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AOGD BULLETIN

Women's Health to a New Horizon

Theme:
"Labor: Interim pain.... joy forever!"

AOGD SECRETARIAT

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Contents

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Events Held

- AOGD was taken over by Team Maulana Azad Medical College at ESI Hospital Basai Darapur on 1st April, 2016 - Dr Sudha Prasad as President and Dr Anjali Tempe as Vice President.
- Monthly Clinical Meeting was held at ESI Hospital Basai Darapur on 1st April, 2016. Interesting cases were discussed.
- CME on "Controversies in Obstetrics" organized by Deen Dayal Upadhyay Hospital, New Delhi on 8th April, 2016.
- 3rd GOAL Conference, 'Controversies to Consensus in Obstetrics & Gynecology' organized by ICOG and FOGSI on 9th -10th April, 2016 at Delhi.
- CME under aegis of Reproductive Endocrinology Subcommittee AOGD and DGF North on 12th April, 2016 at Apollo Spectra Hospitals.
- CME was organized by South Delhi Forum at Vikram Hotel with Baishakhi Cerebrations on 13th April, 2016.
- CME on Common Gynaecological Problems was organized by Max Super Speciality Hospital, Saket on 24th April, 2016.
- Monthly Clinical Meeting was held in the Auditorium of Indraprastha Apollo Hospital on 29th April. Interesting cases were discussed.

Taking over of AOGD by President
Dr Sudha Prasad on 1st April, 2016

Taking over of AOGD by Vice President
Dr Anjali Tempe on 1st April, 2016


CME at DDU Hospital on 8th April, 2016


CME at DDU Hospital on 8th April, 2016


CME at DDU Hospital on 8th April, 2016


CME by South Delhi Forum on 13th April, 2016


CME by South Delhi Forum on 13th April, 2016


First Executive Meeting on 16th April, 2016


First Executive Meeting on 16th April, 2016

Message from the President

Dear Members,

It gives my immense pleasure to welcome you all on behalf of AOGD MAMC team. Improvement of maternal health is a crucial challenge. Quality Antenatal Care is the keystone to improved maternal health. The theme for 2016 - 17 is *Women's health to a new horizon*. I, along with my team at Maulana Azad Medical College take this challenge to equip the obstetricians & gynaecologists with appropriate knowledge and clinical skills on subjects relevant to contemporary practice and to promote preventive and therapeutic health services to all women.

All women deserve access to healthcare that makes their existence more fulfilling and promising. An integral component of women empowerment is good health and that is my endeavour for the year. I look forward to your support in making the year ahead a grand success.

We all will be celebrating the joy of Motherhood this Mothers' Day on 8th May. To commemorate this, our first issue is dedicated to Labor as it is Labor which redefines woman in form of a mother.

Every Mother Counts!

Dr Sudha Prasad
President
drsprasad@yahoo.com

From the Secretary's Desk

Dear Members,

Namaskar! Greetings from Maulana Azad Medical College & Lok Nayak Hospital.

I along with my joint secretaries Dr Poonam Sachdeva, Dr Poonam Kashyap and Dr Niharika Dhiman welcome you all on this journey of AOGD.

I am privileged and greatly honored to be grateful to our President Dr Sudha Prasad, who has entrusted me this responsibility of bridging the gap between AOGD and you. In this direction, we have started SMS and emails services to apprise the members about our activities at regular interval. The website www.aogd.org has been made more vibrant, attractive and interactive and members can give their suggestions or feedback at the home page itself. We are also calling the members to update their contact details. I request all of you to cooperate in providing the details like contact number, email and postal address. You can even search 'facebook' for **AOGD MAMC** and like it to connect with all members.

The chairpersons of various subcommittees were nominated in April, 2016. I invite active participation from all our AOGD members. Any group of gynecologists / social forum interested to organize or participate in outreach activities or mass education programmes can coordinate with AOGD after following a small and short procedure.

Dear members, I request you to stay connected and block 12-13 November, 2016 for your own **Annual Conference at Hotel Ashok**. Your inputs for the conference program are valuable to us.

We would be informing you about the various activities by SMS, emails, website and bulletin.

Dr Ashok Kumar
Honorary Secretary
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From the Editor's Pen

Dear friends,

Greetings and a warm welcome to all!

We are delighted to be a part of this great knowledge-sharing platform. We are honoured to be bestowed with this great responsibility and extend our heartfelt thanks to the President for placing her faith in us. I am blessed to have a team of dynamic and vibrant co-editors, Dr Krishna Agarwal and Dr Nilanchali Singh. Dr Krishna is very prompt in all tasks assigned and Dr Nilanchali, the youngest amongst us, is very enthusiastic and bubbling with new ideas. The editorial team at Safdarjung has indeed done a commendable job and we too will make a sincere effort to bring forth an academic bonanza every month.

Our editions will largely be theme based with flavours of history and humour. A special highlight of every issue will be exploring history of some aspect of Obstetrics or gynecology. The crossword has been formulated with the help of residents. A new dimension has been added to journal scan where Dr Deepti Goswami will discuss the perspective of the study. We have added a new page with jokes and cartoons –'Tickle your funny bone'.

The 'Cesarean epidemic' should be a matter of concern for one and all. In his blog (dated January 9, 2016; Lifestyle, Science, World; TOI), Kiran Khumbhka a health policy expert comments on the "worryingly easygoing manner" in which families and obstetricians in India approach these procedures in the present day. The private sector's C-section rate touches 50 to 80 per cent; a far cry from the 20-25 per cent which too is considered worrisome. It's time to realize that normal delivery should be made the norm before it's too late. This has driven us to dedicate our first issue to certain aspects of labor. The crossword on labor will stimulate your gray cells. Dr Nilanchali has made an enthralling narrative on history of forceps.

With your support and active participation, we will keep striving for a better issue every time. Our motto is:

'Good, better, best-Never let it rest; Till good is better and your better is best.'

Happy Reading !

Dr Sangeeta Gupta
Editor
drsangeetamamc@gmail.com

Monthly Clinical Meeting

- Monthly Clinical Meeting will be held at Dr Ram Manohar Lohia Hospital on 27th May, 2016.

Mechanism of Onset of Labor

Deepika Sharma, Ashok Kumar
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Onset of labor is a natural process. In an attempt to understand the mechanisms involved in it, many theorems are postulated. Two of them are as follows:

1. There is loss of factors which maintain pregnancy.
2. Synthesis of factors which induce parturition.

One of the fascinating facts is that the signals required for the onset of labor are initiated by the mature fetus also. There is an increase in the population of myometrial uterotonic receptors too at term.

The uterine myometrium has few unique features as compared to other muscles in the body.

1. Uterine muscles not only contract but also retract and the degree of shortening at a given time is more than that of striated muscle cells.
2. The myometrium exerts its force in multiple directions which is also aided by the plexiform arrangement of muscle fibres.
3. The uterine fundal fibres contract more than that of the lower uterine segment.

As in other muscle cells, even in the uterine myometrium, interaction between the actin and the myosin filaments and increased excitability of the individual cells is the basic physiological event for contraction to occur[1].

Parturition – An inflammatory process:

With the onset of labor, the uterine anti-inflammatory environment switches over to a pro-inflammatory one. Inflammatory cells infiltrate the myometrium, placenta and the fetal membranes and release

1. Matrix Metalloproteinases (MMPs) which cause cervical ripening
2. Prostaglandins, serotonin and histamine which have direct uterotonic effect on the myometrium.
3. Pro-inflammatory cytokines- IL-1β, IL-6, IL-8 & TNF-α.

The laboring myometrium markedly expresses major monocyte chemo-attractant, chemokine (C-C motif) ligand 2 (CCL-2; also known as MCP-1). CCL-2 mediates local leucocytes (mostly monocytes and T cells) migration into various tissues. There is also upregulation of CXC chemokine – CXCL8, a major neutrophil chemoattractant[2].

The prerequisites for an effective leucocyte migration include

1. An increased concentration gradient of CCL2/ CXCL8.
2. Responsiveness of the immune cells via cognate chemokine receptors.

The myometrium is infiltrated by neutrophils and macrophages both in the upper and the lower uterine segment, more in the lower as compared to upper.

There is increased expression of ICAM-1, endothelial adhesion molecule which facilitates adhesion, arrest and transmigration of leucocytes.

IL-1β induces both basal and store operated Ca^{2+} entry in the myometrial smooth muscle cells. It also stimulates phosphodiesterase activity which results in breakdown of cAMP. It increases both PGF2α & its receptors[3].

In the uterine myometrium, monocytes express an increased levels of CCL2, IL6, MMP2 & MMP9.

There are 9 cytokines whose levels are markedly increased in the laboring myometrium – IL1RA, IL9, CCL2, IL-18, CSF-3, TNF-A, CCL7/MCP3, IL6.

Factors stimulating immune cell proliferation include IL-9 & CSF3.

Chemoattractatnts for ------monocytes- CCL2 & CCL7.

------neutrophils- CXCL8.

Pro-inflammatory cytokines include TNF-A, IL-6, IL-18.

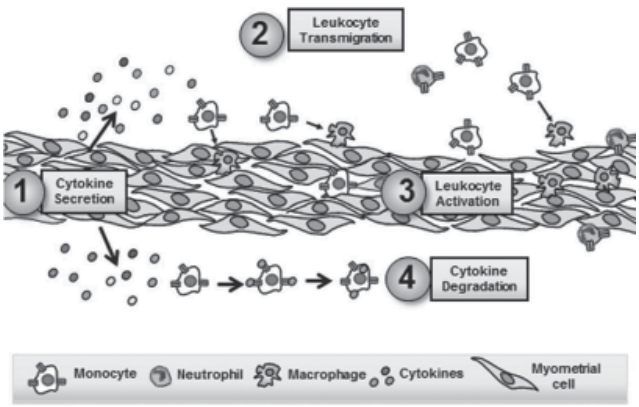
Anti-inflammatory cytokines include- IL1RA.

It is found that there is highest expression of CCL2 & CXCL8.

The most abundant proinflammatory cytokine is IL6.

IL9 & CSF3 are regulators of a variety of hematopoietic cells and act by stimulating proliferation and differentiation of these cells and also by inhibiting apoptosis.

Plasma levels of IL1RA are considered as a marker for monocyte activation & M2 macrophage polarization. Another fact observed is that the concentration of monocytes is 3 times that of neutrophils in the laboring myometrium. The negative feedback by the monocytes itself prevents overwhelming inflammatory response.



Schematic presentation of the hypothetical model for cytokine degradation after labor onset to prevent an uncontrolled inflammatory response. 1) Uterine chemokines secreted during term labor bind to chemokine receptors on circulating monocytes and/or neutrophils, which 2) mediates transmigration of peripheral leukocytes into the myometrium. 3) During migration, a direct contact with uterine SMCs activates leukocytes, and 4) causes selective internalization of chemokine receptors from the plasma membrane to lysosomes for degradation of chemokines[2].

Cervical events

Cervical ripening is also an inflammatory process. There is massive influx of leucocytes into the cervix associated with increase in proinflammatory cytokines and cell adhesion molecules. There is also an increase in the nitric oxide level.

Fetal membranes

There is no leucocyte migration in the fetal membranes but significant upregulation of IL-1β and IL-6 has been observed in labor.

Peripheral blood

Chemotaxis of peripheral blood leucocytes and reactive oxygen species production is greater in the blood samples of laboring women. The circulating leucocytes are an attractive potential therapeutic target also[3].

Physiological mechanisms:

1. Myometrial Changes:
   - As we know, actin interacting with myosin results in activation of ATPase leading to hydrolysis of ATPs. Also there is increase in intracellular calcium which combines with calmodulin which forms the calcium-calmodulin complex resulting in activation of myosin light chain complex.
   - CRH - Corticotrophin Releasing Hormone modulates the expression of large conductance K^+ channels via Ca^{2+}.
   - There are 3 coupling mechanisms occurring in myometrial cells.
     1. Electronic
     2. Ionic
     3. Metabolic
        1. Electronic coupling involves the transmembrane channels known as "Connexon" that form the gap junctions. Each connexon has 6 connexin subunit proteins. 4 important connexins are 26, 40, 43, 45. Connexin 43 junctions are scarce in non pregnant uterus and in abundance during labor.
        2. Ionic coupling involves Ca^{2+} & K^+ channels.
        3. Metabolic coupling includes the metabolites described later in detail.
   - There are 3 major cell surface receptors – G-Protein coupled receptors (GPCRs), ion linked channels and enzyme linked channels.
   - GPCRs act via 2 pathways. One is via adenylyl cyclase activation and other via phospholipase C.
2. Cervical Changes:
   - There are changes in the composition of glycosaminoglycans, proteoglycans and poorly formed collagen fibrils which also contributes to the reorganization and recovery of cervical tissue in the post partum period.

There are 4 phases of parturition

1. Phase of uterine quiescence and cervical competence.
2. Phase of uterine activation and cervical ripening.
3. Phase of uterine stimulation.
4. Phase of recovery.

Phase 1:

There are 4 molecular pathways – neural, endocrine, paracrine and autocrine.

Uterine quiescence is due to following factors-

- Effects for estrogen and progesterone via intracellular pathways
- Myometrial sarcolemmal receptor mediated cAMP, cGMP and ion channels.

a. Progesterone & Estrogen:

- Progesterone causes decreased expression of Contraction Associated Proteins (CAPs)
- Progesterone causes increased expression of transcription factor ZEB1- Zinc finger E- box

binding homeobox protein 1 which inhibits the expression of CAPs.

- Progesterone binding with progesterone receptor results in release of increased coregulatory factors like PSF - Polypyrimidine Tract binding protein associated splicing factor & sin3A/ HDACs- Yeast switch dependent 3 homologue A / Histone Deacetylase Corepressor Complex which inhibits connexin 43 gene.
- With the onset of parturition, there is fall in the progesterone levels and increased estrogen which results in decreased PSF, ZEB1 (due to increased microRNAs) sin/HDAC levels.

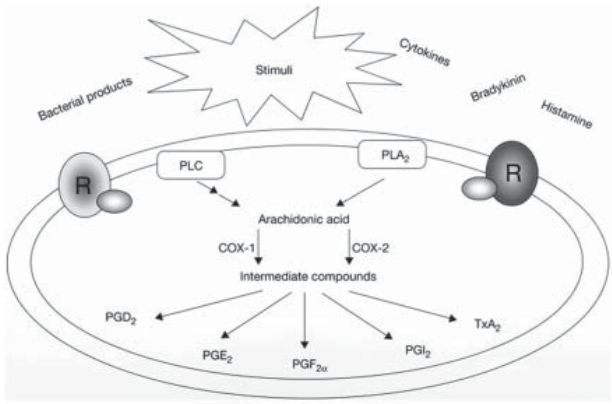
b. Beta adreno- receptors cause Gαs mediated increase in adenylyl cyclase and thus myometrial relaxation via increased cAMP.

c. LH & hCG receptors are GPCRs which also act via Gαs mediated pathway to cause myometrial relaxation.

d. Relaxin:
   - It's a peptide hormone with 2 chains A & B, with a structure similar to insulin.
   - Actions :
     - Lengthening of pubic ligaments
     - Cervical softening
     - Vaginal relaxation
     - Inhibition of myometrial contractions
   - There are 2 relaxin genes – H1 & H2.
     - H1 in decidua, trophoblast and prostate
     - H2 in corpus luteum
   - Relaxin levels are maximum at 8-12 weeks period of gestation - 1ng/ml.
   - Relaxin combines with Relaxin Family Peptide Receptor (RXFP-1) and acts via adenylyl cyclase pathway.

e. CRH- Corticotrophin Releasing Hormone
   - It is synthesized in the placenta and hypothalamus.
   - It has a unique role that it promotes myometrial quiescence during entire pregnancy and aids myometrial contractions with the onset of parturition.
   - This is possible due the differential action of CRH at its receptor CRHR1.
   - In non-laboring myometrium, CRH combines with CRHR1 resulting in activation of Gs adenyl cyclase- cAMP signaling pathway which in turn inhibits IP3, resulting in stabilization of cells.
   - In term laboring myometrium, ionized Ca^{2+} is increased by CRH by increasing IP3 which increases the myometrial contractility.

f. Prostaglandins:
   - PGE2 & PGI2 cause uterine quiescence by increasing c AMP, yet PGE2 can promote uterine contractility by binding to PGE receptor 1 & 3- EP-1 & EP-3.
   - PGE2, PGD2 & PGI2 cause vascular smooth muscle relaxation and vasodilation.



The decidua/fetal membranes interface is rich in tissue macrophages. These cells have a high rate of prostaglandin (PG) and thromboxane (Tx) synthesis and are likely to be involved in the onset of infection-associated preterm labour. Phospholipases (PLC, PLA2) can be activated by a number of receptors (R), releasing arachidonic acid for prostanoid synthesis through constitutive (COX-1) and inducible (COX-2) prostaglandin synthase pathways. The premature release of prostaglandins and inflammatory mediators by macrophages and other cell types is likely to cause contractions in the neighbouring myometrium[4].

g. ANP, BNP & cAMP :
   - ANP & BNP cause smooth muscle relaxation via activation of guanylyl cyclase resulting in increase in cGMP.

   The increased synthesis of uterotonins is also associated with accelerated uterotonin degradation viz

   - Increased Prostaglandin Dehydrogenase (PGDH) which degrade Prostaglandins.
   - Oxytocinase and Oxytocin.
   - Diamine oxidase and Histamine.
   - Catechol-O-Methyl Transferase (COMT) and Catecholamines.
   - Angiotensinase and Angiotension II.
   - PAF Acetyl Hydrolase & PAF.

Phase 2:

As mentioned earlier, progesterone withdrawal is one of the vital events during parturition. It is exemplified by the fact that mifepristone, progesterone receptor antagonist is an effective abortifacient. It also causes cervical ripening and increases the myometrial tissue sensitivity to uterotonins. There are changes in the relative expression of nuclear progesterone receptor isoforms (PR-A, PR-B & PR-C) and membrane bound progesterone receptors. It is a Micro RNA based regulation of progesterone metabolizing enzymes and transcription factors that modulate uterine quiescence.

It is observed that in the late gestation, there is a change in the relative ratio of PR-A to PR-B within the myometrium, decidua and chorion.

In the human cervix, at term, decreased activity of 17α hydroxy steroid dehydrogenase type 2 (HSD) results in net increase in estrogen and also a substantial decrease in progesterone levels.

The increased expression of microRNA 200a in the term myometrium blunts the expression of STAT5b, an inhibitor of 20 α HSD. So, when there is decreased expression of STAT5b it results in increased levels of 20 α HSD which in turn results in increased progesterone metabolism and decreased progesterone function.

Oxytocin receptors:

During the phase 2 of parturition, there is increase in the population of oxytocin receptors in the uterine myometrium, decidua, amnion and chorion which act via phospholipase C pathway which in turn results in increased cytosolic Ca^{2+} levels thus increasing the uterine contractility.

Expression of oxytocin receptors is primarily regulated by progesterone and estradiol.

Progesterone causes increased degradation of oxytocin receptors and inhibits the activation of cell surface oxytocin receptor.

Relaxin:

As mentioned earlier, relaxin causes remodeling of extracellular matrix of uterus, vagina, cervix, breast and pubic symphysis which also plays a role in their growth.

It also promotes cellular proliferation and inhibits apoptosis via GPCRs and RXFP1.

It induces the matrix metalloproteinases (MMPs) which results in degradation of the cellular glycosaminoglycans, proteoglycans and collagen.

Fetal contribution to initiation of parturition:

As the fetus grows, there is an increased uterine stretch, which is also due to increased myometrial tensile strength and increased amniotic fluid pressure.

This uterine stretch results in induction of specific contraction associated proteins (CAPs), connexin 43, oxytocin receptors and Gastrin Releasing Peptide (GRP).

The uterine stretch- mechanotransduction includes

a. Activation of cell surface receptors or ion channels.
b. Transmission of signals through extracellular matrix.
c. Release of autocrine molecules that act directly on the myometrium.

Fetal endocrine cascades:

For the initiation of parturition, signals from fetal hypothalamus pituitary adrenal axis are essential. Also the placental CRH production plays a pivotal role in this process. During labor, there is increase in both maternal plasma and amniotic fluid CRH. There is positive feedback effect of fetal cortisol on placental CRH. CRH combines with CRH receptor and via cAMP pathway cause uterine contraction. CRH augments the contraction inducing potency of a given dose of oxytocin.

Fetal lung surfactant and parturition:

Surfactant Protein A (SPA) inhibits PGF2α in the term decidua and it is observed that the amniotic fluid concentration of SPA falls at term.

Phase 3:

The important mediators which stimulate the uterine smooth muscle contractions are oxytocin, serotonin, histamine, angiotensin II and PAF (platelet activating factor)

Oxytocin:

It is nanopeptide synthesized in the magnocellular neurons of supraoptic and para-ventricular neurons in hypothalamus which is transported with its carrier protein, neurophysin and is stored in membrane bound vesicles in the posterior pituitary.

It is found that at term oxytocin is synthesized directly in the decidual and extra-embyonic fetal tissues and in the placenta which increases the mRNA levels in the myometrium which in turn increase the Monocyte Chemoattractant Protein-1 (MCP-1), IL-8 and urokinase plasminogen activator receptor.

Prostaglandins (PGs):

As pregnancy advances, PGs levels in the maternal plasma, maternal urine and amniotic fluid increase.

The laboring myometrium produces PGs, which is an efficient mechanism of activating uterine contractions. Similar to the oxytocin receptors, PGF2α receptors also increase at term. They are synthesized by fetal membranes and placenta also. Amnion is a major

source of PGs. They degrade the extracellular matrix, weaken the fetal membranes, cause cervical dilatation and alterations in the lower uterine segment.

Angiotensin II:

It also acts via GPCRs- AT1 & AT2. AT2 predominates in the non pregnant state and AT1 in the pregnant.

Endothelin-1:

It is 21 amino acid peptide which causes myometrial contraction via ET-A receptor.

EG-VEGF- Endocrine gland derived endothelial growth factor:

It is also known as prokineticin-1. It has 2 receptors PROKR1 & PROKR2. Is is an angiogenic factor, a new placental growth factor. The circulating levels of EG-VEGF increases towards term and significantly decreases at the time of labor. The same thing holds good for the receptor levels. It is more in the fetal membranes than the placenta. Within the fetal membranes, chorion is the main source. It decreases the MMP2 & MMP9 activities and also increases PGDH expression. It is a local intrauterine factor that is highly produced by the chorion trophoblast cells and acts in both autocrine and paracrine manner to ensure uterine quiescence during pregnancy[5].

Therefore, labor is a complex process right from its onset and results in delivery of the baby.

References

1. Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, et al. Williams Obstetrics 24e; 408-432.
2. Srikhajon K, Shynlova O, Preechapornprasert A, Chanrachakul B, Lye S. A New Role for Monocytes in Modulating Myometrial Inflammation During Human Labor. Biology of Reproduction 2014; 91(1):10, 1–12.
3. Norman JE, Bollapragada S, Yuan M, Nelson SM. Inflammatory pathways in the mechanism of parturition. BMC Pregnancy Childbirth 2007; 7(Suppl 1):S7.
4. Bernal AL. Mechanisms of labour—biochemical aspects. BJOG 2003; 110 (Suppl 20):39–45 .
5. Dunand P, Hoffmann V, Sapin L, Blanchon A, Salomon F, Sergent M et al. Endocrine Gland-Derived Endothelial Growth Factor (EG-VEGF) Is a Potential Novel Regulator of Human Parturition. Biology of Reproduction 2014; 91 (3): 73, 1–10.

Calendar of Monthly Clinical Meetings 2016-17

Months	Name of the Institute
April, 2016	Apollo Hospital
May, 2016	Dr RML Hospital
June, 2016	Army Hospital-Referral & Research
July, 2016	VMMC & Safdarjung Hospital
August, 2016	AIIMS
September, 2016	Fortis Hospital
October, 2016	Sir Ganga Ram Hospital
November, 2016	MAMC & LN Hospital
December, 2016	Hindu Rao Hospital
January, 2017	LHMC
February, 2017	ESI Hospital
March, 2017	UCMS & GTB Hospital
April, 2017	Apollo Hospital

Case Summaries for AOGD Bulletin may be sent by email to the editor/CD mey be handed over on the day of the meeting.

-Dr Sangeeta Gupta

Revised Labor Curves

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Labor curve, a graphical representation of the progression of labor, is prepared by plotting cervical dilatation on vertical axis and time on horizontal axis. It helps the labor attendant to detect deviation from normal labor. Before 1955, there were no labor curves and the researchers only mentioned about the total duration of labor.

Emanuel Freidman, in year 1955, for the first time described the labor pattern and the labor curve[1,2]. Since then this curve has been used world over for almost 40 years. The Friedman curve was based on the labor data collected from 500 nulliparous women from Sloane hospital in New York, in a sample of convenience. He plotted labor progress on a graph with cervical dilatation in centimeters (cm) on Y axis and time in hours on X axis. It generated a sigmoid shaped curve. In spite of the fact that this curve was descriptive and non-representative, it has been used across the globe until recently. The World Health Organization (WHO) partogram (1994) for labor management is based on this curve[3].

However, recent data on labor show that the labor progresses at a much slower rate than what was traditionally considered[4-13].

Concerns with traditional labor curve

The major concern with the use of traditional labor curve is high rate of cesarean section. It was estimated that in year 2011, in United States every 3rdchild was born by cesarean section[14]. It has been now known fact supported by studies that the risk of severe morbidity, both short and long term, is increased with cesarean delivery as compared to vaginal birth[14].

It has been estimated that dystocia, diagnosed on the basis of traditional labor curve, remains to be the most common indication of primary cesarean section[14]. Dystocia is characterized by slow and abnormal progress of labor[15]. Barber et al[16] in a population based study showed that 34% for primary cesarean section were due to dystocia. Consortium on Safe Labor data on 226,668 women revealed that one third of elective cesarean sections were due to previous uterine scar[4]. Therefore reducing the rate of primary cesarean section will have a large impact on total cesarean section rate; and the primary cesarean section rate can be lowered to a large extent if the traditional labor curves are revised.

Gifford et al[17] reported that almost 25 % of cesarean sections, performed for failure of labor to progress, were done even before the woman entered the active phase of labor (at 0 to 3 cm dilatation of cervix). Zhang et al[4] studied pattern of cesarean delivery with respect to labor progress in 226,668 women using data from consortium on safe labor. It was seen that 38% of women with spontaneous labor and 68% of women with induced labor had cesarean section even before 6 cm dilatation of cervix.

The contemporary studies suggest that labor progresses at a much slower pace and cesarean sections which are performed for dystocia can be minimized if the labor curves are re-assessed and revised based on the robust evidence.

Contemporary studies on labor

In light of the evidence suggesting that normal labor progresses at a slower rate than the traditional belief and most of the women if monitored for maternal and fetal wellbeing will deliver successfully with a normal outcome.

Data from consortium on safe labor

In year 2010, Zhang et al[3] reported contemporary patterns of labor using data from the Consortium on Safe Labor which was supported by National Institute of Health. This was a large, multicenter, retrospective study, between 2002 to 2008, with an objective to examine labor patterns in modern US population. The study was conducted in 19 hospitals across United States and the information was retrieved from the electronic medical records of these hospitals. Total number of deliveries was 228,668 in the database and of these 62,415 parturients who had singleton term pregnancy, went in spontaneous labor with vertex presentation, had vaginal birth and normal perinatal outcome were selected. A repeated measures analysis was performed to draw average labor curves for nullipara as well as multigravida (figure 1). These curves revealed that progression of labor is much slower as compared to the traditional Friedman curves in both

nullipara and multigravida (figure 1). Zhang et al also performed an interval censored regression analysis to know the duration of labor stratified for centimeter by centimeter (table 1). They suggested that point of intervention should be guided by 95th percentile rather than the average.

Important features of the new labor curves (figure1 and table 1)

1. Labor in nulliparas and multigravida progresses at almost the same rate before 6 cm cervical dilatation and is much slower than that shown in Friedman's curves.
2. Latent phase appears to be much longer than traditionally thought and time taken for cervix to dilate from 4 cm to 6 cm can be as long as 8 hours in nullipara and 6 hours in multigravida (table 1)
3. Active phase starts beyond 6 cm dilatation of cervix in both nullipara and multigravida.
4. After 6 cm, the curve for the nulliparous woman is much flatter as compared to the multigravida.

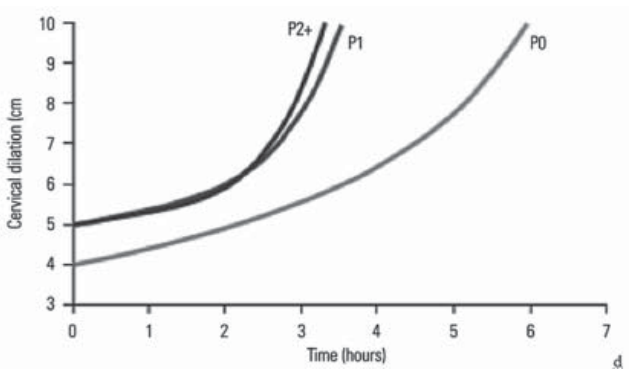

Figure 1: Average labor curves in women with singleton, term pregnancy, spontaneous labor, vaginal birth and normal perinatal and maternal outcomes. P0- Nulliparous, P1-para 1, P2+-para two and more (*Reprinted with permission from ACOG Safe prevention of primary cesarean delivery. Am J ObstetGynecol 2014*)

Table 1: Progression of spontaneous labor in uncomplicated pregnancy, stratified according to the level of cervical dilatation and parity

Dilatation of cervix (cm)	Time to progress (hours) Median (95th percentile)		
	Nullipara	Para1	Para≥2
3-4	1.8(8.1)	--	--
4-5	1.3(6.4)	1.4(7.3)	1.4(7.0)
5-6	0.8(3.2)	0.8(3.4)	0.8(3.4)
6-7	0.6(2.2)	0.5(1.9)	0.5(1.8)
7-8	0.5(1.6)	0.4(1.3)	0.4(1.2)
8-9	0.5(1.4))	0.3(0.9)	0.3(0.9)
9-10	0.5(1.8)	0.3(0.9)	0.3(0.8)
Second stage	0.6(2.8)	0.2(1.3)	--

Modified from Zhang et al (4)

The Consortium on Safe Labor concluded that progress of labor, from 4 to 6 cm cervical dilatation, is slower than the traditional belief and rate of cervical dilatation accelerates after 6 cm. Thus it is recommended that before 6 cm cervical dilatation, labor should be allowed to continue for longer duration and that this may reduce the rate of primary and repeat cesarean deliveries in United states.

Other studies

There are other studies which have addressed the issue of labor duration. However the modern researchers have not reported data on latent phase.

Rouse et al[7,8] in their study suggested that active phase starts when cervix is 4 cm dilated and ≥90% effaced or 5 cm dilated irrespective of effacement. Whereas Peisner et al[8] reported that in women who delivered successfully, 50% started active phase at ≤4 cm, 74% at ≤5cm and 89% at ≤6 cm cervical dilatation.

A study on 500 women demonstrated that when augmentation of labor with oxytocin was extended from minimum of 2 hours to 6 hours, majority of women successfully delivered vaginally with normal maternal and perinatal outcome[9]. The researchers validated the fact that women who had not progressed in spite of 4 hours of oxytocin administration and the oxytocin continued on the judgement of treating physician, then higher proportion of women had vaginal birth with normal neonatal outcomes[10].

Secondary analysis of a multicenter randomized study on fetal pulse oximetry involving 4126 nulliparous women revealed that none of the adverse neonatal outcome was related to the duration of second stage of labor[11]. However studies on multiparous women showed that when the duration of second stage was > 3 hours, risk of neonatal morbidity was significantly increased[12]. Spong et al[13] suggested that the limits for diagnosis of arrest of second stage of labor should be increased by an hour.

American College of Obstetricians and Gynecologists (ACOG) and Society of Maternal and Fetal Medicine (SMFM) recommendations[17]

Though the strategies proposed by Consortium on Safe Labor (Zhang et al,[4]) have not been validated by any randomized controlled trial, ACOG and SMFM Consensus on Safe Primary Prevention of Cesarean Section has come up with some recommendations based on the Consortium on Safe Labor and other studies (table 2). This table compares the recommendations of ACOG and SMFM consensus on Safe Prevention of cesarean delivery with the historical

Table 2: Comparison of Friedman's definitions of labor disorders with the recommendation of ACOG & SMFM

Phase of labor	ACOG and society of MFM obstetric care consensus (2014)	Friedman (1955)
Latent phase duration	It should not be an indication for cesarean section, most women enter in active phase with expectant management, some cease to have uterine contractions, and few enter active phase with augmentation of labor with oxytocin or amniotomy **1B recommendation** **Moderate quality evidence**	>20 h in primi and > 14 h in multi
Start of active phase	6 cm cervical dilatation **1B recommendation** **Moderate quality evidence**	4 cm cervical dilatation
Arrest of labor in active phase	Women with ≥6 cm cervical dilatation, ruptured membrane who fail to progress despite 4 h of adequate uterine activity or 6 h of oxytocin administration and inadequate uterine activity and no cervical change **1B recommendation** **Moderate quality evidence**	Arrest of dilatation-no dilatation for >2h in primi, >1 h in multi Arrest of decent-no decent for >2h in nullipara, >1 h in multigravida
Second stage of labor	Atleast 3 h of pushing in primigravida and 2 h of pushing in multigravida **1B recommendation** **Moderate quality evidence**	>2 hours in nullipara > 1 hour in multi gravida

recommendation by Emanuel Friedman.

Miller et al reviewed the literature and concluded that labor progress is slower than what was thought and is mostly followed by vaginal birth without any complication[18].

Therefore it's time to revise our labor curves and shift from the traditional Friedman's curves to the contemporary curves defined by Zhang et al. These labor curves need to be further validated by prospective multicenter studies and to prove that it actually does not compromise maternal and fetal wellbeing and at the same time significantly brings down the rate of cesarean section.

References

1. Friedman EA. The graphic analysis of labor. Am J ObstetGynecol 1954; 68:1568.
2. Friedman EAPrimigravid labor; a graphicostatistical analysis. ObstetGynecol 1955; 6:567.
3. World health Organization. World health Organization Partograph in management of Labor. Lancet. 1994; 343: 1399-1404..
4. Zhang J, Landy HJ, Branch DW, et al. Contemporary patterns of spontaneous labor with normal neonatal outcomes. Consortium on safe labor. ObstetGynecol 2010; 116: 1281.
5. Zhang J, Troendle J, Mikolajczyk R, et al. The natural history of the normal first stage of labor. ObstetGynecol 2010; 115:705.
6. Zhang J, Troendle JF, Yancey MK. Reassessing the labor curve in nulliparous women. Am J ObstetGynecol 2002; 187:824.
7. Rouse DJ, Owen J, Hauth JC. Criteria for failed labor induction: Prospective evaluation of a standardized protocol. Obstet Gynecol. 2000; 96:671-7
8. Peisner DB, Rosen MG. Transition from latent to active labor. Obstet Gynecol. 1986; 68:448-51.
9. Rouse DJ., Owen J. Hauth JC. Active-phase labor arrest: Oxytocin augmentation for atleast 4 hours. Obstet Gynecol. 1999; 93:323-8.
10. Rouse DJ., Owen J. Savage KG. Hauth JC. Active-phase labor arrest: revising the 2 hour minimum. Obstet Gynecol. 2001; 98:550-4)
11. Rouse DJ, Weiner SJ, Bloom SL. Second stage labor duration in nulliparous women;relationship to maternal and perinatal outcomes. Am J Obstet Gynecol.2009;201:357.
12. Le Ray C, Audibert F, Goffinet F, Fraser W. When to stop pushing, effect of duration of second stage expulsion efforts on maternal and neonatal outcomes in nulliparous women with epidural analgesia. Am J Obstet Gynecol. 2009; 201:361.
13. Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Presenting the first cesarean delivery. Obstet Gynecol. 2012; 120:1181-93.
14. Safe Prevention of Primary Cesarean Delivery. Obstet Gynecol. 2014; 123:693-711.
15. ACOG practice bulletin. Dystocia and augmentation of labor. International J of Obstetrics and Gynecology.2004; 85: 315-324.
16. Barber EL, Lundsberg LS, Belanger K, Pettker CM, Funai EF, Illuzi JL. Indications contributing to the increasing cesarean delivery rate.Obstet Gynecol. 2011;118:29-38.
17. Gifford DS, Morton SC, Fiske M. Lack of progress in labor as a reason for cesarean section. Obstet Gynecol. 2000; 95: 589.
18. Millen KR, Kuo, Zhao L, Gecsi K. Evidence based guidelines in labor management. Obstet Gynecol Surv. 2014; 69: 209-17.

Methods of Induction of Labour –Newer perspectives

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"Success is not final, failure is not fatal, it's the courage to continue that counts".

-Winston Churchill

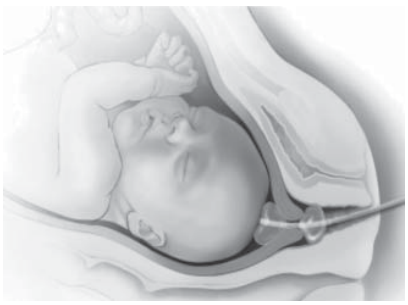
Induction of labour is one of the most used, and probably one of the most effective interventions in modern obstetrics. Globally, labour is induced in 20 to 30% of all deliveries for a variety of reasons, amongst which hypertensive disorders in pregnancy, intrauterine growth restriction, post term pregnancy are the most common[1]. There are varieties of methods available for induction but their judicious use will only determine the effectiveness of induction which is best judged by measuring the time taken to achieve successful vaginal delivery, usually within a predefined time frame and also with good clinical condition of the mother and baby. The other parameters like cost, ease of procurement and administration and patient satisfaction cannot be ignored.

Work up prior to induction

It is very important to determine gestational age accurately before considering induction, to avoid inadvertent post-term and preterm deliveries. Counselling regarding indications, contraindications including risks (possible complications) and benefits of induction should be done with patient and consent should be taken. The condition of the cervix described as cervical ripening is important for successful labour induction assessed by Bishop score. Score of less than 6 generally requires cervical ripening.

Methods of induction

Mechanical methods



Trans-cervical Catheter

Generally, these techniques are used only when the cervix is unfavourable which uses a Foley's catheter with 25 to 50 ml balloon that is placed through the internal cervical os. A modification of this is extra amniotic saline infusion in which constant saline infusion is passed through the catheter into the space between the internal os and placental membranes.

Compared to the use of PGE2 gel, the foley's catheter has comparable induction to delivery duration and caesarean section rates with less maternal and neonatal morbidity. Foley's catheter has fewer side effects and causes no uterine tachysystole so very strict monitoring of uterine contractions is not required during the ripening phase[2]. Foley's catheter causes less risk of scar dehiscence compared to prostaglandins, therefore can be used safely in induction of previous caesarean section. In developing countries where cost is an important limiting factor and very stringent conditions for storage of prostaglandins may not be available, Foley's catheter is a good option. When compared to vaginal application of misoprostol, induction by Foley catheter is associated with reduced uterine hyperstimulation causing adverse fetal heart changes. However, some studies have demonstrated that maternal and neonatal infections were increased with their use [3] but recently many trials have shown that the risk of infection in such cases is insignificant. The practice of Foley's catheter use for induction has now changed with ACOG recommending its use for induction. Because of its safety profile, it is now recommended by WHO for induction of labour particularly in previous caesarean section cases.

Membrane stripping

This method has been recommended by ACOG, NICE, WHO. Most authors agree that membrane sweeping is an effective method to reduce the need for formal labour induction, whereas amniotomy alone is not recommended. It is safe and decreases the incidence of post-term pregnancy without consistently increasing the incidence of ruptured membranes, infection, or bleeding.

Amniotomy

Artificial rupture of membranes also called surgical induction always implies commitment to delivery.

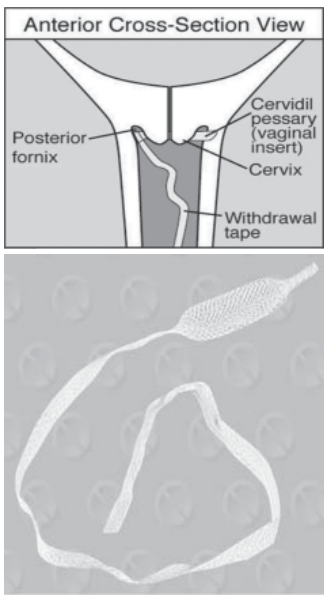
The incidence of chorio-amnionitis also becomes high because of unpredictable and occasionally long interval until labour onset. Recent guidelines by American College of Obstetricians and Gynaecologists (2013a) recommends the use of amniotomy to enhance progress in active labour, but cautions about the increase risks of infection and maternal fever. However NICE and WHO don't recommend this as method of induction[4,5,6].

Pharmacological techniques

ProstaglandinE2

Dinoprostone is a synthetic analogue of prostaglandinE2. It is commercially available in three forms: a gel, a time release vaginal insert (DVI), and 10 mg suppository. All forms of PGE2 become unstable at room temperature and require controlled storage conditions.

DVI available as cervidil contains 10mg insert and it releases prostaglandin at 0.3mg /hr. The insert needs to be removed after 12hrs, after onset of labour or after hyperstimulation. The pregnant woman is laid supine for 2 hrs of insertion.



In recent trials it has been found that DVI and PGE2 are equally efficacious in achieving vaginal delivery within 24 hrs and there is no difference in rates of caesarean section [7] but because of single application DVI has better patient satisfaction. Recently, in various studies it has been concluded that out of intravaginal and intracervical route of administration of PGE2, intravaginal route is preferred because of improved efficacy and easier route of administration. In trials comparing vaginal PGE2 gel to oxytocin for cervical ripening, vaginal PGE2 was more efficacious than oxytocin in achieving vaginal delivery within 24 hours.

While ACOG and WHO recommends the use of vaginal and intracervical PGE2, NICE advocates only intravaginal route.

Caution is recommended when these preparations are used in women with ruptured membranes and also in patients with asthma, glaucoma and previous caesarean section.

Misoprostol is a synthetic prostaglandin E1 that is used in pregnant women for cervical ripening and induction of labour. Originally, misoprostol was prescribed for the treatment of gastric ulcers and is still not registered for obstetrical use and as such constitutes an "off label" prescription.

It is administered orally or vaginally. The tablets are stable at room temperature. It is currently available in the form of 200 mcg and 100mcg tablets.

The United States Food and Drug Administration (FDA) has not yet approved use of misoprostol for IOL.Despite its prevalent use, Drug controller of India also does not permit its use for IOL. As per NICE guidelines, it is only used for IOL in intrauterine dead baby.

WHO and ACOG recommends the use of oral and vaginal misoprostol in dose of 25mcg (low dose).

Misoprostol is, however, not for IOL in women with previous caesarean delivery. Misoprostol is more effective than oxytocin when 50mcg or more higher doses are used but there is higher incidence of tachysystole[8].

Misoprostol has been associated with increased risks of meconium staining of the amniotic fluid and thus of neonatal meconium aspiration syndrome. Although the mechanism of this is unclear, in vitro rat tissue studies have shown that misoprostol, unlike oxytocin, stimulates both bowel and uterine smooth muscle, and it has been postulated that misoprostol may cross the placenta to directly stimulate fetal bowel.[9]

The newer drug ***misoprostol vaginal insert (MVI)*** contains misoprostol as the active ingredient which releases it at a controlled rate of approximately 1/24th of the total dose per hour of insertion. It is safe as it can be removed anytime because of its retrieval tape mechanism at the onset of labour or if any adverse reaction occurs. It is unstable at room temperature. Sustained release preparations are more convenient with single application than multiple dosing leading to better patient compliance.

Phase III testing has recently been completed on a sustained-release misoprostol vaginal insert (MVI) containing 200 mcg of misoprostol (Ferring Pharmaceuticals), and this MVI is expected to be available commercially when FDA approved. The insert was compared with 10mg dinoprostone (PGE2) vaginal insert (DVI) and shown that median time of vaginal delivery was significantly shorter for women who received MVI but there was no difference in rates of caesarean delivery[10].

Oral versus vaginal administration

The studies have shown that comparing oral and vaginal- route of administration, oral misoprostol was associated with a lower rate of uterine tachysystole with fetal heart rate changes, but there was no significant differences with respect to rates of caesarean delivery[11].

Oxytocin

Oxytocin may be used for labour induction or for augmentation. With oxytocin use, the American College of Obstetricians and Gynaecologists (2013b) recommends fetal heart rate and contraction monitoring similar to that for any high risk pregnancy. In general, oxytocin should be discontinued if the number of contractions persists with a frequency of more than five in a 10 min period or more than seven in a 15 min. period or with a persistent non reassuring fetal heart rate pattern. The response is highly variable and depends on pre-existing uterine activity, cervical status, pregnancy duration and individual biological differences. From past till recent times Oxytocin still remains the most efficacious agent for labour induction and with the proper safety protocols in place has an acceptable side effect profile. The side effects are immediately reversible on discontinuation of its use.

Oxytocin dosage

It is typically given as an intravenous infusion and has a half-life of 3 to 6 minutes. Common regimens start at 1 to 6 mU/min with subsequent increases in dose of 1 to 6 mU/min every 15 to 40 minutes to a maximum dose of 20 to 42 mU/min. A 1ml ampule containing 5 units is usually diluted in 500ml of a crystalloid solution and administered by infusion. This mixture results in an oxytocin concentration of 5 mU/ml.

Oxytocin regimens

High-dose oxytocin regimens are more effective than low-dose regimens in achieving a shorter duration of labour when used for induction but higher doses of oxytocin have the potential to increase the risk hyperstimulation with FHR changes which may or may be associated with an increased risk of caesarean delivery for this indication. However, the shorter labours seen with high-dose regimens may also be associated with decreased fetal exposure to infection; chorioamnionitis has also been associated with adverse neonatal neurological outcomes[12,13]. Doses>40mU/min are rarely used in modern practice except following an intrauterine fetal demise.

Regimen	Starting dose (mU/min)	Incremental increase (mU/min)	Interval (min)
Low dose	0.5-1.5	1	15 –40
	2	4, 8, 12, 16, 20, 25, 30	15
High-dose	4	4	15
	4.5	4.5	15-30
	6	6	20 – 40

Uterine tachysystole is more common with shorter intervals.

Risks

Oxytocin has significant antidiuretic action, and when infused at doses of 20mu/min or more, water intoxication can occur if aqueous fluids are infused in appreciable amounts along with oxytocin. This can lead to convulsions and death. In general, if oxytocin is to be administered in high doses for a considerable period of time, its concentration should be increased rather than increasing the flow rate of a more dilute solution. Therefore concentrated oxytocin is best given by infusion pump.

Conclusion

Oxytocin, oral misoprostol, vaginal misoprostol, DVI, MVI, and vaginal PGE2 are all currently available and accepted pharmacologic agents for induction of labour and cervical ripening. In case of unripe cervix, mechanical induction with trans-cervical balloon and oral misoprostol are probably the safest methods.In modern era, Obstetric care providers must balance between efficacy and safety when choosing the best agent for an individual patient and also on availability of resources for monitoring labour. Factors such as cost, convenience, and patient satisfaction also play a key role. At this time oral misoprostol appears to be the most effective and economical primary agent for cervical ripening; oral misoprostol does not require repeated vaginal examinations, and is the treatment most consistently associated with a reduced risk of caesarean delivery. Oral misoprostol is recommended as the first-line cervical ripening agent by the World Health Organization. Vaginal prostaglandins, administered

by intermittent or sustained-release delivery devices remain reasonable options. When induction of labour is started, the condition of the both mother and child should be the indicator for further decision making instead of induction time.

References

1. Kelly Mast, Mieke LG Ten Eikelder, Kitty WM Bloemenkamp et al. Induction of labour: who, when and where? Recent Advances in Obstetrics & Gynecology2015; 26:135-147.
2. Jozwiak M, Oude Rengerink K, Benthem M et al. Foley catheter versus vaginal prostaglandinE2 gel for induction of labour at term (PROBAAT trial): an open – label, randomised controlled trial. Lancet 2011; 378:2095-2103
3. Heinmann J, Gillen G, Sanchez-Ramos L, et al: Do mechanical methods of cervical ripening increase infectious morbidity? A systematic review. Am J obstet Gynecol2008; 19(2):177-199.
4. World Health Organization (WHO), Department of Reproductive Health and Research. WHO recommendations for induction of labour. Geneva: WHO, 2011.
5. National Institute for Clinical Excellence. Induction of Labour. London: National Institute of Clinical Excellence; 2008.
6. Induction of Labor. ACOG Practice Bulletin No. 107. American College of Obstetricians and Gynecologists. Obstet Gynecol. 2009; 114:386–397.
7. Boulvain M, Kelly A, Irion O. Intracervical prostaglandins for induction of labour. Cochrane Database Syst Rev. 2008; 1:CD006971.
8. Alfirevic Z, Kelly AJ, Dowswell T: Intravenous oxytocin alone for cervical ripening and induction of labour. Cochrane Database Syst Rev 4:CD003246,2009.
9. Matonhodze BB, Katsoulis LC, Hofmeyr GJ. Labor induction and meconium: in vitro effects of oxytocin, dinoprostone and misoprostol on rat ileum relative to myometrium. J Perinat Med. 2002; 30:405–410.
10. Wing D, Brown R, Plante L, et al. Efficacy and safety of misoprostol vaginal insert compared with dinoprostone vaginal insert for labor induction. Am J Obstet Gynecol. 2013; 208:S49.
11. Hofmeyr GJ, Gu¨ lmezogluAM, Pileggi C. Vaginal misoprostol for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2010; Issue 10:CD000941.
12. Wu YW, Escobar GJ, Grether JK, et al. Chorioamnionitis and cerebral palsy in term and near term infants. JAMA. 2003; 290:2677–2684.
13. Impey LW, Greenwood CE, Black RS, et al.The relationship between intrapartum maternal fever and neonatal acidosis as risk factors for neonatal encephalopathy. Am J Obstet Gynecol. 2008; 198: 9.

Forthcoming Events

- Thalassemia Control Cell & Deptt of Obst & Gynae, Hindu Rao Hospital will be organizing - Awareness Program on Thalassemia Screening and Diagnosis on 4th May, 2016 at Hindu Rao Hospital. Contact: Dr Suman Lata Mehendiratta, Ph. 9871858597
- FOGSI Infertility Committee and AOGD Infertility Committee is organizing a CME on Gonadotrophins in IUI on 4th May, 2016 at 1:00 pm at The Hotel Lalit, New Delhi.
- The Forum of Obstetricians and Gynecologists of South Delhi will be organizing a CME on the Current Concepts in the Management of Preterm Labor and Mother's Day Celebration on Mother's Day Eve 7th May, 2016. Contact: Dr Anita Sabharwal, Ph. 9810366459
- Monthly Clinical Meeting will be held in Dr Ram Manohar Lohia Hospital on 27th May, 2016.
- Sunrise Hospital, Delhi will be organizing - Workshop on Gynae Laparoscopic Surgery on 16th and 17th July, 2016.
- 19th Post-graduate Practical Course and CME will be conducted from 14th to 16th October, 2016 in MAMC Auditorium, New Delhi. Date: 14th - 16th October, 2016. at Auditorium, MAMC, New Delhi. Details at http://www.mamc.ac.in/
- Quiz on 'Contraception', on 23rd July 2016, 1:00-2:00 pm at Maulana Azad Medical College, New Delhi. For details, contact Dr Rachna Sharma 09873617586.

Induction of Labour in Previous Caesarean Section: Breaking the barriers

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When faced with a challenge, look for a way, not a way out. – David L. Weatherford

One such challenge with obstetricians is rising rate of Cesarean section. A consistent increase in caesarean rate has been observed in both developing and developed countries including India. Caesarean rates are much higher than the recommended level of 5 – 15 % by WHO.[1] The solution seems to be vaginal birth after Cesarean section. Earlier, "Once a caesarean always a caesarean" was the dictum of management for pregnancies with previous caesarean section, which continued for around 50 to 60 years. With emerging evidence, trial of labour after caesarean section (TOLAC) is an acceptable current practice.[2] Another big issue associated with previous Cesarean is Induction of Labour. Since we have limited options available for induction of labour in previous Cesarean and that too with limited dosage, careful selection of candidates for induction may lead to better maternal and fetal outcomes.

Why induction of labour in previous cesarean?

There are three possible outcomes of pregnancies with previous caesarean i.e. successful trial of Labour leading to vaginal birth after caesarean, unsuccessful trial of labour leading to emergency caesarean section and an elective repeat caesarean section. Various safety analysis data suggest that successful trial of labour is safest followed by elective repeat caesarean and thereafter, unsuccessful trial of labour culminating into caesarean section.[3] Hence, if patients are carefully selected, successful vaginal birth after Cesarean has the best outcome. Moreover, TOLAC if successful, is helpful in curbing Cesarean Section rates as the WHO has emphasized upon.

Concerns of labour induction in previous cesarean section

Patients with previous CS are not without risk. The risk of failed induction and the possibility of uterine rupture are major concerns of women undergoing a trial of labor after a previous cesarean delivery, particularly after induction of labour. The most dreaded complication is uterine rupture. Therefore, proper selection of patient and proper counselling plays an important role in management. Patients who undergo induction of labour are at two to three fold increased risk of uterine rupture and around 1.5 fold increase of caesarean delivery in induced and / or augmented labour compared with spontaneous VBAC delivery. The risk of uterine rupture ranges between 0.15 -0.4% in spontaneous labour, 0.54 – 1.4% in induced labour and 0.9 – 1.9% in augmented labour.[4]

Selection of patients for induction of labour

RCOG recommends that a senior obstetrician should take the decision to induce labour, the proposed method of induction, decision to augment labour with oxytocin and the time intervals for serial vaginal examination.[2] Following points should be considered while considering a woman for induction of labour:

- No contraindications of Induction of Labour
- Prior indication for Cesarean Section
- Inter-pregnancy interval
- Prior uterine incision
- History of any previous vaginal delivery
- Prior records of CS to be reviewed in details
- History of any post -op complications

The risk of uterine rupture varies with previous uterine scar. The risk is 0.2-1.5%, 1-1.6% and 4-9% with lower transverse uterine incision, lower segment vertical uterine incision and classical scars respectively.[5,6] Similarly, risk of uterine rupture varies with interdelivery interval i.e. 4.8%, 2.9% and 0.9% for interdelivery interval < 12 months, 12-24 months and > 24 months respectively.[7] One should keep in mind, the contraindications for Induction of labour, which are as follows:

- Previous classical caesarean / inverted T incision
- Previous hysterotomy / myomectomy entering cavity
- Previous uterine rupture

- Presence of contraindications of labour - placenta praevia, malpresentations, cephalo-pelvic disproportion etc
- Patient not willing for induction of labour

Inducing agents

The RCOG guideline, 2015 on birth after previous caesarean quotes: "Although induction and augmentation are not contraindicated in women with previous caesarean delivery, there remains considerable disagreement among clinicians on their use."[2] Different methods –viz. oxytocin, prostaglandins, amniotomy or Foley's bulb can be used. Studies showed that risk of rupture is high with prostaglandins as compared to others.[8]

Oxytocin: Oxytocin augmentation is not contraindicated in women undergoing a TOL after Caesarean section. Medical induction of labour with oxytocin may be associated with an increased risk of uterine rupture and should be used carefully after appropriate counselling. Risk of uterine rupture is dose dependent. Maximum dose recommended upto 20 milliunits/min.[8] It is recommended that the total duration of failure to progress should not exceed 3 hours in active phase. Oxytocin is associated with minimal or moderate risk of uterine rupture. Proper monitoring of patients is essential along with use of infusion pump, if available. Risk of rupture is 1.1% with oxytocin alone and 1.4% with prostaglandins and oxytocin together.[9]

Prostaglandin E2 or Dinoprostone: Medical induction of labour with prostaglandin E2 is associated with an increased risk of uterine rupture and should be used in exceptional circumstances and after appropriate selection and counselling. Obstetrical and maternal history is to be assessed prior to induction as it is associated with significant risk of uterine rupture. Not more than one dose of PGE2 should be used. According to WHO, PGE2 (vaginal inserts 2.5 mg) when compared to oxytocin shows no significant difference in any outcome (CS / instrumental vaginal delivery /epidural analgesia / Apgar score / perinatal death).[8] However, uterine rupture was noted in prostaglandin group but none in the oxytocin group. Up to 2001, there were conflicting data on the risk of labour induction with prostaglandin E2. Several other smaller studies reported that it appeared to be safe, effective, and not associated with an increased risk of uterine rupture.[9]

Prostaglandin E1 or Misoprostol: Prostaglandin E1 (misoprostol) is associated with a high risk of uterine rupture and should not be used as part of a TOL after Caesarean section.[9] In the recent WHO document, comparing misoprostol with oxytocin, it was noted that misoprostol is associated with increased risk of uterine rupture.[8]

Trans-cervical Balloon: Mostly accepted method for cervical ripening in case of previous Cesarean section. A foley catheter may be safely used to ripen the cervix in a woman planning a TOL after Caesarean section.[9]

Patient counselling and documentation

Prior to induction of labour, informed consent with documentation of each and every detail like indication and method of induction, interval of usage with timings, labour monitoring and further plan in case of failure of induction is mandatory. Proper counselling of patients explaining risks, benefits and outcomes is necessary.

Labour monitoring

Monitoring of labour is very important for patients with previous CS undergoing induction of labour.

***Maternal Monitoring*:[9]** Women planning a TOL after Caesarean should have appropriate monitoring in labour. The presence of a devoted birth attendant is ideal. Progress of labour should be assessed frequently, as there is some evidence that prolonged or desultory labour is associated with an increased risk of failure and uterine rupture. Epidural analgesia is not contraindicated. Women who are induced after a previous cesarean delivery have a longer latent phase, but a similar active phase, compared with women who experience spontaneous labor after a cesarean delivery.[10] While making the diagnosis of a protraction or arrest disorder in women who undergo induction of labor after cesarean delivery, it is reasonable for clinicians to apply the same criteria as in women without a previous cesarean delivery.

***Fetal Monitoring*:[9]** Continuous electronic fetal monitoring in labour is recommended for all women attempting TOL after Caesarean, especially after labour induction. The most reliable first sign of uterine rupture is a nonreassuring fetal heart tracing. This may be sudden in onset and may not be related to contractions. Continuous fetal heart rate monitoring is recommended apart from monitoring for signs and symptoms of uterine rupture.

Likelihood of successful induction

Inductions in women with a prior cesarean delivery are successful in approximately 50 percent cases, with

the highest chance of success in women with a prior vaginal delivery and favorable cervix. Calculators are available that estimate the likelihood of successful induction in women with a prior cesarean delivery. Induction results in a lower vaginal delivery rate than spontaneous labor (mean vaginal delivery rate 68 versus 80 percent).[11]

Factors affecting success of induction of labor in previous cesarean

The factors which significantly increase the chance of successful vaginal birth are previous history of vaginal delivery and favorable cervical status (modified Bishop score ≥6). In one large study, successful induction occurred in 91% of women with a prior vaginal delivery and a favorable cervix, 77% of women with a prior vaginal delivery but an unfavorable cervix, 69% of women with no prior vaginal delivery but a favorable cervix, and 45% of women with no prior vaginal delivery and an unfavorable cervix.[12]

Outcomes of labour induction

Most studies of the outcome of labor induction in women with a prior cesarean delivery have compared those undergoing induction with those undergoing spontaneous labor. In 2000 Ravasia et al. reviewed the risk of uterine rupture in women undergoing an induction TOL after Caesarean.[13] In 575 women with a previous Caesarean section, labour was induced with prostaglandin E2 gel (n = 172), intracervical foley catheter (n = 129), or amniotomy and (or) oxytocin (n = 274). The risk of uterine rupture was not increased in women who underwent either amniotomy/oxytocin or foley catheter induction but was significantly increased in those who underwent a prostaglandin E2 induction (P = 0.004).[13]

In 2003, Delaney and Young reported the examination of 3746 women with a prior Caesarean delivery who underwent either induced or spontaneous labour. They found that induced labour was associated with a greater risk of early postpartum hemorrhage, Caesarean delivery and admission to a neonatal intensive care unit. There was a trend toward a higher rate of uterine rupture, but this was not statistically significant.[14]

Conclusion

In conclusion, induction of labour is not contraindicated in women with previous Cesaren but there is considerable disagreement among clinicians on the various methods. Therefore, for success of IOL and fewer complications proper selection of patients, proper methods of induction and proper monitoring is essential.

References

1. WHO Statement on Cesarean Section Rates. April 2015
2. Birth after previous caesarean birth. RCOG. Green top guidelines No-45.Oct 2015.
3. Rosen MG, Dickinson JC, Westhoff CL. Vaginal birth after Cesarean section: a meta-analysis of morbidity and mortality. Obstet Gynecol 1991;77:465–70.
4. Ravasia DJ, Wood SL, Pollard JK. Uterine rupture during induced trial of labor among women with previous Cesarean delivery. Am J Obstet Gynecol 2000;183: 1176-9.
5. Appleton B, Targett C, Rasmussen M, Readman E, Sale F, Permezel M. Vaginal birth after Caesarean section: an Australian multicentre study. Aust N Z J Obstet Gynaecol 2000; 40: 87–91.
6. Shipp TA, Zelop CM, Repke JT, Cohen A, Caughey AB, Lieberman E. Intrapartum uterine rupture and dehiscence in patients with prior lower uterine segment vertical and transverse incisions. Obstet Gynecol 1993; 94:735–40.
7. Lyndon-Rochelle M, Holt VL, Easterling TR, Martin DP. Risk of uterine rupture during labor among women with a prior Cesarean delivery. N Engl J Med 2001; 345:3–8.
8. World Health Organzation: Methods of term labour induction for women with previous caesarean section. April 2015.
9. SOGC guidelines for vaginal birth after previous caesarean birth. Feb 2005.
10. Sondgeroth KE, Stout MJ, Graseck AS, et al. Progress of induced labor in trial of labor after cesarean delivery. Am J Obstet Gynecol 2015; 213:420.e1.
11. McDonagh MS, Osterweil P, Guise JM. The benefits and risks of inducing labour in patients with prior caesarean delivery: a systematic review. BJOG 2005; 112:1007.
12. Grobman WA, Gilbert S, Landon MB, et al. Outcomes of induction of labor after one prior cesarean. Obstet Gynecol 2007; 109:262.
13. Ravasia DJ, Wood SL, Pollard JK. Uterine rupture during induced trial of labor among women with previous Cesarean delivery. Am J Obstet Gynecol 2000; 183:1176-9.
14. Delaney T, Young DC. Spontaneous versus induced labor after a previous Cesarean delivery. Obstet Gynecol 2003; 102: 39–44.

Elective Induction of Labor: Changing scenario

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Elective induction of labor (IOL) is defined as artificial initiation of labor in pregnant women without clinically indicated medical or obstetrical condition. Vogel et al[1] has defined elective inductions as those inductions occurring in the absence of a clear maternal, fetal or medical indication and before 41 completed weeks of gestation. It is one of the most controversial procedures in obstetrics accounting for about 15% deliveries in United States. The ACOG[2], RCOG[3], SOGC[4] and NICE[5] discourage this practice.

Contributing factors

The factors contributing to the increase in the trend of this practice include caregiver convenience, maternal request for relief of discomfort or parental request for logistic issues or social reasons particularly choice of auspicious date for delivery. Other contributing factors are easy availability of cervical ripening agents and concerns for adverse perinatal outcome.

Advantages

Elective IOL offers several advantages to caregivers and mothers. Women prefer delivery during daytime as delivery is more likely to be attended by health professional. Women staying in remote areas may avert the risk of delivering outside a hospital setting. Proponents of elective IOL argue that obstetric complications can occur while waiting for a later gestational age like abruption, hypertension, rupture of membranes and cord prolapse. These conditions add to maternal and fetal morbidity, burden of time in labor and delivery units and costs of added interventions.[6]

Elective IOL and adverse perinatal outcome

For decades, IOL is reported to be associated with adverse perinatal outcomes. The concerns attributable to elective IOL are increased rate of caesarean delivery, postpartum haemorrhage, neonatal respiratory distress and neonatal intensive care unit admission. However, there is conflicting evidence and limited data on impact of elective IOL on perinatal outcome. The limitations of the data available so far is either the studies are underpowered, have used spontaneous labor rather than expectant management for comparison and absence of a universally accepted definition of medical induction versus elective induction.[7]

Maternal outcomes and Elective IOL

There is sufficient data that clearly shows that induction done at 41 weeks or more results in fewer caesareans and perinatal deaths. There is conflicting evidence about the relationship of elective induction at early term and full term gestations (37 0/7 to 40 6/7) and caesarean delivery. In a systematic review by Caughey et al[8], of the 9 RCTs compared elective IOL with expectant management and found that expectant management was associated with increased odds ratio for caesarean delivery (OR, 1.22;95% CI, 1.07-1.39; P<0.01). Stock et al[9] conducted a population based study and concluded that elective induction of labor at term can reduce perinatal mortality in developed countries without reducing the operative delivery. Vidya et al[10] conducted a small study from india and concluded that elective induction does not appear to pose an increased risk to the mother or her fetus in a carefully selected patient population. However, when associated with nulliparity, poor Bishop score, and estimated fetal weight of more than 3.5 kg, it has a statistically significant increase in cesarean rate.

Fetal outcomes of elective IOL

Current available evidence supports the policy of limiting elective induction before 39 weeks as fetal morbidity is of concern. Delivery beyond 39 weeks is beneficial to the newborn with lower risk of pulmonary immaturity, neonatal intensive care units and postnatal deaths. Ehrenthal et al[11] concluded that elective IOL has no impact on macrosomia whereas Stock et al[9] found the rate of shoulder dystocia was actually increased by elective IOL. Guidelines (ACOG) and quality metrics (eg Joint Commission, Leapfrog, National Quality Forum) have already been developed in US aimed at reducing non-indicated deliveries before 39 weeks.[12]

Challenges of elective IOL and expectant management

In the low and middle income countries, there is chronic shortage of health workers, constraint of budget for health expenditure and lack of essential life-saving interventions for mothers and newborns. Providing elective labor inductions in the setting of significant unmet need for medically indicated inductions poses

additional challenge with respect to human and clinical resources and costs. In a resource constraint setting, care must be prioritized based on urgency and medical need. This poses an ethical dilemma too. On the other hand, expectant management in these settings poses its own challenges due to lack of logistics for intensive monitoring of these patients during the waiting period and poor access of women to appropriate health facility.

Future work

First true prevalence, trends and determinants need to be established. Adequately powered and properly designed trials are required to establish the effect on maternal and perinatal outcomes. Most of the data available is from high resource tertiary centres or academic settings, and extrapolating this data on low resource setting is risky. Thus inputs of trials from this segment are essential to draw conclusions. In addition, safety of women and cost-effectiveness of the intervention also need to be addressed.

Conclusion

Despite the growing use of elective IOL and changing evidence with respect to its risks, critical issues on risks, benefits, safety and cost-effectiveness need to be addressed. Clear evidence about benefits in late term and post term is available but important gaps remain in our understanding of the optimal timing of induction at term. Routine induction for all women at term is likely to have many unintended consequences and considerable economic impact. A tempered approach, which balances the risk and benefits of IOL versus expectant management and does not encourage intervention without proven benefit, seems most prudent.[13] It should be performed only in context of informed consent, access to emergency obstetric care and appropriate monitoring and supervision.

References

1. Vogel JP, Gülmezoglu AM, Hofmeyr GJ, Temmerman M. Global perspectives on elective induction of labor. Clin Obstet Gynecol. 2014 Jun; 57(2): 331-42.
2. American College of Obstetrics and Gynecology (ACOG). Induction of labor. ACOG Practice Bulletin No. 107. Vol. 114. American College of Obstetrics and Gynecologists Obstetrics & Gynecology; 2009. p. 386-97.
3. Evidence-based Clinical Guideline No 9. RCOG Press; Jun. 2001 Induction of Labour. Royal College of Obstetricians and Gynecologists: Setting Standards to Improve Women's Health.
4. Crane J. Induction of labor at term SOGC Clinical Practice Guideline, No. 107. Journal of obstetrics and gynaecology Canada : JOGC = Journal d'obstetrique et gynecologie du Canada : JOGC. 2001; 23:717–28.
5. National Institute for Health and Clinical Excellence. Induction of Labour NICE Clinical Guidelines 70. London: National Collaborating Centre for Women's and Children's Health; 2008.
6. Gibson KS, Waters TP, Bailit JL. Maternal and neonatal outcomes in electively induced low-risk term pregnancies. Am J Obstet Gynecol 2014; 211:249.e1-16.
7. Getahun D. Epidemiologic considerations: scope of problem and disparity concerns. Clin Obstet Gynecol. 2014 Jun; 57(2): 326-30.
8. Caughey AB, Sundaram V, Kaimal AJ, et al. Systematic review: elective induction of labor versus expectant management of pregnancy. AnnIntern Med. 2009;151: 252–263.
9. Sarah J Stock, Evelyn Ferguson, Andrew Duffy, Ian Ford, James Chalmers, Jane E Norman. Outcomes of elective induction of labour compared with expectant management: population based study. BMJ. 2012; 344: e2838.
10. Ramasamy V, Thunga S, Nayak SR. Is elective induction safe? A prospectiveanalysis. J Obstet Gynaecol India. 2011 Dec; 61(6): 667-9.
11. Ehrenthal DB, Hoffman MK, Jiang X, et al. Neonatal outcomes after implementation of guidelines limiting elective delivery before 39 weeks of gestation. Obstet Gynecol. 2011; 118: 1047–1055.
12. Darney BG, Caughey AB. Elective induction of labor symposium: nomenclature, research methodological issues, and outcomes. Clin Obstet Gynecol. 2014 Jun; 57(2): 343-62.
13. Caughey AB. Elective induction of labour is associated with decreased perinatal mortality and lower odds of caesarean section at 40 and 41 weeks. Evid Based Med. 2014 Dec; 19(6): 236.

Chairpersons of Subcommittees of AOGD

Names of the Subcommittee	Nominated Chairperson for the year 2016-18
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Multidisciplinary Patient Management Subcommittee	Dr Jyotsna Suri
Rural Health Subcommittee	Dr Achla Batra
Breast Cancer Prevention Subcommittee	Dr Sunita Malik
Cervical Cancer Awareness & Prevention Subcommittee	Dr Mala Srivastava

Crossword - Labor

Compiled and designed by Megha Jindal[1], Sangeeta Gupta[2]
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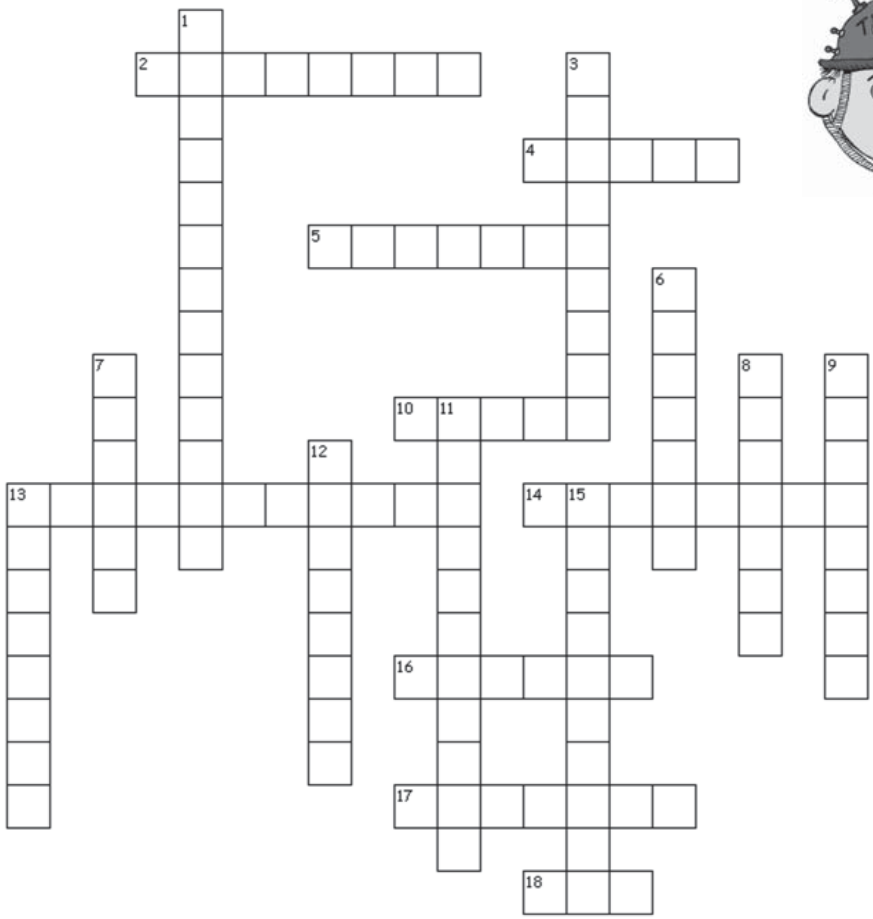

Across

2. Mechanical stretching of cervix enhances uterine activity. This phenomenon is known as _____ reflex
4. According to Hughes, precipitous labour terminates in expulsion of fetus in less than _____ hours
5. Third Leopold maneuver is also known as _____ grip
10. Acronym used for perineal tear classification _____
13. The most common cause of breech presentation is _____
14. In case of shoulder dystocia, the very first manoeuvre used along with suprapubic pressure is _____ manoeuvre
16. What is the name of the manoeuvre for controlled delivery of head by exerting forward pressure on chin while exerting pressure superiorly against the occiput by other hand _____ manoeuvre
17. In induction of labour for anencephalic fetus oxytocin is not preferred due to deficiency of ¬¬¬¬_____ in fetoplacental unit
18. Post placental IUCD is to be inserted within how many minutes of delivery of placenta ?

Down

1. If moulding leads to shortened suboccipit-obregmatic diameter then _____ diameter is lengthened
3. What is the incision given on cervix for delivery of after coming head of breech through incompletely dilated cervix _____ incision
6. During "Face to pubis" delivery the delivery of head occurs by _____
7. In accordance with latest WHO partograph, _____ phase of labour is not plotted
8. If ala of both sides are absent, a transversely contracted pelvis results known as _____ pelvis
9. The main focus of active management of third stage of labour is to prevent PPH by means of prophylactic administration of uterotonic agent immediately after the birth of baby, most preferred drug is ¬¬¬¬¬¬¬¬
11. _____ occurs when vertex fails to descend with sagittal suture in transverse diameter in pelvis
12. The sigmoid shaped curve of labour progress was given by _____
13. Name of the technique used for injection of oxytocin in intaumbilical vein in cases of manual removal of placenta
15. Excessive bleeding into myometrium and beneath the serosa in severe abruption may lead to _____ uterus

(..... answers on page 37)

Meconium Stained Liquor: Lot of fuss for a bit of poo..

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"All men commend patience, although few are willing to practice it" - Thomas Kempis

This is something which happens to us, obstetricians, when a baby passes meconium in labor. We become impatient, trying to do interventions, which are at times, unnecessary. Incidence of meconium stained liquor (MSL) is 2-3% at 37 weeks, 15% at 40 weeks and 30% at 42 weeks. However, meconium aspiration syndrome occurs in 2 out of 1000 live births.[1] Therefore, if Cesarean Section is performed for all MSL patients, 100 Cesarean sections will be required to prevent one Meconium Aspiration Syndrome. More so, passage of meconium in labor is a big management dilemma for obstetricians as it is associated with fetal acidosis, non-reassuring fetal heart patterns and low Apgar scores in some, whereas, is a normal physiological phenomena in others. In current era, when rates of Cesarean section are rising, judicious management of MSL is necessary, to prevent unnecessary Cesarean sections.

What is meconium?

There is very little known about meconium and whether it is a problem at all. Meconium is a mixture of mostly water (70-80%) and others like amniotic fluid, intestinal epithelial cells, lanugo, bile, mucus etc. Following are the postulated causes of passage of meconium prior to delivery:

- The fetal digestive system, when mature, the intestines start working leading to passage of meconium. This is the most common reason of meconium passage in term (15-20%) and post-term (30-40%) babies.
- Cord or head compression, leading to vagally mediated gastrointestinal peristalsis (the same reflex which causes variable heart rate decelerations), may lead to passage of meconium.
- Fetal distress, resulting in hypoxia and intestinal ischemia, relaxes the anal sphincter and increases gastrointestinal peristalsis leading to passage of meconium.

Significance of meconium

Significant meconium stained liquor is defined as either dark green or black fluid that is thick or tenacious, or any meconium stained fluid containing lumps of meconium.[1,2]

Meconium alone cannot be relied on as an indication of fetal distress: *"… meconium passage, in the absence of other signs of fetal distress, is not a sign of hypoxia…".*[3] An abnormal heart rate is a better predictor of fetal distress; and an abnormal heart rate with meconium may provide an even better indication of fetal distress. In addition, thick meconium rather than thin meconium is associated with complications.

The normal breathing efforts of a baby in utero are not sufficient to inhale particulate meconium in significant quantities. It is therefore reasonable to assume that if a baby has inhaled significant quantities of meconium it is because it has been gasping. Meconium can enter the lungs before or during birth but the subsequent hypoxia and persistent pulmonary hypertension that develops may be related to the meconium in the lung or more likely to the preceding hypoxia-ischemia. As long as meconium is not blocking the airway it may be relatively harmless.[3]

Monitoring during labor

Significant meconium liquor is an indication for continuous electronic fetal monitoring (EFM). Lightly meconium stained liquor alone does not indicate a requirement for continuous electronic fetal monitoring.[2] EFM should be considered for any light MSL depending on a risk assessment which should include the stage of labour, volumes of liquor, parity, the fetal heart rate etc. Electronic fetal monitoring should continue for at least one hour in all cases when meconium stained liquor is discovered. The indications of continuous electronic fetal monitoring in MSL in labor are as follows:[2]

- Significant meconium in liquor
- Abnormal fetal heart rate on intermittent auscultation (less than 110 beats per minute (bpm), greater than 160 bpm, presence of decelerations)
- Maternal pyrexia
- Fresh bleeding developing in labor
- Oxytocin use for augmentation
- Maternal request

Role of amnioinfusion

These days amnio-infusion, which was formerly proposed, is reconsidered in countries where midwives and obstetricians carefully monitor the fetal heart rate tracing during labor.[4] Amnioinfusion should not be used for the treatment of meconium stained liquor when strict fetal heart monitoring is possible.[2]

The WHO review from reproductive health library states that, in settings with limited intrapartum perinatal surveillance facilities, amnioinfusion in women with moderate or thick meconium staining of the amniotic fluid could improve some of the perinatal outcomes. The benefits might possibly be due to dilution of meconium or relief of oligohydramios.[5]

Role of fetal blood sampling

This test may aid in diagnosing fetal distress in pregnancies with MSL. Only caregivers trained in fetal blood sampling should perform the test. Being an invasive test, it is not used commonly.

Role of fetal scalp stimulation test (FSST)

It is an alternative and non-invasive method to fetal scalp blood sampling. This test comprises of a firm digital pressure on head or a gentle pinch of fetal head with atraumatic clamp for stimulation. An acceleration of the fetal heart rate of 15 bpm lasting at least 15 seconds is considered positive. Its role in meconium stained liquor is not clear. However, a study has shown poorer fetal prognosis, if FSST is negative in MSL.[6]

The obstetricians approach[7]

Since passage of meconium might be physiological and not associated with fetal distress, it is important that fetus who has passed meconium does not become stressed during labour and delivery, as it could lead to MAS.

- Avoid early amniotomy, so that, even if meconium is present it will remain well diluted.
- There should be documentation of meconium in medical record and partogram.
- Counseling the mother and family is necessary. Meconium is a variation and not necessarily a complication. Decision for further management should be well conveyed.
- There should be a holistic approach for management in light of clinical scenario. A postdated baby with old meconium should be managed differently than a 38 week baby with thick fresh meconium.
- Increased monitoring and intervention may be required if meconium is detected.
- There should be judicious use of oxytocin, else excessive use may lead to fetal distress.
- Early cord clamping is not recommended.
- Immediate suctioning is also not recommended.
- A practitioner trained in neonatal resuscitation should be present.

Cesarean section for meconium stained liquor[2]

There are no studies that address the effect on neonatal morbidity and mortality of immediate delivery by cesarean section when thick meconium is present or suspected early in labor. It has been recommended, however, that all labors with meconium-stained amniotic fluid should be continuously monitored and Cesarean section should be performed in cases of non-reassuring fetal heart rate.

Neonatal management[8]

Vigorous Neonate: Suctioning is not required if the neonate is term and vigorous at birth (i.e. good spontaneous respiratory effort, heart rate > 100 and good muscle tone) and the neonate can remain with the mother and have routine post birth care. Cleaning of mouth and nose, drying the neonate, stimulation and reposition as required are the steps to be carried.

Non-Vigorous Neonate: A non-vigorous neonate at birth shall receive laryngoscopy and tracheal suction under direct vision which shall be carried out immediately by the neonatologist. The neonate should not be stimulated at all, even by drying, until suction is completed. Tracheal suction is performed promptly and before any assisted or spontaneous respirations. The neonatologist should consider the potential benefits of suctioning meconium against the urgent need for other resuscitation methods. For suctioning, the meconium aspirator device is attached to the adapter of the endotracheal tube after intubation, then connected to a negative pressure source (not exceeding 100mmHg).

Monitoring in Postnatal Period: MAS can occur in infants who are in good condition at birth. It is therefore imperative that close observations are undertaken in the immediate postnatal period.

These infants require 4 hourly observations of:

- Respiratory effort
- Temperature
- Heart rate
- Respiration

A newborn needs to be reviewed by a neonatologist if any of the following signs are demonstrated:

- Tachypnoea
- Cyanosis
- Variable hyperinflation or audible expiratory noises (grunting)
- Newborn's condition causes concern at any time

Maternal and fetal outcome: Thin meconium has similar outcomes when compared to clear liquor in labor. Prolonged labor was more common and associated with a particularly worse outcome in women with thick MSL. Caesarean sections rates are increased; failure to progress being the indication in most of the cases. Mean Apgars are lower and the proportions of neonates with poor Apgar scores are higher in cases of thick meconium, but not with thin meconium.[9] Meconium aspiration syndrome is the cause of 2% cases of perinatal mortality. Complications of meconium aspirations are respiratory failure, persistent pulmonary hypertension of the newborn and pneumonia/ sepsis. These are major causes of morbidity and mortality.[10] MSL has reported to be associated with an increased risk of cerebral palsy and death according to a study.[11] MSL is a significant risk factor for neonatal encephalopathy.[12]

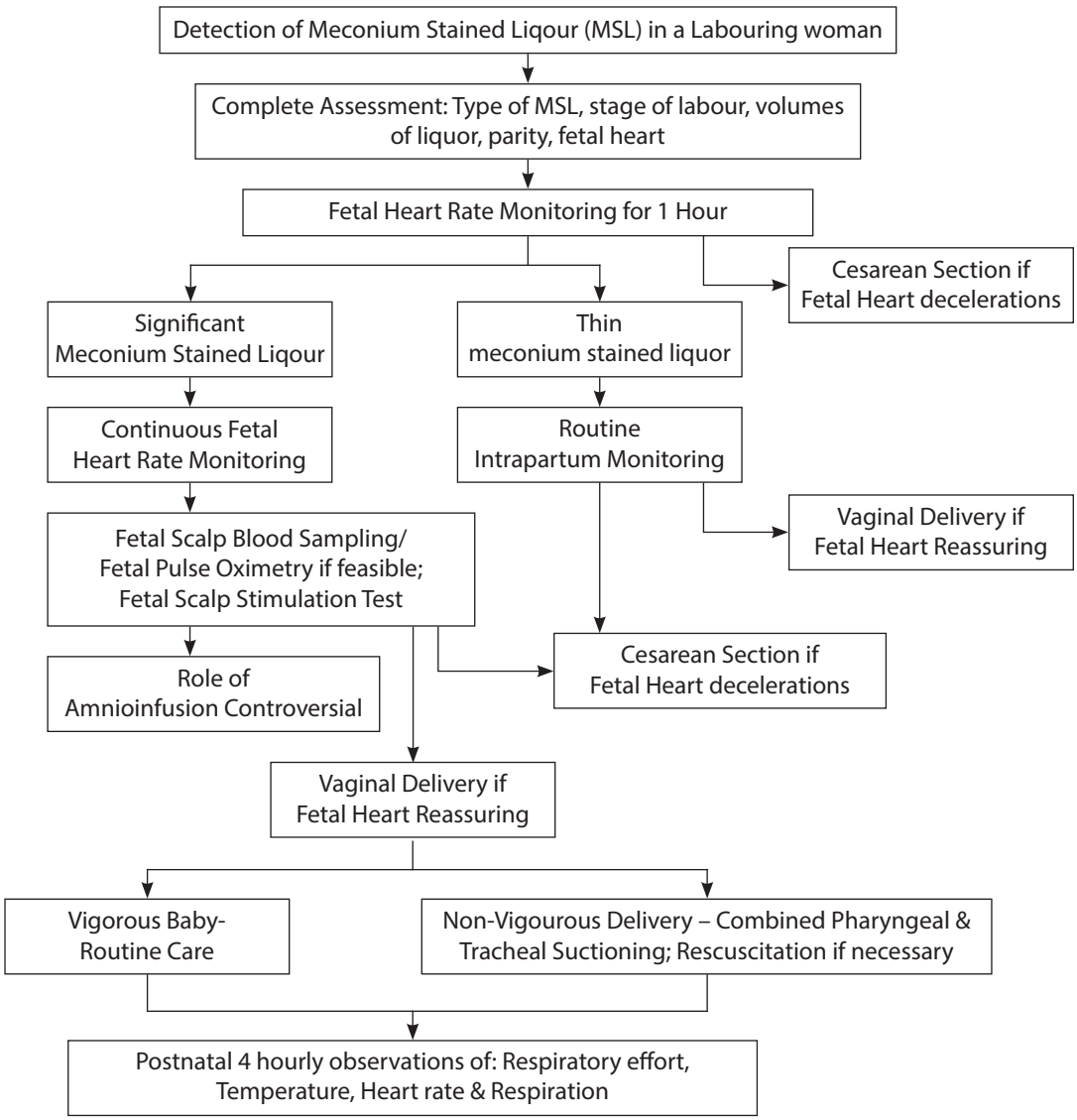
Conclusion

Detecting meconium in amniotic fluid initiates a cascade of event. A CTG machine is often strapped onto the woman reducing her ability to move, discontinuation of oral intake, and increasing risks of having induction or augmentation of labor and therefore, Cesarean section. As the baby may be subjected to airway suctioning after birth, it can cause a vagal response and difficulties with breastfeeding. Baby might require intensive monitoring and admission to nursery. Indeed, many of the interventions implemented because of meconium are, more likely, the cause of complications than the meconium itself. There is a lot of fuss for a bit of poo; which, in the vast majority of cases, is not a problem per se.

References

1. Sithembiso V, Dharmapuri V. Intrapartum and post delivery management of infants born to mothers with meconium stained amniotic fluid: evidence based recommendations. Clinical Perinatology 2006; 29-42
2. National Institute for Health and Clinical Excellence Intrapartum Care: care of healthy women and babies during childbirth. NICE Guidelines 2007
3. Unsworth J, Vause S. Meconium in Labour. Obstet Gynaecol Reprod Med. 2010; 20(10): 289-94
4. Chabernaud JL. Intrapartum and postdelivery management of infants born to mothers with meconium-stained amniotic fluid. Arch Pediatr. 2007; 14(11): 1389-93.
5. Sangkomkamhang US and Lumbiganon P. Amnioinfusion for meconium-stained liquor in labour: RHL commentary (last revised: 1 October 2011). The WHO Reproductive Health Library; Geneva: World Health Organization.
6. Rathore, A. M., Ramji, S., Devi, C. B., Saini, S., Manaktala, U. and Batra, S. (2011), Fetal scalp stimulation test: An adjunct to intermittent auscultation in non-reassuring fetal status during labor. Journal of Obstetrics and Gynaecology Research, 37: 819–824.
7. Meconium Stained Amniotic Fluid Guidelines. Obstetric Clinical Guideline Group, December 2008.
8. Wiswell TE, Gannon CM, Jacob J. Delivery Room Management of the Apparently Vigorous Meconium Stained Neonate: Results of the Multicenter International Collaborative Trial. Pediatrics 2000; 105:1-7.
9. Saunders K. Should we worry about meconium? A controlled study of neonatal outcome. Trop Doct. 2002; 32(1): 7-10.
10. Bhutani, V.K; Chima. R; Sivieri. E.M (2003) Innovative Neonatal Ventilation and Meconium Aspiration Syndrome. Indian Journal of Pediatrics. 2003 May 70 (5) 421-7.
11. Gaffrey, G; Flavell, V; Johnson, A; Squier, M; Sellars, S (1994b) cerebral palsy and neonatal encephalopathy. Arch dis Child neonat Ed 1994; 70: 195-200
12. Adamson SJ, Alessandri LM, Badawi N, Burton PR, Pemberton PJ, Stanley F. Predictors of neonatal encephalopathy in full term infants. BMJ 1995; 311: 598-602.

Algorithm for management of MSL



Congratulations

MAMC awarded prize for maximum number of Cu-T insertions and tubal ligations performed vis-a-vis total deliveries by Mission Director, Central District on 21st March 2016

Journal Scan

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Abstract of the research articles are available free at the journal websites and on PubMed (http://www.ncbi.nlm.nih.gov/pubmed). A summary of the articles has been provided so as to put the findings of the articles into perspective for current clinical practice.

Impact of uterine closure on residual myometrial thickness after cesarean: a randomized controlled trial.

Stéphanie Roberge, Suzanne Demers, Mario Girard, Olga Vikhareva, Stéphanie Markey, Nils Chaillet, Lynne Moore, Gaétan Paris, Emmanuel Bujold

Citation: Roberge S, Demers S, Girard M, Vikhareva O, Markey S, Chaillet N, Moore L, Paris G, Bujold E. Impact of uterine closure on residual myometrial thickness after cesarean: a randomized controlled trial. Am J Obstet Gynecol. 2016 Apr; 214(4):507.e1-6.

Study Question: Do different techniques of uterine closure affect healing of the uterine scar after cesarean section?

Methods

- A double-blind randomized controlled trial that compared three different techniques of uterine closure in 81 women with singleton pregnancies undergoing elective primary cesarean delivery at >38 weeks' gestation. The sample size was calculated so as to observe an estimated difference of 1.5 mm of residual myometrial thickness (RMT) between uterine closures and allow for 10% of loss to follow-up.
- The techniques compared were- (1) single layer locked, including the decidua (controls); (2) double layer with the first layer locked including the decidua and the second layer unlocked and imbricating the first layer; and (3) double layer with the first layer unlocked, excluding the decidua and including the deep part of the myometrium, and the second layer unlocked including the remaining part of the myometrium. All the three techniques involved continuous sutures (polyglycolic acid, size: 0).
- Additional operative information was collected - (1) need for additional suture; (2) closure/non closure of the vesicouterine and parietal peritoneum; (3) duration of surgery. Birthweight, estimated blood loss, and postpartum endometritis were also noted.
- Transvaginal sonography (TVS) was done 6-12 months after cesarean section to assess the position of the uterus (anteverted or retroverted), RMT, and total myometrial thickness (TMT) above the uterine scar.
- Primary outcome was the mean RMT assessed at TVS. Secondary outcomes included: prevalence of severe scar defect defined as the RMT <2.3 mm, mean anterior TMT, healing ratio defined as the RMT x 100/ TMT, mean operative time, mean estimated blood loss at the time of the cesarean, need for and number of additional hemostatic sutures, and prevalence of postpartum endometritis.
- Intent-to-treat analyses using Student test were performed to compare each double layertechnique to the single-layer closure for primary outcome and other continuous variables.

Results

1. Double-layer uterine closure with a first unlocked layer excluding the decidua, compared to locked single-layer closure including the decidua, was associated with
   - A greater RMT (6.1$\pm$ 2.2 mm vs 3.8 $\pm$ 1.6 mm; $P<$ 0.001),
   - A greater TMT (9.5 $\pm$ 1.7 mm vs 8.2 $\pm$ 2.1mm; $P=$ 0.025) and
   - A greater healing ratio (73 $\pm$ 23% vs 54 $\pm$ 20%; P =0.004).
2. Double-layer closure with locked first layer was not significantly different than single-layer closure in
   - RMT (4.8 $\pm$ 1.3 vs. 3.8 $\pm$ 1.6 mm; P = 0 .032),
   - TMT (9.4 + 2.3mm vs. 8.2 $\pm$ 2.1mm; P= 0.074)
   - Healing ratio (60 $\pm$ 21% vs. 54 $\pm$ 20%; P = 0.287).

The trial lacked the power to demonstrate differences in operative time, need for additional sutures, or blood loss among the three techniques.

Conclusion

Double-layer closure with unlocked first layer excluding the decidua is associated with better uterine scar healing than locked single layer including the decidua.

Perspective

Over the years rate of cesarean delivery has increased world over and so has the incidence of placenta accreta and other late complications of cesarean delivery. Evidence suggests that this could be related to the method of uterine closure during cesarean. Traditionally uterine incision was closed in double layers. Then came studies advocating single layer uterine closure. Some studies still found double layer closure to be better [Bujold E et al, Obstet Gynecol. 2010; 116:43-50]. At present, there is no consensus on the method of uterine closure. The present study addressed three aspects of the technique of uterine closure- (1) single or double layer, (2) locking or non-locking sutures and (3) inclusion or exclusion of decidua in suturing. The impact on healing was assessed on the basis of TVS findings of the uterine scar. However, as discussed by the authors, sonographically assessed scar thickness and healing is still a surrogate marker for the prediction of uterine rupture and other adverse outcomes like placenta accreta. A follow up of outcomes in subsequent pregnancies would provide more convincing answer to the question as to which technique of uterine closure leads to better healing and minimizes long term complications after a cesarean delivery.

Infant Outcomes after Elective Early-Term Delivery Compared With Expectant Management.

Jason L. Salemi, Elizabeth B. Pathak, and Hamisu M. Salihu

Citation: Salemi JL, Pathak EB, Salihu HM. Infant Outcomes after Elective Early-Term Delivery Compared With Expectant Management. Obstet Gynecol. 2016 Apr; 127(4): 657-66.

Study Question: Does the risk of neonatal morbidity and infant mortality differ between elective early-term (37-38 weeks of gestation) deliveries and those delivered at 39 weeks of gestation or greater?

Methods

A population-based retrospective cohort study of 675,302 singleton infants born alive at 37–44 weeks of gestation from 2005 to 2009 in more than 125 centers in Florida. The study used data from a statewide maternal and infant longitudinally linked database.

The study population was categorized into exposure groups based on the timing and reason for delivery initiation—four subtypes of deliveries at 37–38 weeks of gestation and a comparison group of expectantly managed infants delivered at 39–40 weeks of gestation. The final exposure consisted of five levels:

- Electively induced delivery, 37–38 weeks of gestation;
- Elective cesarean delivery without a trial of labor, 37–38 weeks of gestation;
- Spontaneous delivery, 37–38 weeks of gestation;
- Medically indicated delivery, 37–38 weeks of gestation (if a medical complication occurred only at delivery and was not present before or during pregnancy);
- Delivery at 39–40 weeks of gestation after expectant management (full-term).

The primary outcomes included respiratory morbidity, neonatal sepsis, feeding difficulties, and neonatal intensive care unit (NICU) admission

Results

Compared with the full-term group, after adjusting for confounders, infants born after early induction did not have increased odds of respiratory morbidity, neonatal sepsis, or NICU admission but did experience slightly increased odds of feeding difficulty (odds ratio 1.18, 99% confidence interval 1.02– 1.36). Infants in the early cesarean delivery group, on the other hand, were at 13–66% higher odds of all four morbidity outcomes. Survival experiences were similar when comparing early inductions and early cesarean deliveries with the expectant management group.

Conclusion

This study cautions against a general avoidance of all elective early-term deliveries.

Perspective

Several studies have reported poorer neonatal outcomes for early-term (37–38 weeks of gestation) compared with later term (39 weeks of gestation or greater) deliveries leading to a change in clinical practice. This study reported the findings of a large population-based study and concluded that elective induction before 39 weeks of gestation was not associated with an increased likelihood of adverse neonatal outcomes. However, study supports the avoidance of purely elective cesarean deliveries before 39 weeks of gestation. The study did not capture information on fetal deaths that may have occurred while expectantly managing pregnancies at 37–38 weeks of gestation until 39 weeks of gestation or greater. Maternal outcomes were not studied. The authors also mentioned that it is not known whether the outcomes can be generalized to women with different racial–ethnic background. The issues that surround the timing and reasons for delivery initiation either by induction of labor or elective cesarean are complicated. The decision in this matter needs to be individualized.

HISTORY UNVEILED

The Birth of 'A Birth Aid'… Forceps!

Nilanchali Singh
Assistant Professor, Obstetrics & Gynecology, Maulana Azad Medical College & Lok Nayak Hospital, New Delhi, India

History is the only true teacher.. the one which brings out the best..

We live in a time of rapid change, a time of progress. However, history plays an important role in leading to this progress. Medicine has evolved tremendously over the years. It is interesting to know how history has molded medicine, over the years. History dictates what answers we seek for existing problems. By learning about the causes and effects of events in history, one may learn better ways to deal with current conflicts. Apart from imparting wisdom, going through pages of history is, also, very interesting. We tend to bring forth some interesting historical myths and facts, pertaining to obstetrics and gynecology in this section, 'History Unveiled'. As per the theme of current issue, I have compiled interesting history of forceps usage over the years.

Ancient Era

The history of Obstetrical forceps dates back to Vedic era, with the firth writings related to unpaired and paired instruments reported back in 1500 B.C. The earliest mention of forceps delivery in the Vedic period is 'Ankush'. Similarly, Egyptian, Greek, Persian and Roman writings mention forceps in writing and paintings. (Figure 1, 2) Wall-carvings in the temple of Kom Ombo (250 B.C.) have been widely cited as an example of forceps usage; but are almost certainly instruments for sacrificial rites.

Figure 1: In 1937, Baglioni displayed a bas relief from the 2nd century A.D., showing the use of forceps of relatively modern design in Rome; however, later on it was found out to be a hoax.

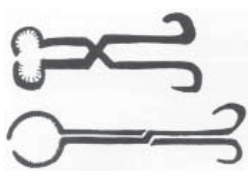


Figure 2: There is also mention of forceps described by Albucasis in ancient era, which had teeth on inner surface for dead fetus.

Middle ages

Midwifery was primitive and mainly controlled by the church in Middle Ages. Only few cloistered celibate were considered pure enough to discuss midwifery. Midwifery practice was immersed in folklore and superstition. Labor management included herbal preparations and fumigations. Mechanical devices were used for delivery assistance occasionally, in cases of obstructed labour. *Instruments were borrowed from surgeons and lithotomists, or at times, utensils from the kitchen were used*. There was no concern of the fetus as it was by now, already dead, due to prolonged obstruction.

Figure 3: When there is a difficult labour with a dead child, place the patient in a sheet held at the corners by four strong men, with her head somewhat elevated. Have them shake the sheet vigorously by pulling on the opposite corners, and with God's will she will give birth.

Many midwives gave instruction on the management of difficult labour. Few were committed to print. An example is Dame Trot, practicing in Salerno in the 11th century. (Figure 3) However, with the 16th century new ideas began to evolve and this is where the story of the obstetric forceps really begins, with the activities of the Chamberlen family.

The Chamberlen family

In the 16th century, the French Protestent, William Chamberlen fled to England from France as the Queen, Catherine de Medici banned Protestant physicians. With the flee of this Frenchman with his two sons Peter Chamberlen, 'the elder', and Peter Chamberlen, 'the younger', the story of forceps as an instrument to deliver live infants began.

Peter, the elder and Peter, the younger were the epitomes of 'man-midwifery'. Whilst it is not entirely

clear which of the brothers invented forceps, it is often accredited to Peter, the elder who became surgeon to the Queen of England. The eldest son of Peter, the younger, Hugh, is credited with continuing the tradition of practising midwifery and the use of forceps.

Their quest to protect their invention led to extensive means of concealment. The instruments themselves were always carried in a gilded chest and revealed once the woman had been blindfolded. The birth subsequently took place under blankets with only the Chamberlens in attendance of the patient. It was through these elaborate measures that the Chamberlens were able to keep the secret of forceps for nearly a century.

In 1670, however, Hugh Chamberlen visited Paris with the intention of selling the secret to the French government in the hope of raising funds. In doing so he was challenged by Francois Mauriceau to deliver a 38-year-old rachitic dwarf with a grossly deformed pelvis who had been in obstructed labour for eight days. Unable to deliver her successfully, Hugh journeyed back home with the secret still intact.

He, later, flee to Holland where he is said to have sold some instruments to a Dutch Obstetrician named Van Roonhuysen. Despite this, models of forceps started to appear from the early 18th century including a flexible model with a lever from the Van Roonhuysen family, which again was kept secret for many years.

Figure 4: William Chamberlein. It still remains a secret who invented the forceps, he himself or one of his sons. Forceps itself was kept a secret for centuries.

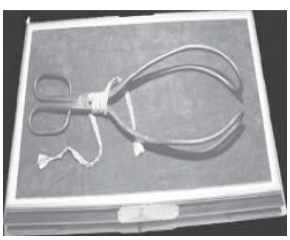


Figure 5: The original Chamberlen instruments were only discovered in 1813 under the floorboards of the attic in their Essex residence, hidden by Peter the younger's wife Ann.

Discovery of 'The Lock, The Pelvic Curve and The Rules'

William Smellie was the most significant obstetrician in the middle of 18th century. He developed his own forceps to prevent loss of children. His instruments included the 'English lock' to allow the blades to be inserted separately into the vagina. He was the one who introduced a pelvic curve. The blades were covered in leather and later thinner material and lubricated with hogs lard to ease insertion and prevent transmission of venereal infection. The rules of applying forceps were developed by Smellie and in 1752 he published the 'Treatise on the Theory and Practice of Midwifery'. Although a great teacher, he was met with violent opposition from some midwives, most notably Elizabeth Nihell who described him as a 'great horse God-mother of a he-midwife'. Although he was familiar with forceps he prided himself in rarely using them, carrying with him a pair of rusty forceps to emphasize their infrequent use.

Figure 6: 'Lobster Forceps' designed by Smellie. His fiercest critics was Dr Burton of York, who claimed that this forceps with a screw mechanism to appose the blades were 'better than any yet'

Tragedies associated

Forceps as a method of obstetric practice was strongly condemned after the tragedy of Princess Charlotte, daughter of King George IV. She died in labour at the age of 21 in 1817. Labour lasted for 50 hours. The baby was, obviously, a stillborn. Princess Charlotte died secondary to a concealed haemorrhage, thus leaving the King without an heir. The management of the labour in which forceps had been kept on standby but were not used was widely criticized. The incident costed life of the obstetrician in charge, Sir Richard Croft, who, later shot himself in his forehead.

Use of forceps as satirical instrument

Forceps has been used in history as a Satirical Instrument. Two examples of this satire are: satire on Martin Van Buren, ex-vice-president of United States of America and Grevy, ex-president of France.

Figure 7: Political satire featuring an ex-president of France, Grevy, and a midwife holding a newborn child by the head in a large pair of forceps.

Figure 8: A satire on Van Buren administration challenged by presidential candidate of US, William Henry Harrison, who is dressed as woman and dethroning Van Buren with a forceps.

Development of modern forceps

Use of forceps were largely restricted at dealing with deliveries in small deformed pelvises resulting in cephalo-pelvic disproportion and prolonged labor in 19th century. Various forceps were designed with focus on axis traction devices. Long forceps were used in Europe and became popular in England. The designs of JY Simpson, Barnes and Anderson gained maximum popularity. With advent of safe Cesarean Section in 20th century, emphasis was put onto fetal well-being, high application of forceps became a rarity.

The Kielland's forceps was however designed in 1916 in Norway, to aid in deep arrests. These forceps were straight with a slight pelvic curve that was parallel to the axis of the handle with a sliding lock. This instrument gained popularity worldover, but not in place of its birth i.e. Norway. Shorter forceps like modified Simpson's forceps and Wrigely's forceps (1935) were developed later to protect fetal head and aid in arrests at lower stations.

The Indian contribution

Sir Kedar Nath Das (1867-1936) was an eminent obstetrician and medical educator of India, who pursued all his medical education in India. He was an author of many publications in national and international journals, including 'Handbook of Obstetrics' and 'Textbook of Midwifery'. He devised a long curved obstetrics forceps, especially for Indian women with smaller pelvis and babies have lower birth weight. He is also an author of 'Obstetrics Forceps: Its history and evolution', which was later published in many foreign languages.

Figure 9: Sir Kedar Nath Das, who designed the Das variety of Long Forceps for Indian Females

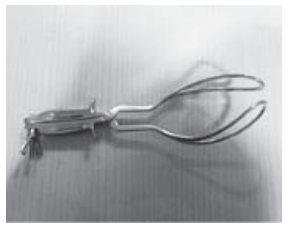


Figure 10: Forceps designed by Kedarnath Das

Conclusion

Obstetrics has come a long way over the ages. A lot of emphasis is on 'Safe Obstetric Practices'. With emergence of safer techniques of Cesarean Section and anaesthesia, forceps delivery has taken a down troll. Due to infrequent usage of forceps, training of young obstetricians in forceps delivery is largely, lacking. However, in areas of low resource settings and even, at times, in areas with all the resources, optimal use of forceps may decrease fetal and maternal morbidity and mortality. Hence, trained obstetricians should pass on this legacy to the budding obstetricians and prevent this 'Dying Art' from dying!

If you stop learning, you stop creating history and become history. –Anonymous

Reference

1. Sheikh S, Ganesaratnam I, Jan H. Birth of Forceps. J R Soc Med Sh Rep 2013: 4: 1–4.
2. Hibbard B. *Milestones in the evolution of the obstetric forceps*. Retrieved from http://www.lmi.org.uk/Data/10/Docs/18/18Hibbard.pdf.

Proceedings of the AOGD Monthly Clinical Meeting, Indraprastha Apollo Hospital, New Delhi, 29th April, 2016

Case - 1

Laser Photocogulation for stage III Twin-twin transfusion Syndrome

Anita Kaul, Chanchal Singh, Ranjana Sharma

A 26-year-old G2P0 with spontaneously conceived monochorionic diamniotic (MCDA) twin pregnancy was referred to the Fetal Medicine unit at Indraprastha Apollo Hospital at 26 weeks gestation in view of twin twin transfusion syndrome (TTTS). Ultrasound confirmed the diagnosis of stage III TTTS – there was oligo-polyhydramnios sequence, donor bladder was not seen and there was reversal of end diastolic flow (REDF) in the stuck donor twin alongwith increased pulsatility index (PI) in the ductus venosus of the recipient. The options of expectant management, immediate delivery vis a vis selective Laser coagulation of placental vessels (SLCPV) was discussed with the couple. Since expectant management would invariably lead to fetal demise with antecedent high risk of neurological morbidity in the surviving twin and high perinatal morbidity and mortality with iatrogenic premature delivery at this early gestation, the couple opted for Laser photocoagulation. The cervical length prior to the procedure was 32 mm on transvaginal scan. Fetoscopic Laser dichorionization under local anesthesia was done within 12 hours of referral. Fetal donor bladder was visible within 24 hours of the procedure. However, the smaller donor had an intrauterine fetal demise 1 week following the procedure. Follow up ultrasound at 4 week post procedure did not show any abnormal brain changes in the surviving twin. Fetal growth for surviving twin was on the 6th centile for gestation with normal amniotic fluid volume and fetal Dopplers. The patient was admitted with decreased fetal movements at 35 weeks. She was delivered by emergency LSCS as CTG was abnormal. A baby girl weighing 2260 grams was born with an Apgar of 7,8. The macerated co-twin delivered at the same time weighed 427 grams. The mother was discharged at postop day 3. The baby went home well after an NICU stay of 5 days and is currently doing well at 16 months of age with normal developmental milestones.

Discussion: TTTS occurs in 10-15% of monochorionic twin pregnancies. Diagnosis is based on ultrasound. Perinatal mortality for untreated TTTS is as high as 80% with an overall survival rate of 27% alongwith 25% rate of neurological damage in the survivors. Evidence suggests that the best way to improve survival without neurological impairment in TTTS is Laser photocoagulation (Level I evidence).[1,2] Laser corrects the underlying etiology and protects the surviving co-twin even when there is intrauterine demise (IUD) of one twin.

References

1. Senat MV, Deprest J, Boulvain M, et al. Endoscopic laser surgery versus serial amnioreduction for severe twin-to-twin transfusion syndrome. N Engl J Med 2004; 351(2): 136-144.
2. Roberts D, Neilson J, Kilby MD, Gates S. Interventions for the treatment of twin-twin transfusion syndrome. Cochrane Database of Systematic Reviews 2014, Issue 1. Art. No.:CD002073. DOI:10.1002/14651858.CD002073.pub3

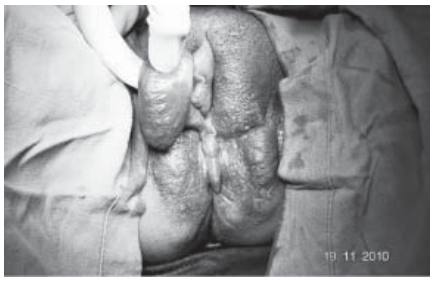
Case - 2

Vulvitis Granulomatosa

Shweta Rajput, S.B. Khanna, Kuldeep Singh, Deepshikha Arora , Kiranbala Dash

Vulvitis granulomatosa is chronic inflammatory hypertrophy of the vulva. It is clinically characterised by painless swelling of the vulva and histologically demonstrated by non caseating granulomas extending deep into dermis.

A 25 yr old unmarried girl came to IAH in february 2016 with complains of pain and itching in the perineal region. On examination multiple ulcers were found on the perineum, extending upto the intergluteal region. She was apparently normal till 2010 when she first developed painless enlargement of labia majora and came to apollo hospital. After thorough investigations she was taken up for surgery



She underwent debulking of labia majora with reconstruction of labia minora and clitoris. The tissue which was sent for HPE reported it to be vulvitis granulomatosa.

She again came to IAH in november 2015 with similar complains of gradual enlargement of the vulva for 2 yrs which was associated with pain and vaginal discharge. On examination bilateral labia majora was enlarged with overlying skin being hypertrophied, hyperpigmented, indurated with multiple erosive ulcers. Also a multilobular growth was seen arising from clitoris. She again underwent surgery in which simple vulvectomy followed by vulval reconstruction was done. The tissue sent for HPE reported it to be vulvitis granulomatosa. In february 2016 when she came with similar complains and multiple ulcers were found in the perineal region, she was treated conservatively with local dressings, after ruling out other infective causes.

Vulvitis granulomatosa is a rare disorder and is considered to be genital counterpart of cheilitis granulomatosa. It is a diagnosis of exclusion and lupus vulgaris, sarcoidosis, mycobacterial infections and crohn's disease needs to be ruled out before reaching to the conclusion. Histological features of vulvitis granulomatosa, Cheilitis granulomatosa and crohn's disease are almost similar, thus differentiation is very difficult.

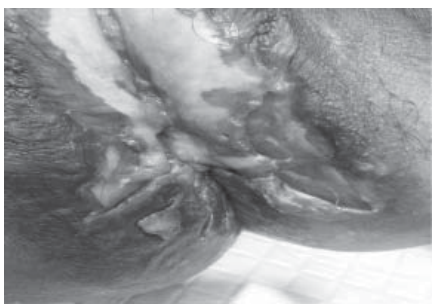


Figure: Multiple ulcer on vulva

Very few cases of vulvitis granulomatosa have been reported so far. No definitive cause and treatment for it has been identified yet., recurrence is very common. Surgical correction has been found to provide symptomatic relief to the patient. Other treatment options are steroids and antibiotics like metronidazole. Recently immunosuppresants like azathioprine and anti TNF monoclonal antibodies like infliximab have been used in the treatment of vulvitis granulomatosa, with promising results.

Case - 3

Congenital Heart Block

Karuna Ratwani , Geeta Chadha, Anita Kaul

A 29 year old G2A1 was referred to IAH at 27 weeks in view of fetal complete heart block (CHB) at 27 weeks. Her previous antenatal care was at Agra where CHB was diagnosed at anomaly scan at 20 weeks. The patient had opted for a second trimester termination in her last pregnancy for the same condition and had not had any further investigation. Maternal investigations showed raised ESR and Anti Ro and Anti La antibodies were positive. The mother was proactively asked about past symptoms and she gave history of dry mouth, recurrent febrile episodes with swelling of salivary glands. A diagnosis of Sjogren's syndrome was made. Ultrasound and fetal echocardiogram confirmed the diagnosis of complete heart block with a fetal ventricular rate of 54 beats per minute. Fetal growth was on the 10th centile with oligohydramnios but end diastolic flow (EDF) was present in the umbilical artery and good fetal movements were present. The patient was managed by a multidisciplinary team comprising of obstetrician, fetal medicine specialist, pediatric cardiologist, rheumatologist and neonatologist. The mother presented with high grade fever at 28 weeks and was managed by IV antibiotics, steroids and hydroxychloroquine. The fetus was followed up with 2 weekly ultrasound. The mother was started on oral dexamethasone and salbutamol; however salbutamol was stopped as mother could not tolerate it and dexamethasone was stopped due to worsening oligohydramnios and maternal hypertension. An elective LSCS was done at 36 weeks and a baby girl weighing 1.68 kg was delivered with an Apgar of 8,9. Baby had bilateral clubfeet (most likely due to persistent oligohydramnios) which is being managed by massage and cast. The baby did not require pacing and is doing well at 1 month age. The mother had another flare in the postpartum period and was managed by IV steroids and HCQS. She is currently well.

Tickle the Funny Bone

Compiled by Sangeeta Gupta[1], Nilanchali Singh[2], Pushpa Mishra[3]
[1]Professor, [2]Assistant Professor, [3]Specialist, Department of Obstetrics & Gynecology, Maulana Azad Medical College, New Delhi, India

Laughter is strong medicine for mind and body.

Recognizing your Patient

One pretty lady meets her gynec doctor in a party and introduces herself and says "Do you remember me?" The doctor gives a naughty smile and replies – "Sorry mam, I can't remember you by just seeing your face."

Keep your Identity

A middle-aged woman had a heart attack and was taken to the hospital. While on the operating table she had a near death experience. Seeing God, she asked, "Is my time up?" God said, "No you have another 43 years, 2 months and 8 days to live."

Upon hearing this, the woman decided to stay in the hospital and have a face lift, liposuction and a tummy tuck. She even had someone change her hair color. Since she had so much more time to live, she figured she might as well make the most of it.

She was released from the hospital but while crossing the street on her way home, she was killed by a car.

Arriving in front of God, she demanded, "I thought you said I had another 43 years?! Why didn't you pull me out of the path of that car?"

God replied, "I didn't recognize you."

Remember these words..

- Smile
- Count your blessings
- When you hear laughter, move toward it
- Spend time with fun, playful people
- Bring humor into conversations

Answers: Crossword - Labor

Across

2. Ferguson	10. OASIS	16. Ritgen
4. Three	13. Prematurity	17. Estriol
5. Pawlik's	14. McRobert	18. Ten

Down

1. Mentovertical	7. Latent	11. Asynclitism	15. Couvelaire
3. Duhrssen's	8. Robert's	12. Friedman	
6. Flexion	9. Oxytocin	13. Pipingas	

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