Obstetrics & Gynaecology

Relavant Anatomy

⇒ Uterus:
  ⇒ Mullerian duct:
    ⇒ Embryologically derived from mullerian ducts (Paramesonephric ducts)

⇒ Mullerian ducts appears at 6wks POG

⇒ Remnants: Disappears of mullerian ducts is 9wks POG

⇒ Remnants are:
  - Uterus masculinus
  - Appendix of Testis

Wolffian ducts:
  ⇒ appears at 6wks, disappears at 9wks in female.

⇒ Remnants:
  - Epoophoron → Cranial remnant of mesonephric duct
  - Paroophoron → Caudal remnant of mesonephric tubule
    - Gartner's duct
  - Hydatid of Morgagni — Ø blood vessel Blood Gartner's duct

- All these are contents of broad lig.
- Paroophoron + in medial part of broad lig.
- All except paroophoron + in lateral part of broad lig.
- Caudal remnant of mesonephric duct IS Gartner's duct.
Location of Gartner's cyst duct + in anterolateral vaginal wall.

- Uterus:
  - Shape: pyriform
  - Shape of cavity: Δ
    (cut section)

  - Shape of cavity:
    - Coronal section: Δ
    - Sagittal section: Cleft like

- There are 2 meso & para mesonephric ducts.

- The mesonephric ducts fuse to form uterus in fetus at 10 wks in caudal to cranial fashion.

- Cavity of uterus form (septa get dissolved) - 18-20 wk post.
  in caudal to cranial fashion.

- wt:
  - Nulliparous: 50-70 gm
  - Multiparous: 80 gm

  length of uterus:
  - Nulliparous: 6-8 cm
  - Multiparous: 9-10 cm

  volume of uterus:
  - Nonpregnant: 10 ml
  - Pregnant: 51 ml
- wt of pregnant uterus at term: 1000gm (1.1kg)
- wt of uterus immediately after delivery: 1600gm (1kg)

In uterus: 5L occurs due to **Hypertrophy** >> **Hyperplasia** in pregnancy.

- wt of uterus after 6wk of delivery: 80-100gm (due to hypertrophy)

**Fundus:**
- Part of uterus above attachment of Fallopian tube

**Body:**
- Body opens into cervix at internal OS.

⇒ **Internal OS:**

- **Anatomical OS**
- **Histological OS**

Histological OS lies below the Anatomical OS. The part below Anatomical & Histological is **Isthmus**

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JAIN STATIONERY
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\[ \text{LUS} \implies \text{It begins to form after 1st Trimester} \]

- Formed by isthmus.
- At term, LUS formed, - Isthmus (70%), Cervix (30%).

Effacement: Taking up of cervix into LUS (Isthmus).

To identify the LUS of uterus is:
- by attachment of loose fold of peritoneum (uterovesical)

\[ \text{Fold of peritoneum} \]

\[ \rightarrow \text{Below loose attachment is LUS} \]

\[ \rightarrow \text{Incision: - 1. Transverse / Pfannensteil} \]

\[ \text{KERR'S Incision} \implies \text{by Dr. Munrothar} \]

2. Vertical Incision \rightarrow KRONIG's

\[ \rightarrow \text{Incision in upper segment: Classical} \]

\[ \rightarrow \text{Risk of rupture of classical scar is 4-9\%} \]

\[ \text{Strong is KERR's} \implies \text{risk is 02-15\% (Theoretically)} \]

Risk for KRONIG's is 1-7\%.

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A-P relations:
- (Antero-Posterior relation)
  - Round lig.
  - Fallopian Tube
  - Ovarian lig.

Sup-Inf. relation: Fallopian Tube superiority placed
- Round lig. & ovarian lig. attached at same level

Tubal cause of failure of ligation is identifying the wrong structure.

Derived from:
- FT: Mullerian ducts
- RL: distal Gubernaculum
- OL: proximal

Which lig. are main supports of uterus:
1. Ant: Pubocervical
2. Post: Uterosacral
3. Lat: Transverse cervical lig. also k/a mackenrodtlig.
4. Down: Levator ani

All main support of true uterus: Transverse cervical lig. (Cardinal lig.)
- Not a support of uterus: Broad lig (not a support). It is only fold of peritoneum.

- Contents of Broad lig:
  - FT / OvL
  - Connective tissue
  - Bladder for supports
  - Remnant of wolffian duct
  - Ovary for supports
  - Cardinal ligament - lower boundary.

- It is from upper part of cervix to lat. pelvic wall.
- Tunnel where arteries pass through it.

- Ulg. responsible for Anteversion & Ante Flexion - Round lig.

  **Anteversion**
  80° Uterus & vagina

  **Ante Flexion**
  80° Uterus & cervix

  90°

  120°

  At level of Internal OS

- Ulg. which prevents Retroversion is - RL + uterosacral

- Blood supply of uterus: Uterine artery - is a branch of Interna iliaca a. (Anterior division)
Uterine artery is an attention artery, bleed more so we do B/L ligation at level of internal os.

If we ligate uterine artery, urine enters pelvic part which is known as water under the bridge.

Which is m/c site of uterine ureteric injury - where it is crossed by uterine artery.

1st: Pelvic brim (where it is crossed by ovarian vessels)

To prevent ureteric injury is - know the Anatomy.

Uterine
Arcuate (branch)
Radial

(Urue the best)

Grande
Basal → Basal endometrium → Regeneration
S spiral → supply superficial/functional endometria.

Radial supplies inner 2/3rd of myometrium

Arcuate supplies outer 1/3rd of myometrium.
**Endometrium**

- D3 (immediately after menstruation - 0.5mm)

**Thickness of Endometrium**
- Mid cycle - 3mm
- Luteal phase - 5-6mm
- Implantation - 10-12mm.

Lining epithelium: Single layer ciliated columnar epithelium.

- Uterus is muscular sigh.

- Myometrium: Thickness (1.5-2.5cm)
  - Outer longitudinal
  - Middle criss cross - lying ligature
  - Inner circular: Sphincter-like act at 3 points
    - a. Rt. Cornu
    - b. Lt. Cornu (opening FT into uterine cavity)
    - c. Internal os

- Anatomical sphincter → Intramural / Interstitial part of FT.

Lymphatic drainage: Int. Iliac (m) some External Iliac.
Fundus of uterus drains into Paracolic group of LN.

Nerve Supply:

→ Labour pain pass through T₁₀ - L₁ nerve segments.

→ Painless labour: we give Epidural Analgesia (sensory block).

Before pain is ↓.

→ Level of Drug given epidural Analgesia - T₁₀ level, Bupivacain 0.25

→ Level of block when there is fetal distress, Instrumental delivery is Pudendal N. Block (S₂, S₃, S₄).

→ The lig. pierced when we give pudendal N. Block is Sacrospinous ligament.

Cervix

→ Embryologically derived from - mullerian duct.

Cervix opens into vagina - Ext. Os

Uterus opens into vagina - Internal Os.

Shape: Fusiform / Spindle shape.

→ Shape of cavity → Transverse (Fusiform / Spindle shape)
Ext. Os: - Nulliparous - Circular
- Multiparous - Transverse Slit

Shape of ext. os. immediately after delivery - slit

Cervix made up of:
- Connective tissue 10-15% - smooth muscle
  - Cx smoothens due to this in effacement

Changes during effacement:
- ↑ hyaluronic acid
- ↓ levels of dermatan sulfate & chondroitin sulfate

Endo Cx

ECTO Cx

Tall columnar epithelium
- Squamous epithelium

Squamo-columnar
Junction seen
at ecto Cx

At Squamo-columnar Junction:
→ Cells undergoing metaplasia so it is Transformation Zone

Bid supply of Cx: - Uterine a.
- Descending cervical arteries

Para-cervical block is given to pull Cx.

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Paracervical block should not be given at 3 & 9 o'clock position due to branches of descending cervical arteries.

Lymphatic drainage:
- Internal iliac
- External iliac

Nerve supply:
- S₂, S₃, S₄, pudendal nerve.

Cervix: Corpus

At Birth: 1 : 1
Before puberty: 2 : 1
After puberty: 1 : 2
Reproductive life: 1 : 3
Menopause: 1 : 1

Fallopian Tubes (FT)

- Derived from mullerian duct
- Unfused cranial part form FT
- 10-12 cm long

Parts from medial to lateral:

1) Intramural | Interstitial -> 2cm - narrowest part: 0.7
2) Isthmus -> 3cm -> 1mm
3) Ampulla -> 5cm - Diameter -> 6mm
4) Fimbrial end ->
Accounts

Anatomical sphincter \rightarrow Intramural

Physiological sphincter \rightarrow Isthmus

\rightarrow Site of fertilization \rightarrow Ampulla

\rightarrow m/c site of ectopy \rightarrow Ampulla. Because it is the site of fertilization

\rightarrow It has highest no. of plicae (mucosal folds)

\rightarrow Conceptus stays in Ampulla for 3 days.

\rightarrow FT has 3 Types of cells

(a) Ciliated Cells

(b) Secretory Cells

(c) PEG cells - Characteristic feature \rightarrow Resting cells of FT

\rightarrow Epithelium of FT: Single layer ciliated columnar epithelium.

\rightarrow Blood supply: Medial part supplied by \rightarrow Uterine artery

\rightarrow Lateral part \rightarrow Ovarian artery.

\rightarrow Pain of unruptured ectopic pregnancy is by \( T_{11} T_{12} L_{1} \)

\rightarrow Nerve Supply: \( T_{11} T_{12} L_{1} \)

\rightarrow Lymph Drainage: - Para-aortic LN

\rightarrow Superficial Inguinal LN (some)

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Vagina.

→ Embryologically: derived from:

upper 2/3rd: mullerian duct → mesoderm of mo (mo

lower 1/3rd: urogenital sinus → endoderm of u6 (uas) ↓ i.e. sinovaginal bulb.

→ vagina has 7-10 cm long

=) 4 walls: Ant. post. later

Post-wall is longer than Ant. by 2 cm

Fornix:

⇒ 4 fornix 2 lat

1 Ant.

1 Post - deepest fornix

- Fold of Peritoneum covering post-wall is Pouch of Douglas (POD)

→ Gut is in POD

→ Uterus making an angle to vagina → 90°

vagina making an angle to horizontal → 45°

→ No glands in vagina.

⇒ Main source of secretions of vagina:

1) Transudation from vaginal walls
2) Cervical secretions
3) Bartholin glands (secrete only at time of coitus)
$pH$ — Acidic (due to Bacilli duoderales form lactic acid)
- Estrogen [mature cells produce glycogen]
- Duodena Bacilli
- If acidic $pH$ is protective against infection

$pH$ throughout Reproductive life is 4 - 4.5

During menstruation
- 6.5 - 7.5
- Post menopausal women
- Prepubertal age

At birth $\rightarrow$ 5.5 - 6.0
(Exposure to maternal hormones)

Pregnancy $\rightarrow$ 4 $\rightarrow$ more acidic

Blood supply:
- Supplied by:
  - Upper $\frac{1}{3}$ $\rightarrow$ descending vaginal branches of uterine a. (uterus)
  - Middle $\frac{2}{3}$ $\rightarrow$ Inf. vesical a. (ant to bladder)
  - Lower $\frac{1}{3}$ $\rightarrow$ middle rectal a. (ant to rectum)

Lymphatic drainage:
- Drains into:
  - Upper $\frac{1}{3}$ $\rightarrow$ Ext. Iliac LN
  - Middle $\frac{2}{3}$ $\rightarrow$ Int. Iliac LN
  - Lower $\frac{1}{3}$ $\rightarrow$ Superficial Inguinal LN.
Nerve supply: S2, S3, S4, pudendal nerve.

**External Genitalia.**

- Genital tubercle
- Clitoris
- Penis
- Genital fold
- Labia minora
- Penile urethra
- Genital swelling
- Labia majora
- Scrotum

**Homologous Structures**

1. **Bulbourethral glands/Cowper's gland:**
   - In superficial perineal pouch
   - In deep perineal pouch
   - Cyst in post-lat wall of vagina

2. **Prostate gland:**
   - **Bartholin's gland:**
     - Epithelium:
       - Columnar: 1 gland beneath labia majora
       - Gland at junct. of ant. 2/3rd urethral glands
     - Epithelium:
       - Transitional of duct

- **Bartholin's gland cyst:**
  - In post-lat wall of vagina.

- **Secretions:**
  - Alkaline
Rx of asymptomatic Bartholin's cyst: No Rx

2. If it produces any symptoms or if it is recurrent cyst: MARSUPIALIZATION (extraction/sing of gland)

Rx of Bartholin's abscess: I & D + word catheter. It prevents recurrence.

Bartholin gland causes adenocarcinoma.

Lymphatic drainage of clitoris: superficial inguinal LN. Labia minora

Gland's clitoris: drain into deep inguinal LN. Labia majora

Glands of Cloquet

Most common cancer: Vulva Can.

Relevant Embryology

Ovary/Testis — genital ridge (gonadal ridge)

Genital ridge appears at 5 wks in fetus in both.

Testis: SRY gene (short arm of Y chromosome)

Appearance: Testis → 7 weeks.

Ovary → 8 wks.

→ For development of ovary — both X chromosomes (XX) required.

Ipsilateral: Disappearance of Mullerian duct by mIS (Sertoli cell of testis)

Ovary: Germinal epithelium which is single layer of cuboidal epithelium

→ 3x2x1 cm.
Ovary lies in Ovarian Fossa (lat. pelvic wall)

Relations of ovarian fossa:

Superiorly: Ext. Iliac vein

Inferiorly: Levatores ani

Anteriorly: Post. Leaephy by Bl

Laterally: Obliterated umbilical vein

Posteriorly: Ureter / Internal iliac vessel

Medially: Ovarian ligament

Laterally: Obturator nerve & vessels

Infundibulopelvic lig.: Suspends the ovary to lat. pelvic wall

→ Ovarian vessels ⊕ in this

Cortex: Follicles in the ovary ⊕ in this. Follicles + stroma

Medulla: Stroma + blood vessels + nerve (vascular region)

Contains Hilus cells

Blood supply to ovary: Ovarian artery: Branch of abdominal vein cobra. at L2 level.

Veins: RT Ovarian vein → IVC

Lt. Ovarian vein → Lt. Renal vein.
nerve supply: hypogastric plexus (T10-T12)

Referred pain of ovary: medial aspect of thigh

Cutaneous Branch of Obturator nerve.

Lymphatic drainage: Paraortic group of LN

⇒ Primordial Germs: - old cells.
   (PGC) - derived from Ectoderm (Germ layer) in origin
   - area is called Epiblast

⇒ Oogenesis

Epiblast

⇒ Yolk sac (3wks POG)
  ↓
  Genital ridge (6wks POG)
  ↓
  Oogonia → 9wks POG
  ↓
  Primary oocyte → 12wks POG
  ↓

Follicle formation: Begins at 14wks
  Complete by 24wks.

⇒ Oogenesis: - start in utero.

⇒ Perimenopausal ovary vol = 7-8ml.
Oogenesis:

- Oogonia (diploid)
  ↓
- 1° oocyte (diploid)
  ↓ mitosis
- 2° oocyte arrested in prophase I - diplotene → Dicyteli

Completed at ovulation → MEIOSIS - I

- 2° oocyte
- 1° polar body
  - more cytoplasm
  - less cytoplasm
  - genetic material
  - genetic material

Completed at fertilization → MEIOSIS - II arrested at metaphase

Female pronucleus

- 2° polar body

From 1° oocyte
  ↓
- 1 ovum (carrying genetic material in centre)

→ Size of mature ovum → 120 μm

→ Size of mature follicle (ovum + corpus luteum) → 18 - 20 mm etc

→ Fertilizable life span of ovum is 24 hrs

→ Size of dormant or primordial follicle = 0.02 mm

→ Size of 1° follicle → 0.1 mm

→ Size of 2° follicle → 0.2 mm

→ Size of 3° follicle → Keeps changing
Spermatogenesis

occurs in → Intrauterine

→ At puberty = 72 days

Spermatogonia (2n)

\[ \downarrow \text{mitosis} \]

1° spermatocyte (2n)

\[ \downarrow \text{meiosis-I} \]

2° spermatocyte → 2° spermatocytes

\[ (n) \]

[Diagram: two cells splitting into two spermatids each]

\[ \downarrow \text{meiosis-II} \]

0 spermatids

2 spermatids

1° spermatocyte form

\[ \downarrow \]

4 spermatids

→ spermatids → sperms

move through epididymis

\[ \downarrow \text{14 days} \]

maturity → motility

→ nuclear material: → golgi apparatus → acrosome/cap

Spermiogenesis: → mitochondrion → middle piece

mito

→ microtubules → Tail
- Fertilizable life span of sperm - 72 hrs
  
  length - 55 μm (50-60)
  
  > Capacitation:
    - Occurs in female genital tract.
    - Begins in cervix & major in FT.

  avg. time required for capacitation - 7 hrs
  
  After capacitation - In Cap FT sperms attain hypermotility.

  1. Sperms undergo acrosomal reaction.
  
  2. Sperms get ability to penetrate protective layer of ovum i.e. zona pellucida.
  
  > Avg. time of sperms after ejaculation to reach site of fertilization is 30 min.

  > Site of fertilization: - Ampulla.

  > After fertilization product formed is zygote conceptus.

  > Conceptus stays in FT upto 3 days.

  > Isthmus stops conceptus to stay in FT.

  > The main cause of movement of conceptus towards uterus is Peristalsis of FT (Cilia contribute).

  > Cilia beats towards uterine cavity.

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→ Conceptus enters uterine cavity → Day 4 (Post fertilization)
   (m) Post ovulation

→ The conceptus enters uterine cavity → morula cavity

→ morula: (16 cell stage)
  Single cell is Blastomere

→ The cell stage does the conceptus enter uterine cavity → 8 cell

→ Implantation begins : $D_6$ (post fertilization) - $D_7$

→ Form of implantation → Blastocyst form.

→ The day the blastocyst form → $D_5$

→ Zonapellucida:
  (2)
  Function: Prevention of polyspermy (by cortical reaction)
  Z.P lost: Just before implantation ($D_5$)

→ Implantation completed by $D_{10}$ (Post fertilization)
Eventually when conceptus goes into uterine cavity,

Capsularis & parietalis Fuse at: 14-16 wks POG (last day from menstruation)

Superfetation: Superfetation in twin pregnancy Theoretically possible

→ at 14-16 wks of POG (after this, superfetation won't occur there is no space in uterine cavity).

Embryonic Stage → upto 8 wks Post Fertilization

→ upto 10 wks from LMP

→ 3-8 wks

Trophoblast

→ CytoTrophoblast

→ Syncitio trophoblast

Source: Cyto trophoblast.

Villous

→ Placental villi

are lined by villous.

Don't express HLA class I

Class II molecules

Entravillous

→ express HLA- Class I,

→ go beyond villi

→ invade decidual, spiral arterioles of mother (in myometrium)

→ Spiral arterioles are resistant to vaso pressor

by regulat Uteroplacental Circulation
Lack of invasion of spiral arterioles by extravillous

In pregnancy, placenta goes ischemic necrosis & release inflammatory mediators & cause preeclampsia.

Fetal membranes:

1) Amnion → now not considered as Trophoblast derivative.
   • It is a derivative of fetal ectoderm
2) Chorion
3) Yolk sac
4) Allantois

Amnion: → Avascular membrane

→ Responsible for Tenisc strength of fetal membrane
   → Inner mass of fetal membrane.

Chorion:

\[ \text{Chorion frondosum} \quad \downarrow \quad \text{Chorion laeve} \quad \downarrow \quad \text{merge to amnion.} \]

Forms placenta

Yolk sac: → 1st site of haematopoiesis.

3wk - 6wk

Hb → Gower 1, 2 → Portland

1st trimester: 6th week → Extramedullary hematopoiesis:

Liver, spleen, LN
→ fetal Hb at term = 18 g%. 

→ Combining HbF = total Hb = 75-80%. (3/4 of the total)

→ Calculation of dose of anti D in an Rh immune mother.

→ Quantitative test: K 8 test (Kleihauer Betke test)

→ test that differentiates fetal RBC from maternal blood

cells in maternal blood contain citric acid phosphate buffer

→ Test that differentiates fetal RBC from maternal blood

→ Test to differentiate fetal & maternal RBC

→ HbF: - less of 2,3 DPG

→ Rh factor

→ It has both acid & alkaline denaturation

→ HbF: alkaline denaturation : APT test

→ Qualitative test

→ RH F:

→ typing:

→ fetal RBC’s are bigger in size but have life span

short i.e. 90 days.

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NOTES FROM JAIN STATIONERY
Bone marrow: The only source of haematopoiesis after 20 weeks

Allantois

The diverticulum that arise from hindgut and grows into the connecting stalk.

Placenta

Hemochorial (no intermingling of maternal & fetal blood).

maternal blood

\[ \begin{array}{c}
\text{Intervillous - I} \\
\text{Villous - V}
\end{array} \]

fetal blood

\[ \begin{array}{c}
\text{I} \\
\text{V}
\end{array} \]

The weight of placenta at term > 500 g

Volume of placenta > 500 ml

Ratio of placenta: Fetus wt at term: 1: 6

When will be the placenta & fetal wt equal: at 17 weeks

Total surface area of villi is 12 m²

Placenta has:

Fetal side / maternal side
placenta anatomically only a product of conceptus, not a maternal product:

→ Fetal placenta:
  → Smooth because it has membranes.

→ Maternal side:
  → Not smooth, divided into lobes
  → Each lobe
    ↓ divided
    3-5 Lobules
    Functional unit of placenta is Lobule/Cotyledon.

→ 1st stem villi
→ 2nd stem villi
→ 3rd stem villi

Solid, mesoderm comes at centre.

D_{13}  \quad D_{16}  \quad D_{21}


troplacental Circulation:

→ Established at D_{12} \text{ to } D_{17}

→ Fetal Circulation established at D_{21}
Uterine blood flow at term is \( \approx \) 750 ml/min.

\[ \rightarrow 10\% \text{ of Cardiac Output} \]

The uteroplacental circulation at term is \( \approx 90\% \).

\[ \rightarrow 450 - 650 \text{ ml/min} \]

Fetal blood volume at term \( \approx 125 \text{ ml/kg} \).

**Intervillous (IV)**

| In 1 intervillous space | \( \approx \) 140 ml of blood (arterioles) |

| How many spiral arterioles in IV space |

\[ \rightarrow 120 \text{ spiral arterioles} \]

\[ \rightarrow \text{O}_2 \text{ saturation} \approx 65 - 75\% \text{ admixed to O}_2 \]

**Umbilical Cord**

| 2 Arteries \( \rightarrow \) 1 vein (left umbilical vein) |

| Down: genetal blood towards placenta |

| Small \( \rightarrow \) Biggers one |

| Oxygenates blood from placenta to heart |

| \( \rightarrow \) Highest \( \text{O}_2 \) Saturation \( \approx 80\% \)

**Notes from**

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UA → UV → DV → DA → FO after birth

U. Artery Umbilicus O. A. Artériás Foramen ovale

⇒ SUA (single umbilical artery): mc vascular anomaly

⇒ ↑ risk of cardiac malformation

⇒ vascular anomaly

⇒ SUA + congenital malformation

⇒ NO ↑ risk of

⇒ aneuploidy (abnormal no.)

⇒ TRISOMY

→ mc vascular problem etc.

→ Avg. length of umbilical cord
  → 55 cm

→ Short cord < 35 cm
  (< 35 cm)

Placenta

→ produces Hormones: Estrogen / 2. Progesterone

  3. Human PL / 4. HCG

→ Estrogen specific for pregnancy → Estradiol / ESTETROL
   E₃
   E₄

⇒ E produced in max amounts in E₂ Estradiol

⇒ Placenta can't synthesize estrogen on its own

because it lacks enzymes 17-hydroxylase.
Placenta uses fetal precursor **DHEAS** to produce **E**.

Placenta can synthesize *progesterone* on its own, by using maternal precursor is **LDL**.

When does placenta take over the function of Corpus luteum → 8 wks (8-10 wks)

**HPL**: Human placental lactogen

In lactation there is no role for **HPL**

HPL only prepares the breast for lactation

HPL appears at maternal serum at −3 weeks

& highest at 34-36 weeks (last trimester)

Functioning of placenta is determined by **HPL**

Main function of **HPL** is: Endocrine (HPL → Lipolysis) is an antinsulin hormone

Fetal growth is determined by Insulin like GF

HPL responsible for Insulin Resistance in Pregnancy.

**HCG**: 2 chains

α β

Non-specific specific chain.

It is shahed by TSH, LH, FSH:

HCG.

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doubling time of HCG → 48 hrs (1-2 days)

HCG can be detected as early as D8 after ovulation

Peak amount of HCG seen at 8-10 wks (60-70 days)

Minimum levels of HCG seen at 16 wks

After 16 wks the value plateaus

After normal delivery/labour, Time taken for HCG to return to normal is 2-4 wks 4-6 wks

HCG starts disappearing from maternal urine after 48 hrs of delivery.

MC used Test: Urine Test UPT test based on Sandwich ELISA

1 IU/ L (20 mIU/ml)

FIA → RIA → ELISA

Critical titre of hCG for Transvaginal Sonography (TVS)

for ectopic

HCG Range is → 1000 - 2000 IU

→ In Trans Abdominal Sonography (TAS) →

Critical titre: 6500 IU
Function of hCG:

1) Which hormone prevent luteolysis → hCG (maintain Corpus luteum of pregnancy)

2) hCG is the first stimulus for testosterone release in pregnancy from male fetus

3) Immuno suppressive

4) Screening & diagnosis:

   a) Molar pregnancy
   b) GHTN: (gestational HTN)
   c) Ectopic pregnancy: (overestimated gestational age: hCG less)
   d) Twin pregnancy
   e) Down syndrome

HCG value >> than the expected age is seen in molar, ectopic, down syndrome.

Down screening: pregnant woman > 35 wks

ACOG universal screening: Every pregnant woman should undergo this Down screening irrespective of age

→ Done in

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Down Screening

1st Trimester

USG

maternal

Serum markers

→ Nuchal Translucency

2nd Trimester

USG

maternal serum markers

→ Nuchal Pd Thickness

→ ≥ 6 mm → abnormal test

→ done btw 16-18 wks

→ quadruple test

3rd → ≥ 6 mm +ve

→ done at 15-20 wks

1st Trimester:

→ Nuchal Translucency done at 11-13 +6 days

→ if thickness ≥ 3 mm, Test +ve.

→ maternal serum markers done at 11-13 weeks

Test is dual test:

Serum β hCG

PAPP-A

(pregnancy associated plasma protein A)

HCG level > normal

β hCG level < normal i.e. expected age

+ve

++ve

Tripple Test

2nd Trimester:

Tripple Test

HCG

AFP

Sr. unconjugated Estriol (SUE 3 )

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Quaduple test: HCG + AFP + SUE3 + Inhibin A

Abnormal

→ Confirmatory for Down's: karyotyping.

1. 1st Trimester: Chorionic villous sampling (can be done after 10 wks but not done < 9 wks because it leads to limb & oro-mandibular defects

   → of 9 wks: Take sample from Chorionic villi.

2. 2nd Trimester:

   → Amniocentesis > 15 wks

   → 11 - 14 wks: Early amnio → Risk of fetal loss

   → 16 - 18 wks: MC Time to do.

   → Sample taken from Amniocytes/fibroblasts

   Dangerous

3. Cardocentesis: Most dangerous abortion > 3 1/2.

   done → 20 wks

→ Fetal skin biopsy is not used for karyotyping.

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2015/10/23 97
TVS

1st evidence of pregnancy on TVS → Gestational sac 'G' sac

'G' sac seen at 4 + 1 day → 4 + 3 days wk = LMP

→ How many days from LMP, we will see 'G' sac on USG

29 - 31 days. (≈ 30 days)

→ How many days after ovulation, we can see 'G' sac on USG

→ (29 - 14) - (31 - 14) = 15 days from ovulation

→ 'G' sac on TAS seen at 5wks

→ 'G' sac on USG can be seen at 5wks

(when we do TAS, we can know TVS also)

→ Cardiac activity on TVS at 5wks POG

→ CA on TAS at 6wks POG.

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09654691327
Physiological Changes in Pregnancy

In pregnancy → BMI ↑ (10-20%)

→ wt. gain is 12.5 kgs.

→ Calorie = 300 kcal/day (350 kcal/day)

→ Na⁺/K⁺ = Retention (due to Estrogen)

⇒ additional H₂O retained is → 6.5 L

- Sr. Na level = Fall (dilutional hyponatremia)

- plasma Osmolality Falls 10 mosm/l

⇒ Pregnancy is a state of insulin Resistance so it is
Characterised by fasting hypoglycemia & postprandial
Hyperglycemia.

⇒ Urine → glycosuria physiological Renal threshold ↓

⇒ Proteinuria - Pathological

WBC count: ↑ (Neutrophilia)

= 20,000 post partum time. (physiological)

Avg. PLT count = ↓ (compare to pre-pregnant count otherwise)
→ All clotting factors ↑ in pregnancy except II & XIII

→ Liver enzymes:
  → Placental Alkaline phosphatase ↑
    (PLAC) Heat stable isoform
  → ALT & AST ↓

→ Gastric emptying time: No change in any trimester
  During labour ↑'s.

→ Lower esophageal sphincter tone ↓ there is ↑ gastritis
  More reflux.

→ Cholestasis → Hormone → estrogen

Chadwick's sign (Jaquimets): Bluish discoloration of
vagina, vulva, cervix at 8 wks.

Hegar's sign: Softening of lower part of uterus Cisterna
  6th wk.

Goodell's sign: Softening of cervix 6 wk

Osiendar's sign: Lat. vaginal Fornix pulsations
  at 8 wks.

Paskacas's sign: One ½ of uterus appears softer than
  the other (lateral implantation)
Palmer's sign: Regular rhythmic uterine contractions

6-8 weeks

Hartmann's sign: At time of implantation, women experience some vaginal bleeding.

CVD

1) ↑ in plasma volume 40% 
   (PV)

2) ↑ in Red cell mass by 20% 
   (Rcm)

3) Hemoilution (Rcm < PV) Hb level falls significantly in pregnancy.

4) Anemia of pregnancy < 11 g/mL

5) Total Iron requirement for pregnancy = 1000 mg.

Iron requirement

1st half - 4 mg/day
2nd half - 6-7 mg/day

6) O₂ carrying capacity of blood ↓ is a function of Hb so ↓

7) Cardiac output ↑ in pregnancy by 40%

8) O₂ demand by tissues ↑ by 20%

9) A-V̄₂ gradient ↓ (In pregnant more oxygenated blood)
⇒ HR ↑ by 20% 10 - 16 bpm
   early Term
   Pregnancy

⇒ Shifted upwards & rotates anteriorly → apex 4th IC

⇒ all heart sounds are loud

⇒ S3 sound heard.

⇒ Systolic murmur - upto grade 2 it is physiological
   (Ejection Sm)

⇒ Diastolic murmur is always pathologic

⇒ Hear the physiological splitting of S1

⇒ In ECG:
   1. Left axis deviation
   2. Atrial & ventricular premature beats

⇒ Chest x-ray: straightening of left heart border

⇒ Cardiomegaly is a pathological finding

⇒ Blood pressure: both SBP & DBP fall.
   but fall in SBP >> fall in DBP due to hormones Estrogen (vasodilator) & Progesterone
   (Resistance against vasopressors)

⇒

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Supine hypotension syndrome:

- Fall in BP in supine
  - Compression occurs
    - ↓ IVC venous return
    - ↓ cardiac output
    - ↓ hypotension

In left lateral position - uteroplacental circulation.

Renal System:

- All abdominal organs - endoderm.
- In renal system:
  - ↑ GFR - ↑ 50%
  - ↓ Sr. Creatinine
  - ↓ Sr. BUN

- Hydro ureter / Hydronephrosis - due to progesterone - cause smooth muscle relaxation

- Dilation of ureter more at R+ side. because dextrorotated (lt. ureter at the pelvic brim)

- Both kidney's enlarged.

Fetal Urinary System:

- Start producing urine 12 wks
  - at term - 650 ml/day
  - 27 ml/day hr
Fetal urine is hypotonic to fetal plasma.

**Respiratory System.**

- ↑ in Tidal volume
- ↑ in alveolar ventilation by 40%
- In pregnancy, no change in respiratory rate, there is only ↑ in depth of respiration due to progesterone which ↑ sensitivity of respiratory centre to CO₂.

- ↓ in CO₂ in pregnancy → ↓ (due to wash out of CO₂)

- Resp. alkalosis (mild)

- pH of blood ↓: No change.
- Serum HCO₃ levels ↓ in pregnancy due to compensatory mechanism by kidneys.

- Diaphragm rises ↑4cm.
- Tranverse thoracic diameter ↑2cm
- ↑ lung capacity → ↓
- Vital capacity no change

- There is a ↓ in expiratory Reserve volume (dead space)

- IRV - no change.
Fetal Respiratory System:

- Lungs derived from germ layer - endodermal in origin due to outpouching from foregut.
- Surfactant synthesis (occurs) begins at 20 wks.
- Surfactant appears in amniotic fluid = 28 wks as early as.
- Earliest Fetal Breathing movements on USG = 11 wks.
- Best test for Fetal lung maturity: phosphatidyl glycerol.
- MC Test used for Fetal lung maturity: Lecithin: Sphingo
  \[ \frac{2}{1} \to \] 8.

L/S ratio changes = contaminant where as
phosphatidyl glycerol doesn't change = contaminant.

Gross Body movements appears as early as 7 wks.

- Swallowing = 10 wks
- Urine = 12 wks
- Breathing movements = 11 wks
- Meconium = 16 wks
- On USG: Sucking = 24 wks
- Hearing = 24 wks
- Light perception = 28 wks.
Fetal Insulin appears at 10 wks.

Fetal glucagon $\to$ 8 wks

Fetal Thyroid Hormones $= 12$ weeks

LH/FSH $= 12$ weeks.

$\Rightarrow$ wt. of Baby at 30 wks $= 300$ gms.

$\Rightarrow$ For abortion wt. $= 500$ gms.

$\Rightarrow$ At term wt $= 3.4$ kg (2.8 kg as per India)

3rd stage of labour $:$ Delivery of placenta.

$\Rightarrow$ Avg. Time $: 15-20$ min.

$\Rightarrow$ Prolonged $: \text{of} > 30$ min.

$\Rightarrow$ Signs of placental separation $:$

1) Gush of blood
2) Suprapubic bulge.
3) Lengthening of Cord.
4) Fundal height $\uparrow$ (placenta goes down $\&$ pushes uterus up)

Fundal lie after delivery lie just below umbilicus.

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→ Placenta lying in vagina > lengthening of cord.
→ Mc method for placental separation.

Placental separations

Schultz (b)

→ Separation begins from centre

→ Retroploental clot:
  → External bleeding occurs
    after entire placenta has separated.
  → Fetal side (A) which +
    at vulva.

→ S. Smooth & shiny
  Schultz (b)

→ Total blood loss is less by:

→ Mc seen in 80% of deliveries.

→ Separation from periphery

→ No Retroploental clot
  → External bleeding occurs
    early in separation.
  → Maternal side (B) which
    present at vulva.

Dirty = Duncans

Total blood loss ↑
mc seen in 20% of deliveries.

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- Normal blood loss after vaginal delivery → 500 ml
  after CS → 1000 ml
  after twin vaginal delivery → 1000 ml
  In CS hysterectomy → 1500 ml

  ➔ PPH: Any blood loss which leads to fall in Hct by > 10%.

  ➔ Shock Index: \( \frac{\text{Heart rate}}{\text{Systolic BP}} \)  
  \( n = 0.5 - 0.7 \)
  → used in acute bleeding pt.
  if \( > 0.9 \) → Requires immediate resuscitation.

  ➔ Measure urine output:
  ➔ Catheterise patient:
  ➔ PPH: It is the leading cause of maternal mortality.

  AMTSL  
  WHO  
  (active mx of third stage of labour)

  FIGO - International Federation of Obstetric Gynecology
  • Modified WHO

  Components:
  1) Uterotonic agent at time of delivery.
  2) Ant. shoulders or immediately after delivery.
     (2 in 1 min)
1st Doc - Oxytocin

- Oxytocin release from
- Octapeptide / posterior pituitary
- Synthesized from hypothalamus.

→ 5 - 10 IU → Intramuscular bolus onset 2 - 3 min, act 2 last longer

→ 10 - 20 IU → Intravenous infusion onset 2 in 1 min

→ Intravenous bolus - not to be given → Hypotension
   - Tachycardia
   - Arrhythmia
   - MI

→ T1/2 of Oxytocin = 3 min

→ Max. no. of Oxytocin R's occur at 2nd stage of labour

→ Oxytocin stored at room temperature

2nd Doc - Methergin → 0.2 mg Intravenous → for prophylaxis

→ Brown colored ampoule due to photosensitivity

→ C/I in: 1. Pre-eclampsia / Eclampsia / Heart

- Problems / RH-ve pregnancy

→ 3. After delivery of 1st twin in twin pregnancy
S/E: It cause Transient Severe HTN.

3rd: Syntometrine → most potent

50 U oxytocin + 0.5 mg methergin

→ not doc due to high cost & not available.

4th: Plasprostol: PGE, analogue.

In prophylaxis: 600 - 800 μg in Per rectally.

→ Not given in IM or IV drug because it available in Tab form.

→ Give in asthma (bronchodilation)

8/6: Hyperpyrexia: α dose

Shivering.

2nd Component: Delivery of placenta by Controlled Cord traction k/LA: modified Brand Andrews method (otherwise uterus eversion occurs)

3rd Component: delayed Cord Clamping:

> 1 min - 3 min

How much extra blood goes to baby if we clamp blood

at 1 min → 80 ml

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4th component - uterine massage.

PPH

1°

Post PPH occurs 2-3 in 24hrs of delivery

mcc - atonic uterus

General causes of PPH:

4Ts:
- Tone (atonic bladder)
- Trauma to genital tract
- Tissue (retained placental bits)
- Thrombosis (defect in coagulation mechanisms)

R° of PPH:

\[
\text{PPH} \xrightarrow{m_x} \text{specific } m_x \\
\text{Non-specific } m_x \downarrow \\
\text{Bimanual uterine massage + call for help.}
\]

\[\text{Uterotonia} \text{ doc oxytocin given 30-40 IU/IV infusion.}\]

\[\rightarrow \text{methergin 0.2mg IM, IV}\]

\[\rightarrow \text{misoprostol } 1000 \text{mg P/R}\]

\[\rightarrow \text{Carboprost } \text{methyl} PGE_2 \text{ analagous.}\]

- 0.25 mg max.
- always give IM.
carboprost:
max. no. of doses in 24 hrs: 8 doses at 1/2 hr intervals.
mc: diarrhea, abdominal pain, nausea vomiting.

Brisk  PPH:
\[ \text{look for completeness} \]
\[ \text{of placenta (maternal side)} \]
uterus shows → intermittent atonicity seen

\[ \text{look for genital tract trauma} \]
\[ \text{uterus} \rightarrow \text{tonically contracted} \]

\[ \text{atomic PPH} \]
\[ \text{(medical) Balloon Tamponade} \]
\[ \text{UAE (uterine artery Embolization)} \]
\[ \text{Surgical methods} \]
\[ \text{uterine compression sutures (8-lynch sutures)} \]
UAE → done in hemodynamically stable person.
→ requires shifting of pt. k requires interventional radiology.
→ effective in PPH where cause is:
   AV malformation.

uterine compression sutures are: -
8-lynch sutures
Hayman's sutures
Cho square sutures.
B/ L uterine a. ligation

B/L ligation of ant. division of int. iliac
reduces the uterine blood flow by 80%. (Artery-synonym)

⇒ Effective in 50%.

⇒ Hysterectomy
C Supracervical Hysterectomy- Subtotal Hysterectomy

⇒ Remove uterus & leave cervix.

2. Genital Tract Trauma (Outside → Inside)

⇒ vulval hematomas /vaginal hematomas.

⇒ perineal tears.

Vulval hematomas: - caused by int. pudendal a.

Vaginal hematoma: - branches y uterine a.

Conservative mx: - monitor vitals
- Analgesics
- Ice compressors.

⇒ Surgically management Indications for:
(1) hemodynamic instability.
(2) ↑ in size
(3) of hematoma cause extreme pain.
Rx: I & D (given incision, remove clots & obliterate cavity)

of bleeder, mx by figure of '8' suture.

mc presentation of hematoma: → pain
& → inability to pass urine. (in post part patient)

on examination: blueish tender swelling

⇒ perineal tears:

degree

1st → only skin (all or) vaginal mucosa torn

2nd → along & vaginal mucosa, muscle layer also torn.

3rd → A → 30–50% of anal sphincter torn (EAS)

B → > 50% of EAS

C → EAS + IAS (Int. anal sphincter)

4th → whenever Rectal mucosa torn

3rd & 4th → medical emergencies because we have to
first repair that C in 24 hrs otherwise there
will be fecal contamination for month.

⇒ or OT (operation theatre)

⇒ ↓ anaesthesia (spinal, epidural)

Rectal mucosa:

↓
IAS

↓
EAS

Episiotomy.
mc method of repair - end to end anastomosis

Better overlapping technique
outcome

⇒ Episiotomy :-

median

- ↑ pain

- ↑ bleeding

- Cosmetically Poor
due to extension.

Cervix:-
⇒ mc site for cervical tear :- 3 o' clock >= 9 o' clock

- more on left side

Uterus ? - Trauma

Inversion

Rupture

Pt. goes to shock
immediately after delivery.
due to neurogenic shock
vasovagal response

Uterus become a Haeorrhagic
atonic shock

Cause of death - Haeorrhagic shock.
Inversion

- Reposition of uterus
- No uterotonics used until reposition of uterus occurs.

1. Reposition of uterus
   ↓
   Hydrostatic method
   'O’Sullivan’s'
   ↓
   OT (laparotomy)

After giving GA, try manual Then do laparotomy.

Q. A lady goes into shock after delivery most probable is:

- PPH

Q. A lady goes into shock immediately just after delivery is:

- Inversion

Q. A lady goes into unexplainable shock after delivery is:

- Amniotic Fluid Embolism (AFE)

Q. A lady dies due after delivery is:

- PPH

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Amniotic Fluid Embolism - Clinical vs' vs exclusion.
(AFE) - A form of defective thrombosis

1st phase:
- Sudden onset of breathlessness
  ↓
  Hypotension
  ↓
  Cardiac arrest
  ↓
  Coma → dies

2nd phase:
- Presents as Hemorrhage as DIC or Consumptive Coagulopathy

DIC

mcc of DIC in obs. is → abruption - mcc

→ Intrauterine dead baby (>4 wks)
  ↓
  Risk of DIC ↑

→ AFE

→ whenever there is an extensive hemorrhage

→ Sepsis

→ Uterine Rupture (Blood loss more)

Rx: - FFP & Cryoprecipitates

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Labour

False labour

True labour

Uterus contractions are going to

– Increased frequency,

2) Intensity

3) Duration of Time

– Show (expulsion of blood & cervix mucus plug)

– Dilation and effacement (cervix becomes thinner & attaches to uterus)

→ 1st gravida Effacement precedes dilatation

→ Multigravida E & D occurs side by side

→ Rupture of membranes (leaking PV)

Fully dilated Cervix = 10cm

Effacement expressed by %.

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Labour

1st stage: → From onset of labour pains to full dilatation

→ It is divided into

  Latent phase   → Active phase

Before ≥ 6 cm dilat → Any time ≥ 6 cm dilatation

Latent phase

Nulliparous

- Prolonged:
  ∘ 12 hrs
  ∘ 20 hrs

Multiparous:

- 8 hrs
  ∘ 14 hrs

Active stage:

a) $C_x$ dilatation rate

b) Rate of descent of head

→ $C_x$ dilatation rate:

Nulliparous: 1.2 cm/hr

Multiparous: 1.5 cm/hr

→ Rate of descent of head:

Nulliparous: 1 cm/hr

Multiparous: 2 cm/hr
3rd stage: From full dilatation to expulsion of fetus

Maternal pushing \( \rightarrow \) helps to expulsion of fetus.

Normal \hspace{1cm} Prolonged

Nulliparous 1 hr \hspace{1cm} 2 hrs \hspace{1cm} [In prolonged - Prog]

Multiparous 30 min \hspace{1cm} 1 hr \hspace{1cm} In Arrest: No prog

\( \Rightarrow \) Labour analgesia \( \downarrow \) prolongs the active phase of labour by 1 hr.

1. Prolongs 3rd stage of labour by 25 min

\( \Rightarrow \) Labour analgesia \( \downarrow \) risk of Caesarian sect

3rd stage: - expulsion of fetus to expulsion of placenta.

4th stage: - Observation period

It is up to 1 hr

- Chills occur: - Drugs \( \rightarrow \) misoprostol due to

1) blood loss

3) temp changes
lie: It is relation b/w the long axis of fetus & maternal spine.

longitudinal lie

Transverse lie

Oblique lie

⇒ Unstable lie is illiar to oblique lie

⇒ when lie is not fixed by 37 weeks it cause unstable lie.

⇒ goes to longitudinal
⇒ as Transverse

⇒ Placenta praevia

⇒ polyhydramnios also

⇒ oligohydramnios cause malpresentation

⇒ but not unstable lie.

Presentation: Part of fetus which is foremost in birth canal

cephalic

shoulder

arm
Cephalic $P_x$: changes in flexion & extension of head

when head is fully flexed - vertex $P_x$

fully extended - face $P_x$

partial extension $\rightarrow$ brow $P_x$ (midposition)

partial flexion $\rightarrow$ sinciput

Transcendent positions:

- Brow $\rightarrow$ Face

- Sinciput $\rightarrow$ Vertex

\[ \Rightarrow \text{Denominator: - Bony part which we feel during examination} \]

$V_x\; P_x \rightarrow$ Occiput

Face $P_x \rightarrow$ Mentum

Breech $P_x \rightarrow$ Sacrum

\[ \Rightarrow \text{Engagement: - Biparietal diameter of fetal head crosses pelvic inlet at birth} \]

\[ \rightarrow \text{Rule It Rules out Cephalo pelvic disproportion (cpp)} \]

\[ \text{Only at level of Inlet} \]

\[ \frac{\text{Engagement}}{} \]

\[ \Rightarrow \text{In 1° gravid, it happens by 37 wks} \]
Primenisitada come to OPD 37 wks POG head is free floating. This appears because

1) CPD
2) wrong dates
3) placenta praevia
4) a deep flexed head (mcc) of not engaged head in 37 wks of POG

In multigravida, engagement will happen at onset of labour.

Station: while doing PV examination,
at which level we feel head is station

Distance b/w ischial spines & pelvic inlet = 5 cm

'o' is level of ischial spines
of head above ischial spines 've
of head below ischial spines '+ve'

When head is engaged, station is 'o'
Ischial spines: - 1. Station is 'O'

2. Pudendal n. block level

3) Levator ani is inserted.

4) Curve of Carus

Forward part of curve of Carus is at

Engaging Pesary: at prolapse, we put this at level of ischial spines.

Engaging diameters: - Due to flexion & extension of head.

1. Transverse engaging diameter: - Bi-parietal Diameter = 9.8

2. Ant.-Post. engaging diameter:

At Fully flexed - At Vx P.: Suboccipito bregmatic = 9.5 cm

At Fully extended - At Face P.: Submento bregmatic = 9.5 cm.

Partial extension - At Brow P.: Mentoverical = 14 cm longest diam. of fetal head.

⇒ Deflexed head (not in full flexion): -

Suboccipito frontal

10.5 cm

Occipito Frontal

11 cm

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Positions: We divide maternal pelvis into 8 cardinal positions.

At full flexed: Occiput is denominator

At point A Left occipito ant. transverse

B LOA

A = MC position of fetus is LOT - 40% of babies

A = MC position of fetus at onset of labour/early labour → LOT

Late labour → LOA

A = What % of babies present in OP at onset of labour → 15-20% of babies.

⇒ Steps of normal labour:

1) Engagement

2) Descent

3) Flexion

Then after reaching level of ischial spine, internal rotation occurs:

LOT

Ischial spine
After Internal Rotation, Occiput lies under line of symphysis pubis.

IR happens by $2\frac{1}{8}^\text{th}$ of circle ← head rotates by $2\frac{1}{8}^\text{th}$

→ Shoulder rotates by $1\frac{1}{8}^\text{th}$

Crowning: when BPD stretches vulval diameters outlet & does not recede back after the contractions.

5) Extension

6) External Rotation (ER)

- Occiput will lie in original position (left)

After ER occiput will take original position i.e. along the left maternal thigh.

Lateral Flexion: delivery of body by Lat. flexion.

→ Normal labour means only by $V_x P_x$

other than $V_x$, it is abnormal labour.

1) Passage

2) Push

3) Passage

To bring labour as normal labour.
1) Passage: i.e. maternal pelvis.

   It has been divided by Caldwell & Malloy on basis of shape of inlet.

1) **Gynecoid** - 50% → mc ○ widest pelvis

2) **Anthropoid** - 25%

3) **Android** - 20% → narrowest pelvis

4) **Platypelloid** - 5%

**Gynecoid pelvis**

1. Cut sect by inlet of pelvis is circular ○

2) Sidewalls are parallel ○

3) Ischiadic spine blunt

**Android pelvis**

beaut shape ❤️

Convergent ❤️

sharp ❤️

Post dimensions:

Ant. dimensions:

Baby lie in post. dimension

mc of op position is Android pelvis ❤️ prominent ischial spinous

DTA ⊕ but not that much → Deep transverse cut - mc
1. **Naegleu pelvis**: when one ala of sacrum is congenitally absent.

2. **Robert's pelvis**: Both alae are absent.

   In Naegleu & Robert's pelvis, Cesarean section has to be done.

3. **Infradiate pelvis**: seen in Vit D deficiency, larger inlet, 0 in Ostandroasia.

4. **Rachitic pelvis**: Seen in Rickets.

5. **Contracted pelvis**: Just about pelvis, it is recurrent indication for CS.

6. **COPD**: Pelvis is inadequate for baby.

   → Non-recurrent indication of Cesarean
- Shortest the height (cm), higher risk of having contracted pelvis (Ht. < 140cm)

- Mc: is manual pelvis assessment to see contracted pelvis.

(a) Contracted Inlet: - AP diameter most imp.

1. Diagonal conjugate: → only measured clinically (under Sp to Sacral promontory)
2. Obstetric conjugate: → shortest AP diameter of inlet, not measure clinically (OC) → DC - 1.5cm.
3. True conjugate: - distance bew. upper border of PS → Sacral promontory (TC)

DC → 12 cm.
OC → 10.5 cm (shortest AP diameter, inlet) for OC > 10.5 cm
TC → 11 cm

- Contracted Inlet: - Shortest AP diameter is less than 10 cm.

Contracted pelvis: DC becomes → 10 + 1.5 = 11.5 cm.

- Contracted cavity: - IID (Interc ischial spine Diameters)

IIA is 10.5 cm

- Contracted < 8 cm.

If we able to touch 2 ischial spines, it is inadequate IID.
Outlet : Inter Tuberosous : Ischial Tuberosus site
distance.

\( \text{\#11 cm} \)

- if we put Fist (4 Fingers) in below Contracted < 8 cm. a ischial spine then it is adequate.

\( \Rightarrow \text{CPD : -} \)

Best to know CPD is 1. Trial of Labour. - only done for mild CPD at inlet

No outlet Trial of labour at outlet & cavity.

Best in CPD 1. Trial of Labour

\( \downarrow \)

2. MRI

\( \downarrow \)

3. manual assessment.

Push :-

\( \Rightarrow \) Uterine Contraction.

\( \Rightarrow \) Pacemaker of uterus is Cornu (i.e. Rt. Cornu > Lt. Cornu) hom

Contract? at Cornu @ 2 cm/sec speed

\( \downarrow \) Spread & depolarize the entire organ in 15 sec

\( \Rightarrow \) Contraction show fundal predominance.
Intrauterine pressure palpable is 10 mmHg.

IUP Painful - 15 mmHg.

If IUP required for cervix dilated = 15 mmHg

If IUP is 40 mmHg, fundus cannot be indented, it is moderate uterine contract.

Adequate uterine contraction:

1) which generated on IUP of 200 - 220 montovideo units

(no. of contractions in 10 min x press. generated by each contraction)

\[ 4 \times 3 \times 40 = 120 \text{ montovideo.} \]

- minimum press. required to push baby in 2nd stage of labour
- is 200 - 220 montovideo units.

2) 3 contractions in 10 min. each contraction extend to 45 sec.

3) Tachy systole > 5 contractions in 10 min.

when Tachy systole cause fetal distress it is called Hyperstimulation. (seen in Augmented labour < labour progress) by drugs etc.

Oxytocin was used for this.
Misoprostol not used for augmentation.

Uterine Press.

1st stage of labour - 40-50 mmHg.

2nd stage of labour → 80-100 mmHg.

Clinical labour: Begins at 80-120 montervideo units.

In 2nd stage of labour, much imp. to push baby is

↑ Intraabdominal press or maternal push.

b) Passenger:-

CPD

Non Vx P x

malpresentation.

⇒ malpresentation:

1a) Occipito posterior: - N presentation

(OP) → It is just malposition but not malP x

⇒ It is V x

Mcc → 1. Android pelvis.

2. Deflexed head

All mal P x in multiparous women except OP
At onset of labour -15-20% babies are in OP engage diameter: Suboccipito frontal or Occipito-frontal

How many are in OP towards the end of labour is 5%.
Most of OP change to anterior rotation occur in OA position.

In OP mc position is ROP (Occiput is in ROP)

1st outcome:

\[ \text{ROP to OA} \]
\[ \text{3/8 - head rotation} \]
\[ \text{2/8 - shoulder rotation} \]

Occiput rotates to right maternal thigh.

\[ n_x \text{ of OP is wait & watch.} \]

OP delivery > than OA delivery. Take time

2nd outcome:

- Transverse arrest.
- Only 1/8th rotation occurs & arrest.
- Seen in Android pelvis.

3rd outcome: If pelvis is adequate

\[ n_x \text{ of DTA is manual Rotation - we we do } C \text{'sect} \]

If pelvis inadequate do 'C' sect.
- Forceps rotation done in adequate pelvis. (Forceps used is Keilland's forceps)

3rd outcome:

→ direct OP/persistent OP

→ seen in [Anthropoidal pelvis.]

"Face To PUBES" position in direct OP.

Direct OP:

- Occiput towards sacrum
- Face towards pubis

Rx:

1. C Section

2. Forceps extraction from OP

(Forceps used in post position caused III & IV type Natum injuries so C section done. In ant. position forceps done)

Transverse lie: Shoulder Rx

Mcc: Prematurity

Mcc of Transverse lie in term pregnancy:

1) Placenta praevia.

when we do PV - we have "Gird iron feel" (Ribs all touching while doing PV.

Pelvis

Rx: - 'C' sectn

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when G2 P1 L1 female c 37 weeks q: Transverse lie position

Then m_x is External cephalic version (ECV)

**Prerequisites for ECV:**

1) POG > 36 weeks (of a ECV must be done before 36 wks
   Premature baby deliver occur)

2) liquor should be adequate. so
   Oligohydramnios is c/I but not polyhydramnios.

3) membranes should be intact.

4) ECV done in labour when membranes a is intact

5) if uterus contractare present so stop contract, so do

   ECV.

6) There should be no c/I for vaginal delivery

7) ECV always done under continuous fetal monitoring
   (USG/NSI)

→ ECV can cause placental abruption & cause
   Fetal distress.

   Of fetal distress occur, "C' secta has to be done.

   No ECV has to be done.
Obstructed labour / neglected shoulder $P_x$; most of baby die (IUD) occur. Then do 'C' section. Don't do vaginal delivery.

In old time, destructive procedures are used to take out baby through vaginal delivery.

Destructive procedures are:

1) Decapitation:
   - Hook separates head & body.

2) Evisceration - cut out abdomen, remove organs & deliver baby.

b) In a dead baby of transverse lie all following can be done except:
   a) Craniotomy - cut open vertex of head, only done for $P_x$.
   b) Decapitation
   c) C-section
   d) Evisceration

Face $P_x$ →
- [Mec of face $P_x$ is Anencephaly]
- [Type of pelvis - Platyphysoid]

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Risk factors for Face P:x:-

1) multiparity
2) prematurity
3) Cystic hygroma, Thyroid swelling so ant. neck mask
4) Cord loops around neck can cause face P:x
5) Hydrocephalus.

For malpresentation:

1) Abnormal pelvis → mc reason

a) multiparity
b) prematurity

Engaging diameters for Face P:x:-

1) Submentooccipital bregmatomic = 9.5 cm.

Delivery Flexion in Face P:x

mento ant. mento Post.

Vaginal delivery (VD) is possible VD not possible so 'c'-section.

Q → 3 cm diameter 50% effaced P/v mentoposterior.

In early labour - mento post. may change so

waist & GB watch.

Late labour - C- sect.

Tim dilated, labour is active, no rotat'd then C- sect.
Brow presentation: military position (partial extension)

Engaging diameter: mentovertical (14 cm - longest)

[Shortest diameter - of fetal head - bimastoid - 7.5 cm]

Rx in Brow Px: C-sect

In early labour: wait & watch
In late labour: C-sect

Breech Px:

Incidences of Breech Px at Term is 3.1%

MC of Breech Px: Prematurity (Transverse Lie)

Breech Px

Complete (Ficcoled)

Incomplete (Frank)

Footling

Highest risk of Cord prolapse

MC for multigravida

For 1st gravida

Least risk of cord prolapse i.e.

In general Vx Px - least cord prolapse
Complete (Fixed): -
- We feel P/v
  → Heel of the feet
  → Ischial Tuberosity
  → Anal opening.
  → External genitalia.

Frank:
- Heel of feet not palpable.
- Hip flex & knee extended.

Footling:

→ Absolute Indications of C'sec:
  Relative Indications of C'sec:
  Can do VD & C'sec but preferably C'sec is done.

1) First gravida.
2) Macrosomia.
3) Previous C'sec - C current breech (scar of uterus injured & death occurs).
4) Hydrocephalus.

→ 37 wks 1st gravida come to amn & breech Px → DO ECV if prerequisites met.

→ G2 P1 L1 38 wks breech Px we do ECV
   If ECV fails, then assisted breech vaginal delivery.
1) For extended arm. Rx: - Lovset's - (classical - another one)
2) For extended legs. Rx: - Pinard's
3) After coming head. Rx: Dr. Burns Marshall, 1. Mauriceau Smailic Viet. (m.b.)

In m.b. + m.b. Flexion and Shoulder Traction.

In Burns Marshall & m.b. alone dorso ant position after coming head (spine towards obstetrician).

By Dorso post. After coming head we do - 1. Prague's
2. Forceps - Pipers - Dorso ant.

By head stuck:
1. ZAVANELL: - Push back baby into vagina & do c'section
2. Symphysiotomy - Destructive delivery.

After coming head of preterm baby i.e. baby head stuck we do: - Cut the Cx - 2 positions i.e. Dubersson:

2 o'clock
10 o'clock.

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→ False labour pains: Rx Sedation
   → No cervical dilatation etc.

→ True labour:
  → Latent phase: Rx Sedation ≤ 6cm
  → Active phase: 1. (>6cm dilatation)
     1. Is the labour is slow
        Yes ↓
        Artificial rupture of membranes
        (Amniotomy)
        ↓ Slow
        Are the contractions adequate
        Inadequate ↓
        Rx: Oxytocin
        Augmentation
        Adequate ↓ slow labour due to
        1. CPD
        2. OP To rule out this, pelvic examination have to be done.

on Rule
1) Presence of Caput succedaneum [Old cephal hematoma]
   Scalp edema
2) Molding

Caput succedaneum
doesn't cross suture lines
1) At the time of delivery it improves in 2 days

Cephal hematoma
doesn’t cross suture lines (persistent)
Not at time of delivery
Appearance in first 3 days
molding:

Grade I: - Parietal bones just touch each other

Grade II: - Parietal bones overlap. They can manually separate.

Grade III: - Overlap cannot be separated.

⇒ CPD: - R x C'-section

⇒ It is a slow process.

⇒ From slow process

⇒ Arrest of labour.

⇒ Active phase of arrest: - No change in Cx dilatation even after 4 hrs of adequate uterine contractions.

⇒ Even after 6 hrs of inadequate uterine contractions.

 Rx of active phase arrest: - C' sect.

⇒ 2nd stage arrest: - Full dilatation (+)

⇒ No change in descent.

The criteria:

- 4 hrs multiparous
- 3 hrs multiparous
- Epidural analgesia.
2) Out epidural analgesia

3 hrs - nulliparas

2 hrs - multiparas.

Rx: 'C' - section.

In CPD:

① Arrest

② Obstruction

- Obstruction Labour

General physical examination

Per abdominal examination

- Tachycardia
- Tachypnoea
- Acidotic breath

Exhausted (dehydrated)

Tonsiloid contracted

Upper segment:

A stretched in inner segment

+ A groove + - Bland's Ring

(or) Contracted Ring

Obstruction may lead to FHR ↓ IUD

- Repeated filling of bladder
  Cause: Hematuria

P/v:

1. Capitulum

2. Hot dry vagina

4. Foul smelling Discharged
m_X:- C-sect X
- Destructive procedure - Craniotomy.

→ R_X 1. By C-sect X: 1) obstructed labour.
  2) neglected shoulder.

  Bandler Ring             Schroeder's Ring
  Pathological             Physiological
  Felt on Perabd. examin.  Felt on P/v examin.

Due to obstruct X → due to incoordinate uterine contract X.

Forceps                   Vacuum.
Instrumental vaginal
Delivery.

Aim:- To cut short the 2nd stage of
labour.

Forceps:- PREREQUISITES

  Fully dilated
  0 no. of obstruct X in path.
  Ruptured membranes
  C good uterine contract X
  E engaged head ("O" stage "O" station)
  P favourable P X

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Now we are using

1) Outlet Forceps:
   a. Wrigleys - old one (not used now)
   b. Simpsons - used for primigravidal women
   - molded head.
   c. Tucker Mc.Lane - multiparous
   - rounded head

Station

At \( \geq +3 \) - Outlet Forceps

\( +2 \) - Low Forceps

\( 0 \to +2 \) - Mid Cavity Forceps

Pre-requisites: - of Outlet Forceps:

1) head on perineum
2) scalp visible at introitus.
3) skull on pelvic floor.
4) sagittal suture of baby's head should be in AP's
diameter
5) In case it is short AP, i.e. \(<45^\circ\) rotation, we
   can apply forceps.

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Forceps

- Training should be used to ease to use.
- Forceps

- No maternal effort.
- Used in maternal exhaustion.
- Used in heart disease.
- Used in fetal distress.
- Used in face P.x.
- Afta forcing head of breech.

- Used for preterm baby.

- It causes more maternal Injuries.

Fetal Injuries incl. forcaps:

1) Intracranial Hemorrhage.
2) Brachial plexus injury.
3) Facial n. palsy.

- Forceps applied in the dimension is occipito mental diameter. - in OA N OP position.

Vacuum:

- Easier to use.
- Requires maternal effort.

A C/I in preterm.

It causes more fetal Injuries.
How we can know that forceps place applied correctly in OA:

1. Correctly applied blades are equidistant from sagittal suture & the blades are locked.
   - Cannot lock the blades = incorrect application.

In OP: Correctness:

- Are equidistant from line forming joining mentum & brow.

Forceps: more severe maternal injuries

- When applied to OP compared to OA.

Vacuum:- Plastic cups, Bell shaped

Applied at flexion point i.e. 3cm ant. to the
post. fontanelle.

6 cm post to ant. fontanelle.

Pressure generated $\rightarrow 0.8 \text{ kg/cm}^2$

$\rightarrow 600 \text{ mm Hg}$

If one instrument fails, go to 'C' section.

Failed vacuum: - When despite 3 pulls there is no descent.

$\rightarrow$ 3 pulls or 3 cuppop offs for vaginal delivery.
Instrumental deliveries are C/I in: - absolute C/I.

1) known coagulopathies in baby.
2) HIV
3) contracted pelvis / CPP
4) bone abnormalities → osteogenesis imperfecta.

**Antepartum Hemorrhage**

(APH)

It is defined as bleeding from or into the genital tract beyond the period of viability i.e. **34 wks**.

**Abortion**

Premature separation of a normally located placenta from decidua.

→ **mC** (i.e. 1 in 200 deliveries).

→ **Risk Factors**: Highest risk factors:
    - Previous H/o abruption 12%

Risk factors for both:
- cigarette smoking
- ↑ maternal age
- multiparity.

**Placenta praevia**

placenta located in lower uterine segment

Incidence 1 in 300

Highest risk:
- previous H/o placenta previa (5%)

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**Abruptio Placenta**

- Risk factors:
  - Strongly related to *pre-eclampsia*
  - Other causes:
    - Trauma (ECV/amniotomy)
    - Folic acid deficiency
    - Cause Abruptio Placenta
    - APLA syndrome → Risk factors
    - Poly & oligohydramnios → Risk factors
  - Fibroid uterus
    - Of placenta above & below
    - Fibroid cause premature separation of placenta.
    - Maternal blood loss

**Placenta Previa**

- *Types:*
  - **Type 1:**
    - Placenta in LUS, does not reach the internal os.
    - C 2 cm away from internal os.
  - **Type 2:** Marginal
    - Placenta reaches the margin of int. os.
Type III  Incomplete / partial - placenta partially covers internal os but moves away when os dilated.

Type IV  Complete / central - placenta completely covers os, don't move away after os dilatation.

Degrees of Placenta previa:

Minor Type:
Type 1 A KB - post uterine wall
Type 2 A - ant uterine wall
Can be delivered vaginally.

Major Type:
Type 2 B
3
4

Types of Abortion:
Revealed
Concealed
Mixed

Blood expelled out of uterus
Retroplacental clot

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PAGE's criteria:

Type 0 - examined for retroplacental bleed
  i.e. retrospectiveasis

Type 1 - Bleeding + Pain + FHR ○

Type 2 - Bleeding + Pain + FHR ↓ (± IUD)
  Fetal distress ⊕

Type 3 - Bleeding + Pain ± IUD + shock
  Mother is hemodynamically unstable
  ☑ Out of DIC.

DIC

Placenta separates from decidua which releases tissue
Thromboplastin into maternal circulation forming microthrombi
in vessels & cause (thrombi) DIC.

Causes: mec of DIC in Obs in Abruption.

Other causes:
1. IUD (>4 weeks) - ↑ risk of DIC
2. PPH
3. APH
4. of mother goes into Sepsis,
5. Amniotic fluid embolism (AFE)
36 wk POG e dead baby in utero Rx is: wait & watch.

The IUD baby expelled e in 4 wks by spontaneous labour.

\( \text{if} > 4 \text{ wks} \rightarrow \text{check coagulat}^{\text{io}} \text{ profile wait \& watch.} \)

\( \text{\fu} \rightarrow \text{How much maternal blood loss to call IUD is} \)

\( \geq 50 \% \text{ of maternal blood volume.} \)

\( \text{Abruption} \)

\( \text{PP.} \)

\( \rightarrow \text{Painful} \)

\( \text{painless} \)

\( \rightarrow \text{ax e P.E. (antece} \text{dent e} \text{vent e} \text{nt)} \)

\( \rightarrow \text{Bleeding per vagina (BPV}) \)

\( \rightarrow \text{altered blood loss} \)

\( \text{(dark color)} \)

\( \rightarrow \text{Tense \& tender} \)

\( \text{(Basal tone \uparrow)} \)

\( \rightarrow \text{Fundal Ht} > \text{POG} \)

\( \rightarrow \text{mal p also in this} \)

\( \rightarrow \text{fetal distress MC in this} \)

\( \text{Uterus Relaxed.} \)

\( \text{Fundal Ht} = \text{POG} \)

\( \text{mal p} \rightarrow \text{MC} \)

\( \text{seen in this.} \)
q PPH: - USG

1st we do Transabdominal scan

→ For pess. located PP is TVS > TAS

For IUD → TAS

To see: -

→ Distance of placenta from internal os.

→ Retroplacental area (to see clot or not in blue placenta & decidua)
  behind placenta.

→ Placenta is low lying. NO Retroplacental area in ANM

In USA is not good to see Abruption.

Clinically confirm by ARM we see blood stained liquor

in Abruption

⇒ AP Hemorrhage: - Terminating of pregnancy/ delivery

1) If Pt. is Haemodynamically unstable → Rx TOP

   next appropriate step is

   Blood Transfusion.

2) If Fetal distress: - Rx: delivery The baby.

3) Continuously bleeding PV: - TOP.

4) 37 weeks = TOP.

5) Dead baby in womb i.e. IUD = TOP.
6) Gross congenital anomaly (GCA) → Rx: TOP (least imp.)
   incompatible to life

⇒ what are indications of 'C'-section:
   1) Haemodynamically unstable mother.
   2) Fetal distress.
   3) of pregnancy < 32 weeks. We do C-sect because Cx not ripened, < 32 wks 80% don't respond to induction.

⇒ Preferred mode of delivery for abruptio placenta:
   1) Vaginal delivery.

→ Most of abruptio placenta people go to precipitate labour (onset of contractions to expulsion) within 3hrs.

→ 2. ARM:
   a. Blood-stained liquor
   b. Helps to induce labour.

   In Abruptio-ass, C-termination no conservative Rx
   1. Temporarily stops separation.

→ In PP:- we can do conservative management
   Indications of conservative mx:
   → NO haemodynamically unstable
   → NO FD fetal distress
   → NO BPV: bleeding per vagina
   → Live baby.

---

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Conservative m x is  **Maccoffe's Protocol**

1) Rest.
2) Maternal monitoring.
3) < 34 weeks → steroids.
   - Betamethasone: 12 mg I/m 2 doses 24 hrs (apart for lung maturity)
   - Dexamethasone given to mother when she has CAH

4) Uterus is mild contractions
   - Tocolytics given in pp but not in Abruption placenta

**Tocolytic Indications:**
- No tocolytic if > 34 wks POG
- To buy time for steroid cover.

**Tocolytics:**
- < 32 wks → Indomethacin
- > 32 → Nifedipin (safest tocolytic)

- Etoxiban
- Atosiban
- Estrasiban → oxytocin receptor antagonists better in heart diseases.

- Progesterone → is not a tocolytic agent.

In heart diseases, [Atosiban > Nifedipin]

  2. Hypokalemic.
- Diazoxide
- Halothane
- Alcohol

- Blood Transfusion (BT): - only symptomatic management.
  All are maccord protocol except Circlage
  A cervical Circlage not done in placenta previa.
  
  Pt. on conservative mx can deliver at 37 wks

  If mild injury → vaginal delivery.
  If severe injury → 'c'-section.

- Double set up examination: P/V in OT
  1) start doing P/V.
  Put finger up to os to feel placenta margins
  If you feel → major → shift pt & do 'c'-sect?
      placenta edge injury
  If you won't feel → minor → ARM - Induce labour.
      placenta edge

  Vasa Praevia
  - Fetal blood vessels + outside umbilical cord & run over
    internal os
  - It is an obstetric emergency do 'c' section to deliver baby
→ Torrrential blood loss of fetus

Adherent Placenta

Placenta not attached to decidua, placenta attach to myometrium

- Accreta
- Increta
- Percreta

Placenta adhere to, invades into, goes through & through myometrium & comes out on serosal surface

No intervening myometrial deciduosa

Histology: character by absent Nitauch's membrane, a layer of fibrinoid.

Adherent Placenta presentation:

Of undiagnosed, it reflects refractory post partum hemorrhage

Highest risk of adherent placenta presentation:

1. Lower segment C- sect° (in part)
2. In current presentation is PP

PP present >> C- sect° LV
More risk in current pregnancy

[Note: PP in previous pregnancy is not risk & for Adherent placenta]
Iy of choice for Adherent Placenta is

MRI > Doppler USG

\[ Rx: \text{elective C-section + Hysterectomy} \rightarrow \text{1st line Rx} \]

(planned)

If Pt. refuses for Hysterectomy, deliver the baby & leave the placenta in situ where placenta undergo autolytic digestion.

Pre eclampsia (P1H)

1) SBP > 140/90 on 2 occasions 4 hrs apart.
2) High BP should develop > 20 wks of POG.
3) No proteinuria.
4) BP will return to normal 2 in 12 wks of delivery.

Preeclampsia:

1) SBP > 140/90 on 2 occasions 4 hrs apart.
2) HTN should develop > 20 wks of POG.
3) 24h urine proteinuria.
4) Proteinuria > 300mg in 24 hr urine sample.

In most it corresponds to +
out proteinuria if any of following are met then also we consider preeclampsia

- PLT count < 1 lac
- Serum Creatinine > 1.1
- Liver enzymes are raised to twice than normal value. 
  [Alk. phosphatase ↑, ALT, AST ↓ in physiological]
- Prol. Edema.
- Cerebral or visual symptoms

All above show end organ failure or damage.

3. **K/A Eclampsia.**
   - All conditions of preeclampsia + seizures.

4. **K/A Chv. HTN**
   - When BP is high even at conception or even at high in the first 20 weeks.
   - BP remains high even 4 hrs after
   - 12 weeks of delivery

Chronic HTN + superimpose Preeclampsia

1. New onset proteinuria — previously no proteinuria she develop proteinuria after conception.
2) End organ damage

3) of proteinuria is preexisting
   a) worsening HTN (HTN not controlled) & new
   b) end organ damage occur

Risk factors for Preeclampsia:
- usually seen in nulliparous or primigravida
  - Chronic HTN
  - Chronic renal disease
  - DM
  - BMI (≥ 25)
    - APLA syndrome
    - molar pregnancy
    - twin pregnancy

Smoking is protective for preeclampsia: In these conditions, early onset of preeclampsia occurs.

Inverse relationship: blood pressure & preeclampsia.
- of pp occur, NO preeclampsia & vice versa, (preeclampsia is a risk for abruptio placentae)

Prediction of preeclampsia:
- By Doppler of uterine artery (normally notching disappears by 22 wks)
  we study here
  → In Preeclampsia → uterine a. notching

Persistence persists at ≥ 22 wks
Roll over test:

- Women lie in left lateral position, check BP.
  - Then roll over to her back to supine, check BP.
  - Maternal press up, mean.
  - Previously used as predictor, (not now).
  - Done b/w 28-32 weeks.

Urinary acid levels: Previously used as a predictor but not now.

- Urinary acid levels are higher in preeclampsia.

Prevention of preeclampsia:

a) Low dose aspirin daily from 12 weeks & continued throughout pregnancy & stop 7 days before delivery.

- Aspirin can also be given in high-risk cases.

Calcium supplementation:

- It has no role in supplementation.
- Has role in calcium deficient people for prevention.
- Routinely calcium supplementation is given for meeting RDA.
Pathophysiology of Preeclampsia:

There is absence of invasion by the extravillous cytotrophoblast into maternal spiral arterioles.

\[ \downarrow \]

Spiral arterioles undergo vasoconstriction

\[ \downarrow \]

Placenta undergo ischemia

\[ \downarrow \]

Ischemic placenta release severe inflammatory mediators

\[ \downarrow \text{cause} \]

Endothelial damage, injury (PTTs attach)

\[ \downarrow \]

Leaky capillaries

\[ \downarrow \]

Edema

\[ \downarrow \text{Intravascular volume: only cells } \oplus, \text{Hemodilution} \]

\[ \downarrow \text{Thrombus formation} \]

\[ \downarrow \text{End organ failure} \]

\[ \rightarrow \text{Placenta is culprit} \]

Specific Rx of preeclampsia is \( T_{op} \).
Preeclampsia

mild

Severe

- Sys BP > 160/110 mm Hg
- End organ injury
- Sr. Creatinine > 1.1

$\rightarrow$ Pulm. Edema
$\rightarrow$ Cerebral & Visual symptoms
$\rightarrow$ Liver enzymes $>$ than double.
$\rightarrow$ PLT count $<$ 1 lac.

\[ 1. Proteinuria > 5 gm/24 hr \rightarrow \text{Renal} \text{now} \]
2. IVQR
3. Oliguria.

Impending Eclampsia:
- Anytime develop seizures.

8/3x:
1) Epigastric pain, due to stretching of liver capsule
2) Nausea, vomiting, (N/V)

3) Headache & Dizziness cause of Central edema Hypoxia.

4) Development of visual symptoms
5) Blurring of vision, scotoma, diplopia, Blindness

1. Occipital lobe hypoxia cause Central Blindness which is

incurable after delivery
2. Retinal detachment cause blindness which is irreversible

\[\text{Rx} \rightarrow \text{specific Rx}\]

\[\text{non-specific}\]

→ HTN ⊕ \(-\text{Rx:- antihypertensives}\)

→ Seizures prophylaxis for seizures
   \[\text{ie mg soy}\]

→ Antihypertensives :

Given to

1) whenever \[\text{DAP is persistently } > 100 \text{mm Hg}\].

2) Case of Impending eclampsia.

\[\text{Doc :} \text{ Pre-eclampsia : labeceol} (\text{combine } \alpha \& \beta \text{ blocke})\]

9) For mild pre-eclampsia \[\rightarrow \text{NO HTN Therapy}\].

3) \[\text{Doc for CHN in pregnancy : } \alpha \text{-methyl dopa}\].

4) \[\text{Doc for acute HTN in pregnancy : } \text{i/v labeceol}\].

Others are: \[\text{i/v Hydralazine}\]

- Nifedipin orally. (\text{sublingual nifedipin cause IVD})

- \[\text{i/v infusion of NTG}\]

- \[\text{Na nitroprusside is reserved for refractory HTN in pregnancy}\].

\[\text{Na nitroprusside cause cyanide toxicity}\]
Diazoxide → not given in pregnancy

0) In Pregnancy
1) Diazoxide
2) ACE
3) β-blockers
4) Furosemide (diuretics) as hypotensive drug → cause IUD

In Congr. In mI + pregnancy pt.'s - Furosemide is given!

Target SBP: 140–150 mmHg
Target DBP: 90–100 mmHg

Termination at:
1. Gestational HTN
   well controlled
   EDD (40 wks)
   40 wks.
2. Preeclampsia (mild)
   37 weeks
Severe Preeclampsia
   34 wks.

Irrespective of POG / TOLB:
1. Impending eclampsia.
2. Eclampsia.
3. HELP
4. abruptio placentae / fetal distress.
5. uncontrolled BP.

Top: Preferred route is Vaginal Delivery.

C-sect → Obstetric indications for 'C' sect

Pregnancy < 32 wk - cx not respond to induct

Preferred anaesthesia for 'C' sect: Epidural / Combined

Paravertebral / Spinal

Epidural

HELPP Syndrome: 3rd Trimester

H - Haemolysis on peripheral smear + of schistocytes or HELLP all

E - Elevated liver enzymes → AST ↑↑ > 70, ser.Bilirubin ↑

L - Low PLT count < 1 lac

P - Pitting Edema

S/sx: epigastric pain

Nausea & vomiting

Others involve to pre-eclampsia.

HELPP Syndrome

dyo → AFLP

→ Acute Fatty Liver Pregnancy

→ more severe hypoglycaemia

→ Hepatalseal Syndrome

→ Coagulant profile, abo

→ Acute pancreatitis.
**TOP**

\[ R_x \text{ of HELLP} = \text{TOP + mgsa} \text{y (prophylactic)} \]

Recurrence rate of HELLP: 27%

Maternal mortality rate: 4%

D/P 1) *Hepatitis*:

- Bilirubin level >10
- Liver enzymes 1000 Times ↑
- Fever
- Hep “E”

3) *Obstetric Cholestasis*:

- 3rd trimester
- MC complaint: pruritis
- Cause: unconjugated bilirubin
- Serum bile acids ↑
- Estrogen hormone

**Eclampsia**

Pre-eclampsia + seizures (GTCS)

1. *Antepartum* → MC

2. *Intrapartum*

3. *Postpartum* → Highest mortality
Postpartum eclampsia: Occurs in 1st 48 hrs of delivery.

- Any seizure other than after 48 hrs of delivery
  - Is other than eclampsia should be considered.

- Cerebral hypoxia / edema cause of eclampsia.

- Mechanism of death in eclampsia due to Cerebral Hemorrhage

If pregnant lady throws fits:

1. Stabilize the patient's position.
2. Secure the airway.
3. (Doc. for eclampsia is MgSO4)

MgSO4 acts:
1. On NMDA Receptors in Brain (Centrally acts)
2. Cerebral vasodilatation

Pritchard's criteria:

Loading dose:
- 4 gm I/V MgSO4 given slow I/V
  - Over 10 min
  - 1 gm/min.

Check PR & HR when giving I/V MgSO4.

10 gm I/M (5 gm in each buttock I/M)

- Maintenance dose given 4 hrs.

Before giving maintenance we have to check:

1. Patellar Reflex
   - Normally seen in pre-eclampsia

2. Urine output (Oliguria due to toxicity)
   - \( \geq 30 \text{ ml/hr} \)

3. Rino-Lo To > 12 min.
5 gm of mgsou I/m given as maintenance dose
when above features satisfied.

Impending Eclampsia -
(prophylaxis)

Dose & Regimen are Same.

\[ Mg\text{ Therapeutic blood levels } = 4-7 \text{ meq} \]

9-12 meq - loss of patellar reflex
- Slurring of speech
- Feeling of warmth
- Respiratory distress
- Dizziness

Of all +, check levels of mg in blood

> 15 meq - Resp. arrest

> 30 meq - Cardiac arrest

At 10 meq - loss of patellar reflex is absent

Antidote \( \rightarrow \) 10 ml 10% of Ca gluconate i/v.

Specific Rx of eclampsia: TOP (irrespective of RGA)

Route: Vaginal delivery
(delivery should occur \( \leq \) in 24 hrs of 1st seizure)

Preferred anesthesia: Epidural or spinal anesthesia given
GA not preferred.
Diabetes in Pregnancy

- Pregnancy is a state of insulin resistance. 
- Hormone is HPLactogen. (HPL)
- Fasting glycosuria & pp.
- Hyperglycemia (post prand) 

Glycosuria: physiological (Renal Threshold Used)

Pre gestational

Gestational Diabetes (GDM)

Type 1, 2 pm

GDM > Type 1 > Type 2

⇒ GDM: - when high blood sugar levels are diagnosed 1st time in pregnancy.

Screening: universal screening of GDM.

2 step approach

1 step approach

ACOG

WHO

IA PDSG

1st Step is Screenponsible

Diagnostic Test

Approach Test

i.e. GCT (challengert)

in 24-28wks

(where we can see insulin resistance normally)

⇒ 3hr OGGT

(Orgal glucose Tolerance Test)

⇒ 72 hrs before Test, unrestricted diet is taken
GCT:
50 gm oral glucose given
↓
1hr
after 1hr Bld sugar > 140

The screening test:

Screening test for Diabetes is
GCT

ΔScHc Test:
F-FBS > 95

100 gm glucose load
Carpenter Cousten criteria:

1hr - 180
2hr - 155
3rd hr - 140

⇒ 2 value are deranged
to make ΔScHc GDM

⇒ Single test / step approach:

→ done in 24-28 weeks.
→ Both screening & ΔScHc Test.

→ better compliance

→ more sensitive than a step.

Test:
[2 hr OGTt] ← Best test for Diabetes.

F-FBS cut off is > 92

Glucose 75 gm WHO / IPA PDSG

1hr - 180
2hr - 153

Any value if deranged she is confirmed case of
GDM

NOTES FROM
JAIN STATIONERY
09654691327
Fetus high risk:

1) Gross congenital anomalies
   (GCA)
   
   only ass. to pregestational DM
   (not GDM)
   
   → Hyperglycemia is fetotoxic
   
   ↓
   
   Free radical injury.
   
   Mc GCA: Cardiac vascular > Neural Tube defects.
   
   in Diabetes.

   Most specific → Caudal Regression Syndrome (Sacral
   NTDs → agenesis)
   
   Among CVS: mc is VSD
   
   Mc specific → TGA
   
   Mc finding → HOCM (not a malformation
   It is physiological adaptation
   which is reversible after birth)

To know risk of anomaly:

For risk assessment → glycosalated HbA1C

HbA1C < 6.5 % no ↑ risk

6.5 - 8.5 % mildly ↑ risk 3%

> 9 % greatly ↑ risk (22%)

Notes from

JAIN STATIONERY
Gautam Nagar
09654691327
To know or pick up anomaly: USG has to be done.

Specifically → Level II USG - done at 18-20 wks
Targeted Anomaly scan.

2) Fetal echocardiography → 18-22 wks.

⇒ Level II USG - done on 1st trimester.

a) → Anencephaly is detected.

b) Earliest at → 10 wks picked up.

c) Confirmatory at 14 wks

Prevention:

1) Insulin Therapy: Tight glucose Therapy in 1st Trimester.

2) Folic acid

IFA - 100 mg Fe 0.5 mg FA folic Acid

Dose of folic acid required for NTD's control is

⇒ 400 × 0.4 mg Folic acid.

RDA:
RDA of FA in Pregnancy = 0.5 mg.

cose of FA prevention of recurrence of NTD's = 4 mg.

diabetic dose = 4 mg.

keto epilepsy in antiepileptics (prophylaxis) = 4 mg.
For Prophylaxis of NTD's → 0.4 mg FA is given
[1 month before to 3 months after conception]

0.4 mg FA – Prophylaxis of NTD.

Risk of NTD is reduced by 72%.

→ mc anomalies in general pregnant population:

CVS >> NTD's

→ macrosomia: wt > 4 kg

Single best USG parameter to know about

IUGR & macrosomia is Abdominal Circumference (AC)

macrosomia:

\[
\text{BPD + AC + FL = EBW} \quad \text{Hadlock's Formula}
\]

EBW > AC

Hyperglycaemia in mother goes to baby & cause hyperglycaemia Fetus

pancreas → ↑ Fetal Insulin → Macrosomia.

maternal Insulin does not cross placenta.

maternal TSH

Erythropoetin

Calcitonin, PTH

NOTES FROM
JAIN STATIONER
GAUTAM NASAR
09654691397
why macrosomia is problem?

- Fat deposited in shoulder's a cause shoulder dystocia.

- Shoulder dystocia is delay in delivery of shoulders by > 1 min after delivery of head.

- SD: It is recognised by TURTLE sign.

- There is a sudden recoil of head towards perineum after delivery.

management for shoulder dystocia:

1) Call for help.

2. Epsiotomy & empty the bladder.


- There is a sudden flexion & abduction of maternal thigh on maternal abdomen

- Suprapubic press: given by help.

- Fundal press is contra indicated.

- Mc Robert's maneuver is success in 90% cases.

- Injury to lat: cutaneous nerve of thigh.

- If Mc Robert's maneuver fails, then.
woods cork screw manouvre.

\[ \Rightarrow \text{clavicle occur} \]

\[ \downarrow \]

symphysiotomy

\[ \downarrow \]

Zavanelli method.

3) IUGR :- of pt. develop (rare) complications like diabetic vasculopathy or preeclampsia

\[ \downarrow \]

uteroplacental insufficiency

\[ \downarrow \]

IUGR.

4) Still Birth :- placenta at certain places undergo hydropic changes (because glucose in placenta absorb H2O)

so baby become macrosomia. & requirement of O2 is required. So hypoxia occurs. It cause IUO.

→ fetal monitoring begins 32 wks.

→ macrosomic babies \( \Rightarrow \) Risk is high.

→ male sex

5) Operative Interventions :-

- Vaginal deliveries.
- C-section.
6) Fetal lung maturity is delayed in diabetic pregnancy.

MC Test: L/S ratio

Best Test = phosphatidyl glycerol.

Newborn:

1) Hypoglycemia.
2) Hypocalcemia.
3) Hypomagnesemia.
4) Hyperbilirubinemia.
5) Polycythemia.
6) RDS (Respiratory Distress Syndrome).
7) HOCM.

Maternal risk of diabetes:

1) Uncontrolled diabetes will cause abortions.
2) Polyhydramnios → d/t polyuria, CSF leak.
3) Diabetes → vasculopathy
   Nephropathy
   Neuropathy.
4) Preeclampsia Risk - 15%.

5) Infections - UTI / candida.

6) Operative interventions - higher in diabetic pregnancy.

7) Psychiatric problems.
   Polyhydramnios >> oligohydramnios
   Oligo can happen less common - uteroplacental insufficiency
     - vasculopathy
     - preeclampsia.

Rx:

Doc in Pregnancy:

Doc in Pregestation / Diabetic - Insulin
   Regular / Lispro / Aspart?
   Long-acting NPH
   ↑ in pregnancy
   Dose ± per:
   1 - 12 weeks → 0.7 U/kg
   12 - 28 → 0.8 U/kg
   28 - 34 → 0.9 U/kg
   > 35 → 1 U/kg.

NOTES FROM
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GAUTAM NAGAR
09654691327
GDM: - Diabetic diet:

40% → Carbohydrates

40% → Fats – < 10% should Saturated FA

20% → Proteins

↓

POC - Insulin

OHA's are not Cl in pregnancy are oral hypoglycemic agents: glyburide / metformin.

Not compliant 2 Insulin or refuse to take.

Indication to start Insulin:–

Fasting level ≥ 95 mg/dl

2 hr pp ≥ 120 mg/dl

Target level:–

Target capillary glucose level:–

Fasting < 95

1 hr pp < 140

2 hr pp < 120

A1C = 100 mg/dl

HbA1C ≤ 6%


**Termination:**

GDM (diet controlled) → EDD is 40 wks.

[on Therapy (Insulin/OHA) → 39 wks for Termination]

Mode of delivery: diabetes is not indication to do 'C' sect.

**Indications for 'C' sect:**

- Fetal wt ≥ 4.5 kg in diabetic pregnancy
- ≥ 5 kg in non-diabetic pregnancy

⇒ Insulin requirements in labour is ↓ because pt. who are in labour are kept on liquid diet or they kept on NPO & oral fluids are given.

Insulin is stopped in labour:

↓ Intensive glucose monitoring

(Blood sugar every 2 hourly)

Molar Pregnancy

1) GTD's Gestational

- Trophoblastic Disease:
  a) Partial mole
  b) Complete mole
  c) Chorio Ca.
  d) Invasive mole or PSTT

GTNeoplasia:

⇒ Chorio Ca.
⇒ Invasive mole
⇒ PSTT (placental site trophoblastic tumor)
Partial mole

Triploid

extra set of chromosomes

is [paternal]

90% are triploid

10% are tetraploid

Complete mole

Diploid

all genetic material is paternal

monospermic

- single sperm
  - duplicate
  - empty ovum

→ 80% monospermic

20% diplospermic

→ trophoblastic proliferation less

→ extensive trophoblastic proliferation

on HPE:

- Some villi
  - Some bld vessels
  - Some fetal tissue

↓

which is congenitally malformed

on HPE:

- Trophoblastic scalloping

Both present & bleeding

PV

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Gautam Nagar
09654691327
HCG is more than expected but not markedly raised

HCG levels are very high

Fundal height < POG

Gold standard for a missed molar pregnancy is HPE

Complete moles: 1. Hyperemesis gravidarum
2. Hypertension
3. Pre-eclampsia

For high risk:
1. Emboli "wings" ARDS
2. Emboli present on X-ray

Full hydatidiform mole

Partial mole:

Partial hydatidiform mole

In some placenta

gSC is more bigger

Partial from diameter of U.S.A (missed abort)

Hematoma

Passage of grape-like vesicles

Ex of choice = USG
Partial

x

Partial mole
↓ 3-5%

GtN

Partial mole
↓ negligible <1%

Chorio Ca

(Theca lutein cysts +
(ovaries B/L)

→ complete mole
↓ 15-20% risk

GtN

→ complete mole
↓ 4% risk

Chorio Ca

Rx: Suction & Evacuation for molar pregnancy.

Family completed size 3 mole → Hysterectomy.

Monitor: weekly HCG
↓ x for 6 months after S&E

monthly HCG

Period of surveillance after evacuation of molar pregnancy is 6 mon.

Contraception of choice - Combined OC pills.

Avg time HCG to return → 9 weeks.

Complete mole = 9 wks

Partial mole = 7 wks.
Risk Fx's → GTN

1) age > 40 yrs

2) HCG > 10^5

3) Large uterine size

4) BJL Large theca lutein cyst (6cm)

   Spontaneously regress 2 in 1-4 mon after evacuation

→ Criteria of GTN

5) when 4 consecutive HCG values are plateau (± 10% of previous values) \( D_1 \ D_7 \ D_{14} \ D_{21} \)

2) 3 consecutive HCG values that are raising (> 10% of previous value) \( D_1 \ D_7 \ D_{14} \)

3) On HPE → Chorio Ca.

4) if HCG above normal even after 6 mon of evacuation

   Even 1 among above criteria met i.e. GTN.

→ Presents 2

1) Continuous BP v. even after evacuation

2) Uterus show subinvolution.
3) Shock (Invasive molereat all wall of uterus in 1 day
and present 2 shock)

4) Persistent B/L Theca Lutein Cyst

5) Present 2 metastasis

Lungs >> vagina >> pelvis >> liver >> brain

Choriocarcinoma:

⇒ Characteristic appearance of lung metastasis go

Chest X-ray is

a) Canon ball appearance.

b) Snow storm appearance.

Choriocar:

- MC seen seen after which type of pregnancy - mole

Complete mole:

- MC after which type of non mole pregnancy - full term delivery.

Vaginal metastasis:

- Bluish sub urethral nodule due to high vascularisation.

GTV staging

Stage 1: Tx confined to uterus

Stage 2: Tx outside uterus but 2 in pelvis

Stage 3: Lung metastasis

Stage 4: Distant metastasis at any other site
MC Chemosensitive: Choriocarcinoma

Chemosensitive

Modified WHO Score

- Score > 7 - High risk
- Score < 6 - Low risk

1. Age ≤ 39
2. HCG < 10^3
3. Type of molar pregnancy
   - Full-term delivery
   - Low risk → good prognosis
   - High risk → bad prognosis
4. Duration < 6 months
   - High risk → bad prognosis
5. Size < 3 cm
6. Metastasis
   - Lungs
   - Liver, Brain
7. Multiagent chemotherapy
   - After chemotherapy recurrence occurs

Low Risk
- Single agent chemotherapy
  - Methotrexate (MTX)
  - Actinomycin D

High Risk
- Multiagent chemotherapy
  - EMA-CO
  - Etopside
  - Vancomycin
  - Cyclophosphamide

Resistance
- Methotrexate
- Actinomycin D
- Etopside
- Cisplatin
Low risk
weekly HCG - N
after value N we have to give
3 cycles chemotherapy
if HCG level N then - monthly HCG has to be done.

Surveillance:
Low Risk = 12 mon
High Risk = 24 mon

Contraceptives: Combined OCP’s

⇒ PSTT: - 1. Chemoresistant Tumor
2. Tx: hysterecasy
3. HCG not raised
4. PLAP: placental Alk. Phosphatase - marker

Ectopic Pregnancy (EP)
Any pregnancy that is implanted outside uterine cavity

⇒ Cornual pregnancy: - It is an ectopic pregnancy
It is implanted in the interstitial part of intramural Fallopian Tube
The growth of pregnancy is lateral to round lig.
Angular pregnancy: Implanted near the opening of Fallopian tube. It is an intrauterine pregnancy. It is close to the cornu.

D/O of A.P is C.P

⇒ The growth of pregnancy lies medial to the round lig.

Heterotopic pregnancy: IUP pregnancy coexist with extrauterine.

⇒ Highest risk of ectopic:

1. Previous 1/3 of ectopic preg.

2. Any kind of tubal surgery.

3. MC Risk Fx is Pelvic inflammatory disease (PID).

4. Contraception: All contraceptives reduce the absolute risk of ectopic preg.

Least risk is C: Combined OCP's.

⇒ Sterilization: High risk if it is a form of tubal surgery of it fails.

IUP

Progesteron only pills

JAIN STATIONERY
GAUTAM NAGAR
0065109122
Sterilization > IUD > Pop

↓

Progestergen > mirena > CuT

which has highest risk of ectopic

1. Sterilization
2. IUD
3. Pop

1. Sterilization - 2nd
2. mirena (progesteron + IUD) - 1st
3. CuT
4. Pop (progesteron ↓ Tubal motility)

5) Cervicitis

6) multiple sexual partners

7) ART (Assisted Reproductive Technique → IVF)

8) Smoking

9) previous H/o Ca

10) Infertility:

Mc site of ectopic preg is Fallopian Tube (Ampulla)

Least common is FT → Interstitial/Intramural

Least common site: Cervical ectopic / ectopic in a "C" seen scar.

→ Ampulla is mc due to 1 site of fertilization occurs at ampula

≈ most no. of plicae
mc outcome of Ampulla ectopic preg. is Tubal abortion

2nd common → Tubal Rupture

Avg. Time at which Ampulla Rupture: 8 wks

Isthmic Rupture: 6 wks of amenorrhea.

Intramural / Interstitial Rupture: 19 wks because uterine musculature supporting it. So preg. is supported

→ Intramural rupture has highest mortality rate (because uterus also rupture in this)

Ectopic Preg. C/F:

Triad: Pain

Amenorrhea

Bleeding

only in 15% of patients.

most consistent s/s x 2 ectopic: Pain > Amenorrhea > Bleed

ectopic → Urnerupted preg. cause pain due to stretching of nerves ie T11, T12, L1

→ Rupture of ectopic preg. cause pain due to peritonitis

on P/Abdomen: 1. Tenderness in lower abd.

2. Rigidity / guarding.

NOTES FROM
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GAUTAM NAGAR
09654691327
N/v: Syncope attack

Vomitting

Shoulder tip pain (due to irritation of phrenic n.)

P/vaginal: 1. Uterus enlarged

Cervical motion tenderness: when examiner move cervix (cm)

that cause pain.

CMT tve 1. Due to inflammation of adnexa—in PID

(salpingitis/operitis)

& PID

3. Adnexal tenderness

4. Adnexal mass (50%.)

5.

⇒ Women is hemodynamically stable, H/o suggests ectopic

She has Triad:

Then 1st do UPT Test (99% of cases it is tve)

T:

IoC: 1. Transvaginal USG. (done ≥ empty bladder

ECTOPIC

TVS:

1. 1st we have to see uterus

1st Finding that raises the suspicion of ectopic is

empty uterine cavity

2. Now look for outside of uterus.

2nd Finding which raises suspicion of ectopic is

Complex Adnexal mass (suspicion goes higher)
3rd: Then do Doppler USG (high vascularity + around mass)

"corpus luteum also show ring of fire sign". If we remove corpus luteum (8-10 wks) the pregnancy is abortive.

4th: We see GSac in extraterine position which also has cardiac activity. Then there is confirmatory to suspect ectopic preg.

Fluid in pouch of Douglas

- Seen in mid cycle, periovulatory time
- PID also
- After tubular abortion
- Also ass $\geq$ rupture of ectopic preg.

$\rightarrow$ minimum fluid in POP - does not significant for rupture

Fluid $\geq 100$ is significant

- Empty GSac is a blighted ovum
  \[\text{NO Yolk Sac $\&$ fetal pole} \]

- NO Intrauterine GSac seen; NO extraterine GSac seen

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GAUTAM NAGAR
09654691327
Adnexa

Minimal fluid in POD

Pt. correlate clinically.

Then we correlate now.

1. Baseline Serum HCG:

   - > 1500 IU
   - < 1500 IU

   Provisional Δs is:

   - Ectopic
     - Ectopic + VIable Intrauterinepreg. (HR +)

   - Non viable IUPreg. (HR absent)

Repeat β hCG in 48 hrs + doubling rate

- β hCG ↑ by 66% of previous value.

  1. β hCG ↑ & it doubles
     - VIable IUPreg

  2. β hCG ↑ & it doesn't double (Klo plateaus)
     - Non viable ectopic preg.

→ TVS + Sr. HCG is best to Δsis for Ectopic

→ TVS has higher sensitivity & specificity.

→ tells locatn of G sac
3) Gold standard for diagnosis of ectopic preg.: Laparoscopic.

Other Ix:

4. Sr. progesterone level $> 25$ ng/mL indicates IU preg.
   $< 25$ ng/mL indicates Ectopic preg. (Nonviable preg.)

5. Caudocentesis: Test used to capture the ruptured blood in Ectopic preg.
   → Not using now.

6. Colpotomy: Opening up POD
   → Low role in diagnosis of Ectopic preg.
   → Used to Rx for pelvic abscess
   → HSG → no role in diagnosis of ectopic pregnancy. Test is C/I HSG.
   Hysteroscopy not used for ectopic diagnosis.

Rx of ectopic preg:
   $m_x$
   Medical $\downarrow$
   Expectorant $\rightarrow$ Surgical

NOTES FROM
JAIN STATIONERY
GAUTAM NAGAR
09654691387
Medical Management

Prerequisites for medical m_x:

Hemodynamically stable.

To do medical m_x:

a) HCG < 5000 IU

b) Sac size < 3.5 cm (4 or cardiac activity)
   < 4 cm (absence of cardiac activity)

c) Preferably absent cardiac activity (relative requirement)

d) Hemodynamically stable.

Drugs we use are:

1. Methotrexate 50 mg/m² I/m injected on D1

   on D4 → sent β hCG.

   D7 → sent β hCG.

→ Successful medical m_x occur:

   when D7 value should fall ≥ 15% from D4 value

   if fall < 10%, then repeat inj methotrexate

   → βhCG sent on D4 & D7

   → we can give medical management up to 3 times m_x.

Failed medical management 3 times of methotrexate can be given.
Expectant Mx:

1) Sac size < 3cm
   Cardiac activity should be absent.

2) HCG < 200 IU & a Falling Trend

3) Monitor vitals 48 hrs till HCG become 0, i.e. < 5

Surgical Mx:

- If pt is hemodynamically unstable.
  - Next step is Blood Transfusion.

Ruptured ectopic

- Failed medical Mx
- C/I to give methotrexate (active liver & kidney problems)
- Future child bearing is not desired [of damage tube & we have to remove]

- C/O Infertility want to go for IVF (ART)

Principles for Surgical Mx:

1. Laparoscopy is always preferred over laparotomy except in unstable vitals [of unstable vitals & unstable]

   In ruptured ectopic & unstable: Laparoscopy
2) **Salpingostomy** is preferred over **Salpingotomy**.

Remove contents by hydro dissect

By hydro dissect

Gastrostomy (not sutured ends)

3) **Salpingectomy**

**Partial**

Future preg is desired

→ Family is complete

**Total**

Salpingectomy indications:
1. IVF
2. If sac size bigger than 5cm
3. If hemostasis can't be achieved

Salpingostomy indications:
1. Severely injured FT
2. Ruptured ectopic
3. Family is complete

---

**Notes from Jain Stationery**

Gautam Nagar
09654691327
Cervical Ectopic Preg.

→ Painless BPV

→ Rubins criteria

→ Palmitic criteria

→ Preferred is medical m_x [of vitals stable].
  
  Vital signs of unstable → surgical m_x

  **ABDOMINAL ECTOPIC**

→ Abd. bleeding not +, only pain +

Criteria: Staddford

A

m_x: Surgical

Ovarian Ectopic

→ Spigelberg classification

→ m_x: Surgical

Heterotopic

→ Very rare preg (1/20,000)

→ Highest risk of ectopic is ART (IVF) i.e. 1/100

→ m_x: Always surgically managed

→ Inj. KCl (Fetotoxic) into ectopic sac under USG or laparoscopic guidance
Repeat Elective 'C' sect

Indications:
1. 0.5% of Classical 'C' Sectn class scar
   scar in upper seg. of uterus [4-9%]
   Risk:
   Prev. LSCS = 0.2 - 1.5%,
   Low vertical = 1 - 7%.

Repeat Cesarean is posted
at 37 wks

2. 1/10 Ruptured uterus → 'C' sec
   Recurrence rate of classical CS - 32%
   Lower sec → 6%

   Elective 'C' sectn is posted as
   Soon as lung maturity achieved
   for fetus at 34 wks.

3. > Prev: 2 LSCS → 'C' sect
   ACOG: If 2 prev. LSCS + 1 vaginal delivery
   She can be allowed for VBAC Trial.

4. 1/10 surgeries in which uterine
   cavity is opened i.e. myomectomy,
   Hysterectomy in which cavity is
   Opened.
Current Indications for classical 'C' sect:

1. Ca Cervix (absoluta Indicat)

2. Dense adhesions b/w bladder & LUS (otherwise bladder will be opened up)

3. Post mortem 'C' sect

4. Impacted shoulder (Transverse lie) & dorso-post.

5. Placenta Praevia - Ant. wall (if don't know let's)

6. VVF Repair

7. A very preterm C sect (because LV segment not formed)

Previous classical 'C' sect is not an indication to do Repeat classical 'C' sect

Repeat elective 'C' sect "Trial of VBAC"

Conditions in which vaginal delivery is c/I i.e. contracted pelvis, CPD PP etc.

Relative Indications:

1. Previous LSCS & breech in current preg.

2. Previous LSCS & macrosomia in current preg.

3. Post term & previous LSCS (>42wks)

[Polyhydramnios (b) twin preg. not indicate]
→ Presence of twin preg. doesn't ↑ risk of rupture of uterus

→ Impending Rupture / Dehiscence / Rupture:

s/sy of IR := most consistent s/sx of IR is changes in fetal HR

i.e. Fetal Bradycardia

what is First fetal heart rate is := Tachycardia.

→ IR is clinical ±s

2) s_x := maternal Tachycardia.

3) Scar Tenderness / pain in scar site

IR may ±may not present ± microscopic hematuria

m_x of IR := Emergency 'C' Section.

Dehiscence := when all layers of myometrium is rupture but

overlying serosa is intact [preg. intrauterine]

→ It is an Intraoperative finding.

→ Rupture := when all layers of myometrium including

serosa is ruptured. Baby is expelled into abd. cavity.

s/sx := 1 maternal Tachycardia.

maternal Hypotension

FHR ↓ (absent)
3. There is a loss of uterine contour (when there is rupture you can’t palpate wall)

4. There is a sudden loss of uterine contractions

5. Fresh BPV

6. Gross hematuria [Bladder rupture or venous plexus b/w bladder & uterus]

7. Loss of station [Due to this, baby expelled into abd. cavity]

Mix of Rupture uterus: 1. Emergency Laparotomy &
2. Suturing back uterus

General Principles in prev. CS sect

1) ECV can be done it is not C/I for prev. CS

2) IPV is C/I. (Internal Pudendal Version)

3) Labour analgesia is not C/I

4) IOL in prev. CS — is not C/I

Induction of labour:

Spontaneous labour is performed

Doc: Oxytocin

For induction

C/I is misoprostol

Used for induction in [Non scarred uterus] IV dose

Misoprostol 25 µg Intravaginally every 4 hourly till

onset of uterine contraction
→ Before induction, we see in non-scared uterus is

**Bishop Score** - 5 Components

- Cervical dilatation
- Effacement
- Position
- Consistency
- Station of head

Total Score = 13

- > 9 → Successful induction is possible
- ≤ 4 → Unfavourable Cx

To make Cx favourable, we give Cerviprime (Dinoprostone, which is PGE2 analogue)

→ Dinoprostone → 0.5 mg, put it in intracervically, 3 doses at 6-8 hr intervals.

→ Cerviprime gel is used in previous Caesarean sections only.

Single application of Cerviprime is allowed.

5) **Augmentation should be avoided in previous C-sect** (It is a risk of rupture). If we want to do augmentation, oxytocin is given but continuous fetal HR should be monitored.

6) **Ideal time for conception after prev. C sect** is

18 mon (max time)

Min. Time: 6 mon
we close uterus in 2 layers in c-section normally.

if we close in 1 layer which has weak integrity of scar
Guidelines: single layer closure doesn't ↑ risk of rupture
2nd layer of closure is for haemostasis.

P. sepsis: c prev. lscs is no indication to do repeat c-section.

Abortion

Preg. loss that occurs < 20 wks or < 500gm (200gm)

mc cause of 1st trimester abortions are Chromosomal anomaly

a) aneuploidy b) monosomy X c) trisomy 16
d) polyploidy

In aneuploidy → Trisomy's are mc

↓
Specific lesion (individual)

↓
Monosomy X - 20% of all

2nd lesion: mc is Trisomy 16 - 16% of all.
2) Congenital malformations in baby.

3) Trauma → Amniocentesis/CVS in 2nd trimester
   
4) Endocrinopathies → uncontrolled diabetes,
   
   ↓

   - uncontrolled Thyroid drugs
   - PCOS

   Uterine malformations are 2nd mc cause in 2nd trimester

5) Cigarette smoking

6) Alcohol

7) APLA Syndrome

8) Environmental

10) Infections: 1. TORCH → cause Isolated preg loss

   2. Streptococcus / Mycoplasma / Bacterial vaginosis

   Syphilis

⇒ Well controlled diabetes & Thyroidism doesn't cause abort

⇒ Luteal phase defect: less progesterone secretion occurs

⇒ Endometrial biopsy > 2 days

⇒ lag btw HPE dating & menstrual dating
Progesterone level < 15 ng/mL.

- Progesterone supplementation

Uterine malformations:

- Congenital anomalies
  - MC is bicornuate > septate
  - MC cong. anomaly associated w/ abortus = septate

  → Cause 2nd trimester abortions

- Acquired
  → MC → Cervical incompetence
  - Characterised by:
    - Painless dilatation of Cx
    - Expulsion of contents
  - It classically presents as recurrent 2nd trimester loss
  - In every close loss:
    - Gestational Age Keep
    - Sing (opposite True for syphilis who)
      - G + A +
  → Rx: Cervical stitch

- Cervical stitch

- Prophylactic
  1. H/o of recurrent
  2. Thin w/diag. which are painless
  3. Has not H/o recurrent
    - Loss but has short
    - Cx length i.e. < 25 cm.
Short CX seen in USG. (See shape of cx canal & internal os).

T → Y → V → U

→ Shape is indicator of cervical incompetence → U

→ Prophylactic: Circumcision is put b/w 12-14 wks.

Circumcision

[absolutely c/I circumcision:]

1) Ruptured membranes

2) Current infect

3) Gross congenital anomalies (Amenapathy)

[Mc Circumcision is McDonalds/Koh.]

Vaginal Indic. of McDonald's fails procedure we use this

→ Remove circumcise at ≥ 37 wks and/or

→ Rupture of membranes

→ Chorioamnionitis (Infect of fetal membranes)

Recurrent preg. loss > 3 ≥ Consecutive routine preg. losses except:

TORCH = Isolate loss.

Most imp is APLA = 15%

APLA → develop post thrombotic.
APLA

Clinical 1 + 2 lab criteria.

Clinical

1. arterial/venous
   superficial/deep
   Thrombosis

2. Preg > 3 losses at < 16 wks

3. > 1 preg loss > 16 wks
   of a morphologically abnormal fetus

4. At least 1 preterm delivery < 34 wks.

Lab criteria.

1. Presence of Lupus anticoagulant (LAC)

2. Presence of IgG & IgM Cardiolipin Abs in medium
to high titre on 2 occasions 12 wks apart.

3. Anti b2 microglobulin

Preterm delivery should be 2° to Severe preeclampsia or uteroplacental Insufficiency.

Rx:- LMW Heparin + Low dose Aspirin Therapy

→ Enoxaparin  \( \downarrow \)
   80mg OD

→ unfractionated Heparin also (Reversible) given Rapidly

→ Start Aspirin as soon as UPT test +ve

Heparin when intrauterine pregnancy is confirmed.

They have to be continued throughout preg.

Heparin is stopped at onset of labour.
Aspirin 5 stop at 5-10 days before labour.

→ Plasmapheresis is used in research purposes, & IV Ig in clinical Trials

→ Not to be used routinely.

→ These are use if 1° line agents fails

→ Can chromosomal anomalies cause Recurrent preg. loss (RPL)

Yes, the Balanced Robertsonian Translocat°.

Parents karyotyping is more imp. in RPL

Conceptus karyotyping is more imp in 1st Trimester loss.

RPL causes:

1) Endocrinopathies

2) Uterine malformations

3) Torch doesn't cause RPL

4) Mycoplasmal, Trp / Syphilis.

Abortions:

Spontaneous

Induced (by obstetrician)

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<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy</td>
<td>N/A</td>
</tr>
<tr>
<td>Threatened abortion</td>
<td>Pain &amp; IV</td>
</tr>
<tr>
<td>Incomplete abortion</td>
<td>Pain &amp; IV</td>
</tr>
<tr>
<td>Complete abortion</td>
<td>Pain &amp; IV</td>
</tr>
<tr>
<td>Missed abortion</td>
<td>Pain &amp; IV</td>
</tr>
<tr>
<td>Spontaneous abortion</td>
<td>Pain &amp; IV</td>
</tr>
</tbody>
</table>

- Rx: \( \rightarrow \) Induce Abortion
- USG: Absent
- Cardiac activity: Close
- + of
- Pain & IV
- Threatened abortion
- Incomplete abortion
- Complete abortion
- Missed abortion
- Spontaneous abortion

- Prostaglandin inj. to maintain prog.
- Bed rest
- AVOID COITUS
- Give weekly FSH inj.
- Uterus only & abort it.
Incomplete

Pain + BPv + H/o
expulsion of products
by uterus
ext.os is open &
products of conceptus (Poc)
are felt in cervical canal
→ Bleed profusely.

Complete

Pain + BPv + H/o expulsion
of products by uterus

→ ext.os closed

→ BPv reduces after
expulsion

Next step: BT + Suction & evacuation of Poc

→ USG: - No retained Poc

seen in uterine cavity

uterine cavity is empty

check HCG if it comes to 0

Induced Abortion

1st Trimester 2nd Trimester

According to MTP act, abortion allowed
upto 20 wks.
1st Trimester

up to 7 wks of POG as per act mp

→ Medical (outpatient medical abortion)

mifepristone: 200mg orally - sent pt to home

after 48 hrs

misoprostol → 800 µg/m p/vaginally

→ Up out patient medical abortion is safe up to 9 wks.

After 7 wks, up to 15 wks

the method is suction & evacuation

→ Press generated = 600 mm Hg

→ End point of suction:
  a. Reduced bleeding
  b. Air bubbles in canula
  c. A feeling of grasping canula by uterus

→ After S&E, check curettage has to be done i.e. deep curettage.

2nd Trimester

→ Abortion occurs > 15 wks

mcc induced abortion is

PG's i.e. misoprostol

→ Surgical D&E (Manual evacuation)
  Ovum Forceps → remove
  → MVA (manual vacuum aspiration)
  done up to 12 wks amenorrhea
  use 60 ml syringe
  press 60 mm of Hg

→ Menstrual regulation: done
  up to 3 wks of amenorrhea

NOTES FROM
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Dilator performs cautery knife

Suction > dilator

Perforation occurs when Hegar dilator passes through cervix.

For clinical incompetence:

Hegar's dilators are used to dilate.

1st Trimester:

Heart Disease:

Progressive dyspnea

Pathological signs

Psychological

4th grade
dyspnea

Cyanosis

Persistently distended neck

Diastolic murmur

Systolic murmur

2nd Trimester:

Orthopnea

Syncope attack

Chest pain

HAPE
Signs

- Pulm. arterial HTN
- Widely split S2

- MC acquired heart disease → MS
- MC congenital heart disease → ASD (in pregnancy)
- Which heart disease in pregnancy has highest mortality rate (>50%)

1) Marfan syndrome → aortic valve involvement
2) Coarctation of aorta, aortic valve involvement
3) Pulm. HTN → Eisenmenger's syndrome → indicates for vaginal delivery

Which common lesion is MC associated with maternal mortality in preg. is MS

Indications for Abortion:

1) MSynd. & AV involvement
2) CoA of AV involvement
3) Pulm. a. HTN →
4) At concept, high NYHA grade 3/4
5) HAS Ejection Fraction < 40%
6) Severe MS / AS

Value area < 1.5 cm²
1) propped up position
   O₂ by her side. in left lat. position. (LLP)

2) LLP

3) restrict I/v fluids @ 75 ml/hr.

4) restrict the no. of I/v examination
    induction of labour

5) IDL is safe & can be done.

6) ARM — can be done
   ↓
   c/I in PP | IUD dead baby
   In polyhydramnios — ARM is done
   In controlled way
   In magg. degree
   Take needle skin & Prick it

7) Vaginal delivery is preferred.
   we have to cut short 1st stage of labour
   for this we use forceps.

8) As soon as membranes ruptured prophylactic antibiotics
   we use is IV Gentamycin & Ampicillin I.V

9) methargin is C/I after delivery.

10) CHF immediate post partum period > 2nd stage labour
    28-32 wks
    Co give
    Rx — give Inj. Lasix is given immediately after delivery
2) Legs should hang down the delivery table to return venous return.

3) Mechanical heart valves are required.

Anticoagulant used for this:

6 - 12 wks - Heparin

12 - 36 wks - Warfarin → chondrodysplasia

36 wks to onset - Heparin. Punctata warfarin embryopathy if we give in 6-12 wks

Severe MS → Rx: Valvotomy (Balloon)

Valve replacement is not done in preg - very high mortality rate.

Valvotomy done at - 18 - 20 weeks

→ C-sect is reserved for obstetric indications.

→ Heart disease: Indications for C-section:

1) CoA & aortic valve

2) Marfan's & aortic root

3) Aortic dissect

4) Severe aortic stenosis.

→ Preferred anesthesia: Epidural > neuraxial > spinal anesthesia

C/O cyanotic heart disease & HbON
Twin Pregnancy

→ MC are dizygotic Twin

[In monozygotic Twins mc is monochorionic 2/3ds

  1/3ds Dichorionic]

After Fertilization (1st 72 hrs): Dichorionic & Diplanionic

4 – 8th day: MC & DA

8 – 12th day: MC & MA

> 12 days: Conjoined Twins | Siamese twins

→ Superfetation - 2nd conceptus during an ongoing preg.

→ Superfounding: Fertilization of 2 ova in same menstrual cycle by 2 different actions of coitus, seen in humans.

→ DC | MC: MC has high risk of complications.

Complications

1) ↑ Cong. malformations

2) ↑ TTS ( Twin Transfusion Synd)

3) ↑ acardiac twinning.
4) preterm labour
5) selective IUGR
6) Still birth rate.

=> DC (Indicators):
1) 2 Separate placenta.
2) Opposite sex twins associated always & DC
3) Twin peak sign or Lambda sign best visualized in 10 wks, i.e. 10 - 14 wks

Twin peak sign: Chorion entering divina dividing membrane
(can be seen up to 20 wks) [11 - 15 wks]

4) > 2 layers in the Intertwin membrane.
4 layers = 2 Amnion, 2 Chorion.

5) Thickness of Intertwin membrane (cut off is 2 mm)
If > 2 mm is DC

Twin Twin Transfusion Syndrome (TTTS):
mc in DC 1DA MCDA
Bama MCMA usually not seen in amniotic

Causes: - , deep A - deep vein
1st Twin = donor -> suffer's & oligo
2nd Twin = recipient -> Poly hydramnios
Suffered 2 CHF - hydrops & die

USG are one Twin - Poly & other Oligo.

Rx: Fetoscopic laser ablation of anastomosis

MA

\[ \text{monoamniotic} \quad \text{monoamniotic} \quad \text{MC complications} \]

\[ \text{Cord entanglement} \quad \text{Cord entanglement} \]

\[ \text{No blood supply to both twins} \quad \text{No blood supply to both twins} \]

\[ \text{They are delivered by plan 'C'. Such at 32 wks because} \quad \text{after 32 wks, cord entanglement?} \]

MC \rightarrow Conjoined Twins

[parapagus] (laterally fused)

Among parapagus, [thoracopagus] is MC

Thin dividing membrane

Thick dividing membrane

MC

DC

What determines outcome:

LIE of 1st Twin

1st Twin Longitudinal \rightarrow VD can happen

2nd: Vx \rightarrow VD
2nd Breed → VD

→ 1st Twin Breach

2nd Twin Vertex

\{ \text{Twin interlocking occur} \}

Refrer 'C sect'N

MA ← 'C sect'N had to be done.

\text{Twin}

→ 1st Twin is in Transverse lie. Rx: 'C sect'N

1st Twin had delivered

a) 2nd Twin is breach → Assisted vaginal breech delivery had to be done.

b) 2nd Twin Transverse → do IPV (Int. pudalic version) in a non scarred uterus.

2nd is Breech extract\(^2\) (under anestesia through VD

\underline{Preterm Labour}

\underline{ACOG:}

37 - 38\(^{+6}\) weeks called as early Term

39 - 40\(^{+6}\) - Term

41 - 41\(^{+6}\) wks - late term

> 42 wks - post term

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In preterm

4 contract in 20 min
8 contract in 80% 60 min -
which dilate 2cm & 80% effaced.

Based on gestational age. mx:

>34 wks — wait & watch

— urine culture
— a swab for Group B Strept

from vagina & Rectum.

Then look for Cervical length.

< 34 wks: Cervical length

>30mm 20-30mm <20mm

NO ↑ risk
kept for observation
for few hrs &
no active mx
→ assess fetal well being
& assess time of labour

↑ risk.

fetal fibronectin (FFN)
vaginal swab

↑FFN >50ng/ml

+ve active mx
← ve no mx

High Risk
active mx
1. **Betamethasone** for lung maturity 20mg

2. **Tocolytic** for 48 hrs
   - < 32 wks Indomethacin
   - > 32 weeks (safest)

3. MgSO4 (24–32 wks) → give MgSO4 for protection, which prevent cerebral palsy.

4. No role of antibiotics in preterm labour.

5. Progesterone has no role in acute preterm labour.

   Not a tocolytic agent.

   Only given for prophylaxis.

**Prophylaxis of preterm labour:**


2. Progesterone (prevent) of PTL

3. Cigarette smoking cessation

→ 32 wks, H/o 2 previous preterm delivery. Appropriate therapy is Progesterone.

32 wks H/o 2 previous preterm delivery; she is in threatened labour → Tocolytics & Steroids.
→ PPROM (preterm premature rupture of membranes) at
< 37 wks
get antibiotics (have definite role)

of > 34 wks PPROM → induction of labour.
< 34 wks → steroids, start tocolytic, antibiotics
mgsqy has to be given.

HIV in pregnancy

→ HIV is not Teratogenic

→ Universal

→ method for screening is: [OPT OUT] Screening
pt. didn't obey the doctor's advice
she don't do all the test that are advised by doctors.

→ HIV doesn't worsen in pregnancy.

→ CD4 count ↓ in pregnancy.

Perinatal Transfusion/Transmission:
(PNT)
PNT rate in the absence of breast feeding = 25%.
PNT rate in + of breast feeding = 35-40%

→ Even Anti-RT by how much PNT is reduced by 63%.

→ CART reduces transmission to <2%.
C section reduces transmission by 50%.

After 4 hrs of membrane rupture by how much does transmission ↑ every hr is 2%

Basic principles we follow in HIV pt. (preg)

→ All HIV +ve pt. should receive ART irrespective of their CD4 count. (CD4 ≤ 500)

CD4 count when to start

≤ 500 delay ART

as early as possible 14 wks 1st antenatal visit (not given in 3rd trimester)

→ Ideal Therapy is Triple drug Regimen

Tenofavir + Lamivudine + efavirenz

↓

WHD: Zidovudine Nevirapine

NACO: Triple drug combination

→ If viral load > 1000 copies → do 'C' section.

C-section is elective 'c' sect. at 38 wks

→ If viral load < 1000 copies → do VO
Instrumental VD is contraindicated

- Invasive fetal monitoring (fetal scalp electrode) is c/i.
- Labour should be induced (fasten process of labour otherwise infection occur)
- DOC for PPH ➔ Oxytocin.
- WHO: Late cord clamping has to be done.
- Baby has to be bathed immediately after delivery.
- Breast Feeding is to be done for a period of >6 months.

During breast feeding, child should receive nevirapine.

- To prevent PIM:
  - Potency: Tripple drug ➔ zidovudine ➔ nevirapine. ↑ single dose given by programme.

Fetal monitoring:
Done in antepartum

1) Fetal movement count:
   a) Count to 10 (lie in LLP for 2 hrs, count baby movements)
   b) 10 movements thr/s.
   c) 10 movements in 2 hrs, baby is not compromised.
   
   if < 10 ➔ risk

Cardiff 10 count ➔ 10 mov. X 12 hrs (outdated now)
2) NST
   a) Baseline FHR 120 - 160 bpm
   - Bradycardia < 110 bpm
   - Tachycardia > 160 bpm
   b) Beat to Beat variability B is 5 - 25 beats/min
   c) Accelerations ↑ in baseline FHR by 15 bpm & it lasts for 15 sec for babies > 32 wks
      ↓ if < 32 wks - ↑ FHR by 10 bpm x 10 sec.
   d) Deceleration:
      1. Early - k/a Type I → cause is head compress "physiological"
      2. Late - k/a Type II → cause is uteroplacental insuff (pathological (obstetric))
      3. Variable k/a Type III → cause is cord compression "physiological"

\[ \text{Early} \]

\[ \text{Late} \]
FHR dips at ht of contraction slowly falls to min. value & persists after contraction

\[ \text{Variable} \]
Fall to min. value is abrupt.

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Reactive NST: Done for 20 min.

Presence of 2 accelerations each of 15 bpm x 15 sec in a 20 min i.e. baby not compromised.

Non reactive NST: < 2 accelerations but time limit up to 40 mins (no late decelerations)

→ of non stress test shows late decelerations is pathological

& Delivery has to be done

1) We did NST → if becomes non reactive NST then NST is

2) BPP Biophysical profile / BP score manning score:
   ⇒ Binary i.e. rate +2 or 0 score.
   ⇒ done by USG max time is 30 min.

→ 5 components
   ① Fetal breathing movements +2 zero
      → at least 1 BM for 30 sec.
   ② Gross body movements → gross em +2 0
   ③ Body tone → see at least flexion, extension, & back folding
      → movement +2 0
   ④ Amniotic fluid → verify pocket
      → see 1 VP at least ≥ 2 cm
   ⑤ NST
      → reactive +2 0
2 Normal volume score is 8/10 & 10/10 → No comp. Fetal

A what score of BPS is borderline score?

A. 6/10 & 8/10 → ↓ AF (Component affected is Amniotic Fluid)

Rx: Repeat BPP in the same day.

If BPS score < 4/10 → Urgent delivery.

⇒ How should NST to be done in high risk pregnancy in

And twice a week (every 72 hrs) ← NST.

⇒ BPP → atleast weekly.

⇒ Doppler —– Chronic components → Amniotic Fluids

35 days to ↓ AF in hypoxia

Acute components → Breathing, movement → Tone (last to go)

In hypoxia

4) Doppler:


2. Uterine a. umbilical a.

3. MCA doppler.


1) Uterine a. notching persisting at 22-24 wks

Predictor of Preeclampsia.
fetal monitoring → IUGR
→ UP insufficiency.

2) MCA dopplex (Best) → indicator of fetal anemia.

- Middle Cerebral Artery
- Peak Systolic Velocity

Upf > 1.5 mom → Fetal anemia.

The waveform with sinusoidal pattern is best for fetal anemia.

→ mc Bid vessel to study UP insufficiency is Umbilical a.

→ S/D ratio only in preg as POG ↑, S/D ratio ↓

S/D ratio in ≥ 28-30 wks → IUGR / UP insufficiency

N → ≥ 3

\[ \text{Forward Flow} \]

\[ \text{In UP Insufficiency} \]

\[ \text{S/D ratio ↑'s} \]

\[ \text{Omnipresent Pathological} \]

\[ \text{A End Diastolic Flow} → \text{Indication of Top if} \]

\[ \text{GEA} > 34 \text{ wks} \]

\[ \text{Reverse EDF} → \text{Indication of Top irrespective} \]
In REDF, if we don't do TOP, baby will die in next 48 hrs.

2 in 1 week, AEDF change to REDF.

MCA doppler → does not show changes early;

Umbilical artery : S/D ratio ↑.

Best aid vessel to show changes is DV → Reversal → Terminal event

VAST : vibro acoustic stimula(?)

High frequency for 1 sec through artificial larynx

FHR ↑ by 15 bpm, baby is not compromised; it's a healthy baby.

Also used for intrapartum monitoring.

Intrapartum monitoring:

1. Continuous CTG (NST)

2. Intermittent auscultation (as good as continuous CTG)

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Intermittent auscultation  (1:1 monitoring)

Low risk  \[\rightarrow\]  High risk.

1st Stage  Listen to HR for every 30 min.

2nd Stage  HR for every 15 min.

3) Fetal scan  Blood pH :- (we want to look for acidosis)

\[7.25 - 7.35\]

7.0 - 7.25 \[\rightarrow \] borderline \[\rightarrow\] indication to repeat testing in 30 min.

< 7.20 - acidosis  \[\rightarrow\] indication for delivery immediately.

4) Fetal scalp stimulation  (non-invasive):

\[\uparrow\] FHR by 10 bpm : pH > 7.20 which rules out acidosis.

5) VAST

6) Fetal O₂ saturation < 30% for a period of 2 min or more

it means baby is compromised.

7) Fetal ECG, where absence of ST elevation means

Compromised baby.

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Anemia

→ mild anemia → Iron DA

→ check → Sr. Ferritin

→ anemia → Hb < 10 mg/dl.

→ severe anemia → Hb < 7 mg/dl.

Rx:
- Oral Tablets except if severe anemia > 36 wks indicate
- of BT (Bid Transfusion)

2. Irrespective of POG pt has S/Sx of CHF

Parenteral Iron:
- 1. non-compliant, 2. Oral

2. IBS (malabsorption)

3. Intolerable to oral iron

@ oral = rate @ 2 parenteral

0.1 gm/day → 0.7 gm/week

Fe 0.1 gm/day → ↑ Hb by 1 gm.

Total Iron preg = 1000 mg

1st day = 3-4 mg/day

2nd half = 6-7 mg/day

→ check complaint by asking color of stool

→ if not responding to Fe therapy → should check

Thalassemia → do electrophoresis.
Gynecology.

Estrogen → Progesterone

Sex steroids

Cholesterol → C_{18} compounds → Cholesterol → C_{21} compound

Carbohydrate

Types

E_1 → Estrone

→ Predominant in menopausal woman

E_2 → Estradiol

E_3 → Estriol

E_4 → Estetrol

E_1 = Estrone

Post menopausal women

Androstenedione → Estrone

Adipose tissue

E_1 → Estradiol predominant in Reproductive phase.

Source:

- In Early proliferative: indirectly Theca cells
- In late proliferative: Granulosa cells of Follicles

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2 Cell 2 Gonadotropin Theory

Acts by

FSH

LH

E2

Granulosa Cells

Theca cells (Stroma) (Theca interna)

Produce

Androgens (19c)

E3

Specific to pregnancy, produced by placenta (also produce E2)

Source: Fetal DHEAS

Corpus luteum produces -> Progesterone (mainly)

E2


⇒ Progesterone Sources:

1. Corpus luteum
2. Placenta (maternal LOL)

a) Progestins

1st generation → Nor ethindione.

2nd gen → Levonorgestrel

3rd gen → Desogestrel/gestodene/norgestimale

New 4th gen → Spironolactone

All progestins have androgenic S/E +

As generations ↑ androgenic S/E ↓ & their abnormal effect in lipid profile also ↓ so
3rd generation progestins have least androgenic.
4th gen. progestins has antiandrogenic.

**Estrogen (E)**
- Binds to Sex hormone Binding globulin (SHBG)
- 1% is Free

End products: glucuronides (sulphur-oxides)

Receptors: Intranuclear

→ Effect in:
  - a) uterus: Non pregnant
    - Thin, copious, watery, cellular
    - Spinberkel test: Stretch like a thread
    - Requires E + high levels of NaCl
  - Pregnant: Growth

→ Cervical mucus: Thin, copious, watery, cellular

→ Ferning disappears on Dig of menstrual cycle.

**Progesteron (P)**
- Cortisol binding globulin (CBG)
- Mainly Albumin
- 2% is Free

Pregnanediol

Intracytoplasmic

Secretory change in endometrium

→ Growth & also smooth m.
- Relaxation, used for prophylaxis of PTL (preterm labour)

Thick, scanty, highly cellular
- Viscosity due to highly cellularity
- No spinberkel test
- No ferning

→ Shows Crystallization (Pregnancy)

→ Ae shows ferning
  - Ae Mucus mixed E
  - Amn. Fluid
  - ie Amn. Fluid leaking

→ Type not permeable to sperms

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**Estrogen**

- Vagina: high karyopyknotic
- Superficial Cells (mature)

**Progesterone**

- Intermediate Cells
  - (Post menopausal women cells)
  - We see is Basal a Parabasal

**Cells**
- Basal: Intermediate: Superficial
- Preovulatory period: 0 : 30 : 70
- Post Parsuto: 0 : 100 : 0
- Post menopausal: 0 : 90 : 0

**Fallopian Tube**
- E ↑ motility
- ↓ secretions
- Caused by retent

**Salt & H₂O retention**

**Effect on**
- Cholesterol: ↑ HDL ↑ TG
- ↓ LDL
  - Cardioprotective

**Growth**
- Responsible for growth
- Cause closure of epiphysis

**Coagulation**
- Procoagulant
- Inhibit fibrinolysis

**Breast**
- Responsible for ductular development
- Upregulate the

**↓ mobility**

- ↑ secretions
- Cause excretition
- ↑ LDL
- ↓ HDL, ↓ TG

**No effect on growth**

**No effect on coagulation**

**Down regulates**
Progesteron acts on estrogen *primed* endometrium

\*Dub: Acute Blood loss \(\rightarrow\) Estrogen

\(\rightarrow\) Chronic Blood loss \(\rightarrow\) Progesterone

\(\circ\) is always have \(-\)ve feed back on FSH

\(\circ\) at low conc., \(-\)ve to LH (Inhibitory act° at LH)

\(\circ\) high conc., \(-\)ve feedback change to \(+\)ve feedback to LH

\(\circ\) at high conc. - has negative feedback on both LH & FSH

\(\circ\) at low conc. - has \(+\)ve feedback on both LH & FSH

\(\circ\) & \(\circ\): \(-\)ve to hypothalamus (GnRH)

on HPE:

- Proliferative effect in endometrium is seen under \(\circ\)

- Leucocytic infiltration in endometrium - Premenstrual phase

- 1st HPE evidence of Progesterone secretion is subvascular

  (Subnuclear vacoulation seen in D16)

\(\rightarrow\) Cork screw glands \(\rightarrow\) Late Secretory phase

\(\rightarrow\) Stromal edema \(\rightarrow\) Late Secretory phase

\(\rightarrow\) Telescoping / Endometrial gland \(\rightarrow\) Proliferation (\(\circ\))

\(\rightarrow\) Spiral arterioles \(\rightarrow\) show vasoconstrict° (Just before menstruation)
Testosterone

→ C19 steroid

→ major source of Testosterone in Females is
  50% - peripheral conversion of androstenedione.
  25% - adrenal gland
  25% - ovary.

In Ovary → which androgen is secreted in max is

Androstenedione > DHEA > Testosterone.

→ Produced in Theca interna (stroma)

→ Leydig cells → Testosterone Δ⁴ Pathway.

→ Sertoli cells → cause spermatogenesis.
  Bind + require spermatogenesis.
  Secrete MIS / Testosterone binding protein / estradiol / relaxin / inhibin

(1) is (1) in bound form i.e. 2 albumin / SHBG

1% free in females, 2% free in males

SHBG - high affinity (1)

SHBG (2) feedback (1)
(Synthesized in liver)

SHBG (2) feedback estrogen

→ low level of SHBG is marker for Insulin resistance.

→ In females: (1) e. responsible for Axillary / pubic hair
  (1) control libido.
End product: oxosteroids (Keto)

Receptor: intracytoplasmic.

→ Main hormone responsible for spermatogenesis.

\[
\begin{align*}
FSH & \mid \text{Testosterone} \mid LH \\
\downarrow \\
\text{Sertoli cells} & \quad \text{Leydig cells}
\end{align*}
\]

→ Blood Testis Barrier: + b/w Sertoli & Sertoli cells.

→ Pituitary: Secrete Gonadotropins (act on gonads which are Trope)

- FSH | LH

- produced from Ant. pituitary

- Pulsatile

- Gonadotropins

- FSH $t_{1/2} = 3-4$ hrs

- LH $t_{1/2} = 2$ min

- Proteins: membrane bound, G coupled proteins.

FSH has $\beta$'s on LH $\alpha$'s on

→ Granulosa cells

→ Theca cells

→ Sertoli cells

→ Granulosa cells (Cells surrounding ova)

→ G coupled coupled proteins

LH R's should be present in Corpus luteum to undergo luteinization.

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FSH main functions:

1. Select a cohort of follicles
2. FSH selects only one and allows for formation of a dominant follicle.

LH function:

1. Ovulation
2. Formation of corpus luteum.

\[ \text{LH} \rightarrow \text{Surge after } 36 \text{ hrs} \rightarrow \text{Ovulation occur} \]

\[ \text{peak after } 12 \text{ hrs} \rightarrow \text{Ovulation occur} \]

\[ \text{750g/mL} \]

What initiates LH surge:

- E present in high amounts → negative feedback changes to positive feedback
- E (Estradiol) initiates LH surge.

Estradiol 200pg for 48 hrs needed for LH surge.

LH surge is because of feedback of \( E + P \) for initiation

For maintenance the surge of LH

→ Which hormone is responsible for meiosis II resumption? LH

→ Meiosis II is resumed → 36 hrs before ovulation

→ LH surge cause luteinization of follicles

\[ \text{LH surge} \rightarrow \text{P synthesis begins before 36 hrs before ovulation} \]

(low levels)
→ Before ovulation :- \[ \uparrow \text{LH} \quad \& \quad \uparrow \text{FSH} \quad \text{(smaller)} \] due to \[ \text{P} \]

→ minimum levels of LH/FSH are seen in luteal phase of menstrual cycle.

→ which hormone is responsible for steroidogenesis of corpus luteum? LH (LH which forms & maintains corpus luteum)

\[ \Rightarrow \text{HCG} \rightarrow \text{corpus luteum dies} \]

→ which hormone rescues the corpus luteum from luteolysis? HCG.

\[ \Rightarrow \text{LH/FSH} \quad \xrightarrow{\text{?}} \quad \text{FSH} \]

\[ \text{GnRH Hypothalamus} \]

→ GnRH is released from arcuate nucleus of hypothalamus

\[ \Rightarrow \text{Decapeptide} \]

→ \[ \downarrow \text{Ty2} \rightarrow 3-4 \text{ min} \]

Neurons - which secrete - \[ \text{GnRH} \] are derived from olfactory placode

→ Neuronal migration is defective in Kallmann Syndrome.

\[ \downarrow \] (mutation in \text{KAL-1 gene})

\[ \downarrow \text{product is anosmin} \]

→ \[ \text{6 is absent} \]

→ lack of anosmin so they have Anosmia.

→ \[ \text{x-linked disorder} \]

\[ \text{NOTES FROM} \]

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Kallman syndrome:

- X-linked disorder
- NO GnRH → LH/FSH (NO)
- Hypogonadotropic hypogonadism

Release:

- At puberty: Pulsatile release of G

Before puberty: HPO axis is dormant.

Neurotransmitters:
- Responsible for pulsatile release of G
  - ↓ GABA
  - ↓ Neuropeptide
  - ↑ Glutamate
  - ↑ Kisspeptin

Pulsatile release is initially at nighttime.

First GnRH act on pulsatile release is LH.

Pulsatile release takes 3-7 yrs to mature HPO axis.

G act on pituitary

\[ \downarrow \text{Frequency of pulses} \quad \rightarrow \quad \text{release} \]

- FSH
- LH

Of frequency of pulses ↑ pituitary preferentially release LH

Of frequency ↓ pituitary preferentially releases FSH

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Luteal phase:

- Begins after LH surge

Causes:
- Release of ovulation
- Formation of Corpus Luteum (CL)

CL produces Progesterone.

- Max level at D22
- 8 days postovulation

Begin in late luteal phase when LH falls

LH falls → CL regresses → P level in serum ↓ (Begin Fall)

(FSH) (withdrawal)

(-ve feedback on GnRH)

GnRH small level ↑

Proliferative phase

FSH release by pituitary

Check FSH level

-ve Feed to check FSH level

Granulosa cells
0 o o cohort of follicles 0 o to grow

Secrets

Release low levels of

E2

Inhibin B

In late proliferation: only one allowed to grow, because it has max no. FSH R's

Dominal follicle (is selective by D8 of menstrual cycle)

Initiates LH

LH releases

(+ve feedback + E2 (high conc.)

astradiol)

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Corpus luteal:

- Life span of CL → 12-14 days.
- Max. activity of CL seen in 8 days post ovulation.
- CL produces secretes 1) Progesterone
  2) Estradiol
  3) Inhibin A

Source of Inhibin B → By granulosa cells of follicle.

↓

Inhibit FSH.

→ Activity → No fluctuation throughout menstrual cycle.

→ Antimullerian hormone → Source (Preantral follicles)

→ Shows no change throughout menstrual cycle.

20/4/16

1) Normal length of menstrual cycle = 21-35 days
   Avg = 28 days

2) Normal blood loss = 80 mL
   Avg = 55 mL (30 mL)

3) Normal no. of day that a woman bleed = 2-7 days
   Avg = 5 days
menorrhagia:

- Blood loss > 80mL
- Duration > 7 days

Hypomenorrhea

- Blood loss ≤ 20mL
- Duration ≤ 2 days

Polymenorrhea

- Cycle < 21 days

oligomenorrhea:

- Cycle > 35 days

→ metrorrhagia: any form of intermediate bleeding.

→ mittelschmerz: midcycle pain and spurring at time of ovulation

→ dysmenorrhea: no associated anatomical cause

→ Progesterone with withdrawal causes release of PGF₂α → ischemia in uterus → causes pain.

(vasoconstrictor)

2° dysmenorrhea: when an underlying pathology + fibroid, polyp

McC is [Endometriosis]

Ovulatory cycle: painful

An ovulatory cycle: painless

menstrual blood: ly arterial
Spiral arteries show vasoconstrict just before ovulation by PGF₂α.

Amenorrhea

1°

Def.: absence of menses by 15 yrs of age in the presence of 2° sexual characters

2°

Absence of menses for 6 consecutive months or 3 consecutive menstrual cycles in a previously menstruating female.

mcc is Pregnancy

mc pathological cause: PCOS

1° Amenorrhea

→ mcc of 1° Amenorrhea: Gonadal dysgenesis

2nd is Vaginal agenesis

Puberty: we can't estimate level of hormones

Female: Growth spurt (Skeletal growth > arm span)↓

Any Tanner stage > 2 → Thelarche - (Breast budding)↓

Pubarche (appearance of pubic hair)↓

Peak height velocity ↓

Menarche
Average age of menarche is 12.5 yrs.

In males:
1. ↑ in Testicular size
   ↓
   Penile growth
   ↓
   Pubarche
   ↓
   ht. velocity

→ Adv. Adrenarche precedes puberty by 2-3 yrs

Precocious puberty:
- In females most pubertal changes
  before 8 yr.
- In males < 9 yr.
  [For white race girls < 7 yrs
   Black girls < 6 yrs]

Precocious menarche: menstruation before age of 10 yrs

Amenorrhea

→

mca is Gonadal dygenesis.

→ Gonadal dygenesis:
1) Turner's - m/c.
2) Pure
3) Mixed
Turner's Syndrome:

- Karyotype is 45XO (one X is lost)
- The characteristic feature is short stature because loss of X
- Ovaries don't produce oestrogen

Short stature occurs due to loss of X gene responsible for growth of long bones.
- Gonadal dysgenesis
  - Streak gonads
    - Ovary - accelerated atresia (accelerated death of follicles occurs before fetus is born)
- Uterus: always small (hypoplastic) - 0 in intrauterine
- Breast: happen at puberty so breast tissue is absent, nipples are widely separated
- Axillary & pubic hair: adrenals glands +
  - Androgens so
  - Axillary & pubic hair + but sparse
- External genitalia: + in utero
  - External genitalia is female like
    - Sometimes it is hypoplastic
- Turner's characterised by:
  - 1) Low postnatal line
3) Webbed neck
4) Shield shaped chest
5) Short 4th metacarpal
6) CVS anomaly: MC is Bicuspid aortic valve, CoA
7) Renal anomaly: Horse shoe kidney
8) Autoimmune diseases: DM, Hashimoto's Thyroiditis

Life span of Turner's syndrome:

- Slightly reduced (due to CVS anomaly otherwise @)
- IQ level — @
- FSH — @ ↑ (No gonads → so no –ve feedback)
  - Gonads are fibrotic

Turner's is k/a Hypergonadotropic Hypogonadism.

- Can they become pregnant? — Yes by surrogate mother.
  - Pregnancy is C/I
  - In pregnancy, mortality is high due to aortic dissect@

in this Turner syndrome

- Pure Gonadal Dysgenesis
- Karyotype 46,XY/46,XX @
- Both are streak gonads
- Stature is @
- Pubic & axillary hair @

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46\text{xy} \rightarrow \text{Sryver's syndrome. (Defect lies in SW region}}
\
s\text{so they are phenotypic females.}

\underline{\text{Kallman's}}

\begin{itemize}
\item \text{Stature}
\item \text{1° Amenorrhea.}
\item \text{a. Kallmann's} \hspace{1cm} \text{much more common}
\item \text{b. pure gonadal dysgenesis. (also correct) - rare disorder.}
\end{itemize}

\rightarrow \text{mc in males compared to females.}

\underline{\text{Mixed Gonadal dysgenesis.}}

\rightarrow \text{mixture of } 46\text{XY} | 45\text{XO}

\rightarrow \text{It is a mosaic pattern}

\rightarrow \text{One gonad is streak & other gonad is poor functionally}

\underline{\text{Testis}}

\rightarrow \text{They have ambiguous ext. genitalia.}

\underline{\text{Hermaphrodites}}

\rightarrow \text{male pseudohermaphrodites:} \rightarrow \text{mc is Androgen Insensitivity syndrome (AIS)}
\rightarrow \text{Testicular Feminisation syndrome (TF5)}

\underline{\text{Gonad \rightarrow Testis}}

\underline{\text{ext. genitalia \rightarrow Female.}}

\rightarrow \text{Female pseudohermaphrodites:} \rightarrow \text{mc Congenital adrenal Hyperplasia (absence of 21 hydroxylase)}

\underline{\text{gonads \rightarrow ovary.}}

\underline{\text{ext. genitalia \rightarrow Virginised (male like).}}
→ True hermaphrodite:

Gonad → ovotestis

Extr. genitalia: ambiguous

→ 2nd mec of 1st amenorrhea is vaginal agenesis.

→ Vaginal agenesis!  gonads 0

→ 2nd sexual characters. (where as Gonadal dysgenesis kallman → absence of 2nd sexual character

1) Mullerian agenesis

2) AIS

3) Imperforate Hymen (1st amenorrhea 2nd sexual character)

4) Vaginal Septa

Mullerian agenesis.

→ Rokitansky Syndrome (MRKH):-

Triad: 1. Mullerian agenesis

characterised by absent vagina
absent uterus

FT are present i.e. distal 1/3rd

2. Skeletal anomalies: 10–15%

- Anomalies mostly in form of
  Hemivertebrae i.e. Cervical Spine

3. Renal anomalies: Renal agenesis > Horseshoe kidney
- Gonad: Ovary (gonadal ridge) karyotype - 46XX

- Breast: N Breast tissue + from Gonads

- Ext. genitalia: + Female-like

- Pubic/auricular hair: + & N

- Androgen: N Female level

- Have their own babies: Yes

- N genetic potential

By -> Surrogate mother

- Rx: Vaginoplasty -> mc Sx is McIndoe

2. Williams

Testicular Feminisation Syndrome (AIS)

- Gonade: Testis

- Karyotype: 46XY

- Ext. genitalia: Female-like

Testis produce N levels of testosterone

Defect: Receptor is insensitive to testosterone

Androgen -> Estrogen

Breast: + They are phenotypic females

Genotypic: Male
pubic / axillary hair — absent (sparsely ☺)

→ They are pseudohermaphrodite.

→ Uterus: absent (Testis produce MIS → Mullerian duct)

→ Can they have babies: NO [only adopt babies]

→ Worst Reproductive outcome: AIS / TFS

→ Undescended Testis (present E in inguinal hernia/abdomen)

mc Tumor to develop in undescended Testes — Gonadoblastoma

mc cancer is Seminoma.

→ Rx: Gonadectomy — after puberty

They are replaced by Estrogen Therapy.

No Gonadectomy in Turner's Syndrome (45X0)

When Female is carrying Y chromosome we can do

Gonadectomy.

→ Best Test to confirm AIS is karyotype.

→ USG

→ MRI

→ Laproscopy

→ Sr. Testosterone level (High in AIS & ☺ in Mullerian agenesis)

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3. **Imperforate Hymen**

- Hymen not opened
- Blood accumulation occurs
- Present & cyclical abd. pain
- ↑ endometriosis
- Can be a medical emergency & urinary retention.

```
Hemato colpos / Hematometra
    / vagina
    /
upars
    /
Bladder outlet obstruction
```

- O/E → Bluish bulging membrane
- Rx: Cruciate incision

- Cribriform Hymen / Sieve Like → multiple small openings

- Septate Hymen ○ 70% 2 openings.

- Vaginal septa:

```
Vaginal septa
```

```
Mc site → Upper 1/3
```

Uterine didelphius - mc mullerian duct defect.

1° Amenorrhea IOC: karyotyping
Amenorrhea

1st Test → UPT → -ve

2 → PCT (progestrone challenge test)

Take oral progesterone for 5 days & then stop

After 1 week

- Bleeding
- No bleeding?

? No progesterone
↓

Anovulate
mc. is PCOS

3. Give E + P challenge test

→ Stop & return.

Bleed → Not bleed

? Deficient in
Estrogen

→ It can happen
at 3 levels

a) Ovary → ? FSH / TSH / Prolactin

m/c = Hyperprolactinemia
m/c = microadenoma of pituitary
< 10 mm

Doc → Cabergine
(long-Acting)

b) Pituitary

c) Hypothalamus

MRI has to be done

a) Ovary → FSH / TSH / Prolactin

TSH → Any type of thyroid abnormality (↑ / ↓) genita

Low & high can cause menstrual
irregularities.

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mri: Tumor, Trauma
Infarct, Infection

In MRI all are (N) Functional
↓ @ in → Hypothalamic amenorrhea due to

a) Anorexia Nervosa
b) Exercise Induced
c) Stress Induced
d) Chronic malnutrition

It is A's of exclusion.

→ 4f (+) does not bleed

damaged endometrium

mc → Asherman's Syndrome

→ Intrauterine synechie (adhesions)

mc = Curetage - mc PPH

1st - Repeated curetage of MTP

-

In India → Genital TB.

Ioc: - Hysteroscopy.

On HSG: may be incidental finding.

R: Hysteroscopic adhesiolysis + High dose CB +
Cu I insert to keep walls separated.
MC pathological cause of primary amenorrhea is PCOS.

PCOS

Menopause: Absence of menses for 12 consecutive months.

Avg. age → 51 yrs. (World)

In India → 47 yrs

Premature menopause: Happens in women < 40 yrs of age.

ASIS: FSH (To Test T Ellis Ovary)

Sr. FSH levels > 40 IU on 2 occasions one mon apart. Is confirmed premature menopause.

MC symptom for menopause are: Vasomotor symptoms i.e.

Hot flashes

Hot Flashes → cause is Δ withdrawal

→ Coincide Δ LH surge

→ Vasomotor symptoms: Rx: Hormone Replacement Therapy.

PCOS / PCOD

→ Klin Stein Leventhal syndrome.

→ Defect lies at level of ovary i.e. ovary stromal hyperplasia.

\[
\text{Ovary} \xrightarrow{\text{Androgens}} \text{Stromal hyperplasia} \xrightarrow{\text{NE}} \text{Total (Theca Cells)}
\]
↑ E₁ → LH+ve → Very follicles begin to develop
FSH-ve → No dominant follicle
No ovulation occurs.

→ Obese
→ Insulin Resistance (30-50%)
→ Hyper Insulinemia
→ Insulin further stimulates stromal hyperplasia

Insulin Resistance
→ HAIR - AN
→ Acanthosis nigricans (cutaneous markers of insulin resistance)
→ Seen at Dark velvety patches
at nape of neck
→ Groin
→ Under breast tissue
→ Axilla.

→ Rotterdam criteria:
  of 2 of the following are met we make a PROVISIONAL diagnosis of PCOS.
  1) Oligomenorrhea / amenorrhea - Characteristic complaint
     Cause: - Anovulation (due to absence of F & no withdrawal)
     2) ↑ Estradiol - Can happen.

  3) Cause Proliferation
  Outgrowth vascular supply
  Avascular necrosis occur.
2) Hyperandrogenemia (Bld levels high) & Hyperandrogenism (Clinical Features due to Blood levels)

Genitalia $\rightarrow$ Sr. Androgen $\uparrow$

i.e. Sr. Testosterone $-$ mildly raised i.e. 70-150ng/dl
value $< 200 \text{ng/dL}$. [$g > 200 \text{ng/dL} -$ 
Secretion Tumor]

Total (we used) 'bcz no definitive value to confer.

$\lessdot$ Free (better)

2. Serum levels of DHEA $\uparrow$

Sr. DHEA $< 700 \mu \text{g/dl}$. [$g > 700 \text{ind} \text{icative of Tumor}$]

(mildly raised)

Genitalia $-$

C/F

Hirsutism

seen in mildly raised levels

seen in PCOS

-> appearance of terminal thick

Coarse hair in male pattern

distribution

-> appearance of acne

-> Gradual in onset &
gradual progression

Mec $\rightarrow$ Idiopathic

and $\rightarrow$ Peed.

Vivilisation

very high levels

Tumor

$\rightarrow$ Clitomegaly

$\rightarrow$ ↑ muscle mass

$\rightarrow$ Temporal resuscit’d of hair.

$\rightarrow$ Hoarseness of voice.

$\rightarrow$ Rapid onset Rapid progress

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Hirsutism

To diagnose Hirsutism

Fearman Gallway:

A score of ≥8 indicate Hirsutism

3rd: USG: appearance of PCOS

a) >12 follicles in the ovary

b) <1cm (2-9mm) size of follicles

c) >10 cc (ovarian volume)

Necklace pattern is seen but not a part of Rother's dom criteria.

D/D of PCOS:

1) PCOS

2) adult onset CAH → cloves D/D, clinically we can't differentiate b/w adult onset CAH & PCOS

3) Tumor >200, >300 DHEA, rapidly progressive onset PCOS

4) Cushing's → Dexamethasone suppression test → we measure is 24 hr urinary cortisol.

→ 48 hr test to differentiate b/w CAH & PCOS.

If Sr level of 17-OHP → >200 ng/dl.

48 hr test we do is ACTH stimulation test

If Sr level of 17-OHP rise to >1500 ng/dl then it is CAH.
→ Can a thin pt. has PCOS → Yes
  only 30-50% obese has PCOS.
→ LH/FSH ratio is used to make Δsis: NOT required,
  - it is just finding.

↓
Ovaries are normal in a pt. with PCOS → Yes.

↓
1) 24 yr old thin girl present with oligo/amenorhea & hirsutism.
  Sr. is raised. USG is (N) → a provisional Δsis is PCOD.

2) A pt. 12 yr old present with menstrual irregularity & oligomenorhea
  are hirsutism. What should be next test of IX-
  a) LH/FSH
  /) Sr. Testosterone → to make Δsis.
  c) USG pelvis. → might be (N)

Lab Findings in PCOD:
1) LH/FSH > 2.1 (are persistently raised)
2) Sr. Androgen levels are raised.
3) Total E2 → E1 is increased while Total E2 is (N)
  Free E2 is ↑ due to low SHBG.
4) SHBG ↓ed because androgen has higher affinity & have
  -ve feed back on SHBG.
5) Insulin resistance: – 45 gm OGTT (N) Female BS > 126
  (I) 110-126 Impaired
  2hr pp > 200
  140-200
6 → Lipid profile → metabolic syndrome (syndrome X)
   → usually abnormal

7 → Sr. prolactin levels are usually \( \square \)
   In up to 25% pt → there may be hyperprolactinemia.

8 → USG.
   C/F of prog: Short Term Complication
   Long Term Complication
   → Hirsutism
   → Irregular cycle
   → [Irritability]
   → Infertility

1. CAD
2. DM type 2
3. Endometrial CA
4. Ovarian CA
5. Sleep apnea syndrome
6. Nephropathy
7. Alcoholic hepatic cirrhosis
8. Psychiatric character (anxiety, depression)

8. Pregnancy complication
   a) Abortion
   b) AM
   c) Preterm labour
   d) Preeclampsia
   e) Still birth

DOC: OCP's:

Doc's: COC's (at low dose E, 3rd gen)

→ Spironolactone
→ Cyproterone acetate
→ Flutamide
→ Ketoconazole
→ GnRH agonists (Continuous)
→ Metformin (not given for lean pts)
2) Infertility - anovulate

1st advise: obese PCOS pt infertility: is

a) wt loss → 5-10% of wlt loss

**Doc:** To induce ovulation re: Clomiphene Citrate (GEMR)

- **Note:** It only has antagonistic action → it has central act to bring ovulation.

**Doc:**

- **Clomiphene Citrate:** (cc):

50 mg for 5 days in menstrual cycle (D2-D6 or D5 to D9) in early part of menstrual cycle.

- **Approved dose is 100mg**.

- **Max. Time we can give is 12 cycles** (past practically 6 mon)

- 80% ovulate → endometrium thin

- 40% will conceive → thick cervical mucus.

- 80 ovulate % more than conceive %

- **Twin pregnancy - 6-8% (<10%)**

**Ovarian Hyperstimulat Synd. OHSS → rage 2 Clomiph Citrate < 1%**

**mc side effect of c.c: Hot flushes**

2° mc: Ovarian cyst format
Indication to stop C.C is: when we see visual symptoms

C.C doesn't cause hypospadias (Diethylstilbestrol causes hypospadias)

C.C is not teratogenic.

The SERM that can be used for ovulation induction is Tamoxifen (Clomiphene citrate, intolerance pt.)

Raloxifene is not to be used in ovulation induction.

if pt not showing any pt response to C.C & obese, we use C.C + metformin (concept occurs)

Second line agents in PCOS:

(a) Gonadotropins (inj LH/FSH)

*Low dose 75 IU - to be given.*

*High dose: → cause twin pregnancy rate 30%.*

*OHSS: 15%.*

(b) Laparoscopic ovarian drilling → destroy stromal tissue
(by laser or cryotherapy)

Untrained → premature ovarian failure is caused.

Third line: GnRH agonist:

Luprolide | goserelin | nafarelin (give s/c, I/m, I/v)

For ovulation → They are given in pulsatile manner.

Can't be given oral route.
Continuous GnRH is given in
- for mX of precocious puberty (for suppress)
- pain relief endometriosis
- reduce size of fibroid
- given in prostate & breast cancers
- Hirsutism.
- Infertility. (For suppress of endogenous gonadotropins)
  
  GnRH agonists  OCP's (LH & FSH)

- all the following can be used in aroid lady PCOS and infertility except:
  a) CC  b) tamoxifen  c) OCP's  e.g. norgestimate, not to conceive.

- 4th is aromatase inhibitors
  Letrozole (not approved due to teratogenicity)

- 5th Bromocriptine - co hyperprolactinemia.
  Safe. [not cabergolin]

Twin pregnancy:

<table>
<thead>
<tr>
<th>Gonadotropin</th>
<th>CC</th>
<th>GnRH agonist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moss:</td>
<td>Gonadotropin &gt; GnRH &gt; CC.</td>
<td></td>
</tr>
</tbody>
</table>
OHSS (Ovarian Hypersimulate Syndrome).

Cause: - 
1. VEGF produced by ovaries.

2. Inj HCG (Trigger for ovulation) → after trigger 36 hrs ovulation occur.

LH surge
↓
ovulation

→ mc in younger females

→ High Follicle Count (PCOS pt's)

→ In pt's 

Prevent of OHSS: - 1. Avoid giving HCG by giving inj LH (or) GnRH

2. Coasting - delaying HCG injection (to let E2 come down then give trigger oplu E2 level is down)

3. IVM - In vitro maturation of oocytes

   NO FSH inj
   ↓
   No hyperstimulation of follicles.

4. 9/6 albumin & dextran.

5. Cryopreservation: - Take out grown eggs from female body & don't fuse E2 current one. Make hCG come down & give it in next cycle.

6. Pregnancy → OHSS severity

In PCOS → IVF is not precise &.
All the following are causes of infertility except:

a) CC
b) IVF (Intrauterine Insemination) now along w CC it is giving
c) metformin
d) IVF

Infertility

→ It is inability to conceive even after 1 yr of unprotected intercourse.

→ Male factor alone cause in 20% cases

→ Male factor contributes in 40% cases.

→ Female factor = 40–55%.

→ Unexplained contribute 10% of cases.

→ Female factors:
  Factors such as Ovulatory = 40% → anovulatory i.e. PCOS.
  Tubal = 30%
  Uterine = 15%
  Cervical = 5%
  Unexplained = 10%.

Tests of Ovulation:

1) Basal Body Temp.:— Raise 0.5°F temp. 0.5–0.8
2) [other tests mentioned]

Biphasic chart which means

First 14 days → not ovulatory
Next 14 days → ovulatory.
2) Cervical mucus:
   - Thick mucus

3) Vaginal cytology:
   - Superficial cells
   - Intermediate cells

   Swab taken from lateral vaginal fornix

4) Sr. progesterone levels - Day 10 of the cycle

   $\geq 3$ ng/ml $\rightarrow$ Then it is ovulatory cycle

5) Endometrial biopsy - In infertile female at least once

   In India: Premenstrual phase
   2 days before the expected date

   HPE  AFB

   Result: on HPE Secretory endometrium $\rightarrow$ It is $\square$ not $\square$

   If it result is proliferative endometrium, it is anovulation
   $\square$ is active, no $\square$

6) Urinary LH kits: To pick up LH surge (not ovulation)

7) Transvaginal USG is used to study ovulation which is

   Follicular monitoring

   Start on D10

   O $\in$ $\in$ $\in$

   Follicle size $\uparrow$ $\rightarrow$ $\uparrow$ mm/day

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- Mature Follicle: 18-20 mm
- Trigger can be given when follicular size is 18-20 mm.
- When a sudden ↓ in size of a growing follicle shows ovulation.
- Free fluid in POP.

Appearance of endometrium in periovulatory phase is called as a Triple layer endometrium (becoz it is seen as bright 3 hyperchoic lines).

b) Luteal phase (secretory phase): seen as single hyperechoic line & post enhancement.

- MC used test is [USG] for follicular monitoring (contapulation ovulation)

Easiest test is: Sr. progesterone levels (Test given on D21)

Very reliable.

Drawback: retrospective study.

- Others tests are:

Tests for ovarian Reserve:

1. a) MC test for ovarian reserve is:

   - Sr. FSH on D3
     - 0-10 IU - Normal level.
     - 10-15 IU - Borderline Reserve.
     - >15 IU - Poor Reserve.

2. b) Sr. Inhibin B on D3 → <45 pg/ml.
3) AFC: Antral Follicle Count < 10 → Poor reserve
done on D3 of menstrual cycle.

4) Levels of Antimullerian hormones → best & latest Test.
   → It doesn't show any change during ovulation
   if level < 1 ng/ml.

5) Clomiphene Citrate Challenge Test:
   on D3 → Test Sr. in FSH
   D5 → D9 give 100 mg O.C.
   on D10 → check FSH level.
   High basal level which rise further after giving cc indicate
   a poor ovarian reserve.

**TUBAL FACTOR**

For assessing Tubal patency: HSG

1. HSG: Done after day D5 → D11 (mostly on day 10)
on postmenstrual phase of menstrual cycle.

   Absolute C/I →
   1. Preg.
   2. Known case of genital TB.
      because it cause dissemination.
      If this is done.
   3. Current pelvic Infection
   4. Contrast allergy

NOTES FROM
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09654691327
Peritoneal spill seen if Tubal Patency, we can see this.

Hecht Wilkinson's cannula.

Dye: - water soluble Iodinate.

Amount is 10ml.

Filling defect: - seen in polyps.

They are seen early in HSG.

Anomalies

Fibroids.

HSG tells about interior luminal side of FT.

HSG is a screening tool.

Of a screening test

Ob(N)


HSG followed by Lap = Best Test.

Cornual Block:

If B/L blocks is at Cornual level it is proximal segment.

If it occurs block occurs at fimbrial it is distal block.

Mid segmental block.

Cornual Block: -

Better prognosis is if B/L Cornual block. (most of cornual blocks are physiological).

HSG is painful test, no anaesthesia is given.
- Cornua is made up of muscles so when dye goes muscle spasm occur. i.e., Cornual spasm occur.

2. Mucus plugs.

Rx: - open or up spasm or dislodge the plug of mucus.

\[\text{Tubal cannulation}\]

If it is spasm -> it opens

If mucus -> it dislodges.

Affective in 90% of pt's is Bil Cornual Blocks.

- Cannulation USG
  done under hysteroscopy (mc)

If we get Bil cornual block on HSG,

\[\text{next step.}\]

Asic Laparoscopy (to look exterior of tubes)

+ Hysteroscopic cannulation

+ Chromopertubation

- Those who don't respond to cannulation (i.e., block is not physiological it is pathological)

So we do

- Surgery
- IVF

If cornual block, IVF is preferred

No surgery performed.
For B/L Cornual block Best Rx: Tubal Cannulation

next step

(a) mid segment Block:

- undergone sterilization
- but some reason they want to reversal surgery for concept
- on what factors concept rate depend is

1) Type of sterilizat:

Best reversal is C

Clips > Falipe ring > modified pomeroy > cautery

2) Type of reanostomosis:

Isthamo isthomic - best (same diameter)

3) After reanostomosis, total length of FT achieved

If length > 4 cm → good outcome.

4) of only cause of infertility is sterilization.

→ Before doing Reversal sterilizat, which test we have to do is

a) Husband Serum analysis is mandatory.

b) Distal block: B/L Fimbrial block.

Laparoscopy

mild disease Rx: Surgery (Fimbrioplasty)

Severe disease Rx: IVF.
Tubal disease where we can make IVF

- Severe B/L disease (endometriosis, 78)
- Proximal / Distal Block
- B/L hydrosalpinx
  - Feto toxic (fluid that enter uterine cavity)
  - So Tubal clipping / salpingectomy Then do IVF.

Genital TB

- It is a Tubal Factor.

- It is always a B L infection

- Inc 1° for this is Lungs > LN > Urinary tract > Bones

- MC Route of spread is hematogenous [others lymphatic, ascending inf, Direct spread]

- MC site → FT involved in 90% of cases.
  1. Endometrium involved in 50-60% cases,
  2. Ovary involved 15-25%.
  3. Cervix - 5-15%.
  4. Least is vagina/vulva → <1% cases.

Among FT, MC part involved is Ampulla

- It is always B/L disease.

- In acute - Tubes are red & inflamed.
- In Chronic - Tubes become thick walled.

If we do HSG on chronic phase
- Hydrocele (when tubal end gets blocked)
- 
- remain Patent (open) → Shows lead pipe appearance / Tobacco pouch appearance

2) Endometrium:

Route of spread → direct spread

In acute Genital TB → Endometrium

In chronic Genital TB → Endometrium present 2 ulcers / adhesions Asherman's syndrome.

3) Ovary:

Tubercles on surface of ovary.
Tubo ovarian mass
Tubo ovarian Abscess.

4) CX:

Present as CX growth D/D CA CX

-> mc complaint pt present as Genital TB:

Inferiflity > Pain > Menstrual irregularities

First menstrual complaint is Menorrhagia (Inflammatory of endomet

mc complaints of menstrual irregularities – oligo / amenorrhagia

-> what % of pt & genital TB is Infertile: up to 70% (45-70)

What % of infertile couples have genital TB:

On world – 10%

In India – 17%

OE mc finding in reproductive age of women: ( Pelvic examination

Tenderness.)
Mc finding in genital TB in adolescent girls is B/L adnexal masses (which is least in reproductive women)

**Agenesis of Genital TB:**

(a) Endometrial biopsy → AFB
    → Culture (Best)

(b) Menstrual blood PCR (on Day 4) → So not best test.
   ↓
   Highly sensitive it picks up dead bacilli also.

**Rx:**

ATT for a period of 6 mon (to cure the disease)

Infertility due to genital TB is IVF

**Cervical Factor:**

→ Antisperm AB → Test is Post Coital Test.
    → Cervical consistency.
    → Cervical mucus is collected on Day 12 – Day 14

Cervical mucus is kept in slide and examined under microscope.

They show forward motility.

♀ Progressive

Antisperm antibodies: if ♀ sperm show circular motility.

---

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Modern test is Immunobead \( \Rightarrow \) Tests for Anti sperm AB.
- Sperm agglutination tests.

\[ R_x : IUI \text{ (directly inseminated sperm into cervix)} \]

0.5ml (processed)

Immunological Infertility \( \Rightarrow \) Exc IUI

**Uterine Factor:**

1) Congenital
2) Endometritis
3) Fibroid
4) DES
5) Retroverted

**Congenital Anomalies**

- mc \( \Rightarrow \) Conging congenital anomaly is Bicornuate uterus \( \Rightarrow \) septal
- mc \( \Rightarrow \) abortion - septate

\[ \Rightarrow \text{which mullerian defect mc are } \Rightarrow \text{Infertility } \Rightarrow \text{unicornuate} \]

ectopic ovary ass. \( \Rightarrow \)

\[ \Rightarrow \text{which mullerian anomaly has best reproductive outcome:} \]

Arcuate > Didelphic > Bicornuate

\[ \Rightarrow \text{which mullerian anomaly where surgery is not required} \]

Arcuate > Didelphic > Bicornuate

\[ \Rightarrow \text{worst reproductive outcome is } \Rightarrow \text{unicornuate} \]
IOCs: MRI (non-invasive)

Bicornuate uterus  

Septate uterus

HSG: For differential basis of these bicornuate & septate:

Fundus

Bicornuate

Septate

Gold standard: Laparoscopy

→ 2D USG is poor to recognize uterine anomalies, so it is not routine

→ SIS (saline infusion sonography) → give good result method to visualize anomalies, non-invasive which can differentiate bicornuate & septate.

Rx:

→ Hysteroscopic resection of septa

→ Tompkins & Jones for septate uterus

In HSG we see:

\[ \text{angle between two horns is } \leq 60^\circ \text{ } \rightarrow \text{septate} \]

\[ < 4 \text{ cm} \]
For Didelphys & Bicornuate

\( \text{by unification} \) \( s_x \) i.e. \( \text{Strussman metroplasty} \)

All are (Strauss's man) \( s_x \) for mullerian anomalies except:

- Tompes
- Genes
- Strauss

\( \sqrt{\text{mc Indoe (vaginoplasty)}} \)

All mullerian anomalies high associat' \& urinary tract (30%)

Must undergo IVP & renal USG.

Mullerian anomaly is highest risk is unicorneate.

\( \Rightarrow \text{DES} \) - cause

a) vagina \( \Rightarrow \) v. adenosis

Clear all Ca.

b) \( C_x \) \( \Rightarrow \) Collar / hood / \( \text{CIN} / \text{adeno Ca} \) \( C_x \)

c) Uterus \( \Rightarrow \) mc is hypoplastic.

Characteristic shape when uterus exposed to DES is

\( \text{T} \) Shape

d) FT - para tubal cyst.

Renal anomalies \( \oplus \) not seen in females/babies.

\( \text{Seen in male babies} \)
Male Factor Infertility

Best factor: Semen analysis (HSA)

Best method of semen analysis is masturbation

- Test done on liquified semen.
- Avg. time for liquifaction = 30 min.
- Max. time for liquifaction = 60 min (HSA)
- Avg. time taken by sperms to reach the site of fertilization is 30 min

Q/S For semen analysis we follow WHO parameters.

WHO PARAMETERS:

Volume > 1.5 mL

pH > 7.2

Total sperm count > 39 million / ejaculate

Sperm Conc > 15 million / mL

Total motility > 40%

Progressive motility > 32%

Vitality (sperms are alive) > 58%

Morphology > 4%

WBC count < 1 million / mL

Aspermia: absence of ejaculate

Azoospermia: absence of sperms in ejaculate
oligospermia: < 15 million/mL
Severe oligospermia: < 5 million/mL
Asthenospermia: reduced motility
Teratoospermia: abnormal morphology
Necrospermia: dead sperms
Globospermia: sperms which lack acrosomal cap; sperms have round head.

Azoospermia

\[ \rightarrow \quad \text{semen fructose} \]

\[ \quad \oplus \quad \text{obstructed epididymis} \]

1. To look obstructed
   - Advise scrotal USG

\[ \quad \downarrow \quad \text{absent} \quad \text{Sr. LH/FSH/testosterone} \]

\[ \quad \text{N} \quad \downarrow \quad \text{but LH & FSH up} \quad \text{Defect lies at gonad} \]

\[ \quad \text{TRUS} \quad \text{(look for seminal vesicles)} \]

\[ \quad \rightarrow \quad \% \text{absent seminal vesicles} \]

\[ \quad \downarrow \quad \text{do} \quad \text{CFTR gene mutation} \]

\[ \quad \text{Obstructive azoospermia (vas is not palpable)} \]

\[ \text{Rx: resect \& reanastomosis of vas deferens} \]

Low fructose
Low pH
Low viscosity

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Rx of mild male factor infertility:

Counts:
- 15 million/ml → IUI
- 5-15 million/ml → IVF
- < 5 million → ICSI

For IVF → sperm most important factor is motility
For ICSI → most important factor is morphology.

Endometriosis:

- Ectopic endometrial glands & stroma

mc site: ovary > peritoneal lig > post. leaf of broad lig.
> FT

mc theory:
1) Theory of retrograde menstruation → Sampson's theory.
2) Coelomic metaplasia.
3) Immunological - Both cell mediated & humoral immunity is reduced.
4) Genetic: K-ras

1st degree of relative is effective risk of endometriosis = 7 times high risk

Endometriosis occur in reproductive age group:
- b'cuz ovarian hormone is high
- Vafe in adolescent / post menopausal women.
In pregnancy → It improves because of decidua of implants.

I.O.C → Laparoscopy

Gold standard → HPE

ASRM divided Endometriosis

minimal → m. implants are superficial isolated.
mild → superficial but multiple implants < 5 cm in diameter.
moderate → superficial & deep implants
severe → superficial + deep implants + Adhesions

Appearance of an ovarian mass (endometrioma of ovary)

Endometrioma:

on USG: Homogenous Ground glass appearance.

DIO: Hemorrhagic Cyst on USG & cyst appears as Fish net appearance.

MC complaint in Endometriosis:

(a) Pain – i.e. dysmenorrhea (MC) > Chronic pelvic pain >

   dyspareunia > lower back pain (least)

Other complaints:

(b) Infertility – inability to conceive.

(c) Menstrual irregularities usually in form of menorrhagia.

Examination Findings:

P/V: Tenderness of POD

- Uterine Tenderness
- Tenderness of utero sacral lig. (nodularity)
— we may even palpate adnexal mass (tube + ovary)

— Retroverted fixed uterus (manually can’t be corrected)

\[ Rx \]

1) For pain — → i° medical

we can do surgical management.

→ medical mx

→ For minimal & mild → Doc is Coop’s & NSAID’s

Pain & menstrual complaints
Just 2 pain but
Regular cycle (2)

→ For moderate pain — → GnRH agonists Continuous manner

→ Progesterone (oral, intrauterine form / IUCD form)

Down

MPA MIRENA DMPA

Caused decidualisation of implant & relieve pain.

→ Pain & depth of implant

Depth determines pain.

3: aromatase Ε’s → cause Ε of synthesis

4: Danazol (androgenic Ε/E)

→ Surgical mx:

1) pt non-responsive to medical mx

2) of pt presents 2 acute severe pain which is intolerable.

Of endometriosis involves Bowel & Urinary Tract.
4) Ovarian cyst:

\[ S_X = \]

1. Adhesiolysis

2. Fulgurate the implants (laser or cryotherapy) 

(Gum)

3. Presacral neurectomy (cut the nerves that cause pain)


**Ovarian cyst / endometrioma**

\[ R_{X OC} = \text{cystectomy} \]

(Aspirat\(\text{ }\) | Laser ablation of cyst wall is not done because recurrence rate is very high)

In infertility cause:

In moderate & severe disease: Tubal factor + Ovarian Factor.

**mcc of infertility is Ovarian factor, infertility**

\[ \rightarrow \ \text{Oogenesis is defective} \]

\[ \rightarrow \ \text{Ovum - poor genetic quality} \]

\[ \rightarrow \ \text{Ovum pick up defective} \]

Cauing

\[ \rightarrow \text{Minimal & mild infertility: } \]

\[ \rightarrow \text{Superovulation } \rightarrow \text{ C.C + IUI \& 3 cycle disease} \]

\[ \downarrow \text{If don't respond} \]

\[ \text{Ivf} \]

\[ \rightarrow \text{Moderate \& severe disease: } \]

\[ \text{Cause infertility: } \rightarrow \text{IVF} + S_X \text{ (Corrective)} \]

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Fibroid.

- mc pelvic Tumor in females

- It is due to smooth tissue proliferati^n.
  - Depend on (E + P)
  - Proliferati^n Prevents apoptosis.

Types:
1. Intramural → mc
2. Subserosal
3. Submucosal

depend on hysteroscopic appearance it divided into 3 types.

Type 0
Type 1
Type 2

entirely intracavitary 750% is intracavitary

Intracavitary

→ hysteroscopically not resectable.

- mc presentation of fibroids: asymptomatic.
  - A woman > 35 yr old 50% develop fibroid.

- mc symptom: bleeding (menorrhagia)
  - Fibroids always cause regular cycles.
  - Which fibroid mc cause menorrhagia be submucosal
  - When fibroid represent 2 metrorrhagia:
    - When there is fibroid polyp (pedunculated submucosal)
Inversion of uterus present or Fundal Submucosal pedunculated

→ [mc present]n of Fundal Fibroid = menorrhagia.

→ Intramural cause → menorrhagia.

→ Subserosal → don't cause menorrhagia

2) Pain :— Dysmenorrhea can be present (but not chief complaint).

→ Fibroid don't cause dyspareunia.

b) When fibroid undergo degenerat° & Torsion.

↓

For degenerat° mc is Hyaline degenerat°

[wombstone — calcified fibroid]

Least common degeneration → Sarcomatous degenerat° from

It is malignancy.

Incidence < 0.5%.

→ mcommonly submucosal develop sarcomatous degener

→ In postmenopausal women: Fibroid ↓ in size.

if there is pain u ↑ in size → shows sarcomatous change

⇒ Red degenerat° : Pain

2nd Trimester:

- acute abdominal pain + N & V + Fever & ↑ TLC

Rx: Conservative put on bed rest

Analgesics

Antipyretics, lot of I/v fluids.

No myomectomy & No antibiotics
Torsion: Pedunculated subserosal fibroid

Present in acute abdomen.

3. Pressure:
   - Ant. uterine wall fibroids → frequency of micturition (initiate)
   - Post. uterine wall fibroid produce urinary retention
   - Urinary retention is feature of cervical fibroids

→ Fibroids cause

4) Infertility
5) b) Recurrent preg. loss
6) c) Preterm labour
4) Abruption > praevia
8) PPH
9) IUGR

Inslor's Cervical Score:- It's a semi-quantitative method, used to assess quantity & quality of cervical mucus.

5 parameters & each is scored from 0-3

1. Appearance of Os:
   0 - Os is closed
   3 - Os is widely open
2. Mucus quantity
   0 - No mucus
   3 - Mucus visible at external Os
3rd parameter: Spinnbarkel
0 - mucus doesn't stretch
3 - stretches to more than 8 cm without breaking.

4. Fermen
0 - no fermenting
3 - all mucus shows good fermenting.

5. Cellularity (No. of leukocytes) is assessed as reverse score:
0 - Full of leukocytes,
3 - No. of leukocytes

Max. score = 15

≥ 12 = Good ovulatory cervical mucus
10-11 = adequate cervical mucus

→ Only cervical dilatation done if Pyometra.
→ mc Benign vaginal Rx - Condyloma.
→ To differentiate bl/w hypothalamic & pituitary amenorrhea - GnRH stimulation test has to be done.
→ Logwheel sign seen in acute salpingitis
→ Postmenopausal hormone levels = (E) ↓ & Gonadotropin ↑
→ Contracept for newly married couple - OCP.
→ Uterine polyp can be removed adequately c Hysteroscopy.
→ Anencephaly can be detected in 13 wks.
Notes from Jain Stationery
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True Broad lig. fibroid  
False Broad lig. fibroid

Denovo b/w layers of broad lig.

Subserosal fibroid uterus

b/w its layers.

Ureter lies medial

Ureter lies lat. to BL.

Loc of fibroid: USG hypoechoic.

Fibroid

Adenomyosis

Endometrial glands and

stroma in myometrium

mc → menorrhagia.

mc → menorrhagia + Dysmenorrh.

Irregular growth of uterus

→ Symmetrical growth so it is

globular globular uterus

Non tender uterus

Tender uterus

10 wks uterus pregnancy

10 wks preg. uterine size

Loc: USG

Loc: MRI

Gold standard: HPE

At minimum distance of deposits

from junctional zone.

Atleast 1 HPF deep

in MRI: Junctional zone is thick

Junctional where endometrial and

myometrium starts is not clear

also seen in endometriosis

Rx: Hysterectomy.
Fibroid is not capsulated. Fibroid has pseudocapsule.

Rx of fibroid:
1) Medical
2) Minimally Invasive
3) Surgical

Medical Rx:
- For symptoms & reduce size

1) Drugs that reduce symptoms:
   a. [OCP for menorrhagia] → Cope's (low dose)
   b. LNG IUD - MIRENA (reduce blood loss) → expensive
   c. Tranexamic acid (antifibrinolytic) - control heavy menstrual blood loss

2) Drugs which reduce size of fibroid:
   a) GnRH agonist: Continuous
      - If we stop GnRH agonist, regrows
      - Max time is 6 mo.
      - GnRH agonist cause Osteoporosis.

In order to give 6 mo. Therapy so give add back Therapy (estrogen) to protect bone.
→ Only used in pre-operative period to reduce intra-operative Blood loss.

Myoma Screw → To remove myoma.

Plane of cleavage lost when we use myoma Screw.

Myomectomy is difficult if we lose plane of cleavage.
b) GnRH antagonists: NO flare response.
   - Initially ↑ LH & FSH → ↑ size.

c) Mifepristone (SPAM)
d) Uprisral

e) Aromatase inhibitors.

- Minimally Invasive methods:
  - UAE: Uterine artery embolization
    - not a 1st line under
  - UAE Done if:
    - not willing/can’t go for Sx
    - not responding medical my
    - Future pregnancy is not desired

MRg HIFU (non invasive technique)
   - MRI guided high intensity focused USG
   - Done in:
     1. 0-7 cm
     2. Of future preg. not desired
     3. < 5cm in no. of fibroid
     4. < 10 cm size
   - Not useful in heavy calcified fibroid.

Surgical mx: Sx

Conservative / Radical

a) Myomectomy
   - Hysterectomy
   - Mx Indication
   - TAH is
t Abd. Hysterectomy (TAH)

Laser / Cryotherapy

a) Myomectomy: 3 types
   - Parts that remove in TAH is
     - abdominal / subserosal
     - Uterus + Cx
     - Laproscopic / Intramural / Sub Total = Uterus Removal
Conservative

Hysteroscopic - only submucosal Pan Hysterectomy: Uterine Cervical + FT

Type I / Type II - KIA TAH + Bil Salpingo Oophorectomy

Laparoscopic best:
- Less operative Time
- Recurrence rate < Lap is
- Less Blood loss
- High than myomectomy
- Less post op stay
- Early ambulatory

Hysteroscopic procedure is bad:
- When Type III Submucosal is operated there will be
  ↑ blood loss.
- For hysteroscopy to visualize the structures distension
  media (saline/glycerine is used)
  - we use more which cause electrolytic imbalance
    1) Water absorption
    2) Electrolyte imbalances (we can't control)
    3) Perforation
    4) Infection

Prolapse

De Lancer's levels of Support:

Level I: Uterosacral / Mackenrodt
  - of weak then uterosacral descent

Level II: Paravaginal Tissue
  - if weak cause Cystocele: (Prolapse of bladder)

Level III: Perineal Body + muscles attached to it
  - Weak Recarocele Post Urethrocele Ant
Enteroccele: Gut protrudes through Pud.

Degree of Prolapse

1st degree prolapse: descent but it lies above introitus

2nd degree: at level of introitus

3rd degree: descent but it lies below introitus

POPQ: pelvic organ prolapse quantification

Level of reference: hymen

Grade 0: no prolapse

1: prolapse lies >1 cm above hymen

2: 0 to 1 cm above or below hymen

3: >1 cm below hymen

4: max prolapse

Baddoo Walker half way system:

Reference point: hymen

Prolapse seen in postmenopausal women

Cause of prolapse occurring in reproductive age:

1. CT disorders
2. Congenital elongation
3. Congenital elongation CT

Risk factors:

1. Prolonged labour
2. Difficult labour
3. Episiotomy
4. Instrumental
5. Multiparity
6. Cough /constipation
7. Menopause

3rd degree enteroccele: descent 60 yr old

Cystocele + Rectocele

Rx: VH + Ant colpotrophy + Post colposuspension

(strengthen wall)
or VH + PFR (Pelvic Floor Repair) vaginal Hysterectomy.

Female is in reproductive age group, family not complete

1) Sling
   - Merselene Tape To suspend uterus. Uterus suspended to

2) Khanna sling
   - One tape to uterus & another to ant supiliac spine.

3) Modified Shirodkar abdom sling: one end Rectus uteru
   - Psoas hook: in middle, Psoas m'attach. other end Rectus sheath

- Also used in poor muscle tone.
- Now using.

4) Cervixpexy
   - one side Cx mesh undergone fission.
   - other side sacral promontory.
   - Outcome is better

5) Manchester repair/Fothergills Repair;
   - 1st perform ant. colpoprhaphy
   - cut & ligate mackenrosti (then cervix is free)

3) Cervical amputation

4) Reattach the mackenrosti anteriorly on Cx

5) Raw area on C & 2 vaginal mucosa
Done in Cervical elongation

- CT in nulliparous women. (women who desire Child bearing)
- Done only if Family is complete.
6) Shirodkar's modification of Manchester.

All steps 1, 2, 4, 5 is done & no cervical amputation

Done in nulliparous women

Not done for cervical elongation.

A lady is 80 yr old (poorly controlled)

Short surgical procedure has to be done i.e.

LeFort's colpocleisis: — done in 30 min.

Scrap ant & post vaginal walls

Then 2 broad vaginal walls come in contact so

that Cx can't come out.

Done in Ca Cx

Pessary indications: — Ca endometriosis

→ 1. 80 yr + CAO + MI + HTN can't be operated.

Indications to use ring pessary.

2. Early pregnancy 2 prolapse: 2nd indication

Ring pessary remove by 16-18 wks

3. Puerperium.

In ulcers — Pessary should be avoided.

Cause of ulcer in prolapse is venous engorgement

(venous return)

Rx: Reposition by

- AG packing (Apridine + Glycerin)

absorb H₂O

Reposition prolapse.

→ Vault Prolapse:

Apex of vagina after hysterectomy → vault.

Abdominal Sx

Vaginal Sx

Sacralopexy [Best outcome] Sacrospinous fixation [Better]

suspend vagina by sacrospinous
Abdominal Sx  Vaginal Sx

Uterosacral suspens 2) Uterosacral suspens

Better is
Sacrocolpopexy > Sacrospinous > Uterosacral

⇒ Colposuspension: Sx for stress urinary incontinence (SUI)
vagina Those are abd. Sx

2 Types of Colposuspension Sx

1. Burch colposusp 2. MMK

Part of urethra suspended is proximal urethra within vaginal neck.

Gold standard is: Colposuspens

In Burch: It is suspended to Cooper lig.
In MMK: It is suspended to peristium of Symphysis pubis

SUI Stress Urinary Incontinence

TOT TVT

Trans Obturator Tape Trans Vaginal Tape

Route Vaginal Sx Vaginal Sx

Day Care Sx Day Care Sx

Mid urethra is Suspended mid urethra is Suspended

Better Sx

TOT > TVT

In this we don't enter Retropubic space (Space of Retzius)
In TOT Complication loss & blood loss is less.

**VVF Vaginal Vaginal Fistula**

\[ \Rightarrow \] **LATZKO Sx:** Sx of VVF Repair.

1. MCC is obstructed labour (in developing countries)
2. MCC is Gynaecological Sx (malignancy) in developed countries
3. Most common Fistula is VVF.

**Urogenital Fistula:**

Urogenital Fistula show dribbling.

*Test of Fistula is 3 Swab Test (To know Type of Fistula)*

- If lower 2 swabs are stained then it is VVF.
- If uppermost swab is wet but not stained blue then it is Ureterovaginal Fistula.

Most imp. Investigation of VVF: [Cystoscopy] (Tells about size & details of fistula)

\[ \rightarrow \] **MC Site:**

- In Obstetric Fistula → High Vaginal Fistula (vault)
- In Sx Fistula → Juxta cervical

**MC Route of Repair of VVF:** Sx Vaginal Route

**MC Sx is Latzko (Flap Repair)**

Abdominal Repair done in

1. In complicated Fistula (vesicle & rectal Coexist)
2. Malignancy induced by Fistula's
3. Fistula's due to Radiotherapy
4. Fistula's not which fail vaginal route

**JAIN STATIONERY**

09654691327
Oncology

Ca Cervix

Mc Ca in woman = Breast Ca (Indian women)
2nd = Ca Cervix

It starts as Infection
    majority will clear Infection, some would persist
    Infection
        preinvasive lesions
            Invasive

avg. time to convert into Ca is \([10\text{–}15\text{ yrs}]\)

I:\-

a) Universal Screening:
    - Begin by 21 yrs of age, irrespective of sexual activity
        Pap smear – screening
            every 3 years Interval

PAP + HPV DNA – 5 yrs (Interval in combination)

Highly Sensitive: HPV DNA only done in women \(\geq 30\text{ yr} \) to pickup
High Specificity: HPV & PAPs persistent infection

If women is HIV+ve the screening should be done Annually up to the end point 65 yrs of age when PAPs in last decade are not suspicious.

If any of PAP or smear is suspicious screening should be done is 75 yrs age.
PAP's: Ayer's spatula

- Bifid end is used for taking PAP
- Blunt end of spatula is used for hormonal assessment for vaginal cytology

Spatula is made up of wood

C/I to take PAP smear:
- Absolutely no C/I for PAP smear if women is severely bleeding we didn't do this.

- PAP should be taken at 1st antenatal visit in preg. women

PAP taken from ecto. Cx - Part that is screening squamo-columnar junction

- Transformation Zone

- By Ayer's spatula we collect secretion and put on slide which is not air dry and put the slide in fixative 95% Ethyl alcohol & 5% Ether

Pap smear report is known as cytology report:

Bethesda classification system 2000 (modified in 2001)

1. Normal Smear
2. Infection (The organism has to be specified)
3. Reactive reparative changes (Once there was infect & now it is healing)
4. ASCUS Atypical squamous cell of undetermined significance
5. LSIL (low grade squamous intraepithelial lesion)
6. HSIL (high grade squamous intraepithelial lesion)
7. Cancer
Transitional Zone: (TZ)

1. As Tz recedes inside the endocervix.
   - In Puberty
   - Pregnancy
   - Women Taking OCs

2. Metaplasia
   - Dysplasia
   - Premalignant condition

Before 2000 system the Pap smear result comes in dysplasia/CIN system.

Now if we get LSIL which corresponds to mild dysplasia.

- CIN-1
  - LSIL: mild dysplasia (when only lower 1/3rd of epithelium is dysplastic)

- CIN-2 (moderate dysplasia) → lower 2/3rd of epithelium is dysplastic

- CIN-3 (severe dysplasia)

- CIS (when greater than lower 2/3rd is dysplastic)

- CIS (entire epithelium is dysplastic)
  - It is a premalignant lesion

Intact Basement membrane (pre-invasive lesion)

Breach Broken BM → Invasive lesion
PAPS - Screening Tool

Confirmatory by Biopsy

Biopsy - from some abnormal area to pick up disease
Colposcope is a microscope to detect abn. area

ectopy is seen on colposcope
→ The max. magnification possible for colposcope is 30 (10-30x)

Biopsy is taken when
→ 1st thing that pick up by colposcope is to detect abn. area
(a) abnormal vessels - reticular/mosaic/punctate
(b) Surface contour - irregular
(c) Ab norm. area colour is paler compared to healthy pink

Visual Inspection (VI)
Both used for screening & pick up abn. areas
VIA
VI 3% acetic acid
3-5% acetic acid
abn. areas stained \( \square \)
aceto white
ab norm. areas
→ Lugol's iodine
3-4%. Lugol iodine

where there is glycogen

metaplastia areas are not stained - Grey we
ab norm. areas didn't stain so
we didn't take biopsy
Ab norm. areas are unstained
from this beam we didn't
need

Colposcopy: HPE report
PAP's cytology

Now colposcopy reports come in CIN 1, 2, 3
PAP smear report = Normal | Infect | Reactive

PAP smear report is ≥ ASCUS

1. ASCUS
2. (Refractory) (Confirmatory)
3. Repeat pap smear after colposcopy, sent pt for risk of HBV DNA

≥ ASCUS → +ve

Report is LSIL

Colposcopic directed biopsy (Ecc is preferred)

Report is HSIL

Colposcopic directed biopsy + Ecc is mandatory

Endocervical Curettage

Report is Cancer

Colposcopic directed biopsy & confirm the lesions.

Management of Colposcopy Report:

1. CIN-1: mild one

Risk of CIN-1 → Cancer is 1%

upto 70-80% of lesions regress spontaneously.
The avg. time for regression is 2 yrs.

- Conservative mx for a period of 2 yrs.
  - PAP smears every 6 monthly.
  - HPV DNA

If CIN-1 is persistent after 2 yrs:

↓

Rx the lesion

↓

Rx is ablation (destroy the lesion or organ)

- i.e. Cryoablation (CO₂ & H₂O) → it's open procedure

  mechanism how it destroy cells is: Crystallization of intracellular H₂O

Cryoablation: During procedure mild pain occur

- Long term complication is also persistent Hsy dys.
  - b) cervical stenosis
  - c) cervical incompetence

Cryotherapy doesn't cause bleeding

→ what % of LSIL convert into HSIL is 10%.

10% HSIL → Ca

2) CIN 2 / CIN 3: Colposcopy confirmed CIN 2 / CIN 3.

↓ 5% ↓ >12% (22%)

Ca Cx → Ca Cx

No role of conservative mx

We rely these decisions.
The Rx is excisional (Remove ab@ cells)

→ Remove T2 of cervix.

Procedure is LEEP/LEEP T2 → Rx of choice

Loop electrosurgical excisional → Large loop excision of T2

LEEP is both diagnostic & Therapeutic (Remove T2) Xsent for

(Take specimen)

→ Margins are not clearly seen by Histopathology.

→ OPD procedure

→ Very short time 2min

→ No training required.

→ New procedure.

CIN 2/ CIN 3

when LEEP is not there

Cone is done.

→ Both diagnostic & Therapeutic

→ Done in OT under anesthesia

→ Conization Indications : (Cone Rx)

Base of cone is T2.

I. a) Unsatisfactory (when entire T2 not visible) Colposcopy.

b) Entire lesion is not visible.

c)

II. When there is discrepancy b/w Cytology & HPE
3) When ECC is +ve in HSIL

4) When micro invasion is suspected

5) Of histology suspected the AdenoCa (glandular columnar cells)

A lady came to report CIN 2 in PAP

We do colposcope directed bx

A 42 yr old P 3 L3 Colposcopy done—CIN 3

Next step: LEEP

Best step:

40 yr old P 3 L 2 CIN-2—LEEP

Hysterectomy done if:

1) Recurrent CIN
2) Pt will not follow up
3) If histology is adeno & family is complete origin endometrium
4) Co-existing other pelvic pathologies

Cancer of Cx

Risk factors:

1) early age of 1st intercourse
2) early age of 1st child birth
3) multiple sexual partners
4) STD’s, HIV; genital herpes
5) low socioeconomic status
6) OCP’s (> 5 yr) minimally ↑ risk of Ca Cx

Notes:

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4) Smoking.

8) Pre-invasive lesion

MC Histology: Squ. cell Ca (40%)

Adeno (25%)

Others (5%)

Squamous cell Ca

Adeno

Smoking is Risk Fx

OCP's as Risk Fx

OCP (x) Smoking is not.

Causative agent:

1 \(\Rightarrow\) HPV:

High Risk: 16, 18, 31, 33, 35, 45, 52, 58

\(\Rightarrow\) Responsible for

70% of pathogenesis

Low Risk: 6, 11

\(\Rightarrow\) Cause genital warts

Caused by HPV

Women

Men

Ca Cx

Penile

Vagina

Anal

Vulva

Oral

HPV infect basal epithelial cells

HPV shows koilocytosis on histopathology

Perinuclear halo

Ballooning of the genital warts have

laryngeal papillomatosis.
MC Type 16 most sensitive.

Squamous Ca.

most specific → Type 18
  → adenoc Ca.

→ Viral protein → malignant transformation
  \[ p53 \leftarrow E_6 \mid E_7 \rightarrow Rb. \]

\[ E_1 \mid E_2 \] required for viral replication

R \(_v\) of HPV: (DNA virus)

Gardasil  Cervarix
  Quadrivalent  Bivalent
  now it protects against 9 16, 18.
  16, 18, 11 → Quadrivalent \(\rightarrow\) can't be given to boys.

act on inactivated protein capsid
  can be given at age 11-12 yrs
  can be given at age 09-26 yrs
  \(\rightarrow\) can be given to boys.
  Dose: 0.5 ml I/m
  0, 2, 6 months 0, 1, 6 months

S/E: Syncopeal attack. (After giving vaccine, we have to observe for 15 min)

Vaccine can't be given preg.
  HIV +ve all should receive vaccine. (From 26 yr old)

There is no \( R_v \) for preinvasive lesions.

After vaccine, screening should continue.
mc mode of spread :- lymphatic > direct hematogenous.

mc age group :- bimodal distribution

1st peak seen in 3rd - 4th decade

2nd peak seen 5th - 6th decade

also seen in post menopausal common.

mc complaint is irregular vaginal bleeding

most specific complaint :- post coital bleeding (PCB)

→ a 35 yr old woman come c h/o PCB next step is clinical examination & PAP smear

→ a 35 yr old woman come c h/o PCB on examination exophytic 3x4 cm mass evaginative growth on the ant. lip of C then we do is punch biopsy -> opa procedure

→ mc of death of Ca Cx is uremia

mc site of metastasis is lymphnode

mc site of metastasis of hematogenous spread is lung

mc LN to be involved is Obturator - 1st L1/2 sentinel

1st LN / sentinel LN -> paraaortic (right side we see hole)

Involvement of ovaries in Ca Cx:

risk < 0.5%

Ovaries are never removed in Ca Cx.

Staging of Ca Cx: Clinical Staging (FIGO)

some Tx can't be used to change the stage of Ca.

all. @ USG

jet CT -> used is allow to look for Changes of hydroureter

jet MRI

jet PET scan
All - Scopy can be done for stage < x-ray, bone scanning

Stage 1:
- microscopic
  1. 1 < 3mm deep < 7mm in horizontal span
  2. 3-5 cm deep < 7mm in horizontal span
- macroscopic
  1. 1 ≤ 4 cm (all micro > A1)
  2. > 4 cm

Stage 2 - involves upper 2/3rd of vagina.
- A: Cut involvement of Parametrium A < 4 cm
- B: Involvement of Parametrium

Stage 3A: Lower 1/3rd of vagina is involved
- 3a.: Tumor reaches the lat. pelvic wall (hydronephrotic
  on CT/IPN scan

Stage 4:
- A: Bladder & Rectum
- B: Distant Spread (if inguinal LN involvement occur
  it change the stage)

- Most imp. prognostic marker for Ca Cx is
  Stage > LN status (whether LN involved or not)

3 cm on ant. lip lower 1/3rd of vagina involved on
Cystoscopy Bullous edema of bladder mucosa.

doesn't change stage (not because of bladder involved)

It is due to lymphatic obstruct

so it 3A.


Stage $\leq 1b_{1}$: $\rightarrow 1^{o}$ - Surgery

$> 1b_{2}$ $\rightarrow 1^{o}$ - Chemoradiation

Stage $1A_{2}$ - If family is complete $\rightarrow$ do Simple Hysterectomy ($\times$)

- Simple extra-fascial Hysterectomy
- Type-I Hysterectomy

$\Rightarrow$ Risk of LN involvement is $< 1\%$ &

Stage 1 of family is not complete $\rightarrow$ do Conization ($\times$)

In case Lymphovascular Space Invasion (LVSI) we can't do Conisation we do Fertilization preserving surgery $\times$

Radical Trachelectomy

We remove cervix + parametrial tissue / Pelvic LN

a small part of vagina.

$\Rightarrow$ a cerclage is done b/w uterine & vagina.

$\Rightarrow$ 'C' secto is done in the patient in preg.

$\Rightarrow$ Stage $1A_{1}$, $1A_{2}$, $1B_{1}$ ($< 2cm$)

Stage $1A_{2}$ - If family is complete $\rightarrow$ is modified Radical Hysterectomy k/A Wertheim's Hysterectomy k/A Type-II Hysterectomy.

If family not complete: - Radical Trachelectomy.

Risk of LN involvement is $7\%$.

Along with Wertheim's hysterectomy + Pelvic Lymphadenectomy has to be done.

Stage $1B_{2}$ -

Family complete: - Radical hysterectomy k/A Melge Hysterectomy k/A Type-III Hysterectomy.

+ Pelvic lymphadenectomy + Para aortic LN sampling.
Family not completed = Radical Tracheectomy < 2 cm
If T4 beyond 2 cm uterus has to be 3 cm

→ All stages > IB2
1 modality is Chemoradiation (Concurrent Chemotherapy)

In chemotherapy: mc agent is Cisplatin > 5FU

These drugs also called as Radiation sensitizers.

Radiotherapy: External Beam Radiotherapy
Brachytherapy: Intracavitary RT

1st we give is EBRT → Brachytherapy
(Lower T4 load)

Radioisotope used in EBRT → Cesium
Brachytherapy → Cesium & Iridium

In EBRT Radiation given to Pelvis - 25# Fractions i.e.

5 fractions every week

\[ \text{#} = 1.8 - 2 \text{ Gy} \]

(4.5 - 50 Gy)

Extended Field RT: Pelvis + Paracervical LN

25 #

1 - 1.2 Gy

Q: Brachytherapy: Inside pelvis we give radiation at 2 points
Point A
Point B

2 cm lat. to uterosacral canal
3 cm lat. to point A (15 cm from canal) & 3 cm above uterosacral canal) & 3 cm above ext. os.

It corresponds to position of

It corresponds to Obturating LN

Radiation dose: 8000 cGy (80 Gy)
Radiation dose: 6000 cGy (60 Gy)
American Brachytherapy Society (ABS Therapy)

Early stage → 80-85 Gy

Point A

- Locally advanced → 85-90 Gy
- Beyond T2

Point B → Early stage → 50-55 Gy

(lat. pelvic wall point)

Locally advanced stage → 55-60 Gy

All in routine pelvic radiation therapy

Inguinal LN is not involved routinely.

Recurrent Ca Cx

1st modality of Therapy is → Give Chemo/Radation for recurrence

a) Sx

b) Chemoradiation → Sx (operable) i.e. total pelvic exenteration

Inoperable → Chemotherapy (palliative)

(due to fibrosis after operation)

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Ovarian Cancer

→ Most cases are sporadic.

→ Presents no non-specific Sx/S.

So there is no universal screening.

→ When there is an adrenal mass + rise suspicion of malignancy.

On Hx & examination then we can tell benign or malignant.

Adrenal mass

Benign masses → malignant masses

Seen in reproductive age extremes of age woman

CI of pain: no pain

Ultrasound: tender → non-tender

Consistency: cystic → variable/heterogeneous.

LOC: transvaginal USG tell b/w adrenal & ovarian mass

On USG: unilocular completely benign

Anechoic

High risk features on USG

1. Anechoic contain solid components

2. Presence of papillary projections

3. Presence of thick septae (2-3 mm thick)

4. C. Ascites

5. Enlargement of LN & marked bowel loops

When we mark Sx + of mass:

Sx of mass (laparoscopy/ laparotomy).

1) Any ovarian mass where which show high risk
features on USG:

1) Any ovarian mass > 4 cm
   
   Adnexal mass > 10 cm

2) Raised CA-125 in postmenopausal women:
   
   $\text{CA-125} < 35$ is $\text{N}$(1)

   CA-125 not used in reproductive age group.

   In reproductive age group CA-125 > 200 $\text{is}$ indicative of a probable lying malignancy.

3) Masses $\oplus$ to acute abdomen either they undergo torsion or rupture.

4) For $\Delta$s (because we don't know origin of malignancy)

   - Reproductive age - Cystic masses
     
     a) 3 cm - 5 cm $\rightarrow$ wait & watch (mostly Follicular cysts)

   b) 5 - 5 cm $\rightarrow$ serial USG on follow

   3, 6, 12

   CA-125 is not a 1st line investigation for masses.

   In reproductive age women.

   In 55 yr old post menopausal women 5 cm benign cystic mass in U/L ovary

   First Ix: USG / CA 125

   For 3 - 5 cm wait & watch.

   OCP's are not to be given routinely for all ovarian masses.

   prevents appearance of new ovarian mass.
6) BRC A 1 | BRC A 1 | MNPC C

7) Post menopausal Hormone Replacement Therapy

Smoking is only a risk factor for mucinous Ca.

Protective Factors:

1) Multiparity
2) Anovulation (no estrogens)
3) Salpingectomy
4) Tubal ligation
5) Hysterectomy

6) OCP’s reduce ovarian Ca risk by 50%.

7) Breast feeding

Types of Ovarian Ca.

1) Epithelial
2) Germ cell
3) Sex cord

90% of all cancers

5-8%

3%

Epithelial Ovarian Cancers: mostly B/L

Serous histology (70-75%) - mc

Mucinous histology (10-15%) → tends to be unilateral

Endometroid

Brenners

Clear cell

Rest:

Mucinous:

1) Unlike serous, mucinous confined to ovary

for long time; so better prognosis.

2) Grow up to large size = 20 cm

3) CA 125 not markedly raised, it is

mildly raised.

4) CA 125 pseudomyxoma peritonei mc at asc

appendical.
Endometroid: It has highest association with existing Endometriosis.

Brenner's Tumor: It has transitional epithelium.

Clear cell Tumor: As in endometriosis.

The peak age incidence for epithelial ovarian Ca is 60 yrs (6th-7th decade).

Nonspecific symptoms:-
- IBS
- Early satiety
- Loss of appetite
- Abdominal conditions like bloating, belching
- Abdominal distension
- Used in late stages
  Among all gynecologic it has highest mortality rate

Most routes of spread - Tumor exfoliation.

- Most are sporadic
- 10% are familial associated with genes BRCA1, BRCA2, HRPC

Familial:
- They occur a decade earlier

Helpful mutations: We advice Risk reducing surgery i.e.

Prophylactic Bilateral Salpingoophorectomy

Ideal age is 35 yrs as soon as family is complete.

Because after 35 yrs, risk ↑

By how much is the risk of breast & ovarian Ca
Reduced:

Ovarian Ca reduced risk by 80%.
Breast Ca reduced risk by 50%.

Of pt. not willing for prophylactic BSO then
a) Prescribe OCP's + screen her.
   Screening is done in pts:
   a) who have less BRCA1/BRCA2/HNPCC.
   b) Family H/o Breast / Ovarian Ca.

[For sporadic case - CA H/o + in young age group & clustering group.]

Screening by 35 yrs age.

d) TVS + CA 125
   every 6 or 12 monthly

Germ Cell

Contribute 5 - 8% of all ovarian Ca.

Peak age incidence 10 - 20 yrs (10 - 30 yrs)

40% of all ovarian neoplasms in 10 - 30 yrs are germ cell Ca.

1) Teratoma: / Dermoid : - Immature teratoma

   mature cystic teratoma.

2) Dysgerminoma.

3) Embryonal All Ca.

4) Endometrial sinus Tumor (Yolk Sac tumors)

5) Chorion.

4) mixed.

Germ cells is U/L (majority)

It present with nonspecific S/Sx + precocious puberty + acu

mostly Produce HCG.
abdomen (rapidly growing)

- Younger girls are mostly used in early stages.
- They are conservatively managed by $S_X$.

Sex chord.

Contribute 3%.

Peak age incidence is in $\rightarrow$ Perimenopausal women

- Granulosa cell tumor (GCT)
- Theca cell tumor
- Sertoli-Leydig cell tumor
- Fibroma
- Fibroma thicoma

$\rightarrow$ Presents $\&$ nonspecific S/S, + Abdominal bleeding + visualiz$^n$

$\rightarrow$ Estrogen (Testosterone seen)

$\rightarrow$ Visualizing tumor of ovary $\rightarrow$ Artenoblast

$\rightarrow$ Used in early stage

$\rightarrow$ Best prognosis

$\rightarrow$ Metastatize late

$\rightarrow$ LN spread is rare

$\rightarrow$ GCT $\rightarrow$ usu $\&$ endometrial Ca.

- MC ovarian tumor $\rightarrow$ Serous cyst adenoma.
- MC ovarian Ca $\rightarrow$ Serous cyst adenoc.
- MC ovarian tumor in preg $\rightarrow$ Dermoid.
- MC ovarian malignancy in preg $\rightarrow$ Dysgerminoma.
- MC Germ cell tumor $:\rightarrow$ Immature $\rightarrow$ Dysgerminoma $\rightarrow$ Teratoma.

Germ cell tumor $\&$ best prognosis $\rightarrow$ Dysgerminoma.
Germ Cell Tumor with
Best prognosis → Dysgerminoma
Worst prognosis → Yolk Sac Tumor

Germ Cell Tumor in acute abdomen → Yolk Sac Tumor
Which Germ Cell Tumor is mc b/l → Dysgerminoma

Risk of b/l in dysgerminoma → 10–15%
B/l in dermoid → Upto 10% (<10%)
Risk of malignancy in dermoid → 0.2–2%
Malignancy → squamous cell ca.

Tumor of ovary present C metastasizes to other ovary → GCT
Which metastasizes to other ovary → GCT

Meig’s Syndrome: Fibroma + Ascites + Pleural effusion
(Rt >4)

Pseudo Meig’s syndrome: Tumor other than Fibroma + Ascites + Pleural effusion
C highest mulleran anomalies: dysgerminoma
Dysgerminoma: Seminoma

Tumor markers

Serous → CA 125
Human epididymal 4 (HE4) (non-specific)
Mucinous → CEA
Dysgerminoma → LDH (PLAP → AFP
HCG +)
GCT → Inhibin

Embryonal Sinus T → ACG + AFP
Endodermal Sinus T → AFP + LDH (not HCG)
Dermoid → no tumor marker
rarely it can secrete HCG

Fibroma → no tumor marker
Chorio Ca → HCG

On HPE
Pseudoma Bodies → Serous:
Cell exner Bodies → granulosa cell tumor
Wallersh cell nest → Brenners
Reinke’s Crystal → Leydeg / hilus cell tumor
Schiller Duval Bodies → endodermal sinus tumor

Rokitansky Protuberance → Dermoid
Signet Ring → Krukenberg

Krukenbergs:
- 2° to ovary
- MC 1° → stomach Ca.

MC route → retrograde lymphatic
almost always 3/1:
capsule of ovary is intact.

Staging of Ovary Ca (FIGO)
Surgical Staging

Stage 1:
A → Tumor confined U/L ovary
B → B/L ovary
C 1 → surgical spill
2 → pre-operative capsule rupture
3 → malignant ascites.

Stage 2: A → extension to tuber / uterus
B → extension to other pelvic structures (pelvic LN)

Stage 3: A 1 → deep retroperitoneal LN (para adnexal LN)
A₀ → microscopic extra pelvic peritoneal deposits (only on peritoneum)
B → macroscopic peritoneal deposits < 2cm & capsule of liver intact
C → macroscopic peritoneal deposits > 2cm

Stage 4:
A → malignant pleural effusion
B → Parenchymal spread to an organ (abdominal) → extraabdominal spread → Inguinal LN.

Procedure for all above is: Staging laparotomy.

Staging Laparotomy:
1) Midline vertical incision (always for malignancy)
2) Ascitic fluid sampling
   Saline wash (50-100 ml) of normal saline if ascitic fluid not present
3) Inspection & palpation of all abdominal organs
4) Random peritoneal biopsies:
   Site: Para colic gutters
   POD / hidden areas
   Surface of Diaphragm (scrapings)
5) Total Abdominal Hysterectomy + BSO
   Type II (simple extraperitoneal hysterectomy)
6) Infracolic Omentectomy (omentum is cut that is hanging from Transverse colon)
7) Pelvic & paraaortic LN sampling
8) Closure

Additional step you do if etiology is mucinous is appendicectomy.
2) Stage 3 & 4 are advanced stage.
   By far, advanced stage is
   as 1st therapy Sx – To reduce tumor load.
   Sx is debulking Sx / cytoreductive Sx

3) Best Tx for suspected ovarian Ca is CT Scan.
   CT scan is used to [see operability] spread but not
   staging.

4) A pt. Ca ovary was operated what was follow up?
   if Ca 125 raised → PET Scan is best. T
   Post Operative Therapy:
   For epithelial all stages except stage 1a & 1b grade I need
   post op Chemotherapy
   Carboplatin + Paclitaxel (for advanced stages)  
   Cisplatin + Paclitaxel (for early stages)
   6 cycles of chemotherapy intravenously

   For sex chord / Germ cell tumors → late stage given chemo.
   ↓
   even early stages Chemotherapy: BEP

   need post op chemo
   Biomycin, etoposide, cisplatin.

In Ca ovary, fertility preserving Sx is unilateral salpingo oophorectomy
   [cystectomy is done in benign]

Fertility preserving Sx indications:
1. If pt has family incomplete stage 1a (Only one ovary involved)
2. Germ cell tumors (because they are UI, can’t be seen in young females)
3. Borderline epithelial tumors k/a epithelial ovarian Ca
   low malignant potential

Borderline epithelial tumors – very good prognosis
   lack stromal invasion, a decade earlier, occur UI
Poorest prognostic markers:-
- Abdominal spread
- Lymph node spread
- Capsule rupture
- Intraoperative spill

**Endometrial Cancer**

Main histology: Endometroid adenocarcinoma.
Others are Papillary serous / clear cell.

Based on histology we have classification:

<table>
<thead>
<tr>
<th>Type 1</th>
<th>Type 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endometroid</td>
<td>Endometrial grade 3</td>
</tr>
<tr>
<td>Grade 1, 2</td>
<td>Papillary / serous clear cell</td>
</tr>
</tbody>
</table>

80% Estrogen responsive

- As it is preinvasive lesion
- Better prognosis

- Seen in obese women
- Early menopause &
- Postmenopausal genes are PTEN / Kras
- Age.

20% Estrogen non-responsive

- No preinvasive lesion
- Poor prognosis

- Clear cell - worst prognosis
- Thin women

Peak age incidence: 60 yrs (6th – 7th decade)

Main symptoms - Irregular bleeding / Post-menopausal bleeding (PMB)

Most specific -> PMB
mcc of PMB in general - sterile endometrial atrophy

mcc of PMB in India → Ca Cx

Q: What % of PMB pts have endometrial Ca - 10%

Common Route of Spread → Direct

mcc of pyometria (collection of pus in uterus) → Ca endometrium

mcc of pyometria in India → Ca Cx

Risk Factors

more no. of menstrual cycle → more risk.

① not followed by ② it is unopposed ②

1. Early menarche
2. Late menopause
3. Nulliparity
4. Obesity
5. DM
6. HTN
7. PCOS
8. Genes BRCA1, BRCA2, HPV etc.

Tamoxifen has no genital uroinary agonistic action

Protective Factors:
1. OCP’s: - 60% protective
2. Multiparity
3. Smoking is protective (break down of ②)
4. Phytoestrogen - bind to ② but don't make act
5. Physical activity
6. Coffee - ① Green Tea
Aasis of Endometrial Ca:

No universal Screening.

⇒ Pt of HNPCC has a risk of endometrial Ca is up to 75%

In these patients, Screening is done.

In 35 yrs of age:

Endometrial Sampling is done.

Fractional Curetage is best method for HNPCC pts.

Done every 6 mon or 12 mon.

⇒ Once the pt. come to PMB rule out Endometrial Ca.

1st Ix is:-

1) TVS - Look for endometrial thickness

If thickness ≤ 4mm → risk of malignancy negligible

> 5mm → have a risk of endo Ca.

Iox: Endometrial Biopsy (minimally Invasive)

→ done in OPD under anesthesia

→ Done by using

a) Pipelle Technique  b) Vabra aspirator  c) Karman's Cannula

Used in India.

Aspira^n is done so it is the Endometrial aspira^n Biopsy.

Gold standard: - Fractional Curetage

For PMB - done in OPD under anesthesia.

FC done in 1) Cervical Stenosis

4) Benign report but persistent

Symptoms. (Inadequate tissue & no endometrial cells not seen)

3) Indecisive report

4) Atypical hyperplasia
1) PAP's
2) Endocervical Curettage
3) Dilate, OS
4) Endometrial Curettage

→ Hysteroscopy → not done.
→ PAPs - Poor IV to 1/10 Endometrial Ca.

show @ report in 50% Endometrial Ca. patients

Q: For PNB: - PAPs + TVS → EBx → FC.

On PAPs → AUS?
2) adenOCa (help to suspect Ca

AUB: - abnorm uterine bleeding
> 80 ml
> 7 day
> 21 days

No anatomical, systemic or endocrine cause then?

table less as "DUB"

"DUB"

usually @ in perimenopausal women or adolescent

80% → anovulatory

Any lady > 45 yrs → ab norm bleeding
1st thing r/o Endometrial Ca.

Only 10% of Endometrial Cancers occur in less than 45 yr old.
45 yr old & 2 menstrual bleeding - x 6 mon

Next step is endometrial sampling.

Beyond 45 yr - we have to do endometrial sampling.

**DUB:**

It is called only when other pathologies are ruled out.

For in adolescent DUB - Combined OCPs

(mod-severe)

Minimal → menstrual calendar

All are 1st line Rx for an adolescent ovulatory DUB except:

a) Methylene Blue (least)

b) Tranexamic acid

c) E alone not 3rd therapy or d) E + P

d) E + P (OCP's)

E alone reserved for girls who presents 2 unstable bleeding.

MC histological pattern: proliferative endometrium

Premenopausal DUB

Pt. present 2

Acute blood loss

Chronic blood loss

(>10 days post-beding)

 unstable vitals

Stable vitals

BT + D & C > E, only give if STOP bleeding in 24 hrs then

Endometrium is thin

Put pt. on OCPs for regular cycles x 3 mo

Progestrone

[Progesterone] 1st line, OCPs
metropathia hemorrhaegica:
- Type of OVB, age group = 40-45 yrs.
- Anovulatory
- 8 weeks of amenorrhea & heavy bleeding
- Bleeding has painless due to anovulatory
- On assessment pt. has thick polypoidal endometrium.
- It has pattern: swiss cheese pattern
  classically secretory endometrium absent.
  Ovaries will show cysts U/L 8/L.
- Lp.: Progesterone (MPA)
- In OVB, break through bleeding Estrogen Q.
- Endometrial Ca:
  There will be preinvasive lesions → Endometrial Hypoplasia
  4 Types:
  1) Simple or atypia . 1%.
  2) Complex or atypia . 3%.
  3) Simple or atypia . 8%.
  4) Complex or atypia . 99%.
  Simple = Both glands & stroma are proliferating
  Complex = Glands proliferate > stroma
  crowding glands
  Back to Back phenomenon

  → Rx of Hyperplasia or atypia:
  → Progesterone Therapy (MPA) for 6 month
  Repeat sample for Endometrial Ca: Biopsy report

  Rx O.C. of Hyperplasia or atypia: - Hysterectomy
  2nd line therapy: (Q)
  But preferred one is megestrol acetate
Staging FIGO

Surgical Staging.

Stage 1:

A: only endometrium is involved
   < 50% of myometrium is involved

B: > 50% of myometrium is involved

Stage 2: Cervical spread (if glands are involved it is not Cervical Spread)

Cervical stroma is involved

Stage 3:

A: Serosa and/or adnexa.

B: vagina and/or parametrium

C₁: the pelvic LN.

C₂: the para-aortic LN.

Stage 4:

A: bladder & bowel involvement

B: distant spread

Inguinal LN.

Procedure: Staging laparotomy.

Here routine omentectomy is not done.

Omentectomy is done if there is type 2.

TAH + BSO [Cervical Spread] Cervix spread to endometrium later.

L: Radical Type 3 hysterectomy

LN adenectomy → if histology is type 2

what is Q for advanced stage: -(3ly): -Debulking Sx
Post Op Therapy: Divide pt's into

Low risk
Intermediate risk
High risk

Histology: Endometriod
Any stage that is not type I (Papillary, etc)
Grade I & II fit into low risk
Stage 3 & 4 = high risk
Only endometrium involved
Endometriod grade 3

Rx: No further Rx
Radiotherapy
Both Radio & Chemo

Rx for recurrence:
1st Rx is local Rx

Rx: Radiotherapy

If pt is inoperable
To reduce viral load
If we can't give local therapy then
and is Chemotherapy (Palliative)

If ER+ve, PR+ve → Good prognostic factor
If recurrence occur in ER & PR+ve pts
Hormonal therapy is given

Contraception

High dose: > 500 pgm Estro Ethinylestradiol
Low dose: < 50 pgm nore 3.5 pgm
Very low dose: ≤ 20 pgm
Lowest possible: 10 pgm
Micro pill (OCP)

- 0.03 mg Ethynyl Estradiol
- 0.15 mg LNG

Both of them contain Salt- Ferrous Fumarate

Main MOA OCP: Inhibition of Ovulation

MC SIE: Break through bleeding (progesterone)

Relation of OCP to various CA:

OCP's to CA CX \rightarrow \uparrow risk of adenocar
Breast CX \rightarrow \text{No} \uparrow \text{risk}

LG SECO
Ovarian CX \rightarrow \downarrow 50%.
Endometrial CX \rightarrow \downarrow 60%.
Colon CX \rightarrow \downarrow \text{risk}
Hepatocellular CA \rightarrow \text{No} \uparrow \text{Risk}
Adenoma (Benign TV) \rightarrow \uparrow \text{risk}

(Gall bladder GB cancer \rightarrow \text{No} \uparrow \text{risk}

If miss 1 pill \rightarrow advise 2 pills as soon as possible

> 2 pills \rightarrow Back up contraceptives
Return of fertility \rightarrow 2 to 3 months (90 days)

OCP's in PID: \uparrow (chlamydia & candida)

\& ectopic: \downarrow,

Infections there is a slight \uparrow

Absolute C/I of OCP's (WHO Cat. IV)

1) Known case of CAD
2) VTE
3) uncontrolled HTN > 160/110
4) DM 2 vasculopathy
5) migraine 2 aura 1 focal neurological deficit
6) k/c/p breast Ca
7) preg.
8) undiagnosed vaginal bleeding
9) age > 35 yr + smoker
10) acute liver disease (acute viral hepatitis)

NSAID — Sabeli [CDRI Lucknow]

Centchroman — active ingredient Ormeloxofine

30 mg tab.

MOA: 2 by implantation
Endometrium out of phase.

Has to be taken twice a week for 1st 3 wks

→ there 1 pill/week.

S/E — delay of menstrual cycle.

Minipill

Minipill

Progestrone only pill

Cerazette: 0.075 mg desogestrel

MOA: 2 of ovulation because it has longer half-life

Minipill MOA — alteration of cervical mucus.

Minipill best in

Breast Feeding women (contracept of choice)

COC — immediately started

WHO — 6 weeks

mc S/E of minipill is → irregular vaginal bleeding

(UB)
absolute C/I for minipill (Cat 4):
- HCJ/o suspected preg.
- Undiagnosed vaginal bleeding.
- Current breast cancer

**DMPA**
- 150mg I/m inj. every 3 months
- max: 10 of ovulation (because dose is very high)

**2 Benefits**
- Reduces sickling
- Reduce sickle cell anemia

**2 Drawbacks**
- 1) Cause osteopenia by ↓ bone density
- 2) Delay in return of period

- Max. delay: 18 mo - avg 12 mo

- Epilepsy by raising seizure threshold

**Implants**

- **Implanon**
  - Single rod
  - 68 mg etonogestrel
  - 81C, 67 μg/day rod releases
  - Once implanted it is effective for 3 yrs.
  - Both insertion & removal are OPD procedures
  - Implants have least failure rate (0.05%)
  - Most effective method among all contraceptives

**Norplant**
- Fan-shaped manner
  - In upper arm model: medial aspect
  - Each rod has 26 mg of LNG
  - Each rod: has a 3cm - 4cm
  - It was effective for a period of 5 yrs


→ mc s/e → irregular VB
absolute c/i: cat 4:
moA → Φ ovulation

⇒ Vaginal Ring: NuvaRing

Composition: EE + Estrogestrel
release: 15 μg/m 120 μg/1 day
moA: Φ ovulation
Put in vagina for a period of 13 weeks
During 1 week ring is free (due to withdrawal bleed & insert new ring)
No systemic s/e of ring

Intrauterine Device
IUO's

CuT 380 A Para
guard
380 → surface area
A → atm8

MIRENA LNG 20
52 mg LNG
release rate @ 20 μg/1/day

○ bead prevents cervical perforation → Radiopaque
○ string → check & removal → It reduces menstrual blood loss

⇒ Free of cost
⇒ Radioopaque
⇒ Base
⇒ 10 yrs ≤ t½
⇒ @ 50 μg/1 day = release rate
⇒ bid loss 80ml
⇒ mc s/e → bleeding
CUT-380 A

MIRENA

mcc → removal is painful

Expulsion rate 16%

It is an emergency contraceptive

CUT device is at risk of PID only in 1st 20 days post insert

Medical complications of contraceptives

Contraception of choice

- Heart disease → Barrier IUD
- DM → barrier
- HIV +ve → IUD

Barrier

most used by couples in India

Safer → Barrier

Sickle cell anemia pt's

Nerdy married couple K living far away

Ideal of choice:

vasectomy > sterilization > IUD

cast

most effective
doesn't ↑ risk of infect

method of contracept

polyfilament (+ infect)

Now

monofilament

AHA

IUD for heart disease you don't need prophylactic antibiotics
HIV → IUD / Barrier to prevent transmission

Intrauterine device (IUD) is the choice of contraception.

HIV patients who are on ART we give OCPs.

MISSING IUD

IUC → USG

If the patient conceives →

1. Simple remove IUD
2. Remove IUD do MTP
3. Leave the IUD in situ
   - If thread of IUD visible. Remove IUD because it is teratogenic & infective?

After removal of IUD & USG to see the location of pregnancy.

Previous ectopic is not a contra indication for IUD use.

(I'm not sure what this means)

Absolute C/I for IUD use:

a) Suspected preg
b) Current pelvic infection (TA, endometriosis, PID) not HIV infection
c) Major uterine malformations
d) Wilson's disease (MIRENA) but cut C/I
   - Breast Ca (CUT) safe but not MIRENA C/I
f) Undiagnosed vaginal bleeding.

Post placental insertion:

When IUD is introduced within 10 min of placental expulsion.
EC don't act after implantation? Anything that acts after implantation is abortifacient.

Q: LNG Tab: 2 Tab 0.75 mg LNG as EC. Both Tab together no side, no risk.

5. Can OCP's used as EC? Yes. High dose OCP's used as EC.

100 mg EE + 0.5 mg LNG. Repeat after 12 hrs.

(Option) 200 mg EE + 1 mg LNG - used as EC.

6. Centromed: is approved as EC. 50 mg - 12 hrs apart.

7. Mifepristone → 10-50 mg.

8. Ulipristal → 20 mg single dose.

Most side effects of EC is nausea & vomiting.

No C/I.

Sterilization

- Non Reversible method

- Child norm → 1 child 1 year old

- The couple can go sterilization

- Consent of spouse is not mandatory

- MC method used for sterilization: laparoscopic sterilization.

- Other: Postpartum sterilization

- Done 2-48 hrs of delivery

- Can be done up to 7 days

Interval sterilization: at 6 wks (42 days)
Breast Feeding women

1. Pop

2. IUD - ↑ of perforation (when child sucks → oxytocin release → Intrauterine uterus).

   IUD can be put in 4 wks after delivery if IUD is not put immediately after delivery.

Emergency Contraceptives / Intercutives

most effective → cut

most effective among Hormonal

- ulipristal > mifepristone > LNG Tab

- MC used: LNG Tab

1. LNG IUD: Is not an Emergency Contraceptive (EC)

2. Misoprostol is never EC it is abortifacient.

3. Pop is not an EC dose is 0.75 mg.

4. LNG Tablet = 0.75 mg is an EC.

EC is effective up to 5 days or 120 hrs.

They are most effective if they are used 2 in 72 hrs.

EC

- moA

   - Hormonal

   ↓

   (Fertilization)

   - Non hormonal IUD

   ↓

Fertilization > Inhibit Implantation

Jain Stationery
09654691387
mc method for post partum sterilization is mini laparotomy.

Size is very small 3cm.

mc method used: Modified Pomeroy/Parkland method.

Pago ➔ Pouch that ligates isthmus

Pomeroy ➔ specimen should sent to histopath.

Modified
Pomeroy

FT damaged is 3cm

For post partum sterilization: we didn't use laparoscopy.

In heart disease pt. ➔ Avoid 24 hrs once she settled

we can do sterilization at same time

laparoscopy

Laparoscopy is used for interval sterilization

we can do D_5 ➔ D_11

can used is CO_2 > N_2O

usual press: around 8-12 mmHg

volume is 2L

max. force generated is 15 mmHg

max. press generated is 15 mmHg.

avg is 10-12 mmHg

No absolute contraindication

Relative contraindication:

1) Massive Obesity

2) Severe Cardiac disease

3) Severe Pulmonary disease
**Intra abdominal adhesions**

**Most effective methods:**

1. Post-partum sterilization
   - minilap (modified Pomeroy)
   - Unipolar not used due to causes death.

   **Least effective:**
   - Halka Clips
     - 2nd least: Bipolar Cautery

   **Risk of ectopic → cauterization:**
   - Halka Clips > fallopian tube > modified Pomeroy > cauterization

   **Ligation:**
   - Most effective:
     - Tubal ligation > modified Pomeroy

   **Failure rates**

   - Life table analysis > Pearl index

**Highly effective Contraceptives**

- Perfect use rate
- Typical use rate

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>Perfect Use Rate</th>
<th>Typical Use Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Implant</td>
<td>0.05%</td>
<td>0.05%</td>
</tr>
<tr>
<td>Sterilization</td>
<td>M 0.1%</td>
<td>F 0.5%</td>
</tr>
<tr>
<td>TUB: mirena</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Cu I</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>IUCD</td>
<td>0.3%</td>
<td>8.4% - 9%</td>
</tr>
<tr>
<td>Vaginal ring</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Asp's DMPA  0.3  0.3

Diaphragm  6%  12%

Sponge (today)
  a) Parous  20%  24%
  b) Nulliparous  9%  12%

Condom  male  2%  18%
          Female  5%  21%

If they are least effective  then based on perfect use
otherwise, not mention  typical use.

Hydrosopic sterilant
called as Essure
introduced hydrosopic fertility in proximal part of
FT  
intramural part.

Outer coil  -> made of nitinol (alloy of nickel & Titanium)

Inner coil  -> Stainless steel

Very effective
Causes foreign body reaction  -> heals by fibrosis
Taken time  -> ~ 3 month

Confirm by HSG  after 3 month
1st 3 month  pt. has to use back up method
of sterilant

on HSG  if fibrosis has occurred

Certificate of ey/ sterilant

Also done in vasectomy
## Vaginitis

**Bacterial vaginitis > Candida > Trichomonas**

<table>
<thead>
<tr>
<th>Causative agent</th>
<th>Gardenella vaginalis</th>
<th>C. albicans</th>
<th>T. vaginalis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not an STD</td>
<td>Not an STD</td>
<td>Yes STD</td>
<td></td>
</tr>
<tr>
<td>Vaginal pH</td>
<td>&gt; 4.5</td>
<td>&lt; 4.5</td>
<td>5-6</td>
</tr>
</tbody>
</table>

**mc complex**
- Foul smelling
- Pruritis +
- White discharge
- Curdy white
- Dysuria +
- Dyspareunia
- Greenish discharge

** IOC**
- saline
- microscopy
- clue cells
- pseudohyphal
- epithelial cells
- bacteria in piggy back

**Gold Standard**
- Gram stain
- culture
- culture
- Nugent score
- Sabouraud's media
- diaton's media
- we find cocobacilli
- of 7-10 Confirmatory
- culture has no role.

**Amine Test**
- +ve
- -ve
- +ve

**10% KOH**
- amine odour
- metronidazole
- 150mg of Fluconazole
- metronidazole
- in pregnancy also
- topical azole like
- Cotrimoxazole

**Rx of Partner**
- If the partner is Rx, he is symptomatic.
- Rx if he is symptomatic.
MC PID: Chlamydia:
Fitz Hugh Curtis Syndrome: perihepatitis & inflammation of capsule
upper Abd. Rib side pain
Anorectalitis

MC with Chlamydia:
Cervicitis & as acute PID.
Chlamydia → Subacute

Acute PID:
Lower Abd. pain
Uterine tenderness
Adnexal tenderness
Cervical motion tenderness

Gold Standard for diagnosis → Laparoscopy

Tx: Confirmatory:
1. Biopsy taken from FT
2. Inc. [Gold Standard → Laparoscopy]
3. EQ → Plasma. All Endometriosis → Confirmatory
4. USG.

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