1 FERTILIZATION, IMPLANTATION AND EARLY DEVELOPMENT OF FERTILIZED OVA

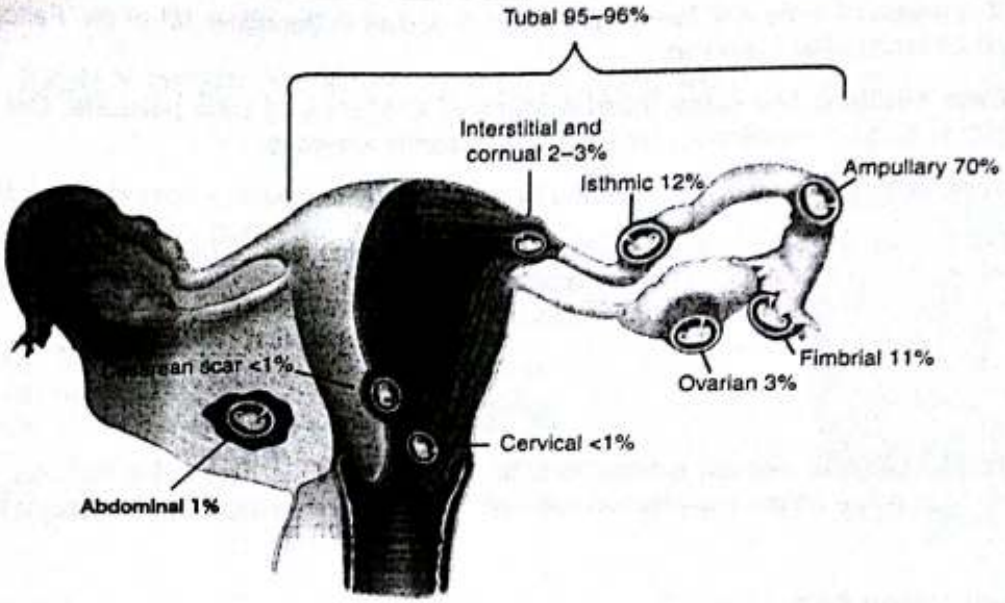


Fig 1:1 Oocyte Fertilization

GAMETOGENESIS

Primitive germ cells are present in the embryo by the end of the **third** week. They start to migrate via the dorsal mesentery and reach the indifferent gonad by the end of the **fourth** week of development. Gametogenesis reduces the number of chromosomes to half and alters the shape of germ cells to suit their functions.

Oogenesis starts in utero and is arrested in the prophase for years to be completed just before ovulation, while spermatogenesis starts at puberty and continues indefinitely. In oogenesis only one mature ovum is produced from an oocyte.

Basic Features of Oogenesis:

- *Proliferation* of germ cells into daughter gonocytes by mitosis
- *Growth* of some daughter gonocytes into primary gonocytes
- First maturation *division* is a reduction division with long prophase
- Second maturation *division* with no prophase.

Pre-Fertilization Changes:

These change occur in the upper part of the female genital tract caused by influx of calcium:

A. Sperm Motility: Testicular sperms are immotile and have poor ability to fertilize the ova. Sperm motility is acquired as they migrate through the epididymis but the maximum velocity (4-mm per minute) is achieved in the female genital tract.

B. Sperm Capacitation: It denotes the changes occurring in the sperm before it is capable fertilization. It is caused by calcium influx and its characteristic features include both increased sperm motility and acquired ability of the sperm to bind to the zona pellucida.

C. Acrosomal Reaction: This reaction involves rupture of the acrosomal membrane with release of proteolytic enzymes that help the sperm to penetrate the zona

FERTILIZATION

It is fusion of male and female pronuclei. It occurs in the distal 1/3 of the Fallopian tube, within the first 24 hours after ovulation.

- **Zona binding:** The sperm head recognizes and binds to zona pellucida; this is associated with acrosomal reaction and release of acrosomal enzymes.
- **Zona penetration:** This is facilitated by acrosomal release.
- **Oolemmal fusion:** Oolemmal fusion is facilitated by integrin B1-6. As soon as the sperm contacts the vitelline membrane two reactions are triggered, first resumption of meiosis, and formation of cortical granules that blocks polyspermy.

IMPLANTATION

Implantation or nidation is imbedding of fertilized ovum in the uterine mucosa. Many important phenomena occur before implantation namely, fertilization, cleavage, and blastocyst formation.

Implantation bed

The endometrium is columnar ciliated resting on a basement membrane. The endometrial glands are simple tubular ones, but become tortuous and full of glycogen under the effect of progesterone. The endometrial stroma is formed of stromal cells, stromal vessels, leucocytes, and macrophages in a matrix of collagen, fibronectin, laminin, and Gags.

During the luteal phase of the menstrual cycle, progesterone causes both stromal cells and glands to enlarge and store glycogen in preparation for nidation, should fertilization occur.

The endometrium suitable for nidation is ***secretory***, with a thickness not less than ***10 mm***, proper in phase with corpus luteum hormones. Leukemia inhibiting factor (LIF) is a cytokine of 1L-6 family that is maximally expressed in the secretory endometrium, and has a role in implantation as it forces trophoblastic differentiation.

The decidua (the mucosa, or endometrium, of the pregnant uterus), shares in the formation of the placenta, and it protects the baby from immune attack. Leukocytes migrate to the endometrium at the time of nidation and in the premenstrual days, helping the process of implantation

Stages of implantation:

1. ***Apposition of blastocyst:*** This is a close contact brought about by pinocytosis of uterine fluid, and the glandular crypts of secretory endometrium.
2. ***Adhesion of trophoblast:*** The zona pellucida disappears and the blastocyst hatches, out to come in contact with the surface uterine epithelium.
3. ***Invasion of the trophoblast:*** Trophoblastic invasion is brought about by a variety of proteases secreted by trophoblast.

Implantation Window:

It is the time interval in which the endometrium is ***receptive*** for the implantation of the blastocyst. This occurs 7-10 days after mid-cycle LH surge.

The blastocyst starts to implant on the 7th day after fertilization (LH +7); it is a slow process, which is complete by 10th day after fertilization (LH +10).

There should be synchrony between the stages of ovular development and that of bed preparation otherwise implantation may fail.

EARLY DEVELOPMENT OF FERTILIZED OVA

- ***The Zygote:*** Fertilization occurs in the lateral end of the fallopian tube. Once fertilization is achieved the *final maturation division* is completed, the second polar body is extruded, and a *zygote* is formed.

- ***The Morula:*** The zygote divides repeatedly by cleavage, as it passes down from the fallopian tube to the uterine cavity, to form a solid mass of cells known as the *morula*. The journey in the fallopian tube takes about 3 days, most of them at the ampullary isthmic junction.

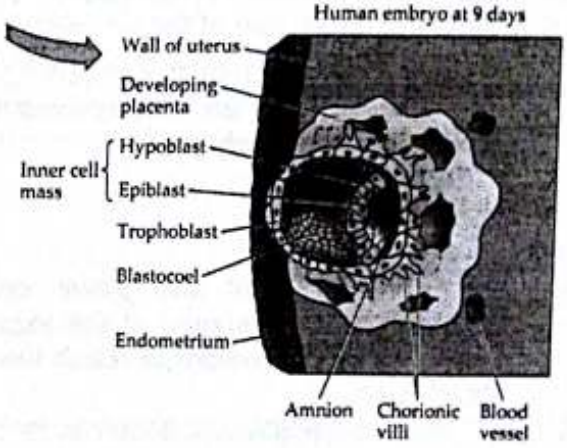


Fig 1:2 The Morula & early blastocyst

- ***The Blastocyst:*** When the morula reaches the uterine cavity it starts to imbibe water and an eccentric space appears dividing cells into two groups:

  1. The *inner cell mass*, (or embryonic cells),
  2. The *outer cell mass*, (or trophoblastic cells), forming the outer cover.

  The whole structure is called the *blastocyst*. It represents the implantation stage. With disappearance of the zona pellucida the trophoblast is exposed to invade the endometrium.

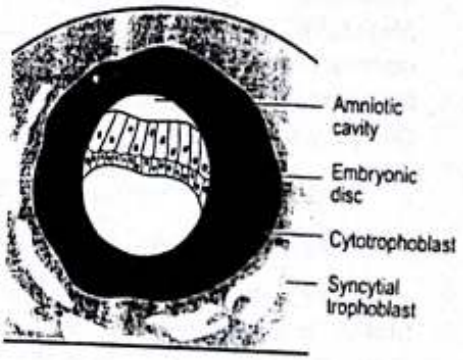


Fig 1:3 The Blastocyst

- ***Trophoblastic Differentiation:*** By the eighth day after fertilization the blastocyst is partiality implanted into the decidua and the trophoblast has differentiated into <u>cytotrophoblast</u> and <u>syncytiotrophoblast</u>.
  The trophoblast is active and secretes human chorionic gonadotropins (hCG) which has a stimulatory effect on the corpus luteum thus indirectly prepares the bed for nidation.

- ***The primitive villi:*** By the tenth day after fertilization, implantation is complete and the trophoblast proliferates greatly and forms primitive villi that open decidual vessels and create primitive intervillous space circulation.

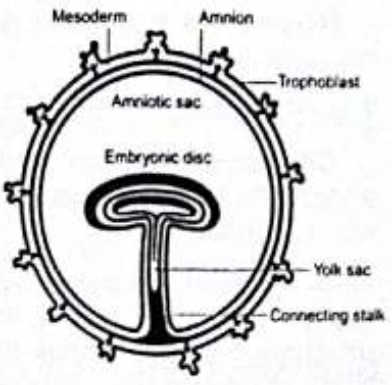


Fig 1:4 The embryonic disc

Arrangement of cells into an Embryo

Two cavities appear in the inner cell mass the *yolk sac cavity* and the *amniotic cavity* separated by the ***bilaminar embryonic disc***.

A mesodermal layer develops in the middle of disc and change the bilaminar disc into ***trilaminar*** one; this mesodermal layer grows outward to form the *extraembryonic mesoderm*.

Multiple cavities appear in the latter that coalesce to form extraembryonic coelom; this separates the extraembryonic mesoderm into two layers; both layers are connected by the ***body stalk*** the forerunner of the *umbilical cord*.

The embryonic disc comprising the ***ectoderm, endoderm*** and ***interposed mesoderm*** will become the ***embryo***.

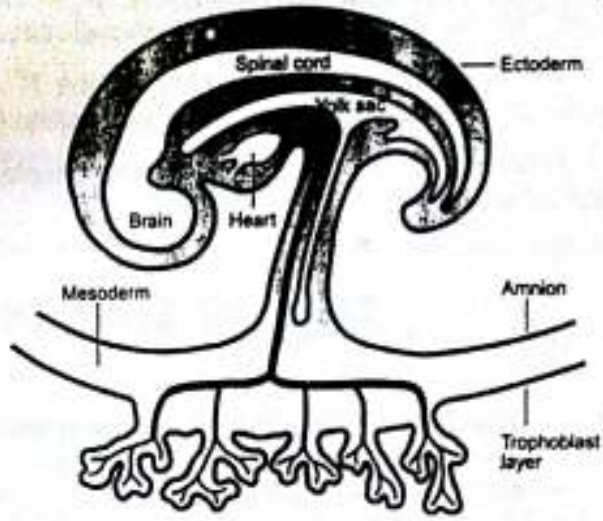


Fig 1:5 Early Embryonic development

The Umbilical cord

As development and growth continue, the amniotic cavity enlarges at the expense of the extraembryonic coelom to reach the wall of the blastocyst.

Part of the yolk sac becomes incorporated in the embryo and the remainder forms a vestigial tube in the *umbilical cord*.

Blood vessels develop in the intraembryonic mesoderm and mesoderm lining the trophoblast, extension of these vessels along the connecting stalk results in the formation of ***two arteries and one vein*** of the umbilical cord, carrying ***only fetal blood***.

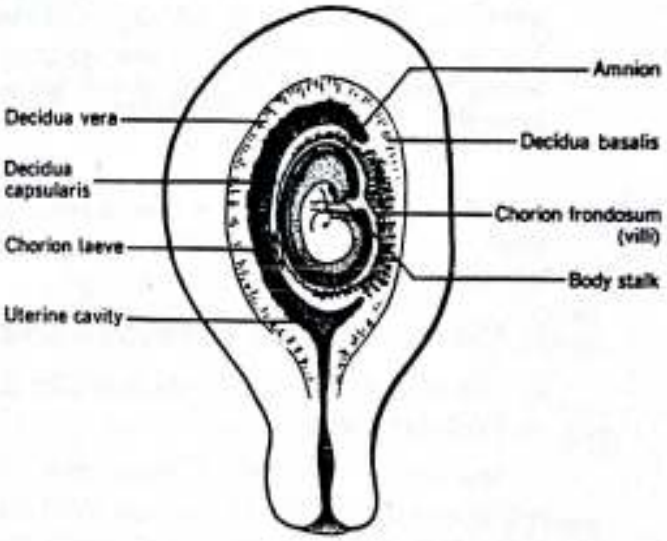


Fig 1:6 Final Embryonic development

The heart and Major Blood Vessels:

Within the embryo the vessels at the cephalic end differentiate to form the heart. By the 7th week from the LMP, while the patient wanders why her menstruation didn't come, the fetus already has a heart and starts pumping blood to and receives nutrient and oxygen from the primitive placenta.

Blood of the fetus does not come in contact with that of the mother and the two are separated by the placental barrier.

The Placenta:

Chorionic villi at first cover the whole blastocyst but later on those facing the decidua capsularis atrophy to form *chorion leave*, while those facing decidua basalis branch and re-branch to form *chorion frondosum*.

The placenta is formed from the chorion frondosum and underlying decidua basalis. There are two types of chorionic villi, the *nutritive villi* floating in the intervillous space and the *anchoring villi* for attachment to the decidua and invasion of the spiral arteries of the myometrium to create low resistance vessels essential for fetal growth and development.

2 THE PLACENTA, CORD, AND FETAL MEMBRANES

The Placenta

- *Anatomy and Histology*
- *Placental Physiology*
- *Abnormalities of the Placenta*
  1. *Shape*
  2. *Size and weight*

The umbilical Cord

- *Anatomy and Histology*
- *Abnormalities of the cord*

The Foetal Membranes

The amniotic fluid

Foetal Circulation

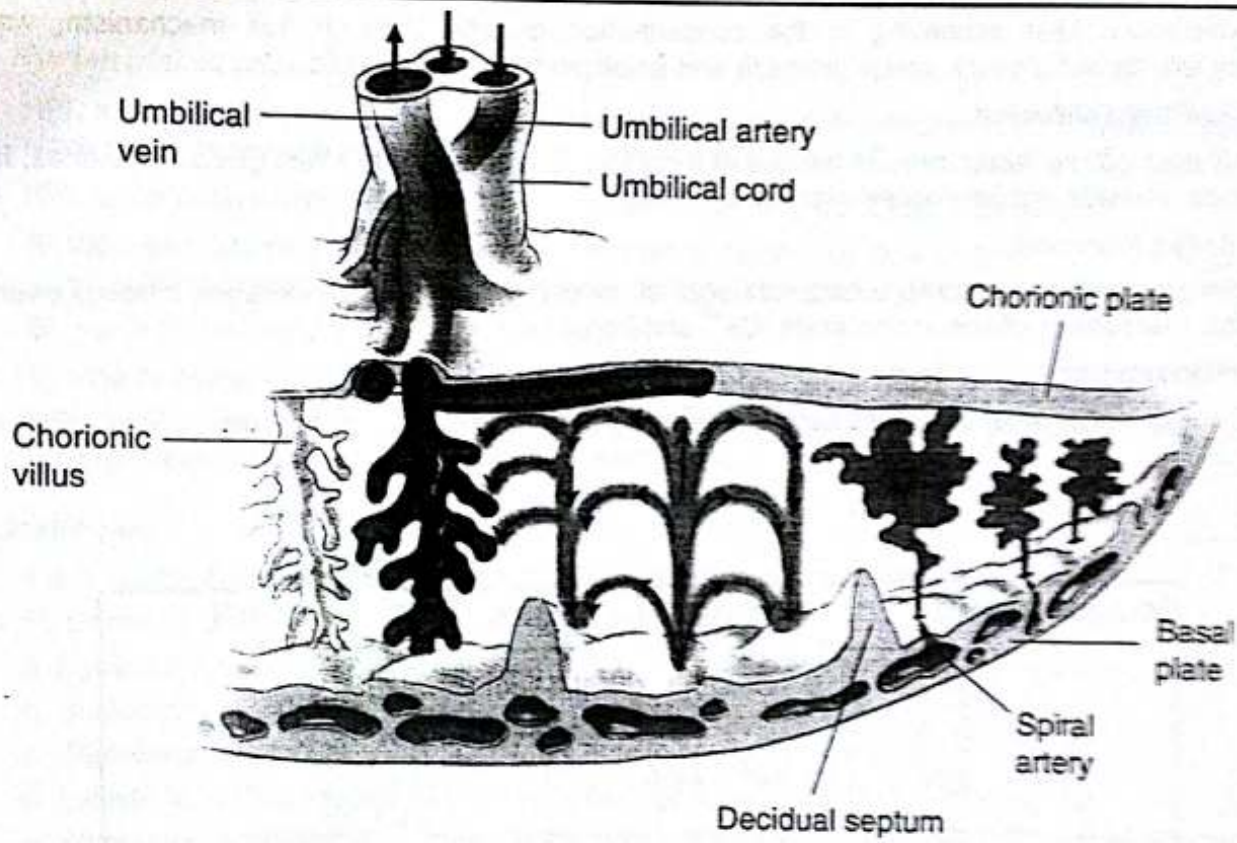


Fig 2:1:The Placenta

ANATOMY AND HISTOLOGY:

- **The placenta** represents the interface between the mother and the fetus. It is formed from the chorion frondosum and the decidua basalis.
- **Morphology:** at term the placenta is discoid in shape, about 20 cm in diameter, 1 inch in thickness at the center, 0.5 inch at the periphery and about 500g in weight. It has 2 surfaces:

1. *Fetal surface:* Smooth, glistening and is covered by amnion which is reflected on the umbilical cord which is inserted at or near the center of this surface.
2. *Maternal surface:* Dull grayish red in color, divided into 10-38 lobules (called cotyledons). Cotyledons are separated by fibrous tissue which is avascular.

- **Attachement:** the placenta is normally attached to the upper uterine segment, 60% in the posterior wall, 40% in the anterior wall.
- **Microscopically:** the placenta is formed of branching villi swimming into blood spaces (intervillous space) in which maternal blood circulates. However, fetal and maternal blood do not communicate as the fetal blood circulates in the vessels inside the chorionic villi.
- **The placental barrier:** tissues that separate maternal and fetal blood are called collectively the placental barrier. Placental barrier is composed of the wall of the fetal blood vessels, the villous stroma, the cytotrophoblast layer then the syncytiotrophoblast layer.

PLACENTAL PHYSIOLOGY:

The human placenta has three main functions, namely transfer of nutrients, production of a group of hormones and enzymes (endocrine and metabolic function), and an immunological role.

A) TRANSFER FUNCTION: can occur through many mechanism:

1. ***Simple diffusion:***

   Substances pass according to the concentration gradient.Through this mechanism, water, oxygen, carbon dioxide, waste products and small particles (MW < 1000) can pass.

2. ***Facilitated diffusion:***

   Diffusion can be faster through the use of a carrier. This mechanism affect glucose, ketones, fatty acids, steroids and fat soluble vitamins.

3. ***Active transport:***

   This mechanism can pump substances against concentration gradient, therefore it needs energy. This mechanism affects amino acids, Ca^{++} and iron.

4. ***Pinocytosis:***

   Through which large molecules such as immunoglobulins and LATS can pass.

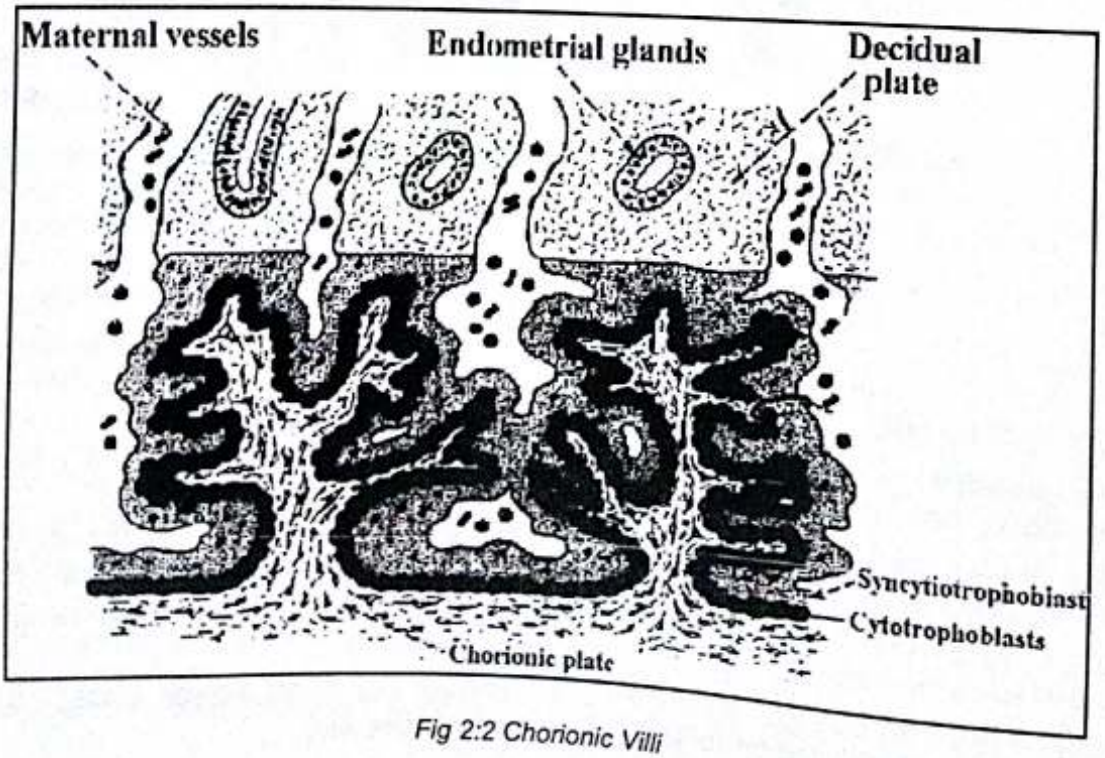


Fig 2:2 Chorionic Villi

B) ENDOCRINE FUNCTION:

1. *Human chorionic gonadotrophin (hCG):*

- It is a *glycoprotein hormone* produced by the *syncytio-trophoblasts*, consisting of an Alpha subunit (similar to all anterior pituitary hormones) and a B-subunit which is specific to hCG.
- hCG secretion starts few days *after fertilization*, reaches a maximum at 8-10 weeks of pregnancy and then slightly decreases till 20 weeks and is maintained till the end of pregnancy where it disappears few days after delivery.
- hCG main function is *maintenance of corpus luteum function* that supports early pregnancy till the development of the placenta.Its level nearly doubles evey 48 hours in the first 6-8 weeks of pregnancy and correlates with the integrity of the pregnancy and its duration.
- hCG levels are abnormally high in cases with vesicular mole, choriocarcinoma, multifetal pregnancy, Rh isoimmunization, and Down syndrome. Levels are abnormally low in cases of impending abortion and ectopic pregnancy. It can be used as a screeniung marker for cases of choriocharcinoma, and follow up of gestational trophoblastic disease (GTD).

2. *Human placental lactogen (hPL):*

- HPL is a large *protein hormone* secreted from the *syncytiotrophoblasts*. HPL starts to appear since early pregnancy and *steadily increases* till before labor.
- HPL is the *main metabolic hormone* of pregnancy carrying the following functions:
  - A. Induces maternal hyperglycemia to maintain a continuous flow of glucose to the fetus via stimulating glycogenolysis, and inhibiting maternal peripheral glucose uptake (anti-insulin).
  - B. maintain free fatty acids flow to the fetus.
  - C. Inhibits gluconeogenesis to preserve amino acids flow to the fetus.
- HPL levels are *high* in twin gestation, Rh-isoimmunization, diabetes and prolonged fasting; while it is *low in* all cases with placental insufficiency.

3. *Oestrogen:*

- It is a *steroid hormone* secreted form the *fetoplacental unit (*placenta and fetus).
- Functions of Oestrogens:
  - a. Hypertrophy and hyperplasia of uterine muscles and Increased uterine blood flow.
  - b. Stimulation of the development of the breast duct system.
  - c. Stimulation of prolactin synthesis.
  - d. Inhibits pituitary gonadotropin secretion leading to inhibition of ovulation.
  - e. Stimulates oxytocin receptor formation and increases uterine excitability in late pregnancy preparing for labor.

4. *Progesterone (P):*

- P is a *steroid hormone* secreted by *syncytiotrophoblasts*. It progressively increases throughout pregnancy then declines during the last week before parturition probably due to placental aging.
- Functions:
  - a. Maintains secretory activity of the endometrium making it suitable for implantation.
  - b. Inhibits uterine activity throughout pregnancy preventing it from expelling the conceptus.
  - c. May have a role in immunological materno-fetal acceptance.
  - d. Responsible for uterine softness (oedema).
  - e. Stimulates the development of the alveolar system of the breast.

C) IMMUNOLOGICAL FUNCTION:

The placenta appears to be an important factor in maternal acceptance of the fetus.

ABNORMALITIES OF THE PLACENTA

A) Abnormalities in shape:

1. ***Placenta membranacea (diffuse placenta):***
   The placenta is large and thin. Part of the placenta may be implanted over the lower uterine segment (placenta previa) causing antepartum hemorrhage. A part of the placenta may be retained after labor causing postpartum hemorrhage.

2. ***Bilobate or multilobate placenta:***
   Placenta is made of 2 or more lobes connected by placental tissue.

3. ***Bipartite and multipartite placenta:***
   The placenta is made of 2 or more separate parts almost equal in size, connected by membranes.

4. ***Succenturiate placenta:***
   The placenta is made of a large part and a small accessory part which are separate, and are connected by membranes.The accessory part may be retained in the uterus leading to postpartum hemorrhage and in this case we find a circular gap in the membranes from which vessels pass to the edge of the main placenta.

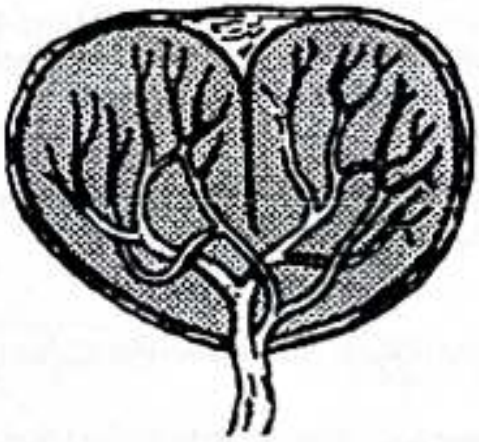


Fig 2:3 Bilobate placenta

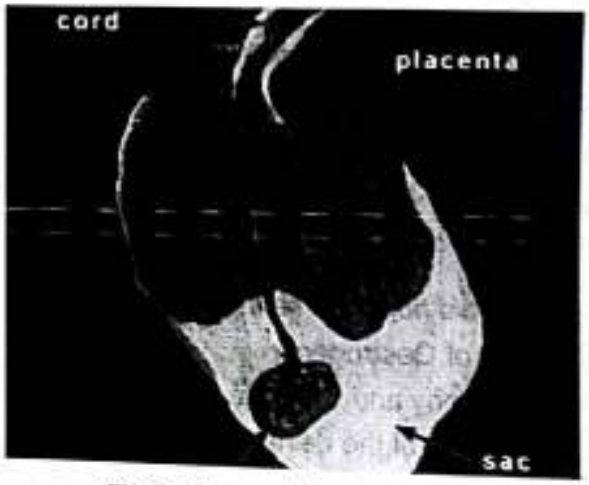


Fig 2:4 Succenturiate Placenta

B) Abnormalities in Size and Weight:

Increase in size and weight in syphilis (syphilitic placenta is not edematous), hydrops fetalis and Gest. Diabetes. Placenta > 600g in wt, and > 5cm thickness on US.

C) Other Abnormalities:

1. Abnormal adhesion of the placenta to the myometrium (placenta accreta, increta and percreta).
2. Abnormal encroachment on the lower uterine segment: placenta previa.
3. Placental calcification: Normally calcium deposit increase as placental age advance and is correlated to placental functions.
4. Placental infarcts:
   a. White infarcts: Fibrin deposition on degenerated chorionic villi. Normal placenta may contain white infarcts and in some of them, calcium deposition may occur.
   b. Red infarcts: Caused by hemorrhage from the maternal vessels of the decidua. They occur more in hypertensive conditions.

THE UMBILICAL CORD (funis)

The Umbilical cord is developed from the ventral (connecting) stalk. At term, it measures about 50cm in length and 1-2 cm in diameter.

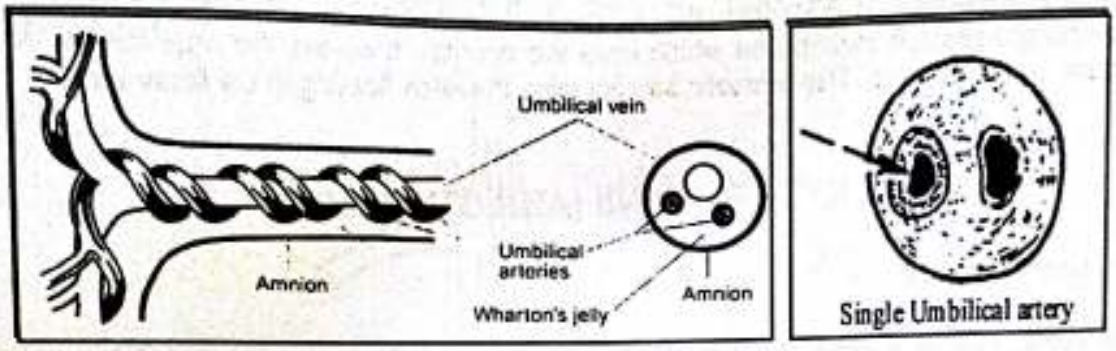


Fig 2:5 The umbilical Cord

The umbilical cord consists of:

1. Amniotic membrane coat.
2. Extraembryonic mesodermal tissue (Wharton's jelly).
3. One umbilical vein carrying oxygenated blood to the fetus and 2 umbilical arteries carrying the deoxygenated blood from the fetus to the placenta.
4. The obliterated coelom (if persistent, it leads to umbilical hernia).
5. Remnants of the yolk sac (vitelline sac) and the obliterated vitello-intestinal duct (if persistent it leads to the formation of Meckel diverticulum).
6. Remnants of the allantois.

ABNORMALITIES OF THE UMBILICAL CORD

1. Abnormalities of insertion:
   a. Marginal insertion in the placenta (battledore), occurs in nearly 8% of pregnancies.
   b. Insertion In the membranes (velamentous insertion), occurs in nearly 2% of pregnancies.

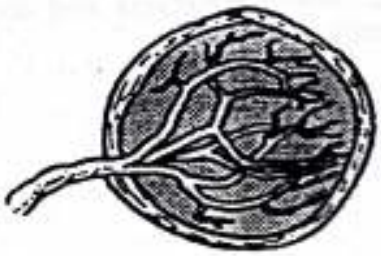


Fig 2:6 Marginal insertion

2. Knots of the cord:
   a. False Knots: Localized collection of Wharton's jelly containing loop of umbilical vessels.
   b. True Knots (1%): Occurs when the fetus passes through a loop formed by abnormally long cord. If tight, asphyxia results from interference of circulation.

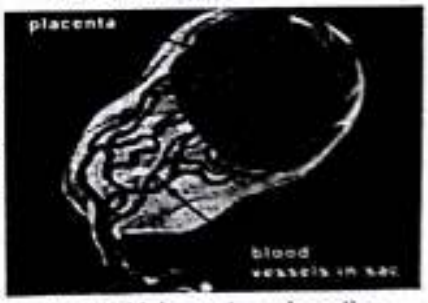


Fig 2:7 Velamentous insertion with vessels connectting to placenta

3. Abnormal length:
   a. Too long (> 60 cm): Predispose to cord prolapse, true knots and coiling loops of the cord around the neck of the fetus.
   b. Too short (< 35 cm): this may be true or apparent (due to coiling around neck). It predisposes to; intrapartum hemorrhage due to premature separation of the placenta, prolonged 2nd stage of labor due to delay descent, and inversion of the uterus.

True knot False knot

4. Vasa previa:

When placental vessels are below the presenting part. This occurs in cases of velamentous insertion of the cord specially when the placenta is low lying. This condition increases the risk of rupture leading to dangerous fetal hemorrhage.

THE FETAL MEMBRANES

1. The chorion:

Is the outer of the two membranes. Its surface is in contact with the uterine wall. It ends at the margin of the placenta to which it is attached. It is less transparent than the amnion.

2. The amnion:

Is a transparent grayish membrane which lines the chorion. It covers the fetal surface of the placenta and the umbilical cord. The amniotic sac contains the fetus floating in the liquor amnii.

THE LIQUOR AMNII (AMNIOTIC FLUID)

Constitution and Contents:

- The amniotic fluid is a clear, pale, slightly alkaline fluid, with a steadily increasing volume throughout pregnnacy, raeching about about one liter at full term (from 0.5 to 1.5). Both the fetus and placenta share in its formation and regulation.
- The liquor amnii is mainly composed of water (98-99%) and it contains albumin, sodium chloride, small amounts of sugars, urea, uric acid, creatinine, ammonia, enzymes and hormones and suspended in it are lanugo hairs, vernix caseosa, and epithelial cells.
- The liquor amnii is not static but there is a continuous turn over of water and sodium, with a balance between production and absorption that keeps the volume within normal ranges for gestational age.

Formation, Functions and abnormalities of amniotic fluid (see chapter 44, for amniotic fluid disorders).

FETAL CIRCULATION

- **One umbilical vein** carries ***oxygenated blood*** from the placenta to the fetus. It divides into a branch joining the *portal vein* and another joining the *inferior vena cava* to the *right atrium* (ductus venosus).

  Most blood of the right atrium passes through the *foramen ovale* to the *left atrium*, *left ventricle* to the *aorta*. Some blood of the right atrium passes to the right ventricle to the pulmonary artery, but most of this blood is directed to the aorta through the ductus arteriosus.
- **Two umbilical arteries** (branches of internal iliac "hypogastric artery") carry ***deoxygenated blood*** from the fetus to the placenta.

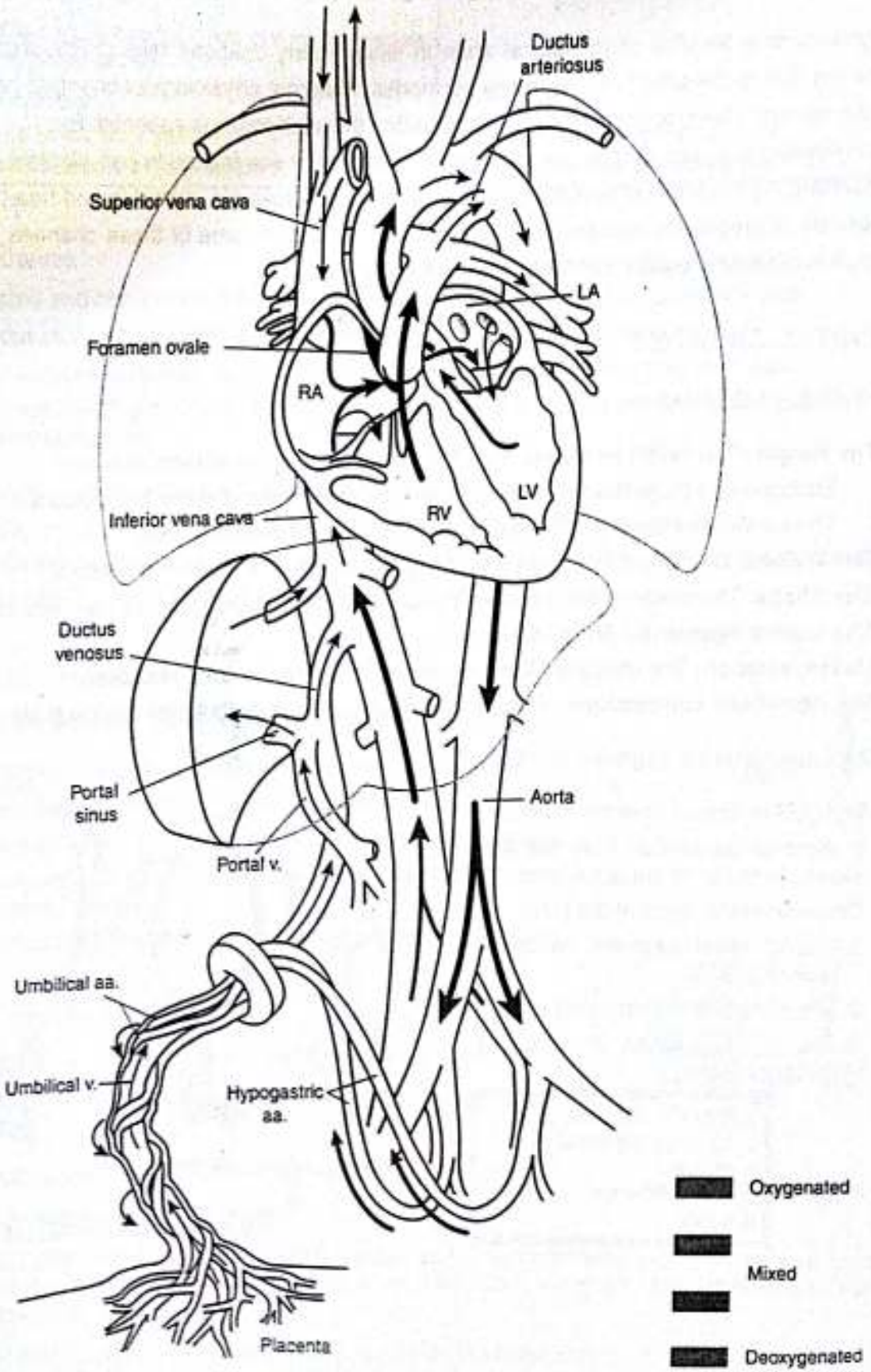
After birth, the following changes occur:

a. *Due to ligation of the umbilical cord*, the large volume of blood returning to the fetus through the umbilical vein stops, hence venous pressure falls in the inferior vena cava and this causes the ductus venosus to close and becomes the ligamentum venosum later on.

b. *Neonatal respiration* creates a negative intrathoracic pressure which opens the pulmonary capillary bed. This results in falling of the pressure in the right atrium while that in the left atrium is increased due to increased amount of blood returning from the pulmonary veins, thus causing functional closure of the foramen ovale (closes anatomically after 1 year).

c. *With diversion of most of the blood into the lungs* and increased oxygen concentration, the ductus arteriosus contracts (prostaglandin mediated) and becomes obliterated to form the ligamentum arteriosum later on.

d. *The umbilical vein is obliterated* to form the ligamentum teres and the *distal ends of the fetal hypogastric arteries are obliterated* to form the hypogastric ligaments (lateral umbilical ligaments).



3 MATERNAL CHANGES DURING PREGNANCY

Pregnancy is a peculiar physiological state in which many changes take place; most of these changes are due to the effect of pregnancy hormones. Maternal physiological changes help adaptation of the woman's body to pregnancy. Understanding these changes is essential for:

- Discriminating between normal pregnancy symptoms and those related to pathological conditions.
- Understanding the effect of pregnancy on pre-existing diseases e.g. diabetes and heart diseases.
- Diagnosis of pregnancy was previously dependent on finding some of these changes, before the evolution of ultrasonography and accurate pregnancy tests.

1. GENITAL CHANGES

A) *The Body of the Uterus:*

- **The Height** : The height increases from 7.5 cm to reach 35 cm at term, due to:
  - Estrogen and progesterone leading to myometrial hypertrophy and hyperplasia.
  - The contents of the uterus i.e. fetus, placenta and amniotic fluid ..etc.
- **The Weight:** The weight increases from 50 grams to reach 1000 grams at term.
- **The Shape:** The shape of the uterus is globular till 14 weeks then it becomes pyriform.
- **The uterine ligaments:** Show hypertrophy.
- **Dextro-rotation:** The uterus is tilted and twisted to the right in 80% of cases.
- **Braxton-Hicks contractions:** Irregular, painless contractions that help placental circulation.

B) *The Lower Uterine Segment (LUS):*

- The LUS is formed from the isthmus
- It starts to be formed from the 4th month, to reach 10 cm at full term.
- Obstetric significance of the LUS:
  1. Site of lower segment cesarean section (LSCS)
  2. Site of rupture in obstructed labor.
  3. Site of implantation in cases of placenta previa.

a. anatomical internal os
b. histological internal os
c. isthmus
c'. former isthmus
d. cervix

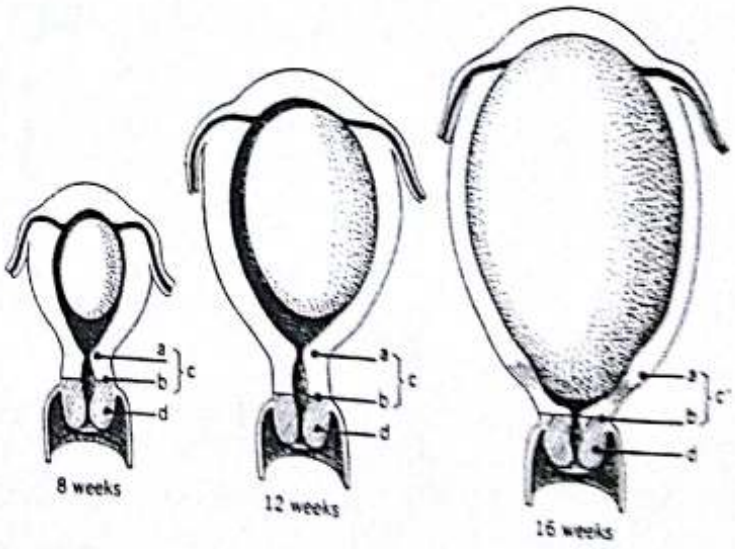


Fig 3:1 Formation of the LUS

Differences between the Upper and Lower Uterine Segments:

Upper uterine segment (UUS)	Lower uterine segment (LUS)
▪ During labor, it is active i.e. it contracts and retracts to become shorter & thicker. ▪ Thick wall: It consists of 3 muscle layers; - Outer longitudinal. - Middle oblique forming figure of 8 for hemostasis at the placental bed. - Inner circular. ▪ Covered by adherent peritoneum. ▪ The membranes are firmly attached.	▪ During labor, it's passive i.e. it dilates and stretches to be thinner and longer ▪ Thin wall: The oblique layer is defective so the LUS shows less retraction. ▪ Covered by loose peritoneum. ▪ The membranes are loosely attached.

C) The Cervix:

- Edema and congestion; it becomes soft "Goodell sign" and bluish "Chadwick sign"
- Mucus plug, which consists of the cervical mucus closing the cervical canal.
- Hormonal erosion may occur, it disappears spontaneously 3-6 months after labor.
- Cervical ripening mainly at the end of pregnancy due to edema and decreased collagen caused by prostaglandins.

D) The Vulva: shows increased vascularity and varicosities may develop.

E) The Vagina: shows increased vascularity which makes it soft, moist and bluish

F) The Ovary: shows increased vascularity and increased size. One of the ovaries contains the corpus luteum, which degenerates after the 10th week.

2. BREAST CHANGES

Due to the effect of oestrogen (E) and progesterone (P)

- Increased size and vascularity (warm, tense and tender).
- Increased pigmentation of the nipple and areola.
- 2ry areola appears (light pigmentation around the 1ry areola
- Montegomery tubercles appear on the areola (dilated sebaceous glands).
- Colostrum-like fluid is expressed at the end of the 4th month

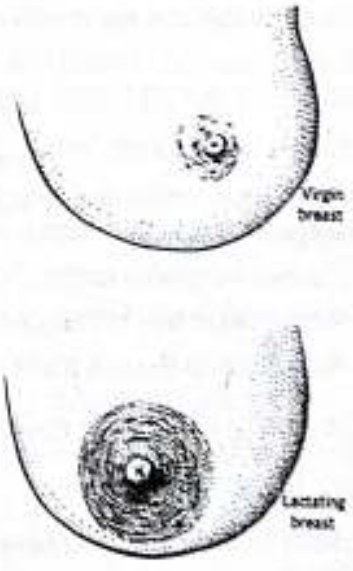


Fig 3:2 Breast changes

3. SKIN CHANGES

A. Pigmentation: Melanocyte Stimulating Hormone MSH + E:

- Linea nigra: Pigmentation of the linea alba, more marked below the umbilicus.
- Chloasma gravidarum: Butterfly pigmentation of the face

B. Striae gravidarum "stretch marks":

These are pink lines in the flanks due to rupture of the subcutaneous elastic fibers as a result of stretch of the abdominal wall during pregnancy. After labor, the color turns white due to fibrosis "striae albicans".

C. Loss of hair: is a common complaint that occurs during pregnancy.

4. METABOLIC CHANGES

A. Protein metabolism: (anabolic state)

Tendency to *nitrogen retention*, with *increased binding globulins* produced by the liver.

B. Carbohydrates metabolism: (diabetogenic state)

- *Progressive increase in insulin requirements* due to insulin antagonism and resistance by;
  1. Placental hormones (human placental lactogen HPL, oestrogen E, and progesterone P).
  2. Production of insulinase enzyme by the placenta
- *Alimentary glycosuria* due to rapid absorption of glucose from the GIT.
- *Renal glycosuria* due to lowering of the renal threshold (increase loss of glucose in urine).
- *Lowered fasting blood glucose* due to tansfer of glucose to the fetus (fasting hypoglycaemia).

C. Fat metabolism: Increased lipolysis to deliver more free fatty aids to the foetus

D. Minerals: Increased requirements of iron, calcium, phosphorus and iodides.

E. Water: Salt and water retention occurs due to the effect of estrogen and progesterone.

F. Weight: The average total weight gain is 9-14 kg, most of it occurs in the 3rd trimester.

5. URINARY CHANGES

A. Dilatation of the ureters: due to:

- *Atony* of the ureteric muscles caused by progesterone and relaxin.
- *Hypertrophy* of the lower end of the ureter caused by estrogen together with pressure of the uterus on the ureter. This efefct is more on the right ureter due to dextro-rotation of the uterus.
- Ureteric atony and pressure of gravid uterus lead to *stasis* of urine that predispose to *pyelitis*.

B. Frequency of micturition: due to pressure of the pregnant uterus on the urinary bladder in the 1st trimester and again with engagement of the head late in pregnancy in last few weeks.

6. GASTRO-INTESTINAL CHANGES

- Emesis gravidarum "morning sickness", and sometimes with increased salivation (ptyalism).
- Heart burn "reflux oesophagitis" caused by relaxation of the cardiac sphincter due to the effect of progesterone and relaxin.
- Decreased gastric acidity (HCl) which, may interfere with iron absorption, leadinhg to anaemia.
- Constipation due to reduced gut motility caused by progesterone.
- Relaxation of the gall bladder with a tendency to stone formation.

7. CARDIOVASCULAR CHANGES

A. The Blood Volume:

- Total blood volume increases almost 40% by the end of 32nd week of pregnancy.
- Plasma volume incresaes by 45% while RBCs increase only 15% leading to haemodilution.
- Haemodilution causes physiological anemia and hyperdynamic circulation (functional murmurs).

B. The Cardiac Output (CO):

- The CO increases to a peak at the 32nd week of pregnancy (about 40% increase) which remains elevated till the end of pregnancy. Increased CO is due to an both increased stroke volume (the main reason) and incrseased in the heart rate (15%) towards the end of pregnancy.

C. The blood pressure (BP):

- A normal BP in general has a mean of **120/80**. During pregnancy a **slight drop** in the BP occurs in the 2nd trimester due to opening of arterio-venous shunts at the placenta.
- Hypertension (HTN) is diagnosed in pregnancy when the BP is **140/90** or more, on two occasions, 6 hours or more apart.
- Supine hypotension syndrome: In the 2nd half of pregnancy, maternal hypotension occurs in the supine position due to pressure of the pregnant uterus on the inferior vena cava.This leads to decreased venous return and cardiac output.

D. Cardiac changes:

1. Dispalcement of the apex of heart In late pregnancy, to the 4th intercostal space, due to elevation of the diaphragm and the heart by the fundus of the growing uterus.
2. Functional murmurs are detected and are usually systolic.

E. Venous stasis:

Pressure of the uterus on the pelvic veins leads to ankle edema, varicose veins and piles.

F. Blood changes:

Increased fibrinogen (up to 600 mg%) and WBCs (leucocytosis up to 12 000/mm^3).

8. RESPIRATORY CHANGES

- Hyperventilation occurs, as progesterone stimulates the respiratory center.
- Limitation of movement of the diaphragm in late pregnancy causes dyspnea.

9. SKELETAL CHANGES

- Increased lumbar lordosis may cause low back pain.
- Relaxation of pelvic joints and ligaments due to progesterone and relaxin may cause pelvic pains.

10. ENDOCRINAL CHANGES

- ***The Pituitary gland:*** The anterior pituitary increases in size and activity, while the posterior pituitary releases oxytocin at the onset of labor.
- ***The Thyroid gland:*** increases in size and activity (physiological goiter). Total T4 is increased but the free T4 is within normal levels.
- ***The Parathyroid gland:*** increases in size and activity to regulate calcium metabolism.
- ***The Adrenal gland:*** increases in size and activity. Cortisol is increased but the main increase is in the bound portion.

4 DIAGNOSIS OF PREGNANCY

Symptoms of Pregnancy:
- *First trimester*
- *Second trimester*

Signs on Clinical Examination:
- *First trimester*
- *Second trimester*

Laboratory Diagnosis
- *Urine pregnancy test*
- *Serum B-hCG*

Ultrasound Diagnosis
- *TAS*
- *TVS*

The diagnosis of pregnancy is one of few important events in a woman's life that should be diagnosed accurately and as early as possible. Diagnosis of pregnancy is based on the triad of:

1. Delayed cycle (missed period)
2. Positive Pregnancy test (urine or serum)
3. Pelvic ultrasonography

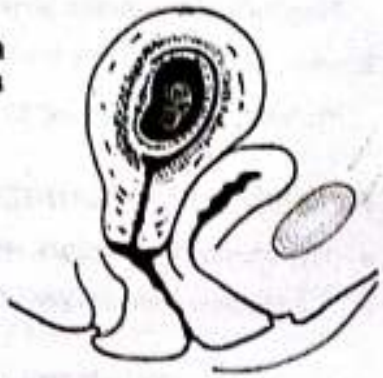


Fig 4:1 Intrauterine pregnancy

SYMPTOMS IN THE FIRST TRIMESTER

Symptoms of pregnancy in the first trimester are only presumptive and are never sure.

1. **Cessation of menses:**

   A missed period is the earliest symptom of pregnancy. It is due to persistent corpus luteum function, under the effect of human chorionic gonadotropin (hCG) produced by the proliferating trophoblastic cells, with consequent continuous production of both estrogen E and progesterone P.

2. **Nausea, with or without vomiting:**

   Commonly occurs between 6-12 weeks, especially in a primigravida, probably related to exposure to increasing levels of hCG. It is variable from one patient to another, and in severe conditions may require hospitalization and I.V. therapy (hyperemesis gravidarum).

3. **Urinary symptoms:**

   Although non specific to pregnancy, yet frequency of micturition is a common early pregnancy complaint. It is due to pressure by the enlarging gravid uterus, and associated pelvic congestion.

4. **Fatigue, weakness and lack of concentration:** Are all common symptoms in the first trimester, but are usually insignificant and nonspecific.

5. **Breast symptoms:** The breast size is usually increased with associated tenderness. Later on dark pigmentation of the nipple and areola will occur, due to increased deposition of melanin pigment in the skin.

SYMPTOMS IN THE SECOND TRIMESTER

Symptoms in the 2nd trimester are presumptive until perception of foetal movements starts

1. Abdominal enlargement: which is gradually progressive.
2. Maternal perception of fetal movements.
   - Quickening is the first perception of fetal movements by the mother.
   - It starts between 16-18 weeks in the multigravida (slightly later in a primigravida).
   - It could be misinterpreted with intestinal and colonic bowel movements.
3. Maternal visualization of fetal movements:

   Vigorous fetal kicks can be felt and seen by the mother as sudden changes in her abdominal contour, sometimes associated with mild pain.

SIGNS IN THE FIRST TRIMESTER

Signs elicited in the first trimester are only presumptive, and sometimes confusing.

Vaginal examination is poorly diagnostic, besides being inconvenient to many pregnant women. Laboratory pregnancy tests and US had largely replaced clinical examination in the first trimester

1. **The vagina:** Becomes congested giving a bluish or purple coloration *(Chadwick's sign)*.
2. **The cervix:** Becomes gradually softer in consistency as pregnancy advances.
3. **The uterus:** Bimanual examination if performed will reveal
   - The uterus becomes soft cystic in consistency, symmetrical enlarged in size, with the fundus of the uterus reaching the symphysis pubis by 12 weeks gestation.
   - ***Hegar's sign:*** where 2 fingers introduced to the anterior vaginal fornix could be approximated to the abdominal hand posterior to the isthmus due to the soft myometrium
4. **Breast signs:** more pronounced in primigravidas *(see maternal changes during pregnancy)*
   - Increased pigmentation of the nipple and areola,
   - Appearance of the secondary areola (light discoloration around the 1ry areola).
   - Montgomery's tubercles on the areola (dilated sebaceous glands).

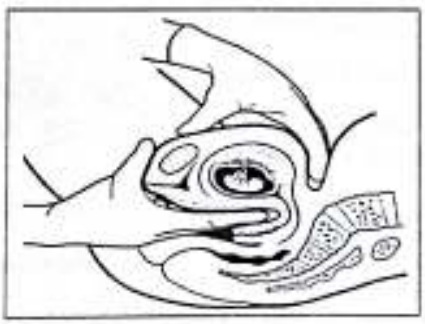


Fig 4:2 Hegar's sign

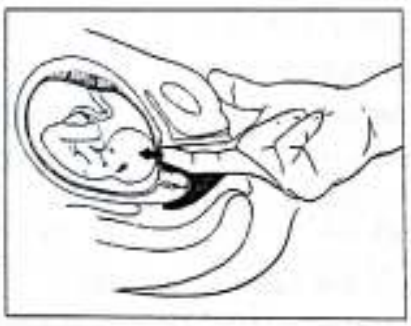


Fig 4:3 Internal ballottment

SIGNS IN THE SECOND TRIMESTER

Signs of pregnancy in the second trimester are considered as sure evidence of pregnancy.

1. Palpation of fetal parts on abdominal examination (see chapter 5).
2. Palpation of fetal movements on abdominal examination.
3. Auscultation of FHS or umbilical artery soufflé, by Sonicaid duplex instruments.
4. Palpation of fetal parts on vaginal examination through the vaginal fornix (internal ballottment).
5. Visualization of fetal echoes by ultrasound or skeleton by x ray.

LABORATORY PREGNANCY TESTS

Laboratory tests for diagnosis of pregnancy are based on the detection of human chorionic gonadotropin (hCG) in the urine or blood samples of pregnant women.

1. Urine pregnancy tests:

Urine pregnancy tests are simple, cheap and reasonably accurate. A positive result will be detectable in the first few days of a missed period, within 5-10 minutes from performing the test.

A) ***ELISA Pregnancy Slide Tests***: *(Enzyme Linked Immuno-Absorbent Assay)* can detect pregnancy starting from 48 hours delay in the cycle (5th week pregnancy).

B) ***Immunologic Pregnancy Tests:*** Detect pregnancy by an antigen antibody reaction using latex particles, positive after one week delay in the cycle (6th week pregnancy).

2. Serum B-HCG tests:

- Detection of the B-hCG fraction in sera of pregnant women is the earliest available method for diagnosis of pregnancy. It is a quantitative test, being negative at levels 0-5 m.IU/ml.
- The test is **positive** at the first day of the missed period with levels > 50 m.IU/ml (i.e. 2 weeks after ovulation and fertilization).
- B-hCG levels rise sharply in the first 8 weeks of pregnancy, nearly doubling every 48 hours, reaching > 800 m.IU/ml. by end of the 5th week , and > 10.000 m.IU/ml. by end of the 6th week.
- B-hCG may also serve as an early predictor of abnormal pregnancies where poorly rising levels may be the first indicator of a pregnancy complication, as abortion or ectopic pregnancy. Abnormally highly rising levels are associated with vesicular mole and choriocarcinoma.

N.B.: *Serum B-HCG test is superior to urine pregnancy test in the early diagnosis of normal pregnancies, and in the follow up of abnormal pregnancies.*

ULTRASOUND DIAGNOSIS OF PREGNANCY

Both Transabdominal ultrasonography (TAS), and transvaginal ultrasonography (TVS), are excellent tools in the early diagnosis of intact pregnancies and pregnancy complications. Both TAS and TVS are considered safe, cheap, readily available and easy to interpret.

Ultrasonography in the first trimester:

- 5-6 week : Gestational sac < 2cm
- 7-9 week : Embryo with pulsations.
- 10-13 week: Fetus with limb and trunk movements

In the first trimester measurements of the gestational sac (GS) size and fetal crown to rump length (CRL) give an accurate estimation of the true gestational age, and the expected delivery date (EDD).

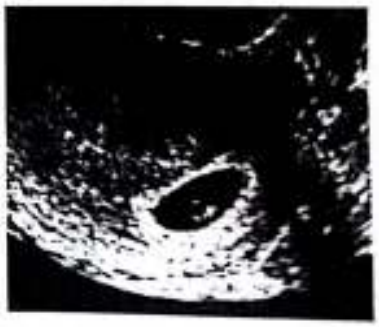


Fig 4:4 G.S. 6 weeks

Ultrasonography in the second trimester:

Combined measurements of the fetal biparietal diameter (BPD), abdominal circumference (AC) and femur length (FL), gives an accurate estimation of the gestational age, and the expected delivery date (EDD).

Calculation of the EDD (Naegle's formula)

- Pregnancy duration is 280 days from first day of LMP.
- Pregnancy duration = 9 month + 1 week.
- Calculation of EDD: LMP + 9 months + 7 days.

  e.g.: If LMP at **01/01/2010**, then EDD will be at **08/10/2010**.

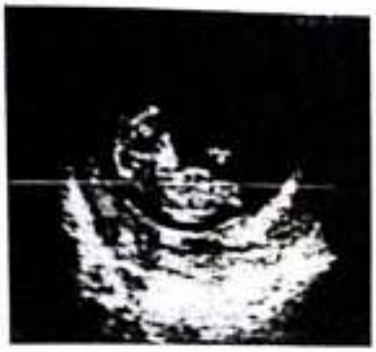


Fig 4:5 Foetus 10 weeks

5 ANTENATAL CARE AND HIGH RISK PREGNANCY

Antenatal Care ANC
- *Objectives of ANC*
- *The Preconception visit*
- *The first ANC visits*
- *Return ANC visits*
- *Instructions to the patient*
- *Nutrition during pregnancy*
- *Vaccination during pregnancy*
- *Drug Intake during pregnancy*
- *Common Complaints during pregnancy*
- *Postnatal care*

The High Risk Pregnancy
- *Identification of high risk cases*
- *Preconception Counseling*
- *Risk score system*
- *Screening for foetal anomalies*
- *Foetal surveillance during pregnancy*
- *Delivery of high risk patients*
- *The elderly primigravida*
- *The grand multipara*
- *Maternal Mortality*
- *Foetal and Neonatal Mortality*

Although pregnancy is considered a normal physiologic event, yet it can be complicated by pathologic processes dangerous to the mother and foetus in about 5-20% of cases. Some of these complications are preventable, others are predictable, allowing early diagnosis and management.

Antenatal care (ANC) is a program of preventive obstetrics, with a main objective to ensure a safe motherhood, culminating in a safe delivery of a healthy foetus.

OBJECTIVES OF ANC

1. Early detection and, possibly prevention, of complications specific to pregnancy, as preeclampsia, eclampsia, and obstetric haemorrhage.
2. Detection and management, or at least amelioration, of any medical disorder complicating pregnancy as anaemia, diabetes mellitus, cardiac, renal, or endocrine disorders.
3. Detection of complications which may affect labour as disproportion and malpresentations.
4. Education of the patient and her family about pregnancy, labour and delivery, the hygiene and diet in pregnancy, and the warning or alarming symptoms that necessitate consultation.
5. Laboratory investigations that may assure the general health and detect medical problems.
6. Finally classification of patients into normal or high risk to put the plan of proper management.

THE PRECONCEPTION VISIT

The ideal first visit should be at a preconception clinic where health education and risk assessment can be directed towards the planned pregnancy.

Advice can be given regarding the avoidance of harmful and teratogenic factors (drugs, cigarette smoking and alcohol intake...), ensuring an optimal dietary intake, and absence or control of chronic medical disorders (as diabetes, hypertension, etc...), in order to allow pregnancy to be started in the optimum conditions.

THE FIRST ANC VISIT

- **Aim:** Thorough history taking and clinical examination to **identify important risk factors:**

- History:
  - Menstrual History: To identify LMP, calculate gestational age, and the EDD (Naegle's formula).
  - Obstetric History: Previous pregnancies provide important clues to potential problems in the current one.
  - Medical History: Medical disorders exacerbated by pregnancy e.g. hypertension, diabetes, heart disease…etc.
  - Surgical History: e.g. uterine surgery as myomectomy, previous CS.
  - Family History: e.g. diabetes, twins, familial disorders.

- **General examination:** Pulse, temperature and B.P., pallor…etc.

- **Abdominal Examination**: abdominal masses, enlarged liver or spleen, hernias,…etc

- **Fundal level assessment** : in relation to pregnancy duration

- **Vaginal examination:** done only if necessary, e.g.: suspected pelvic masses, ectopic pregnancy, etc...

- **Routine laboratory tests:**
  - Blood group and Rh typing, to identify RH negative patients.
  - Complete blood picture: for Hb%, WBCs, and platelets.
  - Blood sugar level: random blood glucose, or fasting and 2 hrs postprandial levels.
  - Complete urine analysis: for character, PH, albumin, sugar, pus cells, RBCs, epithelial cells, etc...
  - Other tests as: TORCH antibodies IgG and IgM, VDRL, hepatitis B and C if necessary, especially in the first pregnancy.

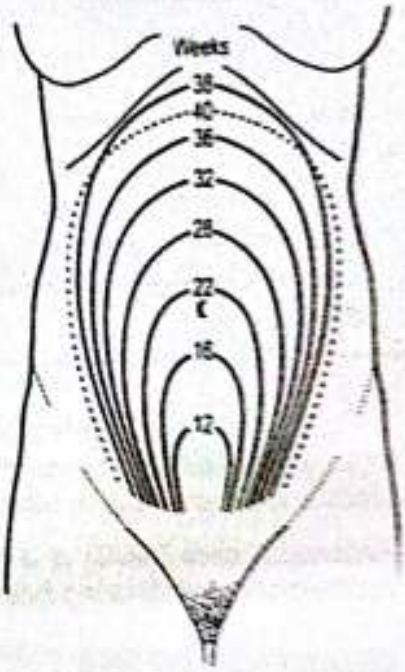


Fig 5:1 Fundal level

RETURN ANC VISITS

Monthly visits are required in the first 6 months, twice weekly visits in 7th and 8th months, then weekly visits in the 9th month until delivery. In each visit important data include;

- Warning symptoms, e.g. Bleeding or regular menstrual like colicky pains, persistent vomiting, sudden escape of liquor amnii, severe persistent headache, blurring of vision, marked swelling of the lower limbs.
- Weight gain: the average weight gain during pregnancy is 11-16 Kg. Excessive weight gain many denote occult oedema (developing preeclampsia) while inadequate weight gain may reflect nutritional deficit or foetal growth retardation).
- Examination for L.L. Oedema: ankle oedema is acceptable in late 2nd and 3rd trimesters.
- Blood pressure measurements: to detect early gestational hypertension or preeclampsia.
- Fundal level: Should be measured and recorded at each visit after 20 weeks.
- Foetal heart sounds, heard by the Sonicaid Duplex instrument, or by Pinnard stethoscope.
- Ultrasonography whenever needed to ensure gestational age, to assure normal foetal growth, to assess foetal well being, to exclude major foetal anomalies, to evaluate placental location and amniotic fluid volume at various pregnancy trimesters.

INSTRUCTIONS TO THE PATIENT

- **Exercise:** Mild to moderate exercise, as walking, and regular daily house work are allowed.
- **Sleep and rest:** Proper night sleep (8 hrs), and adequate periods of rest are advisable.
- **Care of teeth:** To avoid dental caries caused by increased acidity, and septic foci.
- **Bowel habit:** Avoiding constipation by fresh vegetables and mild laxatives if necessary.
- **Clothes:** Avoid tight and too heavy uncomfortable clothing.
- **Breasts:** Regular hygiene by daily washes throughout pregnancy. In the last few weeks, massage of the nipples using lubricant creams might reduce the incidence of cracking. Retracted nipple is withdrawn by the thumb and finger using a lubricant.
- **Sexual intercourse:** Is better minimized in the first trimester to avoid bleeding then gradually allowed. It is completely restricted only if there is recurrent bleeding, threatened abortion, threatened preterm labour, or suspected rupture of the membranes.
- **Smoking:** Should be strictly avoided as it may result in placental insufficiency, delivery of small babies, or may be a cause of premature labour.
- **Travelling:** Only comfortable travelling may be allowed. However, travelling should be avoided in the last month and it is completely prevented in patients with a history of bleeding, threatened abortion, habitual abortion, or premature labour.

NUTRITION IN PREGNANCY

Nutritional Requirements of the pregnant mother should include:

- Caloric requirements average 2300 Kcal/day.
- Protein: 80-100 gm/day, Calcium: 1-1.5 gm/day, Iron: 30-60 mg/day.
- Vitamins and minerals: Especially B, C, D, K
- Folic acid is important for cell division and replication. In the first few weeks, a dose of 400 ug/day has been shown to effectively reduce the risk of neural tube defects.
- Salt restriction, is advisable in cases with marked oedema or tendency to hypertension.

A suitable daily diet in pregnancy should thus include: 400 ml. of milk or its derivatives, one egg, fresh fruits and vegetables about 120 gm of red meat, fish, or liver.

Effect of severe Malnutrition on the mother:

1. Loss of weight, anaemia, and lowered resistance against infection.
2. Decalcification of bones, caries of teeth.
3. Affection of lactation.

Effect of Malnutrition on the foetus:

1. Low birth weight infants.
2. Higher incidence of rickets and anaemia, in severe cases.

Vaccination (immunization) in pregnancy:

- Live attenuated vaccines are contraindicated.
- The vaccines for the following diseases may be given if needed, preferably after the 1st trimester:
  - Tetanus, poliomyelitis, rabies, influenza, cholera and typhoid.
  - Passive immunization against hepatitis A and B may be given.

DRUG INTAKE DURING PREGNANCY

Drug categories during pregnnacy according to FDA classification:

- Group: A Safe during pregnancy
- Group: B Risky in animal, no enough data on humans.
- Group: C Risk in human cannot be ruled out.
- Group: D Risky in human pregnancy, but the benefits may outweigh the risks.
- Group: X Contraindicated in pregnancy, may cause adverse fetal effects.

COMMON COMPLAINTS DURING PREGNANCY

- ***Morning sickness:*** (see: vomiting in pregnancy).

A sensation of nausea, with or without vomiting, which may be more evident in the morning, is common especially in primigravidas. *Management by* reassurance, frequent small light meals, vitamin B6, and if severe certain antiemetic drugs may be given for a short period of time.

- ***Heart Burn:*** dilatation of the cardiac opening of the stomach and oesophageal regurgitation, commonly lead to a sensation of heart burn. Less commonly the cause is some degree of a hiatus hernia.

Management: Frequent light diet, antacids, and allowing 2 hours between meals and sleep.

- ***Constipation:*** due to reduced intestinal motility due to steroid hormones, with continued fluid absorption and pressure by the gravid uterus.

Management includes increased fluid intake, regulation of bowel habits, diet should be rich in fresh vegetables. Mild laxatives may be required.

- ***Haemorrhoids (Piles):*** haemorrhoids are predisposed to by congenital weakness of the walls of the veins, constipation, straining, and prolonged standing.

Management: use local anaesthetic ointment as lignocaine and avoid constipation.

- *Headache:* is one of the commonest complaints, especially those with history of migraine attacks. The condition is aggravated by vasodilatation accompanying pregnancy. It may also be due to nasal congestion or chronic sinusitis, errors of refraction or emotional tension.

Management: In most cases *symptomatic treatment* by use of Paracetamol derivatives. It is important to note that severe and persistent headache in the 3rd trimester may be suggestive of PE.

- *Breast tenderness:* caused by breast engorgement and *managed by* avoiding tight clothes.
- *Breathlessness:* commonly noticed as early as the first 12th week of pregnancy, due to hyperventilation caused by progesterone. In late weeks, the enlarging uterus can cause mechanical pressure. Reassurance of the patient is usually enough in managing the problem
- *Abdominal pain:*

1. Pelvic heaviness or sensation of dragging caused by the weight of the uterus on the pelvic support and the abdominal wall. *Management*: Rest especially in the lateral position.
2. Traction on the round ligament with slight rotation of the uterus can cause abdominal discomfort along the course of the ligament. *Management*: Reassurance, change of position.
3. Braxton-Hicks contractions: infrequent, irregular and not increasing in frequency or strength. *Management:* Reassurance, and if recurrent or severe, mild sedatives or antispasmodics.
4. Flatulence and distension: may be caused by large, fatty meals or intestinal hypotonia, constipation and pressure by the enlarging gravid uterus. *Management:* avoiding large, fatty meals.

- *Urinary symptoms:* frequency, urgency and stress incontinence are quite common in late pregnancy which may be explained by increased intra-abdominal pressure together with pressure on the bladder by enlarging uterus. The most important *management* is to exclude urinary tract infection.
- *Lower limb and Ankle oedema:* Is common in late pregnancy.
  - **Physiological:** due to salt and water retention caused by ovarian, adrenal and placental steroid hormones, pressure of the uterus on the pelvic veins and prolonged sitting or standing.

    Management: minimize long sitting and standing, elevation of legs whenever possible, and mild exercise. Reduction of salt intake, does not usually affect the condition.
  - **Pathological:** (See differential diagnosis of preeclampsia).
- *Leg Cramps:* Transient nocturnal painful cramps, mostly due to accumulation of lactic acid with poor venous drainage due to pressure of gravid uterus and LL oedema. Less commonly it may be attributed to reduced serum calcium or magnesium or elevated serum phosphorus.

  Management includes massage to leg muscles, calcium and magnesium supplementation. Aluminium hydroxide may be given to reduce phosphorus absorption.
- *Varicose veins (V.V):* Is predisposed to by congenital weakness of the wall of the veins (main cause), poor muscle activity, increased venous pressure, obesity and pregnancy induced vasodilatation.

  Management includes: avoiding long standing and sitting, active muscle exercise, elevation of the leg, control of weight gain, and elastic cotton stockings used while lying down and the veins are empty. Surgical or injection treatment is avoided during pregnancy.
- *Backache:* Backache is one of the commonest complaints during pregnancy. It may be explained by increased lumbar lordosis, and relaxation of the back muscles and pelvic joints caused by steroids.

  Management is by frequent bed rest to minimize lordosis, exercise, e.g., walking to maintain muscle strength, light massage to relax tense back muscle and avoiding high-heels.
- *Fatigue:* early in pregnancy, there may be a desire for excessive periods of sleep. In later trimesters fatigue may be explained by anaemia, extra-weight gain, breathlessness, or other systemic diseases.
- *Vaginal discharge (leucorrhoea):* due to excess oestrogen production. No treatment is required except if it is associated with infection by trichomonas, or Candida albicans.
- *Sweating and "feeling of heat":* hot flashes are common probably due to increased peripheral circulation and vasodilatation.

  Management includes frequent rest periods and cold showers and increased fluid intake.

THE PLACE OF DELIVERY

The place of delivery is chosen according to the risk assessment of the case performed by the end of the ANC program. High risk cases should be delivered at hospitals equipped for care of obstetric emergencies including C.S., blood transfusion, maternal ICU service, and neonatal ICU.

POSTNATAL CARE

The mother is still at risk for complications during the immediate & late postpartum period. The new born must be assessed and managed by a neonatologist. See later maternal care during puerperium.

HIGH RISK PREGNANCY

High Risk Pregnancy is a pregnancy complicated by a disease or a disorder that may endanger the life or affect the health of the mother, the fetus or the newborn.

Taking a thorough history and performing a physical examination are the best way to identify the high-risk pregnant women. Once identified, high risk pregnancies should be referred to a center specialized in maternal and fetal medicine.

High risk pregnancy may be associated with:

a) Severe medical conditions affecting the mother such as:
Diabetes Mellitus, cardiac disease grades III and IV, artificial heart valves, systemic lupus Erythematosus (SLE), and sickle cell disease.

b) Recurrent poor obstetrical outcomes such as:
Recurrent pregnancy loss (RPL), recurrent still birth (RSB), recurrent early rupture of membranes (ROM), and recurrent pre term labor (PTL).

c) Obstetrical complications that require specialized care such as:
Severe pre-eclampsia (PE) or eclampsia, HELLP syndrome, severe intrauterine fetal growth restriction (IUGR), and multiple high risk factors.

d) Conditions that may require invasive procedures for fetal diagnosis or therapy as:
Immune and non immune hydrops foetalis, and congenital anomalies or genetic disorders.

PRECONCEPTION COUNSELING:

The obstetrician discusses and explains the following items:

- The high risk factors) and its possible effects on the mother, fetus, and the newborn.
- The importance of proper monitoring during pregnancy and labor.
- The possibility of early intervention and the sequelae of pre term labor.
- Antenatal care in a well equipped antenatal clinic.
- The need to deliver in a well equipped hospital, with warning against home delivery.

CLASSIFICATION ACCORDING TO A RISK SCORING SYSTEM:

For each country or location, a specific risk score is developed to identify high risk cases and to evaluate the magnitude of risk. The scoring system determines the prevalence of risk factors together with the associated peri-natal mortality.

Identification of high risk cases during ANC occurs as follows;

A. Conditions detected during history taking:

- Age; whether young (< 18) or elderly (> 35) Primigravida.
- Parity; whether nullipara (primigravida), or grand multipara (> 4)
- Previous obstetric difficulties, fetal loss or abnormalities
- Medical disorders as; Diabetes mellitus, cardiac or renal disease

B. Conditions observed during general examination:

- Extreme obesity (maternal weight > 120 kg).
- Short stature (less than 150 cm)
- Hypertension (>140/90)
- Severe anemia (Hb <8.0 gm %)
- Cardiac or renal disease.
- Poor weight gain during pregnancy

C. Conditions diagnosed during obstetric examination

- Pre- eclampsia (PE)
- Ante partum hemorrhage (APH)
- Multiple pregnancy
- Malpresentations, and Feto-pelvic disproportion

D. Conditions detected during routine investigations

- Severe anemia, thrombocytopaenia, and hyperglycemia,
- Glycosuria and Albuminuria.
- Rh negative blood typing

SCREENING FOR FOETAL ANOMALIES

- Congenital anomalies: Ultrasonography for foetal anatomy survey for detection of (e.g. anencephaly, NTDs, Limb and skeletal deformities, cardiac and renal anomalies...etc).
- Chromosomal abnormalities, as Down's syndrome (by 1st trimester US, chorionic villus sampling, and 2nd trimester amniocentesis). See later chapter on prenatal diagnosis.

SCREENING FOR INFECTIONS

- TORCH, toxoplasmosis, rubella, cytomegalovirus, herpes simplex.
- Hepatitis B & C and Human Immunity Virus (HIV).

FETAL SURVEILLANCE IN HIGH RISK CASES

1. Correlation between fetal growth and gestational age. (Clinical & US).
2. Daily Fetal Movement Count (DFMC).
3. Non stress test (NST).
4. Contraction stress test (CST).
5. Biophysical profile score (BPPS).

DELIVERY OF HIGH RISK PATIENTS

1. Attention to the risks that may develop during labor and may affect maternal or fetal conditions.
2. The place of delivery should be fully equipped for maternal and foetal resuscitation (maternal and neonatal ICU).
3. Efficient well-trained personnel, specialists and consultants available 24 hours a day.
4. Monitoring of fetal well being during labor (FHR monitoring) and progress of labor (partogram) is essential.

THE ELDERLY PRIMIGRAVIDA

- **Definition:** Primigravida whose age is 35 years or more during pregnancy or delivery
- **Maternal and foetal risks:** increased liability to the following:

1. Medical disorders during pregnancy; as PE, Eclampsia, and GDM (see medical disorders)
2. Intrauterine growth restriction (IUGR), placental insufficiency, and preterm labour (PTL)
3. Increased incidence of chromosomal abnormalities (trisomy 21), and congenital malformations
4. Dysfunctional labour, abnormal uterine action, rigid perineum.
5. Increased rate of CS to reassure foetal safety, due to all previous conditions in addition to: pregnancy after infertility treatment as ovulation induction, IUI, or ICSI (precious pregnancy).

THE GRAND MULTIPARA

Definition: women who had five or more previous deliveries
Maternal and foetal risks: increased liability to the following:

1. Medical disorders as; Anaemia, chronic Hypertension, PE, GDM, etc..
2. Placenta praevia, and placenta accreta in cases of repeat CS
3. Malpresentations and malpositions; due to pendulous abdomen and weak abdominal muscles
4. Uterine inertia and more liability to atonic post partum haemorrhage (PPH)
5. Obstructed labour and rupture of the uterus; due to
   - Oversized babies with malposition and malpresentations
   - Osteomalacia affecting the bony pelvis
6. Increased operative delivery (CS, Forceps, and Ventouse) due to all previous factors

MATERNAL MORTALITY

- **Maternal Mortality:** refers to maternal deaths due to obstetrical causes (during pregnancy, delivery, or puerperium)
- **Maternal mortality rate (MMR):** describes the number of maternal deaths due to obstetric related causes per 100,000 deliveries per year
- **Incidence in Egypt:** 82/100,000 deliveries in year 2000 statistics
- **Common Causes of Maternal Mortality:**
  1. Postpartum Haemorrhage (PPH): 34%
  2. Preeclampsia (PE) and Eclampsia: 22%
  3. Antepartum Haemorrhage (APH): 9.0%
  4. Puerperal and post-abortive sepsis: 8.0%
  5. Rupture of the uterus: 8.0 %
  6. Complications of CS: 7.0%
  7. Other factors as: Pulmonary embolism and DIC, medical and cardiac problems, anaesthesia complications: 12%

- MMR are significantly increase in developing countries due to lack of education, low socioeconomic standards, poor medical services, unawareness of the importance of ANC, and lack of general hygiene.

FOETAL AND NEONATAL MORTALITY

- **Foetal death:** describes foetal death during pregnancy (intrauterine) or during delivery (intrapartum) i.e.; IUFD and IPFD
- ***Causes of IUFD:***
  1. Hypertensive disorders during pregnancy especially PE and eclampsia
  2. Diabetes during pregnancy especially GDM
  3. Placental insufficiency causing severe IUGR
  4. RH incompatibility in sensitized mothers
  5. Congenital foetal anomalies (incompatible with life)
  6. True knots of the cord or multiple tight loops around the neck
  7. Idiopathic causes with unexplained IUFD

- *Causes of IPFD:*
  1. Intrapartum asphyxia; due to severe CPD and obstructed labour
  2. Intracranial haemorrhage
  3. Intrapartum sepsis due to prolonged PROM
  4. Birth trauma using forceps or ventouse instruments

- **Neonatal death:** refers to infant death in the first 4 weeks after delivery, with the highest incidence in the first week after delivery
- *Causes of Neonatal death (NND)*
  1. Prematurity
  2. Neonatal asphyxia
  3. Birth injuries
  4. Congenital foetal anomalies
  5. Haemolytic and haemorrhagic diseases of the new born
  6. Respiratory distress syndrome (RDS)
  7. Neonatal sepsis and infection

6 PRENATAL DIAGNOSIS OF CONGENITAL ANOMALIES

Types of foetal anomalies

- Congenital
- Chromosomal

Value of prenatal diagnosis

Indications of prenatal diagnosis

Techniques used for Prenatal diagnosis

1. Maternal serum screening
2. Ultrasound diagnosis
3. Amniocentesis
4. Chorionic villus sampling (CVS)
5. Cordocentesis
6. Magnetic resonance imaging (MRI)

Screening for Down syndrome (trisomy 21)

- US NT
- US soft markers
- Double marker test (DMT)
- Triple marker test (TMT)

New prenatal diagnostic options

Pre-implantation genetic diagnosis (PGD)
Foetal cells in maternal circulation

Prenatal diagnosis entails in utero diagnosis of fetal congenital or chromosomal abnormalities

Some fetal anomalies are lethal, some may need surgical repair, and others do not interfere with fetal life, growth or functions.

Whenever the mother or fetus are at high risk for such abnormalities, screening tests are triggered as early as possible to avoid remote and late complications.

TYPES OF FETAL ANOMALIES

I. CONGENITAL ANOMALIES

A) Structural anomalies: Abnormalities or malformation in organ development: **e.g.;**

- Neural tube defects (NTDS) as anencephaly, spina bifida, hydrocephalus, encephalocoele.
- Abdominal wall defects (exomphalos) and congenital diaphragmatic hernia.
- Cardiac anomalies as atrial and ventricular septal defects (ASD & VSD),
- Renal anomalies as renal agenesis, unilateral or pelvic kidney, obstructive uropathy,
- Tumors as sacro-coccygeal teratoma, congenital goiter, ...
- Lymphatic channel obstruction as cystic hygromas, and many others,

B) Biochemical anomalies: are *due to enzyme deficiencies as:*

- Phenylketonuria, cystinuria, galactosemia and others.

II. CHROMOSOMAL ANOMALIES: e.g. include:

- Down syndrome (trisomy 21),
- Edward syndrome (trisomy 18)
- Pateau syndrome (trisomy 13), and others.....

VALUE OF PRENATAL DIAGNOSIS OF FETAL ANOMALIES

1. Counseling the parents about termination of pregnancy (only if anomaly is lethal) with identification of any possible maternal hazards.
2. Preparation for postnatal treatment of the new born shortly after delivery.
3. In utero fetal therapy in selected cases (if possible).

INDICATIONS OF PRENATAL DIAGNOSIS OF FETAL ANOMALIES

1. Maternal age more than 35 years
2. Administration of teratogenic drugs or chemicals, especially first trimester.
3. Exposure to teratogenic irradiation especially during early pregnancy.
4. Past history of previous affected fetus of the mother or very near family relatives
5. Obstetric history of 2 or more previous successive spontaneous abortion
6. One or both parents with a defined chromosomal anomaly.
7. Exposure to certain infections in the peri-conceptional period or during early pregnancy.

Maternal age above 35 years as a risk factor for a fetus with ***nondisjunction*** chromosome.

- ***Nondisjunction*** is an error in meiosis that results in a gamete with one chromosome too few, or too many. Fertilization of the gamete with one extra chromosome results in a conception with **47** chromosomes.
- ***Aneuploidy*** occurs when the chromosome number is not an exact multiple of 23. Most common aneuploids are trisomies 21, 18, and 13. Nondisjunction may involve sex chromosomes, therefore abnormalities such as 47 XXY, 47 XYY, also increase with advancing maternal age.

TECHNIQUES USED FOR PRENATAL DIAGNOSIS

1. MATERNAL SERUM SCREENING

A maternal serum blood sample, may allow testing for different markers (hCG, PAPP-A, AFP, and unconjugated E3), which, separately or in combination, may provide important information about the risks of having a child with possible chromosomal defects.

If these markers point to an abnormality it should be confirmed by US and/or amniocentesis or CVS, to reach an ultimate diagnosis.

1. *Maternal serum screening*
2. *US*
3. *Amniocentesis*
4. *CVS*
5. *Cordocentesis*
6. *MRI*

A) Alpha Fetoprotein (AFP):

It is a glycoprotein produced early by the yolk sac, then by the fetal liver and GIT. Normally it rises in the fetal blood peaking at 12 weeks, in the amniotic fluid peaking at 12 weeks, and in the maternal serum it rises peaking at 30 weeks. It is measured by multiple of medians MOMS, (normally from 0.5-2.5 MOMS). Maternal serum alpha fetoprotein (MSAFP) levels are used as markers for foetal birth defects and chromosomal anomalies as follows

- MSAFP is abnormally **increased** in open neural tube defects (NTDS), and turner syndrome.
- MSAFP shows abnormally **decreased** levels in trisomy 21 (Down syndrome), where it is best measured between 16-18 weeks after correction for maternal age, weight and race.

B) hCG: is a product of the placental trophoblasts. Its levels correlate with gestational age in normal pregnancies. Levels are *abnormally increased* in fetuses with trisomy 21 (Down syndrome)

C) PAPP-A: Pregnancy associated plasma protein A is a placental product that is found in *abnormally decreased* levels in Down syndrome fetuses.

D) Unconjugated Oestriol (uE3): a placental product that is *decreased* in Down syndrome fetuses

The Double marker test (DMT)

Combining maternal serum **hCG** with **PAPP– A** in one test performed in the first trimester (11-13 weeks gestation) together with US measurement of the nuchal translucency (NT) at the foetal neck, as shown later, can improve the predictive accuracy for the risk for Down syndrome to > 85%.

- Down syndrome risk is high when the test shows **high** hCG, **low** PAPP– A levels, with increased NT > 3.0 mm at 12 weeks gestation. A high risk score calls for further triple marker test, CVS, or amniocentesis

The Triple Marker Test (TMT):

The combination of **MSAFP, hCG,** and **uE3**, in one test performed in the second trimester (15-16 weeks gestation) yields good information about the risk of trisomies (namely Down syndrome), taking in consideration maternal age, weight, race, and gestational age.

- **Down syndrome** risk is high when the triple marker test shows **high** hCG levels with **low** MSAFP and uE3 levels. A high risk score calls for further amniocentesis or CVS.
- **N.B.:** maternal age related risk for Down syndrome is nearly 1/600 at age < 25 years, rising to nearly 1/100 at age 39, and 1/40 at age 44 years.

2. ULTRASOUND SCREENING AND DIAGNOSIS OF FOETAL ANOMALIES

- Screening by US starts as early as the 9th week and continues till the third trimester.
- US evaluation of nuchal translucency (NT): measurement of foetal nuchal fold thickness at 11-13 weeks gestation shows good correlation with the presence of foetal chromosomal abnormalities. NT thickness > 3.0 mm at 11-13 weeks should call for more invasive diagnostic work up to exclude Down syndrome (DMT, TMT, CVS, and amniocentesis)
- Anencephaly can be excluded by the end of first trimester.
- The second trimester detailed fetal anatomy survey; is usually performed at 20-24 weeks gestation for early diagnosis of important major fetal congenital anomalies as; NTDs, most of the skeletal, cardiac, renal and GIT anomalies, in addition to diaphragmatic and ventral hernias.

3. AMNIOCENTESIS

In amniocentesis an amniotic fluid sample is obtained via a transabdominally introduced needle.

Foetal cells obtained from amniotic fluid will be subjected to Karyotyping, fetal sexing, DNA analysis using polymerase chain reaction (PCR), and enzyme assay to achieve a final accurate diagnosis of several chromosomal, biochemical, and hereditary disorders.

- *Technique:* At 14-16 weeks gestation, a 22 gauge needle is introduced transabdominally, under local anesthesia and ultrasonographic guidance, to aspirate 10-20 ml of amniotic fluid which will be available for culture and chromosomal analysis. Results are reproducible within 2-4 weeks.
- *Risks:* Amniocentesis carries a definite however very small risk of fetal infection, rupture of membranes, accidental haemorrhage, and feto-maternal transfusion with the risk of sensitization in cases of RH negative mothers.

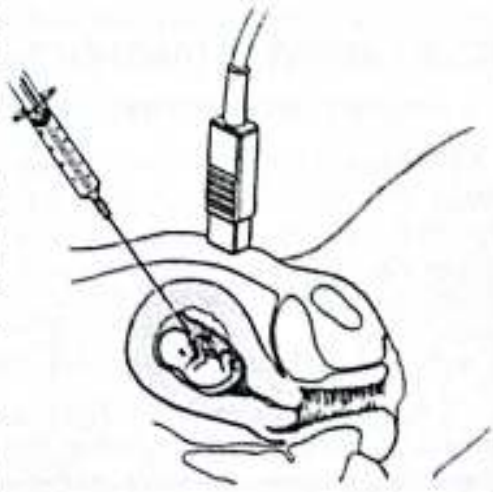


Fig 6:1 Amniocentesis

4. CHORIONIC VILLOUS SAMPLING

a. *Trans-abdominal method:* after 12 weeks gestation, under ultrasonographic guidance, the 22 gauge needle is introduced to get at least 0.5 cc of chorionic villi as a tissue sample.

b. *Trans-vaginal method:* only done from 7-12 weeks for early diagnosis.

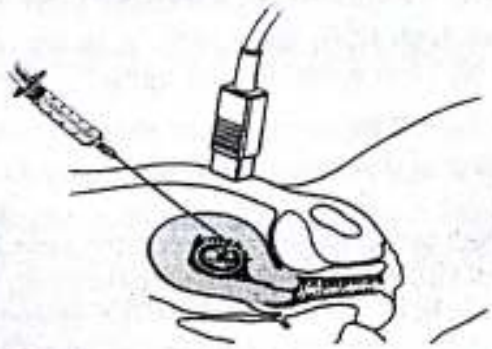


Fig 6:3 Transabdominal CVS

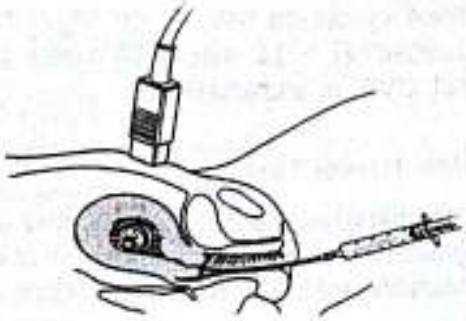


Fig 6:2 Transvaginal CVS

5. CORDOCENTESIS

After 18 weeks gestation, with the same technique of amniocentesis, the needle is introduced into the umbilical cord to aspirate at least 1 ml of fetal blood. The technique needs special experience and training. It carries the same risks as amniocentesis with an additional risk of fetal haemorrhage and bradycardia.

6. MAGNETIC RESONANCE IMAGING (MRI):

Can efficiently diagnose some of the structural anomalies in which ultrasonography is not very sensitive. Although of proven safety, yet its use is limited by its costs and availability when compared to ultrasonography.

Screening for trisomy 21 (Down syndrome) includes;

a. First trimester US scan for NT measurement and nasal bone hypoplasia, together with double marker screening test (DMT) at 11-13 weeks

b. Second trimester MSAFP, or Triple marker test (TMT), at 14-16 weeks .

c. Second trimester US foetal anatomy survey; CVS and amniocentesis as final diagnostic tools in high risk cases at 12 and 16 weeks gestation

NEW PRENATAL DIAGNOSTIC OPTIONS IN THE HORIZON

- **PREIMPLANTATION GENETIC DIAGNOSIS (PIG):**

Early in human gestation, a single cell at the 8 cell stage, or a dozen cells at the blastocyst stage, can be removed without subsequent damage to the fetus. These cells provide sufficient DNA for PCR directed molecular analysis for inherited diseases, or fluorescent in situ hybridization for aneuploidy. Preimplantation biopsy for prenatal diagnosis involves participation in an IVF program and is currently available for a limited number of genetic conditions.

- **FETAL CELLS IN MATERNAL CIRCULATION:**

Acquisition of fetal cells from maternal circulation without invasive procedures is the future in screening for foetal chromosomal problems. There is still difficulty in isolating the very few fetal cells from the overwhelming number of maternal cells. Once isolated, fetal cells provide information about the fetus through PCR, and molecular studies, as well as hybridization for aneuploidy.

7 ABORTION

Spontaneous abortion

Clinical types of abortion

- Threatened abortion
- Inevitable abortion
- Incomplete abortion
- Complete abortion
- Missed abortion
- Septic abortion

Complications of abortion

Induced abortion

Second trimester pregnancy loss

Isthmic Incompetence

Recurrent pregnancy loss

Abortion is the term used to describe foetal loss before viability i.e.; pregnancy termination before capability of the foetus to survive outside the uterus.

WHO definition:

- Pregnancies terminated **before 20 weeks gestation**, or
- Pregnancies terminated in which the foetus **weighs < 500 gm**.
- In many countries **24 weeks** is used as the cut off for foetal viability rather than 20 weeks.

Definitions and Terminologies:

The terms ***miscarriage, early pregnancy failure, or early pregnancy loss***, are all synonyms to the term abortion, and are used interchangeably to describe the same condition.

- **Spontaneous abortion**: is an abortion that occurs accidentally either once or twice
- **Clinical types of abortion:** Threatened abortion, inevitable abortion, incomplete, complete, missed, and septic abortions
- **Recurrent abortion**: is the term used to describe more than 2 successive spontaneous abortions without intervening term pregnancies.
- **Induced abortion:** is an abortion that is usually medically or surgically induced.
- **Illegal or criminal abortion:** is an induced abortion of an intact pregnancy without medical indication for pregnancy termination

BLEEDING IN EARLY PREGNANCY

Obstetric causes	Non Obstetric causes
1. Abortion	4. Cervical erosion, polyps, or carcinoma
2. Ectopic pregnancy	5. Severe Vaginitis
3. Molar pregnancy	6. General systemic disease: HTN , and ITPP

SPONTANEOUS ABORTION

INCIDENCE

- Spontaneous abortion may complicate almost **15%** of all clinically detectable pregnancies
- Nearly **80 %** of spontaneous abortion occur in the in the **1st trimester** (usually <8 weeks)
- After 12 weeks the incidence of spontaneous abortion will sharply decrease (< 20%).
- Clinical types of abortion: threatened, inevitable, incomplete, complete, missed, and septic abortion

Aetiology of Abortion

A) Foetal Causes:

1. Chromosomal abnormalities
   - *Aneuploidy*
   - *Inherited defects*
2. Structural congenital anomalies

B) Maternal Causes:

- *Maternal Infections*
- *Uterine & endometrial causes*
- *Hormone deficiency*
- *Endocrine disorders*
- *Immunologic disorders*

C) Severe Trauma:

- *External trauma*
- *Internal uterine trauma*

D) Drugs & Toxins:

- *MTX, PGLs, Anti-P, etc...*
- *Alcohol, caffeine, etc*
- *Anaesthetic gases, etc...*

RISK FACTORS FOR SPONTANEOUS ABORTION

1. Low socio-economic status
2. Advancing maternal and paternal age
3. Increasing gravidity and parity .
4. Previous and recurrent pregnancy losses
5. Heavy smoking
6. Distortion of uterine cavity: due to congenital uterine anomalies or leiomyomata.

AETIOLOGY OF ABORTION

A. FOETAL CAUSES

1. Chromosomal Abnormalities:

These represent the *single most common cause* for early pregnancy losses, encountered in at least **60%** of 1st trimesteric abortions.

a. Aneuploidy:

Aneuploid abortions tend to occur in early 1st trimesteric miscarriages, with 95% of such chromosomal abnormalities due to maternal gametogenesis errors. They are mostly non-inherited sporadic defects, with low tendency for recurrence. Common examples include;

- *Autosomal trisomies;* are the most common anomalies including 13,16,18,21, and 22
- *45X monosomy;* these cause Turner syndrome that usually end in abortion
- *Triploidy;* is often associated with complete molar pregnancies. In partial moles the foetus is usually dead or severely malformed, and rarely survives.
- *Tetraploidy;* tetraploid abortuses are most often aborted in early pregnancy

b. Inherited Defects; in which one of the parents may carry a *balanced translocation*, are rare but have a tendency for recurrence. It is therefore a significant cause for recurrent pregnancy loss (RPL).

2. Structural Congenital Malformations

Foetal congenital malformations may be a cause of late abortions mostly 2nd trimesteric, especially lethal anomalies incompatible with life.

B. MATERNAL CAUSES

1. Maternal Infections

Maternal infections are an **uncommon** cause for 1st trimesteric abortion, however chorioamnionitis, is an important cause for late 2nd trimesteric pregnancy loss.

1. *Acute febrile illness* with persistent high grade fever.
2. *Acute Infection early in the 1st trimester with organisms and viruses;* as Toxoplasma Gondii (TG), Rubella, Cytomegalovirus (CMV), and herpes Simplex virus (HSV).
3. *Ascending genital tract infections,* (chlamydial, gonorrhoeal, and group B-haemolytic streptococci), leading to chorioamnionitis, and prostaglandin release

2. Uterine and Endometrial causes:

- *Congenital uterine anomalies;* as septate and bicornuate uterus.
- *Uterine leiomyomata;* especially submucus myomas, and endometrial polypi.
- *Intrauterine synaechae;* as Ashermann syndrome.
- *Cervical Incompetence;* is one of the commonest causes of late and recurrent 2nd trimesteric abortion where the weak sphincter mechanism at the internal os cannot retain the advancing pregnancy (see later).

3. Hormonal Deficiency

- *Progesterone deficiency in early pregnancy;* luteal phase defect (LPD) may play a role in early 1st trimesteric abortion. It is controversial whether the associated progesterone deficiency is the cause or the result of the abortion. However it is unlikely that a normal pregnancy can be lost as a result of abnormal hormone production.

4. Endocrine Disorders:

- *Diabetes mellitus (DM);* uncontrolled DM may be responsible for both 1st trimesteric abortion and specific lethal foetal congenital anomalies later in pregnancy.
- *Hypothyroidism;* in its severe forms may cause early embryonic death and abortion.

5. Immunologic Disorders:

- *Systemic Lupus Erythematosus (SLE):* up to 40% of pregnancies are lost in women with SLE before such women will develop the clinical stigmata of the disease.
- *Antiphospholipid syndrome (APS):* may cause recurrent late 1st trimesteric RPL due to a tendency for early thrombosis in decidual vessels.

C. SEVERE TRAUMA:

- *External trauma;* as direct hit on the abdomen, car accidents and fall from heights.
- *Internal uterine trauma;* as sounding the uterus or introducing an IUD during an undiagnosed early pregnancy may result in disturbance of pregnancy and abortion.

D. DRUGS AND TOXINS:

- *Chemotherapeutic agents* (e.g.; Methotrexate), *Prostaglandins* (e.g.; Mesoprostol), *Anti-progesterone* (e.g.; Mifeprostone), and other drugs may cause IUFD followed by abortion.
- Large doses of *alcohol, caffeine*, and some *anaesthetic and toxic gases* are rare causes.

THREATENED ABORTION

Threatened abortion describes mild vaginal bleeding that occurs in the first half of pregnancy < 20 weeks. It may be encountered in up to 25% of pregnancies in the 1st trimester.

In > 50% of cases the pregnancy will continue uninterrupted till delivery, however in the rest of cases pregnancy will end in either an inevitable abortion or a missed abortion (see later).

N.B.: Some women may experience spotting of blood at the time of blastocyst implantation that may be confused with the start of a menstrual cycle (Hartman's sign)

Symptoms and Signs

- *Vaginal bleeding;* is minimal or mild with absence of blood clots
- *Pain;* mild suprapubic dull aching in character, associated with pelvic heaviness
- *Uterine size;* corresponds to the gestational age by LMP
- *Cervix;* is formed and closed on vaginal examination

Laboratory and US Diagnosis

A. Quantitative serum B-hCG assay:

- Levels correlate with the duration of pregnancy, according to LMP
- Levels increase > 66% when repeated after a 48 hours interval (between 5-8 weeks) denoting an intact pregnancy with normal trophoblastic activity.

B. Ultrasonography: TAS or TVS will show;

- Intact pregnancy, with evident cardiac pulsations (in pregnancies > 7 weeks gestation).
- Mild choriodecidual separation and intrauterine bleeding may be elicited in some cases.

Management

Management of threatened abortion is essentially **conservative** through rest and reassurance since the majority of cases will successfully continue their pregnancy to full term

1. ***Physical and Mental Rest;*** is the single most important line of management. Exercise and sexual intercourse are better avoided for few weeks after bleeding stops.
2. ***Hormonal support:*** using natural progesterone by IM injections or via the oral or vaginal route, are commonly used but without enough evidence to support their values, except in cases with corpus luteum insufficiency (LPD).

INEVITABLE ABORTION

In this type of abortion the uterus starts to contract in order to expel its contents. The condition is irreversible, and termination of pregnancy becomes inevitable and mandatory.

PATHOLOGY AND MECHANISM OF ABORTION

- **In the 1st trimester;** partial or complete separation of the decidua basalis occurs, followed by rupture and expulsion of the ovum or embryo through the dilated cervix.
- **n the 2nd trimester;** uterine contractions occur spontaneously, or after rupture of the membranes, leading to cervical dilatation, and expulsion of the foetus followed by the placenta, resembling miniature labour.

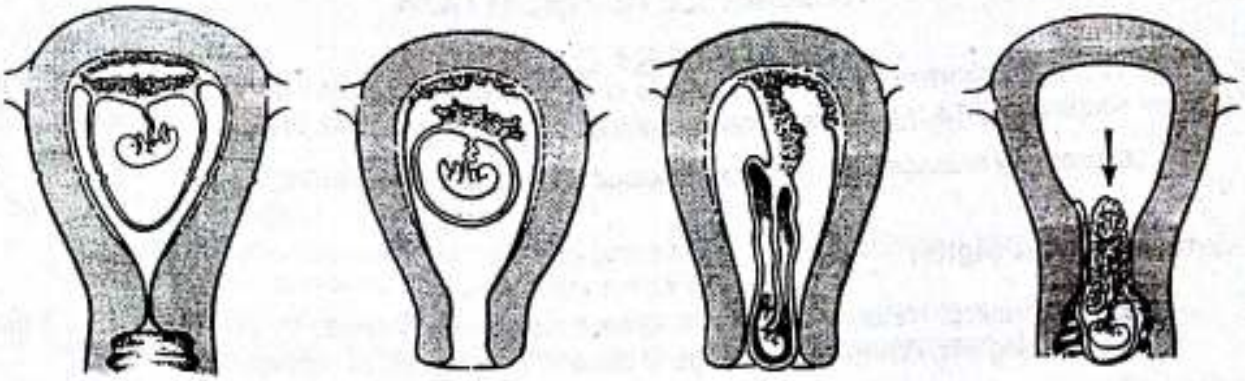


Fig 7:1 Mechanism of abortion

Symptoms and Signs:

- *Vaginal bleeding;* is severe with passage of blood clots. In some cases may lead to hypovolaemic shock
- *Pain;* Severe suprapubic colicky pain due to uterine cramping
- *Rupture of the membranes (ROM);* if occurs is a sign of an inevitable abortion (2nd trimester)
- *Uterine size;* may corresponds to period of amenorrhoea, or slightly smaller
- *Cervix:* internal os becomes dilated as elicited on digital vaginal or speculum examination

Ultrasound diagnosis: (TAS/TVS): reveals;

- Intrauterine products of conception (a dead or living embryo or foetus)
- The internal cervical os opened
- Intrauterine bleeding and choriodecidual separation are evident.

Management of inevitable Abortion: (immediate active management)

1. Stabilizing the patient's general condition in cases of severe bleeding, through

- Hospitalization and IV infusions of saline and lactated Ringer's solutions
- Blood transfusion in severely anaemic patients due to excessive blood loss
- Antibiotics to guard against 2ry infection

2. Evacuation of uterine contents;

Surgical Evacuation (SE) **: <13 weeks**; via Suction of intrauterine contents and curettage of the uterine cavity. The procedure is performed vaginally under general anaesthesia

Medical evacuation (ME) **: >13 weeks;** via ecbolics used to induce expulsion of the products of conception vaginally resembling a miniature labour process. Induction is usually started by oral and/or vaginal prostaglandin E1 (Mesoprostol 200 ug / 4-6 hours) followed by IV drip containing oxytocin and/or Ergometrin, to ensure complete expulsion of all contents.

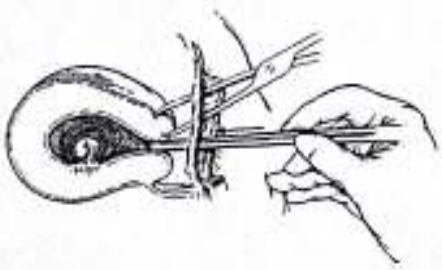


Fig7:2 Suction Evacuation

Fig 7:3 Endometrial Curettage

IN-

INCOMPLETE ABORTION

In incomplete abortion part of the products of conception are expelled during the process of inevitable abortion, while some parts of foetal and/or decidual tissue are retained within the uterus.

If not promptly managed it may be complicated by persistent haemorrhage, and infection.

Symptoms and Signs:

- *Vaginal bleeding:* history of a recent spontaneous abortion followed by persistent painful profuse bleeding with intermittent passage of some of the products of conception
- *Uterus;* usually subinvoluted, soft in consistency, with the cervical internal os still opened.

Ultrasound diagnosis:

- *TAS or TVS;* reveals significant remnants of conception within the uterine cavity

Management of Incomplete Abortion:

- ***Surgical evacuation (SE);*** of the remaining tissue by curettage of the uterine cavity under general anaesthesia through the already dilated cervix
- ***Antibiotics*** are given to guard against 2ry infection

COMPLETE ABORTION

All products of conception have been expelled leaving an empty uterine cavity.

Symptoms and Signs:

- *History of a recent abortion*
- *Vaginal bleeding:* diminishes and gradually stops
- *The uterus:* contracted, returns to normal size, with the internal os closed.

Fig 7: 4 Incomplete abortion; Retained products of conception

Ultrasound diagnosis:

TAS or TVS; will show an empty uterine cavity, with no retained products of conception.

Management:

- Reassurance and follow up by US to ensure an empty uterine cavity with no remnants,
- Some investigations may be performed to clarify possible causes for abortion in an attempt to avoid its recurrence.

MISSED ABORTION

Death and retention of the products of conception in utero for a prolonged period

Symptoms and Signs:

- *Symptoms of early pregnancy;* become less pronounced (nausea, vomiting, etc...)
- *Brownish vaginal discharge;* may be an early symptom or sign
- *Recurrent vaginal bleeding;* is usually dark in colour and minimal in amount
- *Bimanual examination;* reveals a soft uterus, with a size smaller than gestational age

Ultrasound Diagnosis: *TAS or TVS;* shows anembryonic gestational sac, or intrauterine foetal (IUFD), with gestational age less than that calculated based on the LMP.

Complications:

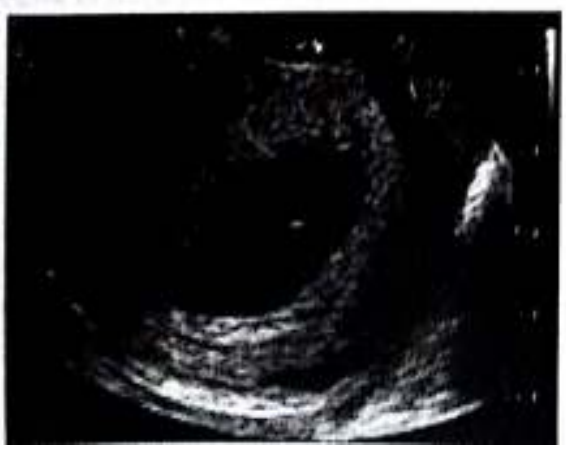
- Sepsis and Infection
- Hypofibrinogenaemia and DIC: because prolonged retention of IUFD may be associated with the release of thromboplastin like substances leading to DIC with subsequent consumption of all clotting factors, resulting in hypofibrinogenaemia and severe haemorrhage.

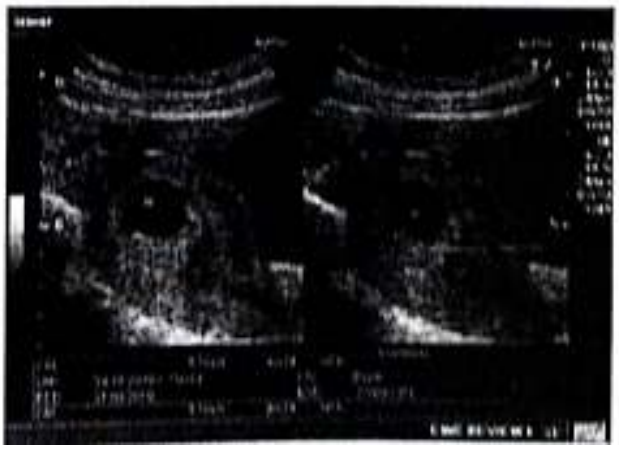
Management in the First Trimester

- **Expectant management:** in most cases, spontaneous expulsion of uterine contents will start to occur within 2-3 weeks of embryonic death, without significant maternal hazards. The case will be thus converted into a spontaneous inevitable abortion which is managed accordingly.
- **Active management (Termination of pregnancy);** If spontaneous expulsion is delayed, or the patient becomes too much anxious about the possible complications of a retained non viable pregnancy, then pregnancy should be immediately terminated via;
    1. **Medical Evacuation (ME):** using **prostaglandin E** (Mesoprostol 400 ug/6 hours) orally or vaginally, to induce uterine contractions and convert the case into inevitable abortion.
    2. **Surgical Evacuation (SE);** under general anaesthesia, from the start or if incomplete expulsion of contents occurred after medical evacuation by PGL E.

Management in the Second Trimester:

- **Expectant management;** for 2-3 weeks awaiting conversion into an inevitable abortion, that will be managed medically by I.V. ecbolics and or prostaglandins.
- **Active management;** If spontaneous expulsion is delayed, or the patient is too much anxious, or laboratory signs of an ominous coagulopathy is evident, then pregnancy should be terminated via medical induction of abortion using;
    1. **Prostaglandin E** (Mesoprostol 400 ug/6 hours); orally or vaginally or both.
    2. **I.V. drip using ecbolics** (oxytocin and Ergometrin)





SEPTIC ABORTION

In septic abortion, severe infection occurs on top of any clinical type of abortion

Risk factors:

- Pregnancies occurring on top of an IUD
- Prior cervical gonococcal or chlamydial infection

Aetiology:

- Prolonged presence of intrauterine retained products of conception especially after a missed or an incomplete abortion.
- Criminal induction of abortion using non properly sterilized instruments

Organisms:

- Mostly B-haemolytic streptococci, staphylococci, E. coli, and Clostridium Welchii

Symptoms and Signs:

- *Persistent fever* associated with lower abdominal pain and malodorous vaginal discharge
- *Lower abdominal and pelvic tenderness* on bimanual vaginal examination
- *Complete blood picture (CBC);* shows increase leucocytic count, with shift to the left in ratio between staff and segmented WBCs (due to increased immature forms of WBCs).

Investigations:

- *TVS/TAS;* may show infected intrauterine retained products of conception
- *CBC:* will show leucocytosis, with shift to the left (abnormal staff/segmented WBC ratio)

Complications of septic abortion:

- *Septic shock* that may predispose to *Acute renal failure*
- *Spread of infection:* leading to salpingitis and pelvic peritonitis.
- *Disseminated intravascular coagulopathy (DIC);* this is triggered by the release of inflammatory mediators (cytokines)

Management of Septic Abortion

- Antibiotics: broad spectrum antibiotics as Cephalosporins, better by IV route
- Immediate complete evacuation of uterine contents (surgically via SE).
- Ecbolics as Ergometrin (Methergin) and prostaglandins (Mesoprostol) are also used to augment expulsion of uterine contents and avoid incomplete abortion

COMPLICATIONS OF ABORTION

1. Haemorrhage:

- *Continuous or prolonged bleeding;* may be associated with incomplete evacuation
- *Severe haemorrhage;* may be associated with late 1st trimesteric and 2nd trimesteric abortion
- *Haemorrhagic shock:* if severe haemorrhage occurs in a short duration it may lead to hypovolaemic shock with; hypotension, tachycardia, tachypnea, oliguria, etc..... . The condition is life threatening if intervention is delayed.

2. Infection (Sepsis):

This may complicate various types of abortion leading to *Post abortive endometritis.*

Septic abortion may lead to serious and even fatal complications (see before in septic abortion))

- *Hypofibrinogenaemia;* In neglected cases of 2nd trimester abortions, prolonged IUFD may lead to release of thromboplastin like substances leading to DIC, consumption coagulopathy, hypofibrinogenaemia, and severe haemorrhage, which may lead to maternal death.

3. Hypofibrinogenaemia and DIC: may complicate cases of missed or septic abortion

4. Complications of surgical evacuation SE: as uterine perforation and cervical laceration

5. RH sensitization: In RH-negative females the risk of sensitization increases with advanced gestational age at the time of abortion. The recommended dose is a single I.M. injection of anti-D immunoglobulin in a dose of 50 ug up to 12 weeks, and 300 ug thereafter.

N.B.: Future adverse pregnancy outcomes: the incidence of infertility, spontaneous abortion, and ectopic pregnancy, do not increase after a non complicated spontaneous or induced abortion.

MATERNAL MORTALITY

The leading causes of death include; Haemorrhage, Infection (sepsis), Thrombo-embolism. In **Induced abortion** anaesthetic complications will be added on top of the list.

INDUCED ABORTION

Inducing abortion is only allowed in cases of intrauterine embryonic or foetal death, lethal foetal congenital anomalies detected by ultrasonography, severe maternal illness that will be worsened by pregnancy, and in rare cases of 1st trimesteric maternal exposure to foetal **teratogens** as; specific viral infections (as Rubella), ionizing radiation (as radiotherapy), or certain toxins .

METHODS AND TECHNIQUES OF TERMINATION OF PREGNANCY

A. Anti-progesterone *(Mifeprostone – RU 486 oral tablets);* is highly effective in pregnancy termination with up to 49 days amenorrhoea (i.e.; < 7 weeks). Its effectiveness is increased with adding oral or vaginal prostaglandin E.

B. Prostaglandin E *(Misoprostol 200 ug tablets);* given orally or vaginally, once every 4-6 hours, is highly effective in pregnancy termination in cases of missed abortion and IUFD both in late 1st trimester and in the 2nd trimester.

C. Surgical Evacuation (SE): via

- *Suction Curettage:* dilatation of the cervix and vacuum aspiration of the uterine contents is suitable for early 1st trimesteric abortion
- *Dilatation and Curettage (D&C):* dilatation of the cervix, evacuation of uterine contents using ovum forceps, and curettage of the uterine cavity, is suitable for late 1st trimesteric abortion

D. Induction of abortion:

- *I.V. drip infusion containing oxytocin;* has long been the traditional method used in 2nd trimesteric induction of abortion. Vaginal prostaglandins may be used to ripen the cervix, and shorten the duration of induction.
- *Prostaglandin E (Misoprostol 200 ug tablet);* recently the use of oral and vaginal prostaglandin E tablets has been used with great success being now the first choice drug in medical induction of 2nd trimester abortion.

SECOND TRIMESTERIC PREGNANCY LOSS

DEFINITION:

Late or 2nd trimester pregnancy loss occurs between 13-24 weeks gestation. After 24 weeks the foetus is potentially viable and birth is referred to as preterm delivery.

INCIDENCE:

Exact incidence is unknown, but generally complicates 1-2% of pregnancies past the 1st trimester.

AETIOLOGY:

- **Early 2nd trimester (13-16 weeks):** causes similar to 1st trimesteric abortion including; foetal chromosomal and structural anomalies, and possibly endocrine disorders.
- **Late 2nd trimester (18-24 weeks):** causes similar to those of the very preterm delivery as;

1. Placental separation: causing intrauterine bleeding and initiating uterine contractions
2. Ascending genital tract infection: including chlamydial, gonorrhoeal, and group B-haemolytic streptococci. Infection leads to both;
   - Chorioamnionitis with erosion and rupture of the membranes
   - Prostaglandin release and initiating labour.
3. Cervical Isthmic incompetence; due to weakness of the sphincter mechanism at internal os.

CERVICAL INCOMPETENCE

(Incompetent isthmus)

Cervical incompetence describes inability of the cervix to retain the conceptus during the 2nd trimester, due to weakness of the sphincter mechanism at the uterine isthmus and internal os.

INCIDENCE: Approximately 0.5 to 1% of pregnancies are complicated by cervical incompetence.

AETIOLOGY

A. Congenital weakness:

1. *Isolated defect;* due to decreased cervical Elastin content
2. *In association with uterine anomalies;* as septate and bicornuate uterus

B. Acquired weakness;

1. *Anatomic distortion:* as with large cervical leiomyomata, and large endometrial polyps
2. *Traumatic distortion:*
   - During labour: cervical lacerations may occur in association with difficult breech delivery, instrumental delivery, and shoulder dystocia.
   - After Gynaecologic procedures: as
     a. D&C procedures; especially if cervical dilatation is excessive and forcible, and the procedure is frequently repeated.
     b. Conization of the cervix, Loop excision transformation zone, and high cervical amputation.

CLINICAL PICTURE SUGGESTIVE OF AN INCOMPETENT ISTHMUS

Recurrent late 2nd trimesteric pregnancy loss (16-24 weeks) with the following characteristics

- Pain is usually mild and well tolerated by the patient
- The clinical course is usually short with progressive rapid cervical dilatation
- Bleeding is usually mild.
- Membranes usually rupture early with the start of abortion. However in some cases it may remain intact till abortion is complete.
- The foetus may be born living or recently dead (stillborn SB), in absence of major congenital anomalies thus excluding a foetal cause for the abortion.

DIAGNOSIS OF ISTHMIC INCOMPETENCE

A. Recent history; of recurrent mid trimesteric abortions, or preterm labour, with the clinical characteristics suggestive of isthmic incompetence (see before)

B. Past history of traumatic procedures to the cervix as forcible dilatation, amputation, conization, etc... (see aetiology),

Diagnosis during pregnancy

US especially TVS has a role in diagnosis and prediction of cases at high risk for midtrimesteric abortion due to incompetence of the isthmus. The following criteria are important;

- Shortened cervical canal length in 2nd trimester (< 2.5 cm) is suggestive of incompetence.
- Widened internal os diameter in early 2nd trimester (in relation to gestational age).
- Funneling of the cervical isthmus
- Bulging and protrusion of the foetal membranes through the dilated cervical canal.

Diagnosis in between pregnancies:

- Passage of a No. 8 Hegar dilator through the internal os easily without resistance
- Hysterosalpingography (HSG): Luteal phase isthmography; showing internal os dilatation of 6 mm or more or funneling of the internal os is also diagnostic.

MANAGEMENT OF ISTHMIC INCOMPETENCE:

A. Cervical Cerclage Operations

- This is the most successful treatment of incompetent cervix, by placing a cervical suture encircling the cervical canal via the vaginal route, under general anaesthesia (McDonald's or Shirodkar procedures).
- In rare occasions an abdominal approach for cerclage via laparotomy may be recommended
- Timing of procedure: at 12-14 weeks, in order to exclude 1st trimesteric spontaneous abortions associated with foetal chromosomal abnormalities.
- The suture is removed at 37 weeks or at onset of labour pains at any gestational age.

A. McDonald's operation:

Four bites of Mersilene tape, or non absorbable silk sutures, are taken as high as possible in the cervix like a purse string. This is the procedure of choice as it is rapidly and easily performed with a high success rate (75-85%).

B. Shirodkar operation:

Mersilene tape or silk suture is inserted under the cervical mucosa surrounding the cervix at the level of internal Os. It is more lengthy and associated with more blood loss, therefore less commonly performed than the McDonald's procedure. It is mainly indicated when there are deep cervical lacerations or failed Mc Donald's.

C. Abdominal cerclage:

A suture is inserted via a laparotomy encircling the cervix at the level of internal Os. It is a complicated procedure that has a limited indication, only in cases with high cervical lacerations or repeated failed Mc Donald's procedure. The patient is delivered by CS at 37 weeks or at onset of labour pains.

Medical measures:

Physical and mental rest, avoid fatigue and exertion, and progesterone support are helpful in cases of RPL, but in most cases are not adequate by themselves, but may be used after cerclage procedures.

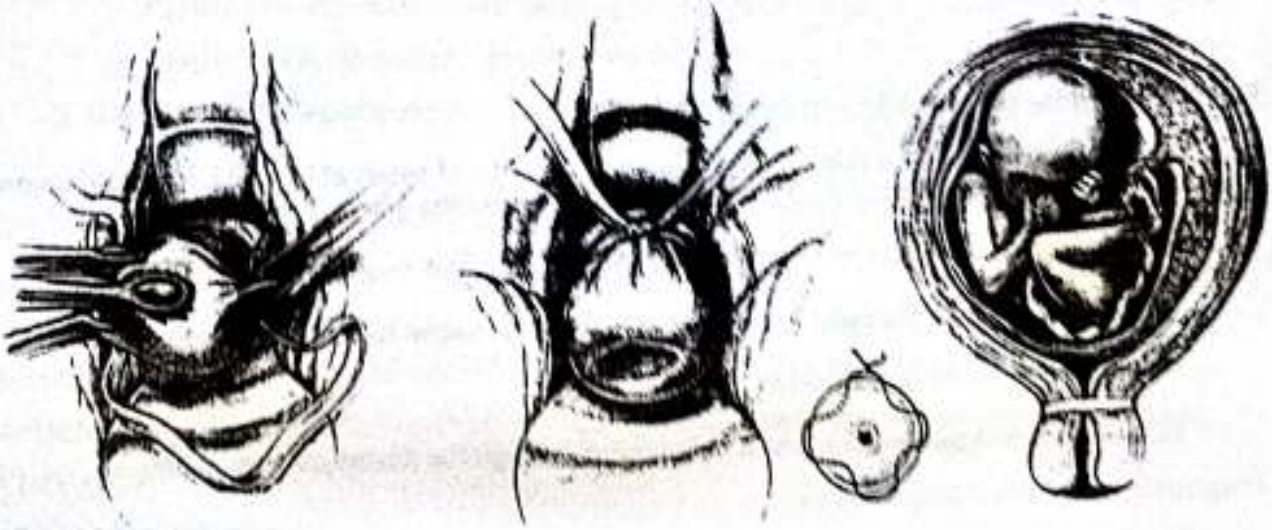


Fig 7:7 McDonald's Cerclage Operation

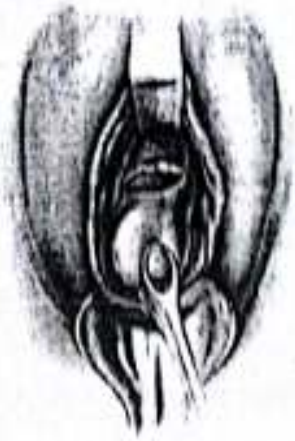


Fig 7:8 Shirodkar Cerclage Operation

Fig 7:9 Abdominal Cerclage Operation

RECURRENT PREGNANCY LOSS (RPL)

DEFINITION:

RPL is the term used to describe the occurrence of more than 2 successive spontaneous pregnancy losses without intervening term pregnancies. It has been previously called **habitual abortion**, and may affect nearly 2% of couples. Most RPL occur in the 1st trimester.

AETIOLOGY

a. Genetic and chromosomal abnormalities
b. Hormonal disorders; as luteal phase defect (LPD)
c. Anatomic abnormalities; as congenital uterine hypoplasia, anomalies, and leiomyomata
d. Autoimmune disorders; as SLE, and Antiphospholipid syndrome (APS)
e. Endocrine disorders; as severe hypothyroidism
f. Thrombophilias; as protein-S and protein-C deficiencies
g. Incompetent isthmus; in 2nd trimesteric habitual abortion

INVESTIGATIONS FOR A CASE OF RPL

Evaluation of the patient is indicated after 3 successive losses; however it may start after the second loss according to the age of the patient and the clinical course of the abortion. The patient may present during pregnancy or in between pregnancies usually after an abortion event.

1. Detailed history and physical examination
2. Chromosomal evaluation of the couple (via blood sampling);
3. Hysterography or hysteroscopy; for evaluation of abnormalities within the endometrial cavity
4. Premenstrual dated endometrial biopsy; to exclude cases with luteal phase defect
5. Thyroid function tests; to exclude severe thyroid disorders
6. Screening for immunologic disorders namely; Lupus anticoagulant (LAC) and anticardiolipin antibodies (ACA) both IgG and IgM for diagnosis of anti-phosholipid syndrome (APS), protein-S and protein-C deficiencies.
7. TVS during late first trimester to detect cervical shortening and herniation of the amniotic sac through the dilated internal os.

Management of cases with RPL

Treatment of RPL depends on identification of the underlying aetiology. However in up to 30% of cases no aetiology is ever identified, and hence measures taken will be empirical rather than specific.

A. Management in between pregnancies

- Maternal and paternal chromosomal studies to detect hereditary disorders
- Cyclic oestrogen (E) and progesterone (P) to increase the uterine size in cases of hypoplasia
- Progesterone (P) support in cases of LPD, in luteal phase of the cycle
- Control of chronic diseases as diabetes mellitus (DM), hypertension (HTN), and thyroid disorders.

- Treatment of maternal infections as Toxoplasma (Rovamycin), Syphilis (Penicillin), Herpes simplex virus HSV (Acyclovir), etc...
- Hysteroscopic removal of a uterine septum
- Myomectomy for submucous myomas and polypectomy for endometrial polyps

B. Management during pregnancy

- Progesterone support for cases of LPD early in 1st trimester. P may also be continued till third trimester to decrease chances of late 2nd trimester abortions and preterm labour
- Low dose aspirin (LDA) and / or, low molecular weight Heparin in cases of thrombophilias and APS
- Cerclage for isthmic incompetence

Key Points in abortion

- *15% of clinically recognized pregnancies will end in an abortion.*
- *Chromosomal defects are responsible for nearly 50% of early 1st trimesteric abortions*
- *Most cases of threatened abortion will successfully continue with their pregnancies till delivery*
- *TAS & TVS play a major role in diagnosis of the type of abortion and assess foetal viability*
- *Inevitable abortion mandates immediate intervention to help complete expulsion of uterine contents either medically or surgically*
- *In cases of missed abortion, an expectant management could be adopted for 2-3 weeks. If delay occurs or complications are anticipated, termination may be initiated either medically or surgically according to the gestational age.*
- *Retained dead foetus for 4 or more weeks is associated with the risk of severe bleeding due to coagulopathy.*
- *Midtrimesteric recurrent abortions due to incompetent isthmus is best managed surgically through vaginal cerclage procedures, mostly McDonald's operation.*
- *RPL in 1st trimester necessitates special investigations to help identifying possible treatable causes.*

8 ECTOPIC PREGNANCY

Locations
Tubal Ectopic
- *Incidence and sites*
- *Predisposing factors*
- *Aetiology and risk factors*
- *Pathology*
- *Clinical picture*
  a. Undisturbed ectopic
  b. Disturbed ectopic
  c. Chronic ectopic
- *Special investigations*
- *Differential Diagnosis*
- *Management of tubal ectopic*
  a. Surgical
  b. Medical by MTX
- *Prognosis*

Other rare types of ectopic gestation
  a. **Ovarian ectopic**
  b. **Pregnancy in a rudimentary horn**
  c. **Cervical ectopic**
  d. **Advanced extrauterine pregnancy**

DEFINITION

Ectopic pregnancy is implantation of the fertilized ovum outside lining of the endometrial cavity.

INCIDENCE

Ectopic pregnancy may occur in 0.5 to 2% of pregnancies. The true prevalence of ectopic gestation remains unknown, as many cases may occur without being clinically diagnosed. The past few decades witnessed an increasing rate of diagnosis of ectopic pregnancies due to:

a. The use of B-hCG and transvaginal sonography (TVS) in the early diagnosis of pregnancy
b. The introduction of assisted reproductive techniques (ART) in the management of infertility.
c. The increased incidence of PID and STDs
d. The frequent use of laparoscopy in diagnosis and management of doubtful cases with adnexal pain and/or pelvic masses

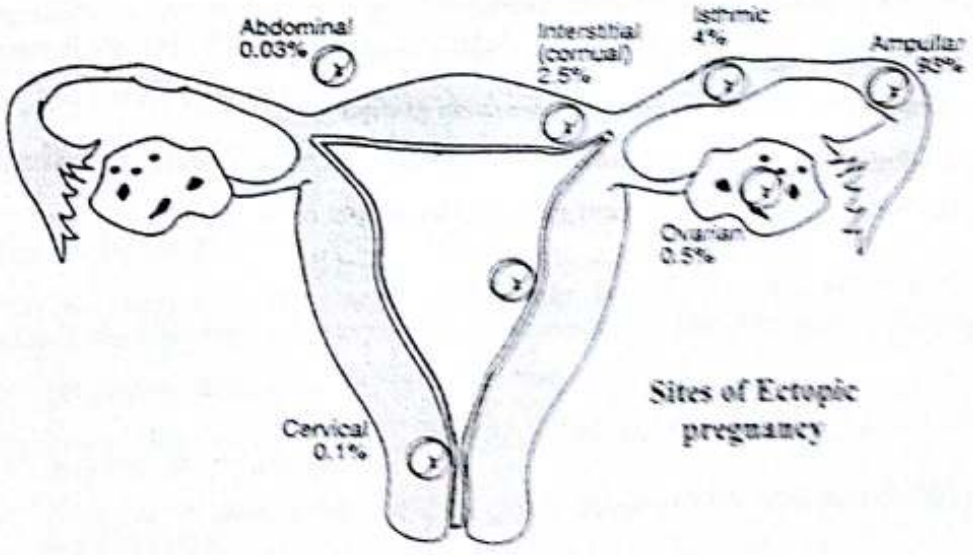


Fig 8:1 Locations and sites of ectopic gestation

LOCATIONS OF ECTOPIC GESTATION

1. **Tubal (96%):** any where in the fallopian tube; mostly at the ampulla.
2. **Ovarian (2.0%):** on the ovary
3. **Cervical (<1.0%):** in the cervix below the internal os
4. **Abdominal (<0.1%):** any where within the abdominal cavity, with adherence to the peritoneum, omentum, or visceral surface of abdominal or pelvic organs.
5. **Heterotopic (< 0.025%):** intrauterine and extrauterine pregnancies existing at the same time

TUBAL ECTOPIC PREGNANCY

Tubal ectopic pregnancy is one of the life threatening obstetric emergencies and a leading cause of maternal death in the first trimester. In many cases it may be followed by tubal damage that affects the patient's future reproductive outcome.

INCIDENCE

Ectopic pregnancy may generally occur in 0.5 to 2% of pregnancies. Tubal ectopic pregnancy constitutes more than 96% of all ectopic gestation.

COMMON SITES for tubal ectopic gestation

1. The Ampulla; the most common site for implantation (80%)
2. The isthmus; less common (12%)
3. The fimbriae; rare (5%)
4. The Interstitial part; rare (3%)

FACTORS PREDISPOSING to tubal ectopic gestation

Any factor interfering with tubal transfer of the fertilized ovum, including:

- Induced narrowing and strictures in the tubal lumen.
- Destruction of a segment of the tubal epithelium
- Peritubal adhesions causing kinking and narrowing of the tube
- Affection of tubal motility causing inadequate tubal transfer
- Rarely occurring congenital factors as; tubal hypoplasia, accessory ostea, and diverticuli.
- Trans-peritoneal migration of the ovum to the contra lateral tube.

AETIOLOGY and RISK FACTORS of tubal ectopic gestation

1. Induced Tubal Narrowing and Strictures

a. Tubal Inflammation (salpingitis):

- *Pelvic Inflammatory Disease (PID); especially gonorrhoeal and chlamydial salpingitis*
- *Puerperal or post-abortive salpingitis*
- *Pelvic peritonitis; in association with appendicitis, generalized peritonitis, etc...*

b. Previous tubal ectopic: causing damage and stricture to a segment of the tubal lumen

c. Tubal stretch: causing narrowing and elongation of the lumen, with entrapment of the dividing morula at the cornual end, as in; broad ligamentary cysts or myomata.

d. Tubal Surgery: as tubal ligation, tubal re-anastomosis, and sometimes attempts at tuboplasty

e. Peritubal adhesions: causing kinking of the tube, commonly occurring in association with;

- *PID and endometriosis*
- *Gynaecologic surgery; as ovarian cystectomy and myomectomy*
- *Pelvic surgery; as appendectomy, if severely inflamed or ruptured*

2. Intrauterine contraceptive device (IUCD): due to its effect on tubal motility and ovum transfer. IUD may also induce and inflammatory reaction with peritubal adhesions. *IUD does not interfere with oocyte fertilization in the fallopian tube*

3. Progestogen only contraception (oral – injectables – implants): have a slightly higher risk of tubal ectopic, compared to non users, due to its effect on tubal motility and ovum transfer.

4. Assisted reproductive techniques (ART): the risk of ectopic pregnancy is increased with the use of drugs to induce super ovulation in ART (IUI, and to a lesser degree in IVF).

5. Cigarette smoking: may cause tubal ciliary dysfunction, thus increasing the risk of ectopic pregnancy, compared to non smokers.

PATHOLOGY OF TUBAL ECTOPIC PREGNANCY

Tubal pregnancy usually becomes **disturbed** in first few weeks after implantation. The narrower the tubal lumen at the site of implantation, the earlier will be the timing of disturbance, due to;

- Decidua is defective and unable to resist invasion by the trophoblast.
- Muscle wall is thin and unable to stretch.

PATHOLOGIC ENTITIES IN TUBAL ECTOPIC

A. Rupture of the pregnancy sac towards the lumen leading to tubal abortion; This is associated with bleeding within the tubal lumen causing one of the following pathologic entities:

- *Haematosalpinx;* tube distended with blood.
- *Tubal mole;* tube distended with old clotted blood including retained remnants of the ruptured gestational sac.
- *Extrusion of gestational sac through the tubal ostium;* associated with intraperitoneal haemorrhage.

B. Tubal Rupture: external rupture of the tube may cause either;

- *Broad ligament haematoma;* rupture towards the floor of the tube into the broad ligament
- *Diffuse intraperitoneal hemorrhage;* rupture through the side walls or roof of the fallopian tube causing intraperitoneal haemorrhage (more common)

C. 2ry Peritoneal Pregnancy; extrusion of viable pregnancy through the tubal lumen, with re-implantation anywhere within the peritoneal cavity (abdominal pregnancy)

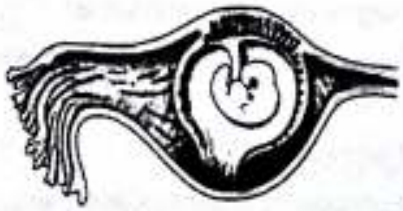


Fig 8:2 Rupture towards the tubal lumen

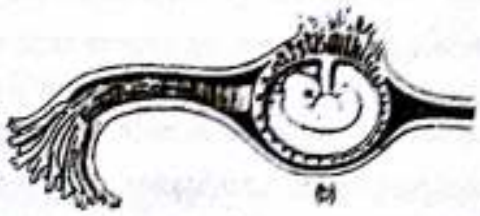


Fig 8:3 External rupture at the roof of the tube

ASSOCIATED UTERINE CHANGES

- The uterus becomes slightly enlarged with softer consistency, due to the effect of ovarian hormones produced by the corpus luteum, and the lining decidua.
- After disturbance of the tubal pregnancy, shedding of the decidua will lead to vaginal bleeding.
- The endometrium on curettage will show decidual reaction with absent chorionic villi

CLINICAL PICTURE OF TUBAL ECTOPIC PREGNANCY

The clinical presentation of a case of ectopic pregnancy may includes either;

A. Undisturbed tubal ectopic pregnancy
B. Ruptured tubal ectopic gestation
C. Chronic cases with pelvic haematocele

A. UNDISTURBED TUBAL ECTOPIC PREGNANCY;

Undisturbed tubal ectopic pregnancy can be easily **missed** or **under diagnosed**. It can be only clinically **suspected** by history and local examination, but diagnosis will only be made after a combination of diagnostic tests including TVS, serum B-hCG, and sometimes laparoscopy.

1. **History** of a short delay in the menstrual cycle
2. **A positive pregnancy test** in urine or blood
3. **Pain:** unilateral lower abdominal or pelvic dull aching pain (due to tubal distension), is usually the main presenting symptom. Pain may be aggravated by exercise, or sexual contact.
4. **Vaginal bleeding:** is usually mild due to shedding of the decidua following pelvic pain.
5. **General examination:** normal pulse, temp. and B.P. (haemodynamically stable)
6. **Abdominal examination:** unilateral mild lower abdominal tenderness

7. Bimanual Pelvic Examination:

- Normal size uterus which is soft in consistency (due to pregnancy hormones)
- Unilateral fullness and tenderness on palpating the vaginal fornices.
- Tenderness on touching or moving the cervix is usually strongly suspicious.

B. DISTURBED ECTOPIC PREGNANCY;

Disturbed ectopic pregnancy is one of the major life threatening obstetric emergencies encountered in the first trimester. If not promptly diagnosed and managed it may lead to maternal morbidity and sometimes mortality.

The clinical presentation differs from undisturbed ectopic pregnancy in the following;

1. Pain: is very prominent in disturbed ectopic pregnancy, and may be;

- Unilateral colicky pain; in cases of tubal abortion
- Unilateral sharp stabbing pain; in association with tubal rupture
- Unilateral shoulder pain; may be present in cases with intraperitoneal haemorrhage due to accumulation of blood under the diaphragm (subphrenic blood collection).

2. Other symptoms; Dizziness, fainting attacks, and palpitation, are associated with disturbed ectopic and intraperitoneal haemorrhage.

3. General examination; according to the severity of intraperitoneal haemorrhage:

If Mild: patients are haemodynamically stable (normal or slightly increase pulse, and normal or slightly lowered B.P.)

If Severe: patients will show the picture of **Hypovolaemic shock**; leading to hypotension with rapidly rising weak pulse, tachypnea, oliguria, and mental confusion.

4. Abdominal examination; picture similar to that of acute abdomen with lower abdominal tenderness and rebound tenderness, guarding, and rigidity.

5. Bimanual Pelvic Examination:

The uterus is normal in size, and soft in consistency

Generalized tenderness at the vaginal vault and fornices

Marked tenderness on touching or moving the cervix.

An adnexal mass may be felt on one side (broad ligament haematoma)

C. CHRONIC ECTOPIC WITH PELVIC HAEMATOCELE

These cases are more difficult to diagnose because;

- A delay in the cycle may not be properly elicited due to a long interval of irregular bleeding, hence a diagnosis of early pregnancy may not be proposed from the start
- Serum B-hCG levels may be very low to be confused with an abortion
- On general examination most cases will be haemodynamically stable
- Abdominal examination reveals mild or absent lower abdominal tenderness
- Bimanual pelvic examination reveals mild tenderness on moving the cervix or palpation of the vaginal fornices, which may be confused with PID.
- A cystic swelling can be palpated in the posterior fornix (pelvic haematocele), which may be confused with a pelvic abscess.
- Most cases will be diagnosed at laparoscopy for chronic pelvic pain and an adnexal mass

SPECIAL INVESTIGATIONS IN ECTOPIC PREGNANCY:

1. Serum B-hCG:

- *A single serum B-hCG* assay is positive for pregnancy, but at levels lower than expected for gestational age according to the LMP.
- *Repeat B-hCG* will show failure of levels to increase > 66% when repeated after a 48 hour interval (Abnormal **serum B-hCG doubling time)**

2. Combined US and B-hCG discriminatory zone; may show the following:

- ***B-hCG discriminatory zone:*** serum B-hCG level at which all viable intrauterine pregnancies will be visualized by US (>2000 m IU/ml by TVS, and >3500 m IU/ml by TAS).
- *Absence of an intrauterine GS with serum B-hCG levels above the discriminatory zone,* is considered the **gold standard test** in diagnosis of ectopic pregnancy
- *Other findings that may be encountered on **TVS;***
  a. Presence of an associated adnexal mass (haematosalpinx, or tubal mole)
  b. Detection of an extrauterine GS or an embryo (rarely elicited although diagnostic)
  c. Fluid in Cul-de-sac or diffuse intraperitoneal haemorrhage (ruptured ectopic)

3. Diagnostic laparoscopy:

- Laparoscopy is conclusive whenever diagnosis is still in doubt. It allows;
- Direct visualization of the fallopian tubes for diagnosis of ectopic gestation.
- Accurately detecting the site of ectopic (ampullary, isthmic, fimbrial, or interstitial)
- Differentiation between undisturbed ectopic with an intact tube, tubal rupture, and chronic ectopic with or without pelvic haematocele.
- Underlying associated or predisposing pathology as endometriosis or PID.
- Most cases of ectopic pregnancy can be successfully managed via operative laparoscopy as described later

DIFFERENTAL DIAGNOSIS OF ECTOPIC PREGNANCIES

a. **Spontaneous abortion:** Bleeding precedes pain and is more prominent, while B-hCG levels are low and declining

b. **Haemorrhagic corpus luteum:** short delay in a cycle with unilateral pain and adnexal mass on US, but pregnancy test is negative

c. **Adnexal torsion:** unilateral lower abdominal pain in association of an adnexal mass on US, without amenorrhoea or bleeding. Pregnancy test is negative

d. **Appendicitis:** pain in right iliac fossa with tenderness and rebound tenderness, not related to menstrual cycle. Pregnancy test is negative, and a CBC shows high TLC.

e. **Pelvic inflammatory disease (PID):** pelvic pain with tender cervix and adnexa in absence of amenorrhoea. Pregnancy test is negative, and CBC may show high TLC.

f. **Pelvic haematocele:** may be associated with haemorrhage and rupture in corpus luteum cyst. Pregnancy test is negative, and so is tenderness in moving the cervix. CBC may show anaemia due to blood loss with a normal TLC.

MANAGEMENT OF TUBAL ECTOPIC PREGNANCY

A. SURGICAL MANAGEMENT

- Most cases of tubal ectopic pregnancy will be managed surgically.
- Cases presenting as disturbed ectopic with shock and/or severe internal haemorrhage should first receive anti-shock measures including; fluid, and blood transfusion, until the general condition is stabilized before surgery is attempted.

1. Salpingostomy: describes performing a linear incision on the anti-mesenteric border of the distended part of the fallopian tube, and extrude all of the intratubal contents.

- **Indications:** in early discovered cases in which tubal damage Is minimal and the patient is haemodynamically stable.
- **Advantage:** managing ectopic while conserving the tube to preserve its function.
- **Disadvantage:** postoperative scarring may cause narrowing of the tubal lumen at the site of salpingostomy incision predisposing to recurrence of ectopic in the same tube

  a. ***Laparoscopic salpingostomy:*** is best choice in early cases of undisturbed tubal ectopic pregnancy, especially with a small tubal mass (<2.0 cm).

  b. ***Salpingostomy via laparotomy;*** is performed whenever laparoscopy was not available, or if the tubal mass was large, or haemorrhage was significant.

2. Salpingectomy: describes excision of the fallopian tube affected by ectopic pregnancy.

- **Indications:** in cases with extensive tubal damage, where sparing the tube is useless or impossible, as with tubal rupture, large tubal masses, or chronic ectopic gestation.

  a. ***Laparoscopic salpingectomy:*** is performed in haemodynamically stable patients

  b. ***Salpingectomy via laparotomy:*** in cases with severe intraperitoneal haemorrhage especially in haemodynamically unstable patients, where laparoscopy will carry an additional risk to the patient.

c.

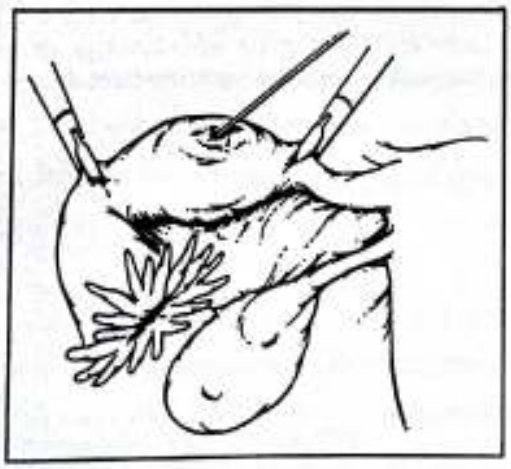


Fig 8:4 Salpingostomy

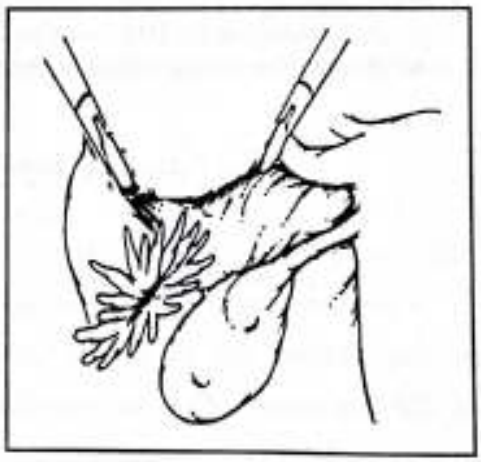


Fig 8:5 Salpingectomy

N.B.: In all surgically treated cases follow up by serum B-hCG is recommended to ensure decreasing B-hCG levels and exclude persistent trophoblastic activity.

MEDICAL APPROACH WITH METHOTREXATE

A medical approach using **Methotrexate (MTX)** may be chosen for certain highly selected cases,

- *Mechanism of action:* MTX acts s a Folic acid antagonist that interferes with DNA synthesis, especially in the rapidly dividing trophoblastic cells.
- *Advantage:* avoiding surgery and conserving the fallopian tube
- *Disadvantage;* it may take 3-4 weeks for an ectopic to resolve, thus needs careful follow up
- *Patient selection:* undisturbed early ectopic gestation with a small tubal mass < 3.0 cm, low serum B-hCG levels <500 m IU/ml, and absent embryonic cardiac pulsations.
- *Dosage:* single I.M. dose of 50 mg/m2
- *Follow up;* serial serum B-hCG daily until levels start to fall then weekly till a nil value is obtained
- *Success rates;* 73-93% success rates have been reported in carefully selected cases.
- *Treatment failures;* laparoscopic surgical management is usually necessary
- *Side effects* (5% of cases); GIT symptoms as nausea, vomiting, diarrhea, and stomatitis.
- *Contraindications;* during lactation, and in women with hepatic or renal disease, or immunodeficiency syndromes.

PROGNOSIS AFTER TUBAL ECTOPIC GESTATION

A. Fertility: future fertility is best preserved in cases with early undisturbed ectopic pregnancy managed with **conservative laparoscopic surgery** (salpingotomy), or by **Methotrexate** treatment. Almost 80% of patients will successfully achieve pregnancy after an ectopic pregnancy.

B. Recurrence: almost 27% of women who achieve pregnancy after an ectopic gestation will have another ectopic pregnancy.

N.B.;

- Rh - ve cases should receive an anti-D Rh immunoglobulin dose if the husband is Rh +ve
- Early detection of ectopic pregnancies with the combined use of TVS and serum B-hCG, and prompt management with medical and surgical techniques (including laparoscopy), resulted in minimizing the serious consequences of ectopic pregnancy over the past few decades.

RARE TYPES OF ECTOPIC PREGNANCY

OVARIAN ECTOPIC PREGNANCY

- **Incidence;** is rare (<2.0% of ectopic gestations)
- **Implantation site:** the fertilized oocyte will be implanted on the ovarian surface.
- **Clinical presentation:** same as tubal ectopic pregnancy.
- **Diagnosis:** is established only at laparoscopy or laparotomy, where the pregnancy sac will be seen occupying the ovary, being attached to the uterus by the ovarian ligament, with the tube on the affected completely free (Spiegelberg criteria).
- **Management:** is by laparotomy or laparoscopy, where the ovary in most cases will be removed (oophorectomy), or in selected cases preserved after removal of embryonic tissue.

PREGNANCY IN A RUDIMENTARY UTERINE HORN

- **Implantation site:** congenital rudimentary horn of the uterus (very rare)
- **Clinical presentation:** persistent lower abdominal pain in early pregnancy
- **Diagnosis:** suspected by TVS, usually late 1st trimester, where an adnexal mass is seen attached to the myometrium as one horn, and not connected to a cervix.
- **Differential diagnosis:** from interstitial ectopic pregnancy by being medial to the insertion of the round ligament at the uterine cornu.
- **Management;** surgically by cornual resection via laparotomy

CERVICAL ECTOPIC PREGNANCY

- **Incidence:** very rare (< 0.5% of all ectopic pregnancies)
- **Implantation site:** The fertilized ovum is implanted in the cervical canal below the internal os
- **Clinical presentation:** persistent bleeding in early pregnancy with pelvic pain
- **PV examination:** the cervix is soft, enlarged, and barrel shaped.
- **Diagnosis:** TVS will show all pregnancy contents in the cervical canal with an empty endometrial cavity and a closed internal os.

Management:

1. Young patients desirous of further fertility can be managed medically by IM MTX injection followed by gentle manual separation of content sunder anaesthesia.
2. Elderly multiparous patients, not desiring further pregnancies can be managed surgically by total abdominal hysterectomy to avoid severe bleeding from the cervix.

ADVANCED EXTRAUTERINE PREGNANCY

- This is the rarest type of ectopic pregnancy, with an unknown location that may be anywhere in the pelvic or abdominal peritoneal cavity.
- Pregnancy may advance to 2nd trimester in some cases, with a great risk of severe life threatening intraperitoneal haemorrhage
- Once suspected or diagnosed immediate laparotomy is indicated with excision of the pregnancy sac from the surrounding adherent tissue.

Key points in ectopic pregnancy

- *Tubal ectopic is the commonest type of ectopic pregnancy.*
- *The ampullary portion of the tube is the commonest site where tubal ectopic occurs*
- *Tubal ectopic may clinically present as undisturbed tubal ectopic, rupture tubal ectopic, or chronic ectopic with pelvic haematocele*
- *Undisturbed ectopic pregnancy is usually diagnosed through a high degree of clinical suspicion, and combined investigations including TVS serum B-hCG, and sometimes laparoscopy.*
- *Most cases of tubal ectopic pregnancy are managed surgically.*
- *Laparoscopic salpingostomy is the best choice for early cases of undisturbed ectopic. A few cases can be managed medically by MTX.*
- *Cases of disturbed ectopic pregnancy are usually associated with tubal rupture and damage together with variable degree of intraperitoneal haemorrhage. Such cases will require salpingectomy after stabilizing their general condition*
- *Some cases may suffer future fertility problems especially those with tubal damage and peritubal adhesions.*
- *Other rare types of ectopic pregnancy include; cervical, ovarian, abdominal, interstitial, and heterotopic ectopic pregnancies*

9 GESTATIONAL TROPHOBLASTIC DISEASE

Terminology and classifications
- *Histologic Classification*
- *Clinical Classification*

HYDATIDIFORM MOLE
- *Incidence & risk factors*
- *Origin & Karyotype*
- *Pathology of the uterus*
- *Associated Ovarian Pathology*
- *Symptoms and Signs*
- *Special investigations*
- *Complications of GTD*
- *Management of molar pregnancy*
- *Recurrence and future fertility*

GESTATIONAL TROPHOBLASTIC TUMOUR (GTT)
- *Histopathologic classification*
- *Clinical classification*

Diagnosis of GTT

Special investigations

Management of GTT
- Non Metastatic
- Metastatic

Recurrence rates

Future fertility

GESTATIONAL TROPHOBLASTIC DISEASE (GTD)

GTD is the general term describing a spectrum of proliferative abnormalities originating from the trophoblastic cells of the placenta i.e.; abnormal placental trophoblastic proliferation.

HISTOLOGIC CLASSIFICATION:

- **Hydatidiform mole** *(benign form)*
- **Invasive mole** *(locally malignant form)*
- **Choriocarcinoma** *(malignant form)*

CLINICAL CLASSIFICATION:

Hydatidiform Mole (benign GTD)	Gestational Trophoblastic Tumour GTT (malignant GTD)
- Complete Mole	- Non Metastatic GTT
- Partial Mole	- Metastatic GTT
	A) Low Risk (good prognosis)
	B) High Risk (poor prognosis)

HYDATIDIFORM MOLE

Hydatidiform Mole is the most commonly encountered **benign** form of GTD, also known as vesicular mole. It may be either **complete** (classic), or **incomplete** (partial).

Fig 9:1 Hydatidiform mole

INCIDENCE AND RISK FACTORS

- **Geographic distribution:** molar pregnancies are more common in some parts of Asia with an incidence of 1/125 pregnancies. In USA and Europe it is rare affecting only **1/1500** pregnancies.
- **Age factor:** increased risk 5-10 times with advanced maternal age > 40 years.
- **Dietary factors;** increased risk with low intake of vitamin A and animal fat
- **Recurrence risk:** the risk of recurrence in a subsequent pregnancy is around 1-2%

ORIGIN AND KARYOTYPE

A. Complete Mole

- In 90% of cases, an **abnormal empty ovum** (in which the chromosomes are either absent or inactivated) is fertilized by **one sperm**, which duplicates its DNA leading to an abnormal 46XX karyotype, with all DNA **paternal** in origin.
- In 10% of cases, an **empty ovum** will be fertilized by 2 sperms, resulting in abnormal 46XX, or 46XY karyotype, with all DNA again **paternal** in origin.

B. Partial or Incomplete Mole

- A partial mole is a **normal ovum** that is fertilized by 2 sperms.
- The resulting karyotype is 69XXX, 69XXY or 69XYY (triploidy).
- Many spontaneous abortions may represent undiagnosed partial moles

PATHOLOGY OF GTD

Hydatidiform mole is one form of abnormal trophoblastic proliferation, causing the uterine cavity to be distended with grape like thin-wall structures (vesicles), secreting large amounts of hCG.

A. COMPLETE MOLE

- ***Gross Morphology:***
  - The chorionic villi are converted into a mass of clear vesicles
  - Size of vesicles vary from few millimeters to a few centimeters in diameter
  - Absence of amniotic sac or foetal tissue within the uterus
- ***Histologic Features:***
  - Marked oedema and enlargement of the villous stroma (hydropic villi)
  - Disappearance of the villous foetal blood vessels
  - Diffuse proliferation of the trophoblastic cells

B. PARTIAL MOLE

Gross morphology:

- Hydatidiform changes are only focal and not generalized
- A foetus or amniotic sac can be identified
- When a fetus is present, it is usually non viable, and exhibits the stigmata of triploidy including multiple congenital malformations and growth restriction.

Histologic Features:

- Only focal hydropic villi
- Only focal trophoblastic proliferation
- Presence of umbilical cord, amniotic membrane, or foetal tissue

ASSOCIATED OVARIAN PATHOLOGY

- Ovarian theca lutein cysts will occur in 25-60 % of **complete moles**, but occur much less commonly in association with partial moles
- Theca lutein cysts represent a form of ovarian hyperstimulation in response to the huge amounts of hCG secreted by the abnormally proliferating trophoblastic cells.
- Cysts are usually bilateral, varying in size from few mm up to 10 cm in diameter. They have a smooth surface, yellowish in colour, and are lined by lutein cells
- Most theca lutein cysts will undergo spontaneous regression within 2-4 months of molar evacuation, although large cysts may undergo torsion, infarction and hemorrhage.

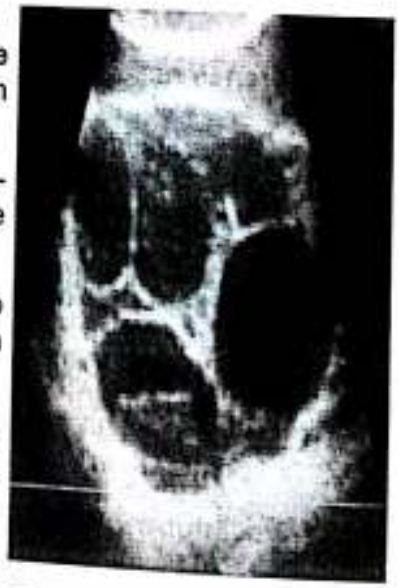


Fig 9:2 associated ovarian theca lutein cysts

CLINICAL PICTURE OF MOLAR PREGNANCIES

Most cases of GTD will be first present as bleeding in early pregnancy, and will mostly be misdiagnosed as threatened abortion. Symptoms and signs are non specific, and the final accurate diagnosis will be only achieved via ultrasonography in addition to serum b-hCG levels.

SYMPTOMS AND SIGNS

1. **Severe nausea and vomiting:** early in pregnancy due to abnormally high levels of E2, P, & hCG.
2. **Recurrent mild vaginal bleeding:** is the most common presenting symptom in the first trimester.
3. **Dark prune juice vaginal discharge:** may occur due to separation of molar tissue from the decidua, disruption of the maternal vessels, and rupture of some vesicles.
4. **Pain:** is usually absent or minimal in most cases, however some cases may experience;
   a. Dull aching pain; due to rapid uterine distension
   b. Colicky pain; due to uterine contractions associated with expulsion of the mole
   c. Sudden severe sharp pain; in association with concealed accidental haemorrhage, invasive mole, perforation and rupture, or torsion of a theca lutein cyst of the ovary
5. **General Examination:**
   - Pallor in association with anaemia due to recurrent or severe blood loss
   - Signs of PE may be present in cases reaching the 2nd trimester (HTN + albuminuria)
6. **Abdominal Examination:**
   - *Fundal level:* larger than calculated gestational age; due to the rapidly proliferating vesicles
   - *Consistency:* uterus is soft and doughy due to absence of amniotic fluid
   - *Foetal parts* and *foetal pulsations* are absent in complete moles past the first trimester
7. ***Vaginal Examination:***
   - The cervix is soft in consistency
   - Enlarged ovaries with theca lutein cysts may be palpable in the pouch of Douglas
   - Passage of vesicles: in association with vaginal bleeding is diagnostic of the condition

SPECIAL DIAGNOSTIC INVESTIGATIONS

A. <u>Ultrasonography</u>;

This is the **gold standard** in diagnosis of molar pregnancy

- ***Complete Mole:*** characteristic **snow storm appearance**, due the presence of vesicles surrounded by blood, in absence of foetal echoes
- ***Partial mole;*** shows embryonic or foetal echoes, amniotic sac, and a placenta partially replaced by vesicular structures.
- ***Ovarian theca lutein cysts:*** if present will appear as multiple thin walled cysts of variable size.

B. <u>Serum B-hCG</u>;

abnormally high serum B-hCG levels, which may reach > 100.000 m IU/ml in the first 8-10 weeks gestation

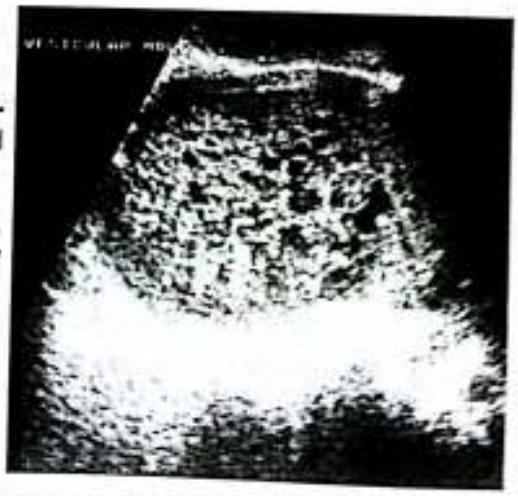


Fig 9:3 TAS of a complete hydatidiform mole (Snow storm appearance)

DIAGNOSTIC WORK UP FOR CASES OF GTD

Once diagnosis of a hydatidiform mole has been established, a work up of further special investigations is immediately started in order to exclude metastases, to asses the risk of developing malignant disease (GTT), and to prepare the patient for surgical evacuation. These include;

1. **Laboratory tests:**
   - Serum B-hCG: abnormally high levels are suspicious of GTT
   - CBC, coagulation studies, baseline renal and liver function tests (preoperative test)
   - Thyroid function tests if clinical hyperthyroidism is suspected (see later)
2. **Abdominal ultrasound:** to exclude liver metastases
3. **Chest X-ray:** to exclude lung metastases
4. **CT or MRI :** to exclude brain metastases

COMPLICATIONS ASSOCIATED WITH BENIGN GTD (Hydatidiform mole)

1. Hyperemesis gravidarum: should raise suspicions for diagnosis of a molar pregnancy
2. Early onset pregnancy induced hypertension (Gestational HTN or PE preeclampsia < 24 wks)
3. Hyperthyroidism: encountered in 7% of cases due to thyroid stimulating effect of HCG. Serum levels of T3 & T4 may be elevated
4. Severe bleeding which may be life threatening
5. Trophoblastic embolization to the lungs causing RDS
6. Complications of ovarian cysts (torsion, haemorrhage, rupture...)
7. Risk of developing an invasive Mole (20% in a complete mole, and 4% in a partial moles)
8. Risk of developing Choriocarcinoma (5% of cases)

RISK FACTORS FOR DEVELOPING MALIGNANT GTD (GTT)

- Age > 40 years
- History of a previous molar pregnancy
- Pretreatment B-hCG levels > 100.000 m IU/ml
- Theca lutein cysts > 6.0 cm

MANAGEMENT OF MOLAR PREGNANCIES (complete and incomplete)

A. Dilatation and Suction Evacuation:

- This is the gold standard in evacuating molar pregnancies, even those > 20 weeks size.
- Vaginal or oral PGL E1 (Mesoprostol) started 24 hours prior to SE may help dilatation of the cervix, and initiation of uterine contractions
- I.V. oxytocin should be given during evacuation to enhance uterine contractility and minimize post evacuation blood loss.
- Ergometrin or PGL are given postoperative to guard against post partum haemorrhage

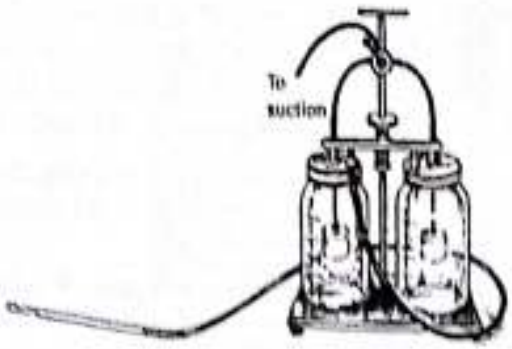


Fig 9.4 Suction evacuation apparatus

B. Hysterectomy:

Abdominal hysterectomy has a limited place in the management of hydatidiform mole.

It may be indicated in the following conditions;

- Older multiparous patients desiring sterilization
- Multiparous patients in cases complicated by severe haemorrhage or infection.
- Invasive mole causing uterine perforation and severe intraperitoneal haemorrhage.

N.B.; *Hysterectomy does not cure or prevent metastases therefore after hysterectomy is performed cases will require follow up by serial serum B-hCG similar to that performed after molar evacuation.*

C. Management of Ovarian Theca Lutein Cysts;

a. Conservative management: most cysts will undergo spontaneous regression

b. Complicated cysts: torsion, rupture, or severe haemorrhage will necessitates laparotomy, and management according to guidelines for ovarian cysts.

FOLLOW UP AFTER EVACUATION OF MOLAR PREGNANCIES

After evacuation of a molar pregnancy, serum B-hCG levels are expected to return to undetectable levels in 9-11 weeks. During this period the following follow up protocol is recommended;

a. **Repeated serum B-hCG** assays made at 48 hours post-evacuation, then weekly till until results are negative for 3 successive weeks, then monthly for 6 months.

b. **Physical examination** and **pelvic US** at regular monthly intervals; to ensure complete evacuation, normal involution of pelvic organs, and disappearance of theca lutein cysts

c. **Birth control;** for 1 year; best using combined oral contraceptive pills (COCs), or progesterone only pills (POPS), progestin I.M. injectables, or subdermal implants.

d. **Role of chemotherapy;**

- Most cases will be cured after suction evacuation, and will not require further therapy
- Patients who develop GTT after a complete mole or a partial mole, as evidenced by plateau or rising hCG levels, or failing to reach negative values in 11 weeks, will require an immediate metastatic work up and start of chemotherapy (see later in GTT)
- Chemotherapy should not be used as a routine prophylactic measure due its possible side effects and toxicity

Future fertility and risk of recurrence

- Normal pregnancy will most likely follow a molar pregnancy.
- The risk of a second molar pregnancy is 1-2 %, while risk of a third one is 33%.
- Pregnancy is allowed after 6-9 months of a negative B-hCG value on the follow up protocol

GESTATIONAL TROPHOBLASTIC TUMOUR (GTT)

Gestational trophoblastic tumour (GTT) is the **malignant** form of GTD, characterized by their aggressive invasion into the myometrium, and their tendency to metastasize.

INCIDENCE

GTT is identified in **1/20.000** pregnancies. It can occur after any type of pregnancy, as follows;

- Following hydatidiform in 50% of cases
- Following abortion in 25% of cases
- Following normal pregnancy in 25% of cases

HISTOPATHOLOGICAL CLASSIFICATION

A. **Invasive Mole:** It develops following 20% of all molar pregnancies, always arising from partial or complete moles. They are characterized by extensive tissue invasion and deep myometrial penetration by trophoblastic cells and whole villi. They are locally invasive tumours that lack the tendency to metastases

B. **Gestational choriocarcinoma:** this extremely malignant tumour is considered a carcinoma of the chorionic epithelium. It occurs in 1/30.000 pregnancies, with 50% developing after a molar pregnancy, 25% following an abortion, and 25% following a term pregnancy. They are usually associated with early blood borne metastases, mostly to the lungs (75%), and the vagina (50%). Ovarian theca lutein cysts are identified in over 1/3 of cases

- *Grossly:* rapidly growing dark red friable mass invading both myometrium and blood vessels, causing haemorrhage and necrosis.
- *Microscopically:* columns and sheets of trophoblastic cells invading the myometrial muscles and blood vessels. There is complete loss of the villous pattern

C. **Placental site trophoblastic tumour:** a rare variant arising from placental implantation site usually following a term pregnancy. It is mainly formed of intermediate trophoblast that produces small amounts of B-hCG. It is a locally invasive tumour that is generally resistant to chemotherapy.

CLINICAL CLASSIFICATION OF GTT

- **Non Metastatic:** disease confined to the uterus (most common form of GTT)
- **Metastatic:** disease spread outside the uterus to the lung, vagina, brain, and liver

Metastatic Low risk (Good Prognosis)	Metastatic high risk (Poor Prognosis)
- Short duration < 4 month	- Long duration > 4 months
- Pretreatment hCG < 40.000 m IU/ml	- Pretreatment hCG > 40.000 m IU/ml
- No previous chemotherapy.	- Failure of previous chemotherapy
- No spread to brain or liver	- Disease spread to brain or liver
	- Disease after full term pregnancy

CLINICAL PRESENTATION OF METASTATIC GTT; according to the site of metastases:

- Lungs: in 80% of cases, presenting by haemoptysis
- Vagina: in 30% of cases, presenting by vaginal bleeding
- Brain: in 10% of cases, showing neurologic symptoms & increased Intracranial tension ICT
- Liver: in 10% of cases, mostly asymptomatic

DIAGNOSIS OF GTT

GTT is suspected if **after termination of a pregnancy** (hydatidiform mole, abortion, or full term delivery), one or more of the following occurs

A) **Persistent vaginal bleeding** continues without US evidence of retained products of conception, and/or

B) Follow up reveals an **increase in serum B-hCG levels** or **failure to plateau in 3 weeks**

Non Metastatic Disease (invasive Mole); is further diagnosed by TVS, Doppler US, and MRI, to detect the presence and depth of invasion of the myometrium by trophoblastic cells. Diagnosis is established without the need for a biopsy specimen for histopathologic confirmation.

Metastatic GTT; is identified by naked eye examination of vaginal metastases, or by US and radiological studies to detect liver, lung, and brain metastases. Metastatic lesions are friable and bleed easily, hence biopsy to confirm diagnosis is not recommended before starting treatment.

MANAGEMENT OF GTT

A. <u>Non Metastatic Disease</u>

1. **Methotrexate (MTX):** MTX is an anti-folic acid that acts by arresting synthesis of DNA, RNA, and proteins. It is the most commonly used **single agent chemotherapy** drug with success rates > 90%
   - Dosage: 50 mg/m2 given as IM injections
   - Side effects include; nausea, oral and gastric ulcers.
   - Toxicity: myelosuppression, hepatotoxicity, nephrotoxicity, alopecia, and pneumonitis
   - Leucovorin (folinic acid); is administered 24 hours after each MTX dose to rescue normal cells from MTX toxicity. Folic acid should be avoided as it interferes with MTX action
2. **Actinomycin D (Act-D):** is an antibiotic that acts on DNA strands, and can be used as a single agent chemotherapy alternative to MTX

Follow up after single agent chemotherapy treatment is essential by serial B-hCG weekly until normal levels are obtained, then monthly for six months.

Failure of single agent chemotherapy in non metastatic disease is rare. It is diagnosed by failure of B-hCG levels to return to normal values during post treatment follow. Management is usually successful through the use of multi drug regimens.

Hysterectomy may be offered to elderly multiparous patients with no desire for future fertility.

Recurrence rates for non metastatic GTT is **< 1%**

B. Metastatic GTT

- Metastatic **Low risk GTT (Good Prognosis): is managed by;**
  - *Single agent chemotherapy (*MTX or Act-D) but with more frequently repeated courses.
  - Follow up as in non metastatic GTT (weekly for 3 months, then monthly for 6 months)
  - Recurrence rates: around 5%
- Metastatic **high risk GTT (Poor Prognosis): is managed by;**
  - *Multi agent chemotherapy;* Etoposide, MTX, Act-D, Cyclophosphamide, and Oncovin.
  - Follow up as in non metastatic GTT, followed by *three additional courses* of chemotherapy after B-hCG becomes negative.
  - Surgery and radiotherapy may have a role in some cases.
  - Recurrence rates: may reach up to 20%

FUTURE FERTILITY

- Contraception by OCPs is recommended for 1 year after negative hCG levels
- Pregnancy should be monitored in first trimester to detect recurrence
- HCG levels should be followed after delivery till they reach zero postpartum

Key Points

- ***GTD are classified into:***
  A) Benign GTD (hydatidiform moles), which may be Complete or Partial moles.
  B) Malignant GTT; may be Non metastatic or Metastatic GTT which is either good prognosis or poor prognosis.
- ***Complete hydatidiform moles** are the commonest encountered form of GTD. They have a 46XX, or 46XY karyotype with all DNA of paternal in origin*
- *Most cases present as threatened abortion until diagnosis is made by abnormally elevated hCG levels together with a characteristic picture on ultrasonography*
- *Management is by suction evacuation and follow-up for 3 months using serial B-hCG titer.*
- *A new pregnancy Is not allowed before 6-9 months from a negative hCG titer*
- *Persistent vaginal bleeding after molar evacuation with high B-hCG titer or a plateau for 3 months is the main presentation for malignant GTT.*
- *A metastatic work up including hCG, blood chemistry, radiography, chest X ray, brain CT or MRI, will allow classification of cases into non metastatic and metastatic low risk or high risk.*
- ***Non metastatic GTT** are treated successfully with single agent chemotherapy.*
- ***Metastatic low risk GTT** are managed by single agent chemotherapy but on more frequent repeated courses*
- ***Metastatic high risk** cases are treated with combination chemotherapy (EMA CO), with cure rated > 80%*
- *Hysterectomy is indicated in selected in elderly multiparous women who do not require further childbearing.*

10 ANTEPARTUM HAEMORRHAGE

Causes of APH

PLACENTA PRAEVIA

- Definition
- Incidence
- Classifications
- Aetiology & Risk factors
- Mechanism of bleeding
- Pathology of PL PRV
- Clinical picture
- Diagnosis & DD
- Management
  a. *Expectant*
  b. *Active*
- Complications

Placenta Accreta

PLACENTAL ABRUPTION

- Definition
- Incidence
- Clinical types of bleeding
- Aetiology & Risk factors
- Pathology
- Clinical picture
- Diagnosis
- Management

Rupture Vasa Praevia

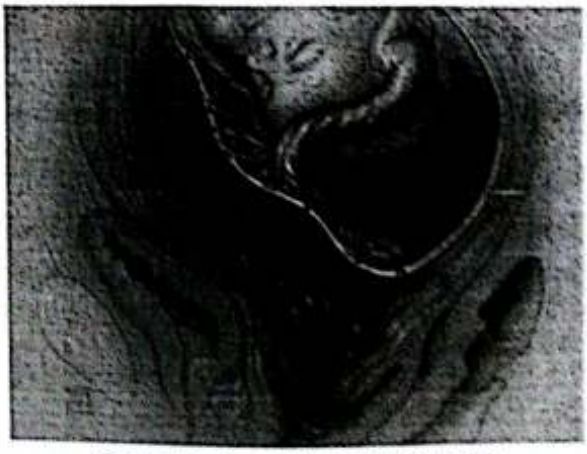
INTRODUCION

Antepartum haemorrhage (APH) describes vaginal bleeding occurring after viability of the foetus (i.e.; from 20 weeks gestation till delivery).

APH is considered one of the most serious obstetric emergencies, being a leading cause of **maternal morbidity** and **mortality** that also carries an adverse effect on the **neonatal outcome.**

CAUSES OF ANTEPARTUM HAEMORRHAGE

1. **Placenta Praevia:** separation of an abnormally low implanted placenta
2. **Placental Abruption:** separation of a normally implanted placenta
3. **Rupture Vasa Praevia:** rupture of foetal vessels due to velamentous cord insertion (very rare)
4. **Abnormal Genital Tract Bleeding:** non obstetric cause are less commonly encountered:
   - Benign conditions of the cervix; as erosion and polyps.
   - Cancer of the cervix with pregnancy (rare).
   - Vaginal trauma (rare)



Complete Centralis (Total PL PRV)

PLACENTA PRAEVIA

(PL PRV)

DEFINITION

PL PRV is a placenta that encroaches partially or totally on the lower uterine segment (LUS).

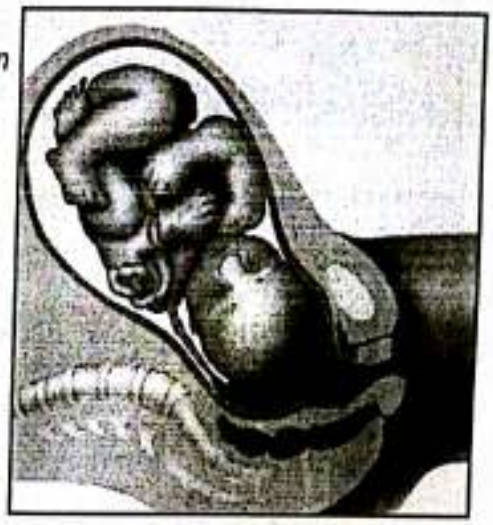


Fig 10;1 Placenta praevia

INCIDENCE

- It is uncommon occurring in around 1/200 – 1/300 deliveries.
- The incidence is increasing in the last 2 decades due to
  - Increasing rates of delivery by CS
  - Higher number of pregnancies at an older maternal age

CLASSIFICATIONS

- PL PRV has been classically classified into 4 grades:
  1. ***Complete Centralis (Total PL PRV):*** where the placenta completely covers the internal os
  2. ***Incomplete Centralis (Partial PL PRV):*** where the placenta partially covers the internal os
  3. ***Marginalis (Marginal PL PRV):*** the lower edge of the placenta reaches but does not cover the margin of the internal os. It could be implanted on the LUS either anteriorly or posteriorly.
  4. ***Lateralis (Low Lying Placenta):*** the lower placental edge encroaches on the LUS, but does not reach the margin of the internal os. This type is of little clinical significance.

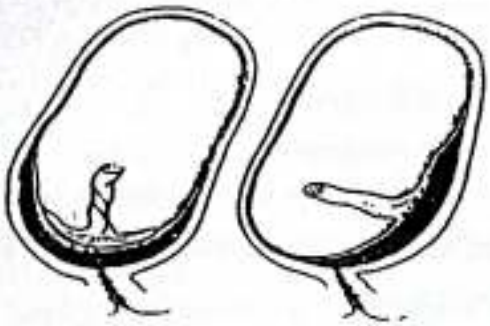


Fig 10:2 Complete and incomplete Centralis (Major PL PRV)

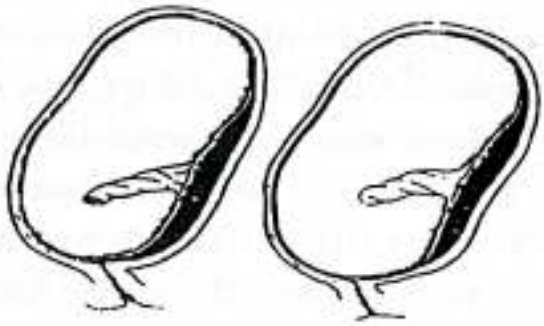


Fig 10:3 Marginalis and Lateralis (Minor PL PRV)

- Recently a more simplified classification divides PL PRV into:
  a. ***Major PL PRV;*** where the placenta is covering the internal cervical os (total or partial)
  b. ***Minor PL PRV;*** where the placenta is sited within the LUS but not covering the internal os (marginal or lateral)

N.B.; Low lying and marginal PL PRV are more commonly encountered than total or central PL PRV

AETIOLOGY AND RISK FACTORS

1. **Advancing maternal age:** incidence reaches > 1% at ages 35 years and more.
2. **Multiparity:** incidence reaches > 2% in women para 5 or greater.
3. **Prior C.S.:** incidence rises from 0.5% for prior one C.S. reaching up to 3.5% in prior 5 C.S.
4. **Multifetal pregnancy MFP:** the incidence doubles in twin pregnancy, and triples in MFP.
5. **Other causes of a large placenta:** as bipartite or multipartite (multiple lobes).

MECHANISM OF BLEEDING in PL PRV

In the second half of pregnancy the relatively inelastic placenta cannot keep pace with the progressive stretching of the LUS due to the accelerated foetal growth. Inevitable separation of the low lying placenta then occurs leading to unavoidable bleeding especially in the third trimester (APH).

The nearer the placenta to the internal os the more will be the risk of;

- Early placental separation and severe APH
- Termination of pregnancy before foetal maturity (preterm delivery).
- Delivery by CS even if the foetus Is dead or non viable

PATHOLOGY OF PLACENTA PRAEVIA

1. **The LUS;** is thin vascular and friable, thus more liable to lacerations.
2. **The placenta:** implanted on the LUS (partially or completely). It may also reach the internal cervical os and cover it (partially or completely).
3. **Placenta Accreta;** higher risk of occurrence due to poor decidual reaction in the LUS, especially at the site of a previous CS scar (see later)
4. **The umbilical cord:** higher incidence of velamentous insertion of the cord and vasa praevia.

CLINICAL PICTURE OF APH DUE TO PL PRV

PL PRV by itself is a symptomless condition until the first episode of bleeding starts usually towards the beginning of the third trimester.

In PL PRV bleeding is from **maternal** and not fetal circulation, and is more likely to compromise the mother's general condition before it affects the foetus.

- SYMPTOMS:
  - **Painless, causeless, and recurrent** bleeding is the classic symptom in APH due to PL PRV
  - Bleeding is usually **bright red** in colour (recently occurring)
  - The first attacks of vaginal bleeding are usually **mild**, but recurrent attacks may be **severe**
  - If labour pains start severe bleeding may cause **hypovolaemic shock** in a short period of time

- SIGNS ON CLINICAL EXAMINATION:

A. General Examination:

- **Single** attack of **mild APH**; the general condition is usually not affected.
- **Recurrent** or **moderate** attacks of **APH**; pallor and anaemia are usually present
- **Severe attack of APH:** signs of hypovolaemic shock may be evident including; Drowsiness, delirium, loss of consciousness, tachycardia, tachypnea, and hypotension (rapid and rising pulse, high RR, low and falling BP). The general condition is in proportion with the amount of vaginal bleeding.

B. Abdominal Examination

- **Fundal level:** corresponds to the calculated gestational age with the abdomen usually lax
- **Foetal parts;** are easily felt, with malpresentations being common; since the low lying placenta will interfere with normal adaptation of foetal head to maternal pelvis
- **Foetal heart pulsations;** are usually audible by the Duplex instrument; as bleeding in PL PRV is mainly maternal in origin. However in cases with severe and recurrent APH, foetal distress and sometimes IUFD may occur

C. Vaginal Examination

- PV examination is generally **CONTRAINDICATED** in cases of PL PRV to avoid further placenta separation that will provoke more severe attacks of vaginal bleeding.
- PV examination may be performed only when **active management** is decided in order to choose between a trial for vaginal delivery versus performing an elective C.S., especially in cases in which labour pains have already started and bleeding is minimal.
- PV examination should be done **ONLY** in a well prepared operating theater under complete aseptic conditions, with anaesthesia, blood transfusion, and a team ready to perform an immediate C.S. (PV examination should NOT be done at home or even in hospital outpatient or emergency room).

- SPECIAL INVESTIGATIONS

Ultrasonography (U.S.) is the **gold standard** in diagnosis of **PL PRV**, with the following benefits.

- Early diagnosis of low lying placenta with high accuracy: as early as the mid-second trimester, even before most cases will start to bleed.
- Diagnosis of partial and complete types of PL PRV; as early as the beginning of 3rd trimester.
- Exclusion of placental abruption and vasa praevia; as causes for APH.
- Other benefits of US: include accurate estimation of fetal gestational age, amniotic fluid volume, assessment of fetal well being, and exclusion of major fetal anomalies.

DIFFERENTIAL DIAGNOSIS of APH due to PL PRV:

- Other obstetric causes of APH (placental abruption and rupture vasa praevia)
- Bleeding to local gynaecologic conditions (cervical polyp, cervical erosion, or cancer cervix)
- Bleeding due to general and systemic causes (severe hypertension, thrombocytopenic purpura, etc.)

MANAGEMENT OF PLACENTA PRAEVIA

Management of PL PRV is either **EXPECTANT** or **ACTIVE** depending on the following factors;

- *Gestational age*: (< or > 37 weeks) i.e.; term or preterm foetus.
- *Severity of APH*: (mild, moderate, or severe), and its effect on maternal or foetal well being.
- *Onset of labour pains;* whether the patient is in labour or not

A. EXPECTANT MANAGEMENT:

Aim: continuation of pregnancy till foetal maturity is achieved or labour pains start

Indications: Mild bleeding in pregnancies < 37 weeks.

Lines of Expectant Management in PL PRV:

- Confirm placental localization and exclude placental abruption by US.
- Rest, reassurance, correction of anaemia, and check bleeding and coagulation profiles.

B. ACTIVE MANAGEMENT:

Aim: immediate **termination of pregnancy** to avoid maternal and or foetal complications.

Indications:

- All Pregnancies **> 37 weeks** (foetal lung maturity achieved) even if bleeding is minimal
- **Severe** or **recurrent** attacks of **bleeding** that affects maternal or foetal condition
- Onset of **labour pains** regardless gestational age
- **Poor foetal** condition; as foetal distress, or major foetal anomaly.

1. *Trial Vaginal Delivery;*

Vaginal delivery could be allowed in cases in which **labour pains** start **spontaneously** in presence of **mild APH** or during expectant management with the following prerequisites;

- Placental localization; *Low lying* or *marginal anterior* PL PRV (minor PL PRV)
- Foetal presentation is *cephalic* in absence of cephalo-pelvic disproportion (CPD)
- Foetal condition; *Normal foetal well being* as assessed by US biophysical profile, umbilical artery Doppler studies, and non stress test (NST).

1. *Induction of Labour (IOL):*

IOL may be attempted in pregnancies > 37 weeks that fulfill the criteria for trial vaginal delivery (see before) in absence of spontaneous labour pains, through either;

1. **I.V. Oxytocin:** to induce or to augment labour pains (unless contraindicated), or
2. **Vaginal PGL E1** (Mesoprostol); 25-50 ug repeated doses until labour pains achieved.
3. **Artificial ROM:** in cases of PL PRV artificial ROM during IOL will decrease the risk of severe bleeding due to the following benefits;
   - Stops the shearing effect and separation of the placenta from lower segment
   - Allows more rapid descent of the head and compression of the bleeding sinuses.
   - Stimulates uterine contractions (prostaglandin release).

3. *Caesarean Section C.S.:*

Indications of C.S. in PL PRV

- Severe APH that may affect maternal or foetal condition.
- Special types of PL PRV; Total or partial PL PRV, and marginal posterior Pl PRV.
- Foetal malpresentations; as face, breech, and shoulder presentation
- Continuous vaginal bleeding during trial for vaginal delivery.
- **Other relative indications** of C.S. as: elderly primigravida, previous C.S., cephalo-pelvic disproportion (CPD), pregnancy complications as PE, GDM, foetal macrosomia, IUGR, etc.

Precautions during performing C.S. for PL PRV:

a. Anti-shock measures available including blood transfusion if needed.

b. Use of Oxytocin, Ergometrin, and/or PGLs to avoid atonic PPH by inducing and maintaining uterine contractility.

c. In cases of uncontrollable bleeding with failure of above measures, we may do:
   - Bilateral ligation of the uterine arteries.
   - Unilateral or bilateral ligation of the anterior division of internal iliac artery.
   - Total abdominal hysterectomy (TAH) if above measures fail (or in grand multipara).

COMPLICATIONS AND EFFECTS OF PL PRV on pregnancy, labour and puerperium:

1. Unavoidable haemorrhage which may be severe causing hypovolaemic shock.
2. Premature delivery (with all complications of prematurity).
3. IUGR and IUFD in cases of recurrent and severe bleeding.
4. Malpresentations and non-engagement of the presenting part leading to more C.S. deliveries.
5. Atonic postpartum haemorrhage (PPH) due to:
   a. Defective retraction in the LUS with inability to close the bleeding sinuses.
   b. Inertia of the upper uterine segment due to anaemia.
   c. Retention of parts of the placenta due to abnormal adhesion to the LUS.
   d. LUS is thin and vascular so liable to be torn during any manipulation.

PLACENTA ACCRETA

This is a rare but serious condition in which the placenta is abnormally adherent to the uterine wall. It is classified into 3 grades of increasing severity:

a. **Placenta Accreta;** the placenta in abnormally adherent to the uterine wall

b. **Placenta Increta;** the placenta is abnormally invading into the uterine wall

c. **Placenta Percreta;** the placenta is abnormally invading through, or perforating, the uterine wall

N.B.; Placenta accreta occurs mostly in association with PL PRV due to the poor decidual reaction in the LUS especially at the site of a previous CS scar.

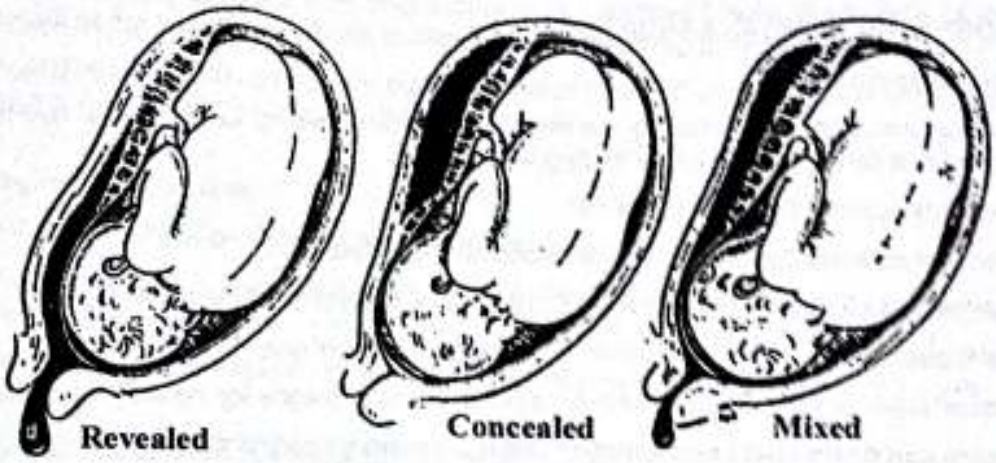
PLACENTAL ABRUPTION (ACCIDENTAL HAEMORRHAGE)

DEFINITION:

Bleeding after 20 weeks gestation due to premature separation of a **normally situated placenta.**

INCIDENCE:

1/200 pregnancies, being variable and more in elderly primigravidas.



VARIETIES OF ABRUPTIO PLACENTAL

Fig 10:5 Varieties of placental abruption

CLINICAL TYPES OF BLEEDING IN PLACENTAL ABRUPTION:

1. Revealed type: All bleeding appears vaginally from the start.
2. Concealed type: All bleeding is retained inside the uterus with formation of retro placental haematoma, with No external bleeding.
3. Mixed type: Bleeding is partially concealed and partially revealed.

AETIOLOGY and RISK FACTORS

The primary cause for placental abruption is **unknown** however several factors are listed;

1. Age, Parity, and Racial factors:
   - Risk increases with advancing maternal age (> 2.5 times > 40 years), and increasing parity
   - Risk is more in Caucasians and African women than European and white Americans
2. Hypertension (HTN):

   Gestational Hypertension, PE, and chronic hypertension are the commonest conditions. The severity of hypertension does not necessarily correlate with the incidence of **abruption**
2. Preterm ROM & PTL: Increased incidence with both preterm ROM and preterm labour (PTL)
3. Smoking: cigarette smoking increases risk for abruption by at least two-folds.

4. **Thrombophilias:** inherited or acquired thrombophilias have been linked to increased incidence of abruption. Factor V Leiden, and prothrombin gene mutation have been associated with placental infarction, abruption and preeclampsia (PE)

5. **Traumatic abruption:**
   - *External trauma to the abdomen;* as in car accidents, fall from a height, or a direct hit.
   - *Obstetric procedures:* as external cephalic version and amniocentesis (rare)
   - *Internal trauma from inside the uterus*: due to;
     a. Sudden decrease in intra-amniotic pressure after ROM in severe polyhydramnios.
     b. Traction on the placenta by a short cord during vaginal delivery especially in cases of precipitate labour.

PATHOLOGY OF PLACENTAL ABRUPTION

- **Placental pathology:**
  - Degenerative arteriolitis affecting the decidual arterioles leading to ischaemia, necrosis, and rupture of a decidual spiral artery leading to;
  - *Haemorrhage* into the decidua basalis
  - *Placental separation* with bleeding from open placental sinuses
  - *Haematoma formation* which expands causing more separation of the placenta.
  - *Retro placental haematoma,* in cases of concealed haemorrhage
  - Placental pathology of *preeclampsia* (infarcts) in cases of pregnancy induced hypertension.
- **Concealed Accidental haemorrhage;** This is more likely to occur due to;
  - Accumulation of blood behind the placenta while its margins still remain adherent to the uterus
  - Adhesion between the foetal membranes and uterus.
  - Deeply engaged presenting part (usually the foetal head) closely applied to the LUS compressing the membranes so that blood cannot make its way out
  - High rupture of membranes with bleeding inside the amniotic sac.
  - Atony of the uterus.
- **Uterine pathology:**
  - *Intra-myometrial haemorrhage* with tearing of muscle fibres, leading to necrosis
  - *Subperitoneal ecchymosis* leading to **Couvelaire uterus** which may be atonic.

CLINICAL PICTURE

I. REVEALED ACCIDENTAL HAEMORRHAGE:

- **Symptoms**
  - Vaginal bleeding: which may be **mild, moderate, or severe**, according to the extent of placental abruption. Bleeding is usually bright red in colour
  - Pain; is usually present but mild
  - Symptoms of PE or eclampsia; may be present and should be searched for
  - Other risk factor or aetiology for bleeding may be evident as; external trauma, heavy smoking, antiphospholipid syndrome, etc.

- **General Examination:**
  - The general condition is in proportion with the amount and rate of blood loss
  - Anaemia will be present if bleeding is severe and recurrent
  - Signs of PE (hypertension and albuminuria) or eclampsia (PE + convulsions) may be present

- **Abdominal examination:**
  - *The abdomen and uterus*; are usually lax,
  - *The foetal parts and movements;* are easily felt through the uterine wall
  - *The fundal level* matches with the duration of gestation according to the LMP
  - *The Presenting part;* NO increased incidence of malpresentations, however in cephalic presentations the head is usually not engaged allowing bleeding to be revealed.
  - *Foetal heart sounds;* are usually audible if bleeding is mild. In cases of marked separation the foetus may be dead or severely distressed.

- **Vaginal Examination:**
  - PV examination is performed **ONLY AFTER** exclusion of PL PRV by US.
  - PV examination is performed **ONLY WHEN** Indications for termination of pregnancy are present, in order to facilitate choice between trial vaginal delivery versus C.S.
  - Precautions during vaginal examination: the same precautions as in placenta praevia.

II. CONCEALED ACCIDENTAL HAEMORRHAGE:

- **Symptoms:**
  - Vaginal bleeding; is usually **absent.**
  - Pain; abdominal pain is **prominent,** and is usually sudden, severe, and progressive.
  - History of a cause, as recent trauma, may be **evident** , or should be searched for
  - Symptoms and signs of PE and or Eclampsia may be present.
  - Attack is usually **single** due to immediate termination of pregnancy in most cases.

- **General examination:**
  - The general condition **may not** correlate with symptoms or the complaint
  - Signs of hypovolaemic shock may be prominent without evidence of external blood loss
  - Neurogenic shock; may occur due to the sudden stretch of uterine serosa by the enlarging haematoma and intramyometrial bleeding

N.B.; In PE, the preexisting hypertension may mask the hypotensive effect of blood loss.

- **Abdominal examination:**
  - *The abdomen;* usually shows localized or diffuse tenderness, guarding, and or rigidity.
  - *The fundal level;* is higher than the calculated gestational age due to the retro-placental haematoma. It may rise gradually in the abdomen with progressive intrauterine bleeding.
  - *Foetal parts and movements;* are not easily felt as the uterus is usually tense and tender.
  - *Foetal heart sounds;* may be normal in cases of **mild** concealed haemorrhage. However with **severe** haemorrhage FHR may be irregular (foetal distress) or inaudible (IUFD).

- Pelvic examination:
  - PV is performed **Only when** termination of pregnancy is indicated and with the same precautions as for PL PRV and revealed placental abruption.
  - PV is performed to facilitate decision of attempting vaginal delivery versus performing C.S. (see revealed placental abruption).

III. MIXED OR COMBINED ACCIDENTAL HAEMORRHAGE

The clinical picture is mostly similar to concealed haemorrhage, in spite of associated vaginal bleeding which is usually moderate in amount and dark red in colour

INVESTIGATIONS IN A CASE OF ACCIDENTAL HAEMORAHGE

1. **Ultrasound:** for placental localization to exclude PL PRV. Diagnosis of a retro-placental haematoma is not easy except if haematoma is large.
2. **Coagulation profile** (bleeding and coagulation times, prothrombin time and concentration, Fibrinogen level, and fibrin degradation products FDP).
3. Complete blood picture (CBC); to check for presence and severity of anaemia.
4. Urine analysis: To detect proteinuria suggestive of preeclampsia (PE).
5. Kidney and liver functions: to anticipate renal dysfunction and diagnose HELLP syndrome.
6. Retinal examination: in cases associated with severe PE and eclampsia.
7. Weiner clot retraction test: if blood failed to form clot or if clotted blood breaks easily that indicates hypofibrinogenaemia.

DIFFERENTIAL DIAGNOSIS

1. Revealed accidental haemorrhage: Other causes of APH.
2. Concealed accidental haemorrhage: acute abdomen, and shock in obstetrics.

MANAGEMENT OF PLACENTAL ABRUPTION

Management of placental abruption is either **EXPECTANT** or **ACTIVE** depending on the severity of the condition, the gestational age, and both the maternal foetal condition.

A. EXPECTANT MANAGEMENT:

Aim: continuation of pregnancy till fetal maturity is achieved or labour pains start

Indications: Mild abruption in pregnancies <37 weeks with good maternal and foetal condition

Lines of Expectant Management;

- Confirm placental localization by US to exclude PL PRV
- Rest, reassurance, correction of anaemia, and check on bleeding and coagulation profile
- Laboratory investigations to check on bleeding and coagulation profiles

B. ACTIVE MANAGEMENT:

Aim: Immediate **termination of pregnancy** to avoid maternal and foetal complications

Indications:

- All pregnancies **> 37 weeks** even if abruption is minimal
- **Severe** or **recurrent** bleeding affecting maternal or foetal condition.
- Bleeding of any amount complicated by **PE** or **Eclampsia**
- **Onset of labour** pains irrespective of gestational age.
- IUFD or foetus with major anomalies incompatible with life.

1. *Trial Vaginal Delivery:*

Vaginal delivery could be allowed when labour pains start spontaneously in cases with mild abruption during expectant management, with the following prerequisites

- Absence of any indication for C.S.
- Foetal presentation; cephalic with absence of CPD, or malpresentations.
- Foetal condition: normal after assessment by (US - Doppler – Biophysical profile - NST).

2. *Induction of Labour (IOL):*

IOL may be attempted in pregnancies >37 weeks that fulfill the prerequisites for a safe trial of vaginal delivery (see before) in absence of spontaneous labour pains, through;

1. **I.V. Oxytocin** drip, or **Vaginal PGL E1** repeated doses; to stimulate uterine contractions.
2. **Artificial ROM** to decrease uterine distention and pain; descend of the head and compression of the cervix with stimulation of uterine contractions.

3. *Caesarean section (C.S.)*

Indications of C.S. in Accidental Haemorrhage:

- Patient in shock (which is the common presentation, in concealed accidental haemorrhage).
- Severe vaginal bleeding, irrespective of the state of cervical dilatation.
- Moderate bleeding and cervix is closed.
- Foetal distress, irrespective of the amount of bleeding.
- Continuous bleeding during trial for vaginal delivery.
- **Other relative indications** of C.S. as; elderly primigravida, malpresentations, CPD, pregnancy complications as PE, GDM, IUGR, etc...

Precautions during performing C.S. in placental abruption:

- Use of Oxytocin, Ergometrin, and PGLs in addition to continuous uterine massage to avoid atonic PPH after delivery of the fetus and the placenta, to augment uterine contractility (see management of atonic PPH)
- If above measures fail to control bleeding we perform:
    1. ligation of both uterine arteries, or anterior division of internal iliac arteries.
    2. Abdominal hysterectomy, if the above measures fail.

ANTI-SHOCK MEASURES IN CASES OF APH:

1. I.V. line for intravenous fluids (saline, lactated ringer, plasma and blood transfusion).
2. Insertion of urethral catheter (to evaluate urine output rate; > 60 ml/hr) .
3. Insertion of a central venous pressure catheter CVP.
4. Blood grouping and cross matching, and screening for clotting factors.
5. Electronic fetal heart rate monitor.

CARE OF THE 3RD STAGE AND PUERPERIUM:

- Measures to avoid PPH due to atony of uterus and/or hypofibrinogenaemia.
- Prophylaxis and treatment of expected anaemia due to revealed or concealed blood loss.
- Prophylactic antibiotics against sepsis.

COMPLICATIONS OF ACCIDENTAL HAEMORRHAGE:

A. Maternal complications:

1. Shock, which is either:

- Hypovolaemic shock (due to excessive blood loss).
- Neurogenic shock (due to severe pain from to uterine distension).

2. ***Hypofibrinogenaemia & DIC:*** This occurs in severe cases due to:

- Tissue thromboplastin is released from the ischaemic, necrotic tissues into circulation leading to formation of thrombin from prothrombin which in turn will transform fibrinogen into fibrin which will form local and disseminated thrombosis and hypofibrinogenaemia.
- Consumption of the clotting factors & platelets in the formation of the retro-placental clot.
- Liver pathology associated with pre-eclampsia (if present).

3. ***Acute renal failure:***

- Renal vasospasm due to hypovolaemia.
- Renal vasospasm due to uterine distension "utero-renal reflex".
- Renal pathology of hypertensive states of pregnancy (if present).
- The result is bilateral tubular necrosis which is reversible or bilateral cortical necrosis which is irreversible.

4. Postpartum haemorrhage:

- Atony due to myometrial damage (Couvelaire uterus) or due to severe anemia.
- Hypofibrinogenaemia and disseminated intravascular coagulopathy (DIC).

5. Sheehan syndrome. Severe pituitary insufficiency due to sudden acute blood loss.

B. Foetal complications:

- Perinatal mortality is 95% in concealed & 50% in the revealed type:
- Intrauterine hypoxia or 1UFD.
- Preterm labor.

Treatment of hypofibrinogenaemia:

1. Human fibrinogen 6-12 gm (expensive and carries risks for hepatitis).
2. **Fresh blood** is the best option as it fills circulation with RBCs, plasma and active coagulation factors.
3. Freshly prepared concentrated plasma or cryoprecipitate (if fresh blood not available).

Emergency First aid Management of A.P.H. at home:

1. History, general and abdominal examination only. **NO PV examination.**
2. Sedation, **I.V. Cannula** to start fluids and even blood transfusion if needed.
3. Put sterile **vulval pad**, to evaluate amount of bleeding, and transfer the patient to the hospital.

RUPTURE VASA PRAEVIA

Definition & aetiology:

This is a very rare condition in which the umbilical vessels in the membranes are passing opposite the internal cervical in case of velamentous insertion of the cord.

Rupture of these vessels will lead to bleeding of fetal origin which is very dangerous.

Diagnosis:

Vasa praevia should be suspected when fetal distress is marked with mild vaginal bleeding and good general condition of the mother. Examination of the blood will show fetal RBCs.

Treatment is by immediate caesarean section.

Key points in Antepartum haemorrhage:

- *In cases of PP the patient presents with painless, causeless, and recurrent bleeding. If bleeding is severe the patient will be in state of hypovolaemic shock.*
- *In concealed accidental haemorrhage the patient usually presents with acute abdominal pain which is sudden, severe, and progressive. Shock if present may be hypovolaemic or Neurogenic.*
- *The fetus is more affected in cases of placental abruption than in PP.*
- *In concealed accidental haemorrhage the patient's general condition may be poor, and deteriorating, in-spite of absent, or minimal, revealed vaginal bleeding. Such cases are also at high risk for development of DIC, and Couvelaire uterus.*
- ***Ultrasonography** is the gold standard in diagnosing the cause of APH.*
- *In cases of **mild APH**, with non recurrent bleeding, conservative management is allowed only until reasonable fetal maturity is achieved.*
- *In cases of **severe APH**, irrespective of the cause, immediate anti-shock measures and pregnancy termination are indicated. Caesarian section is the most appropriate choice in the majority of cases.*
- ***Anti-D immunoglobulin** is to be given to non sensitized RH negative mothers (those without previously detected antibodies) if they experience a moderate bleeding episode in the third trimester while conservative management has been decided.*

11 HYPEREMESIS GRAVIDARUM

The vomiting act and The vomiting centre

Nausea and Vomiting in pregnancy

Hyperemesis Gravidarum

- Pathogenesis
- Pathology
- Clinical picture
- Diagnosis
- DD of NVP

Management of HEG

- Advice & nutrition
- Fluid therapy & TPN
- Medications
- Termination of pregnancy

Vomiting is a reflex emptying of the stomach through the mouth. It may be preceded by a sense of nausea. If severe, it may be followed by dehydration; weight loss, electrolyte and acid base upsets. Finally organ damage may occur and the patient's life may be endangered.

The Vomiting Act

Reverse peristalsis empties the small gut contents into the stomach, and the cricopharyngeous closes the glottis and prevents aspiration of the vomitus. The breath is held in mid-inspiration, the muscles of the abdominal wall contract to increase the intra-abdominal pressure, the cardiac sphincter relaxes and the gastric contents are ejected.

The Vomiting Center

The vomiting center is located in the medulla and can be stimulated by tickling the back of the tongue, stomach distension, and vestibular stimulation (motion sickness). *Acetylcholine* is the neurotransmitter in the vomiting center.

Chemoreceptor trigger zone (CTZ) is located in the fourth ventricle and can be stimulated by emetics, uremia and hCG. *Dopamine* is the neurotransmitter in the CTZ.

NAUSEA AND VOMITING OF PREGNANCY NVP Emesis Gravidarum)

Incidence: NVP in early pregnancy affects nearly 50% of primigravidas, but less in multigravidas

Severity: NVP starts with moderate severity in early pregnancy with a peak at 7-9 weaks that ameliorates by 12 weeks gestation.

Aetiology: In most cases it is related to hCG levels, however psychological conflicts and avitaminosis (Thiamine) worsen the condition and makes it resistant to treatment

Management:

1. NVP of mild severilty; is generally considered a signs of an intact pregnancy that does not call for treatment, apart from patient reassurance
2. NVP of moderate severity: is best managed by reassurance, small frequent light meals, and limited use of antiemetic drugs allowed in pregnancy
3. Severe NVP; also known as hyperemesis gravidarum (HEG) may be a serious condition that usually needs hospitalization, IV fluids, and intense therapy (see later).

HYPEREMESIS GRAVIDARUM
(Pernicious Vomiting of Pregnancy)

- **Definition:** Severe NVP that is not confined to the morning, not responding to regular medications, and affects the patient's general condition.
- **Incidence:** It affects nearly 1% of pregnancies especially in primigravidas, and is strongly related to a psychological background. The patient's general condition worsens rapidly, and becomes very resistant to conventional treatment methods. Although serious, yet deaths from hyperemesis gravidarum are now rare.

PATHOGENESIS of HEG

1. Neurosis theory:

Psychological conflicts play a role as evidenced by rapid improvement on patient isolation.

2. Avitaminosis theory:

- Vitamin B1 (Thiamine) is a water-soluble vitamin and function as a coenzyme for decraboxylase. Its deficiency can lead to heart failure (wet beriberi) and neuritis (dry beriberi).
- Vitamin B6 (Pyridoxine) is a water-soluble vitamin and function as NH2 carrier so it is essential for deamination and transamination.
- Therapy with both of these vitamins improves many cases of HEG

3. Endocrine theory:

- The severity of HEG has a positive correlation with the levels of hCG. This theory explains the increased incidence of HEG multi-foetal pregnancies and hydatidiform mole, and the drastic improvement of hyperemesis after 12 weeks.
- HCG has a TSH action and many cases of HEG may have biochemical thyrotoxicosis. This form of gestational thyrotoxicosis rarely needs antithyroid medications.

PATHOLOGY

- ***Biochemical changes*** in HEG include; dehydration, haemoconcentration, hyponatremia, hypokalemia (renal loss) and metabolic alkalosis. Urine depleted from chloride, as detected by Fantus test, indicates poor prognosis.
- ***Circulatory collapse*** may induce prerenal failure.
- Starvation, ketoacidosis and weight loss; Starvation depletes liver glycogen and the liver in fatal cases shows central lobular necrosis, with abnormal liver function tests in these cases.
- ***Wernicke's Encephalopathy*** is due degenerative changes in the brain, it is related thiamine deficiency and is manifested delirium, ataxia, and nystagmus. MRI in Wernicke's encephalopathy may reveal petechial hemorrhages in the Wernicke area of the brain. Other manifestations of thiamine deficiency are optic neuritis and six-nerve palsy.

CLINICAL PICTURE

- HEG begins as ordinary morning sickness, but instead of being confined to the early morning vomiting is repeated and occurs apart from food intake and may be bilious or blood stained.
- After a time the patient becomes lethargic, emaciated, dehydrated with manifestations of circulatory collapse. End stages show jaundice, hepato-renal failure, neuritis, delirium and coma.

DIAGNOSIS

- HEG is a diagnosis of exclusion, and no single confirmatory test available.
- Vomiting starts in early pregnancy, with a rapidly progressive course, not limited to a period of the day (as morning sickness).
- Severe NVP becomes resistant to antiemetic therapy, and affects the general condition.
- Vomiting beginning after 12 weeks of pregnancy is hardly attributable to HEG and other causes should be ruled out.

INVESTIGATIONS:

Assessment of the general condition of the case including; fluid electrolyte and acid base status, liver and kidney function tests, fundus examination and circulatory assessment.

DIFFERENTIAL DIAGNOSIS:

- **Multifetal pregnnacy, vesicular mole;** should be excluded in every case.
- **Pregnancy related conditions;**, gestational thyrotoxicosis, preeclampsia, HELLP syndrome, acute fatty liver, and pregnancy cholestatsis.
- **GIT disorders:** peptic ulcers, appendicitis, cholecystitis, pyelitis, hepatitis, drug poisoning.
- **Rare associated conditions:** Brain tumours, mismatched blood transfusion....etc.

MANAGEMENT OF HEG

- **General advice:** Early and aggressive treatment, hospitalization, and psychiatric support.
- **Fluid therapy:**
  - The best fluid is normal saline (glucose worsen Wernicke's encephalopathy)
  - Initial loading IL/hr until urine flow exceeds 30 ml/hr
  - Fluid balance chart
- **Feeding:** In severe cases, NO ORAL FEEDING, at the start of treatment, until vomiting becomes c trolled or ameliorated. Instead I.V. fluids and total parenteral nutrition are started.
- **Medications:**
  - Antiemetics: I.V., I.M., suppositories, or oral forms in mild cases. (see table)
  - Thiamine 100 mg intravenously diluted in saline.
  - Prednisolone 40 mg/day

Antiemetic drugs commonly used in NVP and HEG

Receptor blocker	Drug and class group	Trade	Action	Comment
Dopamine	Promethazine C Metoclopramide B	Phenergan Primperan	CTZ	Extrapyramidal effect Hyperprolactinemia
Acetylecholine	Scopolamine B	Hysoine	VC	Dry mouth
Histamine	Meclozine B	Navidoxine	Both	Sedation
Serotonin	Ondansetron C	Zofran	Both	Depression

Total parenteral nutrition: (TPN)

- At first calculate the daily caloric need 30 kcal/Kg/d, and the daily fluid needs 30 ml/Kg/d.
- I.V. hyperalimentation should be via catheter inserted into the subclavian vein.
- Fluid to be given should contain lipid emulsion, dextrose, amino acids electrolytes and vitamins. The prepared solution should be kept in refrigerator.
- The main troubles arise from long placement of intravenous catheter (Line associated sepsis), circulatory troubles and metabolic troubles.

Indications for pregnancy termination in HEG:

Pregnancy termination is the last resort in management of severe cases resistant to treatment, reaching a life threatening situation. It us generally rarely resorted to ifproper management of the case is early and properly started. Indications include;

- Worsening of the vital signs (BP, pulse, respiratory rate, and temp)
- severe dehydration with oliguria and circulatory collapse, without good response to treatment.
- Severely affected liver and or kidney functions pointing to impending liver and or renal failure.
- Termination is via medically induced abortion using PGL E1 (Mesoprostol).
- SE may be needed in some cases

12 ANAEMIA DURING PREGNANCY

Definition
Prevalence
Classification
Effects on pregnancy
- Maternal
- Foetal

Diagnosis
Management
- During pregnancy
- During labour
- During Puerperium

DEFINITION AND PREVALENCE

- Anaemia is the *commonest medical disorder in pregnancy.*
- ***WHO definition:*** anaemia in pregnancy is a Hb concentration of < 11g/ dl (7.45 m. mol/l) and a haematocrit of < 0.33, at any trimester in pregnancy.
- ***Centers for disease control (CDC):*** proposed a cut- off point 10.5 g /dl during 2nd trimester.
- About **50%** of pregnant women worldwide will suffer some degree of anaemia in pregnancy.
- Mild anaemia; Hb levels of 9-11 g/dl. Pregnancy will aggravate any already existing anaemia.

CALSSIFICATION AND TYPES OF ANAEMIA

1. **Physiological Anaemia of pregnancy:** During pregnancy the plasma volume increases more than the increase in the R.B.C. volume resulting in haemodilution anaemia (CBC is normal).

2. Nutritional anaemia

   - ***Iron Deficiency Anaemia:***
     Iron deficiency is the commonest nutritional deficiency anaemia in pregnancy followed by foliate deficiency anaemia. It may either be due to increased iron loss or decreased iron absorption.
   - ***Megaloblastic Anaemia:***
     In megaloblastic anaemia, DNA replication is affected. There is derangement of red cell maturation with production of abnormal precursors known as megaloblasts which can be due to deficiency of *foliate or vitamin B_{12}*

3. Haemorrhagic Anaemia: due to severe or repeat blood loss as in APH, or GIT bleeding.

4. Haemolytic Anaemia:

   - ***Microangiopathic haemolytic anaemia***: occurs in some patients with severe PE, eclampsia, ITTP.
   - ***Acquired immune haemolytic anaemia:*** antibodies of the IgG type against red cell antigens are present in collagen vascular diseases.
   - ***Haemolytic anaemia associated with haemoglobinopathies:*** due to abnormal Hb synthesis; as in cases of sickle cell anaemia, and Beta thalassemia.

5. Aplastic Anaemia: Extremely rare and the mortality rate is about 30%.

CLASSIFICATION FOR SEVERITY OF ANAEMIA

Category	Anaemia severity	Hb level (g/dl)
1	Mild	10.0-10.9
2	Moderate	7.0-10.0
3	Severe	<7.0
4	Decompensated	<4.0

EFFECTS OF ANAEMIA ON PREGNANCY

A) Maternal Effects

- Mild Anaemia: No effect on pregnancy and labour. Mother will have low iron stores.
- Moderate Anaemia: Increased weakness, lack of energy, fatigue and poor work performance.
- Severe Anaemia: Associated with poor outcome as; increased PTL, PE and sepsis

B) Fetal Effects:

- Decreased iron stores due to depletion of maternal stores.
- High risk for an adverse perinatal outcome (PTL, SGA, and increased perinatal mortality).

Iron Requirements in Pregnancy

- Basal iron requirements: 280 mg
- Expansion of red cell mass: 570 mg
- For transfer to the fetus: 200-350 mg
- For the placenta: 50-150 mg
- For blood loss at delivery, 100-250 mg.

After deducting iron conserved by amenorrhoea (240-480 mg), an additional 500-600 mg of iron is required in pregnancy or 4-6 mg/day of absorbed iron.

Because absorption is less than 10% at least 40-60 mg of iron should be available in the diet, so iron supplementation is a necessity in all pregnant women.

DIAGNOSIS OF ANAEMIA I N PREGNANCY

A) Symptoms:

- There may be no symptoms in mild and moderate cases in most pregnant women
- Severe anaemia: patients may complain of weakness, exhaustion, loss of appetite, palpitation and dyspnoea. Rarely congestive heart failure CHF may occur in severe cases.

B) Signs:

- There may be no signs in mild and moderate cases.
- Severe anaemia may be associated with one or more of; pallor, glossitis and stomatitis. Oedema and systolic murmur can be found in some cases .

C) Investigations:

- Complete blood picture (CBC) including Hb% and haematocrit value (Ht).
- Serum ferritin; better picture of stored iron levels < 30 µg/L are diagnostic of iron deficiency.
- Investigations for detection of the cause; as serum iron, peripheral blood smear, and Hb electrophoresis for diagnosis of inherited anaemia and leukemia

MANAGEMENT OF ANAEMIA

1. During pregnancy

A) Prevention: Proper antenatal care. Iron supplementation using oral iron preparations. Proper diet rich in iron and vitamin C.

B) Treatment:

- Oral iron therapy: In mid trimester or early 3rd trimester. Hemoglobin rises from 0.3 to 1.0 g per week, and this is reflected in a significant elevation in Hb/Ht values 2 to 3 weeks after initiation of treatment.
- Parentral iron therapy: For women with severe anaemia in late 3rd trimester or those with poor compliance for oral therapy. Iron dextran is used.
- *Blood transfusion* (Packed cells are preferred): Very rarely required in patients with severe anaemia beyond 36 weeks, associated infection, to compensate blood loss due to antepartum haemorrhage and in patients not responding to iron therapy.

2. During labour

- *First stage:* Oxygen should be ready if dyspnoea develops. Antibiotic prophylaxis.
- *Second stage:* Shortened if necessary to avoid maternal exhaustion.
- *Third stage:* Active management should be done except in very severe anaemia for fear of cardiac failure (any post partum haemorrhage must be treated as these patients tolerate bleeding very poorly).

3. During Puerperium:

- Adequate rest, iron and foliate therapy for at least 3 months.
- Any infection should be promptly treated.

13 URINARY TRACT INFECTIONS IN PREGNANCY

Bacteruria of Pregnancy
- *Definition*
- *Incidence and risks*
- *Investigations*
- *Treatment*

Pyelonephritis During Pregnancy
- *Incidence*
- *Aetiology and predisposing factors*
- *Clinical Picture*
- *Diagnosis*
- *Complications*
- *Treatment*

BACTERURIA OF PREGNANCY

DEFINITION

- *Significant asymptomatic bacteruria:* is defined as the presence of 100,000 or more bacteria per ml of urine in two freshly voided midstream specimens of urine.
- *The mere presence of bacteria* in the urine of the pregnant women (without pus cells) does not signify infection of the urinary tract.

INCIDENCE AND RISKS

- It occurs in about 2-10% of all pregnant women (average 6%).
- ***Pyelonephritis*** will develop in ***25-30%*** of cases with significant bacteruria if untreated but in less than 1% of the patients without bacteruria. Therefore many cases of pyelonephritis in pregnancy can be prevented by finding and treating asymptomatic bacteruria.

INVESTIGATION

- Urine examination for presence of pus cells
- Culture and sensitivity for identifying pathogenic organisms.

TREATMENT:

- **Ampicillin** antibiotic: The infecting organism is most commonly E coli. About 75% of cases are cured by a single 10 days course of Ampicillin (500 mg 8-hourly).
- **Recurrence:** In 1/3 of the cases, bacteruria will recur and treatment should be repeated.
- **Resistance and/or recurrence:** antibiotic should be given according to **culture** and **sensitivity**.
- After delivery resistant and recurrent cases should be offered a full urologic evaluation including an intravenous pyelography (IVP)

PYELONEPHRITIS DURING PREGNANCY

INCIDENCE: About 1-2% of pregnancies may be complicated by pyelonephritis.

AETIOLOGY

A) Predisposing Factors

- *Asymptomatic bacteruria* during pregnancy.
- *Urinary stasis* due to dilatation of the ureter caused by:
  - Atony of the ureter associated with outer compression by the enlarging gravid uterus.
  - Hypertrophy of the wall of the lower end of the ureter.

N.B.: Ppyelonephritis is more common on the right kidneys due to more compression on the right ureter, due to usual slight right inclination (dextrorotation) of the gravid uterus in most cases.

B) Infecting organisms: E. coli is the commonest organism. Others include; Streptococcus foecalis, Staphylococci, Bacillus proteus and Klebsiella Enterobacter.

ROUTES OF INFECTION

- Blood borne (most common).
- Lymphatic spread: ascending infection along the periureteric lymphatics or along the lumen, or Spread by lymphatics from the colon.

CLINICAL PICTURE (usually occurs after 16 weeks):

- **Symptoms:**

1. Acute onset of pain in the loin, usually associated with high fever, and rigors
2. Excessive nausea and vomiting are usually present.
3. Pain is usually preceded by a history of recurrent attacks of burning micturition

- **Signs:**

Tenderness on one or both renal angle regions.

INVESTIGATIONS: (complete urine analysis):

- The urine is acidic, diminished in amount severe cases and contains albumin.
- The urine contains pus cells and micro-organisms. Culture of the urine is always positive.
- CBC may show mild Leucocytosis with shift to the left in ratio of staff to segmented WBcs

Differential diagnosis

1. Appendicitis: tender right iliac fossa, fever is lower, urine is free, Leucocytosis is higher.
2. Acute abdomen, twisted ovarian cyst.
3. Other causes of fever and vomiting during pregnancy.

COMPLICATIONS

1. Chronic pyelonephritis; in recurrent and inadequately treated cases especially ureteric stones.
2. Severe cases inadequately treated or neglected; abortion, PTL, or even IUFD may occur.
3. With less severe infection and even in cases of asymptomatic bacteruria, if proper treatment is not given, foetal growth restriction (IUGR) or preterm labour (PTL) may occur.

TREATMENT

A) General measures:

- Rest, light diet, increased fluid intake
- Alkalanization of the urine, if it is acidic
- Intravenous fluids if there is excessive vomiting.

B) Antibiotics:

- Start with Ampicillin 500 mg 6-hourly, until results of urine culture are available
- Continue according to the results of the culture and sensitivity.

14 HYPERTENSIVE DISORDERS IN PREGNANCY

Hypertensive disorders represent one of the most serious medical problems encountered during pregnancy, complicating around 5-10% of all pregnancies. They may have a serious impact on both maternal and fetal morbidity and mortality, being the second most common cause of maternal mortality in Egypt (26:100,000),

CLASSIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY

1. **Gestational hypertension:** *It is **hypertension alone***, without proteinuria, occurring for the first time during pregnancy usually in the *second half of pregnancy*. It has been formerly known as pregnancy induced hypertension) PIH.
2. **Preeclampsia (PE):** is a pregnancy specific syndrome of ***hypertension*** *and* ***proteinuria***, with or without oedema, occurring mostly in the *second half of pregnancy*.
3. **Eclampsia:** is the occurrence of seizures (fits or convulsions) in a patient with preeclampsia, no attributable to any other underlying cause or disease
4. **Chronic hypertension:** hypertension present *before pregnancy*, or occurring *< 20 weeks gestation*, or hypertension that is *persistent > 12 weeks postpartum*.

- **Hypertension:** is a **BP: 140/90** or **greater** .measured on two occasions 6 hours apart
- **N.B.:** Normally there is a tendency towards a decrease in blood pressure during the first and second trimesters of pregnancy, with a return to pre-pregnancy levels at the beginning of the third trimester.

PREECLAMPSIA

Preeclampsia PE is a pregnancy specific syndrome of ***hypertension and proteinuria***, with or without oedema, occurring mostly in the ***second half of pregnancy***.

PE is a multi-system disorder, characterized by reduced organ perfusion secondary to vasospasm and endothelial activation.

- **Proteinuria:** Urinary protein excretion of **> 300 mg/24 hr**, or persistent 30 mg/dl (1+ dipstick). The degree of proteinuria may fluctuate widely over 24-hour period even in severe cases therefore a single random sample may fail to demonstrate significant proteinuria.
- **Oedema:** Oedema has been abandoned as a diagnostic criterion as it occurs in too many normal pregnant women, and its presence does not affect the pregnancy outcome.

INCIDENCE

- Incidence varies from 3-7 % of all pregnancies.
- More common in primigravidas, especially at the extremes of age.
- PE has a recurrence rate of no more than 20 %.

RISK FACTORE FOR PREECLAMPSIA

1. *First time exposure to chorionic villi:* (Primigravidas)
2. *Exposure to superabundance of chorionic villi:* (Multiple pregnancy – hydatidiform mole)
3. *Preexisting vascular disease:* (Diabetes mellitus, chronic hypertension, autoimmune vasculitis)
4. *Genetic predisposition to hypertension:* (PE in a previous pregnancy, chronic renal disease, obesity of a marked degree).
5. *Family history of PE or Eclampsia.*
6. *Associated abnormal placentation:* Hydrops foetalis, Polyhydramnios).

AETIOLOGY OF PREECLAMPSIA (unknown)

- **Abnormal or defective placentation** in PE results in placental ischaemia or hypo perfusion. The abnormal placenta releases an unknown substance that passes into maternal circulation leading to **vascular endothelial damage** with all its sequelae. This substance may be a cytokine, oxygen free radical, prostanoids or endothelin.

- ***Vascular endothelial damage*** (2ry to abnormal placentation), and ***generalized vasospasm*** (2ry to vasoactive substance release), are responsible for the multisystem hypoperfusion state, with its ischaemic effect on different organs, especially the small capillaries and arterioles. Several organs and systems are affected especially the placenta, kidneys, liver, and the CNS. Changes in these systems are non specific and are usually evident only in late or severe cases.

Role of Endothelium in normal pregnancy:

1. Control of vascular tone through a balance between vasoactive factors:
   - Vasodilators (VD): e.g. Nitric oxide (NO), and prostacyclin.
   - Vasoconstrictors (VC): e.g. Thromboxane, and endothelin.
2. Control of vascular permeability.
3. Control of haemostasis.

In PE endothelial damage leads to:

1. Increased vascular tone → vasoconstriction (VC > VD).
2. Increased capillary permeability → oedema and proteinuria.
3. Platelet thrombosis ---- (DIC in severe cases).

PATHOPHYSIOLOGIC EFFECTS OF PREECLAMPSIA: *(It is a multisystem disorder)*

1. **Cardiovascular system:**
   - Increased vascular responsiveness to vasoconstrictors VC (reduced in normal pregnancy).
   - Generalized VC leading to increased peripheral vascular resistance (PVR) and hypertension.
   - Decreased plasma volume due to vasoconstriction, increased capillary permeability and fall in plasma protein.
2. **Renal system:**
   - Decreased renal blood flow, and glomerular filtration rate (GFR) leading to oliguria.
   - Glomerular and tubular dysfunction lead to:
     - Proteinuria (increased renal tubular permeability)
     - Hyperuricemia (decreased clearance by renal tubules).

	Normal pregnancy	Pre-eclampsia
• Vascular sensitivity to vasopressor	Reduced sensitivity	Increased responsiveness
• Peripheral vascular resistance (PVR)	Peripheral VD results in decrease in PVR	Generalized VC results in Increased PVR resulting in hypertension
• Palsma volume	Hypervolaemia	Decreased (Haemoconcentration)
• Vascular endothelium	Normal and healthy	Endothelial damage
• Renal blood flow and GFR	Increases	Decreased
• Trophoblastic invasion of spiral arterioles	Present	Failure to occur

PATHOLOGIC ANATOMY

I. The Placenta:

A) ***Early:*** *(Failure of trophoblastic invasion of spiral arteries).*

- During normal pregnancy, invasion of the decidual then the Intramyometrial segment of spiral arteries occurs with its replacement by trophoblastic tissue. Trophoblastic invasion will thus convert the narrow muscular vessels into wide flaccid vessels not responsive to vasoconstrictive compounds, with decreased vascular resistance and increased placental blood flow.
- In preeclampsia: the trophoblastic invasion is patchy and the spiral arteries retain their muscular walls. This prevents the increase in placental blood flow leading to placental ischaemia.

B) ***Late:*** Acute atherosis of spiral arteries due to accumulation of fat-laden macrophages and perinuclear halo leading to narrowing of the lumen and placental ischaemia.

C) ***Placental infarcts:*** Placental infarcts are more common and more extensive in PE, leading to placental insufficiency, IUGR, and /or IUFD.

II. **The Kidneys:** *Glomerular endotheliosis* is a characteristic lesion where the glomeruli are enlarged, swollen, bloodless with hypertrophy of intracapilllary cells, leading to decreased GFR.

III. **The Liver:** may show peri-portal or sub-capsular hemorrhage and necrosis..

IV. **The CNS:** may show vasospasm, cerebral oedema, petechial hemorrhage and thrombi.

CLINICAL PICTURE OF PREECLAMPSIA

A) SYMPTOMS

1. **Asymptomatic cases:** PE is asymptomatic in the early and mild cases.
2. **Symptomatic cases:** Symptoms are non-specific. They usually occur late, and more commonly in severe or complicated cases:
   - Persistent headache.
   - Epigastric and right upper abdominal pain.
   - Persistent vomiting.
   - Visual disturbances: blurring of vision, scotoma, diplopia, flashes of light, blindness.
   - Oedema (lower limb, abdominal, or generalized oedema).

B) SIGNS IN PE

PE is a disease of signs rather than symptoms, mainly a hypertension and proteinuria, with or without oedema, occurring in the second half of pregnancy. Regular antenatal care will result in early detection of PE before symptoms are evident

1. **Hypertension:** is considered the *earliest feature* in PE. BP ***140/90 or more*** on two or more measurements 6 hours apart is diagnostic. It is commonly labile from time to time, usually resolving within 6 weeks after delivery.

   N.B.: *A rise of > 30 mm Hg in the systolic or > 15 mm Hg in the diastolic BP pressure is no longer a criterion for diagnosis, but only warrants close observation.*

2. **Proteinuria:** although a late sign, it is an essential criterion in the diagnosis of preeclampsia.
   - A urinary protein excretion of ***> 300 mg / 24 hrs***.
   - Using reagent urine strips: **> +1** in at least 2 random urine samples without collection of 24 hours urine, as proteinuria is usually variable over the 24 hours.
3. **Oedema:** Although a common feature in PE cases, especially severe ones, yet is not included as a criterion in the definition of PE, as it is also a common finding in normal pregnancies.
   - *Early: occult oedema:* abnormal rate of weight gain often precede clinical oedema.
   - *Late: clinical oedema:* more significant is non-dependent oedema of hands and feet. Its absence does not exclude diagnosis of PE.

C) SPECIAL INVESTIGATIONS

I. Evaluation of Severity of PE

1. ***Complete urine analysis for:***
   - Proteinuria:
     - By Dipstick in a random sample (it is a rapid, but an inaccurate test)
     - 24 hours urine collection for total protein (> 0.3 gm = mild, 5 gm = severe).
   - Other tests: specific gravity, epithelial casts, RBCs, and pus cells.
2. ***Serum uric acid*** (N = 3-5.5 mg/dl): Hyperuricemia precedes proteinuria but is non-specific.
3. ***Kidney functions tests:*** serum creatinine, and blood urea (normal except in late severe cases)
4. ***Liver functions tests:*** AST, ALT (N = 8-20 m.IU/ml) for detection of HELLP syndrome.
5. ***Complete blood picture (CBC):***
   - Haemoglobin level: may be low in association with anaemia.
   - Haematocrit: may be elevated due to haemoconcentration.
   - Platelet count: may be low in cases of HELLP syndrome (N: 150.000 - 250.000/mm^3).

6. *Coagulation profile:*
   - Prothrombin time: (N = 11-12 second).
   - Partial thromboplastin time (PTT): (N = 24 -36 second).
   - Serum fibrinogen (300 - 600 mg/dl) and Fibrinogen Degradation Products (FDP).
7. *Fundus oculi examination:* for retinal changes as vasospasm, haemorrhage and exudates.

II. Evaluation of Foetal well being:

- Daily foetal movement count (DFMC): see antepartum assessment of fetal well being.
- Non-stress test (NST): when kick count is non reassuring (see assessment of fetal well being).
- Ultrasonography: for detection of, IUGR, oligo-hydramnios, performing a Biophysical Profile Score (BPPS) test, and Doppler US of foetal umbilical artery and cerebral artery blood flow.

CRITERIA OF SEVERITY OF PE

PE is classified into **mild** or **severe** according to the following criteria :

A) Mild PE: BP < 160/110, mild proteinuria 1+, with mild or no associated symptoms.

B) Severe PE: PE is considered severe if the following criteria are present;

- *Symptoms:* Persistent headache, epigastric pain, visual disorders, or Oliguria (< 30 ml /hr.)
- *Signs:* BP 160/110 or more
- *Investigations:*
  - Persistent proteinuria (+2 or more, or 5 gm or more / 24 hour urine).
  - Elevated Liver enzymes (ALT, AST, Alkaline phosphatase, and bilirubin).
  - Thrombocytopenia (low platelets, especially <100.00/mm$_3$).
- *Presence of complications:*
  - Maternal complications: HELLP, DIC, Heart failure, or pulmonary oedema.
  - Fetal complications as IUGR.

COMPLICATIONS OF PREECLAMPSIA

Most complications of PE occur in the severe cases with prolonged hypertension.

A. Maternal Complications of PE:

1. *Eclampsia:* in 1-2% of cases of PE.
2. *Acute renal failure:* either due to acute tubular necrosis or bilateral renal cortical necrosis.
3. *Placental abruption:* in severe hypertension with retro-placental clot formation...etc
4. *HELLP syndrome:* Haemolysis, Elevated Liver enzymes and Low Platelet count. It complicates 2-4% of cases of PE and is associated with high fetal and maternal mortality.
5. *Cardiac failure* and *acute pulmonary oedema.*
6. *Intracranial hemorrhage:* due to rupture of cerebral vessels.
7. *Hepatic rupture:* due to sub capsular haemorrhage, and resultant internal bleeding and shock.
8. *Disseminated Intravascular Coagulopathy (DIC).*
9. *Retinal detachment and cortical blindness.*

- Maternal Mortality: Hypertensive disorders are the second major cause of maternal mortality.
- Remote complication: Residual hypertension, or recurrence of PE.

B. Fetal Complications of Preeclampsia:

1. Intrauterine growth restriction (IUGR), due to placental insufficiency.
2. Intrauterine fetal death (IUFD), due to severe IUGR or accidental haemorrhage.
3. Prematurity (iatrogenic), due to spontaneous or induced preterm labour.

DIFFERENTIAL DIAGNOSIS OF PREECLAMPSIA

I. Other hypertensive states with pregnancy:

- Gestational hypertension (hypertension without albuminuria).
- Chronic hypertension (hypertension antedating pregnancy, or present < 20 weeks, or persistent > 6 wks after delivery).

II. Other causes of proteinuria:

- Contamination by vaginal discharge, (a catheter specimen is therefore advisable).
- Urinary tract infection, (a complete urine analysis is diagnostic).
- Chronic kidney disease, e.g. chronic nephritis (diagnosed by kidney function tests).

III. Other causes of oedema:

- General causes: e.g. congestive heart failure.
- Varicose veins of lower limbs.
- Pregnancy with oversized abdomen (e.g.: multifetal pregnancies or polyhydramnios).

N.B.: *The proper diagnosis in a case of PE should include the severity of the disease, DD from other causes of hypertension, diagnosis of underlying risk factors if any, and diagnosis of maternal or fetal complications if present.*

PREDICTION OF PE

- No reliable tests for prediction of PE.
- Uterine artery Doppler studies at 18-24 weeks, may reveal a high resistance index (RI), with diastolic notching, that may identify up to 80% of women who will subsequently develop PE.

PREVENTION of PE

The prevention of PE will continue to be extremely difficult as long as the exact aetiology will remain unknown. However, trials have been directed towards the use of:

A) Low Dose Aspirin LDA: (infantile aspirin 75-100 mg daily from 2nd trimester).

- Indication: patients at high risk of development of PE especially those with history of severe PE before 30 weeks in a previous pregnancy, or those with severe IUGR.
- Low dose aspirin acts through prevention of thrombosis and promotion of vasodilatation:
  - It inhibits of platelet aggregation.
  - Inhibition of the release of thromboxane (TX A2), without impairing synthesis of vasodilating prostacyclin produced by the endothelium.

B) Antioxidants: Vitamin C and E.

- They act by inhibition of endothelial cell activation.
- They may be tried in high risk cases in which uterine artery Doppler shows high RI.

TREATMENT OF PREECLAMPSIA

The **goal** of treatment in PE is the **prevention** of complications particularly Eclampsia.

The **only** definitive **treatment** of PE is **termination** of pregnancy (delivery).

The **timing** of termination depends both on **gestational age** and the **severity of PE.**

A. MILD PREECLAMPSIA:

- **Full term** (37 weeks or more):**delivery** by induction of labour or CS.
- **Preterm** (< 37 weeks) : **expectant** management until fetal lung maturity reassured.
  - *Rest:* Both mental and physical rest, (bed rest is preferable in the left lateral position).
  - *Diet:* Ample protein diet, better with salt restriction.
  - *Antihypertensive drugs:* NO documented benefits in mild PE. However the most commonly used drug is alpha methyl dopa (Aldomet), in a dose of 250 mg t.d.s.
  - *Close maternal follow up:*
    - Warning symptoms: headache, visual disturbance and Epigastric pain.
    - Warning signs: BP measurements, and testing for joint hyperreflexia.
    - Laboratory tests: urinary protein assessment, serum uric acid and liver functions, daily maternal weight and urinary output.
  - *Fetal assessment:* DFMC, NST, BPPS, and Doppler US for umbilical and cerebral vessels.
  - *Outpatient management:* cases of mild PE are managed on outpatient basis with twice weekly follow up visits until foetal lung maturity is achieved or spontaneous labour start.
  - *Hospitalization:* will be mandatory if progressive deterioration occurs or a complication develops that necessitates immediate delivery
- **Mode of delivery:** *vaginal delivery*, whether spontaneous or induced, may be allowed under strict fetal surveillance with continuous electronic FHR monitoring. Otherwise *CS is preferable.*

B. SEVERE PREECLAMPSIA: *Immediate delivery after urgent adequate control by:*

- Hospitalization.
- Antihypertensive drugs: MUST be given by parenteral route, to reduce the maternal risks of cerebrovascular accident, which is an important cause of maternal mortality.
  - ***Hydralazine:*** 5 mg IV then 5 mg every 20 minutes. Direct vasodilator. Side effects: tachycardia, flushing, headache.
  - ***Labetalol:*** combined alpha and beta adrenergic blocker. It lowers the BP by decreasing the heart rate and contractility. Dose: 10- 20 mg IV repeated every 10 minutes. Advantages: less headache, less flushing, less tachycardia than Hydralazine.
  - ***Nifedipine:*** calcium channel blocker. Given 10 mg by mouth, repeated in 4-8 hrs. Better not to be used with magnesium sulfate.
- Prophylactic-anticonvulsants: Magnesium sulfate might be used to prevent the development of convulsions in severe PE before termination of pregnancy.

N.B.: *Diuretics:* may worsen haemoconcentration and predispose to thrombosis. They are thus contraindicated except in cases of pulmonary edema or heart failure.

N.B.: *Corticosteroids:* may be given in cases of PTL to decrease hazards of neonatal RDS, or in the treatment of cases complicated by HELLP syndrome...

- **Mode of delivery:**

The mode of delivery will depend largely on both the urgency for termination and the ripeness of the cervix. If conditions are favorable for a safe, well monitored vaginal delivery, then induction of labour may be started, otherwise CS is considered a relatively safe option.

A) Induction of labour:

- Method: IV oxytocin infusion, or vaginal prostaglandins (with or without amniotomy).
- Prerequisites: soft, ripe, effaced cervix, and adequate pelvis, with good fetal condition.
- Monitoring: continuous electronic FHR monitoring.
- Maternal condition: adequate control of Blood pressure and proper pain relief.
- Avoid prolonged and difficult labour, and 2nd stage should be adequately shortened.
- Avoid use of postpartum ergometrin which may increase BP and increase venous return.
- Close observation after delivery to avoid postpartum eclampsia, and PP haemorrhage..

B) Caesarean Section:

- Extremely premature or low birth weight fetus.
- Non reassuring antepartum or intrapartum fetal surveillance tests.
- Cervix is unripe and conditions are unfavorable for vaginal delivery.
- Other indications for CS fulfilled as placenta praevia, oligohydramnios, severe IUGR, previous CS, contracted pelvis ...etc.

Key points in Preeclampsia

- *PE and Eclampsia are the second most common cause of maternal mortality in Egypt.*
- *PE is a disease of signs rather than symptoms, hence the importance of antenatal care in early detection to avoid complications.*
- *Diagnosis is based on presence of hypertension and albuminuria developing at the second half of pregnancy. Oedema is not a prerequisite for diagnosis.*
- *Symptoms in PE appear late and only in the severe cases.*
- *Severe PE lead to multisystem dysfunction as a result of generalized vasospasm and vascular endothelial damage.*
- *PE is very difficult to prevent or to predict, although some parameters as uterine artery Doppler velocimetry studies at 18-24 weeks are promising.*
- *The only definitive treatment for PE is termination of pregnancy to avoid severe maternal and fetal complications.*
- *In mild PE conservative approach is adopted with expectant management until fetal lung maturity is reassured or labour pains start spontaneously. Whenever pregnancy reaches 37 weeks or more termination is better achieved via induction of labour or C.S.*
- *In severe PE hospitalization is necessary to achieve rapid control of BP by antihypertensive drugs, followed by immediate delivery via induction of labour or C.S. which is more frequently used to minimize foetal and maternal complications that may arise if labour is prolonged.*

ECLAMPSIA

An eclamptic convulsion

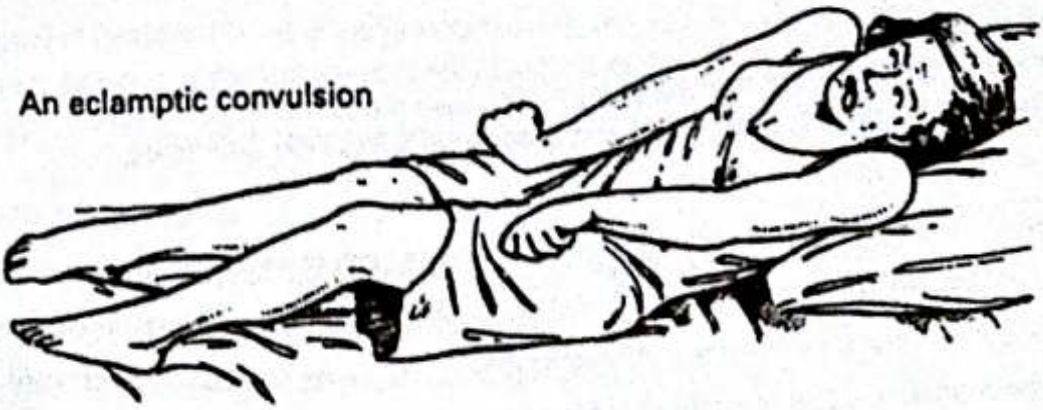


Fig 14:1 Eclamptic fit

Eclampsia is the occurrence of ***seizures*** (convulsions or fits) in a woman with ***preeclampsia*** that cannot be attributed to any other underlying cause or a preexisting disease.

The incidence of Eclampsia ranges around 1:2000 deliveries. It may complicate 1-2% of cases of preeclampsia. **Eclampsia** may occur antepartum (65%), intrapartum (20%), or postpartum (15%).

CLINICAL PICTURE: The eclamptic fit is characterized by:

1. **Premonitory stage:** ½ minute. Twitches of face and hands, rolling up of eyes.
2. **Tonic stage:** ½ minute. Generalized tonic spasm, opisthotonus, no respiration, and cyanosis.
3. **Dome stage:** ½ to 1 minute. Tongue may be bitten, breathing is stertorous.
4. **Coma stage:** patient may recover or fit may recur again.

PATHOGENESIS OF ECLAMPSIA:

The pathogenesis of convulsions is unknown; however it may be secondary to cerebral edema, vasospasm, and brain ischaemia.

DIFFERENTIAL DIAGNOSIS

Epileptic seizures is the most important condition which mimics eclamptic fits.

- Careful history from patients family and relatives will reveal previous fits antedating pregnancy.
- History of treatment with antiepileptic drugs.
- Epileptic fits are preceded by aura of different stages
- No associated hypertension or albuminuria

PREVENTION OF ECLAMPSIA

- Eclampsia is a preventable complication of PE.
- It can be prevented through early diagnosis and proper management of PE
- This could only be achieved through proper antenatal care assessment and follow up.
- Once PE develops it should be thoroughly controlled and managed, and if the disease was progressive termination of pregnancy will avoid the occurrence of eclampsia.
- Post partum eclampsia is difficult to predict or prevent.

TREATMENT OF ECLAMPSIA

Successful management of an established case is based on the following:

- Emergency First Aid measures
- Control of Eclamptic Fits
- Control of B.P.
- Immediate Termination of Pregnancy

A) EMERGENCY FIRST AID MEASURES:

1. The patient is kept in a dark room with minimal noise and light (to avoid irritant stimuli).
2. Insert a mouth gag and/or tongue depressor (to avoid tongue injury).
3. Ensure free airway, and give oxygen by mask if available (to assure adequate perfusion).
4. Apply suction from the mouth (to avoid aspiration).
5. Patient is nursed on her side with head down (to avoid aspiration).
6. An I.V. line is immediately ensured, with a good vein caliber, and CVP monitoring.
7. Urinary catheter is inserted and record volume of urine.
8. Initial I.V. loading dose of an anticonvulsant either:
   - $MgSO_4$: 4-6 gm of a 20% solution over 20 minutes by I.V. drip, or
   - Diazepam 5-10 mg slowly I.V.

B) CONTROL OF ECLAMPTIC FITS:

1. Magnesium Sulfate ($MgSO_4$): Best drug to be used.
   - *Mode of action:*
     - Inhibits muscle contractility by inhibition of neuromuscular transmission
     - CNS depressant.
   - *Dose and Administration:*
     A) Loading Dose: 4-6 gm IV infusion over 20 minutes.
     B) Maintenance dose: either
     - 2 gm / hr by continuous IV infusion of a 20% solution for 24 hours (best route), or
     - 5 gm / 4 hrs by IM into the buttock of a 50% solution for 24 hours
   - *Therapeutic serum level of $MgSO_4$:* 4-7 mEq/L.
   - *Precautions before giving the next dose:* check for:
     - Urine output (not less than 30 ml/hr): toxicity may develop in cases with oliguria.
     - Respiratory rate (not less than 16/min): toxicity may cause depression of respiratory centre.
     - Patellar reflex must be present: toxicity may cause diminish reflexes due to excessive inhibition of neuromuscular transmission.
   - *Toxicity:* Absent knee jerk reflex, marked respiratory depression, and finally cardiac arrest.
   - *Antidote:* Calcium Gluconate 10%, 10 ml, IV route, slowly over 3-5 minutes.

2. Diazepam (valium): 5-10 mg IV.

   Used as an alternative for $MgSO_4$ whenever $MgSO_4$ is unavailable.
   - It is less efficient in preventing or controlling eclamptic fits, however, it is safer to use with markedly less toxicity.
   - N.B.: If given quickly diazepam may cause sudden apnea, and cardiac arrest.

C) CONTROL THE HYPERTENSION:

Hypertension is best controlled by either

- IV Hydralazine, or
- IV Labetalol.

D) DELIVERY OF TH EFOETUS:

After first aid management, control of convulsions, and hypertension, immediate delivery should be carried out either by

- Induction of labour by PGLs and / or Oxytocin; in favorable well controlled cases, where the foetus is cephalic in presentation, head engaged, cervix soft, effaced, and pelvis adequate
- C.S.: seems a better and safer option in the majority of cases as it ensures a more rapid and less traumatizing route of delivery whenever facilities for safe anaesthesia are available

E) POSTPARTUM CARE:

Magnesium sulfate should be continued after delivery for 24 hrs to guard against postpartum fits.

Key points in Eclampsia

1. *Eclampsia is a preventable disease, based on the proper detection and management of PE.*
2. *Proper antenatal care, and prompt delivery of cases with severe PE will significantly decrease the incidence of eclampsia in the near future.*
3. *Eclamptic fits should be differentiated from epileptic seizures, before planning for pregnancy termination.*
4. *Active management of Eclampsia includes control of the fits followed by immediate termination of pregnancy.*
5. *There is no place for conservative prolongation of pregnancy in eclampsia irrespective of gestational age and lung maturity.*

CHRONIC HYPERTENSION

DEFINITION:

Chronic Hypertension is defined as

- Hypertension ***antedating*** pregnancy, or
- Hypertension detected ***< 20 weeks*** gestation, or
- Hypertension that is persistent ***> 12 weeks postpartum.***

INCIDENCE: 2-5% of pregnancies.

TYPES:

A) Essential (primary) Hypertension: in 90% of cases .

B) Secondary Hypertension: in 10% of cases due to;

- Renal disease e.g. chronic nephritis, renal artery stenosis.
- Coarcitation of the aorta.
- Endocrine causes as Pheochromocytoma and hyperthyroidism.

DIAGNOSIS:

Diagnosis of chronic hypertension in pregnancy is based on BP measurements. However in the second trimester the diagnosis may be fallacious due to normal physiologic lowering of the BP.

COMPLICATIONS:

The most important complication is the development of superimposed preeclampsia on top of chronic hypertension. It occurs in 20-25% of cases with aggravation of hypertension and proteinuria.

INVESTIGATIONS:

- ECG, and echocardiography.
- Kidney function tests, thyroid function tests, and blood sugar levels.
- Valinyl mandilic acid (VMA), for detection of pheochromocytoma.
- Fundus oculi examination, in severe cases.

TREATMENT

- Rule out and treat secondary causes of hypertension.
- Antihypertensive therapy: is indicated if diastolic BP is 100 mmHg or more i.e. severe hypertension.
- The aim is to reduce risk of intracranial hemorrhage or heart failure. The blood pressure should be maintained below 160/100.
- Obstetric management is similar to that of pregnancy induced hypertension

15 DIABETES MELLITUS WITH PREGNANCY

Definition
Incidence
Effect of pregnancy on DM
Effect of DM on pregnancy
High risk cases for DM during pregnancy
Diagnosis during pregnancy
Screening for DM
Classification of DM
Management of DM during pregnancy
Time and method of delivery
Care of the newly born IDM
Care during puerperium
Pricella white's Classification of DM during pregnancy

DEFINITION

Diabetes mellitus (DM) is a disturbance in carbohydrate metabolism characterized by hyperglycaemia, glycosuria and microangiopathy.

In pregnnacy, DM may cause many serious complications both to the mother and her foetus.

INCIDENCE: Nearly 1:350 pregnancies.

CLASSIFICATION OF DIABETES MELLITUS:

1. **Gestational diabetes mellitus (GDM):** It is a state of carbohydrate intolerance that has its onset or first recognition during pregnancy.
2. **Established Diabetes mellitus (DM):** It is diabetes mellitus that is already known to be existing before the onset of pregnancy. It may either be:
   - Insulin dependant (type 1),
   - Non insulin dependant (type 2)

EFFECT OF PREGNANCY ON DIABETES

1. **Pregnancy is diabetogenic**, especially if repeated and if the patient is predisposed. Pregnancy may unmask latent diabetes due to the production of insulin antagonists (human placental lactogen, placental insulinase, cortisol, oestrogens and progesterone).
2. **During pregnancy**, established diabetes becomes **difficult to control** as insulin requirements increase during pregnancy and decrease after delivery.
3. **After labour**, utilization of sugar by the breasts may cause **hypoglycaemia**.

EFFECTS OF DIABETES ON PREGNANCY

1. **Abortion, pre-eclampsia, hydramnios and preterm labour** are more common.
2. Tendency to **intrauterine foetal death** in the last month which may be due to ketosis, hypoglycaemia, pre-eclampsia, congenital anomalies, or placental insufficiency.
3. **Foetal Congenital Anomalies (FCA):** higher incidence of FCA (6-9%).

- The most common anomalies are VSD and open neural tube defects
- The most specific anomaly is caudal regression syndrome.

1. **Fetal macrosomia** (oversized foetus 4.5 kg or more) may occur even in the prediabetic stage. This is due to maternal and consequently foetal hyperglycaemia which stimulates hyperinsulinism in the foetus. Foetal hyperglycaemia and hyperinsulinism enhance glycogen synthesis, lipogenesis, and protein synthesis in the foetus.
2. **Monilia vulvo-vaginitis** is common in the pregnant diabetic. Infections specially urinary tract infection are more liable to occur.
3. More liability of **puerperal sepsis**, breast infection and deficient lactation.
4. Higher incidence of **neonatal mortality and morbidity**. May be due to:
   - Respiratory distress syndrome,
   - Congenital anomalies.
   - Neonatal hypoglycaemia "blood glucose less than 30 mg%" resulting from increased insulin production by the hypertrophied foetal pancreas.
   - Over sized foetus with broad shoulders leading to higher incidence of birth trauma,
   - Tendency to hypocalcaemia, hyperbilirubinaemia and hyperviscosity.

N.B.: In early pregnancy: uncontrolled diabetes results in a higher incidence of foetal congenital anomalies. In late pregnancy, the foetus is more likely to be overweight (macrosomic).

Cases at high risk for diabetes during pregnancy:

1. 35 years or more, especially with family history of diabetes.
2. Previous large macrosomic babies (4 kg or more).
3. Previous unexplained intrauterine or neonatal deaths.
4. Previous 2 or more unexplained abortions.
5. Presence or history of major congenital anomalies.
6. Gross obesity, and /or hypertension.
7. Presence or history of polyhydramnios.

DIAGNOSIS OF DIABETES DURING PREGNANCY

1. History; The patient is known to have DM.
2. Symptoms suggesting diabetes; loss of weight, thirst, polyuria, and recurrent pruritis vulvae.
3. Fasting and 2 hours postprandial hyperglycaemia.
4. Abnormal glucose tolerance test (GTT): a raised FBS and lagging GTT establish diagnosis.

- *Screening for diabetes during pregnancy*

  50 gram oral glucose test followed by a 1 hr estimation of blood glucose level:

  - 1 hr Blood glucose < 140 mg%, then screening is negative
  - 1 hr Blood glucose > 140 mg%, then a 100 gm GTT is indicated.

- *Diagnosis of DM by GTT in pregnancy:*

  - Fasting blood glucose; **90** mg/100 ml.
  - 100 gm oral glucose administration test: Normal levels at 1,2,and 3 hours should be **165** mg%, **145** mg%, and **125** mg% respectively. If any 2 or more of these values are elevated, the patient should be considered as having impaired glucose tolerance.

MANAGEMENT OF DIABETES WITH PREGNANCY

1) CONTROL OF DIABETES:

1. **Diet control:** Proper diet supplies 30-35 Kcal/kg/day, 50% of which is of carbohydrate.
2. **Antenatal care (ANC)** visits: more frequent for adequate follow up of maternal diabetic state and fetal well being.
3. Repeated **blood sugar** estimations for better control of blood sugar level.
4. **Glycosylated haemoglobin (HbA_{1c});** It is produced by glycosylation of HbA during the life of the red cells, and can be used as an indicator of the blood glucose levels in the preceding 2-3 months. Normally, it constitutes 3-4% of the total amount of haemoglobin. More than 10% indicate poor diabetic control in the preceding 2-3 months.
5. **Insulin therapy:** is indicated in all cases of established DM, and in cases of GDM, whenever diet alone is not reassuring in control of diabetes. Oral hypoglycemic drugs cannot control properly diabetes during pregnancy, and they may have teratogenic effect on the foetus, as they cross the placental barrier, while the large insulin molecule does not.
6. **Insulin dosage and preparations:**
   - Insulin requirements < 50 units/day: given in a single morning dose with NPH/regular insulin ratio 2:1.
   - Insulin requirements > 50 units/day: given in 2 doses with NPH/regular ratio 1:1. 2/3 the dose is given in the morning as before and 1/3 the dose in the evening.

2) TIME AND METHOD OF DELIVERY

Termination of pregnancy is indicated if there is:

1. Evidence of placental insufficiency (IUGR, Oligohydramnios, or abnormal umbilical Doppler)
2. Fetal lung maturity is achieved (at 37 weeks) to avoid IUFD and fetal macrosomia.
   - Induction of labour, if cervix is favourable and fetus of average weight.
   - C.S. is preferred in cases associated with fetal macrosomia or placental insufficiency.

3) CARE OF THE NEWLY BORN INFANT

1. The infant of diabetic mother (IDM) is more liable to the development of hyaline membrane disease leading to respiratory distress syndrome (RDS).
2. 5% glucose I.V. solution given afetr delivery may be needed to combat the possibility of neonatal hypoglycaemia due to overactive foetal pancreas.

4) PUERPERIUM

- Reduction of insulin dose to prevent hypoglycaemia
- Proper Antibiotic coverage to avoid puerperal sepsis.

Pricella White's Classification of DM with pregnancy

A) Chemical diabetes
B) Onset of diabetes over 20 years age AND duration less than 10 years
C) Onset of diabetes between 10 and 19 years OR duration between 10 and 19 years
D) Onset of diabetes < 10 years OR duration > 20 years OR associated with minimal vascular disease.
E) Diabetic Nephropathy
F) Diabetic Cardiomyopathy
G) Proliferative retinopathy
H) Diabetics after renal transplant.

D.D. OF JAUNDICE IN PREGNANCY

1. **Medical conditions as Infective hepatitis.**
   - Infective hepatitis during pregnancy may cause abortion, PTL, or IUFD. The virus responsible for infective hepatitis may be type A, B, or C.
   - More than half of the babies born to mothers who have had type B hepatitis during pregnancy will shows hepatitis B antigen in their blood, and a proportion of them develop hepatic lesions.

1. **Haemolytic jaundice;** due to RBC destruction, as with Thalassemia.
2. **Surgical conditions** as obstructive jaundice.
3. **Drug toxicity** e.g. chlorpromazine.
4. **Hepatic degeneration** in cases of severe hyperemesis gravidarum or pregnancy induced hypertension (PE and Eclampsia).
5. **Recurrent Cholestatic Jaundice of pregnancy** as a result of oestrogen-induced cholestasis which occurs in some women who are unduely sensitive to oestrogen (so this occurs also with oral contraceptive intake). (6) Other causes of jaundice in pregnancy as haemolytic jaundice from mismatched blood transfusion, OR jaundice from infection with haemolytic organisms, OR the very rare acute fatty liver (acute yellow atrophy) or pregnancy that occurs in late pregnancy, OR congenital hyperbilirubinaemia.

16 CARDIAC DISEASES WITH PREGNANCY

Cardiac output changes during pregnancy
Clinical effects of increases CO
Assessment of patient's cardiac condition
Management during pregnancy
Management during labour
Management in the puerperium
Induction of abortion

Cardiac disease may be associated with 0.5-1% of pregnancies. Rheumatic heart valve diseases account for most of cases in the developing countries. In developed countries, there is more preponderance of congenital heart disease rather than complications of rheumatic fever.

CARDIAC OUTPUT (CO) CHANGES DURING PREGNANCY:

During pregnancy the CO increases gradually from the first trimester reaching a peak of 40% above non-pregnant value by 20 weeks to remain constant until full term. Causes of an increased C.O. during pregnancy include:

1. Increased stroke volume (40-50% above the non-pregnant state)
2. Increase of pulse rate (+10 or 12 beat/min).

CLINICAL EFFECTS:

- **Heart rate and Pulse changes:**
  - Increase heart rate (tachycardia)
  - Obvious capillary pulsation (nail-bed).
  - Water-hammer pulse (increased systolic diastolic difference).
  - Occasional extra-systoles.
- **Apex beat variations:**
  - Elevation to the 4th intercostal space, one inch lateral to normal position.
  - Soft systolic murmur (heard by the stethoscope).
  - Split first sound (earlier closure of mitral valve).
  - Appearance of a loud third sound
- E.C.G. changes:
  - Left axis deviation, deep Q-wave in lead III.
  - Flattening of T wave and inverted ST segment in V2-V4

ASSESSMENT OF PATIENT'S CARDIAC CONDITION

The New-York Heart-Association Grading

Class I:	Organic heart disease without limitation of house hold activity
Class II:	Limitation of activity (dyspnea and chest pain) at ordinary house-hold duties. e.g. at the end of climbing one set of stairs.
Class III:	Limitation of activity at less than ordinary house-hold duties e.g. during climbing one set of stairs.
Class IV:	Dyspnea at rest

N.B.: Pregnancy usually makes the classification worse by one grade.

Other Factors affecting maternal cardiac condition:

- Associated anaemia with pregnancy
- Infections: as urinary tract infections and careous teecth (subacute cacterial endocarditis).
- Associated cardiomyopathy and tight mitral stenosis.
- Hypertensive disorders in pregnancy, and thyrotoxicosis.
- History of recent reactivation of rheumatic fever

MANAGEMENT DURING PREGNANCY (ANC)

- **Frequency:** More frequent than usual, attended by both obstetricians and cardiologists.
- **Examination:**
  - Obstetric examination (maternal and fetal)
  - Cardiac assessment and early detection of heart failure.
  - Recognition of risk factors (anemia, infection, hypertension)
- **Advice:**
  - Bed rest; 9 hours by night, 2 hours by day.
  - Guard against excessive weight gain and risk factors.
  - Dental care and use of an umbrella of antibiotics when there is tooth extraction.
- **Indications for Digitalis:**
  - If already on this regimen before pregnancy has started.
  - In cases of Class II or more or impending heart failure.
- **Hospitalization:**
  - Cases of Class II at 24 -32 weeks of pregnancy and one week before expected delivery date.
  - Cases of Class III and IV are admitted earlier and for longer periods.

MANAGEMENT DURING LABOUR

- *Proper pain relief* to minimize anxiety and tachycardia. Epidural anaesthesia may sometimes cause systemic hypotension, so it contraindicated in cases with right-left shunt.
- *Straining is prohibited*, to minimize venous return and decrease heart rate.
- Delivery in the *Semi-sitting position* with adequate oxygenation.
- *Digitalis* in cases at high risk for heart failure.
- *Shorten the second stage* by low-forceps when necessary.
- *Caesarean section* is done when obstetrically indicated.
- *Antibiotic cover* to guard against SBE.

N.B.: Ergometrin is NOT given ROUTINELY; but only when post-partum hemorrhage occurs. (to avoid sudden increase in venous return, predisposing to heart failure)

MANAGEMENT IN THE PUERPERIUM

There is more liability to heart failure due to increased venous return (reduced obstruction on the inferior vena cava, and mobilization of extracellular fluid).

- Monitoring of patient for at least two weeks (when COP return to pre-pregnant level).
- Breast-feeding is contraindicated ONLY if there is heart failure.
- Proper selection of a method of contraception, for adequate pregnancy spacing.

INDUCTION OF ABORTION

In the past, first trimester termination of pregnnacy was indicated in cases of Class III and IV heart classification, history of failure in a previous pregnancy, recent history of recrudescence of rheumatic fever, and in cases of right- left shunt.

In the past two decades induction of abortion is **rarely indicated** after introduction of recent surgical treatment of heart lesions, and availability of proper medical control of chronic cases.

Apart from some cases with pulmonary hypertension and right to left shunt, termination of pregnancy should not be encouraged in cardiac cases.

17 VENOUS THROMBOEMBOLISM IN PREGNANCY

INTRODUCTION AND INCIDENCE

Pregnancy is a known hyper-coagulable state, with a five-fold risk of venous thromboembolism over the non-pregnant condition. Between 0.5 and 3 of every 1000 pregnancies are complicated by symptomatic deep venous thrombosis (DVT). If untreated 25% may develop pulmonary embolism (PE), of which up to 15% are fatal.

AETIOLOGY

Pregnancy increases the risk for thrombosis due to the changes in the thrombotic and fibrinolytic activities that normally occurs during pregnancy, such as:

1. Increase in the levels of coagulation factors VII, VIII, IX, and X.
2. Increased fibrinogen levels.
3. Increased platelet activation.
4. Decreased protein-S and antithrombin III concentrations.
5. Venous stasis in the lower limbs due to pressure by the gravid uterus.

THROMBOPHILIAS

Some women are predisposed to thrombosis through changes in their coagulation- fibrinolytic systems, which may be either inherited or acquired.

A) Acquired thrombophilia

This is mostly associated with the antiphospholipid syndrome **(APS)**.

- **APS** is the combination of lupus anticoagulant **(LAC)** with or without, anticardiolipin antibodies **(ACA)**, with a history of **recurrent miscarriage** and or **thrombosis**.
- **APS** may or may not be associated with other autoantibody disorders as systemic lupus erythematosis (SLE).

B) Inherited thrombophilias

- Protein-C, protein-S, and Antithrombin III deficiency.

RISK FACTORS FOR DVT / PE DURING PREGNANCY

1. Maternal age > 35 years.
2. Pre-pregnancy weight > 80 kg (obesity).
3. Previous DVT (hyper-coagulability).
4. Pre-existing Thrombophilia (hyper-coagulability).
5. Severe varicose veins VVs (stasis + vasculitis).
6. Prolonged bed rest (stasis).
7. Multifoetal pregnancies (stasis)
8. Severe pre-eclampsia (haemoconcentration).
9. Caesarean section delivery (pelvic surgery).
10. Sepsis, especially pelvic (hyper coagulability + stasis + vasculitis) .

DEEP VEIN THROMBOSIS (DVT)

CLINICAL PRESENTATION

- **Symptoms:** DVT usually presents clinically with ***pain in the calf muscles*** associated with varying degrees of ***redness***, ***hotness***, or ***swelling***, especially ***unilateral oedema***.
- **Signs:**

  - Unilateral leg oedema, in association with redness, hotness, and tenderness

  - Pressure on the sole of the leg during extension results in severe pain in calf muscles

N.B.: Bilateral oedema of the lower limbs is a common finding during pregnancy that may confuse or delay the diagnosis.

INVESTIGATIONS

- **Colour Doppler Ultrasound:**

  - Allows assessment of the deep veins between the knee and the iliac veins and also allows for the dynamic assessment of femoral and iliac veins.

  - It is currently the preferred first-line method for investigating a suspected case of DVT during pregnancy being an accurate, non-invasive procedure.

- **Venography:**
- - Allows excellent visualization of veins both below and above the knee.
- - It is an invasive procedure, requiring the injection of a contrast medium and the use of X-ray. This method is not preferable during pregnancy.

MANAGEMENT

A) **Suspected cases:** Once **DVT** is suspected, **anticoagulant therapy** should be started with heparin, or low molecular weight heparin, in treatment doses until the diagnosis is confirmed or refuted by one of the above-mentioned investigations.

B) **Established cases:** Complete rest and immobilization for the first 24 hours, together with continuation of already started anticoagulant therapy in maintenance doses.

N.B.: in cases of DVT avoid dehydration by allowing excessive oral fluid intake or IV fluid infusion

ANTICOAGULANT THERAPY

1. Heparin and LMW heparin derivatives (Calcium heparin):

- *Advantages:* they are the preferred line of treatment due to:
  1. They do not cross the placenta, and therefore are not teratogenic
  2. Their effect can be stopped within hours by withholding further doses, therefore can be used safely before and after delivery without increased risk for haemorrhage
- *Mode of action:* Heparin prolongs the activated partial thromboplastin time (APTT), while calcium heparins affect factor X activity.
- *Side effects: on prolonged use:* prolonged use > 6 months may be associated with idiosyncratic reaction, thrombocytopenia and higher risk of osteoporosis.
- *The major draw back* in the use of parenteral anticoagulant therapy is the need for *daily* repeated S.C. or I.V. injections, which is troublesome for most patients.

2. Oral anticoagulants:

- *Advantage:* oral intake which is convenient for long use, and easily monitored and adjusted by INR measurement . It is the drug of choice that can be also used safely during lactation
- *Mode of action:* Warfarin prolongs the prothrombin time (PT).
- *Side effects:* The drug crosses the placenta and can cause limb and facial defects in the first trimester, and foetal intracerebral haemorrhage in the third trimesters.
- *Recently* some centres use it in the 2nd trimester, to avoid the prolonged uncomfortable use of heparin derivatives. Late in the 3rd trimester the patient is put back to heparin or LMW heparin, that will be stopped only for few hour during delivery or CS.

ANTICOAGULANT PROPHYLAXIS

- Women with a history of DVT occurring during or following a previous pregnancy are given prophylactic LMW heparin during subsequent pregnancies at least in the last trimester that extends to the end of the puerperium.
- Women with a history of DVT occurring in the non-pregnant state should be screened for thrombophilias, and offered anticoagulant prophylaxis at starting from the second trimester.
- Some women will require full anticoagulation throughout pregnancy as those with artificial heart valves and cases of APS with recurrent DVT, or cases with a history of previous PE

PULMONARY EMBOLISM (PE)

PE is one of the *fatal consequences* of undiagnosed or improperly managed cases *of DVT*. The thrombus readily formed in the lower limb or pelvic veins will fragment, and small emboli will travel through the venous circulation to be entrapped in the lungs.

Clinical presentation

- Mild breathlessness with inspiratory chest pain. associated with tachycardia, hypoxia, pleural rubs and sometimes ECG changes will settle the clinical diagnosis.
- A history of recent DVT event or the presence of clinical evidence of pelvic or lower limb DVT is an important clue for diagnosis and immediate management.

Management

Once suspected, early intervention by full I.V. anticoagulant therapy may be life saving. Full I.V. heparinization is immediately started, with supportive oxygen therapy.

Definitive diagnosis is then required via ventilation / perfusion scan or pulmonary angiography. A positive diagnosis of PE will have a major impact regarding long-term anticoagulation.

18 RH INCOMPATIBILITY (ERYTHROBLASTOSIS FETALIS)

The Rhesus factor
Incidence
Aetiology of RH incompatibility
Clinical Types
Diagnosis during pregnancy
Prophylaxis against maternal sensitization
Management during pregnancy
Neonatal management

It is an immunologic disorder characterized by excessive haemolysis of foetal RBCs by antibodies that pass through the placenta from maternal blood.

Erythroblastosis foetalis (also called haemolytic disease of the newborn), caused by Rh allo-immunization, is an important cause of infant morbidity and mortality.

THE RHESUS FACTOR:

The Rhesus (Rh) factor is a complex antigen that is present on the surface of RBCs. It consists of 3 pairs of genes Cc, Dd, Ee, the most important of which is the D. The D gene is dominant therefore an Rh-positive individual may be homozygous (DD) or heterozygous (Dd) and an Rh-negative individual has a (dd) genotype. The C and E antigens may also lead to incompatibility. But they are quite rare.

AETIOLOGY:

Rh-negative females develop anti-Rh antibodies if they were subjected to:

1. Blood transfusion with an Rh-positive blood. The immune system responds by the production of antibodies against the D antigen.
2. Married to Rh-positive male and get pregnant in Rh-positive baby. At time of delivery or (also disturbance of ectopic pregnancy, antepartum haemorrhage, amniocentesis, or external cephalic version) foeto-maternal haemorrhage occurs (passage of foetal RBCs to maternal circulation). These fetal Rh-positive RBCs stimulate the immune system to produce antibodies against the Rh-positive antigen.

INCIDENCE:

85% of the Caucasian population is Rh-positive while 15% is Rh-negative. However, the incidence of Erythroblastosis foetalis is less than 1% due to:

1. An Rh-negative female may get married to an Rh-negative male.
2. An Rh-negative female may get married to an Rh-positive heterozygous (Dd) male. The baby has a 50% chance of being Rh-negative.
3. ABO incompatibility between the mother and her foetus results in the haemolysis of transfused foetal cells into the maternal circulation before they can induce Rh antibody response.
4. Immunologic variation in the response to Rh-positive antigen. Some Rh-negative women are non-responders and others are to poor responders.
5. The first baby is usually unaffected because on the first exposure to Rh antigen, during the first

pregnancy, the immune system responds by producing IgM antibodies (first immune response) that have a large molecular weight so can not cross the placenta. While on the second exposure, the immune system responds by producing IgG antibodies (second immune response) that have a small molecular weight thus cross the placenta affecting the second baby. It is to be noted that the first baby can also be affected if the mother had previous Rh-positive blood transfusion or previous abortion

CLINICAL TYPES:

In all types, haemolysis of foetal Rh-positive RBCs occurs due to the passage of anti Rh antibodies from maternal to foetal circulation in a previously sensitised pregnant woman.

I. Congenital haemolytic anaemia: (Mildest form)

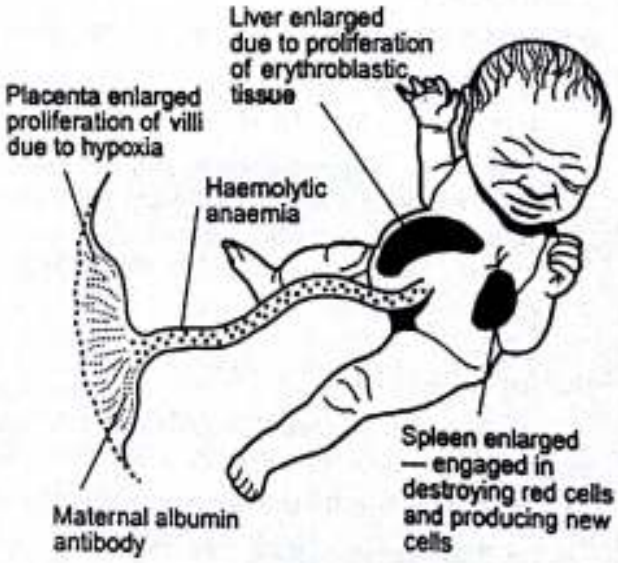
- Haemolysis results in foetal anaemia that develops 2 weeks after birth.
- Due to faster rate of erythropoiesis, erythroblasts (immature nucleated RBCs) are produced, hence, the name Erythroblastosis foetalis.

II. Icterus gravis neonatorum: (Commonest and moderate form)

- The baby is delivered anaemic but never jaundiced at birth as the placenta carries on the function of conjugating foetal bilirubin.
- Hepatosplenomegaly is usually present due to faster erythropoiesis.
- Jaundice develops within 48 hours after birth due to the inability of the foetal liver to conjugate the high amount of bilirubin produced by the haemolysed RBCs.
- **kernicterus** develops when foetal bilirubin level exceeds 20 mg%. Bilirubin crosses the blood brain barrier and becomes deposited in the basal ganglia of the brain stem, a serious condition that may lead to neonatal death or mental retardation.

III. Hydrops foetalis: (Severest form)

- Intrauterine foetal death usually occurs due to severe haemolytic anaemia that leads to heart failure. If the baby was born alive, it dies within few hours.
- The foetus shows generalized oedema, pleural effusion and ascites due to heart failure.
- Hepatosplenomegaly.
- The placenta is large and oedematous.
- If ultrasound is done during pregnancy the foetus shows the "Buddha" attitude due to flexion of the thighs and abdominal distension and shows a halo around the skull due to scalp oedema.



DIAGNOSIS DURING PREGNANCY

Rh group should be checked in all pregnant women at the first antenatal visit and if Rh-negative:

- Ask about previous blood transfusion, foetal anaemia, neonatal jaundice or IUFD.
- Determine the Rh group of the husband and if positive proceed for:
  - **Indirect coomb's test:** to detect the presence and the titre of anti-Rh antibodies (Anti-D IgG). If the titre =1/16 amniocentesis is indicated. If <1/16 repeat the test every 4 weeks.
  - **Amniocentesis:** is done, if antibody titre is >1/16, to determine the amount of bilirubin in the amniotic fluid that reflects the degree of haemolysis in foetal blood.
  - **US:** May show foetal hepatosplenomegaly, oedema, or Buddha attitude.

DIAGNOSIS AFTER LABOUR

Cord Blood is obtained for Rh grouping and if positive:

- Assess haemoglobin level to detect foetal anaemia (normal 18 gm%),
- Assess serum bilirubin to detect jaundice (normal = 2 mg%).
- **Direct Coombs' test:** detects the antibodies absorbed to the RBCs (sensitisation).

PROPHYLAXIS AGAINST MATERNAL SENSITIZATION AND ERYTHROBLASTOSIS FOETALIS:

1. Rh-negative females should never receive Rh-positive blood transfusion.
2. Anti-D immunoglobulin should be given to all Rh-negative **non-sensitised** (negative indirect coomb's test) females married to Rh-positive males in the following conditions:
   - After delivery of an Rh-positive baby to destroy foetal RBCs before they initiate maternal immune response (300 mcg Anti-D I.M. within 72 hours of delivery).
   - At time of abortion, disturbance of ectopic pregnancy, antepartum haemorrhage, amniocentesis, or version (50-100 mcg Anti-D I.M.).
   - Recently, 300 mcg of Anti-D immunoglobulin I.M. are given at 28 weeks of pregnancy is recommended to prevent sensitisation during pregnancy.

NB: In already sensitised women (with Anti-D antibodies in their blood) as detected by positive indirect comb's test or who have a previously affected baby, Anti-D prophylaxis is contraindicated.

MANAGEMENT DURING PREGNANCY:

1. Intrauterine blood transfusion:

This is indicated if the foetus is severely affected before 34 weeks gestation. Rh-negative group O blood is injected into the peritoneal cavity of the foetus or into the umbilical vein. It is done under ultrasonographic guidance and local anaesthesia.

2. Termination of pregnancy:

It is indicated if the foetus is severely affected after 34 weeks gestation. Induction of labour or caesarean section is done when lung maturity is demonstrated by L/S ratio.

NEONATAL MANAGEMENT:

Exchange transfusion:

10-20 ml of blood are withdrawn from the umbilical vein and replaced with the same amount of Rh-negative group O blood and repeated till 80-90% of the blood is replaced.

The aim is to remove the antibodies, remove the antigen (replace the Rh-positive cells with Rh-negative ones), correct anaemia, and correct jaundice (remove bilirubin).

19 ANATOMY OF THE FEMALE PELVIS AND FOETAL SKULL

ANATOMY OF THE FEMALE PELVIS

The female pelvis is formed of two innominate bones, sacrum, and coccyx

- The innominate bones are joined together anteriorly at the symphysis pubis and posteriorly to the sacrum at the sacroiliac joint.
- The coccyx is joined with the sacrum at the sacrococygeal joint.

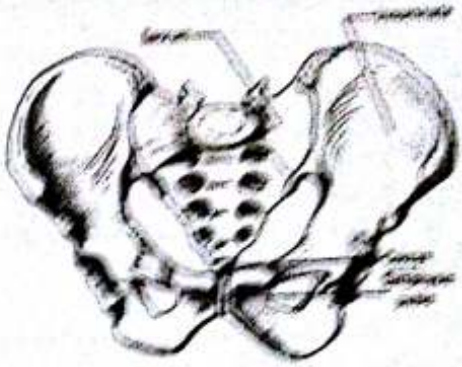


Fig 19:1 Bones of female pelvis

The female pelvis is divided into a false and a true pelvis separated by the pelvic brim.

- **The false pelvis:** has no obstetric importance.
- **The true pelvis:** divided into: inlet, cavity and outlet.

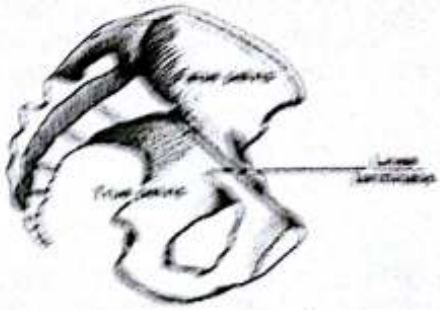


Fig 19:2 True and false pelvis

1. THE PELVIC INLET:

- **Boundaries:** The pelvic inlet is bounded on each side by the promontory of the sacrum, the alum of the sacrum, the sacro-iliac joint, iliopectineal line of the innominate bone iliopectineal eminence, upper border of the superior pubic ramus, pubic tubercle upper border of the pubic bone (the pubic crest) and the upper border of the symphysis pubis.
- **Pelvic inclination:** The plane of the pelvic inlet makes an angle 55° with the horizon in the standing position.
- **Shape of the pelvic inlet:** oval transversely
- **Diameters of the Pelvic Inlet:**

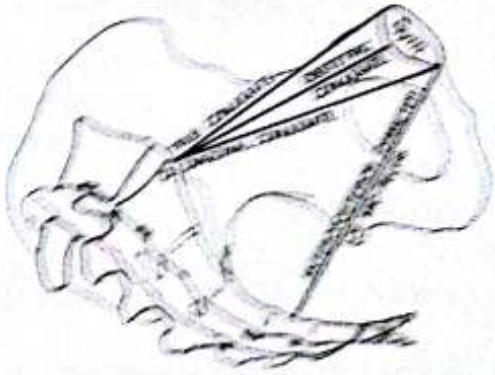


Fig 19:3 AP diameters of the pelvis

A) <u>Anteroposterior Diameters of the Inlet (APD):</u>

- *Anatomical APD (true conjugate):* ***11 cm,*** from the tip of the sacral promontory to the upper border of the symphysis pubis (SP).
- *Obstetric conjugate:* ***10.5 cm,*** from the promontory to the most bulging point on the back of the SP.
- *Diagonal conjugate:* ***12.5 cm,*** from the promontory to the lower border of the symphysis pubis (measured by vaginal examination).

B) *Transverse diameters of the inlet (TD) :*

- *Anatomical TD:* ***13 cm,*** between the farthest points on the iliopectineal lines. It is nearer to the sacral promontory (not used by the fetus).
- *Obstetrical TD:* ***11-12 cm,*** it bisects the true conjugate and is slightly shorter than the anatomical (It is of obstetric importance).

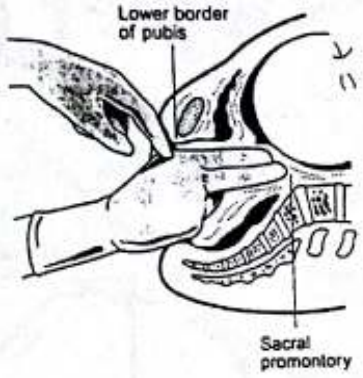


Fig 19:4 Diagonal conjugate

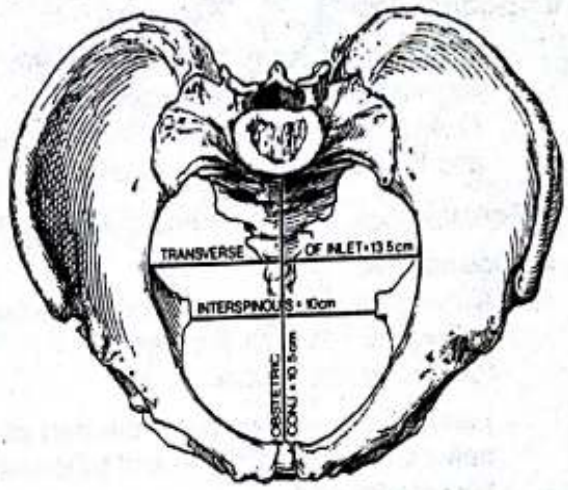


Fig 19:5 TD of the inlet

C) *Oblique diameters of the inlet (OD):*

- *Right and left oblique diameters (ROD & LOD):* ***12 cm,*** The usual diameters for engagement. Extend from the sacroiliac joint to the opposite iliopectineal eminence. The right oblique starts from the right sacro-iliac joint to the left iliopectineal. The right oblique diameter is slightly longer than the left because:
  - The left oblique diameter is shortened by presence of the pelvic colon
  - The left oblique diameter is anatomically slightly shorter.
- *Sacro-cotyloid diameter (SCD):* ***9-9.5 cm,*** extends from the promontory of the sacrum to the right and left iliopectineal eminence. (RSCD & LSCD).

II. THE PELVIC CAVITY

- **Boundaries:** It is bounded above by the pelvic brim, below by the plane of least pelvic dimensions, anteriorly by the symphysis pubis and posteriorly by the sacrum.
- **Planes of the pelvic cavity:** *(APD)*
  - It is *"the plane of greatest pelvic dimensions"*.
  - It is the widest part of the pelvic cavity.
  - It extends from the center of the back of the symphysis pubis to the junction between the 2nd and 3rd pieces of sacrum. It is round with all diameters equal 12.5 cm.
  - Internal rotation of fetal head occurs when the BPD lies in this plane.
- **Characters of the pelvic cavity**
  - The posterior wall is concave and this serves to increase the capacity of the pelvis.
  - It has a short anterior wall (symphysis pubis) and a long posterior wall (sacrum).

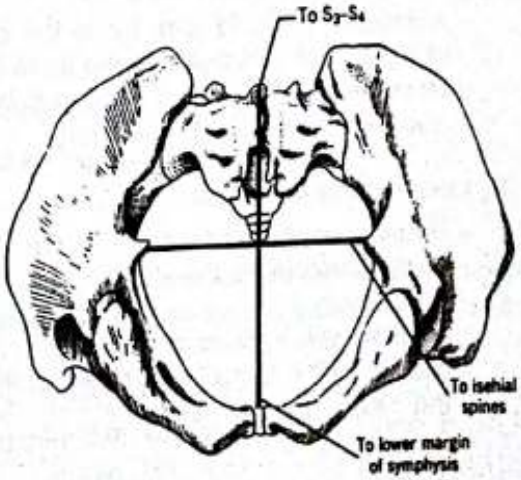


Fig 19: 6 pelvic cavity

III. THE PELVIC OUTLET

a. ***Anatomical outlet***: is a Lozenge shaped area made of 2 triangles in 2 planes.

- The anterior sagittal plane
- The posterior sagittal plane

- **Boundaries:**
  - *Anteriorly:* The lower border of the SP.
  - *Posteriorly:* The tip of the coccyx.
  - *Laterally:* The pubic arch, ischial tuberosities and the sacrotuberous ligament.

b. ***Obstetric outlet***: It is a segment of the pelvis.

- **Boundaries:**
  - *Above:* plane of the least pelvic dimensions.
  - *Below:* The anatomical outlet.
  - *Posteriorly:* The coccyx.
  - Lateral walls: made up of the part of ischium between the ischial spine and tuberosity
  - No anterior wall

It should be noted that coccyx moves backward in the 2nd stage of labor. So the lowest part of the posterior wall of the pelvic cavity becomes the tip of the sacrum and not the tip of the coccyx.

- Plane of the Obstetric Outlet:

It is known as "*plane of least pelvic dimensions*". It extends from lower border of the SP to the tip of the sacrum. It lies at the level of the ischial spines.

A) Transverse diameters:

- *Bituberous diameter:* ***11 cm***, Between the 2 ischial tuberosities.
- *Bispinous diameter:* ***10.5 cm***, Between the tip of the 2 ischial spines.

B) Anteroposterior diameters (APD)

- *Anatomical APD:* ***11 cm***, From the lower border of the symphysis pubis to the tip of the coccyx
- *Obstetric APD:* ***13 cm***, From the lower border of the SP to the tip of the sacrum

C) Longitudinal diameters:

- *Posterior sagittal diameter:* ***7-10 cm***, from the tip of the sacrum to the center of the bituberous diameter.
- *Anterior sagittal diameter:* ***6-7 cm***, from the lower border of the symphysis pubis to the center of the bituberous diameter

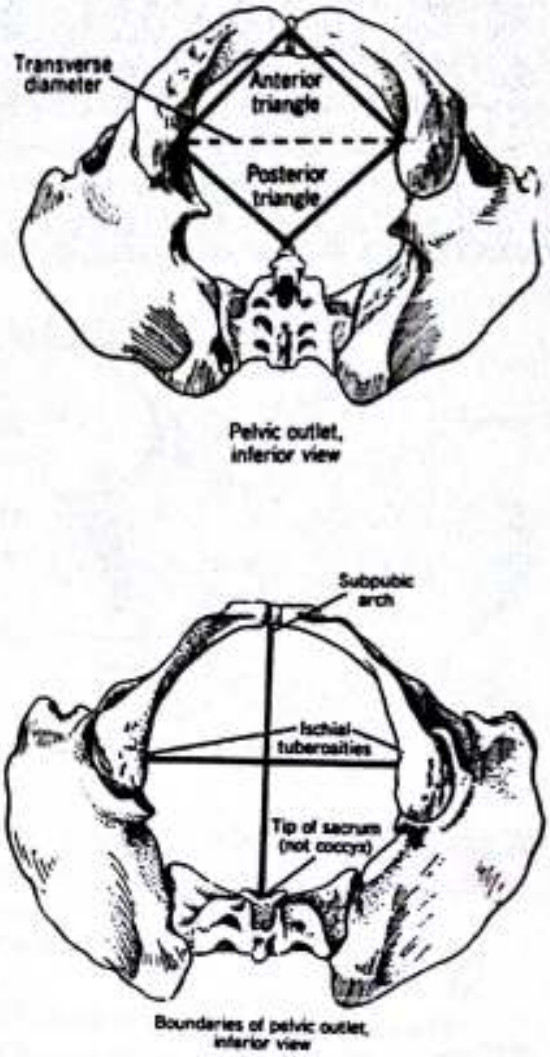


Fig 19: 7 pelvic outlet Inferior view

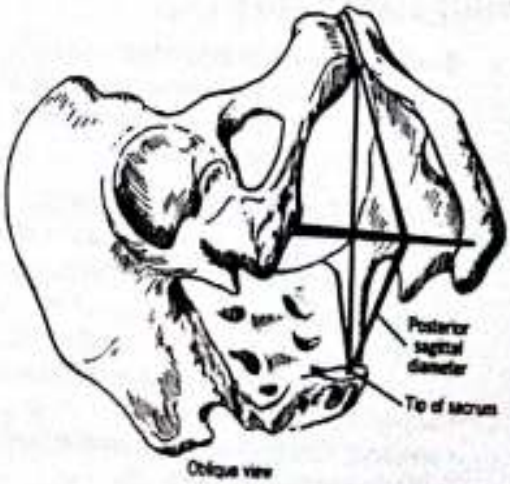


Fig 19: 8 obstetric outlet Inferior view

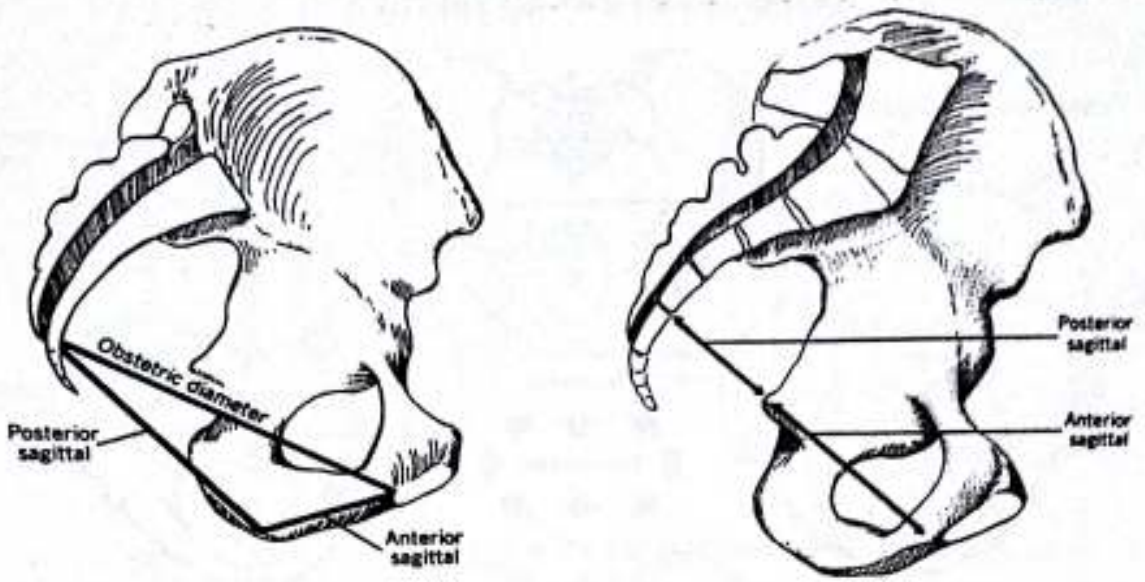


Fig 19: 9 obstetric outlet
Inferior view

Thom's dictum :

For the average sized head to pass through the outlet, the sum of the bituberous and posterior sagittal diameters must exceed 15 cm , accordingly the bituberous should exceed 8 cm.

The subpubic angle :

It is normally between 90° and 100° while in the male it may be reduced to 70°

Importance of the level of the ischial spines:

- It is the level of the levator ani, which is attached to the ischial spines.
- The external os of the cervix & the vaginal vault lie at this level.
- It is the level of the plane of the least pelvic dimensions.
- The obstetric axis changes its direction at this level.
- The head is considered engaged if the vault is felt at or below this level.
- Forceps should not be applied when the fetal head is above this level.
- Anesthetic agent for pudendal nerve block is injected at this level.

Pelvic Axis:

1. **The anatomical axis "Curve of Carus"** is an imaginary line joining the centers of the planes of the inlet, cavity & outlet. It is C-shaped & has no obstetric importance because this is not the route of the fetus during labor.
2. **The obstetric axis** is L-shaped and corresponds to decent of the fetal head during labor i.e. *downwards* and *backwards* to the level of the ischial spines then *downwards* and *forwards*, so it is J-shaped.

VARIATION IN THE SHAPE OF FEMALE PELVIS

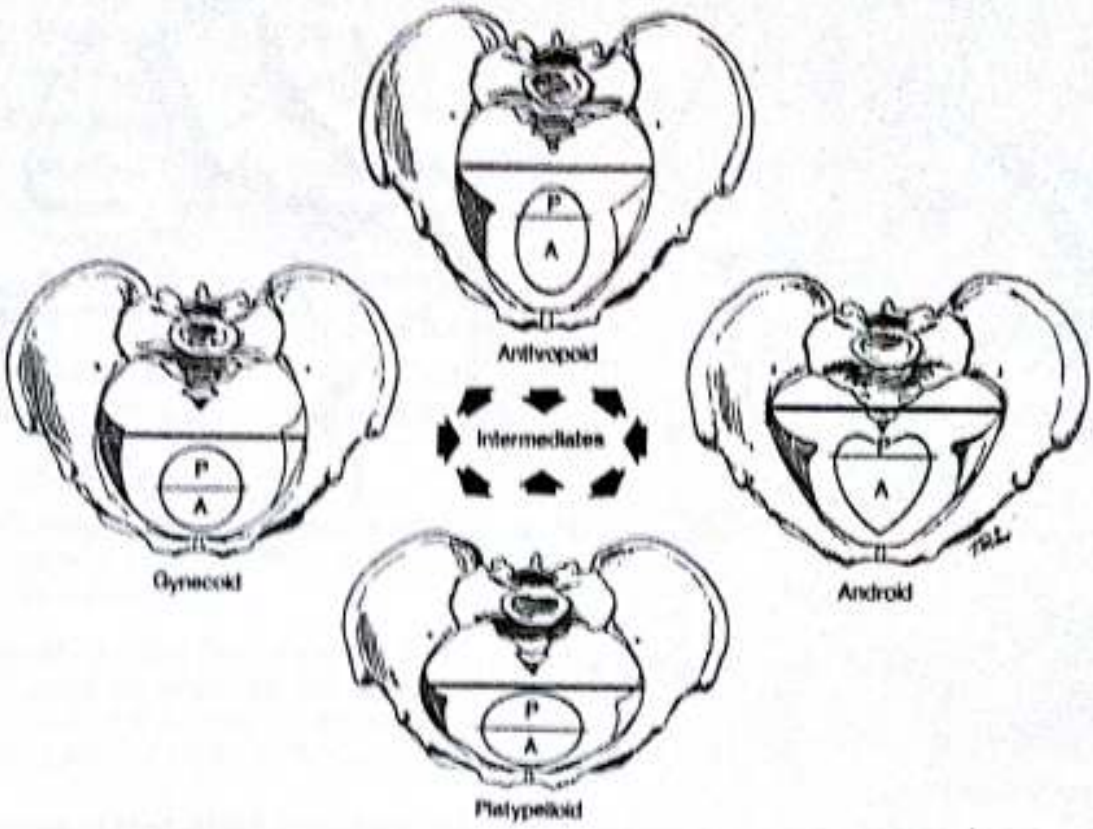


Fig 19: 10 different shapes of the female pelvis

RADIOLOGIC PELVIC SHAPES (Caldwell and Moloy classification):

NB. Gynecoid pelvis is the most common and most favorable.

1. Gynecoid pelvis (50%)	2. Android pelvis (20%)
- The inlet is slightly transverse oval.	- It is heart-shaped with narrow fore-pelvis.
- The sacrum is short & concave.	- The sacrum is long & shallow.
- Wide sacro-sciatic notch.	- Narrow sacro-sciatic notch.
- Wide sub-pubic angle (90-100°).	- Narrow sub-pubic angle "<70°".
- Parallel or slightly converging sidewalls.	- Converging sidewalls.
- Non projecting ischial spines.	- Projecting ischial spines.

3. Anthropoid pelvis (25%)	4. Platypelloid "flat" pelvis (5%)
- Inlet: Longitudinal oval	- Inlet: Transverse oval
- Long anteroposterior diameters	- Short anteroposterior diameters
- Short transverse diameters	- Long transverse diameters.
- Narrow sacrosciatic notch	- Wide sacrosciatic notch.
- Narrow subpubic angle.	- Wide subpubic angle.

ANATOMY OF THE FETAL SKULL

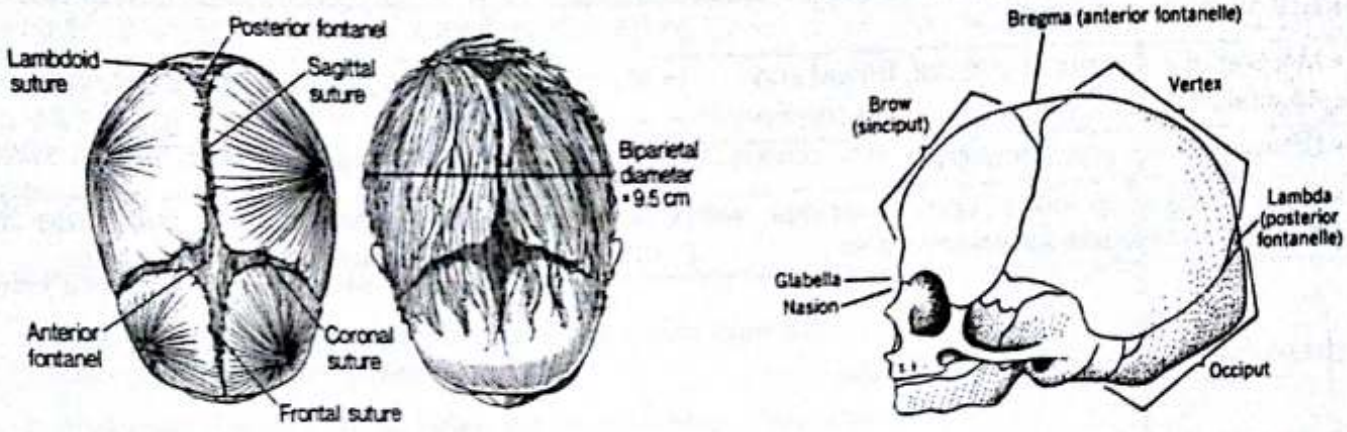


Fig 19: 11 different parts of foetal skull

The foetal skull consists of 3 parts separated by sutures and fontanelles.

1. **Base:** from the chin to the foramen magnum.
2. **Face:** from the chin to the root of the nose and supraorbital margins.
3. **Vault:** consists of three regions.
   - Brow: from the root of the nose and supraorbital margins to the anterior fontanelle (bregma) and coronal sutures. It includes the 2 frontal bones separated by the frontal suture.
   - Vertex: from the bregma and coronal sutures to the posterior fontanelle and lambdoid suture. It consists of 2 parietal bones separated by the sagittal suture. It is bounded laterally by the parietal eminences.
   - Occiput: from the posterior fontanelle and lambdoid suture to the foramen magnum.

Comparison between bones of the vault to that of base and face

Vault	Base and Face
1. Thin and regular.	*Thick and irregular*
2. Develop in membranes.	*Develop in cartilage*
3. United by sutures and can undergo molding	*Don't undergo molding*

4. **Sutures:** Frontal, sagittal, coronal, lambdoid, and temporal sutures.
5. **Fontanelles:**

They are 6 in number present at points of crossing of sutures. 4 fontanelles lie at the anterior and posterior ends of the temporal suture on either side. They are of no clinical importance. The other 2 fontanelles "anterior" and "posterior" have some clinical importance in

- Diagnosis of vertex presentation.
- Position of the occiput.
- Degree of flexion of the fetal head.

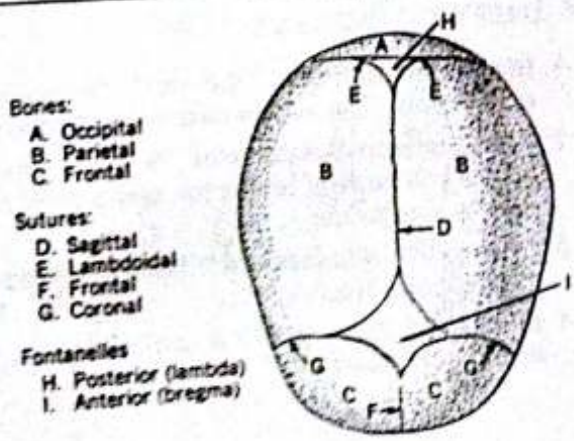


Fig 19: 12 different parts of foetal skull Bones, sutures & fontanelles

Comparison between the anterior and posterior fontanelles

Anterior fontanelle (bregma)	Posterior fontanelle (lambda)
• Large, Lozenge shaped.	• Small, triangular.
• Soft membranous floor.	• Hard, bony floor.
• Meeting of 4 sutures: 2 coronal, frontal and sagittal.	• Meeting of 3 sutures: sagittal and 2 lambdoid.
• Disappears 1.5 years after birth.	• Completely ossified at full term
• The surrounding bone don't over-ride with molding as they are widely separated.	• With molding, one parietal bone over-rides the other and both over-ride the occipital bone.

DIAMETERS OF THE FOETAL SKULL

A. Longitudinal Diameters (LD, *engaging diameters*):

1. ***Suboccipito-bregmatic "9.5 cm":*** from below the occipital protuberance to the center of the anterior fontanelle (bregma). *The engaging diameter when the head is fully flexed*
2. ***Suboccipto-frontal "10 cm":*** from below the occipital protuberance to the anterior end of the anterior fontanelle.
3. ***Occipito-frontal "11.5 cm":*** from the occipital protuberance to the root of the nose. *The engaging diameter when the head is deflexed (occipito-posterior).*
4. ***Submento-bregmatic "9.5 cm":*** from the junction of the chin and neck to the center of the bregma. *The engaging diameter when the head is extended (Face).*
5. ***Submento-vertical "11.5 cm":*** from the junction of the chin and neck to the vertical point (a point on the sagittal suture midway between the anterior and posterior fontanelles)
6. ***Mento-vertical "13.5 cm":*** from the tip of the chin to the vertical point. *The engaging diameter when the head is partially extended (Brow).* Larger than any pelvic diameter.

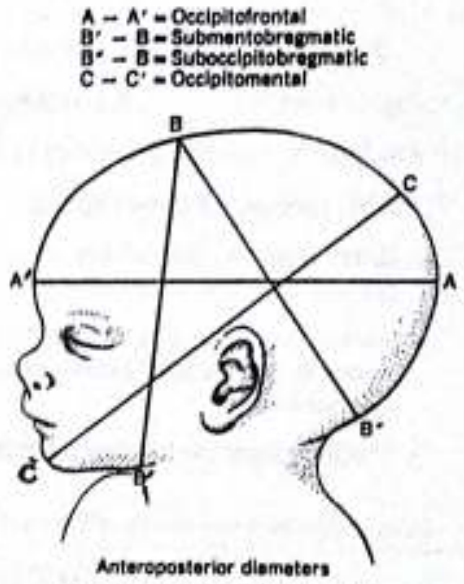


Fig 19: 13 LD of foetal skull

B. Transverse Diameters (TVD):

1. ***Biparietal diameter "9.5 cm":*** Between the 2 parietal eminences. *(The widest transverse diameter)*
2. ***Superparietal-Subparietal "9 cm":*** Between a point above one parietal eminence and a point below the other parietal eminence.
3. ***Bitemporal diameter "8":*** Between the anterior ends of the temporal sutures.
4. ***Bimastoid diameter "7.5 cm":*** Between the tips of the mastoid processes.

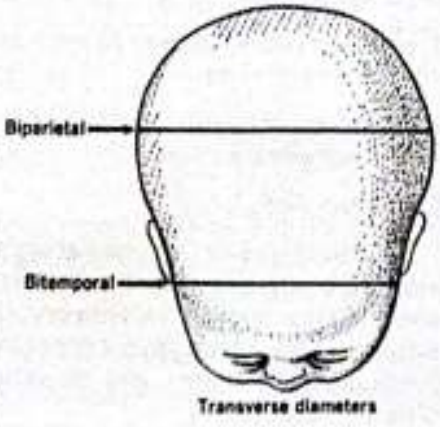


Fig 19: 14 TVD of foetal skull

OBSTETRICAL DEFINITIONS

1. FETAL ATTITUDE: The relation of the fetal parts to each other (Usually complete flexion attitude; in face it is complete extension of the-head).

2. FETAL LIE: The relation between the long axis of the fetus to that of the mother (Longitudinal in cephalic and breech and transverse in shoulder)

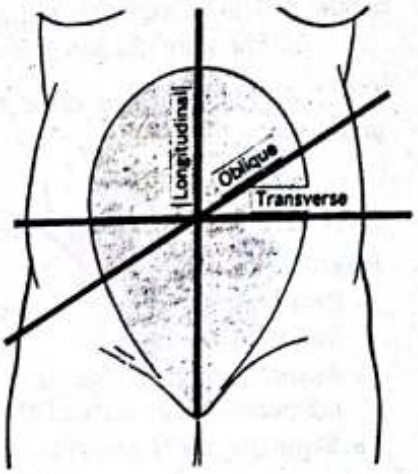


Fig 19: 15 Foetal lie

3. FETAL PRESENTATION

It is that part of the fetus related to the pelvic brim and first felt by vaginal examination. It may be;

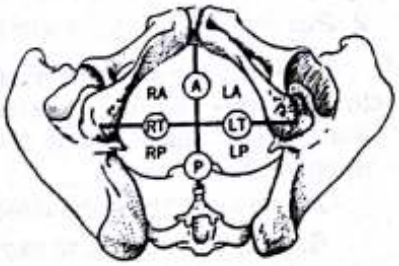
A. Cephalic (96%): Vertex when the head is flexed, face when extended and brow when it is midway between flexion and extension. Cephalic presentation is much more common as the fetus is adapted to the pyriform-shaped uterus (with the larger breech in the wide fundus)

B. Breech (3.5%): The buttocks with or whithout the limbs form the presenting part.

C. Shoulder (0.5%): The scapula with or whithout the arms form the presenting part.

- **N.B.: MALPRESNTATION** is any presentation other than the vertex.

4. FETAL DENOMINATOR: A landmark on the presenting part used to denote the position.
   - In vertex it is the occiput
   - in face it is the chin
   - in breech it is the sacrum
   - in shoulder, it is the scapula.

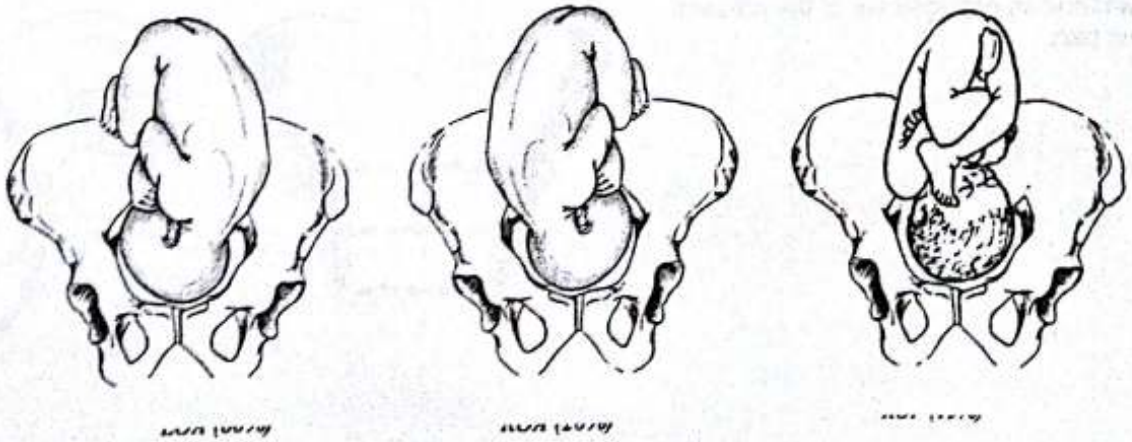


A = Anterior
P = Posterior
RT = Right transverse
LT = Left transverse
RA = Right anterior
RP = Right posterior
LA = Left anterior
LP = Left posterior

5. FETAL POSITION:

It is the relation of the back of the fetus to the right or left side of the mother and whether it is directed anteriorly or posteriorly.

There are 4 common classical positions in vertex presentation:



Occipito anterior positions are more common than occipito-posterior positions because the concavity of the front of the fetus fits into the convexity of the vertebral column (Lumbar lordosis) of the back of the mother.

LOA is more common than **ROA** and **ROP** is more common than **LOP** as in **LOA** and **ROP** the head enters the pelvis in the right oblique diameter which is more favorable than left oblique because: a) The pelvic colon reduces the length of the left oblique.

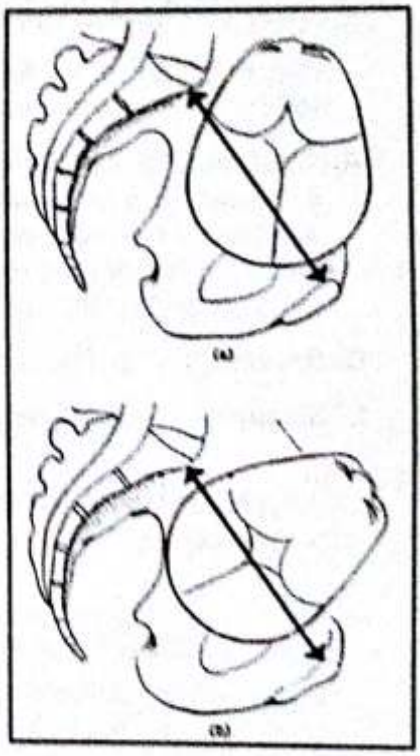
b) The right oblique is usually slightly longer than the left oblique.

N.B.: *In addition there are other 4 positions: right and left occipito-transverse and direct occipito-anterior and posterior.*

SYNCLITISM: When the 2 parietal bones are at the same level.

Asynclitism:

- One parietal bone is at a lower level than the other due to lateral flexion of the head.
- Asynclitism brings shorter diameter to enter the pelvis (Superparietal subparietal 9 cm instead of biparietal 9.5 cm)
- Slight degree of asynclitism occurs in normal labour



Correction of Asynclitism

1. Anterior parietal bone presentation:

The anterior parietal bone is lower and the sagittal suture is near to the promontory. It occurs more in multigravidas due to laxity of the abdominal wall.

2. Posterior parietal bone presentation:

The posterior parietal bone is lower and the sagittal suture is near to the symphysis. It occurs in primigravidas due to tense abdominal wall. Anterior parietal bone presentation is more favorable than posterior because:

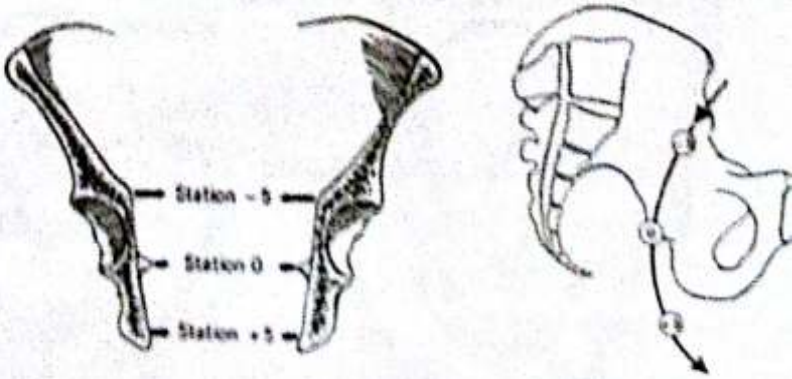
A. The head lies in the direction of the axis of the pelvic inlet.

B. During correction of asynclitism, the head meets only the resistance of the sacral promontory, while in posterior parietal bone presentation the head meets the resistance of the- whole back of the symphysis pubis.

C. Uterine rupture is more likely to occur in posterior parietal bone presentation because the anterior wall of the lower uterine segment becomes markedly stretched.

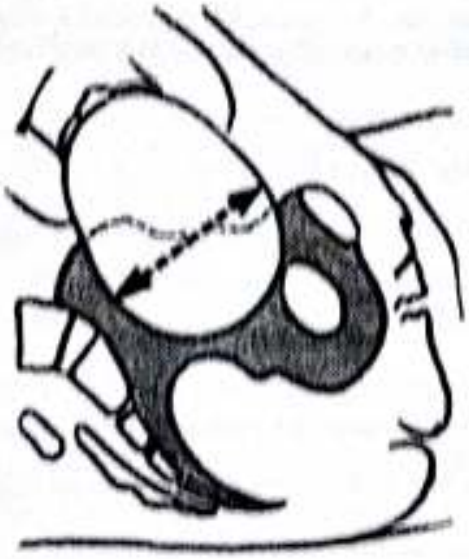
7. STATIONS OF THE PELVIS

Station 0 is marked by the level of the ischial spines. It is the level that determines engagement of the presenting part.

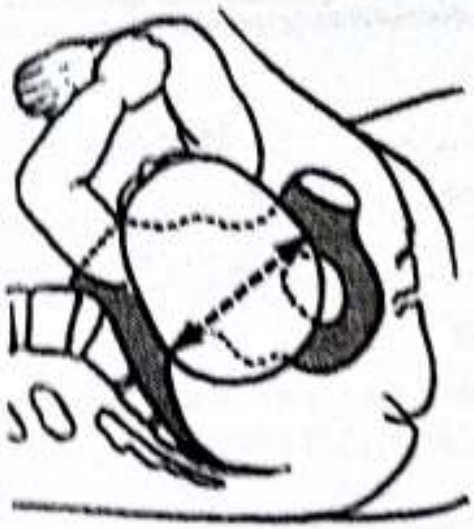


ENGAGEMENT

It is the passage of the widest transverse diameter of the presenting part through the plane of the pelvic inlet (the biparietal in case of the head, the bitrochanteric in case of breech).



Non engaged head



Engaged head

The engaged head can't be grasped by the first pelvic grip but it can be palpated by the 2nd pelvic grip. Vaginally, the vertex is felt at or below the level of the ischial spines.

In primigravidas, engagement occurs in the last 3 or 4 weeks of pregnancy due to the tonicity of abdominal and uterine muscles while in multiparas, the head commonly engages at the onset of labor or at the beginning of the 2nd stage.

Causes of non-engagement in the last 4 weeks in the primigravida:

A. Maternal causes:

1. Contracted pelvis
2. Pelvic tumors
3. Placenta praevia
4. Full bladder or rectum
5. Atony of the abdominal muscles

B. Fetal causes:

1. Occipitoposterior position (commonest cause)
2. Hydrocephalus
3. Hydramnios.
4. Multiple pregnancy
5. Malpresentations as face and brow

20 THE PHYSIOLOGY OF LABOUR

DEFINITIONS

- Labour: is the process of expulsion of the fetus from the uterus through the birth canal.
- Duration of labour: 12-18 hours in the primigravida, and 6-10 hours in the multigravida

ANATOMIC AND PHYSIOLOGIC CONSIDRATIONS

- The myometrium of the uterus comprises three muscles layers;
  - *The inner layer:* in which muscle fibres are arranged in a circular pattern
  - *The thick intermediate layer:* comprises oblique muscle fibers (interlocked muscle fibers).
  - *The outer layer;* which runs longitudinally over the fundus.
- The interaction between the two intramyometrial key proteins **actin** and **myosin**, brings about contraction, while their separation brings about relaxation. An increase in intra-cellular free calcium ions is needed for contraction.
- Gap junctions facilitate cell to cell communication with passage of various products of metabolism and electric current . These gap junctions increase in size and number with actual labour process.

THE ONSET OF LABOUR

The cause of the onset of labor is unknown, however proposed theories include;

1. Progesterone (P) withdrawal: a sharp decline in P level near term may help in initiation of labour pains, as P is known to decreases the frequency of the uterine contractions. Anti-progesterone have been used successfully for induction of abortion in the first trimester.

2. Prostaglandin release: PGLs stimulate uterine contractions and ripening of the cervix. Rupture of the membranes is associated with release of PGLs stored in the foetal membranes, and onset of uterine contractions. On the other hand anti- PGLs may play a role in inhibiting uterine contractions during the management of cases of preterm labour (PTL).

3. Estrogen-oxytocin effect: As pregnancy advances, estrogen (E) increases oxytocin receptors in the uterus, i.e. at the end of pregnancy the uterus is well prepared for oxytocin to act.

4. Placental Oxytocinase decrease: that occurs near term allows oxytocin produced by the posterior pituitary gland to act freely on the uterus, inducing uterine contractions.

5. Uterine distension theory: over distension of the myometrium, as in cases of multifoetal pregnancy and polyhydramnios, may explain the tendency to PTL in such conditions.

6. Fetal cortisol theory: An increase in foetal cortisol in the last trimester is important to complete foetal lung maturity and prepare for the onset of labours. Foetal cortisol deficiency may be associated with post-term pregnancy in cases of anencephaly due to lack of ACTH secondary to pituitary gland aplasia.

THE FORCES OF LABOUR

The powers by which the cervix is dilated and the fetus is expelled through the birth canal include:

1. The uterine muscle contractions and retraction (**true labour pains**)
2. **Auxiliary forces** (maternal bearing down and unfolding of the fetus)

CHARACTERISTICS OF TRUE LABOR PAINS

- True labour pains are involuntary, rhythmic, intermittent, coordinated, and painful.
- They show gradual increase in frequency, strength, and duration, by time.
- They start near the fundus (upper segment) and propagate to the lower uterine segment (LUS)
- Retraction occurs at the end of each contraction leading to descent of the presenting part and dilatation of the cervix.

1. Polarity and fundal dominance:
   - The upper uterine segment (UUS) contracts and retracts i.e. active to expel the fetus
   - The lower uterine segment (LUS) stretches and elongates i.e. passive to dilate the cervix.
   - A physiological retraction ring develops between the UUS and LUS at a level behind the S. pubis. It is normally neither seen or felt.

2. Uterine Contractions are Painful: due to:
   - Compression of the myometrial blood vessels leading to hypoxia
   - Compression of the nerve ganglia
   - Stretch of the peritoneum on the LUS
   - Stretch of the cervix during its dilatation

3. Contractions are followed by Retraction:
   - Following the end of each contraction progressive shortening of the uterine smooth muscle cells occur, i.e. muscle fibres relax but do not return to their original length (retraction).
   - Values of uterine muscle retraction include:
     A. It helps dilatation the cervix in the 1st stage of labor.
     B. Expulsion of the fetus in the 2nd stage.
     C. Separation and expulsion of the placenta in the 3rd stage.
     D. Control placental site bleeding "4th stage of labor"

4. Contractions are Intermittent:
   For temporary relief of pain, and to avoid muscle fatigue and prevent foetal hypoxia.

5. Increase in frequency, strength and duration:
   - The intrauterine pressure at which pain is felt is about 20-25 mm Hg
   - In the active stage of the first stage of labour there are 3 contractions every 10 minutes, each lasts 50-60 seconds, with intrauterine pressure reaching 50-60 mm Hg.
   - The intrauterine pressure in the 2nd stage reaches 80 mm Hg.

6. Contractions are Coordinated:
   - All fibers contract simultaneously to achieve maximal force.
   - The excitation wave starts from a pace maker situated near both cornu
   - Gap junction; allow spread of the wave to the adjacent muscles.
   - Uterine contractions are due to interaction of myosin and actin. This interaction is caused by an enzyme called myosin light chain kinase.
   - Myosin light-chain kinase is stimulated by prostaglandins and calcium ions.

THE AUXILLARY FORCES OF LABOUR:

A. Maternal bearing down in the 2nd stage of labour: leads to increased intra-abdominal pressure helping foetal expulsion and facilitating placental separation and expulsion
B. Unfolding of the foetus: is the final step which helps delivery of the foetus through the birth canal

THE CLINICAL DIAGNOSIS OF LABOUR

SYMPTOMS:

- True labor pains : Progressive rhythmic colicky abdominal pains, starting at the fundus of the uterus, with gradually increasing frequency and intensity. Pain later on is referred to the lower back due to progressive cervical effacement and dilatation.

- The bloody show: expulsion of the mucus plug within the cervical canal streaked with blood, may occur with early cervical dilatation. The blood is due to ruptured small vessels as a result of separation of the bag of waters from the lower uterine segment.

SIGNS:

- Abdominal examination:
  - The abdominal wall becomes rigid and tense during the uterine contraction
  - In between contractions it relaxes making foetal parts easily palpated.

- Vaginal Examination:

1. *Cervical effacement and dilatation:*
   - The most important sign for onset of labour.
   - Before labour starts the cervical canal is formed and the internal os is closed.
   - In the multipara the cervical canal may admit one finger (2 cm) before labour starts.
2. *Bulging bag of fore-waters:* when occurs during uterine contractions is considered a sure sign that labour has started.

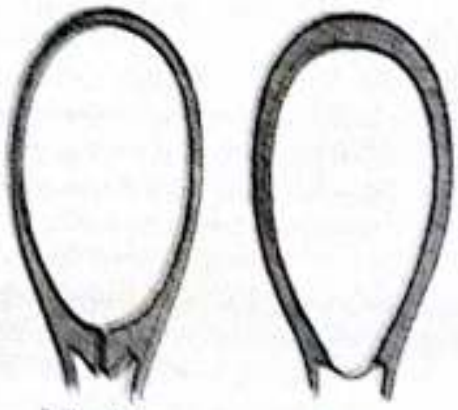


Fig 20.1 cervical changes in labour

Clinical differences between true and false labour pains

	True labor pains	False labor pains
- *Site of the pain*	- Abdominal and back ache	- Abdominal
- *Rhythm*	- Regular	Irregular
- *Frequency, strength, duration*	- Increasing	Stationary or decreasing
- *Bulging of membranes*	- Present	Absent
- *Cervical effacement*	- Usually present	Absent
- *Cervical dilatation*	- Progressive	Absent or stationary
- *Relief by sedation*	- Not relieved	Usually relieved

The COURSE OF LABOR

PRODROMA OF LABOR (pre labour)

- *False labor pains:* see before.
- *Increased vaginal discharge:* due to pelvic congestion.
- *If the head is engaged;* especially in primigravidas, descent of the head in the pelvis results in;
  - Pelvic pressure symptom as frequent micturition and difficult walking.
  - Lightening: relief of the upper abdominal pressure symptoms as dyspnea and dyspepsia.
  - Shelfing: descent with slight forward tilt of the uterine fundus detected in standing position.

THE STAGES OF LABOR

Classically there are **three stages of labor** which include; cervical dilatation, delivery of the foetus, and expulsion of the placenta. Some authorities add the first hours after delivery as a 4th stage since many complications may occur in this period.

THE FIRST STAGE OF LABOR

- It is the stage of **cervical effacement and dilatation**.
- It **starts** by onset of true labour pains, and **ends** by full dilatation of the cervix (10) cm.

The average duration of the first stage; varies from one patient to another;

- In primigravidas: around 10-18 hours
- In multigravidas: it is usually of **shorter** duration (6-10 hours), due to rapid cervical dilatation
- In malpositions and malpresentations labour is **prolonged** due to slow cervical dilatation
- Precipitate labour: occurs when duration labour does not exceed 4 hours.

Cervical ripening:

In late pregnancy, and during prodroma of labour, the cervix becomes softer and thinner due to edema and decreased collagen caused by PGL. The cervix becomes ready for effacement & dilatation.

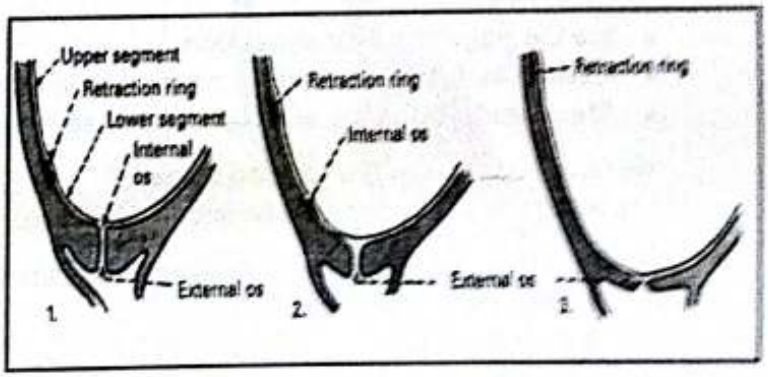


Fig 20:2 cervical effacement and dilatation in a primigravida

Forces Causing Cervical Effacement and Dilatation:

1. Uterine contractions and retractions.
2. Pressure by the bag of waters.
3. Pressure by the presenting part

Patterns of cervical effacement:

- *In primigravidas:* Effacement occurs first followed by dilatation of the external os. Effacement is shortening and incorporation of the cervix into the lower segment (LUS), so that the genital tract is transformed into one canal.
- *In multipara*: Effacement and dilatation of the cervix occur simultaneously.

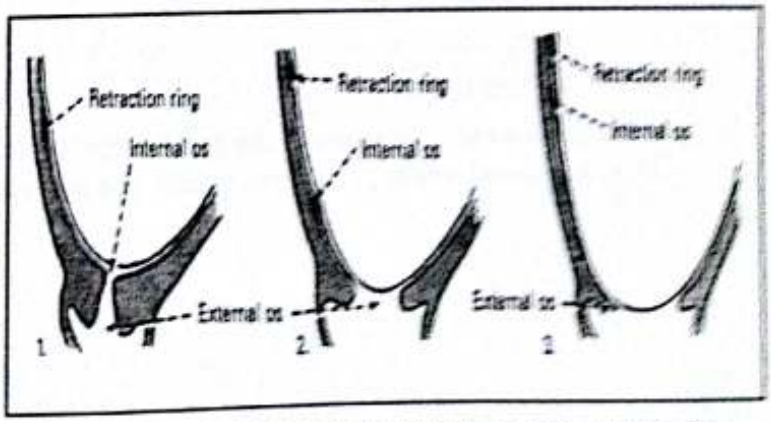


Fig 20:3 cervical effacement and dilatation in a multigravida

Phases of cervical dilatation "Friedman curve":

A) Latent phase

Initial slow phase
Dilatation from 0 to 3 cm in 8 hours

B) Active phase

Rapid phase
Dilatation from 3 to 10 cm in 6 hours

- *Acceleration phase*
- *Maximum slope*
- *Deceleration phase*

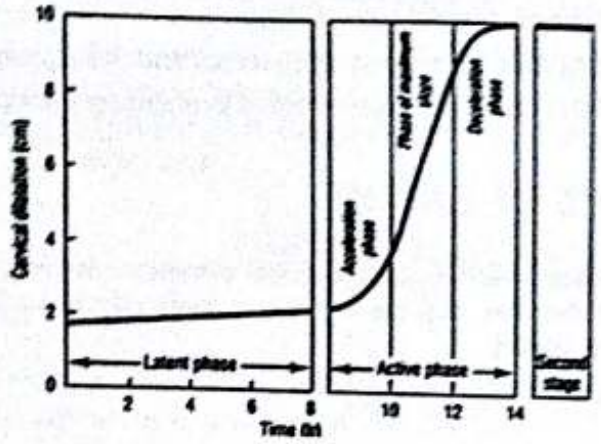


Fig 20:4 The average dilatation curve for primigravidas in labour A relatively flat latent phase & a rapidly progressive active phase, which includes 3 component:- acceleration phase, phase of maximum slope and a deceleration phase.

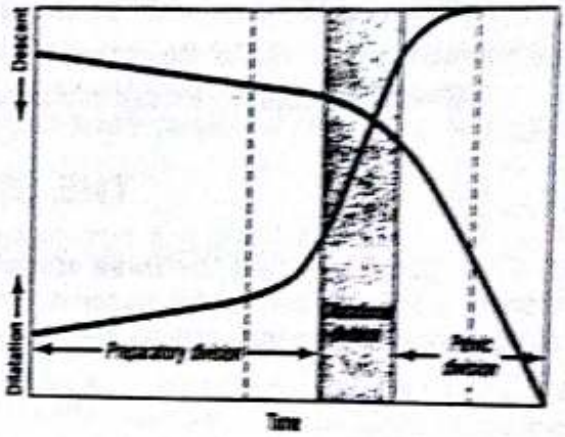


Fig 20;5 Labour course is divided on basis of cervical dilatation & head descent curves into (1) preparatory division, (2) dilatational division and (3) pelvic division including both deceleration phase & second stage.

THE SECOND STAGE OF LABOR

- It is the stage of **Foetal expulsion**
- It **starts** by full dilatation of the cervix. And **ends** by complete expulsion of the fetus.
- The average **duration:** is around **1-2 hours** in a primigravida, and **1/2 an hour** in a multipara.

Mechanism of labour in the 2nd stage:

It depends on the fetal position and presentation.

A) *In Cephalic Occipito Anterior Positions* (discussed with normal labor).

Descent of foetal head
Engagement
Increased Flexion
Internal Rotation
Restitution
External rotation

B) *In Malpositions* (right and left occipito posterior positions), discussed later in a separate chapter

C) *In Malpresentations* (face, brow, breech, and transverse lie), discussed separately with each disorder.

THE THIRD STAGE OF LABOUR

- It is the stage of **expulsion of the placenta** and membranes.
- It **starts** by complete delivery of fetus and **ends** by expulsion of the placenta and membranes.
- The average **duration** is 10-30 minutes in both primi-and multipara.

Mechanism of separation of the placenta

After fetal delivery the uterus becomes smaller and retraction continues, so the placental site diminishes, meanwhile the inelastic placenta is unable to shrink.

a. Schultze mechanism (80%)

- Placenta separation starts at the center.
- The placenta is delivered as an inverted umbrella with its fetal surface first.
- It is associated with less blood loss and less retained fragments.

b. Duncan mechanism (20%)

- Placental separation starts at the lower edge.
- The placenta is delivered sideways.
- It is associated with more blood loss and more retained parts.

Fig 20:5 Schultze mechanism

Fig 20:7 Duncan mechanism

21 NORMAL LABOUR

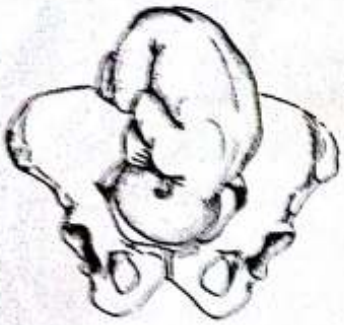
Cephalic

Occipito Anterior positions

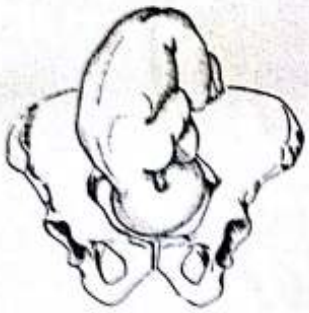
- *Definitions & Positions*
- *Mechanism of Delivery*
  - *Delivery of the head*
  - *Delivery of the shoulders & body*
- *Management of normal labour*
  - *Initial assessment*
  - *The 1st stage*
  - *The 2nd stage*
  - *The 3rd stage*
  - *The 4th stage*
- *Management of the new born*

DEFINITIONS

- Labour: is the term that describes the process of delivery of the fetus from the uterus
- Normal Labour: describes the process of delivery in which spontaneous labour pains result in vaginal delivery of a single living full term foetus, presenting in a vertex cephalic presentation, without maternal or foetal complications.
- Normal labour is therefore a retrospective diagnosis that can be only made after labour is completed.
- Incidence: the majority of labours are normal, hover the true incidence is difficult to estimate
- Duration: the average duration of normal labour is; **14-18** hours in the primigravida, and **6-10** hours in the multigravida
- Positions in normal labour:
  1. Left and Right occipitoanterior (LOA and ROA).
  2. Left and Right occipitotransverse (LOT and ROT).



LOA



ROA

MECHANISM OF DELIVERY IN NORMAL LABOUR

A. DELIVERY OF THE HEAD

Delivery of the head includes; descent, engagement, increased flexion, internal rotation, extension, restitution, and external rotation

1. **Descent:** A continuous movement throughout labour due to:
   - Uterine contractions & retractions.
   - Auxiliary force in the 2nd stage of labour.
   - Straightening of the fetus caused by contraction & retraction of the uterus.
2. **Engagement:**
   - It is passage of the widest transverse diameter of the presenting part through the plane of the pelvic inlet.
   - In cephalic presentation engagement it is the passage of the BPD through the plane of the pelvic brim.

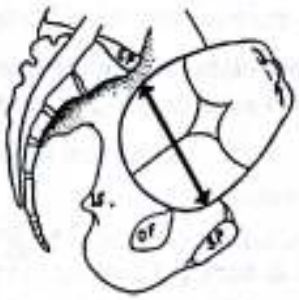


Fig 21;2 Non engaged head

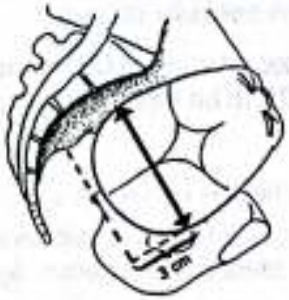


Fig 21:3 Engaged head

3. Increased flexion:

- When the head meets resistance during its descent, the force applied on the sinciput is greater than that on the occiput, leading to increased flexion.
- It is explained by the 2 armed lever theory i.e. the head is represented by two armed levers of unequal lengths.
  - A short arm: extends from the occiput to the atlanto-occipital joint.
  - A long arm: extends from the sinciput to the atlanto-occipital joint.
- Results of increased flexion:
  - The head enters the pelvis with the smallest suboccipito-bregmatic diameter (9.5 cm).
  - The occiput meets the pelvic floor first preparatory to internal rotation.
  - The part of the head occupying the plane of the greatest dimensions is like a circle, as the BPD and suboccipito-bregmatic diameters are both equal (9.5 cm). This will facilitate internal rotation of the head.

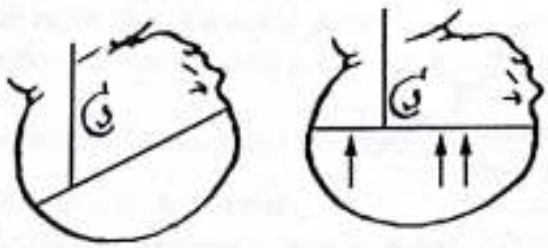
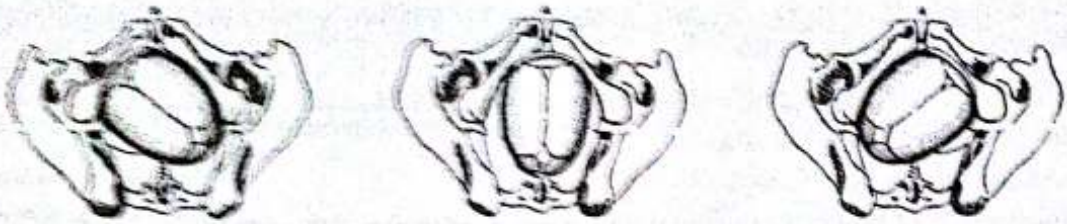


Fig 21:4 flexion of foetal head

4. Internal rotation:

- This means anterior rotation of the occiput 1/8th of a circle (45°) as it meets the pelvic floor first.
- It occurs at the level of the plane of the greatest pelvic dimensions



ROA DOA LOA

- Internal rotation is explained by:
  - Direction of the forward sloping gutter of the levator ani muscles. The direction of the gutter is downwards forwards and medially.
  - Rifling action of the pelvis: The largest available diameter at the inlet is the oblique, while at the outlet is the antero-posterior diameter.
- N.B.: In occipito posterior positions 90% of cases will rotate 3/8th of a circle anteriorly to bring the occiput behind the SP to be delivered as in occipito-anterior (long internal rotation).

5. Extension:

- The suboccipital region hinges under the symphysis pubis.
- The head is acted upon by 2 forces at this level in the pelvis: **downward & forward** force of the uterine contractions, **upward & forward** force of the pelvic floor.
- The net result is passage of the head forward i.e. extension.

6. Restitution:

The occiput rotates 1/8th of a circle in an opposite direction to internal rotation, to undo the twist of the neck caused by internal rotation.

7. External rotation:

Rotation of the occiput 1/8th of a circle in same direction as restitution. It is due to internal rotation of the anterior shoulder, 1/8th of a circle from the oblique to the anteroposterior to the posterior diameter

8. DELIVERY OF THE SHOULDERS AND BODY

- The anterior shoulder hinges below the SP.
- The posterior shoulder is delivered first by lateral flexion of the spine.
- The anterior shoulder then follows, then the rest of the body.

Fig 21:8 delivery of the shoulder

Fig 21:7 ext. rotation

MANAGEMENT OF NORMAL LABOUR

INITIAL ASSESSMENT:

On arrival to the hospital the parturient is subjected to the following:

1. Reviewing the antenatal records if possible
2. Full history and examination

- **History:**
  - Onset of labour pains, their frequency, intensity, and duration.
  - Presence of show
  - Escape of liquor amnii
  - Amount and colour of liquor amnii if ROM
  - Pattern of foetal movements

- **General Examination:**
  - Pulse, Blood pressure, and Temperature
  - Degree of anxiety
  - Degree of dehydration
  - Observation of height and weight.

- **Abdominal examination**
  - For determination of the frequency, duration and intensity of uterine contraction
  - To determine the lie, presentation and position of the presenting part.
  - Engagement of the presenting part
  - F.H.S. (site, rate, rhythm): The foetal heart sounds should be checked especially at the end of a contraction and immediately thereafter, to identify pathological slowing of the heart rate.

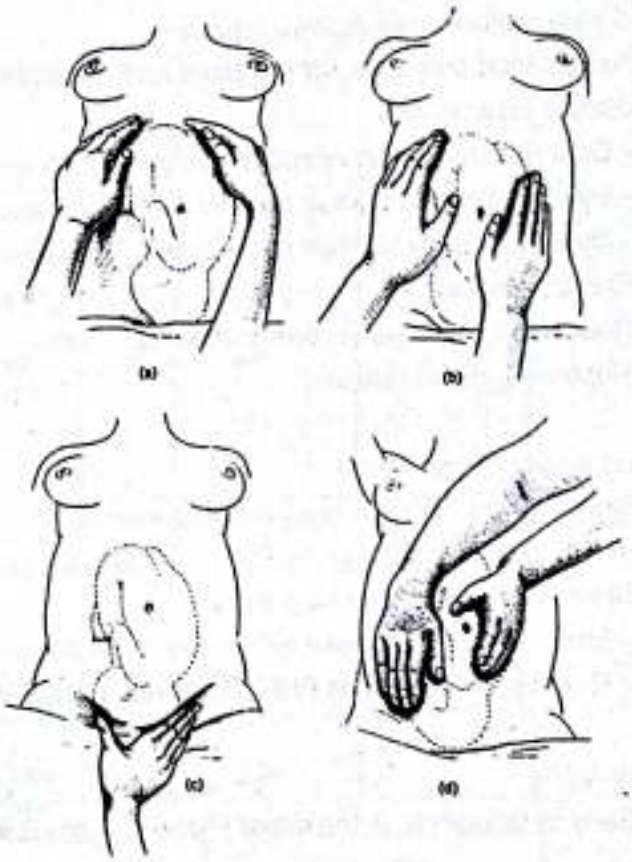


Fig 21:9 Leuopold maneuvers

- **Vaginal examination:**
  - To exclude contracted pelvis.
  - To assess dilatation and effacement of the cervix.
  - The foetal presenting part (position, station and degree of flexion)
  - Condition of the membranes and if ruptured; presence or absence of meconium.
  - To exclude cord prolapse.
  - To determine **station of the head:** When the lowest part of the fetal head is felt at the level of the ischial spines, this is called **zero** station. **Station +1, +2 & +3**, means that the lowest part of the head is 1,2 or 3 cm lower than the ischial spines. **Station -1, -2 & -3**, means the lowest part of the head is 1,2 or 3 cm higher than the ischial spines.
  - **Frequency of vaginal examination:** depends on the obstetrician, but at least it is done twice;
    1. At the start of labour.
    2. If rupture of membranes occurs to exclude cord prolapse.

- **Electronic Foetal Heart Rate Monitoring FHRM:**

  Electronic FHRM is performed on admission, then repeated at intervals during and in between uterine contractions to reassure normal foetal well being throughout labour.

MANAGEMENT OF THE 1ST STAGE OF LABOUR

1. Preparation of the patient
   - Antisepsis: The vulva is shaved & cleaned with an antiseptic.
   - Evacuation of the bladder & rectum:
     - This is done to prevent reflex uterine inertia.
     - The bladder is evacuated by frequent micturition or by a catheter.
     - The rectum is evacuated by an enema, which also prevents contamination.

2. Maternal observation during labour:
   - Pulse, blood pressure, temperature and respiratory rate
   - Uterine contractions:
     - Contractions are observed for frequency, strength and duration
     - By the palm of the hand applied on the abdomen.
     - By a toco-dynamometer i.e. a device applied on the abdomen.
   - Cervical dilatation.
   - Descent of the fetus i.e. pelvic station.
   - Rupture of membranes.

3. Foetal Heart Rate (FHR):
   - Normally the FHS are regular with a rate of 120-160 beats / minute.
   - The aim of auscultating the FHS is to detect fetal distress e.g. bradycardia.
   - Methods of detection of the FHS:
     - Intermittent by the sonicaid or Pinard stethoscope every 30 minutes.
     - Continuous electronic FHR monitoring is indicated in high-risk cases.

3. Nutrition:
   - Early in labour i.e. in the latent phase, sugary fluids are given.
   - In the active phase, oral feeding is avoided, as delayed gastric emptying may lead to vomiting & aspiration if general anesthesia is needed at any time "Mendelson syndrome"
   - If labour is prolonged more than 8 hours, IV fluids as glucose 5% and saline are given.

4. Pain relief:
   - Pethidine 50 mg IM is commonly used.

     Pethidine causes fetal respiratory depression & should be stopped 2 hours before the 2nd stage of labour, to avoid fetal respiratory depression at birth.
   - Epidural analgesia is an alternative.

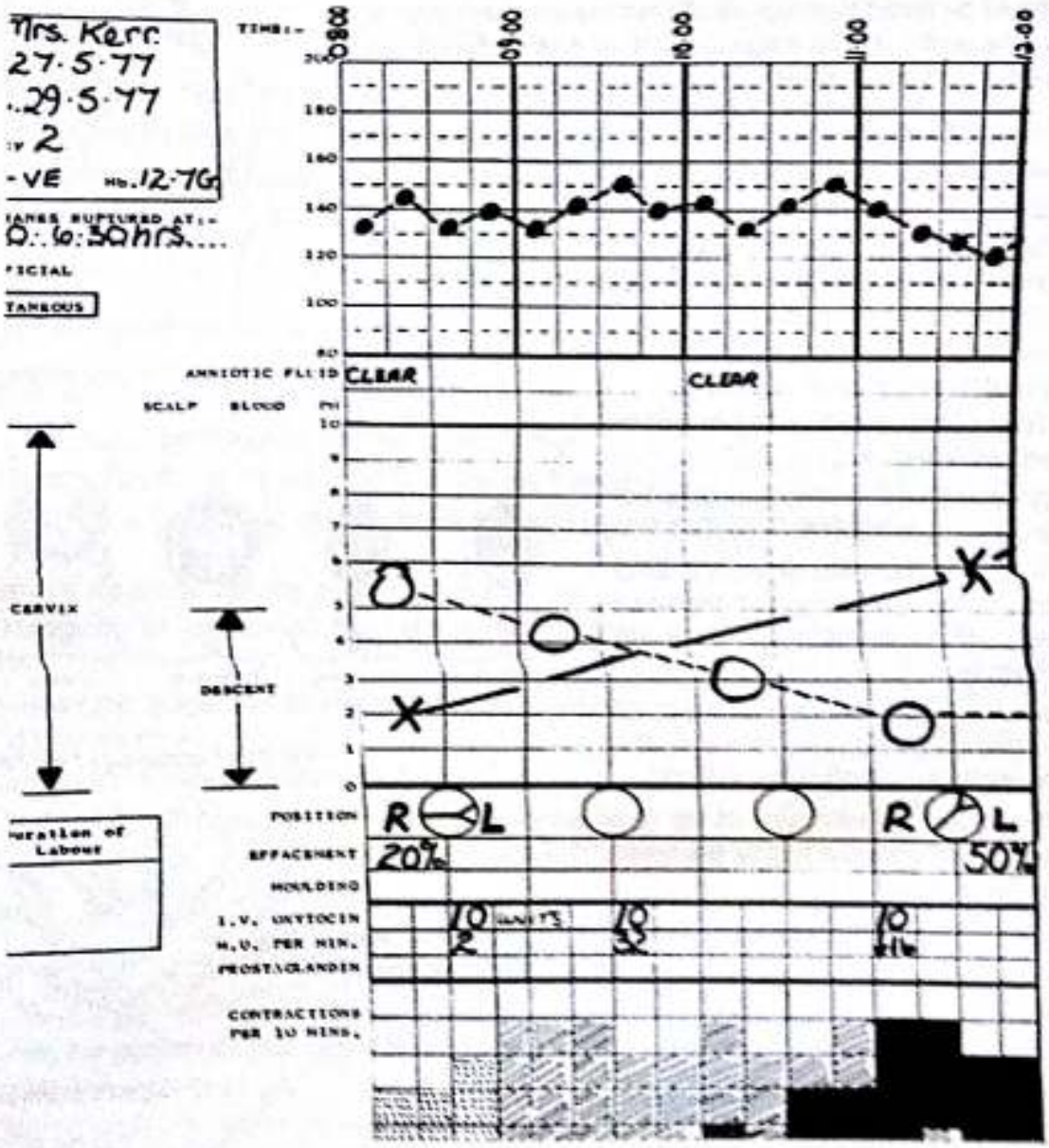
5. Instructions:
   - If the membranes are ruptured: Rest in bed in the lateral position.
   - If the membranes are intact:
   - Walking is allowed in between uterine contractions.
   - Straining (bearing down) should be avoided because:
     - It is useless & exhausts the patient.
     - It predisposes to genital prolapse.

THE PARTOGRAM

It is a graphic record of labour which allows an instant visual assessment of the rate of cervical dilatation against an expected norm according to parity of the women so that active management can be instituted immediately.

Other observations can be recorded on the chart as; the frequency and strength of contractions the descent of the head, timing of rupture membranes, medications given and the basic observations as the blood pressure, pulse rate and temperature.

The Partogram



Items of the Partogram:

1. Vital signs; P, BP, RR
2. Cervical dilatation
3. Descent of the Head (station)
4. Cardiotocograph

MANAGEMENT OF THE 2ND STAGE OF LABOUR

Identification of the 2nd stage:

1. Full dilatation of the cervix (10 cm).
2. Reflex desire to bear down.
3. Rupture of membranes:
   - During the 1st stage of labour, the amniotic sac is divided by contact of the head and cervix into a bag of hind-waters, and a bag of fore-waters.
   - After full cervical dilatation, the hind and fore-waters become continuous leading to increased pressure in the fore-waters and rupture of membranes.
   - It should be noted that rupture of membranes may occur at any time during the 1st stage (SROM) or even before onset of labour (prelabour ROM).

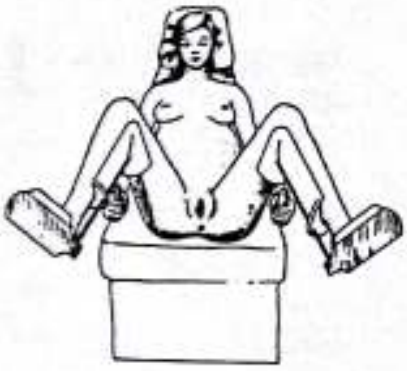


Fig 21:10 lithotomy position

CONDUCT OF LABOUR:

- Delivery is carried out in the delivery room or theater.
- Position: patient is usually put in the Lithotomy position.
- Sterilization: the vulva and perineum are washed by an antiseptic from before backward, and the abdomen, thighs, and legs are covered by sterile towels.
- Instructions: The patient is instructed to strain during contractions and to relax in between.
- Delivery of the head: with prevention of perineal tears:
  - Head is left until crowning occurs before allowing extension.
  - Crowning is identified when the BPD distends the vulval ring and the head does not recede in between contractions.
  - After crowning, extension of the head will distend the vulva by the suboccipito-frontal diameter (10 cm).
  - Before crowning, extension of the head will over-distend the vulva by the occipito- frontal diameter (11.5 cm) with liability to perineal tears.

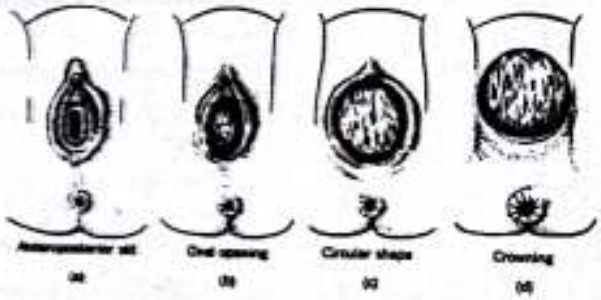


Fig 21:11 crowning of the head

- Perineal support: is done by a sterile dressing when the head appears at the vulva, to prevent extension of the head before crowning.
- Delivery of the head should be:
  - Slow, in between contractions, and without bearing down.
  - Ritgen maneuver: which is controlled extension of the head.

Fig 21:12 Ritgen's maneuver

- Episiotomy:
  - This is an incision made in the perineum at maximum distension of the vulva by the foetal head (crowning).
  - Its aim is to avoid extensive perineal and vaginal tears
  - Indications include: rigid perineum as elderly primigravida, or a large head diameter, as in: malpositions (occipito-posterior and face to pubis), and malpresentations (as face and breech delivery). *See details later in Obstetric Operations.*

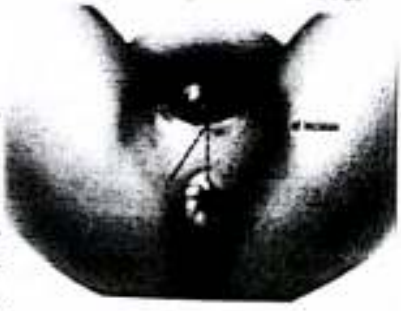


Fig 21:13 episiotomy

- Delivery of the shoulders and body:
  - Gentle downward traction on the head till the anterior shoulder appears under the SP.
  - The head is then lifted upward to deliver the posterior shoulder first.
  - The head is then depressed downwards to deliver the anterior shoulder.

- Clearance of foetal air passages by suction or aspiration is started immediately after delivery of the foetus. It may be even started before delivery of the shoulders and rest of the body.

- Dealing with loops of cord around the foetal neck if present:
  - One loop is slipped.
  - Several loops are doubly clamped and the cord is cut in between.

- The umbilical cord is cut between 2 clamps

- The baby is held from the ankles, and handled to the neonatologist for;
  - Complete clearance of the airways to ensure proper respiration
  - Foetal assessment by the APGAR score at one and five minutes after delivery.

MANAGEMENT OF THE 3rd STAGE OF LABOUR

- Duration: 5-10 minutes, if more than 30 minutes it is considered a prolonged 3rd stage.

A. CONSERVATIVE METHOD:

1. Exclusion of bleeding and uterine atony:
   - The ulnar border of the left hand is put on the fundus
   - A rise of the fundal level of a lax uterus points to bleeding inside the uterus.

2. Signs of separation of the placenta are awaited:
   - The body of the uterus becomes smaller, harder and globular.
   - Suprapubic bulge due to presence of the placenta in the lower uterine segment
   - Elongation of the cord without receding.
   - Gush of blood from the vagina due to expulsion of the retroplacental clot.

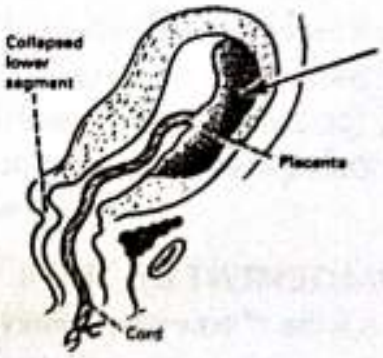


Fig 21:14 separation of placenta

3. Uterine massage:
   Allows contraction of the uterus and controls bleeding.

4. Placental expulsion:
   - Ask the patient to bear down or perform a gentle fundal pressure.
   - Fundal pressure should be very gentle if the uterus is lax, to avoid inversion of the uterus.

5. Uterine stimulants:
   May be used to prevent atonic postpartum hemorrhage (Ergometrine 0.25 mg IM or Oxytocin 5 units IV drip).

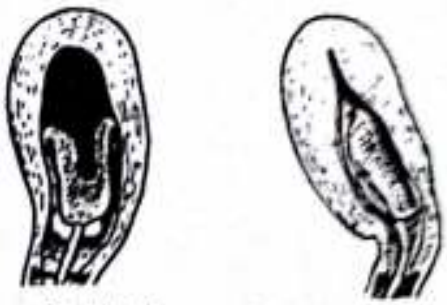


Fig 21:14 separation of placenta

N.B.: Disadvantages of the conservative method:
- Takes longer time
- Risk of postpartum hemorrhage is 5%

B. ACTIVE METHOD "MODERN MANAGEMENT":

1. Uterine stimulants:

- Oxytocin IV infusion drip is added if not already used
- With delivery of the anterior shoulder, ergometrine 0.25-0.5 mg IV is given, to produce strong uterine contractions & thus rapid placental separation.

2. Brandt-Andrews method "Controlled cord traction":

- The left hand is put suprapubic & when the uterus contracts, the uterus is pushed upwards. The other hand exerts gentle traction on the cord.
- Disadvantages of the active method:
  - Rupture of the cord.
  - Acute inversion of the uterus if done on a lax uterus.
  - *Thus, cord traction is avoided if the uterus is lax, to avoid inversion of the uterus .*
- Advantages of the active method:
  - Less duration & less blood loss.
  - Significant reduction in postpartum hemorrhage

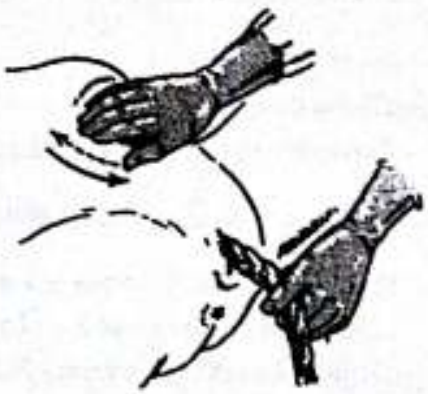


Fig 21:15 controlled cord traction

After placental delivery:

- The placenta is rolled by both hands, to make the membranes like a rope, to avoid missing part of the membranes.
- The placenta is inspected to avoid missing parts.
- Repair of perineal tears, if more than 1 cm or if bleeding.
- The vulva is washed with an antiseptic and covered by a sterile dressing.

Blood loss in the 3rd stage of Labour:

- 200-300 ml from the placental site.
- 100-200 ml from the episiotomy or perineal lacerations.
- During cesarean section, blood loss from the placental site is up to 600 ml.

MANAGEMENT OF THE 4TH STAGE OF LABOUR

- It is the 1st hour after delivery in which postpartum hemorrhage is liable.
- Careful observation to detect postpartum hemorrhage.
- Uterine massage is done every 15 minutes.

MANAGEMENT OF THE NEWBORN

1. Warmth: On a special heated unit with a thermal regulation.
2. Care of respiration:
   - The newborn is placed supine with head lowered & turned to one side.
   - Suction, of the mouth & nose by a catheter connected to a suction pump.
   - If respiration is delayed, respiration is stimulated by slapping the sole or back.
   - Apgar score is done for evaluation of the newborn.
3. Care of the umbilical cord stump:
   - Asepsis: To avoid neonatal tetanus or infections
   - It is ligated by 2 silk ligatures or plastic clamps 4 and 5 cm from the umbilicus.
   - The cord is cut distal to the 2nd ligature to avoid tying an umbilical hernia.
4. Care of the eyes: Penicillin or tetracycline drops are used to prevent infection of the eyes.
5. The weight is recorded.
6. Identification, by an identification band or footprint of the newborn.
7. Detection of congenital anomalies: e.g.; Hypospadius, imperforate anus, hare lip, etc...
8. Vitamin K administration:To prevent hemorrhagic disease of the newborn.
9. Physiological jaundice, may develop after 24 hours.

EFFECTS OF VAGINAL DELIVERY ON THE FETUS:

1. Moulding:
   - Moulding is overlap of the bones of the skull vault, due to compression.
   - Slight moulding, helps easy passage of the head through the pelvis.
   - Marked moulding may cause intracanial hemorrhage.
   - Prolonged head compression may cause foetal bradycardia (vagal stimulation)
2. Caput Succedaneum: see fetal birth injuries.

22 MALPOSITIONS CEPHALIC

Occipito Posterior Positions

Definition & Incidence
Positions
Aetiology
Course of labour
Mechanism of delivery
- *Normal mechanism (90%)*
- *Abnormal mechanism (10%)*

Diagnosis during pregnancy
Diagnosis during labour
Complications
Management
- *1st stage*
- *2nd stage*

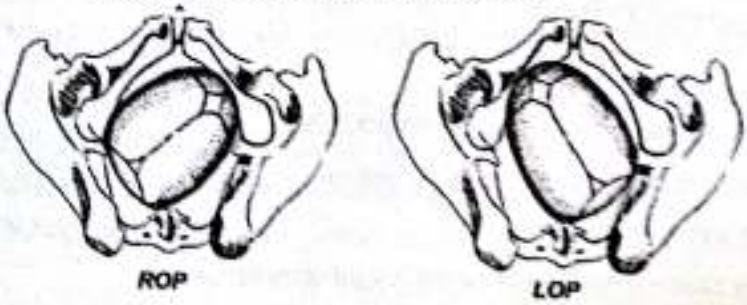
DEFINITION

Occipito posterior (OP) is a cephalic vertex presentation in which the fetal back is directed posteriorly. It is a malposition and not a malpresentation.

INCIDENCE 25% early in labour

POSITIONS (ROP & LOP)

Right OP is commoner than the left OP as dextro-rotation of the uterus favours right OP if the back is on right side.



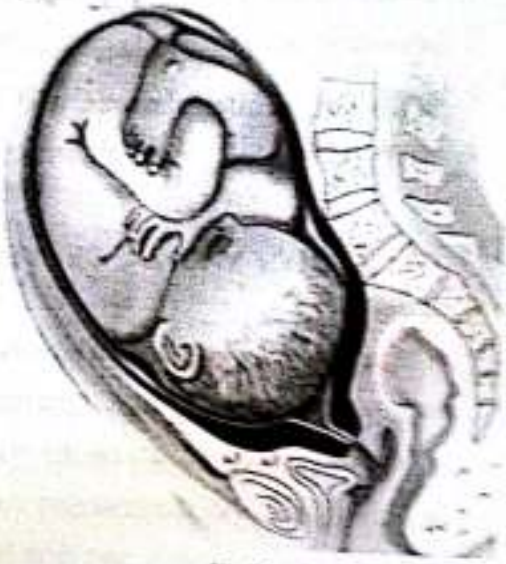


Fig 22.1 ROP position

AETIOLOGY

1. Shape of the pelvis: Narrow fore-pelvis and wider hind-pelvis (the commonest cause) as in android, anthropoid and high assimilation pelvis.
2. Maternal lumbar Kyphosis
3. Anterior insertion of the placenta
4. Other Causes: Twins, pendulous abdomen.

COURSE OF LABOUR

1. Delayed engagement as there is a degree of deflexion
2. Prolonged labour (long head rotation)
3. Uterine inertia (commonly associated with dysfunctional labour)
4. Early spontaneous ROM (lack of fitting of the head on the cervix within the pelvis)

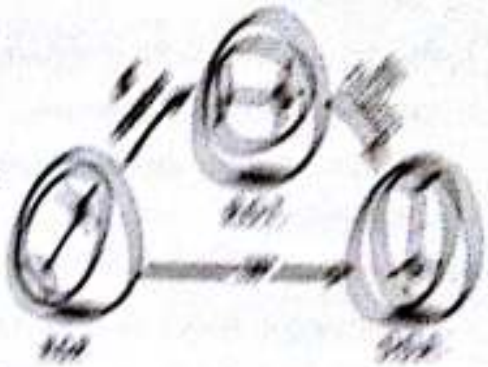
Causes of foetal head Deflexion:

Deflexion is favoured by early descent of the occiput and delayed descent of the sinciput. This occurs as the BPD (9.5 cm) enters the pelvis in the oblique diameter (12 cm), while the bitemporal (8 cm) enters in the wider oblique diameter of the pelvis (13 cm).

MECHANISM OF LABOUR IN OP

I. NORMAL MECHANISM: (90%) Long anterior rotation (OPA)

- Correction of head deflexion into complete flexion occurs as the occiput reaches the pelvic floor first, and rotates anteriorly 3/8th of a circle (long anterior rotation).
- The head is delivered by **extension** as in normal labour.



II. ABNORMAL MECHANISM (10%)

Failure of correction of head deflexion

A. Posterio rotation to direct OP (9%): Face to pubis

- Marked deflexion of the head: causes the occiput to reach the pelvic floor first and rotates anteriorly 1/8th of a circle.
- This means that the **occiput rotates posteriorly** 1/8th of a circle to a DOP position (face to pubis), and the head is delivered in **flexion**.
- Perineal tears are liable to occur because:
  - The vulva is overdistended by occipitofrontal diameter (11.5 cm).
  - The perineum is distended by the baby occiput.

B. Persistent oblique occipito posterior (3%): POP

- **Moderate deflexion of the head**: causes the occiput and sinciput to reach the pelvic floor at the same time so **no rotation occurs**.
- Spontaneous delivery can't occur and labour is prolonged.

C. Deep transverse arrest of the occiput (1%): DTA

- **Mild deflexion of the head**: causes the occiput to reach the pelvic floor first, and rotates anteriorly 1/8th of a circle then **rotation is arrested**.
- Spontaneous delivery can't occur and labour is prolonged.

N.B.: *Shape of the pelvis affects the anteriorly of OP and the size of the pelvis affects the long anterior rotation of the occiput.*

Factors favouring long anterior rotation	Factors interfering with long anterior rotation
Correction of deflexion	Persistent deflexion
Strong uterine contractions	Uterine inertia
Adequate pelvis	Contracted pelvis
Good pelvic floor	Lax or rigid pelvic floor
Intact membranes	Rupture of membranes
	Epidural anaesthesia

DIAGNOSIS OF OCCIPITO-POSTERIOR POSITION

A) During Pregnancy:

I. Abdominal examination:

a. Inspection:

- Flat abdomen below the umbilicus.
- Subumbilical transverse groove.
- Foetal movements near the middle line.

b. Palpation:

- Fundal grip: breech.
- Umbilical grip: back is away from the middle line. Limbs are near the midline
- 1st pelvic grips: head usually smaller and not engaged.
- 2nd pelvic grip: head is usually deflexed.

c. Auscultation:

- FHS are usually heard away from the middle line below the umbilicus.

II. Ultrasound Diagnosis

- Confirm the diagnosis.
- Other routine values (Foetal weight, foetal well being, placental localization, amniotic fluid volume, etc...).

B) During Lbour:

In addition to the above, vaginal examination is done to show:

- Cephalic presentation with the occiput and lambda posterior
- Degree of deflexion is detected
- Presence of caput succedaneum, or moulding of cranial bones.
- Degree of cervical dilatation.
- Rupture of membranes and cord prolapse.
- Exclusion of cephalopelvic disproportion (CPD) and contracted pelvis.

COMPLICATIONS OF LABOUR IN OP POSITIONS

Maternal	Foetal
Prolonged and / or obstructed labour	*Asphyxia*
Birth canal injuries	*Foetal injuries*
Postpartum haemorrhage (atonic and traumatic)	
Puerperal sepsis (prolonged ROM)	

MANAGEMENT OF OP POSITION

A) Initial Assessment:

- Pelvic adequacy should be carefully assessed early in labour to exclude contracted pelvis and CPD. In many cases OP are more associated with android type of pelvis.
- Ultrasonography may be helpful in evaluation of expected foetal weight to exclude an oversized foetus, more liable to CPD. It may also confirm OP postion, and evaluate amount of liquor amnii, to plan for a safe delivery

B) Trial labour:

1st stage of labour:

- Cases of OP are more liable to prolonged labour, PROM and abnormal uterine action. Care should be given to avoid maternal exhaustion (light diet, fluids, glucose 5%, sedation, etc..)
- Continuous FHR monitoring; may help early detection of foetal distress

2nd stage of labour: (after excluding contracted pelvis)

- ***In 90% of cases:*** correction of deflexion occurs with long anterior rotation of the occiput, ending in a in a normal delivery within 1 hour in the 2nd stage.
- ***In 6% of cases:*** short posterior rotation occurs into a DOP (face to pubis), with spontaneous delivery that may be aided by a generous episiotomy or rarely forceps application.
- ***In cases of POP or DTA (4%):*** Labour will be obstructed as there is no mechanism of delivery in such cases. A hidden CPD should be searched for before a decision is made;
  1. **C.S.:** offers the best and safest line of management for both the mother and the foetus
  2. **Instrumental delivery:** are possible alternatives, in selected cases, with an experienced operation, especially in cases where facilities for a CS are not readily available, foetal distress is detected with no availability of transfer of the patient to another centre. Instrumental delivery can be accomplished by either
     - A. The vaccum extractor (ventouse) as a first choice
     - B. Rotation and extraction by Kiell and forceps

Indications of C.S in OP include

- POP and DTA
- Contracted pelvis or marked CPD due to oversized foetus
- Foetal distress during trial vaginal delivery
- Other indications as; Elderly primigravida, placenta praevia, previous CS, IUGR, etc....).

23 MALPRESENTATIONS CEPHALIC
Face and Brow Presentations

Face Presentation
Definitions,
Incidence, & Aetiology
Mechanism of labour
- *Mento-anterior positions*
- *Mento-posterior positions*

Diagnosis
Management
Complications
Brow presentation
Definition , incidence & aetiology
Diagnosis & management

FACE PRESENTATION

- **Face** is a longitudinal lie, cephalic presentation, in which the head is **fully extended**.
- The face presents at the pelvic brim, while the occiput lies in direct contact with the back.
- **The face** is that part of the foetal head that lies between the root of the nose and the chin.
- The **denominator** in face presentation is the chin or mentum (M).
- There are **four** positions:
  - Left mento anterior (LMA), right mento anterior (RMA)
  - Left mento posterior (LMP), right mento posterior (RMP).
- **Mento anterior (MA) positions** are more common than mento posterior (MP), because most of face presentations develop secondary to OP positions with increased extension of the foetal head especially if associated with a flat pelvis.
- **Right OP** usually turns to **LMA**, which makes it the commonest position in face persentation

INCIDENCE: 1/500 deliveries

AETIOLOGY:

Any factor that favours extension of the foetal head, or prevents its flexion.

A. Primary face:

It is rare, being encountered mainly during pregnancy rater than labour

- Mostly idiopathic (most common).
- Excessive tone of the extensor muscles of the neck (rare).
- Tumours of foetal neck such as foetal goitre (rare).
- Anencephaly due to short neck and extended head.

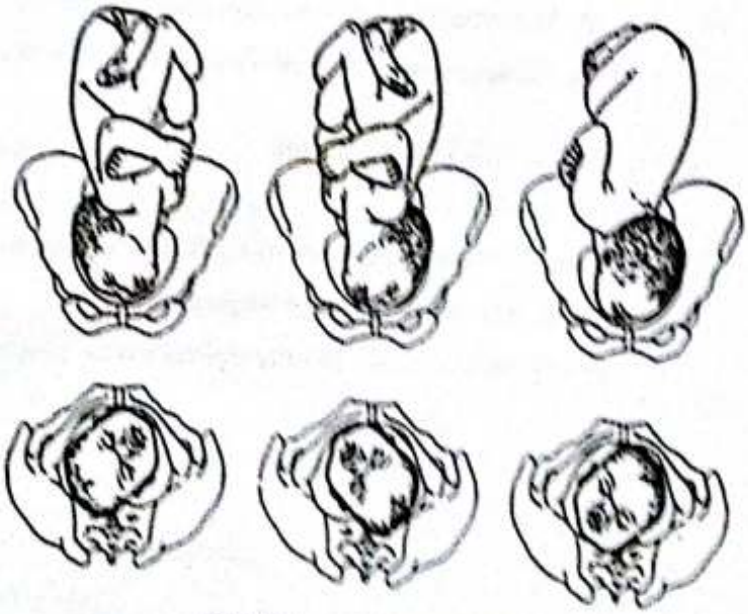


Fig 23.1 positions in Face Presentation
LMA - RMA - RMP

B. Secondary face:

It is the more common form of face presentation encountered during labour

- Contracted pelvis especially inlet in a primigravida.
- Pendulous abdomen in multipara (weak uterine & abdominal muscles).
- Placenta praevia (prevents head flexion at inlet)
- Other causes of malpresentations (prematurity, twins, polyhydramnios)

MECHANISM OF LABOUR:

In absence of contracted pelvis or marked CPD, and with effective uterine contractions, successful vaginal delivery is usually achieved.

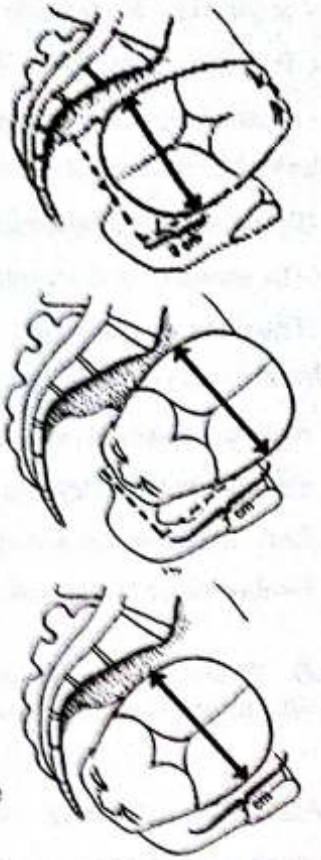


Fig 23:2 engagement in face presentation

A. Mento Anterior Positions (MA):

1. Descent (delayed).
2. Engagement by submento-bregmatic diameter 9.5 cm (delayed).
3. Increased **extension**.
4. Anterior rotation of head $^1/_8$ circle, to bring the chin below the S.P.
5. Delivery of head by **flexion**. Submento-vertical D. (11.5 cm) distends the vulva.
6. Restitution (correction of internal rotation in opposite direction).
7. External rotation (transmitted to the head during shoulder rotation).

Causes of prolonged Labour face presentation:

a. ***Delayed cervical dilatation:*** due to dysfunctional labour.

b. ***Delayed descent and engagement*** of the head:

- Absent moulding of facial bones.
- At the time when BPD is crossing the plane of the pelvic brim, the facial bones are slowly advancing through pelvic floor muscles.

ENGAGEMENT in face: occurs when BPD crosses the plane of the pelvic inlet, at which time the chin is at the perineum partially distending the vulva.

B. MENTO POSTERIOR POSITIONS (MP):

1. **Successful vaginal delivery in 2/3 cases:** Achieved with long internal rotation of the head 3/8 circle anteriorly to bring the chin below the S. pubis where the head is delivered in flexion.
2. **Obstructed labour in 1/3 cases:** Occurs with
   - *Failure of long anterior rotation:* transverse arrest (TA), or persistent, MP (PMP).
   - *Posterior rotation 1/8 circle:* leading to direct mento posterior position (DMP).

N.B.: In **DMP** the head is already maximally extended, and the foetal neck is facing the long sacrum rather than the short symphysis, hence labour is obstructed and vaginal delivery becomes impossible.

Fig 23:3 Long anterior rotation of the chin in face MP position

DIAGNOSIS OF FACE PRESENTATION:

The clinical diagnosis of face presentation rests on suspicion on abdominal examination confirmed only by vaginal examination aided by ultrasonography whenever available.

I. Abdominal Examination: Abdominal findings are poorly diagnostic

- Inspection: No reliable signs.
- Palpation: First Pelvic grip shows an unengaged head.
- Auscultation: No reliable data.

II. Vaginal Examination (during labour):

Distinctive facial landmarks on vaginal examination are:

- The mentum and alveolar margin
- The nose, malar bones, and supra-orbital ridges.

III. Ultrasound Diagnosis:

- Reveals maximal head extension in face presentations.
- Estimation of gestational age, foetal weight, and placental localization.
- Detection of foetal anomalies.
- Evaluation of foetal well being (BPP score & umbilical artery Doppler studies).

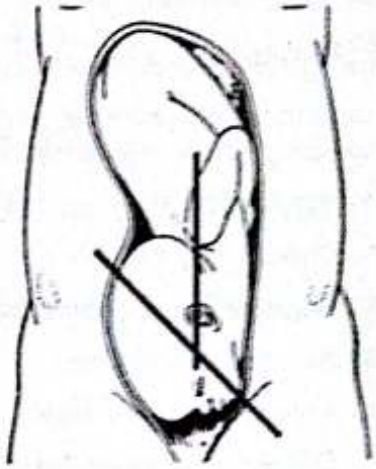


Fig 23:4 face MP

N.B.: Facial Oedema may occur with prolonged head compression in the pelvis. It may distort the facial features resulting in what it is known as (tumefaction).

MANAGEMENT OF FACE PRESENTATION

A) Management during pregnancy:

If suspected during ANC examination near term, efforts should be made to detect the underlying cause, to exclude foetal congenital anomalies, and other causes that call for an elective C.S.

B) Management during labour:

Labour is allowed to progress with the following prequisites

1. Exclusion of contracted pelvis and CPD (by vaginal examination and CPD tests)
2. Ensure effective uterine contractions
3. Close supervision of foetal well being through; continuous electronic FHR monitoring and regular partograms.

In cases with normal progress in the first stage, vaginal delivery may be anticipated.

- ***In Mento-anterior positions:***

Vaginal delivery should be anticipated whether:

1. Spontaneously with a generous episiotomy, or
2. Assisted with low forceps and a generous episiotomy

- ***In Mento-posterior positions:***

Watchful expectancy (Oxytocin in selected cases):

1. $^{2}/_{3}$ of cases will rotate anteriorly, to be delivered as FMA.
2. In the remaining $^{1}/_{3}$ of cases with Deep TA, Persistent MP or Direct MP, Caesarean Section will be the safest available option to avoid obstructed labour.

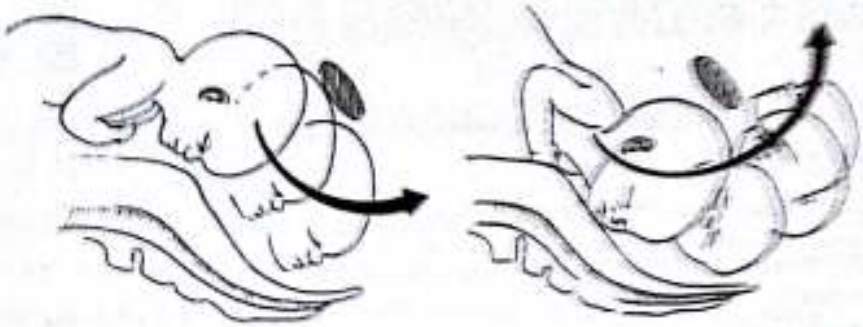


Fig 23:5 delivery in Face MP
Descent, engagement, extension, long internal rotation 3/8 circle anteriorly to MA, and finally delivery in flexion

Indications of CS in Face Presentation:

- MP positions with failed rotation after trial labour (DTA – PMP – DMP).
- Uterine scar as previous myomectomy or CS.
- Contracted pelvis and CPD (of any degrees).
- Other indications of CS as placenta praevia, pelvic tumours, foetal distress.

COMPLICATIONS ENCOUNTERED IN FACE DELIVERIES

A) Maternal:

- Prolonged labour leading to maternal exhaustion and distress.
- Obstetric injuries due to perineal and cervical lacerations & forceps delivery.
- Infection due to PROM and possible manipulations.

B) Foetal:

- Birth trauma and foetal birth injuries.
- Birth asphyxia from hypoxia associated with prolonged labour especially 2nd stage with prolonged use of Oxytocin.
- Congenital anomalies, commonly associated with the condition.

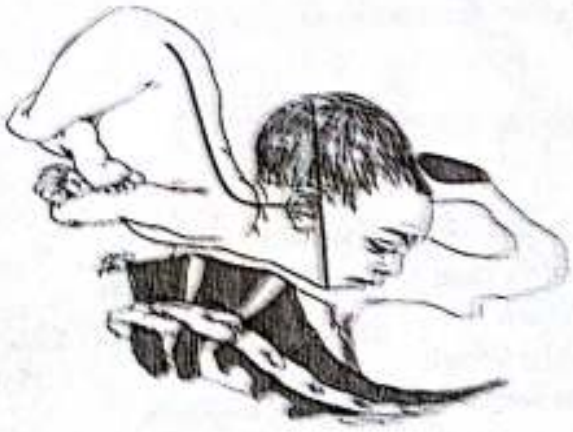


Fig 23:6 Face Presentation, direct mento-posterior DMT, vaginal delivery is impossible

BROW PRESENTATION

- **Brow presentation** is a longitudinal lie, cephalic presentation in which the head is midway between flexion and extension
- **The brow** is tha part of the foetal head that extends from the route of the nose to the anterior fontanelle.
- The capping diameter in brow is the is the mento-vertical diameter (13.5 cm). In the full term baby it is longer than any diameter of the inlet, hence engagement usually fails, and labour will be obstructed.

INCIDENCE: about 1/2000.

AETIOLOGY: The same as face.

TYPES OF BROW PRESENTATION:

- **Transient brow**: usually occurs during conversion of vertex occipito posterior into face presenattion
- **Persistent brow**: rare, and usually ends in obstructed labour.

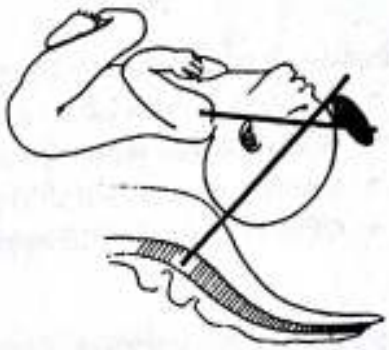


Fig 23:7 Brow presentation

DIAGNOSIS OF FACE:

I. Abdominal examination: poorly diagnostic and inconclusive

II. Vaginal examination (during labour with partially dilated cervix):

- Non-engaged, high presenting part.
- Distinctive landmarks include the frontal bones, the supra-orbital margin, the root of the nose and anterior fontanelle.

III. U.S. Diagnosis:

- To elicit incomplete head extension
- To exclude foetal congenital anomalies (as congenital goitre)
- To exclude multiple loops of cord around the neck
- To evaluate foetal well being, and confirm gestational age.

Fig 23:8 Brow posterior position

Fig 23:9 Brow anterior position

MANAGEMENT OF BROW PRESENTATION

A. Early in the 1st stage:
- Exclude contracted pelvis and CPD; if present, do C.S.
- Give appropriate time for the head to convert into face or vertex, and manage accordingly.

B. In the 2nd stage: (persistent brow):
- Caesarean section is the best and safest option available.

24 MALPRESENTATIONS
Breech Presentation

- **Definition:** Breech persentatioin is a longitudinal lie in which the **buttocks,** with or without the lower limbs, form the presenting part.
- **Incidence:**
  - **3-4%** of singleton term pregnancies (37 weeks or more).
  - More frequently encountered in **preterm deliveries** and in **multifoetal pregnancies.**

TYPES OF BREECH

1. **Complete breech:** Both hips and knees are flexed. The feet present beside the buttocks.
2. **Incomplete breech:** Partial extension is present either at the hipjoint, the knee, or both.
   - ***Frank breech:*** Only the buttocks are presenting (flexed hips and extended knees).
   - ***Footling presentation:*** Partial extension of the hip and knee on one or both sides.
   - ***Knee presentation:*** Extension of the hip and flexion of the knee.

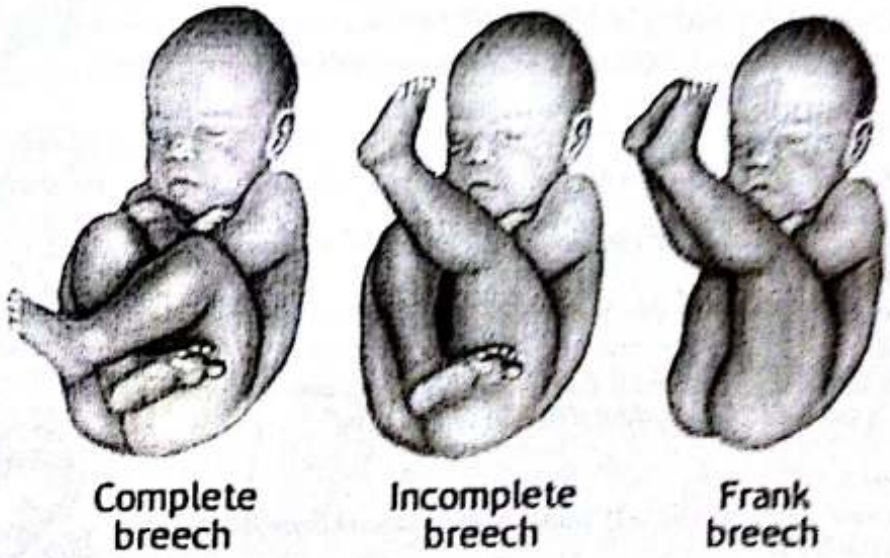


Fig 24:1 types of breech presentation

AETIOLOGY OF BREECH

Any factor that interferes with the adaptation of the foetus to the pyriform-shaped uterus may result in a breech presentation, although most cases are idiopathic.

1. Idiopathic: No identifiable cause in most cases of breech presentation.
2. Prematurity is the commonest cause due to:
   - Relatively excessive liquor (allows free mobility and rotation).
   - Relatively larger head (accommodates in the wider uterine fundus).
3. Failure of spontaneous cephalic version:
   - Breech with extended legs, as the legs act as a splint beside the head.
   - Twins and multifoetal pregnancies (vertex / breech).
   - IUFD (absent muscle tone and foetal kicking movements).
   - Oligohydramnics (inadequate liquor for spontaneous rotation).
   - Polyhydramnios (free mobility of the foetus resulting in unstable lie).
   - Uterine anomalies as bicornuate and septate uterus (prevent spontaneous cephalic version).
   - Uterine fibroid (if it encroaches on the pelvic cavity preventing engagement, and making th ehead better accomodates to th efundus)
4. Hydrocephalus (large head accommodates better to large uterine fundus)
5. Placenta previa (prevents descent of th ehead and favours its accomodation to the fundus of theuterus).

Fig 24:2 R.S.A

Fig 24:3 L.S.A

POSITIONS (Sacrum is the denominator)

1. **left sacro-anterior (L.S.A.)**: The sacrum is felt towards the left obturator foramen.
2. **R.S.A.**: The sacrum is felt towards the right obturator foramen.
3. **R.S.P.**: The sacrum is felt towards the right sacroiliac joint.
4. **L.S.P.**: The sacrum is felt towards the left sacroiliac joint.

N.B.: *Sacro anterior positions are more common than sacroposterior.*

MECHANISM OF LABOUR

A. *Sacro-anterior Positions:*

1. The buttocks:
   - The bitrochanteric diameter (10 cm) enters in one of the oblique diameters of the pelvis.
   - The anterior buttock reaches the pelvic floor first and rotates anteriorly 1/8th of a circle.
   - The anterior buttock hinges below the symphysis pubis and the posterior buttock is delivered first by lateral flexion of the spine.
2. The shoulders:
   - The bis-acromial diameter (12 cm) enters in the same oblique diameter as the bitrochanteric.
   - The anterior shoulder reaches the pelvic floor first and rotates anteriorly 1/8th of a circle.
   - The anterior shoulder hinges under the symphysis, the posterior shoulder is delivered first by lateral flexion of the foetal spine.

Fig 24:4 delivery of breech SA position

3. *The head:*
   - The longitudinal diameter of the head enters in the opposite oblique diameter of the pelvis.
   - The occiput rotates anteriorly 1/8th of a circle.
   - The occiput hinges below the symphysis pubis and the head is delivered in flexion.

B. Sacro-posterior Positions:
   - The anterior buttock and shoulder rotate anteriorly 1/8th of a circle.
   - The occiput rotates anteriorly 3/8th of a circle.

The major risks in vaginal breech delivery arise from the fact that the baby is delivered by successively larger foetal parts through the birth canal (limbs, buttocks, shoulder and head).

This may result in **head entrapment** due to:

- Inadequate maternal pelvic capacity (feto-pelvic disproportion).
- Delivery of the foetal body before full cervical dilatation (especially in preterm breech).
- Extension of foetal head during passage through bony pelvis.

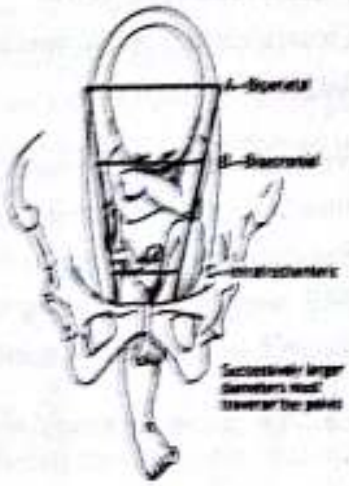


Fig 24.5 delivery of breech in successively larger diameters

DIAGNOSIS OF BREECH PRESENTATION

1. Abdominal examination : (Leuopold's maneuvers)
   - Fundal level: Corresponds to the period of amenorrhea
   - Fundal grip: Head is felt (hard, small, regular, ballotable and tender).
   - Umbilical grip: To detect position of the back
   - Pelvic grip: The buttock is felt (large, soft, irregular, non-ballotable)
   - Auscultation: Foetal heart sounds are heard above the level of the umbilicus.

2. Ultrasound Diagnosis: (during pregnancy and labour)
   - Confirms persentation, in addition to gestational age, foetal weight, and type of breech.
   - Exclusion of congenital anomalies.
   - Detects foetal head hyperextension which may preclude attempts at vaginal delivery.
   - Detects associated conditions as placenta praevia or twins.

N.B.: Contracted pelvis is excluded by X-ray or C.T. scan pelvimetry and not by ultrasound.

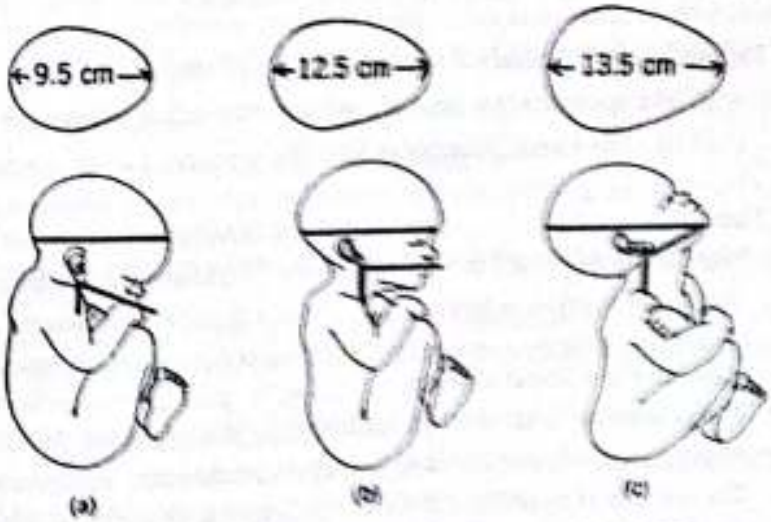


Fig 24.6 Full flexion Suboccipito-bregmatic

Fig 24.7 Partial flexion Occipito-frontal

Fig 24.8 Extension Occipito mental

3. Vaginal examination: (during labour with the cervix dilated)

Landmarks for diagnosing breech on vaginal examination include palpating:

- 3 bony prominences (The 2 ischial tuberosities and the tip of the sacrum).
- The feet beside the buttocks (in complete breech).

Vaginal examination in breech presentation also helps to:

- Exclude contracted pelvis.
- Exclude rupture of the membranes which commonly occurs early.
- Exclude cord prolapse.

Confusion in the diagnosis of frank breech on examination: may occur due to;

- Possible early engagement of the small breech resulting in a lower fundal level
- Restricted ballotment of the foeatl head at the fundus due to splinting by the legs
- FHS being heard at a lower level (below umbilicus).
- Vaginal examination: The feet are not felt beside the buttocks.

N.B.: The proper diagnosis and competent management of breech presentations will significantly reduce both maternal and foetal complications associated with vaginal breech delivery.

MANAGEMENT OPTIONS IN BREECH PRESENTATIONS

1. **External Cephalic Version (ECV):** To convert breech into cephalic presentation.
2. **Elective Caesarean Section (ECS):** see indications for C.S. in breech.
3. **Trial of Vaginal Breech Delivery (TVBD):** In selected cases (see management in labour).

I. EXTERNAL CEPHALIC VERSION (ECV)

ECV allows conversion of breech presentations into cephalic ones in order to be able to perform cephalopelvic disproportion tests, and to avoid foetal and maternal complications associated with vaginal breech delivery.

The procedure should be restricted to well-selected cases, and performed by trained staff member, to avoid unnecessary complications that may result from forcible trials in high risk cases.

- **Timing of ECV:** Usually done between 35-37 wks
  - < 35 wks spontaneous cephalic version may occur without the need for ECV.
  - > 37 wks the foetus is larger in size, the amount of liquor decreased and ECV is difficult.

- **Technique of ECV:**
  - The patient lies down comfortably in the Trendlenberg position.
  - The foetal position is determined, and the bladder evacuated.
  - The head is gently manipulated into the pelvis under ultrasound guidance, in a direction that maintains the flexed attitude.
  - The procedure is sometimes facilitated by giving a prophylactic tocolytic agent.
  - Anaesthesia is better avoided as pain is a safeguard against rough manipulations.
  - The vulva is exposed to discover bleeding due to placental separations.
  - FHR tracings are done before and after procedure.

- **Complications and risks of ECV:**

1. Separation of the placenta leading to accidental haemorrhage.
2. Rupture of the membranes and preterm labour.
3. Cord accidents and foetal bradycardia.

- **Contraindications to ECV:**

1. Placenta praevia (to avoid bleeding and because C.S. is already indicated).
2. Oligohydramnios (no space for version) or poly hydramnios (lie will be unstable).
3. Multiple gestation (difficult version of only one foetus in multifoetal pregnancies).
4. Preeclampsia or hypertension (high risk pregnancy where prolonged vaginal delivery is risky).
5. Plan to deliver by CS for any other reason (any degree of contracted pelvis, uterine scar as previous C.S. or myomectomy, elderly primigravida, etc...)

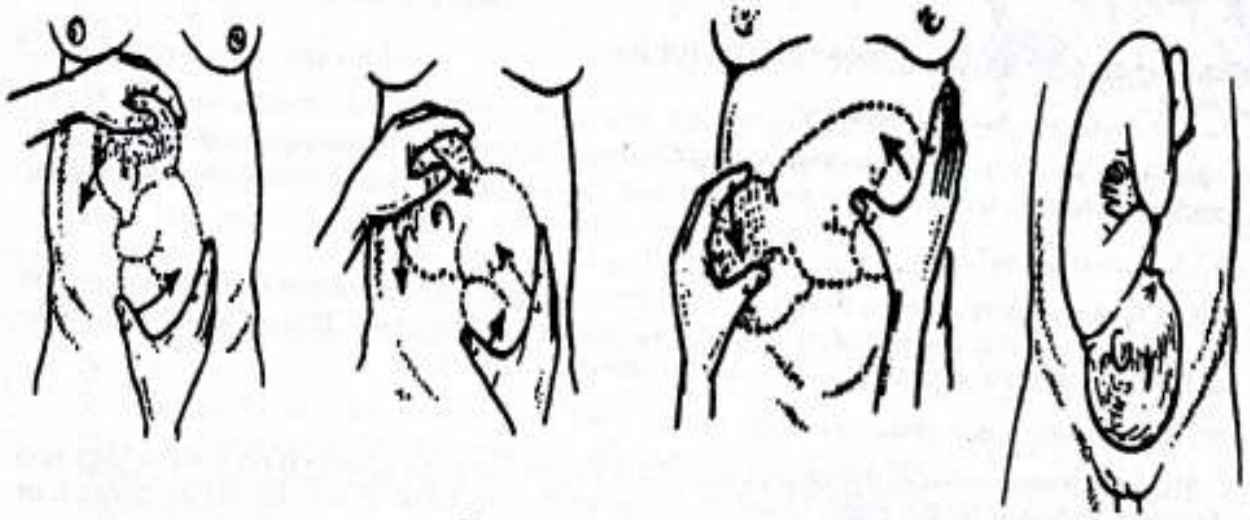


Fig 24:9 External cephalic version ECV

II. CAESAREAN SECTION IN BREECH PRESENTATION:

Due to the **higher maternal and foetal morbidity and mortality** associated with vaginal breech delivery, there is a world wide increase in the rates of C.S. reaching up to 80% in many centres.

- **Indications of CS in breech presentation:**

1. Foetal weight > 3.5 kg: LGA foetuses are more liable to head entrapment.
2. Foetal weight < 2.5 kg: The extreme preterm foetus is very liable to intracranial haemorrhage due to sudden compression and decompression of the foetal head.
3. Footling presentation: to avoid cord prolapse and head entrapment if the feet slip through a partially dilated cervix.
4. Hyperextension of the foetal head: to avoid arrest of the extended after-coming head
5. Any degree of contracted pelvis: There is no place for trial of labour in breech presentation as moulding doesn't occur in the after coming head of breech.
6. Any degree of placenta praevia: (incomplete – complete – partial).
7. Other indications for CS (irrespective of foetal presentation) as: previous C.S. or myomectomy scar, elderly primigravida, prolapsed pulsating cord, foetal distress ...etc.

III. TRIAL VAGINAL BREECH DELIVERY:

- **Criteria to be fullfilled before allowing a trial for vaginal breech delivery:**
  1. Complete or frank breech presentation.
  2. Gestational age > 36 weeks.
  3. Estimated foetal weight (by US) 2.5 – 3.5 kg.
  4. Flexed foetal head (detected by US or plain X-ray).
  5. No evidence of contracted pelvis (by pelvimetry).
  6. No other indications of CS (see before).

- **The following should be available:**
  - Experienced obstetrician.
  - Anaesthesiologist readily available.
  - Assistant ready to perform Kristiller manoeuvre (see later).

- **METHODS FOR VAGINAL BREECH DELIVERY:**

1. Spontaneous breech delivery:

The foetus is delivered spontaneously without any help by the obstetrician other than supporting the foetus. It is not recommended since it may leave the foetus for many complications.

2. Assisted breech delivery (partial breech):

The foetus is allowed to be delivered spontaneously until the level of the umbilicus, then the obstetrician assist in delivery of the shoulders and after coming head. This is the the commonest and safest method used in vaginal breech delivery (see later)

3. Breech extraction (total breech):

The obstetrician extracts the entire body of the foetus starting by traction on the the legs and buttocks, followed by the body, shoulder and after coming head. It is done under general or regional anaesthesia.

Breech extraction may carries many risks to the foetus and mother, therefore its use is restricted nowadays to very few conditions, namely:

- Delivery of the second twin presenting in a breech presentation
- In emergency as prolapsed pulsating cord; with the cervix fully dialted, and facilities for CS are not readily available.

ASSISTED BREECH DELIVERY

A senior well-trained staff member should be attending to minimize any possible complications.

1. **Avoid maternal bearing down** in the first stage before full cervical dilatation is achieved.
2. **Positioning:** The patient is placed in the lithotomy position when the buttocks are seen distending the perineum.
3. **Episiotomy:** is mandatory when the perineum is maximally distended by the buttocks, as it;
   - Decreases the amount of lateral flexion of the spine.
   - Avoids extended perineal tears.
   - Avoids sudden compression and decompression of the foetal head, thus minimizing the risk of intracranial haemorrhage.

4. Delivery of the Buttocks, Legs and Trunk:

- ***Complete breech:*** The feet and legs are hooked out and delivered first followed by delivery of the buttocks (aided by maternal bearing down and without traction).
- ***Frank breech:*** delivery of the buttocks first followed by flexing the leg at the knee by pressing on the popliteal fossa, and sweeping the foot downwards in front of the body.
- ***Pulling down a loop of the cord:*** To avoid cord compression and to detect cord pulsations.
- ***Keep the foetal back always anterior*** and covered by a ***warm towel:*** to prevent premature stimulation of foetal breathing before the after coming head is delivered.

Fig 24:10 vaginal breech delivery

N.B.: Avoid rapid traction on the foetal trunk and body as this may lead to extension of the arms or the head resulting in arrest in delivery of the shoulders or after coming head.

5. Delivery of the Shoulders:

- When the anterior scapula appears under the symphysis pubis, each arm is delivered by hooking a finger in the elbow and sweeping the arm in front of the chest.
- After delivery of the shoulders, the back is rotated anteriorly so as to keep it under the symphysis pubis to ensure anterior rotation of the occiput of the after coming head.
- ***Lovset's manoeuvre:*** If extension of the arms is discovered during delivery:
  - Rotation of foetal trunk 180 °to bring the posterior shoulder below the symphysis pubis. The arm which is now anterior can be hooked by the index finger of the operator and brought down from behind the symphysis.
  - Repeat trunk rotation 180 °in the opposite direction and deliver the second arm similarly.

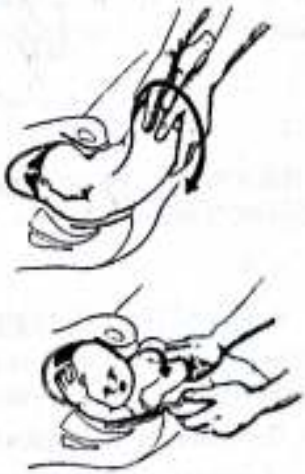


Fig 24:11 Lovset's maneuver

6. Delivery of the after coming Head:

1. Burns-Marshall's method:

- The infant is left hanging, so that its weight exerts gentle traction.
- When the occiput appears under the symphysis, the infant is held from the feet and the body is lifted towards the mother's abdomen. Fracture of the cervical spine may occur if its is done before appearance of the occiput.

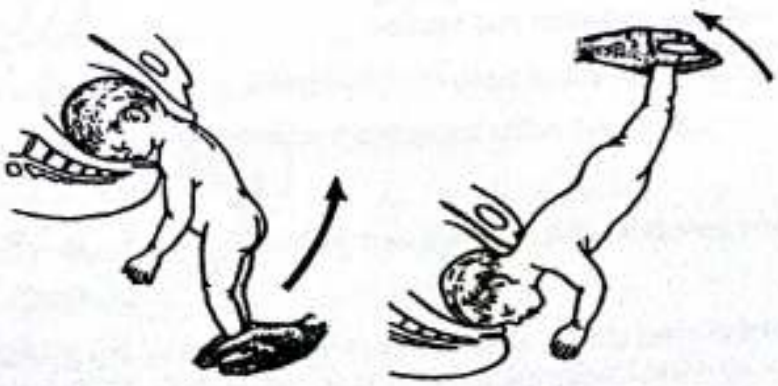


Fig 24:12 Burns-Marshall's Method

2. Mauriceau-Smellie-Veit method:

- **Jaw flexion:** done by the left hand: with one finger put in the mouth to allow flexion of the head, or better 2 fingers put on the maxillae to avoid jaw dislocation.
- **Shoulder traction:** done by the right hand:
  - The index and ring fingers are placed on the shoulders from behind, while the middle finger is put on the suboccipital region, to promotes flexion.
  - Traction is done downward and backwards till the occiput appears under the SP, then downward and forward.

Fig 24 :13 Mauriceau-Smellie-

***N.B.: Kristiller manoeuvre** consists of gentle fundal pressure during uterine contractions. It is done by an assistant to help the other methods because it guides the head into the pelvis, and maintains flexion of the head.*

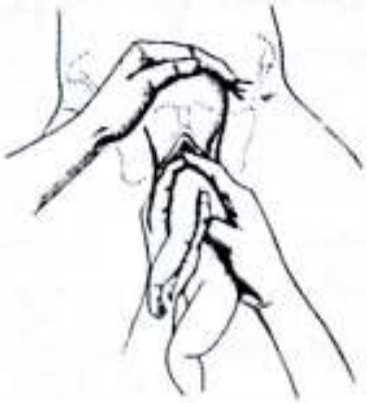


Fig 24:14 Kristiller maneuver

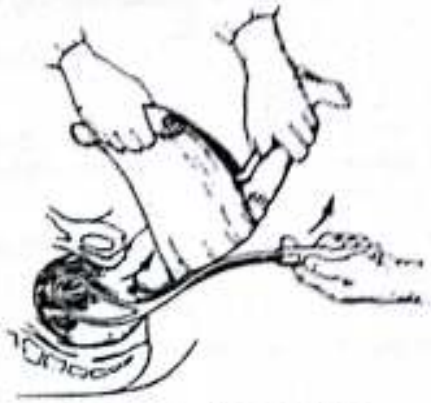


Fig 24:15 The Pipers forceps

3. Use of the Obstetric Forceps:

The use Pipers forceps (a long forceps characterized by having a perineal curve), may offer some advantages in some cases:

- Traction will be applied on the head preventing over stretching of the neck
- Forceps may promote head flexion and protects it from sudden compression and decompression.

Posterior rotation of the head

- In case posterior rotation of th eback occurs, it I smanaged by flexion of the body towards maternal abdomen and traction on the shoulder from behind (Prague maneuver).

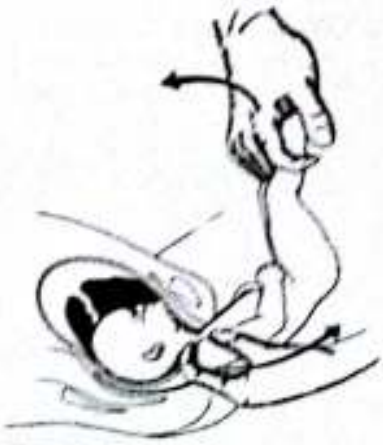


Fig 24:16 The Prague maneuver

COMPLICATIONS OF BREECH DELIVERY

I. MATERNAL COMPLICATIONS:

1. Early rupture of the membranes due to improper fitting of the breech in the pelvis.
2. Prolonged labour due to uterine inertia or arrested breech delivery
3. Birth canal injuries e.g.
   - Perineal and vaginal lacerations (especially with inadequate episiotomy and forcible traction).
   - Cervical tears due to delivery before full cervical dilatation (especially with prematurity)
   - Rupture of the uterus due to obstructed labour, or extended cervical laceration.
4. Postpartum haemorrhage:
   - *Atonic* due to prolonged labour and maternal exhaustion.
   - *Traumatic* due to birth canal injuries (especially cervical tears and rupture uterus).
5. Puerperal sepsis due to:

   Prolonged rupture of membranes, prolonged labour, and birth canal injuries.

II. FOETAL COMPLICATIONS:

Prolonged entrapment of the after coming head may result in severe foetal hypoxia and asphyxia with resultant short and long term adverse foetal and neonatal effects. On the other hand forcible traction on the entrapped head may result in severe foetal birth injuries with marked foetal morbidity and even mortality.

1. **Intracranial haemorrhage:**
   - Due to sudden compression and decompression of foetal head during its passage within maternal bony pelvis or through a non fully dilated cervix. It is the commonest cause of foetal mortality in vaginal breech delivery .
   - Prevention:
     - Fundal pressure is done only during uterine contractions.
     - Episiotomy, generous to minimize head compression .
     - Vitamin K, IM given to the mother early in labour.
     - Forceps delivery when recomended.
2. **Fracture of the Cervical Spine:**

   Prevented by delivering the foetal head after the occiput appears below the symphysis pubis.
3. **Asphyxia,** due to:
   - Cord compression.
   - Premature stimulation of respiration (avoided by the use of warm towels to cover the infant).
   - Prolonged head compression if entrapped within the pelvis.
4. **Injury of Abdominal Organs:**

   Prevented by grasping the infant from the hip, not the abdomen where the liver and kidneys.
5. **Other Foetal Injuries:**

   Fracture of the femur and humerus, dislocation of hip joint, brachial plexus palsy, and sternomastoid rupture, rarely occur usually in difficult vaginal breech deliveries.

6. The Preterm Breech

The extremely premature breech < 34 weeks is better delivered by CS as it is more liable to complications than the term breech especially:

- Intracranial haemorrhage (due to pliable skull bones, affected by rapid compression and decompression).
- Intraventricular haemorrhage (due to capillary fragility, associated with hypoxia).
- Asphyxia neonatorum (due to prolonged head or cord compression).
- Foetal birth injuries as brachial plexus and soft tissue injuries (as liver and kidney).

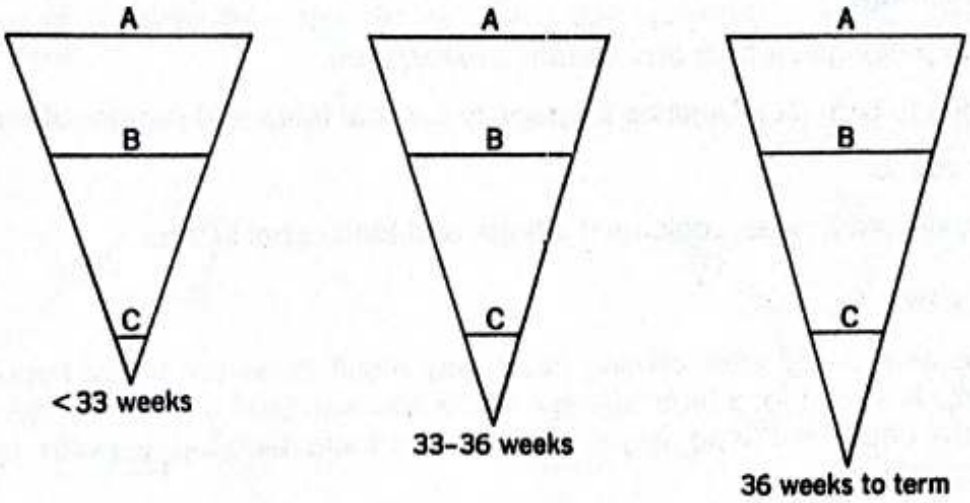


Fig 24:16 Effect of prematurity on relation between head circumference, abdominal circumference, and bitrochanteric diameter , increasing the risk of head entrapment during vaginal delivery through a non fully dilated cervix

Key points in management of breech presentation:

1. *ECV may be offered between 36-37 weeks in carefully selected cases.*
2. *A vaginal breech delivery, when decided, should be well planned.*
3. *An experienced well trained obstetrician should attend all vaginal breech deliveries.*
4. *Nearly 80% of breech presentations will end up in C.S.*
5. *Preterm breeches (<34 weeks), are better delivered by C.S.*

25 MALPRESENTATIONS
Shoulder, Cord, and Complex Presentations

Shoulder presentation
- *Definition & incidence*
- *Positions*
- *Aetiology*
- *Diagnosis*
- *Management*

Cord presentation
- *Definition & incidence*
- *Aetiology*
- *Diagnosis*
- *Management*

Complex presentation

SHOULDER PRESENTATION

DEFINITION:

It is a transverse lie in which the long axis of the foetus crosses the long axis of the mother. The shoulder lies over the pelvic inlet while the head is present at one iliac fossa.

INCIDENCE: 1:200

- Far more common un multiparas than proimigravidas due to their lax abdominal wall muscles
- In primigravidas it usually occurs in presence of uterine anomalies or multifoetal pregnancies.

POSITIONS:

Four possible positions depending on the direction of the back, which lies anterior in 60% of cases and posterior in 40% of cases:

- Left Dorso-Anterior (LDA)
- Right Dorso-Anterior (RDA)
- Right Dorso-Posterior (RDP)
- Left Dorso-Posterior (LDP)

The head lies in:

- Right iliac fossa in RDA & RDP
- Left iliac fossa in LDA & LDP

Fig 25:1 LDP

Fig 25:2 RDP

CAUSES:

1. Lax abdominal wall (pendulous abdominal as in multiparity).
2. Prematurity
3. Hydramnios
4. Twins and multifoetal pregnancy
5. Abnormality in the shape of the uterus;as bicornuate and septate uterus
6. Ovarian tumours, fibroids or placenta praevia (prevents engagement of the fetal head)
7. Contracted pelvis (especially extremely contracted inlet.

DIAGNOSIS

- **Abdominal Examination:**
  - The uterus is broad transversely with fundal level lower than expected for gestational age
  - The head felt in one iliac fossa (neither felt in the pelvis, nor the fundus).
  - When the back is anterior a hard resistant plane is felt across the abdomen.
  - When the back is posterior irregularity of the limbs is felt.
- **Vaginal Examination**
  - The ribs may be felt above pelvic inlet.
  - Later in labour a hand and an arm frequently prolapse and the umbilical cord may be felt.
  - **A prolapsed arm must be distinguished from a leg,** by;
    - A) The elbow is sharper than the knee.
    - B) There is no heel and abduction of the thumb will distinguish a hand from a foot.

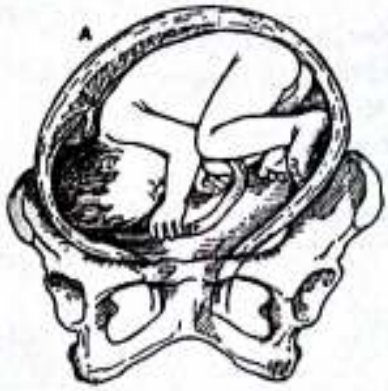


Fig 25:3 Transverse lie RDA

MANAGEMENT OF SHOULDER PRESENTATION

- **During pregnancy:**

  External cephalic version (ECV) could be attempted, when the condition is diagnosed beyond 36 weeeks unless C.S. was indicated or ECV was contraindicated.

- **Early in labour:**
  If the membranes are intact, ECV is done followed by ROM, to maintain a longitudinal lie. If an oblique or transverse lie persists, i.e.failed ECV, C.S. is performed immediately.

- **Late in labour:**
  1. Caesarean section (C.S.) is the safest method of delivery.
  2. In exceptional cases, as second twin, with intact membranes and fully dilated cervix, it is possible to perform artificial rupture of membranes , internal podalic version and breech extraction.

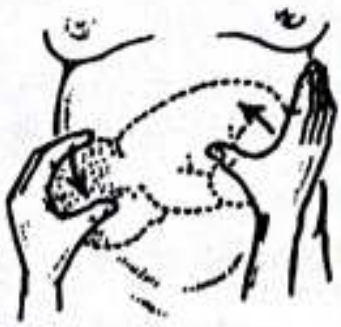


Fig 25:4 ECV

NEGLECTED SHOULDER:

It is considered a serious complication that occurs when the membranes rupture and the arm is prolapsed through the cervical canal into the vagina. If intervention is delayed, the foetus may be lost or severely distressed and the uterus may rupture. Caesarean section is immediately done.

UNSTABLE LIE

When the foetus frequently changes its axis from transverse to longitudinal to oblique after 34 weeks of gestation it is referred to as an unstable lie. The case is managed according to the foetal lie at the onset of labour.

CORD PRESENTATION

DEFINITIONS

- **Cord presentation:** The cord lies below the presenting part with intact membranes.
- **Cord prolapse:** The cord lies below the presenting part with ruptured membranes.

INCIDENCE

It is a rare condition occurring in nearly 1/300 cases. It carries higher risk of foetal morbidity and mortality because of the danger of obstructing the circulation in the umbilical vessels, with consequent foetal hypoxia and asphyxia.

Fig 25;5 Cord Presentation

Fig 25:6 Cord Prolapse Membranes Ruptured

CAUSES AND PREDISPOSING FACTORS:

1. Long cord (above 80 cm).
2. Mapresentations which cause non fitting of the presenting part in to the pelvis: (shoulder - face - breech).
3. High non engaged head (contracted inlet- prematurity - Hydramnios - twins- placenta praevia)

DIAGNOSIS

- **Cord presentation** is seldom discovered early during labor.
  - Cord pulsations can be felt through intact membranes when the cervix is partially dilated.
  - Foetal bradycardia may occur when cord is compressed by the descending presenting part.
- **In Cord Prolapse**, a loop of cord may be felt in the vagina or even present at the vulva.

Every patient should be examined vaginally immediately after ROM to exclude cord prolapse.

- Prolapsed pulsating cord: the foetus is alive, and pulsations are usually felt in the cord.
- Prolapsed non pulsating cord: the foetus is usually dead with no pulsations in the cord.

N.B: Even when cord pulsations are absent, FHS should be checked by Duplex sonicaid instrument or by US to exclude cord compression in a living foetus as a cause for absent pulsations.

PROGNOSIS

- In cases of severe bradycardia foetal prognosis is poor with a stillbirth and neonatal death occuring in > 20% of cases.

MANAGEMENT

- **Prolapsed non pulsating cord:** IUFD is confirmed by US and vaginal delivery is allowed.
- **Prolapsed Pulsating cord:** Foetus living but may be distressed
  - **C.S.** is the safest method, to avoid foetal hypoxia and death.
  - Vaginal delivery may be allowed in case the cervix is fully dilated

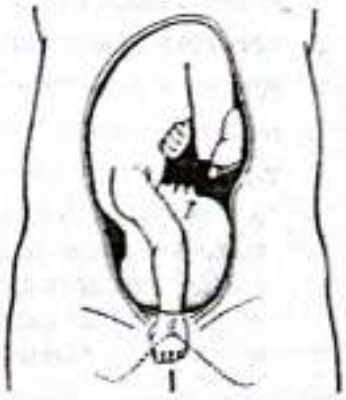


Fig 25:7 Complex presentation

COMPLEX PRESENTATION

- Rarely an arm may present beside the presenting part, which is mostly cephalic.
- In most cases reposition of the limb will allow labour to be completed safely.
- If the cord is prolapsed beside the arm the case is managed as in cord prolapse.

26 TWINS and MULTIFOETAL PREGNANCY

- *Introduction and Incidences*
- *Maternal and foetal risks*
- *Classification of twin pregnancy*
- *Diagnosis*
- *Management during pregnancy*
- *Management during labour*
- *Delivery of the1st twin*
- *Delivery of the 2nd twin*
- *Indications of C.S. in MFP*
- *Complications of MFP*
- *The Discordant twin*
- *Death of one foetus*

INTRODUCTION AND INCIDENCES

- In **1-2%** of pregnancies there is more than one foetus in the uterus.
- The incidence has been **steadily rising** in the last 2 decades due to the liberal use of drugs for induction of ovulation in the treatment of infertility.
- **Twin** pregnancy represent nearly **97%** of multifoetal pregnancies (MFP).
- **Triplets** have an incidence of about 1-2%
- **Quadruplets** and **quintuplets** less than 1%.

MATERNAL AND FETAL RISKS OF MFP:

- Most twin pregnancies will run a safe conduct during pregnancy and delivery, yet the condition is considered a high risk pregnancy (HRP) as it carries higher incidence of maternal and foetal perinatal complications.
- Most cases of quadruplets or more will end in a mid trimesteric abortion or an early PTL.
- The more the number of foetuses in a MFP, the higher the expected complications and the poorer is the prognosis especially concerning the foetal outcome. Risks include;

1. Miscarriage, (risk increases with larger number of foetuses).
2. Preterm labor, (risk increases the larger the number of foetuses).
3. Abnormal fetal growth patterns (IUGR – discordant twins)
4. Abnormal placentation (placenta praevia – accidental haemorrhage).
5. Maternal medical illness (Diabetes, Preeclampsia, Polyhydramnios, ...).
6. Increased fetal perinatal morbidity and mortality due to;
   a. Prematurity, with more changes for neonatal ICU admissions.
   b. Malpresentations and malpositions, leading to prolonged and dysfunctional labour
   c. More incidence of C.S. (abnormal labour, non cephalic 1st twin, placenta praevia, etc..).
   d. Obstetric manipulations during delivery internal podalic version, breech extraction, etc..).
   e. Congenital foetal anomalies (CFA).

TWIN PREGNANCY

CLASSIFICATION

- Twins are classified according to the number of fertilized oocytes into; **monozygotic** and **dizygotic** twins.
- They are classified according to the presence or absence of chorionic and amniotic membranes, into; **monochorionic** and **dichorionic twins**.

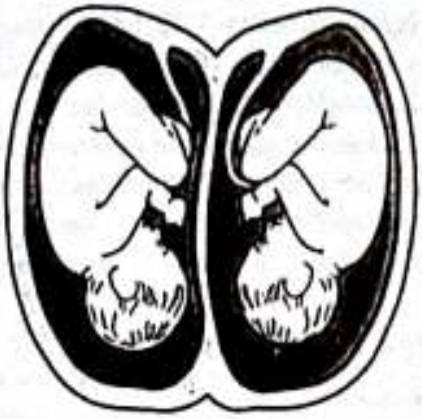


Fig 26:1 Diamniotic Dichorionic twins

I- DIZYGOTIC TWINS (Binovular – non identical):

- Dizygotic twins are the **commoner** type of twins.
- **Two** different ova are fertilized separately by **two** different sperms in one cycle.
- They are **genetically non identical**, and may be of the same or of different sex.
- They are always of the **diamniotic / dichorionic** type

II- MONOZYGOTIC TWINS (uni-ovular – identical):

- Monozygotic twins represent the **less common** type of twins.
- **Single ovum** is fertilized by **single sperm**, followed by division into two cell masses.
- They are **genetically identical**, of similar sex, features and external appearance.
- May be **mono or diamniotic, mono or dichorionic**, according to the stage at which division of the zygote has occurred after fertilization:
  1. **Dichorionic diamniotic (30%):** division occurs within **72h** of fertilization, each fetus will have a separate placenta and a separate amniotic sac.
  2. **Monochorionic diamniotic (65%):** division occurs between **4-8 days** of fertilization, the twins will share a single placenta (monochorionic) but each will be in a separate amniotic sac (diamniotic).
  3. **Monochorionic monoamniotic (5%):** division occurs after **9-12 days** of fertilization. They will share a single placenta and a single amniotic sac.
  4. **Conjoined twins (very rare):** The zygote division occurs **>13 days** after fertilization, here division is incomplete and the twins will be adherent to each other at a certain point, the most common is thoracopagus but it may be craniopagus, ischiopagus.

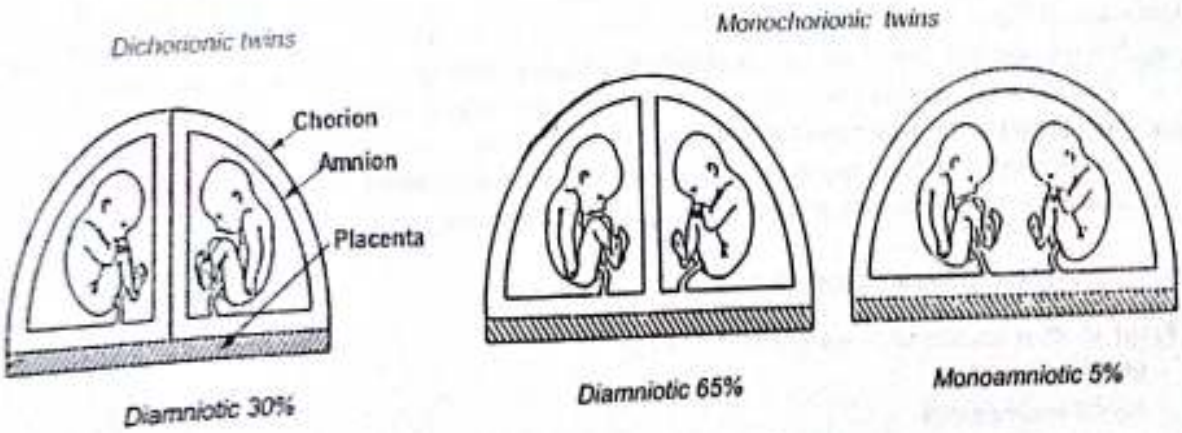


Fig 26:2 Classification of twin pregnancies

INCIDENCE OF TWIN PREGNANCY

A. **Monozygotic twins:** 1/250, nearly constant worldwide.

B. **Dizygotic twins:** 1/89, highly variable depending on many factors including:
   1. Racial factors: Black race and African countries.
   2. Hereditary factors: Families with history of twins.
   3. Maternal age: Slight increased with older maternal age.
   4. Parity effect: Slight increase with multiparity.
   5. Ovulation induction: Increase with Clomiphene and HMG.
   6. Assisted reproductive techniques (ART): IUI, IVF and ICSI.

DIAGNOSIS OF TWIN PREGNANCY

History:

- Past or family history of twins in either wife or husband families.
- Recent treatment with drugs used for induction of ovulation.
- Recent attempts for ART (IUI, IVF and ICSI).

Symptoms:

- Exaggerated symptoms of early pregnancy (nausea, vomiting... etc.)
- Rapid increase in abdominal girth and excessive maternal weight gain.

Signs on Abdominal Examination:

- **Inspection**: Increased abdominal size compared to gestation age.
- **Palpation**: (highly suggestive – rarely conclusive)
  - Fundal level appears higher than calculated gestational age.
  - Two similar fetal parts (2 heads / 2 buttocks) felt at two different poles.
- **Auscultation**: (poorly suggestive – never conclusive)
  - Two different FHR with maximum intensity at 2 different points

Signs on Vaginal Examination (During Labor):

- Cephalic presentation while abdominal palpation was suggestive of breech.
- Small fetal head while abdominal palpation suggested an oversized fetus.
- Small fetal limbs and feet felt vaginally while abdominal examination assumed a large abdomen with an oversized fetus.

Ultrasound Diagnosis of Twin Pregnancy (sure – accurate –early):

- Visualization of more than one **gestational sac** (5-6 weeks).
- More than one **embryo** with positive cardiac pulsations (7-8 weeks).
- Assurance of the exact **gestational age** (1st trimester).
- Determination of **twin zygocity** (number of placentae & chorion).
- Exclusion of **congenital anomalies** and **conjoined twins**.

DIFFERENTIAL DIAGNOSIS OF TWIN PREGNANCY

From all other causes of oversized abdomen as

- Maternal obesity
- Foetal macrosomia
- Polyhydramnios,
- Miscalculation of LMP
- Associated pelvic mass (as large fibroids or ovarian cysts).

MANAGEMENT OF TWIN PREGNANCY

MANAGEMENT DURING PREGNANCY (high risk pregnancy):

- Less exposure to stress and fatigue.
- Better nutrition, adequate vitamins, iron & Calcium supplements
- Closer follow up with more frequent ANC visits.
- Laboratory investigations done more frequently as:
  - Complete blood picture (CBC), to check for anaemia.
  - Blood glucose (BG), to detect gestational diabetes.
  - Kidney functions (serum urea & creatinine), for (PIH).
  - Urine analysis, for early detection of albuminuria in (PE), and pus cells in (UTI).

MANAGEMENT DURING 1ST STAGE OF LABOR:

- Determination of the lie, presentation & FHR of each baby, (US is very helpful).
- Assess the progress of labor by special labor partogram.
- Mild sedation and parental nutrition to guard against inertia and maternal exhaustion.

MANAGEMENT DURING 2ND STAGE OF LABOUR:

1. Delivery of the First Twin

- **Vertex presentation:** vaginal delivery is allowed, with careful observation.
- **Non vertex presentation:**

- C.S. is the **best** option to breech presentation to avoid complications of breech delivery, and the possibility of locked twins (see later).
- C.S. is the **only** option in transverse lie to avoid obstructed labour, as ECV in presence of a second twin is impossible.

2. Delivery of the Second Twin

After delivery of the first twin, the cord is divided long away from vagina, the uterus usually takes a short period of rest, 10-15 minutes, before it regains its contractions during which evaluation of the **2nd twin** is done:

- **Vertex Presentation**: ROM is done, and spontaneous VD is allowed safely.
- **Breech Presentation:** ROM is done, and VD is accomplished usually by breech extraction. This is almost the only indication for breech extraction in modern obstetric practice.
- **Transverse lie:**
  - If Membranes are still intact: do external cephalic version (ECV), ROM, and allow for vaginal delivery VD (ECV – ROM – VD).
  - If ROM already has occurred, or ECV failed: do internal podalic version (IPV) and vaginal breech extraction BE (ROM – IPV – BE).

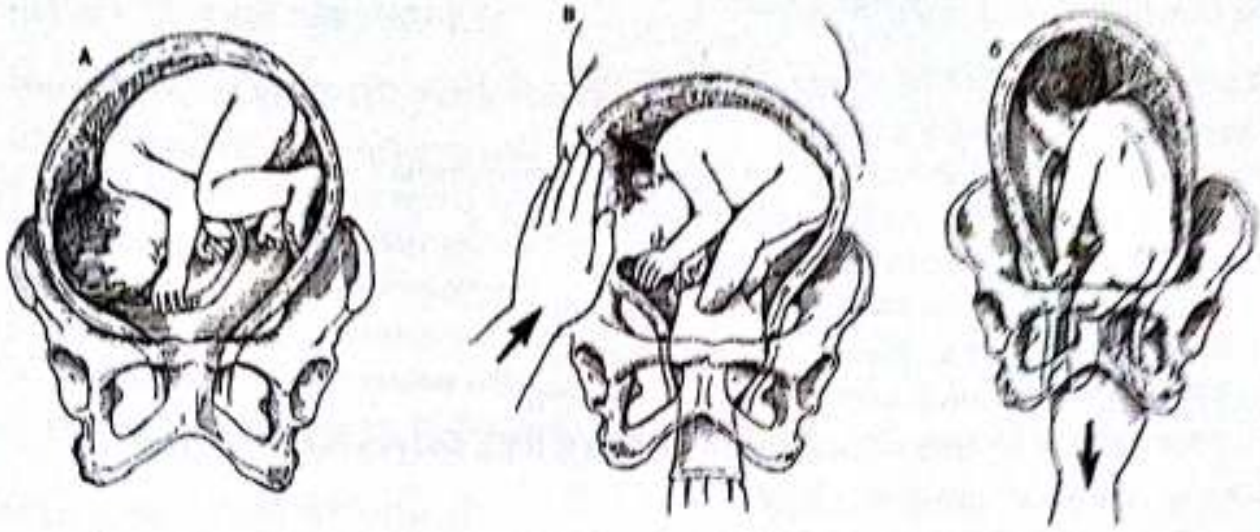


Fig 26:3 Delivery of the second twin (transverse lie) by Internal podalic version (IPV) and vaginal breech extraction

The Locked Twins

This is a rare condition that may occur when the head and neck of the 1st fetus in a breech presentation, is entangled with the head and neck of the 2nd fetus (usually cephalic, or transverse lie),

Both heads are entrapped at the pelvic brim with the body of the first twin has already been delivered through the birth canal.

Dis-impaction under general anaesthesia can be attempted but usually fails. The first twin is thus sacrificed and a C.S. is performed to save the second.

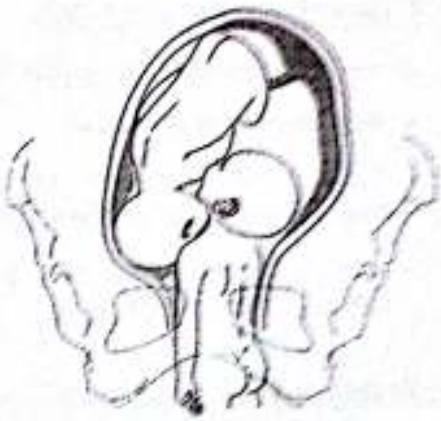


Fig 26:4 Locked twins

MANAGEMENT IN THE 3RD STAGE OF LABOUR:

MFP are more liable to complications in the 3rd stage of labor compared to singleton pregnancies, especially:

1. **Atonic post partum haemorrhage:** due to uterine over-distension. Close follow up, with continuous uterine massage and liberal use of ecbolics.
2. **Traumatic post-partum haemorrhage:** due to possible vaginal or cervical lacerations from internal manipulations or delivery before full cervical dilatation. Exploration of the birth canal is recommended and surgical repair accomplished.
3. **Puerperal sepsis**: due to prolonged delivery, ROM and possible internal manipulations. Broad spectrum antibiotic coverage is a safe option.

INDICATIONS OF C.S. IN MFP

- Non-vertex presentation of the 1st twin (Breech or Transverse lie).
- Retained living 2nd twin (failed ECV or IPV).
- Triplets or more fetuses (relative indication).
- Monoamniotic twins (relative indication).
- Conjoined twins (if good chance for survival).
- Other indications for C.S. as failed progress of labor, fetal distress, previous C.S, previous myomectomy, placenta praevia...etc.

COMPLICATIONS OF MULTIFETAL PREGNANCY

A. MATERNAL COMPLICATIONS: (More severe the more the number of fetuses).

1. During pregnancy: High risk pregnancy due to;
   - 1st and 2nd trimesteric abortions
   - Hyperemesis gravidarum
   - Medical disorder (Anaemia, GDM, PIH, PE, etc.)
   - Placental problems (Placenta praevia – Accidental haemorrhage)
   - Amniotic fluid disorders (Polyhydramnios / Oligohydramnios)
   - Pressure symptoms (urinary and respiratory).
2. During labor: Higher incidence of;
   - C.S. due to (malpresentations, prolonged and dysfunctional labor, PROM).
   - Post partum haemorrhage (atonic and traumatic).
   - Puerperal sepsis (ROM – Internal manipulations).

B. FETAL COMPLICATIONS:

More common in uni-ovular than in bi-ovular twins, in monoamniotic than in diamniotic ones, and in multifoetal compared to twin pregnancies.

1. Abortion: incidence twice as singleton pregnancies
2. Preterm labor (PTL).
3. Intrauterine growth restriction (IUGR).
4. Foetal Congenital Anomalies (FCA).
5. Conjoined twins.
6. Discordant twin growth.

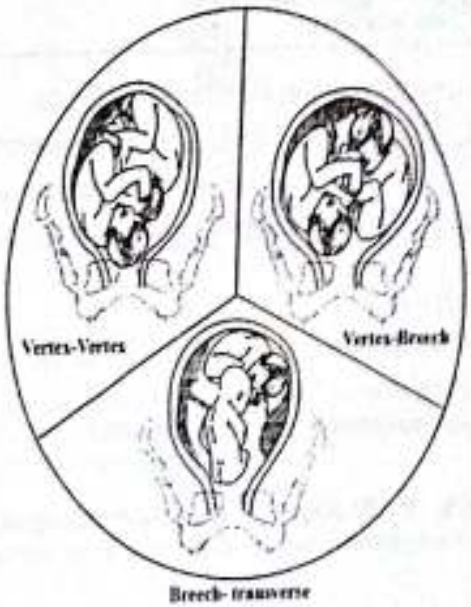


Fig 26:5 different twin presentations

THE DISCORDANT TWINS:

One fetus shows abnormally increased growth, while the second shows severe IUGR with a difference in fetal weights > 25%. The condition may be due to:

- Unequal placental masses.
- Genetic syndromes.
- Twin-to-twin transfusion syndrome. The latter condition is specific to Monochorionic twins only, where abnormal arterial and venous vascular anastomosis within the placenta, result in anemia and hypovolaemia in the fetus showing IUGR, and polycythaemia and hypervolaemia in the overgrowing one.

THE VANISHING TWIN:

Early in the first trimester, one embryo may stop growing, dies and will eventually disappear and become absorbed while the second one continues to grow in a normal pattern. The condition is diagnosed and followed up by ultrasonography.

DEATH OF ONE FETUS DURING PREGNANCY

A) In the first trimester: pregnancy is allowed to continue while the second fetus is closely monitored. There is minimal risks to the mother.

B) In the second trimester:

- The dead fetus is compressed and displaced to one side by the growing sac and fetus.
- Pregnancy commonly continues but the risk of ***hypofibrinogenaemia*** and ***DIC*** is higher.
  - Close monitoring of maternal bleeding and coagulation profiles, fibrinogen and prothrombin
  - Follow up of the living fetus by close ANC and repeated US.
  - Delivery of the living twin should be attempted once it can survive conditions outside the uterus, or if maternal hazards as DIC are anticipated.

27 ANALGESIA AND ANAESTHESIA IN LABOUR AND DELIVERY

CAUSES OF PAIN DURING LABOUR:

1. Stretch of the cervix during dilatation
2. Distention and ischaemia of the muscle wall of the uterus with build up of lactate
3. Stretch of the vagina and perineum is the second stage

PAIN PATHWAY:

1. Pain arising from the uterus is carried by sympathetic nerve fibres to T10,11, 12 and L1 segments
2. Pain arising from the uterus, cervix, vagina and perineum is carried by nerve fibres to S2,3,4 segments (mainly pudendal nerve).

IDEAL PAIN RELIEF should provide *good analgesia*, be *safe* for the mother and foetus, be reversible if necessary and in the same time *reversible* if necessary.

PHARMACOLOGICAL METHODS for pain relief :

1. SYSTEMIC ANALGESIA AND SEDATION

(a) Narcotic Drugs:

1. Meperidine hydrochloride (pethidine)

- Given 50-100 mg IM every 3-4 hrs or 20-50 mg intermittent I.V. doses
- May cause nausea for mother and neonatal respiratory depression if given < 2 hours before delivery is accomplished
- Antidote is Naloxone

2. Morphine Sulphate:

- Rarely used as it ios associated with higher incidence of respiratory depression although 10 times more potent than Meperidine

3.Butorphanol (Stadol)

Five times more potent than morphine with less neonatal respiratory depression.

4. Nalbuline (Nubain):

(b) Non Narcotic Drugs:

usually given in combination with narcotic drugs to produce good analgesia and sedation

1. **Benzodiazepines:** Diazepam (Valium) and Midazolam (Dormicum). Both may cause neonatal hypotonicity, hypoactivity and impaired temperature regulation if given in repeated doses.
2. **Phenothiazine derivatives:** such as Promazine hydrochlride (Sparine)

(c) Inhalation Drugs:

1. **Nitrous oxide gas:** can be inhaled periodically with uterine contractions in a mixture with oxygen (50:50) Entonox apparatus)
2. **Trilene** (trichloroethylene) was supplied by hand-held inhalers, but now abandoned because of its toxic metabolites.

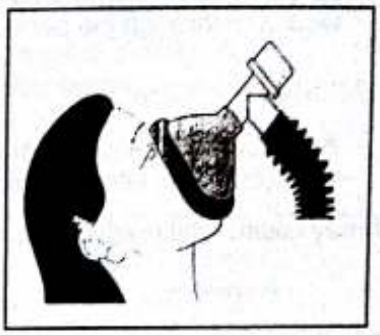


Fig 27:1 Inhalation analgesia

(II) REGIONAL ANALGESIA:

This is pain relief (nerve block) without loss of consciousness.

(a) Epidural Analgesia: It provides the most effective pain relief

- A plastic catheter is introduced into the epidural space (outside the dura) through a needle with a curved tip
- Intermittent doses of a local anaesthetic are injected through the catheter

Disadvantages:

- Motor block with weakness of the lower limbs. Decreasing the dose of the local anaesthetic and adding an opiate (e.g. fentanyl) spare the motor function (mobile epidural)
- Loss of the urge for straining increasing the need for forceps delivery.
- Accidental puncture of the dura.

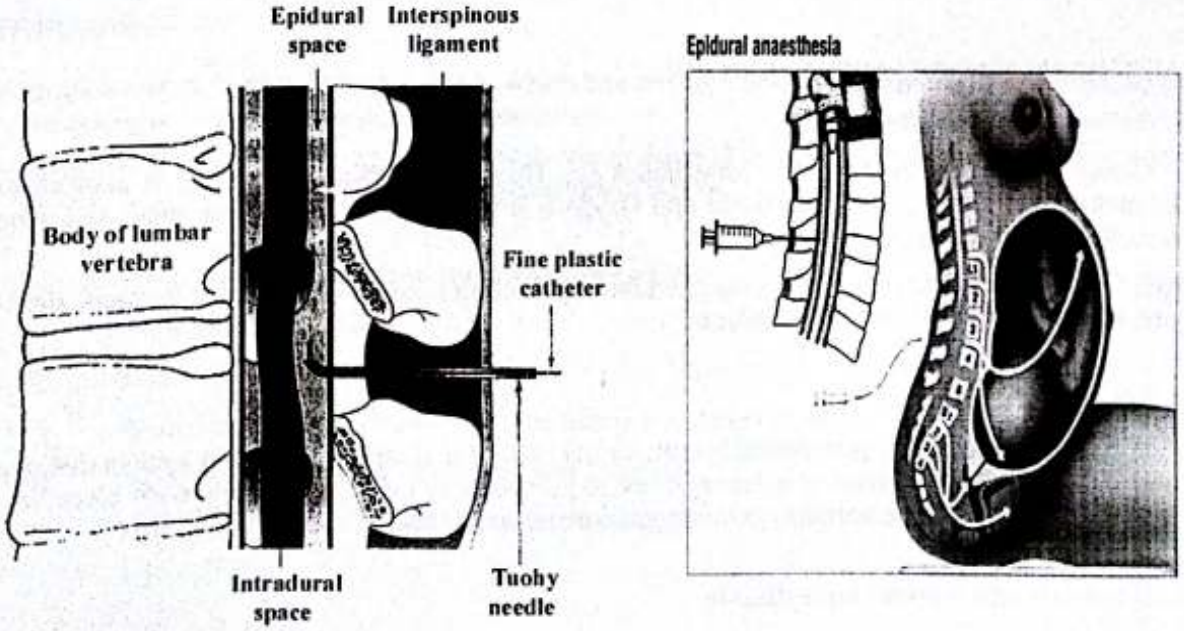


Fig 27:2 Regional Epidural Analgesia

(b) Local infiltration anaesthesia:

A local anaesthetic (e.g lidocaine) is injected at the site of episiotomy or perineal lacerations.

(c) Pudendal nerve block:

A local anaesthetic is injected near the ischial spine where the pudendal nerve crosses (either through the vagina or through the perineum).

(d) Paracervical block:

A local anaesthetic is injected on each side of the cervix through the lateral fornices.

It may cause fetal bradycardia, so rarely used nowadays.

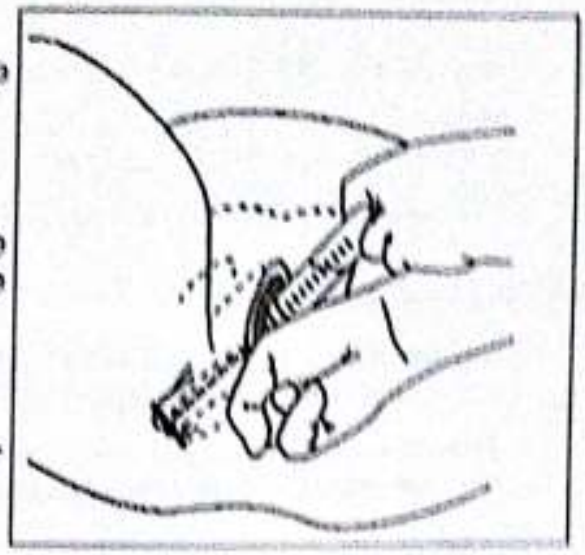


Fig 27.3 Pudendal nerve block

B) NON-PHARMACOLOGICAL METHODS for pain relief:

- **Prepared childbirth:**

  Educating the woman in antenatal classes about the birth process, how to relax, exercising and specific breathing patterns to be used during labour pains.

- **Trans Cutaneous Electrical Nerve Stimulation (TENS):**

  Application of a mild electric current to the skin can result in the reduction of pain.

  Stimulation of cutaneous nerve fibres closes a gate in the spinal cord preventing pain impulses transmission. Also the release of encephalins is a possible mode of action.

- **Acupuncture:** Acts through the gate theory.

- **Warm water baths (underwater birth):** heat is analgesic and the buoyancy of water is relaxing.

ANAESTHESIA FOR CESAREAN SECTION

A. General anaesthesia:

General anaesthesia using I.V. anesthetics (as Thiopental sodium or Ketalar) in addition to inhalation gas drugs (as Nitrous oxide and Oxygen) is safe for both mother and fetus and does not affect uterine contractions

NB: General anaesthesia may be also used for: repair of episiotomy, instrumental (forceps) delivery, and manual removal of the placenta

B. Epidural analgesia:

Is one of the best and safe methods both for the mother and fetus both during vaginal delivery and C.S. It has a slow onset of action and has to preceded by I.V. infusion to increase blood volume (preload) to avoid commonly occurring hypotensive episodes.

C. Subarachnoid (spinal) anaesthesia:

- The local anaesthetic is injected into the subarachnoid space
- It is considered nowadays as the first choice for C.S. analgesia for its strong and rapid action although in some cases it may cause hypotension and headache.

28 CONTRACTED PELVIS AND CEPHALOPELVIC DISPROPORTION

- *Definition and Aetiology*
- *Diagnosis*
- *Clinical pelvimetry*
- *Radiologic pelvimetry*
- *Cephalopelvic disproportion tests*
- *Maternal risks during labour*
- *Foetal risks during labour*
- *Management of labour in contracted pelvis*

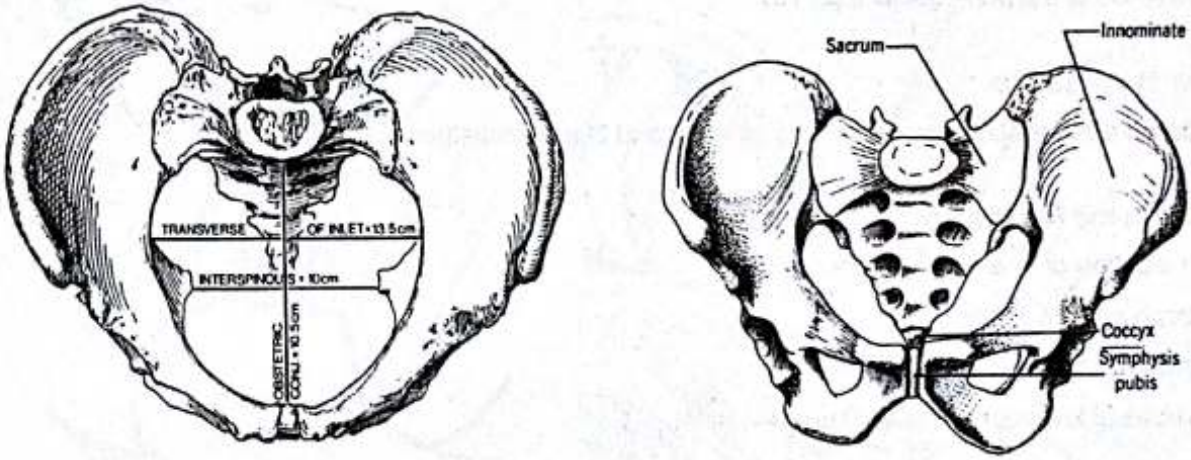


Fig 28:1 The normal gynaecoid pelvis

DEFINITION:

- **Anatomical definition:** It is a pelvis in which one or more of its main diameters are reduced below average normal by one or more centimetres.
- **Obstetric definition:** It is a pelvis in which one or more of its main diameters are reduced to the extent that interferes with the normal mechanism of labour.

Factors which affect the shape and the size of the pelvis:

1. Developmental factors
2. Sexual factors (male or female)
3. Racial factors (dark and white, Caucasians and Africans, etc.)
4. Nutritional factors (lack of calcium and proteins)
5. Metabolic diseases (as rickets and osteomalacia).

AETIOLOGY:

I. Causes in the pelvic bone:

A) Developmental causes (Abnormal shape):

- Small gynaecoid (generally contracted pelvis),
- Small android, small anthropoid, and small flat platypelloid pelvis.

B) Diseases of the pelvic bones and joints:

- Metabolic diseases:
  - *Rickets;* resulting into flat rachitic pelvis and generally contracted pelvis.
  - *Osteomalacia:* resulting into (triradiate pelvis).
- Fractures of the pelvic bones.
- Tumours of the pelvic bones.
- Diseases of the pelvic joints e.g.: T.B.

II. Causes in the spine:

- Dorso-lumbar scoliosis, Lumbar Kyphosis, and Spondylolisthesis.

III. Causes in the lower limbs:

- Dislocation of one or both femurs.
- Atrophy of one or both lower limbs.
- Unilateral fracture or tumour.
- Unilateral lower limb disease (poliomyelitis).

DIAGNOSIS OF CONTRACTED PELVIS

A) HISTORY: *Bad obstetric history* suggestive of contracted pelvis e.g.:

- Prolonged labour ending in C.S, foetal birth injury, or still birth (S.B.).
- Difficult forceps ending in S.B. or foetal birth injury.
- History of trauma or disease of pelvis, spine or lower limbs.

B) EXAMINATION:

1. ***General examination:***

- *Height:* Short stature < 150 cm, is commonly associated with a contracted pelvis.
- *Gait:* abnormal gait suggestive of diseases of the lower limb or spines.
- *Stigmata of old rickets:* as square head, pigeon chest, costal rosary, Harrison's sulcus, spine deformities and bow legs.
- *Dystrophia dystocia syndrome:* Short, obese, muscular appearance, male distribution of hair; may have an android pelvis (favouring occipito-posterior position).
- *Spines:* for deformities in the spines (scoliosis or kyphosis).
- *Lower limb:* for abnormalities.

2. ***Abdominal examination:*** for evidence suggesting contracted pelvis

- Malpresentations; as face, brow, breech and transverse lie.
- Non engagement of the foetal head in the last 3 or 4 weeks in a primigravida.

C) CLINICAL PELVIMETRY

1. ***External pelvimetry at the inlet:*** it has a little significance as it measures diameters of false pelvis.
2. ***External Pelvimetry at the outlet:***
   - *Sub-pubic angle:* direct palpation of the ischio-pubic rami (normally obtuse in females).
   - *Bituberous diameter:* Roughly admits the 4 knuckles of the closed fist or measured by the pelvimeter (11 cm).
   - *Anterior and posterior sagittal diameters:* Measured by Thom's pelvimeter.

NB: ***Thom's dictum:*** The sum of the bituberous and posterior sagittal diameters must exceed 15 cm to allow an average sized head to pass through the pelvic outlet provided that the bituberous diameter is more than 8 cm.

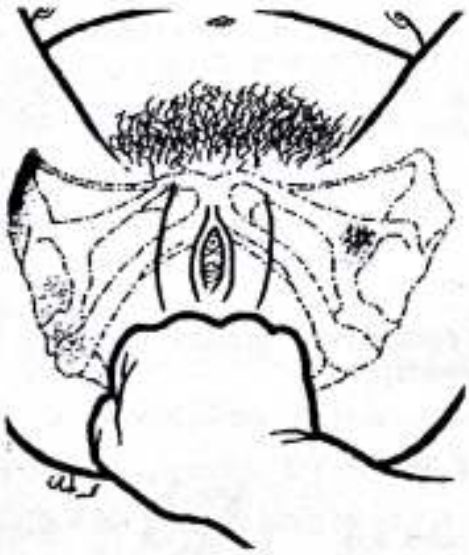


Fig 28:2 external pelvimetry of the outlet; the bituberous diameter

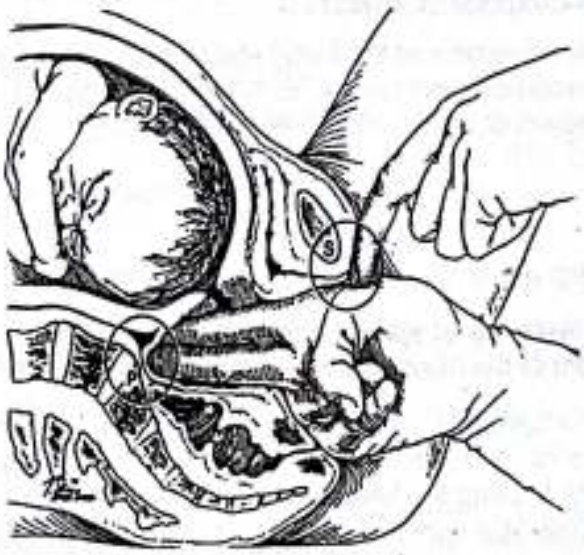


Fig 28:3 Internal pelvimetry of the inlet; The diagonal conjugate

3. ***Internal Pelvimetry: (Diagonal conjugate DC)***

   Measurement of the diagonal conjugate DC for clinical assessment of the pelvic inlet is done through a P.V. examination during ANC (at 38 weeks) or during labour;

   - To measure the diagonal conjugate the head must be *not engaged*.
   - DC lies between the lower border of the *symphysis pubis* and the *promontory of the sacrum*.
   - Normally the DC measures **12.5** cm (i.e.; 1.5 cm > true conjugate)
   - Subtracting **1.5** cm from the measurement of DC gives the length of the *true conjugate TC*
   - Normally the sacral promontory is not easily felt or reached.

- ***Palpation of the sacrum:*** normally it is concave with smooth concavity from above downwards and from side to side (there is no sudden bent).
- ***Palpation of the sidewalls of the pelvis:*** normally it is not converging.
- ***Estimation of the width of the Sacro sciatic notch:*** normally it accommodates 2 fingers.
- ***Palpation of the ischial spines:*** normally it is not felt when opening the index and middle fingers at the same time (not jutting)
- ***Palpation of the sub pubic angle:*** normally it accommodates 2 fingers.

D) RADIOLOGICAL PELVIMETRY:

- Lateral view X ray and C.T. scan pelvimetry can assess pelvic diameters accurately.
- The whole concept of pelvimetry has been replaced by the concept of cephalopelvic proportion and disproportion as a better judgement on the capability of the head to traverse the pelvis
- Nowadays radiologic pelvimetry is seldom resorted to except in very seelcted situations (e.g. if vaginal breech delivery is attempted), as it is expensive, and unavailable in many centres.

ULTRASOUND ASSESSMENT OF THE DIAMETERS OF FETAL HEAD

It is the most accurate method used for assessment of the size of the foetal head both during pre-ganncy or during the 1st stage of labor. It is accomplished by measuring the following diameters:

- Biparietal diameter, (BPD)
- Occipto-frontal diameter (OFD)
- Head circumference (HC).

N.B.: Measurement of the foetal abdominal circumference (AC) will allow in addition:
- Estimation of foetal weight (EFW) and diagnosis of a large for gestational age (LGA) foetus
- Estimation of HC/AC ratio may help in prediction of shoulder dystocia in a LGA foetus

CEPHALOPELVIC DISPROPORTION (CPD) TESTS

CPD depends upon the concept that ***"The head is the best pelvimeter for the pelvis"***

These tests are of special importance in evaluation of a primigravida, especially those with non engagement of the head near the end of 3rd trimester (> 36 weeks).

1. **Pinard's method:**
   - The patient is put in the semi-sitting position with the bladder empty, to bring the foetus in the axis of the pelvic inlet.
   - The operator right hand is placed over the symphysis pubis and the left hand grasps the fetal head and try to push it downwards and backwards in the pelvis.
   - The fingers of the right hand placed over the symphysis pubis can determine the degree of disproportion.

Fig 28:4 Muller-kerr test for CPD

2. **Muller-Kerr method:**
   - The patient is put in the dorsal position, with the bladder empty.
   - The index and the middle fingers of the right hand are put in the vagina to perform the steps of the internal pelvimetry and to detect the station of the head in the pelvis.
   - The thumb of the right hand is put over the SP to determine presence of any disproportion.
   - The head is grasped by the left hand and is pushed downwards into the pelvis.

Interpretation of the results of CPD tests:

A) **No disproportion:** if the head can be *pushed into the pelvis*. Vaginal delivery can usually occur

B) **Moderate disproportion** (1st degree disproportion):
   - The head does not enter the pelvis and stops at same level of the anterior surface of SP.
   - Vaginal delivery *may or may not occur* depending upon the undetermined factors of labour (moulding of the head and yielding of the pelvis).

C) **Marked disproportion** (2nd degree disproportion):
   - The head overrides the anterior surface of the S. pubis.This is usually found in cases with marked degree of *contracted pelvis*.
   - Vaginal delivery cannot occur.

MATERNAL RISKS DURING LABOUR IN CONTRACTED PELVIS

- Prolonged labour and slow dilatation of the cervix (abnormal progress of labour).
- Premature rupture of membranes and prolapse of cord.
- Obstructed labour (may end in rupture of the uterus).
- Higher incidence of instrumental and operative delivery.
- Postpartum haemorrhage (due to atony and lacerations).
- Maternal infection (prolonged labour and instrumental delivery).
- Necrotic genitourinary fistula.
- Rarely, injury of the joints or nerves from difficult instrumental delivery.

FETAL RISKS DURING LABOUR IN CONTRACTED PELVIS:

- Fetal birth injuries: as intra-cranial haemorrhage, fractures of the skull, nerve injuries, etc..
- Intrapartum and neonatal asphyxia.
- Prolapse of the cord, due to the high non engaged presenting part.
- Intra-amniotic infection, due to the prolonged early spontaneous ROM.

MANAGEMENT OF LABOUR IN CASES WITH CPD:

A) **Moderate degree of CPD** (1st degree)

- Trial of labour in selected cases (see case selection).
- C.S. if trial of labour fails or is contraindicated.

B) **Marked degree of CPD** (2nd degree)

- CS if the foetus is living (even if the foetus is dead CS is safer than performing a craniotomy).

TRIAL OF LABOUR (TOL)

It is a test of the undeterminable factors of labour in moderate degree of CPD. It is affected by:

- *Moulding* of the foetal head and *yielding* of the maternal pelvis.
- *Efficiency of uterine contractions* in causing *descent* of foetal head and cervical dilatation.

- **Selection of cases for TOL:**
  - Young healthy *primigravida*, *cephalic* presentation, with a *non engaged* head at onset of labour
  - Muller-kerr test reveals no more than *1st degree CPD*.
  - Cases with bad obstetric history, outlet contraction, and post-maturity are better excluded.
- **Conduct of labour in TOL:**
  - It must be in a ***hospital*** with available facilities for ***CS***.
  - Proper management of the 1st stage of labour: (see management of normal labour).
  - Proper assessment of the progress of labour by the use of ***partogram***.
  - Proper and adequate ***analgesia*** to avoid maternal exhaustion (e.g.: epidural analgesia).
- **Successful TOL:** ends by engagement of the head and safe vaginal delivery
- **Failed TOL:** is managed by C.S. Failed may be due to; failed progress of labour, foetal distress, or marked maternal exhaustion.

Failed progress of labour may occur due to:

- Improperly diagnosed 2nd degree CPD
- Failed long head rotation in OP positions
- Inccordinate uterine action (hypotonic or hypertonic uterine inertia)

INDICATIONS OF C.S IN CONTRACTED PELVIS AND CPD:

1. Marked disproportion if the foetus is living (2nd degree CPD).
2. Moderate disproportion if TOL is contraindicated or fails.
3. Markedly contracted outlet.
4. Contracted pelvis in elderly primigravida.
5. Contracted pelvis associated with complications as malpresentations, or placenta praevia.

CONTRACTED OUTLET "FUNNEL PELVIS":

- Definition: it is a variant of contracted pelvis in which the bituberous diameter is 8 cm or less.
- Features: The pelvic capacity is reduced from above downward.
  - The pelvis is narrow and deep,
  - Sidewalls are converging
  - Transverse diameter of the outlet is reduced
  - A.P. diameter of the outlet is reduced.
- Mechanism of labour: Extreme flexion and moulding occurs at the outlet with backwards displacement of the foetal head. N.B.; Contraction of the outlet interferes with long anterior rotation in O.P. positions.
- Management:

  - According to *"Thom's dictum"*, when the sum of the bituberous and the posterior sagittal diameters is > 15 cm, the bituberous is > 8 cm and the sub-pubic angle is not very narrow: a generous episiotomy is performed and low forceps may be applied.

  - If the sum of the bituberous and the posterior sagittal is <15 cm C.S. is performed.

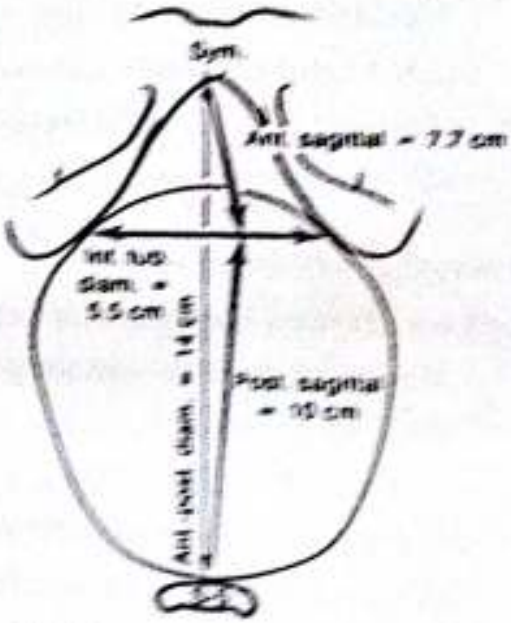


Fig 28.5 Contracted pelvic outlet (bituberous diameter 5.5) cm. Vaginal delivery is still possible due to the long posterior sagittal diameter (10 cm).

29 ABNORMAL UTERINE ACTION

Physiological uterine contractions
Classification of abnormal uterine action
1. Uterine Overactivity
- *Precipitate labour*
- *Obstructed labour*

2. Uterine Inertia
- *Hypotonic inertia*
- *Hypertonic inertia*

Contraction ring
Cervical dystocia

INTRODUCTION

Dystocia (difficult labour) is commonly associated with;

1. **Uterine dysfunction:** abnormal uterine action in which uterine forces are insufficiently strong or inappropriately coordinated to efface and dilate the cervix.
2. **Pelvic contraction:** see contracted pelvis
3. **Cephalo-pelvic disproportion (CPD):** associated with malpresentations or large fetal size.

PHYSIOLOGICAL UTERINE CONTRACTIONS

The physiological control of myometrial activity takes place through th ebalance between many factors including; Oestrogen (E), Progesterone (P), Oxytocin, Prostaglandins (PGLs), cyclic AMP, Calcium, and Beta 2 receptors.

Initiation of labour is explained by a change in balance between these factors, favoring an increase in uterine activity, with onset of labour.

During labour, the uterus exhibits waves of contractions beginning at the fundus, downwards to the lower uterine segment (LUS). Uterine contractions and retraction are paralleled with cervical effacement and dilatation, leading to descent of the presenting part through the pelvis and birth canal.

Assessment of uterine activity should include: Frequency, Amplitude, Duration, and resting tone of the uterine muscle.

- **Tocography** is a graphic recording of the previously mentioned factors
- **Cardiotocography:** is a graphic recording of these factors coupled with FHR.

Uterine work can be evaluated using Montevideo units. Montevideo units are calculated by subtracting the baseline uterine pressure from the peak of uterine contraction pressure for each contraction in a 10 minutes window and adding the pressures generated by each contraction.

CLASSIFICATION OF ABNORMALITIES OF THE UTERINE ACTION

I. Uterine Overactivity:
1. *Precipitate labor:* in absence of obstruction.
2. *Obstructed labor:* in presence of obstruction.

II. Uterine Inertia:
1. *Hypotonic inertia.*
2. *Hypertonic inertia:*

I. UTERINE OVERACTIVITY

1. PRECIPITATE LABOUR:

- It is a labor of duration less than **4 hours**
- It usually occurs due to **strong coordinate uterine contractions** in absence of obstruction in the birth canal, and resistance of the soft tissue of the cervix.
- The patient does not feel except the last contractions during the expulsion of the fetus.

Diagnosis:

- It is a retrospective diagnosis as the patient is usually seen in the 2nd or 3rd stages of labor.
- During the first stage of the labor; the partogram will show rapid progress of cervical dilatation and effacement.

Complications associated with precipitate labour

A. Maternal:

1. Lacerations of the cervix, vagina and perineum predisposing to: postpartum hemorrhage and sepsis which is also predisposed to due to delivery in unsuitable surroundings.
2. Atony: due to uterine exhaustion may lead to postpartum hemorrhage, retained placenta and inversion of the uterus.
3. Shock due to haemorrhage and/or pain.

B. Foetal:

1. Intracranial hemorrhage: due to rapid compression and decompression of the foetal head during delivery through the bony pelvis
2. Avulsion of the cord especially if short
3. Foetal distress due to excessive uterine contractions with short recovery period in between contractions leading to foetal hypoxia

Management:

1. Prophylaxis: A patient with past history of precipitate labor should be admitted to the hospital at the first perception of labor pains.
2. If seen during the short 1st stage of labour; slowing down the course of delivery and prevention of forcible bearing down, can be achieved using sedation and epidural analgesia.
3. If seen during the 2nd and 3rd stages: allow slow controlled delivery of the presenting part, avoid forcible bearing down and ensure complete expulsion of the placenta (inhalation analgesia via NO + O2 inhalation may be used)
4. After delivery: exploration of the birth canal for any injury and manage accordingly.
4. Prophylactic antibiotics if delivery occurred in unsuitable conditions
5. Proper examination of the fetus for detection of any complications.

2. EXCESSIVE UTERINE CONTRACTIONS AND RETRACTION (in presence of obstruction) :

- Excessive uterine contractions and retraction in presence of variable degrees of cephalo pelvic disproportion (CPD) and pelvic obstruction may result in:
- *The active upper uterine segment (UUS):* shows marked retraction and thickening
- *The passive lower uterine segment (LUS);* shows marked stretching and thinning
- *A groove appears* between the UUS and the LUS known as the **retraction ring,** that is seen and felt abdominally as a transverse groove rising with continuous retraction towards the level of the umbilicus. This pathological retraction ring is also known as Bandle's ring.
- Unless the obstruction is properly dealt with, the thinned out LUS will rupture.

II. UTERINE INERTIA

1. HYPOTONIC INERTIA:

Definition: Weak, infrequent and ineffective uterine contractions

Aetiology: Not known but the following factors may be associated:

1. *General factors:*
   a. Primigravida especially elderly.
   b. Anaemia, chronic illness, hypertensive states with pregnancy.
   d. Nervous, anxious patients.
   e. Improper use of analgesics.
2. Local factors:
   a. Over-distension of the uterus (e.g.: twins and polyhydramnios).
   b. Anomalies in development of the uterus (e.g.: unicornuate, bicornuate and septate uterus).
   c. Malpresentations and malpositions
   d. Full bladder or rectum.
   e. Uterine fibroids: Fibroids interfere with proper uterine contractions.
   f. Induction of preterm labour.

Classification:

- ***Primary inertia:*** poor uterine contractions from the start of labor.
- ***Secondary inertia:*** uterine contractions become weaker after a prolonged labour due to uterine exhaustion.

Clinical Picture:

1. Monitoring by **partogram**: reveals prolonged labour at various stages; (prolonged latent phase, protraction disorders, or arrest of cervical dilatation).
2. External monitoring by **toco-dynamometer**; a sensor on the abdomen will reveal infrequent contractions, with poor increase in the uterine tone, and short duration.
3. Abdominal examination by a hand on the fundus; reveals weak infrequent contractions < 3 in 10 minutes, each lasting less than 30 seconds.
4. The mother and the foetus are usually not seriously affected especially when the membranes remain intact, apart from prolonged labor.
5. If inertia persists after delivery of the fetus, there is liability for retention of the placenta (prolonged 3rd stage of labor) and atonic postpartum hemorrhage.

Complications due to prolonged labour:

A. Maternal:

- In the 1st stage: Nervousness, anxiety, exhaustion and starvation ketoacidosis.
- In the 2nd stage: increase liability for instrumental delivery and caesarean section.
- In the 3rd stage: retention of the placenta and postpartum hemorrhage

B. Foetal:
Usually no effect apart from fetal infection in cases with prolonged premature ROM.

Management of Hypotonic uterine inertia:

A) General measures:

1. Proper diagnosis that the patient is truly in the 1st stage of labor (not in the *pre-labour* stage) by proper identification of true labor pains (rhythmic, increase in strength, frequency and duration, accompanied by bulge of the bag of fore-waters, and cervical dilatation).
2. Exclusion of CPD and malpresentations; that are managed accordingly.
3. Proper management of the 1st stage of the labor: see normal labour

B) Uterine Stimulants: Oxytocin stimulation:

- **Aim**: To increase the strength, frequency and duration of the uterine contractions.

- Contraindications:
  - CPD, feto-pelvic disproportion in breech presentation, and malpresentations that may cause obstructed labour as shoulder, persistent brow, and face MP.
  - Incoordinate uterine action, grand multipara, and multifoetal pregnancies.
  - Uterine scar of previous C.S. or myomectomy (relative contraindication)
  - Foetal distress.

- Precautions;
  - *Close observation of FHR* by continuous monitoring to detect foetal distress elicited by significant repeated FHR decelerations especially at the end of contraction (late decelerations). If this occurs infusion is immediately stopped, case reevaluated, and C.S. should be considered.
  - *Oxytocin dose preparation and administration:* Dissolve 5 units in 500 ml of lactated ringer solution so 1 ml contains 10 m.IU of oxytocin. Adjustment of the dose via continuous infusion drip best using automatic computer adjusted infusion pump
  - *Assessment of efficiency of uterine contractions:* either clinically by a hand applied on the patient's abdomen, or better by electronic monitoring via toco-dynamometer (to detect frequency, regularity, duration and strength of contractions), and titrate the dose accordingly

- Operative interference:
  1. *Artificial ROM:* may be effective especially in cases of hydramnios (will relieve the overstretch of the uterine muscles).
  2. *Caesarean section:* if fetal distress occurs before full dilatation of the cervix
  3. *Instrumental delivery* by forceps or ventouse; in case of prolonged 2nd stage with early signs of maternal exhaustion or foetal distress

N.B.: *continue the drip for at least one hour (duration of fourth stage) after delivery of the fetus to guard against retained placenta and atonic postpartum hemorrhage.*

Secondary Hypotonic Inertia:

this condition usually follows prolonged labor with good uterine contractions which has failed to overcome obstruction to delivery in primigravida. Careful examination is needed to detect the cause of obstruction. CS is usually the solution.

2. HYPERTONIC INERTIA

Aetiology: *not known but the following may be associated:*

1. Anxiety.
2. Repeated rough manipulation.
3. Mal-use of oxytocin.
4. CPD, malpresentations and malpositions.

Clinical picture:

1. Painful contractions where pain precedes and outlasts the contractions, with low backache.
2. Irregular uterine contractions with high basal tone in between contractions. This can be best detected by external tocodynamometer, rather than by clinical examination.
3. Cervical effacement and dilatation are slow due to inefficient although strong uterine contractions (detected on partogram).
4. Early ROM is common due to increased intrauterine pressure, and non engagement of the presenting part.

Complications: As abnormal uterine action.

Treatment:

I. General measures: proper management of 1st stage with exclusion of CPD.

II. Specific management:

a. *Use of sedatives, antispasmodics, and epidural analgesia:* may be useful in controlling hypertonic inertia. Normal uterine action with progressive cervical dilatation may be regained and labour progresses normally.

b. *Caesarean section: Is indicated in:* cases of CPD, foetal distress occurs before full cervical dilatation, and in cases where analgesia fails to cause normal uterine action and progressive cervical dilatation.

CONTRACTION (CONSTRICTION) RING

Definition:

It is a persistent localized annular spasm of the uterine muscles, that occurs at any stage of labor, and at any part of the uterus, but usually at the junction of the upper and lower segments.

Aetiology: unknown, but may be associated with

1. Malpresentations and malposition.
2. Rough or repeated intrauterine manipulations (especially under light anaesthesia)
3. Improper use of uterine stimulants e.g. the use of oxytocin infusion in hypertonic inertia.

Diagnosis:

1. Contraction ring is usually preceded by colicky uterus mostly in primigravidas.
2. It is suspected if there is prolonged 2nd stage without any obvious cause.
3. It usually lies opposite the foetal neck, mostly at the junction between the upper and the LUS
4. It is diagnosed **only by PV examination:** by feeling it with a hand introduced inside the uterus.
5. It is suspected in the 3rd stage when it causes hour glass contraction of the uterus with retained placenta and postpartum hemorrhage.

Differential diagnosis: from pathologic retraction ring (see later in obstructed labour)

Management of Constriction ring:

1. Exclude disproportion, malpresentations and malposition.
2. Analgesics and antispasmodics, or better regional (epidural or spinal) anaesthesia.
3. In the 2nd stage, give deep general anaesthesia until constriction ring disappears, then:
   - Deliver the fetus immediately by forceps.
   - If the forceps fails or if the ring is below the presenting part, C.S. is performed
   - If the ring persists in spite of general anaesthesia, a vertical incision of the lower segment is needed to cut the ring.
4. In cases of retained placenta due to hour glass contraction of the uterus in the 3rd stage, give deep general anesthesia then perform manual removal of the placenta.

CERVICAL DYSTOCIA

Definition:

This is a difficulty in labor due to failure of cervical dilatation within a reasonable time in spite of the presence of strong, regular uterine contractions, i.e. no abnormal uterine action.

Types:

1. Organic rigidity (2ry):

a. Stenosis of the cervix by fibrosis following previous trauma or iatrogenic surgical trauma e.g.: cervical amputation, over cauterization, conization, and repeated cerclage procedures.

b. Organic obstruction of the cervix by cervical fibroid or carcinoma.

2. Functional rigidity (1ry):

It is non-dilatation of the external os of the cervix in absence of any organic lesion. The process affects the external os only, so the cervix may be well effaced and the head is well applied to it.

Clinically: The external os is felt as a hard rim.

Complications:

- Complications of prolonged labor and obstructed labor (maternal and foetal distress, rupture of the uterus, etc...if labor is neglected)
- Rarely annular detachment of the cervix may occur.

Management of Cervical Dystocia:

1. C.S. is indicated in most cases in association with:
   A) Cervical stenosis due to fibrosis; if cervix fails to dilate after a reasonable time.
   B) Organic obstruction of the cervix by myomata or pelvic masses
   C) Foetal distress or marked maternal exhaustion
2. In cases of functional rigidity of the cervix: giving time this cervix may dilate with good uterine contractions, together with use of analgesics and antispasmodics may be given.

30 OBSTRUCTED LABOUR

Awareness of the obstetrician about the potential complications of obstructed labour is of utmost importance. Missing the diagnosis, wrong or undue delay in the management or attempting unnecessary manipulations may lead to serious and even fatal complications to both the mother and the foetus.

DEFINITION

It is Failure of delivery of the foetus due to mechanical obstruction.

AETIOLOGY

A) Maternal causes:

1. Contracted pelvis.
2. Soft tissue obstruction e.g. fibroid, ovarian tumours impacted in Douglas pouch.
3. Cervical dystocia.
4. Other less common causes include: contraction ring, vaginal stricture or septum, vulval oedema, haematoma or neoplasms and rigid perineum.

B) Foetal causes:

1. *Macrosomia:* large foetus more than 4 kg. The most important causes are:
   - Constitutional
   - Maternal diabetes and Rh incompatibility
   - Post term pregnancy and
   - localized overgrowth e.g. hydrocephalus or abdominal tumours.
2. *Malpresentations* e.g.:
   - Persistent occipito-posterior.
   - Persistent mento-posterior.
   - Impacted breech.
   - Shoulder presentation (neglected).
3. *Shoulder dystocia:* difficulty in delivery of the shoulders which may be due to: macrosomia, broad shoulders e.g. maternal diabetes, short neck e.g. anencephaly and non-rotation of the shoulders.
4. *locked twins:* very rare (see multifoetal pregnancy).

CLINICAL PICTURE OF OBSTRUCTED LABOUR *"Impending rupture of the uterus"*

A) History:

Polonged labour, with *prolonged ROM* in spite of presence of *good* uterine contractions.

B) General examination:

- The patient is *exhausted* due to prolonged labour.
- Signs of *dehydration* are commonly evident; blood pressure is low, temperature is elevated, pulse is rapid, tongue and mucus membranes are dry.

C) Abdominal examination:

1. The *uterus* is hard and tender, contractions are rapid and strong.
2. Pathological *retraction ring* = BANDL'S RING is seen and felt as a transverse or oblique groove across the abdomen. The ring rises with time.
3. *Foetal parts* are difficult to palpate
4. *F.H.S.* are inaudible or show severe distress.

D) Vaginal examination:

- *The vulva* becomes oedematous, and the *vagina* becomes dry.
- *The cervix* feels oedamatous, **not** well applied on the presenting part unless fully dilated.
- *The presenting part* is **not** engaged, pelvic **caput succidanum** commonly develops in the foetal scalp in vertex presentation. This is of particular importance as.
- *The cause of obstruction* is determined: (Disproportion, Persistent OP, Neglected shoulder etc....)

N.B.: The presence of caput succidanum;

- May hide the sutures underneath, making determination of head position more difficult.
- May give a false impression about the level of the head in the pelvis (station). When the caput gets bigger it gives a false impression that the head is descending. This may encourage the unskilled obstetrician to wait for further "descent" or even apply forceps on a truly non engaged head with all its possible maternal and foetal hazards, including rupture of the uterus, cervical and perineal tears, and foetal birth injuries.

Differential diagnosis of pathological retraction ring:

1. Full bladder (excluded by catheterization).
2. Fundal fibroid (not rising - no signs of obstruction).
3. Contraction ring.

Pathological retraction ring	Contraction Ring
Occurs with prolonged second stage	*Occurs during any stage of labour*
Lies always between the Upper and LUS	*Occurs at any level but usually between Upper & LUS*
Rises up	*Does not change its position*
Felt and seen abdominally	*Not seen abdominally*
- Uterus is tonically retracted, and tender *- Foetal parts cannot be felt* *- Picture of impending rupture uterus*	*- The uterus is not tonically retracted* *- Foetal parts can be felt*
- The mother is distressed *- The foetus is distressed or dead*	*- The mother and the foetus are not necessarily distressed*
Relieved only by delivery	*May relax by antispasmodics or anaesthesia*

COMPLICATIONS OF OBSTRUCTED LABOUR

1. Maternal distress, exhaustion, and dehydration.
2. Foetal distress, hypoxia and neonatal asphyxia.
3. Prolonged rupture of membranes (with its complications).
4. Intra-amniotic infection.
5. Rupture uterus (overstretch of lower uterine segment).
6. Injuries of birth canal: cervical, vaginal and perineal lacerations.
7. Puerperal infection due to:
   - Prolonged labour with prolonged rupture of membranes.
   - Intrauterine manipulations and possible genital tract lacerations.
8. Necrotic obstetric vesico-vaginal fistula (due to prolonged head compression).
9. High perinatal mortality (neonatal asphyxia, foetal birth injuries, and sepsis).

MANAGEMENT OF OBSTRUCTED LABOUR:

- *Immediate C.S. with least possible manipulations is the safest choice*
- *Exploration of the birth canal* (under anaesthesia) is essential after any vaginal manipulation to exclude any traumatic lesion especially rupture uterus.
- *Forceps delivery* should not be attempted as it carries a high risk of complications especially rupture of the uterus.

Difficulties encountered during C.S. in obstructed labour: *(impending rupture uterus)*

- Extension of lower segment incision and subsequent injury of the uterine vessels
- Difficulty in extraction of the foetus due to impaction of the presenting part
- These problems could be avoided by dis-impaction of foetal head vaginally, making an adequate uterine incision and gentle extraction of the foetus.

SOFT TISSUE DYSTOCIA:

This may occur due to soft tissue obstruction as in cases of:

- Large subserous fibroids in the pouch of Douglas
- Ovarian tumours especial solid ones

Diagnosis:

- Clinical examination (abdominal and vaginal)
- Ultrasonography

Management :

C.S. is usually the best option

31 RUPTURE OF THE UTERUS And Cervical Lacerations

- *Incidence*
- *Risk factors*
- *Aetiology*
- *Rupture during pregnancy*
- *Rupture during labour*
- *Pathogenesis and mechanism*
- *Pathology*
- *Clinical picture*
- *Prevention*
- *Management of uterine rupture*

Rupture of the uterus is a potential obstetric catastrophe. It accounts for 20% of maternal mortality from haemorrhage in obstetric practice.

INCIDENCE

Varies from 1/2000 – 1/4000 deliveries. It depends greatly on the level of obstetric care, and is therefore more prevalent in developing countries with inadequate medical services, being an important factor contributing to maternal mortality.

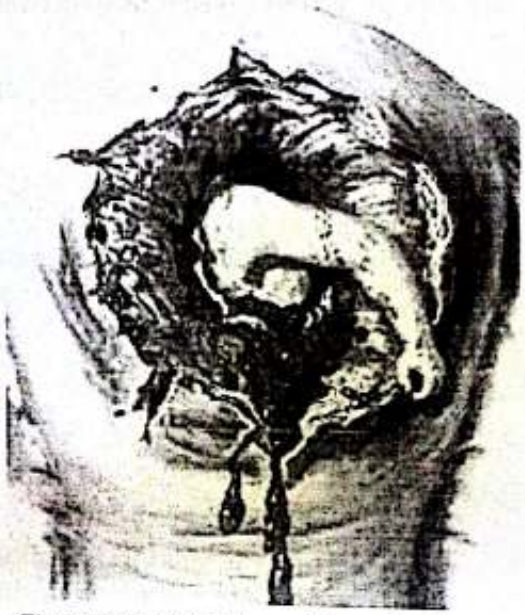


Fig 31:1 Rupture uterus through scar of upper segment C.S.

RISK FACTORS

1. Grand multipara (>90% of cases).
2. Presence of a uterine scar (C.S. > myomectomy).
3. Obstructed labour (as malpresentations and CPD).
4. Obstetric trauma (as with improper use of forceps).
5. Misuse of uterine stimulants (as PGLs & Oxytocin).

AETIOLOGY

A) Rupture During Pregnancy:

1. Spontaneous Rupture:

a. Scar of previous C.S.
- Classic upper segment C.S. scar. (1/3 of cases may rupture in late pregnancy)
- Lower segment C.S. scar (stronger, rarely ruptures during pregnancy).

b. Scar after gynecological operations (e.g. myomectomy, metroplasty, perforation).

2. Traumatic Rupture: *Rare, e.g. car accidents or fall from height.*

B) Rupture During Labour:

1. Spontaneous Rupture:

a. Preexisting uterine scar

b. Obstructed labour
- Malpresentations (face MP, brow, shoulder...)
- Feto pelvic disproportion (contracted pelvis, CPD, hydrocephalus...)

c. Improper use of oxytocin for augmentation of labour

d. Use of PGL E1 for augmentation of labour, e.g. Mesoprostol.

2. *Traumatic rupture:*

a. Forceps application before full cervical dilatation.
b. Breech extraction before fore cervical dilatation.
c. Internal podalic version procedures
d. Difficult manual removal of the placenta
e. Destructive operations
f. Excessive fundal pressure during the second stage

N.B.: In developed countries where rupture of the uterus is a rare event, most cases are due to rupture previous C.S. scar during a trial of vaginal birth after C.S. (VBACS)

In developing countries most cases are due to obstructed labour, inappropriate use of instrumental delivery, and abuse of prostaglandins and oxytocin.

PATHOGENESIS AND MECHANISM OF RUPTURE UTERUS

1. Rupture Uterine Scar: This depends on the following

a. Type of operation:

- C.S. scar is the commonest uterine scar to rupture because of the rising incidence of C.S. in general.
- Myomectomy scars: these are generally less liable to rupture than CS, except if they were multiple, and reaching the endometrial cavity.

b. Site of the scar:

- Classical or upper segment C.S. (USCS): rupture occurs in 4-9% of cases.
- Lower segment C.S. (LSCS): incidence of rupture is only around 0.2-1.5%.

c. Timing of rupture of scar:

- During pregnancy: more liability with U.S.C.S.
- During labour: more with L.S.C.S. especially in prolonged or obstructed labour, because U.S.C.S. is not allowed trial of vaginal delivery.

d. Predisposing factors for scar rupture:

- Improper coaptation of edges
- Improper haemostasis
- Postoperative infection
- Implantation of the placenta over the scar
- Over-distension of the uterus (twins, polyhydramnios...etc)
- Use of uterine stimulants in a scared uterus.

2. Rupture of an Unscarred Uterus:

A. Spontaneous Rupture:

It occurs mostly in cases of obstructed labour, associated with progressive thinning and stretching of the lower uterine segment. Rupture occurs usually late in labour, and affects the over stretched lower segment. It may be transverse or longitudinal, commonly on the left side due to dextro-rotation of the uterus.

B. Traumatic rupture:

It may occur with extension of a cervical tear upwards to the lower segment, commonly as a result of application of forceps before full cervical dilatation.

PATHOLOGY OF UTERINE RUPTURE

1. Complete rupture:
   Complete disruption of the entire myometrial thickness including the peritoneum.
2. Incomplete rupture:
   Rupture does not involve the visceral peritoneum over the uterus, which remain intact.

N.B.:

- ***Uterine dehiscence*** describes separation of a small part of a uterine scar with intact peritoneum and fetal membranes. It is usually asymptomatic.
- Rupture of lower segment may extend to involve the urinary bladder
- Lateral rupture may cause injury of uterine artery, or ureteric injuries.

CLINICAL PICTURE OF RUPTURE UTERUS

1. SPONTANEOUS RUPTURE:

It is usually preceded by clinical picture of *obstructed labour*

Symptoms and Signs:

- Sudden severe abdominal pain followed by cessation of uterine contractions
- Vaginal bleeding, that may be severe.
- General examination: signs of hypovolaemic shock if blood loss was severe.
- Abdominal examination:
  - Fetal parts may be easily felt just beneath abdominal wall muscles.
  - The fetus may take an abnormal attitude.
  - Marked fetal distress, severe bradycardia, or absent FHR if dead.
  - Signs of internal haemorrhage (abdominal wall tenderness and rigidity).
- Vaginal examination:
  - Recession and loss of station of the presenting part.
  - Excessive vaginal bleeding.
  - Site of rupture may be felt vaginally.

2. TRAUMATIC RUPTURE:

It should be suspected if after *difficult or instrumental delivery* the following occurs:

- Excessive vaginal bleeding and hypovolaemic shock develops after delivery
- Placenta is retained after delivery of the fetus, where manual removal reveals the rupture and its site.
- Diagnosis can be confirmed by manual exploration of the uterus including both the cervix and the uterine cavity.

N.B.: Some centres recommend routine exploration of the cervix and uterine cavity after difficult instrumental delivery and in vaginal birth after C.S. (VBACS).

PREVENTION OF UTERINE RUPTURE

Most cases of uterine rupture can be avoided by:

- Use of the partogram for early detection and proper management of obstructed labour
- Proper use of uterine stimulants (the indication, the dose, the rate and monitoring)
- Proper evaluation of patients with previous uterine scars before allowing trial of vaginal delivery (see vaginal birth after C.S. chapter 53).

MANAGEMENT OF RUPTURE UTERUS

Competent management of hypovolaemic shock is started while preparing for immediate laparotomy. The procedure done will depend upon:

a. The patient's general condition, amount of bleeding and state of shock.
b. The type, site, and extent of the rupture.
c. The patient's age, parity, and desire for further fertility.

1. Surgical Repair:

If the tear is limited, the general condition is fair and the patient is young in age demanding further childbearing, then surgical repair with re-suturing the torn muscles and arresting bleeding will be the optimum choice. N.B. Successful pregnancy may occur after repair, but delivery by C.S. will be mandatory at 37 weeks to avoid spontaneous rupture.

2. Abdominal hysterectomy:

It is the standard treatment whenever further childbearing is not wanted, or whenever the rupture is extensive, and blood loss is life threatening.

Fig 31:2 Rupture of lower uterine segment into broad ligament

Key points in rupture uterus

1. It accounts for 20% of maternal mortality from haemorrhage in obstetric practice.
2. Risk factors include Grand multipara, Presence of a uterine scar, Obstructed labour and Misuse of uterine stimulants.
3. Rupture may occur during pregnancy or labour either spontaneous or traumatic.
4. C.S. scar is the commonest uterine scar to rupture while Myomectomy scars are generally less liable to rupture than CS.
5. Spontaneous rupture is usually preceded by clinical picture of obstructed labour.
6. Traumatic rupture should be suspected if after difficult or instrumental delivery.
7. Most cases of uterine rupture can be avoided by proper Obstetric care.
8. Management of rupture uterus is by surgical repair when the tear is limited or by abdominal

CERVICAL LACERATIONS

INTRODUCTION

Obstetric genital trauma encompasses a spectrum of maternal injuries ranging from minor contusions to major lacerations.

Genital trauma may lead to extensive cosmetic and functional damage. The most common injuries at delivery are lacerations of the perineum and vagina, followed by cervical lacerations

Vulval and paravaginal hematomas are uncommon complications and the incidence varies from 1:300 to 1:7000 of deliveries depending on the anatomical region.

AETIOLOGY

1. Delivery of the head before full cervical dilatation:
   - After coming head in breech presentation.
   - Improper use of oxytocin and /or prostaglandins.
   - Faulty application of forceps or ventouse.
   - Some cases of precipitate labour
2. Manual dilatation of the cervix (wrong practice).

PREDISPOSING FACTORS:

1. Oedema of the cervix: as in cases of obstructed labor
2. Scarring and rigidity of the cervix: (excess fibrous tissue in the cervix):
   - Previous cervical cerclage
   - Previous operations on the cervix (e.g. cauterization, amputation, trachelorrhaphy)
3. Placenta praevia: due to marked vascularity of the lower segment and the cervix

CLINICAL PICTURE

Minor cervical lacerations are common in labor and may pass unnoticed. Extensive lacerations however lead to a picture of traumatic postpartum hemorrhage.

1. Excess bleeding during the third stage of labor.
2. Hypovolaemia and shock may occur
3. Uterus is well contracted (i.e. traumatic)
4. Examination of the cervix with good exposure and light will reveal the tear which may be:
   a. Unilateral, bilateral
   b. Annular detachment of the cervix: this type occurs due to prolonged compression of the cervix by the fetal head during prolonged labor, the external os becomes completely detached

COMPLICATIONS OF CERVICAL LACERATIONS

1. Hemorrhage, which is usually revealed and intrapartum or immediate postpartum.
2. Incompetent cervix
3. Infection, which may predispose to puerperal sepsis.
4. Ectropion; eversion of the edges with exposure of columnar epithelium (see gynaecology). It occurs due to bilateral injury with lateral cervical tear, fibrosis and retraction
5. Extension of the tear upwards into the lower segment or broad ligament leading to broad ligament haematoma

PREVENTION:

By avoiding the predisposing factors and proper management of labor

MANAGEMENT

1. Prophylaxis: by avoiding the predisposing factors and proper management of labor
2. Management of shock and blood transfusion may be needed
3. The cervix is exposed under good exposure and illumination grasped by sponge forceps and the tear is sutured with catgut or delayed absorbable sutures e.g. Vicryll or Dexon
   - Suturing should start above the apex of cervical tear to secure bleeding from retracted vessels.
   - Extension to the lower segment necessitates abdominal exploration by laparotomy, and management as rupture of the uterus.

a. and, due to ischemia, there is usually little bleeding.

Fig 31:3 Repair of cervical laceration

32 POST PARTUM HAEMORRHAGE

- *Definition & types of PPH*
- *Primary Post partum haemorrhage*
- *Incidence & Aetiology*
- *Placental site haemorrhage*
- *Lacerations of the genital tract*
- *DIC*
- *Clinical picture of PPH*
- *Complications of PPH*
- *Prevention*
- *Treatment of PPH*

Postpartum haemorrhage (PPH) is one of the leading causes of maternal mortality in Egypt, being responsible for nearly 34% of all maternal deaths. Proper obstetric care and management can prevent most cases of PPH and its complications, thus significantly reducing both maternal morbidity and mortality.

The potential for excessive blood loss after delivery occurs because the blood flow to the placental site is around 600 ml / min. This blood is controlled after delivery by myometrial contraction and retraction that constricts and occludes the opened vessels feeding the placental implantation site.

DEFINITION

PPH is defined as a blood loss > 500 c.c. after vaginal delivery, and > 1000 c.c. following C.S.

The proper estimation of the amount of blood loss after delivery may be difficult and inaccurate therefore the effect of blood loss on the patient's general condition is more important, as it can be affected not only by the amount, but also by the rate of blood loss and the general maternal health prior to delivery (anaemia, hypertension, APH, etc.).

TYPES OF PPH

1. **Primary PPH:**
   Immediate bleeding, or within first 24 hours, after delivery. It is the most important variety as it is associated with acute blood loss that may be life threatening.

2. **Secondary PPH:**
   Bleeding which is delayed > 24 hours, and till the end of puerperium. It is uncommon, the bleeding tends to be mild and chronic, and may even present as a gynaecologic problem.

PRIMARY POST PARTUM HAEMORRHAGE

INCIDENCE

The incidence of PPH varies from 0.5 - 4 % depending on the proper management of labour.

AETIOLOGY

In many cases bleeding may be due to combined factors being both atonic and traumatic, or either of them with associated DIC.

A) Placental Site Haemorrhage (Atonic PPH):

It is mainly due to failure of uterine contraction and retraction and accounts for 75 – 80% of cases of 1ry PPH. Risk factors predisposing to uterine atony are:

1. Over distension of the uterus (e.g. over sized baby, polyhydramnios and twins).
2. Prolonged labour (maternal exhaustion and dehydration)
3. Antepartum haemorrhage (placenta praevia and accidental haemorrhage).
4. Grand multiparity (lax and weak uterine muscles).
5. Precipitate labour (rapid delivery gives no time for efficient uterine retraction).
6. Nervous shock & full bladder lead to reflex atony of the uterus.
7. Retained separated placenta (partial or complete). In these cases the myometrium cannot contract and retract sufficiently due to presence of retained placental tissue.

B) Genital Tract Lacerations (Traumatic PPH):

As occur with rupture of the uterus, perineal, vaginal, or cervical lacerations

N.B.: Traumatic PPH is mostly caused by traumatic and operative vaginal deliveries.

C) DISSEMINATED INTRAVASCULAR COAGULOPATHY (DIC):

DIC and hypofibrinogenaemia can occur due; to placental abruption, retained IUFD, and amniotic fluid embolism (AFE).

CLINICAL PICTURE

A. History: *Ask for the presence of a risk factor*

- **Atonic PPH:** over distended uterus, multifetal pregnancy, polyhydramnios, etc.
- **Traumatic PPH:** traumatic or instrumental delivery.

B. General Examination: *Check for signs of hypovolaemic shock*

- Pallor, rapid weak pulse, low B.P., subnormal temperature, and oliguria.

C. Abdominal Examination: *To check the size and consistency of the uterus.*

- Atonic PPH is usually revealed, but may be partially or entirely concealed.
- In atonic PPH, palpation of the uterus reveals a soft consistency. The fundal level may be higher than expected if bleeding is partially concealed.
- In traumatic PPH, the uterus is firm, and vaginal bleeding continues in spite of a well contracted uterus. The cause of traumatic PPH should be confirmed by PV examination.

D. Vaginal Examination: *Preferably done under anaesthesia*

- To detect bleeding from a perineal, vaginal, or cervical laceration.
- To explore digitally the uterine cavity for retained parts, and for exclusion of uterine rupture.

COMPLICATIONS OF PPH

1. Maternal mortality (PPH represents about 34% of MMR in Egypt).
2. Haemorrhagic shock (due to excessive rapid blood loss, and possible DIC)
3. Acute renal failure (2ry to hypovolaemic shock).
4. Puerperal sepsis (2ry to low immunity and possible manipulations and retained products)
5. Sheehan's syndrome (hypo-pituitarism leading to 2ry amenorrhoea due to hypovolaemic shock)

PREVENTION OF PPH:

1. **Proper antenatal care (ANC):** *For identification of high risk factors for PPH as;*
   - Previous history of PPH
   - Grand multiparity (uterine muscle atony)
   - Hydramnios, twins, oversized fetus (over distension of uterine muscle).
   - Placenta praevia and placental abruption (causes of APH).
   - Correction of anaemia during pregnancy
2. **Proper management of the 1st and 2nd stages of labour:**
   - Avoid difficult and prolonged labour.
   - Avoid difficult and unnecessary instrumental delivery, especially if conditions are not suitable for safe applications.
3. **Proper management of the 3rd stage of labour:** *(The most important preventive measure)*
   - Active management of the 3rd stage; reduces the occurrence of PPH by nearly 50%.
   - Wait for signs of separation before delivery of the placenta. Attempts to express the placenta before its separation are dangerous.
   - Routine use of ecbolics after delivery, especially in high risk cases.
   - Intermittent uterine massage every 15 minutes, and continuous observation for the pulse, temperature, B.P., and vaginal bleeding, throughout the first two hours after delivery.

MANAGEMENT OF PRIMARY PPH

The goal should be to support life and arrest the bleeding. This may be achieved by the following successive steps in orderly sequence:

1. **Anti-shock measures** and blood transfusion, whenever necessary.
2. **Gentle uterine massage:** done by placing the thumb abdominally on the uterine fundus and the four fingers of the same hand behind to stimulate the uterus to contract.
3. **Ecbolics:** must be given with uterine massage. These include:
   - Oxytocin given as an I.V. drip (syntocinon); to increase the frequency and strength of uterine contraction. (It should never be given as direct I.V. bolus, as it may cause serious hypotension and arrhythmias.
   - Methyl ergometrin (methergin); 0.2–0.5 mg, I.M. or I.V., causes tetanic uterine contractions.
   - Mesoprostol (synthetic prostaglandin); given by rectal route, in a dose of 800 – 1000 ug.

4. If bleeding persists the following steps are activated:

   a. If the placenta was retained; it should be delivered immediately by controlled cord traction or manual removal.

   b. If the placenta was already delivered, then perform a vaginal exploration under anaesthesia to reveal:

      - Undiagnosed retained placenta fragments which should be removed, or
      - Vaginal or cervical lacerations that should be sutured and repaired.

   c. Bimanual compression of the uterus may be life saving until a laparotomy is performed.

5. If bleeding persists a Laparotomy is mandatory:

   a. Subtotal hysterectomy: is the standard procedure if bleeding is uncontrollable.

   b. Internal iliac artery ligation: may be attempted if the patient's general condition allows in an attempt to preserve the uterus, if the patient is young and desirous of further fertility. If this procedure fails to control the bleeding, hysterectomy is performed without hesitation.

N.B.: If PPH is due to DIC, treatment is by fresh frozen plasma, fresh blood transfusion, cryoprecipitate, fibrinogen, and platelet concentrate.

SECONDARY POST PARTUM HAEMORRHAGE

Definition: Bleeding which is delayed > 24 hours, and till the end of puerperium.

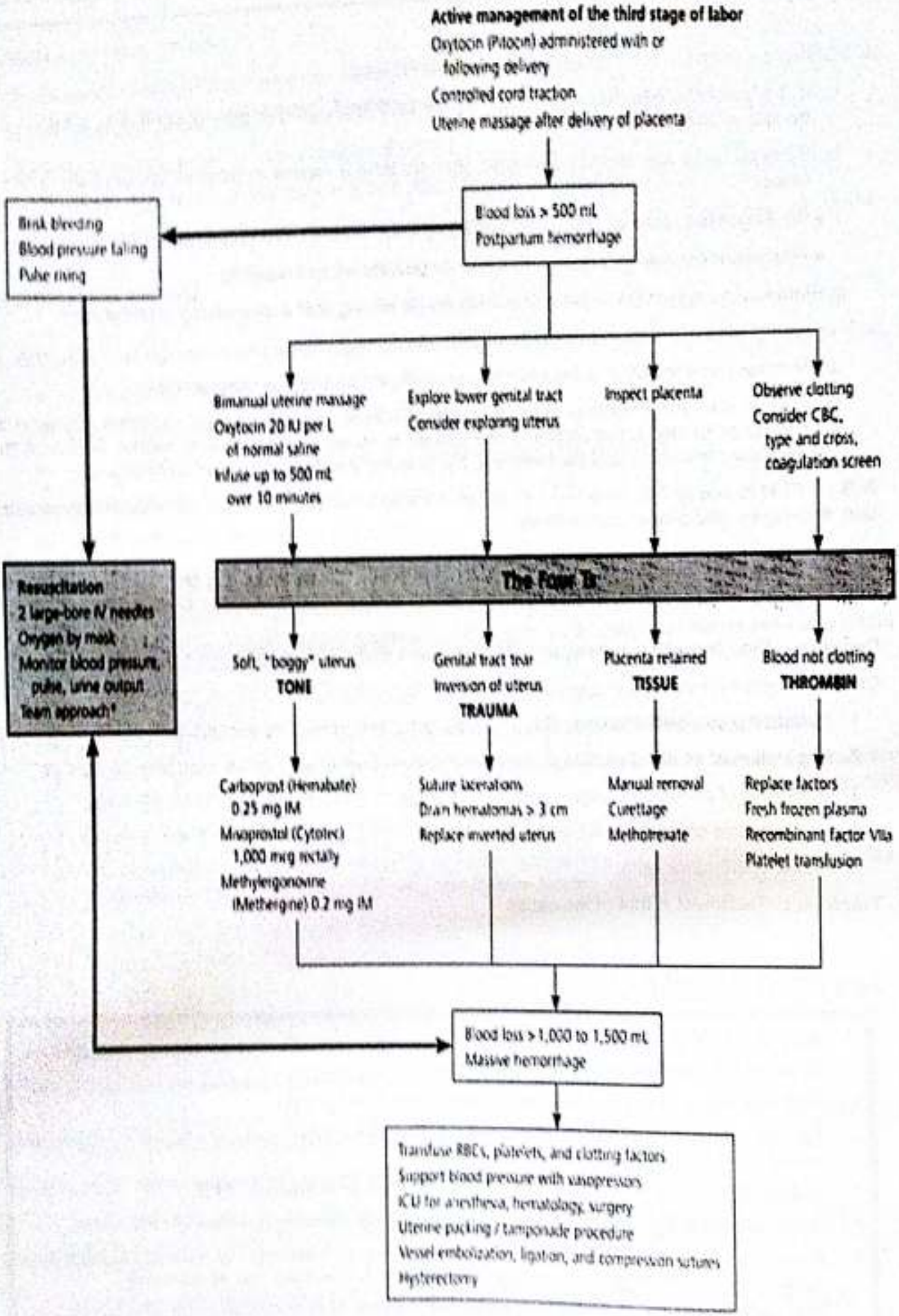
Causes:

1. Retained placental fragments; diagnosed by U.S., and treated by ecbolics and/or D&C.
2. Separation of an infected slough from a laceration in the lower genital tract; give antibiotics.
3. Sloughing of an infected submucous fibroid polyp.
4. Undiagnosed chronic uterine inversion.
5. Rarely choriocarcinoma.

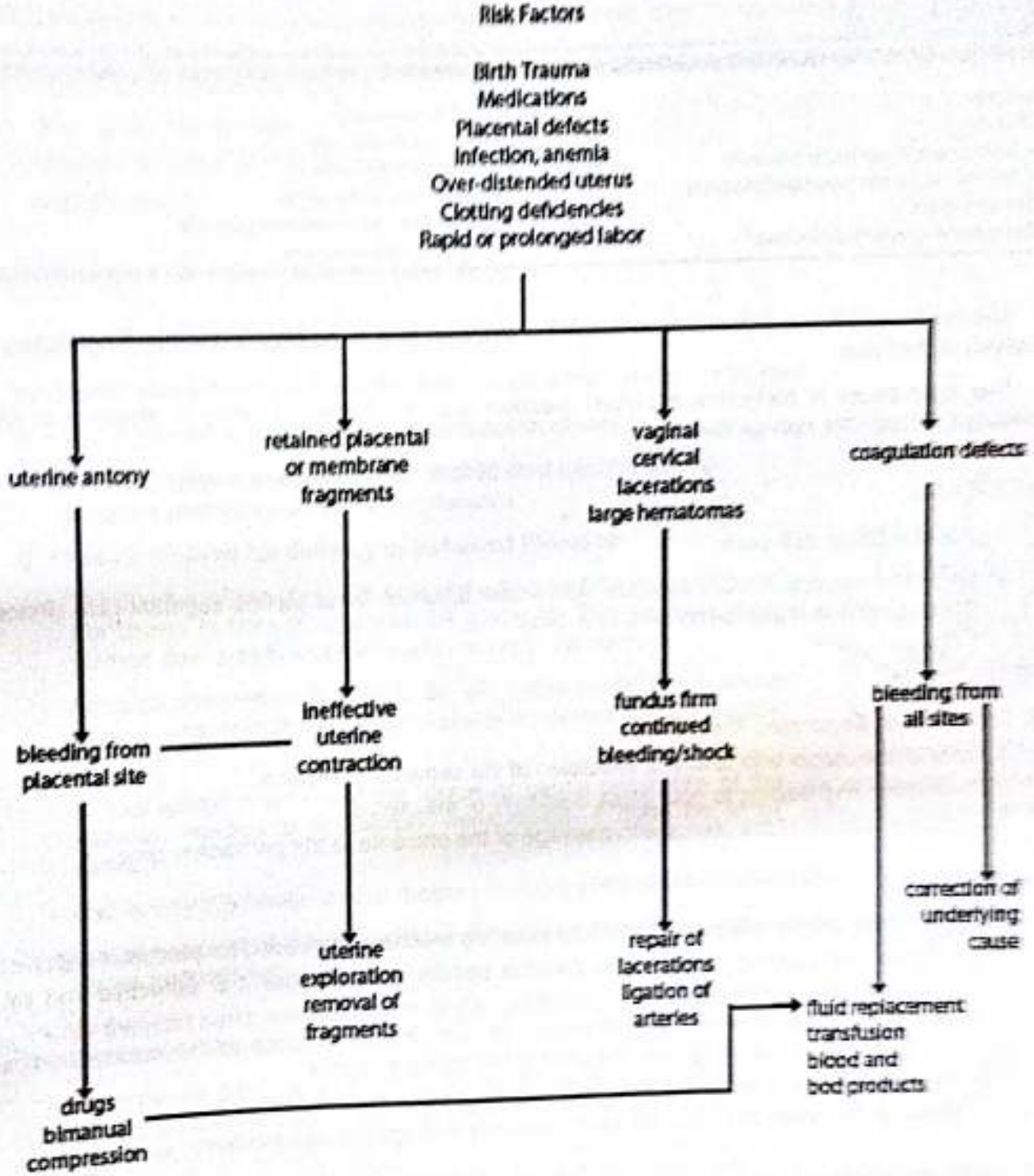
Treatment: Treatment is that of the cause.

KEY POINTS IN PPH

1. *PPH is defined as a blood loss in excess of 500 c.c. after vaginal delivery, and more than 1000 c.c. following C.S. and may be primary or secondary.*
2. *PPH is an important mostly preventable cause of maternal mortality*
3. *Uterine atony is the commonest cause for PPH .Genital tract lacerations or DIC are other possible causes.*
4. *Abdominal palpation of the uterus can differentiate atonic from traumatic PPH.*
5. *Proper management of the third stage of labour is very important in prevention of PPH.*
6. *First aid treatment of Ivy PPH is massage and ecbolics, with exclusion of retained placental fragments. If bleeding is severe and uncontrollable, subtotal hysterectomy may be life saving.*
7. *PPH cannot always be prevented for it occasionally occurs when conditions are in all respect normal*



POSTPARTUM HEMORRHAGE



33 RETAINED PLACENTA AND ACUTE UTERINE INVERSION

Retained placenta is a condition in which the placenta fails to be expelled within 30 minutes after delivery of the fetus.

The main cause is ***defective decidual reaction*** (decidua basalis) leading to absence of line of cleavage through the spongy layer.

INCIDENCE:

- About 0.5% of deliveries.
- Defective decidua is more common with implantation on lower uterine segment LUS (Placenta praevia) or with implantation on a C.S. Scar.

AETEIOLOGY:

A. Retention of Separated Placenta:

1. *Atony of the uterus* with failure of expulsion of the separated placenta.
2. *Contraction ring* leading to hour glass deformity of the uterus.
3. *Complete rupture of the uterus* with passage of the placenta to the peritoneal cavity.

B. Retention of Non-Separated placenta:

1. *Atony of the uterus:* leads to absence of shearing mechanism needed for placental separation.
2. *Defective placentation:* in which the decidua basalis is either absent or defective and so the chorionic villi penetrate the uterine muscles.
   - **Placenta accreta:** the placental chorionic villi are directly attached to the myometrium as a consequence of partial or total absence of the decidua basalis.
   - **Placenta Increta:** The chorionic villi actually invade the myometrium.
   - **Placenta Percreta:** chorionic villi even penetrate through the myometrium.

N.B.; the abnormal adherence of chorionic villi may involve;
- All of the cotyledons (total placenta accreta), or
- Few to several cotyledons (partial placental accreta), or
- A single cotyledon (focal placenta accreta).

CLINICAL PICTURE:

1. Vaginal bleeding: occurs only if part or the entire placenta is separated.
2. Uterine atony: the uterus is lax abdominally and if bleeding occurs it will be severe.
3. Severe shock : Retention of placenta more than 2 hours may cause shock even in absence of haemorrhage (Idiopathic obstetric shock)
4. Vaginal examination can detect:
   - Hour- glass contraction
   - Absence of plane of cleavage: placenta accreta
   - Rupture uterus.

MANAGEMENT OF RETAINED PLACENTA:

1. CASES OF UTERINE ATONY:

A. Gentle abdominal uterine massage: To stimulate uterine contraction.

B. Give ergometrin (I.M.): to ensure contraction of uterus

C. Brandt-Andrews maneuver: (Controlled cord traction and suprapubic pressure) to deliver the placenta.

D. Crede's method for delivery of Retained Placenta:

- The fundus of the uterus is grasped with four fingers behind and the thumb in front to squeeze the placenta. The fundus is then pushed downwards and backwards to expel the placenta.
- Although this method is easy to do, yet it may be difficult in obese patients, unsuccessful in cases of placenta accreta and contraction ring.
- It may cause inversion of the uterus or partial separation of the placenta, leading to postpartum hemorrhage, if done on a lax uterus.
- That is why it is rarely used in modern obstetric practice.

Fig 33:1 Crede's method for delivery of retained placenta placenta

D. Manual removal of the placenta: under general anesthesia, if the above methods fail:

- By the right hand, follow the cord to the placenta and then pass the hand to its lower edge.
- Separate the placenta completely, by a sawing movement from side to side, while the fundus is steadied by the left hand on the abdomen.
- Grasp the placenta and deliver it out and inspect it to detect any missing fragment.

Fig 33:2 Manual separation of the placenta

- Give ergometrin and massage the uterus.
- This operation must be done carefully otherwise you may perforate the uterus.
- Strict asepsis and prophylactic antibiotics are given to guard against puerperal sepsis.

2. CASES OF CONTRACTION RING:

Give the patient **deep general anesthesia**, and then do manual removal of the placenta.

3. CASES OF ADHERENT PLACENTA

- In cases of simple adhesion or partial placenta accreta; manual separation and removal of the placenta is done.
- In cases of placenta complete accreta either:
  - Abdominal Hysterectomy: as a life saving procedure in cases with shock and severe hemorrhage, especially multiparous patients.
  - In young patients and in primigravidas; the placenta may be left in situ to undergo autolysis changes after cutting the cord short. Antibiotics and thorough observation are essential. Such a management is unsafe and may hazardous to many patients.

4. IN CASE OF RUPTURE UTERUS:

- Laparotomy is performed after administrating blood transfusion and antishock measures.
- Placenta is removed from the peritoneal cavity
- The uterus is repaired (in non extensive tears) to preserve the patient's fertility.
- Subtotal hysterectomy may be life saving if rupture was extensive with poor patient's general condition.

COMPLICATIONS OF RETAINED PLACENTA:

1. Shock: haemorrhagic or idiopathic obstetric shock.
2. Postpartum hemorrhage.
3. Puerperal sepsis
4. Subinvolution of the uterus.
5. Retained parts of the placenta may later form a placental polyp and give rise to chorio-carcinoma.
6. Complications of the method done to deliver the placenta and complications of anesthesia.

Key points in retained placenta

1. The placenta fails to be expelled within 30 minutes after delivery.

*2. The main cause is **defective decidual reaction** (decidua basalis).*

3. Defective placentation and abnormal adhesions may be Placenta accreta, Placenta Increta and Placenta Percreta.

4. Complications include Shock, Postpartum hemorrhage, Puerperal sepsis and Retained parts of the placenta.

5. Management according to aetiology either by gentle massage , ecbolics .controlled cord traction and manual removal in cases of atony or inhalation anesthesia in contraction ring or laparotomy in abnormal adhesions.

ACUTE INVERSION OF THE UTERUS

DEFINITION

The uterus is partially or completely turned inside out after delivery of the neonate.

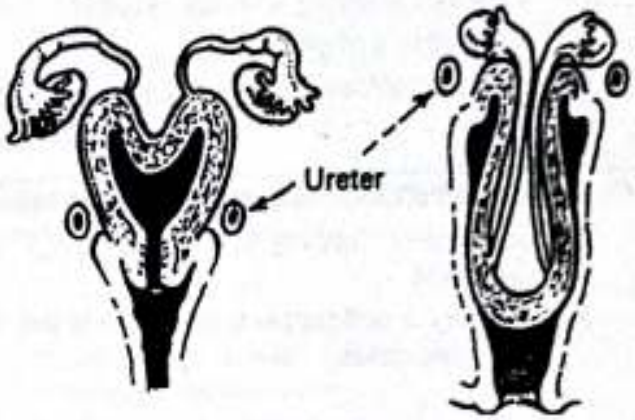


Fig 33:3 acute inversion of the uterus

INCIDENCE

1:2500-6500.

AETIOLOGY

A) Induced Inversion:

1. Vigorous pressure on the fundus (a complication of Crede's method).
2. Traction on the cord especially if the placenta is abnormally adherent.
3. during manual removal of the placenta (can also occur during CS).

Spontaneous:

1. Precipitate labor.
2. Traction by the fetus on a very short cord.
3. Submucous fundal fibroid.
4. Vigorous straining or coughing.

Fig 33:4 submucus fibroid polyp causing acute inversion

Degrees of Uterine Inversion

- *1st degree:* the fundus uteri is depressed but does not pass through the cervix
- *2nd degree:* the fundus protrudes through the cervix
- *3rd degree:* total uterine inversion, dragging on the vagina, protruding outside the vulva

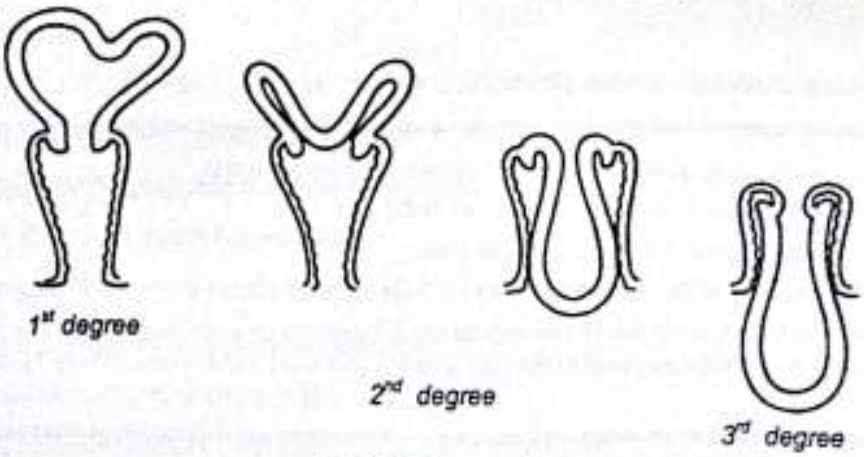


Fig 33:5 degrees of acute inversion of the uterus

CLINICAL PICTURE

Symptoms:

- Pain in lower abdomen
- Vaginal bleeding (usually massive).
- Symptoms of shock and
- Mass protruding from the vulva in 3rd degree cases.

Signs:

1. Profound shock due to massive blood loss, and traction on the peritoneum and adnexa
2. Abdominally: cupping of the fundus in 1st and 2nd degree cases and absent uterus in 3rd degree cases.
3. Vaginally: a soft purple mass is felt in the vagina (in 2nd degree cases) or seen at the vulva (in 3rd degree cases).

MANAGEMENT OF ACUTE INVERSION

- **Prophylaxis:** Avoid vigorous manipulations on a lax uterus or traction on the cord of an abnormally adherent placenta.
- **Definitive treatment:**

1. Two wide bore IV cannulas are inserted. Blood sample is sent for cross-matching. Ringer's lactate IV infusion is started. Plasma substitutes can be also used. The inverted uterus is replaced in the vagina.
2. Under general anesthesia (preferably halothane to relax the uterus), the inverted uterus is repushed out manually with the palm of the hand and the fingers in the direction of the long axis of the vagina. Tocolytic drugs as terbutaline, nitrites or magnesium sulfate can be used to relax the uterus to facilitate reposition.
3. If the placenta is still attached, it is removed to facilitate reposition of the uterus.
4. When normal uterine configuration is restored, the tocolytic agent is stopped and syntocinon infusion is started while maintaining the uterus in position till it contracts.

N.B.:

- Some cases (as hydatidiform) possess for reposition of the uterus. In a minority of patients the abdominal approaches fail and laparotomy for surgical correction is needed.
- Rarely, the condition is discovered later in the puerperium as a **subacute case** presenting by secondary PPH and offensive vaginal discharge or as a **chronic case** presenting by a mass protruding per vagina (PV) (uterine prolapse or fibroid polyp).

Key points in acute inversion of the uterus

1. The uterus is partially or completely turned inside out after delivery.
2. The uterine inversion is either spontaneous or induced.
3. There degrees are present 1st, 2nd, & 3rd degree.
4. Abdominally: cupping of the fundus in 1st and 2nd degree and absent uterus in 3rd degree.
5. Vaginally soft purple mass is felt in the vagina (in 2nd degree) or seen at the vulva (in 3rd degree).
6. Management is by prophylaxis and reduction under anesthesia either manually or by hydrostatic reduction.

34 SHOCK AND HYPOFIBRINOGENAEMIA IN OBSTETRICS

- *Definition*
- *Types and Aetiology*
- *Clinical picture*
- *Hypovolaemic shock*
- *Classification*
- *Pathophysiology*
- *Management*
- *Septicaemic shock*
- *Cardiogenic shock*
- *Neurogenic shock*

Hypofibrinogenaemia

- *Definition and mechanism*
- *Clinical picture & diagnosis*
- *Treatment*

DEFINITION

Shock: *is a state of circulatory impairment characterized by defective tissue perfusion resulting in abnormal cellular function and metabolism.*

This leads to a clinical syndrome of signs of decreased perfusion of vital organs, with possible alterations in the mental status (somnolence) and oliguria (urine output <30 ml/Hr.)

TYPES AND AETIOLOGY OF SHOCK

1. Hypovolaemic shock: secondary to
   - Bleeding (various causes of bleeding in early pregnancy, ante- or postpartum hemorrhage).
   - Other causes of fluid loss (e.g. naso-gastric suction or diarrhea).
2. Distributive shock: secondary to increased venous pooling (i.e. early septic shock, peritonitis, anaphylaxis and neurogenic shock).
3. Cardiogenic shock: secondary to decreased myocardial contractility and function (as in myocardial infarction).
4. Obstructive shock: secondary to mechanical obstruction (i.e. cardiac tamponade, massive pulmonary embolism or thrombosed prosthetic valve).

CLINICAL PICTURE

1. Hypotension (a BP decrease of 50-60 mmHg or BP <100 mmHg) & subnormal temperature.
2. Tachypnea & tachycardia (weak rapid -thready- pulse).
3. Pallor, cyanosis of fingers and cold clammy sweat.
4. Dimness of vision and mental confusion.
5. Oliguria.

Remote complications of shock in obstetrics:

1. Renal failure (due to cortical necrosis).
2. Postpartum anterior pituitary necrosis (Sheehan syndrome).

HYPOVOLAEMIC OR HEMORRHAGIC SHOCK

A healthy pregnant woman can lose 25% of her blood volume (1500ml) before clinical signs of shock are evident.

Conditions that predispose to the development of shock in obstetrics include:

a) Anemia and malnutrition

b) Bleeding in early pregnancy, APH, or PPH

c) Prolonged labor with dehydration and acidosis

d) Hypertensive states with pregnancy, especially PE and eclampsia.

Classification of shock based on extent of blood loss:

Parameter	Class I	Class II	Class III	Class IV
Blood volume lost (%)	*<15*	*15-30*	*30-40*	*>40*
Pulse rate (beats/min)	*<100*	*>100*	*>120*	*>140*
Supine blood pressure	*Normal*	*Normal*	*Decreased*	*Decreased*
Urine output (ml/hr)	*>30*	*20-30*	*5-15*	*<5*
Mental status	*Anxious*	*Agitated*	*Confused*	*Lethargic*

PATHOPHYSIOLOGY OF SHOCK:

A) Compensatory Mechanisms: in early stages of shock

- **Arterioles:** they control capillary blood flow in various organs. They are resistance vessels controlled by the CNS.
- **Venules:** they contain 70% of the total blood volume. They are passive resistance vessels controlled by humoral factors.
- Catecholamine release during hemorrhage causes increased venular tone resulting in auto transfusion from this capacitance reservoir.
- There is compensatory increase in heart rate, systemic and pulmonary vascular resistance, and myocardial contractility.
- There is redistribution of cardiac output & blood volume by selective centrally mediated arteriolar constriction resulting in decreased blood flow to kidneys, splanchnic bed, uterus and skin with relative maintenance of blood flow to the heart, brain & adrenal glands (organs that auto regulate their own flow).

B) Decompensation: when blood volume deficit exceeds 25%.

- Rapid clinical deterioration results from maldistribution of blood flow that causes local tissue hypoxia & metabolic acidosis, producing a vicious circle of vasoconstriction, organ ischemia and cellular death.
- Loss of capillary membrane integrity.
- Increased platelet aggregation resulting in small vessel occlusion.
- Electrolyte shifts: Na^+ & H2O enter skeletal muscles and cellular K^+ is lost to the extracellular space.

MANAGEMENT OF HYPOVOLAEMIC SHOCK

A) General measures:

1. Adequate ventilation providing oxygen by mask, nasal tube or tracheal intubation if needed.
2. Insertion of two wide bore cannula with blood sample collection for blood grouping, Rh & cross-matching, CBC, electrolytes, liver & kidney function tests, blood sugar and coagulation profile (PT, PTT, fibrinogen & FDPs).
3. Warmth, recumbent position with legs slightly elevated.
4. Morphine to alleviate pain and apprehension if needed.

B) Fluid, blood and blood component replacement:

1. Crystalloid solutions as lactated Ringer's solution or normal saline (basic therapy for acute hemorrhage is crystalloid and blood).
2. Colloid therapy (as plasma substitutes) will provide more volume expansion than crystalloids.
3. Whole blood: only used in torrential bleeding.
4. Packed RBCs are usually used.
5. Blood component replacement is rarely necessary with acute component replacement of 5-10 packed RBCs or less. Transfusion is needed when Hb concentration < 8 g / dL or HT <25 %.
6. Red cell substitutes: still under research.

Blood components commonly transfused in obstetrics:

Product	Indication	Content	Effect
Whole blood (450 ml)	*Symptomatic anemia with large volume deficits*	*All components*	*Increases Ht 3-4 volume % per unit*
Packed RBCs (250 ml)	*Symptomatic anemia*	*Erythrocytes*	*Increases Ht 3-4 volume % per unit*
Fresh frozen plasma (FFP 250 ml)	*Deficit of labile and stable coagulation factors*	*All clotting factors*	*Supplies fibrinogen 150 mg per unit and other factors*
Cryoprecipitate (50 ml)	*Hypofibrinogenaemia*	*Factors VIII, VWF, XIII, fibronectin, and fibrinogen*	*Supplies selected clotting factors*
Platelets (50 ml)	*Bleeding from thrombocytopenia*	*Platelets*	*Increases platelet count by 5000-8000/µl per unit*

C) Patient Monitoring:

For vital signs (P, T, BP, RR), urine output, central venous pressure (CVP), pulmonary artery pressure (by Swan-Ganz catheter in selected patients) and repeat laboratory investigations.

D) Vasoactive and Inotropic Agents:

- **Vasoactive drugs;** induce vasoconstriction and increase BP (e.g.; Epinephrine & nor epinephrine)
- **Inotropic drugs:** improve cardiac muscle contractility (e.g.; Dopamine & Dobutamine)

SEPTICEMIC SHOCK

AETIOLOGY:

As pelvic infection is polymicrobial, septic shock may be caused by;

1. Endotoxin producing entero-bacteriaceae family especially E. coli (most common).
2. Aerobic and anaerobic Streptococci (less common)
3. Bacteroids and Clostridium species (uncommon).
4. Exotoxin producing Group A ß-hemolytic streptococci and also Staphylococcus aureus may also be the cause.

PATHOPHYSIOLOGY

Bacterial toxins result in mediator release with:

- Activation of complement, kinins and the coagulation system causing DIC & induction of fibrinolytic state with bleeding.
- Selective vasodilatation with maldistribution of blood flow.
- Leukocyte & platelet aggregation causing capillary plugging.
- Vascular endothelial injury causing profound capillary leakage.
- Early septic shock is a form of distributive shock while in late stages it is both distributive and cardiogenic. The end result is septic shock syndrome with multiple organ failure.

CLINICAL PICTURE

Passes into 3 stages of increasing severity:

- Systemic inflammatory response syndrome (SIRS)
- Severe sepsis, then
- Septic shock.

Multiple organ effects with sepsis and shock:

System		Effect
CNS	*Cerebral*	*Confusion, somnolence, coma and combativeness*
	Hypothalamus	*Fever, hypothermia*
CVS	*BP*	*Hypotension (vasodilatation)*
	Cardiac	*Increased cardiac output (early), myocardial depression (late), tachyarrhythmia*
Pulmonary		*Shunting with hypoxemia, diffuse infiltrates (capillary leak)*
Renal		*Hypoperfusion (oliguria), acute tubular necrosis*
Haematological		*Thrombocytopaenia, Leucocytosis, DIC*

TREATMENT OF SEPTIC SHOCK

1. Aggressive fluid replacement. Oxygenation and ventilation.
2. Administration of Vasopressor and Inotropic agents (see hypovolaemic shock).
3. Broad spectrum antibiotics.
4. Removal of the infectious source.

N.B. Steroids and NSAID: are not beneficial.

Immunotherapy is still under research.

CARDIOGENIC SHOCK

Can also occur in the setting of septic shock or hemorrhagic shock, especially in patients who have baseline cardiovascular disease. Treatment requires invasive monitoring and dealing with the underlying disorder.

NEUROGENIC SHOCK

AETIOLOGY: trauma and tissue damage as in cases of:

1. Disturbed extrauterine pregnancy.
2. Concealed accidental hemorrhage.
3. Difficult forceps delivery or breech extraction (especially if the cervix isn't fully dilated).
4. Difficult internal version.
5. Repeat rough attempts at Crede's method.
6. Rupture of the uterus or cervical tears extending into the lower uterine segment.
7. Acute inversion of the uterus.
8. Rapid evacuation of the uterus as in precipitate labor and polyhydramnios
9. Retained placenta especially for more than 2 hours.

Differences between neurogenic and hemorrhagic shock:

Neurogenic shock	Hemorrhagic shock
The patient is quiet and apathetic	*The patient is restless and anxious with air hunger*
No external or internal bleeding	*External or internal bleeding*
Superficial veins are full of blood	*Superficial veins are collapsed*
Haemoconcentration	*Haemodilution*
Slow pulse	*Weak and rapid pulse*
Slow and shallow respiration	*Rapid and shallow respiration*

TREATMENT

1. General measure: mentioned earlier.
2. Fluid replacement.
3. Vasopressor and inotropic agents.
4. Dealing with the cause.

Key points in Obstetric shock

1. Types include Hypovolaemic, Distributive shock, Cardiogenic shock and Obstructive shock.
2. Remote complications of shock in obstetrics include Renal failure and Sheehan syndrome.
3. Crystalloid solutions as lactated Ringer's solution or normal saline for acute hemorrhage.
4. Colloid therapy (as plasma substitutes) will provide more volume expansion than crystalloids.
5. Packed RBCs are usually used in Obstetric hemorrhagic shock.
6. Early septic shock is a form of distributive shock while in late stages it is both distributive and cardiogenic.
7. Clinically septic shock Passes into 3 stages of increasing severity namely systemic inflammatory response syndrome (SIRS), severe sepsis then septic shock.

HYPOFIBRINOGENEMIA

Disseminated Intravascular Coagulation (DIC) or Consumptive Coagulopathy.

PHYSIOLOGICAL BACKGROUND

In late pregnancy, there is increased concentration of coagulation factors I (fibrinogen: level is 350-650 mg/dL), VII,VIII,IX and X.

Other plasma factors and platelet count (150 000-400 000/cmm) do not change remarkably.

DEFINITION

A widespread hematological condition characterized by *accelerated fibrin formation and lyses.*

- There is consumption of platelets and coagulation factors in variable quantities.
- Signs of hypofibrinogenaemia develop when its level goes below 100 mg/dL

ETIOLOGY: Pregnancy related

Common causes

1. Massive blood loss with inadequate replacement. Massive crystalloid or colloid replacement
2. Placental abruption.
3. Severe pre-eclampsia/eclampsia or HELLP syndrome.

Rare cause

1. Sepsis.
2. Retained dead fetus (for more than 3-4 weeks).
3. Amniotic fluid embolism.
4. Acute fatty liver of pregnancy.
5. Adult RDS, acute hemolytic transfusion reactions, autoimmune disease, hematological malignancies and solid tumors.

MECHANISM

- ***Accelerated coagulation:*** occurs via the extrinsic pathway (thromboplastin from tissue destruction) or the intrinsic pathway (collagen and other tissue components when endothelial integrity is lost). Finally factor X (Prothrombinase) is activated.
  - The formed thrombin (activated factor II) changes fibrinogen (factor I) to fibrin (monomers and polymers-clot-).
  - Factor X can be activated directly by proteases present in mucin of amniotic fluid or neoplasms.
- ***Fibrinolysis:*** the fibrin monomers combine with tissue plasminogen activator and plasminogen which release plasmin. Plasmin lyses the fibrin mono and polymers to form a series of fibrin degradation products (FDPs) including the D-dimer.

CLINICAL PICTURE

1. Postpartum or antepartum hemorrhage.
2. Persistent bleeding from venipuncture sites or after catheter insertion.
3. Spontaneous bleeding from gums and nose.
4. Generalized oozing in surgical fields.
5. Purpuric areas at pressure sites (thrombocytopenia and incoagulable blood).

INVESTIGATIONS: aiming to detect fibrinolysis.

1. FDPs and fibrin D-dimer (normally absent).
2. Prolonged PT and PTT(PTT may be normal).
3. Low fibrinogen, falling antithrombin III & low platelet count (CBC should be done).
4. Weiner test (clot observation test): 5-10 cc of blood in a test tube are incubated at 37°C. A) Normally a clot forms within 3-8 minutes. B) a clot forms after a longer time and dissolves within one hour = hypofibrinogenemia. C) No clot is formed = afibrinogenemia.

TREATMENT

1. It must be directed to the underlying cause to reverse defibrination. Two wide-bore IV cannula are inserted.
2. If PT is > 1.5 times the control value, transfuse fresh frozen plasma (FFP). The goal is to keep PT within 2-3 sec. of the control value.
3. If fibrinogen level is < 100 mg/dL, transfuse cryoprecipitate. Ten units of cryoprecipitate are usually given after every 2 to 3 units of plasma. Each unit of cryoprecipitate increases the fibrinogen by 10 mg/dL. OR give Fibrinogen 4-10 g IV.
4. Platelets should be transfused if the count is < 20 000/cmm or if clinically significant bleeding occurs with a platelet count between 20 000 and 50 000/cmm. Each platelet unit increases count by 10,000/cmm. The usual rate of platelet transfusion is 1-3 U/ 10Kg/ day.
5. Antifibrinolytics as Epsilon Amino Caproic Acid (EACA) 4-6 g IV OR trasylol 2-4 ampoules IV (5 ml ampoule contains 25 000 U), is not recommended in most types of obstetric coagulopathy to avoid organ ischemia and infarction unless all above mentioned measures fail to control bleeding.
6. Heparin infusion trying to stop coagulation is condemned when the vascular system integrity is compromised.

Key points in Hypofibrinogenaemia

1. Pregnancy is a hyper-coagulable state with increased concentration of coagulation factors.

2. Disseminated Intravascular Coagulation is characterized by accelerated fibrin formation and lyses.

3. Common causes include Massive blood loss, Placental abruption and Severe pre-eclampsia/eclampsia.

4. Rare causes include Sepsis, Retained dead fetus, Amniotic fluid embolism and Acute fatty liver.

5. Accelerated coagulation occurs via the extrinsic pathway.

6. Treatment must be directed to the underlying cause to reverse defibrination.

7. If PT is > 1.5 times transfuse fresh frozen plasma.

8. If fibrinogen level is < 100 mg/dL transfuse cryoprecipitate.

9. Platelets should be transfused if the count is < 20 000/cmm or if clinically significant bleeding occurs with a platelet count between 20 000 and 50 000/cmm.

35 ASSESSMENT OF FOETAL WELL BEING

Antepartum assessment of FWB
Placental insufficiency
Antepartum foetal surveillance
- DFMC, NST, US BBP, US CD, OCT
Management of placental insufficiency

Intrapartum assessment of FWB
Causes of intrauterine foetal hypoxia
Intrapartum foetal surveillance
- CTG, FBS,
Management of foetal distress during labour

I. ANTEPARTUM ASSESSMENT OF FOETAL WELLBEING

AIM:

*To detect foetuses at risk of **hypoxia during 3rd trimester of pregnancy**, so that it can be delivered before IUFD, or hypoxic injury. Foetal hypoxia is mostly caused by **uteroplacental insufficiency.***

PLACENTAL INSUFFICIENCY

DEFINTION : Failure of the placenta to deliver adequate oxygenation and nutrition to the foetus.

TYPES

1. ***Acute:*** Suddenly occurring associated with otherwise normal foetus. (Placental separation).
2. ***Chronic:*** Associated usually with intrauterine growth restriction (IUGR - small for date foetus).

AETIOLOGY:

Impairment of the placental circulation due to thrombosis of the vessels or placental infarcts.

1. Hypertensive states of pregnancy (PE, eclampsia, and chronic hypertension).
2. Accidental hemorrhage or placenta praevia.
3. Postmaturity syndrome.
4. Diabetic pregnancy when associated with vasculopathy.
5. Placental infarctions.
6. Idiopathic.

PATHOLOGY

Foetal response to placental insufficiency:

1. Redistribution of blood flow preferentially to the brain and foetal heart.
2. Asymmetric IUGR (see foetal growth disorders).
3. Oligohydramnios due to reduced renal perfusion.
4. Decreased foetal movement to conserve energy.
5. Disturbance of foetal heart rate due to defective autonomic regulation.

DIAGNOSIS OF PLACENTAL INSUFFICIENCY

A) HISTORY:

Careful history taking may reveal the cause of placental insufficiency, e.g. PE, post maturity, etc.

B) CLINICAL SIGNS SUGGESTIVE OF SGA FOETUS OR OLIGOHYDRAMNIOS:

- Poor maternal weight gain during regular ANC follow up (normal = 0.5 kg/wk > 20 wks).
- Small abdominal girth, with undersized uterus (fundal level < Gestational age "GA").

C) ANTEPARTUM FOETAL SURVEILLANCE AFS (Placental Function Tests):

1. ***Daily Foetal Movement Count (DFMC)***

- Recorded 2 days each week, after 30 weeks.
- More than 10-12 gross movements within 10-12 hrs indicate good foetal movements.
- If DFMC is decreased, further foetal evaluation is recommended (NST and US).

2. ***The Non Stress Test (NST):***

- **Idea:** To test FHR changes in response to foetal movements. FHR are recorded by electronic monitoring using a special apparatus with a duplex ultrasound probe attached to the maternal abdomen. Foetal movements are reported by the mother who presses on a special button concomitant with each foetal movement.
- **Procedure:** by external cardiotocography **(CTG)**
  - Basal FHR is recorded and its changes in response to foetal movements are detected.
  - Duration of the test: FHR recordings over 20 min observation period. The test may be extended for another 20 minutes if foetal movements are insufficient.
- **Response to NST:**

a. ***Reactive:*** Normally a rise in FHR of at least 15 bpm, for at least 15 sec. will occur at least twice within a period of 15-20 min. testing.

b. ***Non reactive:*** No FHR changes or changes less than normal (see above).

Management according to NST:

1. *Reactive NST:* repeat every week.
2. *Non reactive NST:* Further foetal evaluation:
   - Ultrasound Biophysical Profile Score (BPPS)
   - Colour Doppler velocimetry studies (foetal umbilical and cerebral arteries)
   - Oxytocin Challenge Test (OCT).

3 ***Ultrasound Biophysical Profile Score (BPPS):***

- **The BPPS:** It is a simple test based on clear ultrasound parameters that can pick up those foetuses at risk of hypoxia. Each tested parameter is given a score (either 0 or 2).
- **Timing of BPPS:** any time in the 3rd trimester and can be repeated freely when needed.
- **Parameters studied:** observation of the following parameters over a period of 30 minutes
  1. Foetal tone (flexion attitude of foetal limbs, head and body).
  2. Foetal body movements (flexion / extension limb and body movements).
  3. Foetal breathing movements FBM (expansion movements of the foetal abdomen)
  4. Amniotic fluid volume (or amniotic fluid index - AFI).
  5. NST (normal: when foetal movements percieved by the mother are associated with a FHR rise of 15 bpm, for 15 secs, twice in 15-20 min. duration).
- **Interpretation of the test:** Each item is scored either 0 or 2, with a maximum 10/10.

- Scores of 8 to 10 denote a normal foetal well being, with normal foetal PH.
- Scores of 8/10 may need repeating the test and /or adding Doppler US evaluation.
- Scores < 8 suggest severe hypoxia. Termination of pregnancy should be considered.
- Scores < 6 denote severe foetal acidaemia, with severely compromised foetal outcome.

- **The modified BPP score:** It combines the non stress test (NST), with the amniotic fluid index (AFI). It is simpler and quicker than the complete BPP, and seems to have the same sensitivity for detecting foetal compromise. If the NST is non reactive, a complete BPP should be done.

4. ULTRASOUND COLOR DOPPLER STUDIES (US CD) OF FOETAL BLOOD FLOW:

Measurement of the resistance to foetal blood flow within the ***umbilical artery*** and the ***middle cerebral artery*** yields excellent information about the state of foetal organ perfusion.

High resistance in the umbilical artery is concomitant with placental insufficiency, while **Low** resistance in the foetal middle cerebral artery points to vascular shift and brain sparing, suggestive of hypoxia. Absent or reversed end-diastolic flow in the umbilical arteries Doppler velocimetry flow studies denotes a severely compromised foetus that needs urgent delivery.

5. OXYTOCIN CHALLENGE TEST (OCT): Rarely resorted to in the presence of US CD.

Monitoring FHR changes on CTG, in response to I.V. oxytocin induced mild uterine contractions.

a) Positive OCT: FHR decelerations obtained after each uterine contraction (foetal distress).

b) Negative OCT: No FHR changes occur in response to contractions (good foetal well being).

The test has formerly been indicated if the NST was non reactive, however OCT is rarely used nowadays being outdated by the safer and easier Doppler US velocimetry studies.

MANAGEMENT OF PLACENTAL INSUFFICIENCY

1. ***Chronic placental insufficiency:*** cases with IUGR are carefully monitored and delivered nearest to 37 weeks gestation. If Doppler studies revealed very high umbilical artery resistance index with poor BPP score, immediate termination of pregnancy is indicated.

2. Cases with ***acute placental insufficiency*** from the start, or those who developed acute on top of chronic insufficiency, with Oligohydramnios, poor BPP scores, abnormal Doppler studies, are for immediate termination of pregnancy irrespective of foetal lung maturity.

N.B.: Delivery by CS, offers the best chance for foetal survival in cases with placental insufficiency when pregnancy termination is indicated. In some selected cases induction of labour may be chosen with continuous electronic FHR monitoring by the CTG. If foetal bradycardia occurs, or if ROM revealed presence of meconium, vaginal delivery is abandoned and CS is rapidly performed.

KEY POINTS IN ANTEPARTUM FOETAL SURVEILLANCE (AFS):

1. *AFS detects foetuses at risk secondary to utero-placental insufficiency, but cannot predict sudden events.*
2. *All pregnant women at high risk for placental insufficiency should do a DFMC.*
3. *The NST should be offered to cases with decreased DFMC or to high risk cases in general.*
4. *The BPP includes a NST with evaluation of specific US parameters. It should be performed and repeated whenever foetal compromise is suspected.*
5. *Doppler umbilical artery studies are indicated whenever an abnormal BPP is encountered. Abnormal findings reflect serious foetal condition.*
6. *Normal AFS tests may be repeated on weekly or biweekly intervals.*
7. *Abnormal AFS tests necessitate termination of pregnancy.*
8. *In most cases CS delivery will offer best chances for foetal neonatal survival.*

II. INTRAPARTUM ASSESSMENT OF FOETAL WELL BEING

AIM:

To detect foetuses at risk for *hypoxia during labour* ***(foetal distress)***, thus minimizing the risks of intrapartum and neonatal foetal death, or severe foetal hypoxic injury (e.g.: cerebral palsy).

PATHOPHYSIOLOGY OF HYPOXIA:

Decreased Oxygen supply to the foetus during labour is associated with decreased elimination of CO2, with resultant foetal respiratory acidosis and decreased foetal blood PH.

CAUSES OF INTRAUTERINE FOETAL HYPOXIA (FOETAL DISTRESS)

1. ***Acute foetal hypoxia:***
   - Cord accidents (cord prolapse, cord compression, true knots, coils around foetal neck, and rupture vasa praevia).
   - Placental separation (accidental Haemorrhage, placenta praevia).
   - Placental compression (prolonged ROM, uterine hypertonicity, obstructed labour).
   - Some congenital and or chromosomal foetal anomalies.
2. ***Chronic foetal hypoxia:***
   - Placental insufficiency: (see before).
   - Maternal hypoxia: (Severe anaemia or excessive haemorrhage, congestive heart failure, severe pulmonary disease, during eclamptic fits, during anaesthesia with improper oxygenation).

DIAGNOSIS OF FOETAL DISTRESS DURING LABOUR

A) CLINICAL DIAGNOSIS:

1. Abnormal FHR recordings by the sonicaid (severe bradycardia, severe tachycardia).
2. Passage of meconium after ROM in (cephalic presentation but not in breech).
   - **N.B.:** Intrauterine asphyxia results in relaxation of the foetal anal sphincter & increased intestinal peristalsis. Meconium, (the thick intestinal particulate secretion in the neonate), will then escape into the amniotic fluid giving it yellowish or greenish colour that appears after rupture of membranes.

B) INTRAPARTUM FOETAL SURVEILLANCE - IFS

The aim of IFS tests is to detect foetal distress occurring during labour. The best available method for such IFS is the use of continuous ***external electronic FHR monitoring***, with or without testing **foetal scalp PH** if foetal distress develops.

1. <u>Electronic FHR Monitoring:</u> (Cardiotocography – CTG)

The CTG entails Continuous electronic FHR monitoring during labour in relation to uterine activity. An external US transducer is placed on the maternal abdomen at a site selected to give the best FHR recordings. Another transducer is placed to record the onset, duration and intensity of uterine contractions and associated increased intrauterine pressure.

- **Interpretation of FHR Monitoring by CTG:**

A) Normal FHR tracings:

1. Normal FHR values: 120-140 beat per minute (b/m).
2. Normal FHR Pattern: beat to beat variability (fluctuations by ± 5 to 10 b/m, periodically).

B) Abnormal FHR tracings:

1. Baseline *bradycardia:* <100 b/m.
2. Baseline *tachycardia:* >160 b/m.
3. *Absence of beat to beat variability*: FHR variation within 1 min is < 5 b/m.
4. *Late Decelerations*: Slowing of FHR at the ***peak*** of the uterine contraction that returns to normal a short period after the contraction ends. Late deceleration is the most dangerous pattern, as it indicates severe utero-placental insufficiency.
5. *Variable Decelerations*: Slowing of FHR which is not related to uterine contractions. It mostly indicates cord compression especially in cases with ROM or Oligohydramnios.

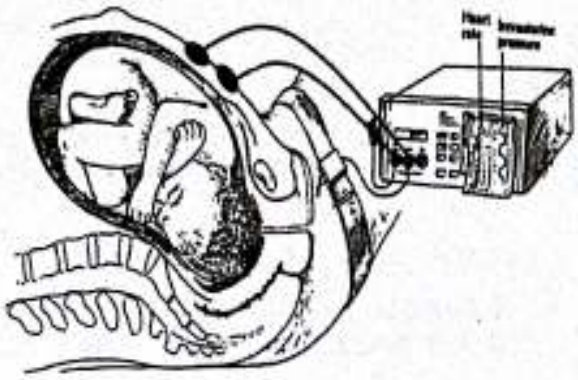


Fig 35:1 External FHR Monitoring

- **N.B.:** Early Decelerations refer to slowing of FHR that starts at the ***onset*** of the uterine contraction and returns to normal with its end. It is mostly associated with head compression within the bony pelvis with reflex stimulation of vagus nerve. Early decelerations are usually harmless.
- **N.B.:** Sinusoidal FHR pattern is a smooth wave like pattern of regular frequency (3-5 cycles/min), and amplitude (5-20 bpm). Short episodes of sinusoidal FHR patterns are considered a variant of normal. Longer episodes may reflect foetal anaemia and severe hypoxia (e.g. accidental haemorrhage).

2. *Foetal Blood sampling During labour (FBS)*

Abnormal FHR patterns on CTG during labour are sometimes misleading, with a resultant increase in the rate of unnecessary CS deliveries. In cases where FHR tracings on the CTG are abnormal, foetal blood sampling (FBS) will be the most accurate method in detecting foetal hypoxia.

The blood sample is taken via a small needle probe applied to foetal scalp after ROM. Few drops of foetal blood are adequate to perform the test, which is known as foetal scalp pH.

- Normal foetal pH ranges from 7.25-7.35.
- In cases of foetal hypoxia:
  Accumulation of CO_2 → Anaerobic pathway for energy → lactic acid.
- This leads to foetal acidosis. If < 7.25 → Mild acidosis. If < 7.2 → severe acidosis.

MANAGEMENT OF FOETAL DISTRESS DURING LABOUR

1. **High risk cases** should be offered a **continuous CTG monitoring** throughout labour.
2. If **CTG** records **abnormal FHR patterns;** the following should be done:
   - Stop oxytocin, if it has been infused (to minimize uterine contractions and decrease intrauterine pressure, thus improving foetal placental perfusion in between contractions).
   - Change position of the mother to left lateral position (allows increased blood supply).
   - O_2 mask to the mother (to avoid maternal hypoxia).
   - I.V. Fluids (to avoid maternal dehydration).
3. If the above measures are **successful** (i.e. FHR patterns return to normal):
   Labour is allowed to proceed under strict observation, if vaginal delivery is rapidly anticipated.
4. **If The above measures were unsuccessful** (i.e.; persistent abnorml FHR patterns)
   An immediate *delivery by CS* will be the best and safest option available.

N.B.:

If foetal distress occurs when the cervix is fully dilated, the membranes ruptured, the presenting part engaged, and the maternal pelvis is adequate, ***immediate vaginal delivery may be encouraged by:***

- Instrumental delivery by the low forceps procedure.
- Breech extraction in cases of complete or frank breech presentations.

KEY POINTS IN INTRAPARTUM FOETAL SURVEILLANCE (IFS):

1. *Cases at high risk for foetal hypoxia should be selected and carefully monitored throughout labour.*
2. *Intrapartum foetal monitoring will detect early foetal hypoxia (foetal distress), preventing many serious foetal and neonatal hypoxic complications.*
3. *IFS has decreased the number of intrapartum foetal deaths, but it has increased the number of CS deliveries.*
4. *FHR accelerations signify normal foetal PH and intact CNS.*
5. *Decelerations are characterized based on the timing with contractions into:*
   - *Early decelerations: head compression vagal response (no hazards).*
   - *Late decelerations: utero-placental insufficiency (serious hypoxia anticipated).*
   - *Variable decelerations: cord compression (especially with ROM or oligohydramnios).*
6. *Whenever IFS points to foetal distress, measures should be taken for immediate delivery:*
   - *Most cases will benefit from immediate CS.*
   - *Some selected cases may continue a carefully monitored trial for a rapid vaginal delivery*

36 FOETAL GROWTH DISORDERS

DEFINITIONS

- ***Low birth weight (LBW):*** Infants with birth weight <2500 gm regardless their gestational age.
- ***Small for gestational age (SGA)*** foetuses are <10th percentile weight for gestational age.
- ***Large for gestational age (LGA)*** foetuses are >90th percentile weight for gestational age.
- ***Appropriate for gestational age (AGA)*** foetuses are between 10th & 90th percentile weight for gestational age.

Birth weight is a function of both gestational age and rate of foetal growth. Therefore the correct assignment of foetal weight (FW) depends on accurate dating of pregnancy.

During the intrauterine period of life, abnormal foetal growth patterns may be either:

1. **Restricted foetal growth** (Intrauterine growth restriction - IUGR - SGA)
2. **Accelerated foetal growth** (Foetal macrosomia - LGA)

I - INTRAUTERINE GROWTH RESTRICTION (IUGR)

DEFINITION

It refers to any foetus that fails to reach its full growth potential (<10th percentile weight according to foetal growth curves).

AETIOLOGY

1. Constitutionally Small foetus:

Small women typically have smaller babies. If a woman begins pregnancy weighing less than 42 Kg, the risk of delivering an SGA infant is at least doubled.

2. Growth Restriction (retardation):

- **Summetrical IUGR**
- **Asymetrical IUGR**

N.B.: These two types are likely to be th econsequence of different causes, time of onset, and duration of events.

1. SYMMETRICAL IUGR (TYPE I):

Symmetric IUGR usually results from ***foetal injury*** very early in development. It is thus ***intrinsic to the foetus*** and constitutes about **20%** of IUGR cases.

Aetiology of Symmetrical IUGR:

A. Poor maternal weight gain: Lack or arrest of maternal weight gain specially after 28th week gestation, is commonly associated with symmetric IUGR.

B. Foetal infections:
   - *Viral:* Rubella, Cytomegalovirus, Hepatitis, Varicella and Influenza, may cause congenital infection and growth retardation.
   - *Bacterial:* Listeriosis, tuberculosis, syphilis. In Syphilis, the placenta almost always increase in size and weight due to oedema and perivascular inflammation.

C. Congenital Malformation:e.g. Serious cardiovascular malformation or renal hypoplasia. The more severe is the malformation, the more likely the foetus to be SGA

D. Chromosomal Abnormalities: Trisomies, especially of chromosome 13, 18, 21, and others, cause some of the most severe forms of IUGR

E. Skeletal Anomalies: e.g. Osteogenesis imperfecta, and other numerous inherited syndromes

2. ASYMMETRICAL IUGR (TYPE II):

Asymmetrical IUGR usually results from ***foetal injury*** later in pregnancy, commonly due to ***maternal diseases*** that are ***extrinsic to the foetus*** .The foetal size is affected via:

1. Reducing uteroplacental blood flow: as with hypertensive disorders.
2. Restricting oxygen and nutrient transfer: as with sickle cell disease.
3. Reducing placental size with infarcts and vasculopathy: as with PIH and diabetes.

The foetus reacts to such ***chronic placental insufficiency*** by redirecting its blood flow to be maintained to the brain and decreased to most visceral organs, a condition known as ***"Brain sparing phenomenon"*** This will result in:

- Abnormal head to abdominal circumference ratio ***(increased HC/AC ratio).***
- Reduced renal perfusion and decreased urine output, leading to ***oligohydramnios.***

Aetiology of Asymmetric IUGR:

1. Vascular disease: Chronic hypertension, Preeclampsia, and Diabetic vasculopathy.
2. Chronic Renal disease: Renal insufficiency is usually associated with type II IUGR.
3. Chronic Hypoxia: Foetuses of mothers with cyanotic heart disease show frequent IUGR.
4. Placental and cord abnormalities:
   - Chronic focal placental abruption, extensive infarction, and chorioangioma.
   - Marginal and velamentous insertions of the cord.

DIAGNOSIS OF IUGR

1. Proper pregnancy dating: history of an accurate LMP, calculation of gestational age (GA) and expected date of delivery (EDD). N.B.: *ultrasound Foetal biometry alone is not considered a sensitive predictor of gestational age in the late second and third trimester.*
2. Symphysial fundal height measurement: Between 20 and 34 weeks gestation, the longitudinal distance measured from the symphysis pubis to the uterine fundus in centimetres, roughly coincides with weeks of gestation. If the measurement is less than 2.0 cm from the expected height, inappropriate foetal growth is suspected.

3. Ultrasound Assessment :

- Decreased BPD and AC measurements < 10th percentile for GA, or altered AC/HC ratio.
- Estimated foetal weight < 10th percentile for gestational age.
- Oligohydramnios associated with IUGR.
- Accelerated placental aging (early grade III placenta < 34 weeks, with calcifications).
- Abnormal umbilical and cerebral artery Doppler flow indices (decreased umbilical and preserved or increased cerebral vascular flow in asymmetrical IUGR).

MANAGEMENT OF IUGR

I. Near Term IUGR:

Prompt delivery is likely to afford the best outcome for the foetus who is growth retarded and diagnosed near term, whether symmetrical or asymmetrical type.

II. Preterm IUGR:

1. *Symmetrical IUGR:*
   - Exclusion of important foetal congenital and or chromosomal anomalies, by detailed US foetal anatomy scan, amniocentesis, or cordocentesis if indicated. Manage accordingly
   - Screening for Toxoplasmosis, Rubella, CMV, Herpes viruses. Treatment individualized.
   - Evaluation of foetal well being (in an otherwise normal foetus):
     1.Daily foetal movement count (DFMC).
     2.Non stress test (NST).
     3.Biophysical profile score, twice weekly (BPPS).
     4.US CD: serial Doppler velocity waveform measurements for maternal uterine and foetal umbilical, middle cerebral, and renal arteries.

N.B.: Once the foetus starts being compromised, termination of pregnancy is advised.

2. *Asymmetrical IUGR:*

Antepartum foetal surveillance (AFS) is started, and if it shows;

- Foetus not severely affected : pregnancy is allowed to continue withrepeated testing
- Foetus severely ill: termination of pregnancy is usualy offered, otherwise IUFD will occur.

COMPLICATIONS OF IUGR

1. **Foetal:** FHR abnormalities during labour, asphyxia and IUFD.
2. **Neonatal:**
   - *Immediate (50%):* meconium aspiration, hypoglycemia, polycythaemia and pulmonary haemorrhage.
   - *Late (2%):* Cerebral dysfunction (mild to cerebral palsy).

II – FOETAL MACROSOMIA

DEFINITION: foetus with absolute birth weight of either >4000g or >4500g.

INCIDENCE: 3% of neonates are >4000g and 0.5% are >4500g at birth.

RISK FACTORS:

1. Maternal diabetes (the most common risk factor).
2. Post-term pregnancy.
3. Maternal obesity (a prepregnancy weight of >90kg), and increased maternal height.
4. Multiparity and prior macrosomic infant.

DIAGNOSIS:

1. Clinical estimates of foetal size, based on Leopold's manoeuvres or fundal height measurements, are often inaccurate and are markedly affected by clinical experience and maternal obesity.
2. Ultrasonic estimates of foetal weight are reasonably accurate with only 15-20% error range, in comparison to actual foetal weight after delivery. However US estimates of foetal weight are more difficult in obese women.

PREVENTION:

1. Meticulous control of maternal diabetes.
2. Obese women should lose weight before conception and once pregnant should gain less weight than the average patient.

MANAGEMENT:

1. During pregnancy: Serial US to chart foetal growth, and exclude anomalies.
2. Induction at about 37 weeks to minimize the need for a CS delivery. This approach is at best controversial, and should be reserved only to highly selected cases.
3. Elective caesarean section (C/S): If USEFW ≥4250 especially in diabetic pregnancies.
4. Vaginal delivery: If attempted, anaesthesia staff and neonatal resuscitation team must be available. Assisted instrumental vaginal delivery MUST BE AVOIDED.

COMPLICATIONS:

a) Foetal Complications:
   - IUFD (in diabetic macrosomia or serious congenital malformations)
   - Birth trauma (shoulder dystocia and brachial plexus palsy)
   - Hypoglycaemia, polycythaemia, hypocalcaemia and jaundice.

b) Maternal:
   - Higher incidence of C/S deliveries.
   - Traumatic injuries of birth canal, postpartum haemorrhage (PPH) and puerperal infection.

37 PRETERM LABOUR AND PREMATURITY

Definition
Incidence and risk factors
Aetiology of PTL
Maternal complications of PTL
Foetal and neonatal complications
Prediction of PTL
Prevention in HRP
Management of patients with PTL
- *Allowing labour to proceed*
- *Attempts to prolong pregnancy (tocolytic therapy)*
- *Use of corticosteroids*
- *Antibiotic therapy*

DEFINITION

Preterm labour (PTL) is defined as the onset of frequent uterine contractions associated with progressive effacement and dilatation of the cervix, before 37 weeks gestation.

INCIDENCE

Nearly 5-10 % of pregnancies are complicated by PTL

RISK FACTORS:

1. Twins and multifoetal pregnancy.
2. History of previous PTL, recurrent or habitual abortions.
3. Low socioeconomic status, and poor nutrition (e.g.; anaemia and vitamin deficiencies ..etc.).
4. Extremes of maternal age (the very young and the elderly gravida).
5. Maternal cigarette smoking.

AETIOLOGY:

1. In at least **20-30%** of cases the cause of PTL will remain **unknown.**
2. **Premature rupture of membranes (PROM).** (20-30%), due to prostaglandin release.
3. **Chorioamnionitis:** responsible for 20-30 % of all causes of preterm labour.
4. **Systemic extrauterine infections:** in 5-10 % of cases, most commonly in urinary tract.
5. **Placental abnormalities:**
   - Placenta praevia (due to early separation with formation of the lower segment).
   - Placental abruption (retroplacental haemorrhage provokes early uterine contractions).
6. **Uterine abnormalities:**
   - Septate and bicornuate uterus (inability to cope with rapid distension associated with increased foetal growth in the 3rd trimester + incompetent cervix in some cases).
   - Uterine leiomyoma (interfere with uterine distension + provokes uterine contractions).
7. **Foetal causes:**
   - Multiple pregnancy (uterine overdistension).
   - Major congenital anomalies as open neural tube defect (anencephaly)
   - Inborn errors of metabolism and chromosomal abnormalities (unknown cause).
   - Foetal death: Spontaneous labour occurs within few weeks of IUFD (hormonal changes).

8. **Uterine over distension:**
   - As with polyhydramnios and LGA foetus, due to increased intrauterine pressure.

MATERNAL COMPLICATIONS OF PTL

- Chorioamnionitis and its sequalae including puerperal sepsis.
- Increased risk for recurrent preterm labour and midtrimesteric abortion.

FOETAL AND NEONATAL COMPLICATIONS OF PTL

1. **Birth trauma:** increased incidence of intraventricular haemorrhage (IVH).
2. **Respiratory distress syndrome (RDS):** due to decreased surfactant (mainly lecithin), which is formed by the alveoli. It decreases the surface tension of alveoli, allowing expansion during inspiration and prevents collapse during expiration. In absence of surfactant the alveoli collapse and are covered by structure less hyaline material. Progressive dyspnoea and cyanosis occurs 1-2 hours after delivery and death usually occurs, unless treatment is rapidly initiated.
3. **Neonatal hypothermia:** Increased heat loss due to decreased subcutaneous fat and immaturity of heat regulation centre.
4. **Neonatal sepsis:** Increased susceptibility to infection due to decreased immunoglobulins transferred from the mother.
5. **Anaemia** due to poor iron storage.
6. **Bleeding tendency** due to hypoprothrombinaemia and capillary fragility.
7. **Malnutrition** due to poor suckling, poor digestion.
8. Increased liability to **hyperbilirubinaemia** due to immaturity of liver enzymes.
9. **Iatrogenic complications** as
   - Retro-lental fibroplasia (due to oxygen excess in the incubator).
   - Rupture pulmonary alveoli (due to high pressure oxygen on the ventilator).
10. **Neonatal mortality:** PTL is a major cause of neonatal death.

PREDICTION OF PTL: (not easily identifiable)

A. Warning symptoms and signs: *Frequently misleading.*
   - Frequent and recurrent menstrual like cramps.
   - Low dull backache and markedly increased pelvic pressure.
   - Increase in amount of vaginal discharge, and or fluid leaking from the vagina.
   - Uterine contractions that are 15 minutes apart or closer.
   - Finding short, soft, partially effaced cervix during vaginal examination.

B. Special investigations: *strongly suggestive.*
   - Transvaginal US to measure length of cervical canal (shortened if < 2.5 cm).
   - Assay of foetal fibronectin in cervico-vaginal fluid (> 50 ng/ml).

DIAGNOSIS OF ESTABLISHED PTL:

- **True labour pains,** as evidenced by palpation and CTG, associated with
- **progressive cervical effacement** (>50%)and **dilatation** (>2.0 cm).

PREVENTIVE MEASURES IN HIGH RISK CASES:

1. Patient **education** about preterm labour.
2. Good **nutrition** and treatment of **anaemia** and vitamin deficiency.
3. **Limitation** of physical and sexual activity.
4. Treatment of **vaginal** and **cervical infections:**
   - GBS +ve cervical cultures (group B streptococcal infection) should receive properly selected antibiotics (ampicillin or erythromycin).
   - Trichomonal vaginal infections are treated with oral or vaginal metronidazole preparations.

MANAGEMENT OF PATIENTS WITH ESTABLISHED PTL:

1. *ALLOWING PTL TO PROCEED AND DEAL WITH THE NEWBORN.*

The foetus is left to be delivered vaginally, or by CS, and managed accordingly in a well equipped neonatal ICU. This may be indicated in the following conditions:

- Membranes ruptured, or cervix > 50% effaced and /or > 2.0 cm dilated.
- The presence of reasonably adequate foetal lung maturity (34-36 weeks)
- Severe IUGR with marked placental insufficiency.
- Foetal congenital anomalies incompatible with survival (as anencephaly).
- Severe maternal illness and disease that precludes continuation of pregnancy as in cases of severe preeclampsia (PE), uncontrolled chronic hypertension, severe or advanced cardiac, renal or liver disease).

During labour:

- Continuous electronic monitoring of preterm foetus during labour is mandatory because preterm infants tolerate hypoxia more poorly than infants at term.
- Avoid prolongation of the 2nd stage of labour.
- Episiotomy: to minimize head compression and decrease intracranial haemorrhage.
- C.S.: is indicated in preterm breech, and the extremely low birth weight (LBW) foetus.
- Vitamin K1: Administered to the neonate to reduce the incidence and severity of IVH.

2. *ATTEMPTS TO PROLONG PREGNANCY AND STOP LABOUR PAINS (TOCOLYTIC THERAPY):*

- Aim of therapy: Prolongation of pregnancy by inhibiting uterine contractions until:
  - Transfer of patient to a centre equipped with a more advanced neonatal ICU.
  - Enhancement of foetal lung maturity by use of corticosteroids.
- Indications: PTL before 34 weeks.
- Contraindications:
  - Maternal conditions contraindicating prolongation of pregnancy as PE, Antepartum haemorrhage (APH), advanced cardiac, renal disease, or distressing maternal illness.
  - Foetal conditions precluding continuation of pregnancy (severe IUGR, serious congenital anomalies incompatible with life, and intrauterine foetal death).
  - Chorioamnionitis and rupture of membranes (relative contraindications).
- Duration of Tocolysis:
  - ***Short term tocolysis***, (mostly required for the 1st 48 hours), until corticosteroids are administered, and mother transferred to a centre with neonatal ICU facilities gives better chances for survival of the premature foetus.
  - ***Long term tocolysis*** is of doubtful clinical value. Furthermore the drugs used have many adverse side effects on long term use.

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- Intravenous Tocolytic Agents :

A. *Beta adrenergic agonists:* Ritodrine HCL & Terbutaline are the drugs commonly used. However their use is associated with several maternal side effects as tachycardia, tachypnia, hypotension and abnormal glucose tolerance.

B. *Magnesium sulphate:* Its use is limited by its high toxicity in larger doses, which are difficult to monitor and to control.

- Oral Tocolytic therapy:

A. *Calcium channel blockers* (Nifedipine). Gaining more popularity in recent years, being safer than IV or oral beta agonist agents, and with fewer side effects.

B. *Prostaglandin synthetase inhibitors* (Indomethacin). Although effective, yet its use is limited by possible complications as Oligohydramnios and premature closure of the ductus arteriosus.

C. *Glyceryl trinitrate:* Results in vasodilatation and muscle relaxation (under trial).

D. *Beta adrenergic agonists:* can also be administered orally, however their use is controversial and their effect is doubtful.

E. *Anti-Oxytocin drugs:* very recently a new generation of antioxytocin drugs given parentarily via IV drip infusion has been tried with very promising results

3. *THE ROLE OF CORTICOSTEROID THERAPY:*

- **Aim:** To accelerate foetal lung maturity and minimize both RDS and IVH.
- **Indication:** PTL < 34 weeks.
- **Value and actions:**
  1. Helps to minimize the incidence of RDS due to hyaline membrane disease (HMD), by enhancing lung maturity via stimulation of type II pneumocytes.
  2. It is also beneficial in decreasing the occurrence of intraventricular haemorrhage IVH, and necrotizing eneterocoloitis (NEC).
- **Dose and Regimen:**
  - **Two** maternal I.M. injection of Betamethazone 12 mg each, given 24 hours apart. **OR**
  - **Four** I.M. injections of dexamethazone 6.0 mg each given 12 hours apart.

4. *THE ROLE OF ANTIBIOTIC THERAPY:*
   - For associated infection or in cases of PROM.
   - For prophylaxis in cases which will adopt a conservative expectant approach.
   - For treatment of early infection or established cases (parentral route by I.V. or I.M.)

KEY POINTS IN PTL:

1. *Prevention of PTL is difficult however selection of high risk cases is beneficial.*
2. *Positive GBS cervical cultures in high risk cases should be properly treated.*
3. *Tocolysis is primarily used to delay delivery until the administration of corticosteroids.*
4. *IM corticosteroids administered to the mother 24 hours before delivery significantly decreases complications of prematurity namely RDS and IVH.*
5. *Management of a preterm foetus should be undertaken in a well equipped neonatal ICU.*
6. *PTL 34-36 wks, carries best chances for neonatal survival (foetal weight >2.0 kg).*
7. *The extreme LBW < 1.0 kg (< 28 weeks), carry the poorest prognosis for the neonate.*
8. *In cases of severe RDS the administration of surfactant to the neonate in the first 24 hours may significantly improve the prognosis for survival.*

38 POST-TERM PREGNANCY

Definition
Incidence
Aetiology
Diagnosis
Clinical features of post-maturity
Effects of prolonged pregnancy
Management of post-term pregnancy

DEFINITION

Post-term or prolonged pregnancy, is a pregnancy that lasts for 42 weeks or more from the onset of the last menstrual period i.e. (294 days)

INCIDENCE: Nearly 8-10 % of all pregnancies are prolonged.

AETIOLOGY

1. **Inaccurate or unknown LMP:** usually results in *miscalculation* which is the commonest cause encountered. There is a high association with *no or late antenatal care (ANC).*
2. **Irregular ovulation:** Variable length of the follicular phase made prediction of the timing of onset of pregnancy difficult. This results in over estimation of the gestational age.
3. **Altered oestrogen / progesterone ratio:**
   - Anencephaly.
   - Decreased foetal production of precursors of estriol.

DIAGNOSIS OF POST-TERM PREGNANCY

The diagnosis of postmaturity in a patient who was not sure of her LMP, and was not attending an antenatal care program (ANC) from the beginning of her pregnancy is usually difficult and frequently misleading.

1. Ensure the accuracy of the date of the LMP, and exclude miscalculation.
2. Correlate the proposed date of LMP with:
   - Timing of her first positive urine or blood pregnancy test.
   - Time of first performed ultrasound scan (best in the first trimester).
   - Date of quickening (around 17 weeks).

N.B.: Ultrasound foetal biometry is very reliable in estimating gestational age in the first trimester. Unfortunately it is less accurate in the late second and third trimesters.

CLINICAL FEATURES OF POSTMATURITY

- The post-term newborns may demonstrate some characteristic features as: wrinkled, patchy peeling of skin, long nails, the infant is open eyed and alert.
- Post-maturity is associated with higher incidence of oligohydramnios and meconium passage with its complications.

EFFECTS OF PROLONGED PREGNANCY

1. Foetal Distress and Oligohydramnios:

Amniotic fluid volume diminishes from nearly 1.0 litre at 36 weeks to less than 0.5 litre at 42 weeks gestation. The result is a loss in protection for the umbilical cord with a higher incidence of cord compression and foetal distress.

Oligohydramnios in post-term pregnancies is caused by decreased foetal urine production due to increased renal artery resistance

2. Meconium Passage:

The incidence of meconium passage is markedly increased especially with foetal distress. Since oligohydramnios is also present, the infant is more likely exposed to aspirate more concentrations of this potentially toxic, particulate meconium.

3. Increased morbidity and mortality for post-term foetus:

a. Restricted growth and placental insufficiency: It increases several folds with resultant meconium aspiration, IUGR, oligohydramnios, and foetal distress.

b. Continued growth with good placental function: macrosomia with its consequences of obstructed labour, C.S. deliveries, shoulder dystocia, and brachial plexus injuries

MANAGEMENT OF POST-TERM PREGNANCY

1. Exclude miscalculation.

2. Assessment of the foetal well being by:

A. From 40 to 42 weeks: (conservative management)

Assessment of foetal well being: weekly examination, daily foetal movement count (DFMC), repeated non stress test (NST), Biophysical profile (BPP), and Doppler studies.

- If foetal assessment is good, we can wait till 42 completed weeks
- If non reassuring foetal condition; termination of pregnancy is indicated.

B. After 42 weeks: (termination of pregnancy)

- ***Induction of labour:*** If good foetal condition and favourable vaginal examination.
- ***Caesarean Section:*** If compromised foetal condition, or unfavourable vaginal examination (non engaged head, non effaced cervix, ?? inadequate pelvis).

39 PRELABOUR RUPTURE OF THE MEMBRANES

Definition
Incidence
Aetiology
Diagnosis
Maternal hazards
Foetal & neonatal hazards
Management options
- *Term PROM > 37 weeks*
- *Preterm PROM < 37 weeks*

DEFINITION

- Pre-labour rupture of membranes **(PROM)** refers to rupture of the foetal membranes spontaneously at any time **prior to the onset of labour.**
- If it occurs before 37 weeks gestation it is known as **preterm PROM.**
- The term *premature ROM* of the membranes, has been formerly used but is now universally replaced by the less confusing and more descriptive term *prelabour ROM*

INCIDENCE:

PROM complicates approximately **10%** of all pregnancies.

AETIOLOGY

In many cases the membranes may rupture spontaneously without any identifiable cause or predisposing factor. In other cases the underlying cause may be:

1. **Infection:** The source of infection is usually bacteria, which are normally present in the vagina (as trichomoniasis), or the cervix (as group B streptococci GBS). Infection could result in PROM through decreasing the tensile strength of membranes.
2. **Cervical incompetence:** The membranes are exposed to trauma and infection.
3. **Polyhydramnios** and multiple pregnancy. Due to increased intra-amniotic pressure.

DIAGNOSIS of PROM

1. **History:** "sudden gush" of fluid from the vagina followed by intermittent trickle.
2. **Speculum Examination:** A sterile speculum examination will reveal pooling of amniotic fluid (AF) in the posterior vaginal fornix. Digital cervical examination should not be performed if the patient is not in labour, because it carries the risk of introducing intra-amniotic infection.
3. **Nitrazine test:** This test detects the alkaline pH of the amniotic fluid in the vagina. Normally vaginal PH is 4.5-5.0, while PH of AF is nearly 7.0. Nitrazine paper turns deep blue at a PH above 6.
4. **Ultrasound examination:** Finding of oligohydramnios by U.S. is suggestive of PROM. Decreasing AF volume on repeated US is a good evidence, especially with continuous fluid leak.

Other microscopic and biochemical tests: as foetal Fibronectin and alpha fetoprotein detected in vaginal pool fluid.

HAZARDS OF PROM

Prolonged PROM > 24 hours before onset of labour may result in the following:

A. Maternal Hazards of PROM:

1. Chorioamnionitis:

- The overall **risk** of chorioamnionitis after PROM is about 20%. Ascending infection is the most likely **mechanism** of infection.
- The **hazards** of chorioamnionitis include maternal septicaemia, septic shock and disseminated coagulopathy. Together with foetal affection as pneumonia and septicaemia, etc.
- **Infection** with **GBS** (group B-haemolytic streptococci) is particularly important as it may cause overwhelming neonatal infection resulting in death or severe neurologic morbidity.
- **Clinical Diagnosis** of chorioamnionitis:
  - Temperature elevation to 38°C or more, **AND** two or more of the following criteria:
  - Uterine tenderness
  - Malodorous vaginal discharge.
  - Maternal and foetal tachycardia.
- **Laboratory Diagnosis** of chorioamnionitis:
  - Complete blood picture (CBC): reveals maternal leucocytosis (WBCs > 14,000) with shift to the left i.e. abnormal ratio between staff and segmented cells.
  - Biochemical markers: Positive C-reactive protein in maternal serum (normal 0-2).
  - Amniotic fluid bacterial cultures: positive for pathogenic bacteria.

2. **Postpartum Endometritis**: As a sequalae of chorioamnionitis.

3. **Placental abruption**: Possibly due to the progressive decrease in maternal surface area and sudden decrease in intrauterine pressure in cases of acute polyhydramnios.

B. Foetal and Neonatal Hazards of PROM:

1. Foetal/Neonatal Infection: The risk is greater with increasing prematurity, with the presence of chorioamnionitis and with increasing time interval between the last vaginal examination and delivery.
2. Respiratory Distress Syndrome (RDS): RDS due to hyaline membrane disease (HMD) is the greatest threat to the foetus when PROM occurs before maturity. It is the major cause of neonatal deaths.
3. Perinatal Asphyxia and Foetal Distress: Though cord compression or prolapse, these complications may also occur secondary to maternal fever and chorioamnionitis.
4. Pulmonary Hypoplasia: It is a major problem in preterm PROM particularly when prolonged. The amniotic fluid contains growth promoting factors for the lungs.
5. Brain damage and Intracranial haemorrhage: With severe chorioamnionitis, intraventricular haemorrhage and periventricular leucomalacia may occur with subsequent development of cerebral palsy.
6. Compression Deformities: Facial and skeletal deformities can occur as a consequence of severe prolonged PROM.

MANAGEMENT OPTIONS IN PROM

A. Term PROM (> 37 weeks):

1. *Short Expectant Management:*
   - Spontaneous labour pains will start within 24-48 hours in approximately 80-90% of cases.
   - Short term expectant management is advisable, waiting for spontaneous labour pains, while the patient is under cover of suitable antibiotics and close foetal monitoring.
2. *Termination of pregnancy* is indicated in:
   - Clinical and laboratory signs of infection.
   - Non reassuring foetal condition. (abnormal NST, abnormal US Doppler indices , severe oligohydramnios...etc).
   - Induction of labour by I.V. oxytocin drip: if conditions are favourable for a safe and rapid vaginal delivery.
   - C.S. is done if its indications are fulfilled (e.g.. previous C.S., myomectomy scar, placenta praevia, certain malpresentations, foetal distress, severe oligohydramnios, etc..).

B. Preterm PROM (< 37 weeks):

1. *Expectant Management* is generally adopted until labour pains start spontaneously or a reasonable state of lung maturity is achieved. In all cases the risks of infection is weighed against the risks of prematurity.
   - *The foetus should be monitored* with; daily foetal kick counts, repeated NST, and ultrasound for BPP scores (once or twice weekly). Repeated white cell counts and C-reactive protein determinations to help in detection of early infection.
   - *Prophylactic antibiotics* given to guard against infection.
   - *I.M. corticosteroids in cases < 34 weeks* (to minimize the risks of RDS and IVH), are not contraindicated except if infection is anticipated or already documented.
   - *Expectant management is discontinued* when foetal lung maturity is documented by amniotic fluid L/S/ ratio, or if gestational age of lung maturation has been reached (34-36 weeks).
   - *Pregnancy should be terminated* irrespective of gestational age, whenever the risks of prematurity are small compared with the risk of infection, (34-36 weeks).
2. *Immediate delivery is indicated if:*
   - Spontaneous labour pains start.
   - Evidence that chorioamnionitis is starting.
   - Foetal condition is non reassuring.

40 AMNIOTIC FLUID DISORDERS

Sources of AFL
Volume & characteristics
Composition
Functions
Evaluation & Measurements

Oligohydramnios
- Aetiology, Diagnosis, Complications, & Management
Polyhydramnios
- Aetiology, Diagnosis, Complications, & prognosis
- Management (moderate & severe cases)

SOURCES OF AMNIOTIC FLUID

The amniotic fluid is a dynamic structure, being continuously produced & absorbed from both maternal and foetal sources, with the foetus contributing more than the mother in its formation.

The main source of amniotic fluid is the foetal urine. Other important sources include transudation from the foetal and maternal circulation and secretion by the amniotic epithelium.

VOLUME AND CHARACTERS OF AMNIOTIC FLUID

The amniotic fluid volume (AFV) depends largely on the gestational age. It is determined by a balance between production and absorption keeping constant volumes at different gestational ages.

Amniotic fluid volume increases gradually during pregnancy to reach a peak of 0.8 – 1.5 litres at term. It then decreases if pregnancy is prolonged beyond term.

The amniotic fluid is characterized by having a clear **aspect**, pale **colour** and a slightly alkaline **pH** (nearly 7.0).

COMPOSITION OF AMNIOTIC FLUID

The amniotic fluid is **99% water** containing many nutrients such as carbohydrates, lipids and proteins as well as hormones and enzymes.

It also contains **foetal excretions** such as uric acid, urea, creatinine and sodium chloride as well as lanugo hairs and vernix caseosa (*white flakes of foetal sebaceous secretions).*

In addition to the above, the amniotic fluid contains many different hormones, enzymes, and mediators as cytokines.

FUNCTIONS OF AMNIOTIC FLUID

- **During Pregnancy:** The amniotic fluid serves in protection of the foetus from external trauma. It maintains a constant foetal temperature, provides a medium for free foetal movement, and muscular development, a medium for foetal excretion, and a source of nutrients for the foetus. It finally prevents adhesions between foetal skin and amniotic sac.
- **During Labour:** Amniotic fluid prevents cord compression. The bag of fore-waters helps in cervical dilatation, and after ROM it serves in sterilization of the birth canal.

EVALUATION OF AMNIOTIC FLUID VOLUME

- **Amniotic fluid pockets** measurements in both longitudinal and A.P. diameters
- **Amniotic fluid index (AFI):** in which the vertical diameters of the largest pocket of fluid in each of the four quadrants of the uterus are measured in cm. The sum of the 4 quadrant measurements represents the AFI: .

Normal AFI (10–15), **Oligohydramnios** (< 5.0), **Polyhydramnios** (> 25).

I. OLIGOHYDRAMNIOS

DEFINITION

Amniotic fluid volume below the 5th percentile for gestational age or less than **500 ml.** By ultrasound (U.S.), oligohydramnios is defined as an **AFI < 5,** or largest fluid pocket **< 2.0 cm**

INCIDENCE: It affects around 3-4% of all pregnancies.

AETIOLOGY

1. Placental insufficiency.
2. Undiagnosed PROM: may be confused with increased vaginal discharge by the patient.
3. Foetal congenital anomalies: Renal anomalies are the most common cause.
4. Drugs e.g. Indomethacin which reduces foetal urine production.

DIAGNOSIS OF OLIGOHYDRMANIOS

1. **History** of leaking amniotic fluid in cases or ROM.
2. **Symptoms:** The patient does not feel progressive abdominal enlargement with advancement of pregnancy.
3. **Signs**: Small abdominal girth and decreased fundal level in relation to gestational age.
4. **Ultrasound diagnosis and benefits:**
   - Diagnosis of oligohydramnios with an AFI< 5
   - Detection of the cause. (e.g. placental insufficiency, renal agenesis, postmaturity...)
   - Evaluation of foetal well-being (BPP score, umbilical and cerebral artery Doppler)

COMPLICATIONS OF OLIGOHYDRAMNIOS

1. **Umbilical cord compression** causing foetal hypoxia with its complications such as meconium aspiration and IUFD.
2. **Pulmonary hypoplasia** and contracture limb deformities, and amniotic band formation, especially with severe prolonged oligohydramnios before 26 weeks gestation.

MANAGEMENT OF OLIGOHYDRAMNIOS

A. **Pregnancy termination**: Considered in patients with placental insufficiency or lethal foetal congenital anomalies e.g. renal agenesis.

B. **Amnio-infusion:**
   - Repeated injection of 250-350 ml warmed saline into the uterus via amniocentesis.
   - Done under ultrasound guidance to improve visualization and help detect foetal anomalies.
   - It may also be done during labour (trancervical infusion), to prevent umbilical cord compression.

II. POLYHYDRAMNIOS

DEFINITION

- Amniotic fluid volume above the 95th percentile for gestational age, or more than **2000 ml** .
- By U.S. it is defined as an AFI ≥ 25 cm or a single pocket of amniotic fluid ≥ 8 cm in the largest vertical diameter.

INCIDENCE :It affects 0.4-1.5% of pregnancies.

AETIOLOGY

The presence of mild late occurring polyhydramnios is frequently encountered without real maternal or neonatal hazards. However in many cases the presence of polyhydramnios especially if early, severe, and rapidly accumulating, reflects a serious underlying maternal or foetal problem.

A. **Idiopathic:** The majority of cases of polyhydramnios are idiopathic. An imbalance may occur between foetal and maternal amniotic fluid production and absorption. The cause may not be detectable even after delivery.

B. **Foetal causes:**

1. Twins especially uniovular with twin to twin transfusion syndrome.
2. Foetal anomalies as:
   - *Anencephaly*: due to passage of foetal CSF into the amniotic fluid, foetal polyuria due to absent secretion of ADH, and failure of foetal swallowing.
   - *Oesophageal and duodenal atresia* due to failure of foetal swallowing of the liquor.
   - *Obstruction of the foetal venous circulation* leading to oedema of the placenta and increased transudation as in cases of foetal liver cirrhosis and erythroblastosis foetalis.
3. Placental chorioangioma.
4. Large placenta: due to increased area of chorionic villi available for transudation.

C. **Maternal causes:**

- Diabetes mellitus: due to increased osmolarity of the amniotic fluid due to increased glucose concentration & foetal polyuria associated with foetal hyperglycemia.
- Severe generalized oedema: Cardiac, renal or nutritional.
- Pre-eclampsia due to placental oedema.

DIAGNOSIS OF POLYHYDRAMNIOS:

1. **Maternal symptoms:**
   - Progressive abdominal enlargement in a short period of time.
   - Respiratory embarrassment due to pressure on diaphragm and lung restriction.
   - Pressure symptoms resulting in abdominal discomfort, and lower limb oedema.
2. **Abdominal Examination:**
   - The abdomen shows over distension and excessive striae.
   - The fundal level is higher than expected for the period of amenorrhea.
   - Foetal parts are difficult to feel but could be felt by dipping.
   - Twins, malpresentations & non-engagement are common.
   - Marked external ballottement, and a fluid thrill may be elicited.

3. **Ultrasound Diagnosis and benefits:**

- Diagnosis of polyhydramnios: AFI ≥ 25, or long pocket > 8.0 cm.
- Evaluation of severity (mild :pocket > 12 cm, severe pocket >16 cm).
- Detection of the cause of polyhydramnios e.g. anencephaly, multiple pregnancy.
- Evaluation of foetal number, presentation, weight and well-being (Doppler and BPP score).

Differential Diagnosis

From other causes that result in a fundal level disproportionate to the gestational age as with miscalculation, multifoetal pregnancy, oversized foetus, and associated uterine myomata and ovarian cysts.

Clinical Types of polyhydramnios

Acute Hydramnios	Chronic Hydramnios
Commonest cause is Uniovular twins and foetal anomalies	
Very rare affecting 1/3000 pregnancies	*More common (0.5-1.5%)*
Develops before 20 weeks	*Usually develops after 20 weeks*
Rapid accumulation of liquor	*Gradual accumulation of liquor*
Usually ends in abortion	*Usually ends in preterm labour*
Marked pressure symptoms	*Less marked pressure symptoms*

COMPLICATIONS AND PROGNOSIS

1. **Effect on pregnancy:**
   - Preterm labour is the most important effect. Acute and early cases may cause abortion.
   - Maternal respiratory distress and discomfort (only in severe and acute cases).
2. **Effects on labour:** higher incidence of:
   - Inertia (dysfunctional labour)
   - Malpresentations (higher incidence of C.S.)
   - PROM (increased intrauterine pressure).
   - Cord presentation and prolapse (foetal distress and urgent C.S.).
   - Accidental haemorrhage if ROM occurs suddenly (acute decrease intrauterine Pressure).
   - Postpartum haemorrhage (atonic with over distension, and traumatic with malpresentations.)
3. **Effects on the foetus**: higher incidence of:
   - Congenital anomalies are commonly associated with polyhydramnios.
   - Prematurity, due to PTL associated with uterine over distension and foetal anomalies.
   - Asphyxia due to: Cord prolapse, or accidental haemorrhage.

MANAGEMENT OF POLYHYDRAMNIOS

I. Chronic polyhydramnios:

The majority of polyhydramnios cases are **mild**, slowly developing cases, with tendency to late occurrence (3rd trimester). Such cases do not require special treatment. Most of them will go into spontaneous labour, probably near term, with good foetal and maternal outcome.

II. Acute polyhydramnios:

Early occurring, rapidly accumulating cases are less common. They usually reflect a severe underlying maternal, foetal or neonatal problem that needs thorough investigation, and are commonly associated with higher maternal morbidity, poor foetal outcome, and higher incidence of preterm labour (PTL).

The management of polyhydramnios should be directed towards:

1. Establishing the cause if present: (to determine foetal prognosis, and exclude anomalies).
2. Relieving maternal discomfort when present (if necessary do amnio-drainage).
3. Assessment of the risk of PTL, due to uterine over distension.
4. Control of maternal diabetes if this was the underlying cause.

A) Mild and Moderate Polyhydramnios: (conservative)

There is no need for any intervention in such cases, apart from reassurance, and establishing the underlying maternal or foetal causes if present. Most of these cases will have spontaneous labour pains usually few weeks before their expected delivery date.

B) Severe Polyhydramnios:

a. *Termination of pregnancy:* (If > 37 weeks or evidence of adequate foetal lung maturity)

- Aim: to relieve maternal distress and pressure symptoms.
- Method: According to foetal well being, foetal lie and presentation, placental localization, and maternal general condition etc...
  - Induction of labour
  - Caesarean section.

b. *Conservative approach* (If < 37 weeks, until foetal lung maturity is adequate):

- Amniocentesis: Removing up to 1.5 to 2 litres of amniotic fluid, via inserting a needle transabdominally under ultrasound guidance, in a slow rate to avoid placental abruption and ROM. The procedure may be repeated if required.
- Drugs as Indomethacin: may decrease amniotic fluid volume by decreasing foetal urine production and increased fluid re-absorption by the foetal lungs. However its use is limited by its effect on premature closure of foetal ductus arteriosus.

c. *Close observation after delivery:* for uterine inertia and postpartum haemorrhage.

41 FOETAL AND NEONATAL ASPHYXIA

Intrauterine asphyxia (foetal distress)
Pathophysiology
Causes of FD
Clinical features & diagnosis
Management of FD

Neonatal asphyxia (asphyxia neonatorum)
Causes of NNA
Clinical features & assessment
Management of NNA
- *Prophylaxis*
- *Active management (resuscitation)*

INTRODUCTION:

Intrauterine foeatal asphyxia (IUFA) and neonatal asphyxia (NNA) are serious situations, which might put the foetus (infant) at risk for loss of life or permanent CNS disability. Therefore, properly trained personnel in well-equipped delivery rooms are essential to the anticipation, recognition and prompt management of these serious conditions.

DEFINITION:

IUFA is a state of inadequate oxygenation and inadequate elimination of CO2, which if allowed to be continued, will result in metabolic acidaemia (umbilical arterial blood pH < 7.2).

PATHOPHYSIOLOGY:

1. Foetal **respiratory centres** in the medulla are inhibited by higher centres in the diencephalon.
2. The **mild temporary anoxia** at birth (due to stoppage of the placental circulation) depresses the higher cortical centres, thus releasing the medullary centres from inhibition.
3. The medullary centres become stimulated by:
   a. Sensory stimuli from the skin, muscles and joints (sensory receptors)
   b. Relative increase in carbon dioxide concentration (chemoreceptors)
   c. Rise in blood pressure at birth (pressor receptors).
4. If foetal **anoxia is marked** or **prolonged**, the lower respiratory centres in the medulla together which the vasomotor centres become paralyzed leading to asphyxia and shock.
5. Thus, if prompt resuscitation is not done at an early stage, the **irreversible damage** to the respiratory centre will result in failure of all attempts at recovery.

Causes of Intra Uterine Fetal Asphyxia (IUFA) also known as Foetal Distress (FD):

1. Maternal causes; leading to imperfect oxygenation of maternal blood.
2. Placental causes; interfering with delivering adequate circulation to the foetus.
3. Umbilical cord compression; leads to obstruction of the circulation between foetus the placenta
4. Prolonged Foetal head compression; within maternal pelvis, laeds to depression of respiratory centres

INTRA-UTERINE ASPHYXIA (IUFA)
FOETAL DISTRESS (FD)

CAUSES OF IUFA

I. Maternal causes: (conditions leading to imperfect oxygenation of maternal blood)

- Severe anaemia, Haemorrhage & shock, Respiratory failure, and heart failure.
- Eclamptic convulsions, advanced pulmonary T.B., pneumonia, and pulmonary oedema.

II. Placental causes:

- Placental compression: interfering with its circulation as in tonically contracted uterus, prolonged labour after rupture of the membranes or as a method of control of bleeding in placenta praevia.
- Placental separation as in accidental haemorrhage.
- Placental insufficiency: (extensive degeneration, multiple infarcts & abnormally small placenta).

III. Causes in th eumbilical cord: Obstruction of the circulation, which may be due to:

1. Tight coils around the neck.
2. True knots of the cord.
3. Prolapsed cord.
4. Compression of the vessels by haematoma of the cord or by blades of the forceps.
5. Rupture of vasa praevia.

IV. Prolonged foeatl head compression :

This will cause oedema and ischemia, which interfere with the blood supply of the medulla leading to depression of the respiratory centre. Prolonged compression may be due to:

1. Contracted pelvis (C/P disproportion).
2. Rigid perineum.
3. Intracranial haemorrhage.
4. Forceps application for a long time.
5. Depressed fracture.

CLINICAL FEATURES OF IUFA (FOETAL DISTRESS FD):

1. **Abnormal Foeatal heart rate (FHR)**
   - *Bradycrdia* (FHR <100 bpm); most dangerous sign
   - *Tachycardia* (F.H.R. >160 bpm)
   - *Irregular FHR* patterns.
2. **Delay of return of the FHR** to their normal rate after uterine contraction. (FHR normally slows down during the uterine contraction, and returns rapidly to normal after it ends).
3. **If continuous electronic FHR monitoring** and CTG would suggest foetal distress:
   a. *Late deceleration* (see assessment of foetal well being).
   b. *Variable deceleration.*
   c. *Loss of beat-to-beat variation in FHR*
   d. *Sinusoidal FHR pattern.*
4. **Passage of meconium in cephalic presentations**, due to relaxation of the anal sphincter due to anoxia and increased intestinal peristalsis.
5. **Foetal acidosis:** Detected by taking blood samples from the scalp of the foetus during labour; pH below 7.2 indicates foetal asphyxia (N. 7.25- 7.35).

MANAGEMENT OF INTRAUTERINE ASPHYXIA (FOETAL DISTRESS)

1. *Elimenation of th ecause of asphyxia if possible:*
   a. The patient should be turned onto her side: this may relieve either umbilical cord compression, or alleviate poor return of blood to maternal heart caused by occlusion of the maternal aorta or IVC by the gravid uterus.
   c. Oxytocin infusion, if started, should be discontinued to decrease uterine activity and improve placental perfusion.
   d. Any hypotension should be corrected by position change, intravenous hydration or vasopressor treatment if severe hypotension due to induction anaesthesia develops.
2. Oxygen *(100%)* should be administered to the mother by facemask.
3. *Atropine* given to the mother may be beneficial in some cases of foetal bradycardia.
4. ***If FD is relieved:*** careful observation by EFHR monitoring till delivery is accomplished
5. ***If foetal distress is not relieved within several minutes***, immediate delivery is indicated;.
   - If the cervix is not fully dilated; C.S. is immediately performed regardless presentation
   - If the cervix is fully dilated; delivered by:
     - Forceps : in cephalic presentation, engaged head, CPD excluded
     - Breech extraction: in breech presentation.

POSTNATAL ASPHYXIA

ASPHYXIA NEONATORUM (ANN)

CAUSES OF ANN

1. Persistence of a state of severe intrauterine asphyxia after birth.
2. Obstruction of respiratory passages by mucus, amniotic fluid, blood or meconium.
3. Paralysis of cardiorespiratory centres, due to cerebral haemorrhage.
4. Depression of the respiratory centres by drugs (morphine or pethidine) or narcotics & anaesthetics given during labour.
5. Congenital malformations: e.g. congenital atelectasis of the lungs or congenital abnormality in respiratory or circulatory system.
6. Prematurity (R.D.S.).
7. Congenital debility.

CLINICAL FEATURES OF ASPHYXIA NEONATORUM:

With initial O2 deprivation the new born develops rapid breathing pattern followed by a period of APNOEA.

A) **Primary apnoea:** It represents the initial phase of apnoea, and is associated with a decrease in the heart rate (bradycardia), and loss of muscle tone (hypotonia). In such cases with simple stimulation and exposure to oxygen, normal respiration is resumed.

B) **Secondary apnoea:** If oxygen deprivation persists, it will lead to irregular gasping, followed by secondary apnoea. It is manifested by further decrease in hear rate, B.P. and muscle tone. Secondary apnoea will not respond spontaneously to stimulation and oxygen, but assisted ventilation is essential, otherwise neonatal death occurs.

N.B.: Clinically, primary and secondary apnoea, are indistinguishable.

N.B.: The older classification of asphyxia neonatorum into asphyxia Livida and asphyxia Pallida has been abandoned and is nowadays considered obsolete.

CLINICAL ASSESSMENT OF THE NEW BORN (APGAR score):

This is a clinical assessment of the severity of asphyxia in the newborn suggested by Virginia Apgar (1952). In this system the child's condition is assessed one minute and five minutes after birth utilizing five features. Either 0,1,or 2 is given to each of the 5 feature with a total degree out of ten :

1. Appearance (colour)
2. Pulse (heart rate)
3. Grimace (reflex irritability)
4. Activity (muscle tone)
5. Respiration (respiratory effort)

Table : The APGAR score

	0	1	2
Appearance (color)	Blue or pale	Body pink, limbs blue	All pink
Pulse (heart rate)	Absent	< 100	> 100
Grimace (reflex irritability in response to catheter in nose)	No response	Grimace	Cough or sneezing
Activity (muscle tone)	Limp	Some flexion of extremities	Active movement
Respiration (respiratory effort)	Absent	Slow, irregular	Strong cry

Apgar score should be done at one and 5 minutes after birth

1. One minute Apgar score: determines the need for immediate resuscitation
2. Five minutes Apgar score is useful index of the effectiveness of resuscitation methods, when low is indicative of infant at higher risk of morbidity and mortality (prognostic)

Importance of Apgar scoring system: It determines the prognosis, helps selection of the management, and evaluation of the newborn

MANAGEMENT OF ASPHYXIA NEONATORUM

A) PROPHYLAXIS:

1. **Proper ANC** for early detection of cases at high risk for FD and NNA.
2. **Proper intranatal care:**
   - Careful observation of FHR, avoid prolonged and traumatic labour (e.g. forceps)
   - Proper oxygenation during anaesthesia, and avoid morphia within 3 hours before labour
   - Episiotomy to shorten head delivery when necessary, especially in breech and prematurity.
   - Proper delivery of the after coming head
   - Vitamin K for all premature and breech deliveries
   - Aspiration of the mucus and meconium from foetal larynx before it starts breathing.

ACTIVE MANAGEMENT (Active Resuscitation of the newborn):

Resuscitation of the new born is an excellent example of a **team work** that needs cooperation and harmony between each member, namely the obstetrician, the neonatologist, the anaesthesiologist and the nursing team.

The **first few minutes** in the new born's life may be crucial in determining both its potential for survival and its future health performance which may not be revealed except after several months or even years.

1. Clearing the air passages:

Holding the infant from the feet and aspirating mucus from the mouth and upper pharynx by a rubber catheter.

N.B.: *The infant's head should not be lowered if intracranial haemorrhage is suspected.*

2. Warming the infant:

Warming is necessary to decrease oxygen requirements and to avoid attacks of apnoea.

3. Oxygen therapy : When necessary, may be supplied by:

- Small mask or stream in front of the mouth and nose (O_2 saturation ...).
- Endotracheal tube is indicated if:
  - 1 minute Apgar score <3
  - Persistent Apnoea.
  - Persistent Bradycardia <100.

4. Artificial respiration: by:

- Endotracheal tube with intermittent positive pressure insufflation
- Mouth to mouth breathing until endotracheal tube is available.

5. Cardiopulmonary resuscitation:

Cardiac resuscitation together with Endotracheal entubation (or mouth to mouth breathing)

- No audible heart beats or
- Heart rate < 100.
- Thumbs are put at the junction of lower and middle 1/3 of sternum to compress the chest gently 100 times per minute.

6. Use of Drugs:

- ***Nalorphine:*** ½ mg into umbilical vein if asphyxia is due to morphia.
- ***Sodium bicarbonate 8.4%:*** If the infant develops acidosis with severe asphyxia.
- ***Epinephrine:*** May be used for cardiac resuscitation (if absent heart beats). Up to 0.5 cc are injected either into *umbilical vein* or *intracardiac*
- ***Antibiotics:*** To prevent pneumonia especially if resuscitation has been difficult.

42 FOETAL BIRTH INJURIES

Foetal birth injuries represent an important commonly avoidable cause of neonatal morbidity and mortality. They vary from minor skin abrasions to severe intracranial haemorrhage. Prevention of serious birth trauma depends mainly upon the art of obstetrics and the experience of the obstetrician, and is considered a reflection of the improvements in antenatal and perinatal care.

TYPES OF FOETAL BIRTH INJURIES

A) Bone injuries:

1. Skull fracture:
   - *Aetiology:*
     - Difficult forceps delivery.
     - Delivery through a contracted pelvis.
   - *Types:*
     - Fracture vault: linear or depressed (associated with intra-cranial haemorrhage so needs surgical intervention).
     - Fracture base: usually associated with intra-cranial haemorrhage.
     - Fracture mandible.
2. Others bone injuries:
   - Spine injuries.
   - Fracture humerus
   - Fracture femur.
   - Fracture clavicle.
   - Dislocation of hip.
   - Dislocation of shoulders.

B) Soft tissue birth injuries:

1. Intra-cranial haemorrhage 9over compression of cranial bones).
2. Cephalhaematoma (instrumental trauma especially ventouse).
3. Nerve Injuries (undue traction on neck, shoulders, and arms).
4. Visceral and Muscle Injuries.
5. Eye Injury.
6. Injury of hymen, or anal sphincter especially in breech presentation, during examination.
7. Skin and scalp Injuries by the scalpel on opening the uterus in a C.S.

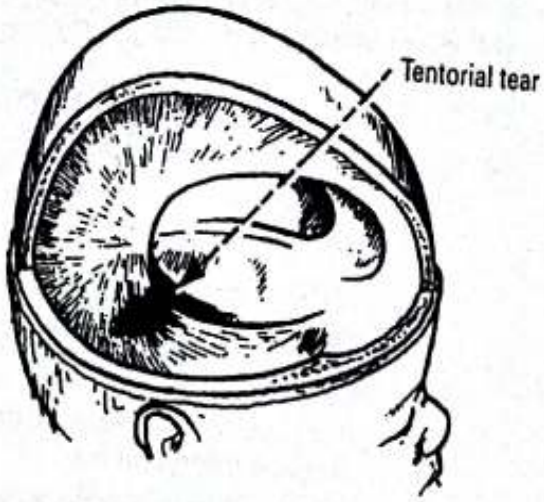
1. INTRA-CRANIAL HAEMORRHAGE

AETIOLOGY:

1. Prematurity, due to:
   - Fragile blood vessels.
   - Hypoprothrombinaemia.
   - Increased susceptibility to birth trauma.
2. Breech delivery: due to sudden compression and decompression of cranial bones.
3. Excessive compression, due to:
   - Excessive moulding, in cases of cephalopelvic disproportion.
   - Excessive compression by forceps (oblique application or persistent locking).
4. Asphyxia: leads to hypoxia of the walls of blood vessels with subsequent leakage.
5. Hemorrhagic disease of the newborn.

Sites of Haemorrhage:

1. Intra-ventricular haemorrhage.
2. Intra-cerebral haemorrhage.
3. Subdural haemorrhage.
4. Subarachnoid haemorrhage,



N.B.: *Subdural and subarachnoid haemorrhages usually develop with traumatic delivery.*

The vein of Galen is torn due to tear in dura at jnction of falx cerebri with tentorium cerebelli (that results from excessive moulding due to increased antero-posterior diameter of the head)

CLINICAL PICTURE:

1. Stillbirth or neonatal asphyxia.
2. Drowsy, refuse suckling with sudden sharp cry.
3. Convulsions and rigidity.
4. Tense bulging anterior fontanelle.
5. Vomiting.

DIFFERENTIAL DIAGNOSIS:

1. Asphyxia neonatorum.
2. Neonatal convulsions.

INVESTIGATIONS: Brain CT scan.

TREATMENT:

Prophylactic Treatment

1. Breech delivery: see breech presentation.
2. Premature delivery: see prematurity.
3. Vitamin K for the mother (10 mg IM early in labour when we suspect difficult delivery)
4. Careful forceps application.

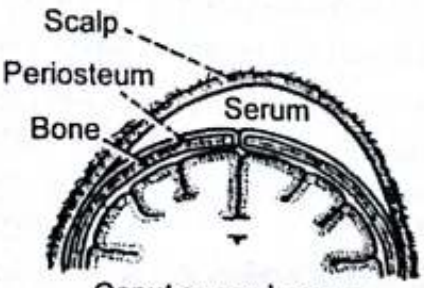
Active Treatment

1. Resuscitation with minimal handling.
2. Chloral hydrate, Magnesium sulphate 50% 1 cc and Luminal.
3. NaCl per rectum for oedema.
4. Dehydrating measures even lumbar puncture.
5. Vitamin K for the foetus (1 mg IM).

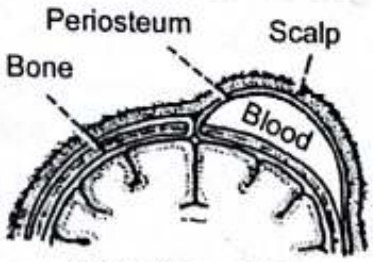
N.B.: Penicillin is used for prophylaxis against infection.

2. CEPHALHAEMATOMA (Sub-periosteal haematoma)

		Caput succedaneum	Cephalhaematoma
Cause		*Obstructed labour*	*Forceps or ventouse*
		Ventouse	*Difficult delivery through contracted pelvis*
Appearance		*At birth*	*Few hours after birth*
Character	*Edges*	*ILL defined*	*Well defined*
	Skin	*May be ecchymotic*	*Normal*
	Sutures	*Overlap sutures and cover more than 1 bone*	*No overlap & limited to one bone*
Complications		*No*	*Calcification, infection and hyper-bilirubinaemia*
Treatment		*No treatment (disappears after 1 -2 days)*	*Expectant treatment (disappears within few weeks)*



Caput succedaneum



Cephalhaematoma

3. NERVE INJURIES

I. Facial Nerve Palsy:

Cause: Compression of the nerve by blade of forceps results in oedema and haematoma around the nerve.

Clinical picture: *unilateral and temporary*

- Absent nasolabial fold.
- Angle of the mouth is deviated to the healthy side.
- Absent blinking on the affected side.

Treatment:

Conservative management. May need corticosteroids.

II. Brachial Plexus Injury:

Cause: Forcible lateral flexion of the head during delivery causes damage of the roots of brachial plexus (oedema and haematoma around the nerves) especially in cases of shoulder dystocia.

Clinical picture:

A) Upper injury (Erb's palsy):

- Injury to C5 and C6.
- Characters: Policeman tip position:
  1. The affected limb is adducted to the body and internally rotated.
  2. Elbow is extended.
  3. Wrist is flexed.

Erb' palsy

B) Lower injury (Klumpke's palsy):

- Injury to C7, C8 and T1.
- Characters:
  1. Wrist drop.
  2. Absent grasp reflex.
  3. Paralysis of small muscles of hands (atrophy).

Treatment:

- *Upper injury (Erb's palsy):* Fixation of the affected limb in pharaoh's position.
- *Lower injury (Klumpke's palsy):*
  - Physiotherapy.
  - Rarely, it may need plastic correction.

4. VISCERAL AND MUSCLE INJURY

A) VISCERAL INJURY:

- e.g. liver, spleen.
- It may occur during breech delivery.

B) MUSCLE INJURY: (Especially sternomastoid muscle)

Cause: due to forcible traction on the head (tilting of the head towards the affected side)

Clinical picture: It may subside or cause permanent torticollis.

Treatment: by passive stretching of muscle several times/day.

43 THE PUERPERIUM

- *Genital Involution*
- *Breast Evolution*
- *Metabolic changes*
- *Mental changes*
- *Physiological changes*
- *Endocrine changes*
- *Management or conduct of the puerperium*
- *Puerperal Exercise*

INTRODUCTION

Puerperium is the few weeks after delivery. It commences after delivery of the placenta and is completed after **four** to **six weeks** later.

During this period genital organs involute, metabolic changes of pregnancy reverse and lactation is established. However, some features of nulliparity are never regained like nipple color, skin striae and skin coloration.

GENITAL INVOLUTION

A) The Uterus:

- *Immediately after delivery* it **weighs** about one k gm, and is felt at or just below the umbilicus.
- *By the end of the first week* it is felt three-finger breadth above the pubes, and after another week it is no longer felt per abdomen.
- *Muscles* of the uterus undergo *autolysis* by proteolytic ferments starting at early puerperium.
- *Placental bed vessels* undergo thrombosis and are replaced by *Elastin*.
- *The decidua* casts off and is replaced by regenerating endometrium (10th day), except at the placental bed where it takes six weeks.
- ***Lochia*** is postpartum genital discharge; it consists of blood and necrotic decidua. The quantity of the lochia diminishes gradually and its color changes from ;
  - Red (Lochia Rubra), to yellow (Lochia Serosa), to white (Lochia Alba).

N.B.: Persistent red lochia for more than two weeks points to the possibility of retained placental fragments.

N.B.: Foetid lochia may result from its accumulation in the posterior vaginal fornix and decomposition not necessarily infection.

B) The Cervix:

- Is normally stretched at delivery and the external os is split and appears as a transverse slit.
- Cervical ligaments involute and gradually regain their role in preventing genital prolapse.

Nulliparous cervix

Parous cervix

C) The Vagina:

- Immediately after delivery the vagina is capacious swollen and bruised.
- Rugae reappear three weeks after labor.
- Pelvic muscle tone and strength are gradually regained.

D) The Vulva:

- The vulva gapes for two to three weeks after delivery but gradually revert to non-pregnant state but with less labial fat and separated labia minora
- Remnant of hymeneal tags known as carunculae myrtiformis.
- Episiotomy and vulval tears may leave tender scar resulting in dyspareunia.

BREAST EVOLUTION

- Prolactin initiates milk secretion on prepared breast (oestrogen & progesterone prepares the breast during pregnancy).
  - For the first three days after labor, the breast secretes colostrum (with high protein and immunoglobulin, less carbohydrate and fat).
  - Secretion of milk commences on the third day and may be associated with marked breast congestion and some pain.
  - Breast-feeding encourages mother-infant bond and provides a source of nourishment quite adapted for the baby needs.
- Lactation may suppress maternal fertility and although lactational amenorrhea is one of the most natural ever known contraceptive method, it should never be considered a reliable method of contraception.

METABOLIC CHANGES

- **Salt and Water:**

  There is a tendency for salt and water retention during pregnancy; this accumulated water is excreted in the first few days of puerperium. As a result, edema of the feet disappears and the weight of the patient diminishes.

- **Blood:**

  - Due to reduction in plasma volume after labor the haematocrit value rises and reaches its normal value by the fifth postpartum day.
  - Both leucocytes and platelets show sharp rise in their count after labor but normalize few days later.
  - An increase in clotting factors during the first 10 days after delivery is associated with a possible risk of deep vein thrombosis.

- **Urine:**

  - Lactose appears in the urine with commencement of lactation.
  - There is an increase of urinary nitrogen due to autolysis of uterine muscles

- **Skin:**

  Skin pigmentation disappears gradually; but may persist as a feature of previou

MENTAL CHANGES

- The psyche certainly changes in pregnancy. Mild degree of depression and emotional liability are common in normal puerperium, is usually self limited lasting for few days (postpartum blues) and needs only reassurance.
- However, rarely women may develop major depression, puerperal psychosis or even schizophrenic reaction. In these women psychiatric intervention is a must.

PHYSIOLOGICAL CHANGES

- **Blood pressure:**

  Cardiac output diminishes after labor and the blood pressure normalizes rapidly unless the case has underling hypertensive disorder or develops residual hypertension.

- **Temperature:**

  Reactionary rise of the temperature can occur after prolonged labor but such rise is transitory, limited to the 1st day, and does not exceed 38°C. Low grade fever may also occur with breast engorgement.

- **Pulse:**

  Pulse slows down after labor and tachycardia after labor should be a cause of concern, it may point to hidden bleeding, infection or embolism.

- **Bladder:**

  - Postpartum diuresis is a feature of early postpartum days.
  - Reflex spasm of the bladder sphincter from painful perineal lesion could result in retention of urine, while difficulty in bladder evacuation may result in excess residual urine.
  - Transient stress urinary incontinence is not uncommon due stretch of the bladder sphincter
  - Peptonuria, lactosuria and creatinuria are features of early puerperium.

- **Bowel:**

  - Constipation: is common due to; to lax abdominal muscles, weak pelvic floor and gaseous distension.
  - Anal incontinence; may occur early due to excessive stretch of the pelvic floor in traumatic labor especially for flatus and loose stool. This usually improves by the end of puerperium.

ENDOCRINE CHANGES

- After delivery, the decrease in estrogen and progesterone levels and continued rise of prolactin concentration result in milk secretion.
- Suckling-induced signals stimulate prolactin secretion and milk yield as well as oxytocin secretion and milk letting. But milk production requires also synergistic action of growth hormone, cortisol and thyroxine.
- The degree to which breast-feeding suppresses GnRH secretion is modulated by nutritional status of the mother, her body mass, psychological adjustment and the intensity of breast-feeding.
- About 40% of lactating mothers have lactational amenorrhea with circulating estradiol in the menopausal range and may continue on this status for six months after labor.
- Menstruation returns by six weeks in non-lactating mothers.
- Estrogen containing contraceptives can affect milk yield and milk composition and some disfavor their use during lactation.

MANAGEMENT OF NORMAL PUERPERIUM

- **Observation:** For vital signs, uterine size and tone, lochia and urine output.
- **Diet:** a well balanced diet of not less than 2600 Cal/day is to be given from the first day. Fibers and extra fluids are important to avoid constipation.
- **Sleep:**
  - Nursing the early neonate, after pains, painful perineal sutures are among the causes of postpartum insomnia.
  - Reassurance, time management, activity regulation and treating pain are essential to ensure good sleeping periods.
- **Rest:** Early ambulation is advised few hours after labor. It improves the patient psychology, helps venous drainage, prevents DVT and allows better evacuation of the bladder and rectum.
- **Breast care:**
  - Breast preparation for lactation starts in the last weeks of pregnancy. The breasts are to be washed, comfortably supported by fitting brassieres and colostrum is expressed out of the nipples. Inverted nipples should be treated antenatally by Waller shield.
  - The child should be put to the breast as soon after delivery as possible. Suckling is initially limited to 3 minutes on each side but subsequently the period is to be increased.
  - The mother has to sit comfortably; the whole nipple is placed in the baby's mouth taking care to maintain a clear airway.
- **Abdominal binder:**

  Binder is not necessary except after twin delivery and should extend from above the umbilicus to the greater trochanter and only for few days.
- **Perineal care:**
  - After each bowel act the perineum should be washed from before backward with antiseptic solution. In general, the perineum should be kept dry and clean.
  - The perineum may become edematous and painful after labor; local ice packs or hypertonic saline swabs are helpful.
  - Perineal skin sutures are usually spontaneously absorbed in 3-4 weeks
- **Bladder care:**
  - Bruising of the vulva during labor, perineal stitches, lax abdominal wall, and recumbent position are among the causes of urine retention after labor.
  - Residual urine in excess of 50 ml is common after labor and the mother should be encouraged to evacuate her bladder.
  - Should catheterization be necessary, strict asepsis is to be followed.
- **Bowel care:**

  Provide fibers and extra fluids to avoid constipation.

  Use of mild laxatives or rectal glycerine suppository once daily is helpful in severe cases.
- **Psychological support:**

  Motherhood is accompanied by psychological stress. Anxiety, the troubles made by the newcomer and lack of confidence are common and support must be offered. Psychological support allays the anxiety, relieves postpartum blues, and prevents puerperal psychosis

- **Postpartum vaccination:**
  - No live vaccine to be given during pregnancy.
  - Rhesus negative mother giving birth of Rhesus positive baby should receive 300 microgram RhoGAM immediately after labor and not later than 72 hours.
  - After delivery if the patient is rubella negative she should be vaccinated.
- **Discharge from the hospital:**
  - Advantages of early discharge are great mother satisfaction, reduction of the hospital costs and avoidance of hospital cross-infection.
  - Disadvantages of early discharge are poor housing, lack of rest, medical problems may arise that needs readmission.
  - In general patients delivered normally are to be discharged on the second postpartum day.
- **Postnatal visits:**
  - At least two postnatal visits are recommended; the first after two weeks and the second at the end of the puerperium.
  - In each visit the woman and her baby should be formally examined. Enquiry is made about breast-feeding, menstrual resumption, sexual satisfaction and dangerous symptoms e.g. pain, bleeding and abnormal discharge.
  - Discuss with patient methods of contraception suitable for her, and let her share in the choice.
- **Puerperal exercise**
  - Postnatal exercise can be done, whether it was normal delivery, forceps, caesarean section, and can still be done in presence of perineal sutures.
  - The aim of these exercises is to improve venous circulation, help genital involution, restore muscle tone, prevent genital prolapse, and encourage good posture.
  - After delivery the patient can start breathing exercise and ankle exercise. From the second postpartum day onward she can start pelvic floor exercise, abdominal wall exercise and pelvic rocking. Women with perineal sutures can postpone pelvic floor exercise till one week.
  - On the second postpartum day, she is allowed out of bed and she has to correct her posture, avoid stooping and sharp bending with straight knees.

44 PUERPERAL PYREXIA And Puerperal Sepsis

DEFINITION

The most accepted definition is;

- A **temperature of 38°C** or higher which
- lasts for **> 24hours** and or **recur within 24 hours**
- during the **first 21 days postpartum** exclusive of the first 24 hours
- Temperature should be taken by mouth by a standard technique at least four times daily.

AETIOLOGY OF PUERPERAL PYREXIA

1. Puerperal sepsis.
2. Breast infection.
3. Urinary tract infection.
4. Respiratory system infection.
5. Thrombophlebitis.
6. General causes of fever as typhoid or malaria

Any case of puerperal pyrexia is considered puerperal sepsis until proved otherwise.

This is because pelvic infections are the most common serious complications of the puerperium, and along with preeclampsia and obstetrical haemorrhage, form the lethal triad of causes of maternal deaths.

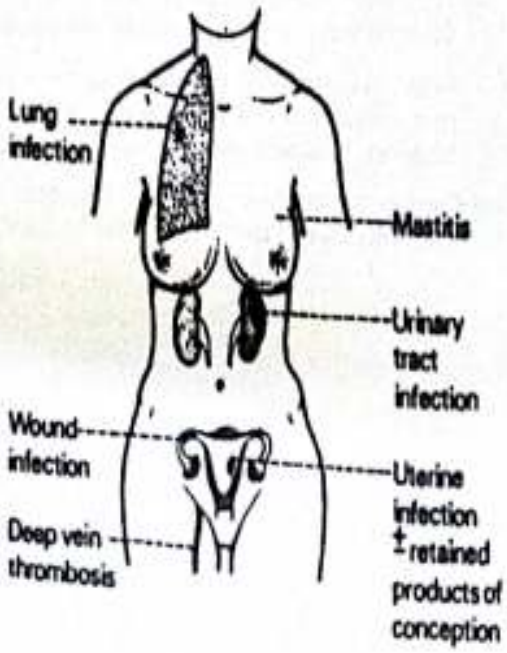


Fig 43:1 Common causes of puerperal pyrexia

PUERPERAL SEPSIS

DEFINITION

It is a type of wound infection of the genital tract that occurs during labour or during the first postpartum 3 weeks.

AETIOLOGY

A. Predisposing factors:

- *General factors* (leading to decreased immunity):
  - Anaemia
  - Antepartum or postpartum haemorrhage
  - Diabetes mellitus
  - Septic focus.
- *Local factors* "in the genital tract":
  - Lack of antiseptic measures.
  - Premature rupture of membranes.
  - Prolonged labour with excessive vaginal examination.
  - Retained parts of placenta or membranes.
  - Intrauterine manipulations (e.g. manual separation of placenta)
  - Instrumental delivery with possible genital tract lacerations.
  - Cerclage sutures.

B. Sources of infection:

- *Exogenous (most important):* from attendants by droplet infection or from unsterilized instruments.
- *Endogenous:* organisms are already present in the genital tract before delivery, e.g. vaginitis or cervicitis.
- *Autogenous:* organisms reach the genital tract from remote site via blood stream e.g. tonsillitis or respiratory tract infection, carious teeth.....etc.

C. Causative organisms:

- *Anaerobic streptococci (most common):* It is non pathogenic and in the presence of dead tissue, it becomes pathogenic and produces mild infection.
- *Group A haemolytic streptococci:* Causes severe infection.
- *Others:* As Staphylococci, E.coli, non-haemolytic Streptococci, Pseudomonas and Clostridia Welchii.

Acute putrid Endometritis	Acute septic Endometritis
Mild type.	*Severe type.*
Caused by low virulent organism (anaerobic streptococci).	*Virulent organism (haemolytic streptococci).*
Good patients resistance.	*Low.*
Uterus is subinvoluted.	*Involuted.*
Uterine cavity is filled with necrotic tissue → necrotic infected discharge.	*It is lined by a pyogenic membrane → scanty discharge.*
Leucocytic barrier is found under the endometrium → limits spread of infection.	*No such barrier.*

PATHOLOGY

1. **Primary Sites of Infection:**

   a. ***The uterus:*** this is the commonest primary site.

   b. ***Infected cervical, vaginal, and perineal lacerations:*** appear usually as ulcers with dirty base, greenish discharge, with surrounding oedema.

2. **Secondary Sites of Infection (local spread):**

   a. Parametritis (pelvic cellulitis):

   - Infection reaches this site via lymphatic spread or directly from cervical or vaginal tears.
   - It is usually unilateral, and may form a mass of exudates pushing the uterus to opposite side.
   - If abscess is formed, it commonly points above the inguinal ligament but may also point in the vagina, rectum, bladder, peri-renal space and occasionally in the gluteal region.
   - It may heal by fibrosis pulling the uterus to the same side.

   b. Salpingo-Oophoritis:

   - Due to lymphatic or vascular spread from the primary sites.

   c. Pelvic thrombophlebitis:

   - Secondary to parametritis or uterine wall veins thrombophlebitis.
   - It may spread to include other pelvic veins or femoral vein or IVC.
   - The affected veins become partially or completely occluded.

   d. Peritonitis:

   - It is either localized pelvic peritonitis (pelvic abscess) or generalized peritonitis.
   - Infection reaches the peritoneum by lymphatics of the uterine wall, along tubal lumen or from cervical and vaginal vault lacerations.
   - In the localized type, effusion collects in cul de sac forming a tender cystic swelling behind the uterus.

3. **Generalized Spread:**

   - Septicemia and septic shock may result from infected emboli in a severe infection with low patient's resistance.

CLINICAL PICTURE: (depends on the pathological type):

1. **Infected lacerations:**
   - Local pain and pyrexia.
   - Wound is hot, red, painful, swollen and covered with purulent exudates.
2. **Local uterine infection:** Endometritis and retained products.
3. **Parametritis:**
   - It starts 7-10 days post delivery, with mild fever, tachycardia, and deep-seated pelvic pain.
   - Locally: usually cervical tear with unilateral tender mass in one fornix.
   - Horse shoe induration around the cervix with extreme tenderness (jumping sign)
   - If abscess is formed it may point in the following site; above the inguinal ligament, the vagina, the rectum, or in the bladder.

4. Salpingo-oophoritis:

- It starts 7-10 days post delivery.
- Generally: there is a deep-seated bilateral lower abdominal pain and tenderness with fever and rapid pulse.
- Locally: tender adnexa with pain on moving the cervix.

5. Pelvic thrombophlebitis:

- It occurs in the second week post delivery.
- It starts with pelvic pain, low-grade fever, rapid pulse inconsistent with degree of pyrexia.
- If the thrombus extends to femoral vein, the whole limb become edematous, white (phlegmasia alba dolens) and not tender.
- If thrombosis extends to IVC, both lower limbs will show massive oedema.

6. Peritonitis:

- *Localized pelvic peritonitis:* giving local pelvic pain and tenderness with accumulation of fluid in the Cul de sac.
- *Generalized peritonitis:* characterized by abdominal distension, vomiting, deterioration of the general condition with shifting dullness.

Pelvic abscess (localized peritonitis)	Generalized peritonitis
• *Pelvic pain and tenderness on the lower abdomen.*	• *Generalized abdominal pain.*
• *Fever and tachycardia.*	• *Same*
• *Tenesmus*	• *Continuous vomiting*
• *Cystic, tender mass in cul de sac*	• *Dehydration*

7. Septicemia:

- The most serious complication of puerperal sepsis.
- Occurs in the first week post delivery.
- There is high shooting fever with tachycardia inconsistent with degree of pyrexia plus rigors.
- It is usually associated with symptoms and signs of generalized peritonitis.
- Finally, skin eruptions with drowsiness and septic shock occurs.

DIAGNOSIS

1. History:

- Pre-existing infection in the genital tract or in the body in general.
- Diseases that lower the patient's resistance as diabetes or anaemia.
- Mode of delivery, premature rupture of membranes and the place of delivery.

2. Examination:

a. General: aiming to

1. Assess the extent of the disease.
2. Exclude other causes of puerperal pyrexia.

3. Discover possible source of infection outside the genital tract. So, look for:
   - Vital signs (pulse, temperature, blood pressure and respiratory rate).
   - Lower limbs for deep venous thrombosis.
   - Cyanosis and purpura (in septicemia).
   - Breast, chest and throat (for infection).

b. Abdominal:

- Tenderness and rigidity.
- Uterine size: subinvoluted uterus.
- Renal angles for pyelonephritis.

c. Local:

- Lochia: amount and nature (excessive, foul odeur).
- Infected lacerations.
- Uterine size and tenderness.
- Pelvic swellings.

3. Special investigations:

- Swabs from vagina and cervix for aerobic, anaerobic cultures and antibiotic sensitivity.
- Urine culture (Catheter specimen).
- Complete blood picture.
- Blood culture at the height of the fever.
- Doppler ultrasound for venous thrombosis (for all pelvic vessels and IVC).
- Chest X-ray for chest infection and blood film for malaria.

PREVENTION OF PUERPERAL SEPSIS

1. During pregnancy:

- Treat any infection especially in the genital tract.
- Improve the general condition of the patient by treating anaemia and diabetes.
- Treat any septic focus.

2. During labour:

- Propoer aseptic and antiseptic measures.
- Avoidance of unnecessary repeated vaginal examination.
- Antibiotics in case of prolonged labour especially with premature rupture of membranes.
- Proper management of genital lacerations.
- Avoidance of retained fragments in the uterus.

3. During puerperium:

- Maintenance of aseptic & antiseptic measures.
- Prevent infected personnel or visitors from visiting the puerperal mothers.
- Sitting in bed to promote lochia drainage.
- Isolation of suspected cases.
- Respiratory and pelvic floor muscle exercises.

TREATMENT:

a. General measures:

- Isolation.
- Light nutritive diet, vitamins, iron and plenty of fluids.
- Frequent measurements of vital signs.
- Analgesics and antipyretics to assure good sleep.

b. Antibiotics:

A combination of antibiotics is given till the results of culture and sensitivity are obtained:

- Cephalosporins for gram +ve organisms.
- Gentamycin for gram -ve organisms.
- Clindamycin or Metronidazole for anaerobic organisms.

c. Promotion of Drainage:

- Fowler's position (Semi-sitting with flexed knees).
- Ergometrin, which increases uterine ability to expel retained placental parts or membranes.
- Removal of retained parts in the uterine cavity (under anaesthesia).
- Removal of sutures in infected wounds.
- If pelvic abscess: surgical drainage if antibiotics fail to cure.

d. Treatment of Complications:

1. Septic thrombophlebitis and DVT:

- Antibiotics.
- Anticoagulants.
- Limb immobilization till fall of the temperature.

2. Generalized peritonitis:

- No oral feeding.
- IV fluids.
- Fix a Ryle tube for gastrointestinal drainage.
- Massive IV antibiotics.

3. Septic shock: See shock in obstetrics

45 INDUCTION OF LABOUR AND ABORTION

Induction of Labour
Prerequisites
Methods
Cervical ripening
Amniotomy
I.V. oxytocin
Prostaglandins

Induction of Abortion
Medical methods
1st trimester
2nd trimester
Surgical methods
Vacuum aspiration[
Abdominal hysterotomy

INDUCTION OF LABOUR

DEFINITION: Artificial termination of a pregnancy by inducing labour

INDICATIONS: Whenever continuing pregnancy will harm the mother or the fetus: e.g.;

- Hypertensive disorders (PE / eclampsia).
- Prolonged pregnancy (Post date).
- Compromised fetus (e.g. IUGR).
- Maternal diabetes mellitus.
- Rhesus iso-immunization.

PREREQUISITES FOR SUCCESSFUL INDUCTION OF LABOUR:

- Ripening of the cervix before the onset of labour makes it favorable for easy dilatation and effacement. It also renders labour more easily inducible whenever indicated. An Unfavorable cervix increases the chances of a difficult prolonged labour, fetal hypoxia and operative delivery.
- The Bishop score takes account of the cervical length, cervical dilatation, cervical consistency, cervical position and station of the fetal head. Scores of 9 or > is favorable for labour induction.

The Bishop score

	0	1	2	3
Dilatation (cm)		*<2*	*2-4*	*>4*
Length (cm)		*>2*	*1-2*	*<1*
Consistency	*Firm*	*Average*	*Soft*	
Position	*Post.*	*Mid Anterior*		
Level	*0-3*	*0-2*	*0-1:0*	*0+*
				Total

METHODS OF INDUCTION OF LABOUR

I. Cervical ripening by Prostaglandins (PGLs):

Prostaglandins are effective in inducing cervical ripening (softening of cervical collagen fibers) which effectively results in success of labour induction.

- *PGL E2 (Dinoprostone);* vaginal tablet or gel
- *PGL E1 (Misoprostol 25 ug);* vaginal tablet inserted into the posterior fornix.

N.B.: repeated small doses of Misoprostol (25-50 ug) can be used to induce uterine contractions

II. Amniotomy (Artificial rupture of membranes)

- This is done to initiate or augment labor, or to allow a fetal scalp blood pH assay.
- The cervix must be at least 2.0 cm dilated to allow safely performing amniotomy
- Amniotomy appears to release a local secretion of endogenous PGLs. It is done by passing a special hook along the fingers or by direct vision using a speculum to rupture the membranes overlying the presenting part, but care must be taken not to damage cervical or fetal tissues.
- The colour and quantity of the liquor removed should be noted.
- Labour may not become established after amniotomy alone and it is usual to stimulate the uterus further by IV oxytocin if contractions are inadequate.
- *Complications*:
  a. Placental separation (abruption): may be caused by sudden reduction in intrauterine pressure specially in polyhydramnios.
  b. Vaginal bleeding: soft tissue and fetal injury should be excluded.
  c. Prolapse of the cord: Specially in ill-fitting presenting pare. FHR monitor is a must after amniotomy.
  d. Infection: Intra-amniotic infection may occur if the interval from amniotomy to delivery is excessive, and both mother and child are at risk. Careful antiseptic techniques and prophylactic antibiotics are strongly recommended whenever delay is anticipated.

III. I.V. Oxytocin drip

- Synthetic oxytocin by continuous IV drip is commonly used after amniotomy to stimulate uterine contractions.
- Doses should be carefully titrated and monitored as the drug is powerful and uterine response to stimulation is generally unpredictable.
- I.V. oxytocin is best administered by a suitable semi-automated infusion system incorporating an accurate drop counter.
- A solution of 2 units of syntocinon in 500 ml of Hartmann's solution (lactated Ringer) is used beginning at a dose of 1 m.IU/min. (10-15 drops/min). This is increased by 1 m.IU/min. every 15 minutes until satisfactory contractions are established.
- ***Complications of Oxytocin Infusion***:
  a. Failure of induction: ripening of the cervix with PGLs should be used first
  b. Hyperactive uterine action: Resulting in dysfunctional labour.
  c. Abnormal FHR patterns: due to foetal hypoxia by over-stimulation of the uterus.
  d. Rupture of the uterus: It is rare in a primigravida, but is more to be expected in grand multiparas, and patients with previous uterine scar 9C.S., hysterotomy, and myomectomy).

INDUCTION OF ABORTION

DEFINITION: Induced termination of pregnancy before fetal viability.

INDICATIONS:

I- Maternal:

- Maternal diseases with pregnancy as cardiac, Diabetes mellitus, Hypertensive disorders
- Severe mental disorders.
- Severe resistant hyperemesis gravidarum.
- Malignancy needing irradiation.
- Whenever a teratogenic drug must be given.
- Some cases of acute hydramnios.

II- Fetal:

- Missed abortion.
- Vesicular mole.
- Fetal congenital anomalies

MEDICAL METHODS FOR INDUCTION OF ABORTION

A. First trimester

1. ***Oral Anti-progesterone; (Mifeprostone 600 mg)*** is effective in early pregnancy termination (5-8 weeks). It is used for 48 hours followed by PGL vaginal pessary.
2. ***Oral and/or Vaginal PGL E1 (Misoprostol 400 ug)***: given in repeated doses, every 4 hours till abortion is accomplished, usually within 24-48 hours.

B. Mid trimester

1. ***Oral and/or Vaginal PGL E1 (Misoprostol)*** 200-400 ug / 4-6 hours in repeated doses .
2. ***High concentration oxytocin*** (50-300IU/500cc over 3 hours) is successful only in cases with ripped cervix.

- **Complications of PGLs:**
  - GIT symptoms: Nausea, vomiting or diarrhea.
  - Cardiac symptoms: Palpitations and mild pyrexia.
  - Reports on uterine rupture and cervical damage, are now emerging after its extensive use, especially in parous women or those with a previous Caesarean section.
- **N.B.;** *Prostaglandins should never be used to augment labour. It might be used to induce labour or to accomplish cervical ripening in term pregnancies.*

SURGICAL METHODS FOR INDUCTION OF ABORTION

A. First trimester:

- Surgical methods are preferred during the 1st trimester due to:
  1. The uterus is small and the contents are not bulky.
  2. The products of conceptions contain no bones.
- Methods:
  1. ***Dilatation and evacuation (D&E):*** Entails cervical dilatation using surgical dilators or Laminaria tent and evacuation of the contents by the ovum forceps followed by endometrial curettage.
  2. ***Dilatation and suction evacuation:*** The technique is similar to D&E, but instead of extracting the conceptus using ovum forceps, we use a suction cannula. This method appears to be with less risk of uterine perforation and can be used for v. mole evacuation whatever the uterine size.
  3. ***Menstrual extraction:*** Early pregnancy (5-7 weeks from LMP) can be terminated as an office setting through modified vacuum evacuation without cervical dilatation or anesthesia. Although it appears simple and easy, it has the higher risk of incomplete evacuation, uterine perforation and infection.

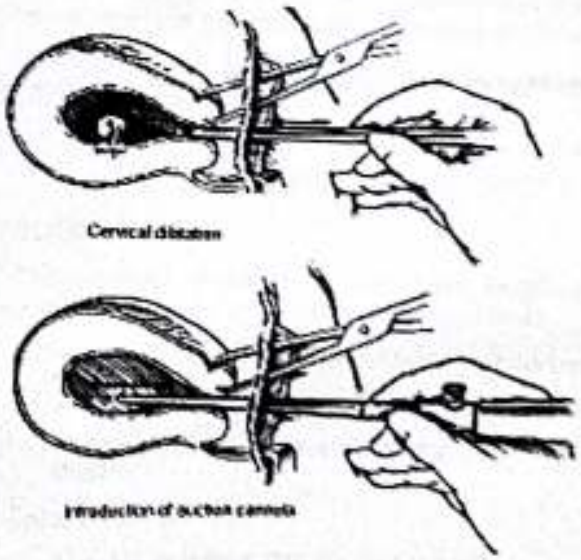


Fig 45: 1 surgical evacuation

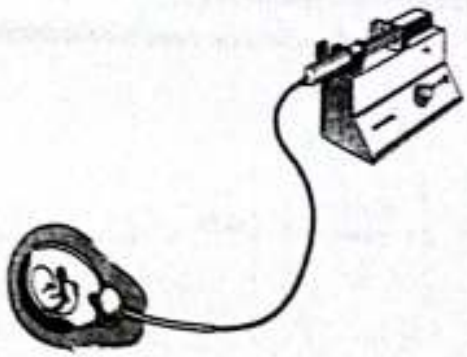


Fig 45: 1 suction evacuation

Complications of surgical vaginal evacuation:

1. Cervical lacerations due to rapid or forcible dilatation.
2. Uterine perforation by sound, dilator, or ovum forceps (rare with suction cannula).
3. Introduction of infection.
4. Hemorrhage and shock: Mostly due to incomplete evacuation.
5. Neurogenic shock if the cervix is dilated with inadequate anesthesia.
6. Complications of general anesthesia.
7. Remote complications as cervical stenosis or incompetence, infertility and intrauterine synaechae.

N.B.: Rh negative women should receive an anti-D IM injection in a dose of 50 ug in 1st trimester and 300 ug in 2nd trimester, for prophylaxis against iso-immunization.

B. Second Trimester:

Surgical methods are now rarely used in second trimester abortion due to the great success of medical agents specially prostaglandins. The following procedures could be done

1. Abdominal hysterotomy:

Emptying of the uterus by the abdominal route through a small suprapubic incision.

- *Indications:*

1. Failed medical induction of 2nd trimesteric abortion by PGL E1 and Oxytocin (rare)
2. Cases with severe life threatening bleeding, that necessitates rapid termination
3. Poor patient condition that cannot withstand long medical induction

- *Procedure:*

- Laparotomy similar to C.S.; performed, during the 2nd trimester on a non viable foetus
- Abdominal incision is usually a transverse Pfannenstiel incision
- Uterine incision may be transverse or sometimes longitudinal due to absence of a well developed lower uterine segment (LUS)

2. Abdominal hysterectomy:

Only done whenever there is an indication for hysterectomy such as cancer cervix

46 INSTRUMENTAL VAGINAL DELIVERY

The Obstetric Forceps
- *Classification of forceps operations*
- *Types of obstetrics forceps*
- *Indications for the use of forceps*
- *Prerequisites for application*
- *Contraindications*
- *Complications*

The Vacuum Extractor (ventouse)
- *Indications for the use of vacuum extractor*
- *Prerequisites for application*
- *Contraindications*
- *Complications*
- *Advantages of the ventouse & forceps*
- *Technical considerations*

***Instrumental vaginal delivery** refers to the use of specially designed instruments, namely the obstetric forceps and the vacuum extractor, to facilitate delivery of the fetal head.*

The decision to choose between both instruments is individualized according to each case and is largely dependant on the clinician's state of experience.

The liberal use of instrumental delivery in obstetric practice has been hindered by the increased maternal and fetal complications associated with their inappropriate use.

In the last 2-3 decades, delivery by C.S. has replaced most of the difficult instrumental techniques.

THE OBSTETRIC FORCEPS

THE INSTRUMENT

- The obstetric forceps is an instrument designed for traction or combined traction and rotation of the fetal head.
- It consists of **two blades**, each having **two curves:**
  - *Cephalic curve* to fit on each side of the fetal skull.
  - *Pelvic curve* to obtain central grip on the head and promote flexion.
- **Each blade consists of:**
  - The Blade proper, that fits on the head.
  - The Shank, that connects the blade to the handle.
  - The Lock, where the two blades cross each other
  - The handle, by which traction is done.
- **Each blade is fenestrated:**
  - To minimize compression
  - To make its weight lighter
  - To prevent slipping as the parietal eminences protrudes through the fenestration

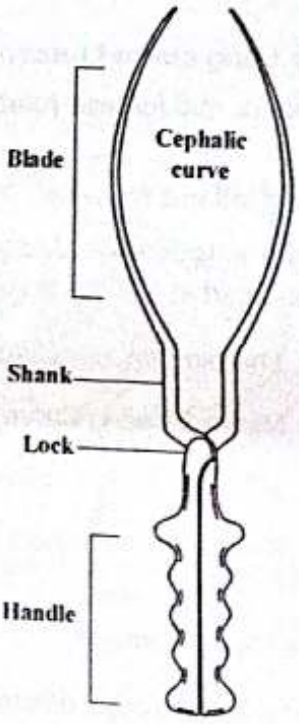


Fig 46:1 The obstetric forceps

CLASSIFICATION OF FORCEPS OPERATIONS

A. Outlet forceps:

- Foetal head is at the perineum
- Sagittal suture in the mid line DOA or DOP; Traction only
- ROA & LOA position; Rotation < 45° and Traction.

B. Low Forceps:

- Head is at station (+2, or more), but not on the perineum
- Rotation is 45° or more (ROA & LOA, ROT & LOT)

C. Mid forceps:

- Foetal head at station (0 to +1)
- Rotation may be > 45 (ROT & LOT, ROP& LOP)

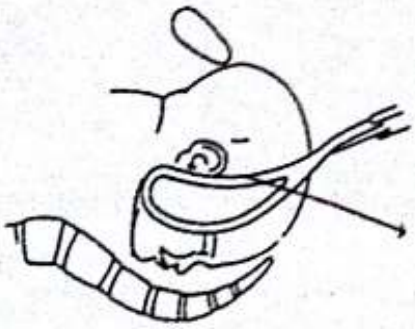


Fig 46:2 Outlet forceps DOA

TYPES OF OBSTETRIC FORCEPS:

1. The Short forceps "11 inches"(shorted shank & handles):

Used for low forceps delivery.

- Curved (Wrigley's forceps).
- Straight (Simpson forceps).

Fig 46:3 the short forceps for outlet traction

2. The Long curved forceps "15 inches":

Used for mid forceps rotation and traction (rarely used in modern obstetrics).

3. The Kielland forceps:

It is a long forceps designed mainly to facilitate traction and rotation of the fetal head in OP positions. It is characterized by:

- The blades are called anterior & posterior.
- Minimal Pelvic Curve: allowing rotation & traction by single application.
- A Sliding lock: to allow application on asynclytic head
- Knobs on the handle that should be directed toward the fetal occiput.

Fig 46:4 kielland forceps for rotation & traction in OP positions

4. The Piper Forceps:

It is a long forceps designed to facilitate delivery of the after coming head in breech presentation.

- *It is characterized by* a long shank with the presence of a perineal curve.
- *Advantages:* It promotes flexion of the fetal head, prevents sudden compression and decompression on the fetal head, and allows safer traction on the after coming head and not on the fetal neck.

1. Prolonged second stage of labour, when maternal exhaustion is eminent.
2. To shorten second stage of labor, in maternal cardiac or hypertensive disorders.
3. Inadequate maternal expulsive forces, as with the use of epidural analgesia.
4. Fetal distress if the cervix is fully dilated with engaged head.
5. Prolapsed pulsating cord when the cervix is fully dilated and engaged head.
6. Some malpositions & malpresentations:
   - Occipito-posterior position after failure of spontaneous rotation.
   - After coming head in breech, to promote flexion and avoid skull compression.

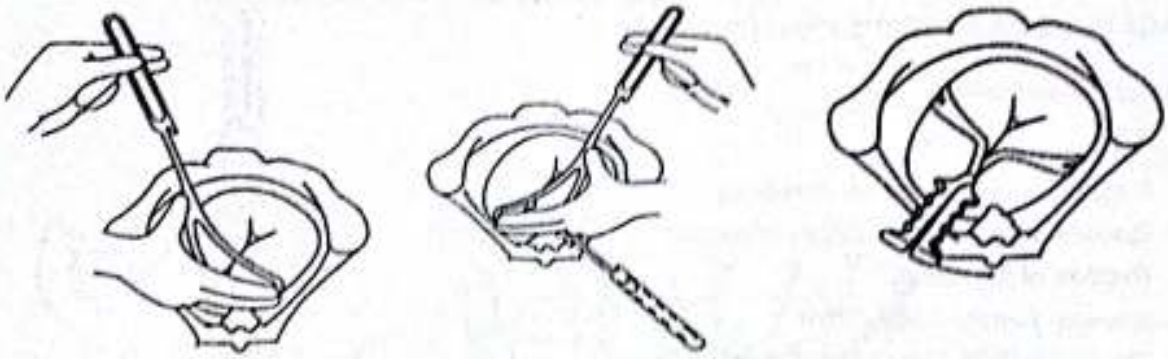


Fig 46:5 Technique of forceps application

1. The cervix should be fully dilated.
2. The head should be engaged.
3. No cephalopelvic disproportion.
4. The membranes (forewater) should be ruptured.
5. Presence of adequate uterine contractions.
6. Antisepsis and anaesthesia.
7. The bladder & rectum should be evacuated.

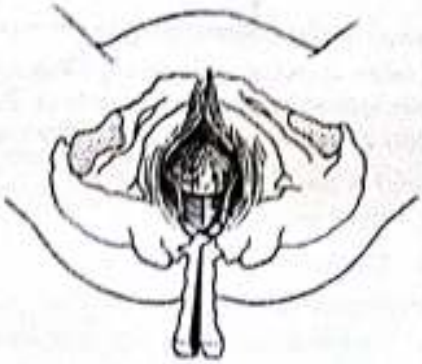


Fig 46:6 traction on footal head after rotation

1. Incompletely dilated cervix
2. Unengaged head
3. Cephalopelvic disproportion or contracted outlet.
4. Intact membranes.
5. Uterine inertia.

N.B: The ideal application of the forceps is the ***Cephalo-pelvic application***, in which the forceps is applied on the sides of the fetal head along the mentovertical diameter, and in the same time along the pelvic side walls. This is only possible in the cases of direct occipito-anterior, direct occipito-posterior or direct mento-anterior positions.

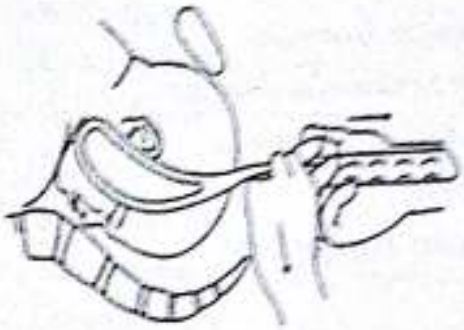


Fig 48: 7 direction of traction on foetal head

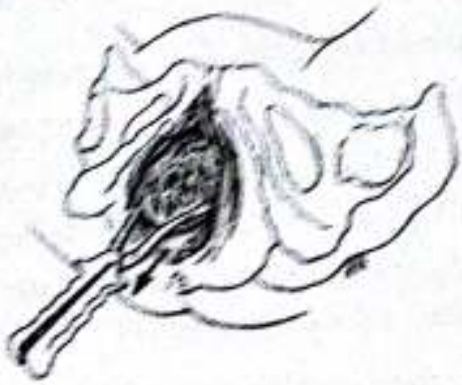


Fig 48:8 traction on foetal head till crowning occurs

COMPLICATIONS OF FORCEPS PROCEDURES:

A) Maternal complications:

1. *Maternal birth injuries:*
   - Vaginal, perineal, and cervical tears.
   - Complete perineal tear (injury of rectum and anal sphincter)
   - Rupture of the uterus.
2. *Postpartum hemorrhage (PPH):*
   - Traumatic PPH, due to maternal birth injuries.
   - Atonic PPH, if applied in cases of poor uterine contractions.

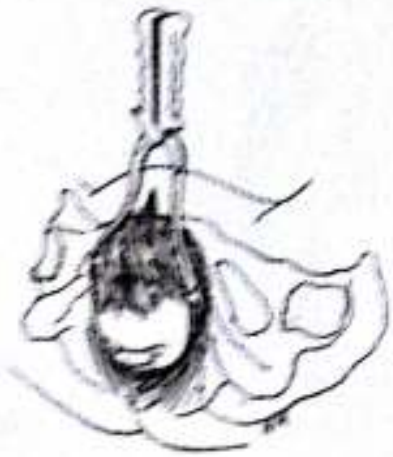


Fig 48:9 Delivery of the head by upwards lifting of the forceps

B) Fetal complications:

1. *Intracranial haemorrhage:*

   Liable to occur with wrong forceps application, leading to marked fetal head compression and elongation of the mento-vertical diameter. This leads to tear of the vein of Galien at the junction of the falx cerebri and tentorium cerebelli.

2. *Head & Skull injuries: e.g.*
   - Skull fractures.
   - Cephal-haematoma.
   - Facial nerve injury.
   - Facial skin bruises and lacerations.

HISTORICAL REVIEW

The introduction of the obstetric forceps in modern obstetrics has been credited to the **Chamberlen's family**, who practiced midwifery in England for four generations, in the 16th century. They kept their forceps as a family secret for nearly 100 years.

During the next 200 years, endless innovations have been introduced to the original simple instrument, including change of materials, introduction of different types of locks, fenestrations on the blade, axes traction pieces, and introduction of the pelvic curve.

Special types of forceps have been designed to facilitate its use in certain situations as with the **Kielland forceps** (especially used for persistent occipito posterior and deep transverse arrest), and the **Piper's forceps** (applied on the after coming head in breech presentation).

The most commonly used forceps nowadays are the short forceps (Wrigley & Simpson forceps), used mainly in low and outlet forceps procedures, especially after the more liberal use of regional (epidural) anaesthesia during labour in many centers.

THE VACUUM EXTRACTOR

INTRODUCTION

The use of ***vacuum–cup deliveries*** to facilitate vaginal birth dates back to the 18th century. The idea was to apply traction on the fetal scalp guiding the head down out of the birth canal. In 1954, Malmstrom, (a Swedish obstetrician), developed the currently used vacuum extractor (the ventouse), which now bears his name.

The original ***Malmstrom ventouse*** used a metal cup applied on the fetal scalp for traction, and a glass jar and pump to create a negative pressure. Current instruments use pliable plastic and polyethylene cups, and electric suction instruments for negative pressure production.

INDICATIONS FOR USE OF VACUUM EXTRACTOR

1. Rotation and extraction of non-rotated occipito anterior position.
2. Rotation and extraction of non-rotated occipito posterior position.

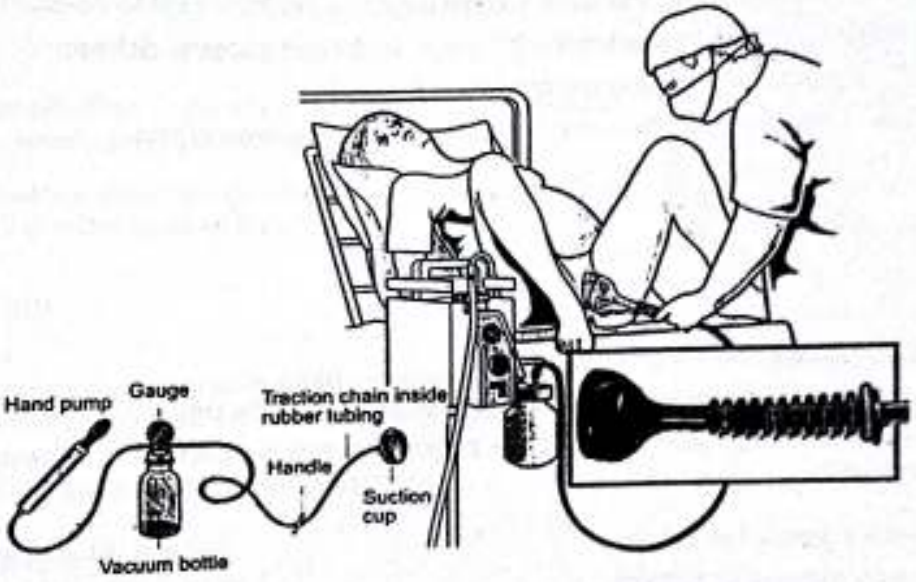


Fig 46:10 Delivery of the head by the use of vacuum extractor

PREREQUISITES FOR THE USE OF VACUUM EXTRACTOR

1. Suitable presentation: In contrast to forceps, vacuum is not suitable for face or after coming head in breech presentation.
2. No cephalo-pelvic disproportion.
3. Rupture of membranes.
4. Adequate uterine contractions.
5. Head well applied on the cervix.
6. Empty bladder and rectum.
7. Adequate anesthesia.
8. Living fetus: Vacuum is not suitable for dead fetus.
9. No fetal distress: Vacuum is time consuming.

N.B: Vacuum extractor can be used during the 1st stage provided that the cervix is > 7 cm dilatation. Also head engagement is not a prerequisite provided no cephalo-pelvic disproportion.

CONTRAINDICATIONS TO THE USE OF THE VACUUM EXTRACTOR

1. Cephalopelvic disproportion or contracted outlet.
2. Non vertex presentations, as in face and breech presentations.
3. Premature infants, to avoid serious complications (see later).
4. Marked fetal distress, as it needs a longer period of application than the forceps.

Advantages of the Vacuum extractor over forceps:

- Allows easy and gentle traction on the fetal head, due to limited force.
- Promotes flexion and helps internal rotation of the fetal head in OP positions.
- Less encroachment on maternal pelvic space, resulting in less trauma to maternal birth canal, and less serious vaginal, cervical and perineal lacerations.

Advantages of the forceps over Vacuum extractor:

- Faster extraction so suitable to shorten the 2nd stage, foetal and maternal distress.
- Can be used in face 9MAP) and aftercoming head of breech.
- Used in preterm fetus to protect the head from compression decompression.
- Can be used for dead fetus.

Complications of the vacuum extractor:

1) Fetal birth injuries including:

- Cephalhaematoma (bleeding into the scalp).
- Scalp lacerations, (excessive force and repeated slipping of the cup).
- Cerebral haemorrhage due to excessive negative pressure specially in preterm and asphyxiated fetus.

2) Maternal birth injuries including:

- Vaginal and perineal lacerations.
- Cervical lacerations (inclusion of cervical tissue within the cup).
- Rarely rupture of the uterus.

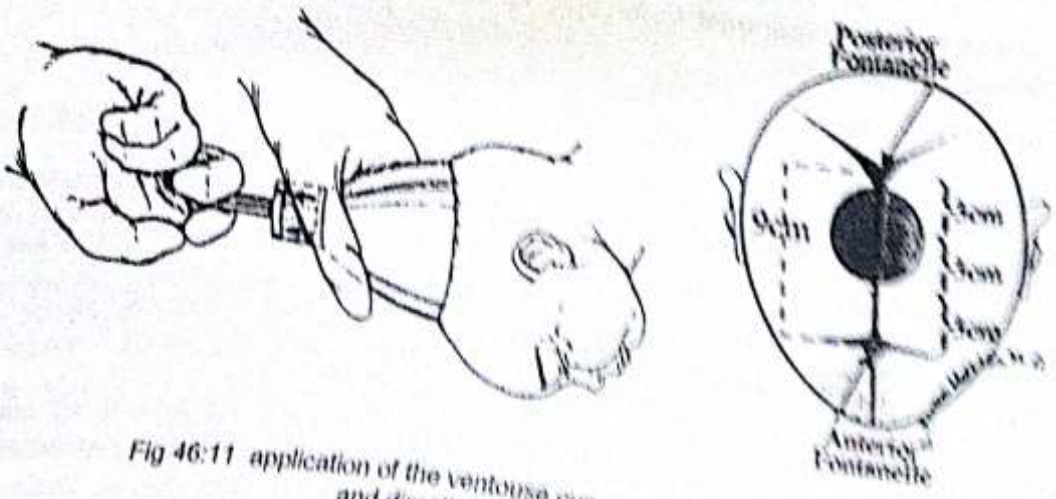


Fig 46:11 application of the ventouse cup on the foetal head and direction of traction

47 EPISIOTOMY

- *Definition*
- *Types*
- *Indications*
- *Timing of the incision*
- *Technique*
- *Episiotomy repair*
- *Advantages*
- *Complications*

DEFINITION:

It is an incision of the perineum during labor.

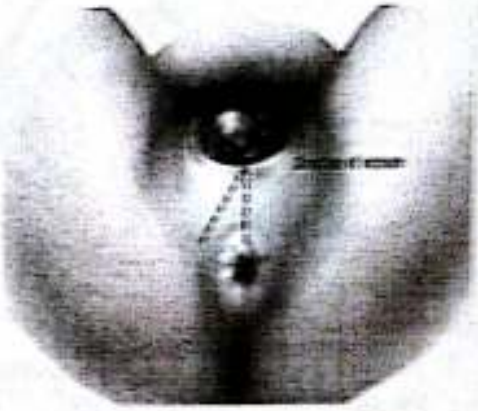


Fig 47.1 Episiotomy incisions

TYPES:

- *Median (Midline) Episiotomy:* Midline incision of the perineum directly backwards towards the anus.
- *Mediolateral Episiotomy:* Directed posterolateral 45° towards the buttocks away from the anus.

INDICATIONS:

A) Maternal:
- Short rigid perineum as in elderly primigravida.
- Perineal scarring such as in previous perineal or pelvic floor repair.
- Mild degree of pelvic outlet contractions.

B) Foetal:
- Face to pubis delivery.
- Vaginal breech delivery.
- Shoulder dystocia.
- Oversized foetus.
- Forceps or ventouse delivery.
- Preterm delivery.

Timing of the Incision:

- Best time to perform episiotomy is when the head is visible during a contraction to a diameter of 3 to 4 cm (just before crowning).
- Before traction by forceps or vacuum extractor.
- Too early episiotomy causes bleeding from the gaping to be considerable.
- Too late episiotomy is useless.

TECHNIQUES OF EPISIOTOMY :

- *Median type:* A vertical incision is made in the perineal body avoiding the fetal presenting part. The incision starts at the forchette till approximately half the length of the perineal body. The incision should extend into the vagina approximately 2 to 3 cm.

- ***Mediolateral type:*** Incision starts at the forchette at a 45 degree angle to the midline of the perineum. The incision should extend into the vagina approximately 2 to 3 cm. When the incision reaches the ischiorectal fossa (with its fat) it is termed ***generous episiotomy.***

Median Episiotomy	Mediolateral Episiotomy
Extension to the anal sphincter and rectum is more common	*Extension to the anal sphincter and rectum is less common*
Easier to repair	*More difficult to repair*
Rare faulty healing	*Faulty healing is more common*
Less pain in the perineum	*More pain in the perineum*
Dyspareunia is rare	*Dyspareunia is more common*
Less blood loss	*More blood loss*

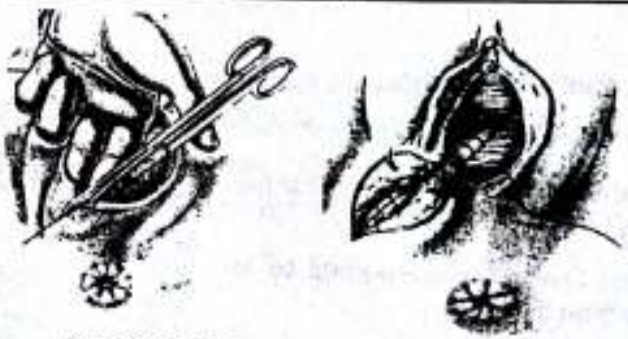


Fig 47:2 Episiotomy incision and repair The

Episiotomy Repair:

1. Vaginal mucosa and submucosa are closed by chromic catgut 3/0 starting 1 cm beyond the visible apex till the hymeneal ring.
2. Interrupted chromic catgut sutures are used to approximate the muscles and fascia of deep and superficial perineal pouches.
3. Closure of the superficial fascia by continuous suture.
4. Closure of the skin by interrupted simple or mattress sutures or alternatively by subcuticular continuous stitches.

Advantages of episiotomy:

1. Clean cut incision which is easy to repair compared to irregular vaginal lacerations.
2. Shorter second stage of labour, thus less fetal and maternal distress.
3. Reduce intracranial haemorrhage in PTL by decreasing compression-decompression effect.
4. Reduce damage to maternal pelvic floor predisposing to vaginal prolapse, stress incontinence and ano-rectal dysfunction.

Complications of episiotomy:

1. Increased blood loss.
2. Extension to anal sphincter (median type) or ischio-rectal fossa (mediolateral type).
3. Haematoma formation.
4. Infection.
5. Perineal pain, dyspareunia and ugly scar.

Contraindications of episiotomy:

1. Adequate perineal size. Episiotomy is not routine even in primigravidae.
2. High suspicion of Cesarean delivery.
3. Extensive perineal lesions such as condyloma accuminata or severe edema.

48 CAESAREAN SECTION

- *Definition and types of C.S.*
- *Indications*
- *Technique of LSCS*
- *Abdominal incision*
- *Uterine incision*
- *Delivery of the foetus*
- *Closure of the uterus and abdominal wall*
- *Advantages of LSCS over USCS*
- *The vertical LSCS incision*
- *Complications of C.S.*
- *Management of a pregnancy with previous C.S.*
- *Vaginal birth after C.S. (VBAC)*

DEFINITIONS

- ***Caesarean section (C.S.):*** is the operation done for delivery of a viable fetus, through an abdominal and uterine incisions.
- ***Hysterotomy:*** is the operation done for delivery of the foetus before viability < 24 weeks, through an abdominal and uterine incision
- ***Primary C.S.:*** the first C.S.
- ***Repeat C.S.:*** C.S. in patients with one or more previous C.S.
- ***Elective C.S.:*** C.S. in a patient not in labour

Fig 48:1 LSCS

TYPES OF C.S. ACCORDING TO OPERATIVE METHOD

1. ***Lower Segment (LSCS):*** using a transverse incision in the LUS (Munro Kerr's technique).
2. ***Upper segment (USCS):*** using a longitudinal incision in the upper uterine segment (UUS).

INDICATIONS of C.S.:

I. Previous CS is nowadays the most common indication.

II. Faults in the Passages (maternal indications):

1. Contracted pelvis: Moderate and marked degrees (commonest cause in primigravidae).
2. Cephalo-pelvic disproportion (CPD) usually detected after failed trial labour (TOL)
3. Cervical dystocia: leading to failed progress of labour and failed TOL.
4. Placenta Praevia (PL PRV): total or partial central PL PRV, or marginal posterior PL PRV.
5. Severe vaginal stenosis, scarring, or masses obstructing delivery.
6. Perineal and vulval lesions as HPV infection forming large condylomata.
7. Pelvic tumors such as low corporeal fibroid or ovarian tumors.

III. Faults in the Passenger (foetal indications):

1. Macrosomia: oversized foetus (the most common cause in multipara).
2. Malpresentations as in:
   - Cephalic OP positions with failed long rotation (DTA, or persistent oblique OP).
   - Face DMP or with failed long rotation (DTA of face, or persistent oblique MP)
   - Persistent Brow presentation
   - Breech presentation when trial vaginal delivery is contraindicated.
   - Transverse lie, when ECV fails or contraindicated
3. Foetal distress during 1st stag of labour: due to;
   1. Placental site bleeding: Placenta praevia or accidental hemorrhage.
   2. IUGR and post-maturity.
   3. Prolapsed pulsating cord with a non fully dilated cervix.
   4. Vasa-praevia: to avoid severe fetal hemorrhage resulting in death.
   5. Hypertonic uterine action, not responding to analgesics
   6. Rh iso-immunization with severe allo-immune reaction.

IV. Maternal medical diseases (act my multiple mechanisms):

1. Hypertensive disorders; such as severe PE and Eclampsia: if termination of pregnancy is indicated while vaginal examination reveals a poor Bishop score.
2. Diabetes mellitus: Because of macrosomia, placental insufficiency, and risks of IUFD.

V. Recurrent unexplained IUFD; an Elective C.S. is usually performed once 37 weeks reached

VI. After successful IVF procedure; This is a **relative indication** as many women my prefer C.S. after a long period of infertility and successful ICSI procedure to avoid hazards and stress associated with vaginal delivery.

TECHNIQUE OF LSCS

- Abdominal wall skin incision
  - Transverse suprapubic incision (Pfannensteil incision): most commonly performed.
  - Longitudinal subumbilical suprapubic incision; suitable in selected cases as previous laparotomy, marked obesity, etc..
- **Opening abdominal wall in layers;** subcutaneous fat, rectus sheath, separation of rectus abdominis muscles, opening the parietal peritoneum of abdominal wall, then opening the loose visceral peritoneum overlying the LUS.
- **Put a Doyen's retractor** in the lower abdominal incision to retract the urinary bladder and protect it.
- **Transverse incision of the LUS;** about 10 cm width (Kerr incision), and rupture the amniotic membrane, if still intact.

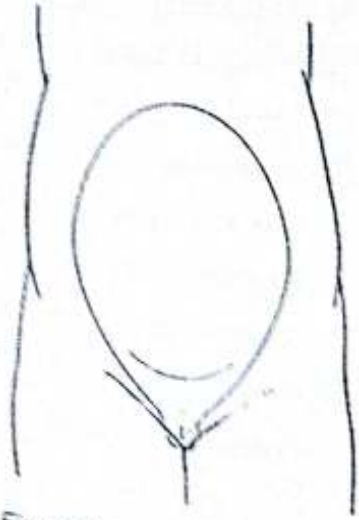


Fig 48:2 Transverse incision in LSCS

- **Foetal Extraction:**
  - A) Cephalic presentations: Lifting the head up by the fingers and palm of one hand (or by one blade of a short forceps) will allow the head to glide outside the uterine incision aided by gentle fundal pressure done by the assistant.
  - B) Breech presentation: do breech extraction (BE)
  - C) Transverse Lie: do Internal podalic version (IPV) and BE
- **After delivery of the foetus;** swab the face and aspirate to clear up respiratory passages.
- **Clamp and divide the cord.**
- **Delivery of the placenta and membranes.**
- **Exploration of the uterine cavity** for remnants of the placenta and membranes and for congenital uterine anomalies.
- **Uterine repair:** suturing the uterine incision in layers:
  1. 1st muscle layer using continuous (No 0-1 vicryl suture material) not including the decidua). Haemostasis and good muscle layer coaptation are a must.
  2. A second continuous or interrupted inverted suture: to reinforce the first layer.
  3. The visceral peritoneum is closed by 2/0 vicryl sutures, but can be left without closure.
- Abdominal toilet and remove the packs.
- Parietal peritoneum is closed by continuous 2/0 vicryl , but can be left without closure.
- Abdominal wall is closed in layers; rectus sheath, muscle approximation, subcutaneous fat in obese patients, and finally skin sutured usually with interrupted or sub-cuticular sutures. .

Fig 48:3 delivery of the head in LSCS

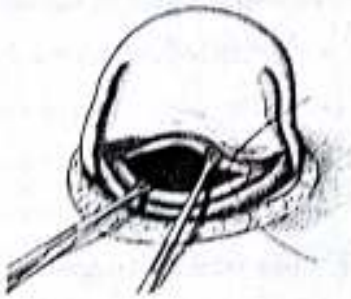


Fig 48:4 1st layer closure of uterine incision

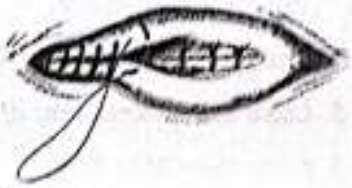


Fig 48:5 2nd layer closure of uterine incision

The Vertical LUS Incision (Kronig's Incision)

Rarely a vertical incision in the LUS may be performed to facilitate delivery of the foetus as the incision can be extended upwards safely avoiding lateral injury of the uterine vessels. Indications include:

1. Constriction ring: to cut the ring.
2. Varicose veins in the broad ligaments (to avoid their injury resulting in bleeding).
3. Head deeply engaged in the pelvis, to facilitate it's extraction and to avoid lateral extension in the transverse incisions.
4. Hydrocephalus or foetal tumours.
5. Some cases of PTL with non properly formed LUS

Fig 48:6 USCS

The Upper segment uterine incision (USCS)

- Abdominal incision: longitudinal subumbilical suprapubic incision (Median incision).
- Uterine incision: Anterior central vertical incision in the upper segment.
- Uterine closure: 2 or 3 layered closure including the adherent visceral peritoneum.
- Indications of USCS: when LUS is not easily accessible as in;
  - Large fibroids, excessive varices, excess adhesions, PL PRV, Impacted shoulder.
  - In PTL where the lower segment is not well formed.
  - Previous successful repair of high VVF (for fear of recurrence), or rarely cancer cervix..

CAESAREAN HYSTERECTOMY:

- Perform CS then remove the uterus.
- Indications:
  - Uterine atony associated with uncontrollable severe postpartum hemorrhage.
  - Couvelaire uterus (severe concealed accidental haemorrhage).
  - Tumours associated with pregnancy as operable cancer cervix.
  - Placenta accreta, Increta and Percreta.

N.B.

- Tubal sterilization can be done during CS when indicated.
- CS can be done using general, epidural or spinal anesthesia.

ADVANTAGES OF LSCS OVER USCS

1. ***Stronger scar: (it ruptures in 0.2%) due to:***
   - Better healing as the lower segment is less active in puerperium.
   - Better coaptation of the edges (lower segment is thin).
   - Less haematoma in the suture line (less vascularity).
   - In subsequent pregnancies, erosion of the incision site by the chorionic villi is rare.
2. ***Less Haemorrhage: because***
   - The placental site is away from the operation area.
   - Lower segment is thin and less vascular.
3. ***Less abdominal distension and ileus:*** because the intestines are away and not manipulated during the operation.
4. ***Less infection due to:*** better peritonization, and better healing.
5. ***Less adhesions and intestinal obstruction.*** wound is low and is covered by peritoneum.
6. ***Less mortality rate.***

COMPLICATIONS OF CS:

I. Intra-operative:

1. Anesthetic complications such as; cyanosis, cardiopulmonary complications.
2. Primary haemorrhage: due to injury of inferior epigastric vessels, uterine vessels, uterine atony or DIC in concealed accidental haemorrhage.
3. Injury to the bladder, colon or ureters.

I. Early post-operative (within 24h):

1. Reactionary hemorrhage: due to slipping of a ligature.
2. Bladder or ureteric injuries.

II. Late post-operative:

1. Wound complications: infection, burst abdomen or incisional hernia
2. Infections: generalized peritonitis, parametritis.

3. GIT: Late diagnosis of intestinal injuries, paralytic ileus, acute gastric dilatation, and intestinal obstruction.
4. Thrombo-embolic complications.
5. Rupture scar in subsequent pregnancy.
6. Abdominal adhesions leading to infertility or intestinal obstruction.

RUPTURE OF CS SCAR IN SUBSEQUENT PREGNANCY:

- **Incidence:** in USCS: 2-4%; while in LSCS, it is 0.2%.
- **Causes: Weak CS Scar, is due to:**
  - Operative faults:
    - Incomplete haemostasis resulting in wound hematomas heal by weak fibrosis.
    - Inaccurate coaptation of the wound edges.
    - Inversion of the decidua in the wound.
  - Post operative infection.
  - Placental insertion over the scar (erosion of the scar by chorionic villi).

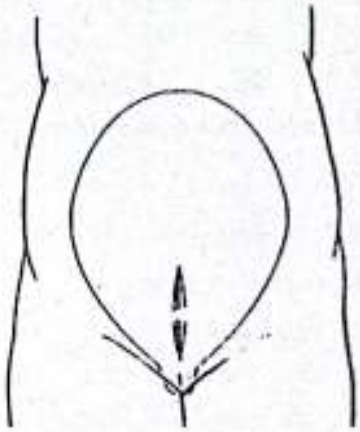


Fig 48:7 rupture USCS scar

  - In USCS scar (the upper segment is not at rest i.e. contractions and retractions, delaying healing).
  - Repeated pregnancies.
  - Over-distension of the uterus by polyhydramnios or twins.
- **Management of pregnancy with previous C.S.:**
  - Trial of vaginal delivery.
  - Repeat CS.

PREREQUISITES FOR TRIAL VAGINAL BIRTH AFTER CS (VBAC):

- Non-persistent cause of previous CS as severe degrees of contracted pelvis and CPD .
- No more than one previous LSCS.
- Previous normal puerperium.
- No tenderness over the CS scar.
- Vertex presentation, with head engaged.
- No associated other obstetric or medical complications.

N.B.: a previous normal vaginal delivery followed by C.S. improves the chances of a safe and successful VBAC

BLOOD TRANSFUSION IN OBSTETRICS

- **Indications:**
  1. Hemorrhage: during pregnancy, CS, or postpartum.
  2. Severe anemia (given very slowly to avoid overloading the circulation or better give packed RBCs).
  3. Puerperal sepsis and septic abortion: small doses of fresh blood (to increase immunity).
  4. Babies with severe Rh iso-immunization:
     a. Exchange transfusion after birth.
     b. Intrauterine transfusion in selected cases.
  5. Hypofibrinogenaemia (DIC).

- **Precautions:**
  1. Cross matching.
  2. Rate of transfusion = rate of blood loss.
  3. Blood should not be very cold.
  4. For every one liter of blood, give 10 cc calcium gluconate (to antidote blood citrate).
  5. Continuous observation.
  6. Monitoring CVP during transfusion in risky cases (anaemia, cardiac, PE cases, ...etc).

- **Complications:**
  1. Major anaphylactic reactions due to incompatibility leading to dyspnoea, cyanosis, rigors, lumbar pain, anuria and jaundice.
  2. Febrile reactions due to presence of pyrogens as blood or apparatus. Stop transfusion and give antipyretics and antihistaminics.
  3. Air embolism.
  4. Circulatory overloading especially in cases of anemia.
  5. Transmission of diseases: AIDS and infective hepatitis.