FUNDAMENTALS OF OBSTETRICS – 1

Sex determination

↓
Decided by SRY gene

↓
Distal end of short arm of Y chromosome

↓
Present
↓
Male

↓
Absent
↓
Female

Turner’s syndrome : 45XO → No Y chromosome → Female

Klinefelter’s syndrome : 47XXY → Y chr. Present → male

Other genes that favour

[Testis]
SOX-9

Ovary
RSPO-1
Wnt-4

Best method of sex determination: Karyotyping

↓
Also best investigation to detect detect in chromosomal number

↓
Aneuploidy

↓
Triploidy
69 chromosomes

↓
Trisomy
one extra chromosome(47)

↓
Monosomy
one less chromosome(45)

Genitalia

MC method used for sex determination Looking at external genitalia

Ambiguous genitalia
↓
Sex can’t be determined by genitalia.
**Embryology**

- **Gonads**
- **Ducts**
- **Germ cells**
- **Ext. genitalia**

**Note:** Most of the urogenital system is **mesodermal in origin** →

- **Except urogenital sinus** → **Endodermal**
  - Intermediate mesoderm
  - Urogenital ridge (5 weeks of gestation)
    - **Gonads**
    - **Renal system**
    - **Ducts**

**Lateral plate mesoderm** → **Genitalia** (dorsosomatic part)

Germ cells originate from **epiblast**
Development of gonads

Till 6 weeks → Bipotential

 Derived from genital ridge
(application-in mullerian agenesis ovaries are normal)

Differentiation depends on SRY gene

- Present
  - Medulla from testis 7 weeks
- Absent
  - Cortex form ovaries 8 weeks

First feature which distinguishes between testis and ovaries is formation of testicular cords

Ovaries → Due to absence of Y chromosome

- Turner's
- Klinefelter's
  - Ovary
  - Testes

But for proper development of ovaries both X chromosomes are needed

In Turner's syndrome

- Ovaries present
- Poorly developed - Streak gonads

Development of germ cells

- Y chromosome present → Spermatogonia
- Y chromosome absent → Oogonia
Spermatogenesis:

Spermatogonia (46 XY) → Mitosis → Primary spermatocyte (46 XY) → Meiosis I → Secondary spermatocyte → Meiosis II → Spermatids → Transformation k/a spermiogenesis → Sperms

Spermatogenesis spermatogonia → Sperms

Spermiogenesis spermatid → Sperms

- Begins at puberty
  - 1 primary spermatocyte = 4 sperms
  - 1 spermatogonia = 16 primary spermatocytes
  - 1 spermatogonia = 16 x 4 = 64 sperms

Time taken for spermatogenesis 72-74 days
Average number of sperms produced per day = 100 million sperms/day

Spermiogenesis

1. Transformation of spermatids to sperms
2. No mitosis / meiosis
3. Time taken 12-14 days
Sperm lack endoplasmic reticulum (specially RER)

Sperm

1. Length 55 microns
2. Fertilizable span - 48-72 hrs
3. Sperms attain motility & maturity in caudal end of epididymis (not in testis)
4. Gene for motility CATSPER
5. Ion for motility Ca^{2+}
6. Sperms remain motile in female genital tract 12 hrs
7. Time taken for sperm to reach the ampulla of tube 30 mins

Hormonal support for spermatogenesis

1. Hypothalamus
   - GnRH
     - Pulsatile
     - Released in pulsatile manner
     - Protein for pulsatility
2. Anterior pituitary
   - KISSPEPTIN
   - KISS-IR receptor
3. LH
4. FSH
5. Leydig cells
6. Sertoli cells
   - Testosterone
   - GnRH
   - Inhibin B
7. Spermatogenesis (other hormones LH, FSH)
1. First stimulus to release testosterone hcg - from Leydig cells

2. LH Receptors in ♂ - Leydig cells
   FSH Receptors in ♂ - Sertoli cells

3. Hormones needed for spermatogenesis
   - Main
     - Testosterone
   - Others
     - LH, FSH

**Leydig and Sertoli cells**
* Begin in intrauterine life

- oogonia (46 XX)

  ↓ mitosis

  Primary oocyte

  ↓ meiosis I but gets arrested in diplotene stage of prophase

  Remain arrested till puberty

  K/a Dicylate state → This state is absent in spermatogenesis

  ↓ Primary oocyte gets surrounded by follicular cells

  ↓ Cortex

  ↓ Medulla

  Granulosa cell

  ↓ Theca cells

  Primordial follicle

Tests for ovarian reserve

when number of follicles in ovaries becomes zero

↓

Ovarian Failure / Menopause
At puberty, Hypothalamus
↓
Pulsatile GnRH
↓
LH released from anterior pituitary
↓
LH surge → meiosis I resumes (hormone dependent i.e., LH)
↓
1° oocyte (46XX)
↓
Secondary oocyte (23X)
↓
First polar body is released (23X)
↓
Meiosis II gets arrested in metaphase → over at the time of fertilization
↓
Second polar body (23X)

Female pronucleus (23X)

Ovulation
01:00:17

* Release of secondary oocyte from primary oocyte
LH surge → meiosis I resumed
↓
Ovulation

* Time between LH surge & ovulation 32-36 hours > 24-36 hours
1° Polar body is released at the time of ovulation
2° Polar body is released at the time of fertilization

Oogenesis special points
01:03:42

* Oogenesis begins in intrauterine life (1st week)
* Ova - largest cell in the body = 120 μ
- Size of resting follicle: 0.02 mm
- Size of follicle just before ovulation = 18–20 mm
- Fertilizable span of ovum = 12–24 hrs

Time table of events
1. Germ cells reach yolk sac at 3 weeks
2. Germ cells reach gonads at 6 weeks
3. Oogonia formed at 9 weeks
4. Primary oocyte formed at 12–16 weeks
5. Follicle formation begins by 15–20 weeks
6. Follicle formation completes by 22–24 weeks

**Number of Follicles**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max at 20 weeks of life</td>
<td>1-2 million</td>
</tr>
<tr>
<td>Birth</td>
<td>4-5 lakhs</td>
</tr>
<tr>
<td>Puberty</td>
<td>Only 500 follicles mature in entire lifetime</td>
</tr>
<tr>
<td>6-7 million</td>
<td>1000 follicles undergo atresia every month</td>
</tr>
</tbody>
</table>

**Initial recruitment of follicles**
Hormone independent

**Testosterone**

Testosterone production

- Begins at 7-8 weeks of life
- Maximum at 15 weeks of life
- Decrease
- Remain low till puberty
- @ 17 years of age = Adult male
- Testosterone in males 5-7 mg/day
- Free testosterone 0.5 to 3 J

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Bound = mainly to SHBG/alumnum

\[ \text{ Estradiol} \quad \downarrow \quad \text{DHT} \]

Testosterone $\xrightarrow{\text{Aromatase}}$ Aromatase Estrogen

\[ \text{Amount} = 50 \text{ mcg/day} \]

20% in testes

Rest in adipose tissue

Role of estrogen in male

1. ↑ bone mineral density
2. Closure of epiphysis of long bones
3. Maintenance of body fat
4. Sexual function

Estrogen excess in $\mathcal{O}$: Gynecomastia.

$\downarrow$ Testosterone $\xrightarrow{5\alpha \text{ Sregistrase}}$ DHT (Dihydrotestosterone)

Most potent androgen

Testosterone: DHT = 10 to 15 : 1

Note: Transition meiosis a/k/a meiosis I
FERTILISATION

When the sperm reaches the female reproductive tract:

I) Capacitation - Ability of sperms to fertilise ova
   Occurs in the female reproductive tract
   Begins in - Cervix
   Majorly in - Fallopian tube

Sperms become motile in - Caudal end of epididymis
Sperms become hypermotile in - Fallopian tube

Within 30 mins - Sperms reach the fallopian tube

a) Acrosomal reaction - Sperm attaches to zona pellucida
   (layer around secondary oocyte)
   Facilitated by receptors - ZP1
   ZP2
   ZP3

b) Cortical reaction - Sperm penetrates the zona pellucida and
   reaches cortex of zygote
   With the help of ZP3, Ca²⁺ ions
   And enzymes are released from cortex

4) Zona reaction - These enzymes make zona pellucida
   Impermeable to other sperms

∴ Function of zona pellucida - Prevents polyspermy
Fertilisation – Zygote formation

- The sperm unites with secondary oocyte
  ↓
  Fertilisation – occurs in ampulla of fallopian tube
  ↓
  Zygote is formed
  ← After 20-30 hours
  ↓
  2-cell stage
  ↓
  4-cell stage
  ↓
  8-cell stage
  ↓
  16-cell stage – called morula (mulberry-shaped)

- For 3 days after fertilization → zygote remains in fallopian tube
  ↓
  Nutrition is provided by secretory cells of fallopian tube

- The zygote (morula) moves into uterine cavity with the help of two factors
  Peristalsis in tube (main factor)
  ↓
  movement of cilia.
  ↓
  factors that ↓ tube peristalsis
  ↓
  ↑ risk of ectopic pregnancy
  ↓
  e.g. Progesterone, copper – T, tubal surgeries

- Fallopian tube has three types of cells
  ↓
  Columnar cells
  Secretory cells
  PEG cells – function not known
Implantation

- Fourth day after fertilization / Day -18 of menstrual cycle
  \[\text{morula enters uterine cavity}\]

- 5\textsuperscript{th} day after fertilization
  \[\text{Zona pellucida is lost} - \text{Zona hatching}\]
  \[\text{Fluid enters morula}\]
  \[\text{Forms blastocyst}\]
  \[\text{Attach to endometrium of uterus}\]
  \[\text{Called implantation}\]

Intra decidual sign

- Types of implantation
  - Superficial implantation
    \[\text{Attaches superficially to endometrium}\]
  - Interstitial implantation
    \[\text{Implanted deep inside endometrium}\]
    \[\text{Occurs in humans}\]
On ultrasound - The normal uterine cavity appears as a straight white line

- The blastocyst does not disturb the central cavity complex / uterine cavity
- intra decidual sign
- 1st sign of pregnancy

- The endometrium - in pregnancy is called Decidua
  3 parts
  - Decidua Capsularis
  - Decidua Basalis
  - Decidua parietalis
  - Separates blastocyst from uterine cavity
  - Separates blastocyst from myometrium
  - Rest of the Decidua

**Important points on implantation**

- Implantation occurs in the form of - Blastocyst
- Begins by - 6 days after fertilization (Day 20 of menstrual cycle)
- Implantation window - Day 20 - Day 31
- Completed by - 10 days after fertilization
- m/C site - upper posterior part of uterus
• Implantation is eccentric (on one side)
  unequal growth of uterus
  ↓
  Piskacek sign
  • True gestational sac eccentrically located
  • Pseudogestational sac is centrally located (the whole decidua is thickened)

• Type of implantation - interstitial implantation
• Sign on ultrasound - intradecidual sign
• Hartmann sign/placental sign - Bleeding at the time of implantation

• Phases of implantation

<table>
<thead>
<tr>
<th>Phase</th>
<th>Main transmitter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apposition</td>
<td>Selectin</td>
</tr>
<tr>
<td>Adhesion</td>
<td>Integrin</td>
</tr>
<tr>
<td>Invasion</td>
<td>Metalloprotease</td>
</tr>
</tbody>
</table>

• Thickness of endometrium at the time of implantation: 10-12 mm

• Thickness of endometrium

<table>
<thead>
<tr>
<th>Phase</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>After menstruation</td>
<td>0.5 mm</td>
</tr>
<tr>
<td>At ovulation</td>
<td>2-3 mm</td>
</tr>
<tr>
<td>In secretory phase</td>
<td>5-6 mm</td>
</tr>
</tbody>
</table>

Decidua

• Endometrium after implantation - Decidua.

- Parts
  Decidua Basalis          Decidua Capsularis  Decidua parietalis
  ↓
  • Functions → Site for
    formation of
    future placenta.
    → Forms the
    maternal side
    of placenta.
Fusion of decidua capsularis and decidua parietalis

- Blastocyst grows and occupies uterine cavity
  \[ \downarrow \]
  Decidua capsularis & Decidua parietalis come closer to each other
* Decidua capsularis and decidua parietalis fuse or uterine cavity obliterate
down
At 14-16 weeks of pregnancy

* For twin pregnancy
  a ovum fertilized by 2 sperms
  In same cycle
  Superfetation
  Seen in humans
  Not seen in humans

* Superfetation - in humans - theoretically is possible till
  down
  14-16 weeks

**Blastocyst**

<table>
<thead>
<tr>
<th>Inner cell mass</th>
<th>Trophoblast</th>
</tr>
</thead>
</table>

* Blastocyst has - 58 cells
  5 cells
  Forms entire fetus

53 cells
  Form trophoblast

* The trophoblast - 8 days after fertilization
  Cytotrophoblast
  Near decidua
  Basalis
  Form villi like structure
  Chorion frondosum
  Forms fetal side of placenta

  Synctiotrophoblast
  Rest of chorion
  Prevents PIH
  Hormone factory of placenta

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**Trophoblastic invasion**

- The cytotrophoblast — converts maternal arteries from (extra villous cytotrophoblast) ↓
  - High resistance to low resistance vessels ↓
  - Known as: trophoblastic invasion ↓
  - Occurs in 2 phases
    - Begins ↓
      - At 12 wks
    - 2nd phase ↓
      - At 16 wks

  This maintains uteroplacental blood flow

- If trophoblastic invasion is absent ↓
  - The resistance in maternal vessels is high and volume ↓
  - Causing uteroplacental insufficiency
    - Leads to
      - PIH
      - IUGR
      - Placenta accreta
        - (Pregnancy induced hypertension)
        - (Intra uterine growth restriction)
  - Function of cytotrophoblast controlled by ↓
    - Natural killer cells of decidua

**Double decidual sac sign on ultrasound**

- Blastocyst grows and occupies uterine cavity ↓
  - Indents central cavity complex
  - Decidua capsularis and parietalis — forms a rings around blastocyst ↓
    - Double decidual sac / Ring sign
Inner ring - Decidua capsularis
Outer ring - Decidua parietalis

Seen only in intrauterine pregnancy (true gestational sac)
PLACENTA – 1

Placenta – introduction

1. Human placenta.
   - **Discoid** - Disc-like in shape
   - **Deciduate** - Sheds off after delivery
   - **Hemochorial** - Lies directly in contact with maternal blood

2. Weight of placenta at term - **500 gms**
3. Ratio of weight of placenta : fetus = 1 : 6
4. Weight of placenta = weight of fetus at **17 weeks of pregnancy**
5. Named cells

<table>
<thead>
<tr>
<th>Named structure</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hofbauer cells</td>
<td>Placenta</td>
</tr>
<tr>
<td>2. PEG cells</td>
<td>Fallopian tube</td>
</tr>
<tr>
<td>3. Langhan cells</td>
<td>Cytotrophoblast</td>
</tr>
<tr>
<td>4. Nitabuch layer</td>
<td>Fibrinoid degeneration - between chorion and decidua</td>
</tr>
</tbody>
</table>

* Zika virus can infect Hofbauer cells

Placenta – formation

- Syncytiotrophoblast
- Cytotrophoblast
- Extra-embryonic mesoderm
- Fetal blood capillary in the villi
- Maternal spiral artery opening into intervillous space (IVS) [At Day 15]

- Primary villi
- a"villi
- Tertiary villi

- Trophoblastic shell
- Mesodermal core
- Vascular

- At D₉
- Formed before D₉ - D₁₁
- Day 12 of fertilization

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Placental membrane/Barrier
Structures separating maternal blood from fetal blood

- Outside
- Inside

- Suminutrophoblast
- Cytotrophoblast
- Extra embryonic mesoderm
- Fetal capillary endothelium

- maternal spiral artery is not included in the placental barrier structures
Reason: Cytotrophoblast replaces the lining of maternal arteries

maternal artery converted from high resistance \(\rightarrow\) Low resistance

Trophoblastic invasion
(This prevents PIH (pregnancy induced hypertension))

Placental circulation

- Uteroplacental (in IVS)
  - Established on Day 15
  - Number of spiral arteries opening in IVS
  - \(O_2\) saturation in IVS \(\rightarrow\) 65-75%
  - Partial pressure in IVS \(\rightarrow\) 35-45 mmHg
  - Uteroplacental blood flow at term (36 weeks)
  - Uterine blood flow at term = 750 ml/min

- Fetoplacental (in villi)
  - Established by Day 17 after fertilization
  - Fetoplacental circulation at term
  - Fetal blood flow at term
  - Rate of delivery of oxygen to fetus

*IVS = intervillus space*
Umbilical Artery and umbilical vein:
- Umbilical vein
  - Carries oxygenated blood
  - $O_2$ saturation = 70-80%
  - Remnant $\rightarrow$ Ligamentum teres
- Umbilical Artery
  - Carries deoxygenated blood
  - $O_2$ saturation = 50-60%
  - Remnant $\rightarrow$ medial umbilical ligament

- Remnant of urachus forms median umbilical ligament
- Umbilical artery - medial umbilical ligament (amni)
- Remnant of Inferior Epigastric Artery $\rightarrow$ Lateral umbilical ligament

Anatomy of placenta at term

Umbilical cord $\rightarrow$ Placenta.
- Fetus $\rightarrow$ membranes $\rightarrow$ uterus

Fetal side $\leftrightarrow$ maternal side
- [Comprises of cords and membranes]
- Forms 4/5th of placenta.
<table>
<thead>
<tr>
<th>Fetal side</th>
<th>maternal side</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Formed of chorion frondosum / Cytotrophoblast / Trophoblast</td>
<td></td>
</tr>
<tr>
<td>• Comprises of 1/3rd of placenta</td>
<td></td>
</tr>
<tr>
<td>• Shiny, grey in colour</td>
<td></td>
</tr>
<tr>
<td>• Fetal membranes and umbilical cord is attached</td>
<td></td>
</tr>
<tr>
<td>• Formed by decidua basalis</td>
<td></td>
</tr>
<tr>
<td>• Comprises of 1/3rd of placenta</td>
<td></td>
</tr>
<tr>
<td>• Dull red in colour</td>
<td></td>
</tr>
<tr>
<td>• Lobes [polygonal areas] ↓</td>
<td></td>
</tr>
<tr>
<td>Lobules / Cotyledons</td>
<td></td>
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</tbody>
</table>

Note:

• Functional unit of placenta. → Cotyledons
  The area supplied by the main stem villi and all its branches

• USG for localization of placenta. → 3rd trimester

• Thickness of placenta. →
  - At term = 2.5cms
  - Thickness ↑ by 1mm every week
  - At 40 weeks = 4 cms (40 mm) [maximum]

Thickness > 4cms → Placentomegaly ↓ Causes

Maternal
1. Diabetes
2. Severe anaemia
3. α- thalassemia

Fetal
1. Hydrops fetalis
2. Infections [syphilis, TORCH]
3. Triploidy

Anomalies of placenta

1. Battledore placenta.
   - Normal placenta → Centre of placenta
   - Battledore placenta → Cord attached to margin of placenta (or)
   - Marginal insertion of cord
a. Extrachorial placenta.

Normally, Fetal side = maternal side of placenta

(chorionic plate)    (decidual plate)

Extrachorial placenta $\rightarrow$ Fetal side $<$ maternal side
[Fetal side is surrounded by maternal side in the form of a ring]

a. Types

Circummarginate
Fetal side is surrounded by maternal side smoothly

\[
\begin{array}{c}
\text{maternal} \\
\rightarrow \\
\text{Fetal}
\end{array}
\]

Circumvallate
Fetal side is surrounded by maternal side with a valve-like thickening in between

\[
\begin{array}{c}
\text{valve like}
\end{array}
\]

maternal $>$ fetal side

Fetal side is depressed, thickened than maternal side, valve like 😞

b. Complications

IUGR [Intrauterine Growth Retardation], APH [Antepartum Hemorrhage]

Other anomalies of placenta

00:45:00

1. Succenturiata placenta.

Small part detaches from main part

Connecting blood vessels 😊
1. Placenta spuria
   - Small part detaches from main part
   - No connecting blood vessels

2. Placenta Bilobata
   - Two equal parts of placenta separated from each other
   - Connecting blood vessels

- Also known as placenta bipartite / duplex

Complications of the above anomalies

a° PPH (>24 hrs - <12 weeks after delivery)

management of the above anomalies

Curettage → But, incase of postpartum women

Curettage is the most common

Cause of Asherman syndrome

(Intrauterine Adhesions)

→ Placenta succenturiata

→ Placenta Bilobata
Fenestrated placenta, placenta membranacea

1. Fenestrated placenta.
   - Central portion of a discoidal placenta is missing
     i.e. missing chorionic plate and villi

2. Placenta membranacea.
   - All of the fetal membranes are covered by functioning villi.
   - Placenta develops as a thin membranous structure.
   - Occupies the entire periphery of the chorion.
   - Serious hemorrhage due to associated placenta Previa / Accreta.

Morbidly adherent placenta

- Nitabuch's layer
  - Layer of fibrinoid degeneration which is present between placenta and decidua
  - Limits the penetration of blastocyst
  - Absent Nitabuch's layer → Morbidly adherent placenta
morbidly Adherent Placenta.

* Types

Endometrium [decidua]

myometrium

→ Placenta Accreta

superficially attached
to myometrium]

→ Placenta Increta

[Deep inside myometrium]

→ Placenta Percreta

[Chorionic villi – attached
to serosa]

Pathogenesis:

1. Absent Nitabuch’s layer
2. Absent Decidua Basalis

Risk Factors

1. Placenta previa in present pregnancy [1st highest risk factor]
2. Previous history of cesarean section [2nd highest]
3. Others: History of uterine curettage
   History of uterine surgery – Myomectomy, Hysterotomy

Clinical Presentation

1. undiagnosed case → Presents as a case of refractory PPH

Morbidly adherent placenta – management

1. Diagnosis

* IOC in antenatal period → USG (Trans Vaginal Ultrasound)
  - USG shows → i) subplacental sonolucent area which
    represents Decidua basalis – Absent
Normal placental adherence

Placenta accreta - Absent subplacental sonolucent line

Placenta percreta.

ii) Heterogenous appearance of placenta

iii) Placental lakes are present

Placenta lakes

- Gold standard → MRI
a. Management

- Elective Caesarean/section
  - (Classical Caesarean-section)
  - Reason: Placenta Accreta is common in placenta previa.
  - To prevent excessive bleed

- Hysterectomy
  - If woman refuses

- Leave the placenta which cannot be removed → Autolysis
- Methotrexate
  - [To prevent Gestational Trophoblastic Neoplasia]
Hormones produced by placenta – Progesterone

1. Progesterone
   - Main sources of progesterone
     - Early weeks of pregnancy → Corpus luteum
       - 7-9 weeks or 8 weeks
     - > 8 weeks → Placenta
     - Luteal placental shift - Functional shift from corpus luteum to placenta
   - Life span of corpus luteum - 10 to 12 weeks
   - Precursor of progesterone - Maternal LDL & cholesterol
   - Rate of production of progesterone - 250 mg/day
     - Incase of twin pregnancy - ≥ 600 mg/day
   - In reactions in pregnancy

- Decidual Reaction
- Arias stella reaction
  - i) Glands become hypersecretory
  - + Changes in nuclei
  - Stroma becomes edematous & rich in glycogen
  - ii) Seen in
    - Intrauterine pregnancy
    - Ectopic pregnancy
    - Molar pregnancy

- Progesterone relaxes smooth muscles in pregnancy
**Estrogen**

- Placenta cannot synthesize estrogen using precursors from the mother [Reason: Lack of 17-α hydroxylase enzyme]
  
  \[ \text{17 OH Progesterone (C₁₇ steroid)} \rightarrow \text{Androstenedione (C₁₉ steroid)} \]

\[ \downarrow \]

Required for estrogen synthesis

- **Fetal Adrenal Glands**
  
  \[ \text{DHEA-sulphate (C₂₃ steroid)} \rightarrow \text{Estrogen} \]

  \[ \text{by placenta} \text{ sulphatase, Aromatase} \]

- *most common estrogen during pregnancy* → Estradiol \( \left[ E_2 \right] \)
- *most specific estrogen during pregnancy* → Estriol \( \left[ E_3 \right] \)

- *Role of estrogen*
  - At term: Uterine contraction

<table>
<thead>
<tr>
<th>↑ Estrogen</th>
<th>↓ Estrogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ placental tissue</td>
<td>↓ Absent / Hypoplastic adrenal glands in the fetus</td>
</tr>
<tr>
<td>- Seen in Erythroblastosis fetalis, Rh incompatibility</td>
<td>- Seen in Anencephaly</td>
</tr>
<tr>
<td></td>
<td>i) Deficiency of placental sulfatase, Aromatase</td>
</tr>
<tr>
<td></td>
<td>ii) Fetal demise</td>
</tr>
<tr>
<td></td>
<td>iii) Down's syndrome - ↓ overall production of C₁₉ steroid in fetus</td>
</tr>
</tbody>
</table>

**Human Chorionic Gonadotropin (hCG)**

- *main site for hormone production* → Syncytiotrophoblast
- **Glycoprotein hormone**
  - Hormone with: Maximum glycogen content
- 2 subunits → \( \alpha \) and \( \beta \)
- α subunits
  i) Non-specific
  ii) Similar in LH, FSH & TSH
  iii) Gene → Chromosome 6

- β subunit
  i) Specific [::. β sub unit is measured for pregnancy]
  ii) Gene → chromosome 19

* hCG
  i) Anatomically similar to LH, FSH & TSH
     but
  ii) Functionally similar to LH only

* hCG detection in blood:
  - 8 days after fertilization
  - Day 22 of cycle
  - 5-6 days before missed period

* Most sensitive test to detect hCG → FIA > RIA
  [FIA: Fluorescent Immuno-Assay
   RIA: Radio Immuno-Assay]

**Levels and functions of hCG**

* Levels of hCG:
  - Increase every 48hrs in early pregnancy [nearly doubles]
    ie about 55-65%
  - Maximum hCG levels: 60-80 days (or) 10th week of pregnancy
    ie hCG = 50,000 - 10^4 IU/L
  - hCG starts to decrease after 10th week
    ↓
    minimum at 16 weeks of pregnancy

![Graph of hCG levels over pregnancy weeks]

- 8-10 weeks
- 16 weeks
Low level of hCG is continued throughout pregnancy

- hCG level becomes normal:
  i) After delivery $\rightarrow$ 1-2 weeks
  ii) After abortion $\rightarrow$ 2-4 weeks
  iii) After partial mole evacuation $\rightarrow$ 7 weeks
  iv) After complete mole evacuation $\rightarrow$ 9 weeks

- $T_{1/2}$ of hCG $\rightarrow$ 36 hours
  ($T_{1/2}$ of LH $\rightarrow$ 2 hours)

- Functions of hCG:
  i) Helps to maintain corpus luteum of pregnancy
  ii) Initial stimulus for Leydig cells to produce testosterone
  iii) Prevents rejection of fetus

<table>
<thead>
<tr>
<th>Levels of hCG: ↑</th>
<th>↓</th>
</tr>
</thead>
<tbody>
<tr>
<td>i) Twin pregnancy</td>
<td></td>
</tr>
<tr>
<td>ii) Molar pregnancy</td>
<td></td>
</tr>
<tr>
<td>iii) Choriocarcinoma</td>
<td></td>
</tr>
<tr>
<td>iv) Down's syndrome</td>
<td></td>
</tr>
<tr>
<td>v) Erythroblastosis fetalis</td>
<td>i) Ectopic pregnancy</td>
</tr>
<tr>
<td></td>
<td>ii) All trisomies except Down’s</td>
</tr>
<tr>
<td></td>
<td>iii) Abortion</td>
</tr>
</tbody>
</table>

- urine pregnancy test
  - Sandwich ELISA test
  - Sensitivity: detects hCG with 20 IU/L

- Critical titre of hCG
  The value of hCG at which 100% cases of intrauterine pregnancy has a visible gestational sac
  - TVS: $\geq$ 1500 IU
  - TRS: 6500 IU (>5000 IU)
Cast scenario

1. UPT – Positive, hCG = 1000 IU, TVS = No gestational sac seen
   \[\downarrow\]
   Repeat hCG after 48 hours
   \[\downarrow\]
   hCG level doubles \[\downarrow\]
   but does not double \[\downarrow\]
   Early intrauterine Pregnancy \[\downarrow\]
   Ectopic pregnancy \[\downarrow\]
   Abortion

2. UPT – positive, hCG = 2500 IU, TVS – gestational sac not visible
   \[\downarrow\]
   Ectopic pregnancy

Human placental lactogen

- Also known as HCS (human chorionic somatomodulin)
- Single chain polypeptide hormone
- hPL is similar to GH (growth hormone), PRL (prolactin hormone)
- Detected at 3 weeks of pregnancy
  - Continues to ↑ throughout pregnancy
  - Reaches maximum at 36 weeks
- marker – To check placental functioning
- Hormone which is produced maximum at term → hPL (1g/dl)
- T1/2 – 30-30 mins

- Functions
  i) Promotes lipolysis in mother
     \[\downarrow\]
     ↑ Fatty acids
     \[\downarrow\]
     used by mother for energy requirement
     \[\therefore\] Glucose is spared for fetus
  ii) HPL brings about insulin resistance
     \[\downarrow\]
     ↑ Glucose level
Hormones which bring about insulin resistance:
- HPL (mainly)
- Estrogen
- Progesterone
- Cortisol

**Fetal membranes**

1. Chorion
   - Formed 8 days after fertilization
   - By cytotrophoblast (chorion laevae)
   - Outer layer

2. Amnion
   - Formed 10 days after fertilization
   - Innermost membrane
   - Avascular
   - Maximum tensile strength

3. Yolk sac
   - Site for hematopoiesis in fetus

4. Allantois
   - Diverticulum derived from hindgut and grows in the connecting stalk

Note
PGE$_3$ - is the prostaglandin which is predominant in fetal membranes

**Umbilical cord**

- Derived from connecting stalk
- Average length - 55 cms
- Range - 30-100 cms
- Short cord - <30 cms
- PH - 7.2
- Connective tissue of the cord - Wharton's Jelly
- Folds of Hoboken
- Early intrauterine life - 4 vessels present
  - Right and left umbilical artery
  - Right and left umbilical vein

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Later

Right umbilical vein - Disappears

At the time of birth

1 umbilical vein

2 umbilical arteries

- Most common vascular anomaly of a cord → Single umbilical artery

- Single umbilical artery
  - More common in diabetic, twin pregnancy
  - Not an insignificant finding
  - ↑ chances of malformation (CVS + Renal)
  - Malformation + single umbilical artery

  Trisomy in fetus

- Velamentous insertion of cord
  - Cord 1" attaches to fetal membranes and then to placenta and before attaching to placenta, it divides
  - In such case, whenever membranes rupture

  Cord gets damaged (umbilical artery and vein also gets damaged)

  Fetal blood loss

  Vasa previa = ↑ perinatal mortality

- Since fetal blood exits through mother's vagina

  Often confused with placenta previa
Singer alkali denaturation test

1) Fetal blood $\rightarrow$ HbF - Resistant to alkali and acid
   - Seen in vasaprevia

2) Maternal blood $\rightarrow$ HbA - Sensitive to acid and alkali
   - Seen in placenta previa

Test done:
- Singer alkali denaturation test / APT test: done if woman presents with bleed per vagina (PV)

  Reagent - NaOH/KOH added

  Colour - remains same
  Blood resistant to alkali
  HbF
  Vasaprevia

  Colour - becomes brown
  Hemolysis occurred
  HbA
  Placenta previa

- To diagnose antenatally [no bleed PV]: Doppler USG
  - Management of vasa previa
  - Cesarean section

Prognosis of vasa previa.
- ↑ perinatal mortality
AMNIOTIC FLUID AND ITS DISORDERS

Amniotic Fluid:
- Specific gravity: 1.008 - 1.010
- Osmolality: 250 mosm/L
- pH: 7.2 - 7.4 (alkaline)
- Rate of turnover of Amniotic Fluid: 500 ml/hr
- In Premature rupture of membranes, pH of amniotic fluid helps to differentiate it from acidic pH of vaginal discharge.
- Nitrazine paper test
  1. Amniotic fluid (PROM) - Nitrazine paper turns blue (due to alkaline pH)
  2. Vaginal fluid - Nitrazine paper remains yellow (due to acidic pH)

Volume of Amniotic Fluid:
- At 12 weeks - 50 ml
- At 16 weeks - 250 ml
- At 20 weeks - 400 ml
- At 32 weeks - 1 litre - maximum at 32 weeks
- At 36 weeks - 900 ml
- At 40 weeks - 800 ml - At term
- At 42 weeks - 800 ml - Post term
- Max. value of amniotic fluid - 32 > 34 wks
- As pregnancy advances - volume of amniotic fluid decreases.

Major contributors of Amniotic Fluid:
- Period of gestation
  - Early weeks (1st trimester): Maternal plasma
  - 12 - 20 weeks: Fetal skin (Transudate)
  - ≥ 20 weeks: Fetal urine
    - Major contributor overall - Fetal urine
- Urine production begins in the fetus at 12 weeks
- Keratinisation of fetal skin at 22 - 25 weeks of pregnancy
- Normally Amniotic fluid is colourless but at term it is straw colored
Colour of amniotic fluid
1. Green coloured (meconium)
2. Golden colour (Bilirubin)
3. Tobacco juice colour
4. Saffron coloured, yellowish green
5. Dark coloured

Conditions
- Fetal Distress
- Breech presentation / Transverse lie
- Listeria infection
- Rh incompatibility
- Intrauterine death
- Post dated pregnancy
- Abruptio placenta

- Amniotic fluid is kept in balance by fetus (swallowing)
- Composition of amniotic fluid: 99% water
- No Nutritive Function

Measurement of amniotic fluid
- Best investigation: USG
  Divide abdomen into 4 quadrants
  \[\text{measure the largest vertical pocket in each quadrant (in cms)}\]
  \[\text{Add } a + b + c + d = \text{AFI (Amniotic Fluid Index)}\]
- Normal AFI = 5 - 24 cms
- If ≤ 5 cm = Oligohydramnios
- If ≥ 25 cm = Polyhydramnios
- Most Sensitive indicator of measurement:
  Single largest vertical pocket (overall)
  \[\text{Normal: } a \leq 8 \text{ cms}\]
  \[< a \text{ cms: Oligohydramnios}\]
  \[\geq 8 \text{ cms: Polyhydramnios}\]
- Normal volume of: 800ml - 2 liters
  \[< 800 \text{ ml: Oligohydramnios}\]
  \[\geq 2 \text{ liters: Polyhydramnios}\]
Oligo and polyhydramnios

**Oligohydramnios**
- mc. of mild oligohydramnios: Idiopathic
- mc. of severe oligohydramnios

  **Congenital malformation**
  ↓
  **Renal Anomalies**

**Polyhydramnios**
- mc. of mild polyhydramnios: Idiopathic
- mc. of severe polyhydramnios

  **Congenital malformation**
  ↓
  **malformations >>**
  NTD-neural tube defects

**Oligohydramnios—other causes**

D - Drugs: Indomethacin, ACE inhibitors
L - Intrauterine growth retardation (IUGR) (Brain sparing effect)
M - Leaking following Amniocentesis

  ↓ blood flow to fetus
  ↓
  **fetal renal blood flow**
  ↓
  **GFR**
  ↓
  oligohydramnios

P - Premature rupture of membranes
P - Post term pregnancy
A - Amnion nodosum*, Chromosomal abnormalities - Triploidy, Trisomy 13*
R - Renal anomalies

1. Multifetal pregnancy
   a. Maternal diabetes mellitus
      ↓
      **Fetal Hyperglycemia**
      ↓
      **Polyuria → Polyhydramnios**

3. High fetal cardiac output:
   a) Fetal Anemia
      - Rh incompatibility
      - Parvo virus infection
      - Hemolysis
   b) α - Thalassemia
   c) Fetal Bartter syndrome

4. Swallowing Defect:
   a) Esophageal atresia
   b) Duodenal atresia
   c) Intestinal obstruction
   d) Cleft lip, cleft palate
   e) Trisomy 18 - mc. trisomy
   f) Trisomy 18 - IUGR + Polyhydramnios
5. When CSF leaks into amniotic fluid:
   6. Chorangioma of placenta.
   7. TORCH infections

**Effect of oligo & polyhydramnios on pregnancy**

1. Oligohydramnios
   \[ \downarrow \]
   Less space
   \[ \downarrow \]
   Fetal parts grow properly
   \[ \text{mc problem: Pulmonary Hypoplasia} \]

2. Amniotic band sequence:
   \[ \downarrow \]
   Less Amniotic fluid
   \[ \downarrow \]
   Amnion wraps around some body structure like a band
   Devascularisation of that part
   \[ \text{mc problem: Limb Amputation followed by Craniofacial Defects} \]

If oligohydramnios occurs in late pregnancy:
\[ \downarrow \]
Organogenesis complete
\[ \downarrow \]
But less space
\[ \downarrow \]
Cord gets compressed
\[ \downarrow \]
Blood supply decreases
Fetal Distress $\rightarrow$ meconium in amniotic fluid

\[ \downarrow \]

meconium Aspiration syndrome

2. Congenital talipes equinovarus (CTEV) - Compression defect

* Time for Delivery in
  * Idiopathic
    * Oligohydramnios $-$ 37 - 38 wks + 6 days

Management of oligohydramnios:

1. Amino infusion with normal saline.

1. Serial Amniocentesis: Only in pregnant females with excessive respiratory discomfort

2. Drugs that can be used to manage polyhydramnios $\rightarrow$ Indomethacin (C/I beyond 32 weeks of pregnancy)

3. After 32 weeks, the chances of closure of ductus arteriosus is increased by 50%, whereas below 32 weeks the risk is less and reversible.
FETUS – 1

- Gestational age = Fetal age = Period of Pregnancy

1st day | 14th day | 28th day
- 1st day: Day of last menstrual period
- 14th day: Ovulation/ fertilisation
- 28th day: missed period

- Calculate Pregnancy
- Definition of Growth period

- On the day of missed period - pt is 4 weeks pregnant
- Fertilisation happened 2 weeks back from the missed period
- An event – happens from x days from day of fertilization. That event will happen x days + 2 weeks of pregnancy

- E.g: 1. Trophoblast divides into cyto and Syncytiotrophoblast 8 days after fertilisation.
  Gestational age – 2 weeks + 8 days

  2. Cardiac activity of fetus seen at 4 weeks of pregnancy,
     i.e, 3 weeks after fertilisation

Fetal growth periods

- Defined from the day of fertilization and not from the 1st day of LMP

<table>
<thead>
<tr>
<th>Growth Period</th>
<th>Normal Definition (From Day of Fertilization)</th>
<th>Definition as per period of Pregnancy (+2 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fertilised Ova</td>
<td>From Day of fertilization upto 2 weeks of pregnancy</td>
<td>uptill 4 weeks of pregnancy</td>
</tr>
</tbody>
</table>
FETUS - 1

- Gestational age = Fetal age = Period of Pregnancy

<table>
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<tr>
<th>1st day</th>
<th>14th day</th>
<th>28th day</th>
</tr>
</thead>
</table>

- 1st day = Day of last menstrual period
- Ovulation / fertilisation
- Missed period

- Calculate Pregnancy → Definition of Growth period
- On the day of missed period → pt. is 4 weeks pregnant
- Fertilisation happened 2 weeks back from the missed period
- An event – Happens from x days from day of fertilization. That event will happen x days + 2 weeks of pregnancy
- E.g: 1. Trophoblast divides into cyto and syncytiotrophoblast 8 days after fertilisation.
  Gestational age = 2 weeks + 8 days
  a. Cardiac activity of fetus seen at 5 weeks of pregnancy.
  i.e., 3 weeks after fertilisation

Fetal growth periods

- Defined from the day of fertilization and not from the 1st day of LMP

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</tbody>
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2. Embryonic Period

From 3wks after fertilisation and up to 8wks after fertilisation from 8weeks → 10weeks of pregnancy

Most teratogenic period is Embryonic period.

3. Fetal period

29wks after fertilisation upto delivery 11 weeks of pregnancy and upto delivery

The total duration of pregnancy = 280 days / 40wks / 9months + 7 days

Trimesters

1st trimester 14 weeks 3rd trimester
up to 13 weeks 28 weeks
+ 6 days 40 weeks

2nd trimester
14 weeks 27 weeks + 6 days

Estimation of fetal age

- Clinically by assessing the fundal height

- Mother experiences a lot of respiratory discomfort at 36 weeks.
- After 36 wks → Head of the fetus goes down into pelvis

Height of the uterus decreases
• At 40 weeks, the height of the uterus corresponds to 32 weeks pregnant uterus. Height → Respiratory discomfort ↓
   This is known as lightening/welcome sign

USG estimation of fetal age:
• Best method
  • Trimester    Best Parameter
  • 1st trimester Crown rump length
  • 2nd trimester Biparietal Diameter > head circumference
  • 3rd trimester Femoral length

• The earlier the USG is done in pregnancy, the more accurately it predicts the fetal age
• Suboptimally dated on USG: if a female has not got any USG done before 20 weeks of pregnancy
• EDD Should not be changed based on USG except in cases of IVF

Crown – Rump length

• It is the measurement taken from the vertex of the fetus to the coccyx
• CRL + 6.5 mm = Gestational age in weeks.
• CRL + 4.5 = Gestational age in days.
• CRL increases by 1 mm/week
• Smallest CRL at which embryo is visible 4–5 mm
• If CRL ≥ 7 mm: no cardiac activity → missed abortion

Biparietal diameter

• Measured from the outer table of one side to the inner table of the fetal skull on the other side

Hasse rule:
• ≤5 months of pregnancy: \( \sqrt{\text{Fetal length}} \) → Gestational age
• >5 months: \( \frac{\text{Fetal length}}{5} \) → Gestational age
• <5 m: Fetal length → vertex to coccyx → CRL
• >5 m: Fetal length → vertex to Heels → Crown Heel length
Assessment of weight of fetus

- clinically - Johnson formula
- USG - Shepherd formula
  - Hadlock formula

Assessment of fetal growth:

- USG
  - Abdominal Circumference of fetus:
  - Abdominal circumference should be measured in a plane where P, U, S are visible
  - P - Portal Sinus
  - U - Umbilical vein
  - S - Stomach

- Abdominal circumference is the Only parameter which tells us about the growth of fetus. It can be IUGR or macrosomia.
- If Ac > 235 cms - Macrosomia.

- Normally \[
\frac{\text{Femoral length}}{\text{Abdominal circumference}} = 20-40\%
\]

- Ratio increased: IUGR
- Ratio decreased: Skeletal dysplasia
### Fetal hematopoiesis

<table>
<thead>
<tr>
<th>Site</th>
<th>Time of Pregnancy</th>
<th>Main Hb Formed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yolk sac</td>
<td>Uptill 6 wks of pregnancy</td>
<td>Grower 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grower 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Portland Hb</td>
</tr>
<tr>
<td>Liver</td>
<td>Upto 20 weeks</td>
<td>HbF</td>
</tr>
<tr>
<td>Bone marrow</td>
<td>After 20 weeks</td>
<td>HbA</td>
</tr>
</tbody>
</table>

**Fetal Erythrocytes:**

1. Early intrauterine life: Fetal RBC's
   - Nucleated and macrocytic (MCV= 180 fl)
   - Later the size decreases to 105 fl; but this does not happen in aneuploid fetuses

2. Difference between adult & fetal RBC:
   - Fetal RBC's are large in size and short life span (90 days)
   - At the time of birth, the Hb of the baby → 16-18 g/dl
     - 75-80% HbF
     - 20-25% HbA
   - But by 6 months of age HbF will be <1%

   - HbF
     1. Less of α, γ DPG
     2. Less carbonic anhydrase (CA)
     3. Higher affinity for O₂
     4. Resistance to acid & alkali
   - HbA
     1. More α, γ DPG
     2. More CA
     3. Less affinity for O₂
     4. Sensitive to acid and alkali

   - Forms basis to two tests
     - APT test/Singer's alkali denaturation test
     - Neihauer-Belke test
Apt test
1. Helps to differentiate between placenta previa and vasaprevia
2. Reagent: 1% NaOH (or) KOH
3. Qualitative test
4. Differentiating b/w fetal and maternal blood

Kleihauer betke test
1. Help to calculate the dose of Anti-D in Rh-ve females
2. Reagent: Citric acid phosphate buffer
3. Quantitative
4. Differentiate b/w fetal R.B.C and maternal R.B.C

Major events in fetal development

<table>
<thead>
<tr>
<th>Event</th>
<th>Seen at</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fetal gross body movements</td>
<td>7 weeks</td>
</tr>
<tr>
<td>2. Gonads formation (Testis&gt;ovaries)</td>
<td>7 weeks</td>
</tr>
<tr>
<td>3. Glucagon synthesis</td>
<td>8 weeks</td>
</tr>
<tr>
<td>4. Internal genitalia (m or f)</td>
<td>10 weeks</td>
</tr>
<tr>
<td>5. Breathing movements</td>
<td>11 weeks</td>
</tr>
<tr>
<td>6. External genitalia</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>12 weeks</td>
</tr>
<tr>
<td>male</td>
<td>14 weeks</td>
</tr>
<tr>
<td>7. Insulin production</td>
<td></td>
</tr>
<tr>
<td>8. HP axis</td>
<td>12 weeks</td>
</tr>
<tr>
<td>9. Fetal urine</td>
<td></td>
</tr>
<tr>
<td>10. Meconium production</td>
<td>16 weeks / 4 months</td>
</tr>
</tbody>
</table>

Fetus produce only IgM, begins at 20 weeks
IgM and albumin cannot cross placenta.
Igs transported to fetus at 16 weeks

* Surfactant production begins at 20 weeks, present in amniotic fluid by 28 weeks
FETUS – 2

- USG during pregnancy
  - Help to confirm pregnancy
  - To differentiate intrauterine, ectopic pregnancy and twin pregnancy
- USG – High frequency – Better Resolution
  Low frequency – Better Penetration
- Early Pregnancy: Frequency – 5-10 MHz
- Late 1st and 2nd trimester: 4-6 MHz
- 3rd trimester: 2-5 MHz
- Alara principle: Expose the pregnant female to USG as low as reasonably advised
- Routine USG: B mode
- To see cardiac activity: M mode

<table>
<thead>
<tr>
<th>Sign</th>
<th>TVS</th>
<th>TAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gestational Sac</td>
<td>4 weeks + 1 day to 4 wks + 3 days</td>
<td>5 weeks</td>
</tr>
<tr>
<td>2. Yolk sac</td>
<td>5 weeks</td>
<td>5.5 weeks</td>
</tr>
<tr>
<td>3. Cardiac activity</td>
<td>5-6 weeks</td>
<td>6-7 weeks</td>
</tr>
</tbody>
</table>

Intrauterine Pregnancy                Ectopic pregnancy
1. True sac is seen                  1. Pseudogestational sac is seen
2. True sac is located eccentrically a. Centrally located.
3. True sac increases in size every day a. Pseudo sac do not grow
4. Double decidual sac sign -ve       4. -ve

1st sign of Pregnancy on USG: Gestational sac
1st sign of Intrauterine pregnancy on USG: Yolk sac
- Mean sac diameter ≥ 2.5 mm: no fetal tissue / yolk sac / no cardiac activity

  ↓

  Blighted ovum

- If CRL ≥ 7 mm + no cardiac activity: missed abortion
- USG helps in detecting congenital malformations

### Congenital malformations and USG

**USG in pregnancy**

- **Routine USG**
  - Type 1 USG
    - Can detect only few congenital malformations
    - Anencephaly can be detected

- **Type 2 USG / TIFFA** (Targetted Imaging for Fetal Anomalies)
  - Can detect all congenital malformations
  - Done in all pregnant females
  - Best time: 16-20 weeks

#### Anencephaly and USG:

- Earliest time to be detected on USG: 10 weeks
- Best time to be detected on USG: 14 weeks
- Signs of anencephaly on USG: Frog eye sign, Mickey mouse sign

#### Signs of Spina bifida on USG:

- Small BPD
- Ventriculomegaly
- Frontal bone scalloping - Lemon sign
- Elongation and downward displacement of cerebellum - Banana sign
- Size of ventral defect ≥ 7 mm
  ↓
  Pathological
  ↓
  Omphalocele  Gastrochisis
  
  - The defect in omphalocele and gastrochisis continues after 12 weeks of pregnancy
  
  - Omphalocele  Gastrochisis
    ↓  ↓
    As covered by sac, defect will have  Defect will have
    smooth contour  irregular contour
  
  - Best time for USG to localise the placenta – 2nd trimester

Other important points about anencephaly:
- Vault or Skull – Absent
- Adrenal glands – Absent or Hypoplastic
  ↓
  ↓ DHEA
  ↓
  ↓ Estrogen production by placenta
  ↓
  Post term Pregnancy
  
  - Polyhydramnios → Increased chances of preterm labour
  
  - MC presentation in anencephaly: Face
• Chances of Recurrence
  • After 1 neural tube defects (NTD): 4%
  • After 2 NTD’s: 10%

• Anencephaly is more common in Female fetuses
• Macrosomia, Respiratory Distress – mc in male fetuses

Prevention of NTD’s:
• Folic acid Supplementation

Management of Anencephaly: Medical termination of pregnancy

**Folic acid supplementation in pregnancy**

- Prevents NTD’s
- Prevents Megaloblastic anemia.
- Prevents IUGR
- Prevents Abruptio Placenta
- Dose of folic acid in pregnancy:
  - 400 mcg/day
  - 1 mg/day
  - 4 mg/day
    [Therapeutic dose]

1. Given to all pregnant females with no history of NTD (Prophylactic Dose)
1. Pregnant female with megaloblastic anemia
1. Pregnant female with h/o NTD baby

2. All pregnant females with Diabetes
2. If pregnant female has a° relative with h/o NTD
2. Pregnant female on antiepileptic drug which increases the chance of NTD
  (Eg: Valproate)

3. All pregnant on antiepileptics which do not cause NTD’s (Eg: Lamotrigine)
3. Pregnant female on any drug which decreases the folic acid absorption
  (Eg: Trimethoprim, Sulfasalazine)
3. If pregnant female has sickle cell anemia
- Prophylactic dose of folic acid - 400 mcg
  \[\downarrow\]
  Prevent NTD
  \[\downarrow\]
  Ideally should be given 3 months before pregnancy
  (atleast be given 1 month before pregnancy, should be continued till
  3 months after pregnancy)

- Tablet provided by Govt of India - 500 mcg

- RDA of folic acid in pregnant female - 500 mcg

- Pregnant female with h/o NTD in previous pregnancy
  \[\downarrow\]
  4 mg/day (Therapeutic dose)
  \[\downarrow\]
  Atleast given 1 month before pregnancy and continue
  3 months after pregnancy
  \[\downarrow\]
  After 3 months, 400 mcg/day given throughout pregnancy

**Diagnosis of Anencephaly:**

- Anencephaly
  \[\downarrow\]
  Screening test
  \[\downarrow\]
  - Maternal serum AFP
    - Routinely done at 15-20 weeks
    - Best done at 16-18 weeks
    - Most Sensitive Biochemical marker
  - Diagnostic test
    - Earlier: Amniocentesis (Invasive)
    - Now: Level 2 scan

**Alpha fetoprotein**

- Early weeks - Synthesised by fetal yolk sac
- Later - by fetal GI & liver
- Major source - Fetal liver
- AFP is present in
  - Fetal serum: Highest at 13 weeks
  - Amniotic fluid: Highest at 13 weeks
  - Maternal serum: Highest at 32 weeks
• t½ = 5-7 days
• Screening test: maternal Serum AFP
  (≥ 2.5 multiples of median (MOM) → +ve
  In twin pregnancy - ≥ 3.5 MOM → +ve

↑ AFP
1. NTD
2. Abdominal wall Defect
3. Pilonidal sinus

↓ AFP
1. Diabetic
2. G - Gestational trophoblastic diseases
3. O - Maternal obesity, overestimated GA
4. A - Abortion
5. T - Trisomy
MATERNAL ADAPTATIONS IN PREGNANCY:
GENERAL METABOLIC CHANGES

Weight gain in pregnancy

- Weight gained in pregnancy 10 - 12 kg (2.5 kg)
- Net weight gained 5-6 kg
- Factors influencing weight gain
  1. Ethnicity
  2. Socioeconomic status
  3. Parity
  4. Pre pregnancy weight
- Smoking doesn’t affect weight gain during pregnancy
- Total amount of water retained in pregnancy 6.5 L

↓ Edema in Pregnancy (physiological pitting)
  ↓ Plasma osmolality (10 m osmol)
  ↓ Conc. of Na⁺, K⁺ in pregnancy inspite of Na⁺, K⁺ retention

Fetus is dependent on mother for

1) Glucose
   ↓
   ↓ Insulin resistance in mother
   ↓ HPL
   ↓ 24-48 wks of Pregnancy
2) Calcium
   ↓ Ca absorption
   ↓ Vit D requirement
3) Thyroxine
   ↓ Dose should be ↑ by 30-50% in pregnancy

Pregnancy is a diabetogenic state
Pregnancy = Fasting hypoglycemia + Post prandial hyperglycemia
Note: 1. Vit D requirements in
   1. Adults  - 2.5 mcg (100 IU)
   2. Children - 5 mcg (200 IU)
   3. Pregnancy - 10 mcg (400 IU)

   a. ↑ Ca requirement in pregnancy
   RDA of Ca⁺ = 1200 mg

Fat metabolism in pregnancy

- Hyperlipidemia in pregnancy
  - VLDL ↑
  - Cholesterol ↑
  - HDL ↑
  - Lipoprotein ↑
  - Apolipoprotein ↑

Changes in genital tract during pregnancy

<table>
<thead>
<tr>
<th>uterus</th>
<th>Non pregnant female</th>
<th>Pregnant female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Weight</td>
<td>50 - 60 gm</td>
<td>1000 gm</td>
</tr>
<tr>
<td></td>
<td>(Williams = 1100 gm)</td>
<td></td>
</tr>
<tr>
<td>2. Length</td>
<td>7 - 5 cms</td>
<td></td>
</tr>
<tr>
<td>3. Volume</td>
<td>5 - 10 ml</td>
<td></td>
</tr>
<tr>
<td>4. Shape</td>
<td>Pear shaped</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
- Due to rectosigmoid in left side, there is dextrorotation of uterus
**Uterine contractility**
- During second trimester: **Braxton hicks Contraction**
  
  1. Sporadic
  2. Infrequent
  3. IUP = 5 - 25 mm Hg
  4. Near term → False labour pain due to ↑ in frequency

**Uteroplacental blood flow**
- **Mid term** = 450 ml/min
- **Term** = 500-750 ml/min

<table>
<thead>
<tr>
<th>Normal Pregnancy</th>
<th>PIH</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Spiral artery: Vasodilation Angiogenesis</td>
<td>No angiogenesis</td>
</tr>
<tr>
<td>Blood flow ∝ (radius)*</td>
<td>↓ VEGF, ↓ PGF</td>
</tr>
<tr>
<td>↑ VEGF, ↑ PGF</td>
<td>↑ S - FLt 1</td>
</tr>
<tr>
<td>2. Levels of VEGF &amp; PGF are ↓ by receptor proteins</td>
<td>↓ No</td>
</tr>
<tr>
<td>S - FLt 1 (Soluble FMS like tyrosine Kinase 1)</td>
<td>*</td>
</tr>
<tr>
<td>3. ↓ Peripheral vascular resistance</td>
<td></td>
</tr>
<tr>
<td>Progesterone</td>
<td>↑ No</td>
</tr>
<tr>
<td>4. Characterised by refractoriness to the pressure effects of Angiotensin II</td>
<td>-</td>
</tr>
<tr>
<td>5. Micro RNA (mir 34, mir 17 - 92) Play important role in remodelling of spinal Arteries</td>
<td>-</td>
</tr>
</tbody>
</table>
• New predictors of PIH
  ↓ VEGF
  ↓ PGF
  ↑ S - FLT 1
  ↓ NO

Cervical changes in pregnancy

- Columnar epithelium of endocervix moves out & cover the squamous epithelium of exocervix → Eversion of cervix Ectropion

- During pregnancy, cervical canal is closed by a mucus plug
  ↓ at the time of labor, when cervix dilates
  ↓ Release of mucus plug + blood
    Show → Definitive sign of true labour

• Thick cervical mucus is due to progesterone
  ↓ Beaded appearance on slide [if membranes have ruptured then amniotic fluid shows fern like pattern on slide]
Vaginal changes in pregnancy

1. Chadwick Sign - Bluish discoloration of vagina.
2. ↑ Doderlein’s bacteria - Lactobacilli
   
   ![Diagram]
   
   - Glycogen in vaginal epithelium → ↑ Lactic acid → ↑ acidity → ↓ PH → Normal PH = 4.5 → In preg. = 3.5
   - Pathogenic bacteria ↓

   - Candida can survive in acidic media. ∴ most common vaginitis in pregnancy is Candidiasis

Ovarian changes in pregnancy

- Ovulation is suspended
- α Subunit of hCG is similar to LH & FSH

   ![Diagram]

   - B/L, multiple Theca lutein cysts
   - Also seen in
   - ↑ hCG
   - Placentomegaly
   - Hyper thyroidism
   - Preeclampsia
   - Molar pregnancy
   - Diabetes
   - Chorio carcinoma
   - Rh iso immunization
   - Twin pregnancy

Changes in breast in pregnancy

1. Montgomery tubercles - modified sebaceous glands
2. Colostrum has everything more in comparison to breast milk except K - Potassium
   - F - Fat
   - C - Carbohydrate
- Extra caloric requirement in preg: +350 kcal/day
  Lactation: +600 kcal/day
- BMR ↑ by 10-20% in pregnancy
MATERNAL ADAPTATIONS TO PREGNANCY: HEMATOLOGICAL CHANGES

Hematological changes during pregnancy

<table>
<thead>
<tr>
<th>Increased</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ 40 - 45 %</td>
<td>- Hemodilution ↓</td>
</tr>
<tr>
<td>→ Maximum - mid</td>
<td>↓</td>
</tr>
<tr>
<td>trimester</td>
<td>Physiological anemia</td>
</tr>
<tr>
<td>* Plasma volume increase in 40%</td>
<td>→ Hb not less than 11gm %</td>
</tr>
<tr>
<td>* RBC volume increase in 20%</td>
<td>* Packed cell volume ↓</td>
</tr>
<tr>
<td>* Number of:</td>
<td>↓</td>
</tr>
<tr>
<td>erythrocytes ↑</td>
<td>RBC volume</td>
</tr>
<tr>
<td>reticulocytes ↑</td>
<td>Plasma volume</td>
</tr>
<tr>
<td>* maternal erythropoietin levels ↑</td>
<td>→ Hematocrit ↓</td>
</tr>
<tr>
<td>2. Hemoglobin mass ↑ (gm)</td>
<td>2. Hemoglobin concentration decreases (g/dl)</td>
</tr>
<tr>
<td>- Oxygen carrying capacity of blood ↑</td>
<td>3. Plasma protein concentration decreases (g/dl)</td>
</tr>
<tr>
<td>3. Plasma protein increases (gm)</td>
<td>4. Albumin decreases</td>
</tr>
<tr>
<td>4. Globulin increases</td>
<td></td>
</tr>
<tr>
<td>Sex hormone binding globulin ↑</td>
<td></td>
</tr>
<tr>
<td>Thyroid binding globulin ↑</td>
<td></td>
</tr>
<tr>
<td>Normal A: G = 1.7 : 1</td>
<td></td>
</tr>
<tr>
<td>Pregnant A: G = 1:1</td>
<td></td>
</tr>
</tbody>
</table>

Normal
5. WBC increase
   TLC = ↑ (15,000)
   Postpartum → TLC = 25,000
   DLC → Neutrophils ↑
       T lymphocytes ↑
   B lymphocytes → Normal
   CD4 : CD8 → Normal

5. Platelet decreases but not less than normal.
   📷 A benign gestational thrombocytopenia
   monocytes ↓

Pregnancy is an immunocompromised state

6. Humoral immunity increases
   T - Helper cell a ↑

6. Cell mediated immunity decreases
   T - Helper cell 1 ↓

T-H1 shift to T-H2 is not seen in pregnant women with PIH

7. Interleukin - 4 increases
   Interleukin - 10 increases
   Interleukin - 13 increases

Diseases mediated by TH-2 flare during pregnancy
   Eg → SLE

7. Interleukin - 2 decreases
   TNF decreases
   Interferon α decreases

Diseases mediated by
   TH - 1 improve in pregnancy
   Eg → Hashimoto’s thyroiditis, multiple sclerosis, Rheumatoid arthritis

8. All inflammatory markers ↑
   ESR ↑
   CRP ↑
   All clotting factors ↑
   Pregnancy is a Hypercoagulable State

8. Factor 11 and 13 decreases
   Factor 13 → fibrin stabilising factor
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TPA</td>
</tr>
<tr>
<td></td>
<td>Plasminogen → Plasmin</td>
</tr>
<tr>
<td></td>
<td>TPA → Tissue plasminogen activator</td>
</tr>
<tr>
<td></td>
<td>During pregnancy → TPA inhibitor increases</td>
</tr>
<tr>
<td></td>
<td>Anticoagulants ↓</td>
</tr>
<tr>
<td></td>
<td>→ Protein C ↓</td>
</tr>
<tr>
<td></td>
<td>→ Protein S ↓</td>
</tr>
</tbody>
</table>

→ The size of spleen increases by 50%

Parameters which remains unchanged during pregnancy 00:23:28

1. B - lymphocyte
2. CD₄ : CD₈
3. Bleeding time and clotting time
4. Anti thrombin time

Changes in iron metabolism during pregnancy 00:24:24

→ All parameters of iron metabolism ↓
→ During pregnancy → Total Iron ↓, S. ferritin ↓
  Except: → Serum transferrin levels
  → Total Iron Binding Capacity (TIBC)

→ Total iron requirement during pregnancy = 1000mg
→ Fetal iron requirement during pregnancy = 300mg
→ Daily requirement of iron during pregnancy
  = 4-6 mg/day

To fulfill in diet → 40 - 60 mg of iron required
• Only 10% of diet iron is absorbed
• Iron supplementation is mandatory during pregnancy
Anaemia Mukti Bharat programe

- To prevent dimorphic anaemia.

\[ \text{Gives} \rightarrow 60 \text{ mg of elemental iron} + \]
\[ 500 \text{ mcg of folic acid} \]
\[ \downarrow \]

- 1 tab per day starting from 4th month of pregnancy
- Ideally continued throughout pregnancy but should be taken atleast for 180 days
- Should be taken for 180 days after delivery \[ \rightarrow \] To replenish iron stores

- Iron salt present \[ \rightarrow \] Ferrous sulphate
- Mala D and Mala N \[ \rightarrow \] Ferrous fumarate
MATERNAL ADAPTATIONS TO PREGNANCY: RESPIRATORY, GIT & RENAL CHANGES

Changes in GI system

- Gastric emptying time
  - Normal in pregnancy
  - ↑ during labour

- Progesterone - Smooth muscle relaxant
  - Relaxed lower Esophageal sphincter
  - ↑ Constipation
  - ↑ Haemorrhoids
  - ↑ Gastroesophageal reflux
  - ↑ Heartburn (pyrosis)

Vomiting during pregnancy / morning sickness
- Due to hCG (main)
- Also due to Estrogen, Progesterone, Thyroxine, Prolactin, Placental GH, Leptins
- Resolves after 14 - 16 weeks of pregnancy

- Hyperemesis gravidarum
  - Excessive vomiting + 1. Significant wt. loss
    2. Dehydration
    3. Alkalosis
    4. Hypokalemia
    5. Starvation Ketosis

Problems

1. Vit K deficiency
   - ↓ Prothrombin levels
   - Coagulopathy
   - Vit K embryopathy
   - Intracranial hemorrhage
   in fetus

2. Preterm labour
   2. Abruptio placenta
   3. Pre eclampsia

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a. Thiamine deficiency
   \[\downarrow\]
   Wernicke encephalopathy
   \[\text{Ataxia} \quad \text{Confusion} \quad \text{Ocular signs}\]

Risk Factors of Hyperemesis gravidarum

- ↑ hCG
- Molar pregnancy
- Twin pregnancy
- Placentomegaly
- Female fetus
- Hyperthyroidism
- ↑ Estrogen
- H. Pylori infection +/-
- Marijuana use

Management of vomiting in pregnancy

1. Mild / morning sickness
   1. Small frequent meals
      a. Vit B6
      2. Doxylamine
      3. Doxylamine + Pyridoxine
      4. Diphenhydramine

2. Moderate vomiting
   1. Promethazine
      a. Prochlorperazine
      3. Metoclopramide
      4. Ondansetron

3. Severe vomiting: IV fluids + Thiamine
   +
   IV metoclopramide
   Or
   IV Promethazine
   Or
   IV Ondansetron

4. Intractable vomiting: Enteral / Parenteral nutrition
Respiratory changes in pregnancy

- Uterus pushes diaphragm up by 4 cm
  - Residual volume ↓
  - Expiratory reserve volume ↓
- Transverse diameter of chest ↑ by 2 cm
  - Tidal volume ↑
  - Minute ventilation ↑
- Circumference of chest ↑ by 6 cm
- Subcostal angle ↑
  - Non pregnant → 68°
  - Pregnant → 103°

Functional residual capacity ↓ (ERV + RV)
Inspiratory capacity ↑ (IRV + TV)
Total lung capacity - Normal / Slightly ↓

Parameters which remain normal in pregnancy
1. IRV
2. TLC
3. Respiratory rate
4. Max. breathing capacity
5. Vital capacity

Renal changes in pregnancy

- Size of kidney ↑ by 1 cm
- Hydroureter (∇), mainly on right side
- Glycosuria is ∇ in pregnancy, proteinuria (> 300 mg/dl) → PIH
- Bladder
  1. Congestion of bladder
  2. Bladder pressure ↑ from 8 - 20 cm of H2O
  3. ↓ Bladder capacity
  4. Urethral pressure ↑
  5. Length of urethra ↑
- Total blood flow ↑ in pregnancy

↓

↑ Renal blood flow → ↑ GFR

↓

↑ Clearance of urea,
Uric acid, Creatinine

↓ S. urea
↓ S. uric acid
↓ S. Creatinine
MATERNAL ADAPTATIONS TO PREGNANCY: LIVER CHANGES

- Changes that occur in liver during pregnancy:
  1. Size of liver remains normal
  2. All liver enzymes decrease in pregnancy
     Except: Serum Alkaline phosphatase - increases
     ↓
     Synthesized by liver and placenta
     ↓
     Heat stable alkaline phosphatase

Cholestasis of pregnancy

- A/k/a recurring jaundice of pregnancy
  icterus gravidarum
- Seen in III trimester
- Basic pathology:
  Bile acids cleared incompletely
  ↓
  Accumulate in plasma
  ↓
  Most common symptom
  (first symptom)
  ↓
  Pruritis
  (palms and soles)
  ↓
  Bilirubin <4-5 g %
  Jaundice
  ↓
  SGOT, SGPT normal or increased slightly
  ↓
  Alkaline phosphatase is increased
  ↓
  Serum Bile acids
• Fetal complications
  - Preterm labour
  - Bile acid $\rightarrow \downarrow$ surfactant $\rightarrow$ RDS: Respiratory distress syndrome
  - ↑ Fetal distress $\rightarrow$ meconium aspiration syndrome
  - Still birth

• Obstetric management
  - Induction of labour: 38–39 weeks of pregnancy
  - Termination of pregnancy: 38–39 weeks of pregnancy

• Medical management
  1. Antihistamines for pruritus
  2. Cholestyramine not preferred - decreases absorption of vitamin K
  3. Drug of choice: ursodeoxycholic acid

• Recurrence rate of cholestasis = 60–70%
  OC pills are contraindicated in these patients

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

**Acute fatty liver of pregnancy**

• Most common cause of acute liver failure in pregnancy

• Associated with recessively inherited mitochondrial abnormalities of fatty acid oxidation

  Abnormality = mutation of LCHAD gene on Chromosome 2

  $\downarrow$ Long-chain L-3 hydroxacyl CoA dehydrogenase
  (responsible for oxidation of long chain fatty acids)
**Pathophysiology:**

- Accumulation of long chain fatty acids
  - Acute liver injury
  - Endothelial cell activation
    - Capillary endothelium becomes leaky
  - Haemoconcentration
  - 3rd space accumulation of fluid
    - Renal failure
      - Uteroplacental blood flow
      - Fetal distress
      - Fetal death
    - Ascites
    - Pulmonary edema

**Clinical features**

- Nausea and vomiting in III trimester
- 1 week later, Jaundice
- In 50% cases $\rightarrow$ ↑ BP, proteinuria and edema

**Investigations:**

- ↑ Bilirubin (≤10)
- ↑ SGOT, ↑ SEPT (≤1000)
- ↑ Alkaline phosphatase
- ↓ All clotting factors, Serum fibrinogen
- ↑ Bleeding time, ↑ Clotting time, ↑ Prothrombin time
- Hypoglycemia
- Hypoalbuminemia

- ↑ S. Urea
- ↑ S. Uric acid
- ↑ S. Creatinine

**Differential diagnosis - HELLP syndrome**
• Management:
  - Immediate termination of pregnancy
  - Mode of delivery → vaginal delivery if possible within 24 hours
    caesarean section otherwise

• Complications: seen in recovery phase
  - Diabetes insipidus
  - Pancreatitis
**MATERNAL ADAPTATIONS TO PREGNANCY: ENDOCRINE SYSTEM CHANGES**

**Endocrine changes in pregnancy**

<table>
<thead>
<tr>
<th>Parameters ↑ during pregnancy</th>
<th>Parameters ↓ during pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Adrenal Gland</td>
<td>1. Adrenal Gland</td>
</tr>
<tr>
<td>All Hormones ↑ (including</td>
<td>DHEA ↓ &amp; DHEA-Sulphate ↓</td>
</tr>
<tr>
<td>aldosterone) ↓</td>
<td>[Dehydroepiandrosterone]</td>
</tr>
<tr>
<td>( \therefore \text{Na}^+ ) &amp; ( \text{K}^+ ) retention</td>
<td></td>
</tr>
<tr>
<td>2. Pancreas</td>
<td>Except</td>
</tr>
<tr>
<td>Insulin secretion - ↑</td>
<td>- ( \uparrow ) Progesterone ( \rightarrow \downarrow ) LH</td>
</tr>
<tr>
<td>Insulin resistance - ↑</td>
<td>due to negative feedback on LH</td>
</tr>
<tr>
<td>3. Pituitary Glands</td>
<td>- ( \uparrow ) Estrogen ( \rightarrow \downarrow ) FSH</td>
</tr>
<tr>
<td>All Hormones released by</td>
<td>due to negative feedback on FSH by estrogen</td>
</tr>
<tr>
<td>Anterior Pituitary - ↑↑</td>
<td></td>
</tr>
</tbody>
</table>

**Changes in thyroid gland during pregnancy**

Thyroid Gland

i) Size of thyroid \( \rightarrow \uparrow \) [But, goitre is pathological]

ii) Level of hcg - ↑

   - \( \alpha \) - sub unit of hcg is similar to levels of TSH 
     \( \downarrow \)
     \( \uparrow T_3, T_4 \) levels
ii) Thyroid Binding globulin - ↑ ↓
  Levels of Free T₃, Free T₄ - Normal ↓
  \[ \therefore \text{Euthyroid during Pregnancy} \]

iv) TSH (Thyroid Stimulating Hormone) - Normal / Slight ↓

v) Iodine (I) requirement - ↑
  \[ \text{Reason} - \uparrow \text{iodine excretion} \]
  - RDA for Iodine during pregnancy = 250 mcg / day
  \( \text{(RDA - Recommended Dietary Allowance)} \)

vi) Most common cause of Hypothyroidism during pregnancy

\[ \begin{align*}
\text{Developing countries} & \quad \downarrow \\
\text{Developed countries} & \quad \downarrow \\
\text{I deficiency} & \quad \downarrow \\
\text{Hashimoto Thyroiditis} & \\
\end{align*} \]

vii) Dose of thyroxine should be ↑ by 30-50% during pregnancy

**CNS changes during pregnancy**

- Size of pituitary gland during pregnancy
  \[ \downarrow \]
  \[ \uparrow \text{by 150\%} \]

- Sheehan’s syndrome
  Post partum necrosis of anterior pituitary due to excessive blood loss [post partum hemorrhage]
  1. **Smoking** is not related to weight gain in pregnancy
  2. BMI ↓ weight gain

<table>
<thead>
<tr>
<th>BMI [kg/m²]</th>
<th>Weight Gain [kg]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under weight [&lt;18.5]</td>
<td>12.5 - 18</td>
</tr>
<tr>
<td>Normal weight [18.5 - 24.9]</td>
<td>11.5 - 14</td>
</tr>
<tr>
<td>Over weight [BMI ≥ 25]</td>
<td>7 - 11.5</td>
</tr>
<tr>
<td>Obese [≥ 30]</td>
<td>&lt; 7</td>
</tr>
</tbody>
</table>
3. All clotting factors ↑ during pregnancy, except factor II, IV
4. Fluid retention in pregnancy → Blood viscosity ↓
   - All iron parameters ↑
   - Blood oxygen carrying capacity ↑
5. Uric acid level - ↓
6. Most common cause of ↓ platelet → Benign gestational thrombocytopenia
7. Character of vagina
   - - Cederlein bacilli ↑ [lacto bacilli ↑]
     - ↓
   - ↑ conversion of glycogen → Lactic acid
     - ↓
   - Vaginal acidity - ↑
     - ↓
   - pH ↓
     - ↓
   - Pathogenic Organisms ↓
8. Globulin, Fibrinogen, Lymphocytes, Transferrin - ↑
9. Acute fatty liver in Pregnancy - Bilirubin < 10 mg / dl
   - Idiopathic cholestatic jaundice - Bilirubin < 5 mg / dl
10. Bile acid - Best diagnostic test for cholestasis of pregnancy
11. Cholestasis of pregnancy should be terminated - 35 - 39 weeks of gestation
   - Acute fatty liver in pregnancy - pregnancy should be terminated immediately
12. Colostrum contains all in excess except - K ↓ F ↓ C ↓
   - Potassium Fat Carbohydrate
13. Diaphragm is elevated by 4 cm during pregnancy.
   Transverse diameter ↑ by 2 cm
   Circumference of chest ↑ by 6 cm

14. Fibrin stabilizing factor ↓ during pregnancy

15. Intermediate cells — Predominate in vagina at the time of pregnancy

16. Inspiratory capacity ↑ during pregnancy

17. • Oxygen carrying capacity of blood ↑ during pregnancy
   • Arterio-venous oxygen gradient ↓ in pregnancy
   • Oxygen consumption ↑ during pregnancy

18. Theca lutein cyst:
   Seen in
   • Twin pregnancy
   • Hyperthyroidism
   • Diabetes

19. Hyperemesis gravidarum is not associated with — Post dated pregnancy
HEART DISEASES IN PREGNANCY

Changes in cardiovascular system in pregnancy

**Increases**

Cardiac output = Stroke volume x Heart rate

Cardiac output:
- Begins to increase after 5 weeks
- Maximum during pregnancy at 28-32 weeks (40%)
- Overall maximum increase in cardiac output:
  - Immediate post-partum (70%)
  - 2nd stage of labour (50%)
  - Late 1st stage of labour
  - 28-32 weeks of pregnancy

**Decreases**

- Main hormone = Progesterone
  - Smooth muscle relaxant
  - Peripheral vascular resistance
  - Pulmonary vascular resistance
  - BP
    - Systolic BP
    - Diastolic BP
    - Mean arterial pressure

- Maximum decrease in BP:
  - Diastolic BP
  - Maximum 2nd trimester
  - In supine position

- Supine Hypotension Syndrome:
  - In late 3rd trimester:
    - Lie supine
    - Gravid uterus presses on IVC
    - Venous return decreases
    - Cardiac output decreases
    - Hypotension
    - Blood flow decreases
    - Fetal Distress

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At 40 weeks:

In comparison to non-pregnant female:

- CO ↑

But compared to CO at 28-32 weeks:

- CO ↓

Pressure of gravid uterus on IVC pooling of blood in lower limbs

Femoral venous pressure:

- Edema
- Varicose veins
- Haemorrhoids

N → 8 mm of Hg
Pregnant female → 20 mm of Hg

→ O₂ Consumption → Arteriovenous O₂ gradient

Normal changes in clinical indications of heart in pregnancy

1. Heart Rate ↑ → Pulse Rate ↑
2. BP ↓
3. Heart rotates upwards and outwards during pregnancy (gravid uterus pushes diaphragm, diaphragm pushes heart)

- Polpitations
- Apex beat is heard in 4th intercostal space, 2.5 cm lateral to mid clavicular line
- ECG

Left axis deviation

4. Heart Sounds:

- S₁ → Loud + wide or prominent split
- S₂ → Normal
- S₃ → Easily heard
5. Murmurs:
   - MC murmur → Ejection Systolic murmur (< 3/6)
   - In 20% → Soft diastolic transient murmur
   - In 10% → Continuous murmur

   • On chest x-ray:
     - Appears → mild cardiomegaly
     - Unaffected in pregnancy:
       1. JVP / Central Venous Pressure
       2. Ejection fraction

Indicators of heart disease during pregnancy

   • Symptoms:
     - Clubbing
     - Cyanosis
     - Paroxysmal Nocturnal Dyspnea
     - Orthopnea
     - Pulmonary edema

   • ↑ JVP
   • $S_a$ → Loud + Prominent split
   • $S_s$
   • Ejection systolic murmur > 3/6
   • Diastolic murmur
   • Chest x-ray: marked cardiomegaly
   • Any arrhythmia

Heart diseases in pregnancy

   • MC Heart disease in pregnancy → Mitral Stenosis
   • MC Cause of heart disease in pregnancy in developing countries
     → RHD
   • MC Heart disease in pregnancy in developing countries
     → Mitral Stenosis
   • MC Cause of heart disease in pregnancy in developed countries
     → Congenital Heart Disease (HSD)
   • MC Cyanotic heart disease during pregnancy → TOF
   • MC Congenital valvular heart disease during pregnancy
     → Mitral Valve Prolapse
Clarks classification of heart diseases

- Class I
  → maternal mortality < 1%
  → All congenital heart diseases

- Class II
  → maternal mortality (15-25 %)

- Class III
  → maternal mortality (25-50%) → Diseases:
    1. Pulmonary Hypertension
    2. Eisenmenger syndrome
    3. Marfan syndrome with aortic involvement
    4. Coarctation of aorta

- Heart disease with highest risk of maternal mortality
  → Eisenmenger syndrome

- MC heart disease associated with maternal mortality
  → mitral stenosis

- Regurgitant lesions are tolerated better than stenotic lesions

- Heart diseases in which best prognosis → Congenital heart disease

- Heart diseases in which pregnancy is contraindicated:
  1. All 3 heart diseases belonging to Clark's class III
  2. Ejection fraction < 30%
  3. Severe mitral stenosis
  4. Severe aortic stenosis
  5. Any heart disease which belongs to NYHA class III or class IV

MTP in Heart disease patient in pregnancy:
- Normally in India → 30 weeks
- In Heart Disease → 12 weeks
- Best method → Suction Evacuation

Management of heart disease patient during labour

1. Rest in left lateral position or propped up position
2. Restrict:
   - Per vaginal examination
   - IV fluids @ 75 ml/hour
3. Best mode of delivery ➔ Vaginal delivery

- Best: Allow patient to go into spontaneous labour
- Induction of labour is safe
- Try vaginal delivery
- If 2nd stage of Labour > 30 minutes
- Still use vacuum or forceps
- Known as prophylactic use of forceps or vacuum

- In normal conditions:
  - 2nd stage is prolonged if:
    - Nulliparous ➔ > 2 hours
    - Multiparous ➔ > 1 hour

- Earlier: In Heart disease forceps was preferred
  - Now: Vacuum is preferred (Lithotomy position is not needed)

**Indications for cesarean section in heart diseases**

1. Any disease which involves Aorta:
   - Aortic aneurysm
   - Aortic stenosis
   - Marfan syndrome with aorta involvement
   - Coarctation of Aorta
2. Recent MI/CHF
3. Emergency valve replacement after delivery
4. If patient has been fully coagulated on warfarin at labour or within 2 weeks of labour

- Anesthesia: Epidural Anesthesia

Conditions in which General Anesthesia is used:
1. Intracardiac shunt
2. Hypertrophic Obstructive Cardiomyopathy (HOCM)
3. Pulmonary Hypertension
4. Severe aortic stenosis
5. Severe aortic regurgitation

Management of third stage of labour in heart disease

- Active management of third stage of labour
  → Inj. methyl ergometrine is contraindicated

- Give inj. Diuretics

- Antibody prophylaxis for infective endocarditis after vaginal delivery → Not needed

Contraception of choice in heart disease

Temporary method
  → Earlier: Barrier
  → Now: IUCD

Permanent method
  → Undergo vasectomy
  If partner refuses
  → Tubectomy

Tubectomy in heart diseases:

Time
  → Best: After 6 wks of delivery
  → 2nd Best: End of 1st week after delivery

Best
  → Minilaparotomy
  → Small incision under umbilicus
  → Done under Local Anesthesia

Laparoscopic sterilisation is Contraindicated
Anticoagulant of choice in pregnancy:

- **Warfarin**
  - Advantage: Strong anticoagulant
  - Disadvantage: Crosses placenta
  - Chondrodysplasia in fetus

- **Heparin**
  - Cannot cross placenta
  - Weak anticoagulant

→ Period of Gestation:
  - < 12 weeks
  - 12 - 36 weeks
  - 36 weeks
  - 6 hours after vaginal delivery and 24 hours after Caesarean

→ Warfarin is not contraindicated during breast feeding

→ If patient goes into labour on warfarin

  ↓

  Management: Cesarean Section

**Mitral stenosis**

- Best time to do surgery
  - 2nd trimester of pregnancy (14 - 18 weeks)

- Surgery of choice
  - Balloon valvuloplasty

- Surgery which is contraindicated in Pregnancy
  - Valve replacement

→ Anticoagulant of choice:
  - → Heparin
  - → Warfarin
  - → Heparin

  ↓

  Stopped 24 hours before labour

→ Restart:
  - Heparin or Warfarin
ANAEMIA IN PREGNANCY

Iron metabolism in pregnancy

- Total amount of iron needed in pregnancy = 1000 mg
  - Amount of iron needed by the fetus = 300 mg
  - Amount of iron lost during delivery = 250 mg

- Daily requirement of iron in pregnancy = 4-6 mg/day
  - Trimester wise,
    - I = 1-2 mg/day
    - II = 4-5 mg/day
    - III = 6 mg/day

- Only 10% of dietary Fe is absorbed
  
  To fulfill requirement of 4-6 mg/day
  
  Amount to be given in diet = 40-60 mg/day
  
  Impossible through diet alone
  
  Fe supplementation is mandatory

- Anemia mukt Bharat programme

  Ferrous sulphate (Fe) = 60 mg + Folic acid = 500 mcg

  Prophylactic dose of Fe To target dimorphic anemia.

  - Should be started from 4th month of pregnancy
  - Continued throughout pregnancy (ideally)
  - At least for 180 days during pregnancy and 180 days after delivery
    → To replenish iron stores
Anemia in pregnancy

Physiological anemia
Due to haemodilution
Hb is never <11g%

Pathological anemia
Due to other causes like Fe deficiency, Folic acid deficiency etc Hb <11g%

* Definitions:
  - Anemia in pregnancy: Hb < 11g%
  - Severe anemia in pregnancy: Hb < 7g%
  - Very severe anemia in pregnancy: Hb < 4g%

* Trimester-wise definition of anemia as per WHO and ACOG

<table>
<thead>
<tr>
<th>Trimester</th>
<th>Hb</th>
<th>Haematocrit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt; 11g%</td>
<td>&lt; 33%</td>
</tr>
<tr>
<td>11</td>
<td>&lt; 10.5g%</td>
<td>&lt; 31 - 32%</td>
</tr>
<tr>
<td>III</td>
<td>&lt; 10.5g - 11g%</td>
<td>&lt; 33%</td>
</tr>
<tr>
<td>Post-partum</td>
<td>&lt; 10g%</td>
<td>&lt; 30%</td>
</tr>
</tbody>
</table>

* One liners:
  - Most common cause of anemia in pregnancy
    Physiological anemia > Iron deficiency anemia
  - Most common cause of pathological anemia,
    Iron deficiency anemia (IDA)
  - First / most sensitive marker of IDA
    S. ferritin <30mg/ml
  - Most common cause of megaloblastic anemia.
    Folic acid deficiency anemia.
  - Dose of Folic acid in megaloblastic anemia
    1mg/day
  - Most common anemia after bariatric surgery
    Vit B12 deficiency anemia.
  - Most common indirect cause of maternal mortality anemia
  - Most common type of anemia of pregnancy in developing countries: Fe + Folic acid deficiency anemia (Dimorphic anemia)
- Screening of Anemia in pregnancy
  - Should be done in all females
  - Done at 1st antenatal visit, repeated between 24-28 weeks
  - Screening is done by doing complete blood counts

**Management of IDA**

1. Oral Iron
   - Therapeutic dose = 2-3 tablets of Fe/day
     it is given till blood parameters become normal
   - 1st blood parameters to improve = Reticulocyte count
     it starts increasing within 7 days and maximum is reached in 10 days
   - HB increases after 3 weeks. Rate of increase of
     HB = 0.7 - 1g/l/week
   - Maintenance dose = 1 tab/day
     it is given throughout pregnancy + 180 days after delivery

- Problems of oral Fe
  - Poor compliance
  - GI side effects
  - Cannot be used in inflammatory bowel disease like ulcerative
    colitis and Crohn’s disease
  - Cannot be used in patients after bariatric surgery

II - Parenteral Iron
   - Examples:
     - Low molecular weight Dextran
     - Iron sucrose
     - Ferric carboxymaltose

- Rate of increase of HB after parenteral iron is same as that of oral iron

- Advantages:
  - Not patient dependent
  - Large amounts (1000mg IV) can be given in a single infusion
  - No GI side effects
  - Can be used in patients of IBD, malabsorption, following
    bariatric surgery
- **Calculation of dose of parenteral iron**
  - **Assume:**
    - Blood volume = 65ml/kg
    - Hb to be corrected till 14g%
    - Each g of Hb = 3.3mg of Fe
  - Dose of Fe = \( \text{(Body weight x (14- patient Hb) x 2.145)} \) (mg) + \( \text{(Irres to replenish stores)} \)
  - Volume of drug to be given = \( \frac{\text{Dose of iron (mg)}}{\text{Concentration of Fe/mL in the drug}} \)
  - Example:
    - Suppose, 60 kg mother with Hb = 8
    - No need for iron stores
    - Using Fe sucrose = 20 mg/ml of Fe
    - Dose of Fe = \( 60 \times (14-8) \times 2.145 \)  
    = 772 mg of iron
    - Volume of drug = \( \frac{772}{20} \) = 38.6 mL IV

**III - Blood Transfusion**

- **Indications of blood transfusion in pregnancy**
  1. Pregnancy < 34 wks + Hb <10g%
  2. Pregnancy >34 wks + Hb <10g%
  3. At time of labour + Hb <10g%
  4. Acute haemorrhage causing Hb <6g%
  5. Any time in patient with heart failure with low Hb
DIABETES IN PREGNANCY

Carbohydrate metabolism in pregnancy

- Insulin resistance in pregnancy
  
  mainly due to HPL
  Other hormones 
  - Estrogen
  - Progesterone
  - Cortisol

  24-48 wks of pregnancy

- Fetus is entirely dependent on mother for glucose requirement
  
  GLUT 1 & GLUT 3

  Facilitated diffusion → glucose reaches the fetus

- In pregnancy - Fasting hypoglycemia & Post prandial hyperglycemia.
- Glycosuria - ○ in pregnancy
- Maternal insulin cannot cross placenta.
- Fetus starts producing insulin @ 12 wks of pregnancy
- Maternal hyperglycemia → Fetal hyperglycemia.

  Hypertrophy of β cell of fetal pancreas

  ↑ insulin in fetus

  ↑ growth of fetus ↓ Surfactant Macrosomia production Neonatal hypoglycemia

  ↓ Chances of RDS
Diabetes in pregnancy

Diabetic female has conceived
↓
Overt diabetes
↓
Blood sugar levels are raised from day 1

Normoglycemic female during pregnancy, develops
insulin resistance
↓
Becomes diabetic
↓
Gestational diabetes
↓
Blood sugar levels are raised after 24-28 wks of pregnancy

In diabetes → ↑ blood sugar from day 1
↓
↑ free radicals
↓
Increased chances of congenital malformation

Free radicals are formed after 24-28 wks
↓
By this time organogenesis is complete
↓
No congenital malformation

Overt diabetes

FBS ≥ 136mg/dl
RBS = ≥ 200mg/dl
HbA1C ≥ 6.5%

Tests to predict the chances of congenital malformation in babies of overt diabetes mother
↓
HbA1C

<6.5% 6.5-9.5 ≥9.5
↓ ↓ ↓
No chances 5% 20%
- Test to detect congenital malformation in babies of diabetic mother – Tiffa – level a ultrasound scan @ 16-20 wks of pregnancy

- Tiffa should be done in overt & gestational diabetes mellitus

- Methods to prevent congenital malformations in babies of overt diabetic mothers
  1. Strict control over glucose
  2. Folic acid supplementation (400 mg/day)

- DOC – Insulin
  All Oral hypoglycemic drug are contraindicated

1. MC system involved in congenital malformation – CVS > CNS
2. MC congenital anomaly seen – VSD > NTD
3. Most specific anomaly – Caudal regression syndrome or sacral agenesis
4. Most specific cardiovascular anomaly – TGA
5. MC cardiac anomaly seen in babies of diabetic mother – VSD
6. MC cardiovascular finding seen in babies of diabetic mother – HOCM
7. Least common cardiac anomaly seen in babies of diabetic mother – TDF

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

Gestational diabetes

- Diagnosis of gestational diabetes

  2 step approach by ACOG

  1 step approach by WHO (DIPS)

2 step approach

  1st step: Screening test: Glucose challenge test (GCT) time
  24-28 wks

  No fasting is required
**Method:** 50 gms glucose to patient

1hr

Check blood sugar levels

- If <140 mg/dl
  - Not diabetic
  - Do next step: i.e., glucose tolerance test

- If 140-180 mg/dl
  - Do next step: i.e., glucose tolerance test

- If >180 mg/dl
  - Diabetes confirmed

**Note:**

1. ACOG recommends universal screening in all pregnant females irrespective of risk factors.

   In low risk females, OGT to be done only once between 24-28 wks.

   In high risk females, OGT to be done @ 1st antenatal visit & repeated at 24-28 wks.

2. High risk females
   - Obese
   - Age > 35 yrs
   - Previous h/o GDM
   - Family h/o diabetes
   - Previous h/o congenital malformation
   - Previous h/o still birth
   - Previous h/o IUGR
   - ≥ 3 abortions
   - Previous h/o macrosomia.
   - Polyhydramnios

3. And step: perform a 3hr 100 gm OGGT (glucose tolerance test)

   Preparation: Overnight fasting (8-10 hrs)

   1st sample = FBS sample

   Give 100 gms glucose
↓
2nd sample 1 hr postprandial
3rd sample 2 hr postprandial
4th sample 3 hr postprandial
Total = 4 samples
If any 2/ > 2 values are abnormal → diabetes is confirmed

<table>
<thead>
<tr>
<th>Carpenter and coustau criteria</th>
<th>National diabetes criteria (India)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting 95 mg/dl</td>
<td>105 mg/dl</td>
</tr>
<tr>
<td>1hr 180 mg/dl</td>
<td>190 mg/dl</td>
</tr>
<tr>
<td>2hr 155 mg/dl</td>
<td>165 mg/dl</td>
</tr>
<tr>
<td>3hr 140 mg/dl</td>
<td>145 mg/dl</td>
</tr>
</tbody>
</table>

One step approach
- Only screening test
- Do a 2hr 75gm OGTT

Directly @ 24-28wks → ask pt to do overnight fasting (8-10hrs)
↓
1st sample = FBS sample
↓
Do a hr 75 gm OGTT
↓
Give 75 gm oral glucose
↓
After 1hr → 2nd sample
After 2hr → 3rd sample

Total 3 samples
If 1 or > 1 value is abnormal, → diabetic

Criteria, for 75 gms GITT
- Fasting 93
- 1 hr 180
- 2 hr 153

Note: Unlike ACOG, WHO recommends tests only in high risk females

DIPS1 criteria, followed in India.
- No fasting needed
- Whenever female comes for antenatal visit
  ↓
  Irrespective of previous meals
  ↓
  Give 75 gm of glucose
  ↓
  After 2 hrs, check blood sugars levels

  2130 2140 2300
  ↓  ↓  ↓
  Glucose GDM Overt diabetes

- This test has to be done in all pregnant females

Management of diabetes in pregnancy

1st step = Diet modification + exercise
  ↓
  2 wks
  ↓
  Aim for metabolic goals
  
  Fasting = 70-95
  1 hr postprandial = < 140
  2 hr postprandial = < 120
  Hb A1C < 6.5

Not attained by 2 wks → Insulin

- DOC for treating diabetes in pregnancy - Insulin
- During pregnancy insulin dose is increased
- During labour (patient is on liquid diet / Nil oral) - Dose of insulin ↓
- Oral hypoglycemic - contraindicated during pregnancy because
  - 1. They can cross placenta → Fetal hypoglycemia
  - 2. Pregnancy - diabetogenic state

  Insulin - strong drug
  OHA - weak drugs
Diabetes in pregnancy: fetal surveillance

- Begin by 32 - 34 wks
- If growth restriction is seen → Start monitoring from 28 wks
- Overt diabetes + GDM patients on insulin
  ↓
  Admit at 34wks
- Methods of surveillance
  1. Daily fetal kick count
  2. Biweekly NST
  3. Weekly biophysical score

Time of delivery
- GDM patient, controlled control on diet: ≥ 39wks to 40wks + 6 days
- GDM control by insulin → ≥ 39 wks to 39wks + 6 days
- Overt diabetes: 37wks to 37 wks + 6 days
  mode of delivery: Vaginal delivery
  If weight of fetus is ≥ 4.5kg in diabetic female
  ↓
  Cesarean section

Complications of diabetes in pregnancy

Maternal complications

↑ Infections  ->  Big placenta  ->  Hyperglycemia in fetus  ->  ↑ Chances of DM in future
↓ Asymptomatic Placenta previa  →  PIH  →  Polyuria  ↓  Polyhydramnios
↓  • Preterm labour  →  PROM

- PROM
- Preterm labour
- Malpresentation
- Abruptio placenta
- Cord prolapse
- PPH
Fetal complications

1) Fetal Hyperglycemia
   ↓
   ↑ Insulin
   ↓

2) Macrosomia / ↑ growth of fetus

   ↑ in wt of fetus ≥ 4.5 kgs
   In India ≥ 4 kgs

Risk factors for macrosomia:
- Male fetus
- Postdated pregnancy
- Diabetes in mother

USG parameter to detect macrosomia = Abdominal circumference
   ↓
   ≥ 35cms → macrosomia

- Organ least affected in macrosomia = Brain
- Management: caesarean section is done

   wt of fetus ≥ 4.5 kg in diabetic female
   wt of fetus ≥ 5 kg in non diabetic females

- During vaginal macrosomia can lead to shoulder dystocia.

Fetal complication: shoulder dystocia

- Delay in delivery of shoulder > 1 min after delivery of head
- Sudden pulling back of head towards the perineum known as turtle sign

Risk factors for shoulder dystocia
(Mnemonic = DOPAP)
- D - Maternal diabetes
- O - Maternal obesity
- P - Fetal obesity
- A - Post dated pregnancy
- P - Anencephaly
   (pseudo shoulder dystocia)
Management: Shoulder dystocia drill

( Mnemonic: HELPERR )

H - Call for help
E - Give episiotomy
L - Flexion of legs against abdomen (McRoberts maneuver) and abduction of hips
- MC nerve injured during this maneuver - Lateral femoral cutaneous nerve of thigh

McRoberts maneuver

McRoberts maneuver and suprapubic pressure

- First & best maneuver in management of shoulder dystocia – McRoberts maneuver
P - Give suprapubic pressure
- Fundal pressure contraindicated
E - Enter vagina: Hold and rotate shoulders
- Woods corkscrew maneuver
- Rubin maneuver
R - Remove the posterior arm of baby
- Jacquemier maneuver / Barnum maneuver
R - Rotate patient to all 4 limbs
- Gaskin or all 4 maneuver
- Wood's corkscrew maneuver
- Last maneuver: Zavanelli maneuver
  - Head of the fetus pushed back into the uterus followed by caesarean section
- Destructive procedures which are not done but can be done are
  1. Cleidotomy - clavicle
  2. Symphysiotomy - dividing pubic symphysis in mother
- m.C. maternal complication of shoulder dystocia - PPH
- m.C. fetal complication - Brachial plexus injury
- m.C. bone injured - Clavicle

Other fetal complications:
  1. ↑ chances of death of the fetus
     - Occurs at <30 wks/<500 gms in ut - Abortion ↑
     - Occurs at >30 wks/≥500 gms in ut - IUD of fetus ↑
     - Occurs during delivery - Still birth ↑
  2. Rarely IUGR may be seen
     1. Diabetes + PIH
     2. Overt diabetes with vasculopathy

Intrauterine death/ IUD

- Signs of IUD on X-ray
  1st sign - Robert sign - presence of gas in great vessels in fetus
  - Becomes +ve within 12-24hrs of fetal death

  2nd sign - Spalding sign
  - Overlapping of cranial bones of fetus
  - Becomes positive within a week of fetal death

  3rd sign - Ball sign
  - Hyperflexion / Hyper extension of fetal spine
Neonatal complications in diabetes

- Prematurity
  - RDS
- Hypoglycaemia (≤40 mg/dL)
- Hyperviscosity Syndrome
  - K^+↓
  - Ca^{++}↓
  - Mg^{++}↓
  - Polycythemia
  + Hyperbilirubinemia

- Anaemia & mental retardation - not seen in babies of diabetic mother

- Best test for assessing lung maturity in fetus of diabetic mother - Phosphatidyl glycerol
  - If present in amniotic fluid
    - Lungs mature
  - If absent in amniotic fluid
    - Lungs not mature

- Tocolytic C/I in diabetic mother
  - β-agonists - Ritodrine
    - Isosuxprine
    - Terbutaline
    - Salbutamol

- Tocolytic of choice in diabetic mother - Nifedipine
PREGNANCY INDUCED HYPERTENSION – 1

Hypertension in pregnancy

- BP more than or equal to 140/90 mmHg on 2 occasions 4 hours apart
- Note in pregnancy – Diastole BP determined by disappearance of sound (korotkoff sound V)
- It should be recorded in sitting position

Two situations

- Hypertensive female has conceived
  - Chronic hypertension in Pregnancy
  - Normotensive female who has conceived & during pregnancy due to placental pathology at 20 weeks → BP increases

<table>
<thead>
<tr>
<th>Chronic hypertension in pregnancy</th>
<th>PIH</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Past history of hypertension present</td>
<td></td>
</tr>
<tr>
<td>* Increase in BP is seen even before 20 weeks of preanancu</td>
<td></td>
</tr>
<tr>
<td>* BP does not come back to normal with in 12 weeks of pregnancy</td>
<td></td>
</tr>
<tr>
<td>* Not present</td>
<td></td>
</tr>
<tr>
<td>* Increase in BP is seen only after 20 weeks of pregnancy</td>
<td></td>
</tr>
<tr>
<td>* BP comes back to normal with in 12 weeks of delivery</td>
<td></td>
</tr>
</tbody>
</table>

PIH

Gestational Hypertension ∆ Pre-eclampsia

- ↑ in BP after 20 weeks of pregnancy
- BP will come back to normal within 12 weeks of delivery
- Not present
- Either proteinuria or
  - Signs of end organ damage

<table>
<thead>
<tr>
<th>Proteinuria</th>
<th>End organ damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Excretion of proteins → more than or equal to 300 mg in 24 hours</td>
<td>* Serum creatinine more than 1.1</td>
</tr>
<tr>
<td>or</td>
<td>* Platelet count less than 1 lakh</td>
</tr>
<tr>
<td>* more than or equal to 30 mg/dl (0.3g/l)</td>
<td>* Liver enzymes are raised to more than 2 times their normal value</td>
</tr>
<tr>
<td>or</td>
<td>Note → ALP is normally raised in pregnancy, so it has no significance</td>
</tr>
<tr>
<td>* urine protein: creatinine ratio more than 0.3</td>
<td>* Pulmonary edema</td>
</tr>
<tr>
<td>or</td>
<td>* Cerebral/visual symptoms.</td>
</tr>
<tr>
<td>* Dipstick method = 1 + = 0.3 g/L</td>
<td></td>
</tr>
<tr>
<td>* Traces is not taken as proteinuria. = (0.15 to 0.3g/l)</td>
<td></td>
</tr>
</tbody>
</table>

**Pre-eclampsia**

- **mild preeclampsia**
  - BP = more than or equal to 140/90 but < 160/110
  - Signs of end organ damage are not there

- **Severe preeclampsia**
  - BP = more than or equal to 160/110
  - Signs of end organ damage present

Chronic hypertension with superimposed preeclampsia
- In a pregnant chronic hypertensive female if at 20 weeks:
  - BP becomes uncontrollable
    - Or
  - Proteinuria
  - Signs of end organ damage present
Pathology

Normally → when spiral arteries open in the intervillous space → Extravillous
Cytotrophoblast → It will replace

Trophoblastic invasion
Lining of the maternal artery and convert them
→ From high resistance vessels
→ Low resistance vessels

• If trophoblastic invasion is incomplete
→ Resistance in maternal artery remains high
→ PIH

<table>
<thead>
<tr>
<th>Normal pregnancy</th>
<th>PIH</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Spiral arteries → Angiogenesis → Diameter ↑ ↓ Pressure ↓ Bought about by: - VEGF - Placental growth factor</td>
<td>Absent</td>
</tr>
<tr>
<td></td>
<td>- VEGF ↓</td>
</tr>
<tr>
<td></td>
<td>- Placental growth factor ↓</td>
</tr>
<tr>
<td></td>
<td>1) Soluble tyrosine kinase like receptor = sFlt 1 ↑</td>
</tr>
<tr>
<td></td>
<td>2) endoglin ↑</td>
</tr>
</tbody>
</table>
- Vasodilators are NO ↑
- Vasoconstrictors →
  Thromboxane A₃ ↓
  Prostacyclin ↓

<table>
<thead>
<tr>
<th>NO ↓</th>
<th>increased</th>
</tr>
</thead>
</table>

- Due to vasoconstriction and increased resistance in spiral arteries → volume of blood coming to IVS ↓ → placental ischaemia → Pale
  → Small

- Placental ischaemia ↓
  Inflammatory mediators released ↓
  acts on capillary endothelium

  ↓

  Capillaries leak

  ↓

  Collection of fluid in third space
  ↓
  Edema

  ↓

  Hemoconcentration → Platelet dysfunction
  ↓
  Thrombosis in blood vessels
  ↓
  Multiple organ dysfunction
In severe preeclampsia, $\rho \propto \frac{1}{v}$

- Blood flow to brain
  - Cerebral hypoxia
  - Convulsions
  - Eclampsia

- Blood flow to kidney
  - RBF ↓
  - GFR ↓
  - S. urea
  - S. uric acid
  - S. Creatinine

- Blood flow to fetus
  - IUGR
  - RBF in fetus

- Oliguria in fetus
  - Oligohydranmios

- Maternal oliguria

- Most common organ involved in PIH: Kidney
  - On HPE
    - Glomeruloendotheliosis

- Another characteristic feature of PIH is:
  - Normally in pregnancy TH₁ response is suppressed and switch to TH₂
  - This does not happen in PIH

Important concepts:

- PIH → Chorionic villi
  - Size of placenta ↑
  - Diabetes

- If a female is exposed to chorionic villi for 1st Time → Prim
  - Risk factor for PIH

- Excessive chorionic villi → Twins/molar
  - Pregnancy → Risk factor for PIH

- Fetus is not prerequisite for PIH

- It is not necessary that these chorionic villi should be intrauterine
  → PI can develop in ectopic pregnancy

Obstetrics & Gynaecology • v2.0 • Marrow 4.0 • 2020
- Eclampsia is a complication of severe pre-eclampsia.
- Eclampsia → Patient has → Generalized Tonic clonic Seizures
  - Occur during Pregnancy
  - Antepartum Eclampsia
  - → most common and worst prognosis
  - Occur during Labor (with in 48 hours of delivery)
  - Intrapartum Eclampsia
  - Occur after delivery (post partum)
  - Post partum Eclampsia

Signs and symptoms of impending eclampsia in patients of severe preeclampsia:
1. Oliguria
2. Epigastric pain (due to stretching of liver capsule)
3. Visual symptoms
   - most common - scotoma
   - Reversible blindness
   - Blurring of vision / diplopia.
   - Visual symptoms are due to HTN Retinopathy
     - Classification → Keith Wagner Baker classification
4. Headache

Risk factors for PIH

- Maternal exposure to placental tissue for first time
  - Primi Gravida
  - New paternity
- Placental tissue
- Syndrome
  - APLA Syndrome
  - Metabolic
  - Gestational (Gestational diabetes)
  - Multiple pregnancy
  - Rh negative Pregnancy
Protective: Smoking

- Patients with placenta previa do not develop pre-eclampsia.
- But there is a positive association between abruption and pre-eclampsia.
- Age of patient more than 35 years is also a risk factor

Tests which can predict PIH:

- Most commonly used test
  - Uterine artery doppler
- Outdated test
  - Giants roll over test
- Recent predictors
  - VEGF = ↓  
  - PLOTO = ↓  
  - SFIT  
  - Endoglin  
  - NO = ↓  
  - Tromboxane A2 = ↑  
  - Urinary calcium excretion

Findings in PIH not predictors:
1. Hemoconcentration
2. Oliguria
3. ↑ uric acid levels

Drug to prevent PIH

- Best → Aspirin (↓ Thromboxane A2) → Dose: 50 - 150 mg OD 12-28 weeks of pregnancy
- Heparin and aspirin
- Calcium supplementation only if female has ↓ calcium
- Regular exercise

No role of:
- Bed rest
- Diet salt restriction
- Fish oil
- Antioxidants
- Heparin alone
Management of PIH

- Definitive management → Termination of pregnancy (TOP)
  - TOP → mild preeclampsia → 37 weeks
    - Severe preeclampsia → 34 weeks
      - Eclampsia
      - HELLP Syndrome
        - Immediate termination
        - Irrespective of Gestational age
  - mode of delivery → Vaginal delivery
    - Cesarian not preferred
    - If cesarian done → Epidural is preferred than spinal
      → GA not given

Management of mild PE:

- Role of antihypertensives +/-
  - Indication for starting antihypertensives:
    1. Severe ↑ BP (≥ 160/110)
    2. If BP is more than or equal to 150/100 then it should be persistently increased

Definitive management: Termination of pregnancy at 37 weeks

Management of severe PE:

- Risks
  - Impending Eclampsia
    - Risk of Hemorrhage
      - To prevent convulsion/
        - To treat in a Hypertensive pregnant female
          - Give antihypertensive
            - DOC
              - DOC → mgSO4
                - For PIH/PE
                  - 1st choice
                    - labetalol
                      - Labetalol
                        - α - methyl dopa
                          - iv labetalol
                          - iv hydralazine
                - Chronic HTN
                  - in pregnancy
                - Hypertensive crisis

Definitive management: Termination of pregnancy at 34 weeks

Anti hypertensives in pregnancy:
* Safest anti-hypertensive: Alpha methylidopa

<table>
<thead>
<tr>
<th>Safe</th>
<th>Contraindicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Labetalol (α + β blockers)</td>
<td>1. ACE inhibitors</td>
</tr>
<tr>
<td>2. α methylidopa</td>
<td>2. Angiotension receptor blocker - Losartan</td>
</tr>
<tr>
<td>3. Hydralazine</td>
<td>3. β blockers</td>
</tr>
<tr>
<td>5. Nitroglycerine</td>
<td>5. Diazoxide</td>
</tr>
<tr>
<td>6. Sodium Nitroprusside (resistant cases)</td>
<td></td>
</tr>
</tbody>
</table>

Management of hypertensive crisis
* DOC → Labetalol iv → Dose → Initial → 20mg
  Maximum total dose → 200mg ↓ 10mins
* iv. Labetalol is contraindicated in asthmatic patients.
  40mg ↓ 10min
  60mg ↓ 10mins
  80mg
* 2nd → DOC iv Hydralazine
* 3rd → Sustained release nifedipine
* 4th → iv NTG
* Last Resort → Sodium Nitroprusside DOC for Refractory HTN
**PIH-2**

### Management of eclampsia

- **1st step in management** → Airway management
  - DOC to Treat and Prevent convulsions → mgSO₄
  - Simultaneously, Anti hypertensive → Labetalol (IV)
    - max.dose → 220 mg
    - [Reason-Hypertensive crisis; BP > 160/110 mmHg]
  - Definitive management → Termination of pregnancy (TOP) immediately irrespective of gestational age

### Magnesium sulphate

- **DOC** to prevent and treat convulsions in a hypertensive female
- **Not** an antiepileptic drug
  - Reason – MgSO₄ can prevent only convulsions due to vasoconstriction because of PIH
    - [pregnancy induced Hypertension]
- **Mechanism of action**
  - Centrally acting
    - acts on NMDA receptors in brain
      - Cerebral vasodilation
      - ↓ cerebral hypoxia.
    - Blocks Ca²⁺ channel
      - ∴ CCB’s (Ca²⁺ channel blockers) should not be combined with MgSO₄
        - Risk of causing respiratory arrest
- **Prophylactic use**
  - Impending eclampsia.
  - Severe pre-eclampsia[PE]
  - HELLP syndrome
Pritchard regimen of MgSO₄

Loading dose

\[ 4 \text{ gm} + 10 \text{ gm} \]
\[
\text{↓} \quad \text{↓}
\]

80% solution 50% of solution [1 mL=5 gm in each buttock]

[slow IV]

* maintenance dose = 5 gm

\[
\text{↓} \quad \text{↓}
\]

given every 4th 50% solution [1 mL = given on alternate
hourly till 24 hrs buttocks each time to prevent abscess]
after delivery
(or)

24 hrs after
last convulsion

Dose calculation

* Vial available → 10 ml vial
  - Contains 50% MgSO₄
  - ∴ 10 ml vial → Has 5 gm of MgSO₄
  - ∴ 1 ml = 0.5 gm

* For IV use → 20% solution required

20 ml syringe

\[
\text{↓}
\]

8 ml of MgSO₄ (~ 4 gms)

+ Add 12 ml of normal saline (NS)

∴ In 20 ml, there is 4 gm of
MgSO₄ = 20%

Therapeutic range of MgSO₄

* Narrow therapeutic range
  → 4-7 meq/L
  (or)
  2-3.5 mmol/L
  (or)
  4.8-8.4 mg/dL
4 Parameters checked before loading dose →
   1. Knee jerk / Patellar reflex - Present
   2. Urine output ≥ 50 ml/hr
   3. Respiratory rate ≥ 12 breaths/min
   4. $\text{SpO}_2$ ≥ 96%

**MgSO$_4$ toxicity and SIBAI regime**

A - MgSO$_4$ Toxicity

1. Signs of MgSO$_4$ toxicity
   a. Loss of knee jerk / Patellar reflex: when MgSO$_4$ ≥ 10 meq/L

   **1st** sign
   b. Slurring of speech & Diaphoresis
   c. Respiratory depression at MgSO$_4$ ≥ 12 meq/L
   d. Respiratory arrest at MgSO$_4$ ≥ 15 meq/L
   e. Cardiac arrest: MgSO$_4$ = 25-30 meq/L

Note:
Oliguria is **not** a sign of MgSO$_4$ toxicity, but is a parameter to be checked before loading dose
[Reason: MgSO$_4$ excreted through kidney]

a. Antidote for MgSO$_4$ toxicity
   - Calcium gluconate [10 ml of 10% of Ca$^{2+}$ gluconate]
   - Alternative: CaCl$_2$

b. Absolute contraindication of MgSO$_4$
   a. Myasthenia gravis
   b. Renal failure

B. SIBAI regime

* Route - i.v only
* Loading dose ⇒ 6 gm i.v X 20 mins
* Maintenance dose ⇒ 2 gm i.v
* If convulsion recurs ⇒ 2-4 gm in 5 mins

Status eclampticus
* Uncontrolled convulsions despite MgSO$_4$ administration
* **DOC** - Thiopentone sodium
HELLP syndrome

- A complication which is seen in 3rd trimester

\[ \downarrow \]

Patient complains of abdominal pain [epigastric / right upper quadrant]

- 85% cases have high B.P., but 15% cases may have normal B.P
- Recurrent rate = 4-7%
- HELLP -

1. Hemolysis
   - LDH ≥ 600IU
   - Serum bilirubin ≥ 1.2
   - ↓ Haptoglobin level
   - On peripheral blood smear: Helmet cells, Tear drop cells, Schistocytes

2. Elevated liver enzymes
   - SGOT & SGPT ≥ 70 IU

3. LP: Low platelet count
   - Platelet < 1 lakh

- Tennessee criteria.
  - To diagnose HELLP syndrome
  - Criteria: LDH > 600 IU
    - SGOT, SGPT ≥ 70 IU
    - Platelet < 1 lakh

- Differential Diagnosis
  - AFLP [Acute Fatty Liver of Pregnancy]
    - Conditions seen in AFLP but not in HELLP
      - i. Hypoglycemia
      - ii. Hepatorenal syndrome
      - iii. Features of DIC
      - iv. Development of a° complications like pancreatitis

- Management of HELLP syndrome
  - 1. Prophylactic MgSO₄
  - 2. Antihypertensives
  - 3. Definitive management → Immediate Termination of pregnancy
Umbilical artery doppler study

a. Normal

- Forward flow
- As the period of gestation ↑, resistance of blood vessels ↓
  ∴ Diastolic flow increases
  - S/D ratio decreases (< 3)

b. PIH

- S/D ratio increases (> 5)
  during diastole → Less blood enters the vessel due to
  ↑ vascular resistance

c. Absent end diastolic flow

- Absent diastolic flow
  ↓
  Indication of TOP at ≥ 34 weeks
If period of gestation is < 34 weeks

- Give corticosteroids
- Continuous fetal monitoring [NST and BPP]

[Reason: absent end diastolic flow within a week Reversal of flow]

**d. Reversed Diastolic Flow**

![Diagram showing reversed diastolic flow with annotations: Forward systolic flow, Reversed diastolic flow.]

Indication for **Immediate** termination of pregnancy [TOP]

1. Indications for immediate TOP irrespective of gestational age in PIH
   a. Eclampsia.
   b. HELLP syndrome
   c. Fetal distress / Abruptio placenta.
   d. Uncontrolled BP
   e. ↑ serum creatinine
   f. Reversal of diastolic flow (Umbilical Artery Doppler)

2. Indications for TOP at 34 weeks in PIH
   a. Severe pre-eclampsia.
   b. Absent end diastolic flow.
IUGR - Intrauterine Growth Restriction:

- **Manifestation of disease:**
  - Due to: maternal, placental, fetal problems
- **Weight of fetus:** < 10 percentile of weight appropriate for that gestational age < 2 SD below
- **First change:** Abdominal circumference of fetus ↓

![Diagram showing macrosomia and IUGR]

**Normal growth rate of fetus**

- **Upto 14 - 15 weeks:** 5 g/day
- **Upto 20 weeks:** 10 g/day
- **From 32 - 34 weeks:** 30-35 g/day
- **Factors influencing growth of fetus:**
  1. Growth of the parents
  2. Hormone responsible for growth of fetus in intrauterine life
     - Insulin like growth factor
  3. Availability of substrates (glucose etc)

![Diagram showing maternal and fetal blood flow]

- **Maternal PIH**
  - $P \uparrow (P \propto \frac{1}{V})$
  - $V \downarrow$ (volume of blood to intervillous)
  - Substrates ↓
  - **IUGR**
* Smoking:
  - Does not affect maternal weight gain
  - Vasoconstriction $\rightarrow$ decrease volume $\rightarrow$ decrease substrates $\rightarrow$ IUGR
  - Active/passive smoking $\rightarrow$ Leads to IUGR

IUGR: classification

```
IUGR (↓ growth) __________________________________________________________

Early in pregnancy ↓ Late in pregnancy ↓
  ↓ No. of cells ↓            ↓ 32 - 34 weeks
  ↓ ANA Type 1 IUGR           ↓ Size of cells ↓
  ↓ Bad Prognosis            ↓ No. of cells: normal
  ↓ Type 1 IUGR              ↓ Good prognosis

Type 1 IUGR: All USG parameters are affected
  Abdominal circumference (AC) ↓
  Head circumference (HC) ↓
  Biparietal Diameter (BPD) ↓
  Femur length (FL) ↓
  Weight ↓

Symmetrical IUGR

Type 2 IUGR: Later in pregnancy
  Blood is collected from peripheral organs and sent to brain
  1st organ: Abdomen affected
    AC ↓
    Weight ↓
    But: BPD = Normal
    HC = Normal
    FL = Normal
    Crown Rump length = Normal

Asymmetrical IUGR

Ponderal Index (PI):

  $P_I = \frac{\text{Estimated Fetal weight}}{(\text{Femur length})^3}$
```
* Type 1 IUGR  
  → PI = Normal  
  → Causes:  
    - Chronic anomaly
    - Torch infections
    - Genetic disease

* Type 2 IUGR  
  → PI = ↓  
  → Causes:  
    - Placental insufficiency
    - PIH
    - Chronic Renal Disease

### Causes of IUGR

<table>
<thead>
<tr>
<th>Maternal</th>
<th>Placental</th>
<th>Fetal</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ PIH</td>
<td>→ Placental insufficiency</td>
<td>→ Chronic Anomaly</td>
</tr>
<tr>
<td>→ Placental insufficiency</td>
<td>→ Abnormal placentation</td>
<td>→ Genetic disease</td>
</tr>
<tr>
<td>→ Chronic renal disease</td>
<td>→ Calcifications</td>
<td>→ Infections</td>
</tr>
<tr>
<td>→ Heart Disease (NYHA Class III/IV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>→ Connective tissue disorder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>→ Infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>→ Diabetes with Vasculopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>→ Diabetes with PIH</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Diagnosis of IUGR

1. Identify mother at risk:
   1) PIH
   2) Chronic Renal Disease
   3) BMI ↓
   4) Less weight gain during pregnancy
   5) Infections

2. Sure about Gestational Age of fetus
   - Trimester
   - USG Parameter
   - 1st Trimester
     (upto 14 weeks) → Crown Rump Length
   - 14-20 weeks → Biparietal Diameter
   - 3rd Trimester → Femur Length
• Overall best time to do USG, to know gestational age: 1st trimester
• Best parameter: Crown Rump Length

Clinical Diagnosis:

- Symphysiofundal height (4 weeks - 32 weeks) ↑ by 1 cm/week

- Lag in symphysiofundal height by 4 cms → IUGR
- Measure the abdominal circumference of mother:
  - Abdominal circumference at 30 weeks = 30 inches
  Then ↑ by 1 inch/week after 30 weeks
- On USG, best parameter to assess growth of fetus → Abdominal circumference

**USG parameter to assess fetal growth**

1. Single measurement for fetal growth: Abdominal circumference (AC)

- To detect IUGR/macroamniota
- Plane for measuring AC: (following landmarks should be visible)
  - Fetal stomach
  - Umbilical vein
  - Portal sinus

plane for measuring AC

umbilical vein
Portal sinus
Fetal stomach
vertebral body and ribs
I. Ratio (Age independent):
   a) \[ \frac{\text{Head circumference}}{\text{Abdominal circumference}} \]

\[ \downarrow \]

Normally in Pregnancy
\[ \rightarrow \text{As pregnancy advances; AC } \uparrow \]
\[ \therefore \frac{\text{HC}}{\text{AC}} = \downarrow \]

In case of IUGR (asymmetric)
\[ \text{HC} = \text{Normal} \]
\[ \text{AC} = \downarrow \]
\[ \therefore \frac{\text{HC}}{\text{AC}} = \uparrow \]

b) \[ \frac{\text{Femur length}}{\text{Abdominal circumference}} = \text{normal} - 2.5\% \]

- In case of asymmetric IUGR:
  \[ \text{FL} = \text{N} \]
  \[ \text{AC} = \downarrow \]
  \[ \frac{\text{FL}}{\text{AC}} = \uparrow \text{ (} > 2.5\% \text{)} \]

- In asymmetric IUGR:
  \[ \text{Blood flow } \downarrow \text{(fetus)} \]
  \[ \downarrow \]
  \[ \text{Renal blood flow } \downarrow \]
  \[ \downarrow \]
  \[ \text{eGFR } \downarrow \]
  \[ \downarrow \]
  \[ \text{Urine output } \downarrow \]
  \[ \downarrow \]
  \[ \text{Oligohydramnios} \]
3. On USG: Amniotic Fluid Volume

- Amniotic Fluid Index
  - $< 5$ cm
  - Oligohydramnios

- Single largest vertical pocket
  - $< 2$ cm
  - Oligohydramnios

4. Placental morphology

- Batai plate
- Chorionic plate
- Cord

- Randomly dispersed echogenic areas
- Smooth indentation on chorionic plate

- Grade 0 Placenta.

- Grade 1 Placenta.

- Dense echogenic areas on batai plate
- Corrada shaped indentations on chorionic plate
- Distinct indentations
- Cotyledon Formation

- Grade 2 Placenta.

- Grade 3 Placenta.

Umbilical artery doppler

Best way to assess IUGR: Doppler

1st change: Amniotic fluid index +

Umbilical Artery Doppler

- Normal $S/D = \downarrow$
- IUGR /PIH $S/D = \uparrow$

$S \rightarrow$ Systolic

$D \rightarrow$ Diastolic
**Absent end diastolic flow**

- Terminate pregnancy @ 34 weeks

**Reverse diastolic flow**

- Immediately terminate pregnancy

---

**Middle cerebral artery and ductus venosus doppler**

**2nd change: middle Cerebral Artery**

- Normal
- IUGR (asymmetric)

- ↑ Blood flow during diastole
- ↓ S/D

**IUGR**

- S/D ratio in umbilical artery: ↑
- S/D ratio in mca: ↓
middle cerebral artery waveform

Color doppler examination of the circle of willis (left). Flow velocity waveforms from the middle cerebral artery in a normal fetus with low diastolic velocities (right, top) and in a growth-restricted fetus with high diastolic velocities (right, bottom).

3rd Artery: Ductus venosus

↓

Flow to IVC ↓

↓

↓ Blood in Ductus venosus

Fetal circulation
Doppler of Ductus Venosus:

- s - ventricular systole
- d - ventricular diastole
- a - atrial systole

1. Decreased blood in atrial systole
2. Absent blood in atrial systole
3. In atrial systole

Normal Waveform

Abnormal ductus venosus waveform

- ↓ Blood flow in atrial systole
- Reversal of blood flow
- Reversal of blood flow:
  - Atrial Systole
  - Ventricular diastole
1. Oligohydramnios
   ↓
   ↓ space in uterus
   ↓
   Cord of body gets compressed
   ↓
   Fetal distress
   ↓
   Fetus passes meconium in amniotic fluid
   ↓
   Fetus swallows meconium stained amniotic fluid
   ↓

2. Meconium Aspiration Syndrome

3. Low Birth Weight

4. Fetal distress / Hypoxia / Acidosis

5. Still birth

* Neonate:
  - Loose Skin
  - ↓ Tone
  - ↑ Hyaline Membrane Disease / Respiratory Distress Syndrome
  - ↑ Intraventricular Hemorrhage
  - ↑ Neonatal Death

Fetal surveillance in IUGR:

* Non - Stress Test → bi weekly

* Management
  1. Ca.
  2. mg
  3. Zn
  4. Anti oxidants
  5. Vit C, Vit E
  6. High Protein Diet (Exception: If pregnant female is malnourished)
ABORTION

Introduction

- Pregnancy loss before period of viability
  or
  Pregnancy loss at < 20 weeks of gestation
- Period of viability:
  USA = 20 weeks
  India = 28 wks
  WHO = 24 wks
- Weight of fetus at 20 weeks = 300 grams
- WHO definition
  “Pregnancy loss with weight of fetus < 500 grams”
  (at 24 weeks of pregnancy)

Classification of abortion

**USA Criteria:**

- Blighted ovum
  ▼
  - If gestation sac is visible but no yolk sac,
  - no fetal pole and no cardiac activity
  ▼
  - Mean Sac Diameter ≥ 25mm
    - No yolk sac
    - No fetal pole
    - No cardiac activity

- Missed abortion
  ▼
  - Gestational sac and yolk sac present fetal tissue identified
  - No cardiac activity
  ▼
  - Crown Rump Length ≥ 7 mm
    - No cardiac activity

- Abortion
  ▼
  - Spontaneous (isolated)
  ▼
  - Induced (MTP)
  ▼
  - Recurrent ≥ 3 pregnancy loss
  (Investigation should begin at ≥ 2 abortions)
Spontaneous abortion

- MC cause of isolated spontaneous abortion - Chromosomal abnormality
  \[ \downarrow \text{(germplasm defect)} \downarrow \]
  \[ \begin{array}{ll}
  1^{st} \text{ trimester} & (fetal cause) \\
  50\% & 35\%
  \end{array} \]

- MC time of spontaneous abortion < 8 weeks

- MC chromosomal defect for abortion:
  - Best answer: Aneuploidy
  - 2nd best: Trisomy
  - 3rd best: Monosomy X (20\%)
  - 4th best: Trisomy 16 (16\%)

- Most lethal trisomy - Trisomy 16
- Most viable trisomy - Trisomy 21

- MC sex chromosome abnormality - Klinefelter's syndrome
  \[ \downarrow \]
  \[ \text{(1 in 1000 pregnancy)} \]

- MC sex chromosome abnormality - Turner's syndrome
  \[ \downarrow \]
  \[ \text{(1 in 2500 pregnancy)} \]

- Most important risk factor for spontaneous abortion
  - Best: Maternal age
  - 2nd best: Previous history of abortion

- Previous history of
  - 1 abortion = 20\% risk of recurrence
  - 2 abortions = 30\% risk
  - 3 abortions = 40-50\%

- Infections can lead to single episode of abortion
- MC time for abortion = 1\textsuperscript{st} trimester (< 8 weeks)
Recurrent abortions

Causes:

1. Uterine causes (MC) (10-15%)
   -> Uterine malformation
     (MC - septate uterus)
     Cervical incompetence (MC)
     Fibroid
     Endometrial polyp

   Uterine causes lead to 2nd trimester recurrent pregnancy loss
   Investigation
   "TVS"
   (diagnose cervical incompetency)
   1. HSG
   2. Saline infusion sonography
   3. Hysteroscopy
   4. Laparoscopy

Causes for recurrent abortion

a. Anti phospholipid Antibody syndrome: (5-15%)
   - Investigations:
     * Antibodies:
       - Lupus Anticoagulant
       - Anti cardiolipin Ab
       - Anti β2 microglobulin Ab

3. Chromosomal abnormality
   Balanced translocation of chromosomes
   Investigation: Karyotyping
4. Hypothyroidism
   ↓
   TSH levels

Probable causes of Recurrent Pregnancy Loss (RPL):
1. Diabetes
2. Increased prolactin levels
3. Luteal phase defect:
   ↓
   In pregnant female progesterone level < 5 ng

Infections:
- Never lead to recurrent pregnancy loss
  ↓
  Except: Syphilis
  ↓
  "Kassowitz law"

  "As number of pregnancy losses increase,
  the period at which the pregnancy ends also increase"

- Syphilis does not lead to early recurrent pregnancy loss
- Investigation: VDRL test

Investigations: No role in recurrent pregnancy loss
1. TORCH test
2. Serum progesterone levels
   - MC cause of Recurrent pregnancy loss - uterine causes
     (10-15%) > AP
   - Anti phospholipid syndrome - several autoimmune diseases
   - Many been linked to poor obstetric outcomes

But it is the only immune condition in which pregnancy loss is a
   diagnostic criteria for the disease
- 5-15% of patients with recurrent pregnancy loss may have
  Antiphospholipid syndrome
- MCC immunological cause for RPL: APLA
- MC cause of 1st trimester recurrent abortions - Idiopathic
  abortions
- MCC of 2nd trimester recurrent abortions - uterine causes
Anti-Phospholipid Antibody syndrome (APLA)

- Antibodies are present
- MC antibodies:
  1. Lupus anti coagulant (misnomer)
  2. Anti cardiolipin Ab
  3. Ab against β groundbreaking Glycoprotein 1
- APLA leads to both arterial and venous thrombosis in placental blood vessels

\[\text{Placental blood supply} \downarrow \quad \text{Placental blood supply stops} \quad \text{PIH} \]

\[\text{IUGR} \quad \text{Abortion} \quad \text{IUD} \quad \text{Still birth} \]

- mc time=2nd trimester
- Size of placenta reduces - uteroplacental insufficiency

**Diagnosis of APLA**

Revised/modified sapporo criteria
(earlier 1 clinical + 2 lab, now 1 lab + 1 clinical)

Clinical:
1. Arterial / venous Thrombosis (superficial / deep)
2. ≥ 3 losses in < 10 weeks (recurrent losses)
3. ≥ 1 loss in > 10 weeks of morphologically normal fetus
4. At least 1 preterm delivery secondary to severe PIH/uteroplacental insufficiency

Lab criteria:
1. Presence of lupus anticoagulant Ab
2. Presence of IgM/IgG anticoagulant Ab
3. Presence of β micro globulin Ab on 2 occasions, 12 weeks apart

- In APLA:
  - APTT prolonged
  - PT - normal
- 'Russel viper venom' clotting time - prolonged

**Management of APLA**

- **Aspirin** (low dose) = 50-100 mg (80 mg)
  
  APLA diagnosed based on history of Abortion

  ![Aspirin Diagram]

  - **Aspirin**
    - Started as UPT +ve
    - Through out pregnancy
    - Stopped 4-6 days before labour

  - **Heparin**
    - LMWH > unfractionated heparin
    - Started only after confirming it is intra-uterine pregnancy
    - Continued throughout pregnancy
    - Stopped at onset of labour

  APLA is diagnosed based on criteria of PTL (preterm labour)

  Give only: **Aspirin**

  Time: at end of 1st trimester

  Continue throughout pregnancy

  - DOC for management of APLA - Warfarin
  
  DOC for management of APLA in pregnancy - Aspirin

**Warfarin**

- Contraindicated in pregnancy
- Reason: It can cross placenta.

  Limit vitamin K activity in fetus
Osteocalcin needs vitamin K for carboxylation

Warfarin embryopathy

Bone and cartilage deformity

Warfarin embryopathy

1. Stippled epiphysis
2. Nasal hypoplasia
3. Limb hypoplasia

Resembles "chondrodisplasia punctata."
(X-linked disease)

Cervical incompetence

- m/c/cause of 2nd trimester recurrent pregnancy loss
- Painless, sudden dilatation of internal OS
  ↓ (2nd trimester)
  Rupture of membranes → Expulsion of fetus

Etiology:

Congenital       Acquired (most commonly)

1. Forceful dilatation
   of cervix (D&C)
2. Cervical surgeries
   (least chances-cryosurgery)
Diagnosis:

- Non pregnant
  - History
    1. Recurrent pregnancy loss
    2. Only of 1st trimester
    3. Painless dilatation of cervix
    4. Number of losses ↑
  - POG at which loss occurs ↓
  - Hegar’s Dilation:
    If No. 8 Hegar’s dilator can be passed
    - Through internal OS
    - Without patient offering any resistance

- Pregnant
  - TVS
  - Cervical length
  1. Length of cervix = < 2 cm
  2. Dilatation = > 2 cm
  3. Shape of cervix on USG = U shaped
  - Normally cervix is “T shaped”
  - As cervix dilates – “Y shaped”
  - “U shaped”

- Hystero cervicography
  - Normal
  - Incompetent os
  - “Funnel shaped appearance”
**Management**

**Cervical cerclage (vaginally) - mc**

- Mc Donald cerclage (mc)
- Shirodkar cerclage

Purse string suture around internal os

Fails

**Abdominal cerclage: “Bensen and Durfee cerclage”**

**Indications for cervical cerclage**

1. **History based indications:**
   - Recurrent abortions of and trimester (≥ 3)
   - History of preterm labour

2. **USG based indication**
   - Length of cervix < 2.5cms
     
     Patient given history of ≥1 a nd trimester abortion

   - Emergency / Rescue cerclage
     
     Cervix has begun dilating and then cervix cerclage
     
     Dilatation < 4 cm and membranes should not be ruptured
- Time for cerclage - 14-24 weeks (12-14 weeks)
  - Cannot do after 24 weeks
- Time to remove suture - 37 weeks of pregnancy
  or
  at the onset of labour
- Earlier removal - Rupture of membrane, preterm labour, fetal distress

**Contraindications for cerclage**

1. Dilatation of cervix ≥ 4cms
2. Membranes are ruptured
3. Presence of gross anomaly of fetus
4. Vaginal bleeding present
5. Presence of pelvic infections
6. Placenta previa (relative contra indication)

**Types of abortion**

<table>
<thead>
<tr>
<th>Types</th>
<th>Definition</th>
<th>Symptoms</th>
<th>P/A examination</th>
<th>Internal OS</th>
<th>USG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Threatened abortion</td>
<td>Process of abortion has begun but it is at a stage from where it can be reversed</td>
<td>Spotting PV</td>
<td>Height of uterus = period of gestation</td>
<td>Closed</td>
<td>Live fetus - cardiac activity present</td>
</tr>
</tbody>
</table>

POC - products of consumption
<table>
<thead>
<tr>
<th></th>
<th>Inevitable abortion</th>
<th>Incomplete abortion</th>
<th>Complete abortion</th>
<th>Missed abortion</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.</td>
<td>Cannot be reversed (POC has not started coming out)</td>
<td>POC has started coming out but process is not yet complete</td>
<td>Entire process of abortion is complete on its own</td>
<td>Fetus dead but patient is unaware</td>
</tr>
<tr>
<td></td>
<td>Bleeding +/- pain in abdomen</td>
<td>c/o = POC coming out + bleeding + pain in abdomen</td>
<td>Initially = bleeding + pain + product of conception comes out → bleeding stops</td>
<td>Brownish vaginal discharge</td>
</tr>
<tr>
<td></td>
<td>Ht of uterus = period of gestation</td>
<td>Ht of uterus less than pd of gestation</td>
<td>Height of uterus less than period of gestation</td>
<td>Ht of uterus less than pd of gestation</td>
</tr>
<tr>
<td></td>
<td>Open</td>
<td>Open + POC can be seen coming out through it</td>
<td>Closed</td>
<td>Closed</td>
</tr>
<tr>
<td></td>
<td>Dead fetus No cardiac activity</td>
<td>Incomplete product of conception</td>
<td>Empty uterus</td>
<td>Dead macerated fetus or CRL &gt; 7 mm, no cardiac activity</td>
</tr>
</tbody>
</table>
Anti-D prophylaxis in RH-negative

Female who has had abortion:

Abortion at

\[ < 12 \text{ weeks} \quad \rightarrow \quad \text{Dose: 50 mcg} \]

\[ > 12 \text{ weeks} \quad \rightarrow \quad \text{Dose: 300 mcg} \]

\[ \rightarrow \text{Given in all types of abortion including threatened and complete abortions} \]

\[ \rightarrow \text{Given in all types except threatened abortion} \]
MEDICAL TERMINATION OF PREGNANCY (MTP)

**MTP ACT**

- MTP act was passed in 1971
- Came into action in 1972
- MTP can be done only up to 20 weeks of pregnancy
- If MTP done at
  - < 12 weeks
    - Single doctor opinion
  - ≥ 12 weeks
    - Two doctors opinion needed

- Consent for MTP
  - If the female ≥ 18 yrs
    - Her consent needed (written)
    - Husband's consent not required
  - If the female < 18 yrs / not mentally sound
    - Written consent from Parents / guardians

- MTP can be done by
  - MBBS Doctor
    - Assisted at least 25 cases of MTP
  - Degree in gynaecology / Diploma in gynaecology & Obstetrics
    - Resident in Obstetrics and gynaecology for 6 months

- MTP can be done in
  - Government hospitals
  - Private centres approved by the government
Indications of MTP

- Maternal
  - Clark's class III heart disease
  - Chronic renal disease
  - Malignant hypertension
  - Psychiatric illness
    - Schizophrenia

- Fetal
  - Radiation exposure
  - Teratogenic drug exposure
  - Rubella infection

- Humanitarian
  - Rape

- Social ground
  - Low socio economical status
  - Failure of contraception

Congenital Rubella Syndrome
  - Mental retardation
  - Sensorineural hearing loss
  - Microcephaly
  - Micropthalmia
  - Congenital cataract
  - Congenital heart disease (PDA)

Methods of MTP

1st trimester methods
  - Medical abortion
    - Misoprostol
    - Mifepristone
    - Methotrexate

2nd trimester methods
  - Menstrual regulation
  - Suction evacuation
  - Vacuum curettage
  - Aspiration
  - Manual dilation

First trimester methods

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MTP - Second trimester methods

- Prostaglandins (PG)
  - PGF - a - Carboprost - Im
  - PGE - a - Dinoprost
  - PGE 1 - misoprost
  - PGE 2 analogue - Esmoprost

- Suction evacuation
  - 0.5 %

- Extra amniotic ethacridine
  - Instillation
  - Of hypertonic solution
  - Urea,
  - Mannitol,
  - Hypertonic saline

Best method of doing MTP

- Period of pregnancy
  - ≤ 7 weeks
  - 7-15 weeks
  - ≥ 15 weeks

Medical abortion

- WHO / ACOG recommendation - Medical abortion can be done upto 9 weeks (63 days)
  - In India - Medical abortion done only upto 7 weeks (49 days)

- Before initiating medical abortion
  - Intrauterine pregnancy should be confirmed on ultrasound
  - Patient should be compliant to follow up / Abortion

Protocol for medical abortion at < 7 weeks and 7 - 9 weeks

At < 7 weeks

- On Day 1 - oral mifepristone - 200 mg (1 tablet)
  - RU 486 / anti progesterone compound - kills the fetus
• On Day 3 - 400 mcg (2 tablets) of misoprost (vaginally or orally)
  
  Teratogenic  
  Contraindicated during surgery  
  Moebius syndrome  

  It is PGE 1  
  Causes uterine contraction  
  Expels dead fetus  

• Within 2 to 3 days the patient will experience heavy bleeding  
• On Day 15 - To ensure that the process of abortion is complete  
  • In 99% - abortion will be complete  
  • In 1% - abortion is not complete  
  
  Dilation & curettage to complete it  

At 7 - 9 weeks  
• On Day 1 - Oral mifepristone - 200 mg  
• On Day 3 - 800 mcg (4 tablets) of vaginal misoprost  
• On Day 15 - To ensure the process of abortion is complete  
• Success rate - 95%

Suction evacuation  

• It is done using plastic cannula - Karman cannula  

• 4 – 12 number is written on cannula  
  • Indicates the diameter of cannula in millimeter  
  • The number of Karman cannula - corresponds to the week of pregnancy or it should be 1 less  

• The cannula is attached to suction machine  
  
  Generates 600 mm of Hg pressure - sucks the fetal parts
Before passing Karman cannula - the internal os dilated ↓ using Hegar dilator

- Complication - uterine perforation
  Occurs while dilating internal os ↓ while doing suction evacuation ↓
  wait & watch ↓ Serious injury ↓
  Let the cannula remain inside ↓
  Patient should be taken for Laparotomy immediately

Menstrual regulation

- Done within 3 weeks after a missed period i.e. 7 weeks of amenorrhea, using a 50 ml syringe

- In rural areas - Manual vacuum aspiration ↓
  using 60 ml syringe and 6 - 12 mm cannula.
  Pressure of 660 mm of Hg
  used up till 12 weeks
  doesn’t require electricity - best for rural settings
Prostaglandins

* Done in 2nd trimester (beyond 15 weeks)

<table>
<thead>
<tr>
<th>Analogue</th>
<th>Name</th>
<th>Route</th>
<th>Dose</th>
<th>Maximum dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>PGF - 2α</td>
<td>Carboprost</td>
<td>IM</td>
<td>250 mcg (3 - 4hrly)</td>
<td>10 doses</td>
</tr>
<tr>
<td>PGE 1</td>
<td>Gemeprost</td>
<td>Vaginal pessary</td>
<td>1 mg (3- 4 hrly)</td>
<td>5 doses</td>
</tr>
<tr>
<td>PGE 1</td>
<td>Misoprost</td>
<td>Oral/ vaginal tablet</td>
<td>200 mcg to 800 mcg (3 - 4 hrly)</td>
<td>4 doses</td>
</tr>
<tr>
<td>PGE 2</td>
<td>Dinoprost</td>
<td>Vaginal pessary</td>
<td>20 mg</td>
<td></td>
</tr>
</tbody>
</table>

Extra amniotic Ethacridine

* A Foley's catheter is placed in between the membrane & uterus

Through Foley's - Pass ethacridine (0.1%)

Dose - 10 ml x weeks of gestation
Maximum - 150 ml
Foley's catheter left for - 4 - 6 hrs

* Abortion begins by - 12 - 48 hrs - Separates membrane from uterus

releases prostaglandins

initiates uterine contraction

abortion
INTRODUCTION AND MANAGEMENT OF RUPTURED ECTOPIC PREGNANCY

Ectopic pregnancy

- Implantation occurs outside uterus

- Ectopic pregnancy

  - mC Site (overall)
  - mC Non-tubal site
  - Overall least common site

  - Fallopian tube
  - Ovary

  - Ectopic in cesarean 0.1%
  - Or
  - Cervical ectopic 0.1%

  - Least common site for ectopic pregnancy
  - (Fertilization)

  - Intrauterine pregnancy
  - In the tubes:
    - Ampulla > Isthmus > Infundibulum > Interstitial

  - Least common

- Least common site of Ectopic pregnancy → Cervical ectopic / ectopic in cesarean scar

- Least common site of Ectopic in tubes → Interstitial

Cornual pregnancy and intrauterine pregnancy

- Cornual pregnancy
  - Ectopic pregnancy which occurs in interstitial / Rudimentary horn of a bicornuate uterus

- Intrauterine pregnancy
  - Close to the angles of uterus
  - AKA Angular pregnancy
**Important points**

- **Ectopic pregnancy**
  - Ends earliest
  - Lasts longest in fallopian tube
  - Lasts longest overall
  - Isthmus
  - Interstitial (myometrium of uterus supports pregnancy at this site)
  - → Abdominal Ectopic
  - → Heterotopic pregnancy
  - Twin pregnancy:
    - 1st pregnancy → Intrauterine
    - 2nd pregnancy → Ectopic

Narrowest part of the tube - Interstitial
and narrowest part of the tube - Isthmus
Risk factors for ectopic pregnancy

1. Highest risk of Ectopic pregnancy:
   Previous history of ectopic pregnancy > tubal surgery
   - Chance of Recurrence:
     - If history of previous one episode: 15%
     - Of Ectopic pregnancy
     - In previous 2 episodes: 30%

2. MC Risk factor: Salpingitis / PID
   - PID includes:
     Salpingitis
     Oophoritis
     Endometritis

3. Other Risk factors: IVF / ART

4. Smoking

5. Early age of intercourse

6. Multiple sex partners

7. Low socio-economic status

8. Any adhesions inside tube:
   - Endometriosis / Appendicitis
   - Theory of Retrograde menstruation
1. Salpingitis isthmica nodosa.

- Zygote can fall in one of the pockets of fallopian tube and implant there.

**Role of contraceptive agents in ectopic pregnancy**

- ↓ the absolute risk of ectopic pregnancy (↓ overall chance of any type of pregnancy)
- But on failure → ↑ chance of ectopic pregnancy
- M.C: Tubectomy > IUCD > POP (progesterone only pills)

  Progestasert > MIRENA > Cu IUCD

- Least chance: OCP and with vasectomy failure

**Symptoms of ectopic pregnancy**

- Most consistent symptom of ectopic pregnancy

  **Pain in abdomen**

  - Ruptured ectopic pregnancy
    - Hemo peritoneum
    - Irritates Diaphragm
    - Shoulder tip pain
      (AKA: Danforth sign)
  - Unruptured ectopic pregnancy
    - Stretching of fallopian tube

- In 50% cases: Triad

  Amenorrhea
  (6-10 wks)
  Pain in abdomen
  Bleeding
• a reactions brought by progesterone during pregnancy:
  
  Converts endometrium into decidua.
  
  Arias Stella Reaction
  
  Characterized by:
  → enlarged glands in endometrium
  → abundant cytoplasm
  → Hobnail Nucleus

  Seen in:
  • Intrauterine pregnancy
  • Ectopic pregnancy
  • Molar pregnancy

• In patients of ectopic pregnancy:
  
  Progesterone is present (less amount)

  Decidua is shed in the form of Decidua cast

  Made of Decidua Vera.

• Signs:
  1. Decidua cast
  2. Cervical motion tenderness
     → Differential diagnosis: PID
  3. Presence of Adnexal mass
Diagnosis and management of ruptured ectopic pregnancy

Diagnosis:
- Clinical Diagnosis
- Complains of: Amenorrhea (6-10 weeks)
  + Pain in abdomen
  + Shock
  +/− Bleeding P/V

\[ \text{urine pregnancy test positive} \]

Ruptured Ectopic pregnancy

Management:

\[ \downarrow \]
Laparotomy
(Preferred)
\[ \downarrow \]
Vitals unstable

\[ \downarrow \]
Laparoscopy
Can be done only if
vitals of patient are stable

- Surgery of choice: Ruptured Ectopic
  \[ \downarrow \]
  Remove the tube which is damaged
  \[ \downarrow \]
  Salpingectomy
  (whether female is nulliparous or multiparous)

- Following are not done in ruptured ectopic
  1. Medical management
  2. Expectant management
  3. Any other surgery except salpingectomy

- Salpingo Oophorectomy is never done in ectopic pregnancy
  (Tubes are removed in ruptured ectopic, ovaries retained)
**Culdocentesis**

- A needle is passed via posterior fornix
- Non clotting blood → Hemoperitoneum
- Blood which clots → Needle pierced an artery

- Doesn't confirm if it's ruptured ectopic pregnancy
MANAGEMENT OF UNRUPTURED ECTOPIC PREGNANCY AND SALPINGECTOMY

Diagnosis of unruptured ectopic

IOC: TVS (Transvaginal USG)

↓

1) Empty uterus
   ("sign: indicates ectopic pregnancy)
2) Pseudo gestational sac
   a) Tubes & Adnexa.
      i) Gestational sac or cardiac activity in tubes
         ↓
         "Bagels sign"
         ↓
         Confirm- Doppler
         ↓
         "Ring of fire appearance"
         a) Complex adnexal mass

Decidua

Decidua basalis
↓
Separates blastocyst from myometrium
↓
Forms maternal side of Placenta.

Decidua capsularis
↓
Separates blastocyst from uterine cavity

Decidua parietalis
↓
Rest of decidua.

Active space

Central Cavity Complex

→ uterine cavity
→ Blastocyst
→ Decidua capsularis
→ Decidua basalis
→ Decidua parietalis

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Intra - decidual sign on USG:

- In early pregnancy as the blastocyst / gestational sac implants in the decidua.

↓

If interstitial implantation occurs and does not displace / deform the hyper echogenic central cavity complex

↓

Called as "intra decidual sign"

Intra decidual sign:

- Gestational sac
- Central cavity complex

Double decidual sac sign

Seen only in intra-uterine pregnancy

- 2nd ring: Decidua parietalis
- 1st ring: Decidua capsularis

Double decidual sac sign:

- 1st sac
- 2nd ring (decidua capsularis)

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Difference between true and pseudogestational sac

True gestational sac
- seen in intrauterine pregnancy
- eccentric in location
- ↑ in size (≤3mm/day)
- Double decidual sign/
  Double ring sign (+)

Pseudogestational sac
- seen in ectopic pregnancy
- Thick decidua
- Centrally located
- Size remains constant
- Double ring sign (−)

TVS findings in unruptured ectopic pregnancy

Empty uterine cavity

Early Intrauterine Pregnancy

Abortion

Ectopic pregnancy

β hCG levels measured

Critical titre of hCG:
- Value of hCG at which gestational sac should be visible on USG (TVS)

TVS = 1500 IU/L (1000-2000)
TAS = 6500 IU/L (> 5000)

β hCG levels:

≥ 1500 IU
- Ectopic pregnancy

< 1500 IU
- Repeat β-hCG after 48 hrs
In an intrauterine pregnancy, β hCG levels roughly double in 48 hrs (≥ 60%)

If hCG double (≥ 60%)

- Early intrauterine pregnancy

hCG↓

Abortion

hCG↑ but do not double (↑ < 60%)

Ectopic pregnancy

Other investigations

1. Serum progesterone levels:
   - ≥ 25 ng/ml → intrauterine viable pregnancy
   - < 5 ng/ml → during pregnancy (abortion / ectopic)

→ IOC: TVS
   - Best: TVS + β hCG
   - Gold standard: Laparoscopy

Investigations not done in ectopic pregnancy:

1. HSG
2. Hysteroscopy
3. Colpotomy - For draining pelvic abscess
Management of unruptured ectopic pregnancy

1. Expectant management
   - Wait and watch
   - Risk of rupture
   - Not preferred
   - DOC: methotrexate

2. Medical management
   - Management of choice
   - Preferred in case of unruptured ectopic pregnancy
   - Laparoscopy (preferred)
   - Laparotomy

Pre-requisites
1. Only in unruptured ectopic pregnancy
2. Hemodynamically stable patients

→ Conditions:
   - Size of ectopic < 3 cm
   - β hCG < 1000 IU
     (Best = < 500 IU)
     and decreasing
   - Cardiac activity
     absent

→ Conditions:
   - Size of ectopic < 4 cm
   - β hCG < 5000 IU
   - Cardiac activity
     preferably absent

Medical management of ectopic pregnancy

→ DOC: methotrexate
   - Single dose methotrexate
   - Dose: 50 mg/m²

→ Day 1: Check βhCG of patient
   Inji. Methotrexate IM
→ **Day 4**: Repeat β hCG
   
   → **Day 7**: Repeat β hCG ≥ 15%
   
   Medical management is successful

→ If fall is < 15% between day 4 and 7
   
   Repeat methotrexate injection
   
   New β hCG levels recorded (Day 1, 4, 7)

→ inj- methotrexate × 3 times
   
   Fall is < 15%
   
   Failed medical management
   
   Surgical management

**Surgical management**

→ Laparoscopic surgery is preferred

→ Surgery depends on age / Parity of patient

Family not complete

Family is complete

**Linear Salpingostomy (Preferred)**

→ Remove ectopic by hydrodissection

→ No suture on incision site

**Salpingostomy**

Similar procedure but with sutures incision site

**Salpingectomy**
### Other sites of ectopic

<table>
<thead>
<tr>
<th>Other sites</th>
<th>Criteria for diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical ectopic</td>
<td>Palman criteria</td>
</tr>
<tr>
<td>Abdominal ectopic</td>
<td>Rubin criteria</td>
</tr>
<tr>
<td>Ovarian ectopic</td>
<td>Studdiford criteria</td>
</tr>
<tr>
<td></td>
<td>Spiegelberg criteria</td>
</tr>
</tbody>
</table>

**Indications of salpingectomy in ectopic**

1. Ruptured ectopic
2. Unruptured ectopic (family is complete)
3. Size of ectopic > 5 cms
4. Hemostasis cannot be maintained
5. Heterotopic pregnancy

→ Earlier: Incidence = 1 in 30,000 pregnancy
   
   now: Incidence ↑ due to ART/IVF

   ↓

   1 in 3000 pregnancy

→ Management of choice → Surgery

↓

Salpingectomy
MOLAR PREGNANCY: HYDATIDIFORM MOLE

Classification

molar pregnancy

Gestational trophoblastic disease

Hydatidiform mole
1. Partial mole
   a. Complete mole

trophoblastic neoplasia

1. Invasive mole
2. Choriocarcinoma
3. Placental Site Trophoblastic Tumor (PSTT)
4. Epithelial Trophoblastic Tumor (ETT)

molar pregnancy

molar diseases
1. Partial mole
2. Complete mole
3. Invasive mole
On HPE, chorionic villi are present

Rest of them
1. Choriocarcinoma
2. PSTT
3. ETT
On HPE, no chorionic villi are present

Molar pregnancy - risk factors

- Benign disease of the chorion with malignant potential
- More common in developing countries → Asians
- Maximum incidence → Philippines
- Risk factors:
  1. Increased maternal age
     (≥ 35y → 2 times the chance
      ≥ 40y → 7 times the chance)
  2. Asian population
  3. Diet: Deficiency of Vitamin A
4. Previous history: Molar pregnancy - 1-4% risk of recurrence
   a Molar pregnancy - 25% risk of recurrence

5. Others: Smoking
   OCP use
   AB blood group

Molar pregnancy: pathology

1) Undue proliferation of trophoblasts
   - Some fetal parts are present
   - No fetal parts are present
   - Partial mole
   - Complete mole
   - ↑ HCG levels
   - PIH
   - ↑ Height of uterus (Height > gestational age)
   - Pregnancy induced hypertension before 20 weeks

2) Hydropic degeneration
   - Effect of ↑ HCG:
     1. Hyperemesis gravidarum: Excessive nausea & vomiting
     2. Thyrotoxicosis: α subunit of HCG is similar to TSH
     3. Theca lutein cysts

   - On USG, Snow storm Appearance
<table>
<thead>
<tr>
<th>Partial mole</th>
<th>complete mole</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Karyotyping</strong>:</td>
<td><strong>Karyotype</strong>:</td>
</tr>
<tr>
<td>Triploid, dispermic</td>
<td>Diploid, monospermic (90%)</td>
</tr>
<tr>
<td><img src="image" alt="Ova" /> Sperms</td>
<td><img src="image" alt="Empty Ova" /> Sperm</td>
</tr>
<tr>
<td>(69 xxx or 69 xxy)</td>
<td>Duplication of genetic material of sperms</td>
</tr>
<tr>
<td>The extra genetic material is Paternal in origin</td>
<td>The entire genetic material is paternal and is Called Androgenesis</td>
</tr>
<tr>
<td></td>
<td>in 10% cases, dispermic</td>
</tr>
<tr>
<td><img src="image" alt="Empty Ova" /> Sperms</td>
<td></td>
</tr>
<tr>
<td><strong>Histopathology</strong>:</td>
<td><strong>Histopathology</strong>:</td>
</tr>
<tr>
<td>- Hydropic degeneration is less</td>
<td>- Hydropic degeneration is more marked</td>
</tr>
<tr>
<td>- Some fetal parts are present</td>
<td>- No fetal parts are present</td>
</tr>
<tr>
<td>- Trophoblastic scalloping present</td>
<td>- Trophoblastic scalloping and inclusion bodies are absent</td>
</tr>
<tr>
<td>- Inclusion bodies present</td>
<td></td>
</tr>
<tr>
<td><strong>Symptoms,</strong></td>
<td><strong>Symptoms,</strong></td>
</tr>
<tr>
<td>- Most common: Bleeding p/v - expulsion of grapelike vesicles</td>
<td>- Similar to partial mole but all symptoms are more marked like:</td>
</tr>
<tr>
<td>- Other symptoms are less marked or absent</td>
<td>- PIH (27% cases)</td>
</tr>
<tr>
<td></td>
<td>- Hyperemesis gravidarum (25%)</td>
</tr>
<tr>
<td></td>
<td>- Thyrotoxicosis</td>
</tr>
<tr>
<td></td>
<td>- Respiratory distress due to embolization of Trophoblastic tissue</td>
</tr>
<tr>
<td><strong>Investigation - USG/TVS</strong></td>
<td><strong>Investigation - USG/TVS</strong></td>
</tr>
<tr>
<td>- Resembles missed abortion</td>
<td>- Snow storm appearance</td>
</tr>
</tbody>
</table>
• Levels of HCG
  ↑ but less than $10^4$ IU/L
• Theca lutein cysts are absent
• Chances to progress to gestational trophoblastic neoplasia (GTN) 3-5%
• Chances of choriocarcinoma <1%
• Immunostaining with p57, Kip 2 → Positive
  (maternal gene) ↓ Ova
                     ↓ Sperms
  • Levels of HCG ≥ $10^5$ IU/L
• Theca lutein cysts are present
• Chances to progress to GTN 15-20%
• Chances of choriocarcinoma 4%
• Immunostaining with p57, Kip 2 will be negative
  Empty Ova

• In molar pregnancy, investigation of choice = TVS
  Investigation of choice in follow up = $\beta$ HCG
  Gold standard investigations = Histopathological examination (HPE)

Management of molar pregnancy

• Treatment of choice: Suction Evacuation
  ↓ Irrespective of gestational age of molar pregnancy
  ↓ If required, curettage is done
  ↓ Tissue is then sent for HPE

• Oxytocin drip should not be started before evacuation as there are chances for embolization - RCOG 2010
• Size of cannula used = 10-12
• Anti-D administration in all Rh−ve mothers, after evacuation
• Theca lutein cysts spontaneously regress following evacuation (Chocolate cysts in endometriosis requires laparoscopic removal)
• Indications for hysterectomy in molar pregnancy
  - Age ≥ 40y with completed family
  - Uncontrolled hemorrhage following suction evacuation
  - Invasive mole
• After suction evacuation or hysterectomy

  Follow up with $\beta$ hCG levels is must
• Hysterectomy does not decrease the risk of developing choriocarcinoma.

**Follow up in molar pregnancy**

• measure $\beta$ hCG weekly

  Till 3 consecutive normal values

  Then monthly for 6 months

• Pregnancy is contraindicated for 6 months following evacuation of molar pregnancy
  Contraception of choice - OCP: Oral contraceptive pills

• Following evacuation, $\beta$ hCG levels become normal after:
  In partial mole - 7 weeks
  In complete mole - 9 weeks

• Prophylactic chemotherapy:
  - Not given to all patients after evacuation
  - Only given to high risk patients like:
    * Age ≥ 40 years
    * HCG levels ≥ $10^5$ IU/L
    * Large uterine size
    * Bilateral theca lutein cysts ≥ 6cms

• Prophylactic chemotherapy, drug of choice
  methotrexate or Actinomycin D (if jaundice present)
ANTEPARTUM HEMORRHAGE

Definition:
Bleeding in or from genital tract beyond period of viability
(in India - 28 weeks)

Causes

- Placental causes (m.c)
  - Abruptio placenta (m.c)
  - Placenta previa
  - Circumvallate placenta
  - Circummarginate placenta

- Fetal causes
  - Vasa previa

Placenta previa and abruptio placenta

Placenta previa
- Placenta in lower uterine segment
- To localize placenta → USG
done in 3rd trimester

Repeat scan done at:
- 36 weeks → minor degrees
- 32 weeks → major degrees

Incidence:
- 1 in 300 pregnancies
- 1 in 200 pregnancies

Recurrence rate:
- 5%
- 12–15%

Risk factor
- Previous history of placenta previa (m.c)
- h/o C-section (a.m.c)
- Previous history of abruptio placenta
Common risk factors:
- Multiparity
- ↑ Maternal age
- Smoking
- Twin pregnancy

APH - m.e. in multiparous
- Smoking is a risk factor

PIH - m.e. in nulliparous
- Smoking is protective

Other risk factors

<table>
<thead>
<tr>
<th>Placenta previa</th>
<th>Abruptio placenta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Placenta is big in</strong></td>
<td>Cigarette smoking + cocaine</td>
</tr>
<tr>
<td>Twins</td>
<td>Folic acid deficiency</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Thrombophilia</td>
</tr>
<tr>
<td>Succenturiate lobe</td>
<td>PIH/HTN</td>
</tr>
<tr>
<td>Placenta bilobate</td>
<td>Abdominal Trauma</td>
</tr>
<tr>
<td></td>
<td>Fibroid</td>
</tr>
<tr>
<td><strong>2 Defective endometrium in</strong></td>
<td>PROM</td>
</tr>
<tr>
<td>Endometritis</td>
<td>Polyhydramnios</td>
</tr>
<tr>
<td>Smoking</td>
<td>Twin pregnancy</td>
</tr>
<tr>
<td>Curettage</td>
<td></td>
</tr>
<tr>
<td>Previous surgery</td>
<td></td>
</tr>
<tr>
<td>(C-section,</td>
<td></td>
</tr>
<tr>
<td>myomectomy)</td>
<td></td>
</tr>
</tbody>
</table>

Classification of placenta previa

Type 1: Anterior & posterior
- Placenta in lower uterine segment (Lus)
  (distance from internal os <4cm)

Type 2: Placenta reaches upto margin of internal OS
• Does not reach upto internal os
  Aka lateral/ Low lying placenta previa

aka. marginal placenta previa
- Anterior & posterior types
  Type 2 posterior
  less dangerous variety

Type 3:
• Placenta partially covers internal OS
  A/k/a Incomplete placenta previa
• Placenta does not cover dilated OS.

Type 4:
Placenta completely covers internal OS.
Aka complete/ Central placenta previa.

Minor degrees of placenta previa:
  Type 1 anterior & posterior
  Type 2 anterior
• Vaginal delivery can be tried

Major degrees of placenta previa:
  Type 2 posterior
  Type 3
  Type 4

Cesarean section - Always

In all posterior varieties of placenta previa:
  Fetal distress on pushing head into pelvis, perabdominally
  → Stallworthy Sign

Classification of abruptio placenta

Page classification:

Grade 0: Retrospective diagnosis
  Based on retroplacental clot

Grade 1: Bleeding PV + pain abdomen (due to thromboplastin)
Grade 2: Bleeding PV + pain abdomen
Fetal distress/death

Grade 3: Bleed PV + pain abdomen
Fetal death + mother in shock
+/- DIC (thromboplastin)

Varieties of abruptio:
1. Revealed variety

2. Concealed variety - blood collected
   Behind placenta
   ↓
   Enters myometrium
   ↓
   Uterus appears bluish / wine coloured
   K.a. Couvelaire uterus
   ↓
   a) Not an indication for hysterectomy
   b) Risk factor for PPH

* mc variety of abruptio → mixed variety

Presentation of placenta previa and abruptio

<table>
<thead>
<tr>
<th>Placenta previa</th>
<th>Abruptio placenta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding in 3rd trimester</td>
<td>Bleeding in 3rd trimester</td>
</tr>
<tr>
<td>a) Bright red in colour</td>
<td>a) Dark red in colour</td>
</tr>
<tr>
<td>b) Painless</td>
<td>b) Pain in abdomen</td>
</tr>
<tr>
<td>c) Causeless</td>
<td>c) Cause</td>
</tr>
<tr>
<td>d) Recurrent in same pregnancy</td>
<td>Trauma (accidental hemorrhage)</td>
</tr>
<tr>
<td>e) Warning hemorrhage</td>
<td>↑ BP (PIH)</td>
</tr>
<tr>
<td></td>
<td>e) No warning hemorrhages</td>
</tr>
</tbody>
</table>
### Placenta previa vs. Concealed abruptio

<table>
<thead>
<tr>
<th>Placenta previa</th>
<th>Concealed abruptio</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General examination:</strong></td>
<td>condition of patient not proportional to blood loss</td>
</tr>
<tr>
<td>Condition of patient proportional to blood loss</td>
<td>uterus - hard, tender, tensed</td>
</tr>
<tr>
<td>P/A: uterus is soft, relaxed, non-tender</td>
<td>FHS - not easily heard</td>
</tr>
<tr>
<td>FHS - easily heard</td>
<td>Fetal parts- not easily felt</td>
</tr>
<tr>
<td>Fetal parts- easily felt</td>
<td>Ht.of uterus &gt; POG</td>
</tr>
<tr>
<td>Height of uterus = period of gestation (POG)</td>
<td>Fetal distress / Fetal death</td>
</tr>
<tr>
<td>FHS ⊕</td>
<td></td>
</tr>
<tr>
<td>P/vi: Contra-indicated in APH</td>
<td></td>
</tr>
<tr>
<td>contraindicated in placenta previa</td>
<td></td>
</tr>
<tr>
<td><strong>IOC (for APH):</strong> Transabdominal USG (screening to differentiate between placenta previa and abruptio)</td>
<td></td>
</tr>
<tr>
<td>IOC for previa: TVS (probe does not cross internal OS)</td>
<td>IOC for abruptio</td>
</tr>
<tr>
<td>* malpresentations more common in transverse lie &gt; breech</td>
<td>Clinical diagnosis &gt; TRS</td>
</tr>
</tbody>
</table>

### Management of abruptio placenta

00:48:50

* Never wait and watch (↑ risk of DIC)*
* No tocolytics*
* Management: Resuscitation of mother + Immediate termination of pregnancy*

Vaginal delivery in abruptio:
1) Misoprost contra - indicated
   - Can cause tachysystole (>5 contractions in 10 minutes)
2) Oxytocin can be used to augment labour
3) Artificial rupture of membranes can be done
If a patient of abruptio develops DIC

First correct DIC

Followed by vaginal delivery

### Management of placenta previa

- **Terminate pregnancy Immediately**
  - Active management
    - In any condition where mother's life is at risk
      - a) Hemodynamically unstable patient
      - b) Continuous bleeding
      - c) Gestational age ≥37 wks
      - d) Fetal distress
      - e) On USG - congenital anomaly incompatible with life

- **Continue the pregnancy**
  - Expectant management
    - Aim: To allow fetus to attain lung maturity
    - Condition: Only if mother's life is not at risk
      - a) Hemodynamically stable patient
      - b) Bleeding has stopped
      - c) Gestation ≤36 weeks
      - d) No fetal distress.
      - e) On USG -
        - Normal fetus or congenital anomaly
        - Compatible with life

### McAfee and Johnson regime

Expectant management of placenta previa

1. Admit the patient
2. Arrange for blood
3. Rh negative female - Anti D (300mcg)
4. Inj.corticosteroid (to attain lung maturity)
5. Tocolytics (if contractions)
   - Best - Nifedipine
Cervical cerclage: Not a component of expectant management

Expectant management

- Carry till 37wks
  - if:
    - Fetal distress
    - Bleeding episode
  - Terminate immediately

Mode of delivery:

- Minor degrees
- Vaginal delivery
  - a) Major degrees of placenta previa
  - b) Fetal distress
  - c) Severe bleeding
    - (irrespective of type of previa)
    - Cesarean section

To decide mode of delivery in placenta previa:

- Assess type of previa
  - USG
    - Double set-up examination
    - Do a P/V examination in OT, after full preparation for a cesarean section

DIC

- Consumptive coagulopathy (all clotting factors are consumed)

Obstetric causes of DIC:

- Abruptio placenta (mA)
- IUD of fetus
- Amniotic fluid embolism
- Septic abortion
- Severe pre-eclampsia / Eclampsia / HELLP syndrome
Diagnosis:
  S. fibrinogen (clotting factor 1): ↓
  Fibrin degradation product (FDP): ↑
  D- dimer: ↑

Management of DIC:
  Blood transfusion
  Cryoprecipitate / FFP
  Platelet transfusion
  Heparin +/-

No role in DIC:
  • EACA (epsilon - amino caproic acid)
  • Tranexamic acid

Recombinant factor VIII A: role in DIC uncertain
TWIN PREGNANCY – TYPES AND ETIOLOGIES

**Twin pregnancy – types**

- **Monozygotic (1/3 – 30%)**
  - Single sperm fertilizes ova.
  - Single zygote
  - Divides into two
    - Always same sex
    - A/a/A identical twins
  - Same phenotype
    - Same blood group
    - Same HLA typing
  - Different finger prints
  - Incidence – constant
    - Throughout the world
      - 1 in 250 pregnancies
      - 4 in 100 pregnancies
    - Due to Assisted reproductive techniques (ART) / In vitro fertilization (IVF)
      - Incidence is ↑

- **Dizygotic (2/3 – 70%)**
  - Two ova fertilized by two different sperms
  - Two zygotes formed
  - Sex may be same / different
  - Incidence – varies from country to country
    - Maximum in Nigeria – 1 in 20 pregnancies
    - Least in Japan – 1 in 200 pregnancies
    - In India – 1 in 80 pregnancies

**Hellin’s rule for incidence in India**

- In India,
  - Incidence of twins – 1 in 80 pregnancy
- Incidence of Triplets – 1 in (80)^3 pregnancy
• Incidence of quadruplets - 1 in (50)^3 pregnancy

• In dizygotic twins a ova fertilized by a sperms
  ↓
  In the same cycle by a different act of coitus
  ↓
  Superfecundation
  ↓
  Seen in humans
  ↓
  Not seen in humans

• Theoretically superfetation is possible in humans
  ↓
  Until uterus is not obliterated - 14-16 weeks

Factors that affect dizygotic twinning

1) Race
2) Maternal family history of twinning
3) Maternal age > 35 years
4) Maternal parity > 4
5) Use of drug like Clomiphene citrate - 5-8%
   Or
   Procedures like ART/IVF

6) Maternal obesity

• Dizygotic twins - Develop from two different zygotes
  ↓
  Zygotes develop their own chorion and amnion
  ↓
  Dichorionic diamniotic (DCDA)

• So m.c type of twin pregnancy - Dizygotic twins / Dichorionic diamniotic twins

Zygosity and chorionicity

• Zygosity - Type of conception
• Chorionicity - Type of placentation
• Prognosis in twins - Depends on chorionicity & not zygosity
- Monochorionic twins - Bad prognosis
- Dichorionic twin - Good prognosis
- Dizygotic twins - Always DCDA
  Always good prognosis

Monozygotic twins

- Number of chorions and amnions
  \[\downarrow\]
  Depends on the time of division
- Amnion - Formed on day-10
- Chorion - Formed on day - 8
- If the zygote in monozygotic twins divides
  \[\downarrow\]
  \(<4\) days
  \((within \ 72\ hours)\)
  \[\downarrow\]
  \(\text{Chorion}\)
  \(\text{Amnion}\)
  \[\downarrow\]
  DCDA
  \[\downarrow\]
  Good prognosis

  \[\downarrow\]
  \([4\ to \ 8\ days]\)
  \[\downarrow\]
  MCDA
  \(\text{(mono chorionic diamniotic)}\)
  \[\downarrow\]
  When they divide
  \[\downarrow\]
  Conjoint twins

  \[\downarrow\]
  \([8-12\ days]\)
  \[\downarrow\]
  MCMA
  \(\text{(mono chorionic mono amniotic)}\)
  \[\downarrow\]
  Some body part has formed

- MC type of monozygotic twins - MCDA
- MC type of dizygotic twins - DCDA

Chorionicity – investigation

- IOC - Trans vaginal ultrasound (TVS)
  \[\downarrow\]
  Time 11-14 weeks
  \[\downarrow\]
  In 1st trimester
**Difference between dichorionic diamniotic and monochorionic diamniotic**

<table>
<thead>
<tr>
<th>DCDA</th>
<th>MCDA</th>
</tr>
</thead>
</table>
| * Sex - Same or different  
100% dichorionic  
* Four layers in between the twins  
* membranes are thick ≥ 2mm  
* Twin peak sign / Lambda sign - is positive |  
Placenta insinuates between intetwin membrane | * Sex-same  
* Two layers in between the twins  
* Thin membranes < 2mm  
* Twin peak sign - is absent  
* T sign is present |
Ultrasound images in twins

- USG of twin peak sign / Lambda sign

  → DCDA
  - Twin peak
  - Lambda sign present

  → MCDA
  - T - sign present
  
  → MCDA

* In MCMA - T - sign is absent

Dichorionic diamniotic – Twins

* They have good prognosis
* 4 layers of membranes – membranes are thick
* Time of delivery – 38 weeks

DCDA
Monochorionic diamniotic and monochorionic monoamniotic – twins

Monochorionic diamniotic
• They have bad prognosis
  ↓
  Because of a complication
  ↓
  Twin-to-twin transfusion syndrome
• 2 layers of membrane (membranes are thin)
• Time of delivery – 34–37 weeks + 6 days
  Ideally – at 37 weeks

Monochorionic monoamniotic
• They have bad prognosis
  ↓
  Because the two fetuses are
  lying in the same amniotic fluid
  ↓
  ↑ risk of cord entanglement
• No layer of membranes in between the twins
• Best time to deliver – 32–34 weeks
  ↓
  Delivered only by cesarean section

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Conjoined twins

- mC variety - Paraphagus - 1st
  Thoracophagus - 2nd

- Least common variety
  Rachiphagus - vertebral columns joined - <1% cases
  Craniophagus - Heads joined - 5% cases

- Worst prognosis - Craniophagus

On ultrasound - findings:
1) Heads of both fetuses lie at same level
2) 4 vessels in the cord seen
3) Relative position of the twins does not change
4) Extension of fetal spine

- They have single chorion single amnion
- Delivered only by cesarean section
TWIN PREGNANCY-COMPLICATIONS & MANAGEMENT

Twin pregnancy – maternal complications

maternal complications

- i) Anaemia
- ii) Polyhydramnios
- iii) Uterine overdistension
- iv) ↑ placental size
  - Preterm labour
  - PROM [Premature Rupture of membranes]
  - Cord prolapse
  - Abruption

uterine overdistension

- Preterm labor
- PROM symptoms

Respiratory difficulty

Edema

↑ Placental size

Placenta previa

↑ PIH [pregnancy induced hypertension]

↑ Hormone synthesis

↑ HPL [human placental lactogen]

↑ Insulin resistance

↑ GDM [Gestational Diabetes mellitus]

↑ HCG [human chorionic gonadotropin]

↓ Hyperemesis gravidarum

↑ Osm
Twin pregnancy – Fetal complications

1. Complication which are never seen in twin pregnancy
   • Macrosomia
   • Post-term pregnancy

2. All complications are related to chorionicity & amnionicity except
   • Pre-term labor
   • IUGR [Intra-uterine Growth Retardation]
   which are related to number of fetus

   - In multi-fetal pregnancy:
     Do fetal reduction
     ↓
   DOC – Intracardiac injection of KCl
   - At 10-13 weeks of pregnancy

3. Most common complication of twin pregnancy → Preterm labor

   Prevention of preterm labor

   ↓

   methods adopted
   methods not adopted

   a. Frequent antenatal visits
   b. Frequent USG [ultrasound]
      - Monochorionic: every 6 weeks
      - Dichorionic: every 6 weeks
   c. Limited physical activity

   Advice
   • No progesterone
   • No tocolytics
   • No circlage
   • No bed rest

4. All complications of twin pregnancy are more in
   • Monochorionic > Dichorionic twins
   • Monozygotic > Dizygotic twins

   Except
   Cumulative risk of chromosomal anomaly

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Fetal complications – Chromosomal anomaly

1. The cumulative risk of chromosomal anomaly in
   * Dichorionic twins > monochorionic twins
   * Dizygotic > monozygotic

   a. Monozygotic
      
      ![Diagram showing monozygotic twinning with risk of chromosomal anomaly]

   b. Dizygotic
      
      ![Diagram showing dizygotic twinning with risk of chromosomal anomaly]

   ∴ Risk of chromosomal anomaly = 2x%

2. Trisomy -a1
   a. Monozygotic
      
      ![Diagram showing trisomy in monozygotic twinning]

   b. Dizygotic
      
      ![Diagram showing trisomy in dizygotic twinning]

   - Test for aneuploidy
   sample from 1 of the twins is sufficient

Complications of monochorionic twins

1. Vascular anastomosis between twins
   - Incase of deep anastomosis ⇒ TTTS [Twin to Twin Transfusion Syndrome]

2. Acardiac twin

3. Twin Anaemia Polycythaemia Sequence [TAPS]
4. Selective IUGR
5. Congenital malformations
6. Specific complication

monochorionic monoamniotic $\rightarrow$ Cord entanglement
  \[ \text{MCMA} \]
  Vascular Anastomosis
  
  Superficial
  1. more common variety
  2. most common type: artery-to-artery anastomosis
  3. Bidirectional flow [Based on pressure]

  $A \xrightarrow{\Downarrow} B$
  ↓
  • No permanent donor
  • No permanent recipient
  ↓
  $\therefore$ No TTTS
  iv) mostly seen in MCMA twins
  ↓
  $\therefore$ TTTS not seen

Deep
  i) most common type:
  1. Artery-to-vein anastomosis
  2. occurs at the level of capillary bed
  3. Unidirectional flow

  $A \rightarrow B$
  ↓
  Permanent donor Permanent recipient
  ↓
  $\therefore$ TTTS seen
  iv) mostly seen MCDA
  [monochorionic diamniotic] twins
  ↓
  $\therefore$ TTTS more common

Twin to Twin Transfusion Syndrome [TTTS]

* Seen in MCDA twins
* Deep artery of twin A $\rightarrow$ Deep vein of twin B

$A \xrightarrow{\text{Donor}} B \xleftarrow{\text{Recipient}}$

1. Anaemia
   ↓
2. Findings
   ↓
   1. Polycythemia
      (Thrombosis)
      $\therefore$ Sinusoidal pattern of FHR
      [seen in donor twin, vasa previa, Rh incompatibility]
      $\uparrow$

* Peak systolic velocity of middle cerebral artery
- Hemoglobin difference ≥ 5g/dl in both twins ⇒ TAPS

TAPS

As a part of TTS

Oligo and polyhydramnios in the twins +

As an isolated feature [≥ 26 weeks]

No oligo and polyhydramnios in the twins

- Peak systolic velocity of middle cerebral artery

Donor Recipient twin
>15 mom <1 mom

a. Donor a. Recipient twin
growth growth

- Discordant growth (Different in growth)

Difference in

- Body weight of fetus ≥ 20%
  (with larger twin being taken as index)
- Abdominal circumference in USG ≥ 20 mm
- Biparietal diameter on USG ≥ 6 mm

3. Oligohydramnios

[ ↓ Blood flow
  ↓ Renal blood flow
  ↓ GFR
  ↓ Urine output]

- Tops ⇒ Twin oligo poly sequence
  i.e. If 1 twin has oligohydramnios, the other twin has polyhydramnios
According to ACOG,

- Definitive features on USG diagnose TTTS are
  - mCDA
  - TOPS

2. ↑ risk of renal failure
   [Renin Angiotensin System is activated]

3. ↑ risk of congestive heart failure
   [Cyclic ANP is activated]

Staging of twin to twin transfusion syndrome

A. Quintero staging - (USG based)
   1. Twin-A: oligo, twin B: polyhydramnios + Bladder of Donor twin - visible on USG
   2. Bladder of donor twin - not visible
   3. Doppler changes
   4. Hydrops in any of the twins
   5. Death of any of the twin

B. CHOP score - For cardiac evaluation of recipient twin in TTTS

Management of TTTS:
- Definitive/Best ⇒ Fetoscopic laser ablation of anastomosis
  - Alternative ⇒ Serial amniocentesis

Twin Reversed Arterial Perfusion (TRAP)

- A → B
  - Normal Acardiac Twin
    - Lacks heart

- Anastomosis - Artery to artery & vein to vein
  - (significant role)
- Pathology of TRAP
  The umbilical artery [carry deoxygenated blood of twin A]
  ↓
  Of twin A
  ↓
  Takes blood to Twin B [acardiac]
  ↓
  Due to gravity
  - Blood reaches the lower limbs of Twin B
    ↓ lower limbs are formed
  - But, since twin B is acardiac – rest of body is not formed

- Varieties of TRAP
  ↓
  Acardius accephalus
  [only lower limbs are formed]
  ↓
  Acardius myelacephalus
  [lower limbs + some part of head]
  ↓
  Acardius amorphus
  [no part of body formed]

Donor twin has – ↑ chance of heart failure
- ↑ mortality

Management of TRAP
1. Volume of blood in acardiac twin – < 50%
   ↓
   Wait and watch, deliver at 37 weeks
2. Volume of blood in acardiac twin – > 50%
   ↓
   Radio frequency ablation
IUD in twin pregnancy

- more common in monochorionic twin
- more dangerous in monochorionic twin (due to anastomosis)

Neurological complications and death
- Preterm labor of the surviving twin in both monochorionic and dichorionic twin
- Aim - To carry pregnancy
  - Atleast till 34 weeks
  - Best upto 37 weeks

Case scenario
Twin pregnancy, in which 1 fetus dies at 32 weeks

management
- monitor the surviving twin
- Carry pregnancy atleast till 34 weeks
Delivery of twins

- most common presentation - both twins vertex
- 2nd most common - Twin A vertex, Twin B: breech

mode of delivery (depends on 1st twin)

\[
\begin{align*}
\text{1st twin - vertex} & \quad \downarrow \\
& \quad \text{vaginal delivery} \\
\text{1st twin - breech/transverse lie} & \quad \downarrow \\
& \quad \text{Cesarean section}
\end{align*}
\]

- 1st twin - breech, 2nd twin - vertex
  \[
  \begin{align*}
  \text{Cannot perform vaginal delivery due to complications of interlocking of twins}
  \end{align*}
  \]

1st twin vertex

\[
\begin{align*}
\text{2nd vertex} & \quad \downarrow \\
& \quad \text{1st twin - vaginal delivery} \\
& \quad \text{Methyl ergometrine is contraindicated after delivery of 1st twin due to tetanic contractions} \\
& \quad \text{Oxytocin can be given}
\end{align*}
\]

- 1st twin - vaginal delivery
- 2nd twin - assisted breech delivery

1st twin breech

- 1st twin - vaginal delivery
- 2nd twin - assisted breech delivery
- Take mother to OT
  \[
  \begin{align*}
  \text{Give general anaesthesia [GA]} & \quad \downarrow \\
  \text{Internal podalic version} & \quad \downarrow \\
  \text{Breech extraction}
  \end{align*}
  \]
### Internal podalic version vs external cephalic version

<table>
<thead>
<tr>
<th>Internal podalic</th>
<th>External cephalic</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Performed in OT</td>
<td>• Performed in OPD</td>
</tr>
<tr>
<td>• under GA</td>
<td>• No anaesthesia</td>
</tr>
<tr>
<td>• Risk of uterine rupture</td>
<td>• Fetal distress</td>
</tr>
<tr>
<td>• Indications</td>
<td>• Indications</td>
</tr>
<tr>
<td>- a&lt;br&gt;rd twin in transverse lie</td>
<td>• Single pregnancy</td>
</tr>
<tr>
<td>[at the time of labor]</td>
<td></td>
</tr>
<tr>
<td>• Absolute contraindication</td>
<td>• Relative contraindication</td>
</tr>
<tr>
<td>- Previous cesarean section</td>
<td>- Previous cesarean section</td>
</tr>
</tbody>
</table>

- Breech
- Transverse lie
- To make it cephalic
- Done antenatally
- ≥ 36 weeks
RH ISOIMMUNIZATION: MECHANISM AND COMPLICATIONS

Rh antigens

- c, C, D (most important), E, e

D antigen

- Present
  - Rh +ve

- Absent
  - Rh -ve

* Located on short arm of chromosome I
* Discovered by Landsteiner and Weiner

Rh negative pregnancy

1. mother : Rh -ve, Fetus : Rh +ve } Complications ⊕

a. mother : Rh -ve, Fetus : Rh -ve } No complications

To find fetal Rh status

If Husband - Rh -ve, if Husband : Rh +ve
wife - Rh -ve, and wife : Rh -ve
Fetus - Rh -ve (100%), fetus can be Rh +ve

1st investigation in an Rh -ve pregnant female

Rh status of husband
Pathogenesis of Rh negative pregnancy

- First pregnancy safe (if IgG develops only 6 months after exposure)
- Next Pregnancy:

  - Mother: Rh -ve
  - Placenta: Enter maternal circulation
  - Fetus: Rh +ve

  - Rh - antigen → Stimulate maternal immune system
  - Rh - antibody formed
    - (Anti-D)
    - IgM type (does not cross placenta)
    - IgG (can cross placenta)

  - Hemolysis of fetal RBCs

Antibody formed in mother → Cause hemolysis in fetus
:: Known as Rh immunization reaction

Fetal manifestations in Rh negative pregnancy

- Hemolysis in fetus

  - Fetal anemia
  - Sinusoidal heart rate pattern
  - Peak systolic velocity ↑ in MCA

  - Collection of fluid in 3rd space (pericardial / pleural effusion, ascites)

  - ↑ Bilirubin
    - Jaundice
    - ≥ 20 mg/dl
    - Hemicencephalus
    - Erythrocytes ↑

    - Known as Erythroblastosis Fetalis
Hemolysis in fetus

↓

Placentomegaly

(↑ blood supply to fetus from placenta)

↓

PIH in mother

Fetal manifestations:
1. Fetal anemia
2. Icterus gravidarum
3. Hydrops fetalis is (Grave / Worst prognosis)

Hydrops fetalis

00:20:17

Diagnosis:
- Fluid in ≥ 2 body cavities (any of the following):
  - Pleural effusion
  - Pericardial effusion
  - Ascites
  - Skin edema → manifests as scalp edema
    ↓
    On use, halo ☺
    known as Buddha sign

Features of hydrops fetalis (not diagnostic criteria)
1. Polyhydramnios
2. Placentomegaly

Fetus → Skin edema. Scalp edema → Bloating appearance → Mirror syndrome
Mother → PIH → Edema → Polyhydramnios → Bloating appearance

Hydrops fetalis

↓

Due to Rh negative pregnancy
↓

Immune hydrops fetalis

Due to any other cause
↓

Non-immune hydrops fetalis (NIHF)
Causes of NIHF:
1. Parvovirus infection
2. Congenital heart blocks
3. α Thalassemia
4. GI disorders: volvulus
5. Renal disorders: polycystic kidney disease
6. Chromosomal anomalies
7. Cystic hygroma
8. Twin to Twin Transfusion syndrome
PRETERM LABOR AND PROM

Preterm labor

- Preterm labor = < 37 weeks
- Before 37 weeks of gestation surfactants are not yet completely synthesized
  ↓
  Lungs are not mature

Surfactants:
- These are greasy material (Saponifying agents) synthesized by type II pneumocytes
  ↓
  Help in smoother deflation of lungs
  ↓
  If it is absent
  ↓
  Respiratory distress syndrome

- 2 components:
  1. Phosphatidyl choline - 80%
     (Maternal serum, fetal serum, amniotic fluid)
  2. Phosphatidyl glycerol - 15%
     (Present only in amniotic fluid)

- Production:
  * Begins - 30 weeks
  * Appears in amniotic fluid - 28 weeks of pregnancy

Lung maturity test:

1) mc done test: Lecithin / Sphingomyelin ratio in amniotic fluid

   ↓
   ↓
   ≥ a  < a

   Lungs mature  Lungs not mature
1. Best test: presence of phosphatidyl glycerol in amniotic fluid

- Present
- Absent
- Lungs mature
- Immature lungs

Best test for assessing fetal lung maturity in
Diabetic mother → Phosphatidyl glycerol

3. Bed side test: Shake/Bubble test
   - If bubbles are present
     - Mature lungs

4. Nile blue sulphate test / Skin test:
   - Amniotic fluid on centrifuging
     - Fetal skin cells
     - Add reagent - Nile blue sulphate dye
     - Look under microscope
     - If fetal skin cells have fat → Orange appearance
       - ≥ 50% → Lungs mature

5. TDX FLm test: Fluorescence polarization test
   - Quantitative test which assess ratio of surfactant and albumin in uncentrifuged amniotic fluid
     - If surfactant: Albumin ≥ 55mg/grams
       "Mature lungs"

6. Lamellar body count
Drugs for hastening lung maturity

**Corticosteroids**

- **Worldwide**
  - Betamethasone
  - Dexamethasone
  - **Dose:** 2 injections, 13 mg each 24 hours apart
  - **Route:** IM

- **India**
  - Dexamethasone
  - **Dose:** 1 injection, 6 mg each 12 hours apart
  - **Route:** IM

- **Contraindications for corticosteroids:** Chorioamnionitis
- **Effects** begins after **24hrs** of last injections maximum effect: 2-7 days
- **Repetitive doses of corticosteroids are not given**
  - It can lead to cerebral palsy in fetus
- **Maximum of 1 repeat dose can be given**
- **Lungs of fetus starts maturing by 34 weeks and completely mature by 37 weeks so**

  - **If preterm labour <34 weeks:**
    - Corticosteroids given
  - **If preterm labour ≥34 weeks:**
    - Corticosteroids are not given

  - **ACOG = Recommended 2017**
  - **Should give corticosteroid**
  - **Not yet followed**

**Preterm labour**

- **Labour begins at < 37 weeks**
- **mc cause - Idiopathic**
- **Causes:**
  1. Infections - Asymptomatic bacteriuria, UTI bacterial vaginosis
a. Over distension of uterus (twin pregnancy, Polyhydramnios)  
   - mc risk factor: Previous history of preterm labour

**Prevention of preterm labour:**
1. Quit smoking
2. Progesterone (PCC)
3. Cervical Cerclage

**Diagnosis:**
- Contractions:
  - $\geq 4$ contractions in 20mins  
    Or  
  - $\geq 8$ contractions in 60mins  
    (+) Any of following
  1. Dilatation of cervix $\geq 3$cms
  2. Length of cervix $\leq 2$cm
  3. Length of cervix between 2-3cms  
     (+)  
     Fetal fibronectin protein should be present
  - length of cervix to predict PTL- 2.5cms

**Fetal fibronectin protein**

- Extracellular matrix protein which is present in interface between the chorion and decidua.
- If at any reason this interface is broken
  \[ \downarrow \]
  Fibronection Protein appears in Cervicovaginal discharge
  $\geq 50$ ng/ml - then test is positive
  \[ \downarrow \]
  Can be due to
  1) uterine contractions
  2) Infections
  3) Abruptio placenta

- FFN protein can be present in Cervicovaginal discharge:
  1) Preterm lab our
  2) PROM
Shape changes in cervix at time of labour:

Normally: 'T' shaped

\[ \downarrow \]

Correlation between the cervical length and changes of the internal cervical os

As cervix dilates

\{ 'Y' shaped, 'V' shaped, 'U' shaped \}

Problems with PTL

Lungs not mature

\[ \downarrow \]

Brain not developed

\[ \text{DOA: (1) Corticosteroid} \]

- Need at least 24hrs to act

\[ \downarrow \]

- Give short term tocolytics

For neuro protection

\[ \text{DOA: (5) mgSO₄, (at < 38 weeks)} \]

Management of preterm labor

\[ \geq 34 \text{ weeks up till 36 weeks + 6 days} \]

\[ \downarrow \]

\[ \text{(1) Send rectal/vaginal swab for group B streptococci} \]

\[ \text{(2) Do not give corticosteroids} \]

\[ \text{(3) Wait and watch} \]

\[ < 34 \text{ weeks} \]

\[ \downarrow \]

\[ \text{(1) Corticosteroids} + \]

\[ \text{(2) Short term tocolytics} + \]

\[ \text{(3) If PTL < 28 weeks} \]

\[ \text{mGnSO₄} \]
mode of delivery:

Earlier:

If PTL < 34 weeks → Cephalic → Cesarean section

 if PTL < 34 weeks → Breech

Now:

If PTL < 34 weeks → Cephalic → Vaginal delivery

if PTL < 34 weeks → Breech → Cesarean section

No role in PTL:

1) Antibiotics
   - unless membranes are ruptured
   - documented infection

2) Progesterone (not a tocolytic)

Tocolytics

- Best tocolytic - Nifedipine
- Best tocolytic in heart disease - Atosiban (oxytocin antagonist)

Dose of Nifedipine: 20mg oral stat

↓ after 4 hrs (every 4 hrs)

10mg oral till contractions subside

↓

10mg 8 hourly x 1 week

- Tocolytic with maximum fetal side effect: indomethacin
- Tocolytic with maximum maternal side effect

"β agonist"

Drugs used

1. Salbutamol
2. Ritodrine
3. Isoxsuprine
4. Terbutaline

Side effects

mc - Tremors
- Hyperglycemia
- Hypokalemia
- Pulmonary edema
- Contraindicated in diabetes

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- $\text{mgSO}_4$ acts as tocolytic at 9-10 meq / L
  \[\downarrow\]
  Toxic
  \[\downarrow\]
  Hence not used as tocolytic

- Drugs with tocolytic effect but not used:
  1. Alcohol
  2. Halothane
  3. Diazoxide

**Premature Rupture Of Membranes (PROM)**

- Normally membranes rupture at end of 1st stage of labour, when cervix is fully dilated
- In PROM membranes rupture before onset of labour
- In preterm PROM membranes rupture before 37 weeks of pregnancy
- Artificial rupture of membranes - Amniotomy
  - Instrument: Kocker’s forceps
  - Indication:
    1) To augment labour (accelerate the process of labour)
    2) In Fetal distress during labour

**Contraindications**

1. Maternal HIV infections
2. Maternal genital herpes
3. IUD of fetus
4. Polyhydramnios
   \[\downarrow\]
   ARM- Contrainindicated, controlled ruptures to be performed

**Management**

- Per vaginal examination is contraindicated
- Sterile per speculum examination (can be done)
Test done for diagnosing PROM

- Nitrazine test
  - Principle: Amniotic fluid
    - Alkaline pH (7.2-7.4)
    - Vaginal discharge
    - Acidic pH (3.5)
  - Introduce nitrazine paper
    - Blue
      - pH - alkaline in amniotic fluid
        - PROM
    - Yellow
      - pH - acidic
        - Vaginal discharge

- Fern test
  - Based on principle:
    1. Cervical discharge of pregnancy
       - allowed to dry
       - beaded appearance
    2. If amniotic fluid is allowed to dry
       - Fern like appearance

Other tests:
- Fibronectin protein test:
  - If fibronectin protein is present in cervicovaginal discharge before 37 weeks of pregnancy
    - Preterm labour or PROM

- Best investigation: USG
  - Amniotic fluid decreases
Risk of PROM/PPROM

① Lungs not matured
② Brain is not properly developed
③ Increase chances of infection

DOC: Corticosteroids
(+)
Short term tocolytics

DOC: mgSO4
(<28 weeks)
Antibiotics

Management of PPROM

≥ 34 weeks to 36 weeks + 6 days

① Corticosteroids not given (ACOG = Corticosteroids should be given)
② Antibiotics given
③ TOL

<34 weeks

① Corticosteroids
② Short term tocolytics
③ mgSO4 (if PROM <28 weeks)
④ Antibiotics
POST TERM PREGNANCY

Post Term Pregnancy: Any pregnancy which goes beyond 42 weeks

* Naegle's formula →
  * Expected date of delivery (EDD) =
  * 1º day of last menstrual period + 9 months and 7 days

if 1º day of LMP = 1º September 2018
  EDD = 8º June 2019

* Only 4% ♀ deliver on exact EDD
* 50% ♀ deliver → 1 week before or 1 week after EDD

* If no delivery - even after 1 week of EDD = After 42 weeks

  Induce labor

* Pregnancy not to be continued beyond 42 weeks

Complications: Post term pregnancy

* Oligohydramnios - meconium aspiration syndrome
* Macrosomia - Fat around shoulder & trunk
  * Shoulder dystocia
  * Intracranial haemorrhage
  * ↑ chance of Caesarean section

* Placental aging - Placental insufficiency

  Fetal distress

* 1º step on approach to post term pregnancy = Review her menstrual history

  Fetal ACTH

  * Fetal adrenal gland → DHEA → Androgens → Placenta Synthesize estrogen

  Signal uterus to start contractions

*: For labor to initiate → intact fetal adrenal gland is required
In Anencephaly: Fetal Adrenal glands - Absent / Hypoplastic

\[\downarrow\]

Post Dated Pregnancy.
MATERNAL PELVIS: NORMAL AND CONTRACTED

Pelvis

- Composed of 4 bones:
  - Sacrum
  - Coccyx
  - 2 innominate bones

- Each innominate bone is formed by fusion of 3 bones:
  - Ileum
  - Ischium
  - Pubis

- Pelvis is divided anatomically into false pelvis and true pelvis by pelvic brim

The boundaries of pelvic brim or inlet (from anterior to posterior) are:
- upper border of pubic symphysis → Pubic crest → Pubic tubercle → upper border of superior pubic rami → Ilipectineal eminence → ilipectineal lines → Sacro iliac joint → Ala of sacral bone → Sacral promontory
- **False pelvis**
  - Lies above pelvic brim
  - No obstetric significance

- **True pelvis**:
  - Lies below pelvic brim
  - Important role in child birth and delivery

- True pelvis can be divided into:
  - **Pelvic inlet**: Lies at level of pelvic brim
  - **Pelvic cavity / midpelvis**: Lies at level of ischial spine
  - **Pelvic outlet**: Lies at level of ischial tuberosity

### Major diameters of pelvis

<table>
<thead>
<tr>
<th></th>
<th>Inlet</th>
<th>Mid pelvis</th>
<th>Outlet</th>
</tr>
</thead>
</table>
| **Antero-posterior (AP) Diameter** | True conjugate
  → Distance between upper border of pubic symphysis and sacral promontory (SP) = 11 cm
  Obstetric conjugate
  → Distance between middle of pubic symphysis and SP = 10-10.5 cm
  Diagonal conjugate
  → Distance between lower border of pubic symphysis and SP = 13 cm | Line joining lower border of pubic symphysis to the junction of S4-S5 vertebra. 12 cm (11.5 cm) | 11.5-13 cm ↓

At the time of labour |

<table>
<thead>
<tr>
<th><strong>Oblique Diameter</strong></th>
<th>Left oblique diameter - from left sacroiliac joint to right iliopectineal eminence - 12 cm</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Transverse Diameter</strong></td>
<td>13 cm</td>
<td>Bispinous diameter / interischial diameter 10 cm</td>
<td>Bituberos 11 cm</td>
</tr>
</tbody>
</table>
Important points related to diameters of pelvis

- Smallest AP diameter of pelvis → Obstetric conjugate
- Largest AP diameter of inlet → Diagonal conjugate
- Out of three AP diameters of pelvic inlet; only diagonal conjugate can be measured clinically
- Critical obstetric conjugate → 10 cm (i.e., if obstetric conjugate is less than 10 cm vaginal delivery is not possible)
- If diagonal conjugate is x cm in a female, then obstetrical conjugate will be x-1.5 or 2 cm
- Interspinous diameter
  - Shortest major diameter of pelvis
  - Most important diameter during delivery

Posterior sagittal diameter

- Posterior sagittal diameter of outlet

  - Distance from the tip of coccyx to the point at which transverse diameter and antero-posterior diameter bisect each other
  - ≥ 7 cm

- Posterior sagittal diameter of mid pelvis → 4.5 cms
- Posterior sagittal diameter of inlet → 4.0 cms

Angle of inclination—angle between plane of pelvic inlet with horizontal plane = 55°.
- High inclination—increase in no. of sacral bones (SS → S4). Fetus will take longer time to get engaged, therefore time of delivery also increases.
* Low inclination—decrease in no. of sacral bones (lumbarization of sacral bone), therefore faster engagement and easier delivery.

2. Subpubic angle—obtuse in females, acute in males

**Significance of ischial spine**

1. Station of fetal head

   ![Diagram of pelvic brim and ischial spine](image)

   a. Internal rotation of fetal head occurs at this level

   3. Deep transverse arrest occurs at this level

   4. Site for giving pudendal nerve block—sacrospinous ligament is pierced while giving block

   5. Levator Ani muscle is attached here

   6. Ring pessary in case of prolapse is inserted at this level

   * Time for pelvic assessment

     - Primigravida = 37 weeks
     - Multigravida = At the onset of labor

**Contracted pelvis**

* Any of major diameter of pelvis ↓ by ≥ 1 cm
Contracted at

- Inlet
  - Obstetric conjugate < 10 cm
  - Clinically: Diagonal conjugate minus 1.5 cm to 2 cm

- Midpelvis
  - Interschial diameter < 8 cm
  - Clinically: Both ischial spines can be touched with 2 fingers of same hand

- Outlet
  - Inter tuberous diameter < 8 cm
  - Clinically: If 4 knuckles cannot be inserted between ischial tuberosities

Varieties of contracted pelvis:

- Naegles pelvis
- Robert pelvis

  → Only 1 ala of sacral bone is present
  → Both ala of sacral bone are absent

management: Cesarean section

Cephalo – Pelvic Disproportion (CPD)

- Fetus is too large or pelvis is too small for this delivery
- CPD can also occur at level of inlet/cavity/outlet

management:
- mild CPD at level of inlet → Trial of labour
- moderate / severe degree of disproportion at: cesarean level of inlet
- Disproportion at level of cavity / outlet: cesarean

- Trial of labour
  - Obstetrician is aware that patient has mild CPD at level of inlet
  - Vaginal delivery is being tried

- Trial of scar
  - Patient is a previous c-section, in whom cesarean was done due to a non-recurring cause like fetal distress
  - Vaginal delivery is being tried in the next pregnancy in the patient
  - Trial of scar = vaginal birth after cesarean (VBAC)

Clinical condition: A GaPi female with previous vaginal delivery has mild CPD at level of inlet

management: Trial of labour
Clinical condition 2: A GaPi female with previous cesarean section due to fetal distress, has normal pelvis and no fetal distress

management: VBAC / Trial of scar

Clinical condition 3: A GaPi female with previous C-section due to fetal distress, has mild CPD at level of inlet and no fetal distress

management: Cesarean section

Trial of labour is contraindicated in previous cesarean patients

Normal varieties of pelvis

- On the basis of shape of the inlet, the pelvis can be of 4 types
- Caldwell and Mokhley classification:
  - Gynecoid 50% → Female type pelvis - most suitable for vaginal delivery
  - Anthropoid 25%
  - Android 20% → male type pelvis - least suitable vaginal delivery
  - Platybelloid 5% → Flat bowl like pelvis

Diagram:

<table>
<thead>
<tr>
<th>Type</th>
<th>Gynecoid</th>
<th>Android</th>
<th>Anthropoid</th>
<th>Platybelloid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shape of inlet</td>
<td>transverse</td>
<td>heart shaped</td>
<td>AP oval</td>
<td>bowl shaped</td>
</tr>
<tr>
<td>Side walls</td>
<td>straight (broad)</td>
<td>convergent</td>
<td>straight (narrow)</td>
<td>divergent</td>
</tr>
<tr>
<td>Sub pubic angle</td>
<td>Obtuse</td>
<td>Acute</td>
<td>Prominent</td>
<td></td>
</tr>
</tbody>
</table>
FETAL SKULL

Diameters of fetal skull

Always AP diameters are greater than transverse diameters

AP diameter

- Mentovertebral [14 cm]
- Submentovertebral [11.5 cm]
- Occipito Frontal [11.5 cm]
  - Longest AP diameter
  - 2nd longest
  - Occipito posterior
  - Brow presentation
  - Face presentation
  - Always C-section

Transverse Diameter: Mnemonic

Miss - Bimastoid Diameter = 7.5 cm (shortest)
Tina - Bitemporal Diameter = 8 cm
So - Super subparietal diameter = 8.5 cm
Pretty - Biparietal diameter = 9.5 cm (largest)
### Parts of fetal skull

<table>
<thead>
<tr>
<th>Part</th>
<th>Definition</th>
<th>Seen in</th>
<th>Engaging diameter</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Vertex</em></td>
<td>Part between anterior fontanelle &amp; posterior fontanelle</td>
<td>Head of baby is fully flexed</td>
<td>Sub occipito bregmatic (9.5 cm)</td>
</tr>
<tr>
<td><em>Brow</em></td>
<td>Part between anterior fontanelle on one side and root of nose &amp; supra orbital ridges on the other side</td>
<td>Head is deflexed</td>
<td>Mentovertical diameter (14 cm) C. section is mandatory</td>
</tr>
<tr>
<td><em>Face</em></td>
<td>Between root of nose &amp; supra orbital ridge on one side &amp; chin on the other side</td>
<td>Fully extended head</td>
<td>Submentovertical diameter (if mento posterior) Sub mento bregmatic (if mento anterior)</td>
</tr>
</tbody>
</table>

### Moulding

Alteration of shape of fetal skull during labor

Grades:

- Grade 1 - Skull bone of fetus touch each other
- Grade 2 - Skull bone overlap each other, but can be separated
- Grade 3 - Skull bone overlap & cannot be separated
Fetal scalp swelling

Caput succedaneum  Cephalohematoma

Scalp

Periosteum

Fetal skull

<table>
<thead>
<tr>
<th>Caput succedenum</th>
<th>Cephal hematoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Diffuse swelling above the periosteum</td>
<td>• Localized swelling below periosteum</td>
</tr>
<tr>
<td>• Cause: Head of fetus stay &gt; in one position for very long time during delivery</td>
<td>• Traumatic – especially instrumental delivery</td>
</tr>
<tr>
<td>• Pitting ☹</td>
<td>• Pitting ☹</td>
</tr>
<tr>
<td>• Cross the suture line</td>
<td>• Doesn’t cross suture line</td>
</tr>
<tr>
<td>• Not associated with # of underlying bone</td>
<td>• Associated with # of underlying bone</td>
</tr>
<tr>
<td>• Jaundice ☹</td>
<td>• Jaundice may be ☺</td>
</tr>
<tr>
<td>• Present at the time of birth &amp; disappears few hours after birth</td>
<td>• Doesn’t appear at birth &amp; disappear after few days</td>
</tr>
</tbody>
</table>
TERMINOLOGY RELATED TO LABOR

Lie

- Relationship between long axis of fetus & long axis of mother
  
  **Longitudinal lie:** MC lie
  - Long axis of mother & long axis of fetus - Coincide with each other

  **Oblique lie:**
  - Long axis of fetus make angle with long axis of mother
  - Do caesarean section

  **Transverse lie:**
  - Long axis of mother & long axis of the fetus are at 90° to each other
  - Always do caesarean section (if baby is alive / dead)
  - ↑ incidence of cord prolapse
  - ↑ incidence of hand prolapse

Unstable lie

- when lie of the fetus is not fixed by 37 weeks
- Causes:
  - Idiopathic (mc)
  - Polyhydramnios
  - Placenta previa
  - Abnormal shape of pelvis

- Oligohydramnios doesn’t lead to unstable lie
- Uterine malformation
- management - Caesarean Section

Presentation

- Part occupying lower part of uterus
- mc Presentation - Cephalic
- Apart from Cephalic, all the other presentations are malpresentations
- mc malpresentation - breech (buttocks)
Neglected Shoulder Presentation:
- Dead baby with shoulder presentation
- Management - C-Section

Presenting part
- Part of presentation directly lying over the internal os
- Well-flexed head - vertex (mo)
- Fully extended head - Face
- Partially extended - Brow → Always do C-section

Denominator
- Bony point of reference on presentation used to describe the position
  - Vertex - Occiput
  - Brow - Frontal bone
  - Face - mentum
  - Breech - Sacrum

Positions
- 0 Vaginal delivery - Position from 1 to 5
- Occipito posterior (mc malposition) - 6 to 8
- mc Position of fetus = LOT
- mc Position during labor = LOT
- mc Position during late labor = LOA
- mc Occipito anterior position = LOA
- mc Occipito posterior position = ROP
- mc Position in breech = LSA
- mc Position in Face = LMA

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NORMAL AND ABNORMAL LABOR

Normal labor and uterine contractions

- Three things are needed for normal labor
  ↓
  3-P's
  ↓
  Passenger  Passage-normal Pelvis  Push-uterine contractions

Uterine contractions

- Braxton hicks contractions - Painless and irregular contractions throughout the pregnancy

- During labor-the contractions are painful and lead to dilation of cervix
  ↓
  Uterine contractions start at the cornu (Pace maker)
  ↓
  Right cornu pacemaker > left
  Fundal predominance
  ↓
  Contraction are predominant over the fundus
  ↓
  Contraction spread
  ↓
  Through out uterus at 2 cm/sec
  ↓
  Depolarizing the whole organ within 15 secs

- Polarity of uterus - isthmus in non pregnant female is 0.5 cms, during pregnancy
  ↓
  Isthmus enlarges - Forms lower uterine segment
  ↓
  The length of lower uterine segment at labor - 10 cm
  ↓
  During labor- The upper uterine segment actively contracts, lower uterine segment dilates passively called as polarity of uterus
  ↓
  Allowing the baby to move out of the uterus out
And upper uterine segment - Contracts & dilate
Pushes the baby down

* Retraction of uterine muscles - During labor, when the upper uterine segment contracts and relaxes
  The muscles will not come back to its original length

The muscles get shortened as the contraction progresses
This is called retraction

Events and intrauterine pressure

<table>
<thead>
<tr>
<th>Events</th>
<th>Intrauterine pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Contraction begin at</td>
<td>10 mm Hg</td>
</tr>
<tr>
<td>* Contraction are painful</td>
<td>15 mm Hg</td>
</tr>
<tr>
<td>* Cervix dilates at</td>
<td>15 mm Hg</td>
</tr>
<tr>
<td>* Fundus cannot be intended at</td>
<td>40 mm Hg</td>
</tr>
<tr>
<td>* 1st stage of labor</td>
<td>40-50 mm Hg</td>
</tr>
<tr>
<td>* 2nd stage of labor</td>
<td>100-180 mm Hg</td>
</tr>
</tbody>
</table>

* Adequate uterine contractions

- Frequency
  - 3 contractions in 10 min
- Duration
  - Each contraction lasting for 45 sec
- Intensity
  - Intrauterine pressure of 65-75 mm or 220 montevideo unit
- Tachysystole - more than 5 contractions in 10 minutes
  ↓
  Due to ↑ pressure in contraction -
  ↓ volume of blood → To fetus
  ↓
  Can lead to fetal distress
  ↓
  If fetal distress → Hyperstimulation of uterus (term no longer used)

**Unit of uterine activity and prelabor**

**Unit of uterine activity**
- Units for measuring uterine contractions
  ↓
  mm of Hg montevideo unit (mv unit)

1 montevideo unit = intensity of uterine contraction × number of contractions in 10 minutes

  e.g. - 4 contractions in 10 minutes
  each lasting for = 35 secs
  generating = 50 mm Hg intrauterine pressure
  ↓
  $4 \times 50 = 200 \text{mv}$

**Prelabor**
- Before true labor - Stage of pre labor
  ↓
  Characterized by
  ↓
  False labor pain  Lightening  Softening of cervix
  ↓
  Ripening of cervix
Cervical ripening

- The changes which occur in cervix at ripening

- Collagen decreases
- Hyaluronic acid increases
- Dermatan sulphate decreases

- During induction of labor—cervical ripening is checked using Bishop's score

Uterus at different times of pregnancy

- 36 weeks: Xiphisternum
- 40 weeks/33 weeks: a/3 distance between umbilicus & xiphisterum
- 28 weeks: 1/3 distance between umbilicus & xiphisternum
- 24 weeks: Upper border of umbilicus
- 20 weeks: Lower border of umbilicus
- 16 weeks: Midway between umbilicus & pubic symphysis
- 12 weeks: Upper border of pubic symphysis

- Lightening: After 36 weeks, head of fetus goes inside pelvis
- The height of uterus↓ like at 32 weeks
- Respiratory discomfort is relieved
- Called welcome sign
### False labor pain and true labor pain

<table>
<thead>
<tr>
<th>False labor pain</th>
<th>True labor pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Constant / irregular</td>
<td>* Regular &amp; Rhythmic (Off &amp; On)</td>
</tr>
<tr>
<td>* Located in lower abdomen</td>
<td>* Located in lower abdomen but it is radiated to back and thigh</td>
</tr>
<tr>
<td>* Does not lead to dilation of cervix</td>
<td>* Leads to dilation of cervix</td>
</tr>
<tr>
<td>* Show is absent</td>
<td>* Show is present (show - blood mixed mucous discharge)</td>
</tr>
<tr>
<td>* On per vaginal examination ↓</td>
<td>* On per vaginal (PV) examination ↓</td>
</tr>
<tr>
<td>Bag of water absent (Fetal membranes+amniotic membranes)</td>
<td>Bag of water present</td>
</tr>
<tr>
<td>* Do not ↑ in intensity, frequency and duration</td>
<td>* They ↑ in intensity, frequency, duration with passage of time</td>
</tr>
<tr>
<td>* Clinically—Pain subsides on rest, sedation, enema</td>
<td>* Pain does not subside</td>
</tr>
<tr>
<td></td>
<td>* True labor pain mark the beginning of true labor</td>
</tr>
</tbody>
</table>

### Stages of labor

<table>
<thead>
<tr>
<th></th>
<th>1st stage</th>
<th>2nd stage</th>
<th>3rd stage</th>
<th>4th stage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>* Begins with onset of true labor</td>
<td>* Begins with full dilation of</td>
<td>* Begins with delivery of baby</td>
<td>* 1 hr observation period after</td>
</tr>
<tr>
<td></td>
<td>pains</td>
<td>cervix</td>
<td>9 ends with delivery of baby</td>
<td>delivery of placenta</td>
</tr>
<tr>
<td></td>
<td>* Ends with full dilation of cervix</td>
<td>* Ends with delivery of baby</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(10cm)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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The document contains information about the phases of labor. It is divided into sections that discuss the first stage of labor focusing on the latent and active phases. Below is the structured representation of the content:

### Phases

<table>
<thead>
<tr>
<th>Phases</th>
<th>1st stage</th>
<th>2nd stage</th>
<th>3rd stage</th>
<th>4th stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal dilation in nulliparous multiparous</td>
<td>Latent phase 12 hrs 8 hrs</td>
<td>1 hr 30 min</td>
<td>Passively -15-25 min, Active management = 5-10 min</td>
<td>1 hr</td>
</tr>
<tr>
<td>Prolonged Nulliparous Multiparous</td>
<td>20 hrs 14 hrs</td>
<td>2 hrs 1 hr (+1 hr each if epidural given)</td>
<td>More than 30 min Retained Placenta</td>
<td>Physiological chills are experienced by mother</td>
</tr>
</tbody>
</table>

### First stage of labor – latent and active phases

#### Latent
- Begins with true labor pain
- Ends with cervix 5 cm dilation
- Duration
  - Nulliparous 12 hrs (average 9.6 hrs)
  - Multiparous 8 hrs (average 5.6 hrs)
- Mainly concerned with cervical effacement and dilation
  - Cervix become short

#### Active
- Begins with cervical dilation ≥ 6 with regular contractions
- Normal minimum cervical dilation rate for
  - Nulliparous 1.2 cm/hr
  - Multiparous 1.5 cm/hr
- Minimum rate of dilation
  - 1 cm/hr
- Mainly concerned with cervical dilation and descent of fetal head
Abnormality of latent phases

Prolonged latent phase

- Definition
- Causes
- Management

- ≥ 20 hours in nulli
- ≥ 14 hours in multipara
- Earlier definition ≤ 8 hrs
- Excessive sedation or epidural analgesia
- Therapeutic rest - BEST
- D.O.C for ripening of cervix
- Poor cervical conditions (e.g., thick, uneffaced or undilated)
- False labour - mCC in multipara
- PGEA (Cinoprost gel)/ cerviprim gel

First stage of labor – abnormalities of active phase

Active phase – normal

- Dilation of cervix
- Descent of fetal head

- Nulliparous – 1cm/hr
- Multiparous – 1.5cm/hr

(According to WHO- minimum should be 1 cm/hr)
Abnormalities of active phase

Protracted Active Phase

Definition

If dilation or descent is happening at slower rate (less than normal rate)

Management

Rule out

Cephalopelvic disproportion (CPD)

If CPD is cause

Rx-cesarean section

Wait & watch

Rule out

Occipito posterior position

If it is the cause

Augment labor by rupture of membranes and starting oxytocin infusion

If the cause is neither of them

Induction of labor and augmentation of labor

Induction of labor

- Pregnant female not in labor

Initiate labor in her

Augmentation of labor

- Pregnant female already in labor

If progress of labor is slow

Accelerating the process of labor

* Done by prostaglandins

  i) PGE-2 = Dinoprost - BREST (cerviprime gel)

  ii) PGE-1 = misoprost

Contraindicated in previous cesarean section patient

* Two methods

  - Artificial rupture of membranes
  - Oxytocin

  PGE-2 released

  Augments labor
Contraindications for artificial rupture of membranes

- The membranes - normally, acts as a barrier between vagina and fetus
- Membranes - rupture normally at the end of 1st stage (after dilation of cervix dilation)
- Artificial rupture - membranes ruptured before full dilation
- Contraindication
  i) Maternal HIV
  ii) Active genital herpes infection
  iii) Intrauterine fetal death
  iv) In chronic polyhydramnios - Simple ARM causes sudden amniotic fluid gush
     ↓
     Causes sudden decompression of uterus
     ↓
     Can lead to detachment of placenta
     ↓
     Abruptio placenta
- So in polyhydramnios - Controlled ARM is done
Active phase arrest

Active phase arrest (cervix does not dilate)

\[ \downarrow \]

Cervix is \( \geq \) 6 cms dilated with membranes ruptured

\[ \downarrow \]

Adequate uterine contractions are present and no dilation for 4 hours

Adequate uterine contractions are not present, oxytocin given and no dilation for 6 hours

• Diagnosis of arrest, should only be made after membranes are ruptured
• Management - Cesarean section

Second stage of labor

• In second stage-fetus gets delivered
• Nulliparous - 1 hour
• Multiparous - 30 mins
• The second stage of labor - abnormalities

\[ \downarrow \]

Prolonged Arrest

• Second stage is said to be prolonged

\[ \downarrow \]

If without epidural

Nulliparous: 2 hrs
Multiparous: 1 hrs

If with epidural

Nulliparous: 3 hrs
Multiparous: 2 hrs

• Management of prolonged stage-2 labor

\[ \downarrow \]

If station \( \geq +2 \)

Forceps

If station \(< +2 \)

Cesarean section
- Arrest of 2nd stage - Prolonged second stage + 1 hr
  - Without epidural
    - Nulliparous: 3 hrs
    - Multiparous: 2 hrs
  - With epidural
    - Nulliparous: 4 hrs
    - Multiparous: 3 hrs

- Second stage arrest - A/H/A obstructed labor
  - Management - Cesarean section

**Obstructed labor**

- In obstructed labor
  - The mother is - Exhausted
    - Tachypnea
    - Acidotic breathing

- Per abdomen, the uterus in pregnancy - two segments
  - Upper uterine segment (UUS)
    - Actually contracts
    - Tonia contract
  - Lower uterine segment (LUS)
    - Passively dilates at the time of labor
    - Dilated and stretched out

A ring can be palpated between UUS and LUS - called Bandl's ring

- Fetal heart sound - Fetal distress / Absent
- P/V examination - Hot, dry vagina.
  - Bleeding present
  - Hematuria Present
  - Fetal head - Moulding
  - Caput can be felt
管理 - 原则

- 永远不要等待，永远不要观察
- 永远不要给予催产素
- 最佳管理 - 剖宫产 + 抗生素

并发症 - 阻塞性分娩

- 子宫破裂
- 膀胱阴道瘘（VVF）
- VVF 在发展中国家 - 阻塞性分娩

Banda's 环和 Schroeder's 环

<table>
<thead>
<tr>
<th>Banda's ring</th>
<th>Schroeder's ring</th>
</tr>
</thead>
<tbody>
<tr>
<td>被见于阻塞性分娩</td>
<td>被见于催产素使用不恰当</td>
</tr>
<tr>
<td>病理环</td>
<td>生理环</td>
</tr>
<tr>
<td>收缩环</td>
<td>收缩环</td>
</tr>
<tr>
<td>位于 UUS 和 LUS 交界处</td>
<td>位于 UUS 和 LUS 交界处</td>
</tr>
<tr>
<td>环向上移动（位置固定）</td>
<td>位置固定</td>
</tr>
<tr>
<td>可以通过腹部（P/A）但不能通过阴道（P/V）</td>
<td>不能通过P/A但可以通过P/V</td>
</tr>
<tr>
<td>管理 - 剖宫产</td>
<td>管理 - 大部分环将自动消失</td>
</tr>
</tbody>
</table>

- 如果没有
- 第一阶段 - 剖宫产
- (垂直切口在 LUS)
- 第二阶段 - 根据胎儿头的位置
- 剖腹产或引产

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Third stage of labor

- Normal duration
  - If passive management
    - 15-25 mins
  - If active management
    - 5-70 mins

- Prolonged 3rd stage of labor ≥ 30 min
  - Retained placenta

- Management for retained placenta
  - Oxytocin
  - If still not delivered
    - Adherent placenta
      - (placenta accreta, increta, percreta)
      - Hysterectomy
    - Non-adherent placenta
      - (detached from uterus but not delivered)
      - Manual removal under general anesthesia

Fourth stage of labor

- It is 1 hr observation period after delivery of placenta
- The mother experiences physiological chills
MECHANISM OF LABOR

- Mechanism of normal labor - manner in which the fetus adjusts itself to pass though the parturient canal with minimal difficulty

- 8 cardinal movements of the head in normal labor
  - Every - Engagement (syncitlc / Asyncitlc)
  - Decent - Descent
  - Female - Flexion
  - I - Internal rotation
  - Choose to - Crowning
  - Employ - Extension
  - Rises - Restitution
  - Extremely - External rotation

### Engagement

Normal vaginal delivery
  - Lie - longitudinal
  - Presentation - cephalic
  - Presenting part - vertex (MC position is left-occipito transverse)

Engagement: when the largest transverse diameter of the fetal head has crossed the pelvic inlet

Biparietal diameter → Largest transverse diameter
- On per vaginal examination → Station 0 - at level of ischial spine
- On per abdominal examination → ≥ ½" of fetal head palpable
Engaging diameters: Fetal skull

- Biparietal diameter (BPD) [most cases]
- Super subparietal diameter

- Transverse
  - Vertex
  - Brow
  - Face

- AP diameter
  - Suboccipito-bregmatic [14 cm]
  - Mentochondio-bregmatic

- Platypelloid pelvis

- Mentococcus position
  - Submento vertical
  - Mentoposterior position

Synclitic & asynclitic engagement

Synclitic: Sagittal suture lies midway between pubic symphysis and sacral promontory, i.e., lies in the transverse diameter of the pelvic inlet.

Asynclitic

- Anterior
  - Sagittal suture pointing towards sacral promontory
  - Mc in multipara
  - A/K/A Naegel's obliquity
  - Anterior parietal presentation
- Posterior
  - Sagittal suture pointing towards pubic symphysis
  - A/K/A Litzman obliquity
  - Parietal presentation
  - Mc in primigravida

Platypelloid pelvis — engagement occurs in marked asynclitism

Descent: Uterine contraction pushes the head of the fetus

Flexion: Head of the fetus touches the Levator ani muscle, resistance of this pushes fetal head
Head reaches the level of ischial spine, occultus of fetus touch levator ani,

↓

**Internal Rotation:** Push the occiput in downward, forward & inward direction. Occiput → Directly behind pubic symphysis → Hart's rule
- Head rotated → 90°(or) 2/8ths of a circle
- Vorsion in the neck of the fetus

↓

**Crowning:** Head is visible at the introitus - episiotomy is done here if indicated

↓

**Extension:** Head is delivered by extension
- Maximum chances of perineal tear, prevented by supporting the perineum by one hand
- Ritgen / Modified-Ritgen maneuver:
  - With right hand bring the chin forward,
  - With left hand give pressure on occiput so that suboccipito bregmatic diameter (9.5cm) is delivered

↓

**Restitution:** Untwisting of the neck of the baby
- Occiput of fetus move in opposite direction of internal rotation by 7/8th of the circle (or) by 45°
- Shoulder will come directly behind pubic symphysis

↓

**External Rotation:** Shoulders move inside
- Move by 7/8th of a circle
- Outside: Head of the fetus rotate, occiput facing the LOT direction
  \[\therefore\text{occiput at external rotation will be facing the same position as it was facing at the time of labor}\]
- Face of the baby is facing towards mothers Right thigh

↓
One liners in mechanism of labor

- Delivery of fetal head during normal vaginal delivery is by Extension
  - In breech & face presentation it occurs by Flexion
- MC position at the beginning of the labor = LOT > LOA
- Occiput moves by 2/8th of circle in LOT position & shoulder by 1/8th of the circle
- After external rotation Face of the baby faces towards Right thigh of the mother and occiput towards original position from where labor began
- Time for episiotomy: After crowning has occurred

Episiotomy

- Surgically planned incision given on the perineum to facilitate the delivery of fetal head
- Not to be given universally to all females undergoing vaginal delivery – as recommended by WHO
- To prevent perineal tear, WHO doesn’t recommend episiotomy, but perineal support at the time of delivery of head of fetus / Ritgen manoeuvre.

Indications for episiotomy:
- Baby is big – macrosomia
- Perineum is Rigid – Primigravida
- Instrumental delivery – Forceps/Vacuum
- Head in breech
- Shoulder dystocia
- Face to pubis delivery

Episiotomy scissors:
- Tip is blunt & is bent at an angle
  ↓
  Prevent fetal head injury
Episiotomy

- Mediolateral (preferred)
  - Lot of muscle fibers are cut
  - Increased bleeding
  - Difficult repair
  - Slow healing
  - More dyspareunia
  - Can never involve anal sphincter
    → Preferred
  - Vaginal mucosa + muscles are cut
    → Equivalent to 2nd degree perineal tear

- Median
  - Very few muscle fibers are cut
  - Less bleeding
  - Easy repair
  - Healing is faster
  - Less dyspareunia
  - If it extents, can involve anal sphincter
    → Not preferred
  - Here only vaginal mucosa is cut
    = Equivalent to 1st degree perineal tear
THIRD STAGE OF LABOR & ATONIC PPH

Third stage of labor

- Begins after the delivery of the baby & ends at delivery of placenta.
- Plays crucial role in PPH.
- Can prevent PPH if managed properly.
- Average duration = 15-20 min.
- If 3rd stage ≥ 30 min = Prolonged 3rd stage / Retained placenta.
- Can be managed.

  Passively
  →
  Actively

  Human placenta is deciduate, it has to shed off after delivery.
  Duration = 15-20 min.
  Changes of PPH & maternal mortality ↑.

  Waiting for spontaneous expulsion of placenta.
  Active management of Third Stage of Labor (AMTSL)
  Duration = 5-10 min.
  Bleeding ↓
  PPH & maternal mortality ↓
  Best method to prevent PPH = AMTSL.

Main event in 3rd stage of labor: Delivery of placenta.

Methods of placenta delivery

- Schultz method
- Placenta starts separating from centre.

- Duncan method
- Placenta starts separating from periphery.
- Retro placental clot is formed
  ↓ (clot is hemostatic)
  ↓ Blood loss
- Bleeding is evident only after entire placenta has separated
- Shiny fetal side of the placenta - comes out first (Schults - shiny)
- Mc method
- No retroplacental clot formed
- Bleeding is evident as soon as placenta separates
- Dull maternal side of the placenta - comes out first (Duncan - Dull)
- Less common method

- Important factor responsible for placental separation is uterine contractions
- In all cases of retained placenta - 1st management:
  ↑ uterine contractions - Oxytocin
  [but mestrine & prostaglandin not recommended]
  ↓ If placenta is still retained
  manual removal of placenta (in OT - GA)
  Cannot find the plane between placenta & uterus
  Morbidly adherent placenta
- Line of separation of placenta - Lies along zona spongiosa
- Living ligature: middle layer of myometrium - has fibres crisscross manner with blood vessels in between them. When uterus contracts - blood vessels get constricted.
  ∴ Atonic uterus is mcc of PPH
- Height of the uterus immediately after delivery; just below the uterus with 20 weeks of pregnancy

**Signs of placental separation**

- Sudden gush of blood per vagina
- Supra pubic bulge
- Height of uterus ↑ slightly
- Apparent lengthening of the cord - Permanent
- Most important sign - Feel the placenta in vagina
  > Lengthening of the cord
### Steps in AMTSL

Step 1 - Administer a **uterotonic agent** within 1 min of delivery of the baby (most important)

Step 2 - Delayed cord clamping

Step 3 - Deliver placenta by **Controlled cord traction / modified Brandt Andrews technique**

Step 4 - Intermittent assessment of uterine tone → By doctor [earlier - uterine massage]

#### Step 1: Administer a uterotonic agent

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Important points</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Oxytocin</em></td>
<td>10 IU (i/m or i/v infusion)</td>
<td><em>Natural Oxytocin</em> ↓ Nonapeptide</td>
</tr>
<tr>
<td></td>
<td><em>Never given IV bolus</em></td>
<td><em>Synthetic Oxytocin</em> ↓ Octapeptide</td>
</tr>
<tr>
<td></td>
<td><em>1r given IV bolus</em></td>
<td><em>Natural oxytocin synthesized by:</em></td>
</tr>
<tr>
<td></td>
<td><em>Hypotension</em></td>
<td>Paraventricular nucleus of hypothalamus</td>
</tr>
<tr>
<td></td>
<td><em>Cardiac arrest</em></td>
<td>Stored in posterior pituitary</td>
</tr>
<tr>
<td></td>
<td><em>Mechanism of action:</em></td>
<td><em>t½ = 3 - 5 min</em></td>
</tr>
<tr>
<td></td>
<td><em>Calcium influx</em></td>
<td><em>Can be given:</em></td>
</tr>
<tr>
<td></td>
<td><em>Releases PGE₂</em></td>
<td><em>Leads to milk ejection</em></td>
</tr>
<tr>
<td></td>
<td>↓</td>
<td><em>1st line drug recommended by WHO</em></td>
</tr>
</tbody>
</table>

**Physiological contraction ↓ Can be used during labour to augment contractions**

- Upper uterine segment contraction
- Lower uterine segment passively dilate
- Onset of action of uterus - to maintain fetal blood flow

### Table

<table>
<thead>
<tr>
<th>Onset of Action</th>
<th>Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 min</td>
<td>3 hrs</td>
</tr>
<tr>
<td>Immediately</td>
<td>1 hr</td>
</tr>
</tbody>
</table>

*Leads to milk ejection*
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose/Route</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>methyl ergometrin</em></td>
<td>0.2mg IM</td>
<td><strong>Tetanic Contraction</strong></td>
</tr>
<tr>
<td><em>aka Ergometrine, methergine</em>*</td>
<td></td>
<td><strong>Cannot use it at the time of labor</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Phototoxic</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Vials are brown in colour</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>In HIV - if used with protease inhibitors → Hypertension</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Never given IV → Hypertension</strong></td>
</tr>
<tr>
<td>Oxytocin can be used</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Syntometrine</em></td>
<td>SU oxytocin + 0.5mg methylergometrine</td>
<td><strong>Not readily available, expensive, highly potent</strong></td>
</tr>
<tr>
<td><em>Carbitocin</em></td>
<td>100mcg – Slow IV</td>
<td><strong>Synthetic Oxytocin</strong></td>
</tr>
<tr>
<td></td>
<td>Prevent PPH</td>
<td><strong>Octapeptide</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>T½ → 85–100 mins</strong></td>
</tr>
<tr>
<td><em>PGE₂</em></td>
<td>600 mcg – Oral</td>
<td><strong>Side effect –</strong></td>
</tr>
<tr>
<td>(Not best drug)</td>
<td></td>
<td><em>hyperthermia</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Chills</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Dose dependent</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Not contraindicated in asthmas</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Contraindicated in previous Cesarean section patients</strong></td>
</tr>
</tbody>
</table>
### Table: Treatment of Third Stage of Labor & Atonic PPH

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostaglandin E2 (PGE2)</td>
<td>* CarboProst (Best drug to prevent &amp; treat PPH)</td>
</tr>
<tr>
<td></td>
<td>* Side effect: Diarrhoea</td>
</tr>
<tr>
<td></td>
<td>* Contraindication: Asthmatics, Suspected amniotic fluid embolism</td>
</tr>
<tr>
<td>Dinoprostone</td>
<td>* 20 mg suppository per rectal</td>
</tr>
<tr>
<td></td>
<td>(PGE2) Off label - Not recommended by WHO</td>
</tr>
</tbody>
</table>

### AMTSL: Step 2 & 3

- **Delayed cord clamping** → ≥ 1 min to 3 min
  - 80ml of blood in cord (≈ 50mg of iron) → Baby
  - Prevent neonatal anemia

- **Indications for early cord clamping** (< 1 min)
  - Birth asphyxia
  - Neonate needs resuscitation
  - Baby is a diagnosed case of heart disease
  - Rh -ve pregnancy
  - In HIV +ve females - According to NACO
  - Not a part of AMTSL

- Delivery of placenta by controlled cord traction or by modified Brandt Andrews Technique
- Make sure placenta is separated → Otherwise uterine inversion

### Uterine Inversion

- MCC - Mismanaged 3rd stage of labor
- Patient immediately goes into shock (Neurogenic shock)
  - Later on there is Hemorrhagic shock
- MCC of death here: Hemorrhagic shock
- **Degrees of uterine inversion**

  ![Diagram of uterine inversion degrees](image)

  **1st degree**: Fundus is within endometrial cavity above the level of internal OS

  **2nd degree**: Fundus reaches up to internal OS or below it

  **3rd degree**: Fundus reaches up to external OS or below it

  **4th degree**: Entire uterus is lying outside introitus

- **Classification of uterine inversion based on duration of labor**:
  - **Acute inversion**: within 24 hrs of labour
  - **Subacute inversion**: after 24 hrs but within a month of delivery
  - **Chronic inversion**: ≥ 1 month of delivery
Management of uterine inversion:
- Resuscitate patient
- Stop uterotonic drugs
- Do not attempt to remove placenta.
- Attempt inversion manually (Johnson method)

![Diagram of uterus inversion]

- Vitals of patient unstable
- Immediate surgical management
- MC - Huntington method
- Other methods - Haultain, Spinelli, Mustner

- Vitals stable
- Give uterine relaxant (Terbutaline/Nitro glycerine)
- Try to reposition uterus manually
- O'Sullivan's hydrostatic method
- Do surgery
- Unsuccessful

- Whenever correction of inversion is successful → Start uterotonic drug → Await spontaneous expulsion of placenta.

AMTSL: Step 4

- Patient after delivery goes into shock - most probable cause - PPH
- Patient goes into shock immediately after delivery - most probable cause - uterine inversion
- Patient goes into unexplained shock after delivery - most probable cause - Amniotic fluid embolism
Step 4: Intermittent assessment of uterine tone
Not a part of AMTSL:
- Early cord clamping
- IV ergometrin
- Uterine massage

Postpartum hemorrhage

- Blood loss in vaginal delivery → < 500 ml
  Cesarean section → < 1 lts
  Twin delivery → < 1 lts
  Cesarean Hysterectomy → < 1.5 lts

- In any case blood loss more than expected values in 24 hrs of delivery or up to 12 weeks of delivery
- ACOG: Blood loss ≥ 1000 ml irrespective of mode of delivery
- Bleeding is accompanied by signs of hypovolemia within 24 hrs of delivery irrespective of route of delivery

PPH

ATLS (Advanced Trauma Life Support) - Classification of PPH

- Class I - blood loss → < 15%
- Class II - blood loss → 15 - 30%
- Class III - blood loss → 30 - 40%
- Class IV - blood loss → ≥ 40%

Classification of PPH:

Primary PPH
- Within 24 hrs of delivery
  - MCC - Atonic uterus

Secondary PPH
- After 24 hrs but before 12 weeks of delivery
  - MCC - Retained placental tissue
- Overall MCC of PPH - Atonic uterus
- Causes of PPH → 4T's
  1st T = ↓ Tone of uterus (mc)
  2nd T = Trauma (2nd mc)
  3rd T = ↓ Thrombin (bleeding disorder)
  4th T = Tissue (Retained Placental Tissue)

Factors predisposing to Atonic uterus:
1) Over distension of uterus:
   - Twins
   - Polyhydramnios
   - Macrosomia (diabetes)
   - Grand multipara (> 5 delivery)
2) Prolonged labor
3) Precipitate labour [onset of labor to expulsion of fetus; Time = 3 hrs]
4) Prolonged Oxytocin
5) Chorioamnionitis
6) Previous history of PPH
7) Antepartum hemorrhage
8) Adherent Placenta.
9) Fibroid uterus – surface area of uterus ↑

* Hypertension does not lead to PPH

Placental anomalies that can lead to PPH:
- Placenta Succenturiata
- Placenta Bilobata
- Placenta Spuria

Management of PPH

- Step 1: Resuscitation of patient
  - Secure a large bore I/V line
  - IV fluids (Crystalloids)
  - Catheterize bladder
  - Blood grouping, Hematocrit, Coagulation profile
* Arrange for blood
  ↓
  Shock Index: Helps to determine when to transfuse blood.
  * HR
      \[\frac{\text{HR}}{\text{Sbp}}\] = Shock index
  * ⊛ \rightarrow 0.5 to 0.7
  * In pregnancy \rightarrow 0.6 to 0.8
  * If shock index \geq 0.9 to 1.1 \rightarrow Indication for blood Transfusion

- Step 2: Distinguish between Atonic PPH & Traumatic PPH
  * Abdominal examination
    ↓
    Uterus cannot be Palpated
    ↓
    Atonic PPH
    Uterus can be Palpated
    ↓
    Traumatic PPH

Management of atonic PPH

1:17:41

Step 1:
* Give uterotonic drug
  * WHO recommended drug = Oxytocin
  +
  Do uterine massage simultaneously
  * Dose of Oxytocin = 30 - 40 IU as IV infusion in NS or RL

[Not in 5% dextrose, as in PPH Oxytocin has to be given for a longer time: we cannot use electrolyte deficient media, it can lead to volume overload & Hyponatremia.]

* If oxytocin is not available - WHO recommended
  * methyl ergometrine: 0.2 mg - dose \rightarrow 1/4
  Repeated = 2 - 4hrs
  Maximum = 5 dose

  * Syntometrine + 0.5 mg
    ↓
    Oxytocin methyl ergometrin
• PGE (misoprost): 800mcg - sublingually  
  Per - Rectal → Dose 1000mcg (Not WHO recommended)

• Carboprost (PGF - α): 250mcg 1/m  
  Repeated - 15 - 90min  
  max dose → 8 (ie: 2mg)

• Inj. Tranexamic acid : 1gm 1/v (slow over 20mins)

management of PPH - Recommended by WHO :

1st Drug → Oxytocin with simultaneous uterine massage & give Inj. Tranexamic acid

  ↓  
  Bleeding doesn't stop

  ↓  
  methylergometrine

  ↓  
  Carboprost (Best drug to prevent & treat PPH)

  medical management should be tried only for 30mins

  ↓  
  If it fails

  ↓  
  Do mechanical methods

• mechanical methods :

  ↓  
  Recommended by WHO  

  ↓  
  Not Recommended by WHO

  ↓  
  • Uterine massage

  ↓  
  • Sengstaken – Blakemore esophageal catheter  
    Or

  ↓  
  • Bakri Balloon Catheter  
    Or

  ↓  
  Condom Catheter

  Bakri balloon catheter → Fill it with warm NS → 300-500 ml

↓ maximum capacity is 500ml
Temporary method recommended by WHO for PPH:
- Done as we wait for help or in transit to higher centre
- Bimanual compression of uterus
- External compression of aorta
- Use military anti shock garment / treatment

If mechanical methods fail → surgical methods

Surgical methods:
- Compression sutures:
  1) B- Lynch sutures: 
     when you tighten the sutures
     Anterior & posterior wall of uterus will meet
     Bleeding stops
a) Other sutures:
  - Haymann suture - No need to open uterine cavity
  - Cho square suture - Sutures in form of squares
  - Gunshella suture

- Bilateral ligation of Arteries supplying uterus:
  - Uterine artery → Site: at the level of internal os
  - Ovarian artery
  - Anterior division of internal iliac artery
    (Posterior division → Lower limb)

  ↓

  Site: 5cm below the bifurcation of common iliac artery
  * Pulse pressure ↓ by 80%

if bleeding is still not controlled → Hysterectomy

↓

Supracervical / Subtotal Hysterectomy
  (only uterus removed, Not cervix)

2nd Best - Total Abdominal hysterectomy (remove uterus + cervix)

if bleeding is still not controlled

↓

Apply pelvic pressure packs

→ Umbrella pack

→ Parachute pack
TRAUMATIC POST PARTUM HEMORRHAGE (PPH)

Causes of traumatic PPH

- Perineal tear
- Cervical tear
- Hematoma
- Rupture of uterus

**Perineal tear**

- A tear which involves perineum
- Classification of perineal tear

**1st degree**
- When vaginal mucosa/skin are torn

**2nd degree**
- 1st degree + vaginal muscles are torn
  - A: < 50% of external anal sphincter tear
  - B: ≥ 50% of external anal sphincter tear

**3rd degree**
- Anal sphincter is involved

**4th degree**
- 1st + 2nd + 3rd + rectal mucosa tear
  - C: External anal sphincter + internal anal sphincter tear

- 3rd degree and 4th degree tear - Complete perineal tear
methods to prevent perineal tear

<table>
<thead>
<tr>
<th>Recommended by WHO</th>
<th>Not recommended by WHO</th>
</tr>
</thead>
<tbody>
<tr>
<td>• No routine episiotomy</td>
<td>• WHO does not recommend episiotomy in all females undergoing vaginal delivery</td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>• modified Ritgen maneuver</td>
<td>• RCOG recommends warm compress to be applied to perineum to chances of tear</td>
</tr>
<tr>
<td>i) manually supporting the perineum with right hand</td>
<td>↓</td>
</tr>
<tr>
<td>while delivering the head of fetus</td>
<td></td>
</tr>
<tr>
<td>ii) Deflexing head of fetus with left hand</td>
<td></td>
</tr>
<tr>
<td>• Advise patient not to bear down while delivering the head</td>
<td></td>
</tr>
</tbody>
</table>

Management of perineal tear

- Time to repair 3rd and 4th degree tear

  - If recognized within 24 hrs of delivery
    - Immediate repair
  - If recognized > 24 hrs of delivery
    - wait for 2 weeks
    - Repair after infection and inflammation subsides
Technique of repair

1st & 2nd degree tear
- Done in labor room under Local anesthesia.
- Steps
  1st - Suture vaginal mucosa (continuous suture using vicryl/polyglactin)
  2nd - Suture muscles (interrupted sutures)
  3rd - Vaginal Skin (mattress suture)

3rd or 4th degree tear
- Done in OT under general anesthesia.
- Steps
  1st - Repair rectal mucosa
  2nd - Internal anal sphincter (end to end anastomosis)
  3rd - External anal sphincter

End to End Anastomosis
Overlapping Technique

4th - Then repair the 1st & 2nd degree tear

1st - Vaginal mucosa
2nd - Muscles
3rd - Vaginal Skin

Important points after repair
1) Pregnancy should be avoided for an year (ideally) but for atleast 6 months after 3rd/4th degree repair
2) In future these females can have vaginal delivery
3) ACOG recommends a single shot of antibiotic at the time of repair

Cervical tear
- M/C site
- Management
- 3/4 clock > 9/4 clock position
  - Repair the tear
Episiotomy

Definition - it is a surgically planned incision given on posterior wall of vagina to facilitate the delivery of fetal head.

- Episiotomy Scissors
  - Bent and with blunt edges

1 - median episiotomy
2 - mediolateral episiotomy

- Types of episiotomy
  - Median episiotomy
  - Mediolateral episiotomy
### Median and mediolateral episiotomy

<table>
<thead>
<tr>
<th>Median</th>
<th>Mediolateral</th>
</tr>
</thead>
</table>
| * The Levator Ani muscle is thick in the periphery and thin in centre  
  ↓
  So less muscles are cut  
  ↓
  Less bleeding  
  ↓
  Repairing - is easy  
  ↓
  Healing is fast  
  ↓
  Dyspareunia is less  
  * Disadvantage - If it extends it  
  Can involve the anal sphincter (3rd or 4th degree perineal tear)  
  It can lead to fecal incontinence  
  * It corresponds to 1st degree Perineal tear | * Even if it extends, it can never involve anal sphincter  
  * Disadvantage - more muscles are cut  
  ↓
  Bleeding  
  ↑
  ↓
  Repair is difficult  
  ↓
  Healing is slow  
  ↓
  Dyspareunia is more  
  * It corresponds to 2nd degree perineal tear |

---

### Indications for episiotomy

1. Rigid perineum (in primigravida females)
2. Shoulder dystocia
3. Macrosomia
4. If delivering after coming head of breech
5. Instrumental delivery (Forceps / vacuum)
6. Face to pubis delivery
7. Suspecting Perineal tear
Hematoma

- MC site of pelvic hematoma
  - Vulval hematoma
- MC artery to form vulval hematoma
  - Pudendal artery
- MC artery to form vaginal hematoma
  - Uterine artery
- MC symptom
  - Pain
  - Difficulty to pass urine

On Local examination
- Bluish coloured swelling

Management
- Conservative
  - Ice pack
  - Analgesics

Indications for surgical management
1) Patient is hemodynamically unstable (shock)
2) Size of hematoma increasing (> 5 cms)
3) Excruciating pain (it indicates hematoma has extended to muscles)

Surgical management
- Drain hematoma
  - If any active bleeder is present
- Obliterate the cavity of hematoma
  - Close by figure of ‘s’ Suture

- In case of broad ligament hematoma
  - Immediate Laparotomy
Rupture of uterus

m.CC  ↓  m.CC in
Previous cesarean section  ↓  non scarred uterus  ↓  Sign of impending rupture  ↓  Sign of Rupture
Obstructed labor  ↓  Fetal tachycardia
Other causes
i) Grand multipara
ii) Mullerian malformation  ↓  So monitor
• Fetal heart rate
• Check for Scar tenderness

• Other signs of uterine rupture
  ↓
  i) Maternal Tachycardia
  ii) Maternal hypotension
  iii) Feel fetal parts superficially
  iv) Uterus contraction - lost
  v) Hematuria
  vi) Fresh vaginal bleed
  vii) Loss of station - peri vaginally
management

- Of uterine rupture
  - Immediate laparotomy
  - Try to repair uterus
    - if not possible
      - Hysterectomy

- Impending rupture
  - Immediate cesarean delivery
OCCIPITO – POSTERIOR POSITION & MALPRESENTATION

Introduction

• At the onset of labor – Only 10% of fetuses are in Occipito–Posterior (OP) position

• Late stage of labor – Only 2% are in Occipito posterior position
  [Reason – 80–90% cases rotate and become occipito–anterior]

• Etiology of Occipito posterior position
  i) most common cause – Idiopathic
  ii) 2nd most common cause – Anthropoid & android pelvis
  iii) more common in nulliparous females
    [OP-position is a malposition not a malpresentation]
    All malpresentations are common in multipara.

• Occipito posterior positions
  (ROP) Right OP > Direct OP (DOP) > Left OP (LOP)
  [most common]  [least common]

• Mechanism of Labor
  i) In 90% cases, normal delivery occurs, as the head rotates 360° of a circle

  ii) Hence, best management of OP position when diagnosed during labor → wait & watch

  iii) During rotation of head occipito posterior → Occipito–anterior
    ⇒ Vaginal delivery

  iv) Sometimes, Head of Fetus starts to rotate
      ↓
    Reaches occipito–transverse
      ↓
    Ceases rotation [no further rotation]
      ↓
    Incomplete forward rotation
      ↓
    [Reason – Prominent ischial spine]
Deep transverse arrest

- Fetal Head – At occipito – transverse position for an hour despite good uterine contractions
  ↓
  Deep transverse arrest

- Occurs at the level of ischial spines
- More common with android pelvis
- Management of deep transverse arrest

  - Android pelvis
  - Other pelvis

  1. Vacuum delivery
  2. Manual rotation of head followed by forceps delivery
  3. Kielland forceps (outdated)
  4. Caesarean section

  Best management

Direct occipito posterior position

- Rarely, if the head of fetus rotates posteriorly
  ↓
  Direct occipito posterior position

- More common with Anthropoid pelvis
- [AP diameter > Transverse diameter]
• **management of DOP**

  ![Diagram](image)

  - Anthropoid pelvis
  - Any other pelvis

  ![Sub-diagram](image)

  - Vaginal delivery can occur
  - Fetus cannot be delivered and if fetus remains in that position for ≥ 30 mins

  ![Sub-diagram](image)

  - Known as Face to pubis delivery
  - Known as Deep sacral arrest

  ![Sub-diagram](image)

  - Cesarean section

**Transverse lie**

- Lie - transverse
- Presentation - Shoulder
- Dorsum of fetus

  ![Diagram](image)

  - Dorso-anterior
  - Dorso-posterior

**Management**

- Most common cause - prematurity > platypelloid pelvis > multiparity
- No mechanism of labor

  ![Diagram](image)

  - Always cesarean section [Despite baby being alive or dead]
  - Chances of cord prolapse is high with transverse lie
  - If transverse lie is recognized during pregnancy, external cephalic version is tried

**External cephalic version**

- **OPD** procedure
- No anaesthesia required
- Should be done under continuous fetal monitoring

  - Fetal distress → indicates cord around the neck

  - Immediate steps:
    - Abandon the procedure
    - Rotate the fetus back to its original position
- If the cord gets
  - Detangled
    - Continue ECV
  - Remains entangled
    - Take up for cesarean section

- Indications for ECV
  i) Breech
  ii) Transverse lie

- Procedure
  - Rotation of fetus per-abdominally
    - Convert to cephalic presentation

- Pre-requisites for ECV
  i) Period of gestation ≥ 36 weeks [lung maturity]
  ii) Adequate Liquor
  iii) membranes intact
  iv) Can be done in early labor if membranes are intact
  v) There should be no contraindications for vaginal delivery

- Contraindications for ECV
  - Absolute
    - Precious pregnancy
      - [eg: pregnancy after 10 years of infertility, women with heart disease]
  - Relative
    - Previous cesarean section

---

Face and brow presentation

1. Face presentation
   - Presenting part is face
   - Attitude is abnormal
     - Normally → Flexion of head
     - Face presentation → Extension of head
   - Denominator is Chin / mentum
Commonest position is left mentoanterior (LMA)
more common in multiparous females
most common type of pelvis involved is platypelloid pelvis
most common cause is anencephaly
Engaging diameter in mento anterior is submento bregmatic diameter

Management of face presentation
- Denominator is mentum

Position: Mento anterior  Mento posterior
(most common - LMA)

Vaginal delivery possible  Only cesarean section

Face presentation - Vaginal delivery
- Delivery of face → Flexion

Normal vaginal delivery
- Delivery of face → Extension

2. Brow presentation
   i) No mechanism of labor
   ii) Management
      - Caesarean section
        [Reason- engaging diameter is mentovertical (4cms)]
      - Per vaginal examination
        Anterior fontanelle and supra-orbital ridges are felt

3. Mandatory cesarean section - indications
   1. Transverse lie
   2. Brow presentation
   3. Face presentation - mentoposterior position
Per-vaginal differentiation

**Face**
1. Alveolar margins—hard
2. Sucking effect ⊗
3. No meconium
4. 3 bony prominences
   forming Triangle
   [mouth + a malar
   prominence]

**Breech**
1. Anus — soft
2. No sucking effect
3. Meconium ⊗
4. 3 Bony prominences
   forming straight line
   [a ischial tuberosities +
   anus]
BREECH

Introduction:
- MC malpresentation: Breech
- In Breech:
  - Lie: Longitudinal
  - Presentation: Pudic

Incidence of Breech:
- At 28 weeks: 20%
- At 34 weeks: baby spontaneously rotates: 5%
- At term: 3-4%
- MCC of Breech: Prematurity
- MCC of Recurrent Breech: Uterine malformation

Types of Breech

- Thigh flexed
  - Knee flexed
    - Flexed breech
      - P/V: feet + Genitalia
      - Buttoks
      - MC in multigravida
      - Chances of cord prolapse = 6%
    - Over all max chance of cord prolapse: Transverse lie > footling > knee
    - If head of the fetus is extended in breech → Stargazer breech
      - C-section

- Thigh extended
  - Knee flexed
    - Knee presentation
      - Footling
        - Maximum chances of cord prolapse
          - C-section
  - Knee extended
    - C-section

- Thigh flexed
  - Knee extended
    - Frank breech
      - P/V: Buttoks + Genitalia
      - MC in primi
      - Least chances of cord prolapse
      - C-section

- Thigh extended
  - Knee flexed
    - Knee presentation
      - Footling
        - Maximum chances of cord prolapse
          - C-section

- Thigh extended
  - Knee extended
    - C-section
Management of breech:

1. If breech patient comes in pregnancy at ≥ 36 weeks and No C/1 to ECV
   \[\downarrow\]
   do ECV in OPD at ≥ 36 weeks

2. If breech comes at labor
   \[\downarrow\]
   \[\downarrow\]
   \[\downarrow\]
   C-section
   Arrested breech
   Breech extraction
   Delivery

Indications for C-section for breech

1. Footing / Knee presentation
2. Stargazer breech
3. Preterm breech (Baby wt < 1.5 kg) and ECV is avoided
4. Post term breech (Baby wt ≥ 3.5 kg + ≥40 weeks)
5. First twin - breech presentation
6. Breech with previous C-section
7. Breech with any other complication of pregnancy - Placenta previa, PIH
8. Breech score 3 or less
9. Primi with breech-relative indication

Assisted breech delivery
- The breech is allowed to deliver spontaneously up to umbilicus and then assistance is given for delivery of shoulder and head
- Delivery happens due to efforts (push of mother) + assistance of obstetrician
- MC type of vaginal breech delivery

Breech extraction
- Mother is ↓ 6A - No efforts (push) by mother
- The entire delivery is done by obstetrician using manoeuvres
- Only indication: Successful internal podalic version in transverse lie of and twin
Assisted breech delivery

- Lie: longitudinal
- Presentation: Podalic
- Denominator: Sacrum
- MC position: LSA (left sacro-anterior)
- Induction and Augmentation (by oxytocin or ARM) should be avoided
- Any delay in 1st stage or 2nd stage of labor should be taken as CPD → C-section
- In breech
  - Make patient nil orally → Give IV fluids
  - Patient should be lying as much as possible, because ambulation can lead to rupture of membranes and cord can prolapse
  - Meconium staining can be normal in breech as long as FHS is normal
  - Do continuous FHR monitoring by cardiotocogram
  - Epidural analgesia can be given for pain relief

Mechanism of breech delivery

- 1st part delivered in breech: Buttocks
- Engaging diameter: Ilioischial diameter (10 cm)
- In RSA position: Buttocks engage in Rt. Oblique diameter
- When buttocks distend the perineum i.e. climbing up of perineum
  - ↓ Give episiotomy

- Buttocks lie in AP diameter during internal rotation such that from RSA, sacrum goes to RST (Right Sacro Transverse)
- Breech is the only exception where denominator (sacrum) does not lie directly behind the pubic symphysis during delivery
- Savage technique: Spontaneous delivery of baby till level of umbilicus
  - ↓ Wrap a towel around baby’s body to prevent cord exposure this is called as savage technique
  - ↓ Shoulder Delivery
In shoulder delivery:

* Engaging diameter: Bis acromial diameter - 13 cms
  
  Lie along Rt oblique diameter of pelvis
  ↓

  Shoulder comes into anteroposterior diameter
  (for this anterior shoulder turns clockwise by 45°)
  ↓

  Anterior shoulder gets delivered first followed by posterior shoulder
  ↓

  Restitution of shoulder by 45° in antclockwise direction
  ↓

  Delivery of head (After coming Head)
  
  Buttocks and shoulder lie in same oblique diameter, Head in opposite oblique diameter

Delivery of After coming Head:

* Engaging diameter: Suboccipitofrontal diameter
  
  Lie along left oblique diameter of pelvis
  ↓

  Occiput moves anteriorly towards and behind the public symphysis
  ↓

  Delivery of Head in flexed position

---

Manoeuvres used in breech delivery

For delivery of Buttocks:

* Delivery of buttocks occurs spontaneously
  
* Only if spontaneous delivery of buttocks is not occurring:
  
  Groin traction (in flexed breech)
  (or)

  Pinard manoeuvre (unlocking the popliteal fossa in frank breech)
Delivery of shoulders:

- Shoulders not in AP Diameter
  - Hold the back of the baby and turn the shoulders in the Anteroposterior Diameter
  - most of the times, delivery happens spontaneously after this manoeuvre
  - if not
  - Rotate the baby so that the posterior shoulder becomes anterior and the delivery happens ↑ lovset's manoeuvre, deliver the anterior shoulder in the same manner, while rotating the baby, the back should always be anterior

For delivery of head:

1. Once shoulder gets delivered
   - Allow the baby to hang by its own weight
   - when nape of neck is visible → Give suprapubic pressure (promotes flexion of neck)
     - Hold the legs of the baby, turn it towards mother's abdomen
     - Head of the baby gets delivered.
     - Burns marshall technique

2. Let the body of the baby rest on the hand
   - Fingers of left hand on either side of cheeks
     - Right hand → one finger each on either shoulder of baby, middle finger on occipital protruberance
     - Shoulder traction and malar flexion
     - mauriceau smellie veit technique
wigand technique → no shoulder traction. one finger each of left hand on either cheek of baby, one finger in mouth. Suprapubic pressure → delivery of head by flexion

3. Delivery of Head by Forceps
   - Forceps used: Neville Barney forceps, Das forceps
   - Overall special forceps used: Pipert's forceps

**Entrapped head**

```
Entrapped Head
  └── Preterm breech
      └── Dührssen incisions on cervix at 2'o clock and 10'o clock positions
          └── Deliver the Head
  └── Term breech
      └── entire body of baby pushed back into uterus and c-section done
          └── Zavanelli's technique
```

- Normal position in breech - dorsoanterior (back facing the pubic symphysis)

In dorso posterior breech (back of baby facing sacrum)

Delivery of the head is done using Prague's manoeuvre

**Groin traction**

**Pinard manoeuvre**
IMPORTANT POINTS ON LABOR

Pain during labor / labor analgesia

- In early stages of labor: Pain due to uterine contractions
  - Nerve supply of uterus: T₁₂–L₁ – pain in these segments

- In later stages of labor: Pain due to dilation of cervix
  - Nerve supply of cervix: S₂–S₅

- For painless labor
  - Block: Epidural analgesia
  - DOC: Bupivacaine

- For Caesarean: Level of anesthesia given at T₄ segment
- For instrumental delivery (Forceps / Vacuum):
  - Pain is due to stretching of the vulva
  \[ \downarrow \]
  - Supplied by Pudendal nerve (S₂–S₅)
  \[ \downarrow \]
  - Pudendal nerve block is done for instrumental delivery

- Site for pudendal nerve block: Ischial spine
- Ligament pierced during pudendal block:
  - Sacrospinous ligament
- Direction of needle: Postero medial

Phases of parturition

- Phases of parturition ≠ stages of labor

<table>
<thead>
<tr>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Phase 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>uterus is quiescent (inactive)</td>
<td>uterus starts preparing for labor</td>
<td>Actual labor phase up till delivery of placenta</td>
<td>Period after the delivery of the placenta +</td>
</tr>
<tr>
<td>includes entire period of pregnancy</td>
<td>includes prelabor</td>
<td>includes stage 1, 2, 3</td>
<td>* Entire puerperium stage 4 + puerperium</td>
</tr>
<tr>
<td>Lightening</td>
<td>Cervix ripening</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
• Cervical softening  Formation of Labour
  begins in phase 1  LUS

**Leopold's manoeuvre**

<table>
<thead>
<tr>
<th>manoeuvre</th>
<th>examiner</th>
<th>examiner's hands</th>
<th>explains</th>
</tr>
</thead>
</table>
| 1st maneuver            | Face of patient | Fundus of uterus | • Lie of fetus
  k/a                    |            |                  | • Presentation
  fundal grip            |            |                  |                               |
| 2nd maneuver            | Face of patient | Either side of maternal abdomen | • Position of fetus
  k/a                    |            |                  | • Back of fetus
  lateral grip           |            |                  | Lt. side _RT. Side
  |            |                  | LOT, LOA, ROT, ROA |
| 3rd manoeuvre           | Face of patient | Pelvis           | Head engaged or not |
  k/a                    |            |                  |                               |
  1st pelvic/pawlik grip |            |                  |                               |
| 4th manoeuvre or 2nd    | Feet of patient | Pelvis           | • Head engaged or not
  pelvic grip            |            |                  | • more comfortable to patient than
  |            |                  | 1st pelvic grip |

Leopold 1,2,3 - Done facing the face of the patient
Leopold 4 - Done facing the feet of the patient
**Rule of 5 / Crichton’s method**

- Tells whether the head engagement
  - Crichton’s method
    - Divide the head into 5 equal parts
    - Numerator tells about how much fetal head is felt per abdomen
  
  \[
  \begin{array}{cccccc}
  5/5 & 4/5 & 3/5 & a/5 & 1/5 & 0/5 \\
  \text{Entire head felt P/A} & \text{Head just got engaged} & \text{Head in pelvic cavity} & \text{Head below pelvic brim} \\
  \text{Free floating head} & \text{Engaged} & \text{Head in} \\
  \end{array}
  \]

**Obstetrical neuropathies**

- MC time: Intrapartum period > Post partum period
- MC nerve injury seen in labor: Lateral femoral cutaneous nerve > Femoral nerve
- MC nerve injury in lithotomy position: Common peroneal nerve
- MC nerve injury due to McRobert’s manoeuvres: Lateral cutaneous nerve of thigh
- MC nerve injury due to usage of forceps: Obturator > Femoral
- MC nerve injury seen in post partum period: Foot drop due to compression of lumbosacral plexus
- MC nerve injury due to c-section: Iliohypogastric and Ilioinguinal
Management of tachysystole

1. Stop the drug given as oxytocics
2. Change the position of patient into left lateral position
3. O₂ by mask
4. Relaxation of uterus by tocolytics. Preferred Drug: Terbutaline

Ferguson Reflex:
- mechanical stretching of cervix
- Enhances uterine activity
  - manipulation of cervix and stripping of membranes leads to release of prostaglandins
  - Hence stripping of membranes leads to induction of labor
  - Rupture of membranes - augmenting the labor

Induction of labor

- It means initiating uterine contractions in a pregnant female
- Before doing induction of labour → Be sure that cervix is ripe
- To know about cervical ripening → Bishop score
  - Components of Bishop score: Mnemonic
    - Delhi: Dilation of cervix - most important component
    - Police: Position of Cervix - max score = a
    - Employed: Effacement of Cervix
    - Special: Station of fetal Head
    - Commandos: Consistency of Cervix - max score = a
  - Each component given a score of 0 - 3
  - If score ≥ 8 - Cervix is favourable & induction is successful
  - Score ≤ 6 - Cervix is not favourable

First ripen the cervix
- In modified Bishop score: Instead of effacement - length of cervix is used
- Max. Bishop score can be 13
methods for cervical ripening:

- Mechanical methods
  - Laminaria tents
  - Osmotic dilators
  - Sweeping / stripping of membranes

- Drugs
  - Prostaglandins
    - Misoprost (tab) - PGE<sub>2</sub>
      - Dose: 25 mcg p/v
      - Can be repeated every 4 hrs
      - Max 6 doses
      - Once the contractions begin after 4 hrs
        - Start oxytocin
    - PGE<sub>2</sub> - Dinoprostone cerviPrime gel
      - 5 ml gel have 0.5mg of drug
      - Put the gel intracervically / in posterior fornix
      - Repeat the gel after 6 hrs
      - Max applications = 3
      - Once the contractions begins after 6 hrs
        - Start oxytocin

Swelling on fetal head

- Swelling above periosteum
  - Collection of blood below periosteum
- Generalized swelling
  - Localized collection of blood
- Occurs because head of fetus stays in one position for a long time
  - Occurs due to traumatic instrumental delivery
* Pitting +
  - Not limited by suture lines
  - Present at the time of birth
  - Resolves by few hours
* Pitting -
  - Limited by suture lines
  - Not present at birth
  - Appears within few hrs of birth and then disappears within few days
* Not associated with fracture of underlying bone
  - Associated with jaundice
  - Do not drain

**Cord prolapse**

* Obstetrical emergency
* Management:
  - Immediately reposit the cord above the presenting part
  - Elevate the buttocks of female
  - Retrograde filling of bladder using Foley's catheter
  - Give O₂ with mask
  - Change the position of patient to left lateral position

\[ \text{Delivery in cord prolapse} \]

\[ \text{Dead fetus} \quad \downarrow \quad \text{Active fetus} \]

vaginal delivery

Exception: transverse lie

\[ \downarrow \]

\[ \text{Check for cervical dilatation} \]

\[ <10 \text{ cms (or)} \quad \downarrow \quad \text{Fully dilated} \]

\[ \text{Not fully dilated} \quad \downarrow \quad \text{Emergency c-section} \]

\[ \text{Check station of fetal head} \]

\[ <\text{a station} \quad \downarrow \quad >\text{a} \quad \downarrow \quad \text{c-section} \quad \text{Forceps delivery} \]

**Chances of cord prolapse:**

Transverse lie > Footing > Knee presentation
INSTRUMENTAL DELIVERY

- Done only when head ≥ +2 station
- Forceps at
  - +2 station
  - +3, +4, +5 station
- Low forceps
- Outlet forceps (Wrigley’s forceps)

Low and outlet forceps

1° Pre-requisites of outlet forceps
   - Scalp visible without separating mother’s labia
   - Fetal skull at pelvic floor
   - Fetal head at perineum
   - Sagittal suture of fetus → Anteroposterior direction
     Or
     Maximum deflection < 45°
   - (Reason - Forceps cannot rotate the head)

2° Direction of pull
   - Low forceps: Downward → Forward → Upward & Forward
   - Outlet forceps: Forward → Upward & Forward
### Forceps and vacuum

<table>
<thead>
<tr>
<th>Forceps</th>
<th>Vacuum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Criteria</strong></td>
<td><strong>1. Criteria</strong></td>
</tr>
<tr>
<td><strong>F</strong> - Favourable position &amp; station</td>
<td>All the criteria as forceps delivery except -</td>
</tr>
<tr>
<td><strong>O</strong> - Os should be fully dilated</td>
<td>- Vacuum is applied when cervix is ≥ 6 cm dilated</td>
</tr>
<tr>
<td><strong>C</strong> - Cervix fully dilated</td>
<td>- Vacuum can rotated head of the Baby</td>
</tr>
<tr>
<td><strong>R</strong> - * membranes should be Ruptured</td>
<td></td>
</tr>
<tr>
<td><strong>E</strong> - * Head should be Engaged</td>
<td></td>
</tr>
<tr>
<td><strong>P</strong> - Pelvis adequate, no Cephalopelvic Disproportion (CPD)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>a. Traction</strong></th>
<th><strong>a. Pressure</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>* Primi - 20 Kgs</td>
<td>* Initial pressure - 0.3 Kg/cm²</td>
</tr>
<tr>
<td>* Multi - 13 Kgs</td>
<td>* Maximum pressure - 0.8 Kg/cm²</td>
</tr>
<tr>
<td><strong>Application</strong></td>
<td><strong>Chignon</strong></td>
</tr>
<tr>
<td>- Applied along occipitomental diameter</td>
<td>- Iatrogenically created Caput succedaneum</td>
</tr>
</tbody>
</table>

**Application**

- Anterior fontanelle
- Flexion point
- Posterior fontanelle
Instrumental Delivery

Centre of cup – applied at flexion point
↓
- 3 cm anterior to posterior fontanel
- 6 cm posterior to anterior fontanel
- Vacuum cup – 6 cm diameter
- Distance between rim of the cup and anterior fontanelle – 3 cm

3. Advantages of forceps:
- Preterm delivery
- Fetal distress
- All pre-presentations where vaginal delivery is possible
  - Vertex
  - After coming head of breech
  - Face – mentoanterior

Cannot be used in:
- Preterm delivery
- Fetal distress
- Can be used only in vertex
- But not in
  - After coming head of breech
  - Face – mentoanterior

Indications & complications of instrumental delivery

<table>
<thead>
<tr>
<th>Forceps</th>
<th>Vacuum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indications</td>
<td>Indications</td>
</tr>
<tr>
<td>Maternal distress</td>
<td>Maternal distress</td>
</tr>
<tr>
<td>Fetal distress</td>
<td>Not in fetal distress</td>
</tr>
<tr>
<td>Prolonged 2nd stage of labor</td>
<td>Prolonged 2nd stage of Labour</td>
</tr>
<tr>
<td>Heart disease/PIH (pregnancy induced hypertension) ↓</td>
<td>Heart disease &amp; PIH ↓</td>
</tr>
<tr>
<td>Cut short 2nd stage of labour ↓</td>
<td>(vacuum &gt; Forceps)</td>
</tr>
<tr>
<td>Prophylactic use of forceps</td>
<td>Reason – Lithotomy position is not required in vacuum</td>
</tr>
</tbody>
</table>

00:19:41
### Complications
- Maternal injury
- More common fetal complications are
  - Facial nerve injury
  - Brachial plexus injury

### Complications (continued)
- Fetal injury
- More common fetal complications are
  - Cephalohematoma
  - 6th nerve palsy
  - Retinal injury

#### Maximum Attempts
### Maximum 3 attempts
- Not delivering
- Failed forceps
- Management - cesarean section

### Maximum 3 attempts (continued)
- Not delivering
- Failed vacuum
- Management - cesarean section

---

**Contraindications of instrumental delivery**

1. Mother - HIV +
2. Fetus - known coagulopathy
3. Osteogenesis imperfecta
4. Contracted pelvis / Cephalopelvic Disproportion (CPD)

---

**Other named forceps**

```
Forceps
  └── Outlet forceps
      ├── India
      │     └── Wrigley forceps
      └── World
          └── Primi multi
              └── Sampson forceps
                  └── Tucker, Mc Lane forceps

  ├── Rotate head of baby
  │     └── Kieland forceps

  └── After coming head of breech
      └── Piper's forceps
```
Introduction of forceps blade

- Fetal head position during labour -
  LOT (Left Occipito - Transverse) / LOA (left occipito - Anterior)
  ↓
  \:. Left blade - introduced 1st

- In case of ROT/ROA (Right occipito - transverse / Right occipito - Anterior)
  ↓
  Right blade - introduced 1st

Parts of forceps

Cephalic curve

Pelvic curve

Blades
Shanks
Handle

Parts
  i) Cephalic curve - Accommodates fetal head
  ii) Pelvic curve - Aligns with bony pelvis
  iii) Shank
  iv) Handle

I. Kielland Forceps

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- Long forceps
  - used to deliver a head above +2 station

a. Wrigley's forceps

- Handle - small
- A short, curved forceps

3. Milne Murrays Forceps

Traction device
- Outdated forceps
- Has a traction device (to deliver head above +2 stations)
CESAREAN SECTION & VBAC

Cesarean section

Abdominal delivery of viable fetus following an incision on skin and uterus

1. Skin Incisions in Cesarean Section
   a. Pfannenstiel incision
      - Curved incision
      - 2 cm above public symphysis
      - Low transverse incision
      - Advantage: cosmetically good
      - most commonly used

   b. Joel Cohen incision
      - Straight incision
      - 3 cms above pubic symphysis

Technique

- Pfannenstiel subcutaneous tissue - cut
- Rectus sheath - cut
- Separation of Rectus sheath from Rectus abdominis
- Peritoneum-cut

- Joel Cohen Skin incision
- Small incision on subcutaneous tissue and separated with fingers
- Small incision and separation of Rectus sheath
- Advantage - ↓ Blood loss
  - ↓ pain (post-operatively)
Better skin incision - Joel Cohen > Pfannenstiel

Uterine incisions in cesarean section

<table>
<thead>
<tr>
<th>Upper uterine segment incision</th>
<th>Lower uterine segment incision</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Classical cesarean section</td>
<td>a. Lower segment cesarean section [LSCS]</td>
</tr>
<tr>
<td>b. Chance of uterine rupture in next pregnancy</td>
<td>b. Chance of uterine rupture ↓[0.2-1.5]</td>
</tr>
<tr>
<td>- ↑[4-9%]</td>
<td>* VBAC [vaginal birth after cesarean]</td>
</tr>
<tr>
<td>- Reason:</td>
<td>- If pelvis adequate, vitals normal ↓</td>
</tr>
<tr>
<td>* upper segment - active contraction and relaxation</td>
<td>VBAC can be performed for next pregnancy</td>
</tr>
<tr>
<td>* lower segment - passive dilation</td>
<td>- VBAC / Trial of scar</td>
</tr>
<tr>
<td>- Prevention of rupture</td>
<td></td>
</tr>
<tr>
<td>* every time a female conceives ↓</td>
<td></td>
</tr>
<tr>
<td>Cesarean section</td>
<td></td>
</tr>
<tr>
<td>c. Sanger incision</td>
<td>c. Merr incision</td>
</tr>
<tr>
<td>- vertical incision</td>
<td>- LSCS</td>
</tr>
<tr>
<td>- upper uterine segment</td>
<td>- Rupture = 0.2 - 1.5%</td>
</tr>
<tr>
<td>- classical incision</td>
<td>- most commonly used</td>
</tr>
<tr>
<td>- Rupture: 4-9%</td>
<td></td>
</tr>
<tr>
<td>d. T-shaped incision</td>
<td>d. Kronig / De lec incision</td>
</tr>
<tr>
<td>- Rupture: 4-9%</td>
<td>- LSCS</td>
</tr>
<tr>
<td></td>
<td>- Rupture = 1-7%</td>
</tr>
</tbody>
</table>

Indications for uterine incision

A. Classical Cesarean incision
   Principle - if lower segment cannot be approached
Absolute indication → Cancer cervix
- Placenta previa with vessels all over the anterior wall of LUS [lower uterine segment]
- Dense adhesions around lower segment - ☑
- Vesico vaginal fistula repair done previously
- Preterm Cesarean section [Improperly formed LUS]
- Post mortem cesarean section

B. Kronig incision
- Constriction ring present

Misgav’s ladach technique

- Skin incision – Joel cohen
- Type of incision – Transverse uterine incision of LUS
- After delivery of baby
  - Closure of uterus → Single layer
- Visceral & parietal peritoneum – Should not be closed
- Closure of abdomen – 2 layers → Rectus sheath + skin

VBAC

- LSCS due to any reason except contracted pelvis
↓
- Next pregnancy → All conditions are Normal, No cephalopelvic disproportion (CPD), Adequate pelvis
↓
  - VBAC/Trial of scar

- Contraindications for VBAC
  - Type of uterine incision – Classical, T-shape [inverted], Kronig
  - Previous History of (H/o) uterine rupture
    1) Previous H/o LSCS + uterine rupture → Recurrence 6%
    2) Previous H/o classical c-section + uterine rupture → Recurrence 32%
  - H/o any surgery where uterine cavity was opened
    1) Hysterotomy
    2) Myomectomy
- Previous h/o ≥ 2 LSCS [unless there is H/o previous vaginal delivery along with a LSCS]
- Any contraindication to vaginal delivery

* Relative contraindications for VBAC
  - Macrosomia in present pregnancy [H/o previous LSCS]
  - H/o puerperal infection
  - Breech in present pregnancy
  - Post term pregnancy
PUERPERIUM

Puerperium

Period after delivery up to 6 weeks postpartum

Immediate  Early  Late

↓   ↓   ↓
with in 24 hrs 24 hrs to 1 week 2nd week to 6th week
of delivery of delivery 6th week

maximum chance of infection in mother

Involution

1) Uterus: Position

- Umbilicus
- Uterus / Just below umbilicus
- 20 weeks of pregnancy
- Till 48 hrs: uterus remains in this position
- Decreases by 1.25 to 1.5 cm/day
- Pubic symphysis → 10-12 day
- Normal position: 4 weeks after

Size (weight) of uterus:

- Immediately after delivery → 1000 gms
- 1st week → 500 gms
- 2nd week → 300 gms
- 3rd week → 100 gms
- 6th week → 60 gms
Clinical significance

**Tubectomy**

- Within 1 week of delivery
- After 6 weeks of delivery
- Post-partum sterilization
- Internal sterilization

methods of sterilization

- Laparoscopic sterilization or minilaparotomy

  - Cannot be done in post-partum period
  - Can be done in post-partum period

- mc method for female sterilization: Laparoscopic sterilization
- mc method of post-partum sterilization: minilaparotomy

Subinvolution

If uterus does not return to its normal position & size in the required time frame

- Causes:
  1. Infection: Endometritis
  2. Retained products of conception
  3. Fibroid uterus
  4. Over distended uterus

Investigation

- USG + Gentle curettage + Antibiotics

mc cause of Asherman's syndrome → Curettage done in postpartum period
Endometrium: Decidua

Decidua

↓

Superficial

↓

- Shed as lochia
- Normally till 15 days after delivery
- Can be seen -24 - 36 days after delivery
- Fishy odour
- Initially alkaline

↓

Acidic

Deep

↓

→ Helps in regeneration of Endometrium
- Begins by 7 days
- Completed by 10 days
- Restored at 16 days
Except at the place where placenta was attached

↓

Occurs by 6 weeks

<table>
<thead>
<tr>
<th>Lochia Rubra (red)</th>
<th>RBC (maternal)</th>
<th>1-4 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lochia serosa (yellow)</td>
<td>mucus + WBC + Exudates</td>
<td></td>
</tr>
<tr>
<td>Lochia alba (white)</td>
<td>Decidual cells</td>
<td>10 - 13 days</td>
</tr>
</tbody>
</table>

Endometritis

- mc site: Placental site
- mc manifestation: Puerperal pyrexia (100.4°F)
- mc Cause of puerperal pyrexia: Endometritis
- mc Organism: Group A streptococci
- mc Organism for late onset Endometritis: Chlamydia.
- In most cases - Mixed infections
  ↓
  - Gram negative bacteria - Ecoli / Klebsiella
  - Anaerobic
- mc / Highest risk of Endometritis - Cesarean section
  ↓
  13 times more chances of Endometritis Compared to vaginal delivery
Other risk factors:
1) Iron deficiency anemia
2) Prolonged labor
3) PROM
4) Bacterial vaginosis

- mc route of spread - direct spread
- Signs:
  - uterine tenderness/fullness.
  - Tests: Tvec, Tesr, Tcpr

Management:
Broad spectrum antibiotics
(Gentamycin + clindamycin or Ampicillin)

Changes in cervix or vagina

Cervix
\[\downarrow\]
External Os
\[\downarrow\]
Nulliparous
\[\downarrow\]
Pin point/
Circular

Multiparous
\[\downarrow\]
Transverse/
slit like

- T-rebuild
  (maximum chance of HPV infection)

Vagina
\[\downarrow\]
- Come back to its normal size and shape
- Rugosites appear in 3 weeks
- Birth trauma is a risk factor for
  \[\downarrow\]
  Prolapse

Breast

\[\downarrow\]
Milk secretion
Prolactin
C/I breast feeding
- **Maximum levels - Pregnancy**
  - **↑ prolactin - amenorrhoea**
  - Hypothalamus
    - GnRH (→)
    - **prolactin**
  - Anterior pituitary
    - LH
    - FSH
  - Ovary
    - **Pregnancy**
    - Progesterone
    - Estrogen
    - **Hypothalamic**
    - **Pituitary**
    - **Ovarian axis**
  - **DOC for ↑ prolactin: Cabergoline**
  - **DOC for ↑ prolactin in infertile female: Bromocriptine**
  - **DOC for ↑ prolactin: Cabergoline**
  - **DOC:** Cabergoline
    - Supressing milk secretions
    - Not used (risk of venous thrombosis)
  - Less milk secretion
    - **Galactogogue**
    - Metoclopramide
    - Nipple stimulation
    - Breast pump
  - HIV in mother - Not a contraindication for breastfeeding.
  - **Mastitis**
    - Infection of breast parenchyma.
    - Most organism - Staph aureus

- Maternal
  - Baby
    - Alcohol
    - IV Drug abuser
    - Breast cancer
    - Anticancer drugs
    - Infections
      - Active herpes on breast
      - Active untreated TB

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- MC source: Infants nose + mouth
- Symptoms: 3-4 weeks
- Unilateral
- Not a contraindication of breast feeding.

- Clinical presentation:
  - Fever
  - Breast engorgement
  - Tenderness

- Management:
  - Penicillin + Dicloxacillin + milk expression

- In 10% it can lead to “Breast abscess”

O/e - Fluctuant mass or fever not resolving inspite of antibiotics.
Management: Incision and Drainage (under general anesthesia)

**Pelvic thrombophlebitis**

Diagram:
- Endometritis
  - Lead to thrombus formation in venous circulation of mother
  - Acts as a nidus - attracts anaerobic bacteria
  - Thrombophlebitis

- MC vein involved - ovarian vein (right > left)
- MC on right side ovarian vein
- Patient presents with spikes of high fever inspite of antibiotics
  + Pain in abdomen + tender mass near cornua of uterus
- IOC: MRI & CT
- Management: High dose antibiotics + Heparin
## Psychological problems in postpartum

<table>
<thead>
<tr>
<th>Postpartum blues</th>
<th>Postpartum psychosis</th>
<th>Postpartum depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seen in 50-60% mildest</td>
<td>&lt;1% serious</td>
<td>10 - 20%</td>
</tr>
<tr>
<td>Gradual onset within 10 days of delivery</td>
<td>sudden onset 10 - 14 days</td>
<td>Gradual onsets 4-6 months</td>
</tr>
<tr>
<td>Withdrawal of Progesterone</td>
<td>adrenal axis</td>
<td>Changes in HPO</td>
</tr>
<tr>
<td>Reassurance + Care</td>
<td>Antipsychotic drugs</td>
<td>SSRI - Fluoxetine</td>
</tr>
</tbody>
</table>

- Not breast feeding
  - Return of menstruation
    - 40% → 6 weeks
    - 80% → 12 weeks
  - Return of ovulation
    - = 7 weeks
    - (5 - 11 weeks)

- Contraceptive of choice in lactating females:
  - 1st choice: Copper IUCD
    - Time of insertion
    - Post-placental
      - CuT 380 A
    - Post-partum
      - with in 48 hours of delivery

If not wait for 4-6 weeks
<table>
<thead>
<tr>
<th>Contraception mode</th>
<th>Non breastfeeding</th>
<th>Breast feeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>LNG = IUCD</td>
<td>Immediately</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Progesterone only pills (POP)</td>
<td>Immediately</td>
<td>4-6 weeks</td>
</tr>
<tr>
<td>Combined oral pills</td>
<td>3 weeks</td>
<td>6 months</td>
</tr>
</tbody>
</table>

- Barrier contraception - when sexual life resumes (usually after 6 weeks)
ANTEPARTUM FETAL SURVEILLANCE

Fetal monitoring

<table>
<thead>
<tr>
<th>Antenatal period</th>
<th>During labor/Intrapartum</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Daily fetal movements</td>
<td>* Fetal heart rate (FHR) auscultation</td>
</tr>
<tr>
<td>* Non stress test</td>
<td>* Fetal scalp pH monitoring</td>
</tr>
<tr>
<td>* Biophysical score (BPS)</td>
<td>* Cardiotocography (CTG)</td>
</tr>
<tr>
<td>* Modified BPS</td>
<td>* Fetal pulse oximetry</td>
</tr>
</tbody>
</table>

Characteristics of fetal heart rate

* Fetal - normal - 110 bpm - 160 bpm
  heart ↓ ↓
  rate <110 bpm ≥ 160 bpm ↓ ↓
  Bradycardia Tachycardia ↓ ↓
  Indicates-1st sign of uterine rupture ↓ ↓
  management- Laparotomy management-immediate caesarean section
  indicates-impending rupture

Fetal heart rate beat to beat variability

* Beat to beat variability - normally between 5 - 25 bpm
* If beat to beat variability is absent ↓
  Compromised fetus
  Fetal acidosis
  Maternal intake - of tranquillizers, phenothiazine,
  analgesics
* Sinusoidal heart rate pattern - FHR becomes fixed
  ↓
  It is ominous
  ↓
  Seen in
  ↓
  Fetal Anemia
  ↓
  Immediate termination of pregnancy
  ↓
  Twin to twin transfusion incompatibility syndrome
  ↓
  Rh
  ↓
  Vasa previa

Sinusoidal pattern

Fetal heart rate - acceleration and deceleration

* Fetal HR acceleration - ↑ FHR on fetal movement
  ↓
  denotes fetus is healthy

* If the fetus is ≥ 32 weeks
  ↓
  FHR - ↑ to 15 bpm for 15 secs

* If the fetus is < 32 weeks
  ↓
  FHR - ↑ to 10 bpm for 10 secs

* If FHR acceleration happens for ≥ 2 mins
  ↓
  Prolonged acceleration
- NST during labor $\rightarrow$ Uterine contractions
  $\Downarrow$
  The blood flow to fetus $\downarrow$ (P $\alpha$ 1/4)
  $\Downarrow$
  If it is a normal fetus
  $\Downarrow$
  Tolerates this $\downarrow$
  (15 bpm for 15 secs)
  $\Downarrow$
  FHR Normal

  If compromised fetus
  $\Downarrow$
  FHR $\downarrow$
  $\Downarrow$
  Fetal HR deceleration

Types of fetal heart rate deceleration – Early deceleration

- The dip in FHR coincides with uterine contraction
  $\Downarrow$
  The dip in FHR begins when uterine contraction begins
  The dip in FHR ends when uterine contraction ends

- Gradual onset, takes $\geq$ 30 secs
- Seen in head compression
- Physiological

Types of fetal heart rate deceleration – late deceleration

- The dip in FHR does not coincide with uterine contraction
  $\Downarrow$
  The dip in FHR begins after uterine contraction begins
  The dip in FHR ends after uterine contraction ends
- Gradual dip, takes ≥ 30 secs
- Seen in uteroplacental insufficiency

most ominous type of deceleration

Late deceleration

Types of fetal heart rate deceleration — variable deceleration

- The dip in FHR have a variable relationship with uterine contraction
- Sudden dip in FHR - < 30 secs
- Seen in cord compression
- In the variable acceleration

<table>
<thead>
<tr>
<th>Happening for short time</th>
<th>If it is persistent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cord compressed for short time</td>
<td>&gt; 60 secs to recover</td>
</tr>
<tr>
<td>Not a problem</td>
<td>Recurrent in 30 mins</td>
</tr>
<tr>
<td></td>
<td>Dip in FHR during deceleration is ≥ 60 bpm</td>
</tr>
</tbody>
</table>

Ominous Findings
Classification of CTG tracings

Class I
- Normal CTG
  - Early deceleration +/-
  - Late or variable deceleration - absent

Class II
- Worst CTG
  - Absent variability of FHR
    - i) Bradycardia
    - Or
    - ii) Late deceleration
    - Or
    - iii) Recurrent variable deceleration
  - Sinusoidal HR pattern

Fetal movement count

- In Antepartum period
  - Daily fetal movement count - normal
    - 10 movements in 12 hours of non rest
    - 10 movements in 2 hours of rest

- Perception of 1st fetal movement - Quickening
  - In primi
    - 18 weeks
  - In multi
    - 16 weeks

- Fetal movements maximum - at 32 weeks
- If ↓ Fetal movements ↓ - sign of fetal distress
- whenever there is ↓ fetal movement

  Screening test - modified biophysical score Non stress test (NST)

  If abnormal

  Diagnostic test - BPS

Modified biophysical score

- NST
- Amniotic fluid index

Indicates - acute insult to fetus

(TNS + modified BPS)

Tells about the acute & chronic insult to fetus

Done for 20 mins

CTG being done

Scanned with CamScanner
Non stress test

NST – for 20 mins

↓

Should get ≥ 2 accelerations

↓

If < 2 accelerations

↓

CTG – Normal / reactive

↓

No intervention required

↓

If in 40 min < 2 acceleration

↓

Non reactive NST

↓

Diagnostic test – BPS

* NST should be done regularly – weekly beginning from 32 weeks

↓

In low risk pregnancy

* In high risk pregnancy (pregnancy induced hypertension, controlled diabetes)

↓

NST done bi weekly

* In case of uncontrolled diabetes – NST daily

Non stress test – in labour

* NST at the time of labour – CTG

↓

Category I

↓

Category II

↓

Category III

↓

Normal

↓

• Immediate caesarean section
Biophysical score or manning score

- It is done by ultrasound for 30 mins.

Mnemonic:

<table>
<thead>
<tr>
<th>T</th>
<th>Fetal Tone</th>
<th>1 extension + 1 flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Fetal breathing movements</td>
<td>1 breathing movement × 30 sec (swallowing of amniotic fluid)</td>
</tr>
<tr>
<td></td>
<td>menigitis</td>
<td>Gross body movements of the fetus</td>
</tr>
<tr>
<td>Always</td>
<td>Amniotic fluid pocket—(single largest vertical pocket)</td>
<td>2-8cms</td>
</tr>
<tr>
<td>Notorious</td>
<td>NST</td>
<td>Reactive</td>
</tr>
</tbody>
</table>

If BPS

10/10

- Normal
- Continue usual monitoring
- Fluid
- Deliver
- Score
  - 4/10: Deliver
  - 7/10: Continue screening

8/10

- Fluid normal
- Continue screening

6/10

- Fluid
- Normal
- Deliver
- Score
  - 4/10: Deliver
  - 7/10: Continue screening

If patient is ≥ 37 weeks

- Deliver
- Score
  - 4/10: Deliver
  - 7/10: Continue screening

If patient is < 37 weeks

- Repeat BPS in 24 hrs
Intrapartum fetal surveillance

- CTG
- FHR Auscultation

<table>
<thead>
<tr>
<th></th>
<th>1st stage of labour</th>
<th>2nd stage of labour</th>
</tr>
</thead>
<tbody>
<tr>
<td>In low risk female</td>
<td>30 min</td>
<td>15 mins</td>
</tr>
<tr>
<td>In high risk female</td>
<td>15 min</td>
<td>5 mins</td>
</tr>
</tbody>
</table>

Fetal scalp pH monitoring

- Normal scalp pH - 7.25 - 7.35
- If pH - 7.20 to 7.25 - Borderline
  - Repeat scalp pH after 30 min
  - If pH < 7.20 - Caesarean section

Fetal pulse oximetry

- Sensor placed on fetal cheek
  - Normal - \( S_{PO_2} \geq 90\% 
  - If \( S_{PO_2} \leq 30\% \) - abnormal - immediate delivery
- Near notocord mesoderm is called axial mesoderm
  ↓
  On either side: Para axial mesoderm
  ↓
  Adjacent to it: Intermediate mesoderm
  ↓
  Followed by lateral plate mesoderm

- Major part of genital tract: mesodermal origin
  ↓
  Except parts which arise from congenital sinus
  ↓
  Endodermal in origin

- Intermediate mesoderm
  ↓
  Urogenital / genital ridge
  ↓
  Gonads
  ↓
  Renal system

- External genitalia - originate from lateral plate (dorso somatic part) mesoderm
Gonads: development

- Develops from genital ridge (at 5 weeks of intrauterine life)
- Mesodermal in origin
- Till 6 weeks, gonads are bipotential/indeterminate
- Sex of baby decided by
  \[ \downarrow \]
  \[ \text{SRY region / Testis determining factor} \]
  \[ \downarrow \]
  \[ \text{Short arm of Y chromosome} \]
  \[ \downarrow \]
  \[ \downarrow \]
  \[ \text{If SRY region present} \quad \text{If SRY region absent} \]
  \[ \downarrow \]
  \[ \text{Y chromosome present} \quad \text{Y chromosome absent} \]
  \[ \downarrow \]
  \[ \text{Gonads: Testis} \quad \text{Gonads: Ovary} \]
  \[ \text{(medulla of gonads: testis cortex: regress)} \quad \text{(cortex: ovary medulla: regress)} \]
  \[ \rightarrow \text{Formed by 7 weeks of IU life} \quad \rightarrow \text{Formed by 8 weeks of IU life} \]
  \[ \rightarrow \text{Other genes: Sox-9} \quad \text{Wnt4} \]
  \[ \rightarrow \text{Testis} \quad \rightarrow \text{Ovary} \]
Primary sex cords
Testicular cords or Seminiferous cords → Rete ovaries

- 1st feature to distinguish between testis and ovary: Formation of testicular cords
- Testicles and ovaries are indistinguishable histologically up to 10-11 weeks of IU life
  - Leydig cell
  - Sertoli cells
  - Blood vessels
  - Germ cells

- Formation of ovary
  - Only in the absence of Y chromosome Proper development of ovary both X chromosomes are needed
- Turner's syndrome (45X0)
  - Gonads - ovary → improperly developed (streak gonads)

Development of ducts
- In early intrauterine life in both male and female - 2 pairs of ducts are present
  1) mesonephric duct: Wolffian duct
  2) Para mesonephric duct: Mullerian duct
- Appears by 6 weeks
  - Disappear
    - Wolfian duct in female
    - Mullerian duct in male
  - 9 weeks

- In males: SRY region present
  - Y chromosome present
  - Gonads: Testis
    - Sertoli cells
    - Leydig cells
      - Secrete Mullerian inhibiting factor/Anti-Mullerian hormone at 7th week
        - Paracrine locally regression of U/L Mullerian duct
          - Mullerian duct regresses in males
          - Remnants: Males
            1. Prostatic utricle
            2. Appendix of testis (Hydatid of Morgagni)

- Appendix of testis: Mullerian duct
- Appendix of epididymis: Wolfian duct

- Secrete testosterone
  - (begins - 8 weeks; maximum - 15 weeks)

  1. Promote Wolfian duct
  2. Form male internal genital organs
     - Seminal vesicle
     - Epididymis
     - Ejaculatory duct
     - Vas deferens

  Gets converted to dihydrotestosterone with help of 5α reductase

  - Formation of male external genitalia.
Anti Mullerian hormone deficiency

**Male**

Anti mullerian hormone deficiency

Chromosome number 46XY

Gonads: Testis

Sertoli cells

AMH is deficient

MB grows in males

Female genital organs present

→ Fallopian tubes

→ Uterus

→ Cervix

→ Upper vagina

Leydig cells

Form testosterone

Wolffian duct grows

→ male genital organs

Seminal vesicle

Epididymis

Ejaculatory duct

Vas deferens

DHT

→ Male external genitelia
1) Less space: intertwining of ducts
   a) Undescended testis: Cryptorchidism
   b) Vas deferens obstruction
   4) Infertility
   5) Herniation of uterus

   ↓

   Uterus hernia syndrome
   (Persisting Mullerian duct syndrome)

   - Not a cause of ambiguous genitalia in males

**Development of ducts in female**

- SRY region absent
  ↓
- No Y chromosome
  ↓
- Gonads: Ovaries

  - Sertoli cells absent
  ↓
  - No Mullerian inhibiting factor/AMH
  ↓
  - Formation of female genital organs
    ↓
    - Fallopian tube
    - Uterus
    - Cervix
    - Upper 2/3rd of vagina

  - Leydig cells absent
  ↓
  - No testosterone in intrauterine life
  ↓
  - Wolffian duct regresses in female
  ↓
  - Female genital tract
    - Remnant: Pronephros
    - Kobelt tubercle, Hydatid of Morgagni
      - Mesonephros - cranial end - Epoophoron
      - Caudal end - paro ophoron
      - Mesonephric duct - Gartner’s duct

- Lower 1/3rd of vagina develops from congenital sinus sinovaginal bulb
- Vaginal epithelium: endoderm of ugs
- Ovaries: genital ridge

**Development of external genitalia**

- Develop from dorso somatic lateral plate mesoderm
- Bipotential
- External genitalia depends on testosterone level in intrauterine life

\[
\begin{align*}
\text{Present} & \quad \text{Absent / resistance to testosterone} \\
\text{male external genitalia} & \quad \text{Female external genitalia} \\
\text{Penis} & \quad \text{genital tubercle} \quad \rightarrow \quad \text{Clitoris} \\
\text{Scrotum} & \quad \text{genital swellings} \quad \rightarrow \quad \text{Labiya majora} \\
\text{Penile urethra} & \quad \text{genital folds} \quad \rightarrow \quad \text{Labia minora}
\end{align*}
\]

- Male external genitalia formed by 14 weeks
- Female external genitalia formed by 11 weeks
- Sex of baby can be determined on USG by 14 weeks

**Homologous organs**

Organs with same embryological origin

<table>
<thead>
<tr>
<th>male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Prostate</td>
<td>Skene glands / Paraurethral gland</td>
</tr>
<tr>
<td>2) Cowper gland/bulbourethral</td>
<td>Bartholin gland</td>
</tr>
<tr>
<td>3) Littre gland</td>
<td>Glands in labia majora and labia minora</td>
</tr>
</tbody>
</table>

**Ambiguous genitalia**

Sex of the baby cannot be determined by external genitalia.

<table>
<thead>
<tr>
<th>male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>- External genitalia look like male due to presence of testosterone</td>
<td>- External genitalia look like female due to absence of testosterone in intrauterine life</td>
</tr>
</tbody>
</table>
Testosterone is absent or resistant

↓

External genitalia will look like female

↓

Ambiguous genitalia in males

mc cause: Androgen Insensitivity syndrome (Testicular feminizing syndrome)

- If a female is exposed to testosterone

↓

Genitalia look like male external genitalia

↓

Ambiguous genitalia in female

mc Cause: Congenital adrenal hyperplasia

3 structures mc affected:

1) Clitoris: Clitoromegaly
2) Labioscrotal folds: Fuse
3) Phallus (penile urethra)

Hermaphroditism

↓

True hermaphroditism

Both ovary and testis present in same individual

↓

Ovotestis

mc karyotype: 46XX

Pseudo hermaphroditism

Gonads of one sex and external genitalia of other

↓

Male pseudo hermaphroditism

→ Gonads - testis

→ External genitalia

Female pseudo hermaphroditism

→ Gonads - ovary

→ External genitalia

↓

Female

mc cause: Androgen Insensitivity Syndrome

Male

mc cause: CAH
Congenital adrenal hyperplasia - steroid pathway

Steroid pathway:
- Pregnenolone → (17 hydroxylase) → 17 Hydroxyprogrenolone → (17,20 lyase) → DHEA
  - (3 β HSD)
  - (11 β hydroxylase) → Deoxy cortisol
- Progesterone → (17 hydroxylase) → 17 OH progesterone → (11 β hydroxylase) → Deoxy cortisol
  - (3 β HSD)
- Androsterone
  - (11 β hydroxylase) → Testosterone

Congenital adrenal hyperplasia

→ MC enzyme: 11 β hydroxylase
→ 2nd MC enzyme: 11 β hydroxylase
→ Least common: 3 β hydroxysteroid dehydrogenase
→ MC gene defect: CYP21A2
  - Codes for 11 β hydroxylase enzyme

ACTH (↑)

↑ Adrenals

Corticosteroids → Mineralosteroid → Progesterone → Androgen

Negative feedback on ACTH

↓

11 β hydroxylase needed

↓

11 β hydroxylase not needed
In 21 hydroxylase enzyme deficiency

- Corticosteroid ↓
  - Negative feedback on ACTH reduced
  - ACTH ↑
  - Progesterone ↑
    - 17-OH progesterone ↑ (screening test)
- Mineralocorticoid ↓
  - Salt, water wasting
  - Androgen ↑

- External genitalia resembles male:
  - Ambiguous genitalia
- Clitoromegaly, phallic formation, labioscrotal fusion
- Precocious puberty (heterosexual)
- Virilisation
- Bone age increased
  - Early epiphyseal fusion
  - Short stature

Heterosexual precocious puberty:

In females cause:

1. Clitoromegaly
2. Deepening of voice
3. Increase muscle mass
4. Hirsutism
5. Virilisation
11 β hydroxylase deficiency

→ Aldosterone deficiency
→ Corticosteroid, mineralo steroid - not formed
→ Progesterone and androgens are formed
→ Female child - Ambiguous genitalia.
→ Deoxy cortisol level increases
  ↓
  mineralocorticoid like activity
  ↓
  No salt water wasting
  No hypotension
  ↓
  Hypertension + Ambiguous genitalia.

↑ Progesterone
↓ progesterone
17 OH progesetone
↑ Androgen
↓ Ambiguous genitalia
↑ Deoxy cortisolone
↓ (mineralocorticoid like action)
↓ Hypertension

Screening test:
17 OH progesterone levels

< 300 ng/dl
↓
Not a case of CAH

300-600 ng/dl
↓
Do diagnostic test

≥ 800 ng/dl
↓
Definitive (100%) diagnosis of CAH

Diagnostic test for CAH:
ACTH stimulation test:
↓ 3/2 hr

17 OH progesterone → ≥ 1500 ng/dl
↓
CAH
Management of CAH

- Mineralocorticoid defect
  - Hydrocortisone

- Surgical
  - Removal of phalus corrective surgery

- Corticosteroid defect
  - Corticosterone

4-5 years of age

<table>
<thead>
<tr>
<th>Age</th>
<th>Corticosteroid of choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood (before puberty)</td>
<td>Hydrocortisone (short acting)</td>
</tr>
<tr>
<td>At puberty</td>
<td>Prednisolone/Dexamethasone</td>
</tr>
</tbody>
</table>

Pregnant female with CAH

- Hydrocortisone (cannot cross placenta)
- At 10 weeks = FISH/ chronic villus sampling

- Male fetus / unaffected
  - Continue Hydrocortisone

- Affected female fetus
  - Corticosteroids which can cross placenta and treat female fetus

  - Dexamethasone
Late onset CAH

Pathogenesis of CAH begins at puberty

↓ cortisol  ↓ mineralocorticoid  ↑ Androgen  ↑ Progesterone
↓ at puberty
↓
Not in intra
uterine life
↓
Female external genitalia +
↓
- Virilisation
- Hirsutism
- Deep voice
- Increase
muscular mass

→ Differential diagnosis: PCOS

Virilisation
→ Female with a * sexual characters resembling male
→ Breast atrophy
→ Increase muscle mass
→ Deepening of voice
→ Increase hair growth → Hirsutism

Scoring system of virilisation:

Prader system
0 = Normal female
5 = Maximum virilisation

Scoring system for hirsutism
Ferriman Gallwey score (≥ 8)
Gartner's cyst vs. cystocele

→ Gartner's cyst:
  > Present in antero lateral wall of vagina

<table>
<thead>
<tr>
<th>Gartner's cyst</th>
<th>Cystocele</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tender, shiny</td>
<td>Not shiny and tender</td>
</tr>
<tr>
<td>No rugosities</td>
<td>Rugosity present</td>
</tr>
<tr>
<td>Not reducible</td>
<td>Reducible</td>
</tr>
<tr>
<td>Cough impulse absent</td>
<td>Cough impulse present</td>
</tr>
<tr>
<td>well defined margins</td>
<td>Not well defined margins</td>
</tr>
</tbody>
</table>

mc cyst in vagina - inclusion cyst
mc cyst in ovary - follicular cyst
MENSTRUAL CYCLE

Oogenesis

- 1st oocyte
- At puberty: meiosis - I (gets arrested in diplotene stage of prophase)
- 2nd oocyte + 1st polar body

Structure of ovary
- Before puberty

- Cortex
  - Primordial follicle
  - Granulosa cell
- Medulla
  - Blood vessels
  - Theca cells

- Number of follicles:
  1) Maximum = at 5th month of intrauterine life (6-7 months)
  2) At birth = 1-2 million follicles
  3) At puberty = 4-5 lakh follicles

- Initial recruitment of follicles - hormone independent

Role of FSH

- At puberty, Hypothalamo - Pituitary - Ovarian - Axis (HPOA) becomes functional
Hypothalamus

Pulsatile manner \(\rightarrow\) Gonadotropin Releasing Hormone (GnRH)

Anterior pituitary
\(\rightarrow\)

FSH (Follicle Stimulating Hormone) release

Functions of FSH:
- Prevents the follicles from undergoing atresia
- Stimulates follicles

Role of FSH:

FSH acts on Granulosa cells
\(\rightarrow\)

Estrogen
Inhibin - B
- Negative feedback on FSH

Negative feedback on FSH
\(\downarrow\)

\(\uparrow\) FSH

Positive feedback on LH

Proliferates uterine endometrium

... never give estrogen alone to a female with intact uterus

Can cause • Endometrial cancer
• Endometrial hyperplasia

All follicles die except one Dominant follicle (i.e., the follicle with maximum FSH receptors on Granulosa cell)

Note:
1. FSH receptors are present on Granulosa cells
2. Granulosa cell tumour of ovary \(\Rightarrow\) Feminizing tumor
3. Tumor marker for Granulosa cell tumor of ovary \(\Rightarrow\) Inhibin - B
Role of LH

Estrogen has positive feedback on LH

\[ \uparrow \text{LH} \]

- LH surge (sudden increase in LH)
  - Initiated by estrogen
  - Estrogen = 400 pg x 24 hrs
- Meiosis I resumes (hormone dependent)
- Ovulation (1° oocyte → 2° oocyte)
- Follicle → Corpus luteum

Acts on Theca cells
- Produce androgens
  - In Granulosa cells, adipose tissue.
  - Androgens → Estrogen (by aromatase enzyme)

Acts on Granulosa cells

Two cell two gonadotropin theory

- Granulosa cell

\[
\begin{align*}
\text{Cholesterol} & \xrightarrow{\text{LH + cAMP}} \text{CYP-17} \\
\text{LH} & \xrightarrow{\text{CYP-17}} \text{Androstenedione} \\
\end{align*}
\]

\[
\begin{align*}
\text{Theca cell} & \text{Aromatase enzyme absent} \\
\text{CYP17 present} & \\
\text{Granulosa cell} & \text{Aromatase present} \\
\text{CYP17 absent} & \\
\end{align*}
\]

- Adipose Tissue

\[
\begin{align*}
\text{Androstenedione} & \xrightarrow{\text{Aromatase}} \text{E}_1 \\
\end{align*}
\]
Role of LH granulosa cell

↑ LH  
↓
Acts on Granulosa cell  
↓
Luteinization of cells  
↓
Small amount of progesterone release even before ovulation (at the time of LH surge)  
↓
This small amount of progesterone has positive effect on LH, FSH (↑LH, ↑FSH)

Note:
1. In females, LH receptors are present on Theca cell, Granulosa cell
2. Progesterone appears earliest in menstrual cycle at LH surge (i.e. 32-36 hrs before ovulation)
3. There is LH ↑ FSH surge before ovulation

Follicular / proliferative phase of menstrual cycle

Day 1  
↓
FSH  
Granulosa cell  
↓
Release estrogen  
↓
Pre-antral Antral Graffian  
↓
Ovulation  
↓
Negative Proliferative feedback on FSH  
Positive Feedback on LH  
↓
LH surge
Note:
1. Ovarian cycle is initiated by FSH
2. Size of follicle, just before ovulation = 18-20 mm
3. For LH surge to occur - Estrogen = 200 pg x 48hrs
4. LH surge $\xrightarrow{32-36 \text{ hrs}}$ ovulation
   (or)
   $\xrightarrow{24-36 \text{ hrs}}$
5. LH peak $\xrightarrow{10-13 \text{ hrs}}$ Ovulation

- Time interval between estrogen peak to LH peak = 14-24hrs
7. LH surge is initiated by estrogen
8. LH surge is maintained by both estrogen and progesterone
9. Before ovulation there is LH + FSH surge
10. Ovulation is due to LH surge only
11. Meiosis - I is resumed due to LH surge (32-36 hrs before ovulation)

**Ovulation**

1° oocyte $\rightarrow$ 1° oocyte

Cumulus oophorus (granulosa cells surrounding 1° oocyte)

Antral cavity

Antral follicle
1. Normally
   - Antral cavity fluid - estrogen + growth factor + LH
   - LH appears in the antral cavity fluid only toward mid cycle

2. Anovulation
   If LH appears in antral cavity fluid early in the cycle
     \[ \downarrow \]
     - Atresia of follicle
     - \[ \downarrow \] mitotic activity of granulosa cell
       \[ \downarrow \]
       Anovulation

**Secretory phase of menstrual cycle**

1° oocyte \( \rightarrow \) 2° oocyte

Follicle \( \rightarrow \) Corpus luteum

**Note**

LH \( \rightarrow \) maintains corpus luteum in a non-pregnant female.

- Corpus Luteum
  corpus luteum starts growing under the effect of LH
  \[ \downarrow \]
  Day 22 of cycle / 8 days after ovulation - attains maximum size & activity

Hormones produced by corpus luteum

<table>
<thead>
<tr>
<th>Hormones released by corpus luteum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progesterone (mainly)</td>
</tr>
<tr>
<td>Estrogen</td>
</tr>
<tr>
<td>Inhibin-A</td>
</tr>
</tbody>
</table>

\[ \downarrow \] Negative feedback
\[ \downarrow \] \text{Supports uterine}

\[ \downarrow \] Secretory action
\[ \downarrow \] FSH

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Note:
- At low concentration of progesterone $\rightarrow$ $\uparrow$ LH, $\uparrow$ FSH
- At high concentration of progesterone $\rightarrow$ $\downarrow$ LH, $\downarrow$ FSH

Corpus luteum degeneration

$\downarrow$ LH

Corpus luteum degenerates

$\downarrow$ progesterone

Support to endometrium is lost
Endometrium sheds
Menstruation

Note:
- Uterine endometrium
  - Superficial
  - Deep layer (Zona basalis)

Sheds at the time of menstruation

$\downarrow$ Estrogen

Vasoconstriction
Release PGE$_2$, PGE$_3$

$\uparrow$ myometrial contractions
Pain during menstruation
(Dysmenorrhea)

$\downarrow$ Inhibin - A

$\uparrow$ FSH (as negative feedback is gone)

Acts on granulosa cell
Release Estrogen
Proliferates deep layer of endometrium

- Deep layer is responsible for regeneration of entire endometrium in next cycle

Day 14

\[ \uparrow \text{corpus luteum} \] Action on corpus luteum

\[ \downarrow \text{Progesterone (mainly)} \]

\[ \downarrow \text{Estrogen} \]

\[ \downarrow \text{Inhibin A} \]

\[ \downarrow \text{Secretory action} \]

\[ \downarrow \text{LH} \]

Day 28

\[ \uparrow \text{menstruation} \]
• 2nd half of menstrual cycle - luteal / secretory phase

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

One liners in menstrual cycle

1. Main hormone in
   • 1st half of cycle → Estrogen
   • 2nd half of cycle → Progesterone

2. Day 1
   
<table>
<thead>
<tr>
<th>Follicular phase</th>
<th>Luteal phase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 14</td>
</tr>
<tr>
<td>Fixed duration</td>
<td>Day 28</td>
</tr>
<tr>
<td>may vary (depends on cycle length)</td>
<td></td>
</tr>
</tbody>
</table>

• Day of ovulation - Count 14 days backwards from the date of next menstruation

   Or

   Ovulation = Total length of cycle - 14
   (eg: 26 day cycle, day of ovulation = 26 - 14 → Day 12)

3. Corpus luteum
   a. LH → Maintains corpus luteum in non-pregnant female
   b. HCG → Maintains corpus luteum in pregnant female
   c. Life span of corpus luteum in non-pregnant female → 10-13 days
   d. Life span of corpus luteum in pregnant female → 10-13 weeks
   e. HCG → Protects corpus luteum from undergoing luteolysis

Levels of hormones

1. All hormones peak at time of ovulation (LH, FSH)
2. Estrogen → Peaks just before ovulation (24 - 36hrs)
3. Progesterone → Peaks at day 22 of cycle (8 days after ovulation)

4. At Day 22
   • Progesterone peaks
   • Size of corpus luteum – maximum
   • All tests for ovulation – done at day 22 of cycle

5. At high concentration of progesterone → Negative feedback on LH, FSH
- Level of LH, FSH → minimum in secretory phase
  or
  Day 22 of cycle
  or
  1 week before menstruation

- Mittelschmerz Syndrome
  midcycle pain abdomen
  or
  Pain in the abdomen at the time of ovulation
MENSTRUAL CYCLE: NORMAL AND ABNORMAL

Menstrual cycle introduction

- Definition:
  Shedding of superficial layer of endometrium

  Initially: High levels of progesterone supporting endometrium
  Eventually: Progesterone level decrease support of endometrium - lost
  ↓
  menstruation

- Most important hormone needed for menstruation
  ↓
  Progesterone

- Progesterone cannot act unless endometrium is primed with estrogen

- In case of postponing /preponing the menses
  ↓
  2-3 days before expected date of menstruation
  ↓
  Start progesterone (e.g. 5mg Primolut-N TDS)
  ↓
  High progesterone levels
  ↓
  Once stopped, levels of progesterone will go down
  ↓
  menstruation starts after 2-3 days
- Female complains of delayed menstruation (10 days)
  ↓
  Sexually active female
  ↓
  urine pregnancy test (UPT)

  ↓
  Positive
  ↓
  Pregnancy
  mc cause of secondary amenorrhea.
  ↓
  Progesterone levels
  ↓
  Support decidua (pregnant endometrium)
  ↓
  No menstruation
  → After delivery: Placenta removed
  ↓
  Progesterone levels
  ↓
  Shedding of decidua
  ↓
  "Lochia"

  ↓
  Negative
  ↓
  → Reduce Progesterone levels
  ↓
  Shedding of endometrium
  ↓
  menstruation
  → Drug: Meprate 10mg (bd/tid)
  ↓
  5-7 days
  ↓
  Increase Progesterone levels
  ↓
  Once stopped, progesterone levels fall down
  ↓
  Loss of endometrial support
  ↓
  menstruation Starts.

- Female with complains of anovulation
  ↓
  No Corpus Luteum
  ↓
  Reduced / Absent progesterone
  ↓
  "Amenorrhoea."

  ↓
  Hormone present: Estrogen
  ↓
  Proliferates endometrium
  ↓
  Shedding of weak endometrium
  "estrogen break through bleeding"
- **Estrogen break through bleeding**
  ↓
  Can occur in anyone
  ↓
  "Anovulatory Cycles"
  ↓
  Irregular, Painless, heavy bleeding

**Normal Menstrual Cycle**

Sudden decrease in progesterone levels

- Release of metallo-proteinase enzyme
- Vasoconstriction
  PGE2α released
  ↓ myometrial Contraction
  Dysmenorrhea (Pain during menstruation)
  ↓ Indication of ovulation (Indirect)

**Dysmenorrhea**

- Pain at the time of menstruation

<table>
<thead>
<tr>
<th>Primary Dysmenorrhea</th>
<th>Secondary Dysmenorrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Pain due to PGE2α ↓ No Pelvic pathology</td>
<td>1) Pain due to pelvic Pathology Example: → Endometriosis (mc) → Adenomyosis → Fibroids → Polyps</td>
</tr>
<tr>
<td>a) Spasmodic dysmenorrhea</td>
<td>a) Congestive dysmenorrhea</td>
</tr>
</tbody>
</table>
### Primary Dysmenorrhea

3) Seen in: Younger age group
   Complains of dysmenorrhea
   ↓
   → Since beginning of menarche, except for initial few months

4) Pain begins 1-2 days before menstruation
   ↓
   Subsides: onset of menstruation (within 12 hrs)
   → Never present after 48 hrs

### Secondary Dysmenorrhea

3) Seen in: Reproductive age group
   Complains of dysmenorrhea
   ↓
   Present since few months (initially absent)

4) Pain is present 3-5 days before menstruation
   ↓
   Does not subside with onset of menstruation

---

### Management of Dysmenorrhea

- **Primary Dysmenorrhea**
  1. NSAIDs: mefenamic acid
  2. Oral Contraceptive pills (OCP’s)
     ↓
     Anovulatory cycles

- **Secondary Dysmenorrhea**
  → Treatment of underlying disease pathology

- **Oral Contraceptive pills (OCP’s)**
  Synthetic Estrogen + Synthetic Progesterone
  ↓
  Reduce natural FSH and Estrogen
  Reduce Natural LH and Progesterone
  ↓
  Anovulation
Advantage:

1) Used for contraception
2) Management of dysmenorrhea
3) Used as OOC in irregular menstrual cycles
4) Protective for ovarian cancer

In OCP’s:

- If estrogen $\rightarrow$ 30-35 mcg (Low dose pill)
- If estrogen $\rightarrow$ < 20 mcg (Very low dose pill)

5) Protective in:
   - Endometrial Cancer
   - Endometrial hyperplasia
   - Fibroids
   - Endometriosis
   \[\text{Hyper estrogenic conditions}\]

6) Management of excessive bleeding.

   Drugs used:
   1) Tranexamic acid
   2) OCPs
   3) Progesterone

### Characteristics of normal menstrual cycle

<table>
<thead>
<tr>
<th>Description</th>
<th>Study based: 24-38 days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Population based: 21-35 days</td>
</tr>
<tr>
<td>1) Normal length</td>
<td>28 days (15% of females)</td>
</tr>
<tr>
<td>2) Average length</td>
<td></td>
</tr>
<tr>
<td>3) Number of days of bleeding</td>
<td>2-7 days</td>
</tr>
<tr>
<td>4) Average days of bleeding</td>
<td>4-6 days</td>
</tr>
<tr>
<td>5) Normal blood loss</td>
<td>5-20 ml</td>
</tr>
<tr>
<td>6) Average blood loss</td>
<td>1st best answer = 30 ml</td>
</tr>
<tr>
<td></td>
<td>2nd best answer = 35 ml</td>
</tr>
<tr>
<td></td>
<td>3rd best answer = 50 ml</td>
</tr>
</tbody>
</table>
Atypical uterine bleeding

Definition: “Any deviation from normal characteristics of menstrual cycle”

Types:

1) menorrhagia.
   - Excessive bleeding at regular cycles
     ↓
     • Blood loss > 85ml
     Or
     Number of days of bleeding > 7 days
     - Seen in Fibroid

2) Hypomenorrhea.
   - Reduced bleeding at regular cycles
     ↓
     • Blood loss < 5ml
     Or
     Number of days of bleeding < 2 days
     - Seen in “Asherman Syndrome”

3) Oligomenorrhea.
   - Longer duration of cycles
   Or
   Number of cycles in a year is less
   Or
   Infrequent menstruation

4) Polymenorrhea.
   - Short Cycles / Frequent menstruation

5) metrorrhagia.
   • Intermenstrual bleeding / Irregular bleeding
   • Seen in Polyps

6) menometrorrhagia.
   - Excessive bleeding at irregular intervals
     (menorrhagia + metrorrhagia)
   - Seen in Fibroid Polyp, Anovulatory Cycles
Abnormal uterine bleeding (AUB) and dysfunctional uterine bleeding (DUB)

- Excessive Bleeding
  
  Pelvic Pathology
  
  No Pelvic Pathology
  
  Causes excessive bleeding
  
  DUB
  
  Causes of abnormal uterine bleeding

Mnemonic: PALMCOEN

P → Polyps
A → Adenomyosis
L → Leiomyoma (Abroid)
m → Malignancy
C → Coagulation disorder

O → Ovulatory dysfunction
E → Endometrial dysfunction
I → Iatrogenic
N → Not yet Classified

Management of excessive bleeding

1st investigation: TVS (Transvaginal Ultrasound)

Check for: Endometrial Thickening

Pre-menopausal women

menopausal women

if (Fiso) ≥ 5mm or (ACOS) ≥ 4mm

LOC: Biopsy

Uniformly Thickened (Endometriosis)

Endometrial aspiration biopsy

Localised / Focal thickening

Hysteroscopy and biopsy

→ OPD Procedure

→ Instrument: Pipelle device / Valtra aspirator
Investigations
- Gold Standard: Fractional curettage
- 2nd best: Dilation and curettage

Dysfunctional Uterine Bleeding (DUB)

- 80% are anovulatory

Young age

Puberty menorrhagia
- Investigations:
  1. TSH levels
  2. Coagulation profile
  3. TVS

Reproductive age:
- Investigations:
  1. TSH levels
  2. Coagulation profile
  3. TVS

Menstruating age

Complications:
- Pregnancy related
- Premalignant lesions

Rule out cancers

Never do:
- Per vaginal examination
- Dilation and curettage

Management of DUB

- Mild - moderate bleeding

1st line management
- Non hormonal
  1. Tranexamic acid
  2. Mefenamic acid
  For Dysmenorrhea

Best
- Oral Contraceptive pills (OCP’s)
- Progesterone
  (5mg BD x 21 days)
  (In reproductive age)
- Mirena - Progesterone IUCD

For irregular cycles
- Severe bleeding
  ↓
  Check for vitals
  ↓
  Vitals Stable  Vitals unstable
  ↓
  High dose of oral Estrogen x 24 hrs
  ↓
  OCPs or Progesterone
  Dilation and curettage
  ↓
  Put patient on IV estrogen
  / OCP / Progesterone
  (Not available in India)

- Bleeding not controlled by medical management
  ↓
  Age ≥ 40 years
  ↓
  Total Abdominal Hysterectomy (TAH) (uterus + cervix)
  Transcervical resection of endometrium or endometrial ablation (TCRE)
  Contraindicated in young females
HORMONES IN GYNAECOLOGY - 1

Natural GnRH

- Released by Arcuate Nucleus of Hypothalamus
- Released in pulsatile manner
- Decapeptide
- T½ = 3-4 minutes
- Neurons which synthesise the GnRH
  ↓
  Present in olfactory placode
  ↓
  Via olfactory nerve
  ↓
  Arcuate Nucleus

  Neuronal migration is absent
  ↓
  Kallmann syndrome

Kallmann syndrome
- X-linked recessive disease
- Mutation: Kal-1-gene
- M : F = 6 : 1
- Hypothalamic failure + Anosmia ± colour Blindness in male
  ↓
  GnRH ↓
  ↓
  LH ↓ and FSH ↓
  ↓
  Estrogen ↓

- Patient complains of: Delayed puberty
  1° Amenorrhea
  Infertility
- Example of Hypogonadotropic Hypogonadism
- Height of the patient is normal
- At time of puberty:
  
  Active Hypothalamus
  
  \[ \text{GHRH} \downarrow \]
  \[ \text{Neuropeptides} \downarrow \]
  \[ \text{Leptin} \uparrow \]
  \[ \text{Kisspeptin} \uparrow \]
  \[ \text{Glutamate} \uparrow \]
  
  Release GnRH
  (pulsatile manner at night)
  
  Initially: LH at night
  
  \[ \text{FSH + LH} \ (at \ night) \]
  
  Pulse frequency of GnRH
  
  \[ \text{Low} \downarrow \]
  \[ \text{High} \downarrow \]
  
  FSH
  
  LH

**Synthetic GnRH**

- Leuprolide
  - Triptorelin
  - Goserein
  - Nafarelin

- High first pass metabolism
  - Orally inactive
  - Given: S.C, I.V, I.M, Nasal sprays

- Can be given by

  \[ \text{Pulsatile manner} \downarrow \]
  \[ \text{Continuously} \downarrow \]
  
  Acts like natural GnRH
  
  \[ \uparrow \text{LH and } \uparrow \text{FSH} \]
  
  \[ \uparrow \text{Estrogen} \]

  Uses: 1. Delayed puberty
  - Kohlmann syndrome
  2. Ovulation induction

  \[ \downarrow \text{LH and } \downarrow \text{FSH} \]

  \[ \downarrow \text{Estrogen} \]

  Uses: In hyperestrogenic conditions:
  1. Endometriosis
  2. Fibroids
  3. Precocious puberty
→ As it ↓ estrogen if used
≥ 6 months

→ Experience menopause like symptoms (m.C → Hot flashes)

∴ Add back therapy
(Estrogen +
Progestosterone administration)

<table>
<thead>
<tr>
<th>Natural and Synthetic LH and FSH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Natural LH and FSH:</strong></td>
</tr>
<tr>
<td>LH</td>
</tr>
<tr>
<td>→ Released by: Anterior</td>
</tr>
<tr>
<td>pituitary</td>
</tr>
<tr>
<td>→ Receptors present on:</td>
</tr>
<tr>
<td>♀: Theca + granulosa cells</td>
</tr>
<tr>
<td>♂: Leydig cells</td>
</tr>
<tr>
<td>→ T ½: 20 min</td>
</tr>
<tr>
<td>FSH</td>
</tr>
<tr>
<td>→ Anterior pituitary</td>
</tr>
<tr>
<td>→ Granulosa cells</td>
</tr>
<tr>
<td>→ Sertoli cells</td>
</tr>
<tr>
<td>→ 3-4 min</td>
</tr>
</tbody>
</table>

• Hormones and Receptors:

  **Hormones:**
  → GnRH
  → LH
  → FSH
  → hCG

  **Receptors:**
  → G-protein coupled
  → membrane receptors

  → Nuclear Receptors
  → present in cytoplasm

**Synthetic LH and FSH:**

• Obtained from urine of post-menopausal ♀
  known as human menopausal Gonadotropin (HMG)

• Has 75 IU of LH and 75 IU of FSH

• Uses: Ovulation induction
  1. Multi-fetal pregnancy = 30%
  2. Ovarian Hyper Stimulation Syndrome (OHSS) = < 5%
Estrogen and progesterone

Estrogen
→ Natural Estrogen
→ \( C_{E} \)

→ Forms of naturals:
\( E_1 : \) Estrone
\( E_2 : \) Estradiol
\( E_3 : \) Estriol
\( E_4 : \) Estriol

→ m.C Estrogen

Reproductive menopause Pregnancy
Age \( \downarrow \) \( \downarrow \) \( \downarrow \) \( \downarrow \) \( \downarrow \) \( \downarrow \)
\( E_1 \) \( E_2 \) \( E_3 \) \( E_4 \)
→ most specific in pregnancy: \( E_1 \)
→ Ratio \( E_1 : E_2 = 2:1 \) in PCOS, this ratio is reversed.
→ Potency wise:
\( E_4 > E_3 > E_2 > E_1 \)

* Sources:
1. Granulosa cells
2. Theca cells
3. Corpus luteum
4. Placenta (uses fetal DHEA)

* Binds to:
→ Sex Hormone Binding Globulin (SHBG) mainly
→ Albumin Free Estrogen: 1%

Progesterone
→ Natural progesterone
→ \( C_{P} \)

→ Synthetic progesterone
→ \( C_{P} \)

Androgens are also \( C_{A} \)

→ Synthetic progesterones
→ 1st generation
→ 2nd generation
→ 3rd generation
→ 4th generation

1st generation
| As
2nd generation
| generation ↑,
3rd generation
| Androgenic side effects ↓
4th generation
→ Least Androgenic side effects seen with 3rd generation progesterone

→ 4th generation progesterones are Anti-androgenic
Eg: Drospirenone cyproterone acetate

* Sources:
1. Granulosa cells
2. Corpus luteum
3. Placenta.

1. Do not bind to SHBG
2. Binds to:
→ Albumin
→ Cortisol Binding protein
Free progesterone: 2%
**Effects of estrogen and progesterone**

**Estrogen**
- On uterus
  - Growth of non-pregnant uterus
- On Endometrium
  - Proliferates

**Progesterone**
- Growth of pregnant uterus
- 1. Stops proliferation
  2. Secretory action (Decidualisation)
    - Supports endometrium
  3. If progesterone is given for a long time
    - Endometrial Atrophy
**Endometrial Biopsy**

1st Half of cycle: At ovulation 2nd Half of cycle

- Glands:
  - Short
  - Simple
  - Cystic
  - Tubular

- Glands begin to coil
- Appearance of subnuclear vacuoles

↓ 1st sign of ovulation on Endometrial Biopsy

↓ Subnuclear vacuoles

- Corkscrew appearance of glands or sawtooth appearance

I. Filled with secretion

- Cervical mucus:
  - Thin, watery, copious, elastic can be stretched between fingers → Spinnbarkeit
  - Under microscope: Ferning
    → due to ↑ Estrogen
    → Na
    → Cl
  - Appears by day 8 of cycle

- Thick viscous
  - Scanty
  - Breaks on stretching
  - Tack

→ Ferning is absent
→ Ferning disappears by Day-18 of cycle
Effects of estrogen and progesterone on vaginal epithelium

**Estrogen**
- Vaginal epithelium:
  - → Estrogen predominates
  - → Superficial cells
- Pink coloured cells (Eosinophilic)
- Pyknotic nuclei
- Predominates in presence of estrogen
- 1st half of cycle

**Progesterone**
- No Hormone predominates
- → Basal / parabasal cells
- Intermediate cells
- Blue coloured cells (basophilic)
- Outlines are not distinct
- Big nucleus
- Predominates when no hormone is predominantly
- Menopause
- Post partum
- 2nd half of cycle
- Pregnancy

**Normal Cervical Cells**

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with marrow edition 4 videos.
Important points

1. Sample for Hormonal study should be taken from: Lateral wall of vagina (upper 1/3rd part)

2. Sample for cytological study should be taken from: Posterior wall of vagina.

3. Maturation index

\[ a : b : c \]

- Parabasal / Intermediate cells
- Basal cells
- Superficial cells

- Maturation index (MI)
  - At pregnancy: \(0 : 95 : 5\)
  - Postpartum: \(100 : 0 : 0\)
  - Menopause: \(100 : 0 : 0\)
  - Secretory phase: \(0 : 70 : 30\)
  - Pre-ovulatory phase: \(0 : 40 : 60\)

4. Karyotypic index / Cornification index
  - No. of cornified squamous cells (due to estrogen)

Other effects of estrogen and progesterone

<table>
<thead>
<tr>
<th>Estrogen</th>
<th>Progesterone</th>
</tr>
</thead>
<tbody>
<tr>
<td>* On Fallopian Tube</td>
<td>→ ↑ motility</td>
</tr>
<tr>
<td></td>
<td>→ ↓ secretions</td>
</tr>
<tr>
<td>* On Breast</td>
<td>→ Ductal development</td>
</tr>
<tr>
<td>* On saltwater</td>
<td>→ retention</td>
</tr>
</tbody>
</table>
• On lipid profile
  → ↑ HDL
  ↑ Triglycerides
  ↓ LDL
  ↓ Cholesterol
  :: cardio protective
  → ↓ HDL
  ↑ LDL

Effects:
1. Smooth muscle Relaxant
2. ↑ Basal Body Temperature
   0.2 to 0.5°C
   or
   0.4 to 0.8°F

• On bones:
  → ↑ Bone mass
  → Epiphyseal closure

• On clotting factors:
  → ↑ Clotting factors
    3, 7, 8, 10
    :: Absolutely contraindicated in Thromboembolism, stroke, CAD

• Effect on FSH and LH
  → Negative effect on FSH
  → Estrogen
    Low conc → negative effect on LH
    High conc → positive effect on LH

  → Progesterone
    At ↓ concentration → Positive effect on FSH, LH
    At ↑ concentration → Negative effect on FSH, LH

USG appearance of endometrium

• USG appearance of endometrium in different phases of cycle
  Proliferative phase:
  → Endometrium appears as thin white line
At ovulation:
→ Trilaminar appearance or triple layered appearance

Secretory phase:
→ Thick white endometrium with a posterior enhancement

Proliferative phase

At ovulation

secretory phase
HORMONES IN GYNAECOLOGY – 2

Drugs related to estrogen

- SERM
  - act as agonist
  - antagonist
- Estrogen Antagonist

- SERM

- Clomiphene citrate
  - ovulation inducing drug
- Tamoxifen
  - management of Breast cancer
- Raloxifene
  - managing osteoporosis
  - S/E: Hot flushes
- Omeloxifene
  - Active ingredient of centchroman /'saheli'

1. Breast
   → Antagonist
   → DOC for treatment of ER + Breast cancer
2. Lipid profile
   → Agonist
3. Bone
   → Agonist
   → used in management of osteoporosis
4. Clotting factors
   → Agonist
5. Uterine Endometrium
   → Agonist
   → Risk factor for endometrial cancer
   → S/E: Hot flushes

Obstetrics & Gynaecology • v2.0 • Marrow 4.0 • 2020
• FDA Approved: Bazedoxifene + Estrogen
  \[ \rightarrow \text{Antagonist of Estrogen} \quad \rightarrow \text{Prevention against:} \]
  \[ \text{on endometrium} \quad \text{1. Osteoporosis} \quad \text{2. Hot Flushes} \]

**Estrogen antagonist and hyper estrogenic conditions**

- Progestrone (cheap): 1st line
- DOC: Continuous GnRH
- Danazol
  - Should not be used in young female, as it has androgenic side effects (hirsutism)
- Gestrinone
- Androgens \( \xrightarrow{\text{Aromatase}} \) Estrogens
  \[ \xrightarrow{\text{Letrozole}} \]
  (Aromatase inhibitor)

**Hyper estrogenic conditions:**
1. Endometriosis
2. Fibroid
3. Endometrial cancer
4. Ovarian cancer

**Risk Factors**
1. Nulliparous ♀
2. Obese ♀ (except endometriosis)
3. Early menarche
4. Late menopause

**Protective Factors**
1. Multiparity
2. Pregnancy
3. Physical exercise
4. Smoking (inhibits aromatase)
Drugs related to progesterone

- Selective progesterone receptor modulator drug
  - Ulipristal (Ella)
- Progesterone antagonist
  - Mifepristone/RU486

- Emergency contraception
- Fibroid

- Contraindication - Pregnancy
  - Liver disease
HIRSUTISM

Androgens in females

- Androstenedione
- Testosterone
- DHEA and DHEA-S

Source:
- Ovary (50%)
- Adrenal gland (50%)
- Minor androgen formed by ovary

DHEA: major source
DHEA-S: Exclusively produced by Adrenals

Testosterone in females:
- Source → 25% - Ovary
  - 25% - Adrenal glands
  - 50% - Androstenedione

Testosterone
[2nd most potent androgen] \[\text{5a reductase}\] → Dihydrotestosterone
[most potent androgen]

- Normal: 20-80 ng/ml
- Binds to SHBG (Sex Hormone Binding Globulin)
  - Albumin
- End product: Oxysteroid

SHBG

↑

Estrogen

↓

All - Androgens
India. - Insulin
P - Progestin
G - Growth hormone
Council - Cortisol
### Hirsutism, virilisation, hypertrichosis

<table>
<thead>
<tr>
<th>Hirsutism</th>
<th>Virilisation</th>
<th>Hypertrichosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excessive growth of male pattern / sexual hair</td>
<td>Entire spectrum of changes in a female exposed to androgens includes: Hirsutism Deepening of voice ↑ muscle mass clitoromegaly, Breast atrophy</td>
<td>Excessive growth of non-sexual hair all over the body</td>
</tr>
<tr>
<td>Scoring: Ferriman Gallwey score 11 sites Score: 0-4 → At each site modified Ferriman Gallwey score 9 sites (forearm, legs excluded) Score 0-4 Total score- ≥ 8: mild Hirsutism ≥ 15: Severe Hirsutism</td>
<td>Prader score: 0 → Female 5 → virilization present</td>
<td></td>
</tr>
</tbody>
</table>
Work up in a patient of hirsutism

Patient of hirsutism

↓

1st test: Androgen levels

↓

Normal

↓

Idiopathic hirsutism

↓

Testosterone

↓

DHEA &
DHEA-S

↓

Androgen secreting adrenal tumours

↓

≤ 200 ng/dl

↓

≥ 200 ng/dl

↓

PCOS (Rotterdam criteria)

↓

+ signs of virilization

↓

+ USG: ovarian mass

↓

Androgen producing tumour of ovary

↓

Late onset CAH (Congenital Adrenal Hyperplasia)

* CAH at the onset of puberty

* Screening test: 17-OH-progesterone

↓

<300 ng

↓

300-800 ng

↓

≥ 800 ng

↓

Rules out CAH

↓

ACTH stimulation test (Diagnostic test)

↓

Early onset CAH

* more common

* Female exposed to androgen in intra-uterine life

↓

Late onset CAH

* Less common

* Female exposed to excessive androgen at puberty
Management of hirsutism

OCP: OCPs

DOC: 

- Estrogen
- Progesterone

\[ \text{Estrogen} + \text{Progesterone} \]

\[ \uparrow \text{Estrogen} \]

\[ \downarrow \text{Free testosterone levels} \]

\[ \downarrow \text{Androgen from theca cells} \]

\[ \downarrow \text{Negative feedback on LH} \]

Normal: in females - Free testosterone \( \rightarrow 1\% \)
males - Free testosterone \( \rightarrow 2\% \)

In hirsutism: Free testosterone in females \( \uparrow \)

Note:
- 3rd or 4th generation progesterone used as OCP to treat hirsutism
  - E.g. Diane 35
  - Yasmin

Treatment

\[ \text{OCP} \times 6 \text{ months} \]

\[ \downarrow \text{No response} \]

\[ \downarrow \text{Anti-androgen drugs} \]

- Spironolactone (best)
- Cyproterone acetate
- Flutamide
- Finasteride

\[ \downarrow \text{No response} \]

\[ \text{GnRH} \]
In post menopausal women - Efflornithine cream
in young females - Laser removal of hair

Note:
Danazol → Cause hirsutism as adverse effect
∴ never used

MC cause of hirsutism

↓
In a young female

PCOS > Idiopathic hirsutism

Rapid onset hirsutism in a young female

Androgen producing tumour of ovary
PCOS

Polycystic Ovary Syndrome [PCOS]

- Aka Stein Leventhal syndrome
- Pathology: Excessive androgen production from ovary
- Androgen level: <400 nanogram

Features

- Hirsutism
- Male pattern baldness
- $\uparrow_{\text{LDL}}$
- $\downarrow_{\text{HDL}}$
- Heart disease

- In adipose tissue:
  - Excessive androgens convert into $\rightarrow$ estrogen ($e_i$)
  - Enzyme: Aromatase
- PCOS is mc in obese female
- In PCOS $e_a : e_i = 1 : a$ [Normal - $e_a : e_i = a : 1$]

$\uparrow_{\text{Estrogen}}$

- Negative feedback on FSH $\downarrow$
  - $\downarrow_{\text{FSH}}$
- Positive feedback on LH $\downarrow$
  - $\uparrow_{\text{LH}}$

$\uparrow_{\text{Chance for:}}$

1. Endometrial Hyperplasia
2. Endometrial cancer
3. Breast cancer
4. ± Ovarian cancer

- In PCOS:
  - $\text{FSH : LH} = 1 : 2 \ or \ 1 : 3$ (Normal - FSH < LH)
  - Test done on: Day 2 to Day 4 of menstrual cycle
- After puberty granulosa cells of small follicles secrete \( \rightarrow \) Anti mullerian hormone

- In PCOS number of small follicles increased in ovary \( \rightarrow \) \( \uparrow \) Anti mullerian hormone

- \( \rightarrow \) no corpus luteum, progesterone causes
  1. Secondary Amenorrhea.
  2. Anovulatory / Irregular cycles (Painless)

**Insulin resistance**

manifested as:

- Acanthosis Nigricans \( \rightarrow \) Hyper pigmented skin in axillary area or nape of neck
• Investigations:

  Serum fasting glucose levels
  \[
  \frac{< 4.5 \text{ in insulin resistance}}{\text{Serum fasting insulin levels}}
  \]

  a) Serum insulin levels > 25 IU

• Complications:
  - Diabetes mellitus

ACOG Recommendation:

In PCOS patients with 75 gm of glucose levels → a hourly glucose tolerance test recommended

Hormones in PCOS

<table>
<thead>
<tr>
<th>Increased</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>LH</td>
<td>FSH</td>
</tr>
<tr>
<td>Androgens</td>
<td>Progesterone</td>
</tr>
<tr>
<td>Estrogen (E₂)</td>
<td>HDL</td>
</tr>
<tr>
<td>LDL</td>
<td>Sex hormone binding globulin (SHBG)</td>
</tr>
<tr>
<td>Anti Mullerian Hormone Insulin</td>
<td></td>
</tr>
</tbody>
</table>

• In PCOS TSH, Prolactin, inhibin remains normal (↑ Prolactin, inhibin → only in few patients)

Diagnosis of PCOS

Rotterdam criteria:

• Any 2 of the following should be there to diagnose PCOS:
  
  1) Hyperandrogenism → Clinically manifests as hirsutism → Biochemically ↑ androgen levels

  a) Ovulatory dysfunction
     - Amenorrhea
     - Irregular cycles
     - Menometrorrhagia

  b) USG evidence of PCOS: Either in one or both ovary
     1) ≥ 12 Follicles in ovary
     2) Follicles should be <1 cm (2-9 mm) in size
     3) Volume of ovary ≥ 10 cc
- Obesity not a diagnostic criteria.
- LDL:HDL ratio not a diagnostic criteria.
- PCOS can be seen in thin females
- PCOS mc in reproductive age group, but can be seen in prepubertal age groups (rare)

Risk factor for PCOS in prepubertal females:
1) Low birth weight
2) Early onset of Adrenarche (before 6 years)
3) Obesity with acanthosis nigricans
4) Heterosexual precocious puberty

- In PCOS:
  - Familial inheritance in seen
  - mc gene mutation $\rightarrow$ Cyp 11 gene
- Necklace pattern on USG is not diagnostic of PCOS
- Ovaries can appear normal on USG

**Syndrome associated with PCOS**

1) HAIR-AN Syndrome

Features:
- Hyperandrogenism
- Insulin resistance
- Acanthosis nigricans

2) metabolic X Syndrome:

Any 3 of the following 5 should be present to diagnose metabolic X syndrome:

1) Abdominal obesity
   - [waist circumference $> 88$ cm or $35$ inches]
2) Triglycerides $> 150$ mg/dl
3) HDL $< 50$ mg/dl
4) BP $\geq 130/85$ mmHg
5) Fasting blood sugar $\rightarrow 110-126$ mg/dl
   - a hour post prandial sugar $\rightarrow 140-199$ mg/dl
Complications of PCOS

Short term complications
1. Infertility
2. Hirsutism
3. Irregular cycles

Long term complications
1. Heart disease
2. Endometrial carcinoma
3. Breast cancer
4. \pm Ovarian cancer
5. Diabetes mellitus
6. Obesity causes
   - Sleep apnea syndrome
   - Metabolic Syndrome
   - Depression
7. Non alcoholic steatohepatitis

* Osteoporosis does not occur due to \( \uparrow \) estrogen

Management of PCOS

1. 1st step: weight reduction

<table>
<thead>
<tr>
<th>Complications</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Insulin Resistance</td>
<td>DOC: metformin</td>
</tr>
<tr>
<td></td>
<td>* mc complication: GI involvement</td>
</tr>
<tr>
<td></td>
<td>* most dangerous: Lactic acidosis</td>
</tr>
<tr>
<td></td>
<td>* Non teratogenic</td>
</tr>
<tr>
<td>2) Irregular cycles</td>
<td>DOC: OCPs (Progesterone should be 3rd/4th generation)</td>
</tr>
<tr>
<td>3) Hirsutism</td>
<td>DOC: OCPs (Progesterone should be 3rd/4th generation)</td>
</tr>
</tbody>
</table>
4) Infertility  
[due to anovulation]  
* Doc in PCOS  
  Letrozole > clomiphene citrate  
* Doc in obese &  
  Insulin resistance  
  Letrozole > clomiphene citrate  
  +  
  Metformin  
* Doc in PCOS patients with  
  ↑ Prolactin levels  
  Clomiphene citrate  
  +  
  Bromocriptine

1° line of management  
* Letrozole (DOC)  
* Clomiphene citrate  
  (2° best drug)  
* Bromocriptine  
  (Ovulation including drug)

2° line management  
1) Human menopausal  
  gonadotropin [HMG]  
2) Laparoscopic ovarian  
  drilling (theca cells of  
  ovary is destroyed)

3° line management  
  synthetic GnRH in pulsatile  
  manner

Side effects of ovulation inducing drugs:  
1) multiple pregnancy  
   a) OHSS  
   b) Menopause like symptoms

Laparoscopic Ovarian drilling:  
- Advantage: No risk of multiple pregnancy or OHSS  
- Disadvantage: Premature ovarian failure

---

Clomiphene citrate

- Selective estrogen receptor modulator (SERM)  
- 2 Isomers: Enclomiphene

  Zuclomiphene → Antagonist of estrogen  
  ↓ levels of estrogen  
  Negative feedback on FSH lost  
  ↑ FSH levels  
  Follicles starts maturing
- Prerequisite for clomiphene citrate: intact hypothalamic pituitary axis
- Side effects:
  1) Menopause-like symptoms
     a) Multiple pregnancy
- Initial dose: 50mg/day
- Maximum dose: 100mg/day
- Schedule: From Day 3 to 6 or Day 5 to 9 of the cycle.
- Follicular monitoring done from Day 10 till the follicles becomes 18-20mm in size (size increase by 2-3 mm/day)
- Injection hCG → Acts like ovulation trigger
  Ovulation occurs after 32-36 hours
- Ovulation occurs in 60-70% patients on clomiphene citrate
  * Pregnancy rate is less due to ↓ Estrogen

```
Thin endometrium  Thick cervical mucus
(does not favour implantation)
```

Side effects:
  1) Menopause-like symptoms → Hot flushes (due to ↓ Estrogen)
  2) ↑ Ovulation → ↑ Chances of ovarian cancer
      → ↑ Multiple pregnancy
      → ↑ OHSS
- If visual symptoms present → Stop clomiphene citrate

**Letrozole v/s clomiphene citrate**

<table>
<thead>
<tr>
<th>Letrozole</th>
<th>Clomiphene citrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Aromatase inhibitor</td>
<td>• Antagonist to estrogen</td>
</tr>
<tr>
<td>• Prevents Androgen</td>
<td>• ↓ Estrogen levels</td>
</tr>
<tr>
<td>• Conversion to estrogen</td>
<td>• Thick cervical mucus</td>
</tr>
<tr>
<td>• Mild ↓ Estrogen</td>
<td>• Thin endometrium</td>
</tr>
<tr>
<td>• Cervical mucus not thick</td>
<td>• ↓ Uterine blood flow</td>
</tr>
<tr>
<td>• Thick endometrium</td>
<td></td>
</tr>
<tr>
<td>• ↑ Uterine blood flow</td>
<td></td>
</tr>
</tbody>
</table>

01:10:50

* Obstetrics & Gynaecology v2.0 * Marrow 4.0 * 2020
ENDOMETRIOSIS

Endometriosis

- Definition
  Endometrial tissue (Glands + Stroma) outside uterine cavity

- Site
  - Most common site – Ovary
  - Ovary > Pouch of Douglas > Uterosacral Ligament > Broad Ligament > Fallopian tube
  - Can occur anywhere in body
  - Rarest site – CNS
  - Can occur in scars of previous surgeries → Scar endometriosis
    * Caesarean section
    * Episiotomy
    * Pelvic floor repair
    * Myomectomy

- Theories related to Endometriosis
  i) Sampson’s theory of implantation
    - Most commonly accepted theory
    - Theory of retrograde menstruation
  iii) Halban – Theory of metastasis.

- Risk factors & Protective factors related to excessive estrogen
  - Risk factors
    * Obesity
    * Nulliparous
    * Early menarche
    * Late menopause
    * Late marriage & late child birth
    * High socioeconomic status
  - Protective factors
    * Multiparity
    * Pregnancy
    * Smoking
    * Physical exercise
Note:
- Gene involved - K-RAS
- Most common age - ≥ 30 yrs
- Endometriosis in puberty → Rule out Mullerian malformation

Endometriosis - clinical presentation

- Most common symptom → Pain
- 2nd most common → Infertility
- 3rd most common → Endometrioma / Chocolate cyst in ovary
- 4th most common → Menstrual symptoms.

1) Pain:
- Most common symptom
- Most common type → 2° dysmenorrhoea
  2° dysmenorrhoea > Chronic pelvic pain > Dyspareunia > Low back ache
- Reason for pain:
  a) Congestion
  b) Prostaglandins
- Pain is related to depth of lesion

Management of pain in endometriosis

<table>
<thead>
<tr>
<th>Mild / Minimal</th>
<th>Moderate / Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Treated like 1° dysmenorrhoea.</td>
<td>- Treat the disease pathology</td>
</tr>
<tr>
<td>1. NSAID</td>
<td>- Aim: to ↓ Estrogen</td>
</tr>
<tr>
<td>2. OCP [Cycle becomes anovulatory]</td>
<td>- Drugs</td>
</tr>
<tr>
<td></td>
<td>1. Progesterone (Cheapest)</td>
</tr>
<tr>
<td></td>
<td>2. GnRH (Best) - given continuously</td>
</tr>
<tr>
<td></td>
<td>3. Letrozole [Aromatase inhibitor]</td>
</tr>
<tr>
<td></td>
<td>4. Danazol → Hirsutism</td>
</tr>
<tr>
<td></td>
<td>. . . not preferred</td>
</tr>
<tr>
<td></td>
<td>5. Gestrinone</td>
</tr>
<tr>
<td></td>
<td>6. mifepristone → Endometrial Atrophy</td>
</tr>
</tbody>
</table>

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Other symptoms in endometriosis

i) Infertility:
   - A most common symptom in endometriosis
   - Infertility
     - Mild infertility
     - Moderate/Severe
       - Reason:
         - Inflammation → ↑ Cytokines
         - Hormonal imbalance (↑ estrogen)
         - Endometriotic tissue → Embryotoxic
         - Management:
           - Management of unexplained infertility
           - Clomiphene citrate + IUI
           - [Intrauterine insemination]

ii) Endometrioma:
   - Most common site of endometriosis → Ovary [30 - 40% cases]
   - Bilateral involvement of ovary
   - Ovary filled with blood
     - Hemosiderin ⇒ Chocolatey / Tarry blood ⇒ Chocolate cyst
   - Cyst is lined by pseudo xanthoma cells
   - Cyst size ≤ 1 cm:
     - Management
       - Cannot be managed medically
       - Surgical management → Laparoscopic surgery
         - < 3 cm → Laparoscopic electrocoagulation
         - ≥ 3 cm → Laparoscopic cystectomy
• Sites where endometriosis cannot be medically managed
  1. Ovary
     a. Bowel
  3. Urinary system

ii) Menstrual symptoms
   - Menorrhagia (↑ estrogen → endometrial proliferation → shedding of endometrium)

One liners

1. Clinical triad:
   a° dysmenorrhoea.
   Infertility Dyspareunia.

2. Per Vaginal examination
   i) Fixed retroverted uterus
   ii) Adnexal mass
   iii) Nodules on uterosacral ligament
       - Cobblestone appearance

Endometriosis - investigations

1° 1st investigation
   • TVS [Trans Vaginal Sonography] → Chocolate Cyst
     [Ground Glass appearance]
i) IOC [Investigation of Choice]
   - Laparoscopy

       Ovary                       Peritoneum

       Chocolate cyst             Powder Burn / Gun shot appearance

       Red cystic lesions        White Scary lesions

       Small - blue - black cystic lesions

       Allen-Master’s Syndrome (Peritoneal defects)

---

ii) Gold standard
   - HPE [Histological Examination]

Note:
   - CA-125 level is ↑↑ in endometriosis.

Endometriosis - management

i) Medical management
   - Main symptom of endometriosis - Pain ⇒ Managed medically

ii) Surgical management
   - Indications:
     - If patient has intolerance to pain
- Not responding to medical management
- Ovary (chocolate cyst) / Bowel / Urinary system involved

- Techniques:
  - Adhesiolysis
  - Fulguration of lesions
  - LUNA - Laproscopic Uterosacral Nerve Ablation
  - TAH (Total Abdominal Hysterectomy) + BSO (Bilateral Salpingo - oophorectomy)

---

**Adenomyosis**

- Endometrial tissue is present inside myometrium
  - earlier known as endometriosis interna
- Age: ≥ 40yrs
- More common in multipara.
- Most common symptoms - a° dysmenorrhoea + menorrhagia

I. Investigations
- 1st investigation - TVS
- IOC - MRI
- Gold standard - HPE

Findings

- On TVS
  i) myometrium → Salt & Pepper appearance [Heterogenous appearance]
  ii) Myometrial cyst
  iii) Subendometrial striations → Venetian blind appearance

- On MRI
  i) Thickness of junctional zone between endometrium & myometrium increases [≥ 10mm]
  ii) Normal, thickness is 5mm

- On HPE
  i) Endometrial glands seen inside the myometrium
  [glands ≥ 0.5 mm deep, below the junctional zone]
  ii) Myometrial hyperplasia & hypertrophy
• Per Vaginal
  i) Symmetrically enlarged uterus
    size - 10 - 12 weeks pregnant uterus size
  ii) Halban sign - Tender uterus
  iii) Mobile uterus

a. Management
   TAH + BSO
FIBROIDS

- mC pelvic tumour in female
- mC solid benign tumour in female
- Smooth muscle benign tumour of uterus
- Estrogen + progesterone dependent tumour
- mC age = 35-45 years
- monoclonal in origin (originates from single myocyte)
- 40% fibroids → Genetic / Chromosomal abnormality
  → mC chromosomal abnormality: Translocation (b/w chr 12 & 14)
  → Followed by: Deletion of chromosome 7
- Gene mutations present in fibroid: MED12 and HMGA2 gene
- Familial inheritance seen (2.5 times ↑ risk with 1° relative)
- Estrogen dependent:
  mC → Nulliparous female
  mC → Obese female
  Smoking → Protective
- mC in African female
- Effect on fibroids:
  → OCP: no effect
  → Pregnancy: no effect
  → Menopause: ↓ size (if size ↑, suspect malignancy)

Classification of fibroids

Types

- mC intruterine
  → Intramural (mC)
  (interstitial)
  → Submucous (a° mC)
  → Subserous
- Extra uterine
Submucous fibroids are further classified into:

- Type 0 (Completely inside uterine cavity)
- Type 1: ≥ 50% in uterine cavity, < 50% in myometrium
- Type 2: ≥ 50% in myometrium, < 50% in uterine cavity

→ Type 0 and Type 1: Removed Hysteroscopically
→ Type 2: Cannot be removed Hysteroscopically

**True and pseudo-broad ligament fibroid**

- True Broad ligament Fibroid
  - Lies lateral to ureter
  - Non-pedunculated
  - Never covered by pseudocapsule
- Pseudo Broad Ligament Fibroid
  - Lies medial to ureter
  - Pedunculated
  - Covered by a pseudocapsule

→ Can get polycythemia instead of anemia.

Important points on Fibroids:

- m.C variety of uterine fibroid
- To begin with all fibroids are
- m.C fibroid causing symptoms
- m.C fibroid causing abortion
- m.C fibroid causing infertility
- m.C fibroid to undergo malignant transformation
- m.C fibroid to undergo torsion

- Intramural Abroid
- Submucous Abroid
- Submucous Abroid
- Submucous Abroid
- Submucous Abroid
- Pedunculated subserous Abroid
- MC fibroid to undergo calcification:
  Pedunculated subserous fibroid → Subserous fibroid on calcification
  * Womb stone
  * Popcorn calcification

- MC fibroid to cause urinary symptoms: Cervical fibroid
- MC fibroid leading to uterine inversion:

- Wandering Fibroid: Pedunculated subserous fibroid with no connection to uterus

**Structure of a fibroid**

- Rubber consistency
- Whorled appearance
- Covered by pseudocapsule

- Pseudocapsule → Blood vessels supplying fibroid are present in pseudocapsule
- Fibroid → Blood vessels

- Most vascular part
  - Periphery of fibroid
  - Calcification begins from periphery

- Least vascular part
  - Center of fibroid
  - Degeneration begins from center

- No mitotic activity is seen in a fibroid
- If mitotic activity is present (>10 mitosis/HPF)
  - Malignant
- m.C degeneration: Hyaline degeneration
- Calcareous degeneration / Calcification: (m.C) subserous fibroid
- Cystic degeneration in post-menopausal female
- Red degeneration: Specific in pregnancy (2nd trimester)
- Least common change: Malignant transformation (0.2-0.5%)
- M.C malignancy: Leiomyosarcoma
- M.C Fibroid: Submucous fibroid
- Histopathological examination (HPE)
  → Loses its pseudocapsule
  → Whorled pattern lost
  → Mitosis present
- Clinically: T size, painful (signs of malignant transformation)

Red degeneration of fibroid

- Specific to pregnancy (m.C → 2nd trimester)
- Aseptic thrombosis in the blood vessels which supply fibroid
  ↓
  Aseptic Necrosis of fibroid
  ↓
  C/F → pain in abdomen, nausea, vomiting
  ↓
  On HPE: Salmon pink in colour

- Investigation:
  → Aseptic but still presents with: Fever
     ↑ WBC
     ↑ ESR
     Reactionary changes

- Management:
  ↓
  Do's
  → Conservative management
  → Analgesics
  → Anti emetics
  → Rest
  ↓
  Don't's
  → No Antibiotics
  → Do not terminate pregnancy
  → Do not do myomectomy
Clinical presentations of fibroid

* MC presentation: Asymptomatic
* MC symptom: Menstrual symptoms
  - MC if metrorrhagia (irregular bleeding)
  - Never leads to amenorrhea
  - Menorrhagia
  - Fibroid polyp

* Pressure symptoms:
  - On vein / Lymphatics: Varicose veins / Edema
  - Intestine & rectum: Dyspepsia / Constipation
  - Urinary retention

* Fibroid doesn't lead to pain in abdomen

* If pain is the primary complaint:
  - Torsion
  - Red degeneration
  - Malignant transformation

* Fibroids can lead to dysmenorrhea
* Fibroids can never lead to secondary dysperunia
  - i. Submucous fibroids – interferes with the ascent of the sperms
  - ii. If fibroid near to the angle of uterus – blocks tubal ostia
  - iii. Hinders with implantation

* 3% cases → Infertility

Effects of fibroid on pregnancy

1. Infertility
2. ↑ Abortion
3. ↑ Preterm labour
4. ↑ Abruptio placenta
5. Malpresentation
6. ↑ PPH
7. ↑ Subinvolution
Investigation and differential diagnosis of fibroids

- Investigation of choice for fibroid: USG (TVS)
- For small submucous fibroid
  \[\text{Hysteroscopy (invasive test)}\]
- Saline infusion sonography
- HSG (Filling Defect) for submucosal fibroids

- Differential diagnosis of Fibroid: Adenomyosis

1. M.C. complaint: menorrhagia
2. Age: 35–45 years (Reproductive age)
3. Nulliparous
4. Irregularly enlarged uterus
5. Size of uterus: 16–20 weeks pregnant uterus
6. Uterus is non-tender
7. IOC: USG

1. M.C.: menorrhagia + dysmenorrhea
2. Age: \(\geq 45\) years
3. Multiparous
4. Symmetrically enlarged uterus
5. Size of uterus: 10–12 weeks pregnant uterus
6. Tender uterus \(\rightarrow\) Halban sign
7. IOC: MRI
Management of fibroid

Asymptomatic
- Follow up by annual pelvic examination (irrespective of their size)
- Indication for surgery in asymptomatic fibroid
  - Torsion of pedunculated fibroid
  - Size of fibroid increasing
  - Suspecting malignancy

Symptomatic
- Best surgical management
- Surgery
- Medical management
- Indications:
  - Patient refuses surgery
  - Contraindication to surgery
  - Correct anemia before surgery
  - Reduce size of fibroid before surgery
  - Patient nearing menopause
- Uterine Artery Embolisation (UAE)
- Magnetic Resonance Imaging (MRI) guided High Intensity Focused Ultrasound (MIRHIFU)

Only in female who have completed their family

Surgical management of fibroid

Remove uterus + Fibroid
- Type 1 Hysterectomy / Total Abdominal Hysterectomy (TAH)

Indications for TAH
- Age of patient ≥ 40 years
- Family complete
- Excessive bleeding at the time of myomectomy
- Fibroid is associated with malignancy

Remove fibroid
- Myomectomy
  - Indications:
    - 1. If family not complete
    - 2. Case of recurrent abortion with fibroids
    - 3. Case of infertility with fibroids
Myomectomy

* Prerequisites:
  1. Anemia should be corrected
  2. Arrange for blood
  3. Consent for TAH
  4. Husband’s semen analysis should be normal
  5. Hysteroscopy + endometrial biopsy if in case of irregular cycles to rule out polyp / endometrial cancer

* Timing:
  → Post menstrual phase
  → Never to be done during pregnancy

* methods to ↓ blood loss during myomectomy:
  1. Pre operatively = GnRH → ↓ vascularity of fibroid
  2. Time: immediate post menstrual phase
  3. Hypotensive anesthesia
  4. Inject vasopressin to serosa overlying the myoma
  5. Tie Torniquet to the uterine Artery
     (≥ 45 min; Release intermittently)

Bonneys myoma screw
Bonneys myoma clamp

used to hold fibroid
used to release the blood supply intermittently

Myomectomy - contraindications & routes

contraindications
1. Large broad ligament fibroid
2. Small multiple fibroid in uterus
3. Associated with genital TB
4. During pregnancy
• Routes of myomectomy

- Abdominal / Laparoscopic myomectomy
- Hysteroscopic myomectomy

→ Laparoscopic myomectomy is preferred:
- ↓ Blood loss
- ↓ Morbidity
- ↓ Hospital stay
- Early resumption of normal activities

- Earlier: Only disadvantage of laparoscopic myomectomy
  ↓
  ↑ Chances of recurrence

→ Laparoscopic myomectomy:
  1. Intramural fibroid
  2. Subserous fibroid
  3. Type 2 submucous fibroid
  4. No. of fibroids < 4
  5. Size of fibroid < 10 cm

Hysteroscopy

- Used to visualise inside the uterine cavity
- Always use a distension media

↓
Diagnostic
- CO₂

Operative Hysteroscopy
- Fluid
  ↓
  Hypotonic Fluid
  (Fluid poor in electrolytes)
  5% → Mannitol
  3% → Sorbitol
  1.5% → Glycine
  ↓
  Isotonic Fluid
  (Rich in electrolyte)
  → Normal saline

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• m.C complication: Perforation
  → Fundal: Observation
  → Lateral: Immediate laparotomy
  → Posterior: Immediate laparotomy

• 2nd m.C complication: Fluid overload (Reported as fluid deficit)
  ↓
  → Hyponatremia
  ↓
  → Cerebral edema

• media
  Alert
  Hypotonic media: 500 ml 1000 ml
  Isotonic media: 1000 ml 2500 ml
  Stop procedure

• Patients who have completed their family:
  ↓
  Uterine Artery Embolisation
  → Via Femoral Artery using polyvinyl alcohol / gel foam
  → mRGIIFu
  → MRI room x 2-3 hours
  → High intensity ultrasound waves are focused on fibroid
  → Contraindications:
    • Size of fibroid > 10 cms
    • Size of weight > 24 weeks
    • Obstruction in path of rays: Bowel / foreign body

• Fibroid slowly - shrinks in size
• Not done in female planning to conceive in future
Medical management of fibroid

↓ Blood loss, but no effect on size of fibroid
1. Tranexemic Acid
2. Progesterone
3. OCP

↓ Size of fibroid, eventually ↓ blood loss

↓ Estrogen
- Continuous GnRH
- Letrozole
- Danazol
- Gestrinone

↓ Progesterone
- Ulipristal
- Mifepristone

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

FIGO classification of fibroid

Type:
0
1 } Sub mucous fibroid
2
3 } Intramural fibroid
4
5
6 } Subserous fibroid
7
8 → Extraterine fibroid = Broad ligament fibroid
MENOPAUSE

- Cessation of periods for 12 months at least, which is permanent
- Age: 51 years (mc)
  - In India → 47 years
- Period from 39 years to 51 years → Perimenopausal period
- Premature menopause → < 40 years
  - Late menopause → ≥ 55 years

Pathophysiology of menopause

- Pathophysiology:
  - ↓ activity of ovary
  - Ovarian failures
  - ↓ No more follicles in ovary
  - ↓ No granulosa cells
  - ↓ No Theca cells
  - ↓ No ovulation
  - ↓ Estrogen
  - ↓ Androgens
  - ↓ Progesterone
  - → Responsible for symptoms of menopause
  - ↓ Libido
  - Amenorrhea (for 2-12 months)
  - ↑ LH
  - mc estrogen in menopause = $E_1$
  - <20pg/ml - Diagnostic

Theca cells

- Androstenedione
- Testosterone
  - (only in granulosa cell)
  - Adipose tissue
  - Granulosa cells

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• Adrenal Gland: Androgen in Tissue → E1

Decreased estrogen in menopause

- Bone mass osteoporosis
- Dryness in vagina
- Negative feedback on FSH
- Sagging Heart disease
- Senile vaginitis
- FSH↑

Osteoblast

Osteoclast

Rank

Ligand

Bone resorption

Estrogen → OPG (Osteoprotegerin)

Diagnostic test / sine qua non for menopause

• ↑ FSH Levels = 240 μIU
• In a menopausal female: serum LH and serum FSH are raised
  Excreted through urine
  Human menopausal Synthetic LH + FSH
  Gonadotropin (HMG)
  - Multifetal pregnancy = 30%
  - Risk of Ovarian Hyper stimulation Syndrome = 5%

• Most characteristic symptom of menopause
  Hot Flushes (vasomotor symptoms)
  ↓ Estrogen
  Coincides with LH surge
  80% of menopausal females

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Management of menopause

- **Management**: Hormone Replacement Therapy (HRT)
  - Indicated in all menopausal females who are experiencing symptoms of menopause
  - Special indications:
    1. Premature menopause
    2. Menopause due to surgery or radiation
    3. Gonadal dysgenesis (e.g., Turner syndrome)
- **MC indication**: Hot flushes

- If female uterus is intact: Estrogen + Progesterone (HRT)
  - Oral / Transdermal
  - Protect from endometrial cancer

- If uterus is absent: Only Estrogen
  - **Standard Dose**  
    - Conjugated estrogen: 0.625 mg
    - Micronized estrogen: 1 mg
    - Ethinyl estradiol: 5 mcg
    - Medroxyprogesterone acetate: 5 mcg
  - **Low Dose**  
    - 0.3/0.4 mg
    - 0.5 mg
    - 2.5 mcg

- **Indications of Transdermal Estrogen patch in HRT**
  - ↑ Risk of Thromboembolism
  - Triglycerides
  - Obese with Metabolic X Syndrome

- HRT can also be used for:
  - Senile vaginitis
  - Osteoporosis

- HRT is not used to ↓ incidence of coronary heart disease

Management of osteoporosis: review

- **1st line management**: Non-Hormonal drugs
  - Bisphosphonate like Alendronate
- 2nd line management:
  - HRT → Uterus is intact: Estrogen + Progesterone
  → Uterus is absent: only Estrogen
  - Tibolone (estrogen + progesterone like activity)
  - SERM → Raloxifene

Osteoporosis
- ↓ Bone mass
  ↓
  ↑ Chance of fracture
  ↓
  M.C. = Vertebral compression fracture
  - Colle's Fracture
  - Fracture of femur neck

- Screening is done in female ≥65 years
  ↓
  DEXA scan
  - Measure bone mineral density
  Compare with
    ↓
    Female of her age
    ↓
    Z score
    Young female
    ↓
    T score

Management of osteoporosis - denosumab
& calcitonin

1. Denosumab
   (Should not be given to patients with ↓ Ca²⁺ levels)
2. Calcitonin
   ↓
   Salcalcitonin
   I.V./Nasal spray
3. Bisphosphonates
   - Alendronate
   - Ibandronate
   - Risedronate
   - Zoledronate → IV
   - 1st line of treatment of osteoporosis
   - Side effect: Gastritis
   - Lead to → Osteonecrosis of jaw (rare) in long term use

4. SERM / Estrogen
   - Raloxifene
     1. ↓ Fracture by 50%
     2. ↑ HDL
        ↓ LDL 10%
     3. ↓ Breast cancer
     4. ↓ No effect on uterine cancer
   - SERM: Bazedoxifene + Estrogen
      Approved by FDA
      osteoporosis + Hot flushes

Parathyroid hormone & drugs used in osteoporosis

- At low concentration parathyroid hormone stimulates osteoblast
  ↓ Bone formation
  At high concentrations - ↑ RANK ligand - stimulates osteoclasts
  ↓ Serum Ca²⁺

- Synthetic parathyroid hormone → Teriparatide
  - Acts on osteoblast
  - Low pulses / Amplitude
  - Can lead to osteosarcoma in bone
  - Should not be used ≥ 2 years
Classification of drugs used in osteoporosis

- Prevent
  - Calcium
    - Daily requirement of ca:
      - Female 31-50 years: 1000 mg
      - Female >51 years: 1200 mg
  - Vit D intake
    - Low risk: Female
      - 600 IU/day
    - High risk: Female
      - 800 IU/day

- Treating
  - Denosumab
  -Raloxifene
  - Bisphosphonate

- Both
  - HRT
  - Tibolone
  - Teriparatide
  - SERM

Hot flushes and senile vaginitis

- 1st line management: HRT
  - Uterus intact
  - Uterus absent
  - Estrogen + progesterone
  - Only estrogen

- 2nd line management:
  - Tibolone
  - SSRI → Fluoxetine, Venlafaxine
  - Clonidine α₂ Adrenergic agonist (Side effects: dry mouth, constipation)
  - Gabapentin
  - Phytoestrogen soy proteins, flax seed

- Raloxifene: Cannot be used to treat hot flushes
- SERM can be used: Ethinyl Estradiol + Bazedoxifene

Senile vaginitis

- Estrogen cream → Premarin for local application
- If symptom less → lubricants (K-Y jelly) can be used
ANATOMY OF FEMALE GENITAL TRACT: FALLOPIAN TUBE AND OVARY

Fallopian tube

![Diagram of Fallopian tube]

**Length of tube:** 7-12 cms (average: 10 cms) or 4 inches

<table>
<thead>
<tr>
<th>Part of tube</th>
<th>Important points</th>
</tr>
</thead>
</table>
| 1. Intramural part | 1.25 cm long (narrowest part)  
0.7 to 1 mm in diameter  
lacks longitudinal muscle  
fibres, made only of circular fibres  
anatomical sphincter |
| 2. Isthmus | 2.5 cm long  
1-2 mm in diameter and narrowest part  
physiological sphincter |
| 3. Ampulla | 5 cm long  
longest & widest part  
max. number of mucosal folds  
plicae |
| 4. Fimbriae | 1.25 cm long |

One liners:

- Fertilization occurs in $\rightarrow$ Ampulla
- Ectopic pregnancy m.c in $\rightarrow$ Ampulla
- Max plicae/mucosal folds $\rightarrow$ Ampulla
- Part cut during tubectomy $\rightarrow$ Isthmus
- Reversal of tubectomy best in $\rightarrow$ Isthmo-isthmic anastomosis
- TB causes block in $\rightarrow$ cornual end of tube
- Gonococcus causes block in $\rightarrow$ Fimbrial end of tube
Lining of the tube

Lining: Ciliated columnar epithelium
Malignant variety of CA: Adeno CA

Movement of zygote towards uterine cavity is due to:
1. Peristalsis of tube
2. Movement of cilia

Cells in fallopian tube:
- Ciliated columnar cells
- Secretory cells
- Peg cells
- Pyruvate secretions
- Provides nutrition to early zygote

Blood supply:
- Medial part → Uterine artery
- Lateral part → Ovarian artery

Lymphatic drainage:
- Para-aortic LN
- Along cornual end
- Along with round ligament
- Superficial inguinal LN

Nerve supply: T₁₁, T₁₂, L₁
In unruptured ectopic → Pain due to stretching of tube
T₁₁, T₁₂, L₁

Development:
- Unfused parts of Mullerian ducts
Ovary

Length: 2 to 5 cms
Width: 1.5 to 3 cms
Thickness: 0.5 to 1.5 cms
Weight: 5-10 gms
Volume: Average volume in reproductive age female: 1-5 cc
         Maximum volume → 20 cc
         Post-menopausal → 3-4 cc (average)
         Maximum → 10 cc

Location: Ovarian fossa (of waller) in lateral pelvic wall
In intrauterine life: Located near T₁₁ segment
         Ovary descends into pelvis with help of gubernaculum

* Uterus divides gubernaculum into 2 parts
  ↓
  Proximal part Distal part
  ↓
  Forms ovarian ligament Forms round ligament

Ligaments related to ovary

1. Ovarian ligament: connects ovary to cornua of uterus
2. Infundibulopelvic ligament / Suspensory ligament of ovary:
   * Connects ovary to pelvic side walls
   * Ovarian vessels and nerves present
3. Mesovarium: Part of broad ligament anterior to ovary
Relations of ovary

- Superior / Anterior: External iliac artery
- Posterior: Internal iliac artery and ureter
- Medial: Ovarian ligament
- Lateral: Infundibulopelvic ligament, obturator nerve

Ovarian tumour → Press obturator nerve
↓
Referred pain to medial side of thigh
Histology of ovary:
1. Germinal/surface epithelium
2. Tunica albuginea
3. Cortex
   - Germ cells
   - Follicular / Granulosa cells
4. Medulla
   a) Myoid like contractile cells
   b) Interstitial cells → Theca cells
   c) Hilus cells → Secrete androgen
5. Stroma
   + Blood vessels and nerves

Origin of the ovary

Arises from genital ridge

Hormones produced:
- Main: E₃ (estradiol)
- E₁ (estrone)
- Androstenedione
- Testosterone
- 17α – OH – progesterone
Blood supply: ovarian artery (branch of abdominal aorta)

Venous drainage:
- Right ovarian vein → Drains into IVC
- Left ovarian vein → Drain into left renal vein

Lymphatic drainage: Para-aortic LN
Nerve supply: Ovarian plexus
ANATOMY OF FEMALE GENITAL TRACT:
UTERUS AND CERVIX

Uterus

Shape of uterus - pyriform

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non-Pregnant</th>
<th>Pregnant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nulliparous</td>
<td>Multiparous</td>
</tr>
<tr>
<td>Weight</td>
<td>50-70 g</td>
<td>60-80 g</td>
</tr>
<tr>
<td>Volume</td>
<td>10 ml</td>
<td>10 ml</td>
</tr>
<tr>
<td>Length</td>
<td>6-8 cms</td>
<td>9-10 cms</td>
</tr>
</tbody>
</table>

Size of uterus in inches: 3.5 x 2.5 x 1.5 inch

Parts of uterus:

Lining of uterus: ciliated low columnar epithelium
Lining of endocervix: high columnar epithelium
Anatomical internal os: the part where broad uterine cavity converts into a narrow cervical canal
Histological internal os: the part where uterine epithelium converts into cervical epithelium
Isthmus: part of uterus between anatomical and histological internal os

Structures attached to cornua of uterus:
Anterior to posterior: R - Round ligament
                      T - Fallopian tube
                      O - Ovarian ligament
From superior to inferior: Fallopian tube, Round and ovarian ligament (at same level)

most common cause (mcc) of failure of female sterilization → Identification of wrong structure

**Isthmus**

Lower most part of uterus
Lies between anatomical and histological internal os
0.5 cm in non-pregnant female
5 cm in pregnant female → Forms lower uterine segment (LUS)

LUS: Formed late in pregnancy (at term); LUS → 70% Isthmus
30% cervix

Size at term: 5 cms
Length at labor: 10 cms
Identified by loose fold of peritoneum attached to it

**Histology of uterus**

3 Layers:
1) Serosa – Outermost
2) Myometrium – Thickest (3.5 cms)
* Fibres arranged in three layers

- Outer layer (longitudinal fibres)
- Middle layer (mesh-like fibres)
- Inner layer (circular fibres)

* Middle layer: fibres run criss-cross to constrict blood vessels
  \[ \text{\& A\%A living ligature} \]

3) Endometrium - inner most layer (glands + stroma present)
   Lining: mixed ciliated & non-ciliated columnar epithelium

Layers of endometrium

- Superficial layer
  - Stratum spongiosum
  - Stratum compactum
    - Shed off during menstrual cycle

- Deep layer
  - Stratum basalis
    - Responsible for regeneration of entire endometrium in the next cycle

Endometrial thickness:
- Immediately after menstruation: 1-2 mm
- Early proliferative phase: 5-7 mm
- Pre-ovulatory phase: 11 mm
- Secretory phase: 7-14 mm
- At time of implantation: 11 mm
Peritoneal relation of uterus

Peritoneal covering of the uterus

Culdocentesis: To check for abnormal fluid in pouch of Douglas (cul-de-sac)

Colpotomy: To drain the pelvic abscess

Blood supply of uterus

1. Uterine artery: branch of anterior division of internal iliac artery
2. Ovarian artery

Uterine artery:
- Cardinal ligament: also known as (aka) Mackenrodt ligament / Transverse cervical ligament

- 2 cm lateral to internal os → Uterine artery directly crosses over the ureter i.e. water under bridge (water-ureter, bridge-uterine artery)

↓

Most Common site of ureteric injury during hysterectomy

Obstetrics & Gynaecology • v2.0 • Marrow 4.0 • 2020
Branches of uterine artery

- Branch (br.) To uterus
- Descending cervico vaginal artery
- Sampson artery

From outside to inside

- U - br. Of uterine artery
- A - Arcuate artery
- R - Radial artery
- B - Basal artery

Ovarian artery

Arcuate artery

Radial artery

Basal artery

Spiral artery

Endometrium

Myometrium

Uterine artery

Uterus - lymphatic drainage, nerve supply, development, position

Lymphatic drainage of uterus:
- Fundus → Para aortic nodes
- Cornua → Along round ligament to superficial inguinal nodes
- Body → External + internal iliac nodes

Nerve supply: T₁ - L₁

Development: From Mullerian duct

Position of uterus:
- Normal: Anteverted, antiflexed position [Fundus towards pubic symphysis]
- Retroverted uterus → Fundus towards sacral promontory
- Angle of anteversion: 90° (between vagina and cervix)
- Angle of anteflexion: 180° (between uterus and cervix)
**Round ligament:** Promotes anteversion & anteflexion passes via inguinal canal and attaches at anterior 2/3 of labia majora.  

**Usual position of uterus**

**Anteverted Anteflexed Uterus**

**Cervix**

- **Endocervix or supravaginal cervix**
  - L.25 cm
  - Columnar epithelium

- **Exocervix or Portio vaginalis**
  - L.25 cm
  - Stratified squamous epithelium

**External OS:** Cervix opens into vagina.
- In nulliparous → Circular / Pinpoint
- In multiparous → Transverse / Slit like

**Length of cervix:**
- Non-pregnant → 2.5 cms
- Pregnant → 4-5 cms

**Transformation zone (TZ):** Squamo-columnar junction
- On per speculum examination:
  - Endocervix: Appears red
  - Exocervix: Appears pink
  - Transformation zone: pale area between the two
• TZ originally located → At external os
• Dynamic point → Keeps shifting under the influence of hormones
  ↓
  moves out/Towards exocervix
  moves in/Towards endocervix
  • menopause (less hormonal state)

  1) Puberty
  2) Pills
  3) Pregnancy

m.C variety of Ca cervix: squamous cell Ca.
m.C site: Transformation zone
m.C site for adeno Ca of cervix: Endocervix

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

**Blood supply of cervix**

• Descending cervix - vaginal artery
• Placed at 3’0 clock and 9’0 clock position
• Para cervical block → At 2 and 10’0 clock position
  Or 4 and 8’0 clock position

**Lymphatic drainage of cervix:**
- Hypogastric / internal iliac nodes
- Obturator nodes
- Pre-sacral / ureteric nodes
- External iliac nodes

m.C node in cancer (ca) cervix → Obturator LN
Sentinal nodes for Ca cervix → ureteric nodes
Superficial inguinal nodes → Not involved in Ca Cervix
If involved → Indicated metastasis (stage IV)
Nerve supply: S₂-S₄ segments

Development: Mullerian duct

Before Puberty → Cervix is longer than uterus

<table>
<thead>
<tr>
<th></th>
<th>Cervix : Corpus ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>At birth</td>
<td>1 : 1</td>
</tr>
<tr>
<td>Before puberty</td>
<td>2 : 1</td>
</tr>
<tr>
<td>At puberty</td>
<td>1 : 2</td>
</tr>
<tr>
<td>Reproductive age</td>
<td>1 : 3</td>
</tr>
<tr>
<td>Menopause</td>
<td>1 : 1 (both atrophy)</td>
</tr>
</tbody>
</table>
MULLERIAN MALFORMATIONS

Mullerian duct

- Forms the internal genital organs
- Appears at 6 weeks of gestation
- Originate from intermediate mesoderm
- Left mullerian duct forms –
  - Left side fallopian tube
  - Left half of uterus
  - Left half of cervix
  - Left half of upper 2/3 vagina.
- The right mullerian duct forms the same components on the right side

Formation
Steps - 1 - Both grow towards each other in midline
Step - 2 - Fusion of both mullerian duct

\[
\begin{align*}
\text{Begins by} & \quad 7-8 \text{ weeks} & \text{Completes} & \quad \text{by 12 weeks} & \text{Fusion occurs} & \quad \text{in below} & \quad \text{upwards direction}
\end{align*}
\]

Step - 3 - Fusion complete and septa formed
Step - 4 - Septa resolves by 5th month of intrauterine life
  single uterine cavity is formed
Step - 5 - Fundus of uterus becomes dome shaped
### Mullerian malformations - mullerian agenesis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Known as</th>
<th>Diagram</th>
</tr>
</thead>
</table>
| Both mullerian duct (md) absent | mullerian agenesis or MRKH syndrome | * Both fallopian tube absent  
* Uterus, cervix and upper vagina - absent  
* Lower vagina present - (develop from sinovaginal bulb)  
* Ovaries - present  
* Ovulation - normal |
### Mullerian malformations-unicornuate and didelphic uterus

<table>
<thead>
<tr>
<th>Condition</th>
<th>Known as</th>
<th>Diagram</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Mullerian duct absent on one side</td>
<td>Unicornuate uterus</td>
<td><img src="image1" alt="Diagram" /></td>
</tr>
<tr>
<td>3. Both sides mullerian duct present but fail to fuse</td>
<td>Didelphic uterus</td>
<td><img src="image2" alt="Diagram" /></td>
</tr>
</tbody>
</table>

#### Mullerian malformations- bicornuate uterus

<table>
<thead>
<tr>
<th>Condition</th>
<th>called as</th>
<th>Diagram</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. If both mo are present, incomplete fusion</td>
<td>Bicornuate uterus</td>
<td><img src="image3" alt="Diagram" /></td>
</tr>
</tbody>
</table>

*One fallopian tube present
*Half uterus, cervix, upper vagina present
*Two fallopian tube, Two cervix, Two uterus, Two upper vagina present

*Two fallopian tube
*Two uterus
*One vagina present
### Mullerian malformation - septate uterus and arcuate uterus

<table>
<thead>
<tr>
<th>Condition</th>
<th>Known as</th>
<th>Diagram</th>
</tr>
</thead>
<tbody>
<tr>
<td>5. Both MD are present ↓ Fusion occurs ↓ Septa formed ↓ Septa fails to resolve</td>
<td>Septate uterus</td>
<td>![Diagram of septate uterus](* uterus appears normal from outside ↓ septa present inside*)</td>
</tr>
<tr>
<td>6. Fundus of uterus does not become dome shaped</td>
<td>Arcuate uterus</td>
<td>![Diagram of arcuate uterus](* uterus is flat on top <em>best reproductive prognosis</em>)</td>
</tr>
<tr>
<td>7. DES (Diethylstilbestrol) related abnormalities</td>
<td>T-shaped uterus</td>
<td></td>
</tr>
</tbody>
</table>

**WHO classification of Mullerian duct anomalies**

- Class I: Mullerian agenesis (Mullerian duct aplasia)
- Class II: Unicornuate uterus
- Class III: Didelphys uterus
- Class IV: Bicornuate uterus
- Class V: Septate uterus
- Class VI: Arcuate uterus
- Class VII: DES related abnormalities/T shaped uterus
Diethyl stilbestrol related abnormalities

- Pregnant woman exposed to DES
  - Causes two types of cancer in female foetus
    - Vaginal clear cell carcinoma
    - Adenocarcinoma of cervix

- Effects of DES in female child
  - Uterus
    - MC - Hypoplastic uterus
    - Characteristic T-Shaped uterus
  - Cervix
    - Cervix hood
    - Cervix collar
    - Cervical intraepithelial neoplasia
  - Vagina
    - Vaginal adenosis

- No renal anomalies in female child

- DES effect in male
  - Hypospadias
  - Cryptorchidism
  - Testicular hypoplasia
  - Can lead to renal anomalies

Diagnosis of Mullerian malformations

- Hysterosalpingography (HSG)
  - Radio opaque dye is injected into uterus and cervix using Leech Wilkinson cannula
  - Serial X-rays used to check for flow of dye
- Time for doing HSG: Day 7 - day 10 (pre-ovulatory phase)
- Contraindications: 
  - Pregnancy
  - Pelvic inflammatory disease (PID)
  - Genital TB
  - Allergic to dye

- Indications:
  1. IOC - for Tubal patency
  2. Mullerian malformations
  3. Fibroid / Asherman syndrome (Honeycomb appearance on HSG)

Investigation of choice for mullerian malformation

On HSG, bicorunate and septate uterus cannot be differentiated

- Both uterus appears same on HSG
- IOC - MRI > 3D ultrasound
- Gold standard investigation
  - Laparoscopy + Hysteroscopy
  - 2nd best - Laparoscopy
Identification of mullerian malformations on HSG

Number of fallopian tubes

- Single fallopian tube
  - Unicorns uterus
    - a vagina or
      - Leech - Wilkinson cannula
    - Didelphic uterus
      - Bicornuate uterus
        - Distance between two horns > 4 cm
        - Angle between two horns > 60°
      - Septate uterus
        - Distance between two horns < 4 cm
        - Angle between two horns < 60°

Important points and management of mullerian malformation

- MC Mullerian malformation - Septate uterus > Bicornuate uterus
- MC problem with mullerian malformation - Recurrent abortions
- MC mullerian malformation associated with recurrent abortion, infertility - Septate uterus
- Reproductive outcome is best with - Arcuate uterus > Didelphic uterus
- Worst reproductive prognosis - Unicornuate uterus
- MC mullerian malformation most likely to be associated with renal anomalies - Mullerian agenesis or unicornuate uterus
management
- ≥ 3 abortions due to malformations - surgery

Clinically
- Imperforate hymen
  - Tensed bluish bulging hymen
- Transverse vaginal septum
  - Normal hymen

USG
- Transverse vaginal septum
  - Septum seen

Development of vagina
00:57:28
- Upper 3/5 vagina - develops from Mullerian duct (mesodermal in origin)
- Lower 2/5 vagina - develops from urogenital sinus (endodermal in origin)
- Both the parts fuse - septum is formed
  - Recanalization of septum occurs
  - Single vagina formed
Transverse vaginal septa

- recanalization of septum does not occur

Transverse vaginal septum

\[\begin{array}{c}
\text{m/c location} \\
\text{Normal menstrual cycle but no visible menstruation} \\
\text{Cryptomenorrhea} \\
\text{menstrual blood collects in vagina and uterus called as hematocolpos and hematometra.}
\end{array}\]

- Cryptomenorrhea - caused by Transverse vaginal septum, Imperforate hymen
- mCC of hematometra - Imperforate hymen
- Per abdomen - bulky uterus
- Per vagina - contraindicated in virgin females

Difference between imperforate hymen and transverse vaginal septum

Can be distinguished

Clinically

Imperforate hymen

Tensed bluish bulging hymen

USG

Transverse vaginal septum

Normal hymen

Septum seen
management of transverse vaginal septa.

Excision of septa.

- If septa is located in lower ⅓ or mid ⅓, vaginal
- If septa is located in upper ⅓, abdominal

- Vaginal epithelium is derived from endoderm of urogenital sinus
- Muscles of vagina are derived from mesoderm of mullerian duct
- Hymen - Remnant of sinovaginal bulb
UTERINE PROLAPSE

Prolapse

Definition: ‘Downward displacement of uterus, cervix or vagina from its normal anatomical position’

- Structures preventing prolapse of uterus / vagina
  ↓
  Supports of uterus / vagina

  Prolapse

  ↓
  uterocervical prolapse   vaginal prolapse

  ↓
  Prolapse of uterus and cervix

  Supports of uterus

  ↓
  mechanical factors

  Ligaments

  muscle

Mechanical factors supporting uterus

Usual Position of Uterus

Anteverted Anteflexed Uterus
1. **Angle of anteversion**
   - Angle between vagina and cervix
   - 90°

2. **Angle of anteflexion**
   - Angle between cervix and uterus
   - 130°
   - 1st step in prolapse
   - "Retroversion of uterus"
Round ligament

Origin cornua of uterus passes through inguinal canal
Insertion: Labia majora

→ keeps uterus in anteverted position
↓
  'Secondary Support'

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

Ligaments supporting uterus

Ligaments with support uterus

- Pubocervical ligament
- Lat. Pelvic wall
- Cardinal / Transcervical ligament / mackenrodt's ligament
- Uterosacral ligament
- Sacrum coccyx

Primary support:

1) Pubocervical ligament
2) Transcervical or cardinal or mackenrodt's ligament (most important)
3) Uterosacral ligament

- Broad ligament does not support uterus.
- Prevention of retroversion of uterus →
  1) Uterosacral ligament (best)
  2) Round ligament (2nd best)

Muscle supporting uterus

- Levator ani muscle (most important support)

  Divides into
  - Pubococcygeal
  - Iliococcygeal

  • Forms pelvic diaphragm
- Urogenital diaphragm

  \[ \downarrow \]

  Superficial fascia of urogenital diaphragm
  \[ \downarrow \]
  Inferior fascia of urogenital diaphragm
  \[ \downarrow \]
  Perineal membrane

- Contains: Deep transverse perineal muscle
- Superficial perineal pouch

Contents:
- 1) Bulbospongiosus muscle
- 2) Superficial transverse perineal muscle
- 3) Ischiocavernosus muscle

- Midline muscles attach to
  \[ \downarrow \]
  Perineal body
  \[ \downarrow \]
  Supports uterus, vagina, and cervix
**Mnemonic: BLESSD**

B → Bulbospongiosus muscle
L → Levator ani muscle (most important)
E → External anal sphincter
S → Superficial transverse perineal muscle
S → External urethral sphincter muscle
D → Deep transverse perineal muscle

→ muscles which does not support uterus
  ↓
  ) Ischiocavernosus
  ) Ischiococcygeus

**Uterine prolapse**

* Due to weak uterine supporting system

**Risk factors:**

1) Menopause
  ↓
  - Atonicity of uterus
  - Asthenia of uterus

2) Repeated childbirth / Birth trauma
  - More in elderly, multiparous
  - Rarely in young, nulliparous
Congenital prolapse

- Uterine prolapse in young nulliparous female

- Risk Factors:
  1) Ehler Danlos syndrome
  2) Spina bifida
  3) Osteogenesis imperfecta
  - Cystocele not seen

- Management:
  "SLING Surgery" / "Cervicopexy"

Symptoms of prolapse:

1) Back ache
   ↓
   Due to stretching of uterosacral ligament

Stretching of uterosacral ligaments

2) Decubitus ulcer
   - Due to: Venous congestion
   - Not due to: Friction between thighs

Management:
Packing with: Acriflavine + Glycerine
   ↓
   Antiseptic
   ↓
   Hygroscopic
Complication and management of prolapse

Complications:
1) UTI
2) Kinking of ureter (procidentia)
   Can cause
   Hydroureter     Hydronephrosis
3) Incarceration of prolapsed part
4) Cause cancer (rarely)

Management of prolapsed
depends on
Age of patient     Parity

Sling surgery / cervicopexy

Indication:
1) Congenital prolapse (young, nulliparous female with prolapse)
2) Young female with prolapse
   planning to conceive in future
   
   - Sling used: "mersilene tape"

   Sling surgery
   Anterior sling surgery     Posterior sling surgery
   ↓                             ↓
   Shirodkar surgery

Obstetrics & Gynaecology • v2.0 • Marrow 4.0 • 2020
- Anterior sling surgery

  - Purandare sling surgery
    - One end of sling attached to
      - Isthmus of uterus
    - Other end of sling attached to Rectus Fascia
    - Example: Dynamic sling
      - Female should have good abdominal tone
    - Complications → Less
    - Success rate → Less

  - Khanna sling surgery
    - One end of sling attached to
      - Isthmus of uterus
    - Other end of sling to
      - Anterior superior iliac Spine
    - Complication: Osteitis
    - No longer used

Shirodkar sling surgery

- Attachments
  - one end - Isthmus of uterus
  - other end - ligament attached to sacral promontory

- Example of

  - Posterior sling
  - Static sling

- Recreates / Strengthens: uterosacral ligament
Complications of Shirodkar’s sling

- Left side
  - Obstruction of sigmoid colon
  - Injury to mesenteric vessels
  - Urethral injury
  - Genito femoral nerve injury
  - "Success rate of shirodkar’s surgery - more"

- Right side
  - No complication

Composite sling surgery:
  - Aka. "Virkud sling surgery"

- Right side
  - Shirodkar sling surgery

- Left side
  - Purandare’s sling surgery

Foothergills repair / manchester surgery

Indications:
- Female with prolapse < 40 years of age
- Do not want to conceive in future
- Wants to retain menstrual cycles

Steps:
1) Amputation of cervix (Supravaginal)
   - Complications:
     - Internal OS injury
     - Recurrent 2nd trimester abortions
     - Cervical stenosis
     - Cervical dystocia
     - Posterior lip of cervix
     - Covered by vaginal flap
     - Sturmdorf suture
2. Plication of cardinal ligament
   - Fothergill's suture passes
     from left vaginal skin
     ↓
     Through left cardinal ligament
     ↓
     Into cervix
   - From cervical tissue
     ↓
     Suture is taken up
     and out of cervix
     ↓
     Through right cardinal ligament
     ↓
     Into right side of vagina
   - In Shirodkar modification of Fothergill repair
     ↓
     Amputation of cervix not done (plication of cardinal ligament done)
     ↓
     Female who wants to bear a child

Ward Mayo vaginal hysterectomy, ring pessary
and Lefort's colpolpleisis

Ward Mayo vaginal Hysterectomy
Indications:
- Female with prolapse ≥ 40 years of age
- Does not want to retain menstrual cycle
- Does not want future childbirth

Ring pessary
Indications:
- Young female who refuse surgery
- Prolapse occurs during pregnancy/puerperium
- Contraindicated for surgery
  Ring pessary → Space occupying device at ischial spine
- Temporary cure
  ↓
  Changed every 3 months
  ↓
  Complication: Vesico-vaginal fistula (VVF)
Lefort’s colpocleisis:
Indication:
- Female ≥ 65 years age contraindicated for surgery
- Procedure - Scraping of Anterior/posterior vaginal wall
- Done under LA

Complications:
- Adhesions
  ↓
- Close vaginal wall permanently

Vault prolapse

- Prolapse of vaginal stump
  which is left post hysterectomy

Management:

“Vault suspension surgery”

- Abdominal route
  ↓
- Sacral colpopexy
  (uterosacral suspension)
  Gold standard method

- Vaginal route
  ↓
- Sacrospinous fixation

- Colposuspension surgery
  ↓
  For ‘stress urinary incontinence’ (not a surgery for vault prolapse)
  ↓
  Management:
  Transobturator tape (TOP) > Transvaginal tape (TVT) > Burch colposuspension
Kegel / Perineal exercises:
- Best method to prevent prolapse
- Strengthen perineal muscles
- Done 10 times/day
- Advised:
  - During pregnancy
  - After child birth

**Classification of uterine prolapse**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Reference point</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) SHAW classification</td>
<td>Introitus</td>
</tr>
<tr>
<td>2) POP-Q classification</td>
<td>Hymen</td>
</tr>
<tr>
<td>3) Baden Walker Halfway</td>
<td>Hymen</td>
</tr>
<tr>
<td>classification</td>
<td></td>
</tr>
</tbody>
</table>
VAGINAL PROLAPSE

Management of Vaginal Prolapse:

- Not dependent on age / parity
- Cystocele
- Urethroccele
- Enteroccele
- Rectocele
- Laxed Perineum

De Lancey's Level of Supports:

- Level 1: Related to Pouch of Douglas
- Level II: Related to Bladder
- Level III: Related to urethra
<table>
<thead>
<tr>
<th>Level</th>
<th>Part of vagina</th>
<th>Structural supports</th>
<th>Defect</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>upper 1/3rd of vagina</td>
<td>• Cardinal ligament  • Uterosacral ligament</td>
<td>• Apical prolapse  • Cervical elongation  • Enterocele  • Vault prolapsed</td>
</tr>
<tr>
<td>II</td>
<td>middle 1/3rd of vagina</td>
<td>• Arcus tendineus fascia</td>
<td>• Cystocele  • Rectocele  • Urethrocèle  • Laxed perineum</td>
</tr>
<tr>
<td>III</td>
<td>lower 1/3rd of vagina</td>
<td>• Perineal body  • Muscles attached to it</td>
<td></td>
</tr>
</tbody>
</table>

**Pelvic Organ Prolapse Quantification System classification**

Reference point : Hymen

Anterior wall of vagina

Aa - Lies 3cm above the hymen on the anterior wall of vagina

Baa - Point which is the most dependent part of the anterior vaginal wall between Aa and Anterior fornix

C - Anterior fornix

Posterior wall of vagina

Ap - Lies 3 cm above Hymen on the posterior wall of vagina

Bp - Point which is the most dependent part of the posterior vaginal wall between Ap and posterior fornix

D - Posterior fornix

- Above Hymen - Indicated with "-" sign
- Below Hymen - Indicated with "+" sign

3 Measurements:

![Diagram of pelvic organs and prolapse quantification system]
1. Genital Hiatus (GH)
2. Perineal body (PB)
3. Total vaginal length

<table>
<thead>
<tr>
<th>Aa</th>
<th>Ba</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>GH</td>
<td>PB</td>
<td>TVL</td>
</tr>
<tr>
<td>AP</td>
<td>BP</td>
<td>D</td>
</tr>
</tbody>
</table>

**Staging of vaginal prolapse**

Stage 1: If the leading point of prolapse is > 1 cm above the hymen.

Stage 2: If the leading part of prolapse is > 1 cm below the hymen.

Stage 3: If the leading part of the prolapse is within 1 cm on either side of the hymen.

Stage 4: Complete Prolapse / Procidentia.
URINARY FISTULA

Types of urinary fistulae

1) ureterovaginal fistula
2) vesicovaginal fistula - MC
3) urethrovaginal fistula

ureterovaginal fistula and vesicovaginal fistula (VF)

<table>
<thead>
<tr>
<th>Complain</th>
<th>Fistula</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Constant dribbling of urine from vagina + Normal urination</td>
<td>Ureterovaginal fistula</td>
</tr>
<tr>
<td>2. Constant dribbling of urine from vagina + No normal urination</td>
<td>VVF</td>
</tr>
</tbody>
</table>

urethrovaginal fistula

Complaint

No constant dribbling of urine from vagina.
At the time of urination- urine from vagina & urethra.

Fistula.

Urethrovaginal fistula.
Methylene blue three swab test

- Three cotton plugs - Placed in vagina
  - Uppermost
  - Middle
  - Lower

  **Corresponds to**
  - Ureterovaginal fistula
  - VVF
  - Ureterovaginal fistula

- with the help of foley's catheter

  Put methylene blue dye in bladder + urethra, wait

<table>
<thead>
<tr>
<th>Observation</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>* If the uppermost cotton swab wet with urine but not blue in colour</td>
<td>Ureterovaginal fistula</td>
</tr>
<tr>
<td>* If middle cotton swab (or middle + Lower) is wet + blue in colour</td>
<td>VVF</td>
</tr>
<tr>
<td>* If only Lower cotton swab is wet + blue</td>
<td>Urethrovaginal fistula</td>
</tr>
</tbody>
</table>

Vesicovaginal fistula

- m.C - Urinary fistula
- m.CC - In developed countries - Surgery (Hysterectomy)
  Developing countries - Obstructed labor
- I.O.C - Cystoscopy
- Best method to collect urine in VVF patient -
  Suprapubic catheterisation
management of VF

Surgical repair

↓

Time of repair

↓

Due to obstructed labor

↓

Repair after 6 weeks (infection and inflammation subsides)

↓

due to surgery

↓

Diagnosed within 24 hrs

↓

Immediate repair

↓

Diagnosed >24 hrs

↓

Wait for 6 months

↓

Due to radiotherapy

↓

Wait for 12 months

ureteric fistulas

• MC - Site of injury - a cm lateral to internal os - water under bridge

• ACM - Site - Pelvic brim

• Highest risk of ureteric injury - Wertheim's hysterectomy

• MC of ureteric injury - Simple hysterectomy / Total abdominal hysterectomy

• MC route of surgery in which ureteric injury is common - Laparoscopic hysterectomy

Management of ureteric fistula

00:22:23

• Time of repair - Immediately as soon as diagnosed

• Technique - Boari flap technique

Mensouria, or menouria.

• Hematuria at the time of menstruation

↓

Seen in

↓

Endometriosis of bladder

↓

Fistula connecting uterus to bladder

↓

Uterovesical fistula
New classification of genitourinary fistulae

- Depending on distance of fistula from external urinary meatus

<table>
<thead>
<tr>
<th>Type IV</th>
<th>Type III</th>
<th>Type II</th>
<th>Type I</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5 cm</td>
<td>2.5 cm</td>
<td>3.5 cm</td>
<td></td>
</tr>
</tbody>
</table>

- If the distance of fistula from external urinary meatus is:

  - < 1.5 cm
  - 1.5 cm - 2.5 cm
  - 2.5 cm - 3.5 cm
  - > 3.5 cm

  - Type IV fistula
  - Type III fistula
  - Type II fistula
  - Type I fistula

a) < 1.5 cms in diameter
b) 1.5 to 3 cms in diameter
c) > 3 cms in diameter.
PELVIC INFLAMMATORY DISEASE (PID)

Definition
- Infection of upper female genital tract which includes
  - Endometritis
  - Salpingitis
  - Oophoritis
  - Peritonitis
- MCC of PID - Chlamydia > Gonorrhea.
- MCC of acute PID - Gonorrhea
- MCC of PID in IUCD users - Actinomycetes
- MCC route of spread of PID - Ascending infection along with sperms
  - Excep - Genital TB
  - Hematogenous spread
- MCC PID in virgin females - Genital TB

Risk factors
- Multiple sex partners - MCC
- Low socio-economic status
- Douching
- Young age of intercourse (10 - 19yrs)
- History of instrumentation

Protective factor
- Barrier methods - Protect against PID

Gonococcal infection
- Squamous epithelium is resistant to gonococcal infection
- MCC site of asymptomatic carriage of gonococcal infection in females - Endocervix
- Other sites - Urethra / Bartholin glands
- Gonococcal vaginitis - not seen in young females
* Theoretically, gonococcal vaginitis can occur in new born females (vagina - lined by transitional epithelium)

**Spread of gonococcal infection**

- From cervix - Spreads to endometrium
  - Causes endometritis
  - Salpingitis
  - Oophoritis

- From Fallopian tube when infection spreads to peritoneum
  - Peritonitis
  - heals by fine violin string adhesions
  - Adhesions between liver capsule and parietal peritoneum
  - leads to Perihepatitis
  - Fitz - Hugh - Curtis syndrome
  - can lead to infertility / ectopic pregnancy
  - violin string adhesions
Diagnosis of pelvic inflammatory disease

- Minimum criteria - Any 1 should be present
  i) Uterine tenderness
  ii) Cervical motion tenderness (also seen in ectopic pregnancy)
  iii) Adnexal tenderness

- Supportive criteria.
  i) Fever ≥ 101.6° F or ≥ 38.3° C
  ii) Raised WBC count
  iii) Raised CRP
  iv) Abundant WBC in discharge
  v) Lab test positive for gonorrhea and Chlamydia

  For gonorrhea:
  - LOC - NAAT (Nucleic Acid Amplification Test)
  - Sample - endocervical discharge > Urine sample
  - Gold standard - Culture (Thayer Martin medium)

  For Chlamydia:
  - LOC - PCR / NAAT
  - Gold standard - culture (mccoy medium)

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

Definitive diagnosis

i) Gold standard for diagnosis - Laparoscopy
   ↓ Because
   ↓
   - Biopsy can be taken from fallopian tube
   - Prognostic scoring for conception can be done - Boer miesel score

ii) Endometrial biopsy - Endometritis

iii) Transvaginal ultrasound - Normally Fallopian tube not visible on USG
     In acute inflammation tubes: inflamed, swollen, occluded distally
     ↓
     Fallopian tubes - visible
Findings on ultrasound

- Distended / ovoid tube
- Thick inflamed wall
- Fluid present in tube
- Incomplete septa (due to adhesion)

Signs on ultrasound (USE)

- Bead on string appearance / cogwheel sign (in transverse section)
  - Due to endosalpingeal folds which form nodules on fallopian tube

- Waist sign
  - On longitudinal section
  - On diametrically opposite places - indentation

Bead on strings appearance of tube in PID

Waist sign

Management of pelvic inflammatory disease

- Antibiotics
  - Against gonorrhoea
  - Against anaerobes
  - Chlamydia
• PID - OPD Management

Regime - 1  Ceftriaxone 250 mg IM single dose  
+  
Doxycycline 100 mg BD X 14 days  
+  
metronidazole 500 mg BD X 14 days

Regime - 2  Cefoxitin 2 gm IM + Probenecid 1 g oral single dose  
+  
Doxycycline 100 mg BD X 14 days  
+  
metronidazole 500 mg BD X 14 days

Indications for hospitalisation in PID
1) Pregnancy
   a) Surgical emergency - Appendicitis (if cannot be ruled out)
   b) Tubo ovarian abscess
   c) Oral treatment

If no response  
Unable to take  
Not compliant

Oral drugs  
(due to nausea/vomiting)  
High fever

parenteral treatment

Regimes for parenteral treatment
• Regime - 1 - Cefotetan 2g IV every 12 hours  
+  
Doxycycline 100mg orally or IV every 12 hours
- **Regimen - 2** - Cefoxitin 2g IV every 6 hours
  + Doxycycline 100mg orally or IV every 12 hours

- **Regimen - 3** - Clindamycin 900mg IV every 8 hours + Gentamycin loading dose IV or IM (3mg/kg), followed by a maintenance dose (0.5mg/kg) every 8 hours.

- Single day dosing (3-5 mg/kg) can be substituted.
GENITAL TB

Introduction
- mostly Secondary infection
  M.C primary site - lungs > lymph nodes
  ↓ via hematogenous route
  Genital tract
  ↓
  M.C site - Fallopian tube
  ↓
  Ampulla.
  Least common site - vagina/vulva.
- M.C PID (Pelvic inflammatory disease) in virgin females

Genital tuberculosis - in fallopian tube

- M.C site - Ampulla
- M.C symptom of genital TB - Infertility
- Hysterosalpingography (HSG) - contraindicated
- If HSG is done
  ↓
  Characteristic findings
  ↓
  i) Lead pipe appearance of tube
  ii) Beaded appearance of tube
  iii) Tobacco pouch appearance of tube
  iv) Golf stick appearance of tube
  v) E/L cornual block
  vi) Endometrium - honey comb appearance - Asherman syndrome
  vii) moth eaten appearance of tube
• M.C. site of blockage in tube due to
  - Genital TB
  - Gonorrhea
  - Cornual end of tube
  - Fimbrial end of tube

• M.C.C. of B/L cornual block on HSG - Physiological spasm
• M.C. pathological cause B/L cornual block on HSG - Genital TB

Endometrial tuberculosis 00:08:26

• From the fallopian tube - infection spreads to endometrium

• M.C. route of spread of genital TB - Hematogenous
• M.C. route of spread of endometrial TB - Direct spread

Endometrial TB - manifestation

  - Pus in uterus
  - Pyometra.
  - M.C. cause of pyometra - senile endometritis
  - M.C. cancer causing pyometra - Cancer cervix
  - Cancer endometrium
  - M.C. cause of hematometra - imperforate hymen

  - Asherman syndrome
  - Intra uterine adhesions
  - Endometrium is thin and defective

• M.C.C. - Postpartum curettage
• Other causes - Dilation & Curettage
  - MTP
  - Genital TB

Asherman syndrome 00:15:35

• Endometrium is thin and defective

  - Hypomenorrhea.
  - Amenorrhea (secondary)

• Diagnosis: I.O.C. - Hysteroscopy
• Managements - Hysteroscopic adhesiolysis
  + Insert balloon catheter
    (to prevent formation of adhesion again)
  + Estrogen + Progesterone
    (to make endometrium thick)

Ovarian tuberculosis

• 3rd site to be involved - Ovary (30%)
  ↓
  Congestion of ovary
  ↓
  Polymenorrhea

• Initially in genital TB - polymenorrhea
  ↓
  Amenorrhea

Diagnosis of genital TB

  Endometrial biopsy
  ↓
  • Done 1 or 2 days before menstruation
  • Also from menstrual blood (within 12 hrs)

  Menstrual blood - sent for

  Culture
  LH media
  (Lowenstein - Jensen media)

  Histopathological examination

  PCR

Management of Genital TB

• Antitubercular therapy (ATT) - for 6 months
  (safe in 1st trimester of pregnancy)

• Management of infertility - IVF (invitro fertilization)
One liners

- m.C. pelvic examination finding in genital TB - Normal findings
  a.m. m.C. - Tenderness

- m.C. pelvic examination finding in Genital TB in adolescent females
  ↓
  B/L. adnexal mass

- Percentage of genital TB patients - infertile - 70%

- Percentage of infertile patients having genital TB
  ↓
  Worldwide - 10%
  India - 17%
# Vaginitis

<table>
<thead>
<tr>
<th>Organism</th>
<th>Bacterial Vaginosis</th>
<th>Trichomonas Vaginitis</th>
<th>Candidiasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (W) flora of vagina: &lt;br&gt; Böderlein's bacilli</td>
<td>Lactobacilli is replaced by: &lt;br&gt; - Gardnerella &lt;br&gt; - Mobiluncus &lt;br&gt; - Ureoplasma ureolyticum &lt;br&gt; - No Inflammation &lt;br&gt; <strong>.: vaginosis not vaginitis</strong></td>
<td>Flagellated protozoa &lt;br&gt; <strong>↓</strong> &lt;br&gt; Trichomonas vaginalis</td>
<td>Candida Albicans</td>
</tr>
<tr>
<td><strong>Most common (me) sexually transmitted disease (STD)</strong></td>
<td><strong>✓</strong></td>
<td><strong>✓</strong></td>
<td>Usually not</td>
</tr>
<tr>
<td>Symptoms</td>
<td><strong>X</strong></td>
<td><strong>✓</strong></td>
<td></td>
</tr>
<tr>
<td>- Dirty white foul smelling discharge &lt;br&gt; - No itching &lt;br&gt; No pruritis</td>
<td>- Yellowish green discharge &lt;br&gt; - Pruritis (⁺) &lt;br&gt; - Dysuria</td>
<td>- Cottage cheese like discharge &lt;br&gt; (or) curdy white discharge &lt;br&gt; - Intense itching &lt;br&gt; - Splash dysuria</td>
<td></td>
</tr>
<tr>
<td>Signs</td>
<td><strong>≥ 4.5</strong></td>
<td>Inflamed vagina &lt;br&gt; <strong>↓</strong> &lt;br&gt; Angry looking vagina (or) Strawberry vagina</td>
<td></td>
</tr>
<tr>
<td>pH of discharge</td>
<td><strong>5-6</strong></td>
<td><strong>&lt; 4.5 Can survive in acidic media. me vaginitis in pregnancy</strong></td>
<td></td>
</tr>
<tr>
<td>Gold standard investigation</td>
<td>Clue cells</td>
<td>Motility/flagellated protozoa</td>
<td>Hyphae/pseudohyphae</td>
</tr>
<tr>
<td>----------------------------</td>
<td>------------</td>
<td>-------------------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Saline microscopy - LOC</td>
<td>- vaginal epithelium to which numerous bacteria are adhered. Gram staining ↓ Nugent Score - look at the number of Gardnerella (0-4) * Mobilincus (0-2) * Lactobacilli (0-4) If score ≥ 7 Bacterial vaginosis ☑</td>
<td>Culture ↓ Diamond media</td>
<td>Culture ↓ Sabouraud media</td>
</tr>
</tbody>
</table>

**DOC:**
- Non pregnant
- Pregnant
  - Treatment of male partner

<table>
<thead>
<tr>
<th>Nonpregnant</th>
<th>Treatment</th>
<th>Pregnant</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metronidazole 500mg TDS × 7 days</td>
<td>Avoid treatment in 1st trimester</td>
<td>Metronidazole 500mg TDS × 7 days (or) Ag stat (oral) 500mg TDS × 7 days</td>
<td>Avoid in 1st trimester Only if symptoms ☑</td>
</tr>
<tr>
<td>Fluconazole 150mg - stat</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Amsel criteria for diagnosing bacterial vaginosis

Any 3 out of the 4 criteria: diagnostic

1) Dirty white foul smelling discharge thinly coating vagina
2) pH of the discharge ≥ 4.5
3) Clue cells ≥ 20%

4) On adding 10% KOH to discharge → Fish like odour
   ↓ known as (k/a)
   
   Whiff test
   Or
   Amine test
   
   Positive

Risk factors for candidiasis:
- Diabetes
- OCP use
- Steroid use
- Antibiotics
- Immunocompromised
- HIV

Recurrent Candidiasis: ≥ 4 episodes in a year

Clue cells
MALE INFERTILITY

Infertility - overview

Definition: Inability of a couple to conceive even after regular unprotected sexual intercourse for over 1 year

If age of female ≥ 35 years

Start investigation after 6 months of trying

Incidence of infertility: 10-15% (Subfertile)

Fecundability: Ability of a female to conceive within a menstrual cycle

Fecundity: Ability of a female to give live birth in a menstrual cycle

Causes of infertility

male infertility

Semen analysis

• 1st investigation to be done in an infertile couple

• Sample - masturbation

• Abstinence 2-7 days

• Sample should not be collected in latex condom

• Study of semen should be done on liquified semen

Liquefaction time: 30-60 mins

Sample should reach lab within 60 mins

• If semen analysis report is abnormal compulsory

Repeat semen analysis
(atleast 1 month)
Hormonal support of spermatogenesis

First stimulus in a baby to produce testosterone: HCG

Sperm pathway
Semen analysis

- WHO 2010 parameters of normal semen analysis gives the lower limit of normal

1. Volume > 1.5 ml
2. pH > 7.2
3. Sperm concentration > 15 million /ml
4. Sperm count > 39 million / ejaculate
5. Average sperm count 60 - 100 million / ejaculate
6. Sperm morphology
   - Strict criteria > 4% normal
7. Total motility > 40%
8. Progressive forward motility > 32%
8. Viability > 58%
9. WBC count < 1 million / ml
   (DOC: any infection in male genital tract → Doxycycline)
10. Total no. of round cells < 5 million / ml (WBC + globular sperms)

\[\text{Difference b/w} \]
\[\downarrow\]
Non - motile viable sperm
\[\downarrow\]
Dead sperm
\[\downarrow\]
Can be used in ICSI (intracytoplasm sperm injection)
\[\downarrow\]
Test
\[\downarrow\]
Hypo osmotic swelling test (osmosis occurs in living sperm)

### Abnormal semen analysis

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. No semen</td>
<td>Aspermia</td>
</tr>
<tr>
<td>2. Sperm concentration &lt; 15 million / ml</td>
<td>Oligospermia</td>
</tr>
<tr>
<td>3. Sperm concentration &lt; 5 million / ml</td>
<td>Severe oligospermia</td>
</tr>
<tr>
<td>4. Non-motile sperm (motility ↓)</td>
<td>Asthenospermia</td>
</tr>
<tr>
<td>5. Sperms with abnormal morphology</td>
<td>Teratospermia</td>
</tr>
<tr>
<td>6. Non viable / dead sperms</td>
<td>Necrozoospermia</td>
</tr>
<tr>
<td>7. No sperms in semen</td>
<td>Azoospermia</td>
</tr>
<tr>
<td>8. ↑ WBC in semen</td>
<td>Leucocytospermia</td>
</tr>
</tbody>
</table>
**Azoospermia**

- **Pretesticular**
  - Hypothalamus/Pituitary
  - LH ↓
  - FSH ↓
  - Testosterone ↓
  - Spermatogenesis absent

- **Testicular**
  - LH ↑
  - FSH ↑
  - Testosterone ↓
  - ±

- **Post testicular**
  - LH normal
  - FSH normal
  - Testosterone normal
  - Normal
  - ↓
  - Best prognosis

**Obstructive azoospermia**

- Testis
- Epididymis
- Seminal vesicle
- Prostate
- VD
- ED

---

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Scanned with CamScanner
<table>
<thead>
<tr>
<th>Block</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Level A</td>
<td>Level B</td>
</tr>
<tr>
<td>In semen-</td>
<td>In semen-</td>
</tr>
<tr>
<td>- Sperms</td>
<td>- Sperms</td>
</tr>
<tr>
<td>+ Fluid from SV</td>
<td>- Fluid from SV</td>
</tr>
<tr>
<td>Good volume</td>
<td>poor volume</td>
</tr>
<tr>
<td>Fructose present</td>
<td>Fructose present</td>
</tr>
<tr>
<td>+ prostate fluid</td>
<td>+ Prostate fluid</td>
</tr>
<tr>
<td>+ Bulbourethral</td>
<td>+ Bulbourethral</td>
</tr>
<tr>
<td>secretions</td>
<td>secretions</td>
</tr>
</tbody>
</table>

If fructose present in semen - indicates block at seminal vesicle - scrotal ultrasound is done to identify the level of obstruction.

**Fructose absent in semen**

```
<table>
<thead>
<tr>
<th>Block in ejaculatory duct</th>
<th>6/L absence of seminal vesicles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transrectal ultrasound</td>
<td>6/L vas deferens, absent</td>
</tr>
<tr>
<td></td>
<td>CFTR gene testing</td>
</tr>
<tr>
<td></td>
<td>(cystic fibrosis)</td>
</tr>
</tbody>
</table>

mixed of obstructive azoospermia

Sperms are present in testis & epididymis

Testis

TESA Testicular sperm aspiration

TESE Testicular sperm extraction

Followed by

ICS

(intracytoplasmic sperm injection)
## Management of male infertility

<table>
<thead>
<tr>
<th>Condition</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Oligospermia 10-15 million / ml</td>
<td>Intrauterine insemination</td>
</tr>
<tr>
<td>2. Oligospermia 5-15 million / ml</td>
<td>In vitro fertilization</td>
</tr>
<tr>
<td>3. Severe oligospermia &lt; 5 million / ml</td>
<td>Intracytoplasmic sperm injection (ICSI)</td>
</tr>
<tr>
<td>4. Obstructive azoospermia</td>
<td>ICSI</td>
</tr>
<tr>
<td>5. Asthenospermia</td>
<td>ICSI</td>
</tr>
</tbody>
</table>
FEMALE INFERTILITY - 1

Female Infertility:
1. Ovarian cause: 40%
2. Tubal cause: 40%
3. Uterine / Cervical cause: 10%
4. Unexplained: 10%

Ovarian Causes

WHO Classification:

Class I
- Hypothalamo pituitary ovarian axis failure - 10%
  - E.g. Kallman syndrome - Hypothalamic failure + Anosmia.

Class II
- Hypothalamo pituitary ovarian dysfunction - 85% cases - Anovulation
  - E.g. PCOS, ↑ Prolactin levels
  - most easily treatable form of infertility

Class III
- Premature Ovarian failure / Premature menopause - 5%

Tests for Ovulation

Basis:
- If female ovulates, progesterone is present
- If female does not ovulate, progesterone is absent, then the major hormone in her is estrogen

Time for tests of ovulation: Done on Day 22 of cycle
  (if regular cycles) or irregular cycles - 1 week before menstruation

<table>
<thead>
<tr>
<th>Test</th>
<th>If female ovulating (Progesterone +nt)</th>
<th>If female is not ovulating (estrogen ++)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cervical mucus study</td>
<td>Ferning -ve</td>
<td>Ferning +ve</td>
</tr>
</tbody>
</table>
Other tests for ovulation

1. Vaginal epithelial study
   Intermediate cells are seen
   Pink colored cells with pyknotic nuclei
   Superficial cells are seen

2. Basal body temperature
   Temp. increases by 0.2 - 0.5°C (or) 0.4 - 0.8°F in the mid cycle
   Temperature remains constant

3. Endometrial biopsy (Earlier: Best test)
   Corkscrew appearance/saw tooth appearance
   Simple cystic tubular glands
   of glands filled with Secretions
   Not the best test now as it’s invasive

Indications for Endometrial biopsy:
- Genital TB
- Leuteal phase defect diagnosis

Important signs of Endometrial biopsy:
- 1st sign of ovulation on endometrial biopsy: Subnuclear vacuoles appear
- Max. stromal edema: Seen on Day 22 of cycle
- Leukocytic infiltration: Seen on Day 26

Hormonal studies and USG

5. Hormonal study:
   - S. Progesterone ≥ 3 ng/ml < 3 ng/ml
   - LH Kits
     LH test positive → Ovulation
     Fertilizable span of ova = 24 hrs, so can have intercourse up to 48 hours
• Hormonal studies: Best test for ovulation.
• Detect ovulation: Best test: S. Progesterone
• Best test to predict ovulation: LH Kit - Testing should be done between 4 pm - 10 pm
• MC test for ovulation:
  ↓
  TVS A/V/A follicular monitoring
• It’s done from the Day 10 of the cycle, everyday the size of follicle increases from a -3 mm/day
  ↓
  Reach a size of 18 - 20 mm
  • Then the size of follicle decreases suddenly
  • Fluid in pouch of d.o.gu.s.
  • Endometrium will appear triple layered →
    Trilaminar appearance of endometrium

• USG of endometrium at different times of cycle
  • In proliferative
    phase → Estrogen ++
    ↓
    Endometrium appears in the form of single white line

• At the time of ovulation → Endometrium shows 3 layers i.e. trilaminar endometrium

• In the Secretory phase → Endometrium appears as a single line but it will be thick with posterior enhancement

Management of anovulation

• Same as management of PCOS

• 1st line:
  • DOC for anovulation in PCOS: Letrozole > Clomiphene citrate
  • DOC for anovulation in obese female with PCOS: Letrozole
  • DOC for anovulation: Clomiphene citrate
Letrozole:

- DOC in PCOS patients
- It's an aromatase inhibitor
- Initial dose: 2.5 mg
- Max. dose: 7.5 mg

a. Clomiphene citrate:

- SERM - *Clomiphene*
  - *Tamoxifen*
- *Raloxifene* - Not used for ovulation induction
- Initial dose: 50 mg/day
- Max. dose: 100 mg/day
- Obese female with anovulation: Clomiphene citrate + metformin
- Increased prolactin levels + anovulation: Clomiphene citrate + Bromocriptine

- Anovulation + obese $\rightarrow$ 1st step:
  - Advise her for weight loss
  - Dietary modification
  - Physical exercise
  - Morbidly obese: Bariatric surgery
  - Simply by weight loss: 10-15% cases ovulate
  - Best time to conceive after Bariatric surgery: 12-18 months

- 2nd line management:
  - hMG (Human menopausal Gonadotropin)

- 3rd line mx:
  - Gonadotropins in pulsatile manner

**Clomiphene citrate** 00:40:16

- ↓ the levels of estrogen
  - → Negative feedback on FSH is gone
  - ↓ FSH
  - Number of follicles start growing

- Prerequisite for clomiphene citrate intact HPO axis
• MC S/E of clomiphene citrate:
  - Hot flushes
  - Menopause like symptoms
  - Can lead to multifetal pregnancy
  - Chances for twin pregnancy: 5 - 8%

• Give clomiphene from D2 - D6 of the cycle
  ↓
  From D9 - DO follicular monitoring
  ↓
  When follicles become 18 - 20 mm in size
  ↓
  Give inj.hCG - acts as ovulation trigger (similar to LH surge)
  ↓ after 36 hrs
  Ovulation

• % Chance of ovulation with clomiphene citrate: 80%

• Pregnancy rates with clomiphene citrate: 30-40% due to dryness of vagina.

• Inj. hCG → Trigger ovulation
  * Sometimes cause ovulation hyperstimulation syndrome (OHSS)
  * To prevent OHSS: Create ideal conditions to give inj.hCG.
    - Estrogen should be: 450 - 1000 pg/ml
    - There should be 1 or 2 follicles which are ≥ 16 mm size.
    - Thickness of endometrium: 8 mm
  * Do not give inj.hCG if estrogen (EA) ≥ 3500 pg

• Chances of multiple pregnancy: Max with hMG (30%) > clomiphene Citrate (5 - 8%) > GnRH

• Chances of OHSS: hMG: 5%
  Clomiphene citrate: 1%

OHSS

• Ovarian Hyperstimulation syndrome.

• All ovulation inducing drugs
  ↓
  Directly or indirectly increasing FSH
  ↓
  multiple follicles gets stimulated
  ↓


↓
↑ estrogen production (E)
Each follicle releases 150 – 200 pg of E

↓
Responsible for OHSS

* If E ≥ 3500 pg, chances of OHSS are 15%
* If E ≥ 6000 pg, chances of OHSS are 40%
* When E levels are increased

↓
↑ inflammatory cytokines like VEGF and interleukins

↓
make leaky capillaries + stimulate RAPS (electrolyte imbalance)

↓
• Cellular components stay inside
• Hemoconcentration
• HCT ↑
• ↑ chances of thrombosis
• ↑ DIC

↓
Liquid components come out through leaky capillaries

↓
Get collected in the third space

• Ascites
• Hydrothorax
• Pleural effusion

• Size of the ovary ↑es due to big follicles
• Becomes tense, may undergo torsion of ovary, rupture, hemorrhage of ovary
• Controlled stimulation of ovaries - to prevent hyperstimulation

Risk factors for OHSS:
1. Age < 35 years
2. Case of PCOS
3. Thin female
4. Anti Müllerian Hormone > 3.3
   * From puberty onwards, the granulosa cells of small follicles secrete AMH
• AMH is increased, that indicates is increase in number of
small follicles

5. If E2 levels ≥ 3500 pg
6. Inj hCG
7. If no. of follicles are increased:
  • If there are ≥ 13 follicles and they are > 11mm in size, that
    predicts OHSS may occur
  • If the follicles are > 20 in number, then the incidence of
    severe OHSS is 15%

8. Pregnancy
  • In pregnancy hCG is produced
    ↓
    Triggers or worsens the OHSS
  • m.c. cause of late OHSS is pregnancy.

Classification of OHSS

\[ \text{Early} \quad \downarrow \quad \text{OHSS} \quad \downarrow \quad \text{Late} \]

• Occurs within 9 days of using
  inj hCG which is used as
  ovulation trigger
  • Occurs > 9 days after inj hCG
  • m.c. of late OHSS : Pregnancy

Grades of OHSS:

Grade 1:
  • mild
  • Patient complains of abdominal distention or discomfort
  • On USG, size of ovary = max upto 5 cms

Grade 2 & 3:
  moderate (Grade 2 and 3)
  Grade 2 moderate: size of ovary: 5-12 cms
  Grade 3 moderate: USG evidence of ascites

Severe OHSS: Grade 4 & 5:
  • Grade 4: Clinical evidence of ascites, pleural, pericardial
    effusion
  • Grade 5: Hemoconcentration
    • DIC
    ↓ Renal perfusion
    → Thrombosis
management:
- Manage hypovolemia - IV fluids, colloids
- Heparin to prevent thrombosis
- For tensed ascites: Paracentesis

Prevention of OHSS:
- By delaying Inj. hCG if E2 ≥ 3500 pg/ml
- Stop Gonadotropins

WHO category III

- Premature menopause / premature ovarian failure
- If menopause occurs in a female < 40 yrs
  - No. of follicles are decreased

Tests for Ovarian Reserve:
1. S. FSH levels on Day 3 of the cycle
   - If ≥ 45 pg → Premature ovarian failure.
2. Day 3 Inhibin levels
   - < 45 IU → Premature ovarian failure.
3. Antral follicle count - TVS - If in both ovaries, follicles < 10, indicates
   - Premature ovarian failure - Done on D3 - Quantitative test

- Anti mullerian hormone
  - < 0.5: Poor ovarian reserve
  - Can be done on any day of the cycle - Best test for Ovarian Reserve
• Outdated test: clomiphene citrate challenge test:
  • On D3 - measure FSH levels
  • From D5 - D10: give clomiphene citrate
  • measure FSH levels on day 10 - FSH ↑↑
• S. Estradiol level - insignificant in case of ovarian Reserve

management:
• Take donor eggs + husband sperms
  ↓
  Do IVF
FEMALE INFERTILITY - 2

Tubal causes of infertility:

* IOC : HSG
  * HSG done with the help of Leech Wilkinson cannula
  * with Leech wilkinson cannula, inject a radio opaque dye in the uterus and tubes
  
  ↓

  Check Dye Spillage → if present - Both tubes are patent & open

  * No need of surgery for U/L block
  * Do HSG on Day 10 - Always Preovulatory

Indications for HSG:

* For tubal infertility
* For uterine pathologies:
  Asherman syndrome
  Polyp
  Fibroid
  mullerian malformations

Drawbacks of HSG:

* Exterior of the tube cannot be visualised.
* Spasm of the cornual end of the tube. It appears like B/L cornual block
* mc of B/L cornual block on HSG - Physiological spasm of cornual end of tube
* mc pathological cause of B/L cornual block on HSG - Genital T.B.
* Gold std. investigation for tubal infertility - Laparoscopic chromopertubation
* Other investigations for tubal infertility - Saline infusion Sonography.

Causes of tubal infertility:

1. Endometriosis
2. PID
3. Salpingitis
4. Genital T.B
   ↓
   All these cause blockage and adhesions in the tube
   ↓
   Infertility

   • Treatment is done only if there is B/L blockage

   Blockage of tube
   ↓
   Proximal / cornual block
   ↓
   midsegmental block
   ↓
   Distal block

B/L Cornual block:

mcc : Physiological sphincter spasm.
   ↓
   Next step: Take a thin guide wire, under hysteroscopic guidance pass it into the tube
   this is called as Hysteroscopic Cannulation.
   This should be followed by Laparoscopic chromopertubation
   ↓
   most of the times, block gets relieved
   ↓ if not relieved
   It means, it's a pathological block (mcc : Genital T.B)
   ↓
   Best management : IVF

B/L Distal block:

   Distal block
   ↓
   mild block
   ↓
   Severe block
   ↓
   Fimbriectomy
   ↓
   IVF

   • Best management of tubal infertility : IVF

mid Segmental block:

   • It means that tubectomy was done in the past
• patient comes for reversal of tubectomy
• 1st step: Husband's Semen analysis
  ↓
  • If normal: Recanalisation / Reversal.

Factors affecting Recanalisation:
1. Technique of sterilisation
   Clips (best) > Falope ring
   • Least possible: Cautery
2. Type of anastomosis:
   • Best if there is isthmo-isthmic anastomosis
3. Reconstruct ≥ 4 cms of the tube chances of reversal are good,

Uterine and cervical causes of infertility

Cervical Causes:
• There's a problem with the cervical mucus
  • Thick and impermeable mucus → Sperms cannot ascend.
  • Impermeable mucus: Insler score
    Score: 10 - 12: Good cervical mucus.
  • Presence of Antisperm Antibodies - immunological cause of infertility
  • For screening: Post coital test / Sims test / Huhner test
    Done on D10 - D14 of the cycle

Test for infertility Day performed
1. Ovulation tests D2a
2. Follicular monitoring D10
3. Tests for ovarian reserve D3
4. AMH Any Day
5. HSG D10
6. Post coital test D10 - D14

• In Sims test: Forward movement of sperms ⇒ presence of anti sperm Ab's

• Diagnostic test for Antisperm Ab's:
  1. Immuno bead Assay
  2. Sperm agglutination test
  3. Sperm mobilisation test
management of Cervix infertility:
- Intrauterine Insemination (IUI)
- m_x of unexplained infertility: Clomiphene citrate f/b IUI X 3 cycles  ↓ fails  IVF

IUI

• Semen taken from male (can be Husband = IUI H Can be Donor = IUI D)

• Process and centrifuge the semen. Can be done in 3 techniques
  i. Swim up technique
  ii. Swim down technique
  iii. Density centrifugation technique

• 0.5 ml of processed and centrifugal semen, with the help of catheter, put in the uterus (by passing area of Cx)

Prerequisites before IUI:
1. Sperm concentration ≥ 10 million/ml
2. Sperm morphology at least 14% normal

Indications of IUI:
1. If male has ejaculatory problems
   • Hypospadias
   • Epispadias
   • Retrograde ejaculation
2. If male has Neurogenic impotence.
3. If male has oligospermia, but sperm count is between 10 - 15 million/ml
4. If male partner has a hereditary disease, risk of transferring the disease to the offspring. In this case, go for IUI D
5. If in female cervix: Antisperm antibodies are present.
6. If female has vaginismus.
IVF

- From male partner: Take semen, process and centrifuge it
- For female: Give clomiphene citrate / hMG / GnRH

  Hyperovulates

  Do a follicular monitoring from D_0 of cycle

  When follicles reach 18 - 20 mm size

  Give inj. hCG (ovulation trigger)

  After 24-36 hrs

  Do oocyte retrieval under USG guidance

  Put both sperms and oocyte into petridish in

  50,000 - 1,5 lakh sperms per oocyte

  Fertilisation occurs and embryo is formed

  On D_3/D_5 implanted in uterus 2 cms below the fundus

- Success rate of IVF: with 1 embryo: 20-30%
- Cumulative success rate: 40-50%

Limitations of IVF:

- Severe oligospermia
- Obstructive azoospermia
- Asthenospermia

Indications for IVF:

In Males: Oligospermia: 5-15 million sperms/ml
  Repeated IUFI Failures.

In Females: B/L blockage of tube

4. In ↓ ovarian reserve

  Do IVF - with Donor Eggs
• In Mullerian agenesis
  ↓
  IVF + Surrogacy

**ICSI**

Intracytoplasmic Sperm Injection

• Everything is same as IVF except a single sperm is injected into the cytoplasm of the oocyte

**Indications of ICSI:**

• All indications of IVF + Limitations of IVF
• Most important parameter in ICSI: morphology of sperm
• Most important parameter for IVF: motility > Sperm concentration

• In semen analysis, most important parameter:
  morphology > motility > Sperm concentration
PUBERTY

Puberty in females
- Average (avg) age of puberty 10.5 yrs
- 1st Sign: Growth spurt
- 1st visible sign: Thelarche (breast budding)
  ↓
  Pubarche (appearance of Pubic hair 
  & axillary hair)
  ↓
  Peak height (ht) velocity
  ↓
  Menarche - 12.5 years
  (onset of menstruation)
Mnemonic: Gross BPH in male

Puberty in males
- 11.5yrs
- Testicular Enlargement
  ↓
  Penile enlargement
  ↓
  Pubarche
  ↓
  Peak Height velocity

Precocious Puberty: <8 yrs <9 yrs
Precocious menstruation:
- Menstruation in <40y of age
- Precocious puberty is more common (mc) in females
- Most common cause (mcc) of precocious puberty in females: Idiopathic (90%)
- In 10% cases, precocious puberty can be due to Brain tumors like Hamartomas, therefore, MRI brain- mandatory
- DOC (Drug of choice): Continuous GnRH

Delayed Puberty:
- If 2° sexual characteristics (breast) not developed by 13 yrs
  If 2° sexual characteristic (breast) not developed by 14 yrs
  - Delayed puberty is mc in boys
  - MCC: Constitutional delay
  - DOC: Pulsatile GnRH
Important points about puberty in females:

- Most important hormone for puberty in females: **Estrogen**

  ![Estrogen diagram]

- In females, for pubic hair development & axillary hair development, the hormones that are responsible are **Androgens**

- **Tanner staging** for breast and pubic (change everywhere) hair development

- **Tanner’s stages – 5 stages**
  - 1st/2nd stage - Less or premature breast or pubic hair
  - 4th/5th stage - Breast and (change everywhere) hair are fully matured

**Precocious puberty**

- Precocious puberty (↑estrogen)
  - Central
    - HPO (hypothalamic pituitary ovarian) axis is stimulated early
      - GnRH↑, Luteinizing hormone (LH) and Follicle-stimulating hormone (FSH) estrogen↑
  - Peripheral
    - Not due to HPO stimulation
      - Due to increase in Sex steroids from some other source
    - Sex steroid sources
      - Ovary
        - Ovarian tumor
      - Adrenal
        - Congenital adrenal hyperplasia (CAH) ↑Androgen
          - Heterosexual precocious puberty

- Isosexual Precocious puberty
- Associated with precocious menstruation

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• mCC of peripheral precocious puberty: GCT (granulosa cell tumor)
• Not associated with precocious menstruation
• Another cause of peripheral precocious puberty:
  McCune Albright Syndrome syndrome
  • Precocious puberty
  • Café-au-lait spots
  • Polyostotic fibrous dysplasia

Causes of heterosexual precocious puberty

• CAH
• Androgen secreting tumor of ovary
• Cushing syndrome
• Exogenous androgens

Adrenarche:
• Onset of production of androgens (DHEA and DHEA sulphate) from the adrenal glands
• Normally starts at 6 years of age
PRIMARY AMENORRHEA

Amenorrhea:

- Absence of menstruation

  Primary
  - Never experienced menstruation
  - Does not attain menarche by 13 years of age, in absence of secondary sexual characteristics or
  - 15 years of age irrespective of secondary sexual characteristics or
  - If there is a difference of 3 or more years between the onset of puberty and menarche

  Secondary
  - Not menstruating for > 90 days with previous h/o normal menstruation or
  - If a female who has oligomenorrhea having less than 9 cycles in an year

Primary amenorrhea:

- Hypothalamus
  - GnRH (Pulsatile)
- Pituitary
  - FSH
  - LH
- Ovary
  - Estrogen
  - Progesterone

Causes → Hallmann Syndrome (46,XX)

Causes → Craniopharyngioma

Causes → Gonadal dysgenesis

- Turner Syndrome (45, XO)
- Pure Gonadal dysgenesis
- Swyer Syndrome (46, XY)
- **Causes:** Mullerian agenesis (46XX)
  - Androgen insensitivity syndrome (testicular feminizing syndrome 46XY)
- Most common cause of primary amenorrhea →
  - Main cause → Gonadal dysgenesis
  - 2nd → Turner's syndrome (45XO)
- Second most common cause is → Mullerian agenesis 46XX
- Third most common cause is → Androgen insensitivity syndrome (46XY)
- Best investigation for primary amenorrhea is Karyotyping

### Primary amenorrhea

- **Case A**
  - With secondary sexual characteristics normal
  - Uterus
    - Present
    - Cryptomenorrhea
    - Karyotype
      - 46XX
        - Mullerian agenesis
      - 46XY
        - AIS
- **Case B**
  - With absent secondary sexual characteristics
  - Height of the patient
    - Short stature
      - Turner syndrome
      - Pure gonadal dysgenesis
      - Swyer Syndrome
      - Kallman Syndrome
    - Normal
Cryptomenorrhea

- Normal menstrual cycles without any sign of bleeding

  ↓

  due to some obstruction in genital tract

  ↓

  Imperforate hymen (m.c)

  ↓

  Tranverse vaginal septum

  ↓

  Vaginal atresia

  ↓

  Cystic Abdominal pain every month but there is no bleeding

  • Blood collects in vagina, cervix and uterus

  ↓

  Hematometra

  ↓

  P/A → Uterus may be palpable

• Most common cause of hematometra → Imperforate hymen

• P/V → Contraindicated in a virgin female
  - Virgin female
  - Placenta previa
  - PROM

• P/R → Bulky uterus

Local examination → Tensed bluish bulging hymen
Management:
- Cruciate incision on hymen

**Mullerian agenesis**

- Chromosome number is 46 XX
  \[ Y \text{ chromosome: absent} \]
  \[ \text{Gonads} \rightarrow \text{Ovaries} \]
  \[ \text{Normal} \]
  \[ \rightarrow \text{Ovulation: normal} \]
  \[ \rightarrow \text{Levels of estrogen normal} \]
  \[ \rightarrow \text{Secondary sexual characters: normal} \]

Problem →
- Both mullerian ducts are absent
  - Fallopian tube
  - Uterus
  - Cervix
  - Upper vagina
  \{ Absent \}
- Primary amenorrhea
- Coital difficulties

Structures present in mullerian agenesis:
1. Both the ovaries
2. Lower part of vagina
3. Rarely → distal part of fallopian tube

If female also has:
1. Renal anomalies (15%−30%), MC renal agenesis → Horse shoe shaped kidney
   \[ + \]
2. Skeletal anomalies → Hemivertebra.
   - Scoliosis
   - Spina bifida
   - Abnormalities of cervical spine
3. Auditory problems → Mayer Rokitansky
   Kuster Hauser syndrome
   (MRKH syndrome)
   ↓
   Abnormal galactose metabolism

Diagnosis:
- USG → uterus is absent
- Karyotyping
- Intravenous pyelography

Management:
1. Cannot initiate menstruation in the patient (uterus absent)
   a. Coital difficulties (in future)
      ↓
      Do vaginoplasty

   Best time               Best method
   Just before or → Mc Indoe
   Just after marriage     Vaginoplasty
                           → Veichetty technique
                           → Williams vaginoplasty

3. Female cannot become pregnant but can have own biological child by IVF + Surrogacy
Androgen insensitivity syndrome

- Genotype → 46 XY
  - Gonads → Testis (normal)
    → Are undescended - present as inguinal hernia
    → Increased chances of malignancy
    → Most common tumor which is seen in abdominal testis / dysgenetic gonads
      ↓
      Gonadoblastoma
      → Most common malignant tumor seen in abdominal Testis / dysgenetic gonads
      ↓
      Seminoma
  - Sertoli cells
    ↓
    Secrete mullerian inhibiting factor
    ↓
    MD regresses
    ↓
    Fallopian tube, uterus, cervix, upper vagina
    Absent
    - Wolffian duct regresses
      ↓
      S - Seminal vesicles
      E - Ejaculatory duct
      E - Epididymis
      D - Vas deferens
      Male internal genital organs absent
      Also resistant to Dihydrotestosterone
      External genitalia resembles female
      Considered as a female child at birth
  - Leydig cells
    ↓
    Testosterone
    Males are resistant to testosterone
Child attains puberty

At puberty → Resistant to testosterone

↓

Secondary sexual characteristics of male do not develop

Testosterone ↑

↓

In adipose tissue

Aromatase

Estrogen

Not resistant to estrogen

↓

Breast development occur corresponding to Tanner stage 4 or 5

Note → In females also for pubic hair and axillary hair, the hormone needed is Androgen. Since these children are resistant to androgen, therefore pubic hair and axillary hair do not develop properly (corresponding to Tanner stage 1 or 2)

Definitive diagnosis: Karyotyping

Management:

1. Continue to accept them as females
2. Gonadectomy → After breast development is complete (14 - 16 years of age)
3. Estrogen replacement therapy
4. Vaginoplasty - Technique and time of process same as Mullerian agenesis
5. Cannot become pregnant
6. Cannot have own biological child

<table>
<thead>
<tr>
<th>Complete AIS</th>
<th>Partial AIS</th>
<th>Reifenstein Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>X-linked</td>
<td>Not</td>
<td></td>
</tr>
<tr>
<td>Recessive</td>
<td>X-linked</td>
<td>disorder</td>
</tr>
<tr>
<td>disorders</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Partial androgen insensitivity syndrome

- Males are partially insensitive to testosterone
- Genotype → 46 XY
  ↓
  Gonads → Testis - Intra abdominal
  Patient complains of inguinal hernia.

- Sertoli cells
  ↓
  Antimullerian hormone
  ↓
  Mullerian duct will regress
- Leydig cells
  ↓
  Testosterone (partially sensitive)

  Fallopian tube
  Uterus
  Cervix
  Upper vagina.
  Absent
  ↓
  Wolffian duct grows
  Dihydro - testosterone (Partially sensitive)
  → Appears as female external genitalia resembling male genitalia.
  Present
  E → Hypoplastic
  S

  → Clitoromegaly
  → Fusion of Labio scrotal fold
  → Considered as female child at the time of birth
  → At puberty → Partially sensitive to testosterone
  ↓
  Testosterone ↑ also have masculine characters
  ↓ in adipose tissue
  → Estrogen
  ↓ Token as virilising
  Secondary sexual Characteristics of female → Breast
  Characters
<table>
<thead>
<tr>
<th>Complete AIS</th>
<th>Incomplete AIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complains of inguinal hernia + primary amenorrhea + breast →</td>
<td>Clitoromegaly or labioscrotal fusion</td>
</tr>
<tr>
<td>Tanner 4 or 5 + less pubic hair and axillary hair</td>
<td></td>
</tr>
</tbody>
</table>

**Reifenstein syndrome**

- Androgen insensitivity
  - Very less
  - Wolffian duct grows
  - External genitalia
    - Will be male
      - Hypospadias
      - Or
        - Bifid scrotum
  - Present
  - E (less developed)
  - D
    - Taken as male child

- Male with ↓ Testosterone
  - Undervirilized male
  - Complains of infertility
  - Gynaecomastia

**Turner syndrome:**

- Chromosome number → 45 XO
  - Y chromosome is absent
    - Gonads → Ovaries
      - Only 1 X chromosome present
        - These ovaries not properly developed → Streak ovaries

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Streak ovaries

- No Sertoli cells
  - Anti Mullerian hormone absent
    - Mullerian duct will grow
      - Fallopian tube, uterus, cervix, vagina (upper) present

- No Leydig cells
  - No testosterone
    - Wolffian duct regresses
      - External genitalia are like female

- Hypoplastic estrogen levels decreased
  - Breast development absent
  - Negative feedback on FSH is gone
    - Levels of FSH ↑
      - In these patients there is mutation of SHOX gene → Short stature

- Streak ovaries → No ovulation
  - ↓ Progesterone
    - LH ↑
Hyponadotropic

↑ LH and FSH

Hyponadism

↓ estrogen

Additional features:
1. Cubitus valgus
2. Short 4th metacarpal
3. Low posterior hairline
4. Webbing of neck
5. Shield shaped chest
6. Widely spaced nipples
7. Increases risk of heart disease
   - Bicuspid aortic valve (m.o)
8. Increased chances of autoimmune disease → Diabetes mellitus
   Hashimoto's
   Thyroiditis

Intelligent Quotient is normal in Turner syndrome
In Klinefelter syndrome IQ is decreased

Management:
- Estrogen alone for 1 year for proper breast development
  Estrogen + Progesterone later on

Pure gonadal dysgenesis

- 46 XX → But due to unknown reasons they have streak gonads
  ↓
  All characters similar to Turner syndrome
  Except → 1. Height of the patient is normal
  2. Additional features of turners are absent

Management: are same as turners
Swyer syndrome

- Chromosome number → 46XY

  ↓

  Gonads → Testis

  ↓

  Dysgenetic testis → increased chances of malignancy

  85% cases → Idiopathic

  15% cases → mutation in SRY gene

  ↓

  Testis are not functioning

  ↓

  Sertoli cells

  ↓

  AMH not produced

  ↓

  MO will not regress

  ↓

  Fallopian tube
  Uterus
  Cervix
  Upper vagina

  Present

  Female internal genital organ present

  ↓

  Wolffian duct
  Regress

  ↓

  Male internal genital organ absent

  Female child at the time of birth

  External genitalia resembles females

  ↓

  IUI
At puberty:

- Testis will not form testosterone
- Adrenal glands will produce insufficient testosterone
- Estrogen absent
  - Some pubic hair
  - Some axillary hair
- Breast development absent
  - Delayed puberty
  - Primary amenorrhea

Management:

- Continue to accept them as female
- Gonadectomy as soon as diagnosis is made
- Vaginoplasty
- Estrogen alone for 1 year → Breast development
  - Followed by Estrogen and Progesterone.

Kallman syndrome:

- Defect in neuronal migration of the neurons which form GnRH from the olfactory placode
- Gene → Kal1
- Levels of GnRH are decreased + anosmia
  - LH and FSH ↓
  - Estrogen ↓
    - Complains of primary amenorrhea or infertility
    - Secondary sexual characteristics are absent
- Height is normal
**Management:** GnRH (Pulsatile)

Example of Hypogonadotropic hypogonadism

**Difference between Turner syndrome v/s Kallmann syndrome**

<table>
<thead>
<tr>
<th>Turner Syndrome</th>
<th>Kallmann Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ 45 XO</td>
<td>→ 46 XX</td>
</tr>
<tr>
<td>→ Streak gonads</td>
<td>→ Gonads → Ovaries</td>
</tr>
<tr>
<td>→ LH and FSH are increased</td>
<td>→ LH and FSH are increased</td>
</tr>
<tr>
<td>→ Short stature</td>
<td>→ Normal</td>
</tr>
<tr>
<td>→ Additional features:</td>
<td>→ Decreased</td>
</tr>
<tr>
<td>- webbing of neck</td>
<td>→ Normal stature</td>
</tr>
<tr>
<td>- cubitus valgus etc</td>
<td>→ Anosmia</td>
</tr>
</tbody>
</table>
SECONDARY AMENORRHoeA

- Amenorrhea for >90 days in a previously normally menstruating woman or <9 cycles in a female with oligomenorrhea.
- MC cause of secondary amenorrhea. - Pregnancy
- MC pathological cause of secondary amenorrhea. - PCOS

Causes

Systemic
- Thyroid dysfunction
- ↑ Prolactin levels
- Severe anemia
- Chronic renal failure

↑ Prolactin levels / Hyperprolactinemia.
→ Negative feedback on GnRH
→ ↓ LH / FSH
→ Amenorrhea.
(Cause of lactational amenorrhea.)

Prolactin levels

PRL ≥ 50 ng/dl
↓ MRI
rule out prolactinoma.

≤50 ng/dl
Follow up the patient

Prolactinoma

- Prolactin secreting pituitary tumor

Microadenoma.  
<1 cm

Macroadenoma.  
≥1 cm

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Scanned with CamScanner
Clinical features

↑ Prolactin
↓
↓ GnRH
↓
2° amenorrhoea
↓
Pituitary tumor
↓
Headache
Visual disturbance

Diagnosis: MRI

Management

microadenoma - medical management
MAC-Cabergoline

macroadenoma

↓
DOC: Cabergoline
↓
- If size of tumor ≥3cm /
  patient not responding
  ↓
  surgery

Cabergoline 2.5 mg twice weekly
1. DOC for ↑ Prolactin: Cabergoline
2. DOC for ↑ Prolactin in infertile women: Bromocriptine
HPO axis causes of 2° amenorrhoea

- Stress
  - Anxiety
  - Excessive physical exercise
  - Anorexia nervosa
  - Bulimia nervosa

- Prolactinoma
  - Sheehan's syndrome

- PCOS / Anovulation
  - Premature menopause
  - (<40 yrs)

Management of Asherman's
  1) Hysteroscopic adhesiolysis +
     - Balloon catheter insertion
  2) Estrogen + progesterone

Sheehan's syndrome

Pituitary gland enlarges during pregnancy by 135%

mainly anterior lobe
Post delivery
↓
PPH
↓
Blood supply to all organs ↓ including pituitary
↓
Necrosis
↓
Sheehan's syndrome

Clinical Features
All hormones formed by anterior pituitary ↓

↓ Prolactin ↓ TSH ↓ FSH
↓ Inability to breast feed ↓ Cold intolerance ↓

\(^{a}\) amenorrhoea.
Work-up for secondary amenorrhea

Hormonal study (1st or best investigation)

↓

UPT  TSH  Prolactin

Normal

↓

Progesterone challenge test

↓

medroxy progesterone acetate
7-10 days & then withdraw

↓

Bleeding +

↓

Anovulation / PCOS

Bleeding -

↓

Next step

estrogen & progesterone challenge test

Estrogen x 21 days
Last 10 days add progesterone

↓

Bleeding +

↓

Check FSH levels

Bleeding -

↓

Uterine cause
Asherman's syndrome

↓

Premature menopause

↓

Pituitary / Hypothalamus MRI

↓

Empty sella turcica

↓

Sheehan's syndrome

↓

Space occupying lesion

↓

Normal

↓

Hypothalamic Prolactinoma.
HYSTERECTOMY

Female genital tract: related anatomy

* Most common site of ureteric injury during Hysterectomy: Water under Bridge

Course & Relations

- Inferior mesenteric artery
- Common iliac artery
- Middle sacral vessels
- Superior rectal artery (superior rectal nerve)
- Ovarian artery
- Internal iliac artery
- External iliac artery
- Middle rectal artery
- Obturator artery and nerve
- Uterine artery
- Umbilical artery
- Inferior vesical artery
- Vaginal artery
- Round ligament (superior vesical nerve)
- Obturator umbilical artery
- Inferior epigastric artery and vein
- Superior vesicle arteries

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### Piver Rutledge classification

- **Types of Hysterectomies and structures removed:**

<table>
<thead>
<tr>
<th>Types of Hysterectomy</th>
<th>Structure Removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ Type I Hysterectomy / Simple Hysterectomy / Total Abdominal Hysterectomy (TAH)</td>
<td>→ Uterus + Cervix → Depending on age of female</td>
</tr>
<tr>
<td>mC type of hysterectomy done for benign cause; most common indication</td>
<td>↓ ↓</td>
</tr>
<tr>
<td>↓ Fibroid</td>
<td>&lt; 45 ≥ 45</td>
</tr>
<tr>
<td></td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>Do not resect</td>
</tr>
<tr>
<td></td>
<td>resect ovary</td>
</tr>
<tr>
<td></td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>Bilateral Salpingo-oophorectomy (BSO)</td>
</tr>
<tr>
<td></td>
<td>TAH + BSO</td>
</tr>
<tr>
<td></td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>A/V/A pan hysterectomy</td>
</tr>
</tbody>
</table>

- → Type II Hysterectomy / Wertheims Hysterectomy / modified Radical Hysterectomy

- → Type III Hysterectomy / Radical Hysterectomy

- → Uterus + Cervix + both tubes + both ovaries + 1 cm of vagina + medial half of cardinal ligament + medial half of utero sacral ligament + part of uterine artery

- → Uterus + cervix + both tubes + both ovaries + entire cardinal ligament + entire uterosacral ligament + major part of uterine artery

- **m.c site of injury to ureter during hysterectomy:**
  - water under bridge / where uterine artery crosses over the ureter
• 1st site: At pelvic brim
• Mc ureteric injury is seen in: Simple Hysterectomy
• Maximum risk of ureteric injury is with: Wertheim's Hysterectomy

Routes and steps of hysterectomy

Routes

Abdominal

Vaginal

Open
Hysterectomy

Laparoscopic
Hysterectomy

Steps of Abdominal Hysterectomy:

Clamps in TAH:
• 1st Clamp: Tubo Ovarian Round Ligament Pedicle
• 2nd Clamp: Uterine Artery
• 3rd Clamp: Cardinal ligament

Risk for ureteric injury:
Laparoscopic hysterectomy > Abdominal Hysterectomy > Vaginal Hysterectomy > Caesarean hysterectomy
- 4th Clamp: Uterosacral ligament

→ In vaginal hysterectomy: order of clamp is reversed

- If during hysterectomy ovary is not to be removed → Infundibulo pelvic ligament is not cut

**Vaginal hysterectomy**

**Advantages:**
1. No visible scar
2. Complications - less
3. Hospital stay - less
4. Quickly mobile
   ↓
   Less chance of thromboembolism
5. Early resumption of daily activities
6. Best for obese and elderly female

**Disadvantages:**
1. Abdominal and pelvic organs cannot be explored
2. Tubo ovarian pathology cannot be dealt with at the same sitting
3. Difficult to perform if size of uterus > 12 weeks pregnant uterus size
4. Difficult to perform if adhesions are present

**Maylard incision:**
1. Above and parallel to Pfannensteil incision
2. Rectus Abdominus muscle is cut
3. Better visibility to explore pelvic cavity
4. Used in gynae - onco surgeries

* incision used to access Retro pubic space /
Space of Retzius → Cherney incision
ENDOMETRIAL CANCER

ENDOMETRIAL HYPERPLASIA

* Types

1. Simple
   - (1) Without atypical cells
   - (2) With atypical cells

2. Complex
   - (3) Without atypical cells
   - (4) With atypical cells

a. Without atypical cells
b. With atypical cells

Changes of malignancy

1) Simple Hyperplasia without atypical cells
   - least chance of malignancy
   - AHA cystic Glandular Hyperplasia

2) Complex Hyperplasia without atypical cells
   - 3%

3) Simple Hyperplasia with atypical cells
   - 8%

4) Complex Hyperplasia with atypical cells
   - Maximum chance of malignancy
   - 30%

* Due to excessive Estrogen

  Proliferates endometrium

  Complains of: Excessive bleeding

  1st investigation: Trans vaginal sonography (TVS)

  Check endometrial thickness

  Pre-menopausal
  - ≥ 12mm
  Post menopausal
  - ≥ 5mm (FISG)
  - ≥ 4mm (ACOG)

10C: Endometrial Aspiration Biopsy
Endometrial biopsy

- Endometrial Aspiration Biopsy
  - OPD procedure
  - Done using Pipelle / Vabrag aspirator / Karman's cannula
  - No anesthesia needed

IOC: Endometrial Aspiration Biopsy

Gold standard investigation:
Fractional Curettage (7 separate samples taken)

2nd best: Dilatation and curettage

Endometrial Hyperplasia is a histopathological finding not a clinical diagnosis

Management of endometrial hyperplasia

- Report: Endometrial Hyperplasia is present
- Management:

  without atypical cells
  → Simple = 1%
  → Complex = 3%

  with atypical cells
  → Simple = 6%
  → Complex = 30%

  IOC: Progesterone

  → Medroxyprogesterone Acetate (12-14 days in a month)

  Total Abdominal Hysterectomy (Best) ±
  Bilateral salpingo-ophorectomy
  young patient; refuses hysterectomy

  Progesterone = megestrol
Endometrial cancer

- Hyperestrogenic condition

**Risk Factors**

1. Nulliparity
2. Obesity
3. Early menarche
4. Late menopause

**Protective Factors**

1. Multiparity
2. Pregnancy
3. Physical exercise
4. Smoking
5. OCPs

- Risk factors for Endometrial cancer:
  - Family History: Hereditary Nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome: Gene mutations of
    1. MLH1 / MSH2 / MSH-6 / EPICAM gene
    2. BRCA 1 / BRCA 2
  - Hypertension
  - Obesity
  - Late menopause, early menarche
  - Diabetes
  - Atypical Endometrial Hyperplasia
  - Unopposed Estrogen $\rightarrow$ HRT (only Estrogen)
    $\rightarrow$ Endometrial cancer
    $\rightarrow$ not ovarian cancer
    $\rightarrow$ PCOS
    $\rightarrow$ Estrogen secreting ovarian Tumor

- Nulliparity
- Tamoxifen
- History of infertility

- Obesity + Hypertensive + Diabetic female has $\uparrow$ chances of Endometrial cancer $\rightarrow$ Corpus cancer syndrome

- MC variety of Endometrial cancer $\rightarrow$ Adenocarcinoma (Endometroid variety)

- Histopathological Examination:
  1. Back to back arrangement of glands
  2. Desmoplastic stroma
Grading of adenocarcinoma

- Grades

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Undifferentiated Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Well differentiated</td>
<td>&lt; 5%</td>
</tr>
<tr>
<td>II</td>
<td>Moderately differentiated</td>
<td>5-50%</td>
</tr>
<tr>
<td>III</td>
<td>Poorly differentiated</td>
<td>≥ 50%</td>
</tr>
</tbody>
</table>

- Most malignant variety of endometrial cancer
  - Clear cell carcinoma (HPE: Hobnail Nucleus)
  - 2nd most malignant: Papillary serous Tumor

- All endometrial cancers can be divided into:

  **Type I**
  - Adenocarcinoma Grade I
    - Grade 2
  - Better prognosis
  - Associated with
    - ↑ Estrogen
    - M.C in obese women
    - ↑ Common in white women
  - Associated with mutation in KRAS and PTEN genes

  **Type II**
  - Adenocarcinoma Grade III
  - Clear cell carcinoma
  - Papillary serous tumour
  - Bad prognosis
  - Not associated with
    - ↑ Estrogen
    - M.C in thin women
  - ↑ Common in black
  - Mutation in P53 gene

- P53 → Gatekeeper for endometrial cancer

Important points on endometrial cancer

- M.C age: 5th - 7th decade
- M.C symptom: Irregular vaginal bleeding (best)
  - 2nd best: Post menopausal bleeding (PMB)
- Most specific symptom: Post menopausal bleeding (PMB)
• Other symptoms:
  
  May be seen  Not seen
  
  1. Pus in uterus  → Loss of appetite
     ↓
    pyometra  → weight loss
            → Cachexia

  2. Foul smelling watery discharge  ↓
     ↓
    Hydorrhea

  3. Pain in abdomen  ↓
     At same time every day  ↓
    AKA simson pain

Post menopausal bleeding (PMB) and pyometra

• MC cause of PMB: Senile Endometrial Atrophy
• MC cause of PMB in India: Cancer cervix
• MC cancer causing PMB: Cancer cervix
• % of PMB patients with Endometrial cancer: 10%
• 1st investigation to do in PMB: TVS
  ↓

Endometrial Thickness

→ Endometrium more thick at a particular place

→ Uniformly thick endometrium
  
  RCOG ≥ 4 mm
  
  FIGO ≥ 5 mm

  ↓

  IOC: Endometrial Aspiration Biopsy

  IOC: Hysteroscopy and Biopsy

Gold standard: Fractional curettage
Pyometra.
- m.C cause of pyometra: Senile Endometritis
- m.C cancer causing pyometra: Endometrial cancer
- m.C cancer causing pyometra in India: Cancer cervix

Screening and staging in endometrial cancer

- m.C route of spread: Direct spread
- m.C site of recurrence: vagina.
- Routine screening is not done
- Screening is done only in HNPCC patients
- Age: 35 years
- Screening method: Fractional curettage (Best)
- Best method to prevent Endometrial carcinoma in patients of HNPCC

\[
\text{Screen for colon cancer by doing colonoscopy (ACOG 2014)}
\]

\[\downarrow\]

→ TAH + BSO after childbearing is complete

→ Not later than 40 years

Staging procedure
- Surgical staging:

Biopsy: Endometrial cancer (+)

\[\downarrow\]

MRI

Surgery → TAH + BSO

\[\downarrow\]

HPE

\[\downarrow\]

Staging

\[\downarrow\]

Post operative management

- Steps of surgical staging in Endometrial cancer
  1. MRI → To know extent of myometrial involvement
  2. Surgery → TAH + BSO
- If cervix or any other structure below cervix is involved
  \[\downarrow\]
  Wertheim's Hysterectomy

3. Lymph Node Dissection

- Type 1 Endometrial cancer
  (Adenocarcinoma grade 1,2)
  \[\downarrow\]
  Extent of spread
  \[\downarrow\]
  Limited to uterus
  \[\downarrow\]
  mR1 report
  \[\downarrow\]
  \(< 50\%\) of myometrium
  \[\downarrow\]
  Size of tumour
  \[\downarrow\]
  \(< 2\ cm\) no lymph node dissection

- Type 2 Endometrial cancer
  (Adenocarcinoma grade 3 papillary serous Tumour
  clear cell carcinoma)
  \[\downarrow\]
  Spread outside uterus
  \[\downarrow\]
  Pelvic + para aortic lymph Node Dissection
  \[\downarrow\]
  \(\geq 50\%\) of myometrium
  involved
  \[\downarrow\]
  \(< 2\ cm\) pelvic lymph node dissection
FIGO staging of endometrial cancer

Stage I: Cancer limited to uterus
   A: < 50% myometrium involved
   B: ≥ 50% myometrium involved

Stage II: Cancer spreads to cervix

Stage III: Cancer spreads beyond uterus and cervix
   A: serosa or Adnexa involved
   B: vagina involved
   C: LN involved
   C₁: pelvic LN involved
   C₂: Para-aortic LN involved

Stage IV: metastasis occurred
   A: Regional metastasis = Bladder or Bowel
   B: Distant metastasis
   Or
   Superficial inguinal lymph node involved

Role of MRI:
1. Extent of myometrium
2. Lower uterine segment involvement
3. Cervix involvement

Role of CT:
1. Pelvic spread
2. Lymph node involvement
3. Metastasis
**Post-operative management**

- Management of choice in Endometrial cancer → Radiotherapy
- Exceptions:
  1. In stage IA Adeno carcinoma Grade 1, 2: Post operative therapy not needed
  2. In stage III and IV: Chemotherapy ± Radiotherapy

| Paclitaxel | Paclitaxel |
| Carboptatin | Doxorubicin |
| + | + |
| Cisplatin (Tap Regime) |

<table>
<thead>
<tr>
<th>Stage</th>
<th>Staging</th>
<th>Post operative Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Surgery</td>
<td>LN Dissection</td>
</tr>
<tr>
<td>Stage IA Grade 1,a</td>
<td>TAH + BSO</td>
<td>≤ 2 cm; No LN Dissection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 2 cm: Pelvic LN Dissection</td>
</tr>
<tr>
<td>Stage IA Grade 3 Stage 1B</td>
<td>TAH + BSO</td>
<td>Pelvic + Para Aortic LN Dissection</td>
</tr>
<tr>
<td>Stage II (cervix)</td>
<td>Wertheim’s Hysterectomy</td>
<td>Pelvic + para aortic LN Dissection</td>
</tr>
<tr>
<td>Stage III Stage IV</td>
<td>Debulking surgery</td>
<td>Pelvic and para aortic LN dissection</td>
</tr>
</tbody>
</table>
CANCER CERVIX

Anatomy of cervix

Transformation zone / Squamocolumnar junction
Endocervix
External OS
Exocervix / Portio vaginalis / Ectocervix
Vagina

i) Endocervix
   - Lies close to uterus
   - Lined by columnar epithelium
   - Per speculum examination—appears red in colour

ii) Exocervix
    - Part of cervix that lies in vagina.
    - Lined by stratified squamous epithelium
    - Per speculum—appears pink

iii) External OS
    - Part where cervix opens into vagina.
    - Shape
      - Pin point / Circular in Nullipara.
      - Transverse / Slit-like in multipara

iv) Transformation zone
    - Squamocolumnar junction
    - Columnar epithelium of endocervix converts to squamous epithelium of exocervix
    - Dynamic point [Depends on hormonal influence & age of the patient]
    - Lies originally at the level of external OS
    - Transformation zone moves outward (towards exocervix)
      under is ↑ hormonal influence [3P's]
- Puberty
- Pregnancy
- Pills

* moves inwards when there is no hormonal influence
  - menopause

* Classification of transformation zone
  Type 1 - lies entirely on exocervix [clearly visualized by colposcopy]
  Type 2 - part of it is in endocervix [can still be visualized by colposcopy]
  Type 3 - entirely inside endocervix [cannot be visualized]

* Per speculum - Appears white (pale)

>Cervix Per speculum examination

>Endocervix (red)
>Exocervix (pink)
>External os
>Transitional zone (white)

Cancer cervix – introduction

00:09:47

1. Most common cancer of cervix - squamous cell carcinoma.
2. Most common site for cancer cervix - Transformation zone
3. Most common site for adenocarcinoma of cervix - Endocervix

4. Lymphatic Drainage of cervix
   H - Hypogastric Lymph nodes [LN]
   O - Obturator Lymph nodes
   P - Paracervical / Ureteric Lymph Nodes
   E - External iliac Lymph Nodes

* Most common lymph node involved in cancer cervix - obturator LN
* Sentinel LN for cancer cervix - ureteric or paracervical LN
* Superficial inguinal LN
  - Not involved in cancer cervix
  - If involved ⇒ Indicates metastasis [i.e. stage IV-B]
Cancer cervix – pathogenesis & Bethesda classification

1. Pathogenesis of cancer cervix
   • Metaplasia
     - Conversion of columnar epithelium of endocervix
       ↓
       Squamous epithelium of exocervix
     - Metaplasia →
       * Absolutely normal
       * Physiological
       * Seen in all females
       * Not precancerous
   • Dysplasia
     In case of cervical infection in females

          Resolves on its own    Persistence of infection
                                          ↓
                                          Dysplasia
                                          * Abnormal
                                          * Pathological
                                          * Precancerous

Classification of Dysplasia

CIN - cervical intraepithelial neoplasia.

CIN 1 - Dysplasia involving ≤ ½ of thickness of cervix
CIN 2 - ½ = ¾ thickness
CIN 3 - ≥ ¾ thickness

Cancer in situ - involves entire thickness of cervix, but overlying membrane is intact
Invasive cancer cervix - overlying membrane is broken

a. Bethesda classification
   CIN - 1 - / LSIL [Low squamous intraepithelial lesion]
   CIN - 2
   CIN - 3 _HSIL [High squamous intraepithelial lesion]
Cancer in situ

Note:
Dysplastic cell - ↑ nucleo - cytoplasmic ratio
Cancer cervix – risk factors

- Most important - HPV (Human Papilloma Virus) infection
  - Increase of HPV infection
  - Intercourse at <18 yrs of age
  - Multiple partners
  - Poor hygiene
  - Low socio-economic status
  - Multi parity

- Smoking
- Immunocompromised states

- Oral contraceptive pills → Adenocarcinoma of cervix
- In utero exposure to DES (Diethylstilbestrol)

Note:
No role for
- Familial inheritance
- Early menarche & Late menopause

Human Papilloma Virus [HPV]

- Epitheliotrophic virus
- Koilocyte

Virus infects epithelium

↓

Nucleus-pusnea towards periphery

↓

Is surrounded by perinuclear halo

Koilocyte
* Viral proteins - Required
  * To attach virus to epithelium → L₁, L₄
  * For viral replication → E₆, E₇
  * For malignant transformation → E₆, E₇
    - E₆: Suppresses p53 gene
    - E₇: Suppresses Retinoblastoma gene

* Diagnosis of HPV - Test for HPV-DNA
  i) PCR
  ii) Southern Blot
  iii) Hybrid capture [Etest]
    - Done in females ≥ 30 yrs age

* HPV Subtypes
  i) Low risk subtype
    - HPV 6, 11 - Genital warts
  ii) High risk subtype
    - HPV 16, 18, 31, 33, 35, 39, 45, 51, 52, 58, 59, 68
    - HPV 16, 18

  ↓

  Male                           Female
  ↓

  Cancer  Cancer cervix,
  penis, anus vulva, vagina

* One liners in HPV
  i) Most common HPV associated with cancer cervix - HPV 16
  ii) Most common HPV associated with squamous cell cancer of cervix - HPV 16
  iii) Most specific HPV for cancer cervix - HPV 18
  iv) Most common HPV associated with adenocarcinoma of cervix - HPV 18
Human papilloma virus – vaccines

HPV vaccines are made from inactivated capsid proteins

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Divalent</th>
<th>Quadrivalent</th>
<th>9-valent</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Brand</td>
<td>Cervirax</td>
<td>Gardasil</td>
<td>Gardasil-9</td>
</tr>
<tr>
<td>* HPV-subtype</td>
<td>16, 18</td>
<td>16, 18, 6, 11</td>
<td>16, 18, 6, 11, 31, 33, 45, 52, 58</td>
</tr>
<tr>
<td>* Protects against</td>
<td>CIN, cancer cervix</td>
<td>CIN, cancer cervix, genital warts</td>
<td>CIN, cancer cervix, epithelial cancer of vulva and vagina</td>
</tr>
<tr>
<td>* Dose</td>
<td>0.5ml, contains - HPV 16 = 20 mcg - HPV 18 = 20 mcg</td>
<td>0.5ml, contains - HPV 16 = 40 mcg - HPV 18 = 20 mcg - HPV 6 = 20 mcg - HPV 11 = 40 mcg</td>
<td>0.5 ml</td>
</tr>
<tr>
<td>* Dosage schedule</td>
<td>0, 1, 6 months</td>
<td>0, 2, 6 months</td>
<td>0, 2, 6 months</td>
</tr>
<tr>
<td>* Route</td>
<td>1.m (Intra muscular)</td>
<td>1.m</td>
<td>1.m</td>
</tr>
<tr>
<td>* Administered to</td>
<td>Girls</td>
<td>Girls &amp; Boys</td>
<td>Girls &amp; Boys</td>
</tr>
<tr>
<td>* Adjuvant</td>
<td>Aluminium sulphate [Adjuvant system 04]</td>
<td>Alum</td>
<td>Alum</td>
</tr>
</tbody>
</table>

One liners in HPV vaccine:
* Age of vaccine administration
  - 9-10 yrs
  - Ideal age is 11-12 yrs
* Most common complication – syncope
• Contraindication
  - Pregnancy
  - Age < 9 yrs
  - Hypersensitivity

One liner in HPV infection
• most common age group for HPV infection → 20-24 yrs
• most common Sexually Transmitted Disease (STD) in USA → HPV
• most common age - CIN → 20-29 yrs
  - Cancer insitu → 20-35 yrs
  - Cancer cervix → 35-39 yrs & 55-60 yrs- bimodal distribution

Diagnosis of cancer cervix and cervical intraepithelial neoplasia

Screening methods

- Pap smear
- Papsmear Co-test
  - [HPV-DNA testing + pap smear]
  - women ≥ 30 yrs

Diagnostic methods

- Rural areas
  - VIA [visual inspection with acetic acid]
  - VILI [visual inspection with Lugol’s iodine]

Lesion-visible
  → Punch biopsy

Lesion-not visible
  → Colposcopy

Papsmear

Screening is universal for cancer cervix

Pap Test
  Age to start: all years irrespective of 1st intercourse

• method of pap smear
  • Instrument used - Ayer’s spatula, Endocervical brush

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Ayerman's spatula  
Endocervical brush

1) Sample site
- Slide 1 - The concave end of Ayerman's spatula is rotated 360° in the transformation zone (near external os) and sample is taken - To detect squamous cell cancer
- Slide 2 - The endocervical brush is rotated in endocervix and sample taken - To detect adenocarcinoma
- Slide 3 - Control slide
  - The other end of Ayerman's spatula is taken to rotate in posterior wall of vagina and sample taken

Ayer's spatula - transformation zone

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Slides of Pap smear

A. Sample site for cytology (Pap smear) - Posterior wall of vagina.
B. Sample site for Hormonal study - Lateral wall of vagina.

iii) Fixative - 95% Ethyl alcohol

- Liquid based cytology
  - Instrument - Cervical brush
  - Fixative - Methanol

ii) No slide preparation done

- Guidelines for Pap smear

  Pap smear was 1st performed by Georgios Papanikolaou
i) Start pap smear – At 21 years of age
   ↓
   Repeat every 3 years
   ↓
   Till woman becomes 29 years of age
   ↓
   From 30-65 yrs of age
   - Pap smear & HPV DNA testing
   (Co-test) done every 5 years
   (or)
   - Continue Pap smear, every 3 year

ii) Stop pap smear – At 65 years
    Criteria to stop →
    • a cotest / 3 pap test – Normal for past 10 years
      consecutively
    • Hysterectomy done for a benign cause

iii) HIV positive female
    • Annual screening
    • Uncertain to stop screening at 65 years

iv) Female treated for CIN-2, 3 & cancer in situ or cancer cervix
    ↓
    Continue screening for 20 years (at least)

v) If a female has taken HPV vaccine
    ↓
    Continue screening as usual

Rural areas – screening methods

<table>
<thead>
<tr>
<th>Active Space</th>
<th>Dysplasia +</th>
<th>Dysplasia - [Normal epithelium]</th>
</tr>
</thead>
<tbody>
<tr>
<td>VIA[5% acetic acid]</td>
<td>white colour (stained)</td>
<td>Pink colour [unstained-Normal in appearance]</td>
</tr>
<tr>
<td>VILI[3-5% Lugol’s iodine]</td>
<td>unstained [glycojen-used up]</td>
<td>Brown / mahogany colour [stained]</td>
</tr>
</tbody>
</table>
Report & action of pap smear

Report - 1
* Normal report
  ▼
  Continue screening

Report - 2
* Unsatisfactory test
  ▼
  Repeat pap test after 2-4 months

Report - 3
* Infection
  ▼
  Antibiotics given, Repeat pap test after 2-4 months

Infection
  ▼
Pap-can detect
  ▼
  i) Human papilloma virus
  ii) Bacterial vaginosis
  iii) Herpes simplex virus
  iv) Trichomonas
  v) Candidia

Pap-cannot detect
  ▼
  i) Chlamydia
  ii) Gonorrhea

Report 4
* ASCUS [Atypical Squamous Cell of Unknown Significance]
  ▼
  5-10% chance in conversion of ASCUS to CIN -2, 3

ASCUS
  ▼
  Age 18-24 yrs
  ▼
  Repeat pap smear
  ▼
  Abnormal
  ▼
  Colposcopy
  ▼
  Colposcopy
  ▼
  Cytotest
  (after 3 yrs)

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Report-5

- LSIL

  ↓

  Growth-visible Growth - not visible

  ↓

  Punch biopsy Colposcopy [colposcopy guided Biopsy]

  +/-

  Endocervical curettage / Sampling

[In case of HSIL, Growth-not visible → Endocervical sampling is a must]

Special situation

- LSIL: Female (21-24 years)

  ↓

  Follow up with cytology [colposcopy-contraindicated]

- LSIL in post menopausal woman

  ↓

  - Repeat pap in 6-12 months if positive → Colposcopy

  - HPV - DNA test if positive → Colposcopy

  - Colposcopy

- LSIL in pregnant female

  ↓

  Wait for 6 weeks after delivery before performing any further testing

- In HSIL

  All cases are followed up with colposcopy & biopsy

Other reports and actions of PAP smear

Case scenario:

Pap smear - Normal, HPV-DNA-positive

↓

Repeat co-testing in 12 months

↓
↓
If HPV-DNA test persists
↓
Colposcopy

Report 6
• ASC-H [Atypical squamous cells, HPE-cannot exclude HSIL]
  ↓
25% chance of conversion of ASC-US to CIN-2, 3
ASC-US
↓
Colposcopy done, irrespective of age (or) HPV-DNA testing

Report 7
•
↓
HSIL
↓
Lesion - Visible
↓
Punch biopsy
↓
Lesion - Not Visible
↓
• Colposcopy
  + (Best)
  Endocervical curettage
• Alternative - visualise &LEEP
  [Loop electro excisional procedure]

Report 8
• Atypical glandular cells
  ↓
Suggests - Endometrial cancer (or) Cancer cervix
  ↓
Test done - Endometrial biopsy + Colposcopy & Endocervical curettage
MANAGEMENT OF CANCER CERVIX

Diagnosis of cervical intraepithelial neoplasia (CIN)

1. Colposcopy
   - magnifying instrument
     - minimum magnification (5x)
     - maximum magnification (40x)
     - OPD procedure
     - No anaesthesia
     - Sensitivity = 50-80%
     - Colposcopy = gold standard [To Evaluate Abnormal Cervical Cytology]

Procedure of colposcopy

1) Biopsy from a rough area
   - Apply 3-5% acetic acid
     - Dysplastic area
     - Normal
     - Metaplasia
       - Appears white
       - Appears pink
       - Appears grey
         (Aceto-white)

2) Biopsy from aceto-white area
3) Biopsy from area with abnormal blood vessels
   - blood vessels such as - mosaic
     - Comma shaped
     - Reticular
     - Punctate

Disadvantage of Colposcopy
- upper 3/4 of endocervix cannot be visualised
Case Scenario

Pap smear - High-grade squamous intraepithelial lesion (HSIL)
Colposcopy - Normal

Earlier - Cone Biopsy  Now - Endocervical sampling/curettage

- Since, it can be a case of dysplasia in endocervix

a. Cone Biopsy

* OT procedure
* Under General Anaesthesia (GA)
* Biopsy must include
  - Endocervix
  - Exocervix
  - Transformation zone

* Complication of cone biopsy
  - Bleeding
  - Injury to internal OS
    \[\downarrow\]
    2nd trimester recurrent abortions

* Indications for Cone Biopsy (Conization)

  Diagnostic  Therapeutic

- Transformation zone in endocervix (or)
- Transformation zone - not completely visible
- Unsatisfactory colposcopy
  [Limit of lesion - not visible]
- Suspecting Adenocarcinoma / Adenocarcinoma, in situ
- Endocervical curettage - positive

- Cancer insitu (or)
  Stage IA, of cancer cervix in young Female
Management of cervical intraepithelial neoplasia

i) Management

- CIN-1 (usually regresses by 6yrs)
  - Follow up by cotesting after 1 year

- CIN-2 & 3
  - LEEP / LLETZ (at any age / parity)
  - Hysterectomy

- Recurrent CIN
  - CIN extending to vaginal fornix
  - Laser

- Negative
  - Persist after 6yrs

  - Routine screening
  - Cryotherapy

Note:
- CIN-1 in 31 – 34 years of age
  - Follow up even if it persists
- Hysterectomy is not done to manage CIN-2 & 3 in any age/parity

ii) LEEP/LLETZ

LEEP  – Loop Electrosurgical Excision procedure

LLETZ  – Large loop Excision of Transformation zone
- OPD procedure
- Under LA (local anaesthesia)
- No admission required
- Cuts & coagulates at same time

- Tissue can be removed up to a depth of 10mm
Cancer cervix

- Most common gynaecological cancer in world
- 2nd most common cancer seen in female worldwide [most common - breast cancer]
- Most common type of cancer cervix - squamous cell carcinoma
- Most common site of cancer cervix - Transitional zone
- Most common site of adenocarcinoma cervix - endocervix
- Most common symptom - irregular bleeding > post coital bleed / contact bleed
- Most specific symptom - post coital bleed

Case scenario
Female with complaints of post coital bleed

- Lesions - visible
  - Punch biopsy
  - If abnormal (or)
  - If bleeding for 24 weeks
  - Colposcopy

- No lesion - visible
  - Pap smear

Cancer cervix

- Most common route of spread - lymphatic spread
- Most common site of metastasis - lymph node
- Most common site of hematogenous spread - Lungs
- Most common cause of death in cancer cervix - uremia / Renal failure
- 2nd most common cause of death in cancer cervix - Hemorrhage

Cancer cervix – staging

- Based on clinical staging
- Investigation → Staging of cancer → Surgery
- Investigations recommended by FIGO-2018
  (Earlier not recommended by FIGO)
  - Ultrasound (USG)
  - MRI (Magnetic Resonance Imaging)
  - CT (Computed Tomography)
  - PET scan (Positron Emission Tomography)

- Best investigation for radiological assessment of tumor ≥1cm size
  ↓
  MRI

- Best investigation for nodal metastasis → PET scan

- Suspected bladder/bowel involvement should be confirmed by biopsy
  ↓
  Bulbous edema of bladder / bowel does not confirm stage - IV

- Cystoscopy / Sigmoidoscopy - Indications
  i) Patients with related symptoms
  ii) Barrel shaped growth on endocervix extending to anterior vaginal wall [may involve bladder]

New FIGO staging of cancer cervix

Stage 1
  - Cancer limited to cervix
  - Extension to uterus is disregarded

- Stage 1A
  - Microinvasive
  - Depth of invasion <5mm
    - 1A
      ↓
      A₁
      ↓
      Depth <3mm
      A₂
      ↓
      Depth = 3-4mm

- Stage 1B - Invasive
  - Depth of invasion ≥5mm
  - 1B
    ↓
    B₁
    ↓
    <2cm
    B₂
    ↓
    ≥2cm
    B₃
    ↓
    ≥4cm
Stage - a

- Upper 2/3rd of vagina involved
  - Stage a
    - A
    - B
      - No parametrium involvement
      - Parametrium involved
        - Stage 2A
          - $aA_1$
          - $aA_2$
            - Size < 4cms
            - Size ≥ 4cms

Stage - 3

- Lower 1/3rd of vagina involved
  - Stage 3
    - 3A
    - 3B
    - 3C
      - Pelvic sidewall not involved
      - Pelvic side wall involved (or)
        - Hydroureter / Hydronephrosis
          - 3C_a
            - Pelvic lymph node
          - Para-aortic lymph node

Stage - 4

- Metastasis
  - Stage 4
Management of cancer cervix – principles

a. Radiotherapy can be used in all stages of cancer cervix [stage I-IV]
b. Radiosensitizer → ↑ sensitivity of squamous cells to radiotherapy
   - Radiosensitizer used in cancer cervix → Cisplatin
     [Cancer cervix → Chemoradiation]
c. Cancer cervix rarely involves ovaries
   ∴ in young females → Ovaries are not removed (in surgical management of cancer cervix)
d. Stage [IA1–IIA] → Surgery
   Stage [IB1, IIB1, IIA2] → Chemoradiation
   (Because surgery is not preferred for tumor size ≥ 4cm
   Stage [IB2, IIA2] → Chemoradiation

Management of cancer cervix- operative management

<table>
<thead>
<tr>
<th>Stage</th>
<th>Young</th>
<th>Old</th>
<th>Lymph node Dissection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage IA1</td>
<td>* Conization</td>
<td>* Type I / Total abdominal Hysterectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pelvic Lymph Node Dissection</td>
</tr>
<tr>
<td>Stage IA2</td>
<td>* Conization / Radical Trachelectomy</td>
<td>* Type II (Werthen's) / modified Radical Hysterectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage IB1</td>
<td>* Radical Trachelectomy</td>
<td>* Type III / Radical Hysterectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage IB2</td>
<td>* Type III Hysterectomy</td>
<td>* Type III / Radical Hysterectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage IIA1</td>
<td>* Size &lt;2cm – Radical Trachelectomy</td>
<td>* Type III / Radical Hysterectomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>* Size &gt;2cm – Type III Hysterectomy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Note:
1) Radical Tracheectomy
   - Removal of cervix + parametrium & anastomosis of uterus & vagina
   - Done only if size of tumor ≤2cm
   - Mode of delivery in a woman post- radical tracheectomy → Cesarean section

Post operative management of cancer cervix

Risk Factors

Intermediate Risk Factors
- Size ≤4cm
- Stromal invasion present
- Lymph vascular space involved

High Risk Factors
- Positive nodes
- Positive margins
- Parametrial involvement

Stage IA1 - No post operative management

All stages except IA1,

No risk factors

Intermediate Risk

- PORT (post-operative radiotherapy)

- Chemoradiation (Cisplatin)

- EBRT = 45-60Gy (external beam radiotherapy)

Stage IB - IV
Stage IIA & IIB

Chemoradiation
Radiotherapy in cancer cervix

Brachytherapy
(or) ERBT
ICRT
(Intracavitary Radiotherapy)

Brachytherapy (intra-cavitory)

- Isotope
  - Low - dose - cesium 137
  - High - dose - iridium 192

- a reference points

<table>
<thead>
<tr>
<th></th>
<th>Point A</th>
<th>Point B</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Location</td>
<td>2cm above 9</td>
<td>2cm lateral to external os</td>
</tr>
<tr>
<td>- Structures</td>
<td>Paracervical / parametrial lymph nodes</td>
<td>Obturator lymph nodes</td>
</tr>
<tr>
<td>- Dose</td>
<td>80gy</td>
<td>60gy</td>
</tr>
</tbody>
</table>

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CANCER OVARY - 1

Functional ovarian cyst

Ovarian tumour / Adnexal mass:
  Functional ovarian cyst
  Benign tumour
  Borderline tumour
  Malignant tumour

Functional ovarian cyst:
  Related to temporary hormonal disturbances

- m.C → Follicular cyst
  [Cyst → Follicle ≥ 3 cms]

- m.C cyst to rupture → Corpus luteal cyst

- Theca lutein cysts: due to ↑ hCG
  Causes: Molar pregnancy
  Twin pregnancy
  Treatment for infertility → Ovulation induction with hCG/
  Clomiphene/hme

  Most functional cyst → Not more than 5-7 cms regress
  spontaneously

Benign vs malignant tumours

<table>
<thead>
<tr>
<th>Benign</th>
<th>Malignant</th>
</tr>
</thead>
<tbody>
<tr>
<td>* m.C in female of reproductive age</td>
<td>Extremes of age</td>
</tr>
<tr>
<td></td>
<td>Young (adolescent)</td>
</tr>
<tr>
<td></td>
<td>Old</td>
</tr>
<tr>
<td></td>
<td>B/L</td>
</tr>
<tr>
<td>* Usually u/L</td>
<td>Initially: No pain</td>
</tr>
<tr>
<td>* Pain present</td>
<td>Variable consistency</td>
</tr>
<tr>
<td>* Cystic consistency</td>
<td>(Cystic + solid)</td>
</tr>
</tbody>
</table>

IOC: TVS > TAS
High risk features of malignancy on USG:
1. Bilateral
2. Solid mass / multilocular cysts
3. Thick (≥ 3 mm) septa
4. Papillary excrescences on the surface
5. Ascites
6. Presence of matted bowel loops & enlarged nodes

Risk of malignancy index (RMI):
(usg score) x (menopausal status) x (ca 125 levels)

\[ \text{USG score} - \text{check} \]
\[ a - b / l \]
\[ A - \text{ascites} \]
\[ M - \text{malignancy} / \text{metastases} \]
\[ S - \text{solid areas} \]

Score ≥ 200 → High risk of malignancy

Management of adnexal mass

Indications for surgery on adnexal mass:
1. Any ovarian mass showing high risk features on USG
2. Size: Ovarian mass > 7 cms
   Adnexal mass > 10 cms
3. Raised CA 125 in post menopausal females (≥ 35 IU)
4. Acute complication of cyst → Torsion (mC - dermoid cyst)
5. For diagnostic purpose

↑ CA - 125 in reproductive age females:
- Not of diagnostic value
- ↑ in: endometriosis
  Fibroid
  PID

Benign mass

Case 1: In reproductive age
No need for CA-125 levels
Assess size of mass on USG

- 3-5 cms
  - Wait and watch
- 5-7 cms
  - Follow-up with serial USG
- > 7 cms
  - Surgery

If cyst doesn’t resolve or cause symptoms like pain and irregular menses, use OCPs

Case a: In extremes of age

- Post menopausal female (epithelial cell tumours of ovary suspected)
  - CA 125
  - If ≥ 35 IU
  - Surgery
  
  **Note:** CA 125 levels in reproductive age:
  - Not of much use
  - ↑ in benign conditions like endometriosis, Fibroid, PID, Genital TB
  - Significant if ≥ 200 IU

Pre pubertal girls (germ cell tumours suspected)
  - AFP / HCG

Adnexal mass in pregnancy

- Not causing symptoms
- Causing complication like acute abdomen / Torsion

1st trimester
- Wait and watch mostly corpus luteal cyst
- Regress by 12 weeks

2nd trimester
- High risk factors
  - ≥ 10 cm
  - Operate
  - Best time: 14 – 20 weeks
  - Immediate surgery
Note:
- MC ovarian cyst diagnosed in pregnancy: Dermoid cyst
- MC ovarian cyst to undergo torsion in pregnancy: Dermoid cyst
- MC time for ovarian cyst to undergo torsion: end of 1st trimester / puerperium

% of ovarian mass to undergo malignant transformation

↓

Post-menopausal women - 30%
Pre-menopausal women - 7%

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

Classification of ovarian tumours

- Cuboidal epithelium → Surface epithelium
  ↓
  Epithelial cell tumours (90%)
  Germ cell tumours (5-8%)
  Sex cord tumours (3%)
  Metastatic tumours of ovary → Least common
<table>
<thead>
<tr>
<th></th>
<th>Epithelial cell tumour</th>
<th>Germ cell tumour (GCT)</th>
<th>Sex cord tumours</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Incidence</td>
<td>m.c tumour (90%)</td>
<td>5 - 8 %</td>
<td>3%</td>
</tr>
<tr>
<td>2. Types</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serous (75 - 80%) (m.c)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Endometroid (8 - 10%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mucinous (5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clear cell (5%)</td>
<td></td>
<td></td>
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<tr>
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<td>Brenner tumour (least</td>
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<td>common)</td>
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<td>3. m.c benign</td>
<td>Serous cystadenoma</td>
<td>Dermoid cyst (Immature</td>
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<td>variety</td>
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<td>cystic teratoma) [m.c</td>
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<td></td>
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<td>benign tumour of ovary</td>
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<td>over all]</td>
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<td>Thank: Teratoma</td>
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<td>Mature → Benign (Dermoid</td>
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<td>cyst)</td>
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<td>Immature → malignant</td>
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<td>YOLK sac tumour</td>
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<td>AKA Endodermal sinus</td>
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<td>tumour</td>
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<td>C: Choriocarcinoma</td>
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<td>D: Dysgerminoma</td>
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<td>E: Embryonal tumours</td>
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<td># Hormone secreting</td>
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<td>tumours of ovary</td>
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<td>I. Estrogen secreting</td>
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<td>tumours (Feminizing</td>
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<td>tumours)</td>
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<td>Granulosa cell tumours</td>
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<td></td>
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<td>Fibroma</td>
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<td>Thecoma</td>
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<td>II. Androgen secreting</td>
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<td>tumours (virilizing</td>
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<td>tumours)</td>
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<td>Sertoli cell tumours</td>
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<td>Leydig cell tumours</td>
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<td>Hilus cell tumours (Hilus</td>
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<td>cell present in</td>
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<td>medulla of ovary)</td>
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<td>III. Gynandroblastoma</td>
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<td></td>
<td>secretes estrogen + androgen</td>
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<tr>
<td>Epithelial cell tumours</td>
<td>GCT</td>
<td>Sex cord tumours</td>
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<tr>
<td><strong>4. M.C</strong>&lt;br&gt;malignant&lt;br&gt;variety</td>
<td><strong>Serous cystadeno Ca</strong>&lt;br&gt;(m.c malignant ovarian&lt;br&gt;tumour overall)</td>
<td><strong>Mature teratoma</strong>&lt;br&gt;[Earlier → Dysgerminoma]</td>
<td>mostly perimenopausal [can be seen at any age]</td>
</tr>
<tr>
<td><strong>5. Age</strong>&lt;br&gt;Elderly female&lt;br&gt;(m.c → 60-70 years)</td>
<td>mostly B/L</td>
<td>mostly B/L</td>
<td>mostly B/L</td>
</tr>
<tr>
<td><strong>6. U/L/BL</strong>&lt;br&gt;Tumour</td>
<td>% B/L</td>
<td>% B/L</td>
<td>% B/L</td>
</tr>
<tr>
<td>Serous cystadenoma</td>
<td>15 - 20%</td>
<td>Dermoid cyst</td>
<td>10%</td>
</tr>
<tr>
<td>Mucinous cystadenoma</td>
<td>10%</td>
<td>Dysgerminoma</td>
<td>15 - 20%</td>
</tr>
<tr>
<td>Serous cystadeno Ca</td>
<td>≥ 50%</td>
<td>Endodermal sinus tumour</td>
<td>100% u/L</td>
</tr>
<tr>
<td>Endometroid</td>
<td>40%</td>
<td>♦ biopsy of opposite&lt;br&gt;ovary → Contraindicated</td>
<td></td>
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<tr>
<td>♦ Brenner's tumour</td>
<td>mostly u/L</td>
<td></td>
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<tr>
<td>7. Growth of tumours</td>
<td>Epithelial cell tumours</td>
<td>Germ cell tumours</td>
<td>Sex cord tumors</td>
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<tr>
<td>8. Symptoms</td>
<td>slow growing tumors</td>
<td>Rapidly growing (most rapid → yolk sac tumor) present with subacute pain in abdomen • Produce hcg ↓ precocious puberty</td>
<td>Granulosa cell tumours: secrete estrogen → precocious puberty → menometrorrhagia → post menopausal bleeding Androgen secreting tumours → virilisation Fibroma → meig syndrome</td>
</tr>
<tr>
<td></td>
<td>→ Nausea vomiting</td>
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<td></td>
<td>resembling irritable bowel syndrome</td>
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<tr>
<td></td>
<td>mucinous: pseudomyxoma peritonii</td>
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<td></td>
<td>brenner’s: pseudomeig syndrome</td>
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<td></td>
<td>bad prognosis</td>
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<tr>
<td>Sex cord tumours</td>
<td>Germ cell tumours (GCT)</td>
<td>Epithelial cell tumours</td>
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</tr>
<tr>
<td>Granulosa cell tumours</td>
<td><strong>mC malignant GCT</strong>: Immature teratoma, Dysgerminoma, Yolk sac tumour, Teratoma, Brenner, Clear cell tumour</td>
<td><strong>Serous variety</strong>: Benign $\rightarrow$ 55%, Malignant $\rightarrow$ 35 - 40%</td>
<td></td>
</tr>
<tr>
<td>Low grade malignancy $\rightarrow$ 5%</td>
<td><strong>Mucinous</strong>: $\rightarrow$ 10%</td>
<td><strong>Brenner $\rightarrow$ mostly benign</strong></td>
<td></td>
</tr>
<tr>
<td>Most malignant</td>
<td>Yolk sac tumour</td>
<td><strong>Clear cell tumour</strong>: High grade malignant tumour</td>
<td></td>
</tr>
<tr>
<td>5% associated with endometrial hyperplasia</td>
<td>Note: Dermoid cyst malignant in $\sim$ 4% of cases</td>
<td>Low malignant potential $\rightarrow$ Endometrioid $\rightarrow$ Endometrial carcinoma</td>
<td></td>
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<tr>
<td>Low malignant potential $\rightarrow$ Endometrioid $\rightarrow$ Endometrial carcinoma</td>
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<td>Epithelial cell tumours</td>
<td>GCT</td>
<td>Sex cord tumours</td>
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<tr>
<td>1A. Staging</td>
<td>Surgical</td>
<td>Surgical</td>
<td>Surgical</td>
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<tr>
<td>TAH + BSO / Debulking surgery</td>
<td>Conservative surgery</td>
<td></td>
<td>Young</td>
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<tr>
<td>↓</td>
<td>→ U/L salpingo oophorectomy</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>stage I &amp; II</td>
<td>followed by chemotherapy</td>
<td></td>
<td>U/L salpingo</td>
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<td></td>
<td>Stage III &amp; IV</td>
<td></td>
<td>oophorectomy</td>
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<td>followed by chemotherapy</td>
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<tr>
<td>↓</td>
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<td>Followed by chemotherapy</td>
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<tr>
<td>Except: Stage IA &amp; IB</td>
<td></td>
<td>in stage III &amp; IV only</td>
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<tr>
<td>Grade I &amp; II</td>
<td></td>
<td>Chemotherapy of choice:</td>
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<td></td>
<td></td>
<td>B - Bleomycin</td>
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<td>Chemotherapy of choice:</td>
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<td>E - Etoposide</td>
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<tr>
<td>Platinum compound +</td>
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<td>P - Cisplatin / Carboplatin</td>
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<td>Paclitaxel</td>
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<tr>
<td>Note: Ovarian tumours are radio resistant, except</td>
<td>Dysgerminoma</td>
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</table>
CANCER OVARY – 2

Dermoid cyst

m.C benign tumour of ovary: Dermoid cyst
m.C benign epithelial tumour of ovary: Serous cystadenoma
m.C solid benign tumour: Fibroma.
m.C benign tumour of ovary in pregnant female: Dermoid cyst
m.C malignant tumour of ovary in pregnant female: Dysgerminoma.

Dermoid cyst
- B/L in $\rightarrow$ 10%
- Risk of malignancy $\rightarrow<2\%$ (m.C malignancy Squamous cell Ca.)

* Derivatives of all 3 germ layers $\oplus$
  m.C $\rightarrow$ Ectoderm (100% cases)

* Unilocular

* Filled with cheesy / Sebaceous material

* Rokitansky protuberance $\oplus$ $\rightarrow$ Teeth / Hair arise from here

X-ray findings in dermoid cyst:

- Teeth / bone pieces
- Rokitansky protuberance
- Dot and dash appearance
- White ball appearance
## Benign tumours of ovary

<table>
<thead>
<tr>
<th>Serous cystadenoma</th>
<th>Mucinous cystadenoma</th>
<th>Brenner tumour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lining: resemble fallopian tube (ciliated epithelium)</td>
<td>Resembles glandular cervical epithelium</td>
<td>Resemble urethral lining (transitional epithelium)</td>
</tr>
<tr>
<td>Malignant: 40%</td>
<td>≤ 20%</td>
<td>Mostly benign</td>
</tr>
<tr>
<td>B/L: 20%</td>
<td>10%</td>
<td>Mostly u/L</td>
</tr>
<tr>
<td>Unilocular cyst filled with clear fluid</td>
<td>Multilocular cyst filled with mucinous material cut section: Honeycomb appearance</td>
<td>Rubber consistency</td>
</tr>
<tr>
<td><strong>Histology:</strong> Psammoma bodies – concentric fine calcific granulations seen on x-ray</td>
<td>Associated with: Pseudomyxoma peritonei</td>
<td>Histology: Walthard cell rest + coffee bean nucleus Associated with pseudo meig syndrome</td>
</tr>
</tbody>
</table>
Note:
- solid benign tumours of ovary:
  fibroma (mC) > Brenner

Pseudomyxoma peritonei and Meig syndrome

Pseudomyxoma peritonei
- Abundant mucoid material in abdominal cavity and pelvis
- mC cause (Overall): Appendix cancer
- mC ovarian tumour associated → mucinous tumours
  Other causes: mucocele of appendix
- Bad prognosis (recurrence ↑)

Meig syndrome
  Ovarian tumour (Fibroma) + Ascites + Right sided pleural effusion

Pseudomeig syndrome
  Any ovarian tumour other than fibroma
  (Brenner's, granulosa cell tumour, thecoma)
  +
  Ascites
  +
  Right sided pleural effusion

Borderline ovarian tumours

Low malignant potential tumours
Younger age groups (30-50 years)
Stromal invasion absent
Incidence: 10%
Management → Conservative surgery
  *U/L Salpingo oophorectomy*

Indications of conservative surgery in ovarian carcinoma: (U/L Salpingo oophorectomy)
1. Borderline tumours
2. Germ cell tumours
3. Sex cord tumours in a young female
4. Epithelial cell tumours in young female (early stages)
Malignant tumours

Etiology:

↑ estrogen; ↑ risk
↑ ovulation; ↑ risk (Theory of incessant ovulation)

Risk factors:

• Nulliparity
• Obesity
• Early menarche
• Late menopause
• Clomiphene citrate /
  ovulation inducing drugs
• PCOD / infertility
• works in asbestos
  factories
• Dysgenetic gonads
• HRT +/-

Protective factors:

• multiparity
• Pregnancy
• Physical exercise, smoking
  (inhibit aromatase enzyme)
• Anovulation
• OCPs
• Breast feeding
• Tubal ligation
• Hysterectomy (protect
  ovary from exposure to
  carcinogens)

Hereditary ovarian cancers

• 5-10% of all ovarian cancers → Hereditary

BRCA 1 gene: Chromosome 17
BRCA 2 gene: Chromosome 18
HNPCC gene mutation

• Characteristic features:
  Occurs 10 years earlier than usual age of onset
  screening can be done (TVS + CA 125)
  best method to ↓ risk of carcinoma → TAH + BSO at 35 years
  of age but not later than 40 years

Malignant ovarian tumours

malignant epithelial tumours:

Associated with p53 & K RAS mutation
m.c → p53 (aggressive type)
Germ cell tumours

1. Dysgerminoma
   - 2nd M.C. malignant ECT
   - Best prognosis
   - Most radiosensitive
   - M.C. ovarian Ca. in pregnancy
   - Associated with dysgenetic gonads (Y chromosome +)
     - Egg Turner – 45 x0/45 xY
     - Suyer – 46 xY

   Note: M.C. tumour associated with dysgenetic gonads
   ↓
   - Gonadoblastoma (Benign)
   ↓ 40%
   - Dysgerminoma

   Tumour markers: Placental ALP, LDH

Gross appearance: Solid pink to tan lobulated mass

Histology:
- Large, polygonal cells rich in glycogen (clear cytoplasm)
- Prominent nuclei
- Eosinophilic fibrous septa

Yolk sac tumours
- Most malignant ECT
- Worst prognosis
- U/L in 100% cases: Biopsy of opposite ovary contraindicated
On HPE
Schiller Duval body
- Central capillary surrounded by tumour cells present in a cystic space

Tumour markers: AFP
  Anti trypsin

Sex cord tumours

Granulosa cell tumours:
On HPE:

\[\downarrow\]

Typically crowded cells, scanty pale cytoplasm
Nucleus → Groove +
(Coffee bean nuclei)
CalExner bodies → Small eosinophilic fluid spaces with rosette arrangement of cells

Tumour markers: Inhibin

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Tumour marker</th>
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<tbody>
<tr>
<td>Non mucinous epithelial tumour</td>
<td>CA 125</td>
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<tr>
<td>Mucinous epithelial tumor</td>
<td>CA 19-9, CEA</td>
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<tr>
<td>Yolk Sac tumours</td>
<td>AFP, Anti trypsin</td>
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<tr>
<td>Choriocarcinoma</td>
<td>hCG</td>
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<tr>
<td>Dysgerminoma</td>
<td>LDH, Placental ALP</td>
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<tr>
<td>Embryonal tumours</td>
<td>hCG, AFP</td>
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<tr>
<td>Granulosa cell tumours</td>
<td>Inhibin</td>
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</tbody>
</table>
Staging laparotomy

Steps:
1. Incision (midline vertical)
2. All abdominal organs inspected and palpated
3. Samples: a) Ascitic fluid - if present
   - if absent → Add fluid and collect peritoneal washings
   a) Diaphragm
   b) Pelvic & Para aortic lymph nodes
4. Biopsy: multiple peritoneal biopsies
5. TAH + BSO + Infracolic omentectomy [Early stages]
   (not omental biopsy)
   Or
   Debunking surgery + infracolic omentectomy [Advanced stage]

Staging of ovarian carcinoma

FIGO staging (2014)

Stage 1: Tumour limited to ovary
- A → One ovary involved
- B → Both ovaries involved
  • Capsule intact
  • No growth on ovarian surface
  • Peritoneal washing - negative
- C → One / Both ovaries involved
  C₁ : Intra-operative rupture of capsule
  C₂ : Pre-operative rupture of capsule
  C₃ : Malignant ascites / Peritoneal washing - positive

Stage 2: Tumour spreads to pelvic organs / Peritoneum below pelvic brim / 1st peritoneal carcinoma.
- A → Fallopian tubes / uterus involved
- B → Pelvic lymph nodes involved
Stage 3: Retroperitoneal lymph nodes involved (RPL LN) or Peritoneum above pelvic brim involved

A₁ → Retroperitoneal node involvement

\[ A₁(0) \quad A₁(1) \]
10 mm \quad >10 mm

A₂ → microscopic involvement of peritoneum above pelvic brim

θ → macroscopic involvement of peritoneum ≤ 2cms or capsule of liver & spleen ± RPL LN

C → macroscopic involvement of peritoneum > 2cm or capsule of liver & spleen ± RPL LN

Stage 4:

A → Pleural Effusion

θ → Metastasis / Parenchyma of liver & spleen / Inguinal lymph node involvement
ANATOMY OF VULVA

Female external genitalia (vulva)

- Mons pubis
- Prepuce
- Frenulum
- Clitoris (erectile, highly vascular)
- Vestibule
- Labia majora
- Fourchette
- Posterior commissure

Fossa navicularis

Female external genitalia

- Mons pubis
- Prepuce of clitoris
- Domes of clitoris
- Urethral opening (meatus)
- Openings of pararectal (Graefe) ducts
- Vestibule of vagina
- Labium minus
- Vaginal opening
- Labium major
- Hypothenar cuninculus
- Opening of greater vestibular ( Bartholin ) gland
- Vestibular (navicular) fossa
- Frenulum of labium
- Posterior labial commissure
- Perineal raphe
- Anus

Clitoris:
Analogous to penis in males
Erectile & highly vascular
Labia majora:
- Round ligament terminates at anterior 1/3 of labia majora
- Has hair follicles, sweat glands, sebaceous glands
- And modified sweat glands known as (k/a) apocrine glands
- Posterior commissure - Posterior meeting point of labia majora
- Homologous to scrotum in males

Labia minora:
- Encloses clitoris from above - K/a prepuce
- And from below - K/a frenulum
- No hair follicle, apocrine glands, sweat glands
- Has numerous sebaceous glands.

- Outer side → Lined by Keratinized squamous epithelium
- Medial (inner) side → Lined by non-keratinized squamous epithelium

- Outer & medial side separated by Hart’s line

- M.C site of vulval Ca
- M.C type of vulval Ca: Squamous cell Ca.

Fourchette: Posterior meeting point of labia minora.

Vestibule:
- Boundaries:
  - Anterior → Clitoris
  - Lateral → Hart’s line (or labia minora)
  - Posterior → Fourchette

- 6 openings:
  1. Urethra
  2 & 3. Openings of paraurethral / Skene glands (homologous to prostate gland in male)
  4. Opening of vagina K/a introitus

- Covered by a thin membrane → Hymen

- 5 & 6. Openings of Bartholin glands
Fossa navicularis:
Area between introitus and fourchette

Types of hymen

1. Annular hymen
2. Cribiform hymen
3. Septate hymen
4. Imperforate hymen

Blood supply of vulva: Internal pudendal artery
Nerve supply: Pudendal nerve
Lymphatic drainage:
- Superficial inguinal lymph node (LN) (sentinel LN)
  \[\downarrow\]
- Deep inguinal / Femoral nodes
  \[\vdots\text{ In vulva cancer (ca) } \rightarrow \text{inguino femoral lymph node dissection done}\]
- Clitoris \(\rightarrow\) Drains directly into deep inguinal nodes
  \[\downarrow\]
  - Lymph node of cloquet / Rosenmuller lymph nodes

Vulva lymphatics:
* Structures that lie within 2cm of midline
  \(\rightarrow\) Lymphatics cross each other
... Bilateral (e/4) inguinal lymph node dissection done for cancer arising in this area.

Clitoris / Fourchette / Anterior labia minora.

- Lymphatics of structures that lie > 2cm away from midline → do not cross
  - Unilateral lymph node dissection.

- Pelvic lymph node involvement in vulval cancer → stage IV &
  - External iliac
  - Internal iliac
  - Common iliac nodes

- Sentinel lymph node biopsy (SLNB) node biopsy done in vulva cancer > Ca cervix

- Dye used in SLNB in vulval Ca: **Isosulphan blue dye**

**Development of vulva**

- Clitoris: Genital tubercle
- Labia majora: Genital swelling
- Labia minora: Genital folds

**Lining of vulva:**
- Lateral to Hart line: Keratinized squamous epithelium
- Medial to Hart line: Non - Keratinized squamous epithelium
- Hymen and Clitoris: Stratified squamous epithelium

**Bartholin glands**
- Pea - sized, oval shaped glands, a in number
- Racemose variety of glands
- Located in superficial perineal pouch between labia minora and majora at 4'0 clock & 8'0 clock position

- Homologous to Cowper glands/ Bulbourethral gland in males (in deep perineal pouch)

- Ducts of these glands open in vestibule Outside hymen → At junction of anterior 3/6th and posterior 1/6th

- Impalpable; if palpable → Bartholin cyst

- Lining: Gland → Columnar epithelium
Duct → Transitional epithelium  
Opening → Squamous epithelium

Bartholin cyst

- Block of Bartholin duct 
- Between labia majora & minora

Gartner's cyst → Anterolateral wall of vagina.  
m.C variety of vaginal cyst → Inclusion cyst

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

Management of Bartholin cyst:

- Asymptomatic  
- Symptomatic  
  - No treatment  
  - Marsupialisation

Bartholin abscess:
  - m.C organism: E.coli > Gonorrhea.  
  - Management: Incision and drainage

Dissection of perineum (from outside to inside)
VULVAL CANCER

- Comprise of 4% of all gynaecological cancers
- Most common variety of vulval cancer → Squamous cell cancer (90 - 92%)
- 2nd most common variety of vulval cancer → melanoma (2-4%)
- Most common site → Harts line > Labia minora > Labia majora
- Types of squamous cell variety of vulval cancer → warty & keratinizing

<table>
<thead>
<tr>
<th>Warty/Basaloid</th>
<th>Keratinizing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Young females</td>
<td>1. Older females</td>
</tr>
<tr>
<td>2. Unifocal</td>
<td>2. Multifocal</td>
</tr>
<tr>
<td>4. Associated with</td>
<td>4. Associated with</td>
</tr>
<tr>
<td>- Human papilloma virus [HPV] infection</td>
<td>- Lichen sclerosis</td>
</tr>
<tr>
<td>- Smoking</td>
<td>- Squamous cell hyperplasia</td>
</tr>
<tr>
<td>- Vulval intraepithelial neoplasia</td>
<td>- Mutation in p53 gene</td>
</tr>
</tbody>
</table>

- Most common age group for vulval cancer ≥ 65-70 yrs

Vulval cancer – Risk factors & Clinical presentation

1. Risk Factors
   a. HPV infection [most common → HPV 16, others → HPV 18, 31, 33, 45]
   b. Herpes simplex virus infection in smokers [Not a risk factor in non-smokers]
   c. Smoking
   d. Chronic immunosuppression
   e. Lichen sclerosis
   f. Squamous cell hyperplasia
   g. Paget's disease (extra- mammary) → Adenocarcinoma of vulva
   h. Vulval intraepithelial neoplasia → Vulval Cancer

4 years
2. Symptoms of Vulval Cancer
   - Pruritis (most common)
   - Ulcer
   - Mass
   - Bleeding
   - Cachexia

Vulval cancer – diagnosis, spread & prognosis

3. Diagnosis of Vulval Cancer
   - Initial Diagnosis
     - If lesion is not visible
       - Vulvoscopy [3% acetic acid]
         - Biopsy
           - Punch biopsy
           - Wedge biopsy

4. Spread of Vulval Cancer
   - Direct Spread
     - Vaginal
     - Urethra
   - Lymphatic Spread
     - Sentinel lymph node (LN) Spread
       - Superficial inguinal lymph node (SIN)
       - LN not involved
         - Pelvic LN
   - Hematogenous Spread

5. Prognostic factors
   a. Single most important prognostic factor → Involvement of LN
      - Involvement of LN
        - Not involved
          - 5 year survival rate = 85%
        - Involved
          - 5 year survival rate = 50%
b. Depth of the lesion

- <1 mm
  - [Good Prognosis]
  - No LN involved
  - LN dissection - Not required
- ≥1 mm
  - LN are involved

Vulval cancer – Staging

Clinical & Surgical Staging done

- Involvement of Lymph nodes
  - Stage 1 & 2 - LN not involved
  - Stage 3 & 4 - LN involved
  - Stage 3c - LN involvement + extracapsular spread
  - Stage 4A2 - LN is fixed & ulcerated
  - Stage 4B - Pelvic LN involved

- Staging
  - Stage 1 - Cancer is limited to vulva, No LN involved
    - Stage 1
      - Stage 1A
        - Tumor Size: ≤ 2 cm
        - Depth of invasion: ≤ 1 mm
      - Stage 1B
        - > 2 cm
        - ≥ 1 mm
  - Stage 2 - Cancer involving lower part of adjacent structures
    - [Lower 1/3rd of vagina, Lower 1/3rd of anus, Lower 1/3rd of urethra] No Lymph node involvement
  - Stage 3 - Any tumor size, Adjoining structure involvement - +/-
    - Lymph nodes are involved
  - Stage 4 - 4A - Upper part of adjacent structures involved
    - [Upper vagina/anus/rectum/urethra]
    - Lymph nodes are involved
  - 4B - Pelvic LN are involved
**Vulval cancer – Management**

- Stage 1 & 2 [No Lymph Node involved]
  - Radical excision of Vulva
    - [Best – management]
      - Similar to partial / Hemi / Modified radical vulvectomy
  - 

- Incase of Benign / Premalignant lesion
  - Wide local excision

- a. Management of Lymph Nodes
  - Principles: a. LN dissection
    - Inguino femoral LN dissection
      - b. Cancer is ≤ 2 cm from midline
        - Bilateral inguino femoral LN dissection
      - c. Cancer is > 2 cm from midline
        - Unilateral inguino femoral LN dissection

**Approach to lymph node dissection in vulval cancer**

- Lymph Nodes [Inguino femoral]
  - Clinically palpable
    - Do a LN Biopsy
      - 
        - +
          - Debugging of involved LN
            - Treatment – same as clinically not palpable LN
        - –
          - 

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Approach to clinically not palpable LN

↓

- Only stage 1A [Size < 2 cm, Depth of invasion < 1 mm]
  - No Lymph node dissection is done
- Rest all the stages
  - Inguinofemoral LN dissection is done

↓

Unilateral LN dissection

Bilateral LN dissection

- Criteria
  i) Size of tumor < 2 cm
  ii) Laterally present
     [≥ 2 cms away from midline]
  iii) LN should not be palpable clinically

- Done for cases which do not fall under unilateral LN dissection
CONTRACEPTION - NATURAL AND BARRIER METHODS

Classification of contraceptives

- Reversible
  - i) Natural methods
  - ii) Barrier methods
  - iii) Combined oral pills (OCPs)
  - iv) Patches and Rings (estrogen + progestosterone)
  - v) Progesterone only pills (POP)
  - vi) Implants
  - vii) Injections
  - viii) IUCD
    - Cu IUCD, mirena

- Irreversible / permanent methods
  - In females
  - Tubectomy
  - Essure (hysteroscopically)

  - In males
  - Vasectomy (no scalpel vasectomy)

Long acting reversible contraceptive (LARC)

Natural methods - calendar / rhythm method

1st day | 7th day | 8th | 14th | 16th | 19th |

Safe | Safe |

In a 28 day cycle

- Unsafe to have intercourse - 8-19 day
- Safe to have intercourse - 1-7 days & 20-28 days
  (1st week and last week of cycle)
Disadvantages of calendar method

i) Can only be practiced by educated females
ii) Couple has to be motivated
iii) Irregular cycles
iv) During breastfeeding or immediately after delivery

- If the cycle is irregular - Based on Ogino's Knaus theory
  
  1st day of unsafe period - calculated by
  
  Shortest cycle - 18
  
  Last day of unsafe period - calculated by
  
  Longest cycle - 11

Cyclebeads or Tirumala method

- A/V/A Standard day method

- Helps female to have a track of her cycle.
- In Tirumala - A movable rubber band is attached
  This rubber band is moved from one bead to another

- Red bead - 1st day of cycle
- Brown beads - Depicts safe period
- White beads - Depicts unsafe period
- Dark brown bead - On day 26
  - Short cycles - Menstruation begins before day 26
  - Cannot rely on Tirumala method

- Tirumala method is used only if the cycle is between 26 - 32 days

- Failure rates - 2/100 women year

**Cervical mucus method or Billings method**

- At the time of ovulation - Cervical mucus is thick
- Cervical mucus is thin and can be stretched in between fingers
  - Safe period
- Cervical mucous thick; Scanty
  - Unsafe period
- Check **basal body temperature** daily (in the morning)
  ↓
  At the time of ovulation - basal body temperature ↑
  ↓
  unsafe period

- **Symptothermic method** - Cervical mucus method
  +

  Basal body temperature

**Barrier methods - male and female condoms**

<table>
<thead>
<tr>
<th>Male condom</th>
<th>Female condom</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Contraceptive</em></td>
<td><em>Protection against</em></td>
</tr>
<tr>
<td>+ protection against sexually transmitted diseases (STD)* Protection against pelvic inflammatory disease (PID)</td>
<td><em>STD not as effective as male condom</em></td>
</tr>
<tr>
<td><em>Protection against ectopic pregnancy</em></td>
<td><em>Protection against pelvic inflammatory disease</em></td>
</tr>
<tr>
<td><em>Protection against HIV</em></td>
<td><em>Protection against ectopic pregnancy</em></td>
</tr>
<tr>
<td><em>Protection against HPV infection</em></td>
<td><em>Protection against HIV</em></td>
</tr>
<tr>
<td><em>Protection against cancer cervix and cervical dysplasias</em></td>
<td><em>not as effective as male condom</em></td>
</tr>
<tr>
<td></td>
<td><em>Protection against HPV</em></td>
</tr>
<tr>
<td></td>
<td><em>Protection against cancer cervix, cervical dysplasias</em></td>
</tr>
</tbody>
</table>
Disadvantages of male condom
1) Contact dermatitis in female partner
2) used only once
3) Sexual pleasure is reduced
4) Failure rates - 2 -15 /100y

Disadvantages of female condom
1) UTI in female partner
2) used only once
3) Sexual pleasure is reduced
4) Failure rates - 5 - 20/100y
5) Cannot be used in females with cystocele, rectocele, retroverted uterus

---

Female and male condoms contraindications and uses

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>STD/PID</th>
<th>Ectopic Pregnancy</th>
<th>Emergency Contraceptive</th>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male condom</td>
<td>↓</td>
<td>↓</td>
<td>X</td>
<td>uterine prolapse / Retroverted uterus</td>
</tr>
<tr>
<td>Female condom</td>
<td>↓</td>
<td>↓</td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

In a HIV patient best contraception
1st - IUCD + Barrier method
2nd - IUCD
3rd - Barrier method

Diaphragm and sponges

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<table>
<thead>
<tr>
<th>Vaginal Diaphragm</th>
<th>Today Sponge</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Not a sperm proof mechanical barrier ↓ Need to add spermicide</td>
<td>* Mushroom shaped disposable sponge - has 1gm of Nonoxynol - 9 (spermicide)</td>
</tr>
<tr>
<td>* Placed in vagina few minutes to 2 hours before intercourse (max - 6 hours)</td>
<td>* Concave side - face cervix</td>
</tr>
<tr>
<td>* Should be removed 6 - 8 hours after intercourse</td>
<td>* Can be inserted 24 hours before intercourse</td>
</tr>
<tr>
<td>* Max time to keep inside body 24 hours</td>
<td>* Should be removed max 6 hours after intercourse</td>
</tr>
<tr>
<td></td>
<td>* Max time to keep sponge inside body - 30 hours</td>
</tr>
<tr>
<td></td>
<td>* Gives continuous protection for 24 hours irrespective of number of times of intercourse</td>
</tr>
</tbody>
</table>

Diaphragm and sponge - contraindications and toxic shock syndrome

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>STD / PID</th>
<th>Ectopic pregnancy</th>
<th>Emergency Contracept</th>
<th>Contraindication</th>
<th>Special uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragm and sponge</td>
<td>↓</td>
<td>↓</td>
<td>×</td>
<td>Diaphragm - prolapsed / Retroverted uterus</td>
<td>* No protection against HIV * No special uses</td>
</tr>
</tbody>
</table>

Toxic shock syndrome

↑ Chances with ↓
Female condom diaphragm
↓ Chances with ↓
Today sponge
Complications of diaphragm and sponge

Diaphragm
• Toxic shock syndrome (TSS)
• Cervical erosion
• UTI

Sponge
• Does not cause TSS
• Allergic reactions
• Vaginal dryness

Spermicide

Disrupts the sperm cell membrane
• Nonoxynol-9
• Oxytocin
• Menfogel

If used alone
• Very high failure rates
So used in combination with barrier contraceptives

Pearl index
• Measures efficacy of contraception
• Best method to measure efficacy - Life table analysis

Formula

\[
\text{Pearl index} = \frac{\text{No. of accidental pregnancies}}{\text{No. of females that used contraceptive} \times \text{Years of use}} \times 100
\]

\[
\text{Calculation of pearl index}
\]

Eg - If a contraceptive ‘x’, used by 150 females, used for 3 years out of 100 - 24 females conceived

By formula A: \[
A = \frac{24 \times 100}{100 \times 3 \text{ yrs}} = \text{Pearl index} = 8
\]

By formula B: \[
B = \frac{24 \times 1200}{100 \times 36 \text{ months}} = \text{Pearl index} = 8
\]
Pearl index for user independent and dependent methods

For user independent method
1) Implants - 0.05%
2) Sterilization - male - 0.1%
   Female - 0.5%
3) IUCD - Mirena - 0.2%
   Cu T380A - 0.4%

For user dependent methods

<table>
<thead>
<tr>
<th>Method</th>
<th>Perfect use</th>
<th>Typical use</th>
</tr>
</thead>
<tbody>
<tr>
<td>OCP's</td>
<td>0.3%</td>
<td>8%</td>
</tr>
<tr>
<td>Ring</td>
<td>0.3%</td>
<td>8%</td>
</tr>
<tr>
<td>Patch</td>
<td>0.3%</td>
<td>8%</td>
</tr>
<tr>
<td>DMPA (Depot medroxy progesterone Acetate)</td>
<td>0.3%</td>
<td>7%</td>
</tr>
<tr>
<td>Sponge</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td>Male condom</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>Female condom</td>
<td>5</td>
<td>20</td>
</tr>
</tbody>
</table>
ESTROGEN AND PROGESTERONE CONTAINING CONTRACEPTIVES

OCP

- Pills with estrogen + Progesterone
  ↓
  Negative feedback on LH
  ↓
  LH surge absent
  ↓
  No ovulation

- Main mechanism of action of OCP: Anovulation

- Main mechanism of IUCD:
  1. Inhibits fertilization
  2nd best: Inhibits implantation
  Never acts by anovulation

- Other mechanism:
  Synthetic Estrogen
  ↓
  Negative feedback on FSH
  ↓
  Inhibit Folliculogenesis

- Progesterone: makes cervical mucus thick

- Classification:
  ↓
  Dose wise
  Progesterone component :
  High dose : > 50 mcg
  1st generation : Norethinone
  Low dose < 50 mcg
  and generation : Levonorgestrel
  Average : 30 - 35 mcg
  3rd generation : Desogestrel
  4th generation : spironolactone
  minimum effective dose
  Gestodene
  of estrogen : 10 mcg
  Norgestimate
  Drospirenone
  Cypotrocone
  acetate
  FDA : Lo Loestrin Fe
4th generation pill progesterone: Drospirenone

Progesterone: Cyprome acetate

Mala – N and Mala – D has same composition
→ Estrogen: Ethinyl Estradiol → 30 mcg
→ Progesterone: Levonorgestrel → 0.15 mg
→ 21 white tablets + 7 brown tablets → Ferrous Fumarate

• Mala-N: Distributed freely by Government of India
  Mala-D: Not available free of cost

OCP usage

00:12:04

• Ideally: start from Day 1 → No backup method
• Can also start: < Day 5 → needed (Day 1–Day 4)
• Day 5 or any day after Day 5: Backup method of contraception X 7 days → AKA Quickstart
* Should not be used
  - Breast feeding female
  - After delivery < 6 weeks
  - In patients who are on drugs which are Enzyme inducers
  - Accelerate metabolism of OCP
  - Efficacy of OCP ↓

  E.g:
  - Phenytoin
  - Phenobarbitone
  - Valproic Acid
  - Rifampicin

* On missing the pill:

  One pill  \[ \rightarrow \]  \[ \rightarrow \]  \[ \rightarrow \]  \[ \rightarrow \]  ≥ 2 pills

  Next day: Can take
  a pill (no backup needed)

  On remembering: Takes one pill
  + 1 pill of the day
  + Backup method
  of contraception
  × 7 days

* Clinical case: Female misses her pill at the end of pack, she doesn't have any bleeding
  management: Next step → Pregnancy Test

* MC side effect: Break through bleeding

**Other uses and disadvantages of OCP pills**

* Non-contraceptive uses of OCP:
  1. Regularise cycles
     DOC: for irregular cycles
  2. Amenorrhea: Management of dysmenorrhea
     management of PMS
3. Pregnancy ↓ LH → ↓ Androgens
   DOC: for Hirsutism
   management of Acne

4. ↓ FSH → ↓ Natural Estrogen

   Hyper estrogenic conditions like:
   → Fibroid
   → Endometriosis
   ↓ Endometrial cancer
   ↓ Ovarian cancer
   ↓ Ovarian cyst
   ↓ Colon cancer
   ↓ Benign Breast diseases

Disadvantages:
- ↑ Depression
- ↑ Cancer cervix – Adenocarcinoma
  - ≥ 5 years
  - Reversible
- ↑ Gall stones
- ↑ Cholelithiasis

* No effect on:
  1. Breast cancer
  2. Hepatic Adenoma
  3. Hepatocellular carcinoma
  4. Gall Bladder cancer

* Clinical case: Female who is on OCP has spotting (on pills)
  Spotting
  ↓
  Doesn’t indicate failure
  ↓
  If spotting doesn’t disappear spontaneously in 1-3 cycles

   Spotting: 1st Half
   ↓
   Estrogen ↑

   Spotting: 2nd Half
   ↓
   Progesterone ↑
- On use of OCP:
  1. Return of fertility: within 3 months
  2. Quickstart: not teratogenic

### OCPs: Contraindications and special uses

<table>
<thead>
<tr>
<th>Contraceptive</th>
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<th>Ectopic Pregnancy</th>
<th>Emergency contraception</th>
<th>Contraindications</th>
<th>Special uses</th>
</tr>
</thead>
</table>
| OCPS          | ↓ Chlamydia and candida | ↓ (least chances of ectopic pregnancy) | Yuzpe method | mnemonics: Banks, Have various, Schemes To provide Home Loans During May; 
B → Breast cancer  
H → Hypertension (moderate-severe ≥ 160/110)  
V → Undiagnosed vaginal bleeding  
S → Smoker ≥ 35 years  
P → Pregnancy  
H → High cholesterol  
H → High triglycerides  
L → Liver disease  
D → Diabetes with vasculopathy  
m → Migraine with aura | Contraceptives of choice:  
1. After H mole evacuation  
2. Young married couple living together  
3. Spacing of Pregnanies  
DOC:  
1. Hirsutism  
2. Irregular cycles  
3. Dysmenorrhea management  
→ Ovarian cyst  
→ Fibroid  
→ Endometriosis |
Other estrogen and progesterone containing contraceptives

- Vaginal Ring
  - Estrogen + progesterone
  - AKA NUVA Ring

- Transdermal patch
  - Estrogen + progesterone
  - AKA ortho evra patch

- Ring made of: Ethinyl Acetate
  - Outer diameter: 54 mm
  - Cross section: 4 mm
- Estrogen: Ethinyl Estradiol
  - Progesterone: Etonogestrel
- Release Rate:
  - Estrogen → 15 mcg/day
  - Progesterone → 120 mcg/day
- Applied in vaginal x 3 weeks
  - 1 week off
    (should be inserted within 3 hours of intercourse)

- 20 cm skin patch
- Estrogen: Ethinyl Estradiol (75 mcg)
  - Progesterone: Norelgestromin (6 mg)
- Release rate:
  - Estrogen: 20 mcg/day
  - Progesterone: 150 mcg/day
- Each patch → 1 week
  - 3 patches in a pack
    - 3 weeks → On
    - 1 week → Off
1. ↑ venous thrombosis and embolism
2. ↑ failure rate in obese female
PROGESTERONE ONLY CONTRACEPTIVES

Types of progesterone only contraceptives:

1) Progesterone only pills (POP)
2) Progesterone Injections
3) Progesterone Implants

Classification of progesterone:

- **Older generation**
  - Includes 1st and 2nd generation progesterone
  - Low concentration
    - MOA: Thickens cervical mucus
    - Long term exposure of progesterone causes:
      - Endometrial atrophy
      - Amenorrhea

- **Newer generation**
  - Includes 3rd and 4th generation progesterone
  - High concentration
    - MOA: Inhibits ovulation

Advantages:

1) Used in lactating females
2) No effect on carbohydrate metabolism
   - Except: Progesterone only pills
3) No effect on lipid metabolism
   - Except: Depot medroxy progesterone acetate (DMPA)
     - ↑ LDL and ↓ HDL
4) No effect on clotting factors
   - In patients with history of thrombosis

   **Management**
   **Contraindications**
   POP
   - Implants
   - Injections

5) **Reduce** pelvic inflammatory disease (cervical mucus - thick)

**Side effects and contraindications**

Side effects:
1) Irregular bleeding (na)
2) Increase of failures
   ↓
   increased chance of ectopic pregnancy.
   (due to reduced peristalsis of tube)
3) Not an emergency contraceptive method.

Contraindications:
1) Undiagnosed vaginal bleeding.
2) Liver disease.
3) Previous history of thrombosis, ectopic pregnancy
   ↓
   Not a contraindication for Progesterone only pills (POP)
4) Pregnancy
5) Breast cancer
**Progesterone only pills / minipills**

- **Older generation**
  - Mechanism of action
  - Thickens cervical mucus
    - Taken at same time (daily)
    - Grace period / Delay: 3hrs acceptable
    - If patient forgets
  - Alternate method of contraception done for 48hrs

- **New generation**
  - i) Desogestrel
  - a) Cerazzette (India)
  - Mechanism of action
  - Anovulation
    - Taken at same time (daily)
    - Delay of 12hrs acceptable

**Advantages of progesterone only pills:**

1) Contraceptive method of choice
2) Can be used in lactating females

POP > Intrauterine contraceptive device.

- In lactating female POP started at:
  - CDC / ACOG recommendation
  - WHO recommendation
  - Immediately after delivery
  - After 6 weeks
  - Currently followed
Progesterone injections

**Depo Provera Injection**
- Depot of medroxy progesterone acetate (DMPA)
- Dose: 100 mg
- MOA: Inhibit ovulation
- Repeat after 3 months
- Delay of 4 weeks acceptable
- Both DMPA and NET En injections

**NET En Injection**
- Norethindrone
- Dose: 150 mg
- MOA: Inhibit ovulation
- Repeat after 2 months
- Delay of 4 weeks acceptable

- Given within first 7 days of cycle
- Are effective within 24 hrs

**Depot of medroxy progesterone acetate (DMPA)**
- **Advantage**
  ① Can be given to lactating female
  ② Reduce seizure frequency
  ③ Decrease sickling in sickle cell anemia
  Contraceptive drug of choice

- **Disadvantage**
  ① Decrease bone mineral density
  ② Delayed return of fertility
  - Mostly by 12 months
  - Maximum: 18 months
Implants

Norplant → Contains 6 rods
    → Not used

Implanon → Single rod
    → Length: 4cm

Nexplanon (recent)
    → Radio-opaque

Etonorgestrel
- Dose: 68mg
- Released at a rate of 67 mcg/day
- MDA: Inhibits ovulation
- Inserted within 5 days of menstrual period
- Inserted by piercing
  6 - 8 cms above medial epicondyle in the non-dominant hand
- OPD procedure, done under LA
- Inserted immediately after delivery
  ↓
  Effective immediately
- Does not decrease bone mineral density
<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>STD / PID</th>
<th>Ectopic pregnancy</th>
<th>Emergency contraception</th>
<th>Contraindications</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progesterone only pills</td>
<td>Decrease</td>
<td>Increase</td>
<td>Not used</td>
<td>1) Undiagnosed vaginal bleeding 2) Liver disease 3) Pregnancy 4) Breast cancer</td>
<td>1) DOC for lactating female 2) In patients with history of thrombosis 3) Used in a smoker ≥ 35 years 4) Patients with migraine (aura) ↓ In cases where estrogen is contraindicated, progesterone can be used</td>
</tr>
<tr>
<td>Depot of medroxy progesterone acetate (omPA)</td>
<td>Decrease</td>
<td>Increase</td>
<td>Not used</td>
<td>1) Undiagnosed vaginal bleeding 2) Liver disease 3) Patients with history of thrombosis 4) Pregnancy 5) Breast cancer</td>
<td>1) Used in lactating female 2) Epilepsy 3) Sickle cell anemia 4) Decrease bone mineral density</td>
</tr>
<tr>
<td>Implanon</td>
<td>Decrease</td>
<td>Increase</td>
<td>Not used</td>
<td>1) Vaginal bleeding 2) Liver disease 3) Patients history of thrombosis 4) Pregnancy 5) Breast cancer</td>
<td>1) Used in lactating female 2) Does not decrease bone mineral density</td>
</tr>
</tbody>
</table>
INTRA UTERINE CONTRACEPTIVE DEVICE (IUCD)

Introduction

IUCD

1st generation

2nd generation

3rd generation

Non medicated

Cu IUCD (copper)

Mirena (progesterone containing IUCD)

Lippes loop

No longer used

Copper IUCD and mirena IUCD – basic structure

Cu T 380 A

Mirena

<table>
<thead>
<tr>
<th>Cu T 380 A</th>
<th>Mirena</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-shaped polyethylene frame</td>
<td>Plastic IUCD with no copper wire</td>
</tr>
<tr>
<td>Surface area of copper wire = 380 mm²</td>
<td>LNG (levonorgestrel) – Progesterone</td>
</tr>
<tr>
<td>A - Copper present on horizontal arms</td>
<td>Coated with barium sulphate – So Radiopaque</td>
</tr>
<tr>
<td>The entire frame work is coated with Barium sulphate – Radiopaque</td>
<td>Delivery system for LNG on vertical limb</td>
</tr>
<tr>
<td>• 3mm ball is present at the end – Prevents cervical perforation</td>
<td>• monofilament thread is present</td>
</tr>
</tbody>
</table>
CuT and mirena IUCD – mechanism of action

<table>
<thead>
<tr>
<th>CuT 380 A</th>
<th>Mirena</th>
</tr>
</thead>
<tbody>
<tr>
<td>* A/K/A Paragard</td>
<td>* A/K/A LNG – 30</td>
</tr>
<tr>
<td>* Distributed free of cost by the government of India</td>
<td>* The total LNG present – 50mg</td>
</tr>
<tr>
<td>* It releases copper (Cu) at 50mcg/day</td>
<td>* It releases – 20mcg/day</td>
</tr>
<tr>
<td>* Approved life span – 10yrs</td>
<td>* Approved life span – 5yrs</td>
</tr>
<tr>
<td>* It can release Cu upto – 10yrs</td>
<td>* It can release LNG up to – 7yrs</td>
</tr>
<tr>
<td>* Expulsion rate : 8 – 10%</td>
<td>* Expulsion rate – 5–6%</td>
</tr>
<tr>
<td>* Mechanism of action</td>
<td>* Mechanism of action</td>
</tr>
<tr>
<td>↓</td>
<td>Causes endometrial atrophy</td>
</tr>
<tr>
<td>i) Prevents fertilization – makes the endometrium spermicidal</td>
<td>Inhibits implantation Does not inhibit ovulation</td>
</tr>
<tr>
<td>ii) Prevent implantation</td>
<td></td>
</tr>
<tr>
<td>iii) Does not inhibit ovulation</td>
<td></td>
</tr>
</tbody>
</table>

CuT and mirena iucd – side effects

<table>
<thead>
<tr>
<th>CuT 380 A</th>
<th>Mirena</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Do not ↓ the risk of pelvic inflammatory disease (PID)</td>
<td>* The progesterone in mirena – makes cervical mucous thick ↓ risk of PID</td>
</tr>
</tbody>
</table>
• Risk of PID is ↑ in 1st month after insertion of CuT
• m.C - PID - Actinomyces
• ↑ ectopic pregnancy if failure occurs
• Side effects -
  i) ↑ m.C - ↑ blood loss
  ii) ↑ m.C - pain in abdomen
  ↓
  m.C.C of removal of IUCD
• Other complications - perforation at the time of insertion

• ↑ ectopic pregnancy if failure occurs (more in comparison to Cu IUCD) because progesterons is a smooth muscle relaxant and decreases peristalsis
• Side effects -
  i) ↑ m.C - irregular bleeding
  ↓
  Endometrial atrophy
  ↓
  Amenorrhea

CuT and mirena IUCD – uses

<table>
<thead>
<tr>
<th>CuT 380A</th>
<th>mirena</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Can be used as an emergency contraceptive ↓</td>
<td>• Cannot be used as an emergency contraceptive</td>
</tr>
<tr>
<td>• Most effective</td>
<td></td>
</tr>
<tr>
<td>• Can be used up to 5 days after intercourse</td>
<td></td>
</tr>
</tbody>
</table>

Timing of insertion of IUCD
• Post placental insertion of IUCD ↓
  If IUCD is inserted within 10 mins of delivery of placenta.
• Postpartum IUCD insertion ↓
  Inserting after 10 mins but before 48 hrs of delivery
- If IUCD is not inserted within 48hrs of delivery
  ↓
  Then inserted after 6 weeks of delivery
  ↓
  Known as Interval IUCD

- In a female, who has not delivered recently
  ↓
  IUCD inserted within 1st 10 days of her cycle

**IUCD with missing thread**

- In a female with IUCD - if missing threads
  ↓

  Causes - Thread has coiled
  IUCD has expelled
  Pregnancy
  Perforation of uterus

- If missing threads
  ↓

  Transvaginal ultrasound (TVS)

  ↓

  Thread is coiled
  Can continue IUCD
  Remove IUCD

  under Hysteroscopy guidance
  Using shirodkar hook or Artery forceps

  Perforation (at the time of insertion)

  ↓

  • If IUCD is in peritoneal cavity completely
    ↓
    • Immediate Laparotomy / Laparoscopy

  • If IUCD is partly in uterus and partly in peritoneal cavity
    ↓
    • Hysteroscopy + Laparoscopy
Hooks for IUCD removal - Shirodkar hook

Pregnancy with IUCD

- Continue with pregnancy
  - If thread is visible
    - Terminate pregnancy only if patient is not willing
  - If thread is not visible
    - Remove IUCD (Preferred method)
    - Continue with pregnancy

Risk to pregnancy with IUCD

- ↑risk of Abortion
  - Preterm Labor
  - Premature Rupture of membranes
- ↑risk of infection
  - IUGR

Contraindications of IUCD – WHO category IV

Absolute contraindications
1) Active PID or PID in past 3 months
2) Puerperal sepsis
3) Distorted uterine cavity - due to
   - Mullerian malformation
   - Fibroids
   - Cancer endometrium
iv) Pregnancy  
v) Undiagnosed vaginal bleeding  \rightarrow Absolute contraindications for any contraceptives  
vi) For Cu. T - Wilson disease  
vii) For Mirena - breast cancer  
viii) Previous history of ectopic pregnancy is not a contraindication

Ideal candidate to use IUCD
* In a female who has at least 1 child
* Female who has monogamous relationship
* Female with no history of PID

Other types of IUCD

In Nulliparous
↓
Frameless IUCD
↓
Cause less Pain
↓

GyneFix 330
↓
Cu cylinders which are threaded on a polypropylene suture
↓
Attached to myometrium

Fibroplant
↓
LNG containing IUCD
↓
* releases 14 mcg/day  
  or  
  20 mcg/day

multi load 375
↓
Flexible arms + spurs
↓
↓
expulsion rate
* it is preloaded in an inserter

multload 375

GyneFix 330
### CuT and mirena IUCD special uses

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>PID/STD</th>
<th>Ec-topic pregnancy</th>
<th>Emergency contraceptive</th>
<th>Contraception (CI)</th>
<th>Special uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cu IUCD</td>
<td>↑</td>
<td>↑</td>
<td>within 5 days</td>
<td>Wilson disease</td>
<td>Contraceptive of choice in:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+ WHO classification category IV</td>
<td>i) Heart disease patients (stable)</td>
</tr>
<tr>
<td>mirena</td>
<td>↓</td>
<td>↑</td>
<td>x</td>
<td>Breast cancer + Same as Cu IUCD</td>
<td>ii) Diabetic patients ii) HIV patient</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>In heavy bleeding</td>
</tr>
</tbody>
</table>

- *In decompensated heart disease patients*
  i) Permanent method of choice vasectomy
  Tubectomy
  
  ii) Temporary method
  Barrier method

### Non steroidal contraceptive – centchroman

- Active ingredient
  - Ormiloxifene
    - SERM (Selective estrogen receptor modulator)
- Mechanism of action
  - Makes endometrium out of phase
  - Implantation won't occur
- Developed in 1991, CDRI Lucknow
- Originally Trade Name ‘Saheli’

- By the Name of CHMRIA – Distributed free of cost by government of India
Side effect
  Delayed menstruation

Dose
  • Initial 3 months – 30mg / twice weekly
  • Later – 30mg/weekly

Other properties
  i) Not teratogenic
  ii) Can be used as emergency contraceptive
  iii) Does not protect against PID.
  iv) Return of fertility – within 4 months of stopping the drug

Centchroman
PERMANENT CONTRACEPTION

Tubectomy 00:05:24

- **mUC** method of contraception worldwide
- Permanent method
- 1st performed by Dr. J. Blundell

Procedure

- A loop is made out of fallopian tube
  - followed by ligation & cut the tube
- **Ligate tube** - 2-3 cms away from the angle of tube
- **mUC reason for failure** - misidentification of structure to be ligated:
  - Round ligament ligated instead of fallopian tube
- **Avoidance of misidentification** - Recognise the tube by Aimbriae
- **For Recanalisation** - methods, of choice is isthmo isthmic anastomosis
- while cutting the tube - At least 1 cm of tube has to be cut
- Recanalisation is best with methods where least possible tube is damaged

![Diagram of tubal ligation](image)

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Methods of tubectomy

Laparoscopic tubectomy
(6 cm incision is made)
- Fallope rings
- clips
- m.C in India
- Flischie
- Hulka

Minilaparotomy
- A small incision (2-3 cm)
is made 2-3 cm above
pubic symphysis
- In post partum sterilization
incision is made below
umbilicus

Timing for tubectomy

Recently delivered
- After 48hrs and
upto 1 week after
delivery
- Post partum
sterilization

Not delivered
- If not done
within 1 week
- To be performed
after 6 weeks
- Interval
sterilization
- Performed within
1st 7 days of
menstrual cycle
- Interval
sterilization

Post partum sterilisation and interval sterilisation

Post partum sterilization
- Can only be done
by minilaparotomy
- Laparoscopy is
contraindicated
because ↑ risk of perforation
of uterus (uterus lies at the
level of umbilicus)

Interval sterilization
- Method of choice
is Laparoscopy
- Uterus after 6 weeks
returns to its normal position

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- MC method used for sterilization - Laparoscopy
- MC method used for postpartum sterilization - mini laparotomy

Laparoscopic sterilisation

Gas ↓ Pressure ↓ Maximum volume of ↓ Instrument ↓
CO₂ ↓ 8-12 mmHg ↓ gas ↓ used to create pneumoperitoneum ↓
Or ↓ Never ≥ 15 mmHg ↓ 2 L ↓ veress needle
(N₂O)

Two methods

Falope ring ↓ Clips ↓
- MC in India
- To be placed at junction of proximal 1/3rd and middle 1/3rd of Fallopian tube
- Damage to Fallopian tube - 3 cm
- Preferred in young females (may require recanalization later on)
- Preferred if tubectomy is done after abortion (tubes are edematous)
- Damage to tube: 4-5 mm

Hence, best suited for recanalization

Laposcopic tubal ligation using Falope rings
Laparoscopic ligation

Fishie clip - made of outer titanium and inner sialastic material

Minilaparotomy – techniques

Pomeroy’s method

1. A loop of tube is made at isthmus part
   a) Single ligature is done – using catgut (absorbable suture)
   b) The loop is cut – using metzenbaum scissors

- The tube is held with - Babcock’s forceps
- Failure rate - 0.4%

Pomeroy Method:
- Babcock’s forceps
- Catgut suture
- Difficult in tubal adhesion

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Minilaparotomy – modified pomeroy’s method

1) A loop is made at isthmus part of tube

2) A window is created in mesosalpinx followed by two ligatures placed at proximal and distal ends of the loop

- Double ligature
- Loop at – Sent for HPE (Histopathological examination)
- Failure – 0.2%
- mC used method for tubectomy
- A/H/A Parkland’s method

Madlener’s technique and uchida technique

Madlener’s technique:

1) A loop is created
2) Single ligature placed
3) The loop is crushed

- It is outdated – due to high failure rate
Uchida technique

- **Success rate is high**
- **Step A** - A solution of normal saline and epinephrine is injected into the serosa of Fallopian tube, as a result - the serosa swells

  \[\downarrow\]

  Incision - given on serosa over the anti-mesentric border

- **Step B** - Muscular part of the tube isolated using Babcock forceps
- **Step C** - Fallopian tube is cut
- **Step D** -
  - The proximal end is tied and left inside the serosa.
  - The distal end is stitched with serosa in a purse string manner.
Irving method, Kroener’s fimbriectomy, Aldridge’s fimbriectomy

**Irving method**

Step A - Tube cut at isthmo-ampullary junction

Step B - A tunnel is made in the myometrium into which the proximal part of the Fallopian tube is buried. Distal part of the Fallopian tube is attached to the broad ligament of uterus (Sutured along)

**Kroener’s fimbriectomy**

- Double ligature at fimbrial end of Fallopian tube followed by cutting of fimbrial end
- No longer used

**Aldridge’s fimbriectomy**

- Modification of Kroener’s
- Double ligature at fimbrial end of Fallopian tube
- Fimbriae placed posterior to peritoneum & sutured with broad ligament
- No longer used
- Least failure rate - Cautery > Uchida > Irving > Modified Pomeroy's > Pomeroy's
- Chances of recanalisation - Clips > Falope rings > Pomeroy's > modified Pomeroy's

- Cautery - Least failure rates
  - No longer used - because damages adjacent tissues ↑ risk of ectopic pregnancy

**Essure device**

- Spring like device
- Inserted into fallopian tube - intramural part
  - Via hysteroscope

- It has - Outer cylinder, inner cylinder
  - Nickel and titanium stainless steel

- Mechanism of Action
  - Irritates fallopian tube
  - Initiates tissue reaction
  - Inflammation → Blocks tube

- It takes 3 months for essure to act - so backup method is needed

- After 3 months - perform **Hysterosalpingography**
  - To confirm L/R blockade of tube

- Success rate - 99%
Male sterilisation – vasectomy

Non-scalpel vasectomy

- It is done using – Two instruments

  ▼

  Ring clamp

  ▼

  Dissecting clamp

Procedure

- Done under local anesthesia.
- Step-1 → Identify and immobilize the vas deferens using ring clamp
  ▼
  With help of dissecting clamp – puncture the skin
  ▼
  Through that puncture – vas deferens taken out
  ▼
  Hold the vas deferens with ring clamp
  ▼
  With the help of dissecting clamp – cut the vas deferens
  ▼
  Both the ends are ligated and put back in place

Non-scalpel vasectomy steps
Non-scalpel vasectomy steps

- Failure rate - 0.1%
- Reversal of vasectomy - microsurgery (vasovasostomy)
  - Fertility - 90%
  - Pregnancy - 70%

- Male does not become sterile immediately after vasectomy
  - So, additional contraceptive used for 15-30 ejaculations or 3 months
  - Perform - semen analysis
  - If azoospermia - is confirmed
  - No additional contraceptive is required

Emergency contraception

- After unprotected intercourse
  - If taking contraceptives
    - Known as emergency contraception / interceptives

- Mechanism of Action
  - Hormonal
    - Inhibit ovulation
    - Inhibit fertilization
    - Inhibit implantation
  - Non hormonal
    - Spermicidal
- Emergency contraceptives (EC) are not abortifacient
- Never interrupt with early pregnancy
- Never interrupt with placental functioning
- Best time to take EC - within 72 hours of unprotected intercourse
- They can be given till 120 hours / 5th day of unprotected intercourse
- Most effective EC - IUCD (Copper-T)
- Most effective hormonal contraceptive - Ulipristal

Emergency contraceptives – drugs

1) Ulipristal
   - It is SPRM – selective progesterone receptor modulator
   - Single dose of 30 mg
   - Trade name – ‘Ella’

2) Levonorgestrel (LNG)
   - Total dose – 1.5 mg stat or 0.75 mg – after 12 hours → 0.75 mg
   - M/C used EC – LNG
   - EC distributed free of cost by government of India – LNG (E pill)

Other drugs used as EC
1) Centchroman
   - 2 tablets of centchroman (60 mg) → after 12hrs → 2 tablets
   - Failure rate 1%

2) Oral contraceptive pills
   - Yuzpe Method
   - Total dose – 100mcg ethynylestradiol (EE) + progesterone
   - After 12 hours
   - Repeat 100 mcg EE + progesterone
   - For high dose pill – 2 tablets → 2 tablets
   - For low dose pill – 4 tablets → 4 tablets
   - For very low dose pill – 5 tablets → 5 tablets
3) mifepristone
   • 10 mg - Single dose

Drugs not used as EC
   1) misoprost
   2) Progesterone only pill
   3) mirena
BASICS OF PREGNANCY

Duration of pregnancy

- Duration: 9 months + 7 days
  - Or 40 weeks
  - Or 280 days
  - From 1st day of last menstrual period (LMP)

- If delivery < 37 weeks = Preterm labour

- If delivery ≥ 42 weeks = Post term labour (294 days)

- Term delivery: 37 weeks to 41 weeks + 6 days

  - Early term: 37w - 38w + 6d
  - Term: 39w - 40w + 6d
  - Late term: 41w - 42w + 6d

Calculation of EDD

- Naegle's formula:
  \[ EDD = 1^{st} \text{ day of LMP} + 9 \text{ months and 7 days} \]

- Naegle's formula is for 28 days cycles

- Example: Suppose, 1st day of LMP is 15 March

  - Cycle length = 28
  - EDD = 15 March + 9 months = December
  - Date + 7 days = 15 + 7 = 22nd Dec

  - Cycle length > 28
    - EDD = 22nd Dec
    - Add difference between cycle length and 28
      - E.g.: if 30 days
    - Subtract difference between cycle length and 28
      - E.g.: if 25 days,

  - Cycle length < 28
    - EDD = 22nd Dec + (30 - 28)
    - E.g.: if 24 days
    - = 24th Dec
Only 4% of females actually deliver on EDD
50% females deliver either 1 week before or 1 week after EDD

**Period of viability**

- UI = 20 weeks
- INDIA = 28 weeks
- WHO = 24 weeks

- Abortion:
  - When pregnancy loss occurs
  - < 20 weeks
  - > 20 weeks
  - Abortion
  - Intrauterine death of fetus
  - WHO definition if weight < 500 g
  - WHO definition if weight > 500 g

**Gravida and parity**

- Gravida: Number of times a female has conceived
  - Present pregnancy is included
- Parity: Number of pregnancy beyond 20 weeks
  - Present pregnancy is not counted
- Twins / Triplets are taken as a single pregnancy

- 1st method: \( G_x P_a \)
  - \( x \)-no. of times of conception
  - \( a \)-no. of pregnancy > 20w

- Examples
  - Case 1: A pregnant female @ 24 weeks has history of 1 full term live baby @ 37 w
    - Answer: \( G_x P_1 \)
  - Case 2: A pregnant female @ 30w has history of twin delivery @ 36w 5 years back and history of abortion 2 years back
    - Answer: \( G_x P_1 \)
• 2nd method: $E_aP_{a+b}$
  \[ A - \text{no. of pregnancy} > \text{ACW} \]
  \[ B - \text{no. of abortions} \]

• Examples
  - Case 1: $E_1P_{10}$
  - Case 2: $E_3P_{11}$

• 3rd method: $E_aP_{a+b+c+d}$
  - Also called as
  - GTPAL system
  \[ a-\text{no. of Term deliveries} \]
  \[ b-\text{no. of Preterm deliveries} \]
  \[ c-\text{no. of Abortions} \]
  \[ d-\text{no. of Live births} \]

**Signs of pregnancy**

• Signs of early pregnancy
  - Goodell's sign: Earliest sign
    Softening of cervix at 6 weeks
  - Hegar's sign:
    [Image of Hegar's sign]

  Softening of the lower part of the uterus on bimanual examination. It is seen at 6-10 weeks. This is because the products of conception are limited to the upper part of uterus. It is the second sign.

The following signs are positive at 8 weeks
  - Osiander sign: Pulsations can be felt in the lateral fornix of vagina.
  - Jacquemier's sign or Chadwick sign: Bluish discolouration of vagina during pregnancy
  - Piskacek sign: Asymmetrical enlargement of uterus in early pregnancy due to lateral implantation
  - Palmer's sign: Feel regular rhythmic contraction of uterus
• Signs of pregnancy:
  - Presumptive signs: Symptoms felt by the patient
    Eg: Amenorrhea, morning sickness, increased urinary frequency
  - Probable signs: Felt by the examiner but is not diagnostic
    Eg: All early signs of pregnancy
  - Positive / absolute signs: 100% patient is pregnant
    1. Feel fetal movements / fetal parts: quickening is the perception of fetal movements by the mother. Fetal parts are felt at 20 weeks
    2. Hear fetal heart sounds → Stethoscope = 18-20 weeks
    Doppler = 10 weeks
    3. USG evidence of pregnancy
    4. If on x-ray, fetal skeleton can be seen
    Note: X-ray is contraindicated in pregnancy

• Pseudocyesis / False pregnancy
  - Patient is not pregnant but believes she is pregnant
  - None of the positive signs of pregnancy is seen

Antenatal visits

• Ideal number of visits:
  - Till 28 weeks = Once/month
  28-36 w = Twice/month
  > 36 w = Every week
  Total = 12-15 visits

• WHO recommends at least 4 visits

• Government of India recommends at least 8 visits
  1st → 20 weeks
  2nd → 32 weeks
  3rd → 36 weeks

• Caloric requirement in pregnancy = +350 kcal/day
  - Trimester wise, 1 trimester = +0 kcal/day
    1st trimester = +350 kcal/day
    11 trimester = +450 kcal/day

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- Requirement for moderately active female = 2200 kcal/day
  Total caloric requirement = 2200 + 350
  = 2550 kcal/day

Vaccination in pregnancy

- All live vaccines are contraindicated in pregnancy
- All killed vaccines can be given in pregnancy
- Vaccines that are absolutely safe
  H → Hepatitis A/B
  I → Influenza
  T → Tetanus toxoid
  R → Rabies vaccine

- Vaccines that can be given in special circumstances
  (during epidemics)
  Tab → Inactive typhoid vaccine
  P → Pneumococcus
  C → Cholera
  m → Meningococcus

- Vaccines given if pregnant female is travelling to endemic area
  Yellow fever
  Inactive polio vaccine

- Vaccines absolutely contraindicated
  
  | MMR          | Atleast one month is needed after taking these vaccines before she can conceive  |
  | Smallpox     |                                      |
  | Chickenpox   |                                      |
  | BCG          |                                      |
  | Herpes Zoster|                                      |
  | HPV          |                                      |

  If pregnant within 1 month, then MTP is not required
MOLAR PREGNANCY: GESTATIONAL TROPHOBLASTIC NEOPLASIA

Gestational Trophoblastic Neoplasia (GTN) - diagnosis 00:00:11

Diagnosed if any 1 of the following is present
1. 4 Consecutive values of hCG shows plateau (≤ 10%) on Day 1, 7, 14, 21
2. 3 consecutive \([D_1, D_2, D_3]\) \(β\)-hCG values (>10%)
3. On Histopathological examination — Choriocarcinoma
4. hCG level remains high even after 6 months of suction evacuation
   [Normally, partial mole — normal hCG by 7 weeks
    Complete mole — normal hCG by 9 weeks]

Gestational Trophoblastic Neoplasia (GTN) - presentation 00:02:54

1. Continuous bleed per vagina even after evacuation
2. Shock [in case of invasive mole]
3. Uterine sub involution
4. Persistence of theca lutein cyst
   [Normally, disappears after 2-4 months of evacuation]
5. Metastasis
   [More common site ⇒ Lungs > vagina > pelvis]

Note:
A: Most common GTN after molar pregnancy — Invasive mole
   ↓
   * Also known as chorioadenoma destrusens
   * Invades myometrium ⇒ Hysterectomy
   * Chorionic villi present
B. Choriocarcinoma. Most commonly develops after molar pregnancy
   * A low risk choriocarcinoma.
C. High risk choriocarcinoma. More commonly develops after full term delivery
D. Most common GTN to develop after full term delivery ⇒ Choriocarcinoma.
Choriocarcinoma

- Tumor marker - $\beta$ hCG
- Most common route of metastasis - Hematogenous
- Most common site of metastasis - Lung > vagina > pelvis (75%)

Lung metastasis in choriocarcinoma
- Occurs in stage 3
- X-ray appearance

  Most common 2nd most common

  Cannon ball appearance Snow storm appearance

- Snow storm appearance

  On ultrasound On chest X-ray

  Complete mole Lung metastasis in choriocarcinoma.

Vaginal metastasis in choriocarcinoma
- Occurs in stage 2
- Most common site of metastasis ⇒ Just below the urethra

[Suburethral metastasis]

WHO score for choriocarcinoma

<table>
<thead>
<tr>
<th>Low Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age of patient</td>
<td>2. Age of patient</td>
</tr>
<tr>
<td>40 yrs</td>
<td>40 yrs</td>
</tr>
<tr>
<td>2. hCG levels</td>
<td>2. hCG levels</td>
</tr>
<tr>
<td>&lt; 10^4 IU</td>
<td>&gt; 10^4 IU</td>
</tr>
<tr>
<td>3. Type of antecedent pregnancy</td>
<td>3. Type of antecedent pregnancy</td>
</tr>
<tr>
<td>Molar pregnancy</td>
<td>Molar pregnancy</td>
</tr>
<tr>
<td>&lt; 4 months</td>
<td>&gt; 12 months</td>
</tr>
<tr>
<td>4. Duration of antecedent pregnancy</td>
<td>4. Duration of antecedent pregnancy</td>
</tr>
<tr>
<td>Lung</td>
<td>Lung</td>
</tr>
<tr>
<td>5. metastasis</td>
<td>5. metastasis</td>
</tr>
<tr>
<td>&lt; 4</td>
<td>&gt; 8</td>
</tr>
<tr>
<td>6. Number of metastasis</td>
<td>6. Number of metastasis</td>
</tr>
<tr>
<td>&lt; 5</td>
<td>&gt; 5</td>
</tr>
<tr>
<td>7. Size of tumor</td>
<td>7. Size of tumor</td>
</tr>
<tr>
<td>&lt; 3 cms</td>
<td>&gt; 5 cms</td>
</tr>
<tr>
<td>8. History of chemotherapy</td>
<td>8. History of chemotherapy</td>
</tr>
<tr>
<td>No</td>
<td>multiagent chemotherapy</td>
</tr>
</tbody>
</table>
- Total score = < 6 \Rightarrow \text{Low risk}
- Total score = \geq 27 \Rightarrow \text{High risk}

Treatment
- Choriocarcinoma are chemosensitive tumors
- In case of:
  - Low risk choriocarcinoma \Rightarrow \text{Single agent chemotherapy - Methotrexate}
  - High risk choriocarcinoma \Rightarrow \text{Multi agent chemotherapy - Bagshaw regime}
  - E - Etoposide
  - m - Methotrexate
  - A - Actinomycin
  - C - Cyclophosphamide
  - O - Oncovin

Follow up
- Low risk \Rightarrow \text{Follow up x 1 year}
- High risk \Rightarrow \text{Follow up x 2 year [check \beta \text{hCG level}]}
RH ISOIMMUNIZATION: MANAGEMENT

Category -1 RH negative pregnancy

Rh -ve pregnant woman at ANC
↓
1st Investigation: Husband’s Rh - Status
↓
If husband is Rh +ve

Indirect Coombs test (ICT) on mother’s blood
at 12, 20, 28 weeks
↓
If negative: mother not yet sensitized (no antibody)
↓
Administer Anti-D to mother
at 28 - weeks of pregnancy
Antepartum prophylaxis

Mechanism:

\[ \text{mother} \]

\[ \text{Fetal RBC destroyed by anti-D} \]

\[ \text{maternal immune system not stimulated} \]

\[ \text{PLACENTA} \]

\[ \text{300 mcg of Anti-D} \rightarrow \text{Hemolyse 15ml of fetal blood} \]

If ICT +ve
↓
maternal immune system stimulated
↓
Anti-D is of no use
↓

Classified as \rightarrow \text{Category 2: RH isoimmunised pregnancy}
One-liners:

1. Rh antigen develops → 30-40 days after fertilization (7.5 weeks of pregnancy)

2. Maximum chance of fetomaternal haemorrhage (FMH): Beyond 28 weeks

3. 0.1 ml fetal blood (minimum requirement to stimulate maternal immune system)

4. Anti-D prophylaxis → Effective for 12 weeks

5. Antepartum prophylaxis: Dose of anti-D
   \[\downarrow\]
   \[300 \text{ mcg (1500 IU)}\]
   \[\downarrow\]
   Neutralize 15ml of fetal blood
   \[[100 \text{ mcg anti-D } \rightarrow \text{ neutralize } 4 \text{ ml of fetal blood}]\]

Anti-D after delivery:

\[\text{Post-partum prophylaxis}\]

\[\downarrow\]

Dose of anti-D calculated based on amount of fetal blood in maternal circulation

15 ml fetal blood → 300 mcg (1500 IU)
4 ml fetal blood → 100 mcg (500 IU)

- For every ml of fetal blood above 4ml (500IU)
  \[\downarrow\]
  Give 125 IU of anti-D (polyclonal antibody)

- Intra-muscular injection → Deltoid or Anterolateral aspect of thigh
Category 2 Rh negative pregnancy

- A/H/A Rh isoimmunised pregnancy
- ICT +ve (at any time pregnancy)
- No role of prophylaxis anti-D administration

- Maternal immune system stimulated and producing antibodies
  ↓
  Determine antibody titre

Antibody titre:
  ↑ 1:16 → Critical titre
⇒ Significant antibodies present
  ↓
⇒ Will cross placenta and cause fetal hemolysis

Antibody titre

↓

Below critical titre

↓ Follow-up antibody titre
  every 4 weekly → up to 24 weeks
  every 2 weeks after that
  ↓
  If titre < critical titre
  ↓
  Deliver at 37-38 wks

↑ Titre ≥ critical titre

↓ Significant antibodies
  ⇒ Cross placenta and cause fetal hemolysis
  ↓ Calculate amount of fetal hemolysis

To calculate fetal hemolysis

1. Amniocentesis
2. Doppler of middle cerebral (mCA) → Peak systolic velocity
Amniocentesis:

- Fetal hemolysis $\rightarrow$ ↑ bilirubin $\rightarrow$ excreted in amniotic fluid
- Collect amniotic fluid in dark-coloured bottle

↓

Spectrophotometric analysis
measure optical density of fluid between
350 - 750 nm.

↓

Bilirubin absorbs light at 450nm.

Bulge at 450 nm - bilirubin

$\Delta \text{OD}$: height of bulge $\rightarrow$ level of bilirubin.

- Bilirubin $\rightarrow$ bulge at 450 nm
- Meconium $\rightarrow$ bulge at 410 nm
- Blood $\rightarrow$ bulge at 412 nm
Liley's graph

1. Liley's graph → plotted after 27 weeks.

Zone 1: mild: Amniocentesis every 4 weekly.
Zone 2: moderate: Amniocentesis every 2 weeks.
Upper part of zone 2 + zone 3 → Severe disease

Pregnancy < 34 weeks
↓
Intravenous blood transfusion

Pregnancy ≥ 34 weeks
↓
Delivery

2. Robertson graph: 12 zones
3. Queenan graph:
   Advantage → From 14 weeks onwards.

Now, middle cerebral artery (MCA) Doppler is used
MCA doppler

Antibody titre $\geq 1 : 10$

$\downarrow$

middle cerebral artery
doppler

$\downarrow$

USG

Hydrops fetalis

MCA Doppler $\rightarrow$ Peak Systolic Velocity (PSV)

$\downarrow$

Normal

$< 1.5$ mOM

$\downarrow$

Severe fetal anemia.

$\geq 1.5$ mOM

Repeat after every
2 a weeks

$\downarrow$

Deliver at
37-38 weeks

$\downarrow$

Hematocrit of fetus
(cordocentesis)

If HCT $< 30$

$\downarrow$

In utero transfusion

$< 34$ weeks

$\downarrow$

Deliver
immediately

$\geq 34$ weeks

1. Pregnancy terminated at 37 - 38 weeks when:
   1. PSV in MCA Doppler $< 1.5$ mOM
      2. If critical titre of antibody $< 1 : 10$

2. Pregnancy terminated at 34 weeks when:
   1. Hydrops fetalis on USG
      2. Amniocentesis $\rightarrow$ Delta OD between upper zone 2
         and zone 3
      3. PSV in MCA Doppler $\geq 1.5$ mOM
Category 3 RH negative pregnancy

Rh negative woman
↓
H/o hydrops fetalis
in previous pregnancy
↓
Directly monitor PSV in
MCA Doppler
(no need to monitor antibody titre)

Delivery in Rh -ve patient

- Vaginal delivery (C-Section: only for obstetrical indications)

After delivery:
1. Inj. methyl ergometrine contraindicated
2. Early cord clamping
3. Tests on fetus:
   Rh status of fetus
   Bilirubin
   Hematocrit
   Direct Coomb’s test

   If fetus is Rh +ve
   and direct coombs test (DCT) is -ve
   ↓
   Administer Anti - D to mother within
   72 hours of delivery
   ↓
   Postpartum prophylaxis
   (protect subsequent pregnancies)

- Anti - D can be given upto 28 days
- Dose of anti - D: 300 mcg (1500 IU)
  ↓
  Neutralize 15ml of fetal blood
Tests after Rh negative delivery

After delivery, calculate fetal blood in maternal circulation

RCOG Guideline: Rosette test within 4 hrs of delivery

Positive

- Fetal blood ≥ 15 ml

- Weihauer Betke Test
  (Quantitative test)

Negative

- Fetal blood < 15 ml
- Dose of anti-D
  - 300 mcg

Principle: HbF → resistant to acid and alkali
HbA → sensitive to acid and alkali

Reagent: Citric acid phosphate buffer.

Procedure:

Reagent + maternal blood → Ghost cells
  (maternal RBCs hemolysed)
  i.e. RBCs elute
  → Test a.k.a. Acid elution test

If fetal blood Θ in sample

- HbF does not hemolyse

- Fetal RBC Θ on microscopy

- Study 25 high power field (HPF)

- Fetal blood (ml) = \[ \frac{\text{Fetal RBC} / \text{HPF}}{\text{maternal RBC} / \text{HPF}} \times 2400 \]

- Calculate does of anti-D

500 IU → 4 ml fetal blood

For every ml beyond 4 ml → add 125 IU anti-D
• Singers alkali denaturation test / APT test
  - Differentiate between fetal blood and maternal blood
  - Quantitative test.

Kleihauer betke test: differentiate between fetal and maternal RBC

Anti-D also given to Rh-ve women after:
  - Ectopic pregnancy
  - Molar pregnancy
  - Chorionic villi sampling
  - Amniocentesis
  - Antepartum haemorrhage
  - Attempting version
  - Abdominal trauma
  - Abortion

\[ \downarrow \]
\[ \begin{array}{ll}
< 12 \text{ weeks} & \geq 12 \text{ weeks} \\
(1st \text{ trimester}) & \\
\bullet \text{ All abortion except threatened and complete} & \text{Give anti-D to all abortions}
\end{array} \]

\[ \rightarrow \text{ anti-D given} \]

Dose:
\[ \begin{array}{ll}
< 12 \text{ weeks}: 50 \text{ mcg} \\
\geq 12 \text{ weeks}: \text{ACOG} \rightarrow 300 \text{ mcg} \\
\text{RCOG} \rightarrow 100 \text{ mcg}
\end{array} \]
ANATOMY OF FEMALE GENITAL TRACT: VAGINA

- Vagina:
  * Fibromuscular canal connecting cervix to introitus
  * Posterior wall longer than anterior wall
  * 4 fornices → Anterior, posterior & 2 lateral
    Anterior fornix: Shallowest
    Posterior fornix: Deepest

- Cervix and lateral fornix of vagina are related to:
  1. Uterine artery
  2. Ureter
  3. Cardinal / Mackenrodt’s ligament

- Vagina has no glands
  Vaginal discharges → Endocervical glands
  Endometrial glands
  Bartholin glands (only at time of intercourse)

  * Cervical discharge → Alkaline (6-8)

Doderlein bacilli and pH of vagina

- Vagina has inhabitant bacteria
  ↓
  Doderlein bacilli (Lactobacilli)
  ↓
  Convert glycogen to lactic acid
  ∴ vaginal pH → Acidic

Doderlein bacilli → Present at birth
  ↓
  Disappear after 10 days
  ↓
  Reappear at puberty
  ↓
  Disappear at menopause
<table>
<thead>
<tr>
<th>Age</th>
<th>Vaginal pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>* At birth / Newborn female</td>
<td>Acidic</td>
</tr>
<tr>
<td>* Childhood</td>
<td>5-8</td>
</tr>
<tr>
<td>(before puberty)</td>
<td>pH changes from alkaline → acidic</td>
</tr>
<tr>
<td>* At puberty</td>
<td>4.5</td>
</tr>
<tr>
<td>* At reproductive age</td>
<td>4-8</td>
</tr>
<tr>
<td>* At menopause</td>
<td>3.5</td>
</tr>
<tr>
<td>* At pregnancy</td>
<td>4-8</td>
</tr>
<tr>
<td>* During menstruation</td>
<td>(blood: Alkaline)</td>
</tr>
</tbody>
</table>

**Lining epithelium of vagina**

Stratified squamous epithelium (non-keratinised)

* In newborn → Vagina lined by transitional epithelium

**Blood supply:**

- upper ⅔: Descending cervico-vaginal artery
- middle ⅓: Internal pudendal artery
- Lower ⅓: Middle rectal artery

**Lymphatic drainage:**

- upper ⅔: Hypogastric, Obturator, Pre-sacral, I
  - External iliac (HOPE)
- middle ⅓: Internal iliac lymph node
- Lower ⅓: Superficial inguinal nodes

**Nerve supply:**

- upper part: S₄-S₅
- Lower part: Pudendal nerve

**Development of vagina:**

- upper ⅔rd: Mullerian duct (mesodermal)
- Lower ⅓rd: Sinovaginal bulb (urogenital sinus) (endodermal)
Broad ligament

Parts of broad ligament:
1. mesosalpinx
2. mesovarium
3. mesometrium

Contents of broad ligament

- Tube structures
  - 1. ureter
  - a. Fallopian tube
  - Note: ovary not a content
- 4 ligaments
  - A: ovarian
  - B: infundibulo pelvic
  - C: Round ligament
  - D: Cardinal ligament
- Vestigial structures
  - 1. Epoophoron
  - 2. Para-epoophoron
  - 3. Gartner's duct
- Vessels
  - 1. Ovarian artery
  - 2. Uterine artery

Levator ani

Pelvis is separated from perineum by:
- Levator ani muscle
  - Pelvic diaphragm
    - Pubococcygeus
    - Iliococcygeus

Note: Ischiococcygeus not a part of levator ani

Pelvic Diaphragm of Female
Inferior View

- Pubourethralis
- Pubo rectalis
- Pubo vaginalis
- Pubo rectalis
- Pubococcygeus proper
- Ischiococcygeus
- Iliococcygeus
Perineum

urogenital triangle:

- Deep perineal pouch: Deep transverse perinei muscle

Attachments of perineal body:
- Bulbocavernosus (Bulbo cavernosus)
- Levator ani muscle
- Muscles of external anal sphincter
- Superficial transverse perinei
- Sphincter urethrae
- Deep transverse perinei

Not attached → Ischiocavernosus
Parts of sphincter urethrae

1. Compressor urethrae
2. Sphincter urethrovaginalis