89	MERLIN	22	476118	B	IV	P	3	4		Y	5	11	35	14

						BISHOPS SCORE					DURATION			
S.NO	NAME	AGE	IP.NO	BOOKED/ UNBOOK	SES	PRIMI/ MULTI	AT START	AT AUGUMEN	2nd/3rd GEL	AUGUMENTAT ION WITH	AMOUNT OF	1ST[hr]	2nd[mt}	DD interval
90	SARITHA	25	477208	B	IV	M	2	6		Y	2.5	7	10	10
91	MENAKA	27	477004	B	III	M	3	6	2			9	30	12
92	JEEVITHA	25	477546	B	IV	P	3	4						
93	ANJALI	22	477655	B	IV	M	3	6		Y	2.5	7	20	9
94	SUGANYA	25	477259	B	IV	P	3	4						
95	JEYANTHI	23	477655	B	IV	P	3	4		Y	5	9	25	12
96	TAMIL	24	477419	B	III	P	2	6		Y	5	10	35	12
97	SARANI	24	477252	B	IV	P	3	4	2			11	25	14
98	ABINAYA	21	477585	B	IV	P	3	6		Y	5	9	30	11
99	RAJI	21	477694	B	IV	P	3	6		Y	5	8	25	12
100	RAJITHA	26	477703	B	III	P	3	4	2					

							APGAR			
S.NO	**NAME**	**MODE OF DELIVER**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
1	RAMYA	LN	5	150			2.5	7	8	
2	FATHIMA	LN	7	100			3.1	6	8	
3	KUSHPO	LSCS(FD)		600		FHA	2.7	4	6	YES
4	KUSHMA	LN	8	200			3	6	7	
5	ESHWARI	LN	5	150			2.4	5	7	
6	AKILA	LSCS(FD)		500		FHA	3	4	6	YES
7	SAMEERA	LSCS(FI)		600			3.3	5	7	
8	KAVITHA	LN	7	200			3.5	5	7	
9	ILAMATHY	LN	5	150			2.7	5	7	
10	SARANYA	LSCS(FI)		500			3.3	6	7	
11	CHANDRA	LSCS(FD)		600		FHA	3.4	4	7	YES
12	LATHA	LN	6	300	NAUSEA		2.9	5	7	
13	SURYA	LN	5	150			3.3	5	7	
14	BRITTA	LN	3	150		FHA	3.14	4	6	YES
15	SARANYA	VD	5	300		FHA	2.7	4	6	YES
16	SABITHA	LSCS(FI)		500			3	5	7	
17	MALAR	LN	4	100			2.5	5	7	
18	ANANDHI	LN	5	200			3	5	7	
19	NALINI	LSCS(FI)		600			2.8	5	7	
20	AKILA	LN	7	200	FEVER		3	5	7	
21	GAYATHRI	LN	7	200			3.4	5	7	
22	DEIVA	LN	4	200			2.7	5	7	
23	SUMATHY	LN	6	150		FHA	2.5	4	6	YES
24	CHANDRA	OF	3	250			3.4	5	7	
25	ANANYA	LSCS(FD)		600		FHA	3.2	4	6	YES
26	SUGANTHI	LN	4	100			3	5	7	
27	BEEVI	LN	4	150			3	5	7	
28	MANJULA	LSCS(FI)	5	600			3.35	5	7	
29	MERCY	LSCS(PD)		500			3.5	5	7	

							APGAR			
S.NO	**NAME**	**MODE OF DELIVER**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
30	RAMYA	OUTLET	5	250	VOMIT		2.7	5	7	
31	SASIKALA	LSCS(FD)		650			3.3	4	6	YES
32	AMBIGA	LN	4	150			2.6	5	7	
33	AMBIGA	LN	4	150			2.6	5	7	
34	LAKSHMI	LN	6	300			3	5	7	
35	MEENA	LN	5	150			3	5	7	
36	RAMYA	LN	5	150			3	5	7	
37	SELVA	LSCS(FI)		650		TTN	3.2	4	6	YES
38	ANBARASI	LN	5	150			2.6	5	7	
39	NIRMALA	LN	5	150			2.9	5	7	
40	RADHIKA	LSCS(FD)		600		TTN	3.1	4	6	YES
41	VASANTHI	LN	4	150			2.7	5	7	
42	SAROJA	LN	5	100			2.5	5	7	
43	ABINAYA	LN	7	150			2.9	5	7	
44	SUGANYA	LN	4	150			2.5	5	7	
45	KALA	LN	3	100			2.5	5	7	YES
46	RADHA	LN	5	150			2.6	5	7	
47	THEN	LSCS(FD)		500		FHA	2.9	4	6	YES
48	KASTHURI	LN	4	100			3.3	5	7	
49	KALPANA	LN	3.5	200			2.5	5	7	
50	MAHESHI	LSCS(FI)		650		FHA	3.1	3	6	YES
51	VEERAMAL	LN	4	200			2.9	5	7	
52	KANCHA	LN	3.5	150			2.5	5	7	
53	KALAI	LSCS(PD)		600			2.9	5	7	
54	PRIYA	LN	3.5	150			3.5	5	7	
55	LEELA	LN	3	100			3	3	7	
56	KASTHURI	LN	5	150			3	5	7	
57	KIMTHIGA	LN	3	100			3.2	5	7	
58	SUDHA	LN	3.5	150	NAUSEA		3	5	7	
59	RAMYA	LSCS(FI)		500			3.3	5	7	

							APGAR			
S.NO	**NAME**	**MODE OF DELIVER**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
60	DHARSHINI	LN	5	150			2.8	3	6	
61	DEEPA	LSCS(FD)		500		FHA	2.8	4	7	YES
62	SELVI	LN	3.5	150			3.5	4	7	
63	MALATHY	LSCS(PD)		500			3.3	4	7	
64	MANJULA	LN	2.5	650			2.7	5	7	
65	BHUNESHI	LN	3	150			2.7	5	7	
66	VENNILA	LN	3	100			3	5	7	
67	UMAPATHI	LN	3	150			3.1	5	7	
68	RANJITHA	LN	4	100			2.7	3	7	
69	GENITA	LN	3.5	100			2.7	3	7	
70	KANAGA	LSCS(FD)		1000		FHA	2.5	3	5	YES
71	RADHA	LSCS(FI)		500			3	5	7	
72	RADHIKA	LN	4	150			3	5	7	
73	ANANDHI	LN	3.5	200			3	5	7	
74	NANDHINI	LN	3	150			2.8	5	7	
75	KEERTHI	LN	3.5	100			3.01	5	7	
76	PUSHPA	LSCS(FD)	5	500	NAUSEA	FHA	3.1	3	6	YES
77	SHEELA	LN	5	250			2.6	4	7	
78	MARITA	LN	3	100			3.2	4	7	
79	PRIYA	LN	3.5	100			2.6	5	7	
80	AYSHA	LSCS(FD)	3	500		FHA	2.9	4	7	YES
81	SATHYA	LN	3	150	VOMIT		2.5	5	7	
82	YAZHINI	LN	3.5	100			2.5	4	7	
83	PRIYA	LSCS(FD)	4	550		FHA	2.5	3	6	YES
84	JEYANTHI	LN	2.5	150			2.5	4	7	
85	MAHA	LN	2.5	150			3.1	4	7	
86	MADHAVI	LN	2.8	100		FHA	2.8	3	6	YES
87	MENAKA	LN	3	150			2.6	4	7	
88	GAYU	LN	2.5	100			2.75	4	7	
89	MERLIN	LN	3	100			3	4	7	

							APGAR			
S.NO	**NAME**	**MODE OF DELIVER**	**3rd STAGE OF LABOUR**	**BLOOD LOSS**	**MATERNAL**	**FETAL**	**B.WT (kg)**	**1min**	**5min**	**NICU admission**
90	SARITHA	LN	3	100			3	5	7	
91	MENAKA	LN	4	150			3	5	6	
92	JEEVITHA	LSCS(FD)	3	550			2.8	3	6	YES
93	ANJALI	LN	3.5	100		FHA	2.8	5	7	
94	SUGANYA	LSCS(FD)	2.5	500		FHA	2.8	3	6	YES
95	JEYANTHI	LN	3.4	100			3.4	5	7	
96	TAMIL	LN	3	150			2.7	5	7	
97	SARANI	LN	3	100			2.9	5	7	
98	ABINAYA	LN	2.5	150			2.7	3	6	YES
99	RAJI	LN	3	100			3	4	7	
100	RAJITHA	LSCS(FI)	3	500			3.5	5	7	