First Edition

# ESSENTIALS OF OBSTETRICS

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# PREFACE

This hand book is written to enlighten and strengthen the core knowledge required for Obstetrics. I hope to contribute my part in teaching the eagerly keen students both at the pre and post graduate level. Reviewing the difficulties I faced in my studies and the challenges my students encounter this book is intended to help guide them and understand the basics of obstetrics.

During the course of writing this book I would like to thank some key persons whose input was of great importance. Those include Dr. Sabahat Habib, Dr. Rubina Noureen (Associate Professor), Dr. Nayyer Sultana (Associate Professor), Dr. Fiza Asif (Assistant Professor), Dr. Sobia Zafar (Senior Registrar), Dr. Sanya Muqaddas, Dr. Lareb Choudhary and in last but not the least we bear thanks to Mr. Aqeel for his I.T contribution.

# DEDICATION

I would like to dedicate this book to my parents whose unconditional love and encouragement has made me what I am today.

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# 1. LABOUR

## LABOUR

The process of regular painful progressive uterine contractions leading to cervical changes (dilatation, effacement, descent of presenting part) resulting in the delivery of fetus and placenta with membranes along with complete contraction & retraction of uterus.

### STAGES OF LABOUR

**First stage:** Describes the time from diagnosis of labour to full dilatation of cervix. 10cm divided in 2 phases

a) **Latent phase** is the time between the onset of labour and 3-4cm dilatation (3 to 8 hours) but shorter in multiparous.
b) **Active phase** is the time between the 3 to 4cm to 10cm (2 to 6 hours) but shorter in multiparous women.

**Second stage:** Describes the time between full dilatation of cervix to the delivery of fetus or fetuses. Subdivided into two phases.

a) Passive phase: is where there is no maternal urge to push. Presenting part of the fetus is relatively high in the pelvis.
b) Active phase: in which presenting part is low and maternal urge to push is present.

**Third stage:** From delivery of fetus or fetuses until complete delivery of placenta and its membranes with complete retraction and contraction of uterus (few minutes). Lasting more than 30 minutes should be considered abnormal.

### MECHANISM OF NORMAL LABOUR

1. **Engagement**: When widest part of presenting part passes through pelvic inlet, it is known as engagement.
2. **Descent:** Descent of head occurs due to uterine contractions in 1$^{st}$ and passive phase of 2$^{nd}$ stage but voluntary by maternal push in active phase of 2$^{nd}$ stage.
3. **Flexion:** In midcavity flexion of fetal head occurs.
4. **Internal rotation**: Upon reaching the sloping gutter of levator ani muscles head will be encouraged to rotate. If it is well flexed, occiput will rotate till sagittal suture comes in AP diameter of pelvic outlet
5. **Extension:** After completion of internal rotation, occiput is underneath the symphysis pubis and bregma is at lower border of sacrum. The head now extends and occiput escapes from underneath the symphysis pubis and distends the vulva this is known as crowning.
6. **Restitution:** Slight jerky rotation of occiput through one eighth of the circle is called restitution.

7. **External rotation:** Occiput further rotates through one eighth of circle to the transverse diameter to deliver the shoulders by aligning them in AP diameter.
8. **Delivery of shoulder and body of the fetus**: Then rest of the body is delivered by lateral flexion.

# Management of Labour

**Definition:** Regular painful uterine contractions with dilatation and effacement of cervix resulting in delivery of baby and placenta.

- Evaluate the patient & review all previous record & establish the diagnosis
- Clinical assessment of patient & label her as low risk or high risk.
- Do CTG & send required investigations

## 1st Stage

**Latent phase:**

- After complete assessment, if patient is in latent phase and there are no maternal and fetal complications, the best place of care is at home**. If high risk, admit the patient.**
- Maintain normal level of activity.
- Patient should take light diet.
- Ensure good hydration
- Simple analgesia is effective (Paracetamol).
- Regular assessment of maternal & fetal well being(4 hourly)
- Vaginal examination (4 hourly).
  Further induction/ augmentation is consultant decision to reduce the risk of poor prognosis and C-section

**Active phase**

- Admit the patient in active phase
- Active phase of labour starts with 4cm dilatation of cervix.
- Maintain Partogram/labour care guide
- Augmentation if indicated.
- Repeat pelvic examination 2 hourly
- Maternal monitoring B.P and temperature 4 hourly and pulse 1 hourly in low risk. In PROM temperature record 1 hourly
- Ensure bladder emptying
- Analgesia (Paracetamol, Tramadol, Entonox, Epidural)
- Keep patient in left lateral position.
- Continuous fetal heart rate monitoring in high risk patients
- Give psychological support

**FHR monitoring**:
Latent phase: low risk: every 30 min, high risk: every 15 min

Active phase: low risk: every 15 min, high risk: every 5 min after every contraction (continuous monitoring if available)

## 2nd Stage

Time: Full dilatation of Cervix to delivery of baby
Duration: Primigravida 2hrs, multigravida 1hour (with epidural Primigravida 3hrs, multigravida 2hrs)

- Shift primigravida to delivery room on crowning and multigravida at full dilatation of cervix.
- Make lithotomy position
- Do aseptic measures
- FHR monitoring after every contraction
- Monitor frequency, intensity, and duration of uterine contractions.
- Give Episiotomy after infiltrating the perineum with 1% of Lignocaine, at crowning & deliver head of baby.
- Avoid fundal pressure

## 3rd Stage

Time: Delivery of baby followed by complete delivery of placenta Duration: 15-30 mints

- 10 units synto × IV given on delivery of anterior shoulder of baby
- Place baby on mother's abdomen. Dry and assess breathing
- If baby delivered with good APGAR, delay cord clamping for 1 min
- Clamp & cut the cord, deliver the placenta by controlled cord traction
- Look for signs of PPH & any genital tract injury
- Stitch episiotomy in 3 layers
- Do PR after stitching episiotomy
- Do bimanual examination
- Place 800ug Misoprostol per rectal × stat prophylactically in high risk patients for PPH
- Per rectal examination

## Post-Natal Care

- Shift the patient to labour ward after cleaning her
- Complete your notes of SVD & mention any specific order
- Check B.P, Pulse, Temperature ½ hourly for 2 hours.
- Anticipate and treat PPH promptly in high risk patient
- Establish breast feeding as soon as possible & explain care for episiotomy
- Mobilize the patient
- Bladder emptying within 4 hours
- Counsel the patient regarding methods of contraception.

# ABNORMAL LABOUR

Labour becomes abnormal when there is poor progress and the fetus shows signs of compromise.

If there is fetal malpresentation, multiple pregnancy, uterine scar or if the labour has been induced, then the labour cannot be considered normal.

**Poor Progress in Labour**:
Defined as cervical dilatation <2cm in 4 hours usually associated with failure of decend & rotation of fetal head.
It depend upon 3 variables:

- POWER
- PASSENGER
- PASSAGE

Abnormalities of any of these can slow the normal progress of labour.

**Dysfunctional Uterine Activity**

- Most common cause of poor progress
- Common in primigravida
- If there is a delay women should be offered artificial rupture of membranes. after 2 hours still poor progress then augment with oxytocin infusion.
- 4 to 5 contractions in 10 minutes considered to be ideal.

**Cephalopelvic Disproportion:**
Anatomical disproportion between fetal head & maternal pelvis. It can be due to large head and small pelvis or combination of both.

**Absolute Cephalopelvic Disproportion:**

- CPD can be absolute if the head is too large to pass through a normal size pelvis as in hydrocephalus.
- It can also be absolute if the head size is normal and pelvis is contracted, metabolic bone disease.

**Relative Cephalopelvic Disproportion:**

- Head size and pelvis is normal but there is abnormal position of head e.g occipitoposterior position and brow presentation

## DIAGNOSIS OF RELATIVE CEPHALOPELVIC DISPROPORTION

Diagnosis of relative CPD can only be done when patient goes into labour.

- Progress is slow despite efficient uterine contractions
- Fetal head is not engaged.
- Vaginal examination shows severe moulding and caput formation
- Head is poorly applied to cervix.
- Relative disproportion can be overcome if malposition is corrected.

**Management Of Cephalopelvic Disproportion**

- In case of hydrocephalus encephalocentesis is an option depending upon the patient decision after she has been properly counseled. This can be followed by normal delivery.
- In all other causes a diagnosis of CPD necessitates delivery by cesarean section.

**MALPRESENTATION:**

It includes face presentation, brow presentation, shoulder presentation.

**FACE PRESENTATION:**

- Incidence: 1 :500
- Complete extension of fetal head
- Presenting diameter is Submentobregmatic which measures about 9.5 cm
- Engagement of head is late & progress is slow because the facial bones do not mould.
- Diagnosed in labour through vaginal examination.
- If chin is mentoanterior position vaginal delivery is possible

**BROW PRESENTATION:**

- Incidence: 1:2000
- Presenting diameter is mentovertical measures 13.5 cm
- Diagnosed during the labour by palpating supra-orbital ridges, anterior fontanelle and nose on vaginal examination
- In persistent brow presentation delivery by c-section.

**SHOULDER PRESENTATION:**

- Incidence: 1: 300
- Results from transverse and oblique lie of fetus.
- Increase risk of cord prolapse and uterine rupture.
- Delivery by c –section.

**ABNORMALITIES OF BIRTH CANAL:**

- Abnormalities of uterus ,and cervix can also delay labour.
- Fibroids in lower uterine segment can prevent the descent of fetal head
- Cervical dystocia (non-compliant cervix which effaces but fails to dilate because of scarring.
- In these conditions c –section is indicated.

**POOR PROGRESS IN 2ND STAGE OF LABOUR:**

Birth of the baby is expected within 3 hours of start of active 2nd stage in nulliparous and 2 hours in multiparous women.

A. SECONDARY UTERINE INERTIA:
   - Most common cause of second stage delay
   - Cervix achieved full dilatation , the uterine contractions become week & inefficient
   - It is associated with maternal dehydration and ketosis.
   - If no mechanical problem then treat with rehydration and I/V oxytocin.
   - Delay can be due to OP position. In this condition head either goes into long rotation to become OA position or to be delivered as face to pubes.

B. Delay may be due to narrow mid cavity (android pelvis) which prevents internal rotation of fetal head resulting in arrest of fetal head at the level of ischial spines in transverse position called deep transverse arrest.

   Instrumental birth should be considered for prolonged second stage.

# INDUCTION OF LABOUR

- 10-20% of all pregnancies are induced
- Overall success rate is about 80-85% at term

## Obstetric Indications:

- Uteroplacental insufficiency (most common indication)
- Severe pre-eclampsia or eclampsia (after maternal stabilization)
- Pre-labour rupture of membrane
- Prolonged pregnancy (41-42 weeks)
- Chorioamnionitis
- Intrauterine growth restriction
- Fetal demise (IUD)
- Oligohydramnios/anhydramnios
- Congenital anomalies

## Maternal indications:

- Severe hypertention
- Uncontrolled Diabetes Mellitus
- Renal disease with deteriorating renal function
- Malignancies (to facilitate treatment of malignancy)

## Contraindications:

- Placenta or vasa previa
- Transverse fetal lie
- Prolapsed umbilical cord
- Prior classic uterine incision
- Active genital herpes infection
- Pelvic structural deformities
- Grand multiparity
- Overdistended uterus
- Non-vertex presentation

## Cervical ripening methods:

**Mechanical methods**

- **Membrane stripping** (stretch & sweep)
  Should be offered to patients prior to formal IOL and at 40 plus week's antenatal visit.
  It involves digitally separating membranes when cervical os admits a finger. Separation of membranes from cervix leads to local release of Prostaglandins. Patient will go in spontaneous labour in <7 days

**Pharmacological methods**

- **Dinoprostone (PGE2)** (Dose: 1 tablet (3mg) or gel (2mg) followed after 6 hours by 2nd dose if labour is not established) ideally. But third dose can be repeated after 6 hours provided feto-maternal condition is satisfactory
- **Misoprostol (PGE1)** according to FIGO: at term doses 25-50 micro gram/vaginal or oral (Only for IUDs)

**Surgical methods**

- **Amniotomy**, alone or with oxytocin, should not be used as a primary method of IOL except there is contraindication for vaginal PGE2 (uterine hyper stimulation)
- Iv oxytocin after amniotomy

## Requirements for induction of labour:

1. Calculate the exact gestation LMP dating scan to avoid iatrogenic prematurity
2. Women with uncomplicated pregnancies should be offered induction of labour beyond 41 weeks.
3. Careful review of the indication,
4. Informed consent.
5. Bishop score should be assessed and recorded. Favorable bishop score is associated with increased chance of successful induction
6. Maternal and fetal wellbeing established with a fetal surveillance test and routine maternal observations/monitoring

**Cervical scoring using modified Bishop's score**

| Score | 0 | 1 | 2 | 3 |
|---|---|---|---|---|
| Dilatation (cm) | <1 | 1-2 | 2-4 | >4 |
| Length (cm) | >4 | 2-4 | 1-2 | <1 |
| Consistency | Firm | Medium | Soft | |
| Position | Posterior | Mid/Anterior | | |

## Monitoring:

1. Facilities should be available for continuous electronic fetal monitoring (EFM) and uterine contractions monitoring.
2. Reassess Bishop score every 6 hours after vaginal PGE2 tablet or gel

## Pain relief:

Induced labour is likely to be more painful than spontaneous labour. Epidural analgesia should be offered to the patient ideally. Infusion of Tramadol and Provasc can be offered in other setups

## Risks of induction of labour:

- Increased hospital stay
- Increase need of analgesia during labour.
- Uterine hyper-stimulation 1%
- Cord prolapse
- Greater risk of uterine rupture during VBAC
- Failure of induction
- Increase need for c-section or instrumental delivery.
- Fetal compromise

- Low Apgar at birth
- Increase risk of instrumental delivery

## Prevention & Management of complications:

1) **Failed induction:** Occurs in 15 percent of cases
   Defined as failure to establish labour after one cycle (2 doses) of treatment
   Reassess maternal & fetal condition for further attempt to IOL/ C.S

2) **Uterine hyper-stimulation:** Occurs in 1-5 percent of cases
   **Def.** >5 contractions /10mins
   **Treatment:** Inj. Terbutaline (bricanyl) 0.25 mg* S/C * stat
   Hydration, Oxygen, CTG
   Re-evaluate the patient
   Emergency CS if FHR changes do not reverse

3) **Cord prolapse:**
   Avoid amniotomy if the fetal head is high

4) **Uterine rupture:**
   If suspected during IOL, do emergency CS

**Note :**
PGE2 appears safer method of induction than misoprostol and amniotomy
There is increased risk of PPH , rupture of unscared uterus , meconium stained liquor , precipitate delivery and bad perinatal outcome with misoprostol.
There is increase risk of cord prolapse and introduction of infection with amniotomy.

# MALPOSITION OF FETAL HEAD

## Incidence 25% early labour 10-15% in active labour

### Occipito-posterior position of the fetal head:

- Means the head enters the pelvis in one of the oblique diameters and occiput is directed posteriorly
- There are two positions
- Right occipito-posterior position ROP (the occiput directed to the right sacro-iliac joint)
- Left occipito-posterior position LOP (the occiput directed to the left sacro-iliac joint)
- The ROP is more common and will be described here.

**Causes:**

1. Android pelvis
2. Anterior placenta
3. Multiparity
4. Idiopathic.

**Fates of O.P position:**

1. Long anterior rotation ($3/8^{th}$ of circle)
2. Short posterior rotation(face to pubes) $1/8^{th}$ of circle.
3. In complete forward rotation(Deep transverse arrest)
4. Persistent O.P position

**Mechanism of labour:**

depends on whether the head is well flexed or incompletely flexed.

**1.The well flexed head/ Long anterior rotation:**

If the head is well flexed the occiput will be at lower level than the sinciput. It will hit the pelvic floor first. Undergoing long anterior rotation through $3/8^{th}$ of a circle to lie behind the symphysis pubis. The rest of the mechanism is same as the right occipito –anterior position

**2.When the head is incompletely flexed / Short posterior rotation:**

- In this occipito-frontal diameter (11.5cm) has to pass through the pelvis, so it leads to difficult and prolonged labour.
- The sinciput will meet the pelvic floor first and rotate anteriorly to lie behind the symphysis and occiput rotates backward by $1/8^{th}$ of a circle to lie in the hollow of sacrum.
- The head will deliver by face to pubes. Root of nose is pressed against the bone
- The vertex is born by flexion and followed by the occiput.
- Then the head extends so the face and chin emerge from the pubic arch.
- The vulval orifice is stretched by the occipito frontal diameter with a difference in size of 1.5cm and a severe perineal tear may result.

**Deep Transverse Arrest / Incomplete Forward Rotation:**

It is defined as:

**"** ***despite of having good uterine contractions and full dilatation of cervix, the head is not descending and is stuck at the level of ischial spines ,with one fontanelle facing to one ischial spine and other fontanelle facing to other ischial spine".***

- In some cases the head becomes arrested with its long axis in the transverse diameter of the pelvis.
- The degree of extension being such, that neither the occiput nor the forehead is sufficiently in advance to influence rotation.
- It results from either incomplete forward rotation of occipito-posterior position or result of failure of the head which enter the pelvis in occipito –transverse position to rotate anteriorly.

## Diagnosis:

**During pregnancy:**

non –engagement of the fetal head before the onset of labour in primigravidae

**During labour:**

Abdominal examination

1. There is flattening of the lower abdomen
2. The limbs are easily felt anteriorly
3. Difficult in defining the back which is felt far in the flank.
4. Difficult to hear the fetal heart sound which is heard in one of the flanks.

## Vaginal Examination;

**Early in labour:**

- Early rupture of membranes
- High presenting part.

**Established labour**

- The position can be determined from the direction of the anterior fontanelle , which can be easily felt behind the pubis
- The degree of flexion of the head can be determined from the fontanelle also
  1. If only anterior fontanelle can be felt the head is poorly flexed.
  2. If both the anterior and posterior fontanelle can be felt the head is less flexed.
  3. If only the posterior fontanelle felt the head is well flexed. Well flexed head is more likely to rotate anteriorly.

**In the second stage of labour:**

Sometimes the position is not recognized until there is delay in the second stage of labour. The diagnosis by vaginal examination may be difficult due to the **formation of caput succedaneum** over the presenting part. In this case the fingers may be passed higher to feel the free margin of the ear which will point to the occiput.

**The course of labour in occipito-posterior position:**

- Prolongation of the 1st and 2nd stages of labour is common
- Ineffective uterine contraction is common because the poorly flexed head fails to press down upon the cervix.
- In the 70% of the cases there will be spontaneous rotation of the occiput to the anterior position
- In about 10% cases the occiput undergoes short backward rotation and delivered in the direct occipito-posterior position(face to pubes).
- In the remining assisted rotation will be required.

**Management of first stage of labor:**

- The 1st stage is managed as in a normal case.
- Nothing can be done to correct the malposition or in influence of head at this stage.
- A partogram is done to monitor the labour.
  Uterine contraction(frequency, duration, strength), fetal heart monitoring, dilatation of the cervix.
- If progressive cervical dilation does not occur augmentation with an oxytocin drip may be tried
- If still no progress in few hours cesarean section is performed.

**Management of 2nd stage of labour:**

- In most cases (70%) provided that the uterine contractions are strong and the woman is able to make good expulsive efforts, the occiput rotates forward and normal delivery takes place.
- In other cases (10%) the baby may be delivered face -to-face pubes without difficult y but there is a great risk of perineal tear.
- **In about 20 % cases** there is failure of the presenting part to rotate and descend in such cases delivered by c/s or rotation can be enhanced by **assistance.**

## The First Step In Assisting Delivery is Rotation of The Fetal Head:

This can be performed by:

1. Manual rotation and forceps delivery.
2. Keilland forceps.
3. Vacuum extractor

**Manual rotation and forceps delivery:**

- Should be done under pudendal block or General anesthesia.
- The head is rotated with the fingers to direct anterior position.
- The shoulder girdle of the fetus should be rotated at the same time as the head by pressure through the abdominal wall by external hand.
- After rotation is completed the obstetric forceps are applied to complete the delivery.

## Deep Transverse Arrest:

- The occiput lies on one side of the pelvis and the sinciput on the other side and the head is badly flexed.
- It is only diagnosed during the 2nd stage after arrest of labour.
- If the head is firmly fixed in the transverse position obstructed labor will occur.

**It is commonly causes by an android pelvis which has the following feature:**

1. Anterior surface of sacrum is straight
2. The ischial spines are prominent.
3. The side walls are convergent.

**Management:**

Deep transverse arrest can be managed in one of the following ways:

- Manual rotation of fetal head to OA position
- Application of mid-cavity rotational forceps
- Emergency caesarean section

When the head is arrested in the transverse position the safest way to deliver the fetus is by cesarean section.

# PRE-TERM DELIVERY

Delivery between 24-36+6 weeks is defined as preterm delivery with weight of the baby above 500grm.
Moderate to late preterm = 32-36+6 weeks
Very preterm birth = 27-31+6 weeks
Extreme preterm birth = 24-26+6 weeks

## Incidence:

### Etiology

- Cervical incompetency
- Infections
- Preterm pre-labor rupture of membranes
- Multiple prégnancies d/t Uterine over distension
- Poly hydramnios
- Pyelonephritis, appendicitis, pneumonia, any surgical procedure.
- Uterine or cervical anomalies
    - Uterine ⟶ Unicornuate uterus, fibroid
    - Cervical ⟶ Cone biopsy, trachelectomy

### Risk Factors

- Low BMI
- Anemia
- Short inter-pregnancy interval (<1y)
- Maternal age (Extremes of age, Teenage and > 35(high risk))
- Nulliparous or grand multiparous
- Previous hx preterm delivery( if one previous preterm risk is 20%, if previous 2 preterm then risk is 35-40%)

### Management

- History
- Examination
- Investigation
- Treatment
    - Steroid cover
    - $MgSo_4$ for fetal neuroprotection
    - Tocolysis e.g. β-agonist like ritodrine, oxytocin antagonist i.e. Atosiban. $Ca^{+}$-channel blocker like nefidil (drug of choice)
    - Progesterone Supplements (for uterine quiescence)
    - Lifestyle modification

**Prevention of Preterm Delivery**

- Progesterone supplement
- Cervical cerclage
- Cervical length surveillance with serial measurement of cervical length throughout the second and early third trimester is now used in women at high risk of preterm delivery.
- Treatment of infection (Bacterial Vaginosis, Candidiasis, Chlamydia/Gonorrhea, Asymptomatic bacteriuria)

# VAGINAL BIRTH AFTER CESAREAN SECTION

- Woman with uncomplicated previous 1LSCS at term with no contraindication for vaginal delivery is a candidate for VBAC (76% success rate)
- Counselling for mode of delivery should be conducted after anomaly scan and the decision should be finalized by 36+0 weeks of gestation.
- ERCS should be conducted after 39+0 weeks of gestation
- Risk of uterine rupture 0.5% (01 in 200)

**Antenatal evaluation for VBAC:**

- Early booking
- Rule out contraindication to VBAC
- Details of previous C.S must be explored.
- Inter-pregnancy interval(risk increased if interval <12 months)
- Gestational age at which previous surgery was done
- Indication for previous C. Section
- Previous H/O any complication in antenatal & post-operative period (wound infection and fever).

**Previous vaginal delivery, particularly VBAC is the single best predictor for successful VBAC (87-90% Success rate)**

| **SELECTION CRITERIA** | **Contraindication to VBAC** | **Factors for unsuccessful VBAC** |
|---|---|---|
| Singleton pregnancy with cephalic presentation<br>No Contraindication to VBAC | Previous uterine rupture<br>Previous classical C-Section<br>Previous uterine surgery, myomectomy , where uterine cavity has been breeched<br>Récurrent cause e-g; absolute CPD<br>Non-recurrent cause e-g; major degree placenta previa,<br>Malpresentation in this pregnancy | Advance gestation > 40 weeks<br>Advance maternal age >40years<br>High maternal BMI> 30kg/m2<br>No previous vaginal births.<br>Fetal macrosomia<br>Inter pregnancy interval < 2years<br>Cervical dilation <4cm at time of admission |

**Antenatal counseling for VBAC:**

- Explain potential risks and benefits
- Discussion to be tailored according to patients, individual circumstances (personal motivation, preference to achieve VBAC, attitude towards the risk of uterine rupture, chances of a successful VBAC e.g. H/O previous vaginal birth.
- Elicit maternal choice for mode of delivery
- If planned for VBAC and spontaneous labour does not start till 40 weeks-take advice from consultant as Induction of labour carries risk of rupture.
- Patient should be informed about 2-3 fold increased risk of uterine rupture and around 1.5 fold increase risk of cesarean delivery in induced/augmented labour ,If planned for Elective C. Section and labour starts before due date, evaluate the patient and take advice from consultant

## Labour and Delivery with Previous 1 LSCS:

- Review detailed history & previous record
- Explain to the patient risks vs benefits of VBAC once again & take written informed consent
- Maintain I/V line & send blood for investigation, grouping and cross matching.
- One to one monitoring
- Continuous FHR monitoring.
- Regular monitoring of maternal sign and symptoms
- Regular (No less than 4 hourly ) assessment of dilation and progress of labour
- Keep her prepared for Emergency LSCS and arrange blood

## Monitoring & Care:

| **First stage** | **2nd & 3rd stage** |
|---|---|
| Pulse half hourly, B.P 1 hourly, input/output record<br>Frequency, intensity & duration of uterine contractions, scar tenderness in 15 minutes.<br>Maintain partogram<br>Adequate analgesia(epidural has relative contraindication)<br>**If required**, augment inadequate contraction with low dose syntocinon infusion after consultation.<br>If no progress for 2 hours do LSCS | Inform senior if 2nd stage >30 minutes (may be shortened by instruments)<br>Do not check previous scar digitally after delivery<br>Inform senior if excessive bleeding or retained placenta (possibility of adherent placenta) |

## The clinical features associated with uterine scar rupture include:

- Abnormal CTG
- Severe abdominal pain, especially if persisting between contractions
- Acute onset scar tenderness
- Abnormal vaginal bleeding
- Hematuria
- Cessation of previously efficient uterine activity
- Maternal tachycardia, hypotension, fainting or shock
- Loss of station of the presenting part
- Change in abdominal contour and inability to pick up fetal heart rate at the old transducer site

# 2. OBSTETRICAL EMERGENCIES

## ANTEPARTUM HAEMORRHAGE

**Definition**: APH is defined as bleeding from/into the genital tract occurring from 24 weeks of pregnancy and prior to the birth of baby.

**Incidence**: 2 to 5%

### Causes:

1. **Placental**
   - Placenta previa (0.4% to 0.8%)
   - Placental abruption (5%)
   - Marginal placental bleeding

2. **Fetal**
   - Vasa previa (0.015%-0.04%)

3. **Maternal**
   - Vulval/ Vaginal Trauma
   - Cervical ectropion
   - Local infection of cervix
   - CA cervix
   - Varicosities
   - Uterine rupture

4. **Unknown**

| **Placenta Previa**<br>**Incidence:** 0.4 to 0.8% | **Placental abruption**<br>**Incidence:** 5% |
|---|---|
| **Risk factors:**<br>Previous uterine surgery, D&C, Cesarean section, myomectomy(cavity breeched)<br>Maternal age > 40years<br>Multiparity<br>Smoking<br>Assisted conception | Family history of abruption<br>Trauma<br>Chorioamnionitis<br>Smoking<br>Pre eclampsia<br>Rapid uterine decompression |
| **Symptoms:**<br>Painless PV bleeding<br>Un provoked sudden onset<br>Bleeding is always revealed | Abdominal pain (continuous)<br>Painful vaginal bleeding<br>Sudden or may follow trauma<br>Bleeding may be concealed or revealed |
| **Signs:**<br>Pallor ++<br>Abdomen soft SFH corresponds to gestational Age<br>Fetal parts palpable easily<br>FHS is present in most of the cases<br>Lie is mostly oblique<br>Presenting part is high | Pallor ++<br>Abdomen hard with rigidity, SFH usually larger than dates<br>Fetal parts difficult to palpate<br>FHS is absent in most of the cases<br>Lie have no association |

# Management:

**Initial presentation**

- Quick Clinical history to rule out the cause
- Rapid clinical assessment
- Vaginal examination must not be performed until placenta previa has been ruled out.

| Maternal | Fetal |
|---|---|
| Pulse, BP, Uterine palpation for size<br>Tenderness and presenting part<br>No vaginal examination until placenta<br>Previa is ruled out | Fetal heart rate<br>CTG<br>Ultrasound |

**Initial resuscitation:**

- Call for help
- Left lateral tilt
- Check and maintain airway and give 100% oxygen by mask (10-15 L/min)
- Pass 2 intravenous lines 14G and take blood for full blood count, clotting profile, cross match six units of blood.
- Keep the women warm
- Until blood is available, Give warmed crystalloids (2 liters) and/or Give colloids as rapidly as needed (1-2 liters) while waiting for blood
- Once available give warmed blood as much as required
- If cross-matched blood is unavailable groups specific blood ( uncrossed matched or O –ve blood )
- Fresh frozen plasma
    - 4 units ( 12-15 ml/kg or total 1 liter for every 6 units of of red cells )
    - OR if PT and APTT > 1.5Xmean control
- Consider platelets concentrate If platelet count is < 50 x $10^9$/liter
- Cryoprecipitate if fibrinogen <1 g/liter

| Cause | Immediate delivery | Conservative management |
|---|---|---|
| Placental abruption | When there is fetal or maternal Compromise irrespective of the gestational age<br>**Mode of delivery:**<br>**Vaginal birth**<br>**1.**Fetus is dead irrespective of gestation<br>**2.**Alive about extremely premature with poor chances of survival<br><br>**Cesarean section**<br>**1.**Alive fetus with massive or mild to moderate abruption at term<br>**2.**Preterm baby with good chances of survival | Little place for expectant management<br>If abruption is mild with normal fetal heart rate then consider prolonging pregnancy for corticosteroid and correction of anemia |

| | | |
|---|---|---|
| | **3.**Uncontrolled hemorrhage, failure of induction, fetal malpresentation, and multiple cesarean section scars | |
| **Placenta Previa** | **1.**Pregnancy has reached term<br>**2.**Hemorrhage profuse and life threatening irrespective of gestational age<br>**3.**Fetus is dead or malformed | **1.**Admit in hospital<br>**2.**Correction of anemia<br>**3.**Steroid cover between 24-35+6 weeks<br>**4.**Plan elective C-section for patient with major degree placenta Previa between 36-37 weeks |

- Consultant should be present at the time of delivery and remain vigilant for PPH.
- Special protocol should be followed for C-section in major degree placenta Previa.
- Presence of consultant gynaecologist, anesthetist and pediatrician.
- At least 6 units of bloods to be ready.
- High risk written consent for hysterectomy should be taken.
- All the uterotonic drugs and surgical material should be there.

**Definitive treatment:**

Definitive treatment is always according to cause, severity of APH, duration of pregnancy, maternal compromise and condition of the fetus.

**Confirm fetal heart rate:**

- If alive consider immediate delivery by Em-LSCS under general anesthesia.
- If no fetal heart sound, confirm fetal death, exclude placenta Previa and induce labour.
- Major abruption at any gestation should be delivered and mode should be according to the fetal heart and previous obstetrical history.
- Minor abruption between 34 to 38wks, consider for admission and vigilant fetomaternal monitoring.
- Do laparotomy in case of uterine rupture.

## Complication of APH

**Maternal:**

- Anemia
- Shock
- Acute tubular necrosis (ATN)
- DIC
- PPH
- Complications of blood transfusion

**Fetal:**

- Hypoxia
- Feral death
- SGA/IUCTR
- Pre maturity

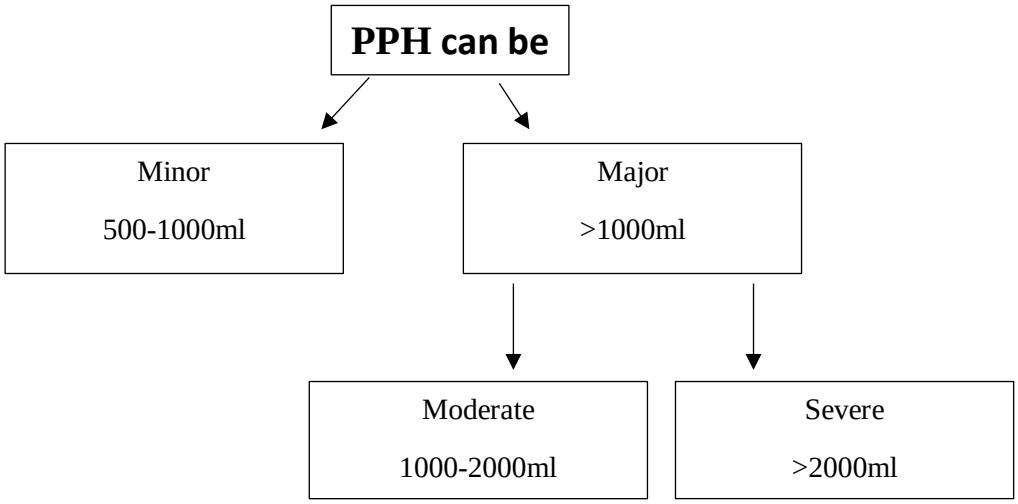
# POST-PARTUM HAEMORRHAGE/SHOCK

## Definition:

**Primary PPH** Loss of 500ml or more blood from genital tract within 24hrs after birth of baby.

**Secondary PPH** Abnormal or excessive bleeding from birth canal between 24hrs and 12 weeks postnatally

Patients presents in state of shock i-e tachycardia or bradycardia, hypotension, pallor, altered state of sensorium.




## Risk Factor.

The causes of PPH are related to abnormalities of one or more of four basic processes, The **FOUR T's,** Tone, Tissue, Trauma and Thrombin.

| **Risk factor** | **The four T's** |
|---|---|
| Multiple pregnancy | Tone |
| Previous PPH | Tone |
| Pre-eclampsia | Thrombin |
| Fetal macrosomia | Tone |
| Failure to progress in second stage | Tone |
| Prolonged third stage of labour | Tone |
| Retained placenta | Tissue |
| Placenta accreta | Tissue |
| Episiotomy | Trauma |
| Perineal laceration | Trauma |
| General anaesthesia | Tone |

## Management

**Pathway of care**

Call for help+ initiate resuscitation
-Check airway and give 100% O2 by mask
-Save 2IV lines of 14G+ send blood for CBC, clotting, c/m 6 units of blood
-Give warm crystalloids(0.9% saline) and colloid rapidly while awaiting blood
-Give warm blood as rapidly as needed
-Pass Foley's catheter
-Check pulse, B.P, Temp and O2 saturation.
-Consider CVP line if there is no contraindication

**Check is the uterus contracted**

NO

Aim is to keep uterus contracted

↓

Bimanual compression

↓

Syntometrine(5IU oxytocin+ ergometrine 0.5mg)

Carboprost 250µg I/M repeated every 15 mins

Then if needed, IV infusion of syntocinon

Misoprostol 80mg (4tabs) into rectum

↓

if still bleeding

↓

Hydrostatic balloon tamponade

↓

Take to theatre for laparotomy
Uterine artery ligation
β Lynch brace suture
Hysterectomy
Embolization

YES

- **If RPOC's** Remove + antibiotics
- **Genital tract** trauma repair ± vaginal packing

**-Uterine inversion** Reduce

**-In case of clotting disorders, give:**

Warm fresh blood
Fresh frozen plasma
cryoprecipitate

# CORD PROLAPSE

Umbilical cord prolapse may be defined as the descent of the umbilical cord through the cervix alongside or past the presenting part in the presence of ruptured membrane. Mean length of umbilical cord at Term is 55-60cm and Normal rage is (35-80cm).

Umbilical cord prolapse (UCP) is an uncommon obstetric emergency that can have significant neonatal morbidity and/or morality.

INCIDENCE: 0.1-0.6%

With breech presentation increases higher to 1%

Factors associated with cord prolapse

| **General** | **Procedure related** |
|---|---|
| Excessive Cord Length. | Artificial rupture of membrane with high presenting part |
| Multiparity | |
| Low birth weight (< 2.5 kg | Vaginal manipulations of the fetus with ruptured membranes |
| Preterm labour (< 37+0 weeks) | External cephalic version (during procedure) |
| Fetal congenital anomalies | Internal podalic version |
| Breech presentation | Stabilizing induction of labour |
| Transverse, oblique and unstable lie | Insertion of intrauterine prcs5urc transducer |
| Second twin | Large balloon catheter induction of labour |
| Polyhydramnios | |
| Unengaged presenting part | |
| Low-lying placenta | |

### Important points

- With transverse, oblique or unstable lie, elective admission to hospital after 37+0 weeks of gestation should be discussed and women in the community should be advised to present urgently if there are signs of labour or suspicion of membrane rupture.
- Women with non-cephalic presentations and preterm pre labour rupture of membranes should be recommended inpatient care.
- Artificial membrane rupture should be avoided whenever possible if the presenting part is high.
- If it becomes necessary to rupture the membrane with a high presenting Part. This should be performed with arrangement in place for immediate caesarean section.
- Upward pressure on the presenting part should be kept to a minimum in women during vaginal examination because of the risk of upward displacement of the presenting part and cord prolapse.

- Rupture of membranes should be avoided if on vaginal examination the cord *is* felt below the presenting part.
- When cord presentation is diagnosed in established labour, caesarean section is usually indicated.
- Cord presentation or prolapse should be excluded at every vaginal examination in labour and after spontaneous rupture of membranes if risk factors are present.
- Fetal heart rate monitoring in labour, the fetal heart rate should be auscultated after every vaginal examination in labour and after spontaneous membrane rupture.

**Diagnosis:**

The diagnosis of cord prolapse should be suspected in any patient who develops FHR Abnormalities after Rupture of membranes. Either spontaneous or artificial. The heart rate abnormalities usually observed are:

1. Sustained bradycardia
2. Variable deceleration

Patient should be re-examined and diagnosis should be confirmed by palpation of cord alongside the presenting part or in the cervix or vagina.

**Management:**

- When cord prolapse is diagnosed before full dilatation, assistance should be immediately called and preparation s made for immediate birth in theatre.
- To prevent vasospasm, there should be immediate replacement of loops of cord lying outside the vagina.
- To prevent cord compression, it is recommended that the presenting part be elevated either manually or by filling the urinary bladder.
- Cord compression can be further reduced by the mother adopting the knee- chest or left lateral (preferably with head down and pillow under the left hip) position.
- Tocolysis can be considered while preparing for caesarean section if there are persistent fetal heart rate abnormalities after attempts to prevent compression mechanically, particularly when birth is likely to be delayed.
- Caesarean section is the recommended mode of delivery in cases of cord prolapse when vaginal birth is not imminent in order to prevent hypoxic acidosis.
- Vaginal birth in most cases, can be attempted at full dilation, if it is anticipated that birth would be accomplished quickly and safely, using standard technique and taking care to avoid impingement of the cord when possible.

# SHOULDER DYSTOCIA

Shoulder dystocia is defined as a vaginal cephalic delivery that requires additional obstetric maneuvers to deliver the fetus after the head has delivered and gentle traction has been unsuccessful in delivering the shoulders.

**Incidence: 0.58% - 0.7%**

## Risk Factor:

### Pre labour

- Macrosomia
- Poorly controlled gestational and insulin-dependent diabetes (infants of diabetic mother have 2-4 fold increases risk of S.D, than infants of same weight born to non-diabetic mother)
- Maternal obesity
- Previous shoulder dystocia (recurrence rate is 25%)

### Intrapartum

- Induction of labour
- Prolong first stage
- Secondary arrest
- Prolong second stage of labour
- Oxytocin infusion
- Instrumental delivery

### Prevention

- Diagnosis and optimal control of gestational and insulin-dependent diabetics,
- Reduction of maternal obesity.
- Careful plan for mode of delivery in women with previous shoulder dystocia delivery.
- Elective caesarian section should be considered with an estimated fetal weight > 4.5kg, to reduce potential morbidity for pregnancies complicated by pre-existing diabetes or GDM.
-

**Diagnosis:**

Timely management of S.D requires prompt recognition always observe for signs of shoulder dystocia.

Difficulty with delivery of face and chin

2. Fetal head remain tightly applied to vulva and even retracting (turtleneck sign).
3. Failure of restitution of fetal head
4. Failure of shoulders to descend

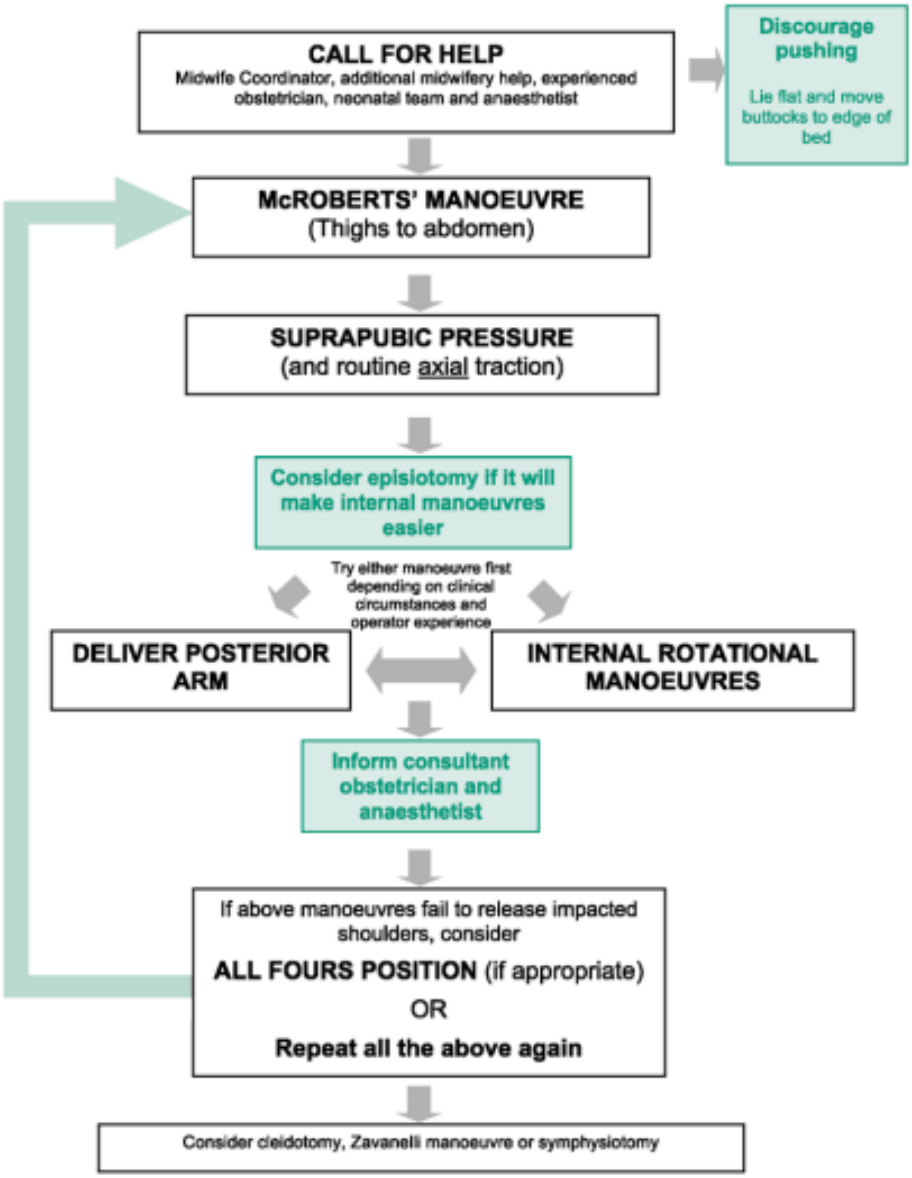
**Management:**

Shoulder dystocia should be managed systematically as shown in algorithm.

**Important Points:**

- Fundal pressure should be not used.
- Mc Robert's maneuver is simple, rapid and effective intervention with success rates as high as 90%
- Head to body delivery time should be less than 5min to avoid hypoxic injury.
- Perinatal mortality rates increase after 5min.

## Algorithm for the management of Shoulder Dystocia




**Baby to be reviewed by neonatologist after birth and referred for Consultant Neonatal review if any concerns**

**DOCUMENT ALL ACTIONS ON PROFORMA AND COMPLETE CLINICAL INCIDENT REPORTING FORM.**

**Maternal complications**

1. Increased perineal trauma (third- and fourth-degree tear) 3.8%
2. Postpartum haemorrhage (11%)
3. Psychological trauma.
4. Vaginal laceration
5. Cervical fear
6. Bladder rupture
7. Uterine rupture
8. Symphyseal separation.
9. Sacroiliac joint dislocation
10. Lateral femoral cutaneous neuropathy

**Fetal complication**

- Brachial Plexus injury (2.3-16% at birth reducing to 1-3% at 12 months of age permanent neurological dysfunction in 10%)
- Fractured clavicle or humerus (1-2%)
- Hypoxic brain injury.
- Pneumothorax.

# PUERPERAL SEPSIS/PYREXIA

The infection occurring at any time between the onset of rupture of membranes , labour and the 42nd day postpartum with fever and one of the following abnormal smell/foul odour of discharge , delay in uterine involution.
Responsible for 10% direct maternal death in Africa and Asia.

**Causes:**

| Infective cause | Non infective cause |
|---|---|
| Genital tract infections includes<br>Prelabour rupture of membranes prolong<br>Caesarean section /episiotomy wound<br>Intrapartum chorioamnionitis<br>Prolong labour<br>Multiple pelvic examination<br>Lacerations of genital tract<br>Cervical cerclage<br>Non genital causes<br>Mastitis(15%)<br>Urinary tract infection(2-4%)<br>Septic Thrombophlebitis<br>Respiratory tract infections | Obesity<br>Diabetes<br>Corticosteroid therapy<br>Immunosuppressant medications<br>Anemia<br>Thrombophlebitis<br>VTE |

**Organism commonly associated with Puerperal sepsis:**

| Aerobes | Anaerobes | Miscellaneous |
|---|---|---|
| Beta hemolytic strep.(A,B,D) | Peptococcus | Chlamydia trachomatis |
| Staph.epidermidis and aureus<br>E.coli,H.influenzae, klebsella, proteus<br>Pseudomonas aeruginosa | Bateriodes and fusobacterium | Mycoplasma hominis<br>Urea plasma urealyticum |

## Diagnosis:

**Detailed Hx and examination**

| Symptoms | Signs |
|---|---|
| Fever & rigors | Pyrexia |
| Diarrhea or vomiting | Tachycardia |
| Breast engorgement | Breathlessness |
| Offensive vaginal discharge | Hypotension |
| Abdominal and pelvic pain | Uterus boggy, tender and large |
| Wound infection | Paralytic ileus |
| Persistent vaginal bleeding/ secondary PPH | Boggyness in pelvis (abcess) |
| Productive cough & urinary symptoms | Indurated adnexae (parametritis)<br>Infected wounds |

## Investigations:

| General | Specific |
|---|---|
| CBC ,Serum electrolytes, CRP | Blood culture |
| | Throat and wound swab/vaginal swabs<br>Urine C/E<br>Serum lactate, ABGs , clotting serum<br>Chest X-ray and pelvic scan |

## Treatment:

| Supportive | Antibiotics | Surgical |
|---|---|---|
| IV Hydration(resuscitation in golden hour)<br>Analgesics<br>Wound care<br>ICE packs for pain from perineum or mastitis. | Broad spectrum antibiotics(Combination of clindamycin and aminoglycosides is appropriate for treatment.)<br>Tetracycline should be avoided in breast feeding. | Incision & drainage of breast abcess<br>Secondary repair of wound dehiscence<br>Drainage of pelvic hematoma and abcess. |

Antibiotics are continued till the patient has made significant clinical improvement and has been afebrile for at least 24 hours in endometritis.

## Prevention:

Increased awareness of general hygiene

- Women with suspected UTI during antenatal period should be investigated
- Advice should be offered with regard to breast feeding.
- Prevention and treatment of pre-existing anemia.
- Rigid Antiseptic measures taken during labour and delivery
- Prophylactic antibiotics and good surgical approach at caesarean.

## Red flags for puerperal sepsis:

- Pyrexia >38◦C
- Sustained tachycardia >90bpm
- Breathlessness(respiratory rate of >20 breaths /min)
- Abdominal/chest pain
- Uterine/renal angle pain
- Anxious /distressed.

# 3. Obstetrical Complications

## MULTIPLE GESTATION

**Definition**

Defined as pregnancies consisting of 2 or more than 2 fetuses with one or more than one amniotic cavities and placenta.

**Incidences**

- 16 in 1000 pregnancies.

**Risk factors for dizygotic twinning**

- Assisted reproductive techniques ( IVF and Ovulation induction)
- Increasing maternal age
- High parity
- Black race
- Maternal family history

**Classification**

- Number of fetuses
- Number of fertilized eggs (zygosity)
- Number of placentas (chorionicity)
- Number of amniotic cavities (amnionicity)
- If division of zygote occur on:

| 1-3 day | 4-7 Day | 8-13 Day | >13 Day |
|---|---|---|---|
| DCDA 70% | MCDA 20% | MCMA 1% | Conjoined Twin |

| Fetal risk | Maternal risk |
|---|---|
| Pre mature delivery :<br>52% before 37 week<br>10.7 % before 32 week<br>7% before 30 week<br>Perinatal mortality is 6 times higher than in singleton<br>10% in triplets<br>Vanished one twin ,one molar with normal<br>Death of 1 twin associated with poor outcome of other co twin<br>IUGR 25%<br>Fetal abnormalities<br>Twin to twin transfusion syndrome (TTTS) occur in 15% monochorionic twins<br>10% in Dia-amniotic and 5% in Monoamniotic<br>Twin reversal arterial perfusion (TRAP)<br>Interlocking twins conjoint twin<br>Umbilical cord accidents<br>Still birth rate is 14.9% | Anemia<br>Miscarriages<br>Preterm labour<br>Pre-eclampsia<br>Polyhydramnios<br>Operative delivery and increased stay in hospital<br>Increased problems with breastfeeding<br>More chances of developing postnatal depression<br>Increased risk of postpartum hemorrhage<br>Increased risk of placental abruption<br>Increased risk of UTI |

**FETAL DEMISE of 1 TWIN:**

- ✔ <14 weeks: fetus gets compressed(fetus papyraceus)
- ✔ > 14 weeks : surviving twin is at risk of death 15 % , neurological damage 26%

## Management:

Aim of management should be prevention of complications , early detection and management

**History:**

- Hyperemesis gravidarum
- Excessive enlargement of abdomen
- Respiratory problem in later half of pregnancy
- Early onset PIH

**P/A Examination:**

- Fundal height larger than gestational age.
- Multiple fetal parts palpable.
- Fetal hearts at 2 different places may be audible.

**Investigations (**baseline investigations and ultrasonography)

**USS: 11WK to 13+6WK for**

- Gestational age estimation (use large baby)
- Chorionicity --- lambda sign/ T sign + membrane thickness
- Screen for down syndrome

**Maternal Care**

- Book at 10 weeks
- Anemia: CBC at booking, 20 to 24 weeks if low repeat at 25 weeks
- High dose iron and Folic acid throughout pregnancy
- B.P + proteinuria at each appointment
- Loprin 75mg daily if risk factor for PIH from 12 weeks
- Anomaly scam at 18 -21+6 weeks
- Serial growth scam

MCMA: At 16th-20 week (every 2 week) then at 24weeks, 28-32weeks , 34-36weeks
DC: 20 weeks – every 4 weeks
**Delivery:** 60% deliver spontaneous <37 weeks
If cervical length is 25mm or less at 23-24 week it is likely to be delivered before 34 week
Offer elective birth:

- M.C: at 36 weeks after steroid
- D.C: at 37 weeks
- Triplets: at 35 weeks after steroid

Delivery should be in a tertiary care hospital (with expert obstetrician/midwife/anesthetist, O.T facility and pediatrician)

**Mode of delivery (DCDA)**

- Vaginal delivery is indicated if the 1st twin is cephalic, there is no medical or obstetrical complication and there is spontaneous onset of labour and no contraindication for vaginal delivery.
- Cross match n arrange blood
- During labour continuous CTG of both twins is done and syntocinon can be used for augmentation with one to one monitoring.
- In delivery room there should be ultrasound machine at bed side two staff nurses n two resuscitation trollies
- Keep pediatrician ;blood bank and OT ready
- Analgesia, ideally epidural
- Deliver the first twin as singleton delivery but avoid syntocinon administration
- When 1st twin delivers then abdominal examination and USG is done to confirm presentation .
  - ✔ If 2nd twin cephalic then vaginal delivery is proceeded
  - ✔ If 2nd twin breech/transverse then do ECV or Internal podalic version.
  - ✔ Add syntocinon 10 unit or double the dose if already going
- Expertise should be available to manage the second twin if in non-cephalic presentation
- Manage the third stage actively and give 600microgram misoprostol prophylactically
- Keep the patient under observation to monitor PPH
- Provide extra support to women in postnatal period to look after two babies
- Complicated monochorionic pregnancies with TTTS or TRAP are generally delivered at 34 weeks usually by C-section
- C-section is advised when the first twin is non-cephalic

**Hazards of induction:**

1. Increase risk of C/Section due to:
    - failed induction
    - fetal heart rate abnormalities
    - second twin (4%)
2. Increase risk of PPH

**High Order Multiples**

INCIDENCE: 1 in 6000-8000

- NICE recommend at least 11 antenatal visits
- Average gestation at delivery 34 weeks
- Do not prolong beyond 36 weeks

# PROTOCOLS OF POLYHYDRAMNIOS

Ultrasound based diagnosis AFI >95$^{th}$ centile for gestation (when deep vertical pocket (DVP) >8 or amniotic fluid index (AFI) >18-25)
Incidence of polyhydramnios is 1-2%.

| Severity | DVP (cm) | AFI (cm) |
|---|---|---|
| Mild | 8-12 | 24-30 |
| Moderate | 12-15 | ≥30-<35 |
| Severe | >15 | ≥35 |

## Common Causes of Polyhydramnios:

Excessive amniotic fluid volume (AFV) result from either an increase in amniotic fluid production or abnormal amniotic fluid regulation and removal.

**Fetal**

- Malformation & genetic anomalies (esophageal atresia, trachea-esophageal fistula, duodenal atresia, anencephaly, diaphragmatic hernia)
- Fetal anemia
- TTTS (causes acute polyhydramnios)
- Chromosomal abnormalities (trisomy 21,18,13)
- Non immune hydrops

**Maternal**

- Diabetes mellitus

## Idiopathic:

**Other -rare causes**

- TORCH infections
- Fetal neuromuscular disorder
- Sacrococcygeal teratoma

**Suspect Polyhydramnios:**

When women present with increased abdominal distension, abdominal discomfort, twin pregnancy, and if on abdominal examination:

- SFH >gestational age
- Tense and tender abdomen with shiny skin
- Fetal parts difficult to palpate
- Excessive fetal movements

## Confirm Diagnosis On Ultrasound

**Management**

It will depend on clinical assessment & investigations for cause of polyhydramnios and hence fetal prognosis.

**History**

- H/O fever, flu like illness in 1st trimester
- If anomaly scan done (any anomaly present )
- H /O anomalies in previous pregnancies
- Any significant drug history e.g. lithium (psychotropic)
- H/O diabetes mellitus

**Investigations**

1. OBS USG for
    - Diagnosis and severity of polyhydramnios
    - Fetal number ,chorionicity , severity of polyhydramnios and fetal growth
    - Fetal anomalies – anencephaly, duodenal atresia, trachea-esophageal fistula
    - Fetal anemia – presence of pericardial effusion / hydrops fetalis
    - MCA peak systolic velocity >1.5 MOM – refer to fetal medicine unit
2. Maternal blood group with Rh factor
3. Indirect coomb's test
4. OGTT
5. TORCH Serology (If indicated)

**Treatment options**

1. Amnioreduction -in case of severe maternal discomfort, respiratory symptoms
2. Pharmacological treatment: NSAID's ( Indomethacin 50 to 200 mg) <32 weeks
3. Cervical cerclage – if <24 weeks and cervical length after amnioreduction is <2.5cm
4. Strict control of blood sugar levels if patient is diabetic

**Time of delivery**

- According to cause
- Induction carries high chance of c/section so better is to wait for spontaneous onset of labour

**Complications anticipated**

- Malpresentation
- Preterm labour
- Cord prolapse
- Placental abruption
- Higher rates of operative delivery
- Maternal abdominal discomfort and respiratory distress
- PPH

**During labour**

- Avoid ARM
- If SROM occurs, immediately look for:
    - Cord prolapse/extremity prolapse
    - Placental abruption.

**NOTE**

After amnioreduction re-accumulation may develop rapidly and repeated amnioreductions can cause chorioamnionitis.

# OLIGOHYDROMNIOS

**Oligohydramnios** is defined as AFI < 5th centile for gestational age, MVP < 2cm , AFI < 5cm
**Borderline oligohydramnios:** AFI 5-8 cm
**Severe oligohydramnios:** AFI <5cm

## Causes Of Oligohydramnios:

| **Maternal** | **Fetal** | **Utero Placental Insufficiency** | **Drugs** | **Idiopathic** |
|---|---|---|---|---|
| Hypertension<br>Chronic kidney disease<br>Diabetes insipidus<br>Maternal dehydration | Rupture of membranes<br>Renal agenesis<br>Posterior urethral valves<br>TTTS<br>Polycystic Kidneys<br>Postdated pregnancy<br>PROM/PPROM | Preeclampsia<br>Chronic hypertension<br>Thrombophilia<br>Collagen vascular disease | ACE inhibitors<br>NSAID's<br>Prostaglandins synthase inhibitor | (65%) |

Suspected if symphysio fundal height is smaller than dates. Confirmed by ultrasound

**MANAGEMENT**

- Initial assessment for cause
- Assess for rupture of membranes
- Assess for IUGR on ultrasound / Doppler USG
- Fetal anomaly scan if not done previously

## Management Of Isolated Oligohydramnios

| | |
|---|---|
| Borderline AFI | Surveillance with 2 weekly growth scan, weekly AFI and Doppler |
| If severe oligohydramnios at 37 weeks | Induction of labour |
| Severe oligohydramnios < 24 weeks | Termination of pregnancy (risk of lung hypoplasia) |
| Severe oligohydramnios at 24-36 weeks (Steroid cover should be given) | Twice weekly CTG<br>Weekly scan for AFI till term<br>Doppler scan weekly |
| If renal agenesis or bilateral multi-cystic kidney disease | Termination of pregnancy |
| Postdates | delivery |

There is some evidence that maternal hydration increases AFI temporarily.

## Intrapartum Monitoring :

Continuous intrapartum FHR monitoring as there is risk of cord compression and IUD during labour pains.

**Complications:**

- IUGR , poor fetal growth
- Pulmonary hypoplasia
- Limb malformation

# PRE-TERM PRE-LABOUR RUPTURE OF MEMBRANES

Rupture of membrane b/w 24-36+6 weeks before onset of labour.
Responsible for 1/3$^{rd}$ of all preterm deliveries.

**Incidence:**
8-10% of all pregnancies
50% women delivered within one week & 75% with in 2 weeks of PPROM (this is not incidence)

**Causes:**

- Ascending infection (vaginal infections)
- APH
- Cervical weakness
- Multiple gestation
- Polyhydramnios
- Smoking
- History of PPROM in previous pregnancy

**Symptoms:**
Sudden gush of watery fluid from vagina, followed by recurrent dampness.

**Signs:**

- Moist vagina
- Liquor draining from cervical OS.
- Smell of liquor

**Diagnosis:**
Sterile speculum Examination:

- Pooling of amniotic fluid is observed.
    - If no pooling of amniotic fluid then

**Bed side Test:**

1. IGFBP-I (Insulin like growth factor binding protein-1) or PAMG-I (placental alpha microglobulin-1) should be considered. These biochemical markers have high level of sensitivity (96%) and specificity (98%). But not available in our hospitals.
2. Fetal fibronectin.
3. Amino-dye test.

**Note:**

- Ultrasound may be useful to support the clinical diagnosis of PRE-PROM.
- No further test is required if women is in established labour.
- If pre-PROM is not confirmed treat the cause for discharge.
- I-cels count + CRP (poor predictive value)

**Risk Assessment:**

- Maternal: Chorioamnionitis, abruption, ↑ risk of operative delivery.
- Fetal: preterm delivery, neonatal infections, cord prolapse, increase risk of pulmonary hypoplasia

## Diagnosis of Clinical Infection Depends Upon:

- **Clinical assessment**
  Pulse, Temperature, uterine tenderness, reduced fetal movement, offensive vaginal discharge.
- **Maternal Test**
  C-reactive protein & white cell count (total leucocyte count to support clinical diagnosis)
- **Fetal Heart Rate Monitoring**
  CTG

If the result of clinical assessment or any of the tests are not consistent with each other then, continue to observe & repeat the test.

White cell count will rise 24hours following administration of corticosteroid and return to baseline 3days following administration.

C-reactive protein most informative.

  - Sensitivity : 68.7%
  - Specificity: 77.1%

**Magnesium Sulphate**:

For neuro-protection of the baby

- In woman who has pre-PROM or is in established labour or is having a planned pre-term birth within 24hours.
- Magnesium sulphate should be **considered** when pre-term birth is anticipated between 30-33+6 weeks.
- Intravenous magnesium sulphate should be **offered** between 24-29+6 weeks of gestation (greatest beneficial effects)
- No effect after 34weeks

**Tocolysis:**

- Only for steroid cover and in utero transfer

# Management:

- Hospitalization
- Consent for prematurity

**Daily Observation:**

- Maternal pulse and temperature 4 hourly
- Fetal heart rate 4 hourly
- FKCC, CTG
- Inspection of vaginal discharge on pad for change of color or odour (meconium/pus), twice a day.
- Uterine palpation for tenderness twice daily

**Alternate day tests:**

- Total leukocyte count
- C reactive protein

**Weekly procedure:**

- Ultrasound for AFI/Biophysical profile

**Antibiotics:**

Tab. Erythrocin 250mg*QID for 10 days following the diagnosis of pre-PROM or until woman is in established labour(whatever is sooner).

- Consider oral penicillin/amoxiclav in whom erythromycin is contraindicated.
- Do not offer co-amoxiclav as prophylaxis of intrauterine infection in patient with Pre-Prom (to avoid necrotizing enterocolitis)

Rationale for the antibiotic recommendation

i- Reduce chorioamnionitis

ii- Prolonged latency
(Significant reduction in no. of babies born within 48hours.)

iii- Improve neonatal outcome
(By reducing risk of neonatal infection and need of surfactant and oxygen therapy).

- Steroid cover: Inj. Dexamethasone 6mg1/M*four doses 12 hours apart (RCOG).
- MgSo4 **(**for neuro-protection, if required)
- Conservative management till 37weeks
- Immediate delivery if signs and symptoms of chorioamnionitis or advance labour develop.

**Delivery:**

- If no contraindication to continuing the pregnancy, woman should be offered expectant management until 37 weeks.

**Subsequent pregnancies:**

- Risk of Pre-PROM in subsequent pregnancies increases
- Short inter pregnancy interval is associated with greater risk
- Patient should be booked with consultant obstetrician
- Clinician may offer, these woman, genital tract screening for infection & serial TVS to determine the cervical length (but evidence supporting these intervention is lacking)

# PRE-TERM DELIVERY

Delivery between 24-36+6 weeks is defined as preterm delivery with weight of the baby above 500grm.
Moderate to late preterm = 32-36+6 weeks
Very preterm birth = 27-31+6 weeks
Extreme preterm birth = 24-26+6 weeks

## Incidence:

**Etiology**

- Cervical incompetency
- Infections
- Preterm pre-labor rupture of membranes
- Multiple pregnancies d/t Uterine over distension
- Poly hydramnios
- Pyelonephritis, appendicitis, pneumonia, any surgical procedure.
- Uterine or cervical anomalies
    - Uterine → Unicornuate uterus + fibroid
    - Cervical → Cone biopsy + cervical

**Risk Factors**

- Low BMI
- **Anemia**
- Short inter-pregnancy interval (<1y)
- Maternal age (Extremes of age, Teenage and > 35(high risk))
- Nulliparous or grand multiparous
- Previous hx preterm delivery( if one previous preterm risk is 20%, if previous 2 preterm then risk is 35-40%)

**Management**

- History
- Examination
- Investigation
- Therapy
    - Steroid cover
    - $MgSo_4$ for fetal neuroprotection
    - Tocolysis
      e.g β-agonist like ritodrine, oxytocin antagonist i.e Atosiban. $Ca^{+}$-channel blocker like nefidil (drug of choice)
    - Progesterone Supplements (for uterine quiescence)
    - Lifestyle modification

**Prevention of Preterm Delivery**

- Progesterone supplement
- Cervical cerclage
  Cervical length surveillance with serial measurement of cervical length throughout the second and early third trimester is now used in women at high risk of preterm delivery.
- Treatment of infection (Bv, CTBS, Asymptomatic bacteriuria)

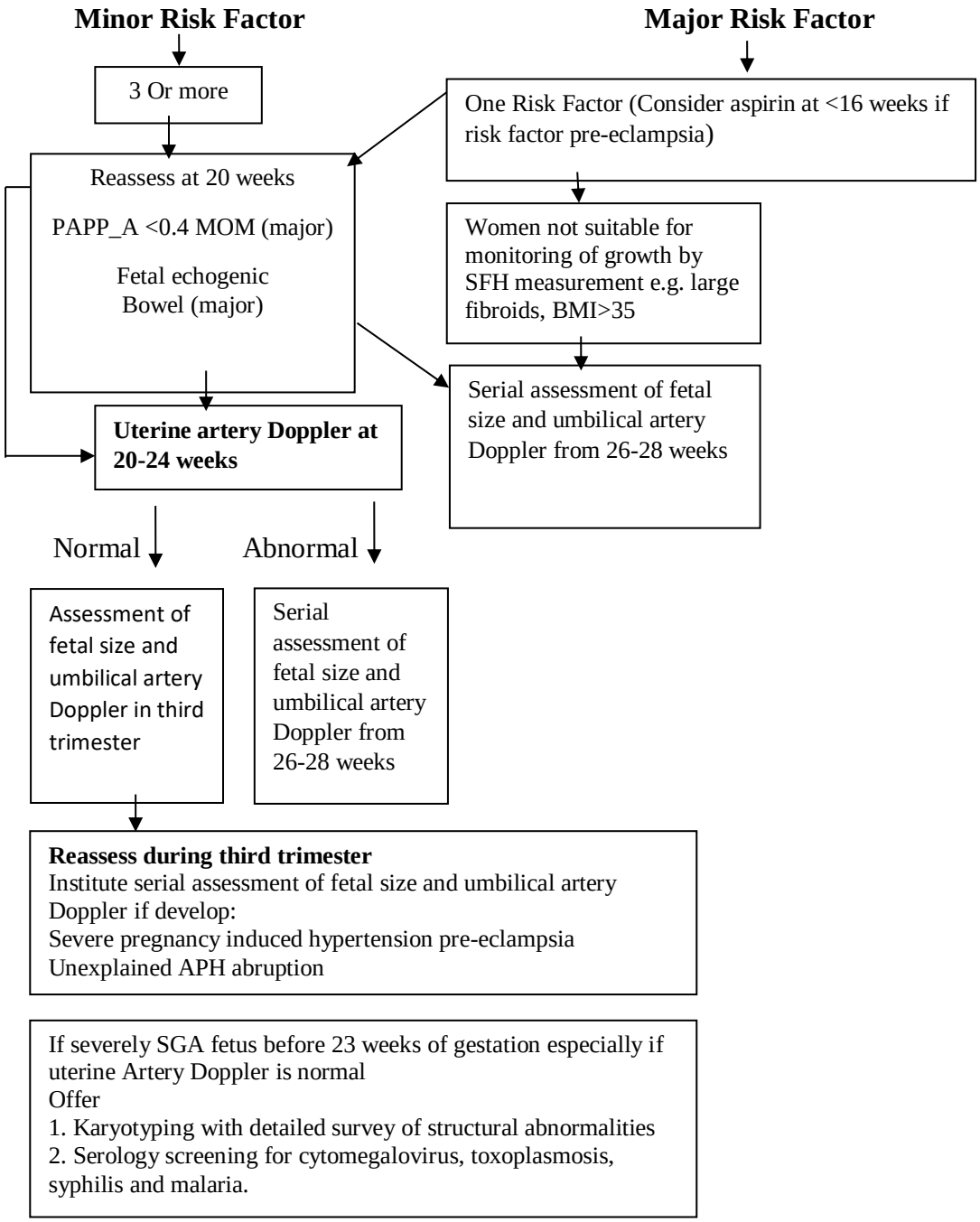
# SMALL FOR GESTATIONAL AGE

**SGA:** is defined as EFW or abdominal circumference <10$^{th}$ centile for gestational age
**D/D:** constitutionally small babies, symmetrical IUGR, Asymmetrical IUGR
**Sever SGA**: EFW or AC < 3$^{rd}$ centile.

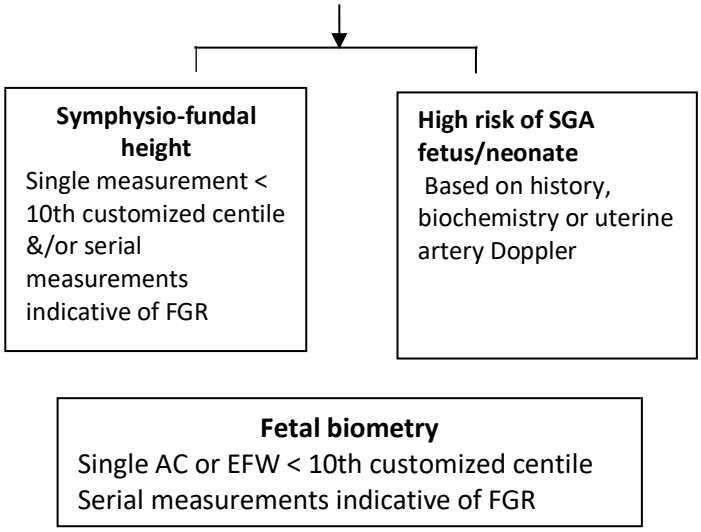
- Serial assessment of growth velocity is superior to single estimate of abdominal circumference to accurately assess the change in fetal size (AC, EFW).
- Two measurements of AC or EFW at least 3 weeks apart are required
- Accuracy of symphysio-fundal height (SFH) measurement to detect SGA is highly variable.
  Sensitivity 27%
  Specificity 88%
- In women not suitable for monitoring of growth by SFH e.g.; large fibroid, BMI > 35, polyhydramnios and multiple pregnancy serial biometry should be routinely performed.
- Serial SFH is reliable after 24 weeks of gestational age to improve prediction of SGA.
- When serial SFH chart shows static or slow growth, advice USG measurement.

| **Booking assessment**<br>Menstrual history<br>Dating scan for confirmation of DOP<br>Detailed history for risk factors |
|---|
| **Minor risk factors for SGA**<br>Maternal age >35 years<br>Nulliparity<br>BMI<20<br>BMI 25-34.9<br>Smoker 1-10 cigarettes per day<br>Previous pre-eclampsia<br>Pregnancy interval < 6 months<br>Pregnancy interval >60 months |
| **Major risk factors for SGA**<br>Maternal age>40 years<br>Smoker >11 cigarettes per day<br>Cocaine<br>Daily vigorous exercise<br>Previous SGA baby, Maternal/Paternal SGA<br>Previous stillbirth<br>Chronic hypertension<br>Diabetes with vascular disease<br>Renal impairment<br>Antiphospholipid syndrome<br>Heavy bleeding similar to menses<br>PAPP-A < 0.4 MoM. (Down syndrome marker)<br>Pre-eclampsia, unexplained APH in current pregnancy<br>Severe PIH, fetal echogenic bowel |

## Women Require Sonographic Surveillance:

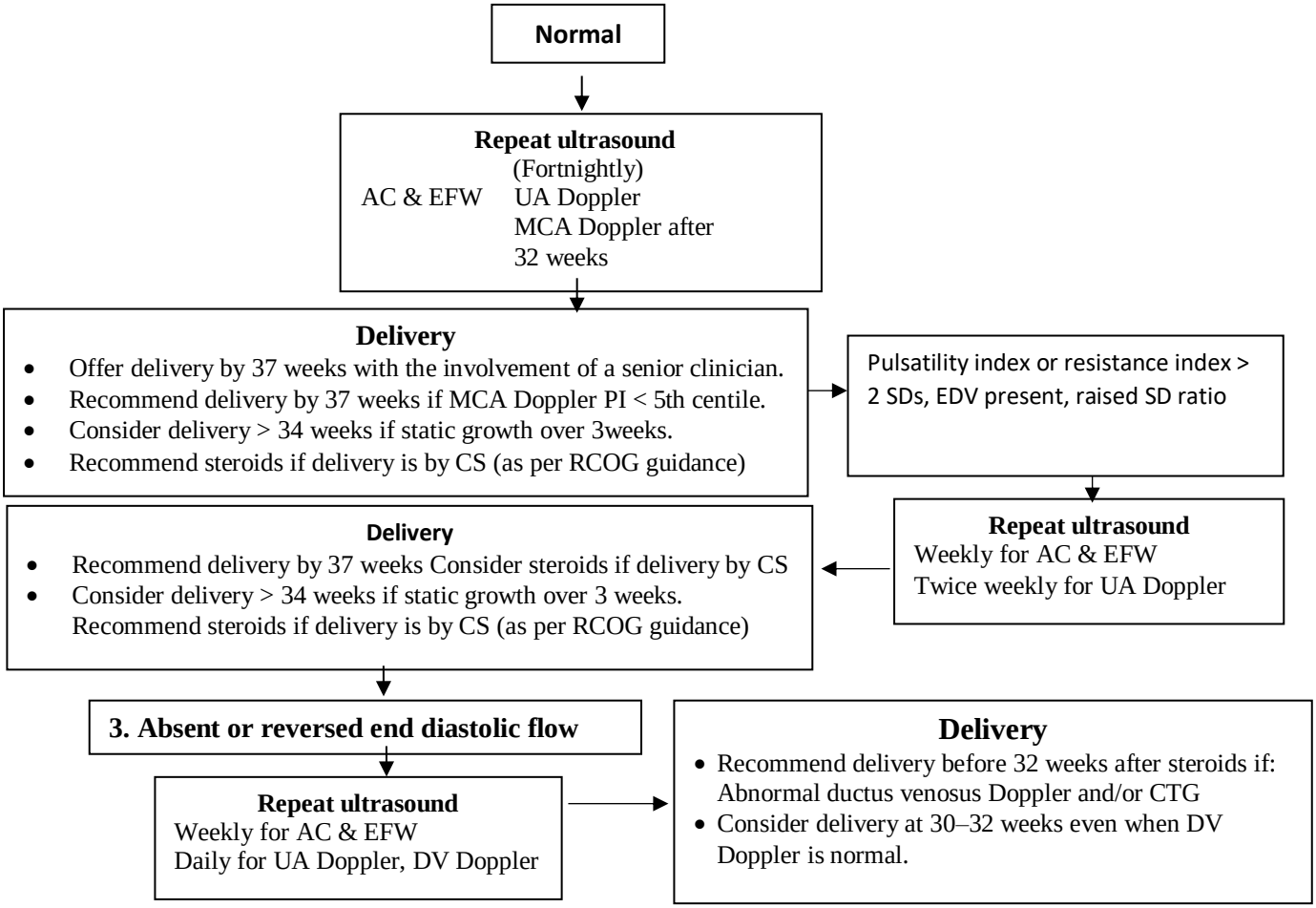


# Management of SGA Fetus




## UA Doppler

1. Normal
2. Pulsatility index or resistive index > 2 SDs, End diastolic velocity present
3. Absent or reversed end diastolic velocity



# THROMBOEMBOLISM IN PREGNANCY AND PUERPERIUM

1. Most common cause of direct maternal death in UK
2. 4 to 5 fold increased risk in pregnancy compared to non-pregnant state
3. In postpartum period risk is increased up to 20 folds
4. Incidence in pregnancy in developed countries without thrombo-prophylaxis is 0.1%
5. While in puerperium 1-2 % and is further increased following emergency Caesarean section

## Risk Factors:

**Virchow's Triad**

1. Blood stasis: blood flow in lower limbs slows up to 50% by 29week and persists for 6 week post partum.
2. Hypercoagulability : increase factor VIII , increase fibrinogen , decrease protein S , impaired fibrinolysis makes pregnancy hypercoagulable state.
3. Vessel wall damage : during delivery by any mean.

**Clinical features of DVT management should be in liaison with hematologist and physician**

- Pain (throbbing and cramping) and swelling in one leg, commonly left leg
- Pain in calf in association with dorsiflexion of foot(positive Homan's sign)
- Tenderness in calf on gentle touch with varying degree of redness and inflammation
- Pyrexia and increased TLC

## Specific investigations:

**Duplex ultrasound**

- Test of choice to diagnose DVT in pregnancy
- If ultrasound is negative but high clinical suspicion of DVT, stop anticoagulants and repeat scan on day 3 and 7

In iliac vein thrombosis doppler USG of iliac vein or magnetic resonance venography or conventional contrast venography should be performed.

**Treatment**:

- Elevation of limb
- Graduated elastic stockings should be used
- Analgesics
- Un fractionated heparin(UFH) and low molecular weight heparin (LMWH**)** is commonly used in pregnancy(dose and duration is same as in PE)

## Venous Thromboembolism

- Formation of a clot in a blood vessel(vein) that breaks loose and is carried by the blood stream to plug in another vessel e.g., lungs, brain, kidney.

## Pulmonary Embolism(PE)

### Diagnosis:

**Most common presentation**

- Mild breathlessness or Inspiratory chest pain, palpitations
- No cyanosis
- Slight tachycardia(>90bpm)
- Tachypnea
- Mild pyrexia(37.5°C)

**Rarely massive pulmonary embolism**
with sudden cardiorespiratory collapse and hemoptysis

### Investigations:

- **Chest x ray**: may identify other pulmonary diseases such as pneumonia, pneumothorax or lobar collapse.
- **Features of PE –** include atelectasis, effusion , focal opacities, regional oligaemia or pulmonary edema
- **ECG:** most common abnormalities are S1Q3T3 pattern , T wave inversion , right bundle branch block
- **In pulmonary embolism:**
  In hemodynamically stable patient 1st line investigation is chest X-ray and ECG. These are insufficient on their own to exclude or diagnose PE
- **Arterial blood gases analysis:** may show hypoxia
- Needs Duplex ultrasound of lower limbs if clinical evidence of DVT
- **Ventilation perfusion scan or CTPA** if no clinical evidence of DVT. (V/Q scan cannot be used if chest X-ray is abnormal)
- **D-dimer** is not used as investigation for DVT/PE in pregnancy but it has highly negative predictive value for DVT/PE.

## Treatment:

- Heparin compounds both LMWH and UFH(unfractionated heparin)can be used during pregnancy but LMWH is the treatment of choice.
- Because of their adverse effects on the fetus.
- **Why warfarin is not suitable for antinatal VTE treatment**
  Miscarriage, prematurity, low birthweight, neurodevelopmental problems and fetal and neonatal bleeding, characteristic embryopathy following fetal exposure in the first trimester vitamin K antagonists, such as warfarin, should not be used for antenatal VTE treatment.

- Before anticoagulant therapy is commenced, blood should be taken for a full blood count, coagulation screen, urea and electrolytes, and liver function tests.
- **LMWH**(Enoxaparin) therapeutic dosage- **1mg/kg SC×BD or 1.5 mg/kg SC×OD** against booking and early pregnancy weight.
- **UFH** loading dose of 80 units/kg, followed by a continuous intravenous infusion of 18 units/kg/hour. UFH is preferred over LMWH in acute life threatening pulmonary embolism.

  Measure APTT level 4–6 hours after the loading dose, 6 hours after any dose change and then at least daily when in the therapeutic range( target APTT ratio is usually 1.5–2.5 times the normal).

  Platelet count monitoring performed every 2–3 d from days 4 to 14 or until heparin is stopped
- Maintenance treatment of PE/DVT.

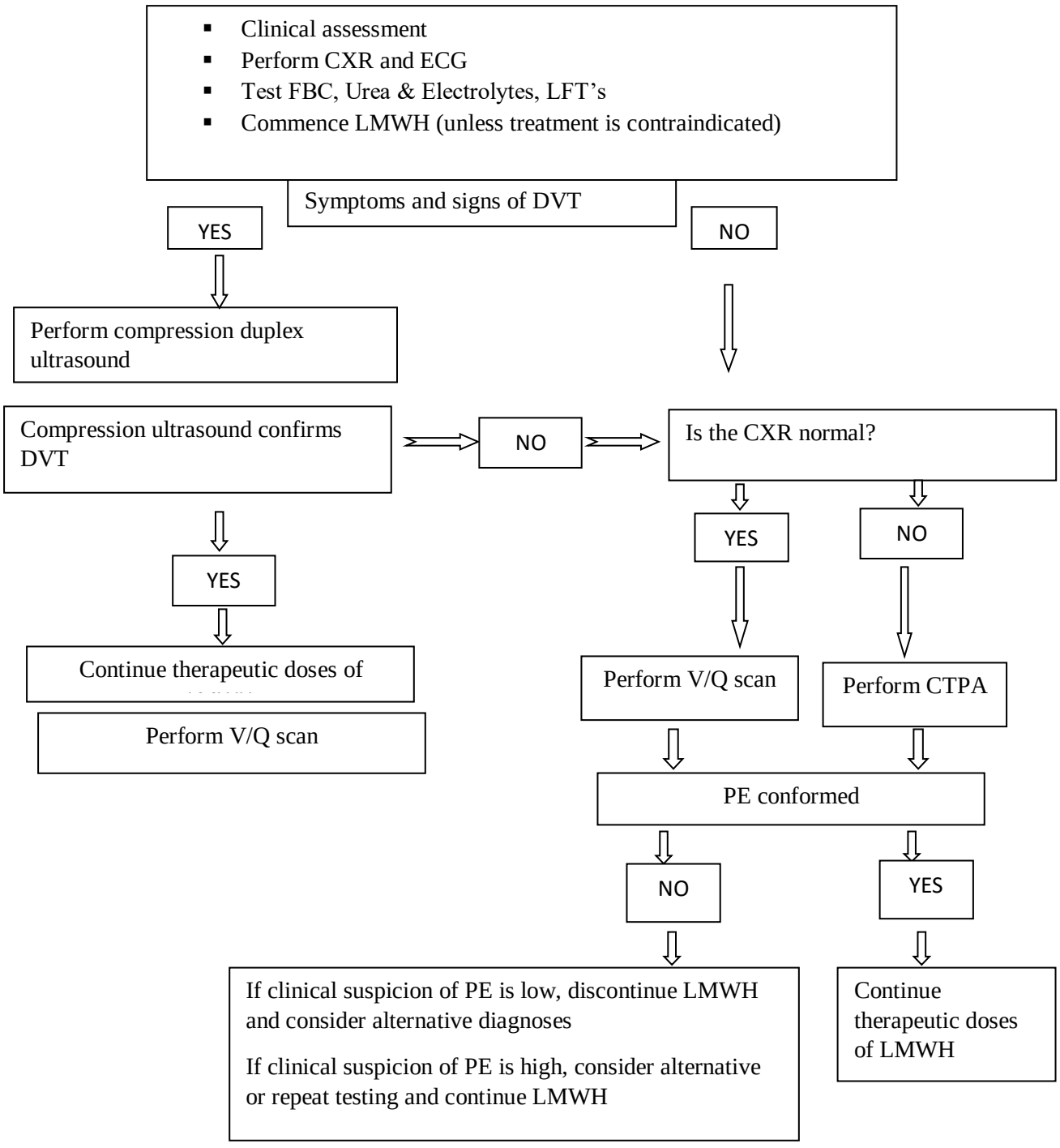
  Treatment with therapeutic doses of subcutaneous LMWH should be employed during the remainder of the pregnancy and for at least 6 weeks postnatal and until at least 3 months of treatment has been given in total.
- Postpartum warfarin should be avoided until at least the fifth day and for longer in women at increased risk of postpartum haemorrhage

  Starting dose is 10mg/day with monitoring of INR-from day 2 daily to maintain the INR between 2.0 and 3.0; heparin treatment should be continued until the INR is greater than 2.0 for at least 24 hours.
- Graduated elastic compression stocking applied to reduce edema
- Encourage mobilization
- Inferior vena cava filters may be considered for recurrent pulmonary embolism or if anticoagulant is contraindicated.
- Heparin/warfarin are not contraindicated in breastfeeding
- Surgical embolectomy may be required in life threatening massive pulmonary embolism.

## Anticoagulation during labour and delivery

- If the women is in labour she should not inject any further heparin and LMWH maintenance therapy should be discontinued 24 hours prior to planned delivery(planned c/s or induction)
- To avoid risk of epidural hematoma regional anesthesia should be avoided until at least 12hrs after the last dose(prophylactic) of LMWH(24hrs if she is on therapeutic dose)
- LMWH should not be given for 4 hours after the use of spinal anaesthesia or after the epidural catheter has been removed, and the epidural catheter should not be removed within 12 hours of the most recent injection
- Postpartum warfarin should be avoided until at least the fifth day and for longer in women at increased risk of postpartum haemorrhage
- Women at risk of hemorrhage e.g., PPH should be managed with Unfractionated heparin and anti-embolic stockings.
- In women on therapeutic dose wound drains should be considered.

## Algorithm for the Management of Suspected PE:



## Thromboprophylaxis in Pregnancy

Assessment of risk factors should be done in pre-pregnancy, pregnancy and in postnatal period.

| **Risk Factors** | **Management** |
|---|---|
| **High risk**<br>Any previous VTE except a single event post surgery | Antenatal prophylaxis with LMWH<br>(high prophylactic dose or therapeutic dose if Antithrombin deficiency, APLS or recurrent VTE) |
| **Intermediate risk**<br>Post surgery VTE<br>High risk thrombophilia<br>**Medical comorbidities** e.g. cancer, heart failure, IBD,type1 DM with nephropathy, nephrotic syndrome, sickle cell disease<br>Any surgical procedure in pregnancy<br>OHSS | Consider antenatal prophylaxis with LMWH |
| **Low risk**<br>BMI >30kg/m2<br>Age >35<br>Parity>3<br>Smoker<br>gross varicose veins<br>current pre-eclampsia<br>multiple pregnancy<br>immobility<br>family history of unprovoked VTE<br>hospital admission<br>IVF/ART<br>Current systemic infection | 4 or more risk factors=prophylaxis from 1st trimester<br>3 or more=prophylaxis from 28 weeks<br>Less than 3 risk factors=mobilize and avoid dehydration- no pharmacological thromboprophylaxis. |

### Suggested Thromboprophylaxis Doses for Antenatal and Postnatal LMWH

| **Weight** | **Enoxaparin** | **Dalteparin** |
|---|---|---|
| <50kg | 20mg | 2500units daily |
| 50-90kg | 40mg | 5000 units daily |
| 91-130kg | 60mg | 7500units daily |
| 131-170kg | 80mg | 10,000 units daily |
| >170kg | 0.6mg/kg/day | 75 units/kg/day |

## Prevention of VTE in Postnatal

| Risk Factors | Management |
|---|---|
| **High risk**<br>Any previous VTE<br>Anyone on antenatal LMWH<br>High risk thrombophilia<br>Low risk thrombophilia + family history | Prophylaxis LMWH for at least 6 weeks |
| **Intermediate risk**<br>Cesarean section in labour<br>BMI≥40kg/m2<br>Any surgical procedure in puerperium except immediate repair of perineum<br>Medical comorbidities | At least 10 days postnatal prophylaxis with LMWH<br><br>For 6 weeks if persisting or > 3 risk factors |
| **Low risk**<br>BMI >30kg/m2<br>Age >35<br>Parity>3<br>Smoker<br>>5days stay in hospital<br>gross varicose veins<br>current pre-eclampsia<br>immobility<br>family history of unprovoked VTE<br>multiple pregnancies<br>Current systemic infection<br>Preterm delivery or still birth in this pregnancy<br>Instrumental delivery-mid cavity forceps<br>Prolong labour >24hrs<br>PPH >1L or blood transfusion | 2 or more risk factor = prophylaxis with LMWH for 10days<br>Less than 2 risk factors = early mobilization and avoid dehydration |

# Rh INCOMPATIBILITY

Occurs when a Rhesus positive fetal red cells cross Rh-ve maternal circulation , causing sensitization of the maternal immune system subsequently giving rise to hemolytic disease of fetus and newborn.

- Does not effect 1st pregnancy as the primary response is weak and only IgM antibodies are produced that do not cross the placenta.
- As little as 0.5ml of fetal blood cells can cause isoimmunization.

| **Potential sensitizing events** |
|---|
| • Miscarriage or threatened miscarriage >12 weeks<br>• Medical/surgical termination at any gestation<br>• Repeated bleeding with abdominal pain before 12 weeks of gestation.<br>• Ectopic pregnancy<br>• Molar pregnancy<br>• Prenatal diagnostic procedures(CVS/amniocentesis)<br>• Abdominal trauma<br>• ECV<br>• Antepartum hemorrhage<br>• Still birth<br>• Traumatic delivery including c/s<br>• Manual removal of placenta |

## Prevention:

Antenatal prophylactic anti D is given if indirect coombs test is negative

- At 28 weeks(single dose)
- At 28 weeks and 34 weeks (2 dose regimen)

Anti D is given as prophylaxis and is useless once sensitization has occurred

| **Weeks** | **Anti D** |
|---|---|
| < 12 weeks spontaneous miscarriage | No anti D |
| < 12 weeks<br>Volume of fetal blood is small so sensitization is unlikely. | Give anti D **250 IU:**<br>if ectopic pregnancy, molar pregnancy, therapeutic termination, repeated uterine bleeding; heavy or associated with pain |
| 12-20 weeks | Give anti D **250 IU**<br>Within 72 hours of sensitizing event and perform kliehauer test |
| >20 weeks | For potentially sensitizing events a kliehauer test is required, pending its result a minimum anti D dose **500 IU** should be given in 72 hours.<br>Further anti D can be given if indicated by kliehauer test. |

In recurrent vaginal bleeding after 20+0 week anti d should be given at a minimum of 6 weeks interval

In Pakistan anti D of 1500IU is available that is routinely used.

## Investigation in sensitized women:

1. Kliehauer test
2. Monitor antibody levels every 2-4 weeks from booking in a sensitized woman

| Anti D level | Outcome |
|---|---|
| <4IU/ml | HDFN is unlikely |
| 4-15IU/ml | Moderate risk of HDFN |
| >15IU/ml | High risk of hydrops fetalis |

3. Measure MCA Doppler (sensitive 100%, false positive 12%)
4. At delivery take cord blood of baby for blood group and blood count .

## Management :

- Surveillance by serial antibody levels and regular ultrasound (growth, polyhydrominos and signs of fetal hydrops )+ Doppler of middle cerebral artery for assessing fetal anemia.
- **Increased MCA** : high probability of anemia
  **Treatment options include delivery / intrauterine fetal blood transfusion.**
- Delivery at a centre where facilities for neonatal support and blood transfusion /exchange transfusion are available.

# 4. Medical Disease in Pregnancy

## HYPERTENSIVE DISORDERS IN PREGNANCY

**Pregnancy induced hypertension:**
It is defined as blood pressure of ≥140/90mmHg recorded on two separate occasions 4 hours apart after 20$^{th}$ week of pregnancy in a previously normotensive woman without significant proteinuria.

**Chronic hypertension**:
Hypertension present at the booking visit or before 20 weeks or if the woman is already taking antihypertensive medication when referred to maternity services.

**Pre-eclampsia:**
New hypertension presenting after 20 weeks with significant proteinuria, more than 300mg/day of protein creatinine ratio more then 30mg/mmol or Albumin creatine ratio more then 8mg/mmol

**Chronic Hypertention with Superimposed Gestation/Hypertention/Pre-eclampsia:**

Exacerbation of hypertention with development of proteinuria or generalized edema that was not present previously.

**Transient Hypertension:**
Development of hypertension during late pregnancy with no signs of pre-eclampsia or pre-existing hypertension.

**Fulminating Pre-eclampsia/Imminent Eclampsia:**
Symptomatic protein uric hypertension.

**Eclampsia**:
A convulsive condition (generalized convulsions and/or coma ) associated with pre-eclampsia.

**Pregnancy Induced Hypertension**
Can be categorized as
Hypertension: blood pressure of 140/90–159/ 109 mmHg
Severe hypertension: blood pressure of 160/110 mmHg or more

## Management of PIH

| Mild to Moderate (B.P140/90-159/109mmHg) | Severe (B.P >160/110 mmHg) |
|---|---|
| ● No need of admission<br>● Manage on OPD basis<br>● Weekly follow up with home<br>● B.P chart (once or twice a week)<br>● Measure proteinuria (once or twice a week) at each visit<br>● Repeat CBC, RFT's, LFT's weekly (Local protocol: Repeat if proteinuria comes +ve, severe HTN, or develop symptoms HTN)<br>● USG for fetal growth 2 weekly(after 28 weeks of gestation)<br>**Target B.P 135/85 mmHg** | ● Admission in hospital<br>● Start antihypertensive drugs<br>● B.P record every 15-30minutes till B.P<160/110mmHg<br>● Measure dipstick proteinuria daily while admitted<br>● Repeat CBC, RFT's, and LFT's weekly<br>● USG for fetal growth 2 weekly(after 28 weeks of gestation)<br>**Target B.P 135/85 mmHg** |

### Timing of Birth:

Patient with uncomplicated PIH should wait for spontaneous onset of labour till 40 weeks and with sever hypertension controlled on antihypertensive should be delivered after 37 weeks and can be induced safely if indicated.

### Intrapartum:

During labour, measure blood pressure:

- Hourly, in women with hypertension
- Every 15–30 minutes until blood pressure is less than 160/110 mmHg in women with severe hypertension.
- Continue use of antenatal antihypertensive treatment during labour.
- Adequate pain management with epidural if available
- Maintain partogram
- Active management of third stage of labour .
- Avoid ergometrine

### Post-natal Management:

Monitor blood pressure daily for first 2 days.
At least once between day 3 and 5.
Reduce antihypertensive if BP less than 130/80 , change methyldopa to alternative within 2 days.

# Pre-Eclampsia

## Incidence: 2-3%

**Risk factors for identifying women at increased risk of pre-eclampsia.**

Any **single** high-risk factor

1. Hypertensive disease during a previous pregnancy
2. Chronic kidney disease
3. Autoimmune disease such as systemic lupus erythematous or antiphospholipid syndrome
4. Type 1 or type 2 diabetes
5. Chronic hypertension

Or **two or more** moderate-risk factors

1. First pregnancy
2. Age 40 years or older
3. Pregnancy interval of more than 10 years
4. Body mass index (BMI) of 35kg/m2 or more at first visit
5. Family history of pre-eclampsia
6. Multiple pregnancy

- Family history in first degree relative increases the risk of pre-eclampsia 4 to 8 fold
- Advise pregnant women at high risk of pre-eclampsia to take 75–150 mg of aspirin daily from 12 weeks until the birth of the baby.
  Chronic hypertension increases the risk of pre-eclampsia to over 20 %

**Symptoms of pre-eclampsia:**

- Severe headache
- Blurring of vision ,flashes of light
- Severe pain just below the ribs, epigastric pain
- Nausea ,vomiting
- Sudden swelling of the face, hands or feet.

**Proteinuria should be checked with dipstick screening**

If dipstick screening is positive (1+ or more) use protein: creatinine ratio to quantify proteinuria.

**(protein: creatinine ratio=30mg/mmol)**

If using albumin: creatinine ratio to quantify proteinuria , use **8mg/mmol** as diagnostic threshold.

## Management :

| **Mild to Moderate**140-159/90-109mmHg | **Severe** B.P >160/110mmHg |
|---|---|
| ● Admission in hospital<br>● - Offer antihypertensive treatment<br>● -B.P monitoring 4 hourly<br>● -Repeat dipstick testing for proteinuria only if indicated by presence of new symptoms<br>● Repeat CBC, RFT's, and LFT's twice weekly<br>● -USG for fetal growth, amniotic fluid volume and umbilical artery Doppler at presentation then after every 2 weeks | ● Admission in hospital<br>● Offer antihypertensive treatment<br>● B.P monitoring every 15-30 minutes till diastolic is <110mmHg.<br>● Repeat dipstick testing for proteinuria only if indicated by presence of new symptoms<br>● Repeat CBC, RFT's, and LFT's thrice weekly<br>● USG for fetal growth, amniotic fluid volume and umbilical artery Doppler at presentation then after every 2 weeks |

## Complications:

| **Maternal** | **Fetal** |
|---|---|
| Eclampsia<br>HELLP syndrome<br>CVA<br>Renal Failure<br>Liver failure<br>Pulmonary edema<br>Placental abruption<br>Increased risk of operative delivery | IUGR<br>Iatrogenic Preterm delivery<br>IUD<br>Oligohydramnios |

### Time & Mode of Delivery:

Offer birth at 37weeks

Early birth is indicated if:

- Uncontrolled B.P even with the use of 3 or more anti-hypertensive in appropriate dose.
- Progressive deterioration in LFT's, RFT's, hemolysis or platelets (or HELLP).
- Ongoing neurological complications such as headache, eclampsia or visual scotoma
- Reversed end diastolic flow of umbilical artery.
- Non-reassuring CTG
- Still birth

Offer course of steroids and intravenous MgSO4 if early delivery is planned in patients of pre-eclampsia.

### Intrapartum Care:

- Measure BP hourly.
- Every 15-30min till BP<160/110mm Hg.
- Continue use of antenatal hypertensive treatment

**Postnatal Management:**

- B.P Monitoring at least 4 times daily till patient is admitted.
- Continue anti-hypertensive treatment when B.P is >150/100 mmHg.
- Self-monitoring plan at discharge
    - Once a day B.P monitoring at home.
    - Be vigilant about neurological symptoms.
- If BP remains raised till 6 weeks post-partum refer to medical specialist to find out the cause of hypertension.

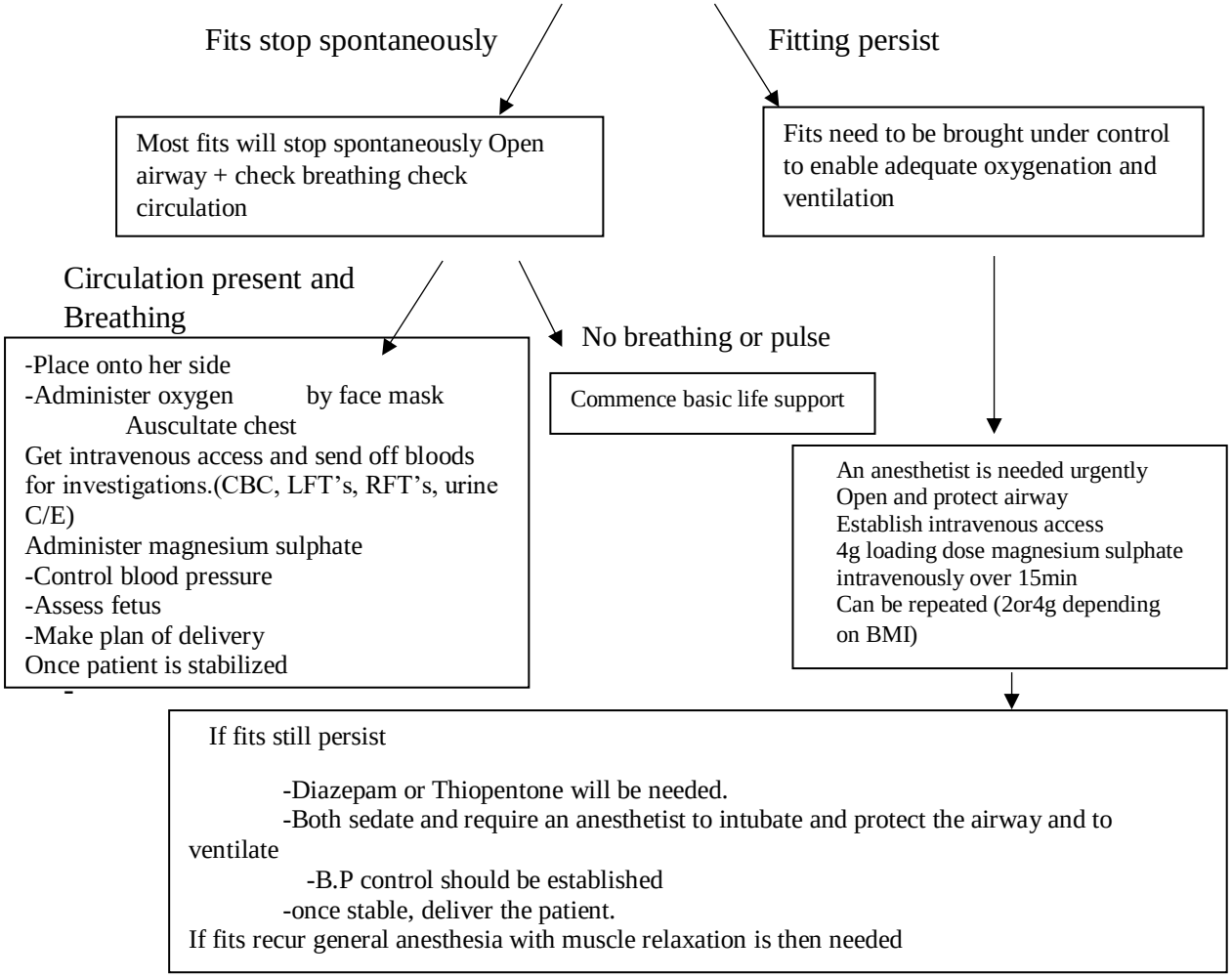
# Eclampsia:

Refers to occurrence of one or more generalized convulsions and/or coma in setting of pre-eclampsia in the absence of other neurological conditions.

Incidence: 27.5 cases per 100,000.

## Protocol for management of eclampsia

Seizures due to eclampsia are usually self-limiting. Call for help and approach patient safely. Turn her onto her side and try to stop her hurting herself during the fit.



# Chronic Hypertension:

### Pre Pregnancy Counselling:

- Weight management
- exercise
- healthy eating and lowering the amount of salt in their diet.
- Advise patients taking ARBs and ACE inhibitors to change to safe drugs for control BP
- Advise to take aspirin 75mg daily from 12 weeks till delivery.
- Aim for a target blood pressure of 135/85 mmHg( during pregnancy).

### Antenatal Appointments:

Weekly appointments in poor control
every 2 to 4 weeks if hypertension is well-controlled

### Monitoring:

Ultrasound for fetal growth and amniotic fluid volume assessment, and umbilical artery doppler velocimetry at 28 weeks, 32 weeks and 36 weeks.

### Timing of birth:

Patient with uncomplicated hypertension should wait for spontaneous onset of labour till 40 weeks and with sever hypertension controlled on antihypertensive should be delivered after 37 weeks and can be induced safely if indicated

### Intrapartum Care:

- Measure BP hourly.
- Every 15-30min till BP<160/110mm Hg.

Continue use of antenatal hypertensive treatment.

### Post-Partum Care:

- Aim to keep BP lower than 140/90mm Hg.
- Measure BP daily for first two days and at least once between day 3-5.
- Continue anti-hypertensive treatment and review within 2 weeks with GP or specialist
- If previously using methyldopa change within 2 days.

# Drugs Used In Hypertension

### Hydralazine

**MOA** relaxes smooth muscles of arterioles which decreases systemic vascular resistance lowering blood pressure.
It decreases. diastolic BP more than systolic.
**DOSE**: 10mg IV slowly.
**Repeat doses:** 5mg IV at 20min interval if necessary.
Effect of single dose last for 6 hours.
If no lasting effect of boluses consider IV injection at 2mg/h increasing by 0.5mg/h as required

**Adverse Effect**
CNS: headache, dizziness, peripheral neuropathy
CVS : palpitations , flushing, reflex tachycardia , angina
GIT : anorexia , nausea
SKIN : sweating , myalgia , skin rashes , fever

**Labetalol**
**MOA:** Reversible adrenorecptor alpha and beta blocker
**Doses:** 50mg IV slowly
If necessary repeat after 20 min as start IV injection of 100mg in 200ml N/S starting at 40mg/h increasing dose at half hourly intervals to maximum of 160mg/h
Oral acute : 200mg after every hour
Chronic: 200mg TDS
MAX 2400mg

**Advantages:**
It increase lung maturity
decreases tachycardia
increases utero-placental blood flow

**Side effect:**
decrease CO

**Nifedipine:**
MOA: bind to voltage gated Ca channels to block Ca channels then to decreases. Ca influx in cardiac and smooth muscle cells
Dose 10mg orally then Adalat Retard 10mg BD
Max 60mg/day
Adverse effect
CNS: dizziness
CVS: cardiac arrest , bradycardia , AV-block, CCF, hypotension, peripheral edema
GIT: nausea ,constipation
SKIN: flushing

**Methyldopa:**
MOA: converted to alpha methyl norepinephrine which is stored in adrenergic nerve granules where it stoichiometrically replace norepinephrine and is released by nerve stimulation to interact with presynaptic central alpha 2 adrenorecptor to decrease synaptic flow to decrease BP
Inhibit dopa decarboxylase to decrease stores of norepinephrine in nervous system
Dec. renal vascular resistance probably alpha methyl norepinephrine is poorer vasoconstriction than norepinephrine
Dose: 750mg/day to max. 4gm
Side effects:
CNS : sedation , nightmares, depression , extrapyramidal sign
CVS : Orthostatic hypotension, rebound hypotension on sudden withdrawal
BLOOD : hemolytic anemia
LIVER : hepatitis

ENDO: lactation

**MgSO4:** Mg levels 2-4 mmol/l

With increasing level: Feeling of warmth, flushes, double vision, loss of reflexes, respiratory depression, cardiac arrest

Dose : 10g IM stat ( 5g on each buttock)

4g IV loading dose, followed by 1g/hr for 24 hours as maintenance dose

Monitor R/R , UOP, reflexes

## PATHOLOGY AND PATHOPHYSIOLOGY

### Placental Bed:

Normal pregnancy

Spiral artery in decidua basalis invaded by cytotrophoblast replaced by fibrinoid.

1st wave of invasion complete by end of 1st trimester

2nd wave of invasion occur early in 2nd trimester involve myometrial segment of spiral arteries

### Preeclampsia:

Primary wave of invasion is impaired ½ to 2/3 of decidual arteries goes physiological change

2nd invasion fails to occur which result in restricted placental blood flow and muscular vessels remain sensitive to vasomotor stimuli.

### Acute Atherosis:

It is non specific vascular lesion seen in PIH.

In it foam cells are present in damaged vessel wall in basal arteries decidua parietalis - intramyometrial segment of spiral arteries:

It is "Necrotizing Arteriopathy" Characterized by fibrinoid necrosis accumulation of lipid laden macrophages + damaged cells , fibroblast proliferation and mononucleated perivascular infiltrate.

"The vascular changes are not specific to PIH are also seen in IUGR without pre-eclampsia."

### Kidney:

Changes seen are

i. Glomerular endotheliosis: Characterized by swelling and lipid vacuolation of cytoplasm of glomerular cells in capillary loops
ii. Endothelial cells: remain normal except for few intracytoplasmic hyaline droplets
iii. Mesangium: increase mesangial matrix
iv. Deposits of IgM + fibrin
v. Proximal tubules dilatation + epithelial thinning of proximal tubules

All these changes cause decrease GFR , decrease Creatinine clearance , Proteinuria d/t glomerular damage

Mechanism of loss of strong -ve charge with normally repel proteins.

Hypocalciuria

Hyperuricemia reflect tubules dysfunction(benefit : act as antioxidant)

### Liver:

Hepatic lesions seen are:

Hemorrhagic , periportal fibrin deposition areas of infarction and necrosis

These changes are not specific to pre-eclampsia can also be seen in obstetric hemorrhage

i. Liver enzyme increase plasma bilirubin increase ( clinically jaundice is occasional)
ii. Sub capsular hematoma → epigastric pain and vomiting.
iii. Liver rupture → shoulder tip pain.

## Help Syndrome:

Hemolysis → characteristic red cell morphology & LDH > 600 units/l
Elevated liver enzymes → AST > 70 units/l
Low platelets → $<100*10^9$/l
It may be present when BP is not grossly elevated
Importance: increased of complication as DIC
Increased risk of neonatal morbidity and mortality

## Brain:

Eclampsia is associated with cerebral vasospasm.
Later increased vascular resistance → loss of cerebral auto regulation → less than resistance & cerebral edema.
Cortical blinding → may be d/t edema or petechial hemorrhages d/t vasospasm in post cerebral circulation.

## Vasomotor function:

In normal pregnancy → down regulation of Angiotensin II receptors → insensitivity to Angiotensin II as well as other agonists as noradrenaline.
In pre-eclampsia → constricted intra vascular volume → loss of acquired insensitivity to Angiotensin II & noradrenaline → decreased production of PGI & NO.

## Coagulation, Fibrinolysis and Platelets:

Normal Pregnancy.

- Coagulation factors increase
  Increase fibrinogen
  Increase factor VIII
  Increase vWF
- Fibrinolysis decrease
  Increase PAI -2(Plasminogen activation inhibitor II)
  Decrease protein S
  Increased resistance to protein C
- Despite of increase procoagulant potential → minor degree of activation of coagulation & subsequent fibrinolysis.
  Increase FDPs & No evidence of endothelial damage.

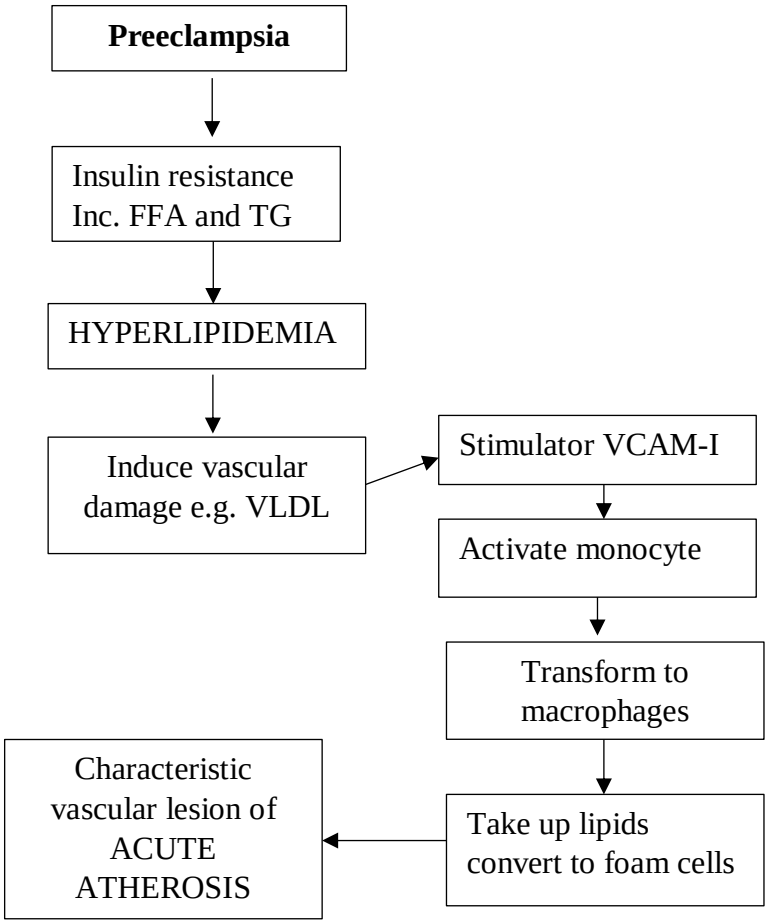
## In Pre-Eclampsia:

- Vascular damage (acute atherosis) → fibrin deposition → coagulation activation more marked in uteroplacental circulation.
- Increase fibronogen, vWF, more than the level in normal pregnancy.
- Coagulation activation is secondary to endothelial activation by:
  Increase level of thromboembolisms & fibronectin → Reflect endothelial damage
  Tissue factor expression on endothelial cells stimulated by cytokine as TNF "Proinflammatory state"

- Increase in endothelial derived tPA → reflecting endothelial activates
- Increase PAI – 1 → reflect endothelial activator
- Decrease PAI-2 impaired placental function

"Routine Coagulation Tests are Essential & Normal Unless Complicated by DIC"
"Normal PT, APTT & slightly prolonged TT, do not imply coagulation activation as these tests are insensitive"

**Metabolic Factors:**
Increase BMI particularly central obesity increase risk factor



# PROTOCOL for DIABETES IN PREGNANCY

Diabetes complicate 10% of pregnancies in Pakistan.
Includes pre-existing DM (Type I (7.5 %), Type II (5%) & GDM (87.5%)).

## Pre-Pregnancy Counseling: Pre-existing DM (Type I & II)

### Information

- Explain to the patient about the effects of diabetes on pregnancy. About the effects of pregnancy on diabetes.
- Explain the general risks and management plan
- Explain the role of diet and exercise
- Tight control of glycemic levels with HbA1c <6% (Target: FBS 90-126g/dl, Pre meal 72-126g/dl) if HBA1c greater than 85mmol/l 30% risk of miscarriage
- Weight reduction if obese(BMI :30kg/$m^2$).
- Stop smoking if smoking.
- Maintain menstrual calendar.
- Folic acid(5mg/day) three month before conception till 12 weeks of gestation.
- Contraception till the optimal control.
- Management of any co morbidity such as retinopathy or nephropathy.
- Control of other medical disorders if any with a drug which can be safely continued in pregnancy (ACE inhibitors, Angiotensin receptor antagonists, statins should be stopped/modified).

### Frequency of monitoring

1. Monitoring of Hb A1C monthly
2. 4 blood sugar levels if on insulin
3. FBS, Pre meal, Post meal, Bed time

### Screening for GDM

Identify risk factors on first antenatal visit on the basis of history and examination and offer 2-hours 75gm OGTT if risk factors present.

**OGTT at booking visit then at 24-28weeks (if prev. OGTT normal)**
H/O GDM in previous-pregnancy.
Previous H/O unexplained, still birth, congenital anomalies

**OGTT at 24-28 weeks**

BMI>27 kg/m2
1st degree relatives with DM
Prev. H/O macrosomia, PCO'S

## Diagnose GDM

**Nice Criteria:**

- FBS ≥ 100mg/dl (5.6mmol)
- A 2hour plasma glucose level of ≥140mg/dl(7.8mmol/l)
- Offer all other pregnant ladies (without risk factors)-FBS for screening of GDM

## WHO Criteria:

- BSF: 5.1mmol/l (91mg/dl)

- 1hour: 10.0mmol/l (180mg/dl)
- 2 hour: 8.5mmol/l (153mg/dl)

## Antenatal Management

- Multidisciplinary approach
- Early booking
- Dating scan with basic investigation
- Frequency of visits at 1-2wks interval
- Monitor for development of hypertension
- If pre-existing DM, start loprin 75mg/day from 12 weeks until birth of baby to prevent preeclampsia
- Optimization of blood sugar at each visit
- **Target level**: BSF ≤95mg/dl
  If patient on insulin fasting >72mg/dl (5.3mmol/l)
  1hr post meal <140mg/dl (less than 7.8 mmol/l)
- **Start insulin** with, diet, exercise & metformin
  If FBS ≥ 126g/dl (7mmol/l) at diagnosis
  FBS 110 -<126mg/dl (6-6.9 mmol/l) and complications such as macrosomia & polyhydramnios
  **If not controlled** with diet, exercise & metformin with1-2 weeks
  Serial growth scans to detect macrosomia and polyhydramnios every 4 week from 28 to 36 week

## Frequency of blood sugar monitoring on daily basis:

- Patient on diet, exercise OR metformin fasting and 1hr postprandial.
- Patient on insulin before control 7BSL (before and 1 hour after each meal, bedtime)

After control 4 BSL fasting, premeal, post meal & bed time
Advise woman regarding risk of sign and symptoms of hypoglycemia.

| At booking | 16 weeks | 20weeks | 28 weeks | 32 weeks | 34 weeks | 36weeks |
|---|---|---|---|---|---|---|
| **1**.Dating scan<br>**2.**Fundoscopy (if not primarily done)<br>**3.**OGTT (If risk factors present)<br>**4**.S/Creatinine+ Proteinuria (if not done previously in 3 months)<br>**5.**If protein 500mg/day refers to nephrology<br>**6**. If >5g/day then thrombo- prophylaxis. | **1.** OGTT (if booked in 2nd trimester) with risk factors<br>**2**.Fudoscopy if previous retinopathy OR Previously fundoscopy not done. | **1.**USG – anomaly scan including 4-Chamber heart view+ outflow tract and vessels. | **1.**Growth scan + AFI<br>**2.**Fundoscopy- to known diabetic only | **1.**Growth scan + AFI<br>Repeat | No additional care pathway. | **1.**Growth scan<br>**2.**Provide information about time, mode & management of birth<br>**3.**Analgesia & Anesthesia<br>>Increased need of insulin during labor.<br>Post-partum issues<br>Contraception (postnatal) |

## Time & mode of birth:

Uncomplicated GDM spontaneous onset of labour can be awaited till 40 weeks.
Uncomplicated type I & type II DM 37-38+6 weeks.
Complicated type I & II (metabolic & other feto-maternal) consider elective birth before 37weeks
Women with DM/GDM controlled on insulin- Delivery to be planned 37-39 weeks.
Omit morning dose of insulin on the day of ELLSCS.

## Intrapartum care:

BSL * 1 hourly (target 4-7 mmol/L) (72-126mg/dl).

- **Type I DM**- Dextrose + insulin infusion from the onset of established labor
- Where BSL is not maintained b/w 4-7mmol/L in type II DM/GDM-consider insulin /Dextrose infusion

## Insulin infusion pump

- 10% Dextrose – 83ml/hr, 500ml: (25 Drops/min)
- 50 Units Actrapid in 50ml 0.9% Salina (Insulin)

The rate of insulin infusion is adjusted based on hourly glucose measurement as mentioned in table

| **Hourly blood glucose (mmol/L)** | **Insulin rate (units per hour = mL per hour)** | **Other action** |
|---|---|---|
| 3.0 or less | 0 | Repeat glucose test, < 3.1 check all lines and infusion pumps, call consultant |
| 3.1–3.9 | 0.5 | Repeat glucose test, give glucose if <4.0 and symptomatic* If hypo confirmed, check all lines and infusion pumps |
| 4.0–6.9 | 1 | |
| 7.0–7.9 | 2 | |
| 8.0–8.9 | 3 | |
| 9.0–10.9 | 4 | Call consultant |
| 11.0–16 | 6 | Stop dextrose, start 0.9% saline, call consultant |
| >16 | 8 | Stop dextrose, start 0.9% saline, call consultant |

Delivery in consultant- led unit
Expertise for instrumental delivery/ shoulder dystopia should be available.
**Anesthesia**: preferably spinal or epidural if GA then blood sugar monitoring half hourly from induction till patient become conscious.

## Post-natal Care

**Neonatal care:**

- BSL every 2-4 hourly routinely for 1st 24 hours.
- Frequent breast feeding(start within 30minuts of birth) until pre-feed BSL at least 2mmol/l
- Monitor for RDS, polycythemia, hyperbilirubinemia, hypocalcemia if indicated admit in neonatal unit if sign and symptoms present or preterm delivery
- Echocardiography(neonatal)f indicated

## Postnatal maternal care:

For insulin treated pre-existing DM dose of insulin should be reduced TO HALF
Women who were diagnosed with GDM (if blood sugar normal after delivery) discontinue blood glucose lowering therapy immediately after birth.

**FOLLOW UP GDM:** For GDM (if blood sugar N after delivery)

- **FBS / Hb A1C at 6-13 weeks postpartum**

| NORMAL | BORDERLINE | DIABETIES |
|---|---|---|
| FBS < 6.0mmol/L | FBS - 6.0-6.9mmol/L (108-126) | FBS > 6.9mmol/L > (126mg/dl) |
| Hb A/c <5.7 % | Hb A/c -5.7-6.4 % | Hb A1C > 6.4 % |

**DO NOT OFFER OGTT ON ROUTINE BASIS**

- Contraception
- Annual Hb A1C

**For preexisting DM**: Refer back to their routine diabetic care.

## Diet & insulin for DM/GDM

**Diet:** In women with BMI>27/kg/m2, gives 25 kcal/kg/day with carbohydrate restriction to 35-40 %.
Moderate exercise for at least 30minuts daily.
**Distribution of carbohydrates:**
**Breakfast:** 10-15% of total energy intake (TEI).
**Mid-morning, evening & bed time snake:** 5-10% TEI
**Lunch & Dinner:** 20-30% TEI
**Insulin:** Recommended initial dose is 0.7 units /kg body weight in 1st trimester.

- 0.8U /kg body weight in 2nd trimester.
- 0.9-1U/kg body weight in 3rd trimester.
- Dose adjusted according to the response to initial therapy.
- Of calculated dose, 2/3rd is given before breakfast divided as 2/3rd NPH insulin and 1/3rdregular
  Insulin one third in evening

# Effects of Diabetes in Pregnancy:

**Fetal risks**

| Antenatal | Intrapartum | Postpartum |
|---|---|---|
| Miscarriage | Shoulder dystocia. | Neonatal respiratory distress syndrome. |
| Congenital anomaly(2-3%major and 7-8% all) | birth injury | Neonatal hypoglycemia. |
| Prematurity | brachial plexus or bone injury(most common clavicle) | Poor feeding. |
| Risk of diabetes | | Neonatal jaundice. |
| Macrosomia | | Hypocalcaemia. |
| Stillbirth | | Hyponatremia. |
| | | Developing diabetes. |
| | | Becoming obese as children or adult. |

**Maternal risks:**

| Antenatal | Intrapartum | Postnatal |
|---|---|---|
| Hypoglycemia | Primary PPH | Secondary PPH |
| Polyhydraminos | Cervical Tears | Developing type 2 diabetes |
| Preterm labor | Perineal injury | infections |
| Anxiety<br>Vaginal infections | Nerve injury | |
| UTI, Worsening of retinal disease | Operative delivery | |
| Worsening of pre-existing renal impairment | | |
| Pre-eclampsia | | |

## Important Points to be Remembered:

- Pre pregnancy pre meal glucose should be 4-7mmol/l(72-126mg/dl)
- Congenital abnormalities 2-4 times more common than low-risk pregnancies
- 3 fold excess of cardiac and neural tube defects. Sacral agenesis is seen only in diabetes
- Malformations account for 40% of perinatal mortalities associated with diabetic pregnancies.
- Accelerated growth patterns are seen in late second and third trimester (associated with poor control).
- Risk of pre-eclampsia is 3 fold in women with diabetes.
- As serum creatinine increases , chances of successful pregnancy diminishes
    1. 80%:125-180µ/L
    2. 75%: 180-220 µ/L
    3. 60%: >220 µ/L
- Average insulin production: 40units/day (1 unit per hour)
- Insulin production/meal :4–6 unit/ meal
- Insulin production increases in pregnancy: 75units/day

# ANEMIA IN PREGNANCY

## WHO definition:

Hemoglobin concentration of less than 11.0g/dl or hematocrit < 30% in pregnancy is defined as anemia in pregnancy.

## WHO definition of anemia:

- Hb <110 g/l in the first trimester
- Hb<105 g/l after 12 weeks
- Hb<100 g/l immediately postpartum

## Incidence: 15 -25%

In Pakistan 40-80%
Iron deficiency 75%
Folic acid 10%

## Severity of Anemia:

Mild anemia hemoglobine 10.9g/dl - 10.0g/dl
Moderate anemia : hemoglobin 7.0g/dl - 10.0g/dl

Severe anemia: hemoglobin 4.0g/dl - 7.0g/dl

Very severe anemia : hemoglobin less than 4.0g/dl

## Types of anemias:

| Nutritional deficiency anemia | Hemoglobinopathies | Rare Types |
|---|---|---|
| Iron deficiency<br>Folic acid deficiency<br>Vit B12 deficiency | Thalassemia<br>Sickle cell disorder | Aplastic anemia<br>Autoimmune hemolytic anemia<br>polycythemia rubra Vera<br>paroxysmal nocturnal hemoglobinuria<br>leukemia<br>Hodgkin's disease |

# Iron Absorption:

Fe++ form from gut
Daily 10% of total iron ingested 1-1.5%
In Pregnancy absorption increases up to 30% (3mg/day)

## Requirement:

4mg/day average
Early half 2.5mg
Late half 6.5mg

Max absorption 3.5mg/day from dietary sources .if diet is insufficient to meet the fetal demand than maternal sources are used.
More than 2 years are required to replenish the maternal stores from the dietary source.

## Physiological Anemia:

In pregnancy, there is a physiological expansion of plasma volume beginning in the first trimester and plateauing by the third , which exceeds the increased production of red blood cells and haemoglobin. The resulting haemodilution contributes to the fall in Hb during pregnancy

- Plasma volume increases by 40%
- Red cell mass increases by 18%
- MCV and MCH remain normal.

## History:

### Symptoms of Anemia:

1. Fatigue, Lethargy, irritability ,lack of interest
2. Shortness of breath
3. Generalized body aches
4. Loss of appetite
5. Poor weight gain
6. Poor concentration

### Signs of Anemia:

1. Pallor(observed on palm, conjunctivae and base of tongue)
2. Koilonychia
3. Brittle nails
4. Tachycardia
5. Systolic Murmur ( observed at mitral area)

## Causes of Iron Deficiency Anemia:

### 1. Dietary habits:

Intake of tea, coffee, PICA, poor intake of iron containing diet i.e.: poultry, fish, red meat, leafy vegetables.

### 2. Malabsorption:

IBS, crohns disease, ulcerative colitis, celiac disease, worm infestation, intake of tea/coffee, simultaneous intake of iron and calcium.

### 3. Loss:

| Medical causes | Obstetric causes | Gynaecological causes |
|---|---|---|
| 1.Epistaxis<br>2.Gum bleeding<br>3.Haematemesis<br>4.Hemoptysis<br>5.Haemorrhoids<br>6.Recurrent UTI<br>7.Worm infestation | 1.Grandmultiparity<br>2.Short inter pregnancy interval<br>3.Antepartum haemorrhage<br>4.Postpartum hemorrhage<br>5.Twin pregnancy | 1.Menorrhagia |

| Effect of disease on pregnancy | |
|---|---|
| **Maternal** | **Fetal** |
| Fatigue<br>Exercise intolerance<br>Ice , clay, starch PICA<br>High cardiac output failure<br>Infection<br>Pre-eclampsia/abruption<br>Increased risk of PPH<br>(moderate anemia increases PPH by 50%, severe anemia increases PPH by 10 fold higher)<br>Preterm labour<br>Maternal depression | Perinatal morbidity/mortality<br>Prematurity<br>Fetal growth restriction<br>Impaired cerebral functions<br>Impaired iron dependent enzyme functions |

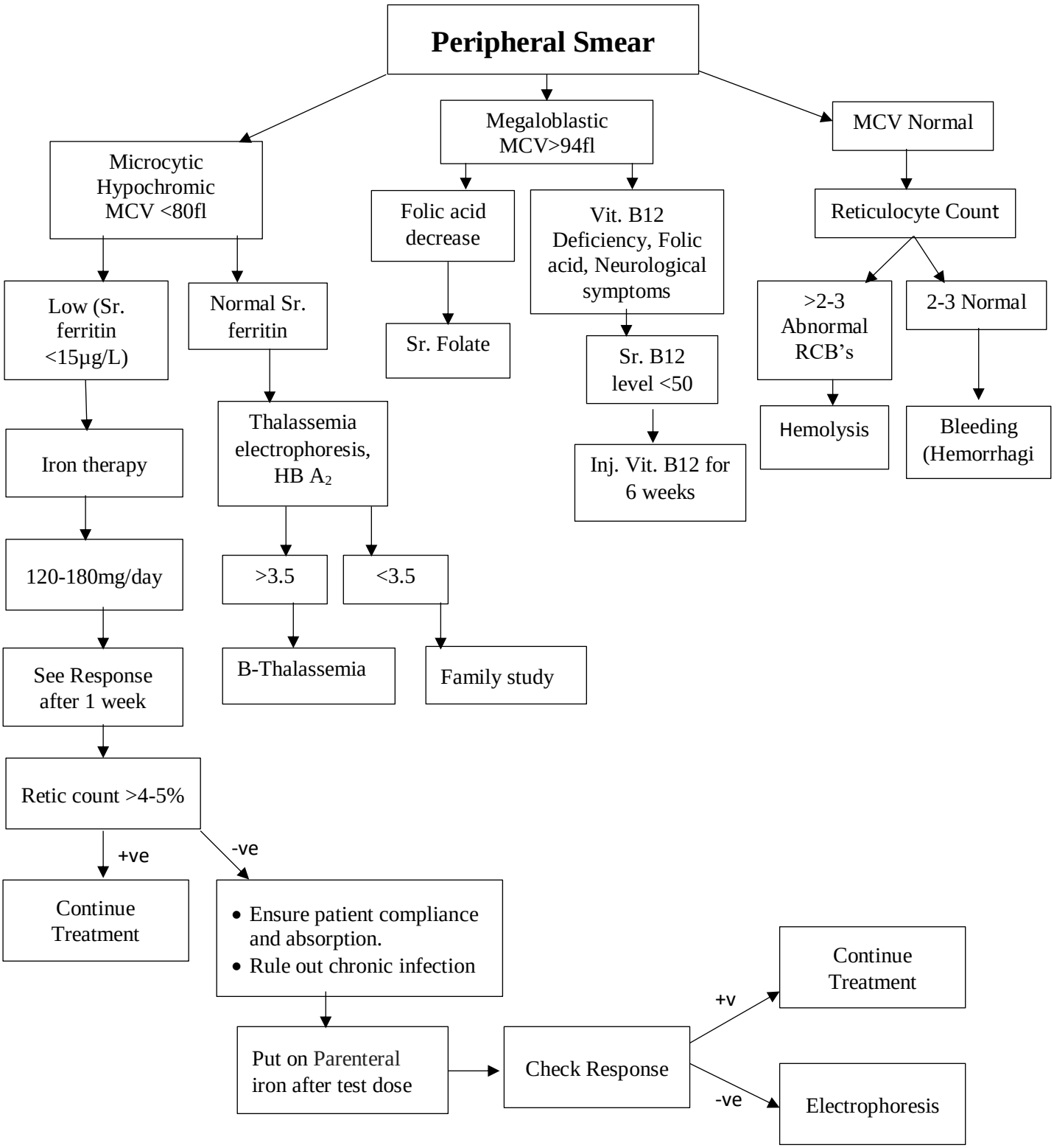
# Investigations:

1. CBC ⟶ Hemoglobin, Red cell indices: MCV, MCH, MCHC

| | **Iron deficiency anemia** | **Megaloblastic anemia** | **Thalassemia** |
|---|---|---|---|
| MCV | reduced | raised | reduced |
| MCH | reduced | raised | reduced |
| MCHC | reduced | normal | normal |

2. Serum Ferritin: Normal value 15-300µg/L
3. Peripheral smear: Microcytic and hypochromic anemia
4. Hb electrophoresis
5. Total iron binding capacity
6. Oral iron trial
   Follow up on compliance is checked by Retics count after trial of oral iron.
7. Stool examination for parasites
8. Urine examination(for UTI)
9. Deoxy uridine suppression test
(to differentiate between folate deficiency and vitamin B12 deficiency)

# Diagnosing Iron Deficiency Anemia in Pregnancy



## Management:

| Treatment of cause | Treatment of disease |
|---|---|
| If medical disorder then treat the relevant disease(upper GI and lower GI bleeds, IBS, ulcerative colitis, celiac disease)<br>If worm infestation(deworm the patient)<br>Advise to reduce intake of tea/coffee<br>Advise to stop simultaneous intake of iron and calcium.<br>Treat infections<br>Management of APH and PPH accordingly. | Before 28 weeks:<br>Iron rich diet<br>120-180mg/day oral iron till Hb 12g/dl then reduce dose to half(follow up by retics count to check compliance)at 1 week interval .<br>28 weeks-34 weeks:<br>Mild anemia: oral iron<br>Moderate to severe anemia: parenteral iron<br>≥34 weeks:<br>Vary from patient to patient.<br>If Hb <8g/dl then transfuse blood<br>If Hb>8g/dl then parenteral iron but keep blood arranged. And active management of 3rd stage of labour. |

# Iron Requirement in Pregnancy:

Iron stores: liver, spleen, bone marrow(contain 700-1000mg iron)

3% iron is absorbed from iron rich diet.

Daily iron requirement in pregnancy: 6.6mg.

Total iron requirement in pregnancy :1360mg

- Fetus and placenta 500mg
- Expansion of red cell mass 500mg
- Loss at delivery 180mg
- Lactation 180mg

# Indications of Blood Transfusion:

- Severe anemia resulting in CCF at any gestation to make the patient asymptomatic
- APH, placenta previa
- Patient at term with hemoglobin <8.0g/dl
- Patient at risk of PPH(polyhydramnios, multiple gestation, grand multiparity)

## Elemental iron content in oral iron preparations:

| Iron preparation | Tablet size | Elemental iron per tablet(mg) |
|---|---|---|
| Ferrous Sulfate | 200 | 65 |
| Ferrous Fumarate | 200 | 65 |
| Ferrous Gluconate | 300 | 35 |

- The newer preparations such as iron carboxymaltose which is given as a single dose over 15 minutes produces a faster response(approximately 10g/L improvement per week)particularly beneficial in women who presents late in pregnancy.
- Therapeutic dose of elemental iron in iron deficiency is 200mg per day .
- Prophylactic dose of elemental iron 30-60mg/day
- Fefol vit → Ferrous sulphate 150mg – elemental iron 47mg
- Iberet folic → Ferrous sulphate 525mg – elemental iron 105mg
- Sangobion → ferrous gluconate 250mg – elemental iron 30mg

# THYROID DISEASES IN PREGNANCY

Maternal Thyroid hormones are essential for maintenance of pregnancy, placental and fetal development

**Pre-Pregnancy Counseling**

- Lowest dose of drugs.
- Achieve euthyroid for 6 months before conception.
- Avoid conception for 6 months if received radioactive iodine.
- PTU is preferred in early pregnancy (<10 weeks)

**Hyperthyroidism**

- Incidence 1 in 500 pregnancies.
- 90% graves disease (autoimmune thyrotoxicosis) associated with hyperplastic goiter and exophthalmos.
- <5% of cases include toxic nodular goiter, subacute thyroiditis or carcinoma.

**Sign & symptoms:**

| General | Specific in pregnancy |
|---|---|
| • Exophthalmos<br>• Periorbital myxedema<br>• Tachycardia & tremors, anxiety, sweating.<br>• Heat intolerance, warm skin<br>• Weight loss, bowel disturbance<br>• Hyperemesis gravidarum, emotional irritability, insomnia, HTN. | • Poor weight gain in the presence of good Appetite .<br>• Tachycardia > 100 beats per minute that is unresponsive to Valsalva maneuver may indicate the disease. |

**Complications:**

| Fetal complication | Maternal complication |
|---|---|
| • Miscarriages, Preterm delivery<br>• IUGR<br>• IUD, still birth<br>• Neonatal thyrotoxicosis | • Pregnancy induced hypertention<br>• Pre-eclampsia<br>• Thyroid storm<br>• Cardiac failure |

**Investigations:**

- Baseline profile
- Obstetric ultrasound
- Specific investigation include Thyroid function test (free T3, T4 with pregnancy specific reference ranges) In normal pregnancy, total T3 and T4 increases, free T4 and T3 remains normal and TSH should be interpreted according to reference values of pregnancy. However, TSH decreases in hyperthyroidism.

(HCG & TSH share a common alpha subunit have similar beta subunit.TSH receptors are prone to stimulation by HCG , so in conditions like molar pregnancy, hyperemesis gravidarum and multiple pregnancy increase thyroid activity has been noted. Patient with hyperemesis gravidarum, molar pregnancy & multiple pregnancy can present with hyperthyroidism).

**Management:**

- Multidisciplinary approach
- Beta blockers for tachycardia (propranolol is recommended therapy)
- Anti-thyroid drugs:

  Propylthiouracil is the first drug of choice(50mg) 2 tablets X TDS (can cause agranulocytosis)

  Carbimazole (5mg) can be used in later months of pregnancy (can cause teratogenicity in 2-4% if exposed during 6-10 weeks gestation)

  **Minimal needed dose should be given as they both cross placenta and can cause neonatal hypothyroidism.**

  Radioactive iodine is contraindicated in pregnancy.

  If medical treatment fails or patient hypersensitive to anti-thyroid drugs then surgery indicated.

  Patient presented in third trimester with marked metabolic signs & symptoms should be hospitalized until disease is controlled.

**Monitoring:**

- Thyroid function test every 4-6 weeks
- Screening for agranulocytosis and hepatitis in those on thiomides
- Anomaly scan.(especially for fetal goiter)
- Serial Fetal USG( 4-6 weekly)
- Umbilical artery doppler monthly form 26-28 weeks in women with grave's disease

**Time and Mode of delivery:**

- It will depend upon maternal and fetal condition.

**Postpartum care:**

- Observe for worsening of symptoms
- Reduce the dose of thyroxin to pre-pregnancy level
- Patient continue to breast feeding if taking Carbimazole
- Check neonatal thyroid function test (cord sample and after 1-2 weeks)

**Breast Feeding:**

- Contraindicated with radioactive iodine
- Relative contraindicated with propylthiouracil (because concentration in breast milk &neonate serum is very low)
- Should be used in lowest possible dose and follow infant with thyroid function tests
- Neonatal TFT's after birth and 1-2 weeks later.

**Post-Partum Thyroiditis**:

- 5-10% of patient develop thyroid dysfunction in first few months post partum
- patient present with severe postpartum depression
- 90% resolve in 6- 10 months. First hyperthyroidism followed by hypothyroidism
- treatment require for3 months then re-evaluate with thyroxine
- condition can reoccur after every pregnancy (70%).
- Follow up with annual TSH T3, T4 measurements.

(histology from thyroid biopsies suggest a chronic thyroiditis with lymphatic infiltration but not fibrosis which is a typical feature of hashimoto's thyroiditis. Initial thyrotoxicosis is not due to increase production but release of preformed thyroxin from destroying follicles. Radioactive iodine and technetium uptake test can help to distinguish b/w postpartum thyroiditis(low uptake) and graves disease(high uptake)but lactation should not be continued during testing.

# Fetal and Neonatal Thyrotoxicosis

**Fetal Thyrotoxicosis:**

- Fetus require maternal T4 for normal fetal brain development during 1$^{st}$ trimester(10 weeks)
- When maternal TSH receptor stimulating antibodies cross the placenta they can cause fetal thyrotoxicosis. Most common if mother not taking anti thyroid drugs.
- Fetal thyroid are capable to respond to these antibodies after 20 weeks.

**Complications of fetal thyrotoxicosis:**

- Fetal tachycardia
- IUGR
- Goiter cause polyhydramnios & obstructed delivery
- Fetal cardiac failure leading to hydrops fetalis and death.
- Intellectual impairment
- Accelerator bone maturation
- Poor weight gain, poor feeding and sleeping

## Treatment

- Maternal antithyroid drugs
- Maternal thyroxine combined with antithyroid drugs if mother is euthyroid

## Neonatal Hyperthyroidism:

- Occur in 10 % of babies born to women with current or past history of graves disease (trans-placental passage of thyroid receptor stimulating antibodies)
- Treatment with antithyroid drugs is required until maternal antibodies are cleared
- Regular TFTs should be checked
- Without treatment mortality rate is 15%

**Thyroid Storm:**
Rare and acute life threatening exacerbation of thyrotoxicosis.

- Occasionally occurs in pregnancy or in puerperium.
- Maternal mortality 10-20 %
- Fetal mortality 20-50%

**Causes:**

- Previously undiagnosed hyperthyroidism with co-existing stress such as labour ,infection, trauma.
- Patient not taking medication.
- Rarely occur in already diagnosed and well controlled patient.

## Sign & symptoms:

- Marked hyperpyrexia
- Dehydration, diarrhea
- Tachycardia
- Atrial fibrillation/Cardiac failure
- Shock
- Psychosis, delirium, coma
- Fetal compromise

**Investigations:**

- Baseline labs
- Thyroid function test
- Serum electrolytes, glucose, calcium
- ECG

**Treatment:**

- Hospitalization
- Correction of precipitating factors
- Supplemental oxygen.
- Propranolol 40mg orally every 6 hours
- Sodium iodide 250-500mg I/V every 6hours
- Propylthiouracil 300-400mg orally every 6 hours
- Adequate hypothermic measures
- Hydration with glucose containing fluids
- Hydrocortisone 50-100mg I/V every 6 hours

# Hypothyroidism in pregnancy

- Incidence ⟶ 1% in pregnancy
- Causes include surgical removal, Radioactive therapy, Autoimmune and Treatment of hyperthyroidism

| Sign | Symptoms |
|---|---|
| • Anemia<br>• Coarse hair<br>• Bradycardia<br>• Cool and dry skin<br>• Weight gain<br>• Carpal tunnel syndromes | • Weakness and tiredness<br>• Cold intolerance<br>• Constipation<br>• Sleep disturbance<br>• Irritability<br>• Altered mental status |

## Complication due to Hypothyroidism:

| Fetal complication | Maternal complication |
|---|---|
| • Miscarriages<br>• Congenital anomalies<br>• IUGR<br>• Preterm delivery<br>• Goiter<br>• Neonatal low IQ<br>• Increase perinatal morbidity | • Hypertention<br>• Pulmonary embolism<br>• PPH |

## Investigations:

- Baseline investigation
- Obstetrical scan
- Thyroid function test (at booking & 28 weeks if euthyroid)

## Management:

- Tab. thyroxin 100 microgram per day till TSH is normal (max dose 300microgram /day)
- Repeat TSH level 4 weekly till normal
- If already on thyroxin then increase the dose by 30% at beginning of pregnancy and check TSH and T4 level 6-8 weekly.
- Avoid taking iron at the same time of oral thyroxin.

# CARDIAC DISEASE IN PREGNANCY

Overall incidence of serious heart disease complicating pregnancy is 1%.
Pre-pregnancy counseling

- Council the women in liaison with cardiologist
- Advise early booking.
- Discuss maternal and fetal risks.
- Obtain update on cardiac status.
- Optimize medical and surgical management.
- Safe and effective contraception
- Review the record (previous surgery, treatment and medication)
- Treat anemia, fever, infection and hypertension

## Advise against the pregnancy in

- Pre-existing pulmonary hypertension.
- Eisenmenger syndrome.
- Marfan syndrome.
- Sever cardiomyopathy.
- Large left to right shunt.
- Severe mitral stenosis <1cm$^2$.

## COMPLICATIONS:

### Maternal Risk

- Incidence of PIH 20%.
- Exacerbation of heart disease .
- Increase mortality, decrease life expectancy.
- Prolog hospital stay.
- Maternal mortality in serious condition can be as high as 30% - 50% (pulmonary hypertension, marfan syndrome)

### Fetal Risk

- Abruption 10%.
- IUGR 15%.
- Perinatal mortality 30%.
- Preterm labour .
- Congenital H.D 2.5-5%.

### Therapeutic Termination in

Cyanotic heart disease (right to left shunt)
Tetralogy of Fallot.
E.M syndrome.
Severe mitral stenosis < 1cm$^2$ in diameter.

## Classification according to NYHA staging

| | |
|---|---|
| Stage 1 | No symptoms and no limitations in ordinary physical activity, e.g. shortness of breath when walking, climbing stairs etc. |
| Stage 2 | Mild symptoms (mild shortness of breath and /or angina) and slight limitation during ordinary activity |
| Stage 3 | Marked limitation in activity due to symptoms, even during ordinary activity e.g. walking short distance (20-100m). Comfortable only at rest. |
| Stage 4 | Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients. |

**Antenatal care**

- Manage in liaison with cardiologist.
- Early booking .
- Access the functional class of patient.
- Avoid or minimize the aggravating factors e.g.
- Treat Anemia infection and hypertension.
- Routine physical examination BP , Pulse , JVP , Heart sound , edema and basal crepitations
- Advise anticoagulation if needed.
    - Congenital heart diseases with pulmonary hypertension.
    - Artificial valve replacement.
    - In or at risk of atrial fibrilization.

**Anticoagulation therapy**

- 1st trimester: LMWH.
- If patient wants to be treated by oral medication.
- Warfarin 13weeks to 36weeks.
- Aim to maintain INR/APT 2.5-3 times normal.

- Prophylactic antibiotic in certain conditions.
- Booking investigations.
- CBC, Urine C/E, Blood Group, Rh factor.
- PT, APTT, INR.
- Viral serology.
- ECG, Echocardiography (if not done in last 5 years).
- Obstetrical USG n fetal growth monitoring.
- X-ray chest with shield (if required).
- Assess indicators of heart failure e.g. cough, edema, tachycardia & basal crept.
- Identify risk factor for the development of heart failure in pregnancy.
- Do not use diuretics empirically, only when needed in case of pulmonary edema.
- Use beta blockers in cases of severe mitral stenosis.
- Replace warfarin with LMWH 10 days prior to delivery or at 36 weeks.
- Discontinue aspirin at 36 weeks.
- In the event of an urgent need to deliver a fully anticoagulated patient, warfarin may be reversed with fresh frozen plasma and Vitamin K
- LMWH with protamine sulphate.
- Mode of delivery and detailed intrapartum care should be written on card.

**Risk factor for the development of heart failure in pregnancy.**

- Respiratory and urinary infection
- Anemia
- Obesity
- Corticosteroid
- Tocolytics
- Multiple gestation
- Hypertension
- Arrhythmias
- Pain related stress
- Fluid over load

## Labour & delivery:

Preferred mode of delivery is vaginal unless contraindicated.

- Spontaneous onset of labour is ideal. Avoid induction of labour, augment with concentrated oxytocin infusion only if necessary (10 units in 500ml of ringers lactate).
- Monitor the labour in fully equipped room.
- For class III and IV monitor with CVP line.
- Avoid mental and physical stress.
- Adequate fluid and electrolyte balance.
- Endocarditis prophylaxis inj. Amoxicillin 2g IV + Inj. Gentamicin 1.5mg/kg IV at the onset of labour or ruptured membranes or prior to C-section followed by Amoxicillin 500mg orally 6 hours later (no routine endocarditis prophylaxis in low risk on complicated labor or delivery)
- Avoid supine position. Ideal is semi recumbent position with 30 degree left lateral tilt.
- Avoid hypotension esp. in case of pulmonary HTN & mitral stenosis.
- Monitor B.P, $O_2$ saturation & intake/output record.
- If needed intermittent $O_2$ inhalation during $1^{st}$ stage of labour.
- Maintain Partogram.
- Monitor fetal heart continuously .
- Adequate analgesia, epidural, if facility is there.
- Avoid lithotomy position.
- In case of severe mitral stenosis & pulmonary edema give 60mg inj. Lasix IV 15 mins prior to delivery (optional & individualize after consultant opinion).
- Shorten $2^{nd}$ stage of labour (instrumental delivery).
- Avoid Valsalva maneuver.
- Avoid ergometrine, instead use oxytocin but don't give repeated IV boluses.
- Use PGE 1 if needed, PGF2 is contraindicated (in case of PPH).
- Continuation dose of Oxytocin; 20 units in 100ml N/S at rate of 10ml/hr.
- Active management of third stage of labour.
- Epidural – excellent pain relief.
- Full resuscitation facility in hand.

## Post-delivery care:

- Keep in HDU for vigilant postnatal monitoring.
- Regular monitoring of pulse, B.P, R.R, chest auscultation and watch for vaginal bleeding.
- Monitor for signs of heart failure.
- In high risk cases, consultation with cardiologist before discharge which is 48 hours after normal delivery.
- In post op cases, keep check on fluid balance, encourage early mobilization & breathing. exercises & provide adequate antibiotic cover.
- Advise appropriate contraception according to cardiac disease.
- Thromboprophylaxis should be given after consultant's advice.

- Discuss and plan for effective contraception. (POP, progestin implants and emergency contraception care category 1 can be used in all cardiac patients. Mirena levonorgestrel IUS is also acceptable for LARC)
- Counselling to limit the family size.

## Cardiac surgery during pregnancy:

When medical treatments fail to control symptoms at any period of gestation.
**Risk Time:** (Time Since Delivery)

5 minutes - increase blood volume (cardiac failure).

5 hours – pulmonary edema.

5 days – DVT, Pulmonary embolism.

5 weeks – Bacterial endocarditis.

## Fetal Effects of Drugs:

Diuretics:

- Neonatal jaundice.
- Electrolyte imbalance.
- $H_2O$ depletion.
- Decrease platelets.

Warfarin:

- Achondroplasia.
- Facial & skeletal anomalies
- Mental retardation.
- Haemorrhage.

Propranolol:

- Bradycardia, hypoglycemia.

# EPILEPSY IN PREGNANCY

Prevalence: 0.5 to 1% of pregnant women.
Diagnosis of epilepsy and epileptic seizures should be made by neurologist.
The risk of death is increased ten-fold in pregnant women with epilepsy compared with those without the condition.

## Pre-pregnancy counselling:

- Avoidance of unplanned pregnancy with effective contraception
- Contraception effective and safe for epileptic patients include CU-T, mirena, depo medroxyprogesterone acetate (these are not effected by enzyme inducing anti-epileptic drugs).
- Efficacy of hormonal contraception(pills, patches, vaginal rings, sub dermal implants) decreases with enzyme inducing anticonvulsants.
- Copper IUD is the preferred method of emergency contraception
- Mothers should be advised that risk of congenital abnormalities in the fetus is low if they are not exposed to AEDs in the periconception period.
- Advise 5mg folic acid for 3 months
- Counsel the couple about effects of pregnancy on disease and disease on pregnancy i.e majority of the babies are born normal, increase risk of major and minor malformations due to anti-epileptic drugs
- Consideration should be give to stopping AEDs who have been seizure free for more than 2 years
- Risk of relapse is 20-50%
- Dose of drug should be at lowest and switch to mono therapy.

## Antenatal care:

- Regular antenatal visits.
- Management should be in liason with physician/Neurophysician
- Women on anti-convulsant drugs should continue folic acid 5mg at least till 12 weeks
- Detailed anomaly scan from $18^{+0}$-$20^{+6}$weeks (to identify cardiac /neural tube defects)
- Counsel family regarding condition and immediately report to hospital in case of fit
- Counsel regarding risk factors for seizures such as sleep deprivation and stress, use of alcohol and sedatives, strenuous exercises.
- There is no recommendation in the antenatal period for vitamin K .
- There is also no need for doubling the dose of steroid in mother for prophylaxis of RDS in newborn and the usual dose should be used.
- If patient presents to you with seizures first time in pregnancy rule out other causes of seizures
- In pregnancy there is altered drug metabolism.
- Routine monitoring of AED level in pregnancy is not recommended.
- Serial growth scan to dated SGA.
- No rule of antepartum CTG

## Causes of Seizures in Pregnancy

- Epilepsy
- Eclampsia
- Encephalitis or meningitis
- Space-occupying lesions (e.g tumour, tuberculoma).
- Cerebral vascular accident
- Cerebral malaria or Toxoplasmosis.
- Thrombotic thrombocytopaenic purpura
- Drug and alcohol withdrawal
- Toxic overdose
- Metabolic abnormalities (e.g hypoglycemia)

## Effect of Pregnancy on Epilepsy:

Exacerbation of disease 25%
Improvement 25%
Unchanged 50%

## Things to be considered:

- Patient should be informed that 2/3$^{rd}$ (67%) will not have seizure deterioration in pregnancy
- Risk of seizures are highest in peripartum period
- Teratogenic drugs are phenytoin, phenobarbitone, carbamazepine, sodium valproate, lamotrigine primidone etc. all cross placenta
- Epilepsy doesn't cause fetal distress during short term hypoxia
- No increased risk of miscarriages unless seizure cause abdominal trauma
- Rate of congenital malformation is approximately 6-8% in infant of epileptic mothers using anti-convulsant drugs
- 90% of mothers using these drugs will deliver children with no evidence of congenital malformations

| **Anti-eepileptic Drugs** | **Effects** |
|---|---|
| Sodium valproate | Neural tube defects, adverse impact on long term neurodevelopment of newborn |
| Phenobarbital + phenytoin | Cardiac defects |
| Phenytoin+ Carbamazepine | Facial Cleft(especially cleft palate) |
| Phenytoin | Hydantoin Fetal syndrome, Craniofacial defects, Growth deficiency, Microcephaly, Mental retardation, Digital dysmorphic features, Hemorrhagic disease of newborn, Malignancy of neural crest origin d/t folic acid antagonisms |

## Labour and Delivery:

- Most appropriate place of delivery in tertiary care
- Anti-epileptic drug should be continued, and patient can safely go for vaginal delivery unless there are continuous seizures causing anoxia requires caesarian section
- Risk of epilepsy in labour 1-2%
- Seizures in labour should be terminated as soon as possible to avoid maternal and fetal hypoxia and fetal acidosis. Benzodiazepines(long acting benzodiazepines such as clobazam) are the drugs of choice.
- Recommended method of analgesia for pain relief in labor include TENS(TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION) nitrous oxide and oxygen (Entonox) and regional analgesia
- Diamorphine should be used in preference to pethidine.
- Continuous fetal monitoring in high risk women.
- No contraindication to use any induction agent.
- Inj vit K should be given to baby immediately after birth to prevent hemorrhagic disease of the newborn

## Post-partum care:

- Mother should be well supported to avoid triggers of seizure.
- Breast feeding is safe with anti-convulsant drugs, although best avoided for few hours after taking medication.
- Observe new born for adverse effects of AED exposure in utero.
- Explain to patient about feeding position i.e sitting on floor
- Review drugs and give appropriate contraception
- Decrease the dose in 4weeks to pre-pregnancy (if the AED dose was increased in pregnancy it should be revised within 10 days of delivery to avoid postpartum toxicity)
- Mother should be screened for depressive disorder in puerperium.

## Contra-Indications to breast feeding:

- Poorly controlled seizures
- After breast feeding infant becomes somnolent rapidly, suggestive of drug effect.

## Risk of developing epilepsy in baby:

- If 1 parent has epilepsy risk is 4%
- If one parent and one sibling, then risk is 10%
- If both parents have epilepsy then 15%

## Drug interactions:

**Antagonists**

- Antihistamine
- Antacids
- Diazepam, OCP, POP.

**Agonists**

- Isoniazid
- Chloramphenicol
- Dicoumarol

**Effect on Nutrients:**
Folic acid requirement increase due to
Competitive malabsorption.
Increased hepatic clearance.

**Enzyme inducing AED increase risk of failure of some hormonal contraceptive, COC.**

| Enzyme inducing AED | Non enzyme inducing AED |
|---|---|
| Carbamazepine<br>Phenytoin<br>Phenobarbital<br>Primidone<br>Oxcarbazepine<br>Topiramate<br>Eslicarbazepine | Sodium valproate<br>Levetiracetam<br>Lamotrigine<br>Gabapentin<br>Vigabatrin<br>Tiagabine<br>Pregabalin |

**<u>Effect of Drugs & Disease on Fetus:</u>**

**Antenatal.**

- Increase congenital anomalies.
- Increase malformations.
- Anoxia.
- Hypoxia.
- Increase risk of C/Section d/t bradycardia.

**Immediate.**

- Haemorrhage
- Drug withdrawal effects.
- Trauma if fit occurs
- Increase PNM

**Late.**

- Increase risk of epilepsy
- psychomotor and cognitive impairment.

# JAUNDICE IN PREGNANCY

## Causes:

- **Most common**: Viral hepatitis (usually hepatitis A)
- Gall stones
- Drug reaction, drug/alcohol abuse.

## Pregnancy specific:

- AFLP (acute fatty liver of pregnancy)
- HELLP syndrome
- Obstetrics cholestasis
- Hyperemesis gravidarum

## History:

- H/O fever, abdominal pain and vomiting
- H/O hyperemesis
- Pale stool and dark urine (obstructive jaundice)
- Drug history (erythromycin, isoniazid, OCPs, methyldopa)
- Severe pruritus esp. on palms and soles(obstetric cholestasis)
- H/O blood transfusion
- Close contact with jaundice patients (Hepatitis A & E)
- H/O easy bruisability
- Bleeding from gums and orifices
- H/O raised B.P

## General Physical Examination: Abdominal Examination:

| | |
|---|---|
| • Fever, Anemia<br>• Discoloration of sclera<br>• Spider Naevi<br>• Petechiae<br>• Palmer erythema<br>• Flapping tremors<br>• Ascites & pedal edema<br>• Clubbing | • Check fundal height, lie, presentation etc.<br>• Liver enlargement<br>• Splenomegaly (Enteric fever, hemolysis) |

## Investigations:

- CBC for retics count
- Urine complete examination for urinary urobilinogen
- Coagulation profile PT,APTT,INR (coagulopathy in fulminant hepatic failure due to lack of vitamin K dependent clotting factors)
- USG abdomen
- LFTs
- If HbsAg positive, then screen for antibodies
- Serology for Hepatitis A & E
- ALT + AST increased in hepatitis, HELLP, AFLP
- Obstetric ultrasound
- Conjugated and unconjugated serum bilirubin
- Total bile acid concertation

## Management:

Multidisciplinary management

| Liver disorders | Management |
|---|---|
| 1. HELLP syndrome | • Immediate delivery of baby after stabilization of mother<br>• Control B.P<br>• Prophylaxis for seizures (MgSO4) |
| 2. AFLP | • Supportive hepatic therapy<br>• Delivery in tertiary care unit |
| 3. Viral hepatitis | • Isolation<br>• Medical consultation<br>• Hepatic supportive therapy<br>• Do not use interferons during pregnancy |
| 4. Cholestasis in pregnancy | • Cap. Urso 250-500 mg BD<br>• Tab vit 10mg OD<br>• Plan of delivery according to peak bile acid level, keeping in view fetomaternal condition |

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