EOGS BULLETIN

MAR 2019

"# Safe Motherhood"
Let Us Reduce Maternal Mortality
By Detecting And Treating High Risk Pregnancies

Dr Nandita Palshetkar,
FOGSI President

WE FOR स्त्री
Safer • Stronger • Smarter

ERODE OBSTETRIC AND GYNAECOLOGICAL SOCIETY

Affiliated to FOGSI - Since 1986.
TN Societies Registration SI No. 144/2018

Office Bearers 2019 - 20

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Monthly Bulletin of Erode Obstetric and Gynecological Society
Editors: **Dr. E.S. Usha & Dr. N. Poongothai**

ERODE OBSTETRIC AND GYNAECOLOGICAL SOCIETY
ACADEMIC MEET

TOPICS

02.30 - 04.00 pm : **Dr. Hitesh Bhatt J** MD., PGDMLS
Medicolegal Expert, Mumbai.
Obstetrics and Gynecology litigations
How to keep ourselves safe?

04.00 - 04.30 pm : **Dr. TV Chitra, PSGIMSR**
Progesterone Vs Cervical encirclage in the management of preterm labour

Date : **17.03. 2019, Sunday**
Time : **2.30 pm - 4.30 pm**
Venue : **Atrium Hotel, Erode**

Lunch : 1.30 pm - 2.30 pm

Sponsored by ***Sun Pharma Spectra Division***

Message from the President & Secretary

Dear Colleague,

Happy Women's Day...

International Women's Day (March 8) is a global day celebrating the social, economic, cultural and political achievements of women.

The theme for International Women's Day 2019 is **"Think equal, build smart, and innovate for change."**

Our FOGSI President insists on reducing maternal mortality by detecting and treating high risk pregnancies. Let us all have clear protocols for identifying and managing the high risk antenatal women. When the woman requires advanced treatment than available in our centre we should shift them safely to higher centres.

This month's meeting by DR Uma Ram and DR Amritha was well attended. Dr Sangeetha presented case scenarios in our monthly meeting.

Dr Vanithasri has continued her article on Hypertension in pregnancy. We request more members to come forward to share their experiences in the meeting and through the Bulletin.

Thank all of you once again.

With regards,

Dr. E.S. Usha MD., DGO.,
Fellow in fetal medicine
President EOGS

Dr. Sri Revathy Sadasivam MD (OG)., DNB (OG)., MICOG., MRCOG (UK)., Masters in Reproductive Medicine (UK)
Secretary EOGS

INTRAPARTUM MONITORING GUIDELINES

Electronic fetal monitoring documentation is an everyday part of obstetric healthcare and is one of the most crucial aspects of validating the provision of quality care.

Electronic Fetal Monitoring vs intermittent auscultation

WHO recommendations on Intrapartum care

- Continuous cardiotocography is not recommended for assessment of fetal well-being in healthy pregnant women undergoing spontaneous labour.
- Continuous CTG should not be used as a substitute for providing supportive, woman-centred intrapartum care.

Indications for EFM

Group 1

- Suspected chorioamnionitis
- Severe hypertension
- Oxytocin use
- Significant Meconium
- Fresh vaginal bleeding in labour

Group 2

- Prolonged rupture of membrane
- Non significant meconium
- Confirmed delay in first / second stage
- Moderate hypertension

Intermittent auscultation

- Every 15–30 minutes in active first stage of labour.
- Every 5 minutes in the second stage of labour.
- Each auscultation should last for at least 1 minute over three contractions if the FHR is not always in the normal range (i.e. 110–160 bpm).
- Auscultate during a uterine contraction and continue for at least 30 seconds after the contraction.

- Record the baseline FHR (as a single counted number in beats per minute) and the presence or absence of accelerations and decelerations.

Documentation basics

- Name / identity
- Date and time
- Ensure the wall clock and CTG clock are in sync
- Check paper speed

Why Document?
"Contemporaneous, complete, and objective documentation is the foundation for continuity of patient care and facilitates communication between all levels of healthcare clinicians."

Documentation

Contractions

- Frequency
- Duration
- Intensity
- Abnormality of resting tone

Fetal heart

- Note the individual features
- Then comment on the category
- Indicate reason for intervention

DR C BRAVDO

- DR Define risk
- C Contractions
- BR Baseline Rate
- A Accelerations
- V Variability
- D Decelerations
- O Overall impression

What to put down and how?

- Tachysystole vs so many contractions per min.
- Individual components to be described before interpretation.

FHR data :

- Numerical baseline rate (in bpm).
- Rhythm (regular or irregular).
- Nature of the changes (gradual or abrupt acceleration or deceleration).
- Interpretation of findings as normal or abnormal.
- Specific actions taken when changes in FHR occur.
- Other maternal observations and assessments.
- Maternal and fetal responses to interventions.

Twins

- If twin use twin monitor.
- If no twin facility
  - Locate the FH with scan and mark site.
  - Do both separately.
  - Make sure the trace is appropriately marked.

Remember

CTG traces fade with time. Hence scan or copy them and store. Newer machines allow storing of traces.

Maternal parameters

- Pulse rate / temp
- Any event : PV / ROM / epidural

Pre-requisites for EFM –Maternal

Patient should be in Propped-up / semi-recumbent position.

Preferable positions are Left lateral position or tilted to either side with a 45 deg wedge. Patient should never be left to lie on her back. In lateral position if the transducer is not making contact with the abdomen a person can hold it manually rather than placing the patient in supine position.

How often?

Routine 2 hourly note will not be sufficient. Notes should be documented

- Whenever the patient is examined.
- When significant interventions /a substantial change in progress or condition.

Do not forget

- Sign
- Print name
- Registration number
- Date and time

Interpretation of CTG

4 features

Baseline
Variability
Accelerations
Decelerations

Classification

Each Feature of CTG is classified as

- Reassuring
- Non reassuring
- Abnormal

CTG feature	Normal	Nonreassuring	Abnormal
Baseline	110-160	100-110,160-180	< 100, >180
Variability	5-25	<5 upto 50min, >25 upto 25 min	< 5 for> 50 min, >25 for > 25 min, sinusoidal
Deceleration	None/ uncompl VD < 90 min	Uncompl VD for > 90 min. Compl VD for <30 min, LD for < 30 min, no msl	Compl VD for > 30 min, LD for > 30 min/ <30min with msl, Prolonged decal > 3min, Acute bradycardia
Accelerations	Present	Absent	
Contractions	<4/10	>4/ 10	Hyperstimulation

CTG trace as a whole is classified as

- Normal
- Suspicious
- Pathological

2015 FIGO classification

	Normal	Suspicious	Pathological
Baseline	110-160 bpm	Lacking at least one characteristic of normality, but with no pathological features	< 100 bpm
Variability	5-25 bpm		Reduced variability for >50 min, increased variability for >30 min, or sinusoidal pattern for> 30min
Decelerations	No repetitive decelerations		Repetitive late or prolonged decelerations during >30 min or 20 min if reduced variability, or one prolonged deceleration with>5min
Interpretation	Fetus with no hypoxia/acidosis	Fetus with a low probability of having hypoxia/acidosis	Fetus with a high probability of having hypoxia/acidosis
Clinical Management	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation.	Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation or if this is not possible expedite delivery. In acute situations (cord prolapse, uterine rupture or placental abruption) immediate delivery should be accomplished.

Tachysystole

- Document tachysystole
- Stop Oxytocin
- Start Tocolysis
- Sub cut terbutaline 0.25 mg
- Consider nasal spray

Interpretation to action

- Never based on CTG alone.
- Depends on the clinical situation.
- The timing in labour.

Use of CTG in fetal hypoxia

Electronic FHR monitoring was introduced with the expectation that it would significantly reduce the incidence of neurologic injury (specifically cerebral palsy) caused by intrapartum interruption of fetal oxygenation. In recent years, it has become apparent that most cases of cerebral palsy are unrelated to intrapartum events and therefore cannot be prevented by intrapartum FHR monitoring. Nevertheless, a significant minority of such cases may be related to intrapartum events and might be preventable.

Criteria to define an acute intrapartum hypoxic event as sufficient to cause CP *(ACOG task force on neonatal encephalopathy and cerebral palsy 2003)*

- Evidence of *metabolic acidosis* in fetal umbilical cord arterial blood obtained at delivery - pH less than 7 and base deficit of 12 mmol/L or more. This criterion indicates that intrapartum interruption of fetal oxygenation does not result in neurologic injury unless it progresses at least to the stage of significant metabolic acidemia .It is important to note that fetal injury is uncommon even when metabolic acidemia is present.
- Early onset of *severe or moderate neonatal encephalopathy* in infants born at 34 or more weeks' gestation.
- Cerebral palsy of the spastic quadriplegic or dyskinetic type.
- Exclusion of other identifiable etiologies, such as trauma, coagulation disorders, infectious conditions, or genetic disorders.

Types of hypoxia

- Acute
- Chronic
- Sub acute
- Evolving

Acute Hypoxia

Causes

Abruption
Scar dehiscence
Cord prolapse

Findings

1. Single prolonged deceleration - the acute hypoxia lasts for less than 3 minutes and then recovers to normal baseline.
2. Prolonged decelerations - lasting for more than 3 minutes.
3. Prolonged baseline bradycardia - the FHR remains below 100 bp(80 bpm in severe cases of hypoxia) for over 10 minutes.

Course

- Fetal PH drop at 0.01/mt.
- 90% recovers by 6mts.
- 95% by 9mts.

Actions

0 – 3: If a deceleration is noted for more than 3 minutes with no signs of recovery the emergency alarm must be raised to summon the on-call team.

3 – 6: Attempt to diagnose the cause of the deceleration. If an accident is diagnosed the aim would be for immediate delivery as soon as safely possible in the fastest route possible (assisted vaginal delivery / caesarean section).

6 – 9: Signs of recovery should be noted (return of variability and improvement in heart rate).

If no signs of recovery are noted, preparation for immediate delivery MUST be started.

9 – 12: By this point in time the deceleration has either recovered, or preparation for an assisted vaginal delivery / caesarean section is in progress aiming for a delivery of the fetus by 12 – 15 minutes.

Sub acute hypoxia

Perfusion to the heart and brain is maintained optimally when the heart is pumping at a certain rate.

When the FHR decelerates, the number of circulations through the placenta is reduced proportionate to the extent of reduction of the FHR and the duration for which it is reduced. Usually, if the duration of the FHR deceleration is shorter than the time spent at the baseline rate, the fetus is able to replenish the oxygen supply and excrete the carbon dioxide. In this situation, the fetus spends more time decelerating and progressively less time at the normal baseline FHR. Typically, the fetus spends less than 30 seconds at the baseline to 'wash-off' carbon dioxide and acid, and spends over 90 seconds building up carbon dioxide and acid. The pH of the fetus has been shown to drop at the rate of 0.01 every 2–3 minutes.

Findings

- Duration of decelerations is two- to three-times greater than the time spent at the base line rate.
- Drop of the FHR < 80 bpm.

Gradually developing hypoxia

This is the most common type of hypoxia in labour . This tends to present with the following order:

1. Evidence of hypoxic stress (decelerations).
2. Loss of accelerations and lack of cycling.
3. Exaggerated response to hypoxic stress (decelerations become wider and deeper).
4. Attempted redistribution to perfuse vital organs facilitated by catecholamines (first sign noted is a rise in baseline).
5. Further redistribution with vasoconstriction affecting the brain (Reduced baseline variability) and bradycardia.

Chronic hypoxia

- This is an antenatal type of hypoxia with implications for intrapartum care. This represents a fetus with reduced reserve (eg; IUGR) and increased susceptibility to hypoxic injury during labour . If there is further hypoxia CTG shows reduced variability and shallow decelerations.
- Pre terminal CTG-mortality-39%

(*Continues in page No.17*)

Dr. C. Rajalakshmi
Born on 6.2.1943

Education

- Schooling at Pudukottai till 8th, then at Lady Sivasamylyergirl's high school, Chennai.
- Graduated from Stanley Medical College, Chennai in 1964.
- Postgraduate from Christian Medical College, Vellore in 1969.
- P.G certification course in Geriatric Medicine.
- Praveen in Hindi.
- Amarbarathi 4th grade in Sanskrit.

Experience

- Worked in CSI hospital, Erode from 1966 to 1973.
- Was the medical superintendant in Seethalaxmi hospital, Gobi 1973-1979.
- Private practice in Arcees Maternity, Erode since 1979.
- Professional bodies
- Founder President of Erode Obstetrics &Gynaecological society.
- Principal - I.M.A School of nursing aid from 1980 – 2000.
- Chairman Ethics committee for medical research - Erode cancer centre till date.
- Had been Joint secretary and vice president of Erode IMA.
- Honors
- First rate physician certificate in 1972 by Dr.Thomas Moulding in Denver, Colorado,for performing button hole sterilisations in Denver General hospital.
- P.C Roy memorial award for meritorious service in profession and society (1997).
- Dr.P.C.Roy Memorial doctor's day award for exemplary service to humanity in medical field(2012).
- Most outstanding woman citizen of erode by Erode Jaycees (1995).

Dr. C.Rajalakshmi being conferred Service Excellence award

Award handed over by our Guest speaker DR Uma Ram

Dr. Vijayarani honouring Dr. C. Rajalakshmi

Credits

- 1981 - The first Laparoscope of Erode District was started at Arcees Maternity.
- 1984 - The first Ultrasound Machine in Erode district was installed at Arcees Maternity.
- 1993 - Routine cancer cervix clinics was started in at Arcees Maternity

Mr. Chandran, Spouse of Dr. C. Rajalakshmi

Dr C. Rajalakshmi giving her acceptance speech

Area of interest:

- Reading books in languages Tamil, Hindi, Sanskrit and English.
- Plays veena and listen to Carnatic music.
- Poems, essays and script writing

SPEAKERS OF THE MEETING ON 10.02.2019

Dr Uma Ram,
Seethapathy Clinic, Chennai

Dr Sangeetha, Kumarasamy Hospital, Erode

Dr Amritha,
Seethapathy Clinic, Chennai

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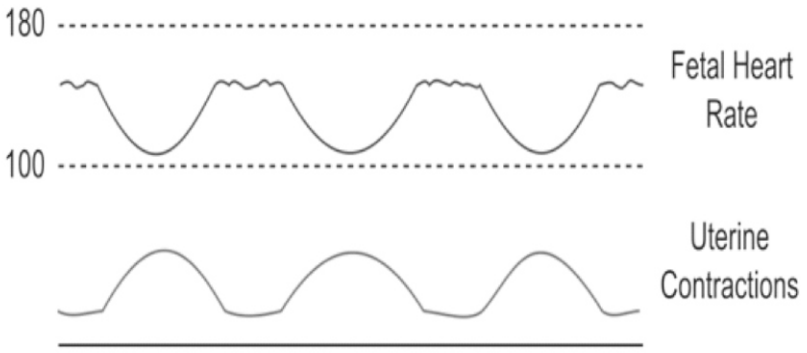
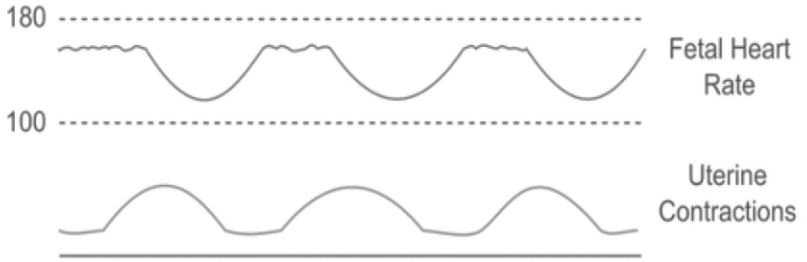
Types of decelerations

Early decelerations - onset with contraction, mirror image of contraction.



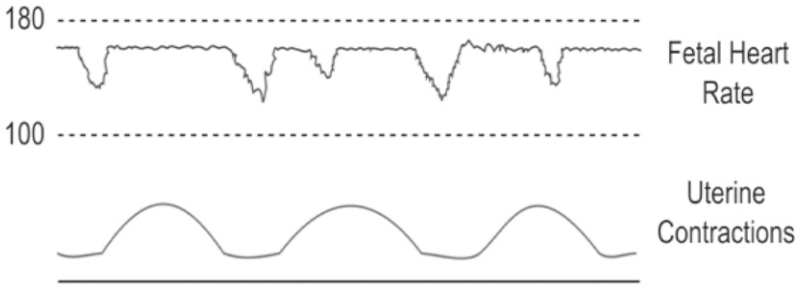
An early deceleration likely represents an autonomic response to changes in intracranial pressure and/or cerebral blood flow caused by intrapartum compression of the fetal head during a uterine contraction and maternal expulsive efforts, although the precise physiologic mechanism is not known. Early decelerations are clinically benign.

Late decelerations - onset 30-60 seconds after onset of contractions, nadir & recovery all out of phase.



In most cases, a late deceleration is a reflex fetal response to transient hypoxemia during a uterine contraction

Variable decelerations - V-shaped decelerations that exhibit a rapid drop (onset to nadir in <30s) followed by a rapid recovery to the baseline.



A variable deceleration reflects the fetal autonomic reflex response to transient mechanical compression of the umbilical cord.

Reliability of CTG

- False positive- 50-60%
- CTG with FBS—False positive-10%
  Negative predictive value is high

(Adapted from the talk given by DR Uma Ram and DR Amritha on 10.02.2019)

IMPORTANT DAYS IN MARCH 2019

Mar-08	International Women's Day
Mar-14	World Kidney Day
Mar-16	National Vaccination Day
Mar-24	World TB Day

PRE ECLAMPSIA HEADACHES

There is a headache for the obstetrician too....

Complications:

1. Eclampsia and other CNS problems.
2. Abruption.
3. HELLP syndrome.
4. Cardio vascular problems.
5. Renal problems.
6. Hepatic problems.
7. Haematological problems.
8. Other systems.

Eclampsia:

- Common in 3 rd trimester.
- Preceeded by headache with or without visual complaints.
- Starts with facial twitching → generalized or focal tonic clonic movement → post ictal confusion/ coma.
- Tachypnea and tachycardia are common after seizures.
- High fever may denote either intra cranial bleed or myolysis and raised metabolic rate due to fits.
- Labour may start spontaneously.
- SPO, fall ALARM
    1. Aspiration.
    2. Pulmonary edema.
    3. Poor respiratory drive due to intracranial events.
    4. Shallow breathing.
    5. Mg sulphate toxicity.
    6. Fluid overload.

Treatment principles

- Control of convulsions.
- Control of blood pressure.
- Delivery of the baby.
- Fluid management and supportive measures.

Control of convulsions:

FIRST AID → lateral positioning, IV line, oral suction, oxygen.

DRUGS: Mg sulphate - Pritchard regime.

Second line drug - Phenytoin loading dose 1000 mg in 100 ml NS infusion over one hour.

Control of hypertension:

- Labetalol -20 mg iv every 10 mins till diastolic BP comes between 90-100mmHg, Maximum dose 200mg.
- Hydralazine -5mg bolus repeated every 15-20 mins for a maximum of 20 mg in one hour. Remarkably effective in prevention of cerebral haemorrhage.
- Maintenance with oral Labetalol or Nifedepine through Ryles tube.

Delivery :

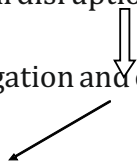
- Timing and mode of delivery should be individualized.
- If delivery is not imminent in reasonable time or in case of other obstetric indications cesarean can be done.
- Fall in BP after delivery (need not be hypotension may be normal range) most often means excessive blood loss.
- Due to poor intravascular volume, they are less tolerant of blood loss compared to normotensive women.

IV fluids:

- Though there is poor intravascular volume fluid infusion should be done with great caution.
- Rapid infusion may result in pulmonary edema.
- In case of C section adequate hydration before surgery may protect from spinal anaesthesia induced hypotension.
- At higher centers severe cases are managed with central venous pressure monitoring which helps in effective fluid management.

HELLP syndrome:

Hepatic endothelial disruption



Platelet activation, aggregation and consumption

Microvascular occlusion, ischemia and Hepatocyte death.

Microangiopathy causing intravascular hemolysis.

Diagnosis: (made by lab parameters alone)

1. Peripheral smear abnormality suggesting hemolysis.
2. Elevated liver enzymes, LDH > 600 U/L suggesting hepatic dysfunction.
3. Low platelet < 100,000- main and EARLIEST coagulation abnormality in HELLP.

Partial HELLP syndrome - one of the above three parameters may be absent. Carries same or higher morbidity and mortality due to delay in diagnosis.

Management :

1. Eclampsia prophylaxis.
2. Antihypertensives.
3. Evaluate fetal wellbeing.
4. Platelet transfusion if count < 20,000 or with significant bleeding tendency.
5. Delivery is the only definitive therapy. Mode of delivery decided according to gestational age and other obstetric parameters.
6. Platelet should be > 80,000 for spinal anaesthesia.
7. Fluid infusion according to urine output.
8. Packed cell transfusion if haematocrit < 30%.

Role of steroids :

It is controversial, still under trial. Dexamethasone 10mg two doses 6 hrs apart followed by 6 mg two doses 6 hrs apart.

Role of conservative management:

Prolonging the pregnancy beyond 24 hours after diagnosis of HELLP may cause higher maternal and perinatal mortality.

Post partum period:

Platelet count returns to normal by 3-5 days.
Careful monitoring for coagulopathy and renal failure.
Patient should be counselled about the recurrence (Pre eclampsia-20%, HELLP- 2-5 % .)

CNS involvement in pre eclampsia:

- Intra cranial haemorrhage-

  Severe occipital or temporal headache followed by convulsion and coma. Motor and sensory deficit will be there. Respiratory paralysis may occur. Volume and location of bleeding decides the prognosis.
- Hypertensive encephalopathy-

Due to severe acute rise in BP, diastolic BP usually > 120 mmHg. Diagnosed by CT or MRI imaging.

Posterior reversible encephalopathy syndrome:

- Diagnosed by clinico- radiological features.
- Headache, altered sensorium, visual disturbance with or without seizures.
- Non convulsive status epilepticus may occur which include stereotypic movements like staring, head turning or eye blinking along with altered sensorium. It is usually confused with post ictal confusion.
- Occurs secondary to inability of the posterior circulation to autoregulate in response of acute rise in BP. Hyper perfusion results in vasogenic edema at parieto occipital region.
- Diagnosed by CT or MRI brain showing edema.
- With effective BP control patient improve in a week.

Post partum circulatory collapse:

- Patient with severe pre eclampsia may develop profound shock after delivery. Common with LSCS than vaginal delivery.
- Usually occurs within first hour after delivery but may happen anytime within 24 hrs.
- If not rapidly corrected may lead to acute renal failure, pan hypo pituitorism, maternal death.
- Decreased plasma volume and misleading haematocrit and electrolyte imbalance are the predisposing factors.
- Patient may not tolerate even normal accepted blood loss.

- **Prevention:**
    1. Proper correction of anemia and electrolyte imbalance.
    2. Antenatal ECHO for anticipated cases.
    3. Proper volume correction.
    4. BE AWARE of the signs and symptoms of circulatory collapse which helps in early intervention.

Pulmonary edema:

Predisposing factors

1. Severe uncontrolled hypertension.
2. Pre existing heart disease.
3. Uncorrected anemia.
4. Rapid or excessive fluid infusion during surgery/ delivery.
5. Patients of chronic hypertension with superimposed pre eclampsia.
6. Compromised renal function.

Renal involvement in pre eclampsia:

- Common with Abruption and HELLP.
- In severe pre eclampsia renal blood flow is compromised due to reduced plasma volume and vasospasm.
- In abruption fibrinogen like material gets depositted in glomerular capillaries which disappear soon after delivery.
- In HELLP thrombotic micro angiopathy causes Acute tubular necrosis.

Severe pre eclampsia, abruption – pre renal ARF

HELLP – Renal ARF

INVESTIGATION	PRE RENAL	RENAL
Urine osmolality	>500	< 350
Urine Na	< 20 Meq/L	>40 Meq/L
Urine/serum creatinine	>40	<20
Serum calcium	↑	N/ ↓
Serum protein/albumin	↑	N/ ↓

Management:

- Volume replacement under CVP monitoring.
- Diuretics use is controversial but 40-60 mg frusedmide with IV fluid challenge may help to open the channels in pre renal failure.
- Pregnancy should be terminated.
- Early pathological changes are reversible. Hence early intervention restores renal function.
- In ATN re epithelialisation takes place within 6 weeks. In the mean time dialysis could be done if required.

Abruptio placenta:

- Anticipate in all cases of pre eclampsia before or during labour.
- 10% of patients develop renal failure.
- 20% of patients develop coagulopathy.
- Platelets, FFP and cryoprecipitate are given according to platelet count and serum fibrinogen level.
- Termination of pregnancy is the definitive therapy.
- If patient is in labour coagulopathy to be corrected with blood component therapy simultaneously.
- If planned for C section, coagulopathy to be corrected before surgery but waiting period should not go beyond 8-10 hrs.

BEING AWARE OF THE COMPLICATIONS HELPS IN EARLY DIAGNOSIS AND INTERVENTION

Dr. K. Vanithasri MD (OG).,

Practical Safety Tip
Perforation of the uterus

Do proper examination to identify the risk factors for uterine perforation before any uterine surgery like short cervix, tight cervix, hard cervix, abnormal mass at cervix, previous LSCS, scarred uterus, acutely anteverted or retroverted uterus, surgical MTP at > 12 weeks, congenital or acquired uterine abnormalities etc.

If possible, consider the other alternatives to the surgery like medical MTP in presence of risk factors.

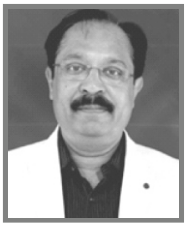
Ask for minor preoperative profile a day before the surgery, if not an emergency. Prepare the cervix with Tab. Misoprostol (200/400) or Prostaglandin gel 3 – 4 hours prior to surgery. Ask the patient to empty her urinary bladder before the surgery. Give proper anaesthesia. Avoid local anaesthesia whenever possible.

Avoid forceful dilatation of the cervix. In difficulty, perform dilatation of cervix & procedure under sonography or hysteroscopic guidance. If uterine perforation is suspected, abandon the Procedure. Most perforations are minor and do not harm much. Lateral wall perforations at cervix or lower uterus can cause haemorrhage from the uterine vessels and broad ligament hematoma. Anterior wall perforations can cause injury to urinary bladder. Use of thermal, grasping or suction instruments can cause injury to bowel, bladder & blood vessels. Immediately perform ultrasonography. Try to identify location of perforation, damage to any organs, if any and severity of the injury. Give intravenous broad spectrum antibiotics. Keep her nil by mouth till further orders.

Observe the vital data of the patient, and watch for any complaints like pain, vomiting, bleeding per vagina and distension of the abdomen etc at every 15 minutes for 2 hours and then at every 30 minutes for minimum 4 hours. If patient has any minor complaints, admit the patient for overnight, keep her NBM, start I.V. fluids and put the foley's catheter. If perforation is with thermal, avulsion or suction instruments or if torrential bleeding occurs, counsel patient & relatives; take written consent for need of laparoscopy, laparotomy or hysterectomy.

Make cross matched blood ready. If patient is hemodynamically unstable, resuscitate the patient. Per form laparoscopy or laparotomy to assess for injury to the blood vessels, omentum, bowel, urinary bladder, broad ligaments & ureters and take the necessary steps to correct the damage. Identify the perforation or rent on the uterus, suture it. Perform Hysterectomy if required.
Put the intra-abdominal drain. If managed conservatively, counsel with the patient about and watch for the signs and symptoms of sepsis, bowel and bladder injury till 2 weeks after the procedure. Do not repeat the procedure till 6 weeks, if possible.

Counsel the patient about possible complications in future like rupture of the uterus in a future pregnancy.



Dr. Alpesh Gandhi
President Elect, FOGSI-2020

THE MEETING ON 10.02.2019

EOGS oath

Attentive Audience

Dr. Sri Revathy participated as faculty in National ISAR 2019 Conference, Mumbai

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- HPV-HR
- INFLUENZA/AB
- S. PNEUMONIAE
- STAPH/PA
- STAPH/MRSA
- NEISSERIA MENINGITIDES
- PSEDOMONAS AERUGINOSA
- HAV
- HEV
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