Effect of Intramuscular Administration of Dexamethasone on the Duration of Labor in Full-Term Primigravida

Prof. Mohamed Samir Fouad, Dr. Mohamed Mohamed Al Khouly, Moustafa Mahmoud Taha Mohamed

Obstetrics and Gynecology Department, Faculty of Medicine Al-Azhar University, Egypt
mstf92@yahoo.com

Abstract: Objectives: to evaluate the effect of dexamethasone on labor duration and to establish whether dexamethasone plays a role in shorting the duration interval between initiation of labor induction and beginning of the active phase of labor in primigravida full-term pregnancy. **Study design:** Case control study included 200 primigravidae with full-term pregnancy classified into two groups: group I (cases) included 100 women assigned to receive a single 8-mg dose of dexamethasone intra-muscular and group II (control) included 100 women will not receive dexamethasone or any other cervical ripening agent. **Results:** The interval between initiation of labor induction and beginning of active phase of labor was shorter in the dexamethasone than in the control group **(2.54±0.94 hours vs. 3.59±0.86 hours; p=0.001).** Dexamethasone group shows shorter duration of active phase of labor than control group **(4.82±0.56 hrs. vs. 5.12±0.58 hrs.)**. Dexamethasone group shows shorter duration of first stage of labor than control group **(7.35±1.15 hrs. vs. 8.69±1.09 hrs.)**. Dexamethasone group shows faster rate of cervical dilatation than control group **(1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.)**. Dexamethasone group shows shorter duration of second stage of labor than control group **(25.09±12.99 minutes vs. 30.73±12.96 minutes).** Oxytocin requirement in dexamethasone group was less than in control group **(5.35±1.49 hrs. vs. 5.97±1.34 hrs.).** **Conclusions**: The administration of dexamethasone found to shorten labor duration.
[Mohamed Samir Fouad,. Mohamed Mohamed Al Khouly, Moustafa Mahmoud Taha Mohamed. **Effect of Intramuscular Administration of Dexamethasone on the Duration of Labor in Full-Term Primigravida.** *N Y Sci J* 2017;10(9):1-12]. ISSN 1554-0200 (print); ISSN 2375-723X (online). http://www.sciencepub.net/newyork. 1. doi:10.7537/marsnys100917.01.

Keywords: Dexamethasone; post-term pregnancy; induction of labor.

1. Introduction

Although administrating corticosteroids is a suggested method to shorten labor duration, the role of these agents in the process of labor is not well understood ***(Kavanagh et al., 2006)***. Several animal studies have shown the importance of corticosteroid secretion by the fetal adrenal glands on the beginning of labor ***(Kavanagh et al, 2006, Wood, Keller-Wood et al., 1991)***, and infusing glucocorticoids in the lamb fetus was also shown to induce preterm labor. These findings have led to the hypothesis that corticosteroids also had an effect on the labor of women ***(Kavanagh, Kelly, Thomas,2006, 2001).***

Different studies have shown the paracrine and autocrine effects of corticosteroids on the human uterus, and receptors for these agents have been detected on the human amniotic membrane ***(Kalantaridou Kavanagh Campbell,2007).***

Kalantaridou et al. (2007) have suggested that the corticotrophin-releasing hormone (CRH), which has been identified in various organ systems, including the female reproductive system, is the principal regulator of the hypothalamic–pituitary–adrenal axis. Circulating placental CRH is responsible for the physiologic hypercortisolism of the latter half of pregnancy and plays a role in the onset of labor. ***O'Sullivan et al. (2007)*** reported that a prolonged gestation is more likely to occur when the fetus has congenital adrenal hyperplasia caused by 21-hydroxylase deficiency, which may be due to an impaired cortisol production. All of these studies show the probable effects of corticosteroids on the labor process. Corticosteroids have been administered intravenously, intramuscularly, and by extra-amniotic infusion in various clinical trials ***(Barkai et al,2007, McLean et al.,2001).***

Induction of labor refers to the process of artificially initiating uterine contractions prior to their spontaneous onset to effects progressive effacement and dilatation of the cervix and ultimately, delivery of the baby ***(Hayman, 2010).***

Induction of labor is one of the most common interventions practiced in modern obstetrics. In the developed World, the ability to induce labor has contributed to the reduction in maternal and perinatal mortality and morbidity ***(Subramanian and Penna, 2009).***

The goal of labor induction is to stimulate uterine contractions before the spontaneous onset of labor, resulting in vaginal delivery. The benefits of labor induction must be weighed against the potential maternal and fetal risks associated with this procedure. When the benefits of expeditious delivery are greater than the risks of continuing the pregnancy, inducing

labor can be justified as a therapeutic intervention ***(Barclay, 2009).***

The success of induction and labor progression is dependent on the condition of the cervix before induction initiation ***(Barclay, 2009).***

In primigravidae, the mean time taken from induction to delivery is between 15 and 20 hours, of which up to 12 hours is spent in the cervical ripening phase before labor itself starts (***Stitely et al., 2000).***

About 10 percent of pregnancies may be prolonged. In general, the longer the truly post-term fetus stays in the uterus, the greater the risk of a severely compromised fetus and newborn infant. Therefore of major importance in handling compromised postdate pregnancies is the use of a suitable method of labor induction. A prolonged gestation is more likely to occur when the fetus has congenital adrenal hyperplasia caused by 2l-hydroxylase deficiency, which may be due to an impaired cortisol production ***(O'Sullivan et al., 2007).***

Glucocorticoids are now known to play key roles in fetal maturation for example in maturation of the lung in anticipation of extra-uterine life and in several species appear to be mediators in the initiation of labor. In humans, the placenta synthesizes CRH, and the exponential rise of this hormone in maternal plasma correlates with the timing of birth ***(Falah N et al., 2014).***

Glucocorticoids derived from the maturing fetal hypothalamus-pituitary-adrenal axis play a crucial role in triggering parturition ***(Challis et al., 2005).***

During pregnancy, large amounts of CRH are released from the placenta and fetal membranes. An increment in plasma CRH concentration occurs during spontaneous labor, with peak value at vaginal delivery ***(Riley & Challis, 2003).*** Placental CRH is also released into the fetal circulation, dehydroepiandrosterone and in vitro CRH directly stimulates sulfate (DHEA-S) production from the fetal zone of the fetal adrenal ***(Sirianni et al., 2005).***

This increase in fetal zone activity correlates with rising levels of maternal estrogen levels through the conversion of DHEA-S to estrogens within the placenta. The increase in the maternal estrogen to progesterone ratio may promote the expression of contraction-associated proteins in the myometrium, thus facilitating the initiation of parturition ***(Mastorakos and Ilias, 2003).***

Cortisol increases the production of prostaglandins in the fetal membranes by either up regulating prostaglandin synthesis (PGHS-2) levels or down regulating 15-hydroxy prostaglandin dehydrogenase (PGDH). It has been very well recognized that increased prostaglandin (PGE_2 and PGF_2) biosynthesis as a result of inflammation-like responses in intrauterine tissues is one of the key events leading to parturition in both term and preterm human labor because these compounds evoke uterine contractions as well as cervical softening and effacement.

2. Patient and Methods

This prospective clinical interventional randomized case-controlled trial was conducted at Al-Hussein university Hospital during the period from 2016 June to 2017 March.

Methodology (plan):

It included 200 participants whom are admitted for labor induction at Al-Hussein University Hospital.

The participants will be randomly assigned by computer list into Group I (Dexamethasone group) N=100 and Group II (Control group) N=100.

The participants of Group I will receive a prefilled syringe with two milliliters (8mg) of dexamethasone intra-muscular, and the participants of Group II will not receive dexamethasone or any other cervical ripening agent.

No cervical ripping agent will be used for induction of labor in either group.

After approval of health committee in Al-Hussein Hospital, a verbal consent was obtained from each candidate after explanation of the procedure in details.

Statistical Analysis:

All clinical and demographic data will be recorded on investigative report form. These data will be analyzed by IBM computer using SPSS (Statistical program for social science version 12) as follows:

Description of quantitative (numerical) variables will be performed in the form of mean, standard deviation (SD) and range.

Description of qualitative (categorical) variables will be performed in the form number of cases and percentage.

Chi-square test will be used to compare qualitative variables between groups.

Fisher exact test will be used instead of chi-square when one expected cell or more less than five.

Unpaired t-test will be used for comparison of quantities variables, in parametric data (SD<50%) of mean.

Paired t-test will be used to compare pre and post quantitative results in the same group.

***p*-value (level of significance):**

$p>0.05$= non-significant.

$p<0.05$= significant.

$P<0.001$= highly significant.

Data were graphically represented using Harvard Graphics program.

3. Results

Data were statistically described in terms of mean ± standard deviation (±SD), median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using Student *t* test for independent samples.

For comparing categorical data, Chi square (χ^2) test was performed. Exact test was used instead when the expected frequency is less than 5. *p*-values less than 0.05 was considered statistically significant.

All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) release 15 for **Microsoft Windows (2006).**

Table (1): Demographic characteristics of the patients (mean ± SD)

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Age (years)	26±4.36	25.63±3.79	0.624*	N.S.
BMI (Kg/m^2)	23.09±1.89	22.78±1.71	0.344*	N.S.
Gestational age on admission (weeks)	40±1.46	40±1.35	0.796*	N.S.

Values are mean ±S.D. & number (percentage) Student t-test*
S.D.: Standard Deviation; N.S.: Non-significant

There were non-significant statistical differences between the two studied groups as regard age, body mass index (BMI), gestational age.

Table (2): Statistical comparison between the two studied groups as regards pulse and blood pressure (vital signs)

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Pulse (bpm)	79.07±6.09	79.4±5.13	0.746*	N.S.
Systolic BP	118±14.59	118±13.38	1.000*	N.S.
Diastolic BP	72.5±8.31	73±8.69	0.748*	N.S.

Values are Mean ± S.D. *Student t-test
S.D.: Standard Deviation; N.S.: Non-significant; bpm: Beat per minutes; BP: Blood Pressure

There were non-significant statistical differences between the two groups as regard pulse and blood pressure (vital signs).

Table (3): Statistical comparison between the two studied groups as regards Bishop score at time of intervention

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Cervical dilatation (cm)	2.43±0.72	2.5±0.62	0.589*	N.S.
Effacement (%)	43.33±7.05	44±10.61	0.686*	N.S.
Consistency **Firm** **Intermediate** **Soft**	 4 (4%) 36 (36%) 60 (60%)	 6 (6%) 34 (34%) 60 (60%)	0.462**	N.S.
Position **Posterior** **Central** **Anterior**	 7 (7%) 53 (53%) 40 (40%)	 7 (7%) 53 (53%) 40 (40%)	0.930**	N.S.
Station of fetal head **-2** **-1** **0** **+1**	 26 (26%) 10 (10%) 54 (54%) 10 (10%)	 26(26%) 10 (10%) 54 (54%) 10 (10%)	0.094**	N.S.
Total Bishop score	7.63±0.66	7.63±0.71	1.000*	N.S.

Values are mean ± S.D. & number (%) *Student t-test
S.D.: Standard Deviation; **Chi-square test; N.S.: Non-significant

There were non-significant statistical differences between the two groups as regard cervical dilatation, effacement, cervical position, consistency, head station and total Bishop score.

Table (4): Statistical comparison between the two studied groups as regards duration between induction of labor and active phase

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Duration between induction of labor and active phase (hr.)	2.54±0.94	3.59±0.86	0.001*	H.S.

Values are mean ± SD *Student t-test S.D.: Standard Deviation; H.S.: Highly Significant

Dexamethasone group shows shorter duration between labor induction and active phase of labor than control group (2.54±0.94 hr. vs. 3.59±0.86 hr.).

There was **a high significant statistical difference** between the two studied groups as regards duration between labor induction and active phase of labor (*p* less than 0.001).

Table (5): Statistical comparison between the two studied groups as regards duration of active phase of labor

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Duration of active phase of labor (hrs.)	4.82±0.56	5.12±0.58	0.006*	S

Values are mean ±S.D. *Student t-test S.D.: Standard Deviation; S: Significant

Dexamethasone group shows shorter duration of active phase of labor than control group (4.82±0.56 hr. vs. 5.12±0.58 hr.).

There was **a significant statistical difference** between the two studied groups as regards duration of active phase of labor (*p* less than 0.05).

Table (6): Statistical comparison between the two studied groups as regards duration of 1st stage of labor

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Duration of 1st stage of labor (hr.)	7.35±1.15	8.69±1.09	0.001*	S

Values are mean ± SD *Student t-test S.D.: Standard Deviation; S: Significant

Dexamethasone group shows shorter duration of first stage of labor than control group (7.35±1.15 hr. vs. 8.69±1.09 hr.).

There was **a significant statistical difference** between the two studied groups as regards duration of first stage of labor (*p* less than 0.001).

Table (7): Statistical comparison between the two studied groups as regards rate of cervical dilatation

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Rate of cervical dilatation (cm/hour)	1.37±0.18	1.28±0.17	0.01*	S.

Values are mean ± S.D. *Student t-test S.D.: Standard Deviation; S.: Significant

Dexamethasone group shows faster rate of cervical dilatation than control group (1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.).

There was **a significant statistical difference** between the two studied groups as regards rate of cervical dilatation (*p* less than 0.05).

Table (8): Statistical comparison between the two studied groups as regards duration of 2nd stage of labor

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Duration of 2nd stage of labor (minutes)	25.09±12.99	30.73±12.96	0.032*	S

Values are mean ±SD. *Student t-test S.D.: Standard Deviation; S: Significant

Dexamethasone group shows shorter duration of second stage of labor than control group (25.09±12.99 minutes vs. 30.73±12.96 minutes).

There was **a significant statistical difference** between the two studied groups as regards duration of second stage of labor (*p* less than 0.05).

Table (9): Statistical comparison between the two studied groups as regards dose of oxytocin required

	Dexamethasone group	Control group	*p*-value	Sig.
Oxytocin requirement (hours)	5.35±1.49	5.97±1.34	0.019*	S

Values are mean ± S.D. *Student t-test S.D.: Standard Deviation; S: Significant

Oxytocin requirement in dexamethasone group was less than in control group (5.35±1.49 hours vs. 5.97±1.34hours).

There was **a significant statistical difference** between the two studied groups as regards dose of oxytocin required (*p* less than 0.05).

Table (10): Statistical comparison between the two studied groups as regards duration of 3rd stage of labor

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Duration of 3rd stage of labor (minutes)	8.57±3.63	9.52±2.99	0.155*	N.S.

Values are mean± SD & number Student t-test* S.D.: Standard Deviation; N.S.: Non-significant

There was no significant statistical difference detected between the two studied groups as regards duration of 3rd stage of labor.

Table (11): Statistical comparison between the two studied groups as regards mode of delivery and its indication

Mode of delivery and indication	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
SVD	83 (83%)	77 (77 %)	0.43**	N.S.
C.S. Failed induction Failure to progress Fetal distress Deep transverse arrest (direct occipito- transverse)	17 (17 %) 4 (23.5 %) 4 (23.5 %) 7 (41.1 %) 2(11.7 %)	23(23%) 8(34.7%) 5(21.7%) 8(34.7%) 2 (8.6 %)		

Values are numbers (percentage). **Chi-square test
N.S.: Non-significant; SVD: Spontaneous vaginal delivery; C.S.: Caesarean section

There was a non-significant statistical difference between the studied groups as regards mode of delivery.

Table (12): Statistical comparison between the two studied groups as regards neonatal outcome

	Dexamethasone group (n=100)	Control group (n=100)	*p*-value	Sig.
Birth weight (gm.)	3150±255.12	3190±271.34	0.407*	N.S.
Apgar score at 1 minute	7.23±0.69	7.1±0.66	0.283*	N.S.
Apgar score at 5 minutes	8.7±0.72	8.65±0.71	0.702*	N.S.
Fetal heart rate disturbance **Yes** **No**	 7 (7 %) 93 (93 %)	 6 (6 %) 94 (94 %)	 0.769**	 N.S.
Meconium-stained liquor **Yes** **No**	 5 (5 %) 95 (95 %)	 6 (6%) 94 (94 %)	 0.697**	 N.S.
Admission to NICU **Yes** **No**	 6 (6. %) 94 (94%)	 8 (8 %) 92 (92 %)		N.S.

Values are mean ± SD & number (%) *Student t-test. **Chi-square test
S.D.: Standard Deviation ; N.S.: Non-significant ; NICE: Neonatal intensive care unit

There were non-significant statistical difference between the two studied groups as regards birth weight, Apgar score at 1 minute, Apgar score at 5 minutes, fetal heart rate disturbance, meconium stained liquor & admission to NICU.

4. Discussion

It is well known that glucocorticoids accelerate lung maturation by enhancing surfactant synthesis in the pulmonary alveolar cells. Evidence has been obtained from early studies that the phospholipid content of surfactant provides a source of arachidonic acid that can be used by the amnion for prostaglandin synthesis. Recently there is direct evidence pointing to surfactant protein A (SP-A) as the key link between the maturing fetus and the initiation of parturition in the mouse ***(Montalbano et al., 2013).***

Glucocorticoids derived from the maturing fetal hypothalamus-pituitary-adrenal axis play a crucial role in, triggering parturition ***(Challis el al., 2005).***

In humans, the placenta synthesizes corticotrophin-releasing hormone (CRH), and the exponential rise of this hormone in maternal plasma correlates with the timing of birth ***(Smith et al., 2007).***

The corticotrophin-releasing hormone (CRH), which has been identified in various organ systems, including the female reproductive system, is the principal regulator of the hypothalamic-pituitary-adrenal axis. Circulating placental CRH is responsible for the physiologic hypercortisolism of the latter half of pregnancy and plays a role in the onset of labor ***(Kalantaridou et al., 2007).***

Cortisol increases the production of prostaglandins in the fetal membranes by either up regulating prostaglandin synthesis levels or down regulating 15-hydroxy prostaglandin dehydrogenase (PGDH) ***(Li et al., 2013).***

Therefore, glucocorticoids also play an important role in human parturition. In the fetal membranes, the actions of glucocorticoids are amplified by the actions of 11β-HSD steroid dehydrogenase type I (11β-HSD1), where 11β-HSD1 converts biologically inert cortisone to active cortisol thereby increasing the local levels of biologically active glucocorticoids. This cascade of events initiated by glucocorticoids may play an important role in the positive feed-forward mechanisms ***(Yang et al., 2007).***

This case controlled trial study was been conducted in the labor ward of Al-Hussein University Hospital to evaluate the effect of intramuscular dexamethasone administration on the duration of labor.

This study comprised 200 pregnant women with full term pregnancy, who admitted to the labor ward for induction of labor because of full-term pregnancy (gestational age ≥40 weeks).

Pregnant women were randomized (assigned) to receive dexamethasone sodium phosphate 8 mg (2 ml) or receive nothing or any other cervical ripening agent.

As regarding our results:

The study showed there were no significant statistical difference between the two studied groups regarding the mean maternal age (years), the gestational age (weeks) on admission, pulse (beat per minute) and blood pressure; No such difference was found regarding body mass index (BMI) and percentage of cesarean section between the two studied groups.

In addition, there were non-significant statistical differences between the two groups as regard primary Bishop score (cervical dilatation, effacement, cervical position, consistency, head station and total Bishop score).

The present study showed that a dexamethasone injection intramuscularly has suggested no significant difference between the 2 groups in the duration of the third stage of labor & the neonatal outcome (Birth weight, APGAR score at 1 minute and 5 minutes, number of cases with fetal heart rate disturbance, meconium stained liquor & neonatal admission to neonatal intensive care unit).

The first stage of labor was shorter in dexamethasone group than control group (7.35±1.15 hrs. vs. 8.69±1.09 hrs.) (p=0.001).

The second stage of labor was shorter in the dexamethasone group than in control group (25.09±12.99 minutes vs. 30.73± 12.96 minutes) (p=0.032). The interval between the initiation of labor induction and the beginning of the active phase of labor was 2.54±0.94 hours in the dexamethasone group and 3.59±0.86 hours in the control group, and the difference was significant (p-value less than 0.001).

The duration of active phase of labor was 4.82±0.56 hours in dexamethasone group and 5.12±0.58 hours in control group, and the difference was significant (p value less than 0.05).

The rate of cervical dilatation is faster in dexamethasone group than control group (1.37±0.18 cm/hr. vs. 1.28±0.17 cm/hr.), the difference was significant (p =0.01).

The mean oxytocin dose consumption on entering active phase was 5.35±1.49 units for dexamethasone group and 5.97±1.34 unites for control group and the difference was significant (p =0.019).

Our findings are in agreement with those observed by ***Maryam Kashanian et al., 2008*** who evaluated the effect of dexamethasone administration on labor duration. A controlled trial including 122 nulliparous women with a full-term pregnancy and a Bishop score of 7 or greater, were randomly assigned to receive a single 8 mg dose of dexamethasone for the case group or placebo for the control group 6 hours before initiation of labor induction.

They found that the interval between initiation of labor induction and beginning of the active phase of labor was shorter in the dexamethasone than in the control group. The duration of the second stage of labor was also shorter in the dexamethasone group.

They concluded that the administration of dexamethasone was found to shorten labor duration by decreasing the interval between the induction and the beginning of the active phase, with no observed maternal or neonatal complications (***Maryam Kashanian et al., 2008).***

Kashanian et al., 2008 reported on the extra-amniotic infusion of a saline solution mixed with dexamethasone through a Foley catheter whose balloon was filled with 15 ml of water, and concluded that the procedure could shorten the duration of labor without significant maternal or fetal risk.

O'Sullivan et al., 2007 concluded that fetuses with congenital adrenal hyperplasia due to 21-hydroxylase deficiency were more likely to have a prolonged gestation, and this may be due to impaired cortisol production.

Hajivandi L et al., 2013 performed clinical trial on 100 eligible nulliparous women in their 40 to 42 weeks of gestation in 2009 who were admitted to Amir Hospital in Ahvaz. For the case group, 8 mg dexamethasone was administered 12 hours before induction and the controls were given 2 ml of normal saline at the same intervals.

There was no significant difference between the two groups in terms of age, demographic characteristics, initial Bishop score, first and fifth minute Apgar score, and meconium difference. There was a significant difference between the two groups (p =0.001) concerning the mean-time interval between the induction and the onset of active phase in the case group (3.1±0.68 hours) and in the control group it was (4.2±1.3 hours). They concluded that intra-muscular dexamethasone reduces the time duration from the induction to the onset of labor phase ***(Hajivandi L et al., 2013).***

In another study, conducted by ***Ziaee et al., 2003,*** that aimed to determine the effect of intra-muscular injection of dexamethasone on induction of labor. Women in 41 weeks gestational age and Bishop score greater than or equal to 7 received intramuscular injections of 10 mg dexamethasone in two doses with 12 hours interval, and the next day, induction was carried out using oxytocin. These patients were compared with patients in similar conditions, but receiving oxytocin.

In this study, more of the patients from dexamethasone group entered active phase than that in control group, and interval between induction and onset of active phase was shorter in this group than in control group. They reported that intra-muscular injection of dexamethasone before labor induction reduced the time between the induction and the active phase of labor (***Ziaei S et al., 2003).***

In another study conducted by ***Barakai et al., 1997*** with the aim to investigate the effect of extra-amniotic normal saline with dexamethasone for induction of labor, the interval between induction and onset of active labor in dexamethasone group was shorter than that in the group that received extra-amniotic normal saline only.

Also, 90.25% of dexamethasone group entered active phase, and 88.37% of control group, but the difference was insignificant. Mean onset of oxytocin to delivery was 7.25±2.86 hours in the case group, and 9.76±3.91 hours in the control group, with a significant difference between the two groups (p =0.002). Results of this study showed that injection of extra-amniotic normal saline was a suitable and inexpensive method for cervical ripening and response to induction. The addition of dexamethasone could help to shorten delivery process and that inducing labor by means of an extra-amniotic infusion of corticosteroids through an intra-cervical Foley balloon catheter reduced the time between induction of labor and delivery. This may indicate a possible role for corticosteroids in the parturition process ***(Barakai et al., 1997).***

Liggins GC, 1968 found that ACTH infusion or cortisol into fetal sheep at more than 88 days of gestation causes parturition**.**

Elliot et al., 1995 showed that betamethazone administration in humans for fetal lung maturity in triplet and quadruplet births is associated with increase uterine contractions and preterm labor with cervical changes requiring tocolysis**.**

Mati et al., 1973 induced labor successfully in six post-date patients by giving 20 mg betamethazone into amniotic fluid. The mean time for onset of labor in the steroid group (67.4±24.3 hrs.) was shorter than the placebo-injected patients (312±142 hrs.), p less than 0.01. They concluded that it is clear that the betamethasone injections accelerated the onset of labor without any harmful effects on infants or mothers.

In contrast to our results, ***Kavanegh et al., 2001 & 2006*** in a review study on the effect of corticosteroids in cervical ripening and induction of labor concluded that, efficacy of corticosteroids in induction of labor was still unknown and required further studies. In 2006, they extended their studies, but arrived at the same conclusion.

References

1. Abiaka C and Machado L (2012): Nitric oxide and antioxidant enzymes in venous and cord blood of late preterm and term Omani mothers. Sultan Qaboos Univ Med J. 2012 Aug; 12(3): 300-5. Epub 2012 Jul 15.
2. ACOG American College of Obstetricians and Gynecologists (2009): ACOG Committee on Practice Bulletins Obstetrics. ACOG practice

bulletin No.l07, August 2009: induction of labor. Obstet Gynecol 2009, 114(2) Parts 1: 386-397.
3. Alexander JM, Mcintire DD and Leveno (2000): Forty weeks and beyond, pregnancy outcomes by weeks of gestation. Obstet Gynecol. 2000 Aug; 96(2): 291-4.
4. Al-Zirqi L, Vangen S, Forsen L, et al. (2009): Effects of onset of labor and mode of delivery on severe postpartum hemorrhage. Am J Obstet Gynecol. 2009 Sep; 201(3): 273.e1-9.
5. Bailit JL Gregory KD, Reddy UM, et al. (2010): Maternal and neonatal outcomes by labor onset type and gestational age. Am J Obstet Gynecol; 202: 245-257.
6. Barclay L (2009): The American College of Obstetricians and Gynecologists (ACOG) issued revised guidelines on when and how to induce labor in pregnant Women. The updated recommendations are published as a Practice Bulletin#107 "Induction of Labor," in the August issue of Obstetrics & Gynecology.
7. Barkai G, Cohen SB, Kees S, Lusky A, Margalit V, Mashiach S, Schiff E (1997): Induction of labor with use of a Foley catheter and extraamniotic corticosteroids. American journal of obstetrics and gynecology, 177(5):1145-1148.
8. Blackburn S (2010): The hypothalamic-pituitary-adrenal axis during pregnancy; J Perinat Neonatal Nurs. 2010 Jan-Mar; 24(1):10-1.
9. Blumenstein M, Hansen WR, Deval D, Mitchell MD (2000): Differential regulation in human amnion epithelial and fibroblast cells of prostaglandin E (2) production and prostaglandin H synthase-2 mRNA expression by dexamethasone but not tumor necrosis factor-alpha. Placenta 21: 210-217.
10. Bollapragada SS, MacKenzie F, and Norrie JD, et al. (2009): Randomized placebo-controlled trial of outpatient (at home) cervical ripening with isosorbidemononitrate (IMN) prior to induction of labor-clinical trial with analyses of efficacy and acceptability; The IMOP study. BJOG; 2009 Aug; 116(9): 1185-95.
11. Caughey AB, Snegovskikh VV, Norwitz ER (2008): Post-term pregnancy: how can we improve outcomes? Obstet Gynecol Surv. 2008 Nov; 63(11): 715- 24.
12. Caughey AB, Sundaram V, Kaimai AJ, et al. (2009): Maternal and neonatal outcomes of elective induction of labor. Evid Rep Technol Assess (Full Rep). 2009 Mar; (176): 1-257.
13. Caughey AB, Sundaram V, Kaimal AJ, Gienger A, Cheng YW, McDonald KM et al. (2009) b: Systematic review: elective induction of labor versus expectant management of pregnancy. Ann Intern Med. 2009; 151(4): 252.
14. Caughey AB, Stotland NE, Washington AE, Escobar GJ (2009) a: Who is at risk for prolonged and postterm pregnancy? Am J Obstet Gynecol. 2009 Jun; 200(6): 683.e1-5.
15. Cejvanovic V, Jimenez-Solem E, Poulsen HE, Andersen JT (2014): NSAID use during pregnancy: maternal characteristics and prescription patterns: nationwide cohort study. Scand J Rheumatol. 2014; 43(5): 424-6.
16. Challis JR, Bloomfield FH, Bocking AD, Casciani V, Chisaka H, Connor K, Li W (2005): Fetal signals and parturition. Journal of Obstetrics and Gynaecology Research, 31(6):492-499.
17. Chanrachakul B, Promsonthi P, Preechapornprasert D (2011): Nitric oxide donors for cervical ripening in first-trimester surgical abortion. Cochrane Database Syst Rev. 2011 Dec 7; (12): CD007444.
18. Chapman K, Holmes M, Seckl J (2013): 11?-hydroxysteroid dehydrogenases: intracellular gate-keepers of tissue glucocorticoid action. Physiol Rev. 2013 Jul; 93(3): 1139-206.
19. Chris J, Ronald S (2008): Danforth's Obstetrics and Gynecology tenth edition 2008, preterm and post term delivery Ch.11 P: 182, Arthur Haney, Ronald Gibbs, Beth Karlan, Ingrid Nygaard; Lippincott Williams & Wilkins.
20. Cunningham, FG. Leveno, KJ. Bloom, SL et al. (2010): Labor induction Ch. 22 P: 505; New York: McGraw-Hill. Williams's Obstetrics. 23rd.
21. Czock D, Keller F, Rasche FM, Haussler U (2005): Pharmacokinetics and pharmacodynamics of systemically administered glucocorticoids. Clin Pharmacokinet. 2005; 44(1): 61-98.
22. Dante G, Bellei G, Neri I, Facchinetti F (2014): Herbal therapies in pregnancy: what works? Curr Opin Obstet Gynecol. 2014 Apr; 26(2): 83-91.
23. Darney BG, Snowden JM, Cheng YW, Jacob L et al. (2013): Elective induction of labor at term compared with expectant management: maternal and neonatal outcomes. Obstet Gynecol. 2013 Oct; 122(4): 761-9.
24. Dean Leduc, Anne Biringer, Lily Lee, Vancouver BC, Jessica Dy (2013): Induction of labor 2013 Clinical Practice Guideline Society of Obstetrics and Gynecology of Canada (SOGC) No. 296, September 2013; J Obstet Gynaecol Can 2013; 35(9).
25. Delaney S, Shaffer BL, and Cheng YW, et al. (2010): Labor induction with a Foley balloon inflated to 30 mL compared with 60 ml: a randomized controlled trial. Obstet Gynecol. 2010 Jun; 115(6): 1239-45.
26. Delaneyn M, Roggensack A, Leduc DC and Ballermann C (2008): Guidelines for the Management of Pregnancy at 41 +0 to 42+0 Weeks; SOGC Clinical Practice Guideline. J Obstet Gynaecol Can. 2008 Sep; 30(9): 800-23.
27. Divon MY, Ferber A, Sanderson M et al. (2004): A functional definition of prolonged pregnancy based on daily fetal and neonatal mortality rates.

Ultrasound Obstet Gynecol. 2004 May; 23(5): 423-6.
28. Donald Briscoe, Hayled Nguyen (2005): Management of pregnancy beyond 40 weeks gestation. J Am Fam Physician. 2005 May 15; 71(10): 1935-1941.
29. Dubicke A, Fransson E, Centini G, Andersson E, Bystrom B, Malmstrom A (2010): Pro-inflammatory and anti-inflammatory cytokines in human preterm and term cervical ripening; J Reprod Immunol. 2010 Mar; 84(2): 176-85.
30. Ducarme G, Chesnoy V, Petit L (2015): Factors predicting unsuccessful labor induction with dinoprostone in post-term pregnancy with unfavorable cervix. J Gynecol Obstet Biol Reprod (Paris). 2015 Jan; 44(1): 28-33.
31. Elliott JP and Radin TG (1995): The effect of corticosteroid administration on uterine activity and preterm labor in high-order multiple gestations. Obstetrics & Gynecology, 85(2):250- 254.
32. Epelman M, Zuckerman-Levin N, Shen-Orr Z, Ben-David S (2007): Parturition itself is the basis for fetal adrenal involution; J Clin Endocrinol Metab. 2007 Jan; 92(1): 93-7.
33. Falah N, Haas DM (2014): Antenatal corticosteroid therapy: current strategies and identifying mediators and markers for response. Semin Perinatol. 2014 Dec; 38(8): 528-33.
34. FDA (U.S. Food and Drug Administration) (2010): Safety Labeling Changes Approved By FDA Center for Drug Evaluation and Research (CDER) – May 2010. Internet address: http: //www.fda.gov/Safety/MedWatch/SafetyInformation/ucm214912.htm.
35. Feinshtein V, Ben-Zvi Z, Sheiner E, Amash A (2010): Progesterone levels in cesarean and normal delivered term placentas. Arch Gynecol Obstet 2010; 281: 387-392.
36. Gagnon-Gervais K, Bujold E, Iglesias MH, Duperron L et al. (2012): Early vs. late amniotomy for labor induction: A randomized controlled trial. J Matern Fetal Neonatal Med. 2012 Nov; 25(11): 2326-9.
37. Galal M, Symonds I, Murray H et al. (2012): Post term pregnancy. Facts Views Vis Obgyn 2012; 4(3): 175-87.
38. Gomez-Lopez N, St Louis D, Lehr MA, Sanchez-Rodriguez EN2, Arenas-Hernandez M (2014): Immune cells in term and preterm labor. Cellular & Molecular Immunology advance online publication 23 June 2014.
39. Gulmezoglu AM, Crowther CA, Middleton P, Heatley E (2012): Induction of labour for improving birth outcomes for women at or beyond term. Cochrane Database Syst Rev. 2012; 6: CD004945.
40. Hajivandi L, Montazeri S, Iravani M, and Dawoodi M, Haghighizade MH (2013): Effect of intramuscular dexamethasone on onset of labor in postdates pregnancy. Journal of Babol University of Medical Sciences, 2013, 15(3), 24.
41. Harper TC, Coeytaux RR, Chen W, et al. (2006): A randomized controlled trial of acupuncture for initiation of labor in nulliparous women. Journal of Maternal- Fetal and Neonatal Medicine; l9 (8): 465-70.
42. Hayman R (2010): obstetrics and gynecology An evidence - based text for MRCOG second edition edited by David M Luesley and Philip N Baker chapter 25 induction of labor; 241-254.
43. Heinemann J, Gillen G, and Sanchez-Ramos L, et al. (2008): Do mechanical methods of cervical ripening increase infectious morbidity? A systematic review; Am J Obstet Gynecol. 2008 Aug; 199(2): 177-87.
44. Hertelendy F and Zakar T (2004): Prostaglandins and the myometrium and cervix; Prostaglandins Leukot Essent Fatty Acids. 2004 Feb; 70(2): 207-22.
45. Hill MJ, McWilliams GD, Garcia-Sur D, and Chen B, et al. (2008): The effect of membrane sweeping on prelabor rupture of membranes: a randomized controlled trial. Obstet Gynecol. 2008 Jun; 111(6): 1313-9.
46. Hofmeyr GJ, Gülmezoglu AM, Pileggi C. (2010): Vaginal misoprostol for cervical ripening and induction of labor; Cochrane Database Syst Rev. 2010 Oct 6; (10): CD000941.
47. Horton JS, Yamamoto SY, Bryant-Greenwood GD (2011): Relaxin modulates proinflammatory cytokine secretion from human decidual macrophages. Biol Reprod. 2011 Oct; 85(4): 788-97.
48. Iliodromiti Z, Antonakopoulos N, Sifakis S, Tsikouras P, Daniilidis A (2012): Endocrine, paracrine, and autocrine placental mediators in labor; Hormones, 2012 Oct-Dec; 11(4): 397-409.
49. Ishimoto H, Jaffe RB (2011): Development and function of the human fetal adrenal cortex: a key component in the feto-placental unit. Endocr Rev. 2011 Jun; 32(3): 317-55.
50. James A (2011): NMS Obstetrics and Gynecology7th edition; Post term Pregnancy Chapter 14. P: 152- 155, by Samantha M. Pfeifer MD, Lippincott Williams & Wilkins.
51. Jozwiak M, Bloemenkamp KW, Kelly AJ, Mol BW, Irion O, et al. (2012): Mechanical methods for induction of labor. Cochrane Database Syst Rev. 2012 Mar 14; 3: CD001233.
52. Kalantaridou S, Makrigiannakis A, Zoumakis E, and Chrousos GP (2007): Peripheral corticotrophin-releasing hormone is produced in the immune and reproductive systems: actions, potential roles and clinical implications. Front Biosci 2007; 12: 572—80.
53. Kalantaridou S, Makrigiannakis A, Zoumakis E, Chrousos GP. (2007): Peripheral corticotrophin-releasing hormone is produced in the immune and

reproductive systems: actions, potential roles and clinical implications. Front Biosci;12:572—80.
54. Karjane NW, Brock EL, Walsh SW (2006): Induction of labor using a Foley balloon, with and without extra-amniotic saline infusion; Obstet Gynecol. 2006 Feb; 107(2 Pt. 1): 234-9.
55. Kashanian M, Fekrat M, Zarrin Z, Ansari NS (2008): A comparison between the effect of oxytocin only and oxtocin plus propranolol on the labor (a double blind randomized trial). Journal of Obstetrics and Gynaecology Research, 34(3):354-358.
56. Kavanagh J, Kelly AJ, Thomas J (2001): Corticosteroids for induction of labor. Cochrane Database Syst Rev; (2): CD003100.
57. Kavanagh J, Kelly AJ, Thomas J (2001): Sexual intercourse for cervical ripening and induction of labor; Cochrane Database Syst. Rev. 2001; (2): CD003093.
58. Kavanagh J, Kelly AJ, Thomas J (2006): Corticosteroids for cervical ripening and induction of labor. Cochrane Database Syst Rev2006 (2): CD003100.
59. Kavanagh J, Kelly AJ, Thomas J (2006): Corticosteroids for cervical ripening and induction of labor. Cochrane Database Syst Rev; (2): CD003100.
60. Kavanagh J, Kelly AJ, Thomas J (2005): Breast stimulation for cervical ripening and induction of labor. Cochrane Database Syst. Rev. 2005 Jul 20; (3): CD003392.
61. Kelly AJ, Kavanagh J, Thomas J (2013) a: Castor oil, bath and/or enema for cervical priming and induction of labor. Cochrane Database Syst Rev. 2013 Jul 24; 7: CD003099.
62. Kelly AJ, Thomas J, Fairclough A, Kavanagh J (2014): Vaginal prostaglandin (PGE2 and PGF2α) for induction of labor at term. Cochrane Database Syst Rev. 2014 Jun 19; 6: CD003101.
63. Kelly E. Ruhstaller and Anthony C. Sciscione (2012): Maternal-Fetal Evidence-Based Guidelines 2nd edition; Edited by Vincenzo Berghella; Part III: Pregnancy complications of labor; C: Special labor issues; Induction of labor; 18, 156. Published: March 13, 2012 by CRC Press Ch. 4 basic concept in endocrinology P: 94; edited by Chard T and Litford R. 4th edition; Springer Verlag, London.
64. Kim SF (2014): The nitric oxide-mediated regulation of prostaglandin signaling in medicine. Vitam Horm, 2014; 96: 211-45.
65. Kistin SJ, Newman AD (2007): Induction of labor with homeopathy: a case report. J Midwifery Womens Health. 2007 May-Jun; 52(3): 303-7.
66. Klimkova M, Parizek A, Velikova M, Hill M, Paskova A (2010): Progesterone neuroactive metabolites in human pregnancy, Ceska Gynekol. 2010 Feb; 75(1): 9-15.
67. Konopka CK, Morais EN, Naidon D, Pereira AM (2013): Maternal serum progesterone, estradiol and estriol levels in successful dinoprostone-induced labor. Braz J Med Biol Res. 2013 Jan; 46(1): 91-7. Epub 2013 Jan 11.
68. Kota SK, Gayatri K, Jammula S, Kota SK, Krishna SV, Meher LK, Modi KD (2013): Endocrinology of parturition; Indian J Endocrinol Metab. 2013 Jan; 17(1): 50-9.
69. Lee SM, Park JW, Park CW, Yoon BH (2012): "Early rupture of membranes" during induced labor as a risk factor for cesarean delivery in term nulliparas. PLoS One. 2012; 7(6): e39883.
70. Li Y, He P, Sun Q, Liu J, Gao L et al. (2013): Reduced expression of 15-hydroxy prostaglandin dehydrogenase in chorion during labor is associated with decreased PRB and increased PRA and GR expression; Am J Pathol. 2013 May, 182(5): 1585-94.
71. Liggins GC (1968): Premature parturition after infusion of corticotrophin or cortisol into foetal lambs. J Endocrinol 42:323–329 - See more at: http://press.endocrine.org/doi/full/10.1210/endo.
72. Lim CE, Ng RW, Xu K (2013): Non-hormonal methods for induction of labour. Curr Opin Obstet Gynecol. 2013 Dec; 25(6): 441-7.
73. Lin MG, Reid KJ and Treaster MR, et al. (2007): Trans-cervical Foley catheter with and Without extra-amniotic saline infusion for labor induction: a randomized controlled trial. Obstet Gynecol. 2007 Sep; 110(3): 558-65.
74. Macones GA, Hankins GDV, Spong CY, et al. (2008): The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring; J Obstet Gynecol Neonatal Nurs. 2008 Sep-Oct; 37(5): 510-5.
75. Maslovit ZS, Lessing JB, Many A (2010): Complications of trans-cervical Foley catheter for labor induction among 1,083 Women. Arch Gynecol Obstet. 2010 Mar; 281(3): 473-7.
76. Mastorakos G and Ilias I (2003): Maternal and fetal hypothalamic-pituitary-adrenal axes during pregnancy and postpartum. Ann N Y Acad Sci. 2003 Nov; 997: 136-49.
77. Mati JKG, Horrobin DF, Bramley PS (1973): Induction of labour in sheep and in humans by single doses of corticosteroids. Br Med J, 2(5859): 149-151.
78. McGill J and Shetty A (2007): Mifepristone and misoprostol in the induction of labor at term. Int J Gynaecol Obstet. 2007 Feb; 96(2): 80-4. Epub 2007 Feb 1.
79. McKay LI and Cidlowski J (1999): Molecular control of immune/ inflammatory responses: Interactions between nuclear factor-K8 and steroid receptor signaling pathways. Endocr Rev. 1999 Aug; 20(4): 435-59.
80. McLean M, Smith R. (2001): Corticotrophin-releasing hormone and human parturition. Reproduction;121(4):493—501.

81. Menticoglou SM and Hall PF (2002): Routine induction of labour at 41 weeks gestation: nonsensus consensus. BJOG; 2002; 109(5): 485.
82. Mesiano S, Wang Y, Norwitz ER (2011): Progesterone receptors in the human pregnancy uterus: do they hold the key to birth timing? Reprod Sci. 2011 Jan; 18(1): 6-19.
83. Moisan MP, Minni AM, Dominguez G, Helbling JC, Foury A, Henkous N, Dorey R, Béracochéa D (2014): Role of corticosteroid binding globulin in the fast actions of glucocorticoids on the brain; 2014 Mar; 81: 109-15.
84. Monen L, Hassaart TH, Kuppens SM (2014): The etiology of meconium-stained amniotic fluid: Pathologic hypoxia or physiologic fetal ripening? Early Human Dev. 2014; 90: 325-8.
85. Montalbano AP, Hawgood S, Mendelson CR (2012): Mice deficient in surfactant protein A (SP-A) and SP-D or in TLR2 manifest delayed parturition and decreased expression of inflammatory and contractile genes. Endocrinology, 154(1):483-498.
86. Morken NH, Melve KK, Skjaerven R (2011): Recurrence of prolonged and post-term gestational age across generations: maternal and paternal contribution.BJOG. 2011 Dec; 118(13): 1630-5.
87. Myatt L and Sun K (2010): Role of fetal membranes in signaling of fetal maturation and parturition; Int J Dev Biol. 2010; 54(2-3): 545-53.
88. Myers ER, Blumrick R, Christian AL et al. (2002): Management of prolonged pregnancy. Evid Rep Technol Assess (Summ). 2002 Mar; (53): 1-6.
89. Neilson JP and Hapangama D (2009): Mifepristone for induction of labor. Cochrane Database Syst Rev. 2009 Jul 8; (3): CD002865.
90. NICE INDUCTION OF LABOR 2008 clinical guideline (2008) b: "Induction of labor"; National Institute for Health and Clinical Excellence; RCOG Press, London. Chapter 4: Induction of labor in specific circumstances; 24-44.
91. NICE INDUCTION OF LABOR 2008 clinical guideline (2008) c: "Induction of labor". National Institute for Health and Clinical Excellence; RCOG Press, London. Chapter 5: Methods of induction of labor; 45-68.
92. NICE INTRAPARTUM CARE (2007) National Collaborating Centre for Women's and Children's Health clinical guideline (2007): Intra-partum Care. London: RCOG Press; Revised reprint 2008; 199.
93. Oberg AS, Frisell T, Svensson AC, Iliadou AN (2013): Maternal and Fetal Genetic Contributions to Post term Birth: Familial Clustering in a Population-Based Sample of 475,429 Swedish Births. Am J Epidemiol 2013 Mar 15; 177(6): 531-7.
94. Olagbuji BN, Okonofua F, Ande AB (2012): Uterine rupture and risk factors for caesarean delivery following induced labor in women with one previous lower segment caesarean section. J Matern Fetal Neonatal Med. 2012 Oct; 25(10): 1970-4.
95. O'Sullivan J, Iyer S, and Taylor N, Cheetham T (2007): Congenital adrenal hyperplasia due to 21-hydroxylase deficiency is associated with a prolonged gestational age. Arch Dis Child 2007; 92(8): 690—2.
96. Park KH, Kim SN, Lee SY, and Jeong EH et al. (2011): Comparison between sonographic cervical length and Bishop score in preinduction cervical assessment: a randomized trial. Ultrasound Obstet Gynecol. 2011 Aug; 38(2): 198-204.
97. Petraglia F,Imperatore A, Challis JR (2010): Neuroendocrine mechanisms in pregnancy and parturition. Endocr Rev. 2010 Dec; 31(6): 783-816.
98. Ramirez, MM (2011): Induction of labor: A Review of Current Methods Obstet Gynecol Clin North Am. 2011 Jun; 38(2): 215-25, ix.
99. Rang H, Dale M, Ritter M and Flower J (2012): Rang and Dale's pharmacology 7th edition; chapter 32; The Pituitary and adrenal cortex; Edinburgh; New York: Elsevier/Churchill Livingstone, 2012.
100. Ribarac-Stepic N, Djurica S, Zakula Z, Koricanac G, Milosevic DP (2005): Molecular basis of glucocorticoid action. Srp Arh Celok Lek; 2005 Oct; 133 Suppl 1: 61-6.
101. Sciscione AC (2014): Methods of cervical ripening and labor induction: mechanical. Clin Obstet Gynecol. 2014 Jun; 57(2): 369-76.
102. Shatz L, Novack L, Mazor M, Weisel RB, Dukler D et al. (2013): Induction of labor after a prior cesarean delivery: lessons from a population-based study. J Perinat Med. 2013 Mar; 41(2): 171-9.
103. Sherman D. Vaknin Z, Kurzweil Y (2010): Foley catheter balloon vs. locally applied prostaglandins for cervical ripening and labor induction: a systematic review and meta-analysis. Am J Obstet Gynecol. 2010 Nov; 203(5): 418-29.
104. Shetty A, Burt R, Templeton A (2005): Women's perceptions, expectations and satisfaction with induced labor a questionnaire-based study. Eur J Obstet Gynecol Reprod Biol. 2005 Nov 1; 123(1): 56-61.
105. Shetty A1, Burt R, Rice P, Templeton A.2005 Women's perceptions, expectations and satisfaction with induced labour--a questionnaire-based study.2005 Nov 1;123(1):56-61.
106. Shripad hebbar (2009): Obstetrics & Gynecology for postgraduates Vol1 3rd Ed, prolonged pregnancy Ch. 20, P: 246, Arulkumaran, Sabaratnam, Gopalan, Sarala & Pratap Kumar et al (2009), Universities Press India.
107. Sifakis S, Angelakis E, Avgoustinakis E, et al. (2007): A randomized comparison between intravaginal misopristol and prostaglandin E2 for labor induction. Arch Gynecol Obstet. 2007 Apr; 275(4): 263-7.
108. Sirianni, M.; Jee, M. J.; Ben? tez, N.; Blakeslee, J. P.; Martel, A. R.; Meurer, G.; Clampin,... AL

(UCO/Lick Observatory, University of California, Santa Cruz, CA 95064.). 1049-1112. Publication Date: 10/2005. Origin: UCP. Astronomy Keywords.
109. Smith R (2007): Parturition. N Engl J Med. 2007 Jan 18; 356(3): 271-83.
110. Stupar ZT, Mikić AN, Bogavac M, Milatović S, Sekulić S (2013): Prediction of labor induction outcome using different clinical parameters; Srp Arh Celok Lek. 2013 Nov-Dec; 141(11-12): 770-4.
111. Subramanian and Penna D (2009): Best Practice in Labor and Delivery, 1st ed. Published by Cambridge University Press: Induction of labor; Edited by Warren R. and. Arulkumaran S. Cambridge University Press, Chapter 18; 195-205.
112. Tan PC, Vallikkannu N, Suguna S, et al. (2007): Transvaginal sonographic measurement of cervical length vs. bishop score in labor induction at term tolerability and predication of caesarean delivery. Ultrasound Obstet Gynecol. 2007 May; 29(5): 568-73.
113. Tobler A, Meier R, and Seitz M, et al. (1992): Glucocorticoids down-regulate gene expression of GSM-CSFNAP1 IL-8, and IL-6, but not M-CSF, in human fibroblasts. Blood, 1992 Jan 1; 79(1): 45-51.
114. Tsotakos N, Silveyra P, Lin Z, Thomas N, Vaid M, Floros J (2015): Regulation of translation by upstream translation initiation codons of surfactant protein A1 splice variants. Am J Physiol Lung Cell Mol Physiol. 2015 Jan 1; 308(1): L58-75.
115. Vaisanen-Tommiska M, Nuutila M, Ylikorkala O (2004): Cervical nitric oxide release in women postterm. Obstet Gynecol. 2004 Apr; 103(4): 657-62.
116. Valha P, Zmrhal J, Feyereisl J. (2010): Non-steroidal anti-inflammatory drugs in pregnancy; Ceska Gynekol. 2010 Feb; 75(1): 69-72.
117. Vayssière C, Haumonte JB, Chantry A, Coatleven F et al. (2013): Prolonged and post-term pregnancies: guidelines for clinical practice from the French College of Gynecologists and Obstetricians (CNGOF) 2013. Eur J Obstet Gynecol Reprod Biol. 2013 Jul; 169(1): 10-6.
118. Voltolini C and Petraglia F (2014): Neuroendocrinology of pregnancy and parturition; Handb Clin Neurol. 2014; 124: 17-36.
119. Vrachnis N, Malamas FM, Sifakis S, Tsikouras P, Iliodromiti Z (2012): Immune aspects and myometrial actions of progesterone and CRH in labor. Clin Dev Immunol; 2012: 937618.
120. Walfisch A, Mei-Dan E, Hallak M (2014): Trans-cervical double balloon catheter with and without extra-amniotic saline infusion for cervical ripening: a prospective quasi-randomized trial. J Matern Fetal Neonatal Med. 2014 Jul 11: 1-6.
121. Wood CE, Keller-Wood M. (1991): Induction of parturition by cortisol: effects on negative feedback sensitivity and plasma CRH. J Dev Physiol;16(5):287—92.
122. Yang Z, Guo C, Zhu P, Li W, Myatt L, Sun K (2007): Role of glucocorticoid receptor and CCAAT/enhancer-binding protein-? in the feedforward induction of 11?-hydroxysteroid dehydrogenase type 1 expression by cortisol in human amnion fibroblasts. J. Endocrinol.; 195:241–253.
123. Yount SM, Lassiter N (2013): The pharmacology of prostaglandins for induction of labor J Midwifery Women's Health. 2013 Mar-Apr; 58(2): 133-44; quiz 238-9.
124. Ziaei S, Rosebehani N, Kazeminejad A, Zafarghandi S (2003): The effects of intramuscular administration of corticosteroids on the induction of parturition. J Perinat Med;31(2):134-9.

8/12/2017