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CHAPTER 31

# Antepartum Haemorrhage (APH)

## INTRODUCTION

**Definition.** Any bleeding from or into the genital tract after the period of viability (28 weeks in India, 24 weeks in Western countries) but before the birth of the baby is termed as antepartum haemorrhage (APH). Even though bleeding in a pregnant woman between 20–28 weeks is labelled as threatened miscarriage, practically, such women are managed as APH cases. APH includes hemorrhages in the first and second stages of labour also.

**Incidence.** 3–5% of all pregnancies.

**Etiology.** Various causes include placental bleeding (placenta previa, abruptio placentae, marginal placental bleeding), extraplacental causes and foetal causes (vasa previa) and are shown in Table 31.1.

**Table 31.1: Various Causes of APH**

| Causes of APH | Percentage |
|---|---|
| **I. Maternal causes** | |
| 1. Placental bleeding | 70 |
| (a) Placenta previa | 35 |
| (b) Abruptio placentae | 35 |
| 2. Marginal placental bleeding and unexplained or indeterminate APH | 25 |
| 3. Extra placental causes | |
| (a) Excessive show | |
| (b) Cervical erosion and ectropion | |
| (c) Local infections of cervix and vagina | |
| (d) Cervical and vaginal trauma | |
| (e) Cervical polyp | |
| (f) CIN and carcinoma cervix | |
| (g) Vulvovaginal varicosities | |
| (h) Post coital bleeding | |
| (i) Scar dehiscence and uterine rupture | |
| (j) Medical causes (Factors VIII and IX deficiency) | |
| 4. Miscellaneous placental and umbilical cord anomalies | |
| (a) Circumvallate placenta | |
| (b) Velamentous insertion of the umbilical cord | |
| (c) Succenturiate lobe | |
| (d) Congenital arterio-venous malformation | |
| (e) Aberrent blood vessel | |
| **II. Foetal causes** | |
| (a) Vasa previa | < 1 |

## PLACENTA PREVIA

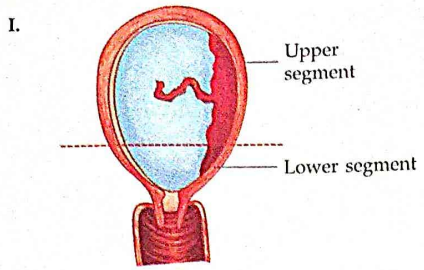
**Definition.** *Placenta previa is defined as a placenta that is partially or wholly situated in the lower uterine segment* **over or very near to the internal cervical os** (< 2 cm from internal os).

**Incidence.** It 1 in 300 to 1 in 400 deliveries (0.3%) in different populations. It accounts for one-third cases of APH.

### Etiology

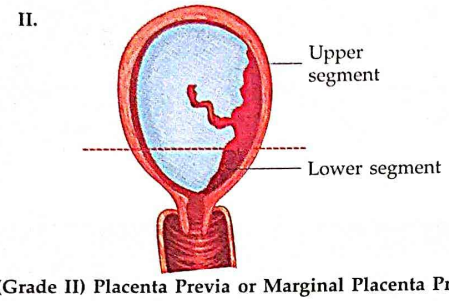
The exact cause of placenta previa is not known. Various factors and accepted theories are as follows:

1. **Age.** It is more common in old age.
2. **Parity.** It is more common in multiparous women
3. **'Dropping down' theory** of fertilized ovum and its implantation in the lower uterine segment instead of the upper segment. This could be the result of poor decidual reaction in the upper segment. It explains central placenta previa.
4. **Defective decidua basalis** as seen in (*i*) elderly patients (*ii*) grand multipara, (*iii*) history of manual removal of placenta, (*iv*) endometrial ablation, (*v*) previous dilatation and curettage, (*vi*) MTP or spontaneous abortion



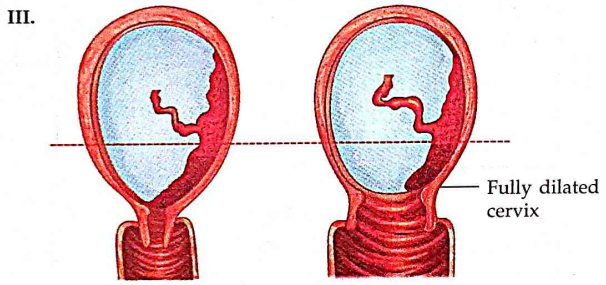

**Type 1 (Grade I) Placenta Previa or Low Lying Placenta**

Placenta implanted in the lower uterine segment with the placental edge not reaching the internal os but is in close proximity to it. It can be attached to (a) anterior wall; (b) posterior wall.



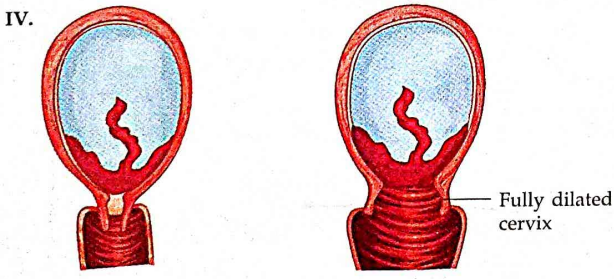

**Type 2 (Grade II) Placenta Previa or Marginal Placenta Previa**

The edge of the placenta is at the margin of the internal os but does not cover it. It can be attached to (a) anterior wall; (b) posterior wall.




**Type 3 (Grade III) Placenta Previa**

Or **Partial/Incomplete Central Placenta Previa.** Placenta covers the os fully or partly but does not cover os on full dilatation of cervix




**Type 4 (Grade IV) Placenta Previa**

**Or Total/Central Placenta Previa.** Placenta covers the os even on full dilatation of cervix

**Fig. 31.1:** Types of placenta previa (Browne's classification)

5. **Hyperplacentosis.** Bigger placenta in multiple pregnancy, anaemia and Rh isoimmunization may encroach on lower segment.
6. **Previous lower segment cesarean delivery.** Incidence increases with each successive cesarean delivery being 0.3% without any cesarean while it is 0.7%, 1.8%, 4% and 10% with previous 2, 3, 4 and 5 cesareans respectively *(2.5 times rise each time).*
7. **Foetal malpresentations** may be the cause or effect.
8. **Uterine anomalies**
9. **Persistence of chorionic** laevae which encroach on the lower uterine segment.
10. **Placental and cord abnormalities.** Circumvallate placenta, battledore placenta, membranous placenta, succenturiate lobe, multipartite placenta, velamentous insertion of the cord
11. **Smoking and drug abuse (cocaine).** Smoking increases the risk of placenta previa (RCOG 2018). Carbon monoxide production during smoking leads to tissue hypoxia with compensatory placental hypertrophy due to possible result of inflammatory or atrophic changes.
12. **Certain ethnic** groups, e.g. Asian women
13. **Maternal Serum AFP.** Women with otherwise unexplained increased screening levels of MSAFP are at greater risk for placenta previa. MSAFP levels of ≥ 2.0 mom at 16 weeks screening were at increased risk for late-pregnancy bleeding and preterm birth.
14. Fibroid uterus increases risk of placenta previa.
15. Assisted reproductive techniques (ART) are associated with increased risk of placenta previa (RCOG 2018).

## Cause of Haemorrhage (bleeding)

The inelastic placenta gets sheared off from the wall of the progressively thinning and dilating lower uterine segment during the last trimester of pregnancy and in labour. **Bleeding occurs from the open maternal sinuses.** Being a physiological phenomenon, the bleeding is called **inevitable.** Hence, it is mostly unprovoked, but sometimes there is history of recent coitus, vaginal examination, external cephalic version or high rupture of membranes.

The blood lost is almost always maternal in origin, but in traumatic separation of the placenta, foetal blood may escape from torn villi

**The mechanisms of spontaneous stopping of bleeding are:**

1. Uterine muscles acting as ligatures.
2. Thrombosis of the open maternal sinuses.
3. Mechanical pressure by the presenting part.
4. Placental infarction

## Classification / Grades

Foetal medicine workshop of National institute of Health has recommended following classification:

1. Placenta previa: The internal os is partially or completely covered by placenta. It can be further total or partial placenta previa.
2. Low-lying placenta: Implantation in lower segment with placental edge lying within 2 cm of perimeter around os but does not cover the internal os.

In olden classification were four grades or types of placenta previa as shown in Fig. 31.1. Type 2 posterior, type 3 and type 4 are called **major (degree) placenta previa** while types 1 and 2 A are called **minor degree of placenta previa.**

### Dangerous placenta previa (Type 2 posterior placenta previa)

Placenta is situated on the posterior wall of the lower uterine segment reaching the internal os.

**Effects**

1. Placenta overlies the sacral promontory, thus reducing the antero-posterior diameter of pelvic inlet by about 2.5 cm (thickness of the placenta).
2. Prevents engagement of the presenting part.
3. Cord compression and cord prolapse can occur causing foetal jeopardy.
4. More chances of foetal hypoxia and mortality due to excessive compression of the placenta if vaginal delivery is allowed.
5. Interferes with its pressure effect on the separated placenta to stop haemorrhage.
6. Type 2 posterior placenta previa is managed like major degree placenta previa (see below) and is an indication of cesarean delivery.

## Clinical Features of placenta previa

**Symptoms**

The main and often the only symptom of placenta previa is vaginal bleeding.

1. Sudden onset of painless, profuse, recurrent and apparently causeless (without trauma, coitus, etc.) bleeding without onset of labour is characteristic of placenta previa. (4P's: Painless and profuse bleeding in placenta previa).
2. The first bleeding called 'warning' haemorrhage is often minor.

Bleeding occurs before 38 weeks and is more severe in major degrees of placenta previa. About 80% of all women with placenta previa bleed before the onset of labour. In olden days, placenta previa was called **unavoidable or inevitable antepartum haemorrhage** as placenta situated in the developing and stretching lower uterine segment ultimately causes bleeding during pregnancy or labour. Chances of bleeding are more if placenta is located partly in upper and partly in lower segment **(type 2 and type 3)** due to differential contraction of upper and lower segments. But when bleeding occurs it is much more severe in **central placenta previa.**

**Signs**

*General Examination*

1. Pallor is proportionate to visible blood loss.
2. The patient may or may not be in shock depending on the amount of bleeding.

*Abdominal examination*

1. The abdomen is **soft.**
2. The uterus is relaxed and **non-tender** corresponding to the period of amenorrhoea.
3. **Malpresentations like breech, transverse or unstable lie are more common in placenta previa (35% cases).**
4. Foetal parts are easily felt and foetal heart is usually normal.
5. The presenting part is floating. **A deeply engaged presenting part usually rules out placenta previa** or points to only minor placenta previa.
6. Foetal heart sound and placental soufflé are well auscultated, unless there is a major separation of the placenta with the patient in exsanguinated condition. **Foetal bradycardia on pressing the head down into the pelvis and its prompt recovery on release of the pressure is suggestive of low lying placenta specially of posterior type (Stallworthy's sign).** But this sign is not very reliable as foetal bradycardia can be due to foetal head compression even in a normal pregnancy.

**Vulval inspection**

Inspection is done to note whether the bleeding is still occurring and the amount and character of blood loss. It is usually fresh (bright red) as the blood quickly trickles down through the cervix from the nearby placenta.

## Diagnosis

### I. Clinical

a. High index of suspicion should be kept if there is foetal malpresentation or free-floating presenting part. Pervaginal bleeding in late pregnancy or presence of predisposing factors arouse suspicion of placenta previa.

b. The mid pregnancy routine anomaly scan after 16 weeks of pregnancy should include placental localisation to identify plancenta previa (placenta lies directly over the internal os) or low lying placenta, i.e., placental edge is less than 20 mm from the internal os in transabdominal or transvaginal scan (RCOG 2018). In all such cases (placenta previa or low lying placenta at level II scan), a follow-up ultrasound examination including a TVS is recommended at 32 weeks of gestation to diagnose persistent placenta previa or low lying placenta (RCOG 2018).

c. A gentle speculum examination performed 24–48 hours after active bleeding has stopped differentiates the condition from other local causes in the vagina and the cervix, e.g. polyp, etc.

d. **Vaginal examination.** A pervaginal examination is contraindicated in a case of APH, unless placenta previa has been ruled out because it may result in serious life-threatening haemorrhage. However, a double set up vaginal examination may be performed (see below).

### II. Investigations

**1. Ultrasonography (USG)** (Fig. 31.2)

Ultrasound is the modality of choice because it is safe, precise and simple with accuracy of 98% after 30 weeks of gestation. Maternal obesity and posterior placenta may obscure the placental view.

*Route*

1. Transabdominal (TAS) ultrasound is the first test performed. It can give false positive results due to full bladder and myometrial contraction.
2. Transvaginal ultrasound (TVS) is better as there is less

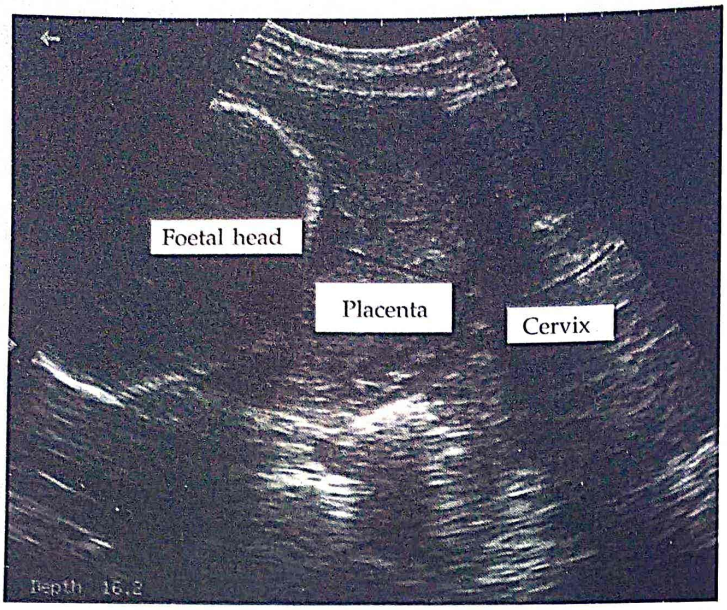



**Fig. 31.2:** Ultrasound showing central placenta previa covering internal os

distortion of the anatomy of the lower uterine segment and cervix. Full bladder is not required for it.

3. Transperineal or translabial ultrasound can also be used. Three-dimensional scan and Colour Doppler flow can also be used.

**Diagnostic criteria.** Placenta within 2 cm of internal os at term is labeled placenta previa. *At TVS the probe being in posterior fornix is not in axis of placenta so there is very less chance of provoking a bleeding However, on internal examination, the fingers come in line with placental axis, increasing chances of provoking bleeding*. Poor imaging is seen with maternal obesity, and posterior placenta previa . Posterior placenta is poorly visualised because of the acoustic shadow from the foetal presenting part and because there is no posterior anatomical landmark like the uterovesical fold anteriorly in relation to which the placenta is defined as previa.

Ultrasound also gives information about presentation and any foetal congenital malformations.

**Placental Migration (King, 1973).** Placental migration is also called **trophotropism**. Up to 5% women have ultrasound evidence of low lying placenta at 16–18 weeks, but only 0.5% have placenta previa at term. It is called placental migration and is due to differential growth of lower and upper segments as pregnancy **progresses**. During third trimester there is tenfold growth of lower uterine segment as compared with the placenta. This apparent movement may be due to:

(a) Development of lower uterine segment (LUS) in later part of pregnancy.

(b) Placenta grows preferentially toward better vascularized fundus (trophotropism) and the placenta over less vascular cervix atrophies.

Placental migration is less likely, if

1. Placenta is posterior
2. Thick (> 1 cm)
3. Lower edge < 2 cm from os
4. History of previous cesarean delivery

**2. Magnetic Resonance Imaging (MRI)** (Fig. 31.3)

MRI is noninvasive, safe but expensive method of diagnosis. Gadolinium enhanced MRI is helpful to diagnose morbidly adherent placenta (See Chapter 25 for details).

Other investigations like radiography, radioactive isotopes $I^{132}$ or $I^{131}$, technetium$^{99}$ study and arteriography were used in the past when ultrasound was not available, but are not used nowadays.

**3. Basic Investigations for Management**

1. Complete hemogram
2. Blood group, Rh factor and cross match
3. VDRL test
4. Urine test
5. Coombs' test, if Rh negative
6. Kleihauer-Betke test, if available

### III. During Cesarean Delivery

Placental implantation and degree on the lower segment can be confirmed during cesarean section.

### IV. After Vaginal Delivery

Examination of the placenta may show (a) A tongue-shaped segment of placenta projecting beyond the main placenta with degeneration. (b) Rent on the membranes on the margin of placenta. (c) Abnormal attachment of the cord like marginal or membranous.

## Differential diagnosis

Bleeding from placenta previa can be confused with any of the other causes of APH shown in Table 31.1. The most common differential diagnosis is abruptio placentae. The differentiating features between the two conditions (placenta previa and abruptio placentae) are shown in Table 31.2.

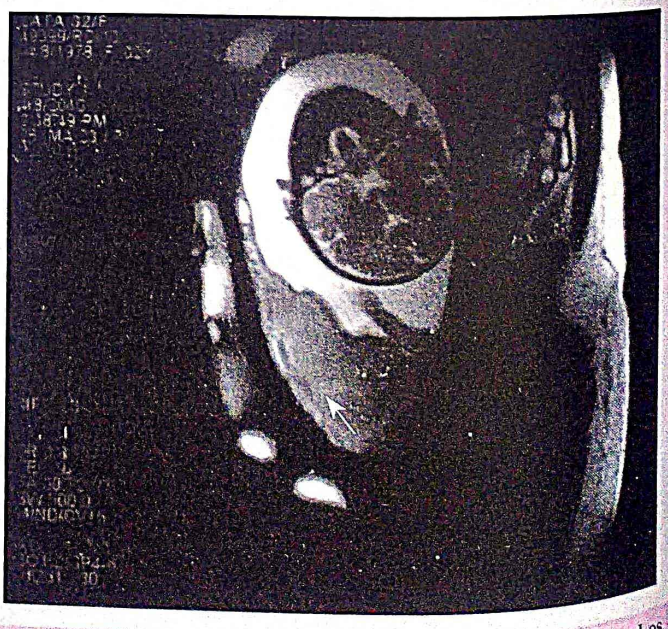


**Fig. 31.3:** MRI showing central placenta previa covering internal os

**Table 31.2: Differences between Placenta Previa and Abruptio Placentae**

| Characteristics | Placenta Previa | Abruptio Placentae |
|---|---|---|
| 1. Definition | It is defined as a placenta that is partially or wholly situated in the lower uterine segment. | It is defined as an antepartum haemorrhage from premature separation of a normally situated placenta in the upper segment |
| 2. Incidence | 1 in 300 deliveries. | 1 in 200 deliveries. |
| 3. Etiology | Not known exactly. It is more common in elderly and multiparous women with hyperplacentosis, previous cesarean delivery and placental and cord anomalies. | Not known exactly. It is common in hypertensive disorders of pregnancy, trauma, thrombophilias, nutritional deficiencies (folate deficiency), sudden decompression of uterus and with past history of abruption. |
| 4. History taking | | |
| Vaginal bleeding | Vaginal bleeding is sudden, painless, profuse, recurrent and apparently causeless (no trauma) and without onset of labour. Blood is bright red and is always revealed in nature. | Vaginal bleeding is usually painful, continuous and is often related to some cause like hypertensive disorders of pregnancy, trauma, etc. Blood is usually dark colored and can be revealed, concealed or mixed in nature. |
| Pain abdomen | Usually absent | Usually present, acute in severe cases |
| Hypertensive disorders of pregnancy | Usually absent | May be present in about 35% cases. |
| 5. General Physical Examination | There may be pallor, sweating, hypotension tachycardia which are proportionate to the amount of revealed blood loss. Hypertension is usually absent. | Pallor, tachycardia, hypotension and shock may be more (out of proportion) in concealed and mixed abruption. There may be hypertension in some cases. |
| Heart and chest examination | Usually normal or tachycardia | Tachycardia is usually present. |
| 6. Abdominal Examination | | |
| Fundal height | Usually as per gestation | Fundal height may be more than gestation due to concealed or mixed haemorrhage. |
| Consistency | Uterus is usually soft and relaxed. | Uterus is usually tense, tender and hard especially in concealed and mixed abruption. |
| Foetal parts | They are easily felt. | They are not easily palpable. |
| Foetal heart sound | It is usually normal and regular. | It may be absent due to placental separation, especially in concealed or mixed abruption. |
| Malpresentations | Malpresentations like breech, transverse lie or oblique lie are more common as placenta does not let head descend down in the pelvis. Presenting part is usually high up due to placenta being there. | Malpresentations are usually absent. Presenting part (usually head) tends to get engaged early. |
| 7. Vaginal examination | It is contraindicated in placenta previa due to risk of brisk haemorrhage. If performed in operation theatre with arranged blood, placenta is felt in the lower segment. | Placenta is not felt in the lower uterine segment within reach of the finger. Instead, soft and friable blood clots are palpable. |
| 8. Investigations | | |
| Ultrasound examination | It demonstrates localization of placenta in the lower uterine segment. It also diagnoses foetal presentation, and foetal heart condition. | Placenta is demonstrated in the upper uterine segment. There is separation of placenta. There may be collection of blood under the placenta in concealed abruption. It also reveals foetal condition. |
| Magnetic Resonance Imaging (MRI) | It is a better diagnostic modality but is more expensive and is mainly used to rule out morbidly adherent placenta. | MRI can quantify the separation of placenta and amount of any concealed abruption. |
| 9. Treatment | | |
| Resuscitation | It is with fluids and blood. | Resuscitation with fluids and blood |
| Mode of delivery | Elective cesarean delivery is the treatment of choice for severe degree placenta previa. | Vaginal delivery after amniotomy and oxytocin is performed. Cesarean delivery may be indicated for foetal distress. |

Other important conditions in differential diagnosis like vasa previa, indeterminate APH and local cervical and vaginal lesions are described in details later.

## Complications

### Maternal

#### During Antenatal Period

1. ***Vaginal bleeding***:. Antepartum haemorrhage with different severity of shock is the most important and inevitable complication of placenta previa. First bout of vaginal bleeding is usually a smaller bleed (warning haemorrhage) but a severe and potentially life threatening haemorrhage can be provoked by a vaginal examination done outside the hospital setting. **Women with placenta previa with raised maternal serum alpha-fetoprotein of more than 2.0 multiple of median (MOM) are at increased risk of bleeding.**
2. ***Malpresentations***. Placenta situated in the lower uterine segment prevents engagement of foetal head which deviates to one side with increased incidence of breech presentation and transverse, oblique and unstable lies. One should always do ultrasound in presence of a malpresentation at term in a primigravida.
3. Preterm labour, both spontaneous and iatrogenic, is more common.
4. Long hospital stay
5. Anaemia
6. Rhesus sensitization in Rh negative women.
7. Hemorrhagic shock with hypotension
8. Adult respiratory distress syndrome
9. Disseminated intravascular coagulation (rare)
10. Acute renal failure (rare)
11. Blood transfusion and its associated complications

#### During Labour

1. Premature rupture of membranes
2. Cord prolapse
3. Intrapartum haemorrhage
4. Slow dilatation of cervix
5. More operative interference
6. Increased incidence of postpartum haemorrhage due to imperfect contraction and retraction of the lower uterine segment, large surface area of placenta with atonic uterus, pre-existing anaemia, association of morbidly adherent placenta and tears in the cervix and uterus due to excess softness and vascularity.
7. Retained placenta is more common due to larger surface area and morbidly adherent placenta necessitating manual removal of placenta.
8. Abruption of placenta can co-exist with placenta previa.
9. Hysterectomy may be needed.
10. Air embolism

#### During Puerperium

1. Secondary postpartum haemorrhage
2. Puerperal sepsis due to increased need of operative interference, pre-existing anaemia and placental site being near to vagina.
3. Non-involution and subinvolution of uterus due to imperfect retraction of uterus, anesthesia and more operative interference.
4. Venous thromboembolism is more common (0.5%).

#### Maternal Mortality

There has been a substantial reduction of maternal deaths in placenta previa throughout the world due to the following reasons.

1. Early diagnosis (diagnosis can even be made prior to bleeding)
2. Avoiding internal examination outside the hospital
3. Free availability of blood transfusion facilities
4. Effective antibiotics
5. Liberal use of cesarean delivery with expert anesthetists
6. Skill and judgement for management.

In developing countries, maternal mortality from placenta previa ranges from 1% to 5% due to inadequate antenatal care, delay in referral and road and transport difficulties. **The causes of morbidity and mortality are haemorrhage, shock and operative delivery.**

## Foetal Complications

The reduction of foetal deaths is mainly due to judicious use of expectant treatment (thereby reducing the loss from prematurity), liberal use of cesarean delivery, which greatly lessens the loss from anoxia and improvement in the neonatal care unit.

1. **Perinatal mortality ranges from 7–25% and is three times higher than the general population.**
   The causes of death are:
   (a) Prematurity
   (b) Asphyxia due to separation of placenta
   (c) Congenital malformations
   (d) Cord accidents
   (e) Maternal hypovolemia and shock
2. **Prematurity.** Spontaneous or iatrogenic
3. **Low birth weight babies** due to preterm labour or chronic placental insufficiency due to repeated small bouts of haemorrhage.
4. Foetal hypoxia can occur due to placental separation and placental and cord compression.
5. **Foetal injuries** can occur due to operative delivery.
6. **Congenital malformations** like spina bifida are three times more common in placenta previa.
7. **Foetal malpresentations** are more common.
8. **Foetal hypovolemia** is more common.

## Management

### Prevention

Placenta previa is not usually preventable as in most cases the etiology is known. The following modalities are beneficial.

1. Universal institutional antenatal care of all women to improve their general health and to correct anaemia.
2. Family planning and limitation of births reduce the incidence of placenta previa.
3. Reducing the rising rate of cesarean deliveries as previous cesarean delivery predisposes to placenta previa.

4. Universal targetted scan of all pregnant women at 18 weeks can diagnose low lying placenta which can later become placenta previa.

### Treatment

The treatment of placenta previa is shown in Flow Chart 31.1.

#### At Home and Transfer to Hospital

After quick history, general physical examination and gentle abdomen examination **(but no vaginal examination)**, once antepartum haemorrhage is visible, patient is shifted in an ambulance to a suitable hospital where there are adequate facilities for blood transfusion, 24 hour cesarean delivery and nursery care. A health care personnel should be with the patient. An intravenous drip is started with Ringer lactate or dextrose saline solution and is continued in the ambulance. Preferably relatives fit to donate blood should also go with the patient. The woman should be lying down during transfer.

#### Treatment at Hospital

***All patients of APH, irrespective of the amount of blood loss, should be admitted. The blood loss should be considered as due to placenta previa unless proved otherwise.***

The bleeding may recur anytime and is unpredictable.

1. In the hospital a quick note is made about the general condition of the patient and the amount of blood loss by inspecting the soiling of her clothes and bed sheets and the blood stains on her thighs and legs.
2. A detailed history is taken from her or reliable attendant about the events before bleeding, any associated pain, backache, any previous bleeding, feeling of foetal movements and date of last menstruation period.
3. A brief clinical examination is performed about general physical condition by noting pulse, blood pressure, respiration, anaemia and heart and chest auscultation.
4. *A light and gentle abdominal palpation is done for uterine* tone, tenderness, contractions, foetal presentation, and foetal heart is auscultated.
5. Blood sample is taken for hemogram, group and crossmatching and 2–4 units of blood depending upon blood loss are arranged.
6. Intravenous access is secured using two wide bore cannulae and a crystalloid (saline or dextrose saline) or colloid (hemaccel) is started.
7. The senior member of team (consultant) should be informed about such a patient.
8. If available, a bedside ultrasound of abdomen is performed to localise placenta, foetal presentation and foetal heart.
9. Some sedation and reassurance are given to calm her.
10. Further management depends on the gestational period, maternal and foetal conditions and whether bleeding has stopped or not.
11. **Vaginal examination is absolutely contraindicated.**

#### Expectant Management (Macafee and Johnson regimen) [Macafee (UK) and Johnson (USA) 1945]

The aim is to prolong pregnancy till the fetus is reasonably mature and if possible till term without compromising maternal condition in a center with round-the-clock facilities for blood transfusion and cesarean delivery. *It should be clear that expectant treatment is meant only for achieving foetal maturity and benefits the fetus by putting some risks on the mother.*

##### *Indications for Expectant Management*

1. Good general condition of the mother (hemoglobin > 10 g/dL).
2. Gestation is less than 37 weeks.
3. Absence of active vaginal bleeding
4. Patient not in labour.
5. Good foetal condition as assessed by ultrasound and non-stress test (NST). There are no major congenital malformations.

##### Conduct of Expectant Treatment

Expectant treatment is done in hospital. Rarely it may be allowed at home if patient lives nearby with 24 hours transport facility and someone to look after her.

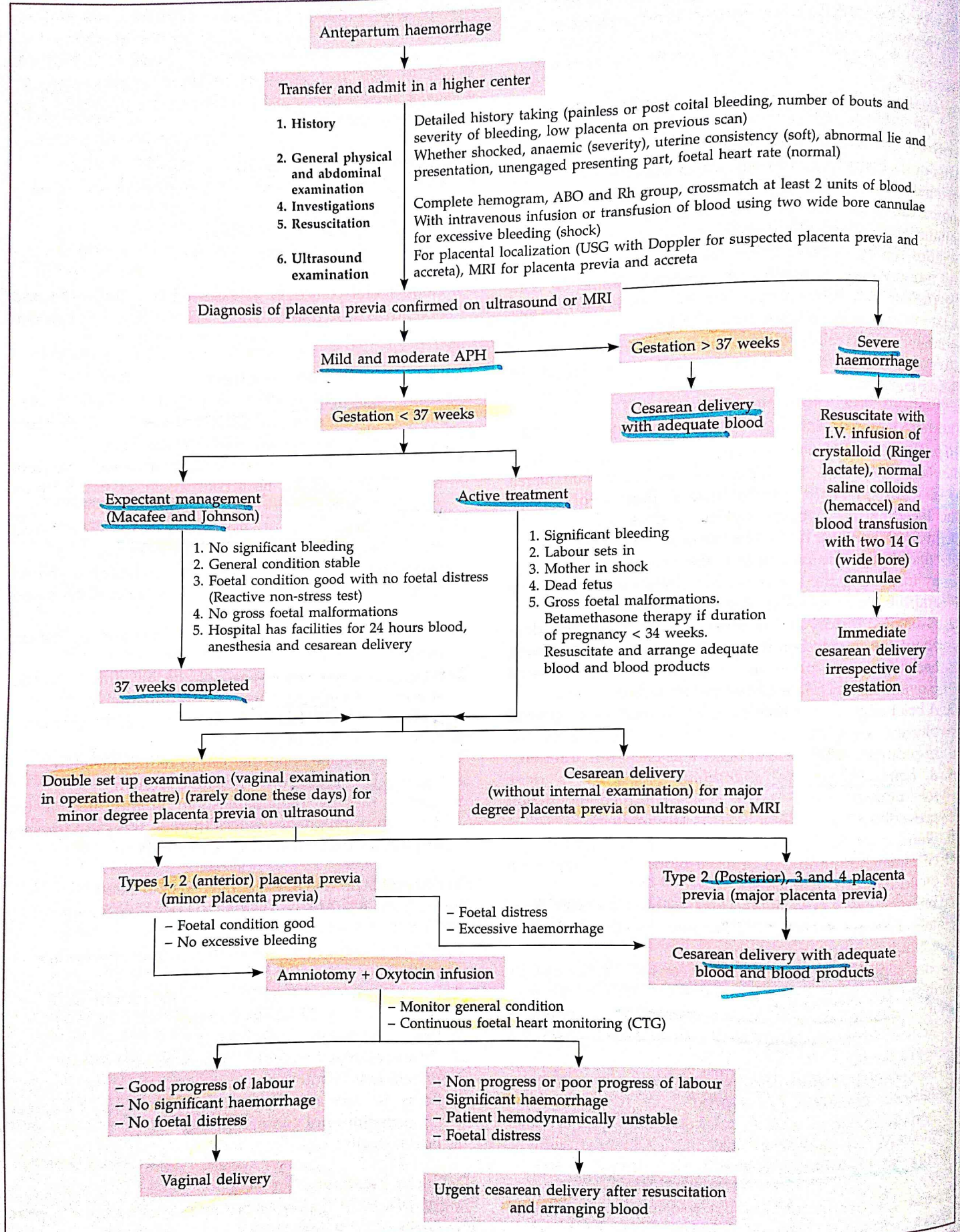
1. Bed rest with bathroom facilities (not to lock the door when alone). Should be accompanied 24 hours by an attendant. Daily monitoring of vitals and foetal well-being.
2. Regular inspection of the vulval pads and foetal monitoring with USG at interval of 2–3 weeks.
3. Investigations like hemoglobin estimation, blood grouping and urine for proteins are done. Keep blood and blood products arranged.
4. Iron, folate and calcium supplementation are given. Blood transfusion is given for blood loss.
5. After 2–3 days of stoppage of the bleeding, a gentle speculum (Sim's or Cusco's) examination is done to exclude local cervical and vaginal lesions for bleeding.
6. Tocolytics should be avoided.
7. Cervical cerclage is not helpful to reduce bleeding.
8. Inj Anti D is given to Rh negative women.
9. Steroid therapy (betamethasone) is indicated if the duration of pregnancy is less than 34 weeks to prevent respiratory distress syndrome in newborn.

##### Contraindications for Expectant Management (Indications for Active Treatment/Termination of Expectant Management)

1. Maternal condition deteriorates despite appropriate treatment.
2. Persistent haemorrhage or excessive haemorrhage
3. Gestational age 37 weeks or more
4. Foetal distress in a viable fetus.
5. Estimated foetal weight 2500 g or more
6. Patient is in labour.
7. Fetus is very small or has lethal major congenital abnormalities not compatible with life.
8. Foetal death

#### Definitive Treatment

Active treatment is performed once fetus becomes mature (37 weeks) or when there is any indication for active treatment.




**Flow Chart 31.1:** Management of placenta previa

**Route of Delivery.** The route of delivery is decided based on type of **placenta previa.**

**Types 1, 2 anterior placenta previa.** Induction of labour with ARM (amniotomy) and oxytocin. Cesarean delivery done only if there is severe bleeding or foetal distress or any other obstetric indication. Rest of the patients deliver vaginally.

**Double set-up vaginal examination**

The vaginal examination is normally contraindicated in placenta previa.

Indications are:

1. Inconclusive USG report
2. USG reveals type I and type II A placenta previa.
3. USG facilities not available.

It is performed in the operation theatre keeping everything ready for cesarean delivery. Anesthetist and nursing staff should be there ready for emergency cesarean delivery. Two scrubbed team should be there; one for emergency cesarean delivery and other for vaginal examination. Vaginal examination conclusively confirms the clinical diagnosis and degree of placenta previa.

PROCEDURE

After taking consent the patient is put on the operation table and an intravenous drip is started using a wide bore cannula. At least 2 units of blood for transfusion are arranged. It is better to put the patient under general anesthesia though examination can be done without anesthesia too. All these precautions are taken so that a quick surgery can be performed if excessive bleeding starts following internal examination. Parts are cleaned and draped. Bladder is catheterized, a Sims speculum is then introduced under all aseptic precautions to inspect the cervix. One finger (index) is gently introduced into the vagina to feel for boggy mass intervening between the presenting part and the finger in the fornices. If the head is felt clearly through all the fornices, then only one finger is gently introduced through the cervical canal. **The placenta feels firm, tough and gritty while blood clot feels soft and friable.** Examination should stop if placenta is felt. The lower segment is explored by sweeping the finger all around the internal os before declaring that no placental tissue is felt. In that case rupture of the membranes may be done to induce labour depending upon the type. There should be two teams ready simultaneously, one vaginal and other abdominal.

CONTRAINDICATIONS OF VAGINAL EXAMINATION

1. Patient in poor general condition due to bleeding.
2. Major degree of placenta previa confirmed by ultrasonography.
3. Conditions like malpresentations, previous cesarean delivery and contracted pelvis which are themselves indications for cesarean delivery.

**Amniotomy.** After a decision for vaginal delivery is made, the labour is induced by low rupture of the membranes using long Kocher's forceps in lesser degree of placenta previa (Type 1 and Type 2 anterior). Cord prolapse is excluded by vaginal examination. Usually labour starts with amniotomy helping in descent of the foetal head which helps in control of bleeding by pressure on the separated placenta. Oxytocin drip can be started, if needed. If labour does not start or bleeding continues cesarean delivery should be performed.

PRECAUTIONS DURING VAGINAL DELIVERY

1. Adequate hydration and blood transfusion if needed through intravenous line.
2. Active management of third stage is done by using intramuscular oxytocin.
3. Proper examination of the cervix and vagina for any tears should be done soon after delivery.
4. Baby's cord blood hemoglobin level is checked.

**Cesarean delivery**

**Cesarean delivery without vaginal examination is the treatment of choice for major degree placenta previa** as the exact location of the placenta can now be determined in almost all cases with the use of ultrasound. It helps not only to reduce the maternal risk but also to improve the foetal outcome.

*The indications of cesarean delivery*

1. Major degree of placenta previa (Type 2 posterior, Type 3 and Type 4) irrespective of foetal condition for maternal sake.
2. Minor degree of placenta previa with excessive bleeding inspite of amniotomy with foetal distress.
3. Presence of other complicating factors with minor degrees of placenta previa where vaginal delivery is unsafe.

Elective cesarean section for placenta previa must be done during daytime so that there are senior doctors of the obstetrics, pediatrics and anesthesia departments either directly performing or supervising the procedures, e.g. cesarean section, resuscitation of the neonate or administering anesthesia. This will help prevent maternal morbidity and mortality as well as foetal morbidity and mortality. Keeping adequate blood in hand is life saving especially in complicated cases like placenta accreta, percreta, etc., or cases with previous history of cesarean delivery in an emergency situation.

However, if patient is in shock and bleeding continues, cesarean section is to be performed in emergency alongwith concomitant resuscitation by starting two intravenous drips using wide bore cannulae and arranging 4 units of blood. One has to be careful in **Jehova witness** women, a religious sect who do not accept blood transfusion. Use of erythropoietin and parental iron or autologous blood transfusion may be used in them. General anesthesia is usually preferred.

Skin incision can be infraumbilical vertical incision which is fast and gives better visualization and also allows classical cesarean delivery if needed. However, transverse incision using Cohen's technique (see Chapter 42[v] for details) can be very fast and gives adequate visualization.

Uterine incision can be converted to an inverted T-, J- or U-shaped incision if lower segment is very vascular or non-existent.

### Type of operation

**Lower uterine segment cesarean section (delivery).** The advantages of lower uterine segment approach are:

1. Familiar technique, as most obstetricians use it
2. The bleeding sinuses at the placental site can be better managed under direct vision.
3. Morbidly adherent placenta, if found, can also be managed effectively.

**The disadvantages are**

1. Dilated vessels on the anterior lower uterine segment especially in anterior placenta previa may bleed excessively when cut.
2. In anterior placenta previa, the placenta either has to be cut or separated to deliver the baby.
3. There is risk of severe foetal anaemia due to foetal exsanguination.
4. The edges of the cut margins are very vascular and friable making suturing very difficult.

### Classical Cesarean Delivery

The advantages of classical cesarean section are:

1. The operation can be performed very fast (However, nowadays the obstetricians are not familiar with it).
2. Fetus can be quickly extracted without disturbing the placenta and without exsanguination.

*Disadvantages*

1. It may be difficult to control haemorrhage from the lower segment which is not visualized properly as it is below the incision.
2. Classical cesarean operation is associated with increased complications.

Most obstetricians these days perform lower uterine cesarean section for placenta previa.

### Difficulties Encountered During Cesarean Section in Placenta Previa

1. The lower segment is not well formed before term.
2. May require lower segment vertical incision.
3. If the placenta lies anteriorly, there can be large dilated vessels. In such a case, ligate them with catgut or vicryl suture and then give a nick in the uterus or feel the lateral margin of placenta and then deliver baby or cut through the placenta to deliver the baby.
4. If there is placenta accreta, hysterectomy or internal iliac artery ligation may be required.
5. If the placental bed bleeds excessively, apply hotpacks, mattress sutures or gel foam.

### Practical Approach to Lower Segment Cesarean and Difficulties Encountered

1. Infraumbilical vertical incision is given though Cohen's transverse incision. It can be quite fast.
2. The dilated veins on the anterior uterine wall are ligated and cut in between while giving the transverse incision.
3. To manage the anterior placenta previa coming in the incision line, a cleavage plane is developed with the fingers between the uterus and the placenta until the membranes are palpable and can be ruptured to deliver the baby. Alternatively, the placenta can be cut to enter into the amniotic sac to extract the fetus. Baby is delivered and the cord is clamped quickly to prevent foetal exsanguination.
4. After the delivery of the placenta, the lower segment is carefully inspected for any bleeding points which are stitched with 1–0 vicryl or chromic catgut sutures.
5. For postpartum haemorrhage due to atonic uterus, quick suturing of the uterine incision, oxytocics (oxytocin drip, ergometrine, prostaglandin $F_2\alpha$) and massaging the uterus with hotpacks usually brings the uterine tone back (see Chapter 25 for details).
6. If hemostasis by sutures fails, tight intrauterine packing with povidine iodine soaked gauze is done to give firm pressure on the oozing area. The edge of gauze is brought through the vagina and is removed vaginally after 24 hours.
7. Isthmic cervical apposition: a suture is passed through the lower flap of the uterine incision 2 cm medial to the lateral border from anterior to posterior and then posterior to anterior , suture is brought back 1 cm medial to the first and tied anteriorly. Same procedure is repeated on the other side. The cervial canal is kept patent while tightening the sutures. This reduces the bleeding in some cases of placenta previa.
8. Setting a balloon device which can be inflated to provide uterine compression to be removed vaginally later can also arrest haemorrhage.
9. B-Lynch sutures can be applied for bleeding. Cho's sutures can be used by placing circular interrupted 1–0 chromic catgut sutures around the lower segment above and below the transverse incision to control haemorrhage (see Chapters 25 and 42[v] for details).
10. Uterine or internal iliac artery ligation can also be tried.
11. Extra oxytocics including prostaglandin $F_2$ alpha, intramyometrial vasopressin can be tried.
12. Pelvic artery embolization has also been used successfully to control haemorrhage.
13. If all these measures fail and in cases of placenta accreta, an early recourse to hysterectomy should be the safest strategy.

The woman should be carefully observed, monitored and cared for in the post operative period for any bleeding.

## Prognosis

Use of expectant management, more liberal use of blood and blood products, avoiding vaginal examination and liberal use of cesarean section (delivery) has significantly improved the maternal and perinatal outcome in placenta previa.

# ABRUPTIO PLACENTAE
## (Syn. Accidental Haemorrhage, Ablatio Placentae, Premature Separation of the Normally Implanted Placenta)

### Definition

*Abruptio placentae is antepartum haemorrhage from premature separation of normally situated placenta in the upper uterine segment.* It is an important cause of maternal mortality (2–5%) and perinatal mortality (22–40%).

Abruptio placentae is the most appropriate term now used in place of accidental haemorrhage. It occurs in 1 in 200 deliveries.

### Etiology

Exact etiology is unknown in majority of cases. Risk factors are:

1. Past history of abruption increases the relative risk of abruption to 10–188 times.
2. Advancing maternal age and parity.
3. Ethnicity more in blacks and white less in Asian women.
4. Single umbilical artery increases risk.
5. Hypertensive disorders of pregnancy like pre-eclampsia, essential hypertension, renal hypertension can all cause abruption and are responsible for 20–50% cases of abruptio placentae. Vasospasm of uteroplacental blood vessels causes anoxic endothelial damage with extravasation of blood in the decidua basalis with abruption.
6. **Trauma.** Fall, blunt external trauma and road traffic accident
7. **Vascular accident.** Supine hypotensive syndrome. Engorged vessels of placenta may rupture.
8. **Nutritional and social factors.** Grand multiparity, advancing age, malnutrition, folic acid deficiency, poverty, hard work, cigarette smoking and cocaine abuse.
9. Thrombophilias including lupus anticoagulant antibody, factor V Leiden, proteins C and S deficiency have slightly increased risk of abruption but no convincing evidence (ACOG 2017).
10. External cephalic version
11. Short cord
12. Sudden decompression of the uterus as seen in cases of twins and hydramnios. It causes reduction in surface area of the uterus and hence shearing of the placenta.
13. Preterm rupture of membranes, prolonged rupture of membranes.
14. Uterine anomalies, fibroid and torsion
15. Abnormal placentation (circumvallate placenta)
16. Severe foetal growth restriction
17. Malformed fetus
18. After snake bite
19. High levels of maternal serum alpha fetoprotein and hCG, decreased inhibin A, pregestational diabetes are also high risk factors.
    High second MSAFP (sick placenta syndrome predisposes to abruption).
20. **Hyperhomocystinemia.** There is 3–7 times higher risk of abruption in hyperhomocystinemia. Elevated levels of homocysteine can damage the vascular endotheliam and cause abruption. This also partly explains the high incidence of abruption in women with folate deficiency anaemia.
21. Subclinical hypothyroidism or antithyroid antibodies increases risk by 2–3 fold.
22. Cocaine abuse.

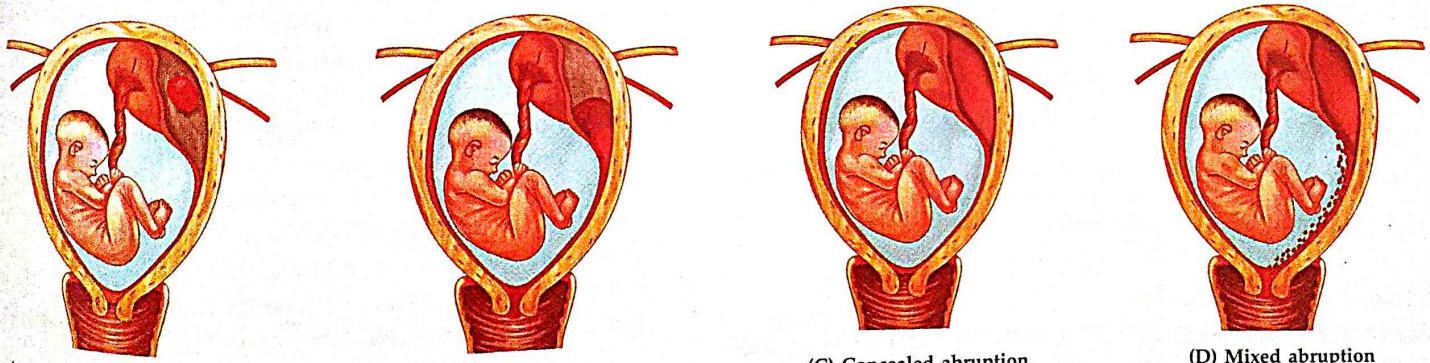
### Clinical types

The clinical types of abruptio placentae are shown in (Fig. 31.4). It is of two types.

1. **Acute.** Haemorrhage at decidual-placental interface.
2. **Chronic.** Uteroplacental insufficiency

### Pathology

In the beginning, there is vasospasm of the uterine vessels, then they relax causing venous engorgement and arteriolar rupture into the decidua. The decidua then splits leaving a thin layer adherent to the myometrium. There is development of a decidual haematoma which leads to



**(A) Retroplacental haematoma**
A very small retroplacental haematoma is formed under placenta.

**(B) Revealed abruption**
The retroplacental haemorrhage is revealed externally from the vagina. It is the **most common** and mild type with general condition being proportionate to bleeding (65–80%).

**(C) Concealed abruption**
The hemorrhagic blood is internal, collecting between the placenta and the uterine wall. It is **rare** but severe type with shock being out of proportion to visible blood loss (20–35%).

**(D) Mixed abruption**
It is a combination of revealed and concealed haemorrhage. It is **quite common** and severe.

**Fig. 31.4:** Types of accidental haemorrhage (abruptio placentae)

further separation, compression and ultimate destruction of the placenta adjacent to it.

Sometimes, a decidual spiral artery ruptures to cause a retroplacental haematoma which expands and disrupts more vessels causing separation of more placental tissue. The escaping blood may dissect the membranes from the uterine wall and eventually appear externally into the amniotic sac **(revealed)** or may be completely retained within the uterus **(concealed)** behind the membranes and even between the muscles **(Couvelaire uterus; see below)**. There is increased intrauterine pressure which further decreases the placental circulation. This adds to the hypoxia of the fetus already present due to separation of the placenta. Routine examination of the placenta after delivery reveals retroplacental clot.

## Couvelaire Uterus (Uteroplacental apoplexy)

Couvelaire uterus is a pathological entity first described by **Couvelaire** in 1912. There is extensive intravasation of blood into the uterine muscles in association with severe form of concealed abruptio placentae. It can only be diagnosed on laparotomy.

**Gross Examination** (Fig. 31.5)

There is patchy or diffuse dark port wine colour of the uterus usually starting from the cornu and then spreading to other areas, especially the placental site. There are petechial hemorrhages underneath the uterine peritoneum. There may be effusion of blood in the peritoneal cavity, broad ligament beneath the tubal serosa and in the substance of ovaries which give port wine stain look.



**Fig. 31.5:** Actual photograph of Couvelaire uterus at time of cesarean section in a case of abruptio placentae (uterus was saved)

**Microscopic Examination**

There is intravasation of blood and fluid in between the uterine muscle bundles causing muscular dissociation in the middle and outer muscle layers. There may be tears in serosa with haemorrhage in the peritoneal cavity. The blood vessels show acute degeneration and thrombosis. It seldom interferes with uterine contractions. **Thus, the presence of Couvelaire uterus during cesarean section is not an indication for hysterectomy.**

**Treatment.** It is same as the treatment of severe abruption (see below).

## Hepatic and renal changes

In severe types of abruptio placentae, a fibrin knot, which is a thrombotic lesion in the hepatic sinusoids, has been described as specific of abruptio placentae. In renal changes, oliguria and anuria may develop due to acute tubular necrosis in mild forms of abruption. Renal cortical necrosis occurs in severe form.

## Disseminated intravascular coagulation or coagulation failure (coagulopathy)

About 5% abruptio placentae cases develop coagulation failure, excess fibrinolysis and hemorrhagic state (see Chapter 41 for details).

## Clinical features of abruptio placentae

Clinical features of abruptio placentae depend upon the type of abruption and are shown in Table 31.3.

## Clinical Grading of Abruptio Placentae

The clinical grading of abruptio placentae is shown in Tables 31.4 and 31.5.

## Investigations

**General**

1. Complete hemogram including Hb estimation, platelet count, leucocyte count and peripheral blood smear.
2. Serum fibrinogen level, prothrombin time (PT), partial thromboplastin time (PTT), Fibrin degradation products (FDP) levels, platelet count, bleeding and clotting time, D-dimer (fibrinolytic activity) levels.
3. Serum electrolytes, blood urea and liver function tests
4. Arterial blood gas analysis
5. Blood group and crossmatching
6. The Kleihauer-Betke test, if available

**Ultrasound Abdomen**

1. To rule out placenta previa
2. To reveal the state of the fetus
3. Retroplacental clots at previous placental site (as seen on earlier ultrasound) is helpful.
4. **Jello sign.** Placenta may jiggle when sudden pressure is applied by transducer.
5. In case of concealed haemorrhage, the placenta can seem thick and globular measuring almost 6cm in diameter.

   However, a negative ultrasonographic finding does not exclude abruption. USG can diagnose abruption in only 15-25% cases.

## Differential Diagnosis

1. **Revealed type.** It has to be differentiated from placenta previa (Table 31.2). Indeterminate causes of APH have to be differentiated (see below).
2. **Mixed or concealed type.** This variety is often confused with: (*i*) Rupture uterus (*ii*) Rectus sheath haematoma

**Table 31.3: Clinical Features in Various Types of Abruptio Placentae**

| | Revealed Type | Concealed and Mixed types |
|---|---|---|
| **Symptoms** | | |
| 1. Vaginal bleeding | Usually slight, continuous, dark red, rarely severe bleeding | Absent but present in mixed type |
| 2. Abdominal pain | No severe pain but discomfort | Acute agonizing pain is present. |
| 3. Preceding symptoms of pre-eclampsia | Edema, headache and vomiting may be rarely present. | Present with higher frequency |
| 4. Past history of pre-eclampsia, essential hypertension, chronic renal disease | May be present | Present with higher incidence. |
| **Signs** | | |
| 1. Shock | Absent | Shock is the main feature. |
| 2. Anaemia | Mild or absent | Always present, moderate to severe |
| 3. Evidence of pre-eclampsia | May be present | Present with higher incidence |
| 4. Abdomen examination | Localized uterine tenderness, normal uterine height, foetal presentation as usual; foetal heart heard. | Tense, tender, hard with rising fundal height and increasing abdominal girth; foetal parts not easily felt and foetal heart not easily audible. |
| 5. Vulval inspection | Usually slight bleeding | Bleeding is absent. It can be there in mixed type. |
| 6. Vaginal examination in an operating theatre | No placenta felt at lower uterine segment but friable and soft blood clot can be felt | No placenta felt at the lower uterine segment |
| 7. Urine output | Normal | Usually reduced |
| **Investigations** | | |
| 1. Placental ultrasonography | Can be helpful in differentiation from placenta previa | Can diagnose sonolucent retroplacental clot in upper segment |
| 2. Blood hemoglobin | Anaemia is proportionate to the blood loss | Severe anaemia, out of proportion to the visible blood loss. |
| 3. Coagulation profile | Usually normal | Usually abnormal<br>1. Clotting time prolonged (> 6 minutes)<br>2. Fibrinogen level is low (< 150 mg/dL)<br>3. Platelet count is low (< 100,000/cm)<br>4. Increased partial thromboplastin time<br>5. Increased fibrin degradation products (FDP) and D-dimer |
| 4. Urine for protein | May be absent | Usually present |

**Table 31.4: Page's Classification (1951) of Abruptio Placentae**

| | |
|---|---|
| Grade 0 | Clinically unrecognized before delivery but diagnosed on examination of placenta after delivery. |
| Grade 1 | 1. External bleeding is slight.<br>2. Shock is absent.<br>3. Uterus is irritable, tenderness may or may not be present.<br>4. Foetal heart is good. |
| Grade 2 | 1. External bleeding mild to moderate.<br>2. Shock is absent.<br>3. Uterine tenderness is marked.<br>4. Foetal distress or death occurs.<br>5. Associated coagulation defect or anuria may be present. |
| Grade 3 | Maternal shock or coagulopathy with foetal death. |

**Table 31.5: Clinical Grading (Sher's) of Abruptio Placentae**

| Grade | Retroplacental clots | Foetal Heart |
|---|---|---|
| I | 150 mL or less | Present. It is diagnosed after delivery. |
| II | 150–500 mL | Abnormal in 92% cases |
| III | As above<br>IIIA without coagulopathy<br>IIIB with coagulopathy | Absent foetal heart |

| Alternative Grading | |
|---|---|
| **Mild haemorrhage** | < 15% blood volume loss<br>No change in vital signs |
| **Moderate haemorrhage** | 15–30% blood volume loss, change in vital signs, tachycardia, low blood pressure |
| **Severe haemorrhage** | > 30% blood volume loss with features of shock |

(*iii*) Appendicular or intestinal perforation (*iv*) Twisted ovarian tumor (*v*) Volvulus (*vi*) Acute hydramnios (*vii*) Tonic uterine contraction (*viii*) Red degeneration in a fibroid

**Points in favor of concealed abruption are.** (*i*) Shock out of proportion to external bleeding. (*ii*) Unexplained severe anaemia (*iii*) Presence of pre-eclampsia (*iv*) Uterus is tense, tender and hard (*v*) FHS is absent (*vi*) Oliguria (*vii*) Presence of bleeding and coagulation disorders (*viii*) Foetal parts difficult to palpate.

## Complications

### Maternal

1. **Hemorrhagic shock.** See Chapter 41 for details.
2. **Postpartum haemorrhage.** See Chapter 25 for details.
3. **Disseminated intravascular coagulation.** Abruption is the most common cause of DIC (see Chapter 41 for details).
4. **Oliguria and anuria**
   There is ischemic necrosis of the kidneys due to hypovolemic hypoperfusion and fibrin deposits from DIC causing acute kidney injury. There can be acute tubular necrosis (reversible) initially. Later, renal cortical necrosis (irreversible) may occur (see Chapter 35[vi] for details).
5. **Puerperal sepsis** (see Chapter 30 for details)
6. **Sheehan's syndrome** (pituitary ischemia) causing failure of lactation (see chapters 20 and 35[iv] for details)
7. **Recurrence.** Risk of recurrence in next pregnancy is about tenfolds with high perinatal mortality.
8. **Death.** Maternal mortality is 2–8 % due to shock, coagulation and renal failure.

### Foetal

High perinatal mortality is seen in abruptio placentae. Perinatal death occurs in 25–30% cases in revealed but 50–100% cases in concealed variety. The causes of death are:

1. Prematurity
2. Asphyxia due to placental abruption.
3. Small for gestational age
4. **Congenital anomalies** (2 to 5 times higher incidence). Anomalies of central nervous system are more common.

## Management

### Prevention

Abruptio placentae is not always preventable. The following modalities may help.

1. Elimination of the known risk factors for placental abruption.
2. Correction of anaemia during antenatal period with hematinics. Routine administration of folic acid from early pregnancy may be of some use.
3. Early diagnosis and timely treatment minimize the serious complications due to placental separation like shock, blood coagulation disorders and renal failure.
4. Prevention, timely diagnosis and appropriate treatment of hypertensive disorders of pregnancy.
5. Chorion villus sampling (CVS) and amniocentesis should be done under ultrasound guidance.
6. **Avoidance of physical trauma.** External cephalic version under anesthesia should be avoided.
7. **To avoid sudden decompression of the uterus.** In acute or chronic hydramnios while performing amniotomy, gradual slow decompression should be done.
8. To avoid supine hypotension, the patient is advised to lie in the left lateral position in the later months of pregnancy.

### Treatment

The patient should be shifted to hospital like a case of placenta previa. The management protocol is shown in Flow Chart 31.2.

Patient is assessed for: (a) Amount of blood loss. (b) Foetal maturity (c) Whether the patient is in labour or not.

Blood is sent for complete hemogram, bleeding and coagulation profile, ABO and Rh grouping and urine for detection of proteins. Ringer's lactate solution drip is started with a large cannula and blood transfusion is arranged. Close monitoring of maternal and foetal conditions is done.

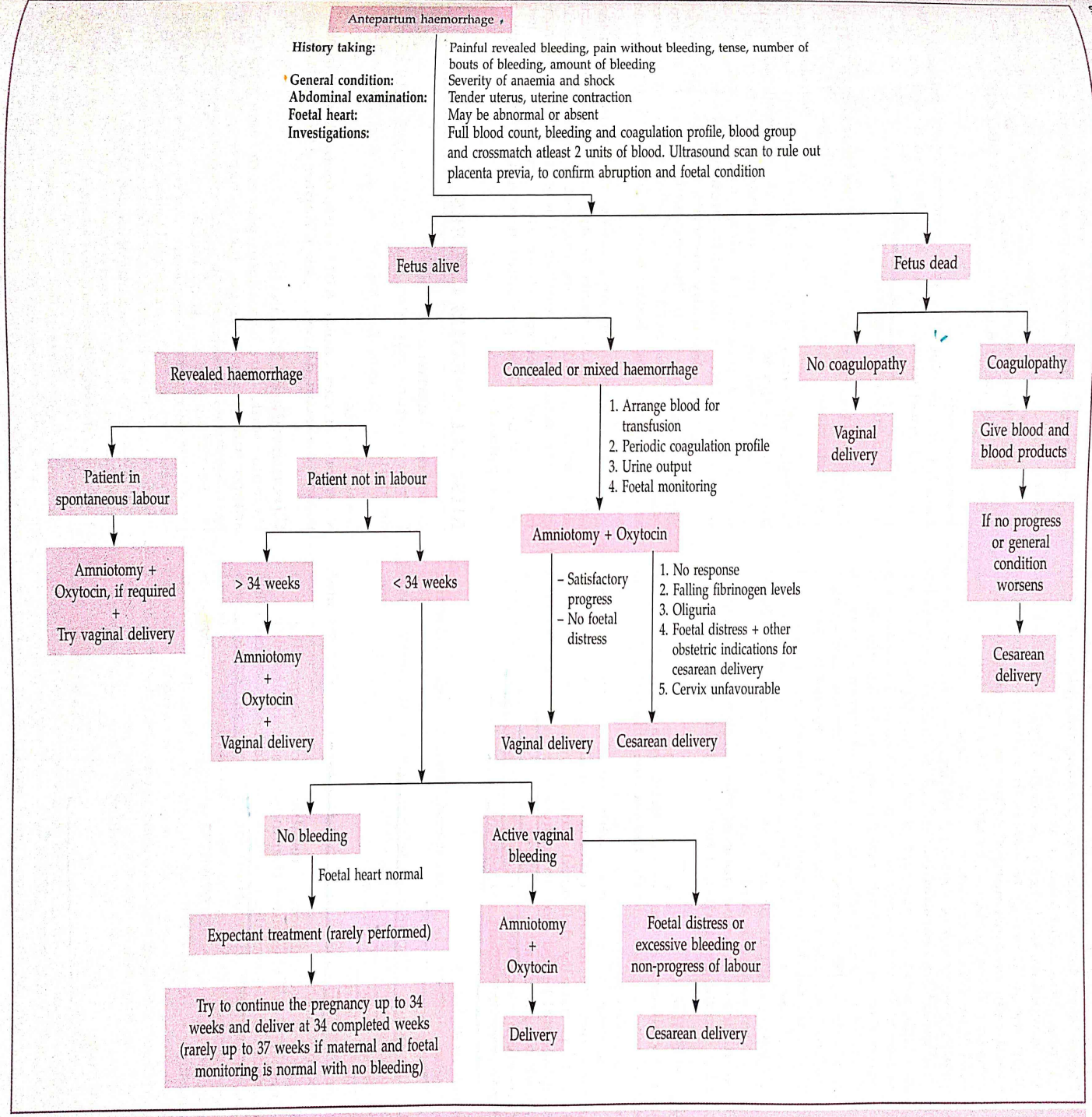
#### Definitive Treatment

***Delivery in all Patients > 34 Weeks***

**Patient is in labour.** Augmentation is done by amniotomy. Rupture of the membranes with escape of liquor amnii not only accelerates labour but it increases the uterine tone, thereby allowing the separated placenta to be compressed between the fetus and the uterine wall. Oxytocin drip can be started to accelerate labour.

**If the patient is not in labour**

1. **Pregnancy is 34 weeks or more.** There is no role of conservative treatment if gestation is > 34 weeks.
   Induction of labour is to be done by amniotomy with or without oxytocin with continuous CTG monitoring.
   **Indications of cesarean delivery (section) are:** (a) Presence of foetal distress. (b) Amniotomy is not possible or amniotomy fails to control bleeding and (c) Associated complicating factors.
   For confusion in the diagnosis with placenta previa, internal examination is performed (Double setup examination) in operation theatre, keeping all things ready for cesarean delivery.
2. **Pregnancy less than 34 weeks**
   (a) **Bleeding, moderate to severe and continuing**—Low rupture of the membranes is done and is quite effective. Oxytocin drip may be added. Labour usually starts soon. Cesarean delivery is often indicated. (b) **Bleeding slight or has stopped**—Conservative treatment is only indicated rarely in small number of patients at < 34 weeks of gestation with a small abruption with normal foetal heart rate. Close observation of the mother and careful foetal monitoring by CTG is done. Betamethasone should be given. Such cases are taken up to 34 weeks when induction of labour should be done. Rarely pregnancy can be taken up to 37 weeks if maternal and foetal monitoring is normal with no bleeding.




**Flow Chart 31.2:** Management plan of abruptio placentae

3. Blood sample is taken for hemogram, bleeding and clotting time and blood is arranged.
4. These women are usually severely hypovolemic. Every effort should be made to restore their effective circulating blood volume with normal saline or hemaccel (colloid) infusion with a large cannula initially followed by sufficient blood transfusion (at least 2 units). Blood pressure may not be reliable in such cases due to severe vasospasm of capillaries. Blood transfusion prevents ischemic renal damage by maintaining blood pressure, prevents blood coagulation disorders and enables the patient to better withstand postpartum haemorrhage. If facilities are available, a central venous pressure line should be inserted for ready route for transfusion and to monitor fluid balance. CVP should be kept at 10 cm water. To maintain adequate organ perfusion, it is necessary to keep a hematocrit of at least 30% and a urinary output of at least 30 mL/hour with liberal intravenous fluids and blood.
5. Amniotomy is to be done at the earliest and oxytocin drip should be started, if not contraindicated.

### Vaginal delivery

Vaginal delivery is the aim as baby is almost invariably dead in severe cases and for the mother vaginal delivery is safer than cesarean delivery.

Following amniotomy with or without oxytocin, delivery usually follows quickly (usually within 6 hours). Placenta is usually delivered quite promptly and contains retroplacental blood clot. Intramuscular oxytocin (10 units) should be given after delivery of the baby to minimize postpartum blood loss. In case of intrauterine foetal death, labour is induced, but amniotomy is avoided.

### Cesarean delivery

Cesarean delivery is usually indicated in the following conditions.

1. Significant abruption where fetus is alive and sufficiently mature to survive.
2. Uncontrolled bleeding
3. Foetal distress
4. Failure to progress in labour inspite of amniotomy and oxytocin drip
5. Maternal condition unstable not responding to resuscitative measures (hemodynamically unstable)
6. Evidence of disseminated intravascular coagulation (DIC)

Cesarean section must be done by an experienced surgeon in the presence of an expert anesthetist with adequate blood bank support. General anesthesia is usually required in patients with hypotension and coagulopathy. If, however, the bleeding and coagulation profile is normal, regional anesthesia can be given. Postpartum haemorrhage due to a poorly contracted uterus must be dealt with accordingly.

### Third stage

Correct management of the third stage of labour is crucial. High dose of oxytocin infusion helps to reduce blood loss.

Examination of the placenta post delivery would enable quantification of the degree and severity of abruption. Anti D is to be given to non-isoimmunized patient in appropriate amount based on a Kleihauer test within 72 hours.

In an event of recurrent episodes of bleeding in a non-sensitised Rh-ve woman beyond 20 weeks, Inc. Anti-D should be given at 6 weekly intervals (RCOG).

### Postnatal Care

Hemodynamic stabililty with normal blood pressure, pulse rate and good urine output are signs of a well resuscitated patient. Coagulation profile should be done as there is increased risk of coagulopathy.

## Indeterminate antepartum haemorrhage (IAPH)

Indeterminate antepartum haemorrhage (IAPH) is a diagnosis of exclusion after ruling out placenta previa, abruptio placentae and local genital tract lesions. Its incidence is quoted to be 2–5%. It is postulated that the origin of bleeding is due to marginal separation of a normally sited placenta leading to a reduced functional reserve. Morbidity is related to preterm delivery.

The patient classically presents with moderate painless vaginal bleeding with the placenta being in the upper segment with no evidence of abruption on ultrasound and there are no local lesions detected on speculum examination.

If the patient remains stable and remote from term, conservative management is appropriate. Close maternal and foetal surveillance are needed. Prognosis is generally good for fetus and mother. There is a reported higher incidence of preterm deliveries and congenital foetal abnormalities. The perinatal mortality may be higher in these patients.

# MISCELLANEOUS CAUSES

## Uterine Rupture

Uterine rupture can also cause APH (see Chapter 26 for details).

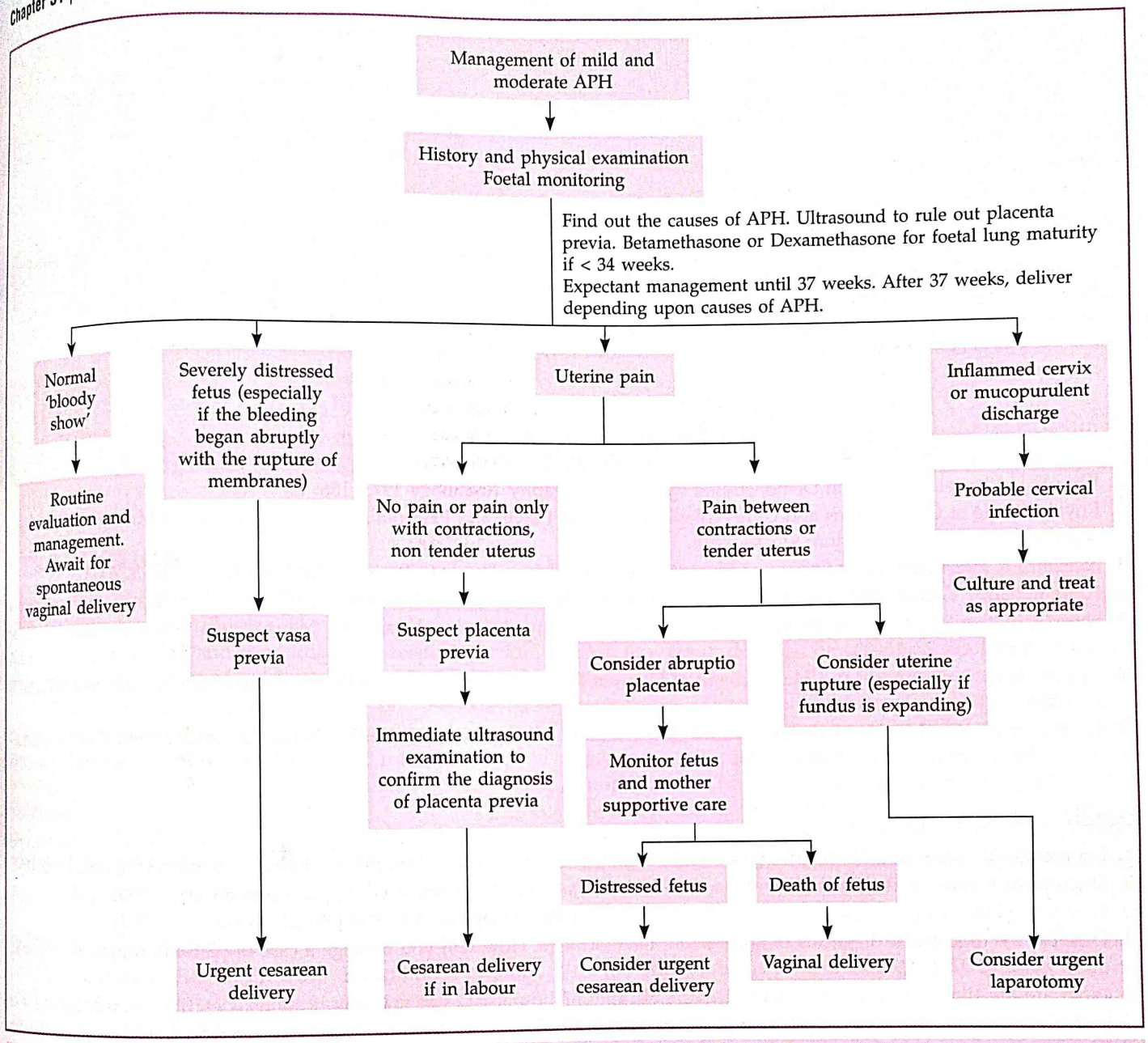
## Vasa Previa

Vasa previa can cause APH with foetal bleeding needing urgent cesarean delivery (see Chapter 24 for details).

## Circumvallate Placenta

Patients with circumvallate placenta can present with antepartum haemorrhage (see Chapter 24 for details). Management is expectant. An ultrasound examination is needed to rule out placenta previa. For severe and persistent bleeding, cesarean delivery is performed. In the absence of other obstetric indications, induction of labour at term is required. Definitive diagnosis is made by examination of the placenta after delivery.

**Local cervical lesions** like cervical intraepithelial neoplasia **(CIN)**, **cervical polyp** and **cervical erosion** are diagnosed on gentle speculum examination performed after bleeding stops in a case of APH. Cervical polyp is removed with a sponge holding forceps. The management of CIN and cervical cancer is discussed in Chapter 37(iii).




**Flow Chart 31.3:** Management of mild and moderate APH

## Key Points

1. Antepartum haemorrhage is an important obstetric condition with high maternal and foetal morbidity and mortality.
2. Placenta previa is a condition in which placenta is situated partly or wholly in the lower uterine segment. National Institute of Health has classified placenta previa to be partially or completely covering the os while low lying placenta with placental edge within 2 cm perimeter of internal os but does not cover it. There are four degrees of placenta previa depending upon the relation of placenta to the internal os. Ultrasound examination is the best modality to make a diagnosis of placenta previa. Low lying placenta detected on ultrasound during early pregnancy should be confirmed in the third trimester by repeat scan as most cases migrate up. Expectant management in the hospital is the management of choice and has improved the maternal and perinatal outcomes in placenta previa.
3. Cesarean delivery is performed for major degrees of placenta previa (Type 2 posterior, Types 3 and 4). Cesarean section for placenta previa should be performed by an experienced surgeon in a well equipped hospital and at least 2 units of blood should be arranged. For anterior placenta previa, surgeon needs to either cut through the placenta or should reach the margin of placenta to rupture the amniotic membranes.

4. Abruptio placentae (ablatio placentae, accidental haemorrhage) is the premature separation of a normally situated placenta whose cause is not known exactly but is associated with certain risk factors like pre-eclampsia, polyhydramnios, multiparity, etc. The various types of abruption are concealed, revealed and mixed. Clinically, it is categorized as mild (< 15% blood loss with no symptoms), moderate (15–20% blood loss with tachycardia and hypotension) and severe type (> 30% blood loss with features of shock).
5. Management depends upon the severity of the condition and amount of bleeding. Conservative treatment is recommended for mild abruption if foetal maturity has not been achieved. For severe abruption, active management with termination of pregnancy is usually recommended. Amniotomy followed by oxytocin infusion is usually started aiming to achieve a vaginal delivery. Cesarean delivery is only done for strict obstetric indications or for foetal distress. Blood transfusion should be arranged for severe variety.
6. Severe abruption is a serious condition and can cause maternal mortality or severe morbidity due to hypovolemic shock, renal failure, postpartum haemorrhage and disseminated intravascular coagulation.

## Suggested Reading

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2. King D L. Placental Migration Demonstrated by Ultrasonography. Radiology 1973; 186: 167.
3. Royal College of Obstetricians and Gynaecologists. Placenta Previa and Placenta Accreta: Diagnosis and Management. Green Top Guideline No. 27a. London: RCOG, 2018.
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7. Oppenheimer L. Diagnosis and Management of Placenta Previa. Journal of Obstetrics and Gynecology of Canada 2007; 29(3): 261–266.
8. Society for Maternal Foetal Medicine. Gyamfi Bannerman C. Management of bleeding in later preterm period. Consult Series no. 44. Am J. Obstetric Gynecology 2017.

## Examination Questions

A. Enumerate the causes of APH. Describe the management of a primigravida admitted at 32 weeks gestation with APH. (DU 1995, 1998)
B. Discuss the management of a third gravida with 34 weeks of gestation with bleeding per vaginum. (DU 1994)
C. Enumerate the causes of antepartum haemorrhage. Discuss the management of abruptio placentae. (DU 1992)
D. Describe the differential diagnosis of antepartum haemorrhage. How will you manage a case of placenta previa at 32 weeks of pregnancy? (DU, 1990)
E. What are the clinical features of a case of severe abruption? Enumerate various complications. Describe the management of severe abruption at 37 weeks of pregnancy. (Many universities)
F. Write short notes on:
   1. Dangerous placenta previa (Type 2 posterior placenta previa) (DU and other universities, many times)
   2. Macafee's regimen for placenta previa
   3. Causes of maternal mortality in abruptio placentae
   4. Complications of abruptio placentae
   5. Abruptio placentae (Many universities)

CHAPTER 32

# Hypertensive Disorders of Pregnancy

## INTRODUCTION

Hypertensive disorders complicating pregnancy are common disorders contributing significantly to maternal and perinatal mortality and morbidity. It is responsible for 14% maternal deaths worldwide including India (WHO 2019). Pre-eclampsia or superimposed on pre-existing hypertension presents the major risk for women causing maternal and foetal serious morbidity and mortality.

### Incidence

Hypertensive disorders complicating pregnancy vary from 5 to 10%, with estimated 50,000–60,000 pre-eclampsia related deaths per year worldwide.

### Definition of hypertension

The National High Blood Pressure Education Program (NHBPEP, USA, 2000) has defined hypertension in pregnancy as a blood pressure (BP) measurement of more than or equal to 140/90 mm Hg. Old recommendation of a rise in systolic BP by 30 mm Hg or diastolic BP by 15 mm Hg is not current recommended in diagnosis of hypertension in pregnancy.

Delta hypertension: Sudden rise in mean arterial pressure (MAP) from baseline BP near term but still in normotensive range. It also signifies pre-eclampsia and can lead to eclampsia and HELLP syndrome.

### Classification

BP is measured in sitting or semi reclining position with back supported on the brachial artery with the mercury sphygnomanometer at the level of the heart. The cuff should be of appropriate size (12.5 to 13 cm in width and 35 cm in length for an average adult). Use of Korotkoff phase V (disappearance of sounds) is used for diastolic BP.

Hypertension during pregnancy is classified into five categories as per recommendation of National High Blood Pressure Education Programm, 2000. The details of diagnostic features of various categories are shown in Table 32.1. ACOG (2019) has modified the classification to some extent. They now take either a systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on two occassion repeated 4 hours apart . BP returns to normal by 12 weeks postpartum. They have also removed the dependence of the diagnosis of pre-eclampsia upon proteinuria and have made it proteinuria or any evidence of maternal compromise. They have also removed the term 'Mild' pre-eclampsia as it is never mild as it causes increased morbidity and mortality and rapid progress is possible. The new name is **pre-eclampsia without severe features** (now called only **'Pre-eclampsia'**) and **pre-eclampsia with severe features (severe pre-eclampsia)**.

ACOG (2019) gives less importance to proteinuria as the studies have proven that there is no difference in maternal and perinatal outcomes with different grades of proteinuria. Hence, delivery decisions should not be made on the basis of amount of proteinuria or change in amount of proteinuria. Also the notion of linear progression of pre-eclampsia without severe features to pre-eclampsia with severe features is inaccurate (ACOG 2019).

### Severity of Hypertensive disorders during pregnancy

Severity of hypertensive disorders during pregnancy are described as **mild** or **severe** disease depending upon various symptoms, signs and investigations as shown in Table 32.2. Some authors add moderate category (diastolic BP 100–109 mm Hg) but it is not universally accepted.

As per the FOGSI GCPR 2019 current classification as per severity is -

**Non-severe pre-eclampsia:**

- Blood pressure ≥ 140/90 mm Hg and ≤ 160/110 mm Hg
- No premonitory symptoms and normal HDP laboratory parameters.

**Table 32.1: Classification of Hypertensive Disorders in Pregnancy (ACOG 2019)**

**I. Gestational Hypertension**

1. Blood pressure (BP) 140/90 mm Hg or more for the first time in pregnancy after 20 weeks of gestation on two occasions 4 hours apart
2. No proteinuria
3. Return of BP to normal within 12 weeks after delivery.
4. Other signs of pre-eclampsia like headache, epigastric discomfort or thrombocytopenia may be present. Final diagnosis is made only after 3 months of delivery.
5. Although transient, may be sign of future chronic hypertension

**II. Pre-eclampsia-Eclampsia**

**A. Pre-eclampsia (New Hypertension and Quantified Proteinuria)**

1. **Basic (least) criteria**
   (a) BP – 140/90 mm Hg or more after 20 weeks gestation on two occasions atleast 4 hours apart in a previously normotensive patient/woman
   (b) Proteinuria > 300 mg in 24 hours or persistent 2+ or more on dipstick random samples or Protein/creatinine ratio > 0.3 mg/dl (ACOG 2019)
2. **Definitive criteria**
   (a) BP 160/110 mm Hg or more
   (b) Proteinuria 2+ on dipstick or 2 g in 24 hours urine
   (c) Raised serum creatinine > 1.1 mg/dl or doubling of serum creatinine in absence of other renal disease
   (d) Thrombocytopenia (platelets < 100,000/mm$^3$)
   (e) Evidence of hemolysis (raised lactic dehydrogenase [LDH] levels)
   (f) Elevated liver enzymes, especially alanine aminotransferase (ALT) or aspartate aminotransferase (AST), twice normal concentration
   (g) Symptoms of severe pre-eclampsia like persistent headache, cerebral or visual disturbances or persistent epigastric pain
   (h) Pulmonary edema

**B. Eclampsia**

1. Development of convulsions in a case of pre-eclampsia which cannot be attributed to another cause.

**III. Chronic Hypertension (of any cause)**

1. A known hypertensive woman becoming pregnant with BP 140/90 mm Hg or more before pregnancy or diagnosed before 20 weeks of pregnancy in absence of gestational trophoblastic disease.
2. Hypertension first diagnosed after 20 weeks but persisting 12 weeks after delivery.

**IV. Superimposed Pre-eclampsia (Development of Features of Pre-eclampsia on Chronic Hypertension)**

1. Onset of proteinuria of 300 mg/24 hours or more after 20 weeks of pregnancy in a woman who had hypertension but no proteinuria before 20 weeks gestation
2. Development of sudden increase in proteinuria, hypertension or thrombocytopenia (< 100,000/mm$^3$) or deranged liver or renal function test in a woman who had hypertension or proteinuria before 20 weeks gestation.

**Table 32.2: Severity of Hypertensive Disorders During Pregnancy**

| Characteristics | Pre-eclampsia without severe features [formerly non-severe (mild) disease] | Pre-eclampsia with severe features (formerly severe disease) |
|---|---|---|
| **Symptoms** | | |
| 1. Headache | Nil | Present |
| 2. Epigastric or upper abdominal pain | Nil<br>Nil | Present<br>Present |
| 3. Visual or cerebral disturbances | Nil<br>Absent | Present<br>May be present |
| 4. Convulsions | Absent | May be present |
| **Signs** | | |
| 1. Systolic BP | < 160 mm Hg | ≥ 160 mm Hg |
| 2. Diastolic BP | < 110 mm Hg | ≥ 110 mm Hg |
| 3. Brisk reflexes or clonus | Absent<br>Nil | Present<br>Present |
| 4. Foetal growth restriction | Nil<br>Nil | Present<br>Present |
| 5. Cardiac dysfunction with pulmonary edema | Absent | Present |
| 6. Oliguria | Absent | Present |
| **Investigations** | | |
| 1. Proteinuria | None to Positive | None to Positive |
| 2. Serum creatinine | Normal | Elevated |
| 3. Platelet count | Normal | Diminished (< 100,000/mm$^3$) |
| 4. Elevated hepatic enzymes (ALT, AST) | Absent | Present |
| 5. Evidence of hemolysis | Absent | Present |

Term 'milk Pre-eclampsia' has been removed (ACOG 2013)

**Severe pre-eclampsia:**

Blood pressure > 160/110 mm Hg with/without premonitory* symptoms with/without abnormal# HDP laboratory parameters. Or

Blood pressure ≥ 140/90 mm Hg with premonitory* symptoms and/or abnormal# HDP laboratory parameters.

***Premonitory Symptoms:** Headache, blurring of vision, vomiting, right upper quadrant pain, sudden excessive weight gain and severe edema.

**#Abnormal HDP Parameters:** Low platelets, elevated liver enzymes, elevated serum creatinine, and abnormal coagulation profile.

Proteinuria is measured in 24 hours urine specimen or by dipstick as negative (< 0.1 g/24 hr.) trace (0.1–0.2 g/24 hrs), 1+ (0.2–0.5 g/24 hrs), 2+ (0.5–1.5 g/24 hrs),

**Table 32.3: Risk Factors for Pre-eclampsia**

| Risk factors | Relative Risk (RR) Wherever Available (Approx.) |
|---|---|
| **Genetic factors** | |
| Race and ethnicity: More common in Blacks and Asians<br>Family history of pre-eclampsia | 3 |
| **Age and parity** | |
| Teenage pregnancy<br>Age more than 35 years<br>Long interval between pregnancies<br>Nulliparity | 1.2<br>2.1 |
| **Partner-related factors** | |
| Change of partner<br>Partner who fathered a pre-eclamptic pregnancy in another woman<br>Limited sperm exposure<br>Pregnancy due to donor insemination | |
| **Presence of underlying disorders** | |
| Chronic hypertension (15–35%)<br>Diabetes mellitus (10–40%)<br>Renal disease<br>Obesity (body mass index > 30 kg/m$^2$)<br>(a) Before pregnancy<br>(b) At booking<br>Maternal low birth weight<br>Polycystic ovarian syndrome<br>Migraine<br>Collagen vascular disorders like SLE<br>Uncontrolled hyperthyroidism<br>Factor V Leiden deficiency, activated Protein C deficiency and thrombophilia<br>Sickle-cell disease or trait and other hemoglobinopathies<br>Anti-phospholipid antibodies (10–40%)<br>Protein S deficiency and hyperhomocysteinemia | 5.1<br>3.7<br>1.8<br>2.8<br>2.5<br>1.5<br>2.5<br>2.8 |
| **Pregnancy-related risk factors** | |
| Multiple pregnancy<br>Hydatidiform mole<br>Hydrops fetalis<br>Congenital and chromosomal foetal anomalies (Trisomy 13, triploidy)<br>Urinary tract infection | 3 |
| **Miscellaneous factors** | |
| Smoking (reduced risk)<br>Psychological strain and stress at working place<br>Previous history of pre-eclampsia<br>Raised blood pressure (diastolic > 80 mm Hg) at booking<br>Prior absuption<br>Use of Assisted reproductive techniques (ART) | 8.4<br>1.4<br>2.0<br>1.8 |

3 + (2.0–5.0 g/12 hrs, 4 + (> 5 g/24 hrs). FIGO classification of Pre-eclampsia (PE) (2019)

1. Early onset PE: with at < 34 weeks of gestation
2. Late onset PE: with at ≥ 34 weeks of gestation
3. Preterm PE: with at < 37 weeks of gestation
4. Term PE: with at ≥ 37 weeks of gestation

## ETIOLOGY

The various risk factors for the development of gestational hypertension and pre-eclampsia are shown in Table 32.3. The exact etiology is not known but is probably multifactorial. Hypertension during pregnancy occurs more often in women exposed to the chorionic villi for the first time (primigravidas); those exposed to excess of chorionic villi (multiple pregnancy and hydatidiform mole); having pre-existing hypertension or vascular disease and in genetically predisposed women. It can occur in the absence of fetus (e.g. hydatidiform mole) and in the absence of chorionic villi in uterus (e.g. ectopic pregnancy).

Various theories put forward are as follows:

### 1. Abnormal Trophoblastic Invasion and Placental Ischemia Theory

According to abnormal trophoblastic invasion and placental ischemia theory, pre-eclampsia is a two-stage placental disease. In the first stage, there is incomplete trophoblastic invasion of spiral arteries with failure of secondary wave and deficient blood supply to the placenta (Fig. 32.1). The second stage includes the effects of placental ischemia on both the mother and the fetus. There is also increased microdeportation of syncytiotrophoblast microvillous membrane particles and glycogen accumulation in syncytiotrophoblasts in patients with pre-eclampsia.

### 2. Genetic Imprinting

Pre-eclampsia and eclampsia may develop based on a single recessive gene or a dominant gene which explains its higher prevalence in daughters of pre-eclamptic women than their daughters-in-law.

### 3. Immunological Factors

According to this most recent theory, pre-eclampsia occurs due to inadequate maternal antibody response to the foetal allograft with vascular damage from circulating immune complexes. Past exposure to paternal antigens (placenta and foetal cells) is protective which explains high occurrence of pre-eclampsia in primigravidas and in pregnancies on change of partner or on donated sperms.

### 4. Maternal Maladaptation to Vascular and Inflammatory Changes (Maternal syndrome)

Ischemic changes in placenta may incite activation of leucocytes with raised cytokine and interleukin levels which may contribute to oxidative stress which is often associated with pre-eclampsia. Oxidative stress markers like malonyldialdehydes, glutathione peroxides, super oxide dismutase, etc., have been found to be increased and

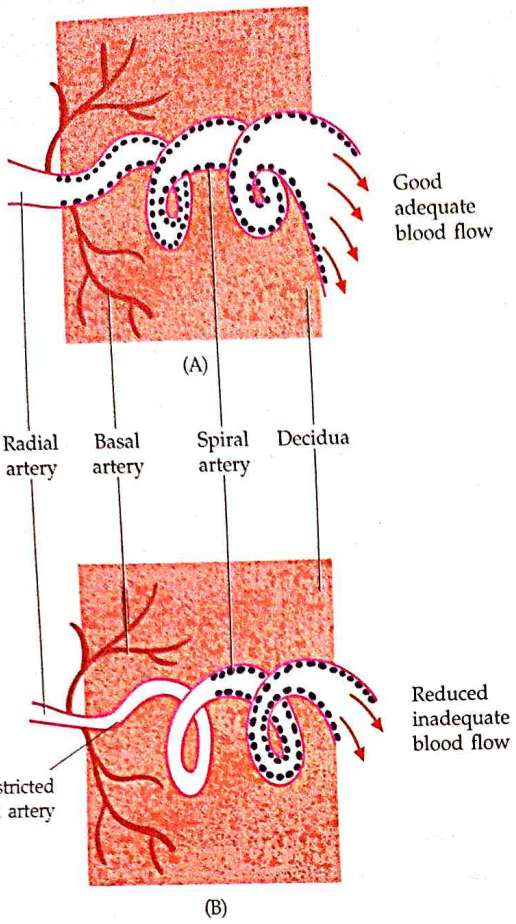



**Fig. 32.1:** Vascular pathology in spiral arteries in pre-eclampsia. (A) Capacitance (dilated) spiral artery in normal pregnancy; (B) Constricted spiral artery in pre-eclamptic pregnancy

antioxidants like lycopene and Vitamin C have been found to be reduced in maternal sera, decidua and placenta of women having pre-eclampsia.

### 5. Very Low Density Lipoproteins (VLDL) Versus Toxicity Preventing Activity

Association of obesity with pre-eclampsia is due to increased free fatty acid (FFA) levels with relative insulin resistance and increased FFA to albumin ratio along with increased lipolytic activity. The levels of vascular endothelial growth factor (VEGF) which plays an important role in mediation of vasculogenesis, angiogenesis, control of the microvascular permeability and vasodilatation are lower in women with pre-eclampsia than controls.

### 6. Increased Pressor Responses

There are decreased prostacyclin ($PGI_2$) (vasodilator) and increased thromboxane A2 (vasoconstrictor) with decreased prostacyclin/thromboxane ratio in women at about 22 weeks of gestation who develop pre-eclampsia later. Nitric oxide (vasodilator) levels are decreased while endothelin-I (vasoconstrictor) levels are raised in pre-eclampsia.

### 7. Angiogenic and antiangiogenic proteins

Trophoblast of women destined to develop pre-eclampsia over produce two antiangiogenic peptides which enter maternal circulation. Soluble fms like tyrosine kinase I (sFlt-I) is a receptor for vascular endothelial growth factor (VEGF). Its elevated levels in PE inactivate and reduce circulating free placental growth factor (PIGF) and VEGF concentration causing endothelial dysfunction. Hence elevated levels of sFlt-I and decreased levels of PIGF are seen in pre-eclampsia and can even be used in its prediction.

Another antiangiogenic peptide soluble endoglin (sEng) is also raised in P.E. It inhibits transforming growth factor beta (TGF-β) from binding to endothelial receptors which decreases endothelial nitric oxide levels.

### 8. Interaction of Various Factors

The above hypotheses are not mutually exclusive, but are probably interactive. It is proposed that in genetically susceptible individuals, immune maladaptation, cytokine-mediated oxidative stress markers cause endothelial cell dysfunction with placental ischemia and increased syncytiotrophoblast deportation.

### 9. Genesis of Pre-eclampsia as a Two-stage Disorder

Genesis of pre-eclampsia can be explained as a two-stage disorder. Decreased placental perfusion (stage 1) is the initiating feature which then results in maternal syndrome of pre-eclampsia (stage 2) (Fig. 32.2).

## PATHOPHYSIOLOGY

Pre-eclampsia is a dynamic and progressive process. It is essentially a syndrome with **multi-organ involvement** with adverse maternal and foetal effects as a consequence of **vasospasm, endothelial dysfunction and ischemia**. The pathological changes in the organs are thrombosis of capillaries, smaller veins and arterioles with haemorrhage and necrosis. The pathological changes in various organs are as follows:

### 1. Uteroplacental Bed

Due to vasospasm of uteroplacental vasculature, the mean diameter of myometrial spiral arterioles is reduced from 500 µm (normal) to 200 µm causing increased perinatal mortality and morbidity through intrauterine growth restriction, abruptio placentae, placental infarction and iatrogenic prematurity. There is increased resistance to blood flow in uterine arteries in women with pre-eclampsia on Doppler velocimetry as compared with normal pregnancies. On histopathological studies of placenta in pre-eclamptic women, there are more chances of premature ageing of the placenta and areas of acute red and white infarcts on the maternal surface of the placenta. Microscopic examination of villi demonstrates increased frequency of various pathologies like syncytial degeneration, excessive proliferation of cytotrophoblasts, increased frequency of syncytial knots, thickening of basement layer and end-arteritis. Due to changes in maternal blood vessels, blood flow through the intervillous circulation and to the fetus is reduced significantly.

### 2. Cardiovascular System

(a) There is severe disturbance in the normal cardiovascular

functions in hypertensive disorders during pregnancy depending upon severity of hypertension, presence of any underlying chronic disease, presence of pre-eclampsia and the duration of pregnancy and disease. There is contracted plasma volume and hemoconcentration in pre-eclampsia along with increased systemic vascular resistance, normal or decreased cardiac output and decreased cardiac preload. Besides, there are increased levels of thromboxane $A_2$ (which cause vasoconstriction and platelet aggregation) and decreased levels of prostacyclin (which causes vasodilatation and inhibition of platelet aggregation) and nitric oxide in patients with pre-eclampsia. The vessels of these women are more responsive to angiotensin-II levels. These changes are responsible for endothelial damage, vasoconstriction and increased peripheral resistance. Central venous pressure (CVP) and pulmonary wedge pressure are reduced in pre-eclamptic women.

(b) **Blood and coagulation.** While most women with pre-eclampsia and eclampsia have a normal coagulation profile, hematological abnormalities in the form of thrombocytopenia (most common), microangiopathic hemolytic anaemia (increased erythrocyte destruction with hemoglobinemia and hemoglobinuria), increased blood viscosity, hemoconcentration and sometimes even disseminated intravascular coagulation (DIC) may be observed in some women, especially with more severe disease.

## 3. Volume Homeostasis

(a) **Fluid and electrolyte changes.** Most women with severe pre-eclampsia have edema (expanded extracellular fluid) which is possibly due to endothelial injury. Apart from generalized edema and proteinuria, reduced plasma oncotic pressure in these women causes filtration imbalance causing further displacement of intravascular fluid into the extracellular space. Usually there is no electrolyte imbalace in pre-eclampsia except after a convulsion when there may be **lactic acidosis** with compensatory respiratory loss of carbon dioxide.

(b) **Endocrine changes.** In normal pregnancy plasma levels of renin, angiotensin II and aldosterone are increased. However, their levels are reduced to non-pregnant levels in women with pre-eclampsia. But their vessels are more responsive to their chemical agents. Levels of vasopressin are normal in pre-eclamptic women. However, levels of atrial natriuretic peptide are increased in pre-eclampsia especially after volume expansion helping in fall in peripheral vascular resistance.

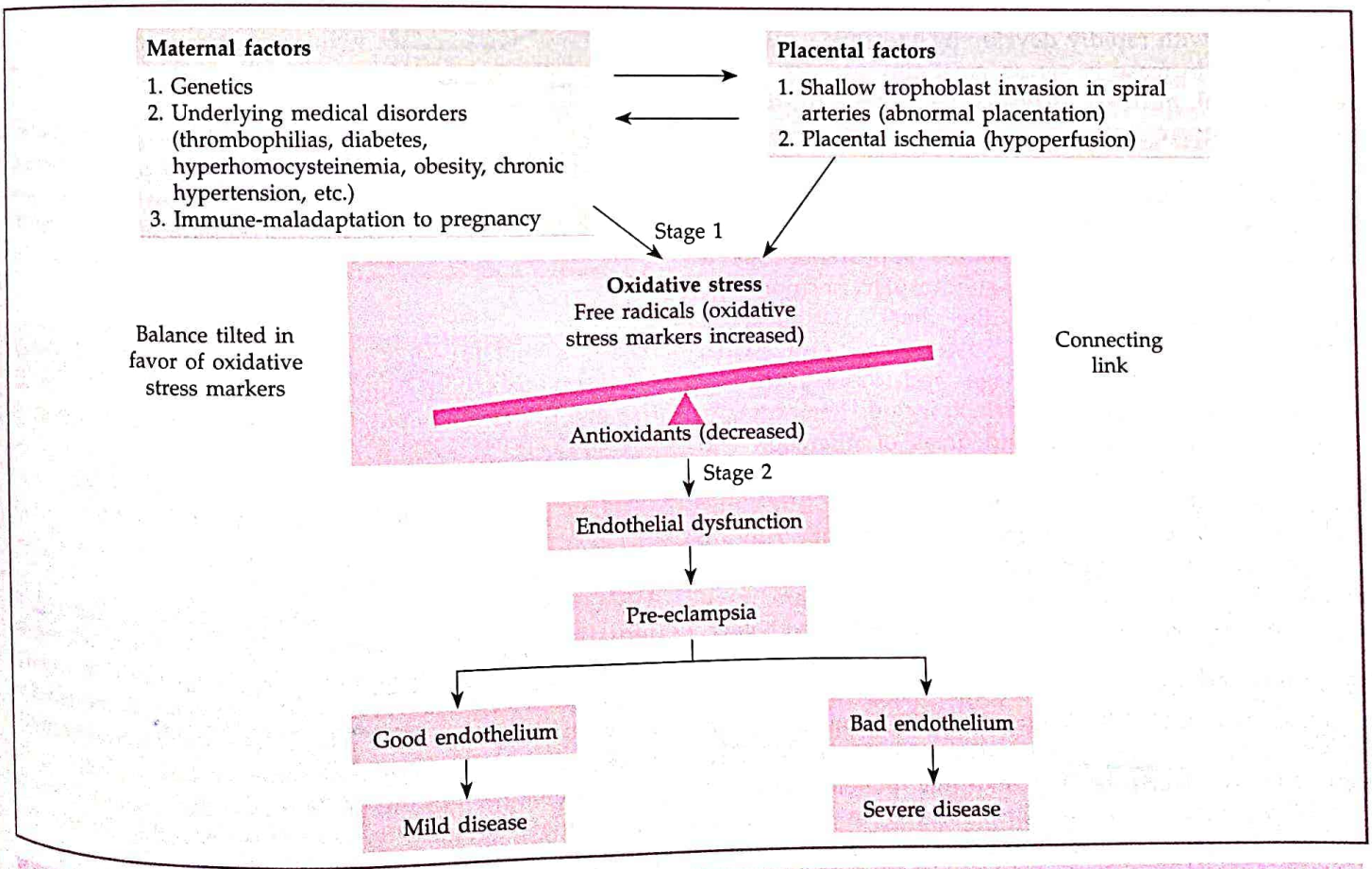



**Fig. 32.2:** Genesis of pre-eclampsia as a two-stage disorder

## 4. Renal Changes

The various changes observed in renal glomeruli in pre-eclampsia are as follows:

(a) Glomerular enlargement. The endothelial cells are swollen, their fenestrations and pedicels are narrowed.
(b) Hyaline, foam cells with 'pouting' of glomeruli and fat in glomerular cells
(c) Deposition of IgM fibrin and thrombi in the glomerular capillaries.
(d) Thickened epithelial tuft with vacuoles
(e) Swelling of mesangial cells
(f) Moderate or gross ballooning of loops

These changes lead on to reduced GFR, increase in blood urea and serum creatinine and proteinuria. There is concomitant tubular dysfunction which explains hyperuricemia in these women. Presence of red cells, hyaline and granular casts in the urine in these women are due to glomerular and tubular damage. Proteinuria occurs due to increased leakage of proteins from glomerular endotheliosis and increased capillary permeability along with concomitant depression of tubular reabsorption. Out of the total protein excreted in urine, albumin constitutes 50–60% and alphaglobulin constitutes 10–15% of the loss. There is reduction in urine output (**oliguria** [less than 500 mL urine in 24 hours]) with severe disease. In severe disease, acute renal failure from **acute tubular necrosis (reversible)** may develop and manifests as oliguria or anuria alongwith rapidly developing azotemia causing up to 1 mg/dL increase in serum creatinine per day. Rarely, **renal cortical necrosis (irreversible renal failure)** may develop in severe cases.

## 5. Hepatic Changes

The hepatic changes of pre-eclampsia are characteristic. Its surface is smooth with mottled appearance due to numerous scattered areas of subcapsular haemorrhage. Periportal haemorrhage in the liver periphery is characteristic. Secondary to vasospasm, intravascular coagulation, endothelial damage and vasoconstriction, various hepatic changes occur which include hemorrhages, periportal fibrin deposition and areas of infarction and necrosis. The hemorrhages in the liver may extend beneath the hepatic (Glisson's) capsule causing its stretching (subcapsular haematoma) or more severe hepatic rupture (causing pain in shoulder). Hepatic changes are seen in about 10% cases of severe pre-eclampsia and are more common in presence of HELLP syndrome described below.

### HELLP syndrome

It was first described by Weinstein in 1985 and is a form of severe pre-eclampsia characterized by hemolysis (H), elevated liver enzymes (EL) and low platelets (LP) (< 100,000 per $mm^3$) and may be observed in about 20% women with severe pre-eclampsia and eclampsia. Usually hemolysis is depicted on periphera smear and it is associated with Haptoglobin levels < 25 mg/dl and total bilirubin > 1.2 mg/dL. Symptoms are nausea, vomiting, epigastric and right upper quadrant pain. Adverse outcome was observed in 40% women with about 1% maternal mortality. The various complications which can occur in them are eclampsia, placental abruption, pulmonary edema, acute renal failure and sepsis.

**Differential diagnosis** is from liver diseases like viral hepatitis, acute fatty liver of pregnancy, renal disease, gall bladder disease, pancreatitis, hyperemesis gravidarum, idiopathic thrombocytopenic conditions and hemolytic uremic syndrome.

There are two types of classifications as shown in Table 32.4. These classes are useful to predict the rapidity of recovery, maternal and perinatal outcomes and need for plasmapheresis.

**Table 32.4: Sub-types of HELLP Syndrome Depending upon Laboratory Abnormalities**

| Mississippi Classification | Tennessee Classification |
|---|---|
| • Class 1 (most severe)<br>1. Platelets < 50,000<br>2. AST or ALT > 70 IU/L<br>3. LDH > 600 IU/L | • True or complete<br>1. Platelets < 1,00,000<br>2. AST > 70 IU/L<br>3. LDH > 600 IU/L |
| • Class 2<br>1. Platelets 50,000–1,00,000<br>2. AST or ALT > 70 IU/L<br>3. LDH > 600 IU/L | • Partial or incomplete<br>– Severe pre-eclampsia with any one or two of the following: ELLP, HEL, EL, LP<br>If all first three parameters present complete HELLP<br>If 2 out of first 3 present: Partial HELLP |
| • Class 3 (least severe)<br>1. Platelets 1,00,000–1,50,000<br>2. AST or ALT ≥ 40 IU/L<br>3. LDH > 600 IU/L | |

ELLP: Absence of hemolysis; HEL: Absence of low platelets; EL: Elevated liver enzymes; LP: Low platelets

### *Criteria for Diagnosis for HELLP Syndrome*

1. Hemolysis, bilirubin more than or equal to 1.2 mg/dL, absent plasma haptoglobulin, schistiocytes in the blood smear (LDH > 600 IU/L).
2. Elevated liver enzymes (AST or ALT > 70 IU/L)
3. Low platelet count, platelets < $100 \times 10^3/mm^3$

#### Management

Women with HELLP syndrome should be treated in tertiary care facility. Treatment is like severe pre-eclampsia, using antihypertensives and anticonvulsants (intravenous magnesium sulphate) along with delivery of the fetus at or more the patient is ≥ 34 weeks - delivered, before 34 weeks - give corticosteroid and deliver, after stabilizing the general condition of the patient. Although role of steroids as treatment of HELLP syndrome is debatable, Mississippi protocol recommends routine use of intravenous dexamethasone 12 mg every 12 hourly for severe form of HELLP syndrome (class 1, class 2) and for mild form (class 3 or partial) with complications (eclampsia, severe

hypertension difficult to control, severe epigastric pain or end organ injury [renal, hepatic, CVS, CNS]) and/or DIC or abruption placentae. As most studies did not find benefit with steroids in HELLP syndrome, the task force of ACOG (2019) does not recommend steroid treatment for it. Blood and blood products should be given as per the natural course of the disease.

Postpartum: complete blood count, liver function test, kidney function tests to be monitored. Antihypertensives and magnesium sulphate to be given for 24 hours after delivery.

## 6. Changes in the Brain

The changes spare the brain stem area but affect posterior hemisphere (causing visual disturbances). Computerized axial tomography (CT scan) and magnetic resonance imaging (MRI) may show the following abnormalities.

1. Cerebral edema and edema in occipital white matter.
2. Cerebral haemorrhage and haemorrhage in ventricles, parenchyma and subarachnoid space.
3. Focal hypodense areas due to petechial haemorrhage and local edema.
4. Normal cortical sulci are lost.
5. Sudden development of hydrocephalus
6. Basal ganglion infarcts
7. Cerebral infarcts

*Headache, visual symptoms and convulsions occur due to brain involvement in the disease process.* Electroencephalogram (EEG) shows increased irritability of cerebral function explaining exaggerated reflexes on examination.

## 7. Visual Disturbances and Retinal Changes

Visual disturbances like blurring of vision are common in severe pre-eclampsia.

The various retinal changes in hypertension modified from classification of American Ophthalmological Society are shown in Table 32.5.

**Table 32.5: Classification of Retinal Changes in Hypertension during Pregnancy**

| | |
|---|---|
| Grade 0 | Normal fundus |
| Grades 1 and 2 | Characterized by slight or moderate degrees of reflex strip arterio-venous narrowing and spasm – the stage of angiospasm |
| Grade 3 | The above with edema, haemorrhage and exudates – pre-eclamptic retinopathy |
| Grade 4 | Papilledema |

Retinal detachment can rarely occur causing unilateral altered vision (rarely total vision loss) and may co-exist with visual defects and cortical edema. Blindness can also occur following eclamptic fit due to extensive vasogenic edema in occipital lobe. Prognosis is good and vision returns to normal within a week. Opinion of an expert ophthalmologist should be taken.

## 8. Changes in the Respiratory system

Various respiratory changes which can be seen are as follows:

1. Pulmonary edema
2. Scattered alveolar hemorrhages
3. Adult respiratory distress syndrome (ARDS)
4. Pneumonia/Patchy pneumonitis
5. Chemical pneumonitis

## 9. Other Organs

There may be subendothelial hemorrhages in heart or focal necrosis or haemorrhage in the myocardium which can affect the conduction system and may cause cardiac failure. Adrenal glands may show haemorrhage and necrosis. Stomach may show hemorrhagic gastritis.

# PREDICTION OF PRE-ECLAMPSIA

A variety of clinical, biological, biochemical and biophysical markers have been tried by various investigators as a screening method for prediction of pre-eclampsia and are shown in Table 32.6A.

Primary clinical assessment for screening and prediction of pre-eclampsia can be objectively performed by 'easy to use' HDP-Gestosis score as per the FOGSI GCPR 2019.

### Process of risk scoring (Table 32.6B)

- This score involves all the existing and emerging risk factors in the pregnant woman.
  Score 1, 2 and 3 is allotted to each clinical risk factor as per its severity in development of pre-eclampsia.
- With careful history and assessment of woman a total score is obtained time to time.
  When total score is ≥ 3; pregnant woman should be marked as 'At risk for Pre-eclampsia'.

### Prevention of Pre-eclampsia

All 'at risk' women should be started with Aspirin 75-150 mg.

**Dose.** The optimum dose of aspirin is unclear; studies have used 60-75-100-150 mg. Low-dose aspirin has a good maternal and foetal safety profile.

### When to start?

Aspirin for this indication should be started even earlier than 12 weeks.

Defective placentation is considered as the causative factor of pre-eclampsia.

Early aspirin balances the levels of thromboxane A2 and prostacyclin which will maintain adequate uteroplacental blood flow and improve placentation without increasing the risks of adverse maternal and perinatal outcomes. Therefore, it appears safe to use low-dose aspirin as a prophylaxis to prevent pre-eclampsia throughout pregnancy until 2 days prior to delivery or cesarean section.

Along with aspirin, calcium 1-1.5 gm daily (in low calcium intake group) is to be started.

**Table 32.6A: Clinical, Biological, Biochemical and Biophysical Markers for Prediction of Pre-eclampsia**

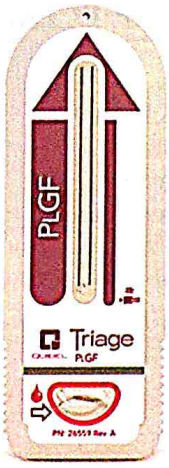
| Methods | Results |
|---|---|
| 1. Past history of high risk factors for pre-eclampsia | Two to fivefold rise in risk of pre-eclampsia. Useful, but does not predict many cases |
| 2. Blood pressure (MAP > 90 mm Hg in 2nd trimester) | 10% positive predictive value (PPV). Not a useful predictor. |
| 3. **Roll over test** done between 28–32 weeks. BP is measured with patient on her side first and then she is asked to roll on her back and BP is checked again. | An increase of 20 mm Hg in diastolic BP from lateral to supine position suggests a positive 'Roll over test' with one-third of such women developing pre-eclampsia in future |
| 4. Angiotensin II (A II) sensitivity test | Rise in BP with A-II infusion. Disappointing positive predictive value (PPV) |
| 5. Biophysical screening, uterine artery Doppler waveform. Uterine artery pulsatility index (UTPI) is also being used. | Diastolic notch in umbilical arteries at 24 weeks in women destined to develop pre-eclampsia. Useful, but limited diagnostic accuracy in predicting pre-eclampsia. Uterine artery pulsatility Index (PI) > 90th percentile for gestational age. |
| 6. Urinary biochemical tests<br>(a) Hypocalciuria<br>(b) Urinary albumin creatinine ratio<br>(c) Urinary kallikrein creatinine ratio<br>(d) Microalbuminuria | Present<br>Increased } Decreased sensitivity<br>Increased<br>Present |
| 7. Human chorionic gonadotropin (HCG) | Increased |
| 8. Maternal serum alpha fetoprotein (MSAFP) | Increased |
| 9. Placental growth factor (PLGF) | Decreased |
| 10. Inhibin-A and Activin A | Increased |
| 11. Bio-markers of insulin resistance<br>(a) Leptin<br>(b) Tumor necrosis factor–α (TNF-α)<br>(c) Sex hormone binding globulin (SHBG) | <br>Increased<br>Increased<br>Decreased |
| 12. Fibronectin | Increased |
| 13. Plasma corticotropin releasing hormone (CRH) | Increased |
| 14. Homocysteine | Increased |
| 15. Adhesion molecules (vascular cell adhesion molecules or VCAM) | Increased |
| 16. Neurokinin B | Increased |
| 17. Soluble forms like Tyrosine Kinase-1 (SFLT1), asymmetric dimethylarginine in second trimester | Increased |
| 18. Placental protein '13' | Decreased |
| 19. PAPP-A | Decreased, useful when used in combination |

**Table 32.6B: FOGSI Gestosis score as per Risk factors**

| Risk factor | Score | Risk factor | Score |
|---|---|---|---|
| Age older than 35 years | 1 | Maternal hypothyroidism | 2 |
| Age younger than 19 years | 1 | Family history of pre-eclampsia | 2 |
| Maternal anaemia | 1 | Gestational diabetes mellitus | 2 |
| Obesity (BMI > 30) | 1 | Obesity (BMI > 35 kg/M$^2$) | 2 |
| Primigravida | 1 | Multifetal pregnancy | 2 |
| Short duration of sperm exposure (cohabitation) | 1 | Hypertensive disease during previous pregnancy | 2 |
| Woman born as small for gestational age | 1 | Pregestational diabetes mellitus | 3 |
| Family history of cardiovascular disease | 1 | Chronic hypertension | 3 |
| Polycystic ovary syndrome | 1 | Mental disorders | 3 |
| Inter pregnancy interval more than 7 years | 1 | Inherited/Acquired thrombophilia | 3 |
| Conceived with Assisted Reproductive (IVF/ICSI) Treatment | 1 | Maternal chronic kidney disease | 3 |
| MAP > 85 mm of Hg | 1 | Autoimmune disease (SLE/APLAS/RA) | 3 |
| Chronic vascular disease (Dyslipidemia) | 1 | Pregnancy with Assisted Reproductive (OD or Surrogacy) Treatment | 3 |
| Excessive weight gain during pregnancy | 1 | | |

I. Marker useful for screening of pre-eclampsia
First Trimester marker: 1. MAP
2. UTPI
3. PLGF
4. PAPP-A
Second and third trimester markers: 1. MAP
2. UTPI
3. PLGF
4. SFLT-1

II. Foetal Medicine Foundation (2016) has developed a tool for "Assessment of Risk of Pre-eclampsia" by Bayes based method using combination of maternal factors and biomarkers in which information about index patient like maternal characteristics, pregnancy dating, medical history, obstetric history are recorded. Alongwith biophysical measurement, mean arternal pressure (MAP), mean uterine artery pulsatility index (UTPI) are added. Alongwith biochemical measurements like placental like growth factor (PLGF) and pregnancy associated plasma-protein-A (PAPP-A) in first trimester and PLGF and sFlt-I in second and third trimester are added. The system than calculates the risk of development of pre-eclampsia. It is considered low risk (< 1 : 100) and these patients are given routine antenatal care. High Risk Patients (> 1 : 100) are started with Aspirin 150 mg at night from < 12 weeks upto 36 weeks.




Quidel triage PlGF Test (ALERE)

sFlt-I levels are increased and PIGF levels are decreased in pre-eclampsia. The levels are correlated with severity and are used for clinical management and decision making. sFlT-I/PIGF ratio is used for prediction and monitoring of pre-eclampsia.
As per the NICE guidelines of 2017- PlGF based screening has been recommended between 20–34 + 6 weeks for suspected pre-eclampsia.

**LEVEL 1 CARE**

BP up to 149/99 mmHg
- Do not admit to hospital
- BP up to 149/99 mmHg
- Do not treat hypertension
- Measure BP no more than ×1/wk
- Test for proteinuria at each visit
- Carry out routine antenatal blood tests.
- If presenting before 32/40 or at high risk of pre-eclampsia, test for proteinuria and measure BP × 2/wk

| | |
|---|---|
| PLGF < 12 very low | Admit for assessment |
| PLGF low 12–100 | Move to level 2 care |
| PLGF > 100 Normal | Continue with level 1 care |

**LEVEL 2 CARE**

Moderate hypertension
(BP 150/100–159/109 mmHg)
- Do not admit to hospital
- Treat hypertension to keep BP < 150/80–100 mmHg.
- Measure BP at least × 2/wk.
- Test for proteinuria at each visit
- Test kidney function, electrolytes, FBC, transminases, bilirubin
- No further blood tests if no subsequent proteinuria

| | |
|---|---|
| PLGF < 12 very low | Admit for assessment |
| PLGF low 12–100 | Continue with level 2 care |
| PLGF > 100 Normal | Return to level 1 care |

**LEVEL 3 CARE**

Severe hypertension
(BP ≥ 160/110 mmHg)
- Admit to hospital until BP ≤ 159/109 mmHg and treat hypertension to keep BP
- < 150/80–100 mmHg
- Do not treat hypertension
- Measure BP at least × 4/wk
- Test for proteinuria daily
- Test kidney function, electrolytes, FBC, transminases, bilirubin at presentation and then weekly

| | |
|---|---|
| PLGF < 12 very low | Admit for assessment |
| PLGF low 12–100 | Admit for BP control then level 2 care |
| PLGF > 100 Normal | Continue with level 1 care |

Management as per PlGF based screening as per the PARROT Trial 2019

## PREVENTION OF PRE-ECLAMPSIA

### Primary Prevention

Primary prevention which means avoiding occurrence of a disease may be possible to some extent by modification of some of the risk factors of pre-eclampsia listed in Table 32.3. Hence, it is recommended to have pregnancies with same partner and to have children at an age when the endothelium is still able to cope with the inflammatory stress associated with pregnant state. Similarly, prevention or effective treatment of obesity or both can result in a significant reduction in the frequency of pre-eclampsia and should be encouraged. Similarly, women with diabetes, chronic hypertension, renal and other medical disorders should have their primary condition under control before conceiving.

Although cigarette smoking is associated with a 30–40% reduction in the risk of pre-eclampsia, possibly through inhibition of interleukin-2 and tumour necrosis factor by nicotine, it cannot be recommended due to substantial harmful effects of smoking on maternal and foetal health. However, using the relative risk of pre-eclampsia in high-risk situations, early diagnosis of pre-eclampsia with timely referral for hospital care can help in their timely management and can prevent them from going into severe pre-eclampsia.

#### Secondary Prevention

Secondary prevention means breaking off the disease process before emergence of obvious clinical disease. The various prevention modalities are shown in Table 32.7 but are not effective. ACOG (2019) recommends low dose aspirin (150 mg) from 12 to 28 week of pregnancy is women at high risk of developing pre-eclampsia (with one or more risk factor shown in table 32.3). ACOG (2019) also recommends calcium supplementation in high risk women with baseline deficit of calcium in doses of 1.2 to 2 gm per day.

#### Conclusion

To date, no effective modality exists for prevention of pre-eclampsia.

## CLINICAL FEATURES

Pre-eclampsia occurs more often in primigravidas and in presence of other high risk factors given in Table 32.6A & B. The onset is usually insidious with slow course of the syndrome. Occasionally, however, there can be acute onset with rapid course of the disease.

### Symptoms

#### Mild symptoms

1. There may be no symptoms in early stages.
2. Mild swelling over ankles especially on getting up from bed in the morning with tightness of the ring on the fingers or toes can be the early symptom.
3. There can be excessive swelling involving feet (Fig. 32.3A), face, abdominal wall, vulva or even the whole body (generalized anasarca) (Fig. 32.3B).

#### Serious Symptoms

Serious symptoms are usually associated with acute onset of the disease.

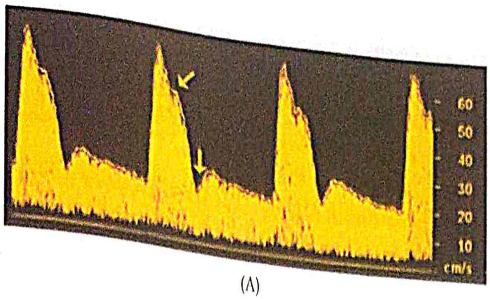
1. **Headache.** Especially in occipital or frontal area
2. **Sleep disturbances**
3. Decreased urine output especially less than 500 mL in 24 hours is a serious symptom.
4. **Epigastric or right hypochondriac pain** is a sinister symptom. There can be associated coffee colour vomiting due to hemorrhagic gastritis. Right hypochondriac pain can be due to stretching of Glisson's Capsule or due to subcapsular haemorrhage in liver.
5. **Visual symptoms.** There may be blurring or diminution of vision or rarely complete blindness due to spasm of retinal vessels, damage in occipital lobe or due to retinal detachment.

### Signs

1. Excessive weight gain especially a rapid gain of more than one pound (half kg) a week or 4 pounds (2 kg) a month in later months of pregnancy is significant.
2. **Edema:** Although not pathognomonic of pre-eclampsia, most women with the disease have visible edema over ankles, especially on rising from bed in the morning (Fig. 32.3B & C). Sudden and generalized edema usually heralds imminent eclampsia.
3. Rise in blood pressure to more than 140 mm Hg systolic and or more than 90 mm Hg diastolic is a diagnostic feature of pre-eclampsia. Blood pressure of more than 160 mm Hg systolic and or more than 110 mm Hg diastolic is diagnostic of severe pre-eclampsia.
4. There may be features of pulmonary edema due to leaky capillaries and low oncotic pressure manifesting as audible crepitations at bases of lungs.
5. Brisk tendon reflexes and ankle clonus
6. Abdominal examination may demonstrate evidence of placental insufficiency like oligohydramnios and foetal growth restriction.

## INVESTIGATIONS

1. **Complete hemogram with platelet levels.** Due to hemoconcentration, there may be raised Hb and hematocrit levels. Low platelets (< 100,000/cm$^3$) are significant and may demonstrate HELLP syndrome.
2. **Coagulation profile.** Bleeding time, clotting time and other coagulation studies are useful for diagnosis of severe disease, HELLP syndrome and their normal value is a pre-requisite for giving regional anesthesia.
3. **Renal function tests**
   (a) **Uric acid**, a measure of renal tubular function, has been used to assess the disease severity in the past. Levels more than 4.5 mg/dL indicate pre-eclampsia. However, recent studies do not demonstrate its clinical usefulness as severe disease




(A)



(B)



(C)

**Fig. 32.3:** (A) Doppler of uterine artery at 16 weeks pregnancy showing diastolic notch (Arrow) and end diastolic flow. Patient later developed pre-eclampsia; (B) Clinical photograph of a woman with pre-eclampsia with marked pedal edema; (C) Clinical photograph of a woman of severe pre-eclampsia with edema all over her body (anasarca). Kind courtesy: Prof Manju Puri, LHMC, New Delhi for (Fig. 32.3C)

**Table 32.7: Various Prevention Modalities for Pre-eclampsia**

1. Bed rest: Not found effective.
2. Dietary sodium restriction: No convincing evidence of its efficacy.
3. Dietary protein and energy intake: No benefit
4. Dietary supplementation with fatty acids: No benefit
5. Control of obesity: Weight reduction not recommended during pregnancy even in obese women
6. Fish oil: Unlikely to benefit in prevention of pre-eclampsia
7. Alcohol intake: Excessive consumption can cause or aggravate maternal hypertension.
8. Diets high in trans-fatty acids associated with decreased incidence of pre-eclampsia (preliminary study)
9. Antihypertensives like methyldopa, labetalol and atenolol evaluated in randomized trials for their efficacy in prevention of superimposed pre-eclampsia. No reduction in incidence of pre-eclampsia found.
10. Role of diuretics not recommended due to risks.
11. Zinc, magnesium, folic acid; no role
12. No definite scientific evidence that any of B complex, vitamins can prevent pre-eclampsia
13. Aspirin: In low dose it inhibits cyclo-oxygenase in platelets inhibiting formation of thromboxane $A_2$ without interfering with prostacyclin generation. *The only women in whom the use of low dose aspirin may be justified are those at especially high risk of developing early onset pre-eclampsia as starting early aspirin has been found to have a favourable effect on prostanoids in these women.* The dose is 150 mg per day starting from 11–14 weeks till 36 weeks. Recent studies have found that starting low dose aspirin at 12 weeks is more effective in prevention of pre-eclampsia. It could reduce incidence of preterm pre-eclampsia (but not term pre-eclampsia) by 62% (ASPRE Trial NEJM 2017). ACOG (2019) also recommends low dose aspirin (100 mg) between 12 to 28 weeks of pregnancy to women at high risk of developing pre-eclampsia.
14. No sufficient data to support the role of heparin and nitric oxide donors.
15. Calcium supplementation in dosage of 1.5–2 gram/day has been found to be useful in prevention of pre-eclampsia in communities where there is deficiency of calcium intake in diet (e.g. India). But larger studies have found it of no use in prevention of pre-eclampsia.
16. Initial evidence supported the use of antioxidants, Vitamin C in dose of 1000 mg with Vitamin E (400 IU) or lycopene 2 mg twice a day starting from 20th week of pregnancy until term. However, recent larger trials have not found antioxidants useful in prevention of pre-eclampsia.

can occur with normal uric acid levels.

(b) **Blood urea** may remain normal or slightly raised.

(c) **Serum creatinine levels** may be more than 1 mg/dL. Raised urea and creatinine are associated with renal involvement, but are not useful as an early indicator of disease severity. Renal function tests are done when the diagnosis is made and are repeated as and when necessary (usually weekly).

4. **Liver function tests.** With severe pre-eclampsia there can be alterations in hepatic function tests. There is usually an increase in serum alkaline phosphatase. In HELLP syndrome, liver enzymes like aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels are raised indicating hepatocellular damage and severe disease.
5. **Urine examination.**
   (a) Urine examination for proteinuria both qualitative and quantitative is performed over a 24 hours specimen. A loss of more than 300 mg (2+ on dipstick) or more in 24 hours is diagnostic of pre-eclampsia. Quantification of proteinuria is no longer used for assessment of severity of pre-eclampsia.
   (b) Urine microscopic examination for hyaline casts or epithelial cells is useful.
6. **Ophthalmoscopic examination** (Fundoscopy) by an ophthalmologist is useful for observing retinal edema, constriction of the arterioles and other fundal findings as shown in Table 32.5. It helps in grading the severity of the disease.
7. **Foetal monitoring**
   (a) Foetal kick chart. There should be at least 10 movements per day.
   (b) Clinical examination should be done for foetal growth, presentation and well-being.
   (c) Non-stress testing twice a week
   (d) Cardiotocography
   (e) Ultrasound examination should be done every two weeks for foetal growth, well-being and amount of amniotic fluid. Biophysical profile (Manning's score) is done for non-reactive NST. Doppler studies for umbilical artery blood flow can be done.

## COMPLICATIONS OF PRE-ECLAMPSIA

Complications occur more often if the patient is left untreated.

### I. Maternal

**1. During Pregnancy**

(a) Eclampsia is seen in 0.05 to 0.5% cases, more so in acute cases
(b) Abruptio placentae
(c) Oliguria
(d) Anuria
(e) Eye complications: Diminution of vision and even blindness
(f) Preterm labour
(g) Cerebral haemorrhage
(h) Coagulation failure and DIC
(i) HELLP syndrome
(j) Adult respiratory distress syndrome (ARDS)

**2. During Labour**

(a) Eclampsia
(b) Postpartum haemorrhage which may be due to atony of the uterus or coagulation failure.
(c) Increased operative delivery
(d) Death

**3. During Puerperium**

(a) Eclampsia usually occurs in first 48 hours.
(b) Postpartum shock which could be due to PPH or vasomotor collapse.
(c) Infection due to increased chances of operative delivery and induction of labour.
(d) Death.

**4. Remote Complications**

Pre-eclampsia is a screening test for future health as many long term complications can occur in them in future. The future complications are as follows:

(a) **Recurrent pre-eclampsia** up to 40% for early onset pre-eclampsia (before 32 weeks) and is 25% for late onset pre-eclampsia (after 32 weeks). Low dose aspirin (150 mg) and calcium supplementation may be advised in subsequent pregnancy.
(b) **Chronic hypertension.** Up to 40% patients especially with early onset pre-eclampsia may develop chronic hypertension (4-fold increase). It may be related to underlying thrombophilias or antiphospholipid syndrome. Hence, close follow-up is necessary.
(c) **Chronic nephritis.** There is a high incidence of chronic renal disease in women with pre-eclampsia (up to 50%).
(d) **Increased cardiovascular morbidity** like ischemic heart diseases (2 fold increase) venous thromboembolism (2 fold increase) and stroke (2 fold increase) in later life
(e) **All cause mortality; 1.5 fold.**

### II. Foetal Complications

1. Intrauterine (foetal) growth restriction (IUGR) due to chronic placental insufficiency (10–25%).
2. Intrauterine asphyxia
3. Intrauterine foetal death due to varying degrees of uteroplacental insufficiency, accidental haemorrhage or acute red infarction.
4. Prematurity either due to spontaneous onset of labour due to accidental haemorrhage or iatrogenic due to induction of labour.
5. Oligohydramnios
6. Placental infarction

### Perinatal Mortality

The perinatal mortality is high being 30% in developing countries mainly due to pre-maturity (spontaneous and iatrogenic), hypoxia and drug effects.

## MANAGEMENT

The objectives of management are as follows:

1. Delivery of baby with least possible trauma to mother and fetus.
2. Birth of a newborn who subsequently lives.
3. Prevention of maternal complications like cerebrovascular accidents, renal failure, pulmonary edema and placental abruption.
4. Prevention of convulsions to avoid maternal and perinatal mortality.
5. Complete maternal health restoration

Diagnosis of pre-eclampsia must be confirmed before treating it. Its management depends upon the following factors.

(a) Severity of pre-eclampsia as determined by various factors as shown in Table 32.2.
(b) Gestational age
(c) Condition of the cervix (Bishop's score)
(d) Maternal condition
(e) Foetal condition

## MANAGEMENT OF PRE-ECLAMPSIA WITHOUT SEVERE FEATURES (MILD PRE-ECLAMPSIA)

Management of pre-eclampsia is shown in Flow Chart 32.1.

Ideally all patients with pre-eclampsia (especially newly diagnosed or persistent or worsening hypertension or proteinuria) should be admitted to hospital at least initially for systematic evaluation which includes the following.

1. Detailed history should be noted to rule out any symptoms of severe disease like headache, visual disturbances, epigastric pain and rapid weight gain.
2. Detailed clinical examination should be done for any edema, brisk reflexes and clonus, chest auscultation for basal crepts and abdominal examination for foetal growth and well-being.
3. Maternal weight should be checked on admission and regularly thereafter.
4. Quantitative estimation should be done for proteinuria on admission and twice weekly thereafter (Not needed once diagnosis of pre-eclampsia is made).
5. Blood pressure measurement with right-sized cuff in the sitting position every 4 hourly, except between midnight and morning.
6. Regular blood tests (weekly for mild hypertension, twice weekly for severe hypertension) should be done for hematocrit, platelets, serum uric acid, creatinine, blood urea and liver enzymes.
7. Weekly ophthalmological examination (fundus) should be performed for retinal changes.
8. Regular evaluation of foetal size and amniotic fluid volume clinically (daily) and ultrasonographically once or twice weekly. If history, examination or investigations point to the diagnosis of severe pre-eclampsia then the management should be altered accordingly.

### Treatment of pre-eclampsia without severe features (formerly Mild Pre-eclampsia)

1. **Rest.** In olden days, bed rest in left lateral position was recommended by most clinicians on the presumption that it decreased the effects of venacaval compression, increased the renal blood flow causing diuresis, increased uterine blood flow and placental perfusion and that it reduced the blood pressure. The current view is that absolute bed rest is not necessary. However, reduced physical activity throughout much of the day is helpful.
2. **Dietary intake of calories and proteins.** Adequate (about 100 g proteins and 1600–2000 calories per day) but not excessive proteins and calories should be given in diet.
3. **Fluid and salt intake.** Usual fluid and salt intake are advised. It should not be restricted or forced.
4. **Sedation.** In olden days mild sedation in the form of diazepam 5 mg or phenobarbitone 60 mg was often prescribed to women suffering from mild pre-eclampsia to calm them and to give them adequate sleep. Current concept, however, is that sedatives and tranquilizers are not prescribed routinely for these women.
5. **Diuretics.** Diuretics are to be avoided as they can have adverse effect on the fetus by causing electrolyte imbalance and by reducing placental perfusion. They can also cause rise in blood urea, uric acid and neonatal thrombocytopenia.

   **Indications of using diuretics.** Diuretics are only given when there are the following indications.
   (a) Pulmonary edema
   (b) Heart failure
   (c) Generalized anasarca (massive edema) causing discomfort to the patient and which does not diminish on rest.
   (d) Sometimes with antihypertensive drugs like diazoxide in women with hypertension and generalized edema. Furosemide in dose of 40 mg per day orally is given.
6. **Role of antihypertensive drugs.** Role of antihypertensive drugs in mild to moderate hypertension is controversial and is not recommended. Main benefit of drug therapy in mild to moderate hypertension is to decrease the rate of progression to severe hypertension.

| | When to start? | Target BP |
|---|---|---|
| ACOG 2019 | SBP > 160 mmhg<br>DBP > 110 mm hg | < 140/90 mmhg |
| NICE 2019 | SBP > 140 mm hg<br>DBP > 90 mm hg | < 135/85 mmhg |

Antihypertensives: They are shown in Table 32.8.

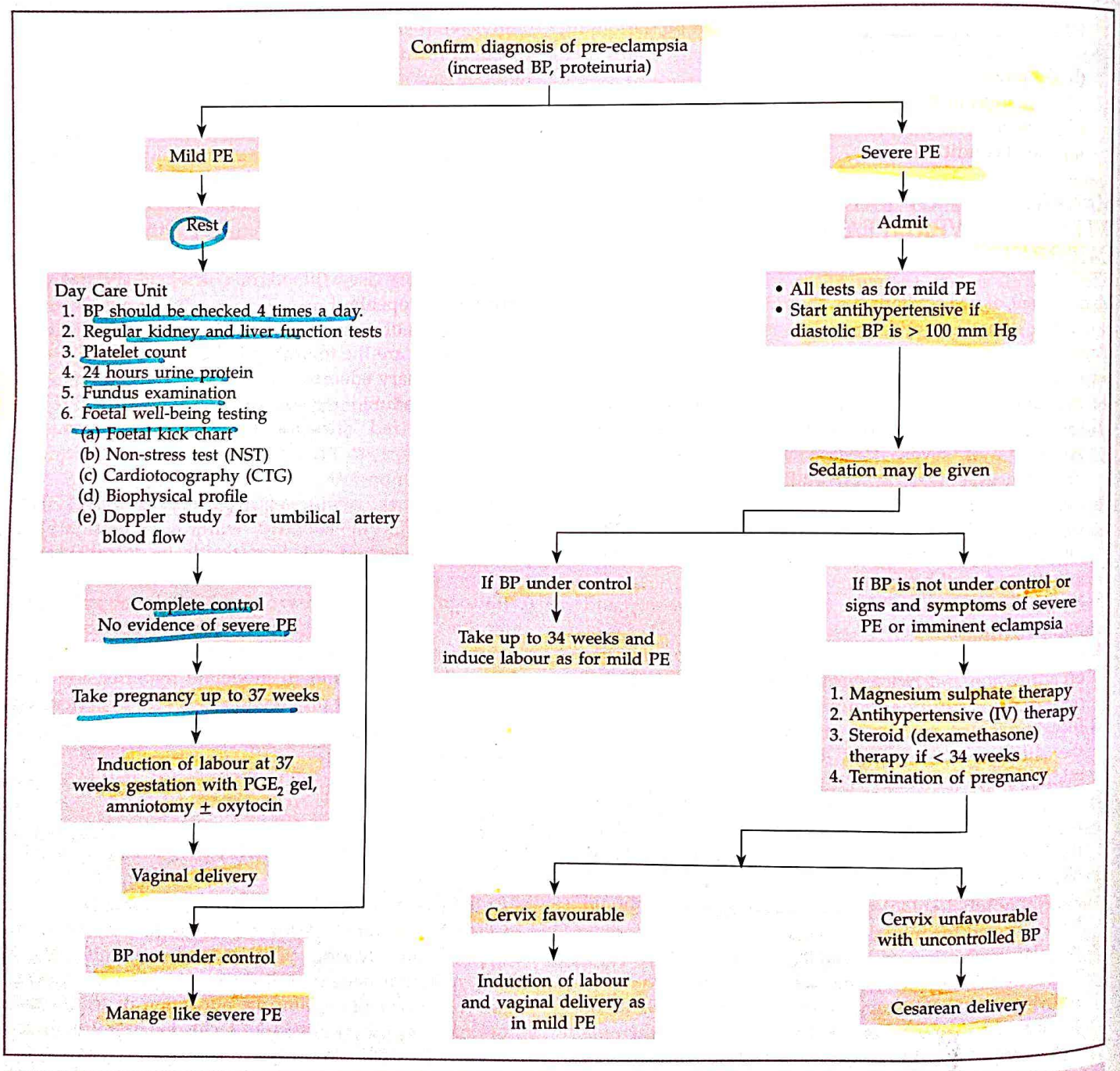
***Current opinion.*** Treatment of mild to moderate hypertension during pregnancy has very small, if any, maternal benefit as it does not appear to prevent superimposed pre-eclampsia, abruption or to decrease perinatal mortality. In fact, it can be harmful to the fetus in the following ways.

(a) It can cause foetal growth restriction.
(b) More premature iatrogenic intervention

(c) Increased neuro developmental abnormalities in babies
(d) Higher frequency of cardiovascular disease and insulin resistance syndrome in adult life.

Antihypertensive drugs can be given to women with a history of previous perinatal loss or with evidence of target organ damage as they now belong to high risk category. The antihypertensive drugs are given as in management of severe pre-eclampsia (see below).

7. **Role of Day Care Unit.** If the patient's blood pressure remains stable and less than 100 mm Hg diastolic without significant proteinuria and she does not have any signs of worsening of her condition, she can be discharged from the hospital. She can rest at home and keep foetal movement count chart. She should get her blood pressure checked everyday and should be evaluated in hospital day care unit every week for maternal and foetal well-being as is popular in western countries (see Chapter 40 for details). She should be counselled about symptoms of worsening of the condition (severe pre-eclampsia) like severe or persistent headache, visual disturbances, epigastric pain, vomiting, any bleeding per vaginum (abruptio placentae) for which she should report to the hospital staff immediately as she now belongs to severe pre-eclampsia category.
8. **Maternal monitoring.** Frequency of monitoring
(a) Blood pressure: Four hourly (except after midnight)




**Flow Chart 32.1:** Management of Pre-eclampsia

**Table 32.8: Commonly used antihypertensives in pregnancy**

| Drug (FDA cagegory) | Dosage | MOA (Mechanism of action) | Adverse Effects | Comments |
|---|---|---|---|---|
| Methyldopa (B) | 250 mg PO TDS<br>Max: 2 g/d | Centrally acting<br>K2 agonist | Dry mouth, somnolence, postural hypotension. Post partum depression | Not very effective in severe hypertension<br>Childhood safety data available |
| Labetalol (C) | 100 mg bd<br>Max: 2400 mg/d | Combined α and β blocker | Discontinue at signs of liver dysfunction, asthma, CHF, Tachy cardia | Ist line agent<br>Fewer side effects<br>Not associated with IUGR |
| Nifedipine (C) | 10–30 mg PO TDS<br>SR tab: 10–20 mg bd<br>Max: 90–120 mg/d | Dihydropyridine CCB-vasodilatation by smooth muscle relaxation | Headache, edema, allergic hepatitis<br>Reflex tachycardia, Hypotension | |
| Atenolol (C) | 50–100 mg PO od | β1 blocker | IUGR masks symptoms of acute hypoglycemia | |

PO: per oral; TDS: Thrice daily; BD: twice daily; SR: sustained release; IUGR: intrauterine growth restriction

(b) Urine proteinuria: Daily
(c) Body weight: Weekly
(d) Biochemical blood tests: Weekly
(e) Any worsening of symptoms

9. **Foetal monitoring**
   (a) Daily foetal movement chart (at least 10 per day)
   (b) Non-stress test: Weekly
   (c) Ultrasound for foetal growth and amniotic fluid: Every fortnight
   (d) Uterine artery doppler studies: weekly
10. **Management of labour.** Patients with controlled mild to moderate hypertension may be allowed to await spontaneous labour and induction after 37 weeks.

**Delivery goals:** As per the FOGSI GCPR 2019-

**Gestational Hypertension:** Pregnancy can be continued till term

**Mild Pre-eclampsia:** To be delivered at 37 completed weeks.

**Severe Pre-eclampsia:** To be delivered after 34 completed weeks.

**Eclampsia:** Should be delivered once mother is stabilized after MgSO4.

(a) **Role of induction of labour.** In controlled mild hypertension, induction can be planned at 37 weeks. If cervix is favourable (Bishop's score > 6), low rupture of membranes (amniotomy) followed by oxytocin infusion is used. If cervix is unfavourable (Bishop's score < 6), prostaglandin $E_2$ gel vaginally or intracervically is inserted for ripening the cervix.

(b) **Indications for early delivery.**
   (i) Worsening of hypertension and/or signs of vital organ involvement
   (ii) If there is intrauterine growth restriction or foetal distress, then early induction of labour is advised.

(c) **Intrapartum care.** Regular monitoring of maternal condition, pulse, blood pressure and urine output. Continuous cardiotocography or intermittent auscultation is performed for foetal monitoring. Vaginal delivery is the aim. Cesarean delivery is to be done for strict obstetric indications. Active management of third stage of labour with ergometrine is relatively contraindicated. Oxytocin 5–10 units intramuscularly or intravenously should instead be used.

## MANAGEMENT OF PRE-ECLAMPSIA WITH SEVERE FEATURES (FORMERLY SEVERE PRE-ECLAMPSIA)

1. Hospitalization in a tertiary centre hospital is required for all patients.
2. Bed rest
3. Daily maternal monitoring for vitals
4. **Antihypertensive treatment.** The goal of this therapy is to keep the diastolic BP between 90 and 100 mm Hg to prevent maternal complications especially cerebral haemorrhage. Methyldopa in dose of 250 mg thrice a day is started initially and increased up to a maximum 500 mg four times a day (2 g/day). Labetalol 100–200 mg 8 hourly can be given. Nifedipine 10 mg 4–6 hourly can also be given.
5. **Corticosteroid administration** (if < 34 weeks of pregnancy). Dexamethasone 6 mg intramuscularly 6 hours apart for four doses for foetal pulmonary maturity.
6. Urine protein testing twice a week
7. Liver function tests, renal function tests, hematologic evaluation daily or on alternate days (as mentioned in mild pre-eclampsia)
8. Daily foetal movement count
9. Daily non-stress test
10. Ultrasound weekly for amniotic fluid volume and uterine artery Doppler
11. Ultrasound every two weeks for foetal growth
12. Look for signs and symptoms of impending eclampsia.

### Indications for Delivery

1. Worsening of maternal condition

2. Foetal compromise
3. Completion of 34 weeks of gestation
4. Premature labour

**Expectant Management for Severe Pre-eclampsia**

Although definitive treatment of pre-eclampsia, especially severe pre-eclampsia and eclampsia, is delivery of the fetus, expectant management in women remote from term has been tried for better foetal outcome without compromising maternal condition. It is especially useful between 28 and 34 weeks of gestation in a tertiary care hospital with intensive maternal and foetal monitoring. Corticosteroids for foetal maturity are given. Antihypertensive drugs are given. Prophylactic anticonvulsant therapy with magnesium sulphate may be required. A few randomized controlled trials using expectant management of severe pre-eclampsia have observed prolongation of pregnancy and better neonatal outcome. However, it is still experimental and cannot be used routinely.

## Management at 34 Weeks Gestation

Termination of pregnancy is the treatment of choice in severe pre-eclampsia if gestational age is 34 weeks or more. First, her general condition should be stabilized.

1. Start antihypertensive therapy (see below in severe pre-eclampsia).
2. Prophylactic anticonvulsant treatment using magnesium sulphate during labour and 24 hours postpartum. The American College of Obsetricians and Gynecologists (2002) recommends that magnesium sulphate should be given prophylactically to patients with severe pre-eclampsia. The Magpie (Magnesium sulphate for Prevention of Eclampsia) Trial (2002) a randomized controlled trial (RCT) on > 10,000 women of severe pre-eclampsia in 33 countries was the largest trial for pre-eclampsia. Primary outcome was eclampsia and death of baby (including stillbirths), secondary outcomes were serious maternal morbidity, toxicity and other side effects, complication of labour and neonatal morbidity. It recommends magnesium sulphate therapy for severe pre-eclampsia as they observed 58% reduction in incidence of eclampsia with magnesium sulphate as compared with placebo with no significant harmful effects of magnesium sulphate on mother and fetus.
3. **Intravenous hydration.** The intravenous fluid must be carefully monitored as there is a risk of pulmonary edema. Ringer lactate or colloids should be used. Central venous pressure line may be used keeping it up to 4 mm Hg.
4. Urine output is carefully monitored using Foley's catheter.
5. Hematocrit, coagulation profile and renal and liver function tests to be done.
6. Administration of blood, platelet concentrates and fresh frozen plasma, if there is coagulopathy.
7. Expedition of delivery

## Management of Labour in Severe Pre-eclampsia

Vaginal delivery is the aim. Cesarean section is to be done only for obstetric indications or unfavourable cervix with uncontrolled BP. Induction of labour is performed depending upon cervical effacement (Bishop's score) as in mild pre-eclampsia using amniotomy and oxytocin drip or prostaglandin $E_2$ gel. In case of spontaneous labour, oxytocin infusion may be given for augmentation of labour. Close monitoring of the patient, foetal heart and uterine activity as in mild pre-eclampsia is performed.

## Indications of Delivering Women with Severe Pre-eclampsia for expedient delivery (within 72 hrs.)

**I. Maternal indications**

1. Uncontrolled hypertension with BP > 160/110 mm Hg despite maximum dose of two anti-hypertensive drugs.
2. Eclampsia
3. Thrombocytopenia (platelets < 100,000/mm$^3$)
4. Pulmonary edema
5. Acute renal failure
6. Persistant severe headache or visual changes
7. Elevated liver enzymes with persistent severe epigastric pain or right upper quadrant tenderness.
8. Labour or rupture of membranes.
9. Abruptio placentae

**II. Foetal indications**

1. Foetal distress (repetitive severe variable or late decelerations).
2. Biophysical profile ≤ 4 on 2 occasions 4 hrs. apart.
3. Amniotic fluid index (AFI) < 5 cm
4. FGR or IUGR (EFW on USG < 5th percentile)
5. Reversed umbilical artery end diastolic flow
6. Foetal death
7. ≥ 34 weeks period of gestation POG

Epidural analgesia can be given if coagulation profile is normal.

*Routine prophylactic forceps are not applied these days but a tendency of prolongation of second stage should be curtailed by forceps or ventouse.* Careful vigilance for any postpartum haemorrhage as these women tolerate even moderate PPH poorly due to contracted blood volume. Hence, blood should always be kept crossmatched for labour. Active management of third stage of labour with ergometrine is contraindicated for the fear of worsening of hypertension. However, oxytocin, can be administered as 5–10 units intramuscularly.

Women with severe pre-eclampsia should be closely observed for at least 24 hours after delivery as eclampsia can develop. Antihypertensives and anticonvulsants are to be continued.

**Postnatal care.** The patient is discharged after her general condition has improved and her blood pressure is under control. She is assessed after 6 weeks in puerperium for hypertension and proteinuria. If hypertension persists, then she should be referred to a physician for evaluation and treatment. She should use contraception to avoid next pregnancy. Oral pills and intrauterine device are safe. Postpartum ligation is avoided for the fear of thrombo-embolism but interval ligation can be performed. Next

pregnancy should only be allowed once her renal status is normal and blood pressure is under control. She should report to hospital in next pregnancy. There is risk of recurrence of pre-eclampsia in subsequent pregnancy (up to 25%) depending upon severity and timing of pre-eclampsia in first pregnancy.

## IMMINENT (IMPENDING) ECLAMPSIA (ACUTE FULMINANT PRE-ECLAMPSIA)

Imminent eclampsia is defined as the sudden worsening in the condition of a diagnosed case of pre-eclampsia. There is a real danger of developing eclampsia necessitating urgent care. It is marked by appearance of severe symptoms such as severe headache, restlessness, vomiting, epigastric pain, visual disturbances and presence of signs like brisk reflexes with clonus and sudden and generalised persistent edema. Disturbed sleep and oliguria (< 400 ml/24 hr) are also signs of it.

**Management**

Management of imminent eclampsia is like eclampsia (given below).

### Hypertensive Emergency (ACOG 2019)

- Two severe BP values (> 160/110) taken 15 minutes apart which can occur antepartum, intrapartum or postpartum WHO (2018) has also recommended use of antihypertension agents in severe hypertension in pregnancy **(strong recommendation)**

  Management
  - First line drugs - Intravenous (I.V.) labetalol, intravenous hydralazine; oral nifediping
  - $MgSO_4$ should be used for seizure prophylaxis.

  Treatment is shown in Flow Chart 32.2 to 32.4.

## ECLAMPSIA

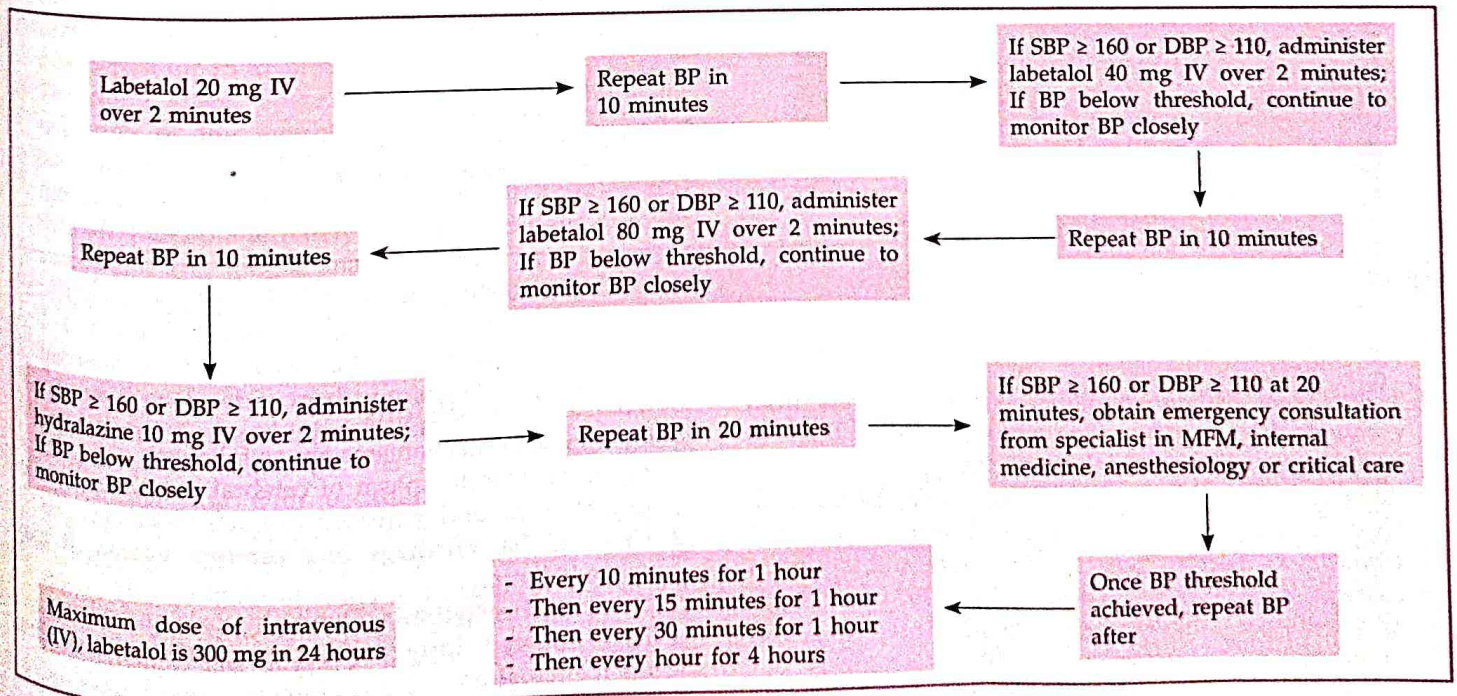
The word eclampsia is derived from Greek word which means 'flashes of light'. Eclampsia is the occurrence of generalized tonic clonic convulsions (seizures) or coma in a woman with pre-eclampsia which cannot be attributed to other causes. It is usually seen in women having signs and symptoms of severe pre-eclampsia or imminent eclampsia like very high blood pressure, severe proteinuria, generalized edema, headache, dizziness, visual disturbances and epigastric pain. Maternal mortality is 4–6% and perinatal loss is up to 45% in eclampsia in India (FOGSI **Consensus Statement, 2008, JOGI 2016).** Eclampsia is responsible for 12% maternal deaths globally. Incidence of eclampsia is 1 in 1500 to 1 in 2000 pregnancies globally, being 1.5 to 10 per 10,000 deliveries in high income countries (2019) but may be as high as 1.5% (0.2 to 5%) of all pregnancies in India (FOGSI GCPR 2019). It is a preventable disease by efficient use of antenatal care as has been seen in western countries.

### Etiopathogenesis

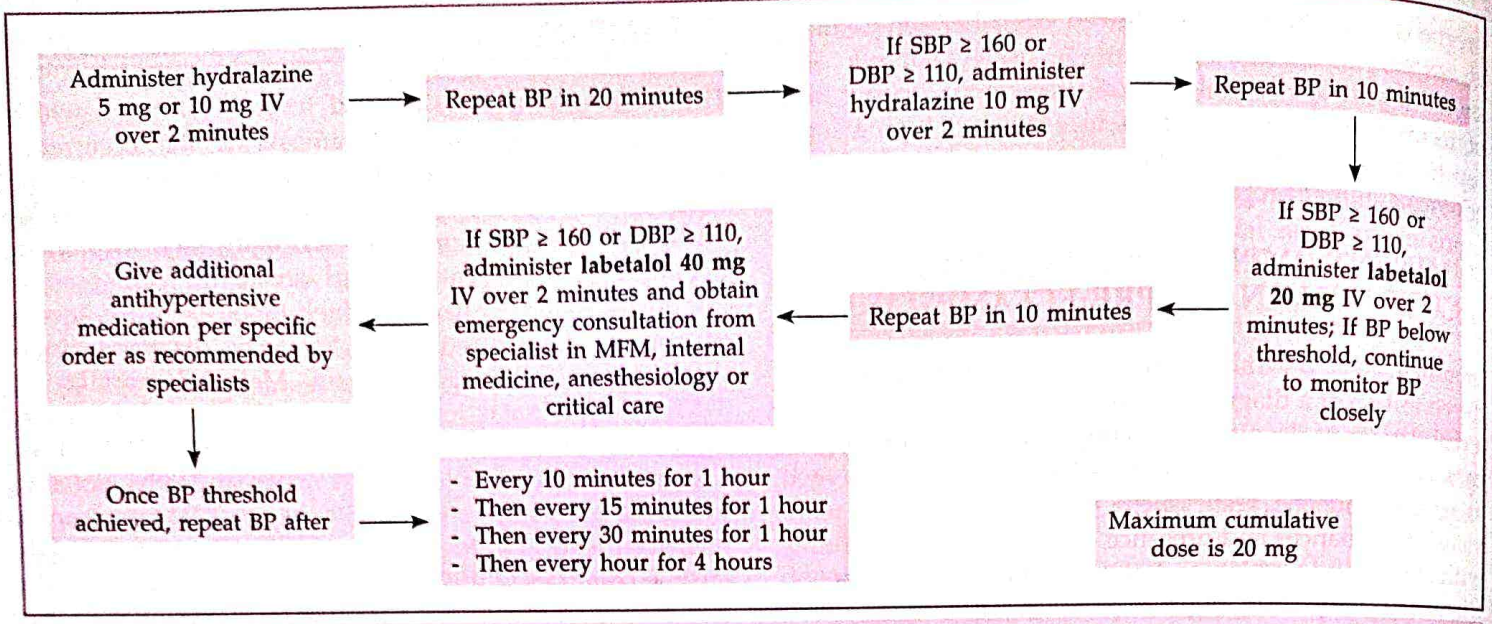
The etiopathogenesis of eclampsia is the same as pre-eclampsia and has been described above.

### Types of Eclampsia

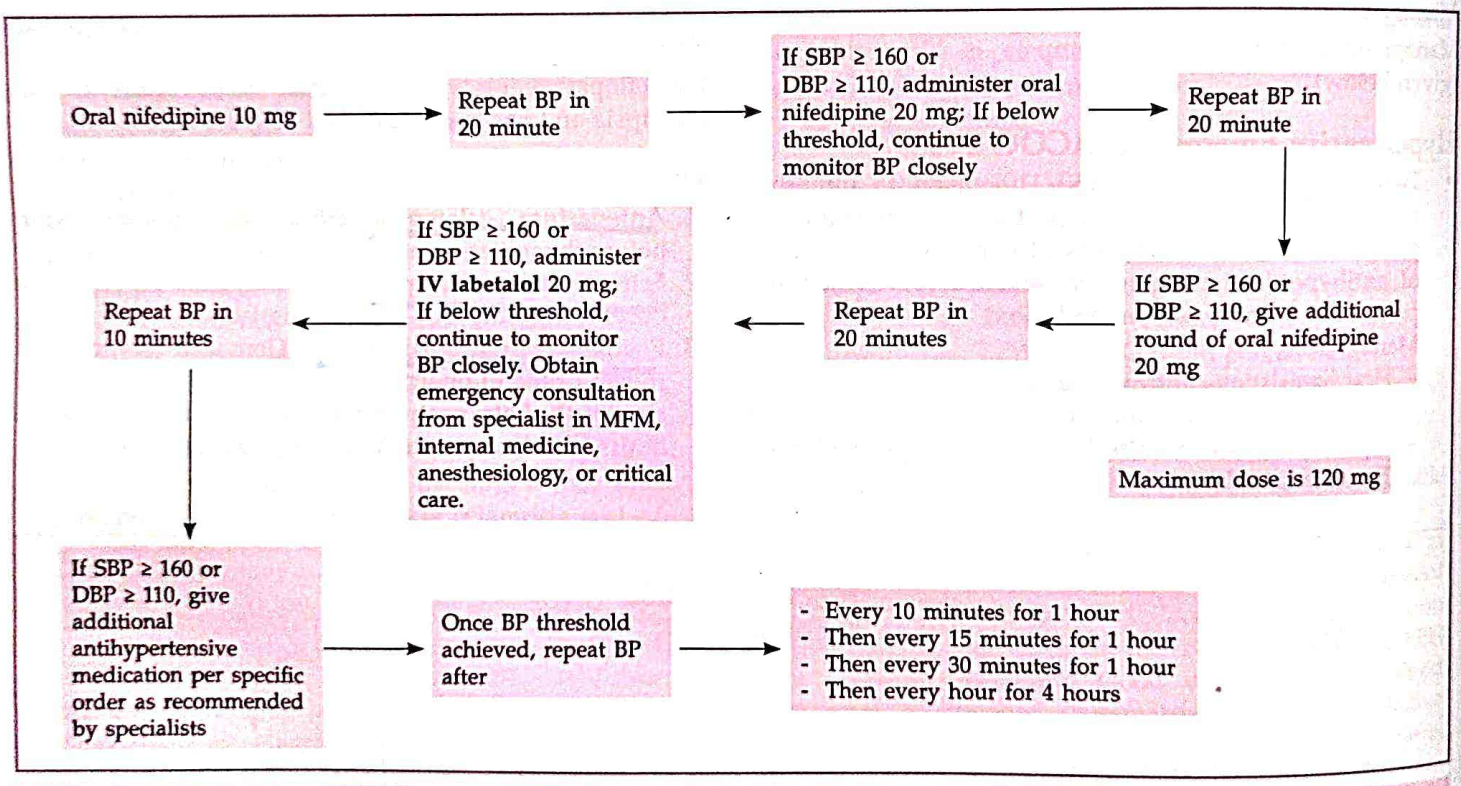
1. **Antepartum eclampsia (50%).** Convulsions occur before the initiation of labour pains.
2. **Intrapartum eclampsia (30%).** Convulsions are observed for the first time during labour.
3. **Postpartum eclampsia (20%).** Convulsions occur for the first time in puerperium usually within 48 hours of childbirth. Late postpartum eclampsia occurs after 48 hours of delivery upto as late as 6 weeks.




**Flow chart 32.2:** Labetalol Algorithm (ACOG 2017)




**Flow chart 32.3:** Hydralazine Algorithm (ACOG 2019)




**Flow chart 32.4:** Oral Nifedipine Algorithm (ACOG 2019)

4. **Intercurrent eclampsia.** When patient recovers from convulsion and pregnancy continues more than 48 hours after cessation of eclamptic fits.
5. **Atypical eclampsia.** Eclampsia occurring before 20th week of pregnancy or more than 48 hours but less than 4 weeks postpartum is exceedingly rare and is known as atypical eclampsia. It is seen in 1 in 1000 pregnancies in developing countries and 1 in 2000 pregnancies in developed countries.

## Causes of Convulsions

Various possible mechanisms are as follows:

1. **Hypoxia.** Due to spasm of cerebral vessels because of hypertension and increased vascular resistance.
2. **Cerebral haemorrhage and cerebral edema.** It may cause irritation.
3. **Cerebral dysrhythmia.** Due to hypoxia and edema
4. **Disseminated intravascular coagulation** in cerebral microcirculation.

Eclamptic seizures are not usually due to hypertensive encephalopathy as they are not usually associated with retinal hemorrhages, exudates and papilledema.

## Clinical Features

Most women, except rare fulminant variety, have signs and symptoms of pre-eclampsia preceding the convulsion. Typically it is a primigravida patient with preceding symptoms and signs of pre-eclampsia who presents to emergency department (casuality) with convulsions or an admitted patient of severe pre-eclampsia has a seizure.

## Prodromal Signs of Eclampsia

Prodromal signs of eclampsia are present in about 80% women and are as follows:

1. Severe and persistant headache (in approximately 80% cases)
2. Epigastric or right hypochondriac pain (in about 20% cases)
3. Photophobia or blurred vision (in about 40–50% cases)
4. Brisk deep tendon reflexes
5. Marked diuresis is seen for about 48 hours.

## Eclamptic Fit or Convulsion

Four stages of eclamptic fit which are epileptiform are recognized.

1. **Premonitory stage.** There is twitching of facial, tongue, hand and feet muscles. Eye balls are turned to one side to become fixed or roll. This may last from a few seconds to half a minute. The woman usually attains unconscious state.
2. **The tonic stage.** The whole body becomes rigid in a generalized muscular contraction, the features are distorted, the arms flexed and the hands clenched, the body being in a condition of tonic spasm. Respiration ceases and tongue protrudes between the teeth. Eye balls become fixed and cyanosis may be present. This may persist for 15 to 30 seconds.
3. **The clonic stage.** There is alternate contraction and relaxation of the voluntary muscles. Suddenly, the jaws begin to open and close violently followed by movement of eyelids. The other facial muscles and then all muscles alternately contract and relax in rapid succession. The tongue may be bitten by the violent action of the jaws (Fig. 32.4). This phase may last for 1-4 minutes. There is froth in the mouth; breathing becomes stertorous or halted and the face may be cyanosed or if not properly protected, the patient may fall from the bed and injure herself.
4. **The stage of coma.** In this stage, the convulsive movements stop with occasional jerks or twitches. The patient lies quiet, breathes stertorously; coma supervenes and the respiration gradually quietens down. The duration of coma is variable. In favourable cases, the patient wakes up after a short time and does not remember anything that has taken place earlier. In very severe cases, the coma persists from one seizure to another and death may result before she awakens.

**Status Eclampticus.** It is a variety of eclampsia in which repeated convulsions occur one after the other. It is a serious condition with high mortality.

## Differential Diagnosis

Eclampsia has to be differentiated from the following diseases which can cause convulsions and/or coma.

1. Cerebrovascular accident (e.g. haemorrhage)
2. Epilepsy (mostly normotensive)
3. Hypertensive encephalopathy (rarely pheochromocytoma)
4. Cerebral malaria
5. Intracranial neoplasms
6. Infections (e.g. meningitis, encephalitis)
7. Metabolic diseases (e.g. hypoglycemia, water intoxication)
8. Thrombotic thrombocytopenic purpura
9. Hysteria
10. Poisoning
11. Cerebral venous thrombosis

## Complications

The following complications can occur in eclampsia.

1. **Injuries.** During an eclamptic fit, injuries varying from bruises to fractures may occur. The tongue is usually bitten and can be so badly injured that it gets swollen (Fig. 32.4). It may fall back and occlude the glottis during the period of unconsciousness causing suffocation and even fatal asphyxia.
2. **Pulmonary.**
   (*i*) Pulmonary edema (5%)
   (*ii*) Pneumonia: Aspiration or infective (7%)
   (*iii*) Adult respiratory distress syndrome
   (*iv*) Pulmonary embolism



**Fig. 32.4:** Clinical photograph of a women with eclampsia with tongue bite

3. Neurological deficit (7%)
4. Hyperpyrexia
5. Renal failure (4%)
6. Placental abruption (10%)
7. **Liver.**
   (*i*) Hepatic necrosis
   (*ii*) Subcapsular haematoma
   (*iii*) HELLP syndrome
8. **Heart.** Acute left ventricular failure due to hypoxia and hypertension.
9. **Hematological.**
   (*i*) Thrombocytopenia
   (*ii*) DIC
10. Eye complications
11. **Puerperal complications.**
   (*i*) Postpartum shock: Dehydration and ketoacidosis
   (*ii*) Puerperal sepsis
   (*iii*) Puerperal psychosis
12. Exhaustion
13. Death

## Maternal Mortality

Mortality varies from 2–30% in different countries. Causes of maternal death are as follows:

(a) Cerebral Haemorrhage (most common cause) - 70%
(b) Cardiac failure
(c) Pulmonary edema
(d) Aspiration
(e) Acute Renal failure
(f) Postpartum shock
(g) Pulmonary embolism

## Perinatal Mortality

It is high in eclampsia. Causes are

(a) Prematurity (85–90%)
(b) Intrauterine asphyxia
(c) Trauma.

## Prevention

Identification of high risk factors and diagnosis and treating pre-eclampsia in time can prevent eclampsia. **Hence, pre-clampsia is not a preventable disease but eclampsia is a preventable disease.**

## Management

Management of eclampsia is shown in Flow Chart 32.5.

**Principles of Management Include:**

1. General management
2. Control of seizures
3. Control of blood pressure
4. Obstetric management
5. Early detection and timely and appropriate management of complications.

If the patient is at home or in a small peripheral hospital, she should be shifted to a tertiary care hospital after giving sedation. Obstetric flying squad, if available, can be useful. Otherwise, a trained junior doctor or nurse should accompany the patient during the journey to clear airway and prevent injury if eclamptic fit occurs on the way.

### Immediate Management

**In the hospital.** The immediate treatment is to prevent maternal injury, provide adequate oxygenation and minimize the risk of aspiration. Detailed history is taken from husband or relative.

1. There must be no noise.
2. Keep the airway clear by regular suction of airways. Mouth gag or padded tongue blade is inserted to prevent biting of the tongue.
3. Avoidance of injuries by using
   (a) Bed side rail
   (b) Physical restraints
4. Keeping patient in lateral position to avoid aspiration.
5. Oxygen is given by a face mask.
6. An intravenous access is started. Intravenous fluids should be given with caution to avoid fluid overload and pulmonary edema. Ringer lactate (crystalloid) solution is given in the quantity equivalent to urinary output of last 24 hours plus one litre for insensible losses (< 2 litres in 24 hours) at rate of 1 mL/kg/hour.
7. Heart rate, blood pressure and respiratory rate are regularly monitored (about half hourly)
8. Monitor the foetal heart rate.
9. Catheterize the patient with Foley's catheter to monitor urine output (hourly). Central venous pressure monitoring is useful in these cases.
10. A complete examination is done after fits are controlled.
11. Antibiotics are given to prevent infection.
12. Control of seizures (see below)
13. Control of hypertension (see below)
14. Delivering the woman safely as soon as possible.
15. Prevention of recurrence of seizures.
16. Early detection and treatment of complications.

## Control of convulsions (seizures)

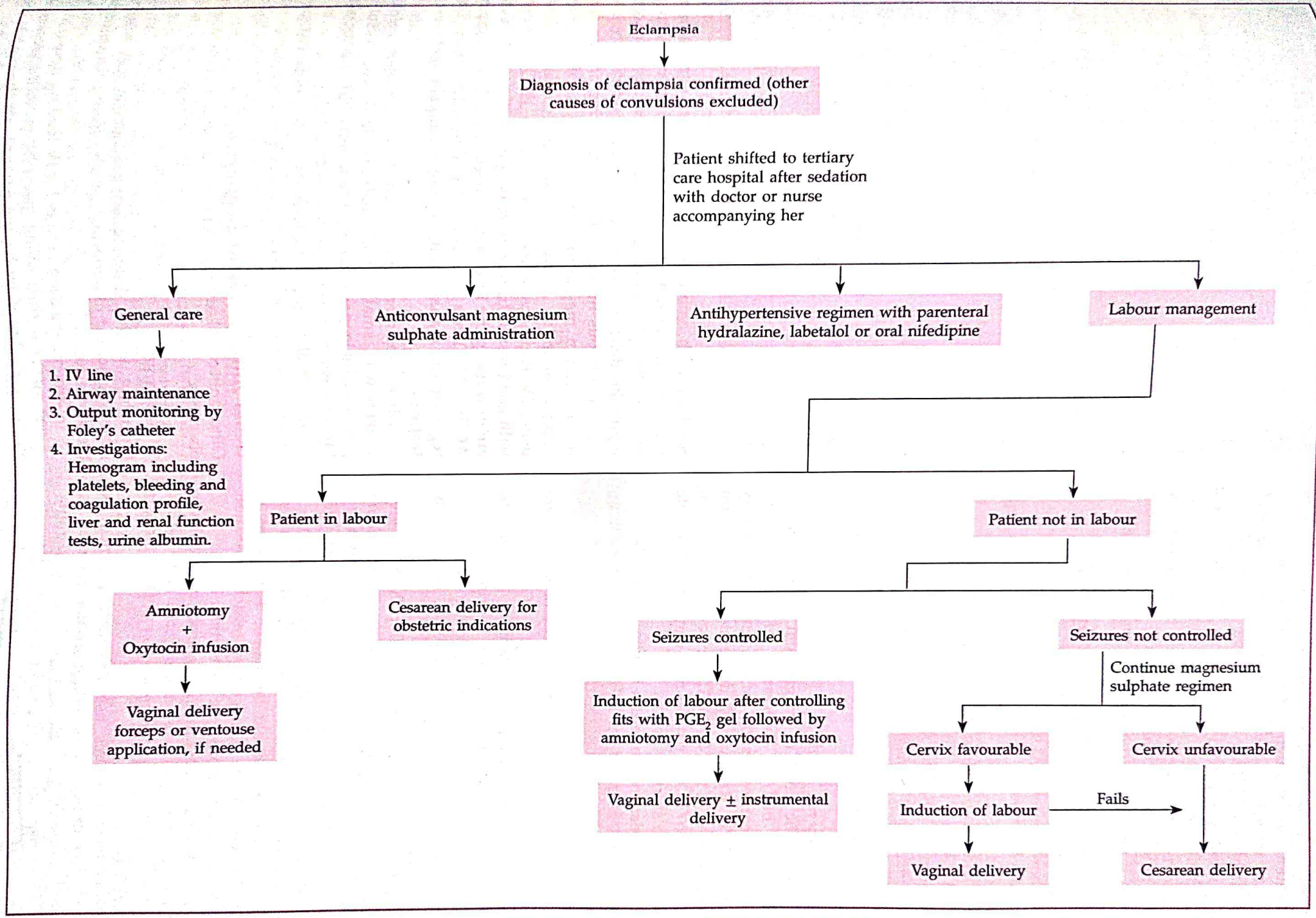
Various anticonvulsant regimens have been tried to control seizures in eclampsia. **It is now clearly proven by large international studies that magnesium sulphate ($MgSo_4.7H_2O$) is the best anticonvulsant and should be the treatment of first choice.**

**The multinational: Eclampsia Trial Collaborative Group (1995)** compared magnesium sulphate, diazepam and phenytoin for eclampsia. It concluded that *magnesium sulphate was associated with a significantly lower incidence of recurrent seizures and lesser maternal mortality (3%) as compared with other regimens.* It is also more effective than other anticonvulsants, does not cause maternal or foetal depression and is associated with reduced perinatal mortality rate. Hence, all doctors should now use only magnesium sulphate for eclampsia.

### Mechanism of Action

Magnesium probably has the following anticonvulsant actions on the cerebral cortex.

1. Reduced presynaptic release of neurotransmitter glutamate.
2. Blocking glutamatergic N-methyl-D-aspartate (NMDA) receptors.
3. Enhancement of adenosine action




**Flow Chart 32.5:** Management of eclampsia

4. Increased mitochondrial calcium buffering and blockage of calcium entry through voltage-gated channels.
5. It causes relaxation of vascular smooth muscles with vasodilatation and decreased vascular resistance.

The patient stops convulsing soon after the initial administration of magnesium sulphate and within an hour or two regains consciousness sufficiently to be oriented to place and time (also see Chapter 14). It increases cerebral blood flow. It also causes dialatation of uterine arteries.

## Various Regimens of Magnesium Sulphate ($MgSO_4$) for Eclampsia

They are shown in table 32.9.

**1. Pritchard Regimen**

(Pritchard et al. USA, 1984).

**Dose.** After initial dose of 14 g (4g IV, 5g in each buttock IM), A dose of 5 g of $MgSO_4$ (50%) is given IM in alternate buttock 4 hourly. *Before administration of the next dose, the monitoring of the following is required*:

(a) *Knee jerk* (deep tendon reflex) *should be present.*
(b) *Urine output should be* > 30 mL/hr. *(> 100 mL in 4 hours)*
(c) *Respiratory rate should be > 14/minute*

Therapeutic level of magnesium is 4–7 mEq/L (2–3.5 m mol/L). **Depression of patellar reflex is the earliest sign of magnesium toxicity (10 mEq/L. Respiratory depression occurs with serum level of 12–14 mEq/L.** Cardiac arrest occurs at 25–30 mEq/L. The patient may complain of double vision and slurred speech.

If possible, measure the serum magnesium levels.

**Side effects.** Warmth, flushing, nausea, vomitting, muscular weakness, respiratory depression and diplopia-biro.

It is discontinued 24 hrs. after delivery or last seizure whichever is late.

If impaired renal function: (S. Creatinine > 1.2 ng/dL → half the maintenance dose is given. If urine output is < 25 mL/hr. → reduce dose according to mg levels.

***Antidote.*** Calcium gluconate 10%, 10 mL (1 g) slowly IV over 3 minutes.

If respiratory difficulty develops stop $MgSO_4$, give calcium gluconate. Mechanical ventilatory support should be provided.

Additional 2 g (20%) is slowly given intravenously for recurrent seizures. If woman is large 4 g may be given.

If seizures continue unabated then intubation may be required. Such patients should be managed in intensive care unit. Further fits are managed by muscle relaxation and intermittent positive pressure ventilation.

It was controlled in past that *combination of magnesium sulphate and nifedipine is to be avoided or given under careful supervision as it can cause sudden hypotension (both act through calcium channel).*

In India, most hospitals use Pritchand regimen as it is safer. The drawback of intramuscular regimen is erratic and unpredictable absorption from this route as there is vasospasm in pre-eclampsia and eclampsia. Moreover, there is risk of gluteal abscesses due to need of high volumes of fluids given intramuscularly (can be minimized by deep IM injection). Total IV regimen though causes predictable effect but may cause higher incidence of toxicity and should preferably be given with continuous maternal ECG monitoring and by doing regular serum magnesium levels. That is why it is less popular in India. However, many obstetricians are now using IV regimens without continuous ECG monitoring with good result. **In fact most hospitals all over the world are now using the intravenous regimen (Sibai or Zuspan). ACOG (2019) has also recommended intravenous Sibai regimen for management of pre-eclampsia with severe features.**

**Table 32.9: Various regimen of $MgSO_4$ for severe pre-eclampsia and eclampsia**

| Name | Loading dose | Maintenance dose |
|---|---|---|
| Pritchard (1984) | 4 g (20% solution) IV over 5 min. and 5 g IM (50% sol) into each buttock (total 14 g) | 5 g IM every 4 hr into alternate buttock |
| Zuspan | Loading dose 4 g IV (20%) over 5–10 min. | 1 g/hr IV infusion (50%) |
| Sibai (2005) (Most commonly used regimen) | Loading dose 4-6 g over 15–20 min. (100 ml Normal Saline) | 2 g/hr IV infusion (maintenance) |
| Low dose (Dhaka) regimen (Chowdhury 2009) (Suitable for lean Asian women) | 10 g: 4 g IV slowly over 15 Min. 3 g IM in each buttock | 2.5 g IM in alternate buttock every 4 hr until 24 hrs after first dose. |

g: gram; I.V.: intravenous; IM: intramuscular; Hr: hour; h: hour, sol.: solution

Dhaka regimen can be used for south-east Asian women with smaller body habitus. It is also useful in patients with jeopardized renal function. With this regimen, $MgSO_4$ toxicity is lower as dose is reduced.

Jana et al (2013) from Kolkata (India) observed good results with low dose magnesium sulphate regimen.

## Other Anticonvulsant Regimens

Other anticonvulsant regimens are not preferred in modern obstetrics.

**1. Phenytoin**

Phenytoin is the drug of second choice and acts by inhibiting spread of abnormal activity from seizure foci to the motor cortex.

In general, the dose used is 1 g IV loading dose diluted in 200 mL of normal saline given by slow infusion over 20 minutes followed by 100 mg 6 hourly for the next 24 hrs.

ECG should be performed. Serum level, if available, is helpful.

Side effects include cardiac toxicity, hypotension, nystagmus, ataxia and lethargy.

#### 2. Diazepam

Diazepam is not often used at present. This regimen was popular in the past.

*Lean Regimen*

A loading dose of 10 mg diazepam IV over 2 minutes followed by IV infusion of 40 mg in 500 mL normal saline for 24 hours. During the next 24 hours – 20 mg of diazepam is infused in 500 mL of normal saline.

Side effects are maternal hypotension and lethargy (hypotonia), respiratory depression and thermoregulatory problems in the newborn (see Chapter 14 for details).

#### 3. Lytic Cocktail or Menon's Regimen (Krishna Menon, 1961 Chennai, India)

In Menon's regimen, a combination of pethidine, chlorpromazine and promethazine were used. Although Menon observed good results with this regimen and it was very popular a few decades back, it is now obsolete and has been superseded by magnesium sulphate regimen.

#### 4. Midozolam has also been used

## Control of Hypertension

The aim of antihypertensive therapy is to keep the diastolic blood pressure between 90 and 100 mm Hg. Drugs commonly used are hydralazine, labetalol and nifedipine (see Chapter 14 for choice of drugs, dosage and side effects).

See above flow chart 32.2 to 32.4 for dosage algorithm of labelalol, hydralazine and nifedipine for severe pre-eclampsia and eclampsia. Other antihypertensive drugs which have been tested in studies but are not commonly used include Verapamil, nimodepine, nitroglycerine, ketanserin and nicardipine. WHO (2018) has advised to avoid angiotensin converting enzyme inhibitors, angiotensin receptor blockers and sodium nitroprusside during pregnancy due to safety concerns.

## Management during convulsion

Use of mouth gag to prevent tongue bite, clearing of air passage of secretions and mucus plug with mucus sucker or suction machine and tilting the head to one side and raising the footend of bed are helpful during a fit. Oxygen therapy should be given till disappearance of cynosis.

**Status eclampticus.** If patient continues to have fits inspite of $MgSO_4$, thiopentone sodium 0.5 g dissolved in 20 mL of 5% dextrose is given slowly under supervision of anesthetist. If it fails then intubation should be done and patient should be shifted to intensive care unit for muscle relaxation and assisted ventilation.

### Management of Complications

1. **Pulmonary edema.** Furosemide 40 mg IV followed by 20 mg IV 6–8 hourly along with aspiration of secretions and oxygen therapy are useful.
2. Heart failure is treated by oxygen therapy, IV furosemide and digitalization.
3. **Treatment of renal failure and anuria.** Dopamine infusion (1 µg/kg) is useful in oliguric phase with raised CVP > 8 mm Hg (see Chapter 35[vi] for details).
4. Hyperpyrexia is difficult to control being central in origin. Cold sponging and antipyretic therapy are tried.
5. Psychosis is treated with trifluoperazine, haloperidol or chlorpromazine (see Chapter 30 for details).

## Obstetric Management

Decision to deliver the patient must be taken as soon as the condition is stabilized. Delaying delivery to increase the foetal maturity will risk the lives of both mother and fetus. After seizure, labour usually starts spontaneously. It can be induced successfully even in patients remote from term using prostaglandin $E_2$ gel.

### Mode of Delivery

Deliver within 4 hrs. of stabilization. Labour ensueors spontaneously in 70–80% cases. Vaginal delivery is preferred as it is safer.

**Assess cervical status.** If Bishop's score > 6 → oxytocin drip followed by amniotomy is performed. Cesarean section is only performed for strict obstetric indications or uncontrolled eclampsia with unfavourable cervix as given in Table 32.10.

**Table 32.10: Indications of Cesarean Delivery in Pre-eclampsia and Eclampsia**

| |
|---|
| **I. Elective Cesarean Delivery** |
| 1. Associated obstetric conditions |
| (i) Cephalopelvic disproportion |
| (ii) Diabetes |
| (iii) Elderly primigravida |
| (iv) Abnormal lie or presentation |
| (v) Placenta previa |
| 2. Worsening maternal condition |
| 3. Foetal monitoring not possible |
| 4. Vaginal delivery not possible in stipulated time |
| **II. Emergency Cesarean Delivery** |
| 1. Rapidly worsening maternal condition |
| 2. Failure to progress |
| 3. Foetal distress |
| 4. Hepatic capsular distension |

Pediatrician should attend delivery. Anesthetist should take care of patient. Regular monitoring of patient should be done in first stage.

*In Second Stage*

Prophylactic forceps are not routinely applied but tendency of prolonged second stage is cut short by forceps

application. Prophylactic intravenous ergometrine at anterior shoulder presentation is contraindicated. Oxytocin 5–10 units IM is given. One must be vigilant for any PPH and shock. Patients should be cared for in puerperium and should be followed up after 6 weeks for any persistent hypertension and proteinuria necessitating physician referral.

## CHRONIC HYPERTENSION DURING PREGNANCY

Chronic hypertension is present in 0.9 to 1.5% of pregnant women (ACOG 2019). Its rate has increased by 67% from 2000 to 2009 in USA mainly due to obesity and increasing maternal age (ACOG 2019). It may cause significant maternal, foetal and neonatal morbidity and mortality.

A women suffering from hypertension may become pregnant. It is defined as a hypertension (BP > 140/90 mm Hg) which predates pregnancy or which persists beyond 12 weeks after delivery. It is diagnosed before 20 weeks of gestation **(in absence of hydatidiform mole or non-immune hydrops fetalis)** and is associated with other features of chronic hypertension like:

1. Retinal changes of hypertension on fundus examination.
2. Associated renal disease or compromised renal function
3. Associated medical disorders which can cause hypertension (see below).

Differences in various types of hypertension are shown in Table 32.11.

### Incidence

The incidence of chronic hypertension during pregnancy is 2–4%.

### Causes of Chronic Hypertension

#### I. Primary Hypertension or Essential Hypertension

Primary hypertension is the most common cause (90% cases) of hypertension in pregnancy.

#### II. Secondary Hypertension

1. Hypertensive states of pregnancy other than pre-eclampsia/eclampsia
2. Secondary hypertension due to renal disorders.
   (a) Acute and chronic glomerulonephritis
   (b) Interstitial nephritis
   (c) Polycystic kidney disease
   (d) Diabetic glomerulosclerosis
   (e) Polyarteritis nodosa
   (f) Renovascular stenosis
   (g) Chronic renal failure with treatment by dialysis
   (h) Renal transplant
3. Obesity
4. Chronic hypertension due to endocrinopathies
   (a) Pheochromocytoma
   (b) Cushing's disease and syndrome
   (c) Primary hyperaldosteronism
   (d) Thyrotoxicosis
   (e) Acromegaly
   (f) Diabeties mellitus
5. Chronic HT due to coarctation of the aorta
6. Collagen vascular diseases
   (a) Systemic lupus erythematosis (SLE)
   (b) Scleroderma

### Risk Factors for Developing Superimposed Pre-eclampsia

1. Presence of renal disease
2. Age (> 40 years)
3. Long duration of hypertension (> 15 years)
4. BP (> 160/110 mm Hg)
5. Presence of thrombophilias
6. Diabetes mellitus
7. Connective tissue disorders like SLE
8. Coarctation of aorta

Differences between pre-eclampsia, essential hypertension and renal disease are given in Table 32.9.

### Complications of Chronic Hypertension during Pregnancy

There is increased incidence of the following complications.

#### Maternal Complications

1. Superimposed pre-eclampsia
2. Abruptio placentae
3. Congestive cardiac failure
4. Malignant hypertension
5. Cerebrovascular accidents
6. Renal damage and failure

#### Foetal Complications

1. Abortions
2. Foetal growth restriction (FGR)
3. Intrauterine death
4. Increased neonatal complications
5. Prematurity

### Management

1. **Preconceptional care.** The women with chronic hypertension (both essential and secondary) should be evaluated by obstetrician and physician before planning a pregnancy. They should be advised about diet, lifestyle and stopping smoking habits. Severity of their blood pressure and renal function tests (or other organ specific tests in other causes of secondary hypertension) should be assessed. Women with severe disease or malignant hypertension, uncontrolled hypertension with complications should avoid conception unless their health improves. Change of antihypertensives and diuretics are necessary. If a woman is on angiotensin converting enzyme (ACE) inhibitors like captopril or enalapril, they must be stopped as they are contraindicated in pregnancy and can cause adverse outcome (see Chapter 14 for details).
2. Detailed history taking
3. Careful clinical examination
4. Investigations. They are aimed at detecting treatable causes of hypertension.
   (a) Blood urea (b) Serum creatinine

**Table 32.11: Differences in Various Types of Hypertension**

| | Pre-eclampsia | Essential Hypertension | Renal Disease Hypertension |
|---|---|---|---|
| 1. Age | Extremes of age, mostly young | Mostly elderly women | Not related |
| 2. Parity | Usually primigravidas | Usually multiparas | Not related |
| 3. Family history | May be present | Usually present | Not related |
| 4. Past history | Pre-eclampsia in previous pregnancy | Pre-pregnant hypertension present | Previous history of renal diseases |
| 5. Onset of hypertension | After 20 weeks of gestation | Before 20 weeks of gestation | Onset is variable |
| 6 Hypertension following delivery | Completely disappears | Persists after 3 months | Persists |
| 7. Cardiac status | Usually normal | Ventricular enlargement in long standing disease | Usually normal |
| 8. Deep tendon reflexes | Usually brisk | Normal | Normal |
| 9. Clonus | May be present | Absent | Absent |
| 10. Proteinuria and other urinary abnormalities | Increased | Usually negative | Positive findings such as low specific gravity, presence of albumin and granular casts |
| 11. Fundus examination | Usually normal; retinal edema, constriction of arterioles, nicking of the veins in severe cases | Silver wiring of the arterioles. Hypertensive retinopathy | Albuminuric retinopathy, cotton wool patches and flame-shaped hemorrhages |
| 12. Hemoglobin and hematocrit | Increased (Hemoconcentration) | No change | No change |
| 13. Platelets | Thrombocytopenia | Normal count | Normal count |
| 14. Liver enzymes | Raised in severe pre-eclampsia and HELLP syndrome | Normal | Normal |
| 15. Renal function tests<br>(a) Blood urea<br>(b) Serum creatinine<br>(c) Serum uric acid | <br>Normal or slightly raised<br>Normal or slightly raised<br><br>Usually raised | <br>Normal<br>Normal<br><br>Normal | <br>Raised<br>Raised<br><br>Raised |

(c) Serum uric acid
(d) 24 hour urinary creatinine clearance
(e) Renal scan
(f) Auto antibody screen
(g) Compliment studies
(h) 24 hours urinary vanillyl mandelic acid (VMA) excretion (suspected pheochromocytoma)
(i) Electrocardiography (ECG)
(j) Echocardiography
(k) Daily foetal kick chart
(l) Regular non-stress test
(m) Ultrasound (fortnightly) for foetal growth, liquor volume, uterine artery flow studies (Doppler)

## Treatment

### Low Risk Group

(If BP between 140–170 mm Hg systolic and 90–109 mm Hg diastolic). Patient is reviewed at the earliest in pregnancy and then at 12 weeks. Blood pressure may fall to normal due to mid-trimester drop. Patients are seen regularly and more often in antenatal clinic and their BP, renal function and urate levels are checked regularly. Any evidence of superimposed pre-eclampsia and foetal growth restriction must be looked for. Antihypertensives started are oral methyldopa, labetalol or nifedipine in suitable doses. Patients with chronic hypertension are followed until delivery and usually have normal vaginal delivery without any major complication with adequate maternal and foetal outcome. If superimposed pre-eclampsia supervenes, the case is managed like pre-eclampsia (given above).

### High risk group

**(If BP is equal to or more then 170/110 mm Hg).** Proper counselling of women is important as there is high rate of maternal and foetal complications. They are

usually admitted to hospital, properly investigated and antihypertensives are started to control their BP. Usually a combination of antihypertensives is given like methyldopa (up to 500 mg four times a day) with labetalol 100–200 mg twice or thrice daily or atenolol 50 mg twice daily (can be increased up to 300 mg BD) and or nifedipine 10 mg three to four times daily. The rest of the management is like severe pre-eclampsia.

### Intrapartum Care

Intrapartum care is like in pre-eclampsia. These women may require termination of pregnancy for superimposed pre-eclampsia or FGR or foetal compromise on Doppler studies. Postnatal counselling of the patient and her husband is important as they should have small family as pregnancy may upset her chronic disease adversely.

### Secondary Hypertension

Secondary hypertension is rare but serious. Pheochromocytoma is associated with 50% maternal mortality if not diagnosed before the onset of labour. Coarctation of aorta is associated with increased risk of aortic dissection and or rupture during pregnancy. Therapeutic abortion may be justified in early pregnancy in women with markedly raised BP with severe renal disease.

## CHRONIC RENAL DISEASE DURING PREGNANCY

The incidence of chronic renal disease in pregnancy is about 0.2% (see Chapter 35[vi] for pyelonephritis and renal failure).

### Effect of Pregnancy on Kidney Disease

The effect depends upon the severity of kidney disease. The risk of renal failure is low (< 5%) in presence of mild disease (serum creatinine < 125 µmol/L), 10% in moderate disease (creatinine 125–250 µmol/L and may be as high as 50% for severe disease (creatinine > 250 µmol/L). Prognosis is worse for women developing superimposed pre-eclampsia. Primary glomerulonephritis has better prognosis while focal glomerulosclerosis, immune nephropathy (IgA nephropathy) and membranoproliferative glomerulonephritis have poor prognosis.

### Effect of Kidney Disease on Pregnancy

*The effect of pregnancy depends upon the severity of renal disease, serum creatinine levels, hypertension and proteinuria.* There is increased risk of superimposed pre-eclampsia in which case perinatal mortality may be up to 50%. There is increased risk of abortions, IUGR, preterm labour and intrauterine death in these women, especially with severe disease. Careful pregnancy surveillance, proper treatment of renal disease in collaboration with nephrologist and improved neonatal care have improved the prognosis for both mother and the newborn.

### Management

Pre-conceptional counselling is important and women with severe disease should be advised against conception. Therapeutic abortion may be justified in early pregnancy in such cases. For mild to moderate disease, pregnancy is continued and careful and regular assessment of the women to assess her kidney function is performed on each visit. Antihypertensive drugs are given in presence of hypertension. Foetal monitoring should be performed on each visit. Management of labour is like pre-eclampsia with aim of vaginal delivery.

## Key Points

1. Hypertensive disorders during pregnancy include gestational hypertension, pre-eclampsia, eclampsia, chronic hypertension and superimposed pre-eclampsia. They are important causes of maternal and perinatal mortality and morbidity throughout the world.
2. Pre-eclampsia is a peculiar syndrome of multiple organ dysfunction during pregnancy which usually manifests after 20 weeks of pregnancy (except in case of hydatidiform mole) and is defined as a hypertension of 140/90 mm Hg or more with proteinuria with or without pathological edema. However, recent ACOG (2013) guideline have removed proteinuria from definition and has classified pre-eclampsia into **pre-eclampsia without severe features (formerly mild pre-eclampsia)** and **pre-eclampsia with severe features (formerly severe pre-eclampsia)**.
3. Exact etiology is not known with probably many factors playing a role. In genetically predisposed individuals immune maladaptation, cytokine mediated oxidative stress markers cause endothelial cell dysfunction with placental ischemia. There is incomplete trophoblastic invasion in the spiral arteries in these women.
4. Vasospasm is a basic contributing factor in its pathogenesis with pathological changes occurring in placenta, kidneys, liver and brain and these organ changes explain most symptoms and signs of the disease. Thus, vasospasm of uteroplacental bed explains foetal growth restriction and placental abruption. Vasospasm of renal vessels causes proteinuria, oliguria and renal failure while liver vessels vasospasm explains its subcapsular haematoma, necrosis and raised liver enzymes. Vasospasm in CNS explains seizures, cortical blindness and papilliedema.
5. The clinical features depend upon the organs involved as explained above. The disease can be mild, severe or eclampsia (convulsions with hypertension and proteinuria).

6. Prediction and prevention of pre-eclampsia is not possible. However, eclampsia can be prevented by timely detection and management of severe pre-eclampsia.
7. Management of pre-eclampsia without severe features (mild pre-eclampsia) is observation, control and monitoring of BP, renal and hepatic functions and delivering at 38 weeks gestation.
8. Management of pre-eclampsia with severe features (severe pre-eclampsia) (diastolic BP > 110 mm Hg with excessive proteinuria with or without other symptoms and signs like blurring of vision, headache, epigastric pain and oliguria) is control of BP with antihypertensive therapy (methyldopa, labetalol and/or nifedipine). Anticonvulsant may be required. Termination of pregnancy is indicated for persistent raised BP.
9. HELLP syndrome is a variant of severe pre-eclampsia characterized by hemolysis, elevated liver enzymes and low platelets and is managed like severe pre-eclampsia by antihypertensives, anticonvulsants and termination of pregnancy.
10. Eclampsia is the presence of convulsions in a case of pre-eclampsia (hypertension with proteinuria) and is a serious condition with high maternal and perinatal mortality. The eclamptic fit (convulsion) has four stages: Premonitory stage, tonic stage, clonic stage and stage of coma. It has many complications involving various organs like coma (cerebral haemorrhage), adult respiratory distress syndrome (lungs), coagulation failure, renal failure and even death.
11. Management should be in a tertiary care hospital by general measures, antihypertensive therapy, anticonvulsant therapy and by termination of pregnancy.
12. For eclampsia, intravenous labetalol and hydralazine are preferred but nifedipine can also be given. Magnesium sulphate is the anticonvulsant of choice. It can be given intravenously or intramuscularly using various regimens. **Currently recommended regimen is the Sibai regimen** with 4–6 g IV magnesium surgical loading dose followed by 2 g IV per hour maintenance dose, a loading dose of intravenous 4 g (20%) magnesium sulphate is given along with 10 g of intramuscular $MgSO_4$ (5 g on each buttock) followed by 5 g intramuscular $MgSO_4$ every four hourly until 24 hours after delivery.
13. Essential hypertension and chronic renal disease can occur during pregnancy and must be differentiated from pre-eclampsia. Superimposed pre-eclampsia is a severe type of pre-eclampsia in chronic hypertensive patients and is treated like pre-eclampsia and with antihypertensive therapy. It has worst prognosis.

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CHAPTER 33

# Multiple Pregnancy

**Multiple pregnancy (syn. multifetal gestation)** is a high risk pregnancy associated with an increased risk of perinatal morbidity and mortality and thus requires special attention. The study of twins is called **gemellology**.

## DEFINITION

**The development of two or more than two fetuses simultaneously in a pregnant uterus is called multiple pregnancy.** Simultaneous development of two fetuses is called **twin pregnancy** and is the most common variety of multifetal pregnancy. The other types are the development of three fetuses **(triplets)**, four fetuses **(quadruplets)**, five fetuses **(quintuplets)**, six fetuses **(sextuplets)** and so on.

## VARIETIES

Multiple pregnancy is of two types. The type and genesis of twinning is shown in Figs. 33.1 A–E.

1. **Dizygotic twins** or **binovular** or **fraternal twins.** It results from fertilization of two ova which have most probably ruptured from two distinct grafian follicles, usually of the same ovary or one from each ovary, by two sperms. The babies bear only fraternal resemblance to each other. It is the most common variety and nearly two-thirds (67%) of all twins are dizygotic. All dizygotic twins have two placentae which are dichorionic and diamniotic (Fig. 33.1 A). Rarely they may be fused.

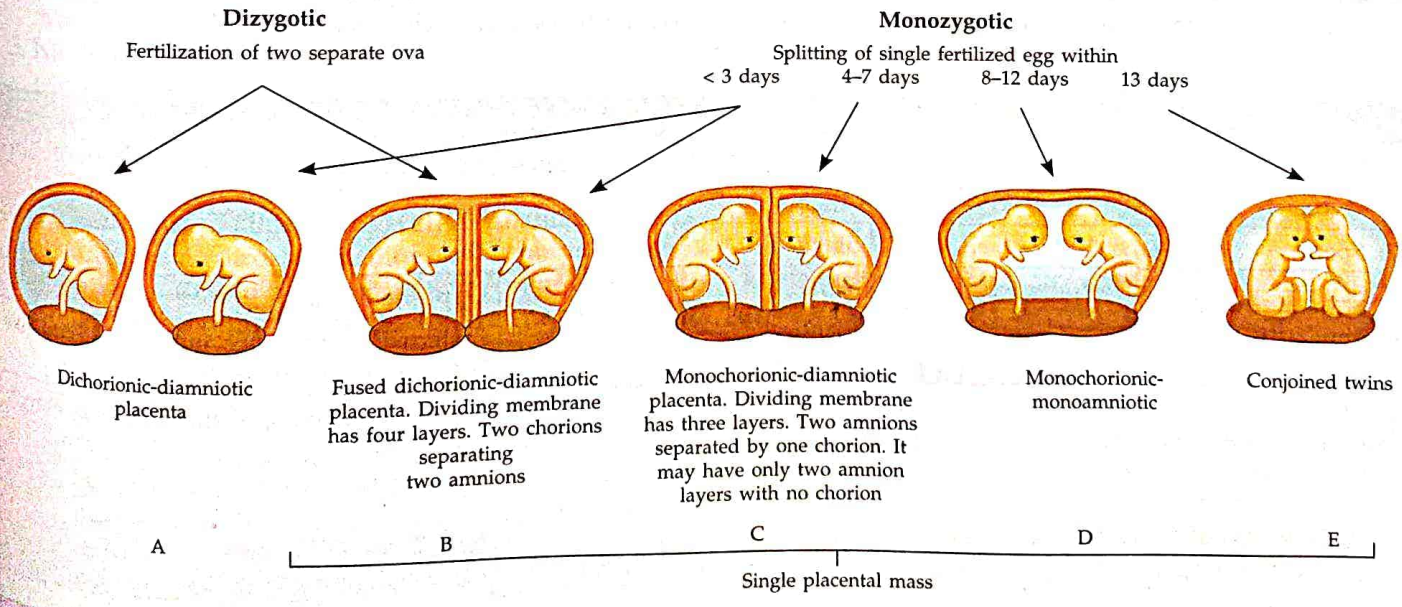



**Fig. 33.1:** Type and genesis of twinning

2. **Monozygotic or Uniovular Twins or Identical Twins.** It results from fertilization of a single ovum by a single sperm and occurs in 1/3rd (33%) of all twin pregnancies. It is of many types depending upon the 'time' when splitting occurs in the embryo as described below.
   (a) If cleavage takes place at about the 8 cells stage, i.e. **within 3 days (72 hours)** after fertilization, the resulting embryo will have two separate or a single fused placenta, two chorions and two amnions **(dichorionic diamniotic)**. It accounts for nearly one-third of monozygotic twin pregnancies (Figs. 33.1A and B).
   (b) If the division occurs when the inner cell mass is forming **(4–7 days)**, the two embryos will develop enclosed by a single chorion, have a single placenta but two separate amniotic sacs **(monochorionic-diamniotic). It is the most common variety accounting for about two-thirds of monozygotic twins** (Fig. 33.1 C).
   (c) If the splitting occurs between **8–12 days** after fertilization, i.e. after differentiation of amnion there is a single amniotic cavity, single chorion and a single placenta **(monochorionic-monoamniotic).** These account for only 1–5% of monozygotic twins (Fig. 33.1 D).
   (d) On very rare occasions, when splitting occurs after the **appearance of the primitive streak**, i.e. after the 13th day of fertilization, it results in the formation of **conjoined twins** within a single amnion and chorion (Fig. 33.1 E).

## DETERMINATION OF ZYGOSITY

The most frequently used method to determine the zygosity of twins is the examination of the placenta at birth. However, there are variations in monozygotic placentation. Microscopic examination of a piece of the septum dividing the two foetal cavities is therefore crucial. In diamniotic-dichorionic placenta there is chorionic tissue present between the two amnions. In monochorionic-diamniotic placentae, the septum consists of two amnion layers without interposing chorion (Table 33.1).

**The finding of a monochorionic placenta is unequivocal proof of monozygosity.**

However, it may be difficult in some cases to decide whether a single placenta is monochorionic-diamniotic or whether it is a fused dichorionic-diamniotic placenta which may be found in both dizygotic and monozygotic twins. Other methods which have been used to determine twin zygosity include study of blood groups, genetic fingerprinting using DNA probes at differences in restriction fragment length polymorphism present in highly polymorphic areas of the genome. **Best period of gestation for determination of zygosity is between 9–12 weeks.**

### Superfetation

In superfetation, **there is fertilization of two ova released in two different menstrual cycles.** Superfetation requires ovulation and fertilization during the course of an established pregnancy, which would theoretically be possible until the uterine cavity is obliterated by the fusion of the decidua capsularis to the decidua by 14–16 weeks. Although known to occur in mares, it is yet unproven in humans.

### Superfecundation

Superfecundation refers to **the fertilization of two ova within the same menstrual cycle but not at the same coitus,** nor necessarily by sperms from the same male. There may thus be two babies from different fathers with different looks and blood groups (e.g. a black and white baby born to a white mother with two instances of sex with a black and a white partner).

**Table 33.1: Differences between Monozygotic and Dizygotic Twins**

| Features | Monozygotic Twin Pregnancy | Dizygotic Twin Pregnancy |
|---|---|---|
| 1. Ovum | Single | Two |
| 2. Sperm | Single | Two |
| 3. Sex | Same | Opposite or same |
| 4. Similarity | Identical | Fraternal resemblance only (similarity like between two brothers or sisters) |
| 5. Placenta<br>(a) Communicating vessel<br>(b) Intervening membrane<br>(c) Thickness of intervening membrane<br>(d) Twin peak sign | Single (43%) or double<br>(a) Present<br>(b) Absent in monochorionic and monoamniotic twin<br>(c) < 2 mm (82–89%)<br>(d) Usually absent (44%) | Usually double occasionally fused at margin<br>(a) Absent<br>(b) Present<br>(c) > 2 mm (95%) usually<br>(d) Usually present (97.3%) |
| 6. Genetic features | Same | Different |
| 7. DNA Microprobe Technique (most definite) | Same | Different |
| 8. Skin grafting from one to other | Accepted | Rejected |

## INCIDENCE

**The frequency of monozygotic twin births is relatively constant throughout the world at approximately one in 250 births and is largely independent of race, heredity, age and parity.** There is now evidence that the incidence of zygotic splitting is increased following assisted reproductive technology (ART). The incidence of dizygotic twinning, however, is influenced remarkably by race, heredity, maternal age, parity and especially fertility drugs (see below).

**Hellin's law (1895)** propounds the following rates of multiple pregnancy.

| | |
|---|---|
| 1. Twins | 1 : 80 |
| 2. Triplets | 1 : $80^2$ (1 : 6400) |
| 3. Quadruplets | 1 : $80^3$ |
| 4. Quintuplets | 1 : $80^4$ |
| 5. Conjoined twins | 1 in 60,000 births |

There has been increase in incidence of twin pregnancy from 1.9% in 1980 to 3.3% in 2009 in USA. The rate of triplets and higher order multiple birth rate declined from 193 per 100,000 births (1998) to 93 per 100,000 birth in 2018 in USA (ACOG 2021).

## ETIOLOGY

The exact cause of multiple pregnancy is not known. **There are variations in the frequency of dizygotic twinning** (varying with maternal characteristics) **as follows:**

1. **Racial.** It is more common in certain races (Africans) than others (Japanese).
2. **Hereditary factors,** especially from the maternal side (history of twin pregnancy in mother or sister).
3. **High parity** (especially para 5 and above)
4. **Rising maternal age.** With a peak at 37 years (due to maximum FSH levels leading to double ovulation)
5. **Nutrition.** Higher rates (25–30%) in taller, heavier women with increased nutritional levels and who receive folic acid supplementation.
6. **Iatrogenic**
   (a) **Use of assisted reproductive techniques.** The treatment of infertility with ovulation-inducing drugs or invitro fertilization and embryo transfer possibly due to a minor trauma to the blastocyst. **Ovulation induction with clomiphene (6–8% risk of multiple pregnancy) or gonadotrophins (16–40% risk) increase the chances of both monozygotic and dizygotic twins, especially the later.**
   (b) **Contraception.** Risk of twin pregnancy increases by two times if the conception occurs within one month after discontinuation of oral contraception (after using for more than 6 months) due to sudden release of gonadotrophins.

## PROGNOSIS

Twin pregnancy is considered a high risk pregnancy. **Maternal mortality** increases (3–7 times) in multiple pregnancy than singleton pregnancy. **Postpartum haemorrhage** which may be due to the larger size of the uterus in twin pregnancy is the most common cause of maternal mortality followed by **anaemia** and **pre-eclampsia.**

Maternal morbidity is increased significantly due to complications and increased incidence of operative interference in multiple pregnancy.

### Perinatal Mortality

Perinatal mortality increases markedly (3 to 11 times) in twin pregnancy than in singleton pregnancy. *Prematurity, growth-restricted fetus and infection are the most common causes. The second twin is at higher risk than the first twin. The monozygotic twins have two and a half times higher mortality than dizygotic twins.* There are higher chances of congenital abnormalities, discordant growth, twin-to-twin transfusion syndrome and malpresentations.

## DIAGNOSIS OF MULTIPLE PREGNANCY

### History

1. Family or past history of multiple pregnancy, especially on the maternal side.
2. History of treatment of infertility with ovulation inducing drugs like FSH plus chorionic gonadotropins or clomiphene citrate or use of assisted reproductive techniques (ART) like in-vitro fertilization.

### Clinical Features

Due to excess of hCG hormone in multiple pregnancy and excessive distension of uterus, minor ailments of normal pregnancy are seen more commonly.

1. Increased nausea and vomiting and hyperemesis gravidarum during early pregnancy.
2. Cardiorespiratory symptoms like palpitation and dyspnea are more common in later pregnancy.
3. Increased chances of swelling and varicosity of legs and vulval veins and hemorrhoids (piles) due to pressure of large gravid uterus.
4. Excessive abdominal distension
5. Excessive foetal movements

### General Physical Examination

Look for pulse, blood pressure, pallor, edema, heart and chest auscultation.

### Abdominal Examination

#### Inspection

There is excessive distension of abdomen.

#### Palpation

1. Symphysiofundal height of the uterus is more than the gestational period, especially in second half of pregnancy.
2. Abdominal girth is increased (> 100 cm at term).
3. Uterus feels full of too many foetal parts.
4. Palpation of two foetal heads or three foetal poles is diagnostic of multiple pregnancy.

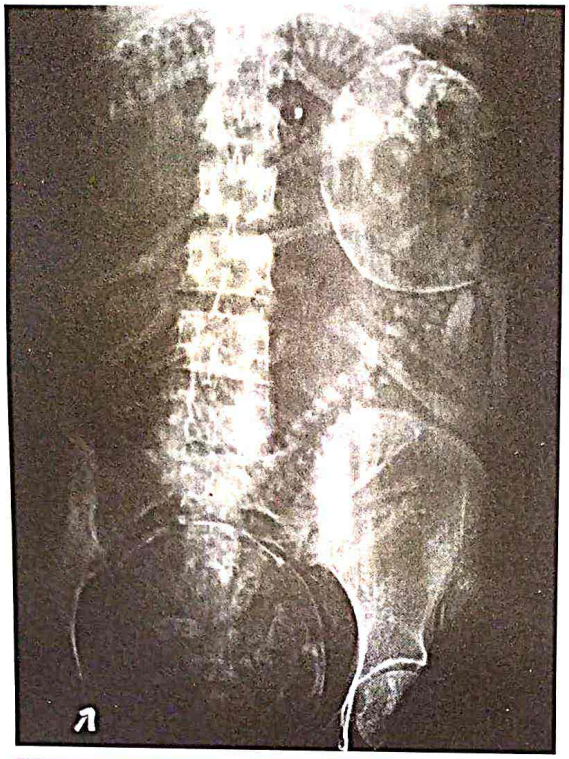


**Fig. 33.2:** X-ray—Abdomen showing twin pregnancy with two foetal heads and two foetal spines

## Auscultation

Presence of *two distinct foetal heart sounds at two different areas at least 10 cm apart with a silent area in between* by two independent observers is diagnostic of twin pregnancy provided the difference in heart rates is at least 10 beats per minute.

However, the abdominal palpation and auscultation can be difficult with polyhydramnios.

## Vaginal Examination

Depending upon the presentation of two fetuses, one foetal head is usually felt on vaginal examination while the other foetal head may be higher up.

Sometimes diagnosis may not be made before the birth of the first baby.

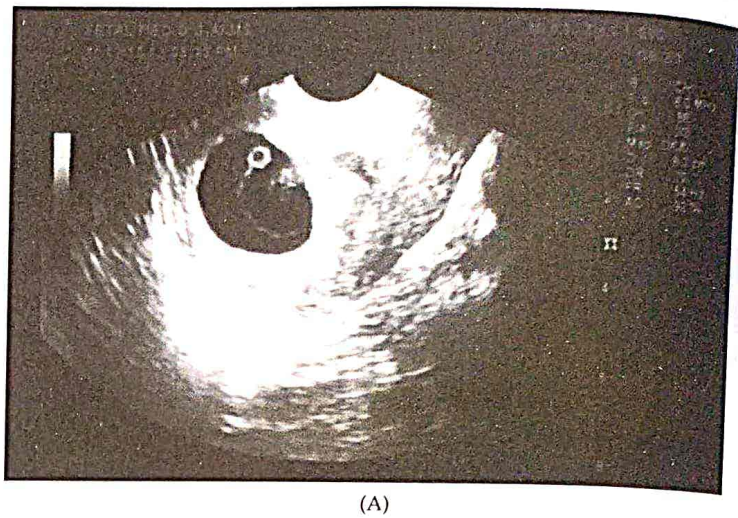
# INVESTIGATIONS

**Radiography.** A straight X-ray of the abdomen can help to confirm the diagnosis by demonstrating two foetal heads and two foetal spines in twin pregnancy (Fig. 33.2). Triplet pregnancy can also be seen. It may occasionally identify conjoined twins. With the availability of safer ultrasound, X-ray is not done in modern obstetrics.

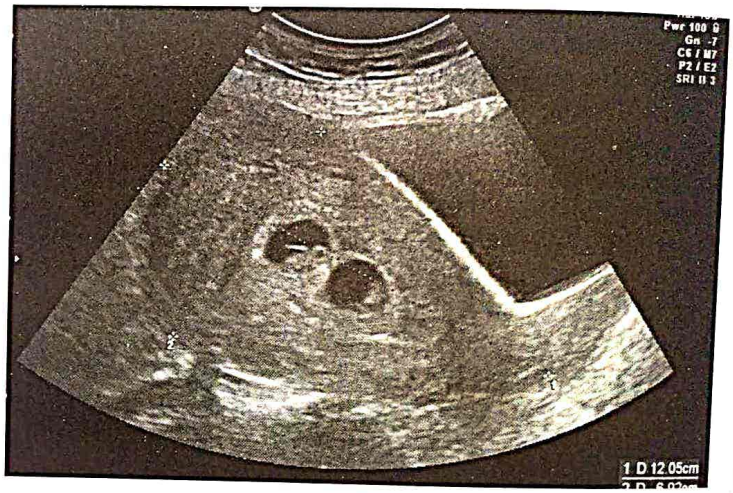
## Ultrasonography

Ultrasonography is an extremely important and diagnostic **modality of first choice** for multiple pregnancy. It is useful for:

1. Diagnosis of multiple pregnancy as early as 8th week of pregnancy. It can also demonstrate vanishing twins with foetal pole in one sac and no pole in the other sac (Fig. 33.3 A). Two gestational sacs are visible in normal twin pregnancy (Fig. 33.3 B). Later, two heads are seen on ultrasound (Fig. 33.4).
2. Foetal viability
3. Chorionicity of placenta
4. Gestation
5. Foetal anomalies
6. Foetal growth monitoring (at every 3–4 weeks interval) for foetal growth restriction
7. Presentation and lie of the fetuses.
8. Twin-to-twin transfusion syndrome
9. Amniotic fluid index of two sacs
10. Localization of placenta
11. Foetal well-being



(A)




(B)

**Fig. 33.3:** (A) Transabdominal scan showing two gestational sacs with foetal pole in one sac and no pole (vanishing twin) in the other sac; (B) Transabdominal ultrasound showing two gestational sacs and foetal poles in a case of twin pregnancy

Uncomplicated monochorionic twin pregnancies should have a first trimester scan and then every 2 weekly scan after 16 weeks to detect any twin to twin transfussion syndroma and twin arterial perfusion syndrome (TAPS).

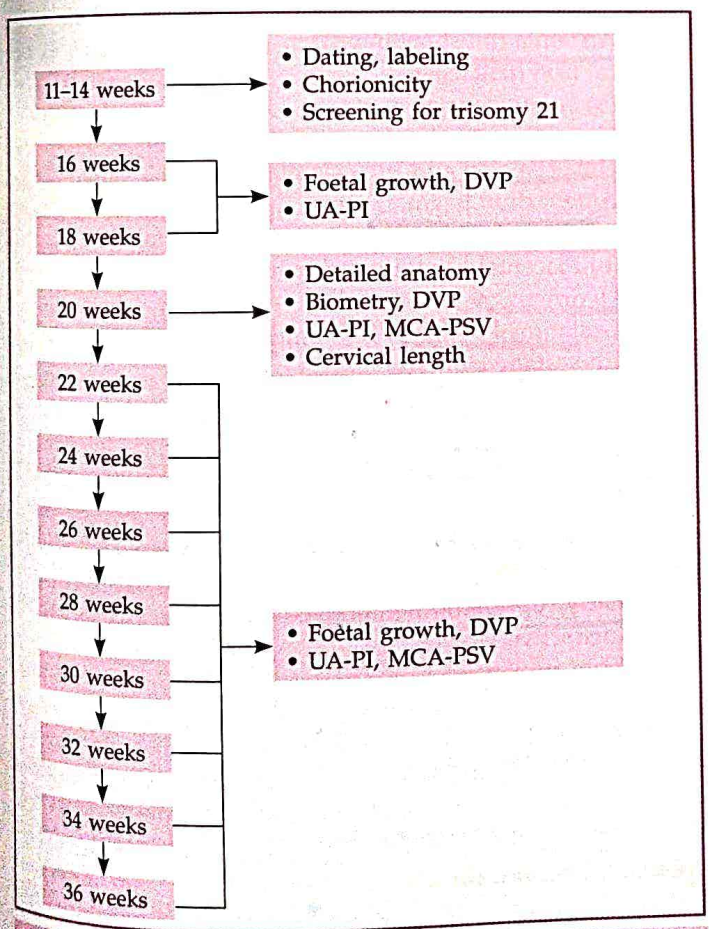
Best time to identify the type of twin pregnancy and chorionicity is 11–14 weeks, when chorion and amnion have not yet fused (at CRL between 45 and 84 mm). The presence of two separate placentas and a thick (generally 2 mm or greater) dividing membrane help in the diagnosis

of dichorionicity. Fetuses of opposite gender are almost always dizygotic.

In pregnancies in which a single placental mass is identified, examining the point of origin of the dividing membrane on the placental surface is helpful. If a triangular projection of placental tissue is seen to extend beyond the chorionic surface between the layers of the dividing membrane, then two fused placentas are actually present. This finding is termed the **'twin-peak'** sign or **lambda sign** and indicates dichorionic placenta (Figs. 33.5 A and C). Monochorionic pregnancies have a very thin (< 2 mm) dividing membrane and magnification reveals only two layers (Fig. 33.5 B). This right-angle relationship between the membranes and placenta with no apparent extension of placenta between the dividing membrane is called **'T' sign**. Ultrasonographic evaluation of the dividing membrane is the easiest and most accurate in the first half of pregnancy because the fetuses are smaller.

Women with uncomplicated dischorionic twin pregnancy should have a first trimester scan, a detailed second trimester anomaly scan and scan every 4 weeks thereafter.

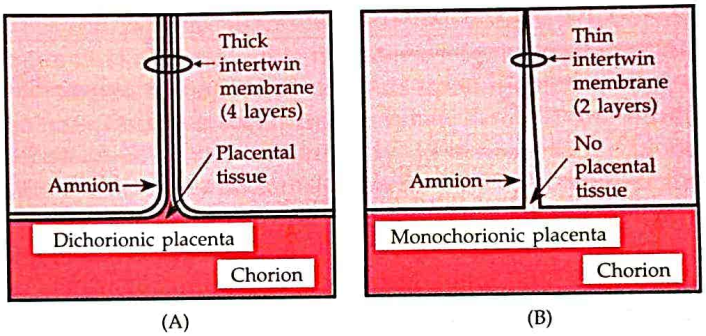
In DCDA twins foetal growth scans have to be repeated every 4 weeks and incase of MCDA twins every 2 weeks. Additionally in MCDA twins Amniotic fluid volume and Umbilical artery PI has to be assessed at 16 wks and MCA-PSV at 20 wks to screen for TAPS and TTTS.

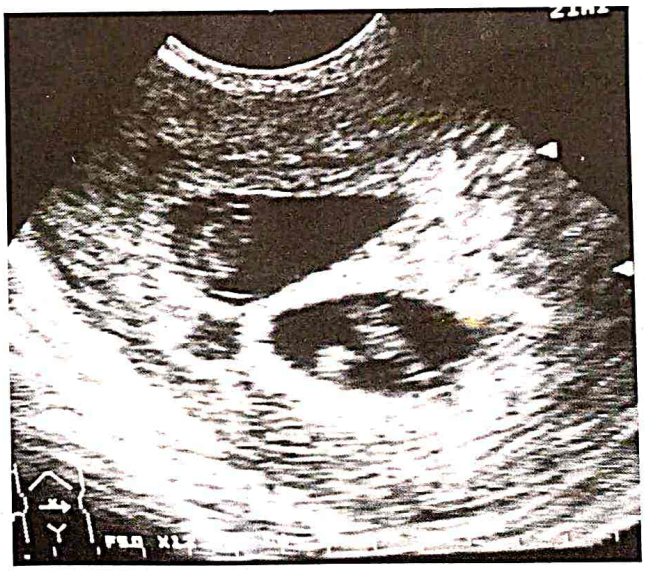



**Flow Chart 33.1A:** Ultrasound monitoring of uncomplicated monochorioni twin pregnancy (ISUOG* Guidelines, 2016)
*ISUOG: International Society of Ultrasound in Obstetrics and Gynaecology



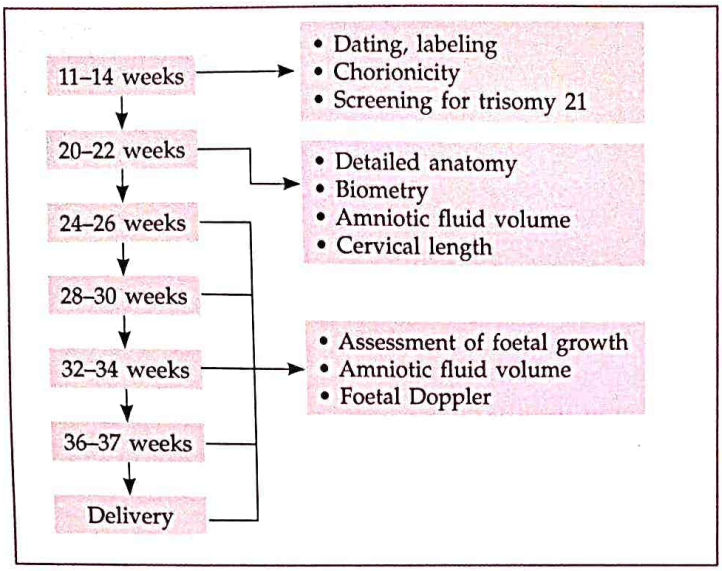

**Fig. 33.4:** Transabdominal ultrasound showing two heads in one field in a case of twin pregnancy



(C)

**Fig. 33.5:** (A) Diagramatic representation of 'twin peak' sign in dichorionic diamniotic twin pregnancy with extension of placental tissue into the inter twin membrane; (B) Diagramatic representation of monochorionic, diamniotic twins, the intertwin membrane is composed of two amnions only with no placental tissue; (C) Twin peak sign on ultrasound in a diamniotic-dichorionic twin pregnancy with extension of placental tissue between membranes. Kind courtesy: Dr Ashok Khurana, Delhi (for Fig. 33.5 C)




**Flow Chart 33.1B:** Ultrasound monitoring of uncomplicated Dichorionic twin pregnancy (ISUOG* Guidelines, 2016)
* ISUOG: International Society of Ultrasound in Obstetrics and Gynaecology

## DIFFERENTIAL DIAGNOSIS

1. Mistaken dates
2. Hydramnios
3. Foetal macrosomia
4. Fibroid with pregnancy
5. Ovarian tumor with pregnancy
6. Ascites with pregnancy
7. Full bladder
8. Hydatidiform mole
9. Closely attached adnexal mass

## EFFECTS ON MOTHER

The various maternal complications are shown in Table 33.2. There is an exaggerated adaptation of all body systems of the mother. The cardiac output is higher, the normal increase in plasma volume during pregnancy is also much greater. The hematocrit and hemoglobin are lower than in singleton pregnancy. The plasma protein levels are lower. Other differences include a slower rate of glucose disposal after a glucose load.

## EFFECTS OF MULTIPLE PREGNANCY ON FETUSES

The various foetal complications in multiple pregnancy are shown in Table 33.3 and important complications are discussed below.

### 1. Vanishing Twin

**When in a twin pregnancy, one of the fetuses aborts or gets reabsorbed within 10 weeks of pregnancy, it is called vanishing twin.** It is seen in up to 20–60% spontaneous twin conception. There may be little accompanying bleeding. It is diagnosed on ultrasound examination by

**Table 33.3: Foetal Risks in Multiple Pregnancy**

1. Abortion
2. Foetal growth restriction
3. Vanishing twin and single foetal death
4. Preterm labour and delivery
5. Stillbirth
6. Congenital malformations
7. Discordant growth
8. Conjoined twins
9. Monoamniotic twins
10. Twin-to-twin transfusion syndrome
11. Twin reversed arterial perfusion (TRAP)
12. Malpresentations
13. Stuck twin
14. Asphyxia
15. Neonatal death
16. Operative vaginal delivery (especially for second baby)
17. Twin entrapment
18. Twin anaemia-polycythemia sequence (TAPS)

**Table 33.2: Maternal Complications in Multiple Pregnancy**

**During Antenatal Period**

1. Miscarriage
2. Increased symptoms of early pregnancy (due to increased hCG levels)
3. Increased minor ailments of pregnancy (due to increased hCG levels)
4. Anaemia (most common)
   (a) Iron deficiency
   (b) Folate deficiency
5. Hyperemesis gravidarum (3 times)
6. Hypertension and pre-eclampsia (up to 25%)
7. Antepartum haemorrhage
   (a) Placenta previa (due to large placenta)
   (b) Placental abruption (due to pre-eclampsia)
8. Hydramnios
9. Gestational diabetes (2–3 times)
10. Cholestatic jaundice
11. Malpresentations
12. Preterm labour
13. Mechanical distress (varicose veins, dependent edema, hemorrhoids)
14. Obstructive uropathy

**During Labour**

1. Early rupture of membranes
2. Ineffective labour
3. Cord prolapse
4. Increased risk of operative delivery
5. Abruptio placentae (sudden shrinkage of uterus after delivery of first twin)
6. Postpartum haemorrhage (due to large uterus and more operative interference, retained placenta)

**During Puerperium**

1. Subinvolution of uterus
2. Infection
3. Failing lactation

observing a viable pregnancy accompanied by a non-viable one. When foetal death occurs during the second trimester, the remains of the baby get compressed and become paper like and flattened by pressure from the survivor **(fetus compressus or papyraceous)** (Fig. 47.62 in Chapter 47). *The vanishing twin can cause problems in screening for neural tube defects (elevated levels of α-fetoproteins in maternal serum and amniotic fluid) and a discrepancy in the karyotyping.* Monochorionic twins have a higher chance of abortion. In dichorionic twins, spontaneous abortion and loss of one or both twins should be kept in mind when diagnosing twins in very early pregnancy by transvaginal USG. Abortion rate is increased in twin gestation.

### 2. Prematurity

Preterm labour is the most important (almost 50%) complication of multiple pregnancies and the main reason for higher perinatal mortality. It can be due to uterine overdistension, hydramnios and intrauterine infections with or without premature rupture of membranes. It may be spontaneous or iatrogenic. Patients with twin-to-twin transfusion and those showing a discordant foetal growth may require a preterm delivery as soon as lung maturity is achieved. Cerebral palsy, microcephaly, porencephaly and multicystic encephalomalacia have been seen to occur more frequently in preterm twins than in preterm singletons. **The mean gestational age at delivery is 37 weeks for twins, 33.5 weeks for triplets, 30 weeks for quadruplets and 29 weeks for quintuplets.**

### 3. Growth Restriction

Growth restriction is more marked in monozygotic twins and occurs at and after 30 weeks of pregnancy. Almost 90% twins are low birth weight due to growth restriction (25%) or due to prematurity.

### 4. Discordant Twins

Discordant twins are due to unequal division of the zygote resulting in unequal placental mass, umbilical cord abnormalities, genetic diseases, twin-to-twin transfusion or placental insufficiency. The difference of birth weight is 25% or more amongst the twins (expressed as a percentage of the larger twin's weight) is termed as discordant growth. **The smaller twin has a higher risk of perinatal complications, long-term physical and intellectual growth restriction and risk of death.**

#### Pathology

In monochorionic twins discordancy is usually due to placental vascular anastomosis that cause hemodynamic imbalances between twins. Occasionally discordancy can be due to structural anomalies.

Dizygotic twins may be due to different genetic potential, one of the placenta may have suboptimal implantation site, in utero crowding and placental abnormalities.

The criteria for diagnosis on ultrasound examination are as follows:

(a) A difference in the head circumference of > 5%.
(b) A difference in the abdominal circumference of > 20 mm.
(c) Increased head to abdomen and femur to abdomen ratios.
(d) A difference of > 25% in estimated foetal weight.
(e) Abnormal umbilical artery Doppler waveforms.

### 5. Single Foetal Death (Acute Intertwin Transfusion)

Single foetal death may occur either early in pregnancy or later as the pregnancy progresses. However, it is uncommon. Morbidity of the surviving fetus depends to a great extent on the chorionicity of the pregnancy. When one monochorionic twin dies in utero, there is a 25% risk of necrotic neurological lesions including cerebral palsy and renal lesions in the survivor. There is high (25%) risk of intrauterine death of the healthy co-twin. These complications are usually due to consequence of severe hypotension occurring during the death of the other twin.

#### Treatment of Co-twin Death

In dichorionic pregnancies, expectant management is indicated. Regular assessment of the pregnant woman's coagulation status is necessary. In monochorionic pregnancies, management depends upon the gestation and the time since the foetal death. When death has occurred within 24–36 hours at an early gestation, foetal blood sampling with rescue transfusion may be considered if the surviving fetus is anaemic. At later gestations, delivery may be more appropriate.

If a co-twin death has occurred some time previously, appropriate imaging of the surviving fetus's brain with either ultrasound or magnetic resonance imaging (MRI) to detect cystic changes is advisable. Cystic changes may warrant termination of pregnancy.

### 6. Twin-twin (or Twin-to-twin) Transfusion Syndrome (TTTS) (Chronic Intertwin Transfusion)

Twin-twin transfusion syndrome is a condition that complicates up to 15% monochorionic twin pregnancies. It is characterized by a relative lack of superficial bilateral vascular anastomoses which protect against hemodynamic imbalances caused by unidirectional deep arterio-venous vessels.

Antenatal criteria recommended for defining the twin-twin transfusion syndrome include the following.

1. Same sex fetuses
2. Monochorionicity with placental vascular anastomoses
3. Weight difference between twins greater than 20%
4. Hydramnios in the larger twin, oligohydramnios in the smaller twin
5. Stuck smaller twin
6. Hemoglobin difference > 5 g/dL.

All of these criteria, except hemoglobin levels, can be determined ultrasonographically.

Quintero *staging system* for twin-twin transfusion syndrome.

Stage I – Polyhydramnios, oligohydramnios (Poly-oli). Deepest liquor pocket ≥ 2 cm syndrome polyhydramnos, deepest liquor pocket < 2 cm: oligohydramnios. Discordant amniotic fluid volume but urine still visible ultrasonographically within donor twin's bladder.

Stage II – Stage I + but urine not visible within donor's bladder.

Stage III – Stage II + abnormal Doppler studies of umbilical artery, ductus venousus or umbilical vein.

Stave IV – Ascites or frank hydrops in either twin.

Stage V – It is most severe form with demise of either twin.

If untreated, the perinatal mortality is extremely high (> 80%). Although twin-twin transfusion is usually a gradual process, it can happen suddenly with the death of one twin, usually the recipient, due to heart failure. **Hence, recipient twin is at higher risk of death than donor.**

**Treatment options.** They are as follows:

1. Amnioreduction treats the polyhydramnios and has a success rate of 60–65%. It is indicated when recipient twin AFI > 40 cm or MVP > 12 cm. The target amniotic fluid index should be kept 15 cm mandating repeated amnio-reductions at longer intervals.
2. Septostomy (making hole in septum) aims to equilibrate the discordant amniotic fluid between the two sacs with a high success rate (up to 85%).
3. Selective foeticide of the donor twin incase of severe FGR can be done using cord ligation. Recipient twin can also be subjected to foeticide by ultrasound guided sclerosant injection, laser coagulation or bipolar diathermy as they have more neurological sequelae and more chances of cardiac dysfunction
4. Laser treatment (using Radio-frequency ablation with Soloman technique) at 18-24 weeks includes ablation of communicating anastomoses with a high survival rate.

However, these procedures are rarely performed in only tertiary referral centers with facilities of foetal medicine and surgery.

## 7. Twin anaemia-polycythemia sequence (TAPS)

It occurs due unbalanced intertwine transfusion across small peripheral arteriovenous anastomosis.

### Diagnostic criteria

**Antenatal criteria:** MCA-PSV > 1.5 MoM in the donor and MCA-PSV < 1.0 MoM in the recipient

**Postnatal criteria:** Interwin Hb difference > 8.0 g/dl and at least one of the following:

- Reticulocyte count ratio > 1.7
- Placenta with only small (diameter < 1 mm) vascular anastomoses

### Staging

| Staging | Antenatal staging | Postnatal |
|---|---|---|
| Stage 1 | MCA-PSV donor > 1.5 MoM and MCA-PSV recipient < 1.0 MoM, without other signs of foetal compromise. | > 8.0 |
| Stage 2 | MCA-PSV donor > 1.7 MoM and MCA-PSV recipient < 0.8 MoM, without other signs of foetal compromise. | > 11.0 |
| Stage 3 | as stage 1 or 2, with cardiac compromise of donor, defined as critically abnormal flow* | > 14.0 |
| Stage 4 | hydrops of donor | > 17.0 |
| Stage 5 | intrauterine demise of one or both fetuses preceded by TAPS | > 20.2 |

*Critically abnormal Doppler is defined as absent or reversed end-diastolic flow in umbilical artery, pulsatile flow in the umbilical vein, increased pulsatility index or reversed flow in ductus venosus.

### Treatment

Intra-uterine blood transfusion for the anaemic twin and partial exchange transfusion for polycythemic twin. Foetal blood sampling post delivery to diagnose anaemia.

## 8. Twin reversed arterial perfusion (TRAP) sequence and Acardiac Twinning

Twin reversed arterial perfusion sequence and acardiac twinning is a complication which occurs in approximately 1 % monochorionic pregnancies. It is characterized by an acardiac twin which receives its blood supply via a large arterio-arterial anastomosis from a normal 'pump' co-twin. This results in absent or rudimentary development of the upper body structures like head or neck. The perinatal mortality of the pump twin is high with death usually occurring from cardiac failure, hydrops or polyhydramnios-induced preterm delivery.

In cases of TRAP, disruption of the acardiac twin's cord is the treatment of choice. However, if this is technically challenging, diathermy or laser treatment of the central vessel may be attempted.

## 9. Monoamniotic twin pregnancies

Monoamniotic twin pregnancies are rare, occurring once in every 12,500 births. The main factors associated with perinatal mortality are umbilical cord entanglement, cord accidents, congenital malformations, preterm delivery and twin-to-twin transfusion.

Ultrasonographic diagnosis is based on the absence of a dividing amniotic membrane and twins of the same sex with the presence of a single placenta. Colour Doppler examination is valuable in detecting umbilical cord entanglement. Presence of variable decelerations is also suggestive of cord entanglement. The optimal route of delivery is by cesarean section after giving a course of two doses of Betamethasone after 34 weeks of gestation to avoid umbilical cord problems.

## 10. Congenital Malformations

The incidence of congenital malformations is significantly increased in twins and higher order multiple gestations compared with singleton pregnancy. Major malformations develop in 2% and minor malformations in 4% twins. This increase is almost entirely due to high incidence of structural defects in monozygotic twins and fall into one of the three following categories.

(a) **Defects resulting from twinning itself** (a teratogenic event). This category includes conjoined twinning, acardiac anomaly, sirenomelia (fusion of lower extremities), neural tube defects and holoprosencephaly.

(b) **Defects resulting from vascular interchange** between monochorionic twins can give rise to dramatic blood pressure fluctuations causing defects such as microcephaly, hydranencephaly, intestinal atresia, aplasia cutis and limb amputation.

(c) **Defect occurring as a result of crowding** like talipes equinovarus (clubfoot) or congenital hip dislocation. Dizygotic twins are also subjected to overcrowding.

## 11. Conjoined twins

Conjoined twins, also called Siamese twins, after Chang and Eng Bunker of Siam (Thailand). They were displayed worldwide by P T Barnum of USA.

They affect 1 in every 200 monozygotic twins. Conjoined twins are uniovular and have the same sex and karyotype. The different types of conjoined twins are:

(a) **Thoracopagus:** Fusion at the chest (40%). It is the most common type (Fig. 33.6).

(b) **Omphalopagus (xiphopagus):** Fusion at the anterior abdominal wall (33%).

(c) **Pyopagus:** Fusion at the buttocks (18%).

(d) **Ischiopagus:** Fusion at the ischium (6%).

(e) **Craniopagus:** Fusion of heads (2%). It is least common.

The outcome of conjoined twins is poor and is dependant largely on the feasibility of surgical separation.

Antenatal USG shows both twins facing each other or on repeated examination heads are at same level and plane. The thoracic cages are in unusual proximity. There is no change in relative foetal positions with time or manipulation. A three dimensional (3D) USG can diagnose the condition very early in pregnancy (see **Fig. 46.2 D** in Chapter 46). An X-ray may be performed with radio-opaque dye in amniotic fluid to confirm diagnosis.

Cesarean section offers a safe method of delivery for the mother and should be done where the diagnosis has been made. Spontaneous delivery may occur in extreme premature cases. Usually, obstructed labour is encountered. Newborns are looked after by neonatologist and usually need separation operations.

## 12. Umbilical cord problems

Umbilical cord problems are more frequent in twin pregnancies and include single umbilical artery and velamentous insertion of the cord. Cord entanglements, cord prolapse and torsion due to focal absence of Wharton's jelly are more common in monoamniotic twins.

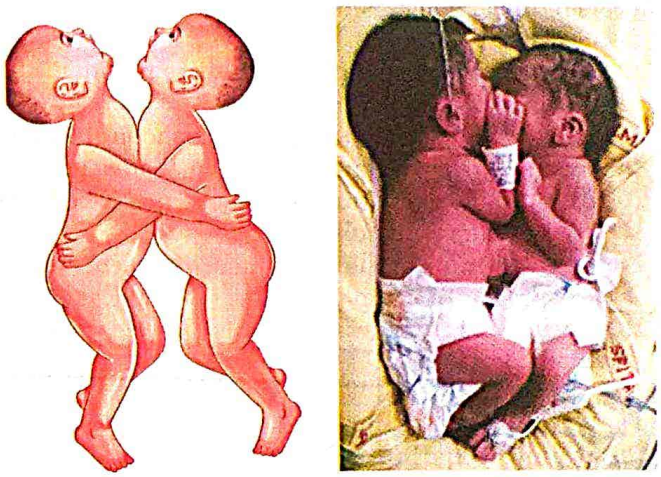


**Fig. 33.6:** (A) Conjoined twins; (B) Clinical photograph of conjoined twins

## 13. Malpresentations

There is a high incidence of foetal malpresentations at the time of delivery in twin gestations. The frequency of different foetal presentations reported is as follows:

| | | |
|---|---|---|
| (a) Vertex-vertex | : | 40% (most common) |
| (b) Vertex-breech | : | 28% |
| (c) Vertex-transverse | : | 7.5% |
| (d) Breech-vertex | : | 9% |
| (e) Breech-breech | : | 6.5% |
| (f) Breech-transverse | : | 2.5% |
| (g) Other combinations | : | 6% |
| (h) Both transverse (0.5%, least common) | | |

Malpresentations are more common in the second fetus. During labour, early rupture of membranes and cord prolapse are likely to occur due to a higher prevalence of malpresentations.

Usually, post maturity and macrosomia are not seen in multiple pregnancy.

# PREVENTION OF MULTIPLE PREGNANCY

Placing three instead of two embryos increase the risk of triplets by 15-fold, from 0.4% to 6% with no increase in the take home baby rate. Legislation to allow only one or two embryo transfer can help to reduce incidence of multiple pregnancies. Similarly, avoiding high risk factors like minimizing use of ovulation induction can lower rates.

# MANAGEMENT DURING ANTENATAL PERIOD

Successful outcome of a twin pregnancy depends on an early diagnosis. High index of clinical suspicion and liberal use of ultrasound examination are the keys to the diagnosis.

## Antenatal Advice and care

1. **Nutrition.** An additional 300 K calories per day dietary supplementation should be advised. Requirement of

protein, minerals, vitamins and essential fatty acids is also increased.

2. **Extra rest** and early stopping of work is recommended to prevent preterm labour.
3. **Iron and other supplements.** (a) She should be given therapeutic dose (200 mg) of elemental iron per day. (b) Extra vitamins, calcium should be prescribed. She should be given 5 mg folic acid per day.
4. **Antenatal visits** should be more frequent (every 2 weekly) to pick up pregnancy complications at the earliest. A minimum of 8 antenatal visits are recommended for DCDA twins and 11 visits for MCDA twins.
5. **Foetal monitoring** is done by 4 weekly ultrasounds for foetal growth and amniotic fluid volume. Non-stress test, Doppler study and biophysical score (Manning's score) are performed regularly. Biophysical score is best for dichorionic diamniotic twin pregnancy.

### Admission to hospital

1. Routine hospitalization for the critical period and for bed rest (as was practice in the past) has not been shown to be helpful.
2. Generally woman with multiple pregnancy are advised rest at home from about 30 weeks to avoid prematurity. Bed rest in left lateral position may increase placental blood flow for better foetal growth and decrease pre-eclampsia but its role is doubted in recent studies.
3. Evidence based medicine does not support prophylactic cervical cerclage or routine sympathomimetics (e.g. ritodrine) to prevent preterm labour in multiple pregnancy.
4. Progestogen therapy: There is no evidence of use of 17-alpha hydroxyprogesterone ceproate (17–0 HP-C) in multiple pregnancy to lower incidence of preterm birth in women without short cervix. Even in short cervix it didn't lower preterm birth risk in studies. There is some evidence that vaginal progesterone (200 mg) may reduce preterm delivery rate before 30 weeks of pregnancy in women with twin pregnancy with short cervix though it is not universally accepted.
5. **Role of pessary.** Silicone Arabin pessary which encircles and compresses the cervix in women with twin pregnancy with short cervix at 18–20 weeks of pregnancy did not reduce preterm delivery rate.
6. Guidelines for the use of corticosteroids are same for multiple pregnancy as for singleton pregnancy (American College of Obstetricians and Gynecologists, 2016).
7. Limited physical activity, early work leave, more frequent antenatal visits and ultrasonographic examination for cervical length and maternal education on the risks of preterm delivery have been advocated to reduce preterm births with little evidence.
8. Emergency admission is needed in case of complications.

### Management of uncomplicated diamniotic twins

It is shown in Flow Chart 33.1B.

## LABOUR MANAGEMENT

As per the NICE guidelines , the timing for planned birth should be:

| Type of twin | Recommended termination |
|---|---|
| DCDA Twins | 37 weeks |
| MCDA Twins | 36 weeks |
| MCMA Twins | $32^{+0}$ to $33^{+6}$ weeks |
| TTS syndrome | $32^{0+}$ to $33^{+6}$ weeks |
| Triplet | 35 weeks |

Patients with multiple pregnancy should preferably be taken care of in tertiary health care centers with anesthesia services and intensive neonatal care unit facilities. It is given in Flow Chart 33.2.

During labour the following precautions are to be taken.

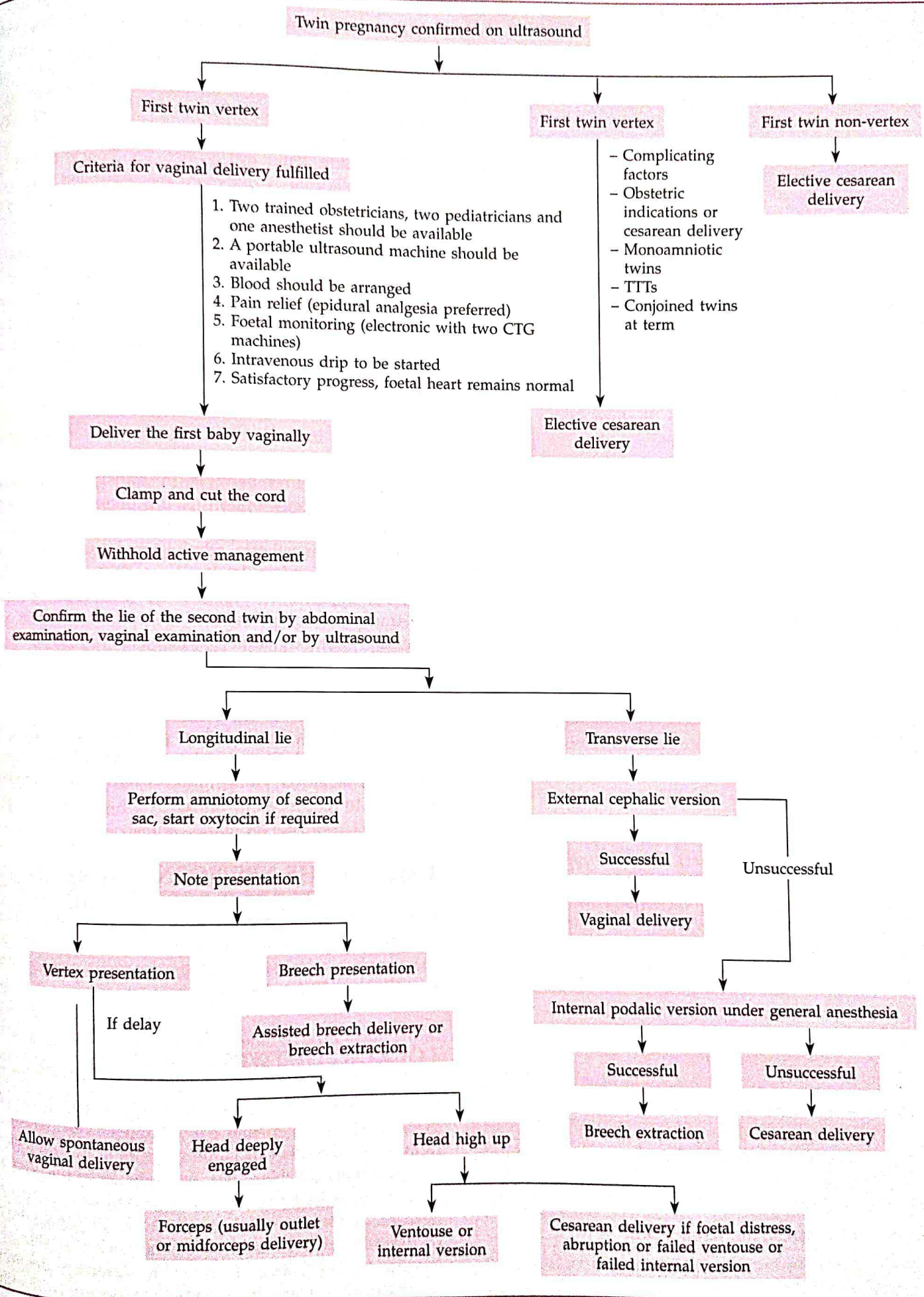
1. The patient should be kept nil by mouth to avoid anesthesia complications in the event of operative interference.
2. Adequate caloric supplementation with intravenous fluids using Ringer lactate or dextrose saline solution.
3. Blood should be crossmatched and kept ready.
4. Continuous intrapartum foetal monitoring using two CTG machines is preferable to detect any signs of foetal distress.
5. An experienced obstetrician skilled in intrauterine identification of foetal parts and in intrauterine manipulations of the fetus should be available at all times.
6. Pain relief, if required, can be given. Epidural anesthesia is ideal as second fetus may need intra-uterine manipulations for delivery. An experienced anesthetist should be kept informed and should be available.
7. Two neonatologists should preferably be present at the time of delivery.

### Delivery of the First Twin

The delivery of the first baby is conducted as in normal labour. A generous episiotomy should be made under local perineal infiltration. An outlet forceps, if needed, should be applied preferably under pudendal block analgesia. Intravenous ergometrine or methylegometrine is contraindicated with delivery of the anterior shoulder of the first baby. Intra muscular or intravenous oxytocin for active management of third stage is withheld. The cord should be clamped at two places and cut in between and kept long for administration of drugs or blood transfusion, if needed. The baby is handed over to the attending neonatologist and labelled as the first twin.

### Delivery of the Second Twin (Flow Chart 33.2)

After first baby is born, abdomen is carefully palpated to know the lie and presentation of the second twin. An ultrasound may be used to confirm the presentation and for foetal heart sounds. A vaginal examination is performed




**Flow Chart 33.2:** Intrapartum management of twins during labour

gently for confirmation of presentation, membrane status and to rule out cord presentation and cord prolapse.

1. For longitudinal lie and vertex presentation with head in pelvis, amniotomy is performed. If there is uterine inertia, a controlled intravenous oxytocin infusion can be used to augment labour. In 80% cases, the second twin is born within 30 minutes of the first. As long as the foetal heart rate remains normal, there is no reason to expedite delivery. If vaginal bleeding occurs suggesting abruptio placentae or there are late decelerations indicating foetal distress or there is cord prolapse, delivery should be conducted immediately by forceps or cesarean section.
2. For longitudinal lie with breech presentation, with good foetal heart condition, amniotomy is done and assisted breech delivery is performed. For foetal distress or placental abruption, breech extraction is done.
3. When the lie is transverse or oblique, an external cephalic version should be performed to convert it into longitudinal lie and vertex presentation followed by amniotomy when head is in pelvis and vaginal delivery is accomplished. If membranes are ruptured or if external cephalic version cannot be done or fails or there is foetal distress or abruption, an internal podalic version should be performed preferably under general anesthesia in operation theatre, followed by breech extraction. ***Infact, an internal podalic version done by an expert for the transverse lie of the second twin is the only acceptable indication for internal podalic version in a live fetus in modern obstetrics.*** Indications of cesarean delivery are given in Table 33.4.

**Table 33.4: Indications of Cesarean Delivery in Twin Pregnancy**

| Indications of Elective Cesarean Delivery |
|---|
| 1. First baby in non-cephalic, especially shoulder presentation<br>2. Conjoined twins<br>3. Congenital abnormality precluding safe vaginal delivery<br>4. Foetal growth restriction in dichorionic twin<br>5. Chronic twin-twin transfusion syndrome in monochorionic twins<br>6. Monoamniotic twins (high risk of cord prolapse or entanglement and foetal deaths)<br>7. Placenta previa<br>8. Contracted pelvis<br>9. Previous cesarean section<br>10. Severe pre-eclampsia |
| **Indications of Emergency Cesarean Delivery** |
| 1. Foetal distress<br>2. Cord prolapse in first baby<br>3. Non-progress of labour<br>4. Collision of both twins |
| **Indications of Cesarean for Second Twin** |
| 1. Second twin transverse, both external cephalic and internal podalic version failed after delivery of first twin<br>2. For a large second twin weighing more than 3.5 kg.<br>3. Prompt closure of cervix after delivery. |

### Management of the Third Stage of Labour

These women are at high risk of postpartum haemorrhage due to large placenta and bigger placental site, overdistension of uterus, anesthesia and uterine manipulations. Active management of third stage is practised by administration of 10 units of oxytocin intramuscularly or intravenously after delivery of the second baby (rule out triplet pregnancy before giving it). The placenta is delivered by controlled cord traction method. It is better to continue oxytocin drip for 1–2 hours and carefully observe the patient

If there is PPH, the uterus should be massaged continuously and intravenous oxytocin infusion drip should be administered simultaneously. The use of 15-methylprostaglandin $F_2$ alpha (carboprost 250 µg IM) intramuscularly is preferred immediately after delivery of the placenta. Blood transfusion may be needed.

### Cesarean Delivery

The various indications of cesarean delivery are given in Table 33.4.

When the first baby is in transverse lie, cesarean section is the only option. If the presentation is breech, most obstetricians favor cesarean delivery.

Epidural anesthesia administered by an experienced anesthetist is preferable. It allows delivery of the babies without hypoxia or depression which could be caused by transplacental passage of general anaesthetic agents. Maternal hemodynamic parameters should be closely monitored.

### Postpartum

Special attention is needed in puerperium because there can be subinvolution, greater chances of infection and failure of lactation. Cooperation of all family members is essential to rear multiple births.

## CARE OF THE NEWBORNS

Care of the newborns is according to their birth weight and gestation. They are mostly treated as preterm neonates and may require admission or more frequent care in nursery (see Chapter 28 for details).

## LOCKING OF TWINS

Locking of twins is a rare situation (occurring 1 in 8000 twin gestations) in which one baby impedes the descent and delivery of the other. It is more common in primigravidas and in smaller babies. For twins to lock, the first fetus must present as breech and the second as cephalic. With descent of the breech through the birth canal, the chin of the first fetus locks between the neck and chin of the second cephalic fetus (**Fig. 33.7**). Cesarean delivery is recommended when the potential for locking is identified.

If the first baby is dead, decapitation of the first baby, pushing up the decapitated head followed by delivery of

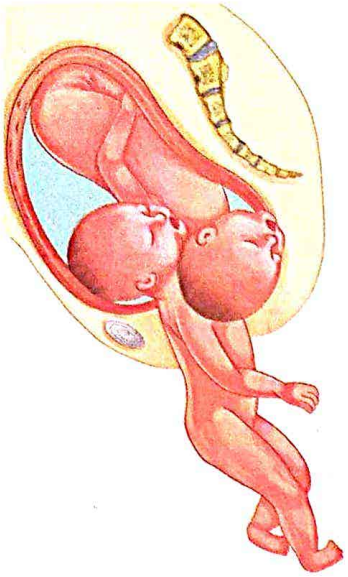


**Fig. 33.7:** Interlocking of twins (first breech, second cephalic)

the second baby is done. Thereafter, the decapitated head is delivered. In situations with less experience with such procedures, a cesarean section may be safer for the mother.

Rarely, interlocking may occur if there are both cephalic presentations with the head of second twin trying to descend into the pelvic cavity along with the first resulting into collision thus impeding the progress. Disengagement can be tried under anesthesia failing which cesarean delivery should be done.

Occasionally, interlocking may occur if one fetus is longitudinal and the second fetus is in an oblique or transverse presentation. It is diagnosed when difficulty is encountered in delivery of the first child. By careful internal examination under anesthesia, the second child is pushed out of the pelvis and if possible, the first child is extracted. Otherwise cesarean section should be performed.

## MANAGEMENT OF TRIPLETS AND OTHER HIGH ORDER MULTIPLE PREGNANCIES

There is excessive distension of abdomen **(Fig. 33.8)** and increased frequency of all the maternal and perinatal complications than twin pregnancy. Mean gestation in triplets is 34 weeks.

Cesarean delivery is recommended with triplets and quadruplets mainly because of difficulties in ensuring adequate monitoring in labour. These fetuses are also more premature **(Fig. 33.9)**, growth retarded and have malpresentations. There is increased risk of malpositions, cord prolapse, reduced placental perfusion and haemorrhage in them. The foetal mortality and morbidity are closely related to the foetal weight, birth order and foetal position.

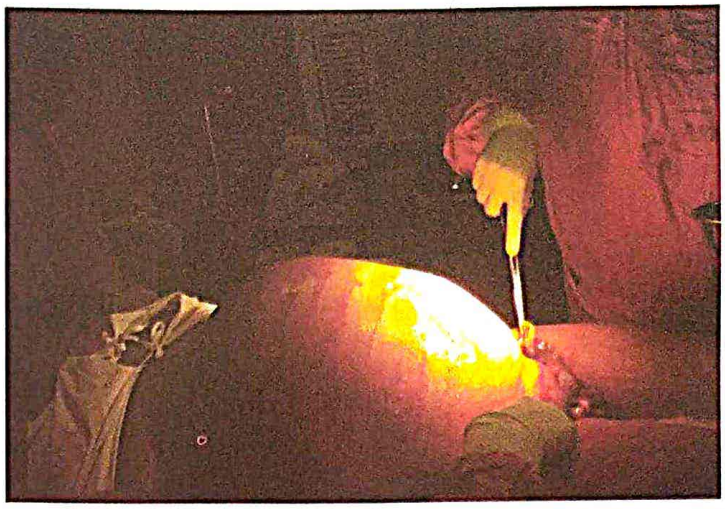


**Fig. 33.8:** Excessive distension of abdomen in Triplet pregnancy at 32 weeks period of gestation



**Fig. 33.9:** Newly born triplets delivered by cesarean section by the author at 35 weeks gestation (1.8 kg, 2.1 kg and 1.8 kg)

**Selective foetal reduction** of high order multifetal pregnancies has developed as an alternative. Between 10 and 13 weeks gestation the fetuses to be reduced receive intracardiac injection of 1–2 mEq of potassium chloride under ultrasound guidance. Reduction to twin pregnancy is associated with lower rate of maternal complications, preterm delivery and neonatal deaths as compared to expectant management of triplet and high order pregnancies. However, there is a 30% likelihood of losing one of the fetuses. Selective foetal reduction is contraindicated in twin pregnancies.

**Selection termination.** In a multifetal pregnancy an anomalous fetus with structural or genetic abnormality can be selectively terminated using bipolar umbilical cord coagulation or radio frequency ablation at midpregnancy after explaining risks to the patient (ACOG 2017)

## Key Points

1. Multiple pregnancy is the simultaneous development of two or more fetuses in the uterus. The incidence of multiple pregnancies is about 1 in 80 and rising due to assisted reproductive technology.
2. Determination of chorionicity is important to allocate pregnancy risk. Prenatal diagnosis using amniocentesis or CVS is suitable in multiple pregnancy.
3. Cervical length measurement may be useful in predicting preterm birth in multiple pregnancy **(level II evidence)**.
4. Maternal complications include higher incidence of anaemia, pre-eclampsia and postpartum haemorrhage. The intrapartum complications in twin pregnancy include abnormal presentations, uterine inertia, cord prolapse, abruptio placentae, interlocking of twins and postpartum haemorrhage.
5. Foetal complications in twin pregnancy include increased incidence of miscarriage, congenital malformations, malpresentations, twin-to-twin transfusion syndrome and low birth weight. Monochorionic monoamniotic placentation may be associated with twin-to-twin transfusion syndrome, twin reversed arterial perfusion (TRAP) and conjoined twins and need management in a higher center.
6. Antenatal management includes more frequent antenatal visits, higher dose of iron (200 mg elemental) and folic acid (5 mg) per day, regular ultrasound examination 3-4 weekly for foetal growth and well-being, foetal surveillance by NST and biophysical scoring.
7. Multiple pregnancy is a high-risk pregnancy and should be managed in a tertiary referral or specialized unit.
8. Vaginal delivery is aimed for if the first twin is presenting by vertex. However, cesarean delivery is recommended if first twin is presenting by breech or transverse lie or other malpresentation or for obstetric indications.
9. After vaginal delivery of the first twin, the presentation of second twin is confirmed. For vertex and breech, vaginal delivery is performed after amniotomy and oxytocin administration. If second twin is presenting by transverse or oblique lie then external version is performed failing which internal podalic version and breech extraction are performed. Infact it is the only indication of internal podalic version for a live fetus in modern obstetrics.
10. The mothers are at high risk of PPH which should be prevented by active management of third stage (using 10 units of oxytocin IM) after delivery of all fetuses. PPH should be energetically treated if it occurs.

## Suggested Reading

1. American College of Obstetricians and Gynecologists: Multifetal Gestations: Twin, Triplets and Higher Order Multifetal Pregnancies: ACOG Practice Bulletin No. 231, June 2021.
2. American College of Obstetricians and Gynecologists: Multifetal Pregnancy Reduction Committee Opinion No. 719, September 2017.
3. Quintero R A, Morales W J, Allen M H et al. Staging of Ttwin-twin Transfusion Syndrome. Journal of Perinatology 1999; 19: 550.
4. Royal College of Obstetricians and Gynaecologists: Management of Monochorionic twin pregnancies. Greentop guideline no. 51, London: RCOG; 2008.
5. Management of twin and triplet pregnancy in the antenatal period. NICE Clinical Guideline 129, National Institute of Health and Care Excellence (NICE), London, 2011.
6. Romero R et al. Vaginal progesterone decreases preterm birth and neonatal morbidity and mortality in women with twin gestation and short cervix: an updated meta-analysis of individual patient data. Ultrasound Obstet Gynecol. 2017; 49: 1303.
7. Society for Maternal Foetal Medicine, Simpson LL. Twin-twin transfusion syndrome. Am J Obstet Gynecol 2013; 208 : 13.

## Examination Questions

A. Define multifetal pregnancy. Briefly discuss various types of twin pregnancy. How will you manage a case of twin pregnancy during antenatal period? (Delhi University)

B. Enumerate various etiological causes and maternal and perinatal complications in multiple pregnancy. How will you monitor the foetal surveillance during pregnancy in a twin pregnancy?

C. How will you manage a primigravida with twin pregnancy at 38 weeks? Enumerate indications of cesarean delivery in multiple pregnancies.

D. Write short notes on:

1. Twin-to-twin transfusion syndrome
2. Discordant twins
3. Interlocking of twins
4. Conjoined twins
5. Vanishing twin
6. Hellin's hypothesis
7. Determination of zygosity in twin pregnancy
8. Differences between monozygotic and dizygotic twins
9. Superfetation
10. Superfecundation
11. Acute inter-twin transfusion
12. Twin reversed arterial perfusion (TRAP) sequence
13. Complications of monoamniotic twin pregnancy
14. Delivery of second twin (Many universities)

# Preterm Labour, Foetal (Intrauterine) Growth Restriction, Intrauterine Death and Postmaturity

CHAPTER 34

## PRETERM LABOUR (PREMATURE LABOUR)

Preterm birth is a leading cause of neonatal mortality and morbidity. It causes significant loss of economy throughout the world as it is very expensive to look after premature babies.

### Definition

American College of Obstetricians and Gynecologists (ACOG) has defined preterm labour as the onset of labour (regular painful uterine contractions associated with effacement and dilatation of the cervix) prior to the completion of 37 weeks (259 days) of gestation (< 37 weeks) counting from the first day of the last menstruation period (LMP) and after the attainment of period of viability . The lower limit varies in different countries being 20–24 weeks in developed countries (22 weeks and 500 g by WHO, 24 weeks in the United Kingdom) and 28 weeks in developing countries including India.

**Preterm (or premature) infant**
Infant born before 37 completed weeks of gestation.

**Late preterm infant (a recently identified category)**
Infant born between 34 and 36 weeks of gestation.

**Moderately preterm infant**
Infant born between 32 and 34 completed weeks of gestation.

**Very preterm infant**
Infant born before 32 completed weeks of gestation.

### Incidence

The incidence of preterm labour varies from 5–15% in different parts of the world and India.

**False labour** and **prelabor.** They have been described in Chapter 16.

### Threatened preterm labour

Threatened preterm labour is defined as the documented uterine contractions with no evidence of cervical change.

### Etiology

Exact etiology is not known in most cases and may be multifactorial. The various high risk factors and causes of preterm labour are shown in Table 34.1.

### Pathogenesis

The exact mechanism is not understood, but the fetus appears to play a synergistic role along with maternal factors like infection and overdistension of uterus as shown in Fig. 34.1.

### Prediction of preterm labour

**Risk factors**

1. Low socio-economic status
2. Low BMI (< 19)
3. Extremes of age
4. Unsupported mother
5. Illicit drug use
6. Smoking
7. Heavy physical activity
8. Previous history of preterm birth
9. Congenital anomalies of uterus

**Symptoms and Signs**

1. Uterine contractions > 4/hr
2. Cervical dilatation > 2 cm
3. Cervical effacement > 80%
4. Bishop's score > 4
5. Vaginal bleeding

**USG Parameters**

| Cervical length | Risk of PTB |
|---|---|
| < 2.5 cm | 17.8% |
| 1.5–2.5 cm | 35% |
| < 1.5 cm | 50% risk before 32 weeks |
| | 90% risk before 34 weeks |

**Table 34.1: Risk Factors and Causes of Preterm Labour**

| |
|---|
| **1. Socio-economic Factors** |
| (a) Lower socio-economic status<br>(b) Extremes of age, young (< 16 years) and high maternal age<br>(c) Unmarried or unsupported mother<br>(d) Underweight (body mass index < 19 kg/m$^2$)<br>(e) Smoking<br>(f) Illicit drug intake<br>(g) Increased physical activity |
| **2. Genital Tract Infection or Colonization** |
| (a) *Chlamydia trachomatis*<br>(b) *Neisseria gonorrhoeae*<br>(c) *Bacterial vaginosis*<br>(d) *Trichomonas vaginalis*<br>(e) *Group B Streptococcus*<br>(f) *Gardnerella vaginalis*<br>Bacteria produce lipopolysachherides and other toxins which activate toll like receptors on decidua and other parts producing interleukin 8 and cytokines like interleukin 1β, IL6, IL8 and tumor necrosis factor (TNFα) which cause cervical ripening and uterine activity. |
| **3. Systemic Infection** |
| Chorioamnionitis—In certain unexplained cases of preterm labour, chorioamnionitis caused by a variety of microorganisms has emerged as a possible explanation |
| **4. Congenital Abnormalities of the Uterus** |
| Septate, unicornuate or bicornuate uterus, arcuate uterus |
| **5. Incompetence of Cervix (Cervical Weakness)** |
| Following cervical conization or previous second trimester spontaneous or induced miscarriage. |
| **6. Maternal Systemic Diseases** |
| (a) Renal diseases like asymptomatic bacteriuria, cystitis and pyelonephritis<br>(b) Hypertensive disorders such as pre-eclampsia or hypertension; spontaneous or iatrogenic prematurity<br>(c) Maternal diabetes—In maternal diabetes there may be hydramnios and this may contribute to preterm labour.<br>(d) Immunological disorders such as SLE, anti-phospholipid syndrome<br>(e) Asthma |
| **7. Past Obstetric Performance** |
| Women with history of recurrent abortions and previous preterm birth. |
| **8. Pregnancy Complications** |
| (a) Multiple pregnancy: due to overdistension of uterus<br>(b) Placenta previa or abruptio placentae<br>(c) Hydramnios (due to overdistension) |
| **9. Trauma may Cause Premature Labour** |
| **10. Genetic** |
| Many preterm deliveries are familial. |
| **11. Iatrogenic: Elective Preterm Labour for Various Maternal and Foetal Conditions** |
| **12. Foetal Factors** |
| (a) Congenital malformations<br>(b) Intrauterine growth restriction<br>(c) Intrauterine death<br>(d) Foetal distress |
| **13. Idiopathic (Majority)** |
| Premature activation of the same systems involved in initiating labour at term. |

## Biomarkers

### Cervico-vaginal fluid

1. **Foetal fibronectin:** It is a glycoprotein produced by foetal amnion in the cervico-vaginal discharge between 24–34 weeks is a predictor of preterm delivery. Levels can be normally high before 22 weeks and > 37 weeks. It is measured by enzyme-linked immunosorbent assay (ELISA), and values exceeding 50 ng/mL are considered positive.

   Therefore, it is not done in patients with leaking per vaginum. However ACOG 2016 does not recommend screening with fibrorectin.
2. **Placental α Macroglobulin-1 (PAMG-1)**
   - called as "Partosure"
   - it has a sensitivity of 80% and specificity of 95% in prediction of preterm birth in 7 days.
3. **Insulin like growth factor binding proein (IGFBP-1)**
   - called as "Actin Partus Test"

- Values >10 µg/L predict preterm birth in 7 days with a sensitivity of 73% and specificity of 61%

4. β-HCG
- a cut off valve of > 32 MIU/ml has a positive predictive value of 89% and negative predictive value of 95% for preterm birth within 1 week.

**Amniotic fluid**

1. Alkaline phosphatase: greater than 90th percentile is considered a predictor.
2. IL-1, IL-6, TNF-α increase

**Serum markers**

1. Corticotrophin releasing hormone (CRH)
2. C-reactive protein (CRP)
3. Alpha-feto protein (AFP)
4. Salivary estriol

## Infection

**Bacterial vaginosis**

In this condition, the normal hydrogen peroxide producing lactobacillus predominant vaginal flora is replaced with anaerobes - Gardanarella vaginalis, myocoplasma hominis, mobiluncus species.

Bacterial vaginosis has been associated with spontaneous preterm labour preterm rupture of membranes and chorioamniotis.

**Genital tract infections**

Currently, screening and treatment to prevent preterm birth in women with chalamydia trachomatis or Trichomonas vaginalis are not recommended. Treatment with erythromycin, azithromycin or metronidazole does not reduce preterm delivery rate.

**Peri-odontal disease**

Oral bacteria, especially fusobacterium nucleatum and capnocytophaga species have been associated with upper genital tract infections and preterm birth. However, antibiotic or metronidazole therapy do not reduce preterm birth.

Unfortunately, till date, screening tests have not been shown to prevent pre-term birth. ACOG 2016 does not recommend use of home uterine activity monitoring, bacterial vaginosis screening, USG for cervial length or fibronectin test in general population for screening for preterm labour.

## Prevention of Preterm Birth

All factors which could lead to prematurity are not clear, but an attempt at prevention can be helpful.

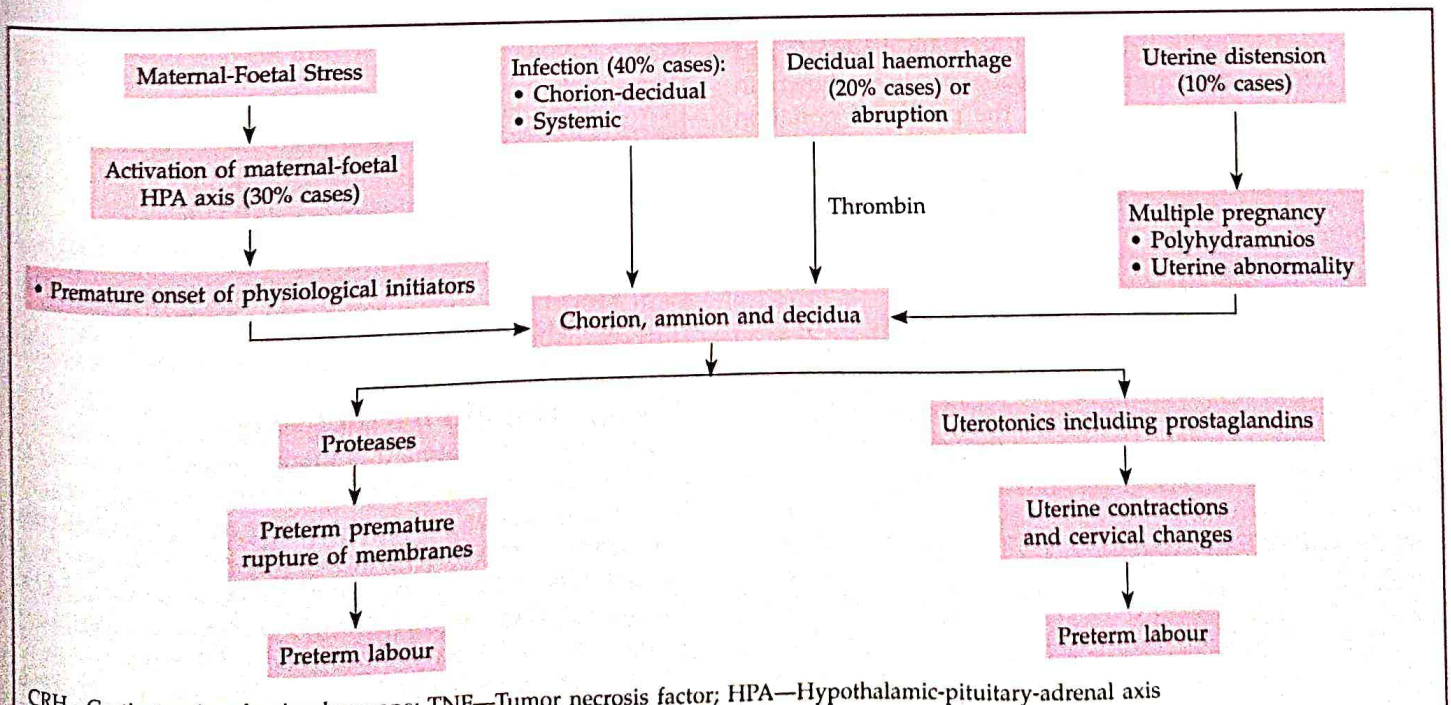
**Primary Prevention**

Primary prevention of preterm birth is the elimination or reduction of risk factors in all women. It includes:

1. Prevent pregnancy in teenagers.
2. Prevent smoking and illicit drug abuse
3. Prevent reproductive tract infections/sexually transmitted infections. Treat asymptomatic bacteriuria and UTIs as their treatment decreases preterm labour.
4. Access of family planning methods to prevent unwanted and frequent pregnancies
5. Preconceptional counselling
6. Improve nutrition and general health of women
7. Decrease factors causing stress and give adequate rest.

**Secondary Prevention**

Secondary prevention includes the identification of




CRH—Corticotropin releasing hormone; TNF—Tumor necrosis factor; HPA—Hypothalamic-pituitary-adrenal axis

**Fig. 34.1:** Pathogenesis of preterm labour

pregnant women who are at risk of preterm delivery and their close surveillance. It includes:

1. **Patient education** to teach them to detect early symptoms of preterm labour (rhythmic backache, sense of pelvic pressure, heavier vaginal discharge, vaginal spotting and abdominal cramps) and also instruct them to reduce physical and sexual activities.
2. **Medical therapy** includes tocolytic drugs, antibiotics, cortisone and use of metronidazole between 14–20 weeks of gestation for bacterial vaginosis.
3. **Role of progestogens.** American College of Obstetricians and Gynecologists (2017) has recommended their use only in women with a documented history of a previous spontaneous preterm delivery and asymptomatic women with a short cervical length (< 2.5 cm). Progestogens maintain uterine quiescence by suppressing cytokine activity.
4. **Cerclage operation** (if cervical incompetence is present). Its empirical use in absence of cervical incompetence does not prevent preterm birth.

## Diagnosis of preterm labour

Diagnosis of preterm labour is made from symptoms, signs and examination.

**Symptoms and Signs of Preterm Labour.** They are shown in Table 34.2.

It is difficult to differentiate between true and false labour prior to demonstrable cervical effacement and dilatation. The following criteria may be used to document preterm labour.

1. Contractions occurring at a frequency of 4 in 20 minutes or 8 in 60 minutes with changes in cervix.
2. Cervical dilatation greater than 1 cm
3. Cervical effacement of 80% or more.

## Management

Management includes prompt and accurate diagnosis and proper referral or appropriate treatment. It is shown in Flow Chart 34.1. Confirmation of the period of gestation is important. The goal is to diagnose the condition in the reversible state and eliminate the risk factors, if possible.

Once the diagnosis of preterm labour is established, the patient is admitted to hospital, advised bed rest, activities are curtailed, adequate hydration is maintained and the following investigations are done to try and find the cause of preterm labour and to monitor maternal and foetal well-being.

**Table 34.2: Symptoms and Signs of Preterm Labour**

| Symptoms |
|---|
| 1. Uterine activity, e.g. painful or painless uterine contractions or frequent tightening<br>2. Lower abdominal pain or menstrual-like cramping<br>3. Pain in low back<br>4. Passage of blood stained vaginal discharge (show)<br>5. Sensation of pelvic or vaginal pressure<br>6. Increased watery vaginal discharge |
| **Signs** |
| 1. Palpable uterine contractions<br>2. Engagement of the presenting part<br>3. Cervical effacement<br>4. Cervical dilatation<br>5. Show (may be bloodstained)<br>6. Bulging membranes<br>7. Rupture of membranes |

## Physical Examination and Investigations

A thorough physical examination as shown in Table 34.3 is required as the risk factors and causes of preterm labour are so varied.

**Table 34.3: Routine Physical Examination in Preterm Labour**

1. Pulse, temperature, blood pressure measurement and dipstick urine analysis
2. Abdominal examination for foetal presentation, foetal heart and uterine activity.
3. Vaginal speculum examination to assess cervix, membrane status and for taking swabs.
4. Digital assessment of the cervix may be useful but is not always necessary.

The routine investigations performed are shown in Table 34.4.

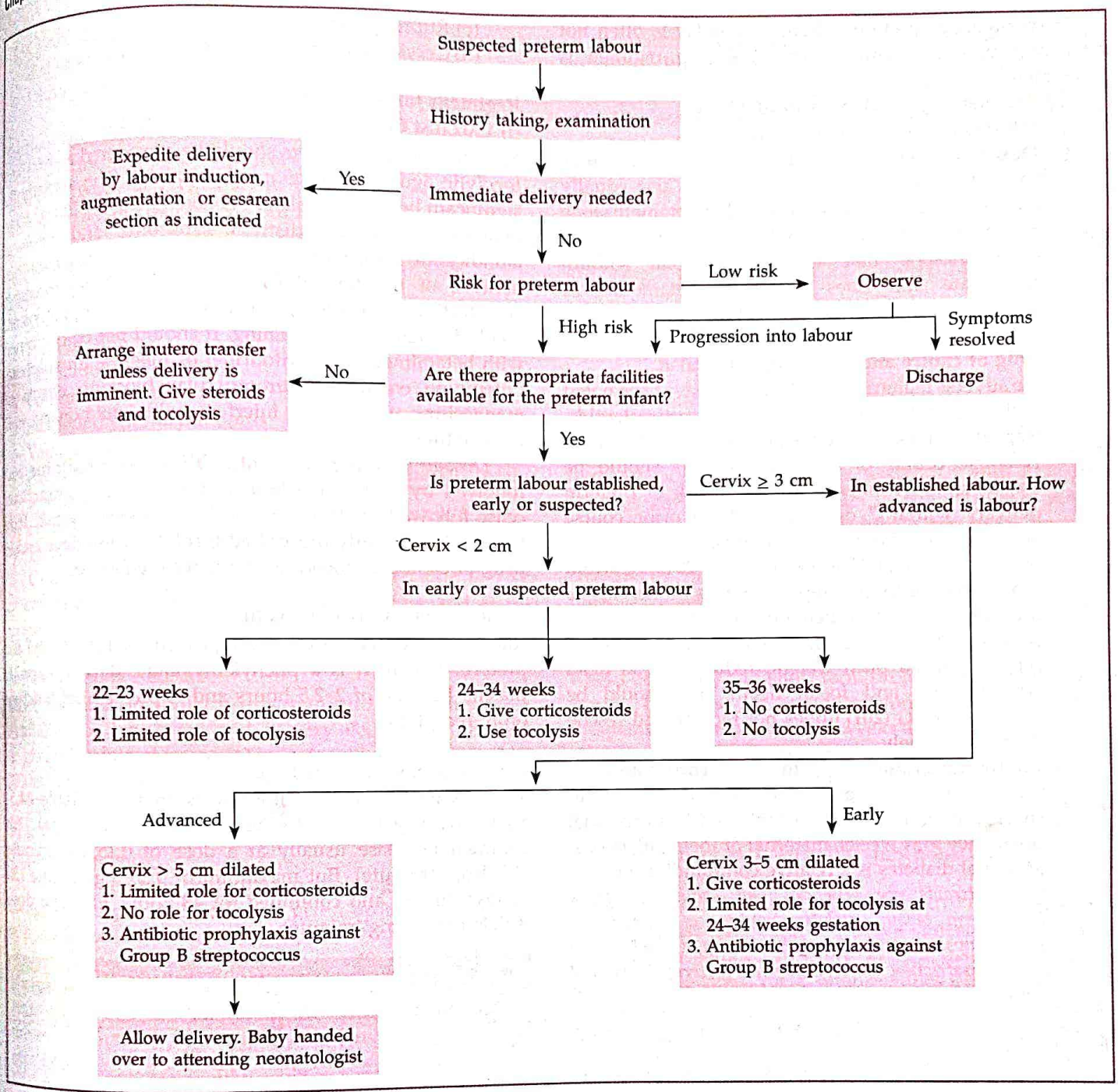
**Table 34.4: Routine Investigations for Preterm Labour**

1. Urine for glucose, proteins, ketones, microscopy and culture.
2. Swabs: One or more of the following should be taken.
   (a) High vaginal swab for gram staining and culture, pH and fern test
   (b) Endocervical swab for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* culture.
   (c) Urethral swab in indicated cases.
3. Cardiotocography (CTG) for uterine contractions and foetal heart.
4. Ultrasound for foetal maturity, foetal anomalies, presentation, liquor assessment, estimated foetal weight. Transvaginal ultrasound assessment for cervical length and funnelling at internal os. On TVS; shape of cervix is T shaped normally, Y shaped in suspicion of PTL and U shaped in preterm labour and cervical insufficiency.
5. Swab from cervico-vaginal area for foetal fibronectin, if available.

## Management of Preterm Labour

The objective of treatment is to give steroids to mother for foetal pulmonary maturity, short term tocolytic therapy to suppress labour, in utero transfer to a center equipped with neonatal care facilities.

1. **Bed rest.** Although role of bed rest is doubtful and can cause thromboembolic complications and bone loss, it is often recommended in left lateral position.
2. **In utero transfer.** If local facilities are inadequate to treat the preterm infant, an inutero transfer is better than exutero transfer as the uterus is the best transport incubator. Exutero transfers may be unavoidable if




**Flow Chart 34.1:** Management of preterm labour

delivery is imminent or if the woman is too ill to be transferred.

3. **Sedation.** Especially useful in threatened preterm labour. Morphine or pethidine with promethazine have been used.
4. **Hydration.** Adequate hydration is advised.
5. **Corticosteroid therapy.** Corticosteroids administered *antenatally to mothers are of proven efficacy in reducing neonatal mortality, respiratory distress syndrome, cerebral and intraventricular hemorrhages by about 40 to 60%* **(level IA evidence).** They stimulate type II pneumocytes in foetal lungs to secrete surfactant. The positive effect of antenatal corticosteroids is apparent soon after administration—maximal 24 to 48 hours later and for up to 1 week but adequate up to 18 days. It should be administered to women at imminent risk of preterm delivery.

**There is general consensus on administering antenatal corticosteroids between 24 and 34 weeks of gestation.** ACOG (2017) recommends single course of steroids between 24 and 34 weeks of gestation with risk of delivery within 7 days including those with ruptured membranes and multiple gestation. A single course of steroids is recommended for pregnant women between 34–36 + 6 weeks at risk of preterm delivery within 7 days who have not received antenatal steroids.

In ideal circumstances, delivery should not occur within 24 to 48 hours following the administration of corticosteroids.

In spontaneous preterm labour, this delay is often not achievable without tocolysis. The following regimen is recommended.

(*i*) **Betamethasone: Two doses of 12 mg intramuscularly 24 hours apart** or

(*ii*) **Dexamethasone** in same dosage or **6 mg 12 hourly for 4 doses** can also be given as it is also equally effective. There is some concern that betamethasone available in India has only one component of betamethasone that is betamethasone sodium phosphate and does not have betamethasone sodium acetate which may not be very effective. Hence, **currently dexamethasone is becoming the drug of choice and it is economical also.**

No adverse maternal or foetal effects have been noted with regard to a single course of corticosteroids.

**Repeat courses of corticosteroids (rescue therapy).** A single course of antenatal steroids should be considered to women who are at risk of preterm delivery within 7 days and whose prior course of antenatal steroids was administered > 14 days before or prior to 24 weeks (ACOG, 2017). Current practice is not to give repeat courses of steroids as they do not serve much purpose and may have adverse effects, e.g. maternal sepsis, neonatal sepsis, reduced birth weight, reduced foetal head circumference and foetal death and should be avoided. ACOG (2017) does not recommend rescue therapy routinely.

**Contraindications to corticosteroids.** Corticosteroids are contraindicated in chorioamnionitis, maternal tuberculosis, porphyria, pregnancy > 34 weeks, maternal or foetal infections. Maternal diabetes is a relative contraindication.

*Corticosteroids are beneficial and should be given in preterm labour, prelabor rupture of membranes, pre-eclampsia, antepartum haemorrhage and foetal growth restriction where iatrogenic preterm delivery is anticipated.*

## 6. TOCOLYSIS

(a) Gestation < 34 weeks to buy time for steroid cover and action
(b) No foetal or maternal compromise
(c) Inutero transfer

*Although tocolysis has not been shown to improve perinatal outcome in metaanalysis, it prolongs pregnancy by at least 48 hours allowing administration of betamethasone and shifting the patient to a center equipped with better neonatal facilities (in-utero transfer).*

### Contraindications

A. Absolute
(a) Thyrotoxicosis
(b) Cardiac disease
(c) Chorioamnionitis
(d) Foetal compromise
(e) Antepartum haemorrhage
B. Relative
(a) Diabetes
(b) Hypertension
(c) Ruptured membranes
(d) Cervix more than 3 cm dilated

### Regimens for Tocolysis

#### (I) Calcium Channel Blockers (Nifedipine)

Calcium channel blockers should be considered as first-line tocolytic agents in developing countries. The statistically significant benefits of calcium channel blockers in reducing neonatal respiratory distress syndrome and neonatal jaundice are even more important in developing countries because of the limited capacities of neonatal intensive care facilities. Side effects include maternal hypotension, headache, nausea and flushing. It should not be combined with betamimetics (e.g. ritodrine) or magnesium sulphate. Nifedipine enhances neuromuscular blocking effects of magnesium which can interfere with pulmonary and cardiac functions.

Dosage is 20 mg oral **(not sublingual) nifedipine** stat followed by 10 mg 4–6 hourly until uterine contractions cease followed by 10 mg 8 hourly for about a week. **It is the most commonly prescribed tocolytic these days** and is drug of choice for tocolysis and is most effective.

#### (II) Ritodrine Hydrochloride

Ritodrine hydrochloride was approved by FDA (USA) for preterm labour. It is a phenylethylamine derivative with plasma half life of 2–2.5 hours and is preferentially active on $\beta_2$ receptors.

#### *Dosage Scheme of Ritodrine*

The dosage regimen of ritodrine is shown in Table 34.5. Start with 0.05 mg/minute (5 drops/minute) till contractions cease usually at a dose of 0.15 mg/minute (15 drops/minute). But maximum of 0.30 mg/minute (30 drops/minute) and continued for 48 hours. Effective dose is 0.05 to 0.15 mg/minute.

**Table 34.5: Ritodrine Regimen (100 mg in 500 mL 5% dextrose)**

| Drops/minute | mg/minute |
|---|---|
| 5 | 0.05 |
| 10 | 0.10 |
| 15 | 0.15 (usual dose) |
| 20 | 0.20 |
| 25 | 0.25 |
| 30 | 0.30 (maximum dose) |

#### *Cautions*

Fluid balance chart
Fluid should be restricted to 2.5 L/24 hours.

#### *Observations*

1. Monitoring of foetal heart and uterine contractions every 15–30 minutes.
2. Record BP and pulse every 15 minutes and temperature every 2 hourly.
3. Auscultate lung bases every 12 hours (any crepts due to pulmonary edema).

4. Check blood sugar every 4 hours (every hour in diabetes), urea and electrolytes every 24 hours.
5. Warn patient of shaking and palpitations.

### *Further Treatment*

Oral ritodrine tablet (10 mg) every 4–6 hours can be given until 37th week of pregnancy or at least up to one week after stoppage of contractions but its role is doubtful.

### *Indications for Cessation (Stopping) of Tocolysis (Complications of Ritodrine)*

1. Maternal tachycardia > 140 per minute
2. Hypotension
3. Palpitations/Chest pain
4. Pulmonary edema
5. Suspected chorioamnionitis
6. Vaginal bleeding
7. Continued labour (continuing contractions or cervix > 5 cm dilated)
8. Hypokalemia
9. Fatal tachycardia > 180 per minute

Currently ritodrine is not much preferred due to risk of complications especially pulmonary edema and is no longer available in USA.

### (iii) Isoxsuprine Regimen

Most hospitals in India, use isoxsuprine which has been available for long time and is very economical. Patient is given loading dose of isoxsuprine-40mg infusion (I.V) (500ml of 5% dextrose at a concentration of 0.8mg/Ml).

Drop rate is set at 8 drops/minute (0.04mg/minute), which is increased by 8 drops every 15-minutes until uterine quiescence

After 15 minutes of infusion stoppage, isoxsuprine-10mg/4 hourly (I.M injection) is given up to first 24-hours of hospitalization

For next 24 hours, isoxsuprine-40mg retard oral capsule are given (BD) as a maintenance therapy and the same oral dose is continued till the time of delivery. Side effects, precautions and contraindications are the same as with ritodrine.

### (iv) Terbutaline

Terbutaline has replaced ritodrine because of its ease of administration. It is given as IV bolus of 250 µg followed by 10–50 µg/minute until the labour stops. Then administer subcutaneously 0.25–0.5 mg every 2–4 hours for 12 hours. A maintenance dose of 2.5–5 mg orally may be given 4–6 times a day. Side effects, precautions and contraindications are like ritodrine.

### (v) Magnesium sulphate

Magnesium sulphate is a safe drug with limited tocolytic efficacy. It can be used in diabetic mothers. A loading dose of 4–6 g over 20–30 minutes is followed by infusion of 1–2 g/hour. It competes for calcium entry into the muscle cells. *There is some evidence that if used between 24 and 32 weeks, for at least 12 hours, $MgSO_4$ has tocolytic and neonatal neuroprotective role by decreasing incidence of cerebral palsy in the newborn* (Beneficial effects of antenatal magnesium sulphate—BEAM study, 2008).

Its use has been approved by Royal College of Obstetricians and Gynecologists London for neuroprotection and to prevent cerebral palsy in newborns if used between 24 to 32 weeks of gestation with preterm labour. It is a membrane stabilizer and reduces motor end plate sensitivity to acetylcholine.

Infact many hospitals in India (including AIIMS, Delhi) are using it for neuroprotection between 24–32 weeks.

#### *Maternal Side effects*

Flushing, perspiration, headache and muscle weakness

#### *Neonatal Side effects*

Lethargy, hypotonia; rarely respiratory depression

Due to concerns for foetal and neonatal bone demineralization and fractures associated with long term in utero exposure to magnesium sulphate, US FDA advises against use of magnesium sulphate for more than 5–7 days to stop preterm labour in pregnant women and has changed its category from A to category D. ACOG (2013), however, continues to support use of magnesium sulphate for short term use (up to 48 hours) in women with severe pre-eclampsia and eclampsia, foetal neuroprotection (< 32 weeks gestation) and short term prolongation of pregnancy (up to 48 hours) for administration of corticosteriods in pregnant women between 28 weeks and 34 weeks gestation who are at risk of preterm delivery within 7 days.

Combination of nifedipine and magnesium sulphate is dangerous and not recommended as nifedipine enhances neuromuscular blocking effect of magnesium interfering with pulmonary and cardiac dysfunction.

### (vi) Indomethacin (Prostaglandin Synthetase Inhibitors)

Indomethacin and ibuprofen are effective and well-tolerated tocolytics especially at less than 32 weeks of gestation. However, foetal abnormalities like patent ductus arteriosus, oligohydramnios, necrotizing enterocolitis, neonatal pulmonary hypertension and neonatal death have been reported in some studies but other studies did not find adverse effects. Dose is 50 mg loading dose orally or per rectally followed by 25–50 mg 6 hourly.

### (vii) Atosiban

It is a nonapeptide (9 amino acids) oxytocin analogue which acts as a competitive antagonist of oxytocin induced contractions.

**Mechanism of action:** it competes with oxytocin and thus prevents increase in intracellular calcium levels and oxytocis induced release of PGF2α.

#### *Dosage*

6.75 mg intravenous bolus over 1 minute followed by continous infusion of 300 µg/minute for 3 hours followed by 100 µg/minute for 45 hours. Maximum dose 330 mg.

It is drug of choice as its side effects are much less as compared with ritodrine. Although it is popular in Europe, it failed to improve neonatal outcome and was found

associated with significant neonatal morbidity. Most studies including meta-analysis did not find its superiority over calcium channel blockers or B-mimetics. It is expensive and is now available in India (also see Chapter 14). It is the safest and is especially useful in heart disease.

(viii) Nitric Oxide Donors

The nitric oxide donor glyceryltrinitrate is also under research to prevent preterm labour. It causes headache.

7. **Prophylactic Antibiotics.** The ORACLE I and II trial has confirmed that antibiotics are not of any benefit in reducing neonatal mortality or serious neonatal morbidity in preterm labour with intact membranes. Cochrane meta-analysis (2013) has also confirmed that animicrobial therapy does not prevent preterm labour in women with intact membrane.

   **Group B Streptococcus.:** Benzylpenicillin or ampicillin should be given in established preterm labour as prophylaxis against early onset neonatal sepsis due to Group B Streptococcus in those with a known carrier status. In cases of penicillin allergy, clindamycin can be substituted. Ideally, it should be administered 4 hours prior to delivery.
8. **Emergency or Rescue Cervical Cerclage.** Rescue cervical cerclage in women with cervical incompetence before 27 weeks has been observed to prevent preterm labour. It is recommended after counselling.
9. **Role of progestogens (SMFM 2017)** (Society of Maternal and Foetal Medicine of USA, 2017)
   (i) Women with history of spontaneous preterm birth. Inj. Hydeoxyprogesterone Caproaite 250 mg intramuscularly weekly from 16 to 36+6 weeks of pregnancy has been recommended to prevent preterm birth.
   (ii) Women with short cervix: Natural progesterone 200 mg vaginally starting from time of diagnosis till 36th week has been recommended.
   However, OPTIMUM study in which 200 mg vaginal progesterone was given between 24–34 weeks in high risk women with prior history of spontaneous preterm birth (< 34 weeks) or with a cervical length of < 2.5 cm or positive fibronectin test, did not show any benefit in reduction in preterm birth or improvement in foetal outcome.

## Management of labour

If tocolysis has failed to stop contractions, labour should be allowed. Presentation should be confirmed by gentle vaginal examination. While in western countries with better neonatal facilities, cesarean delivery is preferred for breech presentation between 26–34 weeks, it should be done with caution in India where neonatal mortality rate is very high. The aim of management of preterm labour is to prevent asphyxia and birth trauma.

### First Stage

The patient lies in bed to avoid early rupture of membranes as intact membranes may reduce trauma to the baby. In case of anticipated traumatic vaginal delivery, cesarean delivery can be considered. The parents should be informed of the likely outcome by obstetrician and pediatrician. The survival rate is poor and handicap rates are very high in preterm babies. There has been dramatic improvement in neonatal outcome of preterm babies in developed countries. Accurate gestation and foetal weight are important prognostic factors. Strong sedatives and oxytocics should be avoided. Labour should be watched by intensive clinical monitoring, if continuous electronic monitoring is not available. Repeated digital examinations are avoided.



**Fig. 34.2:** A preterm newborn baby at 26 weeks period of gestation weighing 800 grams

### Second Stage

An experienced obstetrician should preferably conduct the delivery in presence of a pediatrician. Epidural can be given, if available. Episiotomy is recommended, especially in primigravidas, to minimize head compression.

Forceps are applied only if second stage is delayed and not to act as a protective cage for the soft preterm head during delivery as once thought. Delayed cord clamping (after 1 minute) is recommended if baby is crying. The newborn is immediately transferred to intensive neonatal care unit, with ventilator facilities under the care of a neonatologist (Fig. 34.2).

## PRELABOR (PREMATURE) RUPTURE OF MEMBRANES (PROM)

Prelabor rupture of membranes is defined as spontaneous rupture of the membranes before the onset of regular uterine contractions. When this condition occurs before 37 weeks, it is referred to as preterm prelabor rupture of the membranes (PPROM) (see below). When occurs at or after 37 weeks, it is called term prelabor rupture of the membranes (term, PROM).

## PRETERM PRELABOR OR PREMATURE RUPTURE OF MEMBRANES (PPROM)

**Definition. Preterm prelabor rupture of membranes is defined as spontaneous rupture of membranes before**

**37 weeks of gestation (preterm) in the absence of uterine contractions or before onset of labour (prelabor) (ACOG 2016)**

Incidence. The incidence of PPROM varies in different areas due to different population risk factors and occurs in about 3% of all pregnancies. PPROM precedes about 30–40% cases of spontaneous preterm labour. About 30–40% of PPROM deliver within 48 hours and about 56–63% deliver within 7 days.

In comparison, the term prelabor rupture of membranes (PROM) occurs in 10% term pregnancies and 86% such cases start labour within 24 hours.

## Etiology

The etiology of PPROM is multifactorial but infection plays a significant role. Risk factors for PPROM are very similar to those of preterm labour and are shown in Table 34.6.

**Table 34.6: Causes of PPROM**

1. Lower socio-economic status
2. Past history of PPROM
3. Cervical incompetence
4. Genital tract infection or colonization with
   (a) *Bacterial vaginosis* (b) *Chlamydia trachomatis*
   (c) *Neisseria gonorrhoeae* (d) *Trichomonas vaginalis*
   (e) *Group B Streptococcus* (f) *Gardnerella vaginalis*
5. Oxidative stress induced DNA damage
6. Urinary tract infection
7. Increased friability and decreased tensile strength of membranes (Premature cellular senescence)
8. Low maternal body mass index (BMI) < 19.8 kg/m$^2$
9. Polyhydramnios
10. Short cervix
11. Multiple pregnancy
12. Intrauterine death
13. Foetal abnormalities
14. Iatrogenic invasive procedures, e.g. amniocentesis, foetal blood sampling
15. Cigarette smoking

## Effects of PPROM

1. Preterm labour and birth
2. Pulmonary hypoplasia due to severe oligohydramnios
3. Chorioamnionitis (see below)
4. Skeletal and joint deformities of the fetus due to compression

## Presentation of PPROM

### Symptoms

The classic symptom of PPROM is leakage of fluid from vagina. This fluid is usually colorless. At later gestations, it may contain vernix. Amniotic fluid leakage may be exacerbated by erect posture and straining of abdominal muscles. Amniotic fluid loss is persistent as amniotic fluid is periodically replenished by foetal urination.

Uterine contractions commonly follow PPROM.

### Signs

PPROM is diagnosed when vaginal leak of amniotic fluid is demonstrated.

### Examination

Examination in a case of PPROM is depicted in Table 34.7.

**Table 34.7: Examination in PPROM**

1. Pulse, blood pressure, temperature measurement and urine analysis
2. Abdominal palpation for foetal size, presentation, liquor volume, uterine tenderness, multiple gestation
3. Vaginal speculum examination aseptically with Cusco's speculum
   (a) Demonstrate gushing of amniotic fluid in the upper vagina. Ask patient to cough or apply fundal pressure while observing cervix for fluid loss. The pH of amniotic fluid will be alkaline in contrast to acidic pH of urine. Vernix particles or meconium, if present, are diagnostic of amniotic fluid. If on speculum examination, no amniotic fluid is visible tests like insulin like growth factor binding protein (IGFBP-1) or placental alpha macroglobulin-I (PAMG-I) test on vaginal fluid can be done (Grade B recommendation, RCOG 2019)
   (b) Speculum examination also gives information about dilatation and effacement of cervix.
   (c) For taking endocervical and high vaginal swab for culture sensitivity.
   (d) Exclude cord prolapse
   (e) Collect amniotic fluid for further studies for foetal maturity (see chapters 4 and 24 for details)
4. Assess uterine activity and foetal viability—a CTG is preferred. Stethoscope can be used to hear foetal heart sound.
5. Vaginal examination should be avoided to avoid infection but can be done in advanced labour.

### Investigations

The various investigations performed in PPROM are shown in Table 34.8.

### Diagnosis

1. Non-invasive tests: These tests are done on vaginal fluid
   (i) pH test:
      (a) Litmus paper turns blue when in contact with alkaline amniotic fluid.
      (b) Bromothymol blue turns green
      (c) **Nitrazine test:** Alkaline pH of amniotic fluid will turn nitrazine paper blue in case of PPROM. False positive result can occur in presence of blood and infection.
   (ii) **Fern test:** If fern is seen on slide test of fluid, PPROM is present.
   (iii) Microscopic examination of vaginal fluid to look for
      (a) Foetal fat globules (Sudan III staining)
      (b) Lanugo hair in vaginal smear

**Table 34.8: Investigations for PPROM**

1. Full blood count for leucocytosis and for C. reactive protein (CRP)
2. Urine for microscopy and culture
3. (a) High vaginal swab for Gram staining and culture
   (b) Endocervical swab for Neisseria gonorrhoeae and Chlamydia trachomatis
   (c) Collected vaginal fluid for microbiological culture and foetal lung maturity studies
4. Amniocentesis to provide uncontaminated amniotic fluid for microbiological culture (this is the best indicator of chorioamnionitis) and foetal lung maturity studies, e.g. lecithin/sphingomyelin ratio (not routinely done)
5. Ultrasound examination for foetal assessment, amniotic fluid measurement and biophysical scoring.

(iv) Cytodiagnosis: It can be performed on vaginal fluid by
   (a) Papanicolaou test
   (b) Nile blue sulfate test
   (c) Masson's trichome
   (d) Acridine orange
   (e) Pinacyanol

(v) Amni Sure test: it is a rapid slide test using chromatographic methods to detect traces of PAMG-1 in cervico-vaginal fluid. Threshold of the test for the diagnosis is > 5.0 ng/ml. The test has a sensitivity of 98.7% and specificity of 90%.

(vi) Actim Partus test: immunochromatographic dipstick method with a minimum threshold value of > 25 ug/L . The test has a sensitivity of 95-100% and specificity of 93-98%.

(vii) ROM Plus test: it is a combination of monoclonal/polyclonal ab test detecting placental protein 12 and AFP. It has a sensitivity of 99% and specificity of 91%.

(viii) Amnio-Sense test: It uses a 12×4 cm absorbent pad wherein the central strip changes colour on contact with a fluid of ph > 5.2. It has a sensitivity of 100% and specificity of 65-90%.

2. Invasive tests:
   (i) Transabdominal dye test by intra-amniotic infection of indigocarmine: It is an obsolete test now. It can demonstrate PPROM by colour change.

## Differential Diagnosis

1. Maternal urinary incontinence is common and can be confused with complaint of vaginal fluid loss.
2. Vaginal discharge (candidal or other type of vaginitis) can be mistaken for amniotic fluid.
3. Cervical mucus discharge

## Management

The management of PPROM is shown in Flow Chart 34.2. The woman should be admitted for a period of observation. If PPROM is confirmed, expectant management should be done up to 34 weeks.

### Expectant Management

Uncomplicated PPROM is usually managed expectantly at gestations < 34 weeks.

1. **Diagnosis uncertain**
   (a) Admit the patient
   (b) Perform regular perineal pad checks
   (c) Perform ultrasound for foetal well-being and liquor volume (oligohydramnios goes in favor of ruptured membranes).
   (d) Observe for signs of chorioamnionitis (see below)
2. **Diagnosis certain**
   (If < 34 weeks gestation) Routine conservative treatment is preferred.
   (a) Contact neonatal intensive care unit (NICU)
   (b) Admit for bed rest

The main risk of expectant management is of ascending infection causing chorioamnionitis and foetal infection. In the presence of proven infection, urgent delivery is indicated as antibiotic treatment is usually ineffective while the pregnancy is ongoing.

Routine monitoring in expectantly managed PPROM should be done as shown in Fig. 34.3.

Delivery is expedited when any of the following conditions develop.

1. Chorioamnionitis
2. Onset of spontaneous labour
3. Foetal compromise
4. Foetal maturity is achieved.
5. Maternal disease that may be affected by consequences of PPROM.

There is evidence that with good neonatal care facilities, for PPROM > 34 weeks, a policy of expediting delivery

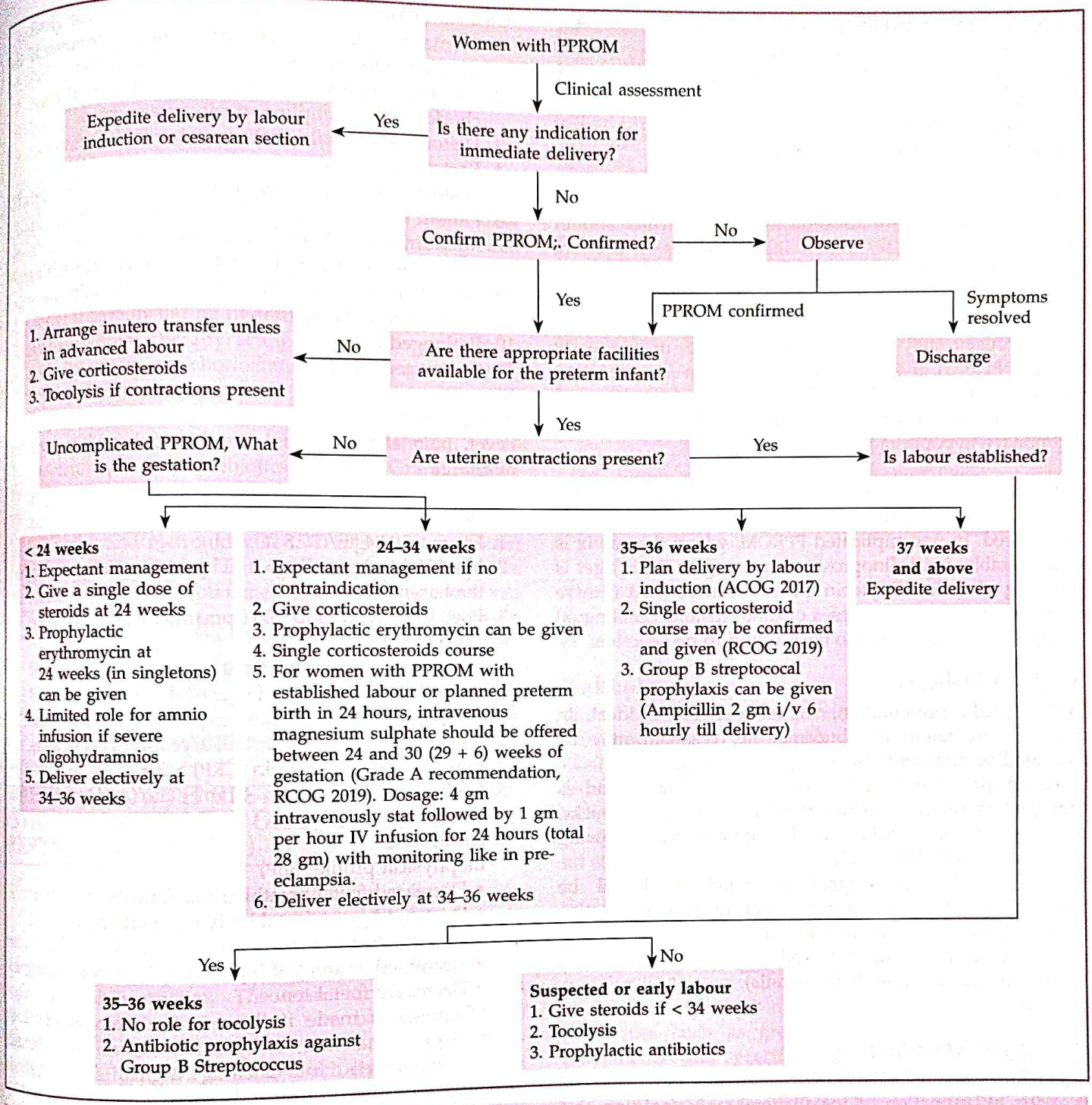
**Maternal markers of infection**

- Temperature measurements 4 times a day
- Maternal pulse 4 times a day
- Twice daily abdominal palpation for increased tone/tenderness (to rule out chorioamnionitis)
- Daily note of vaginal loss (for detection of purulent discharge)
- Thrice weekly total leucocyte count (TLC)
- Thrice weekly C reactive protein (CRP)
- Weekly high vaginal swab, urine routine, microscopic examination and culture sensitivity

**Foetal well-being tests**

- Foetal movement chart
- Non-stress test
- 2–3 weekly growth scan
- Weekly amniotic fluid assessment depending on gestation
- Foetal biophysical profile (including CTG) is of limited value in detecting foetal infection

**Fig. 34.3:** Monitoring in PPROM




**Flow Chart 34.2:** Management of preterm premature rupture of membranes

results in less neonatal infection, maternal infection and shorter hospital stay without adversely affecting neonatal or maternal outcome. ACOG (2016) now recommends delivery after 34 weeks with PPROM. However, delivery can be delayed if no contraindication after discussing the benefits and risks with the patient. However, after 37 weeks expectant management should not be offered and delivery should be expediated.

### Antenatal Corticosteroids

In uncomplicated PPROM, antenatal corticosteroids should be administered.

### Prophylactic Antibiotics

The ORACLE I trial suggests that prophylactic oral erythromycin (not coamoxyclav) 250 mg four times a day should be administered for 10 days or until delivery (whichever is sooner) to singleton pregnancies with PPROM to prevent chorioamnionitis and to reduce neonatal mortality and sepsis RCOG (2019) also recommends oral erythromycin for 10 days.

Compared with antenatal corticosteroids, benefits from prophylactic antibiotics in PPROM are less pronounced.

### Group B Streptococcus

With PPROM, the risk of neonatal Group B streptococcal sepsis is increased—both preterm delivery and PPROM are independent risk factors for Group B streptococcal sepsis. Prophylatic benzylpenicillin or ampicillin is indicated at first presentation and again at intrapartum.

### Tocolysis

PPROM is not a contraindication to tocolysis which should be used for the same limited indications as in preterm labour with intact membranes.

### Amniotic Fluid Infusion

Amnioinfusion may be useful in some cases of very early onset of PPROM. If PPROM occurs at < 24 weeks gestation and results in severe oligohydramnios, infusion of normal saline into the amniotic cavity has been shown to reduce pulmonary hypoplasia.

## Induction of Labour

If vaginal delivery is to be expedited in PPROM, labour may be induced. In uncomplicated PPROM, where the cervix is unfavourable, local dinoprostone (prostaglandin $E_2$) gel is the drug of choice. Oxytocin infusion can be used if there is no response after 1–2 doses of dinoprostone. Sublingual misoprostol ($PGE_1$) has also been found to be effective.

## Mode of Delivery

With cephalic presentation, vaginal delivery is ideal. In breech presentation or abnormal lie, cesarean may be indicated as discussed above in preterm labour.

Cord prolapse is more common in PPROM and is managed by cesarean section at > 26 weeks. If < 26 weeks, an expectant approach may be appropriate following consultation with the couple.

The number of vaginal examinations should be kept to the minimum during labour to reduce infective morbidity. Newborn should be managed by the attending neonatologist and shifted to NICU (Neonatal Intensive Care Unit) (see Chapter 28 for details).

# CHORIOAMNIONITIS

It is the inflammation of foetal membranes (amnion and chorion) and is usually a manifestation of intrauterine infection. In occult chorioamnionitis, there are no obvious symptoms and signs but there is inflammation of chorion and amnion. It can cause pre labour rupture of membranes and preterm labour.

In overt chorioamnionitis, the symptoms and signs are obvious.

In **chorioamnionitis**, the mononuclear and polymorphonuclear leucocytes infiltrate the chorion and the cells are maternal in origin. If the cells are found in amnionic fluid it is called **amnionitis** and when they are found in umbilical cord, it is called **funisitis**. The cells in amnionitis and funisitis are foetal in origin.

## Causes (Etiology)

1. Premature (pre labour) rupture of membranes is the most common cause.
2. Prenatal diagnostic procedures (eg amniocentesis, chorion villus sampling, cordocentesis, etc.)
3. History of amnioinfusion
4. Bacterial vaginosis
5. History of cerclage operation
6. Fibroid uterus
7. Malnutrition and severe anaemia
8. Cases handled by traditional birth attendants (dais)
9. Repeated vaginal examination especially without aseptic precautions
10. Prolonged labour
11. Obstructed labour

## Symptoms

Fever, pain abdomen, malaise and foul-smelling vaginal discharge

## Signs

1. Fever ≥ 100.4 °F/ 37.8 °C
2. Maternal tachycardia (> 100 bpm or > 20 bpm above the baseline)
3. Foetal tachycardia (> 160 bpm)
4. Uterine tenderness
5. Foul-smelling amniotic fluid

## Investigations

1. Maternal leucocytosis (≥ 16000/cc and shift to left)
2. Raised C reactive protein (CRP) > 2.5
3. High vaginal swab for Gram stain and culture and sensitivity
4. Ultrasound
   Biophysical profile (BBP)
   - Decreased foetal breathing movements
   - Decreased gross foetal body movements
   - Non-reactive non-stress test
   - Decreased amount of liquor (amniotic fluid volume)
   - Decreased foetal tone

Diagnosis is made if there is fever alongwith 2 or more signs (maternal tachycardia, foetal tachycardia, uterine tenderness, foul-smelling vaginal discharge) or investigations (leucocytosis, raised CRP).

## Maternal Effects

Chorioamnionitis is a serious condition and can have the following effects.

1. Premature labour
2. Prelabor rupture of membranes
3. Septicemia and septic shock
4. Puerperal sepsis and its complications
5. Subinvolution of uterus
6. Rarely death, if left untreated.

## Foetal effects

1. Prematurity
2. Neonatal sepsis
3. Respiratory distress syndrome

4. Early onset seizures
5. Intraventricular haemorrhage
6. Periventricular leucomalacia
7. Neurological injury including cerebral palsy
8. Foetal and neonatal death
9. Skeletal and joint deformities

### Management

Chorioamnionitis needs to be individualized depending upon etiology and severity. Antibiotic therapy and expedition of delivery are the two key components of management.

1. General care including hydration
2. **Antimicrobial therapy** is the mainstay in prevention and treatment of chorioamnionitis. In women with rupture of membranes, 7 days treatment with ampicillin 500 mg 6 hourly or amoxicillin plus erythromycin should be given. For treatment, broad spectrum parental antibiotics covering Gram positive and Gram negative organisms and anerobes by ampicillin (500 mg IV 6 hourly), gentamicin (60 mg IV 8 hourly) and metronidazole (500 mg IV 8 hourly) are given. Alternatively, third generation cephalosporins (ceftriaxone or cefotaxime) and metronidazole can be given. Betamethasone therapy is avoided in overt chorioamnionitis.
3. Removal of cerclage stitch in case of McDonald suture.
4. **Expedition of delivery** by inducing labour or by cesarean delivery depending upon the case (refer to the section on PPROM) is the most important treatment.

## FOETAL GROWTH RESTRICTION (FGR) (SYN: INTRAUTERINE GROWTH RESTRICTION [IUGR])

Normal foetal growth is discussed in Chapter 5.

Previously called intrauterine growth restriction or retardation (IUGR), low birth weight infants who are small for gestational age (SGA) are now termed as suffering from foetal growth restriction. The term intrauterine growth retardation used in the past has now been discarded as retardation implies retarded mental function. **FGR is defined as failure of a fetus to reach its genetic growth potential inutero putting it at risk of perinatal mortality and morbidity.** Using the World Health Organization (WHO) cut-off, the term SGA is used when the newborn baby's weight is below the tenth precentile for its gestational age (ACOG).

### Incidence

About 3 to 10% infants are growth restricted (12–40% in twin pregnancy). Many infants with birth weight less than the 10th percentile are preterm or small due to neonatal biological factors rather than being pathologically growth retarded.

### Types

Delphi Consensus Criteria is used now for FGR and its classification and is shown in Table 34.9 and Table 34.10.

**Table 34.9: Delphi Consensus Criteria for Foetal Growth Restriction and Classification (In absence of congenital anomalies, based on international Delphi consensus)**

| Early FGR (Formerly symmetrical FGR) (Gestational age < 32 weeks in absence of congenital anomalies) | Later FGR (Formerly asymmetrical FGR) (Gestational age > 32 weeks) in absence of congenital anomalies |
|---|---|
| AC/EFW < 3rd centile or UA-AEDF<br>Or<br>1. AC/EFW < 10th centile combined with<br>2. Ut A PI > 95th centile and/or<br>3. UA PI > 95 th centile | AC/EFW < 3rd centile<br>Or at least two out of three of following<br>1. AC/EFW < 10th centile<br>2. AC/EFW crossing centiles > 2 quartiles on growth centiles<br>3. CPR < 5th centile or UA PI > 95th centile |
| FGR: Foetal Growth Restriction, AC: Abnorminal Circumference, EFW: Estimated foetal weight, UA: Umbilical artery, | |

### Etiology

The various causes of foetal growth restriction are shown in Table 34.11.

### Pathophysiology

There is reduction in the available nutrients and micronutrients to the fetus either due to their less availability to the mother (poverty or malnutrition) or their reduced placental transfer. In some cases there is faulty utilization of nutrients by the fetus. The end result is a small fetus with scanty liquor and reduced glycogen and fat stores.

There seems to be absence of invasion of trophoblast in media of spiral arteries with reduced cytotrophoblastic proliferation and impairment of placental angiogenesis in placenta of growth-restricted fetuses like in pre-eclampsia (see **Fig. 32.1**).

In the early stages of hypoxia, the fetus uses adaptive techniques such as growth reduction, decreased foetal movements and vascular redistribution to reduce its oxygen requirements, in an attempt to prevent hypoxic injury. There is stimulation of erythropoiesis with an increase in foetal hematocrit.

As the fetus becomes increasingly hypoxemic, there may be a decrease in flow to other organs and a corresponding increase in foetal blood flow to the brain, heart and adrenals. Failure of compensatory mechanisms cause abnormal foetal heart rate patterns (Fig. 34.4).

### Complications

#### Maternal

Mother is usually not affected by FGR. However, various maternal diseases like pre-eclampsia, renal or other medical diseases causing FGR may adversely affect her general condition. There is increased incidence of FGR and stillbirths in subsequent pregnancies.

**Table 34.10: Main clinical characteristics of early- and late-onset foetal growth restricton**

| Characteristic | Early-onset FGR | Late-onset FGR |
|---|---|---|
| Main clinical challenge | Management | Detection |
| Prevalence | 30% | 70% |
| Gestational age at manifestation | < 32 weeks | > 32 weeks |
| Ultrasound findings | Fetus may be very small | Fetus not necessarily very small |
| Doppler velocimetry | Spectrum of Doppler alterations that involves umbilical artery, middle cerebral artery and ductus venosus | Cerebral blood-flow redistribution - Typically sparing umbilical artery and ductus venosus |
| Biophysical profile | May be abnormal | May be abnormal |
| Hypertensive disorders of pregnancy | Frequent | Not frequent |
| Placental histopathological findings | Poor placental implantation, spiral artery abnormalities, maternal vascular malperfusion | Less specific placental finding, mainly altered diffusion |
| Perinatal mortality | High | Low. However due to poor tolerance to hypoxia, more chances of still birth and poor long term outcomes. |

**Table 34.11: Causes of FGR**

**Causes of early onset of symmetrical FGR**

1. Genetic factors; constitutional chromosomal and single gene defects (e.g. trisomy 21)
2. Congenital anomalies
3. Inborn errors of metabolism
4. Foetal infections (TORCH and CMV virus)
5. Exposure to teratogens
6. Radiation exposure

**Causes of late onset of asymmetrical FGR**

1. Hypertensive disorders of pregnancy
2. Anaemia
3. Hemoglobinopathies
4. Vasculopathies
5. Chronic renal disease
6. Cyanotic heart disease
7. Chronic partial separation of placenta
8. Extensive placental infarctions
9. Placenta previa
10. High altitude
11. Antiphospholipid antibody syndrome
12. Extrauterine pregnancy

**Causes of Combined Type of FGR (Both early symmetrical and late asymmetrical)**

1. Cigarette smoking
2. Drug abuse
3. Abruptio placentae
4. Placental infarcts
5. Circumvallate placenta
6. Velamentous insertion of umbilical cord
7. Multiple pregnancy

### Foetal Complications

1. Risk of stillbirth due to chronic placental insufficiency
2. Non-reassuring foetal status (foetal distress) due to intrapartum hypoxia, acidosis and meconium aspiration.

### 1. Early neonatal complications

The neonates may have congenital malformations or chromosomal abnormalities. FGR neonates have very less subcutaneous fat and reduced muscle mass and are prone to following complications.

1. Metabolic complications
   - (i) Hypothermia: FGR fetuses are particularly susceptible to hypothermia due to reduced subcutaneous fat and small body mass.
   - (ii) Hypoglycemia due to reduced glycogen content.
   - (iii) Hypocalcemia from relative hypoparathyroidism.
   - (iv) Polycythemia from enhanced erythropoiesis due to chronic intrauterine hypoxia.
   - (v) Hyperviscosity syndrome from polycythemia
2. Complications due to asphyxia
   - (i) Hypoxic ischemic encephalopathy
   - (ii) Persistent foetal circulation
   - (iii) Meconium aspiration syndrome (due to oligohydramnios).
   - (iv) Necrotizing enterocolitis
   - (v) Pulmonary haemorrhage
   - (vi) Electrolyte imbalance
   - (vii) Metabolic acidosis

### II. Late Neonatal Complications

1. Symmetrical growth restricted babies tend to grow slowly after birth. The asymmetrical growth restricted babies tend to catch up growth in early infancy.
2. Increased chances of retarded neurological, intellectual, cognitive and behavioural development and cerebral palsy in infancy especially in symmetrical FGR.
3. *Increased risk of cardiovascular disease, type 2 diabetes and hyperlipidemia in adulthood (Barker's hypothesis 1992) as such a fetus is programmed to exhibit a 'thrifty phenomenon'* with increased food intake, fat deposition and decreased energy output. However, other authors have challenged this concept.

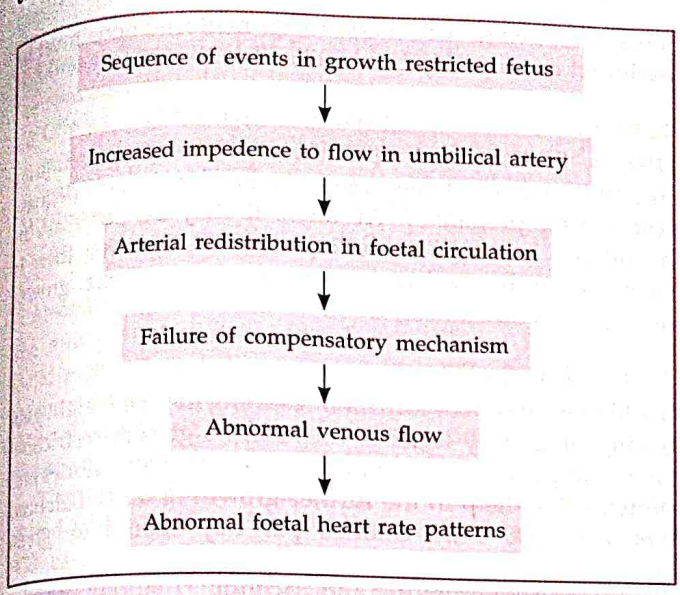



**Fig. 34.4:** Sequence of events in growth restricted fetus

### Mortality

There is 6 to 8 fold increase in intrapartum and neonatal deaths. Most of the deaths occur within first 24 hours.

## Prediction and Screening

Accurate prediction of the pregnancies destined to develop FGR would allow increased vigilance and foetal monitoring to enable interventions to improve outcome.

## History and Examination

The various risk factors and causes should be enquired and observed.

## Maternal Serum Screening

Various biochemical markers (AFP, $uE_3$, hPL and hCG) in the maternal serum in the second trimester have been used to detect foetal growth restriction in later pregnancy. PAPP-A <0.4 MoM in the first trimester is also used as a predictor for development of FGR later.

## Ultrasound Markers

Abnormal uterine artery Doppler velocimetry and bright or echogenic foetal bowel in the second trimester are associated with an increased risk of subsequent FGR. The combination of unexplained elevated maternal AFP with abnormal uterine artery Doppler velocimetry or echogenic bowel is a better predictor of adverse perinatal outcome.

## Scoring Systems

Several different scoring systems utilizing various risk factors for reduced foetal growth have been devised to better predict women at particular risk of having an FGR baby. Due to poor specificity and sensitivity, they are of limited clinical use.

## Diagnosis

### Clinical Examination

1. On obstetric palpation, if the height of the uterus is less by 4 weeks or more than the estimated gestation, foetal growth restriction should be suspected. An early accurate estimation of gestational age is needed for this method.
2. Symphysisfundal (symphyseofundal) height. Carefully performed serial fundal height measurement throughout pregnancy is a simple, safe, inexpensive, and reasonably accurate method to detect small for gestational age fetuses (see Chapters 8 and 9 for details). The uterine height in centimeters coincides with weeks of gestation between 18 and 30 weeks. If the measurement is 2–3 cm less, foetal growth restriction may be suspected. ACOG found it to be 70–85% sensitive and 96% specific. Serial measurements are needed.
3. Static or falling maternal weight after 20 weeks may indicate in FGR.
4. Static or falling abdominal girth measured at umbilicus may indicate FGR. The stage based classification and management of FGR is given in Table 34.12.

### Ultrasound Measurements

#### *1. Foetal Biometry*

In foetal biometry, foetal biparietal diameter (BPD), head circumference (HC), femur length (FL) and abdominal circumference (AC) are measured to estimate the approximate foetal size. Of all these measurements, AC is considered to correlate with foetal weight. An AC within normal range for gestational age rules out FGR while an AC of < 5th percentile is highly suggestive of FGR (ACOG, 2000). It should be measured at the level of bifurcation of hepatic veins in the center of foetal liver. HC should be measured at the level of thalami.

#### *2. Head Circumference (HC) and Abdominal Circumference (AC) Ratio*

It is a useful ratio to diagnose and classify FGR especially late growth restriction. Normally, the HC/AC ratio is > 1.0 before 32 weeks, 1.0 at 32–34 weeks and < 1.0 after 34 weeks. In an late FGR fetus, the HC remains bigger, HC/AC ratio is increased. In early FGR the HC/AC ratio remains normal due to reduced HC and AC. Using HC/AC ratio, 85% FGR fetuses are diagnosed with reduced false negative rate. AC **is the most sensitive parameter to diagnose** FGR. Serial measurements are more useful.

#### *3. Femur length*

Femur length (FL) is relatively spared in late FGR. The normal FL/AC ratio is 22 from 21st weeks till term and does not need accurate gestation. A ratio > 23.5 diagnosis FGR.

#### *4. Amniotic Fluid Volume*

Amniotic fluid volume is usually reduced in late FGR. Vertical pocket of amniotic fluid < 2 cm and amniotic fluid index (AFI) less than 5 indicates oligohydramnios and FGR (see Chapters 4 and 15 for details).

#### *5. Ponderal index (PI)*

It is rarely used now. Ponderal index (PI) is calculated by dividing the estimated foetal weight in grams by the cube

of crown-heel length in centimeters (weight/length$^3$). PI below 10th percentile is taken as FGR. It is usually < 2 in late FGR and 2 or more in early FGR and normal growth.

### *6. Other ultrasonic parameters*

Reduction in foetal facial fat store has been associated with FGR. Foetal structural abnormalities can also be excluded by ultrasonographic evaluation. Ultrasound does not predict all cases of FGR but has high negative predictive value. Transverse cerebellar diameter and AC (TCD/AC) ratio (normal 0.137) can be used for diagnosis of FGR.

#### Doppler Velocimetry

Elevated uterine artery systolic/diastolic (S/D) ratio (> 2.6) and/or presence of diastolic notch are associated with development of FGR. In a normal pregnancy, the diastolic flow increases with time. In a pregnancy complicated by placental vascular insufficiency, diastolic flow decreases leading on to increased in UA S/D ratio to more than 2 standard deviation (SD) above the mean for the gestational age. However, with worsening placental vascular insufficiency, UV waveform can show absent end diastolic flow and finally reversal end diastolic flow indicating foetal hypoxia and need to deliver the fetus urgently (see Chapter 15, Figs. 15.5–15.10 and Chapter 46 and Fig. 46.4 for details). Pulsatility index and sequential changes are used.

## Physical features at birth

See Chapter 28 for details.

# MANAGEMENT

## Uncomplicated FGR before 37 weeks of gestation

If intrauterine restriction is suspected or diagnosed before 37 weeks of gestation, the objective of management will be to monitor the further growth of the fetus and assess foetal well-being. Any major foetal malformations must be excluded as they may be associated with FGR.

### 1. Rest

Rest is particularly important in developing countries like India where women may not be able to get adequate rest during pregnancy. At times it may be necessary to hospitalise her to ensure adequate rest. However, there is no evidence that bed rest causes accelerated foetal growth or improves outcome.

### 2. High Protein Diet

Inadequate and poor nutrition resulting in poor weight gain during pregnancy leads to intrauterine growth restriction. Hence, improving dietary intake with adequate calories and proteins may help in the further growth of fetus. Balanced diet with 300 extra calories per day should be given

### 3. Avoidance of Smoking and Alcohol

### 4. Pharmacological Interventions

Low dose aspirin, essential amino acids, arginine and lycopene are often used in FGR but their role is doubtful. Progestogens especially allylestrenol have been used in various studies to prevent FGR and preterm labour by increasing placental blood flow and secretion of placental enzymes, relaxing uterine muscles with accelerated foetal growth by its beta-adrenergic action. They can be given as allylestrenol (oral), 10 mg twice daily, oral natural micronized progesterone 200–400 mg daily, dydrogesterone 10 mg twice daily in second and third trimester.

Unfortunately no specific treatment improves outcome in FGR remote from term. Nutrient supplementation, attempting plasma volume expansion, oxygen therapy, antihypertensive therapy, heparin and aspirin have all been found to be ineffective (American College of Obstetricians and Gynecologists, 2000).

**Table 34.11: Stage-based classification and management of FGR**

| Stage | Pathophysiological correlate | Criteria (any of) | | Monitoring* | GA/mode of delivery |
|---|---|---|---|---|---|
| I | Severe smallness or mild placental insufficiency | EFW < 3rd centile<br>UA PI > p95<br>UtA PI > p95 | CPR < p5<br>MCA PI <p5 | Weekly | 37 weeks<br>LI |
| II | Severe placental insufficiency | UA AEDV Reverse AoI | | Biweekly | 34 weeks<br>CS |
| III | Low-suspicion foetal acidosis | UA REDV<br>DV-PI >p95 | | 1– 2 days | 30 weeks<br>CS |
| IV | High-suspicion foetal acidosis | DV reverse a flow<br>cCTG < 3 ms<br>FHR decelerations | | 12 h | 26 weeks**<br>CS |

All Doppler signs described above should be confirmed at least twice, ideally at least 12 h apart. GA = Gestational age; L1 = labour induction; CS = cesarean section. * Recommended intervals in the absence of severe pre-eclampsia. If FGR is accompanied by this complication, strict foetal monitoring is warranted regardless of the stage. ** Lower GA threshold recommended according to current literature figures reporting at least 50% intact survival. Threshold could be tailored according to parents' wishes or adjusted according to local statistics of intact survival.

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### 5. Clinical Assessment of Foetal Growth

See above for details.

(i) **Clinical assessment of foetal growth.** Clinically, foetal growth is assessed by maternal weight gain and uterine growth charts which are measured weekly.

(ii) **Ultrasound assessment.** Foetal biometry is performed (see above for details) every 2 weeks.

### 6. Assessment of foetal well-being

The aim of antepartum foetal surveillance is to predict foetal asphyxia and thus prevent foetal death. FGR fetuses are more prone to intrauterine asphyxia. Careful assessment of foetal well-being and timely intervention when asphyxia is suspected improves perinatal outcome.

The tests of foetal well-being are discussed in Chapter 15 and tables 34.13 and 34.14. Dopper has definite role for FGR cases. Doppler studies of uterine arteries, umblical arteries, middle cerebral artery, ductus venosus are done (also see chapters 15 and 46 for details).

(i) **Growth Restriction Intervention Trial (GRIT 2004).** It was a multicentric RCT in European countries to compare effect of immediate delivery within 48 hours of steroid therapy and delayed delivery. There was no significant difference in two groups in perinatal outcome. Hence, delivery is indicated, when foetal danger is anticipated.

(ii) TRUFFLE (Trial of Randomized Umbilical and Foetal Flow in Europe) trial (2015): Ductus venosus Doppler evaluation was compared with foetal heart rate monitoring in 310 pregnancies between 26–32 weeks with FGR. The study concluded that before 34 weeks delaying delivery until ductus venous Doppler or foetal heart rate abnormalities appear is safe and benefits long term outcome.

(iii) DIGITAT (Disproportionate Intrauterine Growth Intervention Trial at Term) study (2010) examined the delivery timing of growth restricted fetuses who were at 36 weeks or older. The women were randomised to induction or expectant management. Neonatal morbidity at birth and neurodevelopmental and behaviour outcomes at 2 years were similar in 2 groups.

**Table 34.13: Tests for Foetal Surveillance in IUGR**

| |
|---|
| **A. Long term (valid for 2 weeks)** |
| 1. Uterine artery Doppler<br>2. Placental morphology<br>3. Foetal biometry |
| **B. Medium term (valid for 1 week)** |
| 1. Umbilical artery Doppler<br>2. Amniotic fluid volume and index |
| **C. Short term (valid for 3–4 days)** |
| 1. Non-stress test<br>2. Biophysical profile score<br>3. Foetal middle cerebral artery (MCA) and ductus venosus Doppler studies |

### 7. Obstetric Intervention

If the foetal growth is satisfactory and the foetal surveillance tests indicate that the fetus is not in distress, then the pregnancy is allowed to continue till 37 weeks. However, if the fetus is not well according to the biophysical profile or for severe degree of FGR, then termination of pregnancy is required as per table 34.12. This decision will have to be taken according to the neonatal care available since these newborns require a good neonatal unit to survive.

Pulmonary maturity tests can be performed and four doses (6 mg each) of dexamethasone or two doses (12 mg each) of betamethasone are given for pulmonary maturation before 34 weeks gestation as they reduce the incidence of hyaline membrane disease, cerebral haemorrhage and death in fetuses weighing less than 1.5 kg.

## TERMINATION OF PREGNANCY

Pregnancy is terminated based on stage of disease and also depends on various factors like gestational age, any congenital abnormalities, severity and type of FGR, past obstetric history, severity of foetal compromise by NST, CTG and biophysical profile.

## WARNING SIGNS

Prompt intervention to be done:

1. Progression of umbilical artery end diastolic flow (EDF) from reduced to absent and then to reverse flow
2. CTG—Lack of variability, presence of late decelerations

**Table 34.14: Interpretation of Antenatal Testing**

| Antenatal testing | Interpretation |
|---|---|
| 1. Positive UA EDV Normal MCA and DV, amniotic fluid pocket > 2, BPS > 8/10 | **Normal. Asphyxia (hypoxia) extremely rare** |
| 2. Same as above with brain sparing MCA | Blood flow redistribution, **hypoxia possible**, higher risk of intrapartum distress |
| 3. UA EDV, DV normal, BPS > 8/10, amniotic fluid pocket < 2 | Significant redistribution, **hypoxia common,** asphyxia/acidemia possible. **Onset of foetal compromise** |
| 4. Significant redistribution, increased DV pulsatility, BPS > 6/10, amniotic fluid < 2 | **Foetal compromise, hypoxia common.** Asphyxia and acidemia likely |
| 5. Compromise by above criteria, BPS < 6/10, amniotic fluid < 2 | **Foetal decompensation** and cardiovascular/ metabolic compromise, **imminent stillbirth, poor outcome** |

UA EDV: Umbilical artery end diastolic velocity; MCA: Middle cerebral artery; BPS: Biophysical score; DV: Ductus venosus; REDV: Reversal of end diastolic velocity

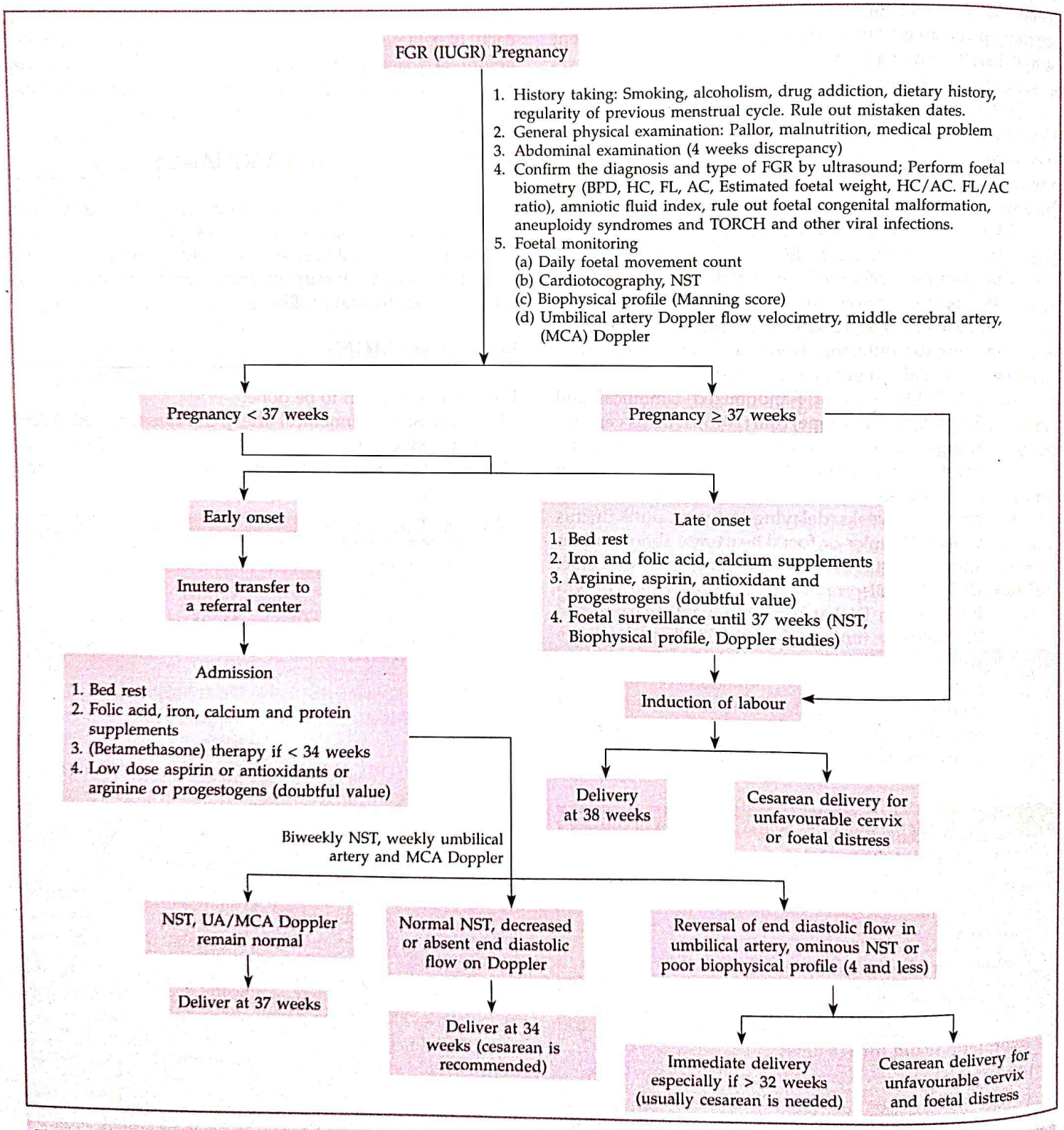
3. Biophysical score < 4
4. Abnormal venous Doppler in presence of absent EDF in umbilical artery
5. Oligohydramnios

## INDUCTION OF LABOUR

Such women should be delivered in well-equipped centers (tertiary referral hospitals) with facilities for intrapartum continuous foetal heart monitoring, foetal blood sampling and neonatal intensive care unit (NICU) facilities.

Mode of delivery depends mainly upon gestational age and any foetal distress. Vaginal delivery is allowed if there is no indication of cesarean delivery and foetal heart rate is normal. Fetuses with major congenital abnormalities should also be delivered vaginally.

Induction of labour is performed depending upon Bishop's score. If Bishop's score is unfavourable (< 6),




**Flow Chart 34.3:** Management of FGR (IUGR)

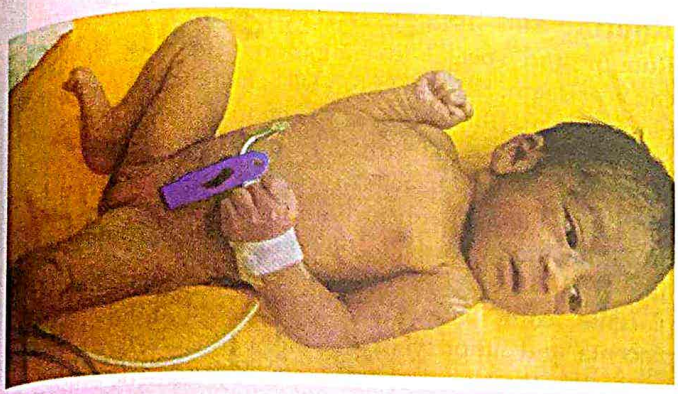


**Fig. 34.5:** A newborn baby with foetal growth restriction delivered at 37 weeks period of gestation weighing 1.4 kg

prostagladin $E_2$ gel is used. For favourable cervix, amniotomy followed by oxytocin drip is started.

**Intrapartum surveillance** is performed preferably by continuous cardiotocography due to high risk of foetal distress (see Chapter 15 for details).

**Cesarean delivery** is performed for foetal distress or jeopardy like repetitive late decelerations, poor biophysical score, reversal of end diastolic flow in umbilical artery, abnormal ductus venosus Doppler and foetal scalp pH acidosis.

### Care during vaginal delivery (Fig. 34.5)

Such women are delivered in tertiary referral centres with neonatal intensive care unit (NICU) facilities to look after FGR neonates. A neonatologist (pediatrician) should be in attendance.

### Immediate care of the baby after birth

A neonatologist (pediatrician) should attend to the baby. The baby is usually shifted to neonatal intensive care unit. (see Chapter 28 for details).

## INTRAUTERINE FOETAL DEATH (IUFD OR IUD)

**Definition.** Intrauterine foetal death (IUFD), intrauterine death (IUD) or stillbirth is defined *as delivery of a baby after period of viability with no signs of life and includes all foetal deaths occurring after 28 weeks (24 weeks in UK, 22 weeks and > 500 g by WHO) of gestation during pregnancy and during labour.*

## INCIDENCE

The foetal death rate (number of foetal deaths per 1000 births) varies from 7.5 per 1000 births in USA to about 35 per 1000 births in India. There has been a gradual decrease in the incidence of IUD due to preconceptional care, better care during pregnancy and labour, prenatal diagnosis of major congenital malformations and their selective termination.

## ETIOLOGY

The various causes are as follows:

### Foetal Causes (25–40% most common)

1. **Congenital malformations.** (*i*) Chromosomal defects (*ii*) non-chromosomal disorders: neural-tube defects, isolated hydrocephalus and complex congenital heart diseases are the most common.
2. **Isoimmunization** due to Rh or other blood groups like Kell, Duffy (see Chapter 38 for details).
3. **Infections.** Congenital syphilis, cytomegalovirus, toxoplasmosis, parvovirus B-19, rubella and varicella infections and listeriosis.
4. **Non immune hydrops**

### Placental Causes (15–25%)

1. Placental abruption and placenta previa through acute placental insufficiency.
2. Placental and cord abnormalities. Pregnancies with circumvallate placenta, velamentous insertion of cord (vasa previa), true knots in the cord, torsion, stricture and haematoma in the cord are rare causes of foetal death. Prolapse of the umbilical cord is an important cause.
3. Chorioamnionitis
4. Intrapartum asphyxia
5. Twin-to-twin transfusion syndrome
6. Fetomaternal haemorrhage
7. Excessive coffee intake

### Maternal Causes (5–10%)

1. Hypertensive disorders, especially pre-eclampsia, due to large placental infarcts, abruptio placentae and markedly small placental size.
2. Diabetes mellitus
3. Medical disorders like heart disease and renal disease
4. Hereditary thrombophilias and antiphospholipid antibodies syndrome due to decidual vasculopathy, placental infarction and placental abruption.
5. Obstetric cholestasis
6. Trauma, sepsis, acidosis and shock
7. Labour complications: Abnormal labour, mismanaged labour and uterine rupture
8. Hyperpyrexia due to any cause
9. Post term pregnancy

### Unexplained (25–35%)

Unexplained causes can be reduced to 10% by meticulous autopsy.

### Pathology

Depending upon the degree of maceration by careful examination of the dead infant, time of death can be determined. There is reddened skin in 6–8 hours, skin slippage and peeling in 8–12 hours (grade II maceration), extensive skin peeling, red serous effusions in chest and abdomen in 2–7 days (grade III maceration). After 5–7 days, aseptic autolysis of the ligaments and liquefaction of other structures like brain occur. These changes are the basis for the typical radiological signs of intrauterine death.

## Diagnosis
Repeated examinations are often required to confirm the diagnosis.

## Symptoms
Up to 50% patients may complain of decreased or absent foetal movements or some women may complain of pain in the abdomen and bleeding per vaginum.

## Signs
There is regression of pregnancy signs like breast changes following death of the fetus.

**Per Abdomen**

1. Stationary or decrease in the fundal height.
2. Decrease in the tone of uterus which feels flaccid. Braxton-Hicks contractions are not felt easily.
3. Foetal movements are absent during palpation.
4. **Foetal heart sounds** are not heard by stethoscope. It is confirmed by ultrasound.
5. **Egg-shell crackling feel** of the foetal head appears after many days of intrauterine death.

## Investigations
The investigations are performed to confirm the diagnosis, to identify the cause of death and to follow-up these parients.

## Ultrasonography
Failure to detect heart motion by real time ultrasound is a reliable evidence of foetal death. It should be done by a trained person. Other findings on ultrasonography include scalp edema and collapsed cranial bones which are overlapping. Ultrasonography can also provide some clue to the cause of death in a few cases and may show abruption, oligohydramnios or signs of foetal hydrops.

## Straight X-ray Abdomen
X-ray abdomen was done in the past before availability of ultrasound. Signs on the X-ray are seen if foetal demise has occurred several days previously. There are five principal radiological signs of foetal death.

1. **Spalding's sign.** There is irregular overlapping of the skull bones on one another because of liquefaction of the brain matter and softening of the ligamentous structures of the skull (Fig. 34.6). Time of appearance is about 7 days after death.
2. **The halo sign.** There is abnormal increase of the cranial soft tissue as a result of maceration **(The halo sign)**.
3. Exaggerated curvature (hyperflexion) of the foetal spine **(Ball's sign)** (Fig. 34.6). As this sign depends on maceration of the spinous ligaments, its development requires several days. Moreover, mild degree of curvature of spine in the living fetus may be misleading.
4. Demonstration of gas in the fetus is an uncommon but reliable sign of foetal death. Demonstration of gas shadow **(Robert's sign)** in the foetal atria or ventricles or in big vessels are visible in 12 hours of intrauterine death. **It is the first sign of IUD.**
5. Crowding of ribs **(concertina effect)**

## Routine and Special Investigations
Routine and special investigations are done to find the cause of death. Hb, ABO Rh, VDRL, fasting and postprandial blood sugar, $HBA_1C$ levels, Kleihauer Betke test, thyroid function tests, renal function tests, coagulation profile, TORCH screening, urine complete examination and lupus anticoagulant and anticardiolipin antibodies. Coagulation profile including bedside bleeding time (BT) and clotting time (CT) are carried out. Naked eye examination of the placenta, cord, baby (Table 34.13) and histology of the placenta and autopsy of the baby (if allowed) may give some clue to the cause of death. Cytogenetic studies by karyotyping of the baby are helpful in the presence of congenital malformation or intrauterine growth restriction. Work up for thrombophilia includes lupus anticoagulant, anticardiolipin level, factor V leiden mutation, antithrombin III levels.

# COMPLICATIONS

1. **Psychological upset.** Carrying a dead fetus causes psychological stress needing special attention and sympathy by the attending medical team and nursing staff.
2. **Blood coagulopathies.** Retained fetus inutero for some time before being expelled can cause consumptive coagulopathy, presumably mediated by thromboplastin

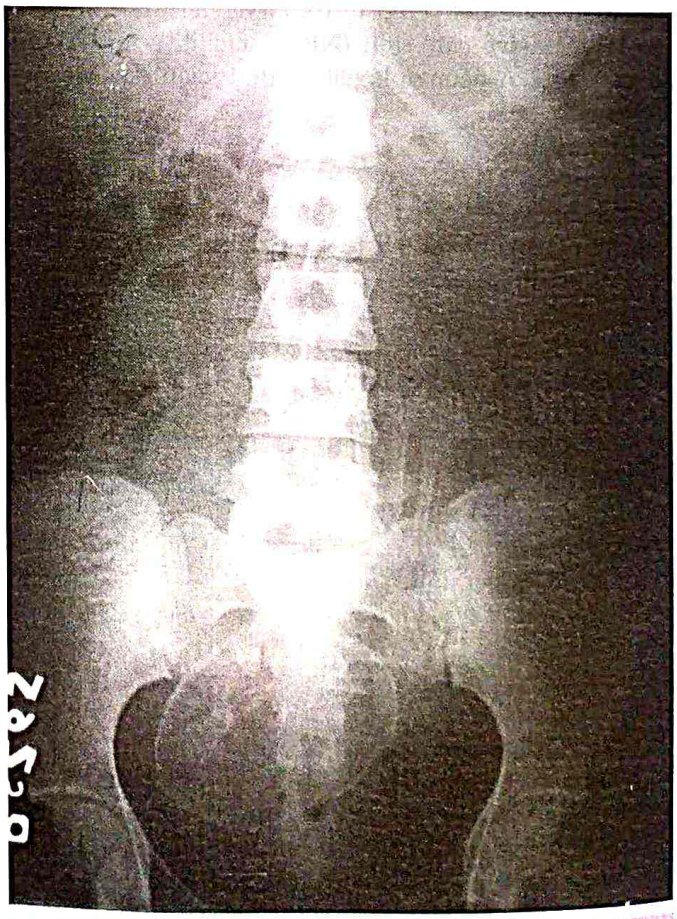


**Fig. 34.6:** X-ray abdomen showing Spalding's sign (overlapping of skull bones) and Ball's sign (hyperflexion of spine) in intrauterine foetal death

## Diagnosis

Repeated examinations are often required to confirm the diagnosis.

## Symptoms

Up to 50% patients may complain of decreased or absent foetal movements or some women may complain of pain in the abdomen and bleeding per vaginum.

## Signs

There is regression of pregnancy signs like breast changes following death of the fetus.

**Per Abdomen**

1. Stationary or decrease in the fundal height.
2. Decrease in the tone of uterus which feels flaccid. Braxton-Hicks contractions are not felt easily.
3. Foetal movements are absent during palpation.
4. **Foetal heart sounds** are not heard by stethoscope. It is confirmed by ultrasound.
5. **Egg-shell crackling feel** of the foetal head appears after many days of intrauterine death.

## Investigations

The investigations are performed to confirm the diagnosis, to identify the cause of death and to follow-up these parients.

## Ultrasonography

Failure to detect heart motion by real time ultrasound is a reliable evidence of foetal death. It should be done by a trained person. Other findings on ultrasonography include scalp edema and collapsed cranial bones which are overlapping. Ultrasonography can also provide some clue to the cause of death in a few cases and may show abruption, oligohydramnios or signs of foetal hydrops.

## Straight X-ray Abdomen

X-ray abdomen was done in the past before availability of ultrasound. Signs on the X-ray are seen if foetal demise has occurred several days previously. There are five principal radiological signs of foetal death.

1. **Spalding's sign.** There is irregular overlapping of the skull bones on one another because of liquefaction of the brain matter and softening of the ligamentous structures of the skull (Fig. 34.6). Time of appearance is about 7 days after death.
2. **The halo sign.** There is abnormal increase of the cranial soft tissue as a result of maceration **(The halo sign).**
3. Exaggerated curvature (hyperflexion) of the foetal spine **(Ball's sign)** (Fig. 34.6). As this sign depends on maceration of the spinous ligaments, its development requires several days. Moreover, mild degree of curvature of spine in the living fetus may be misleading.
4. Demonstration of gas in the fetus is an uncommon but reliable sign of foetal death. Demonstration of gas shadow **(Robert's sign)** in the foetal atria or ventricles or in big vessels are visible in 12 hours of intrauterine death. **It is the first sign of IUD.**
5. Crowding of ribs **(concertina effect)**

## Routine and Special Investigations

Routine and special investigations are done to find the cause of death. Hb, ABO Rh, VDRL, fasting and postprandial blood sugar, $HBA_1C$ levels, Kleihauer Betke test, thyroid function tests, renal function tests, coagulation profile, TORCH screening, urine complete examination and lupus anticoagulant and anticardiolipin antibodies. Coagulation profile including bedside bleeding time (BT) and clotting time (CT) are carried out. Naked eye examination of the placenta, cord, baby (Table 34.13) and histology of the placenta and autopsy of the baby (if allowed) may give some clue to the cause of death. Cytogenetic studies by karyotyping of the baby are helpful in the presence of congenital malformation or intrauterine growth restriction. Work up for thrombophilia includes lupus anticoagulant, anticardiolipin level, factor V leiden mutation, antithrombin III levels.

# COMPLICATIONS

1. **Psychological upset.** Carrying a dead fetus causes psychological stress needing special attention and sympathy by the attending medical team and nursing staff.
2. **Blood coagulopathies.** Retained fetus inutero for some time before being expelled can cause consumptive coagulopathy, presumably mediated by thromboplastin

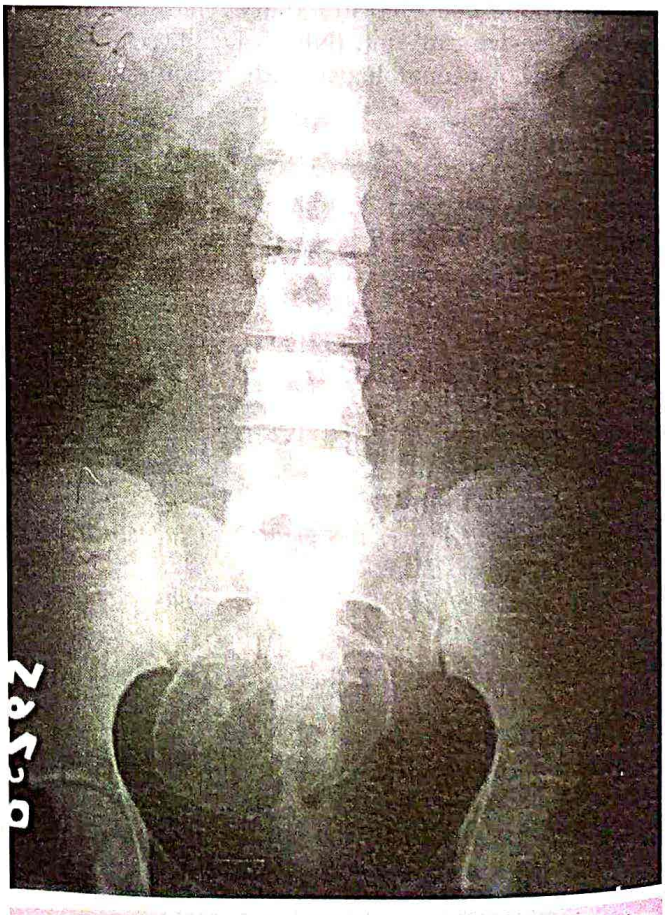


**Fig. 34.6:** X-ray abdomen showing Spalding's sign (overlapping of skull bones) and Ball's sign (hyperflexion of spine) in intrauterine foetal death

from the dead products of conception. If the dead fetus is retained longer (more than a month), about 25% women develop coagulopathy. The fibrinogen concentration falls and in some cases the decrease may reach potentially dangerous concentrations of < 100 mg/dL. Fibrin degradation products are elevated and platelet counts tend to decrease.

3. **Infection.** With intact membranes, infection usually does not start. However, after rupture of membranes, ascending infection can set in causing chorioamnionitis and septicemia.
4. **During labour.** Uterine inertia, retained placenta and postpartum haemorrhage may occur during labour. High doses of intravenous oxytocin may lead to hyponatremia and rupture uterus. DIC may also occur.

## PREVENTION

Intrauterine death cannot be totally prevented, but the following guidelines can reduce its incidence.

1. Pre-conceptional counselling should be performed in all cases so that women conceive when they are in optimum health.
2. Universal and effective hospital antenatal care can pick up high risk cases in regular early stages when the problem can be easily tackled.
3. High risk pregnancies can be detected at the earliest and regularly monitored for maternal and foetal well-being for timely intervention.

## MANAGEMENT

The scheme of management is shown in Flow Chart 34.4.

### Expectant Management

The majority of women (up to 80%) go into spontaneous labour within 2 weeks of foetal death. In the absence of other complications, attempts to evacuate the uterus may be delayed for this interval. The patient and her relatives are usually upset psychologically but should be assured of the safety of non-interference.

### Interference

The indications of interference are:

1. Tendency of prolongation of pregnancy beyond 2 weeks.
2. Wish of the patient due to psychological upset is common.
3. Falling fibrinogen level: The fibrinogen level should be corrected by blood transfusion before interference.
4. Onset of uterine infection

Early termination is favored these days to avoid psychological upsetting of women and preventing complications. Moreover, diagnosis can be made with certainty and prostaglandin $E_2$ gel is available, which is highly efficacious for induction of labour even with poor Bishop's score.

### Methods of Termination

Patient is counselled about induction of labour. **Amniotomy (ARM) is contraindicated for fear of infection.**

1. **Prostaglandins.** If cervix is unfavourable (Bishop < 6), prostaglandin $E_2$ gel vaginally or intracervically is given and repeated every 6 hourly for 3 doses. Alternatively misoprostol ($PGE_1$) 50 µg vaginally or orally every 6 hourly is also effective. Mifepristone (RU486) in dose of 600 mg followed by misoprostol can also be used to reduce induction delivery interval.
2. **Oxytocin infusion.** This is method of choice in cases where the cervix is favourable (Bishop > 6). Initially 5–10 units of oxytocin in 500 mL of Ringer's lactate or saline solution is started intravenously followed by an escalating dose of oxytocin (see Chapters 14 and 18 for details). Secondary abdominal pregnancy must be excluded in a failure case.

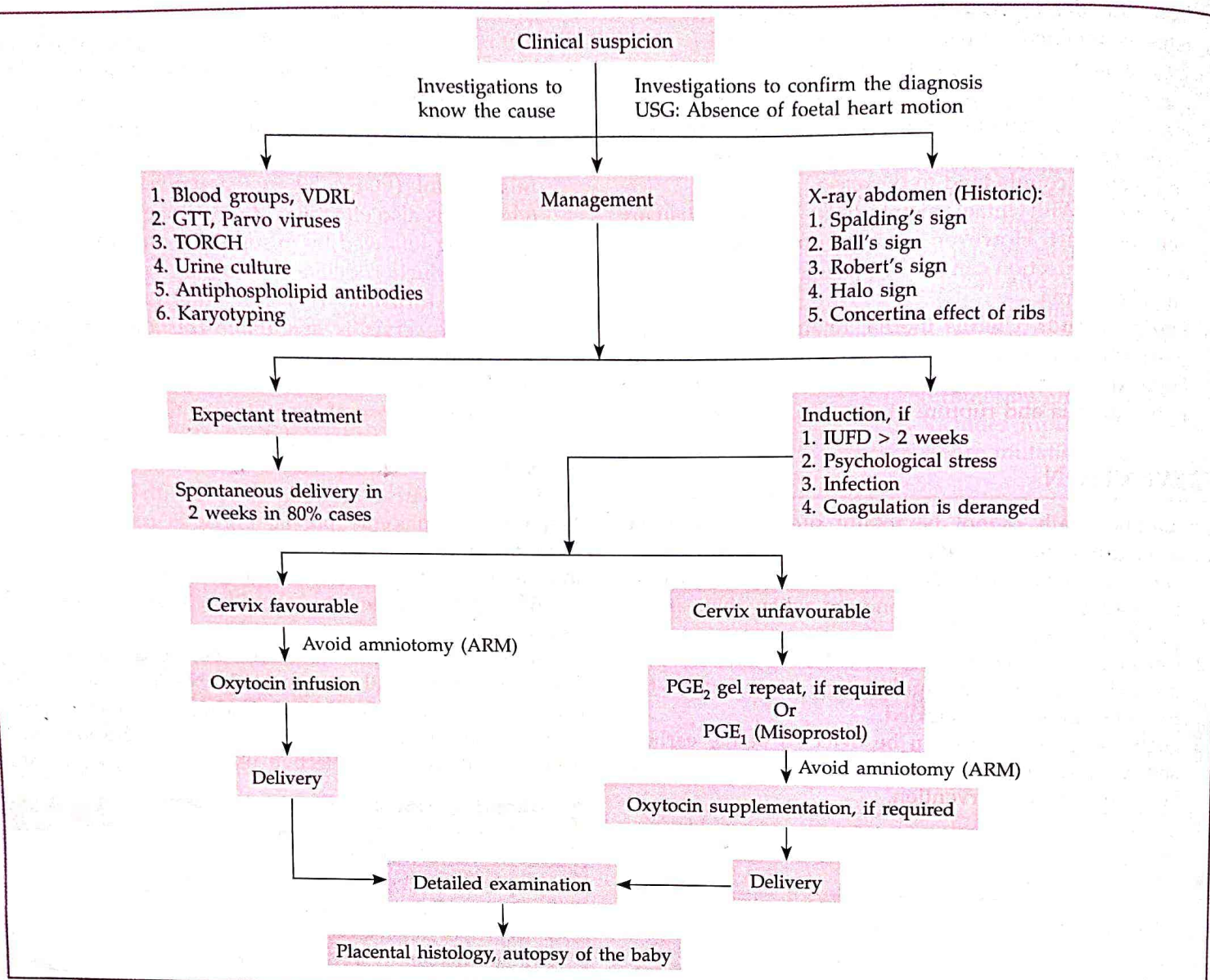
**Place of cesarean section** in a case with IUD is limited. Major degree placenta previa, previous cesarean section (two or more), transverse lie and other strict obstetric indications for cesarean delivery require it.

After delivery, the fetus should be examined carefully as shown in Table 34.15.

**Autopsy.** Patients should be counselled and encouraged for a full autopsy. Valuable information also can be obtained from limited studies. A gross external examination, along with photography, radiography, MRI, bacterial cultures and selective use of chromosomal and

**Table 34.15: Guidance for Examination of Stillborn Infant**

| |
|---|
| **Gross examination of infant** |
| 1. Skin staining<br>2. Colour—Pale, plethoric<br>3. Degree of maceration<br>4. Malformations |
| **Placenta** |
| 1. Weight<br>2. Structural abnormalities—Circumvallate or accessory lobes, velamentous insertion<br>3. Hydropic changes<br>4. Any retroplacental clots<br>5. Meconium staining |
| **Membranes** |
| 1. Meconium stained or cloudy<br>2. Thickening of membranes<br>3. Foul smell |
| **Umbilical Cord** |
| 1. Entanglement around neck, arms, legs<br>2. Prolapse<br>3. Length<br>4. Any true or false knot<br>5. Hematomas or strictures<br>6. Wharton jelly—Normal, absent |
| **Amnionic Fluid** |
| 1. Colour—Meconium, blood<br>2. Volume<br>3. Consistency |




**Flow chart 34.4:** Scheme management of intrauterine foetal death

histopathology studies can help in determining the cause of death.

**Psychological aspects.** Foetal death is psychologically traumatic for the woman and her family. It increases further if there is an interval of more than 24 hours between the diagnosis of foetal death and the induction of labour. She should be encouraged to see her infant for as long as she desires and can have photograph of the child for remembrance. She is at increased risk for postpartum depression and should be closely monitored.

**Bereavement counselling and puerperium.** The obstetrician or midwife (nurse) with special interest and expertise in the area should spend time with the woman and her husband providing them psychological support in simple language and should discuss with them the reason of foetal death. The woman should not lie with other women with live babies as she is at risk of puerperal depression. At the follow-up visit after 6 weeks, doctor should discuss with them the result of autopsy and investigations and planning for future pregnancy.

# POST TERM PREGNANCY (SYN: POSTDATISM, POSTMATURITY, PROLONGED PREGNANCY)

## DEFINITION

The American College of Obstetricians and Gynecologists (ACOG 2016), the World Health Organization (WHO) and the International Federation of Gynecology and Obstetrics (FIGO) have defined prolonged pregnancy as pregnancy at 42 completed weeks of gestation (294 days) from the first day of the last menstrual period.

ACOG (2013) calls 37 to 38.6 weeks as early term, 39 to 40.6 weeks as full term, 41–41.6 weeks as late term and 42 weeks onwards as post term.

## INCIDENCE

Range 4–19%. Average incidence: 10%

## ETIOLOGY

As the exact mechanism of initiation of labour remains unknown, the causes of the prolongation of pregnancy remain obscure. The following factors are related with post-maturity.

1. **Mistaken dates**—An error in calculation of gestational age is the most common cause as many women do not exactly remember their date of last menstrual period (LMP).
2. **Genetic factors.** Heredity may play a role as prolonged pregnancy is seen in generations in Swedish women.
3. **Demographic factors.** No consistent relationship with age and nutrition. **History of postterm pregnancy in the past is the most significant risk factor for prolonged pregnancy**. Sedentary lifestyle and elderly multiparas are at risk.
4. **Foetal causes.** Congenital foetal malformations like anencephaly which disrupt foetal HPA axis and adrenal hypoplasia cause post-datism due to poor foetal cortisol response failing to start labour.
5. **Placental factors.** (a) Placental sulphatase enzyme deficiency causes low estrogen. (b) Deficiency of placental CRH can also cause post datism
6. **Unknown.** Cause remains unknown in most pregnancies.

## DIAGNOSIS

The diagnosis of prolonged pregnancy requires accurate estimation of gestational age (see Chapter 8 for details).

The following are the useful clinical guidelines:

1. Menstrual history. If she reliably remembers date of last menstrual period.
2. From early pregnancy test
3. Early ultrasound in first or second trimester ACOG (2017) considers first trimester sonography to be the most accurate method to confirm gestational age.
4. From date of quickening
5. The clinical findings when a pregnancy is post date by two weeks are as follows:
   (a) **History of false labour pains.** Appearance of false labour pains followed by their disappearance is suggestive.
   (b) **Weight record.** There may be static or falling weight on periodic maternal weight estimation.
   (c) **Girth of the abdomen.** The girth of the abdomen becomes static or gradually declines because of decreasing amniotic fluid.
   (d) **Abdominal examination** shows static height of the uterus. There is more hardness of the skull bones. Due to oligohydamnios, the uterus feels **'full of fetus'**
   (e) **Vaginal examination.** There is increased prominence of foetal parts in post maturity. Cervix is usually ripe in post maturity. The skull bones are felt hard on vaginal examination which suggests mature fetus. Rule out cephalo-pelvic disproportion.

## INVESTIGATIONS

The investigations are performed to confirm the foetal maturity and to assess foetal well-being.

### Tests for Foetal Maturity

1. **Ultrasound abdomen.** It is the preferred modality and gives the following information.
   (a) Assessment of gestational age
   (b) Foetal weight
   (c) Foetal maturity (by BPD, HC, AC and femur length)
   (d) Restriction of foetal growth.
   (e) Loss of foetal subcutaneous fat.
   (f) Amount of liquor (oligohydramnios; amniotic fluid index < 5 cm)
   (g) Placental localization and maturity
2. **Doppler ultrasound** has no proven value till date in the surveillance of postterm fetuses. However, absent and reversal of end diastolic flow of blood in uterine arteries may suggest foetal jeopardy and is indication for termination of pregnancy.
3. **Amniocentesis.** The biochemical and cytological testing of amniotic fluid can tell foetal maturity, but being invasive method it is rarely done.
4. **Straight X-ray abdomen** was done in the past to see ossification centers in the long bones (lower end of femur at 36–37 weeks, upper end of tibia at 38–40 weeks). It is now replaced by ultrasound which gives all information accurately.

### Tests for Foetal surveillance

Assessment of foetal well-being is done by daily foetal movement count, twice weekly non-stress test, biophysical profile or modified biophysical profile (NST and amniotic fluid volume). Doppler studies can be done on umbilical and middle cerebral vessels of fetus. An AFI < 5 cm, poor biophysical profile or abnormal Doppler studies on foetal vessels necessitate prompt delivery (see Chapter 15 for details).

## COMPLICATIONS

### Maternal complications

Usually maternal health is not affected.

Various **intrapartum** complications are as follows:

1. Labour dysfunction (e.g. incoordinate uterine action)
2. Obstetric trauma
3. Increased operative and instrumental delivery
4. Haemorrhage

### Foetal complications

**Antepartum complications**

1. Oligohydramnios: Due to poor placental function leading to decreased renal blood flow and less urine output.
2. Cord compression
3. Macrosomia
4. Placental insufficiency (foetal hypoxia)

5. Foetal distress
6. Meconium stained liquor
7. Loss of subcutaneous fat
8. Foetal growth restriction

**Intrapartum complications**

1. Macrosomia
2. Shoulder dystocia—Greater risk of operative delivery
3. Decreased moulding capacity (because the skull bones are no longer pliable and soft)
4. Cord compression
5. Foetal asphyxia

**Postpartum Complications in the neonate**

1. **Post Maturity Syndrome.** The post mature infant has characteristic appearance with features like patchy peeling and wrinkled skin. It is particularly prominent on the palms and soles with long nails and a long, thin body suggesting wasting. There is advanced maturity because the infant is open-eyed, unusually alert and appears old and worried-looking. Most such post mature infants are not growth restricted. It occurs in 10% pregnancies between 41 and 43 weeks and in 33% at 44 weeks. Associated oligohydramnios substantially increases the likelihood of postmaturity.
2. Increased incidence of neonatal seizures
3. Severe meconium aspiration syndrome
4. Atelectasis of lungs
5. Hypoglycemia
6. Polycythemia
7. Hyperbilirubinemia
8. Low Apgar score
9. Abnormal neurological signs can be there.
10. Increased neonatal intensive care unit (NICU) admissions are needed.

**Perinatal morbidity and mortality** are increased. Stillbirth rates increase by eightfold from 0.7 per 1000 total births at 37 weeks to 5.8 per 1000 total births at 43 weeks.

The major causes of perinatal mortality include prolonged labour with cephalopelvic disproportion, operative intervention, unexplained anoxia and malformations.

## MANAGEMENT

Management of post term pregnancy is shown in Flow Chart 34.5.

### Uncomplicated Group

To reduce perinatal morbidity and mortality associated with prolonged pregnancy, it is necessary to initiate antepartum foetal monitoring from 40 weeks of gestation.

**Antepartum Foetal Surveillance**

(see above)

### Induction of Labour

**Indications**

1. At 42 completed weeks in all pregnancies
2. At any time for abnormal foetal surveillance
3. In India, most obstetricians tend to induce labour before 41 weeks.

**Method**

Stripping of the membranes is done if cervix admits tip of finger. Prostaglandin $E_2$ gel instillation is done in unfavourable cervix. For favourable cervix, amniotomy followed by oxytocin drip are preformed. Early artificial rupture of membranes helps in detection of thick meconium. To prevent its aspiration which may lead to severe respiratory distress and death, immediately delivery is a must. Foetal surveillance is continued. For suspected macrosomia, elective cesarean delivery is performed to avoid shoulder dystocia.

### Complicated Group

**(Associated with complicating factors)**

If post maturity is associated with complicating factors, the morbidity is multiplied as now there are two high risk factors (postmaturity and another factor). Hence, elective cesarean delivery should be performed for high-risk cases like malpresentations, malpositions, cephalopelvic disproportion and previous cesarean delivery. In cases of maternal diseases like diabetes, hypertension, renal diseases, intrahepatic cholestasis, induction of labour is indicated at term.

### Care During Labour

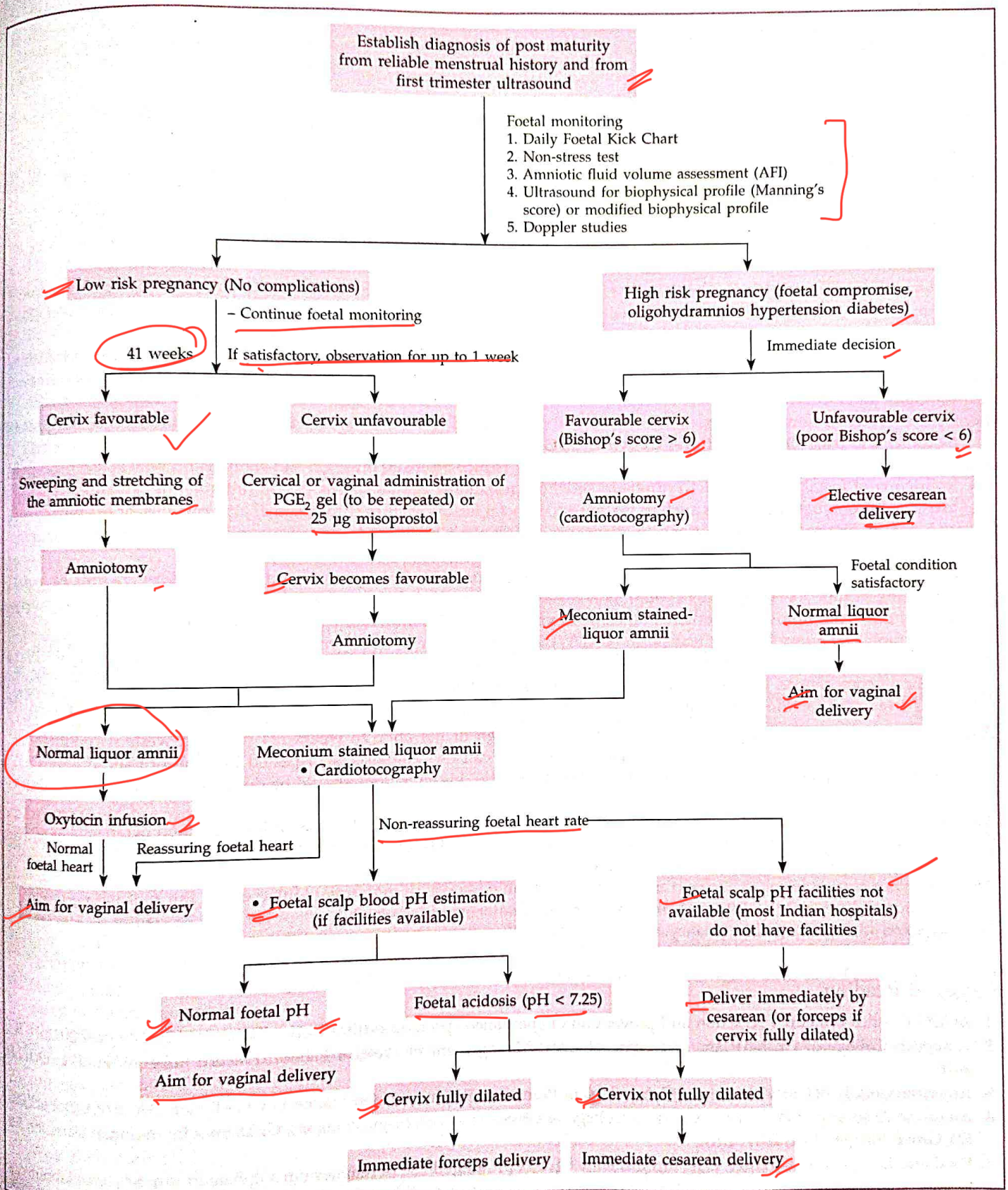
As it is a high risk case, careful vigilance should be maintained. Ideally, continuous electronic foetal monitoring should be used during labour to detect any foetal distress. There is a tendency for prolonged labour. Epidural analgesia is ideal but other analgesics (drotaverine) can also be given. Ideally, partogram should be maintained to detect any deviation of labour. If there is any foetal distress or tendency for prolongation of labour, cesarean delivery should be performed.

Forceps or ventouse delivery can be tried if pre-requisites are fulfilled. The newborn should be attended by the pediatrician. Routine intrapartum suction of pharynx of newborn for meconium is not recommended as such practices do not prevent or alter the meconium aspiration syndrome.

Summary of evaluation and management for post term pregnancy is given in Table 34.14.

**Table 34.14: Summary of Evaluation and Management of Post term Pregnancy**

1. Post term pregnancy is defined as a pregnancy that has extended to or beyond 42 completed weeks.
2. Women with a post term pregnancy with an unfavourable cervix can either undergo labour induction or be managed expectantly.
3. Prostaglandins can be used for cervical ripening and labour induction.
4. The woman should be delivered if there is evidence of foetal compromise or oligohydramnios.
5. Antenatal surveillance should be started between 41 and 42 weeks.
6. A non-stress test, amniotic fluid volume assessment or full biophysical profile (Manning's score) should be performed.
7. Many recommend prompt delivery in a woman with a post term pregnancy, a favourable cervix and no other complication.




**Flow chart 34.5:** Management of post term pregnancy

## Key Points

1. Preterm labour is defined as onset of labour prior to 37 (completed) weeks of pregnancy. Maternal systemic diseases, infections, uterine malformations, pregnancy complications like multiple pregnancy, hydramnios and antepartum haemorrhage contribute to preterm labour.
2. Tocolytic agents are used to inhibit preterm labour whenever indicated. Out of various tocolytic agents, ritodrine and nifedipine are commonly used. Tocolytic agents have no significant effect on perinatal mortality or the prolongation of pregnancy to term but do reduce the number of women delivering within 48 hours to enable the steroids to work for foetal maturity.
3. A single course of two doses of betamethasone 12 mg 24 hours apart given between 28 and 34 weeks gestation results in markedly improved neonatal outcome. There is no evidence of benefit for the use for antibiotics in uncomplicated preterm labour. Spontaneous rupture of membranes before onset of labour is called preterm rupture of membrane (PROM). It is called Term PROM is if it occurs after 37 weeks of gestation and Preterm PROM (PPROM) if it occurs before 37 weeks of gestation.
4. At term, the outcome for women with PROM is as good in women induced immediately as in those managed conservatively. Term PROM is usually a reflection of normal physiology, whereas PPROM is usually caused by pathological processes like infection and antepartum haemorrhage.
5. Accurate diagnosis of membrane rupture is essential and is done by history and speculum examination with avoidance of vaginal examination unless labour is advanced. Corticosteroid use in PPROM reduces the risk of respiratory distress syndrome in newborns.
6. Oral erythromycin but not coamoxyclav used for 10 days after PPROM is associated with significant reduction in neonatal mortality and major morbidity **(level IA evidence,** ORACLE **trial).**
7. In term PROM, early induction with oxytocin appears to reduce perinatal infection and shortens hospital stay.
8. Restriction of foetal growth inutero results in small for gestational age newborns. Foetal (intrauterine) growth restriction may result from maternal factors such as malnutrition, foetal factors such as chromosomal abnormalities or malformations and certain pregnancy complications. A diagnosis of FGR (IUGR) is made on ultrasound examination after accurate estimation of gestational age.
9. Pregnancy with suspected FGR needs to be carefully monitored for foetal growth and foetal well-being. Tests of foetal well-being include foetal movement count, non-stress test and foetal biophysical profile. Proper monitoring and timely intervention will improve the perinatal outcome in pregnancies with intrauterine growth restriction.
10. Use of umbilical artery Doppler waveform (UADW) analysis and use of liquor volume assessment is of benefit in FGR.
11. There is better outcome with timely intervention and delivery for FGR where the fetus is viable and shows signs of significant compromise. Giving steroids and delivery within 48 hours is recommended as delay does not improve outcome **(GRIT trial level I evidence).** There is role of cesarean section in the presence of significant abnormalities of the UADW.
12. Intrauterine foetal death is delivery of a baby with no sign of life after 28 weeks (24 weeks in western countries) of gestation and can be due to foetal, placental and maternal causes. Diagnosis is suspected from history and examination but confirmed by ultrasound (absence of foetal heart activity). Investigations should be done to know the cause of death and for coagulation profile.
13. Management of IUD is conservative initially or by induction of labour after 2 weeks or if coagulation derangement or on maternal request by prostaglandin gel. Newborn dead baby should be carefully examined. Autopsy should be encouraged. The couple should be offered bereavement counselling.
14. Post term pregnancies need antepartum surveillance from 40 weeks of gestation to intervene at the appropriate time. Induction of labour at the optimum time and careful monitoring of labour will reduce perinatal morbidity and mortality associated with prolonged pregnancy.

## Suggested Reading


1. ACOG Practice Bulletin. Prediction and prevention of spontaneos preterm birth. Practice Bullet no. 1234, August 2021.
2. American College of Obstetricians and Gynecologists: Management of Preterm Labour. Practice Bulletin No. 171, October 2016.
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9. Rouse D J, Hirtz D G, Thom E et al. A Randomized Controlled Trial of Magnesium Sulphate for the Prevention of Cerebral

Section 8
MEDICAL, SURGICAL, GYNECOLOGICAL AND NEOPLASTIC CONDITIONS DURING PREGNANCY

CHAPTER 35

# Medical Disorders During Pregnancy

## (i) HEMATOLOGICAL DISORDERS DURING PREGNANCY

### ANAEMIA IN PREGNANCY

Anaemia is the most common medical disorder in pregnancy especially in developing countries. Anaemia contributes almost 40% maternal deaths in developing countries. There is 8–10 fold increase in mortality if Hb is less than 5gm%.

### Physiological hematological changes in pregnancy

Normal physiological changes in blood indices are discussed in Chapter 6 and Table 6.3.

#### Erythropoiesis

Erythropoiesis or formation of erythrocytes (red blood cells) occurs in red bone marrow in adults from hematopoietic stem cells (HSCs) which differentiate into committed erythocyte progenitor cells and erythropoietin cells. Then in the presence of granulocyte macrophage colony stimulating factor (GM-CSF) progenitor cells are converted into early normoblasts → late normoblasts → reticulocytes and finally red blood cells. Hemoglobin (Hb) is the main constituent of red cells which is further made of haem and globin moiety. Haem is iron protoporphyrin containing 4 pyrrole rings and one ferrous ion. Globin molecule in adult Hb is made up of 2α and 2β chains.

#### Factors Required for Erythropoiesis

1. Proteins are required for the biosynthesis of globin chains of hemoglobin. Protein energy malnutrition can lead to anaemia.
2. Hormones:
   (a) **Erythropoietin.** It is produced by kidneys (some by liver) stimulates and increases the number of committed stem cells in the bone marrow that are converted to red blood cell precursors and later to mature erythrocytes. During pregnancy human placental lactogen and progesterone stimulate the secretion of erythropoietin.
   (b) Other hormones like thyroxine and androgens also help in erythropoiesis.
3. Minerals and trace elements:
   (a) *Iron is the most important mineral required for the synthesis of Hb* and **is part of haem moiety.**
   (b) **Trace elements.** Small amount of zinc, cobalt and copper are needed for the synthesis of Hb. Zinc is also important in protein synthesis and nucleic acid metabolism.
4. **Vitamins.** The various vitamins required for erythropoiesis are as follows:
   (a) Folic acid is a coenzyme for '1 carbon' transfer involved in methylating reactions. Vitamin $B_{12}$ is an essential cofactor for conversion of homocysteine to methionine and formation of tetrahydrofolate ($FH_4$) which in turn is converted into polyglutamate and then into DNA. Hence, both folic acid and Vitamin $B_{12}$ are needed for erythropoiesis.
   (b) Vitamin C (ascorbic acid) is needed for the conversion of folic acid to folinic acid which is necessary for the synthesis of purines and pyrimidines which ultimately take part in the synthesis of nucleic acid and nucleo protein with help of vitamin $B_{12}$. It also enhances absorption of iron from the small intestine.
   (c) Pyridoxine (vitamin $B_6$) and riboflavin are also useful adjuncts in erythropoiesis, but are not essential.
   (d) Vitamin A required for cell growth and differentiation and maintenance of integrity of epithelium and immune function is also important.

## Definition

**Anaemia in pregnancy is defined as low hemoglobin concentration resulting in decrease in oxygen carrying capacity of blood.**

**WHO Definition.** *Hb concentration of less than 11 g/dL (7.45 mmol/L) and a hematocrit of less than 33% is defined as anaemia in pregnancy.*

**CDC (Centre for Disease Control, USA) Definition.** *Hb concentration of less than 11 g/dL in first and third trimesters and less than 10.5 g/dL during the second trimester of pregnancy.*

Using 11 g/dL as cut-off for definition of anaemia is probably too high for India and Federation of Obstetric and Gynecological Societies of India (FOGSI) has suggested a cut-off of 10 g/dL for India. *But practically a Hb concentration of < 11 g/dL is taken for anaemia.*

## Severity of Anaemia

| | ICMR | WHO |
|---|---|---|
| Mild | 10–10.9 | 9–10.9 |
| Moderate | 7.0–9.9 | 7.0–8.9 |
| Severe | 4.0–6.9 | < 7.0 |
| Very Severe (Decompensated) | < 4.0 | |

*ICMR- Indian council of Medical Research
*WHO- World Health Organisation

## Prevalence of Anaemia

The global prevalence of anaemia in pregnancy is 38.2%. Countries are categorised as per prevalence of anaemia as follows:

| Prevalence of anaemia (%) | Category of public health significance |
|---|---|
| ≤ 9 | no public health problem |
| 5–19.9 | mild public health problem |
| 20–39.9 | moderate public health problem |
| ≥ 40 | severe public health problem |

India falls in the category of severe health problem. As per National Family Health Survey 4 (NFHS4) (2015–16), overall incidence of anaemia in pregnancy in India was 23.1–61.4% with 23.6–61.7% in urban areas and 19.6–58.1% in rural areas.

| Classification of Anaemia | |
|---|---|
| **Acquired** | **Inherited** |
| IDA (Iron Deficiency Anaemia) | Thalassemias |
| Anaemia caused by acute blood loss | Sickle cell hemoglobinopathies |
| Anaemia of chronic disease | Other hemoglobinopathies |
| Megaloblastic anaemia | Hereditary hemolytic anaemia |
| Acquired hemolytic anaemia | |
| Aplastic/hypoplastic anaemia | |

## Physiological Anaemia in Pregnancy

Blood volume increases by 45%
Plasma volume increases by 55%
RBC mass increases by 30%
Erythrocyte dilution by 5–50%
There is a fall in Hb by 2g/dl
Hemoglobin (Hb) - 10.5g
Hematocrit (Hct) - 30%, RBC - 3.2 million/$mm^3$
Peripheral smear (PS) - normocytic normochromic

### Advantages

1. Decreased blood viscosity decreases load on heart.
2. There is increased placental pertusion
3. There is increased foetal oxygenation.
4. There is protective buffer against blood loss in third stage of labour.

# NUTRITIONAL ANEMIAS

## Iron Deficiency Anaemia (IDA)

Iron deficiency anaemia is the most common type of anaemia in pregnancy. Almost 50% cases are attributed to IDA. The nutritional status of iron depends on long term iron balance and is favored by ingestion of adequate amounts of iron in the diet and through iron supplementation. The various factors affecting the iron status during pregnancy are given in Table 35.1.

**Table 35.1: Factors Affecting the Iron Status in Pregnancy**

| Iron absorption | Iron loss |
|---|---|
| Dietary iron (Haem and non-haem) | 1. Physiological factors<br>2. Basal losses from desquamation from intestines and skin<br>3. Delivery<br>4. Lactation |
| **Enhancers of absorption** | |
| 1. Haem iron<br>3. Meat<br>5. Fermentation<br>7. Gastric acidity<br>9. Low iron stores<br>10. Increased erythropoietic activity (high altitude, hemolysis, bleeding) | 2. Proteins<br>4. Ascorbic acid<br>6. Ferrous iron<br>8. Alcohol |
| **Inhibitors of iron absorption** | **Pathological factors for iron loss** |
| 1. Phytates<br>2. Calcium<br>3. Tannins<br>4. Tea and coffee<br>5. Herbal drinks<br>6. Fortified iron supplements | 1. Hookworm and other helminths infestation<br>2. Haemorrhage from gastrointestinal tract<br>3. Allergies<br>4. Occult blood losses |

In developing countries like India a low bio-availability diet is usually consumed which is a simple routine diet of cereals (wheat, rice, maize), roots or tubers with negligible

amount of meat, fish and ascorbic acid. Such a diet contains phytates, which inhibit iron absorption to as low as 3–4%. In contrast, high bio-availability diet rich in haem iron like meat, fish and poultry is consumed in developed nations with high amount of ascorbic acid for better iron absorption making anaemia uncommon in these countries.

## Iron Requirement during Pregnancy

Iron requirement during pregnancy varies with maternal weight and size and maturity of fetus and is given in Chapter 6. About 800–1000 mg extra iron is required in pregnancy.

Average iron requirement is 6 mg/day throughout pregnancy varying from 2.5 mg/day in early pregnancy, 4–6 mg/day in mid pregnancy to 10 mg/day from 32 weeks onwards.

The absorption of iron is less than 10% which requires at least 40–60 mg of iron in the diet to achieve 4–6 mg of absorption. This is not available in the average Indian diet necessitating supplement of 100 mg iron and 0.5 mg folic acid per day during pregnancy as recommended by Government of India's National Nutrition Anaemia Prophylaxis Programme (see Chapter 45 for details).

## Causes of High Prevalence of Iron Deficiency Anaemia (IDA) in India

The causes of high prevalence of iron deficiency are given in Table 35.2.

**Table 35.2: Causes of High Prevalence of Iron Deficiency Anaemia in Pregnancy**

| | |
|---|---|
| **Dietary habits** | 1. Consumption of low bio-availability diets like maize, rice, beans, wheat with negligible meat, fish, poultry and ascorbic acid<br>2. Food fadism (avoidance of consumption of specific foods in pregnancy)<br>3. Pica: Ingestion of soil or other non-edible substances |
| **Defective absorption** | 1. Helminthiasis (hookworm), amebiasis, giardiasis, celiac disease, tropical sprue |
| **Low iron stores and iron loss** | 1. Low baseline reserves<br>2. Multiple pregnancies at short intervals<br>3. Menorrhagia<br>4. Schistosomiasis, malaria, hookworm infestation |
| **Vitamin A deficiency** | 1. Dietary deficiency |

## Effects of Anaemia on Pregnancy

Mild anaemia may not have any effect on pregnancy and labour other than lower iron stores in mother and fetus. However, moderate and severe anaemia can cause various maternal and foetal effects as shown in Table 35.3.

There is risk of death of mother with severe anaemia at 32–34 weeks of pregnancy, in labour, following delivery and in second week of puerperium due to cardiac failure or thromboembolism.

**Table 35.3: Maternal and Foetal Effects of Anaemia during Pregnancy**

| Maternal Effects |
|---|
| 1. Preterm labour (28%)<br>2. Pre-eclampsia (folate deficiency)<br>3. Inter-current infection<br>4. Cardiac failure<br>5. Poor weight gain<br>6. Uterine inertia<br>7. Maternal exhaustion<br>8. Delayed wound healing<br>9. Postpartum haemorrhage<br>10. Hemorrhagic shock<br>11. Subinvolution<br>12. Puerperal sepsis<br>13. Puerperal venous thrombosis<br>14. Pulmonary embolism<br>15. Failure of lactation |
| **Foetal Effects** |
| 1. Foetal growth restriction<br>2. Intrauterine death<br>3. Increased perinatal mortality<br>4. Iron deficiency in the fetus<br>5. Cognitive and affective dysfunction in the neonate |

## Clinical Features of Iron Deficiency Anaemia

The clinical features of iron deficiency anaemia vary depending upon the duration and severity of anaemia and gestation of pregnancy

### Symptoms

| | |
|---|---|
| Mild anaemia | Usually asymptomatic |
| Moderate anaemia | Weakness, fatigue, lassitude, exhaustion, loss of appetite, indigestion, giddiness, breathlessness |
| Severe anaemia | Palpitation, tachycardia, breathlessness, generalized edema (anasarca) |

### Signs

There may be no signs in mild anaemia.

Pallor, nail changes (depressed nails, koilonychia) cheilosis, glossitis, stomatitis, edema, hyperdynamic circulation as evidenced by a short and soft systolic murmur, signs of congestive heart failure (decompensated anaemia).

Fine crepitations at bases of lungs due to congestion.

## Diagnosis

From symptoms and signs, diagnosis of anaemia is suspected. But it is confirmed by the following investigations. However, all investigations are not required for all cases.

1. **Hemoglobin measurement.** It is the most practical method of diagnosis. Measurement of Hb colorimetrically by **cyanomethemoglobin method** is the most accurate. **Taliquist method** and **Sahli's method** have less accurate results. However, being

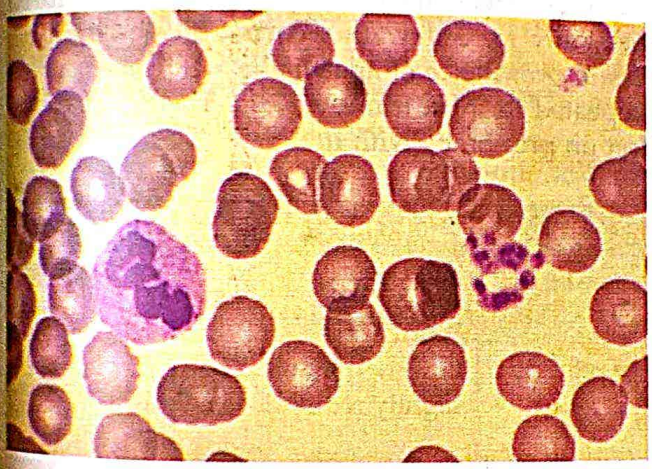
easy, Sahli's method is commonly used (see Chapter 47 for details). Grading of severity of anaemia is done using Hb levels.

2. **Peripheral blood film.** Examination of a properly prepared peripheral blood smear stained with Leishman stain is very useful in studying morphology of red blood cells and elucidating the type of anaemia. A normal peripheral blood film shows normocytic (normal sized RBCs) normochromic (normally colored RBCs) picture (Fig. 35.1 A). In iron deficiency anaemia a typical picture is of **microcytic** (smaller RBCs) **hypochromic** (pale RBCs) with variation in size **(anisocytosis)** and shape (**poikilocytosis**) with or without target cells. (Fig. 35.1 B). Photopencil cells may be seen (cells with a length of 4 times than width like a pencil).
3. **Reticulocyte count.** It may be slightly raised in iron deficiency anaemia but rises more after correction of anaemia with iron therapy. Normal levels are 2–3%.
4. **Hematocrit (normal 32–36%).** It is reduced in iron deficiency anaemia (< 30%)
5. **Blood indices.** They are mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) and are reduced in iron deficiency anaemia. Mentzer's index = MCV × 100/RBC count. It is > 13 in IDA and is < 13 in thalassemia. RDW (Red cell distribution width) is > 14 in IDA and < 14 in thalassemia. Being based on red cell count (inherently inaccurate unless counted by electronic counter), MCH and MCV are not very reliable. However, **MCHC being independent of RBC count is the most sensitive index of iron deficiency anaemia**. As blood indices can also be decreased in thalassemia, the two conditions need to be differentiated. The measurement, normal range and variation in iron deficiency anaemia, thalassemia, anaemia of chronic inflammation and sideroblastic anaemia are shown in Table 35.4A.

An iron deficiency anaemia picture shows the following blood values:

Hemoglobin—less than 11 g%, Red blood cells—less than 4 million/mm$^3$, PCV—less than 30%, MCV—less than 75 f1, MCH—less than 25 pg and MCHC—less than 30%.

6. **Serum iron** is usually below 30 µg/dL in iron deficiency anaemia (normal 60–120 µg/dL).
7. **Total iron binding** capacity is elevated to beyond 400 µg/dL (normal 300–350 µg/dL in non-pregnant state, 300–400 µg/dL in pregnancy).
8. **Serum ferritin.** It is a high molecular weight glycoprotein circulating in concentration of 15–300 µg/L in plasma and gives better picture of stored iron. It is best single indicator of iron deficiency. Level < 30 µg/L slow IDA with 90% sensitivity and 85% specificity. It is indicator of iron stores. **It is unaffected by recent iron intake and is the first abnormal laboratory test in iron deficiency anaemia.**
9. **Serum hepcidin levels.** This peptide is a major regulator of iron availability. Its levels are decreased normally in pregnancy. They are decreased further in IDA like ferritin. Normal median hepcidin levels are 16 ng/ml in first trimester, 11 ng/ml in second trimester, 9.5 ng/ml in third trimester but are < 2.0 ng/ml in IDA in pregnancy.
10. **Transferrin saturation** is 10% or less. **It is the second parameter to be affected in iron deficiency anaemia.**
11. **Free erythrocyte protoporphyrin (FEP) is the third estimation of iron status taking 2–3 weeks to become abnormal after depletion of iron stores.** Estimation of Hb, ferritin, transferrin saturation and FEP help allocating women to one of the following categories:
    (a) **Reduced stores.** Decreased ferritin only
    (b) **Iron deficiency without anaemia.** Decreased ferritin, decreased transferrin saturation and increased FEP
    (c) **Anaemia with reduced iron.** Decreased Hb, red cell indices, ferritin and transferrin saturation, increased TIBC and FEP
12. **Serum transferrin receptor** (STFR) appears to be a specific and sensitive marker and best indicator of iron deficiency in pregnancy (normal 4–9 µg/L). Its levels are increased in iron deficiency anaemia. It is being used more frequently now.
13. **STFR-Ferritin Ratio.** It indicates body iron stores Ratio



(A)




(B)

**Fig. 35.1:** (A) Normal peripheral blood film showing normocytic normochromic red blood cells; (B) Peripheral blood film showing microcytic hypochromic picture due to iron deficiency anaemia with anisocytosis (variation in size of cells) and poikilocytosis (variation in shape of cells)

increases from < 500 (adequate stores) to over 2000 during significant iron depletion.

14. Serum erythropoietin levels: They are less than 75.2 U/L (or mU/ml). They are increased in iron deficiency anaemia.
15. **Bone marrow examination** by staining with potassium ferrocyanate to see characteristic blue granules of stainable iron in erythroblasts is the most accurate method for iron stores, but is not practical in most cases being invasive. Indications of bone marrow examination are as follows:
    (a) No response to iron therapy after 4 weeks
    (b) Diagnosis of Kala azar
    (c) Suspected hypoplastic or aplastic anaemia
    (d) Myeloproliferative disorder
16. As worm infestations are common causes of anaemia, stool examination for ova and cysts should be done consecutively for 3 days in all cases.
17. In areas where schistosomiasis is prevalent, urine examination for occult blood and schistosomes should be performed.
18. As malaria is an important cause of anaemia, peripheral blood film should be examined for malarial parasites in all the cases.
19. Significant bacteriuria should also be ruled out.
20. Other tests are done in selected cases only such as sputum examination and chest X-ray for pulmonary tuberculosis (abdominal shielding should be done), renal function tests in suspected renal disease and serum proteins in hypoproteinemia.

## Prevention of Iron Deficiency Anaemia

The management starts from childhood. Prevent iron deficiency anaemia in adolescent females. Prenatal check-up is necessary to cure anaemia before trying for pregnancy.

1. Dietary modifications:
    (a) Consumption of iron rich foods such as jaggery, green leafy vegetables (like spinach, mustard leaves, green turnip), cereals, sprouted pulses.
    (b) Cooking food in iron utensils.
    (c) Avoidance of excessive tea and coffee intake.
    (d) Avoiding overcooking of food to prevent loss of folate and other vitamins.
2. Iron and folate supplementation of adolescent and non-pregnant women of child bearing age as a public health approach. **Twelve by twelve initiative** is one such initiative aiming to have Hb of 12 g/dL by 12 years of age using prophylactic iron therapy and advising consumption of iron rich foods (see Chapter 45 for details).
3. **Deworming:** It is done using single dose of albendazole (400 mg) or mebendazole 500 mg single dose or 100 mg twice daily for 3 days in second trimester in endemic areas. If prevalence of anaemia is > 50%, then the dose is repeated in third trimester also. There is no adverse effect on pregnancy with it.

**Table 35.4A: Blood Indices in Iron Deficiency Anaemia, Thalassemia and Other Anemias**

| Investigations | Measurement | Normal values | Iron deficiency | Thalassemia | Anaemia of chronic inflammation | Sideroblastic anaemia |
|---|---|---|---|---|---|---|
| 1. MCV (fl) | PCV/RBC | 75–96 | Reduced | Very reduced | Low, normal or slightly reduced | Very low in congenital; Raised in acquired |
| 2. MCH (pg) | Hb/RBC | 27–33 | Reduced | Very reduced | Low, normal or slightly reduced | Very low in congenital; Raised in acquired |
| 3. MCHC (g/dL) (%) | Hb/PCV | 32–35 | Reduced | Normal or slightly reduced | Low, normal or slightly reduced | Very low in congenital; Raised in acquired |
| 4. HbF (%) | HbF/HbA × 100 | < 2% | Normal | Raised | Normal | Normal |
| 5. $HbA_2$ (%) | $HbA_2$/HbA × 100 | 2–3% | Normal or reduced | Raised | Normal | Normal |
| 6. FEP (µg/dL) | | < 35 | > 50 High | Normal | Normal | Normal |
| 7. Bone iron stores | | | Reduced | Normal | Present | Present |
| 8. Serum iron µg/dL | | 60–120 | Reduced | Normal | Reduced | Raised |
| 9. TIBC µg/dL | | 300–350 | Raised | Normal | Reduced | Normal |
| 10. Serum ferritin µg/L | | 15–300 | Reduced | Normal | Normal | Raised |

HbF: Foetal Hb; $HbA_2$: Hemoglobin $A_2$; FEP: Free erythrocyte protoporphyrin

4. Malaria prophylaxis is given in endemic areas using 2 doses of sulfadoxine and pyrimethamine as it also causes anaemia.
5. Iron supplementation in pregnant women.
   (a) **WHO recommendation.** Universal iron supplementation should be given to all pregnant women with 60 mg elemental iron and 400 µg folic acid once daily for 6 months in countries with a prevalence of anaemia < 40% and for additional 3 months postpartum with a prevalence > 40%.
   (b) Ministry of Health, Government of India recommendation is to give 100 mg of elemental iron with 0.5 mg of folic acid in the second half of pregnancy for a period of 100 days (for prevention) to all pregnant women.
   (c) Routine supplementation is considered cost effective in developing countries like India as serum ferritin screening is costly and most women have depleted iron stores who are likely to benefit from it with little problem of overload.
6. **Food fortification.** Fortification of cereals, sugar, curry powder with ferrous salts or chelated iron compounds has been tried. Fortification of common salt with iron has also been successfully done. Double fortified salt contains 1000 parts per million (PPM) of iron with 35 PPM of iodine and 96.5% sodium chloride (NaCl). It provides 100% of dietary iodine and 30–60% of diatary iron daily.
7. Antenatal care for early recognition and prompt treatment of deficiency.
8. Intermittent supplementation (weekly and bi-weekly supplementation) of oral iron has also been found to be effective with lesser side effects and more compliance.

## Treatment

The aim of treating such women is to raise the Hb levels to near normal followed by restoration of iron stores before she goes into labour. The route of administration of iron depends upon the severity of anaemia, duration of pregnancy and any other factors.

### Oral Iron Therapy (Table 35.4B)

Oral iron therapy is the route of choice. As iron is absorbed in ferrous form, salts containing ferrous form of iron e.g. ferrous sulphate (available as fersolate), ferrous gluconate, ferrous fumarate or ferrous succinate should be prescribed. Government of India, Ministry of Health recommends 200 mg elemental iron with 1 mg folic acid per day for treatment until Hb levels come back to normal followed by 100 mg per day as maintenance therapy until 3 months after delivery to replenish the stores. There is no scientific evidence of superiority of one preparation over other and the iron available free of cost in hospital should be prescribed for better compliance. Slow release preparations claimed to have lesser side effects are probably not optimally absorbed.

**Table 35.4B: Oral Iron preparations**

| Preparations | Elemental Iron | Advantages |
|---|---|---|
| Ferrous Sulfate | 65 mg/tablet | - Best efficacy<br>- Has highest gastrointestinal side-effects like vomiting, constipation, diarrhoea |
| Ferrous Fumarate | 50 mg/tablet or 100 mg/capsule | - Best efficacy<br>- Has high gastrointestinal side-effects like vomiting, constipation, diarrhoea |
| Ferrous ascorbate | 100 mg/tablet | - Ascorbic converts ferric to ferrous for better absorption and lesser GI side-effects |
| Iron polymaltose complex | 100 mg/tablet | - Non-ionic iron in a stable complex<br>- Lesser GI side-effects<br>- Absorption not affected by milk or food |
| Ferrous Bisglycinate | 60 mg/tablet or 30 mg/ tablet | - Lesser dose needed and hence lesser side effects |
| Carbonyl Iron | 100 mg/tablet | - Best bioavailability and absorption<br>- Least GI side-effects<br>- Pentacarbonyl iron is reduced to very fine microspheres |

**Advantages**

1. Cheap
2. Fasy to administer
3. No anaphylaxis

**Disadvantage**

1. Poor absorption
2. GI side effects are common and are as follows
   (*i*) Constipation (*ii*) Vomiting
   (*iii*) Epigastric pain (*iv*) Heartburn
   (*v*) Metallic taste (*vi*) Staining of teeth
3. Poor compliance

In case of side effects and non compliance carbonyl iron, ferrous bisglycinate or ferrous ascorbate can be prescribed.

1. ***Indicators of response to therapy***
   (a) Feeling of well-being
   (b) Improved look of the patient.
   (c) Better appetite
   (d) Hematological examination: (*i*) Rise in hemoglobin level (*ii*) Reticulocytosis (0.5% rise) within 7–10 days. (*iii*) Red cell indices returning to normal levels. If no significant improvement occurs clinically and hematologically within three weeks, diagnostic re-evaluation should be done.

2. **Rate of improvement.** The improvement should be evident within three weeks of the therapy. After a few days, the hemoglobin concentration should rise at the rate of 0.3 g to 1.0 g (average 0.7 g/dL) per week.
3. **Reasons for failure to respond to oral therapy** are:
   (a) Inaccurate diagnosis (non-iron deficiency microcytic anaemia like thalassemia, pyridoxine deficiency, lead poisoning)
   (b) Faulty absorption due to associated gastrointestinal disorders
   (c) Non compliance
   (d) Continuous blood loss as in hookworm infestation or bleeding hemorrhoids.
   (e) Inhibition of erythropoiesis by coexisting infection.
   (f) Concomitant folate deficiency.
4. Contraindications to oral iron therapy are:
   (a) Intolerance to oral iron
   (b) Severe anaemia in advanced pregnancy.

## Parenteral Iron Therapy

Parenteral iron therapy is only indicated when the pregnant woman is unable to take oral iron due to side effects or is non compliant. It has the advantage of certainty of its administration. They provide a greater and more rapid iron supply that oral iron therapy without the gastrointestinal side effects of oral substitution and make it possible to avoid blood transfusion with associated risks.

### Indications of Parenteral Iron Therapy

1. Poor tolerance to oral therapy
2. Poor absorption of iron like in chronic diarrhea, ulcerature colitis, celiac disease or inflammatory bowel disease.
3. Non-compliance with oral iron therapy
4. Oral iron not effective
5. Women in third trimester or near term with moderate to severe aemia.
6. Presence of concurrent disease like chronic renal failure when patient is on hemodialysis or being treated with erythropoetin.

### Contraindications

1. Anaemia not due to iron deficiency anaemia (IDA)
2. Iron overload
3. Hypersensitivity to parenteral iron
4. Active infection
5. Liver cirrhosis
6. Acute renal failure
7. First trimester

Calculation of dose of parentral iron (mg) can be done using different formulae.

1. Ganzoni's formula; hemoglobin deficit × weight (kg) × 2.4 + 1000 (for stores)
2. 250 mg × Deficit of Hb %
3. 0.23 × body weight (kg) × (150-patient Hb in g/L) + 500
4. 0.3 × body weight in pounds × hemogobin deficit + 500 mg.

### Iron Preparations

All currently available intravenous iron agents are colloids that consist of spheroidal iron carbohydrate nanoparticles. At the core of each particle is an iron oxyhydronide gel which is surrounded by a shell of carbohydrate. The carbohydrate shell stabilizes the core, slows the release of bioactive iron and maintains the resetting particles is colloidal suspension. They are classified as in Table 35.4C.

Pre-requisites prior to giving parenteral iron therapy:

1. Confirm the diagnosis of iron deficiency anaemia by measuring serum ferritin < 15 ng/ml
2. Stop oral iron therapy 48 hrs prior to administration of parenteral therapy to avoid toxic reactions.
3. Parenteral iron should be given in a health facility in the presence of Hydrocortisone and Adrenaline in the emergency tray.
4. Sensitivity testing to be done prior to preparations where test dose is needed.

**Intravenous Iron:** Two preparations are now used. Oral Iron is stopped 48 hour before parenteral therapy.

1. **Injection Iron sucrose:** It can be administered as IV infusion by diluting 300 mg in 300 ml normal saline over 30 minutes. Total iron requirement is calculated using Ganzoni's formula and 300 mg is given bi-weekly

**Table 35.4C: Classification of Parenteral iron preparations**

| Characteristics | Type I | Type II | Type III | Type IV |
|---|---|---|---|---|
| Strength | Robust, strong | Semi-robust, moderately strong | Labile weak | Mixtures containing at least two different iron complexes |
| Molecular weight (Daltons) | > 1,00,000 | 30,000–1,00,000 | < 50,000 | < 50,000 |
| Examples | (i) Ferric Carboxymaltose intravenous<br>(ii) Iron dextran (Intra muscular or intravenous) | (i) Iron sucrose (Intravenous) | (i) Sodium ferric gluconate<br>(ii) Iron (III) Citrate<br>(iii) Iron (III) sorbitol (intramuscular) | (i) Iron (III) citrate + Iron (III) sorbitol + Iron dextrin<br>(ii) Sodium ferric gluconate + iron sucrose |

(maximum 600 mg per week). It has negligible risk of reaction and does not require test dose. However, it is advisable to give first dose in ward or labour room keeping antihistaminics and adrenaline injection ready for any reaction. Later doses can be given in out door. It is drug of choice in current practice. It is expensive but less than carboxy maltose.

2. **Ferric Carboxy Maltose (FCM).** Macromolecular iron - hydroxide complex of polynuclear (III) hydroxide in carbohydrate shell. **Dose:** 15 mg/kg/day, Maximum - 1000 mg/setting. Repeat dose on day 7 and 14. Not to exceed > 2500 mg.

Although category C drug, it has now been recommended and allowed by Drug Controller of India for use in pregnancy if required. Test dose not required. It was previously given in puerperium. It is diluted in 250 ml normal saline and is given in I.V. infusion over 30 minutes.

At AIIMS Delhi, we are routinely using it now in pregnancy. It is also being used for women who are unable to tolerate oral iron.

#### Advantage

1. Less no. of visits
2. Better compliance
3. Less adverse events
4. It can be given rapidly

#### Disadvantages

Costly (About ₹ 3000 per 500 mg)

**Intramuscular iron:** These are rarely used in current practice due to risk of side-effects. They are cheap. They are iron dextran and Iron sorbitol citrate. Dose is 100 mg/day till total dose. Maximum dose 200 mg/day can be given.

Iron dextran can be given both as IV or IM. When giving IV, 0.5 ml test dose is given diluted in 50 ml of Normal saline over 10-15 mins. After observing for a period of 30-40 mins for no reaction, remaining dose from the 2 ml vial (100mg) is given either undiluted slow IV or as infusion in 250 ml saline over 2-6 hrs.

Z shaped deep intramuscular injections are given to avoid staining.

#### Disadvantages of Intramuscular Iron

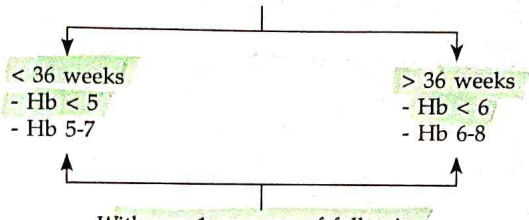
Pain, skin discoloration, abscess formation, reaction in the form of nausea, vomiting, headache, fever, lymphadenopathy, allergic reactions and rarely anaphylaxis. However, there is no extra risk of incurring malignancy at the injection site in humans as was reported in rats.

#### Role of Erythropoietin

Alongwith parenteral iron (iron sucrose intravenously every alternate day on day 2, 4, 6 and so on), injection erythropoietin in dose of 50–150 U/kg body weight every 2-3 days subcutaneously (on day 1, 3 and 5) has been used in severe anaemia for significant increase in Hb and to avoid blood transfusion. It is routinely used in anaemia with renal disease. It can also be combined with ferric carboxymaltose (FCM).

### Blood Transfusion

1. Indications of Blood transfusion




(a) established or incipient cardiac failure or classical evidence of hyponia
(b) Pneumonia or any other serious bacterial infection
(c) Malaria
(d) Pre-existing heart disease, not usually related to anaemia

**Source:** WHO Obstetics Manual 2017

2. Associated infection
3. Patients not responding to oral and parenteral iron therapy.

Packed cells are preferred for transfusion to prevent volume overload. Blood transfusion should be given with extreme care as transfusion reactions, preterm labour (due to release of cytokines and leukotrienes) and overloading of heart can be precipitated.

Exchange transfusion (in which patient's own blood is removed before giving blood transfusion) is used very rarely in some centers for patients with severe anaemia to prevent overloading of heart.

## Care during Pregnancy

Women with anaemia during pregnancy should be seen more often during antenatal period to detect and manage complications of anaemia like heart failure and preterm labour at the earliest. Regular foetal monitoring for growth and well-being is also recommended.

Practical management and protocol of a case of severe anaemia presenting at different gestations is given in Table 35.5.

## Management of Labour

#### First Stage

The patient should be in a comfortable position. Sedation and pain relief should be given. Adequate cross matched blood should be arranged. Intravenous fluids should be restricted. Oxygen should be kept ready and is given for breathlessness. In preterm labour, betamimetics and steroids should be given with caution to avoid the risk of pulmonary edema. Digitalization may be required in cardiac failure due to severe anaemia. The aim is to deliver the baby vaginally. Antibiotic prophylaxis is preferred.

#### Second Stage

The second stage is the most stressful stage for the woman with severe anaemia due to risk of heart failure. A tendency for prolongation of the second stage can be curtailed by use of forceps.

#### Third Stage

Careful vigilance is required as anaemic women tolerate

**Table 35.5: Practical Management Protocol of Severe Anaemia in Pregnancy**

| Pregnancy < 30 weeks | | Pregnancy 30–36 weeks | | Pregnancy > 36 weeks |
|---|---|---|---|---|
| Iron deficiency anaemia ↓ | Folic acid deficiency ↓ | Iron deficiency ↓ | Folic acid deficiency ↓ | Blood transfusion (Parenteral iron with erythropoietin injection can also be tried ) |
| Oral iron therapy ↓ | Oral folate therapy | Parenteral iron ↓ | Oral folate therapy or intramuscular folate | |
| Intolerance or non-compliance ↓ | | Intravenous iron | | |
| Intravenous iron (Rarely intramuscular) | | | | |

any excessive bleeding poorly. Active management of the third stage should be done except in very severe anaemia for fear of heart failure. Any postpartum haemorrhage must be energetically treated.

### Puerperium

The mother should have adequate rest, iron and folate therapy which should be continued for at least 3 months. Any infection must be immediately treated. Puerperal sepsis, failing lactation, subinvolution of uterus and thromboembolism are more common in these patients and should be taken care of. Maternal mortality in severe anaemia can occur in the last trimester, during labour, immediately after delivery and during puerperium due to cardiac failure or pulmonary embolism. The anaemic patient must use an effective method of contraception and should not conceive for at least 2 years giving time for iron stores to recover. Sterilization is preferred if the family is complete. If there is no history of menorrhagia, an intrauterine device can be inserted. Levonorgestrel intrauterine system can be used even in the presence of menorrhagia to give contraception and to reduce blood loss, but it is expensive. Barrier methods can be safely given, but their higher failure rate is a disadvantage.

The baby should be attended by a pediatrician being more likely to be preterm, small for gestational age and more susceptible to infection and anaemia. There is increased perinatal mortality and morbidity and low iron stores in such babies.

## MEGALOBLASTIC ANEMIAS DURING PREGNANCY

In megaloblastic anaemia, DNA replication is affected due to derangement of red cell maturation with production of abnormal precursors known as megaloblasts which can be due to deficiency of folate or Vitamin $B_{12}$. Folate deficiency appears earlier.

### Folate Deficiency Megaloblastic Anaemia

Folic acid together with iron assumes a central role in nutrition during pregnancy. It is reduced first to dihydrofolic acid and then to tetrahydrofolic acid (folinic acid) which is required for cell growth and division. Its requirements are increased in pregnancy to meet the needs of the fetus, the placenta, uterine hypertrophy and expanded maternal red cell mass.

**Incidence** varies from 0.5–3% being more common (up to 5 times) in multiparas and with multiple pregnancy (up to 8 times).

#### Causes

1. **Dietary deficiency.** Food lacking in green vegetables, fruits, liver, kidney with prolonged cooking of foods
2. Hyperemesis gravidarum
3. **Malabsorption syndromes.** Tropical sprue, celiac disease
4. **Drugs.** Anti-epileptic drugs (e.g. Phenytoin, pyrimethamine and trimethoprim) can cause folate deficiency.
5. **Excess utilization:**
   (a) Physiological—Pregnancy and lactation
   (b) Pathological—Hemolytic anemias, malignancies
6. Inflammatory conditions like tuberculosis, Crohn's disease, rheumatoid arthritis, psoriasis, exfoliative dermatitis
7. Hemorrhagic states like peptic ulcer, hookworm infestation, hemorrhoids
8. Hemolytic states such as chronic malaria, sickle cell anaemia and thalassemias lead to increased erythropoiesis.
9. Excess urine folate loss, active liver disease, congestive heart failure
10. **Iron deficiency anaemia.** It not only conceals morphological evidence of megaloblastic anaemia but treatment with iron would lead to a hyperplastic marrow increasing the need for folic acid and thus precipitating its deficiency producing ineffective erythropoiesis.

#### Clinical Features

1. May be asymptomatic
2. Loss of appetite
3. Vomiting, diarrhoea, constitutional symptoms, like unexplained fever

4. On examination, varying degrees of pallor with glossitis, hemorrhagic patches under the skin and conjunctiva, hepatosplenomegaly and polyneuropathy may be observed,

### Effects on Pregnancy

Increased incidence of abortion, growth restriction, abruptio placentae, pre-eclampsia, etc., have been reported in some but not all studies.

### Effects on Fetus

Neural tube defects can be prevented in most (but not all) cases by periconceptional folic acid in dose of 400 µg/day in low-risk cases, 1.5 mg/day in diabetic pregnancies and 4–5 mg/day in high-risk cases. There is an increased incidence of abortion, premature babies, small for date babies and neonatal folate deficiency in neonates born to mothers with folate deficiency.

### Investigations

1. Fall in Hb concentration to < 11 g/dL.
2. MCV > 96 fl, MCH > 33 pg and normal MCHC.
3. **Peripheral blood film.** There is macrocytic (megaloblastic) anaemia with normochromia with hypersegmentation of neutrophils, neutropenia and thrombocytopenia on peripheral blood film (Fig. 35.2 A).
4. Anisocytosis
5. A combination of low serum folate (< 3 ng/mL) and red cell folate (< 150 ng/mL) is diagnostic of folic acid deficiency.
6. Serum iron is usually normal or high.
7. Increased formiminoglutamic acid (FIGLU) in urine following a loading dose of histidine is found in folate deficiency. But the test is rarely done these days.
8. Increased serum lactic dehydrogenase (LDH) levels.
9. Homocysteine levels are elevated in folate deficiency.
10. The deoxyuridine suppression test helps in differentiating between folate and Vitamin $B_{12}$ deficiency.
11. Bone marrow will show a megaloblastic picture, but is rarely required.

### Prophylaxis

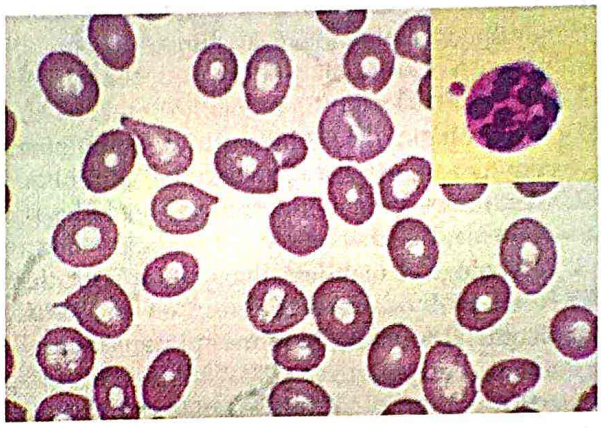
The WHO recommends a daily folate intake of 800 µg in the antenatal period and 600 µg during lactation. However, 300–500 µg present in most iron preparations is enough for prophylaxis. Pregnant women should eat more green vegetables (e.g. spinach and broccoli) and offal (e.g. liver and kidneys). Folate is destroyed by cooking. Even food fortification with folic acid (e.g. wheat floor) is recommended and is already in use in Western countries.

### Treatment

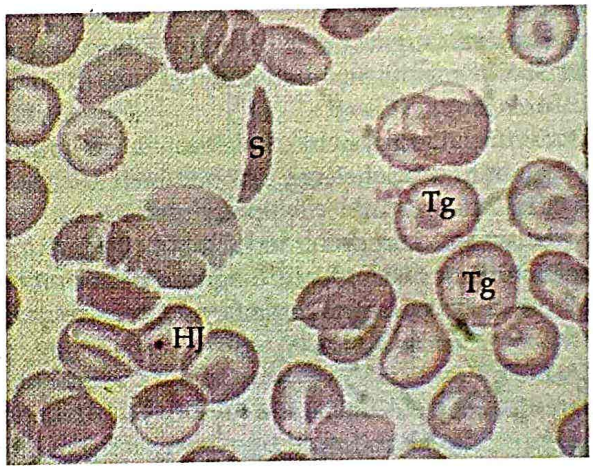
Treatment of established folic acid deficiency is by giving 5 mg oral folate per day which should be continued for at least 4 weeks in puerperium. Response is indicated by a fall in LDH levels within 3–4 days and an increase in reticulocyte count in 5–8 days. Parenteral folate is only indicated in gastric intolerance or for severe difficiency late in pregnancy. Injection containing 15 mg folic acid and 500 µg cyanocobalamin ($B_{12}$) per mL is available and is given as 1 mL IM daily or alternate days for 7–10 injections. Vitamin C is helpful. Associated iron deficiency should be corrected by iron therapy. Blood transfusion is rarely required in severe anaemia.

## Megaloblastic Anaemia due to Vitamin $B_{12}$ (Cyanocobalamin) Deficiency

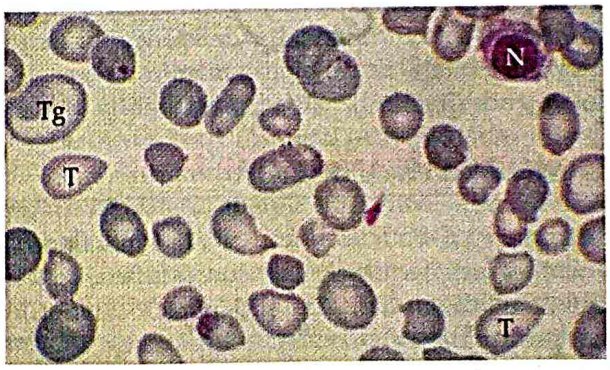
Vitamin $B_{12}$ is synthesized by certain microorganisms. Animals and humans are ultimately dependent on this source. It is found in meat, fish, eggs and milk but not in plants and is not destroyed by cooking. The average daily diet contains 5–30 µg of Vitamin $B_{12}$ of which 1–5 µg is absorbed.



(A)




(B)




(C)

**Fig. 35.2:** (A) Peripheral blood film showing megaloblastic picture in folate deficiency anaemia with macrocytosis, anisocytosis and poikilocytosis with hypersegmented neutrophils; (B) Peripheral blood film showing sickle-cell anaemia with anisocytosis, poikilocytosis, target cells (Tg) and sickle cells (S) and Howell Jolly (HJ) bodies; (C) Peripheral blood film showing thalassemia major with anisocytosis, poikilocytosis, tear drop cells (t), target cells (TG) and nucleated red cells (N).

### Causes

1. Inadequate diet – Strict vegetarians (vegans)
2. Malabsorption
   (a) Defective release of cobalamin from food (gastric achlorhydria, partial gastrectomy)
   (b) Inadequate production of intrinsic factor (pernicious anaemia, total gastrectomy)
   (c) Disorders of terminal ileum (tropical sprue, non tropical sprue, regional enteritis, intestinal reaction)
   (d) Fish tapeworm disease (*Diphyllobothrium latum*)
   (e) Blind loop syndrome

### Clinical Features

1. Hematologic manifestations are a result of anaemia and sometimes purpura results due to thrombocytopenia.
2. Gastrointestinal manifestations include sore tongue, anorexia, diarrhoea
3. Neurological manifestations begin with demyelination followed by axonal degeneration and finally neuronal death. There is numbness, paresthesia, weakness, ataxia, etc.

### Investigations

1. Findings are the same as in folate deficiency. Vitamin $B_{12}$ levels are lower in blood (< 90 µg/l).
2. Serum methyl malonic acid is elevated in Vitamin $B_{12}$ deficiency.
3. Serum homocysteine is elevated in both folate and Vitamin $B_{12}$ deficiency.
4. The deoxyuridine suppression test can differentiate between Vitamin $B_{12}$ and folate deficiency.
5. **Schilling test** used to diagnose pernicious anaemia is not done during pregnancy as it uses contrast.

### Treatment

Parenteral cyanocobalamin (hydroxycobalamine) 1000 µg daily × 1 week followed by weekly for 1 month followed by monthly injections for maintenance therapy (250 µg) are given intramuscularly every month. Treating megaloblastic anaemia due to Vitamin $B_{12}$ deficiency with folate alone may precipitate neurological manifestations. Hence, both folate and Vitamin $B_{12}$ should be given.

## DIMORPHIC ANAEMIA

Deficiency of both iron and folate with findings of both anemias but dominance of one is often seen in tropical countries. Blood film may show macrocytic or normocytic, normochromic or hypochromic pictures. Bone marrow is usually megaloblastic. Treatment is prescription of both folic acid and iron in therapeutic doses. Pregnant women should eat more green vegetables and fruits and should avoid over cooking of food.

## ANAEMIA FROM ACUTE BLOOD LOSS

Severe and acute blood loss in early pregnancy (abortion, ectopic pregnancy and hydatidiform mole) or in late pregnancy (antepartum haemorrhage due to placenta previa or abruptio placentae) can cause severe anaemia. It usually necessitates blood transfusion but can be managed by iron therapy if Hb is > 7 g/dL in absence of ongoing blood loss.

## HEMOLYTIC ANEMIAS

Accelerated red cell destruction stimulated by congenital red cell abnormality or by antibodies directed against red cell membrane proteins.

### Clinical feature

1. Anaemia
2. Jaundice
3. Splenomegaly
4. Pigment gall stones

### Types

It can be classified in various ways as follows:

I. Primary hemolytic anaemia: Various types are
   1. Sickle cell disease
   2. Hereditary spherocytosis

II. Secondary hemolytic anaemia
   1. Lupus erythematosis
   2. Pre-eclampsia syndrome

It can also be classified as:

I. Intrinsic defects:
   1. Hemoglobinopathy
   2. Red cell membrane defect
   3. Red cell enzymes defect

II. Extrinsic defects:
   1. Autoimmune hemolytic anaemia
   2. Paroxysmal nocturnal hemoglobinuria (PNH)
   3. Disseminated intravascular coagulation (DIC)
   4. Thrombotic thrombocytopenic purpura (TTP)
   5. HELLP syndrome
   6. Acute fatty liver of pregnancy

### Hereditary spherocytosis

It is the most common inherited hemolytic anaemia and can be autosomal dominant or recessive. It is due to membrane defects of spectrin, ankyrin or protein 4, 2, moderate band 3. HB: 6–8 g% in 10% patients.

Diagnosis is by increased osmotic fragility and presence of spherocytes in peripheral smear. Folic acid supplementation (4–5 mg per day) is given.

### Complications

Increased foetal loss in first trimester, anaemia, splenomegaly, jaundice, gall stones.

Aplastic crisis (parvovirus induced) can occur.

Splenectomy is indicated in severe cases followed by vaccination and prophylactic pencillin and pneumococcal vaccine.

### Erythrocyte enzyme deficiencies

1. P K deficiency (pyruvate kinase): It is usually autosomal recessive. It is non-spherocytic hemolytic

anaemia which can worsen in pregnancy. There may be PK deficiency in fetus causing nonimmune hydropse and cardiac failure in fetus.

2. G6PD deficiency: It is X-linked recessive disease. It causes periodic non-spherocytic hemolytic anaemia. Precipitating factors are vitamin C, nitrofurantoin, chlorquine, vitamin K

#### Treatment

It is self limiting. Aggressive hydration and folic acid supplementation is given.

### Acquired Hemolytic Anaemia

It can be autoimmune caused by auto antibodies (primary or secondary to malignancies, leukemias or chronic inflammatory diseases) or by infection. There is marked acceleration of hemolytic process during pregnancy which responds to steroid therapy. Diagnosis is by positive direct and indirect Coomb's test and presence of spherocytosis and reticulocytosis on peripheral smear.

#### Drug induced hemolytic anaemia

- Toxic immune complex - Quinine, quinidine, rifampicin, INH, Sulfonamides
- Hapten formation: Methicillin, ampicillin
- Congenital erythrolytic enzymatic defect - G6PD deficiency
- Mild to moderate hemolysis

#### Treatment

Withdrawal of offending drug
Glucocorticoids

#### Paroxysmal Nocturnal Hemoglobinuria

Paroxysmal nocturnal hemoglobinuria is a serious disease in pregnancy with many women developing deep vein thrombosis, cerebral vein thrombosis or Budd Chiari syndrome, especially in puerperium. Close supervision of the patient with adequate hydration is required. Folic acid supplementation and steroids are given Anti-coagulation may be needed.

#### Other Acquired Anemias

HELLP syndrome is characterized by hemolysis, elevated liver enzymes and low platelets and is a complication of severe pre-eclampsia and eclampsia (see Chapter 32 for details).

Other acquired hemolytic anemias are due to severe infection with Gram positive bacteria (exotoxin of *Clostridium perfringes*) and Gram negative septicemia.

## HEMOGLOBINOPATHIES

### Congenital Hemolytic Anemias

Hemoglobinopathies are inherited specific disorders of hemoglobin synthesis (thalassemia) or structure (sickle-cell syndrome) with significant mortality and morbidity throughout the world but are more common in certain ethnic populations like Africans (sickle-cell syndrome) and Asians (thalassemia). There is abnormally in red cell membrane making it susceptible to complement mediated intravascular hemolysis.

#### Hemoglobin Types

Hb consists of a haem moiety and a globin moiety. There are two pairs of globin chains in each Hb molecule. There are three types of normal Hb such as:

***HbA (Adult Hb).*** It has got one pair of alpha ($\alpha_2$) chains (141 amino acids) and one pair of beta ($\beta_2$) chains (146 amino acids) and constitute 97–98% Hb in a normal adult.

***$HbA_2$.*** It is made up of one pair of alpha ($\alpha_2$) chains and one pair of delta ($\delta_2$) chains (differs from β chains by 10 amino acids) and constitutes 2–3% Hb in a normal adult.

***HbF (Foetal Hb).*** It is made up of one pair of alpha ($\alpha_2$) chains and one pair of gamma ($\gamma_2$) chains and is less than 1% in a normal adult. It is the main Hb in fetus and makes 70–80% Hb in a neonate.

Alpha chains are essential for all the three types of Hb and are under the control of two genes (one from each parent).

#### *Alpha Chain Defects*

***Barts Hb (Hb Barts).*** If all four alpha globin genes are defective with no alpha chain production. There are four gamma chains. It results into a hydropic fetus which is not compatible with life.

In alpha thal+ trait, there is one defective gene. In alpha thal trait there are two defective genes from one parent. In homozygous alpha thal+ trait, there is one defective gene from each parent. In these three conditions the patient is asymptomatic.

**HbH disease.** There are three defective genes (one from one parent and two from the other). There is moderate hemolytic anaemia in the patient.

#### Beta Chain Defects

***Beta Thalassemia Major (Cooley's Anaemia).*** In this condition, both beta globin genes are defective and no beta chains are produced. The majority of affected individuals are transfusion dependent for life with all the consequences of iron overload and high mortality.

***Beta Thalassemia Trait (Minor).*** In this condition, one beta globin gene is defective resulting in mild anaemia.

***Sickle-cell syndromes.*** In this condition, the structure of globin chains is abnormal. Sickle Hb (HbS) results from a single beta chain substitution of glutamic acid by valine because of an A for T substitution of codon 6 of the beta globin gene. The various types are as follows:

***Sickle-cell anaemia (HbSS disease).*** This is the more severe disease in which substitution of a single amino acid in the beta globin chain renders it insoluble in the deoxygenated state. This results in alteration of the shape of the red blood cell into a sickle shape (hence the name).

***Sickle cell trait (HbAS)*** is much more common than sickle cell disease and is much milder. It results from production of HbS from one parent and HbA from another parent.

Other Hb variants are HbC and HbE which are much less common.

If both the parents have sickle-cell trait, there is a 1 in 4 chance of the child inheriting both the abnormal genes and thus the sickle-cell disease. The risk increases to 1 : 2 if one partner has two abnormal genes (disease) and the other has trait.

## Sickle-Cell Hemoglobinopathies in Pregnancy

Out of many variants of sickle-cell hemoglobinopathies, homozygous sickle cell (SS) disease and sickle cell hemoglobin C (SC) disease are more common during pregnancy. The frequency of carrier of sickle-cell hemoglobinopathies are as common as 1 in 4 in West Africans, 1 in 10 in Afro-Carribeans, 1 in 100 in Indians and Pakistanis and only 1 in 1,000 in Caucasians.

### Pathophysiology

Chronic anaemia and intravascular thrombosis leading to crisis are the major complications of sickle-cell disease. Although the solubility of HbS is almost equal to that of HbA in oxygenated state, it has only 1/50 solubility in deoxygenated state resulting in aggregation to form rigid crystals. Repeated cycles of agglutination and polymerization lead on to membrane rigidity and irreversible sickle cells which are cleared by reticuloendothelial system. *This reduces average lifespan of red blood cells of sickle-cell patients to only about 17 days in contrast to 120 days of normal erythrocytes.* This results into chronic compensated anaemia with Hb ranging from 6–9 g/dL. Due to excessive red cell destruction, there is associated splenomegaly and gallstone formation.

### Diagnosis

1. Peripheral blood film shows anisocytosis poikilocytosis, Howell Jolly bodies, target cells and sickle cells (Fig. 35.2 B).
2. Sickling test
3. HPLC (High performance liquid chromatography
4. Hemoglobin (Hb) solubility test

### Sickle-Cell Crisis

Sickle-cell crisis is of the following types.

**Vaso-occlusive crisis.** In this condition, the sickle cells have altered motion due to distorted erythrocyte membranes resulting in vascular stasis and hypoxia. This leads to metabolic acidosis which further aggravates the sickling and tissue hypoxia. In addition, there is interaction of sickled erythrocytes with capillary endothelial membrane and microvascular injury. It causes a pro-thrombotic state with ischemic necrosis and infarction of various organs along with increased blood viscosity. All these events lead to painful, vaso-occlusive sickle cell crisis resulting in end organ damage.

**Hematological crisis.** Rarely aplastic crisis can happen in these patients during pregnancy with malnutrition and multiple pregnancies. It usually occurs secondary to infection and manifests as rapidly falling hematocrit with weakness and pallor leading eventually to cardiac failure. It can usually be avoided by prophylactic folic acid supplementation. Rarely splenic autoinfarction can occur.

**Acute chest syndrome.** It is a serious complication resulting from sickling in the lungs compounded by infection, infarction, pulmonary sequestration and fat embolization from bone marrow with development of restrictive chronic lung disease associated with arteriolar vasculopathy. Symptoms are fever, cough, chest pain, shortness of breath, audible crackles, bronchial breathing, lung infiltration on chest X-ray. *It is one of the most common cause of maternal mortality in sickle-cell disease.* The management includes assisted or mechanical ventilation, intravenous heparin, antibiotics and blood transfusion.

### Clinical Features of Sickle-Cell Disease

Clinical features of sickle-cell disease include:

1. Painful crisis
2. Chronic hemolytic anaemia
3. Chest syndrome
4. Higher infection rate
5. Increased risk of cerebrovascular accidents
6. Hyposplenism
7. Avascular bone necrosis

### Effects of Sickle-Cell Disease on Pregnancy

Women with sickle-cell trait are less prone to complications but need to be treated like sickle cell disease only. Various complications during pregnancy are:

1. Miscarriage
2. Infection
3. Pre-eclampsia, abruption
4. Prematurity
5. Foetal growth restriction
6. Thromboembolic events
7. Maternal morbidity
8. Stillbirth
9. Perinatal mortality

### Preconception Care

1. Screening for end organ damage (Echo-cardiography, retinal screening, ferriscan)
2. Partner status
3. Genetic counselling
4. Vaccination (Pneumococci, Meningococci, Hemophilus, influenza)
5. Hydroxyrea to be started 3 months prior to conception.

### Management During Pregnancy

Pregnancies with sickle-cell disease are high-risk pregnancies and should be managed by a team of dedicated hematologist and obstetrician with interest in such problems. Iron supplementation is avoided and given only for proven iron deficiency.

#### *First trimester*

1. Low dose aspirin 75 mg once daily
2. Low molecular weight heparin (LMWH) during hospital admission
3. Prophylactic graduated compression stockings.
4. Prenatal testing can be done using chorion villus sampling or amniocentesis for DNA of abnormal Hb.

*Second and Third trimester*

USG for foetal growth to rule out foetal growth restriction

The patient should be given folate supplementation and penicillin prophylaxis to prevent infections like pneumonia. Kidney and liver functions should be regularly checked. Hb and HbS levels need to be monitored regularly. For anaemia, they need blood transfusions. Routine blood transfusion is not recommended.

Indications for blood transfusion are: acute anaemia, acute chest syndrome, twin pregnancy, previous history of any medical or obstetric complication or for women who were on transfusion therapy before pregnancy to prevent stroke. Dehydration and exposure to cold should be avoided. *As these women are more prone to premature labour, pre-eclampsia and placental abruption, the obstetrician should be careful about them at each visit. There is a risk of missing an obstetric catastrophe in them by thinking it to be due to sickling crisis.* They should be seen every 2 weeks in first and second trimesters and weekly thereafter. At each visit, blood pressure measurement, urine analysis, full blood count, urine microscopy and culture should be performed. Foetal monitoring should be performed by serial ultrasound scans for foetal growth and well-being. In suspected foetal growth restriction, non-stress test, biophysical profile assessment including liquor volume and umbilical artery Doppler velocimetry should be performed.

### Treatment of Sickling Crisis

Treatment of sickling crisis consists of adequate oxygenation, rehydration, treating any infection (parenteral antibiotics), analgesics (parenteral opioids), correcting metabolic acidosis and stabilizing the erythrocyte volume. In unresponsive patients, exchange transfusion may be required.

Routine prophylactic transfusion is not recommended. It is given only in following indications:

1. Acute anaemia
2. Acute chest syndrome
3. Acute stroke
4. Hemoglobin (Hb) < 10 g/dl or fall over 2 g/week.

### Labour Management

1. Labour should be managed in the same way as for heart disease patients.
2. Oversedation is avoided.
3. Epidural analgesia is suited for labour and delivery. Vaginal delivery is ideal. Induction of labour at 38 weeks.
4. Oxygen saturation monitoring
5. Continuous CTG monitoring
6. Avoidance of dehydration and acidosis.
7. Compatible blood should be kept available.
8. Circulatory overload should be avoided.

### Puerperium

Puerperium is the critical time for patients of sickle-cell disease as *there is increased chance of complications of sickle-cell disease during this period.* Early ambulation, hydration, antibiotics and oxygenation are recommended. Adequate analgesia and nutrition should be provided. Breastfeeding is encouraged. The neonate is routinely screened for sickle-cell disease. Low molecular weight heparin (LMWH) for 7 days for vaginal delivery and for 6 weeks for cesarean delivery.

### Contraception Progesterone Containing Contraceptives

The women with sickle-cell disease should have small families. Intrauterine device is avoided due to risk of infection. Low dose oral contraceptive pills can be given as they are effective and reliable method of contraception. Depot medroxyprogesterone acetate (DMPA) is a good alternative. Barrier methods are suitable but have a high failure rate. Male or female sterilization is desirable after completion of family.

## Thalassemia in pregnancy

Thalassemias are common in Asian women with overall prevalence being 1 in 300 to 500. It is common in Punjabi and Sindhi population followed by Rajasthan and Gujarat.

### Thalassemia Screening

The blood picture of thalassemia trait resembles that of iron deficiency anaemia, but there is elevated $HbA_2$ (> 3.5%) in beta thalassemia trait and there is normal or near normal mean corpuscular Hb concentration (Table 35.4). An RBC count higher for a given hemoglobin concentration indicates beta-thalassemia trait. Mean corpuscular hemoglobin < 80 in pregnancy and MCH < 25 rules out beta-thalassaemia. Another screening test called **NESTROFT test (Naked eye single tube red cell osmotic fragility test)** is used in many centers in India for thalassemia trait.

### NESTROFT (Naked Eye Single Tube Red Cell Osmotic Fragility Test)

Nestroft is the test used for screening of patients for thalassemia (in hospitals) based on the fact that red cells with large surface area and volume ratio resist lysis in hypotonic saline indicating lower red cell osmotic fragility, suggesting thalassemia trait. If the tube content is clear and the marker is visible then there is no thalassemia. If it is hazy and the marker invisible it signifies thalassemia. The sensitivity is 94.4% and a negative predictive value of 97.6%. It costs about ₹ 10 per test. It is used in mass screening as in an antenatal clinic. Patients positive for this test are subjected to electrophoresis.

The other methods for diagnosis are:

1. Screening by Hb electrophoresis or Hb high performance liquid chromatography (HPLC)
2. Complete blood count (CBC)
3. **$HbA_2$ estimation.** Confirmation by Hb electrophoresis or gel electrophoresis or high performance liquid chromatography (HPLC)

$HbA_2$ is elevated to > 3.5% in thalassemia trait.

### Preconceptional Care

Aggressive chelation to be given in the preconceptional period to maintain serum ferritin between 1000-2000 ng/ml. A good glycemic control should be maintained. Hba1c

is not a reliable indicator for the same. Serum fructosamine to be maintained < 300 nmol/L. Patient should be clinically euthyroid. A cardiological assesment with routine ECG and T2 weighted cardiac MRI should be done. Liver iron concentration should be estimated using Ferriscan which should be < 7 mg/ g of dry weight. Bone density scan and vit D supplementation to optimise levels prior to pregnancy. Patients should be vaccinated for hepatitis B and splenectomised patients to be given pneumococcal and influenza vaccines.

### Prenatal Diagnosis

If mother has thalassemia trait, father's status is checked. If both have thalassemia trait, there is 1 : 4 chance of fetus having thalassemia major. Prenatal diagnosis is made by chorionic villus sampling (CVS) in the first trimester or amniocentesis or cordocentesis in the second trimester by DNA analysis using molecular techniques. If chorionic villus sampling (CVS) confirms the fetus to be having thalassemia major, termination of pregnancy is indicated after counselling the patient. If the fetus has thalassemia minor (trait), pregnancy is continued as such a baby will have normal lifespan.

### Maternal complications

1. Recurrent pregnancy loss (RPL)
2. Cardiomyopathy
3. Cardiac failure
4. Diabetes mellitis
5. Liver cirrhosis
6. Transfusion related hepatitis
7. Cholelithiasis
8. Hypothyroidism

#### *First trimester*

Penicillin prophylaxis, vaccination, prenatal testing, no iron chelaton in first trimester.

#### *Second and Third trimester*

Blood sugar monitoring, thyroid function tests, Echo cardiography at 28 weeks for cardiac status

Thromboprophylaxis

Splenectomy/platelet count > 6 lac → Asprin 75 mg/day

Splenectomy + platelet count > 6 lac → Aspirin + LMWH (low molecular weight heparin)

**Chelation therapy Indications:**

- Reduced ejection fraction/increased ventricular-atrial ratio on echocardiography
- Liver iron > 10 mg/g liver tissue

Lower dose subcutaneous desferoxamine - 20 mg/kg 4–5 day/week after 20 weeks of pregnancy

Blood transfusion - post transfusion Hb to be 12 g/dl

Labour → IV desferoxamine can be given. Continuous CTG monitoring

**Puerperium.** LMWH; no contraception contraindications

#### *Alpha thalassemia*

Alpha thalassemia is common in South-East Asia. The women with alpha thalassemia trait often become anaemic during pregnancy. They often have mild to moderate hypochromic microcytic anaemia. They require oral iron (100 mg elemental iron per day) and folate (5 mg per day) supplementation. *Parenteral iron is to be avoided.* Women with HbH disease usually have chronic hemolytic anaemia requiring 5 mg folic acid per day. Due to chronic hemolysis, they are often not iron deficient. They usually require blood transfusion to treat anaemia.

In pregnancy harbouring a fetus with Hb Barts, hydrops occurs. There is risk of severe maternal complications including early onset severe pre-eclampsia and intrapartum problems due to delivery of a large hydropic fetus and placenta including maternal soft tissue trauma and primary postpartum haemorrhage.

#### *Beta Thalassemias*

**Beta thalassemia trait (minor).** It is most common in India developing in late infancy and adult life. In this condition $HbA_2$ ($\alpha 2\ \delta 2$) is increased more than 3.5%. HbF ($\alpha_2\ \gamma_2$) is usually increased to more than 2%. There is mild anaemia with microcytic hypochromic picture which can be confused with iron deficiency anaemia so common in Asian countries and can be differentiated from iron deficiency anaemia by blood indices and level of HbA2 and HbF (normal in iron deficiency anaemia but raised in thalassemia Table 35.4). In this condition during pregnancy, there is usually pregnancy stimulated erythropoiesis with normal blood volume expansion with slightly subnormal red cell mass expansion. Peripheral blood film shows anisocytosis, poikilocytosis, tear drop cells, target cells and nucleated red cells (Fig. 35.2 C).

During pregnancy, these women should be given 5 mg folic acid per day with oral iron supplements if the ferritin is low. *Parenteral iron is contraindicated.* If the anaemia is not responsive to oral iron and folate supplementation, blood transfusion is indicated. Usually pregnancy outcome is not affected. However, twofold rise in the risk of foetal growth restriction and oligohydramnios have been reported in such women.

#### *Beta Thalassemia Major*

Beta thalassemia major is a serious disease where both the beta chains are defective. The neonate with this condition is healthy at birth, but becomes severely anaemic as the HbF falls. There is failure to thrive. Such a child needs blood transfusions to survive and there is problem of iron overloading. In the past, most children with beta thalassemia major used to die, but are now reaching reproductive age group with blood transfusions and iron chelation therapy with desferrioxamine. Such women are usually sterile with shortened life expectancy. However, with blood transfusion and iron chelation therapy, some women have had successful pregnancy outcomes under intensive maternal and foetal surveillance by an expert team of dedicated hematologist and an experienced obstetrician. They need cardiac assessment for myocardial function. Their nemia should be treated by blood transfusion. Folic acid (5 mg/

day) supplementation is required. **Iron supplementation is contraindicated.**

### Anaemia of chronic disease

*Causes*

1. Chronic renal disease
2. Parasitic and bacterial disease
3. HIV infection
4. Chronic inflammatory disease

*Treatment*

Treatment of cause like treatment of UTI, tuberculosis, HIV etc. Erythropoietin therapy is useful.

*Renal anaemia*

It is seen in renal failure and renal transplant patients. There is erythropoietin deficiency, chronic inflammation, iron alongwith folic acid and B12 deficiency.

There is decreased half life of erythrocytes.

*Treatment*

Recombinant erythropoetin when hematocrit falls to < 20%

**Dose.** 50–150 U/Kg SC every 2–3 days. It can be given along with iron sucrose.

### Aplastic anaemia

It is characterised by pancytopenia and bone marrow hypocellularity.

1. Hereditary - Fanconi's anaemia
2. Acquired - Radiation
   Viruses - Parvovirus, hepatitis, Epstein-Barr virus (EBV), Systemic lupus erythematosis (SLE)

*Diagnosis*

1. Bone marrow aspiration with cytogenetic analysis
2. Chromosome fragility tests

*Treatment*

Terminate pregnancy in first trimester after counselting.

**Second and third trimester.** Ist line - Erythropoetin and granualocyte transfusion (GM-CSF).

Thymocyte gamma globulin, high-dose steroids, cyclosporin

- Blood transfusion, maintain hematocrit to > 20%
- Platelet transfusion
- Prevent foetal infection
- Splenectomy
- Delivery: vaginal route preferred
- Bone marrow transplant after delivery increases survival rate by 50–80%

## POLYCYTHEMIA IN PREGNANCY

Excessive erythropoiesis can occur in pregnancy in the presence of chronic hypoxia due to pulmonary disorder, congenital cardiac disease or heavy cigarette smoking with chronic bronchitis with poor perinatal outcome.

Polycythemia vera is a myeloproliferative hemopoietic stem cell disease with proliferation of blood cell precursors. There are higher erythropoietin levels with increased blood viscosity and thrombotic complications and increased perinatal mortality.

## PLATELET DISORDERS

Platelets produced by megakaryocytes in bone marrow with a lifespan of 7–10 days play a critical role in normal hemostasis and thrombotic disorders. Normal platelet count 1,50,000–3,50,000/uL ($mm^3$) (150–350 x $10^9$/L) decreases in the second half of pregnancy.

#### Thrombocytopenia

Thrombocytopenia is defined as platelet count of less than 150,000/uL ($mm^3$). Incidence is 7–12% during pregnancy (second most common after anaemia).

**Severity:**

*Mild:* platelet count 100,000 to 1,50,000/µL
*Moderate:* 50,000 to 99,000/µL
*Severe:* < 50,000/µL

*Causes*

1. Defective formation in bone marrow
2. Splenomegaly causing sequestration
3. Excessive destruction
   (a) **Non immunological.** e.g. pregnancy associated thrombocytopenia, hypertensive disorders, abruptio placentae, disseminated intravascular coagulation, HELLP syndrome.
   (b) **Immune disorder.** Thrombocytopenic purpura

**Pregnancy associated (gestational) thrombocytopenia**

1. Occurs in later gestations
2. It is usually mild. (platelet count usually more than 1 L)
3. There is no thrombocytopenia outside of pregnancy
4. It resolves within 6 weeks postpartum
5. There is no foetal/neonatal thrombocytopenia

**Idiopathic (immune) thrombocytopenic purpura (ITP).** It is the most common autoimmune disorder affecting 1–3 per 1000 pregnancies and is due to increased destruction of antibody (usually IgG but can be IgM and IgA) coated platelets in the spleen and other reticuloendothelial systems. If the platelet count is more than 50,000/uL, there are usually no symptoms and no treatment is required. However, if the platelet count falls less than 50,000/uL, then purpura (capillary bleeding with bruises in skin) occurs. Maternal antibodies (IgG) may cross the placenta and cause thrombocytopenia in the fetus associated with risk of intracranial haemorrhage in the fetus especially during labour.

*Management*

Careful history taking, examination and laboratory investigations like complete blood count, peripheral blood

smear HIV and HCV testing. No treatment is required if platelet count is > 50,000/uL. Major bleeding occurs if count is < 10,000 uL. Oral corticosteroids (prednisolone in dose of 1–1.5 mg/kg body weight) are started if platelet count is < 50,000/uL with 70–90% women responding within 3 weeks. They act by inhibiting platelet antibody formation and increasing bone marrow platelets. They are the treatment of choice. Immunoglobulin therapy (I.V.I.G.) is given to patients not responding to oral steroids or with very low platelets. Upto 2 gm is given over 2–3 days Splenectomy is rarely required in pregnancy. It is very expensive. Platelet transfusions are used only in acute situation (haemorrhage or to cover delivery) in consultation with hematologist. Other drugs like azathioprine and cyclophosphamide are rarely indicated in pregnancy. **Guidelines for minimum platelet requirement**

1. In the antenatal period where no invasive procedure is planned >20,000/uL
2. For normal vaginal delivery >40,000/uL
3. Cesarean section/ instrumental delivery >50,000/uL
4. Epidural analgesia > 80,000/uL

New drug romiplostim a thrombopoetin receptor agonist can be used off label for severe and refractory ITP in pregnancy (category C drug) especially prior to delivery.

### *Labour*

The count should be increased to 40,000 for vaginal delivery, 50,000 for cesarean delivery and to 70,000 for epidural analgesia using platelet transfusion. Vaginal delivery is preferred as cesarean section does not confer any benefit. In labour, foetal blood sampling, invasive foetal monitoring and ventouse delivery are avoided because of the risk of cephalhematoma. Traumatic delivery must be avoided although a low cavity forceps can be applied by an experienced obstetrician for the management of second stage. Third stage is important. Secondary postpartum haemorrhage may be seen in them especially between day 4 and day 7 needing treatment.

**Other causes of thrombocytopenia** like pre-eclampsia, infections, drugs and antiphospholipid syndrome need specific treatment of the condition and are discussed in respective chapters.

## THROMBOPHILIAS

Thrombophilia is defined as a predisposition to thrombosis secondary to a hypercoagulable state and can be inherited or acquired.

### Causes

1. Patients with history of venous thromboembolism
2. Deficiency of important protein inhibitors
   (a) Protein C
   (b) Protein S
   (c) Anti thrombin III
   (d) Factor V Leiden
   (e) Prothrombin gene mutation
3. Hyperhomocystinemia
4. Elevated factor VII
5. Antiphospholipid syndrome

### Management

Thrombophilias can cause venous thromboembolism during pregnancy and need treatment with heparin in consultation with a hematologist in dosage of 5000 international units twice daily subcutaneously along with oral low dose aspirin (60 mg). Low molecular weight heparin can also be given in dose of 40–60 mg per day (see also Chapter 35[ix]).

### Effect on Pregnancy

There is increased risk of pregnancy complications such as:

1. Recurrent miscarriage
2. Foetal growth restriction
3. Pre-eclampsia
4. Abruptio placentae
5. Intrauterine foetal death

## INHERITED COAGULATION (BLEEDING) DISORDERS

***Hemophilias.*** They are inherited deficiency of factor VIII (Hemophilia A) or factor IX (Hemophilia B or **Christmas disease**) with genes located in the X chromosome. They are categorized according a plasma levels of factor VIII and IX as mild (5–40%), moderate (1–5%) and severe (< 1%). Genetic counseling is important though hemophilia can also occur for the first time due to mutations. Prenatal diagnosis by chorionic villus sampling in the first trimester is available.

***Management.*** Such women should be managed by an experienced hematologist and obstetrician. Blood level of factor VIII or factor IX (depending upon the type of hemophilia) should be regularly done in pregnancy. Recombinant Factor VIII or desmopressin (DDAVP, synthetic analogue of antidiuretic hormone which increases factor VIII level can be given. During labour, invasive foetal monitoring and instrumental delivery should be avoided. Vaginal delivery is desirable with cesarean section being done only for obstetric indications. In puerperium, there is a risk of secondary postpartum haemorrhage for which recombinant factor VIII or desmopressin can be given. Tranexamic acid 1 g three times daily is also useful in minimizing blood loss.

***Von Willebrand's Disease.*** Von Willebrand factor is a plasma protein causing stabilization of factor VIII and adherence of platelets to injured vessels and is the most common inherited bleeding disorder. It improves in pregnancy but can cause postpartum haemorrhage requiring desmopressin or tranexamic acid therapy.

## Key Points

1. Hematological disorders are common disorders during pregnancy. Nutritional anemias are very common in developing countries causing significant maternal and perinatal mortality and morbidity with iron deficiency anaemia (IDA) being the most common followed by folate deficiency. Anaemia is a Hb concentration of < 11 g/dL during pregnancy. High incidence of anaemia during pregnancy is due to dietary habits and worm infestations. The women may be asymptomatic but can have weakness, exhaustion and rarely cardiac failure in severe anaemia.
2. Diagnosis of iron deficiency anaemia is made by Hb estimation, peripheral blood film (microcytic hypochromic anaemia) and blood indices. Serum iron, serum TIBC and ferritin are rarely indicated for diagnosis. Prevention is by 100 mg elemental iron per day during pregnancy alongwith albendazole therapy for worm infestation.
3. Treatment is by 200 mg elemental oral iron and 5 mg folate per day. Oral iron is the treatment of choice. Parenteral iron (intramuscular or intravenous) is only given if oral iron cannot be tolerated. Blood transfusion is rarely required for severe anaemia after 36 weeks of pregnancy or for any haemorrhage (antepartum or postpartum).
4. Megaloblastic anaemia is usually caused by folate deficiency and is associated with megaloblastic picture on peripheral blood film (PBF) and low serum folate levels. Treatment is by oral 5 mg folate per day. Dimorphic anaemia is deficiency of both iron and folate requiring oral iron and folate therapy.
5. Hemoglobinopathies can be thalassemias (globin chain defects) or sickle-cell syndrome and are associated with significant maternal and perinatal morbidity and even mortality. There can be sickling crisis during pregnancy in sickle cell disease.

## Suggested Reading


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8. Malhotra M, Sharma J B, Batra S et al. Maternal and Perinatal Outcome in Varying Degrees of Anaemia. International Journal of Gynecology and Obstetrics 2002; 79:93-100.
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15. World Health Organisation: Guideline daily iron and folic acid supplementation in pregnant women, 2012. http:\\apps.who.int/iris/bitstream/10665/7770.978924150.
16. National Health Scheme (NHS) Guideline for the management of thrombocytopenia in pregnancy (GL 927) May 2020, U.K. http://www.royalberkshire.nhs.UK
17. World Health Organization WHO Obstetric Manual 2017, Geneva.


## Examination Questions

A. Define anaemia during pregnancy. Enumerate its maternal and foetal effects. How will you manage a case of iron deficiency anaemia at 34 weeks pregnancy with Hb of 8 g/dL. (Many universities)

B. Enumerate the factors required for erythropoiesis and reasons of high prevalence of anaemia during pregnancy in India. How will you manage a case of severe anaemia at 36 weeks of pregnancy? (Delhi University)

# (II) CARDIAC (HEART) DISEASE IN PREGNANCY

## CARDIOVASCULAR CHANGES DURING PREGNANCY

There are significant hemodynamic and cardiovascular changes during normal pregnancy (see Chapter 6, Fig. 6.6 and Table 6.6 for details). In summary, there is significant increase in cardiac output (30–50%), heart rate, plasma volume (40–45%) and red cell volume (15–20%) with slight reduction in blood pressure. While a normal heart can withstand these extensive hemodynamic changes, with cardiac disease the patient may not be able to withstand these changes and there is risk of cardiac failure and death during the following five times.

1. Between 6–8 weeks of pregnancy
2. Between 30–34 weeks of pregnancy
3. Second stage of labour
4. Immediately after delivery
5. Second week of puerperium (cardiac failure and thromboembolism)

**The risk of cardiac failure is increased in the following conditions.**

1. Elderly pregnant women
2. Past history of cardiac failure
3. Risk factors during current pregnancy like anaemia, infection, multiple pregnancy and hypertension
4. Lack of antenatal care
5. Left ventricular hypertrophy
6. Cardiac arrythmias
7. Valvular heart disease
8. Pulmonary hypertension
9. Cardiomyopathy

### Incidence of heart disease

The incidence of clinically significant cardiac disease in pregnancy varies from 0.1% to 4% with average being 0.8%. Although the incidence of rheumatic heart disease has significantly decreased throughout the world due to effective use of antibiotics for group B streptococci, it still continues to be the major disease during pregnancy in developing countries (90–95%). Due to advancement in cardiac surgery, an increasing number of women with congenital heart disease are reaching reproductive age group and are trying conception making it now an important cardiac lesion in pregnancy.

## DIAGNOSIS OF HEART DISEASE DURING PREGNANCY

Many physiological changes during pregnancy mimic a cardiac disease, making diagnosis of heart disease more difficult in pregnancy. It is crucial not to miss the diagnosis of heart disease but equally important is not to make the diagnosis in absence of disease. Table 35.6 gives clinical and diagnostic indicators of heart disease during pregnancy.

**Table 35.6: Clinical and Investigational Indicators of Heart Disease During Pregnancy**

| |
|---|
| **Symptoms** |
| 1. Progressive dyspnea<br>2. Orthopnoea<br>3. Chest pain<br>4. Hemoptysis<br>5. Syncope<br>6. Paroxysmal Noctural Dyspnea<br>7. Noctural cough |
| **Signs** |
| 1. Raised jugular venous pressure<br>2. Cyanosis<br>3. Clubbing of fingers<br>4. On auscultation<br>(a) Pansystolic murmur<br>(b) Late systolic murmur<br>(c) Loud systolic murmur of grade 3/6 or more<br>(d) Diastolic murmur<br>(e) Persistent arrythmias<br>(f) Persistent split second heart sound |
| **Diagnostic Tests** |
| 1. Chest X-ray (with lead apron shield over abdomen)<br>(a) Unequivocal cardiomegaly<br>(b) Enlargement of pulmonary veins<br>(c) Enhanced pulmonary vascular markings<br>2. Electrocardiography (ECG).<br>(a) Cardiac arrythmias<br>(b) Chamber enlargement or hypertrophy<br>3. Echocardiography. Non-invasive and accurate method. It detects various cardiac lesions especially structural abnormalities. |

## CLASSIFICATION OF HEART DISEASE

Heart disease in pregnancy can be classified depending upon etiology (Table 35.7), functional cardiac capacity (New York Heart Association [Table 35.8]) and on risk factors for maternal mortality and morbidity (Table 35.9).

## EFFECTS OF CARDIAC DISEASE ON PREGNANCY

### Maternal effects

The maternal effects of cardiac disease on pregnancy depend upon the etiology, the type of lesion, functional capacity of heart, presence of risk factors and antenatal care. Various maternal complications can be as follows:

1. Congestive cardiac failure
2. Bacterial endocarditis
3. Pulmonary edema

**Table 35.7: Etiological Classification of Heart Disease**

| |
|---|
| **Rheumatic Heart Disease (90–95%)** |
| 1. Mitral stenosis: most common lesion<br>2. Mitral incompetence<br>3. Aortic stenosis<br>4. Aortic incompetence |
| **Congenital Heart Diseases** |
| 1. Non-cyanotic lesions<br>(a) Atrial septal defect<br>(b) Ventricular septal defect<br>(c) Pulmonary stenosis<br>(d) Coarctation of aorta<br>2. Cyanotic lesions<br>(a) Fallot's tetralogy<br>(b) Eisenmenger's syndrome |
| **Miscellaneous Heart Diseases** |
| 1. Miral valve prolapse<br>2. Cardiac arrythmias and conduction defects<br>3. Cardiomyopathy<br>4. Ischemic heart disease<br>5. Hypertensive heart disease<br>6. Thyrotoxic heart disease<br>7. Syphilitic heart disease. |

**Table 35.8: New York Heart Association (NYHA) Classification of Heart Disease during Pregnancy**

| | |
|---|---|
| **Class 1** | **Uncompromised state;** where patients with cardiac disease have **no limitation of physical activity.** They do not have cardiac symptoms |
| **Class 2** | **Slightly compromised state;** when patients with cardiac disease have **slight limitation of physical activity and are comfortable at rest.** When ordinary physical activity is undertaken, discomfort results in the form of excessive fatigue, palpitation or dyspnea. |
| **Class 3** | **Markedly compromised state;** Patients with cardiac disease have **marked limitation of physical activity.** They are comfortable at rest but even minimal activity causes discomfort and symptoms. |
| **Class 4** | **Severely compromised state; Symptoms at rest;** Patients with cardiac disease are unable to perform any activity without discomfort. |

**Table 35.9A: World Health Organisation (WHO) Risk Classification of Cardiovascular Disease and Pregnancy with Management Recommendations**

| Risk Category | Associated Conditions |
|---|---|
| I. **WHO 1** – Risk no higher than general population | 1. Uncomplicated, small or mild:<br>(i) Pulmonary stenosis<br>(ii) Ventricular septal defect<br>(iii) Patent ductus arteriosus<br>(iv) Mitral valve prolapse with no more than trivial mitral regurgitation<br>2. Successfully repaired simple lesions:<br>(i) Ostium secundum atrial septal defect<br>(ii) Ventricular septal defect<br>(iii) Patent ductus arteriosus<br>(iv) Total anomalous pulmonary venous drainage<br>(v) Isolated ventricular extrasystoles and atrial ectopic beats |
| Cardiology consultation once or twice during pregnancy is recommended. | |
| II. **WHO 2** – Small increase in risk of maternal mortality and morbidity | 1. If otherwise uncomplicated:<br>(i) Unoperated atrial septal defect<br>(ii) Repaired Fallot tetralogy<br>(iii) Most arrhythmias |
| Cardiology consultation is recommended in each trimester | |
| III. **WHO 2 or 3** – Depends on individual case | 1. Mild left ventricular impairment<br>2. Hypertrophic cardiomyopathy<br>3. Native or tissue valvular heart disease not considered WHO 4<br>4. Marfan syndrome without aortic dilation<br>5. Heart transplantation |
| Cardiology consultation in each trimester is recommended. | |
| IV. **WHO 3** – Significantly increased risk of maternal mortality (5–15%) or expert cardiac and obstetrical care required | 1. Mechanical valve<br>2. Systemic right ventricle—congenitally corrected transposition, simple transposition post Mustard or Senning repair<br>3. Post-Fontan operation<br>4. Cyanotic heart disease<br>5. Other complex congenital heart disease |
| These women are to be managed by obstetricians and cardiologists monthly or bimonthly | |
| V. **WHO 4** – Very high risk of maternal mortality (25–50%) or severe morbidity; pregnancy contraindicated and termination discussed | 1. Pulmonary arterial hypertension (primary or secondary)<br>2. Eisenmenger's syndrome<br>3. Severe systemic ventricular dysfunction (NYHA III-IV or LVEF < 30%)<br>4. Previous peripartum cardiomyopathy with any residual impairment of left ventricular function<br>5. Severe left heart obstruction<br>6. Marfan syndrome with aorta dilated > 40 mm |
| 1. Pregnancy is contraindicated and termination indicated<br>2. If pregnancy occurs, monthly or bimonthly cardiac and obstetrical monitoring to be done after counseling. | |

| Table 35.9B: Risk Factors for Maternal Cardiovascular Events during pregnancy | |
|---|---|
| 1 | Past history of cardiac event like Heart failure, TIA, stroke before pregnancy or arrhythmias |
| 2 | Baseline NYHA class III or IV or cyanosis in current pregnancy |
| 3 | Left heart obstruction (MVA < 2 $cm^2$, aortic valve area < 1.5 $cm^2$, peak LV out flow tract gradient > 30 mm Hg by echocardiography |
| 4 | Reduced systemic ventricular systolic function, i.e. ejection fraction < 40%. |
| | No risk factors - 5% risk of complications<br>1 risk factor - 27% risk of complications<br>> 1 risk factor - 75% risk of complications |

TIA: Transient ischemic attachs; NYHA: NewYork Heart Association; MVA: Mitral valve area; LV: Left ventricular Complications include pulmonary edema, stroke, TIA, arrhythmias requiring treatment, cardiac arrest, cardiac death.

4. Active rheumatic carditis (rheumatic heart disease)
5. Pulmonary embolism
6. Rupture of aneurysm in coarctation of aorta
7. Premature labour
8. Maternal death

### Foetal effects

The effects on perinatal outcome also depend upon the etiology, type of lesion and functional capacity. Perinatal mortality is particularly high (up to 45%) in cyanotic lesions. Various foetal effects are as follows:

1. Increased chances of miscarriage
2. Prematurity
3. Foetal growth restriction (FGR)
4. *Increased chances of foetal congenital malformations (3–10%) if parents have congenital cardiac disease. Foetal congenital cardiac disease tends to be same as in mother.*
5. Foetal death

## MANAGEMENT OF PREGNANCY

The main aim of management of a cardiac patient is prevention, early diagnosis and prompt and appropriate treatment of cardiac failure during pregnancy. Such patients should always be managed in tertiary care hospitals preferably with facilities of cardiology and cardiothoracic surgery and they should be regularly seen jointly by an obstetrician and a cardiologist.

### Prepregnancy counseling

Ideally all women with heart disease should have preconceptional counseling. The women with contraindication of pregnancy (e.g. Eisenmenger's syndrome) should be advised against pregnancy while women with valvular lesion should conceive when they are in NYHA class I or II. Similarly, women with severe mitral stenosis or congenital heart disease needing cardiac surgery should have surgery before they venture for pregnancy for optimum outcome. Unfortunately, most women with heart disease in India usually report during late pregnancy.

### Role of Medical Termination of Pregnancy (MTP) in heart disease

As shown in Table 35.9 pregnacy is contraindicated and therapeutic abortion is indicated for women with high risk heart disease.

**Absolute contraindications of pregnancy (Indications of MTP)**

1. Eisemenger's syndrome
2. Primary pulmonary hypertension
3. Single ventricle
4. Pulmonary veno-occlusive disease
5. Marfan's syndrome with dilated aortic root (> 4 cm on echocardiography)
6. Dilated cardiomyopathy (peripartum cardiomyopathy) with residual left ventricular function impairment.
7. Bicuspid aortic valve with aortic root > 5 cm.

**Relative Contraindications of Pregnancy (Relative indications for MTP)**

1. Severe obstructive lesion. Left ventricular ejection fraction < 30%.
2. Parous woman with grade III or IV lesion
3. Past history of cardiac failure with heart disease

MTP should be done using suction and evacuation before 12 weeks of pregnancy (preferably before 8 weeks). After this termination may be as dangerous as continuing pregnancy and usually pregnancy is continued in such cases.

### Antenatal care

Whenever cardiac disease is diagnosed in pregnancy, the woman should be referred to a tertiary care center where she should be seen regularly by an experienced obstetrician and a cardiologist. She should be counseled regarding high risk factors for timely diagnosis and treatment of cardiac failure. The following antenatal protocol can be pursued.

1. She should be seen every 2 weeks until 30 weeks of pregnancy and then weekly until delivery.
2. At each visit she should be asked about any increase in dyspnea or limitation of activity.
3. She should be given iron and folic acid as per routine for all pregnant women. Women with > 11 g/dL hemoglobin should receive 100 mg elemental iron with at least 0.5–1 mg folic acid per day, while the women with anaemia should receive therapeutic dose of iron (200 mg per day) and folic acid (5 mg/day). Women with heart disease should not be anaemic as it adds to their morbidity.
4. At each visit she should be asked detailed history of any symptoms suggestive of cardiac failure and her functional cardiac status should be assessed. Increasing dyspnea, orthopnoea, fine crepitations on bases of lungs are indicative of cardiac failure and usually require admission.

5. She is asked to avoid excess work, stress and should have home help.
6. She should avoid infection such as influenza by avoiding contact with people having respiratory infections. She should take care of dental hygiene.
7. She should avoid alcohol, smoking and use of illicit drugs.
8. Some salt restriction is advisable.
9. At each antenatal visit her pulse, blood pressure, weight gain, respiratory rate, jugular venous pressure are checked and auscultation of lungs is performed to diagnose and treat impending cardiac failure.
10. Careful vigilance for any signs of pre-eclampsia.
11. Early diagnosis and treatment of any infection like urinary tract infection, dental infection or respiratory tract infection.
12. All women with rheumatic heart disease should receive penicillin prophylaxis to avoid bacterial endocarditis as injection benzathine penicillin (Penidura LA 1.2 mega units) by deep intramuscular injection after sensitivity test every 3 weeks throughout pregnancy.
13. Foetal monitoring by ultrasound, non-stress test or Manning's biophysical scoring.

### Indications of Admission to hospital

1. Woman with classes III and IV disease should be admitted whenever diagnosed and should stay in hospital until delivery or until their functional cardiac status improves to lower grade.
2. Women with classes I and II disease should be admitted at least 2 weeks prior to expected date of delivery.
3. Worsening of functional cardiac status
4. Cardiac failure, infective endocarditis or anaemia.
5. Appearance of high risk factors like cough, dyspnea, tachyarrhythmias or basal lung crepitations.
6. Women with prosthetic heart valves (mechanical) for heparin switch over in first trimester and at 36 weeks.
7. Pulmonary hypertension

## MANAGEMENT OF LABOUR AND DELIVERY

**It is generally agreed that vaginal delivery is the safest mode of delivery for women with heart disease.** Cesarean section should be done for strict obstetric indications.

### Indications of Induction of Labour

Induction of labour is best avoided and spontaneous onset of labour should be awaited. However, for strict obstetric indications for induction either prostaglandin gel is used or oxytocin should be given in concentrated dosage to avoid fluid overload.

1. Usual obstetric indications
2. Associated pre-eclampsia.
3. Intrahepatic cholestasis of pregnancy (ICP)
4. Non-reassuring biophysical profile

### Indications of Cesarean Section

1. Strict obstetric indications (e.g. major degree placenta previa, contracted pelvis, etc.)
2. Coarctation of aorta where elective cesarean section is indicated to prevent rupture of the aorta or mycotic cerebral aneurysm.
3. Large aortic aneurysm
4. Pheochromocytoma
5. Marfan's syndrome with dilated aortic root
6. Aortic dissection
7. On oral anticoagulants in pre-term labour
8. Warfarin within 2 weeks of labour
9. Severe arortic stenosis (AS)
10. Acute intractable heart failure

**Table 35.10: Current Recommendations for Antibiotic Prophylaxis in Heart Disease in Labour**

| | |
|---|---|
| 1. | Not recommended for either vaginal delivery or cesarean delivery in the absence of infection, except in patients at highest potential risk for adverse cardiac outcomes |
| 2. | Not recommended for non-dental procedures in the absence of active infection |
| Antibiotics are recommended in | |
| 1. | Women with prosthetic cardiac valves including bioprosthetic valves |
| 2. | Past history of bacterial endocarditis |
| 3. | Patient with congenital heart disease with<br>– Unrepaired cyanotic heart disease<br>– Completely repaired CHD with prosthetic maternal or device during the first 6 months after the procedure.<br>– Repaired CHD with residual defects at the site or adjacent to the site of prosthetic patch or device. |
| 4. | Cardiac transplant recepient with valve regurgitation due to structurally abnormal valve. |
| Drugs for antibiotic prophylaxis | |
| **Intravenous**<br>Ampicillin (2 gm) or Cefazolin (1 g) or Ceftriaxone (1 g) (30–60 minutes before procedure)<br>If allergic to pencillin/ampicillin: Clindamycin 600 mg (I/V)<br>**Oral**<br>Amoxicillin – 2 g<br>Vancomycin is given if infection by enterococcus is suspected. | |

### Management of First Stage of Labour

1. Patient should rest in bed in a semi-recumbent position.
2. Intravenous fluids should be avoided or restricted to 50–75 mL/hour.
3. Central venous pressure monitoring is useful especially in high risk patients.
4. Intranasal oxygen, if needed, should be given at a rate of 5–6 liters per minute (in grades III and IV diseases)
5. For pain relief, epidural analgesia is best and is encouraged (contraindicated in Eisenmenger's syndrome, severe aortic stenosis and hypertrophic cardiomyopathy where pudendal block or pethidine injection may be given).
6. Careful monitoring of vitals regularly to see any sign of cardiac failure as follows:
   (a) Pulse rate > 100 beats per minute

(b) Edema feet
(c) Dyspnea (respiratory rate > 24 per minute)
(d) Raised JVP
(e) Cyanosis
(f) Basal crepitations

7. **Prophylactic antibiotics.** As per American Heart Association prophylactic antibiotics are not necessary in all heart disease cases but in moderate and high risk cardiac disease patients only. Recommendation given in Table 35.10. Current recommendations for antibiotics prophylaxis in heart disease during labour are given in Table 35.10.
8. **Invasive monitoring.** By arterial line or pulmonary artery catheterization (PAC) in high risk cases.

### Second Stage of Labour

Prophylactic forceps are not routinely applied for all cases of heart disease. Usually these women have quick spontaneous delivery. If woman does not deliver within 30 minutes of second stage, forceps or ventouse delivery should be applied under epidural analgesia or pudendal block analgesia. Although outlet forceps has been traditionally preferred over ventouse due to no need of maternal effort for forceps, ventouse is preferred these days as it obviates the need of lithotomy position, which may be hazardous for the woman.

### Third Stage of Labour

Intramuscular oxytocin 10 units at delivery of placenta is given except in lesions with left to right shunt or severe outflow obstruction (aortic stenosis). Ergometrine is better avoided to prevent sudden overloading of the heart by the sudden increase in intravascular volume from contracted uterus. Prostaglandins also have adverse cardiovascular effects and are best avoided. Misoprostol 1000 µg per rectum is useful to control haemorrhage but can cause hypotension and rarely, hyperpyrexia. However, postpartum haemorrhage should be energetically treated in these women.

## PUERPERIUM

Women with heart disease must be meticulously monitored in puerperium as there is a risk of mortality from postpartum haemorrhage, anaemia, infection and thromboembolic events. The concept of **'Golden Hour'** especially holds true for heart disease patient. She is observed closely by hourly pulse, BP and respiratory rate. Oxygen is administered. Breastfeeding is allowed except when the patient is in failure. Women on anticoagulant therapy can also breastfeed their babies.

### Contraception

Combined oral pills are contraindicated as they can precipitate thromboembolism. Intrauterine device was avoided for fear of infection (relative contraindication) in past but current WHO eligibility criteria recommend its use (category 2). Levonorgestrel intrauterine system can be inserted under all aseptic precautions and infective endocarditis prophylaxis. Implanon can also be given. Progestogen only pill can be given. Inj. DMPA 3 monthly is a good option. Barrier method (condom) is the best method but has high failure rate. Permanent method like minilaparotomy sterilization or husband's vasectomy can be performed.

### Treatment of Cardiac failure

Treatment of cardiac failure is like in non-pregnant state in collaboration with cardiologist. The patient should be put in propped up position and given oxygen administration. She should be given intravenous furosemide (40 mg) with sedation (morphine 15 mg intramuscularly). Digitalization by giving 0.5 mg digoxin IV followed by 0.25 mg oral digoxin every day is only indicated for atrial fibrillation, otherwise its value is disputed.

## INDIVIDUAL HEART DISEASES IN PREGNANCY–RHEUMATIC HEART DISEASE

### Mitral stenosis

Mitral stenosis is the most common lesion in rheumatic heart disease and is important in pregnancy as women may deteriorate secondary to tachycardia, arrhythmias or due to increased cardiac output.

#### Classification

The classification depends upon valve area on echocardiography **(Echo grading)**.

1. **Normal.** Normal mitral valve area is 4–6 $cm^2$
2. **Mild stenosis.** < 4 $cm^2$ to > 1.5 $cm^2$
3. **Moderate stenosis.** 1.5–1 $cm^2$
4. **Severe stenosis.** < 1 $cm^2$

Symptoms like dyspnea (most common), fatigue, palpitations, cough and hemoptysis usually appear when the area decreases to < 2.5 $cm^2$, while in severe stenosis patient is symptomatic even at rest. Mortality is less than 1% in asymptomatic women but increases significantly (5–15%) in severe stenosis.

#### Complications

Hyperdynamic circulatory changes in pregnancy can cause.

1. an increase in left atrial pressure.
2. increased risk of atrial fibrillation.
3. left heart failure with pulmonary edema.

#### Preconceptional Counselling

Preconceptional counselling is important especially in severe mitral stenosis where women are counseled to get mitral valvotomy (balloon, open or closed) or valve replacement done before conception for optimum maternal and foetal outcomes.

#### Role of Mitral Valvotomy in Pregnancy

Ideally, it should be done prior to conception in severe mitral stenosis. However, many women in India present for the first time during pregnancy with symptoms of severe mitral stenosis with heart failure or atrial fibrillation. Although it is better to withhold elective heart

(b) Edema feet
(c) Dyspnea (respiratory rate > 24 per minute)
(d) Raised JVP
(e) Cyanosis
(f) Basal crepitations

7. **Prophylactic antibiotics.** As per American Heart Association prophylactic antibiotics are not necessary in all heart disease cases but in moderate and high risk cardiac disease patients only. Recommendation given in Table 35.10. Current recommendations for antibiotics prophylaxis in heart disease during labour are given in Table 35.10.
8. **Invasive monitoring.** By arterial line or pulmonary artery catheterization (PAC) in high risk cases.

### Second Stage of Labour

Prophylactic forceps are not routinely applied for all cases of heart disease. Usually these women have quick spontaneous delivery. If woman does not deliver within 30 minutes of second stage, forceps or ventouse delivery should be applied under epidural analgesia or pudendal block analgesia. Although outlet forceps has been traditionally preferred over ventouse due to no need of maternal effort for forceps, ventouse is preferred these days as it obviates the need of lithotomy position, which may be hazardous for the woman.

### Third Stage of Labour

Intramuscular oxytocin 10 units at delivery of placenta is given except in lesions with left to right shunt or severe outflow obstruction (aortic stenosis). Ergometrine is better avoided to prevent sudden overloading of the heart by the sudden increase in intravascular volume from contracted uterus. Prostaglandins also have adverse cardiovascular effects and are best avoided. Misoprostol 1000 µg per rectum is useful to control haemorrhage but can cause hypotension and rarely, hyperpyrexia. However, postpartum haemorrhage should be energetically treated in these women.

## PUERPERIUM

Women with heart disease must be meticulously monitored in puerperium as there is a risk of mortality from postpartum haemorrhage, anaemia, infection and thromboembolic events. The concept of **'Golden Hour'** especially holds true for heart disease patient. She is observed closely by hourly pulse, BP and respiratory rate. Oxygen is administered. Breastfeeding is allowed except when the patient is in failure. Women on anticoagulant therapy can also breastfeed their babies.

### Contraception

Combined oral pills are contraindicated as they can precipitate thromboembolism. Intrauterine device was avoided for fear of infection (relative contraindication) in past but current WHO eligibility criteria recommend its use (category 2). Levonorgestrel intrauterine system can be inserted under all aseptic precautions and infective endocarditis prophylaxis. Implanon can also be given. Progestogen only pill can be given. Inj. DMPA 3 monthly is a good option. Barrier method (condom) is the best method but has high failure rate. Permanent method like minilaparotomy sterilization or husband's vasectomy can be performed.

### Treatment of Cardiac failure

Treatment of cardiac failure is like in non-pregnant state in collaboration with cardiologist. The patient should be put in propped up position and given oxygen administration. She should be given intravenous furosemide (40 mg) with sedation (morphine 15 mg intramuscularly). Digitalization by giving 0.5 mg digoxin IV followed by 0.25 mg oral digoxin every day is only indicated for atrial fibrillation, otherwise its value is disputed.

## INDIVIDUAL HEART DISEASES IN PREGNANCY–RHEUMATIC HEART DISEASE

### Mitral stenosis

Mitral stenosis is the most common lesion in rheumatic heart disease and is important in pregnancy as women may deteriorate secondary to tachycardia, arrhythmias or due to increased cardiac output.

#### Classification

The classification depends upon valve area on echocardiography **(Echo grading)**.

1. **Normal.** Normal mitral valve area is 4–6 $cm^2$
2. **Mild stenosis.** < 4 $cm^2$ to > 1.5 $cm^2$
3. **Moderate stenosis.** 1.5–1 $cm^2$
4. **Severe stenosis.** < 1 $cm^2$

Symptoms like dyspnea (most common), fatigue, palpitations, cough and hemoptysis usually appear when the area decreases to < 2.5 $cm^2$, while in severe stenosis patient is symptomatic even at rest. Mortality is less than 1% in asymptomatic women but increases significantly (5–15%) in severe stenosis.

#### Complications

Hyperdynamic circulatory changes in pregnancy can cause.

1. an increase in left atrial pressure.
2. increased risk of atrial fibrillation.
3. left heart failure with pulmonary edema.

#### Preconceptional Counselling

Preconceptional counselling is important especially in severe mitral stenosis where women are counseled to get mitral valvotomy (balloon, open or closed) or valve replacement done before conception for optimum maternal and foetal outcomes.

#### Role of Mitral Valvotomy in Pregnancy

Ideally, it should be done prior to conception in severe mitral stenosis. However, many women in India present for the first time during pregnancy with symptoms of severe mitral stenosis with heart failure or atrial fibrillation. Although it is better to withhold elective heart

surgery during pregnancy, it can be considered in cases of cardiac failure refractory to medical treatment especially in the second trimester (14–18 weeks) of pregnancy. Percutaneous transcatheter balloon dilatation of the mitral value has largely replaced surgical valvotomy during pregnancy in the modern times. Atrial fibrillation can be a complication for which digitalization, β-blockers or heparin anticoagulation should be used.

**Management of pregnancy and labour in mitral stenosis** is the same as in a case of general heart disease with pregnancy described above. Antibiotic prophylaxis during pregnancy with penicillin and during labour is essential. Early treatment of cardiac failure is required.

During labour, epidural analgesia is ideal with the aim of vaginal delivery and avoiding fluid overload.

### Mitral regurgitation

Mitral regurgitation is usually better tolerated than stenosis in pregnancy but, regular monitoring is required in pregnancy as pulmonary edema and fibrillation can occur. Antibiotic prophylaxis during pregnancy and labour is required.

### Mitral valve prolapse

Mitral valve prolapse may be seen in 12–17% women in child bearing age and rarely causes any complication in pregnancy. If it gets worse then the woman is treated like regurgitation.

### Aortic Stenosis

Aortic stenosis < 1.5 $cm^2$ (normal: 2–3 $cm^2$) is associated with high maternal mortality (8–10% in moderate stenosis, 15–20% in severe stenosis) and perinatal mortality (up to 30%). Standard epidural anesthesia is contraindicated in them and intravenous fluids should be given cautiously and hypotension is avoided. Instrumental delivery can be used and antibiotic prophylaxis is given.

### Pulmonary stenosis

Pulmonary stenosis is rare. If it is severe it can cause right heart failure and atrial fibrillation due to arrythmias.

### Pregnancy after valve replacement surgery

With the advancement in cardiac care and cardiac surgery, most adolescent girls with severe valvular lesions are undergoing valvular replacement surgeries (severe mitral stenosis, combined mitral stenosis and mitral regurgitation, extensive calcification of valve, failed valvotomy). Two types of valves are used. Bio-prosthetic valves (porcine or human allograft) have the advantage of not using anticoagulation but need revision surgery. Mostly mechanical valves are used these days as they have long life but require long term anticoagulation because of the hazard of thromboembolism. **The risk of thrombosis increases 6-fold in pregnancy and 11-fold in puerperium.** The safest option for the mother is to continue warfarin throughout pregnancy as it is a better anticoagulant than heparin with less incidence of thrombosis. However, warfarin crosses the placenta and can lead on to **foetal warfarin embryopathy** which consists of facial and skeletal dysmorphism (chondrodysplasia punctata), nasal hypoplasia, optic atrophy, microcephaly, cardiac defects and intrauterine growth restriction and also causes placental haemorrhage and a high foetal loss rate. The risk of warfarin embryopathy is dose dependant < 3% if dose is < 5 mg per day, but increases to 8% if dose is > 5 mg/day. Hence, some centers recommend giving unfractionated heparin 5000 units twice daily subcutaneously between 6–12 weeks of pregnancy. It is followed by oral warfarin. Normal dosage of warfarin is adjusted keeping prothrombin time (PT) twice and international normalized ratio (INR) 2–3 times of normal followed by oral warfarin to be taken at the same time every day until 36 weeks of pregnancy. Thereafter, it is to be replaced with heparin until 3–4 days of puerperium when warfarin is restarted (it is safe for breastfeeding). Efficacy of subcutaneous low molecular weight heparin to prevent thrombosis in pregnancy is still not fully established but it can be used in place of unfractionated heparin. American Heart Association and American College of Cardiology (2014) and ACOG (2017) have given recommendations for anticoagulation in pregnant patients with mechanical prosthetic valves by recommending either heparin or low molecular weight heparin (LMWH) throughout pregnancy or hepain or LMWH in Ist trimester and after 36 weeks and warfarin in between.

If patient goes into labour on warfarin, then cesarean delivery is preferred to prevent intracranial foetal bleeding. If she is in advanced labour, give intramuscular vitamin K to mother (also to baby after birth). 4 factor prothrombin complex concentrate (4P- PCC, rich in factors 2, 7, 9 and 10) (preferred) or fresh frozen plasma (if 4P-PCC is not available) is given intravenously to the mother to prevent postpartum haemorrhage (PPH).

If patient goes into labour on low molecular weight heparin (LMWH), give protamine intravenously to the mother and continue vaginal delivery.

If patient goes into labour on unfractionated heparin (ufH), stop ufH, allow vaginal delivery. Usually protamine is not required unless there is bleeding.

Risk of PPH (postpartum haemorrhage is least with ufH, intermediate with LMWH and is highest with warfarin.

### Monitoring

For warfarin; INR (International normalised ratio) is kept between 2-3 times of normal.

For ufH; APTT is kept about 2 times of normal.

For LMWH; Usually monitoring is not required. Factor Xa levels can be done and are kept between 0.8-1.2 .

Stop all anticoagulants on day of delivery. Restart after childbirth; 24 hours after cesarean delivery. ufH or LMWH can be started. Add warfarin orally between 5-7 days and then ufH or LMWH can be stopped.

## CONGENITAL HEART DISEASES IN PREGNANCY

Congenital heart diseases in pregnancy are shown in Table 35.11.

**Table 35.11: Congenital Heart Disease in Pregnancy**

| Lesion | Transmission to fetus (risk of congenital HD) in fetus | Pregnancy management | Antenatal management | Intrapartum management | Postpartum management |
|---|---|---|---|---|---|
| Mitral valve prolapse | | Echo cardiography to check for mitral regurgitation | - Arrhythmia surveillance<br>- Infective endocarditis prophylaxis | Same as in heart disease | Same as in heart disease |
| Ventricular septal defects (VSD) and atrial septal defects (ASD) | 6–10% | Screen for pulmonary hypertension; Consider repair of uncorrected lesion | Serial echocardiography<br><br>Foetal echocardiography | Avoid hypertension | Careful fluid balance |
| Pulmonary hypertension including Eisenmenger's syndrome complex | | 1. Counsel against pregnancy<br>2. Echo-cardiography | 1. Terminate<br>2. Obstetric cardiology anesthesiology liaison<br>3. Thromboembolism prophylaxis<br>4. Hospital admission and $SpO_2$ (oxygen saturation) monitoring<br>5. Foetal surveillance | 1. Invasive monitoring<br>2. Fluid balance<br>3. Epidural analgesia<br>4. Strict fluid balance<br>5. IV or inhalated prostacyclin | 1. Oxygen<br>2. Thromboprophylaxis<br>3. Fluid balance<br>4. Prostacyclin or nitrous oxide (NO) |
| Coarctation of aorta | 4–7% | Screen for aneurysms and aortic stenosis | Terminate, if severe | Avoid hypertension, cesarean delivery | |
| Tetralogy of Fallot (TOF) | 5% | Surgical correction | 1. Terminate, if severe<br>2. $SpO_2$ maintenance<br>3. $O_2$ supplementation | Epidural, vaginal delivery | |
| Transposition of great arteries (TGA) | Probably not transmitted | Evaluate cardiac status (especially right ventricle) arrhythmia screen | Echocardiography | 1. ECG<br>2. Cut short second stage of labour<br>3. Electronic foetal monitoring | Fluid balance |
| Patent ductus arteriosus (PDA) | 4% | Screen for pulmonary hypertension | Screen for pulmonary hypertension | Screen for pulmonary hypertension | |
| Marfan's syndrome | 50% | 1. Genetic counselling<br>2. Counsel against pregnancy<br>3. Echo for aortic root dilatation | 1. Serial echo for aortic root<br>2. β-blocker<br>3. Avoid hypertension<br>4. Surgery in extreme cases | Cut short second stage of labour, cesarean section if aortic root dilatation | Vigilance for aortic dissection up to 8 weeks of puerperium |

## MISCELLANEOUS CARDIAC DISORDERS IN PREGNANCY

### Cardiomyopathy

Cardiomyopathy can predate pregnancy in cases of hypertrophic, dilated and restrictive cardiomyopathy. Peripartum cardiomyopathy has following features.

1. Development of cardiac failure in the last month of pregnancy or within 5 months of delivery.
2. Absence of an identifiable cause for the heart failure.
3. Absence of recognizable heart disease prior to the last month of pregnancy.
4. Left ventricular systolic dysfunction on echocardiography (like decreased ejection fraction). Treatment is bed rest, digoxin and diuretics (for heart failure), hydralazine (to reduce afterload), β-blockers and prophylactic heparin (for high incidence of associated thromboembolism). Vaginal delivery is preferred and epidural anesthesia is advisable.
5. Peripartum cardiomyopathy is usually seen in older and obese multiparous women with hypertension. The women usually present with dyspnea, tachycardia or pulmonary edema. The complications are thromboembolism (50% cases), arrhythmias (often fatal) and heart failure. There is a very high mortality (up to 25%) and risk of recurrence in subsequent pregnancy.

### Ischemic heart disease

Ischemic heart disease is rare in pregnancy. However, trends to delay child bearing to thirties and forties, advancing maternal age, obesity and smoking and severe hypertension all put women at high risk for ischemic heart disease. Treatment of acute myocardial infarction is the same as in non-pregnant state with even coronary angioplasty being performed. Labour management is like any other cardiac case but elective delivery within two weeks of infarction should be avoided. β-blockers may be used for tachycardia and epidural analgesia can be given. Vaginal delivery is preferred. The second stage should be shortened with forceps to avoid maternal strain.

## HEART DISEASE WITH PRETERM LABOUR

Patients with heart disease during pregnancy may go into preterm labour. Tocolytic drugs like ritodrine and betamethasone given for foetal pulmonary maturity can make them prone to pulmonary edema which can be serious. Hence, caution must be exercised and limited fluids should be given. Oxytocin antagonist atosiban if available is best in such a case as a tocolytic agent and is superior to β agonists.

## Key Points

1. Heart disease is an important cause of maternal morbidity and mortality.
2. Certain physiological cardiovascular changes in pregnancy can mimic cardiac disease.
3. Heart disease with primary or secondary pulmonary hypertension, severe mitral stenosis, severe peripartum cardiomyopathy and Marfan's syndrome with aortic root dilatation are contraindications for pregnancy.
4. Pregnancy counselling is important and the woman should be advised to try pregnancy only when her cardiac disease is under control.
5. Cardiac disease during pregnancy is classified by New York Heart Association (NYHA) as class 1 being with no limitation of physical activity, class 2 with slight limitation of physical activity, class 3 with marked limitation of physical activity, while class 4 being severely compromised patients who may be symptomatic even at rest.
6. Women with heart disease should be carefully managed jointly by obstetrician and cardiologist by regular and more frequent ANC visits, iron and folate therapy, antibiotic prophylaxis and careful vigilance and early treatment of any cardiac failure.
7. As a general principle women with cardiac disease should go into spontaneous labour. Induction and augmentation are avoided unless absolutely indicated. Aim is to deliver them vaginally. During labour they should be kept in propped up position, minimum fluid should be given to avoid overloading the circulation, antibiotic prophylaxis and to avoid prolongation of the second stage of labour. Active management of the third stage is avoided but PPH is to be energetically treated with oxytocin. Epidural analgesia can be given.
8. Cesarean delivery is avoided but is to be done for coarctation of aorta, Marfan's syndrome with aortic arch dilatation and for strict obstetric indications.

## Suggested Reading

1. American College of Obstetricians and Gynecologists: Use of Prophylactic Antibiotics in Labour and Delivery. Practice Bulletin No. 120, June 2011, Reaffirmed 2016.

# (iii) DIABETES MELLITUS IN PREGNANCY

## INTRODUCTION

### Definition

**Diabetes mellitus is defined as a carbohydrate disturbance characterized by hypeglycemia with peripheral insulin deficiency or insulin resistance.** It is a common metabolic disorder.

### Prevalence

Incidence varies from 1 to 14% in the world (5–8% in Asia including India) depending upon ethnicity, selection criteria and diagnostic tests performed. Most (90%) of these cases have gestational diabetes mellitus (GDM).

### Classification

The various classifications of diabetes are as follows:

**I. Etiological Classification of Diabetes Mellitus**

1. **Type I (1) diabetes mellitus (insulin dependent):** It is a condition of absolute insulin deficiency, previously called insulin dependant diabetes mellitus (IDDM) and is usually seen in young age group (juvenile onset). It is further of two types:
   Type IA: Immune mediated β-cell destruction
   Type IB: Idiopathic β-cell destruction
2. **Type II (2) diabetes mellitus (noninsulin dependent):** Previously called non-insulin dependent diabetes mellitus (NIDDM), it is usually seen with increasing age (can occur in obese adolescents also) and is due to either defective insulin secretion or insulin resistance. Age and the terms of IDDM and NIDDM are no longer used in classification.
3. Other specific type–
   i. Genetic mutation of β cell function
   ii. Genetic defect in insulin action
   iii. Genetic syndromes (Down, Turner, Klinefelter)
   iv. Diseases of exocrine pancreas, e.g. pancreatitis
   v. Endocrinopathies, e.g. Cushing syndrome
   vi. Drug or chemical induced (Steroids or Thiazide)
   vii. Infections (Congenital rubella, CMV)
4. Gestational diabetes mellitus (GDM)

**II. Classification in Pregnancy**

Any degree of glucose intolerance with onset or first recognition during pregnancy is defined as GDM.

ACOG (2017) defines GDM as carbohydrate intolerance during pregnancy. GDM is much more common than pregestational diabetes.

- A1GDM: Adequately controlled without medication/diet controlled
- A2GDM: GDM requiring medication to achieve euglycemia

**Table 35.12: Classification of diabetes in Pregnancy**

| | |
|---|---|
| 1. | **Type 1 Diabetes:** Diabetes resulting from β-cell destruction, usually leading to absolute insulin deficiency<br>(a) Without vascular complications<br>(b) With vascular complications (specify which) |
| 2. | **Type 2 Diabetes:** Diabetes from inadequate insulin secretion in the face of increased insulin resistance<br>(a) Without vascular complications<br>(b) With vascular complications (specify which) |
| 3. | **Other types of diabetes:** genetic in origin, associated with pancreatic disease, drug-induced, or chemically induced. |

**III. White's Classification of Diabetes during Pregnancy** (Table 35.13)

**Priscilla White** (1978) classified diabetes during pregnancy depending upon its severity and duration to formulate the obstetric management and perinatal outcome. It is important for statistical purposes to compare the results of different centers of the world. Vasculopathy and end organ (eye, kidneys, heart) derangements are given more importance due to their significant effects on pregnancy outcome. However, this classification is not often used these days.

### Glycosuria during pregnancy

Glycosuria may be detected on random urine samples in about 5–50% (average 16%) cases during pregnancy

**Table 35.13: White's Classification of Diabetes during Pregnancy**

| Class | Onset | Fasting Plasma Glucose | 2-Hour Postprandial Glucose | Treatment |
|---|---|---|---|---|
| $A_1$ | Gestational | < 105 mg/dL | < 120 mg/dL | Diet |
| $A_2$ | Gestational | > 105 mg/dL | > 120 mg/dL | Insulin |
| **Class** | **Age of onset (years)** | **Duration (yrs)** | **Vascular disease** | **Treatment** |
| B | Over 20 | < 10 | None | Insulin |
| C | 10 to 19 | 10 to 19 | None | Insulin |
| D | Before 10 | > 20 | Benign retinopathy | Insulin |
| F | Any age | Any duration | Nephropathy | Insulin |
| R | Any age | Any duration | Proliferative retinopathy | Insulin |
| H | Any age | Any duration | Heart (cardiomyopathy) | Insulin |
| T | Any age | Any duration | Prior renal transplantation | Insulin |

especially in the mid trimester. It is because of diminished renal threshold due to appreciable increase in glomecular filtration together with impaired tubular reabsorptive capacity for filtered glucose. Blood sugar levels are usually normal and well below the normal renal threshold of 180 mg per 100 mL. It usually does not require any treatment and usually disappears after delivery. However, possibility of gestational diabetes mellitus should be kept in mind.

## GESTATIONAL DIABETES MELLITUS

### Definition

Gestational diabetes mellitus (GDM) is carbohydrate intolerance of variable severity first diagnosed during pregnancy. It generally occurs in the second half of pregnancy.

### Pregnancy as a diabetogenic state

**Effect of Pregnancy on Diabetes.** Pregnancy alters the carbohydrate metabolism in such a way that more glucose is available for the fetus as shown in Fig. 35.3A.

The hormonal changes like increased placental hormones act as insulin antagonizers causing insulin resistance and decreased insulin sensitivity which is further aggravated by increased body weight and caloric intake during pregnancy. Human placental lactogen (hPL) promotes lipolysis making free fatty acids available for mother's own metabolism ensuring availability of adequate glucose for the fetus. Gestational diabetes occurs when the pancreas, despite the production of insulin fails to overcome the effect of these contra-insulin hormones.

### Risk factors for gestational diabetes

The risk factors for gestational diabetes are shown in Table 35.14.

**Table 35.14: Risk Factors for Gestational Diabetes Mellitus**

| Factors in History |
|---|
| 1. Age over 30 years |
| 2. Past history of GDM |
| 3. Family history of diabetes mellitus |
| 4. Bad obstetric history |
| 5. Prior history of macrosomic baby |
| 6. Previous stillbirth |
| 7. Previous foetal anomalies |
| 8. Unexplained perinatal loss |
| 9. History of polycystic ovarian syndrome (PCOS) |
| **Factors in Present Pregnancy** |
| 1. Polyhydramnios |
| 2. Recurrent vaginal candidiasis |
| 3. Recurrent urinary tract infection |
| 4. Obesity (> 90 kg) |
| 5. Congenital foetal anomalies |
| 6. Pre-eclampsia |
| 7. Persistent glycosuria |

### Screening for GDM

Despite tremendous research, there is no consensus about screening for GDM and whether universal or selective screening should be performed. The fifth International Workshop Conference on Gestational Diabetes (2007) had given risk assessment for detecting GDM and changed universal screening to selective screening for GDM. However, American College of Obstetricians and Gynecologists (2017) has now recommended universal screening for all pregnancies.

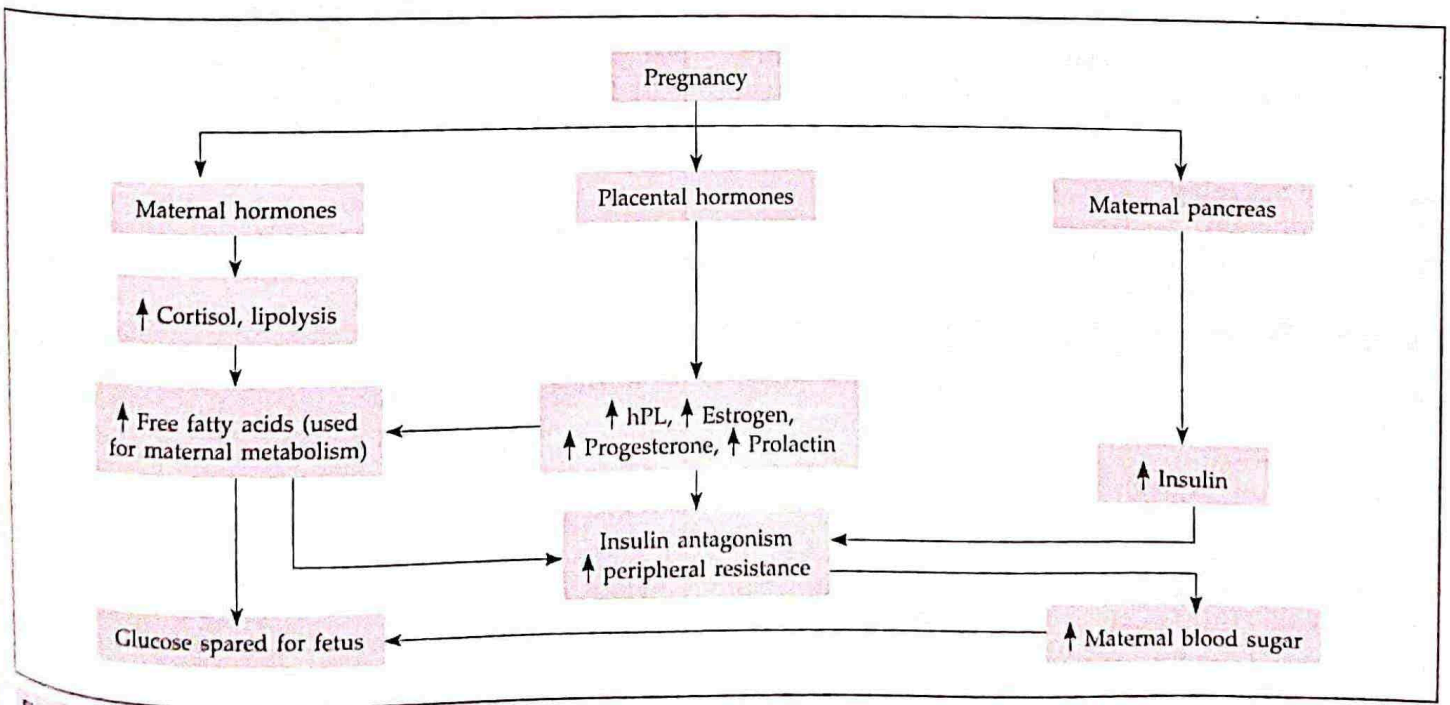



Fig. 35.3A: Changes in carbohydrate metabolism during pregnancy

| Table 35.15: Risk Assessment for Gestational Diabetes Mellitus |
|---|
| Low Risk |
| **Routine screening not recommended.**<br>1. Age less than 25 years<br>2. Weight normal before pregnancy<br>3. No family history of diabetes in first-degree relatives<br>4. Ethnic group with low prevalence of GDM<br>5. No bad obstetric history<br>6. No history of abnormal glucose metabolism |
| Average Risk |
| **Blood glucose testing at 24–28 weeks is recommended.**<br>1. **Women of South-East Asian origin, including Indians**<br>2. Women of Native American, African or Hispanics |
| High Risk |
| **Blood glucose testing at the earliest and repeated at 24–28 weeks or when there are symptoms or signs of hyperglycemia.**<br>1. Obesity (> 200 lbs or > 15%) of non-pregnant ideal body weight)<br>2. Positive family history of diabetes (in siblings or parents)<br>3. History of stillbirth<br>4. History of delivery of a large infant (> 4000 g)<br>5. Glycosuria<br>6. History of unexplained neonatal death<br>7. History of congenital anomaly<br>8. History of pre-eclampsia in multipara<br>9. Polyhydramnios<br>10. History of traumatic delivery with associated neurologic disorder in infant<br>11. Poor reproductive history (> 3 spontaneous abortions in first and second trimesters)<br>12. Recurrent urinary tract infection (UTI), candidiasis<br>13. History of diabetes in previous pregnancy<br>14. Age > 30 years |

**Risk assessment for GDM.** It is shown in Table 35.15. In Indian hospitals screening is usually done as Indian women are at high risk of developing GDM.

## Methods of Screening

### Screening in GDM

Why is there a need to screen?

1. Women with a history of GDM are at increased risk of future diabetes predominantly type 2 DM as are their children.
2. Treatment of GDM reduces serious perinatal morbidity and may also improve the woman's health related quality of life.
3. Treatment of hyperglycemia in pregnancy improves neonatal outcome.
4. Treatment of mild GDM is also beneficial.

These changes have been made in accodance with the results of HAPO study (Hyperglycemia and Insulin Resistance in Pregnancy Outcome) which found found increased adverse foetal outcome with hyperglycemia in the form of primary cesarean rates and neonatal hypoglycemia.

If the initial screening (of Ist trimester, i.e., Ist visit) is negative then retest at 24–28 weeks with 75 g oral GTT.

In women with GDM, GTT has to be repeated 6–12 weeks post partum to see if it is a non-gestational diabetes. And also, they have to be screened life long every 3 years for diabetes and prediabetes.

### *Methods of screening*

1. **Single step method** - (75 of glucose) - It has been recommended by American Diabetes Association (ADA, 2017) and the International Association of Diabetes and Pregnancy Study Groups (IADPSG, 2010). Diagnosis made according to ADA 2017 criteria single (1) value is sufficient for diagnosis) (Table 35.16). Diabetes in Pregnancy Study Group of India (DIPSI) and screening protocol for GDM recommended by Ministry of Health and Family Welfare, Govt. of India, 2018 and followed in most Indian hospitals is shown in Fig. 35.3B. A total of 75 gm glucose mixed in 200 ml water is consumed by the patient over 10-15 minutes. No fasting is needed. Minimum gap between two tests should be 4 weeks. If patient comes only at 29 weeks, do the test only once. If patient vomits in 30 minutes, call her next time to perform the test. If vomiting occurs after 30 minutes, continue the test.

   The advantages of this single step method are:

   (i) The pregnant woman need not be fasting
   (ii) It causes least disturbance in a pregnant woman's routine activities.
   (iii) It serves as both screening and diagnostic procedure.
   (iv) The pregnant woman may not turn up again if asked to come in fasting state the next day.

   **Disadvantage.** Over diagnosis

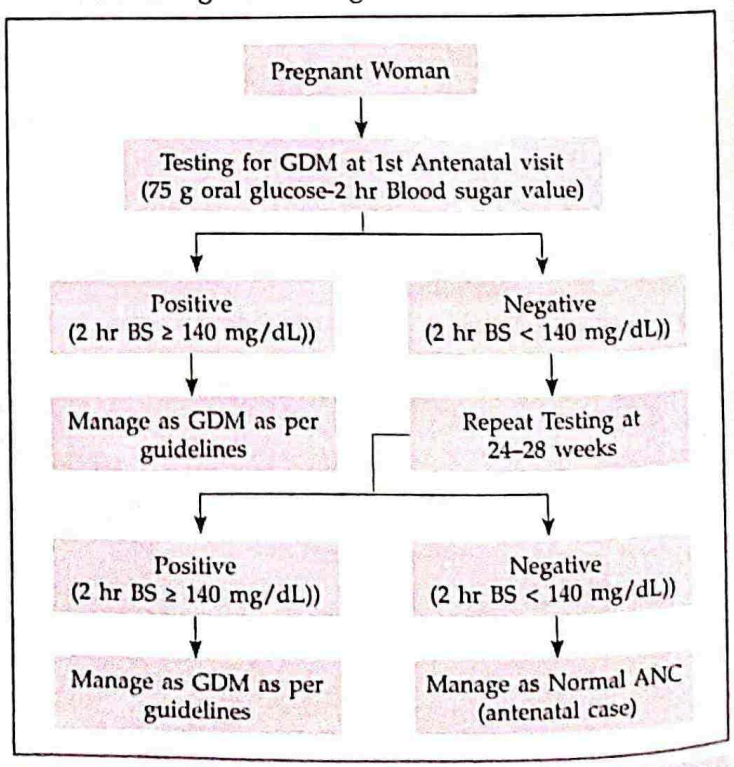



**Fig. 35.3B:** Screening Protocol (Ministry of Health and Family Wellare: February 2018)

2. **Two step (Double step) Method.** This method is recommended by ACOG (2017) and consists of the following two steps.
   (i) **Glucose Challenge Test (GCT)**
   Plasma glucose is measured one hour after 50 g glucose load without regard to the time of the day or the last meal. A value of < 140 mg/dL is taken as normal and 200 mg/dL or more is taken as overt diabetes. A value of plasma glucose of 140 mg/dL–199 mg/dL (or whole blood of 130 mg–190 mg/dL) at 1 hour will identify 80% women with GDM and is taken as cut-off point for glucose tolerance test.
   (ii) **Glucose Tolerance Test (GTT).** GTT is performed if screening test **(glucose challenge test)** shows reading between 140–199 mg/dL. The American College of Obstetricians and Gynecologists (2017) had recommended 100 g, 3 hour oral glucose tolerance test which was originally recommended by O'Sullivan and Mahan and was modified by Carpenter and Coustan. It is shown in Table 35.16. Diagnosis of GDM by various organization is also shown in Table 35.16. Two or more values should exceed the cut off for diagnosis of GDM. World Health Organization and DIPSI (Diabetes in Pregnancy Study Group, India) recommend 75 g, 2 hour oral glucose tolerance test which is often used in Europe and India (Table 35.16).

## Maternal and Foetal Effects of GDM

1. The incidence of congenital malformations in GDM is the same as in general population with no increased risk as disease manifests after organogenesis.
2. More than half of the women with GDM develop overt diabetes in the next 20 years.
3. Fasting hyperglycemia is associated with increased perinatal loss.
4. Increased incidence of foetal macrosomia is due to increase in insulin-like growth factors (IGF-I and II), epidermal growth factor, leptin, adiponectin and also due to maternal obesity.
5. Polyhydramnios
6. Birth trauma
7. Increased frequency of maternal hypertension
8. Increased incidence of cesarean section
9. Recurrence of GDM in subsequent pregnancies in about 50%.

## Management

The aim is to keep fasting plasma glucose at less than 95 mg/dL and the 2-hour postprandial plasma glucose at less than 120 mg/dL.

1. **Diet restriction** (1200–1800 Kcal/day) is recommended especially for obese women (body mass index > 30 kg/m$^2$). Other women should take 30 Kcal/kg/day (2000–2500 Kcal/day). The recommended daily calorie intake in pregnant women with GDM is shown in Table 35.17A. ACOG (2017) recommends carbohydrate intake of 40%, protein 20% and fats 40% of total calorie.
2. **Exercise.** Resistance exercises and walking may diminish need of insulin therapy in overweight women with gestational diabetes.
3. **Regular monitoring of blood sugar levels** is done as shown in Table 35.17B.
4. **Insulin therapy.** Insulin is the first line agent for persistent hyperglycemia in GDM. Human insulin therapy is initiated if fasting plasma glucose exceeds 105 mg/dL or 2 hours postprandial value exceeds 140 mg/dL despite diet therapy. A total dose of 20 to 30 units divided into 2/3rd morning dose (2/3rd intermediate acting insulin and 1/3rd short acting insulin) while rest 1/3rd insulin is given at night (1/2 intermediate, 1/2 short acting) is usually started with regular blood glucose monitoring.
5. **Oral hypoglycemic agents:** They are usually avoided in pregnancy. There is slight increased rate of neonatal admissions, respiratory distress and neonatal hypoglycemia with them. Starting dose of glyburide is 2.5 mg orally with morning meal, increased by 2.5 mg per week to 10 mg once a day. Later, it can be increased to a maximum dose of 10 mg twice daily (total 20 mg/day) beyond which insulin is started. Similarly, metformin given for polycystic ovarian disease can be safely continued in pregnancy. Although USA FDA has not approved glyburide and metformin for treatment of GDM, ACOG (2017) accept them as acceptable second line agents for management of GDM. They both are category B drugs.

**Table 35.16: Diagnosis of GDM using Oral Glucose Tolerance Test (OGTT) as per different organizations**

| Guidelines | Fasting PG mg/dl | Glucose challenge | 1 hr PG mg/dl | 2 hrs. PG mg/dl | 3 hrs PG mg/dl |
|---|---|---|---|---|---|
| WHO (2013) | 92–125 | 75 gms OGTT | 180 | 153–199 | Not required |
| Carpenter-Coustan and ACOG (2017) | ≥ 95 | 100 gms OGTT | ≥ 180 | ≥ 150 | ≥ 140 |
| NDDG | ≥ 105 | 100 gm OGTT | ≥ 190 | ≥ 165 | ≥ 145 |
| IADPSG (2010) | ≥ 92 | 75 gms OGTT | ≥ 180 | ≥ 153 | Not required |
| DIPSI (2006) | Not required | 75 gms OGTT | Not required | ≥ 140 | Not required |

DIPSI: Diabetes In Pregnancy Study Group, India; PG: Plasma glucose; ACOG: American College of Obstetricians and Gynecologists; WHO: World Health Organisation; IADPSG: International Association of Diabetes and Pregnancy Study Group; NDDG: National Diabetes Data Group.

**Table 35.17A: Calorie allotment based on BMI (ADA 2015)**

| BMI (kg/m²) | Calories intake kcal/kg/day | Weight gain (kgs) |
|---|---|---|
| 16.5–18.4 (under weight) | 35 | 11.4–15.9 |
| 18.5–24.9 (normal) | 30 | 11.4–15.9 |
| 25–30 (over weight) | 22–25 | 6.8–11.4 |
| > 40 (morbid obesity) | 12–14 | 7 |

**Table 35.17B: Self-Monitored Capillary Blood Glucose Goals**

| Specimen | Level (mg/dL) |
|---|---|
| Fasting | ≤ 95 |
| Premeal | ≤ 100 |
| 1-hr postprandial | ≤ 140 |
| 2-hr postprandial | ≤ 120 |
| 02 A.M.–06 A.M. | ≥ 60 |
| Mean (average) | 100 |
| Hemoglobin $A_{1c}$ | ≤ 6% |

However, they are still second line treatment as their long term effects on fetus are not known.

## Obstetric Management

Management protocol of GDM women with good diabetic control is shown in Fig. 35.4. Euglycemic women without insulin therapy can await spontaneous onset of labour. *However, they should not be allowed to go post date.* Women requiring insulin or with complications like macrosomia should be delivered at 38 weeks of gestation by induction of labour or by elective cesarean delivery (if estimated foetal weight is > 4.5 kg or for other obstetric indications). *Many hospitals in India deliver all GDM women at 38 weeks.* During puerperium any postpartum haemorrhage or infection should be taken care of. Low dose hormonal pills can be safely given.

Women with GDM should have a 75 g oral glucose tolerance test at 6 to 12 weeks puerperium and every three year intervals thereafter. Fasting hyperglycemia is more important indicator of development of overt diabetes later.

**Follow up.** *There is 50% likelihood of women with GDM to develop overt diabetes within 20 years.* They should be followed up for metabolic disorders or cardiovascular complications. *Recurrence risk in future pregnancies is about 40%. Lifestyle behaviour changes, weight control and exercises between pregnancies are used in reducing incidence of future GDM and overt diabetes.*

# PRE GESTATIONAL (OVERT) DIABETES MELLITUS IN PREGNANCY

Overt diabetes during pregnancy has significant adverse impact on maternal and perinatal outcomes which is closely related to the glycemic control and any underlying renal or cardiovascular disease.

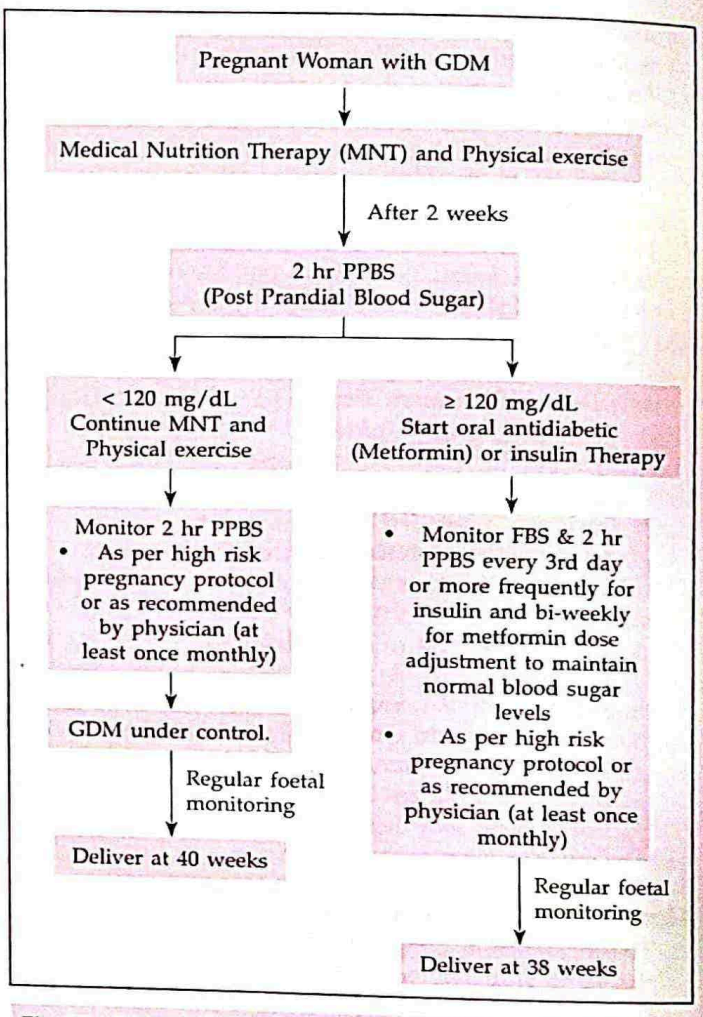



**Fig. 35.4:** Management of GDM (Ministry of Health and Family wellare: 2018, Govt. of India

## Diagnosis

According to WHO (2013), American Diabetes Association (ADA, 2017) and the International Association of Diabetes and Pregnancy Study Groups (IADPSG, 2010), diagnosis of pre gestational diabetes is made if glycosylated hemoglobin ($HbA_1C$) level is equal or more than 6.5 percent, fasting plasma glucose is equal or more than 126 mg/dL (7.0 mmol/L) or random plasma glucose or 2 hour postprandial glucose value is equal or more than 200 mg/dL (11.1 mmol/L) Overt diabetes is usually pre-existing, but may be diagnosed for the first time during pregnancy.

## Classification of diabetes mellitus in pregnancy

Classification of diabetes mellitus in pregnancy is given in Table 35.12. White's classification though complicated is useful in predicting maternal and perinatal outcome but is not very commonly used in clinical practice as its primary focus is on duration of disease and complications of long standing diabetes which are uncommon in pregnancy.

Women with overt diabetes who are in more advanced White's classification (like vasculopathy) are more likely to have maternal and perinatal complications like gestational hypertension, pre-eclampsia, hydramnios, ketoacidosis, foetal growth restriction (FGR) and intrauterine death (IUD).

## Effect of pregnancy on diabetes

As shown in Fig. 35.3A, pregnancy is a diabetogenic state and there is accelerated starvation during pregnancy. There is insulin resistance in pregnancy due to contra-insulin hormones (hPL, estrogen, progesterone, cortisol) and degradation of insulin by the placenta, maintenance of blood sugar during pregnancy is difficult. Insulin requirements are increased during pregnancy. Increased lipolysis due to hPL rises free fatty acid levels which may aid in increased insulin resistance. Due to insulin resistance, pregnant women are more prone to develop ketoacidosis during hyperemesis, infection, prolonged fasting and management of premature labour by the use of $\beta_2$ sympathomimetic drugs (for tocolysis) and glucocorticoids (for foetal pulmonary maturity). There may be worsening of vasculopathy like retinopathy, nephropathy, neuropathy during pregnancy and women with severe vasculopathy should be advised against pregnancy. There is dramatic decrease in insulin requirements during puerperium.

## Effect of Diabetes on Pregnancy

### A. Maternal Complications

The various maternal complications are shown in Table 35.18.

### B. Foetal Complications

Various foetal complications are as follows:

1. ***Increased spontaneous abortion rate*** especially in uncontrolled diabetes.
2. ***Congenital malformations.*** They are seen in overt (pregestational) diabetes as a consequence of poorly controlled diabetes (hyperglycemia) periconceptionally and during early pregnancy. In type I diabetes, the incidence of congenital anomalies is about 5% if their periconceptional glucose control was optimized but rose to 11% if the women presented after completion of organogenesis and is four fold higher than in normal pregnancies. Diabetes is not associated with increased risk of foetal chromosomal abnormalities. The various causes of congenital malformations could be hyperglycemia (which is fetotoxic and causes free radical injury to the fetus), genetic predisposition, ketoacidosis, increased oxidative stress markers (free oxygen radicals) and deficiency of arachidonic acid during periconceptional period and in early pregnancy. Various types of congenital malformations are shown in Table 35.19A. **Cardiac anomalies are most common.**

   **Diagnosis of congenital malformations**

   (a) Glycosylated hemoglobin ($HbA_1C$) estimation before 14 weeks reflects the diabetic control over the preceding 12 weeks. The prevalence of congenital malformation rate increases in proportion to $HbA_1C$ levels (Normal < 6%) as shown in Table 35.19B.

   (b) Estimation of maternal serum α-fetoprotein levels at 16 weeks (increased in neural tube defects)

   (c) Ultrasound scan in late first trimester may diagnose anencephaly. Detailed anomaly scan at 18 weeks for various congenital malformations and foetal echocardiography at 22–24 weeks of gestation (for cardiac anomalies) should be performed. Prevention of congenital malformations is by strict euglycemic control during periconceptional period and in early pregnancy.
3. **Foetal macrosomia.** It can be seen in 30–45% of all diabetic pregnancies. Foetal macrosomia and various other perinatal complications of diabetes can be explained by the **Pederson's (1977) hypothesis** shown in Fig. 35.5. The basic concept is that fetus is not diabetic and its pancreas responds to hyperglycemia by increased insulin secretion which is an anabolic hormone causing deposition of fat, glycogen and proteins. As the fetus receives continuous supply of glucose, amino acids and free fatty acids and there is foetal hyperinsulinemia and increase in insulin like growth factors (IGF-I and II), there is foetal macrosomia and adiposity. The incidence of macrosomia can be markedly reduced by tight diabetic control throughout pregnancy.
4. **Foetal or intrauterine growth restriction** (FGR or IUGR). It is only seen in diabetics with vasculopathy (about 5% cases).
5. **Intrauterine foetal death.** Diabetic mothers especially with uncontrolled diabetes are much more prone to have intrauterine foetal deaths. There can be sudden stillbirths due to placental insufficiency seen in overt diabetes with pre-eclampsia and in advanced diabetes with vascular complications and in maternal ketoacidosis. **Unexplained foetal deaths may occur suddenly in diabetic pregnancies due to hyperglycemia mediated chronic aberrations in transport of oxygen and foetal metabolites. They can also occur due to hyperglycemia induced hypoxia, ketoacidosis, lactic acidosis or hypoglycemia.** Risk of still birth is 1% while perinatal death rate is 1.7%. Tight control of diabetes can prevent most but not all cases of foetal death.
6. **Shoulder dystocia** with difficult delivery is more common due to disproportionate foetal growth and macrosomia.
7. **Foetal birth injuries** like brachial plexus injuries are more commonly seen in diabetic pregnancies.

### Neonatal Complications

Most neonatal complications are related to abnormalities in maternal glucose metabolism. Prevention of most perinatal complications is by proper control of maternal blood sugar levels. The various neonatal complications are as follows:

1. **Respiratory distress syndrome (hyaline membrane disease).** It was thought that foetal lung maturation is delayed in diabetic pregnancies due to inhibition of surfactant (especially phosphatidylglycerol) production by hyperinsulinemia making them more prone to have neonates with respiratory distress syndrome. However,

**Table 35.18: Maternal Complications in Overt Diabetes during Pregnancy**

| Maternal Complications During Pregnancy | Percentage (wherever available) | Causes and Treatment |
|---|---|---|
| 1. Miscarriages (spontaneous abortion) | | Uncontrolled diabetes, proper control of diabetes |
| 2. Infections especially urinary tract infections including pyelonephritis | | Lower body resistance. Glycosuria. Antibiotics. |
| 3. Vaginal candidiasis | | Poor body resistance. Proper control of diabetes. |
| 4. Polyhydramnios | 25–50% | Foetal polyuria due to hyperglycemia, macrosomia, large placenta, increased glucose in amniotic fluid causing increased osmosis. Proper control of diabetes. |
| 5. Gestational hypertension and Pre-eclampsia | 28% | Large placenta, Vasculopathy, Regular BP monitoring. |
| 6. Ketoacidosis | | Poor control. Proper control of diabetes. |
| 7. Worsening of nephropathy with or without renal failure | | Poor diabetes control. Proper control of diabetes. |
| 8. Worsening of diabetic retinopathy | | There can be hemorrhages, microaneurysms and proliferative retinopathy related to duration and severity of diabetes. Laser photocoagulation is treatment of choice. |
| 9. Preterm labour | 28% | Polyhydramnios and infection. Tocolytic agents and steroids should be used with caution |
| 10. Hyperglycemia | 20% | Poor diabetes control. Proper control of diabetes. |
| **During Labour** | | |
| 1. Ketoacidosis | | Poor diabetes control. Proper control of diabetes. |
| 2. Prolonged labour | | Big baby. Proper control of diabetes. |
| 3. Shoulder dystocia | | Macrosomia, Disproportionate growth with increased shoulder head ratio. Proper control of diabetes. Elective cesarean for macrosomia. |
| 4. Increased incidence of instrumental delivery | | Proper control of diabetes |
| 5. Increased incidence of operative delivery | | Proper control of diabetes |
| 6. Maternal soft tissue injuries, perineal tears, vaginal lacerations, cervical tears | | Large baby, shoulder dystocia, Proper control of diabetes. |
| 7. Postpartum haemorrhage | | Maternal soft tissue trauma, instrumental delivery, atonic postpartum haemorrhage. Proper control of diabetes. |
| **During Puerperium** | | |
| 1. Subinvolution of uterus | | Due to increased instrumental or operative delivery rate, maternal soft tissue injury, overdistended uterus due to macrosomia and hydramnios. Proper control of diabetes. |
| 2. Puerperal sepsis | | Due to increased operative and instrumental deliveries, maternal soft tissue injuries, poor resistance to infection. Proper control of diabetes. |
| 3. Failed lactation | | Poorly controlled diabetes. Proper control of diabetes. |

**Table 35.19A: Congenital Malformations in Overt Diabetes during Pregnancy**

| | |
|---|---|
| Cardiovascular defects (5 fold increase) Commonest malformation (> 58% of all anomalies) | 1. Endocardiac cushion defects: Common<br>(a) Ventricular septal defect **(most common anomaly in diabetes)**<br>(b) Atrial septal defect<br>2. Transposition of great vessels **(most specific cardiac anomaly in diabetes)**<br>3. Coarctation of aorta<br>4. Patent ductus arteriosus<br>5. Cardiomegaly<br>6. Situs inversus<br>7. Fallot's tetralogy<br>8. Anomalous pulmonary venous return |
| Nervous system (10 fold increases) | 1. Neural tube defects<br>(a) Anencephaly<br>(b) Open spina bifida<br>(c) Hydrocephalus<br>(d) Meningomyelocele<br>(e) Encephalocele<br>2. Microcephaly<br>3. Other central nervous system defects like holoprosencephaly, anotia and microtia |
| Skeletal Defects (250 fold increase) | Sacral agenesis and caudal regression syndromes: It is **most specific anomaly of diabetes** but is very rare (Fig. 35.6A and B). |
| Renal anomalies | 1. Renal agenesis<br>2. Cystic kidney<br>3. Duplex ureter |
| Gastrointestinal | 1. Duodenal atresia<br>2. Anal atresia<br>3. Rectal atresia<br>4. Small left colon syndrome |
| Miscellaneous | 1. Single umbilical artery.<br>2. Oro-facial defects.<br>3. Hypospadias. |

**Table 35.19B: Relationship between HbA1c level and foetal anomalies**

| HbA1c Levels | Risk of Congenital Foetal Anomalies |
|---|---|
| 6% | 3% |
| 8% | 6% |
| 10% | 20% |
| 12% | 35% |
| 15% | > 40% |

the current concept is that gestational age rather than overt diabetes is likely to be the most significant factor associated with neonatal respiratory distress.

2. **Hypoglycemia** (Blood glucose < 45 mg/dL [2 m mol/L]) due to hyperplasia of foetal β islet cells and related to maternal blood glucose (> 145 mg/dL) during labour. Neonatal gluconeogenesis is suppressed and the maternal nutrients are no longer available. The baby may be lethargic or may have convulsions.
3. **Hypocalcemia** (serum calcium < 7 mg/dL) can be due to prematurity, pre-eclampsia and abnormalities of magnesium and calcium metabolism.
4. **Hyperbilirubinemia.** It could be due to preterm birth or polycythemia with hemolysis.
5. **Hypomagnesemia**
6. **Hyperviscosity syndrome** due to polycythemia
7. **Hypertrophic cardiomyopathy** may occur in infants born to diabetic mothers and is usually seen in presence of foetal macrosomia and hyperinsulinemia. It usually resolves of its own.
8. **Transient tachypnea** of the new born (TTNB)
9. **Birth asphyxia** due to difficult labour and shoulder dystocia.
10. **Birth injuries** due to shoulder dystocia and difficult and prolonged labour.
11. **Long term cognitive development of infants** born is usually normal. Though some studies indicated slightly lower (1–2) intelligent quotient in them.
12. **Perinatal mortality.** The overall perinatal mortality is increased 2–3 times and is principally due to unexplained foetal deaths, shoulder dystocia, difficult labour and due to hypoglycemia, respiratory distress syndrome, polycythemia and jaundice in the neonatal period.
13. **Late effects and Inheritance of diabetes.** The infants born to diabetic mothers may have obesity in adulthood. They are also more prone to have type 2 diabetes. Risk of developing type I diabetes is 3–5% if either parent is affected while that of type 2 diabetes is 40% if both parents are affected.

## Management

### Prepregnancy (Preconceptional) Counselling

For optimum maternal and perinatal outcome in diabetic pregnancy, euglycemia should be maintained during periconceptional period and throughout pregnancy. It is important to have strict control of blood sugar in these women. They should be attended by an endocrinologist (diabetologist), obstetrician and dietician. They should conceive when their diabetes has been well under control. Periconceptional folic acid 400 µg per day is given to prevent neural tube defects and it should be continued in first trimester. Glycosylated Hb (Hb A1C) levels and blood sugar levels should be normal before planning pregnancy to prevent congenital malformations. Proper advice about diet and insulin is given to these women. Women controlled on oral hypoglycemic agents should be started insulin therapy for better outcome and to prevent congenital malformations. Women with polycystic ovarian disease who conceived on metformin should continue to take metformin throughout pregnancy for better maternal and perinatal outcome. The American Diabetic Association

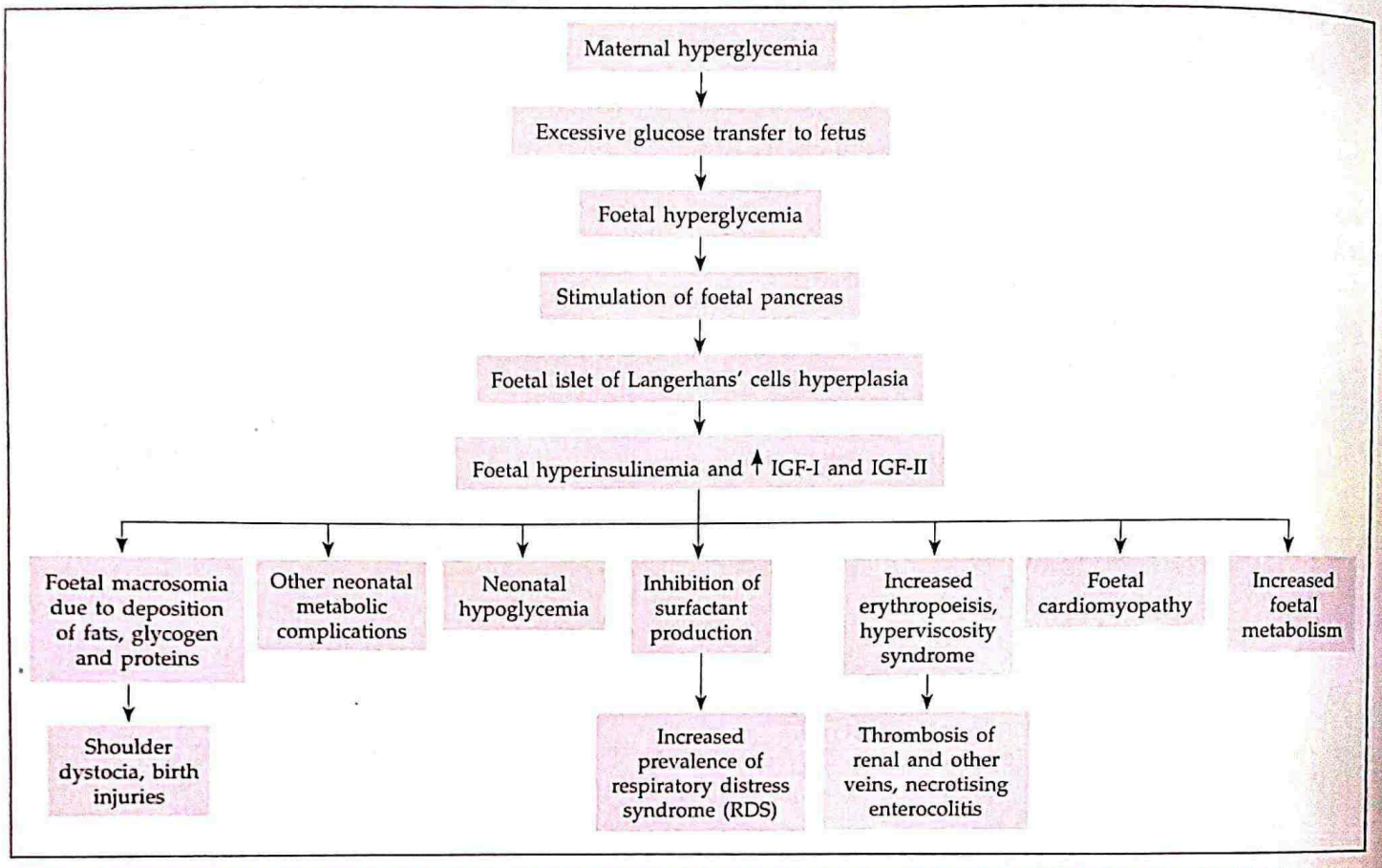



**Fig. 35.5:** Mechanism of foetal and neonatal effects in diabetes mellitus during pregnancy (modified from Pederson's hypothesis)

(1999) has recommended periconceptional glucose control using insulin to achieve fasting capillary plasma glucose between 80–110 mg/dL, 2 hour postprandial plasma glucose to be < 155 mg/dL.

### Aims of Management

1. Achievement of euglycemia during periconceptional period and throughout pregnancy.
2. Careful antenatal care throughout pregnancy .
3. To find out optimum time and mode of delivery and to avoid iatrogenic prematurity.
4. Avoiding maternal complications and their timely detection and management.
5. Foetal monitoring
6. Timely detection and management of foetal and neonatal complications.

### Antenatal Care

1. **Visits.** The women should preferably be seen every 4 weeks up to 20 weeks, then every 2 weeks until 30 weeks and weekly thereafter. Ideally, she should be seen in a diabetic antenatal clinic by an obstetrician with special interest in diabetes and an endocrinologist or physician with special interest in pregnancy with diabetes. Otherwise, she should be seen in routine antenatal clinic and in endocrine clinic regularly.
2. **Diet.** Dietery control is of paramount importance and a dietician of the hospital should give a diet chart to the patient. These women should be given frequent and detailed explanation concerning the need to follow a restricted meal plan during pregnancy to avoid wide fluctuations in blood sugar levels, which are detrimental to the fetus.

*Composition of Diet*

(a) Carbohydrates should make 55% of the calories, coming from complex carbohydrates. About 175–200 g, 3 + meal pattern per day should be consumed to compensate for urinary loss and foetal growth.
Diabetes meal Planner;
Plate Method
Half (1/2) plate: Non-starch vegetables
One fourth (1/4): Carbohydrates,
One fourth (1/4) Proteins plus water or calorie drink.

(b) Proteins should be given about 1.5 g/kg body weight per day (20% calories).

(c) Rest calories (25%) should come from fat out of which 10% should be saturated fat, which can be decreased if patient is obese.

Daily caloric requirements are about 30 calories per kg body weight. The total caloric intake should be split into three small meals and three–four snacks to minimize fluctuations in blood sugar levels.

3. **Education.** All diabetic women require re-education about their diabetes and its control. She is explained

about the utmost need of glycemic control to minimize the complications to her pregnancy and fetus by a tight control. Usually, the women can be taught to give their own injections and should be taught the importance of asepsis and need of using disposable syringes and needles each time. She should also be explained about the early symptoms of hypoglycemia and its immediate management by consuming some biscuits that should be in her bag all the time.

### History Taking

Careful history taking including any symptoms of diabetes and its complications and various complications of pregnancy should be asked each time patient visits antenatal clinic.

### General Physical Examination

A general physical examination should be done and pallor, pulse, blood pressure, jugular venous pressure, respiratory and cardiovascular system are checked at each visit. Fundus examination to rule out diabetic retinopathy.

### Abdominal examination

To confirm the period of gestation to look for the cause of increased fundal height more than the period of gestation (big baby, hydramnios), foetal presestation and foetal heart sounds

### Investigations

All investigations are not required for all cases.

1. Blood pressure on each visit
2. Weight on each visit including
   (a) Pre-pregnancy weight
   (b) Abnormal increase in weight during pregnancy
3. Complete hemogram including
   (a) Hemoglobin
   (b) Total leucocyte count and differential leucocyte count (to rule out infections)
   (c) Platelet count
4. Urine examination for:
   (a) Glucose
   (b) Proteins especially microalbuminuria (30–40 mg/day) (rule out nephropathy)
   (c) Specific gravity
   (d) Volume
   (e) Ketones
   (f) Microscopy (rule out infections)
   (g) Culture
5. Kidney function tests:
   (a) Blood urea nitrogen (BUN)
   (b) Serum creatinine
   (c) Uric acid
6. Hepatic function tests (in presence of gestational hypertension or pre-eclampsia):
   (a) Prothrombin time, activated partial thromboplastin time (APTT)
   (b) Transaminases
   (c) Bilirubin level
   (d) Lactate dehydrogenase
7. Fundus examination for any diabetic or hypertensive retinopathy.
8. Echocardiography if cardiac involvement in pregestational diabetes only (not in GDM).
9. Foetal evaluation:
   (a) Maternal serum α-fetoprotein levels (at 16 weeks gestation) are increased in neural tube defects. (Normally the values are less [80% of normal] in a diabetic patient)
   (b) Targetted ultrasonography (at 18–20 weeks gestation) should be used to evaluate foetal growth and well-being, estimate weight and detect hydramnios, neural tube defects and anomalies. It is repeated for foetal well-being and growth every 4 weeks.
   (c) **Foetal echocardiography.** Due to five fold increase in cardiac anomalies in pregnancies complicated by overt (pre-gestational) diabetes, a foetal anatomic survey including echocardiography is recommended at 22–24 weeks.
   (d) Foetal kick chart daily
   (e) Regular non-stress test
   (f) Biophysical profile
   (g) Doppler velocimetry
10. **Blood sugar estimations.** As urine sugar cannot be relied upon in pregnancy, the patient should measure fasting and 2 hours postprandial blood sugar levels daily using dextrostix strips or glucometer and record it on the chart which she should bring to her obstetrician on each visit, who may need to alter the type and dose of insulin. On each visit the doctor should get a proper venous or whole blood glucose tested in the hospital laboratory to rule out any fallacy in the tests performed by her. Glycosylated hemoglobin should also be tested which is a better indicator of diabetic control over preceeding few weeks. Target is to keep fasting blood sugar of < 95 mg/dl, pre-meal < 100 mg/dl 1, hr. postprandial < 140 mg/dl, 2 hr PP < 120 mg/dl and HbA1C of < 6%.
11. **Insulin therapy.** Pharmacological effects of different insulins are given in Table 35.20.
    Usually combination of short acting insulin (30–50%) and long acting insulin (50–70%) is given in 2–3 doses per day.
    No regimen for insulin administration is applicable to all diabetic pregnant patients. Many of them can achieve control with two doses of a combination of short and intermediate acting insulin. Some women, however, require 3 or more daily doses.
    ***Indications of insulin therapy***
    (a) All type 1 diabetes patients
    (b) Gestational diabetes not controlled by diet alone.
    (c) Type 2 diabetes patients who were on oral hypoglycemic agents before pregnancy.
    ***Principles***
    (a) There are frequent changes in needs of insulin during pregnancy.
    (b) Changes in dose are made in small increments of 2 units at a time.

**Table 35.20: Pharmacological Effects of commonly used Subcutaneous Insulins**

| Types of insulin | Onset | Peak of action (hours) | Duration of action (hours) |
|---|---|---|---|
| I. Short Acting | | | |
| 1. Lispro | < 15 min. | 0.5–1.5 | 3–4 |
| 2. Aspart | < 15 min. | 0.5–1.5 | 3–4 |
| 3. Glulisine | < 15 min. | 0.5–1.5 | 3–4 |
| 4. Regular | 30–60 min. | 2–3 | 4–6 |
| II. Long Acting | | | |
| 1. Detemir | 1–4 hr. | Minimal peak | Upto 24 |
| 2. Glargine | 1–4 hr. | Minimal peak | Upto 24 |
| 3. NPH | 1–4 hr. | 6–10 | 10–16 |

(c) Usually a combination of short acting and long acting insulin is given twice a day. The morning dose of regular insulin is determined by glucose levels 2 hours after breakfast, while evening dose is determined by blood sugar levels after evening meal or bed time. The morning dose of long acting insulin is reflected in blood sugar after lunch and before dinner, while evening dose is reflected in fasting (morning) blood sugar or presence or absence of nocturnal hypoglycemia.

12. All routine antenatal tests are done and prescription is given for iron, calcium and proteins. One should be careful about urinary tract infections and moniliasis (candidiasis) which are more common in diabetic pregnancies and must be treated at the earliest.
13. Hospitalization is required:
    (*i*) If diabetes is not properly controlled at home at any time.
    (*ii*) At about 36 weeks to investigate for stabilization of diabetic control and to decide the mode and timing of delivery.

## Management of Complicated IDDM

1. **Diabetic Nephropathy.** These patients have proteinuria and hypertension in first or second trimester and most of them have increase in proteinuria, blood pressure and serum creatinine. They have superimposed pre-eclampsia and usually have diabetic retinopathy. Foetal effects are prematurity and growth retardation and sometimes-foetal death. These patients ultimately develop end stage renal disease in few years and require renal transplantation. They should be discouraged from conception.
2. **Diabetic retinopathy.** It is also a valid indication for medical termination of pregnancy (MTP). Pregnancy accelerates progression of retinopathy and about 20% of them have proliferative retinopathy which may bleed and can cause sudden visual loss and retinal detachment especially in labour, which is therefore contraindicated in them. Laser treatment is usually required.

### Diabetic ketoacidosis

It can rarely occur and is a medical emergency with blood sugar upto 380 mg/dL, ketonemia, ketonuria and acidosis (pH < 7.3 and low serum bicarbonate < 15 mEq/L).

**Investigations.** Obtain arterial blood gases for degree of acidosis, measure glucose, ketones, and electrolyte levels at 1- to 2-hour intervals.

**Treatment of Diabetic Ketoacidosis.**

1. **Insulin** is the mainstay of treatment, Low-dose, intravenous regular insulin is started with loading dose of 0.2–0.4 U/kg followed by maintenance dose of 2–10 U/hr.
2. **Intravenous fluids.** Isotonic sodium chloride or Ringer lactate is given. Total replacement in first 12 hours of 4–6 L, 1 L in first hour, 500–1000 mL/hr for 2–4 hours, 250 mL/hr until 80 percent replaced.
3. **Intravenous glucose:** Begin 5-percent dextrose in normal saline when glucose plasma level reaches 250 mg/dL (14 mmol/L).
4. **Intravenous potassium:** If initially normal or reduced, an infusion rate upto 15–20 mEq/hr may be required; if elevated, wait until levels decrease to the normal range, then add to intravenous solution in a concentration of 20–30 mEq/L.
5. **Intravenous Bicarbonate:** Add one ampule (44 mEq) to 1 L of 0.45 normal saline if pH is < 7.1.

## Management of Labour in a Diabetic Woman Receiving Insulin

Nowadays, with stress on good glycemic control and foetal surveillance to detect uteroplacental insufficiency, more and more pregnant diabetics are allowed to reach term and go into spontaneous labour. Patients with vascular disease are delivered early if hypertension worsens or if there is foetal growth restriction (FGR). Factors influencing the timing of delivery are control of diabetes, condition of cervix, previous obstetric history and foetal compromise. Aim is to have vaginal delivery. Delivery is planned at 38 weeks to avoid unexplained intrauterine deaths.

## Indications of Cesarean Delivery

Cesarean delivery rate is about 80% in pregestational diabetes. Various relative indications are:

1. Malpresentations
2. Proliferative retinopathy
3. Pregnancy complicated by pre-eclampsia
4. Macrosomia (estimated foetal weight > 4.5 kg)
5. Previous cesarean
6. Foetal distress prior to or during labour
7. Bad obstetric history in elderly patient
8. Elderly primigravida
9. $HbA_{1C}$ > 6.4%

There should be a proper plan of mode and timing of delivery during antenatal period. The majority of

patients are induced or receive a planned elective cesarean delivery. Induction of labour is usually by prostaglandin gel or oxytocin drip and artificial rupture of membranes. Following protocol is used:

1. Any patient on a long acting insulin, should receive the dose of insulin, the night before expected delivery and be admitted to the ward an evening prior to delivery.
2. When a patient on insulin is in labour. Skip (with held) morning dose of insulin. The treatment is shown in Table 35.21.

   **Monitoring**

   **Blood Sugar** Latent phase every 2 hour
   Active phase every 1 hour

   **Urine-Sugar** Ketones every 4 hour
5. Antibiotics should be given prophylactically to minimize infection.

**Table 35.21: Insulin Management during Labour and Delivery (ACOG 2018) recommends following regimen**

| | | |
|---|---|---|
| 1. | Morning dose of insulin is skipped (withheld) | |
| 2. | Start intravenous infusion of normal saline | |
| 3. | In active labour or when blood sugar levels drop to 70 mg/dl, 5% dextrose drip is started at rate of 100–150 ml/hour with aim to keep blood glucose level of about 100 mg/dl | |
| 4. | Blood glucose levels are checked every hour with bedside glucometer to adjust dose of insulin and dextrose | |
| 5. | Regular insulin is given by intravenous infusion using pump at rate of 1.25 U/hr if glucose levels are more than 100 mg/dl | |
| 6. | Older sliding scale is less used now | |
| | Blood glucose | Insulin dose |
| | < 100 mg/dl | No insulin |
| | 100–140 mg/dl | 1 U/hr (4 units of insulin in 1 litre of 5% dextrose at 32 drops/ minute) |
| | 140–180 mg/dl | 1.5 U/hr (6 units insulin in 1 litre of normal saline at 32 drops/minute) |
| | 180–220 mg/dl | 2 U/hr (8 unit insulin in 1 litre of normal saline at 32 drops/minute) |
| | > 220 | 2.5 U/hr (10 unit insulin in 1 litre of normal saline at 32 drops/minute) |

6. In labour, continuous cardiotocography, if available, should be performed, otherwise foetal heart should be regularly heard with stethoscope.
7. Active management of labour is encouraged.
8. Instrumental delivery may be required.
9. Beware of shoulder dystocia
10. Senior obstetrician should be available to conduct delivery.
11. At delivery of the placenta, the insulin infusion rate should be halved in women who were diabetic pre-pregnancy and intravenous insulin and dextrose is continued until the mother eats; the pre-pregnancy insulin regime may then be resumed.
12. In gestational diabetes, insulin may be stopped after delivery. Her blood sugar should be checked 2–4 hourly for 24 hours.
13. **Care of newborn**
    (a) A neonatologist or pediatrician must attend to the newborn. The newborn should be transferred to nursery for 48 hours to manage any neonatal complications at the earliest.
    (b) The baby may be macrosomic and plethoric in poorly controlled diabetes (Fig. 35.6). The baby is carefully examined for Apgar score, any asphyxia and congenital malformations. Any asphyxia is energetically treated.
    (c) Baby's blood sugar should be done at birth, 1 hour, 6 hours, 12 hours. 24 hours, 48 hours and 72 hours as neonatal hypoglycemia is very frequent in them.
    (d) Early and more frequent breastfeeding is recommended.
    (e) Vitamin $K_1$ 1 mg is given intramuscularly.
    (f) Any neonatal complications like hypocalcemia, hypermagnesemia, hypokalemia, etc. should be taken care of by the neonatologist. Perinatal mortality has reduced significantly in recent years due to better management of diabetes during pregnancy and better neonatal care.

## Contraception

All diabetic women must be counselled about contraception. Barrier method is safe and ideal. The low dose combined oral contraceptive pill, injectable progestogens and IUCD can be used in women who had GDM. However, in overt diabetes, low dose combined pill must be used with caution (WHO category 3), IUCD was avoided for fear of infection in the past but WHO now recommends it (WHO category I). Progestogen only pill can be given. Permanent method (sterilization) is done when family is completed.




**Fig. 35.6:** (A) Trans abdominal Ultrasound showing fetus with caudal regression syndrome; (B) Trans abdominal 3D ultrasound showing fetus with caudal regression syndrome. *Kind courtesy: Dr Ashok Khurana, Delhi* . (C) Macrosomic and plethoric baby weighing 4.5 kg born to a diabetic mother by cesarean section

## Key Points

1. Gestational diabetes is a common medical complication of pregnancy and requires screening of high risk cases for early identification and effective control. It usually does not cause increase in congenital malformation rate.
2. Poorly controled GDM can cause significant perinatal morality (up to 4 times). However, with tight control of glucose levels during pregnancy which may require insulin therapy, the perinatal outcome is excellent.
3. Principles of management are tight glycemic control, antepartum foetal surveillance, appropriate timing of delivery, good neonatal care and future counseling. Umbilical Doppler velocimetry is of no proven value in monitoring of fetuses. Beware of shoulder dystocia especially after 39 weeks of gestation and in poorly controlled diabetes.
4. For overt diabetes which is uncommon (1–2% of all pregnancies), management should start pre-conception with good control of blood sugar on insulin. Such women should be managed by a team of obstetrician, endocrinologist (diabetologist), dietician and neonatologist.
5. Overt diabetes is associated with increased foetal congenital malformation rate especially of central nervous system and cardiovascular system with incidence being higher with poor sugar control and high HbA1C levels (> 8.5%) in periconceptional period.
6. Strict euglycemic control with insulin is the aim for optimum maternal and perinatal outcome by keeping fasting glucose less than 100 mg/dL and post prandial less than 140 mg/dL and a HbA1C to be less than 6.5%. A combination of insulin types is better than single insulin type for better outcome.
7. Foetal surveillance by regular ultrasound for foetal growth, non-stress test, biophysical profile is recommended.
8. Usually overt diabetics are delivered at 38 weeks of gestation. Beware of shoulder dystocia. Elective cesarean delivery for macrosomia and other obstetric complications is recommended. All neonates must be monitored for complications like hypoglycemia.

## Suggested Reading

1. American Diabetes Association: Classification and diagnosis of diabetes, Diabetes Care 2017; 40 (1 Supp.): S005
2. American Diabetes Association: Standards of Medical Care in diabetes. Diabetes Care: 2017; 40 (Suppl.): S114.
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# (iv) THYROID AND OTHER ENDOCRINE DISORDERS DURING PREGNANCY

## THYROID DISORDERS

After diabetes mellitus, thyroid disorders are the most common endocrine disorders during pregnancy. The various physiological changes in thyroid during pregnancy are discussed in Chapter 7.

Thyroid hormones are essential for normal foetal brain development and from 12 weeks onwards foetal thyroid produces its own $T_3$ and $T_4$ as maternal TSH does not cross the placental barrier. But during 3rd trimester also, 30% of foetal thyroxine comes from mother.

### Thyroid Function Tests in Pregnancy

The thyroid function tests should be carefully inferred in pregnancy as shown in Table 35.22. Total T3 and T4 increase in pregnancy due to stimulation of thyroid tissue by hCG but there is no change in free T3 and T4 levels due to increase in TBG causing transient fall in TSH in early pregnancy which becomes normal in second and third trimester.

## HYPERTHYROIDISM

Hyperthyroidism occurs in about 1 in 500 pregnancies and is usually (in 95% cases) due to autoimmune thyrotoxicosis (Graves' disease). Rarely women with hyperemesis gravidarum and molar pregnancy may present with symptoms of hyperthyroidism as high levels of hCG may stimulate the TSH receptors. Graves' disease is associated with a hyperplastic goitre and exophthalmos. It is due to thyroid-stimulating autoantibodies or, immunoglobulins which attach to thyrotropin receptors causing their activation resulting into thyroid hyperfunction and growth.

**Table 35.22: Inference of Thyroid Function Tests in Pregnancy**

| Thyroid function in Pregnancy | Inference |
|---|---|
| ↑ Total $T_4$<br>↑ Total $T_3$<br>Normal free (F) $T_4$<br>Normal TSH | Normal in pregnancy |
| ↓ $FT_4$ (mild)<br>↑ TSH (mild) | Normal in third trimester<br>Mild hypothyroidism |
| Normal free $T_4$<br>↑ TSH | May be normal feature in first trimester<br>May represent compensated hypothyroidism<br>Treated hypothyroidism in a non-compliant patient |
| ↑ $FT_4$<br>↓ TSH | May be associated with hyperemesis thyrotoxicosis |
| ↓ TSH<br>↓ $FT_4$ | Pituitary failure or hypothalamic failure<br>Hypothyroidism or non-thyroidal illness |
| Normal free $T_4$<br>↓ TSH | Treated thyrotoxicosis possibly in an intermittent compliant patient. May be a normal feature in first trimester. |

$T_3$: Tri-iodothyronine; $T_4$: Thyroxine, TSH: Thyroid stimulating hormone

### Clinical Features

Many clinical findings of normal pregnancy may simulate hyperthyroidism making diagnosis difficult. However, tachycardia, elevated (> 100/minute) sleeping pulse rate, thyromegaly, exophthalmos, oncholysis and failure to gain weight despite good appetite are features suggestive of hyperthyroidism. Diagnosis is by estimation of serum free $T_4$ and $T_3$ which are elevated and low TSH. Subclinical hyperthyroidism is characterized by abnormally low thyrotropin (TSH) concentration with normal thyroid hormones and may be seen in 1% patients.

### Effects of Hyperthyroidism on Pregnancy

The maternal and foetal effects of hyperthyroidism are shown in Table 35.23.

### Treatment

1. **Medical treatment** with antithyroid drugs is the mainstay of treatment and involves blocking thyroid hormone synthesis. The following drugs are given.
   (a) **Propylthiouracil (PTU) is the drug of choice for its safety and efficacy.** It reduces the titre of TSH receptor antibodies thus directly influencing the etiology of Grave's disease. It inhibits $T_4$ synthesis by blocking the incorporation of iodine into tyrosine, but also by inhibiting the peripheral

**Table 35.23: Maternal and Foetal Effects of Hyperthryoidism during Pregnancy**

| Maternal effects | Foetal effects |
|---|---|
| 1. Infertility<br>2. Abortion<br>3. Preterm labour<br>4. Increased risk of pre-eclampsia<br>5. Cardiac failure (8%) due to dysfunction of cardiac muscles and increased blood volume precipitated by pre-eclampsia, anaemia and sepsis.<br>6. Thyroid storm: It is an acute life threatening hypermetabolic state in women with thyrotoxicosis.<br>7. Death can occur in uncontroled thyrotoxicosis especially due to cardiac failure or thyroid storm. | 1. Abortion<br>2. Foetal growth restriction<br>3. Stillbirth<br>4. Neonatal mortality<br>5. Foetal thyrotoxicosis<br>6. Foetal hypothyroidism<br>7. Foetal goiter<br>8. Perinatal mortality<br>9. Nonimmune hydrops |

conversion of $T_4$ to $T_3$. PTU is preferred in first trimester as it is non teratogenic. However in second and third trimester, carbimazole or methimazole are preferred as PTU can rarely cause liver failure in later pregnancy.

Dose: 300–450 mg daily (maximum 600 mg) is given initially till patient becomes euthyroid followed by 50–150 mg per day (maintenance dose).

(b) Carbimazole and methimazole can cause increased congenital malformations in the fetus like esophageal and choanal atresia and aplasia cutis (methimazole embryopathy). Both PTU and carbimazole are equally beneficial and both cross the placenta. The dose of carbimazole is 20–60 mg orally daily till patient become euthyroid followed by 5–15 mg daily maintenance dose. In 1st trimester change carbimazole and methimazole to PTU to prevent embryopathy (ATA-2017). In 2nd trimester PTU can be changed back to Methiamazole or PTU can be continued throughout pregnancy.

The principles of treatment is to control thyrotoxicosis optimally with the lowest dose of antithyroid medication. Women should be clinically euthyroid with free $T_4$ at upper end of normal range.

Both the drugs can cause transient leucopenia (10%) which does not require stoppage of therapy but may rarely (0.3%) cause agranulocytosis which is serious and requires cessation of therapy. The women are counseled to stop medicine if they develop fever or sore throat and report to hospital for a compete blood count.

During medical treatment $T_3$, $T_4$, TSH levels should be done every 4–6 weeks. Both drugs are expressed in breast milk, but have little effect on foetal thyroid function, so are safe in lactation. Beta blockers (Propranolol) are given for tachycardia and arrythmias.

2. **Surgical treatment** in the form of thyroidectomy is seldom required for few women who are non-compliant to medical treatment or where drug therapy proves toxic. Even after surgery, women with Grave's disease need monitoring as they still have antibodies.
3. **Radioactive iodine therapy is contraindicated in pregnancy as it may cause foetal thyroid gland destruction.** If any woman is given this therapy she should not conceive for 6 months to avoid chromosomal damage in fetus. Inadvertent exposure to radioactive iodine in pregnancy warrants medical termination of pregnancy. It leads to foetal thyroid ablation after 10–12 weeks. Radioactive iodine is an absolute contraindication during breastfeeding and should be done 3 months after breastfeeding cessation.
4. **Management of thyroid storm and cardiac failure.** Treatment of both conditions is similar. Patient is admitted to intensive care unit (ICU). Propranolol is drug of choice 40–120 mg 6–8 hourly. A high dose of propylthiouracil (up to 1 g) is given orally followed by 200 mg every 6 hours. Iodine is given 1 hour after initial propylthiouracil dose to inhibit thyroidal release of $T_3$ and $T_4$ as 500–1000 mg of sodium iodide (weigh against risk of iodism) intravenously every 8 hours or 10 drops of Lugol's iodine orally every 8 hours. Steroids (Dexamethasone) 2 mg intravenously every 6 hourly is recommended by most authorities or Hydrocortisone 100 mg Intravenous 8 hourly.

### Foetal Hyperthyroidism

Maternal thyroid antibodies (IgG) cross the placenta and can cause foetal or neonatal thyrotoxicosis. As foetal thyrotoxicosis may cause pre-mature delivery, hydrops fetalis, foetal death, foetal goitre, polyhydramnios or obstructed labour (face presentation), foetal treatment is required. An ultrasound can demonstrate foetal goiter (Fig. 35.7). Maternal administration of antithyroid drugs also treats fetus as both propylthiouracil and carbimazole cross the placenta. Foetal heart rate monitoring can be used as a method to assess foetal thyrotoxicosis. Alternately, if pregnancy is advanced, delivery followed by treatment of neonate with antithyroid drugs is the treatment of choice. The newborn should be carefully observed by the neonatologist for any features of thyrotoxicosis and should be energetically treated. The baby may have poor feeding, weight gain and sleeping. There may be a goiter which can cause problems with breathing and feeding.

## HYPOTHYROIDISM DURING PREGNANCY

Hypothyroidism occurs in about 1% (0.3–2.5%) of pregnant women and is usually due to autoimmune **Hashimoto's thyroiditis (most common cause of hypothyroidism in pregnancy)** or idiopathic myxedema. It can also occur following surgical or radioactive iodine treatment of Grave's disease or due to dietary iodine deficiency.

As hypothyroidism is associated with subfertility, it is rare to make a new diagnosis in pregnancy. The diagnosis may be difficult as classical symptoms of hypothyroidism

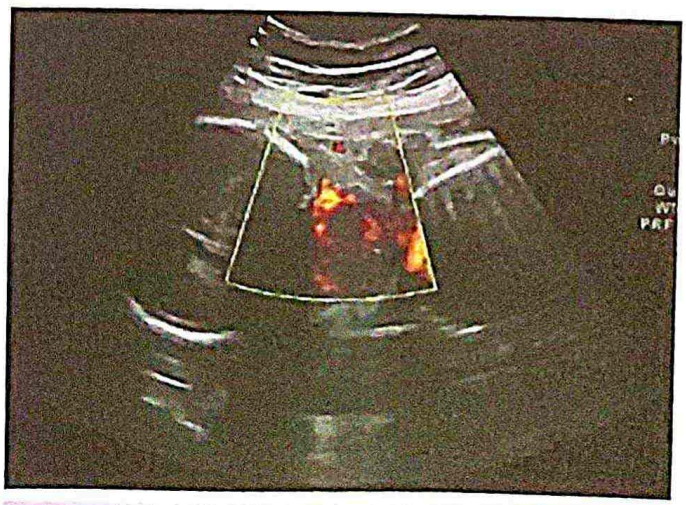


**Fig. 35.7:** Maternal ultrasound showing foetal goiter in a mother with Grave's disease. Kind courtesy: Dr Ashok Khurana, Delhi

such as fatigue, constipation, anaemia, weight gain, hair changes and carpal tunnel syndrome are commonly seen during normal pregnancy also. Diagnosis of clinical (overt) hypothyroidism is made by abnormally high serum thyrotropin level (TSH) accompanied by abnormally low thyroxine level (free $T_3$ and $T_4$). Subclinical hypothyroidism is defined as abnormally high serum thyrotropin (TSH) level with normal thyroxine level. Women with hypothyroidism should be euthyroid before conception to avoid impairment in the neonate.

## Effect on Pregnancy

Maternal and foetal effects are shown in Table 35.24.

**Table 35.24: Maternal and Foetal Effects of Hypothyroidism during Pregnancy**

| Maternal Effects | Foetal Effects |
|---|---|
| 1. Infertility and increased miscarriage rates. | 1. Abortions |
| 2. Anaemia | 2. Stillbirths |
| 3. Pre-eclampsia (31% in over hypothyroidism, 16% in subclinical hypothyroidism) | 3. Foetal growth restriction |
| 4. Abruptio placentae (8%) | 4. Prematurity |
| 5. Cardiac dysfunction | 5. More chances of admission to nursery |
| 6. Postpartum haemorrhage | 6. Subnormal mental development of the neonate |

## Routine Screening

Although subclinical hypothyroidism can cause adverse foetal outcome and adverse neuropsychological development in the neonate, routine screening is not yet practised. It may be considered in high endemic areas like in India. However, iodine supplementation is recommended in endemic iodine deficient areas through universal salt iodization. As India lies in endemic best, routine ANC screening is done (ATA-2017)

If on screening the TSH > 4 mIU/L, treatment with levothyroxine is required (American Thyroid Association [ATA] 2017).

## Treatment

### Overt Hypothyroidism

1. if patient was previously taking levothyrod then increase dose by 30%
2. All overt hypothyroid with TSH > 2.5 mIU/L should be treated.
3. Start levothyrodine as 1.6–2.0 µg/hg/d
4. Maintain target TSH ≤ 2.5 mU/L

### Subclinical Hypothoroidism

**LT4 is recommended for**

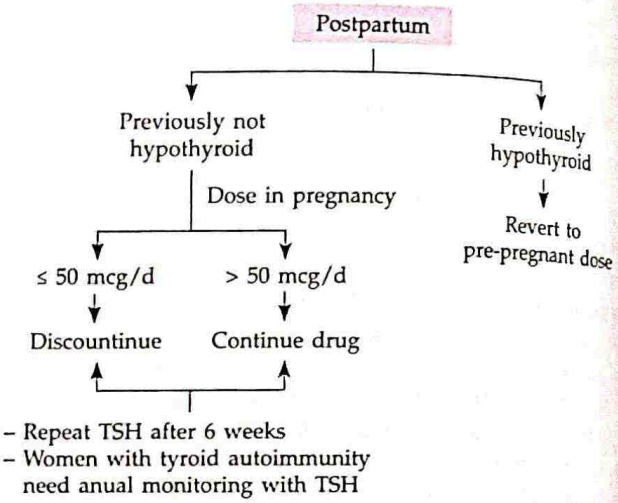
- TSH > 10 mIU/L
- TPOAb, TSH 4–10 mU/L positive

**LT4 can be considered for**

- TPOAb, TSH, 4–10 mIU/L negative
- TPOAb, TSH 2.5–4 MU/L positive

### Monitoring

Every 4 weekly until midgestation (16–20 weeks) → and atleast once nar 28 weeks pOG




## Congenital Hypothyroidism

As clinical diagnosis of hypothyroidism in neonates is usually missed and it is a preventable cause of mental retardation; newborn mass screening for congenital hypothyroidism (1 : 4000) is recommended. It is done by measuring TSH levels (> 80 mu/L) followed by replacement therapy to restore normal intelligent quotient (IQ). Babies with congenital hypothyroidism may be larger for dates with feeding and respiratory difficulties (due to goiter). There may be distension of the abdomen, vomiting, prolonged jaundice, hypotonia, hypothermia, hoarse cry, dry skin, coarse face with large tongue (macroglossia), umbilical hernia, constipation and anaemia in these babies.

Vigorous treatment using thyroxine is recommended.

## Postpartum thyroiditis

Postpartum thyroiditis is an autoimmune thyroiditis with thyroid antiperoxidase antibodies usually occurring during the first year after child birth (1–8 months) in 5–15% women. The woman presents with small, painless goiter with fatigue and palpitations. Antithyroid drugs are ineffective. For severe symptoms beta blockers may be given. About two-thirds of these women become euthyroid while one-third develop hypothyroidism. They require thyroxine replacement therapy.

## Miscellaneous thyroid conditions

For nodule in thyroid during pregnancy, management is like in non-pregnant state except that radioactive iodine scanning is contraindicated. Ultrasound and fine needle aspiration should be used. For thyroid nodule and thyroid carcinoma, surgery can be safely performed in the second trimester (before 24 weeks). In the third trimester it may be delayed to after delivery as most thyroid cancers are well differentiated and pursue a slow course.

# OTHER ENDOCRINE DISORDERS DURING PREGNANCY

## 1. Diabetes Insipidus (DI)

Diabetes insipidus is due to the deficiency of antidiuretic hormone which is rarely seen in pregnancy and is characterized by polyuria and polydipsia. The diagnosis is confirmed by water deprivation test. The drug of choice is desmopressin (1-deamino-8D-arginine vasopressin; [DDAVP]) which is safe in pregnancy and lactation. Plasma electrolytes, fluid intake and urinary output should be monitored.

Labour and delivery are usually uneventful with occasional uterine atony and postpartum haemorrhage.

## 2. Pituitary diseases

(a) **Pituitary adenomas.** They are benign neoplasms which are divided into microadenomas (less than 10 mm) or macroadenomas. They can also be classified according to the hormone they secrete, the 'prolactinomas' being most common. The symptoms may be galactorrhoea, amenorrhoea, hirsuitism, headache or visual field defects. The diagnosis is confirmed by MRI or CT imaging of the head. If there is enlargement of adenoma during pregnancy, bromocriptine or cabergoline therapy is started and visual field testing in each trimester and continued throughout pregnancy. If treatment for a few days does not show improvement, dexamethasone 4 mg, 6 hourly is indicated. If there is no response with the medical therapy, surgery is required.

Radiation therapy is usually contraindicated. Its only indication is post surgical residual cases in order to prevent recurrences.

Breastfeeding is not contraindicated in patients with prolactinomas. The patient should be evaluated for any tumor growth and bromocriptine therapy or cabergoline is restarted if there is persistent hyperprolactinemia.

(b) **Acromegaly.** It is caused by an excess of growth hormone (acidophilic or chromophilic pituitary adenoma). Pregnancy is rare in them.

Symptoms may be due to local expansion of the tumor, headache, visual field disturbances and facial changes. The diagnosis is made by an increase in the level of growth hormone and insulin-like growth factor-I (IGF-I). There is increased risk of developing gestational diabetes.

Treatment includes surgery and or radiation or medical therapy with somatostatin analogues. The newer agents **octreotide** and **lanreotide** have been tried in pregnancy.

**Postpartum hypopituitarism (Sheehan's syndrome)** is directly related to hypovolemia due to obstetric haemorrhage accompanied by vasospasm of the pituitary vasculature. There is a loss of anterior pituitary hormones but posterior pituitary hormones are usually spared. The patient after recovering from the postpartum haemorrhage presents with failure to lactate due to breast atrophy, prolonged postpartum amenorrhoea, loss of pubic and axillary hair, hypothyroidism and persistent hypotension and hypoglycemia . Sella turcica is totally or partially empty on CT scan. They need complete replacement of these hormones (Glucocorticoid replaced first).

More severe form of hypopituitarism involving total pituitary necrosis and failure is called **Simmond's disease** characterized by absence of gonadal function, hypothyroidism and hypoadrenalism.

## 3. Diseases of Adrenal Glands

(a) **Cushing Syndrome.** The most common cause in women is bilateral adrenal hyperplasia but can be due to an adrenal tumor or ectopic ACTH production or pituitary adenoma.

Patient presents with amenorrhea or oligomenorrhea, weight gain, hirsutism, personality changes or muscle weakness. Maternal complications like hypertension, diabetes mellitus and heart failure are common during pregnancy. Diagnosis is by finding elevated plasma cortisol levels without diurnal variation, not suppressed with dexamethasone. CT scan may be required if adrenal cancer is suspected. Determination of 24 hours urinary free cortisol is the best screening test to rule out Cushing syndrome in pregnancy. It is associated with an increased incidence of miscarriages, preterm labour and stillbirth.

**During pregnancy, Definitive treatment is surgical.** Adrenal surgery in the presence of tumor (adrenal, pituitary) in the 1st and 2nd trimesters may be tried. In the third trimester, drug therapy with ketoconazole or metyrapone should be considered.

(b) **Addison's disease.** Primary adrenal insufficiency (Addison's disease) is rare and results when destruction of at least 90% adrenal cortex has taken place. The causes may be autoimmune, granulomatous lesions like TB, other infections, bilateral adrenalectomy, metastatic tumors, etc.

The signs and symptoms include fatigue, weight loss and pigmentation. A plasma cortisol level of less than 20 mg/L is consistent with Addison disease.

Treatment is intravenous hydrocortisone 100 mg 6 hourly, initially. Long-term replacement consists of 12–15 $mg/m^2$ per day of hydrocortisone and fludrocortisone acetate 100 g per day for mineralocorticoid activity. Additional cortisol replacement is recommended during periods of major stress, e.g. surgery, labour and delivery.

(c) **Congenital Adrenal Hyperplasia (CAH).** The enzyme deficiency of 21 hydroxylase (rarely 18 hydroxylase) results in a decrease in cortisol production, leading to increased ACTH stimulation with increase in androgenic cortisol precursors and decrease in production of aldosterone. The androgenic steroids cross the placenta and can cause foetal virilization. This risk is reduced if patients with CAH receive glucocorticoid therapy.

The diagnosis of CAH in the fetus can be done by chorionic villus sampling or cytology of amniotic fluid. Foetal treatment can be initiated with maternal intake of dexamethasone.

In the neonate, ambiguous genitalia occur requiring rapid diagnosis and treatment.

(d) **Pheochromocytoma.** It is a rare catecholomine secreting adrenal tumour which presents as paroxysmal hypertension, headache, palpitations, anxiety, tremors or seizures, chest pain, flushing and nausea or vomiting.

It is rare during pregnancy but when present can be life-threatening for both the mother and the baby.

The diagnosis is made by finding raised levels of 24 hour urinary catecholamines, vanillylmandelic acid (VMA) or metanephrines and by MRI scan for localization of tumor. Treatment is with α-blockers like phenoxybenzamine 10 mg daily to control blood pressure. The dosage can be increased for control of BP. β-blockers may be added if palpitation is present or tachycardia develops. Propranolol 10 mg 3 to 4 times a day is commonly used. **Definitive treatment is surgery in 2nd trimester.**

After 1–2 weeks of medical therapy the patient should be taken up for surgical removal of the tumor. Multispeciality management of the patient is advised. Cesarean delivery under general anesthesia is usually indicated for better control of blood pressure.

## 4. Diseases of Parathyroid Gland

**Primary hyperparathyroidism.** It is rare and is caused by parathyroid adenoma causing prolonged nausea or vomiting or abdominal pain. High serum calcium levels (more than 12 μg/dL) are diagnostic of hyperparathyroidism. There is an increase in perinatal morbidity and mortality due to neonatal tetany or seizures.

**Treatment.** Surgery (removal of parathyroid adenoma) is required preferably in the second trimester.

Medical therapy with oral phosphates (1–1.5 g/day) is indicated only when the patient is unfit for surgery.

**Hypoparathyroidism.** The most common cause is surgical removal of the gland during thyroidectomy or irradiation. The symptoms are those of neuromuscular irritability.

Diagnosis is by persistent low calcium and high phosphate levels.

Treatment is with large doses of Vitamin D and calcium or calcitriol (1, 25 dihydroxy Vitamin $D_3$).

## Key Points

1. Estimation of TSH and free $T_3$ and $T_4$ are performed for accurate diagnosis of thyroid disorders in pregnancy.
2. Graves' disease is the most common form of hyperthyroidism and needs treatment with antithyroid drugs. Both propylthiouracil (PTU) and carbimazole can be safely given during pregnancy and lactation, but PTU is more preferred. Radioactive iodine is contraindicated.
3. Women with hypothyroidism need thyroxine therapy and should be euthyroid before conception to avoid intellectual impairment in the baby.

## Suggested Reading

1. American College of Obstetricians and Gynecologists: Thyroid Disease in Pregnancy. Practice Bulletin no. 148, April 2015, Reaffirmed 2017.
2. American Thyroid Association and American Association of Clinical Endocrinologists. Hyperthyroidism and other causes of thyrotoxicosis: Management Guideline of the American Thyroid Association and American Association of Clinical Endocrinologists. Endocrine Practice 2011; 17:456.
3. Nelson-Piercy C. Handbook of Obstetric Medicine, 4th edition, London, Informa books 2010
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## Examination Questions

A. Write short notes on:

1. Thyroid function tests in pregnancy
2. Hyperthyroidism during pregnancy
3. Hypothyroidism during pregnancy
4. Postpartum thyroiditis
5. Pituitary diseases in pregnancy
6. Cushing syndrome in pregnancy
7. Addison's disease in pregnancy
8. Parathyroid diseases in pregnancy
9. Sheehan's syndrome (postpartum hypopituitarism)

# (v) RESPIRATORY DISORDERS

## INTRODUCTION

Respiratory diseases, particularly asthma are common during pregnancy and are important causes of significant maternal and perinatal mortality and morbidity.

### Physiological respiratory changes during pregnancy

The various physiological respiratory changes during pregnancy are discussed in detail in Chapter 6 and Table 6.7.

In short, there is increased ventilation due to deeper breathing along with increase in vital capacity, inspiratory capacity and tidal volume while there is decrease in residual volume, expiratory reserve volume and functional residual capacity.

## RESPIRATORY DISEASES DURING PREGNANCY

The respiratory diseases during pregnancy are shown in Table 35.25.

### Dyspnea of pregnancy

Breathlessness or difficulty in breathing is common in pregnancy seen in up to 15%, 50% and 75% women in the first trimester, second trimester and third trimester respectively. It is probably due to marked changes in $pCO_2$ levels causing increased ventilatory drive with sensation of hyperventilation. Being physiological, it usually needs no treatment other than reassurance.

### Pneumonia during pregnancy

Pneumonia can occur during pregnancy due to various organisms (see Table 35.25) and is poorly tolerated by women and fetus due to resultant hypoxemia and acidosis. There is no evidence that pregnancy predisposes to pneumonia.

#### Diagnosis

Diagnosis for pneumonia is made from typical symptoms like cough, expectoration, dyspnea and pleuritic chest pain with associated fever, malaise and leucocytosis. Skiagram of chest is must for diagnosis and is done with abdominal shield. Sputum examination for microscopy for pneumococci and for culture can be done but are not essential.

#### Treatment

Patient is hospitalized, oxygen therapy is started along with empirical intravenous antibiotics usually erythromycin alone or with third generation cephalosporins (Cefotaxime) for pneumococcal pneumonia. Results are good. For influenza pneumonia (influenza A and B RNA viruses) **amantadine** or **oseltamivir** is given. Influenza vaccine is recommended during season. For $H_1N_1$ influenza **Oseltamivir** drug can be given along with symptomatic care of the patient. For varicella pneumonia with high morbidity and mortality intravenous acyclovir is started. Pneumonia caused by *Pneumocystis jeroveci (formerly carnii)* infection usually in HIV positive patients is treated with trimethoprim sulfamethoxazole and pentamidine.

**Table 35.25: Respiratory Diseases during Pregnancy**

| |
|---|
| 1. Dyspnea of pregnancy |
| 2. Pneumonias<br>(a) Bacterial pneumonia (*Pneumococcus:* most common)<br>(b) Viral pneumonias<br>(i) *Influenza* pneumonia<br>(ii) *Varicella* pneumonia<br>(iii) Severe Acute Respiratory Syndrome (SARS)<br>(c) *Chlamydia*<br>(d) *Mycoplasma*<br>(e) Fungal pneunomia<br>(f) *Pneumocystis jeroveci (parasitic)* pneumonia |
| 3. Other infectious and granulomatous conditions<br>(a) Tuberculosis (see Chapter 36 for details)<br>(b) Sarcoidosis |
| 4. Bronchitis: Treated like in non-pregnant state |
| 5. Bronchiectasis: Treated like in non-pregnant state |
| 6. Adult Respiratory Distress Syndrome (ARDS) |
| 7. Pulmonary embolism (see Chapter 30 for details) |
| 8. Amniotic fluid embolism (see Chapter 25 for details) |
| 9. Aspiration of gastric contents (Mendelson's syndrome) (see Chapter 19 for details) |
| 10. Asthma<br>(a) Acute asthma<br>(b) Chronic asthma |
| 11. Miscellaneous<br>(a) Cystic fibrosis<br>(b) Carbon monoxide poisoning<br>(c) Pleural effusion<br>(d) Trauma<br>(e) Pneumothorax<br>(f) Pneumomediastinum |

### Severe Acute Respiratory Syndrome (SARS)

SARS is a life threatening infection caused by a corona virus in which there is acute lung injury with consolidation and opacities in lungs with severe respiratory distress. There is high maternal mortality up to 25% in pregnancy. Treatment is ventilatory support, broad spectum intravenous antibiotics like clarithromycin with amoxicillin.

### Adult Respiratory Distress Syndrome (ARDS)

ARDS is a severe form of acute lung disease characterized by breathlessness, hypoxia, stiff lungs with infiltrates on skiagram of chest simulating pulmonary edema. It can be caused by obstetric complications like pre-eclampsia,

eclampsia, infections like septic abortion and puerperal sepsis, abruptio placentae, disseminated intravascular coagulation (DIC), Mendelson's syndrome, amniotic fluid embolism or pulmonary edema. There is severe respiratory depression with ultimately respiratory failure. Ventilatory support is usually required.

## ASTHMA

### Definition

Asthma is caused due to hyperresponsiveness of the airways to various irritants such as cold air, exercise, drugs like aspirin and viral infections. There is a reversible airway obstruction which is more marked during expiration and is due to airway inflammation, bronchial smooth muscle contraction, mucosal edema and mucus hypersecretion.

### Prevalence

The prevalence of asthma during pregnancy is increasing and is 5–9% (status asthmaticus is 0.2%).

### Clinical features

1. Dyspnea
2. Cough
3. Wheezing
4. Chest tightness
5. Use of accessory muscles of respiration.
6. Tachypnea

### Investigations

Lung function tests during attack of asthma show:

1. Abnormal expiratory flow rates
2. Depressed forced expiratory volume (FEV)
3. Decreased forced vital capacity (FVC)
4. Decreased $FEV_1/FVC$ ratio
5. Decrease in $PO_2$
6. Respiratory alkalosis (Decreased $PCO_2$ with increased pH)

### Effects of pregnancy on asthma

There is variable and unpredictable course of asthma during pregnancy. ***There was no change in the course of asthma in 33% women, worsening in 35%, while there was improvement in 28% women, while 4% women were not certain of the change.*** The increased levels of steroids like progesterone and cortisol during pregnancy improve the disease by causing bronchodilatation while increase in prostaglandin $F_2$ and thromboxane levels cause bronchoconstriction.

### Effects of asthma on pregnancy

Effects of asthma during pregnancy are shown in Table 35.26.

Adequate control of asthma can minimize various complications

**Table 35.26: Maternal and Foetal Effects of Asthma during Pregnancy**

| I. Maternal effects | Foetal effects |
|---|---|
| 1. Preterm labour (13%)<br>2. Premature rupture of membranes<br>3. Pre-eclampsia (11%)<br>4. Foetal growth restriction (11%) | 1. Perinatal mortality<br>2. Preterm delivery (12%)<br>3. Low birth weight babies (12%)<br>4. Perinatal hypoxia |
| **II. Maternal complications** | |
| 1. Pneumothorax<br>2. Cor pulmonale<br>3. Cardiac arrhythmia<br>4. Respiratory failure | |

### Management

#### Preconception Counselling

The parents should be counselled. The woman's education is important stressing her the need of proper prophylaxis and management of asthma in periconception, period and during pregnancy. There is increased risk of asthma in children of asthmatic parents.

#### Treatment

Most anti-asthmatic medicines are safe in pregnancy and should be used for proper control of asthma (Table 35.27). It depends upon the severity of the disease as given in Table 35.28.

Salbutamol, terbutaline, salmeterol as 2 to 3 puffs 4–6 hourly.

Triamcinolone, budesonide or beclomethasone in dose of 2–4 puffs 4 times daily.

#### Management of Acute Asthmatic Attack during Pregnancy

Management of acute asthmatic attack during pregnancy is the same as in non-pregnant state.

1. Oxygen therapy should be started.
2. Intravenous hydration is started.
3. $\beta_2$ agonists (salbutamol or salmeterol) are given with a nebuliser using oxygen.
4. $\beta_2$ agonist adrenaline can be administered in dosage of 0.4 mL of 1 : 1000 solution or terbutaline 250 µg subcutaneously every 15–20 minutes for 3 doses.
5. Steroids are the mainstay of treatment and should be given to all patients with severe acute asthma. They can be given as inhaler of budesonide or beclomethasone

**Table 35.27: Safety of Anti-asthmatics in Pregnancy**

| Drug | FDA class |
|---|---|
| Isoprenaline, Orciprenaline, Salmeterol | C |
| Salbutamol | A |
| Terbutaline | B |
| Beclomethasone/fluticasone | C |
| Budesonide | B |
| Monteleukast | B |
| Theophylline, Aminophylline | C |

2–3 puffs every 4–6 hourly. Intravenous hydrocortisone 200 mg stat and then 4 hourly can also be given with $\beta_2$ agonists for severe cases.

6. Severe persistent asthma with symptoms being continuous every day and night. Spirometry results are < 60% of normal. For severe asthma and status asthmaticus, mechanical ventilation is required to avoid hypoxemia and carbon dioxide retention. Humidified oxygen and continuous positive air way pressure is used and a team of physician and anesthetist should attend to the patient.
7. National Heart Lung and Blood Institute, National Asthma Education and Prevention Program Working Group Report (2007) have recommended **step therapy** for chronic asthma during pregnancy (Table 35.28). It is also recommended by Harrison's Principles of Medicine (2018)

**Table 35.28: Step Therapy for Chronic Asthma during Pregnancy**

| Category | Step therapy |
|---|---|
| 1. Mild intermittent asthma (< 2 per week) attacks | Inhaled $\beta_2$ agonists (Salbutamol, Salmetrol, Terbutaline) |
| 2. Mild persistent asthma (> 2 per week) attacks | Low dose inhaled corticosteroids (Budesonide, Beclomethasone fluticasone) preferred. Inhaled cromolyn sodium |
| 3. Moderate persistent asthma (Daily attacks) | Inhaled low dose corticosteriods with long acting $\beta_2$ agonists (Salmeterol). Add oral theophylline or monteleukast for patients inadequately controlled by medium-dose inhaled corticosteroids |
| 4. Severe persistent asthma (Throughout day attacks) | High dose inhaled corticosteroids with long acting β2 agonist (Salmeterol) and oral steroids if needed. **Alternative.** High dose inhaled corticosteroids with oral theophylline and oral steroids. |
| 5. Very severe persistent asthma (continuous) | Oral corticosteroids, long acting $B_2$ agonists, high dose inhaled corticosteroids. |

### Management of Asthma in Labour

1. Clinical evaluation for the severity of disease.
2. Continue maintenance medication of asthma like oral or inhalational medications. Women on steroids for control of asthma should receive parenteral hydro cortisone 100 mg intravenously every 8 hourly during labour. Inhaled steroids prevent bronchial hyperresponsiveness to allergens and should be continued.
3. Avoid using prostaglandins ($PGF_2$) as it can exacerbate asthma. $PGE_1$ and $PGE_2$ are safe for use.
4. Epidural analgesia is ideal for labour.
5. Avoid using ergometrine because it can cause bronchospasm. Oxytocin is safer for use in active management of the third stage and for postpartum haemorrhage.
6. Arterial blood gas analysis
7. Theophylline can be used especially for nocturnal asthma.

**Lactation.** Breastfeeding should be encouraged.

**Contraception.** For temporary use, barrier method is the safest and best. Oral pills and intrauterine device can be used. If family is complete, husband should be motivated for vasectomy.

## COVID-19 INFECTION IN PREGNANCY

### Introduction

COVID-19 (Corona virus disease) pandemic has rocked the whole world with the first case being reported from Wuhan, Hubei Province of the People's Republic of China to the World Health Organization (WHO) on 31st December 2019. The WHO declared corona virus disease 2019 (COVID-19) a Public Health Emergency of International Concern on 30th January 2020 and Pandemic on 11th March 2020. During the first wave by mid-August 2020 almost 213 countries and territories of the world were affected with 19.7 million cases (2.21 million in India) with 7.5 lac deaths (45,000 in India). However then second wave came with India becoming epicentre. By 2021, there had been about 259 million cases worldwide with 5.18 million deaths. India had reported 3.4 crore (34 million) cases of COVID-19 till 24th November 2021 with 4.66 lac deaths. By 24th November 2021, 7.79 billion (7790 million) doses of vaccine had been administered globally (1.18 billion in India) as per WHO estimate. The disease usually spreads by droplet infection, respiratory secretions and direct contact. Fortunately, most cases (80%) present as mild disease and asymptomatic infection. In about 20% cases usually with co-morbidities, the disease can be severe with pneumonia or other complications requiring admission with about 5% being very sick and necessitating ventilator facilities. WHO, Centre for Disease Control and Prevention (CDC USA), International Federation of Gynecology and Obstetrics (FIGO), American College of Obstetricians and Gynecologists (ACOG), Royal College of Obstetricians and Gynecologists (RCOG), Society of Maternal and Foetal Medicine (SMFM), Ministry of Health and Family Welfare (MOHFW), Government of India, Indian Council of Medical Research (ICMR) and Federation of Obstetric and Gynaecological Societies of India (FOGSI) have come with guidelines to manage COVID-19 in general population and in pregnancy. Pregnant women being special and vulnerable group are at risk of acquiring the disease with increased risk of complications to both mother and fetus with risk of morbidity and mortality.

## Etiopathogenesis

COVID-19 is caused by SARS CoV 2 which is a zoonotic disease. It is a single stranded 80-120 nm sized enveloped RNA beta corona virus which uses angiotensin converting enzyme (ACE 2) receptors for its entry into cells using S1 and S2 subunits. Its incubation period is about 5-7 days while period of infectivity is 2 days before onset of symptoms and lasts upto 8 days. Hence asymptomatic and mild symptomatic persons can also transmit infection. The structure of virus has been shown in Fig. 35.8.

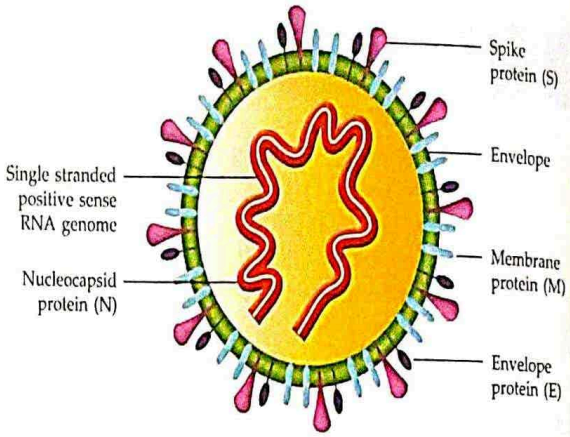



**Fig. 35.8:** Corona virus structure

## Classification of COVID-19 infection

1. **Confirmed case:** Laboratory confirmation of SARS-COV-2
2. **Probable case:** In which testing for COVID-19 is inconclusive or could not be performed
3. **Suspect case:** A patient with acute respiratory illness like fever with cough or shortness of breath or history of travel to a place with community transmission of COVID-19 within last 14 days or in a contact with COVID-19 in last 14 days.

## Clinical features of COVID-19

Common presenting symptoms are pyrexia (fever), cough, sore throat and dyspnea. Various symptoms of COVID-19 are shown in table 35.29.

Various signs of COVID-19 including signs of severe disease requiring hospital admission are also shown in table 35.29.

## Classification of COVID-19 illness severity in pregnancy

The classification of COVID-19 illness during pregnancy is described in Table 35.30.

Frequency wise most patients (upto 80%) have mild to moderate disease while about 15% cases have severe disease and about 5% cases have critical disease. There is high mortality (upto 30- 40%) in critical disease.

**Table 35.29: Symptoms and signs of COVID-19 infection**

| **Symptoms** | | |
|---|---|---|
| 1. Fever | 8. Headache | 15. Abdominal pain |
| 2. Cough | 9. Rhinorrhea | 16. Anosmia (loss of smell) |
| 3. Breathlessness | 10. Nasal congestion | 17. Ageusia (loss of taste) |
| 4. Expectoration | 11. Loss of appetite | 18. Reduced alertness |
| 5. Myalgia | 12. Diarrhea | 19. Reduced mobility |
| 6. Lassitude (lack of energy) | 13. Nausea | 20. Delirium |
| 7. Sore throat | 14. Vomiting | |
| **Common Signs** | **Signs of Severe Disease** | |
| 1. Raised temperature | 1. Respiratory rate >30/min | |
| 2. Breathlessness | 2. Hypoxia with oxygen saturation (SpO2) <94% on room air | |
| 3. Nasal congestion (on examination ) | 3. Tachycardia >110/bpm | |
| 4. Sore throat (on examination) | 4. Systolic blood pressure <90 mmHg | |
| **Complications** | | |
| (i) Pneumonia<br>(ii) Acute respiratory distress syndrome<br>(iii) Acute liver injury (raised liver enzymes)<br>(iv) Cardiovascular complications<br>(a) Cardiac injury (troponin elevation)<br>(b) Acute heart failure<br>(c) Dysrhythmias<br>(d) Myocarditis<br>(e) Prothrombotic coagulopathy<br>(f) Venous and arterial thromboembolism | (v) Acute kidney injury<br>(vi) Neurological complications<br>(a) Impaired consciousness<br>(b) Acute cerebrovascular disease<br>(c) Shock<br>(vii) Cytokine storm<br>(viii) Macrophage activation syndrome | |

| Table 35.30: COVID-19 Illness Classification during pregnancy | |
|---|---|
| Asymptomatic | No symptoms.<br>Positive screen test for COVID-19 |
| Mild disease | Mild flu like symptoms like fever, cough, myalgia, anosmia, no dyspnea or shortness of breath |
| Moderate disease | (a) Lower respiratory tract disease with dyspnea |
| | (b) Imaging (CT Scan) shows pneumonia |
| | (c) Abnormal blood gas analysis |
| | (d) Refractory fever of 39 °C (102.2 °F) or more not relieved with paracetamol |
| | (e) Oxygen saturation is maintained to 94% or higher on room air |
| Severe disease | (a) Respiratory rate of 30 or more breaths per minute |
| | b) Oxygen saturation < 94% |
| | (c) Involvement of lungs in > 90% cases on imaging |
| Critical disease | (a) Respiratory failure requiring mechanical ventilation |
| | (b) Shock |
| | (c) Multiorgan failure or dysfunction |
| Refractory hypoxemia | Persistent inadequate oxygenation and/or ventilation inspite of appropriate and substantial measures to optimize it. e.g., PaO2 < 70 mmHg, PaO2/FiO2 < 150 mmHg |

| Table 35.31 Investigations in COVID -19 |
|---|
| **I. Tests for Covid diagnosis** |
| 1. Nasopharyngeal swab ± oropharynged swab for COVID-19 RT-PCR: Report takes 12 hours. CB-NAAT: gives report in 3-4 hours.<br>or TRU-NAAT |
| 2. Rapid Point of Care (POC) Antigen Detection Test |
| 3. IgG ELISA test (for seroprevalence, not for diagnosis) |
| **II. Other General Laboratory Investigations** |
| 1. Complete hemogram: lymphopenia, coagulation profile, D dimer, low fibrinogen, prolonged prothrombin time, thrombocytopenia |
| 2. Elevated inflammatory markers<br>(i) Raised erythrocyte sedimentation rate (ESR)<br>(ii) Raised C-reactive protein<br>(iii) Raised ferritin<br>(iv) Raised tumour necrosis factor-α and interleukin IL-1, IL-6 |
| 3. Renal function tests maybe deranged: increased serum creatinine, blood urea |
| 4. Hepatic function tests maybe deranged: Increased AST (aspartate aminotransferase)/ALT (alanine aminotransferase), serum bilirubin |
| 5. Increased lactate dehydrogenase (LDH) |
| 6. Radiological imaging<br>(i) Chest X-ray (for pneumonia/ suspect)<br>(ii) CT scan (for pneumonia/suspect): Bilateral peripheral interstitial infiltrates, ground glass opacity or lower lobe consolidation |

## Investigations

Various laboratory investigations depend upon clinical symptoms and severity of disease and are shown in Table 35.31.

For suspected COVID-19 pneumonia, CT scan is investigation of choice even during pregnancy with minimal radiation risk to the fetus and should be done without hesitation in needed patients. Abdominal shield should be used during X-ray chest or CT scan chest.

## CO-RADS scoring on CT chest

| | | |
|---|---|---|
| CO-RADS 1 | No | Normal/Non-infectious |
| CO-RADS 2 | Low | abnormality consistent with infection other than COVID-19 |
| CO-RADS 3 | Indeterminate | Unclear whether COVID-19 is present |
| CO-RADS 4 | High | Abnormalities suspicious for COVID-19 |
| CO-RADS 5 | Very high | Typical COVID-19 |
| CO-RADS 6 | PCR +ve | COVID-19 |

## COVID-19 and pregnancy

Theoretically pregnant women may be more susceptible to COVID-19 due to their increased vulnerability to respiratory infections like Severe Acute Respiratory Syndrome (SARS) infection and Middle East Respiratory Syndrome (MERS) infection which were more common and severe in pregnancy. Fortunately, most International studies have shown that pregnant women are not more prone to develop COVID-19 in pregnancy.

## Effect of COVID-19 on pregnancy

Most studies suggest that pregnant women with COVID-19 are not at higher risk of developing serious disease like COVID-19 pneumonia than non-pregnant women. However, Centre for Disease Control and Prevention (CDC) Surveillance and Advisory and American College of Obstetricians and Gynaecologists (ACOG) suggest that pregnant women with COVID-19 appear to be at an increased risk of Intensive Care Unit (ICU) admissions and mechanical ventilation with risk being more in black and Asian women. However, as in non-pregnant state women with comorbidities like obesity, chronic kidney disease, cardiac disease (both congenital and acquired) are associated with serious disease like pneumonia and

adverse pregnancy outcome and must be treated in tertiary referral centres with facilities for ICU and mechanical ventilation. There is increased risk of thromboembolism in COVID-19 pregnancy which should be looked for in all cases and low molecular weight heparin prophylaxis should be given in high risk or suspected cases. They also need psychological support due to increased risk of anxiety and mental disorders in them. There is no increase in risk of miscarriages (abortion) or congenital malformations (COVID-19 is not a teratogen) in such pregnancies and COVID-19 is certainly not an indication for termination of pregnancy.

Although some studies have observed some adverse outcomes with COVID-19 pregnancies in the form of slightly increased risk of preterm delivery, premature rupture of membranes, maternal sepsis, pre-eclampsia and postpartum haemorrhage. But, most of other studies have not shown any adverse effect of COVID-19 during pregnancy including on foetal growth. Increased preterm labour in some studies may even be iatrogenic. Although cesarean delivery was the main mode of delivery in the initial studies, most recent studies have observed that vaginal delivery can be safely performed in COVID-19 pregnancy and is infact the current practice. In a large study from Mumbai, Nayak et al observed no difference in the mode of delivery, low Apgar score, mortality rate or other maternal complications in COVID-19 pregnant women as compared to non-COVID patients. In general, the consensus is that mild to moderate COVID-19 is not associated with adverse maternal outcomes.

## Vertical Transmission of COVID-19

Although, there have been few case reports of vertical transmission of COVID-19 in newborns including 4 cases in a meta-analysis, most large studies did not find vertical transmission of COVID-19 during pregnancy. These studies also did not find virus in neonatal specimens, breast milk, placenta, amniotic fluid and cord blood. In general, the consensus is that vertical transmission is rare in COVID-19 pregnancies.

## Perinatal and Neonatal Outcome

Most studies have shown that COVID-19 in pregnancy especially without severe disease and comorbidities is associated with normal perinatal and neonatal outcome. Nayak et al from Mumbai observed equal Apgar score, overall birth weight, low birth weight, preterm babies, intrauterine foetal deaths and neonatal admissions in asymptomatic and mild COVID 19 pregnancies and normal controls. However, perinatal and neonatal complications can be higher in severe COVID-19 diseases like pneumonia and other complications or comorbidities.

## Mode of delivery

There was higher cesarean delivery rate (upto 82-100%) in COVID-19 patients in the initial part of pandemic due to lack of proper protocols and lack of time needed to put on Personal Protective Equipment (PPE) and hesitancy to keep COVID-19 positive patient in labour for long periods. However, with the experience of handling COVID-19 positive patients in labour and delivery especially in mild and moderate disease, vaginal delivery is now recommended by most guidelines. Cesarean delivery is done only for obstetric indication or for severe or critical disease. Hence, the current consensus and practice is that vaginal delivery is conducted in most COVID-19 pregnancies with excellent maternal and perinatal outcome especially in mild and moderate disease.

## Antenatal (Prenatal) care of COVID-19 pregnancy

The COVID-19 pandemic had significant impact on outpatient prenatal care due to lock down and closure of outpatient departments. Most pregnant women were managed by teleconsultation which emerged as significant care method during pandemic. The women needing emergency care for their obstetric or other medical or surgical problems could come to emergency or labour rooms which were functional round the clock. The antenatal visits were reduced to bare minimum of 4 at 12, 20, 28 and 36 weeks in low-risk pregnant women. The management of COVID-19 pregnancies during antenatal period is shown in flow chart 35.9.

## Triaging of patients and setting up of obstetric services during covid pandemic

During the peak of pandemic, there was absolute chaos with scarcity of staff and essential resources like PPE kits and the risk of transmission of virus to staff and other patients and family members. There was risk of failure of healthcare system due to sudden influx of patients with lack of beds, ICU beds, oxygen and other essential commodities. As per European experience during the peak of pandemic, all obstetric hospitals were advised to prepone their obstetric units to triage patients which was successfully done at All India Institute of Medical Sciences (AIIMS), New Delhi and most other obstetric hospitals by reorganising and triaging the patients. Thus, at AIIMS, New Delhi we had COVID-19 positive cases in trauma centre, (designated COVID centre equipped with all COVID facilities including trained staff and PPE kits), COVID suspect cases in suspect area and proven COVID negative cases in routine obstetric ward and labour room. It helps in minimising the viral transmission and to use the health care facility specially PPE judiciously and prevents failure of healthcare system. It has been recommended by most guidelines like ACOG, RCOG, FIGO and WHO and various authors. Even COVID positive women can be further triaged as per severity of disease with asymptomatic and mild patients being managed at home, while patients with moderate to severe disease being admitted in COVID positive area and those with critical disease being admitted in Intensive Care Unit with ventilatory support. The patients are checked regularly and can be **downgraded** if they get better and are shifted to lower care. They can be **upgraded** if they worsen and can be shifted to ICU anytime as per the needs of the patients and severity of disease. All COVID positive patients are looked after by

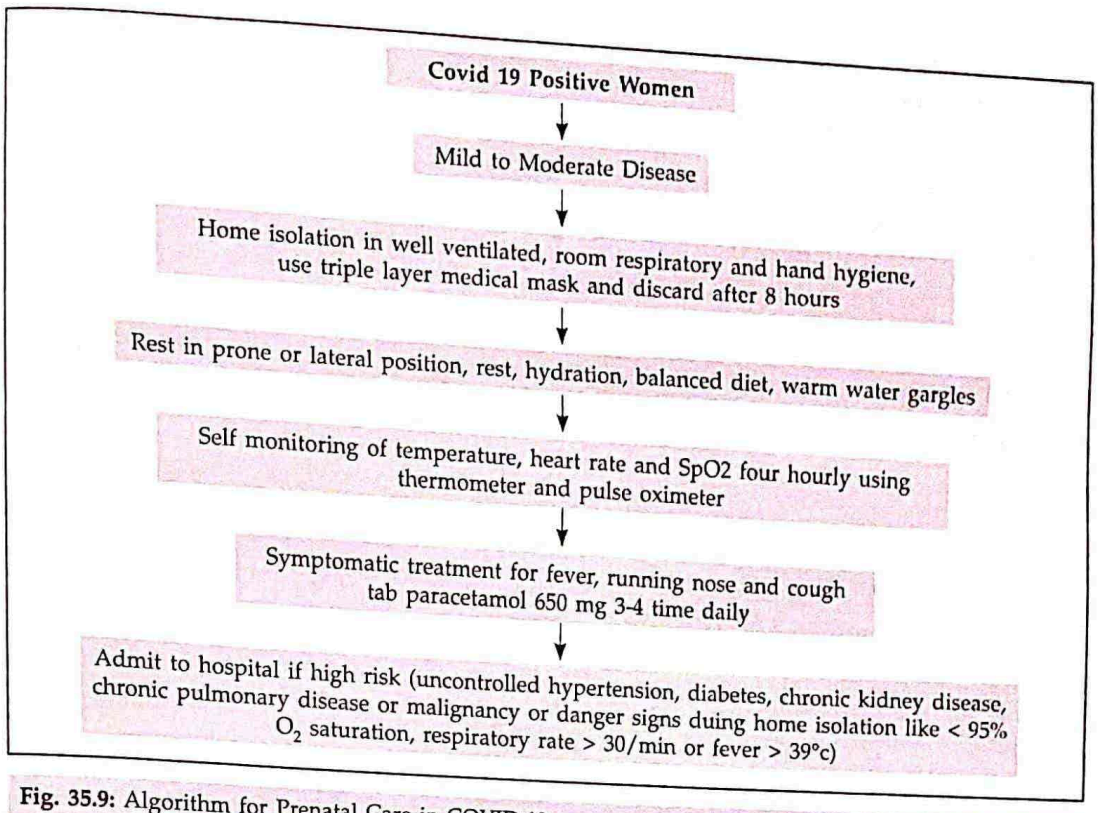



**Fig. 35.9:** Algorithm for Prenatal Care in COVID-19 cases

staff in full PPE in shift duties and with full precautions to prevent exhaustion in staff and to decrease the risk of transmission to them. All the staff are fully trained in use of PPE kits and other precautionary methods especially "donning" (putting PPE) and "doffing" (taking off PPE), which is even more important.

## Management of confirmed COVID-19 positive cases with asymptomatic or mild disease

Most COVID-19 positive pregnant women (more than 80%) are either asymptomatic or have mild disease. If she has no comorbidities like hypertension, diabetes, heart disease, chronic kidney disease, lung disease, HIV or blood dyscrasias or on immunosuppressant medications then outpatient care with 14 day quarantine at home is considered optimal with no need of performing chest imaging (X-ray or CT scan) or tests for inflammatory markers. The management of aymptomatic or mild cases with COVID-19 is shown in flowchart 35.10.

After the patient recovers from infection follow up in hospital or ultrasound may be performed two weeks after infection. However, during home care the patient is regularly monitored through teleconsultation and must be called to hospital for admission if there is any deterioration in health or any concern about her or foetal health. This approach can judiciously use resources without compromising maternal and foetal health.

## Management of confirmed COVID positive pregnant women - Moderate to Severe disease

These patients should be admitted to hospital with multidisciplinary care from Obstetricians, Infectious Disease team and Critical Care Specialists. Initially, they can be managed in COVID positive obstetric ward but can be shifted to ICU if needed and as discussed later. It is shown in flowchart 35.11. If patient has complications of COVID like COVID pneumonia or mucor mycosis, then they should be treated as per protocol. Liposomal amphotericin B is given for mucor mycosis.

## Management of confirmed COVID positive critically ill pregnant women and those with refractory hypoxemia:

It is shown in table 35.32.

## Management of labour (Intrapartum care) in COVID-19 patients

The algorithm for intrapartum care in COVID-19 case is shown in flowchart 35.12. Being a high risk pregnancy, labour of a COVID positive patient should be managed in a tertiary care centre with facilities of maternal and neonatal intensive care and where experience and expertise to look after high risk patients is there and triage facilities have been in place. Most large public hospitals in India have designated COVID positive areas with facilities and trained staff to look after such patients. All the staff looking after these patients should be trained to wear full PPE including N95 mask, coverall, long shoe covers, goggles or long face shield properly during handling of this patients during delivery or cesarean delivery.

## Mode of delivery

As already discussed COVID-19 positivity is not an indication for cesarean delivery in current practice. Indications of cesarean delivery:

(a) Obstetric indications
(b) Severe disease
(c) Critically ill patients with refractory hypoxemia

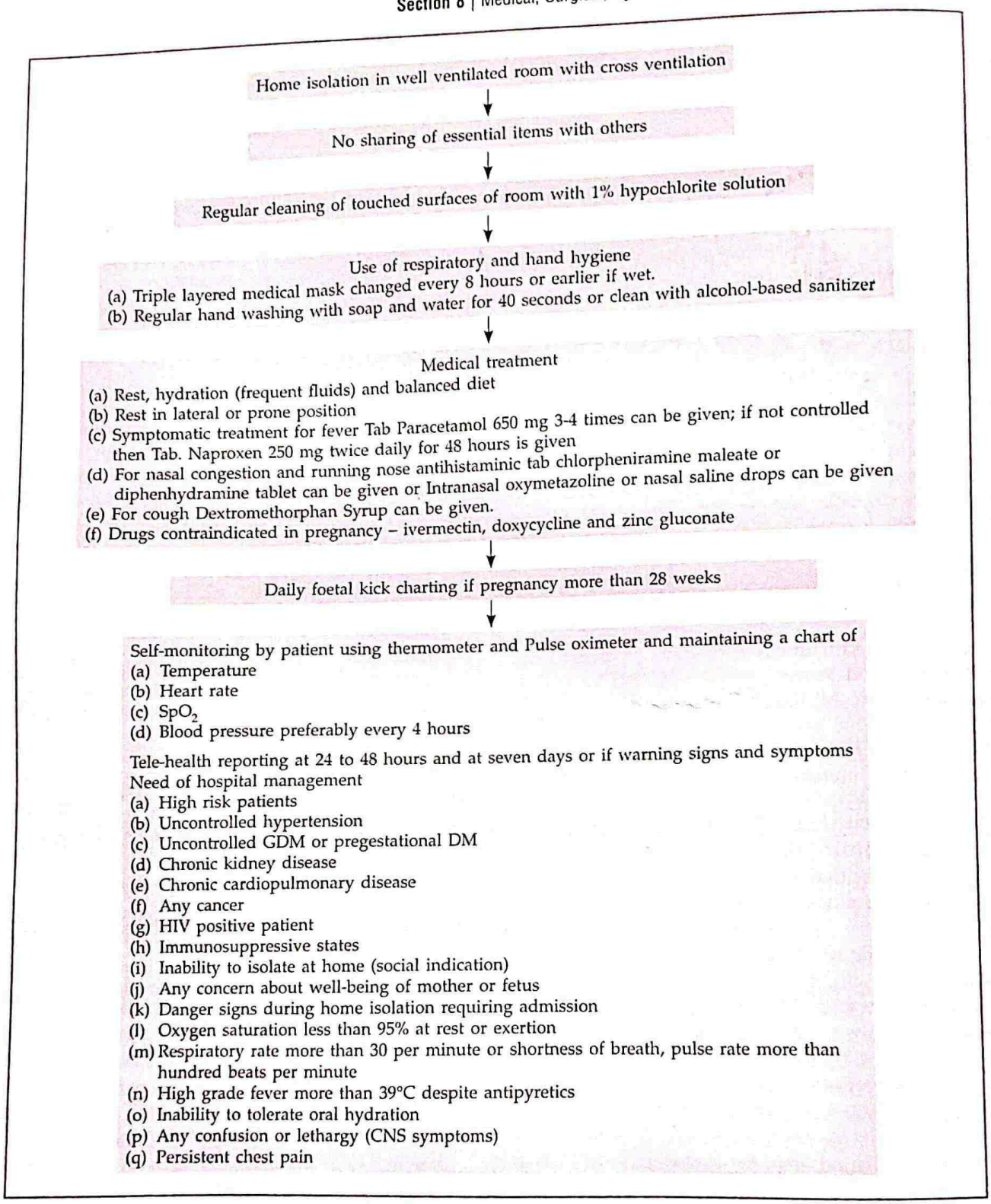



**Fig. 35.10:** Management of COVID-19 positive women with mild disease or asymptomatic cases

## Timing of delivery

It depends upon the severity of disease. For asymptomatic and mild-to-moderate disease,for most patients delivery is based on obstetric indication.

If period of gestation between 37 to 38 weeks 6 days, expectant management can be planned till about 14 days after positive test, or until 7 days after onset of symptoms and 3 days after resolution of symptoms.

If period of gestation is 39 weeks or more then delivery should be considered.

## For severe and critical disease

Multidisciplinary care to first stabilize the patient and then to plan termination of pregnancy. It is reasonable to terminate if gestation is more than 32 to 34 weeks in cases with worsening illness or refractory hypoxemia with $PaO_2$ < 70 mmHg or $PaO_2/FiO_2$ < 150 mm Hg

**Table 35.32: Management of COVID-19 critically ill patients**

**Indications of admission to Intensive Care Unit (ICU)**

(a) Patient is unable to maintain $SpO_2$ ≥ 95% with supplemental oxygen
(b) Rapidly escalating supplemental oxygen need
(c) Hypotension with mean arterial pressure < 65 mmHg in spite of fluid resuscitation
(d) Evidence of fresh end organ dysfunction
- Renal insufficiency
- Liver insufficiency
- Cardiac dysfunction
- Altered sensorium or mental status.

**Indications for intubation -As these patients are critically ill or hypoxemic, mostly they will need intubation as follows**

(a) when oxygen requirements for a target of > 95% $SpO_2$ are
- >15 litre per minute by common nasal cannula or mask
- > 40–50 litre per minute by High Flow Nasal Cannula (HFNC)
- > 60 % $FiO_2$ by venturi mask
- Inability of the patient to protect the airway due to altered mental status (Glasgow Coma Score < 8).

## Intrapartum care

1. Ensure safe institutional delivery as discussed earlier
2. Stop anticoagulants if patient goes in labour. If induction is planned, then low molecular weight heparin (LMWH) is stopped 24 hours before and unfractionated heparin is stopped 6 hours before induction of labour. Unfractionated heparin is preferred near- term patients due to its short half-life. Protamine sulphate should be kept ready as anti-dote for heparin in case patient goes into labour on unfractionated heparin or LMWH.
3. Arrange for blood and blood products.
4. Hourly monitoring of saturation ($SpO_2$ should be ≥ 95%) respiratory rate and temperature
5. Continuous electronic foetal monitoring in all cases as COVID -19 is a high risk pregnancy
6. Epidural analgesia can be given except for patients with thrombocytopenia or on anticoagulants. Pudendal block analgesia can also be given.
7. Birth companion: There are no clear guidelines about it and depends upon hospital and facilities. If birth companion is allowed, the person should be COVID negative and same companion should remain with the patient during labour and immediate postpartum period.
8. Strict intake output chart to avoid fluid overload.
9. Delivery is conducted in separate isolation room by doctor and staff with full PPE and patient must wear mask at all time.
10. Instrumental delivery can be performed if women are exhausted or prolonged second stage of labour but not routinely.
11. Active management of third stage should be practiced. Carboprost is avoided.
12. Although there used to be a debate about early or delayed cord clamping, current consensus is delayed cord clamping which is also practiced in most hospitals in India including AIIMS, Delhi.
13. For cesarean delivery done for severe disease and strict obstetric indications regional anesthesia (epidural or spinal) is preferred as general anesthesia is aerosol generating with high risk of viral transmission to the staff in operation theatre. If however, general anesthesia has to be given for failure of regional anesthesia or where it is contraindicated, then the staff must wear full PPE with FFP 3 mask before entering the theatre along with disposable water-repellent long sleeved gown, double gloves, facial screen, goggles, disposable headgear and gumboots. Cautery to be avoided intra-operatively as it generates aerosols. Obstetricians and other staff should enter the theatre only after anesthesia has been given. After the procedure all staff must doff (put off PPE) as per protocol of the hospital by changing gloves two times and washing hands with antiseptic solution and should preferably take bath, a practice followed in AIIMS Delhi and most hospitals.

## Postnatal (Postpartum) care

It is shown in table 35.33.

**Care of mother:**

1. Mother should wear N95 mask and must practice hand hygiene during contact with the infant.
2. She should practice respiratory etiquettes.
3. She is monitored for pulse, blood pressure, temperature,

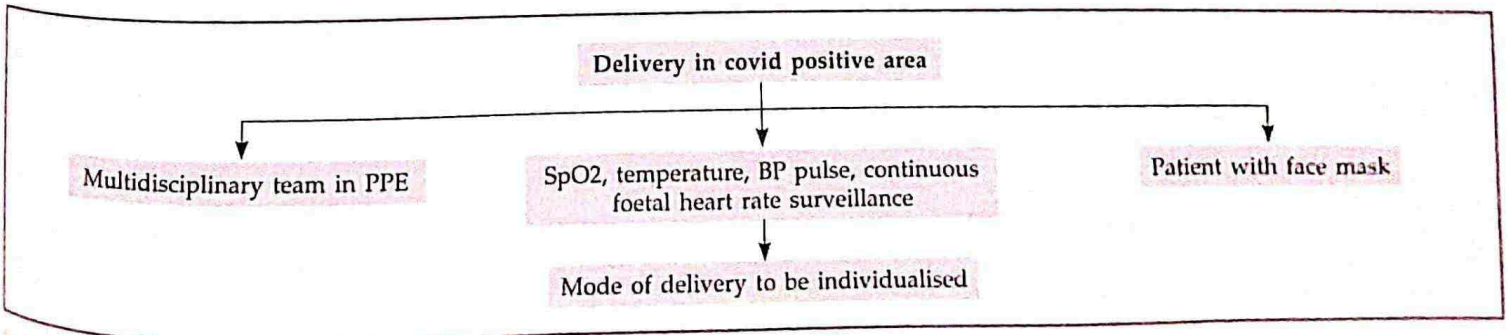



**Fig. 35.12:** Flowchart showing algorithm for Intrapartum care in COVID-19 cases.
[PPE: Personal Protective Equipment; ARDS: Adult Respiratory Distress Syndrome; $SpO_2$: Serum Pressure Oxygen (Oxygen Saturation)]

| Table 35.33: Postnatal care | |
|---|---|
| Rooming in | Encourage rooming in of mother and baby at 6 feet distance |
| Contact and droplet precaution | Mother should wear a mask and practice hand hygiene during contact with their infants |
| Breastfeeding | Breastfeeding should be encouraged. However, if the mother is not well, expressed breast milk may be given |
| Postpartum analgesia | Acetaminophen preferred |
| Postpartum thromboprophylaxis | Individualized risk assessment for venous thromboembolism Prophylactic anticoagulation if indicated (6 hrs after vaginal delivery and 12 hrs after cesarean delivery) |
| Postpartum maternal monitoring | Check vitals, $SpO_2$, temperature, hydration and bleeding per vaginum and input-output charting |
| Postpartum contraception | Long acting reversible contraception/Sterilization |
| Postpartum mental health | Educate patients to prevent postpartum depression |
| Timing of discharge | Depends on mother's need for COVID care/postpartum care in hospital, facility for home isolation and neonatal status |
| Counselling at discharge | Explain period of isolation and need for tele health counseling |
| Vaccination | COVID Vaccination should be given to lactating women |

$SpO_2$, hydration, any bleeding per vaginum and urine output. For postpartum fever, puerperal pyrexia must be ruled out.

4. For pain relief paracetamol or acetaminophen are given
5. Antibiotic are only indicated for obstetric indication or bacterial pneumonia
6. Breastfeeding is encouraged while the mother wear a N95 mask. The infant should not be made to wear a mask.
7. **Thromboprophylaxis:** Early ambulation is encouraged. Stockings can be worn by the patient. Avoid dehydration and give adequate hydration. If she is on thromboprophylaxis start anticoagulant (LMWH or Heparin) 6 hours after vaginal delivery and 12 hours after caesarean delivery.
8. Postpartum contraception is encouraged using long acting reversible contraception (LARC) like intrauterine devices, implants or sterilization as per patient preference and clinical situation.
9. Postpartum mental health – COVID-19 positive women have higher prevalence of depression and psychiatric disorders due to lack of support for themselves and infant by the family due to isolation rules. Adequate education to the patient and family is needed to prevent stress and depression which if present, needs medical treatment and counselling by the psychiatrist and obstetrician. Validated mental health questionnaires like patient health questionnaire (PHQ) can be used.
10. **Time of discharge:** It depends upon maternal and neonatal condition. For asymptomatic and mild disease, patient can be discharged 24 to 48 hours after normal vaginal delivery and 3-4 days of cesarean delivery. Moderate to severe disease patients are kept longer till they recover and show improvement in symptoms at 14 days after appearance of first symptom (up to 20 days in severe or critically ill or severely immunocompromised cases) or at least three days of afebrile period without use of antipyretics.

## Care of the newborn

It is shown in flowchart (Fig. 35.13).

1. The new born is attended by the neonatologist in full PPE at the site of delivery. Suction is avoided unless absolutely necessary.
2. **Cord clamping:** There was some debate about early and delayed cord clamping. The current thinking and practice is delayed cord clamping (after 1 minute) a practice followed in AIIMS Delhi.
3. **Rooming in:** During the early part of pandemic there was debate whether the baby should be separated from mother or kept with her and about breastfeeding. However, most studies have proven that transmission of virus is unlikely by breastfeeding and virus is not detected in milk. However, the current thinking and practice advice by most societies including CDC, Society for Maternal Foetal Medicine (SMFM) recommend rooming in and breastfeeding. At AIIMS Delhi and most other hospitals, rooming in is practiced unless mother or infant are sick. Baby is usually kept at 6 feet distance with caregiver when not feeding and the physical barrier like curtain can be used.
4. **Breastfeeding:** Current practice and recommendation by most societies is that breast feeding is done by mother with following precautions:
   (a) Mother must wear N95 mask
   (b) To observe hand hygiene
   (c) To observe respiratory hygiene
   (d) In case of temporary separation due to severe mental disease, the baby is looked after by the father or other close relative with expressed milk with dedicated breast pump which is disinfected after each use.
   (e) COVID testing of new born: All new born babies born to COVID-19 positive mother are tested for COVID-19 on day 3 and day 5 using RT-PCR.
   (f) New born is given all suitable vaccinations.

## Time of discharge

Counselling and advice at discharge

1. Explain danger signs of puerperal complications and COVID-19 judiciously.

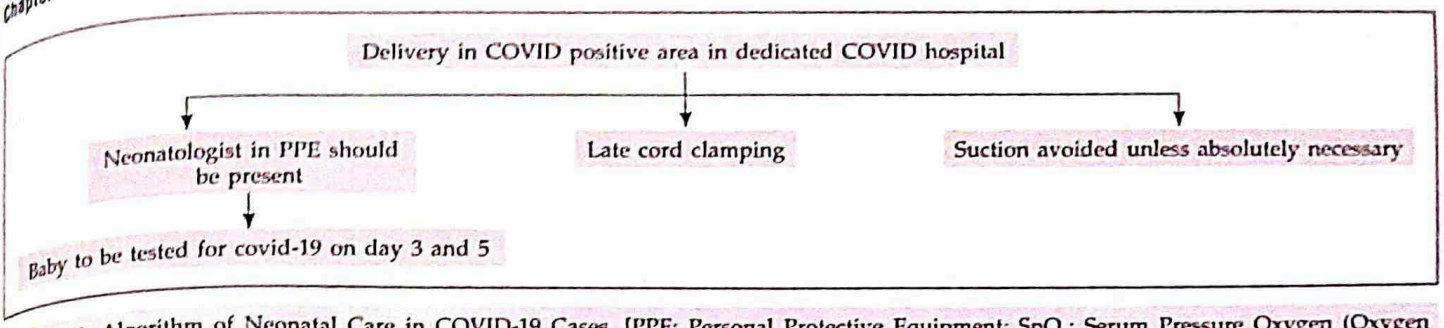



Fig. 35.13: Algorithm of Neonatal Care in COVID-19 Cases. [PPE: Personal Protective Equipment; $SpO_2$: Serum Pressure Oxygen (Oxygen Saturation)]

2. The women should observe all COVID-19 precautions at home.
3. She should be given teleconsultation phone number or hospital number where she can report in case of any emergency to her or the new born. Arrangements should be made for her physical appointment to hospital for any emergency.
4. Routine postnatal advice and care like iron and calcium supplementation, care of, dietary advice and general hygiene is given.
5. Contraceptive advise if PPIUCD or sterilization not already done.
6. Follow up is arranged after she recovers.
7. The Infant vaccinations and care as per routine are advised

## COVID-19 vaccination

It's recommended by all societies and Government of India to have COVID-19 vaccination in normal pregnancy with either Covishield or Covaxin with 2 intramuscular doses 4–6 weeks apart. In fact pregnant women need it more. It can be given even in first trimester. In COVID-19 positive, it should be given 3 months of (90 days) after COVID-19 infection. It can also be given any time in puerperium. There should be a gap of 4 weeks between any two vaccines during pregnancy such as between Tetanus vaccine and COVID vaccine.

## Key Points

1. Pregnancy itself does not usually influence the severity of asthma. Asthma usually does not adversely affect the pregnancy outcome in majority of cases if well controlled.
2. Poorly controlled severe asthma presents more risk to the pregnancy than the medication used to prevent or treat the asthma. Proper counselling of women about the adequate control of asthma during pregnancy is important as decreasing or stopping anti-asthmatic medication can cause serious deterioration of asthma.
3. Treatment of asthma is the same in pregnancy as in non-pregnant state. Most anti-asthmatic medications like inhaled, oral and intravenous steroids, inhalers, nebulizers and intravenous b2 agonists are safe to use during pregnancy and lactation. If the symptoms are not properly controlled with existing medication, an increase in the dose or frequency of inhaled steroids should be done to optimally control the disease for better maternal and perinatal outcome.
4. COVID-19 pandemic has affected the world significantly with high prevalence, morbidity and mortality
5. COVID-19 during pregnancy is a high-risk pregnancy and should be taken care of in tertiary Care Hospital with all facilities and expertise for taking care of COVID positive patients
6. Triaging is strongly recommended where patients are divided into three categories: COVID positive (in COVID positive ward), COVID suspect (in suspect areas) and COVID negative who are kept in routine obstetric wards. It reduces the risk of transmission and helps in judicious use of PPE and other health resources.
7. COVID positive patients are divided into asymptomatic, mild, moderate, severe and critical disease. Asymptomatic and mild disease patients can be managed at home with self-isolation and provision for their admission to hospital if needed is made.
8. Patients with moderate and severe disease need hospital admission in designated COVID facility and are looked after by staff in full PPE. They may require oxygen therapy and are given steroids (methylprednisolone), anticoagulants and in selected cases Remdesivir or tocilizumab. However, doxycycline and ivermectin are contraindicated in pregnancy.
9. Critically ill patients require admission into ICU and may require mechanical ventilation.

10. COVID vaccine can now be given to COVID-19 negative pregnant women anytime during pregnancy in two intramuscular doses (Covaxin 4-6 weeks apart or Covishield at 2 months interval). It can also be given to COVID-19 positive patients after 3 months of COVID-19 infection.
11. Intrapartum care in tertiary referral centres is by staff in full PPE. These are high risk pregnancies, epidural analgesia can be given. Vaginal delivery is allowed. Continuous electronic foetal monitoring is recommended. Normal vaginal delivery is recommended. Instrumental delivery only if indicated. Cesarean delivery is done only for obstetric indications or severe or critical disease. Cesarean should be done under regional anesthesia to avoid aerosol transmission.
12. New born is looked after by the neonatologist. Delayed cord clamping is practiced. Suction is avoided.
13. New born is tested for COVID-19 RT-PCR on day 3 and 5.
14. Rooming in is allowed with six feet distance in room
15. Breastfeeding is allowed with hand and respiratory hygiene (mother must wear N95 mask) and wash hands regularly. If baby is separated due to maternal or neonatal sickness, then expressed milk using a dedicated breast pump is used to feed the baby.
16. All vaccines to be given to the baby.
17. Discharge as per condition of the mother and baby and is usually 24 to 48 hours of vaginal delivery and day 3 or 4 after cesarean delivery in case of mild disease. For moderate to severe disease, longer hospitalization is needed.
18. Mother should practice precautions at home with provision to come to hospital in case of any maternal or neonatal emergency.
19. Thromboprophylaxis and contraception are advised
20. Routine postnatal advices are also given

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# (vi) RENAL DISORDERS DURING PREGNANCY

## PHYSIOLOGICAL RENAL AND URINARY TRACT CHANGES DURING PREGNANCY

Various physiological changes in structure and function in the urinary tract during pregnancy are discussed in Chapter 6.

1. Dilatation of the renal calyces, pelvis and ureters along with the increased vesicoureteral reflux predispose pregnant women to urinary tract infections.
2. Serum concentrations of creatinine and urea are decreased in pregnancy due to increase in renal plasma flow. S. Creatinine > 0.9 mg/dl is suggestive of intrinsic renal pathology.

## URINARY TRACT INFECTIONS (UTIs)

UTIs are the most common infections encountered during pregnancy. In most of the cases, the organism found is *Escherichia coli* (90–95%). Others are *Klebsiella pneumoniae, enterococci, Proteus mirabilis* and *Pseudomonas aeruginosa,* Citrobacter, stayphylococcus saprophyticus, Staphylococcus epidermidis. These can be grouped into asymptomatic bacteriuria, cystitis or pyelonephritis.

### Asymptomatic Bacteriuria

Asymptomatic bacteriuria occurs in about 5% (range 2–7%) pregnancies. It must be treated as about 25% of these may subsequently develop active symptomatic infection during pregnancy.

#### Diagnosis of Asymptomatic Bacteriuria

Diagnosis of asymptomatic bacteriuria is made when a routine midstream urine culture of a clean-voided midstream specimen contains more than 100,000 ($10^5$) colony forming units (CFU) or organisms per mL **(Kass, 1962)**. In a catheter sample even 100 cFu/mL is also significant. Screening by urine culture and microscopy on the first visit is recommended (ACOG). Less expensive tests such as the leukocyte esterase-nitrate dipstick can be used for screening if the prevalence is low in the population.

#### Management

All bacteriuria in pregnancy requires treatment to prevent pyelonephritis and preterm delivery **(Cochrane guideline, level IA evidence)**. Without treatment 25% cases progress to pyelonephritis but with treatment it can be reduced to 24% (Cochrane 2015). It is also associated with increased risk for hypertension, pre-eclampsia and anaemia in mothers and low birth weight in children.

Treatment depends upon culture sensitivity report and is shown in Table 35.34. After initial treatment, periodic surveillance is required.

**Table 35.34: Treatment Regimens for Urinary Tract Infections in Pregnancy**

| |
|---|
| **I. Oral antibiotics** |
| 1. Nitrofurantoin 100 mg QID (4 times a day) or Nitrofurantoin SR (sustained release twice daily) for 7–10 days is the treatment of choice.<br>2. Ampicillin 250 mg four times daily for 3–5 days.<br>3. Amoxicillin 500 mg TDS (3 times a day) (8 hourly) for 3–5 days.<br>4. Cephalexin 250 mg TDS for 3–5 days.<br>5. Oral fosmomycin 3 g satchet with a glass of water at bed time or 1 hr before or 2 hr after meals as single dose therapy is also effective.<br>6. Single dose of amoxicillin 3 g, ampicillin 2 g, cephalosposin 2 g can be given. |
| **II. Intravenous antibiotics (for pyelonephritis)** |
| 1. Cefuroxime 750 mg TDS<br>2. Coamoxyclav 1.2 g TDS<br>3. Gentamicin 2–5 mg/kg/day in 8 hourly divided doses (for organisms resistant to penicillins and cephalosporins and in women allergic to penicillins and cephalosporin). |
| **Duration of treatment** |
| 1. Asymptomatic bacteriuria 3–5 days<br>2. Acute cystisis 7 days<br>3. Acute pyelonephritis: 14 days (IV × 2 days: oral × 12 days) |
| **For treatment failure** |
| Nitrofurantoin 100 mg four times daily for 21 days<br>**Prophylaxis of UTI (for recurrent UTI) for rest of pregnancy**<br>Nitrofurantoin 100 mg OD at bed time<br>Cephalexin 250 mg OD<br>Amoxicillin 250 mg OD |

#### Personal Hygiene Measures

1. Taking plenty of fluids
2. Avoiding delay in post-coital urination
3. Cleaning of the local area from the front to backward direction following defecation.
4. Avoiding douching
5. Cleaning the urethral meatus first while bathing.

#### Self-care Measures

1. Use of probiotics
2. Cranberry products: capsule/juice/tablet for 12 months (avoided during pregnancy)

#### Avoid during Pregnancy

1. Tetracyclines - teratogenic for metal teeth and bones
2. Aminoglycosides: cause ototoxicity for foetus
3. Fluoroquinolones: toxic to the developing cartilages of the foetus
4. Trimethoprim - sulpjhamethoxazole combination: avoided in 1st trimester as it is teratogenic and avoided

in 3rd trimester as it may result in kernicterus in the foetus
5. Nitrofurantoin should be avoided close to term as it may produce neonatal hemolysis.

### Cystitis

Cystitis is an infection of the lower urinary tract which is characterized by burning micturition (dysuria), frequency, urgency, Hematuria, nocturia and suprapubic discomfort. Systemic symptoms and signs are usually missing. It can complicate about 1% pregnancies. **Etiology** is the same as asymptomatic bacteriuria. Rarely hemorrhagic cystitis can cause frank hematuria. If left untreated, cystitis can cause ascending pyelonephritis.

Diagnosis is by urine analysis which reveals pus cells on microscopy (pyuria), bacteriuria and microscopic hematuria, urine culture and sensitivity.

Treatment depends upon the culture sensitivity report. Oral antibiotics are given as shown in Table 35.29. Nitrofurantoin is drug of choice.

## Acute pyelonephritis

The infection of upper urinary tract involving both renal pelvis and parenchyma is known as acute pyelonephritis. **Incidence** is 1–2% and can be reduced to 0.5% by treatment of asymptomatic bacteriuria. **Etiology.** Same as asymptomatic bacteriuria. Pyelonephritis is leading cause of septic shock during pregnancy.

#### Clinical Presentation

Onset is usually acute and presents in the second and third trimesters of pregnancy.

#### Symptoms

1. Fever with rigor and chills.
2. Malaise
3. Back pain usually located in the upper lumbar area radiating to the groin.
4. Anorexia, nausea and vomiting
5. Dysuria and frequency of micturition
6. Uterine contractions

#### Signs

1. Patient is dehydrated and febrile (above 101 °F).
2. Costovertebral tenderness
3. Urine is turbid or bloody.
4. Tachycardia

#### Investigations

1. **Urine.** Physical examination shows acid reaction, high specific gravity and proteinuria. Urine shows leucocytes, red cells, white cell casts and bacteria on microscopic examination or on UTI detection kits.
2. Urine culture and sensitivity test
3. Blood examination shows signs of renal dysfunction such as slightly elevated blood urea nitrogen, creatinine and creatinine clearance depending on the damage.
4. Blood culture if fever > 39°C.

#### Complications

1. Septic shock due to endotoxins.
2. Pulmonary injury (due to endotoxin induced alveolar injury and pulmonary edema)
3. Chronic renal infection
4. Adult respiratory distress syndrome (due to endotoxin induced lung injury)
5. Endotoxin induced hemolysis leading to anaemia.

#### Effect on Pregnancy

There is increased risk of
1. abortions.
2. foetal growth restriction.
3. intrauterine foetal death.
4. premature labour.

#### Treatment

1. Hospitalization
2. Four hourly pulse, blood pressure and temperature charting.
3. Pulse oximetry if there exists signs of adult respiratory distress syndrome.
4. Foetal monitoring
5. Observation for uterine contractions
6. Bed rest
7. Plenty of fluids
8. Easily digestible diet
9. **Intravenous fluids.** Intravenous hydration is cornerstone of treatment. These patients are frequently dehydrated and oliguric and require rapid expansion of intravascular volume with crystalloid solutions like dextrose and dextrose saline.
10. **Intravenous antibiotics.** Suggested treatment regimen for UTI including pyelonephritis is given in Table 35.29. For pyelonephritis, treatment is given intravenously (Inj cefoxitin sodium 2 gm IV, 6–8 hourly or Inj. Ceftriaxone sodium 1 gm daily, ampicillin 500 mg IV 6 hourly, coamoxyclav 1.2 g IV 12 hourly). Once patient is afebrile for 24–48 hours then oral antibiotics are to be started and given for 10–14 days. Recurrence of bacteriuria demands antibiotics treatment for the rest of pregnancy (Table 35.34). Patients not responding to therapy should have renal sonogram and if necessary, a modified intravenous pyelogram with only one or two X-ray exposures to rule out the possibility of obstruction.

## Chronic pyelonephritis

Chronic pyelonephritis is a chronic disease characterized by severe scarring of the kidneys resulting from persistent or recurrent infections usually in patients with vesicoureteral reflux.

#### Complications During Pregnancy

1. Acute pyelonephritis
2. Chronic microcytic anaemia refractory to iron therapy.
3. Chronic hypertension
4. Superimposed pre-eclampsia
5. Hyponatremia if on diuretic therapy
6. Glycosuria

Maternal and foetal prognosis depend on the extent of renal damage.

**Treatment**

Antibiotic prophylaxis is given.

Cap. ampicillin 500 mg or tablet nitrofurantoin 100 mg or capsule cephalexin 500 mg. All are given one capsule every night for the duration of pregnancy.

### Chronic renal disease and renal hypertension

See Chapter 32 for details.

## ACUTE KIDNEY INJURY (AKI) (FORMERLY ACUTE RENAL FAILURE)

Acute kidney injury renal failure is a rare complication in pregnancy in which there is a sudden decrease in renal function with oliguria over a period of hours or days. It is defined as increase in S.creatinine by ≥ 0.3 mg/dl within 48 hour or increase in Serum creatinine to ≥ 1.5 times of baseline over 7 days or urine volume < 0.5 ml/kg/hour for 6 hour. The various causes of acute renal failure in pregnancy are shown in Table 35.35.

**Table 35.35: Various Causes of Acute Kidney Injury (Renal Failure) in Pregnancy**

| |
|---|
| 1. Pre-eclampsia and related syndromes (most common) |
| 2. Obstetric haemorrhage<br>(a) Early pregnancy haemorrhage (abortion and ectopic pregnancy)<br>(b) Antepartum haemorrhage, especially abruptio placentae<br>(c) Postpartum haemorrhage<br>(d) Disseminated intravascular coagulation |
| 3. Infection<br>(a) Septic abortion<br>(b) Chorioamnionitis<br>(c) Puerperal sepsis |
| 4. Drugs: Especially non-steroidal anti-inflammatory drugs |
| 5. Renal diseases |
| 6. Post renal (obstructive uropathy) |

### Diagnosis

The diagnosis for acute renal failure is made by the following parameters.

1. Oliguria (Urine output <0.5 ml/kg/hr for > 6 hours )
2. Rising blood urea and creatinine by 50%
3. Metabolic acidosis
4. Hyperkalemia

### Types

Acute renal failure is of two types. They are:

1. **Acute Tubular Necrosis.** It is a less serious and reversible type of renal failure which is commonly associated with sepsis (septic abortion and puerperal sepsis), hypertension, nephrotoxic drugs, severe hyperemesis gravidarum and acute fatty liver of pregnancy. The kidney lesions in acute tubular necrosis are focal with most characteristic finding being dilatation and flattening of the epithelium of the distal convoluted tubules and the presence of pigmented casts in the lower part of the nephrons. Patient has a high-grade temperature with vomiting and diarrhea. Urine examination may be normal or show brown-pigmented casts or renal tubular cells. Patients may go into shock rapidly and may also have mild jaundice, pallor and cyanosis. Most patients respond to volume resuscitation and vigorous antibiotics in an intensive care unit.
2. **Renal Cortical Necrosis.** Bilateral cortical necrosis with a high mortality rate complicates 15–30% of all cases of acute renal failure associated with obstetric causes. The lesions in kidneys are focal, patchy, confluent or gross resulting from thrombosis of the segments of the renal vascular system. It is a serious type of renal failure which is often irreversible and is due to prolonged and severe renal vasospasm. It can occur in abruption, pre-eclampsia, prolonged intrauterine death, amniotic fluid embolism and acute fatty liver of pregnancy. There is oliguria which may lead to anuria, azotemia and consumptive coagulopathy. There may be extra renal manifestations like cardiac dilatation, congestive heart failure, lethargy and convulsions.

### Clinical features

Oliguria is an important sign of acute impaired renal function. She may appear to be well initially. Hence, a careful record of intake and output is important. The patient is warm to touch, thirsty and gradually becomes irritable and lethargic. There is rise in blood urea and serum potassium levels which causes muscular and ECG changes. In diuretic phase, there is excess of passage of urine but blood urea remains high.

### Management

Early diagnosis is very important as management depends upon the cause. Blood volume replacement is required for haemorrhage, control of BP and delivery for pre-eclampsia and stoppage of nephrotoxic drugs. Patient needs intensive care with adequate hydration. Accurate assessment of fluid balance with the use of a central venous pressure (CVP) line is important. Except for hemorrhagic shock where liberal fluids are given, fluids are only given to replace insensible losses and the previous hour urine output. Iatrogenic fluid overload must be avoided especially in pre-eclampsia where women are hypoalbuminemic and susceptible to pulmonary edema.

Infection should be controlled with appropriate antibiotics in septic abortion and puerperal sepsis.

Blood levels of electrolytes, creatinine and urea should be checked daily. The help of a nephrologist is sought. **Peritoneal** or **hemodialysis** is performed if needed with the aim of keeping blood urea nitrogen to 50 mg/dL. If not already delivered, delivery should be expedited after stabilization of her general condition.

**Table 35.36: Grades of CKD: Green, low risk (if no other markers of kidney disease, no CKD); yellow, moderately increased risk; orange, high risk; red, very high risk**

| Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012 | | | | Persistent albuminuria categories Description and range | | |
|---|---|---|---|---|---|---|
| | | | | A1 | A2 | A3 |
| | | | | Normal to mildly increased | Moderately increased | Severely increased |
| | | | | < 30 mg/g < 3 mg/mmol | 30–300 mg/g 3–30 mg/mmol | > 300 mg/g > 30 mg/mmol |
| GFR categories (ml/min/1.73 m²) Description and range | G1 | Normal or high | ≥ 90 | | | |
| | G2 | Mildly decreased | 60–89 | | | |
| | G3a | Mildly to moderately decreased | 45–59 | | | |
| | G3b | Moderately to severely decreased | 30–44 | | | |
| | G4 | Severely decreased | 15–29 | | | |
| | G5 | Kidney failure | < 15 | | | |

## CHRONIC KIDNEY DISEASE

Abnormality of kidney function or structure for ≥ 3 months detected on abnormalities in markers of kidney damage (albuminuria, hematuria, electrolyte imbalance), low GFR (< 60 ml/min/1.73 $m^2$ ) and detected on imaging.

### Effects of CKD on pregnancy

The maternal and feral outcome depends on:

- Nature and extent of renal disease
- Degree of hypertension
- Presence of coexisting infection and proteinuria

The chronic hypertension and preconceptional proteinuria worsens. There is a 50% increased risk of pre-eclampsia, 30% risk for developing end stage renal disease and has a perinatal mortality rate of about 6-10%.It poses an increased rates of cesarean section upto 60%. CKD is associated with increased risks for spontaneous abortion, therapeutic termination on medical grounds, spontaneous or iatrogenic preterm deliveries, foetal growth restriction secondary to hypertension or uraemia, osteomalacia and increased maternal mortality as well.

### Preconceptional counselling

1. Women with serum creatinine > 2 mg/dl and > 50% impairment of the kidney functions should avoid pregnancy.
2. One must remember the diagnosis of pregnancy in these women is often delayed as they have irregular cycles and impaired fertility. One must prescribe them contraception like progesterone IUCD, progesterone only pills or subnormal implants to avoid conception in high risk women.
3. Genetic screening to be done for inheritable renal disorders,
4. Stop ACE inhibitors and angiotensin receptor blockers pre-conceptionally and optimise the blood pressure <140/90 mm hg and good glycemic control.
5. Stop Mycophenolate Mofetil, Methotrexate and Cyclophosphamide if they are taking. Instead drugs safe for use during pregnancy are Prednisolone, Azathioprine, Cyclosporine, Hydroxychloroquine and Tacrolimus.

### Antenatal management

1. Consider starting Aspirin (75-150 mg)
2. Low molecular weight heparin to be started if urinary protein >300 mg/mmol (nephrotic range )
3. Continue antihypertensives safe for pregnancy like labetalol, nifedipine or methyldopa and maintain BP < 135/85 mm hg.
4. Avoid the use of NSAIDs.
5. Chronic anaemia to be treated with erythropoietin.
6. Women receiving peritoneal dialysis prior to pregnancy need to convert to hemodialysis during pregnancy. Lower levels of sodium and bicarbonate should be used in the dialysate fluid as to mimic physiological changes of pregnancy. Increase the number of hours and sessions per week. Foetal monitoring to be done after each session.

   Dialysis to be initiated when serum urea > 17-20 mmol/L.

## PREGNANCY IN RENAL TRANSPLANT PATIENT

More women are expected to come for pregnancy with more liberal use of renal transplants. They should delay pregnancy for 1–2 years after transplantation to allow graft function to stabilize and immunosuppression reach maintenance levels. Their renal function should be stable (S. creatinine < 1.5 mg/dl, proteinuria < 500 mg/dl) with no hyprtension before trying for conception. These women are immunosuppressed and are therefore more prone to infection. Out of various immunosuppressive drugs, prednisolone, azathioprine and calcineurin inhibitors (tacrolimus and cyclosporine) are all considered safe in pregnancy and lectation **(level II evidence)**. Mycophenolate mofetil (MMF) is contraindicated in pregnancy causing

upto 23% birth defects. Rapamyain is also contraindicated due to insufficient data about it. Women on cyclosporine should not breastfeed. Thre is increased risk of preterm labour (upto 62%), pre-eclampsia (22%), gestational diabetes mellitus (5%), rejectin episodes (2–5%), foetal growth restriction, infection with polyoma virus hominis I (called BK virus). Close surveillance is needed in pregnancy. Any bacteriuria is treated. For recurrent UTI, suppressive therapy is given throughout pregnancy. Renal function tests are done regularly. Any complication like GDM or pre-eclampsia are energetically treated. Regular foetal monitoring is done. Vaginal delivery can be tried. Cesarean delivery for obstetric indication or for obstructed labour by transplanted kidney.

## Key Points

1. Urinary tract infections (UTIs) are common in pregnancy. Asymptomatic bacteriuria (> 1,00,000 [$10^5$] organisms/mL) requires treatment to prevent pyelonephritis and premature labour. Most penicillins (ampicillin, amoxicillin), cephalosporins and nitrofurantoin are safe in pregnancy. Treatment is given orally for 3–5 days for asymptomatic bacteriuria, for 7 days for cystitis and for 10–14 days for pyelonephritis (first intravenously followed by oral drugs).
2. Kidneys may be affected by primary renal diseases or secondary due to obstetric diseases like pre-eclampsia and abruptio placentae.
3. Acute renal failure is rare but can result in serious condition such as septic abortion, abruptio placentae and pre-eclampsia. The early acute tubular necrosis is reversible if energetically treated while the latter acute cortical necrosis may be irreversible.
4. Women with renal transplant which is functioning well can have successful pregnancies. They should, however, delay the pregnancy until 2 years after transplantation. The common immunosuppressives (predinsolone, cyclosporine, azathioprine) can be safely taken in pregnancy.
5. Women with chronic renal diseases with severe renal impairment (high creatinine and urea) are advised against pregnancy until their renal disease is well controled as renal impairment tends to worsen during pregnancy.

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## Examination Questions

A. Enumerate the physiological renal and urinary tract changes in pregnancy. Define asymptomatic bacteriuria. Enumerate its complications. How will you treat a case of asymptomatic bacteriuria during pregnancy?

B. Define acute pyelonephritis in pregnancy. Enumerate its clinical features. How will you manage a case of acute pyelonephritis during the third trimester of pregnancy?

C. Define acute renal failure in pregnancy. Enumerate its causes and its types. How will you manage a case of renal failure at 34 weeks gestation?

D. Write short notes on:

1. Asymptomatic bacteriuria in pregnancy (Many universities)
2. Cystitis in pregnancy
3. Complications of acute pyelonephritis in pregnancy
4. Treatment guidelines for UTIs in pregnancy
5. Renal failure in pregnancy
6. Pregnancy with renal transplant

# (vii) LIVER DISORDERS IN PREGNANCY

Liver is the main organ of metabolism. Overall incidence of liver disorders is 1–4 per 1000 pregnancies. The various types of liver diseases in pregnancy are shown in Table 35.37.

## OBSTETRIC CHOLESTASIS
## (Synonyms: Intrahepatic Cholestasis, Icterus Gravidarum, Recurrent Jaundice of Pregnancy)

### Introduction

Obstetric cholestasis is a hepatic disorder specific to pregnancy which is characterized by pruritis affecting the whole body but particularly the soles and palms with abnormal hepatic function tests. **It is the second most common cause of jaundice in pregnancy after hepatitis and the most common pregnancy specific liver disorder.**

### Prevalence

The prevalence is about 0.2 to 2% being more common in the Indian Subcontinent.

### Etiology

1. **Genetic:** it has a genetic predisposition. Genetic aberrations in the p23 region of the chromosome 21 are associated with membrane dysfunction of the biliary system. Mutations in the the ABCB4 (ATP Binding Casette Transporter 4), MDR3 (Multidrug resistance protein 3) and BSEP (Bile salt export transporter) have been associated with obstetric cholestasis.
2. **Hormonal:** It is associated with high circulating levels of oestrogen as the condition recurs with subsequent pregnancies and during oral pill usage. High levels of sulphated progestrone and 17 beta estradiol glucoronide also inhibit the bile salt export transporter responsible for causing cholestasis.
3. **Environmental:** Selenium acts as a co-factor for the oxidative liver enzymes and thus selenium deficiency results in defective bile formation and secretion.

### Clinical Features

The clinical features commonly present in the third trimester at around 30–32 weeks gestation. Generalized pruritis is the main symptom. It is most severe in night and is more common in palms, soles and trunk. There may be slight jaundice. There are raised bile acids in most of them with levels at least thrice than normal. Liver function tests demonstrate mildly elevated hepatic transaminases (ALT and AST), moderately elevated serum alkaline phosphatase and serum bilirubin (up to 5 mg%). There may be associated dark urine, pale stools, steatorrhea and malaise.

### Diagnosis

The condition is diagnosed by unexplained raised levels of serum transaminases and Gamma glutamic transaminase. While defining raised levels of liver function tests -upper limit of the pregnancy specific range should be used which is 20 % lower than the non-pregnant values. Diagnosis is established when serum bile acids >10 umol/L which is the most specific marker for the diagnosis. Bile acid measurement should be done on a random or non-fasting sample. The diagnosis is established only after ruling out other causes of raised liver functions in pregnancy for example viral hepatitis, liver ultrasound to rule out primary biliary cirrhosis and after ruling out pre-eclampsia or AFLP. In addition other causes of pruritis in pregnancy like eczema, atopy should have been excluded.

Differential diagnosis is from acute or chronic viral hepatitis, gall stones, chronic active hepatitis and primary biliary cirrhosis.

**Table 35.37: Various Liver Diseases in Pregnancy**

| | |
|---|---|
| **Group I** | **Liver disorders caused by pregnancy**<br>1. Obstetric cholestasis (Intrahepatic cholestasis)<br>2. Acute fatty liver of pregnancy<br>3. Hyperemesis gravidarum induced hepatic damage (see Chapter 12 for details)<br>4. Liver damage due to severe pre-eclampsia, eclampsia and HELLP Syndrome (see Chapter 32 for details) |
| **Group II** | **Pregnancy associated liver diseases (Liver disease coincidental to pregnancy)**<br>1. Acute viral hepatitis<br>(a) Hepatitis A, B, C, D, E, G<br>2. Drug induced liver damage.<br>(a) Isoniazid (b) Rifampicin (c) Phenothiazine<br>3. Hemolysis induced liver damage and jaundice including mismatched blood transfusion, malaria, severe septicemic infection |
| **Group III** | **Chronic liver diseases preceding (antedating) pregnancy**<br>1. Chronic hepatitis<br>2. Liver cirrhosis<br>3. Budd Chiari syndrome<br>4. Congenital hyperbilirubinemia<br>5. Autoimmune hepatitis<br>6. Liver tumors<br>7. Liver transplantation |
| **Group IV** | **Diseases of gall bladder**<br>1. Cholecystitis<br>2. Cholelithiasis |

### Effect on pregnancy

With serum bile acids > 40umol/L, there is an increased incidence of premature labour, foetal growth restriction, meconium stained liquor, foetal distress (cardiotocographic abnormalities) in labour and rarely foetal death. With

levels >100 umol/L, there is an increased risk of sudden still births. The exact cause of adverse foetal effects is not known and it does not correlate with severity of symptoms and liver function tests abnormalities. Adverse outcome is more with raised bile acid levels. However, previous animal studies have shown the possible cause of bile acid induced damage is the toxic effect of bile acids on foetal myocardium resulting in anoxic damage and dysrhythmia.

### Management

The woman should be counselled about the various risks of intrahepatic cholestasis. Liver function tests (LFTs) and clotting time should be regularly monitored. Control of symptoms should be achieved with a combination of antihistamines like chlorpheniramine maleate 4 mg BD, fexofenadine 120 mg once a day with local emollients like calamine lotion. The drug ursodeoxycholic acid (UDCA) is the drug of choice in dose of 300–450 mg two to three times daily for better foetal outcome (American College of Obstetricians and Gynecologists, 2015). Recent meta-analysis has suggested that UDCA relives pruritis, lowers bile acid and serum enzyme levels and may reduce neonatal complications like preterm birth, foetal distress, respiratory distress syndrome and neonatal admision in intensive care units (2012). It is continued until delivery. S-adenyl-methionine and Cholestyramine are not recommended. Regular foetal surveillance with ultrasound and non-stress test should be done. Recently, use of rifampicin with UDCA is used in refractory cases as second line. Rifampicin helps in bile excretion by increasing MRP2 ( multi drug resistance protein 2 ) expression. It is started at a dose of 600 mg/day. However further trials and results of TURRIFIC trial on the use of rifampicin are still awaited.

Labour should be induced at 38 (37 completed weeks) weeks gestation to avoid foetal adverse outcome and sudden intrauterine death. However, the Society of Maternal and Foetal Medicine in their latest guidelines have suggested termination at 36 0/7 weeks when serum bile acids >100umol/L given that they have increased risk for still birth (Grade 1B). Vitamin K should be given to mothers to reduce the risk of postpartum haemorrhage. Vaginal delivery is the aim but cesarean delivery is performed for foetal indications.

After delivery, LFTs return to normal and there is no permanent deleterious effect on maternal liver functions. However, symptoms can recur with menstruation and *with combined oral pills which should be avoided in them.* **Recurrence risk of cholestasis in subsequent pregnancies is 90%.**

## ACUTE FATTY LIVER OF PREGNANCY (AFLP)

Acute fatty liver of pregnancy (AFLP) was also previously called acute yellow atrophy of liver. It is a rare (incidence of 0.005%) pregnancy specific liver disease which is a serious and potentially fatal disease complicating a normal pregnancy in **the third trimester.** It is closely related to pre-eclampsia sharing many features and pathophysiology with it. It is more common in primigravidas and in twin pregnancies.

### Etiology

It is caused due to the deficiency of the foetal Long chain hydroxyacyl coenzyme A dehydrogenase which results in accumulation of free fatty acids and oxidative stress and lipotoxicity in the mother. This results in micro vesicular steatosis and mitochondrial dysfunction.

### Symptomatology

Patient usually presents with nausea, vomiting, malaise, loss of appetite and weight, pain in the epigastrium and right upper quadrant with worsening jaundice. Many women (about 50%) have hypertension and pre-eclampsia.

### Diagnosis

Swansea criteria is used for the diagnosis of AFLP as given below:

**Table 35.38: Swansea Criteria for the Diagnosis of Acute Fatty Liver of Pregnancy**

| Six of the features below are required for the diagnosis | |
|---|---|
| Clinical features | • Nausea and vomitng<br>• Abdominal pain<br>• Encephalopathy<br>• Polyuria or polydipsia |
| Laboratory features | • Bilirubin > 0.8 mg/dL<br>• Hypoglycemia < 72 mg/dL<br>• WBC > 11 × $10^9$/L<br>• AST or ALT > 42 units/L<br>• AKI or Cr > 1.7 mg/dL<br>• Coagulopathy or PT > 14 sec<br>• Ammonia > 47 µmol/L<br>• Urate > 340 µmol/L |
| Ultrasonographic features | Ascites or echogenic liver |
| Histologic features | Microvesicular seatosis on liver biopsy |

AKI: acute kidney injury; ALT: alanine transaminase; AST: asparatate transaminase; Cr: creatinine, PT: prothrombin time; WBC: white blood cell count.

Acute fatty liver of pregnancy is a serious disease but is self limiting with symptoms subsiding immediately after delivery and recovering in several days in puerperium. It is an important cause of hepatic coma and precoma and is associated with high fatality rate due to multiorgan failure. Perinatal and maternal mortality and morbidity are increased.

### Treatment

Such women should be treated in intensive care unit by an obstetrician, hepatologist or physician and anesthetist. Intensive supportive measures like adequate intravenous glucose are required. Delivery should be expedited following adequate correction of any hypoglycemia or coagulopathy with concentrated (50%) dextrose, fresh

frozen plasma, cryoprecipitates, platelets and intramuscular vitamin K. Vaginal delivery is preferred. Epidural anesthesia can be given for cesarean delivery if coagulation profile is normal. The patients should be carefully watched for any hepatic encephalopathy and should be treated in a liver unit. The offspring should be screened for hypoketotic hypoglycaemic liver failure. Family should be counselled about 25% chance of recurrence and testing for (long-chain 3-hydroxyacyl (LCHAD) COA) should be offered. In absence of improvement in LFTs after delivery, liver transplantation may be considered.

## VIRAL HEPATITIS IN PREGNANCY

Viral hepatitis is the most common liver disease encountered during pregnancy and **is also the most common cause of jaundice in pregnancy** in tropical countries (90% in India). Malnutrition, poor socio-economic background and inadequate antenatal care are responsible for the high prevalence.

### Types of viral hepatitis

There are six viral agents which cause hepatitis and some other viruses which can affect liver.

1. Hepatitis A
2. Hepatitis B
3. Hepatitis C
4. Hepatitis D
5. Hepatitis E
6. Hepatitis G
7. Epstein Barr Virus
8. Echovirus
9. Yellow fever virus

Except for Hepatitis B, which is a DNA virus, all others hepatitis viruses are RNA viruses.

#### Hepatitis A Virus (HAV)

Hepatitis A virus (HAV) is transmitted by feco-oral route through contaminated food and water. Incubation period is 2 to 7 weeks. Patient presents with acute symptoms like nausea, vomiting, anorexia, headache and flu-like illness. Diagnosis is confirmed by detection of IgM antibody to HAV. It is usually a self-limiting disease with quick recovery and only rarely ending into hepatic failure. There is no chronic carrier state and perinatal transmission is rare. The virus is not teratogenic. There is risk of transmission to newborn due to fecal contamination during delivery. Treatment is the same as in non-pregnant state and involves rest, hydration and balanced diet with high carbohydrates and less fats. Admission to hospital may be needed till the patient eats well. Immunoglobulin (IgG) administration may provide immunoprophylaxis. A single antigen vaccine is safe in pregnancy.

#### Hepatitis B virus (HBV)

Hepatitis B virus (HBV) is a worldwide public health problem. It is transmitted by parental route and is seen in intravenous abusers, homosexuals, women receiving blood products and healthcare workers. It is also transmitted by sexual contact, vertical transmission and through breast milk. It has a longer incubation period of 6 weeks to 6 months. Hepatitis B leads to serious sequelae like chronic active hepatitis, cirrhosis and hepatocellular carcinoma in about 10% women. Ideally all pregnant women should be screened for serum HBsAg (surface antigen). Mother to child (vertical) transmission of the virus is 20% which increased to (70–90%) in women who are HBsAg and HBeAg positive.

Neonatal transmission occurs in about 5–8% infants born to HBsAg positive mothers and mostly (95%) at delivery while the risk of transmission is only 10–20% if mother is only HBsAg positive. An infant infected with HBV at birth (vertical transmission) has a 90% chance to progress to chronic infection in contrast to 40% chance for horizontal transmission between 7–12 months of life and only 10–20% for infections acquired after 1 year of age. Chronic carriers develop chronic hepatitis, hepatitic cirrhosis or hepatocellular carcinoma in up to 25% cases (one fourth). Those who are HBeAg positive are at greatest risk.

##### Presentation

Most infected women are asymptomatic. Mothers with acute infection present with non-specific symptoms like anorexia, nausea, vomiting, malaise and low grade fever (uncommon) with or without arthralgia, skin rash and rarely jaundice. Liver may be enlarged and tender on palpation. Infection usually resolves fully in 90% cases. Out of rest 10%, about 1% may die of fulminant hepatitis while others may become chronic carriers and are at risk of long term complications.

##### Diagnosis

Most cases are diagnosed by routine prenatal serological screening for HBsAg. It is suspected from symptoms and signs. Serum bilirubin, serum transminases (ALT and AST) are very much raised. Serology is performed to detect various viral markers. Confirmation of diagnosis is made by demonstration of HBsAg. HBeAg and antibody (IgM initially and IgG later) to hepatitis B core antigen (HBcAg). Those with HBeAg positivity are highly infective.

### Management

Hepatitis B vaccine and immunoglobulin to be given to mother and also to foetus at birth. The foetus should be given the immunoglobulin within 12 hrs after birth. Antiviral therapy (Tenofovir 300 mg/day) is started if the woman has cirrhosis, raised ALT and HBV DNA > $2 \times 10^6$ copies/ml (Society of Maternal and Foetal medicine, 2016) from 28 weeks to reduce perinatal transmission. Monitor HBV DNA every 2 months after starting Tenofovir and Liver function tests every month. Tenofovir can be stopped 4–12 weeks after birth until mother needs long term treatment.

### Hepatitis C Virus (HCV)

It is a single stranded RNA virus. Its transmission is similar to hepatits B (blood borne). It is prevalent in intravenous drug abusers, hemophiliacs and women with high risk sexual behaviour. About 66% patients show chronic active hepatitis and 20 to 30% may progress to cirrhosis. All blood and blood products are screened for Hepatitis B and C virus now. Vertical transmission occurs in 3 to 6% fetuses with high maternal viremia, intrapartum exposure to viral contaminated maternal blood increasing the perinatal transmission. Diagnosis is by detection of antibody to HCV. There is no vaccine for HCV. Interferon and ribavirin are avoided in pregnancy due to teratogenicity.

All women or male partners receiving Ribavarin to defer pregnancy 6 months after completion of treatment. Breastfeeding is not contra-indicated except if the mother has cracked nipples/bleeding nipples or HIV co-infection.

### Hepatitis D virus (HDV)

Hepatitis D virus (HDV) exists as co-infection with Hepatitis B virus. The co-infection of Hepatitis B and D is serious and increases the risk of cirrhosis. It is transmitted vertically. Hepatitis B vaccination prevents hepatitis D too.

### Hepatitis E Virus (HEV)

Hepatitis E virus (HEV) is water borne which spreads by the feco-oral route. It is the most common cause of epidemic viral hepatitis in India and is more prevalent in the 3rd trimester of pregnancy. **Its infection is more serious and may lead to fulminant hepatitis and hepatic failure.** Postpartum haemorrhage and coagulation failure are the common complications. In presence of hepatic failure, there is high maternal mortality (20–60%). In fact, Hepatitis E infection which causes hepatic coma is one of the common causes of indirect maternal mortality in India, especially in North India. Perinatal mortality is also very high (25–75%) due to prematurity and intrauterine foetal death. In a recent meta-analysis on 4000 patients from Asia and Africa, Jin et al reported maternal mortality rate of 21% and foetal mortality rate of 34% (2016). Recombinant HEV vaccine has been licensed and used in China in non-pregnant state but not yet used in India and other countries.

### Hepatitis G Infection

Hepatitis G infection is blood borne infection which coinfects with Hepatitis B and C in women with history of intravenous drug abuse and can cause vertical transmission.

## CLINICAL FEATURES OF VIRAL HEPATITIS IN PREGNANCY

Most women with viral hepatitis in pregnancy have got almost similar clinical presentation such as:

1. Prodromal stage in which woman complains of anorexia, nausea, vomiting fatigue, general malaise, myalgia, low grade fever and pain in the right upper quadrant of abdomen.
2. Appearance of jaundice 1–2 weeks later with dark urine and clay-colored stool.
3. Mild tender hepatomegaly
4. **Blood tests.** Slight leucopenia, deranged liver function tests with elevated liver transminases, bilirubin and serum alkaline phosphatase. Coagulation profile may become deranged with prolongation of prothrombin time (PT) and partial prothrombin time (PPT).
5. Spider angioma may develop.
6. Serological tests for various hepatitis viral antigens and antibodies.

Acute illness usually resolves rapidly within 2 to 3 weeks. Up to 10% cases of Hepatitis B and C become chronic while 1 to 3% develop into acute fulminant hepatitis.

## DIAGNOSIS

Viral hepatitis is suspected from symptomatology of the woman and signs but confirmed by the laboratory tests. Serum bilirubin is more than 15 mg, transaminases > 1000 units with prolonged PT. Serological markers of various specific viruses like anti HAV, IgM, HBsAg, anti HBc IgM, polymerase chain reaction (PCR) of Hepatitis C and E virus, anti HB-IgM. Liver biopsy if performed may show hepatocellular injury and inflammatory infiltration.

## Management

### Prophylaxis

Availability of safe drinking water and improved sanitation and personal hygiene can prevent most cases of hepatitis transmitted by feco-oral route (hepatitis A and E).

Using disposable syringes and screening blood donors for HBsAg and Hepatitis C virus can help to prevent cases of hepatitis viruses which are transmitted by parental route. Vaccination is available for Hepatitis A and B and should be used by high risk women. Pregnant women who are exposed to Hepatitis A virus infection should receive immunoglobulin 0.01 mL/kg within 2 weeks of exposure and should also receive Hepatitis A vaccine (0.06 mL intramuscularly single dose) which is safe in pregnancy. Women at high risk and healthcare professionals should receive Hepatitis B vaccine and should use universal precautions (see Chapter 36 for details) to avoid infection. Infants born to HBsAg sero positive women are administered. Combined immunoprophylaxis-Hep B vaccine is given at 12 hours of birth then at 6, 10 and 14 weeks. Ig (HBIG) 100 IU is given on opposite thigh. The baby is tested for HBsAg and Anti HbS antibody at 18 months. These measures can prevent infection in the infant from HBV infection in 80–90% cases. Breastfeeding is allowed.

### Treatment of viral hepatitis

As there is no specific treatment available for viral infection, supportive care is used. Most women can be managed on out patient basis. The measures are as follows:

1. Hospitalization in acute phase with adequate rest. Physical isolation is not necessary.

2. Restricted physical activity.
3. Balanced diet which includes high carbohydrate and low fat diet.
4. Intravenous dextrose infusion (10%)
5. Drugs:
   (a) Oral neomycin and lactulose which were popular in past are not routinely given in current practice.
   (b) Avoidance of hepatotoxic drugs.
   (c) Control of severe pruritis by cholestyramines or ursodeoxycholic acid (300 mg twice daily).
   (d) Corticosteroids may be rarely required.
6. Proper handling of bed pan in Hepatitis A and E infections and blood and body fluids in Hepatitis B and C infections.
7. Healthcare workers should use universal precautions and should avoid blood and body fluid contamination and must be careful to avoid needle stick injury.
8. Foetal surveillance and monitoring
9. During labour, vaginal delivery should be the aim. Foetal scalp electrodes and foetal blood sampling should be avoided. Vitamin K 0.5 mg intramuscularly is not routinely administered but given if prothrombin time is prolonged. Complications like hypokalemia, hypoglycemia and hypocalcemia are diagnosed and treated early. Blood and fresh frozen plasma are used in case of postpartum haemorrhage. Breastfeeding is allowed.

## FULMINANT HEPATITIS (HEPATIC COMA, HEPATIC FAILURE)

Fulminant hepatitis is seen in B, D and E ( most common cause in India) hepatitis. It is a serious condition with high maternal and perinatal mortality. Patient is confused and disoriented and may develop encephalopathy or may go into coma. Liver is usually small, prothrombin time is excessively prolonged and disseminated intravascular coagulation (DIC) may complicate the case. Serum bilirubin rises rapidly. There can be generalized edema and ascites. Various complications can be cerebral edema, compression of brain stem, gastrointestinal bleeding, sepsis, renal failure, shock and respiratory failure. There is very high mortality (10–40% maternal, 25–75% perinatal). Treatment is supportive like for hepatitis and acute fatty liver disease. Most women go into labour of their own.

Vaginal delivery is preferred. There is risk of postpartum haemorrhage.

Liver transplantation may be life saving. Interferon therapy can be given in selected patients.

## Key Points

1. Various pregnancy complications like hyperemesis gravidarum, severe pre-eclampsia and HELLP syndrome interfere with liver function tests.
2. Incidental liver diseases like hepatitis can occur in pregnancy contributing significantly to maternal and perinatal mortality and morbidity. Infact, hepatitis is an important cause of maternal mortality in India causing indirect maternal deaths. Hepatitis E is particularly notorious causing hepatic coma and high mortality.
3. Intrahepatitic cholestasis (obstetric cholestasis) is pruritis, abnormal liver function tests causing itching over soles, palms and rest of body. Ursodeoxycholic acid improves symptoms and liver function tests and is the drug of choice. Foetal surveillance is done. Usually the women with obstetric cholestasis are induced at 38 weeks of gestation to avoid perinatal complications.

## Suggested Reading


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# (viii) NEUROLOGICAL AND PSYCHIATRIC DISORDERS IN PREGNANCY

## EPILEPSY

Epilepsy complicates 1 in 200 pregnancies and is an important cause of seizures. *It is defined as a paroxysmal disorder of the central nervous system with an abnormal neuronal discharge with or without loss of consciousness.* In epilepsy, there is predisposition to recurrent unprovoked seizures.

### Effect of pregnancy on epilepsy

During pregnancy, 35% women show increased frequency of seizures, 15% show decreased frequency while 50% have no change. The increased frequency is probably due to subtherapeutic levels of drugs in pregnancy due to increased intravascular volume lowering serum drug levels, increased glomerular filtration enhancing drug clearance, induction of liver and placental enzymes increasing drug metabolism. Nausea and vomiting during pregnancy and decreased gastrointestinal motility and self-discontinuation of medicines by the patients due to fear of teratogenicity may also play a role in increased frequency of seizures.

### Effect of epilepsy on pregnancy

1. Increased frequency of congenital malformations
2. Spontaneous abortions
3. Foetal growth restriction
4. Oligohydramnios
5. Intrauterine death
6. Preterm birth
7. Haemorrhage
8. Hypertensive disorder
9. Cesarean delivery
10. Maternal death (increased mortality)

#### Congenital Malformations

There is increased frequency of congenital malformations in epilepsy, especially in those on anticonvulsant medicines. The prevalence of congenital malformations is about 5% in women on anticonvulsants against the background risk of 1.8%. Although it is agreed that epileptic mothers are more prone to have congenital malformations whether or not they are on anticonvulsant therapy, recent American studies show that untreated epileptics do not have increased chances of congenital malformations.

#### Foetal Anticonvulsant Syndrome

Earlier foetal anticonvulsant syndrome was called **foetal hydantoin syndrome.** It is now known that most antiepileptic drugs can cause the abnormalities.

The major abnormalities include: cleft lip and palate, microcephaly, neural tube defects, congenital cardiac defects, foetal growth restriction and developmental delay.

The minor congenital abnormalities include: low set abnormal ears, flat nasal bridge, hypertelorism, nail and distal digital hypoplasia, epicanthic folds and long philtrum.

It is known that polytherapy (use of more drugs) carries a higher risk of congenital malformations than monotherapy. It may be due to folate deficiency as these drugs interfere with folate metabolism. Sodium valproate is known to cause more chances of neural tube defects. Carbamazepine though previously considered the safest can also cause increased rate of congenital malformations.

There is also increased risk of hemorrhagic disease of newborn due to reduction in Vitamin K dependent clotting factors by anticonvulsants. Other effects of anticonvulsant drugs include neonatal withdrawal effects like jitteriness, convulsions and poor feeding. They are more common with phenobarbitone and are less often seen these days due to less use of phenobarbitone. They can also be seen rarely with carbamazepine and valproate.

### Pre-pregnancy counselling

Pre-pregnancy counselling is very important for epileptic mothers as they can be counselled not to conceive unless their seizures are well under control. They should be seen by obstetrician and neurologist.

The International League Against Epilepsy in their official report published in 2014, have endorsed the new revised defintion of epilepsy as disease of the brain with:

1. At least two unprovoked (or reflex) seizures experienced > 24 hrs apart or
2. One upprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk after 2 unprovoked seizures, occuring over next 10 years (at least 60%) or
3. Diagnosis of an epilepsy syndrome.

Wherever possible epilepsy should be managed using monotherapy.

As per the recommendation of the American Academy of Neurology, 2005 discontinuation of anti-epileptic medication may be considered before pregnancy in women:

1. Who have been seizure free for 2–5 years
2. Display a single seizure
3. Have a normal neurological examination and normal intelligence and
4. Show EEG results that have normalized with treatment.

**Avoid valproate.** Trimethadione is contra indicated.

Folic acid 5 mg should be taken each day during the periconceptional period by women who are taking antiepileptic drugs especially valproate and carbamazepine.

The woman should be counseled about the risk of adequate control of epilepsy, effect of antiepileptic drugs on fetus and the risk of epilepsy in the offspring which is 4% if one parent has idiopathic epilepsy, 10% if one parent and a sibling are affected, but increases to 15% if both the parents have epilepsy. The woman is advised to defer pregnancy till one year of seizure free interval.

**Table 35.39: Dosage and Effects of Anti-epileptic Drugs**

| Anti-epileptic drugs | Usual dosage (per day) | Maternal effects | Foetal effects | Malformation rate (%) |
|---|---|---|---|---|
| 1. Carbamazepine | 600–1200 mg per day in three divided doses | Drowsiness, leucopenia, slight hepatotoxicity, ataxia | Rarely craniofacial and neural defects | 1–2% |
| 2. Phenytoin | 150–300 mg in two divided doses | Gingival hyperplasia, megaloblastic anaemia, nystagmus, hirsutism | Foetal hydantoin syndrome, Craniofacial anomalies, foetal growth restriction, cardiac defect, clefts, finger nail hypoplasia developmental delay | 5–11% |
| 3. Phenobarbitone | 60–180 mg daily at night | Drowsiness | Clefts, cardiac anomalies, urinary tract anomalies | 10–20% |
| 3. Valproic acid | 500–2000 mg in three divided doses | Drowsiness, ataxia, alopecia, thrombocytopenia | Neural tube defects, craniofacial and skeletal defects, low cognitive performance | 10% with mono therapy, higher with polytherapy |
| 4. Lamotrigine | 300–500 mg in two divided doses | | Increased rate of cleft lip and palate | 1% |
| 5. Levetiracetam | 1000–3000 mg in two divided doses | | Skeletal abnormalities, impaired growth | 1% |
| 6. Topiramate | 100 mg per day | | Cleft lip and palate | 2–3% |

## Antenatal care

The main goal of pregnancy management is prevention of seizures by treating nausea and vomiting, avoiding stimuli provoking fits and emphasizing the compliance. As a principle, lowest dosage of anti-convulsant should be given. Serum levels of drugs are not routinely performed in all the cases, but should be done in selected cases like suspected non-compliance, increased seizure activity, use of multiple drugs and presence of side effects of drugs. The woman should be regularly seen in antenatal clinic and in neurological clinic. Quadruple test and screening for foetal anomalies should be performed including detailed anomaly scan in the second trimester and foetal cardiac scan at 20–22 weeks gestation. Drug blood levels may be performed in selected cases. Anti-epileptic drugs must be given in all the indicated cases.

### Anti-epileptic Drugs

1. The aim is to start treatment with monotherapy (single drug) in the lowest possible therapeutic dosage.
2. Various drugs are shown in Table 35.39.

Newer medications **gabapentin, lamotrigine, levetiracetam** and **topiramate** though appear safe, lack detailed data on their use in pregnancy. Initial results on the use of lamotrigine and levetiracetam in pregnancy have shown very promising results with only 1% congenital malformation rate and very high efficacy and they are becoming drugs of choice. **Levetiracetam is considered the best drug in pregnancy with least malformation rate followed by Lamotrigine.** The woman should be regularly seen in antenatal clinic and should be routinely prescribed folic acid 5 mg per day along with iron therapy. Weight gain, symphyseofundal height should be performed on each visit. Foetal growth and well-being should be monitored on each visit by ultrasound scan as foetal growth restriction is more common, Woman may be given Vitamin K in the last week of pregnancy to prevent hemorrhagic disease of newborn.

## Labour management

Vitamin K is given to the woman in the last week of pregnancy and Injection of Vitamin K 1 mg IM is given to the neonate on birth to prevent haemolytic disease of the newborn. Prophylactic Clobazam can be given if there is a previous history of seizure in labour or a recent history of seizure provoked by stress or sleep deprivation. Anti-epileptics to continue in labour, and if patient cannot take orally, a parenteral alternative should be given.

For seizure control during labour, patient can be given Inj. Lorazepam 4mg bolus followed by further dose after 10-20 mins or Inj. Diazepam 5-10 mg slow IV. Continuous electronic foetal monitoring must continue. Vaginal delivery is the preferred route for delivery and cesarean section to be done for routine obstetric indications or uncontrolled seizures. For labour analgesia, TENS, entonox and diamorphine can be used. However, while GA is given Pethidine, Sevofluorane and Ketamine are avoided.

## Puerperium

Usually lower doses of anti-convulsants are required in puerperium. So readjustment of dose is required. Although the drugs are secreted in breast milk and can cause sedation

and withdrawal effects (more with phenobarbitone and benzodiazepines), breastfeeding is encouraged. Vitamin $K_1$ (phytonadione) 1 mg intramuscularly is given to all newborns to prevent hemorrhagic disease of the newborns.

### Contraception

Enzyme inducing anti-epileptics (Carbamazepine, Phenytoin, Phenobarbitone, Topiramate). These drugs reduce the efficacy of contraceptive pills, minipills and depot provera injections by simulating the liver enzymes. Hence they should be given LNG-IUS or Cu-T as the safest options. Depot provera should be given every 10 weeks instead of every 12 weeks. Combined pills, patches and rings should not be prescribed due to high failure rates but for patients who still choose to use COC, their contraceptive efficacy can be increased by using pills with high dose of oestrogen (50 ug), reducing the drug free interval from 7 days to 4 days or using three packs back to back and using an additional barrier contraceptive alongwith.

Non-enzyme inducing anti-epileptic drugs like sodium valproate, Levetiracetam and Gabapentin. Patients taking these can be offered all forms of contraceptions safely.

Women taking Lamotrigine mono therapy with oestrogen containing contraceptive have an increased risk of seizures.

Epileptic women should be careful and should have help for themselves and the baby.

## Status epilepticus

Uncontrolled repeated seizures are known as status epilepticus. Management is the same as in non-pregnant state. Intravenous 20 mg diazepam or phenytoin (50 mg per minute up to 180 mg) should be given. Suction of secretions and maintenance of airway should be done Any precipitating factors like hypoglycemia, infection and non-compliance should be treated energetically. For respiratory depression, endotracheal intubation and positive pressure ventilation may be needed. Neuromuscular blockage or general anesthesia may be required for uncontrolled seizures.

# MISCELLANEOUS DISORDERS

## Multiple sclerosis (MS) in pregnancy

Multiple sclerosis is a multifocal autoimmune disease of the central nervous system (CNS) with relapsing and remitting natural history. Patients may present with optic neuritis (loss of vision, diplopla), muscle weakness, dysarthria, ataxia, hyperreflexia and bladder dysfunction.

Pregnancy usually has no effect on it and vice versa. However, relapses are more common in puerperium.

The primary pathology involves T-cell mediated autoimmune destruction of myelin synthesising oligodendrocytes. The trigger is believed to be induced by environmental agents such as micro-oganisms. Of relevance, in periconceptional counselling is the fact that there is familial recurrence of the order of 15% and there is a 15-fold increased chance of the offspring being affected.

### Effects of Pregnancy on MS

Numerous studies have confirmed a reduction in relapse risk during pregnancy (≈ 70%) albeit with a significantly higher relapse ratio in the postpartum period. Factors predicting postpartum relapse include escalated relapse rates prior to and during pregnancy and a high score for MS disability. Breastfeeding does not augment risk.

### Effects of MS on Pregnancy

Most authors have concluded that MS, does not adversely affect feto maternal outcome significantly. However, women with lesions at or above T6 remain at risk for autonomic dysreflexia.

### Management During Pregnancy

Medical treatment is with steroids (prednisolone), beta interferon, baclofen and azathioprine.

In addition to these disease modifying therapies such as a combination of natalizumab and Interferon (IFN) $\beta_1$a significantly reduce relapse rates with limited but reassuring data regarding use in first trimester. When used in pregnancy the neonate, however, should be monitored for thrombocytopenia and anaemia. **Fingolimod**, another disease modifying agent is not recommended in pregnancy due to teratogenic foetal malformation concerns. Contraception for at least 2 months is warranted post exposure after cessation of therapy.

Vaginal delivery is allowed except when serious disability may make vaginal delivery impractical in which case cesarean delivery should be performed. Epidural anesthesia can be given. Breastfeeding is avoided during interferon therapy. Intravenous immunoglobulins (IUCG) are given to prevent relapse in puerperium.

## Myasthenia gravis

Myasthenia gravis is an immune mediated neuromuscular disorder with easy fatiguability of facial, oropharyngeal, extraocular and limb muscles. Treatment is with longer acting acetyl choline-estrase inhibitors like neostigmine and pyridostigmine. Immunosuppresive therapy are the second line drugs. Plasmapharesis and intravenous immunoglobulin infusions are used for serious exacerbations.

- It has a genetic predisposition
- Patients should be made aware that the maximum risk is within the first year that follows diagnosis.

Thus, sustained improvement should be achieved before planning pregnancy.

Pregnancy usually does not affect the course of myasthenia gravis. However, 33% women may experience worsening of disease. Postpartum exacerbations may be anticipated in women with stable course during pregnancy. Women can deliver vaginally and epidural analgesia can be given. In the second stage, there may be fatigue of skeletal muscles necessitating instrumental delivery. IgG antibodies can cross the placenta and can cause neonatal effects like feeble cry, poor suckling and depression of respiration. Magnesium sulphate is avoided as its use can precipitate severe myasthemia crisis.

### Migraine

Migraine is periodic disorder characterized by episodic attacks of severe headache and autonomic nervous system dysfunction. It can be without aura or with aura (headache with visual scotomas or hallucinations). The traditional belief that migraine does not adversely affect pregnancy has been challenged by many authors who claim 16 fold higher risk of stroke, 5 fold risk for myocardial infection, 2 fold risk for venous thromboembolic disorders and hypertensive disorders. Women should be apprised that the risk for ischemic stroke, is compounded by smoking and oral pill. Foetal limb reduction deformities, maternal pre-eclampsia can occur. Treatment is with simple analgesics like aspirin, ibuprofen for mild migraine, intravenous hydration, parenteral antimetics and opioids for severe migraine. Ergotamine derivatives are avoided in pregnancy. Triptans like sumatriptan can be given, however a recent report on follow up for 36 months attributed emotional and activity related issues in offsprings exposed to therapy in utero. Peripheral nerve blocks can be considered in selective cases.

## PSYCHIATRIC PROBLEMS DURING PREGNANCY

Many psychiatric conditions like mood and anxiety disorders can occur in childbearing years and are more common in first and third trimesters. Anxiety and depression may occur in the first trimester due to unwanted pregnancy for which counselling plays a role. In the third trimester women may fear about the delivery or may doubt about normalcy of the fetus. Counselling with supportive psychotherapy, reassurance and behaviour modification is useful. The classification of psychiatric and mental disorders during pregnancy is the same as in non-pregnant state as written in the Diagnostic and Statistical Manual (DSM-V) which is regularly updated by the American Psychiatric Association. Psychiatric disorders like schizophrenia may co-exist with pregnancy and their presentation and management is the same as in non-pregnant state. There is 1.5 to 3 fold increased incidence of very low or low birth weight neonates or pre-term delivery with these disorders. Great care is needed in prescription of antipsychotic medication during pregnancy as they can cause foetal malformations, foetal growth restriction and prenatal problems. Benzodiazepines are avoided throughout pregnancy and puerperium as they can cause depressed respiration and withdrawl symptoms in the neonate. Olanzepine has been given safely though data on its use in pregnancy is limited.

### Postpartum Psychiatric Disorders

See Chapter 30 for details.

## Key Points

1. Any types of epilepsy grandmal, petitmal (absent), complex partial or myoclonic seizures may complicate pregnancy.
2. Pre-conceptional counselling is very important. The woman should be put on the lowest dose of anticonvulsant drugs, avoiding multiple drug therapy and substituting valproate with lesser teratogenic carbamazepine and giving 5 mg folate supplementation in pregnancy and lactation.
3. Prenatal screening by detailed anomaly scan and foetal echocardiography is recommended and Vitamin K is given to prevent hemorrhagic disease of newborn.
4. Foetal anticonvulsant syndrome can occur with anti-epileptic drugs during pregnancy which includes major and minor congenital malformations especially craniofacial defects.
5. Anti-epileptic drugs are continued in labour. Epidural analgesia is ideal. Vaginal delivery is the aim. Newborn is given intramuscular Vitamin K and breast milk is allowed. Contraception with high dose of oral pills, injectable progestogens DMPA or hormonal or traditional intrauterine devices are given.
6. Psychiatric disorders can occur during pregnancy. Their diagnosis, classification and management is the same as in non-pregnant state using the Diagnostic and Statistical Manual (DSM-IV) of American Psychiatric Association. Most drugs used to treat psychiatric disorders are safe in pregnancy.

## Suggested Reading

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# (ix) CONNECTIVE TISSUE (COLLAGEN VASCULAR) DISORDERS DURING PREGNANCY

The disorders are of two types:

1. Immune complex disease in which connective tissue damage occurs due to deposition of immune complexes. They are also called rheumatic diseases and include systemic lupus erythemotosis (SLE) and rheumatoid arthritis.
2. Inherited disorders of bone, cartilage, skin, vessels and basement membranes. They include diseases like Marfan syndrome, Ehlers Danlos Syndrome and osteogenesis imperfecta.

## SYSTEMIC LUPUS ERYTHEMATOSIS (SLE)

SLE is a relapsing and remitting multi-system connective tissue disorder of autoimmune etiology predominantly affecting women of reproductive age group. It usually affects joints, skin, kidneys and blood. It is associated with antinuclear antibodies, antidouble-stranded DNA antibodies, anti-Smith antibodies and other antibodies like anti-Ro and anti-La and antiphospholipid (aPL) antibodies. The diagnostic criteria are given in Table 35.40.

**Table 35.40: SLICC (2012) Classification Criteria for Systemic Lupus Erythematosus**

| |
|---|
| **Requirements:** ≥ 4 criteria (at least 1 clinical and 1 laboratory criteria) or biopsy-proven lupus nephritis with positive ANA or Anti-DNA |
| **I. Clinical Criteria** |
| 1. Acute Cutaneous Lupus* |
| 2. Chronic Cuteneous Lupus* |
| 3. Oral or nasal ulcers* |
| 4. Non-scarring alopecia |
| 5. Arthritis* |
| 6. serositis* |
| 7. Renal* |
| 8. Neurologic* |
| 9. Hemolytic anaemia |
| 10. Leukopenia* |
| 11. Thrombocytopenia (< 100,000/mm$^3$) |
| **II. Immunologic Criteria** |
| 1. Antinuclear antibodies (ANA) |
| 2. Anti - DNA |
| 3. Anti - Sm |
| 4. Antiphospholipid antibody |
| 5. Low complement (C3, C4, CH50) |
| 6. Direct Coomb's test (do not count in the presence of hemolyti anaemia) |
| **SLICC: Systemic Lupus International Collaboratng Clinics |

### Effect of Pregnancy on SLE

Disease improves in 33% women, remains uncharged in 33% and worsens in 33% pregnancies. There is 7% risk of severe morbidity and increased maternal mortality especially in women with active disease and lupus nephritis. There can be flare up of SLE, especially renal and hematological flare ups during pregnancy and puerperium (more common). The flare up of SLE can be life-threatening especially in puerperium. There is an increased risk for maternal thromboembolism.

### Effect of SLE on Pregnancy

Women with quiescent SLE without renal involvement and Ro/La and aPL antibodies negative have usually normal outcome. However, there is adverse outcome in the form of increased risk of abortions, pre-eclampsia, foetal growth restriction, preterm labour and foetal death in presence of active disease, antiphospholipid syndrome or lupus nephropathy.

### Neonatal Lupus Syndrome

Anti Ro or anti La antibodies can cross the placenta causing neonatal lupus in 5% babies of Ro-positive mothers manifesting as hemolytic anaemia, leucopenia, thrombocytopenia and congenital heart block. **Presence of congenital heart block in a newborn in absence of maternal symptoms should arouse the suspicion of SLE in the mother.** Prevalence is 1–2% in babies of Ro-positive women increasing to 16% and 50% in second and third pregnancies respectively in such women. Foetal echocardiography should be done, Such babies require pacemakers in their early infancy.

Currently, mothers positive for anti Ro and for anti La antibodies are monitored weekly with PR intervals for screening for 1° and 2° heart blocks in the fetus, between 16–28 weeks. Timely surveillance has been shown to reduce the incidence of 3° congenital heart blocks and need for pacemaker in this populations.

### Diagnosis

The diagnosis of SLE is made using the Diagnostic Criteria for SLE (Table 35.40). Maternal blood should be checked for lupus anticoagulant and antiphospholipid antibodies, anti SSA (Ro) and anti SSB (La) antibodies.

### Management

Pre-pregnancy counselling is important with advice to conceive during remission phase with no recent flare in the past 6 months. Non-steroidal anti-inflammatory drugs are discontinued but hydroxychloroquine is continued to avoid flare up of SLE. Low dose aspirin can be used throughout pregnancy. Steroids are the mainstay of treatment. Prednisolone (1–2 mg/kg body weight) and azathioprine are given. They should be seen regularly in clinic for any flare up of the disease. Mycophenolate mofetil, methotrexate and cyclophosphomide are teratogenic and are avoided. In case of flare IV methylprednisolone is given in the dose of 1-1.5 mg/kg for 10 days in case there is no

renal involvement, however in case of renal involvement, higher dose of 10-30 mg/kg is required for 5 days and then eventually tapered. Patient is usually delivered at term except if there is severe pre-eclampsia or progressive renal disease. The women being at high risk of pre-eclampsia and foetal growth restriction (FGR) should be offered serial growth scans and tests for foetal well-being. These women should be seen jointly by obstetrician and rheumatologist or physician. The woman can deliver vaginally but may require cesarean delivery with congenital heart block.

The women should receive steroids in puerperium. Oral pills may affect SLE and intrauterine device may cause infection. Barrier methods are the safest.

## Thrombophilia in pregnancy

Virchow's triad for thrombosis

1. Venous stasis
2. Hypercoagulable state
3. Vascular injury

**Pregnancy is hypercoagulable state due to following reasons**

I. **Increased clotting potential**
   1. Increased clotting factors VII, VIII, IX, X, XII (20–100%)
   2. Increase fibrinogen levels (200–300%)
   3. vonWillebrand factor (vWB) vWf (400%)
   4. Increased clotting factors XI and XIII

II. **Decreased anticoagulant property–Protein**
   1. Decreased free Protein S
   2. Increase in resistance to activated Protein C

III. Decrease fibrinolysis. Increase in plasminogen activator inhibitor.

## CLASSIFICATION OF THROMBOPHILIAS

**Acquired thrombophilias**

1. Antiphospholipid antibody (APLS) syndrome
2. Hyperhomocystinemia
3. Activated protein C resistance

**Inherited**

1. Factor V Leiden mutation
2. Prothrombin gene mutation
3. Antithrombim III deficiency
4. Protein C deficiency
5. Protein S deficiency
6. Methyltetrahydrofolate reductase enzyme deficiency

## ANTIPHOSPHOLIPID ANTIBODY SYNDROME (APS, APLS, APLA)

Antiphospholipid antibody syndrome is an association between clinical features such as thrombosis, poor pregnancy outcome like three or more spontaneous abortions, one or more foetal deaths and one or more preterm births due to severe pre-eclampsia with presence of anticardiolipin antibodies, lupus anticoagulants and anti β-2 glycoprotein on two or more occasions 12 weeks apart. Antiphospholipids (aPLS) reduce hCG release and inhibit trophoblast invasion which may explain miscarriages in them. Typically foetal loss occurs in the second trimester and is associated with severe growth restriction, oligohydramnios and early onset pre-eclampsia possibly due to abnormal placentation related to thrombosis. The International Consensus Statement and ACOG have classification criteria for diagnosis of ALPS which were revised in 2006 in Sapparo city hence called Revised Sapporo criteria, Table 35.41 shows criteria for diagnosis of APLS. At least one clinical and one laboratory test criteria should be present for the diagnosis.

**Table 35.41: Criteria for Diagnosis of Antiphospholipid Antibody Syndrome**

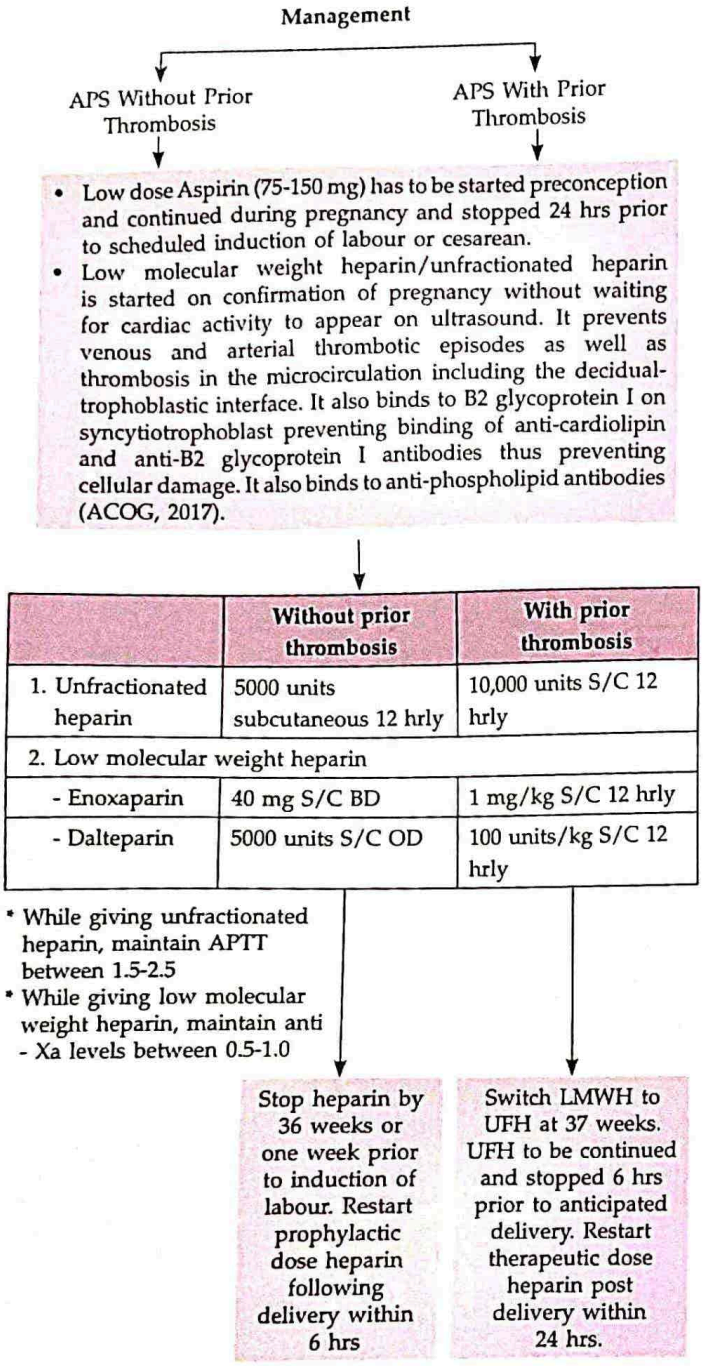
| | |
|---|---|
| **Clinical criteria** | |
| 1. | One or more episode of unexplained venous, arterial or small vessel thrombosis in any organ or tissue |
| 2. | (i) One or more unexplained death of a morphologically normal fetus at or after 10 weeks<br>(ii) three or more consecutive unexplained spontaneous (abortions) before 10 weeks<br>(iii) preterm delivery for severe pre-eclampsia or placental insufficiency before 34 completed weeks. |
| **Laboratory criteria** | |
| 1. | Anticardiolipin antibodies: IgG or IgM in medium or high serum levels identified twice at least 12 weeks apart. |
| 2. | Lupus anticoagulant antibody: Identified twice at least 12 weeks apart. |
| 3. | Anti β-2 glycoprotein IgG or IgM antibody: Identified twice at least 12 weeks apart |

Women with APS are more prone to thrombosis as it is a type of acquired thrombophilia. The women with past history of thrombosis due to APS run the risk of recurrent venous thrombosis. APS increases the risk of early and later abortions, foetal growth restriction, pre-eclampsia and intrauterine death.

A few patients may develop catastrophic antiphospholipid antibody syndrome (CAPS) also called Asherson syndrome characterized by rapid and progressive thromboembolism involving three or more organ systems simultaneously with high maternal mortality.

### Management

If patient is on warfarin preconceptionally, it should be changed to heparin prior to 6 weeks of pregnancy.




Heparin therapy can cause bleeding, thrombocytopenia, osteopenia and osteoporosis. Steroids (prendisolone) is not used in primary APLA but can be used for secondary APS with lupus. Immunoglobulin therapy in dose of 0.4 g/kg daily for 5 days (total 2 g/kg) followed by 1 g/kg monthly is only used for heparin induces thrombocytopenia or with overt disease but is very expensive.

It is mainly used for catastrophic APA and heparin induced thrombocytopenia, Hydroxychloroquine can also be used with low dose aspirin. These pregnancies are monitored by regular non-stress test or Biophysical profile and serial ultrasound for foetal growth. Foetal loss rate is high without treatment. Even with treatment still birth and pre-eclampsia rate are very high especially in triple antibody positive as compared to single anti BOD positive (Pregnants study 2017).

# OTHER CONDITIONS

## Rheumatoid arthritis

Rheumatoid arthritis is a chronic multi-system disease characterized by pain, swelling and tenderness of joints. Rheumatoid arthritis is usually associated with rheumatoid factor and antinuclear antibodies. American College of Rheumatology and European Leage against Rheumatism has given criteria for diagnosis of rheumatoid arthritis as shown in Table 35.35. A score of ≥ 6 is needed for diagnosis. There are no obvious adverse effects of rheumatoid arthritis on pregnancy outcome. The disease improves in majority (90%) women during pregnancy.

### Management

Non-steroidal anti-inflammatory drugs (NSAIDS) are given though can cause abortion, ductus arterosus construction and neonatal pulmonary hypertension. Paracetamol is safe in pregnancy and may be given in maximal doses, if required. Corticosteroids (Prednisolone) can be safely used in pregnancy as indicated. Azathioprine, hydroxychloro quine and sulfasalazine can also be used in pregnancy. Biological disease modifying antirheumatic drug (DMARDs) which are TNF-α inhibitors like infliximab or etanercept can be given but foetal safety is a concern. Oral pills can be used in them.

Most women can deliver vaginally with cesarean delivery being rarely indicated for limitation of hip abduction.

## Scleroderma

Scleroderma may occur as a cutaneous form or as systemic sclerosis or as a part of CREST (Calcinosis, Raynand's phenomenon, esophageal involvement, sclerodactyly and telangiectasia) syndrome. There is no definite treatment for it. With severe disease, there can be adverse effect on pregnancy outcome. Women with renal involvement and hypertension are at an increased risk of hypertension and foetal growth restriction. Women with severe pulmonary fibrosis or pulmonary hypertension should be advised against pregnancy **(level IV evidence)**.

Vaginal delivery is tried. However, if scleroderma produces dystocia due to soft tissue thickening then cesarean delivery is planned. Epidural or spinal anesthesia is performed as tracheal intubation may be difficult due to limited mouth opening and more risk of aspiration due to esophageal dysfunction. Delivery room should be warm, use of warm intravenous fluids, extra blankets is recommended to prevent Reynaud's phenomena. Pregnancy complications can be renal crisis, preterm birth, foetal growth restriction, increased perinatal mortality possibly due to placental anomalies (decidual vasculopathy, acute atherosis and infarcts). Oral pills are avoided in them.

## Key Points

1. SLE is more likely to flare up in pregnancy. Adverse effect of SLE are related to the presence of antiphospholipid syndrome (APS), renal involvement, disease activity and presence of anti-Ro and La antibodies.
2. APS may cause thrombosis, miscarriage, late foetal loss, early onset pre-eclampsia and foetal growth restriction. It is treated in pregnancy with low dose aspirin with heparin or low molecular weight heparin (LMWH). Evidence supports the use of heparin throughout pregnancy in women with APS and past history of thrombosis.
3. Rheumatoid arthritis generally improves in pregnancy but may worsen in puerperium. Steroids are safe in pregnancy to treat connective tissue orders and are preferred over NSAIDS.

## Suggested Reading

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## Examination Questions

A. Define antiphospholipid syndrome (APS). Enumerate its effects on pregnancy. How will you manage a case of APS during pregnancy? (Important)

B. Write short notes on:

1. Systemic lupus erythematosis during pregnancy
2. Antiphospholipid syndrome during pregnancy (Many universities)
3. Rheumatoid arthritis during pregnancy

# (x) DERMATOLOGICAL DISORDERS DURING PREGNANCY

## PHYSIOLOGICAL SKIN CHANGES IN PREGNANCY

Physiological skin changes are common in pregnancy and are not of much importance. Hyperpigmentation is more common in pregnancy and is more marked on the face **(chloasma or melasma)**, along the linea nigra, on the abdomen (striae gravidarum) and areola of breasts. Excess of estrogen levels is the cause (see Chapter 6 for details).

## PATHOLOGICAL CONDITIONS

### Pruritis

Pruritis is common (1–2%) in pregnancy and is usually due to cholestasis. Unfortunately, there is no specific treatment and it worsens with advancing pregnancy. Infected skin conditions like scabies or ringworm must be excluded and opinion of a dermatologist should be sought.

**Treatment is conservative.** Ursodeoxycholic acid, local calamine lotion to the skin and antihistaminics like fexofenadine are given (see intrahepatic cholestasis of pregnancy in liver disorders above).

### Pruritic urticarial papules or poly-morphic eruption of pregnancy (PEP)

PEP is common (0.25–1%). They appear in primigravidas causing severe pruritis and erythematous papular lesions. Topical steroid preparations of betamethasone are useful and oral prednisolone is very rarely required. There is no increase in perinatal morbidity and mortality.

### Papular dermatitis (Syn. Prurigo of pregnancy, prurigo gestation)

Papular dermatitis is uncommon (1 : 300–2400) and is characterized by generalized pruritic eruption. The lesions are soft, red to brown papules which clear after pregnancy but can recur in subsequent pregnancies. Steroids (prednisolone 10–40 mg a day) orally are given. Perinatal mortality may be increased especially, if steroids are not given.

### Herpes gestationalis (Pemphigoid gestationalis)

Herpes gestationalis is rare (1 : 10,000) and peculiar pregnancy dermatosis disorder characterized by severely pruritic widespread eruption with lesions varying from erythematous edematous papules to bullae. Skin of abdomen and extremities is usually involved while the mucus membranes are spared. It is an immunological condition due to deposition of IgG and complement along the basement membrane zone between the epidermis and the dermis. *It may rarely be associated with prematurity, stillbirths and neonatal skin lesions.*

Primigravidae are affected preferentially. Disease tends to occur at earlier periods of gestation and is more severe when it affects subsequent pregnancies. The disease usually manifests in second and third trimesters but postpartum exacerbation are frequent. Early onset disease is more likely to be associated with impaired foetal growth and preterm birth attributable, in part, to placental insufficiency due to deposition of complement along with the amniotic basement membrane. Diagnosis is clinical. However, skin biopsy shows C3 complement and IgG deposition along basement membrane between the epidermis and the dermis.

Treatment is by topical steroid cream for mild cases and prednisolone orally 40 or 60 mg daily for severe cases along with antihistaminics for relief of pruritus. Plasmapharesis, high dose intravenous immunoglobulin (IVIG) therapy or cyclosporin may be required for refractory cases in postpartum period.

### Acne

Acne usually improves in pregnancy. The retinoic acid derivative isotretinoin is absolutely contraindicated as it is one of the most teratogenic drug known to cause 26 fold rise in central nervous system, cardiac and craniofacial abnormalities in the fetus. For severe acne during pregnancy topical benzoyl peroxide, clindamycin or erythromycin gel can be applied. Systemic antibiotic, e.g. erythromycin may be given for not more than 4–6 weeks, being initiated only in second trimester of pregnancy or later.

### Impetigo herpetiformis of pregnancy (Pruritis folliculitis of pregnancy, generalized pustular psoriasis, GPP)

Impetigo herpetiformis is a rare condition which develops in the third trimester. There are multiple sterile pustules around margins of erythematous lesions starting at groin and other flexures. Patients may present with fever, malaise and vomiting. Hypocalcemia may develop. There is increased risk of stillbirths and neonatal deaths. Treatment is by bed rest, admission in hospital, application of cold creams to skin lesions, topical and systemic high potency steroids along with antibiotics for secondary infection. Ultraviolet B-phototherapy is second line therapy. Cyclosporin and TNF antagonists (Infliximab) may be used as third like therapy for resistant case. Medical termination of pregnancy is rarely indicated in severe intractable cases.

### Leprosy (Hansen's disease) in pregnancy

Leprosy is caused by acid fast Mycobacterium leprae and may become overt for the first time during pregnancy or within few months following delivery. With established leprosy there is chance of exacerbation of the lesions during pregnancy. Failure of signs of the disease to reappear during or after pregnancy indicates that the leprosy is definitely arrested. Foetal growth restriction (FGR) is a common problem. Although Mycobacterium leprae have been found in the placenta and cord, congenital infection of the baby is unlikely. However, the baby should be

separated from the infected mother immediately after delivery. When the disease becomes quiescent and non-infectious, the baby may be given to the mother.

**Treatment**

Multi drugs dapsone 100 mg daily, rifampicin 450 mg daily, clofazimine 50 mg daily are used without problem and the treatment is continued for 2 years. Thalidomide is absolutely contraindicated.

Vaginal delivery is allowed.

Breastfeeding is given by mother on anti-leprosy medicines.

## Key Points

1. Due to excess of estrogen and other steroid levels during pregnancy, hyperpigmentation, chloasma (mask) and striae gravidarum are common in pregnancy needing only reassurance or topical creams.
2. There are some pregnancy-specific dermatosis during pregnancy.
   (i) Polymorphic eruption of pregnancy with red urticarial papules and plaques manifest in the late third trimester and is treated by local calamine lotion or topical or systemic steroids and anti-histaminics.
   (ii) Pemphigoid gestationalis (herpes gestationalis) occurs in second or third trimester with erythematous urticarial plaques, vesicles and bullae on skin of abdomen and extremities. It can cause foetal growth restriction and preterm labour. Treatment is by topical and systemic steroids and antihistaminics.
   (iii) Prurigo of pregnancy along with acne may need local steroid cream and antibiotic (clindamycin) cream.
   (iv) Pruritis folliculitis of pregnancy (impetigo herpetiformis) is characterized by masses of itchy red follicular papules in the third trimester of pregnancy and is treated by topical 10% benzoylperoxide, topical steroids and antihistaminics.
3. Leprosy caused by Mycobacterium leprae should be treated by anti-leprosy drugs.

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## Examination Questions

A. Enumerate various physiological cutaneous changes occuring during pregnancy. Enumerate various pregnancy dermatoses. How will you manage a case of herpes gestationalis in the third trimester of pregnancy?

B. Write short notes on:
   1. Chloasma
   2. Striae gravidarum
   3. Pregnancy prurigo
   4. Herpes gestationalis
   5. Impetigo herpetiformis (pruritis folliculitis of pregnancy)
   6. Polymorphic eruptions of pregnancy
   7. Pathological dermatoses of pregnancy
   8. Leprosy during pregnancy

CHAPTER 36

# INFECTIONS AND INFESTATIONS DURING PREGNANCY

## (i) TUBERCULOSIS AND PREGNANCY

### INTRODUCTION

Tuberculosis (TB) is a major public health problem in the world especially in developing countries due to the effect of HIV pandemic. It has been further compounded by the emergence of multidrug resistant tuberculosis (MDR-TB) and extensively drug resistant tuberculosis (XDR-TB) in the recent past. It may be encountered in 1–2% pregnant women in India. Pulmonary TB (PTB) is the most common type of TB during pregnancy but extra pulmonary TB (EPTB) like TB of lymph nodes, bones, kidneys, abdomen and occasionally meningeal TB or miliary TB can occur in pregnancy.

### RISK FACTORS

The various high risk factors for TB are as follows:

1. Past history of TB
2. Family history of TB
3. Close contacts of active cases
4. Poor socio-economic status
5. High prevalence of TB in the community
6. HIV positive patient
7. Intravenous drug addiction
8. Alcohol and other drug addiction

### EFFECTS OF TB ON PREGNANCY

*With timely diagnosis and starting appropriate and effective anti-tubercular treatment, there is no evidence of adverse effects of TB on pregnancy.* However, in women with advanced disease, late diagnosis and inadequate treatment, there is higher perinatal mortality and incidence of prematurity (twofold), small for gestational age babies and low birth weight (< 2500 g) babies. There is also increase risk of antepartum hemorrage and pre-eclampsia.

### EFFECTS OF PREGNANCY ON TB

*Current thinking is that the course of TB is usually unaffected by pregnancy.* Women with TB can have pregnancy unless the disease is very advanced or is multidrug resistant where pregnancy is contraindicated.

### CLINICAL PRESENTATION OF TB DURING PREGNANCY

The presentation of TB during pregnancy is similar to that in non-pregnant state but diagnosis may be delayed by the non-specific early symptoms of TB and the common occurrence of fatigue and malaise in pregnancy.

A patient of pulmonary TB usually presents with cough, expectoration, fever, malaise, fatigue, weight loss, anorexia, lassitude, lethargy and listlessness. She can also present with hemoptysis and chest pain.

A patient of extrapulmonary TB presents with symptoms and signs related to the affected organ apart from fever and anorexia and weight loss, like abdominal pain and distension (in abdominal TB), lymphadenopathy (in TB lymphadenitis), swollen and painful joints (in TB arthritis), headache and neurological symptoms (in TB meningitis).

### DIAGNOSIS

1. **Mantoux test** (Tuberculin skin test) using 1 or 2 tuberculin unit purified protein derivative RT23 (1 or 2 T.U. PPD RT-23) with > 10 mm is considered positive.
2. **Chest radiography** should be performed using abdominal shield after the first trimester in presence of positive tuberculin test and in clinically suspicious cases.
3. **Sputum examination.** There has been change in policy. Instead of three, only two sputum smears should be

examined for acid fast bacilli (AFB). As per National Tuberculosis Elimination Programme (NTEP) of India, diagnosis of PTB is made if at least one of the two smears is positive for AFB.

4. Gastric lavage can also be used.
5. Diagnostic bronchoscopy in selected cases
6. For extrapulmonary TB, investigations corresponding to the affected site should be carried out.
   (a) Fine needle aspiration cytology (FNAC) or biopsy samples from lymph nodes.
   (b) Fluid by tapping from pleural, ascitic or pericardial effusions
   (c) Lumbar puncture for TB meningitis (contraindicated with raised intracranial pressure)
7. Molecular diagnostic tests like gene Xpert and line probe assay on pulmonary and extrapulmonary sample for rapid diagnosis of both drug sensitive and drug resistant tuerculosis.

## TREATMENT OF TB IN PREGNANCY

For latent infection (positive tuberculin test in absence of active disease), isoniazid prophylaxis in dose of 300 mg daily for six months or isoniazid (300 mg) and rifampicin (450 mg or 600 mg, if weight > 50 kg) for 3 months. Pyridoxine (Vitamin $B_6$) 40–100 mg daily is given with anti-tuberculous therapy to prevent peripheral neuropathy. For active disease, category wise treatment should be given preferably under directly observed treatment shortcourse (DOTS) strategy as intermittent therapy or daily therapy as per Table 36.1.

As per WHO 2018 recommendations and adopted by NTEP, category II and III are no longer practised. Therefore, all new patients (pulmonary and extrapulmonary both smear positive and negative) are now given category I treatment. Even patients with drug sensitive relapse cases, treatment failures and treatment after interruption are also given same treatment after ruling out drug resistance. All first line 4 drugs—isoniazid, rifampicin, pyrazinamide and ethambutol are non-teratogenic and safe in pregnancy and should be given even in the first trimester. Streptomycin is avoided in pregnancy as it can cause foetal ototoxicity. Proper diet, rest and appropriate antituberculous therapy (ATT) with pyridoxine is recommended during antenatal period.

The side-effects of the drugs are uncommon and are same as in non-pregnant state like hepatitis, peripheral neuropathy, hepatic enzyme elevation and hypersensitivity (isoniazid), nausea, vomiting, hepatitis, orange discoloration of secretions (rifampicin), optic neuritis, skin rash, reduced visual acuity (ethambutol), hepatotoxicity, hyperuricemia, gastric upset, skin rash and arthralgia (pyrazinamide).

### Treatment of multi drug resistant (MDR) TB in pregnancy

Most second line drugs can cause damage to the genes (ototoxicity with kanamycin and amikacin, teratogenicity with ethionamide and prothionamide). Therapeutic abortion can be considered in presence of MDR-TB in a woman after proper counselling about the potential teratogenic effects of drugs. Those still keen to continue pregnancy must be treated with complete dosage of second line drugs in consultation with an expert after proper counselling.

### Treatment of HIV and TB in pregnancy

The management of pregnant TB women in HIV positive cases is the same as in HIV negative women but may need slight adjustments to avoid drug interaction.

## MANAGEMENT OF LABOUR

After successful antituberculous therapy, obstetric management is similar to other pregnant women. During labour, pulse and respiration should be watched carefully. Ventouse or outlet forceps delivery may be required to cut short the second stage of labour if the patient is not able to bear down well. Cesarean section is only performed for any obstetric indication and not for TB.

## MANAGEMENT OF NEONATE OF A TB PATIENT

In the case of latent TB in the mother (positive tuberculin test) without active TB or sputum negative active PTB, the risk to the infant is negligible. However, if she is sputum positive, then the infant needs evaluation for active TB with chest X-ray, gastric aspirate for AFB to rule out congenital TB. If no active TB is found, the infant should receive isoniazid prophylaxis for 3 months or until after the mother's sputum becomes negative. BCG vaccine is either postponed or given with isoniazid resistant BCG vaccine.

**Table 36.1: WHO Recommended Treatment Regimens for Tuberculosis**

| Tuberculosis treatment | Tuberculosis patients | Initial phase | Continuation phase |
|---|---|---|---|
| New treatment regimen (Formerly category I) including Retreatment regimen (Formerly category II) | 1. New smear positive pulmonary TB<br>2. New smear negative, PTB with extensive parenchymal involvement<br>3. New cases of severe forms of extra-pulmonary tuberculosis<br>4. Drug sensitive: Sputum smear positive relapse<br>5. Drug sensitive: Treatment failure<br>6. Drug sensitive: Treatment after interruption | 2 HRZE<br>2 SHRZE/1HRZE | 4 HRE<br>or<br>6 HE<br>5 HRE |

H: Isoniazid R: Rifampicin Z: Pyrazinamide E: Ethambutol
S: Streptomycin 2 HRZE: 2 months daily therapy 4 HRE: 4 months daily therapy

With a tuberculin positive infant, isoniazid prophylaxis should be given for 6 months after ruling out active TB. In the presence of MDR-TB in mother, INH prophylaxis has no role and should not be given. In such cases, the infant should receive 2 sensitive drugs of index case and BCG vaccination as it has been found to be protective. However, BCG is contraindicated in HIV positive children.

### TB and breastfeeding

Regardless of the mother's TB status, breastfeeding should be given. If she is sputum positive, she should use a mask over her face while breastfeeding the infant. The newborn should not be separated from mother. The only situation where separation is required is mother with MDR-TB.

### TB and contraception

As rifampicin stimulates cytochrome $P_{450}$ enzymes, the metabolism of oral contraceptives is stimulated and their efficacy is reduced. Alternative contraceptive should be used in such cases or following consultation with a clinician, an oral contraceptive pill containing a higher dose of estrogen (35–50 mcg) may be given. If family is complete, tubal ligation is preferred after proper counselling.

### Perinatal (Congenital) TB

Perinatal TB is the infection of the fetus in utero through the hematogenous route via the umbilical vein or aspiration or ingestion of contaminated amniotic fluid through the placenta. Diagnosis of perinatal TB is made by:

1. Tuberculous lesions observed in the first seven days of life
2. A primary complex or caseating granuloma
3. Documented tubercular infection of the placenta or endometrium.
4. Exclusion of tuberculosis infection of an attendant in postnatal period.

For asymptomatic (latent) TB infection, infant is asymptomatic but Mantoux positive, isonizid chemoprophylaxis is given for 6 months in dose of 10 mg/kg body weight. BCG vaccination is given at birth and breastfeeding is continued.

Symptoms of perinatal TB appear in the second or third week of life. Examination and culture of placenta for AFB is useful. Tuberculin skin test is usually negative initially, but may become positive in 4–8 weeks. As it is often difficult to obtain sputum from infants. M. tuberculosis is more easily cultured from gastric aspirate. It is a rare, but serious condition with high (up to 50%) mortality which is usually due to delay in diagnosis and treatment. Treatment is with isoniazid (10 mg/kg), rifampicin (15 mg/kg), pyrazinamide (30 mg/kg) and ethambutol (20 mg/kg) for 2 months followed by isoniazid, rifampicin and ethambutol for 4–10 months.

It should be suspected if aggressive broad-spectrum antibiotics are ineffective and tests for other congenital infections are negative.

## Key Points

1. Using high index of suspicion, early diagnosis and timely and adequate treatment of TB during pregnancy can improve the maternal and perinatal outcomes almost equal to normal pregnant women.
2. First line ATT drugs are safe in pregnancy and should be given even in the first trimester for optimum maternal and perinatal outcome.
3. Women with MDR-TB and HIV with TB in pregnancy should be managed carefully in consultation with experts in the field.

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## Examination Questions

A. What are the effects of tuberculosis on pregnancy? Describe the clinical presentation and management of pulmonary tuberculosis during pregnancy.
B. Enumerate the clinical features, diagnosis and management of extrapulmonary tuberculosis during pregnancy.
C. Write short notes on:
   1. RNTCP treatment of pulmonary tuberculosis during pregnancy
   2. Congenital tuberculosis
   3. Management of newborn of a mother with pulmonary tuberculosis

# (ii) MALARIA IN PREGNANCY

## INTRODUCTION

Malaria is a protozoan infection caused by one of the four species of plasmodium which are *P. vivax*, *P. ovale*, *P. malariae* and *P. falciparum*. Out of 300–500 million persons affected worldwide, about two million people are affected annually in India. In endemic areas, it may complicate pregnancy. Pregnant women are more susceptible to malaria infection.

## CLINICAL FEATURES

Clinical features include fever with rigor and chills, headache, myalgia, malaise, anaemia and jaundice. There can be hepatomegaly, splenomegaly, intravascular hemolysis and thrombocytopenia. Falciparum malaria can be dangerous and life threatening causing black water fever, renal failure, coma and death.

## EFFECTS OF PREGNANCY ON MALARIAL COURSE

There is decreased immunity during pregnancy due to changes in cell mediated immunity and antibody production. There is also diversion of proteins from the immune system for the growth of the fetus declining immunity and increasing parasitemia which increases with advancing pregnancy. The attacks of fever are more frequent in the third trimester and puerperium than in first trimester. Placental and foetal infections can occur.

## EFFECTS OF MALARIA ON PREGNANCY

Malaria affects the health of mother and fetus. Primigravidas are more susceptible than multigravidas and non-pregnant females. It could be due to her recent shifting from a non-endemic area to an endemic area after marriage and she may not have developed immunity towards the malarial parasite. The infected red cells become rigid, sticky and irregular causing blockage of microcirculation due to sequestration. The infected red cells undergo hemolysis. The various maternal and foetal effects are shown in Table 36.2.

Most complications occur due to packing of intervillous spaces by parasites and macrophages especially with *P. falciparum* infection. It interferes with circulation of maternal blood through intervillous spaces impairing foetal growth. The placental infection can be prevented by chemotherapy.

**Table 36.2: Maternal and Foetal Effects of Malaria on Pregnancy**

| Maternal Effects |
|---|
| 1. Pyrexia<br>2. Perspiration<br>3. Pallor<br>(i) Hemolytic anaemia due to hemolysis of infected red cells.<br>(ii) Megaloblastic anaemia due to folic acid deficiency and hemolysis.<br>4. Metabolic acidosis<br>5. Hypoglycemia is seven times more likely due to excessive glucose consumption by the parasites, decreased gluco-neogenesis and increased insulin secretion by quinine and other drugs.<br>6. Jaundice due to hemolysis and liver dysfunction.<br>7. Renal failure and black water fever due to renal microcirculation blockage (P falciparum infection)<br>8. Splenomegaly<br>9. Respiratory distress<br>10. Disseminated intravascular coagulation<br>11. Convulsions (P. falciparum, cerebral malaria)<br>12. Unconsciousness (coma) (P. falciparum, cerebral malaria)<br>13. Death, if left untreated |
| **Foetal Effects** |
| 1. Increased risk of miscarriage<br>2. Preterm delivery<br>3. Foetal growth restriction and low birth weight<br>4. Stillbirths and perinatal death<br>5. Foetal anaemia<br>6. Congenital malaria<br>7. Susceptibility of the infant to malaria |

## DIAGNOSIS

1. The parasite can be seen on a thick peripheral blood smear made during fever.
2. A thin film should also be made to detect the species (Figs. 36.1 A and B).
3. Placental blood and cord blood smear can also be made during delivery.

## TREATMENT

Treatment must be commenced immediately after diagnosis. Commonly used anti-malarial drugs can be safely given in pregnancy. Chloroquine is the treatment of choice for malaria caused by plasmodia sensitive to the drug. Four tablets of chloroquine phosphate 150 mg each are given initially followed by two tablets given after 6 hours and then 2 tablets once a day for 2 days (total 10 tablets). It is safe for the fetus. Pyrimethamine is required to eradicate the exoerythrocytic phase of *P. Vivax* and *P. Ovale* but can cause hemolytic anaemia in the fetus and is preferably delayed until after delivery. Mefloquine orally can also be given for chloroquine resistant vivax infection. For P. falciparum malaria, current treatment of choice is artemisinin based therapy (WHO, 2016, CDC 2013). PREGACT Study Group (2016) observed artemisinin based therapy to be safe in pregnancy for P.falciparum. Dose of

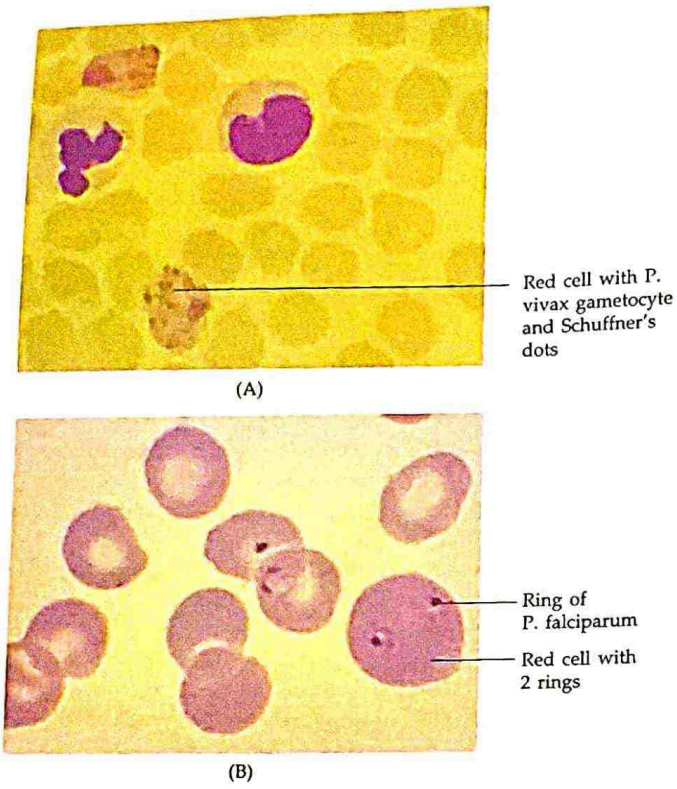



**Fig. 36.1:** (A) Peripheral blood film showing gametocytes of Plasmodium vivax with eccentric red nucleus and Schuffner's dots; (B) Peripheral blood film showing ring stage of Plasmodium falciparum with one red cell showing 2 rings.

artesunate is 4 mg/kg/day (about 200 mg daily × 3 days) with mefloquine 15 mg/kg on day 2 and 10 mg/kg on day 3. For severe or complicated malaria, quinine is given either parenterally or orally (10 mg/kg orally every 8 hourly for 7 days). It can cause hypoglycemia as side effect. Other drugs rarely required are quinidine and tetracycline. Tetracycline is active against chloroquine resistant malaria but can cause maternal hepatotoxicity and foetal dysplasia and foetal dental discoloration. Clindamycin is useful in multidrug resistant malaria.

### Current Treatment of Falciparum Malaria in Pregnancy

**First Trimester:** Quinine therapy

**Second and third trimester:**

*North Eastern States:*

Artemether 20 mg
Lumefantrine 120 mg
Orally for 3 days.

**Rest of India:**

Artesunate: 10 mg/kg
Sulfadoxine Pyrimethamine 500/25 mg
Both orally for 3 days
Primaquine is contraindicated in pregnancy

## CHEMOPROPHYLAXIS

In endemic areas, two tablets of chloroquine (300 mg) are given once every week until six weeks after delivery. Mefloquine can be used in areas with chloroquine resistant strains. Use of protective clothing, mosquito repellants and use of mosquito net is recommended to avoid mosquito bites. Rapid control of fever and correction of iron and folic acid deficiency should also be initiated at the earliest. Folic acid should be given 10 mg per day.

Regular and more frequent antenatal check-up is required and foetal monitoring should be regularly done using ultrasound scan and non-stress test.

A high index of suspicion is required to treat the disease early and thus saving mother and her newborn. The National Malaria Eradication Programme (NMEP) of Government of India is trying to eradicate this disease from India.

Several vaccines are under evaluation (trial), but are not yet available for routine use.

## CONGENITAL MALARIA

Congenital malaria can occur in up to 0.1 to 7% women with malaria especially in fetuses of unprotected susceptible women. Normally maternal antibodies formed by mother cross the placental barrier and protect the fetus. When the passive immunity is poor, congenital malaria can occur in fetus with parasite probably travelling through damaged placenta. Foetal death in malaria is not usually due to congenital malaria but due to maternal hyperpyrexia. However, foetal and neonatal death can occur due to congenital malaria, anaemia, prematurity and foetal growth restriction. Treatment of congenital malaria is by chloroquine or other antimalarials to which the parasite is sensitive.

## Key Points

1. Malaria can complicate pregnancy in endemic areas and pregnancy can enhance the severity of malaria.
2. A high index of suspicion and timely diagnosis and treatment with chloroquine or other drugs (in chloroquine resistant strains) can significantly improve the maternal and perinatal outcome.
3. P. falciparum causing black water fever and cerebral malaria can be life threatening and must be energetically treated at the earliest.

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## Examination Questions

A. What are the effects of malaria on pregnancy and vice versa? How will you treat malaria during third trimester of pregnancy?
B. Enumerate the clinical features of malaria during pregnancy. How will you manage falciparum malaria in labour?
C. Write short notes on:
   1. Plasmodium falciparum malaria during pregnancy.
   2. Chemoprophylaxis of malaria during pregnancy

# (iii) HUMAN IMMUNODEFICIENCY VIRUS INFECTION (HIV)

## INTRODUCTION

HIV, a RNA retrovirus, causes a serious infection which in about 8–10 years leads to acquired immunodeficiency syndrome (AIDS) in which the immune system is so depleted that unusual bacterial and viral infections develop.

## EPIDEMIOLOGY

HIV is increasing in all countries and is a global problem. Globally there were 35.3 million (0.8%) people infected with HIV/AIDS in 2012 (WHO, 2012) with 50.14% women. The prevalence rate of HIV infection among pregnant women is about 0.3% and the vertical transmission rate to the fetus is about 30%. Globally, about 40% of the infected patients are women with majority (80%) being in the reproductive age group (15–44 years). India has the highest population of HIV infected persons next only to Africa. Overall prevalence is 0.4% in India but is up to 1% in high risk areas like Andhra Pradesh, Maharashtra, Karnataka and Tamil Nadu. These four states are responsible for 62% of all HIV infections in India. With National AIDS Control Program India has witnessed 57% decline in incidence of HIV infection from 2.74 lakh cases in 2000 to 1.16 lakh cases in 2011 (0.27%) alongwith 29% reduction in AIDS related deaths with scale up of anti retroviral treatment (ART).

## RISK FACTORS AND MODES OF TRANSMISSION

The various risk factors for HIV, modes of transmission and risk factors for vertical transmission are shown in Table 36.3, Table 36.4 and Table 36.5 respectively.

**Table 36.3: Risk Factors for HIV**

1. Higher (2–17%) male-to-female transmission than vice versa.
2. Larger surface area of contact in women.
3. Less recognition of reproductive tract infections (RTI) or sexually transmitted infections (STI) in women making them more prone to HIV/AIDS.
4. Illicit drug use including cigarette smoking
5. Intravenous drug users sharing needles, waxing, ear piercing
6. Higher need of blood transfusion in women
7. Promiscuous behaviour of either partner
8. Occupation involving frequent travel, e.g. truck drivers
9. Adolescents indulging in unsafe sex practices.

**Table 36.4: Modes of Transmission of HIV**

| | |
|---|---|
| **Sexual** | 1. Homosexual<br>2. Heterosexual (most common); Male-to-female transmission is more common |
| **Parenteral** | 1. Transplacental transmission<br>2. Exposure to infected blood and blood products (most potent). Blood transfusion with infected blood (90–95%)<br>3. Exposure to infected tissue fluids<br>4. Injectable drug users. |
| **Vertical** | 1. Intrauterine transplacental before 36 weeks (20%)<br>2. Intrapartum (50%) ] Peripartum<br>3. Postpartum breastfeeding (30%) ] (80%) |

**Table 36.5: Risk Factors for Vertical Transmission**

**A. Maternal factors**

1. Increased viral load
2. Increased biological efficacy of virus
3. Unprotected sex during pregnancy
4. Illicit drug use
5. Low CD4 and lymphocyte count in maternal blood
6. Presence of reproductive tract infections (RTI) and sexually transmitted infections, (STI) in mother
7. Low maternal Vitamin A, zinc levels
8. Invasive needling procedures in the antenatal period (e.g. amniocentesis, chorion villus sampling). If needed amniocentesis is preferred.
9. Premature rupture of membranes and presence of chorioamnionitis
10. Episiotomy and operative vaginal delivery
11. Concurrent ascending bacterial infection in genital tract.

**B. Foetal factors**

1. Premature fetus
2. Foetal ingestion of the virus
3. Invasive foetal monitoring like foetal scalp electrode application, foetal scalp blood sampling and umbilical cord blood samplings.
4. Prolonged exposure to maternal secretions (first twin)
5. Through breast milk depending on the immune response of the newborn and period of breastfeeding and infectivity of mother.
6. Mixed feeding-infant feeding on both breast and bottle.

## ETIOLOGY AND PATHOGENESIS

HIV is a retrovirus (HIV I and II). The diagramatic structure of the HIV virus is shown in Fig. 36.2, it encodes an enzyme, reverse transcriptase which allows DNA to be transcribed from RNA. The virus integrates within the host genome of the infected host cells producing multiple copies of the HIV genome as DNA. The main targets of the virus are the human CD4 'helper' lymphocytes, which start producing new virus particles and thus lose their central role in the immune response to fight various infections and certain cancers. There is significant fall in levels of (CD4) cells as well as inability of B lymphocytes to produce antibodies to HIV. In this way, HIV causes

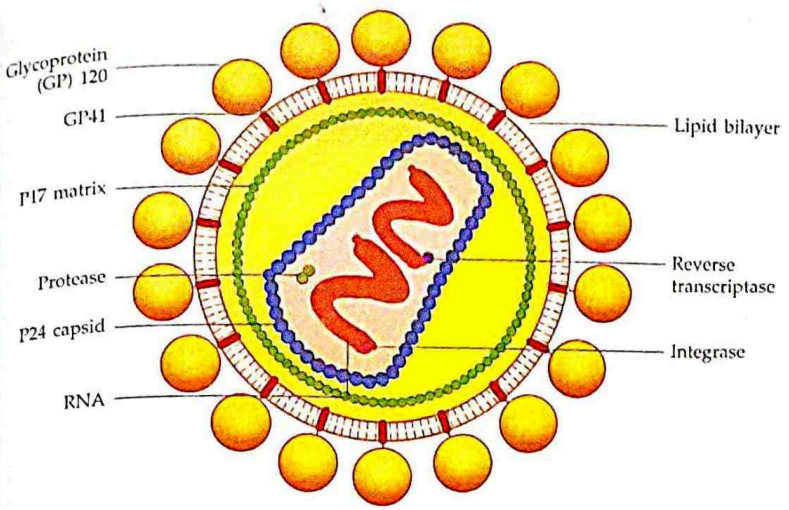



**Fig. 36.2:** Diagramatic representation of HIV-I Virus (virion)

progressive immunosuppression and ultimately the development of AIDS. The incubation period is 1 to 3 weeks. After exposure to virus infection, there is a window period of about 12 weeks in which antibody titre is not detectable. This is stage of primary infection which is highly infectious with CD4 cell count being near normal. Then there is an asymptomatic but infectious period of 3–5 years with detectable antibodies. There is early immunodeficiency with CD4 cell count being more than 500 cells/μL. Then there is an infectious period of 2–3 years with subtle symptoms and signs of immunodeficiency with detectable antibodies with intermediate immunodeficiency (CD4 cell count between 200–500 cell/μL). After another 1–2 years, there is full blown AIDS with symptoms and signs of opportunistic infections in which antibodies may or may not be detectable. It is highly infectious stage. There is terminal illness with CD4 cell count being < 200 cells/μL.

### Perinatal transmission

Vertical transmission rate is about 30% (13–25% in developed countries, 35% in developing countries). The various risk factors for increased perinatal transmission are given in Table 36.5. Viral load and perinatal transmission are directly related; 23% with maternal viral load of > 30,000 copies/ml but only 10% with maternal viral load of < 400 copies/ml. Most transmissions (40–80%) occur in labour. However, with the use of highly active antiretroviral therapy (HAART) during pregnancy, avoidance of invasive needling procedures in pregnancy and invasive monitoring techniques during labour, elective cesarean delivery, treatment of neonate with antiretroviral therapy and avoidance of breastfeeding has dramatically reduced perinatal transmission to as low as 1–2% in developed countries (RCOG 2010). In India by implementing PPTCT guideline 2013 mother to child transmission (MTCT) can be reduced from 35% to < 5% in breast-fed infants and from 25% to < 2% in nonbreast-fed infants.

### Effect of pregnancy on HIV

Pregnancy has not been shown to have an adverse effect on the natural history of HIV. The HIV is non-teratogenic. There is no benefit to maternal health from termination of pregnancy.

### Effect of HIV on pregnancy

Maternal mortality and morbidity rates are not increased in seropositive asymptomatic woman. If properly treated there is no increased incidence of abortion, preterm labour, foetal growth restriction (FGR) and perinatal mortality in HIV positive women. However, adverse perinatal outcome (preterm birth FGR) has been observed with low $CD4^+$ count (< 15%) and in developing countries with symptomatic and advanced disease (stage 2 and more).

### Preconception counselling

1. Routine testing of women of child bearing age group.
2. Screening test (ELISA—highly sesnsitive) is followed by a confirmatory Western blot assay (highly specific)
3. On site HIV testing with pre-post test counselling
4. Reproductive options for HIV descordant and concordant couples
5. Optimizing HIV management in infected partner
6. Timing of initiation of ART and adjustment of ART to optimal regimen for pregnancy.
7. Baseline infection risk, updating immunization
8. Behavioural changes in relation to substance abuse
9. Safe sexual practices

## CLINICAL PRESENTATION

The various clinical features and the WHO staging system for HIV/AIDS is shown in Table 36.6. Patient may be asymptomatic especially after primary illness. The time interval between HIV infection and full blown AIDS is about 10 years, but can be prolonged with antiretroviral therapy.

## DIAGNOSIS

HIV is suspected from the clinical features. The enzyme linked immunosorbent assay (ELISA) is used as a screening test for HIV antibodies with result available in 3 hours. It is highly sensitive (99.5%) but less specific. Rapid tests to detect antibody (immunoconcentration or particle agglulination or immunochromatography) are now commonly used with result available in 15 minutes. It has a sensitivity of 100% and specifically of 98.5%. ELISA kits are available for commercial use. The testing algorithm now used by National AIDS Control Organization (NACO) recommend 3 tests (ELISA/Rapid tests). If all 3 are positive: it is considered

**Table 36.6: WHO Staging System for HIV/AIDS**

| |
|---|
| **Stage 1** |
| Asymptomatic<br>Persistent generalized lymphadenopathy |
| **Stage 2** |
| • Unexplained weight loss < 10% body weight<br>• Recurrent respiratory tract infections (sinusitis, tonsillitis, otitis media, pharyngitis)<br>• Herpes zoster<br>• Angular cheilitis<br>• Seborrhoeic dermatitis<br>• Recurrent oral ulceration |
| **Stage 3** |
| • Unexplained weight loss > 10% body weight<br>• Chronic diarrhea > 1 month<br>• Persistent fever > 37.5 °C > 1 month<br>• Oral candidiasis, leukoplakia<br>• Pulmonary tuberculosis<br>• Severe bacterial infections (pneumonia, empyema, meningitis, bacteremia, joint infection)<br>• Ulcerative stomatitis, gingivitis or periodontitis<br>• Unexplained anaemia, neutropenia, thrombocytopenia |
| **Stage 4** |
| • HIV wasting syndrome<br>• Pneumocystis jiroveci (formerly Carnii) pneumonia<br>• Chronic herpes infection > 1 month<br>• Esophageal candidiasis<br>• Extrapulmonary tuberculosis<br>• Non tuberculous mycobacterial infection<br>• Recurrent septicemia<br>• Cytomegalovirus retinitis<br>• HIV encephalopathy, multifocal leukoencephalopathy<br>• Cryptococcal meningitis<br>• Disseminated mycosis, leishmaniasis<br>• Kaposi's sarcoma<br>• Chronic cryptosporidiasis, isosporiasis<br>• Invasive cervical cancer<br>• CNS toxoplasmosis<br>• Lymphoma |
| **Wasting syndrome** |
| Weight loss > 10% plus either chronic diarrhea, prolonged fever > 1 month or chronic weakness |

reactive (HIV positive). If two negative: It is non-reactive (HIV negative). If 1 negative, 2 positive: It is equivoval. Confirmation of diagnosis in equivoval test is done by Western blot test or immuno fluorescence test (IFA) or HIV NAAT test. Western blot test is highly specific with false positive rate of less than 1 in 10,000.

## MANAGEMENT OF HIV POSITIVE WOMAN IN PREGNANCY

1. The National AIDS Control Organization (NACO) of India recommends that all pregnant women irrespective of their risk factors should be counselled to undergo testing for HIV infection unless they opt out of it **(opt out HIV testing)** rather than **opt** in screening in which the patients have to **opt** for screening. Hence all pregnant women are routinely screened for HIV unless they refuse. If negative, patient is negative for HIV. Retest is recommended at 32 weeks in generalised and epidemics.
2. In HIV positive women, the initial booking visit should include a thorough history and physical examination including baseline fundus examination, neurological and pelvic examination. Such women should be cared in collaboration with physicians treating HIV positive women. Following tests should be performed.
   (a) Complete hemogram
   (b) Urine examination
   (c) Blood group
   (d) Tests for other STDs such as hepatitis B, gonorrhoea, syphilis, chlamydia and herpes
   (e) Serological testing for toxoplasmosis and cytomegalo virus infection
   (f) Tests for tuberculosis
   (g) Serological testing of husband for HIV should be offered.
   (h) Liver function tests
   (i) HIV viral load (RNA) at initial visit and repeated each trimester.
   (j) $CD4^+$ lymphocyte count with flow cytometry at initial visit and repeated in each trimester. If the $CD4^+$ count falls to 200 cells/$mm^3$, the patient is given prophylaxis against Pneumocystis jiroveci and other opportunistic infections.
3. The woman and her partner are counselled about the risk of HIV transmission to the fetus and neonate. Barrier (condom) method is advised for sero discordant couples. Medical termination of pregnancy is only offered on demand or for severe disease (AIDS) after proper counselling. She should also be counselled about the need for regular follow-up, CD4+ count monitoring and antiretroviral therapy to minimize perinatal (mother to child) transmission.
4. Higher folic acic 5 mg/day is given. Iron, calcium and vitamin supplements are necessary to improve the general condition of the patient. Nutritional supplementation may also be required.
   Minimum 4 antenatal visits (registration at 12 weeks) 14–26 weeks, 28–32 weeks and 36–46 weeks. Assign WHO clinical stage screen for TB and STIs. Partner testing. Assess weight gain. Refer to ART centre. Start ART. CD4 count is done.
   Multidisciplinary team: HIV physician, health advisor, obstetrician and neonatologist
   • Screening for diabetes for those on protease inhibitor
   • Consider hepatitis B, pneumococcal and influenza vaccination.
5. Invasive testing like amniocentesis should be avoided.
6. **Antiretroviral therapy.** There have been major changes in recommendation for antiretroviral therapy (ART) during pregnancy by WHO (2013) which has been accepted by NACO (2014). If a patient is diagnosed

**Table 36.7: WHO PMTCT Recommendations**

| |
|---|
| **Option B+** |
| Regardless of CD4 count or WHO clinical stage<br>Initiate and maintain lifelong for ALL<br>• Particularly in generalized epidemic settings.<br>• Settings with limited CD4 testing.<br>• Long duration of breastfeeding.<br>• High rates of fertility.<br>• Limited partner testing; high partner discordancy rates. |
| **Option B** |
| **1. Eligible women (ART required for mother's health)** |
| Initiate and maintain lifelong |
| **Indications for ART for mother's health:** |
| Stage I and II - if CD4 < 500<br>Stage III and IV - irrespective of CD4 |
| **2. Non-eligible (ART given to prevent transmission)** |
| Initiate for ALL; STOP<br>• 1 week after cessation of breastfeeding<br>• Immediate post-delivery (replacement feed) |
| PMTCT: Preventing Mother to Child Transmission |

**Table 36.8: The Dosage and Type of Anti-retroviral therapy for HIV Positive Mother**

| |
|---|
| **Preferred first line therapy** |
| 2 NRTI + 1 NNRTI<br>1. TDF 300 mg + 3 TC 300 mg + EFV 600 mg<br>Single OD dosage drug combination |
| 2. TDF 300 mg + FTC 200 mg OD + EFV 600 mg<br>Single OD dosage drug combination |
| **Alternative first line therapy** |
| 1. AZT 300 mg BD + 3 TC + EFV (or NVP)<br>2. TDF + 3TC (or FTC) + NVP 200 mg OD |
| NNRTI: Nonnucleoside reverse transcriptase inhibitors<br>NRTI: Nucleoside reverse transcriptase inhibitors<br>TDF: Tenofovir; 3TC: Lamivudine; EFV: Efavirenz; FTC: Emtricitabine; AZT: Zidovudine; NVP: Nevirapine |

to be positive for HIV in first trimester for the first time the appropriate management will be to start immediately and continue lifelong ART irrespective of CD4 count or WHO clinical staging (NACO 2014).There has been a shift from option A to Option B or B+ as shown in Table 36.7. NACO operational guideline (Jan 2014) has decided to adopt option B+ for life long ART (Table 36.7) as there is prolonged breastfeeding in India and up to 25–35% women have second pregnancy. Logic for option B+ by WHO is to initiate and maintain ART life long for all particularly in epidemic setting and settings with limited CD4 testing, long duration of breast feeding, high rate of fertility and limited partner testing and high partner discordancy rate. Option B can also be used, if it is not possible to give option B+. The dosage of drugs is given in Table 36.8 while the benefits of option B or B+ over option A are given in Table 36.9.

**Table 36.9: Rationale of Shift from Option A to B+ or B**

| Benefits for Mother and Child | Benefits for Program Delivery and Public Health |
|---|---|
| Improves adherence with once daily, single pill regimen | More effective, lesser adverse effects |
| Prevents MTCT in future pregnancies (early protection) | Same simplified regimen for all pregnant women during pregnancy, labour and post-partum |
| Ensures all ART eligible women initiate treatment (no need for CD4 testing) | |
| Reduces potential risks from treatment interruption | Harmonized with non-pregnant ART regimen |
| Potential health benefits of early ART for non-eligible women | Cost effective |
| Reduces sexual transmission of HIV (92% reduction) | Reduction in number of steps along PMTCT cascade |
| **Major issue now is not "when to start" or "what to start" but "whether to stop"** | |

Antiretroviral therapy is highly effective in reducing the viral load and perinatal transmission. Therapy is given depending upon the gestation at diagnosis. Women with stable CD4+ counts and undetectable viral loads who are already on antiretroviral regimen before pregnancy should continue on the same therapy even during the first trimester. For women diagnosed with HIV infection during pregnancy, treatment should be started as soon as possible. Following regimens can be given.

(a) For newly diagnossed pregnant Women: **HAART (Highly Active Antiretroviral Therapy)** is the best regimen as it can minimize the perinatal transmission to less than 2%. However, it is an expensive treatment and is difficult to provide in healthcare services in developing countries. But with prices of drugs falling significantly and more budgeting on NACO by government, it will become a routine treatment in future. The US Public Health Service Task Fore (2009) has recommended to continue current medication if viral suppression is adequate and patient is tolerating the drugs including efavirenz which can now be used in first trimester (It was category D drug earlier but recent studies and metaanalysis have found no increased rate of birth defects with EFV). WHO (2013) - also recommends use of EFV in first trimester. Usually, two drugs from group A (nucleoside analogs) and one from group B (protease inhibitors) or group C (non nucleoside analogs) or group D are given as shown in Table 36.10.

The triple therapy, if possible, should be the treatment of choice and should be started as soon as possible, continued throughout the pregnancy,

labour and postpartum period. The side effects of antiretroviral therapy include neuropathy, myopathy, lactic acidosis, pancreatitis, hepatitis and mitochondrial toxicity.

(b) **For pregnant women having prior exposure to non nucleoside analogs (NNRTI):** The preferred regimen is to give 2 drugs from group A (nucleoside analogues) along with ritonavir boosted protease inhibitor (LPV/r). Lopinavir (200 mg)/ritonavir (50 mg) combination has to be taken as 2 tablets twice daily (total dose being 800/200 mg per day).

(c) **Nevirapine (NVP) therapy.** It is very cost-effective and is given as single dose (200 mg) orally 4 hours before cesarean section (or at onset of labour and as a single dose to the baby (2 mg/kg) within 72 hours of birth. It was recommended by NACO in India. But there is rapid development of resistance to it. Now option B or B+ are used.

## Care during labour

In recent recommendation by WHO (2013) and NACO (2013), cesarean delivery is to be done only for obstetric indications, otherwise vaginal delivery can be allowed especially if the woman is on ART in pregnancy

1. **Mode of delivery.** Elective cesarean delivery can decrease the risk of vertical transmission by approximately half. Royal College of Obstetricians and Gynaecologists (RCOG, 2010) has recommended elective cesarean delivery at 38 weeks for HIV positive mother taking HAART regimen. Vaginal delivery is only allowed if woman is on HAART with viral load < 50 copies/mL. The American College of Obstetricians and Gynecologists (2016) also recommended elective cesarean if viral load is > 1000 copies/mL or unknown. The morbidity of cesarean is 5–7 times as compared with vaginal delivery in HIV positive women. The value of cesarean section decreases if more than 4 hours have passed between rupture of membranes and delivery. However, vaginal delivery can be recommended if the woman is on combination antiretroviral therapy and if the viral load is < 1000 copies. WHO 2013 and NACO (2013) recommend cesarean delivery only for obstetric indication. During cesarean, ARV prophylaxis is given. Early cord clamping. Avoid rupture of membranes. Use of blunt needles and peripartum antibiotics. Universal precautions to be followed.
2. Factors that increase the risk of vertical transmission of HIV like artificial rupture of membranes (amniotomy), use of foetal scalp electrodes and determination of scalp blood pH should be best avoided.
3. Maternal blood sample is taken for viral load and $CD4^+$ count.
4. **ART.** Zidovudine as loading dose of 2 mg/kg/hr by intravenous infusion may be given 4 hours before cesarean section (or at the onset of labour for vaginal delivery) followed by maintenance dose of 1 mg/kg/hr until cord clamping. For a women diagnosed with HIV first time in labour, A whole blood finger prick test should be done and if a single test comes to be positive, send the samples for confirmatory testing and start the woman with HAART.
5. The obstetrician, pediatrician and other staff attending the women must use universal precautions like caps, masks, gowns, double gloves and protective eyes wear (goggles) and boots for their own safety. The operating table floor should be cleaned with 0.5% sodium hypochlorite or 10% lysol.
6. Avoidance of needle stick injuries by using blunt tipped needles, avoiding resheathing of needles and other measures.

**Table 36.10: Classification of Anti-retroviral Drugs**

| Antiretroviral drugs | FDA Category |
|---|---|
| **A. Nucleoside analogs (Nucleoside reverse transcriptase inhibitors NRTIs)** | |
| 1. Tenofovir (TDF) | B |
| 2. Zidovudine (AZT) | C |
| 3. Lamivudine 3TC) | C |
| 4. Zalcitabane | C |
| 5. Stavudine | C |
| 6. Didanosine | B |
| 7. Abacavir | C |
| 8. Emtricitabine (FTC) | B |
| **B. Protease inhibitors** | |
| 1. Indinavir | C |
| 2. Ritonavir | B |
| 3. Saquinavir | B |
| 4. Lopinavir | C |
| 5. Nelfinavir | B |
| 6. Alazanavir (ATV) | B |
| 7. Darunavir (DRV) | C |
| **C. Non-nucleoside analogs (non-nucleoside reverse transcriptase inhibitors, NNRTIs)** | |
| 1. Nevirapine (NVP) | B |
| 2. Delavirdine | C |
| 3. Efavirenz (EFV) | C (was D earlier) |
| 4. Etravirine ETV | B |
| **D. Entry Inhibitors** | |
| 1. Enfuvirtide | B |
| 2. Maraviroc | B |
| **E. Integrase inhibitors** | |
| 1. Raltegravir | C |
| 2. Elvitegravir (EVG) | C |

7. Disposable syringes and needles should be used which are then disposed in puncture proof containers.
8. Healthcare workers attending these women should avoid contact with potentially infected body fluids. The risk of infection after mucus membrane exposure is about 0.36%.

### Care of neonate

Avoid invasive nasogastric suctioning. Wash away blood from the newborn. Baby should be bathed immediately.

### Breastfeeding

Breastfeeding was contraindicated in past to decrease the mother to child transmission (MTCT) of virus. According to WHO, decision about breast feeding should be made by National Health Authorities and Individual Mothers. According to 2013 National Guidelines on feeding, is exclusive breastfeeding for 6 months and continue feeding is upto 1 year in children who test negative in early infant diagnosis and upto 2 years in children who test positive in early infant diagnosis and receiving ART. Only when breast feeding can not be done or mothers's choice, then replacement feeding is given. Breastfeeding is avoided only when formula feed is acceptable, affordable, feasible, sustainable and safe.

### Antiretroviral therapy to the neonate

1. **Nevirapine therapy.** Nevirapine (NVP) is given as follows:

| Birth weight | NVP daily dose (mg) | NVP daily dose (mL) |
|---|---|---|
| < 2 Kg | 2 mg/kg OD | 0.2 ml/kg once daily |
| 2-2.5 Kg | 10 mg OD | 1 ml OD |
| > 2.5 Kg | 15 mg OD | 1.5 ml OD |
| Give birth dose of NVP within 6–12 hours of delivery | | |

Treatment is given upto 6 weeks irrespective of breastfeeding status. It is given upto 12 weeks if mother has not received ART for adquate duration of at least 24 weeks.

If mother is on life long ART, nevirapine is given to baby for 6 weeks (NACO), 2014, NIH recommends Zidovudine for 4 week.

2. **Zidovudine therapy.** Alternatively, Zidovudine syrup in dose of 4 mg/kg is given to the baby twice daily for the first 4–6 weeks of life (if on replacement feeding). This therapy is preferred in women infected with HIV-2.

Early infant testing: DNA PCR at 6 weeks and 6 months (dried blood sample). If positive, confirm with whole blood. Start ART if positive. Final diagnosis at 18 months by 3 rapid tests. Start-cotrimoxazole prophylaxis for all neonates from 6 weeks to 18 months.

**Postpartum care:** Ensure ART adherescace, Ensure no mixed feeding. Treat cracked nipples and mastitis. Treat depression. Continue antibiotic prophylaxis. Contraception advice as below. Pap smear after 6 weeks.

### Contraception

HIV positive women are strongly advised to use dual contraception or 'double dutch'. It involves using a barrier method (male or female condom) to reduce the risk of sexual transmission of the virus to the partner in combination with a more effective contraceptive method like combined oral contraceptive pills, levonorgestrel intrauterine system or sterilization. Combined oral pills, progestogen only pill and DMPA can be given and are category I (WHO) in HIV infection and AIDS. IUCD is category 2 for HIV infected and category 3 for AIDS. Spermicide is avoided.

### Counselling

Counselling of HIV positive women before, during and after pregnancy is very important so that she can be stressed the need of use of antiretroviral therapy and regular monitoring and proper care during pregnancy and delivery. She should make an informed choice about various decisions about her pregnancy, delivery and contraception.

### Four Prong Strategy plan to reduce MTCT (NACO)

1. Primary prevention of HIV among women of child bearing age.
2. Preventing unintended pregnancy in HIV infected women.
3. Preventing mother to child transmission of HIV during pregnancy.
4. Providing treatment, care and support for infected women and their families

## Post Exposure Prophylaxis (as per NACO Guidelines)

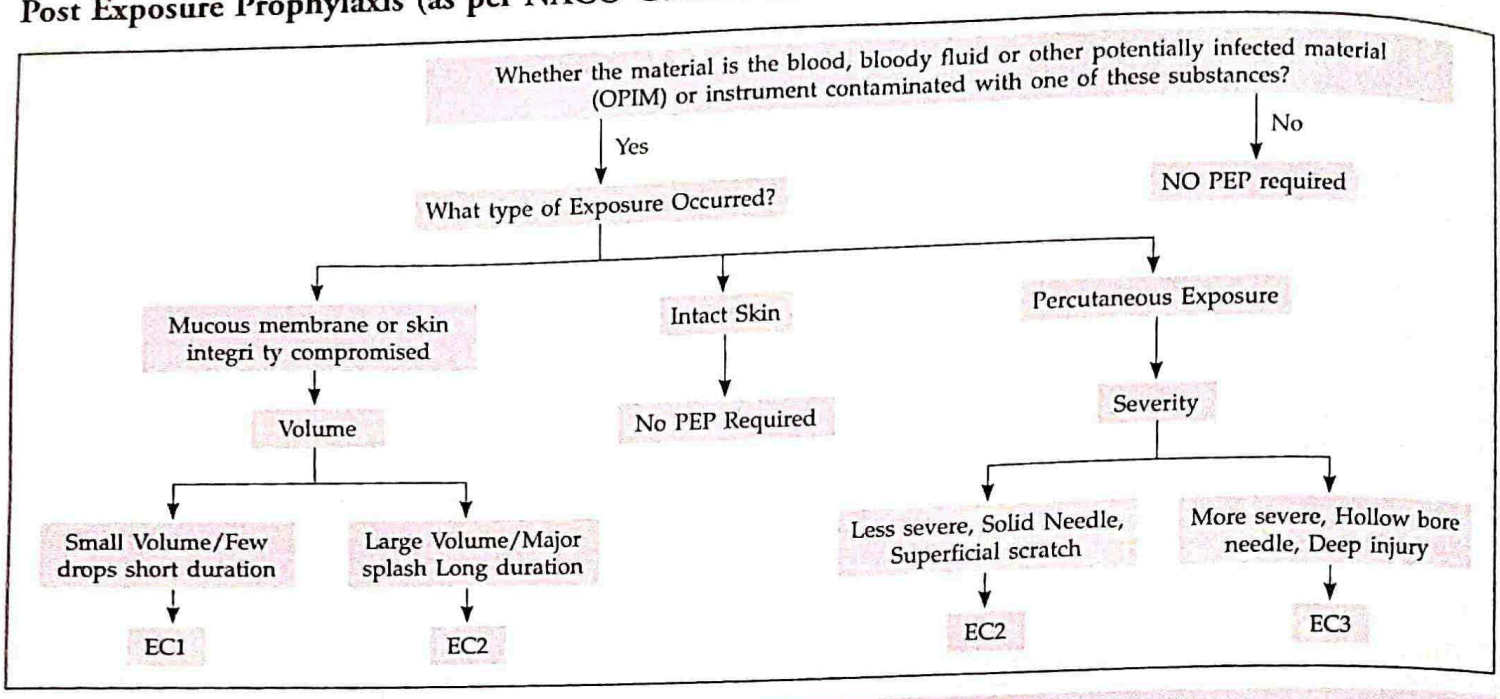



**Fig. 36.3:** HIV Exposure Codes

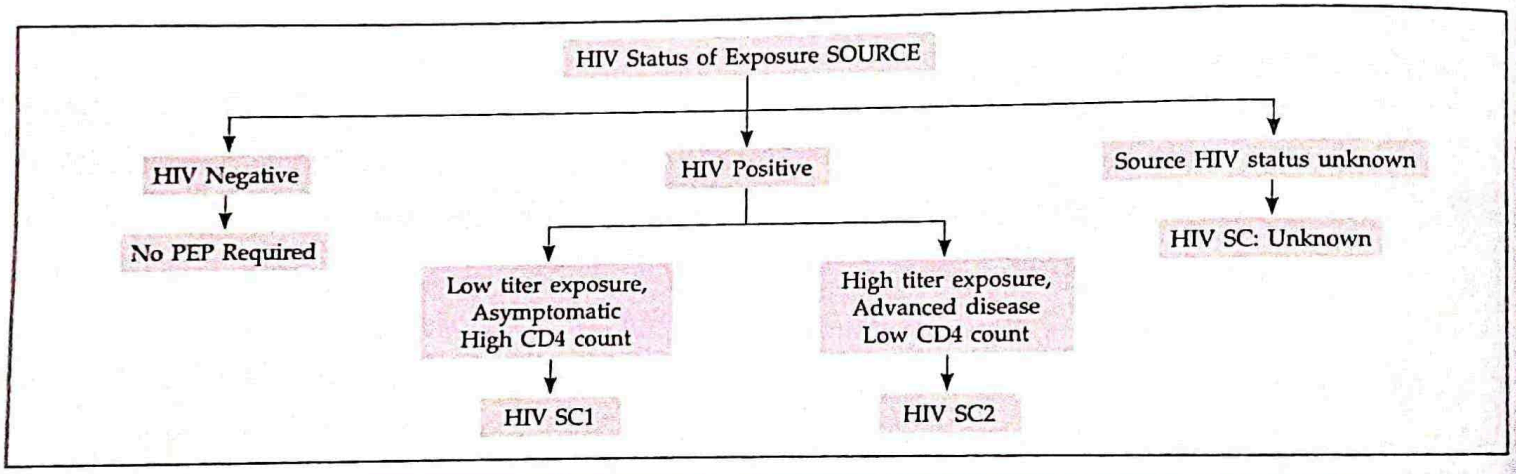



**Fig. 36.4:** HIV Source Codes

**Table 36.11: NACO Recommendations of PEP for HCP based on Exposure and HIV Source codes**

| Exposure Code | Source Code | Recommendatgion for PEP | Duration |
|---|---|---|---|
| 1 | 1 | Not warranted | |
| 1 | 2 | Recommended PEP | PEP is recommended for 28 days |
| 2 | 1 | | |
| 2 | 2 | | |
| 3 | 1 or 2 | | |
| 2/3 | Unknown | Consider PEP if HIV prevalence is high in given population and risk categoorization | |

As post-exposure prophylaxis (PEP) for HIV has its greatest effect if began within 2 hours of exposure, it is essential to act immediately. There is little benefit if > 72 hours have lapsed but PEP can still be used if the health care worker presents after 72 hours of exposure.

**Table 36.12: Recommended PEP regimens**

| Dosages of the drugs for PEP for adults | Recommendation for PEP | Duration |
|---|---|---|
| Tenofovir (TDF) 300 mg + Lamivudine (3 TC) 300 mg One Tab (FDC) once daily (1-OD) | One tab Immediately within 2 hours of accidental exposure, either at day time or at night time | Next day one tab once OD, continue for 4 weeks |
| Lopinavir (200 mg) + Ritonavir (50 mg) Two Tab (FDC) twice daily (2-BD) | Two Tab immediately within 2 hours of accidental exposure, either at day time or at night time | Next day two tab BD, continue for 4 weeks |
| IF LPV/r is not available/ cannot be used, Tenofovir (300 mg) + Lamivudine (300 mg) + Efavirenz (600 mg), One Tab OD may be given for 4 weeks. | | |

## Key Points

1. Opt out HIV testing is recommended by NACO to all pregnant ladies.
2. For HIV positive women, care should be taken by obstetrician and physician interested in HIV infection.
3. Factors causing increased mother to child transmission (MTCT) are low CD4+ count in mother, high HIV virus load, invasive prenatal diagnostic procedures (like amniocentesis, chorion villus sampling) or invasive intrapartum procedures (like amniotomy, foetal scalp electrode application, foetal scalp blood sampling), prolonged interval between rupturing of membranes and delivery, vaginal infection and Vitamin A deficiency).
4. Vertical transmission can be reduced to less than 2% by universal screening, undetectable maternal HIV RNA, antiretroviral (zidovudine/nevirapine or HAART) therapy during pregnancy, avoiding invasive procedures, delivery by elective cesarean section and exclusive formula feeding (avoiding breastfeeding) **(level IA evidence)**.
5. Elective cesarean delivery for HIV positive women at 38 weeks is recommended by RCOG (2010) for > 50 viral copies/mL and by ACOG (2000) with more than 1000 viral copies/mL. However, vaginal delivery is advised by NACO if viral load is less than 1000 copies/mL. NACO and WHO recommend cesarean delivery only for obstetric indications.

## Suggested Reading

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5. Centers for Disease Control and Prevention. HIV among pregnant women, infants and children 2017. httt://www.edc.gov/hiv/group/gender/pregnant women.
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# (iv) TORCH AND OTHER VIRAL INFECTIONS IN PREGNANCY

TORCH includes toxoplasma (T), others like Parvo (O), rubella (R), cytomegalo virus (C) and herpes (H) infection.

## TOXOPLASMA INFECTION IN PREGNANCY

Toxoplasma is caused by a protozoan *Toxoplasma gondii*, with infection being transmitted by eating raw or undercooked meat which is infected with tissue cysts or through contact with oocysts from infected cat feces in contaminated water, soil or litter. It is more common in France. Toxoplasm causes maximum damage in first trimester but maximum transmission in 3rd trimester.

### Maternal effects

Most women are asymptomatic with acute infection being detected by finding IgM specific antibodies. It is a self-limiting disease.

### Foetal effects

Fetus is at risk of infection if mother was seronegative before and acquires infection in current pregnancy. Foetal infection increases with gestation being 15% in first trimester rising to 30% in second trimester and 60% in the last trimester. However, the severity of foetal infection and foetal damage is much greater in early pregnancy (about 60% in first trimester) and decreases significantly later (almost nil at term). **There is increased risk of miscarriages, foetal growth restriction and intrauterine death in toxoplasmosis. Transplacental passage occurs during maternal parasitemia with fetus developing hydrocephalus, microcephaly, cerebral calcifications, choriodoretinitis and mental retardation.**

### Diagnosis

Clinical diagnosis is difficult due to asymptomatic nature of the disease. Detection of toxoplasma specific IgM antibodies, seroconversion for IgG from negative to positive or very high levels of IgG is indicative of acute infection. Routine screening is not recommended in India and it is not a cause of recurrent miscarriages. In presence of maternal infection, foetal infection can be tested by amniocentesis or cordocentesis for detection of IgM antibodies or polymerase chain reaction (PCR) for *Toxoplasma gondii*. Ultrasonography at and after 20 weeks may demonstrate foetal hydrocephalus or brain calcification. Medical termination of pregnancy is offered in presence of foetal affection.

### Prevention

Pregnant women should avoid contact with cat and must not touch cat's feces or should use gloves. She should also avoid consumption of unpasteurised milk and improperly cooked meat.

### Treatment

Treatment of choice for proven toxoplasmosis during pregnancy is spiramycin given in dose of 1 g (3 million IU) 2–3 times orally a day for 3 weeks courses with interruption of 2 weeks drug-free period until delivery. To eradicate parasites in placenta and fetus pyrimethamine 25 mg orally daily with oral sulfadiazine 1 g four times a day for 3 weeks during pregnancy is given alternating with 3 weeks spiramycin (1 g thrice daily). Maternal treatment reduces the risk of congenital infection and its late sequelae but does not completely eliminate it.

## RUBELLA (GERMAN MEASLES) INFECTION

Gregg, an ophthalmologist from Australia was the first to prove the teratogenicity of rubella infection by observing increased incidence of congenital cataracts in children born to mothers with rubella in their first trimester. Rubella is caused by a RNA Togavirus. Gregg triad comprises of cataract, sensory neural hearing loss and PDA (Patent ductus arteriosus). Single most common effect is sensory neural deafness.

### Maternal effects

Mother is usually asymptomatic (50%) or has low grade fever with slight rash which may go unnoticed. There may be arthralgia or arthritis, lymphadenopathy or conjunctivitis, Forchheimer spots (tiny rose colored lesions in mouth). Most serious in encephalis. Post infection thromobcytopenia can also occur.

### Foetal effects

Rubella is one of the most teratogenic agents known especially when contracted in the first trimester (up to 80% risk) causing following effects.

**Congenital rubella syndrome.** The risk of foetal affliction is 90% before 12 weeks, 50% between 13–14 weeks, 25% between 15 and 20 weeks and minimal after 20 weeks.

(*i*) Congenital heart disease: patent ductus arteriosus (common heart defect), septal defects, pulmonary artery stenosis
(*ii*) Central nervous system defects: microcephaly, intellectual disability, mental retardation and meningoencephalitis
(*iii*) Eye defects: congenital cataract, micro-ophthalmia, cloudy retina, retinopathy, congenital glaucoma
(*iv*) Ear defects: Including cochlear defects (sensorineural deafness)
(*v*) Hepatosplenomegaly and jaundice
(*vi*) Neonatal purpura (Blueberry muffin skin lesion)
(*vii*) Radiolucent bone disease

There is increased chance of miscarriages and intrauterine deaths in the affected fetuses. These neonates can be a threat to other infants and adults as they shed virus for many months.

**Extended rubella syndrome** is a delayed disease comprising progressive panencephalitis, hearing loss and type I diabetes developing in the second or third decade of life.

### Diagnosis

If the pregnant woman is exposed to a known case of rubella in pregnancy her blood should be tested for rubella specific IgM and IgG. Presence of IgM in newborn or persistence of IgG after 6 months is diagnostic of congenital rubella. Presence of rubella IgG in absence of IgM shows that she is immune to rubella due to infection in the past or due to past vaccination and no further action is required. Rising levels of IgG may be significant. However, presence of rubella specific IgM especially in early pregnancy in absence of immunity in the mother is an indication of medical termination of pregnancy.

### Prevention

All adolescent girls before planning marriages if not already vaccinated with live attenuated measles mumps rubella (MMR) vaccine, should receive rubella vaccine 0.5 mL subcutaneously. When given during childbearing period, pregnancy should be avoided for one month, rather than 3 months as advised before (Center for Disease Control and Prevention, 2013). Rubella vaccination should be avoided 1 month before or during pregnancy as it is a live virus vaccine. However, termination is not usually recommended if inadvertently vaccine is given as though there is theoretical risk, practically there is no evidence of increased malformations (CDC, 2013).

## CYTOMEGALO VIRUS INFECTION (CMV)

Cytomegalo virus is a DNA virus which is present in body fluids and is transmitted from person to person by contact with infected nasopharyngeal secretions, saliva, urine, semen, cervical secretion or blood. There may be intrauterine or intrapartum infection or neonatal infection from breastfeeding.

Maternal infection is usually asymptomatic or she may have fever, arthralgia, pharyngitis, lymphadenopathy or polyarthrits.

**Foetal effects.** Transmission rates for primary infection are 30–36% in first trimester, 34–40% in second trimester and 40–72% in the third trimester (ACOG 2017). Transmission is < 1% for repeat infection. There are increased chances of **miscarriage, foetal growth restriction, intrauterine death, microcephaly, intracerebral calcification, choriodoretinitis, ventriculomegaly, mental retardation, hepatosplenomegaly, thrombocytopenia and deafness in the newborn.**

### Diagnosis

Diagnosis is by demonstration of IgM in maternal serum though routine screening is not advised (Society for Maternal Foetal Medicine, 2016). Foetal affection is diagnosed by demonstration of CMV IgM in foetal blood on cordocentesis or by ultrasonography, CT scan or MRI of fetus. CMV Nucleic Acid Amplification Testing (NAAT) of Amniotic Fluid is considered gold standard for diagnosis of foetal infection. Congenital CMV infection is diagnosed by detection of virus in saliva or urine (saliva > urine). It's gold standard test in first two weeks. PCR can also be used to detect small amounts of CMV DNA in urine. Urine is better sample for culture of CMV than saliva.

### Treatment

There is no specific treatment for CMV in pregnancy. Antiretroviral therapy with valacyclovir is useful for neonate but is not effective to prevent in vitro CMV infection. Passive immunization with CMV specific hyperimmune globulin decreased risk of congenital CMV infection in some studies. There is no CMV vaccine.

## HERPES SIMPLEX VIRUS (HSV) INFECTION

Genital infection is caused by DNA virus HSV-2 and may be primary episode or a recurrent episode. Transmission is by sexual contact. Infection during pregnancy may be primary or reactivation or recurrent infection with or without symptomatic lesions.

### Maternal effects

HSV may be asymptomatic or there may be painful vesicular or knife-cut lesions on genital organs.

### Foetal effects

The virus can be transmitted to the fetus in three ways; peripartum (most common 85%), postnatal (10%) and intra uterine (least common 5%).

Rarely transplacental passage of the virus occurs causing miscarriages. Fetus is mostly affected by virus shed from cervix or vagina in vaginal deliveries. There is increased chance of **preterm labour, foetal growth restriction** and **death** if the infection is acquired in the last trimester. The risk of neonatal infection is much higher (30–50%) in women who acquire primary HSV infection near time of delivery possibly due to higher viral loads and the lack of transplacental protective antibodies. In contrast, women with recurrent HSV infection have less than 1% risk of neonatal infection.

Various other foetal effects can be **microcephaly, choriodoretinitis, mental retardation, convulsions** and **death.**

### Diagnosis

Though cell culture from lesions is preferred, sensitivity is low. Polymerase chain reaction (PCR) from lesion. is more sensitive. Serological ELISA test for HSV-2 antibody can be done. Routine screening in asymptomatic pregnant woman is not recommended (ACOG 2016).

### Treatment

Mother is treated by oral acyclovir 400 mg three times a day for 7–10 days for active disease and 400 mg thrice daily

for 5 days for recurrent infection. It is safe in pregnancy. Valacyclovir 1 g twice daily for same duration can also be given.

### Mode of delivery

To prevent foetal infection during delivery, elective cesarean delivery is recommended for active primary genital HSV infection or active recurrent genital lesion **(level II evidence)** (ACOG 2016). For women with clinical recurrence of HSV at delivery, there is not an absolute duration of membrane rupture beyond which fetus would not benefit from cesarean delivery (ACOG, 2016).

### Neonatal infection

Neonatal infection with HSV is high following primary infection and can be disseminated disease which is very serious (usually fatal) or localized disease or may be asymptomatic. Neonatal diagnosis is made by PCR testing for viral DNA. Women with active HSV may breastfeed if there are no active HSV breast lesions. They must wash their hands before breastfeeding. Valacyclovir or acyclovir may be used during breastfeeding.

## OTHER VIRAL INFECTIONS IN PREGNANCY

### Chickenpox

Chickenpox is a rare infection caused by Varicella Zoster Virus (VZV) with incubation period of 10–21 days. It spreads by direct contact or via respiratory droplets or secretions. Maternal complications include varicella pneumonia (20%) with high mortality (14%), encephalitis, myocarditis, hepatitis, glomerulonephritis and thrombocytopenia. It attaches to keratinocytes and dendric cells of skin.

#### Foetal effects

Maternal varicella infection in pregnancy can cause **congenital varicella syndrome characterized by limb hypoplasia, limb paresis (deformity), cicatrical skin lesions, microcephaly, cataracts and choriodoretinal scarring.** The risk depends upon the timing of pregnancy being 0.4% in first trimester, 2% from 13 to 20 weeks increasing to 17 to 30% if onset of varicella in the mother is from 5 days before delivery to 2 days after delivery. The risk of foetal varicella syndrome is maximum when mother is infected between 13 to 20 weeks of gestation.

#### Treatment

Ideally seronegative women should receive vaccine before pregnancy as the vaccine is contraindicated in pregnancy. Varicella Zoster Immune Globulin (VZIG) should be given within 72 hours of exposure to all pregnant women with significant exposure to Varicella Zoster Virus (VZV) with no history of chickenpox to reduce the morbidity in mother and newborn. VZIG should also be administered to the neonate within 5 days of delivery.

All pregnant women with varicella zoster infection during pregnancy should receive oral acyclovir (800 mg five times a day) or valacyclovir (1 g thrice a day). In case of varicella pneumonia or neurological symptoms intravenous acyclovir (10–15 mg/kg body weight) every 8 hourly is given.

### Measles

Measles is caused by measles virus which is RNA paramyxo virus. It does not cause teratogenic effect but can cause **miscarriages, preterm labour,** low birth weight and **intrauterine death** due to high-grade fever. Active vaccination is contraindicated in pregnancy. Non-immunized pregnant women exposed to a patient with active measles should be given serum immune globulin 400 mg/kg, intravenously within 72 hours of exposure for protection (CDC 2017). Incidence is low due to routine prophylaxis of all by live attenuated MMR (measles mumps rubella) vaccine which is contraindicated in pregnancy.

### Mumps

Mumps is caused by mumps virus which is a RNA paramyxo virus. It is not teratogenic and does not cause foetal infection. Mumps in pregnancy is like in non-pregnant state. However, maternal infection can cause spontaneous abortions. Its prevalence is very low in pregnancy due to routine prophylaxis of all with live attenuated MMR vaccine (contraindicated in pregnancy).

### Parvo virus

Parvo virus B19 is a small stranded DNA virus causing **erythema infectiosum** or **Fifth disease.** Maternal infection can be asymptomatic or there may be fever, headache, flu like symptoms followed by red rash on face, trunk and extremities. There is 33% vertical transmission rate. It affects the fetus by transplacental route causing abortion, anaemia, non-immune hydrops and stillbirth with an overall foetal loss of 10%. Infact, it is an important cause of non-immune hydrops (in Rh positive patients). Foetal loss is much higher (8–17%) when infection occurs before 20 weeks and is 2–6% after that. Diagnosis is by serological testing for specific IgG and IgM antibodies. Viral DNA can be detected by PCR in maternal serum. Serial ultrasonography is done to see foetal anaemia. Foetal blood sampling is performed in hydrops to assess the degree of foetal anaemia. In utero foetal blood transfusion may improve outcome with most fetuses requiring only one transfusion as hemopoiesis resumes after resolution of infection. There is no specific treatment.

### Influenza

Influenza is caused by influenza virus and usually has no effect on the course of pregnancy. However, virulent infection may cause increased incidence of **miscarriages** and **preterm labour** due to high grade fever. It is not teratogenic but Asian influenza has been reported to cause anencephaly when the infection occurs in early pregnancy. Influenza vaccine can be given safely after the end of first trimester (also see Chapter 35[v] for details).

### Hepatitis virus infection

See Chapter 35[vii] for details.

### Zika virus infection

It is an RNA virus of Flavivirdae family which is acquired by mosquito bite or rarely by sexual transmission. It resembles Rubella infection and may be asymptomatic or causes mild symptoms of rash, fever, headache, arthralgia and conjuctivitis. It attaches to Haufbauer cells of placenta to cause vertical transmission. It is the only mosquito born teratogen known.

**Foetal effects.** It can severely affect the fetus and is a teratogen causing congenital Zika syndrome characterised by microcephaly (small head), lissencephaly (small head with absence of normal convolutions or folds of cerebral cortex), ventriculomegaly, intracranial calcification, eye anomalies, contractures, hypotonia and club foot. Incidence is 15% in laboratory confirmed Zika cases (5% in suspected cases).

**Diagnosis.** Detection of Zika virus RNA in maternal blood or urine by PCR confirms the diagnosis. Zika antibody can also be detected in patient's serum but may cross react with other infections. Ultrasound for foetal cranial defects can be done antenatally. Prevention is by anti-mosquito measures (mosquito nets or insecticide sprays) like for malaria.

**Treatment.** There is no specific treatment or vaccine available. CDC has started us zike pregnancy registry for such cases.

## Key Points

1. Toxoplasma infection during pregnancy may cause miscarriage and may also result in a live born infant with afflictions like hydrocephalus, microcephaly, cerebral calcifications and mental retardation and is treated by oral spiramycin (drug of choice) 3MU twice daily for 3 weeks followed by 2 weeks rest throughout pregnancy.
2. Rubella infection in the first trimester of pregnancy in a non-immunized woman causes major foetal congenital malformations justifying termination of pregnancy. Congenital rubella syndrome involves multiple organs (heart, eyes, ears, liver, bone marrow and bones). Measles, mumps and rubella vaccine (MMR) or rubella vaccine before pregnancy can prevent it.
3. Cytomegalovirus (CMV) infection in pregnancy is usually asymptomatic with the diagnosis being rarely made until the infant is born with CMV affection like microcephaly, mental retardation and cerebral calcification.
4. Symptomatic or asymptomatic herpes virus infection may occur in pregnancy and may result in foetal infection. Most neonatal infections are acquired from active maternal genital disease during delivery. Hence, elective cesarean delivery in the presence of active herpes disease in mother with intact membranes (or within 6 hours of rupture of membranes) can prevent this disease in the newborn **(level II evidence)**.
5. Chickenpox caused by Varicella zoster virus can cause serious maternal complications like varicella pneumonia, encephalitis, myocarditis, hepatitis, etc. and congenital malformations in the fetus and is treated by Varicella zoster immunoglobulin and acyclovir or valacylovir to the mother during pregnancy.
6. Parvo virus B19 infection is an important cause of non-immune foetal hydrops due to transplacental passage of the virus which affects foetal erythroid precursor cells.
7. Zika virus infection in pregnancy can cause microcephaly and lissencephaly in fetus.

## Suggested Reading


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5. Royal College of Obstetricians and Gynaecologists. Chickenpox in Pregnancy. Green Top Guideline No. 13, RCOG London, 2015.
6. Centers for Disease Control and Prevention: General Recommendations on Immunization. MMWR 2002a; 51 (RR-1) : 1.
7. Powrie R O et al (eds). de Swiet's Medical Disorders in Obstetric Practice. Wiley Blackwell, Oxford, U. K. 5th edition, 2010.
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9. Cunningham FG et al. Williams Obstetrics McGraw Hill Education, New York, USA, 25th edn., 2018, p 1209.
10. Driggers RW, Ho CY, Korhonen EM et al. Zika virus infection with prolonged maternal viremia and foetal brain abnormalities. New England Journal of Medicine 2016; 374:2142.

## (v) SEXUALLY TRANSMITTED DISEASES IN PREGNANCY

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment | Comments |
|---|---|---|---|---|---|---|
| 1. Syphilis | *Treponema pallidum* (Spirochete)<br>1. Sexual transmission<br>2. Vertical (maternal-foetal) transmission Maximum transmission to fetus between 10–15th week of gestation. | 1. **Primary syphilis.** Painless chancre with a raised, red firm border with smooth base.<br>2. **Secondary syphilis.** Lesions on palms and soles, cutaneous and mucus rash, condylomata lata (flesh colored papules and nodules) of genitalias, lymphadenopathy<br>3. **Latent syphilis.** Early diagnosed within 12 months. Late: diagnosed beyond 12 months. Clinical manifestations with reactive serological testing in women with untreated primary or secondary syphilis.<br>4. **Tertiary syphilis.** Cardiovascular syphilis, neurosyphilis | VDRL test (Venereal Disease Research Laboratory); It may be false positive. If VDRL is positive, specific tests like FTA-ABS (fluorescent treponemal antibody absorption test) or microhemagglutination assay for antibody to Treponema pallidum (MHA-TP) or the Treponema pallidium passive particle agglutination (TP-PA) tests are done. Reverse sequence (specific tests first and nonspecific test later) can also be done (CDC 2011) Foetal affection is diagnosed by ultrasound or PCR on amniotic fluids. All women should be offered HIV testing. Levaditi stain for syphilis: cork screw organisms (spirochaetes seen) | 1. **Gradually improving obstetric history** called Kassowitz law or (Diday Kassowitz law 1876: abortion only abortion) then stillbirth, then neonatal death followed by congenital syphilis and later even 1–2 normal babies.<br>2. Growth restriction, foetal hydrops<br>3. Neonatal infection, flu-like syndrome, generalized lymphadenopathy, maculopapular rash and condylomata lata.<br><br>**Congenital syphilis**<br>A newborn of a syphilitic mother with signs of congenital syphilis.<br>(i) Large abdomen due to hepato-splenomegaly<br>(ii) Newborn may have edema, ascites and hydropic features.<br>(iii) There may be jaundice with purpura and patechiae, lymphadenopathy, rhinitis or pneumonia. | 1. Early syphilis; Inj. Benzathine penicillinG 2.4 million (megaunits) half on each buttock by deep IM injection after sensitivity test. Second dose after 1 week can be given.<br>2. Late syphilis; Inj. Benzathine penicillin 2.4 megaunits weekly for three doses.<br>3. Neurosyphilis Inj. Aqueous crystalline penicillin G 3–4 million units intravenously every 4 hours for 10–14 days. or Aqueous procaine penicillin, 2.4 million units IM daily with probenecid 500 mg orally four times daily for 10–14 days. For penicillin sensitive patients, erythromycin 500 mg 6 hourly × 14 days or azithromycin 500 mg daily for 3 days. However, non-penicillin regimens do not prevent congenital syphilis. Hence, penicillin desensitization followed by benzathine penicillin G treatment is recommended. Neonatal syphilis is treated by penicillin. | Though syphilis is uncommon now, but it can cause preventable perinatal mortality and morbidity. Thus, screening for syphilis is done in all pregnant ladies at first visit by VDRL test as timely diagnosis and treatment can prevent adverse foetal effects. |

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment | Comments |
|---|---|---|---|---|---|---|
| 2. Chlamydia | *Chlamydia trachomatis* | Meconium stained liquor, pyrexia, pelvic inflammatory disease | Antibodies for chlamydia, ELISA test | Abortion, preterm labour, prelabor rupture of membranes, chorioamnionitis, foetal growth restriction, stillbirth, neonatal conjunctivitis and pneumonia | Treatment of Chlamydia:<br>**First choice:** Azithromycin 1 g orally as a single dose or Amoxicillin 500 mg orally thrice a day for 7 days.<br>**Alternative treatment.** Erythromycin 500 mg four times a day for 7 days or Erythromycin 250 mg four times a day for 14 days. Treatment reduces the number of pregnant woman with positive antibody by 90% and helps prevent pregnancy and neonatal complications **(level IA evidence)** | |
| 3. Mycoplasma | *Mycoplasma hominis* | Pyrexia, pelvic inflammatory disease | C-reactive protein, leucocyte count | Abortion, stillbirth | Erythromycin 500 mg 6 hourly × 7–10 days or Azithromycin 500 mg daily × 3 days or 1 g single dose or Roxithromycin 150 mg BD for 7–10 days. | |
| 4. Ureaplasma | *Ureaplasma urealyticum* | Preterm labour, premature rupture of membranes | Antibodies test, ELISA test | Abortion, preterm labour, stillbirth | Treat like mycoplasma | |
| 5. Gonorrhea | *Neisseria gonorrhoea* | Vulvitis, vaginal discharge, pelvic inflammatory disease, purulent exudates from cervix | Gram staining of purulent exudates and diplococci culture | Chorioamnionitis, preterm delivery, premature rupture of membranes, neonatal conjunctivitis | Treatment of gonorrhea<br>**First choice.** CDC (2015) recommends injection ceftriaxone 250 mg 1 M with 1 gm of azithromycin oral with 7 days abstinence. Alternate regimen is 400 mg oral cefixime with 1 gm oral azithromycin.<br>For Cephalosporin allergy 240 mg i.m. Gentamicin with 2 gm oral azithromycin is given.<br>All infants born to women with gonorrhoea are given prophylaxis against eye infection to prevent ophthalmia neonatorum. The eyes are wiped, two drops of freshly prepared 1% silver nitrate solution are instilled in each eye which is later washed with saline. | |
| 6. Chancroid (soft chancre) | Gram negative streptobacillus (*Hemophilus ducreyi*) | Soft chancre, painful ulcer on vulva, fever, painful lymphadeno pathy | Diagnosis is clinical. Demonstration of Ducrey's bacilli | Chorioamnionitis | Azithromycin 1 g oral single dose is the drug of **first choice**. Erythromycin 500 mg orally thrice a day for 7 days or inj. Ceftriaxone 250 mg IM single dose can be given. | |
| 7. Granuloma inguinale | Gram negative bacillus *Calymmato bacterium granulomatis* (*Donovan bodies*) | Ulcerative lesions of vulva and perineum, elephantiasis, non-suppurative inguinal lymphadenopathy | 1. Intranuclear Donovan bodies on Giemsa stain<br>2. Culture | Obstructed labour | Capsule Ampicillin 500 mg 6 hourly for 7 days.<br>Cesarean may be needed. | |

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment | Comments |
|---|---|---|---|---|---|---|
| 8. Lympho-granuloma venereum | *Chlamydia trachomatis* | Vulval ulcer, fibrosis, suppurative lymphadenitis fistulas in perineum in vulva and redness over perineum and in vagina | 1. Culture<br>2. Antibodies test | Obstructed labour | Erythromycin 500 mg 6 hourly × 21 days is the drug of **first choice**. Azithromycin for 3 weeks can be given. Cesarean may be needed. | |
| 9. Genital warts | Papilloma virus | Warty lesions | Clinical diagnosis, culture | Large lesions may make delivery and episiotomy difficult | Treatment of genital warts: Trichloroacetic acid solution applied topically once a week is an effective treatment for warts during pregnancy. Some prefer cryotherapy, laser ablation or surgical excision. Cautery to warts, Podophyllin therapy is avoided in pregnancy due to concern of maternal and foetal safety. Cesarean may be needed. | |
| 10. Molluscum contagiosum | DNA pox virus | Dome-shaped papules with central umbilication | Clinical diagnosis, culture | | Phenol locally. They can also be removed. | |
| 11. HIV infection (see above for details) | | | | | | |
| 12. HSV infection (see above for details) | | | | | | |
| 13. Vaginitis (bacterial vaginitis, trichomonal vaginitis and Candidal vaginitis), see Chapter 11 for details | | | | | | |

## (vi) MISCELLANEOUS INFECTIONS AND INFESTATIONS IN PREGNANCY

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment |
|---|---|---|---|---|---|
| 1. Listeriosis | *Listeria monocytogenes* Gram positive intracellular bacillus.<br><br>Up to 10% humans may carry it in their intestines. It can be associated with ingestion of contaminated foods like raw milk, meat and ready-to-eat food. | The symptoms are non-specific like fever, nausea, vomiting.<br>There can be septicemia and meningoencephalitis | Culture of organisms from blood, CSF or stools or serological testing. | It can predispose to miscarriages, preterm labour and stillbirth and **meconium stained liquor in premature gestation.**<br>Chorioamnionitis and placental lesions | **Ampicillin and gentamicin combination is the treatment of first choice due to synergism.** Trimethoprim sulfamethoxazole can be given to penicillin-allergic women. |
| 2. GBS infection | Group B Streptococcus, especially *Streptococcus agalactiae*. It colonizes gastrointestinal and genitourinary tract in 20–30% pregnant women. | It may be asymptomatic colonization but can cause **preterm labour, chorioamnionitis, premature rupture of membranes, bacteriuria, pyelonephritis** and **septicemia.** It can cause neonatal septicemia (early onset of disease in the first 7 days of life) or late onset disease with meningitis after 1 week to 3 months after birth. | ACOG (2016), CDC (2010), recommends universal screening of all pregnant women at 35–37 weeks for rectovaginal GBS followed by intrapartum antibiotic prophylaxis in GBS positive women. However, RCOG (2003) and Canadian Task Force on Preventive Health Care (2002) do not recommend GBS screening due to insufficient evidence. | Septicemia, preterm labour, premature rupture of membranes, foetal growth restriction, neonatal sepsis | IV antibiotic, Penicillin G, 5 million units initially at onset of labour or rupture of membranes followed by 2.5 million units every 4 hours until delivery is the **drug of first choice** in GBS positive women. Alternatively, ampicillin 2 g IV Initial dose followed by 1 g IV 4 hourly (or 2 g 6 hourly) until delivery. For penicillin allergic patients, cefazolin, 2 gm I/V initial dose then 1 gm I/V 8 hourly till delivery or clindamycin 900 mg I/V 8 hourly until delivery is given. Vancomycin 1 g IV every 12 hourly is given for penicillin allergic and resistant cases. |
| 3. Typhoid | *Salmonella typhi* | Typhoid fever | Blood culture, Widal test | Abortion, preterm labour, foetal death maternal death | **Fluoroquinolones (ciprofloxacin) and third generation cephalosporins are drugs of choice.** During pregnancy and in areas of quinolone resistance, third generation cephalosporin (ceftriaxone or cefuroxime) or azithromycin can be given. |
| 4. Bacillary dysentery (Shigellosis) | *Shigella species* | Exudative diarrhoea with bloody stools. Abdominal cramps, tenesmus, fever | Stool culture | Abortion, preterm delivery | **Fluoroquinolone (norfloxacin) is the drug of first choice.** Ceftriaxone, azithromycin can be given. |

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment |
|---|---|---|---|---|---|
| 5. Amebiasis | *Entamoeba histolytica* | Amebic dysentery, pain abdomen, malaise, anorexia, weight loss and fever if hepatic amebiasis. | Stool examination; Trophozoites in acute stage, cysts in chronic stage, stool culture, ultrasound liver (if hepatic involvement) | Malnutrition and anaemia can cause foetal growth restriction. | Metronidazole 400 mg thrice a day for 7 days in acute attack. For chronic amebiasis and recurrent amebiasis or chronic cyst passers, metronidazole 400 mg with diloxanide furoate 500 mg are given thrice a day × 7 days. Tinidazole 500 mg BD for 5 days or secnidazole 2 g single dose can also be given. |
| 6. Giardiasis | *Giardia lamblia* | Diarrhea, bulky foul smelling stools, abdominal discomfort, nausea and anorexia (hyperemesis), malabsorption | Cysts and trophozoites on stool examination | Malnutrition can cause foetal growth restriction. | Metronidazole (200 mg thrice a day for 7 days or tinidazole 500 mg BD × 5 days. |
| 7. Hookworm infestation | 1. *Ancylostoma duodenale*<br>2. *Necator americanus* | Very common in rural India due to defecation habits in open fields. Cough, breathlessness, abdominal discomfort, iron deficiency anaemia, hypoproteinemia, even cardiac failure | Stool examination shows cysts of hookworm. Leucocyte count shows eosinophilia. Hemoglobin is low. Peripheral blood film shows microcytic hypochromic anaemia. | Hypoproteinemia and anaemia can cause foetal growth restriction. | 1. Pyrantel pamoate 1 g stat (4 tab) or mebendazole 100 mg BD × 3 days or Albendazole 400 mg single dose (should be avoided in first trimester)<br>2. Treatment of iron deficiency anaemia with iron tablets (Ferrous sulphate) |
| 8. Threadworm infestation (Enterobiasis) | *Enterobius (Oxyuris) vermicularis* | Pruritis vulvae, pruritis ani, vaginitis, pelvic inflammatory disease | Stool examination, perianal swab for eggs | | Pyrantel pamoate 1 g single dose. Mebendazole or albendazole can also be given. |
| 9. Strongy-loidiasis | *Strongyloides stercoralis* | Malabsorption, protein losing enteropathy, iron deficiency anaemia | Stool examination | Foetal growth restriction (FGR) | Ivermectin 6 mg and Albendazole 400 mg single dose is the drug of first choice. Mebendazole (can also be given 100 mg twice a day for 3 days) as drug of first choice. |
| 10. Trichuriasis (Whipworm) | *Trichuria trichuria* | Iron deficiency anaemia, dysentery, gastroenteritis | Stool examination | Anaemia, FGR | Praziquantel 10 mg / kg single dose is the drug of first choice. Albendazole 400 mg single dose can be given. |

| Name of diseases | Causative agents | Symptoms and signs | Investigations | Effects | Treatment |
|---|---|---|---|---|---|
| 11. Tapeworm infestation | Tenia saginata (beef tapeworm), Tenia solium (pork tapeworm), Echinococcus, Diphylobothrium latum (fish tapeworm) | Cysticercosis with T. solium Abdominal discomfort, diarrhoea, constipation and folate deficiency with D. latum infection, Echinococcal cysts can develop in liver, lungs or pelvis. | Stool examination, ultrasound, CT scan | Echinococcal cyst can obstruct labour, if in pelvis | Niclosamide 2 g (4 tablets) single dose or Albendazole in high dose 800 mg daily for 3 days in life-threatening cases. |
| 12. Liver flukes | Schistosoma mansoni, Schistosoma hematobium, Schistosoma japonicum | Hematuria, hepatosplenomegaly, diarrhoea, bladder polyposis | Stool examination, urine examination | Abortion, foetal growth restriction due to malnutrition and proteinuria. Obstructed labour | Praziquantel 40–75 mg / kg single dose is the drug of first choice. Surgical treatment. |
| 13. Filariasis | Wuchereria bancrofti, Brugia malayi | Lymphedema in legs, vulva, perineum | Blood test for microfilariae | Obstructed labour | Diethyl carbamazine 100 mg tablet 5 mg / kg body weight in 3 divided doses for 3 weeks usually after delivery is the drug of first choice. Ivermectin 12 mg single dose with 400 mg albendazole 400 once a year for 5–6 years. Ivermectin 12 mg single dose oral is also the drug of first choice. Cesarean may be needed for obstructed labour. |
| 14. Scabies | Mite (Sarcoptes scabiei) | Epidermal skin lesions (tiny itchy ulcers) | 1. Clinical diagnosis 2. Microscopic examination from ulcer | | Premethrin 5% solution is the drug of first choice. Benzyl benzoate or gammabenzene hexachloride locally. |
| 15. Lice infestation (Pediculosis) | Pediculus capitis and pubis | Pruritis, tiny skin lesions | 1. Clinical diagnosis 2. Microscopic examination | | Ivermectin 12 mg oral single dose is the drug of first choice. Gamma benzene hexachloride locally, 2 applications at a week interval. |

## Key Points

1. All pregnant women should be screened for syphilis and treated to reduce the foetal morbidity and mortality. Screening for syphilis is done by non-treponemal tests like VDRL which may have false positive reaction with acute viral infection, malaria, TB, pregnancy, autoimmune diseases and cancers. Positive test needs to be confirmed by specific treponemal tests like FTA-ABS or microhemagglutination assay (MHA). Treatment is with penicillin, single dose for primary syphilis, 3 doses for secondary syphilis and for longer time for tertiary syphilis. Syphilis causes improving obstetric history with abortion, stillbirth, neonatal death and congenital syphilis. For pencillin allergic patients, desensitization followed by penicillin therapy or erythromycin or azithromycin is given.
2. Gonorrhea can cause miscarriages, premature labour, prelabor rupture of membranes, chorioamnionitis, foetal growth restriction and is treated with cephalosporins. Neonatal gonococcal ophthalmia neonatorum can occur during labour by passage of fetus through infected genital tract needing treatment with silver nitrate drops or erythromycin.
3. Chlamydial infection during pregnancy can cause preterm labour, premature rupture of membranes and low birth weight and neonatal conjunctivitis and pneumonia. Antibiotic therapy, single dose of 1 g of azithromycin or amoxicillin 500 mg 8 hourly for 7 days is the treatment of choice. Alternatively, erythromycin 500 mg 6 hourly for 7 days can be given. Treatment reduces the number of pregnant women with positive antibody by 90% and helps prevent pregnancy and neonatal complications **(level IA evidence)**. Mycoplasma and ureaplasma are also treated in the same way.
4. Listeria infection during pregnancy has been associated with miscarriages, preterm labour, stillbirths and meconium stained liquor in preterm deliveries and is treated with ampicillin and gentamicin therapy.
5. Most intestinal infections can be treated safely during pregnancy with standard medication like mebendazole, albendazole and metronidazole after 12 weeks of pregnancy.
6. Other sexually transmitted diseases can also cause preterm labour, premature rupture of membranes and should be treated with penicillin, amoxicillin or cephalosporins which are safe in pregnancy.
7. Skin lesions like warts, scabies and lice are treated by local solutions (like permethrin) in pregnancy.

## Suggested Reading

1. American College of Obstetricians and Gynecologists: Management of pregnant women with presumptive exposure to Listeria monocytogenes. Committee opinion No. 614; December 2014, Reaffirmed 2016.
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3. American College of Obstetricians and Gynecologists: Prevention of early-onset group B streptococcal disease in newborns. Committee opinion no. 485, April 2011, Reaffirmed 2016.
4. Centres for Disease Control and Prevention: Sexually Transmitted Diseases Treatment Guidelines 2015 : MMWR 2015 : 64(3) 2015.
5. Centre for Disease Control and Prevention: Prevention of Perinatal Group B Streptococcus Disease. Revised Guidelines from the CDC. MMWR 59 (10): 1; 2010.
6. Powrie R O et al ( eds). de Swiet's Medical Disorders in Obstetric Practice. Wiley Blackwell, Oxford, U. K. 5th edition, 2010.

## Examination Questions

A. Describe the various types of syphilis during pregnancy.
B. Enumerate the foetal effects of syphilis during pregnancy. How will you manage a case of syphilis during pregnancy? (DU)
C. What are the adverse effects of chlamydial infection during pregnancy? How will you diagnose and treat a case of chlamydial infection during pregnancy?
D. Write short notes on:
   1. Hookworm infestation during pregnancy
   2. Gonorrhea during pregnancy
   3. Chlamydial infection in pregnancy
   4. Amebiasis in pregnancy
   5. Congenital syphilis (DU)
   6. Obstetric significance of listeriasis during pregnancy

CHAPTER 37

# Abdominal Pain, Surgical Conditions, Gynecological Disorders and Neo-Plastic Conditions during Pregnancy

## (i) ABDOMINAL PAIN AND SURGICAL CONDITIONS DURING PREGNANCY

### I. Obstetric Causes

| Causes | Diagnosis | Management |
|---|---|---|
| First trimester | | |
| 1. Abortions | 1. History<br>2. Examination (speculum and vaginal)<br>3. Ultrasound confirms the diagnosis of the type of abortion. | See Chapter 13 for details.<br>1. Conservative treatment for threatened abortion<br>2. Evacuation for others |
| 2. Hydatidiform mole | 1. History of passage of blood and vesicles.<br>2. Examination. Lack of foetal parts<br>3. Ultrasound confirms the diagnosis. | 1. Evacuation after arranging blood<br>2. Follow up for hCG monitoring.<br>3. See Chapter 13 for details. |
| 3. Ectopic pregnancy | 1. History<br>2. Examination<br>3. Positive pregnancy test<br>4. Serial serum β hCG<br>5. Ultrasound confirms the diagnosis | 1. Laparoscopy or laparotomy<br>(i) Salpingostomy or<br>(ii) Salpingectomy. Medical management in selected cases; see Chapter 13 for details. |
| 4. Ovarian cyst complications (e.g. torsion) | 1. History of cyst<br>2. Examination<br>3. Ultrasound | 1. Laparotomy, ovarian cystectomy; see below for details. |
| 5. Retroverted gravid uterus | 1. History of urinary obstruction or incontinence<br>2. Examination<br>3. Ultrasound | 1. Conservative treatment<br>2. Catheterization<br>3. Bed rest<br>4. Antibiotics if UTI; see below for details. |
| Second Trimester | | |
| 1. Mid-trimester abortion | 1. Bleeding<br>2. Examination: Cervical dilatation and effacement | Shirodkar's or McDonald's suture for cervical incompetence |
| 2. Round ligament pain | 1. Radiating pain on the course of round ligaments. | 1. Conservative management bed rest, analgesics (Drotaverine alone or with mefenamic acid or Ibuprofen and paracetamol combination thrice a day for 5 days. |

| Causes | Diagnosis | Management |
|---|---|---|
| 3. Ectopic pregnancy (cornual or rudimentary horn pregnancy) | 1. Amenorrhea<br>2. Acute abdomen | 1. Surgical treatment. Removal of cornua or rudimentary horn, hysterectomy may rarely be required for intractable haemorrhage (parenteral methotrexate may be tried). See Chapter 13 for details. |
| 4. Red degeneration in fibroid | 1. Known case of fibroid<br>2. Severe abdominal pain<br>3. Vomiting<br>4. Tenderness at site of fibroid.<br>5. Ultrasound | 1. Conservative treatment: Drotaverine with mefenamic acid<br>2. Or aceclofenac Ibuprofen + paracetamol 1 TDS × 5 days<br>3. Or Inj. Pethidine 75 mg + Inj. Promethazine 25 mg – IM 4–6 hourly. See below for details. |
| **Third Trimester** | | |
| 1. Labour pains (most common) | 1. Uterine contractions can be felt.<br>2. CTG detects uterine contractions<br>3. Cervical changes | 1. Manage as labour, if at term (see Chapter 16 for details)<br>2. Tocolytics and betamethasone for preterm labour (see Chapter 34 for details) |
| 2. Fulminating pre-eclampsia | 1. Hypertension<br>2. Proteinuria<br>3. Edema<br>4. Abnormal serum biochemistry | Treat as per protocol of severe pre-eclampsia (see Chapter 32 for details) |
| 3. Acute hydramnios | 1. Known case of hydramnios or multiple pregnancy<br>2. Tense distension of abdomen<br>3. Ultrasound confirms diagnosis | 1. Conservative treatment. Analgesics Drotaverine with mefenamic acid or aceclogfenac or 1 buprofen paracetamol combination 8 hourly<br>2. Amniocentesis may be required for severe distension (500–1000 mL can be drained at one time) (see Chapter 24 for details) |
| 4. Abruptio placentae | 1. APH<br>2. Distress with shock<br>3. Tense tender abdomen<br>4. Ultrasound confirms retroplacental clot | 1. Induction of labour after resuscitation.<br>2. Cesarean delivery, if foetal distress (see Chapter 31 for details) |
| 5. Uterine rupture | 1. Previous history of uterine surgery<br>2. Obstructed labour, shock<br>3. APH<br>4. Hematuria | 1. Resuscitation<br>2. Urgent laparotomy with or without subtotal or total hysterectomy (see Chapter 26 for details) |
| 6. Uterine torsion | 1. Pain abdomen<br>2. Tenderness.<br>3. Ultrasound to rule out abruption | 1. Conservative treatment. Analgesics (Drotaverine with mefenamic acid or aceclofenac or Ibuprofen paracetamol combination). |

## II Non-Obstetric causes (They can occur any time in pregnancy)

| | | |
|---|---|---|
| 1. Acute appendicitis | 1. Pain abdomen at McBurney's point<br>2. Gastrointestinal upset<br>3. Fever<br>4. Leucocytosis<br>5. Diagnosis is difficult due to altered position of appendix.<br>6. Complications like suppuration, gangrene, perforation of appendix and peritonitis are more common in pregnancy.<br>7. Increased chances of abortion and preterm labour. There is foetal loss rate of about 20%. Higher maternal morbidity with delayed diagnosis.<br>8. Ultrasound, CT scan and MRI best modality may help in diagnosis of appendicular abscess. | 1. Early laparotomy by grid-iron (McBurney's incision) or longitudinal incision and appendicectomy,<br>2. Broad spectrum antibiotics (Coamoxyclav 1.2 g IV and Metronidazole 500 mg IV 8 hourly),<br>3. Laparoscopy and laparoscopic appendicectomy have been used successfully in the first half of pregnancy (up to 20 weeks) but should be done by experienced surgeon who can do it even in secondary pregnancy. |

| Causes | Diagnosis | Management |
|---|---|---|
| 2. Intestinal obstruction | 1. Abdominal pain<br>2. Nausea, vomiting<br>3. Constipation<br>4. Abdominal distension<br>5. Distended loops of bowel on X-ray | 1. Nasogastric aspiration<br>2. IV fluids<br>3. Broad spectrum antibiotics<br>4. Laparotomy, if condition does not improve |
| 3. Pancreatitis | 1. Central abdominal pain radiating to back<br>2. Shock<br>3. Raised serum amylase | 1. Nasogastric aspiration<br>2. IV fluids (electrolyte, glucose and calcium levels to be maintained); avoid surgery |
| 4. Peptic ulcer | 1. Past history of ulcer<br>2. Perforation and hematemesis can occur causing acute abdomen and shock.<br>3. X-ray abdomen shows gas under diaphragm, if perforation. | 1. Resuscitation<br>2. Proton pump inhibitors (Omeprazole or Pantoprazole)<br>3. Broad spectrum antibiotics<br>4. Laparotomy, if perforation |
| 5. Cholelithiasis and cholecystitis | 1. Pain in abdomen.<br>2. Ultrasound shows gall stones. | Usually conservative; Surgery may be required. |
| 6. Heart burn and hiatus hernia | 1. Retrosternal pain<br>2. Heart burn<br>3. Vomiting and hemetemesis | 1. Antacids (magnesium and aluminium hydroxide gel 15 mL 3–4 times a day)<br>2. Pantoprazole 40 mg daily<br>3. Metoclopramide, 10 mg 3–4 times daily |
| 7. Pyelonephritis | 1. Pain (loin)<br>2. Frequency and burning in micturition<br>3. Fever with rigor and chills<br>4. Urine culture is positive. | 1. IV antibiotics (coamoxyclav, 1.2 g 8 hourly or sensitive drug)<br>2. Resuscitation See Chapter 35 for details. |
| 8. Renal, ureteral or bladder calculi | 1. Past history of stones<br>2. Severe recurrent colicky abdominal pain radiating from loin to iliac fossa.<br>3. Plain X-ray abdomen and ultrasound help in diagnosis. | 1. Plenty of fluids<br>2. Antispasmodics like drotaverine with aceclofenac or with mefenemic acid thrice a day can be given or analgesics: Ibuprofen + paracetamol thrice a day |
| 9. Porphyria | 1. Abdominal pain<br>2. Psychiatric disturbances<br>3. Autonomic symptoms | 1. Analgesics (paracetamol 1 TDS)<br>2. Antiemetics (metoclopramide 10 mg 1 TDS) |
| 10. Malignant disease (carcinoma rectum, Hodgkin's lymphoma, retroperitoneal sarcoma) | 1. Pain in abdomen<br>2. Mass in abdomen<br>3. Ultrasound<br>4. CT scan<br>5. MRI | 1. Treatment as in non-pregnant state. |
| 11. Vascular accidents (mesenteric vein thrombosis, sickle-cell crisis and ruptured spleen) | 1. Past history of sickle-cell disease<br>2. Splenomegaly<br>3. Severe pain in 0abdomen<br>4. Distension<br>5. Shock | 1. Resuscitation<br>2. Urgent laparotomy and splenectomy in case of splenic rupture. |
| 12. Rectus sheath haematoma | 1. Severe pain and tender swelling at rectus sheath | 1. Conservative treatment<br>2. Analgesics and anti-inflammatory agents |

## General management for pain in abdomen

1. Resuscitation with IV fluids
   Ringer lactate: 1 liter (2 bottles)
   Dextrose saline: 1 liter (2 bottles)
   Blood, if required
2. Inj. Pethidine 75–100 mg IM 4–6 hourly
3. Inj. Promethazine 25 mg or Inj. Metoclopramide 10 mg IM 4–6 hourly
4. IV antibiotics
   i. Inj. Coamoxyclav 1.2 g IV 8 hourly
   ii. Inj. Metronidazole 500 mg IV 8 hourly

## For less severe pain

Oral analgesics can be given.

### If surgical treatment is required

To prevent abortion and premature labour, Inj. hydroxyprogesterone caproate 500 mg IM weekly with ritodrine or isoxsuprive 10 mg 4–6 hourly are given and continued for one week after surgery. The desired surgery is performed (e.g. appendicectomy) after proper counselling of the patient.

# (ii) GYNECOLOGICAL DISORDERS IN PREGNANCY

## FIBROIDS (LEIOMYOMAS) IN PREGNANCY

Fibroids are leiomyomas (benign smooth cell tumors) of uterus which may be classified as submucous (beneath the endometrial lining of the uterus), intramural (confined within the myometrium) or subserous (beneath the uterine serosa). The submucous and subserous may become pedunculated and can undergo torsion or necrosis.

**Incidence.** It is about 2% and is rising.

### Effects of fibroid on pregnancy

Most pregnancies associated with fibroids proceed unevent-fully. All pregnancy and labour complications are increased significantly if the placenta is attached on or near the myoma. Fibroids in the lower uterine segment and cervix are more dangerous as they may obstruct labour and may displace ureter making surgery technically difficult and hazardous. The following problems can occur in pregnancy, especially in submucous fibroids which have worst prognosis.

1. Abortion
2. Premature labour
3. Placental abruption
4. Pressure symptoms
5. Intraperitoneal haemorrhage
6. Torsion
7. Foetal growth restriction

#### Effects on Labour

1. Usually no effect
2. Abnormal presentation, especially breech
3. Obstructed labour
4. Dysfunctional labour
5. Increased incidence of cesarean delivery (6 fold)
6. Postpartum haemorrhage
7. Retained placenta
8. Uterine inversion

#### Effects on Puerperium

1. Increased morbidity
2. Puerperal infection
3. Secondary postpartum haemorrhage
4. Uterine inversion
5. Lochiometra (retention of lochia)
6. Pyometra (collection of pus in uterus)

### Effects of pregnancy on fibroid

Leiomyomas, especially large ones, tend to increase in size especially in the first half of pregnancy in almost 50% cases as a response to increased estrogen. In second trimester and especially in third trimester, the size of myomas may remain unchanged or decrease in size due to estrogen receptor down regulation.

Following changes can occur during pregnancy (due to hypertrophy of the muscle fibers with increased vascularity and edema)

1. Increase in size and change in shape of fibroid.
2. Impaction in pelvis
3. Torsion
4. Degeneration
5. Trauma
6. Infection
7. Incarceration
8. Expulsion of submucous fibroid

### Diagnosis

1. Past history of fibroid
2. Examination
3. Ultrasound is the modality of choice as it can give information about size, site and number of myomas. Use of colour doppler may be useful.
4. MRI can be used.

### Treatment

Classical teaching is not to do any surgical treatment and watchful expectancy is all that is required.

#### Antenatal

1. Routine antenatal follow-up, investigations and treatment are offered unless there are complications.
2. For pressure symptoms, change of posture, bed rest, digital lifting of fibroid from pelvis is tried through vagina or rectum in knee-elbow position. Dietary care and avoidance of constipation are advised.
3. For red degeneration, conservative treatment is offered (see below for details).
4. As a general principle, surgery is avoided and myomectomy is contraindicated due to excessive bleeding. Laparotomy is performed extremely rarely for impaction of fibroid where digital lifting and conservative treatment fail. If the pedunculated subserous fibroid has undergone torsion then the pedicle is clamped, ligated and myoma is excised. Hysterectomy may rarely be indicated where the patient has completed family and there are complications like intractable bleeding making myomectomy a hazardous operation.

#### Intrapartum Management

In general, vaginal delivery is preferred in myomas.

1. Keep vigilance for intrapartum complications.
2. Oxytocic agents, blood and all facilities for operative intervention should be ready.
3. Spontaneous labour is preferred.
4. Induction of labour may be done when indicated (e.g. pre-eclampsia, postdatism).
5. The majority of the myomas, including the anterior cervical myomas, get lifted out of the pelvis either near term or in labour due to uterine contractions and retraction.

6. Cesarean delivery is indicated in the following circumstances.
   (a) If a large fibroid in cervical area is obstructing labour.
   (b) Malpresentations
   (c) Uterine inertia
   (d) Other obstetric indications like oblique lie, transverse lie or other malpresentations.

   Rarely classical cesarean section may have to be done if lower segment is totally obliterated and distorted by multiple fibroids. Traditional teaching is that during cesarean delivery myomectomy should not be attempted and incision should be lower segment transverse away from fibroid to reduce risk of sepsis. However, in current practice many studies have proven the safety and efficacy of concomitant myomectomy with cesarean delivery with rare need of hysterectomy. Hence myomectomy may be considered in suitably selected cases (not all cases) with availability of surgical expertise, adequate blood transfusion especially for large single myoma or myoma in lower segment. However, cesarean myomectomy is still matter of debate and should be done in only selected cases after proper counselling of patient by a senior and experienced obstetrician with expertise in the area with adequate blood and rare need and risk of hysterectomy.
7. There is risk of postpartum haemorrhage and retained placenta.

**Postpartum Period**

Postpartum period is usually uneventful. The following complications may occur.

1. Subinvolution
2. Torsion of subserous myoma requires laparotomy and excision.
3. Infection of the myoma, especially if placenta is situated over it or in septic abortion.
4. Expelled submucous fibroid through cervix presenting with sepsis or secondary postpartum haemorrhage needs shelling out or twisting of the tumor under the cover of antibiotics. Beware of uterine inversion.
5. Secondary haemorrhage
6. Puerperal sepsis.

## Degenerations in fibroid

There can be cystic change or more commonly **red degeneration** (necrobiosis) or carneous degeneration which occurs mainly in the second trimester (14–20 weeks) or during puerperium (most common time) in large fibroids when the growth of the uterus and the fibroid is most active. In this condition ischemic or hemorrhagic infarction occurs due to venous obstruction as an aseptic process **(necrobiosis)**. The fibroid on inspection is reddish in colour being heavy stained with blood pigment giving it a characteristic raw beef appearance on cut section along with a fishy smell. On microscopic examination, there is necrosis and vascular thrombosis in myometrial cells.

**Clinical Features**

1. Severe pain over the fibroid
2. Malaise and fever
3. Constipation
4. Vomiting
5. Rapid pulse
6. Tenderness at the site of myoma
7. Peritoneal rub may be there.
8. Hemogram demonstrates leucocytosis.

**Differential Diagnosis**

All causes of acute abdomen are:

1. Acute appendicitis
2. Acute cholecystitis
3. Twisted ovarian cyst
4. Placental abruption
5. Pyelonephritis
6. Ureteral stone

**Treatment**

Conservative treatment is almost always successful. It is as follows:

1. Bed rest
2. Analgesics. Tablet ibuprofen 400 mg with paracetamol 500 mg thrice a day for 7 days
3. Sedation with Inj. pethidine 75–100 mg with Inj. promethazine 25 mg IM 4–6 hourly. Symptoms usually settle down by second week. *If on laparotomy, performed for acute abdomen, fibroid with red degeneration is detected, no surgical procedure should be performed on the gravid uterus and abdomen should be closed and conservative treatment is continued.* Myomectomy and hysterectomy should never be attempted.

   Cystic change occurs due to hyaline degeneration and is managed conservatively.

## Uterine Artery Embolization and Pregnancies

American College of Obstetricians and Gynecologists (2004) considers uterine artery embolization for myoma a relative contraindication in women wishing to retain fertility. The studies have shown increased complications like miscarriage, abnormal placentation, preterm delivery and postpartum haemorrhage in pregnancies after uterine artery embolization for myomas.

# OVARIAN TUMORS IN PREGNANCY

The ovarian masses could be functional cysts or neoplastic. **The most common neoplasm is benign cystic teratoma** (dermoid cyst) followed by epithelial cystadenoma (mucinous and serous) and rarely other germ cell tumors. Ovarian carcinoma can rarely occur. **Incidence** varies from 1 in 200 to 1 in 2000 pregnancies. 0.2–2% pregnancies are complicated by adnexal mass. Upto 1–3% are malignant.

## Effects of Ovarian Tumor on Pregnancy

Higher rates of:

1. Mechanical distress and respiratory embarassment, if large tumor
2. Retention of urine and other pressure symptoms
3. Malpresentations (breech, transverse lie, etc.)
4. Unengaged head at term
5. Premature labour in presence of complications.

### During Labour

Obstructed labour can occur in an impacted tumor.

## Effects of Pregnancy on Ovarian Tumor

There is increased risk of all complications except malignancy.

1. **Torsion** can occur in 10–12% cases in the first trimester and 20–30% in puerperium. It is due to lax abdominal musculature and sudden change in the anatomical relations of the pelvic organs.
2. **Rupture** of a cyst can rarely occur due to mechanical compression between foetal head and maternal pelvis and may be of no consequence. But rupture of a dermoid or mucinous cyst can cause severe granulomatous peritonitis.
3. **Necrosis** of the tumor can occur in puerperium due to trauma during vaginal delivery.
4. **Haemorrhage** can occur in the substance of the tumor due to trauma and or torsion.
5. **Incarceration.** A tumor can get incarcerated during labour protruding through the vaginal vault and can dissect the rectovaginal septum causing obstructed labour. Tumors can get necrosed and infected.
6. **Suppuration** can rarely occur due to infection of traumatized tissue by organisms from the birth canal during labour or early puerperium.

## Diagnosis

### 1. Clinical Features

(a) Asymptomatic
(b) Abdominal pain
(c) Vomiting
(d) Abdominal distension
(e) Tachycardia
(f) Guarding
(g) Rigidity
(h) Tender palpable mass. Abdominal examination: Ovarian cyst is usually felt separately from pregnant uterus as a separate cystic mass (difficult in later pregnancy). In later months, diagnosis may be difficult. The examination is performed in Trendelenberg's head down position which pushes the ovarian cyst up while uterus remains down with a groove between the two (uterus and ovarian cyst). It is called **Hingorani sign** (Prof. V Hingorani, AIIMS, Delhi)
(i) On bimanual examination. Palpable mass separate from the uterus in lateral or posterior fornix.

### 2. Investigations

(a) **Ultrasound.** Transvaginal or abdominal ultrasound detects the site of origin, nature of the tumor (cystic or solid), any septa or excrescences. It helps in formulating plan of treatment. Colour Doppler images the tumor vascularity aiding the diagnosis of torsion and malignancy.
(b) **Magnetic resonance imaging** is sensitive and specific for diagnosis of dermoids and is an useful adjunct to ultrasound.
(c) **Tumor markers** like CA-125 (for epithelial tumor), hCG, AFP can help in germ cell tumors.
CA-125 may be elevated in first trimester but levels normalize in second and third trimester.

## Differential Diagnosis

1. Uterine fibroid
2. Pelvic hematocoele
3. Retroverted gravid uterus
4. Multiple pregnancy
5. Polyhydramnios
6. Non-gravid horn of bicornuate uterus
7. Rectus abdominis haematoma

## Treatment

An ovarian tumor if more than 10 cm in diameter or between 6–10 cm with septa or solid areas should be removed whenever diagnosed in pregnancy irrespective of the nature, position and symptoms of the tumor, conserving the pregnancy and giving parenteral progesterone and ritodrine or isoxsuprine to keep the uterus quiescent (see above for details). Cysts 5 cm or less can be observed as most undergo spontaneous resolution. If asymptomatic and detected in first trimester, ultrasound examination should be done to look for features of malignancy like thick septa, solid areas, papillary excrescences and size of the cyst. Tumors without any criteria of malignancy can be conserved till the end of the first trimester under careful follow-up. This gives functional cysts, like corpus luteum cysts, time to regress. If still present, elective laparotomy is planned between 14–18 weeks of pregnancy. Similarly, small tumors without features of malignancy detected in the last few weeks of pregnancy can be left till fetus is viable when elective cesarean delivery and cystectomy or ovariotomy are performed in the same sitting.

**Principles of surgery.**

1. Midline insertion
2. No uterine manipulam
3. Peritoneal wash
4. Frozen section to be sent

### Role of Laparoscopy

Laparoscopy, especially gasless laparoscopy, has been used recently to evaluate and remove intermediate-sized cysts with low malignant potential with results comparable to laparotomy. There is higher risk of tumor rupture intraoperatively.

## GENITAL PROLAPSE AND PREGNANCY

Incidence of varying grades of prolapse is about 1 : 250–300 pregnancies. Slight degree displacement of cervix (first degree uterine prolapse) with mild cystocele and rectocele are not uncommon. Pregnancy is uncommon with second degree prolapse and exceedingly rare in third degree prolapse (Fig. 37.1).



**Fig. 37.1:** Actual photograph of third degree uterovaginal prolapse in third trimester of pregnancy

### Effects on Prolapse

1. Hypertrophy and edema of the cervix
2. Aggravation of the prolapse and stress incontinence
3. Excessive vaginal discharge
4. Blood stained vaginal discharge, if decubitus ulceration occurs
5. Rarely symptoms of incarceration may develop from 10–14 weeks of gestation if uterus persists in its prolapsed position. Pessary treatment may prevent this complicaiton.
6. Distension of bladder and urinary retention

### Effects of Prolapse on Pregnancy

1. There may be no effect in mild prolapse.
2. Abortion rate is increased, especially with second and third degree prolapse.
3. Discomfort
4. Intrauterine infection
5. Premature rupture of membranes
6. Premature labour
7. Intrapartum
   (a) Cervical dystocia due to thick and edematous cervix
   (b) Prolonged labour due to failure of cervix to dilate
   (c) Increased surgical intervention rate

#### Puerperium

1. Puerperal sepsis
2. Subinvolution
3. Urinary retention
4. Urinary tract infection

### Clinical Features

1. Excessive vaginal discharge
2. Backache and dragging sensation
3. Stress urinary incontinence
4. Constipation
5. Retention of urine and frequency of micturition
6. Mechanical discomfort due to friction of prolapse with clothes

#### Signs

1. On examination, there is cystocele, rectocele or enterocele and cervical descent.
2. Edematous and hypertrophied cervix which may show decubilus ulcer.
3. Stress incontinence may be demonstrable.
4. Signs of pregnancy

### Management

If the prolapse is only first degree with slight rectocele and cystocele, no special treatment is required and patients can be reassured that prolapse which is worse in the first trimester tends to be less prominent in the second trimester.

1. If second degree or third degree prolapse, it should be replaced and held in place by using a ring pessary which is kept until 20 weeks when uterus grows enough to rise above the pelvis taking cervix alongwith it.
2. If there is edema and congestion of the cervix and prolapsed mass, acriflavin and glycerine or saline pack can be used to relieve the edema (by their hygroscopic action) and mass can be replaced in the vagina. If prolapse cannot be reduced, patients should be kept admitted in hospital. If ring pessary is not sufficient, rarely vaginal introitus can be closed to be reopened at 38 weeks.

#### During Labour

1. Patients should preferably be in bed to avoid premature rupture of membranes.
2. Acriflavin or glycerine packing of vagina helps to relieve edema of cervix.
3. Prophylactic antibiotic with amoxicillin 500 mg 8 hourly is helpful.
4. During decent of the head, cervix should be pushed up during contraction.
5. Ventouse may be applied.
6. If head is low down but cervix is thin and undilated, Duhrssen's incisions at 2 and 10 o'clock positions may be required.
7. Cesarean delivery is preferred for prolonged labour and high foetal head with cervical dystocia.
8. For women who had Fothergill's operation (Manchester repair) for prolapse in the past, cesarean section is usually required for non-dilation of cervix.

#### Puerperium

1. Acriflavin or glycerine pack is helpful if prolapse is persisting.

2. Prophylactic antibiotics are continued.
3. Patients should be in bed.

## RETROVERTED GRAVID UTERUS

Retroversion is seen in 6–19% (average 10%) women in the first trimester. Retroversion of the uterus usually undergoes spontaneous correction between 10th and 13th week, but rarely incarceration of the gravid uterus can occur.

### Etiology

1. It occurs in women who had retroverted uterus before pregnancy.
2. Usually deep sacral concavity with an overhanging promontory prevents upward movement of the uterus.
3. Old pelvic adhesions
4. Pelvic tumor
5. Unknown cause

### Clinical Features and Presentation

1. Spontaneous rectification usually occurs soon after the 12th week with no further problems.
2. If incarceration occurs (12–16 weeks), the following symptoms and signs are there.
   (a) Initially, there is frequency of micturition followed by retention of urine which is acute and patients are in agonizing pain. There can be overflow incontinence.
   (b) Pelvic discomfort
   (c) Low sacral pain
   (d) Constipation

### Signs

1. Patient looks distressed due to severe pain.
2. Lower abdominal distension and tenderness.
3. Fluctuant and tender swelling in the suprapubic region (distended bladder).
4. Bimanual examination confirms the diagnosis by feeling soft mass (uterus) in the posterior fornix and inability to reach the cervix.
   Rectal examination may also be helpful.
   After emptying the bladder, retroverted uterus is felt.

### Differential Diagnosis

1. Pregnancy with impacted fibroid
2. Impacted ovarian tumor
3. Acute hydramnios
4. Pelvic hematocoele

### Pathogenesis

It may happen due to enlargement of a retroverted uterus with subsequent entrapment of fundus below sacral promontary. Cervix is pointed upwards and forwards. Uterus continues to grow. Urethra and bladder are mainly affected. Urethra is elongated along with bladder base which together with edema of bladder neck and neuromuscular imbalance causes urinary retention. Bladder gets distended and infected followed by cystitis and pyelonephritis and even perinephric abscess.

### Effects on Pregnancy

1. Miscarriage
2. Malpresentation
3. Premature labour
4. Unengaged foetal head
5. Uterine rupture in labour due to obstruction

### Treatment

1. Catheterization (Foley's catheter) to keep bladder empty.
2. Bed rest in prone position.
3. To treat urinary infection after culture sensitivity. Capsule amoxicillin 500 mg 8 hourly for 7 days.
4. Usually uterus corrects itself.
5. Continuous drainage of bladder with intermittent knee chest position and sleeping in prone position should be tried as it causes spontaneous correction of retroversion in majority of cases within 48 hours.
6. If spontaneous correction does not occur, manual correction is tried by putting patient in knee-chest position. A pessary (Hodge Smith) is left until 18–20 weeks.
7. Rarely laparotomy may be required to correct retroversion if adhesions are present.
8. Cesarean delivery may be needed if retroversion is diagnosed late in third trimester or when manipulations are unable to correct retroversion. A midline incision may be better. At the time of giving incision on uterus, position should be corrected to avoid incision on vagina or bladder.

# (iii) MALIGNANT DISEASE AND PREGNANCY

Cancer occurring in a pregnant patient is a rare event with incidence being 0.1% to 0.7%. (About 191.5/100.000 maternities) No evidence exists of an increased incidence of cancer in pregnant patients compared with the non-pregnant population. Nor there is any evidence that pregnancy adversely affects either the prognosis or the biology of maternal cancer. The diagnosis of cancer in pregnancy poses a range of problems and management needs to be individualized and is by multi-disciplinary approach involving obstetrician, oncologist, neonatologist, the patient and her family. As a general rule, patient's life is given priority and treatment should not be withheld or unnecessarily postponed in pregnancy. The various malignancies occurring during reproductive age group are cervical cancer, central nervous system tumors, Hodgkin's disease and leukemia. Between the age of 35 and 50 years breast, lung, colon, rectum, uterine and ovarian malignancies can occur.

There is relative increase in incidence due to delayed marriage and child bearing.

## Effect of Pregnancy on Malignancies

There is no reported increase in the incidence of malignancy in pregnancy.

## Effect of Malignancy on Pregnancy

Effects of malignancy are usually secondary to general debility and weakness. Disorders like leukemia can cause thrombocytopenia, anaemia and leucopenia resulting in increased incidence of infections and bleeding. Rarely melanoma, breast cancer, leukemia and lymphoma can metastasize to placenta and the fetus.

## Chemotherapy in pregnancy

The impact of cytotoxic agents on the fetus depends on the time of exposure and is greatest during embryonic period (first trimester). In early pregnancy, abortion can result or congenital malformations may occur. During second and third trimesters, foetal growth and development are impaired. There can also be microcephaly, mental retardation and impaired learning. Antimetabolites especially folate antagonist aminopterin and methotrexate, 5-fluorouracil, 6 mercaptopurine are the most potent cancer drug teratogens. Vinca alkaloids and antibiotics (doxorubicin) are relatively safer in pregnancy.

## Surgery for Cancer

Surgery for cancer can be performed during pregnancy safely especially in second and third trimesters.

## Radiotherapy

Ionizing radiation can cause malformations, abortions or leukemia. Foetal structures like central nervous system, mesenchyme and eyes are most sensitive. Avoidance of abdominal radiation in the first 16 weeks of pregnancy is advised.

# CERVICAL NEOPLASIA IN PREGNANCY

Incidence of abnormal smears (cervical intraepithelial neoplasia) is 10–15 per 1000 pregnancies while incidence of carcinoma in situ and invasive carcinoma of cervix is 1 : 750 and 1 : 2500 pregnancies respectively.

## Abnormal Cervical Cytology

According to 2001 Census Guidelines for the Management of Women with Cervical Cytological Abnormalities, evaluation of a smear is the same as in non-pregnant state. Women with atypical squamous cells of undetermined significance (ASCUS) and low grade squamous intraepithelial lesions (LSIL) can be followed up. Endocervical curettage (ECC) not to be performed.

All women should have cervical cytology in pregnancy if they had not had it before or if there is any irregular bleeding or unhealthy looking cervix. If there is ASCUS or LSIL in two subsequent smears or high-grade squamous intraepithelial lesions (HSIL) in one smear, the patient should be subjected to colposcopy and colposcopic directed biopsy (which may bleed excessively and may need a stitch for hemostasis). Conization is avoided in pregnancy as it tends to be incomplete, associated with excessive bleeding and there is increased risk of abortion and preterm delivery. Loop electrosurgical excision procedure (LEEP) is also avoided in pregnancy and is reserved to exclude invasive cancer.

As per ASCCP Guideline 2006, in pregnant women with LSIL, colposcopy can be deferred until at least 6 weeks postpartum. For non-adolescent women, preferred approach is to perform colposcopy. If no CIN 2 or 3, postpartum follow up, if CIN 2–3 then biopsy should be done like for HSIL.

Patients with HSIL and carcinoma in situ are evaluated with cytology, colposcopy and biopsy to rule out invasive disease. If there is no invasive disease, pregnancy is allowed till term and they can deliver vaginally and re-evaluated postpartum for definitive treatment in the form of conization or total hysterectomy. Microinvasive disease can also be managed similarly.

## Invasive carcinoma cervix

The incidence of invasive carcinoma cervix is about 1 : 2000 pregnancies.

### Effects of Pregnancy on Tumor

There is usually no effect of pregnancy an tumor although some authorities think that the tumor grows more rapidly during pregnancy. Extent of tumor can be underestimated due to cervical, paracervical and parametral softening of pregnancy.

### Effects of Tumor on Pregnancy

There are increased chances of the following complications

1. Miscarriage

2. Preterm labour (often iatrogenic)
3. Haemorrhage
4. Sepsis
5. Cervical dystocia

### Diagnosis

Invasive carcinoma cervix is suspected from abnormal cytology or clinical symptoms and confirmed by histological examination of punch biopsy of cervix. Diagnosis of carcinoma cervix may be missed by attributing vaginal bleeding to threatened abortion and placenta previa or accidental haemorrhage. One should always keep the possibility of carcinoma of cervix in mind as by waiting, an operable cancer may become inoperable.

### Treatment

*During first 20 weeks of pregnancy*

Curative treatment should be carried out to avoid any unnecessary risk to mothers.

Stages I and II A are best managed by radical hysterectomy (with fetus in utero up to 20 weeks) with bilateral pelvic lymphadenectomy.

For advanced stages, radiation therapy is used. External irradiation is started in early pregnancy. Spontaneous abortion usually occurs or pregnancy can be terminated surgically followed by intracavitary irradiation. For 14–20 weeks pregnancy, hysterectomy is first performed followed by external and intracavitary irradiation a week later.

### During Second Half of Pregnancy

A short delay may be allowed to make the fetus viable and steroids are given for foetal lung maturity. At about 34 weeks, cesarean section (usually classical cesarean section) followed by immediate or later radical hysterectomy (Wertheim's hysterectomy) and lymph node dissection for early stage disease or more aggressive radiotherapy for late stage disease should be instituted.

### Labour

Elective cesarean section (classical) is the preferred mode of delivery as vaginal delivery can cause cervical dystocia, injuries and intractable bleeding. Successful pregnancy outcome using classical cesarean has been reported following fertility preserving radical trachelectomy in stages 1B1 and 1B2 cervical cancer.

### Prognosis

The overall prognosis for all stages of cervical cancer during pregnancy is similar to that of the non-pregnant state.

## BREAST CANCER

Pregnancy associated breast cancer (PABC) is defined as cancer that is diagnosed during pregnancy or within 1 year postpartum. Breast cancer accounts for one-fourth of all cancers diagnosed in pregnancy with approximate incidence of 1 in 3000 pregnancies. (0.7% of all breast cancer patients)

### Effect on Pregnancy

Breast cancer usually has no effect on pregnancy. Chemotherapy for breast cancer in the second and third trimesters does not cause any obvious foetal harm. Foetal growth restriction can occur. Placental metastatis is rare.

### Effect of Pregnancy on Breast Cancer

Stage for stage, the outcome is no different from breast cancers diagnosed outside pregnancy. Pregnancy does not have deleterious effect on breast cancer and termination of pregnancy is not recommended.

### Diagnosis

It is often delayed in pregnancy and lactation because of mammary growth. Any lump that persists for more than 2–4 weeks needs work-up. Any suspicious breast lump must have mammography, fine needle aspiration cytology (FNAC) and excisional biopsy. Ultrasound and MRI can be used and are safe. Half of them are estrogen and progestogen receptor (ER/PR) negative and one third are HER 2/ neu receptor positive.

### Treatment

It is like non-pregnant state. Termination of pregnancy does not improve prognosis. Team of obstetrician, surgeon and oncologist should treat them. A modified radical mastectomy which does not require postoperative chemotherapy is normally advised in pregnancy in absence of metastatic disease. Radiation is avoided in pregnancy. Lumpectomy followed by chest wall radiotherapy with appropriate abdominal shielding can be performed though less preferred due to foetal exposure to radiation. Chemotherapy especially for node positive disease can be safely given in second and third trimesters completing the course three weeks prior to delivery to limit the effects of foetal bonemarrow suppression. Tamoxifen is avoided in pregnancy as its safety in pregnancy is not proven. Herceptin (Trastuzumab) is avoided in pregnancy as it can cause miscarriage, foetal renal failure, oligohydramnios and preterm birth. Though stage for stage, pronsosis is same as in non-pregnant stage, breast cancer being found in more advanced stage with overall poorer prognosis in pregnancy.

Other malignancies like lymphomas, leukemias, colorectal cancer are treated as in non-pregnant state.

## Key Points

1. Abdominal pain in pregnancy is a common clinical presentation. Diagnosis is often difficult and delayed and may be masked by pregnancy. Pregnancy and non-pregnancy causes like surgical, medical (gynecological) and gastrointestinal must be considered in differential diagnosis.
2. Urinary tract infections are one of the most common causes of abdominal pain. Preterm labour should always be considered during pregnancy. Similarly, abruptio placentae should be ruled out before considering surgical causes of pain abdomen.
3. Management of most surgical conditions is the same as in non-pregnant state and should not be delayed due to pregnancy. It is difficult to diagnose acute appendicitis in pregnancy and delay in diagnosis causes increased maternal morbidity. There is increased risk of abortion and preterm labour with appendicitis during pregnancy. In appendicitis, delay in surgical treatment can be dangerous and fatal. Hence, early laparotomy and appendicectomy are indicated in such cases. Before surgery, progestogens and uterine relaxants are given parenterally to prevent preterm labour. Diagnosis and treatment can reduce morbidity.
4. Coexistence of uterine fibroids with pregnancy is common. In majority of cases, there may be no effect but they may result in increased incidence of abortion, preterm labour and postpartum haemorrhage. Various types of degenerations are common in pregnancy. Red degeneration can occur during pregnancy and puerperium (more common) with abdominal pain and local tenderness. It is always managed conservatively by analgesics.
5. Most women with fibroids can have vaginal delivery. Cesarean delivery is indicated if fibroids are in lower segment or cervix and are obstructing labour. Myomectomy should not be attempted at the time of cesarean delivery due to extensive haemorrhage. Rarely for a subserous fibroid with a long pedicle, myomectomy may be performed.
6. Ovarian cysts detected during pregnancy are surgically removed if their size is more than 10 cm or more than 6 cm with septa or solid area or features of malignancy. Benign cysts (e.g. dermoid cysts) are best removed between 16 and 20 weeks of pregnancy.
7. Diagnosis and management of cervical intraepithelial neoplasia and carcinoma cervix is the same as in non-pregnant state. Conization is avoided during pregnancy being hazardous but colposcopy and directed biopsy is usually performed. For carcinoma cervix in the first half of pregnancy, Wertheim's hysterectomy for early stages and radiotherapy for late stages are performed. After 30 weeks, cesarean section along with Wertheim's hysterectomy with lymph node dissection can be performed at the same time.
8. Pregnancy does not alter the course of cancer but may cause a delay in diagnosis. Most investigations can be safely used to diagnose cancer. Chemotherapy and radiotherapy in the first trimester can cause abortion or increased chances of foetal congenital malformations.

## Suggested Reading

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CHAPTER 38

# Pregnancy in Red Cell Alloimmunization (Rh-Negative Mother)

## INTRODUCTION

### History

Rhesus factor (first found in Rhesus monkey) was discovered by Landsteiner and Weiner in 1940. Levine et al. in 1941 confirmed that erythroblastosis fetalis was due to maternal Rh isoimmunization. Effective maternal prophylaxis with anti D was developed by Finn et al. (1961) from UK and Freda et al. (1963) from USA. Liley (1961) from New Zealand developed charts (Liley's graphs) for severity of foetal isoimmunization. He also started inutero intraperitoneal blood transfusion. The incidence of Rh isoimmunization has fallen dramatically due to the use of anti D prophylaxis.

In transfusion medicine Rhesus is no longer used as it was a misconception that red cells from Rhesus monkeys expressed human blood group antigen. Current name is CDE blood group incompatibility.

### Incidence

The frequency of Rhesus antigen varies among different countries and ethnic groups. It is 16% in North American and European Caucasians, 8% in African Americans, 4–6% in Africans, 0.3% in Chinese, 0.5% in Japanese, 5% in Indians (8–10% in North India, 3–5% in South India). About 40% of Rh-positive individuals are homozygous at the D locus (DD) while the remainder are heterozygous (Dd).

### Genotype

The fetus receives its genetic components from both parents and thus the blood group of the fetus may differ from the mother. The red cell membranes have various surface antigens like the ABO, Rhesus and Kell groups and all of them are capable of starting an immune response in mothers.

The Rhesus blood group antigens comprise five antigens C, c, D, E and e. As d has not been identified so d indicates absence of the 'D' antigen. The CDE genes of Rhesus factor are located on the short arm of chromosome 1. The most immunogenic of these is the 'D' antigen. Its presence or absence denotes a person as Rh-positive or Rh-negative. In a Rh-negative mother and Rh-positive father, the probability of a Rh-positive fetus will depend upon whether the father is heterozygous or homozygous. A man carrying D on both sets is called homozygous (DD) seen in 40% cases, while a man with D in only one set is called heterozygous (Dd) seen in 60% cases. If the father is homozygous, all the offspring will be Rh-positive (Dd). If the father is heterozygous, half the offsprings will be Rh-positive (Dd) and half will be Rh-negative (dd) (Fig. 38.1).

## PATHOGENESIS OF RH ISOIMMUNIZATION

Isoimmunization is a process in which alloimmunizing antibodies are produced in a person against an antigen from another person of the same species. When the mother is Rh-negative and the father Rh-positive, the fetus can be Rh-positive and this can cause isoimmunization. Unlike ABO groups where anti-A and anti-B are already present in serum, there are no naturally occurring antibodies against Rh groups. Hence, sensitization must take place for antibodies to be formed. Antibodies once formed remain throughout life.

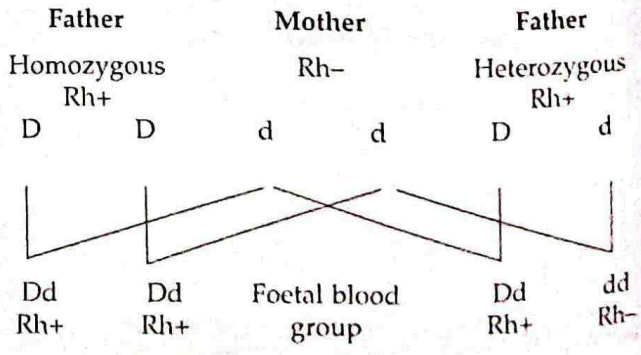



**Fig. 38.1:** Inheritance of Rh group

## Antibodies associated with Hemolytic disease of newborn

1. Anti-D, c, E, e, C, K and Kell antibodies
2. Anti-D, anti-e and anti-K antibodies are most often associated with moderate to severe hemolytic disease of newborn (HDN).

## Mechanism

1. Normally foetal red cells carrying the Rh antigen do not mix with maternal blood. However, some circulating foetal RBCs can enter maternal circulation stimulating an immune response due to **IgM or complete antibody which being a bigger molecule does not cross the placenta and does not affect the fetus. IgG (albumin antibody) is also produced in about 6 months after fetomaternal bleed. Being a tiny molecule IgG is actively transported across the placenta binding to the foetal red cell antigens causing their sequestration and hemolysis if the fetus is Rh-positive.** There is no effect on the Rh-negative fetus due to absence of Rh antigen on red cell surface. Transfer is rare in the first trimester as IgG cannot cross the placenta then. The first pregnancy is unlikely to be affected. If the fetomaternal bleed is less than 0.1 mL, sensitization of the mother occurs but the antibody may not be detected at that time. On a subsequent exposure to the antigen, her previously primed B cells quickly produce IgG which can be detected in the immunized mother in the next pregnancy. However, 1% will have primary immunization before delivery and may show the presence of antibodies during the first Rh-negative pregnancy.
2. Immunization can also occur if a Rh-negative woman has been transfused with Rh-positive blood.
3. Infection with needles contaminated by RhD positive blood
4. D-mismatched allogenic hematopoietic stem cell transplantation

## Factors Deciding the Maternal Response (why all rh-negative women do not get isoimmunized)

Although transplacental fetomaternal haemorrhage is fairly common, the occurrence of Rh-isoimmunization is rare. The degree of isoimmunization depends upon the following factors.

1. *Size of innoculum (volume) of fetomaternal haemorrhage.* As RhD antigen is very potent, even a volume of 0.1 mL (the critical sensitizing volume) of RhD+ erythrocytes in RhD– individual stimulates the production of antibodies.
2. *Magnitude of mother's immune response.* About one-third women are non-responders (variable antigenicity).
3. Strength of the antigenic stimulus also affects the response.
4. *Concurrent ABO incompatibility between mother and fetus.* Risk of sensitization is affected by the ABO blood group of the fetus with lesser risk if it is incompatible with mother's ABO blood group. When ABO incompatible foetal red cells enter the mother's bloodstream, they quickly combine with the naturally occurring anti-A and anti-B agglutinins and are neutralized and cleared by them.
5. Risk of sensitization is highest in first pregnancy and decreases with each subsequent pregnancy.
6. Du positive individual.

**Risk of alloimmunization and after anti-D**

**After Ist Rh-positive pregnancy**

| | |
|---|---|
| ABO compatible (80%) | = 16% |
| ABO incompatible (20%) | = 1.5–2% |
| Postpartum Anti D only | = 2% |
| Ante and postpartum Anti D | = 0.1% |

## Incidence of isoimmunization

A D-negative woman who has delivered a D-positive, ABO-compatible infant has a likelihood of isoimmunization of 16% (2% in ABO incompatible fetus and 2–5% after an abortion); 2% will be immunized by the time of delivery, 7% will have anti-D antibody by 6 months postpartum, and the remaining 7% will be **'sensibilized'**. In sensibilization, anti-D antibodies are produced at a very low level and are not detected during or after the current pregnancy but will be identified early in the next pregnancy.

## Factors predisposing to feto-maternal haemorrhage and sensitization

Sensitization can occur anytime during pregnancy but is more common in the third trimester and during parturition. Although it can occur in the absence of sensitizing events, it is more common in the presence of following conditions.

1. Miscarriage especially late miscarriage
2. Termination of pregnancy
3. Ectopic pregnancy
4. Partial molar pregnancy
5. Medical intervention
   - (*i*) Chorionic villus sampling
   - (*ii*) Amniocentesis
   - (*iii*) Cordocentesis
   - (*iv*) External cephalic version
6. Maternal abdominal trauma
7. Antepartum haemorrhage (more common in abruptio placentae)
8. Manual removal of placenta
9. Cesarean delivery
10. Threatened preterm/Preterm labour
11. Idiopathic

# FOETAL EFFECTS OF RH-ISOIMMUNIZATION (HEMOLYTIC DISEASE)

## Foetal Manifestations

The various foetal manifestations as per severity of the disease are as follows:

### 1. Immune hydrops (Erythroblastosis fetalis)

Immune hydrops is the most severe variety of Rh isoimmunization. In immune hydrops, excessive and

prolonged hemolysis of foetal red cells causes anaemia stimulating marked erythroid hyperplasia of the bone marrow. There is increased extramedullary hematopoiesis in spleen and liver causing their dysfunction. There may be cardiomegaly and pulmonary hemorrhages. Fluid collections in the foetal thorax, abdominal cavity and skin is the hallmark of hydrops. Foetal hydrops occurs at a deficit of > 7 g Hb for that gestation. The placenta is markedly enlarged, edematous, boggy with large, prominent cotyledons and edematous villi. Hydrothorax may restrict lung development causing pulmonary compromise after birth. Ascites, hepatomegaly and splenomegaly may lead to severe dystocia. Hydropic changes are easily diagnosed on ultrasonography. The baby may be hydropic and stillborn with signs of maceration (Fig. 38.2).

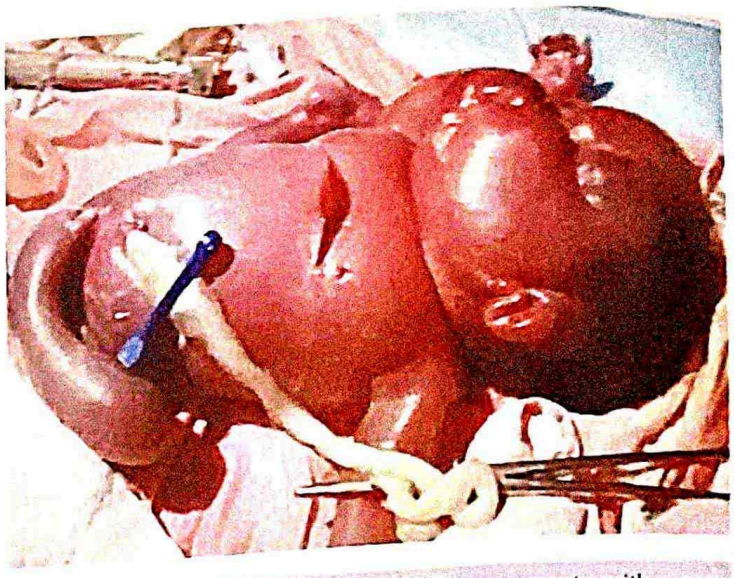


Fig. 38.2: Clinical photograph of a stillborn neonate with severe erythroblastosis fetalis showing signs of hydrops and macerations.

### Pathophysiology

Exact pathophysiology is unknown. Various theories are as follows.

1. Heart failure from prolonged severe anaemia
2. Capillary leakage caused by hypoxia with severe anaemia.
3. Portal and umbilical venous hypertension from hepatic parenchymal disruption by extramedullary hematopoiesis.
4. Lower colloid osmotic pressure due to liver dysfunction and hypoproteinemia.

### Diagnosis

1. Blood group of woman is Rh-negative.
2. Positive maternal Indirect Coomb's test
3. There is usually associated hydramnios.
4. There is usually a history of previous perinatal loss related to hemolytic disease of the newborn in previous pregnancies.
5. **Ultrasound findings.** There may be foetal hydrops, foetal ascites, scalp and skin edema and pericardial or pleural effusion and hepatosplenomegaly in the fetus (see Figs. 38.5 and 38.6).
6. There may be sinusoidal pattern on cardiotocography in severe affection due to foetal anaemia.
7. Maternal X-ray abdomen is not usually needed in modern obstetrics. If done in smaller centers, it shows fetus in so called **'Buddha position'** with a halo around its head due to scalp edema.
8. **Examination of the baby at birth.** The baby may be hydropic and stillborn (Fig. 38.2). If alive the baby has severe pallor (anaemia), grossly swollen body with enlarged abdomen due to ascites and associated hepatosplenomegaly.
9. **Examination of placenta after delivery.** Depending upon the severity of the disease, there may be gross placentomegaly with hydropic changes with liquid emanating from it. On histopathological examination, there is persistence of cytotrophoblastic layer with marked thickening of the chorionic villi.

## 2. Icterus Gravis Neonatorum

It is a type of Rh isoimunization in which baby is less severely affected as compared with immune hydrops. Baby is usually born in good condition but jaundice appears within 24 hours of birth. During in utero stage, excess of bilirubin from foetal red cell hemolysis which was being cleared by the placenta stops after birth leading onto neonatal anaemia and jaundice from continued foetal red cell hemolysis. There is risk of **Kernicterus** in which excess levels of unconjugated bilirubin may cross the blood brain barrier and may damage the central nervous system especially, the basal ganglia and eighth cranial nerve nucleus causing sensorineural deafness. It may occur if serum bilirubin levels in the baby exceed 20 mg/dL and is more likely to happen in premature dehydrated and sick babies. It is prevented and treated by phototherapy, adequate hydration and exchange transfusion (see below).

## 3. Congenital hemolytic anaemia

It is the least severe form of Rh isoimmunization and is also called congenital anaemia of the newborn. As there is very mild hemolysis of foetal red cells occurring in the baby, there is development of mild to moderate anaemia (pallor) in the baby very slowly with hemoglobin usually between 9–10 g/dL. Jaundice is usually absent. Initially due to extramedullary erythropoiesis, there may be mild to moderate hepatosplenomegaly. The condition usually settles down by 6 weeks of life by which time antibodies causing hemolysis are usually exhausted. Such infants usually need iron supplementation but may rarely need blood transfusion or erythropoietin therapy if anaemia becomes severe.

# MATERNAL EFFECTS

The maternal effects of congenital anaemia of the newborn are less marked than foetal effects.

1. Higher chances of **pre-eclampsia, hydramnios** and **macrosomic fetus**. There is risk of coagulopathy and hypofibrinogenemia due to the dead fetus.
2. Increased incidence of **postpartum haemorrhage** due to a placentomegaly and coagulopathy.

3. **Mirror syndrome.** It is caused by vascular changes in the placenta causing pre-eclampsia. The mother develops edema similar to the fetus, hence the name.
4. **Grandmother theory.** Rarely the D-negative female fetus is exposed to D antigen from the mother and sensitized as a result. If such a sensitized female child grows up to bear an Rh-positive fetus, the fetus is affected by antibodies initially provoked by its grandmother's blood. As this is very rare, anti D prophylaxis is not recommended.
5. **Amnestic response.** Presence of an antibody titre in the mother need not necessarily depict the foetal affection. Previously sensitized women may have a higher load during subsequent pregnancies even if the current fetus is D-negative.

## PREVENTION OF RH-IMMUNIZATION

Injection Anti D immunoglobin (IgG) should be given to all Rh-negative mothers who have given birth to Rh-positive fetus.

### Mechanism of action

Exact mechanism is not known. Probable mechanisms are as follows:

1. Rapid macrophage mediated clearance of anti D coated red blood cells (Fig. 38.3).
2. Down regulation of antigen-specific B cells before occurence of immune response.
3. In the past it was believed that anti D blocked the Rh-antigen of foetal red cells but a significant number of RhD antigen sites on foetal RBCs in maternal circulation are not bound by passive anti D. Hence, epitome masking is not the main mechanism of action of anti D.

Anti D immunoglobulin is extracted by cold alcohol fractionation from plasma donated by alloimmunized women and male donors (given repeated injection of RhD+ red cells to develop high titred polyclonal anti D plasma. It is free of viruses and other infection due to use of fractionation, ultrafiltration and screening techniques.

### Current Recommendations for Immunoprophylaxis

1. Prophylactic Anti D immune globulin should be offered to unsensitized RhD negative women at 28 weeks of gestation. Following birth, if the infant is confirmed to be Rh positive, all RhD negative women who are not known to be sensitized should receive anti D immune globulin within 72 hours of delivery (Level A evidence Grade IA recommendation [ACOG 2017]). It is also given for sensitization events shown in Table 38.1. Routine anti-D prophylaxis has to be given regardless of previous dose given for a potentially sensitising event.
2. Although the risk of alloimmunization is low, the consequence can be significant and administration of RhD immune globulin should be considered in cases of spontaneous first trimester miscarriage, especially those that are later in first trimester (level C evidence) (This is a new change. In past Anti D was not prescribed for early first trimester spontaneous abortions). The dose of Anti-D recommended to be given prior to 20 weeks of gestation is 50 ug or 250 IU.
3. Rh D immune globulin should be given to RhD negative women who have pregnancy termination, either medical or surgical
4. RhD immunoglobulin for all cases of ectopic pregnancy in RhD negative women.
5. American College of Obstetricians and Gynecologists (ACOG, 2017) recommends single dose of 300 µg of anti D at 28 weeks of gestation followed by one dose after delivery. This practice is followed in India. This practice reduces the incidence of isoimmunization from 2% with only post delivery anti D to 0.1%. As half life of anti D is only 24 days and protective effect of anti D lasts for about 6 weeks, post delivery anti D is still required despite antepartum prophylaxis.
6. Women who are weakly RhD+ (previously Du positive) are not at risk of RhD alloimmunisation and do not require anti D prophylaxis.
7. **When in doubt, it is better to give anti-D as it causes no harm but not giving it in an indicated case may have serious consequences.** Women undergoing

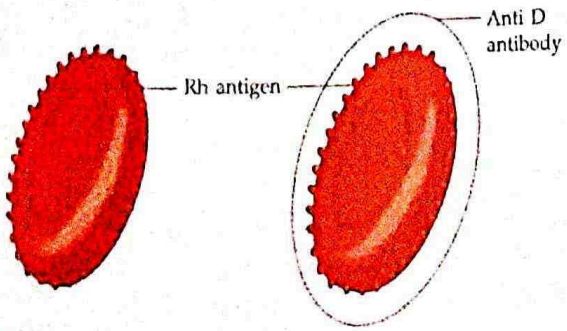



Fig. 38.3: Mechanism of anti D immunoglobulin. It surrounds the foetal RBC (dotted circle) preventing exposure of antigen sites (spikes) thereby preventing antibody formation

**Table 38.1: Indications of Anti-D Immunoprophylaxis**

| Early Pregnancy | Late pregnancy |
|---|---|
| 1. Threatened abortion | 1. Late miscarriage |
| 2. Miscarriage | 2. Amniocentesis |
| 3. Medical or surgical MTP | 3. Cordocentesis |
| 4. Ectopic pregnancy (medical or surgically managed) | 4. Foetal surgery or intervention |
| 5. Hydatidiform mole especially partial (trophoblast cells may express D-antigen) | 5. Antepartum haemorrhage |
| 6. Chorionic villus sampling | 6 External cephalic version |
| 7. Other intrauterine procedures | 7. Routine antepartum prophylaxis (28 weeks) |
| 8. Multifetal reduction | 8. After delivery (within 72 hours) |
| 9. Abdominal trauma | 9. Manual removal of placenta |
| | 10. Intrauterine foetal death |

ligation should also be given anti D as they may later come back for recanalization.

The indications for anti D prophylaxis are listed in Table 38.1. Dose of anti D as per recent NICE guidelines is 300 µgm (1500 IU) (Minimum dose 50 µg).

8. Following the delivery of IUFD fetus, Anti-D prophylaxis should be given within 72 hours of delivery.
9. Anti-D should be given following tubal ligation as the woman may later come for recanalization.
10. D-negative woman presenting with continual uterine bleeding for example in placenta previa should be given a minimum dose of 250 IU of anti-D Immunoglobulin <20 weeks and 500 IU beyond 20 weeks at a minimum of 6 weekly interval.

### Screening and Quantification for Fetomaternal Haemorrhage (FMH)

Initially ACOG recommended screening FMH for high risk cases (abruptio placentae trauma), but this policy identifies only 50% cases. Hence, presently RCOG and ACOG have recommended screening of all women at the time of delivery for excessive FMH. First of all, **Rosette test** is performed which is a qualitative and sensitive test and gives positive or negative result. If rosette is negative (FMH < 2mL), standard dose of anti D (300 µg) is administered. If rosette test is positive (FMH > 2mL), then **Kleihauer-Betke** stain or foetal cell stain using flow cytometry is performed. Kleihauer-Betke test is an acid elution test using citric acid as foetal red cells containing HbF are more resistant to acid elution as compared with maternal erythrocytes containing HbA (Fig. 38.4). Following is the calculation for volume of FMH and dose of Anti D required.

#### Determination of Volume of FMH and dose of Anti D to be given

**First method:**

$$\text{Volume of FMH} = \frac{\text{Percentage of foetal red blood cells}}{} \times 50$$

$$\text{Number of vials of Anti D required} = \frac{\text{Volume of FMH}}{30}$$

**Alternative method.**

$$\text{FMH of 4 mL of foetal blood} = \frac{80 \text{ foetal RBC}}{50 \text{ lower power fields}}$$

= Dose of Anti-D required: 100 µgm: 25 µg/mL of FMH. Every 10 µg of Anti-D required for 0.5 mL of foetal RBCs or 1 mL of whole foetal blood.

A dose of 300 µg of anti D given routinely can neutralise a FMH of 30 mL and 15 mL of foetal RBCs. Up to five 300 µg doses (total 1500 µg) of anti D can be given intramuscularly over 24 hours period. If more anti D is required, then intravenous preparation of anti D in dose of 600 µg every 8 hourly is given till total dose is achieved. It should be done in consultation with the blood bank. Flow cytometry is performed by some centres where Kleihauer test indicates a large FMH as it is more accurate and quantifies FMH better than Kleihauer test. Unfortunately, screening for FMH is not routinely done in India and most hospitals have gone out of practice to do Kleihauer test.

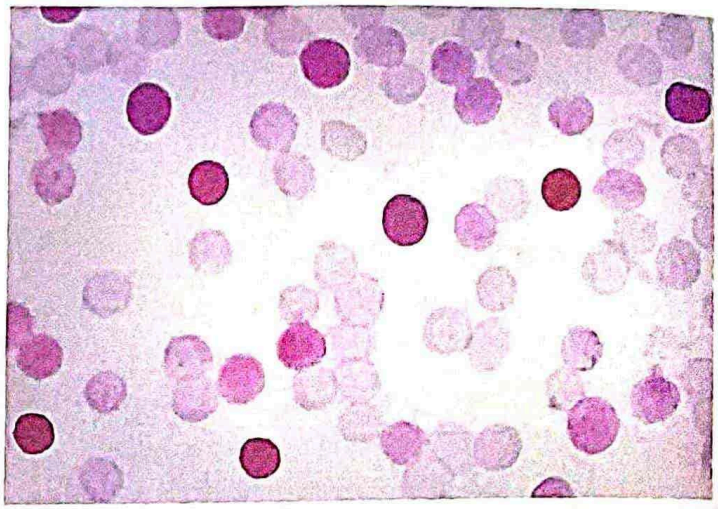


**Fig. 38.4:** (A) Kleihauer-Betke acid elution test showing dark foetal red cells (rich in hemoglobin F) in peripheral smear. Maternal red cells are seen as empty cells.

### Free Foetal DNA Testing in Maternal Serum

It is now possible to establish foetal Rh genotype non-invasively in maternal serum using polymerase chain reaction which picks up foetal DNA even from one cell. If fetus is Rh-negative then anti D is not indicated. However, it is not yet available in India.

### To prevent or minimize feto-maternal bleeding

Aim is to be gentle with abdominal examination and procedures.

1. Do not transfuse Rh-positive blood to Rh-negative females throughout life.
2. Abdominal palpation should be avoided in antepartum haemorrhage cases.
3. External cephalic version should not be done under general anesthesia.
4. Any invasive procedures like chorionic villus sampling or cordocentesis amniocentesis should always be performed under ultrasonic control.
5. Do not use prophylactic ergometrine at the time of delivery of the anterior shoulder to prevent PPH.
6. At cesarean section, blood spilling should be avoided and manual removal of placenta should be avoided.

## MANAGEMENT OF NON-IMMUNIZED MOTHER

If the indirect Coomb's test is negative, the mother is not yet immunized. The test should be performed again at 28 and 34 weeks in a primigravida and monthly in a multigravida after 28 weeks. Administer anti D 300 µg at 28 weeks if ICT is negative. If the indirect Coomb's test is persistently negative, her pregnancy can be continued up to 40 weeks. She is not allowed to go beyond the due date.

At delivery, early cord clamping is done and cord blood is sent for hemoglobin, bilirubin and direct Coomb's test.

## MANAGEMENT OF IMMUNIZED PATIENT

Management of Rh-negative mothers is shown in Fig. 38.9.

### 1. History taking

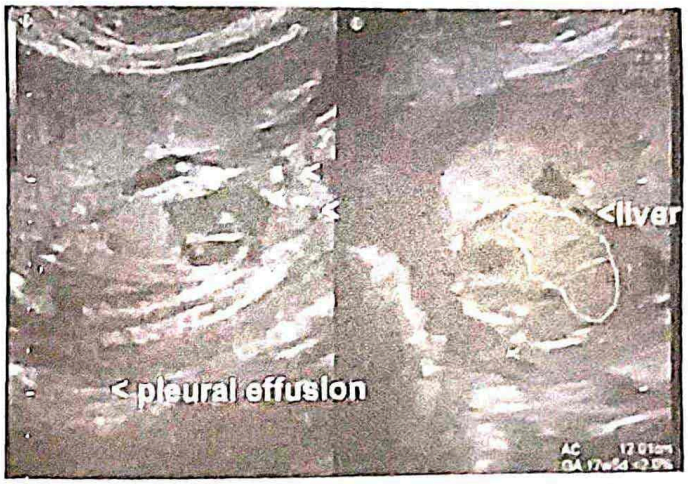
History taking, including obstetric history, is important especially past history of blood transfusion and previous obstetric history. **In Rh-negative isoimmunization, there is worsening obstetric history with first baby being usually normal (unlike syphilis where there is improving obstetric history).**

### 2. Indirect Coomb's test (ICT)

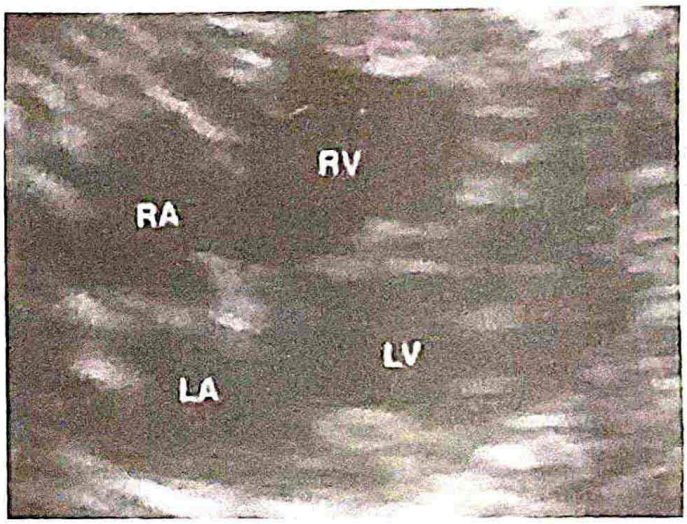
It is used to detect antibodies against foetal RBCs that are present unbound in the mother's serum. If the ICT is positive, it should be repeated in dilution to find out the antibody titre. The critical titre for anti-D antibodies varies from lab to lab and is usually 1 : 16. Management should be individualized after taking obstetric history and the details of gestational age when foetal anaemia developed in the last pregnancy. Anaemia tends to occur earlier and is more severe in next pregnancy and this is an indication for amniocentesis to detect bilirubin. If the titre is less than 1 : 16, it is repeated in every two weeks. Now in many centers it is possible to do a quantitative immunoassay for anti-D antibodies. The safe level is < 4 IU/mL. Once the critical titre is reached, then subsequent titres are not helpful and further evaluation is needed. Serial titres are not useful for monitoring foetal status when the mother has had a previously affected fetus or neonate (ACOG 2017).

### 3. Paternal blood group genotyping

The presence of antibodies as shown by a positive ICT may be due to the amnestic response. Therefore, it is important to know whether the father is heterozygous or homozygous for the D antigen. If the father is homozygous for the D antigen, there is a 100% chance of having an Rh-positive fetus. If he is heterozygous for the D antigen, there is only a 50% chance of having an Rh-positive fetus. This is not cost effective in low resource setting like India.




(A)




(B)

**Fig. 38.5:** (A) Rhesus isoimmunized fetus with hydrops and foetal ascites (arrow). The placenta is large and thick (> 4 cm). (B) Rhesus isoimmunized fetus with hydrops with cardiomegaly
Kind courtesy: Dr Ashok Khurana, Delhi

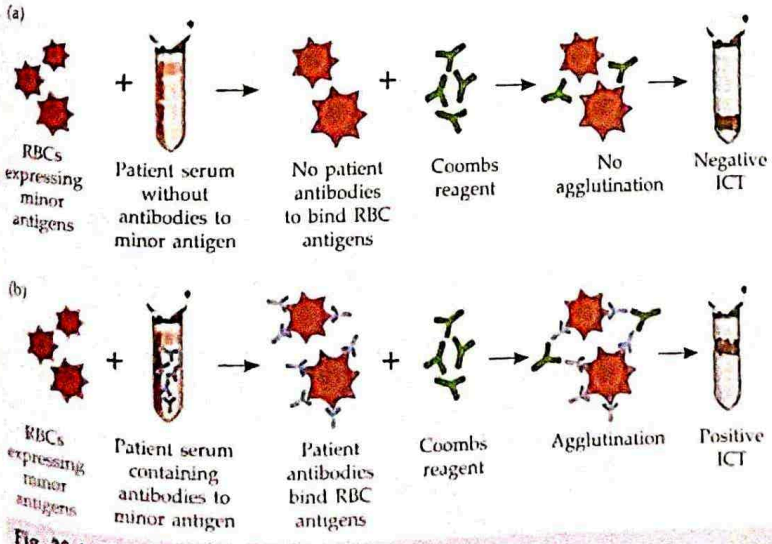


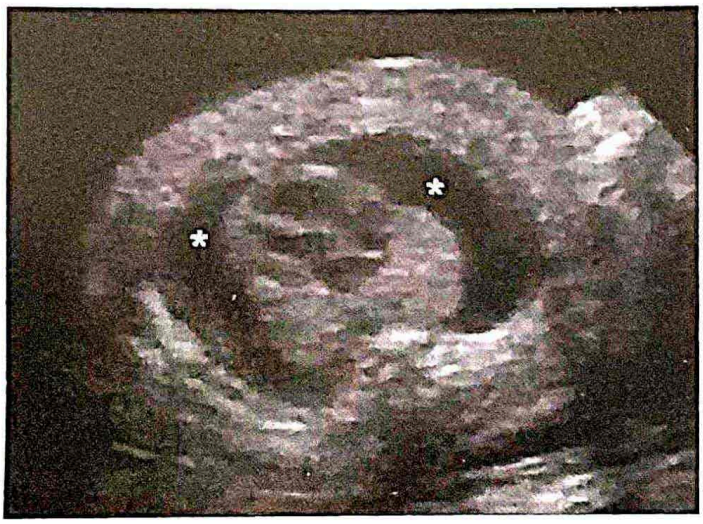

**Fig. 38.4:** (b) Indirect Coomb's Test

### 4. Foetal blood group determination

If the father is heterozygous, there is a chance that the fetus may be Rh-negative. Therefore, in cases of severe immunization in the past, it may be useful to find out the foetal blood group. It is possible only in advanced centers and indicated only when the father is heterozygous for the D antigen and there is a history of severe immunization. Chorion villus sampling or amniocentesis for foetal DNA testing is done. This avoids the need for further testing in the case of an Rh-negative fetus. The foetal genotype can also be identified in the maternal blood sample as foetal DNA circulates in maternal blood in low amount. It is however, not yet available in India.




(A)



(B)

**Fig. 38.6:** (A) Isoimmunized fetus showing hepatomegaly and skin edema; (B) Rhesus isoimmunized fetus showing bilateral pleural effusion
Kind courtesy: Dr Ashok Khurana, Delhi

## 5. Middle Cerebral Artery (MCA) Peak Systolic Velocity (MCA PSV)

Doppler measurement of peak systolic velocity in foetal middle cerebral artery is an appropriate non-invasive method to monitor pregnancies complicated by red cell alloimmunization (level A recommendation) (ACOG 2017).

In most specialized centers (e.g. AIIMS, New Delhi), serial measurements of the peak systolic velocity of middle cerebral artery are done routinely to detect foetal anaemia in Rh isoimmunized pregnancies instead of amniocentesis. In anaemia, blood is shunted preferentially to brain to maintain its oxygenation. The peak systolic velocity of middle cerebral artery is increased due to hypoxia, brain sparing high cardiac output and decrease in blood viscosity due to hemolysis and anaemia. MCA PSV between 1–1.29 MoM depicts mild anaemia, while MCA PSV between 1.29–1.5 MoM depicts moderate anaemia. Patients with MCA PSV values less than 1.5 MoM require regular follow up every 2 weekly and delivery should be planned around 37 – 38 + 6 weeks period of gestation as per ACOG. A value of more than 1.5 multiple of median (MOM) indicates foetal anaemia necessitating foetal blood sampling to determine the need of inutero blood transfusion (see below). After blood transfusion, MCA Doppler is of less value (also see chapters 15 and 46 for details). In fact current standard practice in most centres is to use serial MCV PSV for monitoring of these pregnancies with amniocentesis being rarely performed.

## 6. Ultrasound assessment of fetus is useful for:

1. Accurate dating in the first trimester
2. Foetal growth assessment
3. Identify hydrops and/or foetal ascites (Fig. 38.5 A), cardiomegaly (Fig. 38.5 B), hepatomegaly (Fig. 38.6 A) and pleural effusion (Fig. 38.6 B)
4. Placental thickness (> 4 cm in affected pregnancies) (Fig. 38.5 A).
5. Polyhydramnios
6. Foetal affection like pericardial or pleural effusion (Fig. 38.6 B)
7. Foetal Doppler shows increased velocity (umbilical vein, middle cerebral artery and ductus venosus have been used to detect anaemia instead of amniocentesis).
8. Sinusoidal pattern with loss of normal baseline variability on the cardiotocography.

## 7. Amniocentesis and amniotic fluid bilirubin evaluation

It is of historical interest now and is rarely used in current practice. However, it is discussed in details because of historical importance being land mark work in this field.

Amniocentesis and amniotic fluid bilirubin evaluation is based upon the fact that when hemolysis occurs, bilirubin is present in the amniotic fluid. The amount of bilirubin correlates with the degree of severity. Liley in 1961 established spectrophotometry as a method of assessing amniotic fluid bilirubin concentration.

### Indications

1. The antibody titre is more than 1 : 16 (critical titre) to determine whether the baby will be affected or not.
2. Past history of Rh isoimmunization.
3. Heterozygosity of father to know foetal affection.

### Timing of Amniocentesis

1. **In primigravidas and without past history.** It is performed at 30–32 weeks and again after 3–4 weeks if the report is normal.
2. **Previous Rh isoimmunized baby.** It should be performed 10 weeks before the last stillbirth or foetal affection as foetal anaemia occurs earlier and more severe in subsequent pregnancy. However, it should not be done before 20 weeks.

### Interpretation

Using spectrophotometer, the optical density 'deviation

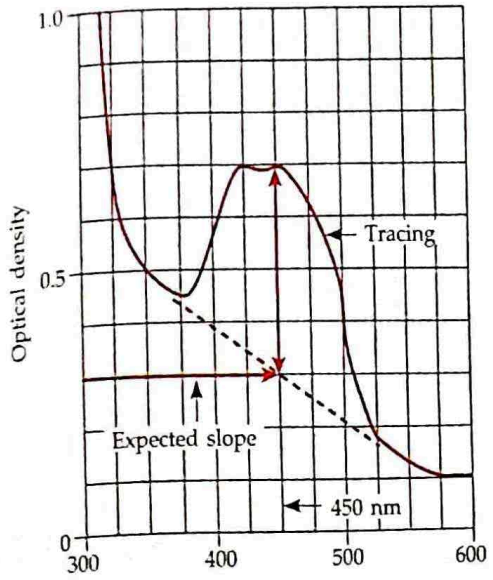



**Fig. 38.7:** Deviation bulge of optical density difference at 450 nm wavelength in spectrophotometric analysis of amniotic fluid in Rh isoimmunization.

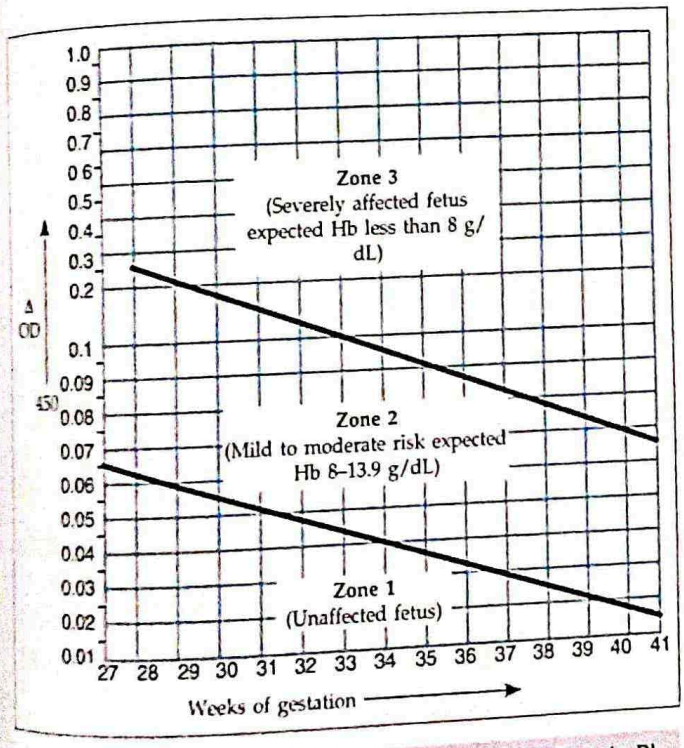



**Fig. 38.8:** Liley's graph to show severity of foetal hemolysis in Rh isoimmunization

bulge' of the liquor is observed at 450 nm wavelength for the severity of foetal hemolysis. The height of the 'deviation bulge' falls within one of the three zones when plotted in **Liley's graph** (Figs. 38.7 and 38.8) depending upon the gestation.

(*i*) **Liley's zone 1 (low zone).** The fetus has only mild disease or is unaffected. The pregnancy can be taken to term. Amniocentesis is repeated in 2–3 weeks.

(*ii*) **Liley's zone 2 (mid zone).** In this zone, the fetus is at moderate risk. It is further divided into **lower zone 2** (where expected foetal Hb is between 11.0 and 13.9 g/dL) and **upper zone 2** (where the foetal Hb ranges from 8 to 10.9 g/dL). Amniocentesis should be repeated in 2 weeks. If the value is increased, cordocentesis should be done. For foetal anaemia (hematocrit < 30%), intrauterine transfusion is indicated to increase foetal hematocrit to about 45%. Fetus is likely to require delivery at 34 weeks gestation.

(*iii*) **Liley's zone 3 (high zone).** Value in zone 3 indicates the fetus is severely affected with expected Hb of < 8 g/dL. Without therapy fetus can die within 7 to 10 days. If pregnancy is more than 34 weeks, delivery is indicated. For pregnancies before 34 weeks, urgent cordocentesis is needed to know foetal Hb, hematocrit and blood group. In the same sitting, intrauterine transfusion is done to increase foetal hematocrit to about 40% (see below for details).

### Modified Liley's Graphs

The original Liley's graph was applicable only to pregnancies beyond 27 weeks or more. Queenan et al. (1993) from USA have developed modified Liley's curves (also called **Queenan chart**) which can be used for earlier gestations beginning at 14 weeks. If there is likelihood of severe foetal anaemia or abnormal amniotic fluid evaluation, it is better to perform foetal blood sampling.

## 8. Foetal Blood Sampling and Intravascular Transfusion

Foetal blood sampling and intravascular transfusion is the best method to assess and treat foetal anaemia and should be done in a regional specialized center (e.g. AIIMS, New Delhi) under ultrasonic guidance (see later).

## 9. Termination of pregnancy

Pregnancy should be terminated in immunized mothers with severe foetal affection. Factors affecting termination of pregnancy are: (*i*) Past history of stillbirth due to Rh-isoimmunization (*ii*) High maternal antibody titre (*iii*) Increased middle cerebral artery peak systolic velocity (*iv*) The optical density difference falling in upper zone 2 or in zone 3 on Liley's graph (*v*) Affected fetus on ultrasound.

## 10. Timing of Delivery

For mild affection, the delivery should be conducted at 38 weeks.

For severe Rh-isoimmunization, it is better to terminate the pregnancy at 34 weeks. Some researchers continue transfusion until 36 weeks but most advocate transfusion up to 34 weeks, give steroids at 32 weeks for foetal maturity and deliver at 34 weeks.

### Where Proper Facilities are not Available

A balanced judgement has to be made between premature termination of pregnancy at 34 weeks vis-à-vis continuation of pregnancy for couple of weeks so that there is good chance of foetal survival exutero. The management of Rh-negative mother is shown in Fig. 38.9.

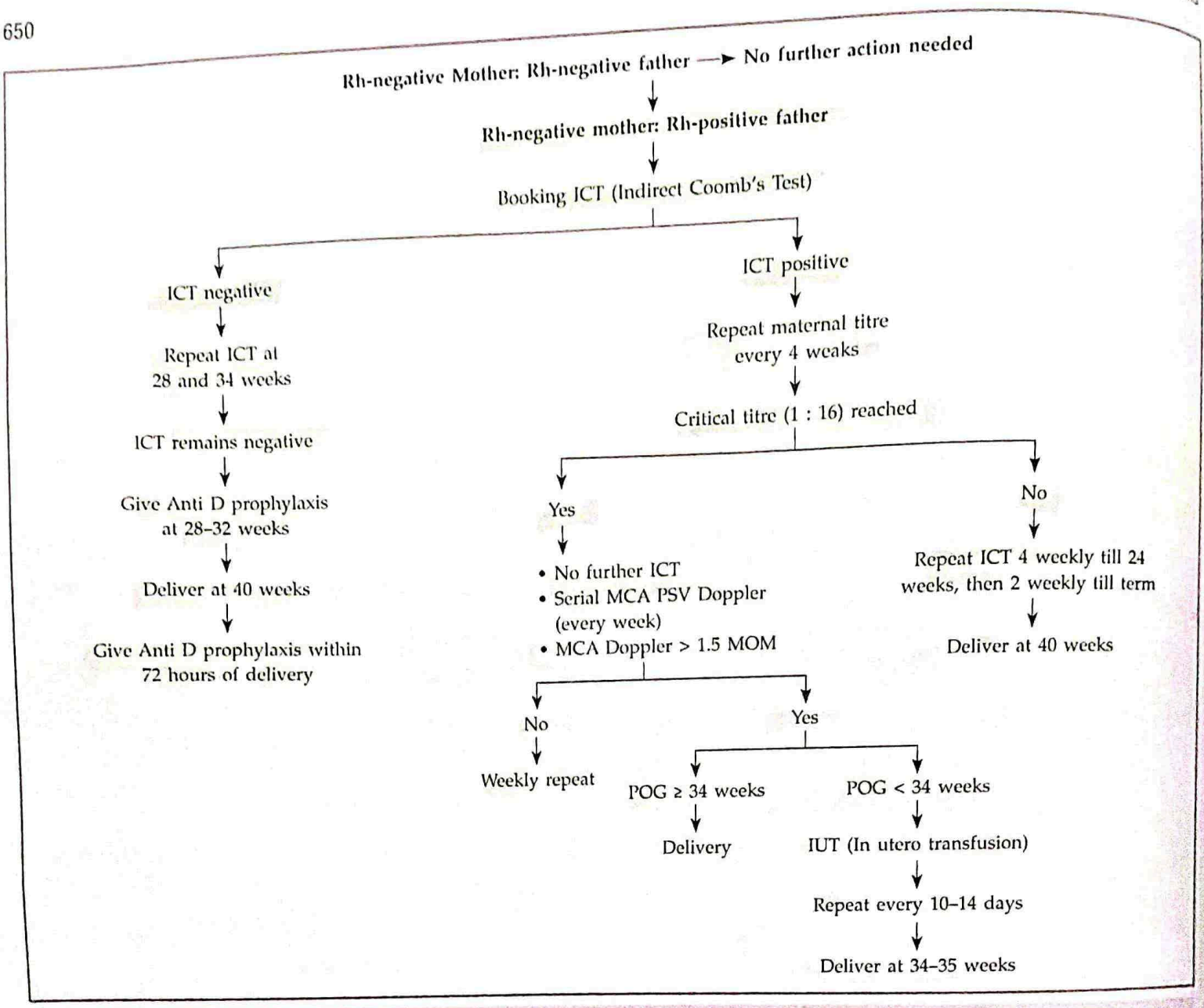



**Fig. 38.9:** Management of Rh-negative mother

## 11. Methods of Termination

### Vaginal Delivery

Induction of labour is performed depending upon Bishop's score. If Bishop's score is < 6, prostaglandin $E_2$ gel is used. If cervix is favourable (Bishop's score ≥ 6), amniotomy followed by oxytocin drip is started. Usually vaginal delivery can be accomplished.

### Cesarean Section

If termination has to be done for severe affection prematurely (34–37 weeks), with unfavourable cervix, cesarean delivery is preferred.

# INTRAUTERINE FOETAL TRANSFUSION

Intrauterine foetal transfusion is performed in a tertiary referral center with facilities for blood transfusion, neonatal intensive care center and expert obstetricians trained in foetal medicine procedures (cordocentesis and intrauterine transfusion). It is required for severe Rh isoimmunization of fetus in utero before 34 weeks gestation with severe foetal anaemia (foetal hematocrit < 30% or single value in zone 3 of Liley's graph). Benefits are to increase foetal RBC count and oxygen carrying capacity and control of foetal anaemia. There are 3 types of intrauterine transfusions, namely:

1. Intravascular (into umbilical vein)
2. Intraperitoneal
3. Combined

## 1. Intravascular Transfusion

Foetal blood transfusion is recommended when the foetal hematocrit is less than 30% which is two standard deviations below the mean at all gestational ages. Foetal hydrops develops with severe foetal anaemia (hematocrit < 15%).

Intravascular transfusion is now favored as it has proven efficacy and safety. The large placental volume allows rapid infusion of a large quantity of blood into foetal circulation. The differences between intravascular and peritoneal transfusion are given in Table 38.2.

**Procedure** (Fig. 38.10)

Under ultrasonic guidance and all aseptic conditions, a 20

**Table 38.2: Differences between Intravascular and Intraperitoneal Transfusion**

| Intravascular transfusion | Intraperitoneal transfusion |
|---|---|
| Blood transfused into the umbilical vein<br>**Advantages**<br>1. Higher survival rate<br>2. Direct estimation of hematocrit can be done.<br>3. Significant reduction in low Apgar score, cesarean delivery<br>**Disadvantages**<br>1. Foetal bradycardia<br>2. Risk of fetomateral haemorrhage<br>3. Volume overload<br>4. Difficult to perform and needs skill | Blood is transfused into the peritoneal cavity.<br>**Advantages**<br>1. Easier to perform<br>2. Acts as a reservoir for sustained release of hemoglobin.<br>**Disadvantages**<br>1. Foetal distress<br>2. Prelabor rupture of membranes (PROM)<br>3. Infection<br>4. Trauma to other organs; 5. Less effective |

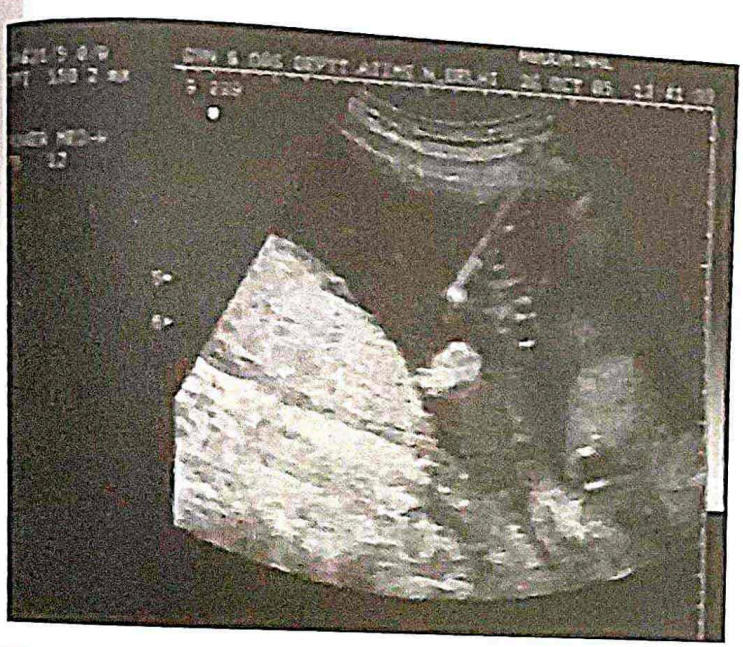


**Fig. 38.10:** Procedure of intrauterine transfusion through umbilical vein
Kind Courtesy: Prof. Deepika Deka, AIIMS, New Delhi

guage spinal needle is passed into the umbilical vein at the placental insertion site (most preferred site) (Fig. 38.10) but other veins like hepatic vein can be used when it is not possible through umbilical vein. One mL foetal blood is aspirated and tested for foetal hematocrit, Hemoglobin packed volume and blood group.

If the hematocrit is below 30%, intravascular transfusion is carried out in the same sitting using a three-way stopcock connected to the needle via extension tubing. Blood used for transfusion should be 'O', RhD negative relatively fresh and crossmatched against mother's blood with screen negative for viruses and irradiated to destroy white cells to avoid graft versus host reaction. Blood is transfused at a rate of 5–10 ml/minute.

It should be packed to a hematocrit of 80–90% to achieve a foetal hematocrit of 50%. The volume of blood transfusion can be estimated by the following **Mandelbrot's formula:**

$$\text{Amount of blood to be transfused} = \frac{\text{Final hematocrit} - \text{Initial hematocrit}}{\text{Hematocrit of transfused blood (usually 80\%)}} \times \text{Foetal placental volume (150 mL/kg)}$$

Example: If initial hematocrit is 30%, estimated foetal weight is 1 kg, then volume of blood transfusion will be

$$= \frac{50 - 30}{80} \times 150 = 37.5 \text{ mL}$$

Usually 40 to 60 mL blood is given in one sitting. Hematocrit falls by 1% per day. Intrauterine transfusion is repeated once hematocrit falls below 30% (7 to 14 days). Prior to transfusion pancuronium can be given in the foetal thigh to decrease foetal movements. It can be started at 18 weeks and can be repeated every 1–2 weeks until 34 weeks.

Cesarean section is advisable considering severity of foetal affection and need of urgent delivery.

Complications of foetal blood sampling and intravascular transfusion include foetal bradycardia, haemorrhage, cord haematoma, tamponade and foetal death.

## 2. Intraperitoneal transfusion

In this case, O negative packed cells (hematocrit 80–90%) are transfused with a 20 gauge needle inserted in the foetal peritoneal cavity under ultrasonic guidance from where they are absorbed by the subdiaphragmatic lymphatics If there is foetal ascites, the ascitic fluid can be first drained before blood transfusion. It is easier to perform and can be started at 18 weeks to be repeated every 2 weeks. The volume of blood to be transfused in mL is calculated as follows:

$$\text{Amount of blood to be transfused (mL)} = (\text{gestational age in weeks} - 20) \times 10$$

Thus, a woman at 30 weeks gestation will require 100 mL (10 × 10) transfusion. However, it has largely been replaced by much superior intravascular transfusion (see Table 38.2).

## 3. Combined Method

Combined method in which initially intraperitoneal transfusion is given followed by intravascular transfusion when it becomes feasible.

Volume to be transfused should not raise the final hematocrit to more than 4 times the initial value because that may cause volume overload and precipitate cardiac failure of the hydropic fetus. Hydrops usually reverses after 1-2 intra-uterine transfusions however, placentomegaly is the last feature to go.

## OTHER THERAPIES

The following therapies were used in the past.

1. **Plasmapheresis.** In this many liters of maternal plasma containing anti-D antibodies was removed in the hope to reduce its level.
2. Administration of promethazine membranes in enteric capsules to decrease phagocytosis by foetal reticuloendothelial system.
3. Immunosuppression with corticosteroids
4. *Intravenous immunoglobulin (IVIG) therapy.* It is given in dose of 1 g/kg maternal body weight to reduce circulating maternal alloantibody levels by almost 50% due to negative feedback by total circulating maternal IgG levels. It also interferes with transfer of maternal antibodies across placenta and reduces IgG coated foetal red cell hemolysis in foetal reticuloendothelial system. It is still used but is very expensive. However, except for IVIG, other therapies have not been found useful.

## PRECAUTIONS DURING DELIVERY AND CESAREAN

(*i*) Continuous electronic foetal monitoring should be performed for foetal jeopardy.
(*ii*) Active management of the third stage should be withheld.
(*iii*) One should be gentle during delivery, cesarean and avoid blood mixing.
(*iv*) For any postpartum haemorrhage, use oxytocin instead of ergometrine.
(*v*) Manual removal of placenta should be avoided as far as possible.
(*vi*) The umbilical cord is promptly clamped to minimize fetomaternal blood mixing to the mother. The cord is kept as long as possible (about 15 cm) as baby may need exchange transfusion.
(*vii*) About 5 mL cord blood must be collected in all case without squeezing the cord.
(*viii*) **Oxalated/EDTA blood.** 2 mL for hemoglobin estimation, hematocrit and peripheral smear for hemolysis.
(*ix*) **Clotted blood.** 3 mL for blood group, direct Coomb's test and serum bilirubin.

### General management of newborn

Birth should be attended by the pediatrician or neonatologist as baby may need resuscitation. O Rh-negative blood should be made available. For affected newborn, immediate resuscitation, supportive therapy, temperature regulation and monitoring, correction of acidosis with sodium bicarbonate, volume expansion, exchange transfusion and assisted ventilation should be available. The Hb concentration, hemotocrit and serum billirubin levels are measured at 4–6 hours intervals.

## EXCHANGE TRANSFUSION IN THE NEWBORN

Neonatal exchange transfusion is a very useful and life saving procedure which removes hemolyzed and antibody coated red blood cells and unattached antibodies and replaces them with donor red blood cells which lack sensitizing antigen in affected fetuses. It helps baby in critical first two weeks after which the infants's own blood forming mechanism usually takes over. **Ultimately the baby's blood group will not change and will remain Rh-positive due to inherent tendency of making Rh-positive cells.**

### Indications

1. Cord hemoglobin value of 10 g/dL or less (or hematocrit of < 30%)
2. Cord bilirubin concentration of 5 mg/dL or more.
3. Increase in newborn's bilirubin at rate of > 1 mg/dL/hour even after phototherapy.
4. Term infants with bilirubin levels ≥ 20 mg/dL.
5. History of Kernicterus or severe erythroblastosis in previous baby in past.
6. Reticulocyte count of > 15% and prematurity (refer to Liley's graph) are additional indications of exchange transfusion.
7. Repeat transfusion is indicated if Hb falls < 10 g/dL or bilirubin rises to 20 mg/dL or more.

**Technique** (Fig. 38.11)

Blood for transfusion should be as fresh as possible. Heparin or citrate phosphate dextrose adenine solution is used for anticoagulation. **Before delivery, blood should be kept ready taken from a type O, Rh-negative donor with low titre of anti A and anti B antibodies and should be tested for compatibility with the mother's serum using indirect Coomb's test. After**

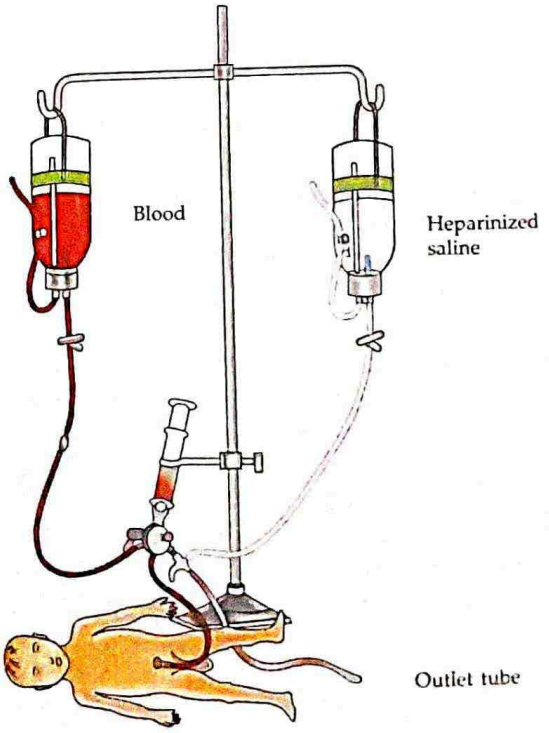



**Fig. 38.11:** Exchange blood transfusion being conducted in an isoimmunized neonate

**delivery, blood should be taken from either type O, Rh-negative donor (preferred) or from Rh-negative donor with same ABO group as baby when mother also has same group after testing for compatibility (crossmatching) with sera of both mother and the baby.** Blood should be gradually warmed to a temperature between 35–37°C and should be kept well mixed by gentle squeezing to avoid sedimentation. Stomach of baby should be emptied by gastric tube to prevent aspiration, body temperature should be kept constant and warm and vital signs should be monitored. An experienced pediatrician (neonatologist) accompanied by a competent assistant should perform the procedure and monitor the baby. The procedure should be performed only in experienced neonatal referral centers.

Under all strict aseptic conditions, the umbilical vein is cannulated with a polyvinyl catheter (about 1 mm diameter) to a distance of about 7 cm in a full term baby when full free flow of blood is achieved indicating the position of catheter to be in a large hepatic vein or the inferior vena cava. As an alternative exchange may be carried out through peripheral veins (for infusion) and arteries (to draw out blood). The exchange is carried out over 45–60 minutes using 35–40 cycles of aspiration of an aliquot of 3–5 mL/kg (less for preterm and sick infant) followed by transfusion of same aliquot (3–5 mL/kg) of donor blood **(pull-push method)** with one cycle taking 60–90 seconds. The goal is to exchange about two blood volumes of the baby (about 170 mL/kg body weight). Hematocrit and bilirubin levels are measured before and after the transfusion. Bilirubin levels are measured every 4–8 hourly. Intensive phototherapy is started to keep bilirubin under control. Repeat transfusion is performed if serum bilirubin rises to 20 mg/dL or more in spite of phototherapy.

Sodium bicarbonate and calcium gluconate are not routinely used but given if acidosis or tetany develop. Baby is continuously monitored during and after transfusion for vital signs, any bleeding or infection at umbilicus site, and estimation of hematocrit, bilirubin and glucose levels. Antibiotics should be given for three days.

### Complications

Exchange transfusion is not without complications and should only be done in tertiary referral centers with adequate technical skills and good blood bank facilities. The complications are as follows:

#### Acute complications

They are observed in 5–10% cases.

1. *Risk of death.* 0.3/100 procedures in experienced hands. Death can occur due to apnea, bradycardia or heart failure or other complications.
2. Transient bradycardia
3. Cyanosis
4. Transient vasospasm
5. Apnea
6. Thromboembolism
7. Congestive heart failure
8. Acidosis due to citrated blood
9. Hypoglycemia
10. Thrombocytopenia
11. Tetany
12. Infection

#### Late complications

1. Late anaemia which may be hemolytic or hyporegenerative bone marrow.
2. Graft versus host reaction manifesing as rash, diarrhea, hepatitis or eosinophilia.
3. Necrotizing enterocolitis is a rare complication.
4. Risk of infection of CMV, HIV and hepatitis from blood transfusion.
5. Inspissated bile syndrome
6. Portal vein thrombosis and portal hypertension possibly due to prolonged trauma or septic umbilical vein catheterization.
7. Kernicterus

### Phototherapy

Use of phototherapy decreases the need for exchange transfusion especially need for repeated exchange transfusion by causing photooxidation (degradation) of bilirubin. Narrow spectrum blue light (450–460 nm) are most effective but broad spectrum white and blue light (420–470 nm) are also effective. Bilirubin (4Z–15Z) in the skin absorbs light energy and undergoes reversible photoisomerization to 4Z, 15E bilirubin which can be excreted in bile without conjugation. Other major structural isomer formed from bilirubin after photochemical reaction is **lamirubin** which can be excreted by kidneys. Baby's eyes should be protected by covering or using dark glasses to avoid damage especially to the retina. Hydration, fluid requirements are increased and IV fluids should be given.

**Intravenous immunoglobulin.** Its use is advocated when serum bilirubin rises to dangerous levels despite phototherapy. Given in dose of 0.5–1.0 g/kg/dose 12 hours apart reduces the need of exchange transfusion possibly by decreasing hemolysis. However, recent studies do not recommend its use.

**Erythropoietin.** Use of subcutaneous erythropoietin 400 U/kg thrice a week for 2 weeks decreases need of repeated transfusion. Supplemental iron 6 mg/kg/day is also given.

### Other medications

Phenobarbitone given parenterally in dose of 5 mg/kg body weight three times a day may clear bilirubin by stimulating its conjugation but is not commonly used these days. Metalloporphyrins (Sn-mesoporphyrin) as a single intramuscular dose on first day of life may reduce bilirubin levels and decrease the need for phototherapy and hospital stay. Ursodeoxycholic acid has also been tried. However, currently only IVIG is used to treat unconjugated hyperbilirubinemia.

### Prognosis

Unaffected baby is normal. For affected babies, prognosis

has improved significantly due to universal use of anti D immunoglobulin, use of phenobarbitone MCA PSV (middle cerebralartery peak systolic velocity) and intrauterine transfusion especially in higher centers. Overall survival in affected fetus after intrauterine transfusion is 84% (92% for non-hydropic fetuses, 70% for hydropic fetuses).

**Long term outcome.** Cerebral palsy and developmental delay is more common in them due to prematurity, though normal outcome is expected in more than 90% cases. In cases where hydrops did not reverse even with intrauterine transfusion, the survival rate is only 40%. The irreversibility of condition despite successful correction of foetal anaemia is not known but may be due to severe injury to foetal endothelium from iron overload caused by intra foetal hemolysis. However, prognosis is not so good in smaller centers where these facilities are not available. Isoimmunized women should be counselled to be donors for anti D preparation.

### Breastfeeding

Breastfeeding is allowed to babies born to Rh-isoimmmunized mother.

### Therapeutic termination and permanent sterilization

Even with history of repeated affection, without any survivors, one should not take a drastic step of permanent sterilization. Termination of pregnancy with RhD+ fetus may also be an option following foetal genotype by chorionic villus sampling. The patients may conceive in near future with Rh-negative man. She may have a pregnancy by artificial insemination from Rh-negative donor. Preimplantation genetic diagnosis and transfer of the Rh-negative embryo (when the husband is heterozygous) or use of gestational surrogate can be resorted for these couples.

## OTHER TYPES OF ANTIBODIES CAUSING ISOIMMUNIZATION

### ABO incompatibility (Isoimmunization)

ABO incompatability is almost always due to mother being group O and fetus being group A or B or AB. Natural anti-A and anti-B antibodies of O group mother are IgG and they readily cross placenta while natural anti-A or anti-B antibodies of group B or group A is IgM and do not cross placenta. Thus, blood group O mother carrying group A or B fetus as is the father will be mildly affected by isoimmunization. Hemolysis due to anti-A is more common than anti-B. However, hemolysis due to anti-B can be more severe necessitating transfusion.

However, sometimes the antibodies may exist as lighter IgG molecules which readily cross the placental barrier after challenge with a previous incompatible pregnancy. The disease is milder than Rh immunization and the affected baby may have neonatal anaemia and jaundice which is usually treated with phototherapy.

### Minor Blood Groups

Rarely sensitization can be caused by minor blood groups which is becoming more common as D sensitization is becoming less common. These can be as follows:

1. **C antigen disease.** It will manifest like D isoimmunization but in much milder form. Treatment is similar.
2. Kell group disease (Kell antibody titres are not appropriate for monitoring as they do not corelate with foetal status
3. K group disease
4. Duffy antigen disease

### Non-immune Hydrops Fetalis (NIHF)

It is defined as excess of extracellular fluid accumulation in two or more body areas such as thorax, abdomen or skin in absence of circulating antibodies against red blood cell antigens. It is usually associated with hydramnios and hydropic and thickened placenta (placentomegaly). Incidence varies from 1 in 1500 to 1 in 3500 pregnancies. In developed nations NIHF is now responsible for majority (75–87%) of cases of hydrops fetalis due to success of anti D gammaglobulin prophylaxis in Rh-negative pregnancies. In India, however, immune hydrops (Rh-negative pregnancies) still continues to be the leading cause of hydrops fetalis but NIHF cases are being encountered more freqnently now.

#### Etiology

The various causes of non-immune hydrops are as follows:

1. Foetal causes
   (i) Anomalies like **cardiovascular** (ASD, VSD and other defects) (22%) (most common), **thoracic** (diaphragmatic hernia, cystic adenomatous malformation [6%]), **lymphatic syndromes** (5.7%) (e.g. cystic hygroma), dwarfism, arthrogryposis ((4.4%), urinary tract obstruction (urethral stenosis, bladder neck obstruction, 2.3%) and miscellaneous anomalies.
   (ii) Aneuploidies (chromosomal) like trisomy 21 and other trisomies, Turner syndrome (13%)
   (iii) Vascular like A V shunts, thrombosis of large vessels
   (iv) Infections. TORCH infection, syphilis, parvovirus B-19 infection (6.7%).
   (v) Multiple pregnancy. Twin-twin transfusion syndrome (TTTS), twin-reverse arterial perfusion syndrome (TRAP).
   (vi) Hematological and miscellaneous (10.4%) like $\alpha_4$ thalassemia (Barts Hb) common in South-East Asian population, anaemia, metabolic diseases.
2. **Placental causes.** Chorioangioma, fetomaternal haemorrhage, A–V shunts, placental trauma, twin-twin transfusion syndrome.
3. **Maternal causes.** Certain medications like indomethacin
4. Idiopathic (17.8%)

### Prognosis

Overall outcome is poor with perinatal death rate between 40–98%. If hydrops develops before 24 weeks, mortality is 95% while if it develops after 24 weeks and has structurally normal heart, mortality is 80% with 20% survival. Risk of recurrence is low for non-genetic cases.

### Diagnosis

1. Targetted ultrasound for congenital anomalies, arrhythmias and complications of twin pregnancies.
2. *Maternal blood tests.* Hb electrophoresis, indirect Coomb's test, Kleihauer-Betke test, serological tests for syphilis, toxoplasma, rubella, cytomegalovirus and parvo virus B-19.
3. Amniocentesis for foetal karyotyping and intrauterine infection.
4. Doppler studies for middle cerebral artery peak systolic velocity (MCA PSV) for severity of foetal anaemia
5. Cordocentesis for foetal hematocrit, infections and for intrauterine transfusion.

### Maternal complications

1. Hydramnios
2. Preterm labour is common due to associated hydramnios
3. Placental abruption due to sudden decompression of hydramnios.
4. Maternal mirror syndrome with mother developing pre-eclampsia and edema like fetus due to vascular changes in swollen and hydropic placenta (placentomegaly)
5. Difficult delivery
6. Postpartum haemorrhage due to uterine atony

### Management

It is individualized depending upon the etiology and after consultation with parents. Termination of pregnancy can be performed before foetal viability. For severe congenital malformations and chromosomal anomalies detected late in pregnancy beyond legal limit of MTP, expectant management is practised. For fetuses without malformations and aneuploides and with treatable conditions like tachyarrythmias or parvo virus infection, aggressive pharmacological treatment is offered. Severe foetal anaemia is treated by cordocentesis and intrauterine blood transfusion.

For twin-twin transfusion syndrome, laser ablation of abnormal vascular anastomoses is performed.

Amniocentesis to remove excessive amniotic fluid to relieve maternal discomfort and to reduce incidence of preterm labour can be tried. Foetal paracentesis, thoracocentesis and thoracoamniotic shunting for pleural effusion have been tried.

If the fetus has no structural or chromosomal abnormality and is mature enough to survive, delivery should be accomplished and may necessitate cesarean delivery. In most cases, however, hydrops cannot be treated and usually foetal death occurs. Vaginal delivery is allowed in such cases.

## Key Points

1. An RhD– mother can develop antibodies when she carries an Rh-positive fetus. These albumin antibodies (IgG) cross the placenta and damage the fetus.
2. Foetal cells can enter the maternal circulation during delivery or rarely even during antenatal period, justifying anti-D prophylaxis 300 µg at 28–34 weeks gestation (Grade IA recommendation). In USA and India routine anti D 300 µgm I M is given at 28 weeks of gestation while in UK and Canada two 100 µg doses are given at 28 and 34 weeks. This practice reduces the incidence of antenatal alloimmunization from 2 to 0.1%.
3. Formation of antibodies in the mother can be prevented by administration of anti-D at the time of abortion and delivery which should be given to the mother within 72 hours of delivery (Grade IA recommendation). Tests for excessive fetomaternal haemorrhage (Rosette test, Kleihauer-Betke or flow cytometry) are done to know additional dose of anti D. If anti D is inadvertently omitted after delivery, it should be given as soon as possible after recognition of omission (Grade IB). Partial protection is achieved within 13 days and there may be an effect up to 28 days. Hence, anti D can be given up to 28 days.
4. Anti-D immunoprophylaxis should also be given to Rh-negative women for miscarriage, ectopic pregnancy, medical termination of pregnancy, chorionic villus sampling, amniocentesis, cordocentesis, antepartum haemorrhage and for external cephalic version (Grade IB recommendation).
5. During antenatal period, Indirect Coomb's Test is done. A titre of 1 : 16 or more is taken positive for isoimmunization and needs further management for it.
6. Serial ultrasound is useful for seeing foetal affection and severity of the disease at 2 weeks' intervals. Foetal affection signs include skin edema, ascites, pleural and pericardial effusion and cardiomegaly and placental thickness. Doppler ultrasound and CTG are useful for foetal monitoring.

**Prognosis**

Overall outcome is poor with perinatal death rate between 40–98%. If hydrops develops before 24 weeks, mortality is 95% while if it develops after 24 weeks and has structurally normal heart, mortality is 80% with 20% survival. Risk of recurrence is low for non-genetic cases.

**Diagnosis**

1. Targetted ultrasound for congenital anomalies, arrhythmias and complications of twin pregnancies.
2. *Maternal blood tests.* Hb electrophoresis, indirect Coomb's test, Kleihauer-Betke test, serological tests for syphilis, toxoplasma, rubella, cytomegalovirus and parvo virus B-19.
3. Amniocentesis for foetal karyotyping and intrauterine infection.
4. Doppler studies for middle cerebral artery peak systolic velocity (MCA PSV) for severity of foetal anaemia
5. Cordocentesis for foetal hematocrit, infections and for intrauterine transfusion.

**Maternal complications**

1. Hydramnios
2. Preterm labour is common due to associated hydramnios
3. Placental abruption due to sudden decompression of hydramnios.
4. Maternal mirror syndrome with mother developing pre-eclampsia and edema like fetus due to vascular changes in swollen and hydropic placenta (placentomegaly)
5. Difficult delivery
6. Postpartum haemorrhage due to uterine atony

**Management**

It is individualized depending upon the etiology and after consultation with parents. Termination of pregnancy can be performed before foetal viability. For severe congenital malformations and chromosomal anomalies detected late in pregnancy beyond legal limit of MTP, expectant management is practised. For fetuses without malformations and aneuploides and with treatable conditions like tachyarrythmias or parvo virus infection, aggressive pharmacological treatment is offered. Severe foetal anaemia is treated by cordocentesis and intrauterine blood transfusion.

For twin-twin transfusion syndrome, laser ablation of abnormal vascular anastomoses is performed.

Amniocentesis to remove excessive amniotic fluid to relieve maternal discomfort and to reduce incidence of preterm labour can be tried. Foetal paracentesis, thoracocentesis and thoracoamniotic shunting for pleural effusion have been tried.

If the fetus has no structural or chromosomal abnormality and is mature enough to survive, delivery should be accomplished and may necessitate cesarean delivery. In most cases, however, hydrops cannot be treated and usually foetal death occurs. Vaginal delivery is allowed in such cases.

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5. During antenatal period, Indirect Coomb's Test is done. A titre of 1 : 16 or more is taken positive for isoimmunization and needs further management for it.
6. Serial ultrasound is useful for seeing foetal affection and severity of the disease at 2 weeks' intervals. Foetal affection signs include skin edema, ascites, pleural and pericardial effusion and cardiomegaly and placental thickness. Doppler ultrasound and CTG are useful for foetal monitoring.

7. Middle cerebral artery peak systolic velocities are periodically assessed. If elevated to > 1.5 MOM, it indicates foetal anaemia and need for cordocentesis and foetal blood transfusion if low hematocrit.
8. Alternatively, amniocentesis is performed for optic density at 450 nm to see foetal affection by plotting on Liley's graph. Affected fetus is delivered at 34 weeks. Before that if severe affection occurs (zone 3 or upper zone 2) intrauterine transfusion is recommended with O negative red blood cells, IUT can be repeated with aim to take the pregnancy up to 34 weeks when delivery is performed.
9. Affected pregnancies should be managed in a tertiary referal center with facilities for amniocentesis, cordocentesis, foetal blood transfusion and specialized neonatal care.

## Suggested Reading

1. American College of Obstetricians and Gynecologists: Prevention of Rh-D alloimmunization. Practice Bulletin No. 181, August 2017.
2. Liley A W. Liquor Analysis in Management of Pregnancy Complicated by Rhesus Sensitization, American Journal of Obstetrics and Gynecology 1961; 82–1359.
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4. Royal College of Obstetricians and Gynaecologists, London for Management of women with Red cell Antibodies during Pregnancy. Guideline Green Top Guideline no. 65, London, May 2014.
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6. American College of Obstetricians and Gynecologists. Management of Alloimmunization during pregnancy. Practice Bulletin No. 75, August 2006, Reaffirmed 2016.
7. Royal College of Obstetricians and Gynecologists, Rhesus D Prophylaxis. The Use of Anti-D Immunoglobulin. Green Top Guideline No. 22, RCOG, London, April 2011.
8. National Institute for Health and Clinical Excellence (NICE). Technology Appraisal Guidance 156. Routine Antenatal Anti D Prophylaxis for Women who are Rhesus D Negative, Nice, London 2011.
9. Crowther C A, Middleton P, McBain R D. Anti-D Administration in Pregnancy for Preventing Rhesus Alloimmunization. Cochrane Database Systematic Reviews 2013; 2: CD000020.
10. Sandler SG, Queenan JT: A guide to terminology for Rh Immunoprophylaxis. Obstet Gynecol 2017: 130 : 633.
11. Cunningham FG et al. William Obstetrics; 25th edn McGraw Hill Foundation, New York, USA, 2018, p. 300–314.
12. Mari G, Deter RL, Carpenter RL et al. Noninvasive diagnosis by Doppler ultrasonography of foetal anaemia due to maternal red cell allo-immunization N. Engl J Med 2000; 342 : 9.

## Examination Questions

**A.** How will you manage a case of Rh-negative pregnancy with ICT negative during pregnancy and labour?
**B.** How will you manage a case of Rh-negative pregnancy with ICT positive (1 : 16 titre) at 30 weeks gestation? (DU)
**C.** Write short notes on:
1. Factors deciding maternal response in Rh-negative pregnancies
2. Immune hydrops (erythroblastosis fetalis) **(DU and other universities, many times)**
3. Icterus gravis neonatorum
4. Active Rh immunization: Indications **(Many universities)**
5. Role of IUT in Rh-negative pregnancy
6. Care of newborn in Rh-negative pregnancy

CHAPTER 39

# Pregnancy with Previous Cesarean Delivery

## INTRODUCTION AND HISTORY

Management of a pregnant woman with a prior cesarean delivery is a controversial topic regarding mode of delivery due to fear of uterine rupture. Due to rising cesarean rate worldwide, obstetricians are seeing more and more pregnant women with previous cesarean delivery.

**Cragin** in 1916 had given the dictum **'once a cesarean, always a cesarean'** as those days cesarean deliveries were performed by the classical upper segment route and anaesthetic agents and antibiotics were in the primitive stage. However, most cesareans are now done by lower segment transverse incision with very low rate of rupture. Hence, trial of labour after cesarean section (TOLAC) and vaginal birth after cesarean (VBAC) is feasible and safe with availability of effective antibiotics, safe anesthesia and blood transfusion. Hence, now the dictum **'once a cesarean, always a hospital delivery'** is more appropriate.

### Effect of Cesarean delivery on Pregnancy and Labour

Although there are no major effects of previous cesarean on the course of pregnancy and labour, the following complications are slightly increased in them.

1. Miscarriages
2. Pregnancy ailments like pain
3. Premature labour
4. Increased chances of repeat cesarean delivery
5. Morbidly adherent placenta
6. Retained placenta
7. Postpartum haemorrhage
8. Increased risk of peripartum hysterectomy

### Effect of pregnancy on Scar

There are higher chances of scar rupture during a subsequent pregnancy and labour. The estimated risk of uterine rupture in women with a prior cesarean delivery with different incisions is shown in Table 39.1.

**Table 39.1: Risk of Uterine Rupture in Previous Uterine Surgeries**

| Types of Previous Surgery | Risk of Uterine Rupture |
|---|---|
| 1. Previous classical cesarean section | 2–9 % |
| 2. Previous T-shaped incision | 4–9% |
| 3. Previous hysterotomy | 4–9% |
| 4. Previous low vertical incision | 1–7% |
| 5. Previous one low transverse incision (LSCS) | 0.2–0.9% |
| 6. Multiple low transverse incisions | 0.9–1.8% |
| 7. Previous rupture in lower segment | 2% |
| 8. Previous rupture in upper segment | 9–32% |

### Healing of the uterine wound

Williams (1931) thought that uterine wound healed by regeneration of the muscle fibers and not by development of scar tissue as confirmed by no trace of prior incision or almost invisible linear scar on repeat cesarean delivery. However, Schwarz and co-authors (1938) showed that healing occurred by fibroblast proliferation and scar tissue at the uterine wound site. In case of healing by proper anatomical apposition of the uterine edges, scar tissue becomes minimal. Factors causing impaired healing and weak scar are as follows:

1. Poor anatomical apposition of uterine cut margins
2. Imperfect hemostasis
3. Wound haematoma hampers healing
4. Presence of infection
5. Poor general condition of patients (e.g. anaemic patients)
6. Overstretching of the lower uterine segment may reduce vascularity of uterine muscles
7. Incision in the upper segment
8. Repeated cesarean deliveries
9. Turbulent post operative course

**Table 39.2: Differences between Lower Segment Cesarean Scar and Classical Scar**

| | | Lower segment cesarean scar | Classical scar |
|---|---|---|---|
| 1. | Suturing | Better apposition as the neat and even cut margins allow perfect apposition without leaving any pocket | Poor apposition of the thick margins of the wound. Blood pockets are formed hindering healing. Increased chances of gutter formation on the inner surface as decidua is often included in the stitch line. |
| 2. | Healing in puerperium | Better healing due to quiescent lower segment | Imperfect due to contraction and retraction of upper segment making sutures loose with imperfect healing. |
| 3. | Stretching of wound | Stretching during future pregnancy and labour is more along the scar | The stretching is usually at right angle to the scar making it weak. |
| 4. | Placental implantation in subsequent pregnancy | Maybe over the scar but much less likely to weaken the scar by placental attachment. | Increased chances of placental implantation over the cesarean scar causing its thinning and dehiscence in subsequent pregnancy and labour. |
| 5. | Scar rupture rates | 0.2–1.5% (much lower) | 4–9% (much higher) |
| 6. | Timing of rupture | In labour | In pregnancy and labour |
| 7. | Maternal mortality in scar rupture | Much less as scar rupture and effects are limited to pelvis | Up to 5% as scar ruptures in general peritoneal cavity causing significant haemorrhage |
| 8. | Perinatal mortality in scar rupture | Much less (12.5%) | Much more (up to 75%) |

## Differences between Lower Segment Scar and Classical Scar

The lower segment scar is much stronger than the classical scar due to factors shown in the Table 39.2.

# INTEGRITY OF SCAR

## Scar of a Classical Cesarean Delivery or Hysterotomy

As shown in Tables 39.1 and 39.2, a classical cesarean or hysterotomy scar is a weak scar and is much more likely to rupture during a subsequent pregnancy and delivery. As the rupture occurs in general peritoneal cavity with significant haemorrhage, there is significantly higher maternal and perinatal mortality and morbidity. Previous classical cesarean and hysterotomy is an indication of elective cesarean section in subsequent pregnancies.

## Scar of a Lower Segment Transverse Incision Cesarean Delivery

As discussed in Table 39.2, the lower segment scar heals in a much better way and is a stronger scar than a classical scar. However, lower segment scar can also undergo dehiscence during labour and one should always be conscious of it in all such pregnancies. Various important points regarding the integrity of lower segment transverse scar depend on the following factors.

### 1. Details of Last Cesarean Delivery

(i) **Indication of previous cesarean delivery.** There is a weak scar if cesarean was done for placenta previa due to imperfect apposition of margins and thrombosis of placental sinuses. Similarly, cesarean done for prolonged and obstructed labour can cause poor scar.

(ii) **Technical difficulty at time of previous cesarean** like lateral extension of angle to involve uterine vessels or tears extending into upper vagina.

(iii) **Technique of cesarean** like one layer closure or two layer closure as some studies show that scar is weak after one layer closure. However, most other studies have observed that scar after one layer closure is equally strong.

(iv) **Type of cesarean section.** Elective or emergency

(v) **Place of cesarean section**

(vi) **Expertise of** the surgeon

(vii) **Postoperative morbidity.** History of blood transfusion, history of prolonged catheterization and history of prolonged hospital stay.

### 2. Past History of Vaginal Delivery

Past history of vaginal delivery following cesarean section though favourable for subsequent vaginal birth may weaken the scar.

### 3. Factors in Present Pregnancy

(i) **Pregnancy occurring too soon after cesarean.** It has been observed that uterus needs at least 6 months for complete involution and restoration of anatomy after a cesarean delivery. Some studies have found an increased risk (up to threefold) of scar dehiscence if interdelivery interval was less than 18 months in contrast to more than 18 months.

(ii) **Overdistension of uterus** in current pregnancy due

to hydramnios or multiple pregnancy puts stretching effect on the scar.

(iii) **Placenta previa.** Attachment of placenta over the scar or placenta previa weaken the scar.

(iv) Weight of baby

(v) Obesity

### Assessment of Scar Thickness and Integrity

**1. In the inter-conceptional period**

(i) Hysterosalpingography performed 6 months after cesarean delivery may demonstrate a wedge depression of more than 5 mm showing the defect in scar.

(ii) Hysteroscopy can also be used to see depression at scar site.

(iii) **Ultrasound.** There are no clear guidelines about scar thickness on ultrasound. A scar of less than 4 mm is usually taken as a weak scar.

**2. During pregnancy**

Ultrasound has been used to assess the thickness and integrity of scar during a subsequent pregnancy with 3.5 mm myometrial thickness at scar site being taken as normal with a low risk of uterine rupture. However, a thinner (< 3.5 mm) scar is often found with low rate of uterine rupture. Ultrasound is also useful in diagnosing placental localization especially on the scar and in diagnosing morbidly adherent placenta (placenta accreta, increta and percreta). Absence of usual subplacental sonolucent zone representing decidua basalis and Nitabuch's layer under the placenta on ultrasound favors diagnosis of adherent placenta.

## SCAR DEHISCENCE (UTERINE RUPTURE)

Scar dehiscence is silent (asymptomatic) incomplete disruption (separation) of uterine scar of previous cesarean delivery with intact peritoneum (also called **incomplete rupture** or **uterine dehiscence**).

### Uterine scar rupture

Uterine scar rupture is called **complete rupture** when all the layers of the uterine wall including the peritoneum are separated.

Women with previous cesarean section are high-risk patients, that is, they have an increased risk regardless of the mode of delivery.

### Features of Impending Scar Rupture

**Symptoms**

1. Dull aching suprapubic pain persisting in between contractions
2. Bleeding per vaginum
3. Bladder tenesmus

**Signs**

1. Tachycardia
2. Hypotension
3. Hematuria on catheterization
4. Scar tenderness
5. Ballooning of lower uterine segment
6. **Cardiotocography (CTG).** There may be increased frequency and intensity of uterine contractions. Abnormal foetal heart sounds with prolonged late or variable decelerations is often the earliest and common sign of scar rupture and should arouse suspicion.
7. Failure of progress of labour without any cause
8. Meconium stained liquor

Although suggestive of scar dehiscence, these are not conclusive proofs and one has to be careful in making diagnosis. High index of suspicion is required.

**Prognosis**

The prognosis depends upon whether it is scar dehiscence (incomplete rupture) or scar rupture (complete rupture) and whether it is in upper segment or lower segment. In low segment scar dehiscence outcome for both mother and fetus is excellent. In case of low segment scar rupture maternal death is rare and perinatal mortality is 12.5%. However, rupture in upper segment (classical cesarean delivery or hysterotomy) which may occur in pregnancy or during labour is much more hazardous. If treated on time maternal prognosis is much better but maternal mortality of up to 5% has been reported from developing countries. However, perinatal mortality is very high ranging from 50–75% with rupture and expulsion of the fetus into the peritoneal cavity. Foetal condition depends upon placental separation. In case of total placental separation compounded by maternal hypovolemia, severe foetal hypoxia, foetal death or severe foetal damage is inevitable. Immediate delivery by laparotomy at the earliest along with adequate resuscitation of mother can salvage both mother and fetus.

**Management**

Scar dehiscence and uterine scar rupture are high risk pregnancies and should have regular frequent check-ups in antenatal units in a tertiary referral center.

**Hospitalization**

1. Women with previous lower segment cesarean delivery are to be hospitalized at 38 weeks.
2. All women with previous classical cesarean, hysterotomy, weak lower segment scar due to high risk factors in previous cesarean section should be admitted at 36 weeks.
3. Emergency hospitalization
   (i) If patient goes into labour any time.
   (ii) Appearance of signs or symptoms of scar dehiscence or rupture any time during pregnancy like pain abdomen, vaginal bleeding or scar tenderness.
   (iii) Any associated obstetric or medical complication.

## PLANNING MODE OF DELIVERY

Whether to perform elective repeat cesarean delivery (ERCD) (also called elective repeat cesarean section [ERCS] in U.K.) or try for trial of labour after cesarean (TOLAC) depends upon the type of previous surgery and the indication.

For previous classical cesarean section or hysterotomy, elective cesarean section is recommended at 38 completed weeks of gestation. Though in India most elective cesarean deliveries are performed at 37 completed (38th) weeks, (2 weeks before expected date of delivery [EDD]) best perinatal outcome has been reported at 38 completed (39th) weeks. Hence, American Academy of Pediatrics and ACOG (2017) recommend elective cesarean delivery during 39th week. For previous lower segment incision, vaginal birth can be tried in selected cases provided the indication of cesarean was non-recurrent, adequate scar strength, average-sized fetus and in absence of any complicating factor.

## Factors Influencing the Mode of Delivery

1. Women with one or more previous vaginal births should be informed that previous vaginal delivery, particularly previous VBAC is the single best predictor of successful VBAC and is associated with a planned VBAC success rate of 85–90%. Previous vaginal delivery is also independently associated with a reduced risk of uterine rupture.
2. **Indication of cesarean delivery.** Elective cesarean section is performed if the indication for previous cesarean was recurrent like cephalopelvic disproportion, failure of progress of labour or dystocia. If the indication was non-recurrent like breech, foetal compromise (foetal distress), placenta previa, abruptio placentae or cord prolapse, vaginal delivery can be planned.
3. **Place of operation (facility) and intraoperative complications.** When the technical skill of the surgeon is doubtful, as in cases of cesarean section done in remote areas or if there were technical difficulties during surgery (tear, extension of scar) then vaginal delivery should not be tried.
4. **Type of scar.** The uterine scar in the upper segment necessitates repeat cesarean delivery. Only if it is a lower segment transverse scar then vaginal delivery is allowed. Lower segment vertical incision is also considered for vaginal birth after cesarean by some obstetricians.
5. History of postoperative infection leading to poorly healed scar like history of fever, foul-smelling discharge or whether the abdominal scar was resutured. In these cases, vaginal delivery is not allowed because there is a threefold increase in the risk of rupture of the uterus.
6. Stitching of uterus in one layer may make the scar weak but there is no consensus.
7. If there is a period of less than 18 months in between two pregnancies there is threefold increase in the incidence of uterine scar rupture, necessitating elective cesarean delivery.
8. If there are more than one scars or a history of previous rupture, elective cesarean is recommended. However, vaginal birth after cesarean can be tried for previous two LSCS if there has been a previous vaginal delivery as recommended recently.
9. **Miscellaneous factors:**
   (i) If there is any doubt of pelvic inadequacy it is better to perform elective cesarean delivery.
   (ii) If the age of the patient is more than 30 years, elective cesarean section is advised by some authors.
   (iii) When the estimated weight of the baby is more than 4 kg, an elective cesarean delivery is recommended by some authors.
   (iv) If there is poor Bishop score of cervix, elective cesarean is performed.
   (v) Place of delivery is important. There should be provision of an operation theatre within 20 to 30 minutes of any sign or symptoms of scar dehiscence.
   (vi) If there is a normal delivery after cesarean section, vaginal delivery is recommended.
   (vii) In case of twin pregnancy (if combined weight is 4 kg or more) and polyhydramnios, an elective cesarean delivery is preferred.
   (viii) Length of gestation beyond 40 weeks needs a cesarean section.
   (ix) Maternal obesity decreases the success of vaginal delivery.

# VAGINAL BIRTH AFTER CESAREAN (VBAC) (PRONOUNCED AS VEEBACK)

With proper case selection, about 2/3rd (60–80%) of these women will be eligible for trial of labour after cesarean (TOLAC) and of these two-thirds will deliver vaginally. A successful trial means a vaginal delivery of a live fetus without scar rupture. A failed trial means when an emergency cesarean delivery is required or there is scar rupture. Current recommendations for TOLAC and VBAC are shown in Table 39.3.

**Table 39.3: Current Recommendations for to TOLAC and vaginal birth after cesarean (VBAC)**

1. Only one previous low segment transverse cesarean delivery. VBAC can also be allowed in women with previous 2 LSCS with a vaginal delivery after counseling.
2. Clinically Pelvis should be adequate on clinical examination.
3. No other uterine scar (e.g. hysterotomy) should be there.
4. No previous uterine rupture should be there.
5. Facilities for anesthesia and cesarean for emergency cesarean delivery should be there.
6. Availability of round the clock obstetrician for monitoring of women in labour and for performing emergency delivery.
7. Proper counseling of women about advantages, disadvantages and contraindications of vaginal delivery, risk of uterine rupture, perinatal mortality and neonatal neurological injury should be done.

## Contraindications for vaginal birth after cesarean (Indications of elective cesarean delivery)

1. Previous classical incision
2. Previous inverted T incision or extension of uterine incision

3. Suspicion of cephalopelvic disproportion (CPD) or contracted pelvis
4. Previous two LSCS without a vaginal delivery
5. Malpresentations
6. Medical or obstetric complications (e.g. placenta previa)
7. Multiple pregnancy was considered a contraindication in the past as operative manipulations may be needed. However, it is no longer a contraindication as recent studies have found low rupture rate (0.7–1.1%) and high vaginal delivery rates (75–85%) in them.
8. Patient's refusal to undergo trial of labour.
9. Previous uterine surgeries - especially in patient who underwent laparoscopic or abdominal myomectomy - where uterine cavity has been breached.

## Management of Labour for vaginal birth after cesarean: Trial of Labour

Informed consent of woman should be taken after discussing the risks and benefits of a trial of labour versus a repeat cesarean delivery as follows:

- Women should be made aware that successful VBAC has fewest complications.
- Women should be made aware that the greatest risk of adverse outcome occurs in a trial of VBAC resulting in emergency cesarean delivery.
- Women should be informed that planned VBAC is associated with 0.5% risk of uterine rupture.
- Women should be informed that elective repeat cesarean section (ERCS) or elective repeat cesarean delivery (ERCD) is associated with small increased risk of placenta previa/accreta in future pregnancy and of pelvic adhesions complicating any future abdominopelvic surgery.

### Monitoring

Trial of labour (TOLAC) should only be conducted in a well-equipped hospital with all facilities for doing an emergency cesarean section. Proper counselling of women is a must in explaining them advantages of vaginal delivery, contraindications of labour, risk of uterine rupture (0.5%) and risk of foetal death and foetal neurological injury with uterine rupture. Ideally, spontaneous onset of labour is awaited. An intravenous line should be set up. Crossmatched blood should be kept ready. The limited data on external cephalic version for breech presentation with previous cesarean delivery suggests that it is successful. Maternal blood pressure and pulse should be recorded half hourly before the active phase has begun and then every 15 minutes in active phase. Labour monitoring for scar dehiscence and for foetal surveillance (preferably electronically) is performed. Epidural analgesia is not contraindicated in a planned VBAC. Although an increased requirement for pain relief in labour should raise awareness of the possibility of an impending rupture (RCOG 2015). Infact ACOG (2017) recommends use of epidural analgesia in TOLAC and VBAC.

1. Induction of labour using mechanical methods (amniotomy or Foley's catheter) is associated with a lower risk of scar rupture compared with induction using prostaglandins.
2. Oxytocin is allowed for augmentation of labour (maximum dose 20–30 mu/minute).
3. Prostaglandins are used with caution. Prostaglandin $E_2$ gel can be used but $PGE_1$ (misoprostol) should be avoided due to risk of uterine rupture.
4. The pediatrician (neonatologist) should be available at the time of delivery.
5. Any demonstration of hyperactivity of uterus (increased frequency or intensity) or abnormality of foetal heart sound (prolonged, late or variable decelerations) on cardiotocography must alert the obstetrician. If in this case the cervix is not fully dilated, a cesarean section should be performed immediately (within 30 minutes).
6. Some authors measure intrauterine pressure by monitoring with a catheter (if available). Any loss of intrauterine pressure is an indication of uterine rupture and immediate laparotomy is indicated. Forceps or ventouse to cut short the second stage of labour can be used but not always required.
7. Intrauterine exploration of the uterine scar after vaginal delivery is not routinely performed nor indicated. However, if there is active bleeding, laparotomy is advised. Otherwise, close observation of vital signs and serial hematocrit determination is needed. Peripartum hysterectomy may be required (see Chapter 42[v] for details).

## Risks of Vaginal Birth After Cesarean (VBAC)

1. Unsuccessful. Needing cesarean delivery (15–20%)
2. Risk of scar dehiscence and rupture (about 0.5%)
3. Maternal morbidity and mortality is not usually increased, but there is slightly more morbidity with scar rupture.
4. Perinatal morbidity and mortality is increased if there is scar rupture. There is risk of foetal death and severe neurological injury including hypoxic ischemic encephalopathy (HIE) in uterine rupture.

## Sterilization

Patients are counselled for tubal ligation at the time of cesarean section.

## Multiple Repeat Cesarean Deliveries

These women require repeat cesarean deliveries. They are at increased risk of complications like bladder injury, haemorrhage, bowel injury, hysterectomy, need of admission to intensive care unit, longer hospital stay, bowel obstruction and pelvic pain.

## Key Points

1. A pregnancy following cesarean section is a high-risk pregnancy as it causes a higher risk of rupture of the cesarean scar during labour.
2. A detailed history and examination of notes of previous cesarean delivery should be made regarding the indication of previous cesarean, whether done in labour, any cephalopelvic disproportion, cesarean in second stage, postoperative period, healing of scar and any complication during the previous cesarean delivery should be asked.
3. Trial of labour after cesarean section (TOLAC) and Vaginal birth after cesarean (VBAC) is a valid option after a previous lower segment cesarean section for non-recurrent indication. Proper counselling of woman about advantages of vaginal birth after cesarean, contraindications, risks, uterine rupture, perinatal mortality and neonatal injury is must.
4. However, elective repeat cesarean delivery (ERCD) should be done for previous classical cesarean section, previous two cesareans (without vaginal delivery), previous inverted T incision or extension of incision and previous cesarean for contracted pelvis at 39th week.
5. Repeated cesarean sections carry increased risk of placenta previa and morbidly adherent placenta with significant maternal morbidity and mortality.
6. The risk of scar dehiscence or rupture in labour after a single cesarean section is approximately 0.5% and is usually higher after more than one cesarean section, hence elective cesarean is offered. However, vaginal birth after cesarean can be tried with previous 2 low segment transverse incisions with a vaginal delivery.
7. Induction of labour in previous cesarean section can be done with caution with Foley's catheter or with oxytocin or prostaglandin $E_2$ gel but misoprostol is avoided.
8. Careful vigilance for scar dehiscence and scar rupture should be kept during labour in vaginal birth after cesarean. Active management of the third stage of labour should be done.
9. Morbidly adherent placenta should be considered for anteriorly placed placenta.

## Suggested Reading

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## Examination Questions

**A.** Enumerate the differences between scar of classical cesarean section and low segment cesarean section. How will you manage a case of 37 weeks pregnancy with previous low segment cesarean section in labour?

**B.** Write short notes on:
   1. Vaginal birth after cesarean (Many Universities)
   2. Scar dehiscence (Delhi University)
   3. Trial of labour after cesarean (TOLAC) (Many Universities)

CHAPTER 40

# Bad Obstetric History and Other Miscellaneous Conditions

## BAD OBSTETRIC HISTORY

### Definition

A pregnant woman is said to have bad obstetric history (BOH) when her obstetric present and future outcomes are likely to be modified or adversely affected by the past obstetric events and complications.

### Causes

**1. Recurrent early pregnancy losses**

**2. Mid-pregnancy loss**

(*i*) Cervical incompetence
(*ii*) Uterine anomalies
(*iii*) Maternal infections—Syphilis, malaria
(*iv*) Endocrinal—Hypo or hyperthyroidism

**3. Late pregnancy loss**

(*i*) Preterm labour with premature rupture of membranes (PROM), foetal growth restriction or multiple pregnancy with foetal loss
(*ii*) Severe anaemia
(*iii*) Pre-eclampsia
(*iv*) Hyperhomocysteinemia
(*v*) Maternal infections—Syphilis, malaria, high fever
(*vi*) Maternal endocrinal defects—Diabetes mellitus
(*vii*) Severe Rh-isoimmunization
(*viii*) Genetic defects
  (a) Chromosomal abnormalities—Down's syndrome, Turner's syndrome
  (b) Gene disorders—Thalassemia, hemophilia
  (c) Polygenic inheritance—Anencephaly, spina bifida, hydrocephalus, congenital heart disease
(*ix*) Immunological factors like antiphospholipid antibodies and anti SLE antibodies
(*x*) Intrahepatic cholestasis of pregnancy
(*xi*) Contracted pelvis and cephalopelvic disproportion (CPD)

**4. Idiopathic**

In number of cases, no cause of abortion or stillbirth can be ascertained.

### Management

The main aim is to elucidate the cause of mishap if possible and to treat it by timely detection and treatment if possible.

**History taking**

1. History of complications in previous pregnancies is very important, e.g. bleeding, medical disorders like high blood pressure, diabetes or Rh incompatibility.
2. Duration of labour, any history of precipitate or prolonged labour, maternal injury, any history of retained placenta or manual removal of placenta. Previous mode of delivery, instrumental delivery or cesarean section, its indication, procedural complications and postoperative complications are important points to be asked in history.
3. Weight and condition of the newborn such as prematurity or any injury to the newborn. In case of stillbirth, detailed history is crucial whether it was a macerated stillbirth or fresh stillbirth, whether intrauterine death occurred before labour or it was an intrapartum death with difficult or prolonged labour, any intervention and details of autopsy if performed. In case of neonatal death, details like weight of the neonate, duration of gestation, any deformity, etc., are to be elicited. Any history of congenital abnormalities, any abnormal karyotyping and use of folic acid in periconceptional and perinatal period are asked.

**Investigations**

1. Complete hemogram
2. Urine complete and culture
3. VDRL
4. ABO Rh

5. Glucose tolerance test in second trimester and repeated at 32–34 weeks
6. Lupus anticoagulant and anticardiolipin antibodies to rule out antiphospholipid syndrome (APLS) (also called antiphospholipid antibodies syndrome [APLA]) as it is associated with complications like recurrent foetal loss, foetal growth restriction, pre-eclampsia, placental abruption, recurrent thrombotic events and thrombocytopenia due to vasoconstriction and thrombosis in placental vessels.
7. HBs Ag, HIV testing
8. Ultrasound scan and Doppler for foetal well-being
9. Thyroid function tests, if clinically indicated
10. TORCH test, if clinically indicated

**Treatment**

Treatment for bad obstetric history is by rectifying or treating the basic cause of bad obstetric history. Depending upon the cause of obstetric mishap, prevention and treatment is accordingly offered. Prepregnancy and periconceptional counselling is very useful especially in medical disorders of pregnancy. Thus, a woman with serious heart disease should undergo cardiac surgery (valve replacement or corrective surgery for congenital heart disease) before she plans pregnancy for optimum outcome. Similarly, a woman with renal condiiton, hypertension, epilepsy or other medical disorders should get her health and medical conditions optimized before planning pregnancy and should be on safer medication and avoid medicines which can harm the fetus (e.g. ACE inhibitors for hypertension must be stopped and replaced with safer medicines). All women should be on folic acid therapy to prevent neural tube defects (400 µg) for low-risk and 4–5 mg for high-risk cases.

In case of recurrent second trimester miscarriages or preterm deliveries due to proven cervical insufficiency (incompetence) and confirmed by ultrasound scan, McDonald stitch (cervical cerclage) should be performed at about 13 weeks of gestation.

Women with heart disease should be seen jointly by obstetrician and cardiologist in all visits to ensure optimum management. Similarly, women with diabetes mellitus should be optimally controlled by obstetrician and endocrinologist to maintain euglycemia in periconceptional period and throughout pregnancy for optimum maternal and perinatal outcomes. Women with medical disorder like nephritis, hepatic diseases, epilepsy, thyroid disorders should be jointly seen by obstetrician and concerned physician for appropriate medical management for optimum maternal and perinatal outcomes. Women with Rh isoimmunization should be referred to a tertiary referral center with facilities to manage such pregnancies (foetal medicine, middle cerebral artery Doppler studies, in utero foetal intravascular transfusion) (see Chapter 38 for details).

If VDRL test is positive, patient is treated with penicillin therapy (see Chapter 36[v] for details). Intrahepatic cholestasis of pregnancy is treated with ursodeoxycholic acid therapy and induction of labour at 37 completed weeks of pregnancy.

Women found to have antiphospholipid antibody syndrome (ALPA or APLS) should be started with unfractionated heparin in dose of 5000–7500 units subcutaneously twice daily till term alongwith 75 mg of aspirin daily from diagnosis of pregnancy till 36 weeks. Instead of unfractionated heparin 40–60 mg low molecular weight heparin once a day can also be given (see chapters 13 and 35[IX] for details).

As there is risk of recurrence of the mishap, such pregnancies should be regularly and carefully monitored by more frequent antenatal clinic visits. Ultrasound should be done every four weeks for foetal growth and well-being.

Assessment of foetal well-being is done by non-stress testing (twice weekly), biophysical scoring (once weekly) (see Chapter 15 for details). Doppler studies on umbilical vessels, uterine vessels, ductus venosus and middle cerebral arteries may be helpful to find out foetal jeopardy, which, if detected, may necessitate early delivery.

**Care During Labour**

Timely delivery usually by cesarean section is required. Inducing labour at least one week prior to the period at which previous mishap had occurred helps in better survival of the baby, except in severe prematurity. It is a judious indication for continuous electronic foetal monitoring during labour.

## TEENAGE PREGNANCY

### Definition

Teenage pregnancy is also called adolescent pregnancy, occurring in teenagers from puberty to 19 years of age. Pregnancy is possible before the start of menstrual periods (menarche), with the first sexual experience and even without penetration.

### Incidence

Teenage pregnancy birth rate is 22.3% in 1000 women in USA (2015) with 78% teen pregnancies being unplanned. In India, early marriage is very common leading to a higher incidence which is about 18–20% with majority being in married women. Adolescent deliveries in hospitals are 4–6%.

### Risk factors for teenage pregnancy

1. Early marriage
2. Poor socio-economic status
3. Exposure to domestic or sexual violence
4. Social isolation
5. Low academic interest and achievement
6. Stress and depression
7. Trouble in school or with the in-laws
8. Limited job opportunities
9. A previous unplanned teenage pregnancy
10. Single-parent homes
11. Drug addiction like smoking, alcohol and substance abuse
12. Non-use of contraceptives

### Prevention of unplanned teenage pregnancies and sexually transmitted infections

1. **School sexual education.** The components of sexual education include excellent sexual information at school, the right to discuss and availability of safe and successful contraceptive methods including emergency contraception and promotion of responsible sexual behaviour.
2. **Promotion of abstinence.** Inculcation of moral values in adolescent girls and educating them about character and monogamous relationship after marriage can help prevent unintended pregnancies as well as sexually transmitted infections.
3. **Avoidance of early marriage.** Use of counselling and using legal means can reduce teenage pregnancies before 18 years.
4. **Empowerment of women.** By increasing female literacy and social awareness as has happened in the state of Kerala.
5. Encourage use of LARC (Long Acting Reversible Contraception) like IUDs, implants.

## Diagnosis

Any teenage woman who presents to a healthcare provider's office with irregular or absent periods should undergo a urine pregnancy test. This should be done even if she refuses history of coitus. An early diagnosis of pregnancy helps the teenager to adjust emotionally and physically. Early prenatal care is essential. Single motherhood is slowly coming up in our society also.

## Complications

### Maternal

There are more chances of the following maternal complications.

1. Abortion
2. Anaemia
3. Malnutrition
4. Pre-eclampsia and Eclampsia
5. Preterm labour is seen in up to 21% cases in teenage pregnancy as compared with 9% in general population.
6. Lower genital tract infections (which may lead to premature labour and delivery)
7. Psychological problems like severe mental and emotional trauma, depression and suicidal tendencies due to unplanned pregnancy and inadequate family support. They are also more prone to puerperal depression and psychosis.
8. Difficult delivery in small pelvis
9. Cesarean section
10. Foetal distress
11. Puerperal endometritis

### Foetal

Infants born to teenage mothers are at a higher risk of:

1. Problems due to prematurity
2. Sudden infant death syndrome
3. Low birth-weight because of prematurity, poor eating habits, smoking and poor antenatal care.
4. Lack of immunization
5. Developmental delays
6. Behaviour problems
7. Infections
8. Exposure to domestic violence
9. Accidental trauma and poisoning
10. Poverty

### Long-term effects

The long-term effects of teenage pregnancy are far reaching. They are as follows:

1. Continued poverty and welfare dependency
2. Social isolation
3. Low level of education
4. Limited job opportunities
5. Depression
6. Further unwanted pregnancies
7. Exposure to domestic violence
8. Child behavioural problems
9. Poor parenting skill
10. Substance abuse
11. Medical problems such as pelvic inflammatory disease, ectopic pregnancy and cervical cancer

## Management

The adolescent girls may not seek abortion until too late due to ignorance and fear or may go to untrained persons for abortion. Hence, criminal and septic abortions are more common among teenagers.

Medical termination of pregnancy services should be made easily available with trained doctors and counsellors should be available to help her make a decision and provide all necessary help and support. She should also be given medication to prevent sexually transmitted infections (see Chapter 36[v] for details).

If her pregnancy has progressed beyond the legal limits of performing MTP (20 weeks), she should be followed up regularly in antenatal clinic. She should have all the necessary investigations as given in Chapter 11. She should be given regular iron and folic acid and calcium tablets and should be adviced about dietary allowances like adequate calorie intake (2000–2500), proteins (60–75 g/day) and green leafy vegetables. Serial ultrasound scan should be done for foetal growth and later foetal monitoring should be performed by foetal kick chart, non-stress test and biophysical tests as given in Chapter 15. Social workers and social organizations should be involved in her care and future options like giving the baby for adoption should be discussed with the girl and her family. Delivery should be conducted in hospital. Baby should be resuscitated by pediatrician. Contraception must be offered during puerperium.

# GRAND MULTIPARITY

## Definition

**It is defined as a pregnant woman who has given birth to four or more viable babies in the past (parity 4 or more) (NICE guidelines 2007).**

The term extreme grand multiparity is used for mothers who have had 10 or more prior viable pregnancies.

## Incidence

About 8% admitted patients in hospitals in India are grand multiparas but they are responsible for about 30% of maternal mortality especially in non-industrialized nations. However, it is decreasing due to acceptance of contraceptives and smaller families by choice.

## Complications

### Antepartum complications

1. Miscarriages
2. Chromosomal anomalies, like Trisomy 21, with increasing maternal age.
3. Obstetric problems:
   (a) Malpresentations and malpositions due to laxity of abdominal wall and high pelvic inclination
   (b) Placenta previa
   (c) Abruptio placentae
   (d) Twin and high order pregnancies
   (e) Preterm labour
4. Medical disorders:
   (a) Increased incidence of iron deficiency and folate deficiency anaemia due to repeated child births without adequate spacing and replenishment of iron and folic acid stores.
   (b) Hypertension and pre-eclampsia
   (c) Cardiovascular defects due to advanced age
   (d) Renal disease
   (e) Impaired glucose tolerance and diabetes
   (f) Varicose veins, piles, venous thrombosis and hiatus hernia.

### Intrapartum complications

There is an increased incidence of:

1. Cephalopelvic disproportion due to:
   a. Macrosomic fetus
   b. Contracted pelvis due to osteomalacia or tuberculosis of pelvis or spondylolisthesis.
2. Malpresentations and malposition
3. Prolonged labour
4. Obstructed labour
5. Rupture uterus due to obstructed labour
6. Cord prolapse
7. Increased operative interference
8. Shock due to excessive bleeding and rupture uterus.

### Postpartum complications

1. Postpartum haemorrhage due to uterine atony, increased operative interference and adherent placenta.
2. Retained placenta
3. Uterine inversion
4. Subinvolution of uterus
5. Failure of lactation

## Management

National Institute of Clinical Excellence (2007) guidelines consider grand multiparas (parity 4 or more) as high risk pregnancies and as a risk factor for PPH and has recommended that they should be advised to give birth in an obstetric unit where emergency treatment options are available (hospital delivery). Anaemia should be corrected. Any medical problem should be controlled. They should be properly assessed by pelvic assessment and ultrasound at about 37 weeks. Elective cesarean is justifiable for any malpresentation or cephalopelvic disproportion.

### Labour management

If pelvis is adequate and malpresentation and malposition is excluded then labour can be tried under careful watch. Delivery should be monitored closely using a partogram. Labour usually progresses quickly in them and any prolonged labour should be taken seriously as uterus of a multiparous woman may rupture unlike primigravidas whose uterus goes into inertia. Difficult vaginal delivery and forceps deliveries are to be avoided. Oxytocin should be used with caution during labour. Blood should be crossmatched and kept ready in the eventuality of any postpartum haemorrhage. Active management of the third stage with oxytocin is recommended. After delivery oxytocin drip can be started to prevent and treat PPH.

### Postpartum

Lactation should be encouraged. Advice about limiting the family should be given. Permanent method of contraception, like postpartum sterlization, is recommended.

# PREGNANCY AFTER 35 YEARS

In the past, the term used was **elderly gravida** to arbitrarily define women over 35 years which was insensitive and is better avoided.

## Incidence

About 10% of all pregnancies occur in women in this age group. It is likely to increase in future due to increased emphasis on women's education, late marriages and more priority to career with tendency to postpone the pregnancies.

Liberal use of assisted reproductive techniques including in vitro fertilization and embryo transfer (IVF ET) and ovum donation have further increased the chance of pregnancies in later ages including in menopausal women.

There are two groups of women.

1. Woman with high fecundity, who married late but conceived soon after.
2. Woman with low fecundity who married early but conceived long after marriage. The second group is more unfavourable as far as obstetric outcome is concerned.

## Complications

### I. Maternal

#### Antepartum

1. Miscarriage
2. Trisomy 21 or Down's syndrome
3. Other chromosomal defects and congenital anomalies

4. Medical problems are more common in elderly patients.
   (*i*) Obesity
   (*ii*) Chronic hypertension
   (*iii*) Overt diabetes
   (*iv*) Impaired glucose tolerance and gestational diabetes.
   (*v*) Pre-existing cardiac problems may cause more problems in pregnancy with older age.
5. Obstetric problems are more common in elderly patients.
   (*i*) Antepartum haemorrhage (placenta previa and abruptio placentae)
   (*ii*) Preterm labour may be seen in up to 19% women (spontaneous and iatrogenic due to medical complications).
   (*iii*) Premature rupture of membranes.
   (*iv*) Post term pregnancies
   (*v*) Multiple pregnancies, especially dizygotic twins (mainly due to infertility treatment)
   (*vi*) More chance of gynecological abnormalities like fibroids and ovarian cysts.
   (*vii*) Foetal growth restriction
   (*viii*) Malpresentations
   (*ix*) Pre-eclampsia

INTRAPARTUM

There is an increased incidence of:

1. Dysfunctional and prolonged labour due to uterine inertia and malpositions like occipito-posterior position.
2. Failed induction with higher oxytocin use.
3. Increased instrumental vaginal delivery rate.
4. Maternal and foetal distress
5. Rigid perineum requiring episiotomy
6. Increased cesarean section rate, i.e. **31.3% vs 13.5%** in general population. The higher rate of cesarean section is due to higher rate of obstetric complications such as pre-eclampsia, malpresentations and prolonged labour.
7. Retained placenta

POSTPARTUM

1. Higher rates of operative delivery
2. Failure of lactation

### II. Perinatal

1. Increased neonatal morbidity (18%)
2. Increased incidence of small for date infants (11%)
3. Increased stillbirths
4. Increased congenital malformations (12% vs 1.28%) (Down's syndrome and other anomalies)

### Management

Pregnancy in women after 35 years is considered high risk due to the various risks given above and need more careful vigilance during antepartum and intrapartum periods.

#### 1. Preconceptional Counselling

Preconceptional counselling is very important in this age group and many women willingly seek it. This is very important for women with pre-existing medical problems in which case pregnancy can be planned after stabilizing the medical condition.

#### 2. Prenatal Diagnosis

Prenatal diagnosis is extremely important considering the increased chance of chromosomal defects. Serum screening and ultrasound screening for Down's syndrome is recommended. Also a targeted anomaly scan at 18–20 weeks is a must. If necessary, chorion villus sampling or amniocentesis may have to be performed to reliably rule out chromosomal anomalies as serum screening may miss up to 30–40% cases of Down's syndrome and other chromosomal anomalies.

#### 3. Antepartum Management

More frequent antenatal visits with antepartum foetal surveillance with serial ultrasound and Doppler. Opinion of a physician may be necessary, if there are associated medical complications.

#### 4. Intrapartum Management

Close monitoring is indicated in labour. Hospital delivery preferably in a tertiary center with good neonatal care facilities is a must. CTG monitoring is ideal. Judicious monitoring of labour is required. A lower threshold for cesarean section is preferred in these women and in many cases, elective cesarean section may be ideal. The newborn should be taken care of by an expert neonatologist.

#### 5. Postpartum Management

Pregnant women who are more than 35 years need better care to avoid postpartum complications and failing lactation. They also need contraception advice.

## OBESITY DURING PREGNANCY

Obesity is defined as 20% increase in body weight. Some consider a weight of more than 90 kg as obesity. Obesity is becoming a major problem throughout the world with 33% adults being overweight and 2.8% women having extreme obesity in USA. It is observed more in diabetic women and women with polycystic ovary syndrome who conceived following ovulation induction. Obesity is often associated with metabolic syndromes like type 2 diabetes, dyslipidemia and hypertension.

**The body mass index (BMI)** also known as **Quetelet index** is calculated as pre-pregnancy weight in kilograms divided by height in meter square ($kg/m^2$). Hence, pre-pregnancy weight must be known to calculate BMI accurately.

1. **Normal.** 18.5 to 24.9 $kg/m^2$
2. **Overweight.** 25 to 29.9 $kg/m^2$
3. **Obesity:** 30 or more $kg/m^2$

   **Obesity.** It can be further classified as:
   (*i*) Class I or mild obesity: BMI 30–34.9 $kg/m^2$
   (*ii*) Class II or moderate obesity: BMI 35–39.9 $kg/m^2$
   (*iii*) Class III or extreme obesity: BMI 40+ $kg/m^2$

Preconceptional counselling: All women of child bearing age with BMI of 30 kg/m$^2$ or more should be advised to lose weight before conception and between pregnancies as it reduces risk of stillbirth, hypertensive complications and foetal macrosomia and chances of successful vaginal birth after cesarean (VBAC) delivery. They should take 5 mg folic acid starting 1 month before conception and in first trimester of pregnancy. However, the women should not try to lose weight in pregnancy or take antiobesity durgs in pregnancy. The hospitals should have facilities to look after obese patients including beds, OT tables, BP cuffs, large wheel chairs, large patient gowns and other equipment. They are high risk pregnancies and need to be attended to in tertiary referal contest. They should be regularly screened for hypertensive disorders, foetal macrosomia and other complications. Iron and calcium with vitamin D supplementation is given. Thromboprophylaxis should be given if cesarean delivery is needed for macrosomia, routine induction of labour at 38 weeks to decrease chances of shoulder dystocia or cesarean births. They should be seen by anesthetist during pregnancy for plan of anesthesia if it is needed. Active management of third stage is recommend. Induction of labour can be done at term.

## Complications

Obesity is often associated with subfertility due to insulin resistance. Marked obesity is hazardous to the mother and fetus. However, weight loss is not recommended in pregnancy but she should limit weight gain.

### Antepartum

1. Increased chances of miscarriages, which rise with increase in BMI.
2. Structural anomalies like neural tube defects are increased.
3. Triple test for Down's syndrome is less reliable in these patients.
4. Increased medical complications like diabetes, hypertension and pre-eclampsia.
5. Increased obesity comorbidities like cholelithiasis, obstructive sleep apnea, fatty liver disease and ischemic heart disease.
6. Intrauterine foetal death due to foetal hypoxia and medical problems.
7. Difficult abdominal palpation and auscultation due to obesity.
8. Increased requirement of frequent ultrasound scans.
9. Post maturity is more common.

### Intrapartum

Obese women are high-risk cases and require hospital delivery.

1. Higher chances of abnormal uterine action and dysfunctional labour.
2. Higher chances of operative delivery and cesarean section.
3. Foetal macrosomia may be associated in obese women with diabetes.
4. Shoulder dystocia should always be anticipated and managed properly.
5. In hypertensive mothers, foetal growth restriction can occur.
6. Anaesthetic complications are more common and intubation may be difficult if the neck is short.
7. Cesarean section is much more difficult to do. Pfannensteil incision and meticulous hemostasis are recommended. There are more complications.
8. Increased risk of post-partum haemorrhage.
9. Increased risk of pelvic infection.
10. Increased risk of wound infection or complication.
11. There is increased risk of stillbirths and perinatal mortality.
12. Increased chances of obesity, diabetes, hypertension and metabolic syndrome in adulthood in children born to obese women. (Barker's hypothesis) (see Chapter 34 for details)

### Postpartum

There may be more chance of deep vein thrombosis and pulmonary embolism especially after cesarean section. Graduated compression stockings, hydration and early mobilization are recommended for cesarean section to prevent thromboembolism. Prophylactic heparin is to be considered. Wound infection may be a complication. Lactation failure is also more common.

## Pregnancy following surgical procedures for obesity

Surgical procedures for obesity are not recommended during pregnancy. However, many women who underwent gastric bypass surgery, gastroplasty (vertical-banded gastroplasty or laparoscopic adjustable gastric banding) or Roux-en-Y-gastric bypass surgery in the past may have pregnancy. The women are advised to delay pregnancy for 12–18 months after surgery during the rapid weight loss phase. The women who underwent gastric band may need adjustment of band during pregnancy. They should be followed up regularly in antenatal clinic and should be evaluated for nutritional deficiencies. Also vitamin supplementation (B12, D, Folic acid and calcium supplement) should be given to them (ACOG 2015). They can have normal delivery.

# HIGH RISK PREGNANCY

## Definition

High risk pregnancy is defined as a pregnancy in which the mother, fetus or newborn are at increased risk of morbidity or mortality during pregnancy, labour or puerperium due to some complicating factors.

## Incidence

About 20% of all pregnancies are high risk. But they are responsible for 50% of all maternal complications, 60% of all primary cesarean deliveries and 70–80% of perinatal mortality and morbidity.

## Screening of pregnancies for high-risk cases

Obstetricians have to be vigilant to screen all high-risk pregnancies from start of pregnancy to avoid any mishaps.

### Factors on history taking

1. **Maternal age.** Below 16 years or above 35 years particularly if the patient is a primigravida.
2. **Past obstetric history.** Previous congenital malformation, macroscopic baby, repeated abortion, repeated preterm labour, small for date fetus, stillbirth or neonatal death, operative delivery like cesarean or forceps or ventouse delivery.
3. **Grand multiparity.** Five or more previous deliveries.
4. **Previous or current medical disorders.** Anaemia, hypertension, diabetes, cardiac, renal, pulmonary and hepatic disease, coagulation defects, hemoglobinopathies, serious infections such as HIV.
5. **Previous or current obstetric disorders.** Antepartum haemorrhage, Rhesus isoimmunizaiton, intrauterine growth restriction, premature rupture of membranes, polyhydramnios, oligohydramnios, postterm pregnancy.
6. **History of surgery or trauma.** Myomectomy, metroplasty, repair of third degree perineal tear, VVF repair and surgery for stress urinary incontinence
7. **Family history** of medical disorder, genetic disorders, multiple pregnancies and poor socio-economic status.

### Factors on examination

1. Short stature (< 145 cm)
2. Underweight
3. Obese (BMI > 30 kg/m$^2$)
4. Hypertension
5. Anaemia
6. Any obvious cardiac, respiratory, orthopedic or neurological disease
7. Uterine enlargement too much or too less.
8. Associated pelvic masses like fibroids or ovarian cysts
9. Associated genital prolapse
10. Old cervical tear or dilated cervix (cervical incompetence)
11. Contracted pelvis

### Factors in current pregnancy

1. Anaemia and other medical disorders
2. Multiple pregnancy
3. Preterm labour and premature rupture of membranes
4. Abnormal presentations and positions
5. Contracted pelvis

### Factors in labour

1. Induction or augmentation of labour
2. Abnormal foetal heart rate
3. Prolonged or obstructed labour
4. Meconium stained liquor
5. Chorioamnionitis
6. Rupture uterus
7. Non-reassuring foetal status (foetal distress)
8. General anesthesia
9. Breech delivery
10. Instrumental delivery

### Postpartum complications

1. Postpartum haemorrhage
2. Retained placenta
3. Shock
4. Inversion
5. Sepsis

### Neonatal complications

1. Asphyxia (poor Apgar)
2. Foetal growth restriction
3. Macrosomia
4. Neonatal seizures
5. Respiratory distress syndrome
6. Metabolic disorders
7. Sepsis
8. Cyanosis
9. Anaemia
10. Congenital malformations
11. Neonatal hyperbilirubinemia
12. Hemorrhagic disease of newborn

## Management

**Pre-conceptional counselling** is important to diagnose high risk factors from history, investigate before pregnancy so that they conceive when their basic medical conditions are under control (e.g. diabetes, epilepsy).

Folic acid (4–5mg/day) therapy should be started in periconceptional period and is continued up to 12 weeks of pregnancy. All women should be properly examined and investigated throughly to pick up any high risk factor. Proper advice for diet, activities, rest and medicines is given.

### Antenatal

Patients with history of previous first trimester abortion should be advised rest and should avoid sexual intercourse. Vaginal examination should be avoided in the first trimester in these cases. Patients with cervical incompetence confirmed on ultrasonography should undergo cervical cerclage operation in the second trimester. Patients having premature labour, unexplained stillbirth, intrauterine growth restriction and many other abnormalities are benefitted by prolonged rest in hospital with close supervision. They should be monitored by antepartum foetal monitoring with non-stress test, Manning score and ultrasound for foetal growth and well-being.

### Labour

Elective cesarean delivery for judicious indications (e.g. contracted pelvis, placenta previa) at 38 weeks of gestation. Most women with high risk factors like diabetes mellitus, nephritis, hypertension and intrahepatic cholestasis usually need induction of labour at 38 (37 completed) weeks of gestation. Close monitoring for the assessment of progress of labour or for any evidence of foetal distress should be done during labour.

It is preferable to monitor fetus during labour by continuous electronic foetal monitoring if possible for all high risk pregnancies.

Cesarean delivery should be performed for non-progress or poor progress of labour or for any non-reassuring foetal status. Such women should be delivered in a hospital with facilities of intensive neonatal care unit and the neonate should be assessed and cared by expert neonatologist or pediatrician.

## Obstetric intensive care and high dependency care

As various pathophysiological conditions can occur during pregnancy, labour and puerperium, some women may need intensive care for cardiovascular or respiratory support. The pregnant women being young and in good health should have better prognosis than many other patients in intensive care unit (ICU). The levels of care are as follows:

1. **Intensive Care Unit (also called medical or surgical ICU):** Most tertiary hospitals have these units operated by medical or surgical intensivists. In many hospitals ICUs are under anesthesiology department, outside the main operation theatre and are managed by anesthesiologists with help from pulmonologists, cardiologists and other medical and surgical specialist. Women needing ventilatory support, intensive monitoring or pharmacological support of circulation are transferred to ICU. Although care is good, there is always a paucity of beds in the ICU, risk of hospital acquired infections (e.g. methicillin resistant Staphylococcus aureus) is high. Only very sick women are transferred to these units.
2. **Obstetric High Dependency Care Units (HDUs).** They are also called **obstetric intermediate care units**. These units are situated within the labour and delivery suites in specialized areas. They cater to an intermediate level of care between a general ward and an ICU. Care is provided by a specially trained maternal-foetal medicine specialist (or an obstetrician with special training and interest in ICU) and specialist nurses with experience and training in critical care obstetrics. The team should include other obstetricians, anesthesiologists and readily available pulmonologists (chest physicians), cardiologists and other medical and surgical specialists. Obstetric HDU can reduce the load on ICU and is a step down unit to an ICU and is much more economical. Besides, the obstetricians can continue to look after their own patients. More sick women which cannot be handled in HDU should be shifted to main ICU. Obstetric HDUs are common in USA and most UK hospitals and should be started in India also for better obstetric care. There should be nurse-patient ratio of 1 : 2 and medical staff should be available round-the-clock. Each hospital should develop their own protocol for admission and care in HDU and should audit the care given. The record keeping should be high quality (preferably computerized) and regular training and education of staff is essential.

### Indications of admission to HDU

1. Hemodynamic instability due to obstetric haemorrhage (antepartum and postpartum haemorrhage, ruptured ectopic and coagulopathy [DIC])
2. Respiratory failure due to fluid overload, pulmonary edema and congestive cardiac failure.
3. Severe pre-eclampsia, eclampsia and HELLP syndrome
4. Severe infections (post operative sepsis, puerperal sepsis or septic shock)
5. Severe medical conditions like heart disease (grade 3 or 4), severe hepatic disease, renal failure, etc.

Maternal management includes round-the-clock monitoring of vital signs (using monitor), heart rate, blood pressure, mean arterial pressure and respiratory rate, oxygen administration and fluid balance. The specific obstetric conditions need to be treated as per protocol of the hospital. Regular laboratory investigations should be performed including blood gas analysis ($pO_2$, $pCO_2$, oxygen saturation, pH, bicarbonate and base deficit), serum electrolytes, renal and hepatic function tests. Imaging (CT scan or MRI) may be done as per indication. Continuous ECG monitoring is performed. Foetal management includes maintaining placental perfusion, regular foetal monitoring and timely delivery as per maternal and foetal indications in collaboration with neonatologists.

There is a need to develop obstetric HDU in all teaching and tertiary hospitals in India for better obstetric outcome. FOGSI (Federation of Obstetric and Gynecological Societies of India) is recommending it for all major hospitals. Many major hospitals like Safdarjang Hospital, Delhi have their own obstetric intensive care units (ICU) or HDU for better maternal and perinatal outcome.

## DAY CARE OBSTETRICS (DAY CARE UNIT)

Day care obstetrics is a recent concept on the pattern of day care minor surgeries. The pregnant woman is given inpatient care on an outpatient basis for the entire day by clinical and laboratory monitoring by a trained obstetrician.

### Indications

1. Medical disorders like hypertensive disorders, diabetes and other disorders.
2. Women with a history of previous stillbirth.
3. History of reduced foetal movements.
4. Anaemic mothers
5. Postmaturity

### Procedure

Foetal heart rate monitoring including non-stress test, biophysical profile, blood pressure, urine analysis, foetal heart rate, GTT, total dose iron infusion and serial recording of weight can be done. Attendances are recorded on a care plan kept with the antenatal record and updated at each visit. The junior doctor discusses her case with the senior obstetrician on a daily basis to review results and follow-up action. A composite assessment of risk can be made and patients may be either admitted or discharged.

**Advantages**
There is a reduction in overcrowding of the inpatient wards. Also the work load in major hospitals can be reduced. It is also advantageous to the woman as she need not undergo hospitalization and be separated from her family. It reduces the hospital costs significantly.

## RURAL OBSTETRICS

India is a country of villages with more than 70% people living in rural areas who have their own problems. The maternal and perinatal mortality and morbidity are higher in villages than cities.

### Problems of Rural Areas

1. Women in rural areas are at an economic disadvantage with lesser facilities.
2. Due to myths and false beliefs in their culture, they often tend to ignore the pregnancy complications as part of a normal pregnancy and do not report on time to health services causing increased mortality.
3. Lack of understanding, lack of trained and skilled staff in villages and lack of equipment and drugs.
4. Due to lack of MTP and contraceptive services, unplanned and unwanted pregnancies are more common. Similarly, unhealthy and illegal abortions by non-trained persons put their lives at risk.
5. They tend to get married early and tend to have pregnancy in young age (teenage pregnancy) and repeated pregnancies at short intervals making them more vulnerable to pregnancy complications.
6. Poor nutrition and high prevalence of worm infestations lead on to increased prevalence of anaemia during pregnancy with all its complications.
7. Women in rural areas do not like the idea of going to a hospital for delivery. Instead, they tend to have home delivery with the help of less trained traditional birth attendants (TBAs or *Dais*) or even family members which is very economical to them. More than 60% deliveries still occur in villages at home putting them at risk.
8. Lack of availability of transport to shift women to city hospitals in case of emergency, especially in hilly and tribal areas.
9. Lack of communication system, especially in hilly and tribal areas.
10. There are social problems in rural areas like poverty, malnutrition, illiteracy and traditions and beliefs to have delivery at home. The transfer to tertiary care centers is made too late putting the life of women and fetus at risk, e.g. rural women often go to government hospitals with massive obstetric haemorrhage, eclampsia and rupture uterus when there is risk of high mortality and severe morbidity.
11. There is gender discrimination and preference for a son. Quite often the women during pregnancy eat leftovers and are treated as inferior to men. There is also preference to have a son causing repeated pregnancies or indulgence in illegal sex determination and female feticide.

### Measures to improve rural obstetrics

1. Training of traditional birth attendants (TBAs). As village women prefer to get delivered by TBAs or *dais*, it is unfair to ignore TBAs. In fact, TBAs can be trained to give tetanus immunization, distribute free iron and folic acid tablets and recognize danger signals and refer them to hospital in time. She can be provided safe delivery kit and can be taught to take care of asepsis. She can also be trained to help in contraceptive services, dietary advice, breastfeeding, referring women for safe MTPs and in vaccination. A well-trained TBA who can cater to a village of about 1000 people and can deliver about 100 deliveries a year, can help in reducing mortality and morbidity by timely referral to a hospital.
2. Better communication and transport system
3. Better understanding, economic upliftment of society, better nutrition, clean drinking water and hygienic toilets can significantly help in improving maternal health.
4. Support from men is vital to improve maternal health. He should also share responsibility about contraception, in child care and in protection against sexually transmitted infections.
5. Strengthening of already existing peripheral health centers and other hospitals can do wonders. Aim should be to have hospital deliveries and 100% antenatal care.
6. Strengthening of community health centers (CHCs) and first referral units (FRUs), as planned in Reproductive and Child Health Programmes (RCH) can do wonders in improving maternal and perinatal outcomes.
7. Strengthening and adequate implementation of already existing maternal programmes like RCH, integrated child development scheme (ICDS), Janani Suraksha Yojana, Rural Health Mission and proper use of Accredited Social Health Activist (ASHA) can help in improving maternal and perinatal outcomes throughout the country including villages (see Chapter 45 for details).
8. Role of non-government organizations (NGOs) and public-private partnership should not be underestimated.
9. Now through Janani Suraksha Yojana (JSY) rural tribal women can also avail the benefits of free ambalance to take them to government hospital and free transport back to their homes, free hospital delivery or cesarean delivery, free hospital stay, some incentive money and free neonatal care and vaccination. It has significantaly increased hospital delivery rates and has reduced maternal and perinatal mortality and morbidity all over India

### Dawn's rule of ten (C S Dawn, Kolkata) Reproductive and Child Healthcare to grow 3 kg baby

It is a simple and easy to remember pictorial method for better maternal and perinatal outcomes in rural India.

1. Ten (5 for low risk) times pregnancy check-up for high risk cases.

2. Ten kg maternal weight gain by adequate diet.
3. Ten hours rest and sleep (2 + 8 hours in day and night)
4. Ten g hemoglobin by diet and iron folate supplementation.
5. By ten months of pregnancy: Two doses of tetanus vaccine (at 18 and 24 weeks)
6. Ten-twelve hours normal labour by trained personnel (high-risk cases in hospital).
7. Ten APGAR score baby.
8. By ten weeks of puerperium contraception is offered.
9. Ten months breastfeed to baby.
10. By ten months, infant immunization to be completed.

## Key Points

1. Bad obstetric history is where the present obstetric outcome is likely to be adversely affected by previous obstetric complications. Its causes may be in early pregnancy, mid pregnancy, late pregnancy and can be medical disorders, genetic disorders or obstetric causes. Detailed history taking and investigations to find out the cause and offending factor are important to plan the treatment. Management includes treatment of the offending cause, like diabetes.
2. Antiphospholipid antibody syndrome (APLS or APLA) with presence of lupus anticoagulant or anticardiolipin antibodies is an important cause of bad obstetric history and can cause recurrent foetal losses, foetal growth restriction, pre-eclampsia and abruption. Its treatment includes low dose aspirin and heparin therapy during pregnancy.
3. Occurrence of pregnancy in adolescent girls (up to 19 years) is called teenage pregnancy and is a high-risk pregnancy. Poor social and economic status, early marriage, stress, etc., are high risk factors for teenage pregnancy.
4. Prevention is by literacy of women, avoidance of early marriage and promotion of sex education in schools and colleges with promotion of contraceptives.
5. There are high maternal and perinatal complications in teenage pregnancies. There are more chances of miscarriages, anaemia, malnutrition, preterm labour, pre-eclampsia, infections and increased rate of cesarean delivery. There are also higher perinatal complications and social problems in such children.
6. Grand multipara is a pregnant mother with four or more previous births. There are higher maternal and perinatal complications which include miscarriages, chromosomal defects, obstetric complications, malpresentations, multiple pregnancy, medical disorders, prolonged labour, cephalopelvic disproportion, cord prolapse, obstructed labour, rupture uterus and more puerperal complications. Careful assessment of patients during antepartum period and in labour is important with timely cesarean delivery for selected cases for optimum outcome.
7. Pregnancy after 35 years is becoming more common and is associated with increase in maternal and perinatal morbidity and mortality due to medical problems and obstetric problems including antepartum and intrapartum complications. There is increased incidence of foetal congenital malformations including chromosomal abnormalities like Down's syndrome, small-for-date infants, perinatal and neonatal mortality. Management is prenatal diagnosis for any chromosomal or congenital abnormality and care during antenatal and intrapartum period.
8. Obesity (BMI > 30 kg/m$^2$) is becoming more common and can be associated with polycystic ovarian disease and insulin resistance. During pregnancy, there are more chances of pre-eclampsia and foetal growth restriction. During labour, there are increased chances of dysfunctional labour, shoulder dystocia and cesarean delivery.
9. Obstetric high dependency units (HDU) are being started in labour wards of many tertiary hospitals for intermediate risk obstetric women (severe pre-eclampsia, haemorrhage, sick women) where expert care is given by a dedicated team of multi-speciality doctors and nurses. Very sick women who cannot be managed in HDU still need to be shifted to main intensive care unit (ICU) of the hospital managed by anesthesiologists and physicians.
10. Day care obstetrics deal with foetal welfare clinics in which women with hypertension, anaemia, previous stillbirth and postmaturity, etc., can be assessed and sent home after doing foetal heart rate monitoring (NST and biophysical score), BP monitoring and urine examination. It avoids unnecessary admissions saving hospital money, reducing workload on doctors and helps women to remain in their homes. Patients can come back to day care clinic regularly (e.g. twice or thrice a week) for continued indications like hypertension. If needed, she can be admitted any time.
11. Rural obstetric problems are related to poor nutrition, poor socio-economic status, poor facilities and home deliveries by untrained persons. Strengthening the already existing PHCs, CHCs and FRUs and proper implementation of newer schemes and adequate use of ASHA and NGOs can definitely reduce maternal and perinatal mortality and morbidity. Free transport to hospital, free hospital delivery and incentive money through JSY has increased hospital delivery rates and decreased maternal and perinatal mortality and morbidity.
12. Ideally, all women should have 100% antenatal care during their pregnancies and there should be 100% hospital deliveries for optimum outcome.

CHAPTER 41

# Traumatic Conditions and Shock in Obstetrics

## TRAUMATIC CONDITIONS

### Trauma

Falls during pregnancy, excessive assaults (like domestic violence and sexual abuse) and vehicular accidents can cause blunt trauma during pregnancy. There can be penetrating wounds by gun shot injuries, stab wound due to knife or due to horns of ox, cow or buffalo.

#### Maternal Effects

1. Haemorrhage
2. Pelvic fractures
3. Intra-abdominal injuries like diaphragmatic, liver, splenic, renal and bowel injuries are less common due to protective effect of gravid uterus.
4. Traumatic placental abruption due to uterine location. Its incidence is 1–6% with minor injuries and 50% with major injuries. Concealed abruption is more common. There is increased incidence of coagulation failure. The clinical features of traumatic abruption resemble spontaneous placental abruption (see Chapter 31 for details).
5. **Traumatic uterine rupture.** It can occur in 1% cases being more common with scarred uterus (previous cesarean section) and with direct excessive trauma to abdomen and uterus or with penetrating trauma (see Chapter 26 for details). The diagnosis may be confused with placental abruption.
6. **Fetomaternal haemorrhage.** Due to tears or lacerations in the placenta in up to 30% cases of trauma.
7. Chorioamnionitis
8. Preterm labour

#### Foetal Effects

1. Prematurity
2. Foetal growth restriction
3. Foetal death

#### Prevention

Use of proper seat belts under the abdomen and over both anterior superior iliac spines and pubic symphysis (Fig. 41.1) with a shoulder strap in cars during accident decrease the frequency of maternal injuries. Wrongly put belt may compress the uterus causing placental abruption, uterine rupture and direct foetal injury.



**Fig. 41.1:** Correct position of seat belt during pregnancy, with one strap over the shoulder and the other under the abdomen over both anterior superior iliac spines and pubic symphysis

#### Management

The initial approach is like that of any trauma victim. Evaluation and stabilization of maternal injuries is the main goal. Intravenous hydration, maintenance of airways, oxygen therapy and shifting to a proper hospital are done.

Complete blood, platelet count and coagulation profile are tested. Kleihauer-Betke test is done for detection of fetomaternal haemorrhage. Anti-D should be given to Rh– women.

Tetanus prophylaxis is given if the patient was not immunized earlier. Cardiotocography is done to assess foetal well-being.

Ultrasound is preferred but CT scan can be required. Foetal monitoring is done by cardiotocography, ultrasound, non-stress testing and biophysical scoring.

Treatment depends upon type of injuries. Conservative

treatment is preferred. Laparotomy may be required for suitable indications. Cesarean is not to be performed if the abdomen is opened for some other injury. Spontaneous vaginal delivery is anticipated later. The fetus is not adversely affected by laparotomy.

## DOMESTIC VIOLENCE IN PREGNANCY

### Definition

**Domestic violence is defined as the violence against adolescent and adult females within the context of family or close relationships.**

### Prevalence

The data about domestic violence in India is not available, but is probably quite high. Prevalence may be as high as 5–21% in pregnancy and 13–21% postnatally.

### Effects on pregnancy

Domestic violence during pregnancy increases the risk of:

1. Threatened abortion
2. Antepartum haemorrhage
3. Foetal growth restriction
4. Premature labour
5. Perinatal death
6. Low birth weight
7. Uterine rupture
8. Neonatal death

ACOG (2012) recommends universal screening for intimate partner violence during antenatal visits and postpartum.

### Diagnosis

Domestic violence may begin or worsen during pregnancy and may cause late booking, poor attendence, repeated visits for minor injuries or non-existent complaints. There may be associated alcohol or drug abuse and partner may always be present with the patient to avoid her complaint.

### Management

There is need for screening for domestic violence and a very high index of suspicion is required on the part of the obstetricians who should ask direct questions about domestic violence. The woman should be seen alone and should be asked about the violence or a survey form about domestic violence should be filled. There may be need to report to police or social security agencies if violence is affecting her health. She should be seen more often in antenatal clinic, should take adequate nutrition, tetanus immunization, iron and calcium supplementation, foetal monitoring and should deliver in hospital for optimum maternal and perinatal outcomes. Evidence indicates that counselling and mentoring may be effective.

## SEXUAL ABUSE IN PREGNANCY

Lifetime prevalence of sexual violence among women in USA ranges from 18% for rape to 27.2% for unsolicited sexual contact. Sexual assault, forced sex or rape has been reported in up to 8% pregnant adolescents in USA mostly by member of the family usually husband or boyfriend. The exact prevalence in India is not known as most women do not report such abuse and obstetricians also avoid asking questions from the patients. Obstetricians need to diagnose and treat it and may need to report to police or social agencies to prevent further assault.

### Effects

1. Increased risk of sexually transmitted diseases
2. Increased risk of urinary infections
3. Increased risk of vaginitis
4. Increased risk of drug use
5. Multiple hospital admissions
6. Physical injuries
7. Uterine rupture
8. Preterm delivery
9. Low birth weight new born
10. Psychological injuries

ACOG (2012) recommends universal screening for intimate partner violence during antenatal visits and postpartum.

### Management

Obstetricians have to care for such women with compassion taking care of their diet, offer iron and calcium supplementations and foetal monitoring. Prophylaxis for sexually transmitted diseases in such victims should be with azithromycin (1 g) oral single dose to cover *Chlamydia trachomatis*, metronidazole or secnidazole 2 g oral single dose to cover *Trichomonas vaginalis* and ceftriaxone 125 mg intramuscularly if there is suspicion of gonorrhea (CDC 2015). Such women should deliver in hospitals.

## CARDIOPULMONARY RESUSCITATION

Cardiopulmonary resuscitation may be rarely required for cardiac arrest during pregnancy due to any reason usually unrelated to pregnancy like trauma, burns and haemorrhage. The steps of cardiopulmonary resuscitation are same as in non-pregnant state. Only difference is lateral displacement of uterus manually by an assistant to avoid uterine aortocaval compression which may hamper resuscitative efforts by reducing forward blood flow and venous return.

### Perimortem cesarean section

See Chapter 42(v) for details.

## SHOCK IN OBSTETRICS

### Definition

Shock is defined as a clinical condition arising out of an inability of the circulatory system to provide adequate tissue perfusion causing cellular hypoxia and organ damage. It is a systemic disorder affecting multiple organ systems. Perfusion may either be decreased throughout the body or distributed poorly (e.g. septic shock). Shock during pregnancy is one of the most difficult problems faced by the

obstetrician and necessitates initiation of management even before full identification of its cause for better survival.

## Types and causes of shock in obstetrics

A. **Hemorrhagic shock.** Hemorrhagic shock due to hypovolemia is the most common cause of shock in obstetrics.

B. **Non-hemorrhagic shock.** The various causes of non-hemorrhagic shock are as follows:

1. **Trauma** due to obstetric operations like difficult forceps delivery, forced breech extraction, internal podalic version, manual removal of placenta, use of Crede's method of placental delivery and cesarean section and acute inversion of uterus.
2. **Fluid loss** due to hyperemesis gravidarum and excessive diuresis can cause hypovolemic shock without haemorrhage.
3. **Septic** shock due to infective conditions (see below for details).
4. **Hypertensive disorders** of pregnancy can cause shock after delivery due to hypovolemia.
5. **Anesthesia**: Anaesthetic accidents can cause shock due to prolonged general anesthesia or deeper planes of anesthesia. Inhalation of acidic gastric contents can cause shock due to Mendelson's syndrome. Spinal anesthesia can also cause shock (see Chapter 19 for details).
6. **Hypertensive drugs**, mismatched blood transfusion or anaphylactic reaction to drugs, like iron dextran can cause shock.
7. **Cardiogenic.** Pump failure due to myocardial inefficiency, e.g. myocardial infarction and cardiac arrhythmias (see below for details).
8. **Neurogenic** shock can sometimes occur after normal delivery due to extreme pain or fear (see below for details).
9. **Pulmonary embolism** developing due to dislodgement of a thrombus from pelvic or leg veins or infected placental sinuses or embolization of air during obstetric manipulations (see Chapter 30 for details).
10. **Supine hypotension syndrome** is a shock like condition of hypotension in supine position (See Chapter 6 for details).

# HYPOVOLEMIC OR HEMORRHAGIC SHOCK

The pathophysiology is shown in Flow Chart 41.1. It is due to excessive blood loss.

1. **Early pregnancy.** Due to abortion, ectopic pregnancy and gestational trophoblastic disease (see Chapter 13 for details).
2. **Antepartum haemorrhage.** Due to placenta previa or abruptio placentae (see Chapter 31 for details).
3. **Rupture uterus, traumatic** PPH and atonic postpartum haemorrhage (see Chapters 25 and 26 for details)

## Clinical Picture

Patients usually present with low blood pressure, a rapid and thready pulse, pallor, cold clammy extremities, air hunger, diminition of vision, oliguria and anuria.

## Phases of Hemorrhagic Shock

### 1. Phase of Compensation

**Blood loss is less than 15% (mild).** No change in vital signs is seen. Postural hypotension is noted.

Sympathetic stimulation is the initial response to blood loss leading to peripheral vasoconstriction to maintain blood supply to the vital organs. There is constriction of the pre- and post-capillary sphincters resulting in inadequate venous return causing reduced cardiac output.

#### *Clinical Picture*

Pallor, tachycardia, tachypnea, sweating and normal blood pressure. At this phase, transfusion, resuscitation and control of haemorrhage are usually effective in restoring the normal circulation and perfusion. There is hyperventilation.

### 2. Phase of Decompensation

**Blood loss is 20 to 35% (moderate).** It is associated with cold and clammy skin, tachycardia, tachypnea, low pulse pressure, low systolic pressure and delayed capillary filling. Blood loss exceeds 1000 mL in normal patient or less if other adverse factors are operating (severe anaemia).

#### *Clinical Picture*

The phase of decompensation is the classic clinical picture of shock. Adequate treatment at this phase improves the condition rapidly without residual adverse effects. If untreated it can become irreversible by passing onto the phase of cellular damage. There is relaxation of pre-capillary sphincters and damage to the microcirculatory bed due to thromboxane $A_2$ and leukotriens.

### 3. Phase of Cellular Damage and Danger of Death

**Blood loss is more than 40% (severe).** It is associated with profound hypotension with only the carotid pulse being palpable. Oliguria and anuria are noted.

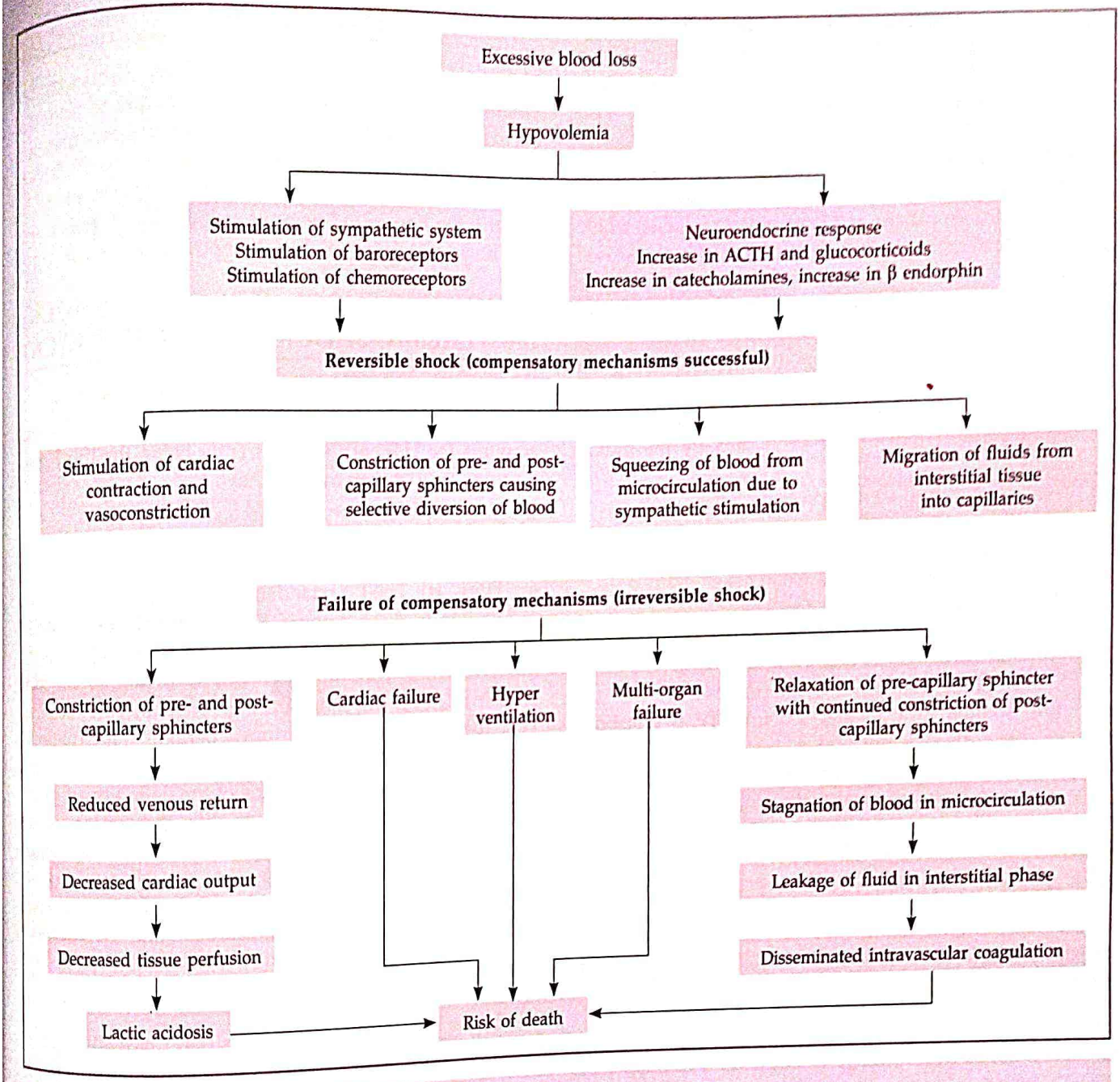
Inadequately treated hemorrhagic shock results in prolonged tissue hypoxia and other effects.

(*i*) There is relaxation of pre-capillary sphincters (arteriolar dilatation) due to metabolites with persistent constriction of post-capillary sphincter. This causes stagnation of blood in capillary bed with its leaking into tissue spaces. All fluids administered will go into tissue spaces without restoring circulatory blood volume.

(*ii*) It is a stage of irreversible shock with severe loss of systemic vascular resistance, severe myocardial depression, decreased cardiac output, non-responsive hypotension and multiple organ system failure.

(*iii*) Hyperventilation due to poor perfusion of lungs.

(*iv*) **Biochemical changes:** The blood sugar is raised probably due to the breakdown of liver glycogen under the influence of adrenaline. Sodium and water retention occur under the




**Flow Chart 41.1:** Pathophysiology of hypovolemic shock

influence of raised levels of aldosterone and antidiuretic hormone (ADH). Potassium levels are raised if there is much cell damage despite the fact that potassium excretion is encouraged by aldosterone. The blood urea can rise. If tissue perfusion is severely reduced, then tissue hypoxia can lead to anerobic metabolism causing metabolic acidosis due to production of lactic acid.

(*v*) Disseminated intravascular coagulation caused by release of thromboplastin from the damaged tissues.

(*vi*) Cardiac failure due to diminished coronary blood flow.

In the phase of cellular damage and danger of death, death is imminent and transfusion alone is inadequate. If recovery from acute phase occurs, residual tissue damage as renal and/or pituitary necrosis can occur.

## Management of Hemorrhagic/ Hypovolemic Shock

In all cases of hypovolemic shock, speed is vital and rapid restoration of the circulating blood volume is the key to successful outcome. The measures are as follows:

1. Establishment of an airway and oxygen therapy at a rate of 6–8 L per minute to maintain oxygen saturation of > 92% and $PaO_2$ of 80–100 mm Hg. Endotracheal intubation and mechanical ventilation are required for severe hypoxia, severe tachypnea and coma to maintain oxygen supply.
2. Establishment of two wide bore (14G) intravenous lines with collection of blood sample for grouping, crossmatching and baseline investigations.
3. Elevation of legs to encourage return of blood from the limbs to the central circulation.
4. Keep the woman warm using appropriate available measures.

5. Volume replacement which is done initially by crystalloid solutions. One to three litres of warm Ringer lactate or (1–2 L) Isolyte M solution should be infused rapidly. Plasma expanders, like, hemaccel may also be used initially and are said to reduce the viscosity and improve the microcirculation. They will remain in circulation for 24 to 48 hours. Plasma protein fraction or fresh frozen plasma and later, when available, whole blood preferably packed cells should be transfused quickly.
6. Insert a Foley's catheter in the urinary bladder to maintain urine output.
7. Positioning of the patient in the Trendelenburg tilt may aid the venous return. In late pregnancy, patients should be nursed on their sides to avoid uterine compression of the inferior vena cava by the gravid uterus.
8. Insertion of central venous pressure line.
9. Drug therapy
   (*i*) **Analgesics.** 10–15 mg morphine intravenously if, there is pain, tissue damage or irritability.
   (*ii*) **Corticosteroids.** Hydrocortisone 1 g or dexamethasone 20 mg slowly (IV). It may reduce the peripheral resistance and potentiate cardiac response to improve tissue perfusion.
   (*iii*) **Sodium bicarbonate.** 100 mEq IV, if metabolic acidosis is demonstrated.
   (*iv*) **Vasopressors.** To increase the blood pressure so as to maintain renal perfusion.
      a. **Dopamine.** 2.5–10 µg/kg/minute by IV infusion is the drug of choice.
      b. **β-adrenergic stimulant.** Isoprenaline 1 mg in 500 mL 5% glucose slowly IV infusion.
      c. **Digitalization** may be carried out if inspite of volume replacement the CVP remains high due to myocardial failure. This is unlikely, except as a terminal event.
      d. **Vasodilators.** Phenoxybenzamine an alpha-receptor blocker is given as 1 mg/kg over 1 hour intravenously if it is felt that the vasoconstriction is persisting inspite of adequate volume replacement and with CVP monitor.

As this management is being done, simultaneously ultrasound examination is carried out to find the cause of blood loss and assess the fetus in antenatal patients and to rule out placental bits in postpartum patients.

Monitoring is done by assessment of central venous pressure, pulse rate, blood pressure, urine output, pulmonary capillary wedge pressure and by clinical improvement in the pallor, cyanosis, air hunger, sweating and consciousness.

In addition to medical management, surgical intervention depending upon the cause of haemorrhage like emergency cesarean for antepartum haemorrhage, laparotomy, internal iliac ligation or hysterectomy for severe postpartum haemorrhage may be required.

## NORMOVOLEMIC SHOCK

Normovolemic shock covers several fairly distinct entities where the blood volume is normal but due to arteriolar or capillary dilatation the capacity of the vascular tree is increased. Thus, the normal volume is insufficient to provide adequate tissue perfusion. Management is by treating the basic condition, resuscitation with fluids with or without vasopressors, like in hypovolemic shock.

## ENDOTOXIC SHOCK (SYN. SEPTIC SHOCK, BACTEREMIC SHOCK, BACTERIAL SHOCK)

Southern Asia and Sub-Saharan Africa contributes to the greatest burden of maternal deaths. Maternal sepsis accounts for 11% of these, currently being the third most leading cause of maternal deaths.

### Definition

In order to reduce the impact of sepsis on maternal mortality, we need a standard definititon for the early recognition of these women. Keeping this in mind, WHO has now issued a new definition of maternal sepsis as "A life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or postpartum period." International Consensus on Sepsis, 2016 (SEPSIS-3 Task Force) Flow Chart 41.2A has shifted the focus of the definition of sepsis from inflammatory response to life threatening organ dysfunction; further clarifying criteria to define organ dysfunction and septick shock.

Organ Dysfunction can be identified as an acute change in total SOFA score of 2 points consequent to the infection.

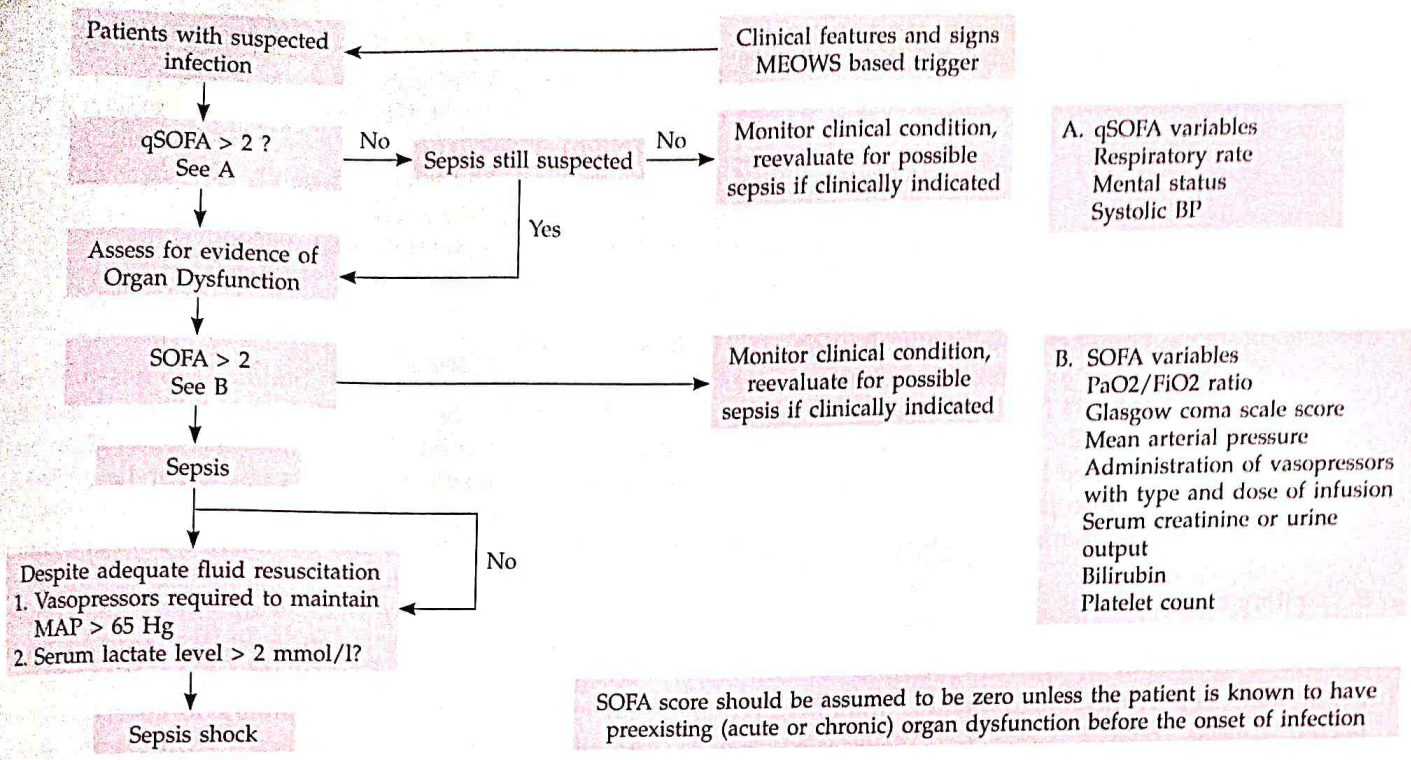
- The baseline SOFA score can be assumed to be zero in patients not known to have preexisting organ dysfunction.
- A SOFA score 2 reflects an overall mortality risk of approximately 10% in a general hospital population with suspected infection.

**Septic shock** is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality.

- Patients with septic shock can be identified with a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP ≥ 65 mm Hg and having a serum lactate level > 2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%.

The SOFA score is not used to guide the management but as a clinical tool to identify women having sepsis and for their prognostication.

The task force suggests that qSOFA criteria be used to prompt clinicians to further investigate for organ dysfunction, to initiate or escalate therapy as appropriate, and to consider referral to critical care or increase the frequency of monitoring, if such actions have not already been undertaken. It is a quick and easy tool not requiring




**Flow Chart 41.2A:** The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

any lab tests for identification of women likely to have sepsis. A "positive" qSOFA Score (≥ 2) suggests high risk of poor outcome in patients with suspected infection. These patients should be more thoroughly assessed for evidence of organ dysfunction. A positive qSOFA Score by itself should not trigger sepsis-directed interventions like initiation of broad-spectrum antibiotics; rather, it should prompt clinicians to further investigate for presence of organ dysfunction or to increase frequency of monitoring. The Sepsis-3 task force recommends that a positive qSOFA Score should prompt the calculation of a SOFA score to confirm the diagnosis of sepsis. This remains controversial, as qSOFA has been shown to be more predictive than SOFA outside of the ICU setting.

**Table 41.1: Sequential [Sepsis-Related] Organ Failure Assessment Score**

| System | Score | | | | |
|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | |
| **Respiratory** | | | | | |
| $PaO2/FIO_2$, mmHg (kPa) | ≥ 400 (53.3) | < 400 (53.3) | < 300 (40) | < 200 (26.7) with respiratory support | < 100 (13.3) with respiratory support |
| **Coagulation** | | | | | |
| Platelet, × 103 $\mu L^{-1}$ | ≥ 150 | < 150 | < 100 | < 50 | < 20 |
| **Liver** | | | | | |
| Bilirubin, mg $dL^{-1}$ (µmol $L^{-1}$) | < 1.2 (20) | 1.2–1.9 (20–32) | 2.0–5.9 (33–101) | 6.0–11.9 (102–204) | > 12.0 (204) |
| **Cardiovascular** | MAP ≥ 70 mmHg | MAP < 70 mmHg | Dopamine < 5 or dobutamine (any dose) | Dopamine 5.5–15 or epinephrine ≤ 0.1 or norephinephrine ≤ 0.1[a] | Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1[a] |
| **Central Nervos System (CNS)** | | | | | |
| Glasgow Coma Scale score[b] | 15 | 13–14 | 10–12 | 6–9 | < 6 |
| **Renal** | | | | | |
| Creatinine, mg $dL^{-1}$ (µmol $L^{-1}$) | < 1.2 (110) | 1.2–1.9 (110–170) | 2.0–3.4 (171–299) | 3.5–4.9 (300–440) | > 5.0 (440) |
| Urine output, mL per day | | | | < 500 | < 200 |

$FIO_2$: fraction of inspired oxygen; MAP: mean arterial pressure, $PaO_2$: partial pressure of oxygen
[a]Catecholamine doses are given as µgkg–1 min–1 for at least 1h.
[b]Glasgow Coma Scale scores range from 3 to 15: higher score indicates better nerological function

| Table 41.2: qSOFA (Quick SOFA) Criteria | |
|---|---|
| qSOFA (Quick SOFA) Criteria | Score |
| • Respirator Rate: ≥ 22 breaths/min | 1 |
| • Altered Mentation: GCS ≤ 15 | 1 |
| • Systolic BP: ≤ 100 mmHg | 1 |

## Etiology

1. Septic abortion, especially criminal septic abortion
2. Chorioamnionitis due to premature rupture of membranes (see chapter 34 for details).
3. Puerperal sepsis especially after emergency cesarean section done for obstructed labour.
4. Retained products of conception
5. Instrumentation of genitourinary tract
6. Acute pyelonephritis
7. Respiratory tract infections

## Causative Organisms

1. Septic shock is polymicrobial in nature. Gram-negative bacilli: *E. coli, Proteus, Klebsiella, Pseudomonas and Bacteroides.*
2. Gram positive aerobes like *β-hemolytic streptococci*
3. Anerobes like *anerobic streptococci, Clostridia* and *Bacteroides*

## Pathophysiology

Pathophysiology of endotoxic shock is shown in Flow Chart 41.2B.

## Clinical Features

Endotoxic shock is a serious condition and mortality may be as high as 50%. Endotoxic shock has two stages.

**Stage I Reversible stage** has 2 phases namely:

**Phase I (Phase of vasodilatation)** (warm phase). Hypotension is associated with fever of 101–105 °F with rigors, tachycardia, tachypnea and flushed skin. Patients are alert. There is hypotension, leucopenia and oliguria.

**Phase II Phase of vasoconstriction.** Patients have cold and clammy skin, cyanosis, jaundice and bradycardia. There is subnormal temperature, cyanosis, occasional jaundice, disseminated intravascular coagulation, leucocytosis and oliguria. There is progressive mental confusion and coma.

**Stage II (Irreversible stage).** Irreversible damage due to prolonged cellular hypoxia results in metabolic acidosis, acute renal failure, cardiovascular failure, pulmonary edema, adrenal failure and ultimately death.

## Differential Diagnosis

Differential diagnosis includes amniotic fluid embolism, pulmonary embolism, adult respiratory distress syndrome (ARDS) and myocardial infarction.

## Diagnosis and Investigations

1. Aerobic and anerobic culture sensitivity of cervical secretion and maternal blood.
2. Complete hemogram, total and differential leucocyte count, fibrinogen, blood and urea, creatinine, serum, electrolytes, blood pH, S. Lactate levels.
4. Markers for sepsis: CRP, Procalcitonin, cytokines (IL-6, TNF-α)
5. X-ray chest, abdomen and pelvis for gas shadow and foreign body (septic abortion)
6. Ultrasound of abdomen and pelvis
7. Take swabs

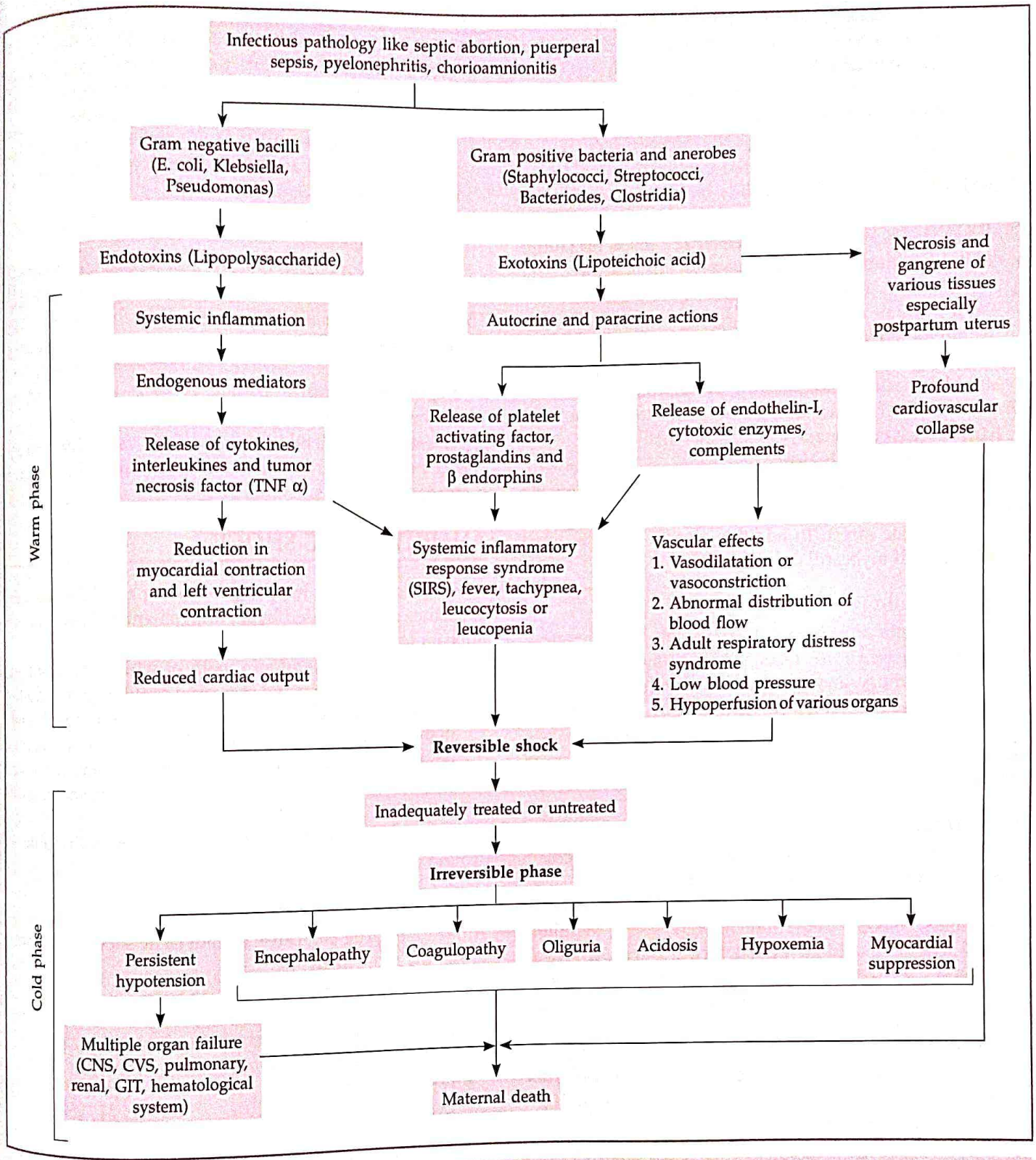
## Management

The surviving sepsis campaign bundle 2018 has combined the 3 hour and 6 hour bundle into the "hour-1 bundle" with explicit intention of beginning resuscitation and management immediately. [3] The sepsis 6 bundle includes:

- Administration of oxygen to maintain SpO2 at >94%
- Taking blood cultures and considering infective source prior to giving antibitotic.
- Administration of intravenous antibiotics. Intravenous antibiotics should be started within 1 hour of sepsis recognition and should include combination therapy (at least two classes of antibiotics to cover a known or suspected pathogen) for patients with septic shock. Combination therapy should not routinely be used for patients without shock. Routinely a combination of ampicillin or cephalosporin with gentamicin and metronidazole by parenteral route is given.
- Checking serial lactates. Results from SSC database has found that patients with lactate > 4 mmol/L had significantly increased mortality and there is a linear relationship between lactate and mortality. A baseline lactate helps in monitoring the resuscitation by targeting its clearance. Another entity is 'cryptic septic shock' that is those who have end organ hypoperfusion without hypotension due to compensation. Difficulty in recognition causes delay and poorer outcomes. Lactate is used to identify this condition.
- Fluid resuscitation with crystalloids guided by lactates. 30 mL/ kg of intravenous crystalloid to be given within 3 hours and should be re-assessed frequently. Commence hourly urine output measurement. Colloids may be associated with increased risk of acute kidney injury.
- Early administration of vasopressors to maintain MAP of > 65 mm Hg. Norepinephrine is the first choice for patients who need vasopressors. Vasopressin or epinephrine can be added. Dobutamine is finally given for patients who still remain unstable.

As per the surviving sepsis campaign guidelines:

1. Blood transfusion to be done when hemoglobin < 7.0 g/dl except in special circumstances such as haemorrhage or myocardial infarction. Indications for platelet transfusion are < 10,000/mm$^3$ without bleeding, < 20,000/mm$^3$ if there is bleeding or the patient receiving chemotherapy and <50,000/mm$^3$ if invasive procedure are planned.
2. Maintain the urine output > 30 ml/kg/hr. Patients with hypoperfusion to be given 1-2 L of crystalloids (Normal saline or Ringers Lactate at the rate of 0.5 ml/ kg/hr within 3 hours and then reassess




**Flow Chart 41.2B:** Pathophysiology of septic shock

Only about 50% pf hypotensive septic patients are fluid responders. In those who are not, aggressive fluid administration may lead to left ventricular diastolic dysfunction, pulmonary edema and higher mortality. Patients who are fluid responsive should be identified prior to further fluid administration using pulse pressure variation and passive leg raising.

3. They help in combatting the relative adrenal insufficiency caused by sepsis and in septic shock; they have a vasopressor sparing role. Inj. Hydrocortisone IV 200 mg/day for patients who are hemodynamically unstable despite fluids and vasopressors.
4. Sodium Bicarbonate is not usually indicated if pH on arterial blood gas is > 7.15.
5. Aminophylline to overcome associated bronchospasm.

6. Surgical treatment to remove retained infected tissues or even laparotomy may be done. (see Chapter 13 and 30 for details)
7. Disseminated intravascular coagulation, if present, is treated. (see below).

## CARDIOGENIC SHOCK

Cardiogenic shock is circulatory collapse caused by failure of the heart to pump blood adequately.

### Etiology

1. Failure of left ventricular ejection due to:
   (*i*) Cardiac arrest due to asystole or ventricular fibrilation
   (*ii*) Myocardial infarction
2. Failure of ventricular filling
   (*i*) Cardiac tamponade
   (*ii*) Pulmonary embolism

Severe valvular disease is the major cause of cardiogenic shock during pregnancy. Any cause of obstetric shock can result in cardiac arrest. In addition, septic shock is often associated with myocardial dysfunction.

### Symptomatology

Patients present with distended neck veins, breathlessness, tachypnea, presence of third heart sound, systolic or diastolic murmurs and generalized edema. It may be followed by sudden collapse with loss of consciousness, absence of pulse including the carotid and femoral pulses, apnea, cyanosis of variable degree and fixed dilatation of pupils.

### Management

Cardiogenic shock is a serious condition needing immediate care by obstetrician, anesthetist and cardiologist.

1. Maintenance of airway by clearing the airway of vomitus, blood, teeth and foreign body. Mandible and tongue should be pulled forward and an airway inserted. Endotracheal intubation should be done as soon as possible to ensure patent airway and adequate oxygenation.
2. Mouth-to-mouth artificial respiration should be given or after inserting a cuffed endotracheal tube intermittent positive pressure ventilation using 100% oxygen should be given.
3. Cardiac massage is given after putting patients on a firm surface.
4. Drugs
   (*i*) Sodium bicarbonate 8.4% solution to counteract metabolic acidosis. Give 100 mL initially and a further 10 mL for each subsequent minute of inadequate circulation.
   (*ii*) Cardiac stimulants can be given solution IV or intracardiac, e.g. adrenaline 0.5–1.0 mg, isoprenaline 4 mg in 500 mL solution IV.
5. Direct current defibrillator is used, if required.

## NEUROGENIC SHOCK

Neurogenic shock may be due to painful conditions like:

1. Acute inversion of the uterus (see Chapter 25 for details).
2. Rapid evacuation of the uterine contents as in precipitate labour and rupture of membranes in polyhydramnios. This is accompanied by rapid accumulation of blood in the splanchnic area due to sudden relief of pressure also called splanchnic shock.
3. Vasovagal stimulation, emotion and fear (associated with trauma)
4. It may play a part in the shock of pulmonary embolism and supine hypotension syndrome.
5. Spinal anesthesia may cause serious hypotension due to blockade of the normal sympathetic vasomotor tone.

### Management

Management includes general measures like fluid replacement, correction of acidosis, vasoactive drugs, corticosteroids, ventilation and elimination of the source of neurogenic stimulus.

## ANAPHYLACTIC SHOCK

Anaphylactic shock happens following the injection of a substance and is caused by an antigen-antibody reaction. It appears to be associated with the release of:

1. Histamine and serotonin, both of which cause smooth muscle contraction and increased capillary permeability.
2. Plasma kinins, which cause vasodilatation. It is thought that in certain sensitive individuals immunoglobulin E is attached to mast cells and when the antigen reacts with it, there is a release of histamine from the mast cells.

Incidence is 3 to 10/1000 with a mortality rate of around 1%.

### Etiology

Almost any drug can cause anaphylactic shock and among those, the most likely are:

1. Antisera and toxoids
2. Penicillin and streptomycin
3. Intramuscular or intravenous iron, especially iron dextran and iron sorbitol
4. Local anesthetics

### Management

1. Definitive treatment is 0.5 ml of 1 in 1000 adrenaline IM, can be repeated after 5 min. if there is no effect. It can also be given IV as 50 µg bolus (0.5 ml of 1 : 10,000)
2. General measures
3. Intravenous fluids
4. Antihistaminics
5. Steroid therapy
6. Lipid rescue is a recently reported intervention for the management of local anesthesia toxicity.
7. Adjuvant therapy → chlorpheniramine 10 mg, hydrocortisone 100 mg.

## ADRENAL INSUFFICIENCY

Adrenal insufficiency gives rise to shock but the exact mechanism is unknown. It is thought that cortisone has an inotropic cardiac action and may sensitize the pre-capillary sphincters to catecholamines. Thus, deficient cortisone will lead to peripheral vasodilatation.

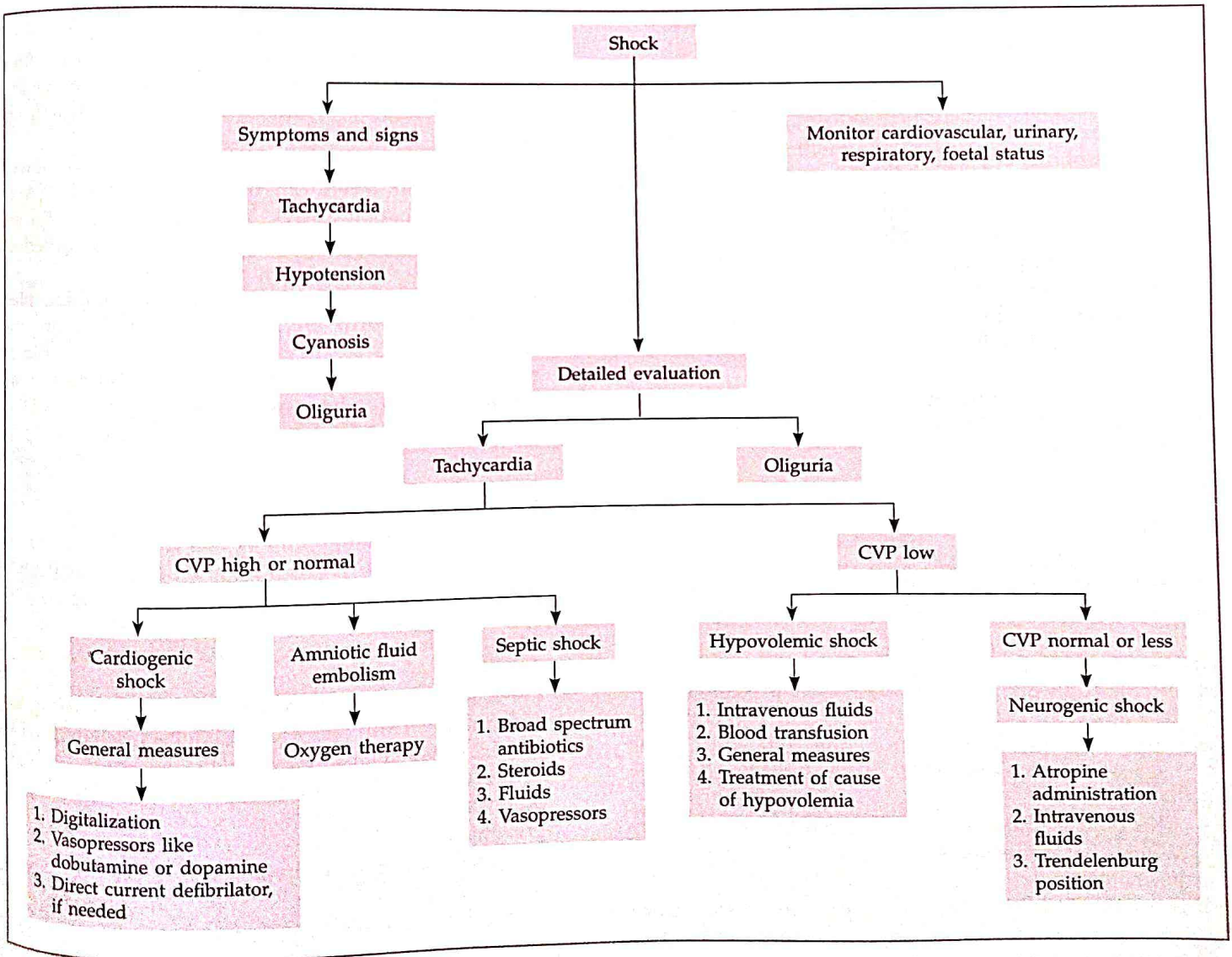
Management is by general measures, fluid replacement and steroid therapy.

## GENERAL MANAGEMENT OF OBSTETRIC SHOCK (Flow Chart 41.3)

Patients of shock must be attended immediately and treatment administered as follows:

1. Intravenous drip is started with Ringer lactate or Isolyte M solution.
2. Adequate crossmatched fresh blood is given as soon as possible.
3. Monitor transfusion with CVP measurements.
4. Oxygenation, artificial ventilation, if indicated
5. Look for specific causes and treat accordingly.
6. Hemostasis, if bleeding
7. Mendelson's syndrome is treated by antacids and general measures (see Chapter 19 for details).
8. Always consider a coagulation disorder.
9. Close observation and follow-up to prevent, diagnose and treat
   (*i*) acute renal failure (see Chapter 35[vi] for details).
   (*ii*) anterior pituitary necrosis (Sheehan's syndrome) (see Chapter 35[iv] for details).
   (*iii*) anaemia (see Chapter 35[i] for details).

The causes of shock during pregnancy may be varied, a quick review of patients' general condition and instituting resuscitative measures are of basic importance. Evaluation of the pulse rate and CVP monitoring provide excellent parameters for rational approach to management.




**Flow Chart 41.3:** Management of shock in obstetrics

# DISSEMINATED INTRAVASCULAR COAGULATION

## Definition

It was first described by Delee in 1901. Disseminated intravascular coagulation is characterized by a systemic activation of the blood coagulation system characterized by microvascular thrombi formation in various organs which can cause multiorgan failure. Consumption causes exhaustion of coagulation proteins and platelets, inducing severe bleeding complications. It is also called **consumptive coagulopathy**. *It is equivalent to the generalized Schwartzman reaction.*

## Types

1. **Acute DIC:** It is a hemorrhagic variety associated with excess plasmin formation with bleeding from various sites.
2. **Subacute or Chronic DIC:** In these patients, manifestation is thrombosis from excess thrombin formation.

## Etiology

### Acute

1. **Abruptio placentae. It is the most common cause.**
2. **Endotoxemia.** Septic abortion, chorioamnionitis, pyelonephritis of pregnancy
3. Amniotic fluid embolism due to leakage of thromboplastin like material from placental system.
4. Severe pre-eclampsia and HELLP syndrome (hemolysis, elevated liver enzymes and low platelets)
5. Hydatidiform mole
6. Intraamniotic hypertonic saline instillation for mid-trimester MTP.
7. Dextran infusion
8. Hemorrhagic shock due to obstetric haemorrhage like postpartum haemorrhage, cesarean section haemorrhage.
9. Acute fatty liver of pregnancy

## Subacute (or Chronic)

1. Missed abortion
2. Intrauterine death (prolonged)
3. Foetal Death Delayed Delivery
4. Discordant one foetal death in multifetal pregnancy

## Symptomatology

Symptoms occur due to three pathological processes.

1. Bleeding from various sites like petechiae, ecchymosis, purpuric spots, bleeding from injection sites, gums; hemetemesis, malena, bleeding per rectum; hematuria before delivery.
   After delivery. Postpartum haemorrhage, bleeding from suture sites, episiotomy, cesarean section site, vulval haematoma
2. **Systemic infection and septicemic shock.** High fever, temperature, irritability, toxic look, tachypnea, tachycardia, BP instability (hypotension)
3. Specific organ involvement
   (*i*) **Brain.** Altered state of consciousness, seizures
   (*ii*) **Lungs.** Respiratory distress or adult respiratory distress syndrome (ARDS)
   (*iii*) **Heart.** Hypotension, cardiac arrest
   (*iv*) **Kidneys.** Renal failure manifesting as oliguria, anuria, acidosis

## Diagnosis

No single laboratory test is sensitive and specific enough to make a definite diagnosis of DIC. A combination of tests in patients with typical signs and symptoms help in making the diagnosis.

1. Platelet count is reduced. In severe pre-eclampsia, qualitative platelet dysfunction may be there.
2. Clotting time especially prothrombin time (PT) and activated partial thromboplastin time (APTT) are prolonged. Thrombin time (normal: < 18 seconds) is prolonged.
3. Antithrombin III and protein C levels are reduced.
4. There is consumption and depletion of various coagulation factors like factors V and VII.
5. Estimation of fibrinogen is useful. There is hypofibrinogenemia but a single reading may not be useful. Sequential values of fibrinogen and other parameters are more useful in making the diagnosis. Value < 50 mg/dL is suggestive of severe hypofibrino genemia.
6. **Fibrin degradation products (FDPs) levels are raised in DIC.** Their levels can be measured by ELISA or latex agglutination assays. However, raised FDPs are not specific for DIC as their levels can be raised in inflammation or recent surgery.
7. **D-dimer levels which detect neoantigens on degraded cross linked fibrin are raised in DIC** but are not specific for DIC as they can be raised in trauma or recent surgery also. **Of all the tests D-dimer assay has been found to be most useful in diagnosing DIC.** Fibrino peptide-A titer is also increased in DIC.

## Management

### Prophylaxis

Prophylaxis is very important, as this is a life threatening complication. Good antenatal care and active management of labour can help in identifying high risk factors and treating them early and energetically as follows:

1. **Abruptio placentae.** Adequate blood transfusion, early delivery, liberal and early use of cesarean section.
2. **Pre-eclampsia eclampsia syndrome.** Early diagnosis and prompt treatment of pre-eclampsia, eclampsia and HELLP syndrome can prevent DIC.
3. **Intrauterine death** can cause DIC. Early termination of pregnancy using prostaglandin gel or oxytocin drip can prevent DIC.
4. **Avoidance of use of hypertonic saline** for second trimester abortion and using prostaglandin or ethacridine lactate can avoid DIC.
5. **Obstetric shock:** Early and adequate treatment in reversible stage by early administration of fluids and blood transfusion can prevent DIC.

### General management

DIC is a peculiar and paradoxical condition. On one hand, there is hypercoagulation with microthrombi formation in microcirculation theoretically requiring anticoagulation. On the other hand, rest of the blood has the tendency to bleed due to consumption of coagulation factors necessitating fibrin treatment. One treatment while treating one component may worsen other components of the disease. Treatment of underlying disease is important like use of appropriate intravenous antibiotics in adequate dosage for any infection, timely and appropriate management of haemorrhage and shock.

### Specific Treatment

The use of various blood and blood products in different obstetric indications is given in Table 41.3.

1. **Massive transfusion protocols.** In severe obstetric haemorrhage, aggressive resuscitation is performed using 5 units of packed red blood cells (PRBC) with 3 units of fresh frozen plasma (FFP) with 6 packs of platelets, in all patients. One unit of cryoprecipitate and 2 mg of recombinant activated factor VII (rVIIa) can be combined in serious cases. Some protocols include IV tranexamic acid (1 gm) also.
2. **Platelet and fresh frozen plasma (FFP) transfusion** as low level of platelets and coagulation factors may increase the risk of bleeding. However, plasma or platelet substitution therapy is indicated in patients with active bleeding and with severe thrombocytopenia (< 50,000/cmL) and in those requiring an invasive procedure or who are otherwise at risk for bleeding complications. ***One unit of FFP (250 mL) raises fibrinogen levels by 10 mg/dL.***
3. **Cryoprecipitate** is prepared from fresh frozen plasma. It is composed of factor VIII : C, factor VIII : von Willebrand factor, 200 mg of fibrinogen, factor XIII and fibronectin in a volume of only 15 mL. It is an ideal source of fibrinogen if levels are very low with oozing from surgical incision. It is very effective for DIC. Each unit of cryoprecipitate raises the fibrinogen level by 10 mg/dL. **Coagulation factor concentrates,** such as **prothrombin complex concentrate** and specific coagulation factors such as **fibrinogen** can be corrected by administration of purified coagulation factor concentrates. **Recombinant activated factor VII A** though approved by US FDA for hemophilia has been found useful in haemorrhage in dose of 60–100 µg/kg intravenously to control DIC promptly but is very expensive. Administration of activated Protein C has been found effective in reducing mortality and organ failure in patients of DIC due to Protein C deficiency. The use of antithrombin III concentrates in patients with DIC due to septic shock has shown a modest reduction in mortality.
4. Regular monitoring by repeated measurement of global clotting tests, such as prothrombin time and activated partial thromboplastin time is useful to monitor the coagulation defect.
5. Anticoagulant therapy
   (*i*) The role and safety of heparin treatment is debatable in patients with DIC who are prone to bleeding. Therapeutic doses of heparin (5000 units IV 4–6 hourly) are indicated in patients with clinically overt thromboembolism or extensive fibrin deposition, such as purpura, fulminans or acral ischemia and in amniotic fluid embolism and retained dead fetus.

**Table 41.3: Use of Various Components of Blood and Plasma in Obstetrics**

| | Blood components | Contents | Amount in mL | Indications | Dangers | Comments |
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| 5. | Cryoprecipitate | Factors I, V, VIII, XIII (Fibrinogen 200 mg) | 15 | DIC, von Willebrand's disease, hemophilia | Hepatitis | Fibrinogen 10 mg/dL per unit infused |
| 6. | Factor concentrates | Factors VIII, IX | 20 | Hemophilia A; Factor IX deficiency (Christmas disease) | Hepatitis | 1 unit = factor activity in 1 mL of pooled plasma |

(*ii*) Patients with DIC may benefit from prophylaxis to prevent venous thromboembolism. **Recombinant tissue factor pathway inhibitor** (TFPI) is being evaluated in clinical trials. Anti-fibrinolytic agents like **epsilon aminocaproic acid** (EACA) (inhibiting plasminogen and plasmin), **trasylol** (inhibiting plasmin) and **aprotinin** (inhibiting non-specific enzymes) have a limited role in patients of postpartum haemorrhage following placental abruption with contracted uterus with normal fibrinogen levels.

6. Delivery is to be expedited once the general condition of patients is stabilized. Surgical treatment is limited to primary treatment for certain underlying etiologies like removal of retained placental bits.

**Complications**

1. Excessive bleeding can seriously complicate the clinical course of patients with DIC.
2. Organ dysfunction and limb ischemia may occur.
3. Death can occur

**Prognosis**

The prognosis of DIC depends on the underlying condition. If the underlying problem is self-limiting or can be appropriately treated, the DIC will disappear and the coagulation status will normalize.

## MATERNAL COLLAPSE IN PREGNANCY

Maternal collapse is defined as an acute event involving the cardiorespiratory system and or central nervous system resulting in reduced or absent consciousness with potential cardiac arrest and death at any stage in pregnancy and up to 6 weeks after birth. If not treated on time, maternal cardiac arrest and death can occur. Incidence is 1 in 36000 pregnancies with case fatality rate of 42%.

**Causes:** It can result from any causes which may or may not be pregnancy-related. A systematic approach must be used to identify the causes of collapse. The causes are

1. **Haemorrhage:** Antepartum haemorrhage especially abruptio placentae, placenta previa, uterine rupture and bleeding due to ectopic pregnancy (especially ruptured ectopic pregnancy) are important causes. Rarely, splenic and hepatic rupture can also occur. It is managed like APH and resuscitation with fluid and blood transfusion.
2. **Thromboembolism:** It is an important cause, necessating thromboprophylaxis. (see Chapter 30 for details)
3. **Amniotic fluid embolism:** It usually occurs during labour or after birth causing hypotension, respiratory distress and hypoxia and is discussed in Chapter 25 in details.
4. **Cardiac diseases:** It is most common overall cause of indirect maternal mortality in developed countries. (Anaemia is commonest cause in India). Certain types of heart diseases like ischemia and sudden arrythmic cardiac death can occur even in a normal heart. Mostly it occurs with known heart diseases like rheumatic heart disease (common in India). Congenital heart disease, cardiomyopathy, coronary artery disease, heart failure and pulmonary edema are other causes. It is discussed in detail in Chapter 35 (ii).
5. Obstetric sepsis especially after abortions or after delivery can cause septic shock and collapse. (see chapter 30 and above for details)
6. **Drug toxicity and overdose:** Substance abuse and therapeutic drug toxicity as with magnesium sulphate in presence of renal impairment and local anaesthetic agents and complications of systemic or epidural anesthesia (total or high spinal block) are important causes of maternal collapse. (see also chapter 25 for details)
7. **Eclampsia:** It is an important cause of maternal collapse especially in developing countries paticularly after 20 weeks of pregnancy in presence of symptoms and signs of pre-eclampsia. (See chapter 32 for details). Rarely, epilepsy especially status epilepticus can cause maternal collapse in known cases of epilepsy. (See chapter 35 (VIII)for details)
8. **Intracranial haemorrhage:** It can rarely be seen in some women especially as a complication of uncontrolled hypertension (particularly systolic hypertension). Rarely, it may result from ruptured aneurysm especially Berry aneurysm or arteriovenous malformation. It manifests as severe headache followed by maternal collapse. CT Scan usually clinches the diagnosis.
9. **Anaphylaxis:** It is a severe life threatening generalized or systemic hypersensitivity reaction causing respiratory, cutaneous and circulatory and sometimes gastrointestinal disturbances and collapse. It may be due to certain drugs, allergens or foods. It causes shock and collapse. (See above for details)
10. **Other causes:** Rarely, other causes may be responsible for maternal collapse in pregnancy as follows:
    (i) Hypoglycemia
    (ii) Hyponatremia
    (iii) Metabolic disturbances
    (iv) Electrolyte disturbances
    (v) Airway obstruction due to aspiration or foreign body
    (vi) Air embolism
    (vii) Tension pneumothorax
    (viii) Cardiac tamponade
    (ix) Aortic dissection
    (x) Hyperthermia
    (xi) Failed tracheal intubation

### Causes of increased risk of maternal collapse in pregnancy

1. Physiological and anatomical changes in pregnancy
2. Aortocaval compression by pregnant uterus

significantly reduces cardiac outflow from 20 weeks of gestation
3. Changes in lung function, diaphragmatic splinting and marked oxygen consumption makes them more susceptible to hypoxia and makes ventilation more difficult
4. Difficult intubation
5. Increased risk of aspiration
6. Risk of rapid loss of large amount of blood due to increased cardiac output and hyperdynamic circulation of pregnancy in conditions like haemorrhage

## Management

Maternal collapse in pregnancy should be managed using standard resuscitation guidelines like Adult basic life support, Adult advanced life support and Automated External Defibrillator Algorithm. If maternal collapse occurs at home or in community, management is as per availability of help like in non pregnant state and ambulance services should be used. The paramedics can resuscitate the patient as per available protocol to them and should shift the patient to hospital after putting Intravenous line and ensuring airway and ABCDE assessment given below.

Manual displacement of uterus to the left should be done to relieve aorto caval compression after 20 weeks of gestation for effective chest compression. In event of cardiac arrest, a left lateral tilt of 15 to 30 degree on a firm surface is helpful.

1. **Airway:** Airway must be immediately cleared. Intubation with a cuffed endotracheal tube should be inserted immediately by an experienced anesthetist.
2. **Breathing:** Supplemental high flow oxygen should be given to avoid deoxygenation. If intubation cannot be done then bag and mask ventilation or use of simple supraglottic airway may be used as an alternative but is inferior to intubation.
3. **Circulation:** If airway is clear but no breathing, chest compression should be started immediately. Two wide bore cannulae (minimum 16 gauge) should be inserted at the earliest. Central venous access can be considered if peripheral veins are not accessible. Aggressive fluid replacement is done. Abdominal ultrasound can be done to rule out concealed haemorrhage. Chest compressions are 100 to 120 per minute with two ventilation breaths after every 30 chest compressions are started.
4. **Defibrillator:** It can be used as in non pregnant state in selected cases.
5. **Drugs:** They are given as in non pregnant state (intravenous adrenaline). Further management is as per condition and are discussed in different chapters.

## Perimortem cesarean delivery

If there is no response to correctly performed CPR in 4 minutes, perimortem cesarean should be done within 5 minutes of collapse (see Chapter 42 for details)

## Key Points

1. Trauma in pregnancy can be due to physical abuse, sexual assault, vehicular accidents (road traffic accidents/blunt trauma and penetrating abdominal injuries). There can be placental abruption, traumatic uterine rupture or fetomaternal haemorrhage in abdominal trauma during pregnancy.
2. Management of trauma is like in non-pregnant state with extra care for fetus and judicious use of radiological investigations and taking care of fetus. Anti-D should be given to Rh– mothers.
3. Domestic violence is violence against females by family members which can start or worsen in pregnancy. It can cause antepartum haemorrhage, foetal growth restriction and perinatal death. Screening for it may diagnose it. Proper antenatal and intrapartum care can improve prognosis. Involvement of police and social agencies may be useful.
4. Sexual assault or abuse in pregnancy is usually by the husband or partner especially in unwanted pregnancies and can cause poor antenatal care with foetal growth restriction. For sexual assault, azithromycin, metronidazole and ceftriaxone are given for prophylaxis of sexually transmitted infections.
5. Cardiopulmonary resuscitation can be required in pregnancy in which uterus should be laterally displaced to avoid uterine compression.
6. Shock is a clinical condition of tissue hypoperfusion causing cellular hypoxia and organ damage and can be hemorrhagic or non-hemorrhagic shock.
7. If blood loss is severe (> 40%) or shock is left untreated in decompensation phase, it can go into irreversible phase of cellular damage with persistent constriction of post-capillary sphincters and stagnation of fluid in tissue spaces, metabolic acidosis and arteriolar dilatation.
8. Treatment of hemorrhagic hypovolemic shock is by intravenous fluid administration, blood transfusion and vasopressors and specific treatment of obstetric conditions like cesarean section for placenta previa, medical and surgical treatment of PPH.
9. Non-hemorrhagic shock can be due to difficult obstetric procedures, fluid loss, septic shock, hypertension, anaesthetic hazard, anaphylactic reaction, cardiogenic or neurogenic shock.

10. Septic shock or endotoxic shock is caused by septic conditions like septic abortion or puerperal sepsis and is managed by replacement of fluids, steroids, vasoactive drugs and combination antibiotic therapy covering all organisms and removal of infected material.
11. Neurogenic shock can be due to acute inversion, rapid evacuation of uterus, vasovagal stimulation, supine hypotensive syndromes. Treatment is by giving fluids, correction of acidosis, vasoactive drugs, steroids, ventilation and elimination of source of neurogenic stimulus.
12. Anaphylaxis can occur due to administration of drugs like iron injection, penicillin and local anesthesia. Management is by general measures, intravenous fluids, antihistamines and steroids. For local anesthesia toxicity lipid rescue therapy has been tried.
13. General management of systemic shock include intravenous fluids, blood transfusion, CVP, oxygenation, treatment of specific causes, hemostasis, early diagnosis and treatment of complications like acute renal failure, pituitary necrosis and anaemia.
14. Disseminated intravascular coagulation (DIC) is a condition of systemic activation of blood coagulation system with microthrombi formation in various organs leading to multiorgan failure and bleeding tendency. It is usually caused by abruptio placentae or other massive obstetric haemorrhage or septic conditions and pre-eclampsia. It is a serious condition with high mortality.
15. Patients of DIC have symptoms of the basic underlying disease causing DIC (e.g. pre-eclampsia, APH or PPH), bleeding tendencies, signs and symptoms of infection and symptoms due to involvement of various organs.
16. Diagnosis is from history, examination and blood tests especially deranged bleeding and coagulation tests, low fibrin levels, raised fibrin degradation products (FDPs) and raised D-dimer levels..
17. Treatment is given for underlying cause, general measures, blood transfusion and blood products. Cryoprecipitates are useful. Role of fibrin and heparin is controversial.

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Section 10
OPERATIVE OBSTETRICS

CHAPTER 42

# Operative Obstetrics

## INTRODUCTION AND GENERAL PRINCIPLES FOR OBSTETRIC OPERATIONS

Before obstetric procedures, the following general principles are followed with only a few changes as per the operation.

1. **Anesthesia and Analgesia.** Local xylocaine anesthesia or paracervical anesthesia may be adequate for minor cases. Pudendal block analgesia is suitable for many obstetric procedures like instrumental deliveries and even episiotomy stitching. Epidural analgesia is becoming popular and feasible for routine labors and can be topped up for any operative procedure including instrumental deliveries and even cesarean delivery. Many minor operations can be done under intravenous sedation with IV analgesia with tramadol or pentazocine or diclofenac. Major operations like cesarean delivery need general or spinal anesthesia or combined spinal epidural analgesia.
2. Most obstetric operations are performed by putting patients in lithotomy position.
3. **Asepsis** is to be practised. Universal precautions to be followed. Always follow no touch technique:
   (a) Surgeon should scrub properly for 2 minutes with soap or scrubbing antiseptic solution (povidine iodine scrub) and should use sterile mask, gown and quality gloves.
   (b) Vulva, vagina and perineum are to be cleaned with antiseptic solution (savlon or povidone iodine solution).
   (c) The pudenda, perineum and thighs are draped in autoclaved sheets.
4. **Evacuation of the bladder.** For ambulatory patients and minor procedures, women pass urine just before the procedure. For major operations (e.g. cesarean delivery), Foley's catheter is to be inserted using aseptic technique.
5. **Bimanual vaginal examination** is performed meticulously under all aseptic conditions. Further procedures are discussed below or in different chapters.

## (i) EARLY PREGNANCY OPERATIONS

The operative procedures performed during early pregnancy are as follows:

### 1. DILATATION AND EVACUATION

The procedure involves first dilating the cervix and then evacuating the foetal products from the uterus. It can be done as one stage procedure where cervical dilatation and uterine evacuation are performed as one operation or as two-stage procedure where gradual dilatation of the cervix is done first followed by evacuation of uterus in the second stage.

#### One stage procedure

**Indications**

(*i*) Incomplete miscarriage
(*ii*) Inevitable miscarriage
(*iii*) Missed miscarriage
(*iv*) Molar pregnancy with favourable cervix or in the process of expulsion.
(*v*) Induced abortion (Medical termination of pregnancy) in first trimester

**Pre-operative Preparation**

(*i*) It may be done under local or general anesthesia.

(ii) An ultrasound to confirm absence of foetal cardiac activity for missed abortion and to note the type of abortion.

(iii) Measurement of hemoglobin and determination of blood group for ABO and Rh typing, urine for albumin and sugar.

(iv) If os is closed, then tab. misoprostol 400 µg vaginally is given 3–4 hours before procedure.

(v) Antibiotic cover preprocedure - one dose of intravenous (cefazolin 1 gm). Postprocedure 5 days oral antibiotic like ciprofloxacin 500 mg twice daily or doxycycline 100 mg twice daily for 5 days.

**Procedure** (Fig. 42.1)

(i) Patients are put in lithotomy position after they have passed urine. The part of perineum along with lower abdomen and thighs are cleaned with savlon and povidine iodine solution. *The cleaning is started from the part to be operated and then continued laterally.* The perineum is draped with autoclaved and cut perineal sheet.

(ii) Procedure is done under aseptic conditions, bimanual examination is performed to assess the size and position of the uterus and for any findings in the fornices.

(iii) Posterior vaginal wall is retracted with Sim's speculum. Cervix is visualized after retracting anterior vaginal wall with anterior vaginal wall retractor or elevator. Anterior lip of cervix is grasped by sponge holding forceps (in gravid uterus) and with tenaculum in early pregnancy. Although volsellum (or vulsellum) is to be avoided being traumatic, practically most obstetricians use volsellum as its grip is strong. Allis forceps can also be used.

(iv) The cervix is dilated gently up to the suitable extent with gradually increasing Hegar dilators. The tip of the dilator should go just beyond internal os and the cervix should be dilated enough to admit the index finger of the operator easily. **As a precaution, the right hand's ring and little finger should rest on patient's perineum to avoid perforation by dilator.**

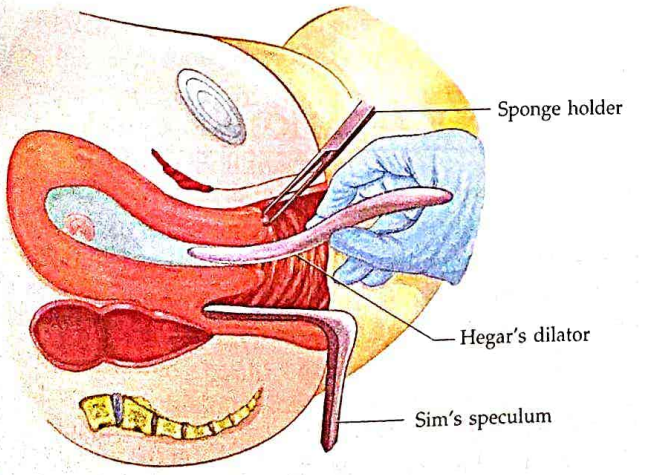



**Fig. 42.1:** Diagramatic representation of dilatation of cervix using Hegar's dilator

(v) Closed ovum forceps is passed into the uterine cavity. The blades of the ovum forceps are opened and detached pieces of products of conception are grasped with the blades. The blades are then closed and the products are removed. The ovum forceps may be rotated through right angle before withdrawing it from the uterine cavity. This ensures better grasp on the products. The ovum forceps may have to be passed many times in the uterine cavity.

(vi) When all the products of conception have been removed, a blunt curette is passed into the uterine cavity and gentle scrapping of all the walls of the uterus is done till the typical grating sensation is felt. Some obstetricians do gentle sharp curettage but it is not recommended due to risk of Asherman's syndrome.

(vii) Intravenous methylergometrine 0.2 mg may be administered during the operation for better uterine contraction and to avoid perforation.

(viii) After removing the Sim's speculum, a bimanual examination is performed and uterus massaged with vaginal fingers and abdominal hand to remove any clot.

(ix) **The completion of procedure is confirmed by grating sensation of uterus on curettage, closure of cervix and stoppage of bleeding.** Rarely ultrasound may be performed in operation theatre using portable machine to ensure complete evacuation.

(x) After ensuring uterine contractility, complete evacuation and minimal bleeding, area is cleaned and a sterile pad is given.

(xi) The products of conception or the material obtained is sent for histopathological examination in spontaneous abortions (not indicated in medical termination of pregnancy).

### Postoperative Care

(i) Pulse, BP and temperature charting are done for 3–4 hours.

(ii) Watch for vaginal bleeding.

(iii) **Anti D injection.** All Rh-negative women (if husband is Rh-positive) should receive Anti D in dose of 50 µg after first and 300 µg after second trimester abortions.

(iv) Patients are encouraged to pass urine.

(v) Antibiotics (Doxycycline 100 mg twice daily for 5–7 days) are continued along with analgesics.

(vi) Hematinics should be given for 4–6 weeks.

(vii) Contraceptive advice is given, avoid conception for 3 months.

(viii) Patients should be asked to come for follow-up with the histopathological report after next menses.

### Complications

Complications of dilatation and evacuation operation are shown in Table 42.1.

### Management of Uterine Perforation

It is suspected when instrument passes further than uterine size or omentum or bowel can be visualized. Management depends upon the site and size of perforation, general

**Table 42.1: Complications of Dilatation and Evacuation Operation**

| |
|---|
| **Early complications** |
| (i) Excessive bleeding<br>(ii) Cervical tear with or without broad ligament haematoma formation<br>(iii) Uterine perforation: Risk is < 1%.<br>(iv) Inadvertent injury to pelvic vessels, intestines or urinary bladder especially for late gestation and evacuations.<br>(v) Shock can occur due to excessive haemorrhage (hypovolemic shock) or due to forceful dilatation of cervix (one-stage operation) causing vasovagal attack or neurogenic shock.<br>(vi) Anaesthetic complications. Inadvertent intravascular injection of a local anaesthetic agent can cause serious systemic toxicity like CNS toxicity (seizures), cardiac toxicity and even mortality. |
| **Intermediate** |
| Pelvic infections; Endometritis, myometritis, perimetritis and parametritis occur in few days |
| **Delayed complications** |
| (i) Pelvic inflammatory disease<br>(ii) Hematometra<br>(iii) Secondary infertility<br>(iv) Asherman's syndrome due to uterine adhesions<br>(v) Cervical insufficiency<br>(vi) Cervical stenosis<br>(vii) Chronic pelvic pain |

condition of patients and the type of the instrument suspected to cause the perforation. Start intravenous drip with crystalloids or colloids. Arrange 2 units of blood. Administer intravenous broad spectrum antibiotics.

(*i*) Stop the procedure immediately. Start intravenous drip if not already on. Call for help.

(*ii*) For perforation caused by small blunt instruments like uterine sound or smaller Hegar dilator, expectant management and monitoring vitals like pulse and blood pressure can be done as it is unlikely to cause trauma to intraperitoneal structures. Give antibiotics.

(*iii*) For perforation due to larger Hegar dilator, ovum forceps or suction cannula, diagnostic laparoscopy should be done to localize the perforation and the amount of bleeding. The intestines and omentum are carefully visualized for any injury. Laparotomy is performed if there is doubt of injury to the bowel, bladder or blood vessels.

(*iv*) For cervical injury with broad ligament haematoma (especially supralevator) or injury of uterine artery, laparotomy and repair of tear or hysterectomy are performed.

(*v*) **Perforation before complete uterine evacuation.** One of the following managements can be performed:
(a) Perform uterine evacuation under laparoscopic guidance. (b) If laparotomy is performed for suspected injury, (*i*) Evacuation can be performed through the uterine rent which is then repaired; (*ii*) Rarely hysterectomy may be needed especially for large and irregular perforation, if woman has living children.
Sick patients should be kept admitted for a few days.

### Dilatation and evacuation as a two-stage procedure

#### Indications

1. Medical termination of pregnancy up to 12 weeks.
2. Missed miscarriage
3. Molar pregnancy with tightly closed cervix. Aim is to avoid forceful dilatation of cervix so as to minimize cervical trauma from mechanical dilatation.

#### Technique

(A) First Stage

Cervical preparation. It is rarely used now. These days misoprostol 400 µgm vaginally 3–4 hour before procedure or sublingually 1 hour before procedure is given for cervical dilatation. Only when there is contraindication to misoprostel or cervix is not responding to misoprostol, then this procedure is used. It causes less pain on insertion, has fewer side effects and is very economical and convenient. Most obstetricians use misoprostol in current practice.

First stage involves insertion of laminaria tent or synthetic hygroscopic dilator (lamicel which is a polyvinylacetate sponge impregnated with anhydrous magnesium sulphate) into the cervix for its slow and gradual dilatation.

Steps of Introduction of Tents (Fig. 42.2)

After ensuring passage of urine, parts are cleaned and draped under all asepsis. Cervix is cleaned with povidone iodine solution and grasped with a tenaculum and suitable size of the tent is chosen. The tent is inserted in the cervix using laminaria tent-holding forceps (Fig. 42.2).

(B) Second Stage—Evacuation

Evacuation is performed after at least 6 hours so that cervix is dilated. The steps of evacuation are the same as in dilatation and evacuation described above.

## 2. SUCTION EVACUATION

Suction evacuation is a technique in which the products of conception are removed from the uterine cavity using a Karman's cannula attached to a suction machine operated by electricity or battery. It is the procedure of choice for medical termination of pregnancy upto 12 weeks and for abortions.

### Common Indications

(*i*) Induced abortion (Medical Termination of Pregnancy [MTP] up to 12 weeks).

(*ii*) Incomplete miscarriage

(*iii*) Inevitable miscarriage

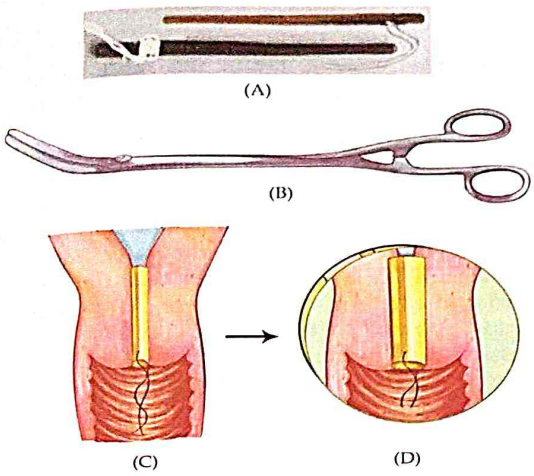



Fig. 42.2: Introduction of laminaria tent for slow cervical dilatation (A) Laminaria tent before introduction; (B) Laminaria tent-holding forceps; (C) Laminaria tent in correct position after introduction and (D) Laminaria tent causing dilatation and softening of cervix

(*iv*) Missed miscarriage
(*v*) Molar pregnancy

### Technique

(*i*) The initial steps used are similar to D and E operation mentioned above. The procedure may be done under intravenous sedation supplemented with paracervical block.
(*ii*) Before suction, dilatation of cervix can be done by slow method (misoprostol 400 µg vaginally 2–4 hours before the procedure or laminaria tents 6 hours before, as given above in D and E) or by rapid method (commonly done). In the latter, the cervix is gradually dilated with gradually increasing Hegar's dilators up to one size smaller than size of suction cannula.
(*iii*) The most appropriate sized suction cannula is inserted into the uterine cavity with the tip placed in the mid uterus. The size of cannula is equal to number of weeks of gestation or one less (e.g. no. 8 cannula for 8 to 9 weeks pregnancy). The cannula is attached to suction machine.
(*iv*) The suction pressure is kept at about 500 mm Hg. The suction cannula is moved upward towards the fundus and then back towards the os and suction is then performed circumferentially (360°) in whole of the uterine cavity (Fig. 42.3). The contents of the suction bottle should be examined for the products of conception and placental tissue (soft, fluffy and feathery).
(*v*) The complete evacuation is ensured by:
  (a) No more tissue is aspirated
  (b) Completely evacuated and contracted uterus grips the cannula.

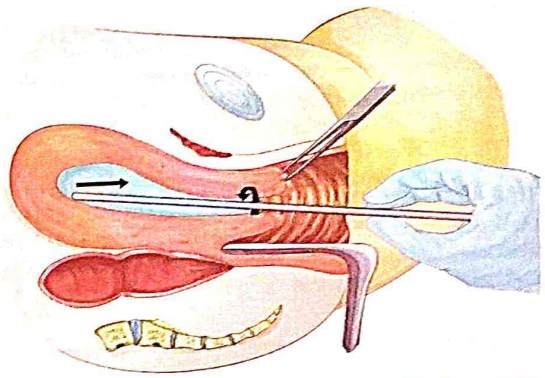


Fig. 42.3: Diagramatic representation of suction and evacuation being performed using a Karman's cannula

  (c) Grating sensation of cannula
  (d) Visible bubbles in the suction cannula.
(*vi*) While taking out the suction cannula from uterine cavity through cervix, the suction should be switched off to avoid injury of internal os and cervix.
(*vii*) The procedure may be completed by gentle blunt curettage to remove any remaining placental or foetal tissue as described above for D and E. As per WHO, routine curettage is not warranted.

### Complications of suction evacuation

The complications of suction and evacuation are shown in Table 42.2.

**Table 42.2: Complications of Suction and Evacuation**

| |
|---|
| **Early Complications** |
| (i) Heavy bleeding. But it is much less than in D and E. |
| (ii) Cervical injury |
| (iii) Uterine perforation |
| **Late Complications** |
| (i) Pelvic inflammatory disease |
| (ii) Secondary infertility |
| (iii) Cervical insufficiency |
| (iv) Uterine synechiae formation (Asherman's syndrome) |

## 3. DILATATION AND CHECK CURETTAGE (D AND C)

Dilatation and check curettage is done in cases of retained products of conception under all aseptic precautions and antibiotic cover. Suction cannula is not used. Gentle curettage is done and tissue is sent for histopathological examination.

The indications, procedure and complications are similar to dilatation and evacuation.

- The uterus contracts around or grips the cannula.
- The patient complains of cramping or pain, indicating that the uterus is contracting.

Complications are like suction and evacuation mentioned above.

## CONTRACEPTION FOLLOWING MISCARRIAGE OR ABORTION

Effective contraception should be initiated soon after evacuation and after proper counselling. Intrauterine device can be fitted in the same sitting. Laparoscopic or minilap ligation can be performed if family is completed after counselling and proper consent. Avoid conception for 3 months in case of incomplete, inevitable or missed abortion and if recurrent pregnancy loss (RPL) with > 2 abortions. Then work up need to be done before next conception. Oral pills can be started on the same day. Injection DMPA (depot medroxyprogesterone acetate) can also be given at the same time.

## Key Points

1. Vaginal examination is performed after cleaning the parts with antiseptic solutions followed by the definitive procedure.
2. The various early pregnancy operations are dilatation and evacuation, suction and evacuation (mostly preferred), dilatation and curettage (rarely done these days) and manual vacuum aspiration. They are performed in lithotomy position, after emptying the bladder, under aseptic conditions.
3. Suction and evacuation is the most preferred operation these days for early pregnancy complications like incomplete abortion, inevitable abortion and medical termination of pregnancy. Before suction, dilatation of cervix is performed either as rapid method or as slow method (use of misoprostol or laminaria tents). Evacuation is then performed using Karman's cannula and suction apparatus. Gentle curettage may be performed in the end to ensure complete evacuation which is made out by grating sensation of uterus which is well contracted with no more products coming out.
4. The early complications are excessive bleeding, injury to cervix, shock, uterine perforation, sepsis and haematoma. The late complications are pelvic inflammatory disease, cervical incompetence and uterine synechiae (Asherman's syndrome). Uterine perforation seen in < 1% cases is a serious complication. It can be managed conservatively if caused by sound or small dilator. However, for perforation caused by large dilator, cannula or curette, laparoscopy or laparotomy is performed as there is risk of bowel or vascular injury. The procedure of evacuation is completed under laparoscopic guidance. Adequate hydration, blood transfusion and intravenous antibiotics are given.
5. Menstrual regulation (aspiration) can be used for medical termination of pregnancy up to 7 weeks of pregnancy using a 50 mL syringe and 5–6 mm cannula.
6. Manual vacuum aspiration is office based procedure using a hand operated 60 mL syringe and a cannula which does not need suction apparatus and electricity and hence is useful in Indian rural settings. It can be performed safely up to 10 weeks (but can be used up to 12 weeks).

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3. American College of Obstetricians and Gynecologists. Medical Management of first trimester abortion, Practice Bulletin No. 143, March 2014, Reaffirmed 2016
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5. World Health Organization Clinical Practice Handbook for Safe Aboriton, Geneva, WHO, 2014
6. American College of Obstetricians and Gynecologists: Induced abortion. In Guidelines for Women's Health Care, 4th edn, Washington 2014.
7. American College of Obstetricians and Gynecologists: Abortion training and education, Committee Opinion no. 612, November 2014, Reaffirmed 2017.

## Examination Questions

A. Enumerate various early pregnancy operations. What are the indications of performing suction and evacuation? Describe the procedure and enumerate its complications.

B. Write short notes on:
   1. Rapid dilatation and evacuation
   2. Two-step dilatation and evacuation
   3. Suction and evacuation (Delhi University and other universities, many times)
   4. Manual vacuum aspiration (Delhi University)
   5. Management of uterine perforation during evacuation
   6. Menstrual regulation

# (ii) OBSTETRIC FORCEPS AND VENTOUSE

## Introduction

Instrumental delivery is an art which should be learnt by all obstetricians for optimum maternal and perinatal outcomes. But at the same time, they should be aware of its limitations and should avoid difficult instrumental delivery in favor of cesarean delivery. Due to fear of medicolegal cases and lack of training, the incidence of forceps deliveries has been falling throughout the world. **In USA, operative deliveries decreased from 9% in 1990 to 3.2% in 2014.** Vacuum was used more commonly as compared to forceps (5:1 ratio) in USA. Exact incidence in India is not known but has decreased significantly. It was 9.31% forceps delivery in AIIMS (Annual statistics 2018) Due to less use of operative vaginal delivery, training is lacking in most hospitals especially for forceps (particularly, rotational forceps). Use of formal training programs of operative vaginal delivery including a manikin and pelvic model can increase use of operative delivery and can decrease complications.

## OBSTETRIC FORCEPS

### Definition

**Obstetric forceps is an instrument designed to assist in the extraction of foetal head accomplishing delivery of the fetus presenting by the vertex (most common), face (mento-anterior) or the after coming head of breech.**

**History:** History of the obstetric forceps is very fascinating. It was first devised in the early 17th century (around AD 1600) by either Peter I or Peter II of the Chamberlain family of England. Three generations kept the use of forceps a secret in the family from 1600 to 1728. The Chamberlain's forceps only had a cephalic curve. Levret from Paris (France) in 1747 added a pelvic curve to the forceps blade. Tarnier a Frenchman in 1877 developed the axis traction device. Since then there have been various modifications. De Lee (USA), in 1920, introduced the concept of prophylactic forceps. William Smellie from England first used forceps for the after coming head of breech. Keilland, a Norwegian obstetrician, introduced the rotational Kielland forceps in 1916, for deep transverse arrest. Kedarnath Das from Calcutta (now Kolkata) wrote a treatise on the subject in 1929 and gave the modified form of forceps (Das's forceps) which are suitable for comparatively small pelvis and small baby of Indian women.

### Components

#### Measurements

**Das forceps.** The total length of forceps is 37 cm; distance between the tips of blades is 2.5 cm and maximum diameter between the two blades is 9 cm.

#### The blades

The obstetric forceps have two blades crossing each other and are called the left or lower blade and the right or upper blade, depending on the side of the pelvis to which they are applied. Each blade is made up of four parts; the fenestrated blade proper, the shank, the lock and the application handle (Fig. 42.5). Tucker-McLane forceps (available in USA, but not in India) has solid blades. In some types, there is, in addition, traction rods and a traction handle.

Each blade is fenestrated for a better grip on the foetal head. The lower part of fenestration of blade has got a slot for fitting the axis traction rod, if needed.

The blade has two curves (Fig. 42.5).

1. **Pelvic curve.** The pelvic curve on the edge of blades conforms to the axis of the birth canal (curve of Carus). It forms a part of a larger circle whose radius is 17.5 cm. The frontside of the forceps is the concave side of the pelvic curve.
2. **Cephalic curve.** It is the curve on the flat surface of blades which conforms to the shape of foetal head and when articulated grasps the foetal head without compression. The radius of the curve is 11.5 cm.

#### The shank

The shank connects the blades and the handle and gives the instrument its length. The shank is parallel in the straight forceps, e.g. Simpson's forceps or crossing as in Tucker-McLane forceps.

#### The lock

The two blades articulate at the lock. Most forceps have the **English lock** which is simpler and more efficient and allows the shank of one blade to slip into the socket of the other. In this type of lock, left blade has to be inserted first. The **French lock** is a pivot lock and is rarely used.

#### Handles

The two handles are apposed on articulation of the two blades. Its length is 12.5 cm. There is a finger guard to provide a firm grip on the forceps for the operator. Whenever forceps have to be applied with the head above

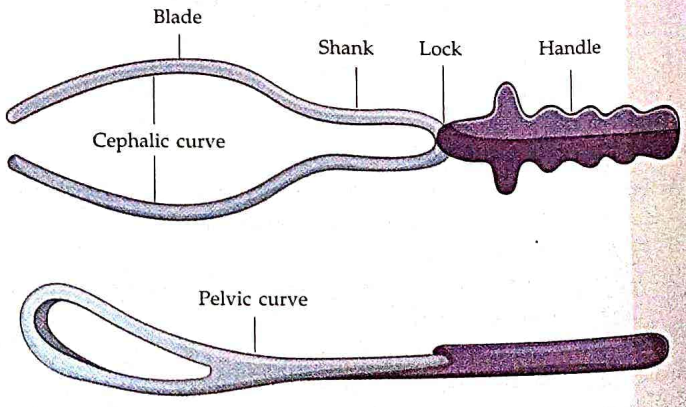



**Fig. 42.5:** Components of forceps

the level of the ischial spines (rarely these days), axis traction forceps, e.g. **Milne Murray's forceps** may have to be used.

In India, **Wrigley's** outlet forceps are most commonly used while **Das** forceps or **Neville Barne's** forceps are used as low mid-cavity forceps.

### Identification of the Blades

ON ARTICULATION OF BLADES

The articulated forceps are put before the pelvis with the tips of the blades directing upwards and the concavity of the pelvic curve being in front. The blade which corresponds to the left side of the maternal pelvis is the left blade, while the one corresponding to the right side is the right blade.

INDIVIDUAL BLADES

The tip of the blade should direct upwards. The cephalic curve directs inwards and the pelvic curve directs forwards.

## Types of forceps

Various types of forceps are shown in Figs. 42.6 A, B and C and Table 42.3.

| Table 42.3: Types of Forceps |
|---|
| **1. Traction forceps** |
| (a) Short—Wrigley's outlet forceps **(Fig. 42.6 A)**, Simpson's short forceps<br>(b) Long—Simpson's long forceps **(Fig. 42.6 B)**, Das's forceps, Elliot's long forceps, Neville Barne's forceps, Haig Ferguson's forceps<br>(c) Axis traction—Tarnier's forceps, Milne Murray's forceps |
| **2. Rotational forceps** |
| (a) Kielland's forceps **(Fig. 42.6 C)** for deep transverse arrest<br>(b) Barton's forceps for transverse arrest in a flat pelvis |
| **3. Special forceps** |
| (a) Piper's forceps for the after coming head of breech |

## Short Curved Obstetric Forceps (Wrigley's) (Fig. 42.6 A)

It is a small, short and light forceps. The instrument is short due to decrease in the length of the shanks and handles. It has a large cephalic curve but a small pelvic curve. Simpson's short forceps are also **outlet forceps**.

## Classification of forceps

American College of Obstetricians and Gynecologists (2007) has classified forceps into mid, low and outlet forceps. An adapted classification is shown in Table 42.4 and Fig. 42.7.

## Forces and functions of forceps

Forceps can provide various forces to aid delivery of head as follows:

### 1. Traction

Traction is the most important function of the forceps. **Traction required is about 18 kg in primigravidas and about 12 kg in multigravidas.**

### 2. Compression

Forceps cause mild compression of foetal head and aid in delivery. It should, however, be minimal to avoid trauma to foetal head.

| Table 42.4: Classification of Forceps and Vacuum Delivery |
|---|
| **A. Outlet forceps** |
| 1. Foetal scalp visible at introitus without separating the labia<br>2. Foetal skull has reached pelvic floor<br>3. Sagittal suture is in anteroposterior diameter or right or left occipito-anterior or posterior position (within 45°)<br>4. Foetal head is at or on the perineum |
| **B. Low forceps** |
| 1. Leading point of foetal skull (not caput) is at station > + 2cm and not on pelvic floor<br>(a) Rotation is 45° or less from occipito-anterior position<br>(b) Rotation is greater than 45° including occipito-posterior position |
| **C. Mid forceps** |
| Leading bony point of skull (station) is between 0 and + 2 cm. Rotation can be 45° or more. |
| **D. High forceps** |
| Head not engaged. Station above ischial spines not included in classification and not recommended. |

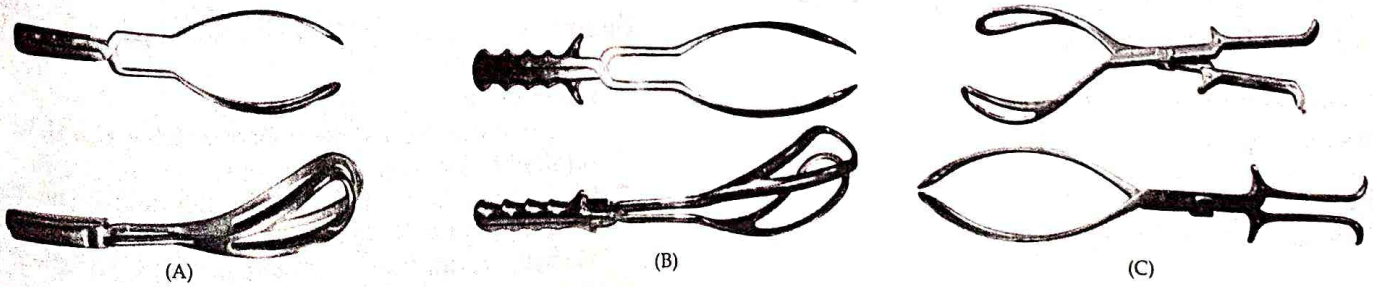



**Fig. 42.6:** Various types of forceps (A) Wrigley's outlet forceps showing cephalic and pelvic curves; (B) Simpson's long curved forceps showing cephalic and pelvic curves; (C) Kielland's forceps

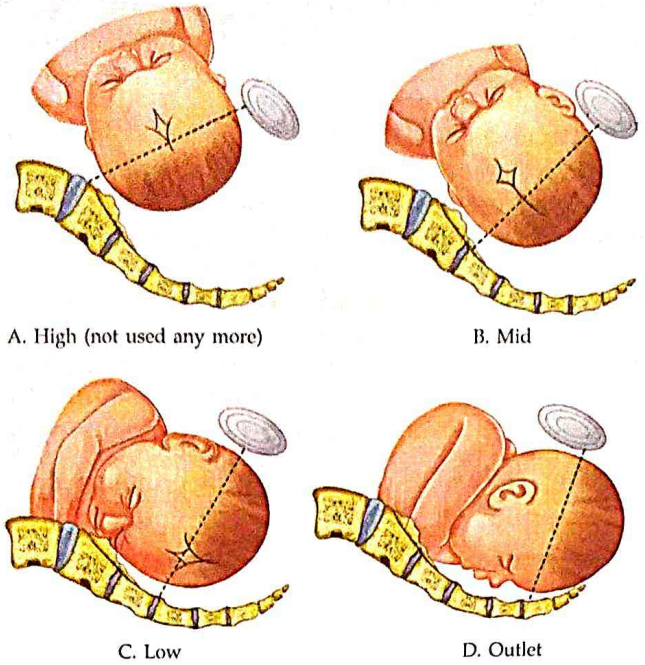



**Fig. 42.7:** Types of forceps delivery

### 3. Rotation

Kielland's forceps are used for rotation of the foetal head. However, in the low forceps operation with the sagittal suture placed obliquely, with the occiput placed within 45°, cephalic application of forceps can cause some rotation. Due to lack of expertise and difficult procedure, these are rarely used in current practice.

### 4. Protection

The forceps blades can provide a protective cage for foetal head from the pressure of the parturient canal which is useful in preterm baby (not used these days). They also control the delivery of the after-coming head in breech presentation reducing sudden decompression.

### 5. Miscellaneous

(*i*) Single forceps blade can be used as a vectis to assist delivery of the head in cesarean delivery.

(*ii*) One forceps blade may be used to explore cervical tears and high vaginal lacerations by retracting vaginal walls away from the tear site.

## Indications and Contraindications

The indications and contraindication of forceps delivery are shown in Table 42.5.

## Pre-requisites of Forceps

The pre-requisites of forceps are shown in Table 42.6.

## Ways of applying forceps

There are two ways of applying forceps. They are:

### 1. Cephalic Method

In cephalic method, the blades are applied in the biparietal diameter irrespective of its position in relation to the maternal pelvis where each ear will be in the center of each fenestra of the forceps. This causes compression in the biparietal diameter where it causes least harm. The blades fit much better and do not tend to slip with a cephalic grip. **This is the preferred method.**

**Table 42.5: Indications and Contraindications of Forceps Delivery**

| **Indications** |
|---|
| **I. Foetal indications** |
| Foetal distress in the second stage of labour with prospects of safe vaginal delivery, e.g. cord prolapse, persistent type II decelerations in second stage. |
| **II. Maternal indications** |
| 1. Maternal exhaustion<br>2. To cut short the second stage of labour. They are relative indications:<br>(i) Pre-eclampsia, eclampsia<br>(ii) Heart disease<br>(iii) Severe anaemia<br>(iv) Sedated patients<br>(v) Other ailments contraindicating pushing<br>3. Also applied to after-coming head of the fetus in breech delivery (Piper's forceps or other mid-cavity forceps)<br>4. Prolonged second stage<br>5. Failure of satisfactory advancement of head for a period of more than 4 hours with epidural analgesia and 3 hours without epidural in primigravidas and 3 hours and 2 hour respectively in multigravidas (ACOG 2020).<br>6. Face anterior presentation<br>7. Previous cesarean delivery |
| **Contraindications** |
| 1. Unengaged foetal head<br>2. Non-willing patients<br>3. Osteogenesis imperfecta in the fetus<br>4. Bleeding disorders of the fetus.<br>5. Position of foetal head not certain<br>6. Inexperienced operator<br>7. If pre-requisitions are not fulfilled. |

### 2. Pelvic Method

In pelvic method the blades are applied with reference to the maternal pelvis on either side. It may cause undue trauma to the foetal head and is avoided.

## Techniques of application of forceps in low forceps delivery

1. Preliminaries
   All pre-requisites as mentioned in Table 42.6 should be fulfilled before applying forceps.
2. Check the indication for application and ensure that a written, informed consent has been taken from women. Verbal informed consent can be taken in emergency situation.
3. If good uterine contractions are not present, start oxytocin drip.

**Table 42.6: Pre-requisites of Forceps**

The pre-requisites of forceps can be remembered by the mnemonic '**FORCEPS**' as follows:

1. **F: Fully** dilated cervix
2. **O: Occipito-anterior, occipito-posterior** and **other** suitable presentations (mento-anterior and after-coming head of breech)
3. **R: Ruptured** membranes
4. **C: Consent** should be obtained. **CPD** (cephalopelvic disproportion) should be ruled out.
5. **E: Effective** uterine contractions, **empty** bladder, **engaged head, episiotomy, epidural** or regional analgesia, **experienced** operator (obstetrician). Forceps or ventouse should only be applied by an operator with adequate knowledge, skills and experience of it. Midpelvic or rotational forceps should be applied only by experienced operator (consultant or trained senior resident) (RCOG 2020).
6. **P: Position** of head must be known. **Pediatrician** to be present. **Patient in lithotomy position.** Place forceps or ventouse delivery have a higher failure rates and should be considered a trial and should be attempted only in place with immediate facilities for cesarean delivery.
7. **S: Sagittal** suture should be in antero-posterior diameter (for outlet forceps) or within 45° (for low and mid forceps), **Stirrups** and lithotomy position, **suitable** and legitimate indication should be there.

4. Patients should be brought to the edge of bed, put in lithotomy position with appropriate preparation and drapes.
5. Bladder should be catheterized.
6. Vaginal examination is performed to know the position, station and presentation of head, cervical dilatation, status of membranes and pelvic size and shape. One should abandon forceps if pre-requisites are not fulfilled or if they are deemed difficult. ACOG (2015) recommends abandoning the procedure if satisfactory progress is not made.
7. Appropriate anesthesia should be given. Pudendal block and perineal infiltration is appropriate. Epidural anesthesia if already given can be topped up.
8. Ensure that uterus is contracting and relaxing as a safeguard against postpartum haemorrhage.
9. Select the appropriate forceps for the application (**Wrigley's** or short **Simpson's** for outlet and **Long Simpson's** or **Das'** for low forceps).
10. A generous mediolateral episiotomy is performed if needed. It can also be given at crowning of head to avoid haemorrhage. The mediolatral epithotomy should be given at 60° angle. It prevents obstetric and sphincter injury in instrument delivery (RCOG 2020).
11. An assistant and the pediatrician should be present before commencing the procedure.

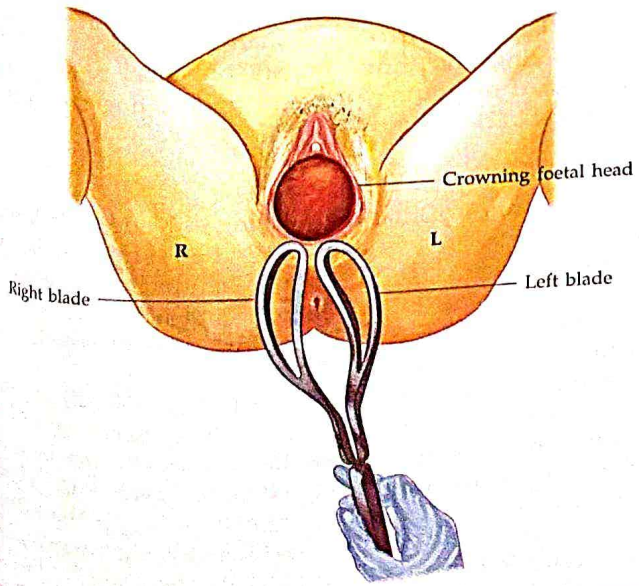



**Fig. 42.8:** Identification of forceps blades

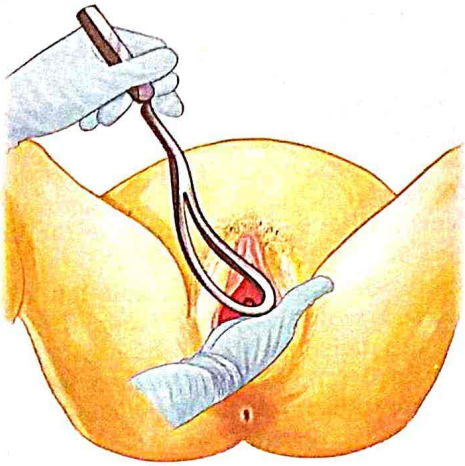


**Fig. 42.9:** Introduction of left blade of forceps under guidance of right hand.

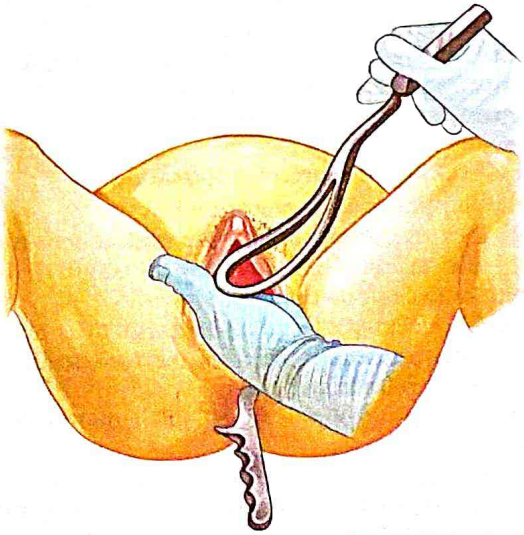


**Fig. 42.10:** Introduction of right blade of forceps

12. A single prophylactic dose of intravenous amoxicillin and clavulanic acid should be given following forceps or ventous delivery as it significantly reduces maternal infection (RCOG 2020)
13. Good standards of hygiene and aseptic techniques are recommended

## Procedure

### Step 1: Identification and Application of the Left Blade (Fig. 42.8 and 42.9)

1. Blades are identified as right and left after holding them locked with the pelvic curve directed upward towards the patient in the position in which they will be when finally applied. Left blade is inserted first.
2. Two or more fingers of the right hand are introduced inside the left posterior portion of the vulva and into the vagina beside the foetal head to guide the blade during application and protect the vaginal wall from its injury. The handle of the left forceps is then grasped between the thumb and two fingers of the left hand as if holding a pen keeping it parallel to the left inguinal ligament. The tip of the blade is gently passed into the vagina between the foetal head and the palmar surface of the fingers of the right hand (Fig. 42.9).
3. Initially the handle and the blade are held nearly vertically. As the blade fits on the foetal head, it is depressed to a horizontal position. Assistant may or may not be asked to hold the blade.

#### Application of the Right Blade (Fig. 42.10)

For application of the right blade, two or more fingers of the left hand are inserted into the right, posterior portion of the vagina as a guide for the right blade which is held in the right hand and introduced into the vagina in the same way like the left blade. The position of the blade is shown in Fig. 42.11.

### Step II: Articulation (Locking) of the Blades (Fig. 42.12)

With correct application, the blades lock perfectly especially if the handles are gently depressed and with a gentle maneuvering of the blades. In case of difficulty in locking, the blades should be withdrawn and reintroduced after confirming the suitability of application. One should never try to forcefully lock the blades.

### Step III: Checking of Correct Application

Appropriate application is confirmed by:

1. Blades lock readily with little separation between them.
2. The blades are equidistant from the sagittal suture in occipito-anterior position.
3. In the occipito-posterior position, the blades are equidistant from the midline of the face and brow and are widely separated.
4. Suitable gripping of the foetal head on the biparietal diameter

### Step IV: Holding of the Articulated Forceps during Traction (Fig. 42.12)

The handle is gripped by putting the middle finger of the right hand in between the shanks of blades of forceps with the ring and index fingers on either side on the finger guard or putting ring and middle finger on either side (Fig. 42.12).

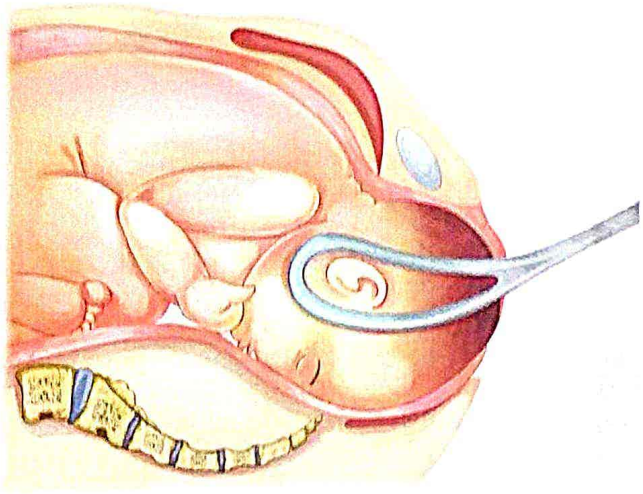


**Fig. 42.11:** The ideal position of the blades relative to the foetal head

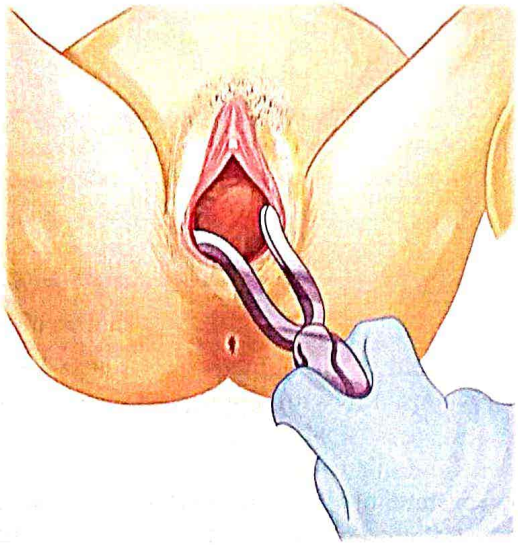


**Fig. 42.12:** Locking of blades and downward traction

### Step V: Traction (Figs. 42.12–42.15)

After proper application and locking of blades, gentle and intermittent traction is exerted. If necessary, rotation to occipito-anterior is performed before traction is applied. Direction of forces in mid forceps and outlet forceps are given in Fig. 42.15. In mid forceps delivery **downward traction** is given first (Fig. 42.12).

**Horizontal traction** is then applied (Fig. 42.13) and the handles are gradually elevated, eventually pointing almost directly upwards. As the handles are raised, the head is extended. Finally **upward traction** is given (Fig. 42.14) in which the four fingers of right hand grasp the upper surface of the handles and shanks, while the thumb puts force on the lower surface. As the vulva is distended by the occiput, a liberal episiotomy is usually performed. During the birth of the head, spontaneous delivery should be

allowed as much as possible. Delivery should be slow and gentle to avoid undue head compression. Traction should, therefore, be intermittent and the head should be allowed to recede during the intervals as in spontaneous labour. In cases of acute foetal distress, delivery should be hastened.

After the vulva has been well distended by the head, the delivery is usually completed by keeping the forceps in place to control the advance of the head. However, the thickness of the blades may increase the likelihood of laceration of vulva or need of a large episiotomy. In such cases, the forceps may be removed and delivery completed by the **modified Ritgen maneuver** by extending and pulling head up with right hand.

After the birth of the foetal head, routine intramuscular oxytocin is administered at the delivery of the anterior shoulder. Routine intramuscular methylergometrine is usually avoided. Episiotomy is stitched in the appropriate manner. Cervical and vaginal exploration is done due to increased chances of lateral wall and other tears. The tears should be sutured properly.

## Outlet Forceps Delivery

The outlet forceps application is now favored in most of the situations as the application and traction are easy. There is minimal trauma to the fetus and maternal soft tissues.

### Procedure

The patients are placed in the lithotomy position and brought to the edge of the table. Parts are cleaned with antiseptic solution and draped. Local analgesia (anesthesia) is obtained with perineal infiltration of 10–15 mL of 1% lignocaine. Pudendal block analgesia may also be given. Vaginal examination is done to fulfill all the pre-requisites and confirm that the head lies in the pelvic outlet with the

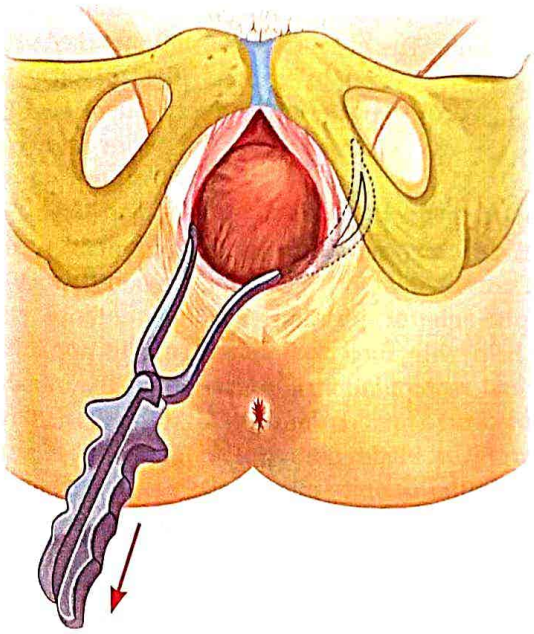


**Fig. 42.13:** Horizontal traction

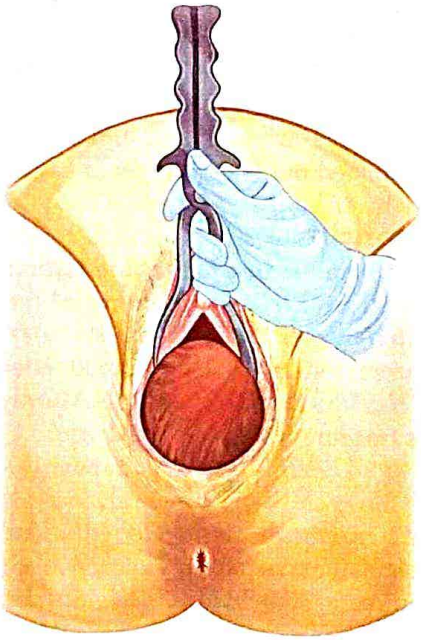


**Fig. 42.14:** Upward traction

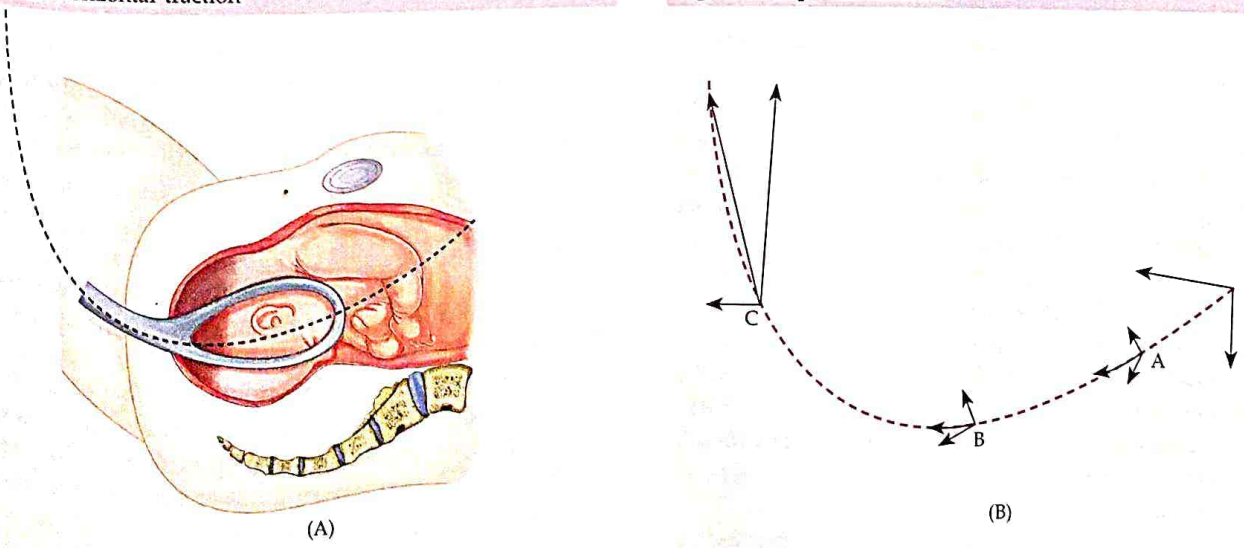



**Fig. 42.15:** Circle or Clock model of direction of forces in forceps and ventouse delivery in clockwise fashion in actual patient [Fig. (A)] and on clock model [Fig. (B)]. Point A: Low and mid forceps application: First downwards and backwards (point A), then forwards (point B) and finally forwards and upwards (anterior) (point C); Point B: Outlet application: First forwards (outwards) (point B) then forwards (outwards) and upwards (anterior) (point C)

parietal eminences lying below the ischial spines. The scalp is visible during the contractions through the introitus.

The internal rotation is completed and the sagittal suture is felt in the antero-posterior diameter of the outlet. The introduction of the blades is like that described in the occipito-anterior forceps. The locking of the blades is usually easy. If there is any difficulty, the handles should be brought down pressing over the perineum. Otherwise, the blades are removed and vaginal examination is repeated to confirm the full rotation of the head. This should be corrected and the blades (its outlet forcep) reinserted.

**Wrigley's** or **Simpson's** forceps are commonly used when the head is on the perineum as there is no fixation screw in them and the operation is easy. The handles of the forceps are held with the operator's right hand and traction made during a uterine contraction. The traction is applied **downward** and **backwards** till the head is crowning, then horizontally and finally **forward and upwards** (Fig. 42.15). These series of directions of pull are followed in gentle succession and usually completed with a single, continuous pull. During the procedure, liberal episiotomy can be given at the right time, i.e. when the head is crowning and the perineum is thinned out.

## Mid forceps delivery

***The most common indication of mid forceps delivery is after manual or Kielland rotataion of foetal head in occipito-posterior position or deep transverse arrest.*** The commonly used forceps is long curved one (**Das, long Simpson's, Neville Barne's or Milne Murray's**) with or without axis traction device.

**Steps**

1. Epidural anesthesia is preferred. Rarely general anesthesia may be required.
2. **Introduction of the blades.** The introduction of the blades is performed after correcting malrotation.
   (a) **Without axis-traction device.** The application of forceps is like outlet forceps mentioned above. The assistant should hold the left handle after its insertion.
   (b) **With axis-traction device.** The left blade is inserted first with the traction rod being kept in close contact with the shank but behind it. After the complete insertion of the blade, the traction rod should be allowed to hang down behind the shank with the assistant holding the application handle to maintain the blade in position. Then the right blade is introduced with the traction rod being kept and maintained in front of the shank until the complete introduction and locking of blades. Traction rod is then allowed to fall backwards so that both rods lie together behind the shanks.
3. **Direction of Traction**
   (a) **Without axis-traction device.** The direction of force is first **downwards** and **backwards**, then **horizontal** and finally **upwards** and **forward** (Fig. 42.15).
   (b) **With axis-traction device.** The direction of forces is as above with the traction rods always remaining parallel and in contact with the shanks. Traction rods should follow the blades during delivery of head. The tractionrods should be removed on delivery of the occiput.

## Forceps in occipito-posterior positions of the vertex (face-to-pubis delivery)

Manual rotation is usually recommended before the forceps is applied. Sometimes, in a case seen late in labour when the head has become jammed in the pelvis and a large caput has developed. The delivery is then performed by long forceps application (Das or Neville Barne's forceps) in the occipito-posterior position. The blades are applied correctly and generous episiotomy is given. Forward traction is given until the nose is visible under the pubic symphysis. The handle should then be elevated till the occiput emerges over the anterior margin of the perineum. The nose, face and chin are then delivered (face-to-pubis delivery) by the downward forces on the forceps. In occipito-posterior delivery, there is a greater tendency for severe lacerations of the perineum to occur.

## Forceps in Face Presentations

Where the chin is anterior, spontaneous delivery is most likely. Occasionally, for the same reasons which delay an occipito-anterior position, a mento-anterior casemay require help with forceps. Utmost care in application of forceps and manipulation is necessary in the introduction of the blades to avoid damage to the soft parts of the foetal face. The blades are applied along the side of the head, along the occipito-mental diameter with the pelvic curve towards the neck. Downward traction is exerted till the chin appears below the symphysis pubis. The face is then slowly delivered by upward traction with the nose, eyes, brow and occiput delivering in succession. Cesarean delivery should be done for mento-posterior presentation.

## Forceps in Brow Presentation

**Forceps are contraindicated in a brow presentation** unless the brow has been converted into a face or a vertex presentation. Cesarean delivery is performed for persistent brow.

## Forceps to the After-coming Head

The forceps may be applied to the after-coming head of breech in one of these two ways.

1. The body of the child may be carried upwards towards the mother's abdomen and the blades of the forceps introduced below the trunk. But excess elevation is avoided to prevent cervical spine injury. The operator then applies traction on the forceps.
2. Occasionally the forceps may have to be applied in front of the trunk, in those cases where the occiput is posterior.

**Piper's** forceps are meant for after-coming head of breech being long and straight. As they are not usually available in India, any mid cavity forceps can be used.

## Forceps to the decapitated head

In some cases, forceps may have to be applied to deliver the decapitated head. Usually, the decapitated head can be easily delivered by fundal pressure and a finger is introduced into the mouth in a manner similar to delivery of the after-coming head of a breech presentation. When this fails, it is advisable to grasp the head with a volsellum, steady it by fundal pressure and apply forceps. If the head cannot be delivered with easy traction, there should be no hesitation in perforating the vault of the skull and then delivering the head.

## Kielland's Forceps

In 1915, Kielland (also spelt as Kjelland) of Norway introduced a new forceps which has been widely used in Europe. The special features of this forceps are as follows:

1. It has a sliding lock which permits satisfactory cephalic application even with gross asynclitism.
2. It has little or no pelvic curve, simplifying its application of rotation.
3. On each handle, a knob indicates the direction of the occiput.

### Method of Application

The pre-requisites are fulfilled and patients' parts are cleaned and draped. **Large medio-lateral episiotomy must be given in all cases.**

#### Identification of the Blades

The articulated blades are held in front of the vulva. The concavity of the slight pelvic curve should be towards the occiput side. Small knob on each blade points towards the occiput. The blades are designated as anterior and posterior. The anterior blade is inserted first.

#### 1. The Wandering Method (Popular Method)

The anterior blade is introduced at the side of the pelvis over the brow or face along the palmar surface of the left hand. The blade is then slided upwards until it comes to lie along the side of the head. The posterior blade is held gently in an almost vertical position with the handle upwards. The fingers of the left hand are inserted into the vagina to reach the posterior surface of the head and the posterior blade is guided to its correct position in the hollow of the sacrum under guidance of the right hand. The handles are depressed slowly and aligned to correct the asynclitism. Once a correct application is made, gentle traction and rotation are started.

#### 2. Direct Method

In direct method anterior blade is put directly with concavity of anterior blade over the foetal head followed by posterior blade in the usual manner.

#### 3. Old Classical Method (Obsolete)

In old classical method, anterior blade is put with direction anteriorly and then rotated 180° to fit on foetal head. It can cause uterine rupture and is now obsolete.

### Limitations

It needs great expertise to apply the Kielland's forceps which is lacking due to lack of training.

### Hazards

#### Foetal

Foetal hazards include facial abrasion, lacerations, facial nerve palsy, depressed skull fractures and intracranial haemorrhage.

#### Maternal

Maternal hazards include vaginal lacerations, vaginal spiral tears, complete perineal tear, cervical tear and uterine rupture etc.

### Current Status of Kielland Forceps

Due to lack of training, Kielland forceps are rarely used in India now as they can be potentially dangerous instrument in inexperienced hands. In fact, some obstetricians feel that the only role of Kielland forceps in modern obstetrics is in museums. Cesarean delivery is usually performed for most of their indications and ventouse is applied for deep transverse arrest.

Hay's forceps are low forceps used for deflexed head. Nargolkar forceps are indigenous forceps designed by Dr. Subhash Nargolkar, Pune, India.

## Types of Forceps Application

### The prophylactic forceps

The term prophylactic forceps has been used to indicate delivery by forceps when there are no strict indications. It was popularized by De Lee from USA. Theoretical advantage of prophylactic forceps is to avoid the damage to the baby's head if it is left on the perineum for a long time and to the pelvic floor by the continued pressure of the head. The various indications are: (1) Severe pre-eclampsia and eclampsia (2) Cardiac disease (3) Past cesarean section (4) Post maturity (5) Small for date fetus (6) To avoid the painful second stage (7) Patients under epidural analgesia. However, there is no proof that prophylactic forceps prevent maternal and foetal damages and they are rarely used for this indication these days.

### Indicated forceps

The term 'indicated forceps' is used when it is applied only for suitable indications. Discontinue attempted forceps if birth is not imminent following three pulls of a corectly applied instrument by an experienced operator (RCOG 2020). Discontinue attempted forceps when forceps cannot be applied easily, the handles do not lock easily or there is lack of progressive descent with moderate contractions (RCOG 2020). Discontinue rotational forceps if rotation is not easily achieved with gentle pressure (RCOG 2020).

### Trial of forceps

**Trial of forceps is a type of the forceps application in which the operator anticipates the possibility of failure and makes advance preparations for cesarean**

**delivery.** It is performed in a case of suspected mid-pelvic contraction in an operating room. The procedure is abandoned if the forceps cannot be applied satisfactorily or there is no descent with gentle downward pull and the delivery is accomplished by cesarean section. It can thus avoid unnecessary cesarean delivery and difficult vaginal deliveries.

### Failed forceps

**The term 'failed forceps' is applied when the forceps application fails to deliver the baby and this failure was not anticipated.**

#### Causes and Management of Failed Forceps

The causes and management of failed forceps are shown in Table 42.7.

#### Prevention

Failed forceps can be prevented. The women needing forceps delivery should be thoroughly checked and selected.

## Causes of difficulty in forceps application

The causes of difficulty in forceps application are shown in Table 42.8.

## Medicolegal Aspects

Although most cerebral palsies or neurological damages in the newborn do not occur due to forceps, the obstetricians are often blamed for them. Such a litigation may run in millions of rupees. Proper notes should be made as follows:

1. Informed consent of patients with signatures of witnesses should be recorded in the case sheet.
2. Indications for forceps delivery should be outlined to patients and written in the record.
3. Proper notes of vaginal findings should be made.
4. Do not re-apply the forceps if once failed.
5. Foetal condition should be recorded.
6. Neonatal outcome like APGAR score, scalp pH should be recorded.

**Table 42.7: Causes and Management of Failed Forceps**

| |
|---|
| **Maternal Causes** |
| 1. Cervix not fully dilated.<br>2. Undiagnosed cephalopelvic disproportion<br>3. Generalized tonic contraction of uterus<br>4. Constriction ring<br>5. Non-distension of vagina and paravaginal tissue |
| **Foetal causes** |
| 1. Malposition (occipito-posterior position)<br>2. Macrosomia<br>3. Deflexed head<br>4. Shoulder dystocia<br>5. Undiagnosed malpresentation (e.g. brow)<br>6. Large foetal head (e.g. hydrocephalus) |
| **Management** |
| 1. Ringer lactate or 5% dextrose solution is started and arrangement for blood transfusion, if required.<br>2. To start parenteral antibiotics.<br>3. To assess maternal and foetal conditions.<br>4. Careful vaginal examination to find out presentation and position of the presenting part.<br>5. Uterine exploration to rule out uterine rupture<br>6. Forceps application is stopped and cesarean is performed. Laparotomy should be performed with suspected uterine rupture. Subtotal or total hysterectomy may be required.<br>7. Craniotomy may be done for a dead fetus. |

## Complications of forceps

The complications of forceps are shown in Table 42.9.

# VACUUM EXTRACTOR OR VENTOUSE

## Definition

Ventouse is an instrumental traction device that is attached to foetal scalp and is used to assist in the delivery of the head by suction forces. The term **vacuum extraction** is more popular in USA while the term **ventouse** (meaning soft cup in French) is used in Europe.

## Instrument

Two components: (a) Suction machine (b) Suction cups

### Suction Machine (Fig. 42.16)

Suction machine can be operated mechanically, manually or by electricity. The latter is preferred which create a vacuum of about 0.8 kg/cm$^2$. Start with 0.2 kg/cm$^2$ and

**Table 42.8: Causes of Difficulty in Forceps Application**

1. **Difficulty in application of blades**
   - i. Cervix not fully dilated
   - ii. Unrotated, malrotated or non-engaged foetal head
2. **Difficulty in locking of blades**
   - i. Application in an occipito-posterior head
   - ii. Wrong introduction of blades: Short application
   - iii. Handles not depressed far enough against the perineum
   - iv. Foetal cord entanglement in the fenestra of forceps blades.
3. **Difficulty in traction**
   - i. Wrong cephalic application of forceps
   - ii. Undetected persistent occipito-posterior position
   - iii. Undiagnosed brow presentation
   - iv. Inappropriate direction of traction forces.
   - v. Constriction ring
   - vi. Pelvic contraction
4. **Blades slipping**
   - i. Blades are not inserted far enough—short application
   - ii. Undiagnosed persistent occipito-posterior position
   - iii. Oblique or antero-posterior application over baby's occiput and face.

**Table 42.9: Complications of Forceps**

| Maternal complications | Foetal complications |
|---|---|
| **Early complications** | |
| 1. Vaginal and cervical lacerations<br>2. Extension of episiotomy and 3rd degree or complete perineal tear.<br>3. Postpartum haemorrhage due to trauma, atonia or both<br>4. Shock<br>5. Anaesthetic hazards<br>6. Nerve injuries—Femoral ($L_{2, 3, 4}$), lumbosacral trunk ($L_{4, 5}$) or sciatic plexus damage ($L_4 - S_3$) with difficult mid forceps delivery<br>7. Colporrhexis<br>8. Rupture uterus<br>9. Pelvic haematoma<br>10. Infection (puerperal sepsis)<br>11. Urinary retention<br>12. Urinary tract infections | 1. Laceration or bruising of foetal skull<br>2. Intracranial injuries, tentorial tears<br>3. Laceration and injury to scalp<br>4. Cephalhematoma<br>5. Fracture of skull<br>6. Cervical spine injury (rotational forceps)<br>7. Neurological injury in the form of facial palsy (Bell's palsy), Erb's palsy<br>8. Hypoxia<br>9. Rarely intraventricular haemorrhage and death |
| **Late (Remote) complications** | |
| 1. Painful scars in perineum<br>2. Painful coitus (dyspareunia)<br>3. Back strain<br>4. Uterovaginal prolapse<br>5. Fecal incontinence due to rectovaginal fistula or complete perineal tear<br>6. Stress urinary incontinence<br>7. Vesicovaginal fistula | 1. Cerebral palsy may rarely occur in difficult mid forceps delivery due to persistent cerebral injury. **Large international studies have confirmed that there is no association between forceps delivery and measures of intelligence in the children, epilepsy and long term neuro developmental outcome.** |

increase gradually with 0.2 kg/cm$^2$ every minute They have fine regulatory switch that can precisely control the vacuum with small increments.

## Suction Cups

### Metal cups (Fig. 42.17)

**Baird's modification,** traction chain is attached at the center, a separate suction hose is attached at the rim or at the side (in occipito-posterior cup), edges of the cup are inverted to facilitate the expansion of **'chignon'**, the artificial caput into the cup to give a firmer grip. Three sizes of cups are available:

(*i*) **6 cm:** Should be the first choice in the application as it is the largest cup.

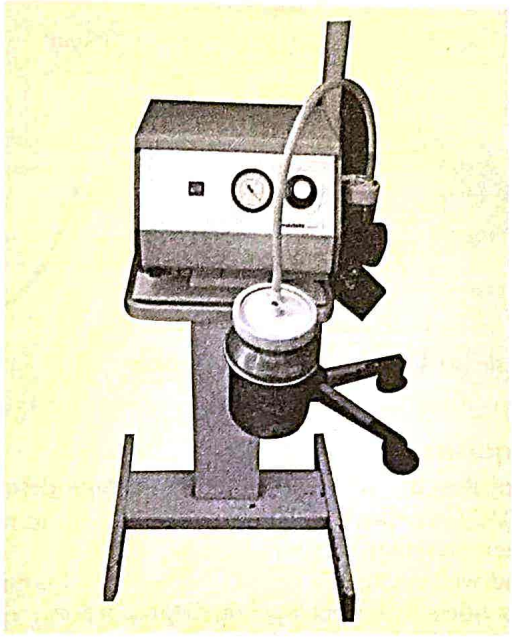


**Fig. 42.16:** Suction machine for ventouse

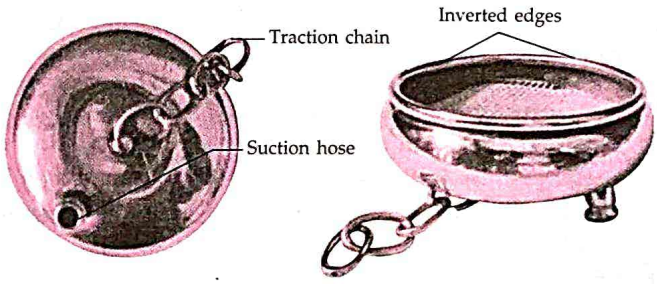



**Fig. 42.17:** Metal cups

(*ii*) **5 cm:** Best suited as OP cup in occipito-posterior position applications.

(*iii*) **3 cm:** Not used in extraction of foetal head.

Metal cups are suitable in cases where rotation is needed.

### Silastic Cups (Fig. 42.18)

Silastic cups are soft cups made of silastic with straight or everted edges with no chignon effect. Traction and suction parts are integrated into the center of the cup causing higher incidence of cup pop-off from lateral traction. Two sizes are available. They are 6 cm and 5 cm.

Various silastic cups are Mityvac M-style or Mityvac M-select or Mityvac mity soft bell (Cooper Surgical). It has a soft bell cup attached by a semi rigid shaft to a hand held pump. Kiwi omnicup (Clinical Innovations) contains a hand held vacuum generating pump which is attached to a rigid plastic mushroom cup through a flexible tubing.

Silastic cups are best used for outlet applications that do not require any rotation of the head. Low applications requiring some degree of rotational movement are better addressed by metallic cups (Fig. 42.18B). Soft cups have higher failure rate than metal cups but have lower incidence of neonatal scalp trauma (RCOG 2020).

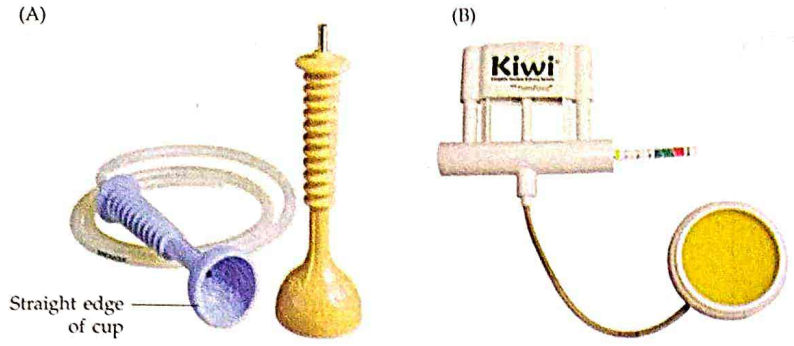



**Fig. 42.18:** (A) Silastic cups; (B) Kiwi Omnicup

## Pre-requisites

Pre-requisites are the same as in the forceps described above (AAP and ACOG Guidelines, 2007)

1. Vertex presentation
2. Head well-engaged
3. No evidence of cephalopelvic disproportion
4. Cervix fully dilated or almost so
5. Bladder should be empty.
6. Ultrasound assessment of foetal head position before assisted vaginal birth is recommended if there is uncertainly on clinical examination (RCOG 2020)

## Indications

1. Maternal distress in the second stage
2. Foetal distress in the second stage. (Forceps delivery is preferred for this indication. However, rarely newer ventouse cup with faster increase in pressure can be used).
3. Prolonged second stage
4. Deep transverse arrest or persistent occipito-posterior position
5. Medical diseases, like pre-eclampsia

## Contraindications

1. Operator inexperience
2. Inability to assess foetal position
3. High station
4. Suspicion of cephalopelvic disproportion (RCOG 2020)
5. Face, brow and other non-vertex presentations
6. Fetuses less than 32 weeks (prematurity) (use with caution between 32 to 36 weeks)
7. Foetal coagulopathy (relative) (RCOG 2020)
8. Known macrosomia
9. Recent scalp blood sampling (relative). Recent RCOG Guideline (2020) allows ventouse delivery after foetal blood sampling.

## Technique of Ventouse Extraction

1. Pre-requisites, preliminaries are the same as for the forceps application.
2. Ensure that the patient is having good uterine contractions along with effective bearing down efforts from the mother. Oxytocin drip can be started.
3. Local perineal infiltration is done with 1% xylocaine or pudendal block analgesia is given.
4. Largest possible cup size should be used.
5. The ventouse apparatus should be properly assembled and tested before use.

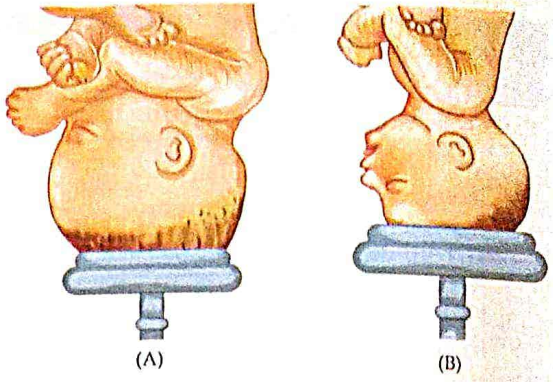



**Fig. 42.19:** (A) Correct application on the posterior fontanelle encouraging foetal head flexion during traction; (B) Incorrect application of ventouse on foetal head causing its deflexion and increased presenting diameter.

## Procedure

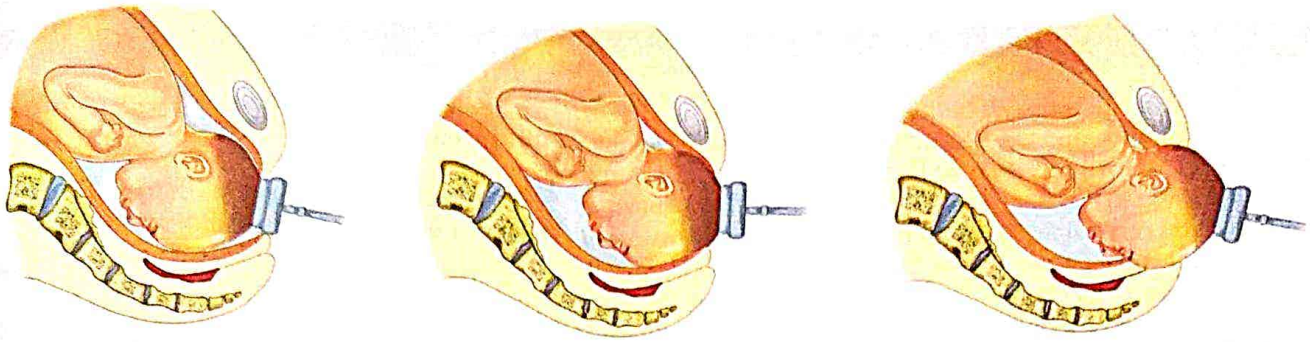
### Step I: Application of the Cup (Fig. 42.19)

1. Largest convenient cup (50–60 m) depending upon cervical dilatation should be used. The cup is lubricated and introduced in the vagina like a ring pessary after retraction of perineum with left hand fingers.
2. The center of the cup should be over the sagittal suture and 2 to 3 cm in front of posterior fontanelle **(flexion point)** with knob of the cup pointing in the direction of occiput (Fig. 42.19 A). This promotes flexion, descent and rotation of the head.
3. Wrong placement of cup (Fig. 42.19 B) is avoided as anterior placement nearer the anterior fontanelle can aggravate deflexion while asymmetrical placement relative to sagittal suture can worsen asynclitism.

Proper cup placement with vertex in outlet position and occipito-anterior position is quite easy. For other situations metal cups may be preferred as they are easier to place in the optimal position. Occipito-posterior cup is more suitable for occipito-posterior position. Liberal episiotomy is given to prevent perineal tear.

### Step II: Creation of Vacuum

1. After placement of cup, confirm that no maternal tissue is entrapped between the cup and foetal scalp.
2. Cup is connected to the suction machine and an initial vacuum of 0.2 kg/cm$^2$ is created (about 250 mm Hg). A repeat check is made to ensure that no maternal tissue is trapped inside the cup. For metallic cups and system, the pressure is increased at a rate of 0.1 kg/cm$^2$ (90–100 mmHg) every minute until a vacuum of 0.7 to 0.8 kg/cm$^2$ (about 580–600 mm Hg) is obtained in 8–10 minutes. The effect of the suction is to draw the foetal scalp into the cup causing its



A: Traction outward and posteriorly (downwards)

B: Traction outward and horizontally (forward)

C: Traction outward (forwards) and anteriorly (upwards)

**Fig. 42.21:** Diagramatic representation of application of ventouse (vacuum extractor) at various stations of foetal head showing direction of forces.

edema or an artificial caput succedaneum **(chignon)** (Fig. 42.20) within rim of ventouse cup, which usually disappears in few hours.

3. In modern ventouse machines using silastic cups, vacuum is raised in a single step to the target level of 0.8 kg/cm$^2$ within a minute and it can even be used for foetal distress cases. Rapid suction causes no harm.



**Fig. 42.20:** Diagramatic representation of chignon formation due to ventouse delivery

**Step III: Traction**

1. Traction is given at right angle to the cup.
2. Traction is exerted intermittently along with the uterine contractions and maternal expulsive efforts.
3. Traction is given by using a two-hand technique-fingers of the left (passive) hand are placed against the edge of the cup pressing it against the scalp to avoid dislodgement of cup while the right (active) hand gives the traction to the instrument.
4. **Direction of traction.** The direction of pull is like mid forceps **first downwards, then horizontal** and at the **end forward** and **upwards** (Figs. 42.15 and 42.21).
5. Manual torque should never be applied to the cup for rotating the head. The head undergoes autorotation with traction automatically.
6. Complete vacuum assisted birth in majority of cases with maximum of three pulls to bring head to perineum. Three additional pulls can be used to ease head out of perineum (RCOG 2020). If there is minimal or no descent with 2 pulls, experienced operator should evaluate the case and look for foetal position or cephal-pelvic disproportion and may discontinue the procedure.
7. The procedure should always be considered as trial, and if there is no early and clear evidence of descent towards delivery consideration for alternative approach preferably cesarean delivery is advisable.

The vacuum is released as soon as the head is crowning. Rest of the delivery is conducted in a normal way.

**Cup Pop-off**

1. Cup pop-off is detachment of cup due to technical failure or wrong or suboptimal placement of cup and merits additional attempts at placement and delivery. Discontinue ventouse delivery if there are two 'pop-offs' of the instrument. After one pop-off experienced operator should take over to give best chance.
2. On the other hand, with optimal placement of cup the cup pop-off indicates relative or absolute disproportion or asynclitism requiring excessive traction forces. In such circumstances, it is better to abandon the procedure in favor of an alternate method of delivery.

**Post operative care and treatment.** They are same as in forceps. The parents should be reassured that bump in newborn's head (chignon) will disappear soon.

## Complications

The complications of ventouse are shown in Table 42.10.

## Trial vacuum and failed vacuum

The trial vacuum and failed vacuum are the same as described under forceps.

## Comparison of vacuum extraction with forceps

Both forceps and ventouse are effective and safe modalities for assisted vaginal delivery. All obstetricians should be well trained in the use of both the instruments as both are complementary to each other. The choice of instrument depends upon the clinical situation. There are no long term neurological deficits with either instrument. Use of second instrument after failure of one should be avoided and used only by an experienced operator after careful assessment. The relative advantages and disadvantages of the two instruments are given in Table 42.11.

## Recommendations regarding vacuum delivery

1. The classification of vacuum deliveries should be the same as that for forceps deliveries (including station).

**Table 42.10: Complications of Ventouse**

| Maternal complications | Foetal complications |
|---|---|
| They are **less common** and can be due to inclusion of maternal soft tissues like cervix or vagina in the cup.<br>1. Vaginal and cervical lacerations<br>2. Vaginal haematoma<br>3. Third degree perineal tear<br>4. Annular detachment of cervix<br>5. Traumatic postpartum haemorrhage<br>6. Rarely vesicovaginal fistula | They are **more common** with ventouse than with forceps.<br>1. Scalp abrasion and ecchymoses<br>2. Scalp sloughing<br>3. Cephalhematoma (haematoma under the periosteum) in about 1% cases<br>4. Sub-aponeurotic (subgaleal) haematoma in about 1% cases. It is not limited by suture lines being subperiosteal<br>5. Intracranial haemorrhage (1%)<br>6. Retinal haemorrhage<br>7. Subconjunctival haemorrhage<br>8. Neonatal jaundice<br>9. Asphyxia in difficult ventouse delivery<br>10. Clavicular fracture<br>11. Injuries of sixth and seventh cranial nerves and Erb's palsy<br>12. Rarely foetal death |

**Table 42.11: Comparative Advantages and Disadvantages of Ventouse and Forceps Deliveries**

| Vacuum (Ventouse) | | Forceps | |
|---|---|---|---|
| **Advantages** | | **Disadvantages** | |
| 1. | No need or less need for anesthesia **(level 1A evidence)** | 1. | Requires anesthesia |
| 2. | Less expertise required | 2. | More operative expertise required |
| 3. | Does not occupy space on the sides | 3. | Occupies space on the sides |
| 4. | Does not require full cervical dilatation | 4. | Requires full dilatation |
| 5. | Promotes autorotation | 5. | No autorotation |
| 6. | It can be used in non-rotated and malrotated occipito-posterior position of head. | 6. | It cannot be used for non-rotated head (only Kielland forceps can be used) |
| 7. | Less traction force needed **(about 10 kg)** | 7. | More traction force needed **(up to 18 kg)** |
| 8. | It can be used when head is higher but engaged. | 8. | High forceps cannot be and should not be used. |
| 9. | Less maternal injuries **(12%)** | 9. | Maternal injuries like complete perineal tears are more common **(29%) (level 1A evidence).** |
| 10. | Less perineal pain after 24 hours. | 10. | More perineal pain after 24 hours **(level 1A evidence).** |
| **Disadvantages** | | **Advantages** | |
| 1. | Requires maternal effort | 1. | It can be used without maternal effort and hence suitable in heart disease and other maternal diseases. |
| 2. | Equipment more complex and expensive | 2. | Less complex equipment and cheap |
| 3. | Takes time to build pressure and is thus not suitable in foetal distress (in newer silastic cups, pressure can be built fast). | 3. | Very suitable in cases of foetal distress being very quick |
| 4. | Cannot be used in other presentations like face, after-coming head of breech. | 4. | Can be used in face anterior and occipito-posterior presentation and after-coming head of breech |
| 5. | Cannot be used in preterm delivery. | 5. | Can be used in preterm delivery |
| 6. | More foetal cephalhematomas, intracranial and retinal hemorrhages **(level 1A evidence)** | 6. | Less foetal cephalhematoma, intracranial and retinal haemorrhage |
| 7. | Failure rate is higher. | 7. | Failure rate is much less. |
| 8. | More maternal worries about the baby due to chignon and foetal injuries. | 8. | Less maternal worries about the baby. |
| 9. | It can be used with 8–9 cm dilated cervix | 9. | Fully dilated cervix only for rotated head (Later it can be used). |
| 10. | 6th nerve palsy, sholder dystocia, cephal haematoma, retinal injury or subgaleal haemorrhage are common | 10. | Facial nerve palsy, branchial, plexus injury, corneal injury are common. |

2. The same indications and contraindications used for forceps deliveries should be applied to vacuum-assisted deliveries also.
3. The vacuum should not be applied to an unengaged vertex, that is, above 0 station.
4. The individual performing or supervising the procedure should be an experienced operator.
5. The operator should be willing to abandon the procedure if it does not proceed easily or if the cup pops-off 2–3 times or for 15–30 minutes ACOG (2020) recommends abandoning the procedure if satisfactory progress is not made.
6. ACOG (2020) recommends against the sequential use of instruments (that is use of forceps after ventouse failure and vice versa).
7. Three pulls are allowed for outlet extraction

## Key Points

1. Obstetric forceps and ventouse are designed to extract foetal head when a quick delivery is going to help mother or the baby and vaginal delivery is expected to be safe.
2. Each forceps has a handle, shank, lock and blade. American Academy of Pediatrics and ACOG (2007) have classified forceps into outlet, low and mid forceps.
3. The forces in forceps can be traction, compression, rotation (Kielland forceps) and protection. They are indicated for maternal exhaustion, to cut short second stage of labour or for foetal distress.
4. Pre-requisites have to be fulfilled before applying them like fully dilated cervix (F), occiput and other suitable presentations (O), ruptured membranes (R), CPD's absence (C), effective uterine contraction with engaged head (E), proper position of head (P) and sagittal suture in antero-posterior diameter or within 45° (S). Non-fulfilment of pre-requisitions are contraindications for forceps.
5. Both forceps and ventouse are useful tools which should only be applied by trained obstetricians for judicious indications to complete delivery with minimum trauma to the mother and the child.
6. Systematic review has shown that as **compared with forceps, ventouse delivery is associated with higher failure rate, more incidence of cephalhematoma and retinal hemorrhages but is associated with significantly lesser use of regional or general anesthesia, lesser maternal perineal and vaginal trauma (tears) and lesser post-operative pain (level IA evidence).**
7. Failure rate is higher with soft (plastic) cups than with metallic cups and soft cups should not be used for rotational deliveries. Use of a second instrument (forceps after failure of ventouse) increases the risks of foetal and maternal damage and should be avoided.

## Suggested Reading

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# (iii) EPISIOTOMY (SYN. PERINEOTOMY)



## INTRODUCTION

### DEFINITION

Episiotomy is a deliberate cut given on the posterior vaginal wall and the perineum (pudenda) for delivery of fetus during the second stage of labour. It is the most common operation in obstetrics. Its incidence has reduced in USA from 65% in 1979 to only 12% in 2012 (75% decline). However incidence is still high in India. It was 60% over all in AIIMS in 2018 (annual statistics).

### Objectives

1. Episiotomy equivalent to a controlled tear and easier to repair in layers as compared to other tears.
2. To increase the available space at the vaginal outlet to enable easy and safe parturition and foetal delivery and to minimize trauma to the foetal head.
3. To reduce excessive stretching and tearing of the levator ani and other perineal muscles, ligaments and pelvic fascia and avoid perineal tears and or lateral vaginal wall tears.

### Advantages

#### 1. Maternal

Episiotomy was used in all women having their first delivery in the past for the following suggested benefits.

(a) A surgically planned cut can be sutured in a much better way as compared with irregular and ragged vaginal and perineal tears which can occur in absence of episiotomy.

(b) Shortening of second stage of labour which is the most traumatic stage for mother.

(c) Decrease in stretching and tearing of levator ani and other pelvic floor muscles, ligaments and pelvic fascia minimizing occurence of cystocele, rectocele, uterine descent, urinary and fecal incontinence later in life.

(d) Decreased pain and discomfort in episiotomy as compared with tears.

#### 2. Foetal

Significant reduction in incidence of intracranial bleeding and injuries especially in preterm babies and in instrumental deliveries.

However, all these benefits were unproven and found to be incorrect by various studies. A number of observational studies and randomized trials have shown that routine episiotomy is associated with an increased incidence of anal sphincter and rectal tears as compared with restricted episiotomy **(level I A evidence)**.

Cochrane review of randomized trials (2017) showed lower rates of severe perineal and vaginal trauma with restrictive use of episiotomy than with routine episiotomy. American College of Obstetricians and Gynecologists (2016) has also recommended restricted use of episiotomy over routine use.

### Indications

The indications of episiotomy are shown in Table 42.12.

**Table 42.12: Indications of Episiotomy**

1. **Markedly short perineal length** which may prevent descent of foetal head
2. **Increased risk of perineal and vaginal tears.** (a) Macro-somia (big baby), (b) Face-to-pubis delivery: (Persistent occipito posterior positions), (c) Assisted breech delivery, (d) Shoulder dystocia, (e) mento-anterior delivery
3. **Operative vaginal delivery.** Forceps (especially mid-forceps) and vacuum delivery
4. **Past perineal surgery.** Past complete perineal tear repair and perineal reconstructive surgeries

Episiotomy should not be performed routinely in all primigravidas but only in judicious and indicated cases. Proper monitoring and care during the second stage of labour can reduce the need of episiotomy and perineal tears. Hence, routine use of episiotomy in all primigravidas as is still the practice in many hospitals in India should be replaced by selected and restricted use as is clear by evidence-based medicine that routine episiotomy does not prevent perineal floor damage, cystocele, rectocele, uterine prolapse or stress urinary incontinence.

### Timing of the Episiotomy

Episiotomy should neither be performed too early to avoid unnecessary blood loss nor too late to avoid stretching and trauma to pelvic floor and perineum. It is usually performed at crowning of head when 3 to 4 cm of foetal head is visible. During instrumental delivery, most practitioners perform episiotomy after application of blades or ventouse cup but it can be given just before their application if perineum is very tight.

## TYPES OF EPISIOTOMY

There are four types of episiotomy as given below and in the Fig 42.22.

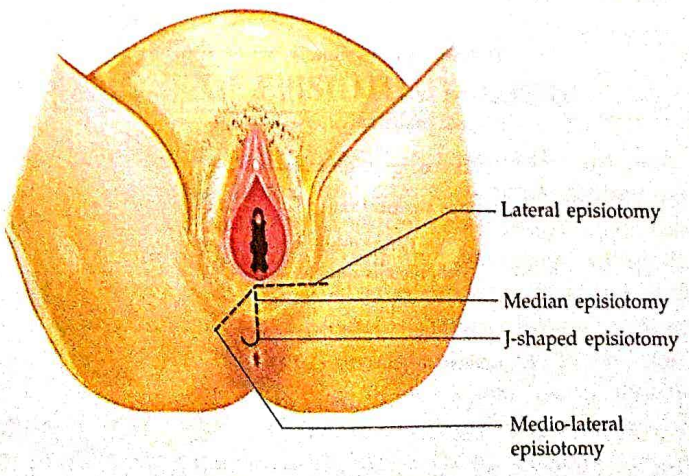



**Fig. 42.22:** Diagramatic representation of various types of episiotomy

## 1. Medio-lateral

The cut is performed downwards and outwards from the center of the lower vulval rim (fourchette) usually to the right side (rarely to left). It is directed at an angle of 60° to the center and at the central point between anal verge and the ischial tuberosity so that the final direction postpartum lies at 45°. A special scissor is available to give adequate angle as there is risk of angle being less.. It is the most commonly performed episiotomy in clinical practice.

## 2. Median

About 2–2.5 cm cut is made from the center of the lower vulval rim (fourchette) directly posteriorly in the midline in the direction of anus. It may injure anal sphincter.

In contemporary practice, only medio-lateral (most common) or median episiotomies (rarely) are performed. Their relative advantages and disadvantages are shown in Table 42.13.

## 3. Lateral

The cut is performed from the center of the lower vulval rim (fourchette) at 90° to midline. It causes excessive bleeding and may damage the Bartholin's duct. It is not used in modern obstetrics.

## 4. J-shaped

The cut is performed from the center of lower vulval rim (fourchette) first straight in midline for 1.5 cm and then is directed laterally towards 7 o'clock position to prevent injury to rectum and anal sphincter. As suturing is not

**Table 42.13: Comparison of Advantages and Disadvantages of Medio-lateral and Median Episiotomy**

| Medio-lateral episiotomy | Median episiotomy |
|---|---|
| **Advantages** | **Advantages** |
| 1. It does not usually cause third or fourth degree perineal tear even on extension.<br>2. The cut can be easily enlarged, if needed | 1. Better anatomic result as suturing is easy and perfect due to even margins.<br>2. Less bleeding<br>3. Very less pain, swelling and tenderness at episiotomy site in puerperium<br>4. Episiotomy heals better.<br>5. Less wound complications and dehiscence<br>6. Less painful intercourse later |
| **Disadvantages** | **Disadvantages** |
| 1. Suturing and apposition of wound margins are less perfect due to uneven tissues.<br>2. Slightly more bleeding<br>3. Pain and swelling at episiotomy site in puerperium.<br>4. Wound complications and gaping are more common.<br>5. More chances of painful intercourse. | 1. The main disadvantage is that if enlargement occurs it can cause third and fourth degree tears, especially in high risk cases which has limited its routine use<br>2. The cut cannot be enlarged, even if needed due to anus and rectum coming in the way |

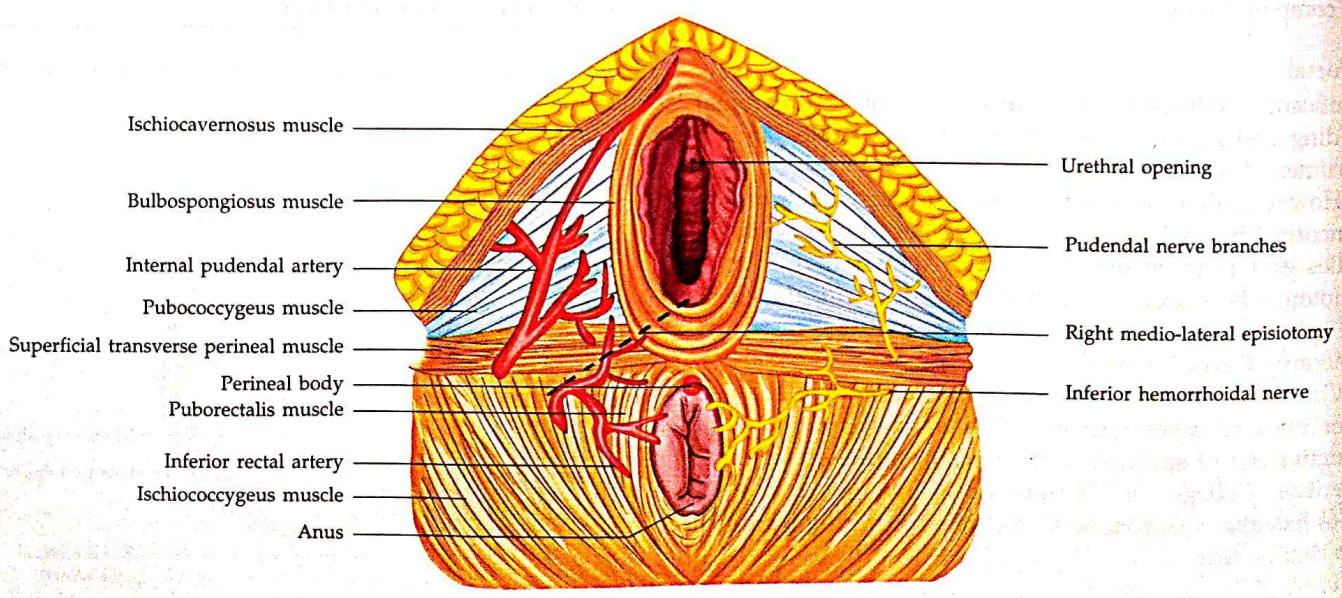



**Fig. 42.23:** Structures cut in medio-lateral episiotomy (dotted line)

| Table 42.14: Structures which are Cut in Medio-lateral Episiotomy | |
|---|---|
| 1. | Fourchette |
| 2. | Posterior vagina |
| 3. | Superficial transverse perineal muscles |
| 4. | Bulbospongiosus muscle |
| 5. | Deep transverse perineal muscles |
| 6. | Some part of the levator ani muscles |
| 7. | Pelvic fascia over the perineal muscles |
| 8. | Branches of pudendal nerve and vessels |
| 9. | Subcutaneous tissue |
| 10. | Skin (epidermis and dermis)<br>Muscles not cut: Ischiocavernosus, Ischiococcygeus |

perfect and there are more wound complications, it is rarely performed in modern obstetrics.

## STRUCTURES CUT IN MEDIO-LATERAL EPISIOTOMY

The various structures cut in the episiotomy are shown in Table 42.14 and Fig 42.23.

## STEPS OF MEDIO-LATERAL EPISIOTOMY

### Step 1: Preliminaries

Written consent is taken. Ensure that patients are immunized against tetanus. The perineal area is cleaned with antiseptic solution and draped with sterile sheets.

The perineum, at the site of episiotomy is injected with 10 mL of 1% lignocaine (xylocaine) solution (Fig. 42.24) in a fan shaped manner. Lignocaine prilocaine (2.5%) cream (EMCA) applied 1 hr before procedure. Bilateral effective pudendal nerve block is very effective.

Paracervical block does not give anesthesia for episiotomy.

### Step 2: Incision (Fig 42.25)

The index and middle fingers of left hand are put into the vagina with palmar surface towards the perineum to expose the vaginal skin, fourchette and perineal body to protect the baby's head.

The perineum is cut with the episiotomy scissors at the peak of uterine contraction (structures cut are described above) under vision. The cut extends from the center of the fourchette (posterior vulval rim) and extends laterally usually to the right (rarely left) at an angle of 45°. The incision should be large enough for easy delivery of the presenting part (Fig. 42.25).

## TIMING OF THE EPISIOTOMY REPAIR

The most common practice is to defer episiotomy repair until the delivery of placenta permiting undivided attention to the signs of placental separation and delivery. A further advantage is that episiotomy repair is not interrupted or disrupted by the obvious necessity of delivering the placenta, especially if manual removal is required. The disadvantage is continuing blood loss from unstitched episiotomy site.

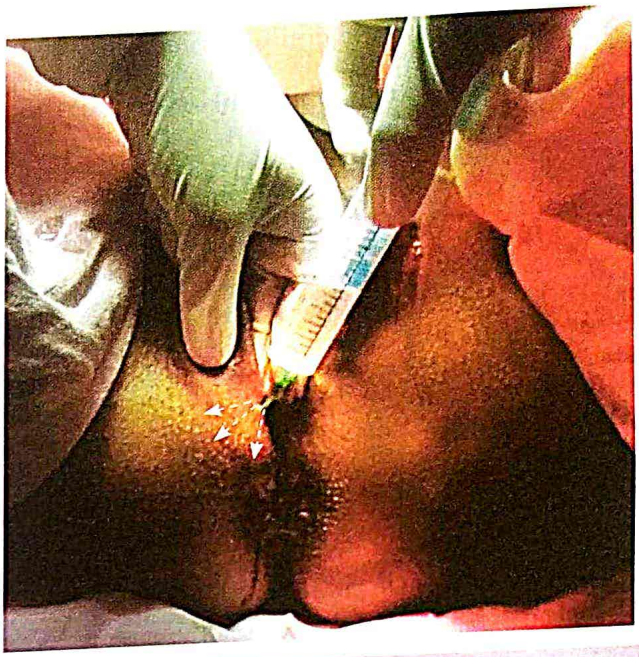


**Fig. 42.24:** Technique of infiltration of local anaesthetic in the perineum for episiotomy

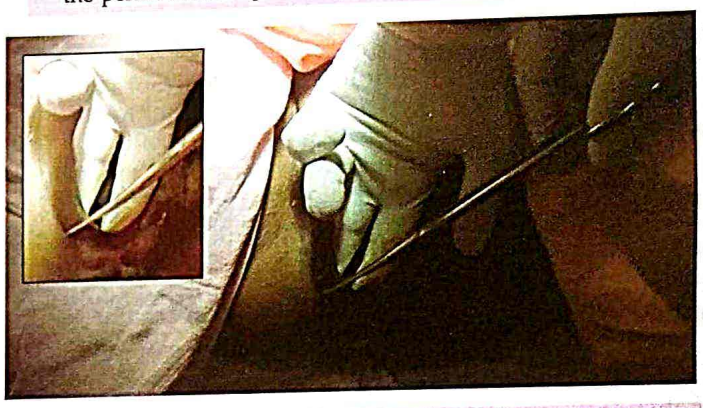


**Fig. 42.25:** Right medio-lateral episiotomy (see inset)

## TECHNIQUE OF EPISIOTOMY REPAIR

Polyglactin (vicryl) sutures result in less short term pain, less use of analgesia and less risk of wound dehiscence than chromic catgut **(level IA evidence)**. It is also available as vicryl rapide which has got the advantage of early absorption but with tensile strength similar to vicryl and is specially suited for episiotomy repair. Poliglecaprone (monocryl) is a newer monofilament, synthetic and absorbable suture and is suitable where more tensile strength is required like in big or irregular episiotomy or in perineal, vaginal or cervical lacerations. Chromic catgut is still used in many hospitals in India as it is cheap and more doctors are familiar with its use.

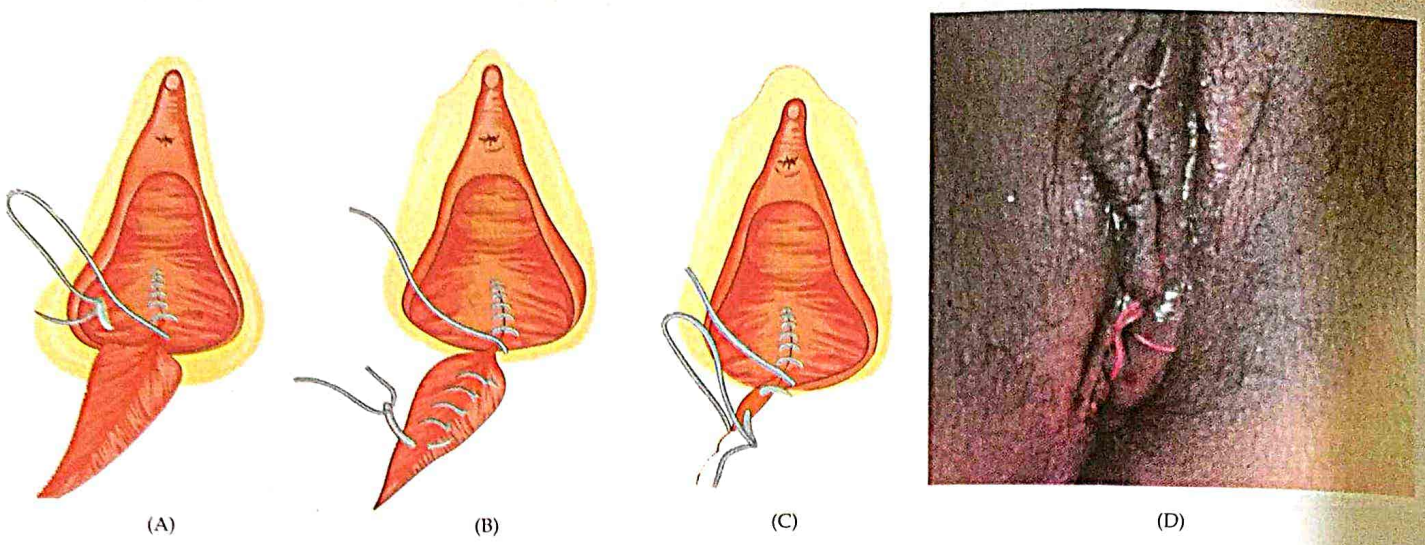



**Fig. 42.26:** Steps of repair of right medio-lateral episiotomy—(A) Repair of vaginal mucosa; (B) Repair of perineal muscles by interrupted sutures; (C) Repair of the skin margins; (D) Actual photograph of perineum after repair of episiotomy

### Repair

Women are put in lithotomy or dorsal position with legs supported by stirrups. Good light source should be available. The episiotomy site is to be cleaned with povidine iodine or savlon solution. Sterile drapes are used. Lignocaine 1% is infiltrated into the episiotomy liberally (10–20 mL).

### 1. Repair of Mucosa (Fig. 42.26 A)

A continuous non-locking hemostatic stitch is used to close the posterior vaginal wall starting from the apex up to the introitus with sutures being placed approximately 0.5 cm from the edge of the incision.

### 2. Repair of Muscles and Fascia (Fig. 42.26 B)

Achieving complete hemostasis and avoiding injury to rectum, muscles and fascia are stitched with vicryl or catgut suture (1–0) using continuous (preferred) or interrupted muscle stitches. All dead space has to be obliterated carefully, otherwise increase chance of haematoma formation in this layer.

### 3. Repair of skin (Figs. 42.26 C and D)

Interrupted mattress sutures with vicryl or catgut 1–0. Subcuticular suture with (vicryl 1–0) can be given if hemostasis can be achieved and the obstetrician is experienced in the technique for lesser post operative pain and better cosmetic results. Continuous suture in all three layers is associated with reduction in pain. Rectal examination should be carried out at the end to ensure that suture is not placed in the anal canal.

## POST OPERATIVE CARE OF EPISIOTOMY

1. **Dressing.** The wound should be cleaned with antiseptic solution usually dettol after each urination or defecation. ACOG and SOGC (2016) do not recommend antibiotics in routine episiotomy unless there is extension leading to third or fourth degree tear and is not really required. Most hospitals in India, tend to give antibiotic. Single dose of second generation cephalosporin is recommended by ACOG (2016) for repair of anal sphincter lacerations. If used, tablet Cefixime 200 mg twice daily or ampicillin or cephlexin 500 mg 6 hourly for 5 days along with anti-inflammatory lbuprofen and paracetamol 1 tablet 8 hourly for 5 days are given. Lactulose 15 mL at bed time can be given to prevent constipation.
2. **Ambulation.** Early ambulation is recommended.
3. Removal of stitches is not required as they are absorbable. If non-absorbable sutures are used, they should be removed on 6th day.

## COMPLICATIONS OF EPISIOTOMY

Immediate complications are as follows:

1. **Traumatic postpartum haemorrhage.** Due to blood loss from episiotomy site. Tremaxamic acid should be given for prophylaxis and treatment.
2. **Extension to a complete perineal tear.** It is more common in median episiotomy, but can occur even in medio-lateral episiotomy especially in instrumental deliveries, particularly mid forceps and rotational forceps and in occipito-posterior presentation with face-to-pubis delivery.
3. **Vulval haematoma.** Vulval haematoma can arise if hemostasis is not properly achieved. It causes severe excruciating pain locally and there is a red tender swelling. Treatment is drainage of the haematoma after cutting one or two stitches, antibiotics and anti inflammatory agents, (see Chapter 26 for details).
4. **Infected episiotomy.** Women complain of fever, pain with episiotomy site looking inflamed and edematous with unhealthy discharge (pus).
   Treatment: (a). Removal of one or two sutures for better drainage of discharge freely, local application of

povidine iodine solution or ointment. (b). Application of magnesium sulphate (sumag) on the area to reduce edema and pain. (c). Oral or parenteral antibiotics like ampicillin, gentamicin and metronidazole for 5 to 7 days.

5. **Wound dehiscence (gaped episiotomy).** This occurs due to sepsis, haematoma formation or inappropriate suturing of episiotomy. The wound should be covered daily with antiseptics until the infection clears and healthy granulation tissue is formed and let it heal by secondary intention. Resuturing of gaped episiotomy can be performed under local lignocaine analgesia after making the margins raw. Parenteral antibiotics like ampicillin with or without gentamicin and metronidazole are given to control infection.
6. **Postpartum pain** which can occur due to inflammation, infection, haematoma or faulty suturing.
7. **Rectovaginal** fistula causing fecal material coming out through vagina.
8. **Necrotising fascitis** with extensive necrosis in the perineal area is a rare complication. Treatment is debridement of tissues and broad spectrum parenteral antibiotics.
9. **Fecal incontinence** due to damage to anal sphincter.

## Delayed (Late) Complications

1. **Sexual dysfunction or dyspareunia.** Painful intercourse can happen due to bad suturing of episiotomy and puckered episiotomy scar or painful inflammation.
2. **Scar endometriosis.** It can rarely occur if endometrial tissue gets deposited at episiotomy site causing painful scar.
3. There is increased risk of third degree perineal tear in future deliveries.

## Key Points

1. Episiotomy is a surgically planned incision on the vagina and perineum to increase available space for the delivery of the foetal head and to avoid perineal tears and excessive stretching of perineal tissues to prevent genital prolapse and urinary incontinence. However, studies do not confirm these benefits. Episiotomy should only be used for judicious indications like for instrumental deliveries, breech deliveries, rigid perineum and where tear is imminent. Evidence does not support routine use of episiotomy for all primigravidas.
2. Medio-lateral episiotomy with incision starting from midpoint of fourchette diagonally towards right side about 2.5 cm from anus is the most preferred episiotomy to avoid third degree perineal tear in case episiotomy extends. The structures cut are fourchette, vaginal wall, superficial and deep transverse perineal muscles, bulbospongiosus and bulbocavernosus muscles, part of levator ani, fascia over muscles, branches of pudendal nerves and vessels, subcutaneous tissue and skin. It is given during crowning of head under 1% xylocaine local analgesia. Repair is done preferably with delayed absorbable polyglactin (vicryl) suture with subcuticular suture to skin if possible for better results. A 3–5 days course of antibiotics and anti-inflammatory agents can be given. Post operatively local dressing with dettol solution after urination and defecation is recommended.
3. Immediate complications include extension of episiotomy, vulval and perineal infection, gaping, complete perineal tear, rectovaginal fistula and necrotising fascitis. Delayed complications are dyspareunia and scar endometriosis.

## Suggested Reading

1. Jiang H, Qian X, Carolli G et al. Selective versus routine use of episiotomy for vaginal birth. Cochrane Database of Systemic Reviews 2 : CD000081, 2017.
2. American Academy of Pediatrics and the American College of Obstetricians and Gynecologists. Guidelines for Perinatal Care, 8th ed., Elk Grove Village, AAP, USA, 2017.
3. American College of Obstetricians and Gynecologists. Prevention and management of obstetric lacerations at vaginal delivery. Practice Bulletin no. 165, July 2016.
4. Cunningham FG et al Williams Obstetrics, McGraw Hill Education, New York, USA, 25th edn. 2018, p 529–538.
5. American College of Obstetricians and Gynecologists. Prophylactic antibiotics in labour and delivery. Practice Bulletin no. 120, June 2011, Reaffirmed 2016.

## Examination Questions

A. Define episiotomy. Enumerate its types, indications and advantages. Describe the technique of episiotomy repair, post operative care and its complications.

B. Write short notes on:
1. Medio-lateral episiotomy (Many universities)
2. Indications of episiotomy
3. Advantages of episiotomy
4. Types of episiotomy
5. Repair of episiotomy
6. Post operative care of episiotomy
7. Complications of episiotomy

# (iv) DESTRUCTIVE OPERATIONS (REDUCTIVE EMBRYOTOMY)

## Definition

Destructive operations are a group of operations that aim at diminishing the bulk of fetus by reducing the size of the head, shoulder girdle or trunk of the dead fetus in order to allow for its vaginal delivery. There is hardly any role of these operations in modern obstetrics. However, an obstetrician practising in rural areas in a developing country may face these situations and should therefore be familiar with the procedures. In a study published from North India, PGI, Chandigarh over 25 year (1983–2003), out of 85,952 deliveries, 230 (0.26%) destructive operations were performed with craniotomy being most common (87.8%) followed by decapitations (5.7%), eviscerations (3.6%) and cleidotomies (2.9%).

## TYPES OF DESTRUCTIVE OPERATIONS

1. Decompression (perforation) of a hydrocephalic head.
2. Craniotomy
3. Decapitation
4. Cleidotomy
5. Evisceration
6. Spondylotomy

The first three are more commonly used.

## 1. Decompression (Perforation) of a Hydrocephalic Head or Craniocentesis

A large hydrocephalic head can be reduced in size by using a large bore 16 or 18 gauge needle introduced per abdomen after emptying the bladder preferably under ultrasonic guidance. It can be done vaginally at about 3 cm cervical dilatation through the fontanelles with a needle. If the fetus presents by breech, the cerebrospinal fluid can be drained via the spinal canal. If spina bifida is present, then a needle or catheter introduced here will help in draining the CSF. Once the head collapses it can be delivered vaginally, after the cervix is fully dilated.

## 2. Craniotomy

**Definition.** In this operation, perforation of foetal skull is done to evacuate the brain matter and then delivering the head.

### Indications

1. Dead fetus in obstructed labour with cephalic presentation (vertex, face and brow). It is the most common indication.
2. Hydrocephalus even in a living fetus. For both cephalic and breech presentations.
3. After-coming head in breech presentation in cases where fetus is dead or its survival is dismal.

### Pre-requisites

1. Fully dilated cervix
2. Dead or hydrocephalic fetus

### Contraindications

1. Severe degree of contracted pelvis (true conjugate < 7.5 cm) as bimastoid diameter (7.5 cm) cannot be compressed even by craniotomy.
2. Suspected uterine rupture, as laparotomy is needed in such cases after resuscitation.

### Technical Aspects (Procedure)

1. **Intravenous fluids.** Ringer lactate, dextrose saline.
2. **IV antibiotics.** Inj. ampicillin 500 mg IV, 6 hourly
   Inj. Gentamycin 60 mg IV or IM, 8 hourly
   Inj. Metronidazole 500 mg IV, 8 hourly
3. **Sedation or anesthesia.** Inj. Pethidine 100 mg and Inj. Promethazine 25 mg IM or general anesthesia.
4. Step I
   (*i*) Pre-requisites are fulfilled. Bladder is catheterized.
   (*ii*) Patients are put in lithotomy position. Parts are cleaned and draped.
5. Vaginal examination is performed for presentation, position, station and pelvic assessment.
6. The two fingers of left hand are inserted into the vagina and are placed on perforation site.
   Perforation site.
   (*i*) Vertex: Either parietal bone
   (*ii*) Brow: Frontal bone
   (*iii*) Face: Orbit or roof of the (palate)
   (*iv*) After-coming head of breech: Occipital bone
7. The Oldham's (Fig. 42.27), Simpson's or Debdas's cranial perforator (like a mechanical drill) are used. The blades are inserted under the protection of fore and middle phalanges of left hand fingers to protect maternal soft tissues. Simpson's is the ideal perforator.

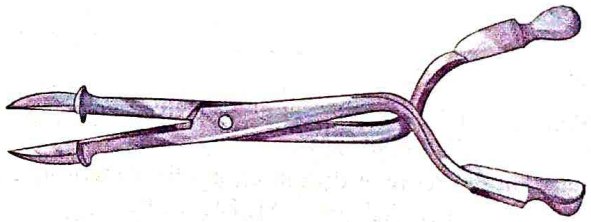


**Fig. 42.27:** Cranial perforator (Oldham's)

8. The foetal skull is perforated at proposed site by rotating movement (Fig. 42.28 A) with an assistant keeping the head steady with his hand per abdomen. The blades of the perforator are pushed through the skull up to their shoulder and are then separated by pressing the handles together to make a large tear in the skull in one direction (Fig. 42.28 B). The same procedure is repeated at right angle to the previous site after turning round the instrument.
   The perforated area resembles a cross or plus sign (+). The points (blades) of the perforator are pushed into the skull and the brain matter is broken up in all

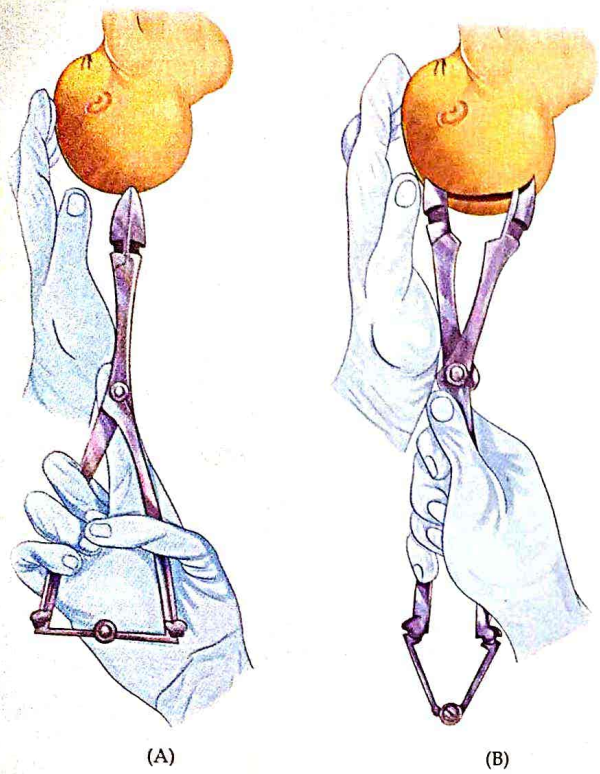



**Fig. 42.28:** (A) Perforation of the head using Simpson's perforator while an assistant fixes the head suprapubically; (B) Separation of the blades by compression of the handle

directions. The instrument is then withdrawn under protection of left hand to avoid trauma to maternal soft tissues.

9. Putting the right index finger in the cranium, brain matter is evacuated. Budin's cannula can also be used. The procedure causes collapse of the skull.
10. Extraction of the foetal head can be done by obstetric forceps (for normal pelvis or mild contraction), cranioclast or large vulsellums. The head can also be delivered with crotchet or hooked with a finger.
11. The traction exerted is downwards, then forwards and then upwards like in forceps delivery.
12. Inj. Oxytocin 10 units is to be given intramuscularly for active management of the third stage.
13. Placenta is delivered by controlled cord traction method.
14. The maternal soft tissues (perineum, vulva, vagina, cervix and uterus) are carefully visualized for any tears or lacerations.
15. Watch for postpartum haemorrhage. Inj. Methyl ergometrine is given 0.2 mg IV or oxytocin drip (10–20 units in 5% Ringer lactate or normal saline solution is started).
16. Self-retaining catheter (Foley's) is left for 5 days.
17. Antibiotics can be given for 5–7 days.

## 3. Decapitation (Fig. 42.29)

### Definition

Decapitation consists of severing the foetal head from the foetal trunk followed by the extraction of the trunk and then the head vaginally.

### Indications

1. In cases of neglected shoulder presentation with the arm prolapsed outside.
2. Interlocked head of twins with death of one fetus.

### Technique

The operation is best done under general anesthesia with decapitating hook (with knife or saw), a long pair of sharp edged blunt pointed scissors or Gigli's wire saw. Blond-Heilder saw is safest and less traumatic and can be used (see chapter 47 and Fig. 47.51 for detail).

### Procedure

1. Pre-requisites, preparation and hydration are similar to craniotomy discussed earlier. Blood transfusion may be given, if indicated.
2. In cases of neglected shoulder presentation, a sling is tied to the prolapsed arm and an assistant makes firm traction on it, so that the shoulder is fixed in the pelvic cavity and the neck comes nearer the pelvic outlet and is accessible vaginally.
3. The operator passes two fingers of his left hand along the shoulder on the groove of the neck anteriorly. Using decapitating hook with knife or saw under guidance of left hand, the neck is severed.
4. Traction is exerted upon the prolapsed arm to deliver the trunk.
5. The head may be delivered by fundal pressure, forking it out with two fingers into the mouth of the decapitated head or with a crotchet or with forceps. Use of oxytocin, intravenous fluids and antibiotics is like in craniotomy.
6. Post operative management is the same as in craniotomy.

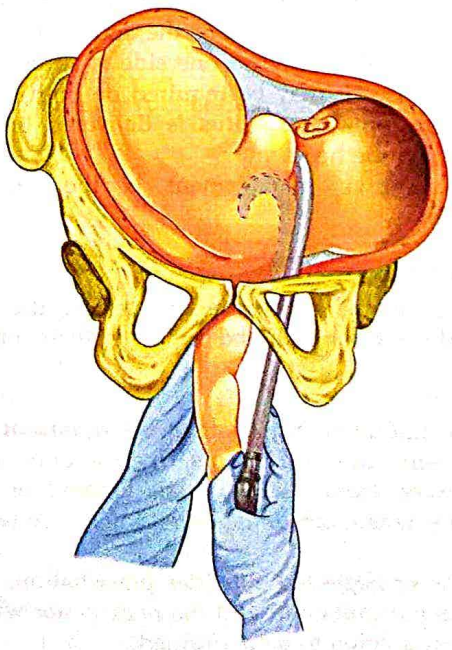


**Fig. 42.29A:** Decapitation being performed in transverse lie with hand prolapse using decapitation saw

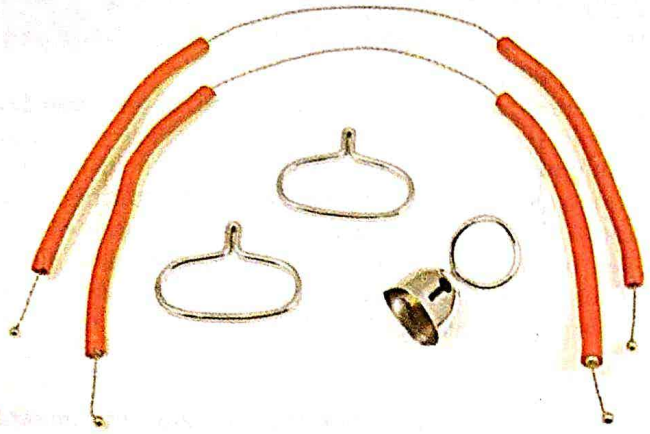


**Fig. 47.29B:** Blond-Heidler decapitation saw

## 4. Cleidotomy (Fig. 42.30)

In cleidotomy operation, division of one or both the clavicles is performed to reduce the bisacromial diameter of the dead fetus.

### Indications

1. In cases of contracted pelvis
2. Macrosomic dead fetus
3. Anencephalic monster
4. Shoulder dystocia with dead fetus

### Procedure

1. Pre-requisites and preparations are the same as in craniotomy.
2. Cleidotomy may be performed by cutting through the clavicle with a pair of scissors passed under the protection of two fingers introduced into the vagina along the anterior aspect of the shoulder (Fig. 42.30). Generally, cleidotomy on one side is sufficient, but in extreme cases it may be required on both sides. After cleidotomy, the dead fetus is delivered by applying traction on the head.

   Post operative management is the same as in craniotomy.

## 5. Evisceration

Evisceration is the procedure of opening the thorax or abdominal cavity or both and removing their contents.

### Indications

1. Where undue enlargement of the thorax or abdomen is present due to tumors, hydrothorax, congenital anomalies, foetal ascites, cystic kidneys or distended bladder where sometimes even for live fetus it can be done.
2. In case of neglected shoulder presentation, where the arm is not prolapsed and the neck is not within reach for decapitation to be performed.

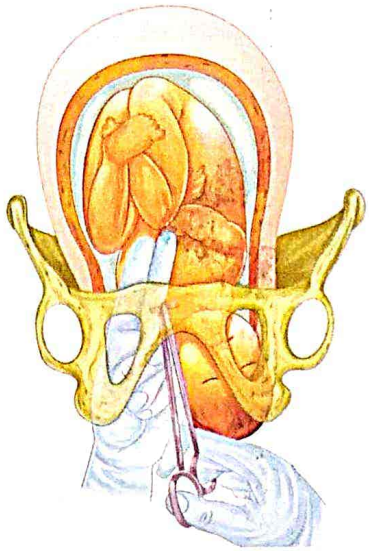


**Fig. 42.30:** Cleidotomy being performed with embryotomy scissors

### Technique

The operation can be done with the perforator or with a straight or curved pair of scissors especially embryotomy scissors, the maternal parts should be protected from injury and the instruments always guided along the fingers of the left hand. When perforating through the thorax, it is necessary to make a fairly big opening which can best be done by cutting away several segments of the ribs so as to make evisceration possible.

## 6. Spondylotomy

In spondylotomy operation, the spinal column of the fetus is divided with embryotomy scissors and the trunk is cut into two halves.

Spondylotomy can be done in cases of neglected shoulder presentation without arm prolapse with neck being too high for decapitation.

# COMPLICATIONS OF DESTRUCTIVE OPERATIONS

The complications of destructive operations are given in Table 42.15.

# CONCLUSION

Destructive operations with the exception of decompression of hydrocephalic head are almost never performed in developed countries and are rare even in developing countries as they represent lack of adequate antenatal and intrapartum services. But they are still performed in centers with inadequate services and are often asked in examination and hence are discussed here.

**Table 42.15: Complications of Destructive Operations**

1. Vaginal, perineal and cervical lacerations
2. Uterine rupture (generally lower uterine segment)
3. Postpartum haemorrhage
   a. Atonic haemorrhage
   b. Traumatic haemorrhage
   c. Both
4. Injuries of nearby organs
   a. Urinary bladder: Vesicovaginal fistula formation
   b. Urethra: Urethrovaginal fistula formation
   c. Rectal wall: Rectovaginal fistula formation
5. Shock:
   a. Hemorrhagic
   b. Hypovolemic
   c. Neurogenic
6. Puerperal sepsis (infection)
7. Non-involution and subinvolution of the uterus
8. Psychological depression
9. Chronic pelvic and abdominal pain

## Key Points

1. The main indications of destructive operations is to avoid a cesarean section. They are performed on the dead fetus to diminish its bulk to facilitate delivery.
2. The various destructive operations are decompression of hydrocephalic head, craniotomy (perforating the head), decapitation (severing the neck of fetus in shoulder presentation), cleidotomy (cutting of one or both clavicles), evisceration (debulking) and spondylotomy (cutting of vertebral column). After destructive operations, maternal soft tissues should be examined for injuries.
3. With the availability of safe cesarean section, there is hardly any place of the destructive operations in modern obstetrics. In fact, decompression of the hydrocephalic head is the only destructive operation performed in modern obstetrics. Otherwise, cesarean section is preferred even for dead fetuses.

## Suggested Reading

1. Umar AG, Maiahu AI, Panti AA et al. Destructive operative vaginal delivery in a tertiary health institution in North Western Nigeria: A ten year review: World Journal of Research and Review 2018; 6 : 68–71.
2. Sikka P, Chopra S, Kalpdev A et al, Destructive operations: a vanishing art in modern obstetrics: 25 year experience at a tertiary care centre in India. Arch Gynecol Obstet 2011; 283 : 929–33.

## Examination Questions

A. What is the role of destructive operations in modern obstetrics? Enumerate various types of destructive operations and their complications.

B. Write short notes on:
   1. Decompression of hydrocephalic head
   2. Craniotomy
   3. Decapitation
   4. Evisceration
   5. Cleidotomy

# (v) CESAREAN DELIVERY AND CESAREAN HYSTERECTOMY

## Definition

**Cesarean delivery is defined as the birth of a fetus through incisions in the abdominal wall (laparotomy) and the uterine wall (hysterotomy) after 28 weeks (24 weeks in western countries) of pregnancy and leaving the uterus in situ.**

The definition excludes extraction of the fetus from the abdominal cavity in uterine rupture and abdominal pregnancy.

Cesarean section being a tautology with both words meaning incision, cesarean birth or cesarean delivery are preferred terms and have been used in this book.

**Primary cesarean delivery.** When the cesarean is performed for the first time on a woman.

**Repeat cesarean delivery.** When the cesarean is done in second or subsequent pregnancies, it is called repeat cesarean delivery or secondary cesarean delivery.

## Historical background and nomenclature

According to legend, Julius Caesar was born by this operation and hence the origin of the term cesarean as delivery by this route was supposed to confer supernatural powers to persons. However, this is not possible as Julius Caesar's mother lived for many years after his birth and until as late as the 17th century cesarean delivery was invariably fatal.

Cesarean section was practised upon the dead women in early times. It was prescribed by an ancient Roman law started by the Numa Pompilius, the second king of Rome in about 715 BC known as Lex Regia, by which it was forbidden to bury a pregnant woman before her child had been cut out for separate burial. Later, in the time of Caesar's the law became **Lex Caesarea** which is the most probable derivation of the present name. The other more logical explanation is that the word cesarean is derived from the Latin verb **'Cedere'** which means **'to cut' and section** is derived from latin verb **'Seco'** which also means to cut. French obstetrician, Francois Mauriceau first reported cesarean delivery in 1668. In 1876, Italian obstetrician, Porro performed successful subtotal hysterectomy which reduced maternal mortality dramatically. Max Sanger, a German obstetrician, was the first to suture the uterine walls after cesarean in 1882. Prior to that there was a deeply rooted belief that the sutures in the uterus were superfluous and harmful by serving as a site of infection. In 1907, Frank mentioned the extraperitoneal cesarean. Kronig in 1912, started lower segment vertical incision which was popularized by DeLee (1922) in USA. The most important modification was the lower segment transverse incision, which was first performed by Kehrer in 1882, but the credit for reintroduction and popularization goes to Munro Kerr from United Kingdom in 1926. This is now universally accepted and commonly employed type of cesarean delivery.

## Incidence

The incidence of cesarean delivery has been progressively rising throughout the world. In the last 15–20 years, there has been two to threefold increase in the cesarean rate from about 10% to 15–20%. In USA, the incidence of cesarean delivery rose from 4.5% in 1970 to peak of 32.9% in 2009 with slight downward trend of 32% in 2015. Rates are very high in Brazil (55.6%), Egypt (51.8%), China (47%) moderate in Australia (33%), 26.3% in Canada, 26.2% in UK. Incidence is rising in India, also. The overall cesarean delivery rate rose from 8.5% in 2005–2006 to 17.2% in 2015–16 in India being lower (12.9) in rural areas (Desai 2017). Prevalence of cesarean delivery was 13.7% in public sector but was much higher (37.9%) in private sector in year 2011 (District Level Health Survey-4). Incidence is higher in tertiary referral centres. It was 53.15% in AIIMS in 2018 (Hospital statistics due to high risk cases). As per WHO at population level, the cesarean rates higher than 10 percent are not associated with reduction in maternal and neonatal mortality rates.

## Reasons of increasing cesarean rates

There has been a rise in cesarean delivery rate throughout the world due to causes given in Table 42.16.

**Table 42.16: Causes for High Cesarean Rates**

| | |
|---|---|
| 1. | Greater percentage of nulliparas. Due to fewer children women are opting for cesarean. Nulliparas are at higher risk of cesarean. |
| 2. | Rising incidence of elderly primigravidas. They have higher risk of cesarean delivery. |
| 3. | Use of electronic foetal monitoring which detects more cases of abnormal or non-reassuring foetal heart rate tracings. |
| 4. | Increased diagnosis of foetal distress by monitoring. |
| 5. | Decline in instrumental delivery, especially forceps |
| 6. | Cesarean for almost all breech presentations |
| 7. | More frequent use of repeat cesarean in women with prior cesarean delivery with decreased rate of vaginal birth after cesarean (VBAC). |
| 8. | Detection of high-risk mothers and pregnancies on time. |
| 9. | Early detection of foetal compromise |
| 10. | Greater expectation of a live baby from a healthy mother with neither obstetrician nor woman willing to take any risk. |
| 11. | Increased rate of obesity |
| 12. | Cesarean delivery on maternal request (CDMR) with increased cesarean delivery and after prediction of auspicious time of birth by astrologers, especially in India. |
| 13. | Increase in pregnancies following assisted reproduction |
| 14. | For sterilization at the same time |
| 15. | Increased rate of induction of labour |
| 16. | Elective cesarean to prevent pelvic floor injuries to prevent urinary and foetal incontinence and genital prolapse. |
| 17. | Increase in rate of cesarean delivery for pre-eclampsia with decrease in rate of induction of labour. |
| 18. | Malpractice litigation related to foetal injury in spontaneous or operative vaginal delivery increases cesarean rate. |

## INDICATIONS

The indications of cesarean delivery can be categorized in several ways. The indications may be divided into:

### 1. Maternal

When safe vaginal delivery is unlikely, labour is contraindicated or prompt delivery is needed to safeguard women's health.

### 2. Foetal

When prompt delivery is needed for foetal well-being or labour and vaginal delivery needs to be avoided for the sake of the fetus.

The various indications of cesarean are shown in Table 42.17.

## ABSOLUTE AND COMMON INDICATIONS AND CONTRAINDICATIONS OF CESAREAN DELIVERY

Absolute and common indications and contraindications of cesarean delivery are shown in Table 42.18.

## OPERATIVE PROCEDURE (LOWER SEGMENT CESAREAN DELIVERY OR LOWER SEGMENT CESAREAN SECTION [LSCS])

### Pre-operative orders and indication of cesarean delivery

1. Fully informed written consent must always be obtained after explaining risks and benefits to patients.
2. Nil orally after midnight or 6–8 hours (6 hrs - clear liquid; 8 hrs - solid) before the procedure in elective cesarean birth.
3. Urine examination
4. Full blood count and blood group
5. Blood should be available if antepartum haemorrhage, fibroid or anaemia is there. As a policy, however, blood must be crossmatched and available for all the cesarean deliveries.
6. Antacid prophylaxis is given using sodium citrate and ranitidine injection to prevent aspiration (see Chapter 19 for details).
7. Pubic hair encroaching on the proposed line of incision are clipped. Shaving in pubic area is routinely practised but should be discouraged as it causes abrasions which may promote infection.
8. If the woman is at high risk for thromboembolism, thromboprophylaxis with the use of mechanical calf compression (by elasticated stockings) and or Inj. Heparin 5000 units subcutaneously should be given 12 hourly for 2–3 days. Low molecular weight heparin (enoxaparin) can also be given (1 mg/kg body weight/day).
9. **Antibiotic prophylaxis.** It is used in all cases depending upon the protocol of the hospital to prevent puerperal sepsis. ACOG (2016) recommends single preoperative IV dose of 1 g of inj. cefazolin half an hour prior to cesarean incision. Injection coamoxyclav 1.2 g IV or any third generation cephalosporin (e.g. cefotaxime 1–2 g) as a single dose, is enough for elective cesarean section or three doses 8 hourly can be given for high risk cases or for suspected infection. High risk women (e.g. ruptured membranes) can be additionally given 500 mg I.V. azithromycin. Women with history of MRSA (methicillin resistant staphylococcus aureus) infection are given additional single dose of intravenous vancomycin. Common antibiotic regimen used in many Indian Government hospitals are:
   Inj. Ampicillin/amoxycillin 500 mg IV 6–8 hourly
   Inj. Gentamicin 60 mg IV or I/M BD
   Inj. Metronidazole 500 mg IV 8 hourly.
   The antibiotics are given for 5–7 days due to high risk of infection as many cases come as mishandled cases by traditional birth attendants and are anaemic and malnourished. However, there is no clear evidence that triple antibiotics are required for prophylaxis.
   During cesarean delivery, antibiotic prophylaxis for infective endocarditis is recommended for patients with prosthetic valves and cyanotic heart disease (not for acyanotic heart disease like ASD or VSD). Either ampicillin or amoxicillin orally or intravenously is recommended.
   For penicillin allergic patients, intravenous 900 mg clindamycin is given with aminoglycoside (gentamicin 60 mg IV). For obese patients, the dose of clindamycin is increased to 900 mg IV.
10. CTG for foetal heart or auscultate with stethoscope or sonicaid.
11. The bladder is emptied by a Foley's catheter which should be kept during the cesarean.
12. The patient is placed in a moderate Trendelenburg position with 15° left lateral tilt while carrying out the operation to prevent supine hypotension.
13. Call the anesthetist and pediatrician. The interval from making a decision to cesarean section should be minimum possible in emergency cases especially in foetal distress (should be less than 30 minutes).
14. **Anesthesia for Cesarean delivery**
    (a) Regional anesthesia in the form of epidural or spinal is preferred (see Chapter 19 for details).
    **Advantages**
    (*i*) The mother is awake and can see the baby at delivery.
    (*ii*) Patient remains conscious during and after cesarean birth.
    (*iii*) Post operative problems, pain and Mendelson syndrome (acid aspiration syndrome) are less after epidural anesthesia.
    (*iv*) Breastfeeding and mobilization can start early.
    **Disadvantages**
    (*i*) The procedure takes longer (may not be suitable for acute emergencies like foetal distress, cord prolapse, antepartum haemorrhage, etc.).
    (*ii*) Sometimes anesthesia is incomplete.
    (*iii*) Some women are apprehensive and want to have general anesthesia.

**Table 42.17: Indications of Cesarean Delivery**

| | |
|---|---|
| **I. Maternal indications** | |
| 1. | **Causes in the bony passage**<br>(a) Moderate to severe degree contracted pelvis<br>(b) Mild degree contracted pelvis with some complicating factor. |
| 2. | **Causes in the soft passage**<br>(a) Cervical fibroid<br>(b) Vaginal atresia (rare)<br>(c) Cervical dystocia<br>(d) Carcinoma cervix<br>(e) Other causes of obstructed labour, like impacted fibroid or ovarian tumors in the pouch of Douglas |
| 3. | **Previous uterine scar and other operations**<br>(a) Past classical cesarean delivery<br>(b) Two or more cesarean deliveries in past<br>(c) Previous one cesarean birth with<br>(i) Recurrent indication like contracted pelvis<br>(ii) Other complicating factors, like malpresentation and antepartum haemorrhage<br>(iii) Slow progress, features of scar dehiscence or foetal distress appear during trial of labour.<br>(d) Previous myomectomy where uterine cavity was opened.<br>(e) Previous hysterotomy or perforation of uterus (relative)<br>(f) Previous Strassman's operation<br>(g) Previous repair of vesicovaginal fistula<br>(h) Previous sling operation for stress urinary incontinence<br>(i) Previous colporrhaphy with cervical amputation<br>(j) Past trachelectomy<br>(k) Permanent cerclage |
| 4. | **Abnormal labour and uterine contractions**<br>(a) Failed induction (b) Protracted labour<br>(c) Arrest of cervical dilatation<br>(d) Arrest of descent of presenting part<br>(e) Abnormal uterine action<br>(i) Uterine inertia (ii) Constriction ring |
| 5. | **Antepartum haemorrhage**<br>(a) Major degree placenta previa (Type II posterior, Type III and IV)<br>(b) Abruptio placentae with foetal distress |
| 6. | **Obstetric complications**<br>(a) Pre-eclampsia<br>(i) Pre-eclampsia not responding to conservative treatment<br>(ii) Severe pre-eclampsia and eclampsia with poor Bishop's score<br>(iii) Failure of induction<br>(iv) Foetal distress<br>(b) Bad obstetric history<br>(c) Elderly primigravida with some complicating factor<br>(d) Prolonged premature rupture of membranes |
| 7. | **Medical conditions**<br>(a) Cardiac disease<br>(i) Uncorrected coarctation of aorta<br>(ii) Pheochromocytoma<br>(iii) Prolonged labour<br>(b) Diabetes<br>(i) Bad obstetric history<br>(ii) Macrosomic fetus<br>(c) Pulmonary disease<br>(d) Cerebral aneurysm or arterio-venous malformation.<br>(d) HIV infection (Relative) - if not on HAART due to increased risk of transmission to fetus |
| 8. | **Miscellaneous**<br>(a) Active Herpes genitalis<br>(b) Cord prolapse<br>(c) Cesarean delivery on maternal request (CDMR) |
| **II. Foetal indications** | |
| 1. | Non-reassuring foetal heart status (formerly foetal distress) |
| 2. | Malpresentations and malpositions of the fetus<br>(a) Face posterior (mento-posterior) presentation<br>(b) Brow presentation<br>(c) Shoulder presentation (transverse lie)<br>(d) Oblique lie<br>(e) Unstable lie<br>(f) Breech presentation. Most obstetricians these days perform cesarean for all breech presentations. However, the following are indications.<br>(i) Any associated complicating factor<br>(ii) Estimated foetal weight > 3.5 kg.<br>(iii) Small premature fetus (in developed countries)<br>(iv) Slow progress of labour<br>(g) Persistent occipito-posterior presentation or deep transverse arrest with borderline pelvis |
| 3. | Foetal macrosomia (> 4.5 kg) |
| 4. | Multiple pregnancy<br>i. Abnormal presentation of first twin<br>ii. Conjoined twins<br>iii. Triplet and higher rate pregnancies |
| 5. | Certain foetal anomalies detected by ultrasound |
| 6. | Abnormal umblical cord Doppler study |
| 7. | Thrombocytopenia |
| 8. | Neonatal birth trauma in past. |

(b) General anesthesia. It is rarely used if patient demands it, where hurried delivery is required (foetal distress and cord prolapse) and in antepartum haemorrhage (not always) and in pre-eclampsia with abnormal coagulation profile where regional anesthesia is contraindicated. General anesthesia may be difficult due to laryngeal edema in pregnant patients.

15. Surgical safety protocol should be used in all cases.

**Table 42.18: Absolute Indications, Most Common Indications and Contraindications of Cesarean Delivery**

**Absolute indications**

These are very few conditions where vaginal delivery is not technically possible or is too dangerous for the mother. In these conditions cesarean delivery is often necessary even for dead fetus to save life of mother.

1. Severe degree of major placenta previa especially central placenta previa.
2. Severe degree contracted pelvis or cephalopelvic disproportion.
3. Large pelvic mass causing mechanical obstruction to the descent of foetal head like cervical or broad ligament fibroid, ovarian tumor impacted in pouch of Douglas.
4. Carcinoma cervix (especially late stages).
5. Vaginal atresia or stenosis

**Most common indications (85%)**

1. Previous cesarean for recurrent indication for recurrent indication is the most common indication.
2. Dystocia or failure to progress
3. Foetal distress (non-reassuring foetal heart rate)
4. Abnormal foetal presentation especially Breech presentation

**Contraindications**

These are very few. It is to be avoided in the following conditions.

1. Dead fetus
2. Very preterm fetus, unlikely to survive
3. Bleeding diathesis

## Technical aspects

Antiseptic painting of abdomen and thighs with chlorhexidine solution or 7.5% povidine iodine solution should be done just prior to cesarean. The instruments tray for cesarean delivery is shown in Fig. 47.89 in Chapter 47. The abdomen should be properly draped with sterile towels.

### Skin Incisions

Two basic skin incisions are used—suprapubic transverse and midline vertical. Paraumbilical (right or left) incision is not usually used for cesarean delivery. Differences between the two are shown in Table 42.19.

#### 1. The Vertical Infra-umbilical Incision

The vertical skin incision extends from about 2.5 cm below the umbilicus to the upper border of symphysis pubis. It is specially indicated in cases of extreme maternal obesity or where fast delivery is required like foetal distress or if suspicion of other intra-abdominal pathology necessitating surgical intervention or where access to the uterine fundus may be required (classical cesarean delivery) or with previous vertical incision.

#### 2. Transverse Incision

Skin incision should preferably be Pfannenstiel's (supra pubic transverse curvilinear incision using sharp dissection called sharp technique) or Cohen's (suprapubic transverse incision with lateral displacement of sheath by fingers called blunt technique) for better cosmetic results.

**Table 42.19: Differences Between Transverse and Vertical Skin Incisions**

| | Transverse incision | Vertical incision |
|---|---|---|
| Cosmetic appeal | More | Less appealing |
| Post operative pain | Less | More |
| Wound dehiscence | Less chance | More chance |
| Incisional hernia | Less chance | More chance |
| Technical skill | More technical skill required | Less technical skill required |
| Time taken | More time consuming | Less time consuming |
| Access to upper abdomen | Less access | Good access and can be extended, if needed |

The anatomical layers incised in both incisions are skin, fat, rectus sheath rectus abdominis muscle (separated), abdominal and visceral peritoneum and uterine muscle. In Maryland incision, the rectus abdominis muscles are cut (transected) on either side towards operating space.

### Packing

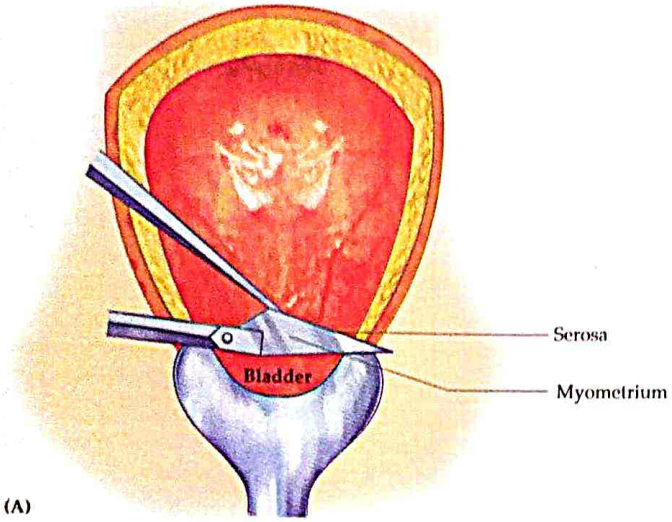
A Doyen's retractor is now inserted. Two tailed large gauze swabs with artery forceps attached to their ends for easy removal later can be used on either side in the lateral recesses of the wound. Packs minimize the contamination of blood and amniotic fluid into the general peritoneal cavity and are specially useful for meconium stained liquor and where infection in amniotic fluid is suspected. Free swabs should not be used as they can be lost. One assistant keeps mopping and sucking the blood and amniotic fluid to keep the operative field clear.

**Uterine incision.** Once the abdomen is opened, any dextrorotation of the uterus should be corrected. A Doyen's retractor is used to visualize the lower segment.

**Peritoneal incision.** After identifying the loose peritoneum over the lower segment, uterovesical fold is opened with scissors. Then bladder is pushed down by blunt dissection (Fig. 42.31). Large blood vessels are sometimes seen coursing over the lower segment. They should be ligated and cut before proceeding with the operation to avoid excessive haemorrhage.

**Muscle incision.** Next, a small transverse incision is made in the lower segment (Fig. 42.32) and extended laterally with the fingers (Fig. 42.33 A) or by scissors (Fig. 42.33 B) with concavity upwards to avoid uterine arteries. If there is inadequate space, rarely a J-shaped or inverted-T incision may be needed. Care should be taken not to injure the uterine vessels laterally. The various types of uterine incisions are shown in Fig. 42.34.

It is very important to make the uterine incision



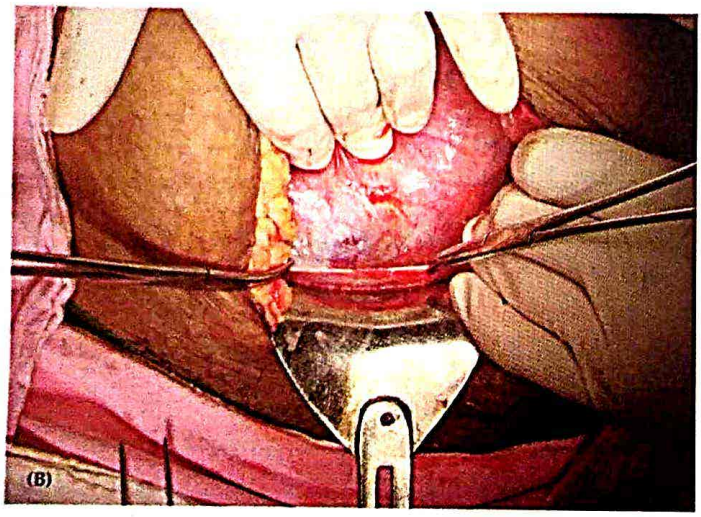




**Fig. 42.31:** Identification and division of loose peritoneum over the lower uterine segment and its separation from the bladder. (A) Diagramatic picture; (B) Actual operative photograph

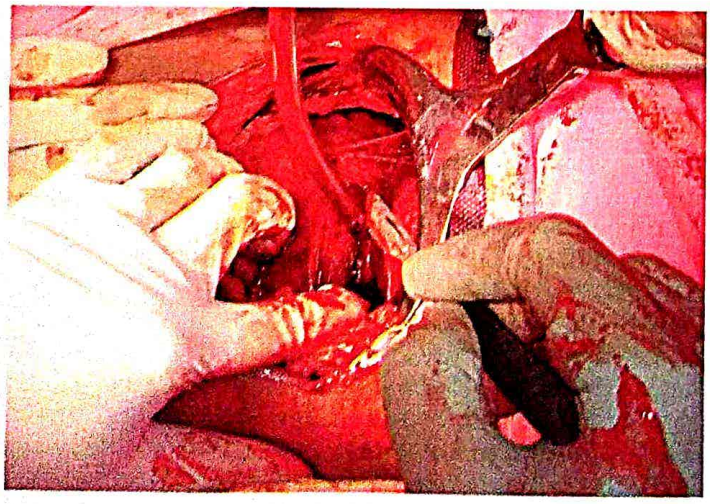


**Fig. 42.32:** Making of small transverse incision in the lower uterine segment.

arge enough (about 10 cm) to allow delivery of the head and trunk of the fetus without either tearing into or having to cut into the uterine arteries and veins which run through the lateral margins of the uterus. If the placenta is situated in the line of incision, it must be either detached or cut. When the placenta is incised, foetal haemorrhage may be severe. Thus, delivery and cord clamping should be performed as quickly as possible in these cases.

### Delivery of the Head

The amniotic membranes are artificially ruptured if still intact. The assistant sucks amniotic fluid mixed with blood with suction. In cephalic presentations, the right hand is slipped into the uterine cavity. Doyen's retractor is taken out and the head is gently levered out of the incision by hooking with fingers carefully inserted between lower uterine flap and the head aided by fundal pressure by assistant or by operator's left hand (Fig. 42.35).

Some obstetricians prefer the use of the obstetric forceps (Wrigley's or Barton's) as vectis to deliver the head especially in the case of a floating head. Ventouse can also be used for extraction of head.

A floating head can be made to engage in the incision by fixing in that position by the left hand or by the assistant and can be delivered out. Rarely internal podalic version and breech extraction may have to be done to deliver the fetus.

When the head is deeply wedged in the cavity, getting the hand well behind the head and levering it out is usually successful. An assistant can push the head up from the vagina below for easy delivery through uterine incision. For deeply impacted and jammed head sometime **Patwardhan technique** can be used for delivery of the fetus. In this technique shoulders which are coming in the incision line are delivered first followed by trunk and limbs and the head is delivered in the end. Use of foetal pillow which is a distensible intravaginal balloon which when inflated elevates the foetal head can be used but not easily available in India.

If the presentation is breech, the feet are first hooked out and the rest of the body delivered as in case of a vaginal breech delivery. In transverse or oblique lie, the lie is corrected to a longitudinal one before the uterine incision is made. In cases where this is not possible, the operator's hand is introduced into the uterus and after locating the foot, it is gently pulled out and the rest of the delivery conducted as in breech. If there is difficulty in delivery of the fetus and extension of the uterine incision is needed, J-shaped, U-shaped or an inverted T-incision may be needed. In the past, after delivery of the head of fetus, the secretions from the mouth and nostrils were sucked out with suction tube to prevent aspiration. This practice is not recommended nowadays.

### Delivery of Trunk (Figs. 42.36 A and B)

The shoulders are then delivered one at a time (usually anterior shoulder first followed by the posterior shoulder) (Figs. 42.36 A and B) using gentle traction aided by fundal pressure. The foetal trunk and lower limbs are then delivered by lateral flexion.

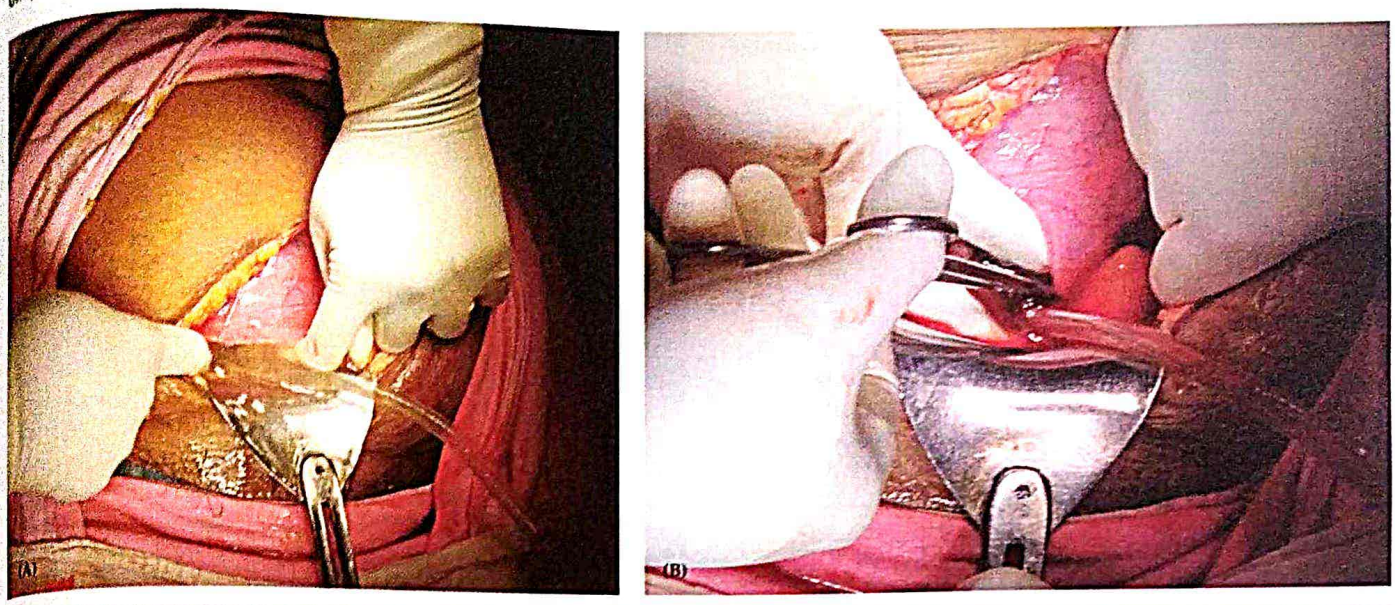



**Fig. 42.33:** (A) Extension of the incision laterally with fingers; (B) Extension of the incision laterally with scissors

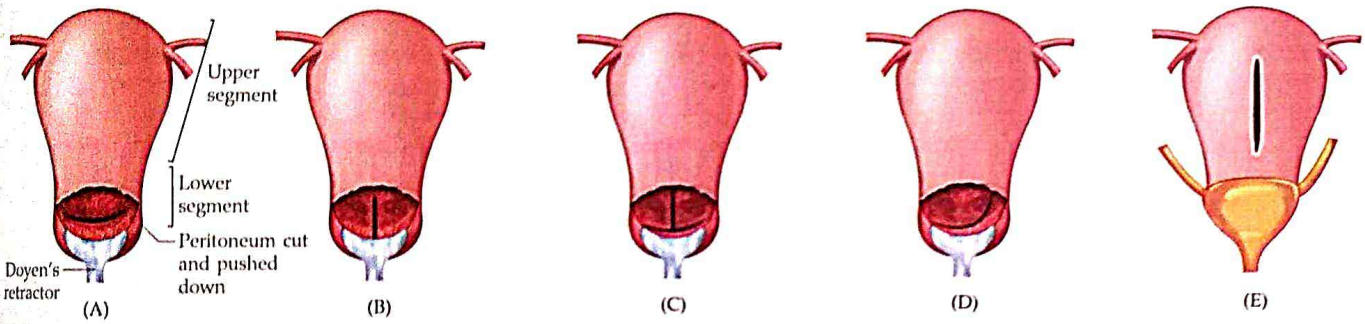



**Fig. 42.34:** (A) Lower segment transverse incision; (B) Lower segment vertical incision; (C) Inverted-T incision; (D) J-shaped incision; (E) Classical incision

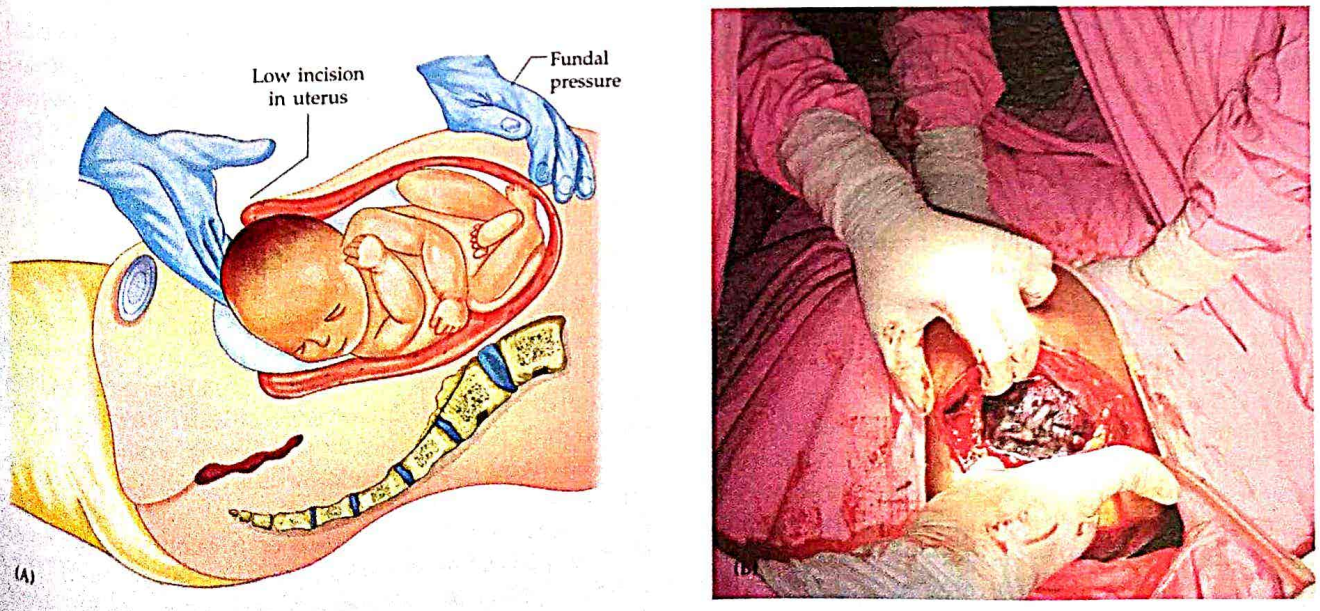



**Fig. 42.35:** (A) Delivery of foetal head with right hand with fundal pressure by left hand or by assistant; (B) Actual photograph

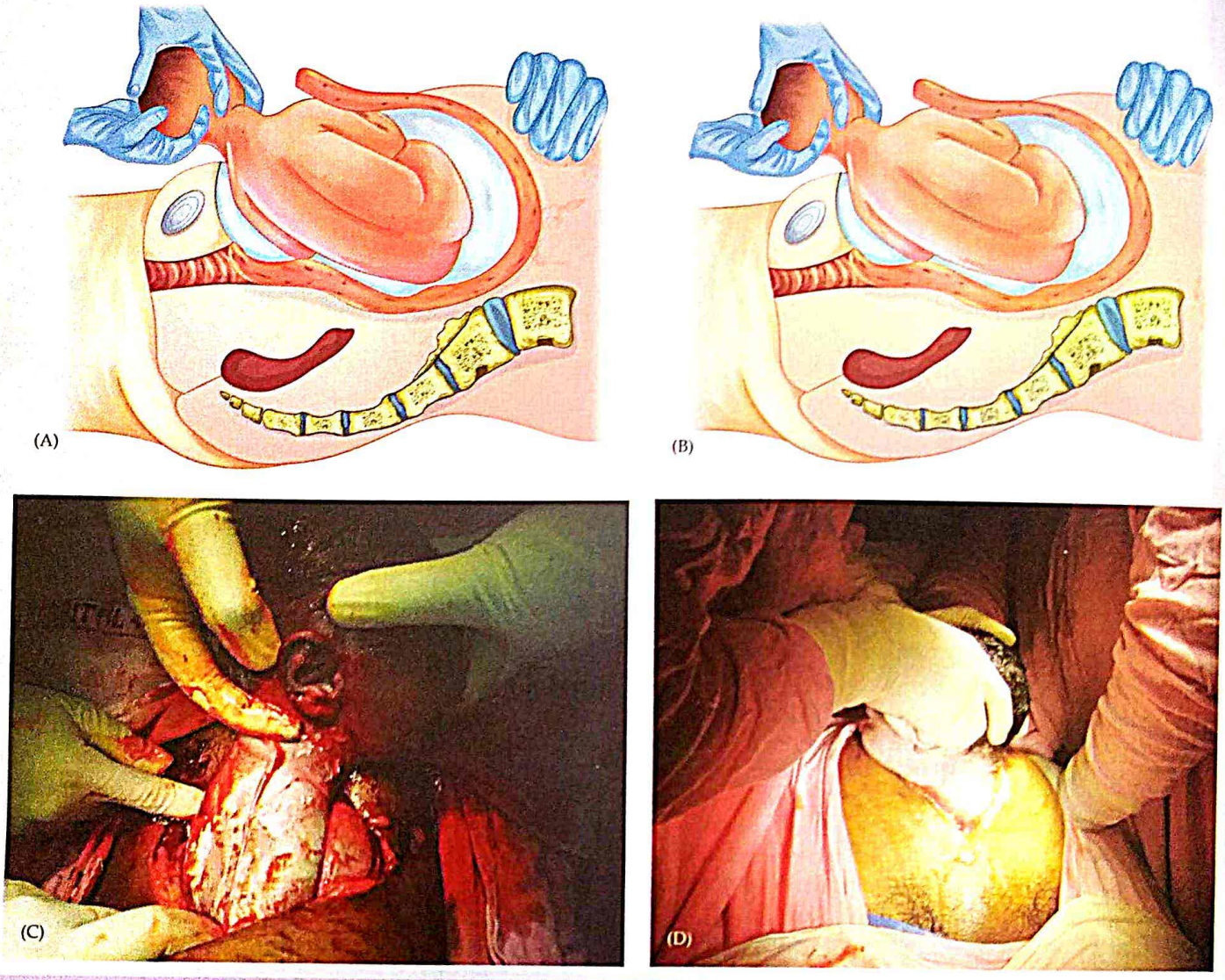



**Fig. 42.36:** As the foetal head is lifted through the incision, pressure usually is applied to the uterine fundus through the abdominal wall to help expel the fetus. (A) Anterior shoulder is delivered; (B) Posterior shoulder is delivered; (C) Actual photograph of delivery of anterior shoulder; (D) Actual photograph of delivery of posterior shoulder

At the delivery of shoulders, anesthetist is asked to start oxytocin drip by adding 20 units of oxytocin in one litre of saline or Ringer lactate solution and to give it at a rate of 10 mL/minute until the uterus contracts satisfactorily, after which the rate can be reduced.

Carbetocin: a longer acting oxytocin derivative can be used instead of oxytoin for better prophylaxis against PPH but is more expensive. Ergometrine or carboprost (PGF2α) are second line drugs used to treat atonic PPH. Misoprostol 400 µgm orally can also be used. Injection tranexamic acid 500–1000 mg IV can be given to reduce blood loss.

Delayed clamping of cord is done (1 minute) unless fetus is distressed or meconium stained (when early clamping is done) and the infant is handed over to the attending pediatrician for resuscitation. The uterine incision is observed for any bleeding sites which should be promptly clamped with Green Armytage forceps or Allis forceps. The placenta is then delivered by controlled cord traction unless it has already been delivered (Fig. 42.37). Many surgeons prefer manual removal of placenta, but spontaneous delivery with some controlled cord traction is better and has been shown to reduce the risk of puerperal metritis and blood loss **(level IA evidence)**. Placenta can also be delivered using placental drainage method in which its blood is allowed to be drained followed by its delivery by pulling it out. Fundal massage, begun as soon as the fetus is delivered, reduces bleeding and hastens placental delivery. The uterus should then be inspected to ensure complete removal of the placenta and membranes. Some authors advise confirmation of a patent cervical canal by introducing a finger in cervix to ensure a patent passage for the drainage of lochia especially in elective cesarean although, it is not necessary in laboring women.

The American Academy of Pediatrics and the American College of Obstetricians and Gynecologists (2017) recommend attendance of a qualified person who is skilled in neonatal resuscitation (usually a pediatrician) in the operative delivery room, with all equipment needed for neonatal resuscitation, to care for the neonate. Most hospitals in India routinely call a pediatrician or

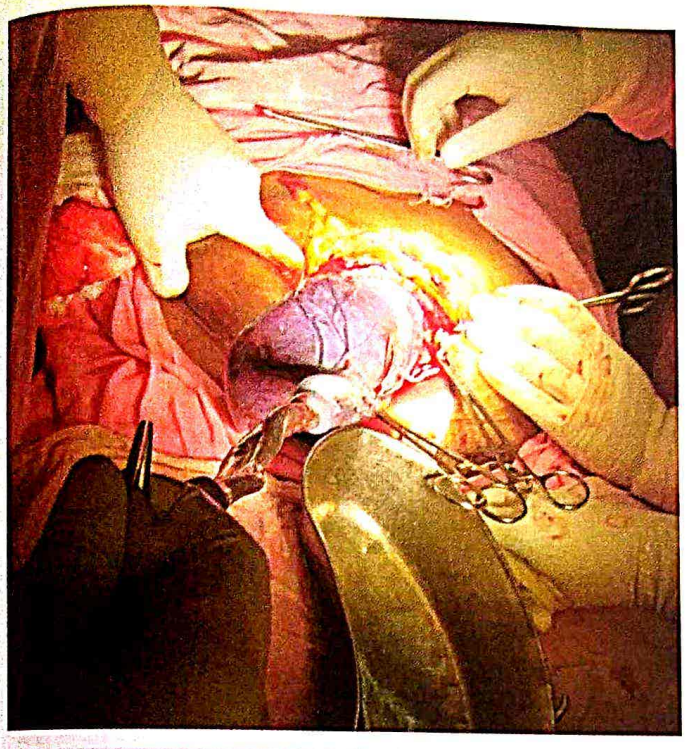


**Fig. 42.37:** Placenta being delivered by controlled cord traction (actual photograph)

neonatologist for all cesarean deliveries but certainly for high risk cases. Obstetricians should also be trained in foetal resuscitation.

**IUCD Insertion:** If consent has beam taken CuT 380A can be inserted in upper fundus through uterine wound with its threads guided towards cervix before closing the uterus. Bard-Parker blade (scalpel with blade) can also be used for cesarean operation usually 22 number blade is used. It has a handle with a notch to fit the blade and is usually held by pencil grip.

### Suture of Uterine Wound

Doyen's retractor is reinserted. Repair of the incision may be performed with the uterus in situ or following exteriorization (delivery of uterus outside the abdominal cavity) for better visualization and repair; especially in case of lateral extension of the incision margins.

Placement of Green-Armytage forceps or Allis tissue forceps on the inferior uterine edge in addition to each angle (total four) improves visualization and reduces blood loss further, especially in the presence of bleeding venous sinuses. Closure should be performed in either single or double layers with continuous or interrupted sutures using delayed absorbable synthetic polyglactin (vicryl) 1 or 1–0 suture though chromic catgut can also be used. For single layer closure, whole thickness of myometrium is stitched in one layer. For two layer closure, inner two-thirds of myometrium is stitched in first layer and outer one-third of myometrium is stitched in second layer. The initial suture should be placed just lateral to the incision angle and the closure continued to a point just lateral to the angle on the opposite side. A running stitch is often employed and this may be locked to improve hemostasis (Fig. 42.38 A). If a second layer is used, an inverting suture or horizontal suture should overlap the myometrium (Figs. 42.38 B and D). While stitching the uterus, needle-stick injuries on surgeon and assistant should be avoided. **A single-layer closure is associated with reduced operating time with no statistically significant difference in the use of extra hemostatic sutures, incidence of endometritis, decrease in post operative hematocrit or need of blood transfusion (level IB evidence).** Though there is some concern of increased risk of scar dehiscence in future pregnancies with one layer closure, it appears to be safe and many hospitals now routinely have a policy of one layer closure. Large CORONIS trial on cesarean delivery did not find any difference in outcome with use of catgut or vicryl, one layer closure or two layer closure and peritoneal closure or non-closure. Its follow-up study also did not find any long term adverse effects of either techniques.

After repair, the incision is assessed for hemostasis and 'figure-of-eight' sutures can be inserted to control bleeding.

Visceral peritoneum can be closed with 1–0 plain or chromic catgut, if needed. The uterus, tubes and ovaries are inspected. Elective or emergency procedures like tubal ligation, ovarian cystectomy for dermoid cyst can be performed. Abdominal packs are removed, if inserted. Many surgeons routinely place drains in the subfascial space to reduce haematoma formation, but such management is most appropriate in the presence of infection or in the obese, where a large 'space' is potentially left. In all cases, attention to hemostasis must be done.

### Abdominal Closure

The peritoneal cavity is cleaned and swabs and instrument count is performed.

There is no need to close the parietal peritoneum or rectus muscles as it is proven by studies that their closure is not required. Peritoneal closure may result in a higher incidence of adhesion formation **(level 1A evidence)**. If stitched polyglactin (vicryl) or chromic catgut number 1–0 suture can be used. Drain can be left in obese patients, high risk cases or where hemostasis is an issue.

Rectus sheath should be closed with a delayed absorbable suture with good tensile strength (Fig. 42.40). Polydioxanone or polyglactin (vicryl) no. '1' are ideal sutures for transverse incision while non-absorbable double loop nylon, prolene or delayed absorbable polydioxanone is ideal for vertical incision and high risk cases in transverse incision especially repeat cesarean delivery. Catgut should not be used for the rectus sheath.

There is no need to take stitches in the Camper's fascia and subcutaneous fat unless it is more than 2 cm thick when it is better to stitch it with chromic catgut 1–0 or 2–0 suture. The skin is usually stitched by non absorbable interrupted nylon (3–0), prolene, silk or polyester sutures which have to be removed later (Fig. 42.41 A). It can also be closed by absorbable subcuticular vicryl rapide or poliglecaprone (monocryl) (Fig. 42.41 B) for better cosmetic

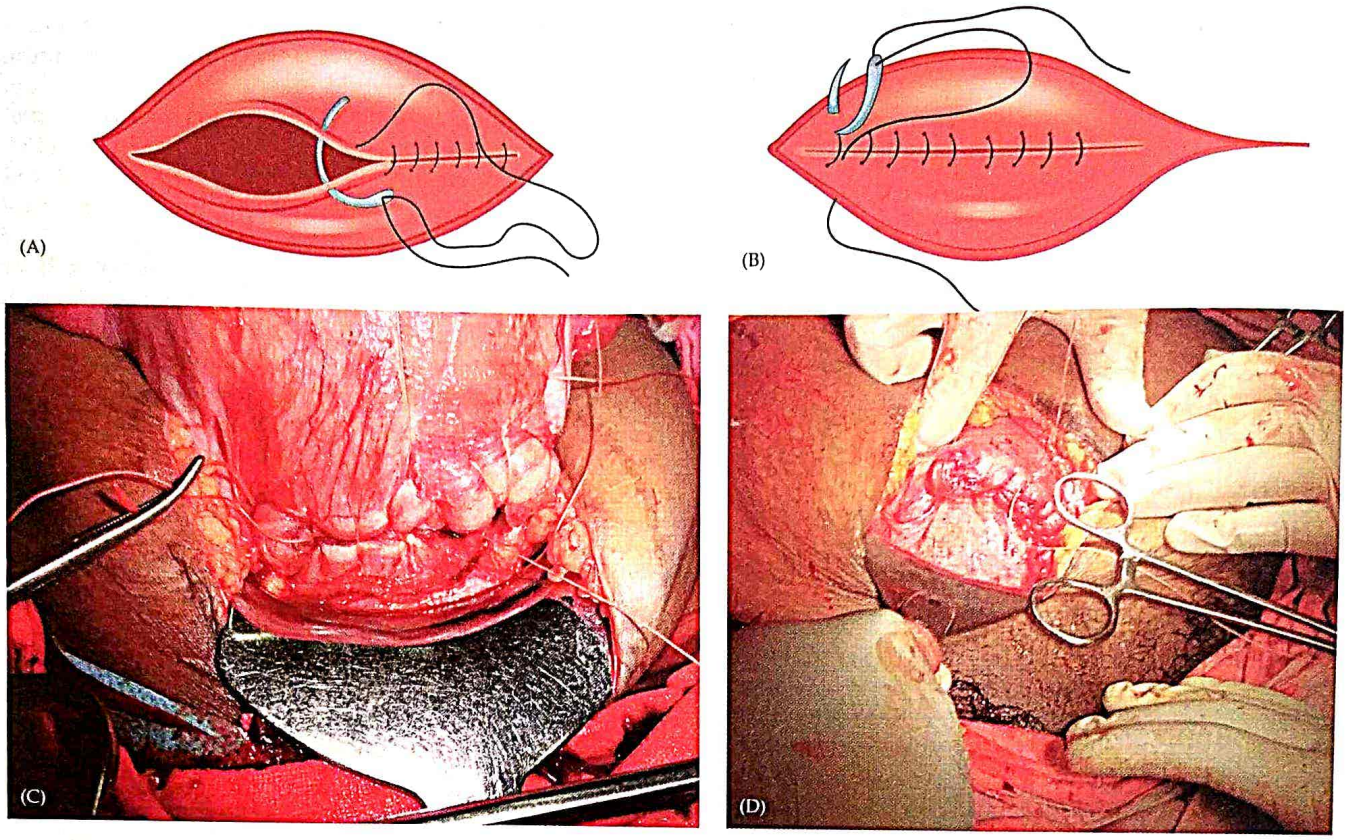



**Fig. 42.38:** (A) Closure of the first layer of uterine incision (inner two-thirds of myometrium) with polyglactin (vicryl), 'O' (1-0) suture; (B) Closure of the second layer of the uterine incision (outer one-third of myometrium) with polyglactin (vicryl), no 'O' (1-0) suture; (C) Actual photograph of first layer closure; (D) Actual photograph of second layer closure

results and less post operative discomfort. Surgical staples can also be used (Fig. 42.41 C). Complete hemostasis must be achieved at all levels of stitching.

Operation should not prevent early breastfeeding. Notes must be meticulously written in the file.

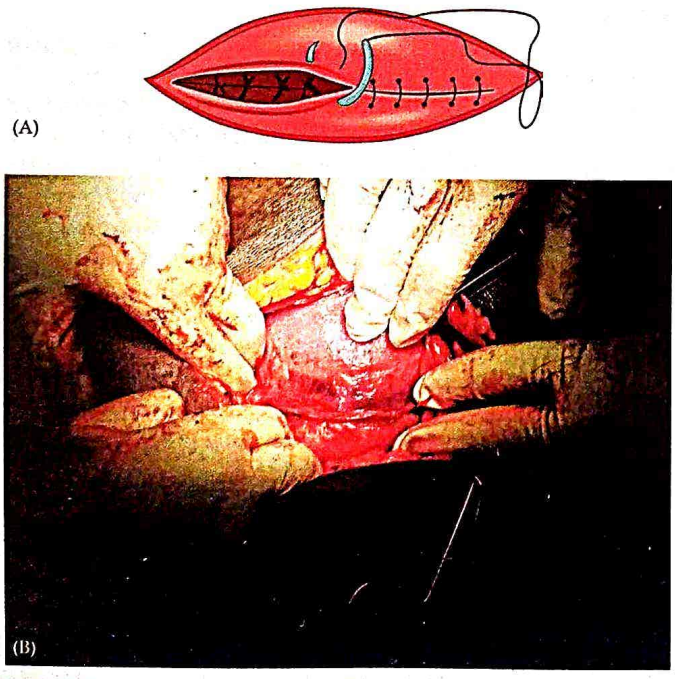



**Fig. 42.39:** (A) Closure of visceral peritoneum (uterovesical pouch) with chromic or plain catgut no. 1–0 or 2–0 suture; (B) Actual photograph of closure of visceral peritoneum

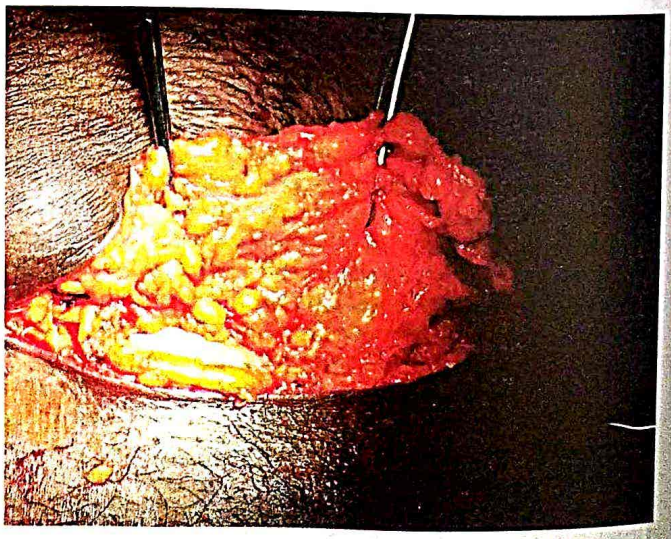


**Fig. 42.40:** Closure of rectus sheath with polyglactin (vicryl). No 'I' suture (actual photograph).

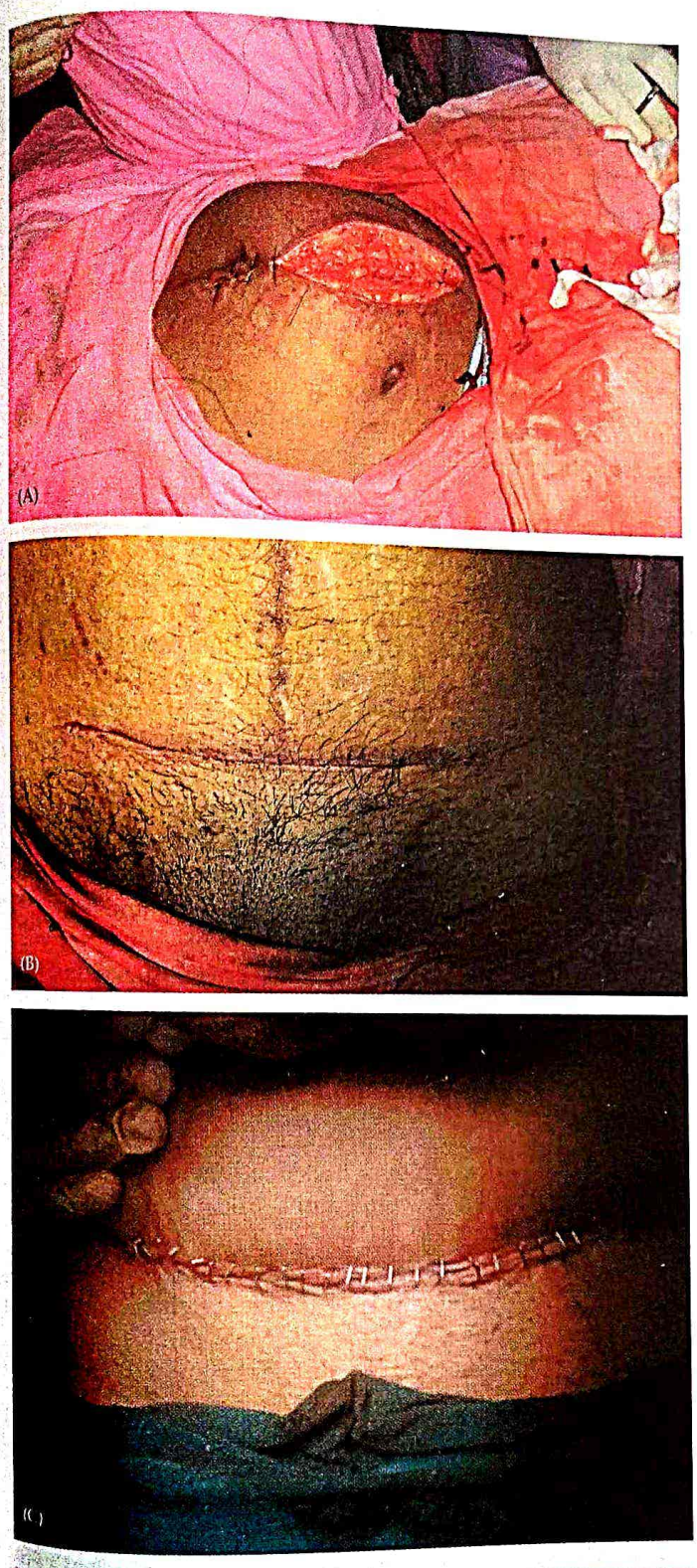


Fig. 42.41: (A) Closure of skin with interrupted 3–0 nylon suture; (B) Closure of skin with 3–0 monocryl (monofilament) subcuticular suture; (C) Closure of skin with staplers

## JOEL-COHEN AND MISGAV LADACH METHOD OF CESAREAN DELIVERY

Misgav Ladach method was started by Stark from Israel's Misgav Ladach hospital which is a very fast technique and is especially suitable for foetal distress patients in primary cesarean deliveries. Abdomen is opened by transverse 10 cm Cohen's incision at a higher level than Pfannenstiel incision. The rectus sheath is cut by a transverse 3 cm incision and then the incision is extended on both sides using surgeon's four fingers (two fingers below and two fingers above) in the center of the wound. By this the perforating vessels are not torn but are displaced laterally. The peritoneum is opened with fingers. Uterus is opened by a 1 cm stab in the center with extension with fingers, fast delivery of head, closure of uterine wound in one layer, non-closure of visceral and parietal peritoneum followed by closure of only rectus sheath and skin. The technique is faster and is associated with lesser blood loss and less post operative pain. However, it may be difficult to perform it with previous scar due to fibrosis and adhesions.

## POST OPERATIVE CARE

**First 24 hours (first day of operation).**

1. Patients are not allowed orally for 6 hours. After spinal anesthesia, they should not sit for six hours to avoid spinal headache. Foley's catheter is usually left for 24 hours so that the patients do not have to worry about passing urine or retention of urine and can have uninterrupted sleep. Some surgeons use in and out catheter for cesarean section. In that case the patients have to pass urine in 6 hours of operation. Recent studies and meta-analysis have confirmed the safety and less urinary tract infection rate without the use of catheter with cesarean being done after patients passed urine.
2. The patients should be carefully monitored for at least 6–8 hours by monitoring general condition. Pulse, temperature, blood pressure, vaginal bleeding and uterine tone are to be regularly checked for any postpartum haemorrhage.
3. **Fluids.** About 2–2.5 litres of the Ringer's lactate or dextrose saline are infused. Routine blood transfusion in all cases is not given. It is given only for excessive blood loss **(blood loss in cesarean delivery can be upto one litre)**
4. Oxytocin drip is continued for 6–8 hours in high risk cases like antepartum and postpartum haemorrhage. Injection methyl ergometrine 0.2 mg or inj. prostaglandin $F_2\alpha$ (carboprost) 250 µg intramuscularly may have to be given.
5. Prophylactic antibiotics are usually prescribed parenterally for the first 48 hours as inj. coamoxyclav 1.2 g IV 8 hourly or combination of inj ampicillin 500 mg IV 6 hourly, inj. Gentamicin 60 mg IM/IV 8 hourly and inj. metronidazole 500 mg/IV 8 hourly.
6. Analgesics in the form of injection diclofenac 75 mg or inj. Ketorolac 30 mg IM 8 hourly or inj. paracetamol 1 g (100 mL infusion) 6–8 hourly are given. Inj. Tramadol 50 mg intravenously 8 hourly can be given for severe pain preceded by antiemetic metoclopramide (10 mg) or ondansetron (4 mg) parenterally.
7. **Ambulation.** Early ambulation is encouraged. After 6 hours in spinal anesthesia, she can sit up and can briefly be out of bed and walk with the help of an assistant.

She should perform deep breathing exercises, move her legs and feet to prevent deep vein thrombosis.

8. **Breast care.** She should support her breasts without marked compression to minimize discomfort. Early breast feeding within 2–4 hours is started.

**First Post operative day or day 2 of operation**

1. Pulse, blood pressure, temperature are checked. Heart and lungs are auscultated.
2. Vaginal bleeding is seen, uterine contour is checked.
3. IV fluids (2 litres), IV antibiotics and IM analgesics are continued like on the first day.
4. She can walk without assistance.
5. Oral clear fluids are started in the morning in form of water, tea, soup, coconut water. If she tolerates them, biscuits can be given with tea.
6. Bowel sounds are ausculated with stethoscope and are usually present.
7. Catheter is removed.

**Second Post operative day or day 3 of operation: Revised treatment**

1. Temperature, pulse, respiration and blood pressure are checked.
2. Oral semi-solid diet like lentils (dalia) and rice with pulse (khichri) are given.
3. Tablet cefixime 200 mg twice day × 5 days or tab cephalexin 500 mg 6 hourly × 5 days are given. Alternately capsule amoxycillin with clavulanate 375 mg TDS × 5 days or
   Capsule amoxycillin 500 mg 8 hourly × 5 days with
   Tablet metronidazole 400 mg TDS × 5 days
   Inj. Gentamicin 60 mg IM 8 hourly × 5 days can be given to high risk cases.
4. Tablet diclofenac or ibuprofen with paracetamol TDS for 5 days.
5. If patient has not moved her bowels, lactulose 15–30 mL at bed time can be given.
6. Ambulation is allowed.
7. For any cough or upper respiratory tract infection, steam inhalation is useful. A cough syrup like codeine 2 teaspoons can be prescribed 2–3 times a day.

**Day 4 of operation.** Light solid diet is allowed. Full diet from day 3–4, if passed stools. Dressing is changed.

**Day 5 of operation.** Normal diet is allowed.

**Day 6 or 7 of operation.** Stitches are removed on day 5 or 6 in transverse incision and on day 7 in vertical incision.

**Day 7 or 8 of operation.** Patient are discharged, if all is well. These days patients are discharged early on day 3 or 4 especially for transverse incision. She should come back to hospital on day 7 or 8 for stitch removal. She should be advised about contraception. Baby should be seen by pediatrician every day and before discharge. All immunizations (at birth) should be given to the baby and future follow-up is arranged.

During the first week she should take care of herself and baby only. Driving can be advised after 2 weeks. She can do house hold work after 2 weeks. Return to work is variable (6–12 weeks). However, maternity leave is of 6 months in India for new born care. Sexual activity can be started once she feels comfortable (usually 6 weeks) with use of contraception (e.g. condom or IUCD or progestogen only pill)

## COMPLICATIONS

Although cesarean has become very safe recently due to better anesthesia, blood and good antibiotics, it must never be forgotten that cesarean delivery is a major abdominal surgical procedure and carries with it all the risks of a major surgery.

### 1. Intra-operative Complications

The patients undergoing cesarean delivery is at risk of many complications which are not faced by a patient undergoing a vaginal delivery.

(*i*) **Extension of uterine incision in difficult cesarean delivery.** Uterine arteries may be involved leading on to broad ligament haematoma formation. The lateral apex of the extension should be identified and the suture placed just lateral to that point.

(*ii*) **Bladder and ureteral injuries.** Bladder injury is seen in 1.4/1000 procedures while ureteral injury is seen in 0.3/1000 procedures. They are more common in previous cesarean cases.

(*iii*) **Gastrointestinal injury.** Prior abdominal surgery and pelvic and abdominal infection leading to adhesion formation are common risk factors.

(*iv*) **Uterine atony and primary postpartum haemorrhage.** See Chapter 25 for details of management.

(*v*) **Morbidly adherent placenta like placenta accreta.** It is now the second most common indication for post cesarean hysterectomy (most common being atonic PPH).

(*vi*) **Anaesthetic complications** (see Chapter 19 for details).

a. Difficult intubation
b. Anaphylaxis
c. Aspiration
d. Mendelson's syndrome
e. Hypotension
f. Total spinal blockade
g. Cardiac arrest

(*vii*) **Difficulty in delivery of the head**

(*viii*) **Shock**

### 2. Post-operative Complications

The major sources of morbidity and associated mortality are related to complications of maternal sepsis, anesthesia and thromboembolic disease and its complications.

#### Maternal Complications

***Immediate Complications***

(*i*) **Postpartum haemorrhage**

(*ii*) **Shock**

(*iii*) **Anaesthetic complications.** Similar to intraoperative complications (see above).

(*iv*) **Infections.** Endometritis is the most common post operative infection after cesarean delivery with

incidence being 10–20 times higher for cesarean delivery than for vaginal delivery. Risk factors are prolonged labour, premature rupture of membranes, obesity, repeated vaginal examinations (see Chapter 30 for details). Other infections are septic pelvic thrombophlebitis, upper respiratory infection, urinary tract infections, wound infection and pelvic abscess. Routine prophylactic antibiotics can reduce incidence of infection by 60%.

(v) **Wound complications.** Incidence of wound infection is 1–4.5%. The causative organisms are Staphylococcus aureus, Streptococci, anerobes and Gram negative bacilli. Wound swab should be sent for cuture sensitivity. There can be wound disruption (dehiscence) followed by abdominal pain and serous or serosanguinous discharge from the wound causing partial burst or complete burst abdomen.

(vi) **Haematoma** formation can occur in 2.5–5.5% cases in retrovesical area or rectus sheath causing infection and fever and may need relaparotomy.

(vii) **Retained foreign body** like sponges or instruments can occur.

(viii) **Psychological effect**

(ix) **Thromboembolic disorders.** The incidence of various thromboembolic disorders like deep vein thrombosis and pulmonary embolism is more after cesarean delivery than normal delivery. Septic thrombophlebitis can also occur (see Chapter 30 for details).

(x) **Paralytic ileus**

(xi) **Intestinal obstruction.** The obstruction can occur from adhesions or bands or from paralytic ileus or injury to bowel.

(xii) **Respiratory complications**

(xiii) **Lactation failure**

*Late Sequelae*

(i) **Secondary postpartum haemorrhage**

(ii) **Incisional hernia**

(iii) **Scar endometriosis**

(iv) **Vesicovaginal fistula**

*Remote Complications*

(i) **Gynecological complications** like menstrual dysfunction, chronic abdominal and pelvic pain and chronic backache.

(ii) **General surgical complications.** Incisional hernia at abdominal incision due to poor stitching of rectus sheath, intestinal obstruction from adhesions and bands.

(iii) **Future pregnancy.** There is risk of scar rupture in subsequent pregnancies (see Chapter 39 for details). There is also risk of placenta previa and accreta in subsequent pregnancy. There is also increased risk of bladder injury in next cesarean.

### Foetal Complications

Iatrogenic prematurity and risk of hyaline membrane disease or respiratory distress syndrome is more following cesarean delivery than vaginal delivery especially when foetal maturity is not certainly known. Foetal injury at the time of cesarean delivery like laceration on scalp, face, buttocks (in breech presentation) can occur due to knife injury at the time of incision to uterus. There can be rarely neurological injuries (Erb's paralysis, Klumpke's paralysis) or fracture clavicle or other bones in difficult extraction of fetus.

## RESEARCH STUDIES IN CESAREAN DELIVERY

Caesar trial from United Kingdom compared single vs double layer closure of uterine incision, closure vs nonclosure of the pelvic peritoneum and liberal vs restricted use of subrectus sheath drain and did not find any major differences in outcome. Overall maternal infectious morbidity was 17 %. Coronis (2013) trial was conducted by National Perinatal Epidemiology Unit, Oxford (funded by Medical Research Council, UK) on 15,944 women from 19 hospitals in 7 countries including India (coordinated by the author in India) to see the short term outcome and differences of 5 interventions—Blunt vs Sharp Abdominal Entry, Exteriorization vs Nonexteriorization of Uterus during Uterine Closure, One Layer vs Two Layer Closure of Uterus, Closure vs Nonclosure of Visceral and Parietal Peritoneum and Catgut vs Vicryl Sutures. There were no statistically significant differences with any of the five surgical intervention pairs. Hence, either of the five pairs of techniques can be safely performed. The long term 3 years follow-up study was also published (CORONIS follow-up study 2016). The study observed all the five interventions to be equally safe and effective on longterm outcome. There was no difference in long term parameters like scar dehiscence or rupture or other morbidities in the different groups.

### Classification of Urgency of Cesarean Delivery (RCOG 2010)

Recent guidelines for time limits within which Cesarean section be performed.

**Category 1.** Immediate (15–30 mins) threat to the life of the woman or fetus. Includes cesarean section for severe prolonged foetal bradycardia, foetal scalp blood pH < 7.2, cord prolapse and uterine rupture should be done preferably in 15 minutes but certainly within 30 minutes.

**Category 2.** Maternal or foetal compromise which is not immediately life threatening. Includes conditions like antepartum haemorrhage, non-progress of labour with maternal or foetal compromise, but not to the degree of Category 1. These women should be delivered within 30 minutes if possible.

**Category 3.** No maternal or foetal compromise but early delivery required. Includes NPOL without maternal or foetal compromise, women booked for elective cesarean section admitted with ruptured membranes or in early labour. It is recommended that these women be delivered within 75 minutes.

**Category 4.** Elective planned cesarean section timed to suit the woman and staff.

## CLASSICAL CESAREAN SECTION

The advantages and disadvantages of lower segment versus classical cesarean are shown in Table 42.20.

### Indications

1. Where lower uterine segment cannot be exposed or if the bladder is densely adherent (previous cesarean) or lower segment fibroid.
2. Invasive carcinoma of the cervix
3. In morbidity adherent placenta, e.g. percreta in lower segment if diagnosed antenatally by ultrasound examination. It is becoming important indication.
4. In some cases of transverse lie with macrosomic fetus with hand prolapse.
5. Some cases of anterior placenta previa.
6. Urgent obstetric problems, e.g. cord prolapse and severe acute hypertension.
7. Previous classical scar
8. Some foetal malformations
9. Post mortem cesarean

However, many indications are relative. Infact Cohen's incision can be very fast. Infact carcinoma cervix is probably the only practical indication of classical cesarean in modern obstetrics.

### Steps

1. Bladder is evacuated.
2. Abdomen is cleaned and draped.
3. The abdomen is opened by midline or right paramedian incision but the skin incision is slightly longer.
4. The various layers of the abdominal wall are separated as done in lower segment cesarean section.
5. The uterine incision is a vertical incision into the body of the uterus above the lower uterine segment with a scalpel and then with scissors. The incision is extended till the surgeon gets enough room to deliver the baby.
6. The incision bleeds profusely as there are plenty of large vessels at the lower end of the incision in the uterus. The thickness of the segment is more and is not stretched and thinned out as in lower segment. These vessels have to be ligated as soon as the baby is delivered.
7. Repair of uterine incision is usually done in two layers as the thickness of upper segment is more. The approximation has to be done carefully so as not to leave any dead space. The first layer is a continuous layer with suture number 1 or 1–0 polyglactin (vicryl). This takes care of the inner half of the incision.
8. The outer layer is closed with same suture, by figure of eight or continuous layer. While doing this, the assistant should compress the myometrium on each side of the incision medially as each suture is placed and tied.
9. If the serosa could not be approximated it should be sutured with continuous suture with 1–0 or 2–0 chromic catgut.
10. The rest of the abdomen is closed as done in LSCS.

**Note.** The incision is made in the lower uterine segment vertically by some obstetricians. The low vertical incision can be extended upto the fundus if more surgical space is required.

## EXTRAPERITONEAL CESAREAN SECTION

It was practised in the past for severe sepsis. It can be done by the lateral approach (lateral incision parallel to inguinal ligament) or median approach (paravesical section). The bladder is denuded of the uterus and the lower segment is opened extraperitoneally through cave of Retzius. It is abandoned in current practice due to higher bladder injury rate and availability of effective antibiotics.

## PERIMORTEM CESAREAN DELIVERY

Perimortem cesarean delivery is rarely done in modern era. It is done after taking a prior written permission from relative. Prognosis of the fetus depends upon how quickly the cesarean is performed after maternal death being good if done within 4 minutes of collapse with failed CPR in absence of pre-existing maternal condition.

The operation is classical cesarean (see above) done by the most experienced personnel present, preferably obstetrician. Prompt resuscitation (wrapping clothes in winter) is immediately done and the newborn is transferred to the nursery. The uterus is stitched in one layer and the same is done for the abdomen.

## MATERNAL AND PERINATAL MORTALITY IN CESAREAN SECTION

### Maternal mortality

In USA, on 1.5 million pregnancies, maternal mortality rate was only 2.2 per 100,000 cesarean deliveries as compared to 0.2 per 100,000 vaginal deliveries. Maternal mortality increased to 13 per 100,000 with repeat elective cesarean delivery as compared to 4 per 100,000 in women undergoing trial of labour. Overall maternal mortality ranges between 6 per 100,000 and 22 per 100,000 procedure but can range from 0.1 to 1% in adverse circumstances. The causes of fatality are: (1) Excessive bleeding and shock (2) Anaesthetic complications (3) Sepsis (4) Thromboembolic complications.

### Perinatal mortality

Cesarean is safe for baby. The foetal death rate depends largely on the nature of condition for which the operation is performed. It also depends upon whether it was emergency or elective, indication of cesarean and foetal condition at time of cesarean. It is much higher for emergency cesarean and foetal distress.

**Table 42.20: Advantages and Disadvantages of Lower Segment Cesarean Delivery versus Classical Cesarean Delivery**

| Characteristics | Lower segment cesarean delivery | Classical cesarean delivery |
|---|---|---|
| Definition | In this type of cesarean delivery, the uterine incision is in the lower uterine segment which may be transverse or vertical ( usually transverse is performed in majority of cases. | In this type of cesarean delivery the vertical incision is made on the uterus which extends into the upper uterine segment or fundus. |
| Frequency | It is almost always performed in modern obstetrics. | It is rarely performed in modern obstetrics. |
| Indications | It can be performed for almost all the indications of cesarean delivery. | It is only performed for selected indications where there is difficulty or danger to enter the lower segment like carcinoma cervix, bladder densely adherent to lower segment, placenta previa with anterior implantation especially with placenta increta or percreta. |
| Operative Procedure | 1. Although it is technically slightly more difficult to do, all obstetricians are trained in it from the very beginning.<br>2. Less haemorrhage<br>3. The margins of lower segment wound are thin and stay close to each other, so perfect stitching and apposition is possible.<br>4. It can be closed in 1 or 2 layers.<br>5. It is more difficult to extract fetus in conditions like placents previa, increta or percreta or transverse lie with hand prolapse. However, in such cases vertical lower segment incision can be given. | 1. Although it is technically slightly easier to do, most obstetricians have gone out of practice to do it due to lack of training in it as it is rarely performed.<br>2. More haemorrhage<br>3. The margins of upper segment wound are very thick and tend to go away from each other, hence perfect stitching and apposition is not possible.<br>4. It always needs two or three layers closure.<br>5. It is technically easier to extract fetus through classical incision in conditions where lower segment is not accessible. |
| Post Operative Care and Complications | 1. Blood loss and shock are less common.<br>2. Blood transfusion is less commonly required.<br>3. Need for hysterectomy—ess common.<br>4. Need for intensive care unit and admission—less common.<br>5. Maternal death risk—lower<br>6. Puerperal infection—less common. The infection is localized to pelvis.<br>7. Perinatal morbidity less<br>8. Post operative adhesions and bands – less common.<br>9. Recovery – faster and better with early return to normal activities and work. | 1. They are more common.<br>2. More commonly required.<br>3. More common<br>4. More common<br>5. Higher<br>6. More common infection is more generalized spreading to upper abdomen.<br>7. More possibly due to preterm babies<br>8. More common<br>9. Slower and poorer with late return to normal activities and work. |
| Healing of scar | Scar of lower uterine incision heals much better due to following reasons.<br>1. Perfect surgical closure of thin wound margins.<br>2. Bleeding and haematoma at wound site—less common.<br>3. The lower segment remains relatively silent during puerperium favoring faster and better healing.<br>4. Less chances of gutter formation under the wound favouring scar healing. | Scar of classical cesarean heals poorly due to following reasons.<br>1. Less perfect closure of thick wound margins.<br>2. More common<br>3. Upper segment involutes much more with more contraction and relaxation impairing wound healing<br>4. More chances of gutter formation under the wound impairing scar healing. |
| Remote complications and behaviour during future pregnancies | 1. Remote complications like intestinal obstruction due to adhesions and bands, incisional hernia are less common.<br>2. Vaginal birth after cesarean can be tried.<br>3. Scar dehiscence and rupture:<br>a. It is much less (0.2-1.5%)<br>b. It usually ruptures only during labour.<br>c. Morbidity and mortality is much lower for low segment scar rupture.<br>d. It can usually be repaired in case of scar rupture and uterus can be saved.<br>e. Perinatal outcome even in scar rupture is excellent as fetus tends to stay in uterine cavity and is often alive. | 1. They are more common<br>2. It is an indication of repeat cesarean delivery. vaginal birth after cesarean is contraindicated.<br>3. Scar dehiscence and rupture<br>a. It is much more common being 4–9%.<br>b. It can even rupture during pregnancy.<br>c. Morbidity and even mortality is higher for classical scar rupture.<br>d. It usually needs subtotal or total hysterectomy in cases of scar rupture.<br>e. Perinatal outcome in classical scar rupture is usually poor as fetus tends to come out in peritoneal cavity and is usually dead. |

**Causes of Perinatal Mortality**

1. Prematurity
2. Respiratory distress syndrome (can be avoided by performing cesarean at 39 weeks rather than 38 weeks).
3. Hypoxia
4. Sepsis
5. Intra-cranial injuries due to difficult extraction like in obstructed labour, deeply impacted head, transverse lie and breech.
6. Rarely undiagnosed foetal malformations or conditions (e.g. pulmonary hypoplasia)

## HOW TO REDUCE HIGHER CESAREAN RATES

There is an epidemic of cesarean deliveries throughout the world which is a matter of serious concern as previous cesarean is associated with increased chances of placenta previa and morbidity adherent placenta in subsequent pregnancies often necessitating cesarean hysterectomy. Cesarean on demand in the hope of reducing the risks of perineal floor injuries, stress incontinence, relaxed perineum and prolapse in future is not justifiable. WHO has recommended a cesarean rate of 10–15%. American College of Obstetricians and Gynecologists had recommended a cesarean rate of 15.5% in USA and a vaginal birth rate after a prior cesarean (VBAC) of 37%. However, it has not been achieved.

### Methods to reduce cesarean rates

1. More liberal use of external cephalic version for breech presentation and transverse lie can lower cesarean rates.
2. Assisted breech delivery can be accomplished in suitable cases.
3. Maintenance of partograph and active management can reduce cesarean rate.
4. More liberal use of foetal scalp pH in foetal distress for foetal acidosis can also lower incidence of cesarean for this common indication.
5. There may be some role of ammioinfusion in selected cases.
6. Symphysiotomy can be done in some cases.
7. Destructive operations can be done in some cases.
8. Michael Robson (2001) from United Kingdom has classified cesarean section into 10 groups classification depending upon the indications and contribution of each indication towards cesarean rate and has recommended methods to lower cesarean births, especially in common indications (Table 42.21). WHO has also recommended use of Robson's classification to reduce cesarean rates.

## CESAREAN HYSTERECTOMY

Cesarean hysterectomy is removal of uterus at the time of cesarean section after delivery of the fetus. This operation, though a major operative decision, should not be left till too late, as the risk of operative complications, maternal morbidity and mortality increase with increasing haemorrhage.

**Table 42.21: ROBSON's 10 Group Classification of Cesarean Section**

| | |
|---|---|
| 1. | Nulliparous, single cephalic, ≥ 37 weeks, in spontaneous labour. |
| 2. | Nulliparous, single cephalic, ≥ 37 weeks, induced or CS before labour. |
| 3. | Multiparous (excluding previous CS), single cephalic, ≥ 37 weeks, in spontaneous labour. |
| 4. | Multiparous (excluding previous CS), single cephalic ≥ 37 weeks, induced or CS before labour. |
| 5. | Previous CS, single cephalic, ≥ 37 weeks. |
| 6. | All nulliparous breeches |
| 7. | All multiparous breeches (including previous CS) |
| 8. | All multiple pregnancies (including previous CS) |
| 9. | All abnormalities (including previous CS) |
| 10. | All single cephalic, ≤ 36 weeks (including previous CS) |

CS: Cesarean section

**Indications**

1. **Uncontrollable (intractable) atonic postpartum haemorrhage is the most common indication.**
2. Morbidly adherent placenta (accreta, increta and percreta) with bleeding is the **second most common indication.**
3. Uterine rupture
4. Multiple incisions where repair of incision is difficult.
5. Grossly infected uterus
6. Cancer cervix (Wertheim's hysterectomy can be done)
7. Tears and laceration in uterus extending into broad ligament with haematoma.

**Peripartum Hysterectomy.** Hysterectomy performed at or following delivery is called peripartum hysterectomy. It includes hysterectomy carried out with cesarean delivery (see above) or even after vaginal delivery.

**Supracervical (subtotal) hysterectomy** is usually performed in emergency (unplanned) situation as it is faster, causes less haemorrhage with reduced risk of injury to adjacent organs (bladder, ureter) and less post operative morbidity (Fig. 42.42). However, if possible, total hysterectomy can also be done.

**Steps.** The round ligaments are clamped, cut and ligated close to uterus on both sides and broad ligament is opened. The utero-ovarian ligaments and fallopian tubes are also clamped, cut and ligated on both sides. Bladder is mobilized in midline and laterally. Bilateral uterine vessels are clamped, cut and ligated close to the uterus. Corpus of the uterus is removed by putting two clamps on cervix. For total hysterectomy, clamps are put on lateral vaginal angles and Mackenrodt and uterosacral ligaments on both sides and cervix separated from vagina. Vagina is closed at vault

**Complications.** Apart from all the complications of cesarean, chances of bladder and ureteric injuries, haemorrhage, shock and infection are higher.

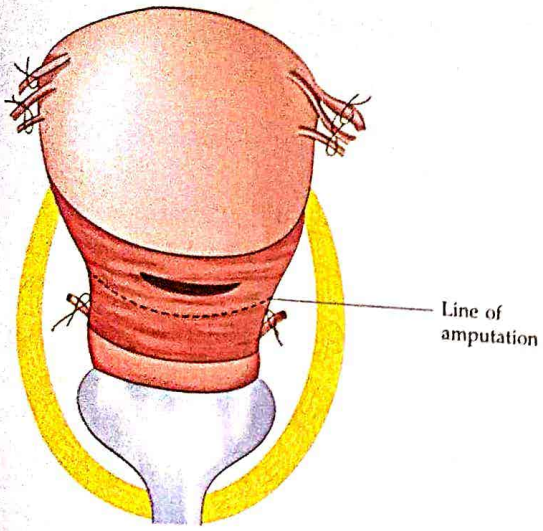



Fig. 42.42: Diagramatic representation of cesarean subtotal hysterectomy showing line of amputation by interrupted line

## SYMPHYSIOTOMY

Symphysiotomy is the division of the symphysis pubis to increase the pelvic dimensions. The incision is given in the midline from above downwards until the arcuate ligament is cut. It may be appropriate in Africa where incidence of contracted pelvis is high and women avoid cesarean due to cultural reasons. All diameters of pelvis are permanently enlarged. Transverse diameter of outlet is enlarged more than other diameters.

The fetus is delivered spontaneously or by instrumental delivery especially with liberal episiotomy.

In **pubiotomy**, pubic bone is divided on one side of symphysis pubis joint.

**Complications.** Hemorrohage, injuries to urethra and bladder, septic infection, osteitis pubis, retropubic pain, urinary incontinence and vesicovaginal fistula. It is not usually done in India. Delayed complication may include difficulty in locomotion.

## Key Points

1. Cesarean delivery (section) is the birth of a fetus through incisions in abdominal wall and uterus after 28 weeks of gestation. It is one of the most common operations in obstetrics with an incidence of about 15–20% (20–30% in USA). The incidence is still increasing due to lesser threshold of performing cesarean for at risk mothers, almost universal cesarean for breech, more use of electronic foetal monitoring, decline in use of difficult instrumental deliveries.
2. The indications can be absolute or relative and in essence are where vaginal delivery is either not possible or is risky for the mother or fetus. Indications can be in bony passage, soft pasage and in passenger (fetus). Contracted pelvis or cephalopelvic disproportion, central placenta previa, carcinoma cervix and vaginal obstruction are absolute indications while previous cesarean, dystocia or failure to progress, foetal distress and breech presentation are common indications.
3. Informed written consent is always taken, blood is arranged and antibiotics are given. Epidural or spinal anesthesia are preferred over general anesthesia. Incision is usually transverse (Pfannenstiel's or Cohen's) in the skin and lower segment transverse incision in the uterus. After delivery of the fetus, placenta is delivered by controlled cord traction **(level 1A evidence)**. Oxytocin drip is given. Uterus can be stitched in one or two layers, there is no need to stitch the visceral or parietal peritoneum. Rectus sheath is stitched by delayed absorbable polyglactin (vicryl) or non-absorbable prolene or nylon (in case of vertical incision) while skin can be stitched by subcuticular absorbable stitches, non-absorbable nylon or silk or staplers.
4. The various complications can be intraoperative like extension of uterine incision to involve uterine vessels, bladder and ureter injuries, intestinal injuries, haemorrhage, shock, morbidly adherent placenta, anaesthetic complications like Mendelson's syndrome (aspiration pneumonitis) and injury to the baby. Post operative complications can be postpartum haemorrhage, shock, infection, thromboembolic events, wound infection and dehiscence, intestinal obstruction and remote complications like chronic pelvic pain, mentrual dysfunction, intestinal obstruction and scar rupture in future.
5. Lower segment cesarean section is almost always performed as it is associated with less blood loss, better apposition of uterine wound, less chances of gutter formation and less chances of scar rupture (0.5 to 1.5%) in future pregnancies. All the complications are higher for classical cesarean section which is rarely performed for carcinoma cervix with pregnancy, perimortem cesarean or where lower segment is not approachable. Risk of scar rupture is 4 to 9% in classical cesarean section.
6. Antibiotic prophylaxis using the antibiotic effective against common organisms significantly reduce the risk of endometritis in all types of cesareans. However, repeated courses of antibiotics for prophylaxis are not necessary.
7. Thromboembolism prophylaxis should be used for high risk cases to prevent pulmonary embolism and deep vein thrombosis.
8. Repeat cesarean section increases the risk of placenta previa and morbidly adherent placenta.
9. Placenta previa in patients with previous cesarean scar may be associated with placenta accreta and may cause uncontrollable uterine haemorrhage necessitating cesarean hysterectomy. Such a cesarean should be preformed by an experienced senior obstetrician and adequate blood should be arranged.

# (vi) MISCELLANEOUS OPERATIONS

## VERSIONS

### Definition

*Version is a manipulative procedure in which the presentation of the fetus is altered artificially either substituting one pole of a longitudinal presentation for the other or converting an oblique or transverse lie into a longitudinal presentation.*

### Types of version

Version is of three types namely:

1. **External version** is performed by external abdominal manipulation.
2. **Internal version** is performed by inserting a hand into the uterine cavity and the other hand abdominally.
3. **Bipolar version** is performed by inserting two fingers into uterus through the cervix and the other hand on the abdomen.

Another classification is as follows:

**Cephalic version**

In this type of version, head (cephalic pole) is made to present in the lower pole of the uterus by external manipulations.

**Podalic version**

In this type of version, breech (podalic pole) is made to present usually by internal manipulaitons.

### External cephalic version

### Introduction and Definition

**External cephalic version (ECV) involves turning the fetus externally (abdominally) to bring the cephalic pole into the lower pole of the uterus,** i.e. it is done to convert a breech or transverse lie to a cephalic presentation. ECV has been practised since the time of Hippocrates and has got renewed interest recently. The average success rate of the procedure is about 60%, and it reduces the incidence of breech presentation at delivery from 3–4% to 1–1.5%. The success rate is even higher for transverse lie.

Both the Royal College of Obstetricians and Gynaecologists UK (RCOG) and the American College of Obstetricians and Gynecologists (ACOG, 2016) recommend that all women with an uncomplicated breech presentation near term (at 36 weeks in nulliparous women and at 37 weeks in multiparous woman) should be offered an attempt at version **(level IA evidence)**. It reduces the rate of non-cephalic presentation at the onset of labour and decreases the rate of cesarean delivery.

### Indications of ECV

1. Uncomplicated breech pregnancy at term
2. Transverse lie at term (not in labour)
3. Second fetus of twin pregnancy in transverse lie after the delivery of the first twin.

### Contraindications of ECV

**Absolute Contraindications**

1. Indications for cesarean delivery irrespective of foetal position (e.g. placenta previa, contracted pelvis, etc.)
2. Multiple pregnancy
3. Significant foetal or uterine anomaly
4. Foetal death
5. Declined consent
6. Non reassuring foetal status
7. Hyperextended foetal head

**Relative Contraindications**

1. Past history of two cesarean deliveries Scarred uterus – with history of myomectomy, metroplasty. Single previous cesarean delivery is not a contraindication.
2. Maternal cardiac disease
3. Hypertensive disorders of pregnancy
4. Maternal diabetes
5. Maternal obesity (BMI > 20% of ideal) (Procedure is less likely to succeed)
6. Foetal growth restriction
7. Oligohydramnios
8. Isoimmunization
9. Ruptured membranes
10. Nuchal cord
11. Structural uterine anomaly
12. Early labour
13. Prior abruption

### Timing of ECV

ECV was previously performed at 32–34 weeks, due to small fetus and abundant liquor making the procedure easy. However, the present ACOG recommendation is to perform ECV at 37 weeks. Delaying up to 37 weeks has the following advantages, even though it may be slightly more difficult at this time.

1. Fewer procedures are needed as spontaneous version occurs in many cases.
2. Reversion to breech after the procedure is less likely.
3. If foetal complications develop during version, they can be managed by immediate cesarean delivery, as the fetus will be mature avoiding iatrogenic prematurity.
4. Some of the contraindications to version and indications for cesarean delivery, like foetal growth restriction, may be evident only in the later weeks.

### Pre-requisites

Following pre-requisites should be fulfilled before trying ECV.

1. There should be no contraindication to vaginal delivery
2. Absence of foetal anomaly that may interfere with vaginal delivery
3. Gestational age ≥ 37 weeks
4. Estimated foetal weight < 3.5 kg
5. Complete breech presentation or transverse lie
6. No hyperextension of foetal head
7. Facilities for continuous foetal heart rate monitoring should be available
8. Skilled obstetrician and pediatrician should be available
9. Facility for immediate cesarean delivery should be present in the center

## Pre-procedure Preparation

1. Informed consent should be taken after explaining the nature, risks, success and failure of the procedure. Consent for emergency cesarean delivery should also be taken in case of foetal distress due to ECV.
2. Patient should be fasting for 6 hours.
3. Intravenous access is obtained.
4. Bladder should be empty and the woman relaxed and comfortable.
5. The procedure should be performed by an experienced operator, making sure operation theatre is available.
6. Ultrasound should be done to assess foetal position, confirm breech presentation or transverse lie, amount of liquor, placental localization and rule out any gross congenital foetal malformations.
7. Cardiotocography (CTG) monitoring should preferably be available during the procedure.
8. A reactive non-stress test (NST) should be obtained prior to the procedure.
9. Foetal heart should be auscultated every 2 minutes, if continuous CTG is not available and the procedure interrupted if bradycardia occurs or if the woman experiences discomfort. Maternal pulse and blood pressure should be taken every 5 minutes, especially if a tocolytic is being used.
10. After successful version, the position of the fetus should be maintained manually for a few minutes.
11. With multiple failed attempts, the procedure should be abandoned in that sitting.
12. If the first attempt has been without a tocolytic, the procedure may be repeated under tocolysis a week later. ECV should not be done more than 4 times or a maximum of 10 mins overall.
13. Anti-D immunoglobulin (300 mg) is given to all Rh-negative women.

## Procedure

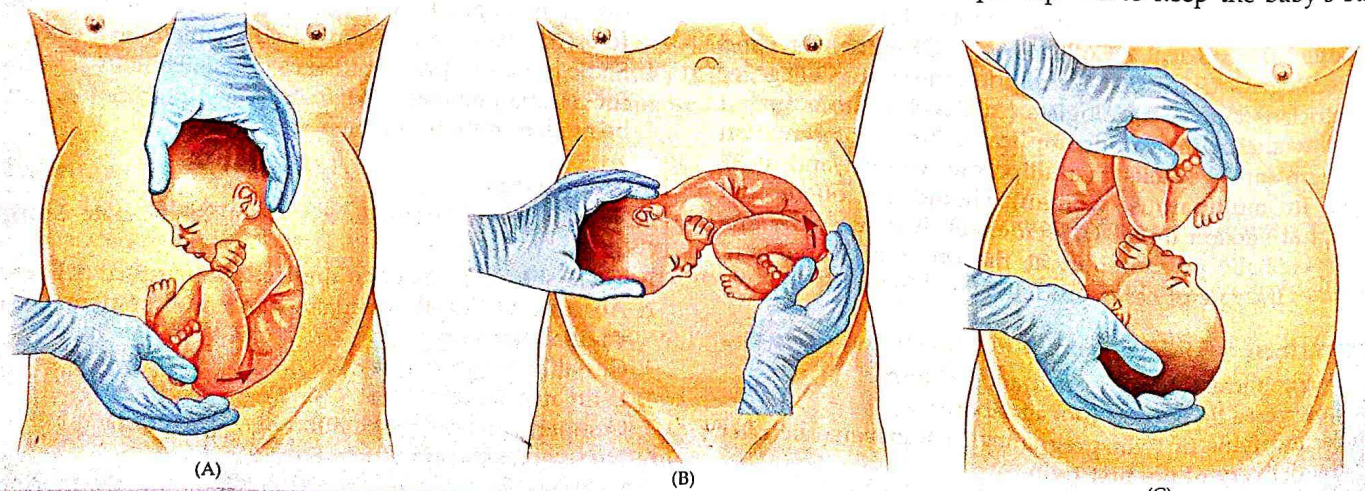
**In breech presentation.** The procedure is performed at or after 37 weeks in the labour room. Drinking plenty of fluids before attempting ECV may optimize liquor volume making ECV easier. Six hour fasting is done. Intravenous drip is started. There is insufficient data about use of analgesia including epidural or spinal before ECV and **it should not be done under general anesthesia**. Vibroacoustic stimulation of fetus and amnio infusion before ECV have been tried but not routinely recommended. Tocolytic agents (Terbutaline 250 µg subcutaneously or isoxsuprine 50–100 mg intravenously) can be given for uterine relaxation. Ultrasonographic examination is performed to confirm breech (or transverse lie), amniotic fluid volume, placental localization and to rule out foetal anomalies. Cardiotocogram should be used for foetal heart reactivity. Injection anti D is given to Rh-negative women.

## Steps

1. Each step of the procedure is carefully explained to the woman and she is asked to keep her abdominal muscles fully relaxed and inform the operator in case of any discomfort. Position of the foetal back is confirmed.
2. The woman is placed on a firm examination table with the foetal back towards the operator (if a backward flip is to be attempted) or opposite to the operator (if forward somersault is to be done). She should be tilted on her side towards the operator with a wedge/cushion under the back.
3. Talcum powder is spread on the abdomen, to allow the skin to slide under the operator's hands and prevent friction. Ultrasound jelly can also be used in place of powder.

**Step 1** (Fig. 42.43 A)

The breech is disimpacted out of pelvis gradually using continous abdominal pressure by right hand and is pushed into one iliac fossa which was occupied by foetal back. It is preferable to turn the foetal head first with left hand while the right hand pushes the breech. The two methods to rotate the breech may be a backward flip or a forward somersault. The basic principle is to keep the baby's back



(A) (B) (C)

**Fig. 42.43:** Steps of external cephalic version—(Breech—LSA): (A) Mobilization of the buttocks to the iliac fossa with the back upwards using both hands; (B) Rotation of the trunk holding the poles and maintaining flexion of the trunk; (C) Change of hands to prevent crossing after the lie becomes transverse. The cephalic pole is being brought to the lower pole of the uterus and the lie becomes longitudinal

flexed and follow the shortest path to a cephalic position. Slow and steady pressure should be used, rather than repeated jerky movements. Continuous pressure on the uterus should be limited to 5 minutes.

**Step 2** (Figs. 42.43 B and C)

In **forward somersault (roll) method**, each hand grasps one of the foetal poles and the buttocks are elevated from maternal pelvis and displaced to one iliac fossa. The buttocks are then slowly guided towards the fundus while the head is directed towards the pelvis.

**Alternate method**

**Backward flip.** If the above method fails, backward flip is attempted.

**Post procedure care.** Non-stress test is performed for foetal heart. The women should come for regular follow-up in clinic and report for any bleeding or leaking or uterine contractions.

## Complications

1. Risks to the mother include discomfort, need for emergency cesarean delivery and possible negative psychological effects in the event of failure.
2. The foetal risks are in the form of non-reassuring foetal heart rate patterns (non-reactive tracings, transient bradycardia) which are the most common and may persist for 20–40 minutes.
3. Placental abruption
4. Fetomaternal haemorrhage
5. Isoimmunization
6. Preterm labour
7. Prelabor preterm rupture of membranes
8. Uterine rupture
9. Amniotic fluid embolism
10. Brachial plexus injury
11. Rarely, it may be a cause of perinatal mortality. Foetal loss rate used to be quoted 1% in old studies but is much less in recent studies.

Small procedure related risks must be weighted with the risk associated with persistent breech presentation, including cord prolapse. Cesarean delivery can also result in trauma to the baby and elective cesarean may carry the risks of transient tachypnea of the newborn and pulmonary hypertension.

However, ECV has been reported to be cost effective from studies in the USA and UK.

## Factors Associated with Successful Version

1. Multiparity
2. Non-engaged presenting part
3. Flexed breech (extended legs are a hindrance to version)
4. Adequate liquor
5. Non-anterior placenta

## Factors Associated with Failure of ECV

1. Nulliparity
2. Anterior, lateral or cornual placentation
3. Decreased or increased liquor
4. Descent of breech into pelvis
5. Maternal obesity
6. Posteriorly located foetal spine
7. Frank breech
8. Short umbilical cord

## External Version in Transverse Lie

The steps are similar as in breech, except that the first step is not required. Hence, it is easier. The complications are also similar. Ultrasound must be done before version to exclude placenta previa and uterine malformations as transverse lie is more common in them.

# EXTERNAL PODALIC VERSION

The external podalic version can be tried in case of failure of the external cephalic version especially in the transverse lie of the second twin. It is very rarely performed.

# INTERNAL PODALIC VERSION

Internal podalic version consists of turning the lie of the fetus by inserting a hand into the uterine cavity and seizing one or both feet and bringing them down and transabdominally pushing the upper portion of the foetal body upwards with the other hand. The operation is followed by breech extraction.

## Indications

1. Delivery of the second twin is the only indication of this procedure in modern obstetrics with live fetus.
2. **Rare indications.** Cesarean facilities are not available with the following possibilities.
   (*i*) Transverse lie with cervix being fully dilated
   (*ii*) Cord prolapse with transverse lie
   (*iii*) Transverse lie with very preterm, non-salvageable or grossly malformed baby incompatible with life.

## Contraindications

1. Big baby
2. Contracted pelvis
3. Obstructed labour with impending rupture
4. Scanty liquor
5. Septate uterus
6. Previous cesarean delivery

## Timing and Pre-requisites

1. Cervix must be fully dilated
2. Membranes absent but recently ruptured, ensuring adequate liquor
3. Baby of average size
4. No contraindications to vaginal delivery

## Pre-procedure Preparation

1. Informed consent is mandatory, explaining the nature, risks and success of the procedure and consent for emergency cesarean delivery.

2. Bladder should be empty and the woman relaxed and comfortable.
3. Good uterine relaxation is necessary.
4. The procedure should be performed by an experienced operator.
5. Pediatrician should be available.
6. Per-abdominal examination should be done to assess foetal position, amount of liquor and foetal heart sounds.
7. Per-vaginal examination should confirm that cervix is fully dilated and pelvis is adequate.
8. Membranes, if present, should be ruptured.

## Procedure

1. It is performed under deep anesthesia, preferably general anesthesia.
2. Place the woman in lithotomy position and continuously monitor foetal heart rate.
3. Under all aseptic precautions, one hand is introduced into the uterine cavity in a cone-shaped manner and taken along the thigh of the baby.
4. If the lower limbs are on the mother's right side, the left hand should be employed and if on the mother's left it is preferable to use the right hand for convenience. However, many obstetricians find it easier to use their right hand irrespective of the position of podalic pole.
5. The foot is identified by the prominence of heel and one foot or both feet are brought down gently.
6. The abdominal hand of operator or hand of assistant aids in pushing the head up towards the fundus to facilitate rotation.
7. The baby is delivered by breech extraction.

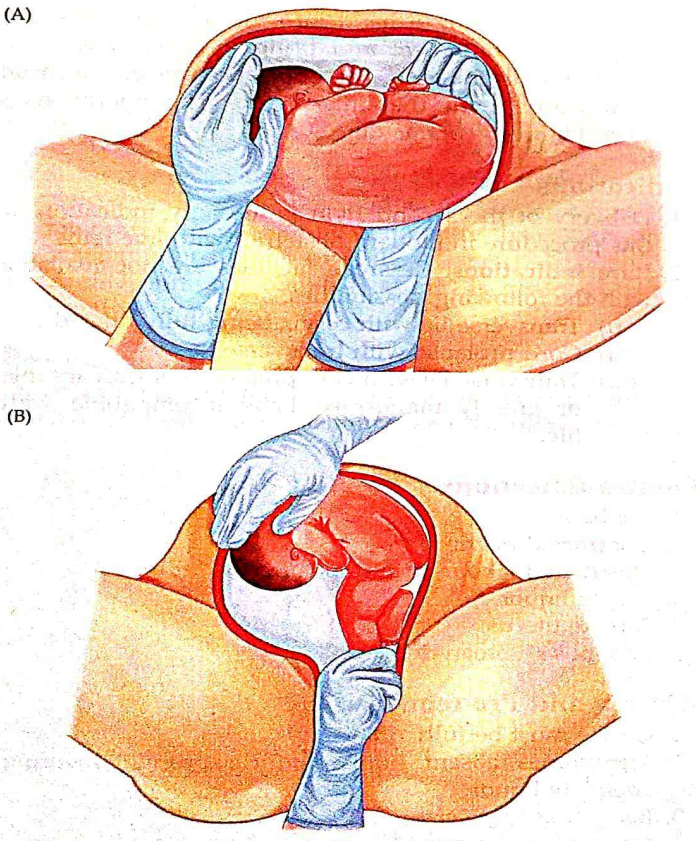



**Fig. 42.44:** Principle steps of internal podalic version—(A) To introduce the right hand to grasp the upper leg in dorso-inferior position with the head lying in the right iliac fossa; (B) Internal podalic version in dorso superior position to give traction on the leg gripping in a cigarette holding fashion, the other hand pushes up the head externally

## Post-procedure Care

1. Uterine rupture should be ruled out.
2. Lacerations to the genital tract must be looked for and managed.
3. Oxytocics should be given to avoid postpartum haemorrhage.

### Maternal Risks

1. Abruptio placentae
2. Tears of cervix, vagina and perineum
3. Uterine rupture
4. Postpartum haemorrhage
5. Increased morbidity

### Foetal Risks

Perinatal mortality can be there mainly due to hazards of breech delivery.

1. Hypoxia
2. Intracranial injury
3. Stillbirth
4. Cord prolapse

## Bipolar version

Bipolar version was first performed by Braxton-Hicks. It is not routinely done and is almost obsolete in modern obstetrics.

### Indications

It was performed in the past for minor degree placenta previa with dead, malformed or very preterm fetus (nolsalvagable). It can be a life-saving procedure in smaller places where facilities for cesarean delivery are not available and patient can not be shifted to a bigger hospital. The cervix should be at least 2 cm dilated for manipulations in which head is pushed to one iliac fossa and one leg is grasped at ankle and is brought down compressing the placenta to stop bleeding. The other hand kept on abdomen helps in the procedure.

# OTHER MISCELLANEOUS OPERATIONS

## Culdocentesis (needling) (Fig. 42.45)

Culdocentesis is done to see blood or pus in pouch of Douglas (POD) in suspected cases of ectopic pregnancy or pelvic abscess. The procedure is explained to the patient and a written, informed consent of the patient is taken. Put the patients in lithotomy position after they have passed urine. The parts are cleaned and draped. Cervix is exposed

by either Sim's posterior vaginal wall speculum or Cusco's bivalve speculum. Posterior lip of the cervix is caught by a tenaculum or vulsellum. 16–18 gauge spinal needle with 20 mL syringe is introduced in pouch of Douglas just below the posterior lip of the cervix and aspiration is done. If pus is coming out, it is pelvis abscess. Send the pus for culture. If it is non-clotting blood, ruptured ectopic pregnancy is suspected. If aspirated blood which clots, a vein or artery may have been injured. Remove the needle and reinsert and aspirate.

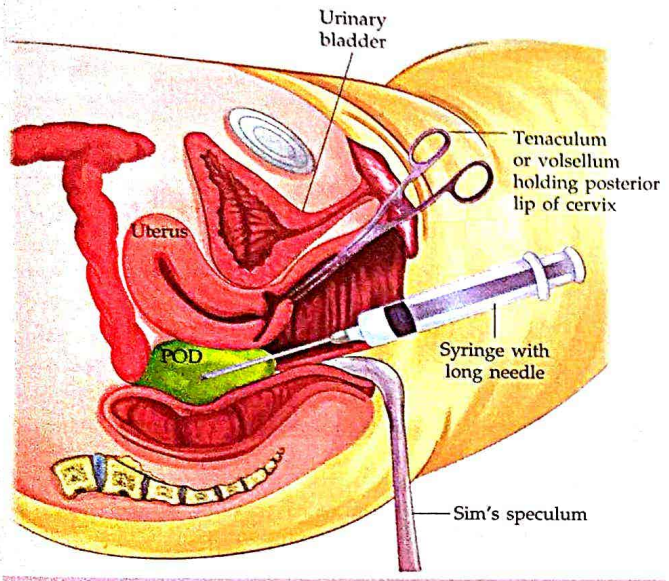



**Fig. 42.45:** Culdocentesis

## Colpotomy (Fig. 42.46)

Colpotomy is cutting open of pouch of Douglas in case of pelvic abscess which can be a complication of abortions, especially illegal and septic abortions and puerperal sepsis.

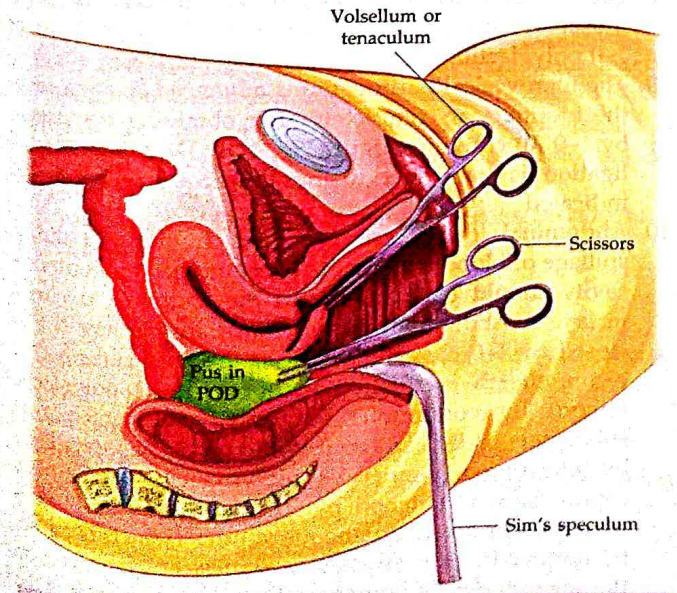



**Fig. 42.46:** Colpotomy

The procedure is explained to patient and consent is taken. The patient is taken to the operation theatre. After passing urine, she is put in lithotomy position. Sedation/ Regional anesthesia is given. Perineum is cleaned with antiseptic solution and drapped. The cervix is exposed by Sim's speculum and anterior vaginal wall retractor. Posterior lip of the cervix is caught with a volsellum or tenaculum. Vagina below the cervix is grasped between two Allis forceps. The middle part of vagina is cut by a pair of scissors. Pus will come out. Right index finger is put inside to break all loculi of pus. A drain is put so that the drainage of pus continues. On second or third day, when pus discharge stops, the drain is removed. The pus is to be sent for culture and sensitivity. Broad spectrum antibiotics are given.

If the abscess is situated anterior or lateral to the uterus, drainage may be performed by ultrasound guided placement of catheter drain. When the abscess cannot be reached by any of the above methods, an open laparotomy is carried out. Pus is removed, pelvic cavity is irrigated and a drain is kept for a day or two. Broad spectrum antibiotics are given.

## Cervical tear (Fig. 42.47)

See Chapter 26 for details.

## Hysterotomy

It is a procedure performed between 13–28 weeks of gestation (24 weeks in western countries) in which evacuation of uterus is performed by incising the anterior wall of uterus. Simultaneous sterilization can be performed. It is usually done by abdominal route but can rarely be performed vaginally. By law, hysterotomy is performed only up to 20 weeks (legal limit) but can rarely be done for hydatidiform mole even later.

### Indications

With effective methods of medical termination of pregnancy available, hysterotomy is rarely required nowadays.

1. Medical termination of pregnancy (induced abortion) in the second trimester where other methods of termination fail or where other methods are contraindicated. This is to be done up to 20 weeks only (legal limit of MTP).
2. In selected cases of hydatidiform mole when suction evacuation cannot be used for some reason and the uterus has to be conserved.
3. Painless vaginal bleeding in second trimester with cervix remaining unfavourable. The bleeding is mostly due to low-lying placenta.
4. In preterm, prolonged rupture of membranes with signs of chorioamnionitis setting in without labour.

### Preparation

Injection tetanus toxoid (TT) 0.5 mL intramuscularly is given before operation (if not already given) and informed written consent is taken from the patient and her husband or one relative. Pre-operative investigations and pre-anaesthetic check up are done. Anesthesia can be general,

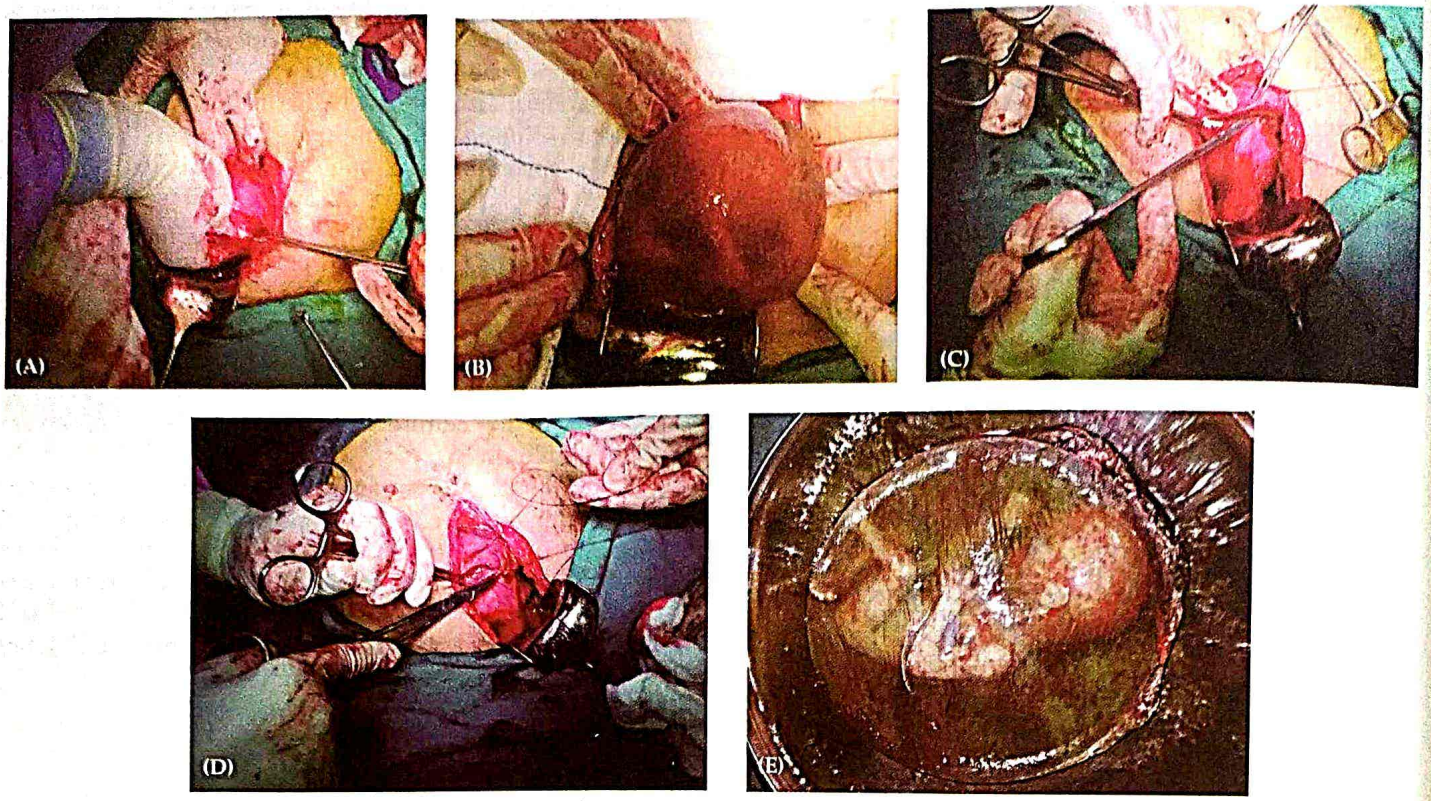



**Fig. 42.47:** Procedure of abdominal hysterectomy performed by author: Actual photographs
(A) Transverse incision (vertical incision can also be given) on the lower part of uterus after opening the uterovesical pouch and retracting the bladder; (B) Amniotic sac is being removed; (C) Hysterotomy wound being stitched in layers apposing the muscles; (D) Closure of the peritoneum or uterovesical pouch; (E) Fetus-en-caul extracted during hysterotomy

spinal or epidural. Intravenous broad spectrum antibiotics are given. One to two units of blood are arranged.

### Steps of Abdominal Hysterotomy

Technique is similar to the cesarean section except that the incisions in the abdomen and uterine wall in hysterotomy are smaller.

1. Abdomen is cleaned with savlon and povidine iodine solution. Abdomen is draped.
2. Abdomen is opened by a Pfannensteil incision or low vertical infraumbilical incision. Peritoneal cavity is entered.
3. **The peritoneal cavity must be completely and meticulously packed to avoid spillage of uterine contents to reduce the risk of scar endometriosis.** Uterus may or may not be delivered out of abdominal cavity.
4. Lower fold of peritoneum from uterovesical pouch is incised transversely and the bladder pushed down. Doyen's retractor is inserted.
5. The uterine incision may be:
   (a) Transverse incision as low as possible (Fig. 42.47 A)
   (b) Low vertical in the region of the lower uterine segment. The incision should be about 5 cm long and deep.
6. The incision is deepened till the amniotic sac bulges out through it (Fig. 42.47 B). The amniotic sac is separated from the wall using right index finger.
7. Oxytocin drip with 10 units is started. Uterus is compressed and the amniotic sac is coaxed out of the uterine incision avoiding its rupture. Thus, fetus is removed En-caul (Fig. 42.47 B). Placenta is also removed in the similar fashion. Uterine cavity is cleaned with a gauze covered finger. Oxytocin drip is continued.
8. Uterine incision is closed in two layers, the deep muscular and superficial seromuscular layer with or 1–0 polyglactin (vicryl) sutures (Fig. 42.47 C).
9. The uterovesical peritoneum is advanced so as to cover the uterine scar completely after obtaining hemostasis by 1–0 (0) chromic catgut (Fig. 42.47 D). Bilateral tubal ligation is performed using Pomeroy's technique, if indicated.
10. Abdominal packs are removed carefully avoiding spillage of blood and products of conception. Peritoneal cavity should be cleaned and the abdomen is closed in layers. Abdominal peritoneum may or may not be closed by 1–0 chromic catgut continuous sutures. The rectus sheath is stitched with number 1 polyglactin (vicryl) by continuous sutures. If the amount of subcutaneous fat is more it is stitched in separate layer by interrupted number 1–0 vicryl or chromic or plain catgut. Skin is stitched by interrupted nylon sutures or staples or by subcutaneous sutures (which need not be removed). Skin stitches or staples are removed on the seventh day of operation. Post operative care is the same as for cesarean delivery.

## Complications

### Early Complications

1. Haemorrhage
2. Injuries to other viscera like bladder, intestine etc.
3. Anaesthetic complications
4. Peritonitis
5. Intestinal obstruction

### Delayed Complications

1. Menstrual problems—menorrhagia or irregular periods
2. **Scar endometriosis (1%). It is the main problem** which can be reduced to some extent by avoidance of contamination of uterine blood into abdominal incision.)
3. Scar rupture and dehiscence in future pregnancies is like classical cesarean scar (can be avoided by ligation at the time of hysterotomy).

## Key Points

1. External cephalic version (ECV) is turning the fetus externally (abdominally) into cephalic presentation to convert a breech presentation or transverse lie to cephalic presentation and has got renewed interest with success rate of about 60%. It has been recommended by RCOG (London) and ACOG for all uncomplicated breech presentations near term. It is contraindicated in cases where cesarean delivery is indicated (placenta previa, contracted pelvis) or there is significant uterine or foetal anomaly. The procedure involves first lifting the breech out of pelvis and converting breech into transverse lie followed by further rotation into cephalic presentation. Complications include foetal distress, preterm labour, antepartum haemorrhage and premature rupture of membranes and isoimmunization.
2. External cephalic version should be offered to all women with an uncomplicated breech presentation at term **(level IA evidence)**. ECV has been demonstrated to be associated with significant reduction in the risk of cesarean section rate **(level IA evidence)**.
3. Internal podalic version is turning the lie of the fetus to breech by intrauterine manipulations and its only valid indication in present day obstetrics is for the second twin in transverse lie and is usually combined with breech extraction in such cases.
4. Hysterotomy is a miniature version of cesarean section before 20 weeks (legal limit of MTP) of pregnancy in which products of conception are extracted out of uterus by cutting through abdominal wall and uterine wall. It is a method of medical termination of pregnancy where other methods have failed, it is rarely required in modern obstetrics due to availability of highly effective prostaglandins for second trimester termination of pregnancy. The steps are like cesarean section. Scar endometriosis is a particular problem of hysterotomy.

## Suggested Reading

1. Hofmeyr G J, West HM and Kulier R. External Cephalic Version for Breech Presentation at Term. Cochrane Database Systematic Reviews 2015: 4 : CD000083.
2. Royal College of Obstetricians and Gynaecologists. The Management of Breech Presentation, Guidelines no. 20 B. London: RCOG Press, March 2017.
3. Royal College of Obstetricians and Gynecologists: External Cephalic version and Reducing the incidence of Term Breech Presentation. Green Top Guideline no. 20A, London, RCOG Press 2017.
4. American College of Obstetricians and Gynecologists. External Cephalic Version. Practice Bulletin No. 221, May 2020.
5. American College of Obstetricians and Gynecologists. Mode of Term Delivery in Singleton Breech Delivery. Committee Opinion no 340, July 2006, reaffirmed 2016.
6. Royal College of Obstetricians and Gynecologists. Operative Vaginal Delivery, Green Top Guideline No. 26, RCOG, London, February 2011.

## Examination Questions

A. Define external cephalic version. Enumerate its indications and contraindications and describe the steps.
B. Describe the steps of external cephalic version. Enumerate its complications and factors associated with its failure.
C. Write short notes on:
   1. External cephalic version (Many universities)
   2. Role of external cephalic version in breech presentation
   3. Role of ECV in transverse lie
   4. Complications and failure of ECV
   5. Internal podalic version
   6. Bipolar version
   7. Colpotomy
   8. Culdocentesis
   9. Hysterotomy

Section 11
CONTRACEPTION

CHAPTER 43

# Control of Conception (Contraception)

## INTRODUCTION

### Demography

**Demography deals with the study of human population and the vital events like crude death rate, birth rate and other parameters related to it.** The world population became staggering 7.66 billion on Ist January 2019. It was 6 billion in 1999. A total of 70% people live in developing countries and the population is increasing at a rate of 150 million per year. World's population is expected to reach 9 billion by 2050.

India with only 2.4% global landmass is the second most populous country, after China, with 1364.5 million people in 2019 which is 17% of the world's population. Infact, every sixth person in the world is Indian. The population of India rises by 16 million per year (equivalent to the total population of Australia). With China controlling its population successfully by adopting one child norm, India will surpass it in 2050 or probably earlier with estimated 1628 million population.

Such a large population expansion adversely affects the national economy and the health of mother and child as high parity is associated with higher maternal and perinatal mortality and morbidity. Controlling the population growth needs great emphasis. Contraception is the need of the hour. Around 2/5th of all pregnancies are unintended. By fulfilling unmet needs of contraception, we can avoid 55 million unwanted pregnancies, 22 million abortions, 90,000 maternal deaths. In India 94.5% women have knowledge of contraceptives but only 50.2% practice it with gap of 44.3% (SRS 2014).

### Aims and objectives of control of population and family planning

1. To control population growth for better standard of living.
2. To bring down the birth rate to sustainable level by increasing contraception usage. Out of about 168 million eligible couples in the reproductive age group of 15–45 years, only about 44% practise effective methods of contraception. We need to increase the unmet needs of contraception.
3. To prevent too early, too frequent and too many pregnancies.
4. To prevent unsafe abortions by use of contraception.
5. To reduce high maternal and perinatal mortality and morbidity.
6. Society upliftment so that food, clean water, clothing, housing, education and proper healthcare can be provided to all.

### Family welfare programme

India became the first country in the world to launch a government-sponsored Family Planning Programme in 1952 within the existing infrastructure of the health services. It was later integrated with Maternal and Child Health (MCH) Programme.

Since 1996–97, a major policy reform has been introduced by the Department of Family Welfare. The entire range of family planning, maternity and child health services have been brought under the **Reproductive and Child Health (RCH) Programme** (see Chapter 45 for details). These have been further strengthened by the **National Population Policy (NPP) (2000)** to address the unmet needs for contraception and the **National Rural Health Mission (NRHM) (April, 2005)** and **National Health Mission** (combination of National Rural and Urban Health Mission) (see Chapter 45 for details).

### Family Planning 2020

WHO's Department of Reproductive Health and Research launched Family Planning 2020 in 2012 to support the right of women and girls to decide freely and for themselves whether when and how many children they want. It

aims to expand access to family planning information, services and supplies to an additional 120 million women and girls in 69 of world's poorest countries by 2020. It gives more importance to spacing methods especially IUDs. India Family Planning 2020 goal is to increase the modern contraceptive usage from 53.1% to 54.3% and to satisfy the demand of modern contraceptives by 74% by 2020.

*The Government of India takes full responsibility of providing funds, specifying guidelines and proposing the organizational structure for our family planning programme, while the state governments are responsible for actual implementation of the programme through their health system.*

## NATIONAL FAMILY PLANNING

The government has drawn out comprehensive National and State roadmaps and also district action plans adopting a decentralized planning approach focusing on operationalization of facilities and delivery of services. Financial incentive are given as per Table 43.1A.

**Table 43.1A: Financial Incentives for Accepting Sterlisation Procedure**

| States | | Acceptor | ASHA/ Health worker | Others | Total |
|---|---|---|---|---|---|
| 11 High focus states (UP, BH, MP, RJ, CG, JH, OD, UK, AS, HR, GJ) | VAS. | 2000 | 300 | 400 | 2700 |
| | TUB. | 1400 | 200 | 400 | 2000 |
| | TUB. (PPS) | 2200 | 300 | 500 | 3000 |
| Mission Parivar Vikas Districts | VAS. | 3000 | 400 | 600 | 4000 |
| | TUB. | 2000 | 300 | 500 | 2800 |
| | TUB. (PPS) | 3000 | 400 | 600 | 4000 |
| Other High focus states (NE states, J&K, HP) | VAS. | 1100 | 200 | 200 | 1500 |
| | TUB. | 600 | 150 | 250 | 1000 |
| Non High focus states | VAS. | 1100 | 200 | 200 | 1500 |
| | TUB. (BPL + SC/ST only) | 600 | 150 | 250 | 1000 |
| | TUB. (APL) | 250 | 150 | 250 | 650 |

UP: Uttar Pradesh; BH: Bihar; MP: Madhya Pradesh; RJ: Rajasthan; CG: Chhitisgarh; JH: Jharkhand; OD: Odissa, UK: Uttrakhand; AS: Assam; HR: Haryana

### Key Highlights of the Programme

#### A. India's Strategy

1. Increased focus on spacing services without disturbing the sterilizaton pie.
2. Voluntary adoption of Family Planning based on felt need of the community.
3. Focus on male participation.
4. Right based approach to Family Planning.
5. Expanding contraceptive choices.

#### B. Contraceptive Basket of Choice Under National Family Planning Programme

1. Temporary methods
   (a) Condoms (Nirodh)
   (b) Oral contraceptive pill
      (i) COC's (Mala-N)
      (ii) Centchroman (Chhaya)
      (iii) Emergency Contraceptive Pills (Ezy Pill)
      (iv) Progesterone only Pills-under pilot
   (c) IUCD - CuT 380A, CuT375
   (d) Injectable Medroxy Progesterone Acetate (ANTARA)
2. Permanent methods
   (a) Male sterilization (Conventiional, Vasectomy/NSV)
   (b) Female sterilization (Minilap/Laparoscopy)

#### C. Augmenting the demand throgh ASHA schemes for Family Planning

(i) Home delivery of contraceptive
(ii) Ensuring spacing at birth.
(iii) Pregnancy test kits – for early management of pregnancy.

#### D. Promoting Quality Sterilization Services Sterilization Compensation Scheme

The compensation package has been enhanced in 2014 for 11 high focus high TFR states.

(i) Higher package for Post-Partum sterilization and Male sterilization
(ii) Higher package for (Mission Parivar Vikas) district.

##### Mobile Terms Dedicated Family Planning Services

The scheme has been introduced in high focus states in 2014–2015 to provide sterilization services in areas where there is the dearth of service providers.

##### Scheme for Ensuring Drop Back Services to Sterilization Clients

It was launched in 2015 and can be availed as per demand from the beneficiaries.

##### National Family Plannng Indemnity Scheme

The scheme was revised in 2013 and is now being operated by the state governments directly with NHM finding as per table 43.1B.

1. The clients are indemnified in the unlikely events of death, complications and failures following sterilization.
2. The providers/accredited intuitions are indemnified against itigations.

#### E. Promoting Quality IUCD Services

(i) Interval IUCD – Can be provided in all public health facilities by a trained provider in OPD
(ii) PPIUCD – Inserted within 48 hour after delivery.

| Table 43.1B: Amount of Compensation under Indemnily Scheme | | | |
|---|---|---|---|
| | Claims arising out of Sterilisation operation | Amount (₹) | Additional as per Hon'ble SC Directives |
| A | Death at hospital/within seven days of discharge | 2,00,000 | 2,00,000 |
| B | Death following Sterilisation (8th–30th day from discharge) | 50,000 | 50,000 |
| C | Expenses for treatment of Medical Complications | 25,000 | 25,000 |
| D | Failure of Sterilisation | 30,000 | 30,000 |
| E | Doctors/facilities covered for litigations upto 4 cases per year including defense cost | 2,00,000 (per case) | |

(iii) PAIUCD – Inserted within 12 days of abortion in PHC and other health facilities.

**Extended PLPPSA Scheme – Programme Linked Payment Plan to Service Providers, Acceptors and ASHA's**

(a) Trained/skilled empanelled providers inserting PPIUCD/PAIUCD is given ₹ 150 per insertion.
(b) ASHA accompanying the client is given ₹ 150
(c) Beneficiary is given ₹ 300 per insertion.

Provider's base for IUCD services is being increased by utilizing the army of doctors qualified in ISM (Unani, Siddha and Homeopathy) after giving them structured training for provision for IUCD.

**F. Generating demand and awareness for Family Planning Services**

(i) Improved counseling through RMNCH counselors.
(ii) Celebrating of World Population Day and fortnight (11th July – 24th July).
(iii) Celebrating of Vasectomy fortnight (21st November to 4th December)

**G. FP-LMIS**

Family Planning Logistic management information system has been incorporated in National Family Planning Programme to ensure the right commodities, in the right quantities reach right place at the right time.

**H. Public Private Partnership**

It is assumed that effective collaboration with the private sector in the form of public private partnership would address the unmet need in the Family Planning significantly.

# CONTRACEPTION

**The term contraception includes temporary or permanent measures designed to prevent pregnancy from unprotected sexual intercourse.** Fertility control includes both fertility inhibition (contraception) and fertility stimulation (treatment for infertility).

*Ideal contraceptive method should be easily available, easy to use, cheap, widely acceptable, safe and highly effective. It should require minimal motivation, maintenance and supervision. Ideal contraceptive is yet to be discovered.*

## Measurement of efficacy of a contraceptive

The efficacy of any method of contraception can be calculated in a variety of ways. Most studies use efficacy as the number of pregnancies experienced during hundred woman years (HWY) of use of the method (a woman year is defined as 12 cycles) and are standardized by 1-year, 2-year, 5-year cut-off points.

The failure rate of any contraceptive is calculated according to the following **Pearl index** formula:

$$\frac{\text{Number of accidental pregnancies} \times \text{1200 (number of months in 100 years)}}{\text{Number of patients observed} \times \text{Months of use (total months of exposure)}}$$

**Example:** If 100 couples used a contraception method for a period of 3 years (36 months) with 24 pregnancies, the pregnancy rate is calculated to be:

$$24 \times 1200/100 \times 36 = 8$$

Efficiency is considered high if the pregnancy rate is below 10, but it is said to be low if pregnancy rate is more than 20. The commonly used contraceptive methods and their efficacy is given in Table 43.1. As shown, typical failure rate is higher than ideal usage as it includes practical problems which are very common in real world e.g. forgetting a pill, use of antibiotics with pill, etc., which decrease its efficacy. The difference is less for methods less dependent on consumers like intrauterine devices. Life time amalysis: Number of pregnancies per 100 women in 12 months of use: not much used.

## WHO eligibility criteria for contraceptives (WHO 2017)

The safety of any method of contraception is of paramount importance to both the user and the prescriber. The World Health Organization (WHO, 2017) has produced a classification system to aid selection of the most appropriate method. The suitability of different contraceptive methods is categorized in the presence of a specific illness or condition by weighing the health risks against the benefits of using a particular contraceptive method when any of these conditions are present. It was updated in 2017. WHO has developed a wheel showing various indications and contraindication of various methods in different conditions for ease of use. There are four categories.

**Category 1:** A condition for which there is no restriction for the use of the contraceptive method. **These are indications for using the method. Use method freely in any circumstance.**

**Category 2:** A condition for which the advantages of

**Table 43.1C: Contraceptive Failure Rates of Various Methods in the First Year of Use (WHO, 2018)**

| Methods | Pregnancy Rate (%) | |
|---|---|---|
| | Lowest Expected | Typical use |
| 1. None (no method) | 85.0 | 85.0 |
| 2. Periodic abstinence (rhythm method) | | 25 |
| (i) Calendar | 9 | 25 |
| (ii) Ovulation method | 3 | |
| (iii) Temperature | 2 | |
| 3. Withdrawal (coitus interruptus) | 4 | 27 |
| 4. Lactational amenorrhoea | 0.9 | 2 |
| 5. Barrier methods | | |
| (i) Cervical cap | | |
| Parous women | 26 | 32 |
| Nulliparous women | 9 | 16 |
| (ii) (a) Diaphragm | 20 | |
| (b) Diaphragm plus spermicide | 6 | 16 |
| (iii) Sponge | | |
| Parous women | 20 | 32 |
| Nulliparous women | 9 | 16 |
| (iv) Spermicides | 18 | 29 |
| (v) Condom male | 2 | 15 |
| (vi) Condom female | 5 | 21.0 |
| 6. Inrauterine devices | | |
| Levonorgestrel intrauterine device | 0.1 | 1 |
| Cu T 380A | 0.6 | 0.8 |
| 7. Hormonal contraception | | |
| (i) Combination pill | 0.16 | 9 |
| (ii) Progestin-only pill (mini pill) | 1.2 | 9 |
| (iii) Patch | 0.3 | 8 |
| (iv) Injectable progestin | 0.3 | 3 |
| (v) Nuva ring (vaginal ring) | 0.3 | 8 |
| (vi) Implants | 0.38 | 1 |
| 8. Emergency contraception | > 75% reduction | |
| 9. Female sterilization | 0.5 | 0.5 |
| 10. Male sterilization | 0.10 | 0.15 |

using the method generally outweigh the theoretical or proven risks. **These are indications for using the method with some caution. Generally use method.**

**Category 3:** A condition for which the theoretical or proven risks usually outweigh the advantages of using the method. **These are relative contraindications for the method. Avoid use of method** unless other more appropriate methods are not available.

**Category 4:** A condition which represents an unacceptable health risk if the contraceptive method is used. **These are absolute contraindications for the method. Never use the method.**

## METHODS OF CONTRACEPTION

The methods of contraception can be temporary or permanent.

### I. Temporary contraceptive methods (spacing methods)

1. Natural methods
2. Barrier methods
3. Intrauterine contraceptive devices
4. Hormonal contraception
   - (*i*) Oral contraceptives
   - (*ii*) Injectable contraceptives
   - (*iii*) Implants
   - (*iv*) Vaginal ring
   - (*v*) Transdermal patches
   - (*vi*) Male contraceptives

### II. Permanent contraceptive methods (terminal methods)

1. Female sterilization (tubal occlusion or tubectomy)
2. Male sterilization (vasectomy)

## TEMPORARY METHODS

Temporary methods are reversible methods which are commonly used to postpone pregnancy or to space births, but are also frequently used by the couples with completed family.

All family planning methods that attempt to identify fertile time in each cycle and then modify sexual behaviour are called Fertility Awareness Based Methods (FAB). When fertility awareness methods involve sexual abstinence during fertile period, they are called Natural Family Planning Methods (NFP). They are as follows:

1. Rhythm method /calender method
2. Basal body temperature method
3. Cervical mucus method / Billing method
4. Sympto-thermal method
5. Standard days method using cycle beads
6. Two day method
7. Ovulation detection
8. Coitus interruptus
9. Lactation amenorrhea method (LAM)
10. Abstinence

Important methods are discussed below.

### 1. Natural Methods

#### (i) Rhythm (calendar) method

Rhythm or calendar method **is approved by the Roman Catholic Church.** It relies upon the fact that there are only

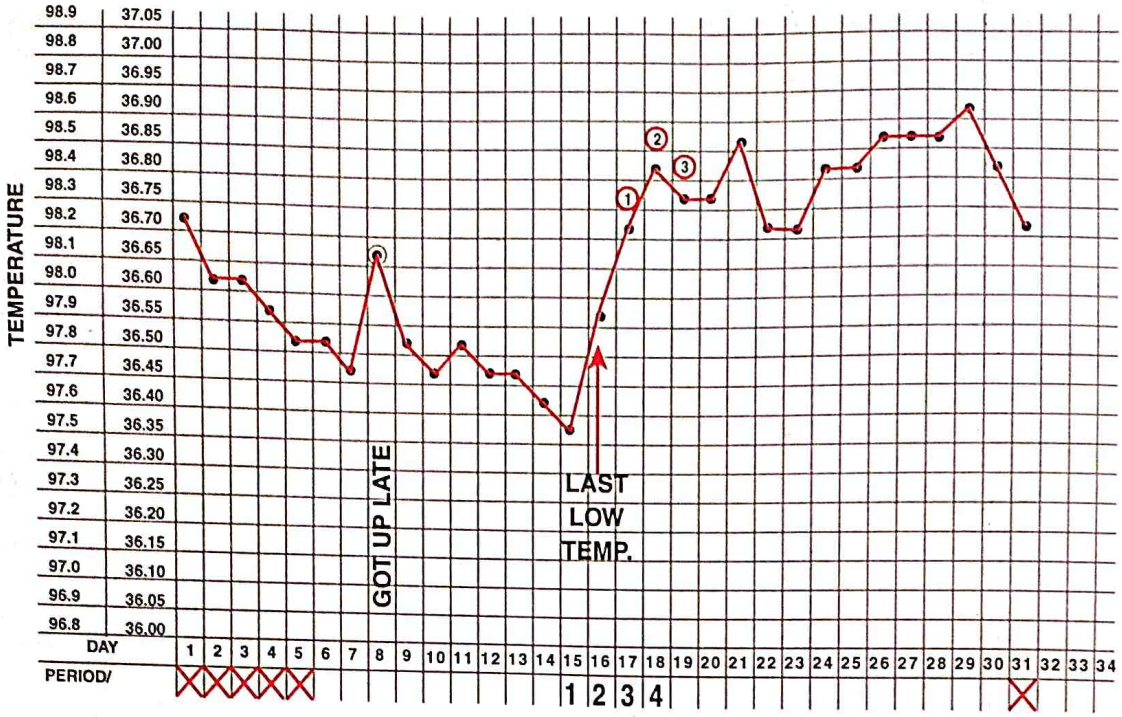



**Fig. 43.1:** Basal body temperature chart showing biphasic curve with temperature rising in the second half of cycle. Coitus is avoided during the fertile phase; X: Days of menstruation

certain days during the menstrual cycle when conception can occur. Following ovulation, the ovum is viable within the reproductive tract for a maximum of 24 hours while the sperms can survive for 3–7 days but can fertilize up to 3 days. The various rhythm methods are calender method, temperature method or cervical mucus method. The woman maintains a record of first day of menstruation (day one) by marking on a calender for 6 months and notes the longest and shortest cycles. The first unsafe day is calculated by subtracting 18 days from the shortest cycle (S) while the last unsafe day is calculated by deducting 11 days from the longest cycle (L) (S-18 to L-11). In a regular 28 day cycle, the unsafe period is from day 7 to day 21. The users of **temperature method** (recorded in the morning on awakening) avoids intercourse until the third day of the rise of temperature (Fig. 43.1). The users of **cervical mucus method (Billing method)** avoid intercourse during the fertile phase 2 of the cycle when the mucus is copious, translucent, thick and can be easily stretched between the testing fingers **(no coitus in pre-ovulatory and ovulatory phases)**. The effectiveness can be improved by combining the mucus method with the calendar or temperature methods to give a failure rate of 2.6 pregnancies per HWY **(level II evidence)**.

### Advantages

1. There are no physical side effects.
2. The method is economical.
3. It does not interfere with natural fertility.
4. There are no health risks involved.

### Disadvantages

1. The method failure rate is high (10–30 per 100 women years).
2. The method offers no protection against sexually transmitted diseases, including HIV and hepatitis B.
3. It can inhibit spontaneity and places a mental strain on the couple.

**Symptothermal method:** combined method based on basal body temperature (BBT) and Billing's method: most effective method in natural methods.

### (ii) Lactational Amenorrhea method

Ovulation occurs at 4 weeks in non-breast feeding or partial breast feeding women while it occurs at 10 weeks in fully breast feeding women. Hence contraception is needed from 3rd weeks in 1st group and 3rd month in second group (rule of 3). Lactational amenorrhea method is appropriate for 6 months postpartum in women who are fully breastfeeding their infant day and night and who are preferably amenorrheic **(Bellagio criteria)**. During lactation ovulation is suppressed and the cervical mucus is thick.

### Advantages

All the advantages of breastfeeding (see Chapter 29 for details).

### Disadvantages

1. The pregnancy rate in the first 6 months after delivery is 2 per hundred women years. Later it increases to 6 per hundred women years between 6 and 12 months postpartum.
2. It does not protect against STDs.
3. Protection rapidly diminishes when the infant is not fully breastfed.

**Overall breastfeeding has prevented more pregnancies in the world (especially developing countries) than all the methods of contraception put**

together. But it cannot be relied upon in individual cases. During breastfeeding, additional contraceptive in the form of condom, intrauterine device or injectable steroids should be given for better contraception.

### (iii) Coitus interruptus or withdrawal method

Coitus interruptus or withdrawal method means discharge of semen outside the female genitalia at the end of sexual intercourse, by withdrawing penis shortly before ejaculation. This method is not barred by Roman Catholic Church and by holy Quran and is used worldwide by Catholics and Mohammedans.

#### Advantages

1. It needs no appliance or medical supervision.
2. It is free of cost.
3. It causes no harm.

#### Disadvantages

1. It needs great motivation and self control by the male partner, is stressful and hampers sexual pleasure.
2. The female partner may develop sex neurosis, pelvic congestion syndrome with dysmenorrhea or hypothalamic amenorrhea due to fear of pregnancy.
3. High failure rate (up to 18 per HWY) as precoital secretions may contain sperms.
4. Risk of accidental deposition of sperms into the vagina.

## 2. BARRIER METHODS

The traditional methods of contraception used by men or women were designed to prevent the entry of live sperms into the cervical canal. For this to be achieved, two conditions must be fulfilled.

1. Direct insemination of the cervix during ejaculation must be made impossible.
2. Any sperms deposited in the vagina must be immobilized before they can reach the cervix. For the greatest protection, therefore, two elements should be used: a mechanical barrier protecting the external os and a dose of chemical spermicide in the vagina.

### i. Male Condoms (Figs. 43.2 A and B)

#### Types

Most condoms are made of latex (plain or treated with spermicide), polyurethane and silicone. Rubber condoms are also manufactured. Latex condoms are 0.3–0.8 mm thick and 15–20 cm long, 3–3.5 cm in diameter. Sperms that are 0.003 mm in diameter cannot penetrate condoms. Condom available free of cost at government hospitals is 'Nirodh'. Many market brands are available some being lubricated. The spermicide immobilizes or kills the sperms providing additional protection in case of breakage or leakage. Condom is an effective contraceptive method as well as a protector against STDs. The male condom is the only one proven to be effective against HIV infection.

#### Instructions for Use

Prospective users need clear instructions to avoid pregnancy and STDs. These are:

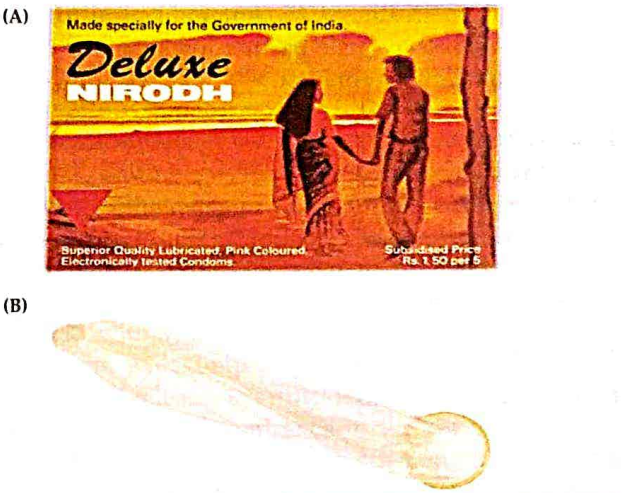



**Fig. 43.2:** (A) Male condom (Nirodh) available free of cost at government outlets in sealed cover; (B) Male condom opened up

1. A condom must be placed on the penis before it touches the partner. Some men penetrate without condom and wear condom just before ejaculation. It is not advisable due to high failure rate as pre-ejaculate may contain spermatozoa.
2. The tip of the condom should extend beyond the end of the penis to provide a reservoir to collect the ejaculate.
3. After coitus, the condom should be held at the base as the still erect penis is withdrawn.
4. Some couples use condom only in pre-ovulatory and ovulatory phases by combining rhythm method with condom use. Failure will be high in such cases.

#### Efficacy

Failure rates are less than those quoted for vaginal barrier methods and spermicides and are 2% for ideal users and 15% for typical users.

#### Advantages

1. Hygienic, easy to carry and disposable
2. Prevents sperm allergy and formation of sperm antibodies
3. Erection enhancement
4. Very useful for couples having infrequent coitus or who are nearing menopause
5. Relatively inexpensive
6. No systemic side effects
7. No interference with menstruation
8. Can be used along with other contraceptive methods to enhance protection
9. Does not adversely affect pregnancy, if method fails.
10. Causes male involvement in contraceptive effort.
11. Non-contraceptive benefits
    - (*i*) Best overall choice for prevention of STDs, including HIV
    - (*ii*) Prevents pelvic inflammatory disease
    - (*iii*) Prevents ectopic pregnancy and tubal infertility
    - (*iv*) Prevention against CIN and carcinoma cervix by preventing HPV infection, if regularly used.

(v) As a treatment for immunological infertility. It is used by male partner for 3 months to prevent antibody formation.

**Disadvantages**

1. Latex allergy can occur.
2. It hampers sexual pleasure by preventing direct contact.
3. Risk of breakage of condom, slippage or leakage of semen
4. Must be used with every act of intercourse.
5. Not reusable

## Newer Male Condoms

Polyurethane condoms

**Advantages**

1. More resistant to heat and oxidation.
2. Thinner material
3. Can be used with lubricant.

**Disadvantage**

Higher breakage risk during use.

## ii. Female Condom (Figs. 43.3 A and B)

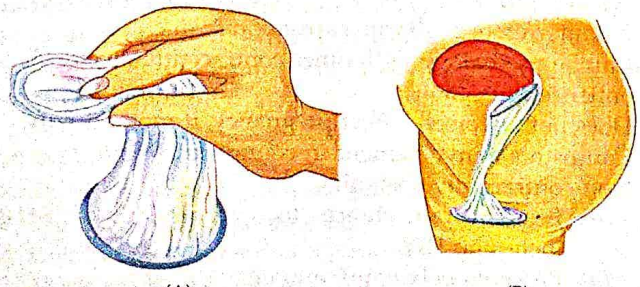
Female condom is a newer introduction to the group of female barrier contraceptives. It acts by providing a physical barrier lining the vagina, occluding the cervix and partially covering the perineum. It is made up of thin (0.05 mm) polyurethane sheath, 7.8 cm in diameter, 17 cm in length, less prone to tearing as it is stronger than latex. It is pre-lubricated from inside with silicone-based lubricant. It contains two flexible polyurethane rings at each end. The inner smaller ring is the closed end of the sheath serving as insertion mechanism and internal anchor, while the outer bigger ring forms the external opening lying outside the vaginal orifice after insertion (Figs. 43.3 A and B). Two brands are available in India: Femshield and Femidom (FCI).

**Insertion Technique**

Follow the instructions given on package or follow the following instructions.

1. Hold the pouch with open end hanging down.
2. Spread labia with fingers of one hand.
3. Use thumb and middle fingers of the other hand to squeeze the inner ring into a narrow oval for facilitating insertion, use the index finger as guide, the inner ring and pouch are pushed deep into the vagina with the outer ring resting outside the vulva.

Female and male condoms should not be used together due to risk of slipping, tearing and displacement.



(A) (B)

**Fig. 43.3:** (A) Female condom (Femidom); (B) Female condom in position in vagina

**Removal**

Female condom is removed soon after coitus by traction on the outer ring.

**Advantages**

1. Protection against HIV infection as it is impenetrable to the virus.
2. Protective against STDs
3. Easy to use.
4. Available over the counter
5. No health hazards

**Disadvantages**

1. High failure rate (5 to 20 per 100 women years)
2. Small risk of toxic shock syndrome
3. Increased risk of urinary tract infections
4. Unsuitable for use by women with deficient perineum, uterine prolapse, large cystocele, rectocele and retroverted uterus.
5. Currently recommended for single use.
6. More expensive than male condom.
7. Interferes with sexual pleasure
8. Produce sounds during coitus.

## NEWER FEMALE CONDOMS

1. FC2 (Female Condom 2) - Made of synthetic latex, half as expensive as FC1.
2. VA Feminine Condom - Preformed lubricated latex pouch with a V-shaped outer frame.
3. Path Women's Condoms - Undergoing trials developed by taking women's views and feedback.

## iii. Dutch cap (Diaphragm) (Fig. 43.4)

Dutch cap type consists of a thin rubber dome attached to a rubber covered circular metal rim which may be a flat watch spring or a coil of thin wire. This measures the approximate diameter size. The diaphragm is held in position partly by the tension of the spring and partly by the muscles of the vagina. The diaphragms are made in sizes ranging from 55–100 mm.



**Fig. 43.4:** The diaphragm

**Instructions for Use**

Diaphragm users should be given the following instructions.

1. The diaphragm should be inserted 3 hours prior to sexual intercourse.
2. Spermicide cream or jelly should be placed in the dome of the diaphragm prior to insertion.

3. The diaphragm should be left in place for approximately 6 hours (but no more than 24 hours) after the last coitus.

**Advantages**

1. There are no method related health risks.
2. Diaphragm is an effective contraceptive which if used properly offers some protection against STDs.
3. There is some protection against cervical cancer as compared with women using no contraceptives.

**Disadvantages**

1. Initially a physician or trained person needs to demonstrate proper technique of insertion of diaphragm.
2. It can only be used after complete involution of uterus after delivery (at least 6 weeks).
3. There is risk of toxic shock syndrome if left in vagina for long period.
4. It cannot be used for torn perineum or with large cystoceles.
5. It has a high failure rate of 8–20 per HWY which can be lowered by use of spermicides.

**Care of the Diaphragm**

After removal, the diaphragm should be washed with soap and water, rinsed and dried completely.

### iv. Cervical Caps (Fig. 43.5 A)

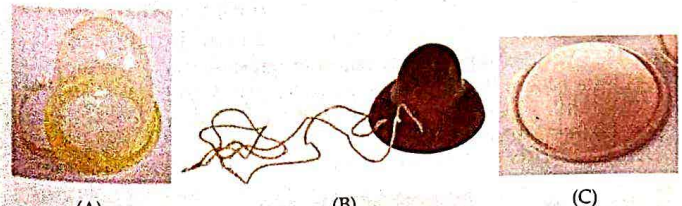
Cervical cap is shaped like a thimble with a raised rim and is made of rubber or plastic and covers the cervix at its base.

Failure rates of 8–20 per hundred women years are reported. Not popularly used in India.

Vinule cap is also a cervical cap with threads attached to it (Fig. 43.5 B).

### v. Vault cap (Dumas cap) (Fig. 43.5 C)

Vault cap is a bowl-shaped suction cap which clings to the vaginal roof to cover the cervix.



(A) (B) (C)

**Fig. 43.5:** Various types of cervical and vault caps
(A) Cervical cap; (B) Vinule cap; (C) Vault (Dumas) cap

### vi. Spermicides

Spermicides are harmless, easy to use and fairly effective, chemical contraceptives which do not need help from the doctor or nurse. However, they have the disadvantage of having to be inserted shortly before coitus. The risk of pregnancy when a chemical method alone is used is greater than when combined with a mechanical barrier.

Modern spermicides contain surface active agents that damage the sperm cell membranes. The agents currently used are nonoxynol 9, octoxynol 9 and benzalkonium chloride. Most preparations contain 60–100 mg of these agents in various applications.

Chemical contraceptives are made up in the form of foam tablets, creams, pastes, jellies (contained in metal or plastic tubes with screw-on caps) (Fig. 43.6 A) or aerosols (pastes or creams compressed into strong containers with a gas). The spermicide is released in the vagina. Vaginal contraceptive sponge (Fig. 43.6 B) is made of polyurethane impregnated with 1 g of nonoxynol 9 as a spermicide. It is 5.5 × 2.5 cm with dimple on one side and a satin loop on other side. It releases spermicide during coitus, absorbs ejaculate and blocks the entrance to the cervical canal. It should be kept for 6 hours after intercourse (maximum wear time 30 hours). Its failure rate is about 10/HWY. It does not have much protection against STDs and HIV.

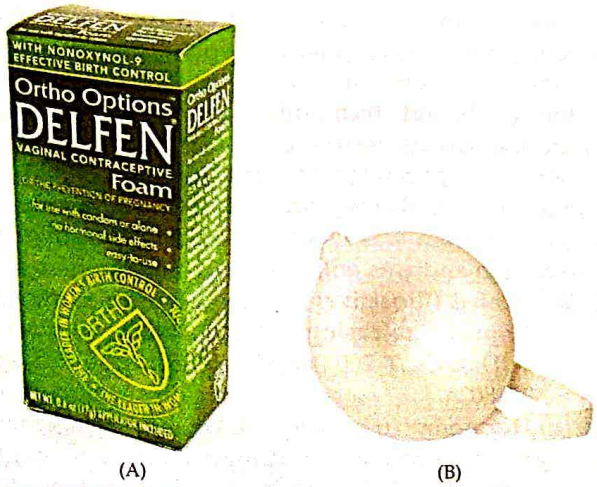
**Advantages of Spermicides**

1. Easy to use
2. There are no method-related health risks.
3. Offer some protection against STDs.
4. Antiviral properties of nonoxynol make it protective against risks of cervical cancer.

**Disadvantages of Spermicides**

1. Local irritation, allergy, discomfort and vaginitis
2. High failure rate of 10–25 per hundred women year
3. Effectiveness lasts for 1–2 hours only.
4. Incomplete protection against STDs
5. Increased risk of acquiring HIV due to genital lesions and trauma.
6. Interrupts spontaneity of coitus
7. Needs to be repeated at every act of coitus
8. Considered to be messy

Use of barrier methods, especially condoms, is encouraged with other contraceptive methods, to protect against sexually transmitted infections STIs **(dual contraception)**. It is strongly recommended for sexual intercourse with a person other than regular partner.




(A) (B)

**Fig. 43.6:** (A) Chemical contraceptive (spermicide) foam; (B) Vaginal contraceptive sponge (Today)

### Fertility awareness combined methods (FACMs)

**Natural methods** (rhythm method, coitus interruptus and lactational amenorrhoea method) and **barrier methods** (male and female condoms, diaphragm, spermicides) are also called **fertility awareness based methods**. When fertility awareness methods involve use of condoms during fertile period, it is called fertility awareness combined method.

## INTRAUTERINE CONTRACEPTIVE DEVICES (IUCD) [SYN. INTRAUTERINE DEVICE (IUD), COIL]

**IUCD is a reversible birth control device placed in the uterine cavity for contraception.** In 2007, 17% women of child bearing age used IUDs in developing countries and 9% in developed countries. Thus 180 million women used it worldwide. In India only 1.7% women used IUD as compared to 45% in China. Worldwide IUCD is the second most commonly used family planning method after voluntary female sterilization.

**History.** Grafenberg (Germany) introduced a ring of silkworm gut and silver wire in 1929 while Ota (Japan) introduced Ota ring made of gold or gold plated silver in 1934. However, they went into disrepute due to risk of pelvic infection. The Lippes loop (Jack Lippes, Buffalo, USA 1962) was made of polyethylene with barium sulphate to make it radio opaque and nylon threads to be hung through cervix for easy detection and removal. Medicated devices Copper T and Copper 7 were developed in 1969 (Zipper and Tatun). Closed devices (Grafenberg ring, Birnberg bow) were tried and discarded due to risk of strangulation of intestines. All IUDs in use now are open devices without the risk of bowel strangulation.

### Types of Devices (Figs. 43.7 A-G)

Intrauterine devices are divided into different types according to their shape and content.

### Classification based on the content of IUD

1. **Inert (non-medicated).** They are also called **First Generation** IUDs and are made of plastic. Lippe's loop, Saf T coil, Margulis spiral, Soonawala-Z and other types are some examples. These IUDs required the **push out technique** for introduction into the uterine cavity resulting in uterine perforation and other complications at times. Dalkon shield which had a central membrane to increase contact with endometrium was associated with high rate of pelvic infection and was withdrawn.
2. **Medicated (Bioactive) Devices.** They are of two types.
   (*i*) **Copper releasing IUDs (second generation IUDs).** Cu T 200, Multiload 250, Cu T 375 and Cu T 380 A.
   (*ii*) **Hormone releasing IUDs. (third generation IUDs).** Progestasert (progesterone releasing) and Levonorgestrel Intrauterine System (LNG-IUS or Mirena) which release levonorgestrel.

### WHO Categorization of IUDs

WHO (1987) divided IUDs into three groups according to the pregnancy rate, indicating their contraceptive efficacy.

1. **Group I.** Where pregnancy risk is more than 2 per 100 women years, e.g. Lippe's loop (Fig. 43.7 A), CuT, CuT 200, CuT 200 B (Fig. 43.7 B).
2. **Group II.** Where pregnancy rate is between 1.0 and 2.0 per 100 women years, e.g. Nova T, Multiload (Fig. 43.7 E to H), Cu 250, CuT 220C.
3. **Group III.** Where pregnancy rate is less than 1.0 per 100 women years (usually less than 0.5), e.g. CuT 380A (Fig. 43.8 A), Multiload Cu 375 (Fig. 43.7 H) and LNG IUS (Fig. 43.8 B).

#### Unmedicated IUDs

The Lippe's loop is a polyethylene device in the shape of a double 'S' with a small polyethylene filament protruding through the cervix to facilitate identification (Fig. 43.7 A). It is available in three sizes; 31 mm, 27.5 mm and 25 mm. The loop introducer and plunger are kept sterilized by putting them in a solution of 1 in 1000 acriflavin in water or savlon or similar antiseptic for 24 hours. It is not in use in India any more.

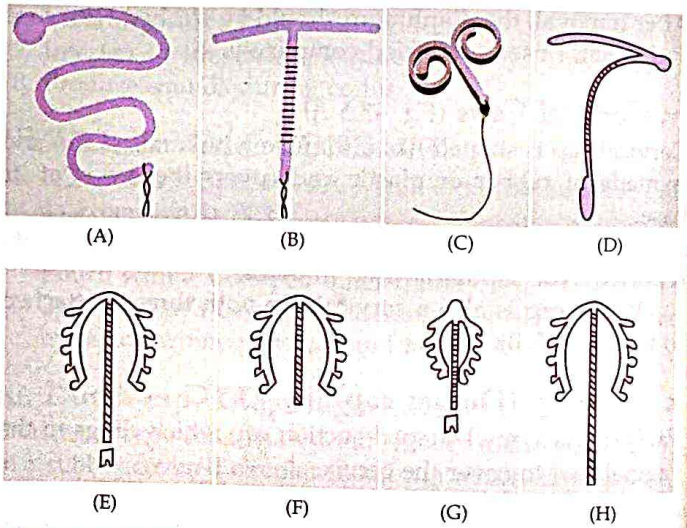



**Fig. 43.7:** Various types of intrauterine devices.
(A) Lippe's loop; (B) Copper T 200B; (C) Saf T coil; (D) Copper 7; (E) Multiload Cu 250; (F) Multiload Cu 250 short; (G) Multiload 250 mini; (H) Multiload Cu 375

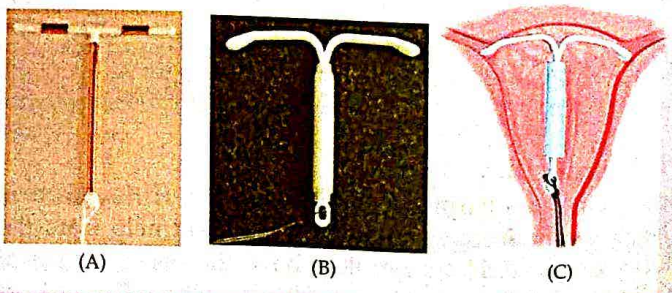



**Fig. 43.8:** (A) CuT 380A; (B) Levonorgestrel intrauterine system (LNG-IUS); (C) A LNG-IUS in position in uterus

*Advantages of IUDs over other temporary methods of contraception*

1. Very effective (failure rate 0.1 to 2.0/HWY)
2. Safe
3. Convenient to use
4. Does not need a strong motivation on the part of the user.
5. May be used successfully in lactating women.
6. Indicated in women in whom oral pills are contraindicated or cause side effects.
7. Can be used over long intervals of time.
8. Does not require repeated usage before each sexual intercourse.
9. Affordable (CuT 380 A is supplied free of cost by the Government of India)
10. Does not interfere with sexual activity or pleasure.
11. No interaction with drugs
12. No hormonal side effects
13. Immediately reversible and is ideal for spacing.
14. LNG IUDs reduce dysmenorrhea and menorrhagia.
15. Some women are now using CuT 380 A even as a permanent method up to 1 year after menopause.

*Disadvantages*

1. Menstrual irregularity
2. Trained personnel needed to insert and follow up IUD.
3. Insertion is painful.
4. Does not prevent against STD
5. Increases incidence of PID
6. Woman cannot stop IUCD use on her own.
7. They need strong motivation.
8. There is slight risk of ectopic pregnancy.
9. Risk of expulsion
10. Dysmenorrhea

## Advantages of Newer IUDs (CuT 380A, Multiload 375 and Levonorgestrel IUD) over the inert IUDs

1. They are very effective (almost equal to sterilization). Pregnancy rate is 0.1 to 0.3/HWY.
2. They work for 5–10 years (CuT 380A: 10 years, LNG-IUS: 5 years), multiload 375 : 5 year.
3. They are easily reversible on removal of the device.
4. Expulsion rate is very low.
5. Well tolerated with minimal need of removal.
6. Ectopic pregnancy is highly unlikely with LNG-IUS and CuT 380A (< 0.02/HWY).
7. Incidence of PID is very less with LNG-IUS due to protective effect of progestogen against infection.
8. Non-contraceptive benefits of LNG-IUS include:
   (a) Reduction of blood loss during menses which is useful for women with anaemia, menorrhagia and abnormal uterine bleeding.
   (b) It reduces pain and dysmenorrhea in endometriosis, adenomyosis and premenstrual tension syndrome (PMS).
   (c) It can be used as a substitute for hysterectomy in menorrhagia and abnormal uterine bleeding. Infact LNG-IUS has decreased incidence of hysterectomy in western countries for menstrual dysfunction cases.
   (d) It can be used as medical management of endometriosis and fibroids.
   (e) It can be used as the progestogenic component of combined HRT.

## Disadvantages of Third Generation IUDs

1. LNG-IUS is costly (about ₹ 5000) and is not available through government channels.
2. It causes amenorrhea in 20–50% cases.
3. It can cause minor side effects like acne, dizziness, headache breast tenderness, nausea, vomiting and weight gain.
4. It cannot be used in conditions like deep vein thrombosis, ischemic heart disease, classical migraine and breast cancer.

## Description of the Commonly Used IUDs

**CuT 380A** (Fig. 43.8 A). It is a polyethylene and barium sulphate T-shaped frame (for X-ray opacity) on which copper is wound. There is 314 $mm^2$ of fine copper wrapped around the stem and 33 $mm^2$ copper bracelet on each arm with a total of 380 $mm^2$ of copper. Two polyethylene filaments 10.5–12.5 cm extend from the base of the stem. Insertion is by withdrawal technique. It has a plastic ball at base to prevent cervical penetration. It is the only copper T which is currently approved for use. Release of copper is about 50 µg per day. It is effective for 10 years. **It is supplied free of cost by the Government of India through the National Family Welfare Programme.**

Now, the Government of India is providing CuT 380A instead of copper T 200 or 200 B as its cost to government is only marginally high (about ₹ 80) and it is more effective with fewer side effects. Many couples are now using it even as a permanent method as it can be changed after 10 years and can be removed one year after menopause. Even for couples needing spacing, it can be removed whenever they want conception even after 2–3 years. Hence, almost all copper Ts in use now are Cu T380As. It is also used by many gynecologists after adhesiolysis in Asherman's syndrome for prevention of reformation of adhesions (usually after removing copper).

**Copper T 200.** It was also called Gynae T and was the most commonly used CuT till recently in India and was supplied free of cost by the Government of India. However, it has now been replaced by CuT 380A (see above). It was a T device made of polypropyelene frame impregnated with barium sulphate for better radioopacity. It had 120 mg of 0.25 mm diameter copper wire wrapped around the stem with an overall 200 $mm^2$ of copper surface area. There were two monofilament nylon threads at the end of vertical stem for easy detection and removal of the device. Copper was released at a rate of 50 µg per day. Its effective life was 3 years. Copper T 200 B (Fig. 43.7 B) was like CuT 200 in all respects but had slightly more copper (total 124 mg) and longer effective life (4 years). It had 215 $mm^2$ copper surface area.

**Copper 7** (Fig. 43.7D). It was also called gravigard and had a shape of number '7' with 200 $mm^2$ copper wire wound round the vertical stem (35.9 mm long). It could be inserted by both push technique or withdrawal technique and had effective life of 3 years.

**Copper T 200 C:** It is a modified version of CuT 200 developed by the Population Council. It has got five copper sleeves on vertical stem and two (one each) on transverse arms with a total copper surface area of 220 $mm^2$ with effective period of 3 years and is inserted by withdrawal technique. Gravigard copper 7 intra uterine device was 7 shaped device with 200 $mm^2$ copper but is not much used now.

**Multiload copper devices.** These are copper releasing devices made of polypropyelene and have copper wrapped around the vertical shaft only. The two arms are flexible plastic serrated fins which keep the device in situ in uterine cavity. The vertical shaft is radio-opaque while the arms are radiolucent. They are available in pre-loaded special inserters of withdrawal type helping in their insertion high up in the uterus. There is no plunger. Multiload Cu 250 (Fig. 43.7E) has 250 $mm^2$ copper surface area releasing about 75 µg per day of copper. It is not available through government channels and has to be bought from market in about ₹ 250 and has effective life of 3 years. Multiload Cu 250 is also available in two other Multiload Cu 250 mini (Fig. 43.7F) for uterus with intracavitary length of 5 cm and CuT 250 mini (Fig. 43.7G) for uterus with an intracavity length of less than 5 cm. Multiload Cu 375 has got 375 $mm^2$ of copper surface area and has effective life of 5 years. Multiload devices are available in sterilized packs and can be introduced without any handling which is an advantage. They are popular amongst private practitioners and rich patients. Since 2012, multiload Cu375 has been made available free of cost through National Family Welfare Programme of Govt. of India.

**Hormonal IUDs.** Progestasert and levonorgestrel

**Progestasert** – It is a T shaped device with a capsule containing (38 mg) progesterone that releases 65 micrograms progesterone per day in the uterine cavity. It is effective for 1 year. It has been mostly replaced by newer hormone containing IUDs and is not marketed any more.

**Levonorgestrel intrauterine system** (LNG-IUS) (Mirena) (Fig. 4.3 B and C): It is a long acting progestin releasing device and is approved for use in USA. It has a T-shaped radio opaque polyethylene frame on whose stem a steroid reservoir made of polydimethylsiloxane-levonorgestrel mixture is wrapped. It contains a total of 52 mg LNG with a release rate of 20 µg per day. It has got two brown threads attached to the stem. Its effective life is for 5 years. It is much more efficacious than copper devices with failure rate of 1–3 per 1000 women. It mainly acts by local progestogenic effects. It is almost as effective as sterilization but is easily reversible. It has many other health benefits for women like treatment of menorrhagia, anaemia, reduction of pain and dysmenorrhea in endometrosis and helpful effect in fibroid uterus. **Disadvantages** are its high cost (about ₹ 5000), irregular bleeding, oligomenonhea and sometimes amenorrhea and other minor side effects like nausea, vomiting, headache, dizziness and weight gain. **It cannot be used as a post coital contraceptive.** In USA only CuT 380 A and LNG IUS (Mirena) are approved for use.

Another LNU-IUD by the name of Skyla has been approved by USFDA in 2013. It is a smaller device (1.1 × 1.2 inches) as compared to mirena (1.3 × 1.3 inches) with smaller inserter tube (0.15 inches) as compared to mirena (0.19 inches). It releases only 14µg LNG per day and is effective for 3 years.

## Mechanism of Action of IUD

Exact anti-fertility mechanism is not clear. Several possible mechanisms are as follows:

1. **Chemical and cellular changes in the endometrium.** All unmedicated and Cu devices produce an inflammatory or foreign body reaction which in turn causes cellular and biochemical changes in the endometrium and tubal fluids **(aseptic inflammation)**. Prostaglandin levels increase and the fibrinolytic mechanism needed for hemostasis is affected. Numerous polymorphs, giant cells, mononuclear cells, plasma cells and macrophages appear in the endometrium as well as in the uterine and tubal fluids. These cells engulf or consume sperms and ova by the process of phagocytosis and thus prevent fertilization. When inserted post coitally, IUD can prevent implantation of fertilized ovum.
2. **Increased tubal motility.** Resulting in quick migration of the fertilized ovum into the uterine cavity before the endometrium is receptive.
3. **Impaired** sperm ascent
4. **Copper devices.** Local anti-fertility effect by preventing implantation through enzymatic interference.
   **In nutshell IUDs prevent implantation of fertilized ovum.** (Students should answer this in examination)
5. **Levonorgestrel-IUD.** *It hinders implantation in the endometrium secondary to the effect of the local action of the progestogen on the endometrium.* It also prevents passage of spermatozoa through the cervical mucus by making it thick, scanty and hostile. It also prevents ovulation and luteal phase activity. There is no increase in serum progesterone.

## Timing of Insertion

1. **Interval insertion.** Interval insertion includes insertion of IUD at least 6 weeks after parturition or MTP or abortion. Insertion of IUD is done soon after menstruation when the cervix is usually softer, more dilated for easier insertion and excludes early pregnancy. It can even be inserted during menstruation but most Indian women do not prefer IUD insertion during menstruation. However, IUD can be inserted at any time of period after excluding pregnancy.
2. **Insertion during lactational period.** IUD can be safely inserted during lactational period after ruling out pregnancy. IUD does not affect lactation in any way. It is preferable to use smaller device during this period.
3. **Post abortal.** IUD can be inserted safely immediately at the end of evacuation of uterus both after medical termination of pregnancy or after spontaneous abortion

after ensuring empty uterus. After medical abortion it is advisable to insert IUD after 2 weeks after ensuring compete abortion and empty uterus. Often women accept IUD at time of abortion. However, there is 5–10 times chances of expulsion of IUD if inserted after second trimester MTP.

4. **Immediate postpartum** (< 48 hours) or **post placental insertion.** IUD is inserted following delivery of the placenta both after normal delivery and after cesarean section. **Government of India is recommending it now.** Large and long sponge holding forceps (Kelly's forceps) have been devised to introduce CuT 380A near fundus. There is no need to use loop of catgut stitch for anchor. The expulsion rate is slightly high.
5. **Post coital insertion within 48 hours of delivery.** Copper devices **(not** LNG-IUS**)** can be used for post coital contraception up to 5 days after unprotected coitus.

## Technique of Insertion of Copper T, Cu T 380A

(Figs. 43.9 A-G)

1. No sedation or anesthesia is required. To reduce cramping pain, tablet drotaverine alone or with mefenamic acid or ibuprofen (NSAID) may be given 30 minutes before insertion.
2. **'No touch' insertion** method is preferred. This includes:
   (*i*) To load the CuT in the inserter while both parts are still in the sterile package.
   (*ii*) Cleaning the cervix with antiseptic before IUD insertion.
   (*iii*) Avoid touching the vaginal wall or speculum blades with the uterine sound or loaded IUD inserter.
   (*iv*) Passing both, the uterine sound and the loaded IUD inserter, only once through the cervical canal.

**Procedure**

Intrauterine device is usually inserted as an outpatient procedure. The woman lies on the examination operating table in dorsal or lithotomy position after passing urine. Vulva and perineum are inspected for any infection or abnormalities. Speculum examination is performed to see condition of cervix and vagina and to rule out any infection. Bimanual examination is performed to note down the size, shape and position of uterus (anteverted and retroverted) and any adnexal masses, and to rule out any contraindication for IUD insertion. Although IUD insertion can be performed through Cusco's speculum, use of Sim's speculum is preferred. Posterior vaginal wall is retracted with adequate-sized Sim's speculum. Anterior vaginal wall is retracted (elevated) with anterior vaginal wall retractor. Vagina and cervix are scrubbed with povidone iodine or other antiseptic solution. Anterior lip of cervix is gently caught with Allis forceps or tenaculum (avoid volsellum as it is painful). Using a uterine sound passed through cervix, length of uterine cavity is measured and uterine position (anteverted or retroverted) is made out. As per uterine length, the suitable length of the inserter is adjusted for the IUD insertion. The horizontal limbs of the device are bent to lie along the vertical limb. Now the device is pushed into the proximal end of the inserter so that the arms are trapped inside the tube. The arms of CuT should not be kept bent for more than 5 minutes as it may cause the arms to remain bent and the device may fail to fit properly in the uterine cavity (as per Government of India guidelines). The inserter with the device placed inside is then introduced through the cervical canal ending just below the fundus.

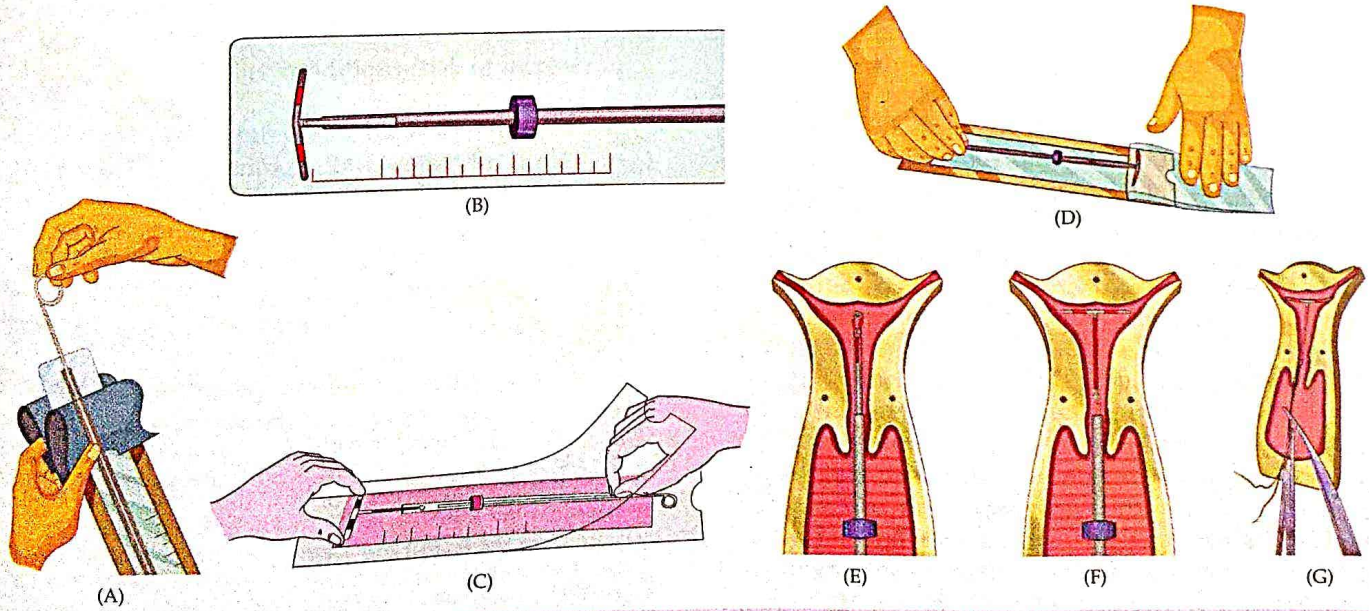



**Fig. 43.9:** Steps of insertion of Copper T by withdrawal method. (A & B) Flexible plastic inserter with plastic plunger; (C & D) CuT is negotiated into the top end of the inserter. Ideally it is done by no touch technique; (E & F) CuT loaded inserter with plunger is introduced into the uterine cavity; (G) The inserter is retracted over the plunger leaving CuT inside the uterus and the plunger and inserter are withdrawn; CuT in position.

After positioning it by the guard, the inserter is withdrawn keeping the plunger in position without pushing the IUD **(withdrawal technique)**. Hence, the IUD remains at the same place in the uterine cavity without the risk of perforation (a risk possible with push technique) and CuT is now in position with thread hanging out through cervix. The extra thread is cut with scissors leaving behind only about 2.5 cm thread protruding through the cervix. Allis forceps or tenaculum is removed and the Sim's speculum is taken out. The woman is asked to sit down for few minutes, If she is fine with no pain, she is allowed to stand and walk and can go home.

**Technique of Insertion of Lippes Loop** (Fig. 43.10)
Lippes loop is not used any more. The procedure of insertion is by **push technique** shown in **Fig. 43.10** with other precautions being same as Copper T.

**Insertion of Multiload 250 and 375** (Figs. 43.11)
It is the easiest method of inserting an IUD and is performed by withdrawal technique without use of a plunger. The wrapping is removed and the inserter tube with the preloaded IUD is carefully inserted into the uterus until it reaches the fundus. When the IUD touches the fundus, it is released into the uterine cavity by withdrawing the inserter tube. Being flexible it can be easily compressed in either direction and can be easily inserted. During removal the thread is just pulled out and the multiload comes out easily with the multiload bending in opposite direction.

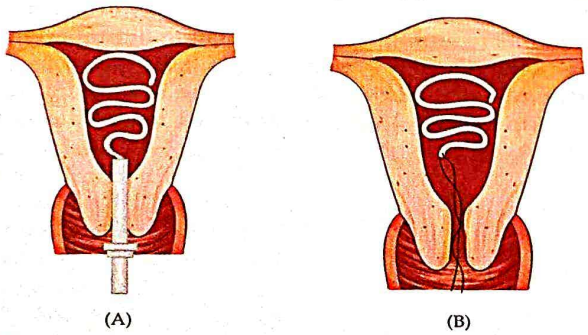



**Fig. 43.10:** Steps of insertion of Lippes loop. (A) Lippes loop is being pushed into the uterine cavity; (B) Lippes loop in position in uterus after withdrawal of the inserter

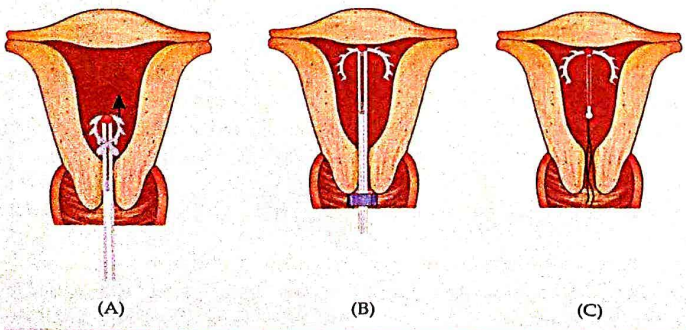



**Fig. 43.11:** Steps of multiload insertion

## Insertion of LNG-IUS (Mirena)

(Fig. 43.8 B, C and Fig. 43.12)
Grasp the upper lip of the cervix with a tenaculum and apply gentle traction to align the cervical canal with the uterine cavity. Carefully sound the uterus to measure the uterocervical length. The uterus should sound to a depth of 6 to 9 cm. Insertion of LNG-IUS into a uterine cavity less than 6.0 cm by sounding may increase the incidence of expulsion, bleeding, pain, perforation and possibly pregnancy.
The Evo InserterTM has been devised to simplify the insertion procedure for inserting the LNG-IUS. There have been no changes in the LNG-IUS itself. The technique of LNG-IUS insertion is shown in Fig. 43.12. It is effective for 5 years.

## Newer LNG-IUS

1. Fenulis - shorter sidearms, device well-suited for nulliparous women
2. Fibroplant: Not available in India at present
   (*i*) Frameless IUS
   (*ii*) 2 versions releasing 14 µg/day and 20 µg LNG/day
   (*iii*) 5 years
   (*iv*) Suitable for uterus of any shape and size.
3. Emily LNG IUS is economical (about ₹ 2700) but is effective for only 3 years.

## WHO Eligibility criteria for use of Intrauterine Devices (WHO, 2017)

**Who Category 1: No Restrictions on Use: These women can use IUDs safely and effectively (indications of IUDs)**

1. **Age.** + 20 years, parous women

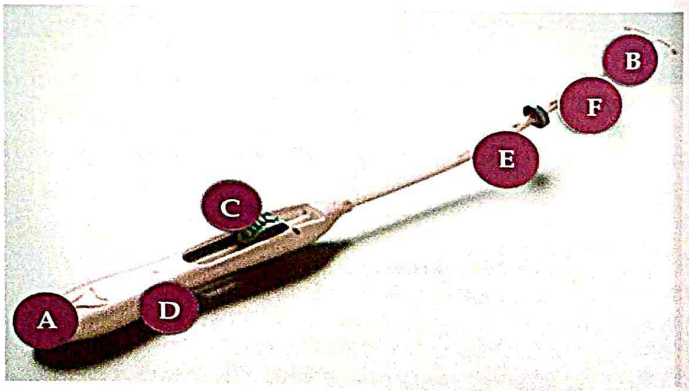



**Fig. 43.12:** (A) Threads have been placed inside the inserter handle: • handling of threads is no longer necessary, • no risk of accidental thread entanglement during insertion, • please note that the Mirena® EvoInserter™ is designed for single use only. Once the system has been deployed it cannot be re-loaded; (B) Mirena® is already pre-loaded in the correct horizontal position for insertion: (C) One-step loading of the IUS: • simplified loading by pushing the slider forward to its furthest position, (D) More ergonomic shape of slider and handle. (E) Centimeter scale is visible on both sides of the insertion tube. (F) Smaller insertion tube outer diameter: • 4.4 mm compared with 4.8 mm.

2. Smoking any age and amount of smoking
3. Obesity
4. Neurological conditions like headaches including migraine with or without aura, stroke and epilepsy
5. Cardiovascular conditions like thromboembolic disorders, uncomplicated valvular heart disease and ischemic heart disease
6. Endocrinological conditions like thyroid disorders and diabetes mellitus
7. Liver and gall bladder diseases
8. Receiving antibiotics or other drugs including rifampicin, griseofulvin and anti-epileptic drugs.
9. Gynecological/Obstetrical conditions other than malignancies and current infection
10. Medical conditions like malaria, non-pelvic tuberculosis and schistosomiasis.

**WHO Category 2: Advantages outweight risk: Here IUDs can be used in judicious indications**

1. **Age.** 10 to 20 years
2. Gynecologic/Obstetric conditions
   (*i*) Nulliparity (it used to be a contraindication in past but not any more)
   (*ii*) Menstrual patterns with prolonged or heavy bleeding/severe dysmenorrhea
   (*iii*) Endometriosis
   (*iv*) Past PID without subsequent pregnancy
   (*v*) Uterine/Cervical abnormalities (including fibroids) that do not distort uterine cavity or interfere with IUD insertion.
   (*vi*) Postpartum < 48 hours
   (*vii*) Post abortion, second trimester
   (*viii*) Vaginal bleeding
   (*ix*) Vaginitis without cervical pus/discharge
3. Cardiovascular conditions
   Complicated valvular heart disease (prophylactic antibiotic is advised)
4. Chronic disease/Other conditions (risk of increase of blood loss)
   Thalassemia (WHO category 1 for LNG – IUS)
   Iron deficiency anaemia (1 for LNG – IUS)
   Sickle-cell disease (1 for LNG – IUS)
5. STD/HIV risk (advise condom use). HIV positive women well-controlled on ART can use IUD.
6. Anti-retroviral therapy
7. Immunosuppressive treatment

**WHO Category 3: Risks outweigh advantages: Avoid using IUD unless absolutely necessary (relative contraindications of IUDs)**

GYNECOLOGIC/OBSTETRIC CONDITIONS

1. **Postpartum.** 48 hours–4 weeks
2. Benign gestational trophoblastic disease
3. Increased risk of STD/HIV (advise condom use)
4. HIV positive or AIDS (advise condom use; dual contraception)
5. Ovarian cancer (WHO category 4 for LNG - IUS)
6. Severe thrombocytopenia

**WHO Category 4: Do not use (absolute contraindications of IUDs)**

GYNECOLOGIC/OBSTETRIC CONDITIONS

1. PID, current or within past 3 months
2. Current STD or STD within past 3 months
3. Known pelvic tuberculosis
4. Unexplained or suspicious vaginal bleeding
5. Malignant gestational trophoblastic disease
6. Cervical and endometrial cancer
7. Uterine/cervical abnormality incompatible with IUD insertion
8. Uterine fibroids with distortion of uterine cavity.
9. Puerperal or postabortal sepsis
10. Suspected Pregnancy (Avoid mentioning it as first thing in examination as there is no indication of using IUD in pregnancy. Examiners get irritated by this answer in examination).
11. Puerperal sepsis

## Complications

### Early Complications

1. **Vasovagal attack or fainting.** It can rarely occur in anxious women, nulliparous women or with a large device possibly due to distension of uterus. Ensuing that the women is not fasting and giving a spasmolytic medicine (e.g. drotaverine with mefenamic acid) can reduce the incidence.
2. **Spasmodic pain.** Mild to moderate suprapubic pain for few minutes to an hour can occur due to uterine distension. It is much less common with modern IUDs and LNG IUS. Use of anti-spasmodic medication (drotaverine with mefenammic acid) half an hour before the procedure can reduce the incidence. However, persistent pain (beyond one week) can be due to faulty position of IUD, uterine perforation, expelling IUD, disparity in size of IUD and uterine cavity, PID or ectopic pregnancy. Persistent pain needs to be properly investigated and may necessitate removal of IUD.
3. **Uterine perforation.** It can rarely occur (1 in 1000 insertions) if push technique is used instead of withdrawal technique or if IUD is fitted in lactational period causing severe pain (see below for details).

### Late Complications

1. **Excessive vaginal discharge or leucorrhea:** It may occur especially in first month, usually due to foreign body reaction. If there is persistent vaginal discharge with pain then pelvic infection should be suspected.
2. **Lower abdominal or pelvic pain.** It can occur due to infection or due to uterine distension by the larger device especially if used in nulliparous women and in lactational period. Use of drotaverine and mefenamic acid or ibuprofen and paracetamol tablets can reduce the pain.
3. **Dysmenorrhea.** Painful periods can occur with IUD. Use of drotaverine with mefenamic acid or accelofenac can reduce it.
4. **Menstrual dysfunction.** IUD use is associated with increased bleeding problems like prolonged and heavy bleeding. Studies have found an increase of blood loss

of 20–30%. Women should be informed that heavy bleeding (menorrhagia) can occur with IUD use.
Causes of menorrhagia include:
(i) Increased production of plasminogen activating enzymes due to IUD causing lysis of fibrin clot.
(ii) Increased endometrial vascularity
(iii) Hormonal asynchronization
Increased bleeding and pain are responsible for most IUD removals (up to 2–10 /100 users in first year). Sometimes metrorrhagia (intermenstrual bleeding) can also occur due to mechanical damage to the endometrium.
Use of NSAIDS or antifibrinolytic agents (tranexamic acid) 500 mg thrice daily during menses can reduce the amount of blood loss especially in first 3 months of IUD use after which bleeding tends to settle down.
LNG IUS on the other hand can cause amenorrhea, spotting, oligomenorrhea and pre-menstrual syndrome.

5. **Anaemia.** The increase in menstrual blood loss associated with the use of copper IUDs has the potential to cause iron deficiency anaemia. LNG-IUS reduces the amount of blood loss and reduces the incidence of anaemia.
6. **Pelvic inflammatory disease.** It is a sexually transmitted infection of genital tract usually caused by Chlamydia trachomatis and can cause chronic abdominal pain, ectopic pregnancy and tubal factor infertility. Asymptomatic chlamydial infection may be there in up to 11% women of reproductive age group necessitating screening for it before inserting IUD. Uterine instrumentation carried out as a part of IUD insertion may result in iatrogenic PID from endocervical chlamydial infection. However, the risk of developing PID following IUD insertion is very low (less than 1%) in women who are at low risk of sexually transmitted infections. In India, routine screening for Chlamydia is not cost effective and is not performed or recommended. Routine prophylactic antibiotics (doxycycline or azithromycin) for all cases of IUD insertion is not recommended. However, if the IUD user develops PID, testing for the relevant organism should be done and appropriate antibiotics should be started (doxycycline with metronidazole for 7–14 days). There is no additional benefit of removal of IUD once PID is being treated with appropriate antibiotics. However, if the infection reoccurs then the device should be removed. Risk of PID is less with newer IUD's (Copper T380 A and LNG IUS).
7. **Actinomycosis.** Actinomyces israelii are commensal bacteria of genital tract. It is a gram positive, slow growing, anerobic bacterium which can rarely cause suppurative infection. They may be identified on cervical smears but are not predictive of disease. IUD users may have a higher risk of infection with actinomycosis (up to 7%). Their presence in a cervical smear in woman with a current IUD requires an assessment to exclude pelvic infection. If there is no evidence of pelvic infection, then routine removal of IUD is not indicated and no antibiotic treatment is needed (ACOG 2017). However, if symptoms of pelvic infection like fever, weight loss, abdominal pain, vaginal discharge or abnormal uterine bleeding develops, then IUD should be removed and a course of antibiotic (penicillin or tetracycline) is given.
8. **Infertility.** It can occur due to pelvic infection and tubal block but is rare. Return to fertility occurs immediately following removal or expulsion of IUD.
9. **Ectopic pregnancy.** Overall IUD users are 50% less likely to have ectopic pregnancy than women using no contraceptive. The incidence of ectopic pregnancy in IUD users is very less varying for 0.25 to 1.5 per 100 women years (only 0.2 for CuT 380 A and CuT 375). However, when pregnancy occurs with IUD in situ, the chance of it being ectopic are much higher ( 6–30% )than in general population (0.5–1.4 %). It is mainly because of pelvic infection especially salpingitis. Avoiding use of IUD in women with previous ectopic pregnancy and with multiple sexual partners can reduce the chance of ectopic pregnancy. Progestasert a progesterone IUD (not in use any more) increases risk of ovarian ectopic pregnancy.
10. **Expulsion.** Expulsion of IUD may be partial or complete and occurs in about 3–8% (average 5%) women in the first year especially in first 3 months of use, mostly during menstruation (especially first menstruation after insertion). Expulsion rate is higher in postplacental insertion, post second trimester abortion insertion, older devices, nulliparous and young women and with faulty technique of insertion. Uterine contractions and disparity in size of device and uterine cavity are responsible for expulsion. Expulsion can be prevented by insertion of IUD by a trained person, with insertion of the device high up in the uterus and keeping the device in the inserter for less than 5 minutes to avoid losing its memory (shape). Women should be advised to check for the presence of IUD threads by inserting a finger in the vagina following menstruation and also check her pads during the menstruation as the device may be expelled. If she cannot feel the thread, she should report to the doctor who sounds the uterus with uterine sound if the thread is not visible on speculum examination. If on sounding, no device is felt inside, then ultrasound pelvis or X-ray abdomen should be done to exclude perforation (see below). In case of expulsion, ultrasound and X-ray abdomen shows negative result for IUD. For a case of expulsion and in absence of PID, another modern IUD (CuT 380A, Cu T 375or LNG IUS) can be inserted. In case of incomplete expulsion, the device should be pulled out with an artery forceps and a new modern IUD is inserted.
11. **Misplaced IUD (missing thread).** In this condition the tail (thread) of the IUD is not seen through the cervix. The causes include:
(*i*) Expulsion of the device
(*ii*) Curling of thread inside the uterus
(*iii*) Tearing and expulsion of the thread
(*iv*) Pregnancy
(*v*) Uterine perforation
Sounding of uterus, X-ray abdomen and/or ultrasound

can localize the IUD. Management of uterine perforation is discussed below

12. **Uterine Perforation.** Perforation of the uterus is a serious but uncommon complication of IUD insertion (less than 1 in 1000) being lower with newer IUDs. The risk of perforation is related to the skill of the healthcare and professional inserting the IUD. Majority of the perforations occur during insertion due to faulty technique or with push out technique (used for Lippes Loop) and is much less if the insertion is performed by skilled healthcare professional using withdrawal technique. Occasionally, the perforation is initiated during insertion (partial perforation) and later completed by uterine contractions (complete perforation). Perforation may not cause any symptoms and may remain unnoticed (more common with inert Lippes Loop). Sharp abdominal pain during IUD insertion, missing thread and vaginal bleeding after insertion are features of perforation. Sometimes IUD may cause cervical perforation which may be partial and the stem of IUD may protrude through the cervix into the vagina causing dyspareuria and the device can be felt by women.

**Diagnosis**

(i) If patient has missed period, urine pregnancy test should be done.

(ii) Speculum and vaginal examinations are performed followed by passage of uterine sound which can detect an IUD if in utero. If probing is negative, the IUD is either expelled out or has perforated the uterus.

(iii) **Ultrasound** (transvaginal and transabdominal) scan is a preferential modality to diagnose whether IUD is in uterus or has perforated and lodged outside in the peritoneal cavity (Fig. 43.13). It can also diagnose partial perforation.

(iv) **X-ray abdomen and pelvis.** Anteroposterior (detects lateral displacement) and lateral (detects anteroposterior displacement) skiagram of abdomen is performed after inserting uterine sound or another IUD in uterine cavity to localize the relative position of perforated IUD in relation to the uterine probe (Fig. 43.14).

(v) **Hysteroscopy** can diagnose a misplaced or partially perforated CuT in the uterine cavity which can also be removed in the same sitting. However, in case of complete perforation, hysteroscopy may be negative.

(vi) **Laproscopy.** It can detect as well as remove a misplaced or perforated IUD from the peritoneal cavity and also diagnose if the device has entered the intestines or other vital structures.

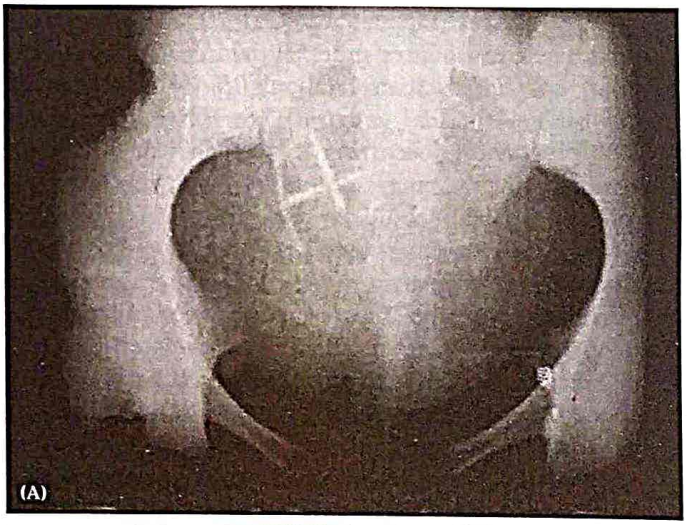
**Management**

(i) **Lippes Loop.** Even though it does not produce reaction in peritoneal cavity due to its inert nature and can be left as such, most women would prefer its removal to avoid injury to intestines and to allay their fears. Its removal can be done by laparoscopy, laparotomy and even by colpotomy.

(ii) **Copper devices.** These devices produce excessive peritoneal inflammation leading to good




**Fig. 43.13:** Ultrasound abdomen and pelvis showing misplaced Copper T



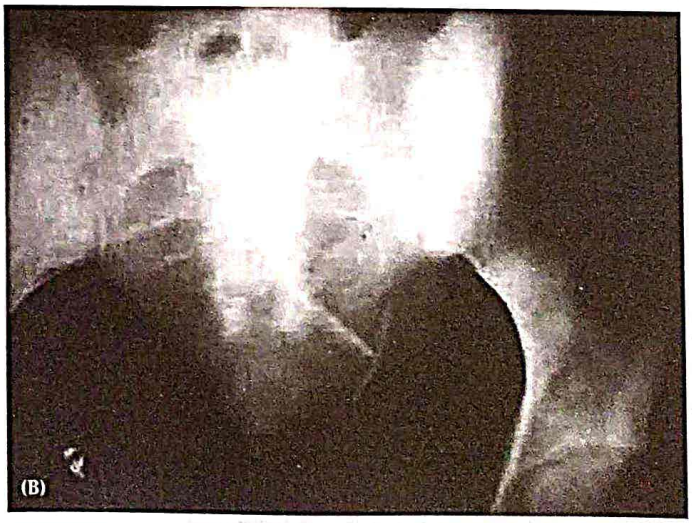




**Fig. 43.14:** (A) Plain X-ray abdomen showing misplaced copper T towards right side; (B) Plain X-ray abdomen and pelvis showing misplaced copper T towards left side

amount of adhesions with omentum, intestines and other adjoining structures. Hence, their removal is mandatory. If the device is inside the uterus, it can be removed by Shirodkar's hook, artery forceps (if the thread is visible) or by uterine curette. Removal under hysteroscopic visualization is the best procedure. For a device partially perforating (some in endometrium and myometrium, rest in peritoneal cavity) removal by both hysteroscopy and laparoscopy may be required. Removal can be done laproscopically by an experienced operator especially if there is no intestinal injury. It usually needs removal by laparotomy especially if intestines are involved. Sometimes, the device may enter small or large intestine necessitating resection anastomosis or colostomy to remove the device. Help of surgeon or gasteroenterology surgeon may be required.

(iii) **LNG-IUS.** If perforated, they can cause intraperitoneal bleeding and should always be removed by hysteroscopy, laparoscopy or laparotomy.

13. **Pregnancy with IUD.** The reported pregnancy rate per 100 women years of use are more than 2 for group I devices (Lippes loop, CuT 200, not used these days), between 1–2 for group II devices (CuT 250, CuT 220c) and is less than 1 (about 0.5 %) for group III devices (0.7 for CuT 380 A, 0.2 for LNG IUS). About 30% pregnancies occurring in women using IUD are ectopic. If pregnancy occurs with device in situ, removal of IUD should be done before 12 completed weeks of gestation whether or not she intends to continue the pregnancy.

Miscarriage is the most common complication of pregnancy with IUD in situ (50–60% miscarriage rate in intrauterine pregnancy against a background risk of about 13%). If women wish and is within legal limits, termination of pregnancy and removal of IUD can be done in the same sitting. If removal is not possible (thread not visible) and women wish to continue pregnancy, she should be informed about the risk of miscarriage, infection, preterm labour, premature rupture of membrane and foetal growth restriction. There is no evidence of higher rate of congenital malformations in the fetus with IUD in situ. Usually after delivery of baby, IUD is expelled embedded in placenta and membranes.

14. **Other Concerns**

(i) **Return to fertility** is very fast with almost 70% women conceiving within 1 year of IUD removal. In a monogamous relationship, there is very little risk of infection and infertility due to IUD. However, there may be risk of infection in case of multiple sex partners or sex workers. In such cases dual contraception (IUD with condom) should be used.

(ii) **Continuation rates of IUD.** Almost 70–90% women continue IUD at the end of 1 year. Abnormal uterine bleeding, pain and pelvic infection are important cause of discontinuation.

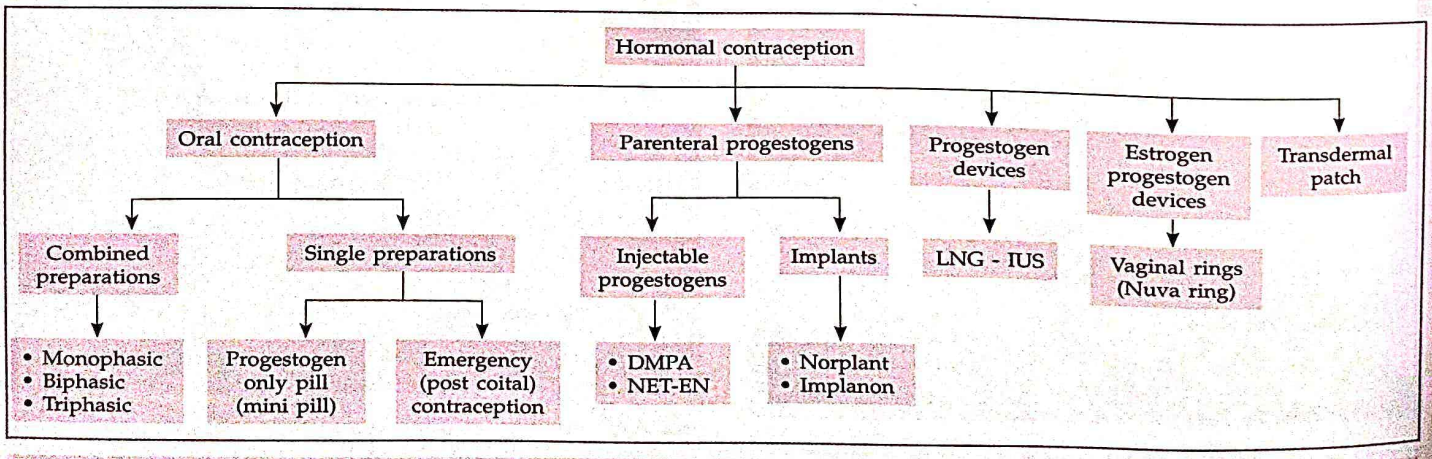
(iii) **Cervical or endometrial malignancy.** There is absolutely no evidence that IUDs promote cervical or endometrial malignancy.

## Indications of removal of IUD

1. Abnormal uterine bleeding not responding to medical treatment.
2. Acute pelvic inflammatory disease (PID)
3. Missing thread
4. Uterine perforation
5. Partial expulsion of device
6. Pregnancy with IUD in situ
7. The woman wants to conceive.
8. After the lifespan of the device
9. One year after onset of menopause.

# HORMONAL CONTRACEPTION

Hormonal contraceptives are the steroidal hormones to prevent pregnancy. They are shown in Flow Chart 43.1.




**Flow Chart 43.1:** Various types of hormonal contraception

## Oral Contraceptives (The Pill)

The oral contraceptive (OC) commonly called **'the pill'** is widely accepted and most effective method of fertility control.

Combined oral contraceptive (COC) pill is combined synthetic estrogen, ethinyl estradiol (EE) and synthetic progestogen, e.g. levonorgestrel.

### History and Development

Gregory Pincus, a biologist along with John Rock and Garcia after field trials in Puereta Rico showed for the first time in 1956 that a pill Enovid containing 10 mg norethynodrel (progestogen) and 0.15 mg of mestranol (estrogen) was an effective contraceptive. It was approved by US FDA in 1959. Later, doses of both progestogen and estrogen were reduced to avoid side effects without reducing efficacy.

Three types of pills are now used in contraception.

1. Combination pills containing both estrogen and progestogen.
2. The sequential regimen is estrogens for first 16 days and progestogen added in the last 5–6 days.
3. Progestogen only pill is also known as 'mini pill'.

### Generation of Pills

1. **The first generation** oral contraceptives contain 50 µg or more of ethinyl estradiol.
2. **The second generation** oral contraceptives contain 30–35 µg of ethinyl estradiol with progestogens (progestin) such as levonorgestrel, norgestimate and other members of the norethindrone family (e.g. Mala N, Mala D). Students can remember Mala N (No cost, free by Govt.), Mala D (Decreased cost, subsidized by Govt.)
3. **The third generation** oral contraceptives contain desogestrel or gestodene with 20–30 µg ethinyl estradiol (e.g. Femilon, Loette). Minesse contains only 15 µgm ethinyl estradiol. One preparation contains cyproterone acetate (2 mg) with ethinyl estradiol (Diane 35, Krimson 35) and is useful in patients with acne and hirsuitism.
4. **The fourth generation pills**. The estrogen content is like third generation pills but progestogen is either drospirenone, dienogest or nomegestrol. The fourth generation pill available in India contains ethinyl estradiol and drospirenone (progestogen) with weak anti-mineralocorticoid activity and is useful in patients with polycystic ovarian disease (PCOD) e.g. Yasmin, Yaz, Dronis. It does not cause weight gain. Infact, women may lose some weight on it due to its mineralocorticoid activity. It makes the skin of women smooth and they may look better. Another preparation contains ethinyl estradiol and dienogest (freedase 30) is available in India now.

### Mechanism of Action

Oral pills act in the following ways.

*1. Ovulation suppression*

Ovulation suppression is the most important effect. Both the hormones prevent ovulation by suppression of hypothalamic gonadotrophin releasing factors which in turn prevents pituitary secretion of follicle-stimulating hormone (estrogen effect) and luteinizing hormone (progestogen effect). The absence of peak release of FSH and LH from the anterior pituitary oral pills inhibit follicular growth in the ovary.

*2. Endometrial Effect*

Oral pills (especially progestogen) cause regression and atrophy of endometrial glands, decidualization and edema of endometrial stroma making the endometrium unfavourable for implantation.

*3. Cervical mucus changes*

Progestogens thicken the cervical mucus and make it viscous and scanty with low spinnbarkeit and poor ferning inhibiting sperm passage.

*4. Alteration of ovum transport*

Alteration of ovum transport by interfering with motility and secretions of the fallopian tubes.

*5. Acceleration of luteolysis*

Even though accidental breakthrough ovulation occurs, the other mechanisms prevent conception. Apart from their anovulatory action, estrogens stabilize the endometrium to prevent breakthrough bleeding which gives better cycle control.

The net effect of estrogen and progestogen is extremely effective ovulation suppression, inhibition of sperm migration through cervical mucus and creation of an unfavourable endometrium for implantation providing almost absolute protection against conception when taken daily for 3 weeks in a month. **In a nutshell, combined oral pills prevent ovulation** (students should answer this in examination).

### *Effectiveness*

Oral pills are the most effective method of contraception if used correctly and consistently with failure rate being only 0.1 to 1 per hundred women years in first year. Typical failure rate is 1.8 HWY which may increase to 8 HWY for improper use. The failures are mostly due to missed pills, delay in starting the next course and stoppage of the drug due to side effects or fear without resorting to other contraceptive measures.

### *Indications and Contraindications*

Indications and contraindications of OC pills are shown in Table 43.2.

### Advantages (benefits) of combined oral contraception

1. Most important benefit is very effective contraception.
2. Easily reversible
3. Effective at all ages
4. Not coitus dependent
5. Simple and attractive
6. No effect on future fertility. Whenever women want

**Table 43.2: Medical Eligibility Criteria for Initiation and Continuation of Combined OCS/Combined Injections/ Transdermal patches and Vaginal Rings (WHO 2017)**

**WHO Category 1: No Restriction of Use (Freely Used) (Indications)**

1. Age: Menarche to 35 years
2. Parity: Nulliparous/parous
3. Postpartum: Non-breastfeeding ≥ 21 days
4. Postabortion: 1st/2nd trimester, immediate post abortion
5. Past ectopic pregnancy
6. Cardiovascular disorders
   i. Varicose veins ii. Minor surgery

**Indications (WHO Category 2: Advantages Outweigh Risks): Can be used with caution**

1. Age: > 35 years
2. Obesity: More than 90 kg weight
3. Cardiovascular disorders
   i. History of hypertension
   ii. Family history of deep vein thrombosis (DVT)/ pulmonary embolism (PE)
   iii. Superficial thrombophlebitis
   iv. Uncomplicated cardiovascular disease
   v. Major surgery without prolonged immobilization

**Relative Contraindications to COC (WHO Category 3: Risks Outweigh Advantages): Avoid use**

1. Age: ≥ 35 years having < 35 cigarettes/day
2. Postpartum:
   i. Breastfeeding women ≥ 6 weeks to < 6 months
   ii. Non-breastfeeding women < 21 days
3. Hypertension
   i. Adequately controled BP
   ii. Systolic 140–159 or diastolic 90–99
4. Hyperlipidemia
5. Diabetes
6. Nonfocal migraine

**Absolute Contraindications to COC (WHO Category 4: Not to be used)**

1. Age: > 35 years taking ≥ 35 cigarettes/day
2. Postpartum: Breastfeeding women < 6 weeks' postpartum
3. Cardiovascular disorders
   i. Hypertension (systolic ≥ 160 or diastolic ≥ 100)
   ii. Deep vein thrombosis/pulmonary embolism (DVT/PE)
   iii. Ischemic heart disease
   iv. Severe or multiple risk factors for venous or arterial disease
   v. Atherogenic lipid disorders
   vi. Significant structural heart disease
   vii. Pulmonary hypertension
4. Neurological disorders
   i. Focal migraine
   ii. Transient ischemic attacks
   iii. Past cerebral haemorrhage
   iv. Vascular malformations of the brain
5. Multiple disorders (old age, smoking, diabetes, hypertension)
6. Rare factors (past thrombin mutations, Protein S, Protein C, antithrombin deficiencies)
7. Major surgery with prolonged immobilization

more children, regular ovulation returns within 3 months on stopping the pill in 98% cases.

### Non-contraceptive benefits of oral pills

The non-contraceptive benefits of oral pills are given in Table 43.3.

### Minor side effects

Oral pills produce several metabolic, functional and organic changes (0.5–15%). Side effects are more common in first few cycles and fall down to insignificant levels after the third cycle of continued use. The majority of the side effects are related to estrogen/progestogen balance of the tablet and can be reduced by prescribing a pill with different balance. Thus, women having estrogenic side effects can be offered a pill with lower dose of estrogen (20 µg) while women with progestogenic side effects can be prescribed a newer progestogen like drospirenone (Yasmin). The minor complications or ailments are as follows:

1. **Gastrointestinal upset.** Due to estrogenic effect, OC pills may cause nausea, retching emesis and anorexia. These symptoms are temporary and settle down with 2–3 months of continued use of OC pills. They are less with low dose and triphasic pills. They can be reduced by taking pills at bed time, with milk or by taking triphasic pills.
2. **Headache and migraine.** These are estrogenic side effects which settle down with continued usage.

**Table 43.3: Non-Contraceptive Benefits of Oral Contraceptives**

1. Reduced menstrual blood loss by up to 50% by controlling both heavy menstrual bleeding (formerly menorrhagia) and intermenstrual bleeding (formerly metrorrhagia) and prevention of anaemia.
2. Stabilization of menstrual cycle
3. Improved dysmenorrhea from endometriosis and other causes.
4. Treatment of pre-menstrual syndrome
5. Treatment of Mittelschmerz's syndrome (mid-cycle ovulation pain)
6. Reduction in pelvic inflammatory disease by 50% (due to thick cervical mucus) and reduced uterine motility
7. Decreased risk of ectopic pregnancy
8. Protection against endometriosis and treatment of pelvic pain due to endometriosis
9. Protection against fibroid uterus (30%) and treatment of bleeding due to leomyomas
10. Increased bone density and prevention of osteoporosis
11. Reduction in various benign breast diseases (50–70%)
12. Improvement of acne
13. Inhibition of hirsutism progression
14. Improvement in rheumatoid arthritis
15. Prevention of functional ovarian cysts (50%)
16. Protection against cancers like endometrial cancer by 50% (for 15 year), ovarian cancer by 40%, colorectal cancer by 40% and choriocarcinoma.

However, persistent and severe migraine necessitates their discontinuation.

3. **Effect on weight.** While estrogens produce edema and cyclical weight changes, progestogens have anabolic effect causing fat and tissue deposition and increased appetite.There may be edema feet due to sodium retention. Newer pills containing drospirenone progestin (Yasmin) do not cause any weight gain or may even cause slight weight loss due to their anti-mineralocorticoid activity.
4. **Skin changes.** There can be hyperpigmentation of face and forehead due to estrogen. There can also be acne and oily skin due to androgenic effects. Use of low dose and triphasic pills cure these effects.
5. **Breast changes.** The estrogens in OC pills can produce edema of breasts causing heaviness and tenderness. These symptoms tend to settle down with continuous use and wearing of tight brassieres. On positive side, OC pills reduce incidence of benign breast diseases due to progestogens.
6. **Excessive vaginal discharge.** Excessive vaginal discharge or leucorrhea can be due to cervical hypertrophy and congestion and cervical erosion due to estrogenic effect. Vaginal candidiasis is more common in women on oral pills. Excessive vaginal discharge may be due to Trichomonal vaginitis or due to sexually transmitted infections like Chlamydia especially if the women has more sexual partners. Concomitant use of condoms is helpful to prevent infection. Pelvic and vaginal infections should be treated.
7. **Breakthrough bleeding.** It is a condition of bleeding in between periods due to lower estrogen levels and is more common with low dose pills (20 µg estrogen) or triphasic pills. It is not harmful and settles down with time. The women should be counselled about its harmless nature. Changing to 30 µg estrogen OC pill can treat the condition.
8. **Irregular bleeding and menorrhagia.** These can sometimes occur due to low hormone levels. Continuous use of pills especially 30 µg estrogen or use of long term continuous OCs like seasonale can be useful in these women. Usually OC pills reduce amount of blood loss.
9. **Oligomenorrhea.** It is more common with low dose (20 µg) estrogen pills due to lack of proliferation of endometrium and regression of endometrium due to progestin effects. It is less with triphasic pills. It needs only reassurance and is not harmful but rather prevents anaemia in Indian women. However, if women are worried then they should be shifted to 30 µg estrogen pills or triphasic pill or can use IUD.
10. **Amenorrhea.** It is often transient and is not harmful. She should be counselled about it. If she misses only one period without missing pills during the cycle, she can continue with pills of next cycle. If she missed few pills along with missing the period, urine pregnancy test should be performed to rule out pregnancy followed by new pack of OC pills in case of negative test. Amenorrhea continuing for few months is due to endometrial hypoplasia caused by OC pills and should be managed like oligomennorhea discussed above. Shifting to triphasic pills usually is effective. Persistence of amenorrhea for 6 months or more may be observed in 1 in 100 women on long term pills use and in women who used to have menstrual dysfunction before starting the pills. It is named as **post pill amenorrhea** and should be investigated and treated like a case of secondary amenorrhea. OC pills should be stopped. There may be microadenoma of pituitary needing treatment with bromocriptine or cabergoline.
11. **Psychosexual problems.** Most women feel good on OC pills and have better sexual life due to loss of fear of pregnancy. However, sometimes pill users may have depression and decreased libido due to psychological factors or as a side effect of progestin causing vaginal dryness. Prescription of daily 50 mg of pyridoxine tablet for few months may be useful.
12. **Miscellaneous side effects** like hyperlipidemia, muscle cramps and leg cramps and vision dimness due to edema of cornea may occur occasionally. Persistent corneal edema and hyperlipidemias usually necessitate stoppage of OC pills.

### Major side effects and risks

Most of the risks of OCs were reported in 1960s and 1970s when pills with high estrogen content were used. Low dose OCs and new progestins are much more safe.

#### I. Cardiovascular diseases

1. **Myocardial infarction** especially in heavy smokers, hypertensive, diabetic or obese women.
2. **Ischemic stroke.** The relative increased risk in pill users is about 1.5 times as compared with non-users being more in smokers and in hypertensive women.
3. **Hemorrhagic stroke.** In hypertensive women with smoking and more than 35 years of age, risk increases to 10 times.
4. **Hypertension (Estrogen).** Up to 1% risk through renin-angiotensin system. Low dose pills have got less risk. The changes tend to revert to normal 3–6 months after stoppage of pill.
5. **Vascular complications.** (a) **Arterial thrombosis.** Low dose pills do not increase the risk of arterial thrombosis in the low risk women. (b) **Venous thromboembolism** (VTE). It includes deep vein thrombosis (DVT), pulmonary embolism and cerebral venous sinus thrombosis. Use of combined oral contraception is associated with an increase risk of VTE 3–5 times more than non OC users. Estrogens may cause vasculitis, increased coagulability and thrombosis in susceptible individuals possibly by its effects on cortisol binding. The absolute risk is small increasing from 5 per 100,000 women year in non OC pill users to 15 per 100,000 women years in users of combined OC pill containing norethisterone or levonorgestrel (3 fold increase) rising to 25 per 100,000 women years in women taking OC pills containing gestodene or

desogestrel (5 fold increase). However, one must not forget that pregnancy is associated with highest risk of VTE with incidence being 60 per 100,000 women years (12 fold increase). Women with positive thrombophilia risk like carrier of factor V Leiden mutation are at increased risk of VTE but its routine screening is not recommended before starting COCs in general population. Smoking, obesity, hypertension, diabetes are other high risk factors.

6. **Lipid profile.** There is no data to suggest that low dose pills cause significant adverse effect on lipid profile.

### II. OC pills and cancers

**Oral pills decrease three cancers (endometrial, ovarian and colorectal) and increase two cancers (breast and cervical).**

#### *1. Breast Cancer and Oral Pills*

Breast cancer is the most fearful complication of oral pills in the mind of patients and doctors. Data from large studies is reassuring. There is a small increase in the risk in current users of the pill **(relative risk 1.24)** which gradually disappears over 10 years after discontinuation. It is usually localized in the breast and has better prognosis.

#### *2. Cervical Cancer*

Most studies did not find an increased chance of cervical cancer in OC pill users. However, some studies observed a slight increase (1.3 to 1.8 fold) in the risk of cervical cancer with COC use of more than 5 years. It could be due to lack of use of condoms with COC or presence of multiple sexual partner increase the risk of HPV infection which is a high risk factor for cervical cancer and CIN. Hence, regular cervical smears are recommended to these women along with advice for safe sexual practices and regular condom use.

#### *3. Liver Cancer*

Slight increase in incidence of liver adenoma has been reported in women on CoCs (1–2 per 100,000 users). Earlier studies observed a slight rise in liver cancer risk with more than 8 years use of OC pill (1 per 100,000). The risk is slightly higher in the developing countries due to risk of associated hepatitis B virus infection.

#### *4. Endometrial Cancer*

COC use protects against carcinoma endometrium. Using COC for more than 12 months causes 50% reduction in the risk.

#### *5. Epithelial Ovarian Cancer*

There is 40% risk reduction in epithelial ovarian cancer by COC use due to suppression of ovulation.

#### *6. Colorectal Cancer*

It is decreased by use of COCs.

### III. Effect on Liver and Gall Bladder

There may be slight increase in risk of cholestasis and cholestatic jaundice in COC users. Slight increasd risk of cholelithiasis and cholestatic jaundice due to bile stasis in gall bladder may be the cause. OC pills should be avoided in those who have had hepatic dysfunction or recent jaundice but can be given 6 months after recovering from viral hepatitis.

### IV. Metabolic Effect

There is no evidence of increased risk of clinical diabetes in COC users. Low dose pills or triphasic pills should be given to diabetic woman under supervision.

The effect on the lipid profile depends on the relative doses of estrogen/progestin and the type of progestin. The level of sex hormone binding globulin (SHBG) is increased.

### V. Effects on reproduction

In more than 90% cases ovulation usually returns within 3 months of stopping the drug.

### VI. Risk to fetus

There is no extra risk of significant congenital malformation in fetus when COC is taken during early pregnancy inadvertently. Risk of congenital malformation is about 2–3% (same as in general population).

### VII. Lactation (estrogen + progestin)

Combined oral pills decrease production and the quality of the milk (reduction of protein and fat content). OC pills are otherwise not harmful to the infant. Progestogen only pill is a better alternative for the lactating mothers.

### Patient Selection

OC pills are most suited for the following classes of patients.

1. Young adolescents in unstable relationships.
2. Recently married women desirous of postponing their first pregnancy.
3. Women desirous of spacing their pregnancies.
4. Women with endometriosis.
5. Women in need of short-term or long-term reversible contraception.
6. Women suffering from acne.
7. Those suffering from menorrhagia, polymenorrhea and dysmenorrhea.
8. Postpartum or postabortal contraception
9. Family history of ovarian cancer.
10. Women with polycystic ovarian disease or repeated anovulatory cycles.
11. Perimenopausal women

Thus, any women of reproductive age group without any systemic disease and contraindications listed is a suitable candidate for combined pill therapy. **Growth and development of the pubertal, sexually active girls are not affected by the use of 'pill' as was once thought.** Hence, they can be given to pre-pubertal girls also, if needed.

### Patient Work-up Prior to Prescribing OC Pills

Though patients work-up prior to prescribing OC pills is not mandatory in all cases, the following guidelines should be fulfilled.

1. Document detailed personal and family history and record weight, blood pressure, physical examination findings including breast examination and pelvic examination. A Pap smear should be taken at the first examination if not done before.
2. Repeat these annually.
3. A mammography is recommended in all women aged 35 years or above, in women with family history of breast cancer in first-degree relatives or whenever a breast examination reveals suspicious findings on clinical examination.
4. Laboratory tests should include: (*i*) complete blood count (*ii*) lipid profile and (*iii*) fasting and postprandial blood sugar levels.

### Instructions for Starting OC Pills

Any brand from Table 43.4 can be started. Some common brands of COC in common use are shown in Fig. 43.15 while some low dose pills (EE 20 µg) are shown in Fig. 43.16. Fig. 43.17 shows how to start the pill pack. The following should be explained to patients. Schedule options for starting OC pills.

1. Immediately after a first trimester abortion.
2. One week after a second trimester miscarriage of 13–28 weeks.
3. Six to eight weeks after delivery
4. Start the new pill pack on the first day of menses or on the first Sunday after start of menses. This assures the patients of a menses-free weekend, a convenience for individuals with outdoor lifestyles but requires use of backup contraception for one week.
5. The pill should be taken everyday at the same time without fail like before retiring to bed, after brushing teeth or removing contact lenses.
6. Call the patients for follow-up after three months to resolve their doubts and to assess how they have been tolerating the pill. Many minor side effects resolve with passage of time. Thereafter, the patients should be asked to come for follow-up after a year or must report earlier in case of any complications.
7. Annual check-ups are important for long-term monitoring of patients' well-being.

### Disadvantages and precautions of combined oral pills

Disadvantages and precautions of combined oral pills are given in Table 43.5.

### Pharmacology

Almost all oral pills now contain estrogen as ethinyl estradiol (mestranol is almost obsolete).

Almost all currently available progestins are 19-nortestosterone derivatives and one is an aldosterone derivative. Although individual progestins are initially chosen because of their progestogenic potencies, they are often compared and prescribed based on their presumed estrogenic, anti-estrogenic and especially their androgenic effects. The various popular OC pills with formulation and dosage and cost available in India are shown in Table 43.4.

(A)




(B)

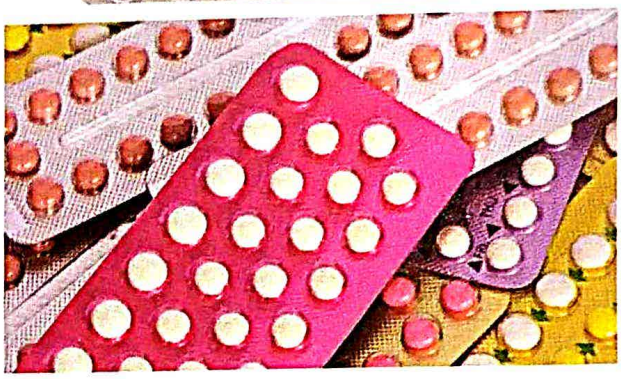


**Fig. 43.15:** (A) Actual photograph of oral pills supplied by the Government of India (B) Actual photograph of some commonly used standard combined oral contraceptive pills




**Fig. 43.16:** Actual photograph of low dose pill (ethyl estradiol 20 µg) elogen.




**Fig. 43.17: A.** Actual photograph of a packet of oral pills (MALA-N) with arrows demonstrating sequence of how to take pills from the packet.

**Table 43.4: Current Common COC Pills Available as Contraceptives in India**

| Name of Pill | Formulation | Tablet/Packet | Cost in Rupees (1 NR) |
|---|---|---|---|
| 1. Mala N (Govt. of India) | Levonorgestrel 0.15 mg + EE 30 µg | 21 hormone tab. plus 7 iron tab. – 28 tab. | Free of cost at Govt. hospitals (N = No cost) |
| 2. Mala D (Govt. of India) | D-levonorgestrel 0.15 mg + EE 30 µg | 21 hormone tab. plus 7 iron tab. – 28 tab. | Low ₹ 3 subsidized by Govt. (D = Decreased cost) |
| Available at National Family Welfare Programme and at commercial market. Rest COCs are available at commercial market. | | | |
| 3. Ovral G used for menorrhagia and contraceptive in women needing high estrogen (e.g. women on antituberculous or antiepileptic drugs) | Norgestrel 0.5 mg, EE 50 µg | 21 tablets | ₹ 50 |
| 4. Ovral L | Levonorgestrel 0.15 mg, EE 30 µg | 21 tablets | ₹ 44 |
| 5. Novelon | Desogestrel 0.15 mg, EE 30 µg | 21 tablets | ₹ 69 |
| 6. Loette | Levonorgestrel 0.10 mg, EE 20 µg | 21 tablets | ₹ 61 |
| 7. Femilon | Desogestrel 0.15 mg, EE 20µg | 21 tablets | ₹ 75 |
| 8. Elogen | Desogestrel 0.15 mg, EE 20 µg | 21 tablets | ₹ 80 |
| 9. Triquilar Triphasic with three incremental hormones | (a) Levonorgestrel 0.05 mg, plus EE 30 µg<br>(b) Levonorgestrel 0.075 mg, plus EE 40 µg<br>(c) Levonorgestrel 0.125 mg, EE 30 µg | 21 tablets<br>6 tablets (day 5–10)<br>5 tablets (day 11–15)<br>10 tablets (day 16–25) | ₹ 38 |
| 10. Yasmin | Drospirenone 3 mg + EE 30 µg | 28 tablets<br>21 hormone tablets plus 7 inert tablets | ₹ 300; It does not cause weight gain and is good for skin. |
| 11. Yaz, Dronis 20 | Drospirenone 3 mg + EE 20 µg | 28 tablets<br>24 hormone tablets plus 4 inert tablets | ₹ 250; Same as above |
| 12. Qlaira: New quadriphasic (4 phasic) COC pills | Dienogest + Estradiol valerate<br>The tablets are to be taken as specification pack | 28 tablets pack<br>2 Dark yellow tablest contain 3 mg estradiol<br>5 medium red tablets contain 2 mg estradiol and 2 mg dienogest<br>17 light yellow tablets contain 2 mg estrdiol and 3 mg dienogest<br>2 dark red tablets contain 1 mg estradiol<br>2 inactive white tablets | Not yet available in India. |
| 13. Minesse | 15 µg EE + 60µg gestodene | | |

## Counselling

1. Avoid smoking
2. Encourage women at risk of STD to additionally use the condoms (dual contraception).
3. Explain about back-up method of contraception in case it becomes necessary because of missed pills, vomiting or diarrhea undermining pill effect, travel schedules, upsetting timing of pill intake etc.
4. **Management of missed pills.**
   (i) Missed 1 or 2 pills or started new pack 1–2 days late: Take hormonal pills as soon as possible.
   (ii) Missed ≥ 3 pills in first and second week or started new pack ≥ 3 days late: Take hormonal pills as soon as possible. Use back up method (e.g. condom) for 7 days.
   (iii) Missed ≥ 3 pills in third week: Take hormonal pill as soon as possible. Skip non-hormonal pill. Start new pack without break. Use back up method (e.g. condom) for 7 days.
   (iv) Missed non-hormonal pill: Keep taking oral pills. Discard missed pill. Start new pack as usual.
   (v) For severe vomiting or diarrhoea: If vomits within 2 hours of taking pill, take another pill. If vomiting for more than 2 days, continue

**Table 43.5: Advantages and Disadvantages of Combined Oral Pills**

| Advantages | Disadvantages |
|---|---|
| 1. Most effective method of contraception if used properly. (Failure rate is 0.1/HWY). The women feels reassured of their efficacy and can plan her career indirectly helping her progress in life as has happened in West. | 1. They require counselling and strong motivation as most women have heard stories about their side effects and complications. |
| 2. Excellent cycle control with good bye to irregularity of periods helping her to plan her life engagements better. | 2. They require initial history taking and examination to find-suitability and need regular follow-up for any complications. |
| 3. OC pills are usually well tolerated in most women. | 3. Women have to remember to take tablet every day. Many Indian women may not remember. Missing pills is common lowering their efficacy. |
| 4. There are many non-contraceptive benefits to the women like treatment of menorrhagia, dysmenorrhea, improvement of acne, treatment of anaemia, reduced incidence of ovarian, endometrial and colorectal cancers. | 4. Risk of drug interactions with anti-tuberculous, anti-epileptic drugs and antibiotics when their efficacy may be reduced. |
| 5. Modern low dose pills with newer progestins further reduce the side effects, make skin better looking with no gain in weight. | 5. Cannot be used by all women with many contraindications, including lactating mothers, thromboembolism, medical disorders. |
| 6. They have immediate reversibility on stopping the pill and women can conceive quite quickly. | 6. Modern pills are expensive (Yasmin ₹ 350 per month). However, (Mala-N) is provided free of cost through government centres. |
| 7. The oral pills have provided empowerment to women. They can now decide when to have a baby. Infact invention of oral pills has been hailed as one of the most important invention of 20th century. | 7. In selected women they can have side effects and complications, some of which can be serious and life threatening (eg. thromboembolism). They do not protect against PID. |

hormonal tablet, skip non-hormonal pills: Use backup method (e.g. condom) for 7 days.

5. Warn about side effects like nausea and vomiting, breast discomfort, dry vagina, etc. Women complaining of nausea should be advised to take the tablet soon after the night meal, assure the patient that this symptom tends to settle down after a few cycles. Always rule out the possibility of pregnancy when counselling an OC pill user complaining of severe nausea.
6. To report potentially serious side effects like headaches, chest pain, eye or speech problems.
7. To report missed menses. If no pill was missed contact the doctor, further action depends on whether the patient has experienced such episodes of missing menses earlier whilst on OC pills and the degree of concern about pregnancy, advise a urine pregnancy test, if negative she should start the next pack at the usual time.

### Drug Interactions

Many drugs interact with OC pills and may reduce their efficacy. The women should be informed about it especially if they are on long term medication. Barbiturates, sulfonamides, rifampicin, anticonvulsants, certain antifungal drugs (griseofulvin and ketoconazole) stimulate hepatic enzymes hastening the metabolism of OC pills thus reducing their efficacy. In such cases higher dose of OC pills containing 30–50 µg of ethinyl estradiol may be used or non-hormonal methods of contraception (eg. IUD) may be used. Women taking broad spectrum antibiotics (chloramphenicol, tetracycline, ampicillin, amoxicillin, doxycycline) for some illness should use additional barrier method during this time as they may reduce absorption of ethinyl estradiol from intestines. However, many studies have found OC pills to be equally effective in women taking antibiotics. OC pills may interfere with anti-diabetic drugs requiring additional dose of the later and may enhance the effect of steroids. Ascorbic acid enhances the efficacy of OC pills.

### Warning features of OCs and indications for stopping the pill

Most women take the OCs without much problem, However, serious adverse events can happen in less than 1 per 100 women years which indicate stopping the pill immediately and consulting the physician to tackle their medical problem and offer them the alternative effective method of contraception (eg. IUD or barrier method). The various indications of seeking urgent medical attention and stopping the pill are shown in Table 43.6.

## Follow-Up of OC Users And Duration of Use

Scheduled return visits are not mandatory if the woman is fine and does not develop any problem mentioned in Table 43.6. Many clinicians first examine them after 3 months of use and then every 1–2 years for any complication, checking of blood pressure, heart, chest auscultation, breast examination, gynecological examination and cervical smear whenever possible.

If there are no contraindication for continuing the pill (eg. obesity with age more than 35 and smoking), the pill can be continued up to the menopause. However, in Indian

**Table 43.6: Indications For Stopping the Pill**

1. Severe and throbbing unilateral headache (severe migraine) with or without scotoma.
2. Chest pain with or without dyspnea (may indicate pulmonary embolism)
3. Visual problems like double vision, blurring of vision or blindness.
4. Acute and severe pain and swelling in one leg (may indicate deep vein thrombosis)
5. Jaundice (yellowness of sclera and skin)
6. Excessive depression
7. Excessive weight gain
8. Missing two consecutive menstrual periods
9. Excessive white or foul smelling vaginal discharge mixed with blood
10. Post coital or persistent abnormal uterine bleeding after 3 months of pill use
11. Six weeks before major operation
12. Women wanting to conceive

conditions it is better to use them for postponement of first pregnancy and for spacing up to 5 years. After completion of family, permanent method is desirable.

## Multiphasic Oral Pills

These pills were developed to decrease the total monthly consumption of hormones (estrogen and progestogens) but maintaining the efficacy equivalent to COCs. They are of two types.

1. **Biphasic Pill.** It contains constant dose of 35 µg of ethinyl estradiol with lesser dose of progestogen for first 10 days followed by higher dose for the next 11 days. However, because of their higher failure rate, they did not become popular and are not available in India.
2. **Triphasic pills.** They are popular and available in most countries including India. The triphasic pill available in India is Triquilar containing varying amount of estrogen ethinyl estradiol (EE) and gradually increasing dose of progestogen levonorgestrel (LNG) for equivalent efficacy and to prevent breakthrough bleeding. The first 6 days tablets contain 30 µg EE with 50 µg LNG, next 5 days tablets contain 40µg EE with 75 µg LNG while the last 10 days tablets contain 30 µg EE along with 125 µg LNG. The 21 days tablets are followed by one pill free week just like any standard COC for withdrawal bleeding. They have lesser adverse effects on carbohydrate and lipid metabolism and have better cycle control.
   However, they are more expensive and end up in supplying higher amount of estrogen than low dose pills and are now less commonly used.

### New formulations and OCs of the future

Attempts are being made to find out newer agents which can be used effectively and conveniently with less side effects and risks.

*1. Continuous use OCs*

Continuous use of monophasic pills can reduce the number of menstrual cycles and the side effects including migraines, headaches, pre-menstrual syndrome and mood changes.

One formulation, **Seasonale** available in western countries is US FDA approved. It contains 0.15 mg levonorgestrel and 30 µg EE (same doses as those of low-dose pills). Women take a pill every day for 84 days (12 weeks) and then take hormone-free pills for 7 days. This results in 4 withdrawal bleeds a year rather than 12. Seasonale has become a popular method of contraception in recent years in western countries and no extra changes in lipid profile and endometrium have been found so far. **Seasoniqe** is also (84/7day tablet) containing LNG 0.15 mg and EE 30 µg followed by 10 µg EE in 7 days.

**Femcon Fes.** New chewable spear mint flavored tablet containing EE 35 µg + Norethindrone, 0.4 mg (21 days). Placebo contains 75 mg ferrous fumarate. It is useful for those women who cannot swallow pills.

*2. Centchroman*

Centchroman (ormeloxifene) developed by the researchers at Central Drug Research Institute, Lucknow, India is a non-hormonal, synthetic-chemical, once-a-week OC. It is available freely in the Indian market at a relatively low cost in the name of 'Centron' and 'Saheli' containing 30 mg of centchroman. The recommended dose is one tablet twice weekly starting from the 1st day of menses for the first 3 months and then once a week irrespective of menstruation. Ministry of Health and Family Welfare, Govt. of India launched Mission Parivar Vikas on 5th Sept. 2017 by including centchroman in the National Family Planning under the brand name **Chhaya** and is providing it to suitable women free of cost. It is effective reversible method of contraception, is safe for all age women, safe for breast feeding and return to fertility is prompt after stoping the pill. Aim is to decrease total fertility rate to 2.1 by 2025 and fulfill contraceptive demand to 74% by 2020.

Chhaya is non-hormonal non-steroidal once a week contraceptive pill. It does not prevent ovulation. It prevents implantation of fertilized ovum in the uterus and causes endometrial changes. It is available free of cost under Govt. of India **'Hum Do'** scheme. Take one pill (30 mg) twice a weak for first 3 months. From 4th month take one pill once a week on the first pill day. The first pill can be taken on the first day of the menstrual cycle or any other day provided pregnancy has been ruled out.




**Fig. 43.17: B.** Actual photograph of Chhaya (Centchroman) provided free of cost by Govt. of India

**Centchroman acts by altering the endometrium and thereby preventing implantation.** The side effects include headache, nausea, vomiting and weight gain. It does not offer protection against STDs. The drug is contraindicated during lactation in women with allergic diathesis, those suffering from polycystic ovaries, hepatic dysfunction and cervical dysplasia. Return of fertility is often delayed.

Overall results indicated that the centchroman oral pill users had complete satisfaction and reported no side effects. The pill was considered 99% effective, with a mean duration of use as 11 months. However, more trials are needed to know more about this OC. **It has also been used for management of heavy menstrual bleeding (formerly menorrhagia) and fibroids in double the dose with same schedule.**

*3. Antiprogesterone RU-486 (mifepristone)*

It is already used for inducing abortion with prostaglandins like misoprostol and for postcoital contraception. Administration of RU-486 either continuously or at regular intervals (e.g. weekly) is likely to have a contraceptive effect by suppressing ovulation. It has potential for use as a once-a-month pill during the luteal phase and is now being tried.

## Injectable Combination of Estrogen and Progestogen

The mode of action, indications and contraindications of injectable combination of estrogen and progestogen are similar to COCs. This combination is given to overcome the problem of irregular menstrual bleeding.

(*i*) **Cyclo-provera or cyclofem.** This consists of 25 mg depot medroxyprogesterone acetate and 5 mg estradiol cypionate.

It is administered monthly as a deep intramuscular injection.

(a) This method is as effective as Depo-Provera, but is not associated with menstrual irregularity.

(b) The method is rapidly reversible.

(*ii*) **Mesigyna (Norigynon).** It contains norethindrone enanthate 50 mg plus estradiol valerate 5 mg (given once a month intramuscular infection). Both cyclofem and mesigyna are highly effective with pregnancy rate at 1 year of use being only 0.2–0.4 per hundred women years.

## Vaginal Rings

In order to reduce the side effects of systemically administered hormonal pills and to overcome the surgical procedures required for the use of implants, silastic vaginal rings have been developed. These rings are 54 mm in diametrer and 4 mm in thickness.



**Fig. 43.18:** Actual photograph of Nuva ring

**Nuva Ring** (Fig. 43.18). It is now available in India. It is a soft vaginal ring releasing 15 µg ethinyl estradiol and 120 µg etonorgestrel. Women wear it for 3 weeks in a month and then remove it for a week when they have the withdrawal bleeding. A new ring is then used in next month. It works like COC with similar indications and contraindications. Failure rate after perfect use is 0.3% but after typical use is 8%. Its not suitable in prolapse. Side effects are less and are headache, leucorrhea, breast tenderness and nausea. It is expensive.

*Advantages*

1. Self-insertion and removal
2. Other advantages of estrogen progestin contraception hold true.

*Disadvantages*

1. A few patients complain of local discomfort and irritation.
2. Expulsion can occur.
3. Failures have been reported.
4. Side effects of estrogen progestin have been reported by a few users.

*6. Skin Patches* (Fig. 43.19)

Hormone patches that elute 150 µg of progestogen (norelgestromin) and 20 µg of EE have been developed for contraception. Ortho Evra is one such patch. Each patch lasts for 1 week, three patches are required per cycle followed by a 1 week patch free interval. These patches are applied over the buttocks or abdomen, but should not be placed over the breasts. They are not yet available in India. Failure rate is 1/HWY. **Twirla** is once weekly patch releasing 30 µg ethinyl estradiol and 120 µg of levonorgetrel per day. It is applied once weekly for three weeks with no patch fourth week and uses skinfusion technology.

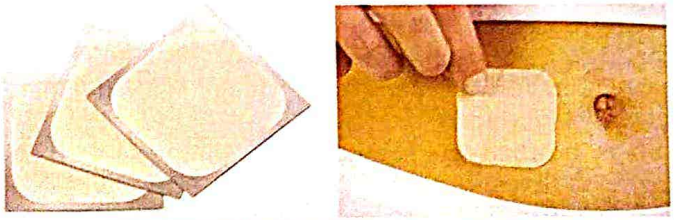


**Fig. 43.19:** Actual photograph of contraceptive patch—Ortho Evra

*Disadvantages*

1. Breakthrough bleeding
2. Allergic skin reactions
3. Mastalgia
4. Headache and nausea
5. Expensive

## Progestogen (Progestin) Only Pill (POP) (Fig. 43.20)

*Types*

Each tablet contains one of the following progestogens—levonorgestrel (0.075 mg), norgestrel (0.030 mg), desogestrel (0.075 mg), norethindrone (0.035 mg) and ethynodiol diacetate (0.500 mg).

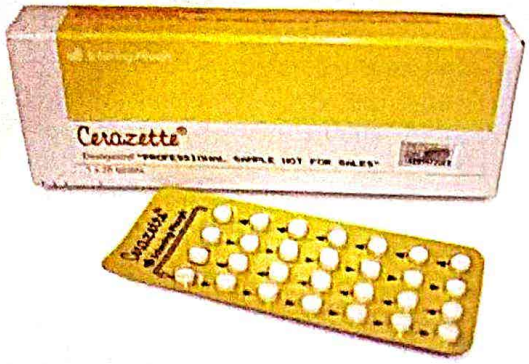



**Fig. 43.20:** Actual photograph of progestogen (progestin) only pill (mini pill)

### *Indications*

These are indicated for use by nursing mothers 6 weeks after delivery, women in whom estrogens are contraindicated or when estrogen related side effects render the combined OC pill unsuitable for the patient.

### *Contraindications*

Contraindications of progestogen only pill are shown in Table 43.7.

**Table 43.7: Contraindications of Progestin Only Pills (POPs) (WHO, 2017)**

| |
|---|
| **I. Absolute contraindications (WHO Category 4)** |
| 1. Breast cancer (current or past) |
| **II. Relative contraindications (WHO Category 3: Risks Outweigh Advantages)** |
| 1. Breastfeeding (< 6 weeks postpartum)<br>2. Cardiovascular disorders<br>i. Current DVT/PE<br>ii. Current ischemic heart disease<br>3. Migraine with aura<br>4. Liver disorders<br>i. Active viral hepatitis<br>ii. Severe cirrhosis<br>iii. Benign/Malignant liver tumors<br>iv. Drugs that effect liver enzymes (rifampicin and some anticonvulsants, e.g. phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine) |

### *Mechanism of Action*

The act by progestogen only pills:

1. Rendering the cervical mucus thick and non-receptive to spermatozoa thus inhibiting penetration of sperm (most important effect).
2. Rendering endometrium atrophic and hostile to implantation.
3. Premature luteolysis
4. Inhibition of ovulation in about 60% cases.

They do not kill the sperms.

### *Prescription of Mini pills*

They are started after 6 weeks of delivery in lactating women and within first 5 days (preferably first day) of the period. Mini pills must be taken daily at the same time of the day. Afternoon is the best time so that mucus plug become hostile for intercourse at night. However, any other time (e.g. bed time) can also be chosen for convenience. Desogestrel containing POP (cerazette) 75 µg desogestrel is used daily and is better. Safety margin is 12 hours. For missed pill backup method (e.g. condom) should be used for 48 hours.

### *Advantages*

1. As there is no effect on quality and quantity of milk, they are best pills for breastfeeding. Infact this is their major indication.
2. Absence of estrogenic side effects
3. They are useful for women older than 40 years.
4. They can be given to women with medical disorders like diabetes, epilepsy, smoking and history of thromboembolism where combined pills are contraindicated. They can also be freely used in obese, hypertensive and women with varicose veins.
5. They decrease the chances of PID and endometrial cancer.

### *Disadvantages*

1. Irregular vaginal bleeding is common and is seen in 20–30% cases and is a common cause for discontinuation.
2. Minor effects related to progestogens like acne, mastalgia and headache can occur.
3. Amenorrhea can occur. In such cases intrauterine and ectopic pregnancy should be first ruled out.
4. Higher failure rate (0.5–2/HWY)
5. Must be taken at same time daily.
6. For dose delay of > 3 hours, backup method should be used for 48 hours. For desogestrol containing POP, safety margin is upto 12 hours.

It is to be noted that POP is less effective and more expensive than COC and even one tablet cannot be missed. Hence, it is only used where COCs are contraindicated as COCs are drugs of first choice for contraception.

### *Effectiveness*

The typical average failure rate of mini pills as commonly used is 3–10% in the 1st year of use. If used effectively and consistently in breastfeeding women, the failure rate is only 0.5% in the 1st year. The failure rates are higher in younger women than in women over 40 years of age.

## Progestogen (Progestin) only Injectable Contraceptives

Their action is similar to progestogen only pill. These block LH surge and suppress ovulation, cause thickening of the cervical mucus, thinning of the endometrial lining (atrophy) and altered tubal motility.

*Types*

### *Depot Medroxy Progesterone Acetate (DMPA)*

Depo-Provera (DMPA) comes as microcrystals suspended in an aqueous solution given as 150 mg deep intramuscular injection by the Z-track technique (gluteal or deltoid) without local massage every 3 months.

The injection should be given within the first 7 days of the current menstrual cycle, otherwise a backup method is necessary for 1 week.

The efficacy of DMPA method is equal to that of sterilization. The indications and contraindications are like POP (see **Table 43.7**).

**Antara Program:** Recently Ministry of Health and Family Welfare, Govt. of India has made injeciton DMPA available free of cost through family welfare program all over India (DMPA 150 mg intramuscular use).

New formulation of DMPA (Depo- Sub Provera) contains 30% less progestogen than DMPA (104 mg instead of 150 mg) and is given subcutaneously rather than by intramuscular route. It is available as prefilled single use syringe (unijet) with short needle and can be given by community worker in community.

### *Norethindrone enanthate (Noristerate, NET-EN)*

Norethindrone enanthate is given in a dose of 200 mg intramuscularly every 2 months. This progestin acts in the same way as Depot-Provera, and has the same problems.

#### Side Effects

The following need to be noted:

1. Irregular bleeding
2. Weight gain related to increased body fat rather than fluid retention
3. Delayed return of fertility
4. Side effects like headache, nausea, dizziness, breast tenderness, psychological effects like loss of libido, depression, fatigue and nervousness have been reported.
5. Acne
6. Increase in hair growth (hirsuitism) and loss of scalp hair

#### *Counselling*

Proper counselling about the following points is important for the successful use of norethindrone enanthate.

1. There is potential for weight gain.
2. Patients should be warned about amenorrhea and irregular bleeding. This is important as patients may think they are pregnant.
3. Importance of returning for timely injections.
4. Lack of protection from STDs.
5. Delay in return of fertility after stopping medications.

## HORMONAL IMPLANTS

Hormonal implants are progestogen implants for contraception and are as follows:

### Norplant

The norplant system consisted of 6 capsules with 216 mg of levonogestrel (LNG) with each capsule having 36 mg of LNG (Fig. 43.21). Each capsule was 34 mm long and 2.4 mm wide. The rate of release of LNG was 85 µg per day during first six months, 50 µg per day by 9 months and 30 µg per day later. It was approved for 5 years though effect may last longer (up to 7 years). However, it was discontinued in 2008.

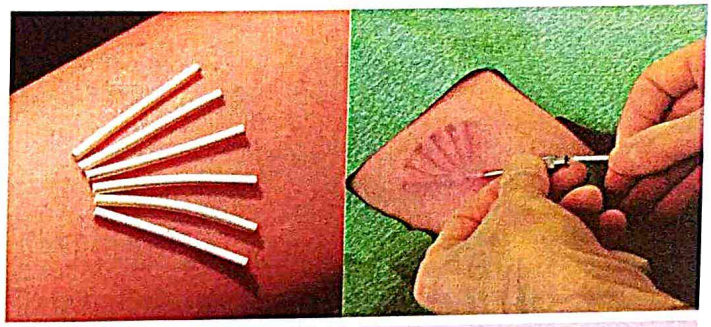


**Fig. 43.21:** Photograph of Norplant

#### Mechanism of Action

1. Norplant made cervical mucus scanty, thick and hostile to sperm penetration.
2. It suppressed ovulation in 50% cases by inhibiting hypothalamopituitary axis.
3. It depressed endometrial growth inhibiting implantation.

#### *Insertion*

The six silastic capsules of norplant were inserted under the skin in the inside of upper arm (6 cm above cubital fossa) or in forearm of left arm (in right-handed people). They were inserted in a fan-shaped manner under local anesthesia with the help of a special trocar through a small incision without the need of suture. Insertion can be done within day 1–7 of cycle (preferably day 1), immediately after abortion or 6 weeks after delivery. The doctor inserting was trained to insert and remove them.

#### *Removal*

The capsules were removed preferably after 5 years (rarely up to 7 years) of insertion. Contraceptive effect passed off immediately after removal.

### Norplant-2 (Jadelle) (Fig. 43.22)

**Norplant-2** (Jadelle) is composed of two levonorgestrel rods. Each rod measures 43 mm in length and 2.5 mm in diameter, contains 75 mg of the drug and releases the drug at the same dose as the 6 capsules. It is effective for 5 years and is approved by US FDA.

### Implanon

Implanon is a long-acting single rod subdermal implant (Fig. 43.23). The rod measures 4 cm in length and 2 mm in diameter. The core of the implant contains 68 mg of crystalline etonogestrel, dispersed in a matrix of ethylene vinyl acetate copolymer. The initial release rate is 60–70 µg per day and declines to 25–30 µg at the end of the third




**Fig. 43.22:** Photograph of hormonal implant Jadelle (Norplant-II)

year. Its use was recommended for 3 years. It was licensed for use in the United Kingdom but has now been replaced by nexplanon.

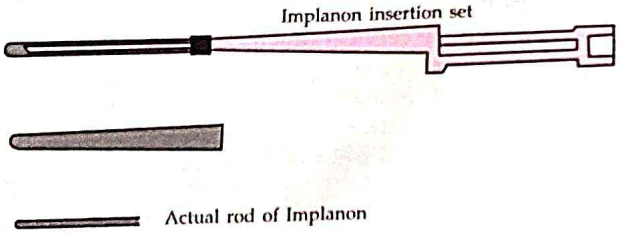



**Fig. 43.23:** Diagramatic representation of implanon

Mechanism of action and side effects are the same as norplant.

**NEXPLANON.** Implanon has now been replaced by nexplanon which contains the same amount of drug as implanon and has many similar characterstics, mechanism of action and side effects. It can be seen on X-ray.

**CAPRONOR.** It is a LNG containing capsule which is biodegradable working for 1 year and does not need removal.

**LEVOPLANT (Sino-Implant II.** It contains 2 rods of levonorgestrel 75 mg each. It works for 5 years.

### *Advantages of Hormonal Implants*

1. Implants are highly effective (pregnancy rate of 0.2–0.5 per 100 women year) comparable to oral pills.
2. Effective within 24 hours, it provides long-term contraception (3–5 years).
3. Fertility returns immediately on removal of the capsules.
4. Contraception is free from the act of sexual intercourse.
5. No estrogenic side effects.
6. Decreased incidence of anaemia.
7. Low risk of ectopic pregnancy.
8. No interference with breastfeeding.
9. Does not have any adverse effect on the lipid profile.
10. It is useful for women with completed family who do not want sterilization operation.

**Thus, implants are considered as 'reversible sterilization'.**

### *Disadvantages and Side Effects of Hormonal Implants*

1. This method requires a minor surgical procedure for insertion and removal of the implants and is painful.
2. Women suffer from side effects like mastalgia, weight gain, irregular menses, amenorrhea, hirsuitism, hair loss and sometimes altered libido.
3. No protection from STDs.
4. Diminished efficacy in epileptics on treatment.
5. Irregular bleeding and amenorrhea in a few cases.
6. High cost
7. Local reaction can occur in same cases.

### *Complications*

Capsule expulsion, inadvertent deep insertion of capsule, inadequate insertion, local infection and keloid formation. Removal may require sonographic or screening guidance. In India, the implants are not in common usage and are used only in research studies.

## Long Acting Reversible Contraceptive (LARC) Methods

LARC methods are defined as methods requiring administering less than once per cycle or month. LARC methods include copper intrauterine devices (non-hormonal) and three progestogen only methods of contraception (intrauterine systems, injectables and the implants). Intrauterine device and implants are more popular. They require a single act of motivation for long term use eliminating adherence and user dependence for effectiveness. As per WHO Medical Eligible Criteria for Contraceptive use (2017), LARC methods have few contraindications and almost all women are eligible for implants and intrauterine device. The American College of Obstetricians and Gynecologists (2015) recommended that LARC methods should be offered as first line contraceptive methods and should be encouraged as options for most women due to advantages of extreme efficacy, safety and non-user dependence and adherence and their potential to reduce unintended pregnancies. LARC methods should be part of counselling on all contraceptive methods and all appropriate candidates including nulliparous women and adolescents should be encouraged to use these methods. It can reduce unintended pregnancy rates as has happened in France where LARC methods are commonly used.

## EMERGENCY CONTRACEPTION (INTERCEPTION, MORNING AFTER PILL, POSTCOITAL CONTRACEPTION) (Fig. 43.24)

### *Definition*

Emergency contraception is a method of contraception used as an emergency procedure before menstruation is missed, to prevent pregnancy following unprotected intercourse or expected failure of contraceptives. **The overall risk of pregnancy is about 8% following single unprotected intercourse.**




**Fig. 43.24:** Actual photograph of emergency contraceptive tablets

### *Indications*

1. When there is a risk of pregnancy due to rupture or slippage of condoms (condom accident), detection of a defect in a diaphragm after its use or premature ejaculation in couples practising coitus interruptus regularly.
2. When unprotected isolated intercourse happens at some odd time in couples using regular contraceptive.
3. For aged couples having infrequent coitus
4. Missing pills with coitus
5. Expelled intrauterine device
6. Ejaculation on external genitalia
7. Following a single act of unprotected coitus in young girls.
8. Cases of rape and incest

### *Contraindications*

There are no contraindications to the use of emergency contraception.

### *Caution*

The morning after pill should not be used by women in whom pregnancy is suspected or by women with history of thromboembolic episodes or severe cardiovascular disease, migraine or active severe liver diseases.

### *Mechanism of Action*

The emergency contraceptive acts either by stopping or postponing ovulation or by interfering with postovulatory events necessary for implantation and longevity of the blastocyst. They also cause altered tubal motility, interfere with corpus luteum function and induce cervical mucus changes.

### *Methods and Dose*

1. Levonorgestrel (progestin) 0.75 mg, two doses given at 12 hours interval at the earliest after unprotected coitus but within 72 hours. It may be given up to 120 hours (5 days), but efficacy will be much lower. It is very successful and without any side effects. A single dose of 1.5 mg (2 tablets) is equally effective. Failure rate is 1%. It is the method of choice for emergency contraception in current practice being very effective and economical (₹ 35). In 2009, US FDA has approved the use of progestin only emergency contraception (called **Plan B**) without a prescription **(over the counter)** to women 17 years and older due to its efficacy, safety and intent in preventing unwanted adolescent pregnancies.
2. Combined estrogen and progestogen pills **(Yuzpe method)** is also effective. It consists of oral intake of 100 µg ethinyl estradiol with 1 mg of d-levonorgestrel in two divided doses 12 hours apart starting within 72 hours of unprotected intercourse (Total 4 tablets of ovral each containing 50 µg of ethinylestradiol and 0.25 mg d-levonorgestrel. Alternately, 4 tablets of low dose COCs can be taken 12 hours apart. Failure rate is 3.2%. Popular in past, its use is now superseded by LNG.
3. Conjugated equine estrogen 15 mg. The drug is taken orally twice daily for 5 days beginning soon after the exposure but not later than 72 hours.
4. **Mifepristone (RU 486):** It is antiprogesterone which blocks the action of progesterone on the endometrium causing sloughing and shedding of decidua preventing implantation. It is used as a simple dose of 25–50 mg with success rate of 99.1% (failure rate 0.9%). Recent studies show that even 10 mg is effective if administered after intercourse but before ovulation. A dose of 200 mg prevents implantation also. The drug is nonteratogenic and does not prevent ectopic pregnancy and may cause delayed periods. It is expensive.
5. **Ulipristal acetate (Ella one):** It can be used as a single tablet of **30 mg** within 5 days of intercourse. It is a synthetic progesterone hormone receptor modulator. It inhibits or delays ovulation and suppresses endometrium preventing implantation. Failure rate is upto 2%. It can cause mood changes, headache and should be avoided in severe liver disorders and severe asthma. It is very expensive.
6. Centchroman (ormeloxifene) in dose of 60 mg (two tablets) twice (repeated after 12 hours) in first 24 hours of coitus can prevent implantation with failure rate of 1–2%.
7. Prostaglandin suppositories are being tried for emergency contraception due to their luteolytic effect on the ovary and increased motility of fallopian tube. It also prevents implantation. However, it is still experimental and not used clinically.
8. **GnRH analogues:** Given in low dose daily buserelin (GnRH analogue) prevents ovulation. It is being tried but is expensive and can not be given by oral route (subcutaneous injection) and is not routinely used for emergency contraception yet.
9. Alternative to the above hormonal pills, emergency contraception can be achieved by inserting an IUD (not LNG-IUS) within 5 days of an unprotected intercourse which can be continued later. Failure rate is only 0 to 1% (15 times more effective than Yuzpe Regimen). It is useful for married women needing continued contraception.

   Infact ACOG (2017) has hailed IUD to be the most effective method of emergency contraception.

### *Advantages*

The efficacy of the 'morning after' pills is 98% for prevention of pregnancy provided the pills are taken within 72 hours of coitus.

### *Disadvantages*

Side effects include nausea and vomiting, spotting and irregular vaginal bleeding. Menstruation generally follows within 7–10 days of ingestion of medication.

Emergency contraception is now available over the counter (OTC) but should not be misused. Proper counselling is important. The women should be encouraged to use regular contraceptives and should not use emergency contraceptives too often.

EZY emergency contraceptive pill is now available free of cost under Govt. of India 'Hum Do Scheme'.

## PERMANENT METHODS MALE STERILIZATION

### Vasectomy

Vasectomy is popular in western countries (40% couples seek vasectomy in USA) but is less used in India with only 1% couples accepting it.

#### Advantages

1. Simpler operative procedure
2. Does not require hospitalization
3. No long-term side effects on physical, psychological, endocrinal and sexual function
4. Low failure rate
5. Does not alter sexual function
6. Costs are lower
7. Surgical reversal possible

#### Disadvantages

1. Permanent procedure
2. Does not protect against STDs
3. Not effective immediately. It requires about 3 months or 20 ejaculations before it becomes effective.
4. Not free from surgical risks
5. Some men suffer from psychological ill-effects.

#### Operative Technique and Post Operative Care (Fig. 43.25)

The operation can be performed by trained MBBS doctors and surgical specialists. Written consent is taken from acceptor after counselling and fulfilling criteria and ruling out contraindications. Blood Hb and urine are tested and tetanus toxoid is given. Vasectomy is performed at outpatient clinic or camp. He comes with scrotum shaved. He takes diazepam 10 mg orally 30 minutes before operation.

1. The scrotum is cleaned with antiseptic solution Vas is isolated and fixed under skin high up in the scrotum by the thumb and index finger. The area (subcutaneous tissue and sheath) is infiltrated with 5 mL 1% lignocaine solution.
2. **Scrotal incision.** About 1 cm incision is made transversely to divide skin, dartos muscle and sheath to expose white cordlike vas which is dissected for 3 cm by picking with an Allis forceps avoiding vessel injury (Figs. 43.25 A and B). Two silk ligatures or chromic catgut sutures are securedly placed on dissected vas 2 cm apart (Fig. 43.25 C). About 1 cm portion of vas is removed (Figs. 43.25 C and D).
3. Proper hemostasis is done. Skin closure is done by a single silk suture or nylon.
4. The procedure is repeated on the other side through second incision.
5. Gauze dressing is placed on the incision sites and suspensory bandage is worn for a week.
6. He leaves clinic after resting for half an hour. He can take up his work on the same day. It is better to send the excised segment for histopathological examination to confirm and for medico-legal reasons.
7. A course of antibiotics along with anti-inflammatory drugs is prescribed.
8. To avoid strenuous exercise for 2 days and avoid using a bicycle for 7 days.
9. He should use some form of scrotal support for 1–2 weeks to reduce discomfort.
10. He should use contraceptive measures (e.g. condom) for the next 3 months until his semen analysis shows absence of spermatozoa.

### Minivasectomy (non-scalpel vasectomy-NSV)

Dr. Li (1974) in China developed this procedure. Under local anesthesia, vas on one side is fixed by 3 fingers and vas fixing forceps. A tiny puncture is made on the skin by fine ended mosquito type dissecting forceps. Vas is made bare on widening puncture hole. A loop of vas is

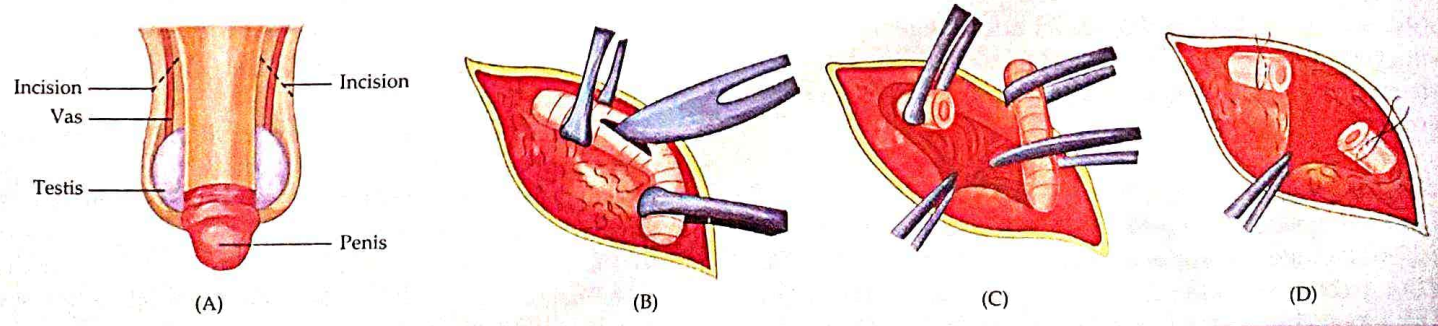



**Fig. 43.25:** Operative steps of vasectomy. **(A)** Skin incisions on both sides; **(B)** Cutting open of sheath of the vas; **(C)** A portion of the vas is being removed; **(D)** The two ends of the vas are being ligated

dissected. 1 cm is removed in between 2 silk ligatures. Vas ends are allowed to drop; testicular end of vas is covered with fascia. Suture is not needed for small puncture site. The same is repeated on the other side. NSV is as effective as conventional vasectomy but with least complications. Many centers in India are now practising NSV and it is available free of cost in most big government hospitals.

#### Effectiveness of Vasectomy

Highly effective with failure rate of 9.4 per thousand procedure. Failures result from unprotected intercourse too soon after vasectomy, incomplete occlusion of the vas deferens or recanalization.

#### Complications

Safer than tubectomy. Mortality is 8 per 100,000 procedures and morbidity very less. Compared with vasectomy, female tubal sterilization has a 20-fold increased complication rate, a 10 to 35 fold failure rate, and a 3-fold increased cost. **There is no evidence of increased chance of prostrate or testicular cancer in vasectomized men.**

##### *Early Complications*

1. Pain
2. Swelling
3. Discomfort
4. Haematoma (0.4–1.6%): They usually resolve in 1–3 weeks on rest, scrotal elevation and gradual mobilization. Rarely incision and drainage of haematoma may be needed for large and unresolving hematomas.
5. Local infection (1%): Rarely abscess formation may require drainage.
6. Anaesthetic complications like hypersensitivity and anaphylaxis can rarely occur.

##### *Delayed Complications*

1. **Sperm granuloma.** It manifests as a palpable area of induration at vasectomy site or in the epididymis in 0.1–0.3% cases. On microscopic examination, there is granulation tissue penetrated by microcanaliculae with spermatozoa and is a foreign body reaction with granuloma formation. It usually settles down with time. Persistent painful granuloma may need excision.
2. Antisperm antibodies can be demonstrated in 40–60% cases.
3. Psychological problems like sexual dysfunction or neurosis in anxious males.
4. Failure of vesectomy can occur in 0.1 to 1.2% cases due to reconnection of cut ends of vas (spontaneous recanalization).

### Newer Techniques

Vas deferens plugs or clips have been developed and are under investigation.

**RISUG (Reversible inhibition of sperm under guidance).** It has been developed by Prof S. K. Guha at I.I.T. Kharagpur, India. The vas deferens on both sides are injected with copolymer of styrene maleic anhydride (SMA) in solution of dimethyl sulfoxide.The drug produces an electric charge that nullifies the electrical charge of the spermatozoa preventing them from penetrating the ovum.The advantages include high efficacy for more than 10 years, convenience, less invasive, reversible (using another injection of sodium bicarbonate solution) and has minimum side effects (scrotal swelling and rarely kidney problems).It is still under phase 3 trials in India under ICMR. Results are very promising.

## FEMALE STERILIZATION

Female sterilization is a surgical procedure intended to end a woman's ability to become pregnant and involves ligation with or without resection or blocking both the fallopian tubes so that ovum and sperm cannot meet. The first tubal ligation was performed by **Dr J Blundell** of London in 1823.

Female sterilization is the most widely used contraceptive in the world with about 190 million couples relying on it. It is four times more commonly done than vasectomy in the world. In India, almost 98% of total voluntary sterilizations are female sterilizations.

### Indications

1. Permanent method for family planning after completing family is the main indication.
2. Multiparity after two or more children (though as per latest guideline even a woman with one living child can opt for sterilization).
3. **Obstetric indications.** During second or higher cesarean delivery provided other pre-requisites are fulfilled and the woman consent for it without any pressure.
4. **Surgical indications.** During conservative surgery for genital prolapse (e.g. Manchester repair) with completed family. During other gynecological operations with completed family like repair of complete perineal tear, repair of vesicovaginal fistula or rectovaginal fistula or laparoscopic or surgery laparotomy for gynecological indications (e.g. ectopic pregnancy, ovarian cyst removal etc.).
5. Medical disorders at high risk of pregnancy (certain types of severe heart disease like Eisenmenger syndrome, severe diabetes mellitus with complications, chronic kidney diseases, severe hypertension) as they worsen during pregnancy and may endanger her life.
6. Psychiatric disorders
7. Breast cancer and other malignancies
8. Eugenic indications with repeated congenital foetal anomalies or hereditary disorders like hemophilia, Tay Sach's disease and Wilson's disease. Sometimes for severe Rh isoimmunized mother sterilization is performed though with modern treatment and intrauterine transfusion, most women can have live fetus.

### Contraindications

There are no absolute contraindications to female sterilization. The relative contraindications are discussed in

Table 43.8. The risk of pregnancy must be weighed against the risks of the sterilization procedure.

**Medical eligibility criteria for sterilization.**

1. **Accept (A).** There is no medical reason to deny sterilization to a person with this condition.
2. **Caution (C).** Procedure is normally conducted in routine setting, but with extra preparation and precautions.
3. **Delay (D).** Procedure is delayed until condition is evaluated and or corrected.
4. **Special (S).** Procedure should be undertaken in a well-equipped setting.

## Time of operation

The operation can be performed at the following times.

1. **Interval Ligation or Sterilization**
   Interval sterilization is performed 6 weeks after delivery or at any time in non-pregnant women. It should preferably be performed within 7–10 days of onset of menstruation to avoid undetectable early pregnancy. If performed in second half of menstrual cycle or during lactational amenorrhea, concomitant vacuum aspiration or dilatation and curettage of uterus may be performed along with sterilization to avoid failure.
2. **Postpartum Ligation (Puerperal ligation)**
   It is done most conveniently in the early puerperium within 72 hours for women who have completed the family and desire sterilization.
   Advantages of postpartum ligation
   (*i*) It is technically easier as uterus is enlarged and close to the anterior abdominal wall with easy and ready access to fallopian tubes
   (*ii*) Postoperative and postpartum convalescence period can coincide, a particularly important scenario in India where women do not get enough time to rest.
   (*iii*) If ligation is not done, the woman may come back with pregnancy for MTP with higher morbidity.
3. **Sterilization during cesarean section.** If the woman is undergoing cesarean section with completed family, it is ideal.
4. **During Medical Termination of Pregnancy (MTP).** Sterilization (laparoscopic or minilaparotomy) can be easily combined with suction evacuation performed for medical termination of pregnancy. Women can be easily motivated for it after proper counselling. If medical abortion is done, (mifepristone or misoprostol) then ligation should be performed after the completion of abortion procedure (about 2 weeks after administration of mifepristone or misoprostol) after ensuring empty uterus.
5. **Sterilization following spontaneous abortion** can be performed provided she fulfils the criteria.
6. **Sterilization along with any other surgery** (e.g. Manchester repair, ovariotomy and ectopic pregnancy)

## Methods of female sterilization

The basic fundamental principle in female sterilization is breaking the continuity of both fallopian tubes by removing a small segment of both tubes. The operation is popular as tubectomy in lay people. With advancement of laparoscopy, occlusion (blockage) of both tubes is usually performed using rings (e.g. Fallope rings, Yoon rings) or clips (e.g. Hulka clips, Filshie clips) without resecting a part of tubes which is much simpler and smaller operation with better length of tubes available in case recanalization is required in future. Hysterectomy, though, has sterilization effect, is not performed only for sterilization purpose. If a woman undergoes hysterectomy for some gynecological indication, she can not be given a certificate of sterilization as per rules.

**Table 43.8: Relative Contraindications of Female Sterilization (WHO, Category 3), WHO 2017**

| |
|---|
| **1. Psychiatric illness** |
| **2. Physical illness** |
| i. Fever<br>ii. Jaundice or chronic liver disease<br>iii. Anaemia (< 7 Hb)<br>iv. Chronic systemic diseases like heart disease, uncontrolled diabetes, hypertension, thyrotoxicosis, tuberculosis, bronchial asthma<br>v. Any cancer<br>vi. Skin infection at operative site<br>vii. Pelvic masses or adhesions or infection<br>viii. Bleeding or clotting disorders<br>ix. Ongoing pregnancy<br>x. Multiple scars of previous pregnancy |
| **3. Allergy to local anesthesia (alternative anesthesia is provided)** |
| **4. Gross obesity** |
| **5. Relative contraindications of postpartum ligation** |
| i. Puerperal fever<br>ii. Between 7 and 42 days<br>iii. Severe pre-eclampsia/eclampsia<br>iv. Antepartum or postpartum haemorrhage with severe anaemia<br>v. Genital tract trauma<br>vi. Puerperal psychosis |

**Laparoscopic tubal ligation should not be done concurrently with second trimester abortion and in the postpartum period**. Where equipment and trained staff are available, the laparoscopic approach to the fallopian tubes is quicker and results in less minor morbidity compared with mini laparotomy. The procedure should be performed as a day care whenever possible.

## Pre-requisites

### *A. Criteria for Eligibility (Case Selection)*

(Self-declaration by clients should be the basis for compiling this information.)

1. Clients should be married.
2. Female clients should be above the age of 22 years and below the age of 49 years.
3. The couple should have at least one child whose age is above one year unless the sterilization is medically indicated.

4. Clients or their spouses/partners must not have undergone sterilization in the past (not applicable in cases of failure of previous sterilization).
5. Clients must be in a sound state of mind so as to understand the full implications of sterilization.
6. Mentally ill clients must be certified by a psychiatrist and a statement should be given by the legal guardian/spouse regarding the soundness of the client's state of mind.

## Pre-operative preparation

The couple is counselled about the permanent nature, irreversibility and failure rate of operation.

### *A. Informed Consent*

1. Detailed history is taken and physical examination performed to rule out any medical or surgical disorder. Hemoglobin and urine anlysis for sugar and albumin are done.
2. Client must sign the consent form (Annexure 1) for sterilization before the surgery without coercion in full conscience. The client is informed about the irreversibility and rare failure of the procedure.
3. The consent of the spouse is not required for sterilization.

## Surgical techniques

Important points to be kept in mind about surgical techniques are:

1. The operating surgeon should identify each fallopian tube clearly by following it up to the fimbrial end.
2. The site of the occlusion of the fallopian tube must always be within 2–3 cm from the uterine cornua in the isthmic portion (this will improve the possibility of reversal if required in the future). Care must be taken to avoid damage to the blood vessels, ovaries and surrounding tissues.
3. Excision of at least 1 cm of the tube should be done. Use of cautery and crushing of the tube should be avoided.

### *Routes*

1. Abdominal
2. Vaginal

### *Abdominal*

1. Conventional
2. Mini laparotomy
3. Laparoscopy

Though female sterilization can be done in camps, these are discouraged these days due to higher complications in camps.

## Traditional (Conventional) Laparotomy Technique

I. **Surgeon.** The procedure is usually performed by a gynecologist trained in sterilization, but can also be performed by general surgeon or even junior doctor who is trained and certified to perform the operation.

II. **Place of surgery.** It is usually performed in recognized and certified operation theatre with adequate facilities. It was done in outpatient settings and mass camps in past but not any more done due to risk of complications.

III. **Type of Anesthesia/Analgesia.** Female sterilization can be done under general, spinal and epidural anesthesia. Local anesthesia with sedation may be adequate for some cases.

IV. **Preparation.** Abdomen is cleaned with antiseptic solution (povidone iodine) and draped with surgically autoclaved drapes.

V. **Types of Incision.** It depends upon the type of sterilization. For the postpartum ligation, the incision is made subumbilically 2 cm below the fundus of uterus. The incision 3–4 cm in length and is usually transverse (preferred due to early ambulation and less risk of incisional hernia) but can be median or right paramedian.

VI. **Opening of Abdomen and Identification and delivery of Fallopian Tubes.** Abdomen opened layer wise in usual way. The abdominal wound is retracted to one side with the small retractor, pelvic organs are explored, any adhesion are separated. One tube is identified by fimbrial end, grasped with babcock forceps and brought out for sterilization. The same procedure is repeated on other side. In case of postpartum ligation, only fundus of the uterus is felt. The index and middle finger of surgeon (or index finger alone) are used to palpate and bring out one fallopian tube by palpating the posterior surface of fundus of uterus and posterior leaf of broad ligament. The tube must be identified by fimbrial end to avoid ligation the round ligaments. The same procedure is repeated in the opposite side.

## Mini Laparotomy (Mini-Lap) Technique

In this case, the female tubal sterilization (tubectomy) is performed through a small incision (< 2.5 cm) given in the anterior abdominal wall using a device in the uterus (e.g. Hulka-device which contains teeth like vulsellum to hold anterior lip of cervix along with a rod which goes in uterine cavity) which keep uterus anteverted and brings fallopian tubes near anterior abdominal wall. Dr Uchida from Japan has popularized this technique.

### Operative steps

1. Abdomen is cleaned and draped. The surgery is performed by an experienced operator with training in mini laparotomy technique.
2. **Anesthesia.** It is usually performed under local analgesia with some sedation. However, for anxious patients, short general or spinal anesthesia can be given.
3. **Type of incision.** It is like in laparotomy described above. The exception is that the incision is very small (< 2.5 cm). Abdomen is opened in usual way.
4. **The procedure.** Uterine manipulator (Hulka variety mentioned above) is inserted by an assistant and uterus and one fallopian tube are brought closer to anterior abdominal wall in the incision area. The

tube is grasped with babcock forceps and sterilization procedure performed (see below). The procedure is repeated on the opposite side.
5. Abdomen is closed in a routine way. Subcuticular skin stitches may be applied as they do not need removal and give better cosmetic result.
6. Patient is discharged after 24–48 hours after assessing her condition.

*Advantages*
1. Mini laparotomy is a safe, effective, convenient method of female sterilization.
2. It can be done as an outpatient procedure.
3. It can be performed even by junior doctors in primary health centers and camps.
4. It can be used for mass sterilization for population control.
5. Most complications are minor.
6. It does not need special training or special equipment.
7. It can be performed soon after childbirth, after abortion or for interval sterilization.

*Drawbacks*
Drawbacks as compared with laparoscopy are:
1. Higher chances of wound and pelvic infections
2. Longer duration and severity of post operative pain
3. Longer convalescence period
4. Longer incisional scar than that needed for laparoscopic sterilization.

## Vaginal ligation
Tubal ligation through the vaginal route is also called as **colpotomy tubal ligation** which is performed in non-pregnant cases. It can be done through anterior colpotomy with prolapse surgery while in other cases it is performed through posterior colpotomy. Its only advantage is avoiding an abdominal scar. Vaginal ligation as compared with laparotomy is much more difficult to perform and needs longer operating time and more expertise. It cannot be done for larger uterus or associated tubo-ovarian masses. Difficulties occur in about 6% cases owing to interference by the bowel, adhesions and fixation of tubes and ovaries and the presence of tubo-ovarian masses. Trauma and injury of the ovaries and tubes and uncontrollable haemorrhage may need urgent laparotomy. Even in experienced hands, failure to ligate tubes vaginally happens in 2–3% cases needing laparotomy for sterilization. Serious pelvic infection occurs in 2–3% cases although mortality is rare. Failure happens in 1 in 300–500 cases. Other complications are same as in transabdominal methods though with slightly higher incidence. Due to increased rate of infectious morbidity, it is not commonly done these days. Even for prolapse surgery (Manchester repair), laproscopic sterilization is performed these days rather than vaginal ligation.

Transvaginal ligation can be done by two methods.
1. **Colpotomy** (making incision in pouch of Douglas) and bringing tubes out in vagina. Occlusion of tubes is done by ligation and excision or by mechanical devices (clips and rings)
   Current status: Not in use.
2. **Culdoscopy.** Vaginal ligation is done using culdoscopy with occlusion of tubes by mechanical devices (clips and rings) or electrocoagulation (unipolar or bipolar)
   Current status: Not in use.

## Techniques
**Pomeroy's Technique** (Figs. 43.26 A–C)
Using a babcock forceps, a segment of the fallopian tube from isthmic part is lifted up through the laparotomy incision. The loop is ligated with plain or chromic catgut suture. The segment is resected. The procedure is performed on both sides. After the absorption of suture, the cut ends remain widely apart reducing chance of recanalization (Fig. 43.26 C). Plain catgut is better to ensure early absorption of suture with early separation of the tubal ends.

**Advantages.** Pomeroy's technique is the most simple, safe and effective procedure. The failure rate is 1 in 300–400 operations. It can be used in laparotomy, mini laparotomy or vaginal routes.

**Parkland Procedure** (Fig. 43.27 A)
The fallopian tube is separated from the adjacent mesosalpinx for about 2.5 cm. The freed fallopian tube is ligated proximally and distally with 1–0 chromic catgut suture and the intervening segment of about 2 cm is excised and inspected for hemostasis. This method avoids the initial intimate proximity of the cut ends of the fallopian

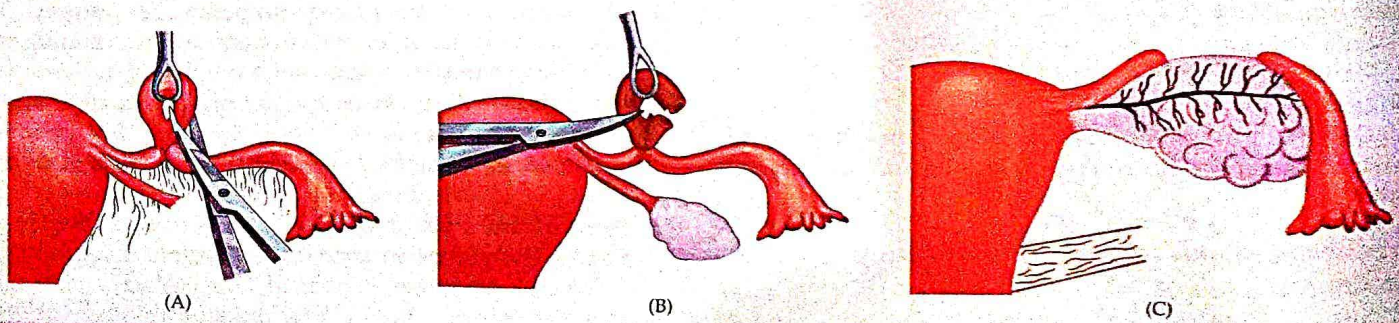



**Fig. 43.26:** Operative steps of Pomeroy's technique of female sterilization. (A) A segment of the fallopian tube from isthmic part is lifted up and the loop is ligated with plain or chromic catgut suture; (B) The segment is resected; (C) The retracted ends of the tube after absorption of the stitch

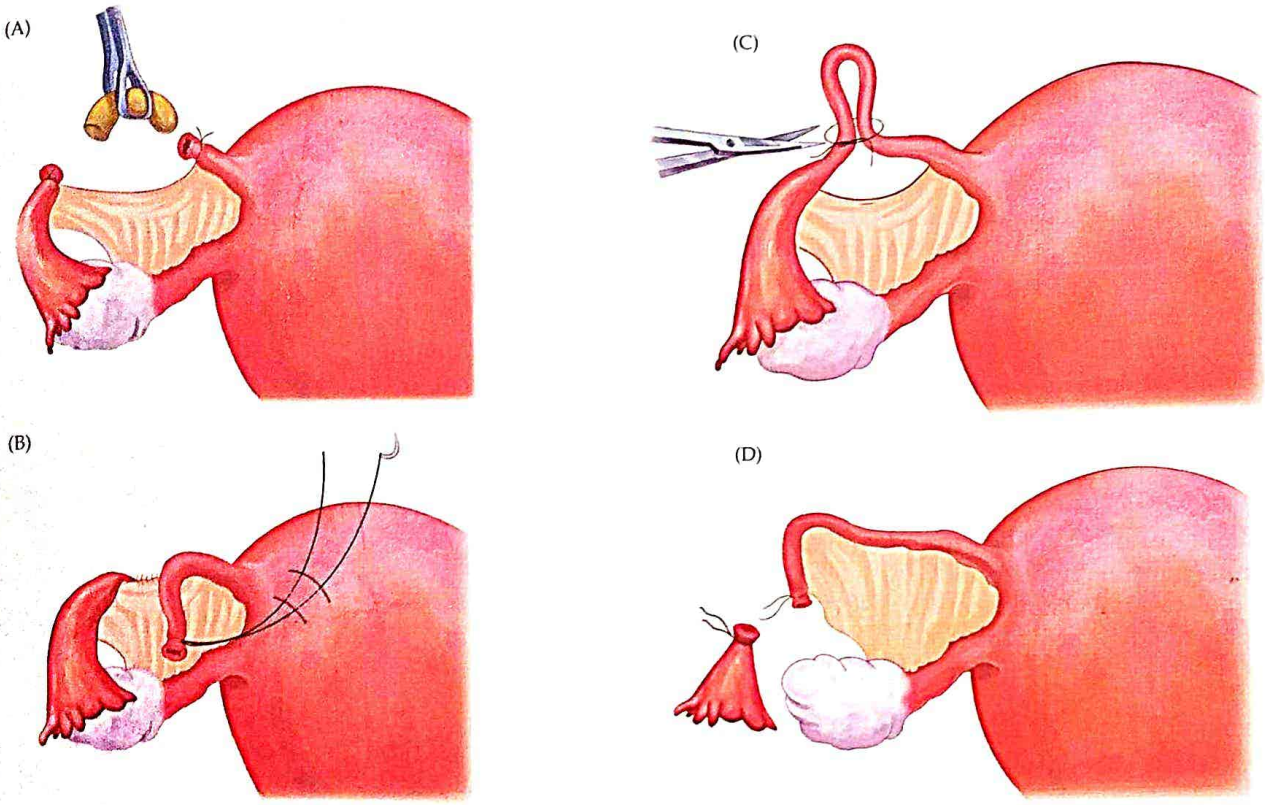



Fig. 43.27: **(A)** Parkland method in which a segment of tube is ligated proximally and distally with chromic catgut suture and about 2 cm segment is excised; **(B)** Irving's procedure in which the medial cut end of tube is buried in the myometrium while the distal end is buried in the mesosalpinx; **(C)** Madlener's procedure of female sterilization with use of non-absorbable (silk) suture and crushing of tube; **(D)** Kroener's fimbriectomy with ligation of ampullary end of tube and resection of fimbria.

tube as seen in Pomeroy's technique reducing failure rate. The failure rate is less than 0.25% (< 1 in 400 procedures).

**Irving Method** (Fig. 43.27 B)
Irving method is the most difficult to perform but has a very low failure rate (< 0.1%). The cut fallopian tube is separated from the mesosalpinx sufficiently to free a medial segment of tube. The freed proximal stump is buried within a tunnel created in the myometrium posteriorly and the distal stump is buried within the mesosalpinx.

**Madlener's Technique** (Fig. 43.27 C)
In this technique, a loop of fallopian tube in the middle third is crushed at the base and ligated with silk. The procedure is very simple and easy. However, due to its very high failure rate (0.3 to 2%), it is now almost obsolete.

**Uchida Method**
Uchida method tries to prevent fistula formation in tubal sterilization failure. The fallopian tube is ligated with No. 0 chromic catgut suture near uterotubal junction (proximally) with resection of 5 cm segment of tube and also ligated distally. The distal stump is exteriorized while the proximal stump is buried within the leaves of the mesosalpinx. It has very low failure rate.

**Kroener's Fimbriectomy** (Fig. 43.27 D)
In Kroener's fimbriectomy, the fibrial end of the tube is doubly ligated with silk sutures and then removed. Though its failure rate is almost nil, recanalization if needed in future is not possible.

**Shirodkar's 96 Method of Tubal Sterilization**
Prof. V N Shirodkar from Mumbai, India performed tubal sterilization after cutting tube by ligating tube in 96 method in which proximal end was inverted upwards (9) while the distal end was inverted downward (6). It is not commonly done.

## Complications of tubal ligation

Complications of tubal ligation depend on method of sterilization, and anesthesia, routes of ligation, place of operation, selection of patient and technical capability of the operator. Following mini laparotomy, major complications happen in 1.8% and minor complications in about 14% cases, (WHO, 1982). ICMR study found about 8% minor complications occurring after conventional laparotomy for sterilization.

**Operative Complications**
1. Anaesthetic hazards
2. Bowel injuries
3. Bladder injuries
4. Injuries of tubes and ovaries
5. Broad ligament haematoma
6. Uterine bleeding and uterine perforation

### Post-operative Complications

1. Wound infection (the most common)
2. Wound haematoma
3. Wound dehiscence
4. Pelvic infection
5. Peritonitis
6. Urinary infection
7. Pelvic haematoma
8. Intraperitoneal haemorrhage
9. Bowel fistulae
10. Bladder fistulae

Complications are more frequent with vaginal than with abdominal tubal ligations and following cesarean section and abortion rather than after interval ligations.

## Laparoscopic Sterilization (Figs. 43.28 and 43.29)

Female sterilization with the use of an operating laparoscope is becoming more and more popular as it can be done through tiny holes under sedation and local anesthesia on an outpatient basis with patient going home the same day. However, this procedure needs some special instruments (Fig. 43.28) and special training.

#### Procedure

Under local or general anesthesia, parts are cleaned and draped. Pneumoperitoneum is created with Verres needle using about 2 liters of carbon dioxide. With the help of trocar and cannula, laparoscope is inserted and sterilization is performed by one of the following methods.

### *Methods of Tubal Occlusion*

#### *1. Electrocoagulation*

Electrocoagulation was used in the past and had low failure rate. But due to serious burns of other organs, vessels and the abdominal wall which may occur especially with unipolar electrocoagulation it is not used these days. There is also little chance of doing recanalization due to damage of tube.

#### *2. Clips*

Two types of clips are mostly used, the spring-loaded clip (Hulka-Clemens clip) and silicon-titanium clip (Filshie clip). The clips are more suitable in young women who may need reversibility of sterilization and in post abortal cases when the tubes are edematous. The clips cause least damage to the tube (about 5 mm) whereas tubal damage is 3 cm with the Falope ring and 3–4 cm with the Pomeroy's technique. However, they are more expensive and are not commonly used in India though Filshie clips are now available in India.

#### *3. Falope rings*

Falope rings were introduced by Yoon in 1974. At present in India, this technique is usually adopted. The rings can be applied through a single or double puncture technique. The Falope ring is made of silicone rubber with 5% barium sulphate. The outer diameter is 3.6 mm, the inner diameter 1.0 mm and the thickness 2.2 mm. The specially equipped laparoscope is loaded with two Falope rings and is inserted in standard fashion. After identifying one of the fallopian tubes, the tongs of the band applicator are extended and the tube grasped about 3 cm from the uterus, choosing a thin portion of the tube (Fig. 43.29 A). The tube is then brought into the applicator. The band is pushed over the knuckle of the tube (Fig. 43.29 B). The tongs are then released and the tube is dropped. If the double-incision technique is followed, the band applicator is passed through the second trocar. The other tube is then occluded in the same manner.

Occasionally the tube is transected. This is managed by grasping the stumps with the tongs and applying a band to each. The bleeding can also be controlled by applying the band to the bleeding point or by use of cautery.

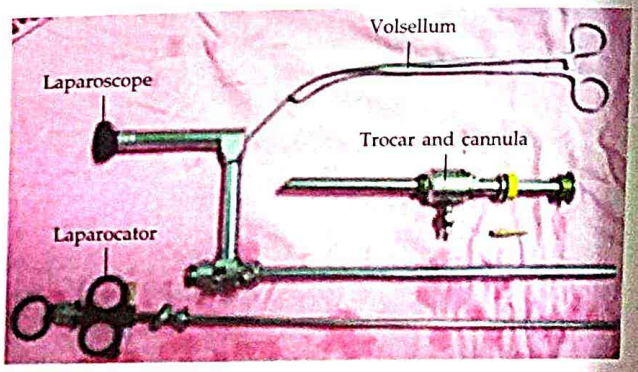



**Fig. 43.28:** Instruments used in laparoscopic sterilization

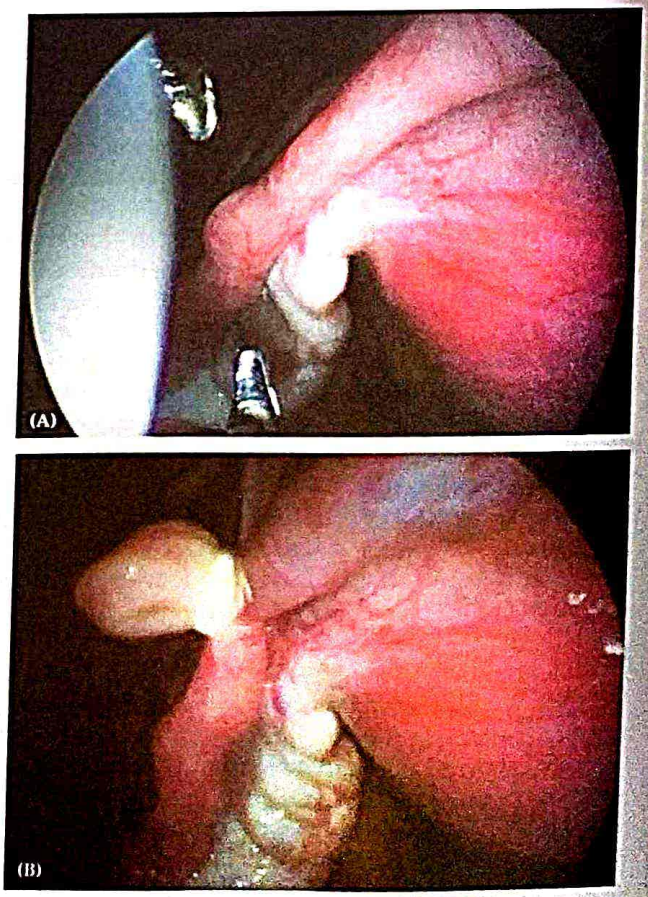



**Fig. 43.29: (A)** Laparoscopic view of uterus and fallopian tubes with sterlization being performed with falope rings; **(B)** Falope rings in place on fallopian tubes

Ensure placement of falope rings on the fallopian tubes by following the tubes up to fimbrial ends. After ensuring hemostasis and no injury to any organ, the laparoscope is withdrawn followed by cannula after allowing the intraperitoneal gas to escape out. The small abdominal wound is stitched with 3–0 non-absorbable nylon suture. It can also be stitched with absorbable polyglactin (vicryl) or chromic catgut suture which do not need removal (although removal is better to avoid leaving foreign body). Patients can be discharged in 3–4 hours and returns after one week for suture removal.

### Advantages of the Laparoscopic Sterilization

There are many advantages of laparoscopic sterilization making it the method of choice in India.

1. Laparoscopic sterilization is a very effective method of contraception. It is better than any temporary method and almost as effective as mini laparotomy or conventional methods of sterilization.
2. It is effective from the time of operation and needs no continued motivation.
3. It has a very low complication rate.
4. It can be performed safely in an outpatient department and in camps, in a short time of 5–15 minutes on an average.
5. It can be performed easily under local anesthesia, eliminates the need for hospitalization.
6. It requires a small incision, leaves a very small scar and eliminates the need for prolonged rest beyond a day or two.
7. Post operative pain, discomfort and restriction of activity are minimal.
8. Unsuspected pathological conditions can be diagnosed during the incidental exploration of the pelvic and abdominal organs and can be managed.
9. It is highly reversible. A success rate of about 80% or even more can be achieved by recanalization particularly following use of clips (e.g. Filshie clips).

### Disadvantages

1. The complications are rare but can be serious, even endangering life.
2. It usually requires insufflation of the abdomen with attendant risk of post operative chest and shoulder pain and rarely cardiorespiratory distress, gas embolism and sudden cardiac failure.
3. The equipment is costly and difficult to maintain. Maintenance facilities are as yet poor in India, even in the cities.
4. It requires considerable and relatively difficult training.
5. It is not recommended in early puerperium, in very obese patients and in patients with pelvic adhesions due to infection or previous operations. Although laparoscopic sterilization can be performed in these cases with care and experience, the risks are greater. Besides, there are absolute contraindications like heart disease, gross respiratory dysfunction, abdominal hernias and abdominal tumors mainly due to pneumoperitoneum risk.

### Complications

Actual complications are rare (0.9–1.6%) but can be dangerous with laparoscopic sterilization. Their frequency is directly related to the surgical expertise of surgeon and other factors like aseptic care, proper choice of cases and anesthetist team. The US collaborative review of sterilization (crest) study on 10,863 women who underwent tubal sterilization from 1978 to 1986 looked for complications and failure rates of various methods. They are as follows:

1. Wound sepsis
2. Abdominal wall haematoma
3. Injury of the large vessels
4. Uterine injury
5. Intestinal injury
6. Burns of the gastrointestinal tract and other pelvic organs (only where fulguration technique is followed. However, it is not used these days)
7. Surgical and mediastinal emphysema
8. Cardiorespiratory embarrassment
9. Regrets: Up to 7% women may have regrets by 5 years (crest study).
10. Rarely, even death can occur.

In view of these possible life-threatening complications, the presence of a trained anesthetist with anaesthetic equipment and facilities for laparotomy are essential.

### Failure Rates

Failures can occur due to surgical errors (account for 30–50% cases), fistula formation, spontaneous reanastomosis (recanalization), equipment failure, defective current in electrocautery ligation, luteal phase pregnancy (patient already pregnant, can be avoided by doing ligation in follicular phase) and without any reason. The typical failure rate following female sterilization in USA is 0.4% in the 1st year (CREST study). In India, large studies reported a failure rate of 0.2–1.3% following laparoscopic sterilizations. The Pomeroy's method has the least failure rate (1 in 300–400 procedures) while the Madlener's, technique has higher rate (2%). The failure rate is increased when it is done through vaginal route or when combined with cesarean delivery or hysterotomy.

### Mortality

The mortality rate following female sterilization is about 1.5 per 100,000 procedures in USA but is about 10/100,000 in India. The three major causes of death after female sterilization are anaesthetic hazards, infection (particularly tetanus in developing countries including India) and haemorrhage if the tying sutures on the tubes are too lax. Most of these deaths are preventable.

### Reversibility

The woman and her partner must be counselled that sterilization is a permanent method. Unfortunately death of children can occur necessitating request for recanalization. Sterilization reversal is expensive, difficult and a major operation (can be done by laparotomy or laparoscopy) needing microsurgical techniques. Success of recanalization

depends upon the method of sterilization and condition of tubes and is higher (up to 75%) for laparoscopic fallope rings and clips and is poor for electrocoagulation techniques. There is risk of ectopic pregnancy (up to 10% cases of reversal).

**Legal Issues**

Government of India has started Family Planning Insurance Scheme to safeguard the interests of patients and doctors and to give a boost to the sterilization operations as follows;

1. Death of a patient at hospital or within 7 days of sterilization operation: ₹ 2 Lakhs
2. Death of a patient from day 8–30 days of sterilization operation: ₹ 50,000
3. Expenses for treatment of medical complications: ₹ 25,000
4. Failure of sterilization: ₹ 30,000
5. Doctors/Facilities covered for litigations upto ₹ 2 Lakhs per 4 cases per year including defence costs.

All doctors performing female sterlizations need to be empanelled to take benefit of the scheme.

## Contraceptives for different age groups and Medical conditions

1. **Adolescent contraception:** For married adolescent girls, combined oral contraception is most effective and best method. DMPA and norplant can also be used. In unmarried sexually active girls, barrier method should be used to prevent pregnancy and to prevent sexually transmitted disease also. They can also take combined pill if they are regularly sexually active. They should also have access to emergency contraceptive. IUCD is avoided in them.
2. **Newly wed couples:** Combined oral pills are best for them for few years till they do not want pregnancy.
3. **After miscarriage:** Combined pills can be given IUCD can also be inserted.
4. **After child birth:** Post placental IUD insertion is a good choice and is encouraged by Govt. of India (see above for details).
   (*i*) **For nonlactating mothers:** After 3 weeks of childbirth combined pills can be given or IUD can be inserted. Inj. DMPA and progestogen implants can also be used.
   (*ii*) **For lactating mothers:** IUCD a good choice after about 10 weeks of childbirth. Progestogen only pill is also a good alternative. DMPA and progestogen implants can also be given. Combined pills are not given.

   Thus for spacing IUCD is best method especially in lactating women. After completion of family, male or female sterilization can also be performed.
5. **Contraception after 35 years of age:** Male or female sterilization can be performed if family is completed. IUCD (CuT380A) can be used upto 1 year beyond menopause. For women having menorrhagia, LNG-IUS (Mirena) can be inserted. Combined low dose pills can be used if the woman is not obese, non-smoker with no medical disease. In presence of these conditions, progestogen only pill can be used. For women nearing menopause with less frequent coitus, barrier methods can be used with emergency contraception and MTP as a backup method.
6. **Contraception for HIV positive mother:** Condoms are ideal for them to prevent transmission of infection from one partner to other. For better efficacy, dual method (barrier with combined pills) is preferred. IUCD can be inserted if the woman is on medication and does not have PID. Sterilization can be performed after family is completed.
7. **Women at risk of sexually transmitted diseases:** They should use dual contraception (barrier with combined pills).
8. **Women with medical disorders:** The risk of pregnancy is weighted against the risk of contraceptives. After completed family, male or female sterilization can be performed. In women with heart disease and diabetes mellitus, IUCD can be inserted with caution due to risk of pelvic infection. Heart disease is category 2 (can be used) for IUCD.

   For women with hypertension, heart disease and diabetes mellitus, hepatic disorders, breast cancer, past thromboembolic disorders, combined pills are contraindicated (category 3 and 4).

   Progestogen only pill can be given in heart disease but is contraindicated in breast cancer, hepatic diseases and thromboembolic disorder. Postcoital (emergency contraception) with levonorgestrel can be safely given to women with medical disorder.

   Women with antiepileptic drugs (except sodium valproate) and on antituberculous drugs (especially rifampicin) which are enzyme inducers cause interaction with combined pills leading onto increased failure rate of OCs necessitating higher dose (50 mgm of ethinyl estradiol).
9. **Women with Psychiatric diseases:** For a woman with severe psychiatric disease or severe mental retardation where she may be unfit to bear children, sterilization should only be performed after written opinion from psychiatrist. Sometimes, court permission may be needed. Written consent of husband or legal guardian is sought for sterilization. Emergency contraceptive can be used and safely given.

GATHER Aproach for family planning: It is a counseling approach using the acronym

G: Greet; A: Ask; T: Tell, H: Help; E: Explain; R: Return. It has documented effectiveness in family planning programs with more satisfied customers who use contraception more.

CAFETERIA approach in family planning is a strategy adopted by Govt. and uses a 'basket of choices' from a five official methods female: sterilization, male sterlization, intrauterine contraceptive devices (IUCD), Oral contraceptions and condoms. It is very effective.

Post abortion First trimester: Implants and intrauterine devices are MEC-1 (Medical Eligibility Criteria),

Second trimester: IUCDS are MEC-2.

Post septic abortion: IUCD are MEC-4 (contraindicated).

## NEWER AND UNDER TRIAL CONTRACEPTIVES

### Male Contraceptives

1. **Gossypol.** It was developed in China in 1972 and is still in use there. It is an aldehyde derived from seeds, stem and roots of cotton plant and is now available as pure forms of gossypol acetic acid and gossypol formic acid. It inhibits spermatogenesis, decreases epididymal sperm motility and decreases conversion of proacrosin to acrosin. It is administered orally 10–20 mg daily for 3 months and thereafter 20 mg twice weekly. The adverse effects are exhaustion, reduced libido and delayed restoration of fertility. The most severe side effect is hypokalemic paralysis seen in 1% cases. Using gossypol acetic acid with potassium supplement can reduce this adverse effect.
2. **GnRH analogues.** They cause suppression of gonadotropins and spermatogenesis. However, their cost is very high and they can reduce libido in many cases and hence are not in much use.
3. **Testosterone injections.** Various testosterone preparations like testosterone enanthate, testosterone buciclate, testosterone undecanoate, progestogen depot medroxy progesterone acetate (DMPA), antiandrogen and progestogen cyproterone acetate have been tried to suppress spermatogenesis and testosterone secretion. They may produce reversible azoospermia in 40–70% men and oligospermia in others. However, add back therapy with longer acting testosterone is needed to maintain sexual function.

   Testosterone enanthate 20 mg intramuscular weekly provides reversible contraception for 1 year while injection testosterone undecanoate 1000 mg initially followed by 500 mg monthly has been found effective for upto 2 years in Chinese studies.

   Intravas devices: Vas deferens occlusion by percentaneous injection of polyurethane elastomer plugs or urethane device fitted with nylon threads blocks vas and is inserted through scrotal incision: 94% success rate.

### Female Contraceptives

1. **Centchroman.** See under hormonal contraception above
2. **Combined injectable contraceptives.** See under hormonal contraception above
3. **Transdermal delivery system.** See under hormonal contraception above
4. **Vaginal rings.** See under hormonal contraception above
5. **Combined ring.** See under hormonal contraception above
6. LNG **rod.** Recently developed implant with a total 150 mg of levonorgestrel (75 mg/rod) is found to be very effective for five years.
7. **ST-1435 single rod implant or nestorone implant.** It is a single modified silastic implant with a new progestogen crystals encased in a silastic capsule releasing hormone at rate of 100 µg/day. Initial results are promising. Side effects are like LNG implant.
8. **Chinese No. 2 implant.** It is called sino-implant which is like jadelle but contains more (150 mg) of LNG.
9. **Biodegradable implants.** They are being developed. They contain a biodegradable polymer E-caprolactone instead of non-biodegradable silicone for faster release of LNG. It works for one year. It works like norplant by suppressing ovulation and making cervical mucus hostile.
10. **Biodegradable injectable contraceptives.** It uses norethindrone alone or in combination with ethinyl estradiol as a microsphere (< 0.1 mm diameter) using a biodegradable lactide glycolide as a copolymer. It is administered as an injection deep in the gluteus maximus muscle. Its mechanism of action is by suppression of ovulation and cervical mucus change. Disadvantage is that it can not be removed after injection.
11. **GnRh agonists and antagonists.** They are not yet routinely used but are promising. The GnRh agonist like buserelin and antagonists like otrorelix inhibit pituitary response to natural GnRh. They prevent ovulation by inhibiting ovarian follicular growth and also suppress endometrial growth. The side effects are like menopausal syndrome, hot flushes, decreased libido, osteopenia. Add back therapy can be used.
12. **Intratubal chemical methods.** Quinacrine pellets have been inserted in the fallopian tubes transcervically to result in permanent sterilization by producing scarring to block the fallopian tubes. Although they were used for many women in past, a pregnancy rate of 3, 10 and 12 percent was observed after 1, 5 and 10 years of use in large Vietnamese studies. Side effects are headache, dizziness, backache, vaginal discharge and itching. They are not used at present. Other agents are silicone plugs, methylcyanoacrylate and erythromycin but none are in practical use.
13. **Transcervical method.** Essure system in which cornual end of fallopian tube is occluded with a microinsert with fine stainless steel inner coil and an expendable outer coil of nickel and titanium alloy under hysteroscopic control. It incites a chronic inflammatory response and tissue ingrowth causing tubal blockage. Hysterosalpingography is required 3 months after surgery to confirm occlusion of tubes. Side effects include abnormal uterine bleeding (AUB), perforation of uterus or fallopian tubes due to device migration and allergy to nickel. It has now become available in India with promising results (Fig. 43.30) but is very expensive. It has a high success rate of about 99%.
14. **Adiana.** Newer method of hysteroscopic female sterilization. Two step process controlled thermal damage of endosalpinx followed by insertion of a biocompatible matrix plug within the tubal lumen. Not yet available in India.
15. **Ovablock.** Method of inserting a rubber tube in bilateral tubes under hysteroscopy. Not yet available in India.

16. **Newer IUDs.** Frameless IUDs (Gynefix) contain six bracelets of copper that are crimped on to a string and affixed to the fundal myometrium. It contains a total of 330 $mm^2$ of copper. It may cause lesser side effects like bleeding, pain and infection. However, they have a higher expulsion rate and are not used in USA. A threadless or butterfly IUD is also being developed to lower side effects but is still not in use.
17. **Immunocontraception.** Use of body's natural immune defence mechanisms to provide protection against an unplanned pregnancy. It requires the production of a controlled time limited and non-pathogen immune response to components of the reproductive process. Various possible vaccines are shown in Fig. 43.32B.
18. **hCG vaccine.** It was developed by Dr. G P Talwar from AIIMS, New Delhi
    Beta HCG vaccine. A recombinant HCG-β-LTB vaccine adsorbed on alum with *Mycobacterium indicus pranii*. Three injections at fort. Nightly intervals followed by booster on day 60 or 120. It is approved by drug controller of India and has extensive toxicology studies in 2 species of rudents and marmosets. It is under phase II clinical trial. It prevents implantation. Results appear promising with only 1 pregnancy in 1224 cases.

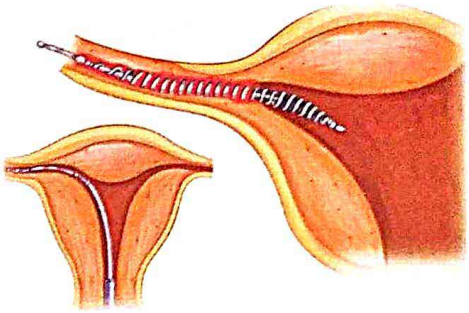


**Fig. 43.30:** Hysteroscopic placement of ESSURE microinsert

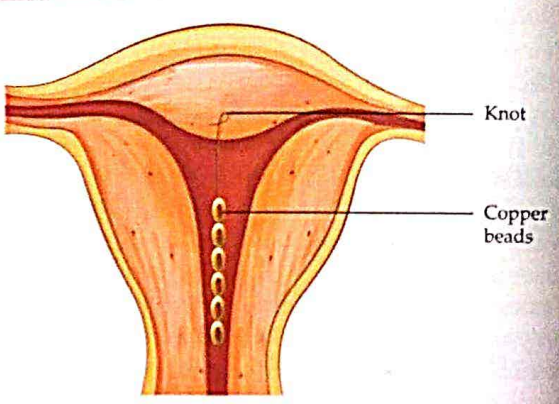



**Fig. 43.31:** Gynefix (frameless IUD) in position in uterus

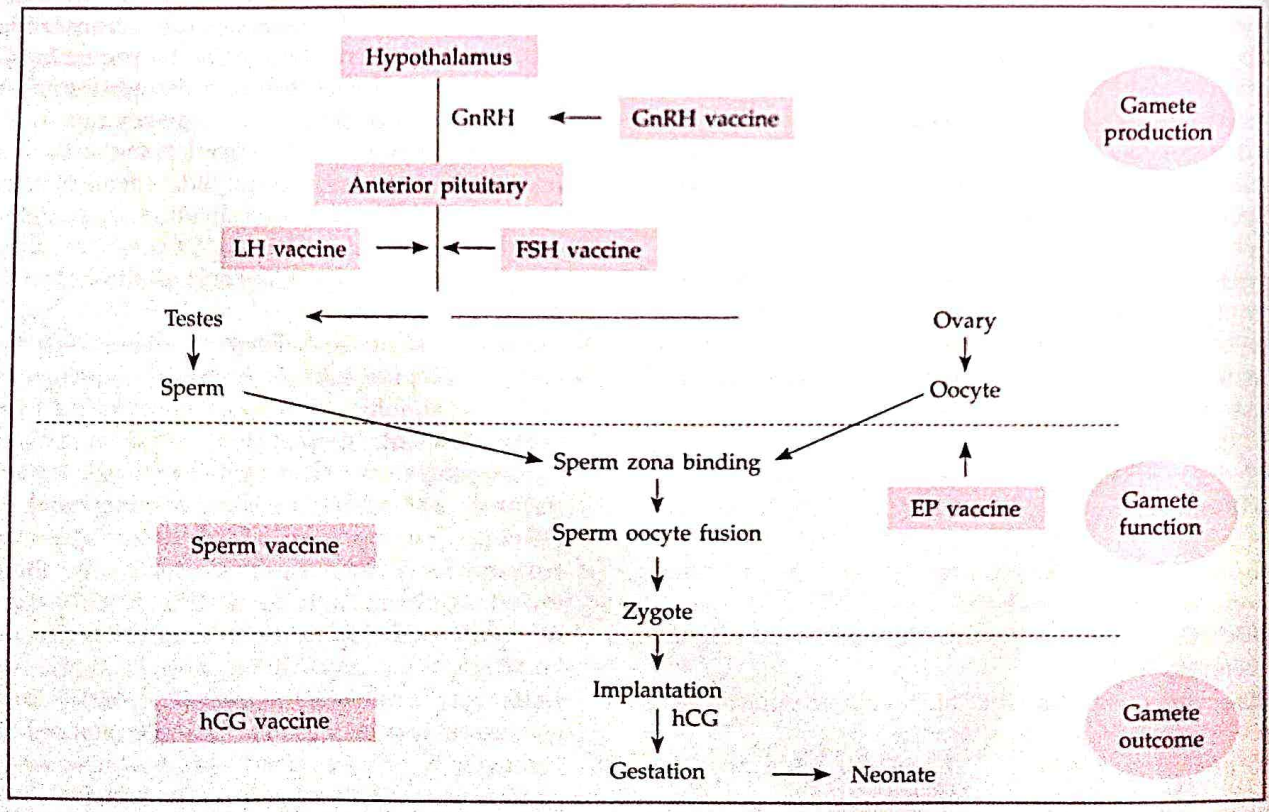



**Fig. 43.32:** Various vaccines to prevent pregnancy in both men and women.

**Table 43.9: Comparison of Mini laparotomy (Mini-lap) Sterilization and Laparoscopic Sterilization**

| Characteristics | Mini-lap Sterilization | Laparoscopic Sterilization |
|---|---|---|
| 1. Definition | Female sterilization performed through mini laparotomy incision (2.5–3 cm) usually using some uterine elevation and manipulation. | Female sterilization performed with laparoscope using some pneumoperitonium and uterine elevation and manipulation. |
| 2. Equipment | It does not need any special instruments or maintenance and is very economical. | Laparoscope and laparocator are costly and need ongoing maintenance. |
| 3. Training | It does not need special training and can be performed even by junior doctors after minimal training with minimal assistance. | It requires special training and expertise and can only be performed by gynecologists and surgeons trained and certified to perform laparoscopic sterilization. Good assistance is also needed. |
| 4. Place of operation | It can be performed in primary health centers and camps, though it should be performed in accredited centers. | It is usually performed in fully equipped operating room with trained gynecologist, anesthetist, electricity and laparoscope and laparocator but can also be done in camps (not preferred). |
| 5. Timing of operation | It can be performed at any time including in postpartum period, with termination of pregnancy or at interval. | It cannot be performed in puerperium and with large uterus. |
| 6. Contra-indications | No specific contraindications. | Laparoscopic sterilization is contraindicated in cardiovascular and respiratory diseases, morbidly obese women and in anticipated intra-abdominal adhesions and in puerperium. |
| 7. Complications | Higher chances of wound and pelvic infections. | Less wound and pelvic infections. Minimal complications. However, rarely serious complications like bowel injury, injury to blood vessels and surgical emphysema can occur. |
| 8. Post operative pain | Longer duration and severity of post operative pain. | Lesser post operative pain. |
| 9. Convalescence | Longer convalescence period. Patients need to stay in hospital for 2–3 days. | Shorter convalescence period. Patients can be discharged in 4 hours. |
| 10. Scar | Longer incisional scar | Very tiny scar. |
| 11. Efficacy | Very effective. Failure rate is only 0.1–0.3%. | Very effective. Slightly higher failure rate of 0.2–0.6%. |
| 12. Possibility of recanalization | Recanalization is possible but difficult and with poorer results due to bands, adhesions and smaller tubal length left behind after excision (3–4 cm of tube is destroyed). | Recanalization is possible and is easier and more successful (80% success) due to lesser adhesions and better tubal length especially with Filshie and Hulka clips where only 4 mm of tube is destroyed while 2–3 cm of tube is destroyed with falope rings. |

## Key Points

1. Temporary (spacing) methods of contraception include the natural methods, barrier methods, oral contraceptives, intrauterine contraceptive devices, injectables and implants. Male and female sterilization are permanent (terminal) methods.
2. Diaphragm, cervical cap, female condom and spermicides are barrier methods used by women and the condom is the barrier method used by men. The condom especially male condom gives protection against STDs, including HIV.
3. Natural contraception methods are rhythm method, coitus interruptus and breastfeeding. Conventional contraceptive methods are condoms, vaginal diaphragm, spermicides and rhythm method. Fertility awareness based methods include: natural contraception and barrier methods.
4. Copper containing IUCDs and the low dose oral contraceptives are provided free by the National Family Welfare Programme in India.
5. Intrauterine device (IUD) has been proven to be a safe and effective method for fertility control with the advantage of being a one time procedure without need of regular use of tablets or barrier contraceptive. The IUD is inserted in OPD without anesthesia under all asepsis by 'No touch' and withdrawal method. It prevents implantation of the fetilized ovum. **The life for CuT 200 is 3 years, multiload 250 is 3 years, multiload 375 is 5 years, CuT 380A is 10 years and LNG-IUS is 5 years.** Both CuT 380A and Multiload 375 are now available free of cost by Govt.
6. The contraindications of insertion of IUD are pelvic infection, abnormal uterine bleeding, genital TB, choriocarcinoma, carcinoma cervix. The failure rate is about 0.5–2 per HWY. Complications of IUD include pelvic pain, vasovagal attack, uterine perforation, menstrual dysfunction (menorrhagia), pelvic inflammatory disease (PID), ectopic pregnancy, spontaneous expulsion and infertility. Menstrual dysfunction, active PID, uterine perforation, pregnancy, missing thread, partial expulsion, one year after menopause and the woman wanting to conceive are indications of removal.
7. IUD may also be used for emergency contraception up to 5 days and following hysteroscopic adhesiolysis in Asherman's syndrome. LNG-IUS has many advantages like reduction in blood loss and is a treatment of menorrhagia. It is a safe and effective method of contraception but is very expensive.
8. Combined oral contraceptives are one of the best methods of contraception with many health benefits but have major side effects and indications of withdrawal. They **act by inhibition of ovulation**, by causing endometrial hypoplasia and by changing character of cervical mucus. The pill is started from day one of a cycle and is continued as 'three weeks on and one week off' regimen. The newer low dose pills have low estrogen and better progestins like drospirenone which are very safe. The pill can be taken up to the menopause in non-obese and non-smoker women in absence of contraindications but under supervision.
9. Major absolute contraindications of combined pills are thromboembolism, estrogen dependent tumor, liver diseases, severe migraine, cardiac disease and diabetes. Minor side effects of pills are nausea, vomiting, breakthrough bleeding, mastalgia, leg cramps, weight gain, hypomenorrhea and amenorrhea. Major complications are rare and include depression, hypertension and thromboembolic manifestations. Progestin only pill (mini pill) contains norgestrel 30 µg, LNG 75 µg or desogestrel 75 µg. Its main indication is in breastfeeding women, as it does not reduce quantity and quality of breast milk. It can be given to women with contraindications to estrogens.
10. Progestogen injection DMPA (every 3 months) and NETEN (every 2 months) are effective but cause menstrual dysfunction and amenorrhea. Norplant is safe and effective for long term use. Norplant (implant) is inserted subdermally for long term use (5 years). It is safe, effective and is considered as 'reversible sterilization'. Implanon is a single rod delivery system. DMPA infection is now available under family planning program free of cost by Govt. of India as 'Antara program'
11. Emergency (post coital) contraception is the use of contraception after unprotected intercourse. Usually LNG in dose of 1.5 mg in single or two divided doses 12 hours apart is used within 72 hours. IUD can be used upto 5 days. Yuzpe regimen (estrogen progestin combination) is not popular. Ulipristal acetate (Ella-one) 60 mg is most efficient and can be given upto 120hours (5 days)
12. Centchroman (ormeloxifene) developed by Central Drug Research Institute, Lucknow, India is a non-hormonal synthetic chemical used as once a week pill in dose of 30 mg. It alters the endometrium preventing implantation. It is also useful for fibroids and menorrhagia. It is now supplied free of cost by Govt. of India as (Chhaya).
13. Vasectomy is a very safe and effective method of permanent male sterilization which can be done under local anesthesia Other contraceptive methods or condoms should be used for at least 20 ejaculations or for 2–3 months after vasectomy. Non-scalpel vasectomy is even better and is performed under local anesthesia without need of scalpel or suture but needs training.
14. Female sterilization is a safe, effective and most commonly performed surgical procedure as permanent method. It can be done by laparotomy (less commonly) or mini laparotomy in puerperium or interval sterilization. Laparoscopic sterilization using falope rings or clips is the most commonly performed procedure which can be done as day care surgery but cannot be done in puerperium and needs laparoscope and trained surgeons to perform the procedure.
15. Hysteroscopic sterilization includes essure placement or quinacrine pellets but are not commonly used.
16. Newer pills, injections, IUDs, vaccines are being developed with better safety, efficacy and lesser side effects.
17. The prescription of contraceptives depends upon age, medical or surgical condition, general and social condition and needs to be individualized.

Section 12
MISCELLANEOUS AND CURRENT TOPICS

CHAPTER 44

# Audit, Medicolegal Aspects, Consent PC PNDT Act, Ethics, Communications, Counselling, Skilled Training and Biomedical Waste Management in Obstetrics

## AUDIT IN OBSTETRICS

### Definition

**Audit in obstetrics includes the systematic and critical analysis of the quality of obstetric care rendered to women.**

Audit seeks to improve patient care and outcomes through systematic review of care against defined criteria and the implementation of change. While research is concerned with discovering right things to do, audit is concerned with ensuring that the right thing is done. Audit can evaluate the structure (organization), process and outcome of care against an agreed standard and can be used for prevention, diagnosis and treatment of patients. Studies have shown that audit is an effective method of improving the quality of care.

### Examples of Audit

Confidential Enquiries into Maternal Deaths in UK is the longest running audit in obstetrics in the world which has helped to significantly reduce maternal mortality rates in UK. The audit study of perinatal mortality in teaching hospitals in India has helped in lowering it. Any aspect of obstetric practice can be audited. Average time taken between decision-making and actual performance of cesarean can be audited and causes for delay can be found. It will improve services in future.

### Undertaking Audit and Audit Cycle

Audit requires a multi-disciplinary approach with involvement of all the departments involved in patient care. It has five principal steps referred to as the audit cycle (Fig. 44.1). Guidelines for medical audit:

1. Health authorities and medical staff should define explictly their respective responsibilities for the quality of patient care.
2. Medical staff should organise themselves in order to fulfill responsibilities for audit and for taking action to improve clinical performance.

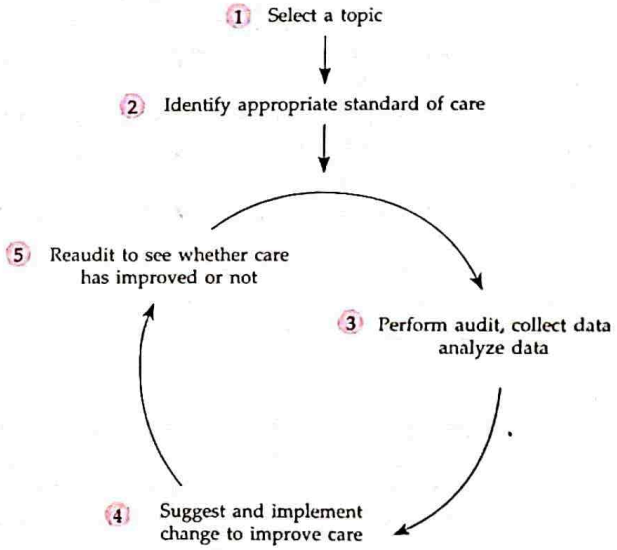



**Fig. 44.1:** Audit cycle

3. Each hospital and speciality should agree a regular programme of audit in which doctors in all grades participate.
4. The process of audit should be relevant, objective, qualified, repeatable and able to effect appropriate change in organisation of the service and clinical practice.
5. Clinicians should be provided with the resources for medical audit.
6. The process and outcome of medical audit should be documented.
7. Medical audit should be subject to evaluation.

### Significance of an Audit

1. Audit is an essential learning tool which reviews the current practice, identifies problem areas and suggests methods of improvement.

2. With the help of audit, non-evidence based and less effective practices can be replaced with the evidence based and more effective procedures.
3. It can be used to increase awareness and better understanding amongst physicians and their patients.

**Drawbacks**

Performing audit is a hard and dedicated work needing lot of time, effort and staff commitment. Some providers dislike it as they are already busy in their clinical practice. Persuation and confidentiality are effective ways to convince each person involved in patient care to use audit in their practice.

## MEDICOLEGAL ASPECTS IN OBSTETRICS

Obstetrics is one of the most litigious and high risk speciality as obstetricians deal with two lives—the mother and the fetus. There may be an adverse maternal or perinatal event even without negligence, e.g. birth of a baby with cerebral palsy for which usually the antepartum factors are responsible but the parents often blame that timely cesarean was not done for intrapartum hypoxia leading to cerebral palsy in the newborn.

There is a growing awareness amongst patients and their relatives that the adverse outcome may have been a result of an error. Most litigations arise out of the suboptimal care that may have been due to negligence, malpractice or incompetence. Malpractice is failure of rendering professional services that exercise a degree of skill and learning commonly applied under all circumstances in the community by the average prudent reputable member of the profession which leads to injury, loss or damage to the patient.

Once an obstetric patient comes to a doctor, the doctor needs to render medical care and attention as per the established norms. If the doctor fails to provide adequate care, she may be rendered negligent. Failure to provide standard care may be an act of omission or commission. If the court of law proves the doctor guilty of providing suboptimal care due to negligence, incompetence or malpractice, the doctor will have to compensate the complainant (plaintiff) as per ruling of the court or shall have to appeal in a higher court.

### Common areas of Medicolegal Cases in Obstetrics

1. Maternal causes
   - (*i*) Foetal heart rate (FHR) monitoring recognition and action on abnormal FHR pattern.
   - (*ii*) Death
   - (*iii*) Severe trauma, including fecal incontinence or urinary incontinence
   - (*iv*) Retained swabs in abdomen or in vagina
   - (*v*) Management of dystocia
   - (*vi*) Use of oxytocin
   - (*vii*) Decision to incision time in cesarean delivery.
   - (*viii*) Failure to notify the high risk problem to seniors.
2. Foetal causes
   - (*i*) Perinatal death
   - (*ii*) Cerebral palsy
   - (*iii*) Other foetal handicap
   - (*iv*) Foetal trauma, e.g. fractures
   - (*v*) Resuscitation of the newborn

Instrumental delivery is an important area of litigation.

### Methods to reduce medicolegal cases

1. **Communication.** The obstetrician should communicate with the woman in simple and easy language preferably in her mother tongue which she can easily understand. Her husband and relatives (e.g. mother-in-law or father-in-law) should also be involved in communication and decision-making (e.g. cesarean delivery) (see below for details of communication).
   The obstetrician should always speak the truth and honestly and clearly tell the facts to patients and their husbands, especially while breaking the bad news like informing about death of fetus or a congenital malformation in the fetus.
2. **Informed Written Consent.** Informed consent give patients the power of participating in decisions concerning their own management.
   The patients should be given honest, unbiased, up-to-date information about their illness, its likely outcome and the risks and benefits of different interventions in easy to understand language.
   **According to law, to treat a competent adult without obtaining his/her consent amounts to assault which is a punishable offence.** To be able to give an informed consent, the patient must
   - (*i*) be mentally competent to make the decision.
   - (*ii*) have adequate information upon which to base the decision.
   - (*iii*) reach a decision voluntarily, without undue influence from health professionals, family or friends.

   However, in an emergency situation, we may provide medical treatment to anyone who needs it to save life or prevent significant decline in her health even without written consent, e.g. treating a woman with eclampsia or performing cesarean section on a women with major degree placenta previa with acute haemorrhage. We should try to take husband or relative's consent and should inform her as soon as she has recovered from the problem.
3. **Case Note Documentation.** Good medical records are essential for the delivery of quality medical care, without which we cannot treat patients with competence and safety. Accurate medical records are our best defence against allegations of negligence and medical malpractice. They should be correct, clear, comprehensive and chronological and should be carefully preserved.
4. Practice of evidence based medicine and adherence to established management protocols of the hospital are mandatory. Any deviation from agreed protocol should be clearly mentioned in notes including its reason.
5. Proper training and supervision of junior and senior residents are essential. Consultants should perform

difficult and life-threatening cases, e.g. cesarean for major degree placenta previa with suspicion of placenta accreta.
6. Discussion with other specialists like physicians, cardiologists, endocrinologists, etc., for high-risk cases.
7. The staff should attend continuing medical education (CME) programs to update their knowledge.
8. Audit meetings, mortality meetings and perinatology meetings are helpful to improve the quality of care.

## CLINICAL RISK MANAGEMENT (CRM)

Clinical risk management aims to identify adverse events by using either incident report or systematic screening of records in cases of maternal and perinatal mortality and severe morbidity.

A risk management team of obstetricians, neonatologists and anesthetists, review reported cases of adverse events for any human errors and failures.

The principal aim is to reduce the frequency of adverse events and harm to patients and to reduce the medicolegal cases.

### Consent

Defined as voluntary agreement, compliance or permission for a specified act of purpose.

**Types**

- Implied Consent - for routine medical care which doesn't involve significant risk
  - Examination of patient coming to OPD.
  - Administration of drugs to indoor patients
- Expressed Consent
  - May be given verbally or in written

Situations where consent is not required

**Medical emergencies:**

- If the patient is incapable of giving consent and has no attendant – a life saving procedure can be done as per IPC 92 without consent

**Medical examination under Sec 53, CrPC**

- Examination of accused by medical practitioner at the request of police officer.

## PRE-CONCEPTION AND PRENATAL DIAGNOSTIC TECHNIQUES (PC PNDT) ACT

To stop the menace of female feticide causing dramatic decline in child sex ratio, the Parliament of India enacted the Prohibition of Sex Selection Act in September 1994. The amended PC PNDT act came into effect on 14 February 2003.

PC PNDT act is an act to provide for the prohibition of sex selection, before or after conception. It also regulates clinical laboratories and ultrasound centers to diagnose genetic chromosomal abnormalities or certain congenital malformations or sex-linked disorders and to prevent the misuse of sex determination leading to female feticide.

The Act has 8 chapters which deal with various aspects of genetic diagnosis including registration of clinics, allowance of genetic tests for appropriate indications, maintenance of records and penalty for offences.

Any healthcare worker contravening any of the provisions of the Act or rules or the husband or any other relative compeling the pregnant woman to undergo prenatal diagnostic test shall be punishable with imprisonment for a term which may extend up to three years and fine of ₹ 10,000. On any subsequent conviction, imprisonment may extend up to five years and fine may extend up to ₹ 50,000. The State Medical Council can cancel offender's registration for 5 years for first offence and permanently for subsequent offence.

All offences under this act are cognizable (a police office can arrest the offender without warrant), non-bailable (getting bail is not the right of accused. The courts have discretion to grant bail) and non-compoundable (Parties to the case can not settle the case out of court and decide not to prosecute).

**Rules of PC PNDT Act**

1. PC PNDT act rules describe the definition of various terms, qualification of employees, sale of ultrasound machines, registration of genetic clinics, maintenance and preservation of records.
   As per act gynecologist is a person who has done postgraduate qualification in obstetrics and gynecology. For genetic counseling center, the gynecologist must have 6 months experience in genetic counseling or 4 weeks training in genetic counseling. For genetic clinic, the gynecologist should have adequate experience in prenatal diagnostic procedures and should have performed at least 20 procedures (e.g. amniocentesis, chorion villus sampling, cordocentesis etc.) under supervision of experienced gynecologist in the field. Medical geneticist should have > 2 years experience in speciality after qualification.
2. All clinics conducting ultrasound must be registered and only doctor qualified under the act can use diagnostic techniques such as ultrasound. They guide about taking written consent in specified form before doing the procedure and that any person conducting ultrasound or imaging shall give declaration on each report that the sex of the fetus is neither detected nor disclosed to anybody by words, signs or in only other manners.
3. They also guide about analyses of the genetic test and that prenatal diagnostic procedures are done under ultrasonic guidance to prevent damage to fetus and placenta.
4. The rules also give details of procedure for search and seizure of any institution by the authorities and the institutions have to provide facilities to the authorities for inspection of their centers.
5. Every institution and clinic shall predominantly display in its premises a notice in English and regional language to the effect that disclosure of sex of fetus is prohibited under law.

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2. All clinics conducting ultrasound must be registered and only doctor qualified under the act can use diagnostic techniques such as ultrasound. They guide about taking written consent in specified form before doing the procedure and that any person conducting ultrasound or imaging shall give declaration on each report that the sex of the fetus is neither detected nor disclosed to anybody by words, signs or in only other manners.
3. They also guide about analyses of the genetic test and that prenatal diagnostic procedures are done under ultrasonic guidance to prevent damage to fetus and placenta.
4. The rules also give details of procedure for search and seizure of any institution by the authorities and the institutions have to provide facilities to the authorities for inspection of their centers.
5. Every institution and clinic shall predominantly display in its premises a notice in English and regional language to the effect that disclosure of sex of fetus is prohibited under law.

6. At least one copy each of the Act and these rules shall be available on the premises for the clients. The code of conduct should be observed by persons working at genetic counselling center, genetic clinic, genetic laboratories and ultrasound centers. Various forms need to be filled before doing prenatal tests like form B for certificate of registration, form G for consent, etc.
7. Maintenance of records: For a period of 2 years from the dose of procedure or in the event of any legal proceeding till the final disposal of it.

The PC PNDT act is an important step in reducing sex-selective abortion and its strict implementation is necessary for improving the fast decreasing female/male ratio in India. Medical professionals must keep the sanctity of their profession by not indulging in illegal sex determination and female feticide. At the same time, it is equally important that no woman should be denied prenatal diagnostic tests for judicious indications due to fear of the Act.

The act permits and regulates the use of prenatal diagnostic techniques for detection of chromosomal anomalies, genetic metabolic diseases, hemoglobinopathies, or sex linked genetic diseases, congenital anomalies or other abnormalities or diseases as may be specified by the central supervisory board, age > 35 year, history of ≥ 2 spontaneous abortions or foetal losses, exposure to teratogenic agents and family history of mental retardation or physical deformities or any other genetic disease.

## BIOETHICS IN OBSTETRICS

'Ethics' is a generic term for referring to the moral code of conduct in a civil society. Bioethics denotes the systemic study of the moral dimensions—including moral vision, decisions, conduct and policies—of the life sciences and healthcare.

The Medical Council of India established the Code of Ethics in 1956 (revised in 2002) to be followed by all the registered medical practitioners in the country. Any violation of the Code may lead to 'penalties' including cancellation of registration. The Indian Council of Medical Research brought out the revised detailed "Ethical Guidelines for Biomedical Research on Human Subjects" in 2000 to guide researchers about the research.

## COMMUNICATION IN OBSTETRICS

Communication is effective talking to patients by which physicians and patients relate to each other and attempt to achieve therapeutic goals. Effective communication has been shown to be essential to the patients' satisfaction with the care they receive and helps in development of trust between the doctor and the patient. It can help in reducing medicolegal cases. Honest and true communication about the condition and its management should be given.

## COUNSELLING IN OBSTETRICS

Counselling is a way of working with patients and trying to understand their feelings and help them in making decision about her care, maintaining eye-to-eye contact with her and by being nice and helpful to her. The doctor should be very patient, gentle and empathic with her and should give her practical help by using simple and easy to understand language.

GATHER approach is used for effective communication and better doctor-patient relationship.

G—Greet the patient
A—Ask about her health and her family
T—Tell patients all the available options with their merits and demerits.
H—Help them in choosing the appropriate treatment
E—Explain everything in details
R—Return visit after treatment

Special empathic but true communication is to be given to the family in case of maternal death or severe maternal morbidity. The patients and their husbands are to be counselled in a simple but factual information in case of intrauterine death, stillbirth or birth of a baby with congenital malformation.

While examining patients, empathic attitude should be used and they should agree for the examination. During examination by a male doctor, a female attendant should be present.

## SKILLED TRAINING IN OBSTETRICS

Imparting of obstetric skills to trainee doctors (residents) in obstetrics is crucial to enhance their skills in dealing with difficult obstetric cases like instrumental deliveries, conducting breech delivery, etc. Obstetric arts like mid cavity forceps deliveries, destructive operations, breech delivery are gradually disappearing. There is real need to train young obstetricians in various obstetric arts such as:

1. Instrumental deliveries
2. Manual rotation of head
3. Breech deliveries, especially delivery of afer-coming head of breech
4. Cesarean deliveries, especially in difficult cases
5. Intrauterine manipulations like internal podalic version
6. External cephalic version
7. Shoulder dystocia drill
8. Cardiopulmonary resuscitation of pregnant mothers and newborns.
9. Managing postpartum collapse like acute inversion of uterus
10. Appropriate management of postpartum haemorrhage.

**Methods of Training**

1. **Use of simulations.** To avoid experimenting on real patients, use of simulations can sharpen skills of young obstetricians. Simulations can be taught on computer programs, models and mannequins. Thus, intubation and cardiopulmonary resuscitations can be taught on mannequins. Mechanism of labour in various presentations and breech delivery can be taught on models (doll and dummy).
2. **Training programs** can improve doctor-patient relationship in obstetrics and can help in communication skills.

3. **Use of actors and actresses** to break bad news can sharpen the communication skills of young doctors.
4. **Courses.** The developed countries have developed courses for training in obstetric skills and drills. They are as follows:
   (*i*) Managing obstetric emergencies and trauma **(MOET, United Kingdom)**
   (*ii*) Advanced Life Support Training in Obstetrics **(ALSO, USA)**
   (*iii*) Advanced Labour and Risk Management (ALARM, Canada)

Unfortunately, such courses are not yet well establilshed in India. However, Federation of Obstetric and Gynecological Societies of India (FOGSI) and Indian College of Obstetricians and Gynecologists (ICOG) have started making guidelines for various obstetric conditions and have started accrediting centers for various sub-specialities (e.g. ultrasound courses, foetal medicine courses, etc.) to enhance skills in training of obstetricians in obstetrics.

## BIOMEDICAL WASTE MANAGEMENT IN OBSTETRICS

Biomedical waste means any waste generated during the diagnosis, treatment, immunization and research on humans or animals, activities including abortuses, placenta, discarded medicines and chemical waste, soiled linen, catheters, etc. **Biomedical Waste Management and Handling Rules 1998** were enacted by the Goverment of India to suitably dispose the biomedical waste to avoid its adverse effect on human health and environment. It was updated in 2016.

Biomedical waste should be segregated, packed, transported and stored properly before finally disposing it off. Biomedical waste is divided into 10 categories (Table 44.1). COVID-19 waste is also managed in same way.

**Table 44.1: Categorization of Biomedical Waste and Management**

| |
|---|
| **Category No. 1** |
| It includes **human anatomical waste** which is incinerated or buried deeply. |
| **Category No. 2** |
| It includes **animal waste** also treated like Category No. 1 |
| **Category No. 3** |
| It includes **microbiology and biotechnology wastes** treated by local autoclaving or microwaving or incineration. |
| **Category No. 4** |
| It includes **waste sharps** (e.g. needles, syringes, blades, scalpels, glass, etc.) which should be disinfected by chemical treatment or autoclaving or microwaving and mutilation and shredding. |
| **Category No. 5** |
| **Discarded medicines and cytotoxic drugs** are incinerated or disposed in secured landfills. |
| **Category No. 6** |
| **Solid waste contaminated with blood and body fluid** is incinerated or autoclaved. |
| **Category No. 7** |
| **Solid Waste.** Disinfection by chemical treatment/ autoclaving/shredding |
| **Category No. 8** |
| **Liquid waste.** Disinfection by chemical treatment (1% hypochlorite solution) and discharge into drains. |
| **Category No. 9** |
| **Incineration ash.** Municipal landfill |
| **Category No. 10** |
| **Chemical waste.** Chemical treatment and discharge into drains. |

### Colour coding

It is important to segregate biomedical waste and put them in properly colour-coded bags. The biomedical wastes and the colored bags in which they are to be put are as follows:

| | | |
|---|---|---|
| 1. **Yellow** (plastic bags) | Category 1, 2, 3, 6 | Incineration or plasma pyrolysis or deep burial |
| 2. **Red** (disinfected containers/ Plastic bags) | Category 3, 6, 7 | Autoclaving or chemical treatment or microwaving and then for recycling (not for land fill) |
| 3. **Black** (plastic bags) | Category 5, 9, 10 | Municipal waste |
| 4. **White/White translucent:** (puncture leak or tamper proof containers or plastic bags) | Category 4 (waste sharps including metals) and Category 7 | Autoclaving or microwaving or chemical treatment and destruction by shredding, mutilation or encapsulation |
| 5. **Blue:** Cardboard boxes with blue colored marking | Glassware | Disinfection, autoclaving, microwaving and then sent for recycling. |

There is punishment in form of imprisonment of 5 years and/or fine of one lakh rupees or both for not properly disposing off the biomedical wastes.

## UNIVERSAL PRECAUTIONS

These precautions should be followed strictly in all patients irrespective of their HIV status, especially while handling blood and various body fluids. They are as follows:

1. Use of gloves, plastic aprons, goggles, mask and occlusive band depending on the severity of risk.
2. Washing hands thoroughly with soap and water after contamination.
3. Avoiding needle prick or sharp injury.
4. Any blood or body fluid splashes on doctor's body should be rinsed thoroughly with plenty of running water.
5. All healthcare workers should be protected against Hepatitis B and tetanus immunization.
6. Management of accidental contaminated needle prick injury
   (*i*) Encourage the wound to bleed.
   (*ii*) Clean with soap and water thoroughly.
   (*iii*) Cover the cut part with waterproof dressing.
   (*iv*) In case of HIV positive, Anti-retroviral therapy (see Chapter 36 for details).

## Key Points

1. Audit is a methodical and systematic analysis of quality of healthcare. While research gives new results, audit tells us what we are doing is right or not. Any aspect of healthcare can be audited and patient care can be improved.
2. As obstetrics deals with two lives and there may be adverse maternal and perinatal outcomes, litigation is common. Maternal death, severe morbidity, cerebral palsy in child or foetal trauma are common causes of medicolegal cases.
3. Proper communication with patients and their husbands and family members in a honest and truthful way can help in reducing complaints. Informed written consent must be taken from all women before performing any procedure, except in certain emergency situtations when it becomes necessary to save a life and there is no time to get written consent. Proper maintenance of cases and notes is crucial.
4. Clinical risk management deals with early identification of adverse events and aims to prevent them from happening in future by making a team of workers who can identify, analyse and control risk.
5. PC PNDT Act was enacted to regulate and prevent misuse of prenatal diagnostic techniques and check sex-selective abortions. It laids down punishment to healthcare workers involved in it. The doctors can play deterrent role in stopping female feticide.
6. Ethics, bioethics, communication and counselling are emerging as very important aspect of healthcare. All obstetric patients have the fundamental right to have the information and evidence-based management offered to them during their obstetric care.
7. All obstetricians should care for their patients with empathy and scientifically after proper communication and counselling. The women should be involved in their care after proper explanation to them.
8. Skill training is very important in obstetrics to teach the young obstetricians various obstetric skills like instrumental and breech deliveries and cesarean section. Use of simulations is helpful.
9. It is the responsibility of all healthcare workers to properly segregate, pack, transport and store the biomedical waste. Proper disposal of biomedical waste is important for human health and environment. Animal and human wastes are incinerated. Proper colour coding of waste is important.

## Suggested Reading

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# Safe Motherhood, Epidemiology of Obstetrics, Maternal and Perinatal Mortality and Morbidity, Vital Statistics, Reproductive Morbidity and Health Programmes in Obstetrics

CHAPTER 45

## INTRODUCTION

**Quality of healthcare delivery system in a country as a whole is reflected by various vital statistics.** Maintenance of accurate vital statistics regarding vital events like births and deaths, analyzing them and taking preventive measures on their basis can lower maternal and foetal deaths.

## SAFE MOTHERHOOD INITIATIVE (SMI)

Disturbed by the high maternal mortality prevailing in developing countries of the world, WHO launched **'Safe Motherhood Initiative'** (SMI) at a conference in Nairobi (Kenya) in 1987. **The global objective was aimed at lowering of maternal deaths by at least half by** AD 2000. This deadline was then extended to 2010 and again to 2015. It was proposed to achieve this objective by enhancing the quality and safety of lives of girl children and women through a combination of health and non-health strategies. Maternal and Child Health is the area of priority for WHO.

### Strategies to Reduce Maternal Mortality and Achieve Safe Motherhood

1. Improve the status of women through education, health, career opportunities and vocational training to enable them to achieve economic independence and become equal partners in nation building.
2. Reduce high fertility through family planning.
3. Upgrade tertiary care hospitals and district hospitals to contemporary acceptable levels of healthcare. Make transfusion services more easily accessible through networking of blood banks.
4. Improve obstetric services by making antenatal care more easily accessible, encourage supervised institutional deliveries or home delivery under qualified supervision for low-risk cases. Strengthen referral systems and transport facilities to ensure timely transfer of high-risk patients.
5. Improve standard of care for women in labour.
6. Ensure easy accessibility of medical termination of pregnancy (MTP) services.
7. Maintain appropriate training of health personnel to deal with obstetric emergencies and to be equipped to undertake essential obstetric functions in primary healthcare centers and first referral units (FRUs).
8. Utilize services of media (television, radio, news media) to promote women's health awareness amongst the public.

From time to time the Government of India started many health programs to improve maternal and child health (see below).

### Epidemiology of Obstetrics

*Epidemiology in obstetrics deals with various vital statistics of maternal and perinatal outcomes* which are the sensitive index of the quality of healthcare delivery system of a country.

## VITAL STATISTICS

*Vital statistics relate to life and death events* and need standard and uniform definitions to allow comparison of data in different parts of the world.

### Maternal Mortality (death)

#### Definition

**As per the Tenth Revision of the International Statistical Classification of Diseases (ICD-10), a maternal death is defined as death of a woman while pregnant or within 42 days of termination of pregnancy irrespective of the site and duration of pregnancy from any cause related to or aggravated by pregnancy or its management but not from accidental or incidental causes.** Hence, deaths from abortion and ectopic pregnancy are included in it.

Maternal deaths are sub-divided into three sub-groups—**direct, indirect** and **incidental deaths** (see below).

**Table 45.1: Maternal Mortality Ratio (per 100,000 live births) in Different Countries**

| Developed countries | MMR | Developing countries | MMR |
|---|---|---|---|
| 1. United States of America | 23.8 | 1. India | 407 in 2000<br>212 in 2007–09 (Sample Registration System, 2011)<br>130 in 2014–2016 (SRS 2018),<br>113 in 2016–18 (SRS 16–18) |
| 2. Overall Europe | 16 | 2. Pakistan | 178 |
| 3. United Kingdom | 10 | 3. Bangladesh | 143 |
| 4. Japan | 5 | 4. Nepal | 258 |
| 5. Singapore | 10 | 5. Sri Lanka | 32 |
| | | 6. China | 27 |

NFHS-4: National Family Health Survey; UNICEF: United Nations International Children Education Fund, SRS 2011, 2018.

### Maternal Mortality Ratio (MMR)

Maternal mortality ratio is defined as the number of maternal deaths per 100,000 live births. It has reduced globally from 341 in 2000 to 246 in 2010 and 216 in 2015, from 352 in 2000 to 206 in 2010 and 164 in 2015 in South East Asia (It is only 16 in Europe, WHO 2015). In India, it has declined from 167 in 2011–2013 to 130 in 2014–2016 (Sample Registration System, Registrar General of India). It is lowest in Kerala (46) and highest in Assam (237) and is 201 in Uttar Pradesh. The denomination is large as number of maternal deaths are very few in developed countries.

### Maternal Mortality Rate

Maternal mortality rate is the number of maternal deaths per 100,000 women of reproductive age (15–45 years) and reflects the medical risk of pregnancy and level of fertility in a population. It was 113 per 100,000 live births in India in 2016-18 SRS (Sample Registration System) as compared with only 0.5 in USA.

### Incidence of Maternal Mortality

A comparison of maternal mortality rate in different countries is shown in Table 45.1. The maternal mortality is much higher in developing countries than developed countries.

Maternal mortality has dropped remarkably in the developed countries due to improved obstetric care, status of women, nutritional status, better sanitary and living conditions and due to spending of large sums of money towards welfare and healthcare in these countries. Surprisingly, there was a rise in maternal mortality in USA being 23.8 per 100,000 live births in 2017 which was highest in previous 40 years. It could be due to revised definition of maternal death, more reporting of maternal deaths, increase in mothers with severe chronic health conditions and increase in elderly mothers (> 40 years). The MMR was higher amongst blacks and Hispanics as compared to whites due to difference in availably, access or less and utilization of health services.

Sri Lanka has low MMR. Low maternal mortality of China is due to universal peoples' education on maternal and child health care (MCH), rise in age of marriage, use of family planning with one child norm and universal maternity care. **In India, MMR fell from 725 in 1982 to 582 in 1994, 407 in 2000, to 256 in 2006 and 230 in 2008, 167 in 2012 and to 130 in 2015.** Recent special bulletin on maternal mortality in India showed significant fall in maternal mortality to 130 per lakh live births with Kerala, Tamil Nadu and Maharashtra showing only 46, 66 and 61 MMR (Sample Registration System, 2018, Office of Registrar General, India).

### Causes of Maternal Mortality (Fig. 45.1)

Bhaskar Rao, FOGSI—1982, Bedi N, ICMR 1994, WHO 2015, NFHS-4, (2015–16)

#### I. Direct Maternal Deaths

Direct maternal deaths **relate to deaths resulting from complications of pregnancy, labour or the puerperium due to interventions,** omissions or incorrect treatments or from a chain of events resulting from any of the above. **Amongst the leading causes of direct maternal deaths are haemorrhage, sepsis, pre-eclampsia and eclampsia, abortions and obstructed labour. Almost 66% of maternal**

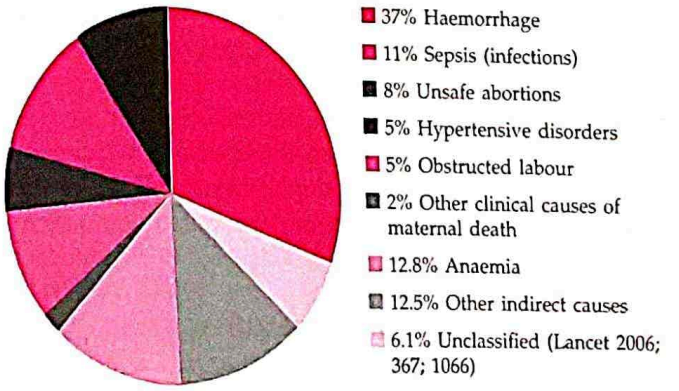



**Fig. 45.1:** Causes of maternal mortality (Source: WHO, UNICEF, UNFPA 2001).

**deaths are due to direct causes, most of which are otherwise largely preventable.**

1. ***Haemorrhage.*** The most common single preventable cause is haemorrhage. It is responsible for 25 to 50% (average 37%) deaths. Atonic postpartum haemorrhage is the most important cause followed by antepartum haemorrhage, bleeding from abortions, rupture uterus and ectopic pregnancy.
2. ***Sepsis.*** About 11% of maternal deaths are caused by sepsis like puerperal sepsis. Septic abortions are responsible for 8% of all maternal deaths.
3. ***Pre-eclampsia and eclampsia.*** These account for almost 5% of all maternal deaths with most women being without prenatal care.
4. ***Prolonged and obstructed labour.*** About 5 of women die as a result of prolonged, neglected and obstructed labour due to dehydration, exhaustion and infection. They may suffer from uterine rupture or septic or hemorrhagic shock.

### II. Indirect Maternal Deaths

Indirect maternal deaths include deaths resulting from previous existing diseases or diseases that developed during pregnancy but got worsened by physiological effects of pregnancy or stress of labour. **They cause about 34% maternal deaths.**

1. ***Anaemia.*** It accounts for (12–15%) of all maternal deaths and is a contributory factor in many other deaths. The women may die during pregnancy or labour due to congestive cardiac failure.
2. ***Hepatitis.*** In India, epidemics of infective hepatitis occur from time to time due to poor sanitation and hygiene. Pregnant women are 7–10 times more likely to suffer from hepatic failure, coma, coagulation failure, postpartum haemorrhage and death. During an epidemic, hepatitis may account for 10 to 15% of all maternal deaths. In some parts of North India like Delhi, hepatitis especially Hepatitis E is the leading cause of maternal mortality due to hepatic coma.
3. ***Heart disease.*** Heart disease accounts for 5% of all maternal deaths with rheumatic heart disease with a tight mitral stenosis being particularly vulnerable for mortality.
4. ***Tetanus.*** About 1% maternal deaths result from tetanus. Routine administration of tetanus toxoid to all pregnant women as part of prenatal care has helped to protect many lives.

### III. Incidental Deaths

Incidental maternal deaths are those deaths which result from accidents, malignant neoplasms, etc. They are not counted in maternal mortality and are called non-maternal deaths.

### Factors Affecting Maternal Mortality

Socio-economic causes like poverty, illiteracy and maternal health status are closely interlinked. All advanced countries have registered concurrent changes of lowering their birth rates by elevating the status of women who then participate in decision-making related to reproductive health like family size, contraception and MTP services.

A maternal death is usually the end result of inadequately or inappropriately managed complications arising during pregnancy and childbirth. The status of women in society plays a major role in their survival; being best in communities where women are educated, are aware of their rights and have an equal status in society as men like in western countries. Kerala and Tamil Nadu in India have lower mortality.

### Three Delays

Delays may occur between the time a woman develops a life-threatening complication and the time she receives appropriate treatment. **The first delay** is when complications arise, the woman and her family may not recognize the significance of the problem and may not seek medical care. **The second delay** may be related to the distance between the woman's home and the appropriate health facility, lack of transport, difficult access due to lack of or damaged roads, floods, etc. Finally, even after the woman reaches the facility, there may be delay in her receiving appropriate care for various reasons **(the third delay).**

### Interventions to Prevent Maternal Deaths

The following measures are to be taken to decrease maternal mortality ratio.

1. Promotion of family planning services, delay in age of marriage and child birth.
2. Early registration of pregnancy and hospital antenatal check-ups.
3. Dietary supplementation and prophylaxis for anaemia to be given to every pregnant woman and tetanus toxoid.
4. The presence of a skilled attendant at delivery and provision of safe abortion.
5. Drugs and supplies are essential in ensuring maternal survival.
6. A functional referral system to a facility which can provide comprehensive emergency obstetric care including cesarean section, laparotomy and blood transfusions.
7. Essential obstetric care should be provided at the doorstep of the pregnant women, i.e. at first referral level hospitals.
8. Medical disorders of pregnancy particularly anaemia, diabetes, hypertension and cardiac disease which are important indirect causes of maternal deaths should be managed jointly by different specialists in tertiary hospitals.
9. There should be provisions for good anaesthetic facilities, blood bank, specialist services in the labour room in the hospitals.
10. Maternal mortality review should be conducted regarding the cause of death and any avoidable factors and remedial measures should be taken from time-to-time to reduce maternal deaths. Confidential enquiries into maternal mortality in UK

has helped to lower maternal mortality in that country. Every maternal death is carefully assessed to find out the cause and to prevent it in future without penalizing the doctors. In United Kingdom, the reporting of maternal and infant deaths is now funded through the National Institute for Clinical Excellence (NICE) and is managed by a consortium of Royal Colleges (Royal College of Obstetricians and Gynaecologists, Midwives, Paediatrics, Pathologists, Anesthetists and Public Health). It is now called Mothers and Babies, Reducing Risks through Audits and Confidential Enquiries across the UK (MBRRACE-UK). It releases 'Saving Lives, Improving Mothers Care'. Its fifth annual report was published in 2018. In USA, CDC maintains it.

11. Training courses for training of health workers, traditional birth attendants and private practitioners should be arranged which will greatly help in reducing maternal mortality.
12. Foster partnerships among government, NGOs and healthcare providers.
13. Reviews of maternal deaths can help. They are as follows.
    (*i*) Community based maternal death reviews (verbal autopsies)
    (*ii*) Facility based maternal death reviews
    (*iii*) Confidential enquiries into maternal deaths
    (*iv*) Surveys of severe morbidity (near misses)
    (*v*) Clinical audit of deaths and near misses

## REPRODUCTIVE MORBIDITY

**Reproductive health** has been defined as a **'state of complete mental, physical and social well-being in all matters related to the reproductive system and to its functions and processes'.**

It is estimated that for each maternal mortality there are about 20 cases of morbidity causing long term sequelae like prolapse uterus, urinary incontinence, etc., causing immense suffering to women and economic loss to the country.

### Reproductive Morbidity

Reproductive morbidity refers to diseases affecting the reproductive system, although not necessarily as a consequence of reproduction and can go beyond reproductive years.

### Complications of Pregnancy and Delivery

Out of around 120 million births occurring per year in India, about half of them experience some complications (acute morbidity) and between 15 and 20 million develop disabilities (chronic morbidity).

### Acute Maternal Morbidity

Acute maternal morbidity conditions are acute conditions like antepartum haemorrhage, postpartum haemorrhage, obstructed labour, sepsis, etc.

### Chronic Maternal Morbidity

Chronic maternal morbidity are chronic conditions which are sequelae of child birth like genital prolapse, obstetric fistula, urinary stress incontinence, pelvic inflammatory disease, abnormal uterine bleeding, Sheehan's syndrome, infertility, choriocarcinoma, etc.

### Direct Obstetric Morbidity

Direct obstetric morbidity are the conditions occurring during pregnancy or labour and are directly related to obstetrics like haemorrhage (antepartum and postpartum), puerperal sepsis, hypertensive disorders, complications of abortion (spontaneous or induced), e.g. Sheehan's syndrome. Choriocarcinoma, obstetric fistulae and uterovaginal prolapse.

### Indirect Obstetric Morbidity

The indirect obstetric morbidity is not caused but worsened by pregnancy like malaria, tuberculosis, hepatitis, etc., which are more serious during pregnancy. There may be exacerbation of disease like rheumatic heart disease, chronic hypertension, sickle-cell anaemia and breast cancer during pregnancy.

### Non-Obstetric Morbidity

Non-obstetric morbidity are conditions which may coincidentally occur during pregnancy, delivery or puerperium but unrelated to pregnancy like an attempted suicide or burn accidents.

## PERINATAL MORTALITY

**Perinatal death is defined as death of fetus during the perinatal period which is from 28 weeks (24 weeks in UK) of gestational age or more than 1000 g birth weight to first week of neonatal life.** It includes late foetal deaths in utero, stillbirths and early neonatal deaths. **Perinatal mortality rate (PNMR) is defined as number of such deaths per 1000 live births.**

Perinatal mortality provides a better insight into the likely hazards facing foetal life at the time of childbirth and reflects the quality of medical care available in the community.

### Incidence of Perinatal Mortality Rate (PMR or PNMR)

The incidence of PNMR in India has decreased from 150 to 48.5/1000 births in 2005–2006 (NFHS-3) and to 26/1000 in 2013 (16/1000 in urban areas, 28 per 1000 in rural areas) but is still much higher than that of 10 to 20 per 1000 births in the developed countries (9.98 in USA in 2013). About 60% of all perinatal deaths occur in low birth weight babies. PNMR is higher in rural India as compared to urban centers in our country.

### Factors affecting perinatal mortality

#### Maternal Age

Obstetric risks are the lowest between the ages of 20 and 30 years. The younger age groups are susceptible to preterm births, illegitimate pregnancies, inadequate prenatal care and cephalopelvic disproportion during labour. The elderly patients are more prone to hypertension, gestational

diabetes, renal disease, incoordinate uterine activity, non-progress of labour, dystocia and foetal distress.

PARITY

**The lowest perinatal mortality is in the second pregnancy.** The highest incidence in the primigravida is due to pre-eclampsia and a higher incidence of dystocia during labour. *Pregnancy-related risks start increasing after three deliveries.* Pregnancies in quick succession, anaemia, inadequate prenatal care, inadequate rest, malpresentations, preterm births and a higher incidence of multiple births cause increased perinatal deaths.

SOCIO-ECONOMIC FACTORS

It is higher in poor women.

### Causes of Perinatal Mortality

The causes of perinatal mortality including neonatal deaths are shown in Table 45.2B.

**Table 45.2B: Common Causes of Perinatal Morbidity**

1. Prematurity (about 18%)
2. Infections (16%)
3. Birth asphyxia (10%)
4. Congenital anomalies (5%)
5. Foetal (intrauterine) growth restriction (low birth weight babies)
6. Birth trauma
7. Respiratory distress syndrome
8. Metabolic and other problems
9. Miscellaneous causes

In United Kingdom, **Confidential Enquiries into Still Births and Deaths in Infancy (CESDI)** used to audit all perinatal deaths trying to find causes and prevent them. Now **Mothers and Babies Reducing Risk through Audit and Confidential Enquiries across the UK** (MBRRACE-UK) maintains it through 'Saving Lives Improving Mothers Care'. Its fifth report was released in 2018. In USA, CDC maintains it.

### Wrigglesworth Classification

**Category 1** Congenital defect or malformation (lethal or severe)
**Category 2** Unexplained antepartum foetal death
**Category 3** Death from intrapartum asphyxia, anoxia or trauma.
**Category 4** Immaturity
**Category 5** Infection where there is clear microbiological support for the diagnosis.
**Category 6** Other specific causes
**Category 7** Accident or non-intrapartum trauma
**Category 8** Sudden infant death, cause unknown
**Category 9** Unclassifiable (this category is used only as a last resort)

### Strategies to Reduce Perinatal Mortality

1. Improving the standards of living
2. Raising the social status of women
3. Universal prenatal care for all pregnant women.
4. High-risk approach to pregnancy care
5. Strengthening the referral system
6. Facilitating early transport of high-risk mothers to suitable centers.
7. Raising the standards of intrapartum care, e.g. by adopting partograms.
8. Better neonatal care and establishment of neonatal intensive care units.
9. Maternal steroid administration in preterm labour reduces perinatal mortality **(level IA evidence).**
10. Maternal dietary supplementation of malnourished women reduces perinatal mortality **(level IB evidence).**

### Under Five Child Mortality

As per WHO-CHERG 2012 estimates, the causes of child mortality in the age group 0–5 years in India are:

1. Neonatal causes (52%)
2. Pneumonia (15%)
3. Diarrhoeal disease (11%)
4. Measles (3%)
5. Injuries (4%) and
6. Others (15%)

## PERINATAL MORBIDITY

The major illnesses of neonates from birth to first four weeks of life are included under perinatal morbidity. Major causes are prematurity, growth restriction, perinatal asphyxia, trauma, malformations and infections.

# HEALTH PROGRAMMES IN OBSTETRICS

## REPRODUCTIVE AND CHILD HEALTH (RCH) PROGRAMME

**Reproductive and Child Health (RCH) is defined as 'people have the ability to reproduce and regulate their fertility, women are able to go through pregnancy and child birth safely and couples are able to have sexual relations free of fear of pregnancy and of contracting disease'. The RCH programme is described below.**

## THE REPRODUCTIVE AND CHILD HEALTH (RCH) PHASE I PROGRAMME

**RCH phase 1 programme was started by the Government of India in 1997 to lower maternal mortality to 100/100,000 births by 2010.** It had four components: Family Planning, Child Survival and Safe Motherhood, Client Approach to Healthcare, and Prevention and Management of Reproductive Tract Infections (RTIs) and AIDS.

## THE REPRODUCTIVE AND CHILD HEALTH PHASE-II PROGRAMME

**RCH phase II programme was started on 1 April, 2005 with an aim to reduce maternal and child morbidity and mortality with emphasis on rural healthcare.**

## The Major Strategies under the RCH phase II

**1. Essential Obstetric Care**

(*i*) Institutional delivery
(*ii*) Skilled attendance at delivery
(*iii*) Permitting auxillary nurse midwives (ANMs) to use life-saving drugs and to carry out certain emergency interventions.

**2. Emergency Obstetric Care**

(*i*) **Operationalizing first referral units** (FRUs) with skilled attendance at birth. The minimum services to be provided by a fully functional FRU are:

a. 24-hour delivery services, including normal and assisted deliveries
b. Emergency obstetric care, including surgical interventions like cesarean sections.
c. Newborn care
d. Emergency care of sick children.
e. Full range of family planning services including laproscopic sterilization services.
f. Comprehensive Abortion services:
   - Provision of Manual Vacuum Aspiration (MVA) facilities and medical methods of abortion 24 × 7 in Primary Health Centres.
   - MTP services are to be made available in all District Hospitals and Sub-district level hospitals with priority given to 'delivery points'
   - Medical Abortion Drugs (Mifepristone + Misoprostol for upto 7 weeks and Ethacridine lactate for 12 to 20 weeks) are to be included in the essential drug list.

   Comprehensive and safe abortion services are provided at public health facilities including 24 × 7 PHCs/FRUs (DHs/SDHs/CHCs) including the Delivery Points. Supply of Nischay Pregnancy detection kits to sub centres for early detection of pregnancy is undertaken.

   **Nischay-Pregnancy Detection Kit**
   - To detect pregnancy at the earliest
   - To save lives lost to unsafe abortion practices
   - Available at the sub-centre level and with ASHA

   Capacity Building of Medical officers is carried out routinely in safe MTP Techniques. ANMs, ASHAs and other field functionaries are trained to provide confidential counselling for MTP and promote post-abortion care including adoption of contraception.

   Routine orientation and training of ASHAs to equip them with skills to create awareness on abortion issues in women and the community and facilitate women in accessing services is undertaken.
g. Treatment of sexually transmitted infections (STI)/reproductive tract infections (RTI).
h. Blood storage facility at all FRUs and above
i. Essential laboratory services
j. Referral (transport) services

(*ii*) Operationalizing peripheral health centers (PHCs) and community health centers (CHCs) for round-the-clock delivery services.

3. **Strengthening referral system** by involving local self-help groups, non-government organizations (NGOs) and women groups.

**Village Health and Nutrition Day:** Organizing of Village Health and Nutrition Day (VHNDs) at Anganwadi center at least once every month to provide ante natal/post partum Planning and nutrition are the part of various services being provided during VHNDs.

**Newer Interventions:** Screening of Gestational Diabetes Mellitus, screening for Hypothyroidism for high risk group during pregnancy, De-worming during pregnancy, Medical Methods of Abortion, birth companion during delivery, Maternal Near Miss programme and Technical and operational Guidelines for screening for Syphilis during pregnancy are newer initiatives to improve Maternal Health services.

## Misoprostol Distribution

Time of distribution - 8th month of pregnancy
Dose - 3 tablets of 200 mcg each (600 mcg)
Counselling - twice at least at 1 week's interval

## New Initiatives

1. **Training of MBBS doctors** in life-saving anaesthetic skills for emergency obstetric care. Government of India is also introducing training of MBBS doctors in obstetric management skills. Federation of Obstetric and Gynecological Society of India (FOGSI) has prepared a training plan for 16 weeks in all obstetric management skills, including cesarean section operation.
2. **Setting up of blood storage centers** at FRUs according to Government of India guidelines.
3. **Provision of RTI/STI services:** Under NHM (National Health Mission), provision of STI/RTI care services is a very important strategy to prevent HIV transmission and promote sexual and reproductive health under the Naitonal AIDS Control Program (NACP III) and Reproductive and Child Health (RCH II). Enhanced syndromic case management (ESCM) with minimal laboratory tests is the cornerstone of STI/RTI management under NACP III. Services are being provided to all FRUs, CHCs and at 24 × 7 PHCs through various kits of different colors for different STIs (Table 45.3).

### Janani Suraksha Yojana (JSY) (April 2005)

It was started to reduce maternal and infant mortality through encouraging delivery at health institutions and focussing at institutional care among women in below poverty line families.

Features of Janani Suraksha Yojana

(*i*) JSY is 100% centrally sponsored scheme.
(*ii*) Under National Rural Health Mission, it integrates the benefit of cash assistance to mothers for institutional deliveries as shown in Table 45.4. The Accredited Social Health Activist (ASHA) works

**Table 45.3: Various kits available through NACP III for varoius STIs/RTIs**

| STI/RTI Syndromic diagnosis | Kit prescribed | Name of drugs |
|---|---|---|
| Urethral discharge/anorectal discharge/ cervical discharge/painful scrotal swelling/presumptive t/t | Kit-1 Gray | Azithromycin 1 g stat<br>Cefixime 400 mg stat |
| Vaginal discharge (vaginitis) | Kit-2 Green | Secnidazole 2 g stat<br>Fluconazole 150 mg stat |
| Genital ulcer disease: non herpetic | Kit-3 White | Benzathine penicillin 2.4 MU im stat<br>Azithromycin 1 g stat |
| Genital ulcer disease: non herpetic (allergic to penicillin) | Kit-4 Blue | Azithromycin 1 g stat<br>Doxycycline 100 mg BD 14 days |
| Genital ulcer disease: herpetic | Kit-5 Red | Acyclovir 400 mg TDS 7 days |
| Lower abdominal pain (PID) | Kit-6 Yellow | Cefixime 400 mg stat<br>Doxycycline 100 mg BD 14 days<br>Metro nidazole 400 mg BD 14 days |
| Inguinal Bubo | Kit-7 Black | Azithromycin: 1 gm OD stat<br>Doxycycline 100 mg BD for 21 days |

**Table 45.4: Financial Assistance to Mothers for Institutional Delivery (in Rupees)**

| | Rural Area | | | Urban Area | | |
|---|---|---|---|---|---|---|
| Category | Mother's package | ASHA's package* | Total Rs. | Mother's package | ASHA's package** | Total Rs. |
| LPS | 1400 | 600 | 2000 | 1000 | 400 | 1400 |
| HPS | 700 | 600 | 1300 | 600 | 400 | 1000 |

LPS: Low performing states (e.g. Uttar Pradesh, Bihar), HPS: High performing states (e.g. Delhi, Kerala)
Note: In LPS, all women are given cash incentive while in HPS only below poverty line (BPL), SC and ST women are given cash incentive
* ASHA package of ₹ 600 includes ₹ 300 for ANC and ₹ 300 for facilitating institutional delivery
** ASHA package of ₹ 400 includes ₹ 200 for ANC and ₹ 200 for facilitating institutional delivery

as a link health worker between the women and government.

During the year 2006–2008, about 28.11 lakh pregnant women benefited from the scheme, out of which 18.72 lakhs had institutional deliveries.

### Janani Shishu Suraksha Karyakram (JSSK)

Government of India has launched Janani Shishu Suraksha Karyakaram (JSSK) on Ist June, 2011, which entitles all pregnant women delivering in public health institutions to absolutely free and no expense delivery including Cesarean section.

The initiative stipulates free drugs, diagnostics, blood and diet, besides free transport from home to institution, between facilities in case of a referral and drop back home.

Similar entitlements have been put in place for all sick new-borns accessing public health institutions for treatment till 30 days after birth. In 2013 this has been expanded to Sick infants and antenatal and postnatal complications.

The following are the Free Entitlements for pregnant women:

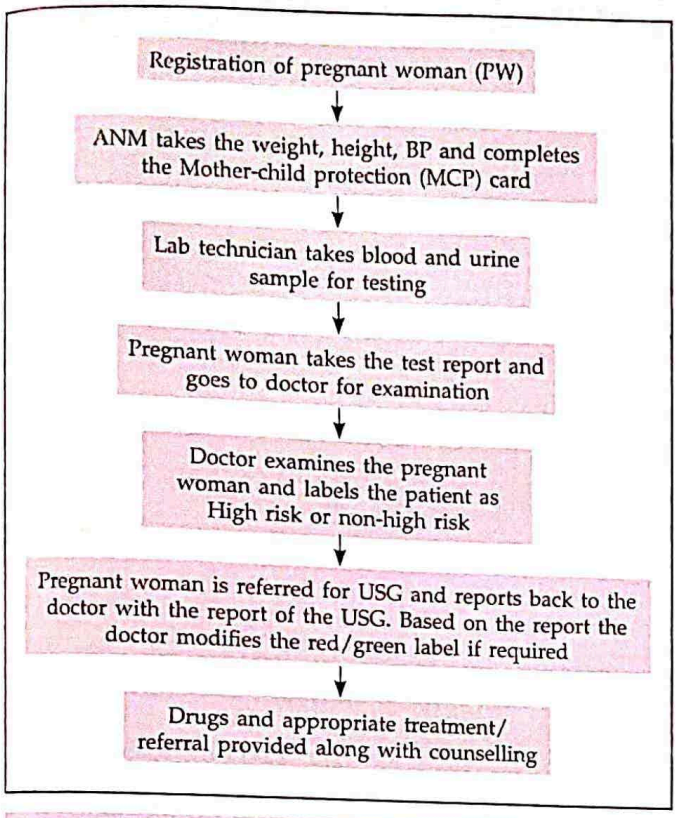
- Free and cashless delivery
- Free cesarean section
- Free drugs and consumables
- Free diagnostics
- Free diet during stay in the health institutions
- Free provision of blood
- Exemption from user charges
- Free transport from home to health institutions
- Free transport between facilities in case of referral
- Free drop back from Institutions to home after 48 hrs. stay

### Pradhan Mantri Surakshit Matritva Abhiyan (PMSMA)

Carrying forward the vision of our Hon'ble Prime Minister, the Pradhan Mantri Surakshit Matritva Abhiyan was launched in 2016 to ensure quality antenatal care to pregnant women in the country on the 9th of every months. Package of services:

- Routine antenatal check-up
- Diagnostic services
- Identification and management of high risk pregnancy
- Counselling for nutrition, family planning, birth preparedness, newborn and postnatal care
- Other-communication for behavioural change health system strengthening for providing quality services.

Referral transport. It is shown in Flow Chart 45.1.




**Flow chart 45.1:** Flow of beneficiaries on the 9th of every month, i.e., on the day of PMSMA

## MAA (Mother's Absolute Affection) Programme

MAA is a countrywide intensified breast-feeding promotion campaign by Ministry of Health stated on 5th August 2016 targeting

- All states and union territories
- Around 3.9 crore pregnant and lactating mothers
- 8.8 lakh ASHAs
- 1.5 lakhs subcentres
- 17,000 Birthing Facilities/Delivery Points

The goal of the 'MAA' Programme is to revitalise efforts towards promotion, protection and supprt of breastfeeding practices through health systems to achieve higher breastfeeding rates through mass media, orietation of ASHAs giving incentive to ASHA.

## Components

1. Early initiation of breastfeeding; immediately after birth, preferably within one hour.
2. Breast-milk alone is the best food and drink for an infant for the first six months of life. No other food or drink, not even water.
3. After 6 months of age, babies should be introduced to semi-solid, soft food (complementary feeding) but breastfeeding should continue for up to two years.
4. From the age of 6–8 months a child needs to eat two to three times per day and thereafter, three to four times per day starting at 9 months – in addition to breastfeeding.
5. During illness, children need additional fluids and encouragement to eat regular meals and breastfeeding infants need to breastfeed more often. After illness, children need to be offered more food than usual, to replenish the energy and nourishment lost due to the illness.
6. Benefits of breastfeeding to the body and mother as below:

**(a) Benefits for the baby**

(i) Early skin-to-skin contact keeps the baby warm.
(ii) It helps in early secretion of breast milk.
(iii) Feeding first milk (colostrum) protects the baby from diseases.
(iv) Helps mother and baby to develop a close and loving relationship.
(v) Decreased risk of illness such as diarrhea, pneumonia, ear and throat infections.
(vi) Improved intelligence.
(vii) Ensures development and growth.

**(b) Benefits for the mother**

(i) Helps womb to contract and the placenta is expelled easily.
(ii) Reduce the risk of excessive bleeding after delivery
(iii) Reduces the risk of breast cancer, uterine cancer and ovarian cancer
(iv) Lessens osteoporosis
(v) Benefits child spacing
(vi) Promotes post-partum weight loss
(vii) Financially viable

## LaQshya

In order to further accelerate our decline in maternal and newborn mortality in the coming years, Health Ministry has recently launched 'LaQshya - Labour room Quality Improvement Initiative'. LaQshya program is a focused and targeted approach to strengthen key processes related to the labour rooms and maternity operation theatres which aims at improving quality of care around birth and ensuring Respectful Maternity Care (RMC). Launched on November 2017 by Ministry of Health and Family Welfare, Govt. of India.

1. **To reduce maternal and newborn mortality and morbidity** due to APH (Antepartum Haemorrhage), PPH (Postpartum Hemmorrhage), Retained placenta, Preterm labour, Pre-eclampsia and Eclampsia, Obstructed labour, Puerperal sepsis, Newborn asphyxia, and Sepsis, etc.
2. **To improve Quality of care**
   (*i*) During the delivery
   (*ii*) Stabilization of complications
   (*iii*) Enable an effective two-way follow-up system
   (*iv*) Immediate post-partum care
   (*v*) Ensure timely referrals
3. **To enhance satisfaction of beneficiaries** visiting the health facilities and provide Respectful Maternity Care (RMC) to all pregnant women attending the public health facility (Table 45.5).

**Quality Improvement (QI) Cycles:** The Fulcrum of LaQshya: The initiative prioritizes local problem

**Table 45.5 Recommended Minimal Human Resources for the Labour Rooms of CHC/SDH/DH/Medical Colleges under LaQshya**

| No. of Deliveries (per month) | Staff Nurse (with LDR) | Staff Nurse (without LDR) | MO | House-keeping | DEO | Guard |
|---|---|---|---|---|---|---|
| 100–200 | In LDR facility there should be 4 staff nurses per LDR unit (1 for each shift and 1 back up) | 8 | 4 MO, 1 OBG/EmoC, 1 Anesthetist/LSAS, 1 Pediatrician | 4 | 1 | 4 |
| 200–500 | | 12 | 1 OBG (Mandatory) + 4 OBG/EmoC +1 Anesthetist +4 LSAS +1 Pediatrician +4 MO | 8 | 1 | 6 |
| > 500 | | 16 | 3 OBG (Mandatory) + 4 EmoC +1 Anesthetist +4 LSAS +1 Pediatrician +4 MO | 12 | 1 | 8 |

LDR: Labour Delivery Recovery, LSAS: Life Saving Anaesthetic Skills; EMOC: Emergency Obstetric Care, MO: Medical Officer; OBG: Obstetrician and Gynecologist, CHC: Community Health Centre; SDH: Sub District or Divisional Hospital, DH: District Hospital; DEO: Data Entry Operator

solving thereby ensuring ownership and accountability at the facility level through formation of Quality Circles and Quality Teams at the intervention facilities. QI methodologies (Plan-Do-Check-Act [PDCA] cycle) will be used to drive and sustain change through 6 defined QI cycles.

1. Real-time Partograph generation, usage of safe birth and surgical safety check-list and strengthening documentation practices for generating robust data for driving improvement
2. Presence of birth companion during delivery, Respecful Maternity Care and enhancement of patients' satisfaction
3. Assessment, triage and timely management of complications including strengthening of referral protocols
4. Management of Labour as per protocols including Active Management of Third Stage of Labour (AMTSL) and rational use of Oxytocin
5. Essential and emergency care of newborn and pre-term babies including management of birth asphyxia, timely initiation of breast feeding as well as Kangaroo Mother Care (KMC) for pre-term newborn.
6. Infection Prevention including Biomedical Waste Management

The Quality Circles will work on the selected themes and improve processes using QI methodologies.

Labour Room Quality Circle:

- Training
- IT (Information technology) Tools
- HR (Human resource)
- Quality Tools

Table 45.6 shows Dos and Don't of LaQshya.

**4. Vandemataram Scheme**

Vandemataram scheme is a voluntary scheme wherein the private doctors can volunteer themselves for providing safe motherhood services. The enrolled doctors will display 'Vandemataram logo' at their clinic. Iron and folic acid tablets, oral pills, tetanus injections, etc., will be provided by the District Medical Officers for free distribution to beneficiaries. The cases needing special care and treatment can be referred to the government hospitals, who have been advised to take due care of the patients coming with Vandemataram cards.

**5. National Rural Health Mission (NRHM, 2005–12)**

NRHM was started on 12 April 2005 by the Government of India to deliver health services for 'underprivileged' as a strategy for 'Health For All'. The objectives are:

1. To provide integrated comprehensive primary health care services.
2. For horizontal integration of vertical national health programs.
3. To cover all the villages in 18 states through, approximately, 2.5 lakh village-based Accredited Social Health Activists (ASHA) (see below).
4. NRHM has started with the activities of selection and training of ASHA.

**Expected outcomes of NRHM:**

(*i*) Infant mortality rate (IMR) to be lowered to 30/1000 by 2012
(*ii*) Maternal mortality rate (MMR) to be lowered to 100/100,000 live births by 2012
(*iii*) Total fertility rate (TFR) to be 2.1 by 2012
(*iv*) Sex ratio (0–6 years) to be 935 by 2012
(*v*) Increasing utilization of FRUs-bed occupancy by referred cases from less than 20% to over 75%.

**Table 45.6 Dos and Don't of LaQshya**

| | Dos | Don'ts |
|---|---|---|
| 1. | Providing privacy to pregnant women during the intrapartum period, by way of separate labour room or at least a private cubicle | Induction and augmentation of labour without sound clinical indications |
| 2. | Presence of birth companion during the labour | Any verbal or physical abuse of the pregnant women |
| 3. | Freedom to choose a comfortable position during birthing (squatting, standing, etc.) | Insisting on conventional lithotomy position for the delivery |
| 4. | Adherence to clinical protocols for management of labour | Immediate clamping and cutting of the umbilical cord |
| 5. | Use of Labour beds instead of tables | Separating baby from the mother for routine care and procedure |
| 6. | Place baby on mother's abdomen | 'Out of Pocket Expenditures (OOPE) on drugs, diagnostics, including demand by the staff for gratuitous payment by families for celebration of the baby's birth. |
| 7. | Initiation of Breast feeding within one hour of birth | |

### 6. NRHM Extended (NRHM [National Rural Health Mission] + NUHM [National Urban Health Mission] = NHM [National Health Mission]) ASHA (Accredited Social Health Activist)

ASHA - One of the key components

To provide every village in the country with a trained female community health activist (ASHA) or Accredited Social Health Activist. Selected from the village itself and accountable to it, the ASHA will be trained to work as an interface between the community and the public health system. Following are the key components of ASHA:

- ASHA must primarily be a woman resident of the village married/ widowed/divorced, preferably in the age group of 25 to 45 years.
- She should be a literate woman with due preference in selection to those who are qualified upto 10 standard wherever they are interested and available in good numbers. This may be relaxed only if no suitable person with this qualification is available.
- ASHA will be chosen through a rigorous process of selection involving various community groups, self-help groups, Anganwadi Institutions, the Block Nodal officer, District Nodal officer, the village Health Committee and the Gram Sabha.
- Capacity building of ASHA is being seen as a continuous process. ASHA will have to undergo series of training episodes to acquire the necessary knowledge, skills and confidence for performing her spelled out roles.
- The Ashas will receive performance-based incentives for promoting universal immunization, referral and escort services for Reproductive and Child Health (RCH) and other healthcare programmes, and construction of household toilets.
- Empowered with knowledge and a drug-kit to deliver first-contact healthcare, every ASHA is expected to be a fountain head of community participation in public health programmes in her village.
- ASHA will be the first port of call for any health related demands of deprived sections of the population, especially women and children, who find it difficult to access health services.
- ASHA will be a health activist in the community who will create awareness on health and its social determinants and mobilise the community towards local health planning and increased utilisation and accountability of the existing health services.
- She would be a promoter of good health practices and will also provide a minimum package of curative care as appropriate and feasible for that level and make timely referrals.
- ASHA will provide information to the community on determinants of health such as nutrition, basic sanitation and hygienic practices, healthy living and working conditions, information on existing health services and the need for timely utilisation of health and family welfare services.
- She will counsel women on birth preparedness, importance of safe delivery, breast-feeding and complementary feeding, immunization, contraception and prevention of common infections including Reproductive Tract Infection/Sexually Transmitted Infections (RTIs/STIs) and care of the young child.
- ASHA will mobilise the community and facilitate them in accessing health and health related services available at the Anganwadi/sub-centre/primary health centers, such as immunisation, Ante Natal Check-up (ANC), Post Natal Check-up supplementary nutrition, sanitation and other services being provided by the government.
- She will act as a depot holder for essential provisions being made available to all habitations like Oral Rehydration Therapy (ORS), Iron Folic Acid Tablet (IFA), chloroquine, Disposable Delivery Kits (DDK), Oral Pills and Condoms, etc.
- At the village level it is recognised that ASHA cannot function without adequate institutional support. Women's committees (like self-help groups or women's health committees), village Health and Sanitation Committee of the Gram Panchayat, peripheral health

workers especially ANMs and Anganwadi workers and the trainers of ASHA and in-service periodic training would be a major source of support of ASHA.

## NATIONAL DIGITAL HEALTH MISSION

It was started on 15th August by the PM to create a national digital health ecosystem that supports universal health coverage by creating national health ID for every Indian. Its implementation will be done in 3 phases and with the pilot phase already started.

1. It comprises of six key **building blocks.**
   (i) **Health Data:** Health Care provider facility will enrol in NDHM Registry to become Health Information Providers (HIPs); who will keep a digital copy of patients' personal health records, HIUs (Health information users) are the individuals/organisations who will get access to health data if consent is given by the individual.
   (ii) **Health ID:** Single health ID and unique health ID based on Aadhaar can be created by self from a mobile or a web application or by health care facility/organisation.
   (iii) **Health Registries:** Health Workforce Registry will have information about doctors, nurses, paramedical staff, ASHAs etc. through Digi Doctor platform. Healthcare Facility Registry will provide information about healthcare facility including hospitals, clinics, diagnostic centres, pharmacies etc.
   (iv) **Health Claims:** Standard e-Claim form can be used for any health insurance claim through a Health Claims Platform (HCP)
   (v) **Health Data Analytics:** Every HIP is expected to generate aggregated data on the health information that is being managed by them.
   (vi) Open Telemedicine and e-Pharmacy Network
2. **The National Health Authority (NHA)** has been given the mandate to design, build, roll-out and implement the mission in the country.
3. The core building block of the mission is that the health ID, DigiDoctor and Health Facility Registry shall be owned, operated and maintained by the Government of India.
4. Private stakeholders will have an equal opportunity to integrate and create their own products for the market. The core activities and verifications, however, remain with the government.
5. The citizens will be able to give their doctors and health providers one-time access to this data during visits to the hospital for consultation.

### RMNCH+A (Reproductive, Maternal Newborn, Child and Adolescent Health)

The Government of India adopted the Reproductive, Maternal, New-born, Child and Adolescent Health (RMNCH+A) framework in 2013 and it essentially looks to address the major causes of mortality among women and children.

### 3 Year Action Agenda 2017–20

Launched on August 2017 by Niti Ayog

- Reprioritize goals
- Healthcare system in the country must prioritize public health
- Shift from being curative to preventive.

Health Goals to be Achieved by the Year 2020 (Table 45.6A)

**Table 45.6A: Health Goals to be Achieved by the Year 2020**

| S.N. | Indicator | Goal |
|---|---|---|
| 1. | MMR | 120/100,000 live births |
| 2. | IMR (Infant mortality rate) | 30/1,000 live births |
| 3. | U-5 mortality rate | 38/1,000 live births |
| 4. | Total fertility rate | 2.1 |
| 5. | % of underweight children | Reduced by 3 point per annum from NFHS-4 |
| 6. | Reduce incidence of TB to 130/100,000 (2015 estimate: 217/100,000) | |
| 7. | Reduce incidence of Malaria (Annual Parasite Incidence) to less than 1/1,000 in 90% of districts | |
| 8. | Eliminate Kala Azar and Lymphatic Filariasis | |
| 9. | Reduce premature mortality from cardiovascular diseases, cancer, diabetes or chronic respiratory diseases by 1/4th of National Family Health Survey-4 (NFHS-4) levels | |
| 10. | Reduce Out-of-Pocket Spending (OOPS) to 50% of the total health expenditure | |

## INTENSIFIED NATIONAL IRON PLUS INITIATIVE (I-NIPI)

The reduction of anaemia is one of the important objectives of the POSHAN Abhiyaan launched in March 2018. Complying with the targets of POSHAN Abhiyaan and National Nutrition Strategy, intensified Iron-Plus Initiative (I-NIPI) strategy of the Anaemia Mukt Bharat Compaign has been designed to reduce prevalence of anaemia by 3% per year among six target beneficiary groupsie. Children, adolescent boy and girls, women in the reproductive groups (20–24 years), through six interventions and six institutional mechanisms.

Approximately 450 million beneficiaries - nearly 50% of the country's population will be reached

### Prevalence of Anaemia in India and Targets

According to the National Family Health Survey 4 (NFHS-4), 2015/16, anaemia prevalence across all ages is extremely high in India being 50% in pregnanr women and 25% in lactating women.

The target of Anaemia Mukt Bharat is to reduce it to 32 percent and 40% respectively by 2022.

The various programs on anaemia are shown in Table 45.6B.

| Table 45.6B: Evoluiton of Anaemia Control Program | |
|---|---|
| 1970 | National Nutritional Anaemia Prophylaxis Program (NNAPP)<br>60 mg iron supplementation for PW and 20 mg for 1–5 yr × 100 days |
| 1991 | National Nutritional Anaemia Control Program (NNACP)<br>60 mg iron changed to 100 mg |
| 2007 | 5–10 years age group added |
| 2013 | National Iron Plus Initiative (NIPI)<br>Weekly and biweekly supplementation.<br>Test and treat, Life cycle approach |
| 2018 | Anaemia Mukt Bharat- I-NIPI Program Intensification<br>PW 60 mg × 180 days, IFS for WRA 6 × 6 × 6 strategy, Life Cycle Approach |

## Six Interventions

Six Interventions are as follows:

1. Prophylactic Iron and Folic Acid supplementation (Table 45.6C)
2. Deworming (Table 45.6D)
3. Intensified year-round Behaviour Change Communication Campaign (Solid Body, Smart Mind) foucsing on four key behaviours
   - Improving compliance to Iron Folic Acid supplementation and deworming
   - Appropriate infant and young child feeding practices,
   - Increase in intake of iron-rich food through diet diversity/quantity/frequency and/or fortified foods with focus on harnessing locally available resources.
   - Ensuring delayed cord clamping after delivery (by 3 minutes) in health facilities

   Intensified 360-degree Information Education Communication (IEC)/Behaviour Change Communication (BCC) for Anaemia Prevention and Behaviour Change.

   Focus on Social mobilization and behaviour change: 4 key behaviours
   (i) Compliance to Iron Folic Acid supplements and deworming
   (ii) Appropriate Infant and Young Child Feeding (IYCF)
   (iii) Increase intake of iron-rich, protein-rich and vitamin C rich foods through diet diversification and consumption of fortified foods.
   (iv) Practice of delayed cord clamping in all health facility deliveries followed by early initiation of breastfeeding within 1 hour of birth
4. Testing and treatment of anaemia, using digital methods and point of care treatment, with special focus on pregnant women and school-going adolescents.

   **Test and Treat Strategy:**
   (i) Use of digital hemoglobinometers.
   (ii) In two age group to begin with school going Adolescent girls and boys 10–19 years, Weekly Iron Folic Acid Supplementation (WIFS) beneficiaries, using Rashtriya Bal Swasthya Karyakram (RBSK) mobile teams.
   (iii) Pregnant women at all ANC contact points.
   (iv) At all high case load facilitie at block level and above, hemoglobin level estimation will be done using Semi-Auto Analyzers.
   (v) This may be extended to all age groups, later.
5. Mandatory provision of Iron and Folic Acid fortified foods in government-funded public health programs (Table 45.6E)
6. Intensifying awareness, screening and treatment of non-nutritional causes of anaemia in endemic pockets with special focus on malaria, haemoglobinopathies and fluorosis.

   Integrated malaria and anaemia testing in endemic regions, beneficiaries who report with recent fever

| Table 45.6C: Prophylactic Dose and Regime for Iron Folic Acid Supplementation | |
|---|---|
| **Age Group** | **Dose and Regime** |
| School-going/Out-of-school adolescent girls, 10–19 years of age | **Weekly**, 1 Iron and Folic Acid tablet each tablet containing 60 mg elemental iron + 500 mcg Folic Acid, sugar-coated, BLUE COLOUR |
| Women of reproductive age (non-pregnant, non-lactating) 20–49 years | **Weekly**, 1 Iron and Folic Acid tablet each tablet containing 60 mg elemental Iron + 500 mcg Folic Acid, sugar-coated, RED COLOUR |
| Pregnant women and lactating mothers (of 0–6 months child) | **Daily**, 1 Iron and Folic Acid tablet starting from the fourth month of pregnancy, i.e., second trimester, continued throughout pregnancy (minimum 180 days during pregnancy) and to be continued for 180 days, postpartum each tablet containing 60 mg elemental Iron + 500 mcg Folic Acid, sugar-coated, RED COLOUR |

| Table 45.6D: Dose and Regime for deworming | |
|---|---|
| **Age Group** | **Dose and Regime** |
| School-going/Out-of-school adolescent girls, 10–19 years of age | Biannual dose of 400 mg albendazole (1 tablet) |
| Women of reproductive age (non-pregnant, non-lactating) 20–49 years | Biannual dose of 400 mg albendazole (1 tablet) |
| Pregnant women and lactating mothers (of 0–6 months child) | One dose of 400 mg albendazole (1 tablet), after the first trimester, preferably during the second trimester |

**Table 45.6E: Anaemia management protocol for Pregnant Women**

| |
|---|
| All pregnant women to be screened at each ANC visit |
| **If Hb 10–19 g/dl (mild anaemia) or Hb 7–9.9 g/dl (moderate anaemia)** |
| 2 tablets IFA (60 mg elemental Iron, 500 mcg Folic Acid) daily, orally |
| OR |
| IV Iron Sucrose or Ferric Carboxy Maltose (FCM) late in pregnancy or in case of non-compliance |
| **Follow up after 2 months:** |
| If normal Hb - continue IFA Prophylaxis |
| If Hb is not improved (< 1 g/dl rise in one month) – Investigate/refer |
| **If Hb 5.0–6.9 g/dl (severe anaemia)** |
| Hospitalisation, evaluation, blood transfusion |

and being screened for anaemia, to be screened for malaria also and vice versa. Fixed days for screening for anaemia at PMSMA sites and annually by RBSK teams in schools will provide screening for malaria under National Vector Borne Disease Control program (NVBDCP). Anaemia Mukt Bharat plays a key role for utilisation of long-lasting insecticides net (LLINs) by all target groups especially pregnant mothers and under five children by promoting IEC/BCC. Indoor Residual Spray (IRS) before and after monsoon season to be carried out with appropriate insecticides in school premises and residential areas.

Screening for hemoglobinopathies, integrated with ANC services during 1st trimester in endemic regions. Women with severe anaemia to be referred to higher centers, if found screening positive then their husbands should also be screened for the carrier status. If the couple is found positive then they should be referred to higher centers for prenatal diagnosis before 20 weeks of pregnancy.

To provide premarital and preconception screening and counselling for informing the community about the preventive options and management as per the national guidelines on prevention and control of hemoglobinopathies 2016.

Coordinated action with National program for the prevention and control of fluorosis (NPPCF), use of safe drinking water, focus on diet corrections (calcium, magnesium, vitamin C) by dietary diversity.

## Six Institutional Mechanism

1. Intra-ministerial coordination
2. National Anaemia Mukt Bharat Unit
3. National Centre of Excellence and Advanced Research on Anaemia Control
4. Convergence with other ministries
5. Strengthening supply chain and logistics
6. Anaemia Mukt Bharat dashboard and digital Portal-one-stop shop for anaemia

## ASHA incentive for Anaemia Mukt Bharat

ASHA will be provided a total of ₹ 150 per month per ASHA for covering at least 70% of the beneficiaries for IFA supplementation in two age groups: children 6–59 months and WRA.

Prophylaxis with iron should be withheld in case of acute illness (fever, diarrhoea, pneumonia, etc.) and in a known case of thalassemia major/history of repeated blood transfusion.

All women in the reproductive age group in the pre-conception period and up to the first trimester of the pregnancy are advised to have 400 mcg of Folic Acid tablets, daily, to reduce the incidence of neural tube defects in the fetus.

## Indradanush Scheme

- Launched by The Ministry of Health and Family Welfare (Govt. of India)
- On December 25, 2014.

The Mission Indradhanush, depicting seven colours of the rainbow, aims-to cover all those children by 2020 who are either unvaccinated, or are partially vaccinated against seven vaccine preventable diseases which include

- Diphtheria
- Whooping cough (Pertussis)
- Tetanus
- Polio
- Tuberculosis
- Measles
- Hepatitis B

### Intensified Mission Indradhanush (IMI)

- To reach each and every child under two years of age and all those pregnant women who have been left uncovered under routine immunization programme.
- Focus on improving immunization coverage in select districts and cities to ensure full immunization to more than 90% by december 2018.

### Menstrual hygiene scheme

- Pack of 6 sanitary napkins - 'Freedays'
- ₹ 6 per pack
- Available with ASHA worker

### Revised Menstrual Hygiene Scheme

- ₹ 6 for a pack of six sanitary napkins by ASHA through door to door sale
- Out of sale proceeds, the ASHA gets an incentive amount
  - ₹ 1 per pack
  - free pack of sanitary napkins per month
- Balance fund - recouped (returned) to the state health society account to be utilised for procurement in the following year.

## Dakshta

To strengthen the compentency of the providers of the labour room, including medical officers, staff nurses and ANMs (Auxiliary nurse midwife) to perform evidence-based practices as per the established labour room protocols and standards.

Clinical update cum skills standardization training: The initiative will undertake a short customized clinical update cum skills standardization training for the providers of the labour rooms. This will be a three-day activity which will be conducted by designated trainers at identified training sites.

All providers of labour rooms, irrespective of their training status in the 21 day in-service SBA trainings, will be eligible for these trainings.

### Weekly Iron Folic Acid Supplementation

- Launched in 2013
- School age children
- Community based
- Orientation of teachers and parents regarding health, nutrition, diet, etc.
- Health card and Hb estimation of all children
- Set of 8–10 slides on importance of anaemia prevention to be distributed to all schools
- Organize Painting, poster competition
- Incorporate a chapter on anaemia in 5th–8th standard textbooks
- Deworming, Organize iron day
- Repeat Hb after 6 months

Iron + Initiative

**Table 45.7: Dose and Mode of Iron and Folic Acid**

| Age group | Iron dose (elemental Fe) | FA dose | Mode |
|---|---|---|---|
| 6m (month) –5 years | 20 mg | 100 mcg | Biweekly, Syrup |
| 5–10 years | 45 mg | 400 mcg | Weekly, Tablet |

- Beneficiaries receive iron and folic acid supplementation irrespective of their Iron/Hb status

**Biannual deworming** with Albendazole > 1 year of age

## Adolescent friendly health clinics

- PHC (Primary Health Centre) - **weekly** by ANM (Auxilary nurse midwife)/MO (Medical officer)
- CHC (Community Health Centre)/DISTRICT – **daily**
- Nutrition
- Substance abuse
- Injury, violence, non-communicable diseases
- Mental health
- Reproductive and sexual health - ICTCs (Integrated Counselling and Testing Centres)
- Gender sensitization

## NATIONAL STRATEGIC PLAN FOR HIV/AIDS - 2017-2024

Launched on December 1, 2017

"Paving the way for an AIDS free INDIA"

PMTCT (Prevention of mother to child transmission of HIV) → EPTCT (Elimination of parent to child transmission of HIV)

**Table 45.8: National Strategy Plan for HIV (NACP 2017)**

| Objective | Outcome |
|---|---|
| Reduce new infection by 80% by 2024 | New infection reduced from > 100,000 to < 21,000 |
| Link 95% of estimated PLHIV (Patients living with HIV) to services by 2024 | 2.1 million PLHIV know their status |
| Ensure ART (Anti-Retroviral therapy) initiation and retention of 90% of PLHIV for sustained viral suppression by 2024 | 1.90 million PLHIV on ART are retained and have sustained viral suppression |
| Eliminate mother to child transmission of HIV and syphilis by 2020 | Attain dual elimination by 2020 |
| Eliminate HIV related stigma and discrimination by 2020 | HIV/AIDS considered as a chronic manageable disease |
| Facilitate sustainable NACP (National AIDS control program) service delivery by 2024 | 1. Integration of identified components of NACP with NHM (National Health Mission)<br>2. Domestic funding for NACP is 100% |

Elimination of Parent to Child Transmission of HIV and Syphilis

**Goal:**

- Eliminating new HIV infections in infants by 2020
- Improving maternal, new-born; further child survival and health in the context of HIV

**Targets:**

- Testing of 95% of pregnant women for HIV and Syphilis
- Putting 95% of estimated positive pregnant women on ART
- Achieving an MTCT rate of less than 5% by 2020

### Elimination Strategies for HIV and Syphilis

- Universal ANC Check-up
- Single prick for HIV and Syphilis
- Treat all who are Syphilis positive
- Active case detection for partners with testing and treatment for HIV and Syphilis
- Available supply of commodities and drugs

- Collaboration with other agencies
- New detection and point of care tests

**Package of services currently offered**

- The HIV testing and counselling (pre-test and post-test) in antenatal patients
- All HIV positive pregnant women, including those presenting in labour and breast-feeding, are to be initiated on lifelong triple ART, irrespective of CD4 count and WHO clinical stage.
- The recommended duration of NVP (nevirapines) for the infant is a minimum of 6 weeks but can be extended to 12 weeks, if the duration of ART during pregnancy is < 24 weeks.

## NATIONAL IODINE DEFICIENCY AND DISORDERS CONTROL PROGRAMME (NIDDCP) AND NATIONAL GUIDELINES FOR SCREENING OF HYPOTHYROIDISM DURING PREGNANCY

National Goitre Control Programme (NGCP) running since 1962 was renamed as "National Iodine Deficiency Disorders Control Programme (NIDDCP)" in August, 1992 keeping in view the wide spectrum of Iodine Deficiency Diorders (IDD) like mental and physical retardation, deaf mutism, cretinism, still births, abortions, etc. The programme is being implemented in all the State/UTs. It is a 100% centrally assisted programme. Its goal is to bring the prevalence of IDD to below 5% in country and to ensure 100% consumption of adequately iodated (15 ppm) at the household level through supply of iodated salt in place of common salt and by laboratory monitoring of iodated salt and urinary iodine excretion and through health education and publicity.

Ministry of Health and Family Welfare is the nodal Ministry for implementation of National Iodine Deficiency Disorders Control Programme (NIDDCP).

### National Guidelines for Screening and Treatment of Hypothyroidism during Pregnancy 2014

It was started to reduce maternal, foetal and neonatal complications associated with hypothyroidism during pregnancy and childbirth by screening pregnant women at risk for hypothyroidism and by early diagnosis and treatment of hypothyroidism during pregnancy.

**High Risk Factors for Screening of Hypothyroidism in First Antenatal Visit**

1. Resident of moderate to severe iodine insuficiency areas.
2. Obesity (pre-pregnancy/first trimester Body Mass Index [BMI] ≥ 30 kg/m$^2$)
3. History of prior thyroid dysfunction or prior thyroid surgery.
4. Symptoms of thyroid dysfunction or the presence of goiter.
5. History of thyroid dysfunction in first degree relative (parents/siblings/children)
6. History of diagnosed mental retardation in family/previous births
7. Know case of autoimmune diseases like Type I diabetes/Systemic Lupus
8. Erythematosus (SLE)/Rheumatid Arthritis (RA)/Addison's disease/Coeliac disease, etc.
9. History of recurrent miscarriages, pre-term delivery, intrauterine demise.
10. Pre-eclampsia/eclampsia, abruptio placentae
11. History of infertility (inability to conceive after one year of unprotected intercourse)
12. Use of amiodarone or lithium or recent administration of iodinated radiologic contrast

**Overt hypothyroidism** - serum TSH > 2.5–3 mIU/l with low FT4 levels or TSH > 10 mIU/l irrespective of FT4

**Subclinical hypothyroidism** - serum TSH between 2.5 and 10 mIU/l with normal FT4

Normal values:
Ist trimester - 0.1–2.5 mIU/l
2nd trimester - 0.2–3 mIU/l
IIrd trimester - 0.3–3 mIU/l

### Treatment

Levothyroxine - category A drug, to be taken orally, in the morning empty stomach and nothing should be taken for at least half an hour after intake of the medicine as per Flowchart 45.2.

If dose is missed on one day, she should take the same as soon as she remembers and should not eat anything for the next half hour, if she misses the tablet altogether, she should take double the dose on the next morning.

Deliver uncomplicated cases at PHC/CHC under supervision of Medical Officer.

Refer cases with associated complications at a higher centre for delivery under the supervision of an obstetrician.

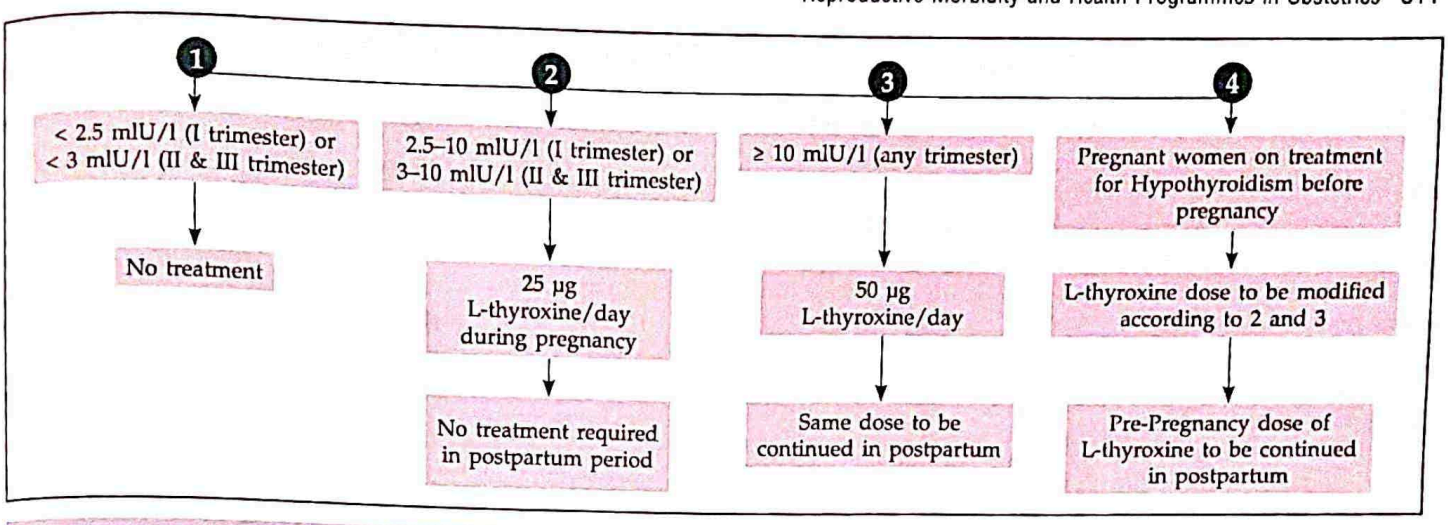
## MILLENIUM DEVELOPMENT GOALS (MDG)

In **September 2000**, representatives from 189 countries met in New York to adopt the United Nations Millennium Declaration in the area of development and poverty eradication. They are called Millennium Development Goals (MDG) and are as follows in relation to Obstetrics.

1. Reduce by two-thirds, between 1990 and 2015, the under-five mortality rate (Goal 4).
2. Reduce by three-quarters, between 1990 and 2015, the maternal mortality ratio (Goal 5).
3. Combat HIV/AIDS, malaria and other diseases
4. Skilled attendance at 90% births by 2015.
5. To reduce the infant mortality rate below 35 per 1000 live births by 2015.
6. Access to reproductive health services for all by 2015.
7. Other goals are gender equality, poverty reduction, education of girls and women.

Though there has been progress in health indicators but MDGs were not achieved.

**The Global Strategy for Women's, Children's and Adolescents' Health (2016–2030)** envisions a world in which every woman, child and adolescent in every setting realizes




**Flow Chart 45.2:** Treatment of Hypothyroidism in Pregnancy

their rights to physical and mental health and well-being, has social and economic opportunities, and is able to participate fully in shaping prosperous and sustainable societies.

### Sustainable Development Goals 3

By 2030

- To decrease global MMR < 70/100,000 live births
- To decrease (Neonatal mortality rate) NMR < 12/1,000 live births, under-5 mortality < 25/1,000 live births.
- Universal access to sexual and reproductive health care services - family planning, information, education, integration of reproductive health into national strategies and programmes.
- Universal health coverage - financial risk protection, access to safe, effective, quality and affordable essential medicines and vaccines for all.

## PREVENTION AND CONTROL OF HEMOGLOBINOPATHIES IN INDIA THALASSEMIAS, SICKLE CELL DISEASE AND OTHER VARIANT HAEMOGLOBINS

Hemoglobinopathies are autosomal recessive common genetic disorders worldwide and in India. Prevalence of β-Thalassemia carrier is 3–4% while HbS is 5–48% (tribal communities). HbE is common in the North Eastern states, HbD is present in about 2% of people in Punjab. As the disease affect children born to "healthy" carrier couples, this makes prevention and "carrier" detection an important public health intervention.

Prevention and Control of Hemoglobinopathies in India was launched in 2016 by the National Health Mission with the mission to improve case of all Thalassemia, Sickle Cell Disease and other haemoglobin variants like Hb D and Hb E patients for their better future and to lower the prevalence of hemoglobinopathies through screening and awareness strategies.

### Prevention and Control Strategies

1. Carrying out awareness, education and screening programmes in the community and schools.
2. Establishing laboratories for carrier screening for hemoglobinopathies and newborn screening for sickle cell disease at the district level.
3. Screening pregnant women and their husbands to prevent the birth of children affected with thalassemia major or sickle cell disease.
4. Screening of newborns, young children and adolescents for prevention and detection of hemoglobinopathies for early intervention and management.
5. Establishing Prenatal diagnostic centers in Medical Colleges in the States where required.
6. Management of patients with Thalassemia and Sickle Cell disease.
7. IEC strategies and modules
8. Reporting and monitoring
9. Training
10. Human resource management and budget allocation

**Universal antenatal screening and prenatal diagnosis of**

Under this provision all pregnant women attending public healthcare facilities at all the levels are to be screened for carrier status as per screening guidelines and are to be followed up with prenatal diagnosis where required as per Table 45.9A.

### Screen for Beta Thalassemia

Prenatal diagnosis-preventing the birth of an affected child of "at risk couple"

Hospitals with facility for obstetrical care, NICU and a genetic lab can serve as centres for prenatal diagnosis.

**Pre-requisites of prenatal sampling include:**

- Thalassemia carrier status of the couple under investigation
- Blood group of the mother to prevent Rh incompatibility, if present
- Pre test counseling, written informed consent of couple

### Foetal sampling methods available for prenatal diagnosis (USG guided)

1. **CVS:** between 10–12 weeks of gestation. (trans-abdominal route or trans-cervical)

2. **Amniocentesis:** after 16 weeks of gestation or if foetal position prevents CVS
3. **Foetal blood sampling - (cordocentesis):** at 18–20 weeks of gestation. Sampling is done by cardiac puncture or from the hepatic vein.

Foetal blood analysis by complete blood, Hb fraction analysis by cation-exchange, HPLC and DNA based tests identify the defect at the gene level and provide final confirmation of the defect

Choices available at an 'at risk' couple:

- Termination of the affected child
- Not to have children
- To adopt children
- To proceed to in-vitro fertilization (e.g. PGD)

## Diagnosis of Hemoglobinopathies at Birth (Table 45.9B)

Four good quality dried blood spot card should be taken and dispatched to the laboratory within 24 hours of collection.

**Technique used for analysis of newborn samples:**

Three techniques for Newborn dried blood spot sample as primary screen.

- High performance liquid chromatography (HPLC)
- Isoelectric focusing (IEF)
- Capillary Electrophoresis (CE)

**Table 45.9A: Prenatal Diagnosis**

| | |
|---|---|
| **Step 1** | Micro-planning for antenatal screening and referral for prenatal diagnosis. Screening of all pregnant women on first visit (Ist trimester) to CHC/PHC by tube tests (NESTROFT, DCIP test, Solubility test) along with routine Hb, Urine test for protein, Blood Group, HIV and Blood sugar. |
| **Step 2** | If Screening test positive, refer the patient along with husband by 104/108 service to District Hospital/DEIC for further testing |
| **Step 3** | The pregnant woman to be admitted to District Women Hospital under JSSK for conduction of CBC and Hemoglobin HPLC test of both wife and husband |
| **Step 4** | If wife and husband both are detected to be hemoglobinopathy carriers on Hb-HPLC testing, an Ultrasound test (USG) of the wife (the pregnant woman) to be done to determine the gestational age of the fetus and provision of counseling the couple |
| **Step 5** | One of the following steps to be followed depending on gestational age:<br>a. If gestational age 12 weeks or less, couple to be referred to nearest tertiary centre for prenatal diagnosis by CATS through coordination with the DH/DEIC<br>b. If gestational age > 12 weeks but < 20 weeks, consultation for possible PND by amniocentesis at the tertiary centre<br>c. If gestational age > 20weeks counseling for possible outcomes of pregnancy and follow up of pregnancy. If the baby born is an affected child determined by DNA based tests, then after family counselling, early intervention by registration in DH/DEIC for management and care is required. |

**Table 45.9B: Screening for Hemoglobinopathies**

| **1. Initial Screening** | | |
|---|---|---|
| **NESTROFT Test (Naked Eye Single Tube Red cell Osmotic Fragility Test)** | **DCIP test (Di-Chlorophenol-Indo Phenol)** | **Solubility Test** |
| For Beta thalassemia trait<br>Sensitivity of 90–100% specificity of 85.47%<br>PPV 66%, NPV 97–100% | For detection of hemoglobin E carriers, especially in eastern states like Assam and Bengal<br>Sensitivity 100%<br>Specificity 98.7%<br>PPV 98.6%, NPV 100% | For Hb S, it is rapid (5 min), reliable with minimal observer variaiton.<br>Sensitivity 100%<br>Specificity 91.66%<br>PPV 80%<br>NPV 100% |
| Complete blood count (CBC) (Sample positive for tube test in field are also subjected to CBC in lab) | Hb: 12–17 gm/dl<br>MCV: 80–100 fi<br>MCH: 27–32 pg | Severe anaemia with microcytic hypochromic red cell indices Hb < 9g/dl<br>MCV: 50–70 fi; MCH: 12–20 pg) |
| **2. Diagnostic test** | | |
| High performance liquid chromatograph:<br>HbA: 96–98%<br>HbF: < 2%<br>HbA2: 2.2–3.3∞ | HPLC pattern in β-thalassemia<br>HbA: 0–30%<br>HbF: 70–100%<br>HbA2: 2–5% | HPLC pattern in Sickle cell syndromes<br>HbA: 0–30%<br>HbS: > 50%<br>HbF: < 50% |
| **3. Confirmatory test** | | |
| DNA Based test<br>Reverse Dot Blot hybridization, ARMS, Gap PCR, DNA Sequencng for unknown mutations<br>(Specially to be used where HPLC is non contributory and for all prenatal diagnostic testing) | | |

## Key Points

1. Safe Motherhood Initiative (SMI) was started by WHO in 1987 to reduce maternal deaths and to promote maternal and child health.
2. Various strategies to reduce maternal mortality and to achieve safe motherhood are by improving the status of women in society, reducing fertility through use of family planning and MTP services, improving referring and referral units, easy access to obstetric services with improved standard of care in labour.
3. Maternal death is defined as death of a woman during pregnancy, child birth or within the 42 days of puerperium by causes related to or aggravated by pregnancy.
4. Maternal mortality ratio (MMR) is the number of maternal deaths per 100,000 live births and is high in developing countries like India (130 in 2014–16 it was 212 in 2009–10, 256 in 2006 [407 in 2000] as compared with 10 in UK and 27 in China).
5. Direct maternal deaths are due to causes related to or aggravated by pregnancy and are the most common causes (66%) with haemorrhage, sepsis, pre-eclampsia and eclampsia and prolonged and obstructed labour. Anaemia, hepatitis, heart disease and tetanus are important causes of indirect maternal deaths.
6. Various interventions to prevent maternal deaths are promotion of family planning services, dietary supplementation and prophylaxis for anaemia, antenatal booking and hospital deliveries, improvement of healthcare facilities and training of healthcare workers.
7. Reproductive morbidity refers to long term sequelae of complications of pregnancy and labour like genital prolapse, obstetric fistula, urinary incontinence, Sheehan's syndrome, pelvic inflammatory disease, etc.
8. There is massive burden of reproductive morbidity in India and other developing countries affecting her general health and negatively affecting the economy of the country. Proper care of women during antenatal period and childbirth can significantly reduce this morbidity.
9. Perinatal mortality rate refers to all foetal deaths (> 1000 g or > 28 weeks) and early neonatal deaths (first week of neonatal life) per 1000 total births. The perinatal mortality rate (PNMR) in India is 48.5 per 1000 live births, 26 in 2013.
10. The various causes of perinatal mortality are prematurity, low birth weight, birth asphyxia, birth trauma, infections, congenital malformations and respiratory distress syndrome.
11. Improving standard of living, social nutritional status of women, universal prenatal care, strengthening of referral system, hospital deliveries and improving maternal and neonatal care facilities can reduce perinatal mortality.
12. Maternal steroid administration before preterm labour reduces perinatal mortality **(level IA evidence)**.
13. Dietary supplementation in chronic malnourished women reduces perinatal mortality **(level IB evidence)**.
14. Reproductive and Child Health (RCH) Phase I Programme was started in 1997 to improve maternal and perinatal outcomes.
15. RCH Phase II was started in 2005 to reduce maternal and child morbidity and mortality with emphasis on rural health care. It includes essential obstetric care (institutional delivery and skilled attendance at delivery), emergency obstetric care (operationalizing first referral units, PHCs and CHCs for round-the-clock delivery service).
16. RCH Phase II also includes new initiatives like training of MBBS doctors, setting of blood storage centers, Janani Suraksha Yojna, safe abortion and Vandemataram scheme.
17. In Janani Suraksha Yojana, there is a cash incentive to all women in low performing states to deliver in government hospitals or centers (₹ 1400 in rural areas and ₹ 1000 in urban areas). In high performing states, the cash incentive is given to below poverty line and SC and ST pregnant women.
18. Government of India has given high priority to maternal and child health and various health programs have been started to improve their health.
19. National Nutritional Anaemia Prophylaxis Program was started by the Government of India in 1970 to provide 60 mg elemental iron and 500 µg folic acid per day for all pregnant women, lactating women, family planning acceptors and children between 1 and 12 years. It has now been increased to 100 mg elemental iron and 500 µg of folic acid per day.
20. Twelve-by-twelve Initiative aims to have Hb of 12 for all adolescents so that they reach adulthood and parenthood without anaemia.
21. Millennium Development Goals (MDGs) aim to reduce under five mortality by two-thirds by 2015 (MDG, 4), maternal mortality by three-quarters by 2015 (MDG, 5) and combat HIV/AIDS, malaria and other diseases. They were not achieved.
22. Sustainable development goal 3 aims to decrease MMR to < 70 and NMR to < 12.
23. Other important programs are JSSK, PMSMA, MAA, LaQshya, Indradhanush, National Stretegic Plan for HIV/AIDS.

CHAPTER 46

# Imaging in Obstetrics

## ULTRASOUND

Use of ultrasound has revolutionized the practice of obstetrics. It was first used in obstetrics in 1958 by Ian Donald from Glasgow, United Kingdom (UK). The introduction of high frequency transducers with real time ultrasonography supplemented by Doppler and three and four dimensional ultrasound has made accurate foetal and maternal assessment possible.

An ultrasound machine with transabdominal and transvaginal probes is shown in Fig. 46.1. Ultrasonography uses high frequency (2–10 MHz) sound waves to create images and converts the reflected waves into a visual image on the screen. These sound waves are emitted through the transducer placed on the maternal skin with its transmission being enhanced by means of a coupling gel. Higher frequency transducers produce better image resolution. The received waves are dependent on the degree of attenuation, absorption and reflection by the tissues. The received wave is converted into electric signals which are then converted into an image.



**Fig. 46.1:** Photograph of an ultrasound machine with transabdominal and transvaginal probes

Transducers can be sector, linear or curvilinear. In real time imaging, moving pictures are produced. Commonly used B-mode ultrasound uses grey scale. M-mode is a linear display of the events of the cardiac cycle in real time and is used to measure foetal heart rate and detects abnormalities in rate and rhythm. Three-dimensional (3D) ultrasound uses a third dimension (Figs. 46.2 A to 46.2 F) while four dimensional (4D) ultrasound is 3D in real time. Thus, in three dimensional (3D) ultrasound, apart from horizontal and vertical dimensions of fetus, antero-posterior dimensions are also visible like in 3D movies to better delineate the foetal anatomy especially in foetal congenital malformations. Doppler velocimetry is used to assess the blood flow through different vessels and is useful in assessing foetal well-being.

### Methods

Transabdominal scanning (TAS) is used in the second and third trimesters. Its probe has a frequency of 3.5 MHz. Usually in early pregnancy, a full bladder is essential to act as a window for proper visualization. The transvaginal scan (TVS) probe with a higher frequency of 5 to 7 MHz is commonly used in the first trimester as most structures can be seen about one week earlier than with abdominal ultrasound. The bladder should be empty for transvaginal ultrasound.

**Table 46.1: Timing of Appearance of Ultrasound Findings in Early Pregnancy on Transvaginal Ultrasound**

| | |
|---|---|
| Gestational sac | 4 ½ week |
| Yolk sac | 5 week |
| Foetal node | 5 ½ week |
| Foetal heart | 6 week |

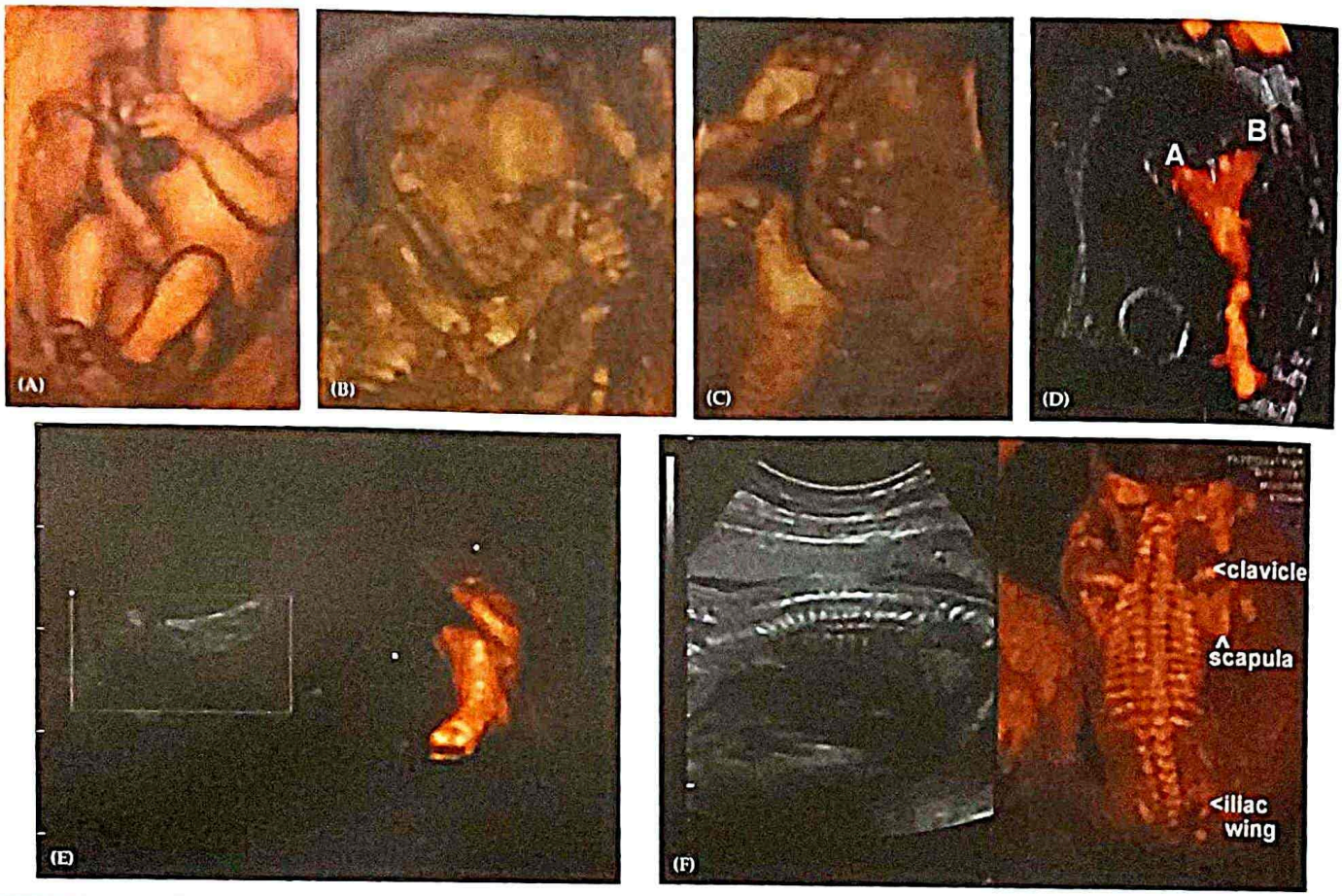



**Fig. 46.2:** (A) A 3D ultrasonic picture of a normal fetus; (B) A 3D ultrasonic picture of a normal fetus showing normal face, ear and fingers; (C) A 3D picture of a fetus with bilateral cleft lip; (D) A 3D power Doppler ultrasound of conjoined twins at 10 weeks gestation; (E) A 3D ultrasonic picture of a normal fetus with a normal foot; (F) A 3D ultrasonic picture of normal foetal spine and other bones
Kind courtesy: Dr Ashok Khurana, Delhi

A two-stage scanning can be done; an initial (standard) scan at booking (preferably at 10 to 14 weeks) and a targeted anomaly scan at around 18–20 weeks (see Chapter 14 for details). A first trimester scan may be needed for specific indications. A third trimester scan is performed to assess growth and well-being if indicated, as in suspected foetal growth restriction or high-risk pregnancies. However, if only a single scan is possible, it should be at 18–20 weeks.

### Safety

Diagnostic ultrasound has been shown to be completely safe for the fetus. But it is recommended that the usage should not be abused.

## FIRST TRIMESTER ULTRASONOGRAPHY

### Indications

1. To confirm foetal viability
2. To confirm the period of gestation (Table 46.1)
3. To establish the site of pregnancy—intrauterine (Figs. 8.5 and 8.6 in Chapter 8) or extrauterine (Fig. 13.16 in Chapter 13).
4. To confirm the number of fetuses in multiple pregnancy and to see chorionicity and amnionicity (**Figs. 33.3, 33.4** and **33.5** in Chapter 33).
5. To evaluate early pregnancy complications like threatened abortion, missed abortion, incomplete abortion and blighted ovum (see **Fig. 13.7** in Chapter 13 for details)
6. To diagnose hydatidiform mole (**Fig. 13.25** in Chapter 13).
7. To detect any adnexal mass, like ovarian mass.
8. To detect uterine anomalies.

With transabdominal scanning all the structures are visualized about one week later.

### Characteristics of normal intrauterine gestational sac are as follows (Figs. 8.5 and 8.6 in Chapter 8)

1. Eccentric location
2. Regular outline
3. Double decidual sac sign
4. Presence of a yolk sac
5. Good choriodecidual reaction

The double decidual sac sign is the normal appearance of the intrauterine gestational sac. It is seen as two concentric echogenic rings separated by a hypoechoic space. The rings are the decidua parietalis and decidua capsularis. This is very useful to differentiate an intrauterine pregnancy from ectopic pregnancy.

Normal sac grows around 1.1 mm per day (till 20–25 mm). Increase of less than 0.6 mm per day indicates abnormal development.

First trimester is the time when foetal maturity can be estimated with greatest accuracy by **gestational sac size between 5–10 weeks and by crown rump length (CRL) between 6–14 weeks. Overall the best parameter to determine foetal age is crown rump length in first trimester (11–13 + weeks).** A CRL of ≥ 7 mm with no cardiac activity seen on mean sac diameter of ≥ 25 mm with no yolk sac indicates missed abortion.

### 10–14 Weeks Scan

10–14 weeks scan is performed for accurate estimation of gestation, screening for Down's syndrome and other anomalies by measuring nuchal thickness (Fig. 14.1) and presence of other aneuploidy markers and to detect certain gross foetal anomalies like anencephaly (see Chapter 14 for details). The American College of Radiology has recommended its name to be **standard** or **basic ultrasound examination** instead of level 1 scan.

## SECOND TRIMESTER OR 18–20 WEEKS TARGETED SCAN OR EXAMINATIONS OR TARGETTED IMAGING FOR FOETAL ANOMALIES (TIFFA)

Second trimester targeted scan is done to rule out foetal anomalies (see Chapter 14 for details). It was called level 2 in older nomenclature. It can pick up various congenital malformations like hydrocephalus, spinabifida and meningocele or meningomyelocele (Fig. 46.3 A). Fetus with anencephaly on ultrasound shows **'Frog eye sign'** with prominent eyeballs or **Mickey mouse sign.** Fetus with spina bifida can have cranial signs like small biparietal diameter, frontal bone scalloping called **Lemon sign** (Fig. 46.3 B), elongation and downward displacement of cerebellum **(Banana sign)** (Fig. 46.3 B). These two signs are part of Chiari II malformation (or Arnald Chiari malformation) in which partial displacement of cerebellum and other posterior fossa structures herniate. Foetal congenital diaphragmatic hernia (Fig. 46.3 C), abdominal wall defects like exomphalos (Fig. 46.3 D) foetal renal

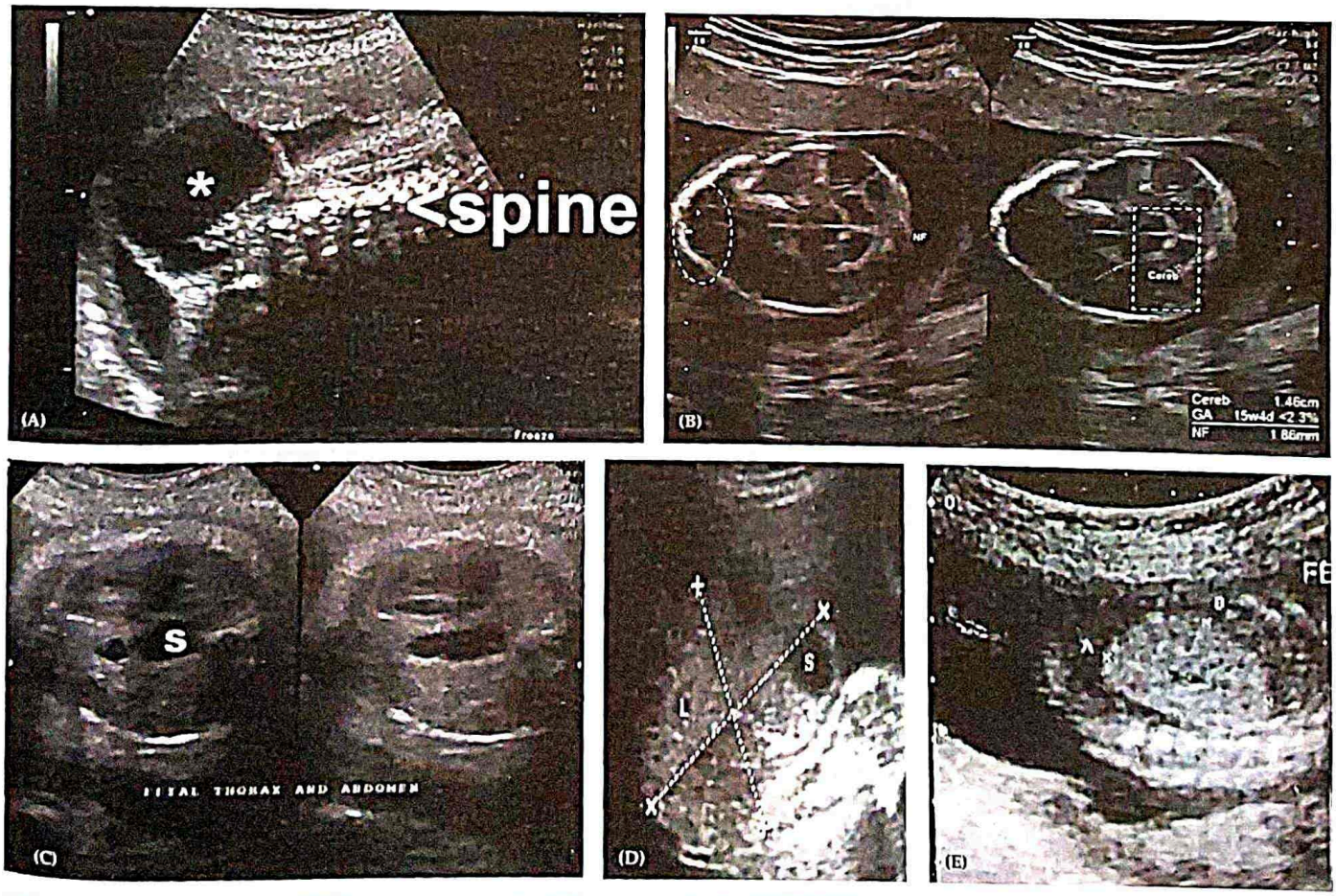



**Fig. 46.3: (A)** Transabdominal ultrasound at 20 weeks gestation showing foetal lumbar meningomyelocele (star *); **(B)** Ultrasound of a case of foetal spina bifida showing frontal scalloping (Lemon sign: encircled with a circle) and elongation and downward displacement of the cerebellum causing effacement of cisterna magna (Banana sign; encircled with a rectangle); **(C)** Transabdominal ultasound showing foetal congenital diaphragmatic hernia; **(D)** Ultrasound picture showing foetal exomphalos; **(E)** Ultrasound picture shows foetal polycystic kidney disease
Kind courtesy: Dr Ashok Khurana, Delhi for Figs. 46.3 A to C
Kind courtesy: Dr Prerna Gupta's thesis (AIIMS, New Delhi) for Fig. 46.3 D and E (Guides: Prof. S Kumar and Prof. Raju Sharma)

**Table 46.2: Indications of Ultrasound in Second or Third Trimester**

1. Estimation of gestational age and evaluation of foetal growth (see Chapter 14 for details)
2. Determination of foetal presentation and lie (see Chapter 16 and 23)
3. Multiple gestation (see **Figs. 33.3, 33.4** and **33.5** in Chapter 33 for details)
4. Discrepancy in gestational age and uterine height, foetal growth restriction and intrauterine foetal death (see Chapter 34 for details)
5. Suspected pelvic mass
6. Cervical incompetence (see **Fig. 13.9** and Chapter 13 for details)
7. Biophysical profile (see Chapter 15 for details)
8. Observation of intrapartum events
9. Amount of liquor and to diagnose polyhydramnios **(Fig. 24.10)** and oligohydramnios **(Fig. 24.11)** in Chapter 24.
10. Bleeding during pregnancy: Placental abruption and Placenta previa, (see **Fig. 31.2** and Chapter 31 for details)
11. Localization of placenta and its anomalies (see chapters 24 and 31 for details)
12. Diagnosis and follow-up of congenital anomalies (see chapters 14 and 21 for details) (**Figs. 21.3 A** and **21.5 A**)
13. PROM and foetal weight estimation (see Chapter 34 for details)
14. As an adjunct in prenatal diagnostic and therapeutic procedures like amniocentesis and foetal therapy (see Chapter 14 for details).
15. In Rh isoimmunization (see **Figs. 38.5** and **38.6** and Chapter 38 for details)

anomalies like foetal polycystic kidney disease (Fig. 46.3 E) can also be diagnosed by ultrasound.

### Indications of Ultrasonography in Second and Third Trimesters of pregnancy

The various indications are shown in Table 46.2.

## DOPPLER IN OBSTETRICS

The term 'Doppler' is based on the **Doppler effect wherein the returning frequency of waves is altered by the movement of a target which is blood in the blood vessels in the region of interest.**

At present pulsed Doppler is used in obstetrics as it is independent of angle of insonation and is particularly useful for assessment of areas of high blood flow velocity and of vascular or non-vascular areas. Its prolonged use should be minimized in pregnancy by using less of colour flow box and avoiding heating of tissue.

Commonly used indices are:

(*i*) S/D ratio = $\frac{\text{Systole (S)}}{\text{Diastole (D)}}$

(*ii*) Resistance Index (RI) = $\frac{\text{Systole} - \text{Diastole}}{\text{Systole Peak Velocity}} \left(\frac{S - D}{S}\right)$

(*iii*) Pulsatility Index (PI) = $\frac{\text{Systole} - \text{Diastole}}{\text{Mean}} \left(\frac{S - D}{M}\right)$

Most modern ultrasound machines have Doppler facilities and are programmed to calculate various indices themselves and are very user-friendly.

Commonly examined vessels are the uterine artery (Fig. 46.4 A and 46.4 B), umbilical artery (**Figs. 15.5, 15.6, 15.7** and **15.8** in Chapter 15), middle cerebral artery (**Fig. 15.10** in Chapter 15 and Fig. 46.4 C), ductus venosus (**Fig. 15.9** in Chapter 15) and other venous channels like umbilical vein, inferior vena cava and superior vena cava.

### Uterine Artery (Figs. 46.4 A and B)

Normal tracing of uterine artery is shown in Fig. 46.4 A. *The uterine artery PI of more than 1.45 at 18–22 weeks of gestation with diastolic notching is considered abnormal and is a predictor of pre-eclampsia and foetal growth restriction* (Fig. 46.4 B). The PI is more useful in foetal and placental vessels as they are elastic vessels. Typically the PI of umbilical artery declines from 2 in the second trimester to 1

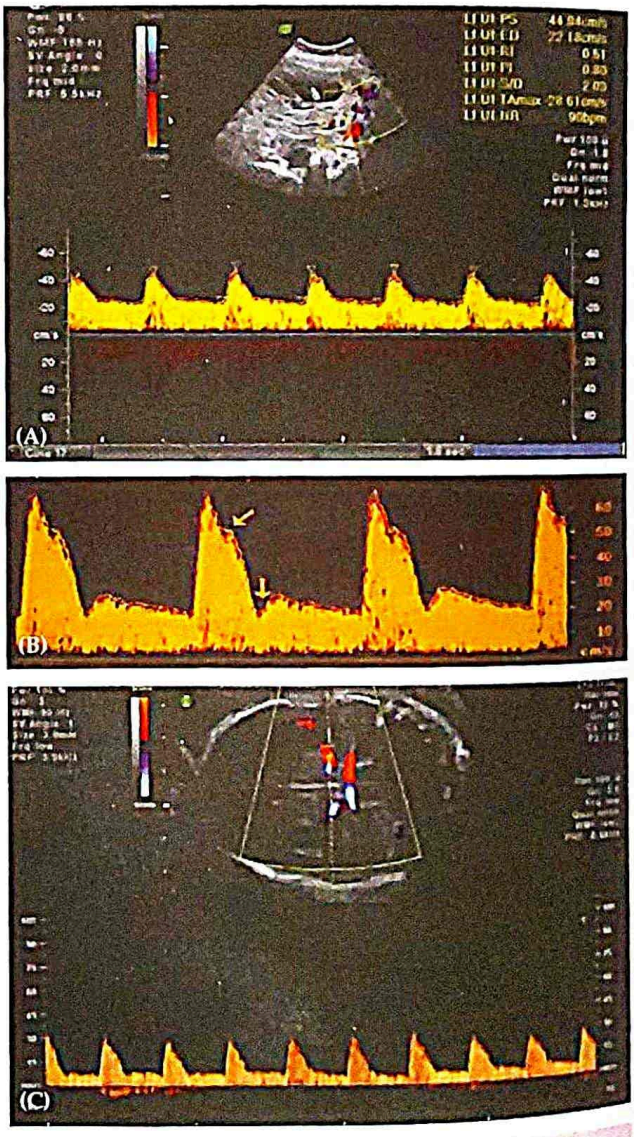



**Fig. 46.4: (A)** Normal tracing of uterine artery Doppler; **(B)** Doppler of uterine artery with diastolic notch (arrow) at 18 weeks gestation. The woman later developed pre-eclampsia; **(C)** Doppler of foetal middle cerebral artery peak systolic velocity (Normal). Kind courtesy: Dr Ashok Khurana, Delhi

near term. From 18–22 weeks onwards it should not show absent end-diastolic velocity. In middle cerebral artery, the redistribution occurs when there is foetal compromise and typically PI should be > 1.45 before term, falling to 1 by term. Ductus venosus peak velocity is normally 50 cm/second with small 'A' waves, 'A' wave < 5 cm/second is abnormal.

**Umbilical Artery** (Figs. 15.5, 15.6 and 15.7)
Resistance in the umbilical artery decreases with gestation. In foetal growth restriction, the flow decreases to the foetal viscera, with decreased flow in the umbilical artery and an increased resistance in the umbilical circulation. A corresponding decrease in the end diastolic velocity is noted. **An end diastolic flow of near zero is indicative of grave foetal distress, a reversal of the diastolic flow is almost a terminal event (see Chapter 15 and Figs. 15.7 and 15.8 for details).**

**Foetal Middle Cerebral Artery (MCA)** (Fig. 46.4 C)
The fetus responds to asphyxia by increasing flow to the cerebral hemispheres, i.e. a decrease in resistance. A decreased resistance in the MCA is a sign of a compromised fetus and increased flow is a compensatory sign, the so called **'brain sparing effect'**. However, a continued insult to the fetus causes a decrease in flow (i.e. a return to the normal) as a terminal event. Reversal of the 'brain sparing' effect in a fetus is indicative of grave foetal distress. Middle cerebral artery Doppler is particularly useful for Rhesus isoimmunization pregnancies (see **Fig. 15.10** in Chapter 15 and Fig. 46.4 C and Chapter 38 for details).

Conventional ultrasound fails to identify the onset of hypoxia in the fetus. Doppler ultrasound studies of the foetal circulation in foetal growth restriction and other hypoxic states are useful in such cases for prognosis and to make a decision for termination of pregnancy. In such cases, absent end diastotic flow occurs in umbilical arteries which is an indication of termination of pregnancy as there is risk of intrauterine death. Middle cerebral artery and ductus venosus Doppler is also useful in these cases to determine time of termination of pregnancy.

**Indications of Doppler Studies**
Indications of Doppler studies are shown in Table 46.3.

## Current status of doppler

Cochrane database reveals that there is 30% improvement in perinatal mortality for normally formed fetus and below 32 weeks of gestation if umbilical arterial assessment is performed in high-risk pregnancies and due course of action is subsequently taken.

Judicious use of Doppler in high-risk cases can help in making obstetric decisions. However, its role in low risk cases in still controversial and is not recommended in current practice as it does not improve outcome **(level IA evidence)**.

# X-RAYS IN OBSTETRIC PRACTICE

X-ray abdomen during pregnancy was popular before ultrasound era for seeing multiple pregnancy **(Fig. 33.2)**, malpositions and malpresentations **(Figs. 23.21, 23.26, 23.34** in Chapter 23) and gross congenital malformations like anencephaly **(Fig. 21.5 B** in Chapter 21) and for pelvimetry in contracted pelvis **(Fig. 22.13** in Chapter 22). It can still be used in rural hospitals where ultrasound is not available but X-ray machine is available. However, radiological investigations may be required to diagnose maternal conditions co-existing during pregnancy which must be quickly diagnosed and treated as the delay may be harmful for mothers. Thus, a woman having pulmonary tuberculosis can undergo X-ray chest with abdominal shielding to protect the fetus. MRI should be preferred, wherever possible.

To avoid inadvertent exposure to X-rays in early pregnancy, it is important to avoid all radiological investigations in non-pregnant women during their luteal phase of menstrual cycles due to the possibility of pregnancy. Thus, hysterosalpingography, intravenous pyelography and barium meal should be done in the first half of menstrual cycle. Radiation should be avoided in the first trimester of pregnancy.

**Table 46.3: Indications of Doppler Studies in Obstetrics**

| S. No. | Indications | Vessels studied | Findings |
|---|---|---|---|
| 1. | Foetal growth restriction | This is the most valid indication. Umbilical arteries are usually studied, Ductus venosus and middle cerebral arteries may be studied. | Absent or reversal of end diastolic flow (see **Figs. 15.5** to **15.10** in Chapter 15) |
| 2. | Rhesus isoimmunized pregnancies | Middle cerebral arteries | Increased flow initially (see **Fig. 15.10** in Chapter 15, Fig. 46.4 C and Chapter 38 for details) |
| 3. | Multiple pregnancies | Umbilical arteries | Doppler can also detect twin-to-twin transfusion syndrome (see Chapter 33) |
| 4. | Ectopic pregnancy | Uterine arteries | Decreased flow to endometrium and more flow to adenexal mass. |
| 5. | Gestational trophoblastic disease | Uterine arteries | Persistent more blood flow to endometrium |
| 6. | Recurrent postpartum haemorrhage | Uterine arteries | Abnormal arterio-venous malformations |

**Radiation dose.** The SI unit of radiation dose is the gray (Gy) with one Gy representing the absorption of I joule (J) of energy by 1 kg of the material. Rad was used in the past and represented the absorption of 100 ergs of energy by 1 g of material.

| | |
|---|---|
| 1 Gy | = 100 rads |
| 100 m rad (millirads) | = 1 m Gy (milli Gray) |
| 1 rad | = 1000 millirads |

## Effects of radiation

Exposure to significant amounts of radiation during pregnancy can cause the following foetal effects.

I. **Stochastic effects:** They do not have threshold dose. Severity is not dose related. It includes radiation carcinogenesis and hereditary effects.

II. **Deterministic effects:** A threshold dose below which no effect is seen. Worsening of the effect as dose increases. All early effects and most normal tissue late effects are deterministic.

1. **Teratogenesis.** Certain foetal malformations like microcephaly can be caused by high dose radiation (> 150 rads) (see Chapter 14).
2. **Miscarriage**
3. **Mental retardation and decrease in intelligence quotient** (IQ) in the child later.
4. **Carcinogenesis.** There can be higher risk of childhood malignancies, especially leukemias and cancers in adult life.
5. **Mutagenesis.** Alteration of germ line genes which can affect future generations. An exposure of 50–100 rads can cause it.

Radioactive substances can localize to certain organs causing more damage in them like Iodine$^{131}$ in foetal thyroid, iron$^{59}$ in the foetal liver.

A foetal dose of 1 Gy (100 rads) can kill 50% embryos while 5 Gy (500 rads) can kill 100% embryos or fetuses before 18 weeks gestation. Gestational age is important and an exposure of > 0.1 gray (Gy) or 10 rads usually results in death of the embryo before 2 weeks.

Estimated foetal radiation in various commonly performed radiological investigations is shown in Table 46.4.

Radiographs may be still useful in assessing pelvic capacity in cases of suspected cephalopelvic disproportion or before deciding on a vaginal delivery in a case of previous cesarean delivery. They may also be useful in diagnosis of foetal malpresentation in smaller centers where ultrasound is not available.

Guidelines of American College of Radiology and American College of Obstetricians and Gynecologists (ACOG) are as follows:

1. No diagnostic procedure results in a radiation dose that threatens the well-being of the developing embryo and fetus.
2. Foetal risk is almost negligible with exposure to up to 5 rads (0.05 gray) with risk of malformations increasing significantly above doses of more than 15 rads.

**Table 46.4: Estimated Dose of Foetal Radiation in Various Radiological Investigations**

| I. Investigation | Foetal dose in mrads (millirads) |
|---|---|
| 1. Chest radiography | 100 |
| 2. X-ray pelvimetry (2 films) | 1000 |
| 3. X-ray lumbar spine | 400 |
| 4. Pelvic radiography | 200 |
| 5. Hip and femur radiography | 300 |
| 6. Abdominal X-ray | 250 |
| 7. Intravenous pyelography | 480 |
| 8. Retrograde pyelography | 600 |
| 9. Barium meal | 300 |
| 10. Barium enema | 1000 |
| **II. Computerized axial tomography** | |
| 1. CT scan chest | 16 |
| 2. CT head | 0 |
| 3. CT abdomen | 3000 |

3. Women should be counselled that X-ray exposure from a single diagnostic procedure does not result in harmful foetal effect and that exposure to less than 5 rads is not associated with an increase in foetal anomalies or pregnancy loss.

# COMPUTED (COMPUTERIZED) TOMOGRAPHY IN OBSTETRICS

Computed tomography (CT) is an imaging technology that enables crosssectional imaging in which there is display of sections of the body using X-ray absorption measurements. Although CT pelvimetry was once popular, especially in breech presentation, it is not used these days due to radiation hazards. MRI is preferred in pregnancy. CT scan does not have much role in obstetrics but may be used for any coincidental maternal conditions like brain tumors and chest conditions in which case an abdominal shield should be used to protect the fetus.

## Magnetic Resonance Imaging (MRI)

MRI provides more specific information with excellent tissue contrast and multiplanar views with no ionizing radiation. It is safe in pregnancy. MRI is not recommended in the first trimester and use of contrast media is contraindicated in pregnancy.

### Indications of MRI in Obstetrics

1. Diagnosis of foetal congenital abnormalities. Ultrasound remains the modality of choice for detection of foetal

congenital malformations. MRI is particularly useful to better diagnose the cleft lip and palate defects in the fetus. It also diagnoses foetal central nervous system anomalies like foetal hydrocephalus with spina bifida (Fig. 46.5 A) and foetal congenital diaphragmatic hernia (Fig. 46.5 B).

2. MRI is helpful in localization and diagnosis of placenta previa (**Fig. 31.3** in Chapter 31). It also diagnoses placenta accreta, increta and percreta.
3. It helps to evaluate the vascularity of a polyp, detect the viable component of a gestational trophoblastic tumor and diagnose uterine arteriovenous malformations.
4. MRI pelvimetry can be beneficial in breech presentation.
5. In cases of first convulsive fit in pregnancy, an MRI of the brain is indicated to rule out any hemorrhages or other causes of fits.
6. Diagnosis of pelvic masses in pregnancy, including red degeneration of a uterine leomyoma with pregnancy.
7. For diagnosis of rare forms of ectopic pregnancy like ovarian and abdominal ectopic pregnancy.
8. For diagnosis of acute maternal conditions in pregnancy like acute fatty liver of pregnancy and pulmonary embolism.
9. Diagnosis of postpartum complications like abscess, haematoma and ovarian vein thrombosis.

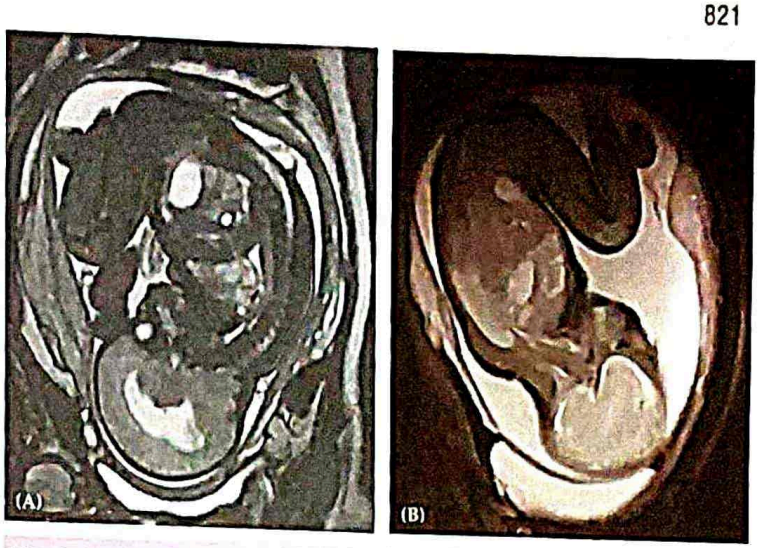



**Fig. 46.5: (A)** MRI during pregnancy showing hydrocephalus with spina bifida; **(B)** Foetal MRI showing foetal congenital diaphragmatic hernia. Kind courtesy: Dr Prerna Gupta's thesis (AIIMS, New Delhi); (Guides: Prof. S. Kumar and Prof. Raju Sharma)

## POSITRON EMISSION TOMOGRAPHY (PET) SCAN

There have not been many studies on the role of positron emission tomography during pregnancy and at present it is not used in pregnancy. However, in later pregnancy PET can be used for serious maternal conditions, like maternal cancer, to see the spread of the disease in the body.

## Key Points

1. Ultrasound has revolutionized the practice of obstetrics. It uses high frequency sound waves to create images. The transducer can be sector or linear while ultrasound can be B mode or M mode. Three dimensional ultrasound is also available these days for better delineation of foetal anatomy.
2. Ultrasound can be transabdominal or transvaginal with the latter being better in picking up early pregnancy and its complications, placental localization and cervical status. Transabdominal ultrasound is usually used for second and third trimesters scanning.
3. Basic or standard ultrasound examination is performed between 10–14 weeks for basic foetal examination. This scan can be used for screening for Down's syndrome by measurement of nuchal fold thickness (NT) (cut off > 3 mm), absent or hypoplastic nasal bone and absent flow in ductus venous.
4. Various soft markers for chromosomal disease which are indicators of karyotyping are nuchal edema, renal pyelectasis, hyperechogenic bowel, choroid plexus cysts, shortened long bones, echogenic intracardiac foci, mild ventriculomegaly, clenched fists and rocket bottom feet.
5. Apart from increased nuchal fold thickness, there is no strong evidence at present that other soft markers are helpful in identifying Down's syndrome **(level IA evidence)**.
6. American College of Radiology has recommended targeted or detailed foetal anomaly scan at 18–20 weeks of gestation which is the most effective method to identify a wide range of foetal abnormalities **(level IB evidence)**.
7. Screening the whole population rather than the selective screening is the only reliable method to detect foetal anomalies and is recommended but may not be possible in countries like India **(level IA evidence)**.
8. Screening for foetal anomalies reduces the perinatal mortality rates through identification and termination of affected pregnancies **(level IA evidence)**.
9. Ultrasound is useful for foetal biometry, foetal anomalies and diagnosis of foetal growth restriction, multiple pregnancies, presentation and lie, cervical incompetence, amount of liquor, placental localization, biophysical profile and as an adjunct to prenatal diagnostic procedures and foetal therapy.
10. Routine scanning for screening for foetal growth restriction in low-risk population does not increase pick up of SGA and does not lower perinatal mortality **(level IA evidence)**.

CHAPTER 47

# Obstetric Instruments, Specimens, Drugs, Spots and Laboratory Tests

## (i) OBSTETRIC INSTRUMENTS

In obstetrics, a number of instruments are used from gynecology and general surgery, apart from some which are used only in obstetrics.

The student should know and discuss the following features of each instrument.

1. Identification
2. Material of which the instrument is made and method of sterilization of the instrument.
3. Parts of the instrument with their special functions
4. Indications for use
5. Contraindications
6. Technique of use, including any anesthesia
7. Advantages and disadvantages
8. Dangers and complications

As a general principle, instruments made of stainless steel are sterilized by autoclaving or boiling except the sharp instruments (e.g. scissors) which are sterilized by chemical methods. Instruments made of plastic or rubber are usually for single use only or are sterilized by boiling or chemical methods. Instruments with fiber optic glass (e.g. laparoscope) are sterilized by chemical methods. In the discussion below, the instruments are made of stainless steel and are sterilized by autoclaving or boiling unless mentioned otherwise.

### SIM'S SPECULUM (Fig. 47.1)

Sim's speculum was invented by J Marion Sims of USA for examining patients of vesicovaginal fistula (VVF). This is the most commonly used speculum in obstetrics. It is called a duck-bill speculum due to its peculiar shape and is either double ended (bivalve) (Fig. 47.1) or single ended. A bivalve speculum has different sized blades for different sized vagina. (smaller blade for nulliparous women and the bigger blade for multiparous women). It has a handle in the center and the rounded and atraumatic blades at right angles to the handle. A trough runs along the entire length of the instrument so that blood or secretions collecting in the concave blade drain off along the trough in the handle. It is essentially used for speculum examination of all obstetric and gynecological patients.



**Fig. 47.1:** Sim's bivalve speculum

**Indications**

1. Examination for obstetric trauma such as cervical tears, vaginal tears after delivery.
2. Cleaning the vagina after childbirth.
3. Insertion or removal of an IUCD.
4. Dilatation and curettage and suction evacuation
5. Repair of obstetric trauma to the cervix and vagina.
6. Packing uterine cavity and vagina for uncontrolled atonic postpartum haemorrhage.
7. Collection of urine for culture in case of vesicovaginal fistula.

**Technique of Use**

The blade is lubricated with an antiseptic solution or sterile jelly. The labia minora are held apart by thumb and index fingers of the left hand and the blade is inserted in between, its transverse axis in the long axis of the labia. Once in the vagina, it is rotated through 90° so that the blade retracts the posterior wall of the vagina. Often

Sims' anterior vaginal wall retractor (Fig. 47.3) has to be used along with this instrument for adequate exposure of the cervix. The posterior vaginal wall is examined as the speculum is withdrawn. The instrument can be used in lithotomy position, dorsal position with legs drawn up or Sims' left lateral position.

**Sims's Triad:** Sim's speculum for examination and surgical treatment of vesicovaginal fistulae (VVF), Sim's left lateral position for examination of VVF, Sim's silver wire for repair of fistula.

### Advantages over Cusco's Speculum in Obstetrics

1. It is safe to use in conditions like placenta previa as it is less traumatic.
2. Vaginal walls can be examined.

### Disadvantages over Cusco's speculum in obstetrics

1. An assistant is required to hold it.
2. It cannot be used in the middle of the bed as the patient has to come at the edge of the table. Hence, it cannot be used on unconscious or very sick patients.
3. Anterior vaginal wall elevator is usually required for satisfactory examination.

(See textbook of gynecology for gynecological uses of Sims' speculum).

## CUSCO'S SPECULUM (Fig. 47.2)

Cusco's speculum has two blades connected by a hinge so that they can open and close around a transverse axis. The blades are concave inside with rounded ends and have handles at right angles with a nut and screw arrangement to fix the distance between the two blades. It is available in three different sizes for different sizes of vagina. The upper blade has a notch at its outer end through which a volsellum may be passed inside without obstructing the field of vision.

### Indications

1. Inspection of the vagina and the cervix as in Sim's speculum.
2. Procedures on the cervix and uterine cavity as with Sim's speculum especially insertion or removal of an IUCD.

### Procedure

The lubricated closed blades are passed between labia minora, transverse axis of the blades in the long axis of the labia. Once inside the vagina, the blades are rotated through 90° and are opened till the cervix is exposed satisfactorily. The blades are fixed in this position by the screw.



**Fig. 47.2:** Cusco's bivalve speculum

### Advantages

1. No assistant is required.
2. Anterior vaginal wall elevation is not required.
3. It can be used even in the middle of the bed or on unconscious patients.

### Disadvantages

Cusco's speculum can incite haemorrhage in placenta previa as its upper blades are put in the vagina blindly which can inadvertently separate the placenta. Hence, it is avoided in speculum examination for antepartum haemorrhage (APH) patients.

## SIM'S ANTERIOR VAGINAL WALL RETRACTOR OR ELEVATOR (Fig. 47.3)

Sim's anterior vaginal wall retractor or elevator is a long instrument with spoon-shaped ends which have transverse serrations on either surface. The loops make an angle of 15° with the shaft and are angled in opposite directions *(Do not confuse it with uterine curette in examination)*. It is a much bigger instrument with bigger and atraumatic serrations.

### Indications

1. Retraction of the anterior vaginal wall to expose the cervix in conjunction with Sims' speculum. The transverse serrations on its looped end provide friction against rugous vagina aiding in efficient retraction.



**Fig. 47.3:** Anterior vaginal wall retractor

## SPONGE HOLDING FORCEPS (Fig. 47.4)

Sponge holding forceps is 22.5 cm long with ring-shaped ends which have transverse serrations on the inner surface for better grip. A ratchet catch on the handles locks the blades on closure and prevents the forceps from slipping.

### Indications in Obstetrics

1. Cleaning of the abdomen and vulva with antiseptic solution for delivery and cesarean section.
2. Insertion of a Foley's catheter extra-amniotically for second trimester pregnancy termination using ethacridine lactate.
3. To hold the cervical lips during cervical cerclage and other operations during pregnancy.
4. To hold the membranes after delivery of the placenta in vaginal and cesarean delivery.

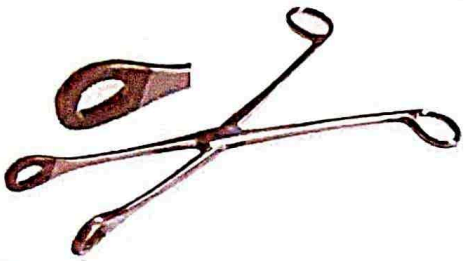


Fig. 47.4: Sponge holding forceps

5. Diagnosis and repair of a cervical tear after difficult deliveries using 2–3 sponge holding forceps (see Chapter 26 and **Fig. 26.2** for details).
6. To hold the edges of the uterine wound during cesarean section instead of Green Armytage forceps.
7. As a clamp for umbilical cord.

**Caution**

Sponge holding forceps should not be used for evacuation as it is more traumatic and loses the sensitivity after locking and can pull uterine wall or intestines if there is perforation due to loss of sensitivity. Ovum forceps being more sensitive is used for evacuation.

## MULTIPLE TOOTHED VULSELLUM (OR VOLSELLUM) (Fig. 47.5)

Multiple toothed vulsellum is of two types: a curved vulsellum called Tealey's vulsellum and straight vulsellum called Jacob's vulsellum.

**Uses**

1. Vulsellum is rarely used in obstetrics to catch anterior lip of cervix for dilatation and evacuation of uterus. Traumatic and pregnant cervix being soft, volsellum is often replaced by sponge holding forceps or Allis forceps in obstetrics.
2. In contraception, vulsellum can be used for IUCD insertion and removal.

**Indications of Holding Posterior Lip of Cervix**

1. For culdocentesis (needling) in suspected hemoperitoneum as in ruptured ectopic pregnancy.
2. For vaginal tubal ligation
3. Anterior lip of cervix is lacerated/deficient.
4. For culdoscopy
5. For colpotomy in case of pelvic abscess.

## TENACULUM (JARCHO'S) (Fig. 47.5 C)

Tenaculum is straight or curved, 20 cm long and similar in design to a volsellum except that there is a single tooth at the end of each blade. The grip of the instrument is more secure than that of a volsellum because its bite is deeper. It is especially suitable for a small cervix, e.g. nulliparous cervix because two teeth can grip a much smaller area than the five teeth of a volsellum.

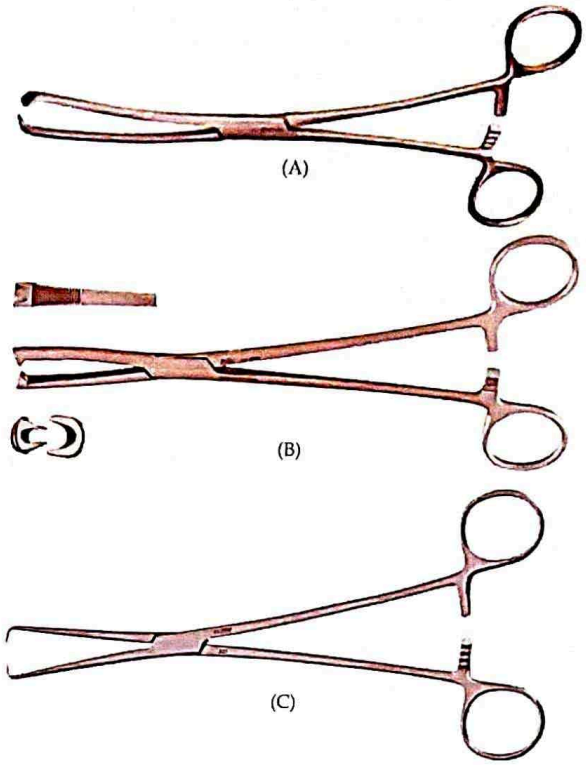



**Fig. 47.5:** (A) Curved (Tealey's) vulsellum; (B) Straight (Jacob's) vulsellum; (C) Tenaculum

**Caution**

As the teeth of a tenaculum take a deeper bite into the cervix, the risk of cervical tissue getting torn by the instrument is greater than that with a volsellum.

## TOWEL CLIPS (Fig. 47.6)

Towel clips are made of steel and are in two shapes like an artery (Backhaus' towel clip) and like a clip (Doyens' towel clip). They are used to fix the drapes over the operative area on abdomen (suprapubic area for cesarean delivery) and on the perineum for repair of episiotomy and perineal tears. They are also used to separate the operative area from anesthetists during cesarean delivery. They are also used for fixing the diathermy cables, suction tubes, laparoscopic camera cables and fiber-optic light cable to the draping sheets avoiding their piercing with them.

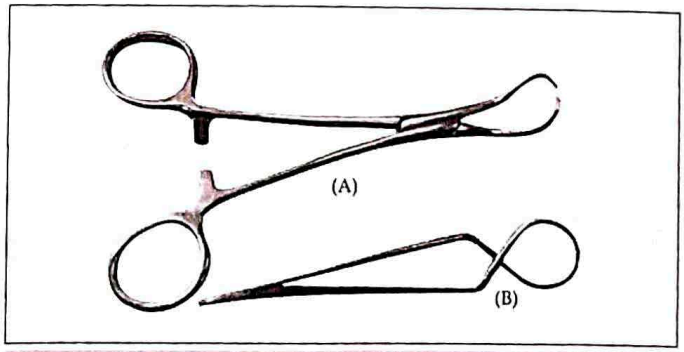



**Fig. 47.6:** (A) Backhaus' towel clip; (B) Doyens' towel clip

## ALLIS TISSUE FORCEPS (Fig. 47.7)

Allis tissue forceps is available in two sizes 12 cm and 17 cm in length with inward curved blades having 4–6 fine teeth. There is a ratchet lock on the handles.

**Uses**

1. To hold cervical anterior lip in medical termination of pregnancy like suction evacuation.
2. To hold the edges and apex of vaginal mucosa in episiotomy repair.
3. To hold tough structures like rectus sheath and aponeurosis in cesarean section, laparotomy and cesarean hysterectomy.
4. To hold levator ani and external sphincter muscle ends on either side during repair of third and fourth degree perineal tears.
5. During first layer closure of uterine incision in cesarean delivery, four Allis forceps are used (one each at two angles and upper and lower flaps) instead of Green-Armytage hemostatic forceps. It gives a better grip and is frequently used for this indication.

**Caution**

Due to sharp teeth of Allis tissue forceps, it is a traumatic instrument and should not be applied to the skin.

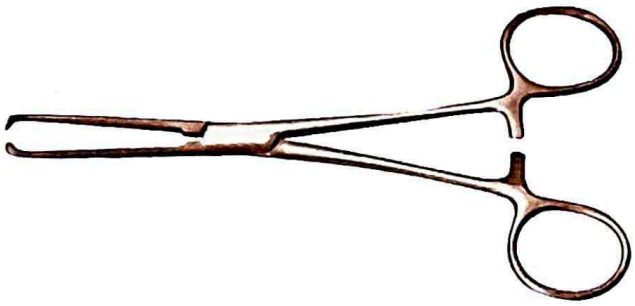


**Fig. 47.7:** Allis tissue forceps

## ARTERY FORCEPS (Figs. 47.8 A and B)

There are different varieties of artery forceps. They can be either straight (Fig. 47.8 A) or curved (Fig. 47.8 B).

Artery forceps are used to catch bleeders in episiotomy, perineal tears and cesarean delivery. They are also used to catch and pull the tip of needle by assistant during suturing. They are also used to hold the thread during stitching.

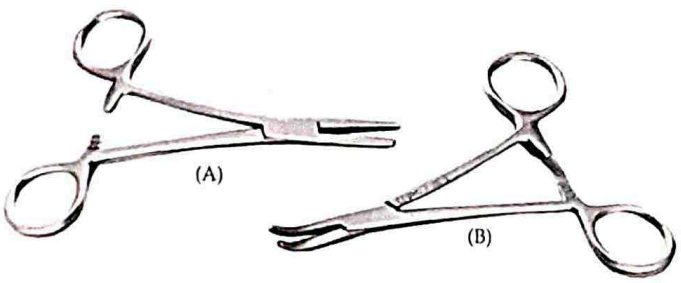



**Fig. 47.8: (A)** Straight artery forceps; **(B)** Curved artery forceps

## BARD PARKER HANDLE WITH BLADES (Figs. 47.8 C, D, E, F and G)

It is scalpel knife handle on which various blades can be fitted as per the need and operation. Commonly used blades are 11, 12, 15 and 22 (Cesarean and hysterectomy)

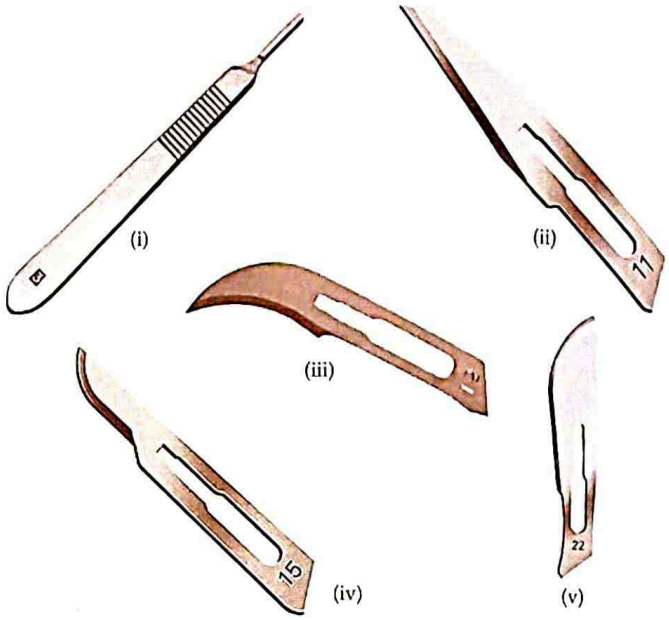



**Fig. 47.8:** (c) (i) Bard Parker Handle; (ii) Size 11 Blade; (iii) Size 12 Blade; (iv) Size 15 Blade; (v) Size 22 Blade

## KOCHER'S HEMOSTATIC FORCEPS (Fig. 47.9)

Kocher's hemostatic forceps are long stout forceps with teeth at ends which fit into each other.

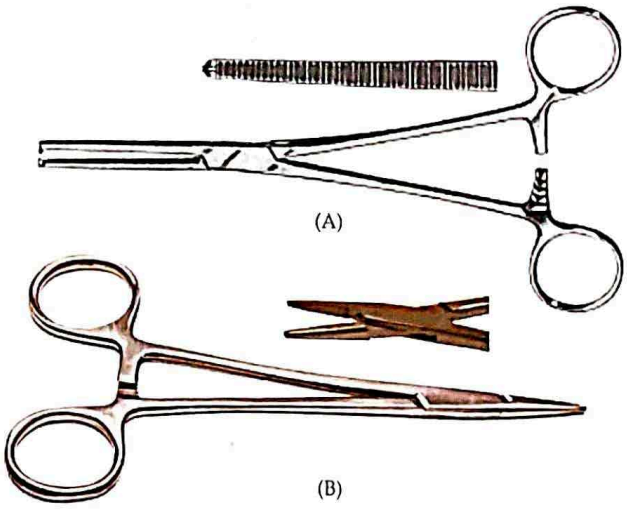



**Fig. 47.9:** (A) Kocher's hemostatic forceps; (B) Needle holder

**Uses**

1. For low amniotomy (artificial rupture of membranes) for induction and augmentation of labour.
2. Clamping the umbilical cord as it has a firm grip and occludes the umbilical vessels efficiently.
3. Rarely it can be used as a pedicle clamp.

The students may be asked about umbilical cord and its

abnormalities and indications, methods and complications of induction of labour.

## NEEDLE HOLDER (Fig 47.9 B)

Needle holder is a long instrument in the shape of a long artery forceps. However, its tip is much smaller as compared with an artery forceps and may have a vertical groove for better holding of needle and to avoid its wobbling during suturing.

**Uses**

1. Needle holder is used to hold surgical needles while suturing episiotomy and perineal tears.
2. It is also used to hold the surgical needle while performing cesarean delivery and cesarean hysterectomy operations.

## SIMPLE RUBBER CATHETER (Fig. 47.10)

Simple rubber catheter is made of rubber which should be used only once but can be sterilized by autoclaving or boiling or by chemical methods. It is used to drain urine temporarily. It is removed once bladder is emptied for the following indications.

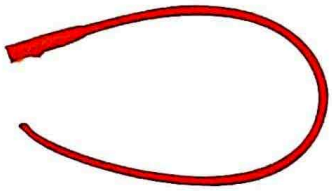


**Fig. 47.10:** Simple rubber catheter

1. In antenatal period for urinary retention like in retroverted gravid uterus, impacted ovarian cyst in pouch of Douglas or cervical fibrid causing urinary retention.
2. **Intrapartum uses.** It can be used for urinary retention in labour when head compresses urethra making voiding of urine difficult like in second stage. It is also used before, during and after instrumental deliveries and destructive operations to evacuate bladder.
3. *Puerperium.* For catheterization of bladder and for output charting in cases of severe postpartum haemorrhage, retained placenta and other serious conditions.
4. *Miscellaneous uses:*
   - (*i*) For tourniquet effect (e.g. myomectomy surgery)
   - (*ii*) To give oxygen as a substitute to nasal catheter.
   - (*iii*) To remove mucus from newborn's nostrils
   - (*iv*) Diagnosis of tracheoesophageal fistula in newborn. The catheter is passed down the esophagus and gets arrested.
   - (*v*) Diagnosis of imperforate anus in newborn in which catheter cannot be passed

## FOLEY'S CATHETER (Fig. 47.11A)

Foley's catheter is a single use self-retaining urinary catheter made of latex rubber sterilized by exposure to gamma radiation. Usually No. 14 or 16 F (French size) catheter is used for catheterization of a female urethra. The catheter has two channels: a big channel for drainage of urine and a small one for inflating the subterminal bulb which makes the catheter self-retaining. The capacity of the bulb is 15 to 50 mL (specified by the manufacturer). It has a solid tip and two subterminal openings for drainage of urine.

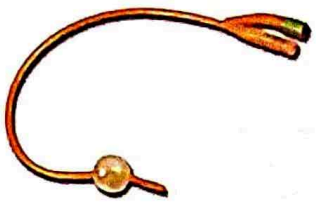


**Fig. 47.11:** (A) Foley's catheter

**Uses**

1. Retroverted gravid uterus with retention of urine.
2. Management of obstetric conditions for accurate monitoring of the urine output. (*i*) Severe pre-eclampsia during labour. (*ii*) Eclampsia (*iii*) Abruptio placentae (*iv*) Disseminated intravascular coagulation (DIC).
3. In prolonged obstructed labour, destructive operations, rupture uterus and suspected bladder injury. To prevent development of a vesicovaginal fistula due to ischemic necrosis of the bladder wall and vagina and to treat such a fistula the catheter is kept in situ for 10 to 15 days.
4. Ripening the cervix for induction of labour. A No. 20 F catheter is passed through the cervix and its bulb is inflated with water to 50 mL.
5. Induction of second trimester abortion for extra-amniotic administration of ethacridine lactate or prostaglandins etc.
6. Cord prolapse. If the fetus is alive and viable, the bladder is inflated with 500 mL of normal saline using Foley's catheter to displace the presenting part up to relieve cord compression. This is aided by administration of ritodrine infusion to relax the uterus.
7. For application of cerclage operation with herniation of membranes, inflated bulb of Foley's catheter is used to push the membranes upwards.
8. In atonic postpartum haemorrhage, a 24 French Foley's catheter is introduced in the uterus, its balloon is inflated with 60–80 mL normal saline to provide tamponade effect to the uterine surface (see Chapter 25 for details).

## MALECOT CATHETER (Figs. 47.11 B)

It is self-retaining catheter with Malecot wings to promote catheter retention.

**Uses:**

1. Vaginal and cervical reconstruction surgery.
2. After drainage of hematometra it can be left in cervix and uterus for better drainage.



**Fig. 47.11:** (B) Malecot catheter

## MUCUS SUCKER (Fig. 47.12)

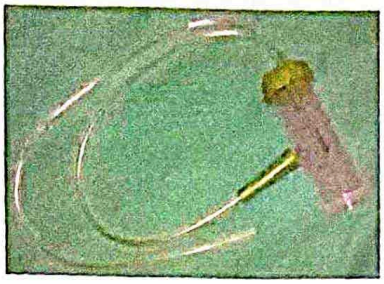


Fig. 47.12: Mucus sucker

Mucus sucker is made of glass which is sterilized by boiling, autoclaving or in hot air oven. It is 42 cm long, has a glass safety chamber in the center and two rubber catheters at either end. The mucus aspirated through one end will enter and collect in the safety chamber first and would not enter in the other end. There is an opening with a stopper at the top of the chamber to remove the mucus collected in the chamber. It is of two types: disposable (once only use preferred) and metal (for repeated use after sterilization).

### Uses

Mucus sucker is used to remove mucus and other secretions from nose, mouth and pharynx of the newborn after delivery of the head prior to the respiratory efforts for better aeration of lungs and to prevent development of asphyxia.

Metal sucker can cause injury to the pharynx of the neonate. However, it still has a place in case of electricity failure and in domestic practice.

Students may be asked about the neonatal resuscitation in labour room, Apgar scoring, meconium aspiration and asphyxia neonatorum (see chapters 27 and 28 for details).

## UTERINE DILATORS (HEGAR'S DILATOR) (Fig. 47.13A)

Uterine Hegar dilator is a solid rod curved and somewhat tapering toward the tip and may be single ended or double ended. This curve is shallow and the dilating portion is within terminal 1.5 cm of the dilator. There is a difference of three millimeters in the diameter near the tip and the maximum dilating portion of the dilator. The smallest size is 1/2 and largest size is 11/12 (the numbers demonstrate the diameter of dilator in mm).

### Indications in Obstetrics

1. Prior to suction evacuation for first trimester pregnancy termination.
2. Prior to suction evacuation of a hydatidiform mole.
3. Before insertion of IUCD
4. Before removal of impacted IUCD in uterus by curettage.
5. Retrograde dilatation of the cervix when it is found to be closed at the time of hysterotomy or an elective cesarean section (usually dilatation is done with finger).
6. Diagnosis of an incompetent os. Passage of a size 8 Hegar's dilator through the internal os in the non-pregnant state without resistance or causation of pain is said to be diagnostic of incompetent os.

### Procedure to Use

The smallest dilator is held like a pen and its lubricated (with lignocaine jelly) tip is passed into the cervical canal. The dilator is held in position for 2–3 seconds and is then removed so that the next dilator may be passed (see Chapter 42 for details). Hegar's dilator has a rapid increase from the diameter at the tip to the maximum diameter. Hence, it may fail in some cases to dilate the cervix.

The amount of dilatation depends upon the procedure performed for evacuation of products and is usually one size smaller than the size of Karman's cannula used (e.g. for 8 and 9 weeks pregnancy, size 8 Karman's cannula is used while dilatation is performed up to size 7 for air tight contact and evacuation). When dilatation and evacuation is performed then more dilatation is needed (size 10–12 Hegar) so that ovum forceps can be easily negotiated in the uterine cavity.

### Complications

1. Irreversible damage to the cervix if too much dilatation is done forcibly.
2. Haemorrhage from injury to the descending cervical artery
3. Cervical perforation, uterine perforation (see Chapter 42 for details of management).
4. Introduction of infection
5. Incompetent os from excessive dilatation of the cervix.

## HAWKIN'S AMBLER DILATOR (Fig. 47.13 B)

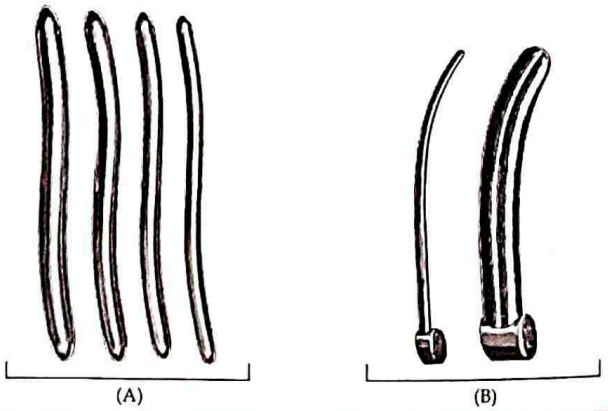



Fig. 47.13: (A) Hegar's dilators; (B) Hawkin's Ambler dilators

Hawkin's Ambler dilator is a single ended cervical dilator with a disc-shaped finger grip at the non-dilating end. It is less curved than Hegar's dilator. Its sizes vary from 3/6 to 18/21. The indications, procedure to use and complications are same as Hegar's dilator given above.

**Features.** It is a graduated malleable metallic dilator and may even work where Hegar's dilators fail.

## UTERINE SOUND (Fig. 47.14)

Uterine sound is 30 cm long, of which 5 cm length is that of its handle. It is bent to an angle of 150° at a distance

of 2½ inches from its tip, i.e. at a distance of normal uterocervical length. It is a pointed and malleable metallic instrument which is marked in centimeters on one side and in inches on the other side. As a general principle, pregnancy or suspected pregnancy is a contraindication to uterine sounding for two reasons. The sound may rupture the membranes and so may cause abortion. Even during dilatation and suction evacuation the uterine cavity is not sounded because the uterus is very soft during pregnancy and can be perforated easily by the sound.



**Fig. 47.14:** Uterine sound

**Uses**

1. It helps in delineating the position of the uterus and the length of the uterine cavity in dilatation and evacuation and dilatation and curettage operations.
2. It is used to sound misplaced copper T inside the uterine cavity.
3. To find the direction of the cervical canal and its length prior to insertion of laminaria tents.
4. To find the direction of the cervical canal in the first trimester pregnancy termination by dilatation and suction evacuation.
5. Diagnosis of incompetent os. Passage of an olive tipped uterine sound through the internal os without resistance or pain is diagnostic of incompetent os **(Shirodkar's test)**. It is done in non-pregnant state.
6. Diagnosis of a vesicular mole. Passage of the sound for the distance of at least 10 cm without resistance or rupture of membranes is diagnostic of a vesicular mole **(Acosta-Sison's test)**. The test is false positive in presence of foetal death and maceration and prior to 12 completed weeks of pregnancy, i.e. prior to fusion of decidua capsularis and decidua parietalis.

## OVUM FORCEPS (HAYWOOD SMITH'S) (Fig. 47.15)

Ovum forceps may have spoon shaped, blunt and fenestrated ends which just come in contact with each other when the forceps is closed. This achieves a good grip on the structure held without nipping it at the base. Thus, during removal of an adherent piece of retained tissue or placenta, the underlying uterine wall is not nipped. There is no catch (lock) on the handles so that the instrument has no crushing action and any structure inadvertently held that should not be held is not damaged (compare with sponge holding forceps which has a lock which should not be used for evacuation).

**Uses**

1. First trimester pregnancy termination to remove the products of conception after rapid dilatation of the cervix.
2. Completion of first trimester spontaneous abortion
3. Removal of bits of placenta and membranes after second trimester incomplete abortion.
4. Removal of bits of placenta in puerperium for secondary postpartum haemorrhage.

**Procedure**

The cervix is exposed, held with sponge forceps and dilated sufficiently to permit the passage of the ovum forceps. The ovum forceps is inserted in the uterus with the closed blades. After reaching the uterine cavity, the blades are opened to catch hold of the products of conception. The blades are then closed and the products are brought out. This is repeated several times and all the products (both lying free in uterine cavity or attached to the uterine wall) are removed without injuring the uterine wall as it has got no lock and the obstetrician knows how much force is being applied. (See Chapter 42 for details of operation).

**Complications**

1. Uterine perforation
2. Injury to intra-abdominal structures like small bowel, colon, omentum, etc., secondary to uterine perforation especially if perforation is not diagnosed and evacuation of the uterine contents is continued.
3. Intrauterine infection
4. Incomplete abortion

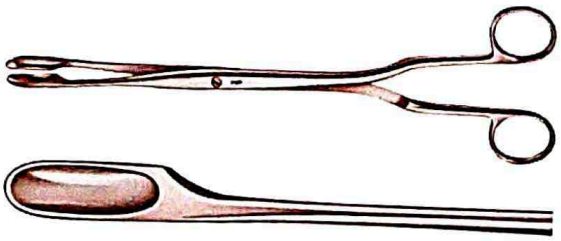


**Fig. 47.15:** Ovum forceps

### Uterine curette (Fig. 47.16 A)

It is long metallic instrument with two different sized ends for uses in different patients. The most common instrument has got one blunt end (for obstetric use) and one sharp end (for gynecological use). However, some instruments have both sharp ends with different sizes.



**Fig. 47.16: (A)** Uterine curette

**Uses**

1. In dilatation and curettage for medical termination of pregnancy (less preferred). It can be used at end of suction evacuation to confirm complete emptying.
2. Blunt curette is used for dilatation and evacuation for incomplete miscarriage and medical termination of pregnancy.

3. Rarely after evacuation of molar pregnancy, blunt curettage can be done (though it is not practised these days).

Procedure and complications are like ovum forceps (see above).

Students may be asked about operative details of dilatation and curettage, dilatation and evacuation and complication (see Chapters 13 and 42 for details).

## FLUSHING CURETTE (Fig. 47.16 B)

Flushing curette is a type of metallic blunt curette which was used in the past for dilatation and evacuation by flushing the uterus with betadine or other antiseptic solutions. It is not used in modern obstetrics.



**Fig. 47.16: (B)** Flushing curette

### Post Placental IUCD Insertion Forceps (Kelly's forceps) (Fig. 47.17)

Post placental IUCD insertion forceps is a large forceps resembling sponge holding forceps used for insertion of IUCD Copper T 380 A just after delivery of the baby and placenta high up in the uterus near fundus. The Government of India is now encourging it as women are more amenable to accept contraception at this time and it has been found to be safe and effective. There is slightly increased risk of expulsion (5%). But still there is 95% continuation rate which is very good as women often don't come for contraception later.

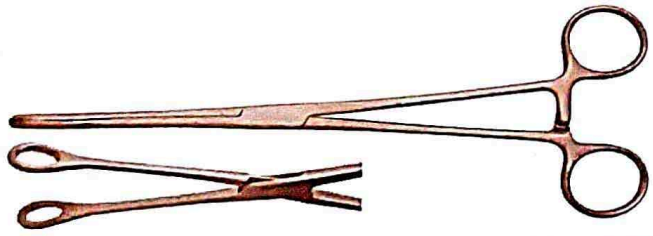


**Fig. 47.17:** Post placental IUCD insertion forceps (Kelly's forceps)

### Uterine dressing forceps (Fig. 47.18)

Uterine dressing forcep is a long forcep with two blades having transverse serrations.

**Uses**

1. For cleaning the uterine cavity after dilatation and evacuation operation.
2. For dilation of the cervix and drainage of lochiometra (collection of lochia in uterus).
3. For dilatation of the cervix and drainage of pyometra (collection of pus in uterus).
4. It can be used to pack the uterine cavity with long ribbon gauge in postpartum hemmorhage

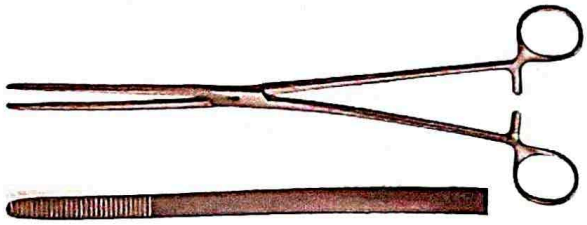


**Fig. 47.18:** Uterine dressing forceps

## LAMINARIA TENTS (Fig. 47.19)

Laminaria tents are slow cervical dilators made from dried seaweed Laminaria digitata or Laminaria japonica. They are 6–8 cm long, shaped into cylindrical forms ranging in diameter from 2 to 4 mm for a small tent to 8 to 10 mm for a large tent. A string is looped through its one end for easy removal. It is sterilized by gamma irradiation or ethylene oxide gas or absolute alcohol.

**Uses**

Laminaria tents are used to achieve slow cervical dilatation in the following conditions.

1. Second trimester pregnancy termination in conjunction with
   a. intramuscular prostaglandins.
   b. intra-amniotic hypertonic saline.
   c. intravenous oxytocin infusion.
2. Evacuation of a vesicular mole
3. Ripening of the cervix for induction of labour.

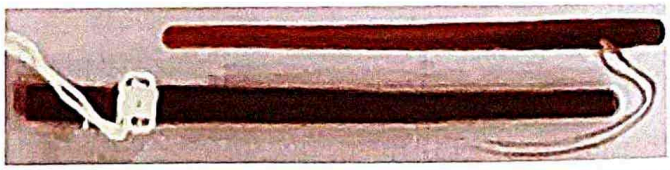


**Fig. 47.19:** Laminaria tents

### Procedure

Sterile tent is grasped with laminaria tent introducing forceps and inserted into the cervical canal until its distal end has just passed beyond the internal os (see **Figs. 42.2 A** to **42.2 C** in Chapter 42). Laminaria tents are intensely hygroscopic and they swell 3 to 5 times of the original diameter in moist environment such as the cervical canal. The greatest degree of swelling occurs within the first 4 to 5 hours after insertion though further dilatation continues subsequently (see Chapter 42 for details).

## LAMINARIA TENTS INTRODUCING FORCEPS (Fig. 47.20)

Laminaria tents introducing forceps is a long metallic forceps (can be confused with uterine dressing forceps). It has got a lock and a groove (depression) on each blade for firmly holding the laminaria tent for its better and easy insertion in the cervix.

### Complications

1. Infection
2. *Failure.* If the tip does not project just beyond the internal os but lies in the cervical canal, the part of the canal lying above the tent does not get dilated.
3. Migration of the tent into the uterine cavity.
4. *False passage.* The tent may be passed into a false passage.
5. *Uterine rupture.* It has been claimed that the risk of uterine rupture is increased when prostaglandins are administered for induction of second trimester abortion with the tents in the cervical canal.

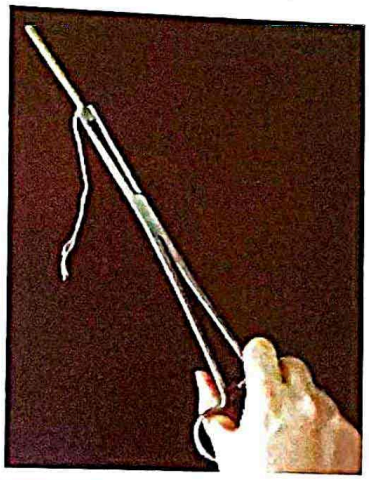
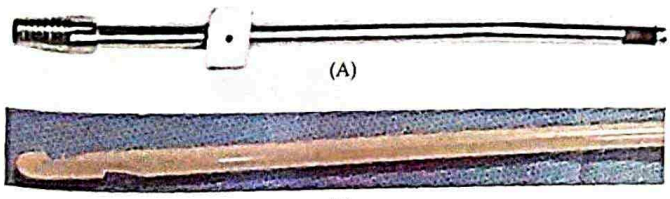


**Fig. 47.20:** Laminaria tents introducing forceps

## SUCTION CANNULA (Fig. 47.21)

Suction cannula is made of stainless steel or plastic. The steel cannula is sterilized by boiling or autoclaving. The metal cannula is 25 cm long, available in diameters of 14 and 16 mm and has a rounded tip and two subterminal openings. Steel cannulas are not used in modern obstetrics as they are more traumatic.



(A)



(B)

**Fig. 47.21: (A)** Metallic suction cannula; **(B)** Plastic suction cannula (Karman's type)

A plastic cannula (Karman's type are mostly used these days) is sterilized by gamma rays or by exposure to ethylene oxide gas or immersion in antiseptic solutions. It should be disposed off after one use. The flexible cannula is available in 4–12 mm diameter sizes. The size of the Karman's cannula is usually one size less or equivalent to the weeks of pregnancy being terminated (e.g. for 8 and 9 weeks pregnancy, size 8 Karman's cannula is used). Plastic cannula is preferred being less traumatic and is transparent through which sucked products can be seen. It has a rounded tip and two subterminal sharp triangular openings with overhanging convex hoods. Its tip bends without blocking its opening and collapses without perforating the uterus.

#### Uses

1. Medical termination of pregnancy during the first trimester is done by suction evacuation after rapid cervical dilatation.
2. Completion of inevitable abortion during first trimester of gestation.
3. Large suction cannulas are used for suction evacuation of vesicular moles.
4. Large suction cannulas are used for suction evacuation during the early second trimester pregnancy terminations.

#### Procedure

See Chapter 42 for details.

#### Complications

1. Uterine perforation
2. Damage to bowel or omentum by aspiration into the cannula after uterine perforation.
3. Haemorrhage from incomplete evacuation, uterine perforation, etc.
4. Postabortal infection of the genital tract.
5. Tip of the flexible plastic cannula may break inside the uterine cavity and be retained. It is removed by suction with a large cannula or by a curette. Hysteroscopy is useful in detection and removal of the broken tip.

Students may be asked about various types of abortion, vesicular mole, steps and complications of suction and evacuation.

## LONG STRAIGHT HEMOSTATIC FORCEPS (Fig. 47.22)

It is a long metallic forceps which resemble a straight artery forcep used for hemostasis but has limited use in obstetric practice. It can rarely be used as a pedicle clamp (proper Kelly's clamps are preferred) during cesarean hysterectomy. Rarely it can be used for cord clamping when disposable umbilical cord clamps or Kocher's clamps are not available.

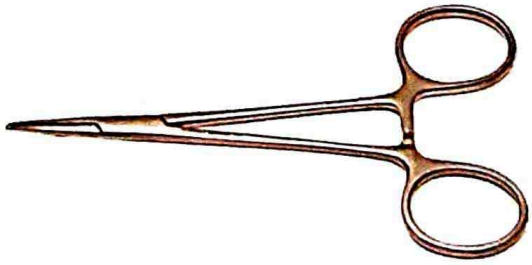


**Fig. 47.22:** Long straight hemostatic forceps

## LONG STRAIGHT SCISSORS (Fig. 47.23)

They are stout metallic scissors with sharp edges for better cutting. They should not be autoclaved but should be chemically sterilized to maintain their sharpness.

### Uses

1. To give cut of episiotomy as an alternative to episiotomy cutting scissors (see below).
2. To cut the umbilical cord between two Kocher's clamps after delivery of baby.
3. To cut the sutures (vicryl or catgut) after tying knots in cesarean delivery and cesarean hysterectomy.



**Fig. 47.23:** Long straight scissors

## UMBILICAL CORD CUTTING SCISSORS (Fig. 47.24)

Umbilical cord cutting scissors are made of stainless steel and sterilized by immersion in glutaraldehyde solution (being sharp). Their blades are curved so that on closing they meet at their tips leaving a gap in between.



**Fig. 47.24:** Umbilical cord cutting scissors

### Indications

Umbilical cord cutting scissors are used for cutting the umbilical cord of the newborn baby. The cord tends to slip while being cut with ordinary scissors because it contains Wharton's jelly and is covered by smooth amniotic membrane. This instrument avoids that as the tips of the blades meet around the cord.

Students may be asked about the time of clamping the umbilical cord, advantages and disadvantages of early clamping and cutting the cord.

## DISPOSABLE CORD CLAMP (Fig. 47.25)

Disposable cord clamp is a plastic clamp available in a sterile pack. It has got serrated margins and a lock for better hold of umbilical cord for effective closure of umbilical arteries and vein. After few days, it itself falls off alongwith the remaining umbilical cord stump. Sometimes aluminium metal clips are also used which are sterilized by immersing in antiseptic solution.

Students may be asked about distance of cord clamp from umbilicus, advantages and disadvantages of early cord clamping (see Chapters 16 and 27 for details).

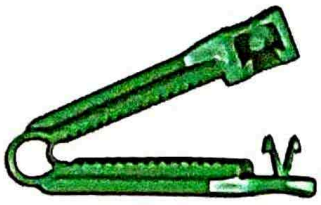


**Fig. 47.25:** Disposable cord clamp

## EPISIOTOMY CUTTING SCISSORS (Fig. 47.26)

Episiotomy cutting scissors have a suitable curvature (bent on edge) for cutting episiotomy. They are angled on one side and one blade is more sharply pointed than the other. Thin blade is kept outside on the vulva and the thick blade with blunt tip is kept in vagina to avoid injury.

The students can be asked about indications, types and complications of episiotomy (see Chapters 16 and 42 for details).

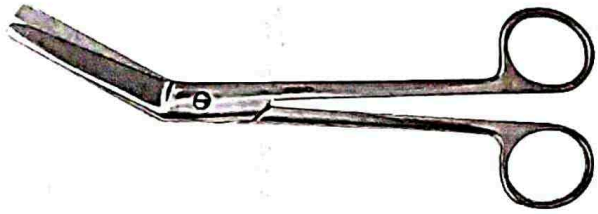


**Fig. 47.26:** Episiotomy cutting scissors

## DOYEN'S RETRACTOR (Fig. 47.27)

Doyen's retractor has a blade with less depth and a uniform curvature. This instrument is used for retraction of the abdominal wall and the bladder suprapubically during abdominal operations such as:

1. Cesarean section
2. Operation for ruptured ectopic gestation
3. Obstetric hysterectomies



**Fig. 47.27:** Doyen's retractor

Doyen's retractor is used after the peritoneum has been opened. The broad retracting surface achieves good retraction. The solid blade compresses the cut edges of the abdominal wall and achieves a reduction in blood loss from injured vessels.

After holding the foetal head with right hand, the Doyen's retractor is removed for sometime for better delivery of foetal head and body. It is reinserted after delivery of the baby for retraction of bladder for easy suturing of uterine wound. After stitching is completed, peritoneal toilet is done and then the retractor is removed.

Students may be asked about types of cesarean, its

indications, contraindications, steps, complications and methods to reduce cesarean rates (see Chapter 42 for details).

## DEAVER'S RETRACTOR (Fig. 47.28)

Deaver's retractor is a long strip of metal which is bent into the shape of retractor. It is available in three sizes: small, medium and large. The blade is thin. Hence, the instrument occupies little space and is a good retractor.



Fig. 47.28: Deaver's retractor

Deaver's retractor is sometimes used in cesarean and cesarean hysterectomy for retraction of the sides of the incision and intraperitoneal structures during operations.

## MORRIS RETRACTOR (Fig. 47.29)

Morris retractor is a right angled stout retractor. It can also be used instead of Doyen's retractor to retract bladder and abdominal wall in cesarean delivery and cesarean hysterectomy.



Fig. 47.29: Morris retractor

## GREEN-ARMYTAGE HEMOSTATIC FORCEPS (Fig. 47.30)

Green-Armytage hemostatic forceps is an atraumatic clamp with wide ends which have transverse ridges on inner side.

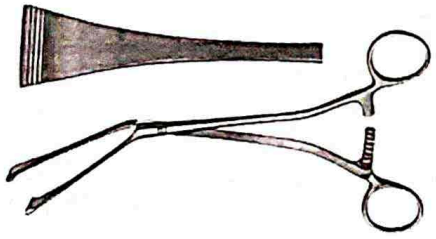


Fig. 47.30: Green-Armytage hemostatic forceps

### Use

Its only use is to catch hold of the uterine wound edges in lower segment cesarean delivery (not in classical cesarean delivery). Out of total four Green-Armytage forceps one each is used for the two angles of the cesarean uterine wound and one each is applied to the two flaps. A bleeder can also be effectively caught with it for better hemostasis. It ensures that uterine margin is not missed during cesarean stitching. Allis tissue forceps are more often used these days as they give better grip (see Allis forceps and Chapter 42 [v] for details).

## BABCOCK'S FORCEPS (Fig. 47.31)

Babcock's forceps is a non-traumatic instrument with fenestrated triangular blades and grooved jaws. The small forceps is about 10 cm long and has small blades.

### Uses

1. To hold the Fallopian tubes in the following operations.
   a. Tubal ligation
   b. Tuboplasty
   c. Salpingectomy for tubal ectopic gestation
2. Dissection of vascular sheaths, ligation of the anterior division of the internal iliac artery.
3. Third degree perineal tear repair to hold the mucosal edges and the sphincter
4. Rectovaginal fistula repair

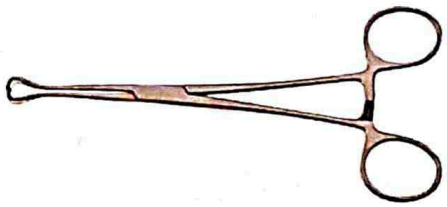


Fig. 47.31: Babcock's forceps

## PINARD'S FOETAL STETHOSCOPE (Fig. 47.32)

Pinard's foetal stethoscope is made of either aluminium or wood. French doctor Pinard (1880) developed it. It is funnel-shaped with a broad flat disc with a central perforation. It is attached at the narrow end of the funnel at right angles to its long axis. The rim of the broad end is rounded to avoid pain to patients during pressing the instrument onto the abdominal wall.



Fig. 47.32: Pinard's foetal stethoscope

### Indications

Pinard's foetal stethoscope is used to hear foetal heart sounds from maternal abdomen in the antenatal and the intrapartum periods.

Autoclaved instrument is also used to listen to foetal heart by obstetrician just before giving incision during cesarean to ensure that fetus is alive. The broad and open end of the instrument is placed firmly at 90° to the maternal abdomen where foetal heart is expected to be best heard. The obstetrician puts their ears firmly to the narrow aural end for better hearing of the foetal heart.

The students may be asked about antepartum and intrapartum foetal monitoring (see Chapters 15 and 17 for details).

## ULTRASONIC DOPPLER (DOPTONE) (Fig. 47.33)

The 'Doptone' is considered as one of the most important instruments that were invented in obstetrics which has quite considerably changed the practice of obstetrics since the 1960s. It is a hand held ultrasound tool. It works by sending a high frequency sound wave into abdomen, and then retrieving the sound wave as it bounces back. The doptone is useful underwater, where other monitoring tools might be damaged. As it is hand-held, a doptone cannot be used when continuous monitoring is required.




**Fig. 47.33:** Ultrasonic Doppler (Doptone)

Students may be asked about various aspects of foetal heart rate monitoring, non-reassuring foetal status (foetal distress), cardiotocography (see Chapters 15 and 17 for details).

## CARDIOTOCOGRAPH (Fig. 47.34)

Cardiotocograph is a machine to record the foetal heart rate and contractions of uterus during labour.

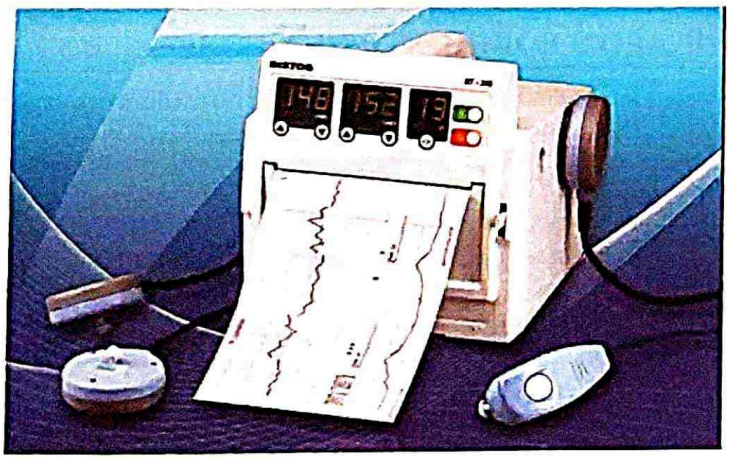


**Fig. 47.34:** Cardiotocograph

The cardiotocograph has a disc-shaped ultrasound transducer which is strapped onto the mother's abdomen over the area of the uterus. The ultrasound waves are reflected from different surfaces in the uterus and detected by the transducer. The foetal heart moves every time it beats and this beating movement is detected by the changing ultrasound waves being reflected back to the transducer. From this, the cardiotocograph can calcutate the foetal heart rate. A second ultrasound transducer can also be used at the same time so that twins can be monitored. The second transducer has a different frequency so the two signals can be separated. The details are given in chapters 15 and 17.

## OBSTETRIC FORCEPS

Obstetric forceps are a pair of stainless steel instrument designed to extract foetal head.

The details of parts of forceps, types of forceps, their application, technique, indications, contraindications and complications are given in Chapter 42[ii].

## WRIGLEY'S FORCEPS (Fig. 47.35)

Wrigley's forceps is a light weight, 27.5 cm in length forceps having an English lock. It is used for outlet forceps delivery and has more pronounced cephalic curve with a normal pelvic curve. It can also be used during cesarean delively for extraction of foetal head. It is one of the most commonly used forceps and is often asked in examinations (see Chapter 42[ii] for details).

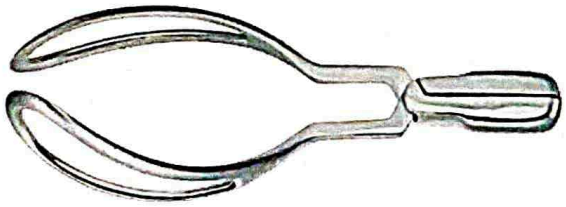


**Fig. 47.35:** Wrigley's forceps

## SIMPSON'S SHORT FORCEPS (Fig. 47.36)

Simpson's short forceps is a short forceps like Wrigley's forceps (27.5 cm) with a short shank, small handle and with identical blades. They do not have a pelvic curve. They have an English lock. Usage is similar to Wrigley's forceps.

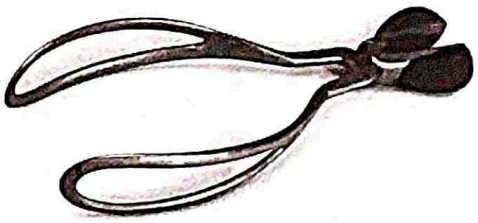


**Fig. 47.36:** Simpson's short forceps

## LONG CURVED OBSTETRIC FORCEPS

Long curved obstetric forceps are commonly used in low mid forceps operations. It has long blades and shanks with English lock. The various types of long curved forceps are Simpson's long forceps, Haig Ferguson's forceps and Das forceps discussed later. The students should be aware of different types of obstetric forceps, their parts and the curves, identification of blades, types of forceps application, functions of the obstetric forceps, common indications of forceps delivery, pre-requisites to be fulfilled before application of forceps, steps of forceps application,

direction of pull during delivery. They should also be aware of prophylactic forceps, trial of forceps and failed forceps (see Chapter 42[ii] for details).

## SIMPSON'S LONG FORCEPS (Fig. 47.37)

Simpson's long forceps is 35 cm long and the maximum distance between blades is 8.5 cm. It has a shallow cephalic curve with 11.25 cm radius while the radius of the pelvic curve is 17.5 cm. Shanks are 6.25 cm long, parallel, diverging rapidly from the English lock so that the distension of maternal perineum is marked. Blades are relatively long measuring 12.5 cm.

Indications are like any long forceps used for low mid cavity forceps.

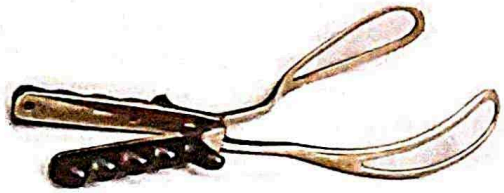


**Fig. 47.37:** Simpson's long forceps

## HAIG FERGUSON'S FORCEPS (Fig. 47.38)

Haig Ferguson's forceps are long curved obstetric forceps with their blades having fenestration in their blades (F for Ferguson, F for fenestration).

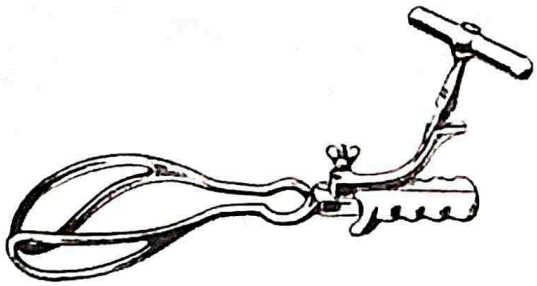


**Fig. 47.38:** Haig Ferguson's forceps

## DAS FORCEPS (Fig. 47.39)

Devised by Dr. Kedarnath Das from Kolkata, India, they are long but lighter forceps suited for Indian women. They are commonly used for low mid cavity application.

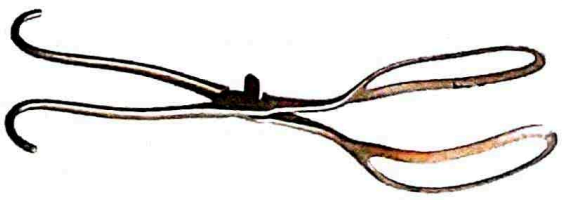


**Fig. 47.39:** Das forceps

## PIPER'S FORCEPS (Fig. 47.40)

Piper's forceps are long forceps specially suited for the delivery of the after-coming head of breech. They are not usually available in India (see Chapter 23 for details). They have additional perineal curve.

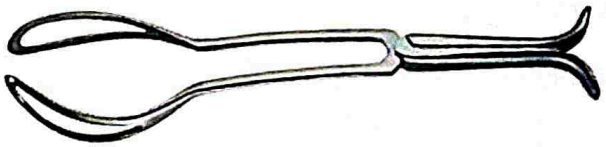


**Fig. 47.40:** Piper's forceps (Reverse pelvic curve)

## KIELLAND FORCEPS (Fig. 47.41)

Kielland forceps are rotational forceps without a pelvic curve and can be applied in antero-posterior position. They are suited for delivery of foetal head in occipito-posterior and occipito-transverse position (see Chapters 23 and 42[ii] for details).

They need extensive training as they can cause spiral tears. They are hardly ever used in India.

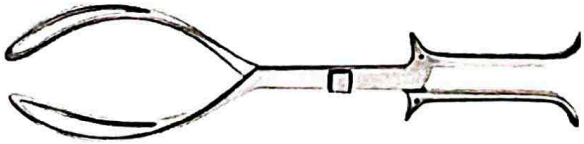


**Fig. 47.41:** Kielland forceps

## AXIS TRACTION DEVICE (Fig. 47.42)

Axis traction device is used for giving traction to the long forceps and is not generally used these days as high mid forceps delivery is not performed any more.

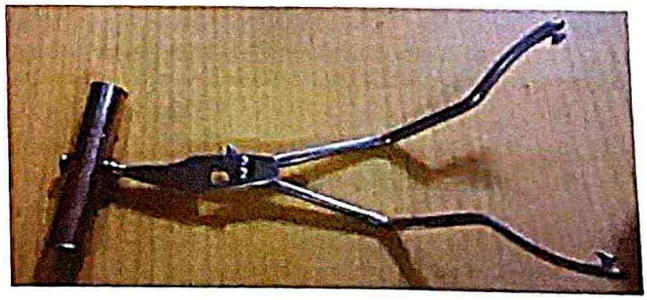


**Fig. 47.42:** Axis traction device

## VACUUM EXTRACTOR OR VENTOUSE (Figs. 47.43 A and B)

Vacuum extractor or ventouse is an obstetric instrument designed for extraction of foetal head. It consists of suction cups made of metal-like steel (Fig. 47.43 A) or silastic (Fig. 47.43 B).

Silastic cups are more pliable. The cup works by producing a physiological swelling **(chignon)** on the foetal scalp.

The students should know the details of procedure, pressure raising, indications, contraindications, complications, causes of failure, relative advantages and disadvantages of ventouse over forceps (see Chapter 42[ii] for details).

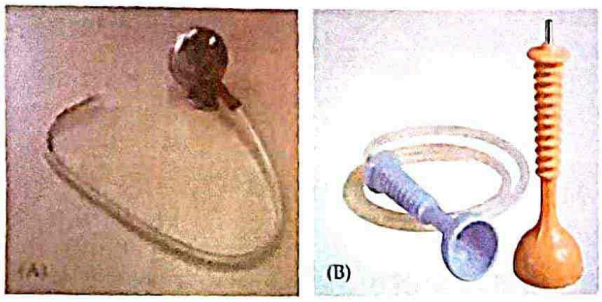



**Fig. 47.43:** Vacuum extractor or Ventouse; **(A)** Metalic cup; **(B)** Silastic cup

## AMNIOTOMY HOOK (HAMILTON'S) (Fig. 47.44)

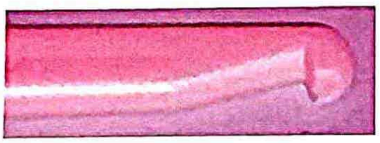


**Fig. 47.44:** Amniotomy hook (Hamilton's)

It is made of stainless steel but can be made of wood also (single use) with its end being angulated with sharp tip to rupture membranes in low amniotomy. The students should know details of indications, contraindications and complications of amniotomy and induction and augmentation of labour (see Chapter 18 for details).

## DREW SMYTH'S CATHETER (Fig. 47.45)

Drew Smyth's catheter is an S-shaped instrument with a side opening to drain liquor amnii. It has a spring loaded stylet with a blunt tip.

### Indications

1. *High amniotomy.* It was used in the past for high amniotomy in cases of hydramnios by passing it between the uterine wall and the membranes to rupture the membranes at high level and drain the liquor amnii slowly. It is not used in modern obstetrics. Instead, controlled low amniotomy is performed (see Chapter 18 for details).
2. To drain a hydrocephalic head through a spina bifida in case of a breech delivered up to the head.

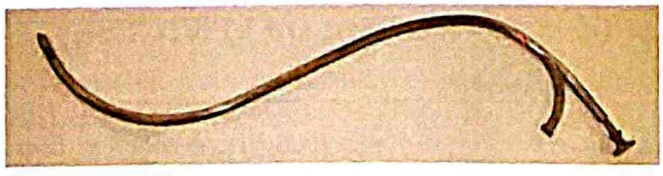


**Fig. 47.45:** Drew Smyth's catheter

## INSTRUMENTS FOR DESTRUCTIVE OBSTETRIC OPERATIONS

The destructive instruments have a limited role to play in modern obstetrics. However, the destructive operations and instruments are still asked in practical examination with some examiners being very fond of them. Hence, some details of the instruments are given here. (see Chapter 42[iv] for details of destructive operations)

## CRANIOCLAST (Fig. 47.46 A)

### (German: kranion, skull: klae; to break in pieces)

Cranioclast is a sturdy forcep consisting of two concavo-convex blades.

Convex blade is pushed in through the opening in the skull and the concave blade is applied on the outside preferably over face. Both the blades face in the same direction and are locked and screwed. The head is firmly grasped and partially crushed. After crushing the vault or the base of the skull, it can be used for extraction of the head by traction.

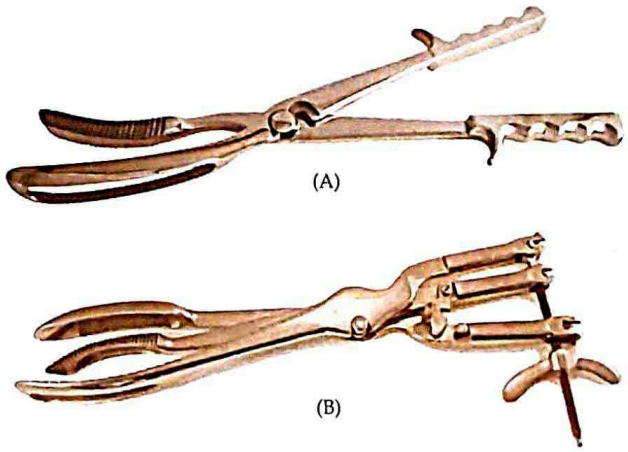



**Fig. 47.46: (A)** Cranioclast; **(B)** Cephalotribe

## CEPHALOTRIBE (Fig. 47.46 B)

### (German: kephale, the head; tribo, to bruise)

Cephalotribe is a 42 cm long instrument made up of three strong blades, one central and two laterals marked 1 and 2 joined with a screw handle. The central serrated blade is passed through the perforation hole in the skull while the two other blades are placed on sides of head (see Chapter 42 for details).

### Indications

1. To crush the vault and base of foetal skull after craniotomy.
2. To extract foetal head after craniotomy and crushing.
3. Dead fetus with obstructed labour in:
   a. Vertex presentation with contracted pelvis
   b. Persistent brow

c. Impacted mento-posterior
d. Deep transverse arrest
e. Arrested after-coming head in breech

4. Hydrocephalus
   In modern obstetrics it is hardly ever used.

## OLDHAM'S PERFORATOR (Fig. 47.47 A)

Oldham's perforator is a long instrument with long blades with triangular cutting edge with only one shoulder. The blades have outer cutting edges and they open when the stout handles are moved apart. There is no spring or lock.

### Procedure

See Chapter 42[iv] for details.

### Uses

1. Craniotomy
2. Sometimes, it can be used for evisceration. (See Chapter 42 for details)

## SIMPSON'S PERFORATOR (Fig. 47.47 B)

Simpson's perforator is 28.5 cm long with blades having triangular tips with outer cutting edges with two shoulders on the blades to prevent slipping and excessive penetration of the foetal head by the blades during craniotomy. The handles have a flat spring in between them with a locking system.

### Indications

Same as Oldham's perforator.

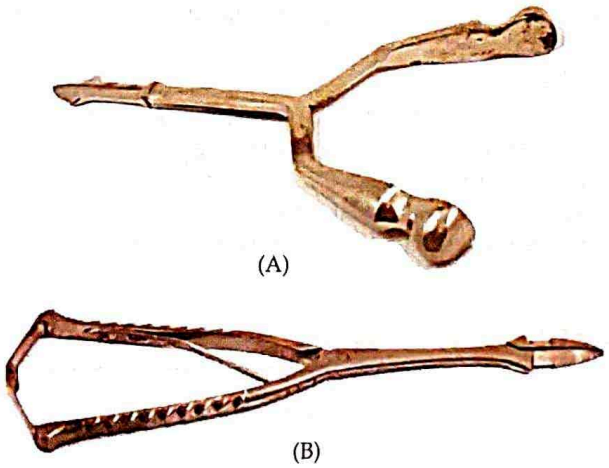



**Fig. 47.47: (A)** Oldham's perforator; **(B)** Simpson's perforator

## DEBDAS'S CRANIOTOMY SET (Fig. 47.48)

Dr Debdas from Jamshedpur has devised this simple equipment for performing craniotomy. After preliminary examination and preparation for vaginal delivery in case of obstructed labour with dead fetus, the foetal head is drilled. Suction cannula is inserted in the hole and brain matter is removed by sucking out with cannula and suction machine or manually. When the foetal head becomes much smaller, suction cannula is removed. Two Foley's catheters are inserted through craniotomy hole and their bulbs inflated with 30 mL saline each. Foetal head is then extracted by pulling on the Foley's catheters.

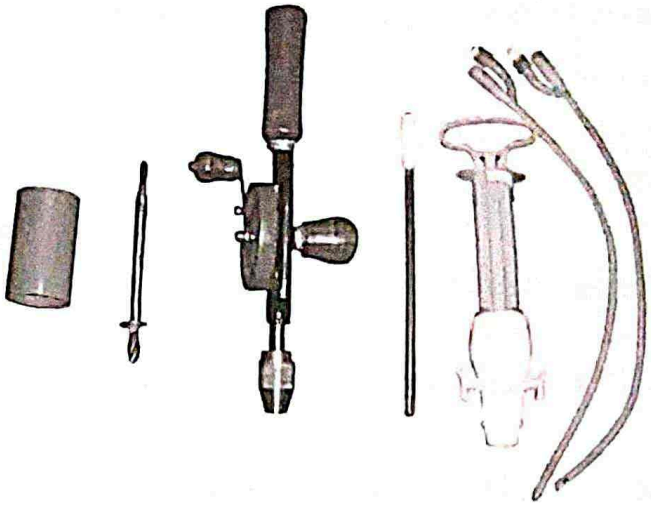


**Fig. 47.48:** Debdas's craniotomy set

## HOOK WITH CROCHET (Fig. 47.49)

### Uses of Crochet

1. To extract the decapitated head
2. To extract a perforated after-coming head of a dead fetus.
3. To extract a hydrocephalic head

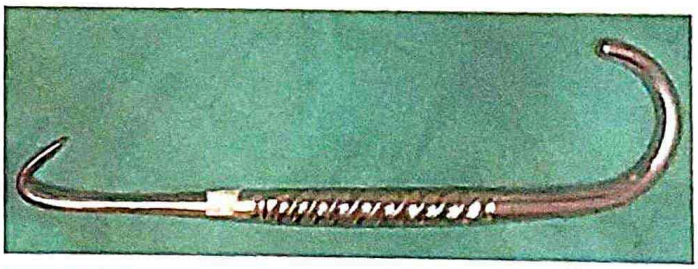


**Fig. 47.49:** Hook with crotchet

### Indications for the Use of the Hook

1. To apply anterior or posterior groin traction for a breech extraction in a dead fetus as the instrument is traumatic.
2. To pull down a foetal leg in case of a transverse lie with dead fetus and advanced labour but in the absence of imminent uterine rupture.

## DECAPITATION HOOK WITH KNIFE (JARDINE'S) (Fig. 47.50)

Decapitation hook with knife is used in decapitation operation in neglected transverse lie. For decapitation

of the first baby of a locked twin, a simple scalpel or embryotomy scissors are enough.

The students should be aware of management of transverse lie and neglected shoulder presentation and its complications. In modern obstetrics, decapitation is hardly ever used except rarely in rural areas. Most obstetricians perform cesarean delivery in a case of transverse lie even for dead fetus in modern era for the sake of the mother (see Chapters 23 and 42[iv] for details).

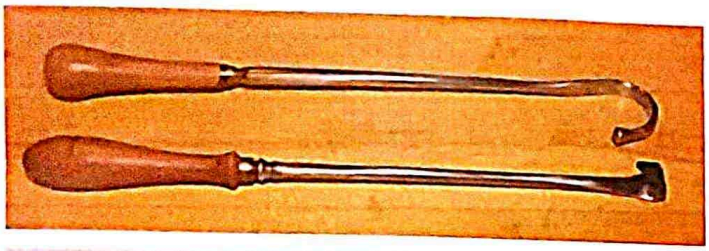


**Fig. 47.50:** Decapitation hook with knife

## BLOND-HEIDLER DECAPITATION SAW (Fig. 47.51)

Blond-Heidler decapitation saw is a long wire saw-made by twisting together long metal wires. The saw is covered with rubber tubing except over 7 cm of its central part which cuts through foetal neck. The rubber covering protects maternal soft parts from injury during the sawing. The ends of the wire saw have knobs so that handles can be fitted to the ends. A thimble with a wire loop on top is supplied along with the saw.

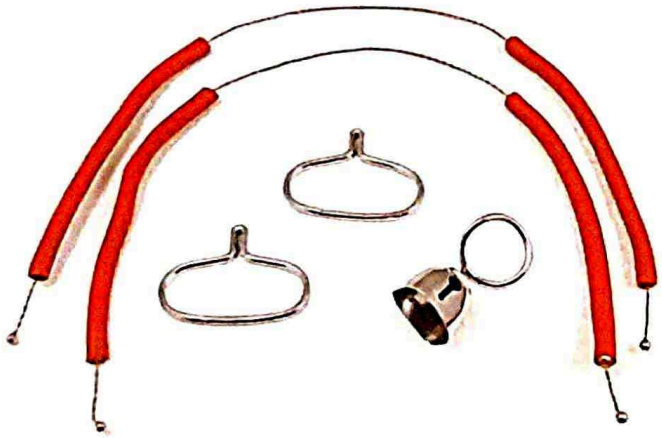


**Fig. 47.51:** Blond-Heidler decapitation saw

### Indications

The saw is used for decapitation (see Chapter 42[iv] for details).

## WILLET'S SCALP TRACTION FORCEPS (Fig. 47.52)

Willet's scalp traction forceps has T-shaped ends of the blades with oblique serrations. One of the blades has two tiny teeth lying side by side which fit in two holes on the other blade to achieve a secure grip on foetal scalp which is made further stronger by a ratchet lock on the handles.

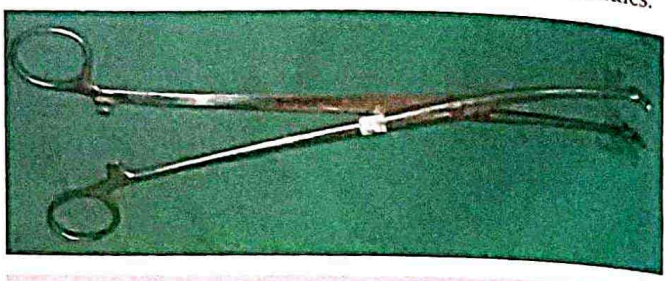


**Fig. 47.52:** Willet's scalp traction forceps

### Indications

1. Willet's scalp traction forceps was used in the past to control bleeding due to type 1 or 2 placenta previa. After rupturing the membranes, the foetal scalp was grasped by the forceps and a continuous traction was applied compressing the placental site and controlling bleeding. It is not used any more.
2. To give traction on the foetal head after craniotomy, to hasten delivery
3. In olden days, it was used to give scalp traction to deliver foetal head during cesarean section.
4. In olden days, it was used to give scalp traction to prevent recurrence of cord prolapse after replacement of the prolapsed cord above the level of the foetal head.

### Complications

1. Laceration of foetal scalp, avulsion of the scalp
2. Haemorrhage from foetal scalp injury.
3. Foetal scalp infection
4. Accidental injury to maternal genital tract.

**Current status:** Not used in modern obstetrics.

## GIANT VOLSELLUM (Fig. 47.53)

Giant volsellum is a large and stout instrument with large teeth for better and firm grip.

### Uses

It is used during destructive operations to extract the foetal parts out due to its very strong grip and traction power. It is especially useful in evisceration operation but can be used in other destructive operations also (see Chapter 42[iv] for details).

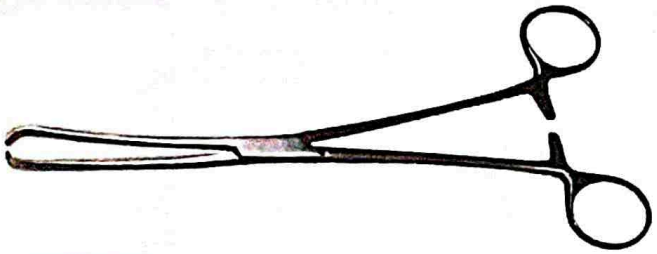


Fig. 47.53: Giant volsellum

## DUBOIS' EMBRYOTOMY SCISSORS (Fig. 47.54)

Dubois' embryotomy scissors is 27 cm long, curved on flat and rather stout. It is sterilized by immersion in lysol or glutaraldehyde solution.

### Indications

1. Evisceration
2. *Decapitation*. To cut soft tissues of foetal neck after dislocation of atlantoaxial joint with a Braun's hook.
3. Spondylotomy
4. Cleidotomy

The students should know the indications of evisceration, cleidotomy. They should be aware of the dangers and complications of destructive operations including post operative care (see Chapter 42 [iv] for details).

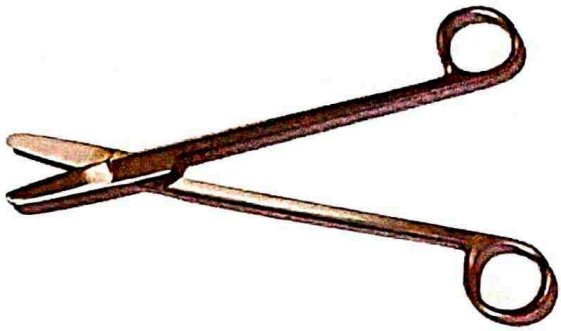


Fig. 47.54: Dubois' embryotomy scissors

# (ii) OBSTETRIC SPECIMENS

Most students are asked about a specimen during their obstetric viva in practical examination. As there are only few obstetric specimens, students can score good marks in this part by preparing them thoroughly. The students need to identify the specimen and then describe it. Further questions can be asked related to the incidence of the problem, symptoms and signs in patients due to it, prevention, diagnosis and management of the condition.

## Anencephaly specimen

**Description of specimen** (Fig. 47.55)

It is specimen of an anencephalic fetus. There is absence of the major portion of the brain, skull and scalp.

This condition is incompatible with life. Most fetuses are either stillborn or die soon after birth due to abnormal development of brain. Eyes are protruding due to absence of cranial vault 'Frog Eye Sign' or Mickey Mouse Sign. Facial features are ugly. Neck is very short.



**Fig. 47.55:** Specimen of an anencephalic baby with absence of major portion of the brain, skull and scalp.

Students can be asked about its etiology, associated anomalies, diagnosis during ante-natal period, incidence, recurrence rate, obstetric complications management and prevention in future by periconceptional folate supplementation (400 µg daily in low-risk cases and 4–5 mg daily in high-risk cases) (see Chapter 21 for details).

## Specimen of hydrocephalic baby (Fig. 47.56)

**Identification**

Hydrocephalic baby

**Description**

It is a fully grown hydrocephalic baby showing massive enlargement of head. The fontanelles and suture are very wide. It is an abnormal accumulation of cerebrospinal fluid in the ventricles causing enlargement of the head. Sutures are also wide. The presence of a hole in head shows that aspiration of cerebrospinal fluid was done prior to delivery to reduce the foetal head diameter for easy delivery. Students can be asked about its incidence, recurrence, etiology, diagnosis, prognosis and management during labour (see Chapter 21 for details).



**Fig. 47.56:** Specimen of a fully grown hydrocephalic baby with massive enlargement of the head with wide sutures and fontanelles.
Kind courtesy: Department of Gynecology, MAMC, Delhi.

## Specimen of spina bifida (Fig. 47.57)

Spina bifida is a neural tube defect and the most common malformation of the spinal cord with failure of dorsal fusion associated with a failure of full development of one or more vertebrae. The most common site is the lumbosacral area, but it may occur anywhere. There may be herniation of the neural elements into the sac. Spina bifida is classified into:

1. **Spina bifida occulta.** The defect is not visible externally. Its presence may be indicated by a hairy patch.
2. **Meningocele.** Herniation of only the dura into the sac.



**Fig. 47.57:** Specimen of a preterm baby showing lumbosacral spina bifida with meningomyelocele with defect in the vertebrae with herniation of dural sac containing spinal cord, meninges and nerve roots.

3. **Meningomyelocele.** Nerve roots, spinal cord and meninges are incorporated in the sac (Fig. 47.57).

The students may be asked about the prognosis and management of different types of spina bifida. It can be prevented by periconceptional folic acid therapy as discussed under anencephaly (see Chapter 21 for details).

## EXOMPHALOS (Fig. 47.58)

Exomphalos is the result of persistence of the physiological hernia of the midgut loop until after birth. Sometimes, large sac ruptures during birth or within a few hours after birth if not treated surgically. An unruptured sac is very thin, translucent and jelly-like. **Exomphalos minor** is small and has the umbilical cord attached to its summit. It may contain a loop of small intestine or Meckel's diverticulum. Usually it does not rupture and does not cause foetal death. **Exomphalos major** is much bigger and has the umbilical cord attached to the inferior aspect of the sac rather than to its summit (Fig. 47.58). It is called complete when the whole of the midgut loop occupies the hernia sac which includes lower part of the duodenum, jejunum, ileum, cecum, appendix, ascending colon and two-thirds of the transverse colon. A portion of the liver is present in the sac in nearly all the cases. The exomphalos is called **partial** when only the cecum and the lower ileum are found in the sac. Exomphalos differs from a true umbilical hernia in that the sac is not covered by normal skin. **It differs from gastroschisis in that the abdominal wall defect is lateral in the latter.**

Students may be asked about its prognosis and management and associated anomalies (see chapters 21 and 28 for details). They should be able to differentiate between gastroschisis and exomphalos.



**Fig. 47.58:** Specimen of an aborted fetus with exomphalos major with a large abdominal wall defect with herniation of major portions of the gut.

## Specimen of hydatidiform mole (Fig. 47.59)

*Hydatidiform mole is a commonly asked specimen in obstetric practical examination .*



**Fig. 47.59:** Specimen of a complete (total) hydatidiform mole showing grape-like vesicles with no evidence of any foetal tissue.

### Identification and Description

It is a specimen of complete hydatidiform mole with vesicles and no demonstration of foetal tissue.

The students can be asked about pathology, symptoms, signs, diagnosis, management, complications of evacuation, follow-up, prognosis and recurrence rate (see Chapter 13 for details).

There can be a specimen of partial hydatidiform mole in which some parts of foetal tissues are seen along with hydatidiform mole. The discussion is almost same except that the chance of choriocarcinoma is much less (see Chapter 13 for details).

## Specimen of rupture uterus (Fig. 47.60)

### Identification

Lateral uterine rupture of lower segment, cervix and partial involvement of upper segment.

### Description

It is a specimen of rupture uterus removed at laparotomy. Uterus is enlarged with complete lateral uterine rupture in the lower segment involving cervix and extending into upper segment. The uterus has been removed because it was not amenable to repair.

Students may be asked about etiology, symptomatology, signs, diagnosis, differential diagnosis and management of rupture uterus (see Chapter 26 for details).

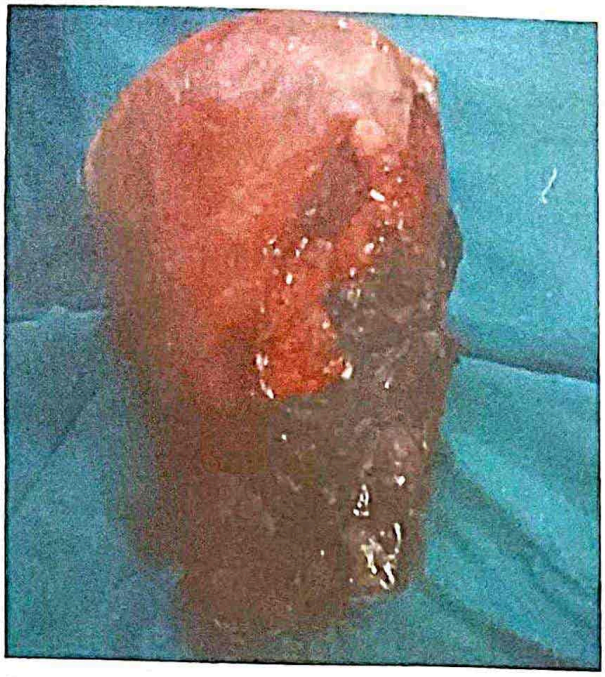


**Fig. 47.60:** Specimen of rupture uterus with complete lateral uterine rupture in the lower segment involving cervix and also extending into the upper segment.
Kind courtesy: Prof. Smiti Nanda, PGIMS, Rohtak

## Specimen of placenta of twin pregnancy

(Fig. 47.61)

There is a single placenta with two cords and two separate amniotic sacs. It is from diamniotic dichorionic pregnancy with two separate amniotic sacs and four membranes (two chorions, two amnions). Students may be asked about monozygotic, dizygotic twin pregnancies, their differences, incidence, predisposing factors, differential diagnosis, complications and management of multiple pregnancies (see Chapter 33 for details).



**Fig. 47.61:** Specimen of a placenta of a twin pregnancy showing a single placenta with two cords and two separate amniotic sacs from a diamniotic dichorionic pregnancy.

## Specimen of fetus papyraceus (Fig. 47.62)

With the death of a fetus and formation of fetus papyraceus, the corresponding placenta shows atrophy and may be identified as fibrotic tissue which may show calcification. It can complicate a twin pregnancy with one normal fetus and other fetus papyraceus. The students may be asked about management of twin pregnancies with one fetus papyraceus and other normally growing fetus. In such a case, conservative management is performed with careful and regular follow-up of patients in antenatal clinic. Usually the healthy twins grow well and is delivered at term (see Chapter 33 for details).



**Fig. 47.62:** Specimen of a fetus papyraceus with compressed fetus

## Specimen of tubal ectopic gestation

(Fig. 47.63)

This is a specimen of total salpingectomy removed either on laparotomy or laparoscopy. The fallopian tube is enlarged and congested. Its external surface is ruptured. The edges of the rent in the tubal wall are irregular, shaggy and a blood clot is adherent at that site. Cut surface of the tube shows a blood clot. Students may be asked about etiology, symptomatology, diagnosis and management (including medical management, minimal invasive treatment and laparotomy) of ectopic pregnancy (see Chapter 13 for details). The students should also know of other types of ectopic pregnancies like ovarian and abdominal pregnancies.

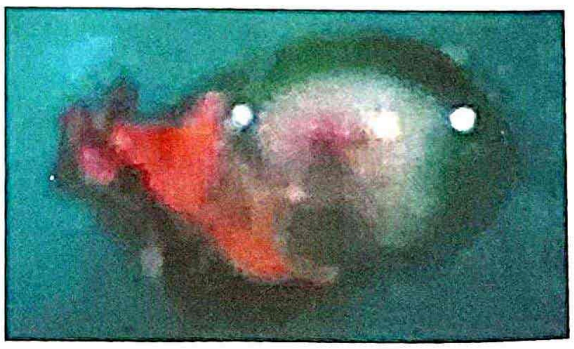


**Fig. 47.63:** Specimen of salpingectomy with ruptured ectopic pregnancy showing enlarged, congested and ruptured tube with irregular, shaggy edges and blood clots.

## Specimen of circumvallate placenta (Fig. 47.64)

Circumvallate placenta is characterized by a thick, round, white, opaque ring 1 to 3 cm in diameter around the periphery of the placenta on the foetal surface. It is seen more commonly in multiple pregnancies, especially triplet and higher order pregnancies.

The significance of circumvallate placenta is that it can cause increased chances of abortion, antepartum haemorrhage, preterm labour, foetal growth restriction and retained placenta and membranes (see Chapter 24 for details).



**Fig. 47.64:** Specimen of a circumvallate placenta showing a thick, round, white opaque ring around the periphery of the placenta on the foetal surface.

## Battledore placenta (Fig. 47.65 A)

The point of umbilical cord insertion into the placenta is usually eccentric, being a little on one side of the center of the placenta. When it is at the edge of the placenta, it is called battledore placenta. It is associated with increased incidence of cord presentation and cord prolapse (see Chapter 24 for details).

## Velamentous insertion of cord (Fig. 47.65 B)

In this condition, which is often associated with Battledore placenta, the branching of vessels in the umbilical cord occurs in the umbilical cord itself before its attachment to the placenta. It is called **vasa previa** if it is in lower uterine segment. Its importance is that it can cause antepartum haemorrhage which can be of foetal origin (identified by tests based on resistance of foetal blood to alkali denaturation) (see Chapter 24 for details). There can be foetal exsanguination and foetal death if not treated on time.

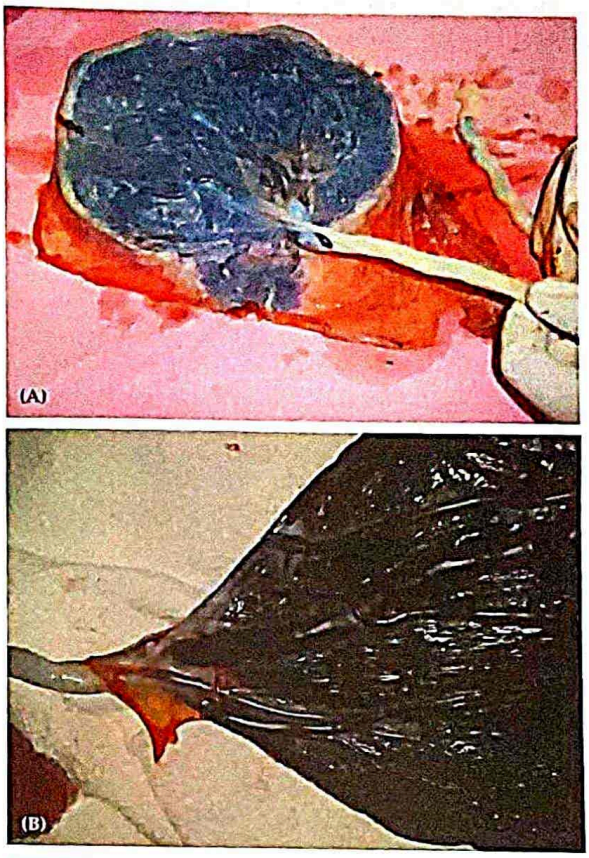
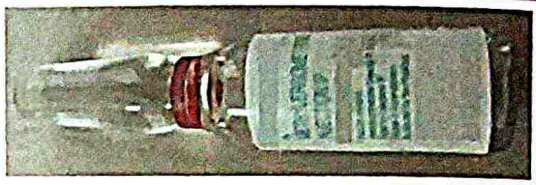
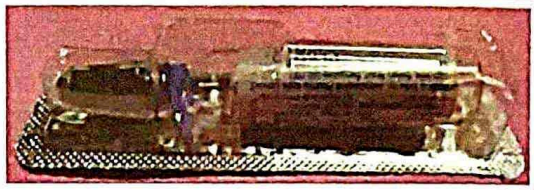
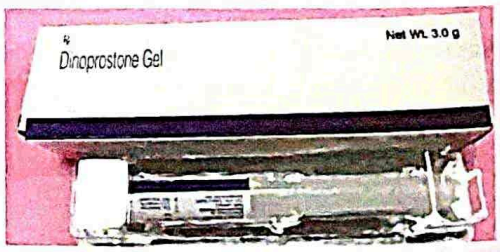
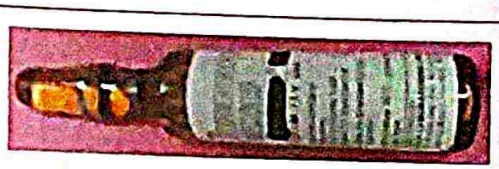
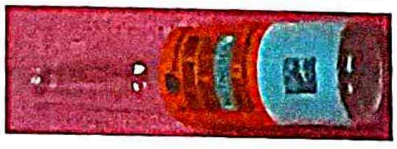



**Fig. 47.65: (A)** Specimen of a battledore placenta with umbilical cord attached to the margin of placenta; **(B)** Specimen of velamentous insertion of umbilical cord with battledore attachment of placenta. Note that the branching of vessels of umbilical cord is occuring in the cord itself before its attachment to the placenta.

## (iii) OBSTETRIC DRUGS AND SPOTS

| | | |
|---|---|---|
| 1. | **Oxytocin injection.** Used for induction and augmentation of labour, cesarean delivery, active management of third stage of labour (10 units IM) and management of postpartum haemorrhage. It is usually given as IV in drip in Ringer lactate or normal saline. It is also used for oxytocin challenge test, first and second trimester abortions and evacuation of hydatidiform mole (see chapters 13, 14, 18 and 25 for details of dosage, drip rate, side effects, etc.). |  Fig. 47.66: Oxytocin (5 units) |
| 2. | **Methylergometrine (Methergin) injection** used for treatment of postpartum haemorrhage as 0.2 mg IV injection. Also used to contract uterus after evacuation in first and second trimester abortions and hydatidiform mole. For active management of third stage of labour, oxytocin is now used. It is used as alternative to oxytocin when oxytocin is not available (see Chapters 14, 16 and 25 for details). |  Fig. 47.67: Methylergometrine (0.2 mg) |
| 3. | **Injection Carboprost 250 µg (Prostaglandin F2α)**<br>Used for second trimester termination of pregnancy or missed abortion as 250 µg IM 3 hourly for up to maximum 10 doses. Also used in prevention and treatment of postpartum haemorrhage (see chapters 13, 14 and 25 for details of dosage, side effects, contraindications, cost, etc.). |  Fig. 47.68: $PGF_2\alpha$ (carboprost) (250 µg) (₹ 250–300) |
| 4. | **Misoprostol tablets ($PGE_1$) tablets**<br>Used for induction of medical abortion (400–800 µg) with mifepristone tablet (up to 9 weeks of pregnancy), second trimester termination of pregnancy (off label) induction of labour, (25 µg), prevention (600 µg) and management of postpartum haemorrhage (800 µg) (see Chapters 13, 14 and 25 for details of indications, contraindications, dosage, side effects and cost). |   Fig. 47.69: Misoprostol tablets (200 µg) (₹ 8) |
| 5. | **Dinoprostone ($PGE_2$ gel)**<br>Question: Uses in obstetrics, dosage and side effects<br>Answer: Used mainly for induction of labour especially in cervical priming with poor Bishop's score (< 6) as 0.5 mg cervical or high vaginal gel to be repeated in 6 hours time. Also used before medical termination of pregnancy (see Chapters 13, 14 and 18 for details). |   Fig. 47.70: Dinoprostone gel (0.5 mg) (₹ 250–300) |
| 6. | **Magnesium sulphate injection**<br>Magnesium sulphate injection is used for prevention and treatment of eclampsia as IV injection of 4 g, IM injection of 10 g (5 g in each buttock) (total 14 g) stat follwed by 5 g IM every 4 hourly (see Chapters 14 and 32 for details). Before giving next injection patellar reflex should be elicted, urine output should be > 25 mL/hour and respiratory rate should be > 16/minute. It is also used for preterm labour. Its antidote is 1 g 10% IV calcium gluconate injection (see Chapters 14, 32 and 34 for details). |  Fig. 47.71: Magnesium sulphate injection (20%, 50% solution) |

| | | |
|---|---|---|
| 7. | **Phenytoin injection.** Used for epilepsy and severe pre-eclampsia (see Chapters 32 and 35 for details). It is no longer used for severe pre-eclampsia or eclampsia (magnesium sulphate is now used). |  Fig. 47.72: Phenytoin injection |
| 8. | **Ritodrine injection.** Uses; Preterm labour (see Chapter 34 for details), to relax uterus for external cephalic version and before cerclage operation (see Chapters 13 and 42 for details) |  Fig. 47.73: Ritodrine injection (10 mg) |
| 9. | **Isoxsuprine injection.** Used for preterm labour (see Chapter 34 for details) and to relax uterus for external cephalic version and before cerclage operation. |  Fig. 47.74: Isoxsuprine injection (10 mg) |
| 10. | **Labetalol injection.** Used for severe pre-eclampsia, eclampsia and severe hypertension (see Chapters 14 and 32 for details of dosage) IV 10 mg then 20 mg after 10 minutes going up to 40 mg and maximum 50 mg for control of hypertension. |  Fig. 47.75: Labetalol injection (5 mg/mL) |
| 11. | **Heparin injection.** Used for deep vein thrombosis and pulmonary embolism (see Chapter 30 for details).<br>Also used for antiphospholipid antibody syndrome (see Chapters 13, 35 and 40 for details) in dose of 5000 units IV or S.C. twice daily up to 34 weeks of pregnancy.<br>**Low molecular weight Heparin (Enoxaparin)** can also be used for same indications as heparin in dosage of 1 mg/kg body weight as single dose but is expensive. |     Fig. 47.76: (A) Heparin injection (5000 units); (B) Low molecular weight heparin injection (40 mg) |
| 12A. | **Betamethasone injection.** Used for pulmonary maturity in threatened preterm labour (12 mg IM every 24 hours for 2 doses) (see Chapter 34 for details). Dexamethasone injections can also be used (6 mg I/M 6 hourly for 4 doses). |  Fig. 47.77A: Betamethasone injection (12 mg) |
| 12B. | **Drotaverine injection.** Used as analgesic for pain relief in labour (see Chapter 19 for details). Also used for pain relief in post operative period and for colicky abdominal pain like renal and ureteric colic. |  Fig. 47.77B: Drotaverine injection 20 mg (2 mL) |

13. Students should be able to identify the cardiotocogram and various abnormal patterns (e.g. decelerations), their interpretation and management (e.g. foetal blood sampling or immediate delivery) (see Chapter 17 for details).

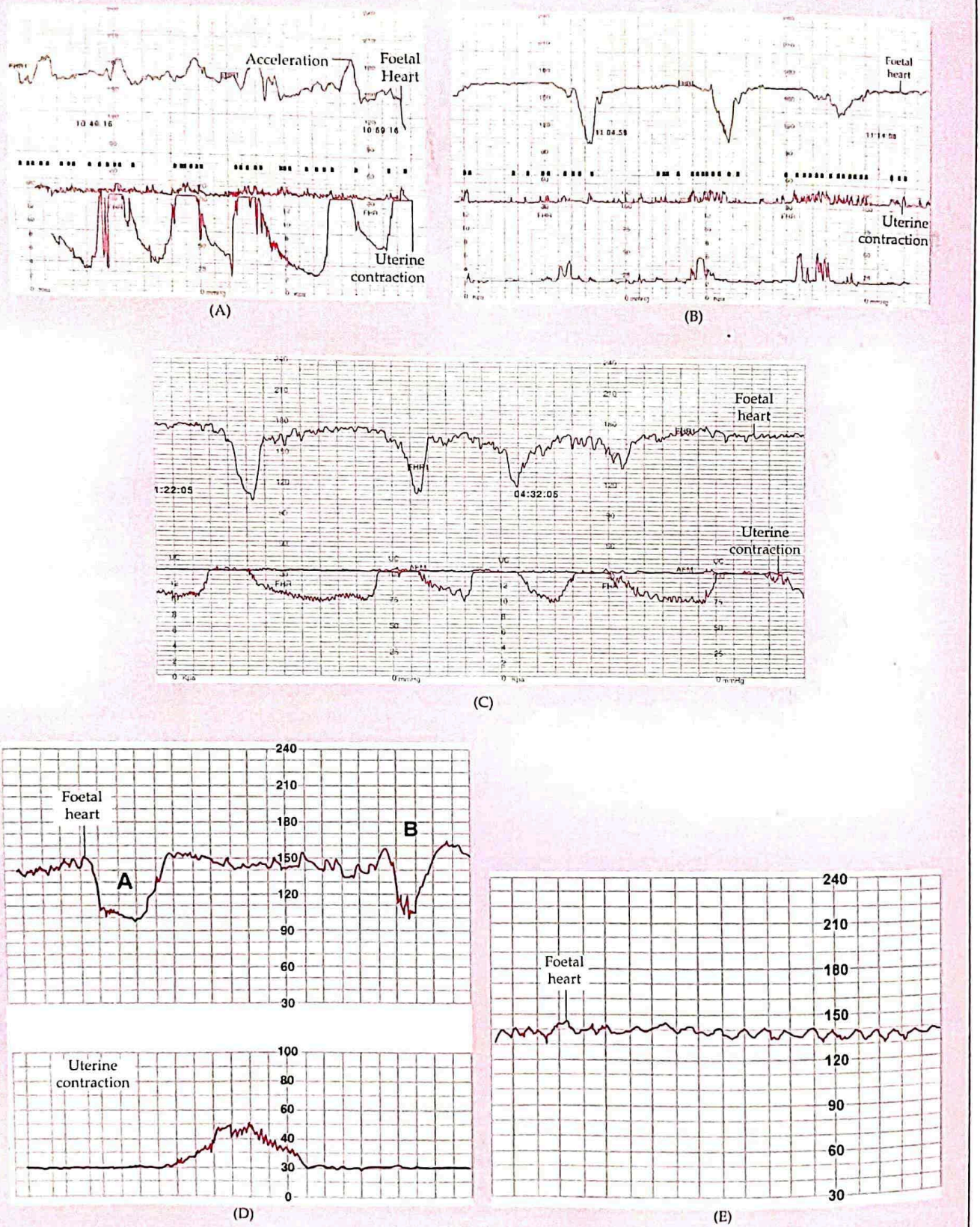



**Fig. 47.78:** Cardiotocogram (CTG strips) **(A)** Normal CTG; **(B)** CTG showing early decelerations; **(C)** CTG showing late decelerations; **(D)** CTG showing variable decelerations; **(E)** CTG showing sinusoidal pattern

14. Students should know the indications of non-stress test, its interpretation, false positives and what to do if non-stress test is non-reactive (eg. to perform biophysical profile) (see Chapter 15 for details).

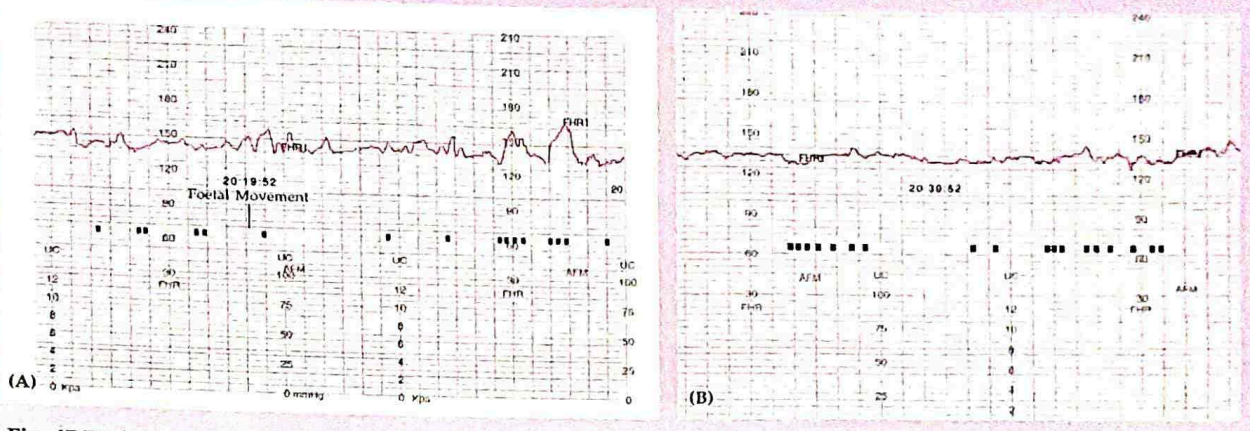



**Fig. 47.79: (A)** Non-stress test strip with reactive (normal) trace; **(B)** Non-stress test strip with non-reactive (abnormal trace)

15. Students should know the etiology, vanishing twin, complications, chorionicity, antenatal and intrapartum management of multiple pregnancy (see Chapter 33 for details).



**Fig. 47.80:** Ultrasound showing three gestational sacs in early triplet pregnancy.

16. Students can be asked about etiology, presentation, diagnosis, complications, management and follow-up of molar pregnancy. Diagnosis is by ultrasound and serum β hCG levels. Management is evacuation using suction and evacuation with Karman's cannula. They are followed up by urine and serum β hCG levels (see Chapter 13 for details).

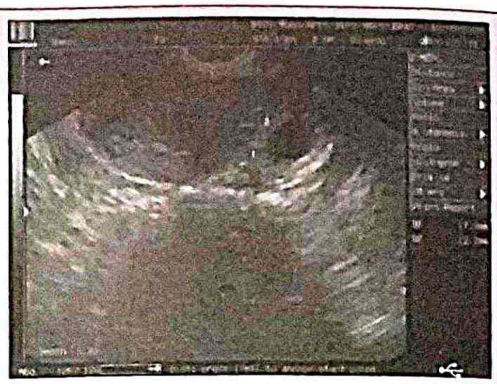
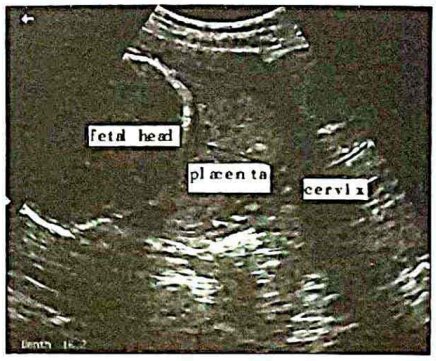
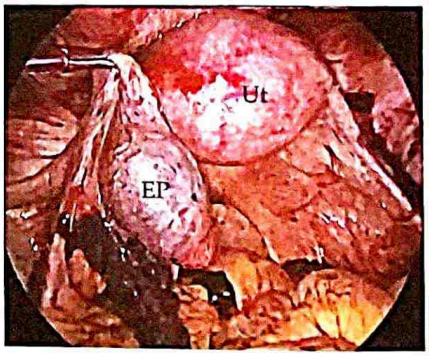
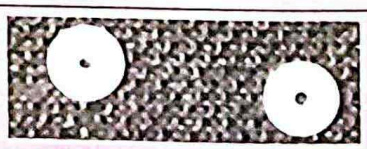


**Fig. 47.81:** Ultrasound showing snowstorm appearance in a case of hydatidiform mole

17. Students should know about various types of abortions and their management (see Chapter 13 for details). Blighted ovum and missed abortion can be managed conservatively, medically (using misoprostol) or surgically (evacuation) (see Chapter 13 for details).



**Fig. 47.82:** Ultrasound picture of blighted ovum

| | | |
|---|---|---|
| 18. | Students may be asked about etiology, presentation, diagnosis, complications and management (including expectant, medical and surgical) of Ectopic pregnancy (see Chapter 13 for details). |  **Fig. 47.83:** Ultrasound showing an empty uterus and an adnexal mass in a case of ectopic pregnancy. |
| 19. | Students may be asked about etiology, clinical features, diagnosis, differential diagnosis and management of placenta previa (see Chapter 31 for details). |   **Fig. 47.84:** Ultrasound showing major degree placenta previa covering the os. |
| 20. | Students should know about etiology, reasons of increased incidence, clinical features, complications and management (medical, surgical including laparoscopic) of ectopic pregnancy (see Chapter 13 for details). |   **Fig. 47.85A:** Laparoscopic picture of unruptured ectopic pregnancy |
| 21 | **Falope rings (Yoon rings).** They are used for female tubal sterilization on laparoscopy. Ring is put on isthmic part of fallopian tube (see Chapter 43 for details). |  **Fig. 47.85B:** Actual photograph of Falope rings in laparoscopy |

Sometimes, there may be spots of X-rays (e.g. malpresentation, intrauterine death, pelvimetry, misplaced copper T) or other ultrasounds (e.g. misplaced copper T, malpresentation, incompetent cervix, uterine anomalies, foetal anomalies, various types of abortion, retroplacental haematoma or other obstetric problems), magnetic resonance imaging pictures (e.g. placenta previa, pelvimetry, foetal anomalies). The pictures have been shown in corresponding chapters of the book and the questions shall be from those topics. The spots are usually used to open a topic and then further discussion on that subject is continued in the examination.

# (iv) LABORATORY TESTS IN OBSTETRICS AND THEIR INTERPRETATION

Students need to be aware of common laboratory tests required for the day-to-day management of obstetric patients as they help in better management of the patients and these are often asked in examination.

## SI UNITS

SI units (International System of Units) are the internationally agreed upon basic units adopted in 1960. Students should be conversant with them as administration of medicines and the reports of chemical analysis are now expressed in SI units. The meter (m) is the basic SI unit of length. Kilogram (kg) is the basic SI unit of mass. Mole is the SI unit of amount of a substance with a mass equal to its molecular weight in grams. One millimole is one-thousandth of a mole (m mol/L), while one micromole is one-millionth of a mole (μ mol/L).

Medicines are administered in milligrams, grams and micrograms. Liquid medicinal preparations are administered in milliliters and liters.

1 mg = 1000 μg,

1 mg/mL = 0.1 μg/100 mL = 1 mg/L

The reports of chemical analysis are now made in millimoles per liter (m mol/L) rather than in milligrams per 100 milliliters (mg/100 mL).

$$\text{Conversion – (m mol/L)} = \frac{\text{mg/100 ml} \times 10}{\text{Mol. weight}}$$

## CELSIUS TEMPERATURE SCALE

**Celsius temperature scale** (°C) is SI derived unit named after the inventor Celsius.

**Conversion of Fahrenheit to Celsius**—Subtract 32, multiply by 5 and then divide by 9, e.g.

$$100°F = \frac{(100 - 32) \times 5}{9} = 37.8\ °C$$

**Conversion of Celsius to Fahrenheit**—Multiply by 9, divide by 5 and then add 32, e.g.

$$37°C = \frac{37 \times 9}{5} + 32 = 98.6\ °F$$

## URINE EXAMINATION

Urine examination is of crucial importance in managing pregnancies complicated by diabetes mellitus, hypertension, renal disease and urinary tract infection. The various tests performed on urine are as follows:

1. **Physical examination.** Colour, volume (normal 750–2000 mL/day), specific gravity (normal 1.001–1.035), pH (5–8) and odour are checked. Oliguria (< 500 mL) can occur in hypertensive disorders, renal disease, obstetric shock and renal failure. Low pH is observed in diabetic ketoacidosis, high pH in vegetarians, metabolic alkalosis, urinary tract infection with urea splitting organisms, while foul smell is seen in bacterial infection.
2. **Biochemical examination.** Testing for proteins, sugar, ketone bodies as described later in details.
3. **Microscopic examination** for white blood cells, pus cells (normal < 5 pus cells/HPF), bacteria (infection), casts (renal disease), crystals (stones) and epithelial cells.
4. **Bacteriological examination.** If pus cells with or without bacteria are present on microscopic examination or there are clinical symptoms or signs of urinary tract infection, urine is sent for culture and sensitivity (see Chapter 35[vi] for details). Collection of midstream urine in the culture bottle at laboratory is ideal so that urine can be put to culture immediately.
5. **Immunological tests.** Urine is tested for pregnancy tests which is based on hCG in urine. These days urine pregnancy test kits are available in market which detect hCG in urine and are useful in diagnosis of pregnancy and gestational trophoblastic disease (see Chapters 8 and 13 for details).
6. **Hormonal tests.** Urine can also be tested for various hormonal levels like estriol, progesterone, hCG and other hormones which guide us about the normalcy of pregnancy (see Chapters 7 and 8 for details).

### Specimen collection

For most of the routine investigations, first morning urine is collected in a dry, clean container or vial. For bacteriological studies, 'clean catch' midstream urine is collected in a wide mouth sterile culture bottle. In case of sick patients, who cannot give urine sample, a catheter sample can be taken for testing. For protein testing, random sample may be used while for glucose testing, 2 hour postprandial (post meal) sample is preferred.

### Tests for proteins

I. **Qualitative tests.** The presence of proteins in urine is tested and are of value in hypertensive disorders during pregnancy, renal diseases and in urinary tract infections. They are as follows:

1. **Heat acetic acid test.** It is shown in Table 47.1.
2. **Dipstick test** (Fig. 47.86). These days easy-to-use reagent strips are available in the market for testing proteins in the urine. The strip is made of stiff absorbent cellulose material with the testing end being impregnated with the indicator 'tetra-brom-phenol-blue' which




**Fig. 47.86:** Actual photograph of Uristix for estimation of urine albumin and sugar

**Table 47.1: Heat Acetic Acid Test for Urine Proteins**

| Methods | Observation | Interpretation |
|---|---|---|
| 1. Fill 3/4th of a test tube with urine. Hold obliquely and heat the top half using Bunsen's burner. | Urine becomes turbid (cloudy precipitate). | Presence of proteins or phosphate |
| 2. Acidify the urine by adding few drops of 5% acetic acid. | Cloudiness disappears | Presence of phosphate |
| | Cloudiness (turbidity) persists or increases | Presence of proteins |
| | (i) No turbidity | Absent proteins in urine |
| | (ii) Barely visible turbidity | Trace (5–10 mg proteins/100 mL of urine) or 0.05 to 0.1 g/L |
| | (iii) Distinct turbidity | + (10–30 mg proteins/100 mL of urine) |
| | (iv) Moderate turbidity | ++ (40–100 mg/100 mL of urine) |
| | (v) Heavy turbidity | +++ (200–500 mg/100 mL of urine) |
| | (vi) Heavy flocculent precipitate | ++++ (500–1000 mg/100 mL of urine) |

is buffered to pH of 3. The Albustix detects albumin while multistix also tests for other urinary constituents like urinary pH, ketone bodies, specific gravity and glucose. Uristix tests both proteins and glucose with separate colour coding for them (Fig. 47.86).

**Method.** The test end of the reagent strip is dipped in the urine and taken out immediately. Any colour change is compared with the colour chart available with the strip. Turning of test end to green or blue represents a positive test for proteins. The interpretation from trace to ++++ is as follows:

**Table 47.2: Quantitative Estimation of Urinary Proteins by Dipstick**

| | |
|---|---|
| Trace | 10 mg/dL |
| + | 30 mg/dL |
| ++ (2+) | 100 mg/dL |
| +++ (3+) | 300 mg/dL |
| ++++ (4+) | 2 g/dL |

**II. Quantitative Urinary Protein Test.** It is usually performed in pre-eclampsia, other hypertensive disorders and renal diseases in pregnancy.

**Method.** The woman collects all urine sample starting from the first morning sample in bottles for total 24 hours. Testing is done in biochemistry laboratory or laboratory medicine for quantitative estimation of urinary proteins in 24 hours by Biuret method using Esbach's albuminometer.

## Tests for reducing sugars

Normal urine may contain 1–1.5 g/24 hours of reducing sugars and 50–300 mg of glucose/24 hours.

I. Qualitative Tests

The tests are useful in diabetic pregnancies.

1. **Benedict's test.** It tests reducing sugars in urine. It works on the principle that cupric ion is reduced to cuprous oxide by glucose (Table 47.3).

   False positive test can come during pregnancy due to other sugars or lowered renal threshold for glucose and blood sugar estimation is indicated.

**Table 47.3: Benedict's test to Detect Urine Sugars**

| Method | Observation | Interpretation |
|---|---|---|
| Take 5 mL of Benedict's qualitative reagent (containing copper sulphate, sodium citrate and sodium carbonate). Add 8 drops (0.5 mL) of urine to it. Boil the mixture on a Bunsen's burner for about two minutes and allow to cool. | Colour change indicates presence of reducing sugars as follows: | |
| | (i) Green colour | Trace 0.5 g/100 mL |
| | (ii) Green precipitate | + 0.5–1 g/100 mL |
| | (iii) Yellow | ++ 1–1.5 g/100 mL |
| | (iv) Orange | +++ 1.5–2 g/100 mL |
| | (v) Brick red | ++++ > 2 g/100 mL |

2. **Dipstick Tests** (Fig. 47.87). The easy-to-use reagent strips available are made of absorbent cellulose with the test end being impregnated with buffered mixture of glucose oxidase and peroxidase. They provide quick, specific, qualitative and semi-quantitative colour tests for detecting glucose in urine and are specific for glucose.

   **Method.** The test end of the strip is dipped in urine and taken out immediately and the test is read after one minute. Change in



**Fig. 47.87:** Actual photograph of Ketostix for estimation of urine ketone and sugar

colour of the test end indicates presence of glucose. The colour change is compared with colour chart on the bottle for quantitative estimation of urine sugar.

| Table 47.4: Quantitative Estimation of Urinary Glucose by Dipstick | |
|---|---|
| Trace | 0.1 g/dL |
| + | 0.25 g/dL |
| ++ (2+) | 0.5 g/dL |
| +++ (3+) | 1 g/dL |
| ++++ (4+) | 2 g or more/dL |

II. **Quantitative estimation of urinary glucose.** Rough estimation is done by uristix as described earlier. For accurate estimation, urine sample is sent to laboratory for testing by glucose oxidase method.

## Urine tests for ketone bodies

In a normal person, blood ketone bodies levels are about 1 mg/dL with less than 1 mg/24 hours being excreted in urine. The various ketone bodies are β-hydroxybutyrate, acetoacetate and acetone. Ketonemia and hence ketonuria can occur during pregnancy even in normal conditions due to accelerated starvation. It can also occur with intake of high fat, low carbohydrate diet, starvation, diabetic ketoacidosis and in pre-eclampsia.

**Rothera's Nitroprusside Test.** It is shown in Table 47.5

# BLOOD TESTS

## Blood collection

Blood sample is taken under all aseptic conditions wearing sterile gloves using disposable syringe and needle.

## Hemoglobin Estimation

The capillary blood is tested by Sahli's hemoglobinometer (Table 47.6).

## Blood sugar estimation

Blood sugar estimation is important for screening for gestational diabetes mellitus (GDM) during pregnancy and for management of GDM and type I and type II diabetes mellitus during pregnancy (see Chapter 35[iii] for details).

**Dextrostix method.** These days consumer-friendly reagent strips are often used being rapid, convenient and semi-quantitative for estimating blood glucose levels. The strip is impregnated with chromogen system which converts into oxidized chromogen by the action of enzyme glucose oxidase and is read with the colour chart.

**Glucometer method.** Now-a-days blood glucometer as shown in Fig. 47.88 is used for glucose estimation as it is a sensitive and accurate method.

**Method.** The reagent strip is inserted into the glucometer. A drop of blood from the finger tip or venous sample is applied to the test end of the reagent strip. The reading of blood sugar comes automatically on the glucometer. The method gives blood sugar in mg/dL of whole blood.




**Fig. 47.88:** Actual photograph of glucometer and reagent strip

The test can be done by the patient herself or by her husband after teaching them. However, during pregnancy, tests should sometimes be cross-checked by the doctor.

| Table 47.5: Tests for Urinary Ketone Bodies | | |
|---|---|---|
| **Method** | **Observation** | **Interpretation** |
| 1. **Rothera's Test.** Take 5 mL urine in a test tube. Saturate it with ammonium sulphate. Add 0.5 mL of freshly prepared sodium nitroprusside solution. Add liquor ammonia along the sides of the test tube. | A purple ring appears at the junction of the liquids. | Presence of ketone bodies (acetone and acetoacetate) |
| 2. **Gerhardt's ferric chloride test.** Add 10% ferric chloride solution drop-by-drop to 5 mL urine in a test tube. | Purplish colour | Presence of acetoacetate |
| 3. Reagent Strip Test<br>(i) **Ketostix (Fig. 47.87).** It is commonly used these days as it is user-friendly. The test end of the strip is impregnated with a buffered mixture of sodium nitroprusside and glycine. The test end of the strip is dipped in fresh urine sample. Compare the colour of the test end with the colour chart on the side of bottle supplied by the manufacturer. Ketostix also detects urine sugar.<br>(ii) **Multistix.** It checks for proteins, sugar, ketone bodies, pH and RBCs in urine. | Change in colour of strip. | Presence of acetone and acetoacetate |

**Table 47.6: Tests for Hemoglobin Estimation**

| Method | Observation | Interpretation |
|---|---|---|
| 1. **Sahli's hemoglobinometer method.** Blood from finger prick or venous sample is drawn in Hb pipette till the mark (20 cu mm or 0.02 mL). Hemoglobinometer tube is filled up to 20 mark (3 g) with N/10 HCl. Blood from the pipette is then blown into the Hb meter tube. Mixture is stirred with the glass rod and kept for 10 minutes. Distilled water is added with a dropper till the colour of the solution matches with the comparator colour provided. Take reading from the upper meniscus. | The principle of the test is that N/10 HCl converts Hb into acid hematin. Brown colour of the acid hematin is compared with the brown colour of the comparator. | Normal Hb is > 11g/dL (11–14g/dL). < 11 g/dL is anaemia. (see Chapter 35 [i] for details) |
| 2. **Cyanomethemoglobin method.** In this method, Hb is oxidized to methemoglobin by potassium ferricyanide whose colour is compared with standard of known Hb value colorimetrically. It is more accurate but expensive and less commonly used. | | |

**Glucose Challenge Test (GCT)**
See Chapter 35(iii) for details.

**Glucose Tolerance Test (GTT)**
GTT is performed if glucose challenge test is positive or for other obstetric indications like hydramnios (see Chapter 35[iii] for details).

## Collection of cord blood sample

Collection of cord blood sample is especially relevant in Rh-negative women but is required in other pregnancies also. It is collected from the placental end of the severed cord. To avoid contamination of the blood with Wharton's jelly, the cord should not be squeezed. The sample can be taken by opening the Kocher's forceps from the placental end of the cord or by syringe and needle (see Chapter 38 for details).

## Collection of vaginal blood for foetal hemoglobin

Collection of vaginal blood for foetal hemoglobin is useful in cases of antepartum haemorrhage with suspicion of vasa previa to differentiate between maternal blood and foetal blood.

### Method

About 2 mL blood is carefully collected from the vagina of women in EDTA vial for alkaline denaturation test **(Apt's test)** which detects foetal blood (see Chapter 31 for details).

# PAP SMEAR

Pregnancy time can be utilized for screening for cervical intraepithelial neoplasia and cervical cancer. Speculum examination is performed. If there is no obvious infection in vagina and no bleeding, surface scrape is taken with Ayre's spatula from ectocervix by rotating the tip of spatula by 360° to collect sample from ectocervix and endocervix. Cytobrush to take sample from endocervix can be used but may cause bleeding. The sample is spread on a glass slide, fixed with 95% alcohol and sent to cytopathology laboratory for staining and reading the results (see Chapter 37[iii] for details). Alternatively liquid pap smear is also taken these days in which the tip of the brush is rotated all around ectocervix and endocervix and then broken into the liquid bottle and sent to the cytopathology laboratory.

# VAGINAL CYTOLOGY

Vaginal cytology used to be very popular few decades back to check hormonal levels, especially to see hypoestrogenic conditions for diagnosis and prognosis of miscarriage and placental insufficiency. However, it is hardly ever used in modern obstetrics due to availability of better methods.

### Method

Using Ayre's spatula, cells are obtained from **the upper third of lateral vaginal wall** and the slide is fixed with alcohol, stained with Papanicoloau stain and the vaginal cells are examined under microscope. During normal pregnancy due to excessive progesterone levels, there is preponderance of **intermediate cells (navicular cells).** In case of high-risk pregnancies like threatened abortion, missed abortion, postmaturity and placental insufficiency, there may be progesterone deficiency shifting the maturation index to right under estrogen effect. Availability of better ultrasound and other foetal monitoring techniques has made it unpopular in current practice.

# (v) DISINFECTION AND STERILIZATION IN OBSTETRICS

### Sterilization

Sterilization is a process by which all microorganisms, including spores, are destroyed and is ideal.

### Autoclaving

Autoclaving is a method of sterilization in which steam is used under pressure for killing microorganisms, including spores. It is done for all linen used in labour room and operation theatre and non-sharp surgical instruments. After washing, cleaning and drying the linen and instruments, sister incharge makes a pack (e.g. delivery pack, episiotomy pack, cesarean pack). It is then sent to the Central Sterile Supply Department (CSSD) of the hospital for autoclaving. There is an indicator which is fixed to the pack of outer surface of surgical drum (signaloc) whose colour is green. After autoclaving, its colour changes to black. Hence, colour of the indicator tells us whether the pack or drum is autoclaved or not. Autoclaving is done by use of steam under pressure at 120 °C for 20–30 minutes under 20 lb (10 kg) pressure (students can remember 120, 20 and 10).

### Disinfection

Disinfection is usually performed on inanimate objects like floors, instruments, etc. Disinfection only kills vegetative bacteria leaving spores. Various methods of disinfection are as follows:

1. **Boiling.** Instruments and syringes can be disinfected by boiling for 30 minutes. It is rarely used these days as disposable syringes and needles are normally used.
2. **Chemical disinfection.** For disinfection of skin (patient's operation site or surgeon's hands), povidine iodine is usually used.
3. **Organic disinfectants.** Like lysol, glutaraldehyde, citraldehyde 2%, cetrimide and formaldehyde solution. Fibreoptics (laparoscope, hysteroscope, cystoscope) and sharp instruments (scissors) are disinfected by putting them in glutaraldehyde (cidex) solution for 10 minutes.

In modern practice, plastic syringes and needles, disposable gloves, catheters, transfusion sets, Ryle's tube and scalpel blades are used which are once-a-use only and then are disposed off. Gauze dressings, gloves, surgical instruments and theatre apparel are usually autoclaved as described earlier.

### Preparation of Operative Site in Patients

The abdomen and vulva can be shaved (though shaving is not necessary these days) and scrubbed with soap and water one hour before operation on the operation table. The skin of the operating site is prepared by using povidine iodine solution (cetrimide and alcohol were used in the past). It is scrubbed on abdomen for at least 3 minutes in concentric circles starting from the area of incision and proceeding outward towards the periphery of the field. Its action usually remains for about one hour.

Vaginal swabbing for examination or delivery is done by 1 : 1000 cetrimide lotion. For vaginal examination, cetrimide solution or dettol can be used as antiseptic and for lubrication.

### Clothing and Scrubbing of Doctors and Nurses

Shoes and clothes should be removed and kept in the changing room from where operation theatre clothes and shoes or chappals are worn. They must wear theatre suit, plastic apron, sterile cap, masks, gown while conducting a delivery or performing a cesarean section. Surgeon and scrub nurse should scrub their hands for 6 minutes with soap and water using brush to clean nails and to scrub up to the arm above the elbows. These days povidine iodine or hibitane scrub is more commonly used which are better disinfectants and cause less dryness of skin.

# (vi) REFERENCE VALUES IN OBSTETRICS

**Table 47.7: Reference Values—Normal Reference Values in Hematology in Women**

| | | Non-pregnant | Value in third trimester of pregnancy |
|---|---|---|---|
| **Investigations** | **Units** | **Value** | |
| Hemoglobin (Hb) | g/dL | 11–16 | 11–14 |
| Packed cell volume (PCV) | % | 35–48 | 32–36 |
| Red Cells | | | |
| RBC count | $10^6/mm^3$ | 4–5 | 3–5 |
| • Diameter | µm | 7–8 | 7–8 |
| • Distribution width | % | 13–15 | 13–15 |
| • Lifespan | days | 120 | 120 |
| • Mean corpuscular volume (MCV) | fl | 80–97 | 75–96 |
| • Mean corpuscular hemoglobin (MCH) | pg | 27–33 | 26–31 |
| • Mean corpuscular hemoglobin concentration (MCHC) | % | 32–36 | 30–35 |
| • HbA | % | 90–97 | 90–97 |
| • $HbA_2$ % | % | 2–3 | 2–3 |
| • HbF % | % | < 2 | < 2 |
| • Red cell mass | mL/kg | 23–29 | 23–29 |
| Total leucocyte count TLC | mL/cmL | 4000–7000 | 10000–15000 |
| Serum transferrin receptor protein | µg/L | 4–8 | 4–8 |
| Red cell protoporphyrin | µg/dL | 30–50 | 30–50 |
| ESR | mm/1st hour | 0–20 (mean 10) | 13–70 (mean 40) |
| Platelet count | lakhs/cu mm | 1.5–4.0 (mean 2.5) | 1.4–4.2 (mean 2.1) |
| Reticulocyte count | % | 0.5–2.0 | 0.5–2.0 |
| Serum iron | µg/dL | 60–175 | 60–75 |
| Total iron binding capacity (TIBC) | µg/dL | 300–350 | 350–400 |
| Transferrin (percentage) saturation | % | 30 | 15 |
| Serum ferritin | µg/L | 15–300 (mean 18) | 12–116 |

**Normal Reference Values of Other Common Blood Chemical Tests**

| **Investigations** | **Units** | **Non-pregnant** | **Third trimester of pregnancy** |
|---|---|---|---|
| S. Alanine aminotransferase ALT/SGPT | U/L | 7–41 | 2–25 |
| S. Amylase | U/L | 20–96 | 15–81 |
| S. Aspartate aminotransferase AST/SGOT | U/L | 12–38 | 4–32 |
| S. Bicarbonate | mmol/L | 22–30 | 20–24 |
| S. Bile acids | µ mol/L | 0.3–4.8 | 0–11.3 |
| S. Bilirubin total | mg/dL | 0.3–1.13 | 0.1–1.1 |
| S. Bilirubin conjugated | mg/dL | 0.1–0.4 | 0–0.1 |
| S. Calcium total | mg/dL | 8.7–10.2 | 8.2–9.7 |
| S. Chloride | meq/L | 102–109 | 97–109 |
| S. Cholesterol | mg/dL | < 200 | 219–349 |
| S. $PCO_2$ | mm Hg | 38–42 | 25–33 |
| S. Creatinine | mg/dL | 0.5–0.9 | 0.4–0.9 |
| 24 hours creatinine clearance | mL/minute | 91–130 | 50–166 |

| | | | | |
|---|---|---|---|---|
| B. Glucose fasting | | mg/dL | 65–100 | 65–100 |
| B. Glucose PP (post prandial 2 hours) | | mg/dL | < 140 | < 140 |
| S. Lactate dehydrogenase | LDH | units/L | 115–221 | 82–524 |
| S. Lipids | total | mg/dL | 400–800 (650) | 500–1200 (1000) |
| S. Lipoprotein – low density | LDL as cholestrol | mg/dL | < 100 | 101–224 |
| S. Lipoprotein – high density | HDL as cholestrol | mg/dL | 40–60 | 48–87 |
| S. $PO_2$ | | mmHg | 90–100 | 92–107 |
| S. Phosphate | alkaline | mg/dL | 33–96 | 38–229 |
| S. Phosphate as phosphorus | | mg/dL | 2.5–4.3 | 2.8–4.6 |
| S. Proteins | total<br>albumin<br>globulin<br>A/G ratio | g/dL<br>g/dL<br>g/dL | 6.7–8.6 (mean 7)<br>4.1–5.3<br>2.0–3.5 (2.7)<br>1.7 : 1–2 : 1 | 5.6–6.7 (6)<br>2.3–4.2<br>2.5–3.8 (3)<br>1 : 1 |
| S. Potassium | | mEq/L | 3.5–5.0 | 3.3–5.1 |
| S. Sodium | | mEq/L | 136–146 | 130–148 |
| B. Urea Nitrogen | | mg/dL | 7–20 (4) | 3–11 (3.1) |
| S. Uric acid | | mg/dL | 2.5–5.6 | 3.1–6.3 |
| B – Blood | | | | |
| S – Serum | | | | |

| Name of tests | Method of blood collection for test | Amount of blood | Normal value | Abnormal value |
|---|---|---|---|---|
| 1. Hemoglobin | EDTA powder (2 mg/mL blood) vial | 2 mL | 11–14 g/dL | < 11 g/dL<br>< 7 g severe anaemia |
| 2. Hemogram (MCV, MCH, MCHC, PCR, etc.) | Same as above | 3 mL | PCV > 38%<br>(see Chapter 35[i] for details) | < 38 |
| 3. Blood group (ABO and Rh) | Plain vial (clotted blood)<br>Add few drops of 3.8% sodium citrate solution | 2 mL | Normal blood group AB, O, AB<br>Rh-positive | Rh-negative |
| 4. STS (VDRI) | Clotted blood in plain vial | 2 mL | Negative | Positive |
| 5. Blood glucose | Oxalate fluoride | 2 mL | Fasting 60–100 mg/dL<br>Postprandial 100–140 mg/dL | > 110 mg/dL<br>> 140 mg/dL |
| 6. Urea, creatinine, non-protein nitrogen (NPN) | Plain vial (clotted blood) | 5 mL | Urea 9–25 mg/dL<br>Creatinine 0.6-1 mg/dL | > 25 mg/dL<br>> 1 mg/dL |
| 7. Serum bilirubin | Plain vial (clotted blood) | 3 mL | < 1 mg/dL | 2 mg/dL and above |
| 8. Plasma proteins | EDTA vial | 3 mL | 4–7 g/dL | < 4 g/dL |
| 9. Blood culture | Glucose broth medium 50 mL | 5 mL | Sterile | Positive for aerobic, anerobic bacteria, typhoid or any other pathogen |
| 10. Coomb's test (indirect or direct) | Plain vial (clotted blood) | 2 mL | Negative | Positive |
| 11. Serum βhCG | Plain vial (clotted blood) | 2 mL | | |

| Name of tests | Method of blood collection for test | Amount of blood | Normal value | Abnormal value |
|---|---|---|---|---|
| **Blood Coagulation studies** | | | | |
| 1. Bleeding time<br>Finger pricking is done and time taken to stop bleeding is checked. Blood is soaked on filter paper every 15 seconds. | Not applicable | Not applicable | **Duke's method**<br>1–6 minutes<br><br>**Ivy's method**<br>1–5 minutes | 6–10 borderline<br>> 10 abnormal |
| 2. Coagulation time<br>(i) Capillary tube method (Wright's tube method) | Blood is collected into a capillary tube from finger puncture. Capillary tube is placed on table for 3 minutes breaking 1 cm of tube every 30 seconds after 3 minutes. The end point is formation of fibrin thread between the broken ends. | 3 mL | 5–10 minutes | > 10 minutes, e.g. DIC |
| (ii) Lee and White method | Plain vial. Time taken by blood collected from vein to clot is taken. | | 4–9 minutes | > 10 minutes, e.g. DIC |
| 3. Clot observation test (Weiner) | Plain dry vial. Venous blood is kept in a dry test tube at 37 °C | 5 mL | Clot forms by 10–12 minutes (6–12 minutes) | If clot fails to form by 30 minutes, e.g. coagulation disorder |
| 4. Clot retraction time | Same as above | Same as above | Clot retracts by 30 minutes | > 30 minutes |
| 5. Prothrombin time (PT) | Sodium citrate 3.8% (0.5 mL) solution is added. | 4.5 mL | 11–15 seconds | > 17 seconds. Prolonged in coagulation failure |
| 6. Thrombin time (TT) | Same as above | Same as above | 10–15 seconds | > 15 seconds |
| 7. Fibrindex or Thrombin test | Plain vial | 5 mL | Formation of clot within 1 minute | > 1 minute |
| 8. Platelet count | EDTA vial | 2 mL | 1.5–4 lac/cu mm or 150–400 × $10^3$/$mm^3$ | < 100,000/cu mm<br>< 70,000 in DIC, HELLP syndrome, ITP, dengue |
| 9. Fibrinogen | EDTA vial | 2 mL | 200–450 mg/dL | < 100 mg is critical. |
| 10. Fibrin degradation products (FDPs) | Special tube supplied with kits | 5 mL | 500 µg/100 mL<br>(5 µg/mL) | Increased in DIC |
| 11. Euglobin clot lysis time (ECLT) | Citrate solution | 4–5 mL | 2–4 hours (> 90 minutes) | Shortened in fibrinolytic syndromes |
| 12. D-dimer (fibrin breakdown products) | EDTA vial | 2 mL | 20 mg/dL | Increased in DIC |
| 13. Partial thromboplastin time (PTT) | | | 40–100 seconds | |
| 14. Activated Partial Thromboplastin Time (APTT) | | | 35–45 seconds | |

| Name of tests | Method of blood collection for test | Amount of blood | Normal value | Abnormal value |
|---|---|---|---|---|
| Miscellaneous Test | | | | |
| 1. Modified Kleihauer Betke acid elution test or blood film from maternal blood is made on a dry glass slide. It is fixed in 80% alcohol (ethanol) solution for 3 minutes, dried in air and sent to laboratory. | EDTA vial | | 2 mL venous blood | |

## INSTRUMENTS TRAY FOR CESAREAN DELIVERY (Fig. 47.89)

See Chapter 42 [v] for details

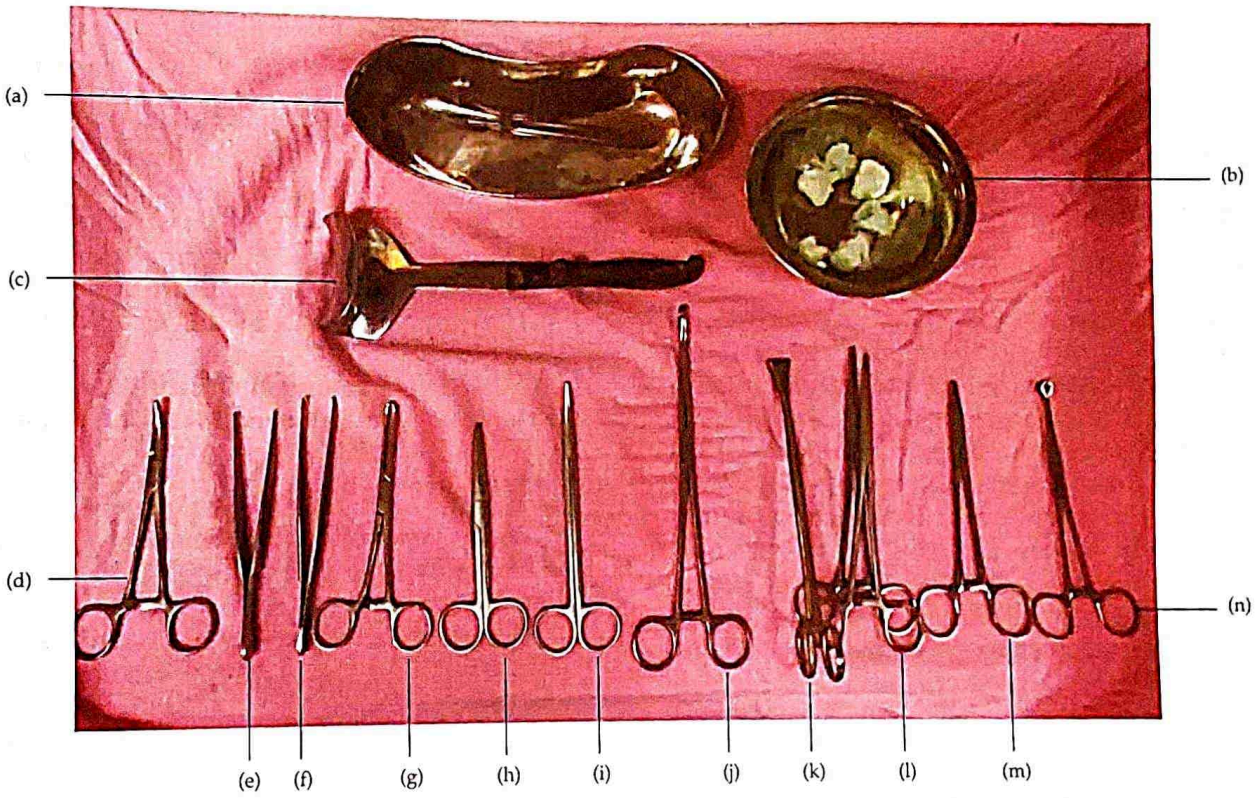



**Fig. 47.89:** (a) Scalpel Bard Parker handle with blade in a kidney bowl; (b) Gauge pieces in a bowl; (c) Doyen's retractor (to retract bladder); (d) Curved artery forceps; (e) forceps (to lift uterovesical fold); (f) Toothed forceps; (g) Allis' tissue forceps (to hold angles of uterine incision); (h) Mayo suture cutting scissors; (i) Metzenbaum dissecting scissors; (j) Sponge holder (cleaning abdomen, removing membranes, vaginal cleansing); (k) Green armytage hemostatic forceps; (l) Hemostatic clamps (artery forceps); (m) Needle holder; (n) Bebcock's forceps

## Suggested Reading

1. Cunningham F G. Reference Table Normal Laboratory Value in Uncomplicated Pregnancies. Williams Obstetrics, 25th Edition 2018, McGraw Hills, New York, USA, p. 1255–1272.
2. Kratz A, Pesce M A, Fink D J. Laboratory Values of Clinical Importance, in Jameson JL, Fauci AS, Kasper DL et al. (eds) Harrison's Principles of Internal Medicine, 20th edition McGraw Hill, New York, USA, 2018, p: Appendix A1–A16.
3. Parulekar SV Practical Gynecology and Obstetrics, 5th edition, 2014, Vora Medical Publications, Mumbai.

CHAPTER 48

# Theory and Practical Examination in Obstetrics

A rough guide is given about various examinations. However, the students are requested to check the exact details from their own college and university.

## MBBS EXAMINATION

Examination of Obstetrics and Gynecology is held at the end of eighth semester (Final Professional or Fourth Professional). The subject is divided into Obstetrics and Gynecology with more weightage being given to Obstetrics. Students have clinical posting in Obstetrics and Gynecology 2–3 times in different semesters with ward leaving assessments at the end of each hospital posting. Internal assessment is very important as students need to pass in it to pass the examination. Without passing in internal assessment, the students may not be allowed to appear in the final examination or may not pass the examination even if she/he passes the theory and practical examinations. Hence, students should take their hospital postings seriously as not only they learn the practical aspects of the subject by seeing patients and attending bedside classes, but score crucial marks in internal assessment which is essential to pass the final examination. By attending hospital postings carefully, they can attempt theory papers better and can answer well in practical examination as mostly management of a particular problem is asked which can be answered better if a similar patient has been seen.

The marks are divided into three parts.

1. Internal assessment
2. Theory examinations
3. Practical examination

The pattern and marks vary slightly in different universities but essential pattern remains the same.

For example, in Delhi University (Maulana Azad Medical College, Lady Hardinge Medical College and University College of Medical Sciences), the Obstetric and Gynecology examination is of 200 marks with internal assessment being of 50 marks. In All India Institute of Medical Sciences, New Delhi, the total marks in Obstetrics and Gynecology are 300 with internal/class assessment being very high and is of 150 marks (half of total marks). The division of marks in Delhi University and in AIIMS are given below.

**Marking Pattern in Obstetrics and Gynecology in Delhi and Most Universities**

| | Marks |
|---|---|
| **A. Theory Paper** | |
| 1. Theory Paper I (Obstetrics) | 40 |
| 2. Theory Paper II (Gynecology) | 40 |
| **B. Internal Assessment** | |
| 1. Theory | 25 |
| 2. Practical | 25 |
| **C. Practical Examination** | |
| 1. Long case (Obstetrics) | 35 |
| 2. Short case (Gynecology) | 15 |
| 3. Obstetrics viva | 10 |
| 4. Gynecology viva | 10 |
| Total | 200 |

**Comments**

1. Passing marks are 50% in theory and practical examinations separately.
2. Marks of viva are added in theory.
3. MCQs are usually not asked in Obstetrics and Gynecology paper.
4. All questions are to be attempted.

**Marking Pattern in Obstetrics and Gynecology in AIIMS, Final Examination**

Greater emphasis is laid on Obstetrics than on Gynecology and more questions are usually framed from Obstetrics. A

similar emphasis on Obstetrics is usually laid in the clinical examination also. The minimum pass marks in the theory and clinical examination are 50%.

| | | | Marks |
|---|---|---|---|
| A. Theory Examination | | | |
| Section A | | | 37 |
| Section B | | | 38 |
| Class assessment | | | 75 |
| | | Total | 150 |
| B. Clinical and Practical Examination | | | |
| Obstetrics | | | 30 |
| Gynecology | | | 15 |
| Viva-voce: | Obstetrics | 12 | |
| | Family Welfare | 8 | |
| | Gynecology | 10 | 30 |
| Class assessment | | | 75 |
| | | Total | 150 |
| | | Grand Total | 300 |

## Theory Paper

In most universities (e.g. Delhi University), there are separate theory papers of Obstetrics and Gynecology. In Obstetrics, in theory paper (40 marks) there are usually six questions with 4 long questions and two questions with two short notes each (total 4 short notes) (see Sample Paper below). In AIIMS, there is a common theory paper of Obstetrics and Gynecology. Section A is Obstetrics with two long questions and one question of 3 short notes while section B is of Gynecology with similar pattern (see Sample Paper below).

The questions cover almost all the Obstetrics with one (sometimes two) short note from neonatology section. Usually at least one question (sometimes two) from Operative Obstetrics (cesarean section, instrumental deliveries or other operations), one question from normal labour, one-two questions from abnormal labour which include abnormal lies and malpresentations are asked. Medical disorders during pregnancy are very important and questions always come from them especially from chapters like anaemia in pregnancy, heart disease, hypertension and diabetes. Sometimes questions are given from physiology of pregnancy and early pregnancy complications (see sample question paper below).

The Gynecology paper is also of 40 marks with similar distribution of marks with one short note or one question from contraception.

*Sample Delhi University Paper*

**OBSTETRICS INCLUDING SOCIAL OBSTETRICS**

Time: 3 hours Maximum Marks: 40

*Attempt all questions.*

*Use a separate answer sheet for each question.*

1. What are the causes of pre-term labour? How will you manage a woman at 32 weeks of pregnancy with labour pains? 6
2. Write short notes on:
   (a) Anti-D prophylaxis (b) Indications of LSCS 8
3. Classify severity of heart disease in pregnancy. How will you manage a primigravida with heart disease in spontaneous labour? 6
4. Define antepartum haemorrhage (APH). Enumerate complications of abruptio placentae. How will you manage a primigravida presenting at 34 weeks with abruptio placentae? 6
5. Write short notes on: 8
   (a) Indications and pre-requisites of outlet forceps delivery
   (b) Strategies to minimize perinatal transmission of HIV
6. What are the predisposing factors for multiple gestation? Describe the management of a second gravida with previous normal delivery presenting at 37 weeks with twin gestation in early labour? 6

*Sample AIIMS Paper*

**Final MBBS Examination**

Time: 3 hours Maximum Marks: 75

*Attempt all questions.*

OBSTETRICS AND GYNECOLOGY

Write Section A and Section B on separate answer sheets

**Section A (Obstetrics)**

1. Enumerate physiological cardiac changes during pregnancy. Describe the management of a primigravida with Rheumatic Heart Disease (RHD) with mitral stenosis in labour. 10
2. Enumerate absolute indications of cesarean section. How will you manage a 25 years old second gravida with previous cesarean section at 36 weeks of pregnancy? 10
3. Write notes on: 6 × 3 = 18
   (a) Partograph
   (b) Asphyxia neonatorum
   (c) External cephalic version

**Section B (Gynecology)**

1. Write classification of ovarian tumours. How will you manage a 28 years old para two with an ovarian cyst? 10
2. Discuss differential diagnosis of a 25 years old woman presenting with 2 months amenorrhea, abdominal pain and bleeding per vaginum. 10
3. Write notes on: 6+6+5 = 17
   (a) Medical abortion
   (b) Contraindications and complications of combined oral contraceptive use
   (c) Syndromic management of acute pelvic inflammatory disease (PID)

If some difficult question comes, which students did not answer well, it is often asked in practical in viva. So students must read the question they did not do well in theory.

## Practical Examination

It consists of a long Obstetric case of 35 marks, Obstetric viva of 10 marks, Gynecology case of 15 marks and Gynecology viva of 10 marks (Total marks 70).

Obstetrics is given more weightage with 45 marks.

Obstetrics long case is the most important aspect of practical examination and is quite often the deciding factor in passing the examination being of 30–35 marks. Usually antenatal case is given. Sometimes, puerperium case can also be given if sufficient number of antenatal cases are not available (a common situation as large number of students [about 30–35] are examined per day). Students get scared to get a case of puerperium. However, there is a clue here to the students. There are only two chapters on puerperium in the whole book (normal and abnormal puerperium), while almost two-thirds of the book is about antenatal cases. A smart student can prepare two chapters of puerperium very well and can score better marks than in an antenatal case as even the examiners have a soft corner for a case of puerperium while they expect good answers in antenatal cases like pregnancy with anaemia.

Two sample antenatal cases and one sample puerperium case are given in this chapter as a rough guidance to students. The students should prepare the Obstetric case very well and in complete details taking history of present illness, past history, medical history, obstetric history, family history, perinatal history, etc. (see sample history and Chapter 11 for details). There is plenty of time given to students to prepare their obstetric case. They must examine the patient in details including general physical examination.

Abdominal examination includes inspection, palpation, percussion and auscultation. At inspection, details of previous scars (e.g. cesarean section scar) are important.

Palpation is very important. Usually examiners want the students to palpate the abdomen properly by performing all the Leopold maneuvers correctly and to demonstrate them to the examiner (see Chapter 11 and sample case below for details).

If there is previous cesarean scar, the candidate is asked about scar tenderness. The uterine scar (not skin scar which may be vertical or transverse) which is almost always transverse and in suprapubic area should be palpated for any tenderness. Foetal heart should be auscultated as beats per minute. The candidate should answer that foetal heart can be heard in left spinoumbilical line (in left occipito-anterior position), 140 beats per minute, regular in rhythm.

In the end, candidate can summarize the case and tell the final diagnosis, e.g., Mrs. G K, a 28-year old Gravida 3, Para 1, Abortion, 1 live issue 1, with 34 weeks pregnancy with Rheumatic heart disease with mitral stenosis and with NYHA class 2 with false labour.

Examiner then usually asks the management of the case. The student should restrict to the management of that particular case and not the general management of that condition. The management includes both investigations and treatment and not just the treatment. Only appropriate investigations should be done and told. Candidates should tell the management as done in their hospital rather than from textbooks as they can then justify the treatment by saying that is how the case is managed in their hospital. The examiners usually accept such an answer and the internal examiners always support it even if external examiners do not agree with it.

Long cases are from obstetrics and are usually antenatal cases but can be a case of normal puerperium. Any of the following cases can be given.

1. Normal antenatal case in late pregnancy
2. Anaemia in pregnancy
3. Multiple pregnancy (usually twin pregnancy)
4. Heart disease with pregnancy (usually RHD with mitral stenosis)
5. Pregnancy with diabetes mellitus (usually gestational diabetes mellitus)
6. Pregnancy with pain abdomen (usually false labour pains)
7. Pregnancy with bad obstetric history
8. Pregnancy with hypertension (usually pre-eclampsia)
9. Pregnancy with breech presentation
10. Pregnancy with previous cesarean section
11. Pregnancy with intrauterine growth restriction
12. Pregnancy with hydatidiform mole
13. Pregnancy with threatened miscarriage

Usually undergraduates are not given sick patients like antepartum haemorrhage, severe pre-eclampsia, transverse lie, but due to non-availability of good cases any case can be given with advice to avoid palpation in sick cases.

### Obstetric Specimens

One obstetric specimen usually is asked in obstetric viva. Following is the list of common specimens.

1. Anencephalic baby
2. Hydrocephalic baby
3. Baby with other congenital malformations, like spina bifida or exomphalos
4. Specimen of hydatidiform mole
5. Rupture uterus
6. Specimen of placenta of twin pregnancy
7. Ectopic pregnancy (tubal pregnancy)
   **(see Chapter 47 for details)**

### Obstetric Instruments

In Obstetric viva, every student is asked at least one instrument out of the following list (see Chapter 47 for details of instruments). Students may be asked about Obstetric forceps and their application.

#### Obstetric Instruments usually kept in Examination

1. Forceps
   (*i*) Wrigley's forceps
   (*ii*) Das forceps (Long forceps)
   (*iii*) Keilland's forceps
2. Other instruments
   - Pinard's stethoscope
   - Disposable mucus sucker
   - Simple rubber catheter
   - Episiotomy scissors
   - Flushing curette
   - Curved artery forceps
   - Kocher's hemostatic forceps
   - Sim's speculum
   - Cusco's bivalve speculum

- Anterior vaginal wall retractor
- Cervical dilators
- Uterine sound
- Doyen's retractor
- Allis tissue forceps
- Karman's cannula
- Volsellum
- Foley's catheter (obstetric indications)
- Umbilical cord tie
- Ventouse cups (both metallic and silastic)

3. Instruments used for destructive operations like embryotomy scissors, decapitation hook, decapitation knife or saw, Breech hook with crotchet, Willet's scalp traction forceps

**Drugs**

Usually one drug is asked from each student. There are vials, tablets, injections or gels used specifically in Obstetrics from the list below. Students are usually asked the indications of drug, contraindications, dosage, route of administration, uses, side effects and any precaution. Students must know details of commonly used drugs, especially oxytocics (ergometrine, oxytocin, prostaglandins), magnesium sulphate (for several pre-eclampsia and eclampsia), tocolytics and anti-hypertensive medicines (see chapters 14 and 47 for details).

**Drugs for Obstetric Viva**

1. Oxytocics
   (*i*) Oxytocin injection
   (*ii*) Ergometrine or methyl ergometrine injection
2. Prostaglandins
   (*i*) Misoprostol tablet
   (*ii*) $PGF_2\alpha$ injection (Carboprost injection)
   (*iii*) Dinoprostone ($PGE_2$) gel
3. Antihypertensive drugs like, labetalol injection
4. Tocolytics like
   (*i*) Ritodrine injection or tablet
   (*ii*) Isoxsuprine injection or tablet
5. Anticonvulsants like
   (*i*) Magnesium sulphate injection
   (*ii*) Phenytoin injection
   (*iii*) Pethidine injection
   (*iv*) Diazepam injection
6. Analgesics and anaesthetic drugs
   (*i*) Lignocaine injection
   (*ii*) Bupivacaine injection
   (*iii*) Midazolam injection
   (*iv*) Pentazocine injection
   (*v*) Diclofenac injection
7. Anticoagulants
   (*i*) Heparin injection
   (*ii*) Low molecular weight heparin
   (*iii*) Oral Warfarin tablets

**Maternal Pelvis, Fetus and Foetal Skull**

Doll (fetus) and dummy (maternal pelvis) are very favorite of all the examiners. Every student is almost invariably asked any one aspect of maternal pelvis and fetus and foetal skull. The examiners expect every student to answer such questions nicely. The students must prepare it very well. They must memorize all the diameters of maternal pelvis at inlet, mid cavity and outlet (see Chapter 10 for details). They must know all the diameters of foetal head in various presentations. They must know mechanism of labour in various presentations and need to demonstrate it in front of examiners on the doll and dummy model. The examiners tend to change topic with each student to avoid repetition of questions and to avoid boredom. The examiner usually puts the foetal head in a particular presentation or position and ask the students to identify it, tell the name of denominator in that position, presenting diameter, how much is the diameter and the management. For example, the examiner may put foetal head in left occipito-anterior position for the first student. The candidate needs to say that it **is left occipito-anterior position, occiput** is the **denominator, suboccipito-bregmatic diameter** which is **9.5 cm**, is the **presenting diameter**. The student is then asked to demonstrate the mechanism of labour and should be able to deliver the fetus on doll and dummy model. From second student the examiner may ask **occipito-posterior position**, when the diameter is **occipito-frontal** (11.5 cm). From the next student they may ask **deep transverse arrest** and its management, then mento-anterior presentation, mento-posterior presentation, brow presentation, breech presentation, transverse lie, oblique lie and so on. In this way, students can be asked any of the normal or abnormal presentations and positions including the presenting diameters in them and the mechanism of labour. If the examiner is asking about **mento-posterior presentation, brow presentation, transverse lie**, students should not panic but consider themself lucky as there is no mechanism of labour. They have to just identify the presentation (e.g. **brow presentation**), tell the denominator and diameter (e.g. **chin, mentovertical diameter** which is 14 cm in brow) and say that **cesarean delivery** needs to be peformed as there is no mechanism of labour and students can easily score good marks. If a student is weak and is not answering questions well related to abnormal presentation, examiner may ask diameters of foetal skull, maternal pelvis and mechanism of labour in a normal patient that is in occipito-anterior position. If he/she is able to answer normal labour, examiners tend to pass him as that much every student is expected to know. If a student does not answer even mechanism of normal labour, examiner may be unable to pass her or him (see chapters 10, 16 and 23 for details.)

**Miscellaneous**

Sometimes a **CTG strip** showing normal or abnormal tracing (e.g. early deceleration, late deceleration, variable deceleration, sinusoidal pattern), (see Chapters 15 and 47 for details) may be kept especially in DNB examination. Normal or abnormal **ultrasound** or **X-ray findings** may also be kept (e.g. malpresentations, twin pregnancy).

If a student is not answering well, the examiner may ask him, as a last resort, what are the *investigations performed*

*in a normal antenatal woman, what is a routine prescription for a normal antenatal woman, management of normal labour, mechanism of normal labour and management of postpartum haemorrhage.* These are the simple questions which every student, aspiring to be a doctor, must know to at least pass the examination.

## Sample cases in obstetrics

For the convenience of students and better understanding, three sample cases are given below, one of which is often given in examination. For detailed history taking and examination, please refer to Chapter 11.

### Anaemia in Pregnancy Case

History: 22 years old, Mrs. V W/O Mr S S, Resident of Vill. Shampur, District Madhubani, Bihar.

$G_2P_{1+0+0+1}$, 37+1 weeks (37 weeks 1 day).

LMP (Date of last menstrual period: 10 November),

EDD (Expected date of delivery: 17 August)

POG (Period of gestation: 37 weeks 1 day on 28 July)

Admitted from antenatal clinic for blood transfusion.

**Main complaints**

1. Period of amenorrhoea 37 weeks 1 day
2. Easy fatiguability (1 month)
3. Swelling of feet (1 month)

#### History of Present Illness

Patient was a known case of anaemia before conception and had easy fatiguability off and on with shortness of breath.

It was a spontaneous conception.

**1st trimester**

No history of excessive nausea, vomiting, fever, X-irradiation, bleeding per vaginum or drug intake. Took folate supplementation.

**2nd trimester**

Booked at 16 weeks; Hb = 9.8 g.

Started on iron supplements twice a day, calcium supplements twice a day. Triple test was done for Down's syndrome screening which was negative.

Next visit at 20 weeks.

Routine antenatal investigations and ultrasound done which was within normal limits.

Received two doses of tetanus toxoid. Quickening started at 20 weeks of gestation.

**3rd trimester**

No history of pica, bleeding per rectum or vaginum, hematuria, fever with chills. No history of cough with expectoration, chest pain, palpitation, gastrointestinal or urinary complaints. No history of pain abdomen or discharge per vaginum. There has been history of swelling of feet with no relation to posture or rest or raised blood pressure. No history of headache, blurring of vision, epigastric pain.

Lost to follow-up for two and a half months and then reported with weakness and pallor.

Hb = 7.6 g; admitted, investigations sent.

Planned for parenteral iron therapy.

Received two doses of iron in first week of July intravenously uneventfully but did not complete the course. Discharged herself against advice.

Came after symptoms worsened over last 2 weeks.

She has received 1 unit of blood transfusion after admission (27 July). Blood tests were sent before and after transfusion.

**Obstetric history:** Married for 4 years

$G_2P_{1+0+0+1}$

1. 3 year back full term vaginal delivery at private hospital in Madhubani, Bihar, male child, 2.5 kg, alive and healthy.
   No antenatal, intrapartum, postpartum complications including PPH.
   Used barrier method (condoms) for contraception.
2. Current pregnancy

**Menstrual history**

Previous cycle 3–5/27–29 days, regular, average flow, no dysmenorrhea.

**Past medical history**

No past history of diabetes, hypertension, severe anaemia

**Past surgical history**: Not significant

**Family history**: Mother hypertensive on oral tablets. No family history of diabetes, genetic disorder or multiple pregnancy.

**Personal history**

Vegetarian, non-smoker, non-alcoholic

2000 Kcal diet, 45 g protein/day

Deficient in green leafy vegetables

Similar to pre-pregnancy diet pattern

Middle class family

Educated up to Class 8.

Husband: Small farmer educated up to class 10. Poor socio-economic status

Income: ₹ 2000 per month

**Examination**

**General physical examination**

Height 153 cm, weight 65 kg

Prepregnancy weight: 53 kg

Body mass index (BMI) 22.6 kg/$m^2$

Pallor + (moderate)

Bipedal pitting edema+

Pulse: 76/min; Blood pressure: 120/70; afebrile;

Respiration Rate: 15/minute

Breasts: Physiological changes

Thyroid: Normal

Chest, Bilateral vesicular breathing all over the chest without any accompaniment (respiratory system normal). Both heart sounds audible at all four areas without any murmurs (Cardiovascular system: Normal).

**Per abdomen:** Linea nigra and striae gravidarum present, no surgical scar

No hepatosplenomegaly

Fundal height: 36 weeks

Symphyseo fundal height (SFH) = 30 cm; Abdominal girth = 33 inches.

Estimated foetal weight by Johnson's formula: 30–12 × 155 = 2790 g, average liquor.

Grips (Leopold's manouvres):

On first Leopold grip, a broad non-ballottable pole suggestive of breech is felt at fundus.

On second Leopold grip, foetal back is felt on left side while limbs are felt on right side.

On third Leopold grip (Pawlik's grip): a ballottable foetal pole suggestive of foetal head is palpable, 5/5 head palpable, non-engaged.

On fourth Leopold grip: Findings of third grip are confirmed. A ballottable foetal head is felt, 5/5 palpable non-engaged.

**Single fetus in longitudinal lie with cephalic presentation with back on left** (possibly left occipito-transverse or anterior position).

**Auscultation**

FHR 144 beats per minute in left spinoumbilical line.

**Per vaginum examination**

(Findings are given; students do not have to do vaginal examination)

Cervix admitting one finger. 20% effaced. Soft, posterior, head at –3 station, membranes felt, no leaking.

Pelvis adequate.

**Diagnosis**

22 year old, $G_2P_{1+0+0+0+1}$ at 37 weeks 1 day period of gestation with moderate anaemia (received one unit blood in this pregnancy).

Examiner usually interrupts the student and asks related questions like confirmation of diagnosis, investigations, indication of bone marrow testing, medical treatment, indications of parenteral iron therapy, calculation of iron dose, techniques of giving parenteral iron, indications of blood transfusion, etc. Management during labour, including management in first stage, second stage and third stage is asked. Management during puerperium, especially contraceptive advice is very important (see Chapters 11 and 35[i] for details).

**Hypertension with Pregnancy**

Mrs. S K, aged 30 years, W/o Mr A K G, Resident of Kotla Mubarakpur, New Delhi.

**Main complaint**

Known case of hypertension with 34 weeks gestation.

**History of present illness**

LMP 2nd October 2020 EDD 9th July 2021

POG 34+6 sks

$G_3 P_{1+0+1+1}$

Pregnancy was confirmed by urine test.

**1st trimester**

She is a regular antenatal patient at the hospital after confirmation of pregnancy.

No history of excessive nausea and vomiting, bleeding or discharge per vaginum, fever or rash in the first trimester. There is history of increased frequency of micturition. There is history of folic acid intake from 8th week to 20th week of gestation.

**2nd trimester**

Felt quickening at 20th week

There is history of increased frequency of micturition.

Proteinuria (+1).

There is history of two doses of tetanus immunization and iron, folate and calcium intake. Triple test was done for Down's syndrome screening which was negative. History of targetted scan at 18 weeks which was normal.

**3rd trimester**

Took anti-hypertensive medication in the form of methydopa 500 mg thrice daily from 20 weeks onwards.

No history of pain abdomen, bleeding or discharge per vaginum.

No history of headache, blurring of vision, dizziness or decreased urine output. No history of urinary or bowel complaints, cough with expectoration or chest pain or palpitation.

**Menstrual history**

Previous cycles regular 2–3 days/28 days; No history of use of contraception.

**Obstetric history**

Married for 11 years

$G_3 P_{1+0+1+1}$

$G_1$ 2011—Full term, normal, vaginal delivery at private hospital, 2.5 kg female baby, breastfed for 9 months, alive + healthy. No hypertension in that pregnancy.

$G_2$ 2015—Spontaneous abortion at 8 weeks, followed by dilatation and currettage.

$G_3$ 2019—Present pregnancy

**Treatment history** - Methyldopa 500 mg thrice daily for hypertension.

**Past history**—Known hypertensive for past 10 years.

**Family history**—No family history of hypertension, diabetes, genetic disorder, multiple gestation.

**Dietary history**—She consumes about 2200 calories diet with about 50 gm proteins per day.

**Examination**

She is conscious, cooperative, well-oriented in time, space and person. Height: 155 cm, Weight: 68 kg

Pre-pregnancy weight not known.

Pulse—88 beats per minute, respiration rate 16 min, BP 130/86 mm Hg, afebrile.

No pallor, cyanosis, clubbing, icterus, lymphadenopathy, edema.

Thyroid (N), Jugular venous pressure (JVP) not raised.

Breasts normal, normal pregnancy signs, no inversion of nipple

Chest—B/L air entry normal, no added sounds.

CVS—Apex beat 5th left intercostal space, $S_1$ $S_2$ normal, No murmur.

Per abdomen; Inspection—linea nigra, striae gravidarum present, abdomen uniformly distended.

Palpation; Fundal height corresponds to 30 weeks period of gestation. On first Leopold grip broad non-ballotable breech is felt at fundus. On second Leopold grip smooth back is felt on right six while nodular limps are felt on left side. On third (Pawlik's) grip ballotable head is felt, not engaged 4/5 palpable above pubis. On fourth grip findings of third grip are confirmed.

Cephalic presentation, right occipito-anterior position, unengaged head.

Foetal heart heard in right spinoumbilical line, 140 beats per minute, regular in rhythm.

**Final diagnosis:** A 30-year old $G_3P_{1+0+1+1}$ at 34 + 6 weeks of her third pregnancy with known case of hypertension on methyldopa with foetal growth restriction.

Then the students will be asked the management of the case which includes investigations, complications, management during labour and treatment (see Chapter 32 for details).

### Sample Case of Puerperium

A case of puerperium may be given, if adequate number of antenatal cases are not available. Students need not get panicky. They should take the case as a case of puerperium and not as an antenatal case. Students often waste precious time and lose marks by telling all the details of antenatal history including trimester history which is not relevant as the woman has already delivered. Only pertinent points of antenatal period need to be told and rest history should be of puerperium as given below.

Mrs R K, 25 years old, W/o Mr A R, Address: 42, Vijay Mandal Enclave, Hauz Khas, New Delhi—110016.

She is a case of second day of normal puerperium. She had a full term normal vaginal delivery yesterday (or had an emergency cesarean delivery for foetal distress in case of cesarean puerperium) at 4 p.m. and a live male baby, weighing 2.9 kg with APGAR 8/10 and 10/10 at 1 and 5 minutes respectively was delivered. She was a booked case of our hospital and had regular antenatal care. She received two doses of tetanus toxoid, received iron, folate and calcium supplementation. There were no antenatal problems. There is no history of fever **(to rule out puerperal pyrexia)**, excessive bleeding per vaginum **(to rule out postpartum haemorrhage)**, pain or swelling at vulva **(to rule out vulval haematoma or episiotomy complications)**, no history of excessive distension of abdomen **(to rule out non-involution or subinvolution of uterus)**, pain or abnormal discharge from wound site **(in case of cesarean section to rule out wound complications)**. She is breastfeeding the infant well with no difficulty in breastfeeding, pain, swelling or abnormal discharge from breasts **(to rule out breast complications like failing lactation, cracked or inverted nipples, mastitis or breast abscess)**.

No history of abnormal sleep rhythm, sadness, depression or abnormal or violent behaviour **(to rule out postnatal depression and puerperal psychosis)**.

No history of pain, swelling or tenderness in legs **(to rule out thrombophlebitis or deep vein thrombosis of legs)**.

No history of chest pain or breathlessness **(to rule out pulmonary embolism)**.

No history of foul-swelling discharge per vaginum **(to rule out puerperal sepsis)**.

No history of burning or frequency of micturition or difficulty in passing urine **(to rule out urinary tract infection or local injury)**.

History of passage of motion and no history of constipation **(to rule out bowel problem)**.

Some details of other histories can be given.

**General physical examination** is like antenatal case.

**Examination of legs**, calf tenderness, demonstration of **Homan's sign** (dorsi flexion of foot causes pain in leg) **to rule out deep vein thrombosis**.

**Breast examination:** Breasts are full with normal nipples. No redness, cracking of nipple or retraction of nipple.

**Abdominal examination:** Striae gravidarum present. Uterus well involuted to 2 cm below umbilicus (8 cm above pubic symphysis). No tenderness.

**In case of cesarean puerperium:** Cesarean wound is healthy, no inflammation, gaping or abnormal discharge.

**Local examination:** No tear or vulval haematoma, episiotomy wound healthy, lochia rubra is present, no foul smell.

**Examination of baby:** The students must not forget about the baby and must take history and examine the baby and tell details to the examiner, e.g. baby is fine with no complications, accepting breastfeeding. Baby has been given BCG vaccination. Weight is 2.9 kg, Temperature is 37.3 °C. No pallor, heart and chest examination normal.

**Final diagnosis: A puerperal woman with day 2 of normal puerperium with normal baby.**

Usually examiner interrupts the students at the time of history and may ask them about some complications of puerperium, e.g. about various types of lochia. Candidate may be asked about management of various puerperal complications, like deep vein thrombosis, puerperal sepsis, subinvolution of uterus, postnatal depression, puerperal psychosis, breast complications (e.g. mastitis, breast abscess) (see Chapters 29 and 30 for details). Students should read chapters on normal and abnormal puerperium well to score good marks. Usually good marks are given in a case of puerperium, if reasonable answers are given.

## Objective Structured Clinical Examination (OSCE)

OSCE is just another way of practical examination which is a very fair system in which all the students of that batch are asked the same questions on a format to give uniformity to the examination. MRCOG Part-2 practical examination conducted by Royal College of Obstetricians and Gynaecologists, London is totally OSCE with 12 stations on different aspects of obstetrics and gynecology. Usually, in

India, OSCE is not part of clinical examination but is being introduced in DNB examination. In AIIMS, MBBS final practical examaination 5–10 marks OSCE is held. It is very easy. Basically, there are 5 stations with first station of an instrument (e.g. Wrigley's forceps) with definite questions (e.g. write two indications, one complication): Second station is a specimen (e.g. rRupture uterus, questions: identify the specimen, cause, management), etc. Third station can be a drug (e.g. oxytocin) with questions like (2 indications, 2 side effects), fourth station may be a X-ray (e.g. pregnancy with anencephaly) with questions (identify the condition, management and prevention) or ultrasound (e.g. twin pregnancy with two foetal heads) with questions (diagnosis, complication, management). Fifth station may be a cardiotocogram strip (e.g. Late [(Type 2) deceleration] with questions (identification, two causes, management). In this way, every student will be asked the same questions, so there is no disparity.

## Postgraduate MD or MS or DNB examination in Obstetrics and Gynecology

### Theory examination

Most universities including Delhi University have four theory papers with some variation.

Paper 1 : Anatomy and Physiology in Obstetrics and Gynecology
Paper 2 : Pathology in Obstetrics and Gynecology
Paper 3 : Obstetrics including Neonatology
Paper 4 : Gynecology and Family Planing

### AIIMS (New Delhi) is slightly different

Paper 1 : Basic Sciences
Paper 2 : Obstetrics (including Neonatology)
Paper 3 : Gynecology
Paper 4 : Recent Advances and Demography

### DNB (Diploma of National Board of Examinations)

Paper 1 : Basic Sciences
Paper 2 : Obstetrics (including Neonatology)
Paper 3 : Gynecology
Paper 4 : Recent Advances

#### Marking Pattern

Each MD paper is of 100 marks.

In each paper there are three long questions of 25 marks each. The fourth question contains five short notes of 5 marks each. DNB paper has 10 questions of 10 marks each (total 100 marks). Candidates have to separately pass in theory examination by scoring at least 50% marks. In many universities (e.g. Delhi University) grades are given instead of marks where A: 70–80%, B+: 60–70%, B: 50–60%, B–(minus): 40–50 (fail), C: clear cut fail. While B– (minus) can be compensated by B+ or A, score C shows blunder or dangerous practice and usually cannot be compensated by better performance in other parts.

In DNB at least 200 marks (out of total 400 marks) have to be obtained to pass theory examination. In DNB, only candidates who have cleared theory examination are allowed to appear in practical examination.

#### Practical Examination

There are some variations in different universities but essence remains the same. In Delhi University (combined for various Delhi hospitals) up to 7 candidates can be examined in one day. If there are more candidates examination is held for many days but each candidate has to appear only on one day. In AIIMS, only 3 candidates can be examined in one day. If there are more students, then it is held for 2–3 days but all candidates have to come for all 2–3 days.

#### Delhi and Most Other Universities

1. Obstetric long case (antenatal case)
2. Obstetric short case (Puerperium, normal, abnormal or cesarean case)
3. Gynecology long case
4. Obstetric Viva: Doll and Dummy, forceps, ventouse, obstetric specimen, obstetric drugs, instruments.
5. Gynecology and Family Planning Viva: Instruments, specimen drugs used in gynecology are kept. In family planning various contraceptives like condom male or female, combined oral pills, intrauterine devices including (LNG-IUS), progestogen only pill, emergency contraception are kept.

There are six examiners (3 internal, 3 external) with three pairs (one internal and one external) each. The candidates are divided in such a way that each student faces every examiner. Thus, first set of examiners takes obstetric long case of first 2–3 candidates, gynecology long case of second 2–3 candidates and obstetric short case of third 2–3 candidates and so on.

There are two tables combined for viva with first table containing obstetric viva and three examiners (2 external, 1 internal) while the second table contains gynecology and family planning viva with three examiners (usually 1 external, 2 internal). First 3 examiners (usually external examiners ask more questions) ask about obstetrics while the second 3 examiners ask about gynecology and family planning but all examiners listen to the answer and can intervene at any stage.

Marking pattern is by grades A: 70–80%, B+: 60–70%, B: 50–60% (pass), B–(minus): 40–50 (fail), C: clear cut fail

The candidate has to score at least 5 Bs to pass the examination, B minus can be compensated by B plus or A (e.g. if a candidate has scored 2 B+, 1 B, 2 B–, he or she passes the examination). Obstetric long case is given slightly more weightage. However, if a candidate has scored 'C' at any place which shows blunder or dangerous practice, he or she is usually failed and it cannot be compensated by B+ or A.

AIIMS: There are four examiners (2 external, 2 internal)

1. Obstetric long case
2. Gynecology long case
3. Obstetric short case
4. Gynecology short case

Two examiners each (1 external, 1 internal) take 2 parts (usually obstetric long case + gynecology short case or gynecology long case with obstetric short case).

In viva, all 4 examiners sit on the table which contain

1. Dummy and Doll
2. Specimens (usually gynecology)
3. Instruments
4. Contraceptives
5. X-ray, CTG strip, ultrasound, etc.
6. Drugs

The candidates have to usually overall pass in cases and viva separately but it is at the discretion of examiners and slightly lower grade can be compensated by a better grade in other parts. Practical is given more importance but theory and internal assessment can help in deciding in a borderline candidate.

**DNB:**

| | | |
|---|---|---|
| 1. | Obstetrics long case | 80 marks |
| 2. | 3 short cases (20 marks each) (usually 2 gynecology and 1 obstetric cases, but can be different) | 60 marks |
| 3. | Post operative round (discussion of a post operative case) | 40 marks |
| 4. | OSCE: 10 spots of 2 marks each (2 questions)<br>i. Obstetric instrument<br>ii. Gynecology instrument<br>iii. Contraception<br>iv. Obstetric specimen<br>v. Gynecology specimen<br>vi. Obstetric drug<br>vii. Gynecology drug<br>viii. Cardiotocogram (CTG) strip<br>ix. X-ray or ultrasound picture<br>x. Doppler picture | 20 marks |
| | There may be some variation from candidate to candidate and clinical case scenarios can also be kept. | |
| 5. | Obstetric viva (2 separate examiners): Doll and Dummy, forceps, ventouse, obstetric drugs, specimens and instruments | 50 marks |
| 6. | Gynecology and Family Planning (2 separate examiners): gynecology instruments, specimens, family planning (various instruments, devices, pills, etc.) | 50 marks |

Thus, out of total 300 marks in practical in DNB examination, a candidate has to score at least 150 marks (50%) to pass the examination.

### Diploma in Gynecology and Obstetrics (DGO) Examination of Various Universities

Usually there are 2 theory papers

i. Obstetrics 100 marks
ii. Gynecology 100 marks

Each paper has two long questions of 30 marks each (60 marks) and 4 short notes of 10 marks each (40 marks) (Total 100 marks).

Practical examination is of 300 marks. There is one Obstetrics long case of 100 marks. One Gynecology long case of 100 marks, one table viva of Obstetrics of 50 marks, one table viva of Gynecology of 50 marks. There are 4 examiners (2 external, 2 internal). One pair of examiners (one internal, one external) takes Obstetric long case with Gynecology table viva while the other pair takes Gynecology long case with Obstetric table viva for fairness though all 4 examiners sit together for table viva. Candidate has to score 50% marks in both theory and practical to pass the examination.

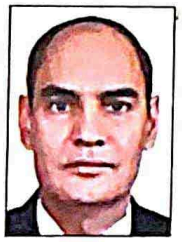
### Summary

The questions may be asked in any format, but the essence remains the same. Ultimately, it is the knowledge of Obstetrics and Gynecology which is being tested. If students have attended their clinical duties seriously, have read the subject well, they will be able to answer the questions well in theory as well as in practical. Best of luck for your examination.

## THE BOOK

- The **Third Edition** of **Textbook of Obstetrics** has been systematically divided into 12 sections.
- This latest third edition of the book contains all the latest developments in the obstetrics including COVID-19 in pregnancy.
- Amended MTP Act has been incorporated in Chapter 13.
- Maternal Collapse in Pregnancy has been added in Chapter 41.
- Evidence based medicine and guidelines from the prestigious institutions have been incorporated throughout the book.
- Various Health Programs in Obstetrics have been given in more details.
- There is a detailed chapter on obstetric instruments, specimens, drugs and spots and laboratory tests for better performance in practical examination.
- A separate chapter on theory and practical examination in obstetrics has been included with sample papers, sample cases and marking patterns prevalent in undergraduate and postgraduate examinations in obstetrics.
- All other chapters have also been thoroughly updated by including latest developments and advances.

## THE AUTHOR



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He has written and edited five books including Textbook of Gynecology, Textbook of Gynecological Nursing and contributed chapters in **Progress in Obstetrics and Gynecology** edited by Prof. John Studd from United Kingdom. He has presented over 200 papers in various International and National Conferences and has been awarded many times by Royal College of Obstetricians and Gynecologists (RCOG), London and won Indian Council of Medical Research's Amrut Mody Unichem Prize on his research work on anemia during pregnancy. His paper on 'Role of Lycopena in Prevention of Pre-eclampsia' published in International Journal of Gynecology and Obstetrics got the best paper award in the year 2003.

Dr. Sharma has been involved with many research projects with Oxford University, UK, Indian Council of Medical Research, Department of Science and Technology and Ministry of Health, Government of India.

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