

KASR El-AINI

Essentials of OBSTETRICS

Department of
Obstetrics and Gynaecology
Cairo University

Kasr El Aini

ESSENTIALS OF OBSTETRICS

Fifth Edition 2020

Professor ABDELMEGUID RAMZY

*Professor of obstetrics and Gynaecology - **Cairo University***

Head of editorial board 2019-2020

Chief Editor

Professor KHALED RASHEED

*Professor of Obstetrics and Gynaecology **Cairo University***

Advisory and IT Committee

*Professor **Hossam Badrawy***
*Professor **Ihab Soliman***
*Professor **Ahmed Elminawy***
*A. Professor **Moaataz Elsherbiny***

Honorary Committee

*Professor **Nabil Abdelhakim***
*Professor **Mohammed Abdelhady***
*Professor **Mohammed Elmeleigy***
*Professor **Raafat Reyad***

Editorial Board

*Professor **Reda Ismail***
*Professor **Gamal Abdelsamei***
*Professor **Tarek Fawzy***
*Professor **Magdy Ibrahim***
*Professor **Alaa Elabrashy***
*Professor **Ahmed Soliman***
*Professor **Mamdouh Sheeba***
*Professor **Mahmoud Salem***
*Professor **Nadin Alaa***
*Professor **Sherif Negm***
*Professor **Mostafa Abdelbar***
*A. Professor **Reham fouad***

Department of Obstetrics and Gynaecology
Cairo University
2020

Preface to the fifth Edition

Hello dear students.

This is the 5th edition. Our book is now 15 years old, but is even growing younger, as we are keen to update the information within in accordance with the latest studies in our field. You will find in the current edition, in an appealing form, full of illustrations whether in the book itself or on the adjoining CD.

This edition, in its final version, met my vision into upgrading the book into the digital mobile platform. So that you, can study anywhere and any place you go, make it on the road, in the coffee shop, or club.

In addition I asked our active editorial board to add quick response codes (QR) alongside each chapter to link to external sources for optional further reading and explanatory videos. This will help broaden your vision on the topic for better understanding the facts and make it easier for you to accumulate the knowledge and retain the information in accordance with scientific fact stating that the brain assimilates knowledge more readily in a picture form.

Thanks to the enthusiasm and perseverance of the editorial board, headed by Prof. **Khaled Rasheed**, and the valuable contribution of the faculty members of the department in Cairo University, this edition in its final version met my expectations, and I hope it will meet yours as well.

Our book is a valuable addition to our department's efforts to upgrade our education standards to meet and hopefully surpass the most recent education advances worldwide.

This book will be a valuable learning tool for both our undergraduate students as a requirement, as well as for our postgraduate students as a baseline for further reading sources.

I hope you make good use of this rich source of medical information both in your undergrad education and in your practice later on in your career

Abdel Maguid Ramzy

Head of department of Obstetrics and Gynaecology 2018-2019

Faculty of medicine, Cairo University

CONTENTS

	Page
BASIC OBSTETRICS	
1. Obstetric History, Examination And Pregnancy Diagnosis	1
2. Fertilization, Implantation, And Early Embryonic Development	6
3. The Placenta, Cord, Membranes, And Foetal Circulation	10
4. Maternal Physiologic Changes During Pregnancy	17
5. Antenatal Care And High Risk Pregnancy	21
6. Prenatal Diagnosis Of Foetal Chromosomal And Congenital Abnormalities	29
EARLY OBSTETRIC COMPLICATIONS	
7. Spontaneous Abortion	35
8. Ectopic Gestation	45
9. Molar Pregnancy	53
NORMAL AND ABNORMAL LABOUR AND DELIVERY	
10. Anatomy Of Female Pelvis And Foetal Skull	61
11. Physiology and Stages of Labour	72
12. Management of Normal Labour and Delivery	76
13. Mechanisms of Delivery in Cephalic Presentation	81
14. Mechanisms of Delivery in Foetal Malpresentations	90
15. Multiple Gestation	99
16. Foetal Surveillance during Pregnancy and Labour	104
17. Abnormal Labour	109
18. Cephalo-pelvic Disproportion	116
19. Obstetric Trauma during Labour and Delivery	119
20. Complications of the 3rd Stage of Labour	124
LATE OBSTETRIC COMPLICATIONS	
21. Antepartum haemorrhage And intrapartum haemorrhage	139
22. Term and Preterm Premature Rupture of the Membranes	149
23. Preterm Labour and Post term Pregnancy	151
24. Amniotic Fluid Disorders	155
25. Foetal Growth Disorders	159

MEDICAL AND SURGICAL COMPLICATIONS

26. Preeclampsia and Hypertensive Disorders 164

27. RH Iso-immunization 173

28. Medical and Surgical complications 176

- GIT and Hepatic Disorders 178
- UTI and Renal Disorders 180
- Thyroid Disorders 181
- Diabetes Mellitus 182
- Seizure Disorders 185
- Anaemia and Haematologic Disorders 186
- Thromboembolic disease 188
- CVS Disorders 190

THE NEW BORN AND THE PUERPERIUM

29. Foetal and Neonatal asphyxia 193

30. Foetal Birth Injuries 198

31. The Puerperium Normal and Abnormal 202

OBSTETRIC PROCEDURES AND OPERATIONS

- Induction of Abortion 210
- Induction of Labour 213
- Analgesia and Anaesthesia during Labour 215
- Episiotomy 218
- Operative Vaginal Delivery (forceps and Vacuum Extractor) 220
- Caesarean Section and Prior CS Delivery 225

01 OBSTETRIC HISTORY, EXAMINATION, AND EARLY PREGNANCY DIAGNOSIS

- *Obstetric History*
- *Obstetric Examination*
- *Diagnostic Aids*
- *Diagnosis of Pregnancy*

OBSTETRIC HISTORY

1. PERSONAL HISTORY:

Including; Patient's name, age, address, occupation, marital status, gravidity and parity.

2. COMPLAINT:

- The patient may have ***no complaint*** and is coming for ***routine antenatal care*** (ANC).
- The patient may complain of ***symptoms related to her present pregnancy*** (in the patient own words), as nausea, vomiting, vaginal bleeding, pelvic pain, etc....).
- The patient may complain from ***symptoms related to a previously known medical disorder*** dating before the present pregnancy (as DM, chronic hypertension, cardiac disease, bronchial asthma, autoimmune disease, etc.....)

3. PRESENT HISTORY:

- ***Calculate the Expected Delivery Date (EDD):***
  - Naegele's rule (LMP + 7 days + 9 months).
- ***Calculate the duration of pregnancy*** using the following steps:
  - Subtract the today's date from the date of LMP (months and days).
  - Convert the result into weeks considering that every 3 months = 13 weeks.
- ***Analysis of the present complaint;***
  - Onset, Course, and duration of each complaint starting with the most prominent one
  - Symptoms relevant to the present complaint or disease
  - Ask about associated symptoms (see gynaecological history and diagnosis).
- Ask about ***important warning symptoms*** during pregnancy;
  - **In 1st trimester:** vaginal bleeding, and abdominal pain.
  - **In 2nd trimester:** abdominal enlargement, quickening, vaginal bleeding, vaginal discharge, abdominal pain, LL swelling, and dysuria.
  - **In 3rd trimester:** Foetal movements, vaginal bleeding, vaginal gush of fluid, headache, blurring of vision, epigastric pain, nausea and vomiting, LL swelling
- Ask about ***special investigations done in pregnancy*** specially Hb, Rh, blood glucose, urine analysis and ultrasound scan.
- Ask about any ***medications taken in pregnancy***.
- Ask about ***exposure to drugs and other teratogens*** in early pregnancy.

4. PAST, MENSTRUAL, OBSTETRIC and FAMILY HISTORY:

See gynaecological history and diagnosis.

OBSTETRIC EXAMINATION

1. GENERAL EXAMINATION: see gynaecological history and diagnosis (*Essentials of Gynaecology*)

- *Pulse, temp, BP*, and RR (respiratory rate).
- *Weight & height:* to define body mass index (BMI) and class of obesity if present.
- *Gait and stature:* Normal gait or limp, and presence of extreme kyphosis or scoliosis.

2. ABDOMINAL EXAMINATION: as gynaecology plus:

A. FUNDAL LEVEL:

- ***Manual method;*** using the ulnar border of the left hand, the fundal level correlated with duration of pregnancy in relation to symphysis pubis (SP), umbilicus, and xiphisternum (XS):
  - At 12 weeks; fundal level is at the upper border of the symphysis pubis
  - At 20-22 weeks: fundal level is at the umbilicus
  - At 36 weeks: fundal level is at the XS.

  N.B.: after engagement, the fundal level descends below the XS.
- ***Metric method:*** the distance between symphysis pubis and fundal level, in cm, correlates with duration of gestation.

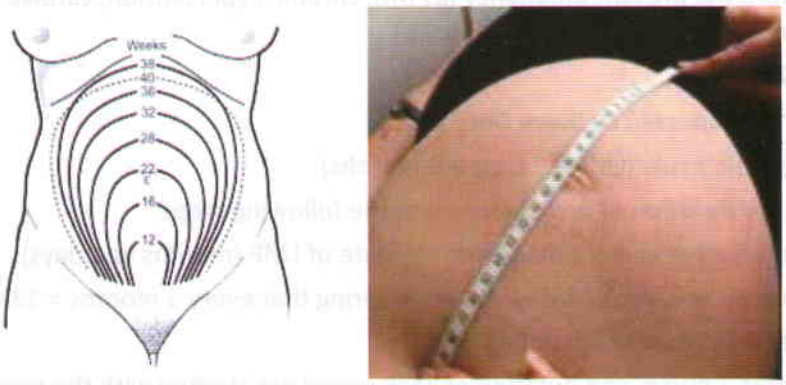
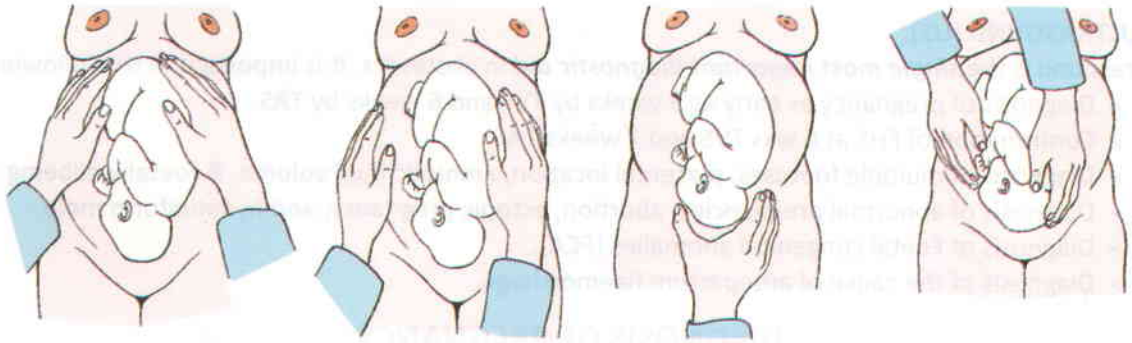


Fig 1-1: Fundal level manual and metric assessment

B. LEOPOLD MANOEUVRES: (see figure)

- ***Fundal grip:*** to determine the **foetal pole** occupying the uterine **fundus**. Normally the fundus contains the foetal breech near term.
- ***Umbilical grip:*** to determine the direction of the **foetal back** and spine;
  - *Right or left:* in relation to maternal midline
  - *Anterior or posterior:* in relation to the back and spine of the mother
  - *Presence of a foetal head* at either side denotes transverse lie
- ***1st pelvic grip:*** gripping the lower uterine segment (LUS) above the SP helps to define;
  - *Cephalic presentations:* head is felt small, globular, hard, ballottable and tender.
  - *Breech presentation:* breech is felt large, soft, not ballottable, and not tender.
  - *Shoulder presentation:* the LUS feels empty.
- ***2nd pelvic grip:*** Done after 1st pelvic grip to confirm head engagement and flexion.



Fundal grip | umbilical grip | 1st pelvic grip | 2nd pelvic grip

Fig 1-2: Leopold maneuvers for abdominal obstetric examination

3. AUSCULTATION OF FOETAL HEART SOUNDS

- In the 2nd trimester FHS can be audible by sonicaid which will also detect FHR
- This is the simplest accurate method to ensure foetal life throughout pregnancy
- Point of maximum intensity will depend upon gestational age, foetal lie, and presentation
- FHS are most commonly heard below the umbilicus near to the middle line or on either side



Fig 1-3: auscultation of FHS by sonicaid

4. LOCAL EXAMINATION:

- Local examination is ***not routinely*** performed during pregnancy except when needed
- It may be performed to **ensure** diagnosis in certain obstetric conditions as; vaginal bleeding during early or late pregnancy, PROM, diagnosis of onset of labour, evaluation of cephalopelvic disproportion and contracted pelvis, and associated conditions as uterine leiomyomas.
- ***During 2nd trimester:*** to obtain vaginal swabs for culture in certain cases
- ***During 3rd trimester:*** at 36-38 weeks: to confirm foetal presentation, determine head engagement, pelvic capacity, and cervical status (ripening, effacement, and dilatation).
- ***During labour:*** to confirm onset of labour, cervical status, and station of foetal head

COMMON INVESTIGATIONS AND DIAGNOSTIC AIDS

A. LABORATORY TESTS:

- Blood group, RH factor, CBC, Fasting and post prandial BS, complete urine analysis
- Kidney FT, Liver FT, bleeding and coagulation profiles, PT & PC
- Vaginal cultures for Group B streptococcal infection (GBS)

B. SERUM HCG ASSAYS:

- Serum B-hCG are positive in levels > 50 m IU/ml at the 1st day of a missed period
- Levels increase rapidly with trophoblastic proliferation nearly doubling every 55 hours
- At levels > 1500 m IU/ml an intrauterine G.sac can be seen by vaginal US (TVS)
- At levels > 6500 m IU/ml an intrauterine G. sac can be seen by abdominal US (TAS)
- Abnormal serum B-hCG and abnormal doubling time can be very beneficial in differentiating a normal pregnancy from abortion, ectopic, or hydatidiform moles

C. ULTRASOUND (US):

Ultrasound is the ***single most important diagnostic aid*** in obstetrics. It is important in the following:

- Diagnosis of pregnancy as early as 5 weeks by TVS and 6 weeks by TAS
- Confirmation of FHS at 6 wks TVS and 7 weeks TAS
- Diagnosis of multiple foetuses, placental location, amniotic fluid volume, & foetal wellbeing
- Diagnosis of abnormal pregnancies; abortion, ectopic pregnancy, and hydatidiform mole.
- Diagnosis of Foetal congenital anomalies (FCA).
- Diagnosis of the cause of antepartum haemorrhage.

DIAGNOSIS OF PREGNANCY

Diagnosis of pregnancy is based on the **triad** of:

- Delayed Cycle (missed period)
- Positive Pregnancy test (urine or serum).
- Pelvic Ultrasound (TAS/TVS) showing intrauterine pregnancy.

A. DELAYED MENSTRUAL CYCLE (missed period)

A missed period is the *earliest symptom* of pregnancy.

- *Cause:* persistent corpus luteum **(CL)** function
- *Mechanism:* **hCG** produced by the proliferating trophoblasts stimulates **CL** for continuous production of both oestrogen **(E)** and progesterone **(P),** thus preventing hormone withdrawal and endometrial shedding that normally occur in absence of a pregnancy.



Fig 1:4 positive urine pregnancy test ELISA

- *Associated symptoms in 1st trimester:* as nausea, with or without vomiting, frequency of micturition, fatigue, weakness and lack of concentration, breast enlargement.
- *Associated symptoms in 2nd trimester:* gradual abdominal enlargement and perception of foetal movements (quickening).

B. POSITIVE PREGNANCY TESTS

Laboratory tests for diagnosis of pregnancy are based on the detection of **hCG** in urine or blood samples of pregnant women.

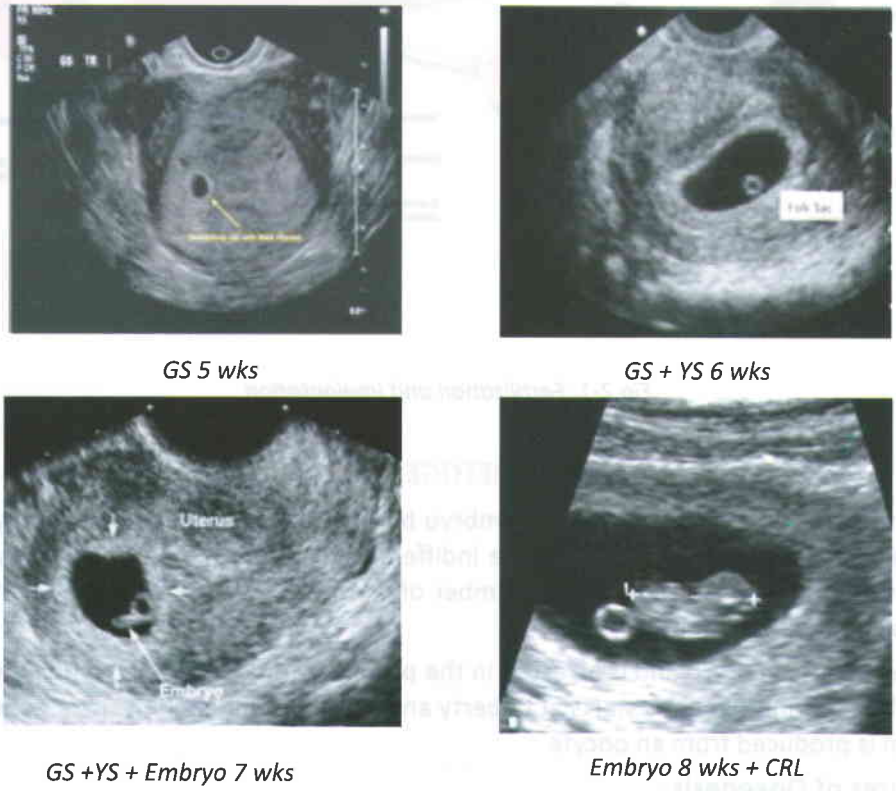
- **Urine ELISA tests:** Enzyme Linked Immunosorbent Assay slide tests are simple, cheap, accurate, and readily available. A positive result will be detectable in the first 48 hours of a missed period.
- **Serum β-hCG tests**
  - It is the *earliest* available method for accurate diagnosis of pregnancy.
  - The test is positive within *1st 48 hours* of a missed period (levels > 50 mIU/ml).
  - β-hCG levels *rise sharply* in the first 8 weeks of pregnancy, nearly doubling every 48-58
  - Levels usually > 1500 mIU/ml. by end of the 5th week, and > 10,000 mIU/ml by end of the 6th week (calculated from the LMP).
  - 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 since it is a quantitative test of high sensitivity and extremely low false positive values.

C. ULTRASOUND DIAGNOSIS OF PREGNANCY

TAS and TVS are the mainstay in the diagnosis of an intrauterine pregnancy. US is safe, cheap, readily available, and easy to interpret.

- **First trimester US:**
  - 5-6 weeks : Gestational sac < 2 cm with no embryonic echoes
  - 7-9 weeks : Embryo with positive heart pulsations.
  - 10-13 weeks: Foetus with early trunk movements
  - Measurements of foetal crown to rump length **(CRL)** give the most accurate dating of pregnancy, and hence the most acceptable expected delivery date (EDD).
- **Second trimester US:**
  - Confirmation of foetal life (FHR)
  - Foetal biometry for estimation of gestational age, foetal weight, and growth patterns.
    - ⇒ Biparietal diameter (BPD)
    - ⇒ Abdominal circumference (AC)
    - ⇒ Femur length (FL)



GS 5 wks | *GS + YS 6 wks*

GS +YS + Embryo 7 wks | *Embryo 8 wks + CRL*

Fig 1-5: TVS in diagnosis of early pregnancy

N.B.: 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**.

02 FERTILIZATION, IMPLANTATION, AND EMBRYONIC DEVELOPMENT

- *Gametogenesis*
- *Fertilization*
- *Implantation*
- *Embryonic Development*

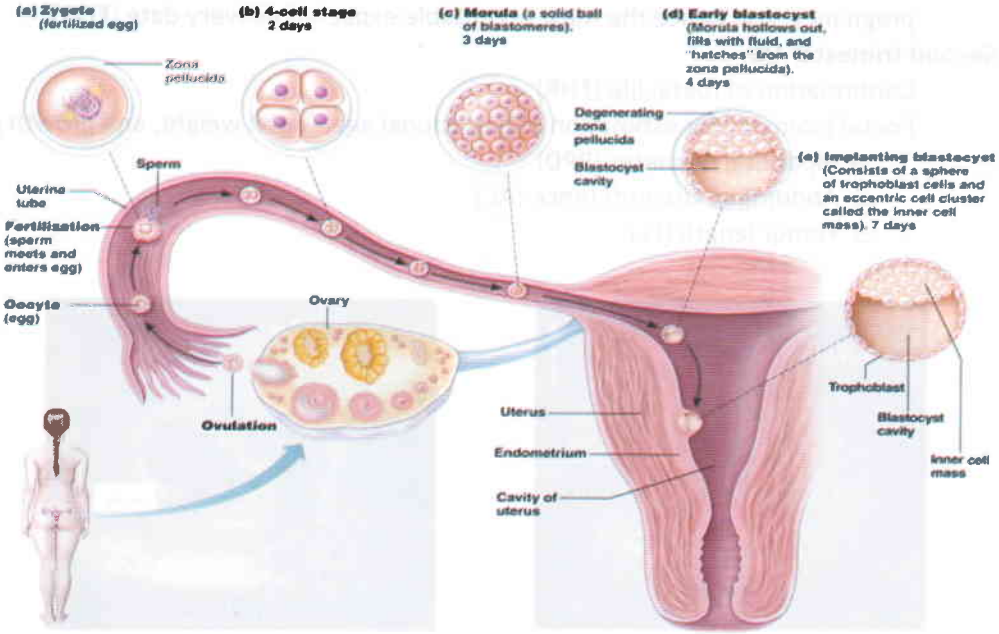


Fig 2-1: Fertilization and Implantation

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 meiotic *division* is a reduction division with long prophase
- Second meiotic *division* with no prophase.

Pre-Fertilization Changes:

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 of 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.

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.

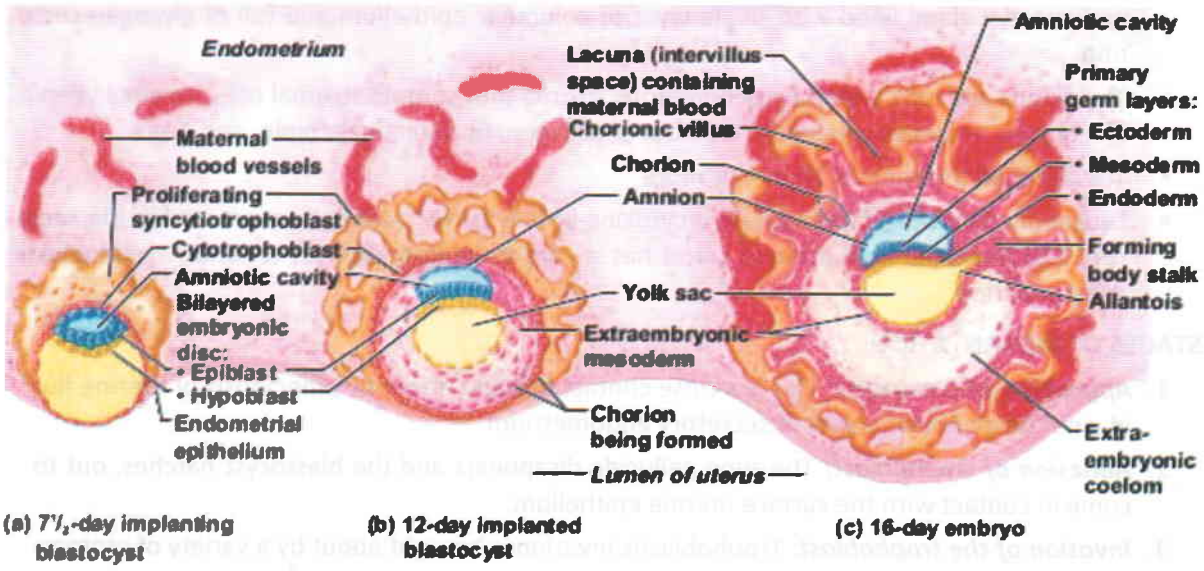


Fig 2-2: Implantation and Development of fertilized ova

- ***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.
- ***Trophoblastic Differentiation:*** By the eighth day after fertilization the blastocyst is partiality implanted into the decidua and the trophoblast has differentiated into *cytotrophoblast* and *syncytiotrophoblast*. 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.
- ***The secondary villi:*** when extra embryonic mesoderm fill the core of the trophoblastic 1ry villi, it is termed 2ry villi (day 12)
- ***The tertiary villi:*** when blood vessels enters the 2ry villi, they are transformed into tertiary villi (day 17)

IMPLANTATION

Implantation or nidation is imbedding of fertilized ovum in the uterine mucosa.

IMPLANTATION BED (The decidua)

- The decidua is a developed stage of secretory endometrium that was formed under oestrogen (E) and progesterone (P) control during the 2nd half of the menstrual cycle. It consists of endometrial gland lined with single layer of columnar epithelium and full of glycogen secretion
- The glands are surrounded by oedematous stroma that contain stromal cells, stromal vessels, leucocytes, and macrophages in a matrix of collagen, fibronectin, laminin, and Gags.
- The decidual thickness is 10 mm or more
- Leukaemia inhibiting factor (LIF) is a cytokine of IL-6 family maximally expressed in the secretory endometrium and decidua , and has a role in implantation as it forces trophoblastic differentiation.

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.
4. ***After implantation, the decidua is divided into:*** decidua basalis; deep to the blastocyst, decidua capsularis; covering the blastocyst, and decidua vera; the rest of the decidua.

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.

DEVELOPMENT AFTER IMPLANTATION (EMBRYONIC STAGE)

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 *extra-embryonic mesoderm*.

Multiple cavities appear in the latter that coalesce to form extra-embryonic coelom; this separates the extra-embryonic 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***.

The Umbilical cord

As development and growth continue, the amniotic cavity enlarges at the expense of the extra-embryonic coelom.

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 intra-embryonic 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 foetal blood***.

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

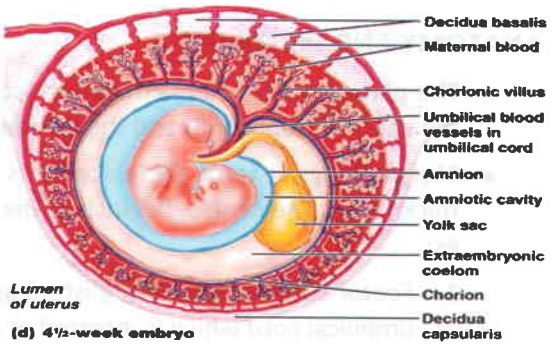


Fig 2-3: Early embryonic development

03 THE PLACENTA, CORD, AND FOETAL MEMBRANES

- *Anatomy and histology*
- *Placental physiology*
- *Placental abnormalities*
- *The umbilical cord*
- *The foetal membranes*
- *Amniotic fluid*
- *Foetal circulation*

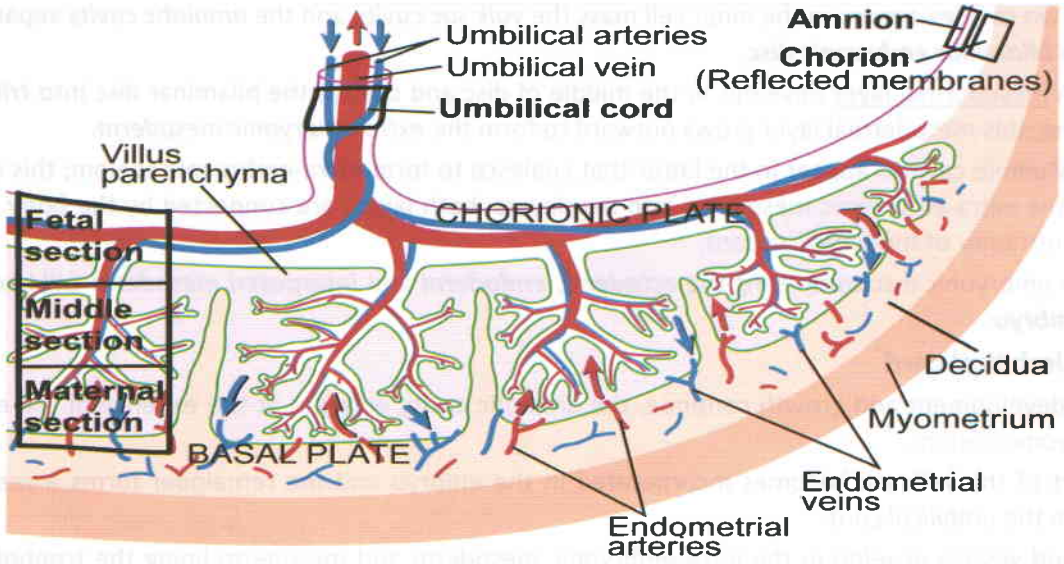


Fig 3-1: The Placenta

ANATOMY AND HISTOLOGY

- **The placenta** represents the interface between the mother and the foetus. 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 centre, 0.5 inch at the periphery and about 500 gm in weight. It has 2 surfaces:
  1. *Foetal surface:* Smooth, glistening and is covered by amnion which is reflected on the umbilical cord which is inserted at or near the centre of this surface.
  2. *Maternal surface:* Dull greyish red in colour, divided into 10-38 lobules (called cotyledons). Cotyledons are separated by fibrous tissue which is avascular.
- **Attachment:** 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, foetal and maternal blood do not communicate as the foetal blood circulates in the vessels inside the chorionic villi.
- **The placental barrier:** tissues that separate maternal and foetal blood are called collectively the placental barrier. Placental barrier is composed of the wall of the foetal 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.

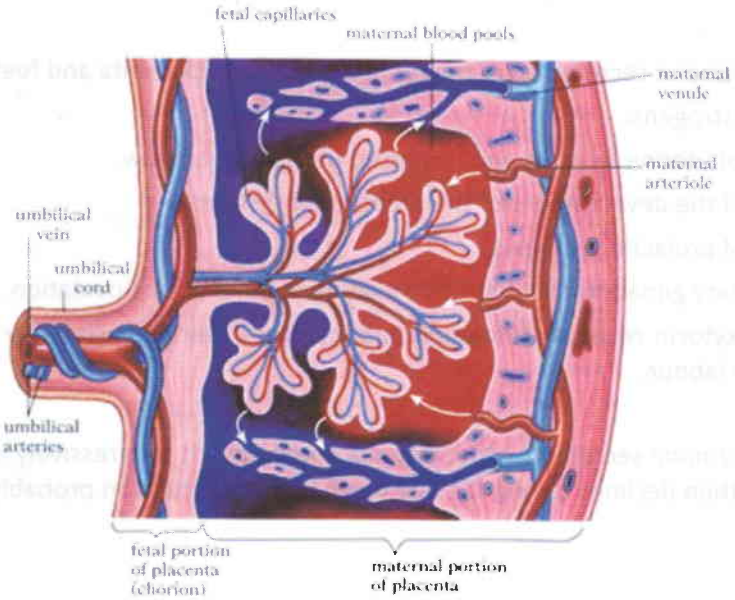


Fig 3-2: Chorionic Villi

B) ENDOCRINE FUNCTION:

1. Human chorionic gonadotrophin (hCG):

- It is a *glycoprotein hormone* produced by the *syncytiotrophoblast*, consisting of an α-subunit (similar to all anterior pituitary hormones) and a β-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 every 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, multifoetal pregnancy, Rh isoimmunisation, and Down syndrome. Levels are abnormally low in cases of impending abortion and ectopic pregnancy. It can be used as a screening marker for cases of choriocarcinoma, and follow up of gestational trophoblastic disease (GTD).

2. Human placental lactogen (hPL):

- HPL is a large *protein hormone* secreted from the *syncytiotrophoblast.* HPL starts to appear since early pregnancy and *steadily increases* till before labour.
- 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 foetus via stimulating glycogenolysis, and inhibiting maternal peripheral glucose uptake (anti-insulin).

  B. maintain free fatty acids flow to the foetus.

  C. Inhibits gluconeogenesis to preserve amino acids flow to the foetus.
- HPL levels are *high* in twin gestation, Rh isoimmunisation, 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 foetus).
- Functions of Oestrogens:
  a. 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 labour.

4. Progesterone (P):

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

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 haemorrhage. A part of the placenta may be retained after labour causing postpartum haemorrhage.

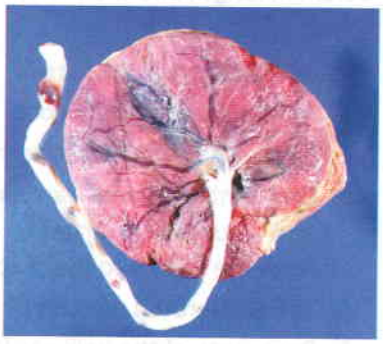


Fig 3-3: Normal placenta

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 haemorrhage and in this case we find a circular gap in the membranes from which vessels pass to the edge of the main placenta.

B) ABNORMALITIES IN SIZE AND WEIGHT:

Increase in size and weight in syphilis (syphilitic placenta is not oedematous), hydrops foetalis and Gest. Diabetes. Placenta > 600 gm in weight, and > 5 cm thickness on US.



Fig 3-4: Bipartite placenta



Fig 3-5: Succenturiate Placenta

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 haemorrhage from the maternal vessels of the decidua. They occur more in hypertensive conditions.

THE UMBILICAL CORD

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



Fig 3-6: The umbilical Cord

The umbilical cord consists of:

1. Amniotic membrane coat.
2. Extra-embryonic mesodermal tissue (Wharton's jelly).
3. One umbilical vein carrying oxygenated blood to the foetus and 2 umbilical arteries carrying the deoxygenated blood from the foetus 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.

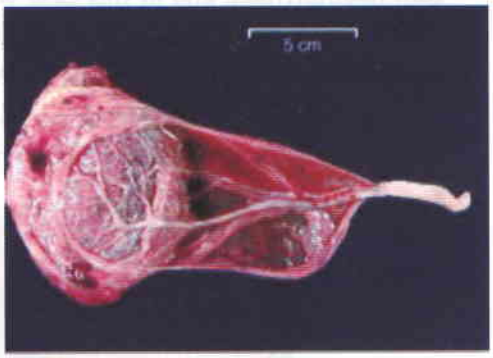


Fig 3-7: Velamentous cord insertion

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 foetus passes through a loop formed by abnormally long cord. If tight, asphyxia results from interference of circulation.
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 foetus.
   b. Too short (< 35 cm): this may be true or apparent (due to coiling around neck). It predisposes to; intrapartum haemorrhage due to premature separation of the placenta, prolonged 2nd stage of labor due to delay descent, and inversion of the uterus.
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 foetal haemorrhage.



Fig 3-8: True and False Knots of the cord

THE FOETAL 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 greyish membrane which lines the chorion. It covers the foetal surface of the placenta and the umbilical cord. The amniotic sac contains the foetus 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 pregnancy, reaching about one litre at full term (from 0.5 to 1.5). Both the foetus 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).

FOETAL CIRCULATION

- **One umbilical vein** carries ***oxygenated blood*** from the placenta to the foetus. 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 foetus 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 foetus 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 foetal hypogastric arteries are obliterated* to form the hypogastric ligaments (lateral umbilical ligaments).

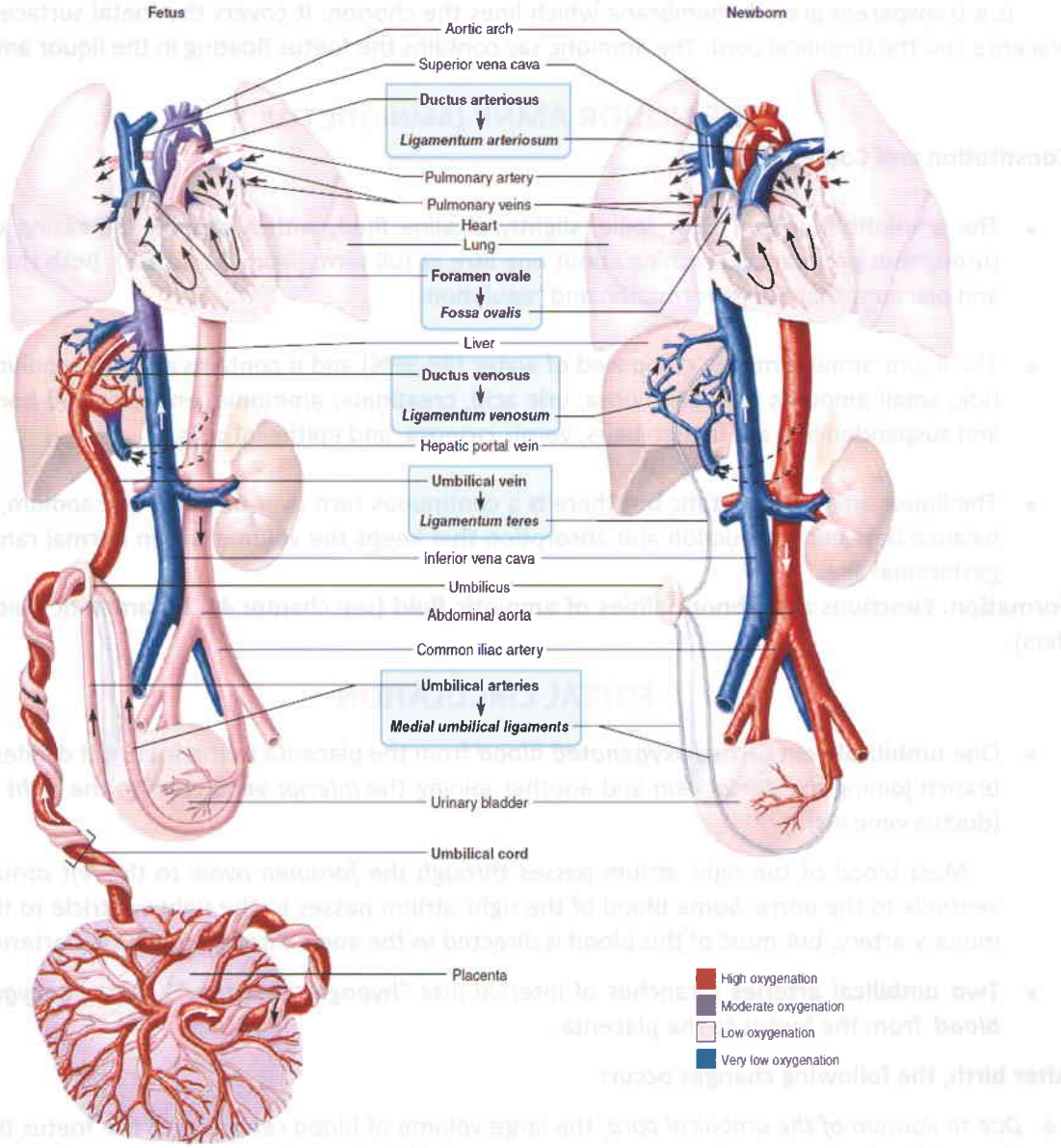


Fig 3-9: Foetal Circulation

04 PHYSIOLOGY OF THE MENSTRUAL CYCLE

- *Genital changes*
- *Breast changes*
- *Skin changes*
- *Metabolic changes*
- *Urinary changes*
- *Gastro-intestinal changes*
- *Cardiovascular changes*
- *Respiratory changes*
- *Skeletal changes*
- *Endocrinal changes*

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:
  - Oestrogen and progesterone leading to myometrial hypertrophy and hyperplasia.
  - The contents of the uterus i.e. foetus, placenta and amniotic fluid ..etc.
- **The Weight:** The weight increases from 50 gm to reach 1000 gm 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.

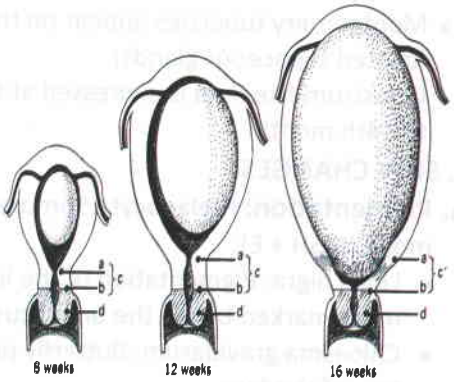
B) The Lower Uterine Segment (LUS):

- 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 4-1: Formation of the LUS

DIFFERENCES BETWEEN THE UPPER AND LOWER UTERINE SEGMENTS:

Upper uterine segment (UUS)	Lower uterine segment (LUS)
▪ Active during labour ▪ Contracts and retracts to become shorter & thicker. ▪ Thick wall: It consists of 3 muscle layers: – *Outer longitudinal* – *Middle oblique (for haemostasis)* – *Inner circular.* ▪ Covered by adherent peritoneum. ▪ The membranes are firmly attached.	▪ Less active during labour ▪ dilates and stretches to be longer and thinner ▪ Thin wall: It consists of 3 muscle layers: – *Outer longitudinal* – *Middle oblique layer is defective* – *Inner circular* ▪ Covered by loose peritoneum. ▪ The membranes are loosely attached.

C) The Cervix:

- Oedema and congestion; it becomes soft "Goodell's 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 labour.
- Cervical ripening mainly at the end of pregnancy due to oedema 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) progesterone (P) & prolactyin (PRL)

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



Fig 4-2: 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



Fig 4-3: Linea nigra



Fig 4:4 Striae gravidarum

B. Striae gravidarum "stretch marks":

These are pink lines in the flanks due to rupture of the subcutaneous elastic fibres as a result of stretch of the abdominal wall during pregnancy. After labour, the colour 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 transfer of glucose to the foetus (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 oestrogen 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 oestrogen together with pressure of the uterus on the ureter. This effect 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 *urinary tract infection (UTI).*

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 esophagitis" 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, leading 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 increases by 45% while RBCs increase only 15% leading to hemodilution.
- Hemodilution causes physiological anaemia 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 increased 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. Displacement 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 oedema, 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 centre.
- Limitation of movement of the diaphragm in late pregnancy causes dyspnoea.

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 labour.
- ***The Thyroid gland:*** increases in size and activity (physiological goitre). 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.

05 ANTENATAL CARE AND HIGH RISK PREGNANCY

- *Antenatal Care*
- *Postnatal Care PNC*
- *The High Risk Pregnancy*
- *Maternal Mortality*
- *Foetal and Neonatal Mortality*

ANTENATAL CARE

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. 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, AIDS, hepatitis B and C if necessary, especially in the first pregnancy.

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 µg/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 pregnancy according to FDA classification:

- ***Group: A*** Safe during pregnancy. Examples are supplementary vitamins & levothyroxine
- ***Group: B*** Risky in animal, no enough data on humans. Examples are penicillin & cephalosporin
- ***Group: C*** Risk in human cannot be ruled out. Examples are corticosteroids & NSAID.
- ***Group: D*** Risky in human pregnancy, but the benefits may outweigh the risks. Tetracycline & streptomycin
- ***Group: X*** Contraindicated in pregnancy, may cause adverse foetal effects. If really needed, termination of pregnancy is indicated . Examples are anticancer chemotherapy & warfarin.

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. In same cases GIT motility regulators are given.

- ***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:*** *f*requency, 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 (VV):*** 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 and 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 diseases or disorder that may a endanger the life or affect the health of the mother, foetus, or the newborn.

High risk pregnancy may be associated with:

a. ***Personal factors:*** elderly primigravida, pregnancy above 40 years and grand multipara (5th pregnancy or more)
b. ***Severe medical conditions affecting the mother:*** such as uncontrolled diabetes mellitus, cardiac disease grades III and IV, artificial heart valves, systemic lupus Erythematosus (SLE), and sickle cell disease.
c. ***Recurrent poor obstetrical outcomes such as:*** recurrent pregnancy loss (RPL), recurrent still birth (RSB), recurrent early rupture of membranes (ROM), and recurrent pre term labour (PTL).
d. ***Obstetrical complications*** that require specialized care such as: severe pre-eclampsia (PE) or eclampsia, HELLP syndrome, severe intrauterine foetal growth restriction (IUGR), and multiple high risk factors.
e. ***Conditions that may require invasive procedures for foetal diagnosis or therapy as:*** Immune and non-immune hydrops foetalis, and congenital anomalies or genetic disorders.

High risk pregnancy is identified by proper assessment through history, clinical examination, and special investigation of the mother during preconceptional visit, the 1st antenatal visit or during the return antenatal visits.

Once identified, the mother should be transferred to a specialized center ready for such high-risk cases.

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 previa, 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:** 52/100,000 deliveries in year 2013 statistics
- **Common Causes of Maternal Mortality:**
  1. Obstetric Haemorrhage: 29.6%
  2. Preeclampsia (PE) and Eclampsia: 14.9%
  3. Puerperal and post-abortive sepsis: 3.7%
  4. Other factors as: Pulmonary embolism and DIC, medical and cardiac problems, anaesthesia complications: 51.8%
- 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



06 PRENATAL DIAGNOSIS OF FOETAL CONGENITAL MALFORMATIONS

- *Foetal Abnormalities*
- *Screening tests*
- *Diagnostic tests*
- *Prenatal genetic screening*
- *Screening for Down syndrome*

Prenatal diagnosis entails in utero diagnosis of foetal **congenital** or **chromosomal** abnormalities, to facilitate dealing with such problems appropriately during pregnancy or immediately after delivery. Prenatal diagnosis can be achieved in early pregnancy through;

- ***Screening tests:*** as *maternal blood sampling* and *level 2 ultrasonography* **and/or**
- ***Diagnostic tests:*** as *foetal sampling* and level *3 ultrasonography*.

FOETAL STRUCTURAL CONGENITAL ANOMALIES

These describe abnormalities or malformation in organ development as occurs in:

- *Neural tube defects (NTDS)* as anencephaly, spina-bifida, hydrocephalus, etc...
- *Abdominal wall defects* (exomphalos) and congenital *diaphragmatic hernia*.
- *Cardiac anomalies* as ASD , VSD, and transposition of great vessels
- *Renal anomalies* as renal agenesis, unilateral or pelvic kidney and obstructive uropathy.
- *Tumours* as sacro-coccygeal teratoma, congenital goitre, ...
- *Lymphatic channel obstruction* as cystic hygromas, and many others

FOETAL CHROMOSOMAL ANOMALIES:

- ***Aneuploidy*** occurs when the chromosome **number** is not an exact multiple of **23**. Most common aneuploidy are **trisomy 21** (Down syndrome), trisomy **18** (Edward syndrome) and trisomy **13** (Pateau syndrome).
- ***Nondisjunction*** is an error in **meiosis** that results in a gamete with one chromosome too few, or too many. They increase with advancing maternal age. Fertilization of the gamete with one extra chromosome results in a conception with **47** chromosomes. Nondisjunction may involve sex chromosomes resulting in abnormalities such as 47 XXY, 47 XYY, etc...

N.B.: Maternal age **above 35** years is a risk factor for a foetus with ***nondisjunction*** chromosomal defect, **aneuploidy**, and some structural **congenital anomalies**. The incidence increases with advancing maternal age.

HIGH RISK CASES FOR PRENATAL SCREENING AND DIAGNOSIS

1. Maternal **age** more than **35** years (increased chromosomal and structural anomalies) Cases with a history of **familial** genetic disease, past **obstetric** history of previous babies with congenital or chromosomal anomalies, and cases with previous recurrent pregnancy loss.
2. History of exposure to **teratogenic drugs** or large doze ionizing **irradiation** in early pregnancy.
3. Exposure to certain **infections** in the pre-conception period or during early pregnancy.

TYPES OF PRENATAL TESTS

- **SCREENING TESTS:**

Screening tests should be *cheap, safe, non-invasive,* and *readily available*, with a reasonable diagnostic accuracy & low false negative rates (sensitivity). The aim of screening is to select a subgroup which will benefit from more complicated expensive diagnostic procedures. Prenatal screening include;

- Maternal *serum biochemical markers* tests (double marker DMT & triple marker TMT)
- US screening for nuchal translucency (NT)
- New techniques as *cell-free foetal DNA* (cfFDNA).

DIAGNOSTIC TESTS:

Diagnostic tests have a high degree of accuracy with low false positive rates (specificity), they are generally more complicated, more expensive, and sometimes more invasive, that is why they are reserved to high risk cases with positive screening tests only and not to general low risk population. Prenatal diagnosis include;

- *Chorionic villus sampling* (CVS) and
- *Amniocentesis* for chromosomal anomalies
- *Advanced ultrasonography* (level 2) for foetal anatomy survey to diagnose various types of structural congenital anomalies and point to markers for possible chromosomal defects.

SCREENING TESTS FOR FOETAL CONGENITAL ABNORMALITIES

1. MATERNAL SERUM BIOCHEMICAL MARKERS

A maternal **serum** blood sample, may allow testing for different **markers** which **separately** or in **combination**, may point to an increased risk of having a child with a chromosomal defects.

a) ***AFP:*** Alpha Fetoprotein is glycoprotein produced by the yolk sac, foetal liver and GIT. Levels are **increased** in cases with open neural tube defects **(NTDs)** and **Turner** syndrome, while, levels are abnormally **decreased** in cases with trisomy 21 (Down syndrome).

b) ***HCG:*** human chorionic gonadotropin levels correlate with gestational age in normal pregnancies. Levels are abnormally **increased** in cases with trisomy 21.

c) ***PAPP-A:*** Pregnancy associated plasma protein A is a placental protein that shows abnormally **decreased** levels in trisomy 21 foetuses.

d) ***UE3:*** Unconjugated Oestriol levels are **decreased** in association with trisomy 21 foetuses

THE DOUBLE MARKER TEST (DMT)

Combining maternal **serum markers** (hCG & PAPP-A) together with **US** measurement of the nuchal translucency **(NT)** at the foetal neck in one test performed at **11-13** weeks gestation, as shown later, can improve the predictive accuracy for the risk for **Down syndrome** to **> 85%**, with a false positive rate of < 5%. This will allow for early diagnosis and early intervention when indicated.

- **Down syndrome** foetuses usually show **high** hCG, **low** PAPP-A levels, together with increased NT > 3.0 mm at 12 weeks gestation. A high risk score calls for further diagnostic tests.
- **Risks** for **trisomies** 18 & 13 (Edwards and Pateau syndromes) can be included in screening by the Double marker test (DMT).

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, taking in consideration maternal age, weight, and gestational age.

Down syndrome risk is high when the test shows **high** hCG levels with **low** MSAFP and uE_3 levels. The test is less commonly used nowadays in favor for the double marker test which is both more accurate and provides and earlier first trimesteric diagnosis.

2. ULTRASOUND SCREENING

- **US MEASUREMENT OF NUCHAL TRANSLUCENCY (NT):**

  Measurement of skin fold thickness of the foetal neck at 11 -13 weeks gestation, shows good correlation with the presence of foetal chromosomal abnormalities. NT thickness **> 3.0 mm** weeks should call for more invasive diagnostic work up to exclude Down syndrome as CVS and amniocentesis (see later)

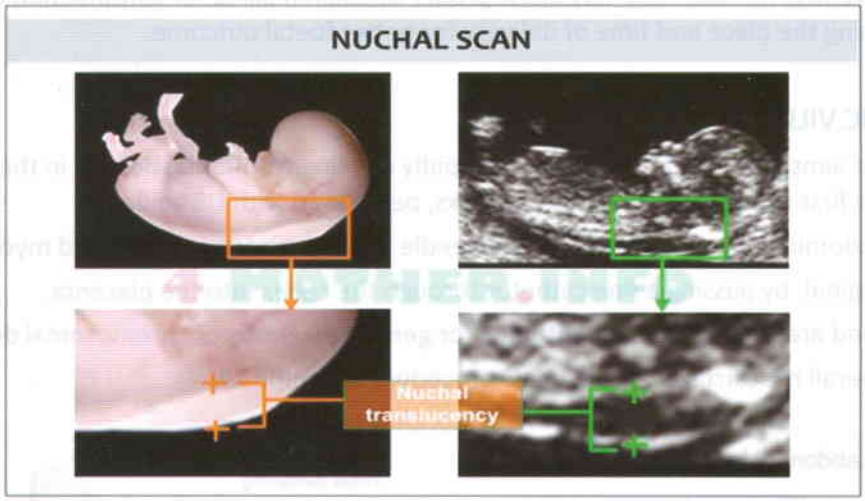


Fig 6-1: Nuchal translucency

- **Adjusting maternal age:** The risk of Down's syndrome increases with **maternal age** from **1/600** before 25 years to **1/40** with age > 44 years. The individual risk for each woman may be calculated by taking her age-related risk and then adjusting this on the measurements obtained for the NT, hCG and PAPP-A.
- **Adding soft markers:** Improving the accuracy of the results of the screening tests for Down's syndrome can be refined by adding further soft markers such as measuring the **nasal bone**. These increase the sensitivity of the test and reduce the false positive rate. However prenatal diagnostic test must still be performed to reach a definitive diagnosis.
- **Explaining the risk score:** It is also important to explain to women that this initial test is simply a **screening** test and not a diagnostic test. A **low-risk** result of even **1:1000** still carries a very remote chance for having an affected baby, while with a very **high risk** score of 1:10 , 9 out of 10 or 90% of fetuses would be normal. The diagnostic tests available to her should be explained.

3. CELL FREE FOETAL DNA (cfFDNA) for screening and diagnosis:

Fragments of **foetal DNA** are released in minute amounts by the foetus and the placenta into maternal circulation with increasing concentration from 10 weeks gestation onwards. By amplifying cfFDNA using **PCR** it is possible to check the foetal genotype, define the sex, and exclude many chromosomal abnormalities starting **10-12** weeks gestation.

The test although being a novel promising **non-invasive diagnostic** technique with high accuracy yet its use is limited by an expensive test, not readily available in many centres, and is still of **limited** capacity in diagnosing only 5-12 chromosomes at a time compared to CVS and Amniocentesis which give a complete accurate genetic diagnosis of all 23 pairs of chromosomes in human pregnancies.

DIAGNOSTIC TESTS FOR FOETAL CONGENITAL ABNORMALITIES

1. ULTRASOUND DIAGNOSIS OF FOETAL CONGENITAL ANOMALIES

a. **First Trimester:** by end of first trimester some important anomalies can be diagnosed as anencephaly, cystic hygromas, and some skeletal abnormalities

b. **Second Trimester Anomaly scan:** in the 2nd trimester between **18-22** weeks a detailed foetal anatomy survey is usually performed for diagnosis of important major foetal congenital anomalies as; NTDs, most of the skeletal, cardiac, renal and GIT anomalies, in addition to diaphragmatic and ventral hernias. This may allow proper decision making for continuation of pregnancy or planning the place and time of delivery for better foetal outcome.

2. CHORIONIC VILLOUS SAMPLING (CVS)

CVS procedure aims at taking a sample of the rapidly dividing **trophoblastic** cells in the placental bed during the first trimester as early as **11** weeks, performed with US guidance;

- Transabdominal: by passing a 22 gauge needle through abdominal wall and myometrium
- Transvaginal: by passing a fine catheter through the cervix into the placenta.

- Cells obtained are subjected to **karyotyping** for genetic diagnosis of chromosomal defects
- N.B.: the overall miscarriage risk for a CVS procedure is around **2%**

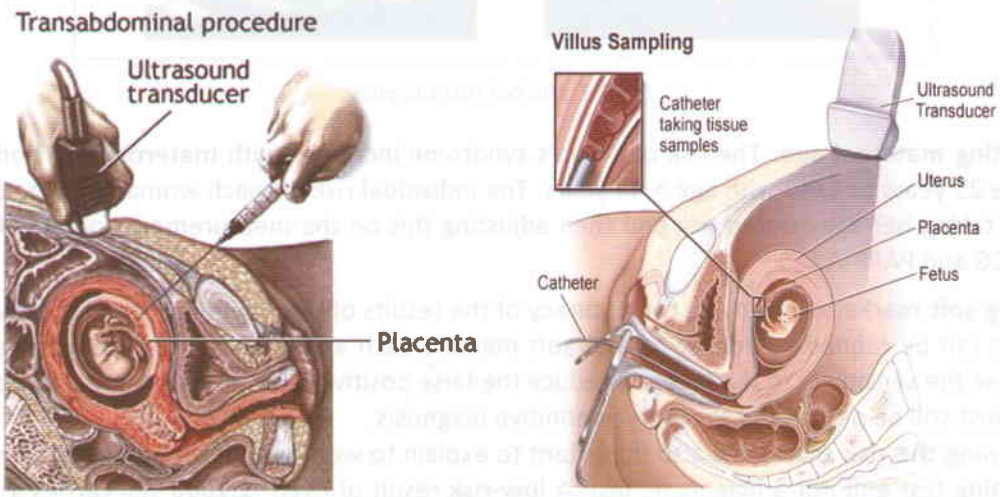


Fig 6-2: Chorionic villus sampling CVS
Prenatal Diagnosis of Foetal Congenital Abnormalities

3. AMNIOCENTESIS

Amniotic fluid contains amniocytes and fibroblasts shed from foetal membranes, skin, and GIT. In amniocentesis a sample (15-20 ml) of amniotic fluid is obtained via a **fine needle** 18-22 gauge introduced through the **abdominal wall** and myometrium under **ultrasound** guidance.

- *Timing of amniocentesis:* **14-16** weeks gestation to obtain adequate fluid sample.
- Foetal cells obtained will be subjected to **Karyotyping**, foetal sexing, **DNA** analysis using polymerase chain reaction (PCR), and enzyme assay to achieve a final accurate diagnosis of several chromosomal, biochemical, and hereditary disorders within **2-4** weeks.
- *Risks of procedure;* apart from a **1-2%** risk of **miscarriage**, amniocentesis carries also a definite however very small risk of foetal infection, rupture of membranes, accidental haemorrhage, and feto-maternal transfusion with the risk of sensitization in cases of RH negative mothers.
- **N.B.:** The advantage of *CVS* over *amniocentesis* is that it is performed earlier allowing earlier diagnosis and possible interventions. It is also preferred in diagnosis of haemoglobinopathies providing a larger cell volume for DNA testing inspite of its slightly higher miscarriage risk.

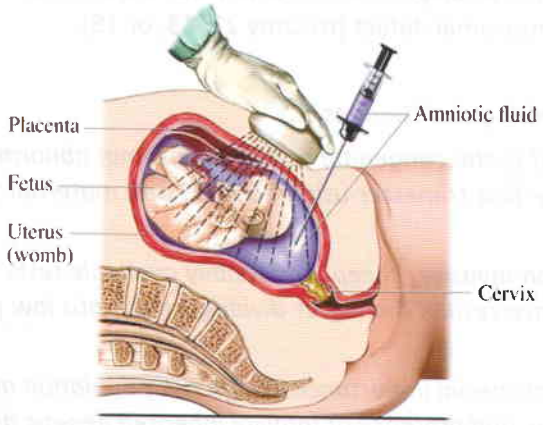


Fig 6-3: Amniocentesis

4. CORDOCENTESIS

- *Cordocentesis* entails obtaining foetal blood sample or injecting foetal RBCs through the umbilical cord in utero
- *Indications:* It is performed when fetal blood is needed, or when a rapid full culture for karyotype is needed. The most common reason is for suspected severe fetal anemia such as in cases of RH incompatibility with availability of immediate intra-uterine transfusion with packed RBCs.
- *Technique:* A needle is passed under ultrasound guidance through the abdominal wall and myometrium and, most commonly, into the umbilical cord at the point where it inserts into the placenta.
- *Timing:* Cordocentesis can be performed from about 20 weeks' gestation. The risk of miscarriage varies with indication and position of the placenta.

PREIMPLANTATION GENETIC DIAGNOSIS (PGD):

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 foetus. These cells provide sufficient DNA for PCR directed molecular analysis for inherited diseases, or fluorescent in situ hybridization for aneuploidy. Preimplantation biopsy for PGD involves participation in an IVF program and is currently available for a limited number of genetic conditions.

SCREENING TESTS FOR TRISOMY 21 (DOWN SYNDROME):

a. First trimester:

- *US scan:* for abnormal NT measurement and Nasal Bone hypoplasia at **11-13** weeks
- *Double Marker Test (DMT):* US + MSAFP & PAPP-A at **11-13** weeks

b. Second trimester:

- *Triple Marker Test (TMT):* MSAFP, hCG, & UE3 at **14-16** weeks.
- *US Foetal Anatomy Survey (FAS):* for soft markers at **18-22** weeks as hypoplastic nasal bone, tricuspid regurge, short femur, pelvicalyceal dilatation, and cardiac echogenic focus, etc...

DIAGNOSTIC TESTS FOR TRISOMY 21:

- *CVS:* done starting **11** weeks in cases with high risk screening tests (US, DMT or TMT)
- *Amniocentesis:* done at **14-16** weeks in cases with high risk screening tests refusing CVS
- *Cell free foetal DNA:* starting **10** weeks can be used as non-invasive screening test (however expensive) or as diagnostic test whenever US, DMT, or TMT show a definite high risk result for a chromosomal defect (trisomy 21, 13, or 18).

Key points in Prenatal Screening and Diagnosis

- *Prenatal diagnosis of foetal congenital and chromosomal abnormalities can be achieved in utero as early as the first trimester using both US and maternal serum biochemical markers.*
- *Screening include non-invasive, cheep, and readily available tests with reasonable accuracy and low false negative results aiming at dividing cases into low risk and high risk population*
- *Screening tests are of special importance in high risk population according to maternal age, past obstetric history, and presence of familial inherited genetic disorders.*
- *Non-invasive Screening tests in 1st & early 2nd trimester include US, DMT and TMT tests*
- *Invasive Diagnostic tests as CVS in 1st trimester and Amniocentesis in 2nd trimester are reserved to cases with positive screening tests and high risk population*
- *Cell free FDNA is a non-invasive test that is valuable for both screening and diagnosis as early as 10 weeks gestation.*
- *Compared to cfFDNA, CVS and amniocentesis have a better capability of giving a full genetic diagnosis for all 23 pairs of chromosomes.*

INTENDED LEARNING OBJECTIVES (ILOs):

- To understand what does foetal congenital abnormalities include
- The difference between chromosomal abnormalities and congenital malformations
- The aim of screening and its different available methods
- The methods of diagnosis, their techniques, limitations and accuracy
- The future trends in screening and diagnosis

07 SPONTANEOUS ABORTION
EARLY PREGNANCY COMPLICATIONS

- *Types of spontaneous abortion*
- *Aetiological and Risk factors*
- *Complications of abortion*
- *Septic abortion*
- *Recurrent abortion*
- *Cervical Insufficiency*

Abortion is the term used to describe pregnancy loss before foetal **viability.** According to the WHO definition this applies to pregnancies **> 20** weeks gestation and foetal weight **> 500** gm. Viability (the capability to survive after delivery) may be extended to **> 24** weeks gestation in centres with less advanced neonatal services.

Spontaneous abortion (miscarriage) will occur in nearly **10-15%** of clinically recognizable pregnancies with nearly **80%** occurring **< 8.0** weeks gestation. The incidences however sharply **declines** with advancement of pregnancy reaching **2-3%** between **12-28** weeks gestation.

Although some cases of spontaneous abortion are **symptomless**, being recognized accidentally during routine US examination, yet the majority are clinically recognizable by the combination of both vaginal **bleeding** and lower abdominal **pain** of variable severity.

TYPES OF SPONTANEOUS ABORTION

1. THREATENED ABORTION

- Threatened abortion describes pregnancies complicated by **mild** vaginal **bleeding < 20th** week. **Pain** is not prominent but may occur as dull aching pelvic pain with mild bleeding.
- **PV** examination reveals a closed cervix, and **US** reveals an intact pregnancy.
- Almost **25%** of pregnant women will experience some degree of mild vaginal bleeding in their 1st trimester, which does not affect the course of their ongoing pregnancy.
- Almost **25-50%** of cases with threatened abortion may end up with a pregnancy loss.
- Threatened abortion is associated with a slightly increase **risk** for a preterm birth.

2. INEVITABLE ABORTION

- Inevitable abortion describes pregnancies complicated by **significant** vaginal **bleeding** and **prominent** colicky lower abdominal **pain.**
- **PV** examination reveals a partially **dilated** internal cervical os
- **US** may reveal placental separation with products of conception still totally **retained** in utero.
- Inevitable abortion will eventually end by **expulsion** of the products of conception and termination of pregnancy. The foetus may be alive or dead at the time of US scan.

3. INCOMPLETE ABORTION

- Incomplete abortion describes **incomplete expulsion** of the products of conception through the partially dilated internal os with some products of conception still **retained** in utero.
- In association to vaginal **bleeding** and **pain**, the patient will describe **passage** of **shreds** of skin or liver like tissue which represent some products of conception and blood clots.
- **PV** examination reveals a subinvoluted uterus, with a partially **dilated** cervix
- **US** reveals **retained** products of conception.

4. COMPLETE ABORTION

- Complete abortion describes **complete** expulsion of **all** products of conception through the cervix. **Pain** and cramps will **stop** and **bleeding** gradually becomes **minimal**.
- **PV** examination reveals an almost **normal** size uterus and a **closed** internal os
- **US** reveals an **empty** cavity with slightly thickened decidua and **no** products of conception.
- **Symptoms** of pregnancy will rapidly **disappear** and pregnancy **test** will rapidly **decline**.

5. MISSED ABORTION

- The term missed abortion is used when **death** of an embryo (or foetus) occurs but is **retained** in the uterus without expulsion, usually for several weeks.
- The condition may be **symptomless** at the start but after some time vaginal brownish **discharge** and recurrent bleeding may start to occur, with loss of symptoms of pregnancy.
- **PV** examination will reveal a smaller than expected uterus with closed internal os.
- **US** examination is diagnostic revealing intrauterine **foetal demise** or anembryonic sac, with all content in utero. It will also determine gestational age at which pregnancy has stopped.
- **Coagulation problems** may develop with hypofibrinogenaemia and DIC if pregnancy has been prolonged for several weeks, especially in the 2nd trimester due to the release of thromboplastin like substances leading to hypofibrinogenaemia and DIC.



Fig 7-1: Clinical types of spontaneous abortion



Fig 7:2 An-embryonic sac



Fig 7:3 IUED (absent FHR)

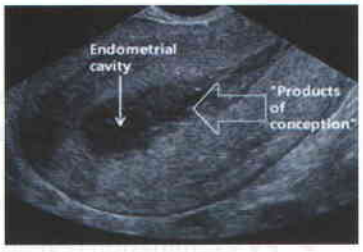


Fig 7:4 Incomplete abortion

AETIOLOGY AND RISK FACTORS OF SPONTANEOUS ABORTION

It is sometimes difficult to identify the exact cause for a spontaneous abortion whether due to **foetal, maternal,** or **combined** foetal and maternal causes.

(A) FOETAL CAUSES

1. **Foetal Genetic Abnormalities:** gross chromosomal abnormalities of the embryo or foetus are the commonest cause of spontaneous abortion in > 70% of cases of 1st trimester pregnancy loss. Aneuploidy is the commonest disorder including monosomy, trisomy, and triploidy. They usually occur sporadically as gene mutations.
2. **Mendelian inheritance:** Autosomal or X-linked dominant or recessive disease
3. **Foetal congenital anomalies (FCA):** major FCA incompatible with life as anencephaly
4. **Foetal Congenital Infections (FCI);** as TORCH with severe foetal affection

(B) MATERNAL CAUSES

1. **Uterine Abnormalities:**
   a. ***Congenital:*** as septate and bicornuate uterus
   b. ***Acquired:*** as large uterine leiomyomas, endometrial polyps, and intrauterine adhesions
   c. ***Cervical incompetence:*** see later
2. **Immunologic Disorders as:** Antiphospholipid syndrome **(APS):** where some women with SLE produce antibodies against their own vascular system and feto-placental tissue causing thrombosis and late 1st trimester recurrent pregnancy loss (see later).
3. **Severe trauma:**
   - *External:* as with car accidents, fall from heights, or direct hit on the abdomen.
   - *Internal:* as with attempts to induce illegal abortion mechanically, or during foetal sampling by amniocentesis, and chorionic villous sampling (CVS).
4. **Maternal Chromosomal Factors:** occasionally foetal chromosomal abnormalities may occur as a result of balanced translocations or inversions in either or both parents. These cases are met with more in cases of unexplained recurrent pregnancy loss (see later)
5. **Drugs:** some teratogenic drugs may cause **foetal demise** in utero.

FACTORS INCREASING THE RISK OF PREGNANCY LOSS

1. *Increased maternal age:* incidence increases with maternal age > 35 years and more > 40 years
2. *Multiparity:* incidence increases with increased parity > 5
3. *Previous and recurrent pregnancy loss;* especially > 2 successive abortions
4. *Heavy smoking and regular alcohol intake*
5. *Psychosocial stress, and domestic violence*
6. *Infections as Mycoplasma, Listeria and Toxoplasma*
7. *Medical disorders;* as severe thyroid disorders, uncontrolled DM, and SLE
8. *Morbid maternal obesity*

COMPLICATIONS OF ABORTION

1. **Haemorrhage**; which if severe may cause haemorrhagic shock
2. **Infection**; may cause endometritis, salpingitis, and peritonitis, all leading to infertility
3. **Septicaemia**; which if severe may cause septicaemic shock, DIC, and renal failure
4. **Complications of Surgical Evacuation SE**: as uterine perforation and intestinal injury

MANAGEMENT OF SPONTANEOUS ABORTION

A) Threatened Abortion:

- ***Reassurance*** by US to ensure an intact pregnancy with > 90% chances to continue safely
- ***Physical*** *and* ***mental rest*** may decrease stress, and help patient stabilization.
- ***Progesterone*** *support:* the use *of* oral, vaginal, or IM PRG although popular, yet is at least controversial without evidence of being really beneficial, except in cases with luteal phase defect.

B) Inevitable Abortion:

Hospitalization and immediate Anti-shock measures for cases with severe blood loss, followed by termination of pregnancy to control pain and excessive blood loss, via:

- ***Surgical Evacuation (SE) and curettage:*** for 1st trimester inevitable abortions.
- ***Medical Evacuation (ME):*** using IV oxytocin drip for 2nd trimester cases to facilitate rapid expulsion of intrauterine contents with minimizing blood loss, and decreasing risks of infection.

C) Incomplete Abortion:

- ***Surgical Evacuation****:* is indicated in cases where bleeding is excessive or contents are considerable. It is also indicated in cases with retained placenta of a 2nd trimester abortion.
- ***Medical evacuation:*** in cases where bleeding is mild and contents are minimal the use of oral or transrectal **Misoprostol** (PGL E1) effectively helps rapid expulsion of retained products of conception without the need for surgery. Ecbolics as **Ergometrin** (oral or IM) have been also used for the same purpose.

D) Missed Abortion:

- ***Conservative Approach:*** wait few weeks for spontaneous expulsion if minimal or no bleeding
- ***Surgical Induction of Abortion:***
- *Suction Evacuation:* for cases with a small anembryonic sac after failed conservative approach
- *Surgical evacuation and curettage:* for cases of IUFD < 12 weeks.
- *Hysterotomy (CS <24 weeks):* rarely resorted to in late 2nd trimester IUFD with failed medical induction of abortion using repeated high dose Misoprostol.
- ***Medical Induction of abortion:***
- *Misoprostol (PGL E1):* 200 ug tablets (oral, sublingual, or vaginal) is the first choice for medically induced abortion in 2nd trimesteric IUFD. Doses of 400 – 600 ug repeated every 4-6 hours, will usually result in complete expulsion of all contents within 1-3 days of starting induction.
- It can be used for both late 1st trimester and 2nd trimester IUFD with doses adjusted according to gestational age, number of foetuses, and presence of a previous uterine scar.

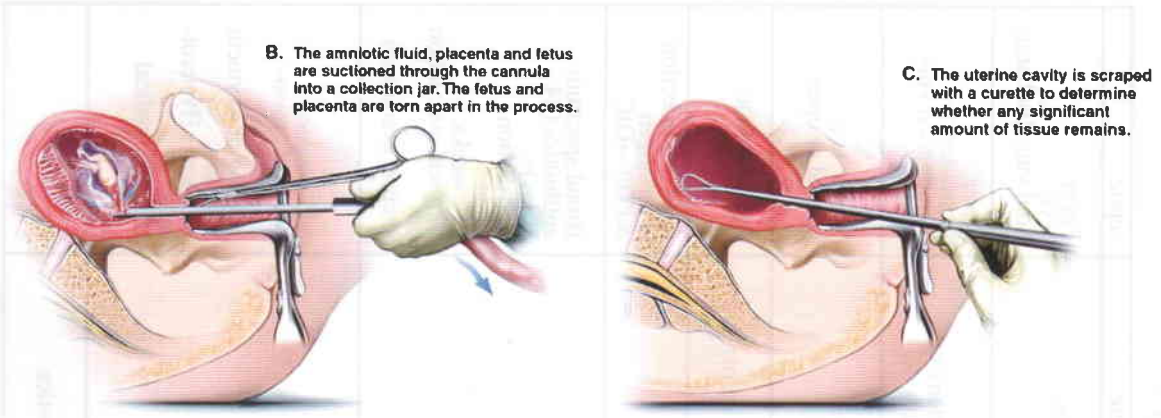


Fig. 7-4: Surgical evacuation by suction and curettage

SEPTIC ABORTION

Septic abortion describes any type of abortion that is complicated by **infection** whether missed, inevitable, or incomplete. Commonly involved bacterial organisms include *β-haemolytic Streptococci, Staphylococci, E. coli, and Clostridium welchii.*

RISK FACTORS

- Prolonged presence of retained products of conception (POC) as missed or incomplete abortion
- Pregnancy in presence of an IUD.
- Positive cultures for group B-haemolytic streptococcus infection (GBS) in early pregnancy.
- Criminal induction of abortion using non properly sterilized instruments.

DIAGNOSIS

- ***Symptoms;*** persistent **fever** with recurrent lower abdominal **pain**, vaginal **bleeding,** and malodorous vaginal **discharge**, in a patient with missed or incomplete abortion.
- ***Signs:*** the uterus is **soft** and may be **tender** and **subinvoluted** on bimanual examination.
- ***Ultrasound Findings:*** retained **POC** with heterogonous echo pattern and haematometra
- ***Laboratory investigations:***
  - ***CBC:*** will show increased ***TLC***, with shift to the left and sometimes *toxic granules.*
  - *Elevated **ESR,** and C reactive protein **(CRP)***

MANAGEMENT OF SEPTIC ABORTION

- **Antibiotics:** broad spectrum IV antibiotics covering both **aerobic** and **an-aerobic** organisms including Cephalosporins, Flagyl, and Clindamycin
- **Ecbolics** as Ergometrin (Methergine) and prostaglandins (Misoprostol) are also used to augment complete expulsion of all retained POC.
- **Surgical Evacuation:** to ensure **complete** evacuation of uterine contents, with gentle curettage.

SEQUEL OF SEPTIC ABORTION

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

All patients had a delay in the menstrual period with positive pregnancy symptoms and pregnancy test which is followed by :

	Threatened Ab.	Missed	Inevitable	Incomplete	complete	septic
Symptoms 1-Bleeding 2-cramping Pelvic pain	Usually mild + Usually absent -ve	Usually mild + Usually absent -ve	++or +++ Present +ve	++or +++ Present +ve	+ or –ve absent -ve	Fever +ve Foul vag. discharge
General exam. Bp Pulse (rate and volume)	Normotensive Average	Normotensive Average	May be shocked Hypotension Raid &low volume	May be shocked Hypotension Raid &low volume	Normotensive Average	Fever Normotensive Rapid+good volume
Pelvic exam. 1-Bleeding 2-Int. OS	Mild + closed	Mild + closed	Moderate/severe opened	Moderate/severe opened	Mild + closed	-ve or +ve Closed or open
Ultrasound exam (better T.V)	I.U gestational sac With living fetus	I.U gestational sac With dead fetus	Dead or living fetus +open Int. os.	Retained POC +open Int. os.	Empty uterus	Retained POC
More investigations	B-hCG quantitative	-CBC → infection -Fibrinogen &FDP→DIC				-CBC → infection -Fibrinogen &FDP→DIC
Treatment	Rest :physical ,sexual And reassurance	≤12 weeks Medical : misopristol 800 mg Surgical evacuation or suction evacuation	Resuscitation + Antishock measures ≤12 weeks: Surgical evacuation or suction evacuation	Resuscitation + Antishock measures ≤12 weeks: Surgical evacuation or suction evacuation		Broad spectrum antibiotics (24 hours)+ rehydration Then ≤12 weeks: Surgical evacuation or suction evacuation
	Progesterone 100 mg I.M or 400 mg vaginal	>12 weeks: Medical : misopristol 800 mg or oxytocin Infusion Surgical : Hysterotomy If failed medical	>12 weeks: Medical : misopristol 800 mg or oxytocin Infusion Surgical : Hysterotomy If failed medical	>12 weeks: Medical : misopristol 800 mg or oxytocin Infusion Surgical : Hysterotomy If failed medical		>12 weeks: Medical : misopristol 800 mg or oxytocin Infusion Surgical : Hysterotomy If failed medical
	Ttt of thyroid disease or DM	Antibiotics	Antibiotics	Antibiotics	Antibiotics	

RECURRENT PREGNANCY LOSS

Recurrent pregnancy loss (RPL) is the term used to describe the occurrence of more than 3 successive spontaneous abortions without intervening term pregnancies. It has been previously called **habitual abortion.** It may affect nearly 2% of couples. Most RPL occur in the 1st trimester.

AETIOLOGY

a. Anatomic uterine abnormalities; as uterine anomalies and submucus leiomyomas (SML)
b. Immunologic disorders as antiphospholipid syndrome (APS)
c. Thrombophilias; as protein-S and protein-C deficiencies
d. Genetic and chromosomal abnormalities
e. Endocrine disorders; as severe hypothyroidism, and uncontrolled DM
f. Incompetent isthmus; habitual 2nd trimesteric abortion (see later)

EVALUATION OF CASES WITH RPL

Evaluation may start after the second loss rather than waiting for a third abortion

1. **Detailed History:**
   - Age, parity, and family consanguinity
   - Chronic medical diseases and endocrine disorders; as DM, thyroid disorders, HN,
   - History of each previous abortion; timing, course, and presence of foetal abnormalities
   - Previous investigations including US, HSG, and hysteroscopy
   - Previous laboratory tests for thyroid function, DM, and Thrombophilias
   - Previous treatment during or in-between pregnancies if any.
2. **Bimanual pelvic examination:** to exclude local uterine causes as myomata
3. **TVS** to detect uterine anomalies, leiomyomas or signs suggestive of incompetent cervix.
4. **Hysteroscopy;** for evaluation of abnormalities within the endometrial cavity
5. **Chromosomal karyotyping** for the couple to exclude inherited disorders
6. **Laboratory test** for thyroid function, DM, HbA1C, etc.... if has not been previously performed
7. **Screening for APS** through testing for; Lupus anticoagulant (LAC), Anticardiolipin antibodies (ACA), Antithrombin III, protein-S and protein-C deficiencies, and B2 glycoprotein.

MANAGEMENT OF CASES WITH RPL

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

1. **Surgical correction of anatomic abnormalities;** removal of a uterine septum, endometrial polyp, or submucus leiomyomas (SML)
2. **Folic acid** 800 ug daily started before pregnancy and throughout 1st trimester
3. **PRG support** in luteal phase and throughout 1st trimester. Some recommend to continue till 3rd trimester as prophylaxis against 2nd trimester loss and PTL.
4. **Control of DM** and **thyroid disorders** before and during pregnancy
5. **Treatment of maternal infections** as Toxoplasma (Rovamycin), Syphilis (Penicillin), Herpes simplex virus HSV (Acyclovir), etc...
6. **Low dose aspirin (LDA)** 75-81 mg/day throughout pregnancy to protect against micro-thrombi and placental insufficiency
7. **Low molecular weight Heparin** in cases of thrombophilia and APS especially with history of IUGR, severe placental insufficiency, and recurrent late 1st trimester IUFD
8. **Cerclage** for cases strongly suspected to have isthmic incompetence (see later)

CERVICAL INSUFFICIENCY

(INCOMPETENT CERVIX)

Cervical insufficiency or incompetence is defined as inability of the uterine cervix to retain a pregnancy during the **2nd trimester** in absence of uterine contractions. The condition is uncommon complicating only **< 0.2%** of all pregnancies

AETIOLOGY AND RISK FACTORS

1. **Congenital Isthmic weakness** due to;
   - Congenital decreased cervical Elastin content
   - Congenital uterine anomalies as septate and bicornuate uterus
2. **Acquired isthmic weakness:**
   a) In association with *submucus leiomyomas and submucus fibroid polyps*
   b) In association with *Cervical trauma and lacerations:*
      - During difficult vaginal and instrumental deliveries
      - During forcible excessive cervical dilatation in a D & C procedure
      - After cervical conization for treatment of high grade CIN lesions
      - After cervical amputation during Manchester operation for uterine prolapse

   c) In association with *multifoetal pregnancy (MFP):* due to rapid progressive overstretch of the isthmic region and shortening of the cervix by multiple gestation.

SUGGESTIVE CLINICAL AND US FINDINGS

- **History** of previous **one** or **more** mid-trimester abortion or PTL characterized by a sudden onset, rapid and relatively painless course, with a foetus born with no signs of serious FCA.
- **Signs** in the **2nd trimester:** the cervix may admit tip of finger, with the membranes occasionally protruding through the dilated internal os.
- **US criteria:**
  - Shortened cervical canal < 2.5 cm in length
  - Funneling at the region of the isthmus (Y shaped isthmus)
  - Protrusion of membranes into the cervical canal through the dilated internal os.

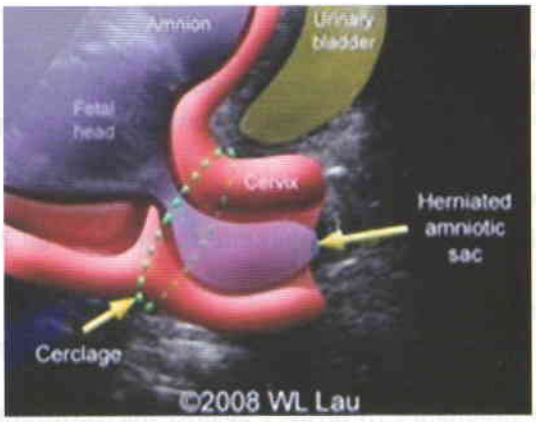


Fig 7-6: Protrusion of membrane



Fig 7-7: Short cervical canal by TVS

CERCLAGE PROCEDURE FOR ISTHMIC INCOMPETENCE

(A) GENERAL MEASURES:

- Physical and mental **rest**, and especially avoid fatigue and exertion.
- Prophylactic **progesterone** support given orally or vaginally on daily doses throughout the 2nd trimester helps in minimizing PTL, hence is also used in cases with RPL, especially those attributed to cervical insufficiency, whether a cerclage procedure will be performed or not.

(B) CERVICAL CERCLAGE PROCEDURES:

- Cervical **cerclage** procedures describe the placement of a circumferential suture into the cervix, encircling the upper cervical canal at the cervico-vaginal junction, with **success** rates approaching **75-85%** in prevention of recurrent mid-trimester abortion and PTL due to incompetent isthmus
- It is generally performed as an **elective prophylactic** procedure, under general or regional anesthesia, at **13-16** weeks gestation in patients with a previous **history** suggestive of an incompetent isthmus. Occasionally it can be performed as an **emergency** procedure at gestational ages up to **24** weeks whenever alerting signs as cervical dilatation, funneling of the isthmus, and intracervical protrusion of the membranes are detected.
- Cerclage suture is **removed** as an office procedure, at **37** weeks, or at onset of **labour** pains to avoid traumatic cervical lacerations, or at the end of a **caesarean** delivery.
- **Vaginal Cerclage procedures:** are the most commonly performed procedures including;

➢ ***McDonald's procedure:*** Four bites of Mersilene tape, or monofilament non absorbable sutures, are taken as high as possible in the cervix externally like a purse string. This is by far the most popular procedure being rapidly and easily performed with excellent results.

➢ ***Shirodkar's procedure:*** Mersilene tape or silk suture is inserted under the vaginal mucosa surrounding the cervix at a level as near as possible to the internal Os. It is more lengthy and associated with more blood loss but a more anatomical procedure. It is mainly indicated when there are deep cervical lacerations or failed Mc Donald's.

- **Abdominal Cerclage Procedure:** In **rare** occasions an abdominal approach for cerclage via laparotomy may be recommended in cases of recurrent abortion with previously failed vaginal cerclage and in cases where vaginal approach is difficult with distorted cervical anatomy. The patient is delivered by **CS > 37 weeks** and cerclage suture left in situ.

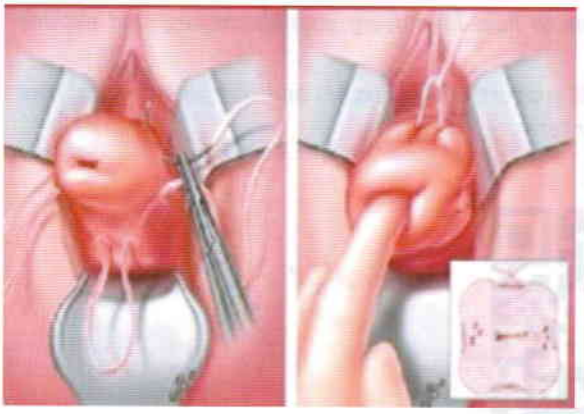


Fig 7-5a: McDonald's cerclage

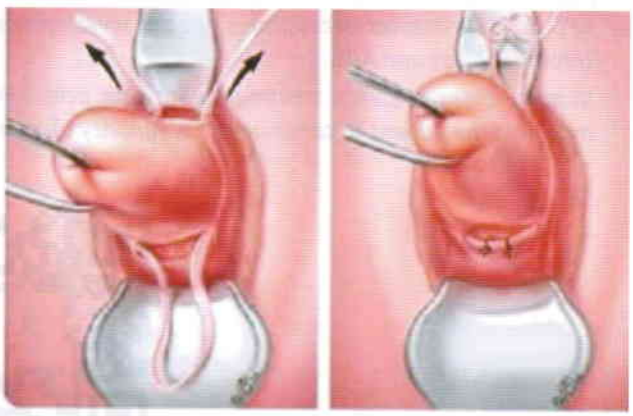


Fig 7-5b: Shirodkar's procedure

GENERAL CONSIDERATIONS IN THE MANAGEMENT OF A CASE OF ABORTION

- An **inevitable abortion** with excessive blood loss and signs of **haemorrhagic shock,** should be managed by immediate **resuscitation** with IV infusions of **crystalloids**, and **ecbolics.** Blood type should be matched for possible **blood transfusion** when necessary.
- An **incomplete abortion** that is **infected**, should be managed vigorously by IV **antibiotics** to avoid overwhelming **sepsis** that may lead to haemorrhage, septicemia, DIC, and rarely death.
- Cases with prolonged 2nd trimester **IUFD** should be carefully monitored for developing **coagulopathy** by repeated measurements of fibrinogen levels, FDPs, PT, PC, and INR. Pregnancy is immediately terminated whenever laboratory evidence points to an impending bleeding tendency.
- Non sensitized **RH-ve** patients should receive an IM prophylactic immunoglobulin **(Rho-GAM).** The recommended dose is a single I.M. injection of anti-D immunoglobulin in a dose of 50 µg up to 12 weeks, and 300 µg thereafter.
- Post abortion **counselling** should be offered to all couples few weeks after a complete abortion, for reassurance, proper history taking, reaching a possible aetiology, and discussing chances of recurrence, and the proposed preventive measures if possible.

Key Points in Abortion

- *10-15% of clinically recognized pregnancies will end in an abortion.*
- *Chromosomal defects are responsible for > 50% of early 1st trimesteric abortions, hormonal and anatomic defects come next.*
- *The clinical types of abortion include; threatened, missed, inevitable, incomplete, and complete abortion*
- *TAS & TVS play a major role in diagnosis of the type of abortion and assess the duration of pregnancy and possible underlying causes*
- *In most cases of threatened abortion pregnancy will successfully continue uninterrupted*
- *In missed abortion pregnancy is terminated either medically or surgically according to pregnancy duration*
- *Inevitable abortion may be accompanied by severe bleeding leading to hypovolemic shock that requires correction and resuscitation before intervention which is usually surgical to control both bleeding and pain and to ensure complete evacuation of all POC.*
- *Incomplete abortion may cause serious sepsis if not adequately managed.*
- *Retained non-viable POC for 4 or more weeks may be associated with hypofibrinogenaemia, DIC, and severe bleeding due consumption coagulopathy. In such cases pregnancy should be terminated whenever any laboratory evidence points to an impending bleeding tendency*
- *Mid-trimester recurrent abortion due to cervical insufficiency is best managed by one of vaginal cerclage procedures, the most commonly used procedure being McDonald's operation.*
- *RPL in 1st trimester necessitates special investigations to help identifying possible causes, and exclude thrombophilias and APS.*
- *In many cases empirical treatment with Folic acid, PRG support, LDA, Clexane, and careful antenatal care may help the patient to continue her pregnancy till near term.*



08 ECTOPIC PREGNANCY
EARLY PREGNANCY COMPLICATIONS

- *Tubal Ectopic Pregnancy*
- *Pregnancy of unknown location*
- *Ovarian Ectopic Pregnancy*
- *Advanced Extrauterine Pregnancy*
- *Cervical Ectopic Pregnancy*
- *Management of ectopic pregnancy*

DEFINITION and OVERVIEW

Ectopic pregnancy is a pregnancy in which the gestational sac **implants** anywhere **outside** the **endometrial** cavity. It is considered one of the common obstetric **emergencies** in early pregnancy, and an important cause for maternal **morbidity**, which may also affect the future female **fertility**.

INCIDENCE:

0.5-2% of all pregnancies may be ectopic, according to associated risk factors present.

SITES OF ECTOPIC GESTATION

1. **THE FALLOPIAN TUBE:** is the most common site **> 95%.** Within the tube **> 70%** will occur in the **ampullary** portion, **12%** in the **isthmic** portion, **11%** in the **fimbrial** portion, and **< 2%** in the **interstitial** portion of the tube.
2. **OTHER RARE SITES**: the **ovary** 3%, the **cervix** 1%, within a **CS scar** <1%, or in a **rudimentary horn**.
3. **ADVANCED INTRAPERITONEAL PREGNANCY:** very rarely the gestational sac may implant and grow within the **intraperitoneal cavity** resulting in an **advanced abdominal pregnancy** < 1%.

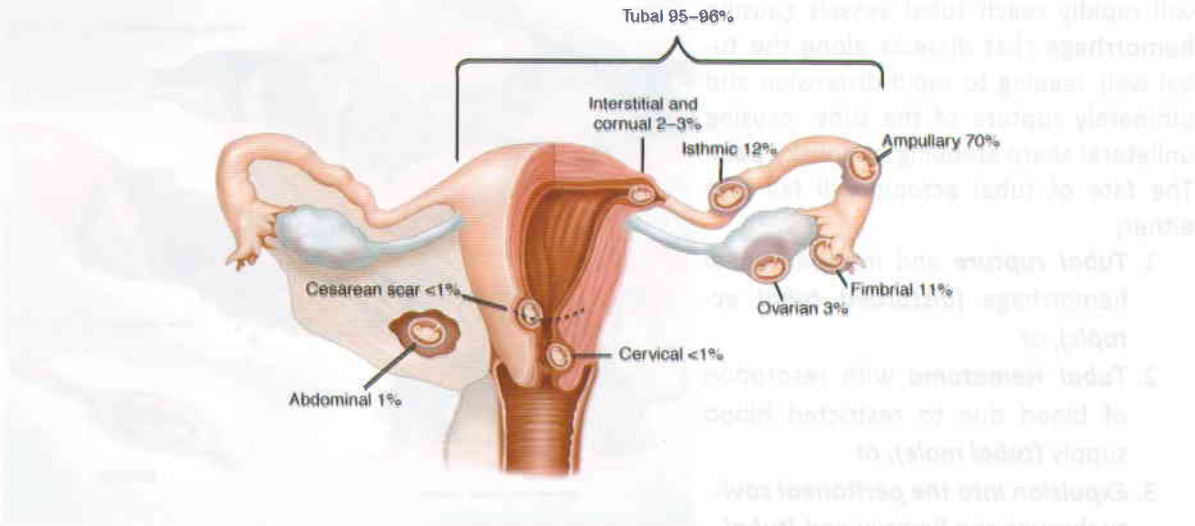


Fig 8-1: Sites of ectopic pregnancy

AETIOLOGY OF TUBAL ECTOPIC PREGNANCHY

- **No identifiable aetiology** can be sited in almost **1/3** of cases of ectopic pregnancy.
- **Tubal stricture;** caused by inflammation, scarring, kinking and adhesions is the **commonest** aetiological factor encountered.
- **Abnormal tubal motility;** secondary to a progesterone effect is a less common cause.

RISK FACTORS FOR TUBAL ECTOPIC PREGNANCY

1. **Previous ectopic gestation:** recurrence rates for an ectopic pregnancy may reach up to **30%.**
2. **Endo-Salpingitis:** inflammation of the tubal mucosa causes destruction and scarring of tubal lumen with strictures associated with partial or complete tubal obstruction.
   - *Gonococcal* or *chlamydial* salpingitis causing severe PID
   - *Puerperal* or *post abortive* salpingitis (following puerperal sepsis or septic abortion)
   - *Postoperative* pelvic peritonitis (following pelvic surgery, rupture appendix, etc...).
3. **Endometriosis:** leading to peritubal adhesions causing tubal kinking and stenosis.
4. **Pelvic and tubal surgery:** leading to tubal scarring or pelvic adhesions
5. **Progesterone effect:** results in alteration of tubal motility thus interfering with oocyte and sperm transfer. PRG effect may be encountered in association with
   - *PRG* only contraceptive pills (POPs)
   - Injectable *Gestagens* and subdermal *Implants*
   - *Levo-norgestrel IUDs (Mirena & Progestasert).*
6. **ICSI** procedure is a known risk factor possibly due to multiple ovulation and high PRG support.
7. **Cu IUD:** prevents intrauterine pregnancy rather than extrauterine. Its presence may alter tubal motility and induces or fulminates PID.

N.B.*: Ectopic pregnancies in sites other than the fallopian tubes do not necessarily share the same risk factors as tubal ectopic pregnancies*

PATHOLOGY AND NATURAL HISTORY OF TUBAL ECTOPIC PREGNANCY

The mucosa of the tubal epithelium cannot resist **trophoblastic invasion** due to absence of a decidual layer. The eroding trophoblasts will rapidly reach tubal vessels causing **hemorrhage** that dissects along the tubal wall, leading to rapid distension and ultimately rupture of the tube, causing unilateral sharp stabbing or colicky **pain.** The fate of tubal ectopic will fall into either;

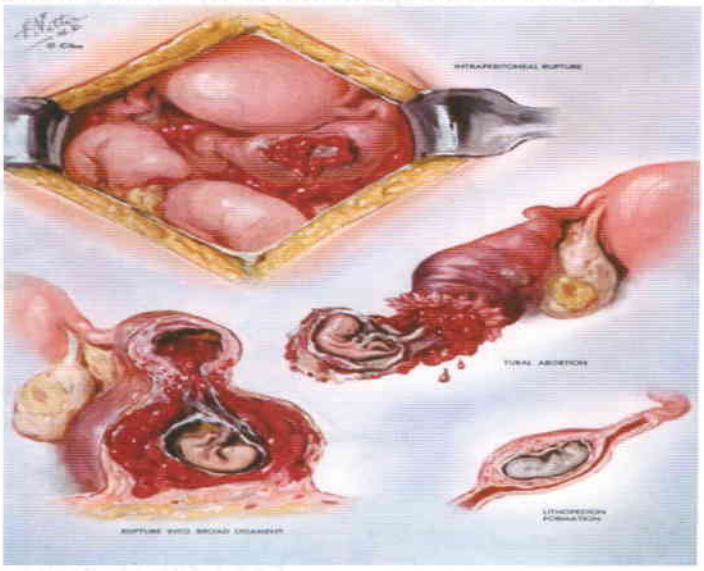


Fig 8-2 Pathology and fate of tubal ectopic gestation

1. ***Tubal rupture*** and intraperitoneal hemorrhage ***(disturbed tubal ectopic)***, or
2. ***Tubal Hematoma*** with resorption of blood due to restricted blood supply ***(tubal mole),*** or
3. ***Expulsion into the peritoneal cavity*** through the fimbrial end ***(tubal abortion)*** or
4. ***Advanced abdominal pregnancy*** where the expelled blastocyst may re-implant within the peritoneal cavity allowing the pregnancy to continue for an undetermined period of time.

During the short period of amenorrhoea in an ectopic pregnancy the following usually occurs

- **Irregular bleeding:** due to irregular shedding of the endometrial lining with lack of normal PRG support, and also due to haemorrhage occurring within the fallopian tube.

➢ **Serum B-HCG** levels usually show initially lower than expected levels, associated with an abnormally slow rise when repeated after 48 ***(abnormal B-hCG doubling time)***.

N.B.; in a normal pregnancy, serum B-hCG levels usually show a steady daily rise almost **doubling** every **55 hours** i.e. an increase by **> 75%** when repeated after **48 hours**. An ***abnormal doubling time*** is one of the hallmarks in suspecting an ectopic gestation.

CLINICAL PRESENTATIONS IN ECTOPIC PREGNANCY

The common finding in all ectopic pregnancies is that symptoms occur in the settings of a **positive** pregnancy test in women in the child bearing period.

The clinical presentation of **tubal ectopic** pregnancies varies from mild lower unilateral abdominal **pain** and minimal vaginal **bleeding** to symptoms and signs of an **acute abdomen** with **hypovolaemic shock** due to marked intraperitoneal haemorrhage. In sites other than a tubal ectopic clinical presentations may vary remarkably.

The diagnosis of ectopic pregnancy requires high index of **suspicion** together with a **combination** of diagnostic tools including pelvic **US,** repeated serum **B-hCG** levels, and diagnostic **laparoscopy**.

1. EARLY UNDISTURBED TUBAL ECTOPIC PREGNANCY

A) SYMPTOMS & SIGNS:

- ***Delayed cycle;*** with a positive pregnancy test.
- ***Unilateral lower abdominal pain;*** dull aching in character, usually not severe.
- ***Mild recurrent vaginal bleeding;*** usually falsely diagnosed as threatened abortion.
- ***General examination*** is normal in haemodynamically stable patients.
- ***Abdominal examination:*** unilateral lower abdominal tenderness.
- ***Vaginal examination:*** marked pain and tenderness on touching or moving the cervix. Adnexal fullness or an adnexal mass can sometimes be felt during bimanual examination.

2. RUPTURED OR DISTURBED TUBAL ECTOPIC

A) SYMPTOMS & SIGNS: in addition to delayed cycle and recurrent bleeding there may be;

- ***Severe lower abdominal pain;*** sharp stabbing or colicky in nature.
- ***Symptoms suggestive of internal haemorrhage;*** as irritability, drowsiness, and sometimes loss of consciousness which is relevant to the amount of internal bleeding.
- ***General signs*** of **intraperitoneal haemorrhage** including tachycardia, hypotension, oliguria, tachypnea, etc.... In cases with massive bleeding picture of haemorrhagic shock
- ***Abdominal examination:*** marked abdominal tenderness, rigidity, and rebound tenderness.
- ***Vaginal examination:*** tenderness on touching or moving the cervix with adnexal fullness

INVESTIGATIONS IN A CASE OF ECTOPIC PREGNANCY

A) SERUM B-HCG:

Quantitative serum **B-hCG** showing pregnancy levels with abnormal doubling when repeated in 48 hours (in a normal pregnancy levels increase steadily showing a 75% rise every 48 hours)

B) ULTRASONOGRAPHY (TAS or TVS):

- ***Absence** of an **intrauterine** gestational sac:* with serum B-HCG levels **> 1500** mIU/ml for TVS & **> 6500** mIU/ml for TAS (B-hCG US discriminatory zone). This is the commonest US finding
- *Presence of a unilateral **adnexal mass**:* haematosalpinx or a tubal haematoma formation.
- *Presence of an **intact extrauterine** gestational sac:* with or without an embryo (very rare)
- *Diffuse intraperitoneal **haemorrhage:*** in cases with disturbed ectopic pregnancies.

C) LAPAROSCOPY:

- When diagnosis is still in **doubt** laparoscopy will offer an excellent chance for;
- **Visualization** of the whole peritoneal cavity and detection of a suspicious adnexal **mass**.
- **Differentiation** between **tubal** and **ovarian** ectopic pregnancies
- Laparoscopy will also offer a unique opportunity for primary **surgical intervention** whenever indicated (see later).

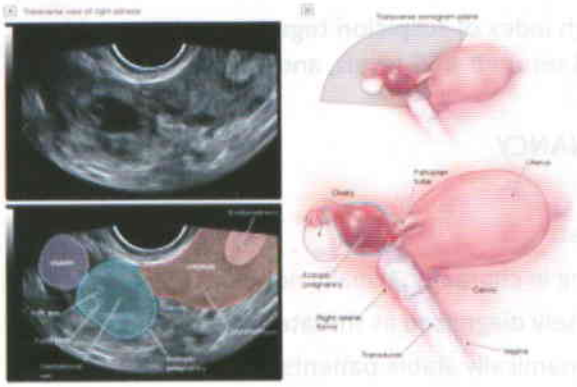


Fig 8-3: TVS right unruptured tubal ectopic pregnancy

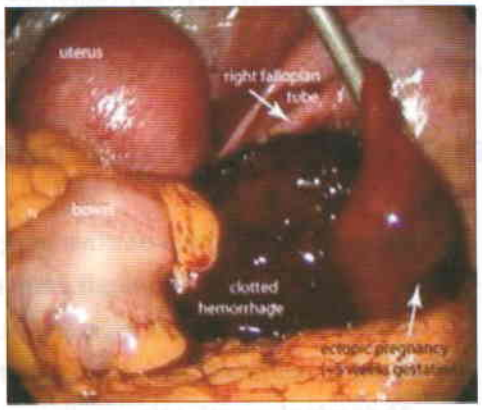


Fig 8-4: laparoscopy right ruptured tubal ectopic

MANAGEMENT OF TUBAL ECTOPIC PREGNANCY

Rarely an early tubal ectopic pregnancy may undergo spontaneous resolution, with initially low serum B-h GG levels which show a steady gradual decline on follow up after 48 hourly, Such cases can be managed **conservatively** (expectant management) without the need for either medical or surgical intervention, until h- CG levels return to non- pregnant levels.

The majority of diagnosed tubal ectopic pregnancies will need either medical or surgical intervention as discussed below.

A) SURGICAL MANAGEMENT

Surgical management of an ectopic pregnancy is indicated in cases with **disturbed** ectopic gestation, in cases with a large adnexal **mass**, or in cases with significant intraperitoneal **haemorrhage**.

Operative **Laparoscopy** is usually the preferable primary approach whenever the skilled team and equipment are available. **Laparotomy** will be reserved to complicated haemodynamically unstable cases with massive haemorrhage, or larger adnexal masses.

1. **SALPINGECTOMY:** the whole fallopian tube is completely excised whenever it is damaged by rupture or severe distension.
2. **SALPINGOSTOMY:** the tube is opened by a linear incision on its antimesenteric border at the

implantation site, the products of conception are removed, and haemostasis established. The tube is left open without closure of the incision. Suturing the incision (**salpingotomy**) adds no benefits and may predispose to future tubal strictures.

N.B.; Any surgical intervention in a patient with haemorrhagic shock will be preceded by immediate **resuscitation** with Saline, crystalloid, and blood transfusion when necessary.

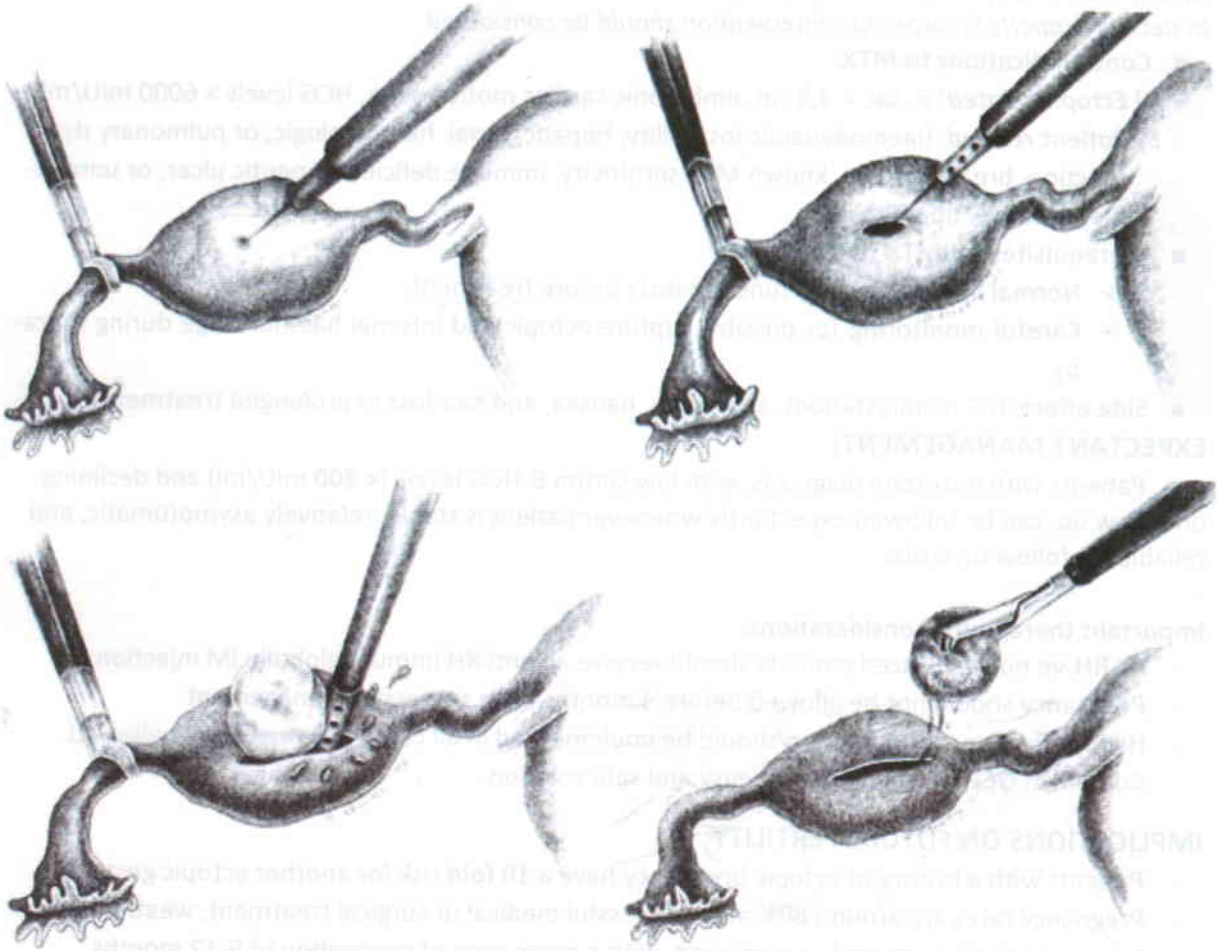


Fig 8-5 Laparoscopic Salpingostomy for undisturbed tubal ectopic pregnancy

MEDICAL MANAGEMENT WITH METHOTREXATE (MTX)

Ambulatory medical management with **MTX** has replaced surgical diagnosis and treatment in **many** cases of early **unruptured** ectopic pregnancies. Randomized trials showed no significant differences in tubal patency, future recurrent ectopic rates, or future fertility between MTX medical treatment and tube sparing laparoscopic surgery (Salpingostomy & Salpingotomy).

- **Indications:** MTX is suitable for early **undisturbed** cases of ectopic gestation with **low** serum B-HCG levels (< 6000 mIU/ml), and a **small** adnexal mass **<3.5** cm, in **absence** of intraperitoneal haemorrhage
- **Mode of action:** MTX is a folic acid antagonist that inhibits **DNA** synthesis and cell replication.
- **Single dose protocol & Follow up regimen:**

- Single IM injection regimen; 1mg/kg body weight.
- Repeat serum B-HCG at days **4 & 7** from 1st injection to insure regression by at least **15%** between return days
- Weekly follow up tests to ensure a constant **15%** decrease in serum B-HCG levels until reaching undetectable levels (< 5 mIU/ml).

N.B.: *if HCG levels fail to decline properly another IM dose of MTX may be given and if levels still fail to decline properly laparoscopic intervention should be considered.*

- **Contraindications to MTX:**

  A) Ectopic related: G. sac > 3.5 cm, embryonic cardiac motion seen, HCG levels > 6000 mIU/ml

  B) Patient related: haemodynamic instability, hepatic, renal, haematologic, or pulmonary dysfunction, breast feeding, known MTX sensitivity, immune deficiency, peptic ulcer, or unreliable for follow up visits.

- **Prerequisites for MTX therapy:**
  - **Normal** liver and kidney function tests before treatment.
  - **Careful monitoring** for possible rupture ectopic and internal haemorrhage during therapy.
- **Side effect:** GIT manifestations, stomatitis, nausea, and hair loss in prolonged treatment

EXPECTANT MANAGEMENT:

Patients with uncertain diagnosis, with low serum B-HCG levels (**< 200** mIU/ml) and declining on follow up, can be followed expectantly whenever patient is stable, relatively asymptomatic, and reliable for follow up visits.

Important therapeutic considerations:

- All **RH-ve** non sensitized patients should receive an anti RH immunoglobulin IM injection
- **Pregnancy** should not be allowed before 3 months from successful management
- Highly effective **contraception** should be implemented in all cases till pregnancy is allowed. Combined **OCP** will give the best easy and safe solution.

IMPLICATIONS ON FUTURE FERTILITY

- Patients with a history of ectopic pregnancy have a **10 fold** risk for another ectopic gestation
- Pregnancy rates are around **80%** after successful medical or surgical treatment, weather the tube is surgically removed or preserved, with a mean time of conception of 9-12 months
- Fertility rates are similar after expectant management, medical, or surgical intervention

DIFFERENTIAL DIAGNOSIS OF ECTOPIC PREGNANCY

1. Other causes of **bleeding** in early pregnancy; as abortion, and molar pregnancies
2. Other causes of **pain** in association with pregnancy;
   a. *Gynaecologic:* torsion or ruptured ovarian cyst, acute PID, or degenerated myoma
   b. Non Gynaecologic causes: acute appendicitis, pyelonephritis, pancreatitis

PREGNANCY OF UNKNOWN LOCATION (PUL)

PUL is the term used to describe a **possible ectopic** pregnancy (abnormal B-HCG doubling and absent intrauterine gestational sac), without reassuring evidence about being extrauterine. The term is used until a **final diagnosis** is made which could be either; a normal intrauterine pregnancy, an abnormal intrauterine pregnancy (abortion), or an ectopic pregnancy.

MANAGEMENT OF UNCOMMON TYPES OF ECTOPIC PREGNANCY

OVARIAN ECTOPIC PREGNANCY

Ovarian ectopic produces similar symptoms and signs to tubal ectopic with slightly less prominent pain. Diagnosis is difficult without laparoscopy which can be used for treatment.

- Aim of treatment is to remove ectopic pregnancy while preserving reasonable ovarian tissue.
- Oophorectomy is sometimes mandatory to prevent or stop severe haemorrhage.
- Early cases will respond to MTX treatment, however early diagnosis is difficult.

CERVICAL ECTOPIC PREGNANCY

Cervical ectopic usually present with severe vaginal bleeding rather than pain. Attempts to remove products of conception from the cervix might fail leading to profuse bleeding. US will show contents filling the cervical canal below internal cervical os with empty endometrial cavity.

- IM injection of MTX and arterial embolization can be used if bleeding is mild and sac is small.
- Trans-cervical aspiration using an oocyte aspiration needle is another alternative
- Hysterectomy is reserved for large cervical ectopic, with life threatening vaginal bleeding

ADVANCED ABDOMINAL PREGNANCY

Pregnancies rarely implant within the abdominal cavity (e.g.; on omentum, bowel, or visceral peritoneum), where it may sometimes proceed to 2nd or rarely third trimester.

- Diagnosis is usually only made at laparotomy where removing the placenta poses a great risk being attached to important vital organs and sometimes major vessels. Any attempt at removal may cause massive internal haemorrhage.
- In most cases it is best to leave the placenta attached allowing for spontaneous resorption with or without adjunctive systemic MTX therapy.

CS SCAR PREGNANCY

The presence of a Gest. Sac within a previous CS scar is being reported with increasing frequency.

- Diagnosis: via TVS that may be aided by MRI to differentiate from intrauterine implantation.
- Systemic MTX, uterine artery embolization, and suction curettage are all treatment options
- In some cases surgical intervention via hysterectomy may be necessary especially if deep invasion of scar has occurred.



Fig 8-6 Diagram for tubal ectopic pregnancy



Fig 8-7 diagram for ovarian ectopic pregnancy



Fig 8-8 US picture of cervical ectopic

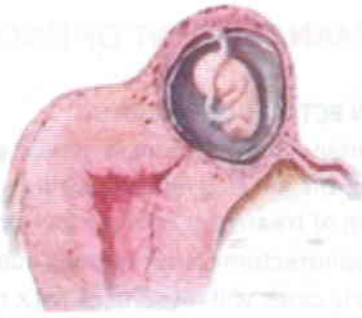


Fig 8-9 Diagram for Rudimentary horn pregnancy

KEY POINT IN ECTOPIC PREGNANCY

- *Tubal ectopic is the commonest site representing > 96% of diagnosed ectopic pregnancies*
- *Some ectopic pregnancies will be never diagnosed ending in a very early tubal abortion or a small tubal mole formation which will be spontaneously absorbed*
- *Diagnosis of ectopic pregnancy needs a high index of suspicion together with the use of a combination of diagnostic tools at the same time*
- *The triad that raises high suspicion includes a short period of cycle delay, together with serum B-hCG levels above the discriminatory zone without US evidence of an intrauterine gestation sac.*
- *Abnormal b-hCG doubling values and presence of an adnexal mass call for starting management.*
- *Management of ectopic presence will vary according to whether it is ruptured or unruptured ectopic gestation, size of the sac, B-hCG level and presence of massive intraperitoneal haemorrhage*
- *Medical treatment by MTX is acceptable in early undisturbed cases with a small sac and low B-hCG levels. It kills dividing trophoblastic tissue and spares the patient surgical intervention.*
- *Laparoscopy is the best modality for cases unsuitable for MTX that will need either salpingostomy if the tube can be preserved or salpingectomy if the tube has been ruptured and damaged.*
- *Follow up after management is mandatory until B-hCG levels completely decline.*
- *Long term sequel of an ectopic pregnancy may have a negative impact on future fertility due to tubal damage, peritubal adhesions, and persistence of the underlying predisposing factors.*
- *Ovarian ectopic and PUL are rarely diagnosed beforehand and usually only diagnosed and managed during laparoscopy or laparotomy*
- *Cervical ectopic can be easily diagnosed only by TVS and should be carefully handled as it may cause serious bleeding that ends in a total hysterectomy as a lifesaving procedure*

INTENDED LEARNING OBJECTIVES (ILOs)

- To know what is an ectopic pregnancy and its common sites (tubal & extratubal)
- To understand the risk factors for an ectopic gestation
- To be aware of its impact of an ectopic pregnancy on maternal health and future fertility
- To understand its various clinical presentations and the tools for proper diagnosis
- To know how to choose between different treatment modalities
- To know how to deal with emergency cases with massive internal haemorrhage
- To understand the aim and extent of surgical and nonsurgical treatments
- To understand the basic principles of the surgical procedures by laparoscopy or laparotomy
- To take a quick glance on diagnosis and management of ectopic pregnancy in sites other than the fallopian tubes
- To be aware of the future hazards of an ectopic pregnancy on female fertility and how to prevent recurrence rates

09 MOLAR PREGNANCY AND GESTATIONAL TROPHOBLASTIC DISEASE

- *Hydatidiform Mole*
  - ➢ *Complete Mole*
  - ➢ *Partial Mole*
- *Gestational Trophoblastic Neoplasia GTN*
  - ➢ *Invasive Mole*
  - ➢ *Choriocarcinoma*
  - ➢ *Placental site Trophoblastic Tumour*
  - ➢ *Epithelioid Trophoblastic Tumour*

GESTATIONAL TROPHOBLASTIC DISEASE (GTD)

Gestational Trophoblastic Disease **(GTD)** is the term used to describe a spectrum of **cellular proliferations** arising from the villous placental **trophoblasts**, including 5 clinico-pathological forms;

- **Hydatidiform Mole** (complete and partial)
- **Invasive Mole**
- **Choriocarcinoma**
- **Placental site Trophoblastic Tumour (PSTT)**
- **Epithelioid Trophoblastic Tumour (ETT)**

The term Gestational Trophoblastic Neoplasia **(GTN)** has been applied collectively to the latter **four** forms which can **progress**, **invade** and **metastasize** and may cause mortalities if untreated.

HYDATIDIFORM MOLE (HDFM)

Molar pregnancies (hydatidiform mole) are **rare** but are the most common type of **GTD** occurring in nearly **1:600** pregnancies in Europe and USA, but may reach up to **1:300** in Caucasian women, and up to **1:125** pregnancies in some Asian countries.

Molar pregnancies represent the **benign** form of **GTD** and are either **complete** or **partial moles**.

RISK FACTORS

- **Age:** increased risk 5-10 times with extremes of age (< 20 years & > 35 years).
- **Dietary factors;** increased risk with low intake of **vitamin A** and **animal fat**
- **Previous molar pregnancy:** the risk of recurrence in a subsequent pregnancy is around **1-2%**



Fig 9-1: Complete HDFM



Fig 9-2: Partial HDFM

PATHOLOGY AND KARYOTYPE

A. COMPLETE MOLE

A complete mole originates from **fertilization** of an **empty ovum** (one with absent or inactivated chromosomes), that will be carrying **paternal DNA only**. This might occur through either;

- **Duplication** of the **DNA** of **one** sperm resulting in a **46 XX** karyotype (90% of cases), **or**
- Fertilization by **2 sperms**, resulting in a **46 XX**, or a **46 XY** karyotype (10% of cases)

- **Gross Morphology:**
  - The uterine cavity will be distended with **grape like** thin-wall **vesicles** that vary in size from few millimetres to a few centimetres in diameter (distended chorionic villi).
  - Hydatidiform changes are **generalized** with **absence** of **amniotic sac** or **foetal tissue**.
- **Histological features** include:
  - 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

A partial mole originates from fertilization of a **normal ovum** by **2 sperms** leading to an embryo with **triploidy**. The resulting karyotype is **69XXX, 69XXY or 69XYY**. Many spontaneous abortions may represent undiagnosed partial moles.

- **Gross Morphology:**
  - Hydatidiform changes are only **focal**, with **presence** of **amniotic sac** or **foetal tissue**.
  - The **placenta** shows large areas with molar changes and variable sized vesicles
  - When a **foetus** is present, it is usually non-viable, and exhibits the stigmata of triploidy including multiple congenital malformations and growth restriction.
- **Histologic Features:**
  - Focal hydropic villi with only focal trophoblastic proliferation.
  - Presence of umbilical cord, amniotic membrane, or foetal tissue

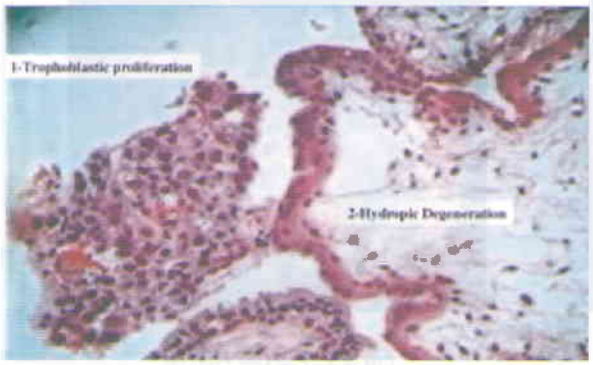


Fig 9-3: Histology of complete mole



Fig 9-4: Histology of partial mole

C. ASSOCIATED OVARIAN THECA LUTEIN CYSTS

Ovarian theca lutein cysts commonly occur in **25-60 %** of **complete moles**, as a form of ovarian hyperstimulation (OHS) in response to the huge amounts of hCG secreted by proliferating trophoblastic cells. They are much less commonly associated with partial moles.

- ***Macroscopically:*** Cysts are usually bilateral, varying in size (few mm up to 10 cm in diameter), with a yellowish colour and a smooth surface.
- ***Microscopically:*** cysts are lined by lutein cells
- ***Fate and complications:*** Most theca lutein cysts will undergo spontaneous regression within 2-4 months of molar evacuation. Larger cysts may be complicated by torsion, infarction and haemorrhage.



Fig 9-5: Theca lutein cysts NE appearance



Fig 9-6: Theca lutein cysts US picture

CLINICAL FEATURES IN MOLAR PREGNANCIES

1. Recurrent early pregnancy bleeding is the chief symptom. Many cases are misdiagnosed as threatened abortion at its first clinical presentation.

2. Other symptoms include;

- Severe ***nausea and vomiting*** in early pregnancy (due to high hCG levels)
- Characteristic ***discharge*** described as dark **prune juice** in colour and nature
- ***Pain*** is usually **absent** or **minimal**, compared to cases of spontaneous abortion, due to:
  - Rapid uterine distension of growing vesicles
  - Separation and haemorrhage in utero with starting expulsion (abortion)
  - Uterine invasion or with complicated theca lutein cysts (torsion and haemorrhage).

3. General Examination:

- ***Pallor*** due to anaemia in relation to the amount of blood loss.
- ***Hypovolemic shock;*** in cases of sudden severe bleeding.
- ***Signs of preeclampsia (PE)*** are rarely encountered in cases reaching the 2nd trimester as hypertension and albuminuria due to abnormal placentation

4. Abdominal Examination:

- *Fundal level:* larger than calculated gestational age due to 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

5. 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

DIAGNOSTIC INVESTIGATIONS IN A MOLAR PREGNANCY

1. Ultrasonography is the **gold standard** in diagnosis of molar pregnancy;

- ***Complete mole:*** shows a characteristic picture of **snow storm** appearance due to presence of vesicles surrounded by blood in absence of foetal echoes or amniotic sac.
- *Ovarian theca lutein cysts* are usually present in association with complete moles
- ***Partial Mole:*** An **embryo** usually in the first trimester, commonly associated with multiple foetal congenital anomalies **(FCA)** and vesicular **hydropic** changes within the **placenta**.

2. Serum β-hCG;

- Abnormally high serum β-hCG levels, reaching >100,000 m IU/ml in the first 8 weeks gestation are usually the hall mark of GTD due to abnormal trophoblastic proliferation.

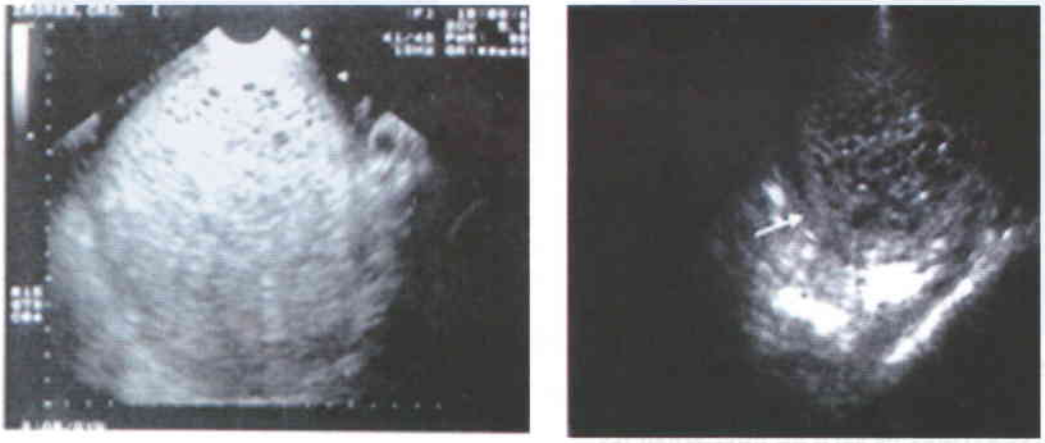


Fig 9-7: US showing snow storm appearance characteristic of a complete HDFM

COMPLICATIONS ASSOCIATED WITH BENIGN HDFM

Severe nausea & vomiting (Hyperemesis gravidarum): due to abnormally elevated hCG levels

1. *Severe bleeding* when starting expulsion which may lead to hypovolemic shock
2. *Early onset pregnancy induced hypertension* (Gestational HTN or preeclampsia PE)
3. *Hyperthyroidism*: in 7% of cases may occur due to thyroid stimulating effect of hCG.
4. *Trophoblastic embolization* to the lungs causing RDS
5. *Complications of ovarian cysts* (torsion, haemorrhage, rupture...)
6. *Risk of developing an invasive Mole* (**20%** in a complete mole, and **4%** in a partial moles)
7. *Risk of developing Choriocarcinoma* (**5%** of cases)

DIAGNOSTIC WORK UP FOR MOLAR PREGNANCIES

Once diagnosis of a molar pregnancy has been established by **US** a metastatic work up should be immediately implemented to exclude GTN before attempts to evacuate the mole.

1. **β-hCG** levels to asses risk for malignant disease (GTN) and as a baseline for follow up after molar evacuation (according to post-molar regression curves)
2. **Exclude metastases** by chest X-ray, abdominal US, and MRI for the brain
3. **Prepare** for immediate **evacuation** (CBC, KFT, LFT, TSH, T3 & T4, and coagulation profile)

MANAGEMENT OF COMPLETE AND PARTIAL MOLES

A. SUCTION EVACUATION CURETTAGE:

- This is the **gold standard technique** for evacuating molar pregnancies
- Performed by a **suction evacuation apparatus** with cannulas introduced through the cervix
- **Vaginal PGL E1** (Misoprostol) may be used to ripen the cervix for easier dilatation
- **IV Ecbolics** are used to minimize blood loss and enhance uterine contractility.
- **Gentle curettage** of the uterine cavity is performed after suction to ensure complete molar evacuation with no residual trophoblastic tissue or hydropic villi.

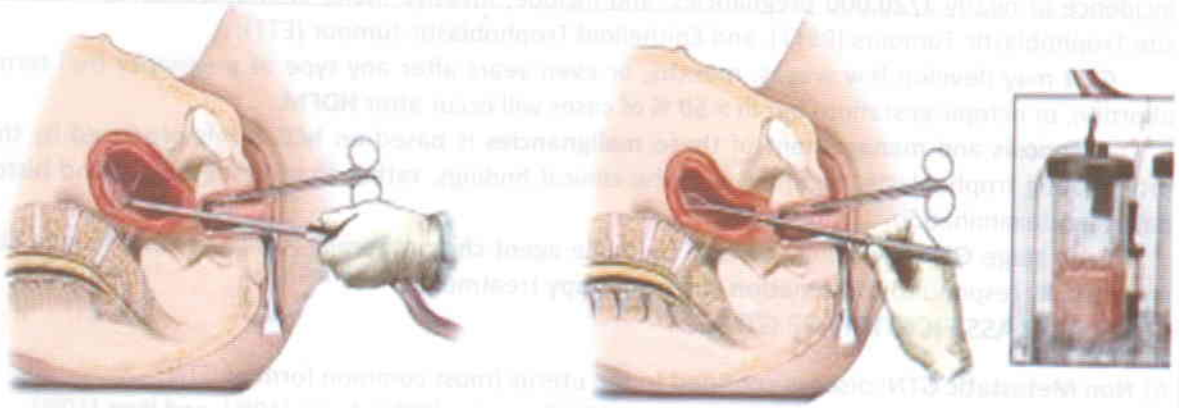


Fig 9-8: Suction evacuation and curettage

HYSTERECTOMY:

Abdominal hysterectomy has a limited place in the management of HDFM only in elderly patients with severe bleeding, infection, invasion, or high risk for GTN. It may be also indicated in presence of associated pathology as multiple myomata or adenomyosis in a multiparous woman.

FOLLOW UP AFTER EVACUATION OF MOLAR PREGNANCIES

- **Repeated serum B-hCG** assays made at 48 hours post-evacuation, then weekly until results are negative for 3 successive weeks, then monthly for 6 months.
- **Physical examination** and **pelvic US** at regular monthly intervals; to ensure complete evacuation, normal involution of pelvic organs, and disappearance of theca lutein cysts.
- **Contraception for 1 year** after evacuation of a HDFM is highly recommended and best achieved by hormonal contraception (COCs, POPs, Injectable contraceptives, or subdermal implants).

PERSISTENT TROPHOBLASTIC DISEASE (PTD)

PTD is the term used to describe cases with incomplete evacuation of a molar pregnancy, showing persistently higher than expected **β-hCG** levels (abnormal post-molar regression curves).

Treatment is usually successfully achieved via Methotrexate chemotherapy. In some cases with significant residual molar tissue in the uterus as detected by US, a repeat curettage will be necessary.

FUTURE PREGNANCIES AND RISK OF RECURRENCE

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

RISK FACTORS FOR DEVELOPING MALIGNANT GTD (GTN)

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

GESTATIONAL TROPHOBLASTIC NEOPLASIA (GTN)

GTN is the **malignant** form of **GTD**, characterized by their aggressive invasion into the myometrium, and their tendency to metastasize. They are rarely occurring trophoblastic tumours with an incidence of nearly **1/20.000** pregnancies, and include; Invasive mole, Choriocarcinoma, Placental site Trophoblastic Tumours (PSTT), and Epithelioid Trophoblastic tumour (ETT).

GTN may develop few weeks, months, or even years after any type of pregnancy (full term, abortion, or ectopic gestation) but in **> 50 %** of cases will occur after **HDFM**.

Diagnosis and **management** of these **malignancies** is based on **hCG** levels produced by the proliferating trophoblasts, together with the clinical findings, rather than tissue biopsy and histopathologic examination.

Early stage **GTN**, are highly **curable** by single-agent **chemotherapy**, while advanced stage disease usually respond to **combination chemotherapy** treatment.

CLINICAL CLASSIFICATION OF GTN

A) **Non Metastatic GTN:** disease **confined** to the uterus (most common form of GTN)
B) **Metastatic GTN:** disease **spread** to the lung (80%), vagina (30%), brain (10%), and liver (10%).
Metastatic GTN are further divided into **low** risk and **high** risk cases according to;

Low Risk Metastatic GTN	High Risk Metastatic GTN
Occurs < 4 months after a previous pregnancy	*Occurs > 4 months after a previous pregnancy*
No previous chemotherapy treatment	*Previous failed chemotherapy treatment*
No spread to brain or liver	*Spread to brain or liver*
Good prognosis	*Poor prognosis*

HISTOPATHOLOGICAL CLASSIFICATION

A. INVASIVE MOLE:

- These tumours have a tendency for **deep myometrial penetration** yet with a **limited** ability for metastases (more like locally malignant tumours).
- They can occur after complete moles (20%), or partial moles (4%).
- **Grossly:** deep myometrial invasion with focal areas of necrosis and haemorrhage
- **Microscopically:** deep myometrial penetration by trophoblastic cells and whole villi.

B. GESTATIONAL CHORIOCARCINOMA:

- A highly **malignant** trophoblastic tumour usually associated with **early** blood borne **metastases**, mostly to the lungs and the vagina.
- 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** tumour arising from placental site **intermediate** trophoblast
- It usually occurs following a **term** pregnancy, and produces moderate amounts of **B-hCG**.
- It is a **locally invasive** tumour that is generally **resistant** to chemotherapy.
- **Hysterectomy** is a good option for management of such tumours

D. EPITHELIOID TROPHOBLASTIC TUMOUR:

- This **rare** tumour develops from chorionic type **intermediate** trophoblast
- It is generally resistant to chemotherapy that is why better managed by hysterectomy
- Adjuvant chemotherapy may be offered to high risk cases.

FIGO	ANATOMICAL STAGING
I	*Disease confined to the **uterus***
II	*Outside uterus but limited to **genital tract** structures (vagina, ovaries & adnexa)*
III	*Extends to the **lungs**, with or without known genital tract involvement*
IV	***All** other metastatic sites*

DIAGNOSIS OF GTN

1. Persistent **vaginal bleeding** with abnormal **high hCG** levels that either rise, plateau, or fail to reach a nadir **3** weeks after a recently terminated pregnancy (mostly HDFM or abortion).
2. **Clinical Examination:**
   - Normal or slightly sub-involuted uterus
   - Theca lutein cysts of the ovary may be felt as bilateral cystic adnexal masses
3. **TVS/TAS:**
   - May or may not show evidence of retained products of conception.
   - Theca lutein cysts if present are accurately detected by US
4. **MRI** is useful in evaluation myometrial **invasion** if present
5. **CT** scan, Chest **X-ray**, and abdominal **US**: are all useful in identifying non metastatic from metastatic cases, and in the latter low risk from high risk GTN.

MANAGEMENT OF GTN

A. Non Metastatic and Low Risk Metastatic GTN

➢ **Methotrexate (MTX):**

- MTX is the drug of choice as a **single agent** chemotherapy with success rates >90%
- It is an anti-folic acid that acts by arresting synthesis of DNA and RNA.
- *Dosage:* 50 mg/m^2 given as repeated IM injections
- *Side effects;* nausea, oral and gastric ulcers, myelo-suppression, 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 is avoided as it interferes with MTX action
- *Follow up protocol* by **weekly** serial hCG levels until negative then **monthly** for 6 months

➢ **Actinomycin D (Act-D): 2nd drug of choice**

This is the second drug choice for non-metastatic and low risk cases of GTN. It is an antibiotic that acts on DNA strands and can be used as an alternative to MTX.

B. Metastatic and High risk Non metastatic GTN

➢ **Combination Chemotherapy:**

- Indicated in the rare cases of **MTX failure**, and in **high** risk **metastatic** cases.
- High cure rates are usually achieved however side effects and toxicity are higher than single agent chemotherapy

➢ **Hysterectomy:**

- May be offered to **elderly** multiparous patients with no desire for future fertility
- It is indicated in cases **resistant** to chemotherapy (as epithelioid trophoblastic tumours and placental site trophoblastic tumours).
- It can be also offered to the rare cases with **recurrence** after chemotherapy **(< 1%)**
- Adjuvant combined chemotherapy may still be necessary in the postoperative period.

SUBSEQUENT FERTILITY AND PREGNANCY

- Women with prior GTD or successfully treated GTN usually do not have impaired fertility
- Their pregnancy outcome is usually normal
- Recurrence risk approaches 2% for GTD
- Hormonal contraception 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 at 6 weeks postpartum

PROGNOSTIC SCORING SYSTEM (modified by FIGO)

The WHO adopted a scoring system for **prognosis** in cases of GTN based on variable factors including: patient's age, type of antecedent pregnancy, interval months from the index pregnancy, pretreatment serum hCG, largest tumour size, site of metastases, number of metastases, and previous failed chemotherapy (single agent or combination drugs). The lower the score the better will be the prognosis and the cure rates.

Key Points

HYDATIDIFORM MOLE

- ***HDFM** is the **benign** form of **GTD,** and are either complete or partial*
- ***Complete HDFM** are the commonest encountered form and are characterized by abnormal karyotypes (46XX, or 46XY) with all DNA being paternal in origin due to fertilization of an empty ovum.*
- *Most cases present as threatened abortion until diagnosed both by US & hCG levels*
- *They hace a characteristic US snow storm appearance in association of markedly elevated hCG levels*
- ***Management of HDFM** is via immediate termination of pregnancy by Suction Evacuation.*
- ***Follow-up** by serial weekly hCG levels for 3 months then monthly levels for 6 months.*
- *A new pregnancy Is not allowed before 6-9 months from a negative hCG titre*
- *Recurrence risk is 2%*

GTN

- ***Malignant forms of GTD are known as GTN.** They include invasive mole, choriocarcinoma, placental site trophoblastic disease, and epithelioid trophoblastic tumour.*
- *They can occur after any pregnancy event but most commonly after HDFM*
- ***Persistent vaginal bleeding** after molar evacuation with high B-hCG titre or a plateau for 3 months after molar evacuation is the main presentation for malignant GTN.*
- *Once suspected a **metastatic work** up is initiated including serum hCG, US, blood chemistry, radiography, chest X ray, brain CT or MRI.*
- *At the end of such work up diagnosis will be established, and cases further classified into non-metastatic and metastatic, low risk and high risk cases.*
- ***Non metastatic GTN** are successfully treated with **single agent** chemotherapy usually MTX.*
- ***Metastatic low risk GTN** 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 rates > 80%*
- ***Hysterectomy** has a limited role in management of GTN. It may be useful in elderly multiparous women who do not require further child bearing especially those with tumours resistant to chemotherapy, or whenever the myometrium is destroyed by excessive invasion.*

10 ANATOMY OF THE FEMALE PELVIS AND FOETAL SKULL

- ***Anatomy of the female pelvis***
- *Inlet , Cavity, Outlet*
- *Variations in the shape of a female pelvis*
- ***Anatomy of the Foetal skull***
- *Diameters of the Foetal skull*
- *Obstetrical Definitions*

ANATOMY OF THE FEMALE PELVIS

The female pelvis is formed of two innominate bones, the sacrum, and the coccyx

- The **innominate bones** are joined together anteriorly at the symphysis pubis **(SP)** and are attached posteriorly each to one side of the sacrum at the right and left sacro-iliac joints.
- The **coccyx** is joined with the sacrum above at the sacro-coccygeal joint.
- The **pelvic brim** is an imaginary line separating the pelvic cavity into the **false** pelvis above, and the **true** pelvis below. The false pelvis has no obstetric importance.
- **The true pelvis:** is further divided into an **inlet**, a **cavity** and an **outlet**.

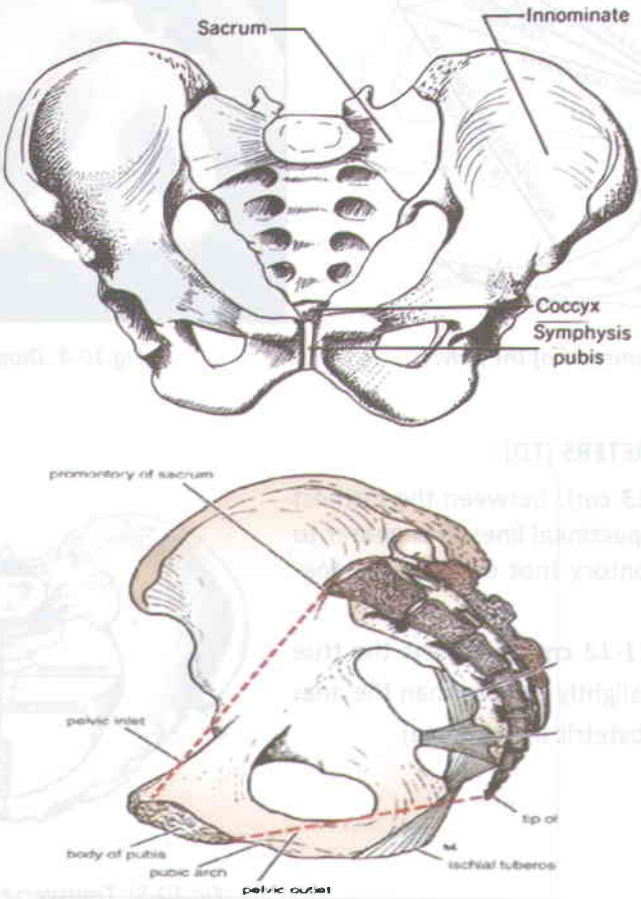


Fig 10-1,2: Bones of female pelvis and planes of pelvic inlet and outlet

THE PELVIC INLET (Brim)

The inlet is **oval** in shape with largest diameters being the transverse and oblique diameters. Its plane makes an **inclination** angle 55° with the horizon in the standing position.

Boundaries of the Pelvic Inlet:

- **Posteriorly:** the promontory of the sacrum, the alae of the sacrum and the sacro-iliac joint
- **On each side:** the iliopectineal line of the innominate bone, the iliopectineal eminence and the upper border of the superior pubic ramus.
- **Anteriorly:** the pubic tubercle, upper border of the pubic bone and upper border of the SP

A) ANTERO-POSTERIOR DIAMETERS (APD):

- *The Anatomical APD or the True Conjugate* ***(11 cm)****:* from the tip of the sacral promontory to the upper border of the symphysis pubis (SP).
- *The Obstetric conjugate* ***(10.5 cm)****:* from the sacral promontory to the most bulging point on the back of the SP. It is 0.5 cm shorter than the true anatomical conjugate
- *The Diagonal conjugate:* ***(12.5 cm):*** from the sacral promontory to the lower border of the symphysis pubis. It is the diameter that can be measured clinically by PV examination.

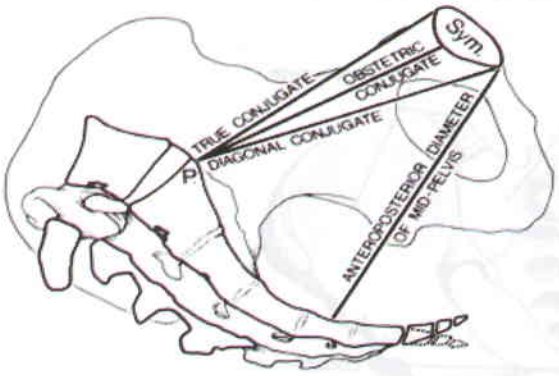


Fig 10-3: AP diameters of the pelvis

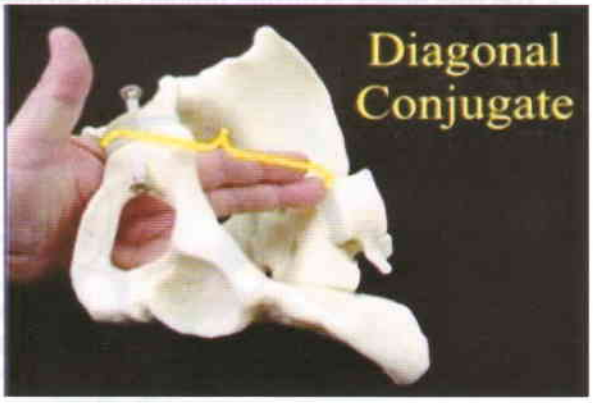


Fig 10-4: Diagonal conjugate

B) TRANSVERSE DIAMETERS (TD):

- *Anatomical TD* ***(13 cm):*** between the farthest points on the iliopectineal lines. It is nearer to the sacral promontory (not used by the foetus).
- *Obstetrical TD* ***(11-12 cm):*** it bisects the true conjugate and is slightly shorter than the anatomical (It is of obstetric importance).

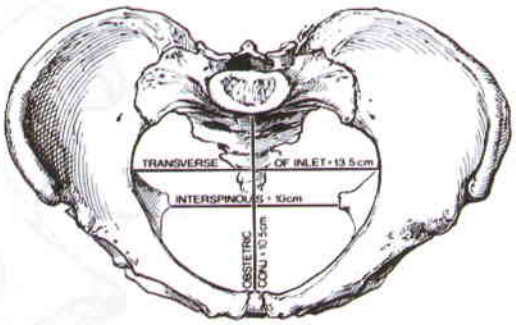


Fig 10-5: Transverse Diameters of the inlet

C) OBLIQUE DIAMETERS OF THE INLET (OD):

- ***Right Oblique Diameter (ROD) = 12 cm:*** from **right** sacroiliac joint to **left** iliopectineal eminence.
- ***Left Oblique Diameters (LOD) = 12 cm:*** from **left** sacroiliac joint to **right** iliopectineal eminence. It is slightly shorter than ROD being encroached upon by the descending pelvic colon.

N.B.: The ROD and LOD are the usual diameters for engagement.

- ***Sacro-Cotyloid Diameter SCD = 9-9.5 cm:*** extends from the promontory of the sacrum to the right and left iliopectineal eminence. The right SCD ends at the right eminence and vice versa.

THE PELVIC CAVITY

The pelvic cavity is an **imaginary segment** bounded **above** by the pelvic brim, **below** by the plane of least pelvic dimensions, **anteriorly** by the symphysis pubis and **posteriorly** by the sacrum.

- The plane of the pelvic cavity lies in its widest part that extends from the centre of the back of the symphysis pubis to the junction between the 2nd and 3rd pieces of sacrum.
- It is rounded in shape with all diameters equal 12.5 cm.
- It has a short anterior wall (symphysis pubis) and a long posterior wall (sacrum).
- The posterior wall (sacrum) is concave and this serves to increase the capacity of the pelvis.
- Internal rotation of foetal head occurs when the BPD lies in this plane.

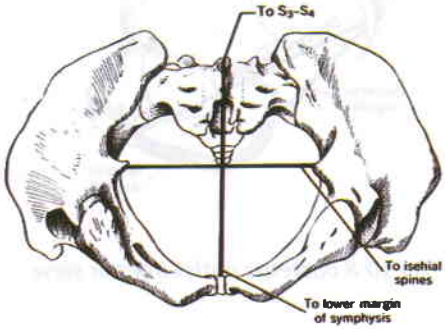


Fig 10-6: The pelvic cavity

THE PELVIC OUTLET

The pelvic outlet is a lozenge shaped area made of **2 triangles** (the anterior and posterior triangles), in **2 planes** (the anterior and posterior sagittal planes)

- **Anatomical outlet:** is a **lozenge** shaped area the boundaries of which are;
  - *Anteriorly:* The lower border of the SP.
  - *Laterally:* The pubic arch, ischial tuberosities, the sacrotuberous & sacrospinous ligaments.
  - *Posteriorly:* The tip of the coccyx.
- **Obstetrical outlet:** is a **wedge** shaped segment the boundaries of which are;
  - *Above (the roof):* the plane of the least pelvic dimensions
  - *Below (the floor):* the anatomical outlet
  - *Laterally:* the part of the ischium between the ischial spine and the ischial tuberosity
  - *Anteriorly:* the lower border of the SP
  - *Posteriorly:* the coccyx. N.B.: the coccyx moved backwards during 2nd stage of labour.

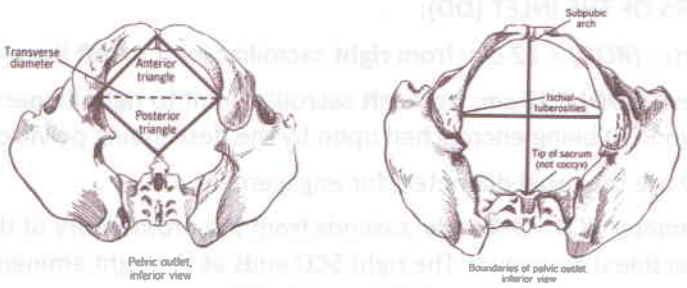


Fig 10-7: Pelvic outlet Inferior view

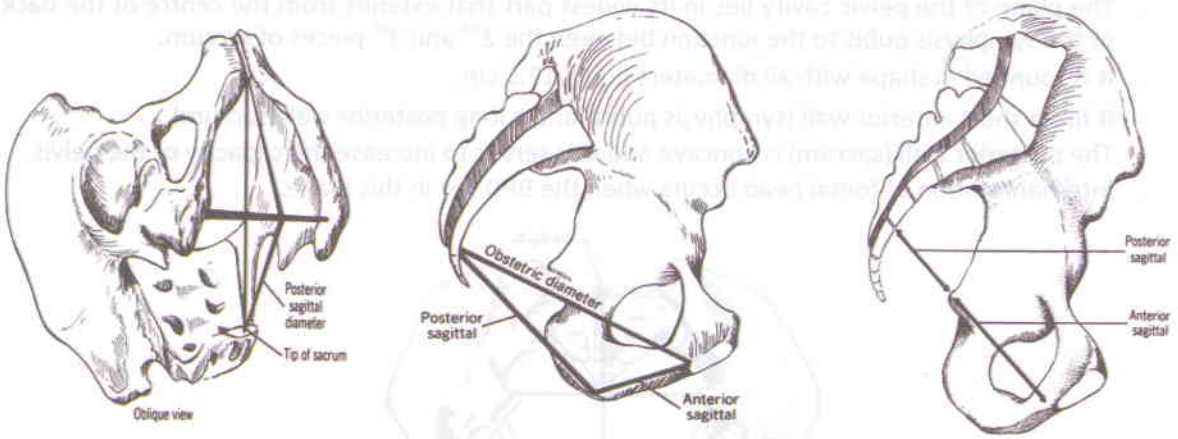


Fig 10-8 obstetric outlet Inferior view

- **Transverse Diameters:**
  - *Bituberous diameter:* ***11 cm,*** between the inner aspects of the 2 ischial **tuberosities.**
  - *Bispinous diameter:* ***10-10.5*** *cm,* between the tips of the 2 ischial **spines.**
- **Anteroposterior Diameters:**
  - *Anatomical APD:* ***11 cm,*** from the lower border of the **SP** to the tip of the **coccyx**
  - *Obstetric APD:* ***13 cm,*** from the lower border of the **SP** to the tip of the **sacrum**
- **Longitudinal Diameters:**
  - *Anterior Sagittal Diameter:* ***6-7 cm,*** from the lower border of the symphysis pubis to the centre of the Bituberous diameter
  - *Posterior Sagittal Diameter:* ***7.5-10 cm,*** from the tip of the sacrum to the centre of the Bituberous diameter.

PLANES OF THE PELVIS

A) PLANE OF THE INLET

- Passes with the boundaries of the pelvic brim making an angle of 55◦ with the horizon (pelvic inclination angle)

B) PLANE OF THE MIDCAVITY (Plane of the greatest pelvic dimensions)

- Passes from the middle of the posterior surface of the SP anteriorly to the junction of the 2nd and 3rd sacral vertebrae posteriorly crossing laterally through the centre of the acetabulum and the upper part of the greater sciatic notch.
- It is a **rounded** plane with a diameter of **12.5** cm.
- Internal rotation of the head occurs when the BPD occupies this wide plane, while the occiput will be touching the pelvic floor at the level of the ischial spines at the plane of least pelvic dimensions.

C) PLANE OF THE OBSTETRIC OUTLET (plane of the least pelvic dimensions)

- Passes from the lower border of the SP anteriorly, to the ischial spines laterally, to the tip of the sacrum posteriorly.

D) PLANE OF THE ANATOMICAL OUTLET

- Passes with the boundaries of the anatomical outlet and consists of 2 triangular planes with a common bases which is the bituberous diameter.
  - *Anterior sagittal plane:* its apex at the lower border of the SP
  - *Posterior sagittal plane:* its apex at the tip of the coccyx

THE SUBPUBIC ANGLE

It is normally between 90° and 100° while in the male it may be reduced to 70°

NB. A narrow subpubic angle will get the head pushed posterior necessitating generous episiotomy

IMPORTANCE OF THE LEVEL OF THE ISCHIAL SPINES:

- It is the level of the levators ani muscle, which is attached to the ischial spines.
- It is the level at which lies the external os of the cervix and the vaginal vault.
- 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 the level of the ischial spines.
- Forceps should not be applied when the foetal head is above this level.
- Anaesthetic agent for pudendal nerve block is injected at this level.

THE OBSTETRIC AXIS

- It is an imaginary line representing the way passed by the head during labour
- It is J-shaped passing *downwards* and *backwards* along the axis of the inlet, till the level of the ischial spines, where it passes *downwards* and *forwards* along the axis of the pelvic outlet.

VARIATIONS IN THE SHAPE OF FEMALE PELVIS

Caldwell – Moloy Classification 1933

The female pelvis is usually gynaecoid in shape, however in many occasions it may have some features of android and or anthropoid pelvis encountered in the male type of pelvis. This may have a clinical impact on the foetal presentation and position and may affect the mechanism of labour.

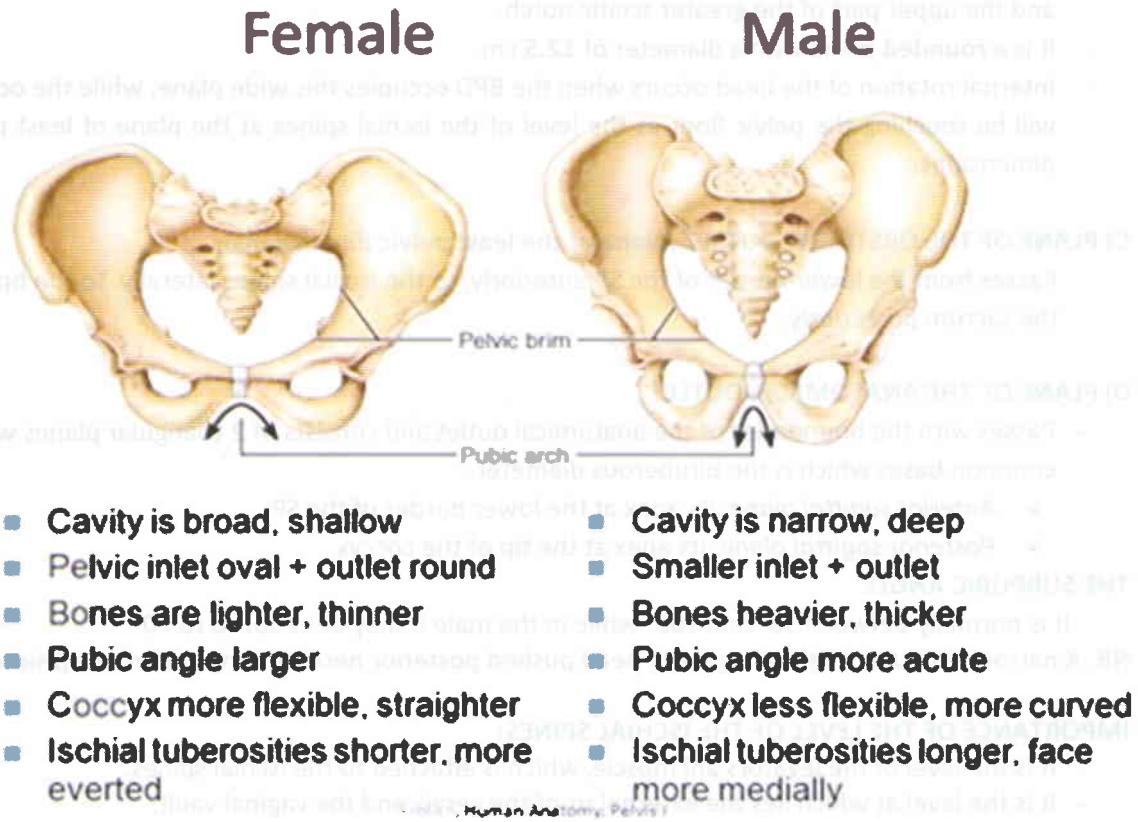


Fig 10-9: Difference between male and female pelvis inlet

ANATOMY OF THE FOETAL SKULL

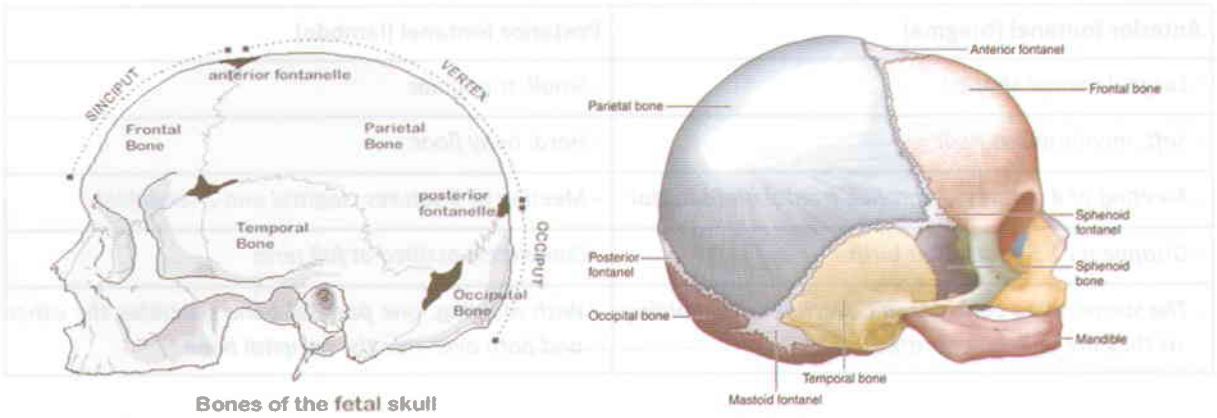


Fig 10-10: Different parts of foetal skull

The foetal skull consists of 3 parts separated by sutures and fontanels.

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.

A) Brow: from the root of the nose and supraorbital margins to the anterior fontanel (bregma) and coronal sutures. It includes the 2 frontal bones separated by the frontal suture.

B) Vertex: from the bregma and coronal sutures to the posterior fontanel and lambdoid suture. It consists of 2 parietal bones separated by the sagittal suture. It is bounded laterally by the parietal eminences.

C) Occiput: from the posterior fontanel and lambdoid suture to the foramen magnum.

- **Sutures:** Frontal, sagittal, coronal, lambdoid, and temporal sutures.
- **Fontanels:** They are **6** in number present at points of crossing of the sutures. The anterior and the posterior fontanelles are the only 2 of clinical importance, in diagnosis of vertex presentation, position of the occiput, and degree of flexion of foetal head

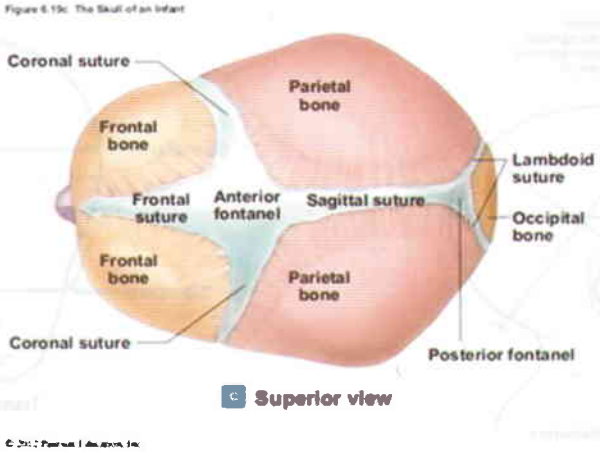


Fig 10-11: Foetal skull, bones, sutures & fontanels

COMPARISON BETWEEN THE ANTERIOR AND POSTERIOR FONTANELS AT TERM

Anterior fontanel (bregma)	Posterior fontanel (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 doesn't override with molding as they are widely separated.	*- With molding, one parietal bone overrides the other and both over-ride the occipital bone*

DIAMETERS OF THE FOETAL SKULL

A. Longitudinal Diameters (LD). The Engaging Diameters:

1. ***Suboccipito-bregmatic (SOB) "9.5 cm":*** from below the occipital protuberance to the centre of the anterior fontanel (bregma). *The engaging diameter when the head is fully flexed*
2. ***Suboccipito-frontal (SOF) "10 cm":*** from below the occipital protuberance to the anterior end of the anterior fontanel. It is the diameter that extends the vulva in vaginal delivery.
3. ***Occipito-frontal (OF) "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 (SMB) "9.5 cm":*** from the junction of the chin and neck to the centre of the bregma. *The engaging diameter when the head is fully extended (Face).*
5. ***Submento-vertical (SMV) "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 fontanels)
6. ***Mento-vertical (MV) "13.5 cm":*** from the tip of the chin to the vertical point. It is the engaging diameter when the head is incompletely extended (brow presentation). It is larger than any pelvic diameter hence engagement will not normally occur in an average sized head

B. Transverse Diameters (TVD):

1. ***Biparietal diameter (BPD) "9.5 cm":*** Between 2 parietal eminences (*widest transverse diameter)*
2. ***Bitemporal diameter (BTD) "8":*** Between the anterior ends of the temporal sutures.

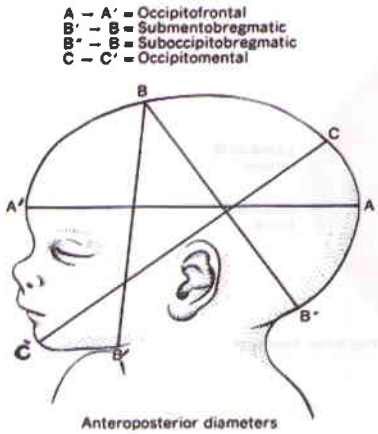
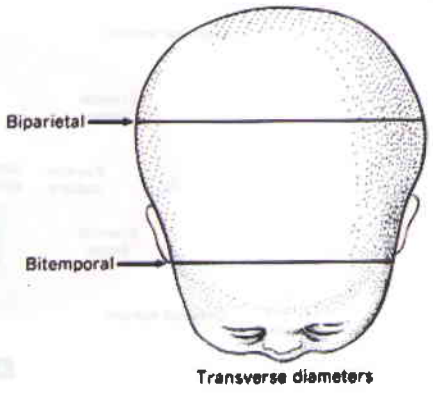


Fig 10-12: LD of foetal skull



10-13: TVD of foetal skull

OBSTETRICAL DEFINITIONS

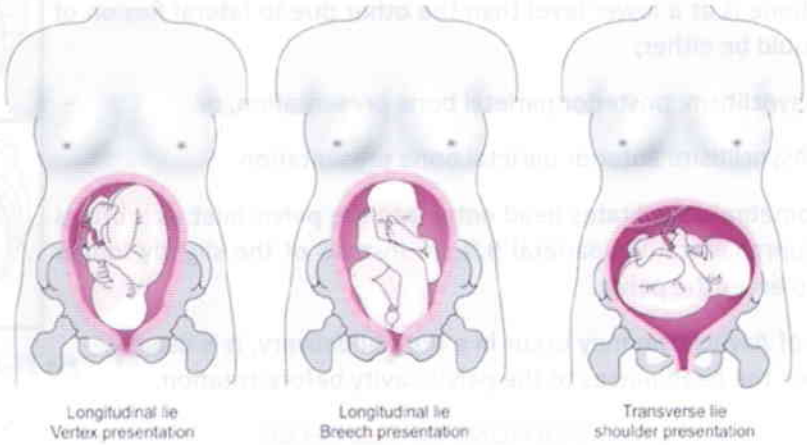


Fig 10-14: foetal lie & Presentation

- **FOETAL PRESENTATION:** It is the lowest part of the foetus related to the pelvic brim and first felt by vaginal examination. It may be cephalic, breech, or shoulder presentation:
  - **A) Cephalic Presentation:** The most commonly encountered presentation as it easily allows for better adaptation of the foetus to the pyriform shape of the uterus with the broader buttocks and thighs lying at the wider fundus. Cephalic presentations include;
    - ***Vertex (95%)***: the commonest in full term pregnancies at the onset of labour
    - ***Face (0.5%)*** *and* ***Brow (0.1%)***.
  - **B) Breech (3.5%):** The **buttocks** with or without the limbs form the presenting part.
  - **C) Shoulder (0.5%):** The **scapula** with or without the arms form the presenting part.
- **MALPRESENTATION:** It is any presentation other than the vertex including face, brow, breech, shoulder, cord and complex presentation.
- **FOETAL LIE:** The relation between the longitudinal axis of the foetus to that of the mother. Foetal lie may be either **longitudinal** or **transverse**.
- **FOETAL ATTITUDE:** The relation of the foetal parts to each other (flexion or extension attitude)
- **FOETAL POSITION:** It is the relation of the back of the foetus to the **right** or **left** side of the mother and whether it is directed **anteriorly** or **posteriorly**.
- **FOETAL DENOMINATOR:** A landmark on the presenting part used to denote the position either anterior or posterior, whether to the right or to the left in relation to the spine of the mother.
  - The **Occiput** in vertex presentation, the **Mentum** (chin) in face presentation
  - The **Sacrum** in breech presentation, and the **Scapula** in shoulder presentation

NOTES OF CLINICAL IMPORTANCE

- Occipito anterior positions **OAP** are more common than Occipito posterior positions **OPP** because the concavity of the front of the foetus fits into the convexity of the vertebral column (Lumbar lordosis) of the back of the mother.
- The right oblique diameter of the inlet is slightly longer than the left oblique (due to encroachment of the descending pelvic colon on the latter), this results in more prevalence of LOA over ROA, and ROP over LOP.
- OP positions are considered a malposition not a malpresentation.

ASYNCLITISM:

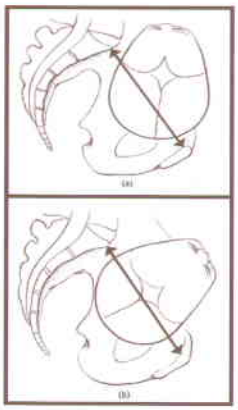


Fig 14-16: Asynclitism

- One parietal bone is at a lower level than the other due to lateral flexion of the head. It could be either;
  - **Anterior** Asynclitism: posterior parietal bone presentation, **or**
  - **Posterior** Asynclitism: anterior parietal bone presentation
- Asynclitism sometimes facilitates head entry into the pelvic inlet as it brings the shorter Supraparietal Subparietal **9.0** cm instead of the slightly longer BPD **9.5** cm, to enter the pelvis.
- Slight degree of Asynclitism may occur in a normal delivery, it is usually corrected when the head passes to the pelvic cavity before rotation.

STATIONS OF THE PELVIS

- Station -3: describes a head with the **BPD** above the pelvic **brim**
- Station -2: the head starts to enter the pelvic inlet with **BPD** behind **SP**
- Station -1: the head is deeper in the pelvic cavity but **not** yet engaged
- **Station 0**: The head is **engaged,** with the **BPD** passing the plane of the pelvic inlet and the **vault** felt on PV examination **at** the **ischial spines.**
- **Station +1:** The BPD has below plane of the inlet and the vault felt by PV examination **below** the level of both ischial spines
- **Station +2:** The head deep in the pelvis resting on pelvic floor muscles
- **Station +3:** the head distending the perineum at the introitus

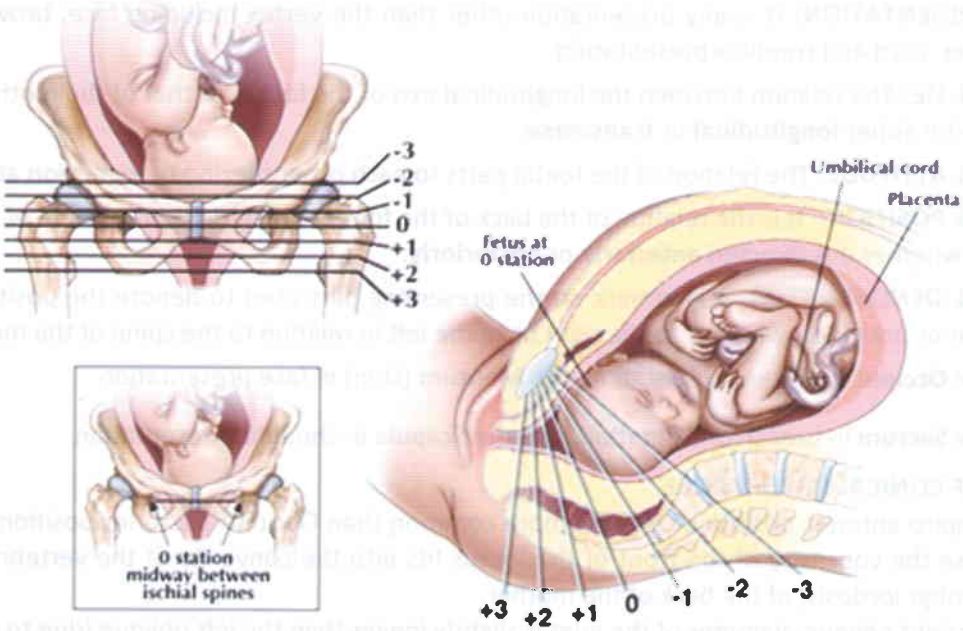


Fig 14-17: Stations of head descent

ENGAGEMENT

Engagement describes passage of the widest **transverse** diameter of the presenting part through the plane of the pelvic inlet (the **BPD** in case of the head and the bitrochanteric **BTD** in case of breech).

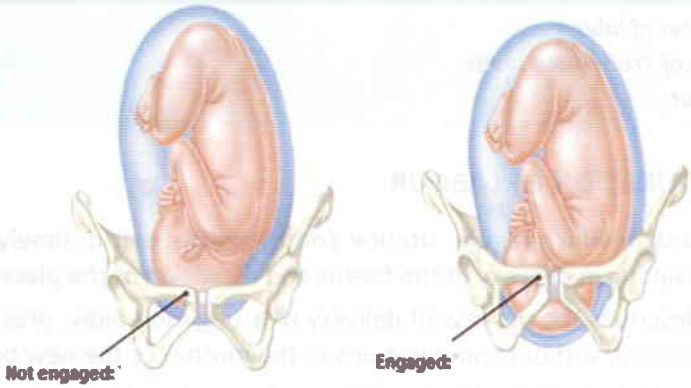


Fig 14-18: Engagement of foetal head

- Abdominally: The engaged head can't be grasped by the fingers of the right hand by the first pelvic grip but it can be palpated by the 2nd pelvic grip.
- Vaginally: the vault is felt at or below the level of the ischial spines on PV examinations.
- In a primigravida engagement of the foetal head may occur as early as 37-38 weeks due to the strong tonicity of the abdominal and uterine musculature.
- In multigravidas, and some primigravidas, engagement will occur only during the first stage of labour due to the down forces of contractions and retractions induced by true labour pains.

CAUSES OF DELAYED HEAD ENGAGEMENT IN A PRIMIGRAVIDA

MATERNAL CAUSES	FOETAL CAUSES
1. *Contracted pelvis* 2. *Placenta previa* 3. *Pelvic tumors* 4. *Full bladder or rectum* 5. *Atony of the abdominal muscles*	*1. OP position (commonest cause)* *2. Malpresentations (face, brow, breech)* *3. Multiple pregnancy* *4. Foetal Macrosomia (large baby)* *5. Prematurity & polyhydramnios* *6. Foetal head anomalies (hydrocephalus and anencephaly)*

11 STAGES OF LABOUR

- *Physiology and Onset of labour*
- *The characteristics of True labour pains*
- *The stages of labour*

DEFINITIONS AND DURATION OF LABOUR

- **Labour** is a process in which regular uterine contractions result in timely cervical effacement and dilatation, resulting in delivery of the foetus and expulsion of the placenta.
- **Normal labour** describes the process of delivery of a singleton baby, presenting by the vertex, through the birth canal, without complications to the mother or the new born.
- **Duration of Normal labour** varies from one patient to another, however;
  - In a *primigravida* duration is usually within 12-18 hours
  - In a *multigravida* duration is usually shorter being nearly 6-10 hours

PHYSIOLOGIC CHANGES PRIOR TO ONSET OF LABOUR

a) *Increased* ***Oxytocin*** *and* ***Prostaglandins (PGL)*** *levels* with multiplication of their **receptors**.
b) *Formation of* ***gap junctions*** between myometrial cells to facilitate cell to cell communication with passage of products of metabolism and electric current
c) *Formation of the* ***lower uterine segment (LUS)***, where the upper uterine segment (UUS) myometrium becomes thicker and LUS myometrium thinner.
d) ***Increased myometrial tone*** with irregular intermittent contractions due to interactions between myometrial proteins (**actin** & **myosin)** caused by myosin light chain kinase enzyme stimulated by PGL and calcium ions which show increased intracellular calcium influx.
e) ***Cervical Softening:*** due to increased water content secondary to increased levels of oxytocin and PGL which lead to breaking of the Disulphide linkage of collaged fibres within the cervix.
f) ***Cervical Effacement:*** The soft cervix gradually thins out with shortening of cervical canal.
g) ***Cervical Dilatation:*** this occurs when the passive LUS is thinned out and pulled up by the active UUS during the first stage of labour. Cervical dilatation is expressed in cm, being slow in the latent phase (till 3-4 cm), then more rapid in the active phase till complete dilatation reaching almost 10 cm.

ONSET OF LABOUR

The exact cause for onset of labour is **unknown**, theories and hypothesis include;

1. ***Progesterone (PRG) withdrawal:*** onset of labour is preceded by sharp decline in PRG levels
2. ***PGL Increase:*** PGL induce ripening of cervix and can initiate labour pains
3. ***Placental Oxytocinase decrease:*** allow pituitary gland oxytocin to act freely on myometrium
4. ***Uterine distension:*** as in cases of polyhydramnios and multi-foetal pregnancy (MFP) is commonly associated with PTL.
5. ***Increased foetal Cortisol:*** towards end of 3rd trimester may accelerate physiologic changes

CHARACTERISTIC OF TRUE LABOUR PAINS

- Start in the **UUS** near the fundus as **low** amplitude **short** duration involuntary, infrequent pains
- Gradually **increase** in frequency, amplitude and duration, with shorter intervening periods of rest
- **Retraction** occurs at the end of each contraction leading to permanent shortening of the muscle leading to descent of the presenting part with upward pulling of the LUS.
- During labour the **UUS** contracts and retracts, while **LUS** stretches and dilates resulting in progressive cervical effacement and dilatation.
- The physiologic **retraction ring**: is the demarcation between the active UUS and the passive LUS. It rises upwards towards the umbilicus with progress of labour.
- Uterine contractions are **painful** due to; compression of nerve ganglia, hypoxia to uterine muscle, stretch of peritoneum over LUS, and the stretch of cervical dilatation.
- Pain is felt at **intrauterine pressure** of 20-25 mm Hg. It reaches 50-60 mm Hg during active 1st stage of labour, and reaches 80 mm Hg at the 2nd stage of labour.

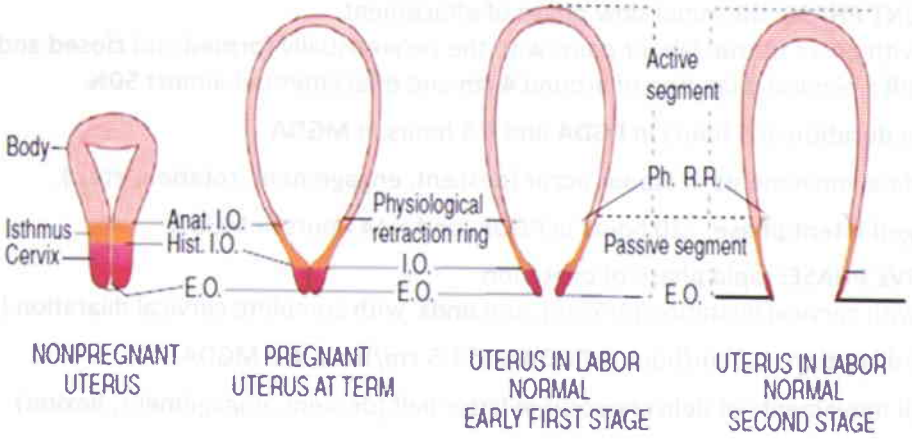


Fig 11-1: Uterine changes prior to and during labour

Comparison between true and false labour pains

	True labour pains	***False labour pains***
Site of pain	*Abdominal*	*Abdominal*
Rhythm	*Regular*	*Irregular*
Frequency, and strength	*Increasing*	*Stationary or decreasing*
Bulging membranes	*Present*	*Absent*
Cervical effacement	*Present*	*Absent*
Effect of sedation	*No effect*	*May relieve pain*

THE AUXILLARY FORCES OF LABOUR

1. Maternal bearing down during the 2nd stage of labour
2. Unfolding of the foetus during passage through the birth canal

THE STAGES OF LABOUR

Labour is classically divided into **3 stages** that include; cervical effacement and dilatation, delivery of the foetus, and delivery of the placenta. The 4th stage is the 1st 2 hours after delivery.

I. THE FIRST STAGE OF LABOUR

Stage of Cervical Effacement and Dilatation

- **Starts** with the onset of labour pains with the cervix formed and closed (around 3-4 cm in length & usually < 1.0 cm in diameter).
- **Ends** with full cervical effacement (very thin cervix) and complete dilatation (almost 10 cm wide)
- In a primigravida **(PGDA)** effacement precedes dilatation while in multigravidas **(MGDA)** effacement and dilatation occur simultaneously in a relatively shorter period.
- **Duration** of 1st stage is around 12-18 hours in a **PGDA** and 6-12 hours in **MGDA**.
- **Abnormalities** include; prolonged latent phase and delayed active phase

PHASES OF FIRST STAGE LABOUR

A) THE LATENT PHASE: the initial slow phase of effacement

- **Starts** with onset of true labour pains with the cervix usually **formed** and **closed** and proceeds slowly **till** a cervical dilatation of around **4 cm** and effacement of almost **50%**.
- Average **duration**: 6.5 hours in **PGDA** and 4.5 hours in **MGDA**
- **No** cardinal movements of labour occur (descent, engagement, rotation, etc...).
- **Prolonged latent phase**: >20 hours in PGDA, and > 14 hours in MGDA.

B) THE ACTIVE PHASE: rapid phase of dilatation

- **Starts** with cervical dilatation (4-5 cm), and **ends** with complete cervical dilatation (10 cm)
- **Rate** of dilatation: 1.2 cm/hour in PGDA and 1.5 cm/hour in a MGDA
- Cardinal movements of delivery occur in latter half (descent, engagement, flexion)

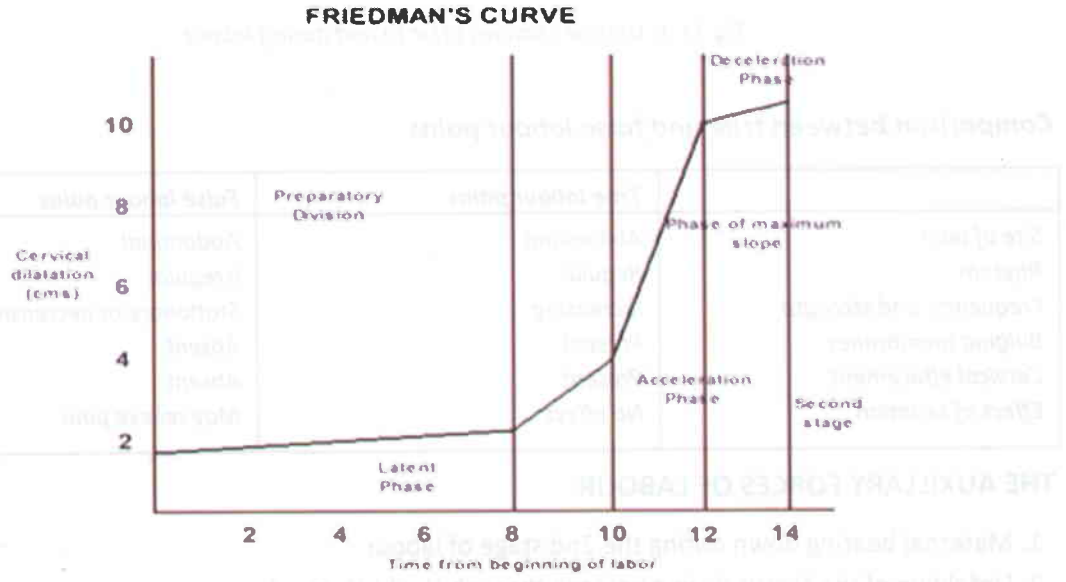


Fig. 11-2: First stage of labour

II. THE SECOND STAGE OF LABOUR

Stage of Delivery of the Foetus

- **Starts** by full cervical dilatation (10 cm) and **ends** by complete expulsion of the foetus
- Maternal **pushing down** efforts facilitate descent and delivery of the foetus through birth canal
- **Duration** in a **PGDA** 1-2 hours and in **MGDA** < 1 hour.
- **Abnormalities** include: prolonged 2nd stage and arrest of descent

III. THE THIRD STAGE OF LABOUR

Stage of Delivery of the Placenta

- ***Starts*** with delivery of the foetus and ***ends*** with expulsion of the placenta
- ***Mechanism*** *of placental separation:* myometrial **contractions** and **retraction** shear off the anchoring villi from the decidua basalis separating and expelling the placenta
- ***Signs of placental separation*** *and descent* include: gush of blood, elongation of the cord, and suprapubic bulge (the placenta descends to fill the LUS).
- *Average* ***duration*** is 10-30 minutes both
- in PGDA and MGDA
- ***Abnormalities*** include; prolonged 3rd stage (see retained and adherent placenta)



Fig 11-4: Delivery of the placenta

12 MANAGEMENT OF NORMAL LABOUR AND DELIVERY

- *Management of the 1st stage*
- *Management of the 2nd stage*
- *Management of the 3rd stage*
- *Management of the 4th Stage*
- *Care of the new born*

MANAGEMENT OF NORMAL LABOUR

Once the patient has been admitted in the 1st stage of labour, the following should be done;

- **Obtaining** comprehensive **data** on the **admission** sheet, to triage patients into normal and high risk cases and select the appropriate management teams and strategies.
- **Patient preparation;** through reassurance, education, advices for nutrition, cleansing and antiseptic measures, together with pain relief options.
- **Monitoring the progress of labour;** through a **Partogram** to allow for early detection of abnormal and or prolonged labour (see later).
- **Foetal surveillance;** throughout labour through repeated FHR auscultation or continuous electronic FHR monitoring. This will allow picking up cases with foetal distress and offer suitable management options (see chapter 13).
- **Special team** for the management of **high risk** pregnancies and dealing with **complications**.

DATA ON AN ADMISSION SHEET

- *Personal history:* Name, age, LMP, EDD, and Gestational age
- *Obstetric history:* Numerical 4 digit Code and patterns of previous deliveries
- *Medical disorders* recent or old complicating pregnancy to document high risk pregnancies
- *Abdominal examination:* for fundal height, foetal lie and presentation, and FHR auscultation
- *PV examination:* for cervical effacement (0-100 %), dilatation (0-10 cm), head station (-3 to +3), position of the head (LOA, ROA, ROP, LOP)
- *Membranes:* intact or ROM and duration of amniotic fluid leakage
- *Molding of the skull bones and presence of caput succedaneum*, for possible CPD
- *Evaluation of the bony pelvis,* to exclude *contracted pelvis* and *CPD.*

DATA ON ULTRASOUND EXAMINATION:

- *Foetal biometry & foetal weight* estimation (BPD, HC, AC, FL, etc.) to exclude SGA & LGA
- *Confirming presentation and position:* cephalic OA/OP, breech, and transverse lie
- *Amniotic fluid volume* and *placental localization:* to exclude PL Praevia and accreta
- *Exclude major foetal anomalies:* anencephaly, NTDs, hydrops Foetalis, etc.
- *Presence of associated pathology* as leiomyomas or ovarian masses
- *Evaluation of foetal wellbeing:* umbilical artery Doppler & biophysical profile if available

MANAGEMENT OF THE FIRST STAGE OF LABOUR

The **1st stage** of labour is the stage of **onset** of **true labour pains** and **cervical dilatation.** Its duration ranges from **12-18** hours in a primigravida and few hours less in a multipara.

A) Preparation of the patient:

- Ask the patient to evacuate the bladder and preferably the rectum.
- Cleansing the vulva and perineum using antiseptic solutions as Betadine or Chlorhexidin.

B) Pain relief: This may be achieved either by

- *Intermittent IM injections* of Pethidine 1mg/kg/4 hours, **or**
- *Epidural Analgesia* during the active phase of labour and throughout the process of delivery.

C) Nutrition: Light diet, oral fluids, and juices; to avoid hypoglycaemia

D) Instructions and Education: reassurance and avoid bearing down in the 1st stage

E) Monitoring Progress of labour using the PARTOGRAM:

- The Partogram is a graphic record for the progress of labour that allows an instant visual assessment of the rate of cervical dilatation and effacement plotted against descent of foetal head in comparison to an expected norm.
- A Partogram will allow early diagnosis of prolonged and abnormal labour, and whether this will occur in the latent or active phase.
- Other data that can be included in a Partogram include; timing of ROM, medications, frequency of uterine contractions, and basic observations as the Pulse, temp., and BP.

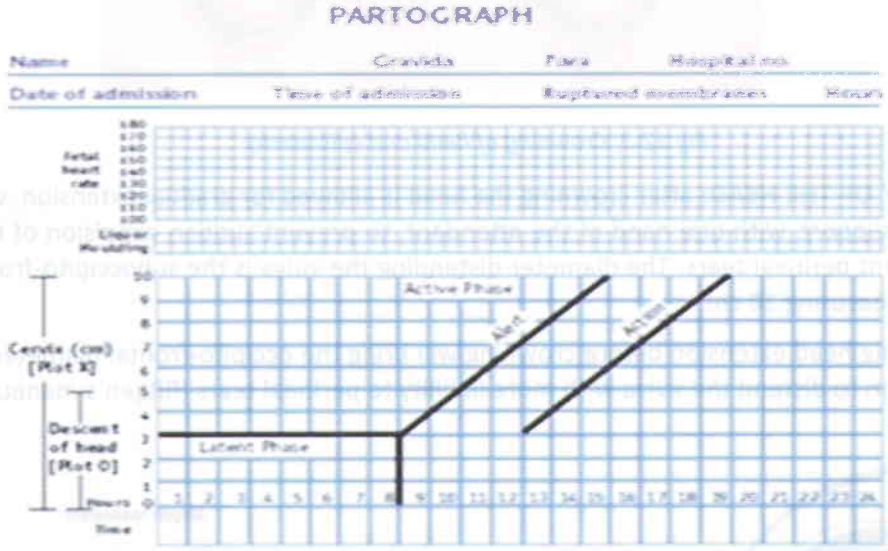


Fig 12-1: The Partogram

INTRAPARTUM FOETAL SURVEILLANCE (IFS):

Foetal surveillance during labour can be achieved via either;

1. **Intermittent** FHR recording by **Sonicaid** in normal low risk cases
2. **Continuous Electronic FHR Monitoring** by Cardiotography **(CTG)** in high risk cases. This will allow early detection and proper management of cases of foetal distress during labour together with selecting the most suitable intervention for the best outcome (see chapter).

MANAGEMENT OF THE SECOND STAGE OF LABOUR

The **2nd stage** of labour is the stage of **delivery of the baby**. It starts by full cervical dilatation and ends by expulsion of the foetus through the natural birth canal. It has an average duration of 2 hours in a primigravida and < 1 hour in multiparas.

- **Place of delivery:** patient is transferred to a specially equipped delivery room
- **Maternal position:** Lithotomy position is the commonest and most preferable
- **Ensure** evacuation of bladder by voiding or a urinary catheter
- **Sterilization:** the vulva and perineum are cleansed with antiseptic solution (Betadine)
- **Sterilized dressings** and **gowns:** The patient is completely covered by sterilized gown from the umbilicus to the legs and feet leaving only the region of vulva and perineum exposed.
- **Maternal bearing down:** which was prohibited in the 1st stage is now encouraged during the peak of uterine contractions with the head moving from station +1 to +2 and +3.
- **Ensure adequate analgesia** and if not available perform a local infiltration anaesthesia before performing an episiotomy
- **Episiotomy:** is performed when needed just before crowning of the head to avoid extensive perineal tears and facilitate head delivery.

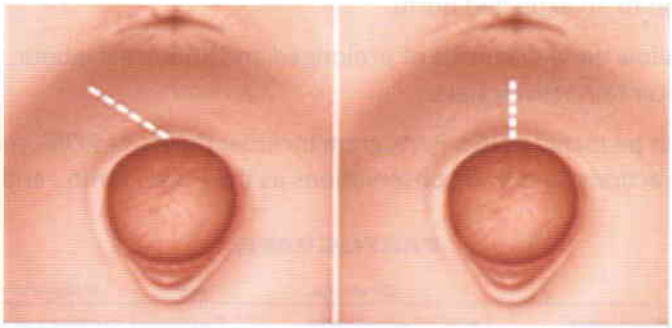


Fig 12-2: Crowning of head and episiotomy

- **DELIVERY OF THE HEAD:** after crowning the head is allowed for gradual extension, with adequate *perineal support*, with one hand of the attendant, to prevent sudden expulsion of the head with consequent perineal tears. The diameter distending the vulva is the suboccipito-frontal diameter (SOFD) measuring 10 cm.

N.B.: allowing head extension before crowning will bring the occipito-frontal diameter (OFD) measuring 11.5 cm to distend the vulva with more liability to perineal tears (Ritgen's maneuver)

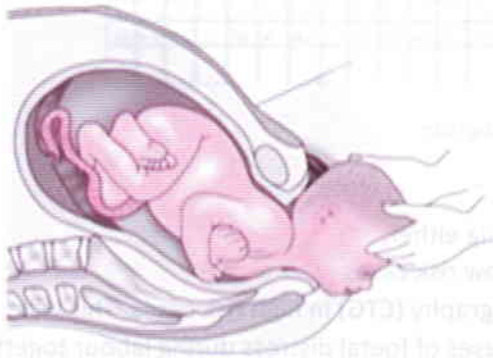


Fig 12-3: delivery of the head



Fig 12-4: Ritgen's maneuver for controlled extension

DELIVERY OF THE SHOULDERS AND BODY:

- The shoulders enter the pelvis in an oblique diameter opposite to that previously occupied by the head, descend through the pelvic cavity till anterior shoulder reaches the pelvic floor muscles then rotate anteriorly at the same time of external rotation of the head bringing the anterior shoulder directly behind the SP.
- Gentle downwards traction on the head till anterior shoulder appears under SP
- The head is then lifted upwards to deliver the posterior shoulder first
- The head is again pushed downwards to deliver the anterior shoulder
- The rest of the body slips down easily after delivery of the shoulders

- **Clamping the Umbilical Cord:** after delivery immediate care of the new born starts by clamping the umbilical cord and cleaning the air passages by nasal suction.



Fig. 12-6: Delivery of the anterior and posterior shoulders

MANAGEMENT OF THE THIRD STAGE OF LABOUR

The **3rd stage** of labour is the stage of delivery of the stage of **delivery of the placenta**

- **Duration** of this stage ranges from 10-30 minutes
- Wait for signs of **placental separation** and descent: gush of blood, elongation of the cord and suprapubic bulge.
- Gentle uterine **massage** is performed suprapubically on the uterine fundus to ensure uterine contractility and minimize bleeding
- Gentle **traction** on the cord will bring the placenta out of the introitus
- **Ecbolics** as Methergin (IM) and oxytocin drip (IV) are usually given immediately after delivery of the foetus to ensure rapid placental separation and avoid postpartum haemorrhage (PPH). Normal blood loss in 3rd stage < 400 ml.
- **Brandt-Andrews** method for active delivery of the placenta: It entails continuous controlled cord traction with the right hand while the left hand is pushing the uterus upwards suprapubically. Active placental delivery allows for reducing the time of 3rd stage, decreasing amount of blood loss, and decreasing chances for a retained placenta.
- **N.B.:** Vigorous manipulations may result in cord avulsion, and rarely acute inversion of the uterus.
- **Inspection** of the **placenta**: is mandatory to detect any missing cotyledon that may cause PPH
- **Inspection** for **laceration**
- **Episiotomy repair**: after delivery of the placenta the birth canal is inspected for vaginal, cervical, or perineal tears which will be dealt with by immediate surgical layered repair of lacerations and or episiotomy if present

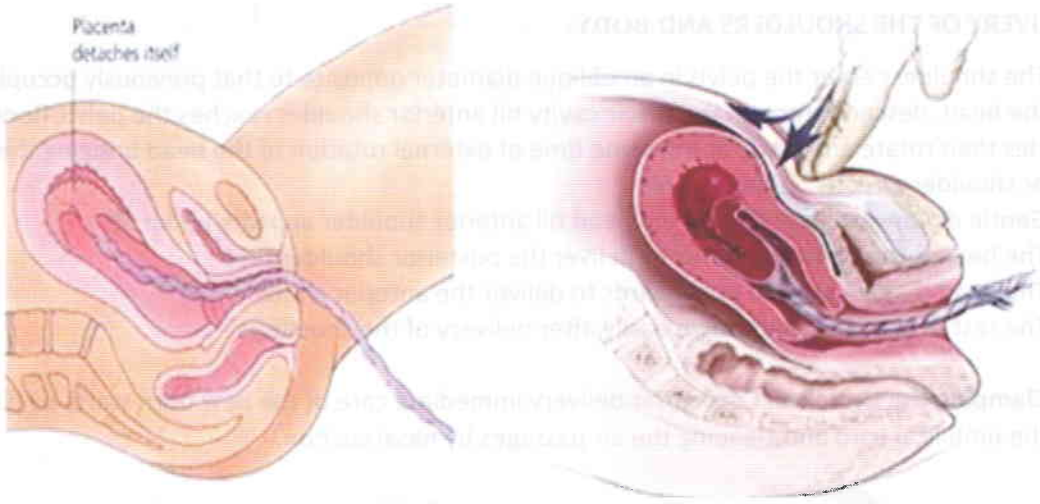


Fig 12-7: Conservative and active methods for delivery of the placenta

The **4th stage** of labour is described by some being the **first 1-2 hours** after delivery of placenta.

- It is the period in which the most serious **complications** of the 3rd stage will be arise and be detected and managed, as retained placenta, postpartum haemorrhage PPH, cervical lacerations, rupture of the uterus, obstetric shock, uterine inversion, etc.
- It is managed by careful observation for the vital signs, amount of vaginal bleeding, and degree of uterine contractility.
- Repeated gentle uterine massage for the first hour together with prophylactic ecbolics are advisable, as they improve the outcome and minimize the risks of PPH

CARE OF THE NEW BORN

1. **Warmth**; best under direct heat from a special unit (servo)
2. **Clear** the airways: by suction of secretions in the oral cavity and nose
3. **APGAR SCORE** is done at 1 & 5 minutes to evaluate foetal condition (see later)
4. **Identification** of the baby by arm and foot ID bands, then by the footprint
5. Examination for common **FCA** as: undescended testes, hypospadias, clubfoot, hare lip...
6. **Vitamin K** administration: for prevention of haemorrhagic disease of the new born
7. Care of the **eyes**; by use of antibiotic eye drops

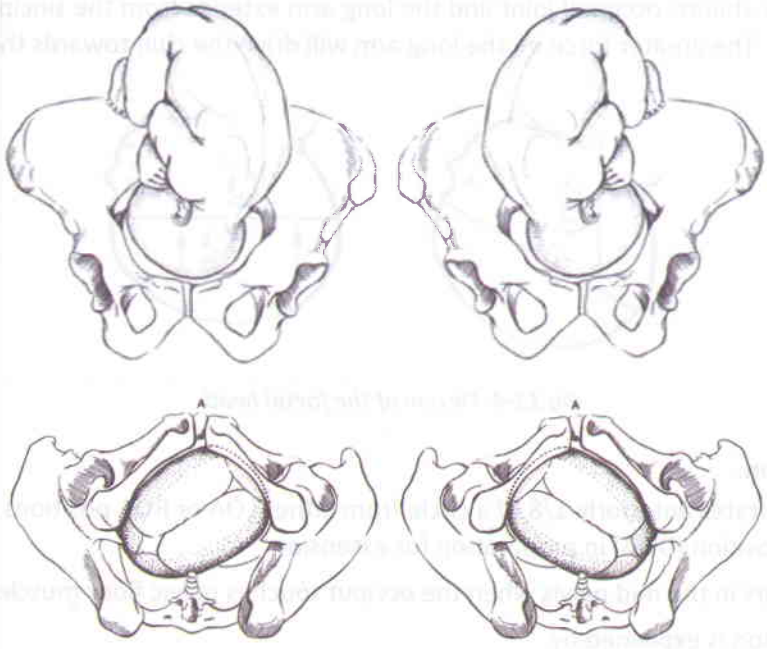
13 MECHANISMS OF DELIVERY IN CEPHALIC PRESENTATIONS

- *Occipito Anterior Positions*
- *Occipito Posterior Positions*
- *Face Presentation*
- *Brow Presentation*

OCCIPITO ANTERIOR POSITIONS

Occipito Anterior (OA) position is a **longitudinal** lie, cephalic presentation, with the foetal **back** directed **anterior**.

- **Incidence:** It is the most commonly encountered position being identified in about **70%** of cases in the 1st stage of labour.
- **Denominator:** the occiput (a bony prominence that lies just below the posterior fontanel)
- **Positions:** Left Occipito Anterior (LOA) and Right Occipito anterior (ROA)
- **PV Diagnosis:** palpation of the posterior fontanelle and lambdoid suture to the right or left in relation to the symphysis pubis (SP)
- **The course of labour**: in **OA** positions vaginal delivery is usually easily achieved in a reasonable period of time in presence of good uterine contractions, normal size gynaecoid pelvis and in absence of marked cephalo-pelvic disproportion (CPD). Maternal and foetal complications are usually minimal and thus OA positions are considered the model for normal labour and delivery.



LOA (60%) *ROA (20%)*

Fig. 13-1: Left and right Occipito-anterior position

MECHANISM OF DELIVERY IN OCCIPITO ANTERIOR POSITION

A) DELIVERY OF THE HEAD

1. **Descent:** a continuous through the birth canal in late 1st stage and 2nd stage of labour
2. **Engagement:**
   - Passage of the BPD (9.5 cm) through the plane of pelvic inlet
   - Clinically the vertex will be felt vaginally at the level of ischial spines by PV exam.

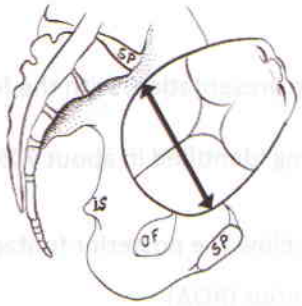


Fig 13-2: Non engaged head

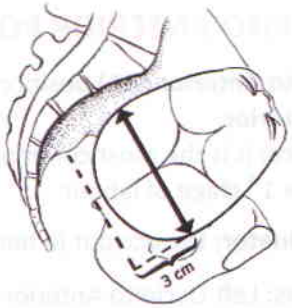


Fig 13-3: Engaged head

3. Increased Flexion:

- Flexion approximates foetal **chin** to the **chest** allowing the head to enter the mid pelvis with the least possible transverse and longitudinal diameters (BPD & SOBD **9.5** cm), facilitating rotation.
- Flexion movement is explained by the 2 armed lever theory; where the short arm extends from occiput to the atlanto-occipital joint and the long arm extends from the sinciput to the atlanto-occipital joint. The greater force on the long arm will drive the chin towards the chest.

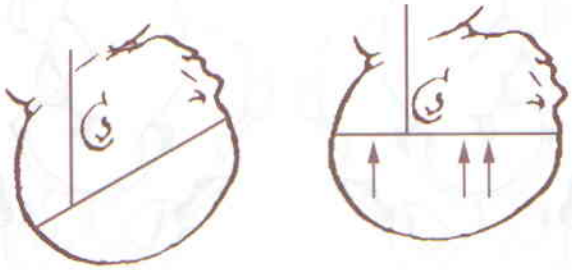


Fig 13-4: Flexion of the foetal head

4. **Internal Rotation:**

- The occiput rotates anteriorly 1/8 of a circle from either LOA or ROA positions to a direct Occipito anterior position (DOA) in preparation for extension.
- Rotation occurs in the mid pelvis when the occiput touches pelvic floor muscles
- Internal rotation is explained by
  - The direction of levators ani muscles; that slopes downwards forwards medially
  - The rifling action of the pelvis; where the largest diameter of the inlet is oblique while that of outlet is longitudinal forcing rotation in the mid-cavity.

5. **Extension:**
   - The sub-occipital region hinges under the symphysis pubis (SP)
   - The head is acted upon by two forces finally resulting in extension;
     - The downwards forwards force of uterine contractions and maternal pushing
     - The upwards forwards force of pelvic floor resistance

6. **Restitution:**
   - The head rotates back 1/8 of a circle in a direction opposite to that of internal rotation to undo the twist of the foetal neck caused by internal rotation.

7. **External Rotation:**
   - It is due to internal rotation of the anterior shoulder and occurs for another 1/8 of a circle in the same direction of restitution.

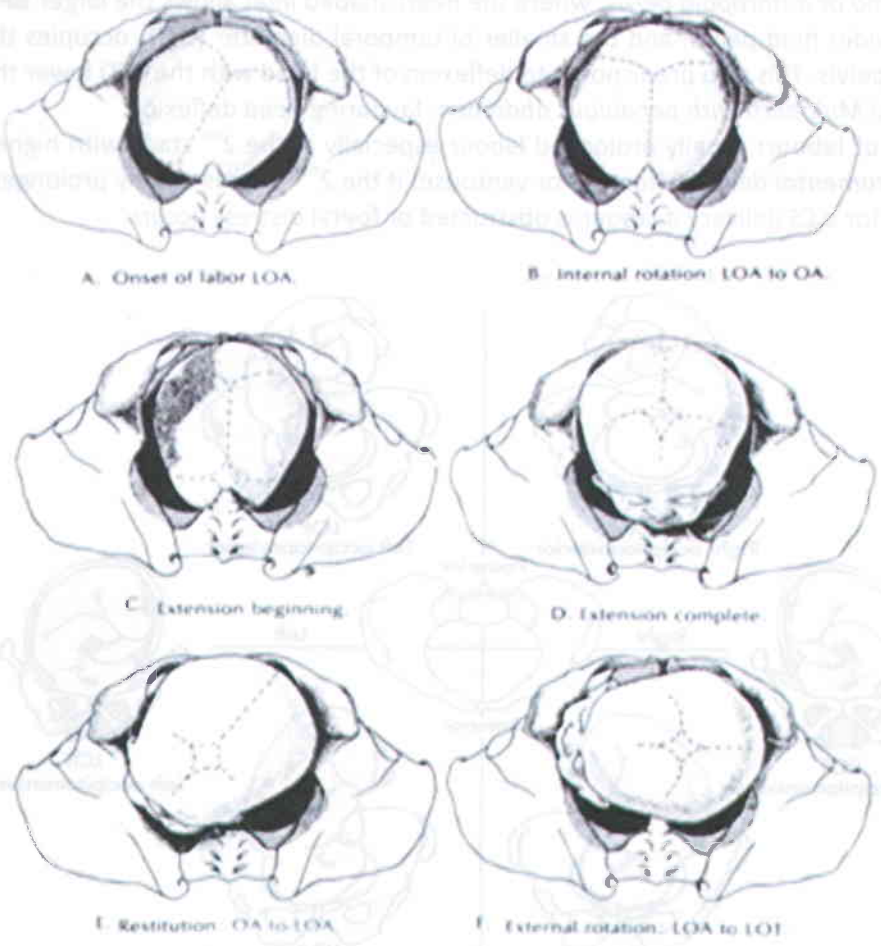


Fig 13– 5: Movements of head during normal vaginal delivery

B) DELIVERY OF THE SHOULDERS AND BODY

- The anterior shoulder hinges below the SP after anterior rotation 1/8 of a circle
- The posterior shoulder is delivered first by lateral flexion of the spine
- The anterior shoulder is then delivered followed by rest of body that slips outwards

OCCIPITO POSTERIOR POSITIONS

Occipito posterior (OP) position is a longitudinal lie, **cephalic** presentation **with the foetal back** directed **posterior**. It is considered a **malposition rather than a malpresentation.**

- **Incidence: OP** positions are encountered in around **20 %** of cases at the onset of labour
- **Denominator**: the occiput
- **Positions:** Right and Left Occipito posterior **(ROP – LOP)**
- **PV Diagnosis:** palpation of the posterior fontanelle and lambdoid suture posteriorly to the right or left in relation to the sacrum
- **ROP** are more common than **LOP** as the descending colon encroaches on the left oblique diameter of the inlet making it slightly smaller than the right oblique diameter.
- **Aetiology:**
  - *Idiopathic:* in many cases no underlying aetiology can be identified
  - *Android or anthropoid pelvis;* where the heart shaped inlet allows the larger **BPD** to occupy the wider hind-pelvis, and the smaller bi-temporal diameter **(BTD)** occupies the narrower fore-pelvis. This also predisposes to deflexion of the head with the BTD lower than the BPD
  - *Grand Multipara with pendulous abdomen:* favouring head deflexion
- **Course of labour:** usually prolonged labour especially in the 2nd stage with higher possibility for ***instrumental*** delivery (forceps or ventouse) if the 2nd stage is unduly prolonged, and more liability for a ***CS*** delivery if labour is obstructed or foetal distress occurs.

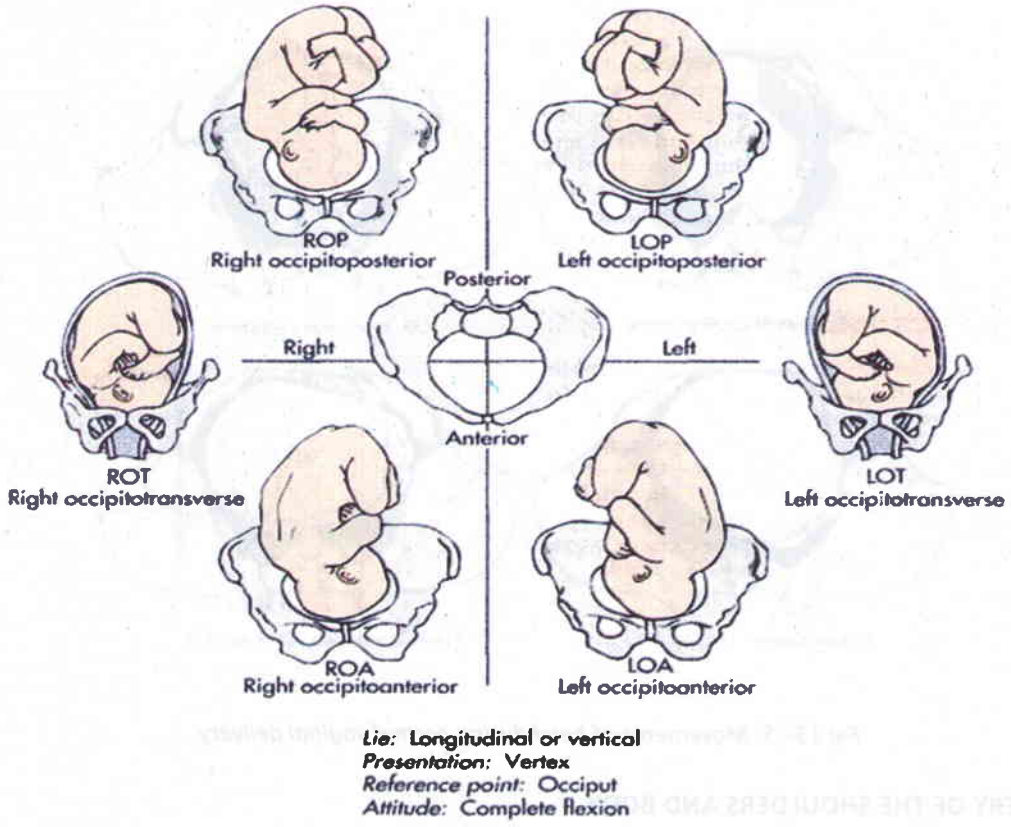


Fig 13-6: Right and left Occipito posterior positions

MECHANISMS OF DELIVERY IN OP POSITIONS

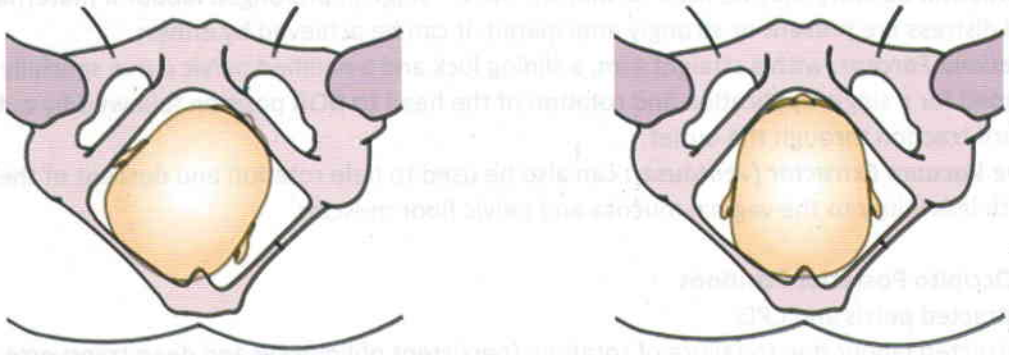
Descent and **engagement** as in OA position with BPD (9.5) cm traversing the pelvic inlet, this is followed by correction of **deflexion**, then log internal **rotation** with the following probabilities;

A. ANTERIOR ROTATION 3/8 OF A CIRCLE to a DOA Position (90%):

In around 90% of cases, and in presence of good uterine contractions and absence of CPD, correction of deflexion will occur, the Occiput will reach the pelvic floor and rotate anteriorly 3/8 of a circle or 135 degrees, from OP to OT to ROA or LOA. Once OA position is achieved, labour will then proceed normally to DOA position, where the occiput hinges below SP and the head will be delivered with a movement of **extension**.

B. POSTERIOR ROTATION 1/8 OF A CIRCLE to a DOP Position (6%)

In almost 6% of cases deflexion of the head allows for posterior rotation of the occiput 1/8 of a circle towards the sacrum in a DOP position. The head is delivered in **flexion** as **face to pubis**.



Right occiput-posterior (ROP) *Direct occiput-posterior (DOP)*

Fig 13-7: Posterior rotation of the occiput to DOP (face to pubis)

C. FAILURE OF ROTATION AND DEEP ARREST of the head (4%)

Failure to correct deflexion will bring both occiput and sinciput at the same level with failure of either part to reach the pelvic floor first resulting in **failure of rotation**. Labour will arrest with the sagittal suture either in an oblique diameter (persistent OP) or transverse position (deep transverse arrest). These positions may predispose to an **obstructed labour**.

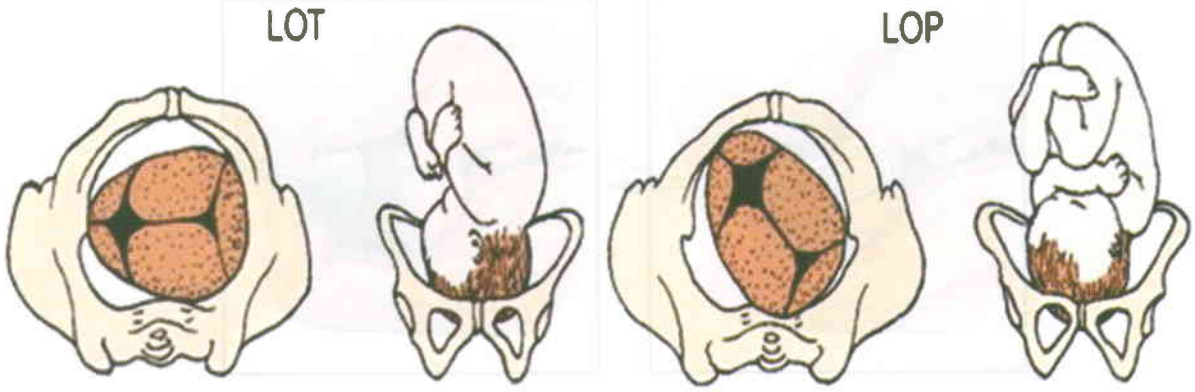


Fig 13-8: Deep arrest of the head in transverse or oblique position

Factors favouring internal rotation	*Factors interfering with internal rotation*
– *Correction of deflexion* – *Strong uterine contractions* – *Adequate pelvis no CPD* – *Good pelvic floor musculature* – *Intact membranes*	– *Persistent deflexion* – *Weak uterine contractions* – *Contracted pelvis or CPD* – *Poor pelvic floor musculature* – *Early PROM*

MANAGEMENT DURING LABOUR

A) Watchful Expectancy (Trial Labour):

In more than **90%** of OP successful head **rotation** will occur and **vaginal** delivery will be achieved in presence of adequate uterine contractions and sedation, and in absence of CPD.

B) Instrumental Delivery:

Instrumental delivery may be used to shorten the 2nd stage in prolonged labour if maternal or foetal distress are present or strongly anticipated. It can be achieved by either:

- ***Kielland Forceps:*** with a straight arm, a sliding lock and a nullified pelvic curve specially designed for a single application and rotation of the head to DOA position followed by outward traction through the outlet.
- ***The Vacuum Extractor (ventouse):*** can also be used to help rotation and descent of the head with less injury to the vaginal mucosa and pelvic floor muscles.

C) CS in Occipito Posterior Positions

- Contracted pelvis and CPD
- Obstructed labour due to failure of rotation (persistent oblique OP and deep transverse arrest)
- Prolonged labour with fetal distress or marked maternal exhaustion.

- ***N.B.: Prerequisites for instrumental delivery include:***
  - Exclusion of contracted pelvis and CPD
  - Engagement of the head with ROM and full cervical dilatation
  - Adequate analgesia preferably regional (epidural) analgesia.

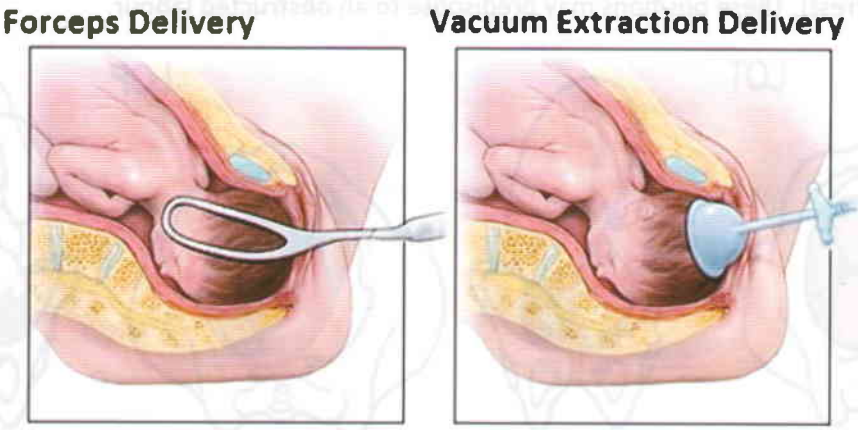


Fig 13-9 Kielland Forceps and Ventouse delivery in OP position after rotation to DOA

FACE PRESENTATION

Face presentation **is a longitudinal** lie**, cephalic** presentation**, with the head fully extended.**

- **Incidence:** It is an uncommon **malpresentation** occurring in around **1/500** deliveries.
- **Face:** is the part lying between the root of the nose and the chin (Mentum)
- **Positions:** Right and left mento anterior and mento posterior (RMA - LMA - RMP - LMP).
- **PV Diagnosis:** landmarks include the chin (mentum), alveolar margin, and malar bones.
- **MA** occur > **MP** positions as they usually develop 2ry to full **extension** of **OP** positions.
- **LMA** is the commonest position as it occurs 2ry to full extension of a head in the **ROP** position.
- **Aetiology:**
  1. *Idiopathic:* in many cases no underlying factor for face presentation can be identified.
  2. *Contracted pelvis* especially contracted inlet in PGDA.
  3. *Pendulous abdomen* in grand multipara.
  4. *Anencephaly:* the baby presents by the face instead of the absent vault.
- **Course of labour:** usually prolonged both 1st and 2nd stage with higher CS rates due to; delayed descent and engagement, difficult rotation in android pelvis especially in MP positions, absent moulding of maxillary bones, PROM, and delivery of the head ina longer vertical diameter.

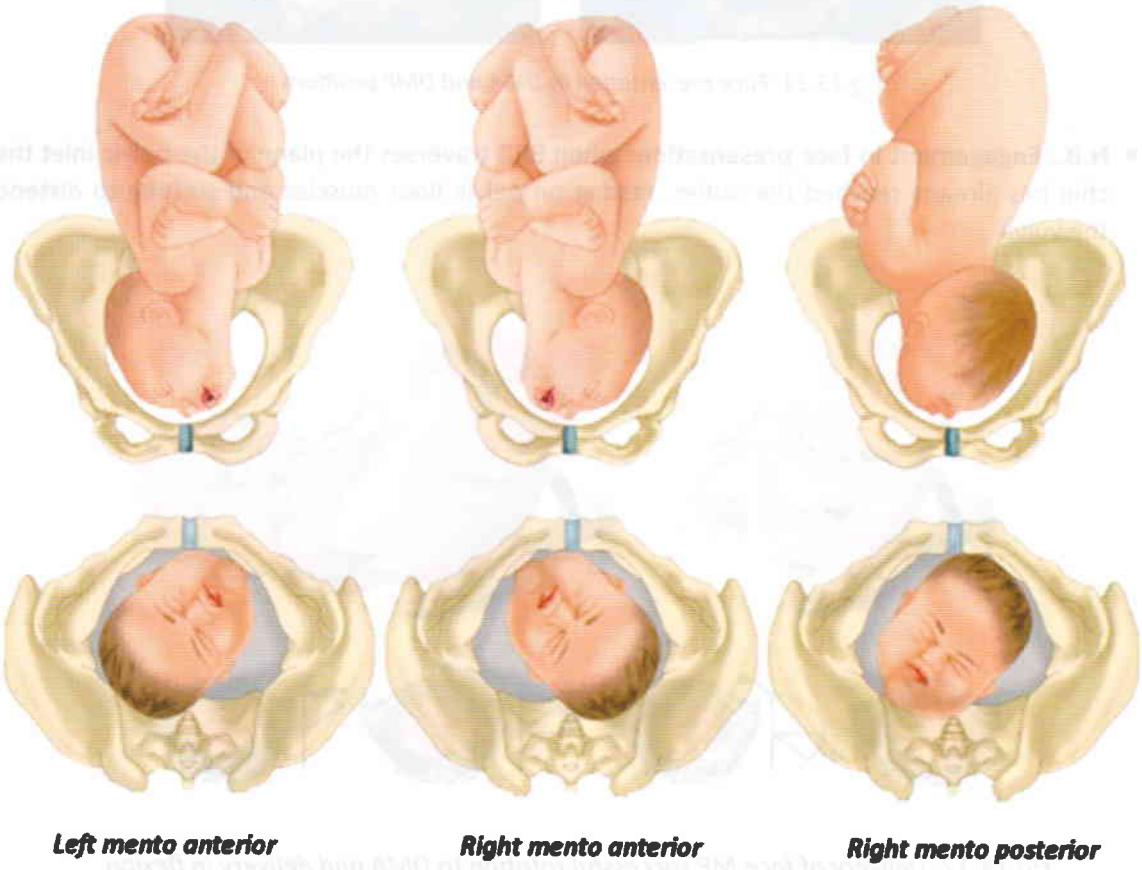


Fig 13-10: Face presentation MA and MP positions

MECHANISM OF DELIVERY IN FACE PRESENTATIONS

A) MENTO ANTERIOR POSITIONS (RMA – LMA)

- Delayed **engagement** of the head with **BPD** (9.5 cm) passing through the pelvic inlet.
- ***Rotation*** *of the Mentum* ***1/8*** *of a circle anterior from RMA & LMA to* ***DMA*** *position.*
- **Delivery** of the head in **flexion** with the **SMV** diameter (11.5 cm) distending the vulva.
- Flexion in **DMA** can occur as the longer neck is facing the shorter symphysis pubis (SP).
- Successful delivery will thus be achieved in face MA positions **in absence of CPD**

B) MENTO POSTERIOR POSITIONS (RMP – LMP)

- In **2/3** of cases successful long anterior **rotation 3/8** of a circle to a **DMA** position and delivery
- In **1/3** of cases **failure** of anterior **rotation** will lead to **obstructed labour** to either;
  - **Arrest** of face in either **oblique** or **transverse** diameters, or
  - ***Posterior rotation*** of the Mentum to direct MP **(DMP).** In such cases the head cannot flex facing the long sacrum, and cannot extend being already fully extended.



Fig 13-11: Face presentation in DMA and DMP positions

- **N.B.: Engagement in face presentation:** when BPD traverses the plane of the pelvic inlet the chin has already reached the outlet, resting on pelvic floor muscles and starting to distend the vulva

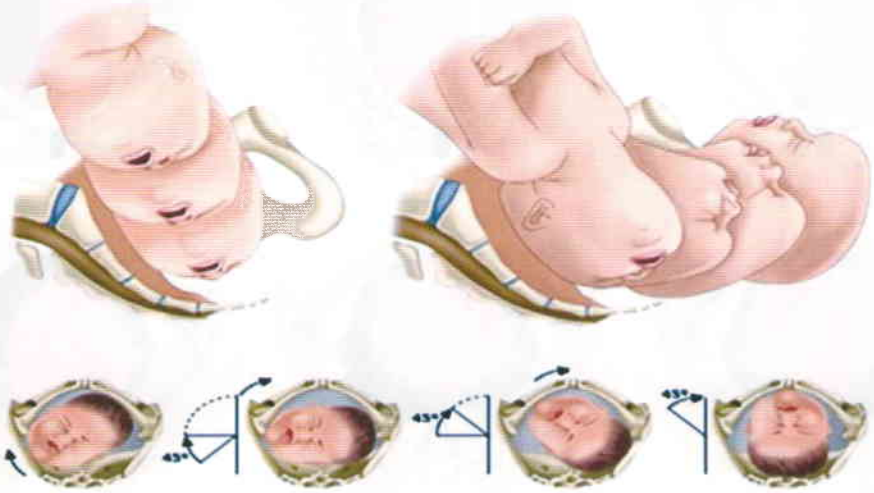


Fig 13-12: Delivery of face MP successful rotation to DMA and delivery in flexion

MANAGEMENT DURING LABOUR

- **Watchful expectancy** for **trial labour** as most **MA** positions and **2/3** of **MP** positions will deliver vaginally in presence of good uterine contractions, proper sedation and absent CPD.
- **Adequate episiotomy** will be mandatory as the diameters distending the vulva are larger than that for Occipito-anterior position
- **Forceps delivery:** are sometimes indicated to shorten the 2nd stage of labour in caqses where foetal distress or maternal exhaustion are present or strongly anticipated
- **Indications of CS in Face Presentation**
  - Persistent Oblique, Transverse or Direct MP positions predisposing to an obstructed labour
  - CPD, foetal distress, or marked maternal exhaustion.
  - Other obstetric indications for a CS as a previous CS, myomectomy scar, PL PRV, MFP, etc...

BROW PRESENTATION

Brow presentation is a **longitudinal** lie, **cephalic** presentation, with the head **incompletely** extended. It is a rare **malpresentation** occurring in **< 1/2000** delivery.

- **Brow:** is that part lying between the root of the nose and the anterior fontanel
- **Engaging diameter** in brow is the mento vertical diameter **MVD** (13.5 cm) which is larger than any diameter in the pelvic inlet making engagement impossible. Labour will thus be doomed to be obstructed unless the presentation is corrected allowing engagement.
- **Transient Brow:** usually occurs during conversion of OP position into face presentation
- **Persistent Brow:** head stops midway between flexion and extension and fails to correct neither to face (complete extension), or to OP (convert to incomplete flexion).
- **PV Diagnosis:** landmarks include the anterior fontanelle and the root of the nose

MANAGEMENT DURING LABOUR

1. Exclude CPD and contracted pelvis, followed by watchful expectancy; waiting for transient brow to turn face or OP, then continue trial of labour
2. **CS** is indicated in all cases of **Persistent** brow due to failure of engagement and obstructed labour

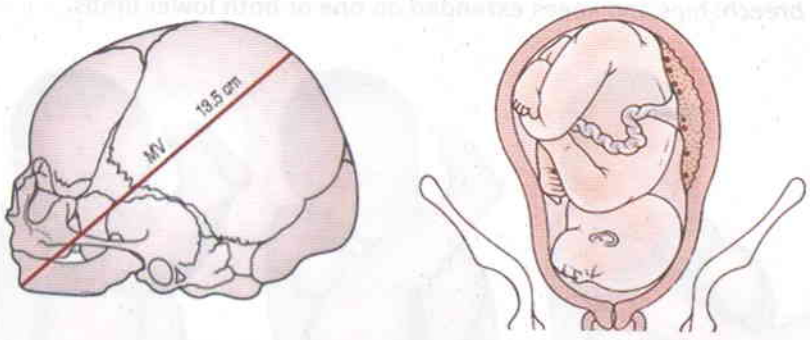


Fig 13-13: Brow presentation and MV diameter

14 MECHANISMS OF DELIVERY IN FOETAL MALPRESENTATIONS

- *Breech Presentation*
- *Shoulder presentation*
- *Cord presentation and Cord prolapse*
- *Complex Presentation*

BREECH PRESENTATION

Breech presentation is a **longitudinal lie** in which the **buttocks,** or the **lower extremities,** present into the maternal pelvis.

- **Incidence:** It is the commonest **malpresentation** being encountered in **3-4 %** of **term** pregnancies at the onset of labour. The incidence is much higher in **preterm** labour and multifoetal pregnancies **(MFP).** Before 28 weeks gestation almost 25% of foetuses are presenting as a breech.
- The **Denominator** in breech is the **sacrum**, and **positions** include;
  - Right and left Sacro-anterior position (RSA & LSA)
  - Right and Left Sacro-posterior positions (RSP & LSP).
  - Sacro-anterior positions are generally more common than Sacro-posterior positions

TYPES OF BREECH

1. **Complete breech:** hips and knees are both **flexed** with feet presenting beside the buttocks.
2. **Incomplete breech:** partial or complete **extension** is present at hip, knee, or both;
   - ***Frank breech:*** hips **flexed** and knees **extended** (buttocks only form the presenting part)
   - ***Footling breech:*** hips and knees **extended** on **one** or **both lower limbs.**

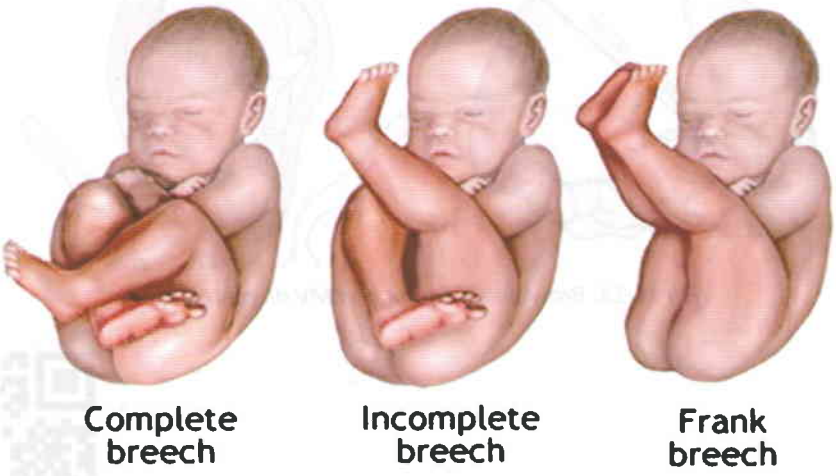


Fig 14-1: Common types of breech presentation

AETIOLOGY

Any factor that interferes with adaptation of the head and buttocks to the pyriform shape of the uterine cavity may predispose to breech presentation although most are idiopathic:

1. **Idiopathic:** in at least 1/3 of cases no underlying aetiology can be detected
2. **Prematurity:** This is the major factor predisposing to breech presentation with an incidence of around 25% before 28 weeks gestation, declining gradually to only 3-4% at 36 weeks. This is due to the unstable foetal lie associated with the small size foetus and relatively larger amniotic fluid volume till spontaneous rectification into vertex occurs < 36 weeks.
3. **Twins** and Multifoetal pregnancy **(MFP)**: if one is vertex then the second will be mostly either breech or transverse lie
4. **Uterine abnormalities;** that interferes with foetal adaptation to the shape of uterine fundus as septate and bicornuate uterus, large uterine leiomyomas, and placenta praevia (PL PRV)
5. **Foetal Congenital anomalies:** as hydrocephalus and anencephaly
6. **Other causes of malpresentations as;** polyhydramnios, contracted inlet, etc...

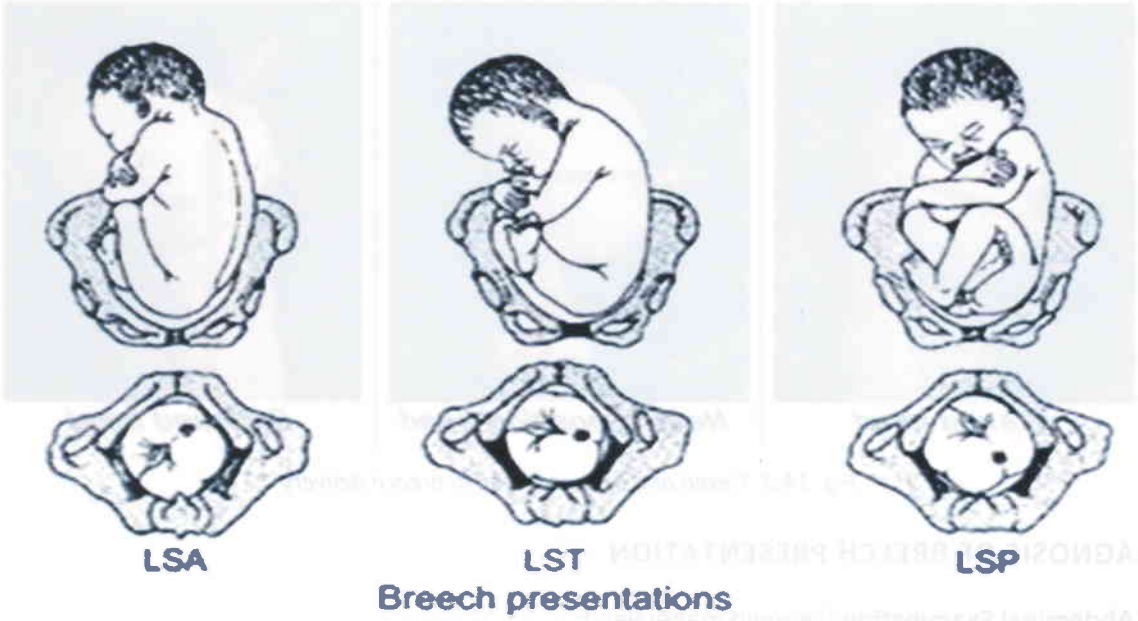


Fig 14-2: SA and SP positions

RISKS ENCOUNTERED WITH BREECH PRESENTATION

1. Inability to asses foeto-pelvic disproportion with the foetal head in the fundus
2. Increased incidence of PROM, dysfunctional labour and prolonged labour.
3. Increased incidence of head entrapment, if delivery of the body occurs before full cervical dilatation, predisposing to foetal and neonatal asphyxia and intracranial haemorrhage
4. Vigorous traction on the foetal trunk, to accelerate head delivery, may result in serious foetal birth injuries, together with maternal cervical and vaginal lacerations.
   - **N.B.:** Due to various uncalculated maternal and foetal hazards, almost **80%** of breech presentations will be delivered by **Caesarean section** in many centres all over the world

MECHANISM OF DELIVERY IN VAGINAL BREECH DELIVERY (VBD)

1. **Descent:** during the 1st stage with good uterine contractions and retraction
2. **Engagement:** when bitrochanteric diameter **(9.5-10 cm)** traversing the plane of the inlet
3. **Internal rotation:** the anterior buttock reaches the pelvic floor first, rotates **1/8** circle anteriorly, hinges below the SP with the posterior buttock facing the sacrum.
4. **Delivery of the buttocks:** the posterior buttock and hip are delivered through the distended vulva by lateral flexion of the spine with the sacrum at a transverse position
5. **Delivery of the shoulders:** the shoulders enter the pelvis in the same oblique diameter as the buttocks, the anterior shoulders reaches the pelvic floor first, rotates **1/8** circle anterior, hinges below the SP, the posterior shoulder is delivered followed by the anterior.
6. **Delivery of the after-coming head:** the head enters the pelvis in an oblique diameter opposite to that of the buttocks and shoulder. The chin touches the pelvic floor muscles, the head rotates bringing the occiput to hinge below the SP. The head is then delivered in flexion.

N.B.: in Sacro posterior positions the occiput will enter the pelvis in a posterior position and has to rotate 3/8 of circle anteriorly to DOA position to be delivered.

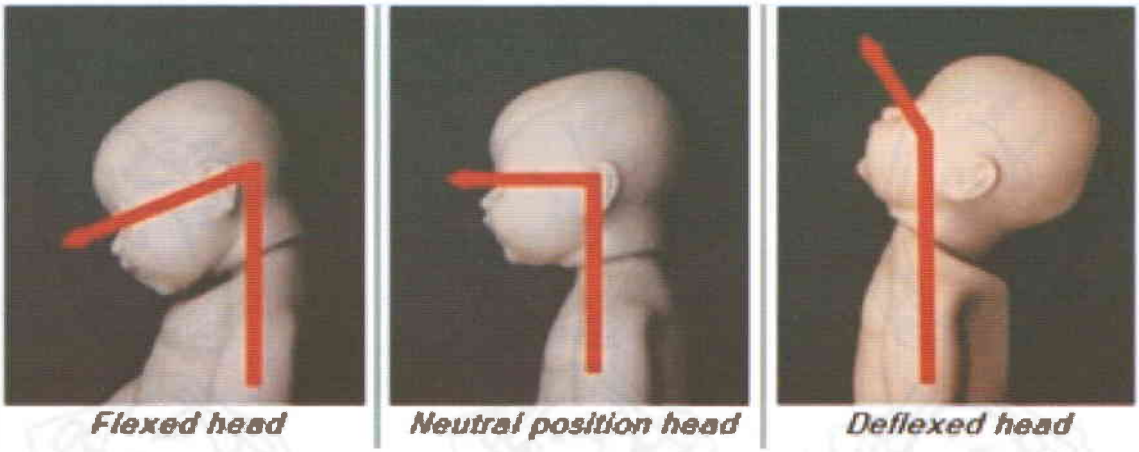


Fig. 14-3: Flexed and deflexed head in breech delivery

DIAGNOSIS OF BREECH PRESENTATION

A. Abdominal Examination (Leopold maneuvers):

- Fundal grip shows a head (hard, rounded, regular, and ballotable)
- Pelvic grip shows a buttock (soft, globular, and irregular)

B. US Examination: Longitudinal lie with head at fundus and buttocks at pelvis

C. PV during labour: with the cervix partially dilated and ROM, shows the soft buttocks, with its characteristic bony landmarks (the tip of sacrum and the 2 ischial tuberosities).

MANAGEMENT OPTIONS IN BREECH PRESENTTAIONS

A. EXTERNAL CEPHALIC VERSION (ECV) to allow trial vaginal delivery as cephalic presentation

B. TRIAL VAGINAL BREECH DELIVERY (VBD) in selected cases (see later)

C. ELECTIVE CS when indicated and when both ECV and VBD are unsafe (see later)

A) EXTERNAL CEPHALIC VERSION

External cephalic version (ECV) describes conversion of breech into cephalic presentation via rotating the foetal body through manipulations on the maternal abdomen. The aim is to correct the malpresentation, avoid hazards of vaginal breech delivery, and minimize the rate of CS

- **Timing:** best at 35-37 weeks but can be done even at onset of labour, however early ECV < 35 weeks may predispose to recurrence of the malpresentation, while late ECV > 37 weeks is more difficult due to larger foetal size and less amniotic fluid ate ECV may be difficult with a larger size foetus and less amniotic fluid
- **The Procedure:** see figure for description

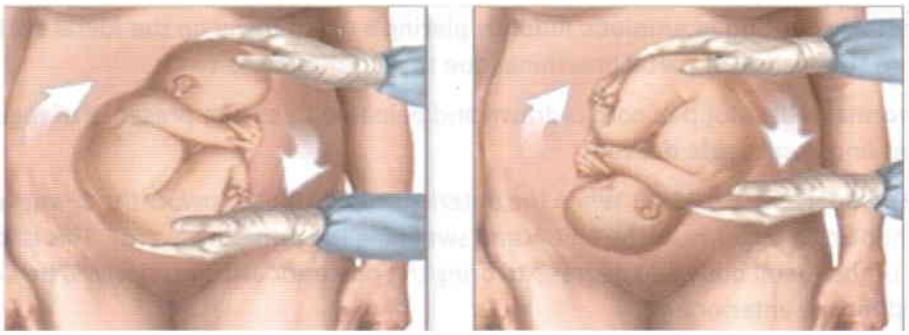


Fig 14:4 external cephalic version ECV

- **Risks and complications of ECV**
  - Placental abruption and accidental haemorrhage
  - ROM and initiation of PTL
  - Cord accidents with loop around foetal neck
- **Contraindications of ECV**
  - MFP, oligohydramnios, uterine anomalies and leiomyomas.
  - When CS is already indicated: as in previous uterine scat (CS or myomectomy), Pl PRV, preeclampsia, foetal macrosomia, contracted pelvis, etc...

B) CAESAREAN SECTION IN BREECH PRESENTATION

CS is offered to almost **80%** of cases with **breech** presentation due to the unpredictable outcome and hazards associated with vaginal breech delivery especially in a **primigravida.**

Indications of CS in Breech Presentation

1. Foetal weight > 3.5 kg (LGA foetus) and < 2.0 kg (extremely preterm or SGA foetus)
2. Footling breech presentation: probable ROM, and delivery before full cervical dilatation
3. Extension of foetal neck by US: to avoid arrest of descent and failure of head rotation
4. Twins and MFP when first is breech (to avoid locked twins)
5. Other Obstetric indications for a CS as: contracted pelvis, PL PRV, Previous uterine scar, elderly primigravida, infant of diabetic mother with macrosomia, etc...

C) VAGINAL BREECH DELIVERY (VBD)

Vaginal breech delivery (VBD) is allowed whenever initial evaluation reveals an average size, full term baby, with normal progress of 1st stage of labour, in absence of contracted pelvis or suspected disproportion. The patient is prepared as in normal labour (see before).

- **Avoid maternal bearing down** before the buttocks reach the vulvar ring
- **Ensure full cervical dilatation** before attempting delivery
- **EPISIOTOMY** should be performed in **all cases** when the buttocks distend the vulva. It should be adequate to allow for lateral flexion of the spine and easy delivery.
- **Delivery of the Buttocks and Legs:** after episiotomy the buttocks and legs are delivered slowly with controlled perineal support keeping the back always anterior.
- **Avoid foetal aspiration of amniotic fluid:** by placing a warm towel on the foetal back to avoid premature stimulation of foetal breathing (due to cold stimulus).
- **Ensure normal FHR:** a loop of cord is drawn and palpated to ensure presence of regular heart pulsations and ensure safe delivery
- **DELIVERY OF THE SHOULDERS:** *When the anterior scapula hinges below the SP* the arm is delivered by hooking a finger near the elbow and sweeping the arm downwards. This is followed by rotation of the foetal body 180 degrees to bring the posterior arm anteriorly to be delivered as described for the anterior arm.
- **DELIVERY OF THE AFTER-COMING HEAD:**

With the baby hanging down by its weight the head descends until the occiput hinges behind the SP, followed by either;

A. BURNS- MARSHAL'S METHOD: when the occiput appears anteriorly hinging below the SP, the infant's legs and feet are held and lifted upwards in a sweeping movement towards maternal abdomen. The head will flex gently and deliver vaginally.

B. JAW FLEXION SHOULDER TRACTION METHOD *(Mauriceau-Smellie-Veit manoeuver)*: with the back anterior the foetus is held with the ***left hand*** of obstetrician. The index and middle fingers of the ***right hand*** start gentle traction at the foetal neck downwards while the index and middle finger of the left hand keep pressing on the maxilla to promote flexion of the head, with the assistant exerting a gentle suprapubic pressure to guide the head down through the pelvis. When the Occiput is traversing the pubic arch the baby is lifted outwards and upwards, to deliver the head in flexion.

C. FORCEPS DELIVERY: A specially designed long forceps (Piper's Forceps) has been formerly used is some cases as it promotes head flexion and allows traction from the head preventing overstretch of the foetal neck. It is rarely used in modern obstetrics as its use has been associated with maternal birth canal injuries and most cases with anticipated difficult delivery are usually offered CS.

THE EXTREMELY PRETERM BREECH

The extremely preterm breech < 32 weeks, with a low birth weight < 2.0 kg is a high risk for VBD and is better offered delivery by CS due to more liability to birth injuries and less tolerance to asphyxia and head compression. It is also more vulnerable to delivery of the buttocks through a non - fully dilated cervix with consequent head entrapment (see later).

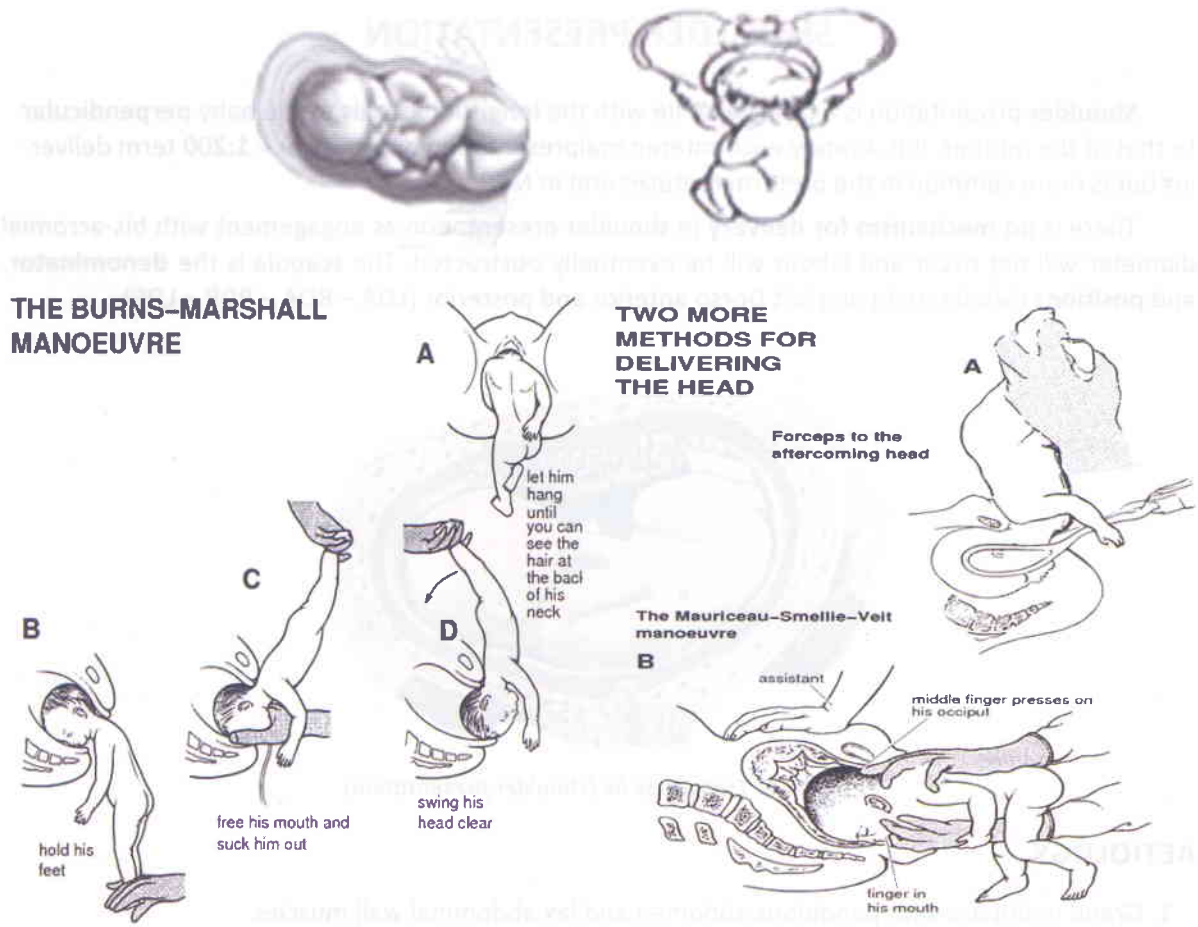


Fig. 14-5: The 3 Methods for Assisted Vaginal Breech Delivery

DIFFICULTIES AND COMPLICATIONS DURING VBD

A. **Extension of the Arms before delivery of the Shoulders:** managed by rotating the foetal trunk 180 degrees to bring the posterior shoulder anteriorly below the SP and sweeping the anterior arm down, then repeating the same procedure with the posterior shoulder *(Lovset's Maneuver)*.

B. **Posterior Rotation of the Head:** with the occiput directed posterior. Delivery can be accomplished by flexing the whole body towards maternal abdomen, and performing downwards traction on the shoulders from behind by the other hand to deliver the head in flexion *(Prague's Maneuver)*.

C. **Foetal Head Entrapment:** due to either delivery before full cervical dilatation or an undiagnosed cephalopelvic disproportion (CPD). The consequences are serious and may end in severe foetal birth injuries primarily due to excessive traction on the neck and sudden head compression and decompression. Complications include; intrapartum hypoxia, intracranial haemorrhage, brachial plexus injury, fracture of the clavicle, rupture of the liver or spleen, and hip dislocation.

D. **Maternal complications**: include maternal exhaustion from a prolonged labour, birth canal injuries, postpartum haemorrhage, and puerperal sepsis from extensive maneuvers and prolonged ROM.

SHOULDER PRESENTATION

Shoulder presentation is a **transverse lie** with the **longitudinal axis** of the baby **perpendicular** to that of the mother. It is a rarely encountered malpresentation occurring in < **1:200** term deliveries but is more common in the preterm foetuses and in MFP.

There is **no mechanism for delivery** in shoulder presentation as engagement with bis-acromial diameter will not occur and labour will be eventually obstructed. The scapula is the **denominator**, and **positions** include: right and left Dorso anterior and posterior (LDA – RDA – RDP – LDP)

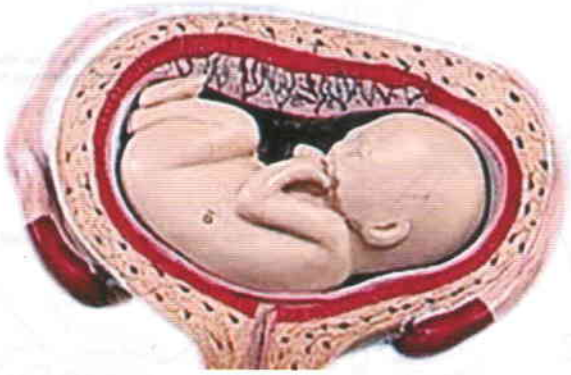


Fig 14-6: Transverse lie (shoulder presentation)

AETIOLOGY

1. Grand multipara with pendulous abdomen and lax abdominal wall muscles
2. Prematurity, polyhydramnios, MFP, PL PRV, and contracted pelvic inlet.
3. Septate and bicornuate uterus, and pelvic masses as leiomyomas and large ovarian swellings

DIAGNOSIS

A. **Abdominal Examination** (Leopold manoeuvers): Fundal level is lower than expected, with head felt on one side and buttocks on the other side of the midline by the umbilical grip.
B. **PV Examination:** with cervix partially dilated during labour shows a high non engaged presenting part with the ribs felt above the pelvic inlet
C. **Ultrasound Diagnosis:** is the most important tool that gives a rapid easy and definitive diagnosis and helps identifying the aetiology, and exclusion of foetal and uterine anomalies and PL PRV.

MANAGEMENT OF SHOULDER PRESENTTAION

1. EXTERNAL CEPHALIC VERSION:

- ECV entails correcting transverse lie into a cephalic presentation through rotation of the body 90 degrees on its axis, i.e.; 2/8 of a circle. ECV is best performed during late pregnancy > 34 weeks gestation, but can be practiced even at onset of labour.
- ECV can be performed whenever a single foetus, of average birth weight, is presenting in a transverse lie, with adequate amount of liquor amnii, intact membranes, absent uterine anomalies, whenever safe vaginal delivery is expected after malpresentation correction.

2. CAESAREAN SECTION:

- CS is indicated whenever ECV is unsafe or impossible to perform as with; uterine anomalies, MFP, PL PRV, uterine scars as CS or myomectomy, ROM, or foetal distress

3. INTERNAL PODALIC VERSION (IPV) and Breech Extraction During Labour

- The main indication of this procedure is for delivery of the second twin presenting as a transverse lie. It can be sometimes performed in the rare occasion in which a multipara is presenting late in labour with the cervix fully dilated and membranes intact.
- It is the method by which the transverse baby is delivered from the uterus during CS

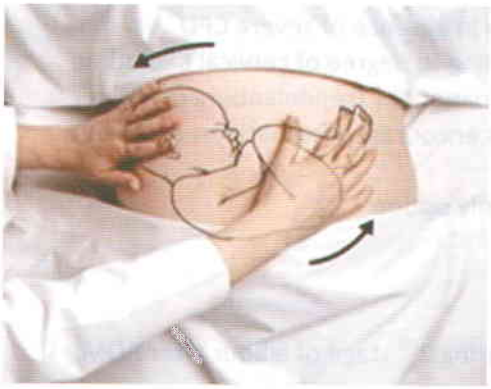


Fig. 14-6: ECV



Fig. 14-7: IPV and breech extraction

NEGLECTED SHOULDER: when after ROM and partial cervical dilatation one arm is prolapsed through the vagina. CS is the only solution to avoid IUFD and rupture of the uterus.

UNSTABLE LIE: when the foetus continuously changes its axis from longitudinal to transverse presentation after 34 weeks.

CORD AND COMPLEX PRESENTATION

CORD PRESENTATION:

In cord presentation the cord lies below the presenting part with **intact membranes**. It is a rare condition (< 1/300 deliveries), which once discovered early in labour the patient should be offered an immediate **CS.** It occurs more commonly in presence of malpresentations as breech and transverse lie in which there is delayed or absent engagement of the presenting part.



Fig 14-8: Cord presentation



Fig 14-9: Cord prolapse

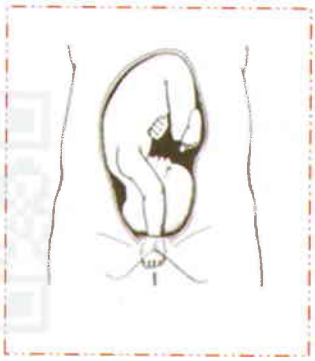


Fig 14-10: Complex presentation

CORD PROLAPSE:

The cord lies below the presenting part **after ROM**. It is considered one of the most acute obstetric emergencies as it is life threatening to the foetus if still living at the time of diagnosis.

Diagnosis:

- PV examination reveals loops of cord felt through the partially dilated cervix or upper vagina.
- Cord is palpated for presence of foetal pulsations to exclude IUFD
- If cord is non pulsating IUFD should be confirmed by sonicaid or better by US

Management:

a) Cases with confirmed IUFD are left for vaginal delivery in absence of severe CPD
b) Cases in which the foetus is living are managed according to degree of cervical dilatation:
   - First aid measures as reposition of the cord, with patient in Trendelenburg position
   - If cervix fully *dilated* immediate vaginal delivery is encouraged (sometimes aided by instrumental delivery)
   - If cervix is not fully dilated: immediate CS is the only option

COMPLEX PRESENTATION:

Rarely an arm may present beside the head or buttock during 2nd stage of labour after ROM.

Management: reposition of the arm and allowing labour to continue safely.

Key Points in Breech Presentation

- *Breech presentation is a malpresentation occurring in 3-4% of term deliveries without an identifiable aetiology in most cases, However in other cases uterine anomalies, leiomyomas, MFP, PL PRV and foetal congenital anomalies may be present*
- *In breech presentation it is difficult to assess CPD and if head entrapment occurred during delivery serious foetal and maternal hazards may arise which can all be avoided by CS delivery*
- *ECV can be offered to those cases that are favourable for a trial vertex vaginal delivery*
- *VBD can be allowed for carefully selected cases usually multipara with good obstetric history and average size foetus at term*
- *VBD necessitates attendance of a well experienced team and use of epidural analgesia*
- *The LBW and extremely premature breech (<32 weeks) are best delivered by CS*
- *Delivery of the baby during CS follows the rules of breech extraction and should be slow and gentle*

15 MULTIPLE GESTATION
TWINS, TRIPLETS, AND MORE FOETUSES

- Twin pregnancy
- Triplets and More foetuses

Multifoetal pregnancy **(MFP)** describes pregnancies in which **two** or **more foetuses** are present in the uterus (twins, triplets, quadruplets, quintuplets, etc...).

It may occur either **spontaneously**, with a strong **genetic** and **familial** background, which is the less commonly encountered form, or it may be **iatrogenic induced**, as with the use of multiple ovulation protocols in the management of **infertility**, especially with the use of assisted reproductive techniques **(ART)** as in IUI and ICSI procedures. The latter is the more commonly encountered form with a steadily rising incidence throughout the last 2 decades.

INCIDENCE

Nearly **1-2%** of all pregnancies are MFP. The incidence of iatrogenic twinning has been showing a steady rise in the last decade to **> 3%** due to the use of superovulation protocols in ART.

- **Twins**: is the commonest form occurring in nearly **97%** of MFP.
- **Triplets**: is rare occurring in only **1-2%** of MFP, and is mostly iatrogenic
- **Quadruplets**: very rare occurring in **< 1%** of MFP and is almost always iatrogenic.

OBSTERICAL RISKS IN A MFP

MFP is considered as a high risk pregnancy **(HRP)** being associated with several maternal and foetal risks that increase with the increase in the number of foetuses in utero, including;

1. **Preterm labour (PTL)** and **Preterm PROM:** this is a major risk in MFP. It usually occurs at a mean gestational age of **36** weeks in twins, and even earlier in triplets and quadruplets.
2. **Medical disorders complicating pregnancy;** as hyperemesis gravidarum **(HEG)**, gestational diabetes mellitus **(GDM),** preeclampsia **(PE)** and nutritional anemia (iron and folate deficiency).
3. **Increased CS rates:** due to polyhydramnios, malpresentations, abnormal labour, and placenta praevia. CS rates may exceed **> 50%** in twins and more in triplets and quadruplets.
4. **Other risks** as; spontaneous abortion, low birth weight **(LBW)**, atonic postpartum haemorrhage **(PPH)**, foetal congenital anomalies **(FCA)**, and conjoint twins (see later).

MECHANISMS AND CLASSIFICATION OF TWINNING

A) Fraternal or **Dizygotic twins** (DZT); this is the commoner form of twinning that occurs due to fertilization of two or more eggs in a single cycle (> 1 zygote). They are genetically non identical, of different or same sex, and are always dichorionic / diamniotic **(DC/DA)**, i.e. 2 embryos, each having its own amniotic sac and its own placenta.

B) Identical or **Monozygotic** twins (MZT); this is the less commonly encountered form in which twinning occurs due to splitting of a single embryo (one zygote in which cleavage will occur). They are genetically identical having the same sex and same features. The type of twins will depend upon the time duration between fertilization and cleavage as follows;

- ***Day 1-3;*** **Dichorionic / Diamniotic (DC/DA)** 2 placentae / 2 sacs (30%)
- ***Day 4-8;*** twins are **Monochorionic / Diamniotic (MC/DA)** 1 placentae / 2 sacs (65%)
- ***Day 9-12;*** twins are **Monochorionic / Monoamniotic (MC/MA)** 1 placenta / 1 sac (5%)
- ***After 12 days;*** **conjoined twins** result due to incomplete cleavage division (MC/MA).

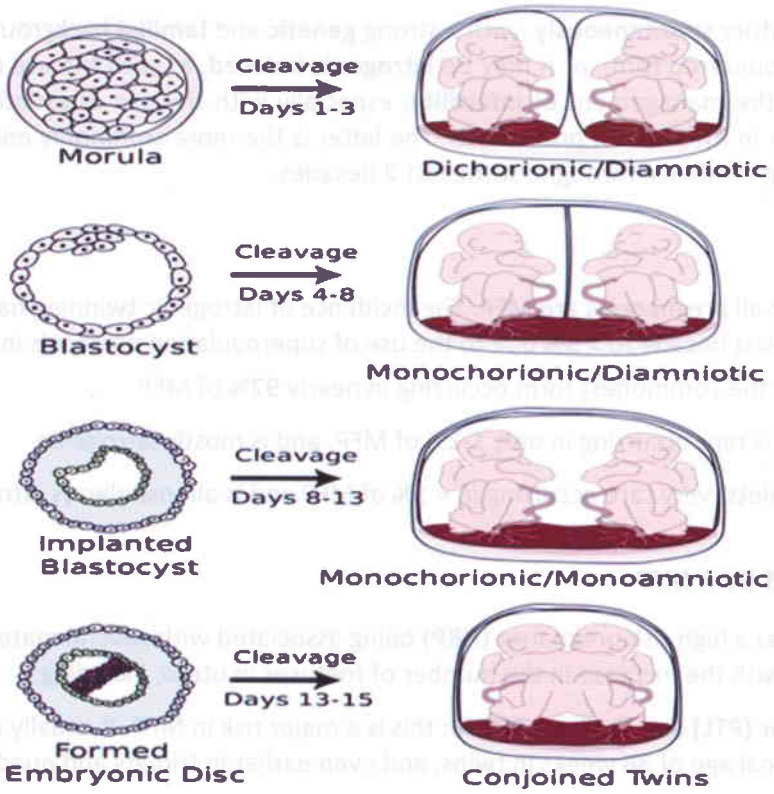


Fig. 15-1: Cleavage effect on type of twin pregnancy

ADDITIONAL RISKS FOR MONOZYGOTIC TWINS INCLUDE

1. **Discordant twins;** twin to twin transfusion syndrome **(TTTS)** may occur in nearly **15%** of MZT sharing the same placenta. The **donor twin** gets less blood supply resulting in intra uterine growth restriction (IUGR), oligohydramnios and anaemia. The **recipient twin** gets more blood supply resulting in excessive growth (macrosomia, polyhydramnios, and polycythaemia).
2. **Conjoined twins:** twins may be adherent at the chest (thoracopagus), head (craniopagus), or sacrum (ischiopagus). Such cases are usually lethal, however in some cases surgical separation and correction may be successful but in many instances sacrificing one baby.
3. **More incidence of CS:** due to polyhydramnios and malpresentations in the first twin, and high incidence of cord prolapse after ROM. If membranes rupture delivery of a second non cephalic twin is more complicated.

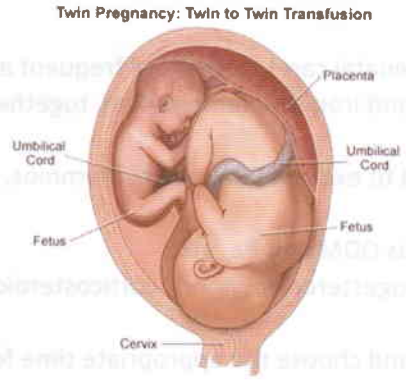


Fig. 15-2 Discordant twins



Fig. 15-3: Conjoined twins thoracopagus and ischiopagus

DIAGNOSIS OF TWINS AND MFP

- Exaggerated **symptoms** of early pregnancy as nausea, vomiting, and HEG
- **Inspection:** abdominal girth larger than expected gestational age in 2nd & 3rd trimesters
- **Palpation:** higher fundal level with multiple foetal poles and limbs felt
- **Auscultation of FHS:** several points of maximum intensity and different FHR
- **TAS:** US is the gold standard for diagnosis of MFP. benefits include
  a. *Estimation of number* of foetuses and the true *gestational age* (GA)
  b. *Determination of zygosity* (MZT or DZT), and *chorionicity* (MC or DC)
  c. *Diagnosis of congenital anomalies (FCA), conjoined twins, PL PRV, and polyhydramnios*
  d. *Evaluation of foetal wellbeing* for each foetus (biophysical profile and Doppler)
  e. *Diagnosis of 2nd twin presentations:* vertex / breech / transverse.



Fig. 15-4: 2D US of DZT



Fig 15-5 3D US of MZT (MC/MA)

DETERMINATION OF ZYGOSITY

Determination of zygosity is the most important step after MFP has been first diagnosed, as it strongly affects the prognosis and the plan of management. US determinants for zygosity include;

DZT	MZT
– *Two separate gestational sacs in 1st trimester*	– *One gestational sac in 1st trimester*
– *Two separate placenta in 2nd trimester*	– *One placenta in 2nd trimester*
– *Same or different gender*	– *Always similar gender*
– *Thick chorionic membrane with Y shaped insertion*	– *Thin membrane with T shaped insertion*

- N.B.; in some cases in babies of the same gender, determination of zygosity may be difficult even after inspecting the placenta after delivery. Such cases may need further HLA typing or DNA analysis to confirm being an identical type of twins.

MANAGEMENT DURING PREGNANCY

MFP is managed as a high risk pregnancy (see HRP in Prenatal care), with more frequent antenatal care visits, improved nutrition, more vitamin, calcium, and iron supplementation, together with;

- *Early and repeated US scans;* to determine zygosity, and to exclude FCA, polyhydramnios, discordant twins, and placenta praevia.
- *Early diagnosis and management of medical disorders;* as GDM and PE, etc...
- *Avoid factors that increase the risk of PTL;* more rest, progesterone support, corticosteroids for lung maturity at 28-20 weeks (see preterm labour)
- *Select patients in which delivery by CS will be indicated* and choose the appropriate time for pregnancy termination.

MANAGEMENT DURING LABOUR

Vaginal delivery is allowed in twin pregnancy whenever the first baby is presenting as vertex presentation irrespective of the second twin, which may be either breech or transverse lie.

A) Delivery of the 1st twin: vertex cephalic presentation (see normal labour)

B) Delivery of the 2nd twin: according to foetal presentation

- *Cephalic presentation:* do artificial ROM and proceed as vaginal delivery
- *Breech presentation:* do artificial ROM and vaginal breech extraction(VBE)
- *Transverse lie:* do either;
  - External Cephalic Version (ECV), ROM, and proceed as cephalic vaginal delivery, **or**
  - Internal podalic version (IPD), ROM, and proceed as vaginal breech extraction (VBE).

C) Immediate postpartum care:

- Ecbolics (Oxytocin, Ergometrin, or Mesoprostol) for prevention of atonic PP haemorrhage due to uterine overdistension
- Exploration of the cervix and vagina to exclude cervical and vaginal lacerations that predispose to guard against traumatic PP haemorrhage
- Antibiotics for prevention of puerperal sepsis associated with internal manipulations during delivery especially of the 2nd twin

INDICATIONS OF CS IN TWINS AND MFP

1. Non vertex presentation of the 1st twin: to avoid locked twins (see later)
2. Triplets & more MFP: delivery of the 2nd & 3rd twins will be unsafe with malpresentations
3. Monoamniotic twins: as more incidence of polyhydramnios, malpresentations, and cord entanglement. ROM is an additional risk for the 2nd twin if transverse lie.
4. Conjoined twins: to avoid obstructed labour, rupture uterus and foetal birth injuries
5. Other indications of CS as: PL PRV, previous CS or myomectomy, associated severe PE or GDM, failed progress of labour, etc...

SPECIAL CONDITIONS RELATED TO MFP

1. **THE VANISHING TWIN:** disappearance of one or more GS in previously diagnosed MFP at early gestation US. Pregnancy usually continues safely with the remaining foetus or foetuses.
2. **THE DISCORDANT TWINS:** in case of MZT due to TTTS (see before), leaving one IUGR foetus and a 2nd LGA foetus compromising the foetal outcome for both foetuses especially the IUGR.

3.DEATH OF ONE FOETUS DURING PREGNANCY:

- **During 1st trimester:** pregnancy usually continues with minimal risks to the mother and to the other foetus but with a higher incidence of abortion and PTL.
- **During 2nd trimester:** pregnancy usually continues without significant hazards except for the increased risk of hypo-fibrinogenaemia, DIC, PROM and PTL. Close observation and early detection of Coagulopathies and prevention of PTL should be encouraged.

4.**THE LOCKED TWINS:** locking of twins during vaginal delivery rarely occurs when the 1st twin is presenting as breech and the second as cephalic or transverse lie. Both heads will be entrapped in the pelvic brim and labour will be obstructed. Emergency CS may salvage the 2nd twin while the first one will be mostly sacrificed.

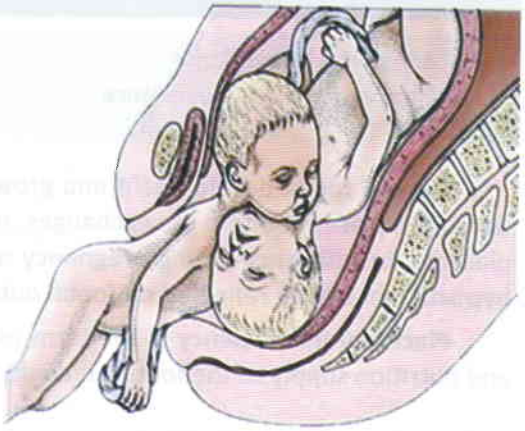


Fig. 15-6: Locked twins

Key points in twins and MFP

- *MFP describes the presence of 2 or more foetuses in utero during pregnancy*
- *It could occur spontaneously mostly genetic based, or induced usually during the course of infertility management with the use of protocols for induction or augmentation of ovulation*
- *MFP is a high risk pregnancy and the risks increase with increased number of foetuses*
- *Risks include PTL, PROM, medical disorders, malpresentations, SGA and IUGR, FCA*
- *In general a MFP is at increased risk for CS reaching > 50% especially if >2 or if MZT*
- *Diagnosis of MFP id best achieved by US which can also accurately detect many problems throughout pregnancy as number of foetuses, gestational age, determination of zygosity, discordant twins, conjoint twins, PL PRV, polyhydramnios, and 2nd foetus presentation during labour*
- *MZT are at higher risks of discordant twins due to TTTS, PTL, PL PRV, FCA, and conjoint twins*
- *Management during pregnancy is directed towards a more strict ANC program, with more frequent ANC visits, investigations, US, vitamin and diet supplementations, and planning for the timing and type of delivery weather vaginal or CS*
- *During labour vaginal delivery is allowed when the 1st twin is presenting by the vertex*
- *Delivery of the 2nd twin needs special skills especially when non vertex presentation*
- *Immediate postpartum care after delivery is of utmost importance*

16 FOETAL SURVEILLANCE ANTEPARTUM AND INTRAPARTUM

- *Antepartum Surveillance*
- *Intrapartum foetal surveillance*

Normal foetal development and growth depends on both **genetic** factors and **placental** functions. The placenta offers gas exchanges, nutrient transfer, and elimination of foetal wastes, hence placental **insufficiency** during **pregnancy** or **labour**, will end up in a state of foetal deprivation and **hypoxia** that will be reflected on foetal outcome and wellbeing **(foetal distress)**.

Placental insufficiency is the term used to describe in-utero failure of adequate oxygenation and nutrition supply to the foetus. It could be either acute or chronic;

1. **Acute Insufficiency:** Suddenly occurring, usually during labour, associated with an otherwise normal foetus, as in;
   - *Cord accidents* as cord prolapse, cord compression, true knots, coils around the neck, and ruptured vasa praevia.
   - Sudden *extensive placental separation* (accidental Haemorrhage, placenta praevia).
   - *Placental compression* (prolonged ROM, uterine hypertonicity, obstructed labour).

2. **Chronic Insufficiency:** Occurring over **weeks** and is usually with intrauterine growth restriction **(IUGR)** and a small for gestational age **(SGA)** foetus, as in;
   - *Idiopathic;* without an explainable aetiology
   - *Postmaturity* syndrome and *placental infarcts*
   - *Medical disorders;* as chronic hypertension, preeclampsia, antiphospholipid syndromes, and diabetic pregnancies associated with vasculopathy
   - *Maternal hypoxia;* as with severe anaemia, excessive haemorrhage, congestive heart failure, severe pulmonary disease and during eclamptic fits.

Foetal Response to Chronic Placental Insufficiency:

1. Decreased foetal movement, due to decreased oxygen supply
2. Oligohydramnios due to reduced renal perfusion.
3. Asymmetric IUGR due to redistribution of blood flow preferentially to the brain and foetal heart known as vascular shift phenomenon (see foetal growth disorders)
4. Disturbance of foetal heart rate due to defective autonomic regulation
5. Abnormal FHR patterns during labour due to severe hypoxia, and academia.

Clinical Assessment of foetal growth during pregnancy can be achieved by recording maternal weight gain, together with the fundal height and width. These measures are only subjective and liable to fallacies especially in obese women, oligo and polyhydramnios, and in multiple gestation

Assessment of **foetal wellbeing** can be achieved through a group of objective tests based on understanding of maternal and foetal physiology, and feto-placental blood flow changes in foetuses exposed to acute and chronic **hypoxia**. This is known as foetal **surveillance** and can be achieved both **antepartum** (during pregnancy), and **intrapartum** (during labour).

ANTEPARTUM FOETAL SURVEILLANCE (APFS)

Antepartum Foetal Surveillance **(APFS)** will allow **identification** of pregnancies at high risk for **placental insufficiency**, and **evaluation** of foetal **wellbeing** to avoid foetal loss.
Foetal surveillance is performed during routine PNC in all pregnancies but is done more frequently in pregnancies at a higher risk for placental insufficiency.

CASES AT HIGH RISK FOR PLACENTAL INSUFFICIENCY

a) Postdate pregnancies
b) Oligohydramnios
c) Intrauterine growth restriction (IUGR)
d) Medical disorders as preeclampsia, hypertensive disorders, and gestational diabetes mellitus
e) Recurrent APH associated with placental abruption or placenta praevia

1. DAILY FOETAL MOVEMENT COUNT (DFMC)

Recording the number of foetal kicks over a period of 1 hour after **30** weeks gestation may help in selecting cases at risk for placental insufficiency, for further evaluation.

- **Interpretation of results:**
  - **More** than **10-12** gross movements within **1** hour indicate **good** foetal wellbeing.
  - **Less** than **10-12** gross movements, need extension of the test to another **1** hour
  - **Decreased DFMC** calls for further tests for evaluation of wellbeing (see later).

2. THE NON STRESS TEST (NST):

Electronic monitoring of **FHR changes** in relation to **foetal movements**, known as the NST, allows evaluation of foetal wellbeing and selecting pregnancies at high risk for acute placental insufficiency during labour.

NST is performed any time in the 3rd trimester using the same **Cardiotogography** (CTG) instrument used for continuous FHR monitoring during labour. A duplex US attached to the mothers abdomen will record **baseline FHR** together with **FHR changes** in relation to **foetal movements** which are reported by the mother by pressing on a special button with each foetal movement.

- **Interpretation of the results:**
  - ***Normal Baseline FHR*** lies between **120-140** bpm
    - ***Normal*** *(reactive foetus):* when FHR **accelerations** of **> 15** bpm, lasing **> 15** seconds occur with each foetal **movement** during **15-20** minutes test duration.
  - ***Abnormal Basal FHR*** < 100 bpm (**bradycardia**), or > 160 bpm (**tachycardia**)
  - ***Abnormal NST*** *(Non-reactive foetus):* when FHR accelerations are absent in relation to movements, or occur by < 15 bpm, or last < 15 seconds.
  - ***Non Reassuring NST:*** when movements are minimal or accelerations are not consistent with each movement.

- **Management according to results:**
  - Reactive NST: repeat after one week
  - Non reassuring NST: extend the period of the test to another 20 minutes
  - Nonreactive NST: immediate Biophysical profile score and Doppler tests

3. THE BIOPHYSICAL PROFILE SCORE (BPPS):

The BPPS is a simple test based on **ultrasound** evaluation of foetal **tone** (flexion / extension), foetal **movements** (limbs and body), foetal **breathing** activities (expansion movements of the abdomen), **amniotic fluid** volume (using AFI), and **placental** maturity **grading** (Grannum score). Each tested parameter is given a **score** (either 0 or 2), with a complete score of **10/10**, in a duration of **30** minutes US examination.

The BPPS is performed any time in the **3rd trimester** and can efficiently pick up those foetuses at risk of hypoxia, and when normal can be repeated every 2 weeks for follow up.

- **Interpretation of the test:** Each item is scored either 0 or 2, with a maximum 10/10.
  - Scores of **10/10** denote a normal foetal wellbeing, with normal foetal PH.
  - Scores of **8/10** may need repeating the test and /or adding Doppler US evaluation.
  - Scores **< 8/10** suggest severe hypoxia. Termination of pregnancy should be considered.
  - Scores **< 6/10** denote severe foetal acidemia, with poor foetal outcome.
- The **modified NST** includes a **BPPS** plus **NST** whether reactive or nonreactive

COLOR DOPPLER STUDIES OF FOETAL BLOOD FLOW

Measurement of the foetal blood flow indices of the foetal **umbilical** artery**, middle cerebral** artery, and **ductus venosus,** yield excellent information about the state of foetal organ perfusion, in addition to US findings of a SGA foetus, asymmetric IUGR, and oligohydramnios.

Colour Doppler Indices Suggestive of Placental Insufficiency:

a) **High** resistance bed for umbilical artery blood flow (increased resistance and pulsatility index)

b) **Low** resistance bed for middle cerebral artery, denoting the vascular shift phenomenon

c) **Absent** or **reversed** umbilical artery end diastolic flow.

d) **Reversed** flow in the foetal ductus venosus.

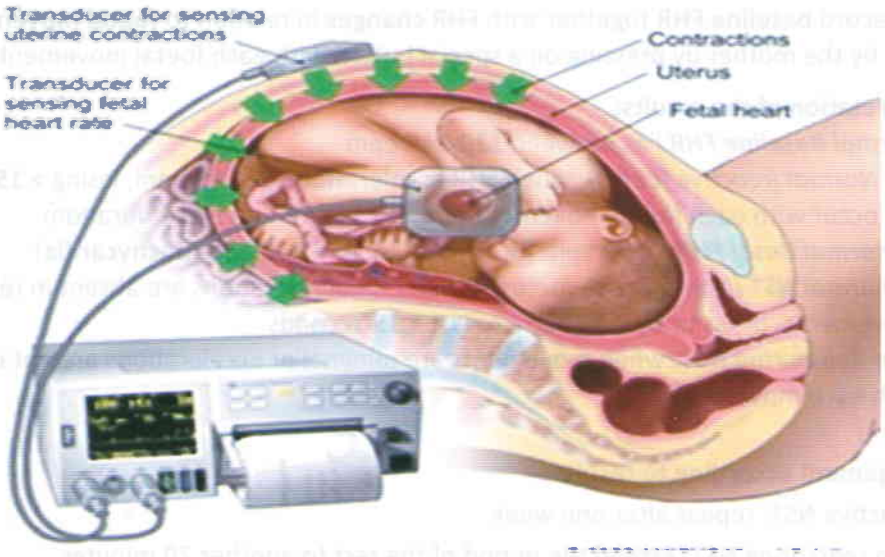


Fig. 16-1: NST using Cardiotogography CTG with electronic FHR Monitoring

INTRAPARTUM FOETAL SURVEILLANCE (IPFS)

Childbirth is a normal **physiologic** process that will usually end **safely** both for the mother and her foetus, however **20-30%** of pregnancies are considered high risk, with **50%** of perinatal morbidity and mortality occurring in this group. High risk cases will requires a close **FHR monitoring** during labour to ensure a good foetal outcome other than traditional FHR auscultation by a Sonicaid.

METHODS OF MONITORING FHR DURING LABOUR

A) AUSCULTATION OF THE FHR

Intermittent auscultation of the FHR by the **Sonicaid** is the standard time honoured technique in evaluation of foetal wellbeing during labour.

- For a **low risk** pregnancy FHR should be listened to every **30** minutes in the 1st stage and at least every **15** minutes in the 2nd stage of labour.
- For a **high risk** pregnancy FHR should be listened to every **15** minutes in the 1st stage and every **5** minutes in teh2nd stage of labour.
- Persistent bradycardia < 100 bpm is an ominous sign for hypoxia and foetal distress

B) CONTINUOUS ELECTRONIC FHR MONITORING

The use of electronic FHR monitoring **(EFM)** during labour is based on the recognition that under hypoxic conditions, the resultant foetal acidosis stimulates **chemoreceptors** and **baroreceptors** in the peripheral arterial circulation that influence the FHR by giving rise to periodic **FHR changes** in relation to uterine contractions.

An external **tocodynamometer** is placed on the abdomen to record the frequency and intensity of uterine contractions, and a duplex **US probe** fixed on the abdomen will continuously record FHR with the results plotted on a screen and a special paper that will show basal FHR together with FHR changes in relation to uterine contractions.

- **Normal Baseline FHR:** 110-160 bpm, **Tachycardia**: > 160 bpm and **Bradycardia**: < 100 bpm
- **Normal Beat-to-beat variability:** fluctuations of +/- 5 to 10 bpm periodically

FOETAL HEART RATE PATTERNS DENOTING FOETAL DISTRESS

- **Decreased beat-to-beat variability:** a prolonged flat baseline with absent beat to beat variability is the result of foetal acidosis
- **Early decelerations (head compression):** slowing of FHR with **onset** of contractions with recovery by its end. Early decelerations are not usually associated with foetal distress,
- **Late decelerations (uteroplacental insufficiency):** slowing of FHR at the **peak** of the uterine contraction, and recovery short after it ends. Severe repetitive late decelerations usually indicate foetal metabolic **acidosis** and low arterial PH.
- **Variable decelerations (cord compression):** slowing of FHR which is not related to uterine contractions. Decelerations have more rapid drop and more rapid return to normal, and are mostly related to transient cord compression especially with oligohydramnios and ROM.

OTHER SIGNS OF FOETAL DISTRESS DURING LABOUR

A) FOETAL TACHYCARDIA

- Foetal **tachycardia** is not usually a reliable sign for foetal distress. It may occur to improve the placental circulation when the foetus is **mildly** distressed
- **Short** periods of tachycardia (15-30 minutes) are sometimes associated with **excessive** oxytocin augmentation of labour

Prolonged periods of tachycardia can be associated with elevated maternal **temperature** of intrauterine **infection**.

B) MECONIUM IN AMNIOTIC FLUID

- *Early passage meconium;* occurs any time before ROM, and varies from light yellowish staining to thick dark green particulate meconium. It is associated with lower 1 & 5 minute Apgar scores and with the risk of meconium aspiration.
- *Late passage meconium;* during the 2nd stage after a period of clear amniotic fluid is usually associated with events as cord compression or uterine hypertonicity that causes foetal distress.
- *In breech presentations;* meconium passage is not reliable since it may occur normally with compression of the abdomen prior to delivery

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 scalp pH ranges from 7.25 to 7.35.
- Intrauterine foetal hypoxia leads to acidosis with foetal pH below 7.25 when mild and below 7.2 with severe. This occurs due to accumulation of CO_2 and lactic acid.

MANAGEMENT OF FOETAL DISTRESS DURING LABOUR

If **CTG** records **abnormal FHR patterns;** the following should be done:

a) Stop oxytocin, if it has been infused (to minimize uterine contractions and decrease intrauterine pressure, thus improving foetal placental perfusion in between contractions)
b) Change position of the mother to left lateral position (allows increased blood supply).
c) O_2 mask to the mother (to avoid maternal hypoxia).
d) I.V. Fluids (to avoid maternal dehydration).

- If FHR patterns return to **normal**, labour is allowed to proceed under strict observation
- If **abnormal** FHR patterns are persistent or recur, immediate delivery is advisable through;
  - **Emergency CS:** this is the safest option whenever cervix is not fully dilated.
  - **Vaginal delivery:** when the cervix is fully dilated and the presenting part engaged, delivery can be encouraged spontaneously or aided by a **low forceps** procedure.
  - **Vaginal breech delivery** can be performed via **breech extraction** in cases of complete or frank breech presentations, with a fully dilated cervix and an adequate pelvis.

KEY POINTS IN FOETAL SURVEILLANCE:

1. *APFS 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 in the 3rd trimester.*
3. *The NST should be offered to cases with decreased DFMC or to high risk cases in general.*
4. *Abnormal NST calls for performing a BPPS and Doppler velocimetry studies.*
5. *Normal NST may be repeated on weekly or biweekly intervals.*
6. *Abnormal NST confirmed by BPPS and Doppler studies necessitate termination of pregnancy.*
7. *Cases at high risk for foetal hypoxia should be selected and carefully monitored throughout labour.*
8. *Cases with normal FHR patterns are allowed to continue their trial for a safe vaginal delivery*
9. *Abnormal FHR patterns are managed according to their severity and impact on foetal outcome*
10. *Persistent late decelerations: indicate serious intrapartum foetal hypoxia*
11. *Persistent abnormal FHR patterns necessitate intervention usually with an emergency CS unless the cervix is fully dilated and vaginal delivery is rapidly anticipated.*

17 ABNORMAL LABOUR

- *Abnormal labour patterns*
- *Precipitate labour*
- *Prolonged labour*
- *Cervical Dystocia*
- *Shoulder Dystocia*
- *Obstructed labour*

ABNORMAL LABOUR PATTERNS

Normal labour describes the process of a spontaneous delivery, of a single living foetus, through the natural birth canal, during an average period of time, without maternal or foetal complications, and without instrumental or surgical intervention.

Prerequisites for such a normal labour usually require an average size foetus, in a cephalic vertex presentation (mostly an Occipito anterior position), after onset of spontaneous labour pains, in absence of contracted pelvis or cephalopelvic disproportion (CPD).

The **average time** expected is estimated to be around **12-18** hours in a **primigravida**, but significantly less in a multigravida. The duration will vary during the latent or active phases of labour from one patient to another according to the parity, gestational age, and pelvic adequacy.

Abnormal labour patterns include precipitate labour, prolonged labour and obstructed labour.

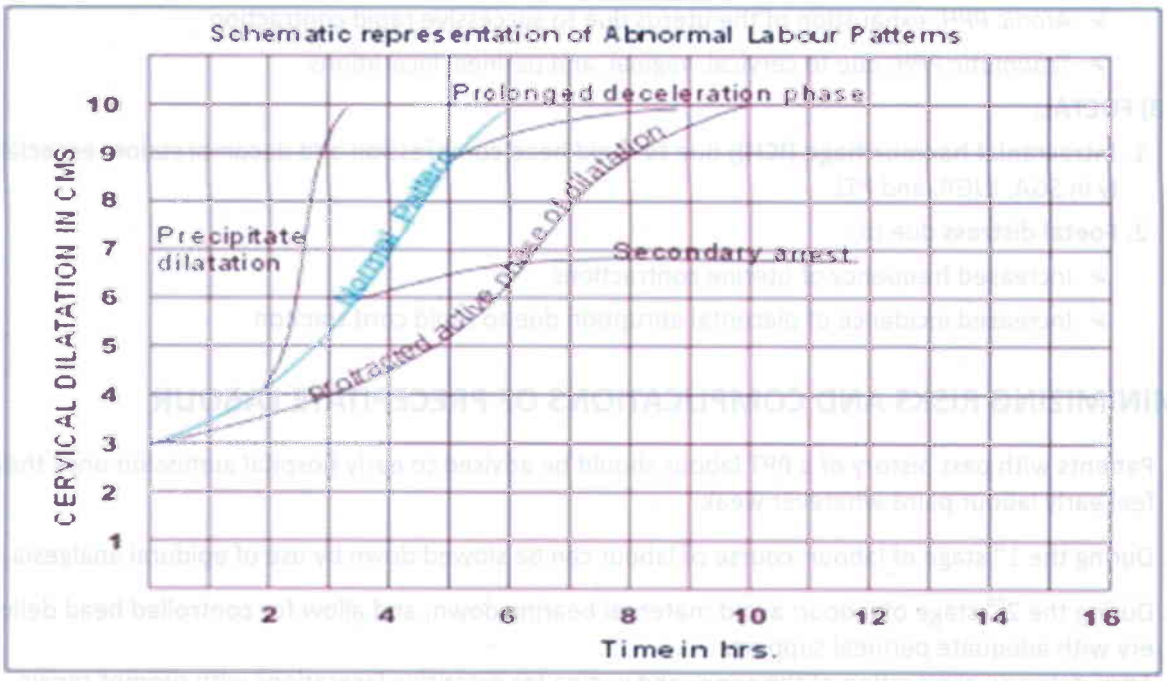


Fig 17-1: Abnormal labour patterns

PRECIPITATE LABOUR

- **Precipitate labour** describes a very rapidly occurring labour of duration **< 4 hours** from onset of labour pains till expulsion of the foetus. It is uncommon, occurring relatively more in multiparas than in primigravidas.
- **Clinical presentation:** Patients commonly present in **late 1st stage** of labour, or even in the **2nd stage**, with a short course labour, in which the patient feels only the last contractions associated with involuntary expulsive forces of the foetus.

AWTIOLOGY AND RISK FACORS

1. Multiparous women with a soft lax birth canal from repeated successive deliveries
2. Previous history of precipitate labour
3. Cervical incompetence (recurrent mid-trimesteric abortions or PTL)
4. Small baby as in intrauterine growth restriction (IUGR) and preterm labour (PTL)
5. Uncontrolled prostaglandin (PGL) induced labour

MATERNAL AND FOETAL RISKS

A) MATERNAL:

1. **Cervical lacerations:** due to rapid dilatation and forced delivery before full cervical dilatation
2. **Vaginal and perineal tears:** due to rapid distension of the birth canal without enough time for full stretch of muscles and ligaments together with uncontrolled head delivery
3. **Postpartum haemorrhage (PPH):**
   - *Atonic PPH:* exhaustion of the uterus due to successive rapid contraction
   - *Traumatic PPH:* due to cervical, vaginal, and perineal lacerations

B) FOETAL:

1. **Intracranial haemorrhage (ICH);** due to rapid head compression and decompression, especially in SGA, IUGR, and PTL
2. **Foetal distress** due to;
   - Increased frequency of uterine contractions
   - Increased incidence of placental abruption due to rapid cord traction

MINIMIZING RISKS AND COMPLICATIONS OF PRECEPITATE LABOUR

1. Patients with past history of a PPT labour should be advised to early hospital admission once they feel early labour pains whatever weak.
2. During the 1st stage of labour: course of labour can be slowed down by use of epidural analgesia.
3. During the 2nd stage of labour: avoid maternal bearing down, and allow for controlled head delivery with adequate perineal support.
4. After delivery; exploration of the cervix and vagina for extensive lacerations with prompt repair.
5. Guard against atonic PPH by use of ecbolics during the 3rd stage of labour
6. Care of the new born; for early diagnosis of foetal asphyxia and birth injuries

PROLONGED LABOUR

- **Prolonged labour** describes a labour of a prolonged duration
  - **> 20** hours in a **primigravida** (PGVDA)
  - **> 14** hours in a **multigravida** (MGVDA)
- **Failed or delayed progress of labour** is a diagnosis based on repeated PV examination throughout labour with findings plotted on a **Partogram,** compared to the norms expected for a normal labour, including cervical dilatation and descent of foetal head
- **Clinical presentation:** according to Partogram during labour

a) Prolonged 1st stage: delayed cervical dilatation and head descent in latent or active phase.

- Delayed or arrested cervical dilatation in the **latent** phase (< 4.0 cm dilatation)
- Delayed cervical dilatation in the **active** phase (> 4.0 cm dilatation)
  - Less than 1.0 cm/hour in PGDA
  - Less than 1.5 cm/hour in MGVDA
- Delayed or arrested descent of the foetal head station.

b) Prolonged 2nd stage: delayed delivery after full cervical dilatation

- **2** hours in a PGVDA
- **1** hour in MGVDA

AETIOLOGY

1. Faults in the **powers**:
   - Inadequate uterine contractions (hypotonic inertia)
   - Uncoordinated uterine contractions (hypertonic inertia and dysfunctional labour)
2. Faults in the **passages**: contracted pelvis and cephalopelvic disproportion (CPD)
3. Faults in the **passenger**: foetal macrosomia, malpresentations (face, brow, breech & shoulder)
4. Cervical dystocia (see later)

MATERNAL AND FOEATL RISKS

1. Maternal **exhaustion**; due to prolonged duration of confinement and labour pains
2. Foetal **distress**; due to prolonged duration of uterine contractions and prolonged use of oxytocin
3. Postpartum **haemorrhage**; due to uterine atony and exhaustion or traumatic difficult delivery
4. Puerperal **sepsis**; due to prolonged rupture of the membranes (ROM), & excessive manipulation
5. Risks of prolonged use of **ecbolics** and **PGL**

MANAGEMENT

a. ***In cases of hypotonic inertia:*** proved by CTG measurements of the frequency and intensity of uterine contractions, labour pains can be **augmented** by the use of **Oxytocin** given as IV infusion drip in escalating doses till adequate contractions are obtained on CTG, and progress of labour is reassured by repeat PV examination.

b. ***In cases of hypertonic inertia:*** with frequent strong uncoordinated uterine contractions, the use of strong sedation (as Morphia), or regional (epidural) analgesia may help normalizing uterine action and improving cervical dilatation and effacement.

c. In cases of marked cephalo-pelvic disproportion (CPD): CS is the proper management

d. In cases of prolonged 2nd stage in absence of CPD:

- If head is engaged; instrumental delivery by the **forceps** or **ventouse** may be used to shorten the 2nd stage and avoid maternal and foetal distress
- If head is not engaged in presence of adequate uterine contractions **CS** will be the best and safest option for delivery to be accomplished without maternal or foetal hazards.

CERVICAL DYSTOCIA

Cervical dystocia describes **failure of** timely **cervical dilatation,** in absence of uterine inertia, leading to a prolonged or arrested 1st stage of labour.

AETIOLOGY:

a. ***Idiopathic** cervical **rigidity;*** of unknown aetiology

b. ***Organic obstruction** of cervical dilatation:* due to pelvic masses or cervical leiomyomas

c. ***Iatrogenic** cervical **fibrosis;*** due to

- Healed lacerations (from previous deliveries, or previous cerclage procedures)
- Following partial amputation: as in (Fothergill's operation) in cases of uterine prolapse
- Following cervical conization (in treatment of high grade CIN lesions).

MANAGEMENT:

- **Sedation** and epidural **analgesia** may help cervical effacement and dilatation in cases of idiopathic or functional cervical rigidity associated with hypertonic uterine inertia
- **CS** in cases with rigid cervix or extensive fibrosis due to stenosis or amputation

SHOULDER DYSTOCIA

Shoulder dystocia occurs when delivery of the foetal shoulder is **delayed** after delivery of the head. It is a **rare** condition complicating **< 1%** of vaginal deliveries.

Shoulders are usually **entrapped** in the anteroposterior diameter **(APD)** of the cavity with the anterior shoulder impacted behind the symphysis pubis **(SP)**. The head recedes backwards shortly after its delivery (turtle sign), and labour may be obstructed.

Foetal macrosomia is the most important risk factor especially those with a large abdominal circumference (AC) / head circumference (HC) ratio. It is mostly encountered in association with uncontrolled gestational diabetes mellitus **(GDM)** and foetal **hydrops** (immune of nonimmune).

Although shoulder dystocia is usually attributed to a large for gestational age **(LGA)** foetus, yet **50%** occur with foetal birth weight **< 4.0** kg.

RISKS AND COMPLICATIONS

1. **Foetal asphyxia and IUFD:** due to brain hypoxia associated with delayed delivery
2. **Foetal birth injuries:** Attempts at delivery of the impacted shoulder may be associated with fracture clavicle, fracture humerus and or brachial plexus injury (Erb's palsy).
3. **Maternal birth injuries:** due to rough intrauterine manipulations including rupture uterus, cervical, and vaginal lacerations.

PREVENTION AND PREDICTION

In many cases it is difficult to predict occurrence of shoulder dystocia to avoid its complications, however the following measures may be of help;

1. Proper assessment of foetal weight before delivery by clinical and US evaluation
2. Proper control of maternal GDM especially in 3rd trimester
3. CS should be offered to LGA and cases with established macrosomia

MANAGEMENT DURING LABOUR

During labour, once shoulder dystocia is suspected (turtle head sign), the following maneuvers are immediately applied in succession;

1. *Mc Roberts maneuver:* maternal thigh flexion with suprapubic pressure. This is the most effective and successful procedure with best possible outcome.
2. *Wood's cork screw maneuver:* internal rotation of foetal shoulders to oblique plane
3. *Manual delivery of the Posterior arm*
4. *Zavinelli maneuver:* cephalic reposition back into vagina and CS.

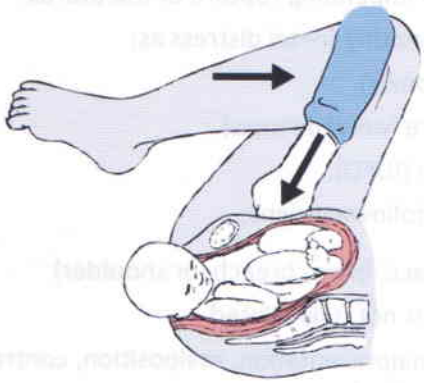


Fig. 17-2: McRoberts manoeuvre



Fig. 17-3: supra pubic pressure

OBSTRUCTED LABOUR

Obstructed labour describes failure of delivery of the foetus due to **mechanical** obstruction.

AETIOLOGY

1. *Contracted pelvis* and marked degrees of *Cephalo-pelvic Disproportion (CPD).*
2. *Foetal macrosomia* and extremely *LGA foetus* > 4.5 kg.
3. *Non corrected malpresentations and malpositions* as:
   - Persistent oblique and transverse Occipito Posterior positions (OPP).
   - Persistent oblique and transverse face Mento Posterior (MP), and direct face MP.
   - Brow and shoulder presentation.
   - Breech impacted in the pelvic inlet.
4. *Soft tissue obstruction* by cervical and uterine leiomyomas, and large ovarian swellings.
5. *Cervical dystocia* (see before).
6. *Locked twins* (rarely when 1st twin is breech and second cephalic or transverse lie)
7. *Contraction ring:* very rarely contraction at any uterine segment will obstruct delivery

CLINICAL PICTURE AND DIAGNOSIS

A) HISTORY: of prolonged labour with long ROM in presence of adequate contractions.

B) GENERAL EXAMINATION: Signs of maternal exhaustion and dehydration including; tachypnea (rapid respiration), tachycardia (rapid pulse), hypotension (low BP), oliguria, and dry tongue.

C) ABDOMINAL EXAMINATION:

- ***Palpation:*** tense tender uterus with persistent tonic contractions (except if ruptured).
- ***Pathological Retraction ring (Bandl's ring);*** it is a transverse groove that can be seen and felt abdominally at the demarcation between the upper and lower uterine segments. It shows a rising level upwards towards the umbilicus in cases of impending rupture of the uterus.
- ***Auscultation:*** FHS usually show abnormal patterns denoting foetal distress as;
  - ➢ Persistent Late Decelerations (denoting foetal hypoxia)
  - ➢ Persistent bradycardia < 100 bpm (denoting severe foetal distress).
  - ➢ Inaudible FHS in cases of intrauterine foetal death (IUFD).

D) VAGINAL EXAMINATION may reveal one or more of the following signs:

- ➢ *Non engagement of the presenting part* (vertex, face, brow, breech, or shoulder)
- ➢ *Oedematous cervical rim* may still be felt if cervix is not fully dilated
- ➢ *The cause of obstructed labour* may be elicited (malpresentation, malposition, contracted pelvis, CPD, cervical dystocia, or soft tissue obstruction)
- ➢ *Caput succedaneum* in vertex cephalic presentation due to scalp oedema

- **N.B.** *caput succedaneum may give a false impression of head engagement where the scalp is felt low in the vagina while the skull bone is still at a higher non engaged level (-2, or -3).*
- **N.B.** *Bandl's ring should be differentiated from a full bladder or a uterine leiomyoma*

Differentiation between Retraction ring and Contraction ring

Pathological Retraction Ring	Contraction Ring
– *Occurs with prolonged 2nd stage*	– *Occurs during any stage of labour*
– *Lies always between UUS and LUS*	– *Occurs at any level*
– *Rises upwards towards umbilicus*	– *Does not change its position*
– *Felt and seen abdominally*	– *Not seen, and only felt vaginally*
– *Uterus is tense and tender*	– *Uterus not tonically retracted*
– *Maternal and foetal distress common*	– *Distress may not be present*
– *Relieved only by delivery*	– *Relieved only by deep anaesthesia*

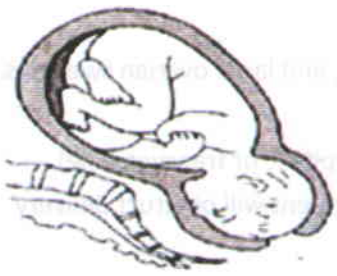


Fig. 17-3: Pathological retraction ring



Fig. 17-4: Contraction ring

COMPLICATIONS OF OBSTRUCTED LABOUR

1. Complications of prolonged labour; maternal exhaustion, foetal distress, ROM, chorioamnionitis, neonatal asphyxia, and IUFD
2. Rupture of the uterus which may necessitate laparotomy for repair, or hysterectomy
3. Cervical, vaginal, and perineal lacerations: from traumatic manipulation
4. Necrotic vesico-vaginal fistula (VVF): due to prolonged head compression on the bladder
5. Puerperal sepsis: due to prolonged ROM and traumatic manipulations

MANAGEMENT

1. **Emergency CS** is the best and **safest** option even in cases with IUFD to avoid rupture of the uterus
2. Instrumental delivery (obstetric forceps or ventouse) for cephalic presentations should be **discouraged** as the head is not engaged and attempts for delivery may be complicated by rupture of the uterus, with high maternal morbidity and mortality from bleeding and sepsis.
3. *Cervical dystocia & shoulder dystocia* are managed as previously described (see before)

18 CEPHALOPELVIC DISPROPORTION AND CONTRACTED PELVIS

- Contracted pelvis
- Cephalopelvic disproportion test

CONTRACTED PELVIS

From the **clinical** point of view a **contracted pelvis** is a pelvis in which one or more of its **main diameters** are **reduced** to the extent that **interferes** with **normal** delivery.

AETIOLOGY OF CONTRACTED PELVIS

1. **Idiopathic** small pelvis: gynaecoid, android, anthropoid, etc...
2. **Metabolic disease**: as Rickets and Osteomalacia
3. **Trauma** or **diseases affecting pelvic bones:** as fractures, tumours, and TB joints
4. Diseases of the **spine:** severe dorso-lumbar kyphoscoloiosis, and spondylolisthesis
5. Diseases of **lower limbs (LL)**: hip dislocation, fracture femur, poliomyelitis, etc...

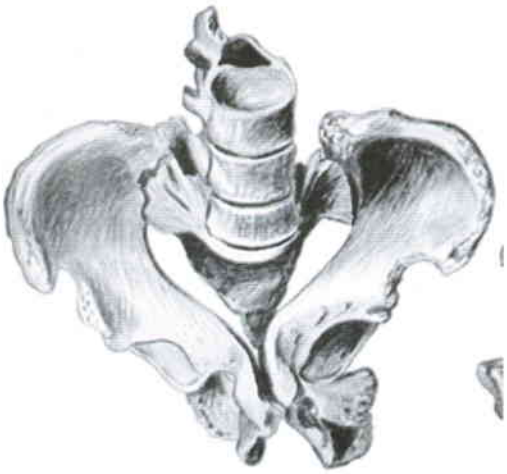


Fig. 18-1: Osteomalaecic pelvis

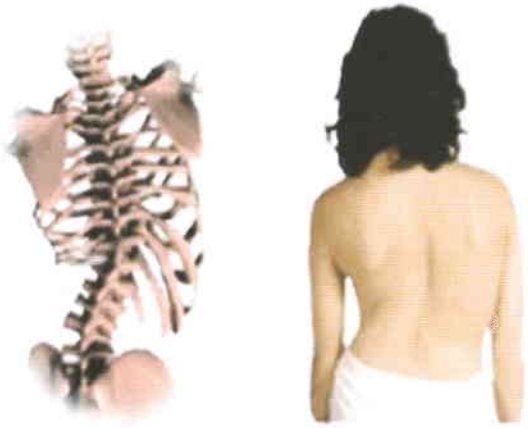


Fig. 18-2: Scoliosis

DIAGNOSIS OF CONTRACTED PELVIS

A) HISTORY:

- History of pelvic trauma or disease of the spine or LL (see aetiology)
- History of previous prolonged difficult or obstructed labour (suggestive)

B) GENERAL EXAMINATION:

- Short stature < 150 cm, limping and abnormal gait, spine and LL deformities
- Malpresentations as face, brow, an transverse lie (contracted inlet)
- Non engagement of the head during the 1st stage of labour

C) CLINICAL EXAMINATION OF THE PELVIS:

Digital PV examination of the pelvis in early 1st stage of labour entails palpation of;

1. The **sacrum** (flat in android, curved in gynaecoid pelvis)
2. The **side walls** of the pelvis (converging in android and anthropoid pelvis)
3. The **sacro-sciatic notch** (normally wide admits 2 fingers)
4. The **ischial spines** (jutting in android pelvis)
5. The sub-pubic angle (normally accommodated 2 fingers)

D) CLINICAL PELVIMETRY OF THE PELVIS

1. ***The Sub-pubic angle:*** obtuse and ischio-pubic ramus accommodates 2 fingers
2. ***The Bituberous diameter (11 cm):*** (transverse diameter of the outlet) roughly admits the 4 knuckles of the closed fist of the hand.
3. ***The Diagonal conjugate DC (12.5 cm):*** **DC** is measured between the lower border of the SP and the promontory of sacrum. It is 1.5 cm larger than the true conjugate TC



Fig 18-3: Bituberous diameter



Fig. 18-4: Diagonal conjugate

E) RADIOLOGIC PELVIMETRY

Lateral view **X-ray** and **CT scan** can accurately measure pelvic dimensions (pelvimetry). However it is seldom used in modern obstetrics which focuses on cephalopelvic proportion and disproportion rather than metric pelvic measurements.

CEPHALOPELVIC DISPROPORTION TESTS

Cephalopelvic disproportion **(CPD)** tests are **clinical** tests to assess possibilities of the foetal head to be delivered vaginally in **primigravida**, based on the concept that **the head is the best Pelvimeter.**

1. PINARD'S METHOD:

- Patient in semi-sitting position with bladder empty
- The operator with his **left hand** grasps the **foetal head** and tries to push it downwards and backwards in the pelvis along the pelvic axis
- The **fingers** of the operator's **right hand** placed over the **SP** can determine the degree of disproportion and weather head overrides SP or passes smoothly through the inlet

2. MULLER-KERR METHOD

- Patient in dorsal position with bladder empty
- The operator performs a digital **PV** examination with the fingers of the **right hand** performing the steps of internal **clinical pelvimetry**
- The **thumb** of the **right hand** is placed over the **SP** to determine the degree of disproportion if present
- The operator with his **left hand** grasps the **foetal head** and tries to push it down through the pelvic inlet with the thumb of the right hand in over the SP trying to evaluate the presence and degree of CPD

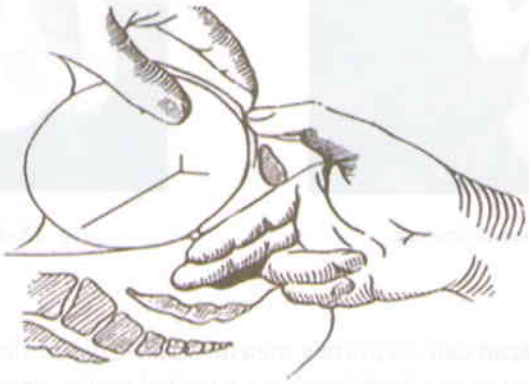


Fig. 18-5: Muller- Kerr test for CPD

INTERPRETATION OF THE RESULTS OF CPD TESTS

A) No CPD: the head can be pushed into the maternal pelvis passing behind the SP

B) Moderate CPD (1st degree):

- The head **stops** at the same level of the **SP**.
- Vaginal delivery is possible after **trial labour** (testing undetermined factors of labour and allowing for molding of the head and yielding of the pelvis) **C) Marked CPD** (2nd degree):
- The head **overrides** the anterior surface of the SP.
- Vaginal delivery cannot occur and trial labour should not be allowed and **CS** performed

19 OBSTETRIC TRAUMA DURING LABOUR AND DELIVERY

- Rupture of the uterus
- Cervical lacerations
- Vaginal lacerations
- Perineal lacerations

RUPTURE OF THE UTERUS

Rupture of the uterus is one of the most **life-threatening** obstetric emergencies that may lead to serious maternal morbidity, or even mortality due to massive **obstetric haemorrhage**.

In many cases rupture of the uterus can be avoided or prevented by adequate antenatal care, proper selection of high risk cases, and adequate intrapartum care during labour and delivery.

TYPES OF RUPTURE

A) Complete Rupture: describes complete tear of **all layers** of the uterine musculature with the foetus being **totally** or **partially extruded** within the peritoneal cavity.

B) Scar Dehiscence: describes separation of the uterine musculature at the site of a **previous LSCS** scar that includes **all** layers **except** the visceral peritoneum. The foetus will thus be kept in utero with no foetal parts extruded into the general peritoneal cavity.

INCIDENCE

- **Complete Rupture:** over all incidence **< 2:1000** deliveries depending on the level of obstetric care.
- **Rupture LSCS scar:** during trial Vaginal Birth after CS (VBAC) has a risk of **2:1000** to **9:1000**
- **Rupture USCS scar:** The risk increases 10 times to reach **1-2%**
- **Scar Dehiscence risk** in a previous LSCS scar is nearly **1%** in late 3rd trimester or during labour.

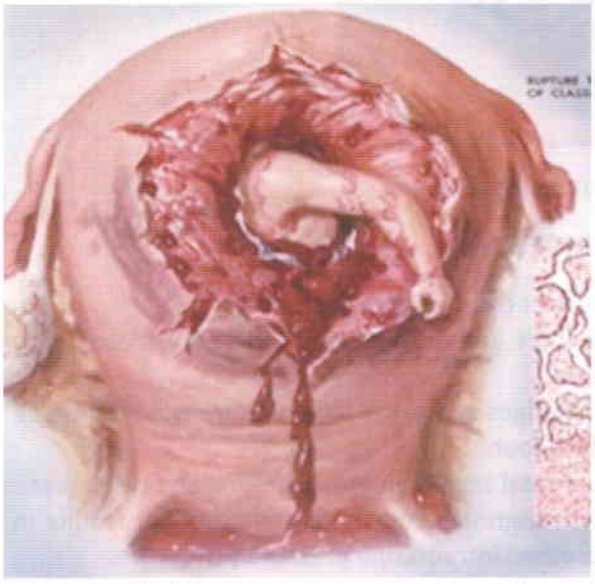


Fig 19-1: Rupture UUS CS scar

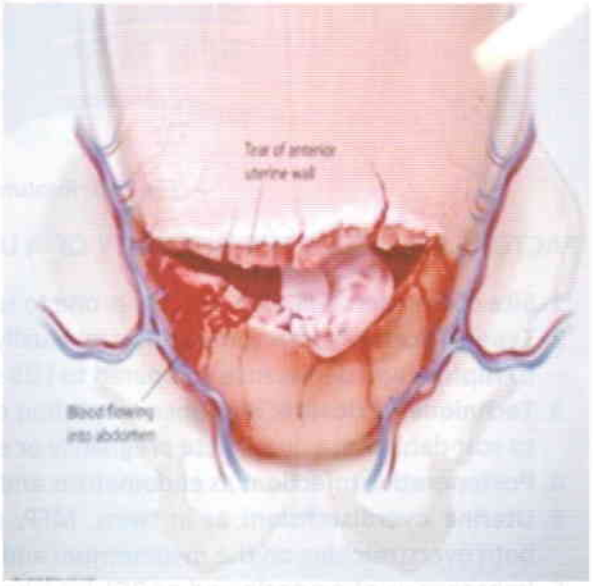


Fig 19-2: Rupture LUS CS scar

RISK FACTORS

1. UTERINE SCAR:
- *Upper uterine segment:* as USCS, myomectomy, and scar of a healed uterine perforation
- *Lower uterine segment:* as LSCS and myomectomy scar
- *Old cervical scar:* as old healed cervical lacerations, cervical myomectomy & repeat cerclage.

2. GRAND MULTIPARA: (> 5 deliveries): due to
- *Weaker and thinner* uterine musculature; due to repeated deliveries
- *Tendency to larger foetus* due to multiparity and possible gestational DM
- *Pendulous abdomen* with tendency to malpresentations and malpositions
- *Tendency to a narrower pelvis* due to possible calcium deficiency & acquired Osteomalacia.

3. TRAUMATIC RUPTURE:
- *External Trauma:* as car accidents, fall from heights, and stab wounds
- *Rough Obstetric manipulations:* as during external cephalic version for breech presentation, internal podalic version for a transverse lie, difficult forceps procedures, and manual separation of the placenta,

4. OBSTRUCTED LABOUR: due to
- ➢ Contracted pelvis and marked cephalopelvic disproportion
- ➢ Malpositions and Malpresentations as in OP, Face, Brow, and Transverse lie, etc...

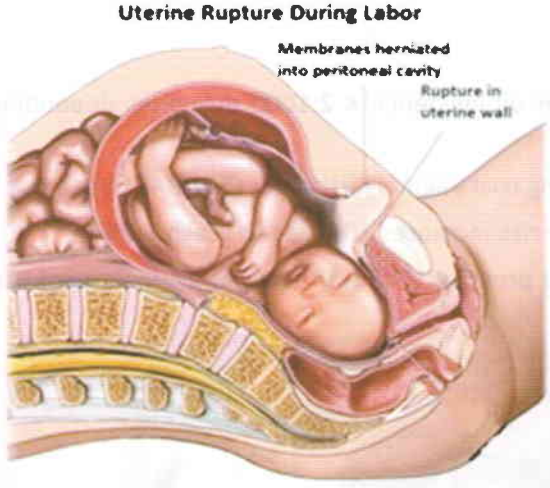


Fig 19-3: Rupture LUS during labour

FACTORS AFFECTING THE INTEGRITY OF A UTERINE SCAR

1. **Site of the scar:** LUS scars are less prone to rupture and dehiscence than UUS scars.
2. **Type of scar:** Myomectomy scars are usually multiple and mostly at UUS, thus are more prone to rupture and dehiscence compared to LUS scars.
3. **Technique of closure:** improper coaptation of the edges and single layer closure will predispose to scar dehiscence during late pregnancy or early labour.
4. **Postoperative infection:** as endometritis and puerperal sepsis with poor healing and weak scar
5. **Uterine overdistension:** as in twins, MFP, polyhydramnios and foetal macrosomia, results in both overstretching on the myometrium and increased intrauterine pressure (IUP).
6. **Improper us of oxytocin and or PGL** for labour induction or augmentation especially in presence of undiagnosed CPD or unknown prior uterine scar.

DIAGNOSIS

A) SYMPTOMS

- Sudden severe abdominal **pain** followed by cessation of uterine contractions
- Sudden onset of vaginal **bleeding** that may be severe
- Sudden cessation of **foetal movements** (usually associated with IUFD)

B) SIGNS

- **General Examination:** may reveal signs of **hypovolemic shock** due to rapid blood loss
- **Abdominal Examination:** may reveal easily palpable foetal parts just below abdominal wall, abnormal foetal lie, severe foetal bradycardia or absent FHS.
- **Vaginal Examination:** recession of the presenting part to a higher pelvic station or even to an abdominal position. Vaginal bleeding is usually severe.

C) ABDOMINAL ULTRASOUND

- Confirms an abnormal foetal lie and detect presence or absence of FHR to exclude IUFD

MANAGEMENT OF RUPTURE OF THE UTERUS

1. RESUSCITATION AND ANTISHOCK MEASURES:

This is the ***first step*** prior to any surgical intervention in cases with massive blood loss.

2. EXPLORATORY LAPAROTOMY AND SURGICAL REPAIR:

Aiming at arresting uterine bleeding, repairing the torn myometrium, and reforming the uterus back to its normal configuration. Successful pregnancy usually occurs after adequate surgical repair however, delivery by ***CS will be mandatory*** for fear of rupture uterine scar.

3. ABDOMINAL HYSTERECTOMY:

Hysterectomy is indicated whenever rupture of the uterus is extensive beyond proper repair, and bleeding is severe risking the life of the patient, especially in elderly grand multigravidas with no desire for future fertility.

CERVICAL LACERATIONS

AETIOLOGY

1. **Delivery** of the foetal presenting part before **full** cervical dilatation
2. **Rapid passage** of the foetal head through the birth canal
3. **Delivery** through a previously **scarred cervix**

RISK FACTORS

1. **Precipitate labour:** due to rapid cervical dilatation and rapid delivery of the head
2. **Preterm delivery:** the small head may be delivered through an incompletely dilated cervix
3. **Excessive maternal bearing down** during 1st stage of labour
4. **Delivery of the after coming head in breech:** before full cervical dilatation
5. **Difficult Instrumental delivery:** forcible forceps or ventouse application and traction especially before full cervical dilatation (wrong application)
6. **Excessive use of oxytocin or PGL:** leading to rapid dilatation & excessive expulsive forceps.
7. **Previous scarring of the cervix:** as in previous cerclage procedures, cervical amputation, cervical conization, or previously repaired cervical lacerations during previous deliveries.

DIAGNOSIS (See traumatic post partum haemorrhage)

1. Inspection: excessive vaginal bleeding immediately after delivery of the foetus or placenta
2. Abdominal examination: fundus of the uterus is well contracted
3. Vaginal examination: cervical tear is sometimes palpable on PV examination
4. Exploration of the cervical lips under good light using vaginal speculum and ring forceps to visualize the cervical laceration and start immediate repair

MANAGEMENT

Surgical repair to stop bleeding and restore normal anatomy (see figures)

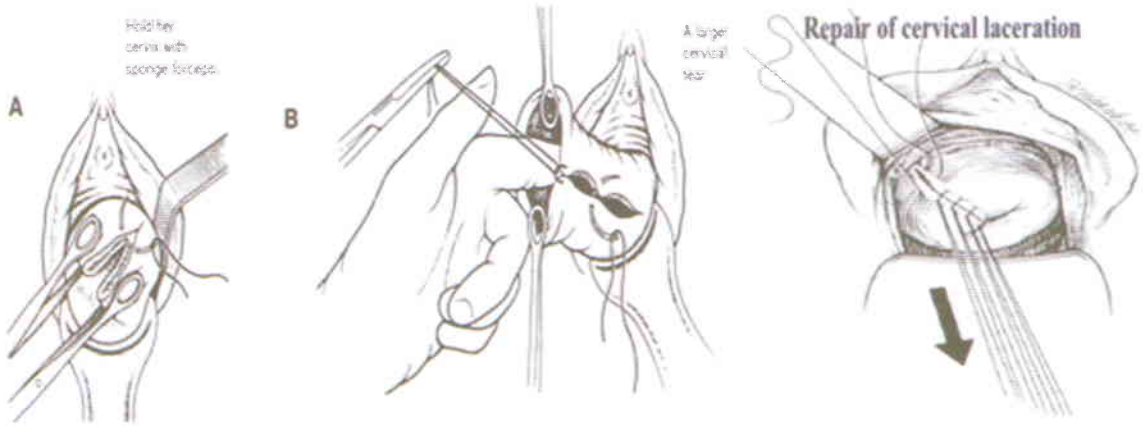


Fig 19-4: Repair of cervical laceration

VAGINAL AND PERINEAL LACERATIONS

Most vaginal deliveries will be associated with minor vaginal tears that do not necessitate surgical repair without causing late side effects or complications. However severe lacerations must be promptly dealt with as they may cause serious morbidity and late complications.

AETIOLOGY

1. **Precipitate labour:** due to the rapid passage of head through the birth canal, without adequate perineal support (Ritgen's manoeuvre).
2. Delivery of an **LGA or** a **macrosomic foetus** without an adequate episiotomy
3. **Instrumental delivery** especially in absence of an adequate episiotomy
4. **Rigid perineum** with tight vaginal and strong perineal muscles

CLASSIFICATION OF TYPES OF LACERATIONS

A. **First degree tears:** involve only **vaginal mucosa**

B. **Second degree tears:** involve the vagina and the muscles of the **perineal body**

C. **Third degree tears:** involve the vagina, perineal muscles, and the **anal sphincter**

D. **Fourth degree tears:** involve the vagina, perineal muscles, anal sphincter, and **anal mucosa**

MANAGEMENT

A. **First degree tears:** suture repair is often not needed except if severe bleeding is present

B. **Second degree tears:** suture repair is necessary to avoid disfiguring and painful scar

C. **Third degree tears:** layered suture repair is mandatory to avoid anal incontinence

D. **Fourth degree tears:** layered suture repair is mandatory and if not properly managed may lead to anal incontinence and or recto-vaginal fistula formation.

20 COMPLICATIONS OF THETHIRD STAGE OF LABOUR

- *Postpartum haemorrhage (PPH)*
- *Retained Placenta*
- *Obstetric Shock, DIC, and hypofibrinogenaemia*

The **3rd stage** of labour is the stage of **delivery** of the **placenta**. Its duration is variable ranging from **10-30** minutes. Adequate **myometrial contractility** is the key to successful placental separation and expulsion. It also acts as a physiologic-anatomic ligature that controls postpartum blood loss from dilated myometrial arterioles and decidual veins after placental separation.

The most common **obstetric complications** encountered in the **3rd stage** of labour include postpartum haemorrhage **(PPH), retained placenta** and rarely **uterine inversion.** These complications may be associated with haemorrhagic shock, DIC, and hypofibrinogenaemia.

POST PARTUM HAEMORRHAGE

PPH is defined as excessive blood loss after delivery that affects the patients' general condition. It is estimated to be around **> 500 ml** following vaginal delivery and **> 1000** ml following CS.

The **general condition** of the patient is affected by severable factors including;

- The **amount** of blood loss
- The **Rate** of blood loss and the **duration** of bleeding
- The presence of a **prior medical condition** as; maternal anaemia, cardiac, renal, and hepatic disease, hypertension, preeclampsia, and bleeding disorders.
- Genetic factors and vitamin D deficiency may also play a role in the severity of PPH.

CAUSES OF PPH

1. Uterine **Atony**
2. Genital tract **Trauma**
3. **Coagulation disorders;** inherited or acquired (ITTP, von Willebrand disease, DIC & Hypofibrinogenemia)
4. **Uterine inversion** (very rare)

N.B. in many cases PPH is multifactorial including both trauma and atony that may be accentuated by presence of retained placental fragments, Placenta praevia, placental abruption or coagulation disorders

CLASSIFICATION OF PPH

A) IMMEDIATE or PRIMARY PPH: occurring **immediately** after delivery, and throughout the first **24** hours postpartum. It may complicate **0.5-4%** of all deliveries, and is mostly related to uterine atony, genital tract trauma, retained placenta, and coagulation disorders

B) DELAYED or SECONDARY PPH: This is excessive bleeding occurring any time during the puerperium starting > 24 hours after delivery. It is less commonly encountered and is mostly associated with retained placental fragments, and puerperal sepsis.

AETIOLOGY AND RISK FACTORS IN PPH

A) UTERINE ATONY:

Uterine **atony** is the **most common** cause for **1ry PPH** encountered in **> 75 %** of cases. It occurs due to absence of the physiologic-anatomic ligature effect of the contracting uterine musculature on the myometrial arterioles and decidual veins, resulting in uncontrolled placental site bleeding.

- ***Risk Factors for ATONIC PPH:***
  - History of *previous atonic PPH* (possibly genetic or familial)
  - *Grand multipara* with 5 or more deliveries (laxity in uterine musculature and larger fetuses)
  - *Overdistension* of the uterus as in twins & MFP, polyhydramnios and foetal macrosomia
  - *Myometrial Exhaustion* in prolonged & precipitate labour, and improper use of oxytocin
  - *Placenta Praevia (PL PRV)* because of the relatively deficient muscular content of the lower uterine segment (LUS) compared to that of the upper segment
  - *Placental abruption* (concealed & mixed types) where retroplacental haematoma formation dissects in-between myometrial layers interfering with contractility
  - *Large uterine leiomyomas* (interfere with efficient myometrial contractility)
  - *Chorioamnionitis* and intrauterine infection (weak uterine muscle)
  - *Severe maternal anemia* and *vitamin D deficiency* (poor contractility)

B) GENITAL TRACT TRAUMA:

Trauma during labour and delivery is the **2nd most common** cause for **1ry PPH,** mostly in association with traumatic cervical, vaginal, and perineal **lacerations**, or **rupture** of the uterus;

- ***Risk Factors for TARUMATIC PPH:***
  - *Previous uterine scar;* CS, myomectomy, or healed perforation during D&C
  - *Traumatic delivery of an oversized baby* as in macrosomia and shoulder dystocia
  - *Insufficient episiotomy;* especially with a large foetal head and rigid perineum
  - *Delivery before full cervical dilatation* as in precipitate labour and preterm breech
  - *Difficult instrumental delivery* as mid-forceps procedures

C) RETAINED PLACENTA or PLACENTAL FRAGMENTS:

This is the **3rd common** cause for **1ry** PPH and the **leading** cause for **2ry PPH,** where retention of the whole placenta or undelivered placental fragments impairs myometrial contractility

- ***Risk factors for RETAINED PLACENTA;***
  - *Uterine atony;* leading to incomplete separation and failure of expulsion
  - *Adherent placenta;* as focal or partial placenta accreta
  - *Rupture uterus;* where placenta may be expelled in peritoneal cavity
  - *Contraction ring;* preventing placental expulsion (very rare)

D) COAGULATION DISORDERS: these are **rare** but may be **fatal** causes of PPH (see obstetric shock);

- *Idiopathic thrombocytopenic purpura (ITP)* associated with abnormal platelets or shortened life span
- *Von Willebrand disease* (factor VIII deficiency)
- *Hypofibrinogenemia and DIC* due to consumption coagulopathy associated with placental abruption or prolonged IUFD.
- *Amniotic fluid embolism:* a rare condition associated with 80% mortality where a significant amount of amniotic fluid escapes into maternal circulation after rupture of the membranes, resulting in fulminating consumption coagulopathy, intense bronchospasm, and vasomotor collapse (see obstetric shock).

E) ACUTE UTERINE INVERSION:

Uterine inversion is a condition in which turning inside out of the uterus may occur in the 3rd stage of labour. It is very **rarely encountered** occurring in < 1: 20.000 deliveries.

- ***Risk factors for UTERINE INVERSION;***
  - *Vigorous Pressure* on the soft uterine fundus from *above*
  - *Excessive Traction* on the cord of a partially adherent placenta from *below*
  - *Fundal submucus leiomyoma,* of moderate size, facilitates dipping of the uterine fundus into the uterine cavity by pressure of its weight and uterine contractions.

DIAGNOSIS OF A CASE OF PPH

PPH is a **clinical diagnosis** based on clinical evaluation of the **amount** of bleeding and **rate** of blood loss occurring after delivery of the foetus, and its effect on the patient's **general** condition.

- **General** examination and **vital signs** will diagnose hypovolaemic **shock** if present and its degree.
- Abdominal **palpation** of the uterine fundus will diagnose uterine **atony** and subinvolution.
- **Speculum** vaginal examination will reveal genital tract **lacerations** by naked eye inspection.
- **Digital vaginal** examination allows for exploration of the uterine cavity for **retained** fragments, rupture of the uterus, or dehiscence of a CS scar.
- **Ultrasonography** is the gold standard for diagnosis of retained placental cotyledons. It can also detect intraperitoneal haemorrhage and broad ligament haematoma in uterine rupture.
- **Laboratory tests** are important for evaluation of the effect of the amount of bleeding on the blood picture, the severity of hypovolaemic shock, and the diagnosis of **coagulopathies**.

CLINCAL PICTURE AND EXAMINATION

A) ATONIC PPH;

- The uterus is **soft** and **hypotonic** with a rising fundal level due accumulating haematometra
- Speculum examination reveals **dark** placental site bleeding pouring through the cervix.
- Ultrasonography shows an enlarged uterine cavity distended only with blood

B) TRAUMATIC PPH;

- The uterus is **firm** in consistency with **good** contractility
- Speculum examination shows profuse **fresh** bright **red** bleeding from extensive cervical, vaginal, or perineal lacerations.
- Digital exploration of uterine cavity may reveal uterine rupture or scar dehiscence

C) RETAINED PLACENTAL OR PLACENTAL FRAGMENTS;

- *Subinvoluted uterus* with a high fundal level and inefficient contractility
- *Inspection* of the delivered placenta will show *missing cotyledons*
- Digital exploration of the uterine cavity through the cervix reveals retained contents
- *Ultrasonography* will show retained placental tissue in utero confirming diagnosis

D) ACUTE UTERINE INVERSION;

- Abdominal palpation of a fundal depression due to cupping of the fundus into the cavity
- Vaginal examination reveals palpation of the inverted fundus through the internal os
- Speculum examination reveals a mass dilating the cervix and protruding to the vagina

MANAGEMENT OF A CASE OF PPH

A) RESUSCITATION AND CORRECTION OF HYPOVOLAEMIC SHOCK if present:

- Insertion of a wide bore IV cannula and urinary catheter
- Continuous monitoring of Vital signs (pulse, temp, BP, & RR) and urine output
- IV infusion with saline/dextrose or lactated Ringers solution
- Blood or blood components reserved for emergency

B) MANAGEMENT OF ATONIC PPH:

1. **ECBOLICS** immediately started to induce myometrial contractility in as follows;
   - ***Oxytocin IV infusion***; Syntocinon 20 u /500 ml saline by rapid infusion
   - ***Ergometrine IM***; Methergin 2.5 mg if atony persists
   - ***Prostaglandin (PG) F2 a***; Carboprost 250 ug IM
   - ***Prostaglandin (PG) E1;*** Misoprostol 800 ug transrectal (4 tablets 200 ug each))

2. **UTERINE MASSAGE** starts together with ecbolic medications as follows;
   - ***External Uterine Massage*** to stimulate uterine contractility by gentle continuous massage of the fundus by the palm & 4 fingers of the left hand with the thumb anterior
   - ***Bimanual uterine compression*** by placing the closed fist of one hand vaginally and the other hand abdominally squeezing the uterus between both hands

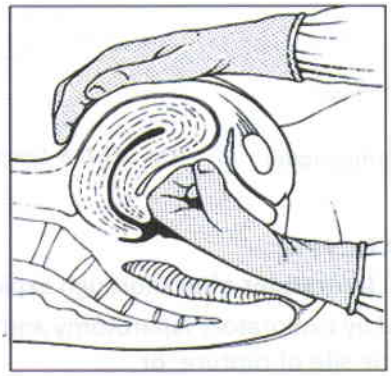


Fig 20-1: Bimanual uterine compression

3. **EXPLORATION OF THE GENITAL TARCT**;
   - ***Inspection*** of the ***perineum, vagina***, and ***cervix*** for extensively bleeding lacerations.
   - ***Exploration of the uterine cavity;*** digitally by 2 fingers introduced through the dilated cervix to detect retained placental fragments, ***rupture*** of the uterus or ***dehiscence*** of a prior CS scar.

4. **INTERVENTIONAL METHODS:** If the above methods fail to control atonic one of the following interventions may be of help in the following sequence without delay;
   - ***Intrauterine Balloon Catheter*** (Bakry's balloon) inflated at an adequate pressure to induce *internal tamponade* to stop bleeding from atonic uterus
   - ***Uterine Arteries Embolization;*** in haemodynamically stable patients with persistent moderate bleeding an interventional radiologist may insert percutaneous catheters through the femoral artery with retrograde injection of thrombogenic material to block the uterine arteries and minimize bleeding.

5. **LAPAROTOMY:** is kept as the **last final** resort in haemodynamically unstable patients not responding properly to the previous measures. Steps are done as follows in sequence;
   a) **Rupture of the uterus** is managed primarily by surgical repair and reconstruction. If failed supracervical hysterectomy is performed as an emergency procedure.
   b) **Intact myometrium with no rupture or lacerations;**
      - **B-lynch sutures** embracing the uterus may control bleeding by acting as an *external tamponade* (see Fig....)
      - ***Bilateral uterine arteries ligation*** to minimize uterine blood flow and stop placental site bleeding
      - ***Bilateral internal iliac artery ligation*** to decrease pelvic blood flow and venous pressure minimizing the amount and rate of blood loss.
      - ***Subtotal (supracervical) or Total hysterectomy;*** with ovarian preservation may sometimes be a last step performed as a lifesaving procedure.



Fig. 20-2: Bakry's balloon (internal tamponade)

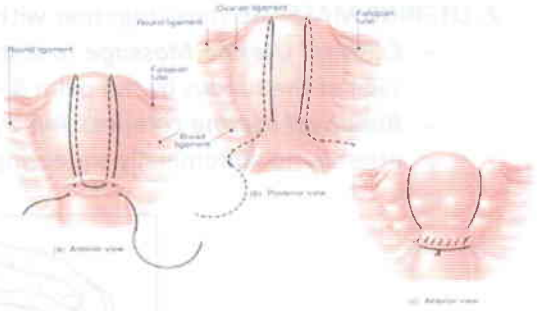


Fig. 20-3: B-Lynch sutures (external tamponade)

C) MANAGEMENT OF TRAUMATIC PPH:
- Primary **suturing** of genital **lacerations** after thorough exploration to arrest bleeding
- Rupture uterus is managed by exploratory laparotomy and either;
  - **Anatomical repair** of the site of rupture, **or**
  - **Hysterectomy** if damage is beyond repair or patient extremely haemodynamically unstable.

D) MANAGEMENT OF RETAINED PLACENTAL FRAGMENTS
- **Manual separation** and delivery of the placenta under anesthesia if retained placenta
- **Digital** removal of placental **fragments** through the open cervix under anesthesia
- **Surgical** evacuation of uterine cavity under anesthesia using **ovum** or **ring** forceps

E) MANAGEMENT OF ACUTE INVERSION OF THE UTERUS
- *Resuscitation* and management of haemorrhagic and/or neurogenic shock
- *Reposition* under anesthesia (manual or hydrostatic reposition)
- Management of *chronic inversion* of the uterus (see book essentials of gyneacology)

COMPLICATIONS OF PPH

1. Complications of severe hypovolaemic shock (acute renal failure, DIC, ...)
2. Complications of massive blood transfusion (DIC, DVT, pulmonary embolism, ...)
3. Complications of acute blood loss (Sheehan's syndrome)
4. Maternal morbidity (hysterectomy), and mortality in case of delayed management.

SECONDARY POST PARTUM HAEMORRHAGE

Delayed or Secondary **PPH** describes **excessive** vaginal bleeding commencing **> 24 hours** after delivery that may occur or recur any time throughout the **puerperium** (6 weeks after delivery).

AETIOLOGY

1. **Retained placental tissue** and fragments is by far the most common cause (> 50%)
2. **2ry Haemorrhage** from cervical, vaginal, or perineal lacerations
3. Sloughing of an infected **submucus fibroid polyp (SMFP)**
4. Undiagnosed **chronic inversion** of the uterus
5. **Choriocarcinoma** (rare). See diagnosis and management in early pregnancy bleeding

DIAGNOSIS

- **History:** severe bleeding within the period of puerperium
- **Inspection:** may reveal infected lacerations, SMF Polyp, or chronic inversion of the uterus
- **Palpation:** the uterus may be subinvoluted due to retained blood and tissue fragments
- **Ultrasound:** may diagnose retained placental tissue, placental polyp, or SMF Polyp

MANAGEMENT treat the cause;

a. **Ecbolics** may help to extrude and expel some retained placental tissue
b. **Surgical evacuation** under anesthesia using ring or ovum forceps for retained missed placental fragments or cotyledons.
c. **Secondary sutures** in cases with gaping vaginal or perineal lacerations
d. **Chronic inversion of the uterus** is treated surgically (see book Essentials of Gynaecology),

RETAINED PLACENTA

Retained placenta describes a condition in which the placenta **fails to be delivered** within **30 minutes** after delivery of the foetus. It may complicate **0.5%** of vaginal deliveries.

AETIOLOGY

A) FAILURE OF EXPULSION after placental separation due to:

- Uterine atony
- Contraction ring
- Rupture of the uterus

B) FAILURE OF EXPULSION due to a **MORBIDLY ADHERENT PLACENTA (MAP)**

This is a rare condition in which the decidua basalis is totally or partially defective leading to direct adherence and/or invasion of the chorionic villi into the myometrium. Absence of a plane of cleavage in such cases interferes with placental separation. There are **3 types** of morbidly adherent placenta;

a. **Placenta Accreta:** chorionic villi become **adherent** to the myometrium (most common type)
b. **Placenta Increta:** chorionic villi **invade** into the myometrium (less common type)
c. **Placenta Percreta:** chorionic villi **penetrate** through the myometrium reaching serosa and may invade the bladder wall (rarest and most serious type)

CLINICAL PICTURE

Failure of spontaneous placental expulsion within **30 minutes** from delivery of the foetus, in-spite of attempts at gentle cord **traction** and suprapubic pressure.

The clinical picture will vary according to underlying aetiology whether associated with uterine **atony** or **rupture** of the uterus (see atonic and traumatic PPH). **Rarely** placental retention is due to;

- **Contraction Ring;** vaginal examination with 2 fingers of one hand inside the uterus through the dilated cervix will reveal the condition. Severe PPH is usually present
- Morbidly **Adherent Placenta (MAP)**; bleeding is usually **not severe** because no placental separation has occurred. Digital exploration through the open cervix will reveal absent plane of cleavage between the placenta myometrium. Attempts at manual removal of an adherent placenta may provoke severe PPH.

MANAGEMENT OF RETAINED PLACENTA

1. **Uterine Massage and Ecbolics** to promote contractility and facilitate expulsion, ***if failed*** ,
2. **Controlled Cord Traction and Suprapubic Pressure** *(Brandt Andrew's method),* ***if failed,***
3. **Manual separation of the placenta** under anaesthesia by introducing one hand inside the uterus creating a plane of cleavage separating the placenta plus continuous cord traction
4. **Dealing with retained placental fragments**;
   - *Digital evacuation* by 2 fingers exploring the uterine cavity through the cervix, **or**
   - *Surgical evacuation* under anaesthesia using a ring or ovum forceps
5. If due to **Contraction ring**; use deep general Halothane anaesthesia to relax the uterus followed by steps of manual separation and cord traction.

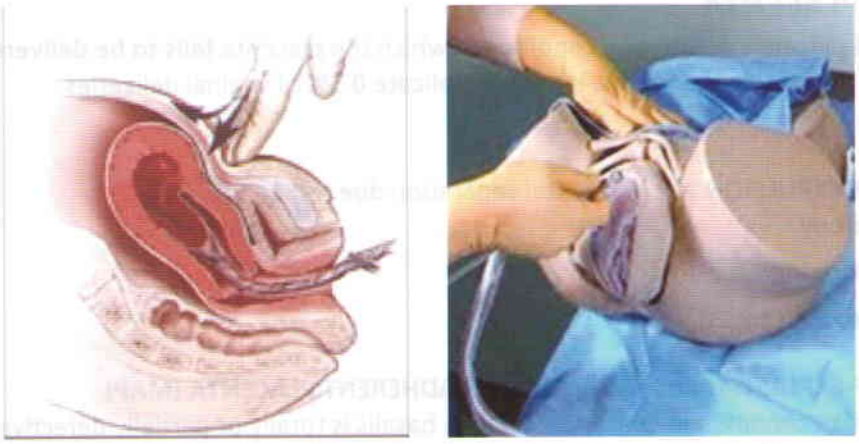


Fig. 20-5: Brandt- Andrew's method of controlled cord traction and suprapubic pressure

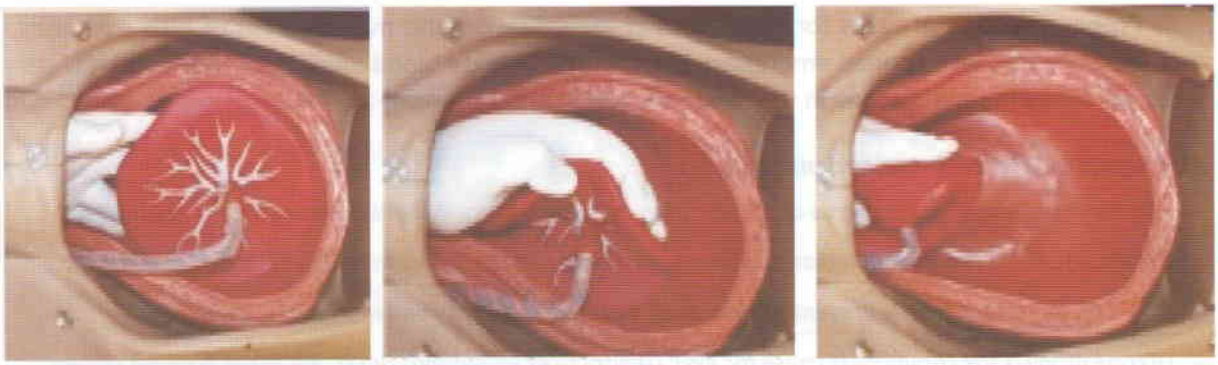


Fig. 20-6: Manual removal of the placenta 1, 2, &3

MANAGEMENT OF MORBIDLY ADHERENT PLACENTA

A) **Focally Adherent** Placenta is managed by gentle **manual separation** under anaesthesia with prompt use of uterine massage and ecbolics

B) Placenta **Accreta, Increta,** and **Percreta** are best managed by an immediate postpartum abdominal hysterectomy to avoid intractable bleeding during useless attempts of separation. This major surgical procedure usually requires an experienced team supported by vascular surgery and urology team. Almost all cases of MAP occur in association with a placenta praevia implanted on a scar of prior repeated CS. Diagnosis should be made antepartum, and cases managed electively by **CS hysterectomy** with adequate blood transfusion facilities.

C) Conservative management: in cases of MAP diagnosed for the first time during performing a CS for PL PRV, if facilities are not available for a CS Hysterectomy with adequate blood transfusion, the upper segment uterine incision may be closed and placenta left in situ without attempts for removal, and the patient transferred postoperatively to a tertiary center that can deal with such a complication

COMPLICATIONS OF RETAINED PALCENTA

1. Atonic PPH
2. Obstetric shock
3. Retained fragments with 2ry PPH
4. Puerperal sepsis due to infection in retained necrotic tissue and attempts at manual removal

PREVENTION AND MINIMIZING COMPLICATIONS OF PPH can be accomplished via;

a. Proper antenatal care and selection of cases with high risk factors (see antenatal care)
b. Proper management of 1st and 2nd stages of labour (see management of normal labour)
c. Active management of 3rd stage of labour (see management of normal labour)
d. Proper inspection of placenta after delivery (to detect a missing cotyledon)
e. Proper use of oxytocin and ecbolic drugs during labour and immediately after delivery
f. Avoid prolonged labour and difficult instrumental delivery
g. Selective use of episiotomy and ensure proper repair
h. Prompt diagnosis and management of traumatic lesions causing PPH

PROGNOSIS

PPH is one of the major causes of maternal **morbidity** and a leading cause of **maternal mortality** in Egypt and many developing countries. Improving obstetric care services and obstetric skills will directly lead to significant decrease in both mortality and morbidity in PPH.

Proper selection of cases for VBAC, proper identification and management of high risk cases, and presence of a well-trained emergency team with adequate blood banking facilities and ICU services will result in rapid decline in the incidence and complications of 1ry PPH.

Key points in PPH and retained placenta

- *PPH describes blood loss in excess of 500 c.c. after vaginal delivery, and more than 1000 c.c. following C.S.*
- *It is classified as primary (immediate), or secondary (delayed) according to timing of its occurrence*
- *PPH is an important mostly preventable cause of maternal morbidity and mortality*
- *Uterine atony is the commonest cause for PPH .Genital tract lacerations or DIC are other possible causes.*
- *Abdominal palpation of the uterus can differentiate atonic from traumatic PPH.*
- *Proper management of the third stage of labour is very important in prevention of PPH.*
- *First aid treatment of 1ry PPH is massage and ecbolics, with exclusion of traumatic lacerations and retained placental fragments.*
- *If bleeding is severe and uncontrollable, subtotal hysterectomy may be lifesaving.*
- *PPH cannot always be prevented for it occasionally occurs when conditions are in all respect normal.*
- *In cases with retained placenta uterine atony is usually the commonest aetiology*
- *Defective placentation and abnormal adhesions is the most dangerous cause.*
- *Complications include Shock, Postpartum haemorrhage, Puerperal sepsis and Retained placental fragments*
- *Management according to aetiology either by gentle massage , ecbolics ,controlled cord traction and manual removal in cases of atony or inhalation anaesthesia in contraction ring or hysterectomy in abnormal adhesions.*

OBSTETRIC SHOCK

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 OBSTETRIC SHOCK

1. Hypovolemic shock: secondary to Bleeding (bleeding in early pregnancy, APH, PPH).

- Other causes of fluid loss (e.g. nasogastric suction or diarrhoea).

2. Early neurogenic & Septic shock

HAEMORRHAGIC SHOCK

A healthy pregnant woman can lose 25% of her blood volume (1500 ml) before clinical signs of shock are evident.

Conditions that increase risk of shock in obstetrics include:

a) Moderate & Severe Anaemia and malnutrition
b) Bleeding in early pregnancy, APH, or PPH
c) Prolonged labour 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 haemorrhage 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.

MANAGEMENT OF HYPOVOLEMIC 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 haemorrhage 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.

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) Inotropic Agents:

- **Inotropic drugs:** improve cardiac muscle contractility (e.g.; Dopamine & Dobutamine)

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)	*Hypofibrinogenemia*	*Factors VIII, VWF, XIII, fibronectin, and fibrinogen*	*Supplies selected clotting factors*
Platelets (50 ml)	*Bleeding from thrombocyto-penia*	*Platelets*	*Increases platelet count by 5000-8000/µl per unit*

SEPTICEMIC SHOCK

AETIOLOGY:

As pelvic infection is polymicrobial, septic shock may be caused by;

1. Endotoxin producing enterobacteriaceae 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) n 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		*Thrombocytopenia, Leucocytosis, DIC*

TREATMENT OF SEPTIC SHOCK

1. Aggressive fluid replacement. Oxygenation and ventilation.
2. Administration of Vasopressor and Inotropic agents (see hypovolemic shock).
3. Broad spectrum antibiotics.
4. Removal of the infectious source.

N.B. Steroids and NSAID: are not beneficial.

Immunotherapy is still under research.

NEUROGENIC SHOCK

AETIOLOGY: trauma and tissue damage as in cases of:

1. Disturbed extrauterine pregnancy.
2. Concealed accidental haemorrhage.
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 labour and polyhydramnios
9. Retained placenta especially for more than 2 hours.

Differences between neurogenic and haemorrhagic shock:

Neurogenic shock	Haemorrhagic 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*
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.

DIC IN OBSTETRICS

(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.
2. Massive crystalloid or colloid replacement
3. Placental abruption.
4. 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 37ºC. 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 obstetric shock and DIC

- Types include Hypovolaemic, Distributive shock, Cardiogenic shock and Obstructive shock.
- Remote complications of shock in obstetrics include Renal failure and Sheehan syndrome.
- Crystalloid solutions as lactated Ringer's solution or normal saline for acute hemorrhage.
- Colloid therapy (as plasma substitutes) will provide more volume expansion than crystalloids.
- Packed RBCs are usually used in Obstetric hemorrhagic shock.
- Early septic shock is a form of distributive shock while in late stages it is both distributive and cardiogenic.
- Clinically septic shock Passes into 3 stages of increasing severity namely systemic inflammatory response syndrome (SIRS), severe sepsis then septic shock.
- *Pregnancy is a hyper-coagulable state with increased concentration of coagulation factors.*
- *Disseminated Intravascular Coagulation is characterized by accelerated fibrin formation and lyses.*
- *Common causes include Massive blood loss, Placental abruption and Severe pre-eclampsia/eclampsia.*
- *Rare causes include Sepsis, Retained dead fetus, Amniotic fluid embolism and Acute fatty liver.*
- *Accelerated coagulation occurs via the extrinsic pathway.*
- *Treatment must be directed to the underlying cause to reverse defibrination.*

21 ANTEPARTUM HAEMORRHAGE AND INTRAPARTUM HAEMORRHAGE

- *Placental abruption*
- *Placenta Praevia*
- *Vasa Praevia*
- *Abnormally Invasive Placenta*

ANTEPARTUM HAEMORRHAGE

Antepartum hemorrhage **(APH)** is the term used to describe bleeding occurring in the **second half** of pregnancy from the time of foetal **viability** till **delivery** (i.e.; bleeding between **24-40** weeks).

Bleeding in **APH** is mostly **maternal** in origin, and when severe may directly affect maternal vital signs and general condition before affecting foetal wellbeing. Less commonly bleeding may be **foetal** in origin rapidly affecting foetal wellbeing before significant maternal affection.

Severe APH represents one of the most acute **obstetric emergencies** that remains a leading cause of maternal morbidity and mortality especially in developing countries, inspite of the improvements in antenatal care services.

CAUSES AND DIFFERENTIAL DIAGNOSIS OF APH

1. OBSTETRIC CAUSES: (detected by ultrasound examination)

- *Placental abruption:* bleeding from separation of a normally implanted placenta
- *Placenta Previa:* bleeding from separation of an abnormally implanted placenta
- *Vasa Previa:* bleeding from the umbilical cord (see later)

2. GYNECOLOGICAL CAUSES: (detected by speculum examination)

- *Vaginal trauma;* as bleeding after sexual intercourse
- *Vaginal lesions;* as HPV is not uncommon
- *Benign cervical conditions:* as erosion or mucus polyps
- *Cervical cancer* during pregnancy is a rare yet possible cause

3. GENERAL OR SYSTEMIC CAUSES: (detected from medical history of the patient)

- *Anticoagulant therapy:* using drugs as Heparin, Clexane, and oral anticoagulants
- *Idiopathic thrombocytopenic purpura (ITP)*
- *Hypertension (HTN)* only if severe

N.B.: *Vaginal bleeding should be always differentiated from;*

- Urinary tract bleeding; as bleeding in association severe UTI or renal stones
- Rectal bleeding; as in cases of bleeding piles

PLACENTAL ABRUPTION

Placental abruption describes bleeding due to **separation** of a normally implanted placenta on the upper uterine segment (UUS). It may complicate up to **1%** of all pregnancies, and is considered the most common cause for antepartum and intrapartum haemorrhage. In 2/3 of cases abruption is classified as severe based on associated maternal, foetal, and neonatal morbidity.

Bleeding from placental abruption, also known as **accidental haemorrhage,** significantly affects foetal wellbeing and is an important cause for intrauterine foetal death (IUFD), preterm labour (PTL), intrapartum haemorrhage (IPH), intrapartum foetal bradycardia and distress, and may be associated with a serious maternal coagulopathy and DIC.

RISK FACTORS

1. Maternal **hypertension** and severe preeclampsia **(PE);** due to defective placentation and rupture of spiral arterioles secondary to vasospasm with elevated BP
2. **Sudden ROM:** in cases with polyhydramnios due to associated rapid decompression
3. **Traumatic abruption;** may occur due to external trauma as car accidents, fall from height or direct hit on the abdomen.
4. **Smoking:** increases the risk significantly relevant to the number of cigarette packets
5. **Thrombophilias,** Factor V Leiden and prothrombin gene mutations, have been associated with IUGR, PE, IUFD, placental infarcts, and placental abruption.

TYPES OF BLEEDING IN PLACENTAL ABRUPTION

1. **Revealed type:** where blood dissects its way between the placental membranes to the internal os, to be revealed externally through the vagina (this is the commoner type).
2. **Concealed type:** blood remains entrapped forming a retro-placental haematoma with no clinically detectable external vaginal bleeding (least common type).
3. **Mixed type:** where bleeding is partially concealed and partially revealed (the 2nd most common type). Unlike with PL PRV, the amount of vaginal bleeding in mixed type of placental abruption does no correlate with severity of blood loss and degree of placental separation.

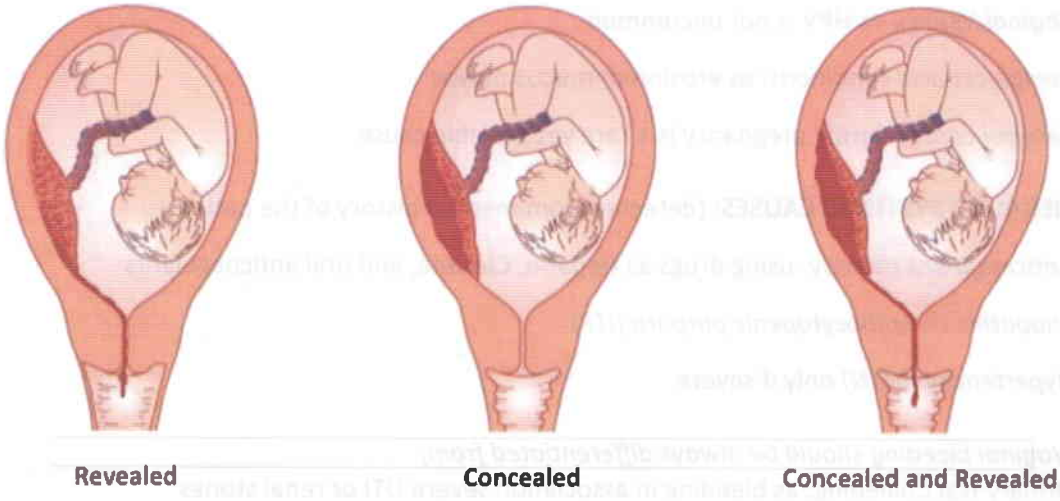


Fig 21-1: Clinical types of placental abruption

SYMPTOMS & SIGNS

A) **Revealed** Haemorrhage (commonest type): patients usually present with a mild to moderate attack of vaginal **BLEEDING** which is usually associated with mild to moderate lower abdominal sharp stabbing type of **PAIN**.

B) **Concealed** Haemorrhage (rarest type): bleeding may be absent, but **PAIN** and **FOETAL DISTRESS** may be significant (foetal bradycardia).

C) **Mixed** or **Combined** Haemorrhage: both **BLEADING** and **PAIN** are present however the amount of vaginal bleeding and intensity of abdominal pain may **not correlate** with the general condition of the patient nor with associated foetal bradycardia if present.

GENERAL EXAMINATION:

- ***Mild placental separation and bleeding:*** does not affect the vital signs or the general condition
- ***Severe placental separation and bleeding:*** is usually associated with **haemorrhagic shock.**
  - The degree of shock may not correlate with the amount of external bleeding in cases with combined or concealed haemorrhage.
  - Hypotension may be masked by the essentially elevated BP in cases with pre-eclampsia (PE)
  - DIC may occur in cases with concealed haemorrhage with large retroplacental haematoma.

ABDOMINAL EXAMINATION:

- *Uterine tenderness* is usually present with severity related to the degree of abruption and amount of retroplacental bleeding.
- A *rising fundal level* may be noticed in cases with an accumulating retroplacental hematoma in cases with concealed accidental hemorrhage.
- **N.B.: Vaginal examination** could be performed ONLY after EXCLUSION of PL PRV by US (as DD being the other common cause of APH)

ULTRASOUND DIAGNOSIS:

- TAS is a gold standard in identifying placental site and hence **exclusion** of PL PRV
- TAS can detect a large retroplacental **haematoma** in cases with combined or concealed HGE

EVALUATION OF MATERNAL AND FOETAL WELLBEING

- **Maternal Vital signs:** pulse, temperature BP, and respiratory rate
- **Laboratory tests:** CBC, liver & kidney function tests, in addition to coagulation profile
- **Assessment of fetal wellbeing** through FHR tracing, CTG, and Doppler US.

PATHOLOGY AND COMPLICATIONS OF SEVERE PALCENTAL ABRUPTION

1. **Hemorrhagic shock** which might be complicated by acute renal tubular necrosis.
2. **Sheehan syndrome** (acute pituitary necrosis) may also occur as a result of hemorrhagic shock.
3. **DIC** due to thromboplastin release from the damaged muscle fibres and consumption of clotting factors in the retroplacental hematoma (hypofibrinogenaemia)

4. **Couvelaire's uterus:** in cases of concealed HGE when entrapped blood in a retroplacental haematoma dissects its way within myometrium to reach the serosal surface leading to bruises like lesions with failure of uterine contractions and atonic postpartum hemorrhage.
5. **Foetal Complications:** include foetal distress, severe IUGR, IUFD, preterm delivery with high neonatal morbidity and mortality especially in cases of severe concealed accidental hemorrhage.

MANAGEMENT

Once diagnosed, placental abruption will be managed **actively** by immediate termination of pregnancy, or **expectantly** by conservative prolongation of pregnancy according to:

- Severity of bleeding and type of APH (revealed, concealed, or mixed)
- Effect of APH on maternal condition and foetal well being
- Gestational age at the onset of APH
- Onset of true labour pains

A) ACTIVE MANAGEMENT by IMMEDIATE DELIVERY in cases with;

1. *Severe bleeding* compromising maternal or foetal lives, **regardless** gestational age
2. *Gestational age > 37 weeks* **regardless** amount of bleeding
3. *Spontaneous onset of labour pains,* regardless amount of bleeding and gestational age

B) EXPECTANT MANAGEMENT to DELAY DELIVERY in preterm pregnancies < 37 weeks to allow more time to achieve better lung maturity whenever:

1. *Bleeding is mild* not affecting maternal or foetal well being
2. *Labour pains* are absent

Expectant management is continued until any indication for active management arises (lung maturity achieved, onset of labour pains, or an attack of severe APH).

- Vaginal delivery is attempted in cases with mild to moderate bleeding whenever possible
- CS is indicated whenever bleeding is severe affecting maternal or foetal wellbeing, or in presence of any other obstetric indication for CS (Prior CS, CPD, Breech presentation, etc...)

N.B.: Non sensitized **Rh-ve** mothers will deserve an **Anti-D immunoglobulin** injection during their expectant period of management.



Fig 21-2: Couvelair's uterus in concealed accidental haemorrhage

PLACENTA PRAEVIA

Placenta praevia **(PL PRV)** describes **implantation** of the placenta partially or totally on the lower uterine segment **(LUS)**. It is classified according to its relation to the internal cervical os into **total (central), partial**, and **marginal** PL PRV. The nearer the placental margin to the internal os, the more will be the liability to earlier and more severe type of **APH**.

PL PRV is the 2nd **most common** obstetric cause of **APH,** being preceded only by placental abruption. However it is considered the **most common** cause of **severe APH** and a leading cause of maternal and foetal **morbidity** and **mortality** during pregnancy and delivery.

PL PRV may sometimes **adhere to** and/or **invade** the myometrium especially at the site of a previous CS scar, leading to an abnormally invasive placenta **(AIP),** one of the most life threatening obstetric complications encountered nowadays especially with the increasing rates of CS.

INCIDENCE

PL PRV with all its types may be encountered in around **0.5%** of **all** pregnancies, and accounts for more than **20%** of all causes of late obstetric haemorrhage.

The incidence of PL PRV has been showing a ***progressive rise*** in the last 2 decades due to increasing maternal age and increasing incidence of repeated deliveries by CS.

PREDISPOSING AND RISK FACTORS

1. **Increasing** maternal **age:** incidence **> 1%** in women > 35 years of age
2. **Increased parity: i**ncreased number of pregnancies and deliveries nay affect the size and shape of the LUS predisposing to low placental implantation.
3. **Increased placental size** (as in **GDM, twins,** & **multifoetal** pregnancies) is one of the most important risk factors for a PL PRV.
4. **Previous PL PRV** increases the risk of a future PL PRV in a next pregnancy.
5. **Previous CS:** increases the risks of both PL PRV and accreta.

MECHANISM OF BLEEDING IN PL PRV

Bleeding in PL PRV occurs due to **unavoidable separation** of the placenta as a result of stretching of the LUS while the pregnancy is ongoing. Stretching of the LUS may occur either;

- **Gradually** at late 3rd trimester (causing mild recurrent APH), **or**
- **Suddenly** with the onset of labor pains and uterine contractions causing rapid separation with sudden onset of severe bleeding

▪**N.B.:** The lower the placental implantation on the LUS, the more will be the chances for earlier and more severe patterns of bleeding due to earlier and more extensive placental separation.

PLACENTAL MIGRATION

- Some patients will have a degree of encroachment of their lower placental margin on the LUS as revealed by US examination done **< 24weeks** gestation. With the development of the LUS, a relative upward **placental migration** occurs, with almost **90%** of these resolving by the third trimester.
- **Complete** and **partial** PL PRV are less likely to resolve, compared to **marginal** PL PRV with only 10% of cases resolving by the third trimester.

CLASSIFICATION AND TYPES OF PL PRV

PL PRV is classified according to the relationship of the placenta to the internal cervical os into;

- **Total PL PRV** (central or complete): the placenta totally covers the internal os (most serious).
- **Partial PL PRV:** the placenta partially covers the internal os (IO).
- **Marginal PL PRV:** the lower placental edge stops at the margin of the internal os.
- **Lateral PL PRV:** the lower placental edge stops > 2.0 cm above the internal os

N.B.:

- **Central PL PRV** whether total or partial is also known as **Major PL PRV**
- **Marginal** and **Lateral PL PRV** are also known as **Minor PL PRV**

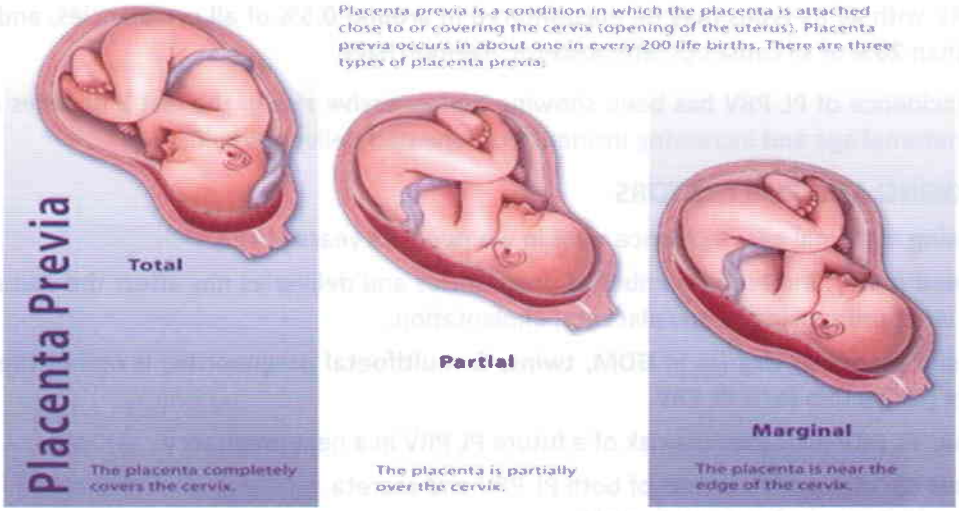


Fig 21-3: Classification of the types of PL PRV

SYMPTOMS & SIGNS:

- **No Symptoms:** 1/3 of cases of PL PRV may be asymptomatic till the onset of labour
- **Vaginal Bleeding:** painless recurrent **vaginal bleeding** in the **3rd trimester** is the most common clinical presentation in the majority of cases (APH).
- **General Examination:**
  - *Mild APH:* no significant signs
  - *Severe APH:* signs of acute massive blood loss including; pallor, tachycardia, tachypnoea, rising pulse, falling BP, oliguria, and loss of consciousness (haemorrhagic shock).
- **Abdominal Examination:** shows no specific signs, however **malpresentations** especially breech and transverse lie are more frequently encountered as the low lying placenta will usually interfere with adaptation of the presenting part through the pelvic inlet.
- **Vaginal Examination (PV):** should not be attempted for diagnosis as it may provoke placental separation with consequent severe uncontrollable bleeding.

ULTRSOUND DIAGNOSIS

A) **TRANSABDOMINAL US (TAS):** this is the **gold standard** for diagnosis of PL PRV with almost **95%** accuracy. It is also accurate in classifying PL PRV into total, partial, or marginal according to its relation to the internal cervical os. If the lower placental margin is > 2.0 cm away from the internal os it may be then considered a low lying placenta (lateral PL PRV) which carries less risk for bleeding and separation, and does not contraindicate vaginal delivery.

B) **TRANSVAGINAL US (TVS):** is more reliable than TAS in measuring the distance between lower placental margin and internal os to differentiate a marginal from lateral PL PRV when in doubt.

N.B.: Magnetic Resonance Imaging (MRI) is a useful and accurate tool for diagnosis of PL PRV, however its use is limited by its higher expenses, lack of availability, and complexity of the procedure when compared to ultrasonography.

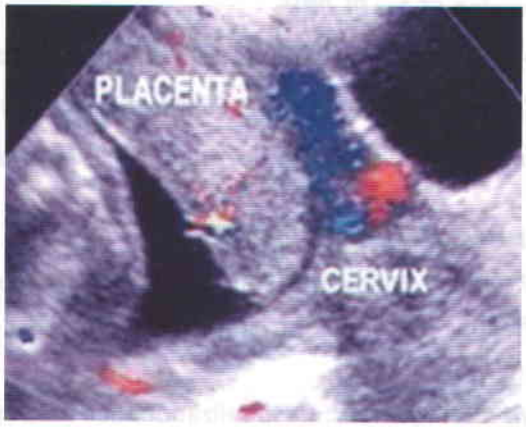


Fig 21-4: US picture of a partial PL PRV

MATERNAL AND FOETAL RISKS AND COMPLICATIONS IN PL PRV

1. Severe **APH** leading to hypovolemic **shock** with all its complications including **DIC**
2. Severe postpartum haemorrhage **(PPH):** due to increased vascularity of the LUS and its defective retraction, together with possible retained placental fragments and uterine atony.
3. Increased risk of **CS hysterectomy** due to uncontrolled bleeding from dilated placental bed on the LUS which has a less contractile and retractile power than the upper uterine segment.
4. Increased risk of **abnormally invasive placenta** especially in presence of previous CS scar, with all its associated complications (see later).
5. **Foetal complications** primarily increased perinatal morbidity and mortality due to prematurity, IUGR, and massive blood loss.

EVALUATION OF MATERNAL AND FOETAL WELL BEING:

A) ***Maternal laboratory test:*** CBC, liver & kidney function test, blood glucose level, PT, PC, INR.

B) ***Tests for foetal wellbeing:*** NST, BPP, UA Doppler velocimetry studies, amniotic fluid index

N.B.: See details in haemorrhagic shock and foetal surveillance tests

MANAGEMENT OF PL PRV

All cases of PL PRV (total, partial & marginal) will be delivered by CS. The timing of the procedure will depend upon many variables including;

- The *severity of APH* if present, and its effect on maternal and/or foetal wellbeing.
- The *gestational age* at time of diagnosis (term or preterm).
- Presence or absence of *uterine contractions* (onset of labor pains).

A) ACTIVE MANAGEMENT BY IMMEDIATE CS:

1. *Severe bleeding* compromising maternal or foetal lives, **regardless** gestational age
2. *Gestational age > 37 weeks* **regardless** amount of bleeding
3. *Spontaneous onset of labour pains,* regardless amount of bleeding and gestational age

- ***Precautions during performing CS for PL PRV:***
  - Immediate **resuscitation**, anti-shock measures and **blood transfusion** before surgery
  - Liberal use of **ecbolics** (Oxytocin, Ergometrin, & PGL) to avoid uterine atony
  - Consider the need for bilateral **uterine artery ligation** or **CS hysterectomy** if required.

B) EXPECTANT MANAGEMENT to DELAY DELIVERY:

In preterm pregnancies < 37 weeks to allow more time to achieve better lung maturity whenever:

1. *Bleeding is mild* not affecting maternal or foetal well being
2. *Labour pains* are absent

- ***Prerequisites for expectant management:*** Hospitalization, bed rest, blood bank reservation, and biweekly evaluation of maternal and foetal wellbeing performed on regular basis and continued until any indication for active management arises (best achieved lung maturity, or onset of labour pains, or an attack of severe vaginal bleeding, or non-reassuring tests of foetal wellbeing)

C) Cases with Lateral PL PRV (lower edge 2 cm or more above the internal os) can be offered a trial vaginal delivery at onset of labour pains whenever bleeding is minimal.

- **NB:** Unsensitized Rh-ve patients should receive anti-D Ig during the expectant management

RUPTURE VASA PRAEVIA (FOETAL BLEEDING)

Vasa praevia is a very rare condition in which the umbilical **cord** vessels **cross over** the internal os (as in velamentous insertion of the cord, or succenturiate lobe of the placenta). During labour, progressive cervical dilatation may lead to separation and rupture of these vessels leading to foetal bleeding with associated foetal distress, severe bradycardia, and ultimately intrapartum **foetal death** unless the condition is urgently recognized and dealt with properly by an **emergency CS.**

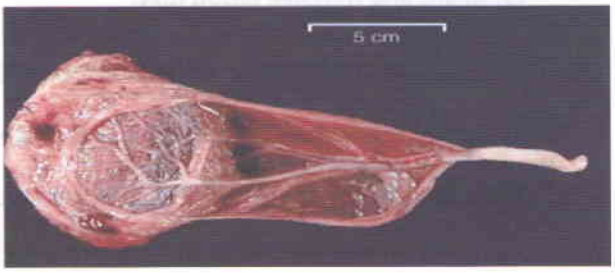


Fig 21-5: Velamentous cord insertion with vasa praevia

ABNORMALLY INVASIVE PALCENTA (AIP)

(Placenta Accreta, Increta, & Percreta)

This is a **rare** but a serious, and may be **fatal**, condition in which the placenta **adheres** to or **invades** the myometrium. It 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.

TYPES OF ADHERENT PLACENTA

- **Accreta;** the placenta is adherent to the myometrium (most common 80%)
- **Increta:** placenta invading the myometrium short of serosa
- **Percreta:** placenta perforating the myometrium reaching the serosa, and may invading bladder wall as well.

DIAGNOSIS DURING PREGNANCY

- Diagnosis can be achieved by second level **expert US** (2D or 3D US and colour Doppler).
- US reveals absence of a plane of cleavage between placental and myometrium denoting adherence with presence of blood lacunae in cases of invasion
- Diagnosis can be also achieved by **MRI** however with no significant benefit over expert US

DIAGNOSIS DURING LABOUR

Some cases of focal placenta accreta will be discovered during labour after dekivery of the foetus when the placenta is totally or partially retained leading to severe atonic postpartum haemorrhage PPH (see later and see complications of 3rd stage of labour)

MANAGEMENT

The majority of cases will require CS hysterectomy as a lifesaving procedure. Ovaries are preserved during surgery (*see also Placenta Accreta in PPH)*.

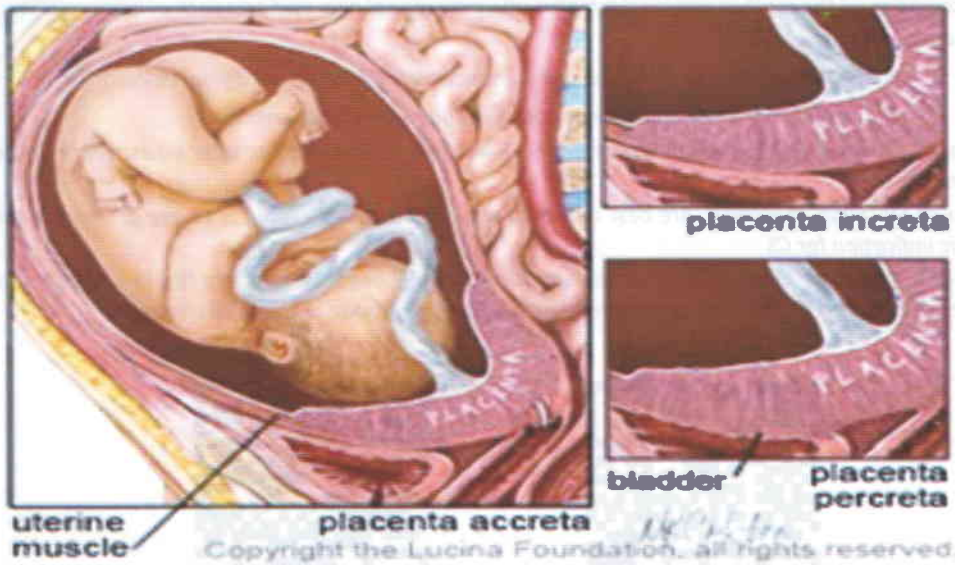


Fig 21-6: Types of adherent placenta

MANAGEMENT OF A CASE OF APH

1. *Grading severity* of bleeding and its effect on *maternal* and *foetal* well being
2. *Hospitalization* and *resuscitation* measures for cases with haemorrhagic shock (see later)
3. *Immediate TAS:* for placental localization, and confirmation of gestational age
4. *Exclusion of presence of labour pains:* by CTG and vaginal examination ONLY when indicated
5. *Choice between active and expectant management* according to gestational age, severity of bleeding, maternal condition, and foetal wellbeing.
6. *Choice between vaginal delivery & CS* whenever termination of pregnancy is indicated or labour pains have already started

RESUSITATION MEASURES

- Insertion of a wide bore IV cannula and start immediate saline and crystalloid infusion
- Insert urinary catheter and monitor urine output in relation to fluid intake
- Continuous monitoring of vital signs (Temp, BP, Pulse, and RR)
- Blood group/RH matched and adequate amount of blood reserved

Key Points in APH

- *Late obstetric bleeding is most commonly due to PL PRV, less commonly placental abruption, and rarely ruptured vasa PRV.*
- *Cases with PL PRV mostly present with painless, causeless, and recurrent vaginal bleeding. If bleeding is severe the patient will be in state of hypovolemic shock.*
- *Major PL PRV is usually associated with severe life threatening APH*
- *Placental abruption is usually of the mixed type, presenting with painful vaginal bleeding.*
- *Concealed haemorrhage is associated with sudden, severe, and progressive abdominal pain. Shock if present may be hypovolemic or neurogenic or both.*
- *In concealed accidental haemorrhage the patient's general condition may be poor and deteriorating inspite of minimal revealed vaginal bleeding. Such cases are also at high risk for development of DIC, and Couvelair's uterus.*
- *Foetal distress is more frequently encountered in cases of placental abruption than in association with PL PRV.*
- ***Ultrasonography** is the gold standard in diagnosing the cause of APH.*
- *Management of a case of APH will depend upon several factors including the severity of APH, its effect on the condition of the mother and/or the foetus, the gestational age, and presence of labour pains.*
- *Management may be either:*
  - ***Active;** immediate termination of pregnancy whether by CS or vaginal delivery ,or*
  - ***Conservative:** allowing continuation of pregnancy and observation*
- *Almost all cases of **PL PRV** will be delivered by **CS** whether immediately or after an expectant period of management*
- *Cases of placental abruption. Are best delivered vaginally unless bleeding is severe or in presence of an obstetric indication for CS*
- *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.*

22 PREMATURE RUPTURE OF THE MEMBRANES TERM AND PRETERM

- *Hazards and complications*
- *Diagnosis of chorioamnionitis*
- *Management of PROM*

Premature rupture of the membranes **(PROM)** is the term used to describe spontaneous rupture of the foetal membranes before the onset of labour. It may complicate up to **10%** of all pregnancies.

If PROM occurs **before 37** weeks gestation (**preterm PROM**), it may be associated with significant maternal and/or foetal morbidity resulting in one of the most challenging obstetrical complications.

AETIOLOGY

1. **Idiopathic:** in many cases PROM will occur spontaneously without an identifiable aetiology
2. **Infection:** ascending infection from the lower genital tract (LGT) is the **most common** risk factor causing PROM mostly due to decreasing the tensile strength of foetal membranes. Common organisms include Group B streptococci (GBS) and Trichomonas Vaginalis (TV).
3. **Cervical Incompetence:** bulging membranes through the dilated cervix are exposed to trauma and infection that may cause erosion and PROM.
4. **Polyhydramnios and Multifoetal pregnancy (MFP):** are commonly associated with PROM mostly due to chronically increased intra-amniotic pressure
5. **Local membrane defects** and **cigarette smoking** have been proposed as risk factors

HAZARDS AND COMPLICATIONS

A) PRETERM LABOUR (PTL):

In most cases labour will start within **few hours** after ROM. If this occurs < 37 weeks gestation it will lead to PTL with all serious foetal hazards related to prematurity including respiratory distress syndrome (RDS), birth trauma, PDA, neonatal asphyxia & sepsis (see details in PTL)

B) INFECTION (CHORIOAMNIONITIS):

Prolonged ROM may be easily complicated by ascending **LGT** infection in at least **20%** of cases. The most serious infection is caused by **GBS** that may lead to both;

- *Maternal hazards;* chorioamnionitis, septicaemia, septic shock and puerperal sepsis
- *Foetal hazards;* serious neurologic morbidity, foetal pneumonia, respiratory distress syndrome (RDS) and neonatal death

C) PLACENTAL ABRUPTION:

ROM in cases of polyhydramnios and MFP may result in a sudden decrease in intra-amniotic pressure leading to placental separation with all maternal and foetal hazards of accidental haemorrhage (see details in placental abruption)

D) OLIGOHYDRAMNIOS:

- *Hazards during labour;* cord compression leading to foetal bradycardia and hypoxia
- *Hazards of prolonged ROM:* if pregnancy continues for few weeks or months after preterm PROM the foetus will be at risk for pulmonary hypoplasia with severe RDS, compression deformities of the limbs, and amniotic band syndrome (see amniotic fluid disorders)

CLINICAL PRESENTATION

- ***Symptoms***: Sudden gush of a significant amount of colourless fluid through the vagina
- ***Signs:***
  - *Inspection:* reveals clear, colourless, odourless fluid flowing out of the vagina
  - *PV examination:* should be avoided to minimize risk of ascending infection unless in labour
- ***Ultrasound:*** reveals decreased amount of liquor amnii around the foetus. Amniotic fluid pockets less than 2 cm or amniotic fluid index (AFI) < 5 points to oligohydramnios.

CONFIRMING DIAGNOSIS OF PROM

Sterile speculum examination shows the following positive criteria essential for diagnosis;

- ***Pool positive:*** clean watery amniotic fluid collected in the posterior vaginal fornix
- ***Nitrazine positive:*** pooled fluid turns **PH** sensitive Nitrazine paper into **blue** colour
- ***Fern positive:*** fluid elicits a fering pattern under microscope, when left to dry on a glass slide
- ***AmniSure test:*** highly accurate test that measures placental alpha macroglobulin

DIAGNOSIS OF CHORIOAMNIONITIS

A) **Clinical Diagnosis:** maternal **fever** and uterine **tenderness** in confirmed case of **PROM** in absence of urinary tract infection (UTI) or respiratory tract infection (RTI)

B) **Laboratory Diagnosis:** increased **TLC**, abnormal **DLC** with increase staff/ segmented WBC ratio (**left shift),** with **toxic granules**, and increased **CRP**

MANAGEMENT OF PROM

Management of PROM will vary according to gestational age, presence of uterine contractions or evidence of infection.

1. In presence of ***uterine contractions:*** labour is allowed to proceed, **regardless** gestational age, as tocolytic agents are contraindicated for fear of masking evidence of maternal infection (see PTL)
2. Evidence of starting ***chorioamnionitis:*** cervical cultures obtained, IV therapeutic broad spectrum antibiotics started, and pregnancy is promptly **terminated** (induced labour or CS)
3. If **NO** labour pains and **NO** chorioamnionitis management will be according to gestational age;
   - ***Preterm PROM:*** Conservative management including hospitalization, IM Betamethasone for lung maturity, cervical cultures, and Ampicillin / Erythromycin 7 days prophylactic therapy.
   - ***Term PROM > 37 weeks:*** pregnancy should be promptly terminated by either;
   - **Induction of labour:** labour is induced by oxytocin or PGL
   - **CS:** in cases unfavourable for vaginal delivery or if failed induction of labour

N.B.: In centres with advanced neonatal ICU services 34 weeks rather than 37 weeks may be adopted as the gestational age at which conservative management can be abandoned and pregnancy terminated.

23 PRETERM LABOUR AND POST TERM PREGNANCY

- *Preterm labour (PTL)*
- *Post Term pregnancy (PTP)*

PRETERM LABOUR

Preterm labour **(PTL)** is defined as onset of **true** labour pains any time **after** foetal **viability** but **before 37** weeks gestation. PTL is the commonest underlying factor for **prematurity** which is the most common cause for neonatal **morbidity** and **mortality**. PTL may complicate **5-10%** of pregnancies

- ***True labour pains:*** are rhythmic uterine contractions that lead to progressive cervical effacement and dilatation.
- ***Foetal viability:*** is the gestational age at which a foetus can survive after delivery. Its lower margin varies from **20-24** weeks according to neonatal ICU (NICU) facilities available.
- ***Foetal lung maturity:*** is assumed to be completed by **37** weeks gestation which defines term from pre-term babies.
- ***Early PTL:*** is PTL occurring **< 34** weeks gestation. It is the more serious type of PTL
- ***Late PTL:*** is PTL occurring **> 34** but **< 37** weeks. It carries less foetal risks than early PTL

INCIDENCE AND RISK FACTORS

PTL may complicate **5-10%** of ALL pregnancies. In most cases there is **NO** identifiable aetiology making it mostly unpredictable, however **RISK FACTORS** for PTL include;

A) OBSTETRIC RISK FACTORS:

- Previous PTL, PROM, IUGR & IUFD
- Cervical incompetence and recurrent pregnancy loss (RPL)
- Short cervical canal < 2.5 cm by TVS > 24 weeks gestation
- Uterine anatomic abnormalities; as septate or bicornuate uterus and large leiomyomas
- Uterine over-distension as: twins & MFP, polyhydramnios, and foetal macrosomia.

B) MEDICAL FACTORS AND CONDITIONS

- Genital tract and urinary tract infections (GTI & UTI) during pregnancy
- Hypertension, preeclampsia, and gestational DM necessitating early pregnancy termination
- Severe anaemia and malnutrition

C) ENVIRONMENTAL AND BEHAVIORAL FACTORS

- Successive short interval deliveries < 18 months
- Excessive smoking and substance abuse
- Excessive physical activity, competitive sports, heavy exercise, and psychosocial stress

COMPLICATIONS AND HAZZARDS OF PREMATURITY

1. **Respiratory Distress Syndrome (RDS):** decreased lung surfactant (mainly Lecithin), will predispose to lung collapse with loss of alveolar wall elasticity in terminal air spaces.
2. **Birth Trauma:** fragile cerebral vessels may easily lead to intracranial haemorrhage (ICH) and intraventricular haemorrhage (IVH) 2ry to head compression and hypoxia.
3. **Neonatal Sepsis:** due to immature and relatively incompetent immune system.
4. **Necrotizing Enterocolitis (NEC):** bacterial infection in small intestine leading to necrosis
5. **Retinopathy of prematurity**
6. **Cerebral Palsy (CP):** more liable to occur 2ry to birth trauma, hypoxia, ICH, IVH, infection, and perinatal infections

PREDICTION OF PTL (Difficult)

In most cases PTL will be **unpredictable** inspite of the previously cited risk factors. Irregular repeated uterine **cramps** and excessive vaginal **discharge** may raise suspicions, however the following may help prediction;

a. **TVS** showing short **cervical canal** length < 2.5 cm after 24 weeks gestation.
b. **Foetal Fibronectin (FFN)** in cervical discharge: FFN is a protein matrix produced by foetal cells that acts as a glue binding trophoblasts to maternal decidua. It leaks into the vagina if PTL is likely to occur. It is measured by repeated vaginal swabs from vaginal pool at posterior fornix. The test It is valid between 24-32 weeks;
   - *Negative test (absence of FFN):* < 1% chance for a PTL within 2 weeks
   - *Positive test (presence of FFN):* 50% chance for PTL within 2 weeks

MANAGEMENT DURING ONSET OF PTL

A) CONFIRM DIAGNOSIS OF PTL: True labour pains with progressive cervical dilatation and effacement that occurs between 24 – 37 weeks gestation confirmed by;

- ***Cardiotography (CTG):*** recording at least 4 contractions in 20 minutes
- ***PV examination:*** showing cervical effacement 50% or more and dilatation at least 2 cm

B) HOSPITALIZATION: bed rest, sedation, and adequate hydration with IV Dextrose 5%, together with monitoring the frequency and intensity of uterine contractions with **CTG**

C) MANAGEMENT OF A CORRECTABLE CAUSE as **GTI** and **UTI** via proper antibiotic therapy according to culture and sensitivity obtained by vaginal swabs and urine samples.

D) ULTRASOUND EXAMINATION: for confirming gestational age, foetal weight, foetal presentation, placental localization, amniotic fluid volume, and evaluation of foetal wellbeing by biophysical profile and umbilical artery colour Doppler velocimetry studies. Ultrasound can also diagnose many risk factors as presence of polyhydramnios, foetal macrosomia, MFP, and leiomyomas.

E) INITIATING CORTICOSTEROID THERAPY: for minimizing hazards of prematurity through:

- ***Enhancing lung maturity:*** IM corticosteroid act via stimulation of foetal type II pneumocyte surfactant production < 34 weeks gestation thus minimizing the risk of neonatal RDS.
- ***Minimizing incidence of NE, ICH, and IVH.***
- ***Dose Regimens:*** either
  - *Betamethasone (24 mg/24 hours):* 2 IM doses **12** mg each **24** hours apart, **OR**
  - *Dexamethasone (24 mg/48 hours):* 4 IM doses **6.0** mg each **12** hours apart

F) TOCOLYTIC THERAPY IN PTL:

- **Aim of Tocolysis:** drugs are used in attempt to stop uterine contractions and prolong pregnancy for at least **48-72** hours to get **benefit** from administration of IM corticosteroids, and transfer the patient to a tertiary centre d with advanced neonatal ICU facilities.
- **Contraindications to Tocolysis:**
  - *Obstetric conditions as:* placental abruption, ROM, and chorioamnionitis.
  - *Foetal conditions as:* foetal distress, and lethal anomalies
  - *Maternal conditions as:* severe preeclampsia (PE), eclampsia, and advanced cervical dilatation and effacement.

TOCOLYTIC AGENTS

1. Calcium Channel Blockers (Nifedipine):

- *Mode of action:* decrease intracellular calcium thus decreasing myometrial contractility.
- *Route of administration:* loading dose with 30 mg orally, followed by maintenance dose of 20 mg/6 hours for 24 hours, then 20 mg/8 hours for another 24 hours.
- *Side effects:* headache, flushing, hypotension and tachycardia (vasodilatation effect).

2. Magnesium Sulphate:

- *Mode of action:* $MgSO_4$ is a competitive inhibitor of calcium
- *Route of administration:* Loading dose IV followed by IV infusion.
- *$MgSO_4$ Toxicity* includes; muscle weakness and respiratory depression.
- *Dosage monitoring:* monitor respiratory rate and deep tendon reflexes (knee jerk)
- *Antidote for toxicity:* IV calcium gluconate
- ***N.B.:*** $MgSO_4$ reduces the risk and severity of cerebral palsy **(CP)** in surviving extremely preterm neonates < 28 weeks

3. β-Adrenergic Agonists (Ritodrine hydrochloride):

- *Mode of action:* β2 receptor stimulants result in myometrial relaxation.
- *Route of administration:* slow IV infusion drip in doses titrated to control uterine contractions.
- *Side effects:* maternal and foetal tachycardia, HTN, hyperglycaemia, and pulmonary oedema.
- Contraindications: cardiac disease, DM, and hyperthyroidism
- **N.B.:** The use of B-mimetic drugs has been declining due their marked maternal side effects.

4. Prostaglandin Synthetase Inhibitors (Indomethacin):

- *Mode of action:* decrease smooth muscle activity by decreasing PGL production.
- *Side effects:* oligohydramnios, in utero closure of ductus arteriosus, and NEC.
- *Contraindications*: gestational age > 32 weeks

5. Anti-Oxytocin (Atosiban):

- *Mode of action:* act by blocking intra-myometrial oxytocin receptors
- *Route of administration:* IV infusion with titrated doses that inhibit uterine contractions

MANAGEMENT DURING LABOUR AND DELIVERY

- *Vertex presentations:* vaginal delivery is allowed with foetal acidosis and trauma avoided
- *Breech presentations:* CS gives better neonatal outcome with foetal weight < 1500 gm.

- **N.B.:** intrapartum continuous foetal monitoring is essential with special attention to abnormal FHR patterns

PROPHYLACTIC IM PROGESTERONE (PRG) ADMINISTRATION

In cases with a history of a **previous** PTL, administration of **weekly** IM 17 a-OH **PRG** Caproate from 20 weeks gestation onwards, has been shown to minimize incidence of a recurrent preterm delivery.

POST TERM PREGNANCY (Prolonged Pregnancy)

Prolonged or **Post term** pregnancy **(PTP)** is a pregnancy that continues beyond **42** weeks gestation (249 days), estimated by the **LMP.** Nearly 6-12% of pregnancies may extend post term, however the incidence has been declining in the last decade owing to policies of pregnancy termination before reaching 42 weeks gestation to avoid **risks** and **complications** of postmaturity.

Accurate pregnancy **dating** supported by 1st trimester **US** are the golden standard for diagnosis of a PTP, however both will be lacking in the majority of cases making sure diagnosis quite difficult.

Most cases with PTP are **idiopathic** with **NO** identifiable **aetiology**, while in at least 20-30% of cases pregnant women proper history taking reveals an **unreliable** LMP.

- **N.B.:** lack of a proposed **labour initiating factor** produced by foetal adrenals has been implicated as an aetiological factor. This has been related to cases with **anencephaly** due to absent foetal hypothalamic-pituitary regulation.

RISKS OF POSTMATURITY

A) **Large for Gestational Age foetus (LGA):** In almost 80% of PTP normal placental function is maintained leading to advanced foetal growth and an LGA foetus with a higher risk for a difficult vaginal delivery, and both maternal and foetal traumatic birth injuries (see obstetric trauma).

B) **Placental Insufficiency:** In almost 20% of cases placental function declines by ageing, scarring, and fibrin deposition, leading to foetal **dysmaturity** syndrome and intrauterine growth restriction **(IUGR),** with hazards of foetal acidosis and hypoxia during labour compromising foetal outcome.

C) **Higher CS delivery rates:** LGA, and IUGR are associated with higher CS rates to improve foetal and maternal outcome by avoiding birth trauma, oligohydramnios, meconium aspiration, and neonatal hypoxia.

- **N.B.:** PTP has a **2-3 fold** risk of increased **perinatal mortality**.

MANAGEMENT OF PTP

1. ***Confirming diagnosis:*** via proper dating of pregnancy through meticulous history taking, reaching a reliable LMP or an accurate 1st trimesteric US examination.
2. ***US confirmation of gestational age:*** using reliable parameters as BPD, AC, HC, FL, together with long bones ossification centres and placental grading.
3. ***Evaluation of foetal wellbeing:*** to facilitate decisions for pregnancy prolongation or termination
   - **Daily** foetal Kick counts and **Biweekly** non stress test (NST).
   - **Weekly US** including: biophysical profile, umbilical artery Doppler, and amniotic fluid index.

4. Active Pregnancy termination: this is indicated whenever

- PTP has been firmly confirmed to have passed **41** weeks **or**
- Evidence of placental insufficiency; as oligohydramnios & umbilical artery Doppler changes.

- **Induction of labour** is weighed against **CS** according to favourability of the cervix, estimated foetal weight and presence or absence of evidence of placental insufficiency.
- **Bishop scoring** is the method to evaluate favourability of the cervix for induction (see induction of labour)

24 AMNIOTIC FLUID DISORDERS

- *Oligohydramnios*
- *Polyhydramnios*

THE AMNIOTIC FLUID

SOURCES AF AMNIOTIC FLUID

- **Foetal:** urine production, amniotic membrane, and transudation from foetal circulation
- **Maternal:** placental chorionic membrane and transudation from maternal circulation

AMNIOTIC FLUID VOLUME and COMPOSITION

The amniotic fluid (AFL) is continuously produced and absorbed from both foetal and maternal surfaces with a more pronounced foetal contribution.

Amniotic fluid volume (AFV) shows gradual increase with gestational age reaching a maximum volume of 1-1.5 L at 34-36 weeks. This is followed by a slight decline the last 4 weeks, then a sharper and more rapid decline occurs after 40 weeks gestation.

The AFL is composed of **99% water**, and is characterized by having a **clear** aspect, **pale** colour, and slightly **alkaline PH** (nearly 7.0). It contains **carbohydrates, lipids, proteins, enzymes**, and **mediators** as cytokines. It also contains **foetal excretory products** as; uric acid, urea, creatinine, and sodium chloride, together with vernix caseosa and lanugo hair.

FUNCTIONS OF THE AMNIOTIC FLUID

1. Protection of the foetus from external trauma
2. Maintains constant surrounding temperature suitable for foetal growth
3. Provides a suitable medium for foetal movement for muscular development and neuromuscular coordination.
4. Provides a source for foetal excretion and a source of foetal nutrients
5. Allows for foetal lung distension and development
6. Prevents occurrence of intra-amniotic bands, adhesions, and foetal compression deformities

EVALUATION OF AMNIOTIC FLUID VOLUME

Clinical evaluation of AFL is only subjective and seldom accurate especially in presence of maternal obesity, twins and MFP, PI PRV, and uterine leiomyomas. US is the gold standard in evaluation of AFV and diagnosis of volume disorders as oligohydramnios and polyhydramnios

A. AFL pockets measurement in longitudinal and AP diameters in cm length

B. AFL Index (AFI) in which the vertical diameter of largest pocket is measured in each of the four abdominal quadrants. The sum of 4 quadrants gives an estimate of AFI.

- **Normal liquor** (AFI: 10-15), **oligohydramnios** (AFI < 5.0), and **polyhydramnios** (AFI > 25).

OLIGOHYDRAMNIOS

Oligohydramnios is defined as **diminished AFV**. By US it is diagnosed when measurement of the largest vertical **AFL pocket is < 2 cm**, or an **AFI < 5** after 28 weeks (3rd trimester)

INCIDENCE

- Oligohydramnios affects almost 3-4 % of all pregnancies
- Incidence is significantly increased in association with IUGR and placental insufficiency

AETIOLOGY

1. Undiagnosed **ROM**
2. **Placental insufficiency:** as in IUGR, Preeclampsia (PE), and post term pregnancy (PTP)
3. **Foetal causes:** Foetal congenital anomalies (FCA) as renal agenesis and renal hypoplasia
4. **Drugs;** Indomethacin Is known to decrease AFV due to decreased renal perfusion

SYMPTOMS AND SIGNS

1. History of leaking amniotic fluid (ROM)
2. Inappropriate growth of maternal abdominal girth especially in the 3rd trimester
3. Inspection: reveals smaller than expected abdominal contour
4. Abdominal examination: lower than expected fundal level for gestational age;

RISKS AND COMPLICATIONS

A) During pregnancy:

- Complications related to IUGR and placental insufficiency (see later)
- Pulmonary hypoplasia, compression limb deformities, and amniotic adhesions

B) During labour

- Cord compression with foetal hypoxia and foetal distress
- Meconium passage and foetal meconium aspiration

MANAGEMENT

1. **Termination of pregnancy:** in cases that reached near term or late preterm especially if associated with placental insufficiency or lethal anomalies
2. **Amnio-infusion:**
   - Intra-amniotic saline injection (250 – 300 ml) in an attempt to prolong pregnancy in cases of the extremely preterm pregnancy, to avoid complications of long term oligohydramnios.
   - Amnio-infusion is rarely performed during labour, in cases with ROM, via a trans- cervical catheter to avoid cord compression and foetal distress necessitating CS.

POLYHYDRAMNIOS

Polyhydramnios is defined as **excessive AFV** above the 95th percentile for gestational age, or **> 2000 ml** after 28 weeks (3rd trimester). By US it is diagnosed when measurement of the largest vertical **AFL pocket is > 8 cm**, or an **AFI > 25** after 28 weeks (3rd trimester)

INCIDENCE: it may be associated with **0.5-1.5 %** of pregnancies

AETIOLOGY

1. **Idiopathic:** most cases are possibly due to an imbalance between AFL production and absorption of an unknown aetiology which may not be discovered even after delivery
2. **Foetal causes:**
   - Twins and MFP: due to large surfaces producing AFL (placenta and membranes)
   - Placental tumours: as chorioangioma
   - FCA: as anencephaly, open NTDs, oesophageal atresia, and duodenal atresia
   - Hydrops foetalis: due to large placenta & excessive transudation from foetal circulation
3. **Maternal causes:**
   - Gestational diabetes mellitus (GDM): most common cause
   - Rarely with PE: in association with placental oedema

SYMPTOMS AND SIGNS

1. Progressive rapid enlargement of abdominal girth especially in 3rd trimester
2. Respiratory embarrassment and pressure symptoms only in severe cases
3. Inspection: shows larger abdominal contour than expected for gestational age
4. Fundal level: is higher than expected for gestational age
5. Foetal parts are not easily palpated due to excessive surrounding AFL

EVALUATION OF SEVERITY

1. Severe cases are associated with disabling respiratory and pressure symptoms
2. US measurements showing AFI > 25 and or vertical AFL pocket > 8 cm
   - **Mild**: AFL pocket > 8 cm
   - **Moderate**: AFL pocket > 12 cm
   - **Severe**: AFL pocket > 16 cm

RISKS AND COMPLICATIONS

1. PTL due to uterine overdistension, with foetal prematurity complications
2. Dysfunctional labour due to malpresentations, macrosomia, PROM, and associated MFP
3. Placental abruption due to suddenly decreased intraamniotic pressure after PROM
4. Risks of associated foetal anomalies or maternal GDM

CLINICAL TYPES OF POLYHYDRAMNIOS

A) **Acute Polyhydramnios:** rapidly accumulating AFL usually < 20 weeks causing early symptoms. It is usually associated with uniovular twins or major foetal anomalies and ends mostly in abortion or extremely PTL.

B) **Chronic Polyhydramnios:** slowly accumulating AFL usually > 24 weeks. It is less serious type unless severe, mostly idiopathic, or associated with GDM. It causes late symptoms and pregnancy is usually completed successfully or ends in late PTL

MANAGEMENT

1. Mild and Moderate Polyhydramnios:

Mild and moderate cases are usually chronic and late developing. Cases are best managed conservatively till spontaneous labour pains start. During this period the patient will need, reassurance, follow up, and exclusion of MFP, FCA, and GDM by US.

2. Severe Polyhydramnios:

- ***Term pregnancy > 37 weeks:*** termination of pregnancy by either induction of labour or CS according to foetal presentation, pelvic adequacy, and bishop score
- ***Preterm pregnancy < 37 weeks:*** Repeated amniocentesis removing 1-1.5 litres every time to prolong pregnancy until lung maturity is established and to give more time for IM corticosteroids to enhance lung maturity.

N.B.: Indomethacin (Anti-PGL) may decrease AFV through decreasing foetal urine production and enhancing foetal lung absorption, due to a decrease in renal blood via enhancing vasopressin effects. Its use > 32 weeks gestation is associated with a higher risk of intrauterine premature closure of foetal ductus arteriosus leading to neonatal cardiac and respiratory problems. It is thus only used when its benefits outweigh the risks.

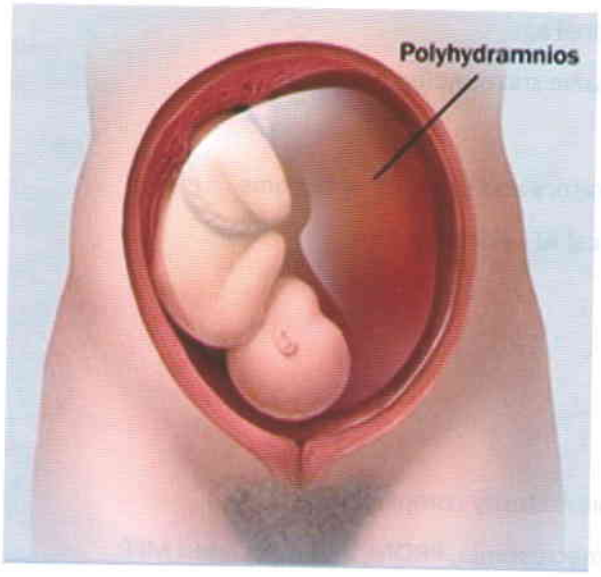


Fig. 24-1: Polyhydramnios

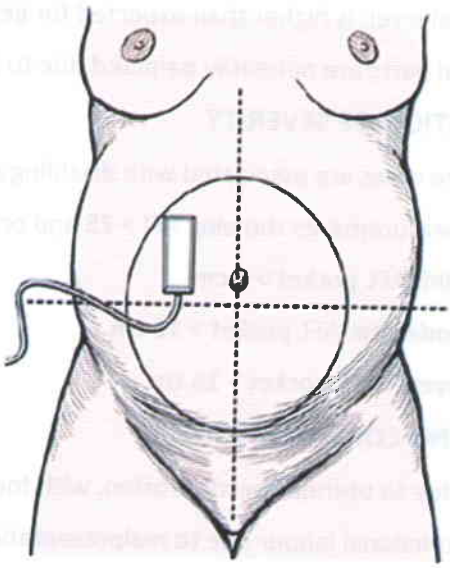


Fig. 24-1: 4 quadrant assessment of AFI by US

25 FOETAL GROWTH DISORDERS

- *Intrauterine Growth Restriction IUGR*
- *Intrauterine Foetal death IUFD*
- *Foetal Macrosomia*

Foetal birth weight a function of both gestational age and rate of foetal growth.

Estimation of gestational age is based on a *reliable LMP* dating and/or an *early US* scan, while foetal growth rates are affected by both *placental function* and *genetic* or constitutional factors.

- ***Appropriate for gestational age (AGA):*** foetuses between 10th - 90th percentile for their GA
- ***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.
- ***Low birth weight (LBW):*** Infants with birth weight <2500 gm regardless their gestational age.

ESTIMATION OF FOETAL WEIGHT

A) ***Clinical Estimation:*** via clinical assessment of fundal level and abdominal girth is an unreliable subjective method with low accuracy.

B) ***US Estimation:*** via combined measurements of BPD, HC, AC & FL, is an objective method giving rather accurate foetal weight estimates with less than 10% error range.

I- INTRAUTERINE GROWTH RESTRICTION (IUGR)

IUGR refers to growth patterns in which the foetus fails to reach its full growth potential. These foetuses are generally **SGA** with weights <10th percentile according to foetal growth curves. SGA foetuses may be either constitutionally small babies or growth restricted ones.

I. CONSTITUTIONALLY SMALL FOETUS:

Pregnant women who are underweight at the beginning of pregnancy < 42 kg have a doubled risk of having a LBW foetus weighing < 2.5 Kg. This is considered a constitutional factor.

2. INTRAUTERINE GROWTH RESTRICTION IUGR

IUGR may be **symmetrical** or **asymmetrical.** These two types are likely to be the consequence of different causes, time of onset, and duration of events.

A) SYMMETRICAL IUGR (TYPE I):

Symmetric IUGR usually results from foetal **injury** very **early** in development that is **intrinsic** to the foetus as with intrauterine infections and chromosomal abnormalities. It constitutes about **20%** of IUGR cases.

B) ASYMMETRICAL IUGR (THYPE II):

Asymmetric IUGR usually results from foetal **injury later** in pregnancy, commonly due to maternal disease that is **extrinsic** to the foetus. This type may constitute up to 80% of IUGR. The foetal size is affected via some degree of **placental insufficiency** secondary to reduced placental blood flow, reduced placental size, placental vasculopathy and infarcts, or reduced oxygen and nutrient transfer.

AETIOLOGY OF IUGR

A) SYMMETRICAL IUGR:

1. **Intrauterine Infections:** *(Congenital infections)*
   - ➢ *Viral infections:* Rubella, Cytomegalovirus, Hepatitis, Varicella and Influenza
   - ➢ *Non-viral infections:* Listeriosis, T.B. and syphilis. In Syphilis, the placenta almost always increase in size and weight due to oedema and perivascular inflammation.
2. **Chromosomal Abnormalities:** as trisomies 13, 18 and 21
3. **Congenital Malformation:** as cardiovascular malformation, renal hypoplasia, and skeletal anomalies (osteogenesis imperfecta) and many other inherited disorders.

B) ASYMMETRICAL IUGR:

1. **Maternal vascular disease:** Chronic hypertension, Preeclampsia, and Diabetic vasculopathy.
2. **Antiphospholipid syndrome:** antibodies cause vascular lesions in uterine and placental bed leading to diminished bllod supply and impaired placental infusion.
3. **Chronic renal disease:** usually associated with type II IUGR.
4. **Chronic Hypoxia:** heavy smoking, poor nutrition, drug abuse and cyanotic heart disease
5. **Placental and cord abnormalities:**
   - ➢ Chronic focal placental abruption, extensive infarction, and chorioangioma.
   - ➢ Marginal and velamentous insertions of the cord.

- **N.B:** In cases with chronic placental insufficiency, the foetus reacts to restricted placental perfusion 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:
  a. Preserved HC while decreased AC measurements by US ***(increased HC/AC ratio).***
  b. Reduced renal perfusion and decreased urine output, leading to ***oligohydramnios.***

DIAGNOSIS OF IUGR

1. **Clinical Examination:** a fundal level lower than expected (2 finger breadths lower than GA) and longitudinal symphysial fundal height (2 cm less than GA) can only **suspect** a SGA foetus, given a reliable GA dating by an accurate LMP and/or early 1st trimesteric US scan.

2. **Ultrasound Assessment:**

   a) Foetal Biometry: measurements of the BPD, HC, AC & FL in the 2nd & 3rd trimester is still a less sensitive predictor of foetal GA than CRL measurements in the 1st trimester.
   - ➢ Type I IUGR: Symmetrically SGA foetus with reduction in all biometry measurements (BPD, HC, AC & FL) giving an estimated foetal weight < 10th percentile for GA.
   - ➢ Type II IUGR: biometry measurements reveal discrepancy between HC and AC measurements with increased HC/AC ration due to brain sparing phenomenon.

   b) Oligohydramnios with decreased AFI measurements is common with type II IUGR.

   c) Accelerated placental aging (early grade III placenta < 34 weeks, with calcifications).

   d) Abnormal umbilical and cerebral artery Doppler flow indices (decreased umbilical and preserved or increased cerebral vascular flow in asymmetrical IUGR).

COMPLICATIONS OF IUGR

1. During pregnancy and labour:

- Intrapartum Hypoxia leading to Intrauterine foetal death (IUFD)
- FHR abnormalities during labour, asphyxia and intrapartum foetal death (IPFD).

2. During Neonatal period:

- *Immediate (50%):* meconium aspiration, polycythaemia and pulmonary haemorrhage.
- *Late (2%):* Cerebral dysfunction (mild cerebral palsy).

MANAGEMENT OF IUGR

A) TERM and NEAR TERM IUGR:

- Prompt **delivery** is indicated in **all full term** pregnancies that have completed **37** weeks gestation regardless the type or aetiology of IUGR.
- Delivery is also recommended by most authorities in **near term** pregnancies that have completed **34** weeks gestation as this will offer them the best foetal and neonatal outcome even if admission to a NICU was required.
- Termination of pregnancy is through either **induction** of labour or **CS** according to obstetric indications, guidelines, facilities, and available team experience (see induction of labour).

B) PRETERM IUGR:

Prolongation of pregnancy till foetal **viability** or until reaching **near term** or **term** is a better option for a **preterm** IUGR foetus after exclusion of serious foetal congenital or chromosomal abnormalities that necessitate immediate pregnancy termination.

During this period of **conservative** management the following measures and guidelines should be strictly followed, until a decision for termination of pregnancy is taken;

1. GENERAL MEASURES:

- *Physical and mental rest:* hospitalization is individualized according to each case
- *Stop smoking* and *improve nutrition*: to improve placental functions
- *Screening for TORCH* and treatment when indicated or if of value
- *Low dose aspirin (LDA);* 78-81 mg daily may help improving placental blood flow

2. REGUALR FOETAL SURVEILLANCE:

- *Daily foetal kick count* done by the patient
- *Bi-weekly Non stress test (NST);* for evaluation of foetal well being
- *Bi-weekly US* for assessment of a *biophysical profile score* and *amniotic fluid index*
- *Weekly US Doppler* for umbilical artery and middle cerebral artery *blood flow* assessment

3. PREGNANCY TERMINATION: Pregnancy is terminated whenever;

- The foetus reaches maturity (term or near term), or
- Foetal surveillance indicates foetal hypoxia with increased chances for IUFD or IPFD
- Termination of pregnancy is achieved weather by induction of labour or CS according to obstetric guidelines and facilities (see induction of labour).

INTRAUTERINE FOETAL DEMISE (IUFD)

IUFD describes foetal death in utero **> 24 weeks** gestation but before onset of labour. It may complicate **<1%** of all pregnancies. Prolonged IUFD carries certain maternal hazards that should be dealt with properly to avoid severe maternal morbidity.

AETIOLOGY

1. **Idiopathic:** In at least **50%** of cases no aetiology can be identified
2. **IUGR,** Preeclampsia **(PE),** and Gestational Diabetes Mellitus **(GDM)**
3. Placental **abruption, umbilical cord** accidents (as true knots, and tight loops around the neck)
4. Erythroblastosis Foetalis **(EBF)**, and some serious foetal congenital anomalies **(FCA)**
5. Foetal and maternal **Infections**
6. Antiphospholipid syndrome antibodies **(APS),** and **Thrombophilia's**

DIAGNOSIS

- IUFD is **suspected** when the mother notices cessation of previously felt foetal movements.
- Examination reveals **absent FHS** by Sonicaid, with a fundal level less than gestational age.
- **US** is the **gold standard** for confirming diagnosis of IUFD;
  - *Recent IUFD:* absent FHR is the only finding
  - *Old IUFD;* SGA foetus with collapsed skull bones and oligohydramnios.
  - *US may suggest* a probable cause for IUFD (FCA, EBF, IUGR, Foetal macrosomia, etc...)

MATERNAL RISKS AND HAZZARDA ASSOCIATED WITH IUFD

a. ***Risk of the aetiological factors;*** as PE, GDM, Placental abruption, infections, etc...

b. ***Risk of coagulopathy:*** prolonged IUFD is associated with thromboplastin release from the placenta, leading to DIC and consumption coagulopathy. Fibrinogen levels are decreased while fibrin degradation products (FDP) are increased, with elevated PT and APTT, finally leading to severe maternal haemorrhage.

MANAGEMENT

A. Watchful Expectancy: labour pains usually start spontaneously 2-3 weeks from IUFD. During this period of watchful expectancy the following measures should be taken:

- *Dealing with predisposing maternal cause*, if present, as GDM, HTN, PE, etc.....
- *Monitoring for coagulopathy*; fibrinogen level, FDPs, platelet count, PT, APTT, etc...

B. Active Management: termination of pregnancy by **induction** of labour or **CS** is indicated in the following conditions;

1. Watchful expectancy period extending more than 3 weeks
2. Difficulties in controlling maternal disease: as GDM, HTN, and PE
3. Clinical or laboratory evidence of an impending coagulopathy or DIC
4. Women who cannot psychologically tolerate watchful expectancy period.

- **N.B.:** Non sensitized RH –negative women should receive an IM injection of 300 ug Anti-RhD immunoglobulin once IUFD is diagnosed since delivery may not occur until > 72 hours.

II. FOETAL MACROSOMIA

The term foetal **macrosomia** is used to describe a newborn who is significantly larger than average, with a birth weight exceeding 4000 gm regardless its gestational age.

Macrosomia may affect around 5% of neonates, however the risks associated increase significantly with a foetal weight > 4500 gm which occurs in < 0.5% of newborn infants.

RISK FACTORS

1. Maternal **diabetes** is the most common and most important risk factor for foetal macrosomia. Gestational diabetes mellitus (GDM) and uncontrolled pre GDM are of more risk than well controlled diabetic patients.
2. **Other risk factors** include; Post-term pregnancy (PTP), maternal obesity (pre-pregnancy weight > 90 kg), rapid weight gain during pregnancy, grand multipara, and a previous LGA baby.

COMPLICATIONS

A) Foetal Complications:

- IUFD (in diabetic macrosomia or serious congenital malformations)
- Birth trauma (shoulder dystocia and brachial plexus palsy)
- Hypoglycemia, polycythaemia, hypocalcaemia and jaundice.

B) Maternal:

- Higher incidence of C.S. deliveries (due to prolonged, or obstructed labour).
- Traumatic injuries of birth canal: (rupture uterus, cervical and vaginal lacerations)
- Postpartum haemorrhage (PPH), weather atonic (due to uterine overdistension), or traumatic (due to lacerations).

DIAGNOSIS

1. **Clinical** estimates of foetal size and weight based on fundal height measurements, are often unreliable and are markedly affected by clinical experience and maternal obesity.
2. **Ultrasound** estimates of foetal weight are reasonably accurate with only 10-15% error range, however US estimates of foetal weight are more difficult in obese women.

PREVENTION

1. Proper evaluation of risk factors during pre-conception visits weight loss in obese women
2. Early diagnosis of GDM and meticulous control of established cases.
3. Control of weight gain during pregnancy at regular antenatal care visits.

MANAGEMENT

1. **During pregnancy:** regular antenatal care, control excessive weight gain, repeated investigation to discover GDM, serial US estimates for growth curves, and anomaly scans.
2. **Induction of labour >37 weeks:** to minimize the need for a CS delivery. This approach is at least 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.





26 PREECLAMPSIA ECLAMPSIA AND HYPERTENSIVE DISORDERS

- *Preeclampsia (PE)*
- *Eclampsia*
- *Gestational hypertension (G-HTN) Chronic*
- *hypertension (C-HTN)*

During **pregnancy** both systolic and diastolic blood pressures show a **constant decline** starting in the late first trimester, reaching a nadir by **24-28** weeks, followed by a **gradual rise** to pre-pregnancy levels by the end of the 3rd trimester. Blood pressure (BP) may also show some changes in relation to posture, stress, emotional state, and method of measurement.

Hypertension (HTN) during pregnancy is diagnosed with a **systolic** BP **140** mm Hg or **more** and/or **diastolic** BP of **90** mm Hg or **more**, measured at **2** occasions at least **4** hours apart after a period of **rest**, preferably in the **sitting** position, using the **5th** Korotkoff sound as a mark for diastolic pressure.

Hypertensive disorders in pregnancy, especially **preeclampsia** and **eclampsia** are considered worldwide a leading cause for maternal and perinatal **morbidity** and **mortality** in obstetrics, with mortality being mostly related to **CNS** haemorrhage.

Classification of Hypertensive Disorders during Pregnancy
▪ ***Gestational hypertension*** *: hypertension alone first occurring > 20 weeks gestation*
▪ ***Preeclampsia*** *: hypertension + Proteinuria first occurring > 20 weeks gestation*
▪ ***Eclampsia*** *: preeclampsia + grand mall seizures*
▪ ***Chronic hypertension*** *: hypertension alone diagnosed before pregnancy or < 20 weeks gestation*
▪ ***Superimposed PE*** *: Preeclampsia developing on top of chronic HTN*

Based on the American College of Obstetricians and Gynaecologists Executive Summary: hypertension in pregnancy 2013

GESTATIONAL HYPERTENSION

Gestational hypertension **(G-HTN)** is diagnosed when sustained **hypertension without proteinuria** occurs, for the first time, in the second half of pregnancy **> 20 weeks** gestation.

G-HTN is not usually associated with specific **symptoms** apart from mild recurrent headaches encountered in some patients.

Laboratory **tests** show no alterations in kidney or liver function tests, with absent proteinuria on repeated urine analysis.

Preeclampsia **(PE)** should be always and repeatedly ruled out, since **30%** of cases with G-HTN may develop PE later in pregnancy.

MANAGEMENT:

- **Conservative** outpatient management with close observation on repeated antenatal care visits and repeated urine analysis to rule out PE.
- **Antihypertensive drugs**; are only indicated with increasing BP levels (Methyldopa oral)
- **Foetal surveillance** for proper evaluation of foetal wellbeing including daily foetal movement count (DFMC), non-stress test (NST) and repeated US (see chapter on foetal surveillance)

PREECLAMPSIA

Preeclampsia **(PE)** is a **pregnancy specific** syndrome of **hypertension** and **proteinuria** occurring for the first time in the **second half** of pregnancy (> 20 weeks gestation). It may complicate **3-7%** of all pregnancies, especially cases with high risk factors. **Eclampsia** describes PE with grandmall **seizures**

DIAGNOSTIC CRITERIA OF PE

a) Sustained elevation of the **BP** to **140/90** mm Hg or **more** on **two** occasions **4** hours apart

b) Proteinuria: > 0.3 g in a 24 hour urine collection, or >1+ on a dipstick test, or a protein/ creatinine ratio of > 0.3.

In absence of proteinuria **PE** can be also diagnosed if **new onset** hypertension **> 20** weeks is associated with a recent **organ dysfunction** manifested by one or more of the following;

- Thrombocytopenia (platelets < 100.000)
- Elevated serum liver transaminases (AST & ALT > twice the norms)
- Rising serum creatinine, CNS symptoms, DIC and pulmonary oedema.

PE is generally associated with the development of rapid onset generalized **oedema**, especially facial and hand oedema, but they are **not diagnostic** of PE being common in normal pregnancies.

RISK FACTORS

1. **Primigravidas:** PE occurs **8 times** more frequently in the **first** than in subsequent pregnancies
2. Multifoetal pregnancy **(MFP)** and hydatidiform mole **(HDFM)** with abnormal placentation
3. Extremes of **age** especially the very young primigravidas and the very elderly ones
4. Diabetes mellitus **(DM)** and marked **obesity**
5. Chronic hypertension **(C-HTN)** and chronic **renal disease** with poor organ perfusion.
6. History of **Previous** PE or Eclampsia or **Family history** of PE or Eclampsia
7. Systemic Lupus Erythematosus **(SLE)** due to small vessel disease.

PATHOPHYSIOLOGY and THEORIES

PE is called a disease of **theories** because genetic, immunologic, vascular, hormonal, nutritional, and behavioral factors have all been proposed as theories for its occurrence. Because the disease starts to **resolve** shortly after delivery, a lot of attention has been payed to the role of the **placenta** and **uteroplacental-foetal interface** in the development of the disease.

The origin of PE is considered multifactorial with **inadequate uteroplacental perfusion** being central to its development leading to placental ischaemia and hypoxia.

During a normal pregnancy, cytotrophoblastic **invasion** of intra-myometrial spiral vessels renders these vessels **wider** and **less responsive** to vasoconstrictor substances, leading to a state of **decreased** vascular resistance and **increased** uteroplacental blood flow.

Inadequate cytotrophoblastic **invasion** of the intra-myometrial spiral arterioles will result in **failure** to establish the normal **low resistance** uteroplacental circulation with a resultant placental **ischaemia** with involvement of secondary **mediators** responsible for;

- Vascular **endothelial** dysfunction
- Local and systemic **vasospasm**
- Activation of the **coagulation** system

Diffuse **vasospasm** is potentiated by an increase in the **vasoconstrictive** PGLs (thromboxane & PGL $F_{2\alpha}$) and a decrease in the **vasodilator** PGLs (prostacyclin & PGL E_2).

PATHOLOGY

Three major pathologic lesions are associated with PE and Eclampsia

1. Lack of **decidualization** of the myometrial segments of the spiral arteries
2. Glomerular capillary **endotheliosis**
3. **Ischaemia**, **haemorrhage**, and **necrosis** in many organs secondary to arteriolar constriction

These characteristic pathologic lesions will result in;

A) Intravascular Volume Constriction; capillary ischaemia will lead to endothelial injury and loss of intravascular fluid volume into the interstitial space causing **oedema**.

B) Decreased Organ Perfusion; utero-placental unit, kidneys, liver, brain, & heart causing;

- IUGR and oligohydramnios
- Decreased renal clearance of metabolites and proteinuria
- Sub-capsular liver haemorrhage and necrosis.
- Cerebral oedema, petechial haemorrhage, and thrombi
- Retinal haemorrhage.
- Vascular endothelial damage leading to platelet thrombosis and DIC in severe cases.

CLINICAL AND LABORATORY MANIFESTATIONS

PE is a disease of **signs** rather than symptoms, with diagnosis based on **BP** measurements together with **laboratory** investigations including **urine** and **blood** samples. It is often a **progressive** disease and its severity is graded both by the severity of hypertension and the degree to which other organ systems are affected. PE is **graded** into mild (non-severe) and severe PE.

CRITERIA FOR MILD (NON-SEVERE) PE:

– Sustained **hypertension > 140/90** mm Hg

– **Proteinuria**: > 0.3 g in a 24 hours, or dip stick test 1-2 +, or Protein / creatinine ratio > 0.3.

– Mild non-specific **symptoms** and **signs:** as recurrent **headaches**, rapid **weight** gain and **bloating,** with pitting lower limb **oedema**.

– Normal or mild changes in CBC, platelet count, kidney and liver functions

CRITERIA FOR SEVERE PE
– ***Severe Hypertension:*** *sustained systolic BP* ***160*** *or* ***more*** *and/or diastolic BP* ***100*** *or* ***more***
– ***Renal Insufficiency:*** *serum Creatinine* ***> 1.1*** *mg/dl or doubling of baseline values*
– ***Elevated Liver Enzymes:*** *AST & ALT increased* ***> twice*** *their normal values*
– ***Thrombocytopenia:*** *Platelet count* ***< 100.000*** */uL.*
– ***Epigastric pain*** *or right upper quadrant pain: due to stretch of liver capsule*
– ***Cerebral*** *or* ***visual disturbances***
– ***Pulmonary oedema***
– ***Oliguria < 750 ml/24 hours***
– ***Foetal jeopardy*** *in the form of asymmetrical IUGR, oligohydramnios and basal FHR changes in NST*

A variant of **severe PE** with high morbidity is the **HELLP** syndrome that describes cases with **H**ypertension, together with **H**aemolysis, **E**levated **L**iver enzymes and **L**ow **P**latelets. It may complicate 5-10% of cases with PE. In contrast to the classic PE presentation the patient with **HELLP** syndrome is more likely to be multiparous, older in age, < 36 weeks gestation with only **50%** having **severe** elevations in their BP.

EVALUATION OF SEVERITY OF PE

- **Symptoms** as; headache, visual disturbances, nausea and vomiting, epigastric pain, etc...
- **Clinical examination** including;
  - BP measurements, weight gain and pitting LL oedema
  - Neurologic examination: for patellar and ankle reflexes to exclude hyperreflexia.
  - Fundus examination: to detect retinal haemorrhage and exudates
- **Laboratory tests:** Oliguria, Proteinuria, CBC, Platelet counts, Liver functions, Kidney functions, and coagulation profiles (bleeding and coagulation time, PT, APTT, INR, etc...).
- **Foetal surveillance:** DFMC, NST, BPPS, and Doppler studies (see foetal surveillance)

MANAGEMENT OF PREECLAMPSIA

The only definitive cure for PE is **termination** of pregnancy. Once the placenta is delivered a rapid decline in most of the signs and symptoms of PE will occur.

The goal of management of PE will be to **prevent** maternal complications of severe PE while minimizing the neonatal hazards of **prematurity**.

The **decision** for **termination** of pregnancy and the route of delivery will be strongly affected by the **severity** and progression of PE, the **degree** of foetal compromise if present, and the **gestational age**.

GUIDELINES FOR MANAGEMENT

a) **Mild** cases with **NON** severe **NON** progressive PE with **NO** evidence of **foetal** compromise, will be managed **conservatively**, not to be delivered unless gestational age is **37** weeks or older.

b) Cases with **Severe** or **Progressive** PE **> 34** weeks, or with associated **foetal compromise**, or with **HELLP** syndrome, should be immediately **delivered** after a brief period of **stabilization**.

c) Cases with **Severe** PE **< 34** weeks in which the **foetus** is **not** compromised, can be offered an expectant management in which the case is **stabilized**, and the BP controlled with **delivery delayed** to **> 34** weeks gestation, whenever possible, to ensure adequate lung maturity.

MEASURES FOR STABILIZATION OF A PATIENT WITH SEVERE PE

- **Hospitalization** is necessary for severe PE until stabilization is achieved or termination of pregnancy is indicated.
- **Antihypertensive therapy:** (see later)
  - Mild non-progressive disease does not need antihypertensive therapy.
  - Cases with **severe** or **progressive** PE will receive treatment to control the rising BP and avoid cerebral haemorrhage. Therapy is immediately started for control of BP, and continues throughout stabilization period, delivery, and early puerperium.
- **Delivery** is indicated whenever pregnancy reaches **37** weeks, or **earlier** if signs and symptoms of worsening disease develop, or if there is evidence of foetal compromise.
- **Initial stabilization** in cases with **preterm severe PE** is achieved through hospitalization and;
  - *Antihypertensive Therapy*: ***Labetalol*** and ***hydralazine*** as drugs of choice (see later)
  - *Seizure Prophylaxis:* ***Mg SO4*** *IV* as loading and maintenance doses (see later)
  - *Enhancement of lung maturity: IM* ***Betamethasone*** *or* ***Dexamethasone*** *24 mg in 24 hr* for minimizing risks of prematurity (see preterm labour and prematurity).
- **Termination of pregnancy** can be achieved by either
  - Induction of labour by IV oxytocin in cases with favourable Bishop score (>7)
  - Elective CS in cases with severe PE, HELLP syndrome, or foetal distress.

ANTIHYPERTENSIVE DRUGS IN PE/ECLAMPSIA

Antihypertensive medications are indicated in **all** cases with **severe** PE (BP ≥ 160/110), as well as all cases associated with **HELLP** syndrome or eclamptic **seizures.** The goal for therapy is to **control** the BP during stabilization period, and **avoid** complications as placental abruption and cerebral haemorrhage.

BP should be lowered gradually (not too rapid) to levels **not lower** than **130/80** mm Hg to avoid a sudden decrease in uteroplacental perfusion that may cause acute foetal distress.

1. **Methyldopa:** is an alpha receptor inhibitor that has long been the drug of choice in the management of **chronic hypertension** and moderate degrees of **PE**. It has been lately reported to be associated with IUGR and postpartum depression (dose: 250 mg orally 2-6 tablets daily)
2. **Labetalol:** is an alpha and beta receptor inhibitor (nonselective beta blocker) that is currently the most popularly used medication for safe and efficient control of BP in cases with **severe PE** and **Eclampsia** (dose: 20 mg oral tablets 1-2 tablets daily).
3. **Hydralazine:** is an efficient drug that acts directly on the blood vessels leading to rapid vasodilatation and lowering the BP. It is used in cases with severe and progressive PE and impending eclampsia, however an IV bolus dose may be associated with a rapid drop in BP which is sometimes hazardous to the foetus as it may compromise placental perfusion.
4. **Calcium Channel Blockers:** act as direct vasodilators and are effective in cases with progressive severe PE especially in preterm PE as they also have a tocolytic effect. They are not contraindicated in association with Mg SO_4, but may be associated with some undesirable side effects as headaches and tachycardia. Dosage: Epilat 10 mg oral tablets twice daily till control.
5. **Magnesium Sulphate (Mg SO_4):** this drug has long been used both IV & IM mainly for seizure prophylaxis in cases with severe PE + hyperreflexia (impending eclampsia). It has also beneficial antihypertensive, sedative, and tocolytic effects. It promotes diuresis, decreases brain oedema, and improves foetal outcome and minimizes cerebral palsy.
   - *Loading Dose:* 5 g slowly IV in 100 ml fluid over 15-20 min, followed by
   - *Maintenance dose:* IV infusion 1-2 g/h till stabilization
   - **N.B.:** for mode of action, toxicity, blood levels, antidote, and monitoring therapy see eclampsia.

PREVENTION AND PREDICTION OF PE

To date there are **No reliable** tests available for the prediction of PE, or its course and severity.

- ***Serum biomarkers*** in the 2nd trimester as pregnancy associated specific proteins (PAP) have been proposed, and promising results are now appearing.
- ***Uterine Artery Doppler*** ultrasound at **18-24** weeks gestation may show **high** resistance flow with **diastolic notching** that may identify up to **75%** of women at risk for PE later in pregnancy
- ***Low Dose Aspirin (LDA)*** 75-100 mg **daily** from **18-34** weeks gestation has been shown to minimize the risk for recurrent PE and IUGR in high risk cases with a previous history of the disease. LDA acts by inhibiting platelet aggregation and inhibition of thromboxane A2 (vasoconstrictor PGL) without impairing synthesis of prostacyclin (vasodilator PGL)

COMPLICATIONS OF SEVERE PE

1. HELLP syndrome, and severe organ dysfunction: renal, hepatic, retinal, and cerebral
2. Oligohydramnios, IUGR, placental abruption, and IUFD
3. Progression to eclampsia
4. Maternal mortality namely due to cerebral haemorrhage

ECLAMPSIA

Eclampsia is the occurrence of **seizures** (convulsions or fits) in a woman with **PE** that cannot be attributed to any other underlying cause or pre-existing disease. It is considered a true obstetric emergency that needs an expert team for proper management.

INCIDENCE AND AETIOLOGY

Eclampsia may complicate **1-2%** of cases of **PE** with an incidence of around 1:2000 deliveries.

The exact aetiology is **unknown** however **severe diffuse cerebral vasospasm** may be the chief underlying factor that causes poor cerebral **perfusion** and cerebral **oedema.**

TIMING and CLINICAL PICTURE

- **Antepartum:** during pregnancy (65%)
- **Intrapartum:** during delivery (20%)
- **Postpartum:** after delivery in early puerperium (15%).



Fig 24-1: Eclamptic fit

The patient with **severe PE** will experience;

- Sudden unexplained **tonic-clonic seizures** that may persist for **1-2 minutes**.
- During the fit the **tongue** may be bitten and **cyanosis** may occur.
- Seizures are followed by a short period of **coma** followed by **recovery**
- Convulsions may **recur** without preceding signs or symptoms.

DIFFERENTIATION FROM EPILEPSY

Epileptic seizures differ from eclamptic fits by having a **history** antedating pregnancy, being preceded by **aura**, and lacking associated **hypertension** and proteinuria.

MANAGEMENT OF ECLAMPSIA

1. **Protect** the mothers **tongue** (mouth gag) and **ensure airway**
2. **Stop convulsions:**
   - **$MgSO_4$** bolus IV dose 5 g followed by **maintenance** with IV infusion 2 g/hr
   - **Diazepam** 5-10 mg slowly IV to control convulsions in absence of $MgSO_4$
3. **Lower diastolic BP** < 100 mm Hg with IV hydralazine and or labetalol
4. **Prompt delivery** irrespective gestational age with either **induction of labour** or **CS** according to obstetric indications and foetal condition
5. **Postpartum care:** $MgSO_4$ should be continued for 24 hr to guard against postpartum seizures.

Mg SO_4 THERAPY AND TOXICITY

- **Mode of action:**
  - Anticonvulsant action through inhibition of neuromuscular transmission
  - CNS depressant with a sedative effect
  - Tocolytic with a mild antihypertensive effect
- **Dose and administration:**
  - Loading dose: 5 g as an IV dose on 100 ml fluid over 15-20 min
  - Maintenance dose: 1-2 g/hr as an IV infusion on 500 ml fluid
- **Therapeutic serum Mg level:** should be kept < 8.0 mg/dL (N: 4.5-9.5 ml/dL)
- **Precautions to prevent toxicity:** ensure the following before giving next dose
  - Normal urine output (> 30 ml/hr)
  - Normal respiratory rate (> 16/min)
  - Normal patellar (knee jerk) reflex
- **Antidote in case of toxicity:** Calcium Gluconate 10%, 10 ml by IV route over 3-5 minutes.

Clinical Features of Mg So_2 Toxicity	Serum Mg levels
– *Loss of patellar (knee jerk) reflex* – *Somnolence and Slurred speech* – *Respiratory depression and paralysis* – *Cardiac arrest*	*8-11 mg/dL* *10-12 ml/dL* *15-17 mg/dL* *30-35 mg/dL*

PROGNOSIS and MANAGEMENT in SUBSEQUENT PREGNANCIES

Eclampsia is considered a complication of severe forms of PE that carries more **risk** for;

a) **Maternal Complications** as: organ dysfunction, DIC, and cerebral haemorrhage.

b) **Foetal Complications** as: placental abruption, foetal hypoxia and IUFD.

During the **postpartum** period, patients with eclampsia should be closely monitored till full recovery has been achieved.

In their **future pregnancies** they are considered at high risk for recurrent PE and dealt with by;

- Close monitoring during prenatal care
- LDA prophylaxis to minimize the risk of recurrent PE and IUGR
- Uterine artery Doppler velocimetry for early detection of cases with diastolic notch.

The current goal in PE and hypertensive disorders in pregnancy is to;

- **Identify** the disease as early as possible
- **Monitor** its effects on both the mother and the foetus
- **Stabilize** the patient if the disease is severe
- **Deliver** the baby before there is major disease induced maternal or foetal complications.

CHRONIC HYPERTENSION DURING PREGNANCY

DEFINITION AND AETIOLOGY

The diagnosis of chronic hypertension **(C-HTN)** requires either known hypertension **before pregnancy** or the development of hypertension **< 20** weeks gestation.

C-HTN may be associated with **2-5%** of pregnancies, the majority of which (> 90%) will have **essential hypertension**, while the rest will have **secondary hypertension** that has renal, vascular, endocrinologic, or behavioral causes as; pheochromocytoma, hypothyroidism, coarctation of the aorta, drug abuse, etc...

Risk factors for chronic hypertension during pregnancy include; obesity, advanced maternal age, positive family history, diabetes mellitus, chronic nephritis, and systemic Lupus Erythematosus (SLE).

MANAGEMENT

Management starts by a thorough history, examination, and laboratory tests to **differentiate essential** from **secondary** hypertension and to exclude **superimposed PE** and **IUGR**.

A) ANTIHYPERTENSIVE THERAPY:

- In many cases BP will decrease to normal in the 2nd trimester and **no medication** will be needed
- Lowering the BP to levels **< 140/90** mm/Hg does not give much benefit, and in fact may decrease uteroplacental perfusion leading to iatrogenic induced IUGR
- Cases with **BP < 160/105** mm/Hg may need only regular follow up with reassurance and life style modification.
- Cases with **BP 160/105** mm/Hg or **more**, will require treatment with **antihypertensive** drugs;
  - ***Alpha Methyldopa;*** has long been considered the safest oral antihypertensive drug used during pregnancy.
  - ***Calcium channel blockers*** and ***Labetalol;*** are now used extensively and are considered safe and highly efficient as well.
  - *B-blocking agents;* are **not** recommended as they may be associated with **IUGR**
  - *Diuretics* are **not recommended** for possible associated plasma volume reduction
  - *Angiotensin converting enzyme inhibitors* are **contraindicated** as they may be associated with foetal congenital anomalies (FCA), renal failure, oligohydramnios, and IUFD.

B) FOETAL SURVEILLANCE for diagnosis of IUGR:

- Daily maternal foetal movement count (DFMC) in the 3rd trimester.
- Twice weekly NST in cases with decreased DFMC
- Serial US examination every 3-4 weeks after in the 3rd trimester for early detection of IUGR, and oligohydramnios, in association with Doppler flow studies of the foetal umbilical artery

C) SUPERIMPOSED PE:

- Superimposed PE will ***occur*** in **15-25%** of cases with chronic hypertension during pregnancy.
- It will be ***diagnosed*** when a significant **increase** in hypertension occurs or if **proteinuria** develops in a previously non proteinuric hypertensive patient.
- ***Management*** will be according to the guidelines for **severe PE**.
- ***Complications include:*** increased incidence of IUGR and IUFD, placental abruption, and end organ failure (hepatic, renal, or cardiac)

PROGNOSIS DURING PREGNANCY

- **Good prognosis:** in cases with **controlled** BP **< 180/110** and good end organ perfusion
- **Bad prognosis with poor outcome;** in cases with **severe HTN** > 180/110 mm/Hg and poor end organ perfusion as with retinopathy, nephropathy, and left ventricular hypertrophy.

SEQUELAE AND OUTCOMES

PE is primarily a disease of the first pregnancy, however women with severe preterm PE are at high risk (40%) for **recurrent PE,** and are the ones that may benefit from the use of antepartum low dose aspirin prophylaxis in subsequent pregnancies.

A **history** of PE raises the risk for cardiovascular disease in later life, so as a history of gestational hypertension will increase the risk of developing chronic hypertension later in life.

Some of the serious **complications** of **PE,** as cerebrovascular accidents and renal failure, may have long term maternal morbidity effects.

The maternal **mortality** rate from hypertensive disorders in pregnancy will vary according to the severity of the disease and the, the socioeconomic standard, and the quality of antenatal care provided.

At present there is **no** proven way for **prevention** of PE, however high quality prenatal care should prevent the majority of severe complications associated with the disease, including eclampsia.

Key points in PE and ECLAMPSIA

- *PE is diagnosed when new onset hypertension in association with proteinuria will occur for the first time in the 2nd half of pregnancy.*
- *The aetiology of PE is unknown but it is related to vascular endothelial damage and arteriolar ischaemia resulting in some degree of organ dysfunction.*
- *The severity of PE is graded by both the degree of hypertension and the degree of organ dysfunction.*
- *In it severe form PE will have serious maternal and foetal morbidity and sometimes mortality*
- *Mild PE will need close observational follow up until either delivery, or progression of the disease*
- *Severe PE will need urgent management by stabilization followed by plan for delivery according to the severity of the disease, the gestational age and foetal condition, before serious maternal or foetal complications may arise.*
- *Eclampsia is a preventable disorder based on early detection and management of PE*
- *Once occurs immediate management by controlling the fits and prompt termination of pregnancy irrespective of gestation age is indicated*
- *There is no place of conservative management or prolongation of pregnancy in cases with eclampsia as it is a life threatening condition*

27 RH ALLOIMMUNIZATION ERYTHROBLASTOSIS FOETALIS

- *Clinical types*
- *Diagnosis*
- *Prophylaxis*
- *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 alloimmunization, 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 foetal 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.
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.

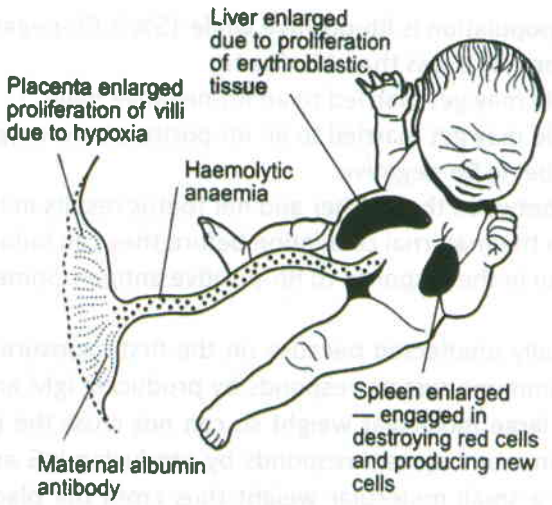


Fig 27-1: Hydrops foetalis

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 Coombs' 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.
  - **Ultrasonography:** May show foetal hepatosplenomegaly, and generalized oedema.
  - **Doppler US:** To assess middle cerebral artery (MCA) peak systolic velocity (PSV) which increases proportionate to the degree of foetal anaemia

DIAGNOSIS AFTER LABOUR

Cord Blood is obtained for Rh grouping and if positive:

- Assess haemoglobin level to detect foetal anaemia (normal 18 gm%), n 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 µg Anti-D I.M. within 72 hours of delivery).
   - At time of abortion, disturbance of ectopic pregnancy, antepartum haemorrhage, amniocentesis, or version (50-100 µg Anti-D I.M.).
   - Recently, 300 µg of Anti-D immunoglobulin I.M. are given at 28 weeks of pregnancy is recommended to prevent sensitisation during pregnancy.

N.B.: 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 Rhnegative ones), correct anaemia, and correct jaundice (remove bilirubin).

28 MEDICAL DISORDERS DURING PREGNANCY

- *Gastrointestinal Disorders*
- *Hepatic Disorders*
- *Urinary tract Infections*
- *Thyroid Disorders*
- *Diabetes Mellitus*
- *Seizure Disorders*
- *Anaemia*
- *Cardiac Disease*
- *Thromboembolic Disorders*

GASTROINTESTINAL DISORDERS DURING PREGNANCY

NAUSEA AND VOMITING OF PREGNANCY (NVP)

NVP is a very common condition that may affect **50-80%** of pregnant women during the **first** trimester being most pronounced between **7-11** weeks. The condition is usually tolerable and is mostly expressed as morning sickness with few vomiting episodes throughout the day.

In most cases the condition is **self-limited** and starts to **decline** spontaneously buy the end of the 1st trimester. In rare cases it may progress to hyperemesis gravidarum (HEG).

- **Aetiology:** still not clearly understood, however NVP correlates with first exposure to **hCG** and its severity correlates with hCG levels.
- **Management:**
  - Reassurance: as the condition is usually self-limiting
  - Instructions: to eat small frequent protein rich meals and avoid recumbent position after meals, as well to avoid known triggers to nausea as; strong odours, coffee & fatty foods
  - Antiemetic therapy: Pyridoxine (vitamin B6), and anti-emetics of first line (as Doxylamine).

HYPEREMESIS GRAVIDARUM (HEG)

HEG is defined as **severe persistent NVP** of the **1st trimester** that **affects** the maternal general condition**,** and is associated with **ketosis** and **loss of > 5%** of total **body weight**. Nausea and vomiting will be severe, recurrent, not related to meals, and not responding to first line antiemetic treatment.

HEG may complicate **1-2 %** of **all pregnancies,** being more prevalent and severe in primigravida **(PGDA)**, multifoetal pregnancy **(MFP)**, and gestational trophoblastic disease **(GTD)**.

- **Aetiology**: exact aetiology is unknown but acceptable theories include;
  - *Hormonal changes* as high hCG and E2 levels
  - *Psychological abnormalities*
  - *Gastric dysrhythmias, and Impairment of mitochondrial fatty acid oxidation*
- **Clinical and Biochemical Changes in Severe HEG:**
  - *Signs of Dehydration:* as hypotension, oliguria, dry tongue, and decreased skin turgor
  - *Electrolyte imbalance:* decreased Na & K levels (due to excessive loss)
  - *Ketonuria* with depleted urinary chlorides (due to starvation ketosis)
  - *Haemoconcentration* and *decreased BUN* (decreased plasma volume)
  - Slightly *elevated liver enzymes* (AST & ALT) and decreased liver glycogen

- **Complications**
  - *Dehydration,* hypotension, pre-renal failure and liver failure in severe cases
  - *Malory Weiss syndrome ;* haematemesis due to vascular tears and rupture with vomiting
  - *Wernick's Encephalopathy:* is a rare complication due to **degenerative** brain changes related to **vitamin B1** (Thiamine) deficiency. It is manifested by delirium, ataxia and nystagmus. **MRI** reveals petechial haemorrhage in Wernick's area in the brain.

- **Management**

A) Mild cases: treatment as NVP in addition to stronger antiemetic drugs

B) Severe cases: hospitalization, IV fluids, and NPO are mandatory;

- IV normal saline and lactated Ringer's solution are important to correct dehydration and electrolyte imbalance, and avoid prerenal failure
- Liver supports and correct ketosis and Ketonuria
- Thiamine (vitamin B1) 100 mg IV diluted in saline infusion
- 2nd line antiemetic drugs are used (most are class B category) including;
  - *Metclorpramide* (Primperan) IM or suppository (Dopamine receptor block)
  - *Meclozine* (Navidoxine) IM, or suppository (histamine receptor block)
  - *Ondansetron* (Zofran) IV infusion, or IM (serotonin receptor block)
- The few patients who do not respond to the above mentioned medical therapy may require **nasogastric feeding** or total parenteral nutrition **(TPN).**
- Induction of abortion has a very limited place in management and is rarely resorted to.

GASTRO-OESOPHAGEAL REFLUX DISEASE (GRD)

Some degree of gastro-oesophageal reflux occurs in nearly 70% of pregnant women, usually described as substernal discomfort aggravated by meals and recumbent position.

- **Management:**

A) Reassurance and instructions as;

- Avoid eating large and late meals
- Avoid recumbent positions especially after meals

B) Drug treatment in resistant cases as;

- Antacids can be taken 1 hr after large meals
- He receptor antagonists as Cimetedine & Ranitidine (Zantac 150 mg orally)
- Proton pump inhibitors as Omeprazole (Controloc 20 mg orally)

HEPATIC DISORDERS DURING PREGNANCY

HEPATITIS B Virus (HBV)

Hepatitis B is a **DNA virus** that is spread by **infected body secretions**.

Maternal infection occurs by sharing contaminated needles, sexual intercourse with infected male partner, or receiving infected blood during the process of blood transfusion.

The disease may be **transmitted** to the **foetus** either;

- *During pregnancy:* Trans-placental infection is **rare** occurring mostly in the 3rd trimester
- *During vaginal delivery:* exposure of the foetus to infected maternal genital secretions from HB surface antigen (HBS Ag) positive mothers is the **common** route of infection

Most **HBS Ag +ve** mothers are **asymptomatic** and are discovered only during prenatal **screening**.

Once detected they should be subjected to tests for **liver functions** (AST & ALT) and **viral load** (PCR).

- **MANAGEMENT HBS Ag positive Mothers**

A) During pregnancy: Women with a **high** viral load should receive **treatment** in the **3rd** trimester to minimize the risk of vertical transmission and protect the new born during vaginal delivery.

B) Vaginal delivery is safely allowed**,** while CS is reserved only for obstetric indications.

C) During first 24 hours postpartum: neonates of HBS Ag positive mothers should receive both;

- *Passive immunization* with HB **immunoglobulin** (HBIG) **and**
- *Active immunization* with HB **vaccine** within the 1st 24 hours after delivery.

D) Breast feeding is allowed in neonates who received immunization in 1st 24 hours.

HEPATITIS C VIRUS (HCV)

Women with **chronic hepatitis C** (HCV +ve) have a very **low risk** of transmitting the virus to their baby during pregnancy (**< 5%** vertical transmission).

Most cases are **asymptomatic** with normal liver functions and are mostly diagnosed either before pregnancy or during prenatal **screening** if performed routinely.

It is important to be **aware** of the presence of the virus in order to **monitor** maternal health during pregnancy and **minimize** the risks of transmission to the baby.

- **Risk of Transmission** of **HCV** increases with;
  - Acute stage (newly acquired HCV infection)
  - Co infection with HIV

During **delivery** to **minimize risk** of transmission prevent the use of instrumental delivery forceps or ventouse and avoid scalp electrodes to protect skin of the baby from harmful abrasions

Hepatitis C infection **does not** usually **harm** the baby during pregnancy or delivery however pregnant women may experience some abnormalities in their liver function tests.

Breast feeding is allowed as it **does not** increase the **risk** of transmission of HCV to the newborn, except in case the mother will have severely cracked and bleeding nipples.

- **Treatment During Pregnancy**

Standard medications for treatment of HCV are **CONTRAINDICATED** during pregnancy as they are unsafe to the unborn baby.

INTRAHEPATIC CHOLESTASIS OF PREGNANCY (ICP)

ICP is an uncommon condition that may affect **1-4%** of all pregnancies. It is characterized mainly by **cholestasis** and **pruritus** in the **second half** of pregnancy (> 20 weeks) **without** other major liver dysfunction.

ICP usually runs a **benign course** with no serious maternal hepatic sequela but has a tendency for **recurrence** in subsequent pregnancies, and is sometimes associated with **meconium** stained amniotic fluid and rarely foetal demise **(IUFD).**

- **Aetiology:** exact aetiology is **unknown** and may be multifactorial, however *Gene mutation* in the MDR3 gene is associated in 15% of cases.
- **Diagnosis:**
  - *Symptoms:* itching > 20 weeks without rash or abdominal pain, is the main symptom, being more intense on the palms and soles
  - *Signs:* Jaundice may be rarely observed
  - *Laboratory tests:* elevated levels of serum bile acids with normal or mildly elevated AST, ALT & bilirubin
- **Management:**
  - *Symptomatic treatment:* with cold baths, emollients, and anti-histaminic drugs
  - *Medical treatment:* with oral ***ursodeoxycholic acid*** tablets is effective in ameliorating pruritus, and reducing serum levels of bile acids and bilirubin.
  - *Postpartum follow up:* rapid improvement occurs with normalization of laboratory tests.
  - *Foetal Surveillance:* Intrapartum surveillance for foetal wellbeing is important and in some cases timing delivery > 37 weeks may be indicated if treatment fails to control the condition.

ACUTE FATTY LIVER OF PREGNANCY (AFLP)

AFLP is a very **rare** but an extremely **serious** condition that may affect 1:14000 of all pregnancies in the **3rd trimester**. It is associated with diffuse micro-vesicular **fatty infiltration** of the liver that may lead to **hepatic failure**.

- **Aetiology:** Unknown, but may be related to an inborn **error** of **metabolism** leading to enzyme deficiency resulting in intrahepatic accumulation of fatty acids (LCHAD).
- **Symptoms & Signs** usually include;
  - *Nausea and vomiting, abdominal pain, and jaundice*
  - *Hypoglycaemia* is frequently present and may be severe
  - *Hypertension* and *preeclampsia (PE)* may be present in up to 50% of cases
  - *Coagulopathy* may develop in late cases with *hepatic and renal failure*
  - *Hematemesis may occur in association with DIC*
- **Diagnostic Tests:**
  - Increased PT & PTT, hyperbilirubinemia, hypoglycaemia, and hyperuricemia
  - Moderate elevation of ALT & AST, BUN, and serum creatinine
  - Liver biopsy is diagnostic, but not usually performed for hazards of bleeding
- **Treatment:**
  - Prompt **delivery** with intensive **supportive care** is indicated once diagnosis confirmed
  - Management in **ICU** unit with specialized medical team
  - The neonate should be tested for enzyme LCHAD deficiency.

URINARY TRACT INFECTIONS DURING PREGNANCY (UTI)

ASYMPTOMATIC BACTERIURIA

This is the **commonest UTI** in pregnancy affecting almost **6%** of all pregnant women.

The condition is usually **diagnosed** during **screening** for pregnant women, between 12-16 weeks gestation, when a *fresh clean catch* urine sample is positive in absence of UTI symptoms. If untreated up to **30-45 %** of cases may develop **acute pyelonephritis**.

- **Laboratory Diagnosis:** positive **urine culture** from a fresh midstream sample showing **> 100.000** colony forming unit (CFU) of a single organism (usually being E. coli).
- **Treatment:** single agent outpatient oral antibiotic according to culture and sensitivity
- **Follow up:** repeat midstream urine examination 3 weeks after treatment to ensure cure.

ACUTE CYSTITIS

This is an acute UTI **localized** to the bladder **without systemic** findings. If not properly treated it may progress to **acute pyelonephritis**, which may cause maternal and foetal hazards as IUGR, PTL, and IUFD

- **Symptoms:** urgency, frequency, and burning micturition
- **Laboratory Diagnosis:** positive **urine culture** showing **> 100.000** CFU of a single organism
- **Treatment:** single agent outpatient oral antibiotic according to culture and sensitivity
- **Follow up:** repeat midstream urine examination after 3 weeks to ensure eradication of infection

ACUTE PYELONEPHRITIS

This is an **acute UTI** involving the **upper** urinary tract with **systemic** findings that usually manifest in the 2nd or 3rd trimesters with pronounced symptoms of acute **cystitis** followed by systemic manifestations as nausea and vomiting in association with **rigors**, **chills**, and **pain** in the flanks

- **Signs**: high fever, tachycardia, and tender loin (right is more common due to dextro-rotation of the uterus with more pressure on the right ureter)
- **Significance:** if not early detected and adequately managed acute pyelonephritis may lead to serious maternal and foetal complications as:
  - Sepsis, anaemia, and pulmonary dysfunction
  - PROM and PTL with marked foetal morbidity and/or mortality
- **Laboratory Diagnosis:** positive **urine culture** showing **> 100.000 CFU** of a single organism
- **Management:**
  - ➢ Hospitalization is necessary with IV fluid infusion, antipyretics and pain killers
  - ➢ IV antibiotics according to culture and sensitivity.
  - ➢ 3rd generation Cephalosporins (Ceftriaxone) may be started immediately until results of culture and sensitivity are available
  - ➢ Foetal surveillance to assure foetal wellbeing

THYROID DIEASE DURING PREGNANCY

PHYSIOLOGIC CHANGES DURING PREGNANCY

- *Physiologic goitre:* increased size with advancing pregnancy due to increased blood flow
- *Decreased plasma Iodine:* increased glomerular filtration rate (GFR) during pregnancy leads to enhanced iodine excretion, and consequently a decrease in plasma iodine.
- *Increased total T3 & T4:* because E2 results in increased thyroid binding globulin (TBG).
- *No change in free T3 & T4.*

HYPERTHYROIDISM

- **Aetiology:** Graves disease, Toxic goitre (nodular or diffuse), and Hydatidiform Mole (HDFM)
- **Clinical Features:** Inadequate weight gain during pregnancy and sometimes weight loss, in association with increased resting pulse, thyroid enlargement, and exophthalmos
- **Laboratory diagnosis:** markedly **elevated** serum total and free **T4** with **normal** or low **TSH** levels
- **Course and Effects on Pregnancy:**
  - *Mild and/or well controlled cases;* usually have no effect on maternal or foetal outcome.
  - *Severe and/or uncontrolled cases:* may be associated with increased incidence of spontaneous abortions, PTL, IUGR, and increased perinatal morbidity and mortality
- **THYROID STORM:** is a life threatening **hypermetabolic** state characterized by pyrexia, tachycardia, and severe dehydration that develops as a complication of untreated cases of hyperthyroidism.
- **GRAVES DISEASE:** is the commonest cause encountered during pregnancy, due to **autoimmune** production of TSH receptor antibodies **(TSHR-Ab)** that causes increased thyroid hormone production independent of thyrotropin. TSHR-Ab can cross the placenta causing foetal hyperthyroidism.
- Diagnosis of Graves disease will include the clinical features of hyperthyroidism (see before) in association with **elevated** levels of free **T4** and **TSHR-Ab**, together with **low TSH** levels.
- **Treatment of Hyperthyroidism:**
  - *Propylthiouracil (PTU)* oral tablets is the drug of choice during pregnancy (50-200 ug/day).
  - *ß-blocking agents*, steroids, and iodine, are used in severe cases.
  - *Subtotal thyroidectomy in 2nd trimester:* in cases resistant to antithyroid medications (rare).
  - *Thyroid Ablation with radioactive iodine* is **contraindicated** in pregnancy because it can cross the placenta destroying the foetal thyroid.

HYPOTHYROIDISM

- *Mild hypothyroidism* does not affect fertility or alter the course of pregnancy.
- *Severe hypothyroidism* is usually due to a **primary thyroid defect** and is commonly associated with anovulation and infertility. If pregnancy occurs it might be associated with increased incidence of spontaneous abortion.
- **Controlled cases** are usually associated with normal pregnancy and foetal outcomes

- **Diagnosis**: TSH levels are **elevated** with normal or **low** T4 values.
- **Treatment:** oral thyroid hormone (Eltroxin) is the drug of choice with an increase of about 30% above the pre-pregnancy dose to achieve adequate control on the disease.

DIABETES MELLITUS WITH PREGNANCY

Diabetes mellitus **(DM)** is a disorder in carbohydrate metabolism characterized by hyperglycaemia and glucosuria. Prolonged disease will be associated with microangiopathy. DM occurs in nearly **1:350** pregnancies

TYPES OF DIABETES MELLITUS IN PREGNANCY

- **Gestational Diabetes Mellitus (GDM):**
  - **GDM** describes carbohydrate intolerance having its **onset** or **first recognition** during pregnancy.
  - It is mostly due to an **anti-insulin** diabetogenic effect of **placental hormones** as human placental Lactogen **(HPL)**, placental **insulinase**, cortisol and progesterone **(P).**
  - Around 35% of women with GDM will develop overt DM in 5-10 years after delivery.
- **Overt Diabetes Mellitus (ODM):**
  - **ODM** describes DM that is known to be present **before** the onset of pregnancy. It may be either;
    - ***Insulin Dependent (type 1):*** juvenile onset, caused by pancreatic islet cell deficiency
    - ***Non-insulin dependent (type 2):*** adult onset, caused by insulin resistance

WOMEN AT RISK FOR GDM

1. Marked Obesity (class 2 or more with BMI > 35)
2. Maternal age > 35 years (elderly gravidas)
3. Foetal macrosomia in a previous pregnancy (foetal weight > 4.5 kg)
4. Unexplained IUFD, repeated FCA, and recurrent pregnancy loss

SCREENING and DIAGNOSIS OF DM IN PREGNANCY

- For **All** pregnant women between **24-28** weeks (maximum hormonal anti-insulin effect)
- **High risk** cases are screened at their **first** antenatal visit and **repeated** at 24-28 weeks

- **Screening Test:** oral glucose challenge tests

  A) 50-g 1 hour oral glucose tolerance test (OGTT);
  - Normal values **< 140** mg/dl: denoting a **negative** screening test for GDM
  - Abnormal values **≥ 140** mg/dl: require repeat screening with a **100-g** 1hour **OGTT.**

  B) 100-g 1 hour OGTT:
  - Normal values **< 165** mg/dl: denoting a negative screening test
  - Abnormal values **≥ 165** mg/dl: denoting a positive test for GDM

- **Diagnostic Tests:**
  - Fasting blood sugar (FBS) **> 95** mg/dl
  - 100-g 1 hour **OGTT** values **> 200 mg/dl** in presence of **FBS** > 95 mg/dl.

MATERNAL RISKS IN DIABETIC PREGNANCIES

1. Difficult glycaemic control due to the anti-insulin effect of placental hormones which is more pronounced in the 2nd & 3rd trimesters
2. Increased incidence of diabetic ketoacidosis (DKA) and preeclampsia (PE)
3. Increased incidence of abortion, PTL, CS, instrumental deliveries, and maternal birth injuries
4. Increased chances for chorioamnionitis, puerperal sepsis and wound infection

FOETAL RISKS

1. Foetal congenital anomalies **(FCA):**
   - Most common are VSD and open NTD
   - Most specific is caudal regression syndrome
2. Foetal **macrosomia** (weight > 4.5 kg):

   Maternal hyperglycaemia leads to foetal **hyperglycaemia** and **hyperinsulinemia**, resulting in enhanced glycogen synthesis, lipogenesis, and foetal protein synthesis.
3. Foetal **birth injuries** during instrumental delivery and **shoulder** dystocia due to macrosomia
4. **IUFD** in cases with prolonged poor glycaemic control and diabetic keto-acidosis **(DKA)**

NEONATAL RISKS AND PROBLEMS

1. **Hypoglycaemia:** due to persistent hyperinsulinemia 2ry to hyperglycaemia
2. **Hypocalcaemia:** due to failure of neonate to increase parathyroid hormone synthesis
3. **Polycythaemia:** due to elevated erythropoietin 2ry to relative intrauterine hypoxia
4. **Hyperbilirubinemia:** due to breakdown of excessive neonatal RBCs
5. **Respiratory distress syndrome (RDS):** caused by delayed pulmonary surfactant production

MANAGEMENT OF DM DURING PREGNANCY

In general diabetic pregnancies are at high risk for poor maternal and foetal outcome unless good glycaemic control is achieved starting from early pregnancy till delivery and puerperium.

During pregnancy DM may be revealed for the first time (GDM), blood sugar levels will become more difficult to control, with a higher tendency to hyperglycaemia, DKA, FCA, polyhydramnios, macrosomia, maternal and foetal birth injuries and higher chances for sepsis during and after delivery.

Management of a diabetic pregnancy entails three important measures to ensure best outcome;

- Proper glycaemic control; including diet, exercise, oral hypoglycaemic drugs and insulin
- Foetal surveillance and exclusion of FCA, polyhydramnios and macrosomia
- Proper timing and preferable route for delivery (NVD or CS)

ANTEPARTUM GLYCAEMIC CONTROL

- **Diet Control:** supplying 30 Kcal/kg/day offers good control in 80% of cases of **GDM**
- **Home** blood glucose **monitoring:** 4 times daily checks by the patient herself to keep
  - FBS **< 90** mg/dl, **and**
  - 1h after meal **< 140** mg/dl
- **Oral Hypoglycaemic Drugs (OHD):** these were formerly contraindicated because of concerns of crossing the placenta and causing foetal and neonatal hypoglycaemia. Recently **Metformin** have been safely recommended for GDM not responding to diet control.
- **Insulin Therapy:** Insulin is a large molecule that does not cross the placenta and can be safely used to maintain normal blood glucose levels in cases with type 1 & 2 diabetes and almost 15% of cases with GDM (with failed diet and/or OHD control).
- **Titrating Insulin Doses:** Total daily insulin units = body weight in Kg x 1. Doses are divided 2/3 in the morning and 1/3 at the evening (NPH and regular insulin 50/50).
- **Glycosylated haemoglobin (Hb A1C):** measures glucose bound to maternal RBCs. It reflects glycaemic control over the life span of RBCs ranging from 60-120 days. Levels should be kept below 7 % (N: 4.5-6.5) and repeated every 4-6 weeks.

ANTEPARTUM FOETAL SURVEILLANCE

1. Screening for Foetal Anomalies:

- Cases of uncontrolled **Overt DM** may suffer hyperglycaemia early in pregnancy and are thus at a higher risk for development of FCA especially NTDs, Cardiac anomalies, and Caudal Regression Syndrome (rare but specific).
- Cases with **GDM** develop hyperglycaemia in the 2nd half of pregnancy and are thus more prone to foetal macrosomia and polyhydramnios.
- Screening for foetal abnormalities include;
  - First trimester **US** scan for NTDs, anencephaly and nuchal translucency (NT)
  - **Double** marker test at 11-13 weeks and **Triple** marker test at 16-18 weeks for trisomies and NTDs.
  - **US** foetal **anomaly**-scan at 18-22 weeks to assess for structural anomalies as cardiac anomalies and caudal regression.

2. US Assessment of Foetal Growth and Amniotic fluid volume: every 4 weeks in 3rd trimester

- **Macrosomia** and **Polyhydramnios** are commonly associated with poor diabetic control
- **IUGR** may be associated with long standing overt DM with vasculopathy.

3.Foetal Surveillance:

- Non stress test **(NST)** and amniotic fluid index **(AFI)** every 2 weeks in the 3rd trimester for cases showing growth disorders and or poor glycaemic control.
- Biophysical profile: performed at the monthly US scan

TIMING DELIVERY IN DIABETIC PREGNANCIES

- In cases with **good** glycaemic control and **no growth disorders** or polyhydramnios, pregnancy can be prolonged even up to **40** weeks and **vaginal delivery** may be allowed.
- In cases with **poor** glycaemic control associated with **growth disorders** and or polyhydramnios, pregnancy should be **terminated** whenever foetal lung maturity is ensured (>37 weeks), or whenever foetal surveillance points to increased risk of IUFD.
- **Corticosteroids** prophylaxis given at 28-32 weeks to enhance lung maturity and minimize neonatal complications (IM Dexamethazone 24 mg/24 hours on 2-3 divided doses).

INDICATIONS FOR CS IN DIABETIC PREGNANCIES

- Foetal **macrosomia** (weight **≥ 4.5** Kg) for prevention of obstructed labour, shoulder dystocia, and foetal birth injuries.
- **IUGR**: for prevention of Intrapartum hypoxia, cerebral palsy, and still birth (SB).
- **Obstetric** indications as Breech, Transverse lie, placenta praevia, contracted pelvis, etc...

CARE OF THE NEWLY BORN INFANT

- Infant of diabetic mother **(IDM)** is more liable to the development of hyaline membrane disease leading to respiratory distress syndrome (RDS).
- 25% glucose I.V. solution given after delivery may be needed to combat the possibility of neonatal hypoglycaemia due to overactive fetal pancreas.

CARE IN PUERPERIUM

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

WHITE CLASSIFICATION FOR DM IN PREGNANCY BY PATHOPHYSIOLOGY

Class A1	*GDM with normal FBS not requiring insulin*
Class A2	*GDM with elevated FBS requiring insulin*
Class B	*Overt DM onset after age 20 years and duration < 10 years*
Class C	*Overt DM onset age 10-19 years and duration 10-19 years*
Class D	*Overt DM onset before age 10 years or duration > 20 years*
Class E	*Overt DM with calcified pelvic vessels*
Class F	*Overt DM with nephropathy*
Class R	*Overt DM with proliferative retinopathy*

SEIZURE DISORDERS IN PFREGNANCY

Seizure disorders may affect **0.5%** of women in childbearing age. They may be either;

- **Partial Seizures:** do not involve both hemispheres
  - *Simple:* with no loss of consciousness
  - *Complex:* with loss of consciousness
- **Generalized Seizures:** involve both hemispheres
  - *Absence type:* duration < 20 seconds formerly known as petit-mal
  - *Tonic-clonic type:* duration lasting up to several minutes formerly known as grand-mal

COURSE DURING PREGNANCY:

- In almost **75%** of cases the condition will remain **controlled** on the same dose of anticonvulsant therapy.
- In around **25%** the condition may **worsen** due to higher hepatic microsomal activity during pregnancy leading to lower blood level of anticonvulsant drugs with seizures occurring more frequently.

Pregnancy complications

- Usually minimal with appropriate prenatal and antenatal adjustment of treatment doses

Effect of Anticonvulsant Drugs on the **Foetus** and the **Newborn**

- Increased incidence of foetal congenital anomalies (FCA) 3-7%
- Increased incidence of cerebral palsy (CP), seizures, and mental retardation (MR)
- Maternal use of phenytoin drug products may lead to neonatal deficiency of vitamin K dependent clotting factors (factors II, VII, IX, and X)

MANAGEMENT

- Extra folic acid supplementation (800 µg daily) before and during early pregnancy
- Monitoring anticonvulsant blood level in each trimester to adjust the dose - Mono drug therapy with the lowest possible dose controlling fits.
- Foetal anomaly screening by US and double and triple markers.

ANAEMIA DURING PREGNANCY

DEFINITION AND PREVALENCE

Anaemia is an extremely **common** condition in pregnancy and postpartum period. In its severe forms it carries number of health risks to both the mother and the child. It is estimated that about **50%** of women world-wide will suffer some degree of anaemia during pregnancy, the vast majority will be related to **iron** deficiency.

During pregnancy anaemia is **diagnosed** with haemoglobin (Hb) levels **< 11** gm/dl in the 1st and 3rd trimesters, and **< 10.5** gm/dl in the 2nd trimester. Haematocrit (HTC) is usually **< 33**

Maternal **signs** and **symptoms** are usually non-specific, but can include: fatigue, pallor, dyspnea, palpitations and dizziness. They are all only prominent in **severe** types of anaemia.

Anaemia is **classified** according to its **severity** as being:

- *Mild: Hb 10-10.9 g/dl*
- *Moderate: Hb 7-10 g/dl*
- *Severe: Hb < 7 g/dl*
- *Decompensated: Hb < 4 g/dl*

CAUSES OF ANAEMIA IN GENERAL

In the simplest of terms, anemia results from either; impaired RBCs production, increased RBCs destruction, or excessive blood loss.

PREVALENCE OF ANAEMIAS IN PREGNANCY

Acquired Anaemia	Hereditary Anaemia
Iron- deficiency anaemia	*Thalassaemias (alpha and beta)*
Anaemia due to acute blood loss	*Sickle cell haemoglobinopathies*
Anaemia of inflammation or malignancy	*Other haemoglobinopathies*
Megaloblastic anaemia (folate & B12 deficiency)	*Hereditary haemolytic anaemias*
Acquired haemolytic anaemias	
Aplastic or hypoplastic anaemias	

- **Iron deficiency** anaemia is by far the **commonest** type of anaemia encountered during pregnancy mainly due to high prevalence before pregnancy together with increasing iron demands, improper dietary intake and inadequate supplementation during pregnancy.

  Anemia due to **blood loss** is the **2nd most** frequently encountered cause in association with;
  - Early pregnancy bleeding; as with abortion, ectopic and molar pregnancies
  - Antepartum haemorrhage (APH); as with placenta praevia and placental abruption
  - Intrapartum haemorrhage (IPH) and Postpartum haemorrhage (PPH).
- **Megaloblastic** anaemia due to Vitamin B_{12} and Folate deficiency rarely occurs in pregnancy especially with the usual vitamin B and Folate supplementation before and during the 1st trimester.
- **Haemolytic** anaemias (acquired and hereditary), beta **thalassaemia**, and other **haemoglobinopathies** are diagnosed during childhood and are rarely encountered during pregnancy.
- **Alpha thalassaemia** (thalassaemia minor) is an exception as it is more common in **Caucasian** and Middle-east population, where a mild form of anaemia will be present not responding to oral or parenteral iron intake and is characterized by a normal serum ferritin levels.

PHYSIOLOGIC ANAEMIA OF PREGNANCY:

During pregnancy both the plasma volume and red cell mass show a significant increase to meet pregnancy demands. However the **increase** in plasma volume exceeds the increase in the red cell mass leading to a state of **haemo-dilution**, usually referred to as physiologic anaemia of pregnancy. These changes are more pronounced by the end of the **2nd** trimester.

By the end of **3rd** trimester plasma volume **decreases** gradually with a relative **increase** in the red cell mass leading to a state of mild **haemoconcentration** with spontaneous elevation of Hb & HTC levels, with correction of physiologic anaemia.

EFFECTS OF ANAEMIA ON PREGNANCY OUTCOME

- **Mild** and **moderate** anaemia do not carry real maternal or foetal risks or hazards.
- **Women** with **severe** anaemia are at **higher risk** for preterm labour (PTL), postpartum haemorrhage (PPH) and puerperal sepsis.
- **Babies** of women with **severe** anaemia are at an increased risk of IUGR, LBW, PTL, and susceptibility to neonatal sepsis.

IRON REQUIREMENTS IN PREGNANCY (nearly 1000 mg)

- Basal iron requirements around 250 mg + expansion of red cell mass around 450 mg
- For transfer to the foetus: 250 mg, and for the placenta: 50 mg

▪ **N.B.:** Because iron absorption from food is only 10% it can't supply all iron needs, so iron supplementation is a necessity in all pregnant women.

LABORATORY WORK UP IN DIAGNOSIS OF IDA

A complete blood count (CBC) showing **microcytic** hypochromic anaemia with low Hb & HTC levels is the mainstay in diagnosis. In addition the following will be confirmatory;

- Low **MCV** < 80 FL/cell
- Normal or high red sell width distribution **RDW** > 15
- Low serum **ferritin** < 30 ng/ml, and low serum **iron** levels
- Increased total serum iron-binding capacity **TIBG**

MANAGEMENT OF IDM ANAEMIA

A) Prevention:

- Proper **diet** rich in iron and vitamin C
- Iron **supplementation** using oral iron preparations.

B) Treatment:

- ***Oral iron therapy:*** Haemoglobin rises from 0.3 to 0.5 g per week.
- ***Parenteral iron therapy:*** only on cases with severe anaemia with either;
  - Poor compliance for oral therapy.
  - Inadequate GIT absorption of iron
- ***Blood transfusion*** (Packed cells are preferred): in cases with acute blood loss, antepartum, intrapartum, or postpartum

N.B.: During Puerperium:

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

CARDIAC DISEASE DURING PREGNANCY

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 CHANGES DURING PREGNANCY

During pregnancy the cardiac output **(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 CO during pregnancy include:

1. Increased stroke volume **40-50%** above the non-pregnant state
2. Increase of pulse rate by **10-12** 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

ASSESSMENT OF A 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 carious teeth (subacute bacterial endocarditis).
- Hypertensive disorders in pregnancy, and thyrotoxicosis.
- Associated cardiomyopathy and tight mitral stenosis.
- History of recent reactivation of rheumatic fever

MANAGEMENT DURING PREGNANCY

- **Frequency:** More frequent than usual, attended by both obstetricians and cardiologists.
- **Examination:**
  - Cardiac assessment and early detection of heart failure.
  - Recognition of risk factors (anaemia, 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 haemorrhage 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 pregnancy 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.

VENOUS THROMBOEMBOLISM IN PREGNANCY

INTRODUCTION AND INCIDENCE

Pregnancy is a known hypercoagulable 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 (hypercoagulability).
4. Pre-existing Thrombophilia (hypercoagulability).
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 (hypercoagulability + 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 Xray. 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 thrombophilia, 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

Pulmonary Embolism **(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 lifesaving. Full I.V. heparinisation 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.

29 FOETAL AND NEONATAL ASPHYXIA

Intrauterine asphyxia
- *Pathophysiology*
- *Causes*
- *Clinical features*
- *Diagnosis*
- *Management*

Neonatal asphyxia
- *Causes*
- *Clinical features*
- *Management*
  - *Prophylaxis*
  - *Active management*

INTRODUCTION:

Intrauterine foetal 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 CO_2, which if allowed to be continued, will result in metabolic acidemia (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 Foetal 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, leads 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) n 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 previa.
- Placental separation as in accidental haemorrhage.
- Placental insufficiency: (extensive degeneration, multiple infarcts & abnormally small placenta).

III. Causes in the umbilical 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 previa.

IV. Prolonged foetal 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 Foetal heart rate (FHR)**
   - *Bradycardia* (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. *Elimination of the cause 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.
   b. Oxytocin infusion, if started, should be discontinued to decrease uterine activity and improve placental perfusion.
   c. 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 O_2 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 neonataorum 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 (1953). 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. **A**ppearance (colour)
2. **P**ulse (heart rate)
3. **G**rimace (reflex irritability)
4. **A**ctivity (muscle tone)
5. **R**espiration (respiratory effort)

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) n 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 newborn'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.



30 FOETAL BIRTH INJURIES

Types of foetal birth injuries
- *Bone injuries*
- *Soft tissue injuries*

Intracranial haemorrhage
Cephalhaematoma
Nerve injuries
- *Facial nerve palsy*
- *Brachial plexus injury*

Visceral and muscle injury

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) <u>Bone injuries</u>:

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. n Fracture humerus n Fracture femur.
- Fracture clavicle.
- Dislocation of hip.
- Dislocation of shoulders.

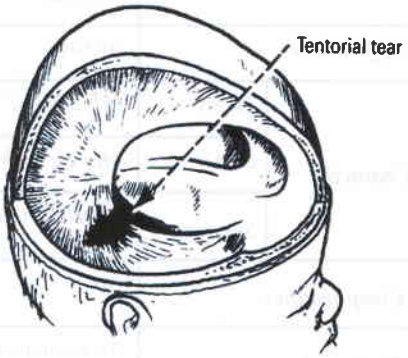
B) <u>Soft tissue birth injuries</u>:

1. Intra-cranial haemorrhage (over compression of cranial bones).
2. Cephalhematoma (instrumental trauma especially ventouse). 3. Nerve Injuries (undue traction on neck, shoulders, and arms).
3. Visceral and Muscle Injuries.
4. Eye Injury.
5. Injury of hymen, or anal sphincter especially in breech presentation, during examination.
6. Skin and scalp Injuries by the scalpel on opening the uterus in a C.S.

I. INTRACRANIAL HAEMORRHAGE

AETIOLOGY:

1. Prematurity, due to:
   - Fragile blood vessels.
   - Hypoprothrombinemia.
   - 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. Haemorrhagic 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 junction 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 fontanel.
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.

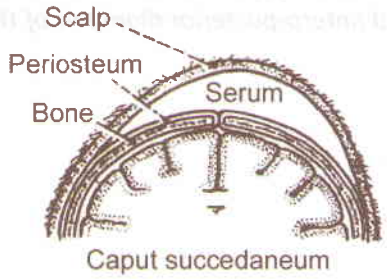
Active Treatment

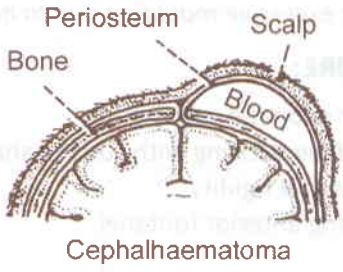
1. Resuscitation with minimal handling.
2. Chloral hydrate, Magnesium sulphate 50% 1 cc and Luminal.
3. NaCl per rectum for oedema.
4. Dehydrating measures even lumbar puncture.
5. Vitamin K for the foetus (1 mg IM).

N.B.: Penicillin is used for prophylaxis against infection.

2. CEPHALHEMATOMA (Sub-periosteal hematoma)

		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)*





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*

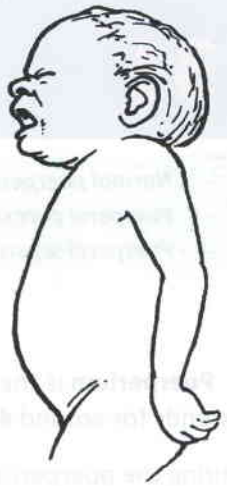
- Absent nasolabial fold.
- Angle of the mouth is deviated to the healthy side.
- Absent blinking on the affected side.

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.


Erb' palsy

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.

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

VISCERAL INJURY:

- e.g. liver, spleen.
- It may occur during breech delivery.

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.

31 THE PUERPERIUM NORMAL AND ABNORMAL

- *Normal puerperium*
- *Puerperal pyrexia*
- *Puerperal sepsis*

NORMAL PUERPERIUM

Puerperium is the period of time that **starts** in the early **post partum** period after delivery and extends for around **4-6 weeks later**.

During the puerperium involution of the genital organs occur hand to hand with several maternal physiologic, metabolic, endocrine, and mental changes.

INVOLUTION OF GENITAL ORGANS

A. THE UTERUS:

- ***Size:*** gradual shrinkage in size together with myometrial autolysis. The fundus reaches the umbilicus immediately after delivery, and is felt at the symphysis pubis (SP) 2 weeks later then finally regains its pre-pregnancy size **4 weeks** post partum.
- ***Endometrium:*** the decidua gradually casts off and is replaced by regenerating endometrium in 4-6 weeks
- ***Cervix::*** gradually reforms and closes within 3-4 weeks. Stretched cervical ligaments involute and become thickened preventing uterine prolapse
- ***Lochia:*** lochia is the postpartum genital **discharge** that consists of blood and necrotic decidual cast.
  - Lochia rubra: profuse and red postpartum discharge in the first 2 weeks
  - lochia serosa: small amount yellowish discharge in 3rd week
  - Lochia alba: scanty whitish discharge that continues till end of puerperium

B. THE VAGINA:

- Vaginal rugae reappear in 3 weeks
- Pelvic floor muscles regain their usual tone and strength in 4-6 weeks **C. THE VULVA:**
- The gapping that occurs during delivery remains for 2-3 weeks then gradually disappears but not completely to pre-pregnant size and shape
- Episiotomy scar is completely healed in 4 weeks if had been performed **D. THE BREASTS:**
- Prolactin (PRL) and nipple suckling initiate milk secretion from the breast glands previously primed by both oestrogen (E) and progesterone (P).
- For the first 2 days the breast secretes colostrum (the first milk produced by the breasts) which is rich in protein and immunoglobulin, while low in carbohydrates and fat.
- Proper milk secretion commences at the 3rd day and may be associated with breast engorgement in the form of pain and congestion.
- Lactation suppresses ovulation and fertility and may be associated with amenorrhoea in up to 50% of lactating women. However this does not preclude the need for the use of an appropriate method for contraception.

PHYSIOLOGIC CHANGES

A. **Blood Pressure:** cardiac output diminishes rapidly after labour with normalization of BP

B. **Temperature:** transient rise < 38°C may occur in 1st 24 hours in prolonged labour, and with breast engorgement in the 3rd day with starting lactation.

C. **Pulse:** Transient rise occurs during labour with rapid decline after delivery

D. **Urinary bladder:** transient postpartum diuresis occurs in 1st few days after delivery sometimes associated with mild stress urinary incontinence (SUI). Retention may occur after use of regional anaesthesia till bladder regains its normal tone.

E. **Bowel:** constipation is common due to lax abdominal muscles and weak pelvic floor, together with gaseous distension. Anal incontinence may occasionally occur due to overstretching of pelvic floor muscles and ligaments but is usually spontaneously corrected by the end of puerperium except of injury of the anal sphincter has occurred.

METABOLIC CHANGES

- **Salt and water:** correction of salt and water retention of pregnancy gradually occurs with promotion of diuresis, relief of oedema, and rapid weight loss ¡ **Blood:**
  - Reduction in plasma volume occurs with rise in haematocrit level within 1 week
  - Increase in leucocytes and platelets immediately after delivery followed by a drop to normalize within 1-2 weeks.
  - Increase in clotting factors during 1st 10 days post partum with an increased risk of deep venous thrombosis (DVT).
- **Urine:** increased urinary lactose excretion with lactation, and increased blood urinary nitrogen (BUN) due to myometrial muscle autolysis.
- **Skin:** pigmentation and chloasma gradually disappear but linea-negra may persist for a long period after delivery.

ENDOCRINE CHANGES

a. Sharp **decrease** in both **E & P** together with **rise** in **PRL** is the main initiative for lactation

b. Suckling induced signals **stimulate oxytocin** release which acts as a milk letting factor

c. Growth hormone, Cortisol, and Thyroxine are slightly elevated and play a role in lactation

d. PRL induced suppression of GNRH is variable, and result in lactational amenorrhoea and decreased fertility according to the intensity of breast feeding

MENTAL CHANGES

1. Emotional liability with mild degree of depression commonly occur during the puerperium
2. Severe postpartum depression rarely occur but might bear serious consequences

POSTPARTUM CARE (PPC) and MANAGEMENT DURING PUERPERIUM

Postpartum acre **(PPC)** for women during the puerperium is an extension of both antenatal care **(ANC)** provided during pregnancy and intrapartum care **(IPC)** during labour and delivery.

1. **General health care:** as physical and mental rest and reassurance
2. **Observation:** for amount of lochia, breast feeding, and occurrence of bleeding or fever
3. **Balanced diet:** providing at least 2500 kcal/day, rich in fibres to minimize constipation
4. **Breast feeding:** encourage suckling by the neonate every 2-3 hours together with additional amounts of water, fluids, and juices.
5. **Breast care:** using cleansing and moisturizing agents to avoid infection and nipple cracking
6. **Abdominal wall exercise:** active exercise starts few days after delivery and throughout puerperium. Abdominal binder is not necessary except in multiparous women with pendulous abdomen.
7. **Perineal care:** keeping a dry pad on the region of perineum is advisable with regular cleansing with antiseptic solutions (Betadine) minimized infection in genital wounds.
8. **Care for episiotomy:** regular cleansing with antiseptic solutions every 4-6 hours together with use of oral antibiotics twice daily for the first 4-6 days.
9. **Bladder care:** encourage the mother for frequent emptying of the bladder till it regains its normal tone and capacity
10. **Bowel care:** avoid constipation by taking extra fluids and high fibre diet. Some laxatives are allowed without affecting the new born especially locally acting glycerine suppositories.
11. **Immunization;** Anti-D immunoglobulin is given to RH negative mothers that deliver an RH positive baby preferably within the first 24-48 hours after delivery (RhoGAM 300 µg/IM).
12. **Discharge from Hospital:** usually within 24 hours from delivery in absence of maternal complications. CS may need 48 hours before discharge until bowel movements return.
13. **Postpartum Exercise:** The aim of puerperal exercise is to restore venous circulation, help genital involution, prevention of genital prolapse, and restoration of normal posture.
14. **Post partum visits:** the patient is best seen 3-4 weeks after delivery in order to reassure general health, follow up genital involution, promote breast feeding, and plan for contraception.
15. **Contraception counselling:** once marital sexual life is resumed a need for a method of contraception is preferable to allow proper pregnancy spacing and family planning. Barrier contraception should be started immediately till a choice for hormonal contraception or IUD is taken according to each cases individually (see contraception in lactating women).

PUERPERAL PYREXIA

DEFINITION

The most accepted definition is;

- A **temperature of 38°C** or higher which
- lasts for **> 24 hours** and or **recur within 24 hours**
- during the **first 21 days postpartum** excluding 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 (mastitis).
3. Urinary tract infection (UTI).
4. Respiratory system infection.
5. Thrombophlebitis and DVT.
6. General causes of fever as typhoid or malaria ***Any case of puerperal pyrexia is considered puerperal sepsis until proved otherwise.***

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.

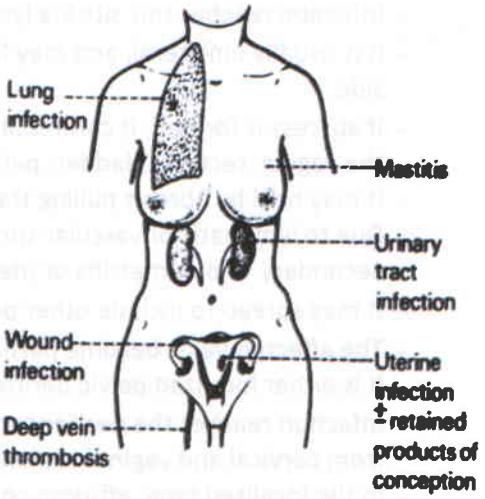


Fig 31-1: Common causes of puerperal pyrexia

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.

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:**
   - Septicaemia 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 oedematous, 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. **Septicaemia:**
   - 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 septicaemia).
   - Breast, chest and throat (for infection).

b. Abdominal:

- Tenderness and rigidity.
- Uterine size: the uterus may be subinvoluted.
- Renal angles for pyelonephritis.

c. Local:

- Lochia: amount and nature (excessive, foul odour).
- Infected lacerations.
- Uterine size and tenderness.
- Pelvic swellings.

3. Special investigations:

- Swabs from vaginal fornix 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:

- Proper 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 placental or membrane 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 & antibiotic cover).
- 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

32 OBSTETRIC PROCEDURES AND OPERATIONS

- Induction of Labour
- Induction of Abortion
- Analgesia And Anaesthesia
- Episiotomy
- Instrumental delivery
- Caesarean Section

INDUCTION OF ABORTION

DEFINITION: Induced termination of pregnancy before fetal viability.

INDICATIONS:

I- Maternal indication as:

a. Severe maternal diseases uncontrolled with pregnancy as cardiac, Diabetes mellitus, Hypertensive disorders

b. Malignancy needing irradiation, surgery, or chemotherapy.

c. Recent intake of a known highly teratogenic drug.

d. Severe mental disorders, disabling the mother for care of her offspring.

II- Foetal:

- Missed abortion, Vesicular mole and lethal congenital anomalies

MEDICAL METHODS FOR INDUCTION OF ABORTION

A. First trimester

1. ***Oral and/or Vaginal PGL E1 (Misoprostol 400 µg)***; 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 µg / 4-6 hours in repeated doses.
2. ***High concentration oxytocin*** (50-300 IU/500 cc over 3 hours) is successful only in cases with ripped cervix.

- **Complications of PGLs:**
  - GIT symptoms: Nausea, vomiting or diarrhoea.
  - 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.

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.

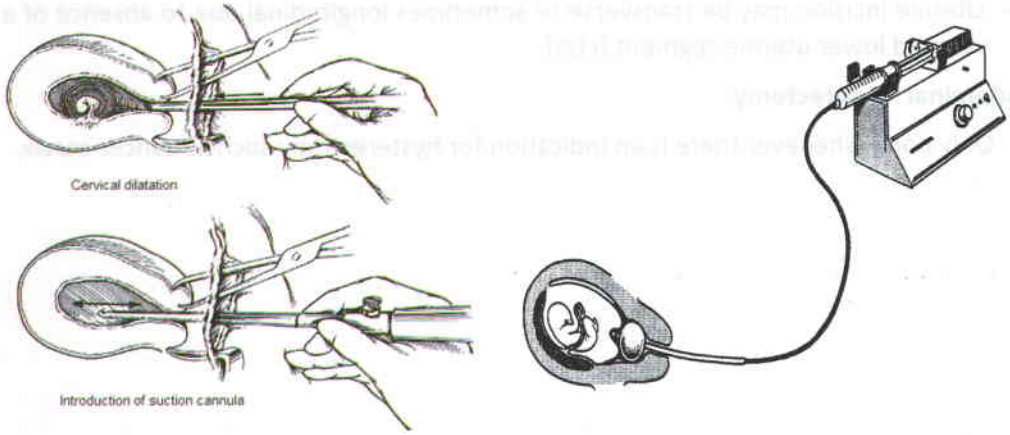


Fig 32-1&2: Induction of abortion

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. Haemorrhage and shock: Mostly due to incomplete evacuation.
5. Neurogenic shock if the cervix is dilated with inadequate anaesthesia.
6. Complications of general anaesthesia.
7. Remote complications as cervical stenosis or incompetence, infertility and intrauterine synechiae.

N.B.: Rh negative women should receive an anti-D IM Injection in cases < 20 wks & 300 mg in cases 20-24 weeks, 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 Pfannensteil 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.

INDUCTION OF LABOUR

DEFINITION: Artificial termination of a pregnancy by inducing uterine contractions and or cervical ripening after foetal viability

INDICATIONS: Whenever continuing pregnancy will harm the mother or the foetus: e.g.;

- Hypertensive disorders (PE / eclampsia).
- Prolonged pregnancy (Post date).
- Compromised foetus (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 favourable for easy dilatation and effacement. It also renders labour more easily inducible whenever indicated. An Unfavourable cervix increases the chances of a difficult prolonged labour, foetal hypoxia and operative delivery.
- The Bishop score takes account of the cervical length, cervical dilatation, cervical consistency, cervical position and station of the foetal head. Scores of < 7 (or 5) indicates PGs use.

The Bishop score

Parameter/score	0	1	2	3
Position	Posterior	Central	Anterior	
Consistency	Firm	Intermediate	Soft	
Effacement	Formed 0- <30%	30- <50%	50-80%	> 80%
Dilatation	Closed	1-2 cm	3-4 cm	> 5 cm
Head station	-3	-2	-1, 0	+1, +2

METHODS OF INDUCTION OF LABOUR

I. Cervical ripening by Prostaglandins (PGLs):

Prostaglandins are effective in inducing cervical ripening (softening of cervical collagen fibres) which effectively results in success of labour induction. *PGL E1 (Misoprostol 25 µg);* vaginal tablet inserted into the posterior fornix is the most commonly used drug.

N.B.: repeated small doses of Misoprostol (25-50 µg) can be used to induce uterine contractions

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.*

II. Amniotomy (Artificial rupture of membranes)

- This is done to initiate or augment labour, or to allow a foetal 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 foetal 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 foetal 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 mIU/min. (10-15 drops/min). This is increased by 1 mIU/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 (C.S., hysterotomy, and myomectomy).

ANALGESIA AND ANAESTHESIA IN LABOUR AND DELIVERY

CAUSES OF PAIN DURING LABOUR:

1. Stretch of the cervix during dilatation
2. Distension and ischemia 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 is 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.

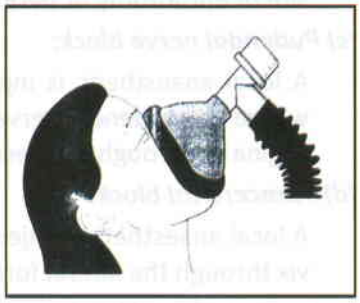


Fig 32-2: Inhalation analgesia

(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.

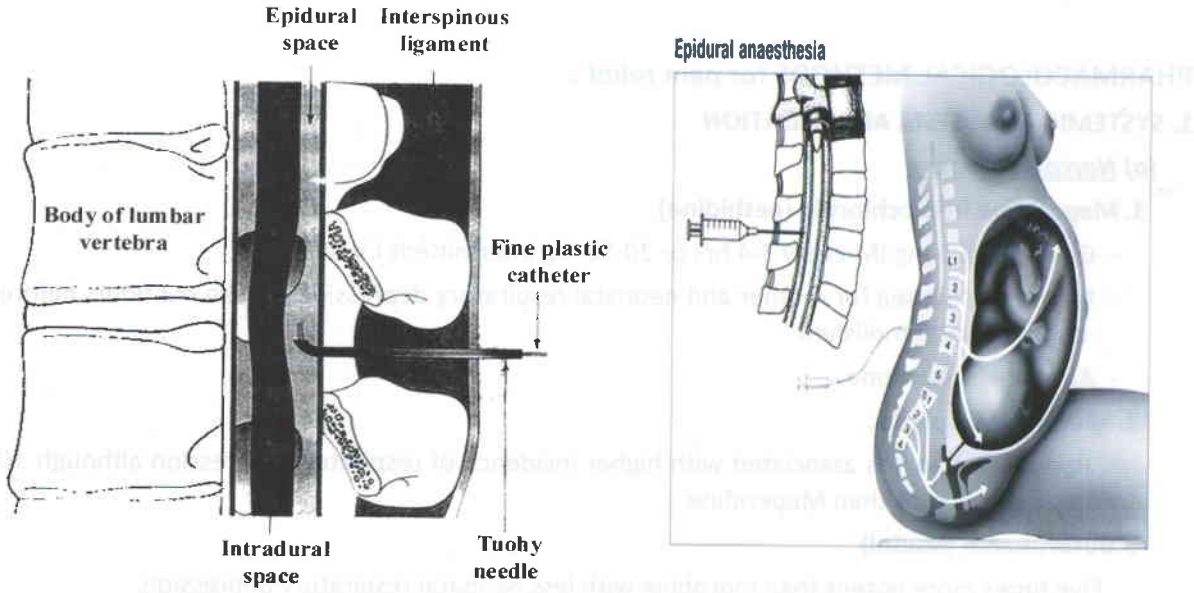


Fig 32-3: Regional Epidural Analgesia

(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 foetal bradycardia, so rarely used nowadays.

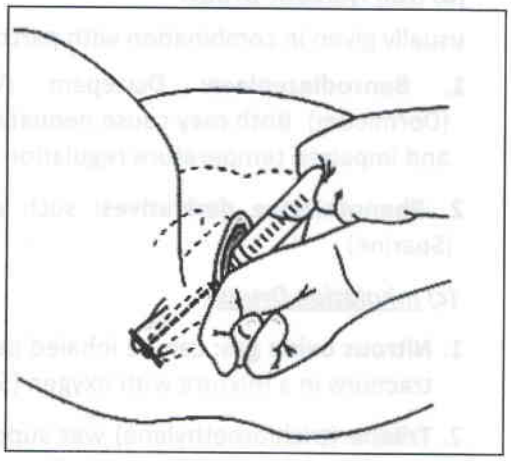


Fig 32-4: Pudendal nerve block

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 CAESAREAN SECTION

A. General anaesthesia:

General anaesthesia using I.V. anaesthetics (as Thiopental sodium or Ketalar) in addition to inhalation gas drugs (as Nitrous oxide and Oxygen) is safe for both mother and foetus 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 foetus 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.

EPISIOTOMY

DEFINITION:

It is an incision of the perineum during labour.

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.

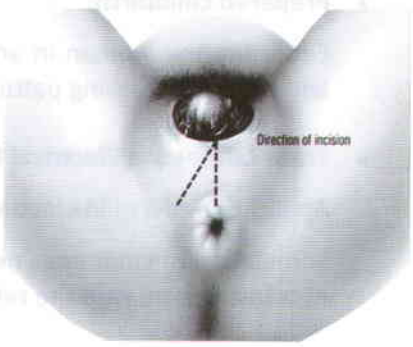


Fig 32-5: Episiotomy incisions

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 foetal 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*

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.

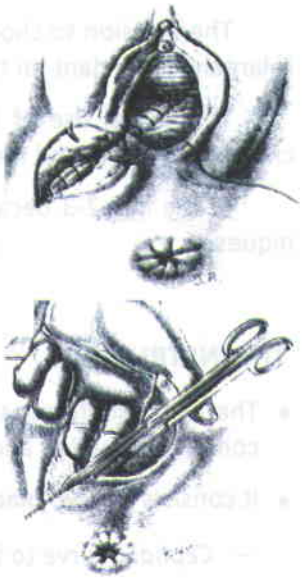


Fig. 32-6: Replace by Episiotomy

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 foetal 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 Caesarean delivery.
3. Extensive perineal lesions such as condyloma accuminata or severe oedema.

INSTRUMENTAL VAGINAL DELIVERY

Instrumental vaginal delivery *refers to the use of specially designed instruments, namely the obstetric forceps and the vacuum extractor, to facilitate delivery of the foetal 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 foetal 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 foetal head.
- It consists of **two blades**, each having **two curves:**
  - *Cephalic curve* to fit on each side of the foetal 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

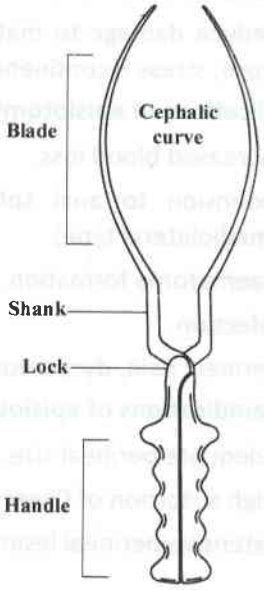


Fig 32-7: The obstetric forceps

TYPES OF OBSTETRIC FORCEPS:

1. The Short forceps "11 inches" (shorted shank & handles):

2. The Long curved forceps "15 inches":

Used for mid forceps rotation and traction (rarely used in modern obstetrics).

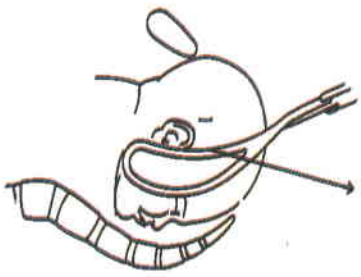


Fig 32-8 short forceps DOA

3. The Kielland forceps:

It is a long forceps designed mainly to facilitate traction and rotation of the foetal 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 foetal occiput.



Fig 32-9: The short forceps for outlet traction

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.

INDICATIONS FOR THE USE OF FORCEPS:

1. Prolonged second stage of labour, when maternal exhaustion is eminent.
2. To shorten second stage of labour, in maternal cardiac or hypertensive disorders.
3. Inadequate maternal expulsive forces, as with the use of epidural analgesia.
4. Foetal 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 32-10: Kielland forceps for rotation & traction in OP positions

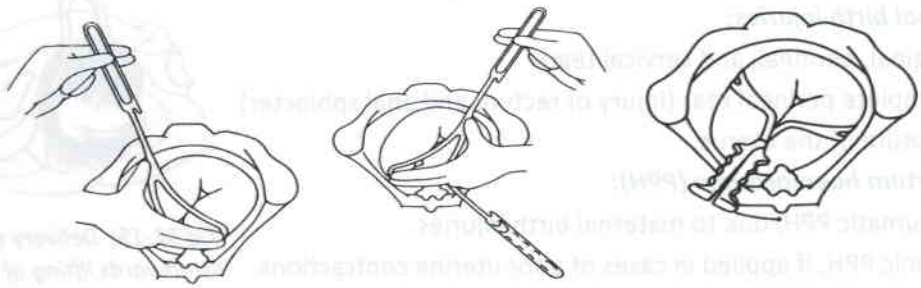


Fig 32-11: Technique of forceps application

PREREQUISITES FOR THE USE FORCEPS:

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.

CONTRAINDICATIONS TO THE USE of FORCEPS:

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 foetal 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.

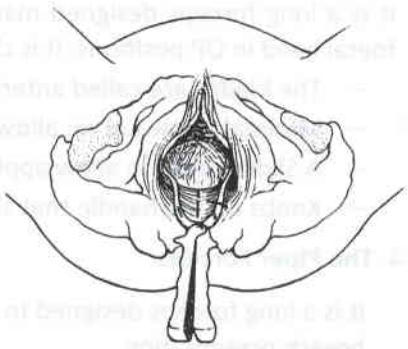


Fig 32-12: traction on foetal head after rotation

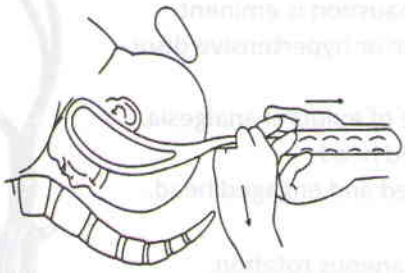


Fig 32-13: direction of traction on foetal head

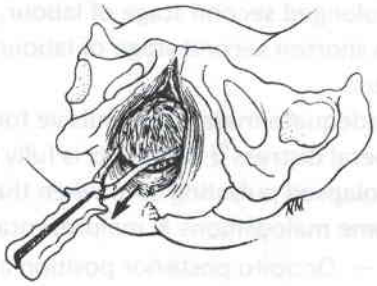


Fig 32-14: traction on foetal head till crowning occurs

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 haemorrhage (PPH):

- Traumatic PPH, due to maternal birth injuries.
- Atonic PPH, if applied in cases of poor uterine contractions.



Fig 32-15: Delivery of the head by upwards lifting of the forceps

B) Foetal complications:

1. Intracranial haemorrhage:

Liable to occur with wrong forceps application, leading to marked foetal head compression and elongation of the mento-vertical diameter. This leads to tear of the vein of Gallen at the junction of the falx cerebri and tentorium cerebelli.

2. Head & Skull injuries: e.g.

- Skull fractures.
- Cephalhematoma.
- Facial nerve injury.
- Facial skin bruises and lacerations.

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 foetal 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.

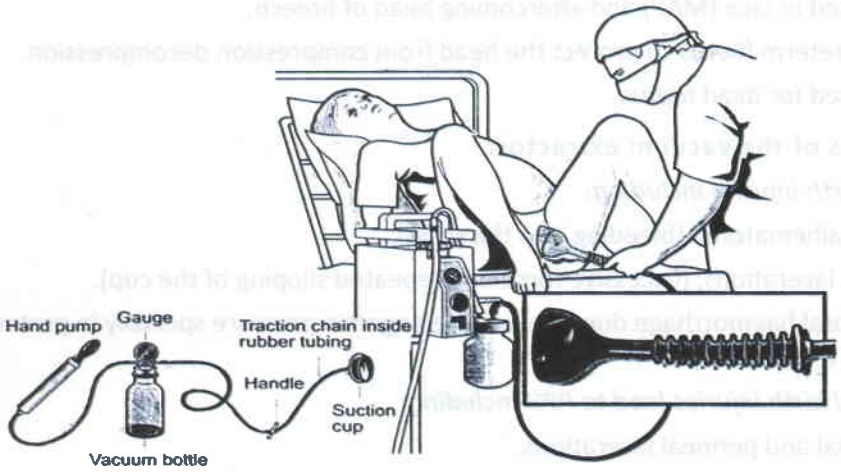


Fig 32-16: Delivery of the head by the use of vacuum extractor he forceps

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 anaesthesia.
8. Living foetus: Vacuum is not suitable for dead foetus.
9. No foetal 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 foetal 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 foetal head, due to limited force.
- Promotes flexion and helps internal rotation of the foetal 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 (MAP) and aftercoming head of breech.
- Used in preterm foetus to protect the head from compression decompression.
- Can be used for dead foetus.

Complications of the vacuum extractor:

1) Foetal birth injuries including:

- Cephalhematoma (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 foetus.

2) Maternal birth injuries lead to PPH including:

- Vaginal and perineal lacerations.
- Cervical lacerations (inclusion of cervical tissue within the cup).
- Rarely rupture of the uterus.

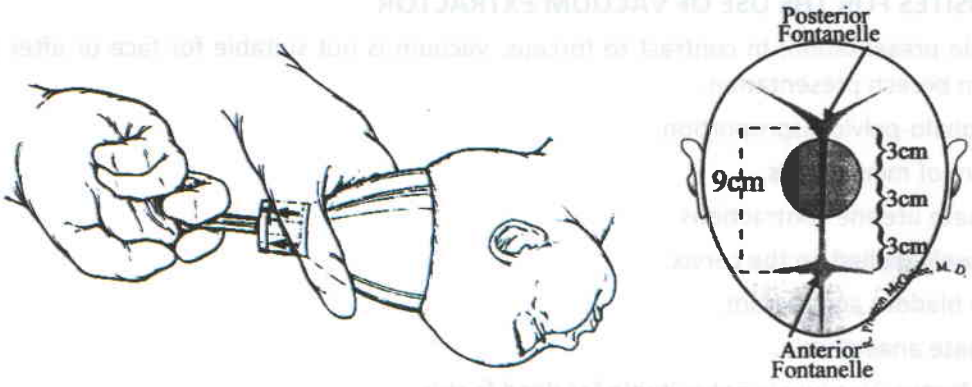


Fig 32-17: application of the ventouse cup on the foetal head and direction of traction

CAESAREAN SECTION

DEFINITIONS

- ***Caesarean section (C.S.):*** is the operation done for delivery of a viable foetus, 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
- ***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
- Emergency C.S: C.S in a patient undergoing trail vaginal delivery or an acute obstetrics complication as APH, eclampsia, etc

TYPES OF C.S.

1. ***Lower Segment (LSCS):*** using a transverse incision in the LUS (Munro Kerr's technique), most common type.
2. ***Upper segment (USCS):*** using a longitudinal incision in the upper uterine segment (UUS), rarely mdicated.



Fig 32-18: LSCS

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 primigravidas).
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 Previa (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 tumours such as low corporeal fibroid or ovarian tumours.

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 foetal haemorrhage 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.
- **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.

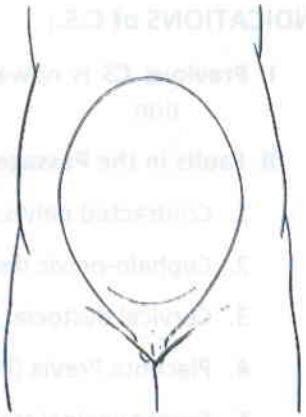


Fig 32-19: Transverse incision in LSCS



Fig 32-20: Delivery of the head in LSCS

- **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.

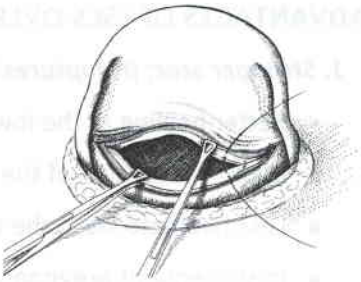


Fig 32-21: 1st layer closure of uterine incision

- 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.

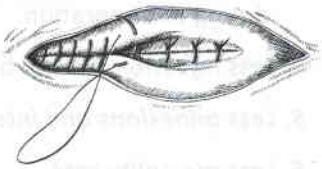


Fig 32-22 : 2nd layer closure of uterine incision

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 haemorrhage.
  - 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 anaesthesia.

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. Anaesthetic 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 haemorrhage: 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: 2-9%; while in LSCS, it is 0.2%-0.9%.
- **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).
  - 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. Haemorrhage: during pregnancy, CS, or postpartum.
  2. Severe anaemia (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 isoimmunisation:
     a. Exchange transfusion after birth.
     b. Intrauterine transfusion in selected cases.
  5. Hypofibrinogenemia (DIC).
- **Precautions:**
  1. Cross matching.
  2. Rate of transfusion = rate of blood loss.
  3. Blood should not be very cold.
  4. For every one litre 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 anaemia.
  5. Transmission of diseases: AIDS and infective hepatitis.

Design and Printing by
MCS Computers - Tel.: 33360906

Publisher
University Book Centre
SAYED MAHMOUD
103 Matahaf EL Manyal, Cairo
Tel: 002-01091111168 - 01223698600