COMPARATIVE STUDY OF INTRAVAGINAL MISOPROSTOL WITH INTRACERVICAL DINOPROSTONE GEL FOR INDUCTION OF LABOUR

By

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IN

OBSTETRICS AND GYNAECOLOGY

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2017

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ABBREVATIONS

PG	-	Prostaglandin
CTG	-	Cardiotocogram
GAG	-	Glycosaminoglycans
NICU	-	Neonatal Intensive Care Unit
ACOG	-	American College of Obstetrician and Gynaecologists
PPH	-	Post Partum Haemorrhage
LSCS	-	Lower Segment Caesarean Section
FHR	-	Fetal Heart Rate
µg	-	Micro gram
MBS	-	Modified Bishop's Score
mg	-	Milligrams

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INTRODUCTION

Labour is a final consequence of Pregnancy and is inevitable. The timing of onset of labour may vary widely, but it will happen sooner or later.

Induction of labour implies the artificial initiation of uterine contractions after period of viability for the purpose of vaginal delivery where as augmentation of labour is a process of stimulation of uterine contractions that are already present but found to be inadequate[1]. Induction of labour is indicated when continuation of pregnancy risks the life of mother or fetus. The baby should be delivered in a good condition, in an acceptable time frame and with minimum maternal discomfort and least side effects.

In order to be successful, induction of labour must lead to adequate uterine contractions which increases in frequency, duration and progressive dilatation of cervix. It should result in vaginal delivery, as there is little purpose in bringing about labour as a mere preparation for caesarean section[1]. The aim is to achieve vaginal delivery with minimal risk to mother and fetus.

The cervix is an organ of diverse properties. Ripening of the cervix takes place before the onset of labour resulting in increased softening, effacement. Pharmacologically and physiologically prostaglandins have two direct actions associated with labour. They are ripening of the cervix and myometrial contractility. Induction of Labour was one of the first indications for the use of prostaglandins in obstetrics.

The method of administration that has been well known is endocervical Dinoprostone or prostaglandin E_2. Though this is widely used, the disadvantage is that it is expensive and required refrigeration for storage with warming before use. Later, a comparably cheap, safe and effective vaginally administered Prostaglandin,

which claims to have limited side effects available with the name Misoprostol or PGE_1 in tablet form. It does not need any refrigeration.

A number of recently published clinical trials abroad and in India have shown that intravaginal Misoprostol is an effective agent for induction of labour and cervical ripening at term, when compared to other methods of labour induction.

In this study, intracervical dinoprostone (PGE_2) gel is compared to intravaginal misoprostol in the induction of labour and its efficacy and safety for the mother and fetus.

NEED FOR THE STUDY

Induction of labour is common procedure of obstetric practice[2]. It is indicated in 10% - 15%[2] of pregnant women and it should lead to regular uterine contractions and progressive dilatation of cervix in order to achieve vaginal delivery. Common indications for induction of labour are postmaturity, preeclampsia/ eclampsia, antepartum hemorrhage, chronic hydramnios, renal diseases, COPD, congenital anomalies, mild IUGR. Induction of labour with unfavourable cervix may lead to prolonged labour[3] or failure of induction. So, cervical ripening is essential to get a successful outcome.

Local prostaglandins act by increasing collagenase which causes beakdown of collagen in cervical tissue and by altering glycosa-aminoglycans (GAG) and proteoglycans in cervical tissue which causes collagen fibers dispersion[4]. The drug used for induction of labour should reduce the interval between initiation of labour and delivery of baby, should need less repeated doses and should cause minimal side effects to mother and fetus. Oral misoprostol is not preferred as in low doses it is not effective where as in high doses it causes hyperstimulation, uterine tachysystole, GIT disturbances[5].

Both misoprostol and dinoprostone are prostaglandins. Misoprostol is PGE_1 analogue and dinoprostone is PGE_2. Both act locally on cervix and uterus. There is need to assess which is better prostaglandin in terms of efficacy and safety.

AIMS & OBJECTIVES

- To compare efficacy of induction of labour with Dinoprostone gel and Misoprostol in induction of labour with respect to induction delivery interval, Oxytocin Augmentation, type of delivery, cost effectiveness.
- To study the maternal and fetal outcome in both groups.

REVIEW OF LITERATURE

The need for termination of pregnancy for safeguarding the maternal and fetal health has been recognized and the search for effective and ideal method for induction of labour has been going on for more than 10 decades.

Blanchette Howard A, Sandhya Nayak[6], 1999, studied on cervical ripening and induction of labour involving 81 patients in the cerviprime group and 145 patients in misoprostol group. Intravaginal Misoprostol administered with an initial dose of 25ug with subsequent dosage option of 25 and 50ug every 4 hours to a maximum of 6 doses as needed. In the Cerviprime group it was 0.5 mg was administered intracervically every 6 hours a maximum of 3 doses. The mean time to delivery interval was significally shorter with Misoprostol (19.8+/_10.4 hours) than with PGE2 (31.3+/- 13.0 hours). Delivery within 24 hours of induction was significantly more in Misoprostol group . So, they concluded that, Misoprostol is more effective in cervical ripening and induction of labour and is as safe for patients who don't have a history of caesarean birth when compared with $PGE_{2.}$

Chunch Frank J and B.Joyce Huffaker[7], 1995, did a randomized controlled study, studied 103 patients with PGE1 induction, 50ug intravaginally or with prepidil gel, 0.5 mg intracervically, every 4 hours until active labour. Induction to delivery interval was significantly shorter with the misoprostal group (11.4 vs 18.9 hours <0.001) and fewer patients in the misoprostal group required oxytocin augmentation . No significant differences were noted in mode of delivery or an adverse maternal, fetal or neonatal effects. So, they concluded, intravaginal misoprostal is a more effective, lower cost agent for induction of labour than in intracervical dinoprostone gel and is comparable in safety.

Kolderup Lindsay, Lynn McLean[8], 1999, did a randomized controlled study of labour induction and patients were randomly assigned to receive either 50 ug of intravaginal misoprostol every 4 hours or 0.5 mg of intracervical dinoprostone (PG E2) every 6 hours. 159 women were randomly assigned to receive misoprostol (n=81) or prepidil (n=78). Mean time of induction to delivery was significantly less/shorter in the misoprostol group (19.50 hours) than in prepidil group (28.52 hours) P=0.005). Only 58% of women in the Misoprostol group needed Oxytocin augmentation when compared with 88% woman receiving prepidil (P=0.00002). In conclusion misoprostol was more efficacious than prepidil for labor induction. However, there is significantly increased incidence of abnormal FHR tracings and the trend in increased deliveries for fetal distress with misoprostol dosing of 50 ug every 4 hours, was observed and was the point of concern in this study.

Deborah A.Wing Ann Rahall[9], in 1995 studied 276 patients with indications for induction of labour and unfavorable cervices. They were randomly assigned to receive either 25ug intravaginally every 3 hours with a maximum of 8 doses or PGE2 gel form 0.5 mg intracervically every 6 hours with a maximum of 3 doses. The average mean interval induction to vaginal delivery was shorter in misoprostol group (1323.0+/- 844 minutes) than in the dinoprostone group (1532.4 +/- 706.5 minutes) (P<0.05). Need for oxytocin augmentation of labour occurred more commonly in the dinoprostone group (72.6%) than in the misoprostol group (45.7%) (P<0.0001). There were no significant differences in the routes of delivery. So they concluded that intravaginal misoprostol and dinoprostone for cervical ripening and labour induction were equally effective and the cost of misoprostol is lesser compared to that of dinoprostone.

David Buser, Gerardo Mora[10], 1997, in their study of 155 women admitted for

induction of labour who were randomized to receive one of two methods: intravaginal misoprostol, 50ug every 4 hours upto 3 doses and intracervical dinoprostone gel, 0.5ug every 6 hours upto a maximum of 3 doses, Misoprostol was more effective than dinoprostone in cervical ripening (P=0.01), induction of labour (P<0.001), shortening the duration of labour (P<0.001). In conclusion, misoprostol was found to be more effective agent for cervical ripening and labour induction.

Varaklis et al,[11] 1995, in his study concluded Misoprostol to be more effective than Dinoprostone but further work was needed regarding ideal dosing regimen.

In 2000, Jose L.Bartha[12] compared efficacy, safety, and tolerance of intracervical dinoprostone with oral misoprostal for cervical ripening and labour induction . 200 women were randomized into 2 groups to receive either single dose of oral misoprostol, 200 ug or 0.5 mg of dinoprostone intracervically every 6 hours for a maximum four doses. The intervals from adminstration of the drug to active phase of labor, and to rupture of membranes and induction to delivery time were significantly shorter with the misoprostol group. In conclusion, a single dose of 200 ug oral misoprostol was more effective for cervical ripening and labour induction than 0.5 mg of intracervical dinoprostone.

In 2001, Michigan State University, U.S.A. French L.[13] conducted Cochrane study on oral misoprostal in induction of labour. In the author's aspect, oral PG consistently resulted in more frequent gastro-intestinal side effects in particular vomiting compared with the other groups in the study. He concluded that there were no clinical advantages of oral PG over the other methods for induction of labour.

In 2001, Neiger R. Greaves PC [14] compared the efficacy of intracervical dinoprostone to intravaginal misoprostal (Cytotec) for pre-induction cervical

ripening. 61 patients admitted for induction of labour, whose cervices were unfavorable (Bishop score: 4) were randomly divided into 2 groups to receive either intravaginal placement of a 50 micrograms misoprostol tablet or intracervical administration of dinoprostone gel. Five women (17%) in the dinoprostone group and eighteen women (56%) in the misoprostol group achieved cervical ripening within 12 hours (P=0.007). Fewer doses of misoprostol were required to achieve cervical ripening, and the interval from induction of labour to delivery was short as well. Sixteen patients (50%) in the misoprostol group required oxytocin, whereas 26 (90%) in the dinoprostone group required oxytocin augmentation (P=0.008). There was no significant difference in mode of delivery or neonatal outcome between the two groups. They concluded that vaginal misoprostol appears to be a more effective as cervical ripening agent than cervical dinoprostone.

In 2003, Agarwal N, Gupta A[15], conducted prospective clinical trials to assess the safety and efficacy of 6-hourly vaginal misoprostol versus intracervical dinoprostone for induction of labour. 120 pregnant women requiring induction of labor were recruited. Cases were randomized to receive either 50ug vaginal misoprostol 6 hourly (group 1, n = 60) or 0.5 mg intracervical dinoprostone 6 hourly (group Ii, n = 60). Results, such as change in Bishop's score, need of oxytocin, induction delivery interval; complications like tachysystole, hyperstimulation, abnormal fetal heart rate, and meconium passage were compared between two groups. They found that Bishop Score rise, after 6 hour of initiation of therapy was significantly higher in the misoprostol group when compared to dinoprostone. The need of oxytocin augmentation was less in misoprostol when compared to dinoprostone group. Induction delivery interval was shorter in misoprostol group. 12.8 +/- 6.4 hour versus 18.53 +/- 8.5 hour in dinoprostone group (P=<0.01). Side

effects in misoprostol group are tachysystole, abnormal heart rate pattern. They concluded that vaginal misoprostol 50 ug 6-hourly is safe and effective for induction of labor with lesser need of oxytocin augmentation and shorter induction delivery interval.

In February 2003, Amali U Lokugamage[16] conducted a study to compare the safety and efficacy of intracervical dinoprostone and intravaginal misoprostol for induction of labour and to quantify the clinical response to suspicious cardiotocographic (CTG) readings The induction to delivery interval, delivery within 12 hours and deliver within 24 hours were all shorter in the misoprostol group. There were no differences in rates of oxytocin augmentation, tachysystole and hyperstimulation syndrome and neonatal outcome. They concluded that intravaginal misoprostol led to a shorter, more efficient labour, and although there was more anxiety related to the CTG, there was no increase in neonatal adverse effects.

In April 2003, D. Garry[17] compared the safety and efficacy of vaginal misoprostol versus dinoprostone vaginal inserts for cervical ripening and labour induction. 200 singleton gestations with an indication for cervical ripening and induction of labor were randomized to receive either 50 ug of misoprostol intravaginally every 3 hour or a 10-mg dinoprostone vaginal insert every 12 hour for a maximum of 24 hour period. The interval from start of induction to vaginal delivery was significantly shorter in the misoprostol group. Women receiving misoprostol were more likely to deliver vaginally both in < 12 hour and < 24 hour. A non-reassuring fetal heart rate tracing was the indication for 71.4 %(20/28) of cesarean deliveries in the misoprostol group compared to 40% (14/35) in the dinoprostone group (p=0.03). Neonatal outcomes remained the same in both groups. They concluded that intravaginal misoprostol and dinoprostone are safe and effective

medications for use in cervical ripening before labour induction. Misoprostol results in a shorter interval from induction to delivery. However, Cesarean delivery for a non-reassuring fetal heart rate tracing was more common in misoprostol group.

In January 2005, Patrick S. Ramsey[18] characterize the frequency and timing of cardiotocographic abnormalities associated with the use of 3 commercially available prostaglandin analogues, misoprostol, dinoprostone gel, and dinoprostone pessary, as labour pre induction agents. 111 women undergoing induction of labour with an unfavorable cervix were randomized to receive either misoprostol 50 ug every 6 hours x 2 doses, dinoprostone pessary 10 mg x 1 dose for every 12 hours intravaginally and dinoprostone gel 0.5mg every 6hrs x 2 doses. Oxytocin induction was initiated per standardized protocol. Cardiotocographic tracings were blindly reviewed, with abnormalities coded using established definitions. They concluded that cardiotocographic abnormalities are more frequent after misoprostol administration compared with the dinoprostone analogues. The early onset and frequent nature of the tracing abnormalities associated with misoprostol raises concern for the potential use of misoprostol for outpatient cervical ripening.

In March 2005, Marjorie Meyer[19] conducted a study to determine whether a single outpatient dose of intravaginal misoprostol (versus intracervical dinoprostone gel) reduces the oxytocin use for induction. Despite the numerous trials examining misoprotol for induction, the efficacy of a single outpatient dose of misoprostol followed by oxytocin induction is unknown. Patients with a term, vertex, singleton pregnancy and a Bishop score of 6 or less were randomly assigned to receive misoprostol (n=42, 25 ug intravaginally) or dinoprostone gel (n=42, 0.5 mg intracervically) the evening before oxytocin induction. Patients were monitored for 3 hours after administration and discharged to home if fetal assessment was

reassuring, for readmission the next morning for oxytocin. They concluded that a single dose of misoprostol adminstered in the outpatient setting significantly decreases oxytocin use, largely due to labour within the ripening period with a favourable outcome.

In 2006, Murthy Bhasker Krishnamurthy[20] compared the safety, efficacy, cost and fetal outcome of misoprostol with that of combination of dinoprostone and oxytocin for induction of labour. 72 women were randomized to receive either misoprostol 25 ug intravaginally every 4 hours for a maximum of 8 doses (study group n=37) or dinoprostone 0.5 mg intracervically 6 hourly for a maximum of 3 doses followed by oxytocin if necessary (control group n=35). Induction delivery interval was significantly shorter in the study group. Failure to progress was the main indication for cesarean section in the control group. Fetal distress was more common in the study group than in the control group but was not significant. Neonatal outcome was comparable in the two groups. The cost of therapy was more effective and highly inexpensive alternative to the combination of dinoprostone and oxytocin for labour induction.

In 2007, Sifakis S[21] conducted randomized study to compare the effectiveness, safety, and side effects of 6 hour vaginal misoprostol versus vaginal prostaglandinE2 (PGE2) for labour induction. 50 microgram of misoprostol was inserted intravaginally in the misoprostol group (204) women), of misoprostol was given intravaginally in the PGE2 group (211 women). In both groups, the dose was repeated every 6 hour for a maximum of three doses, until active labor was achieved. Artificial rupture of membranes(ARM) and oxytocin infusion was used during labour in both groups where it was indicated. The mean interval from the initiation of labour to induction to delivery was shorter for the misoprostol group

than for PGE2 group. In conclusion, the intravaginal administration of 50 ug misoprostol at 6 hour interval (maximum there doses) is comparable in safety and more effective for induction of labor than 3 mg intravaginal PGE2.

In 2007, Lapaire o[22] compared the efficacy of vaginal misoprostol versus dinoprostone for induction of labour in patients with preeclampsia according to the WHO criteria. 98 patients were retrospectively analyzed in this study pattern. A total of 47 patients received 3 mg dinoprostone suppository every 6 hour interval (max. 6 mg/24 hour) whereas 51 patients in the misoprostol group received either 50 ug misoprostol vaginally every 12 hours, or 25 ug every 6 hour (max. 100 ug/24 hour). The probability of delivering within 48 hour was more than three times higher in the misoprostol than in the diinoprostone group. They concluded that misoprostol may have some advantages compared to dinoprostone, including improved efficacy and less cost of the drug, even in cases of preeclampsia.

In 2007, Denguezli W[23] compared the efficacy and safety of intravaginal misoprostol versus dinoprostone cervical gel for cervical ripening and labour induction. 130 patients were randomly assigned to one of the following two treatment groups: (1) intravaginal misoprostol and (2) intracervical dinoprostone gel. A total of 50 ug of misoprostol was placed in the posterior vaginal fornix every 6 hour for a maximum period of 24 hour and 0.5 mg of dinoprostone was adminstrated in the cervix every 6 hour, for a maximum period of 24 hour. The Bishop score was significantly higher in the misoprostol group, 6 hour after the onset . The Caesrean delivery rate for fetal distress was higher in the dinoprostone group. The tachysystole and hyperstimulation syndrome rates were slightly increased in the misoprostol group than in the dinoprostone group without reaching the level of statistical signfications. In conclusion, misoprostol is more effective than dinoprostone gel

application in the cervical ripening and labour induction. There is a tendency for an increase in the rate of tachysystole and hyperstimulation syndrome.

In September 2008, Calder AA[24] compared the efficacy and safety of a 25ug vaginal tablet of miscoprostol with dinoprostone (3-mg vaginal tablet) in cervical ripening and labour induction. 626 Women were randomized to receive either misoprotol (n = 318), initially 25 micrograms (50 micrograms in nulliparous women with Bishop score < or = 4) followed by 25 micrograms after 4 and 8 hours, or dinoprostone (n = 308), initially 3 mg followed by 3 mg after 6 hours. In conclusion, low-dose misoprostol is efficacious in cervical ripening and labour induction and demonstrates an equal fetal and maternal safety profile to Dinoprostone.

In October 2008, Prager M[25] compared the efficacy and safety of induction of labour by vaginal application of dinoprostone or misoprostol or transcervical insertion o a balloon (Bard) catheter. 592 women were randomized to induction of labour using intravaginal dinoprostone (2 mg once every 6 hours) or misoprostol (25 micrograms once every 4 hours) or a transcervical balloon catheter. The shortest mean induction-to-delivery interval was obtained with the catheter (12.9 hours versus 16.8 and 17.3 hours for dinoprostone and misoprostol, respectively). The efficacies of the two prostaglandins are equal. The maternal and neonatal outcomes associated with each of the three procedures were similar. In conclusion, induction of labour with a transcervical balloon catheter is effective and safe and can be recommended as the first choice. The two prostaglandins, dinoprostone and misoprostol, were shown to be equally effective and safe, while misoprostol costs significantly less and is easier to store.

In July 2009, Sebiha Ozkan[26] compared efficacy and safety of vaginal misoprostol (PGE1 analog) with dinoprostone (PGE2 analog) vaginal insert for labor

induction in term pregnancies. 112 women with singleton pregnacies of 37 weeks of gestation, and low Bishop scores underwent labour induction. The subjects were randomized to receive either 50 ug misoprostol intravaginally every 4 hour to a maximum of five doses or a 10 mg dinoprostone vaginal insert for a maximum of 12 hour. Time interval from induction to vaginal delivery was found to be significantly shorter in misoprostol group when compared to dinoprostone group. Vaginal delivery rates within 12 hour were found to be significantly higher with misoprostol induction. In conclusion, using vaginal misoprostol is an effective way of labor induction in term pregnant women with unfavorable cervices, since it is associated with a shorter duration of labour induction and higher rates of vaginal delivery within 12 hour. Misoprostol and dinoprostone are equally safe, since misoprostol did not result in a rise in maternal and neonatal morbidity, namely, tachysystole, uterine hyperstimulation, cesarean section rates.

PHYSIOLOGY OF CERVICAL RIPENING & INDUCTION OF LABOUR

The primary aim of labour induction is to achieve vaginal delivery by initiating the uterine contractions. Thus, the obstetrician is attempting to induce prematurely the two interlinked components of labour, cervical ripening and uterine contractility. Cervical ripening, whether physiological or pharmacological, is the conversion of rigid cervical sphincter meant for maintenance of pregnancy to a soft, compliant and readily dilating structure. The objective of the pharmacological induction of a physiological process is an attempt to mimic the natural process as closely and safely as possible.

Physiology of cervical Ripening

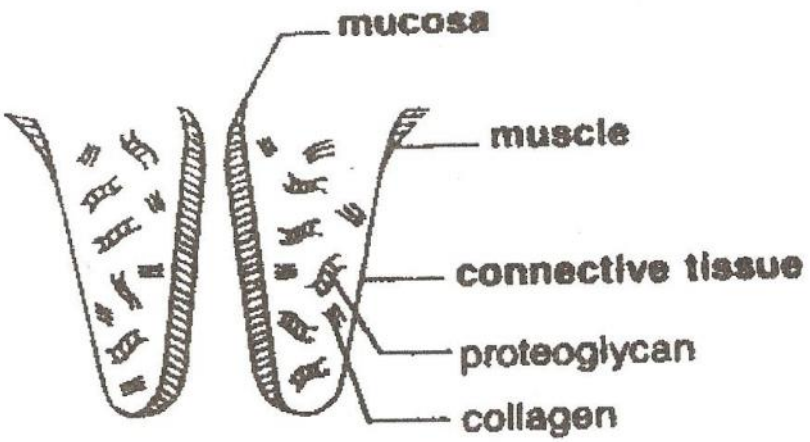
Changes in cervical connective tissue: - predominantly formed element of cervix is the collagen fibrils (type 1), which are bound together into dense bundles conferring on cervix, the rigidity which characterizes its non-pregnant and early pregnant state. The collagen is embedded in a ground substance, comprising large molecular weight proteoglycan complexes containing veriety of glycosaminoglycans (GAG). The most abundant GAG in the cervix are chondroitin and dermatan sulphate. Both are highly negatively charged and hydrophobic. Hence, repel water and are responsible for firmness. Hyaluronic acid binds least strongly with the GAG molecules and will act to destabilize the collagen fibrils, while GAG containing iduronic acid as opposed to glucoronic acid such as dermatan sulphate binds strongly and promotes tissue stability (Obrink, 1973). Changes in the proteoglycan / GAG composition can therefore alter the collagen binding and lead to collagen breakdown.

In non-pregnant state, the cervix consists of around 80% water which increases to around 86% in the late pregnancy. The collagen fibrils and GAG is produced by

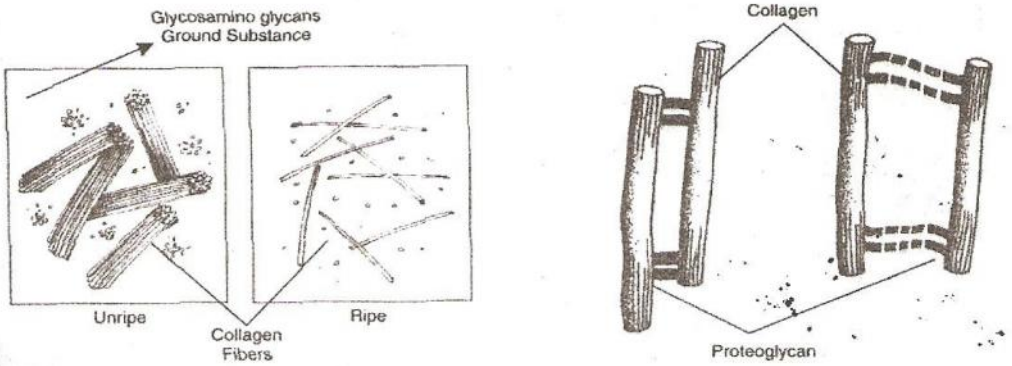
fibroblasts, which constitutes the major cellular component of the cervical tissue. A small amount of elastin is also present in cervix. It has also been shown that the incompetent cervix has absent or reduced elastin fibers Furthermore there is a decrease in elastin during pregnancy. These findings suggest that elastin has an important role in cervical physiology.

The changes associated with cervical ripening include a decrease in collagen fibres within the tissue, a change in GAG content and an increase in water content. Fibroblast activation occurs and local prostaglandin production raises. An inflammatory infiltrate also occurs at term along with this ripening process. stroma becomes highly vascularised and oedematous. While the above changes are widely accepted, the mechanism whereby they occur is still unclear and controversial.

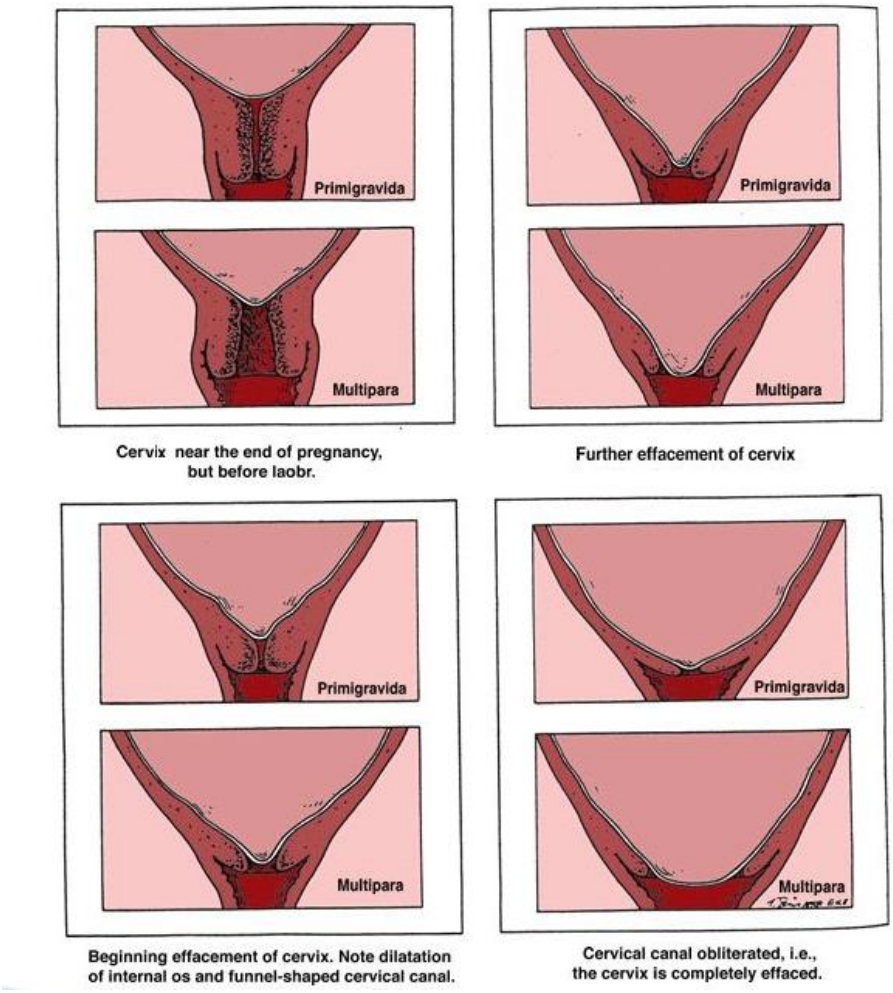
The cervical connective tissue at term show widely scattered and dissociated collagen fibrils with an increase in ground substance when compared to early pregnant and non-pregnant state (Danforth et al).[27]



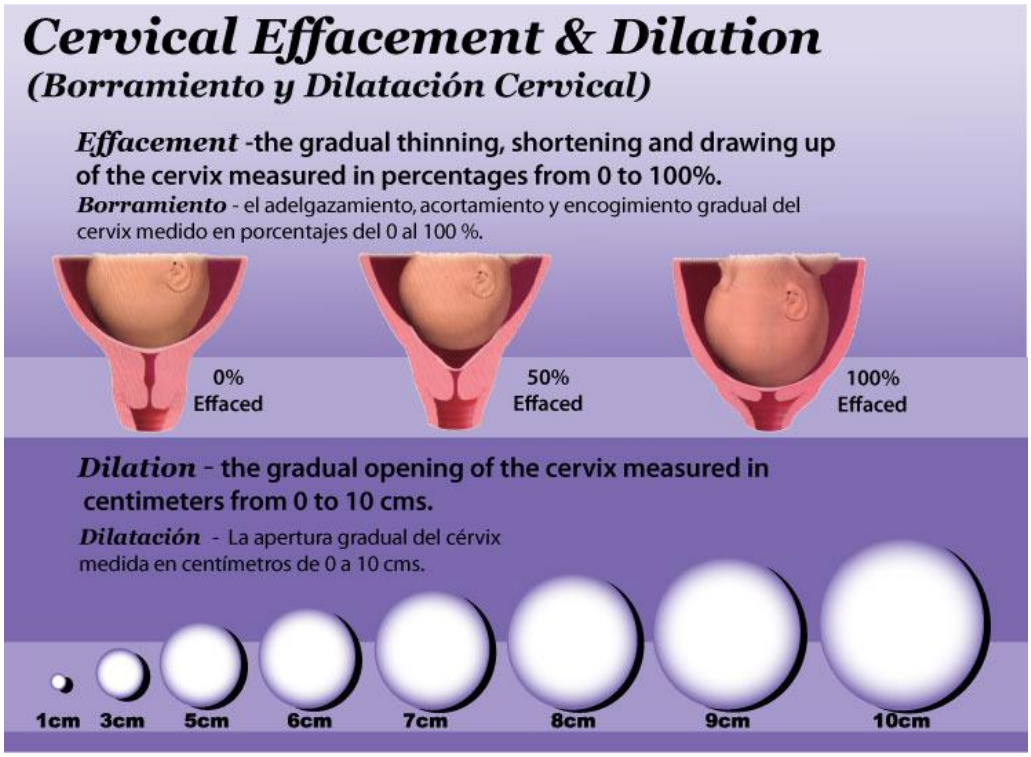
Structural components of the cervix



Physiology of cervical ripening



CERVICAL CHANGES IN PREGNANCY AND LABOUR



CERVICAL EFFACEMENT

A variety of mechanisms have been proposed to explain the reduction in collagen concentration, including increased collagenolysis. Collagen can be brokendown by only two enzymes; (1) Collagenase produced by fibroblasts and leucocytes and (2) Elastase produced by microphages, polymorphs and eosinophils. The collagen fragments by these enzymes can further be broken down by non-specific proteases. The changes of cervical ripening do not appear to be simply due to collagen breakdown as a change in GAG but also change in water content. Overall the total GAG concentration in the cervix probably does not change significantly during labour. However there appears to be relative increase in hyaluronic acid and relative decrease in chondroitin sulphate, compared to non-pregnant cervix.

Control of cervical ripening:-

The above discussion assumes that cervical ripening is an active process due to increased uterine activity. In normal study, cervical ripening occurs even when the cervix is physically isolated from the uterus and ripening can occur in the absence of detectable uterine activity.

Prostaglandins (PG):

Prostaglandins undoubtedly play a major role in the control of cervical ripening . The main prostaglandins produced by cervix are PGE2, PGI2 & to a lesser extent PGF2. There production increases at term, suggesting that they have physiological role in ripening and a further sharp increase accompanies parturition. In addition, amniotic fluid concentration of PGE2 & PGF2 have been shown to correlate directly with cervical ripening in women at term who were not in labour (Calder). Natural and synthetic PGs can ripen the cervix at any stage in pregnancy. There are two possible pathway in which PGs can bring about ripening.

Firstly, they could induce collagen breakdown and later they could alter the collagen binding and tissue hydration by altering the GAG / proteoglycan composition.

A further possible mechanism is that PGE2 induced proteolytic breakdown of proteoglycan complexes which would also cause increase in free hyaluronic acid content.

Oestriol:

Oestriol can stimulate PG production where there has been previous exposure to progesterone and has been used to bring about cervical ripening in the clinical situation. This effect may be due at least in part, to induction of PG synthesis. In addition, oestradiol has been linked to an increase in collagenase activity (Mochizuki & Tojo).[28]

Progesterone:

Progesterone appears to have an inhibitory effect on cervical ripening and parturition in animal studies where, a fall in progesterone at term results in ripening and initiation of labour and has anti inflammatory effect. This possibly is supported by the ripening effect of anti-progestin on cervix prior to termination of pregnancy.

Methods of ripening of cervix prior to induction of labour

The continuation of pregnancy requires that the cervix remains closed and that uterus is quiet and not contracting. Both these conditions need to be reversed to initiate labour. The ways in which this is achieved are unknown but there is evidence that suggests the fetus itself plays an integral part. The cervix, which contains little smooth muscle and is predominantly connective tissue with collagen as its main component, must undergo a process called ripening, where it becomes soft and pliable. This allows its shape to change from being long and closed to being short,

thinned (effaced) and opening (dilating). In parallel with this, the uterus, with predominantly smooth muscle cells, must begin to respond to the stimuli which cause these cells to contract in the waves that characterize labour.

In recent years, it has been recognized that both the components of labour (cervical and uterine changes) involve prostaglandins, inflammatory mediators and other agents. Most methods of induction seek to exploit these components in order to initiate labour.

A review of range of methods that have historically been applied to induction of labour reveals that they can be classified into three categories:-

1. Pharmacological/drug based methods.
2. Non-Pharmacological methods.
3. Surgical methods.

Pharmacological-based methods:

Prostaglandins (PGE2):

Prostaglandins are capable of stimulating uterine contractions resulting in labour. Prostaglandins can be administered by various routes: vaginal, oral, intravenous, extra-amniotic and intra cervical.

Vaginal PGE2 [29,30,31] :-

The vaginal preparations of PGE2 are used in the form of tablets, pessaries, suppositories. In women with an unfavourable cervix, all regimens of vaginal PGE2 are significantly associated with uterine hyperstimulation with fetal heart rate (FHR) changes, improved cervical status within 24 hours, reduction in the need for oxytocin augmentation and reduced incidence of meconium-stained liquor. In women with a favourable cervix, all regimens of vaginal PGE2 are more effective than placebo. No treatment in achieving vaginal birth within 24 hours. The drug cost of vaginal PGE2 tablets, gel and slow-release pessaries are similar. Vaginal PGE2 is the preferred method of induction of labour, unless there are specific clinical reasons for not using it (in particular, the risk of uterine hyperstimulation). It should be administered as a gel, tablet or controlled release pessary.

The recommended regimens are:

- One cycle of vaginal PGE2 tablets or gel : one dose, followed by a second dose after 6 hours if labour is not established (up to a maximum of two doses)
- One cycle of vaginal PGE2 controlled release pessary : One dose over 24 hours When offering PGE2 for induction of labour, healthcare professionals should inform women about the associated risks of uterine hyperstimulation.

Oral PGE2:-

Evidence suggested that, for women with an unfavourable cervix, oral PGE2 is associated with a reduction in caesarean birth rate when compared with placebo. However, oral PGE2 is no more effective as a cervical priming method than vaginal/intracervical PGE2, or oral/intravenous oxytocin. For women with a favourable cervix, oral PGE2 achieved similar maternal and fetal outcomes to oral oxytocin or oral oxytocin plus aminotomy. Gastrointestinal side effects including vomiting were frequently reported by women treated with oral PGE2

Extra-amniotic PGE2[32]:-

Evidence suggested that, for women with an unfavourable cervix, extra amniotic prostaglandins lessen the requirement for oxytocin augmentation when compared with placebo. There are insufficient data to determine its effectiveness when compared with intravenous oxytocin and mechanical methods. For women with a favourable cervix, extra-amniotic PGE2 is comparable to vaginal PGE2 in achieving vaginal birth within 24 hours. Extra-amniotic PGE2 is no more effective than vaginal PGE2 in achieving vaginal birth within 24 hours. Extra-mniotic PGE2 is no more effective than vaginal PGE2 and is a more invasive procedure. Extra-amniotic PGE2 should not be used for induction of labour.

Intracervical PGE2[33] :-

For women with an unfavorable cervix, intracervical PGE2 is less effective than vaginal PGE2 and confers no benefit. For women with a favorable cervix, it achieves similar maternal outcomes as vaginal PGE2. Intracervical adminstration is invasive.

Intravenous oxytocin alone[34]:-

Oxytocin has been used alone, in combination with amniotomy, or following cervical ripening with other pharmacological or non-phamacological methods.

However, it is important to distinguish its role as an induction of labour. In women with an unfavorable cervix and intact membranes, the use of intravenous oxytocin alone when compared with vaginal PGE2 as an inducing agent results in fewer vaginal births within 24 hours, a lower Bishop score at 24 hours and more caesarean births. In women with a favorable cervix, the use of intravenous oxytocin alone when compared with vaginal PGE2 as an inducing agent results in fewer vaginal births within 24 hours. Inravenous oxytocin alone should not be used for induction of labour.

Amniotomy with intravenous oxytocin[35] :-

In women with a favorable cervix, one trial reported that the use of intravenous oxytocin with amniotomy was associated with postprtum haemorrhage and reduced women's satisfaction. This is likely to apply to women with unfavourable cervix as well. In addition, as this method required intravenous access and continuous monitoring, it is necessarily more invasive than the use of vaginal PGE2 and will limit women's mobility during induction. Amniotomy with oxytocin should not be used as a primary method of induction of labour unless there are specific contraindications to the use of vaginal PGE2.

MISOPROSTOL :-

Misoprostol is a synthetic prostaglandin that can be given orally, vaginally or sublingually. It is effective in causing uterine contractions. Oral misoprostol usually comes in tablets of 25, 50, 100, and 200 micrograms.

- **Oral misoprostol[35,36,37.38.39,40]:-**

Evidence suggested that, irrespective of cervical status, oral misoprostol is more effective than placebo as an labour induction agent. There is no significant difference in maternal and fetal outcomes between oral misoprostol (200 micrograms) and

intracervical PGE2. The use of oral misoprostol (100 micrograms) is more likely than oxytocin to be associated with meconium-stained liquor. Oral misoprostol 50 micrograms or 100 micrograms achieve similar maternal and fetal outcomes. Oral misoprostol (50-100 micrograms) is less likely than vaginal PGE2 to result in caesarean birth (borderline significance). Oral misoprostol has similar efficacy to vaginal PGE2 gel in terms of vaginal birth within 24 hours.

- **Vaginal misoprostol :-**

Evidence suggested that, for women with an unfavourable cervix, vaginal misoprostol is more effective than placebo as labour induction agent. Vaginal misoprostol (50-100 micrograms) is more likely than vaginal PGE2 to produce a favourable cervix within 24 hours, achieve birth within 24 hours, and cause uterine hyperstimulation. Vaginal misoprostol (50-100 micrograms) is more likely than intravenous oxytocin to cause uterine hyperstimulation without FHR changes. Vaginal misoprostol at lower dose (minimum 25 micrograms) was more likely than high dose (maximum 50 micrograms) to cause uterine hyperstimulation with and without FHR changes. Vaginal misoprostol (50 ugm) is less likely than vaginal misoprostol tablet to cause uterine hyperstimulation with FHR changes, but more likely to need oxytocin augmentation and epidural analgesia. Vaginal misoprostol is more likely than Isosorbide Mononitrate to achieve earlier birth and don't need oxytocin augmentation. Tachysystole and uterine hyperstimulation are less likely in women given vaginal Isosorbide Mononitrate. There were more reports of headaches, nausea and dizziness in the Isosorbide Mononitrate group.

- **Buccal/Sublingual misoprostol :-**

For women with an unfavourable cervix, there were insufficient data to determine the effectiveness of buccal/sublingual misoprostol as compared with oral

and vaginal misoprostol. Compared with PGE2, any misoprostol is more effective in achieving vaginal birth within 24 hours and lessening the need for oxytocin use, but any misoprostol is associated with higher risks of hyperstimulation and increased meconium staining. Caesarean birth rates were similar between the two interventions.

Mifepristone[41]:-

Mifepristone, also known as RU 486, is an antiprogestin and has been developed to antagonise the action of progesterone. Mifepristone now has an established role in the termination of pregnancy, in combination with prostaglandins, during the first and second trimester. There is concern from the latest evidence that mifepristone may be associated with ischaemic changes in the fetal kidney when labour was induced using mifepristone at between 16 and 28 weeks of gestation. The efficacy and safety of mifepristone as an induction agents needs to be established. Mifepristone should only be offered as a method of induction of labour to women with intrauterine fetal death.

Hyaluronidase[42]:-

The level of hyaluronic acid increases markedly after the onset of labour. Cervical injection of hyaluronidase was postulated to increase cervical ripening. Evidence suggested although intracervical hyaluronidase may be effective in improving cervical ripening and reducing caesarean birth rates,it is an invasive procedure that women may find unacceptable when alternative available methods such as vaginal PGE2 are less invasive. Hyaluronidase should not be used for induction of labour.

Corticosteroids[41]:-

Corticosteroids are postulated to have a promoting effect in induction of labour but their role in the process of labour is not well understood. The available evidence relating to the effects of corticosteroids for cervical priming and induction of labour is limited. Corticosteroids should not be used for induction of labour.

Oestrogens[43]:-

The increase in the serum oestrogen-to-progesterone ratio that occurs before the onset of labour is believed to activate prostaglandin production, which in turn stimulates cervical ripening. Oestrogens and placebo achieved similar maternal and fetal outcome there was insufficient evidence to determine the effectiveness of oestrogen for cervical ripening. Oestrogen should not be used for induction of labour.

Vaginal nitric oxide donors:-

Nitric oxide is considered a fundamental mediator of cervical ripening without causing uterine contractions or adverse effects on the mother and fetus. Vaginal glyceryl trinitrate and nitric oxide donors have not been shown to be of any particular benefit when compared with vaginal PGE2 as labour induction agents, although they seem to be associated with less uterine hyperstimulation. However, there are significant side effects such as headaches and palpitation associated with its use. Vaginal nitric oxide donors should not be used for induction of labour.

Non-pharmacological methods:

Membrane sweeping[44,45,46,47]:-

Stripping/sweeping of the membranes was used as a method for inducing labour at least as early as 1810. Increased local production of prostaglandins following vaginal examination for membrane sweeping provides an explanation for the effect

of this procedure on pregnancy duration. Vaginal examination allows an assessment of the condition of the cervix which informs clinical decision making.

Carried out in late pregnancy, when consideration is being given to induction, it offers the opportunity to undertake membrane sweeping. If the women are on the threshold of spontaneous labour, a membrane sweep may be all that is required to initiate it, thus reducing the need for formal induction of labour. The procedure entails passage of the examining finger through the cervix so that it can be rotated against the wall of the uterus beyondthe internal cervical os, thereby stripping the chorion away from the decidua (the deciduas is the richest source of PGE2 within the uterus). Clearly if the cervix does not admit a finger it may not be possible to strip the membranes but in such cases massaging around the cervix in the vaginal fornices may achieve a similar effect.

Compared with no sweeping, sweeping reduces the need for formal induction of labour. Additional membrane sweeping may be beneficial. Membrane sweeping is an important and integral part of preventing prolonged pregnancy, and should be scheduled to be discussed with the woman at her routine antenatal visit. Prior to formal induction of labour, women should be offered a vaginal examination for membrane sweeping.

At the 40 and 41 week antenatal visits, nulliparous women should be offered a vaginal examination for membrane sweeping.

At the 41 week antenatal visit, parous women should be offered a vaginal examination for membrane sweeping. When a vaginal examination is carried out to assess the cervix, the opportunity should be taken to offer the woman a membrane sweep. Additional membrane sweeping may be offered if labour does not start spontaneously.

Breast stimulation:-

It is known that breast stimulation results in the production of endogenous oxytocin in pregnant and non-pregnant women, causing uterine contractions. There is evidence that breast stimulation may be effective as a method of induction. However, interpretation of the results was problematic owing to the poor quality of the studies reviewed and the heterogeneous populations, including high-risk women from developing countries. There is inconsistency in the timing, methods and frequency of breast stimulation described in these studies, making guidance on this method difficult.

Surgical methods:

Amniotomy:-

Amniotomy is the deliberate artificial rupture of the membranes(ARM), used for induction of labour. The procedure is only possible if the membranes are physically accessible. Although there is limited evidence for amniotomy when the cervix is unfavorable, the practice is not recommended because of the invasiveness of the procedure and the potential risks of infection when amniotomy is performed at the start of labour. In the case of an unfavorable cervix, although amniotomy appears to be effective it is associated with more frequent need for oxytocin augmentation when compared with vaginal PGE2. Amniotomy alone should not be used as a primary method of induction of labour unless there are specific clinical reasons for not using vaginal PGE2, in particular the risk of uterine hyperstimulation.

Mechanical methods:-

Mechanical methods used for induction of labour include various types of balloon catheters or laminaria tents introduced into the cervical canal or into the extra-amniotic space. In unfavourable cervix, when compared with all prostaglandins

given by any route, mechanical methods do not improve the rate of vaginal birth within 24 hours nor do they reduce the caesarean birth rate. They may reduce the incidence of uterine hyperstimulation but increase the risk of neonatal infection. The value of mechanical methods of inducing labour in women with an unfavourable cervix is doubtful. Since these methods are associated with less hypertonicity, they may reduce the risk of uterine rupture in the presence of a previous caesarean section scar. For women with a favourable cervix, there was no evidence to determine the effects of mechanical methods as an induction agent. Mechanical procedures (balloon catheters and laminaria tents) should not be used routinely for induction of labour.

PROSTAGLANDINS

Prostaglandins were first discovered and isolated from human semen in the 1930s by Ulf von Euler of Sweden. Thinking they had come from the prostate gland, he named them prostaglandins. It has since been determined that they exist and are synthesized in virtually every cell of the body.

Prostaglandins are like hormones in that they act as chemical messengers, but do not move to other sites, but work right within the cells where they are synthesized.

Chemistry:

Prostaglandins are unsaturated carboxylic acids, consisting of a 20 carbon skeleton that also contains a five member ring. They are biochemically synthesized from the fatty acid, arachidonic acid.

The unique shape of the arachidonic acid caused by a series of cis double bonds helps to put it into position to make the five member ring.

Prostaglandins are unsaturated carboxylic acids, consisting of a 20 carbon skeleton that also contains a five member ring and are based upon the fatty acid, arachidonic acid. There are a variety of structures with one, two, or three double bonds. On the five member ring, there may also be double bonds, a ketone, or alcohol groups.

Classification:

Prostaglandins are classified as:

PG series A to I – depending upon the ring structure and substitute on it. Subscript 1, 2, 3 – indicate number of double bonds on the side chains.

Type of PGs in relation to source:

Sl No.	Female Reproductive Tissues	Type
1	Maternal blood during pregnancy, labour & abortion	E2, F2α
2	Amniotic fluid in pregnancy & labour	F2α
3	Umbilical blood	E, F
4	Fallopian tube	F2α
5	Menstrual blood and endometrium	F
Male Reproductive tissues		
1	Seminal fluids	E1, E2, E3, 19-OH E1, E2, F1, A1, A2

Prostaglandins are also classified as: -Natural.

-Semi-synthetic.

Natural PGs are PGF2a and PGE2. These are most commonly used in pregnancy because they do not have adverse effect on pregnancy & fetus. While semi-synthetic PGs are 15 methyl PGE2 analogues, 16 phenol PGE2 analogue are used after delivery or termination of pregnancy becasue their adverse effect on pregnancy & fetus are not yet proved.

PHARMACOLOGICAL ACTIONS & CLINICAL SIGNIFICANCE

Metabolic effects:

Various endocrine glands e.g. Thyroid, adrenal, ovary, parathyroid glands augment secretions of their hormones by the action of PG on Adenylcyclase.

Actions of prostaglandins on Platelets:

PGE1 elevates platelets AMP levels & inhibits aggregation of platelets. PGE2 inhibits aggregation of platelets. PGE2 has biphasic effect, promotes aggregation at low (less than 1 micro-mol) concentration but inhibits it at high concentration. Thromboxane- A2 promotes aggregation of platelets. PGE2 elevates platelets AMP levels & inhibits aggregation of platelets.

Vascular effects:

PGE1, PGE2 and $PGF_{2\alpha}$ has vasodilator effect with little effect on BP.

On Smooth muscles:

$PGF_{2\alpha}$ and PGD_2 and TXA_2 are potent broncho-constrictors.

PGE_2 is a broncho-dilator.

Immune Effects: Inhibits T & B cells of immune system.

GI Effects:

PGE2 inhibits gastric secretions but stimulates pancreatic & intestinal secretions thus increases intestinal motility.

Nervous System:

PGs have sedative & tranquilizing action by releasing epinephrine from sympathetic nerve endings. They can also cause marked rise in temperature.

Uterus:

PGs cause softening of cervix and contraction of gravid uterus where as, they cause relaxation of non-gravid uterus in vitro.

Reproductive effects:

The physiological role of PGs in female reproductive process include sperm transport, ovulation, leuteolysis, menstruation, spontaneous abortion, labour & closure of umbilical blood vessels after birth.

Over a period of two decades the PGE & PGF, their synthetic analogues & PG-synthetase inhibitors have undergone numerous clinical trials in the following areas in Obstetrics & Gynaecology.

1. Menstrual Regulation
2. Termination of first trimester pregnancy
3. Pre-evacuation cervical dilation in first trimester pregnancy
4. Termination of second trimester pregnancy
5. Termination of abnormal pregnancy
6. Pre-induction cervical ripening
7. Induction & augmentation of labour
8. Management of third stage of labour (P.P.H)
9. Use of PG-synthetase inhibitor (Dysmenorrhoea, Pre-term labour, Polyhydramnios, D.U.B., etc.)

Adverse Actions:

These are not troublesome with smaller doses, but could be severe with large doses. They include nausea, vomiting, diarrhoea, headache, chills, fever & vasodilation.

Preparations:

a) Natural:

1. Tab. 0.5 mg by mouth for induction of labour Maximum dose 1.5 mg.
2. Tab. 3 mg by vaginal route for induction of labour.
3. I.V. solution 1 mg/ml and 10 mg/ml dilution.
4. Extra-amniotic solution 10 mg/ml with diluents.
5. Intracervical gel (CERVIPRIME GEL) 0.5 mg for ripening of cervix.
6. Dinoprost (PGF2 alfa) is available as solution containing 5 mg of salt per milliliter. It is used intra-amniotically to induce abortion. It is used less often now than in past.

b) Semi-synthetic:

8. Carboprost- (Prostin, Prostodin) is 15-methyl-PGF2 alfa analogue with longer duration of action. 250 microgram/ml for deep intramuscular use. It can be used for abortion with an interval time of 1.5 to 3.5 hours up to maximum dose of 12mg. For PPH, it can be uses in the intervals of 15 min to 90 mins depending on the need up to the dose of 2mg (8 doses).
9. Gemeprost (Cervagem) - 1 mg vaginal pessary.
10. 16-Phenoxy-PGE2 (Sulprostone) is available for I.V/I.M and extra amniotic use.

c) Synthetic : Misoprostol (PGE1)

Misoprostol is a synthetic prostaglandin that can be given orally,

vaginally or sublingually. It is effective in causing uterine contractions. It can be used as:

1. Oral misoprostol (25-200 micrograms) for induction of labour.
2. Vaginal misoprostol (25-100 micrograms) for induction of labour.
3. Per rectal imisoprostol (800-1000 micrograms) for treatment of Postpartum haemorrhage.

INDUCTION OF LABOUR

Williams stated that, “Induction implies stimulation of uterine contractions before the spontaneous onset of labor, with or without ruptured membranes”[48].

Renu Mishra stated that “Induced labour is the one in which pregnancy is terminated artificially any time after fetal viability is attained by a method that aims to secure delivery.”[49]

Duru shah and Sudeshna Ray, mentioned that, “Iinduction of labour is an intervention intended to artificially initiate uterine contractions resulting in progressive effacement and dilatation of the cervix. This should ideally result in the birth of the baby through vaginal route (RCOG 2001).”[50]

Cervical ripening refers to a prelabour phase when cervix changes its characteristics such as consistency, position, effacement and dilatation,. Induction refers primarily to an attempt to produce regular uterine contractions along with cervical changes to go into active phase of labour. In clinical practice, however the two often have many overlapping features and the difference becomes relatively unimportant compared with the ultimate outcome of successful vaginal delivery without fetal or maternal compromise.

INDICATIONS FOR INDUCTION (ACOG Practice Bulletin 107, August 2009).

ACOG states that there are number of health conditions that warrant induction of labour. Some of the indications for induction include (but not limited to):

1. Pregnancy Induced Hypertension
2. Premature Rupture of membranes
3. Abruption placentae
4. Chorioamnionitis
5. Supected
    - Absence of fetal well – being
    - IUGR
    - Postterm pregnancy
    - Isoimmunization
6. Maternal medical problems
    - Diabetes mellitus
    - Renal disease
    - Chronic pulmonary disease
    - Cardiac disease
7. Fetal demise

CONTRAINDICATIONS FOR INDUCTION (ACOG Practice Bulletin 107, August 2009).

Some of the contraindications include (but not limited to) are:

1. Major degree of cephalo – pelvic disproportion and contracted pelvis.
2. Placenta praevia or vasa praevia.
3. Prior classical caesarean section.
4. Cord prolapsed
5. Prior myomectomy or uterine unification surgery.
6. Active gential herpes infection.
7. Pregnancy following repair of Vesicovaginal fistula.
8. Malpresentation.
9. Invasive cervical carcinoma.

RISKS OF INDUCTION:

A. MATERNAL

1. Psychological upset
2. Need for emergency caesarean delivery
    - Due to fetal distress
    - Due to failed induction
3. Placental abruption
4. Precipitate delivery.
5. Abnormal uterine action.
    - Uterine hyper tonicity
    - Incoordinate uterine action
    - Uterine rupture
6. Atony of uterus due to paralysis of myometrial fibrils due to hyper stimulation syndrome.
7. Water intoxication and electrolyte imbalance.
8. infection
9. Amniotic fluid embolism

B. FETAL

1. Iatrogenic prematurity
2. Fetal hypoxias due to
    - Uterine hypertonus
    - Placental site retraction
    - Cord complications
    - Abruptio placentae
3. Neonatal jaundice in association with oxytocin.

FACTORS TO BE CONSIDERED WHEN ELECTING TO INDUCE ARE,

1. Patients informed consent
2. Estimation of fetal pulmonary maturity
3. Estimation of fetal maturity and gestational age
4. Pelvic adequacy
5. Readiness of cervix-by modified Bishop's Scoring system.
6. The presumed ability of the fetus to tolerate the labour
7. Stability of maternal condition.
8. Uterine intergrity.

MATERIAL AND METHODS

Source of Data:

- 150 Patients admitted to labour ward of OBG Dept of SHRI B M Patil medical College and Research Hospital with an indication for induction of labour from October 2014 to june 2016.
- It is a prospective cross- sectional comparative study.

- **Indications for Induction in Our Study**
    - Mild pre eclampsia.
    - Severe pre eclampsia
    - Post dated pregnancy.
    - Mild polyhydramnios.
    - Mild oligohydramnios.
    - Gestational Diabetes Mellitus.
    - Chronic hypertension.
    - Mild IUGR.
    - Chorioamnionitis.

Inclusion Criteria:

- Indication for labour induction
- Singleton pregnancy.
- Gestational age more than 28 weeks.
- Vertex presentation.
- Bishop score ≤ 5.

Exclusion Criteria:

- Previous L.S.C.S or any uterine surgery
- Mal presentation
- Contracted pelvis or cephalopelvic disproportion.
- Antepartum haemorrhage.
- Unsatisfactory CTG.
- Severe IUGR.
- Active genital herpes.
- Pelvic tumors
- Bronchial asthma.

Method of Induction:

- After informed consent had been obtained, the patients selected for the study were evaluated initially by modified Bishop's score and admission test for fetal well being. Patients with a modified bishops score ≤5 and a positive admission test were induced.
- 75 patient with an indication for labour induction received with 50µ g of intravaginal misoprostol and repeated for a maximum of 3 doses every 6 hours as needed.
- 75 patients with an indication for induction of labour received 0.5 mg intracervical dinoprostone gel and repeated for a maximum of 3 doses every 6 hours as needed.
- After drug insertion, patients were monitored for signs of labour, maternal vital signs, fetal heart rate and progress of labour. The fetal heart rate was monitored by either intermittent auscultation or continuous fetal heart rate monitoring. A partogram was strictly maintained in all patients.

- Oxytocin was started depending on the modified Bishop's score and in the absence of adequate uterine contractions after 6 hrs of the last dose, or for augmentation of labour in case of an arrest of dilation. Oxytocin was started at the dose of 5 units in 500ml RL in Primigravida and 2.5 units in 500ml RL in multigravida and titrated accordingly.
- Membranes were ruptured, when the cervix was completely effaced with a cervical dilatation of more than 3 cms or at onset of active stage of labour.
- The data collection included indication for indication, maternal age, parity, gestational age on entry into the study, modified Bishop's Score at time induction, induction – delivery interval, oxytocin augmentation, type of delivery, Apgar score of the baby, maternal and neonatal complications.
- The results observed were subjected to statistical analysis.

DEFINITIONS AND CRITERIA:

1. Induction was considered as 'failed induction' if contractions did not start or if bishop score did not increase at end of 24 hours.
2. Tachysystole was defined as more than 5 uterine contractions per 10 minutes without fetal heart rate (FHR) changes, for 2 consecutive 10 minute periods.
3. Hyperstimulation was defined as exaggerated uterine response accompanied by fetal heart rate decelerations or tachycardia.

Modified Bishop's Score.

	0	1	2	3
Dilatation(cms)	**Closed**	**1-2**	**2-4**	**>4**
Length (cms)	**More than 4**	**2-4**	**1-2**	**<1**
Consistency	**Firm**	**Medium**	**Soft**	**------**
Position	**Posterior**	**Midline**	**Anterior**	**------**
Level of head	**-3**	**-2**	**-1; 0**	**+1,+2,**

Total Score -13

Favorable Score- 6-13.

Unfavorable score- 0-5.

Data collected:

- Maternal age
- Gestational age
- Indication for induction
- Modified Bishop score at induction
- Partograph
- Oxytocin augmentation
- Type of delivery
- Induction delivery interval
- APGAR score of baby
- Maternal and fetal complications
- Meconium stained liquor.

Procedure for Dinoprostone gel instillation:

Patient was taken on the edge of table

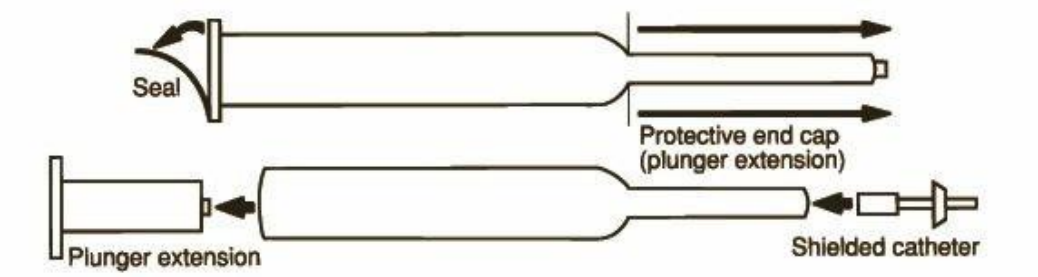
1. Cleaning ,painting &draping were done.
2. Cervix was visualized with Sims speculum.
3. Anterior lip of cervix was caught with the sponge holder.
4. Preloaded gel was instilled below internal os.
5. Patient was asked not to get up from the bed for a period of 30 mins.
6. Instillation of gel was repeated after 6 hours as required upto 3 doses.

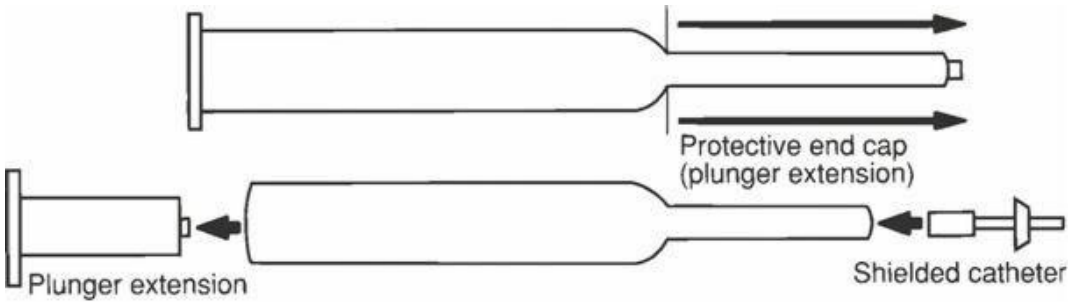
Procedure for Misoprostol Tablet insertion:

1. Patient was taken on the edge of table
2. Cleaning, painting & draping were done.
3. Tablet. Misoprostol 50 microgram was made wet with sterile distilled water or NS and then inserted in the posterior fornix.
4. Patient was asked not to get up from the bed for a period of 30 mins**.**
5. Misoprostol 50mcg was repeated after 6 hours as required up to 3 doses.

Misoprostol 50 micro gram Tablet

Dinoprostone





OBSERVATIONS AND RESULTS

Total number of patients studied was 150. 75 patients were induced with 50 µgms intravaginal Misoprostol tablet and repeated every 6th hourly up to 3 doses. And the other 75 patients induced with 0.5mg intracervical Dinoprostone gel and repeated every 6th hourly up to 3 doses.

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square (χ^2)/Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables was tested with the unpaired t-test. If the p-value was < 0.05, then the results will be considered to be significant. Data were analyzed using SPSS software v.23.0.

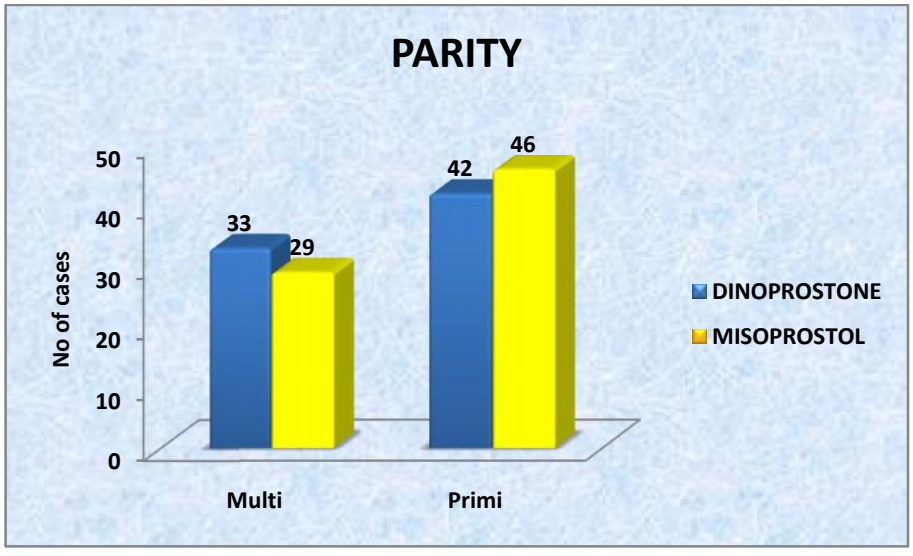
The following observations were made:-

Table 1: Distribution of cases by Parity between study groups

PARITY	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
Multi	33	44.0%	29	38.7%	0.507
Primi	42	56.0%	46	61.3%	
Total	75	100.0%	75	100.0%	

Parity was compared in both groups and found to be almost similar, with no statistical significance (P=0.507). Primigravida formed the largest group in the study being 56% and 61.3% in dinoprostone and misoprostol group respectively. Multigravida in dinoprostone and misoprostol groups were 44% and 38.7% respectively.

Graph 1: Distribution of cases by Parity between study groups



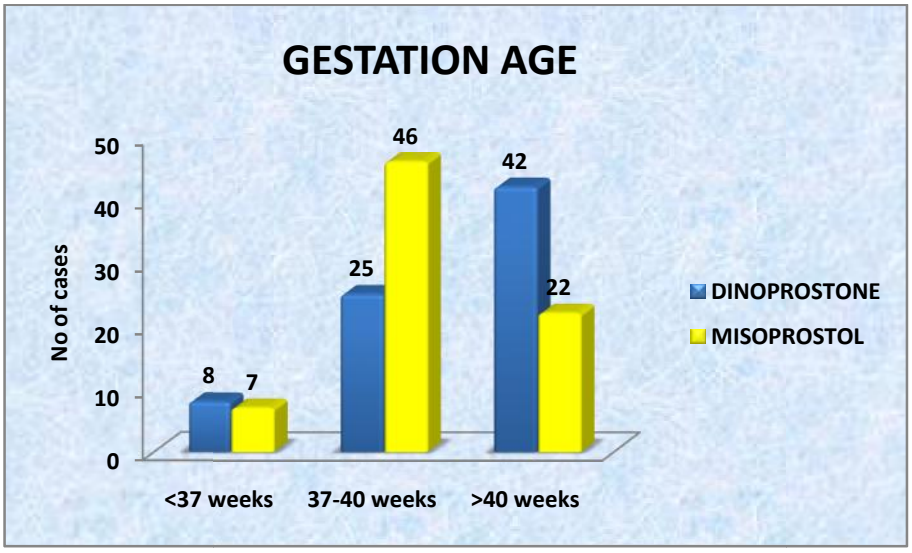
The above graph represents the tabular data showing distribution of cases by parity between study groups.

Table 2: Distribution of cases by Gestation Age between study groups

GESTATION AGE	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
<37 weeks	8	10.7%	7	9.3%	
37-40 weeks	25	33.3%	46	61.3%	0.002
>40 weeks	42	56.0%	22	29.3%	(Sig)
Total	75	100.0%	75	100.0%	

From the above table when gestational age was compared it was seen that there was more number of patients between 37 to 40 weeks (61.3%) in misoprostol group and more number of patients with gestational age more than 40 weeks (56.0%) in Dinoprostone group. This was statistically significant (P<0.002).

Graph 2: Distribution of cases by Gestation Age between study groups



The above graph represents distribution of cases by gestational age between study groups.

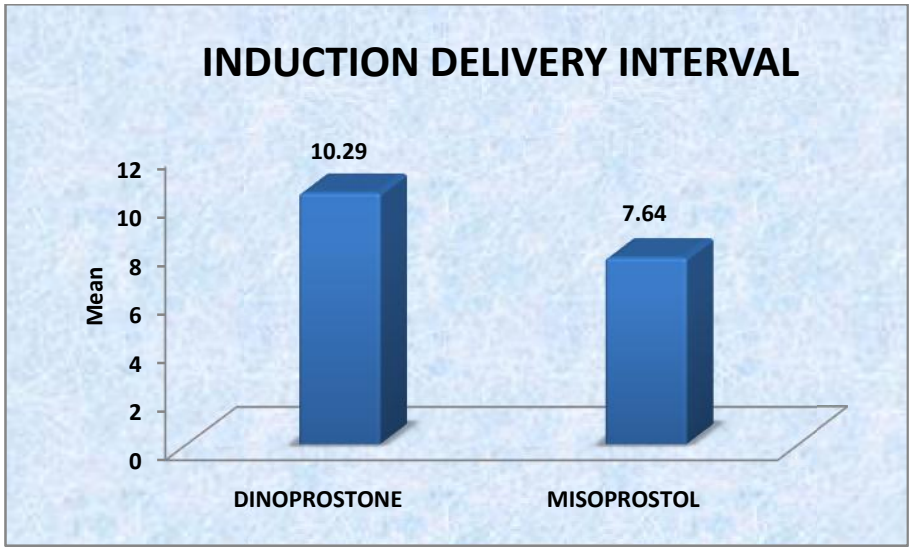
Table 3: Mean Induction Delivery Interval between study groups

MEAN INDUCTION DELIVERY INTERVAL	DINOPROSTONE	MISOPROSTOL	p value
Mean±SD	10.29±7.19	7.64±5.75	0.014 (Sig)

P<0.05 significant

The mean induction delivery interval in dinoprostone is 10.29 ± 7.19 hours. The mean induction delivery interval in misoprostol is 7.64± 5.75 hours. Mean induction delivery interval is shorter in Misoprostol group and is statistically significant.

Graph 3: Mean Induction Delivery Interval between study groups



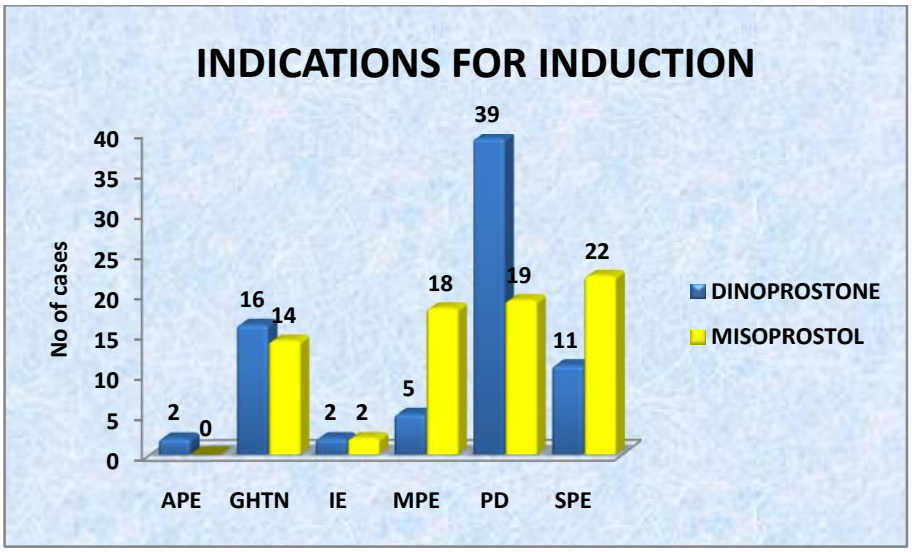
The above graph represents the induction to delivery interval between Dinoprostone and Misoprostol groups. It was shorter in Misoprostol group.

Table 4: Distribution of cases by Indications for Induction between study groups

INDICATIONS FOR INDUCTION	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
APE	2	2.7%	0	0.0%	0.001 (Sig)
GHTN	16	21.3%	14	18.7%	
IE	2	2.7%	2	2.7%	
MPE	5	6.7%	18	24.0%	
PD	39	52.0%	19	25.3%	
SPE	11	14.7%	22	29.3%	
Total	75	100.0%	75	100.0%	

The largest group for induction in Dinoprostone group was Post dated pregnancy (52.0%) and in Misoprostol group was severe preeclampsia (29.3%). There was statistical significance as the p value is 0.001.

Graph 4: Distribution of cases by Indications for Induction between study groups



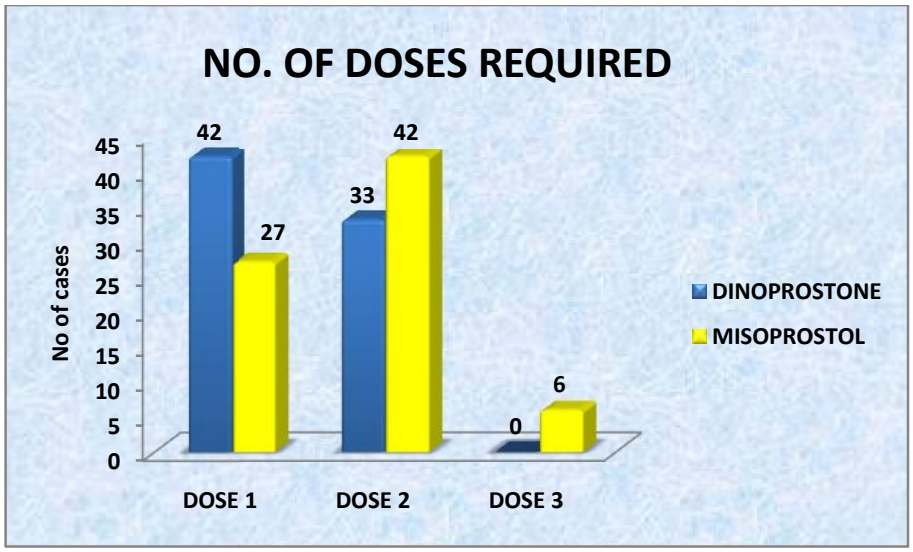
The above graph represents the distribution of cases by indication between study groups. Post dated pregnancy was significantly high as an indication for induction in Dinoprostone group and severe preeclampsia in Misoprostol group.

Table 5 : Distribution of cases by No. of doses required

NO. OF DOSES REQUIRED	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
DOSE 1	42	56.0%	27	36.0%	0.025 (Sig)
DOSE 2	33	44.0%	42	56.0%	
DOSE 3	0	0.0%	6	8.0%	
Total	75	100.0%	75	100.0%	

The above table represents the distribution of cases according to total number of doses required. In Dinoprostone group maximum number doses required is 2 and where as in Misoprostol group it is 3. The difference is statistically significant (p value = 0.025).

Graph 5: Distribution of cases by No. of doses required between study groups



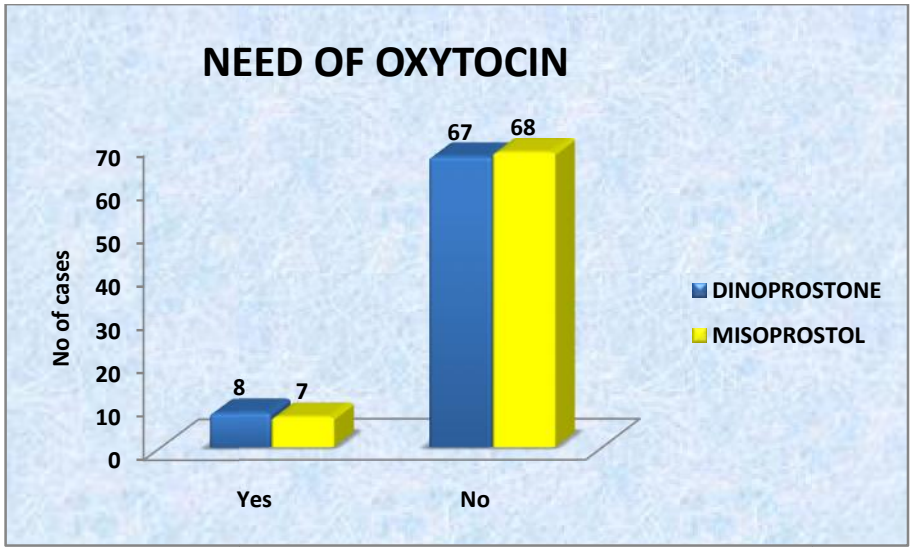
The above graph represents the total number of doses required in Dinoprostone and Misoprostol group.

Table 6: Distribution of cases by Need of Oxytocin between study groups

NEED OF OXYTOCIN	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
Yes	8	10.7%	7	9.3%	0.785
No	67	89.3%	68	90.7%	
Total	75	100.0%	75	100.0%	

Need for oxytocin augumentation was almost equal in both the groups and statistically not significant. Need for oxytocin was 10.7% and 9.3% in Dinoprostone and Misoprostol group respectively.

Graph 6: Distribution of cases by Need of Oxytocin between study groups



P> 0.05, Not significant. (NS)

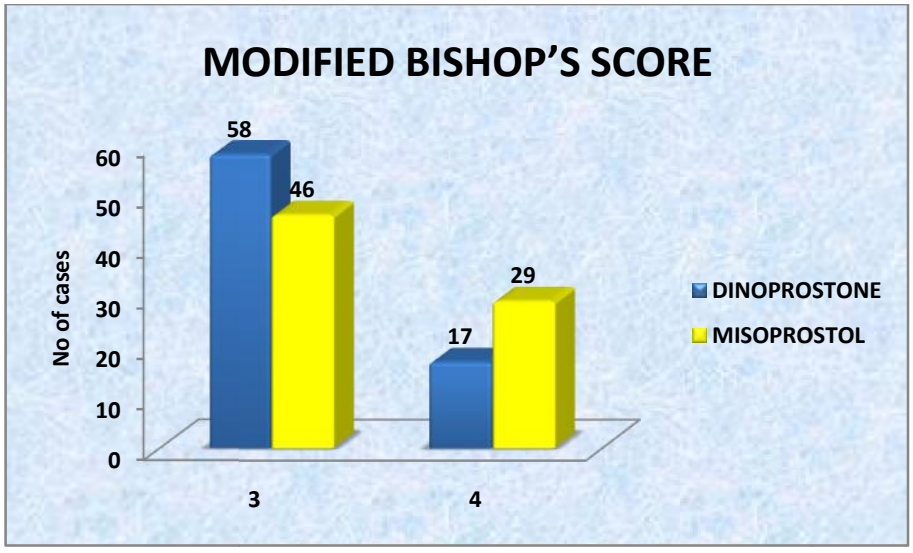
The above graph represents the need for oxytocin augmentation in both the dinoprostone and misoprostol group.

Table 7: Distribution of cases by Modified Bishop's Score between study groups

MODIFIED BISHOP'S SCORE	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
3	58	77.3%	46	61.3%	0.034 (Sig)
4	17	22.7%	29	38.7%	
Total	75	100.0%	75	100.0%	

The p value related to Modified bishop score in both the dinoprostone and Misoprostol group is 0.034 and is statistically significant. 77.3% and 22.7% of patients had MBS of 3 and 4 respectively in Dinoprostone group. In Misoprostol group, 61.3% and 38.7% of patients had MBS as 3 and 4 respectively.

Graph 7: Distribution of cases by Modified Bishop's Score between study groups



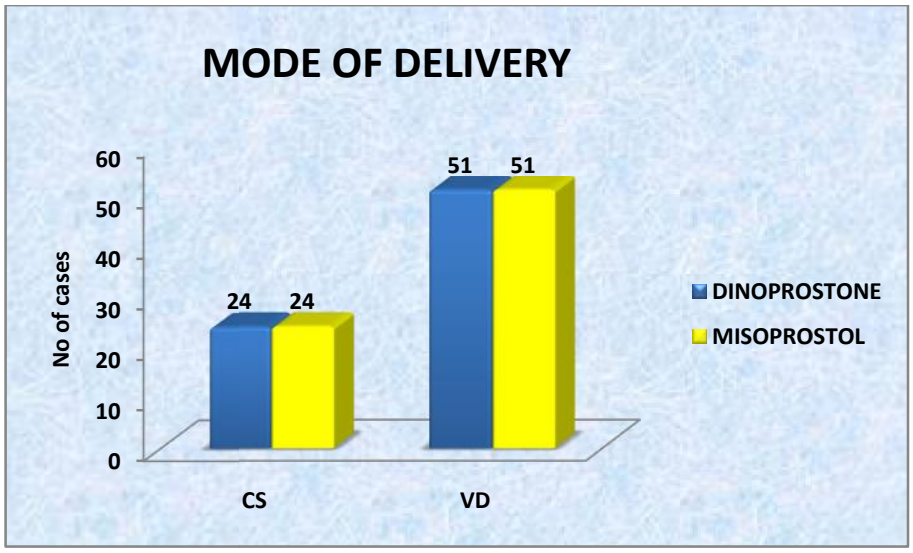
The above graph represents the MBS of both Dinoprostone and Misoprostol group.

Table 8: Distribution of cases by Mode of Delivery between study groups

MODE OF DELIVERY	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
CS	24	32.0%	24	32.0%	NA
VD	51	68.0%	51	68.0%	
Total	75	100.0%	75	100.0%	

The mode of delivery in both the groups was same. 32.0% and 68.0% of patients in both the Dinoprostone group and Misoprostol group underwent C- Section and vaginal delivery respectively.

Graph 8: Distribution of cases by Mode of Delivery between study groups



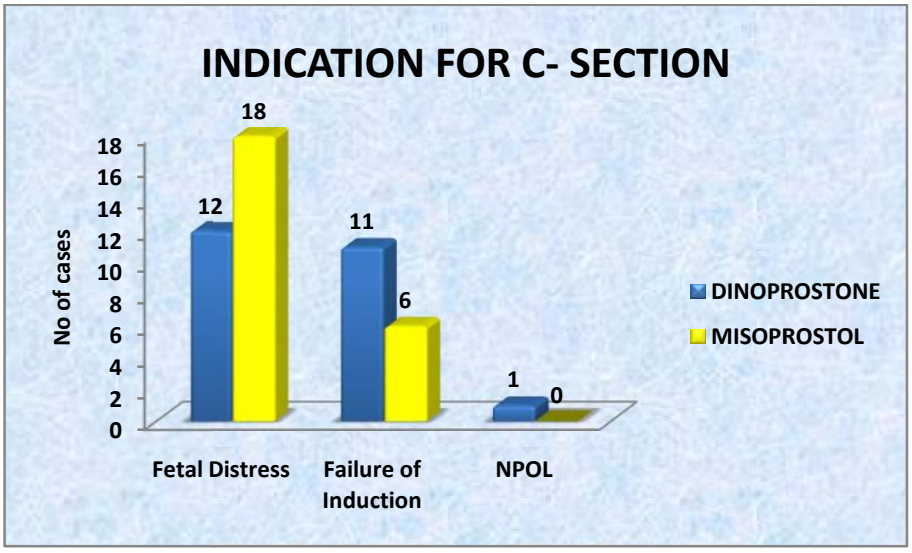
The above graph represents the mode of delivery in both dinoprostone and misoprostol group.

Table 9: Distribution of cases by Indication for C- section between study groups

INDICATION FOR C- SECTION	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
Fetal Distress	12	16.0%	18	24.0%	0.299
Failure of Induction	11	14.7%	6	8.0%	
NPOL	1	1.3%	0	0.0%	
Total	24	32.0%	24	32.0%	

The above table indicates the indications for C- section in the present study. In both the groups, C- section rate was same and doesn't have any statistical significance. But when analyzed, Dinoprostone group has higher rate of induction failure and where as Misoprostol group has higher rate of fetal distress which where indictions for C- section.

Graph 9: Distribution of cases by Indication for C- section between study groups



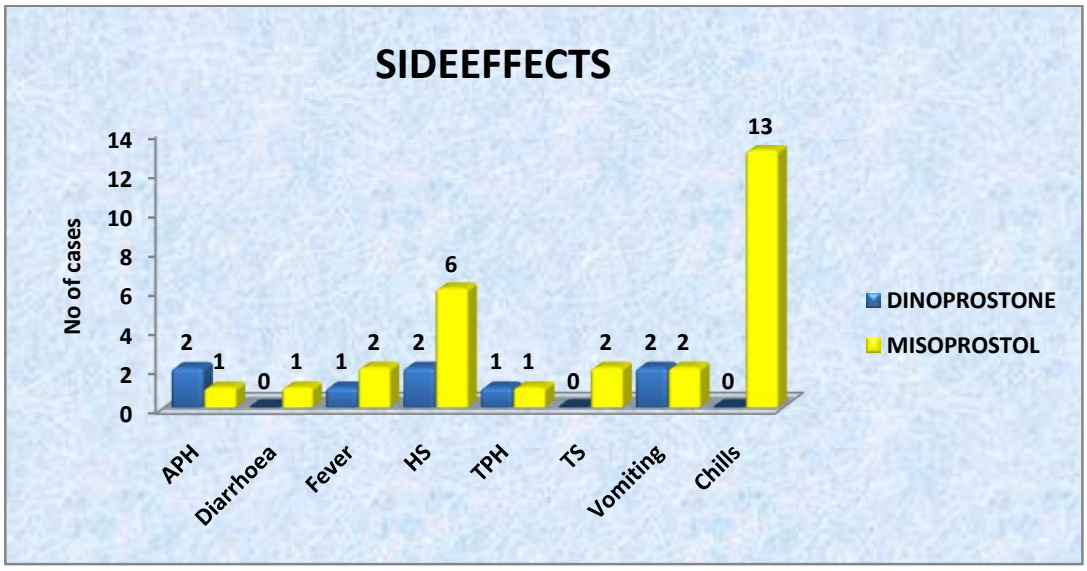
The above graph represents the indications for C- Section which was statistically not significant.

Table 10: Distribution of cases by Side-effects between study groups

SIDEEFFECTS	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
APH	2	2.7%	1	1.3%	0.003 (Sig)
Diarrhoea	0	0.0%	1	1.3%	
Fever	1	1.3%	2	2.7%	
HS	2	2.7%	6	8.0%	
TPH	1	1.3%	1	1.3%	
TS	0	0.0%	2	2.7%	
Vomiting	2	2.7%	2	2.7%	
Chills	0	0.0%	13	17.3%	
Total	8	10.7%	28	37.3%	

The above table represents the side effects in both the Dinoprostone and Misoprostol groups. With this data it is evident that there is higher incidence of side effects in Misoprostol group (37.3%) and is statistically significant (p value = 0.003). The major side effect in the Misoprostol group was chills.

Graph 10: Distribution of cases by Side-effects between study groups



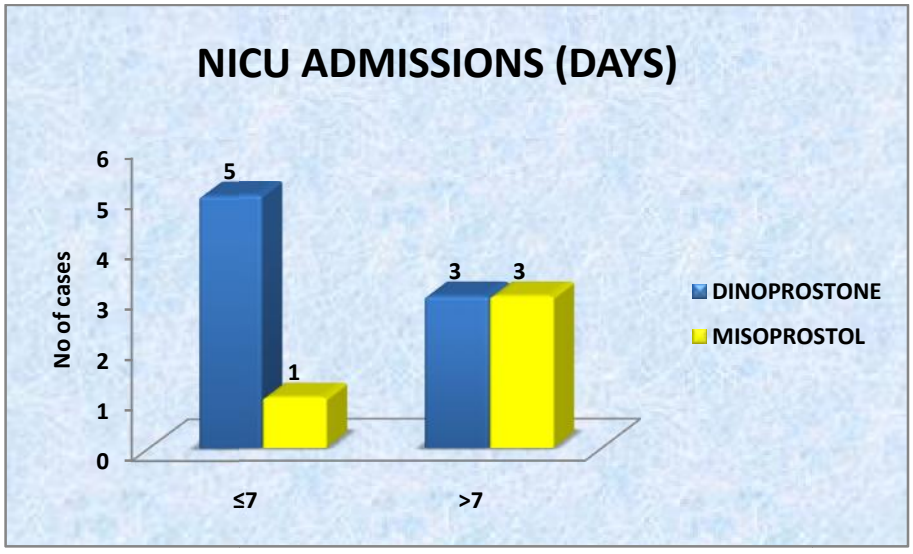
The above graph represents the side effects on mother in both the groups and is more in Misoprostol group.

Table 11: Distribution of cases by NICU admissions (days) between study groups

ICU ADMISSIONS (DAYS)	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
≤7	5	6.7%	1	1.3%	0.221
>7	3	4.0%	3	4.0%	
Total	8	10.7%	4+1	6.7%	

The above table shows the mean number of days the babies were admitted in NICU. One baby from misoprostol group was taken against medical advice (AMA) which was represented as +1. NICU admissions were double in the dinoprostone group when compared to misoprostol group. There is no statistical significance in NICU admissions in both the groups.

Graph 11: Distribution of cases by NICU admissions (days) between study groups



In the Dinoprostone group 5 babies were kept in NICU for less than 7 days and 3 babies were admitted for more than 7 days.

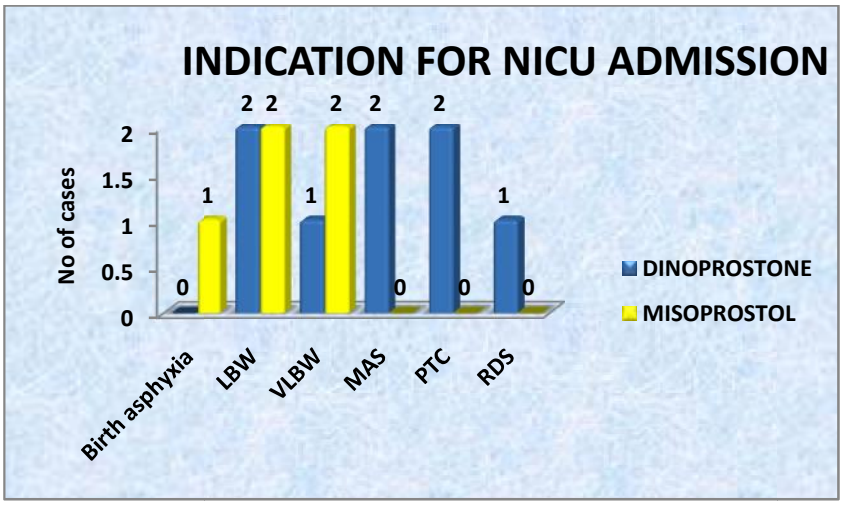
In the Misoprostol group out of 4 babies 3 babies were admitted for less than 7 days and 1 baby was admitted for more than 7 days.

Table 12: Distribution of cases by Indication for NICU Admission between study groups

INDICATION FOR NICU ADMISSION	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
Birth asphyxia	0	0.0%	1	1.3%	0.38
LBW	2	2.7%	2	2.7%	
VLBW	1	1.3%	2	2.7%	
MAS	2	2.7%	0	0.0%	
PTC	2	2.7%	0	0.0%	
RDS	1	1.3%	0	0.0%	
Total	8	10.7%	5	6.7%	

The above table represents the indication for NICU admissions in both Dinoprostone group and Misoprostol group. There is no statistical difference between both the groups.

Graph 12: Distribution of cases by Indication for NICU Admission between study groups



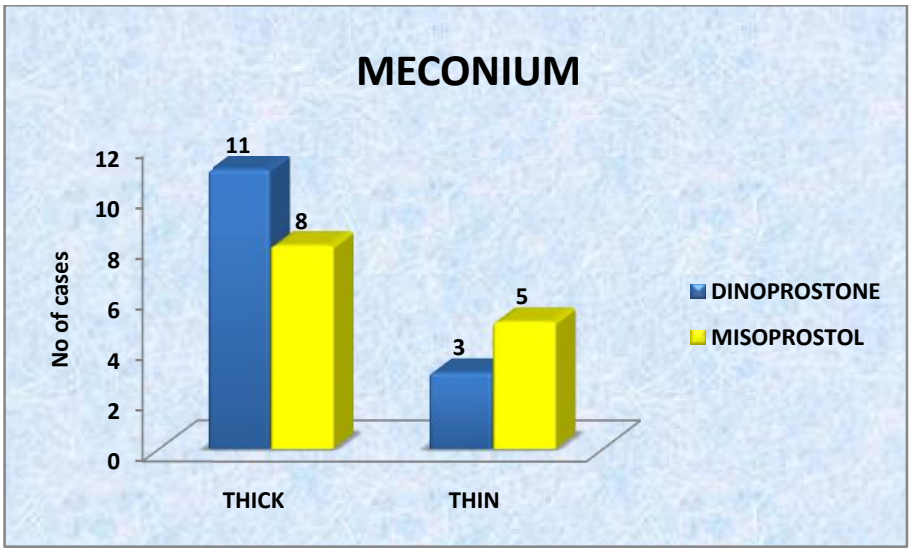
The above graph represents the indication for NICU admissions. In both the groups, LBW as indication for NICU admission was same. In the Misoprostol group 1 baby got admitted for birth asphyxia, 2 babies admitted for VLBW. In Dinoprostone group 2 babies admitted for MAS, 1baby admitted for VLBW, 2 babies admitted for PTC and 1 baby admitted for RDS.

Table13 : Distribution of cases by Meconium between study groups

MECONIUM	DINOPROSTONE		MISOPROSTOL		p value
	N	%	N	%	
THICK	11	14.7%	8	10.7%	0.612
THIN	3	4.0%	5	6.7%	
Total	14	18.7%	13	17.4%	

The above table represents the data regarding meconium stained liquor. The percentage of meconium stained liquor is 18.7% in dinoprostone group and 17.4% in misoprostol group. However, we could not rule out that if meconium stained liquor was due to induction or post dated pregnancy.

Graph 13: Distribution of cases by Meconium between study groups



The above graph represents the meconium stained liquor in both the groups which was statistically not significant.

DISCUSSION

In the present study 150 patients were studied with indications for induction of labour of which 75 patients received intracervical Dinoprostone gel containing 0.5mg and 75 patients received intravaginal Misoprostol tablet 50µ g and same dose was repeated after 6 hours as required up to maximum of 3 doses.

Patients' characteristics:

- Parity: There is no statistical significance regarding parity in both the groups.
- Gestational age: Majority of patients are of above 40 weeks of gestational age in Dinoprostone group where as in Misoprostol group, majority of cases are in between 37 to 40 weeks of gestational age. It is statistically significant (p= 0.002).

Indication for induction:

Dinoprostone group has high number of cases with indication as post dated pregnancy while Misoprostol group has high number of cases with indication as severe preeclampsia. And it is statistically significant (p= 0.001).

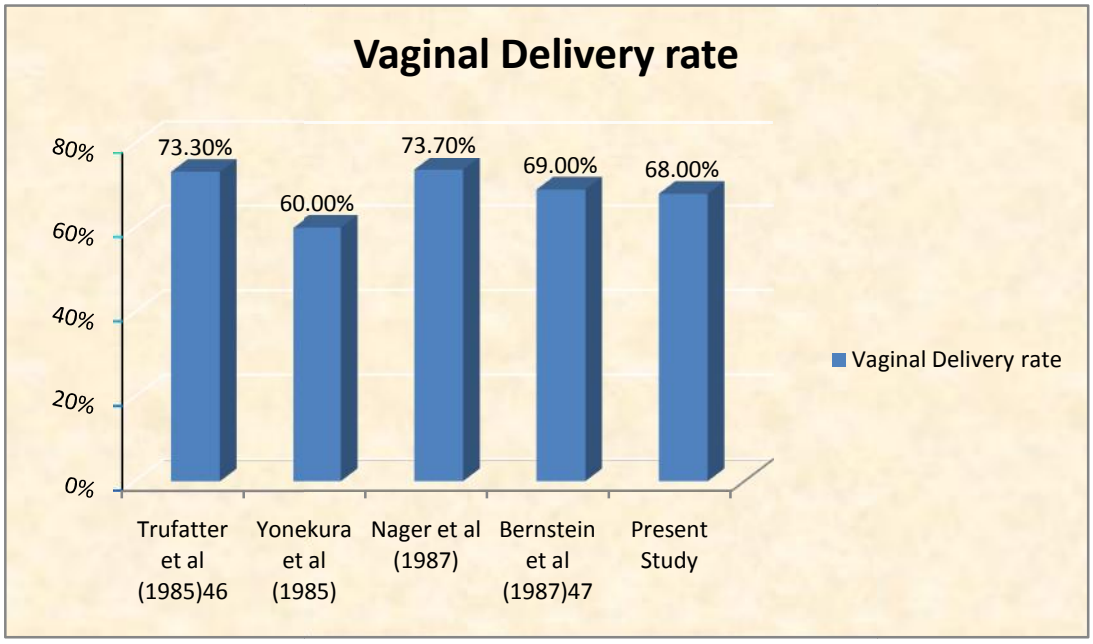
Response to Drug:

Vaginal Deliveries

The rate of vaginal deliveries was 68% in both Dinoprostone group and in the Misoprostol group.

Table -14: Dinoprostone Vaginal Delivery Rate

DINOPROSTONE	
Authors and year	Vaginal Delivery rate
Trufatter et al (1985)	73.3%
Yonekura et al (1985)	60.0%
Nager et al (1987)	73.7%
Bernstein et al (1987)	69.2%
Present Study	68.0%

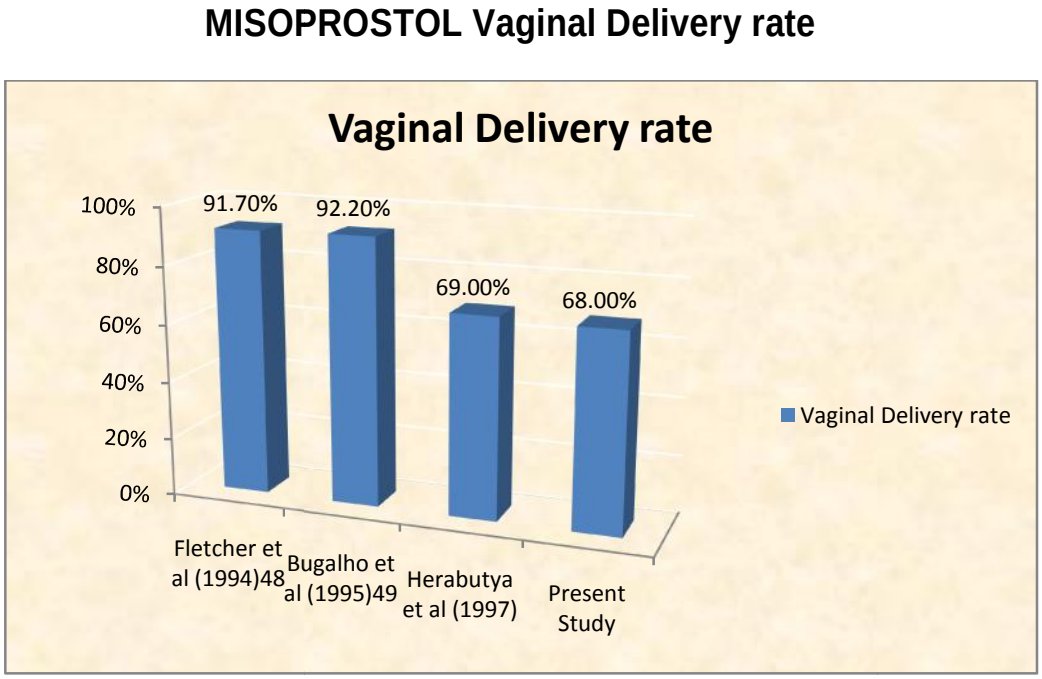


Graph- 14 Vaginal Deliveries rates with Dinoprostone according to Authors

In present study, the rate of vaginal delivery in the Dinoprostone group is consistent with the studies of Bernstein et al (1987)[47]

Table-15: Misoprostol Vaginal Delivery Rate

MISOPROSTOL	
Authors and year	Vaginal Delivery rate
Fletcher et al (1994)	91.7%
Bugalho et al (1995)	92.2%
Herabutya et al (1997)	69.0%
Present Study	68.0%



Graph 15: Vaginal Deliveries rates with Misoprostol according to authors

The vaginal delivery rate with Misoprostol group in present study is comparable to the studies of Herabutya et al (1997) in which vaginal delivery rate was 69%.

Bishop's Score :

In the present study there is statistical difference in regard to Bishop score prior to induction in both the groups (p= 0.034). Majority of cases had 3 as their Bishop score. When both the groups are compared, Dinoprostone group had more number of cases with Bishop score 3 and more number of cases in Misoprostol group had Bishop score 4.

Induction to vaginal delivery interval:

In the present study it was seen that the induction delivery interval was shorter in the Misoprostol group compared to Dinoprostone group ,10.89 ± 7.28 hrs and 7.83 ± 5.63 hrs respectively. This was statistically significant (P<0.05).

Table-16: Induction to vaginal delivery interval

DINOPROSTONE	
Authors and year	induction delivery interval
Trufatter et al (1985)	13.3 ± 6.2
Yonekura et al (1985)	13.1 ± 8.1
Nager et al (1987)	10.1 ± 2.1
Bernstein et al (1987)	12.3 ± 16.5
Present Study	10.89 ± 7.28

In the present study the induction – delivery interval of Dinoprostone is comparable to the studies of Nager et al (1987) and Bernstein et al (1987).

Table -17: Induction to vaginal delivery interval

MISOPROSTOL		
Authors and year	Dosage Max Dose	IDI (hrs)
Sanchez Ramos et all (1993)	50µ g 4hrs (600 µ g)	11 ± 7.3
Fletcher et al (1994)	100 µg (100 µg)	15.6 ± 12.5
Wing et al (1995a)	50 µg 3 hrs(300µ g)	15.1 ± 8
Wing et al (1995b)	25 µg 3 hrs(200µ g)	22.1 ± 14.5
Bugalho et all (1995)	50 µg 12 hrs(200µ g)	10.4
Present Study	50µg 6hr (150 µg)	7.83 ±5.63

In the Misoprostol group it has been shown that by various dosages of Misoprostol used the induction – delivery interval also varies. Our present study uses 50µg Misoprostol every 6^{th} hourly with an induction delivery interval of 7.83 ± 5.63 hrs which is comparable to the studies of Bugalho et al (1995) who has used 50µ g Misoprostol 12^{th} hourly to a maximum of 200µ g with an induction delivery interval of 10.4hrs and Sanchez Ramos et al (1993) who used 50µ g Misoprostol 4^{th} hourly to a maximum of 600µ g with an induction delivery interval of 11 ± 7.3hrs.

Table -18: Induction to vaginal delivery interval

Authors and year	DINOPROSTONE	MISOPROSTOL
Varaklis et al (1995)	22.4 ± 10.9 (0.5mg 6hrs)	16.0 ± 7.7 (25µ g 2hrs)
Wing Da et al (1995)	23.5 ± 14.5 (0.5mg 6hrs)	15.1 ± 8.0 (50µ g 3hrs)
Herabutya et al (1997)	21.36 ± 13.09 (1.5mg)	19.14 ± 10.6 (100µ g)
Ozgur et al (1997)	8.2 ± 5.9 (0.5mg)	7.6 ± 1.9 (100µ g)
Blanchette et al (1999)	31.3 ± 13.0	19.8 ± 10.4
Kolderup et al (1999)	28.52 (0.5mg 6hrs)	19.5 (50µ g 4hrs)
Present Study	10.89 ± 7.28 (0.5mg 6hrs)	7.83 ± 5.63 (50µ g 6hrs)

Various authors in their studies have compared the efficacy of Misoprostol and Dinoprostone in relation to induction – delivery interval.

In the present study the outcome of induction delivery interval is much shorter than the various studies and almost comparable to the studies of Ozgur et al (1997).

FAILED INDUCTION:

Failed inductions were those cases in which contractions did not start or bishop did not improve at the end of 24 hours and were taken up for caesarean section with failure of induction as an indication.

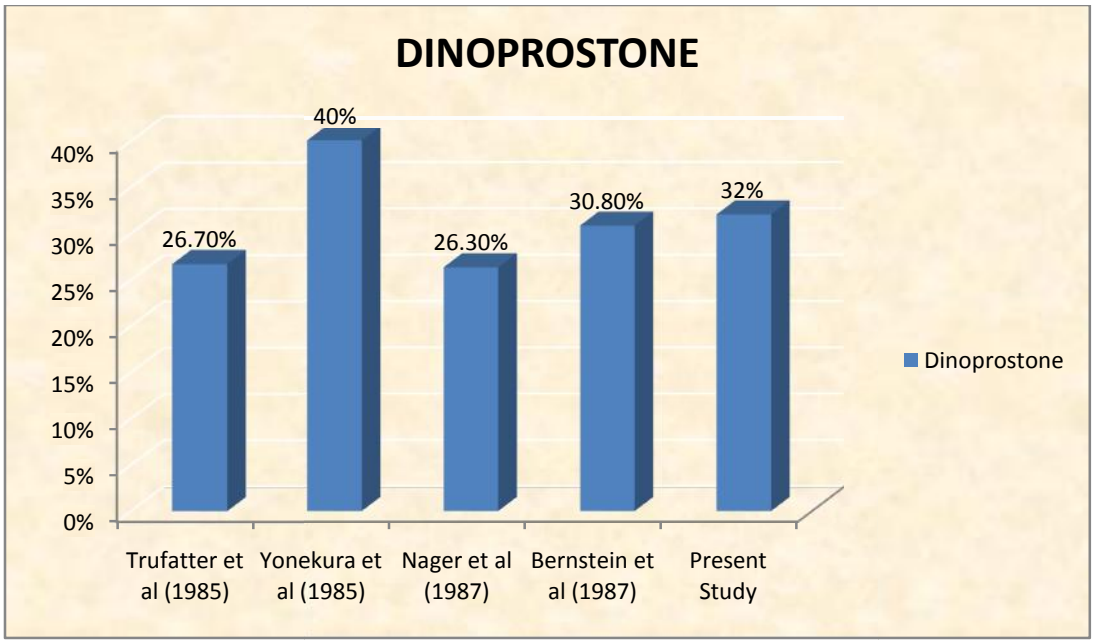
Caesarean delivery rates in the present study are 32% in both the Dinoprostone group and the Misoprostol group. The other indications were fetal distress, non-progression of labour. In the Dinoprostone group, failure of induction formed the major indication for caesarean delivery and in the Misoprostol group fetal distress formed the major indication for caesarean delivery.

In the Misoprostol group it was seen that two cases which had fetal distress, preoperatively it was found to have thick meconium stained liquor.

Table -19: Caesarean Section Rate in Dinoprostone group

DINOPROSTONE	
Author and year	**C.S. Rate**
Trufatter et al (1985)	26.7%
Yonekura et al (1985)	40%
Nager et al (1987)	26.3%
Bernstein et al (1987)	30.8%
Present Study	32%

Graph 16: Caesarean rate in various study groups in regard to Dinoprostone

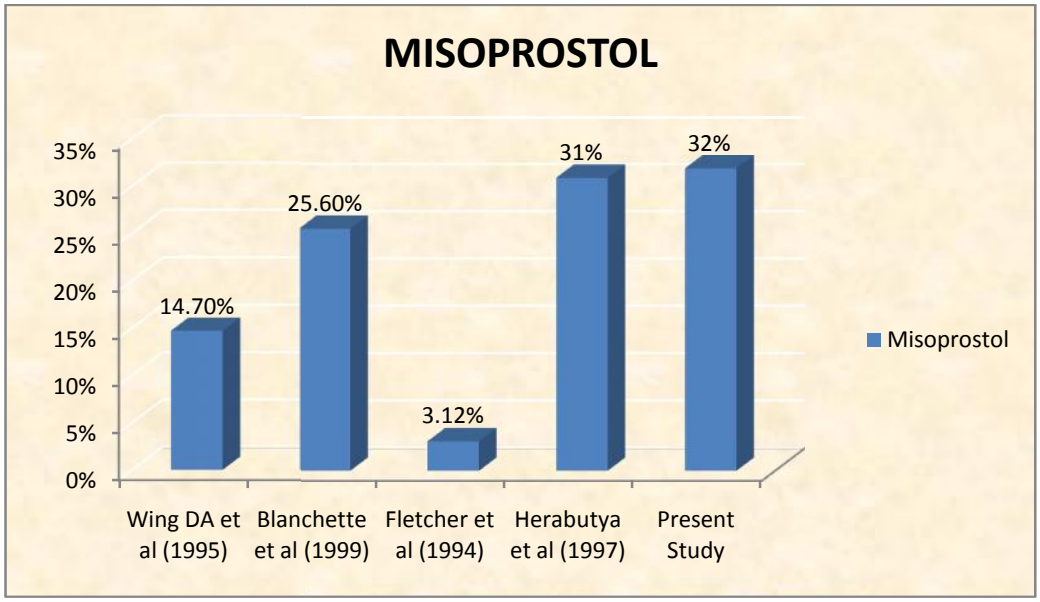


In our study the caesarean section rate with Dinoprostone was 32%, which is consistent with the studies of Bernstein et al.

Table -20: Caesarean Section Rate in Misoprostol group

MISOPROSTOL	
Author and year	**C.S. Rate**
Wing DA et al (1995)	14.7%
Blanchette et al (1999)	25.6%
Fletcher et al (1994)	3.12%
Herabutya et al (1997)	31%
Present Study	32%

Graph 17: Caesarean rate in various study groups in regard to Misoprostol



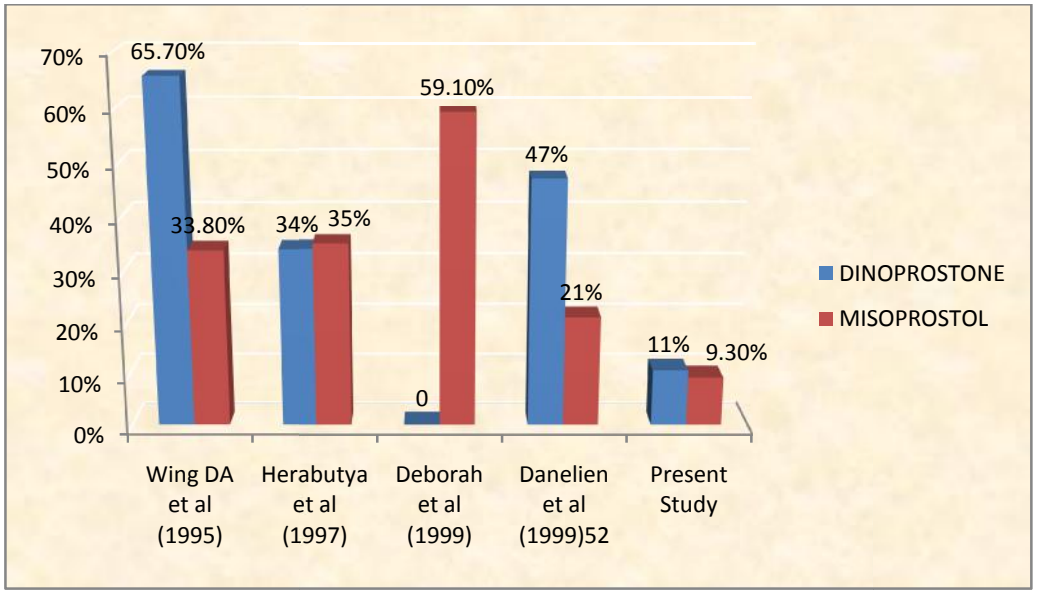
In Misoprostol group the caesarean section rate was 32% which is consistant with the studies of Herabutya et al.

OXYTOCIN AUGMENTATION:

Oxytocin was started depending on the modified Bishops score and in absence of adequate uterine contractions after 6hrs of last dose, or for augmentation in case of arrest of dilation.

Table -21: Oxytocin Augmentation

Author and Year	DINOPROSTONE [dosage (max dose)]	MISOPROSTOL [dosage(max dose)]
Wing DA et al (1995)	65.7% [0.5mg 3hrs(3)]	33.8% [50µ g 3hrs(6)]
Herabutya et al (1997)	34% (1.5)	35% (100 µ g)
Deborah et al (1999)	-	59.1% [25 µ g 4hrs(6)]
Danelien et al (1999)	47% [1 mg 6hrs(3)]	21% [52 µ g 4hrs(4)]
Present Study	10.7% (0.5mg 6hrs)(3)	9.3% 50µ g 6hrs)(3)



Graph 18: Oxytocin Augmentation

In the present study the requirement for oxytocin augmentation was more in the Dinoprostone group – 12% than in the Misoprostol group – 9.3%, this was statistically insignificant. In this study need for oxytocin was very low when compared to all other studies in both the groups.

LIQUOR

The incidence of thick meconium stained liquor was 18.7% and 17.4% in Dinoprostone and Misoprostol groups respectively. More number of patients in the Dinoprostone group were induced for postdatism and found to have thick meconium stained liquor. It was not known whether the thick meconium was due to the drug or due to the indication for induction which was postdatism.

Maternal side Effects

The maternal side effects observed were chills, tachysystole, hyperstimulation, vomiting, diarorhea, fever and PPH.

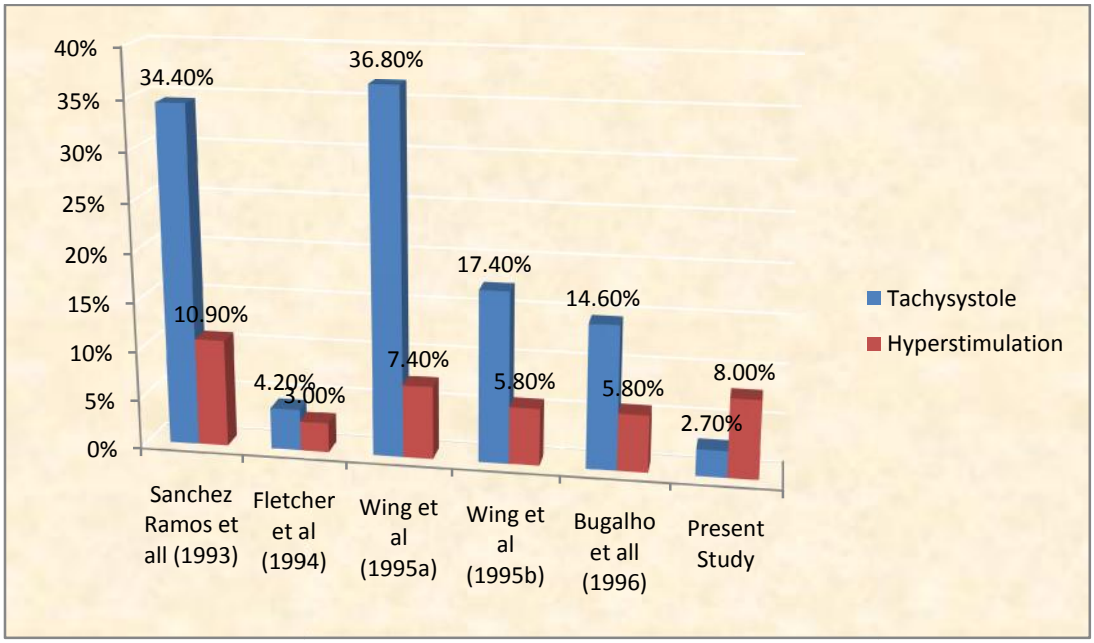
In the Dinoprostone group the major side effects were vomiting – 2.7% and PPH of which traumatic – 1.3% and 2.7% atonic. Vomiting was noticed in patients who had rapid dilation of the cervix and could have been a cause of the same. .

The major side effects observed in the Misoprostol group was chills 17.3%, hyperstimulation 8% , tachysystole 2.7%, fever 2.6% and vomiting 2.7% . Our observations are nearly consistent with the studies of Fletcher et al (1994) and Wing et al (1995a) in regard to tachysystole and hyperstimulation respectively . The difference in the incidence of tachysystole and hyperstimulation by different authors could probably be attributed to the different dosing regimens.

Misoprostol group had 1 patient with traumatic PPH and another one with atonic PPH. Both were treated promptly.

Table-22: Incidence of side effects with Misoprostol

Author and Year	Dosage	Tachysystole	Hyperstimulation
Sanchez Ramos et al (1993)	50µ g q 4hrs	34.4%	10.9%
Fletcher et al (1994)	100µ g single dose	04.2%	3.0%
Wing et al (1995a)	50µ g q 3hrs	36.8%	7.4%
Wing et al (1995b)	25µ g q 3hrs	17.4%	5.8%
Bugalho et all (1996)	25µ g q 3hrs	14.6%	5.8%
Present Study	50µ g 6hrs	2.7%	8.0%



Graph 19: Incidence of side effects with Misoprostol

In present study, incidence of side effects with Misoprostol group is comparable to Wing et al (1995 a) and Scanchez Ramos et al (1993) studies.

NEONATAL OUTCOME:

The mean birth weight and mean APGAR scores in both groups did not show any major difference.

The incidence of NICU admission was 10.6% in Dinoprostone group and 4.9% in Misoprostol group. The indications for NICU admission were meconium aspiration syndrome, birth asphyxia, preterm care, respiratory distress syndrome, very low birth weight, low birth weight. There was an increased incidence of meconium aspiration syndrome in Dinoprostone group and birth asphyxia in the Misoprostol group. As discussed earlier, meconium stained liquor incidence was more in Dinoprostone group, hence the meconium aspiration syndrome incidence was more in Dinoprostone group. If we exclude this particular factor, incidence of NICU admissions in both the groups are almost equal.

Mundle and Young (1996) evaluated the effect of Misoprostol for labour induction on neonatal outcome. They found that neonatal outcome was similar in both the groups (PGE_1 and PGE_2 groups), cord blood acid base analysis did not differ between both the groups. No neonate met the ACOG criteria for birth asphyxia in their study.

Sanchez Ramos et al (1998) their meta analysis found no differences in incidence of low 5minutes apgar score and admission to NICU between Misoprostol and control groups.

CONCLUSION

Misoprostol and Dinoprostone are safe and effective for cervical ripening and labour induction.

Misoprostol is cost-effective when compared to Dinoprostone. Misoprostol is stable at room temperature and does not need refrigeration whereas Dinoprostone requires refrigeration.

Induction to delivery time was shorter in misoprostol group when compared to dinoprostone group.

No. of doses required was less in Dinoprostone group when compared to Misoprostol group. Use of oxytocin was equal in both the groups.

Disadvantages with Misoprostol are chills, uterine tachysystole and hyperstimulation with further fetal distress. Therefore further work is needed to determine the ideal dosing to prevent such complications.

LSCS due to failure of induction was more in cerviprime group and LSCS due to fetal distress was more in misoprostol group.

NICU admissions in both the groups are almost equal in both the groups.

SUMMARY

In the present study, 150 singleton pregnant women who consented for the study and in whom cervical ripening and labour induction was indicated were studied. 75 women received Misoprostol-50ug in the posterior vaginal fornix and other 75 patients received intracervical Dinoprostone-0.5mg gel.

56% were primigravidae and 44% multigravidae in Dinoprostone group when compared to 61.3% primigravidae and 38.7% multigravidae in Misoprostol group.

In Dinoprostone group, 10.7% patients had gestational age of less than 37 weeks, 33.3% patients had gestational age between 37 to 40 weeks and 56% patients had gestational age more than 40 weeks. In Misoprostol group, 9.3% patients had a gestational age of less than 37 weeks, 61.3% patients had gestational age between 37 to 40 weeks and 29.3% patients had gestational age more than 40 weeks.

77.3% and 22.7%of patients in Dinoprostone group had a modified Bishop's score of 3 and 4 respectively prior to induction. In Misoprostol group 61.3% and 38.7% of patients had a modified Bishops score of 3 and 4 respectively.

In Dinoprostone group, 56% required a single dose 0.5mg of gel. The remaining 44% required 2 doses.

In the Misoprostol group, 36% required a single dose, 56% required second dose and 8% required 3 doses.

In Dinoprostone group, 10.7%, required oxytocin augmentation, compared to 9.3% in Misoprostol group.

In the Dinoprostone group the mean induction delivery interval was 10.29± 7.19hrs. In the Misoprostol group the mean induction delivery interval was 7.64 ± 5.75 hrs.

68% cases had a vaginal delivery and 32% had caesarean section in both the groups.

The rate of failed induction was 14.7% in Dinoprostone group which was major indication for C- Section. In the Misoprostol group 8% cases were failed induction, 24% fetal distress which was major indication for C- Section.

There was 10.7% incidence of side effects of Dinoprostone of which vomiting 2.7% and PPH 4% were seen commonly. In Misoprostol group, chills 17.3%, tachysystole 2.7% and hyperstimulation 8% formed the major side effects out of an incidence of 37.3%.

The mean Apgar score was 6.9 at 1 minute and 8.9 at 5 minutes as 6.7 at 1 minute and 8.8 at 5 minutes in Dinoprostone and Misoprostol groups respectively.

There was a 10.7% incidence of thick meconium stained liquor in Misoprostol group, compared to 14.7% incidence in Dinoprostone group.

There was 10.7% and 6.7% incidence of NICU admission in Dinoprostone and Misoprostol groups respectively.

BIBLIOGRAHY

1. Kudagi B.L, Sailaja. L, Pravin Kumar. R, Suresh Babu. C: A Comparative study of intravaginal misoprostol with intracervical dinoprostone gel for induction of labour in pregnancy. Asian J Pharma Clin Res 2013:l6 (2): 174-178.
2. Waraich S, Naqui Z: Comparision of intravaginal Misoprostol and Dinoprostone for Induction of Labour: Pakistan Journal of Surgery 2009: 25(3): 209-213.
3. Van Gemund N, Scherjon S, Lecessie S, Van Leeuwen JH, Van Roosmalen J, Kanhai HH: A randomized trial comparing low dose vaginal misoprostol and dinoprostone for labour induction. BJOG 2004,111: 42-49.
4. Pooja Deodhar, Suman Rai: Comparative Study of Misoprostol versus Dinoprostone for Induction of Labour. Indian Medical Gazette, Dec-2013, 454-457.
5. S. Kulshrestha, P. Sharma, G. Mohan, Sunita Singh, Saroj Singh: Comparitive study of Misoprostol Vs Dinoprostone for induction of labour. Indian J Physiol Pharmacol 2007: 51(1): 55-61.
6. Blanchete Howard A, Sandhya Nayak. Comparison of the safety and efficacy of intravaginal misoprostol (PGE1) with those of dinoprostone (PGE2) for cervical ripening and induction of labour in community hospital. Am J Obstet Gynaecol, 1999;180:P1551-9.
7. Chuck Frank J and B.Joyce Huffaker. Labour induction with intravaginal misoprostol versus intracervical prostaglandin E2 gel (prepidil gel): randomized comparison. Am J Obstet Gynaecol, 1995;173:P1137-42.

8. Kolderup Lindsay, Lynn McLean. Misoprostol is more efficacious for labour induction than prostaglandin E2. Am J Obst & Gynaecol, 1999;10:P1543-50.
9. Deborah A.Wing, Ann Rahall. Misoprostol: An effective agent for cervical ripening and labour induction. Am J Obstet Gynaecol, 1995;172:P1811-6.
10. David Buser, Gerardo Mora. A randomized comparison between misoprostol and dinoprostone for cervical ripening and labour induction in patients with unfavorable cervices. Obstet Gynaeocl, 1997;89:P581-5.
11. Varakalis K, Gumina .R. Randomised controlled trial of vaginal misoprostol and intracervical PGE2 gel for induction of labour at term. Obstet Gynaeocl, 1995; Oct. 86 P541 – 4.
12. Jose. L. Bartha; Rafael Comino – Delgado; Oral Misoprostol and Dinoprostone fro Cervical Ripening and labour Induction. A Randomized comparison Obstetrics and Gynecology 2000; Vol: 96, No.3, September P465– 9
13. French.L, Oral prostaglandin E2 for induction of labour. Cochrane database of systematic reviews 2007, issue 1. Art. No. CD003098. DOI: 10. 1002/14651858. CD003098.
14. Neiger R, Greaves PC. Comparison between vaginal misoprostol and cervical dinoprostone for cervical ripening and labor induction. Tenn Med. 2001 Jan;94(1):25-7.
15. Agarwal N, Gupta A, Kriplani A, Bhatla N, Parul. Six hourly vaginal misoprostol versus intracervical dinoprostone for cervical ripening and labor induction. J Obstet Gynaecol Res. 2003 Jun;29 (3): 147-51.
16. A mali U L okugamage; S ophie F F orsyth; K eith R S ullivan ; H azem E I R efaey; C harles H R odeck Dinoprostone versus misoprostol: a randomized

sudy of nulliparous women undergoing induction of labor Acta Obstetricia et Gynecologica Scandinavica Vol: 82, No: 2, February, 2003 P1133-137.

17. D. Garry; R. Figueroa; R.B. Kalish Randomized Controlled Trial of Vaginal Misoprostol Versus Dinoprostone Vaginal Insert for Labor Induction Journal of Maternal-Fetal and Neonatal Medicine Vol: 13, No: 4, April, 2003 P 254-259.
18. Patrick S. Ramsey; Lane Meyer; Becky A. Walkes; Denise Harris; Paul L. Ogbur; Jr; Robert H. Heise; Kirk D. Ramin Cardiotocographic Abnormalities Associated with Dinoprostone and Misoprostol Cervical Ripening Obstetrics and Gynecology Vol: 105, No: 1, January, 1 2005 P85-90.
19. Marjorie Meyer; Jeannie Pflum; Diantha Howard Outpatient Misoprostol Compared with Dinoprostone Gel for Preinduction Cervical Ripening: A Randomized Controlled Trial Obstetrics and Gynecology Vol: 105, No: 3, March, 1 2005 P 466-47.
20. Murthy Bhaskar Krishnamurthy, Arkalgud Mangala Srikantaiah Misoprostol alone Versus a Combination of Dinoprostone and Oxytocin for Induction of Labor Journal of Obstetrics and Gynecology of India Vol: 56, No:5, October, 2006 P413-416.
21. Sifakis S, Angelakis E, Avgoustinakis E, Fragouli Y, Mantas N, Koukoura O, Vardaki E, Koumantakis E. A randomized comparison between intravaginal misoprostol and prostaglandin E2 for labor induction. Arch Gynecol Obstet. 2007 Apr; 275(4): 263-7 Epub 2006 Oct 7.
22. Lapaire O, Zanetti-Dallenbach R, Weber p, Hosli I, Holzgreve W, Surbek D. Labor induction in preeclampsia: is misoprostol more effective than dinoprostone? J Perinat Med. 2007; 35(3): 195-9.

23. Denguezli W, Trimech A, Haddad A, Hajjaji A, Saidani Z, Faleh R, Sakouhi M. Efficacy and safety of six hourly vaginal misoprostol versus intracervical dinoprostone: a randomized controlled trial. Arch Gynecol Obstet. 2007 Aug: 276(2): 119-24.
24. Calder AA, Loughney AD, Weir CJ, Barber JW. Induction of labour in nulliparous and multiparous women: a UK, multicentre, open-label study of intravaginal misoprostol in comparison with dinoprostone . BJOG. 2008 Sep;115(10): 1279-88.
25. Prager M, Eneroth-Grimfors E, Edlund M, Marions L. A randomized controlled trial of intravaginal dinoprostone, intravaginal misoprostol and transcervical balloon catheter for labour induction. BJOG. 2008 Oct; 115 (11) : 1443-50. Epub 2008 Aug 19.
26. Sebiha Ozkan; Eray Caliskan; Emek Doger; Izzet Yucesoy; Semih Ozeren; Birol Vural Comparative Efficacy and Safety of Vaginal Misoprostol Versus Dinoprostone Vaginal Insert in Labor induction at Term: a Randomized Trial Archives of Gynecology and Obstetrics Vol: 280, No:1, July, 2009 P 19-24.
27. Danforth D N et al ; Connective tissue changes incident to cervical effacement. Am J. Obstet Gynaecol 1960:86: P 939-945.
28. Mochizuki M. et al A study on the effect of dehydroepiandrosterone sulfate on so-called cervical ripening. Acta obstet Gynoecol scand 1978; 57: 397-401.
29. Kelly AJ, Kavanagh J, Thomas J. Vaginal prostaglandin (PGE2 and PGF2a) for induction of labour at term. Cochrane Database of Systematic Reviews 2003; (4): CD003101.
30. EI-Shawarby SA, Connell RJ. Induction of labour at term with vaginal prostaglandins preparation: a randomized controlled trial of Prostin vs

Propess. Journal of Obstetrics and Gynaecology 2006; 26(7):627-30.

31. Joint Formulary Committee. British National Formulary. 54th ed. London: British Medical Association and Royal Pharmaceutical Society of Great Britain; 2007.
32. Hutton E, Mozurkewich E. Extra-amniotic prostaglandin for induction of labour. Cochrane databse of Systematic Reviews 2001; (2): CD003092
33. Boulvain M, Kelly A, Irion O, Intracervical prostaglandins for induction of labour. Cochrane Database of Systematic Reviews 2008;(1): CD006971
34. Kelly AJ, Tan B. Intravenous oxytocin alone for cervical ripening and induction of labour. Cochrane Database of Systematic Reviews 2001;(3):CD003246.
35. Howrath GR, Botha DJ. Amniotomy plus intravenous oxytocin for induction of labour. Cochrane Database of Systematic reviews 2001 ;(3):CD003250.
36. Alfirevic Z, Weeks A. Oral misoprostol for induction of labour. Cochrane Database of Systematic Reviews 2006 ;(2):CD001338.
37. Paungmora N. Comparison of oral and vaginal misoprostol for induction of labor at term: a randomized controlled trial. Journal of Obstetrics and Gynaecology Research 2004; 30(5): P358-62.
38. Kipikasa JH, Adair CD, Williamson J, et al. Use of misoprostol on an outpatient basis for postdate pregnancy. International journal of gynecology and Obstetrics 2005; 88(2): P108-11.
39. Gherman RB. Oral misoprostol vs. intravaginal prostaglandin E2 for preinduction cervical ripening. A randomized trial. Journal of Reproductive Medicine 2001;46(7): P641-6.
40. Zhang A, Leng W, Zhang X, et al. Effect of mifepristone on ultrastructure of

fetal kidney in second trimester of pregnancy. Journal of Jilin University 2006; 32(5): P 854-7.

41. Kavangh J, Kelly AJ, Thomas J. Hyaluronidase for cervical ripening and induction of labour. Cochrane database of Systematic Reviews 2006 Apr 19;(2): CD00309.
42. Kavangh J, Kelly AJ, Thomas J. Cortiosteroids for cervical ripening and induction of labour. Cochrane Database of systematic reviews 2006 ;(2): CD003100.
43. Thomas J, Kelly AJ, Kavanagh J. Oestrogens alone or with amniotomy for cervical ripening or induction of labour. Cochrane Database of Systematic Reviews 2001 ;(4):CD003393.
44. Boulvain M, Stan C, Irion O, Membrane sweeping for induction of labour. Cochrane Database of Systematic Reviews 2005 ;(1):CD000451.
45. Cammu H, Haitsma V. Sweeping of the membranes at 39 weeks in nulliparous women: a randomised controlled trial. BLOG: an International journal of obstetrics and Gynecology 1998; 105(1):P 41-4.
46. Berghella V, Rogers RA, Lescale K. Stripping of membranes as a safe method to reduce prolonged pregnancies. Obstetrics and Gynecology 1996;87(6):P 927-31.
47. Wiriyasirivaj B, Vutyavanich T, Ruangsri RA. A randomized controlled trial of membrane stripping at term to promote labor. Obstetrics and Gynecology 1996; 87(5 Pt 1):P 767-70.
48. Williams. Induction and Augmentation of Labor. In: F. Gray Cunningham et al. Williams Obstetrics. 24th ed. United States of America. Mc Graw- Hill Education.2014.523-534.

49. Renu Mishra. Induced labour. In: Renu Mishra. 7th ed. New Delhi. Wolters Kluwer India Pvt. Ltd.2014.496-513.

50. Duru Shah and Sudeshna Ray. Induction of Labour. In: S. ArulKumaran, Gita A, Leonie K P. 3rd ed. Hyderabad. Universities Press (India) Private Limited.2011.368-386.

51. Truffater F K, Donette Bowers, Stanley A G, Allan P K, Preinduction cervical ripening with PGE2 (Prepidil) gel, American Journal of Obstetrics and Gynecology, 1985:153: P 268-271.

52. Bernstein Paul, Leyland Nicholas, Paul Gurland, Doughlas Gare, Cervical repining and labour induction with PGE2 gel-A placebo controlled study, American Journal of Obstetrics and Gynecology,1987:156: P 336-340.

53. Fletcher H, Mitchell S, Fredrick J, Simon D, Brown D, Intravaginal Misoprostol versus Dinoprostone as a cervical ripening and labour inducing agents: Obstetrics and Gynecology, Feb-1994:83(2): P 244-7.

54. Bugalho A, Bigne C, Machungo F, Farnandes A, A low dose veginal misoprostol for induction of labour with a live fetus. International journal Obstetrics and Gynecology:1995:49: P 149-155

55. Sanchez Ramos L, Kaunitz AM, Wears RC, Delka I, Gaudier FL, Misoprostol for cervical ripening and labour induction: A meta analysis, Obstetrics and Gynecology,1998: 89: P 633-642.

56. Herabutya Y, Prasertsawet P, Pokpirom J, A comparision of Intravaginal misoprostol and intracervical PGE2 gel for ripening of unfavourable cervix and labour induction. Journal Obstetrics and Gynecology, Aug-1997: 23(4), P 369-74.

57. Peter Danilien, Belinda Porter, Natalia Ferri, Jenny Summers, Allen Templeton 1999 Aug, Misoprostol for induction of labour at term: A more effective agent than dinoprostone vaginal gel. International journal Obstetrics and Gynecology, Aug- 1999:106; P 793-797.

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SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 22-11-2014 *at* 3-30pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title Comparative study of intravaginal misoprostol with intracervical dinoprostone gel for induction of labour.

Name of P.G. student Dr. Ramya Deva. Dept of OBG.

Name of Guide/Co-investigator Dr D.B. Taju, Prof & HOD. Dept of OBG

for

DR.TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

BLDE UNIVERSITY'S SRI BM PATIL MEDICAL

COLLEGE VIJAYAPUR-586103

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,

INFORMED CONSENT FOR PARTICIPATION IN

DISSERTATION/RESEARCH:

I the undersigned…… s/o. D/o. W/o…….aged……years ordinarily resident of……. do here by state/ declare that Dr…….. of ……. Hospital has examined me thoroughly on…… at …….. and has explained to me in my own language……. that I am suffering from ……..disease (condition) and this disease/ condition mimic following diseases…….. Further Doctor…….informed me that he/ she is conducting dissertation/ research titled"……

…….under guidance of Dr…….requesting my participation in the study. Apart from routine treatment procedure of doing the video assisted laproscopic thoracoscopy treatment, the preoperative, operative, post operative & follow up observations will be utilized for the study as the reference data.

Doctor…….has also informed me that during conduct of this procedure……..like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstance it may prove fatal in spite of anticipated diagnosis & best treatment made available. Further Doctor has informed me that my participation in this study help in evaluation of results of the study which is useful reference for treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/ video graphs taken upon me by the investigator will be kept secret and not accessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary based on information given to me, I can ask any clarification during the course of treatment/ study related to Diagnosis, Procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can with draw from my participation in this study at any time if I want or investigator can terminate me from study at any time from the study but not the procedure of treatment & Follow up unless I request to discharge.

In view of anticipated/ unexpected complications during the course of study, that I will be treated free of cost, as explained by the investigator.

After understanding the nature of dissertation or research, Diagnosis made, mode of treatment I the under signed Shri/ smt…….under my full conscious state of mind I agree to participate in the said research/ Dissertation.

Signature of patient:

Signature of Doctor:

Witness 1.

2.

Date:

Place:

PROFORMA

NAME: I.P.NO:

AGE: D.O.A:

OCCUPATION: D.O.D:

S.E.STATUS:

BOOKED/UNBOOKED :

ADDRESS: L.M.P:

PRESENTINE COMPLAINTS:

- I TRIMISTER
- II TRIMISTER
- III TRIMISTER

OBSTETRIC HISTORY:

- MARRIED LIFE
- CONSANGUINOUS/NON-CONSANGUINOUS

GRAVIDA - PARA - LIVING - ABORTION –

NO	MONTH & YEAR	PREGNANCY EVENTS	LABOUR EVENTS	METHOD OF DELIVERY	PUEPERIUM	BABY NOTES

MENSTRUAL HISTORY –

- AGE OF MENARCHE
- PREVIOUS MENSTRUAL CYCLES

L.M.P- E.D.D:- CORRECTED E.D.D –

PAST HISTORY

- H/O HYPERSENSETIVITY TO PROSTAGLANDINS
- H/O TUBERCULOSIS
- H/O DIABETES
- H/O HYPERTENSION
- H/O PREVIOUS OPERATIONS
- H/O MALARIA/VIRAL INFECTIONS
- H/O BLOOD TRANSFUSIONS
- OTHERS

FAMILY HISTORY

- H/O DIABETES
- H/O TUBERCULOSIS
- H/O ASTHMA
- H/O TWINS
- H/O CONG. MALFORMATIONS
- H/O EPILEPSY
- OTHERS

PERSONAL HISTORY –

- DIET
- SLEEP
- APPETITE

- BOWEL
- BLADDER
- OTHER HABITS

GENERAL PHYSICAL EXAMINATION –

- UPPER EXTREMITY
- PALLOR - PULSE
- ICTERUS
- LYMPHADENOPATHY - BLOOD PRESSURE
- THYROID
- - RESPIRATORY RATE
- LOWER EXTREMITY
- OEDEMA - TEMPERATURE
- VARICOSITIES
- SPINE

SYSTEMIC EXAMINATION

- CVS
- RS

ABDOMINAL EXAMINATION –

- HEIGHT OF THE UTERUS - UTERINE ACTIVITY
- PRESENTATION - HEAD-ENGAGED/UNENGAGED
- LIQUOR-ADEQUATE/LESS/MORE - FHS

PER VAGINAL EXAMINATION –

- MODIFIED BISHOPS SCORING – 0HRS
- CERVIX
- STATION
- PELVIS

- DILATATION
- CERVICAL LENGTH
- POSITION-POSTR/MID/ANTR
- CONSISTENCY-FIRM/MEDIUM/SOFT

DIAGNOSIS – INVESTIGATIONS –

BLOOD –

- Hb%
- BT
- CT
- BLD GP - Rh
- HIV - Hbs Ag - VDRL

URINE –

- ALBUMIN
- SUGAR
- MICRO

ULTRASOUND –

- Amniotic Fluid index

OTHERS –

INDICATION FOR

INDUCTION –

INDUCING AGENT-MISOPROSTOL/DINOPROSTONE METHOD-

INTRA VAGINAL/INTRACERVICAL DOSAGE –

INTRAPARTUM MONITORING - CONTINUOUS ELECTRONIC MONITORING/INTERMITTENT AUSCULTATION

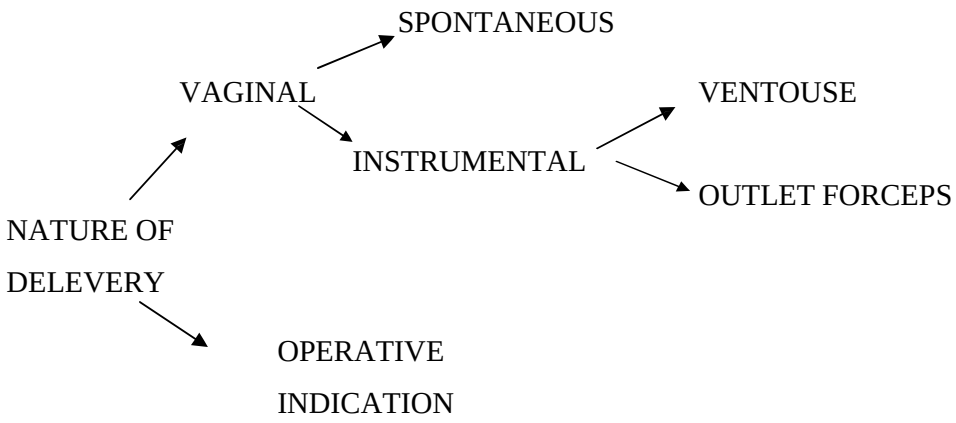
OBSERVATIONS –

- INDUCTION-DELIVERY TIME INTERVAL

DURATION

- 1^{st} STAGE.
- 2^{nd} STAGE.
- 3^{rd} STAGE.

DELIVERY NOTES –



PLACENTA –

- COMPLETE/INCOMPLETE/ANY INFARCTS
- WEIGHT

COMPLICATIONS DURING –

- 2^{nd} STAGE.
- 3^{rd} STAGE.

BABY NOTES –

- LGA/AGA/SGA
- WEIGHT (Kg)
- SEX-MALE/FEMALE
- TIME OF BIRTH
- DATE OF BIRTH
- APGAR SCORE
- CONG. ANOMALIES

IN CASE OF FAILURE/INTERRUPTION

REASON-

SUSEQUENT MODE OF DELIVERY-

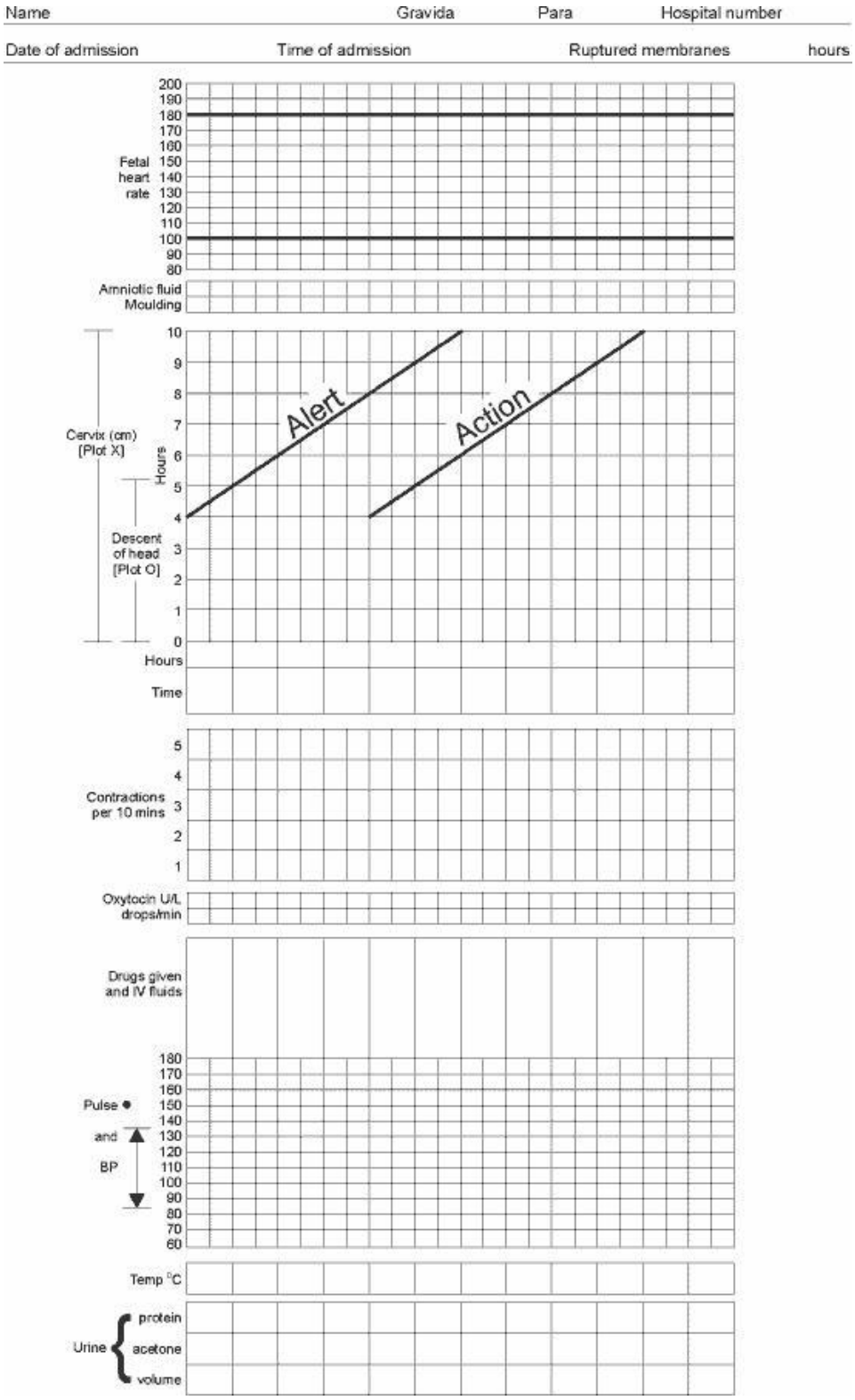
SIDE EFFECTS OF DRUG IF ANY-

- IN NEONATE
- IN MOTHER
    - VOMITING
    - DIARRHOEA
    - FEVER
    - CHILLS
    - UTERINE TACCHYSYSTOLE
    - UTERINE HYPERSTIMULATION

TREATMENT GIVEN FOR SIDE EFFECTS- ANY

REMARKS-

PARTOGRAM


Name
Gravida
Para
Hospital number
Date of admission
Time of admission
Ruptured membranes
hours
Fetal heart rate
200
190
180
170
160
150
140
130
120
110
100
90
80
Amniotic fluid
Moulding
Cervix (cm) [Plot X]
Descent of head [Plot O]
Hours
10
9
8
7
6
5
4
3
2
1
0
Alert
Action
Hours
Time
Contractions per 10 mins
5
4
3
2
1
Oxytocin U/L drops/min
Drugs given and IV fluids
Pulse
and
BP
180
170
160
150
140
130
120
110
100
90
80
70
60
Temp °C
Urine
protein
acetone
volume

KEY TO MASTER CHART

MBS	-	Modified Bishop's Score
ITDT	-	Induction to delivery time
SPE	-	Severe Preeclampsia
MPE	-	Mild preeclampsia
PD	-	Post dated pregnancy
VD	-	Vaginal Delivery
CS	-	caesarean section
F	-	Female
M	-	Male
APPH	-	Atonic Post partum haemorrhage
TPPH	-	Traumatic Post partum haemorrhage
HS	-	Hyper stimulation
TS	-	Tachysystole
F	-	Fever
D	-	Diarrhea
MAS	-	Meconium Aspiration syndrome
BA	-	Birth asphyxia
PTC	-	Preterm care
GHTN	-	Gestational Hypertension
FOI	-	Failure of Induction
FD	-	Fetal distress
AMA	-	Against medical advice.
AG	-	APGAR score
MSE	-	Maternal side effects.
C	-	Chills
V	-	Vomiting

MASTER CHART

DINOPROSTONE

S.NO	IP.NO	NAME	AGE	PARITY	GA	IFI	MBS	ITDT	MOD	IFTN	OXYTOCIN	MSE	SEX	Wt	AG AT 1M	AG AT 5M	MECONIUM	INDICATION FOR NICU ADMISSION	NO. OF DAYS IN NICU
1	11910	MAHANANDA	21	PRIMI	41	PD	4	4H	CS	FD	-	-	M	2.5	7	9	THICK	-	-
2	12996	NEELAMMA	20	PRIMI	32^{+5}	SPE	4	10H	VD	-	-	-	F	1.4	6	9	-	LBW	15
3	12810	SAVITRI	25	G3P2L2	42^{+2}	PD	4	13H	VD	-	-	-	F	3.5	7	9	THICK	-	-
4	13253	MALLAMMA	30	G4P3L2D1	35^{+3}	SPE	4	30H	CS	FOI	-	-	F	2	7	9	-	-	-
5	13673	POOJA	19	PRIMI	41^{+1}	PD	4	9H 30M	VD	-	-	-	F	2.5	7	9	-	-	-
6	13953	PREETHI	19	PRIMI	41^{+5}	PD	3	7H 40M	VD	-	+	-	M	2.9	7	9	THIN	-	-
7	14189	BHAGYASHREE	21	PRIMI	40^{+1}	MPE	3	17H 35M	CS	FD	-	-	F	2.8	7	9	THIN	-	-
8	14830	SHOBHA	25	PRIMI	35	MPE	4	7H	VD	-	-	V	M	2.9	7	9	-	PTC	4
9	15003	ASHWINI	22	G3P1L1A1	41^{+4}	PD	3	26H	CS	FOI	-	-	M	3.2	7	9	-	-	-
10	16036	SHANTABAI	23	G2P1L1	41^{+4}	PD	3	6H 55M	VD	-	-	V	M	2.5	7	9	-	-	-
11	16749	MADHAVI	25	G3P2L2	41	PD	3	6H 55M	VD	-	-	-	M	3	7	9	-	-	-
12	19116	LAXMI	19	G2A1	42	PD	4	7H	VD	-	-	F	F	3.1	7	9	-	-	-
13	20273	BHARATHI	18	PRIMI	42	PD	4	6H	VD	-	-	HS	M	2	7	9	-	-	-
14	20706	LAXMI	28	G2P1L1	35	SPE	3	6H 20M	VD	-	-	-	F	2.28	7	9	-	-	-
15	18487	SEEMA	25	G2P1L1	43	PD	3	8H 45M	VD	-	-	-	M	3	7	9	-	-	-
16	21214	LAXMI	27	PRIMI	39	MPE	3	6H 30M	VD	-	-	-	M	2.6	7	9	-	-	-
17	21384	RAJESHWARI	22	PRIMI	37^{+2}	MPE	4	25H	CS	FOI	-	-	F	2.8	7	9	-	-	-
18	21853	RENUKA	20	PRIMI	44	PD	3	2H	CS	FD	-	-	M	2.8	7	9	THICK	RDS	4
19	24292	USHA	20	PRIMI	34	APE	3	11H 30M	VD	-	+	-	F	1.29	7	9	-	VLBW	14
20	24910	KAVITA	24	G2P1L1	39	GHTN	4	7H	CS	FD	-	-	M	2.6	7	9	-	-	-
21	25734	SHOBHA	24	G2P1L1	41	PD	3	7H 45M	VD	-	-	-	M	2.5	7	9	-	-	-
22	26084	SAVITA	22	PRIMI	41	PD	3	2H 40M	CS	FD	-	-	F	2.4	7	9	-	-	-
23	26229	SHOBHA	38	G2A1	37^{+2}	GHTN	3	5H 10M	CS	FD	-	-	F	1.62	7	9	THICK	LBW	7
24	880	SHARANAMMA	24	G5P3L3D1	38^{+2}	GHTN	3	5H 15M	VD	-	-	-	M	3.1	7	9	THICK	MAS	3
25	787	SABITA	22	PRIMI	32	APE	3	26H	CS	FOI	-	-	F	1.3	7	9	-	PTC	14
26	1395	SUJATA	24	PRIMI	39^{+6}	GHTN	3	4H 58M	CS	FD	-	-	M	3.3	7	9	-	-	-
27	3199	KAVITA	26	G3A2	39^{+1}	IE	3	25H 50M	CS	FOI	-	-	M	3.8	7	9	-	-	-

28	3613	SHALURA	26	G2A1	40^{+3}	PD	3	13H	VD	-	+	-	M	2.7	7	9	-	-	-
29	4387	BHARATHI	23	G2P1L1	41^{+2}	PD	3	11H 25M	VD	-	+	-	F	2.9	7	9	-	-	-
30	5992	RENUKA	24	PRIMI	39	IE	3	3H 45M	VD	-	-	TPH	M	2.5	7	9	-	-	-
31	7406	TABASUM	20	PRIMI	39^{+6}	GHTN	3	11H 20M	VD	-	+	-	M	3.6	7	9	-	-	-
32	7941	LAXMI	26	G5P1L1A3	39	SPE	3	24H	CS	FOI	-	-	F	1.6	7	9	-	-	-
33	8117	DEEPA	18	G4A3	38^{+2}	GHTN	3	7H 20M	CS	FD	-	-	M	2.8	7	9	-	-	-
34	8656	LAXMI	21	PRIMI	36^{+2}	SPE	3	24H	CS	FOI	-	-	F	2.47	7	9	-	-	-
35	9712	KAMALABAI	27	G2P1L1	39^{+1}	GHTN	3	23H 30M	CS	FOI	-	-	F	2.8	7	9	-	-	-
36	10522	ASHWINI	19	PRIMI	40^{+3}	PD	3	10H 50M	VD	-	-	-	M	2.8	7	9	THICK	-	-
37	10510	KAVITA	25	PRIMI	40^{+2}	PD	3	4H 35M	CS	FD	-	-	M	3.2	7	9	THIN	-	-
38	7086	AMBIKA	20	PRIMI	41	PD	3	8H 40M	VD	-	-	-	F	2.7	7	9	THICK	-	-
39	7065	SHASIKALA	28	G3P2L2	40^{+6}	PD	3	4H 15M	CS	FD	-	-	M	3	7	9	THICK	-	-
40	7612	VIDYASHREE	23	PRIMI	42	PD	4	6H	CS	FD	-	-	M	2.5	7	9	THICK	-	-
41	7897	LAXMI	28	PRIMI	40^{+6}	PD	3	6H 20M	CS	FD	-	-	F	2.4	7	9	THICK	-	-
42	10216	VIJAYALAXMI	21	G3A2	40^{+1}	GHTN	3	6H 05M	VD	-	-	-	M	2.9	7	9	-	-	-
43	10581	PUSHPA	28	G4P2L2A1	35^{+6}	SPE	3	24H	CS	FOI	-	-	M	2.4	7	9	-	-	-
44	11520	SAVITRI	22	G2A1	39^{+3}	MPE	4	3H 25M	VD	-	-	HS	M	2.4	7	9	-	-	-
45	12235	NARSAMMA	33	G3P2L2	40^{+2}	PD	3	18H 10M	VD	-	-	APH	F	2.3	6	8	-	-	-
46	12823	KANTA	24	PRIMI	40^{+5}	PD	3	7H 40M	VD	-	+	-	M	2.98	7	9	-	-	-
47	13279	BIBIAISHA	21	PRIMI	41	PD	3	7H 30M	VD	-	-	-	M	3.2	7	9	-	-	-
48	13383	BHARATHI	20	PRIMI	40^{+1}	PD	4	10H	VD	-	-	-	F	4.3	7	9	-	-	-
49	13266	SHREEDEVI	25	G5P4L4	38	SPE	3	24H 30M	CS	FOI	-	-	F	1.4	7	9	-	-	-
50	4547	LAXMI	19	PRIMI	39^{+4}	SPE	3	8H 45M	VD	-	-	-	F	2.5	7	9	-	-	-
51	4773	BHARATHI	20	PRIMI	40^{+2}	PD	3	17H 7M	VD	-	-	-	M	2.4	7	9	-	-	-
52	5064	ROOPA	23	PRIMI	40^{+2}	PD	3	22H 15M	CS	NPOL	-	APH	M	3.9	7	9	-	-	-
53	5108	SUKADEVI	22	G4P3L3	40	GHTN	3	11H 28M	VD	-	-	-	F	2.9	7	9	-	-	-
54	14509	NEELAKKA	20	PRIMI	40^{+6}	PD	3	6H 10M	VD	-	-	-	F	2.6	7	9	-	-	-
55	14698	SHOBHA	20	PRIMI	38	GHTN	3	6H 50M	VD	-	-	-	F	2.6	7	9	-	-	-
56	14877	REKHA	22	PRIMI	41	PD	3	9H 13M	VD	-	-	-	F	2.7	7	9	-	-	-
57	15085	AARTI	22	PRIMI	41	PD	3	8H 20M	VD	-	-	-	M	2.5	7	9	-	-	-
58	15324	REVATI	19	PRIMI	40^{+1}	SPE	3	3H 10M	VD	-	-	-	M	2.5	7	9	-	-	-
59	15410	JYOTIBAI	23	G2P1L1	37^{+2}	GHTN	3	10H 15M	VD	-	-	-	F	2.39	7	9	-	-	-
60	16508	VANISHREE	20	PRIMI	38^{+5}	GHTN	3	10H 45M	VD	-	-	-	M	2.7	7	9	-	-	-
61	20370	LAXMI	25	G3P2L2	40^{+6}	PD	3	5H 16M	VD	-	-	-	F	3.6	7	9	-	-	-
62	3326	MANJULA	22	PRIMI	38^{+2}	SPE	3	16H 10M	VD	-	-	+	F	2.4	7	9	-	-	-
63	34664	SUBADRA	21	PRIMI	37^{+6}	GHTN	3	9H 37M	VD	-	-	-	F	3.6	7	9	-	-	-
64	37841	BHAGYASHREE	22	PRIMI	39^{+6}	GHTN	3	24H	CS	FOI	-	-	F	2.8	7	9	-	-	-
65	39183	NEELAMMA	23	G3P2L2	39^{+5}	GHTN	3	5H 20M	VD	-	-	-	M	2.5	7	9	-	-	-
66	28810	GANGA	18	PRIMI	41	PD	3	4H 20M	VD	-	-	-	F	2.6	7	9	-	-	-

67	29283	ISHWARI	27	G3P2L1D1	41	PD	3	6H 40M	VD	-	-	-	M	3.2	7	9	-	-	-
68	29585	ASHWINI	21	PRIMI	39^{+3}	GHTN	3	11H	VD	-	-	-	F	2.6	7	9	-	-	-
69	30122	KAMAKSHI	25	G5P2L2	41^{+2}	PD	3	8H	VD	-	-	-	M	3.1	7	9	-	-	-
70	31265	SHREEVANI	30	G2P1L1	40^{+2}	PD	3	6H 33M	VD	-	-	-	F	3.3	7	9	-	-	-
71	31380	JANABAI	20	PRIMI	41^{+2}	PD	4	8H 45M	VD	-	-	-	M	2.9	4	6	THICK	MAS	7
72	33636	SUJATHA	20	G2A1	40^{+2}	PD	3	8H 30M	VD	-	+	-	F	2.7	7	9	-	-	-
73	33826	SHABANA	20	PRIMI	40^{+3}	PD	4	4H 20M	VD	-	-	-	F	2.6	7	9	-	-	-
74	33977	LALITHA	20	PRIMI	38	SPE	4	6H 30M	VD	-	-	-	M	2.1	7	9	-	-	-
75	2378	SHAILASHREE	24	G4P3L3	40^{+1}	PD	4	22H 05M	VD	-	+	-	F	3.7	7	9	-	-	-

MISOPROSTOL

S.NO	IPNO	NAME	AGE	PARITY	GA	IFI	MBS	ITDT	MOD	IFIN	OXYTOCIN	MSE	SEX	Wt	AG AT 1M	AG AT 5M	MECONIUM	INDICTION FOR NICU ADMISSION	NO. OF DAYS IN NICU
1	11910	MAHANANDA	20	PRIMI	40^{+2}	PD	3	5H	CS	FD	-	C	M	2.8	7	9	THIN	-	-
2	13186	NANDA	24	PRIMI	35^{+1}	IE	3	15H	CS	FD	-	v	M	1.9	6	8	-	LBW	14
3	13280	JYOTHI	25	G4P3L3	40^{+1}	PD	3	6H	VD	-	-	-	M	3.1	7	9	-	-	-
4	13398	BASALINGAMMA	24	G4P1L1A2	41	PD	4	11H	VD	-	+	C	M	3.2	7	9	-	-	-
5	13635	VAISHALI	31	G2A1	40^{+1}	PD	3	5H 40M	VD	-	-	HS	F	2.4	7	9	-	-	-
6	13612	RESHMA	19	G4P1L1A2	41	PD	4	5H 20M	CS	FOI	-	-	F	2.8	7	9	THICK	-	-
7	14021	BHAGHYASHREE	20	PRIMI	42	PD	4	4H	VD	-	-	HS	M	2.7	6	9	THIN	-	-
8	14140	PARVATI	21	PRIMI	39	MPE	3	4H 45M	VD	-	-	TS	M	3.6	6	9	-	-	-
9	16047	VEENA	28	G2P1L1	42	PD	3	6H	VD	-	-	C	F	2.9	7	9	-	-	-
10	17110	REKHA	24	PRIMI	42	PD	3	4H 48M	CS	FD	-	C	F	3.1	7	9	THICK	-	-
11	18597	DEEPA	26	PRIMI	41^{+6}	PD	4	5H 45M	VD	-	-	F	F	3.1	7	9	-	-	-
12	19984	KALAVATHI	25	G2A1	40	MPE	3	5H 10M	VD	-	-	F	M	3.14	7	9	-	-	-
13	20422	RUKMINI	26	PRIMI	39^{+1}	GHTN	4	5H 55M	VD	-	-	-	M	2.6	7	9	THICK	-	-
14	18255	DEEPA	30	G2P1L1	36^{+5}	MPE	3	4H	VD	-	-	HS	F	3.2	7	9	-	-	-
15	20743	BEBI	21	G3P2L2	41^{+6}	PD	4	10H 15M	VD	-	+	APH	M	2.8	7	9	THICK	-	-
16	21117	LAXMI	30	G2P1LI	38^{+3}	SPE	3	8H 32M	VD	-	+	-	F	2.9	7	9	-	-	-
17	21105	AISHA	20	PRIMI	37^{+2}	MPE	3	5H 5M	VD	-	-	-	M	3.8	7	9	THIN	-	-
18	21356	HEERABASU	25	G2P1L1	38^{+1}	MPE	3	3H 30M	CS	FD	-	-	F	3.1	7	9	-	-	-
19	21603	JAYASHREE	26	PRIMI	37^{+5}	MPE	3	3H 15M	CS	FD	-	-	M	2.6	7	9	-	-	-
20	23050	SAROJINI	30	G4P2L2A1	41^{+3}	PD	3	3H 43M	CS	FD	-	-	M	2.5	7	9	-	-	-
21	24273	SHASHADA	22	PRIMI	42^{+3}	PD	3	13H	CS	FD	-	C	M	2.2	7	9	THIN	-	-

22	24905	KAVERI	23	G2P1LI	38+5	SPE	4	6H	VD	-	-	D	F	2.8	7	9	-	-	-
23	25123	DRAKSHAYANI	23	G2A1	37^{+5}	SPE	3	9H 30M	CS	FD	-	-	M	3.2	4	5	THICK	BA	AMA
24	25458	ROOPA	24	PRIMI	38^{+2}	GHTN	3	5H	CS	FD	-	-	F	3.5	7	9	-	-	-
25	25742	BHAGHYASHREE	22	PRIMI	35	SPE	3	24H 30M	CS	FOI	-	-	F	1.7	7	9	-	-	-
26	26251	CHOUDAMMA	22	PRIMI	38^{+5}	MPE	3	5H 16M	CS	FD	-	-	M	3.3	7	9	THICK	-	-
27	778	VASANTHI	23	PRIMI	42	PD	4	4H 20M	VD	-	-	C	F	2.8	7	9	THIN	-	-
28	1141	KAVITHA	21	G2P1L1	41^{+3}	PD	3	5H 12M	VD	-	-	-	F	2.9	7	9	THICK	-	-
29	1739	SHAMALA	24	G2A1	40^{+6}	GHTN	3	27H	CS	FOI	-	-	M	2.9	7	9	-	-	-
30	3201	SHRUTHI	20	PRIMI	37	GHTN	4	25H 58M	CS	FOI	-	-	M	2.7	7	9	-	-	-
31	4205	ANURADHA	18	PRIMI	34^{+1}	SPE	3	14H 10M	VD	-	+	C	M	1.7	7	9	-	-	-
32	4895	JYOTHI	20	PRIMI	41^{+4}	SPE	4	4H 30M	CS	FD	-	-	M	2.8	7	9	-	-	-
33	5044	SEEMA	26	G2A1	39^{+4}	MPE	3	3H 32M	CS	FD	-	-	F	2.9	7	9	-	-	-
34	6615	REKHA	21	PRIMI	38^{+5}	SPE	4	6H 40M	CS	FD	-	-	M	2.5	7	9	-	-	-
35	7610	SEEMA	22	PRIMI	38^{+6}	SPE	3	6H 50M	VD	-	-	-	F	3.3	7	9	-	-	-
36	8004	MANGALA	20	PRIMI	37^{+3}	MPE	4	4H	CS	FD	-	C	M	2.3	7	9	-	-	-
37	8665	LAXMI	22	PRIMI	40^{+3}	PD	3	27H 55M	CS	FOI	-	-	M	2.9	7	9	-	-	-
38	9711	SUJATHA	19	PRIMI	38^{+2}	SPE	4	23H 30M	CS	FOI	-	-	F	3.2	7	9	-	-	-
39	10466	SANGEETA	20	G2A1	37^{+5}	GHTN	3	8H 30M	VD	-	-	v	M	2.5	7	9	-	-	-
40	7108	VAISHALI	19	PRIMI	37^{+2}	SPE	4	6H 20M	VD	-	+	-	M	2.2	7	9	-	-	-
41	8729	REKHA	30	G3P2L2	36^{+3}	SPE	3	4H 15M	VD	-	-	HS	M	1.6	2	5	-	LBW	7
42	7391	SHAHIN	20	PRIMI	37^{+5}	GHTN	4	6H 10M	CS	FD	-	-	M	2.9	7	9	-	-	-
43	7904	HARIMA	23	G3P2L1D1	37	GHTN	3	5H 30M	VD	-	-	-	F	2.2	7	9	-	-	-
44	8426	BHAGHYASHREE	21	PRIMI	38	SPE	3	6H	VD	-	-	-	M	2.7	7	9	-	-	-
45	11046	LAXMI	19	PRIMI	39	MPE	3	7H 25M	VD	-	-	-	M	2.8	7	9	-	-	-
46	11804	BASAMMA	20	PRIMI	39^{+6}	MPE	4	7H	VD	-	-	-	M	2.5	7	9	-	-	-
47	11264	VANISHREE	19	PRIMI	37+4	SPE	4	4H	CS	FD	-	C	M	2.5	7	9	-	-	-
48	12382	VEENA	22	PRIMI	38^{+5}	GHTN	3	1H 10M	CS	FD	-	-	F	2.4	7	9	THICK	-	-
49	12829	SHREEDEVI	21	PRIMI	40	MPE	3	16H	CS	FD	-	-	F	2.9	7	9	-	-	-
50	13137	SUJATA	30	G3P2L2	39	SPE	3	8H 50M	VD	-	-	-	F	2.5	7	9	-	-	-

51	13557	SANGITA	27	G2P1L1	39	SPE	3	16H 50M	VD	-	-	-	F	2.2	7	9	-	-	-
52	12816	SNEHA	22	PRIMI	37^{+1}	GHTN	3	5H 14M	VD	-	-	-	M	2.2	7	9	-	-	-
53	5366	VAISHALI	20	PRIMI	40^{+2}	GHTN	4	4H 30M	VD	-	-	TPH	F	2.6	7	9	-	-	-
54	5647	RAZWANI	20	PRIMI	40^{+2}	PD	3	10H	VD	-	+	-	M	2.7	7	9	-	-	-
55	5784	HAZARATHBEE	25	PRIMI	38^{+3}	SPE	3	3H 25M	VD	-	-	HS	M	2.8	7	9	-	-	-
56	5937	GIRIJA	20	PRIMI	37^{+2}	MPE	3	6H 45M	VD	-	-	-	F	2.6	6	8	-	-	-
57	14753	AFRINBANU	22	PRIMI	39+2	SPE	4	3H 30M	VD	-	-	-	F	2.5	7	9	-	-	-
58	13968	DANAMMA	22	PRIMI	34^{+4}	SPE	3	8H 30M	VD	-	-	-	M	1.6	5	7	-	-	-
59	14998	RESHMA	22	PRIMI	40^{+2}	GHTN	3	15H 30M	VD	-	-	-	F	3	7	9	-	-	-
60	15336	SHIVALEELA	23	G2P1L1	37^{+1}	SPE	4	3H 20M	VD	-	-	TS	F	2.1	7	9	-	-	-
61	15800	MAHADEVI	19	PRIMI	40	MPE	3	14H 5M	VD	-	+	-	F	3.1	7	9	-	-	-
62	16172	BHAGHYASHREE	22	PRIMI	37^{+5}	GHTN	4	5H 50M	VD	-	-	-	M	3.1	5	6	-	-	-
63	16537	GEETA	22	PRIMI	35^{+3}	IE	3	6H 50M	VD	-	-	C	F	1.7	7	9	-	VLBW	14
64	18549	GODAVARI	23	G2P1L1	41^{+2}	PD	4	6H 5M	VD	-	-	-	F	2.8	7	9	-	-	-
65	19429	PRABHAVATI	21	PRIMI	39^{+6}	SPE	3	5H 55M	VD	-	-	-	F	2.8	7	9	-	-	-
66	19766	ARCHANA	22	G4P2L2A1	37^{+3}	SPE	4	2H 47M	VD	-	-	HS	M	3.2	7	9	-	-	-
67	19753	VIJAYALAXMI	23	PRIMI	37^{+2}	MPE	4	6H 15M	VD	-	-	-	F	2.6	7	9	-	-	-
68	32986	IRAMMA	21	G2P1L1	38^{+1}	SPE	4	4H 5M	VD	-	-	-	M	3.4	7	9	-	-	-
69	35441	RUBINA	22	PRIMI	38^{+3}	MPE	3	7H 20M	VD	-	-	-	F	1.8	7	9	-	VLBW	15
70	37580	GEETA	20	G4A3	37^{+5}	MPE	3	8H 40M	VD	-	-	C	M	2.6	7	9	-	-	-
71	38006	BHAGHYA	20	PRIMI	37^{+2}	MPE	4	5H 25M	VD	-	-	C	F	2.6	7	9	-	-	-
72	28758	SANGEETA	20	G2P1L1	37	GHTN	3	8H 50M	VD	-	-	-	F	2.3	7	9	-	-	-
73	32588	SUREKHA	24	PRIMI	39	PD	4	6H 58M	VD	-	-	-	F	2.5	7	9	-	-	-
74	33257	SAVITHRI	22	G4P3L3	38	PD	4	5H 25M	VD	-	-	-	M	2.3	7	9	-	-	-
75	33926	SUPRIYA	26	G4P3L3	40^{+3}	GHTN	4	7H 44M	VD	-	-	C	M	2.8	7	9	-	-	-