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Term cell differentiation.

What determines sex in fetus : SRY gene on short arm, chromosome Y.

Others :

If SRY gene is present.

↓

Gonads = Testis (7 wks).

(Gonads develop from Genital ridge)

Sertoli cells

↓

Mullerian inhibiting factor

(A glycoprotein, secreted at 7wks)

It has paracrine action.
ie it acts locally.

↓

Leads to ipsilateral regression of Mullerian ducts in males by 9 wks.

↓

Remanants of Mullerian duct in males : Appendix of Testis
Prostatic utricle

* Appendix of Testis
≃ Hydatid of Morgagni.

Leydig cells

↓

Testosterone.

(Its production begins at 9wks (intra-uterine).
Maxm produc^n @ 14-18 wks

5α-reductase

Promotes the growth of wolffian duct in males

↓

Int. Genitalia

S - Seminal vesicle
E - Ejaculatory duct
E - Epididymis
V - Vas deferens

Formn of DHT

↓

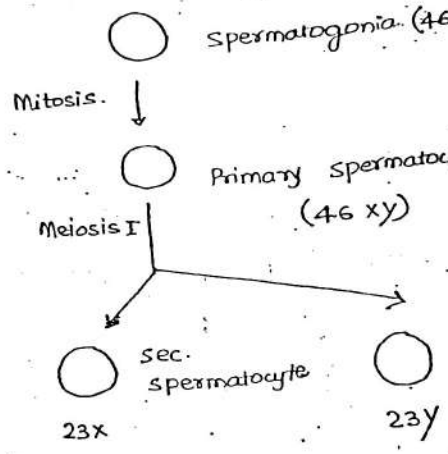
Ext. genitalia in males.

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21. Cells which lie outside Blood testis barrier.

Spermatogenesis.



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Imp. points on spermatogenesis :

- Begins at puberty.
- Time taken : 72-75 days.
- one spermatogonia forms - 16 primary spermatocyte.
- one spermatocyte gives rise to 4 sperms/ spermatids.
- One spermatogonia gives rise to 64 sperms/ spermatids.

Spermiogenesis :-

- Spermatids change to sperms.
- No mitosis / No meiosis
- Time taken : 14 days.

Part of spermatid.		Part in sperm.
Nuclear material	—	Head of sperm.
Golgi body	—	Acrosomal cap.
Mitochondria	—	Middle piece.
Microtubules	—	Tail / axial filament.

* sperms lack ER (especially RER).

Sperms.

- 55 µm.
- Life span - 72 hrs.
- * sperms attain motility & maturity in - Cranial end of epididymis.

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Hormonal support of spermatogenesis :

Hypothalamus
↓
GnRH.
↓
Ant. pituitary

* 1st stimulus for leydig cells to produce testosterone : HCG.

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Sperm pathway -

Spermatogenesis occur @ seminiferous tubules of Testis. (Sertoli cells)

↓

Motility & maturity @ Epididymis (cranial end).

↓

Vas deferens.

↓

They are released along with seminal vesicle fluid. (60% volm + Fructose).

+ Prostatic fluid. + Bulbourethral gland secretion.

↓

The semen which is released should liquify in 20-30 minutes.
(Liquifacn d/t : prostatic fluid)

Ideally semen analysis is done on liquified semen.
But if no liquifcn occurs in 60 mins : semen analysis on unliquified semen.

Note : If there is azoospermia with low semen volume & absent fructose → Block is below the level of seminal vesicle.

Men c congenital absence of vas def., suffer c seminal vesicle agenesis (CAVD)
They have low semen volume, low PH & low fructose.
Spermatogenesis is, however, normal.

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* Absence of spermatozoa } in semen.
  But presence of Fructose }

  Likely diagnosis may be : Mumps orchitis.

Capacitation :

- Ability of sperms to fertilize ova
- Begins in female reproductive tract (cervix).
- Major part occurs in Fallopian tube.
- Time taken : 7 hours
- After capacitation, sperms become hyper-motile.

Acrosomal rxn :

- occurs after the sperm binds to zona pellucida
- Zona pellucida has sperm receptors : ZP_1, ZP_2 & ZP_3, which mediate acrosomal rxn (Main : ZP_3).
- Acrosomal rxn occurs because enzymes like hylauronidase, acrosin, etc. are released.

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In females

SRY gene Absent
- Whether ovary is Present/absent depends on Y chromosome.
- For dev. of ovary 2X chromosomes are needed.

↓

Gonads = ovary.

Sertoli cells absent
↓
Mullerian I.F. absent
↓
Mullerian duct grows into
- Fallopian tube
- Uterus
- Cervix
- Upper 3/5th Vagina.

Leydig cells absent
↓
Testosterone absent
- Wolffian duct regresses
- Ext. genitalia looks like female.

Lower part of vagina develops from urogenital sinus (sino-vaginal bulb)

Most common cause of ambiguous female genitalia : Presence of testosterone in intra-uterine life [congenital adrenal hyperplasia]

Remnants of wolffian ducts in females :-

Part of Wolffian duct		Remnants in females.
1. Pronephros	— forms —	Hydatid of Morgagni/Kobelt tubercle.
2. Mesonephros.		
• Cranial end.	———	Epo-ophoron/organ of Rosenmuller
• Caudal end.	———	Para-oophoron.
3. Wolfian duct proper	———	Gartner's duct

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All remnants of wolffian duct is present in lateral part of broad ligament, except paraoophron, which is present in medial part of broad ligament.

* Wolffian duct ≅ Mesonephric duct
Mullerian duct ≅ Para-mesonephric duct.

* organ of Rossenmuller :
- consists Vertical tubules. (Not Horizontal tubules)
- Lined by cuboid epithelia (Not by columnar epithelia).

Oogenesis.

(46 XX) oogonia
↓ MITOSIS.
(46 XX) — Primary oocyte.
↓ MEIOSIS I : arrested in Diplotene (Prophase)
* This arrested stage is known as Dictyate stage. (upto puberty)
↳ This stage is absent in spermatogenesis.

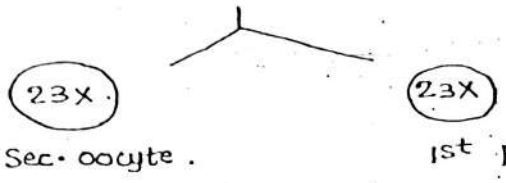
At puberty, Meiosis I is resumed.
Meiosis is hormone dependent. → LH
∴ Meiosis I is resumed 24-36 hours before ovulation.

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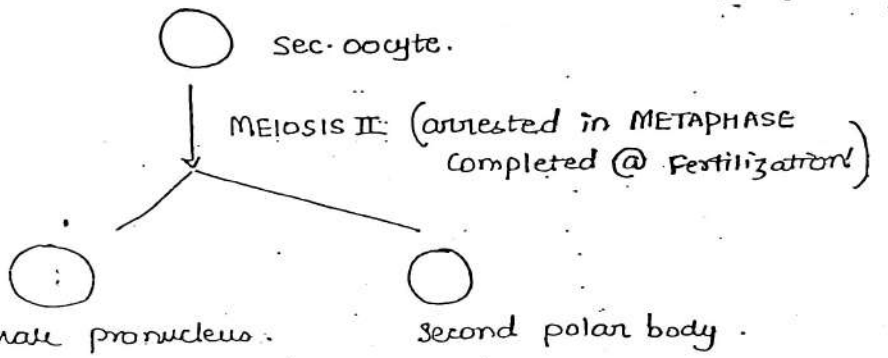
At puberty :

The arrest is over.



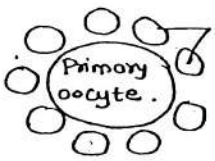
Sec. oocyte. 1st polar body.

- Release of sec. oocyte from primary is k/a ovulation.



Female pronucleus. Second polar body.

- size of ova : 120 microns (largest cell of body).



PRIMORDIAL follicle. (size : 0.02 mm)

* size of follicle just before ovul^n = 18-20 mm

* Max^m no. of follicles @ 20 wks (5th month of intra-uterine life) — 6-7 millions.

At birth : 1-2 millions

At Puberty : 4-5 lakhs.

400-500 follicles mature in entire lifetime.

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1000 follicles every month.

- Germ cells are derived from Epiplast/ectoderm.
  ↓
  Reach yolk sac (by 3 wks)
  ↓
  Genital ridge (6 wks)
  ↓
  Oogonia are formed (9 wks)
  ↓
  1st oocytes (12 wks)
  ↓
  follicle formⁿ begins @ 14 wks'.
  & completed by 24 wks.

- Fertilization:
  - site: ampulla (FT)
  - zygote undergoes cell division.

  ↓
  2, 4, 8, 16 celled zygote.
  ↓
  16 celled zygote (Morula).

* zygote stays in Fallopian tube for 3 days.
  Then it moves towards uterine cavity d/t:-
  (i) Peristalsis of tube
  (ii) Movement of cilia

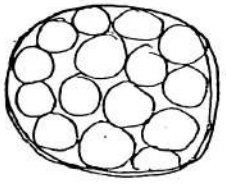
- Nutrition to the zygote in F.T. is provided by secretory cells of FT. in the form of pyruvate.

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* zygote enters the uterine cavity : Day 4 after fertiliz^{n}.

days	14th day	18th day
	Ovulation	zygote enters uterine cavity.



— zona pellucida
(on 5th day zona pellucida is lost & this is K/a zona Hatching).

Morula

- As the Morula enters uterine cavity, fluid enters into it. & Now it is K/a BLASTOCYST.

In Blastocyst, cells are arranged in 2 manners.

↳ ~~*~~ TROPHOBLAST : which lines the Blastocyst.

↳ Inner cell mass :

Implantation :

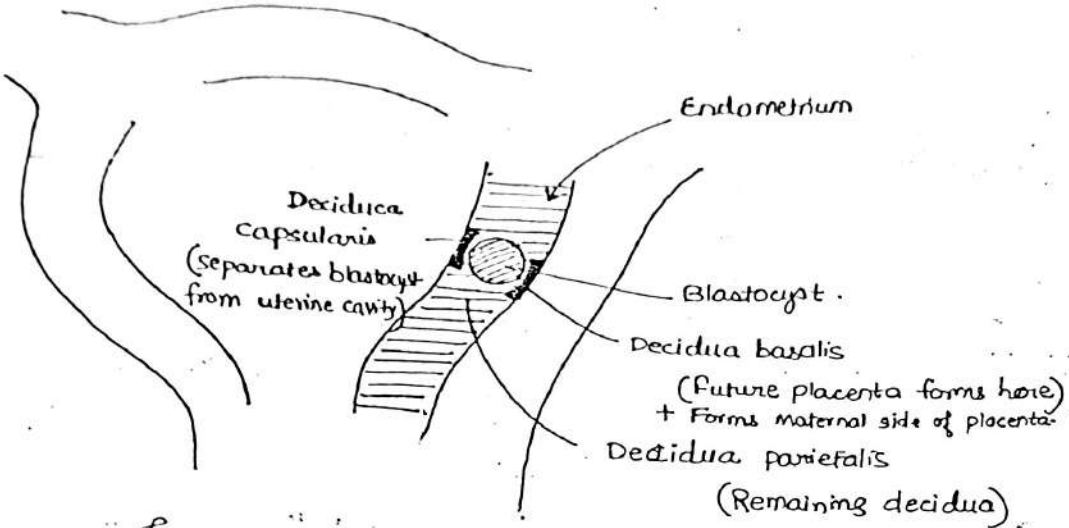
occurs in the form of Blastocyst.

6-7 days. days after fertilization : Beginning.

Completed by 10 days after fertilization

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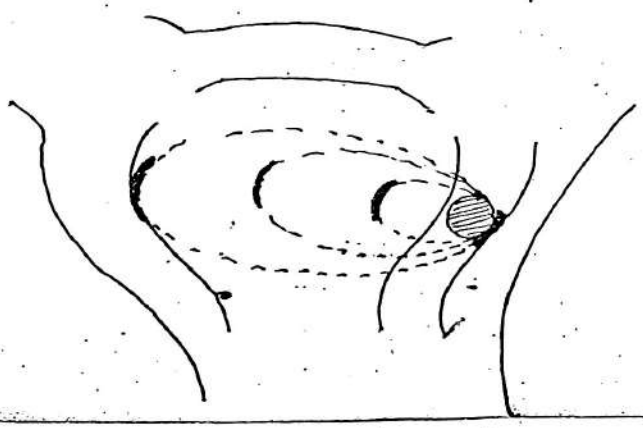
• Interstitial implantation.

- Thickness of endometrium at time of implantation = 8mm
In some females, bleeding occurs at the time of implantn — HARTMAN'S sign.

- After Implantation, Endometrium is K/a Decidua.

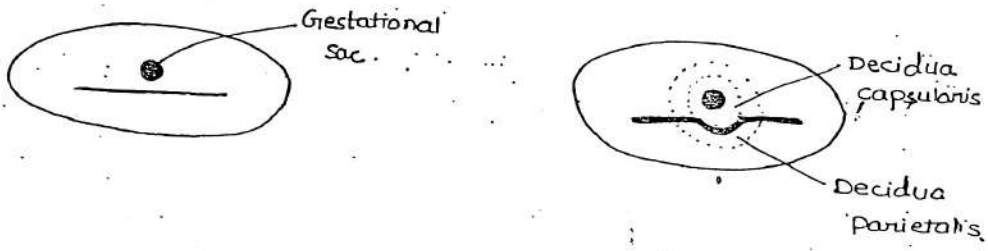
- As the fetus grow, Decidua basalis & decidua parietalis fuse by 16 wks. (uterine cavity obliteration).

∴ Superfetation could be possible only before uterine cavity obliters ie. by 16 wks.



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• Double ring sign.
(at

~~Double ring sign is absent in Ectopic Pregnancy.~~

• Embryonic period : 3-8 wks after fertilization.
(5-10 wks after pregnancy).
↳ Most ~~teratogenic~~ period.

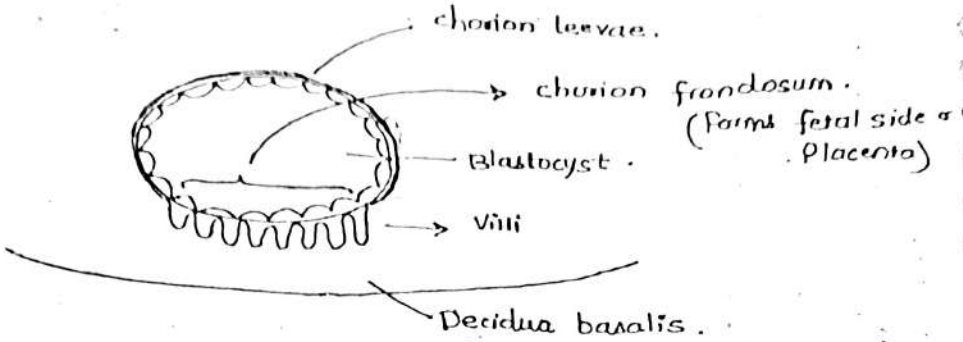
Fetal period : From 9 wks after fertilization uptil delivery.

First germ layer formed in fetus : Endoderm.

Trophoblast < Cyto-trophoblast
Syncytio-trophoblast. | This differentiation occurs 8th day after fertilisn.

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- Fetal membranes.

Amnion.
- Formed @ Day 10, 11. - after fertilisn.
- Derived from ectoderm
- Innermost fetal memb.
- Avascular : provides tensile strength to fetal memb.

Chorion.
Formed 8 days after fertilisn
- Chorion leevae : fuses ⊂ amnion.
- Chorion frondosum

Yolk sac.
First site for hematopoiesis

Allantois.
- A diverticula that arises from hindgut & grows into connecting stalk.

+ Prostaglandin predominantly present in fetal memb. - PGE_2
∴ When fetal memb. are ruptured during labour → Prostaglandins are released. & labour proceeds @ faster rate

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Site of hematopoiesis

1. Yolk sac.	3-6 wks.	Gower 1, 2 Portland.
2. Liver (mainly) & spleen.	≥ 6 wks.	HbF.
3. Bone marrow.	≥ 20 wks.	HbA.

Fetal RBCs have larger size.
but short life span. (90 days)

Fetal Hb. at term = 18 gm %. (75-80% is HbF).

Switch over of fetal Hb. to Adult Hb. begins at 36 wks & is completed by 6 months after birth. (HbF < 1% @ 6 months)
This shift is carried by Gluco-corticoids.

HbF	HbA
– Less of 2,3 DPG. → ∴ Higher affinity for O_2.	– More 2,3 DPG. ∴ Less affinity for O_2
– Less of carbonic anhydrase	– More
– Resistant to acid & alkali	– Sensitive to acid & alkali ∴ gets hemolysed.

* Apt test / singer alkali denaturation test.
– It is a qualititative test done in vasa previa, to differentiate b/w maternal & fetal blood.

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Blood coming from mother's vagina is collected in a test tube.

Add NaOH.

↓

1. If color of blood remains same, blood is not hemolysed.
   ∴ It contains HbF. → Indicates Vasa previa (-ve test)
2. If color of blood is brown. — it means blood has hemolysed.
   ∴ It contains HbA → Indicates placenta previa (+)ve test).

* Thus, Apt test is used to differentiate fetal blood from maternal blood.

* Test to differentiate fetal RBC & maternal RBC.
  ↳ Kleihauer Betke test.
  - Reagent : citric acid phosphate buffer.
  - This is a quantitative test.
  - originally, this test was used to calculate the dose of anti-D in Rh(-)ve females.

Placenta

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→ Villous = Fetal blood = 350 ml.

Inter-villous space = Maternal blood (150 ml)

∴ Volume of placenta = 350 ml + 150 ml = 500 ml.

Villi formation stages:

Fetoplacental circulation is established by 17-21 days.

In the placenta, maternal & fetal blood donot mix & each other & are separated by

(i) syncytio - trophoblast
(ii) Extra-embryonic mesoderm
(iii) Fetal capillary endothelium

: Placental membrane or Placental barrier.

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Wt of placenta at term = 500 grams.
Ratio of wt of placenta : Fetus = 1:6.

Fetal side of placenta	Maternal side of placenta
(i) - Shiny & grey.	(i) - Dull red
(ii) - Membranes attached to it. Cord attached to it.	(ii) - Divided into polygonal areas - Lobes. Each lobe is divided into lobules / cotyledon.
(iii) - Formed by trophoblast or chorion frondosum.	(iii) - Formed by decidua basalis
(iv) - Forms 4/5th part of placenta.	(iv) - Forms 1/5th of placenta

* Functional unit of placenta: Cotyledon/Lobule.
* Placenta formn begins by 6 wks.
~~& completed (anatomically) by 16 wks.~~
Physiologically, placenta keeps on maturing till 3rd trimester.

Term placenta

1. No cyto-trophoblast
2. Syncytio-trophoblast thickness ↓
3. stroma decreases
4. Fetal capillaries increase & move towards periphery
5. Hofbauer cells are present.

* USG done for placental localisation - in 3rd trimester

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Placental circulation:

(A) Uteroplacental circulation: Present in intervillous space

- No. of spiral arterioles in Intervillous space : 120
- O_2 saturation in IVS = 65-75%.
- Utero-placental blood flow at term = 450-650 ml/min
  (* uterine blood flow at term = 750 ml/min)
- Utero-placental circuln is established at Day 12 after fertilizn
- Partial pressure in IVS = 35-45 mm Hg

(B) Feto-placental circulation:

- is via umbilical artery & umbilical vein.
- Feto-placental circuln is established ~~after~~ at Day 17-21 after fertilizn
- Fetal blood volume at term = 125 ml/kg.
- Feto-placental blood flow at term = 400 ml/min
- Rate of O_2 delivery to fetus = 8 ml/kg/min.

	umbilical artery	umbilical vein.
O_2 Saturn	50-60%.	70-80%.
Pressure	60-80 mm Hg	8-10 mm Hg
Remnant	Medial umbilical lig.	Ligamentum teres.

- Median umbilical ligament is a remnant of urachus.

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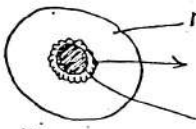
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Anomalies of placenta.

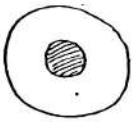
- Normally the maternal side of placenta (Basal plate) & fetal side (chorionic plate) . are equal.

But if fetal side is small ⇨ Extrachorial placenta.

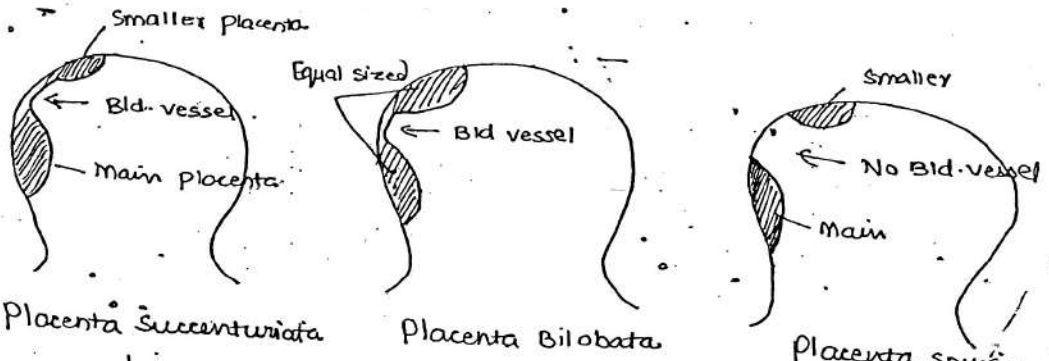
- Circumvallate: This transition is not smooth.
- Circummarginate: The transition is smooth.



• CIRCUMVALLATE.



• CIRCUM-MARGINATE



Placenta Succenturiata — Placenta Bilobata — Placenta spuria.

↓

Smaller placenta is attached to main placenta via a bld. vessel.

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When umbilical cord is not attached to centre, but on any one side ⇒ K/a Lateral insertion of cord. & Such a placenta is K/a BATTLEDORE PLACENTA. 11.

During implantation, the limit of penetration by the blastocyst is governed by Nitabuch's layer, which is a layer of fibrinoid degeneration, present in placenta. It is present b/w the decidua basalis & trophoblast.

Placenta accreta
- Morbidly adherent placenta
- The placenta is not attached to the decidua.
- It is attached to myometrium.

Placenta accreta	Pl. increta	Pl. percreta
Placenta is attached superficially to myometrium	Pl. ~~is attract~~ invades into myometrium	Pl. attached to serosa

- Decidua basalis is absent
  Nitabuch's layer " " - Histologically } in above conditions.

- Highest Risk factor for placenta accreta : ~~Pla~~ Placenta previa/in, Present pregnancy.
  - Placenta previa in previous preg. is not a RISK FACTOR
  - Previous caesarean (2nd Highest)
  - Previous D&C.
  - Previous MTP,

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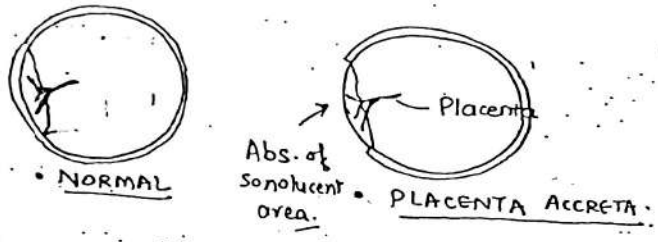
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Presentation :

If undiagnosed :- Presents as refractory PPH.

If Diagnosis - In antenatal Period.

TVS :- 1) Absence of subplacental sonolucent area (which represents decidual basalis)
(18c)



2) Myometrial thickness < 1mm

3) Intraplacental lakes. - This has the highest predictive value for placenta accreta & is used for screening purposes.

4) On Doppler's - vascularity is ↑

~~If USG & Doppler's are inconclusive~~ = MRI (Gold std)

Management :-

1) C.S. + Hysterectomy (If diagnosed).

If Pt. refuses hysterectomy, deliver the baby & leave the placenta inside for autolytic digestion.
+
Keep monitoring β HCG of pt. & USG monitoring.

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Chandraharan; from Chennai, has proposed PPP Procedure for Placenta accreta. (When Pt. refuses hysterectomy)

P - Peri-operative placental localisn
P - Pelvic devascularisation
P - Placental non-separation treated ☆ myometrial excision.

Functions of placenta :-

Mainly Endocrinal.

HORMONES :

I. Progesterone :

* Till placenta is functional, main source of progesterone :- Corpus Luteum
* CL. of Pregnancy is maintained by HCG.
* Placenta takes over the function of CL : 8-10 wks.
* CL of pregnancy degenerate : 12 wks.
* Precursor of progesterone, from which placenta synthesises it :- Maternal LDL.

* Progesterone is smooth muscle relaxant
* Arias stella rxn :- Adenomatous changes that occur in endometrium (d/t Progesterone).

II. Estrogen

- Placenta lacks 17 hydroxylase enzyme, so it can't synthesise estrogen using maternal precursor.
It depends on fetal DHEA Sulphate for estrogen synthase
* Main product of fetal adrenal gland — DHEA Sulfate

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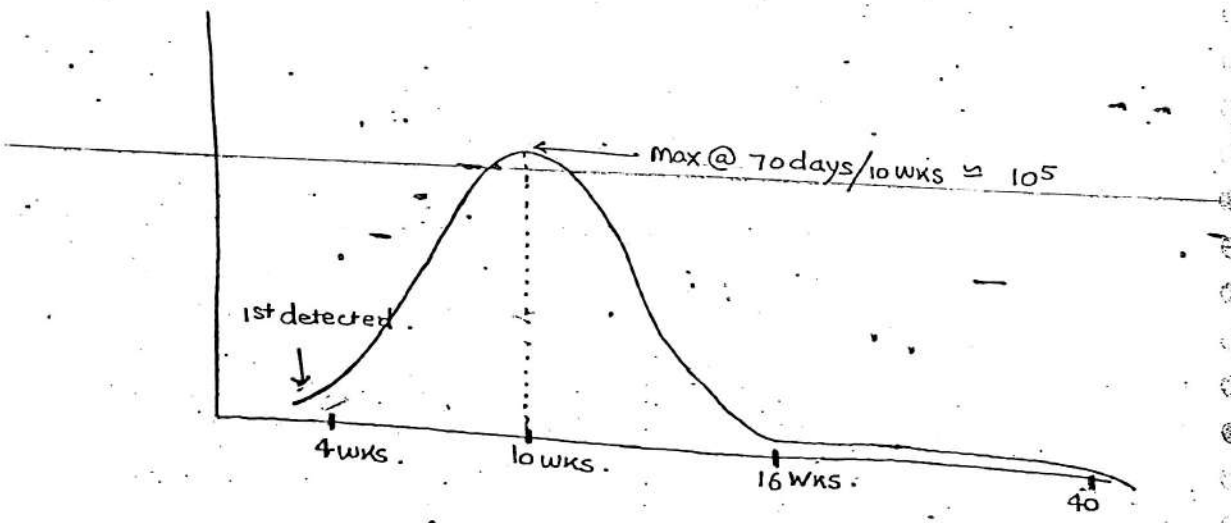
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- most specific estrogen during preg :- E_3 estriol / E_4 estriol
- Max^{na} produced " (mc) " :- E_2 estradiol.
- Estriol - marker for fetal well being (In olden times).

Fetal adrenal gland ← Fetal ACTH
↓
DHEA-S
↓
Estrogen. (↑ signals to the uterus to start contracting at term).

III. HPL.

IV. HCG



- After delivery, it disappears from urine - 48 hours
  from blood - 2 weeks

After abortion. → 4-6 weeks.

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Down syndrome

Trisomy 21.

Extra chromosome can be d/t.

- **Non-disjunction.**
  - in 95%
  - Recurrence = 2%.
- **Translocation of chrosomes.**
  - in 4% cases
  - B/w chr. 3/21.
    14/21.
    21/21 :- Balanced transloc^{n}. (Recurrence rate = 100%).
- **Mosaic**
  - in 1% cases
  - Recurrence rate = 2%.

As age ↑, risk of down syndrome ↑.

At 30 years : 1 in 1000
35 " : 1 in 300
40 " : 1 in 100
45 " : 1 in 30

All Pregnant females should be screened for Down syndrome, irrespective of their age

Screening methods :-

1st trimester –

① USG : ↑Nuchal translucency ≥ 3.5 mm. ∴ It is (+)ve for D.S.
- ↳ should be measured b/w 11–13 wks.
- ↳ Also seen in trisomies other than down syndrome, Turner's syndrome, CHD's & congenital Diaphragmatic Hernia.
- ↳ should be measured in mid-sagittal plane when crown-rump length is 45–84 mm.

② Biochemical markers : Measured at 11–13 wks.

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(i) PAPPA (Pregnancy associated Plasma protein A) or. (schwanger shaft- protein) → Low
(ii) HCG. → High.
} Dual test

2nd trimester :

① USG : Nuchal fold/skin thickness.
↳ Measured in trans cerebellar view.
↳ If ≥ 6mm. → Then +ve for D.S.
↳ Measured b/w 15-20 wks.

② Biochemical test :

(i) Triple test : HCG. (↑) + AFP (↓) + unconjugated estrogen (↓).

(ii) Quadruple test : HCG. (↑) + AFP (↓) + unconj. estrogen (↓) + Inhibin (↑)

Definitive diagnosis : KARYOTYPING.

- Viable cells are needed for diagnosis.
- They are arrested at metaphase, using colchicine → G-Banding
  ∴ It takes longer time (7-14 days) for result to come
- Cells can be obtained by :
  ① chorionic villi sampling
  ② Amniocentesis.
  ③ Cordocentesis.
  ④ Fetal Tissue Biopsy is NOT DONE.

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Chorionic villi sampling:

Study material : Trophoblast / Chorionic villi

Time : ≥ 10 WKS

Most commonly done : 11-13 WKS.

Never do it at ≤ 9 WKS.
(as it can lead to Fetal limb defects).

Rate of fetal loss 1%.

Amniocentesis

Fibrocytes / Fibroblasts.

≥ 15 WKS

16-18 WKS.

Earlier, amniocentesis was done b/w. 11-14 wks But now not done d/t Post procedure abortion

Fetal loss :- 1 in 300-500

Cordocentesis :-

- Time ≥ 20 WKS
- Blood - From umbilical vein (site: Placental end of cord)
- Fetal loss = 3% (very high).
- Not used, these days, for genetic diagnosis.
- Earlier, it was used for determining fetal blood group. (Now it is done by analysis of cell free fetal DNA present in maternal circulation)
- ONLY INDICATION, today :- To confirm fetal anemia, prior to Intra-uterine transfusion in Rh-ve female.

For early results, instead of Karyotyping, FISH is done.
- In FISH, a no. of chromosome specific & gene specific DNAs & fluorescent probes, are used.
- cells are visualised here in Interphase (∴ early results in 6-8 hours)

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= FISH is available for chromosome 13, 18, 21, X & Y

- Non-invasive pre-natal test : cell free Fetal DNA.

Fetal cells can be isolated from maternal blood & can be used for diagnostic purpose

Advantage :- Non-invasive test.

Disadvantage :- Only a screening test (Not diagnostic) & cost is very high.

Imp. points/one-liners.

- Mc. complicn of chorionic villi sampling : Fetal loss.
- mc. " " " done before 9wks : Fetal limb defects.
- chorionic villi sampling can detect - chromosomal anomalies, Hemoglobinopathies, Inborn errors of metabolism. But it can't detect neural tube defects.
- Amniocentesis can detect everything that chorionic villi sampling detects + Neural tube defects.
- If a pt. comes with a history of down syndrome in previous baby, then instead of screening test, diagnostic tests should be done directly.

In 1st trimester :- chorionic villi sampling (Karyotyping)
2nd " :- Amniocentesis (Karyotyping).

- In Twin Pregnancy, Dichorionic diamniotic twins, the chromosomal anomalies are to be detected in both the twins. But in monochorionic twins, the anomalies need to be detected in only one of the twins.

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Alfa-feto protein:

Glycoprotein ; synthesised by fetal yolk sac – in early preg.
& by fetal GIT & liver – in late " .

- Max^m amount is seen in fetal blood . (Time : 13 wks) .
(concentration) .

- In maternal serum , it can be detected b/w 15-20 wks.

- Raised in :- • Neural tube defects
• Abd. wall defects (Gastrochisis, Pilonidal sinus, cystic hygroma , etc).

- Decreased in : G – Gestational Trophoblastic disease
O – over-estimated Gestational age / obesity
A – Abortion .
T – Trisomy .

Anencephaly :

Ioc : Targetted USG
(Targetted imaging for Fetal Anomalies – TIFFA : 3D Ultrasound)

Earliest diagnosed congenital anomaly by USG .

Earliest diagnosis :- By TVS (at 10 wks).

Best diagnosis :- at 14 wks.

On USG. < Mickey mouse sign
Frog eye sign .

Best biochemical marker for anencephaly (NTD) :- Acetyl cholinesterase

Recurrence rates :- After 1 NTD = 5%.
After 2 " = 10-15%.

Dss · Fetal adrenal gland hypoplastic / absent → DHEAS absent → Estriol ↓ → Post dated Preg.

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umblical cord

- Develops from connecting stalk
- In early fetal life, cord has 2 arteries & 2 veins, but later rt. umb. vein disappears, leaving only the original lt. vein (Left is left) Thus, at term, Umb. cord has 2 arteries & 1 vein.
- Length = 55 cm. (30-100 cm)
- < 32 cm :- Short cord.
- Umb. arteries has 'folds of Hoboken' across their lumen & its connective tissue is K/a Wharton's jelly.
- Cord pH = 7.2.

- Sequence of closure

  umb. artery → umb. vein → Ductus venosus → Foramen ovale ← Ductus arteriosus

- mc vascular anomaly of cord — Single umbilical artery.
  Incidence = 0.7 to 0.1 %.
  (↑ in twins preg. / black/diabetic female)

Congenital Anomalies in Single umb. artery :-

Renal anomaly > CVS anomaly

Single umb. artery + Cong. anomalies ⇒ Always do Karyotyping (as trisomy 18 increased)

Velamentous insertion of cord

Cord is first attached to membs. & then to placenta.
It gives rise to Vasa previa.

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Vasa previa

Velamentous insertion of cord
↓
Cord is attached to fetal memb.
↓
When Cx dilates & memb. ruptures.
↓
Cord gets injured.
↓
Cord has umb. artery & vein → Injured (Bleeding)

This Bleeding is of fetal origin & will lead to fetal death.

~~But because this is~~

- APT test
- Antenatally - can be detected by Doppler USG
  ↓
  Management = Elective C.S.

During labour - Emergency C.S.

~~CVS of fetus~~.

Fetal respiratory system

- ~~Endodermal~~
- Surfactant synthesis begins at 20 wks.
- Surfactant appear in amniotic fluid @ 28 wks.
- Earliest fetal breathing movement. - seen by 11 week: USG
- M/c test done for fetal lung maturity - L:S ratio in amniotic fluid
  L:S ratio > 2 → lungs are mature
  Best test = phosphatidyl glycerol in amniotic fluid.
  Bed side test = Shake test / Bubble test.
- Drugs to enhance fetal lung maturity : Corticosteroids (< 34 wk)

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Recent ACOG Guidelines.

- If c.s. is being done bef b/w 34-37 weeks — then also corticosteroids is given.
- But if vaginal delivery is done b/w 34-37 wks — No steroids.
  (Because in vaginal del. — Baby is in stress & itself produces steroi

DOC : Betamethasone

12 mg : 2 doses - 24 hrs apart - IM.

- Also decreases the incidence of Necrotising enterocolitis & Intraventricular hem.

Dexamethasone (DOC in India)

4 doses : 6 mg each - 12 hours apart. - IM

* Repeated doses of steroids : cerebral palsy.

Amniotic fluid

Sp. gravity = 1.008 to 1.010

Osmolality = 250 mosm/L

Rate of turnover = 500 cc/hr.

PH : 7.2 to 7.4 - This forms the basis of Nitrazine test in PROM.

Maxm volume = 36-38 wks.

Composition : 98-99 % water.

Colour of amniotic fluid : Straw colored (at term).

- Green : Fetal distress -> Breech & transverse lie -> Listeria infecn
- Golden : Rh. incompatility (Due to Bilirubin).
- Tobacco juice : IUD
- Saffron (Yellowish-Green) : Post dated pregnancy.

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Source

Early Preg - A.F is ultrafiltrate of maternal plasma

12 - 20 wks - Transudate across fetal skin

≥ 20 wks - Fetal urine. (Main contributor - Overall)

Water in amniotic fluid is replaced once in 3 hrs.

Fetal urine produc^{n} at term = 650 ml/d.
(at 20 wks = 5ml/hr, at 40 wks = 52 ml/hr).

B/w 37 wks & 38 wks + 6 days : Early term

B/w 39 wks & 40 wks + 6 days : Term

B/w 41 wks & 41 wk + 6 days : Late term

After 42 wks : Post term

What % of females deliver on EDD = 4%.

50%. deliver either 1 wk before or 1 wk after EDD.

1st step : Review of her menstrual His.

Congenital anomaly that leads to Post dated preg
= Anencephaly.

Amniotic fluid is seen in pockets on USG.

Largest vertical pockets of each quadrant of abdomen is added to give = Amniotic fluid index. (5-25 cm).

<5cm : oligohydramnios

>25cm : Polyhydramnios.

Measure the largest pocket (overall). = 2-8 cm.

<2 = oligo.

>8 = poly

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Oligohydramnios

- AF < 200ml
- AFI < 5

* Mc cause of mild oligohydramnios } Idiopathic.
  " " " Poly " }
* Mc. cause of severe oligohydramnios } Gross congenital anomaly.
  " " Poly "

oligo : Renal agenesis (Mc)
poly : Cleft palate > NTD ! !

Problems ~ oligohydramnios :

- If oligohydramnios occurs early in preg.
  ↓
  Uterus will expand less.
  ↓
  Less space for fetal parts to grow properly.
  - Pulmonary Hypoplasia (Mc).
  - Limb reduc^n defects.
- If oligohydramnios occurs late in preg (Fetal parts already developed)
  ↳ less space ⇒ cord compression.
  ∵ cord has umb. artery & umb. vein /
  Fetal bld supply ↓
  ↓
  Fetal distress
  ↓
  Fetus passes meconium → in A.F.

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fetus swallows — meconium stained A.F.

↓

Meconium Aspiration syndrome.

Mngmnt: Amino-infusion & Normal saline.

Polyhydramnios:

AF ≥ 2L

AFI > 25 cms.

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Physiological changes during Pregnancy.

Probable signs of Preg.

1. Goodell's sign : Earliest sign (6 wks) → Softening of cervix.
2. Hegar's sign : 6-10 wks.
   Softening of isthmus → when bimanual examⁿ is done, vaginal & uterine fingers touch each other

Restall signs (+)ve at 8 wks.

3. Chadwick's / Jacquemin's sign. – Bluish discolorⁿ of vagina.
4. Osiander's sign – Pulsation in lat. fornices.
5. Piskacec sign – d/t Lateral implantation

Other probable signs are :-

- Braxton's Hick contractions – Painless, irregular contractions, felt from 2nd trimester onwards. Intrauterine pr : 5-25 mm Hg during these contracⁿs.

~~Ext. & int. ballotment~~

Definitive signs of Pregnancy :-

1) Fetal movements can be felt
2) USG evidence of Preg
3) FHS either by stethoscope / Doppler
   ↓ 18-20 wks ↓ 10 wks.
4) On X-ray : Fetal skeleton is visible (16 wks)
   But X-ray is C/I in Preg.

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Quickening :- 18 wks (primi) ; 16 wks (Multi).

Presumptive signs of pregnancy - All the symptoms.

* BMR ↑ by 10-20%.
* Total water retained - 6.5 L.
* Na & K retention occurs during preg.
  But d/t water retention, serum Na & K ↓.
  Plasma osmolality ↓.
* Extra calorie req : 350 Kcal/d.

Total wt. gain = 11 Kg (1 kg - in 1st trimester
5 kg - 2nd Trimester
5 kg - 3rd "
)

Net wt. gain = 6 Kg.

Wt. gain depends on
- Race/ethnicity.
- Parity.
- Socio-economic status.
- Preconceptional wt / BMI
- Associated conditions like D.M.

* Smoking doesn't affect maternal wt. gain during preg.
  Rather, it affects fetal weight gain.

	BMIs	Recommended wt. gain
Underwt.	< 18.5.	12.5 to 18 kg
Normal.	18.5 - 24.9	11.5 to 14 kg
Overwt.	≥ 25	7 - 11.5 kg
Obese.	≥ 30	< 7 kg.

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Changes in Hematological System

↑ (Increase)	↓ (Decrease)
- Blood volume (30-40%.)	- Hemodilution → Viscosity of blood ↓ Plasma osmolality ↓
- Plasma. " (40-50%.)	
- RBC " (20-30%.)	
- Hemodilution during Pregnancy. → Physiological anemia in preg. (But Hb will never be less than 11 gm/dL)	- ↓ Packed cell volume (RBC volm / Plasma volm) → Hematocrit.
- Hb mass ↑ (gm) ⇩ O_2 carrying capacity ↑	——— But Hb concentration ↓ (gm/dL)!
- WBC count ↑	——— Platelet count ↓ (Benign Gestational Thrombocytopenia)
= Plasma protein amount ↑	——— Plasma protein conc^n ↓
Albumin ↑ — GLOBULIN	——— Globulin ↓ — ALBUMIN (ALBUMIN / GLOBULIN = 1·7 : 1 (Normal) In Preg. ⇒ 1 : 1)
- Clotting factors ↑ (including fibrinogen).	——— Factor XI & XIII
Preg is Hypercoagulable state. ↓ ↑ ESR & C-reactive protein	* BT & CT are unaffected during Preg.

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- Anemia < 11gm/dL
  Severe anemia < 7 gm/dL
  Very severe anemia < 4gm/dL.

DD: ~~Iron deficiency~~

* Mc : Iron-def. (In developing countries = Dimorphic)
  ↳ Iron def + folic acid def.

* 1st sign in Iron def. anemia : ↓ S. Ferritin (most sensitive)

* Iron-requirement during Preg :- 1000 mg
  (300mg - Required by fetus)

Per day requirement < 1st half : 3-4 mg/d
2nd half : 6-7 mg/d

Iron-supplementation is must in Preg.

Tab supplied by GoI = Fe + Folic acid :
(100mg) (500µg)
↓
Ferrous sulphate. (Ferrous fumarate : In OCPs)

- After giving iron,

1st parameter to ↑ = Reticulocyte count

↑ in reticulocyte count occurs after 7 days of giving Iron
Max^m ↑ " " " 14 days " "

Hb increases after 3 days " "
(0.7 - 1 gm per week).

Best measure for Hb : cyanomethemoglobin method.

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Indications for Blood transfusion in Anemia.

1) If anemia is d/t hemorrhage (eg. APH)
2) If a female presents c severe anemia beyond 36 wks of Pregnancy.

Rate of increase in Hb is same for parenteral/oral Iron.

Changes in GIT

No change in Gastric emptying time during Preg
It ↑ during Labor.
↑ chances of Reflux oesophagitis as progesterone relaxes LES
splenic area ↑ by 50%.

Changes in Liver

No change in size & Histology.
All enzymes including AST, ALT, GGT decrease.
except ALP → doubles (d/t heat stable placental ALP)

Changes in Gall bladder

cholestasis. (d/t estrogen)
Progesterone ↓ tone
Both these findings along c ↑ cholesterol favour GB stone formation.

Cholestasis of Pregnancy.

2nd MCC of Jaundice in Preg. (MCC - Hepatitis)
↑ S. Bile acid — ∴ Itching/Pruritis (1st symp.) — Palms & soles
↳ More common in 3rd Trimester.
Bilirubin never more than 5
Liver enzymes are mildly ↑ (Never more than 250)

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- Best investigation / Earliest : Measure Bile acid
- It is a recurrent condition
- ∴ In pts. who had cholestasis in preg., OCPs shouldn't be used.

DOC : Ursodeoxycholic acid

2nd DOC - Cholestyramine

- ↑ chances of sudden IUDs in cholestasis.
  ∴ Pregnancies should be terminated b/w 37 - 38 wks
- If pt. has cholestatic jaundice, pregnancy should be terminated at 36 wks.

Changes in Endocrine system

- Pituitary ↑ by 135% (d/t estrogen stimulation).
- S. prolactin levels ↑ (maxm levels)
- But Incidence of Prolactinoma doesn't ↑ in preg.

Changes in CVS.

Increased	Decreased
• Cardiac output = (Stroke Vol x HR)	• Progesterone ⇒ Smooth muscle relaxant
• Preload	• Peripheral vas. resistance ↓ (∴ BP ↓)
• O_2 requirement	• After load ↓
	• ↓ Arterio-venous O_2 gradient

C.O. - Maxm ↑ : Immediately after labour (DELIVERY) > 2nd stage of labour > Late 1st stage of labour > 28-32 wks.

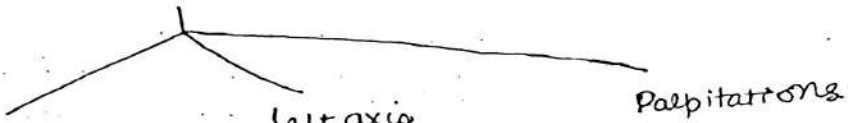
* Central venous Pr.
  Pulmonary cap. wedge Pr. } unaffected

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Changes in clinical indicators of Heart during Preg.

1) HR, Pulse ↑
2) BP ↓
3) Heart rotates upward & outward during pregnancy.



4) S_1 = Loud, exaggerated splitting during Preg.
   S_2 = Normal
   S_3 = Easily heard.

5) Murmurs : ESM (less than grade 3)
   Mammary murmer (continuous murmer).

6) on - X-ray - Mild cardiomegaly
   - Straightening of left ~~half~~ heart border

Supine Hypotension / Mengert's syndrome

When a preg. female lies supine in late 3rd trimester.

↓

Gravid uterus Presses against the IVC.

↓

Venous Return to Heart ↓ (CO.↓)



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Indicators of Heart disease in Pregnancy.

- Clubbing, cyanosis, Raised JVP
- Persistent splitting of S_2 / 4th H.S.
- Diastolic murmer
- Systolic murmer (ESM > Grade 3)
- Persistent arrhythmia.
- cardiomegaly, Pulmonary edema.

- Mc Heart disease during Pregnancy = Mitral stenosis.
- Mc. Congenital Heart disease " = ASD.
- " cyanotic CHD " = ToF
- " Congenital Valvular Heart disease = Mitral valve prolapse.

Clarke's classific^{n} (Based on maternal mortality)

- **CLASS I** (Maternal mortality less than 1%.)
- **CLASS II** (Maternal mortality 15-25%.)
- **CLASS III**
  - ✓ Pul. Hypertension
    - 1° ~~Poorooth~~
    - 2° ⇓ Essinmenger's Syndrome

* Termination of preg. / MTP is advised for class III diseases.

(Time: 12 wks. for MTP:
⇓
By suction/evacuation)

- ✓ Marfan syndrome
  - ↳ aortic involvement
- ✓ Coarctation of aorta

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- HD with max risk of maternal mortality :- Essinmenger's syndrome.
- Most HD leading to mat. mortality :- MS.
- HD in which Preg. is C/I —
  (1) clarke's class III disease.
  (2) Ejection fraction < 40% (WHO < 30%)
  (3) Severe MS (Valve area < 1.5-2 cm^2)
  (4) Severe AS (Valve area < 1 cm^2)
  (5) HD belonging to NYHA III/IV.
- Mc. fetal complicⁿ of HD — Prematurity & IUGR

Management of HD during labour -

- Left lateral position / O_2 bedside.
- Restrict PV examination.
- Restrict fluid to 75ml/hr
- Allow pts. to go into spontaneous labour → Induction of labour

* Trial of Labour - C/I
- Labour analgesia is given (Epidural analgesia) - Imp.
- Mode of delivery: Vaginal delivery
  If delivery doesn't occur in ½ hrs, then ventouse/Forceps should be used. (Vaccum or ventouse: Better b/w two)

HD where C.S. is indicated. (Wherever aorta is involved)
- Aortic aneurysm
- Coarctation of aorta
- Marfan syndrome c aortic involvement
- Severe symp. aortic stenosis.
- Acute severe CHF / Recent MI

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- Need for emergency valve replacement immediately after delivery
- A pt. who is fully anticoagulated ↑ warfarin at the time of labour / in 2 wks of labour.

- Anaesthesia given during LSCS in HD : Epidural anaesthesia

* C/I of epidural anesthesia : (GA is to be given)
  1) Intra-cardiac shunts
  2) HOCM
  3) Severe AS., Severe AR
  4) Pulmonary HT

- Post partum management :
  - Give diuretics.
  - Methyl ergometrine is C/I. (Oxytocin is given)

* C/I of Methyl ergometrine.
  T - After delivery of 1st Twin
  O - Organic HD.
  P - Pre-eclampsia
  E - Eclampsia
  R - Rh (-)ve Preg.

Choice of Anticoagulant during Preg.:

Period of Gestation	Anticoagulants of choice.
< 12 wks	Heparin (warfarin causes CHONDRODYSPLASIA of fetus)
12-36 wks.	Warfarin
≥ 36 wks.	Heparin.

→ If a female is on warfarin during labor → C.S.
→ & Restart - 6 hrs after vag. del. & 24 hrs after C.S.

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Why switchover to Heparin at 36 wks?

↳ Warfarin is very strong anti-coagulant

↳ No-antidote available for warfarin.
(Antidote available for Heparin)

Best time to do surgery during MS = 2nd trimester.

Earlier surgery of choice was valvotomy.

Today surgery " " - Transcatheter balloon dilz

Valve replacement is C/I during preg.

Best permanent method of contraception in HD.

↳ Vasectomy of Husband

↳ Tubectomy — by Mini Laparotomy - after 1 wk of delivery (Local anesthesia).
(Laparoscopy - C/I)

Temp. methods:

↳ OCPs : C/I

IUCDs : are taken as WHO class 2 (ie. Benefit outweighs Risk)

Progesterone containing pills/implants.

Barrier method (Best) : But- High failure rate.

Changes in Renal system during Pregnancy:

- Size of kidney ↑ (1cm)
- Hydronephrosis, Hydro-ureter.
- Congestion of bladder.
- Bladder capacity ↓, Bladder pr ↑
- Renal blood flow ↑ ∴ GFR ↑
  ie. Filtering capacity ↑ : → ↑ increased clearance of urea, uric acid & creatinine
  ↓
  ∴ S. urea/uric acid/creatinine ↓

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Diabetes In Pregnancy.

- Pregnancy is a diabetogenic state, a/w -
  - Hyper-insulinemia
  - Insulin resistance. (max: 24-28 wks).
    - ↳ d/t : H - HPL
      P - Progesterone
      E - Estrogen
      C - Cortisol.

- Maternal insulin can't cross placenta
- Fetus starts secreting insulin by 12 wks.

* Main source of fetal energy is glucose.
This glucose is transferred by facilitated diffusion across the placenta using GLUT1 & GLUT3.

Maternal Hyperglycemia
↓
Fetal Hyperglycemia.
↓
Hyperplasia & Hypertrophy of β cells of fetal pancreas.
↓
↑ Insulin secretion by fetus (Hyper-insulinemia)

- Macrosomia.
- Inhibits Surfactant Production. → ↑ incidence of RDS.
- Neonatal Hypoglycemia.

* D/t maternal hyperglycemia → placenta undergoes hydropic changes (as glucose imbibes water), so it becomes non-functional at places. This creates a dysparity in demand & supply → Hypoxia → sudden IUD.

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MC time of IUD in diabetic mother : last 2 wks of Pregnancy.

Diabetes.

Diabetic pt. becomes Pregnant
↓
OVERT DIABETES.
(Blood glucose abnormal from day 1)

↓
Hyperglycemia
↓
Free radicals
↓
Congenital malformns in fetus.

Pt. was normoglycemic before Pregnancy.
During preg, because of Insulin resistance, she becomes diabetic
↓
GESTATIONAL DIABETES.

↓
Free radicals will be formed by 24-28 wks.
By that time organogenesis is complete.
∴ No congenital malformns

In Diabetic females :-

Which test predicts the risk of congenital malformations :-

HbA1c.

- < 6.5% → No risk
- 6.5 - 8.5 → mod. risk
- > 9.5 → High risk

To detect congenital malformn — best test :- level 2 USG @ 18-20 wks. (TIFFA)

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In overt diabetics :- Fetal cardiography (@ 22-24 wks) to detect Heart anomalies (Not done in Gestational DM).

Mc. system involved in congenital malformⁿs :-
CVS > CNS.

Mc. anomaly : VSD > NTD.

Most specific anomaly : caudal regression syndrome (sacral agenesis)

Most specific cardiac anomaly : TGA.

Most common " " : VSD

" " CVS finding in diabetic mother : HOCM

To reduce anomalies :-

- control Bld sugar (insulin).
- Folic acid.

(Normally folic acid given to all preg. women :- 400 µg, to Prevent NTDs. This should be given ideally one month before conception & continued for 3 months after conception. This is K/a prophylactic dose of folic acid)

But the dose of folic acid given to -

(i) Females c previous H/o NTDs.
(ii) Diabetic females
(iii) Females on anti-epileptic drugs
(iv) Females c sickle cell anemia.

} is 4mg. (K/a Therapeutic dose) ↓ chances of NTDs by 72%.

* Dose of F.A. to t/t Megaloblastic anemia in Preg : 1mg/day
* RDA of Ca^{2+} in Preg : 1000 mg
* RDA of I_2 " : 250 µg

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Diagnosis of GDM

2 step approach (ACOG)

- 1st step = Screening test (universal for Indian populn).

GLUCOSE CHALLENGE TEST.
(Time: 24 - 28 WKS).

Method : 50 gm glucose (irrespective of previous meal).

↓

Level ≥ 140.

↓

Screening (+)ve

- 2nd step = Confirmatory test

3 hour OGTT (100 gm.)
(GLUCOSE TOLERANCE TEST).

FBS < 95
1hr < 180
2hrs < 155
3hrs < 140.

} Carpenter & Coustion Criteria

If any 2 volume are abnormal, pt. is diabetic.

* If value of GCT. ≥ 180 – don't go for GTT: Diabetes confirmed

National diabetic criteria :

FBS < 105 mg/dL. = (5.8 mmol/L) → Divide mg/dL by 18 to get into mmol/L.
1hr < 190
2hrs < 165
3 hrs < 145

1 step approach (IAP, WHO)

Single test :
2 hr. OGTT (75 gm)
(Time : 24-28 WKS)

FBS < 92.
1 hour : < 180
2 hour : < 153.

If any of the values is abnormal – pt is diabetic.

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Indications of screening for GDM

↓

All High risk females.

↳ Previous H/o GDM., Big baby, Baby ⊂ congenital form^{n} in previous pregnancy

↳ family H/o DM

↳ obese female.

↳ Age > 30 years.

↳ Previous H/o still birth.

Management:

① Diet & exercise — For 3 wks.

Metabolic goals to be achieved at the end of 3 wks:-

- Pre-meal value : 70-95 mg/dl
- 1 hour (P.P.) : ≤140
- 2 hours (P.P.) : <120
- HbA1C < 6%

If these goals are not met : INSULIN.

* Oral Hypoglycemics are not advised during pregnancy, as they can cross placenta & cause fetal hypoglycemia.

only oral hypoglycemic drug approved for use:
Glyburide / Metformin.

Dose of insulin ↑, as the gestational age ↑.
But ↓ during labour (because pt is on liquid diet).

- overt diabetics & GD pts on insulin are admitted at 34 wks.

- Time & Mode of delivery:-
  - GDM controlled on diet : 40 wks
  - GDM controlled on insulin : 38 wks.

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- overt diabetics : 38 wks.
- Preferred mode : Vaginal del.
- If wt of fetus is > 4.5 kg in diabetic pts : CS.

* All pts. of GDM should have a ~~@~~ 75 gm GTT at 6 weeks. If it is normal then repeat it every 3 years.

* Contraceptive advice :
  - GDM & overt diabetes ∈ vascular complicns :- low dose OCP.
  - If pt. has High BP / Dyslipidemia / obesity : IUCD can be used.
  - With vascular complication :- avoid OCP (it can cause thrombosis).

Complications of Diabetes :

Maternal	Fetal	Neonatal	Adult life.
• Poly hydramnios ↓ Preterm labour / PROM / PPH. ↑ infecns. (UTI / Asymptomatic bacteriuria) In future they can have DM.	• Hyperglycemia ↓ β cell Hyperplasia ↓ Hyperinsulinemia ↓ ↑ growth of fetus (MACROSOMIA). ↳ SHOULDER DYSTOCIA. • ↑ abortion / IUDs / Still births.	• Prematurity ↓ RDS. (Best test to asses fetal lung maturity is :- Phosphatidyl Glycerol). • Hypoglycemia • Hypocalcemia • Hypomagnesemia • Hypokalemia • Polycythemia • Hyperbilirubinemia → HYPERVISCOSITY SYNDROME.	• obesity • DM • Heart disease

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Macrosomia :

wt. of fetus $\geq$ 4.5 kg. (in India $\geq$ 4 kg).

MCC — Mother < Diabetic / obese.
- Past dated preg.
- Male fetus

Best USG parameter to detect macrosomia – Abd. circumference

USG parameters.

To determine Fetal age.

In 1st trimester : Crown Rump length (overall – Best). – Best time
2nd " : Head circumference / Biparietal diameter
3rd " : Femur length

* The only USG parameter which tells about fetal growth is Abd. circumference.

- For estimating fetal wt. on USG, 2 formulas are used :-

Hadlock's . Shepherd's.

- For estimating fetal wt. clinically : Thomson's formula

Management of macrosomia :-

If wt. of fetus > 5 kg in non-diabetic females or > 4.5 kg in diabetic females } C.S.

Complicn of macrosomia : SHOULDER DYSTOCIA.

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Shoulder dystocia: Difficulty in delivery of shoulder (Delay of more than 1 min after delivery of head).

* Turtle sign

Risk factors:
- D - Maternal Diabetes
- O - Mat. obesity / Fetal obesity (macrosomia)
- P - Post dated pregnancy
- A - Anencephaly.

Management: By shoulder Dystocia Drill.

1. call for help.
2. Liberal episiotomy
3. Stop giving Fundal pressure
4. Give supra-pubic pressure.
5. 1st manuever/Best manuevre/most effective :-

Mc ROBERT'S MANUEVRE. → Flex & abduct the female's thigh. (Nerve that could be injured : Lat cutaneous nerve of thigh)

If these measures fail :- 2nd line measure :- WOOD'S COCKSCREW MANUEVRE.

If this also fails, 3rd line measures :-
1. Cleidotomy.
2. Symphysiotomy.
3. Zavenelli's manuever - Push the head of the baby back (delivered) & Perform C.S.

* Mc complicn following shoulder dystocia : PPH
Mc fetal complicn : Brachial plexus injury > #clavicle > # Humerus

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IUD : Death of fetus beyond the period of viability.

1st X-ray sign to become +ve : ROBERT'S SIGN (↓ Gas in Great vessels of fetus)

→ SPALDING SIGN - overlapping of cranial ~~sig~~ bones

** ↳ Hyperflexion/Hyper-extension of fetal spine : BALL'S sign

Management : Induction of labour (vaginal del : preferred mode)

* If transverse lie c IUD → C.S.

* Destructive procedures are not done.

EQ. All of the following can be seen in neonate of diabetic mother except.

✓(a) Anemia

(b) IUGR

(c) Hyperbilirubinemia

(d) Hypocalcemia.

Q. ~~All are seen in Gestational DM except.~~

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Pre-term Labour :

- Labor that begins before 37 wks.
- mcc : Idiopathic
- 2nd mcc : Infection (Bacterial vaginosis, Asymp. bacteriuria)

∴ Highest risk of pre-term labour is :- If there is previous H/O Preterm Labour.

Suppose, a $G_2 P_{1+0}$ female Previous H/O preterm labour,

Prophylactic measures taken to Prevent Pre-term Labour :-

1. Progesterone :- It prevents pre-term labour. But if Preterm labour begins, it can't be used to tt it (ie-not a Tocolytic).
2. Stop smoking
3. cervical circlage

Diagnosis :-

① USG :

Normal length of cervix during preg : - 4 cm.

* If length becomes < 2.5 cm. ⇒ Pre-term labour.
* U shaped cervix. ⇨ " " "
  (Normal : T shaped)
* Dilatation of cervix > 2cms. ⇨ " "

② Fibronectin Level : By ELISA (B/w 24–34 wks)

If Fibronectin > 50 ng/mL ⇨ Pre-term labour.

③ Uterine contracs : 4 contracs in 20 min / 60 min } ⇨ Pre-term labour
or 8 " "

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Preterm lab. → Fetal lungs do not mature → RDS.

Management:

- Preterm lab. ≥ 34 wks. : wait & watch.
  send Rectal & vaginal swab for detecting Streptococcal infecⁿ, Group B.
  (Normally done : 36-38 wks).
- Pre-term lab. < 34 wks. — steroids.
  — short term tocolytics (so that steroids get time to act)
  — $MgSO_4$ (Neuroprotective role)

* There is no role of antibiotics in pre-term lab. ! !
Antibiotics are used only when membs. are ruptured.

Tocolytics:

- Best tocolytics : Nifedipine
- Best " in HD pts : Atosiban

- Tocolytic ∈ max maternal side effects : β agonists / β mimetics.
  (Salbutamol, Ritordine, Isoxuprine, Terbutaline)
  β mimetics can lead to Tremor (mc side effect), Hyperglycemia, Hypokalemia, & Pul. edema.
- $MgSO_4$ acts as a Tocolytics @ concn of 9-10 meq/L.
- Indomethacin is also used as tocolytics. But it leads to closure of ductus arteriosus. (∵ it has max fetal side effects)

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In PROM, there is risk of chorioamnionitis,

∴ If PROM occurs > 34 wks.

Management :
- Antibiotics.
- Induce labour
- Minm pv-examination.
  (Per speculum examn is done).

Nitrazine test : For diagnosis of PROM.

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Pregnancy induced hypertension.

Hypertension in pregnancy is defined as systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on 2 occasions, 4 hrs apart.

Hypertensive female conceives. ↓ Chronic HT in preg.	A Normotensive female becomes hypertensive during pregnancy (d/t Placental pathology) ↓ PIH.
- Increase in BP is seen before 20 wks of preg.	- Increase in BP is seen after 20 wks of pregnancy.
- BP will not come back to normal after 12 wks of delivery.	- BP will return back to normal after 12 wks of delivery.

Placental pathology that leads to PIH.

Trophoblastic invasion.

Normally,

1st step: @ 10-12 wks - The wall of the decidual part of the artery is replaced by Trophoblast.

2nd step: @ 16-18 wks - Trophoblast will replace the myometrial part/segment of the artery.

When the 2nd step doesn't occur → TROPHOBLASTIC invasion is incomplete. → Maternal arteries have high resistance & pressure. → PIH.

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Trophoblastic invasion is incomplete

↓

Blood supply to placenta ↓

↓

Placental ischemia

↓

Release of inflammatory med. & vasoactive substances & that will lead to ENDOTHELIAL INJURY.

↓

Capillaries will become leaky.

↓

Movement of fluid into extravascular space

- End organ failure (ie. PIH is a multiorgan disorder) ⇦
  - Hemoconcentration
  - Platelet number & functions become defective.
- Collection of fluid in Third space ⇩ EDEMA.

• Definitive management in PIH : Termination of preg.

- Placenta is small in case of PIH.

* causes of big placenta:
  - DM
  - Rh - incompatibility
  - TORCH infection.

- Mc Hematological finding in PIH — ↓ Platelet count.

** Till her platelet count is Normal, other hematological findings are always normal ∴ other tests like (PT, APTT) are not done.

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PIH

Gestational Hypertension.

↳ All 3 characters of PIH are seen.

Pre-eclampsia

↳ (i) All 3 characters of PIH
+
(ii) Proteinuria or signs of end organ damage

Now,

Mild & severe Pre-eclampsia can't be differentiated on the basis of proteinuria.

Proteinuria -

Excretion of protein in urine > 300 mg/day.
or > 30 mg/hour dl.

Signs of End organ damage -

- Platelet count < 1 lakhs
- S. creatinine > 1.1 mg/dL
- Raised liver enzymes to more than Normal Value.
- Pulmonary edema
- Visual symptoms / cerebral symptoms.

Mild pre-eclampsia : Pre-eclampsia without severe features.
BP > 140/90 but < 160/110.

Severe pre-eclampsia :
BP > 160/110 OR Signs of End organ damage

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3 criterias have been removed, which were used to differentiate b/w Mild & Severe Pre-clampsia.

(i) Amount of proteinuria.
(ii) Oliguria.
(iii) IUGR.

- Chronic HT c̄ super-imposed Pre-eclampsia.

In a chronic HT pregnant female -

(i) BP suddenly becomes uncontrollable after 20 wks, OR
(ii) New onset proteinuria after 20 wks, OR
(iii) Signs of end organ damage after 20 wks.

Risk factors for PIH.

(i) Previous H/O PIH.

Recurrence rate in
Mild pre-eclampsia : 5%
Severe " " : 25%.

(ii) Primigravida.

(iii) Obesity, DM, Chronic HT, Chronic Renal disease, Extremes of maternal age.

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3 types of pregnancy in which PIH is common

Twin
Molar
Rh-ve

2 syndromes in which PIH is common.

Antiphospholipid syndrome.
Metabolic X syndrome

Protective factor : Smoking, (as it ↓ IL-2 & TNF)

Characteristic changes of pre-eclampsia are seen in Renal system. & that change is :- Glomerular capillary endotheliosis.

- GFR ↓ in PIH, because Renal blood flow ↓
∴ S. urea, S. creatinine & S. uric acid are increased in PIH.

~~Other~~

Eclampsia

= Severe pre-eclampsia + Generalised Tonic clonic seizure or coma.

- Incidence : 1 in 1500 (In India : 1-5%)

Signs of impending eclampsia in a pt. of severe pre-eclampsia :-
Epigastric pain (d/t stretching of Liver capsule)
Headache
Oliguria
Visual symp. (SCOTOMA - MC. ; Blindness can also occur, however mostly reversible after delivery)

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But when Blindness is d/t retinal detachment → Blindness is irreversible

∴ Fundal exam is done in pts.

Convulsions : Reason

∴ cerebral blood decrease

↓

Cerebral anoxia

↓

Convulsions.

Depending on the time, convulsions can be of 3 types :-

Antepartum eclampsia (mc) - worst prognosis
Intrapartum "
Postpartum "

To be called as 'post partum eclampsia', convulsions should occur in 48 hours of delivery.

* Most common MRI finding in eclampsia.

Sub cortical white matter edema

* Mcc of death in eclampsia : Intra cranial bleeding.

* HELLP syndrome - 1st described by Weinstein.

↳ A complication of severe pre-eclampsia.

↳ In DIC, coagulation profile is deranged. But in HELLP Syndrome, coag. profile is normal.

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35.

- In 15% of pt HELLP syndrome, BP can remain normal
- more common in 3rd Trimester
- MMR - 1%
- Recurrence rate : 25%
- symptoms - Nausea, Vomiting, Malaise, Epigastric pain.
- on investigation :-

H - Hemolysis
- LDH > 600
- ↓ Haptoglobin (Most specific)
- Peripheral smear shows signs of hemolysis like schistocytes, Burr cells, Helmet cells

EL - Elevated Liver enzymes.
- AST/ALT > 70

LP - Low platelet count (< 1lac).

- criteria for diagnosing HELLP syndrome : Tennesse Criteria

Platelet < 1 lac.
AST > 70
LDH > 600

- Mississippi classification :-

Class I : most severe.
Platelet < 50,000.

Class II : Platelet : 50000 - 1 lakh.

Class III : Platelet : Normal (1-1.5 lakh), But AST & LDH ↑

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Management : Immediate termination of preg.

Pt. < 34 wks — Steroids. → Then terminate Preg.
Pt. > 34 wks — Terminate.

Differential diagnosis :-

Acute fatty Liver of pregnancy. (AFP).
↳ Hypoglycemia
↳ Deranged coagulation profile
↳ Hepato renal syndrome
↳ Acute Pancreatitis.
} → Absent in HELLP syndrome.

Management of AFP : Termination of pregnancy.

Prediction of PIH.

1. Doppler of uterine artery :

It shows diastolic notch in normal preg. females, but disappears by 24 wks.

If the notch persists beyond 24 wks, it predicts PIH.

Predictors no longer used :
- Hemoconcentration
- Uric acid levels
- Hypocalciuria
- ↑ Homocysteine levels
- Giant roll over test.
  ↳ ♀ turned from Left lat position to supine position
  BP ↑ by 20 mm Hg (instead of decreasing)
  ↳ Best time to do this test : 28-32 wks.

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Drugs to prevent PIH

① Aspirin - 50-150 mg/day.

Should be given after 1st trimester; continued throughout Preg. stop 7 days before labor.

② calcium -

only given to those ♀ who are calcium deficient

* No role of following in preventing PIH.
  - ↳ salt restriction
  - ↳ Anti-oxidants.
  - ↳ Fish-oil
  - ↳ Vit C, D, E.

Management of PIH.

↳ Termination of preg.

↳ Antihypertensives.

↳ Given when BP > 160/110 (ACOG Guidelines)
BP > 150/100 (NICE ")

↳ safe:- Labetalol : Antihypertensive of choice in PIH/chronic HT/ acute hypertensive crisis
Hydralazine : 2nd Best.
α-methyl dopa
Nifedipine
Nitroglycerine
Sod. nitroprusside (used only in refractory cases)

Labetalol : 20mg initially
↓ If not controlled in 10 mins
40 mg
↓ Not controlled in 10min
80 mg → Repeat 80 mg every 10 min.

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Max dose : 220 mg

Hydralazine

5mg initial dose followed by 5-10mg

Antihypertensives c/I :-

- ACE inhibitors
- Losartan
- Diazoxide
- Diuretics.

Management of mild pre-eclampsia

- Role of antihypertensives : +/-
- Def. management : Termination of preg. (@ 37 wks)
  Mode : Vaginal del.

Management of severe pre-eclampsia

- Prevention of convulsions : $MgSO_4$
- Antihypertensive
- Def. management : Termination of preg (@ $\geq$ 34 wks)
  Mode : Vaginal del.

Indications for immediate termination irrespective of gestational age.

1. Impending eclampsia
2. Eclampsia
3. Fetal distress/ Placental abruption
4. Uncontrolled BP / Rising S.creatinine

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5. HELLP syndrome
6. Reversal of end diastolic flow in umblical artery doppler.

Umblical artery Doppler.



Normal : • forward flow

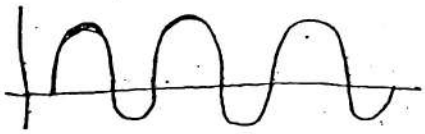
Progesterone is a smooth muscle relaxant.

• As ⊛ period of gestation ↑, resistance of vessels ↓,
∴ Diastolic blood flow ↑ i.e. S/D ratio ↓

In PIH. : S/D ratio. ↑
(Because : Resistance of vessels ↑)



→ ABSENT END DIASTOLIC FLOW
↳ severe PIH.
(Term^n of preg ≥ 34 wks)



→ Reversal of Diastolic flow
↳ Immediate termination of Pregnancy.

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Management of eclampsia

- 1st line management :
  - Airway management.
  - ↳ Then drug to ↓t convulsion : $MgSO_4$.
  - ↳ Antihypertensive. (Labetalol)
- Def. management :- Term↑ of Preg (irrespective of Gestational age)
  Mode - vaginal
  If vaginal delivery doesn't occur in 24 hrs, C.S.

Magnesium sulphate :

- Blocks NMDA receptors in Brain & Peripherally blocks Ca channels (∴ should be used cautiously ↑ CCBs (Nifedipine)).
- Mc regime for giving $MgSO_4$

Loading dose : 4gm slow IV → Followed by 10gm IM (5gms in each buttock)

Followed by

Maintenance dose :- Every 4 hrs, 5gms in alternative buttocks.

Therapeutic range :- 4 to 7 meq/L.

* Before giving next dose, check
  - Knee jerk
  - Urine output (>30ml/hr)
  - R-R. (>14/min)
- If any of these is absent, omit the dose of $MgSO_4$.

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$MgSO_4$ toxicity

- 1st sign : Absence of Knee jerk
  ↳ @ $MgSO_4$ ≥ 10 meq/L.
- Respiratory distress — @ Mg = 12 meq/L
- " arrest — @ Mg = 15 "
- Cardiac " — @ Mg = 30. "

Continue giving $MgSO_4$ till 24 hrs after delivery or 24 hrs after last seizure,

SIBAI REGIME.

Loading dose : 6 gm IV.
Then 2g/hr IV infusion — Maintenance

If convulsion recurs, then 2g IV. is given

Uses of $MgSO_4$

↳ severe pre-eclampsia
↳ Impending eclampsia
↳ Eclampsia
↳ HELLP
↳ Neuroprotective — to prevent cerebral palsy
↳ Pre-term labour

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PPH

Normal blood loss after vag. del < 500 ml
C.S. < 1000 ml
C.S. hysterectomy < 1500 ml

Blood loss more than these levels after delivery (upto 12 wks

- Primary
  - in 24 hrs
  - causes : 4TS
    - ↓Tone (mc)
    - Trauma
    - Tissue (Retained placenta)
    - Thrombin (Bleeding disorder)
- Secondary
  - 24 hrs – 12 wks after delivery
    - Retained placenta (mcc)
    - other causes :-
      - Placental site TT
      - AV malformn
      - Endometritis.

* Placenta succenturiata, Placenta bilobata, P. spuria → PPH

Diagnosis :

1) Shock index –
   HR / Systolic BP.
   Normally : 0.5-0.7
   If it becomes 0.9 - 1.1 → Blood transfusion / Immediate resuscitn

2) Obstetrical shock index
   N. = 0.7 – 0.8
   If > 1 : BT

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3rd stage of labour : Del. of Placenta : 15-20 min.

Time of 3rd stage, on active management : 5-10 min

Prolonged 3rd stage : >30 min

Signs of Placental separn :

1. Gush of Blood
2. Suprapubic bulge.
3. Lengthening of cord.
4. Fundal ht. ↑ (schrodar's sign).

(just after delivery, fundal Ht. → just below umblicus)

Methods of placental seprn

Schultz method	Duncun method
– Mc method	– Less common
– Shiny fetal side comes out first	– Dirty maternal side comes out first
– Retroplacental clot formed – Hemostatic ∴ Less Bleeding	– No retroplacental clot formed ∴ More Bleeding
– Placental sep. begins from centre	Periphery
– 80%.	– 20%.

* Line of sep. of placenta lies along zona spongiosa

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Active management of 3rd stage of labour :

(I) Uterotonics at the time of delivery of shoulder.

Oxytocin	Methergin	Syntometrine
- IM/IV infusion - Never IV Bolus because* it can cause Hypotension & cardiac arrest. - Dose = 10 IU.	0.2mg IM Side effect- Transient hyp	= oxytocin (5U) + Methyl ergometrine (0.5

WHO recommends use of only oxytocin ~~only~~ in 3rd stage.
If oxytocin is unavailable, others uterotonics should be used.

* Carbetocin = ~~toxema~~
- 100 µg IV.
- Synthetic oxytocin
- longer $t_{1/2}$

* Misoprost = PGE1
600 µg orally

OXYTOCIN

- Nonapeptide (Naturally occuring), octapeptide (synthetic)
- Synthesised in periventricular nucleus of hypothalamus.

Route	Time of onset	Duration of action.
• IM Bolus	3 min	• 3 hrs
• IV infusion	Immediate	1 hr

- $t_{1/2}$ = 3 min (3-5 min)
- Stored at 2-8°C (Kept in fridge). | At room temp., its shelf life = 3 months

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Methergin

- Methyl ergometrine
- Highly photosensitive
- Comes in brown colour ampoules.

Misoprost (PGE1)

- Used in: Asthmatic Patients. ($PGF_{2\alpha}$ causes Bronchospasm. ∴ Not used).
- Can't be used in Previous C.S. Patients.
- Side effects:- Hyperpyrexia, shivering.

(II.) Delayed cord clamping – i.e. after 1-3 min.

- Cord clamping before 1 min = Early cord clamping.
- Extra blood that goes to baby, when de cord clamping is delayed = 80 mg (50mg iron).
- Tray of the baby should be kept below the level of abdomen of mother

(III.) Delivery of placenta by CCT. (Modified Brandt Andrew technique).

(IV.) Earlier = uterine massage.

Latest WHO guideline = Intermittent uterine tone assessment.

* Disadvantage of Active management of 3rd stage of labour : RETAINED PLACENTA.

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Management of PPH :

1. Gen. Resuscitation
2.

- Atonic PPH (uterus {Not palpable per abdominally})
- Traumatic PPH. (uterus palpable abdominally) → Genital tract injury.

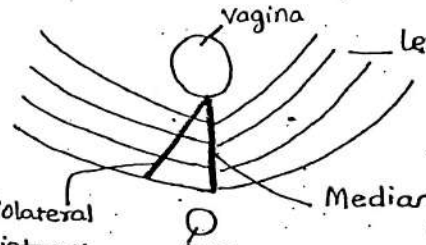
Traumatic PPH.

d/t :- (i) Perineal injuries.
(ii) Hematoma (vaginal or vulival)
(iii) Uterine rupture.

(i) Perineal tear

1° = only skin of Vagina is torn.
2° = Muscle + skin torn.
3° = Anus involved.
↳ A : < 50% ext. anal sphincter torn
B : > 50% " " "
C : Ext. + Int. anal " "
4° = All + Rectal mucosa involved

- Perineal tear can be prevented by giving episiotomy.



1) Mediolateral episiotomy. (PREFERRED)
Median episiotomy (≃ 1° tear)
↳ Muscle not cut → ∴ less Bleeding.
Repair fast, Healing fast, Less dyspareunia
↳ complication, chance of tear of anal sphincter.
∴ NOT PREFERRED.

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Episiotomy is not routinely given.
It is given only in anticipated perineal tear like:-

(i) Primi
(ii) Macrosomic babies
(iii) Instrumental delivery
(iv) Breech
(v) Shoulder Dystocia.
(vi) Face to pubes delivery.
(vii) Previous H/O Perineal tear repair.

* 1° & 2° tear: Managed in Labour room.
3° & 4° Perineal tear: Repaired in 24 hrs in OT
(spinal/epidural/Gen. Anesthesia)

Sequence of repair:
Inside to outside: Rectal mucosa
↓
Int. anal sphincter
↓
Ext. anal sphincter. → End to End anastomosis (mc method)
→ Overlapping technique (Best method).

Suture for repair :- Prolene

* Suture to repair episiotomy - Vicryl.

(ii) Hematoma

Vulval (Pudenal Artery.)
Pain, Hypotension.
Inability to pass urine
O/E Bluish tender swelling

vaginal (vaginal artery : source)

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Conservative management is done.

Surgical indic^{n} :-

If size of hematoma is increasing
Shock
Excessive pain.

} I&D + obliterate the cavity
If bleeder is present, figure of 8 suture.

Management of Atonic PPH.

① Medical management

Doc : oxytocin IV.

If oxytocin is unavailable or bleeding doesn't stop on oxytocin, then other drugs are recommended.

• Oxytocin	—	30-40 IU. IV.	
• Methergin (Methyl ergometrine) or Ergometrine	—	0.2 mg Im or IV. 0.25 mg Im or IV	Repeat every 15 min
• Carboprost ($PGF_{2\alpha}$)	—	250 µg IM. Max^{m} – 8 doses at 30 min interval.	side effect ↳ Diarrhoea
• Misoprost (PGE_1)	—	800 µg sublingual or Per rectal.	
• Inj. Tranexa (Tranexamic acid)	—	500 mg. Im/IV.	

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If medical methods fail → Mechanical methods.

I. Recommended by WHO for treating PPH.

1. Uterine massage
(It was not recommended by WHO, as prophylaxis against PPH, but it recommends for t/t PPH).

2. Use of SBEC (SB oesophageal catheter)
↳ sengstaken.
Condom Catheter / Bakeri catheter : when SBEC is unavailable

II. Method Recommended by WHO, as temporary measure till appropriate care is available. ! !

1. Bimanual compression of uterus.
2. Military antishock garment.
3. External aortic compression

III. Not Recommended by WHO : uterine packing.

If Bleeding still doesn't stop. → Surgical methods.

1. Compression sutures.
   - MC = B-lynch sutures.
   - Hayman suture.
   - Guenshella "
   - cho square "

2. Stepwise uterine devascularisation
(i) Unilateral, then B/L uterine artery ligation — at or just below Int. os.

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(ii) U/L, than B/L utero-ovarian anastomosis ligation —
@ just below ovarian ligament.

(iii) B/L ligation of ant. division of int. iliac A.
site :- 5cm distal to bifurcation of common iliac artery

↓

- Pulse pr. = ↓ by 85%.
- Blood flow = ↓ by 50%.
- MAP = ↓ by 25%.
- It helps in controlling PPH in 40-50% pts.
  But 20-30% pts. may still need Hysterectomy.

Last resort : Hysterectomy

Subtotal : uterus → is removed : Preferred
Total : uterus + cervix is removed.

If Bleeding continuous, even after hysterectomy —
Apply Pelvic Pressure pack ← Umbrella pack
Parachute pack.

Embolisation is done via femoral Artery.
using Polyvinyl Alcohol or polyurethane foam.

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Retained Placenta

- If placenta is not delivered by 30 minutes after delivery.
- It is the only drawback of active management of 3rd stage of labour
- Causes: - Previous H/O Retained placenta (Recurrence rate = 6%)
  - Adherent placenta

Management:

Placenta separated → Retained → 30 min { Oxytocin 10 IU IM/IV infusion + CCT } ⇩ Manual removal of placenta (under GA) + Antibiotics.

Adherent placenta

* WHO doesn't recommend use of Methyl ergometrine / Prostaglandins for management of Retained Placenta.

* A female goes into shock after delivery ⇨ PPH (Mcc)
  A female goes " " immediately after delivery ⇨ Uterine inversion
  A female goes into unexplained shock after delivery ⇨ Amniotic fluid embolism

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Uterine Inversion.

- Mismanaged 3rd stage of labour is the Mc cause.
- Initially the ~~see~~ shock is Neurogenic.
  Because the Inverted uterus loses its tone, there is also Hemorrhagic shock
- Mcc of death : Hemorrhagic shock.
- Management :
  1. Reposition the uterus (Johnson's method)
  2. Hydrostatic method. (O Sullivan method)
  3. Vaginal surgeries (Kustner's surgery, Spinelli surgery)
  4. Abdominal surgery (Huntington's method, Robinson's method, Haltane's method

Mc indicn for peripartum hysterectomy : Abnormal placentation. (>PPH > uterine rupture)

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Antepartum hemorrhage

Any blood loss that occurs beyond the period of viability upto delivery.
(28 wks. / 22 wks - WHO / 24 wks - India)

Causes:

Placental	Fetal
• Placenta previa	• Vasa previa
• Abruptio	
• Circumvallate	
• Circum marginate	

	Pl. Previa	Abruptio
	- Low lying placenta.	Placenta is located
	Time - USG = 3rd trimester	
- Recurrence :	5%	12%.
- Incidence :	1 in 300	1 in 200.
Risk fac:	smoking ↑ Mat. age ↑ Mat. Parity	
Highest risk :-	Prev. C.S.	
Mc risk fac :-	Prev. H/o Placenta Previa	Mc. risk :- Previous Abruptio
Other risk fac:	Myomectomy D&C Endometritis Twin Preg., ↑ Mat. AFP. Succenturiate lobe	Other risk fac : - Smoking, cocaine abuse. - Folic acid-def. - Thrombophilia, ↑ B.P. - H/o abdominal trauma - Twin Preg, Polyhydramnios, Fibroid, PROM

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Classification of Placenta Previa

Type I : Placenta is in lower ut segment.
< 2 cm from int. os (Lateral placenta previa).
- Anterior.
- Posi-

Type II : Placenta reaches upto margin of Int. os. (Marginal P.P.)
- Ant.
- Post. (Dangerous Variety)

Type III : Placenta covers int. os incompletely. (Incomplete P.P.)

Type IV : Placenta covers int. os. completely. (Complete/central P.P.)

In Type I post. & Type II post. → stallworthy sign (+)ve. ! !
↓
(Push the head of the fetus inside pelvis → Cord compression → Fetal distress)

Varieties of Abruptio Placentae.

Concealed variety	Revealed
Blood collects behind placenta	Blood comes out.

Classification : Page

Grade 0 : Retrospective diagnosis (Retroplacental clot)
Grade 1 : Bleeding PV + Pain + FHS Normal.
Grade 2 : " + " + Fetal distress / IUD.
Grade 3 : " + " + IUD + shock ± DIC.

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cause of DIC in Abruptio

Premature separ^n of placenta

↓

Tissue thromboplastin is released ⇒ DIC.

* Obstetrical conditions that lead to DIC.

1. Abruptio placenta (mc)
2. IUD
3. Amniotic fluid embolism
4. Severe pre-eclampsia / Eclampsia / HELLP.
5. Septic abortion.

* Management of DIC :

1. BT :
2. Fresh frozen plasma : when fibrinogen level < 100 mg/dl
   1 unit of FFP raises fibrinogen by 10 mg/dl.
3. Cryoprecipitate
4. Platelet transfusion : ~~when Platelet < 50000~~
5. Recombinant factor VII a
6. Heparin ±

* EACA (Epsilon amino caproic acid) — is not used in DIC.

First correct DIC → Then vaginal delivery.

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Pl. previa.	Abruptio.
C/F :- Painless, causeless	- H/o abd. trauma or PIH
- Recurrent bleeding.	- Pain in abdomen
- Bright red blood	- No Recurrent bleeding.
- Initially small bleeding k/a warning hemorrhage followed by massive bleeding.	- Dark red color blood
	- No warning hemorrhage.
Gen. examn : Condition of pt. ∝ amount of blood loss.	Abruptio (concealed) condn of pt. is not proportionate to amount of blood loss.
P/A : • uterus is soft, relaxed, & non tender	• uterus is tense, tender & rigid
FHS is easily heard / Fetal parts are Palpable	FHS is not easily heard / Fetal parts not easily Palpable.
• Ht of uterus = Period of gestation	• Ht of uterus > Period of gestn (Blood enters uterine myometrium → Gives it a blue color ↓ 'COUVELAIRE UTERUS' (Not an indicn for Hysterectomy)
P/V : C/I	
Ioc : TVS	clinical diag./TVS
Malpresentations: seen < Transverse lie (mc), Breech	Not seen.

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	Pl. previa.	Abruptio. (concealed)
IUGR :	Not seen	seen (as PIH is a risk fac)
Congenital malformn. :	seen.	seen.

Management of Abruptio.

Principles < Never wait & watch.
Never give tocolytics.

↓

Always : Immediate Termn of Preg.

Preferred mode : Vaginal del.

Indicn for C.S. : Fetal distress
Preg. < 32 wks.

If Abruptio leads to DIC, First correct DIC, Then vaginal delivery.
(No C.S. is done in DIC).

Management of Pl. previa

1. Resuscitation

↓

Immediately Terminate Preg.
↓
Active management.

↳ In hemodynamically unstable pts.
↳ Active bleeding

Let the ♀ continue Preg:
↓
Expectant management.
(Done = Lungs of fetus mature)
But, Don't put mother's life at risk.
↳ In hemodynamically stable.
↳ No active bleeding.

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↳ Gest. age ≥ 37 wks Pt is in labour	↳ Gest. age < 37 wks.
↳ Fetal distress	↳ No fetal distress.
↳ Fetal malformation on USG which is incompatible ∑ life	↳ On USG, either the fetus is Normal or, any anomaly that is compatible ∑ life.

Steps in Exp. Management : Macaffee & Thomson regime - upto 37 wks

- ↳ Hospitalize
- ↳ Arrange blood
- ↳ Rh -ve → Anti D
- ↳ Inj corticosteroid (Lungs maturity
- ↳ Any contracs present :- short term tocolytic (Nifedipine)

Mode of delivery : < USG
Double setup exam : (PV exam in OT)

Minor degree (Type I & Type II ant.) → Vaginal delivery.

Major degree & Dangerous variety (Type II post) (Type III & Type IV) → C.S.

- If pt is having persistent excessive bleeding → C.S.
- In placenta previa, Normally LSCS is done, but if placenta is located anteriorly, classical CS. is done.
- Placenta previa in present preg. ∑ previous H/O C.S. is a risk fac. for placenta accreta.

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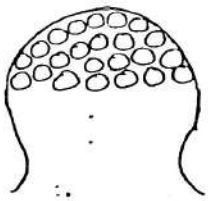
Molar Pregnancy

- Gestational T. diseases
  - 1. Partial mole
  - 2. Complete mole
  - } H·mole
- G. T. Neoplasia
  1. Invasive mole
  2. Choriocarcinoma
  3. Placental site Trophoblastic tumor
  4. Epithelioid Trophoblastic tumor

H·mole :· Bening disease c Malignant potential

Incidence – 1 in 400 (India)

Maxm incidence : Phillipers (1 in 80)



→ UNDUE PROLIFERATION OF TROPHOBLAST (At some places Hydropic degeneration)

- No Inner cell mass → Complete mole (No fetal part Present-
- some Inner cell mass → ∴ Fetal part is present- → Incomplete mole
- Bleeding PV
- ↑ βHCG
  - → Hyper-emesis gravidarum (Morning Sickness is d/t HCG)
  - → Hyperthyroidism (α unit of HCG resembles TSH)
  - → Theca lutein cyst of ovary (βHCG ≈ LH)
- Fundal Ht.

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USG : Snow storm appearance.

PIH. (< 20 wks) : D/t incomplete trophoblastic invasion.

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True pelvis.

1. Inlet : Pelvic brim. level
2. Mid pelvis : Ischial spine. level
3. outlet : Ischial tuberolisty level.

In female pelvis, shape of pelvic inlet : Transverse oval.

Pelvis makes an angle 55° ∠ Horizontal → Angle of inclination

Inlet : AP diameters :-

True conjugate → upper border of Pubic symphis. ⟷ sacral promontory (11 cm).

Obs. conjugate → Mid of pubic symphysis ⟷ sacral promontory (10-10.5)

Diag. conjugate → Lower border of Pubic sym. ⟷ " (12cm)

Transverse dia of inlet : 13 cm.

Mid pelvis : AP diameter → 12cm

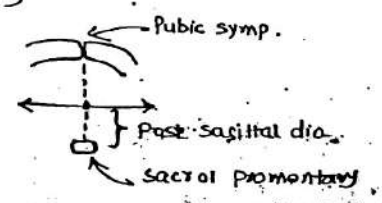
Transverse dia → Dist. b/w 2 ischial spines - 10 cm
(Bispinal/interspinous) → Smallest diameter
∴ Plane of Mid pelvis → Plane of least Pelvic dimension

Outlet : AP diameters : 11.5 to 13.5 cm. during labour.

Transverse dia : Dist. b/w 2 Ischial tuberosity - 11 cm.
(Bituberous/intertuberous)

Post. sagittal diameter :

Inlet	Mid pelvis	Outlet
4 cm	6 cm	7cm



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Which Ap diameter of inlet can be measured clinically?

↳ Diagnol Diagonal conjugate.

Diagonal conjugate − 1cm = True conjugate.

Diagonal " − (1.5 to 2cm) = obs. "

* Minm value of obs. conjugate at which vaginal delivery is possible = 10cm :

If obs. conjugate < 10cm : Pelvic inlet is contracted.

* clinically, c one hand, both the ischial spines should not be palpated.

If both ischial spines are palpated c one hand, it means the Mid-Pelvis is contracted. That means interspinous dia < 8cm

* If 4 knuckles of one hand can be inserted b/w ischial tuberosity, it means intertuberous diameter is normal & Outlet of pelvis is normal.

Intertuberous dia < 8cm :— outlet contracted.

* Subpubic angle

♀ = 85-90° (obtuse)

♂ = 80-85° (Acute).



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Contracted Pelvis.

Types :

1) Naegle's Pelvis : If anyone ala of sacral bone is absent.

2) Robert's pelvis : Both ala of sacral bone ~~is~~ are absent.

3) Tri-radiate pelvis - vit D def.

4) Rachitic Pelvis - In Rickets

Mode of del : C.S.

Time for assessment : In Primi - 37 wks.
In Multi - at onset of labor.

* If Ht. of female < 140 cm : cephalo pelvic Dis-proportion is suspected

CPD

Means either the baby is too big OR Pelvis is too small for delivery. ~~(∴ It doesn't mean - Pelvis is contracted)~~

CPD can occur at level of inlet, mid pelvis & outlet.

If mild cpd occurs at inlet - we can try vaginal del. - This is K/a Trial of Labor.

* Trial of scar : ~~trial~~ vaginal del. after C.S. (VBAC)

Trial of labour is C/I in previous C.S. patients.

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A G_1P_{1+0} female - Previous Normal del.

↓

This time mild CPD at the level of inlet

↓

Management : Try vag. del. K/a TRIAL OF LABOR.

A G_2P_{1+0} female - Previous LSCS. because of fetal distress.

↓

This time · ~~Previous~~ Pelvis Normal

No fetal distress.

↓

A G_2P_{1+0} : Previous LSCS because of fetal distress.

↓

This time Pelv mild CPD at the level of inlet

No fetal distress.

↓

C.S.

Best method for CPD assessment : Trial of labour

Best investigation for CPD " : MRI > CT > X-ray pelvimetry

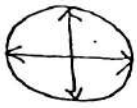
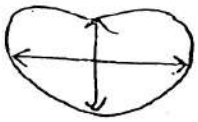
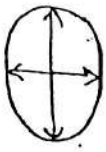
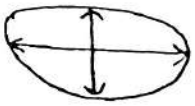
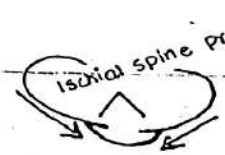
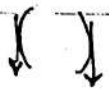
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Types of Normal pelvis.

Caldwell Mohoy Classificn

- Gynaecoid pelvis – 50%.
- Anthroid pelvis – 25%.
- Android " – 20%.
- Platypelloid " – 5%.

	Gynaecoid.	Android.	Anthropoid	Platypelloid.
Type :	Female	male		Flat bowl like
Inlet :				
Diameter	T > AP.	T > Ap	AP > T	T ≫ AP.
Shape	Transverse oval.	Heart-shaped	A-p. oval	Bowl like
				
Pelvic Side wall	 straight	 convergent.	 Narrow	Divergent.
Subpubic angle	obtuse	Acute.		

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- Diameters of fetal skull

AP	Transverse
- Longer than transverse	Miss - Bimastoid (7.5cm)
- Longest : Mento vertical (14cm)	Tina - Bitemporal (8.5cm)
2nd " : Sub mento vertical & occipito-frontal } 11.5cm	SO - Super sub parietal (9cm)
	Pretty - Biparietal (9.5cm)

* Mento vertical dia : in Brow presentn (Always c.s. is done)

- 1st step of labour : Engagement

- When largest transverse diameter of fetal skull has crossed the Pelvic inlet, it is k/a Engagement.

- Time : 38 wks (in primi) / onset of labour (in Multi)

* Lightening - The sense of relief felt by a Preg. female because head of fetus goes down into pelvis so, the Fundal Ht. ↓.
∴ Respi discomfort decreases.

- P/A : less than 2/5th head will be palpable
& P/V : Head can be felt at zero station. (@ Ischial ppine Level)

- Engaging diameters :
  - Transverse diameter : Biparietal (Exception: super-sub parietal in platypelloid)
  - Ap diameter :
    In Vertex — Sub-occipito bregmatic (9.5 cm)
    Face — Sub-mento bregmatic or Sub-mento Vertical

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Brow — Mento-vertical

- When head enters the pelvic brim, such that sagittal suture lies mid way b/w Pubic symphysis & Sacral promontory, it is k/a- SYNCLITIC ENGAGEMENT (only in 25% cases).

- If sagittal suture is deflected, then it is k/a asynclitic engagement.

Anteriorly → Ant. asynclitism (Naegle's obliquity) → More common in Multi

Posteriorly → Post. asynclitism (Litzman obliquity) → More common in Primi

* Marked asynclitism occurs in - Platypelloid pelvis.

ISCHIAL SPINE - Importance:

1. Zero station
2. Site for giving Pudendal Nerve block. (Pierce sacrospinous lig.)
3. Levator ani insertion
4. Curve of carus (forward curve)
5. Ring Pessary in prolapse is kept at this level.
6. Ext. os lies at this level.
7. Plane of least Pelvic dimension lies at this level.

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Steps of Normal labour:

1. Engagement
2. Descent & Flexion.
3. At the level of ischial spine fetal head rotates – Int. rotn (3/8th)
4. Crowning | Episiotomy is done after crowning
5. Head is delivered by extension
6. Restitution. (To relieve the twisting of the neck)
7. Now shoulders lie in LOA position. So they rotate 1/8th of the circle & lie directly behind pubic symphysis. This is visible externally in the form of ext. rotation of feral head, such that face of the baby lies towards mother's rt. thigh & occiput faces in the same direcn, in which it originally faced at the onset of labor.

Uterine contractions:

- Pacemaker: cornua of uterus (Rt. cornua > Lt. cornua)
- Rate of spread of contraction: 2cm/sec.
- Contractions depolarise the uterus in 15 sec.
- Contractions are strongest at the fundus.

Event	Intra-uterine Pressure
Contractions are palpable at	10mm Hg
" Painful	15 " "
Cx dilates	15
Fundus can't be indented	40
1st stage of labour	40–50mm.

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2nd stage of labour : 80-100 mm Hg

Adequate uterine contractions mean - 3 contractions in 10 minutes, each lasting for 45 sec., $\bar{c}$ intrauterine Pr. of ~~200-250~~ 65-75 mm Hg.

* Montevideo unit

No. of contractions in 10 min × Intra-uterine pr.

Labor begins when MV. unit = 80-120 MV units.

* Tachysystole = >5 contractions in 10 min.

When tachysystole causes fetal distress, it is N/a

Hyper-stimulation

↳ Seen in augmented labour (OOC-oxytocin)

oxytocin ARM.

Abnormal uterine action:

1. Hypotonic contractions

< 3 contracs in 10min lasting < 45 secs

& MV ≤ 180 / Intrauterine Pr. < 60.

2. Hypertonic contraction

Precipitate labour Obstructed labour/Bandl's ring.

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Pre-labour : characterised by :

1) Lightening
2) Ripening of cervix . = softening of Cx
3) False labour Pains .

False labour Pain	True labour pain
	- They ↑ in intensity, frequency & duration
	- Regular & rhythmic
opposite ⇦	- Radiates to back & thigh
	- leads to dilⁿ of Cx
	- show is present
	- Bag of membs. present.
	- Never relieved by sedation & enema

Stages of labour :

1st stage

From onset of True lab. pains upto Cx is fully dilated (10cm)

- Latent phase (≤5cm dilⁿ of Cx) → 12 hours (Nulli) / 8 hrs (Multi)
  - Prolonged : > 20 hrs (Nulli), > 14 hrs (Multi)
- Active ph. (6–10 cm) → 1 hour (Nulli) / 30 min (Multi)
  - Prolonged : 2 hrs (Nulli), 1 hr. (Multi)

2nd stage

From full dilⁿ Cx to expulsion of fetus.

3rd stage

Delivery of placenta → 15–25 min (Passive mngmnt) / 5–15 min (Active M.)
- Prolonged : >30 min (Retained placenta)

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4th stage : 1 hour observn period.
- Physiological chills are felt

- **Latent phase.**
  - True lab. pain → Cx dil (5 cm).
  - 2 actions < Effacement Cx (shortening of Cx)
    Dilatation Cx

D/d of prolonged latent phase : False labour.

Lab. Pain relieved on Sedation / Rest	Lab. Pain not relieved on sedation / Rest

- **Active phase.**

Cx ≥ 6 cm dilated.

2 actions < Dil. of cervix
Descent of fetal head.

Dil Cx	Descent
1.2 cm/hr → Nulli	1 cm/hr – Nulli
1.5 cm/hr → Multi	2 cm/hr – Multi
(1 cm/hr - WHO)	

Protracted active phase : Dilⁿ / Descent is at lesser rate.

CPD	O.P. position	Rest condⁿ
↓	↓	↓
C.S.	wait & watch.	Augment labour (oxytocin ± ARM)

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Active phase arrest.

If Cx is more than 6 cm dilated & Memb. are ruptured. & there is no dilatation for 4 hours or more, in presence of adequate uterine contractions.

Management : CS.

Second stage.

↳ said to be prolonged.

If Four Epidural	Epidural
> 2 hrs (in Nulli)	> 3 hrs (Nulli)
> 1 hour (in multi)	> 2 hrs (Multi)

Management :

If Head station ≥ +2 : Forceps

" " < +2 : CS.

Arrest of 2nd stage

Four epidural	Epidural
> 3 hrs (Nulli)	> 4 hrs (Nulli)
> 2 hrs (Multi)	> 3 hrs (Multi)

Second stage arrest → Obstructed lab. → C.S.

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Obstructed labour

- Mother : Exhausted, Tachypnoic, dehydrated, acidotic breathing.
- P/A : Lower uterine seg : stretched
  upper uterine seg : contracted tonically.
  Ring @ the junc^{n} of upper & lower seg : Bandl's ring.
  FHR distress / IUD
- P/V : Dry hot vagina. (Because entire liquor has drained out already d/t Rupture of membs.)
  Fresh bleeding
  Caput / moulding Present.
  Hematuria

Complication : 1. Rupture uterus
2. Vesico-vaginal fistula.

Management- :

1. Resuscitation
2. Correc^{n} of dehydration
3. Antibiotics.
4. Never wait & watch.
5. Never give oxytocin.
6. C.S. always (whether baby is alive/dead)

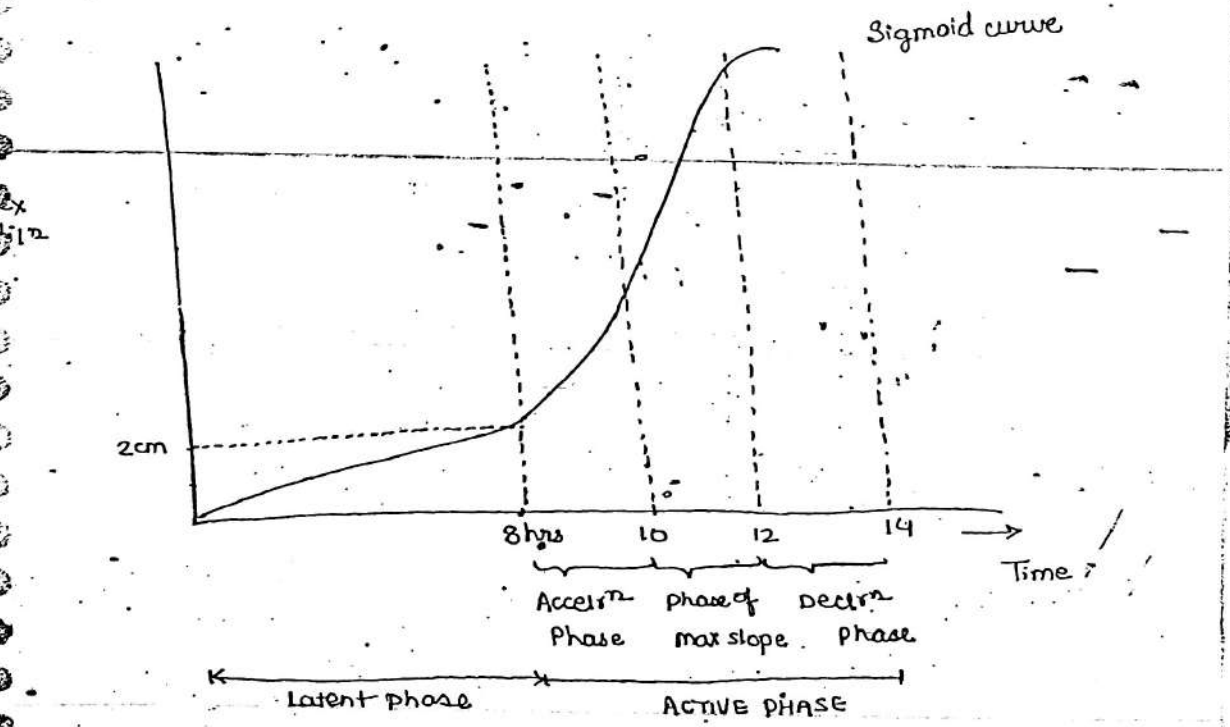
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Bandl's Ring	Schroader's ring
↳ In obstructed labour	↳ In Injudicious use of oxytocin / hyperstimulation of uterus.
↳ Pathological ring	↳ Physiological ring.
↳ can be feel P/A	↳ can be feel P/V.
↳ Retraction ring	↳ Constriction Ring
↳ Leads to Rupture uterus	↳ Doesn't

Friedman curve

- 1st partograph.



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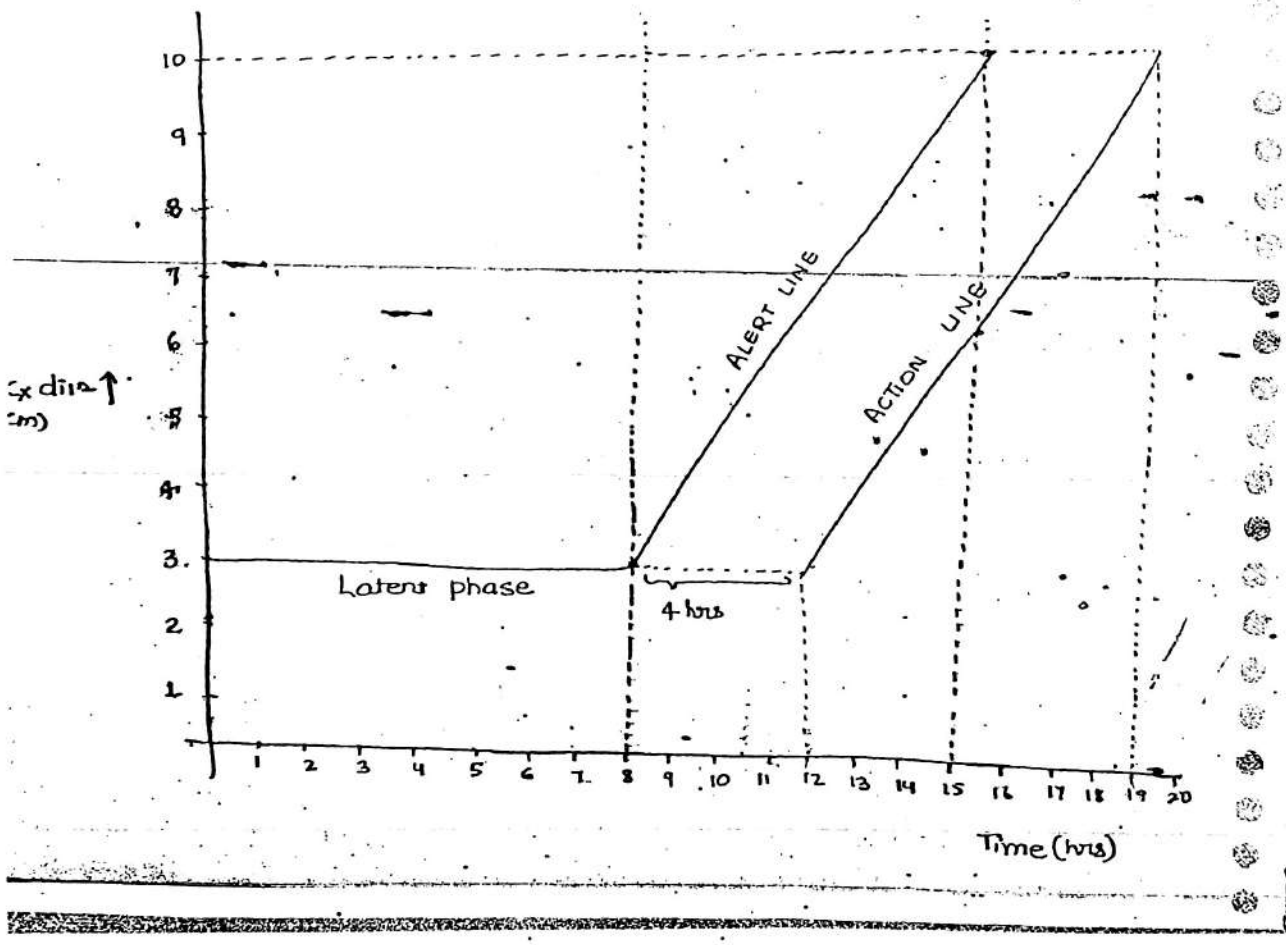
2nd partograph :- Philpot & Castle (1972)

* Concept of alert line & action line
* Labour is said to be Normal till the time it lies to left of alert line

3rd partograph : Composite partograph (by WHO)

- Given in Ghana as a part of safe motherhood initiative.
- Based on following principles :-

(i) Latent phase is said to prolonged, if it is more than 8 hrs.
(ii) Active phase ($\geq$ 3 cm) - Minm rate of Cx dila : 1 cm/hr.
(iii) Time gap b/w alert line & action line = 4 hours.

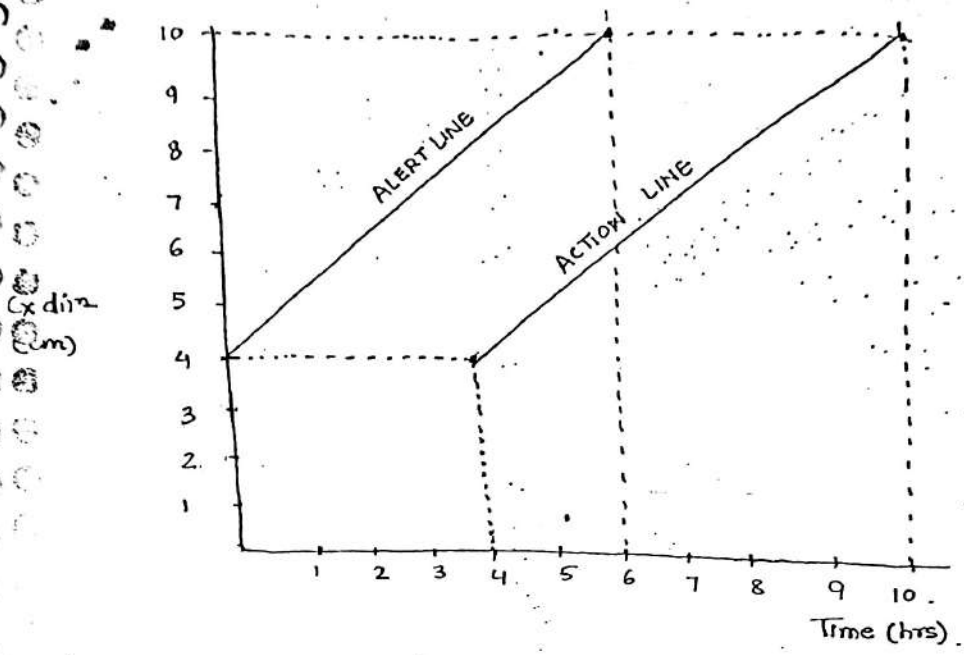


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Modified WHO partograph:

1. Latent phase removed
2. Active phase begins from 4 cm. & minm dil. = 1 cm/hr.



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Breech

Incidence : 20% (at 28 wks) , 5% (at 34 wks) , 3-4% (at term)

Because, baby spontaneously rotates at 34 wks. & becomes cephalic.

∴ Mcc of Breech : Prematurity.

Cause of Recurrent Breech : Septate / Bicornuate uterus.

Varieties :

1) Both thighs and knees are flexed : Complete / Flexed.
   - Presenting part : Feet, Buttocks & Genitalia
   - More common in Multiparous
   - chances of cord prolapse = 6%

2) Thighs Flexed. & knee extended : Frank.
   - More common in primi
   - Presenting part : Buttocks & Genitalia
   - chances of cord prolapse - minm

3) Thighs extended, & knee flexed : Knee
   - Presenting part : Knee
   - High chances of cord prolapse : ∴ C.S.

4) Thighs extended. & knee extended : Footling
   - Presenting part : Feet.
   - Highest chance of cord prolapse - ∴ C.S.

In all cases of Breech - Head of the baby is flexed.

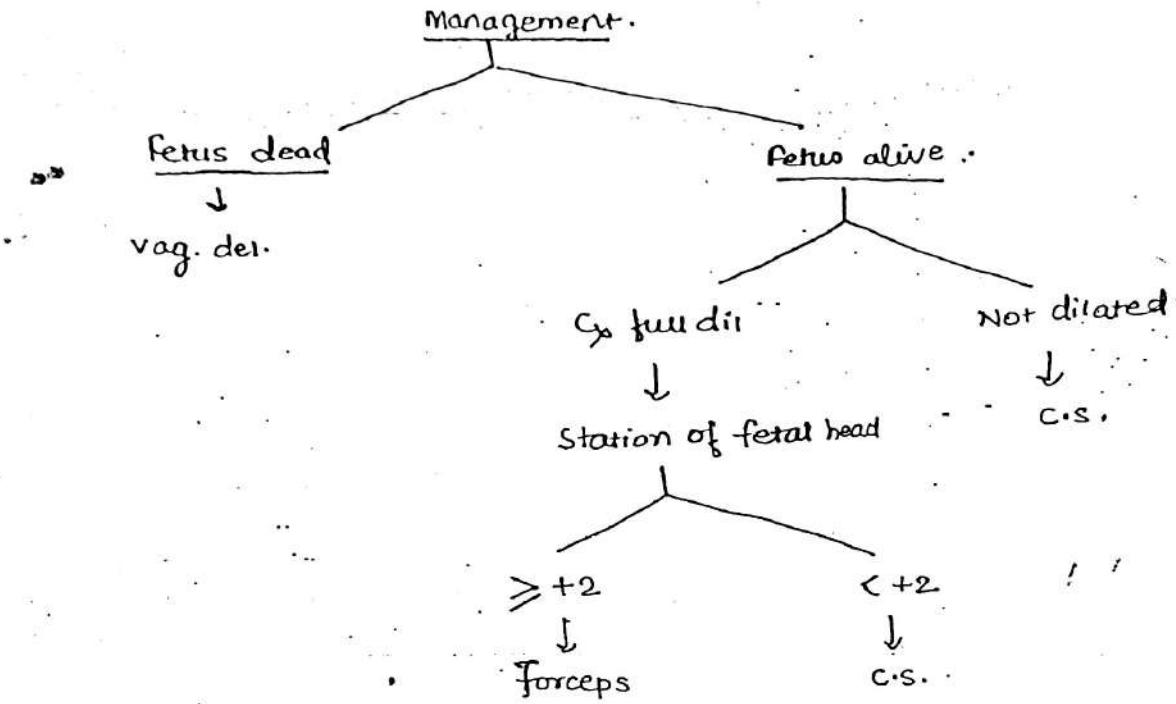
If Head of the baby is extended in Breech : Star gazer breech ∴ (Indicn for C.S.).

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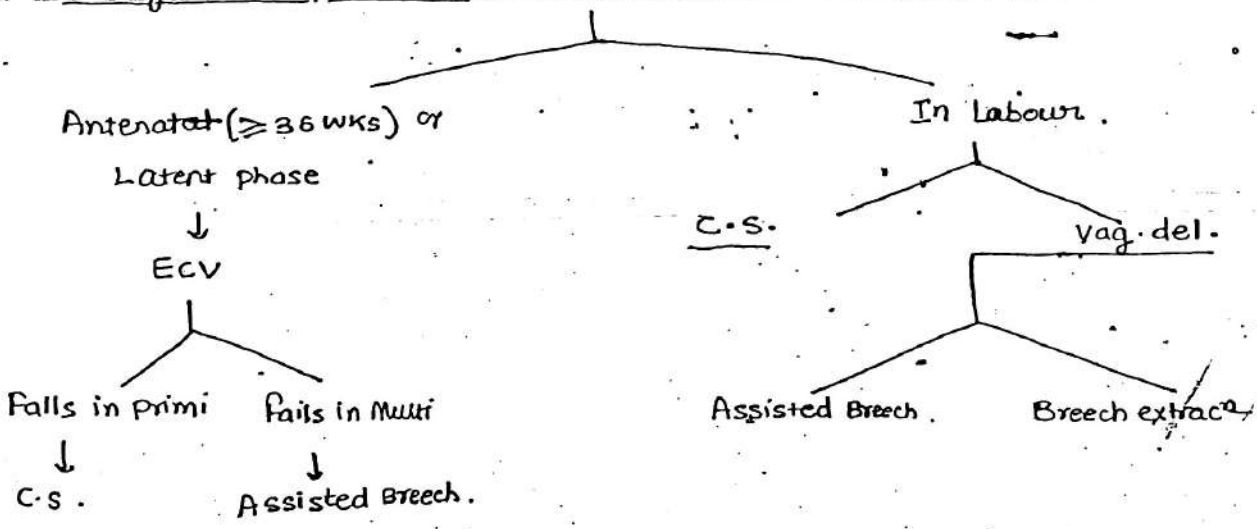
Cord prolapse.

TL > Footling > Knee.



~~Primi pt.~~, ~~37 wks~~, ~~Antenatal chk up~~ → ~~present.~~

Management of Breech.



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External cephalic Version

- An OPD procedure, doesn't require any anaesthesia.
- Pre-requisites:
  - Preg ≥ 36 wks.
  - Liquor – Adequate.
  - Memb. – Not ruptured
  - No C/I for vaginal del.
  - Single preg.

Note: ECV can be done in latent phase of labour.

continuous FHR monitoring should be done

- Outcome is better, if uterus is relaxed. ∴ single shot of Tocolytic is given :- Terbutaline.
- In primi (with breech), if any C/I to ECV – CS.
  In Multi (" "), " " – Assisted Breech.

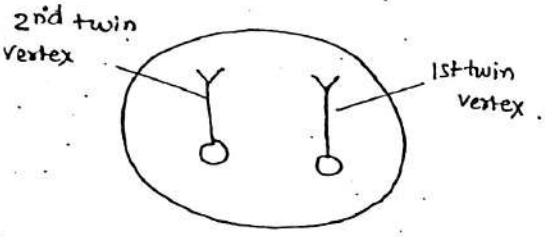
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Delivery in twins.

- Mode of delivery depends on presentation of 1st twin :-

If 1st twin is vertex : Vaginal del.
" " Breech or Transverse lie : C.S.



M/c Presentation.

↓

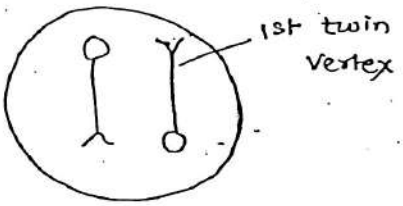
Vag. del. of 1st

↓

Don't give methergin

↓

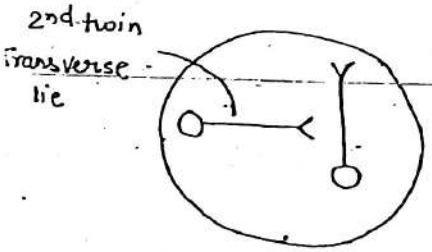
Vag. del. of 2nd.



Vag. del. of 1st

↓

Assisted Breech of 2nd



Vag. del. of 1st twin

↓

Take Pt. to OT. & Give G.A.

↓

- Insert hand inside uterus, hold the feet of 2nd twin & rotate to make it

Breech : INT. PODALIC VERSION

↓

Complete the delivery ~~out~~ any mat. effort ⇨ Breech extraction.

Internal podalic version:

Only indicated : If 2nd twin is Transvers lie.
Done in OT under GA.
There is a risk of uterine rupture.

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Assisted Breech Delivery

Denominator – sacrum
Mc position – LSA

Mostly in Breech position : Dorsum is anterior

If Dorsum is posterior : Then prague manuvere is done.

Delivery :

1) Delivery of Legs & Buttocks.

Engaging diameter : Bitrochanteric dia. (10 cm)

Manuvere used : 1. GROIN TRACTION
2. If legs are extended
↳ PINARD'S MANOUEVER!!
(Popliteal fossa)

2) Delivery of Shoulders.

Engaging diameter : Bisacromial dia. (12 cm).

Manoiwere used : LOVSET'S MANOUEVER.

3) Delivery of Head.

- Head engages in opposite oblique diameter to that of buttocks in breech.
- Engaging diameter :

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Mcc of death in Breech : Intracranial hemorrhage.

Stuck head.

Preterm — Term

- Duhressen incision on Cx. Zavanelli's manouver

Two incisions : 2 'o' clock &
10 'o' clock
Positions.

- Mc breech presentation : Frank
- " " in primi : Frank
- Least chances of cord prolapse : Frank.

Presentations in which C.s. has to be done :

- Transverse lie
- Brow
- Mento post. face

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Forcep del. (Pre-requisites)

F - Fav. position & station

O - Os full dilated

R → Membranes should be ruptured
Head rotated.

C - Uterus should be contracting

E → Episiotomy s/b given
Bladder s/b empty
Head s/b engaged.

P → Pelvis s/b adequate
No CPD

Applied along occipito mental diameter

Vaccum.

- can be applied when $C_x \geq 6$ cms dilated
- Vaccum can rotate the head of baby

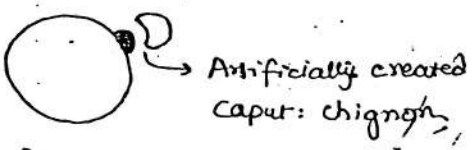
6 cm { ◇ — Ant. Fontanelle
3 cm { • — Flexion pt.
△ — Post. Font.

Vaccum is applied at Flexion pt

Pressure / Traction

Primi = 20 kg
Multi = 13 kg

Initial pr = 0.2 kg/cm²
Max ⊥ pr = 0.8 kg/cm²



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	Forcep	Vaccum
Indics:-	1) Mat. distress. 2) Fetal " 3) Prolonged 2nd stage of lab. 4) Prophylactically in H.D. pts.	same indics except :- - Vaccum isn't used in fetal distress In H.D. pts. – Vaccum is preferred. (Because we don't have to bring the pt. in lithotomy position in Vaccum)
Advantages:	Presentations in which forceps can be used: 1) All presentations except in those where CS. has to be done (ie Transv. lie / Brow / Mento. Post) • Vertex • OP • Breech – after coming head ↳ Piper's • Face – Mento ant.	can be used only in Vertex presentation (Disadvantage)
	2) Forcep can be used in Pre-term babies.	– can't be used in pre-ter (Disadvantage)
Complics:	1) Lead to more of mat. injury Fetal injuries more common ∈ forcep: • Facial N. palsy • Brachial plexus injury.	– Leads to more of fetal injur Fetal injuries more common :- • Cephalhematoma • Retinal injury • Sixth N. palsy.

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Max no. of pulls
↓
3
↓
Not delivered
⇒ Failed forcep.
↓
C.S.

Max no. of pulls
↓
3
↓
Not delivered
⇒ Failed vacuum
↓
C.S.

Absolute C/I for Instrumental del.

1) CPD / contracted Pelvis.
2) HIV female
3) Known coagulopathy in fetus
4) Osteogenesis imperfecta

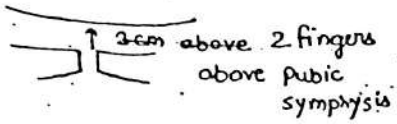
Caput succaderum	Cephalhematoma.
↳ Scalp edema.	↳ Subperiosteal bleeding
↳ Swelling above the ~~present~~	↳ Swelling below periosteum
Periosteum / bone ↓ ∴ It can cross the suture lines.	↓ It can't cross.
↳ Present as soon as baby is born & starts ↓ after birth	↳ It presents few hours after birth. It ↑ for 3 days & then ↓.

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Caesarean section

Skin incisions



3cm above
Pubic symphys

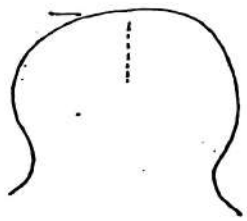
Suprapubic curved incision in which rectus sheath is opened using sharp incision K/a sharp technique.

PFANNENSTIEL INCISION

Little above the Pfannenstiel incision.
Suprapubic transverse incision where rectus sheath is opened using blunt incision

JOHN COHEN INCISION

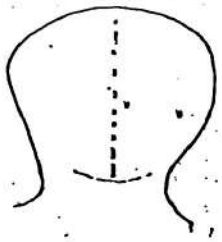
Uterine incisions



Sanger's incision.

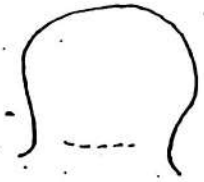
Ut. rupture chances = 4-9%.

Classical C.S.



Rupture = 4-9%.

T-shaped incision



Rupture = 0.2-1.5%.

⇦ Kerr incision (LSCS)



Rupture = 1-7%.

Kronig / De Lee incision (LSCS)

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Current indicⁿs for classical C.S.

1) Ca cervix (absolute indication).
2) Dense adhesions b/w bladder & lower ut. seg.
3) Post mortem c.s.
4) Impacted shoulder (Transverse lie).
5) Placenta previa (Placenta on ant. wall).
6) VVF Repair
7) A very pre term c.s. (because lower seg. is not formed by that time)

Kronig incision:

Indication : Constriction Ring.
Danger : It can extend into Cx.

* Ideal time to conceive after c.s. = 18 months.
Minm " " " = 6 months.
Level of anaesthesia in c.s. = T_4 seg.

~~If Elective c.s. is done for done for no medical reason, then it~~ should not be done at 39 weeks..

Absolute c/I for VBAC.

1) Types of incisions on uterus ← Classical, T shaped, Kronigs.
2) H/o Prev. ut. rupture.
Recurrence of ut. rupture = 32% (classical cs), 6% (LSCS)
3) Any Surgery where ut. cavity was opened ← Hysterectomy, Myomectomy.
4) H/o Prev. 2 LSCS.
5) If any c/I to vaginal delivery (Eg.- Placenta Previa)

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Relative c/I :-

- Macrosomia in current preg.
- Breech in current preg.
- Post term preg

* Most imp. prognostic factor for VBAC : Previous H/O Vaginal delivery

Closure of uterine incision.

Single / Double layer.

No effect on outcome of VBAC.

* If there is H/O Prev. Puerperial infection VBAC is a relative c/I as previous scar would not have healed properly.

* VBAC can be done only if indication for previous C.S. was a non-recurring cause.

* Induction is not c/I in previous LSCS pt. (Doc : oxytocin)

*

Bishop score.

D - Dilatation of Cx (most imp. parameter)
P - Position of Cx
E - Effacement of Cx
S - station of fetal head
C - Consistency of Cx

Induction of labour is successful ∵ a score of 9 or more.

If score < 4 ⇨ First ripen/soften the cervix → then induce labour.

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OT.

Agents used for Cx ripening & Induction of labour.

PGE_2 (dinoprost)

PGE_1 (misoprost.) → ~~oo~~ C/I in previous C.S. for induction of labour (oxytocin is used instead).

Augmentation of labour should be avoided in prev. CS.

Labour analgesia is not C/I in prev. C.S.

Rupture uterus

↳ Rupture of entire uterus along ± serosa/peritoneum.

If peritoneum is intact → K/a scar dehiscence.

Sign of Impending Rupture	Sign of Rupture.
1. Fetal Tachycardia	1. FETAL BRADYCADIA. (Mc sign/Earliest sign/ Most consistent sign). FH.S. absent as the baby dies.
2. Mat. Tachy "	2. Mother – Tachy, Hypotension (shock).
3. Scar tenderness.	3. Hematuria, Bleeding PV
4. Microscopic hematuria.	4. Loss of station
⇩	5. Absent ut. contracn
Immediate C.S.	⇩
	Immediate Laparotomy

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Ectopic pregnancy

↳ Pregnancy outside uterine cavity.

Mc site : Fallopian tube.

↓

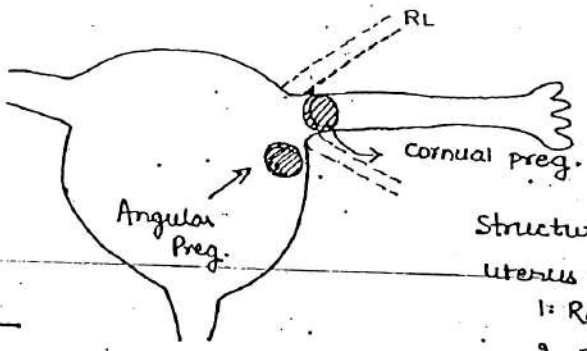
Mc - Ampulla (because fertilisn occurs in ampulla).

Lc - Interstitial (FT).

Least common site (overall) — caesarean scar or cervical ectopic

Heterotopic ectopic — Twin - 1 ectopic + 1 intrauterine
Ectopic occuring Non functional horn of bicornuate uterine.

Cornual preg : Ectopic preg in interstitium of FT.
↳ should be differentiated from Angular Pregnancy.



Structures attached to CORNUA of uterus :-

1. Round ligament — Ant
2. F.T. ↓
3. ovarian ligament. — post.

Angular pregnancy : Intrauterine Preg. lying close to angle of uterus.

If Preg. is lateral to Round lig. (RL) :- cornual preg
" medial " :- Angular "

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Highest risk for ectopic.

1) Prev. H/o Ectopic Preg. < 1. Prev. Ectopic = 15%
2. " " = 30%

2) H/o Tubal surgery.

* Mc risk factor : PID

Other — Multiple sex partners.
Early age of intercourse.
In utero exp to DES (Diethyl stilbesterol).
Infertility.
ART
Smoking.
Tubal adhesions (Endometriosis).

Contraception

All contraceptive agents reduce the risk of (absolute) Ectopic.
But if contraception fails ⇨ Ectopic.

M/c : Sterilization > IUCDs > POP

(Mc: Progestasert)

Least common : OCPs.

Fate of ectopic pregnancy.

- Abort (= Tubal abortion) → Mc : @ Ampulla
- Continues to grow → Tubal Rupture → Mc : @ Isthmus

* Ectopic Preg. has shortest life span : @ Isthmus.

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Ectopic preg. Lasts longest in :- 1) Abdominal preg.
2) Heterotopic "
3) Tube :- Interstitium.
(Because myometrium supports ectopic. → upto 12 wks
∴ Most dangerous site)

Symptoms of Ectopic preg :

- Most consistent symp. : Pain abdomen.

Ruptured ectopic.
↓
Pain is d/t hemoperitoneum
↓
Irritates Diaphragm
↓
Shoulder tip pain
(DANFORTH SIGN)

Unruptured ectopic
↓
Pain is d/t stretching of tube.

- Triad of :- • Abd. pain
• Amenorrhea (6-8 wks)
• Bleeding PV

Signs of Ectopic. preg :

- on P/V :- uterus is enlarged
- cervical movement is tender: characteristic
  - Ectopic
  - PID.
- Adnexal mass can be palpated (in 50% cases).

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Diagnosis

- **Ruptured Ectopic**
  - ↳ Pt in shock
  - ↳ Amenorrhea (6-8 wks) → Bleeding PV & Abd. Pain.
  - ↳ UPT +ve. (Urine preg. test)

⇩

Immediate Laparotomy.

Surgery :- Ruptured tube → Remove it → salpingectomy (in both nulliparous/multi)

* Instead of laparotomy, laparoscopy can be done also, if vitals of pt. is stable.
* No role of medical/expectant management or any other surgery.

- **Unruptured**
  - ↳ UPT (+)ve in 99 % cases.

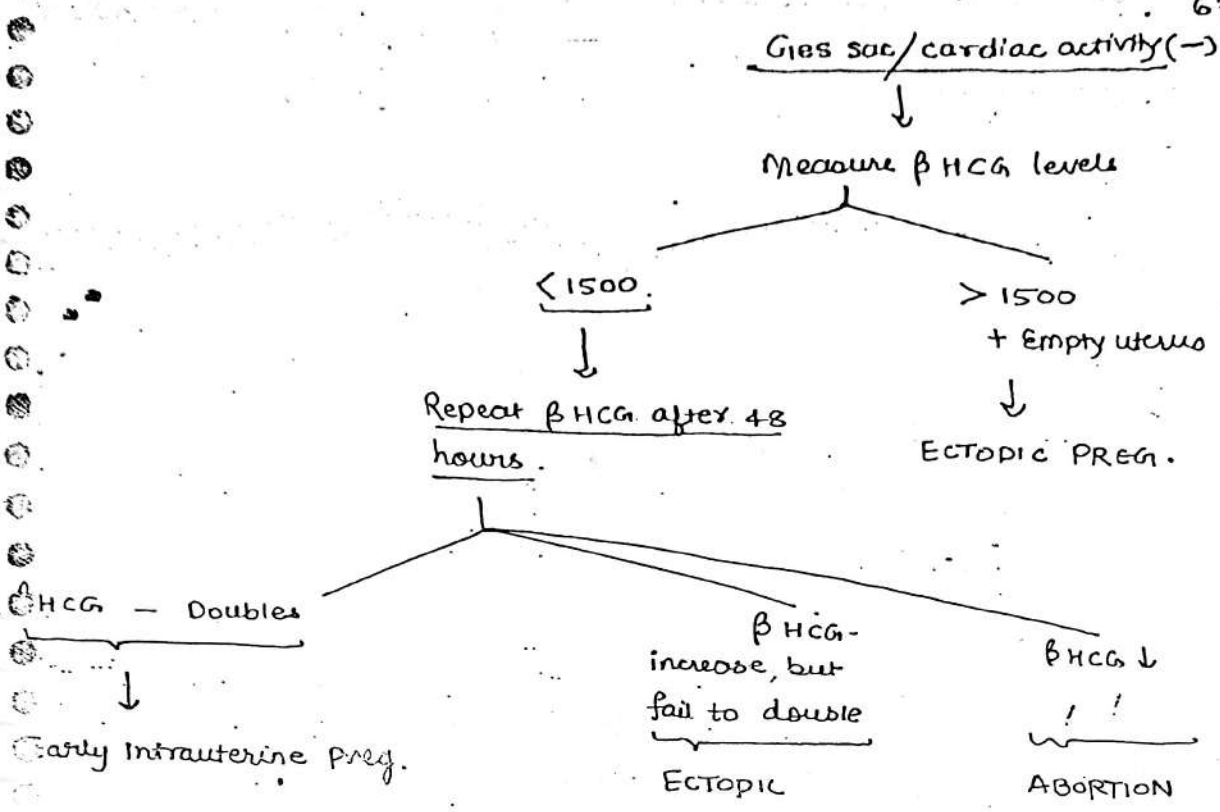
↓

IOC = TVS

- Ges. sac (+) → Intra-uterine preg.
- Ges. sac (-)/ Empty uterus → 1st finding on TVS that suggests ectopic preg. → CHECK ADNEXA (Tube & ovary)
  - Ges sac/cardiac activity + → Doppler USG → ↑ Vascularity in Tube (RING OF FIRE APPEARANCE) → ECTOPIC
  - Ges sac -/cardiac activity -

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Best Investigation : TVS + β HCG.

Gold std : Laparoscopy

Other investigations : 1) S. progesterone

2) Culdocentesis → Blood in case of ruptured ectopic

Investigations Not done : HSG
Colpotomy (It is done in pelvic abscess)
Hysteroscopy

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Management of unruptured ectopic

- **Expectant management** : wait & watch.

  Criterias → 1) Pt. hemodynamically stable
  (ie. Ectopic unruptured)
  2) Cardiac activity absent in Ectopic preg.
  3) Size of ectopic < 3 cm.
  4) βHCG < 200 & should be decreasing.

* Risk of rupture in expectant management.

- **Medical management** :

  Doc : METHOTREXATE (single dose)

  Criteria – 1) Pt. hemodynamically stable
  2) Cardiac activity Preferrably absent.
  3) size of ectopic < 3.5 cm (if cardiac activity +ve)
  < 4 cm (if cardiac activity absent)
  4) βHCG < 5000

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Doc : Methotrexate : 50 mg/m² I.M.

Day 1 = Injection

Day 4 = check β. β HCG.

Day 7 = check β. HCG. – If values fall more than 15% from 'Day 4' value

⇩

THERAPY SUCCESSFUL.

If the fall is < 15%,

Repeat inj. Methotrexate : – Day 1 (New)

check β HCG → Day 4

check β HCG → Day 7

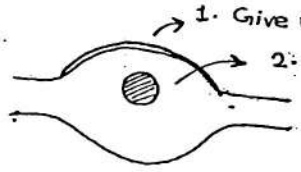
Failed Med. management :- 3 times injection given → Still the required fall in β HCG is not seen.

Surgical management :- Laparoscopy (Preferred)
Laparotomy

Surgery depends on Parity of female.

♀ is Multiparous + completed family = salpingectomy

♀ is Nulliparous + Not completed family = LINEAR SALPINGOSTOMY



1. Give incision.
2. Remove ectopic preg by Hydro-dissection
3. Leave the tube as such.
(Don't close incision site)

⇩

Same procedure, but if suture is applied in the end :→ SALPINGOTOMY

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Ectopic pregnancy occurs in young females.
So salpingo-oopherectomy should never be done in them.

Indications of salpingectomy in Ectopic :-

1) Ruptured ectopic preg
2) In unruptured ectopic, if family is complete
3) If size of ectopic > 4 cm.
4) If Hemostasis can't be obtained.

Site of Ectopic	Diagnostic criteria
1. Cervical ectopic	Rubin / Palman criteria.
2. Abdominal ectopic	Studdiform criteria.
3. Ovarian ectopic	Spigelberg criteria.

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by.

Abortion

Loss of fetus.

- Before the period of viability (20 wks).
- < 500 gm. wt.

Abortion

- **Spontaneous** (single episode)
  - 1st Trimester
    - MCC: chromosomal anomaly (50% cases)
  - 2nd trimester → same (20% cases)
- **Recurrent** (≥ 3 abortion)
  - 1st trim
  - 2nd trim

Spontaneous

Mc time : < 8 wks.

- Chromosomal anomaly → Aneuploidy ✓ (Best)
  - Trisomy
  - Monosomy X = 20% of 1st trim. abortion
  - Trisomy 16 = 16% of abortions.
- Germplasm defect
- Infections

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Recurrent abortions

- 1st trimester
  - Mcc : • Idiopathic.
    - ↓
    - • Immunological. (Antiphospholipid antibody synd)
    - ↓
    - • Endocrinal (DM, PCOD, Thyroid, ↑ Prolactin, Luteal phase defect)
    - ↓
    - • chr anomalies (4%) (Balanced translocⁿs of chromosomes)
  - ∴ Karyotyping should be done.
- 2nd. trimester. (d/t : uterine anomalies)
  - Congenital
    - ↓
    - Septate / Bicornuate uterus
  - Cervical incompetence.

- TORCH infecⁿs never leads to Recurrent Pregnancy loss (RPL).
- only infecⁿ that leads to RPL – syphillis.

Syphillis follows 'Kassowitz Law' → As the no. of preg loss increases, the gestational age at which loss occurs also increases. so syphillis can't lead to Early RPL.

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Cervical Incompetence:

1) Sudden painless dilⁿ of Cx ∴ expulsion of fetus.
2) Mcc of RPL in 2nd trimester.
3) Gestational age decreases ∴ every preg. loss (Opp. to syphillis)

Diagnosis.

Non-Pregnant.

Pass Hegar dilator no. 8
Can't any resistance..
(through int. os).

On Histocervicography:
Funnel shaped appearance.

Pregnant.

- TVS (10c)
  - Length of Cx < 2.5cm
  - Diameter of os > 8mm
  - Shape of Cx = Ushape.
  - Funneling of membranes.

* Cervical index = Funnel length + $\frac{1}{\text{Endocervical length}}$.

Normal = 0.32
In cervical incompetence > 0.52

Management: Cervical circlage (Put the stitch)

Prophylactic (History indicated)
- ≥ 3 abortions of 2nd trim. or 3 times Pre-term lab.

Therapeutic (USG-indicated)
- ≥ 1 mid-trimester abortion + Cx length < 2.5 on USG.

* It should not be done if there is no H/o abortion & Cx length < 2.5.
- Cx length > 2.5, but funneling of membrane.

Rescue (Physical exam indicated)
- Cx dilⁿ < 4cm & Membrane should not be ruptured.

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C/I for circlage -

- current infection
- Memb. ruptured
- Gross congenital anomaly
- Cx > 4cm dilx.

Methods of doing circlage -

1) Vaginal method :-

(i) Mc Donald : Mc method.

(ii) Shirodkar method.

(ii) Abdominal method :

It is done only when vaginal circlage fails.

Benson & Durfy circlage.

ANTI-PHOSPHOLIPID ANTIBODY SYNDROME (APLA).

↳ Antibodies are formed against phospholipids

3 antibodies :- 1) Lupus anticoagulant (MC)
2) Anticardiolipin antibody
3) Anti β2 microglobulin antibody.

↳ Pathology : These antibodies lead to thrombosis in arteries & veins (including placental blood vessels)

PIH ↵ ↳ ∴ size of placenta decreases

Blood flow absent → Abortion, IUD, Still birth.

Blood flow to fetus ↓ → It can lead to IUGR.

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Diagnostic criteria : Revised sappro.

1 clinical & 1 lab criteria should be fulfilled.

clinical → 1) Arterial/venous Thrombosis. (Superficial/Deep).
2) ≥ 3 losses in < 10 wk
3) ≥ 1 loss in > 10 wk.
4) Atleast one preterm delivery secondary to PIH or uteroplacental insufficiency.

Lab. : - Presence of LAC (Lupus anticoagulant) ab.
- Presence of IgM/IgG anticardiolipid antibody
- β_2 micro globulin Ab.

} on 2 occassions 12 weeks apart.

Other findings - APTT ↑ , PT = Normal
- Russel viper's venom clotting time ↑.

Management.

LMW Heparin + Low dose aspirin.
(80 mg OD)

Doc: Enoxapin

Should be started if - intrauterine preg.

Should be started as soon as UPT +ve

Continue throughout preg.

Heparin stopped @ onset of labour
Aspirin " @ 5-10 days after labour

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Types of abortion

Type of abortion	complain	P/A	Int. os	Diagnosis	Imp. points
1. Threatened (Process of abortion has begun, but is reversible)	Spotting (1 or 2 drop of blood) ± Abd. pain.	Ht. of ut = POG	closed	USG = Live intra-uterine preg.	Mngmnt: 1) Reassuran 2) Avoid intercourse 3) Value of bed rest & Progesterone is uncertain. 4) Anti-D.
2. Inevitable Process of abortion has reached a stage from where it can't be reversed)	Bleeding + Pain abdomen.	Ht. of ut. = POG	open.	Live/dead fetus	Insert vagina misoprost to complete the process.
3. Incomplete abortion Process of abortion as begun. & Products of conception starts coming out)	Pain + Bleeding + Poc coming out.	Ht. of ut < POG	Open. + Poc coming out.	USG: Always do USG. to know the amount of	Conservativ mngmnt if
4. Complete abortion. Entire process of abortion is over)	Initially pain & bleeding were Present. Poc came out but now bleeding has stopped	Ht. of ut < POG.	closed	Empty Uterus	—

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Missed abortion (Fetus is dead inside the uterus but pt. is unaware)	No bleeding. Dirty brown discharge.	Ht. of ut. closed < POG	Mean sac diameter > 25 mm OR Crown-rump length ≥ 7mm & No fetal heart beat.	↓ βHCG levels.

Anti-D prophylaxis for early bleeding :

- Ectopic Preg in Rh⊖ve ♀
- Following Suction-Evacuation of H.mole — in Rh⊖ve ♀
- All abortions > 12 wks (including threatened)
- All abortions where uterus is evacuated medically/surgically.
- Threatened abortion < 12 wk, if bleeding is heavy, recurrent or associated c Pain.

Anti-D is not given in :-

- Threatened abortion less than 12 wks.
- Complete " " " , if nothing has been done.

Dose = 5 µg (< 12 wks)
300 µg (≥ 12 wks)

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Methods of MTP :

Best method upto 7 wks : Medical abortion

≫ 7-15 wks : Suction Evacuation

≥ 15 wks : Prostaglandins (PGE1/E2)

1st trim

↳ Medical (till 9 wks/7 wks in India)
↳ Suction Evac.
↳ Menstrual regulⁿ
↳ Manual vaccum aspirⁿ

2nd trim

↳ PGs
↳ Suction Evac. using Drum forcep.
↳ Extra-amniotic Ethacridine
↳ Intraamniotic
↳ Hypot

Med. abortion

< 7 week Pregnancy.	7-9 wks. Pregnancy.
↳ Day 1 = 200 mg Mifepristone	↳ Day 1 = Same
↳ Day 3 = 400 µg Microprost oral or vaginal.	↳ Day 3 = 800 Misoprost (vaginal)
↳ Day 15 = Clinical examⁿ to ensure that abortion is complete.	↳ Day 15 = Same.

* If < 1% = curettage is required
* Misoprost is teratogenic → Mobius syndrome.

Imp. Points on Suction Evacuation :-

↳ Karmann cannula
↳ Pressure generated = 600 mm Hg
↳ Before inserting the cannula Dilate Cx using Hegar's dilator
↳ ~~If perforation of uterus~~

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If Perforation occurs during Procedure.

- While dilating → Wait & watch
- while doing suction & Evac → Don't remove canula from ut. & Immediate laparotomy.

Menstrual regulation

Done in 3 weeks after a missed period. ie. 7 weeks of amenorrhea.

Manual vacuum aspiration

- Take a 60 ml syringe & a cannula.
- Pressure : 660 mm Hg.
- used upto 12 wks of preg
- No electricity required.

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Endometrial Hyperplasia

Simple ① ↓ — without atypical cells
Complex ↓ ④ — with atypical cell
③ ②

① - Na least chance of malignancy = 1% | ≃ cystic glandular Hyperplasia
② - 3%
③ - 8%
④ - 30%.

- When a pt. comes ♀ excess bleeding,

1st investigation : TVS

On TVS

Premenopausal ♀ — Post meno. ♀

Endometrial thickness

≥ 8mm — ≥ 4mm.

Curettage & Biopsy. (HPE)

Endometrial hyperplasia

♀ out atypia. — ♀ atypia.

Simple — complex

⇩

DOC = Progesterone
Medroxy progesterone Acetate } X 6 months
in last 12-14 days of cycle }

⇩

Hysterectomy
(If pt. is young & refuses Hysterectomy)
↓
Megestrol.
(Progesterone).

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Endometrial ca

	Type 1	Type 2.
-	Adeno ca : Grade 1, 2	Papillary serous tumor clear cell tumor Adeno ca : Grade 3.
-	Mainly d/t excessive estrogen	
∴	Endometrial hyperplasia present	— Absent.
-	More common in obese ♀	— In Thin ♀
—	Better prognosis	
—	In pre & post menopausal ♀	— only in post menopausal ♀
—	White ♀	— Black ♀
—	↑w PTEN. & KRAS	— P53.
—	Stable	— Aggressive.

Management of Endometrial ca

Stage IA (Grade 1,2)	TAH + BSO, NO post-op therapy
Stage IA (Grade 3)	TAH + BSO, + Radiotherapy
Stage IB.	TAH + BSO; + Radiotherapy
Stage II	Wertheims. + Radio
Stage III	" + Radio + chemo.
Stage IV.	- Debulking surgery + Radio + chemo.

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Post menopausal Bleeding

1st investigation : TVS

- Endometrial thickness < 4mm → Repeat TVS after 3months
- ≥ 4mm
  - IOC: Endometrial aspirn biopsy using Pipelle, Vibra-aspirator & Karmann curette
  - Gold std. investign: Fractional curettage.

If on TVS, instead of generalised thickening, we get a polyploidal mass, in that case, instead of endometrial aspirn biopsy, Hysteroscopic guided biopsy should be done.

Lymphadenectomy : Indicns (in endometrial ca).

- Tumor size > 2cm
- Tumor Histology – Endometroid grade 3 / papillary serous / clear cell
- More than ½ of myometrium involved.
- Tumor extends to cervix / extra-uterine

If all condns are present : Pelvic & Para-aortic Lymphadenectomy
If size of tumor > 2 cm, & no other condn :- Pelvic Lymph...
If none of these condns present (eg.- Adeno ca grade 1 & 2 belonging to Stage IA) → No lymphadenectomy.

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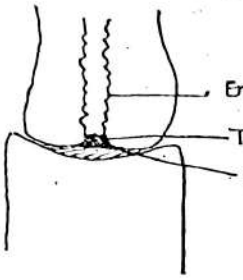
Anatomy of cervix

2 parts :

1) Endocervix : Closer to uterus — columnar epi
2) Ecto " : Closer to vagina — Squamous epi ⟷ Squamo-columnar junctn (Transformn zone)

↓

at level of ext. os.



Transformn zone → Keeps on changing in response to age & hormonal influence

- Moves out-in: • Preg, Puberty & OCP
- Moves inside: • ↑ age.

On per speculum exam :- Endocervix : Red
Exocervix : Pink
→ Transformn zone : WHITE.

- Mc site of Cx ca : Transformn zone
- Mc variety of Cx ca : Squamous cell ca
- Mc site for adenoca : Endocervix.

→ In all females, columnar epithelium of endothelium changes into squamous epithelium of exocervix → K/a Metaplasia
— ↳ physiological ; Not precancerous.

- Squamous epithelium of vagina & exocervix has 4 types of cells :

1. Basal & Parabasal cells : They predominate when there is no hormonal predominance. Ex. In post menopausal females.
2. Intermediate cells : Are Basophillic cells, which predominates, when Progesterone is predominant. (Preg & 2nd half of Menstrual cycle)
3. Superficial cells : Eosinophillic cells, which predominate, when there is excessive estrogen. (1st half of menstrual cycle)

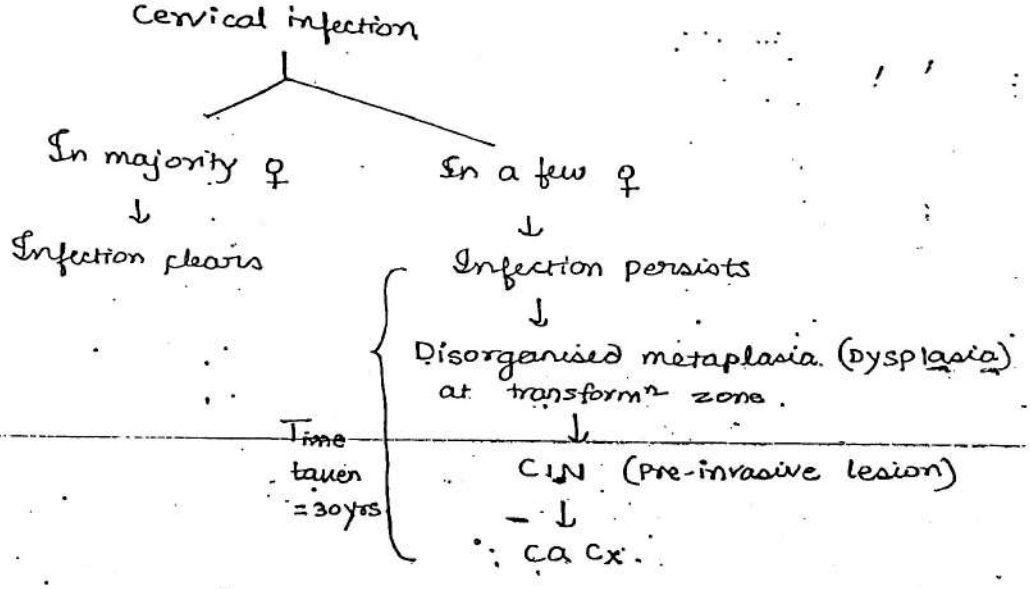
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* For Hormonal study, sample should be taken from lat. side of Vagina.

Cancer cervix

- mc cancer in India in females — Breast ca
- mc cancer of genital tract in females —
  - Worldwide : Cx ca
  - India : Cx ca
  - Developed countries : Endometrial ca



- **Dysplasia :**
  - Always pathological ; pre cancerous ; only in few females.
  - Dysplastic cells — Big cells & Big nucleus & less cytoplasm.
    ∴ ↑ Nucleo-cytoplasmic ratio.
    — Mitotic figures.

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Q CIN = cervical Intra-epithelial Neoplasia.

< 1/3 cervical epithelial dysplastic = CIN 1
1/3 – 2/3 " " = CIN 2
> 2/3 " " = CIN 3
} (21-30 years age group)

Complete Cx epithelia dysplastic
↓
overlying memb.
Intact → ca in situ (30-35 years age)
Broken → Invasive ca. (35-39 years – 1st peak, 60-64 years – 2nd peak)

Bethseda classific^n

CIN 1 = LSIL = Low grade sq. intra-epithelial lesion.
CIN 2,3 = HSIL = High grade. " "
(* LSIL = Low sq. Intra-epithelial lesion)

Risk factors for CIN / ca Cx.

- Early age of intercourse, Multiple Partners.
- Early age of 1st child birth.
- Multiparity (* Low parity is a risk fac for, ca endometrium & ovary)
- Low socio-economic status.
- STDs
- Smoking. (sq. cell ca)
- ocp. (Adeno ca).
- Most imp: • Hpv infection.

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HPV (Booklet)

- When HPV affects cervical epithelium, the changes which are seen are K/a - Koliocytic change. (perinuclear halo).

- High risk subtypes : HPV 16, 18 (70% cases)
  Other High risk - HPV-31, 33, 45, 52, 58.
  Low risk - HPV-6 & 11 → cause Genital warts (condyloma acuminata)

- Babies born to females c Genital warts have laryngeal Papillomatosis. Hence active genital wart Pt. in labour → C.S.

- Viral protein responsible for malignant transform^{n}.
  E_6 - affects P 53 gene
  E_7 - " Retinoblastoma gene.

- Viral protein required for replic^{n} : E_1 & E_2.

∴ Screening test Cx : Pap smear

↳ 1st performed by - Georgios Papanikolaou

↳ Starts @ 21 years of age.

↳ B/w 21-29 years : Screening should be done 3yearly using only Pap smear

↳ 30-65 years : 5 yearly screening using Pap smear (increases specificity) + HPV DNA (increases sensitivity).
OR
3yearly pap.

↳ ♀ is HIV (+), or exposed to DES : Annual Screening - till we get 3 consecutive Normal. & then 3 yearly screening.

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- Screening is to be continued for 65 years if last 10 years Pap - Normal

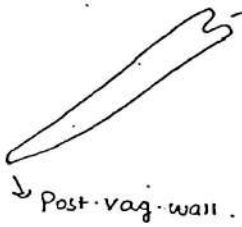
OR

75 years - if last 10 years Pap - Suspicious.

- After vaccine - screening still continues.
- After Hysterectomy for benign cause - screening stops.

Instrument used for pap smear

① Ayer wooden spatula



② Wooden ~~Brush~~ Cytobrash.



Tz.	Endo Cx	Post. Vag. wall
1st slide.	2nd slide	3rd slide = control slide.

Fixative = 95% Ethyl alcohol + 5% ether

Liquid Based cytology

- Better than conventional Pap smear
- uses cervical brush/ broom
- Fixative - Methanol / ethanol

* C/I for pap : None
Relative c/I : Bleeding.
During preg : Pap is done in 1st ante natal visit.

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- Alternative to pap in rural areas :

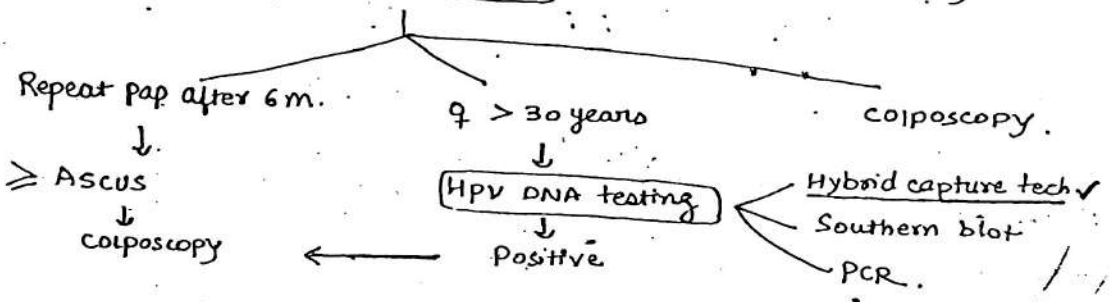
1. VIA - Visual inspection with 5% acetic acid - white/stained areas : Abnormal
2. VILI - " " Lugol's iodine - unstained yellow areas : Abnormal
(stained areas - Black-Brown).

* Lugol's iodine = 5% Iodine
* Schiller's " = 0.3% "

- Infections detected by pap :
  - Trichomonas
  - Candida
  - HSV
  - Actinomyces
  - HPV

Not detected : Gonorrhea & chlamydia.

- Result of pap smear :
  1. Normal
  2. Infection : Give antibiotic ; Repeat pap after 3 m.
  3. Reactive changes :
  4. Atypical squamous cells of unknown significance (ASCUS)



  5. LSIL
  6. HSIL.

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Management of LSIL

LSIL on pap smear.

↓

See Cx

- Lesion visible → Punch Biopsy
- No lesion visible → Colposcopy / colposcopic directed biopsy.

+/- Endocervical curettage.

HSIL { Lesion visible : Punch Biopsy; Lesion not visible : Colposcopy } + Endocervical curettage.

Colposcope

- Magnification : 5X to 30X.
- Focal length : 30 cm
- ~~Can't visualise endocervix.~~
- Invented by Hin salmann.
- OPD procedure ; No anesthesia needed.

First take Biopsy from any rough area or any area appearing white before applying acetic acid

↓

Now Apply 3-5% acetic acid

↓

Take Biopsy from acetowhite areas.

& Take Biopsy from any area where abnormal blood vessels are present (Mosaic / Punctate / Reticular vessels)

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Acetowhite areas.

When aceto-acetic acid is applied on cervix

↓

Normal epithelium - Pink

Dysplastic epithelium - White.

(Dysplastic cells have ↑ N:C ratio

∴ ↑ Nuclear proteins ⇨ coagulate in presence of acetic acid & appear white → acetowhite areas).

Suppose, Pap smear report shows HSIL. & colposcopy report shows Normal. → Problem could be in endocervix.

∴ In such case: Cone biopsy is done.

Cone shaped area of Cx is cut – It must include endocx, Tz & exocx.

It is an OT procedure. & done under GA.

Diagnostic indics of cone biopsy are:-

1) Discrepancy b/w Pap smear & colposcopy.
2) Unsatisfactory colposcopy.
3) Pap smear report is HSIL. & endocervical curettage is (+)ve.
4) Suspicion of Microinvasion / Adenocarcinoma.

Therapeutic indics:-

1) To T/t Ca in situ. or Stage IA1 of Ca Cx in young female

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Management of CIN.

CIN I

↳ follow up.

↳ Normally regresses in 2 years.

↳ If CIN I > 2 years

⇩

CRYOSURGERY

CIN II & CIN III.

At any age group.

⇩

LEEP

(Loop electro excisional procedure

LLETZ (Large loop excision of Transform zone)

In CIN + VIN = Laser

(vaginal IN)

Indications of hysterectomy in CIN.

1) Recurrent CIN
2) CIN c/w any pelvic pathology for which hysterectomy has to be done
3) Adenoca
4) Pt. can't come for follow up.

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Carcinoma cervix.

A pt. of Cx ca has 5 cm growth which extends to lower ⅓rd of vagina, hydrouter is present, on cystoscopy bullous edema of bladder mucosa is seen.

↳ Stage III B'

Management

Stage 1a1 → 1b1 = Surgery

- 1a1 : Conization or Simple hysterectomy
  1a2 : Wertheim hysterectomy
  1b1 : Radical " "

One more surgery which is done in ca Cx : Radical Trachelectomy.

- 80% of Cx + Parametrium + 1 cm of vagina + pelvic L.N. are removed
- Option for women ≤ stage IA1, IA2 & IB1 if tumor < 2 cm

Stage wise management :

- Ca in situ. & stage 1a1 ≤ lymph vessels are not involved.
  ↳ young female : conization
  old " : simple hysterectomy (TAH + BSO)
- stage 1a2 or stage 1a1 ≥ lymph vessels involved.
  ↳ young female : Radical trachelectomy + Pelvic lymphadenectomy
  ↳ old " : Type II / Wertheim's Hysterectomy + "
- Stage 1b1
  ↳ family complete : Type III / Radical hysterectomy + Pelvic & Paraaortic lymphadenec.
  ↳ family incomplete : Radical Trachelectomy + Pelvic & Para-aortic L.A.
  (But if tumor > 2 cm → Hysterectomy)

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Dysmenorrhea : Pain during menses

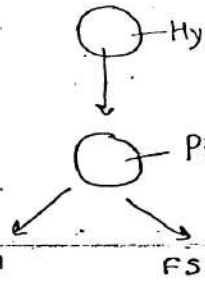
Primary		Secondary
No Pelvic pathology for Pain	–	Pelvic pathology responsible for pain · Endometriosis, fibroid
Seen in young girls at the onset of Menarche	–	Seen in Reproductive age g. (>30 yrs).
Rx : (1) NSAIDS. (2) Make the cycle anovulatory – OCPs.		Rx (1) Treat the underlying cause.

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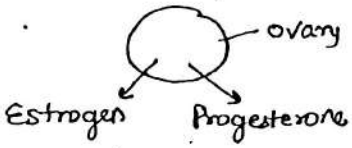
Amenorrhea = Absence of menstruation

Primary amenorrhea	Sec. Amenorrhea.
- Never experienced menstruation or Menarche.	- Earlier menstruating normally, but now absent menstruation
↓	
No menarche by 13 years of age in presence of sec. sexual characteristics OR By 15 years of age irrespective of presence of sex sexual characteristics	

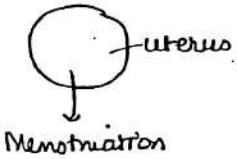


Kallman syndrome (46 XX)

Craniopharyngioma



Turner syndrome (45XO)
Pure Gonadal dysgenesis (46XX)
Swyers syndrome. (46XY)



MRKH syndrome (46XX)
Testicular feminising syndrome / Androgen insensitivity " (46XY)

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Mc cause of 1° Amenorrhea = Turner
2nd " " = MRKH
3rd " " = Testicular feminizing

Best Tx for 1° amenorrhea = Karyotyping
" 2° " = Hormonal study

MRKH syndrome (46XX) - Phenotype: Female

↳ Mullerian agenesis (Both Mullerian ducts absent).
Skeletal abnormalities (mc - cervical spine).
Renal anomalies (Renal agenesis (mc)
Horse shoe shaped kidney)
] TRIAD

↳ ∵ Mullerian duct is absent ∴ Uterus, FT, Cx & upper part of vagina are absent. (In some cases small part of FT may be present)

∴ ↳ No menstruation (∵ uterus absent)

OVARIES - Normal → ∴ Estrogen Normal : Sec. sexual characters (N)

Management

- These females can never menstruate
- ∵ they have short vagina → vaginoplasty is done

Technique
- Mc Indoe Technique
- Williams.
- Laparoscopic vaginoplasty
  ↳ Veichetti technique.

Time
just before/after marriage.

- These females can never become Pregnant. But they can have their own biological child (∵ ovaries are (N) → IVF + surrogacy).

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Clinical case I

- Whenever a ♀ presents ⊼ 1° amenorrhea ⊼ (N) Sec. sex characteristics (Breast dev.)

Imperforate Hymen (Cryptomenorrhea)

↳ C/O : (i) Cyclic abd. pain + No bleeding.

↳ O/E : Hymen bluish & bulging.

P/A – Mass (Uterus ⊼ blood collected in it → Hematometra).

P/V exam should not be done in virgin females.

On Per Rectal examination :–

Uterus is present & Bulky.

↳ Mgmt : Incision of Hymen :–

All the Blood will come out.

MRKH Synd. & Androgen Insensitivity syndrome

↓

Uterus absent.

Androgen Insensitivity syndrome (AIS)

(i) Complete androgen Insensitivity Syndrome

↓

TESTICULAR FEMINISING SYNDROME.

(ii) Incomplete androgen Insensitivity syndrome

(iii) Reifenstein Syndrome

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1. Complete AIS (46 XX). : Males have complete insensitivity towards Testosterone.

X linked

Gonads = Testes → Intra-abdominal ← Inguinal Hernia.
chances of malignancy.

Sertoli cell → Mullerian I.F. → Mullerian duct structures absent.

Leydig cells

Complete Insensitivity towards Testosterone

Wolffian duct structures absent
S
E
E
D.

Female Genitalia Present.

Spermatogenesis absent.

∴ Taken/considered as ♀ babies.

At puberty : Resistant to Testosterone.

2° sex characters of ♂ will not develop.

Gets converted to Estrogen → Proper Breast development (Tanner stage 4 & 5).

But pubic & axillary hairs will be less developed (Tanner stage 1 & 2)

♀ complaining of 1° amenorrhea. + Breast development proper + Pubic hairs absent; Inguinal Hernia present.

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AIS	MRKH
46 XY	46 XX
Gonads = Testes ↑	ovary
Barr body = Absent	Test = N Present
Not a/w Renal anomalies	a/w Renal anomalies. (∵ IVP is always done)

* Best test to differentiate b/w two = KARYOTYPING.

Management of AIS.

1. Let them be ♀
2. ∵ Intra-abdominal Testes → Gonadectomy is done.
   (Time : After breast dev. is complete)
   = 14-15 years.
3. Estrogen Replacement therapy.
4. They can't have their own biological child.
5. Vaginoplasty — Tech. & Time is same like MRKH.

2. Incomplete AIS.

- X-linked recessive
- less severe (little responsive to Testosterone)
- Genotype = (46 XY)

↓

Gonads = Testes. → Intrabdominal → Inguinal Hernia.

Sertoli cells	Leydig cells
↓	↓
MIF	Testosterone
↓	
Ut, Cx, Ft, Vag. absent	

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Time Responsive to Testosterone

Wolffian duct structures
Present, but underdeveloped.

♀ ext. genitalia
but resembles ♂
↳ clitoromegaly
↳ Partial fusion of labia.

These babies are considered as ♀ at the time of birth.

↓

At puberty : Testosterone

Partially ER sensitive.
↓
Virilizing features/Male like
(↑ muscle mass, Deep voice tone, etc.)

Estrogen.
↓
Feminising characters
(Proper breast dev.)

♀ c/ Proper breast development (2° sex character : N)
+ Inguinal Hernia + clitoromegaly

3. Refeinstein syndrome.

↳ They are males who are under virilized
↳ They have male genitalia, but bifid scrotum + Hypospadias.
↳ Int. genitalia
- S
- E
- E
- D

} tnt but less developed.

↳ c/o : Infertility.
↳ Not inherited as X-linked recessive.
↳ Male like bodies c/ Gynaecomastia.

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Clinical case II:

Pt. c/o 1° amenorrhea c absent sec. sex. characteristic.

- Height : short → TURNER's SYND.
- Height : (N) → Pure gonadal dysgenesis
  - 46 XX
  - 46 XY → SWYER's SYND.
- Kallman Syndrom (more common)

- In pure gonadal dysgenesis, the part of X chrosome responsible for formn of ovary is missing. ∴ They have streak gonads (although they have 46 XX). Physical characters of Turner's are absent here.

SWYER's SYNDROME.

↳ Example of Dysgenetic testes

Karyotype = 46 XY

- 85 : 1 cases = Idiopathic
- 15 : 1 cases = d/t inactive of SRY gene

↓ Testes

↓ Dysgenetic

Anti Mullerian Hormone - absent:

- FT
- Ut.
- Cx
- Upper vag.

+

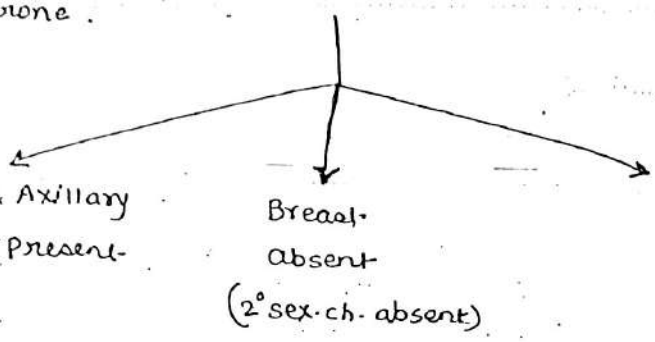
Leydig cells will not form Testosterone

- Wolffian duct regress.
  - S. E. D. D. - Absent
- female genitalia

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At puberty : Adrenals will produce small amounts of Testosterone.



↳ LH/FSH ↑

↳ 1° amenorrhea (No ovaries)

Mgmt : • Gonadectomy, as the diagnosis is made.
• Let them be ♀
• Estrogen alone for 1 year (for breast dev.) & Then Estrogen + Progesterone.
• They can become preg. ✗ → IVF ± donor eggs.

Kallman's syndrome :

↳ Hypothalamic failure + Anosmia

↳ can be seen in both ♂ & ♀

↳ Defect lies in : Kal 1 gene (on short arm of X chr.)
↓
Codes for Anosmin 1
↓
Responsible for migration of GnRH neurons from Olfactory epithelium to Hypothalamus.

↳ Problem : GnRH ↓ → LH/FSH ↓ → Estrogen ↓
→ sec. sex. ch. absent
→ Delayed Puberty
→ Infertility
Normal Ht. + Anosmia.

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Mngmnt :

↳ Estrogen + Progesterone / Gonadotropin / Pulsatile GnRH.

Secondary amenorrhea : YOUTUBE.

• Endometriosis

→ Occurience of Endometrial tissue (Stroma + glands) outside uterus.

Theories :-

MC :- ① Theory of retrograde menstruation / Implantation :- By sampson

② Coelomic metaplasia — By Meyer & Ivanoff

③ Metastasis :- By Halban

Predisposition

~~Risk~~ R/F : Mutation of KRAS

∴ If a family member is affected :- 7 times more chances of having endometriosis.

Age group :- 25-35 years / Rarely seen in pubertal females.

↳ If seen : Rule out Mullerian Malformations : By MRI

MC site : Ovary > Pouch of Douglas > uterosacral lig > Broad lig > FT.

* Endometriosis can occur anywhere in the body. (Lungs, Nose, Thoracic wall, etc.) It can occur in scars of certain surgeries i/a Scar endometriosis (like CS. scar, Myomectomy scar, Episiotomy scar, Pelvic floor repair scar).

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It is an estrogen dependent condⁿ. ∴ More common in Nulliparous, obese ♀. smoking protects.

In pregnancy → Progesterone → decidualization of endometrial tissue
↓
∴ Regress.

∴ Progesterone is used in mgmt.

Ix of choice : Laparoscopy

↳ CA125 levels ↑.

↳ USG : Chocolate cysts. / Endometrioma in ovary.
↓
Ground glass appearance.

On Laparoscopy : (i) Blue black areas on peritoneum : K/a powder burn appearance / Gunshot appearance.

(ii) White healed areas :

↳ C/F :

Mc. complain :- Pain (Sec. dysmenorrhea > chr. Pelvic pain > Dyspareunia > Low backache)

Severity of pain ∝ Depth of lesion

* 2nd most common complain : Infertility

Infertility is mostly because of defective folliculogenesis or defect in Ovum pickup. (ie. mostly d/t ovarian cause).

In severe cases, Infertility may be d/t ovarian & tubal cause.

* 3rd mc. complain : ovarian mass

* 4th " " : Menstrual problem (Menorrhagia)

↳ on P/v exam :- Tender uterus, Tender adnexa, Tender pouch of Douglas
- Uterosacral ligament on palpation :- Nodularity (cobblestone appearance)
- Uterus will be retroverted & fixed.

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Mgmt.

↳ If pain is minimal / mild pain : NSAIDS / OCPs.

↳ If Pain is moderate / severe :- Reverse the disease Process.

1st drug – Progesterone (POP/DMPA/Mirena/ implants).

DOC – GnRH (continuous).

Other drugs – Aromatase inhibitors (Letrozole)
Danazol (Androgenic side effects)
Gestrinone.

At which sites medical management not used :-

1. ovary (for t/t of chocolate cyst).
2. Bowel.
3. Urinary system.

Mgmt of Endometrioma.

- <3 cm → Laps electro coagulation
- >cm. → Lap.S. cholecystectomy.

Mgmt of Infertility

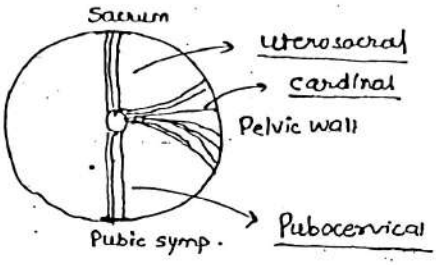
- Mild / Minimal disease → (unexplained infertility) → Clomiphere Citrate + IUI (X 3 times) → ↓ Fails. → IVF
- Severe / moderate disease → (ovarian + Tubal disease) → IVF

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Support of uterus :

I. Ligaments :-



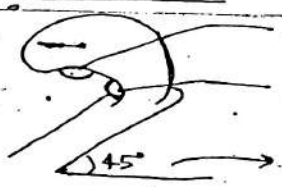
NOTE
1. Pubocervical ligament
2. Cardinal ligament or → Main
Transverse cervical lig. or
Mackenrodt's lig.
3. Uterosacral lig.

* Broad ligament doesn't support

II. Muscles :- (=Muscles attached to perineal body = Muscles cut during episiotomy)

1. Levator ani = Pelvic diaphragm → Main.
2. Superficial & deep transverse perenii (Urogenital diaphragm)
3. Bulbospongiosus
4. Ext. anal sphincter muscle
5. " Urinary " "

I. Mechanical support



Angle of anteflexion = b/w ut. & C_x = 120°
Angle of anteversion - b/w vagina & C_x = 90°
45° → Angle which vagina makes ∠ horizontal

In anteflexion : fundus Points towards bladder
In Retro " sacrum

Ligaments which prevent Retroversion : Uterosacral ligaments.
" " Promote anteflexion & anteflexion : Round ligament.
↓
∴ secondary support.

* for managing Retroverted uterus, Pessary used : Hodge smith Pessary.

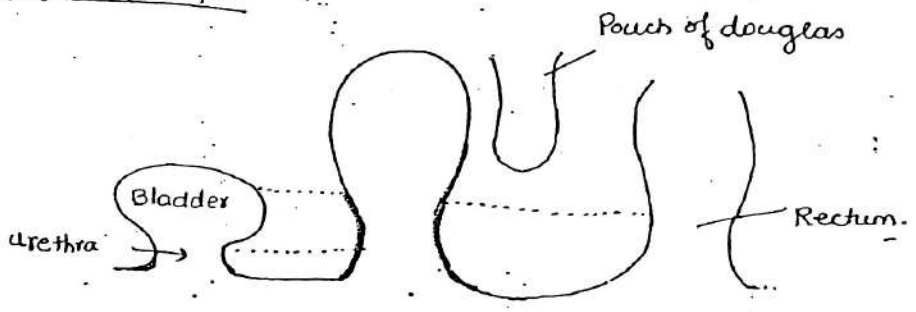
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De lancyes level of support of uterus.

Level 1	:	Uterosacral lig & Mackenrodt "	Defect leads to uterine prolapse.
Level 2	:	Paravaginal muscle Levator ani "	Defect of cystocele.
Level 3	:	Urogenital diaphragm & Perineal body	Defect of Rectocele & urethrocele.

Vaginal prolapse



Upper 2/3rd of Ant vag. wall prolapse : cystocele.
Lower 1/3rd " " : urethrocele

Upper 1/3rd of post. vag. wall prolapse : Enterocele
Middle 1/3rd " " : Rectocele
Lower 1/3rd " " : Laxed perineum

* Repair of cystocele & urethrocele : Ant. colporraphy
Repair of enterocele - 1. Moscowitz repair } Not done nowadays
- 2. (Halbane repair) }
'Mc Call culdoplasty' is used now to repair enterocele.

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Repair of Rectocele & laxed perineum : Post-colpoperineorrhaphy

Uterine prolapse : Prolapse is more common in elderly multiparous female.

If prolapse is seen in young nulliparous ♀ → Congenital prolapse

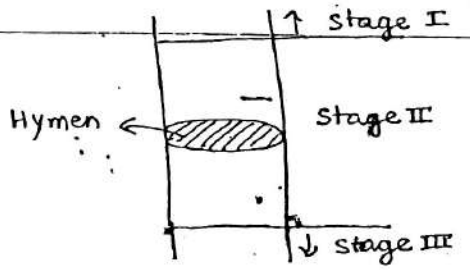
In congenital prolapse : cystocele is never seen.

↳ Risk factor :→ Marfan syndrome
Ehler-Danlos syndrome.
Spina bifida

Classifn of uterine prolapse : POP-Q classifn

Reference point - Hymen.

If prolapsed part lies >1 cm above hymen : stage I prolapse.
" " lies in 1cm of hymen (either side) : stage II "
" " lies > 1cm below hymen : stage III "



Mgmt :-

1) Congenital prolapse : Cervicopexy / sling surgery
2) If prolapse occurs in ♀ who desires ♱ child. : Cervicopexy / sling surgery

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Sling surgery

Material for making sling - Mercelene tape

except in purandare sling Sx - where fascia lata is used as a sling.

Name of Sx	sling attached @	Comments
1. Purandare sling (Not done now)	Ant to: Isthmus Post to: Post. Rectus sheath	- Type of dynamic sling - used in ♀ c good abd. tone - leads to retroversion of uterus & deepening of Pouch of douglas.
2. Khanna sling :	Ant to: Isthmus. ASIS.	Not done now.
3. Shirodkar sling	• cervix. : to ant longitudinal ligament on sacral Promontory on other side. —	- static sling. - can be done in ♀ ♂ who don't have good abd. tone - on the left side, sling pass below mesentry of sigmoid colon, it can lead to:- (i) Intestinal obstruction (ii) Injury to mesentric vessels/ureter/ Genitofemoral Nerve in Psoas muscle.

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Nowadays :-

Ompatite sling is done ; K/a Virkud sling.

- Rt side → Shirodkar's sling
- Lt side → Purandre sling.

If female < 40 years, who doesn't want any preg. in future, but wants to retain uterus for menstrual func^{n} →

Manchester surgery / Fothergill's repair.

- Amputation of Cx.
  - The raw area of Cx should be covered by vag. epithelium
  - ↳ K/a : 'Steurndoff suture'
- cut the cardinal lig. from side & tie them in front of uterus.

Complic^{ns} :- Incompetent os → ↓t : 2nd trimester abortion
∴ Cx fibrosis → stenosis → Cx dystocia (CBD)

* Shirodkar's modific^{n} of Fothergill's repair :- Cervical amputation is not done → ∴ can be done in ♀ who want preg. in future.

If ♀ > 60 years + H.D., DM. → Lefort's colpocleisis or Dani's repair
↳ Done under local anaesthesia

If ♀ > 40 years & doesn't want preg. & doesn't want to retain uterus. → Ward Mayo's vag. hysterectomy

Prolapse during pregnancy / purperium is managed by Ring Pessary.

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Ring Pessary - is not a cure for prolapse.

It is a temporary method, if Prolapse occurs -

(i) in Preg. ♀ (In this case, Pessary should be removed around 16-18 wks. → uterus becomes abdominal organ.)

(ii) in Puerperium.

(iii) unfit for surgery.

Pessary can lead to vaginitis & Dyspareunia. It has to be changed every 3 months. If not removed timely, can ↳ to VVF.

Pessary is kept @ level of Ischial spine

* Relative C/I of Pessary : Decubitus ulcer.
↳ caused by venous congestion
↳ Rx = Uterine Packing ~~with~~ with acriflavine (↓ antiseptic) & glycerine (↓ hygroscopic agent)

Vault Prolapse : Prolapse of vaginal stump after hysterectomy.

It is more common after Vag. hysterectomy than abd. hysterectomy.

Sx: 1. Trans abdominal sacral colpopexy
2. Trans vaginal sacro-spinous fixation
3. Utero sacral suspension (abdominally or Transvaginally).

* Colpo suspension is not done in vault prolapse. It is done in stress urinary incontinence.

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Urinary incontinence:

Dx : 1) Descent of urethra Neck of urinary bladder.
2) Intrinsic cause.

Mgmt :- Earlier concept

Bladder neck descent : Colposuspension

Intrinsic causes : Sling surgery (Aldridge / Mc guire sling)

Various tests were available, which differentiated b/w the above 2 causes :-

Boney's test, Q tip test.

Recent protocol:

Colposuspension.

Bursch colposuspension — Marshall Marchaitti Krantz surgery (MMK).

- Abdominal Sx.
- Proximal urethra/ Bladder neck is suspended to some pelvic structures.

Proximal urethra is Suspended to cooper's lig.

Proximal urethra is Suspended to Pubic symphysis (osteitis pubis : complica)

Gold std. surgery till date was : Bursh colposuspension.

Latest surgery : TOT (Trans-obturator tape) & TVT (Transvaginal tape)

Both are vaginal Sx. Middle urethra is suspended

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TOT is better than TVT because,

In TOT, retropubic space is not entered.

∴ Less bleeding & less complications

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Ovarian causes of infertility :

Tests for anovulation :

1. Indirect :

(i) Basal body temp. = ↑ by 0.5° in ovulation

(ii) cervical mucus study

Prog:- Ferning absent (Day 21) in ovulation

* Insler's score :

Appearance of ext. os.
Quantity of the mucus & } are seen.
ferning

In Modified Insler's score :- cellularity of mucus is also seen.

- If Insler's score > 12 : Good for sperms

(iii) Vaginal cytology

Estrogen → superficial cells
Progesterone → Intermediate "

* Maturation Index : The no. of parabasal & basal (a), Intermediate cells (b), & superficial cells (c) .— seen in 100 cells which are counted.

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Maturation Index represent - Hormonal index

Cornification index / Karyopyknotic index :- index represents no. of superficial cells, cornified under the influence of estrogen

(iv) Hormone estimation

If on Day 21, Prog. > Again 3 ng/ml. ⇨ ovulation has occured

Endometrial biopsy.

In Regular cycle : on D21.
In Irregular " : 7 days before expected day of menses
On D21, secretory endometrium : ovulated
Proliferative " : Not ovulated

Follicular monitoring

- Is done from D 10.
- done by TVS.
- Follicles grow by 2-3 mm/d.
- Just before ovulation, size of follicle = 18-20 mm.
In clomiphene induced ovuln, size = 19-25 mm
- After ovulation :- size suddenly ↓
Fluid in Pouch of douglas
Endometrium appears trilaminar

Direct evidence: Laparoscopy

* Most commonly done :- Follicular monitoring
* Best :- Endometrial Biopsy
* Easiest : Serum Progesterone level

Mgmt of anovulation :- In PCOD

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Decreased ovarian reserve.

→ less no. of follicles.

Test:

1) Day 3 serum FSH level.

Follicles are less
↓
∴ Estrogen ↓
↓
No negative feedback
↓
FSH ↑.

- FSH ≥ 15 : Poor ovarian reserve.
  ≥ 40 : Premature ovarian failure.

2) Serum Inhibin B level.

In poor ovarian reserve patients,
level of Inhibin B on D.3 < 45 pg/ml.

3) Anti Mullerian Hormone.

↳ Produced by Granulosa cells.

∴ In poor ovarian reserve: ↓ Anti Mullerian Hormone

This is best test for ovarian reserve, as it can be done on any day of the cycle.

4) Antral follicle count:

on day 3, using TVS
Follicles > 6 : (N)
Follicles < 4 : Poor ovarian reserve.

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⑤ Clomiphene citrate challenge test - D_3

on D_3 check the level of FSH.

- Give Ø clomiphene : D_5 to D_9

D_{10} : check FSH.

- A further drop of FSH, on D_{10} : Poor ovarian reserve.

Management :

IVF using Donor oocytes.

- Tubal Infertility

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Male infertility

1) semen analysis.

- Vol > 1.5ml
- PH = 7.2
- Total sperm count > 39 million per ejaculate
- Viscocity < 3
- Sperm conc^{n} > 15 million/ml.

  < 15 → oligospermia
  < 5 → Severe "
  No sperms in semen — Azoospermia
  No semen — Aspermia.

- Total motility > 40 %

  > 32 % should be progressively motile
  (< 32 % → Asthenospermia)

- Morphology — > 4 % should be Normal
  < 4 % = Teratospermia.

Oligospermia

Mild to Mod. oligospermia (5-15 million/ml)

If count is b/w 10-15 million/ml : IUI
5-15 million/ml : IVF

Severe oligospermia { < 5 million/ml) : ICSI

> 58 % of sperms should be viable
Leucocytes s/b < 1 million/ml
Round cells " < 5 " "

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Indications for doing IUI : Male :-

1) If sperm count = 10-15 million/ml.
2) If male has ejaculatory problems like - Hypospadias, Epispadias.
3) Retrograde ejaculation
4) If there

Female :-

1) cervical factor infertility
2) cervical stenosis.

IUI can be done ~ Husband's semen = IUI H.
or " : " Donor's sem = IUI D

Ix for IUI D :-

1) If male partner has genetic disease, which could be transmitted to off spring

0.5ml of washed, processed, concentrated sperms are technique

- Swim-up tech
- Swim down tech
- Density gradient centrifugation (Best)

} Methods of sperm Conc^n

IVF :

♂ → Sperm → Wash, process & Intubate for 3-4 hrs to Promote sperm capacitation.

♀ → Give clomiphene/Gonadotrope so that she Hyperovulates → Follicular monitoring. → At least 2 follicles are ≥ 17-18mm. & endometrium = 8mm. → Give ovul^n trigger (HCG) →

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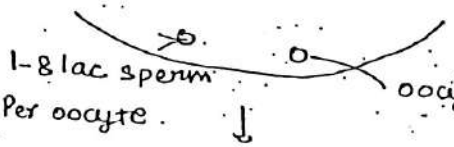
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After 24-36 hrs

↓

Oocyte Retrieval
Transvaginally under
USG Guidance.
(under GA)

↓



↓

Fertilisation

- Embryo transfer on Day 3.
- Blastocyst transfer on Day 5.
  ↓
  Better because it has lower failure rate.
  Maxm no. of Blastocyst that can be transferred = 3.

Site of transfer = 1.5 - 2 cm below the fundus.

- Success rate = 20-30 %.

Cumulative Success rate = 50 %.

Ix for IVF:
1) Tubal diseases
2) ↓ ovarian reserve — ± Donor eggs
3) Mullerian agenesis (MRKH) — IVF + Surrogacy
4) If ♀ has severe genetic disease & has a risk of transferring → ± Donor eggs
5) Oligospermia → Males

→ Females

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ICSI : Intracytoplasmic sperm injection.

Single spe

Single viable sperm → (injected into) Cytoplasm of oocyte.
(Mature metaphase II egg)

* Ix :-
1) Severe oligospermia
2) Motility < 5 % of sperms
3) In obstructive azoospermia, when sperms are surgically retrieved from Testes.

* Methods of surgical retrieval of sperms :

TESA = Percutaneous : Testicular sperm aspiration
TESE = Testicular sperm extraction
MESA = Microsurgical Epididymal sperm aspiration.
PESA = Percutaneous epididymal sperm aspir[n].

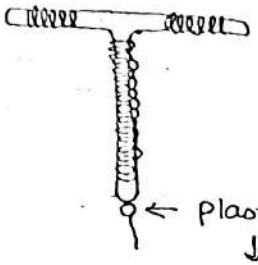
For testicular causes : Epididymal aspirn is not an option.

+ In semen analysis, the most imp. factor :-
Sperm morphology > motility > conc[n].

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CuT 380 A



← Plastic ball
↓
Prevents perforation.

380 = Total surface area of Cu wire.

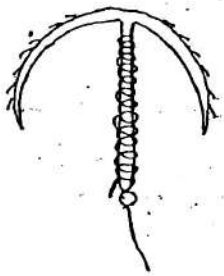
314 → Vertical stem
33 mm^2 → Each arm.

↳ Free of cost by GoI
↳ Releases Cu @ 50 mcg/day.
↳ Inserted by - withdrawal technique or No touch method
↳ Life span : 10 years.

CuT 380 Ag (silver)

↳ mc used CuT world wide!!
↳ Silver core
↳ No Cu wire on arms.
↳ Life span = 10 years

Multiload 375

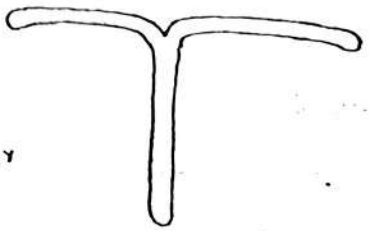


↳ Cu = 375 mm^2
↳ 2 flexible arms & fins (to keep it in its position)
↳ Arms are radiolucent
Stem is Radiopaque.
↳ Available in a preloaded inserter consist of a plunger.
↳ Life span = 5 years.

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Mirena



No copper wire.

↳ 52 mg of LNG ; elutes LNG @ 20 mcg/day.

↳ Life span = 5 years

↳ Decreases Blood loss ∴ used in menorrhagia

↳ T/t of dysmenorrhea, Endometriosis, Fibroid, adenomyosis

↳ HRT.

↳ Drawback :- Irregular bleeding

Can't be used as emergency contractive, unline Cu IUDs.

Essure



↳ spring like device

↳ Introduced in FT via vagina using hysteroscopy.

Inside FT → Initiates Tissue → Inflammation

↳ Success rate : 99%.

↳ Available in India.

↳ For first 3 months, use backup method

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Missing thread of IUDs

↓

Do a TVS to detect whether IUD is inside the uterus or Perforn has occured & it has gone inside peritoneum

- **Uterus**
  - ♀ wants to Continue using IUD → ↓ Do nothing
  - ♀ wants to discontinue → ↓ Remove using Shirodkar's hook or artery forcep or Remove using hysteroscope.
- **IUCD has led to perforn** (mc time for perforn : At the time of insertion)
  - Partly in uterus & Partly in Peritoneum → ↓ Hysteroscopy + Laparoscopy
  - Inside Peritoneal cavity → ↓ Laparoscopy or Laparotomy

If a ♀ becomes pregnant c IUCD.

3 options : 1) Abortion
2) USG Guided Removal of IUD
3) Continue her preg. c IUD in.

↓

(1) IUCD is not teratogenic.
(2) Complicns :- Abortion, Infecn, Pre-term lab, PROM.

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Emergency contraception:-

Best time = Within 72 hours of unprotected intercourse.
Can be taken upto 120 hours.

Drug used		Dosage.
Cu	= Copper containing IUCDs.	upto 5 days; Failure rate: 0-1%
OC	= OCPs.	Yuzpee method: High dose = 2 tab 12 hrs 2 tab; Low dose 4 tab 12 hrs 4 tab
M	= Mifepristone	— 15 mg
E	= Estrogen	— 15 mg
Lets	= LNG	— 0.75 mg 12 hrs 0.75 mg.
Catch	= Centchroman (saheli)	— 50 mg.
U	= Ulipristal (Ella) ↓ Unavailable in India.	— 30 mg single dose (can be given upto 120 hrs)

Most effective method : Cu containing IUCDs.
Most effective Hormonal method : Ulipristal > Levo.
Not used as emergency contractives : Mirena, POP, Misoprost.

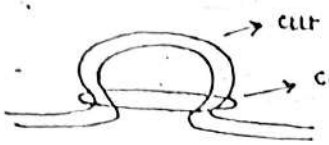
Female sterilization :

Eligibility criteria – 1 child 1 year old
Age b/w 22–44 years.

- Minilaparotomy (Small incision < 3cms)
  - MC technique : POMEROY'S
- Laparoscopically
  - Gas = CO_2
  - Intra-ab. pr = 8-12 mm Hg (Max : 15)
    - Fallopean rings
    - Clips

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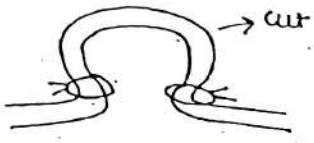
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Failure rate :- 1 in 300 to 400.

Tubal damage : 3-4 cm.

Modified pomeroy's technique



- Fallope ring (More common in India)
- Clips
  - Hulka clip
  - Filshe clip

Placed @ junc^{n} of proxima & middle 1/3rd of tube

- Failure rate = 0.2 - 1.3%.
- Tubal damage = 3 cms.

(* In clips tubal damage is 4-5mm. ∴ It is best suited method for Tubal recanalization)

In conventional methods, least failure rate is seen by:

Uchida method > Irving > Modified Pomeroy > Pomeroy.

* Mc method of female sterilization in India : Laparoscopic ligation
* Mc " Post partum ♀ steriliz^{n} in India : Minilaparotomy.
* Interval sterilization : Delivery ⟶ 6 wks ⟶ steriliz^{n}.
* Contraceptive ℂ highest risk in Ectopic preg : Sterilization.

(cautery > Madlener tech > Modified Pomeroy)

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Rh (-)ve.

Rh antigens discovered by - Landsteiner & weiner

5 Rh antigens : C, c, D, E, e. } short arm of chr. 1.

Main Ag.

+nt → Rh (+)ve

-nt → Rh (-)ve.

Preg. ♀ Rh (-ve) → check Husband's Rh status

Rh(+ve)

- Homozygous → 100% Rh (+)ve babies.
- Heterozygous → 50% Rh +ve, 50% Rh (-)ve

Analyse cell free fetal DNA present in mat. circulation

- Rh (-)ve. → Nothing to be done
- Rh (+)ve → further Ix.

Actual management.

Rh (-)ve female. getting preg. for 1st time.

Mother: (Rh-)

Antibody will form in mother.

↓

IgM (can't cross placenta).

↓ 6 months

IgG

↓

can cross placenta.

(But by that time baby is born.

∴ 1st preg = safe)

0.1ml

Fetal: (Rh+)

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In next preg: This rxn occurs fast

↓

IgG antibodies formed in mother

↓

Cross the placenta

↓

In fetal circuln Ag-Ab RKn occur

↓

Hemolysis

- **Fetal anemia.**
  - Sinusoidal HR pattern
  - Peak Systolic velocity ↑ in Middle cerebral artery.
- **Jaundice.**
- **Hepatosplenomegaly**
- **Placento-megaly**

HYDROPS FETALIS.

↳ Collection of fluid in 2 or more of the following compartments :-

1) Skin edema - can be seen on scalp - Buddha sign / Halo sign
2) Pleural effusion
3) Pericardial "
4) Ascites.

Other features seen, but not a diagnostic criteria :-

↳ Polyhydramnios
↳ Placentomegaly.

Hydrops fetalis. can occur dt

- Rh-ve preg ↓ Immune Hydrops fetalis
- other causes (Parvovirus infen, Thalassemia, etc.) ↓ Non-Immune Hydrops fetalis

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If Rh isoimmunisation occurs, the preg. ♀ will have PIH which leads to edema. Fetus has Hydrops fetalis which leads to edema in fetus.
∴ Both mother & fetus have edema → MIRROR SYNDROME.

In a Rh ⊖ve ♀ : Indirect coomb's test.
(@ Booking visit, 28 wks, 36 wks).

If ICT ⊖ve @ 28 wks :- Give anti-D to mother.
(300 µg = 1500 IU).
↓
Sufficient to neutralise 12 ml of feto-maternal hemorrhage.

* Feto-maternal hemorrhage can be estimated by : Kleihauer Betkey Test!

If Indirect coomb's test → (+)ve.
It means : antibody titre > 1:4 or 4 IU.
↓
Perform Monthly titre till 24 wks, then once in 2 wks.
↓
If antibody titre is > critical level, chances of Hydrops fetalis ↑

Titre = 1:4 to 1:16.
(4 to 15 IU/L)
↓
Moderate Risk

CRITICAL TITRE
- Titre > 1:32 (Ab > 15 IU/L)
- Albumin titre > 1:16

↓
Hydrops fetalis
↓
Check how much hemolysis has occured By Peak ↑ systolic vel. of MCA

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Peak systolic vel. of MCA.

< 1.5 MoM (Multiples of Meridian) → Deliver @ 37-38 wks

≥ 1.5 MoM → Fetal anemia (+)ve

- Preg < 34 wks. → Intra-uterine transfusion ('O' Rh-ve, Hb = 22-24 g/dL)
- Preg ≥ 34 wks → Deliver

* New concept: '34' wk replaced by '36' wk

Indications of Giving Anti D:

1. Prophylactically at 28 wks.

1st trimester

- After ectopic
- After H. mole evacuation
- All abortions > 12 wks
- Any abortion < 12 wk, where medical/surgical intervention was done
- CVS (chorionic villi sampling)
- After trauma

Later.

- Amniocentesis
- Trauma
- ECV/IPV
- After (N) del
- After C.S.
- Manual Removal of Placenta.

* Anti-D should be given to Rh +ve ♀ after delivery to protect her next Pregnancy

↓

Check fetus blood group = Rh+ve
Direct coomb's test = -ve

- Best time to give anti-D, after delivery ⇒ within 72 hours.
  Max^m within 28 days after delivery.

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Ovarian cyst & Pregnancy

- Mc ovarian cyst diagnosed in preg. – Dermoid cyst.
- Mc. ovarian tumor to undergo torsion in preg: – Dermoid
- Mc time for ovarian cyst to undergo " " – at the end of 1st trim / Puerperium.
- An ovarian cyst diagnosed during Puerperium s/b removed immediately.
- Mc ovarian malignancy in Preg: Dysgerminoma.

Management

Depends on size of cyst

- < 5 cm. → Wait & watch.
- 5-10 cm → MRI / USG
  - Solid areas Present → Operate in 2nd Trim.
  - Clear → Wait & Watch.
- > 10 cm → Operate in 2nd Trimester.

* In case of emergency (torsion, rupture) – do surgery irrespective of size of cyst. & Period of gestation.

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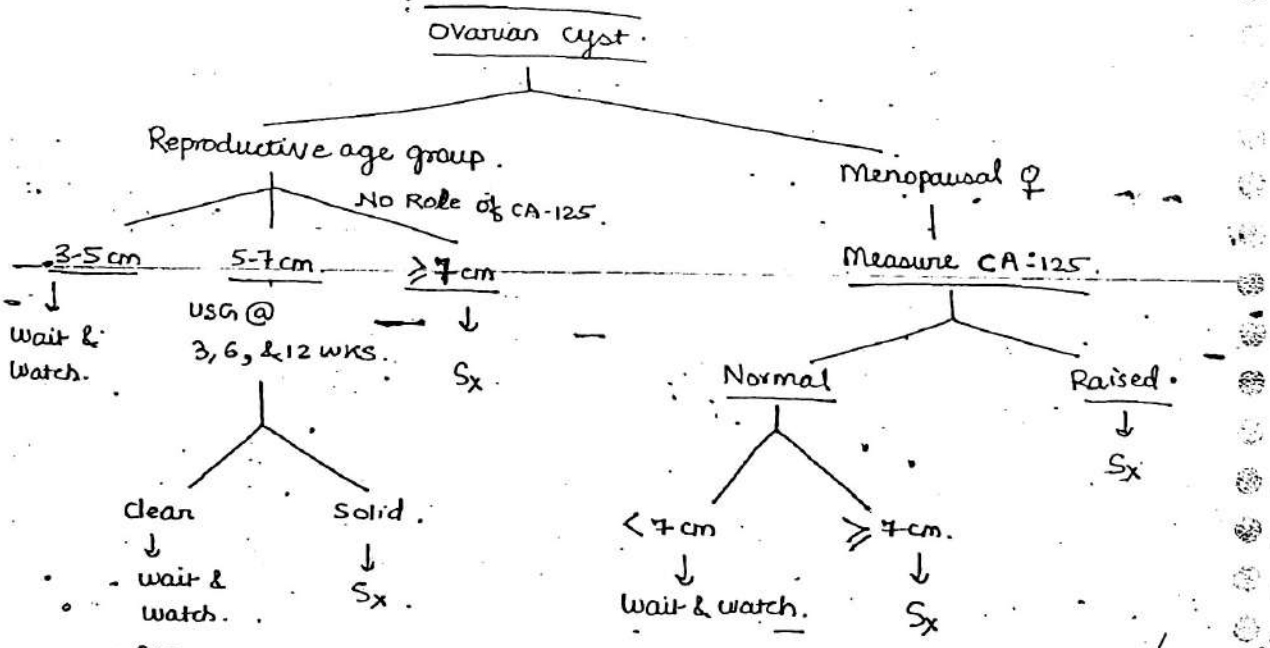
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Non-preg ♀ ℂ ovarian mass : Indic^ns for surgery :-

1. Any ovarian mass which has high risk features on USG.
   - Size > 10 cm.
   - Solid
   - B/L.
   - Ascites present
   - LN enlargement
   - Thick septa

2. Any ovarian mass > 7cms
   " adnexal mass > 10cms
   ] irrespective of age of pt

3. Raised CA125 levels in post menopausal ♀
4. Acute complic^n of cyst – Torsion
5. Diagnostic purpose.



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Twin Pregnancy

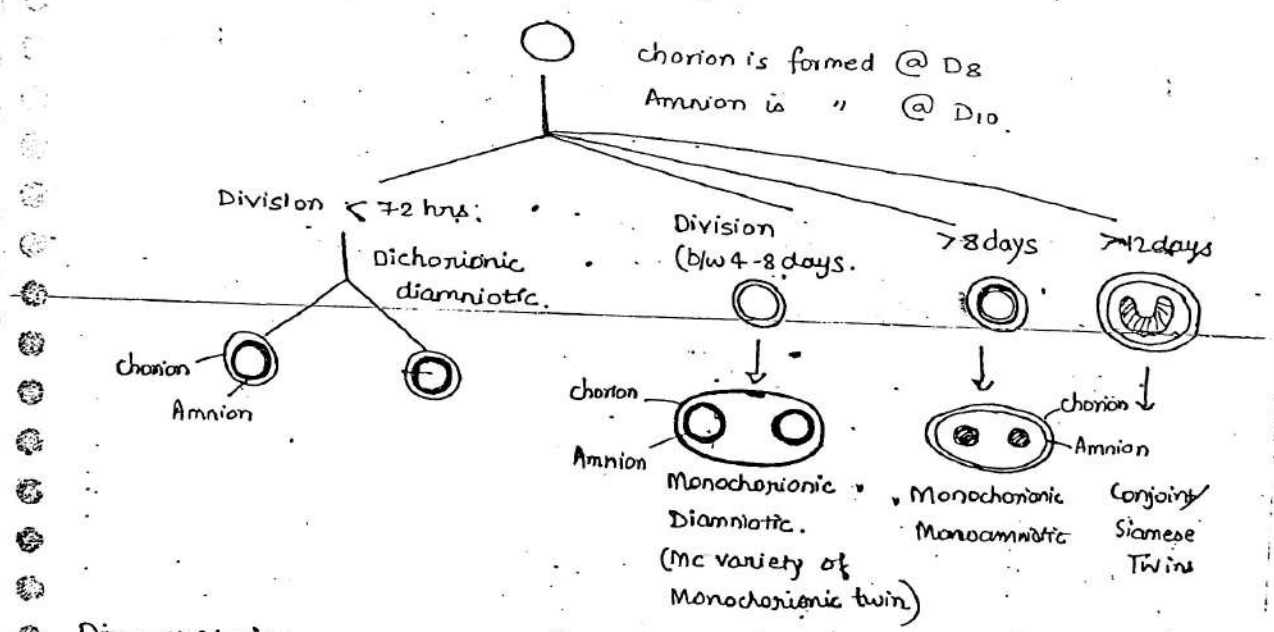
Dizygotic (more common)

- ♀ ♀ – ova
- ♂ ♂ – sperm.
- ↓ ↓
- Zygote zygote.
- ↓
- Each forms its own chorion & amnion. → Dichorionic Diamniotic.
- Same / Different sex.

Monozygotic.

- ♀ – ova
- ♂ – sperm
- ↓
- Zygote
- Divides
- Always have same sex.

Monozygotic twins.



Dizygotic twins

Highest incidence – Phillipens.
Lowest " – Japan

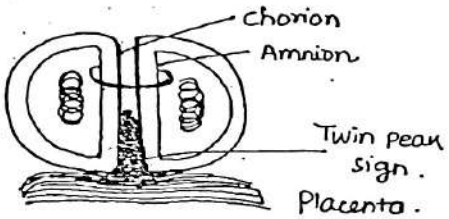
Monozygotic: Incidence is constant throughout the world (1 in 250)

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Incidence of twin is ↑ these days d/t < IVF

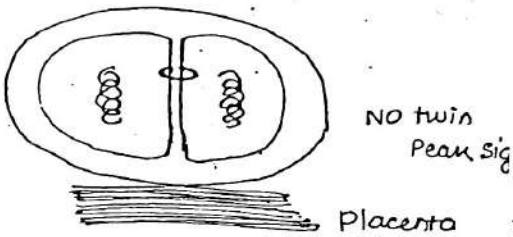
* Mc variety of twin Preg - Dichorionic diamniotic
* Mc " of monozygotic twins - Monochorionic diamniotic.

Dichorionic Diamniotic



4 layers
Thickness of memb. > 2m

Monochorionic Diamniotic.



2 layers
Thickness of memb. < 2.

* Twin peak sign is d/t CHORION
* Twin peak sign : 11-14 weeks (Best time to see)

Complications in Monochorionic twins :

1. Twin to Twin transfusion syndrome.
2. Twin anemia polycythemia sequence.

Complications only in Monochorionic Monoamniotic twin.

↳ cord entanglement.
∴ Absolute indication for c.s. (b/w 32-34 weeks).

Conjoint twins : Mc part that fuses : Thorax. — Thoracopagus.
↳ Always c.s. is done.

* Mc. fetal complication of twin preg. = Prematurity.

Time of delivery : In Monochorionic monoamniotic = 32-34 wks (c.s.)
In Monochorionic diamniotic : 36 wks.

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Dichorionic diamniotic : 37 wks.

* Twins ⊂ previous C.S. — C.S.

Twin to Twin transfusion syndrome :-

↳ Vascular connection is found b/w twins such that deep artery of one is connected to deep vein of another.

DONOR ⇨ RECIPIENT.

↓

- Oligohydramnios — Polyhydramnios
- Anemia — Polycythemia
- ↓ Growth. — DVT
- CHF — CHF
- — N. Growth.

Discordant growth.

↳ More common in Monochorionic diamniotic twins.
(* In MC MA twins : anastomoses are superficial
∴ No Twin to Twin TS)

Mgmt : Fetoscopic laser ablation of the anastomoses.

Staging : Quintero staging :-

Stage 1. - Poly/oligo sequence
- Bladder of oligohydramnios fetus can be seen, Doppler N

Stage 2. - Poly/oligo sequence
Bladder of oligo not seen., Doppler N.

Stage 3. - Poly/oligo sequence
Bladder of oligo not seen, Doppler Abn.

Stage 4 — Hydrops of one or both.

Stage 5. — Death of one or both.

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Antepartum fetal Monitoring

(1)

① Fetal Movements :-

Atleast 10 movts in 2 hours pd of Rest.

If pt c/o ↓ fetal movts :-

↓

NST (Non Stress Test) :- screening test

↓

Non Reactive.

↓

Biophysical score --- confirmatory test

Fetal Heart

① Baseline N FHR = 110–160 bpm

↓ < 110 → Bradycardia

↑ > 160 → Tachycardia.

② Beat-Beat variability = 5–25 bpm

③ Acceleration = ↑ in Baseline FHR by 15 bpm x 15 secs.

NST is said to be Normal = 2 accelerations (Reactive)

in 20 minutes

If FHR becomes fixed = Sinusoidal Heart Rate pattern. seen in fetal Anemia like

① Vasa Previa

② TTTS

③ Rh incompatibility

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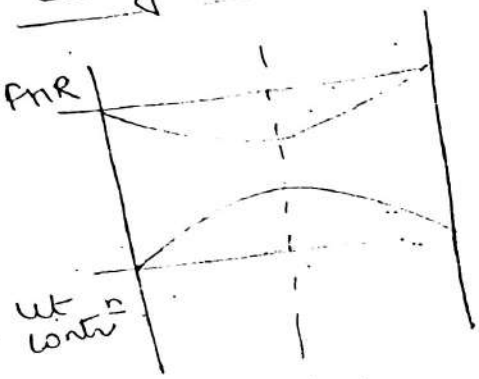
(4) Decelerations = ↓ in FHR by 15 bpm lasting for 15 sec.

Type I deceleration = Early deceleration

Type II " = Late "

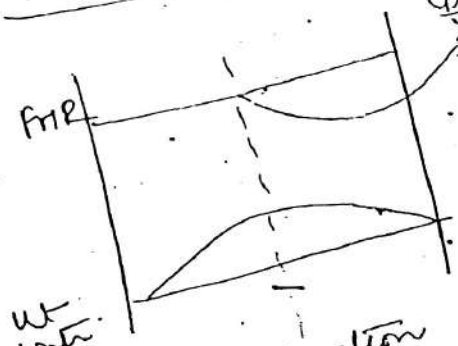
Type III " = Variable ".

Early Deceleration



- occurs Gradually >30 secs
- > seen in head compression
- ↓ physiological

Late Deceleration



Gradual >30 secs

> The FHR starts dipping at utrontraction & continues after contraction

> seen in utero placental insufficiency.

↓ Pathological

↓ Most ominous

Variable deceleration



- No fixed relation
- sudden fall in FHR / abrupt (<30 secs).
- Due to cord compression

↓ physiological

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(2)

When a female C/O = ↓ fetal movt

NST

1

- ≥2 accelerations in 20 mins → Reactive NST → Repeat NST weekly or twice weekly (72 hrs) in high risk pregnancy
- <2 accelerations but NO Late deceleration → Repeat NST for 20 more minutes i.e 40 minutes
  - ≥2 accelerations → Reactive
  - <2 acceleration → Non Reactive → BPS (confirmatory test)
- Late deceleration → Delivery

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Biophysical score / Manning score

- (done for ~~20~~ 30 minutes by USG)

Marker	Score +2	Score 0
T = Fetal Tone	1 flexion + 1 extension of limb	None
B = Fetal Breathing movt	≥ 1 breathing movt lasting for 30 secs	–
Meningites – Gross body Movt	≥ 3 body movt	–
Always – Amniotic fluid volume	Single vertical pocket > 2cms	–
Notorious – NST	Reactive	Non Reactive

Note: All markers of BPS – indicate acute state of fetus except – AF volume which is a long term marker (chronic state)

1st Marker to be affected = NST
↓
fetal breathing movt
↓
fetal Tone

In acute hypoxia

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(:)

- Biophysical score.

Normal

ie 10/10 or
8/10 with fluid Normal
(ie AFVal = 2/2)

} Fetus is Normal
Repeat BPP weekly

Management

Borderline

- 8/10 with ↓ fluid
or
6/10

} < If pregnancy ≥ 36 wks → deliver
If preg < 36 wks → Repeat BPS on same day. If it remains same → deliver.

Abnormal

≤ 4/10

Deliver
↳ usually LSCS needed

Modified BPS →
> Developed by Clarke
> NST + AF ~~outcome~~ measured
> ~~Processing~~ ~~Parameters~~ ~~etc~~ ~~prepared~~.
> If NST Reactive + AF ~~outcome~~ index ≥ 5 → Test is Normal.
> If test is abnormal → do a BPS

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General principles for Antepartum Monitoring

> Time for initiating monitoring = 32-34 weeks.
In high risk – it can begin = 26-28 weeks.

> In absence of obstetric GI to vaginal delivery, the vaginal delivery is the preferred mode of delivery. with continuous monitoring
If persistent late decelerations are present → LSCS done

Intra Partum Monitoring

(1). Continuous CTG or Intermittent auscultation of FHS.

FHS auscultation	Low risk	High Risk
1st stage	30 mins	15 mins
2nd stage	15 mins	5 mins

(2) Fetal scalp pH.

7.25 – 7.35 = N.
7.20 – 7.25 – Borderline
↓
Repeat in 30 mins.
< 7.20 – Cesarean / delivery

- Not to be done in HIV +ve females
- Avoid vacuum till 1 hr after it.

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Fetal distress term has now been replaced by Non Reassuring fetal states (4)

① CTG - • Persistent Bradycardia
• Persistent Late deceleration
• Persistent variable "

② Fetal scalp pH < 7.2

③ Liquor - meconium stained

④ Low apgar score at 1 minute of birth

Mgt of fetal distress

First stage
↓
C/section

2nd stage
↓
Station
- ≥ +2 → ↓ Forceps.
- < +2 → ↓ C/section.

Remember:
- At station ≥ +2, Forceps is quicker than cesarean.
- Ventouse Vacuum is not preferred in fetal distress

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Important Points on Anatomy

UTERUS

> Wt in Non pregnant female < Nulliparous: 50-70g, Multiparous = 80g

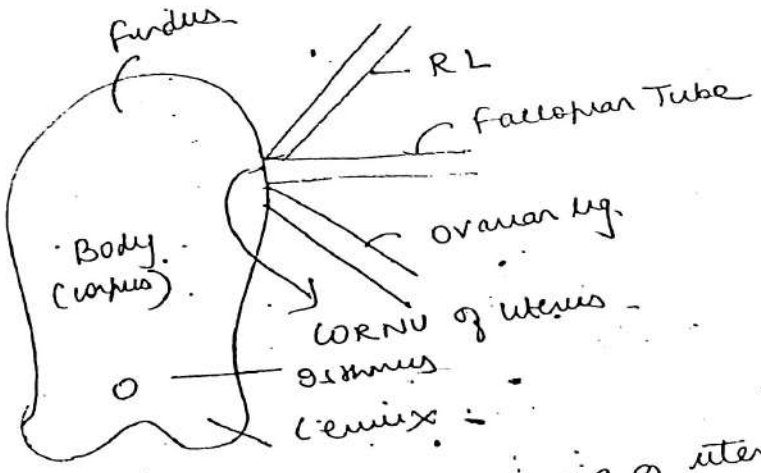
> Wt of pregnant uterus = 1000gm.

Q when does wt of uterus become Normal (Non pregnant like) after delivery = 6 weeks after delivery

> Length of Non pregnant uterus < Nulliparous = 6-8 cm, Multiparous 9-10 cms

> Length of pregnant uterus = 35 cms.

> Capacity of uterus < Non preg = 10 ml, Preg = 5000 ml



Structures attached to cornua of uterus.

Ant to Post:- R = Round lig
T = fallopian Tube
O - Ovarian lig

Superior to Inferior:- supero most is FT
Then RT & O at same level.

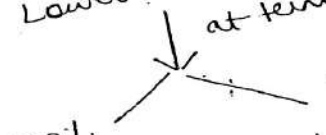
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> Round ligament –
- Derived from Gubernaculum
- Keeps uterus in anteverted position
- Attached to Labia majora
- Supplied by A, branch of uterine A runs within the ligament

> Canal of Nuck – fold of peritoneum in fetus that contains the Round ligament & ends into inguinal canal

> Isthmus :-
- Lies between Anatomical internal os & Histological int os
- Forms Lower uterine segment during pregnancy & at term is formed by



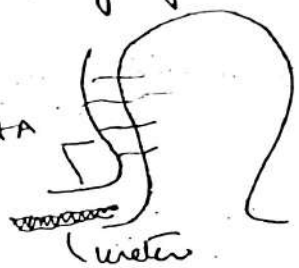
∴ Non pregnant length of isthmus = 0.5 cms

BLOOD SUPPLY OF UTERUS.
① Uterine A (mainly) – br of ant div of int Iliac A.
② Ovarian A.

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> Note :- 2cms lateral to Cx, uterine A lies just above ureter K/a water under bridge & it is the M/C site of uretric injury.
2nd M/C site = Pelvic brim



① Branches of uterine A to Uterus
V = (Ut A Branches)
A → Arcuate A.
R - Radial A
B - Basal A
S - Spiral A

② Uterine A - gives Sanpson A to supply RL

③ Descending vagino cervical A which supplies Cx & upper part of vagina.

Nerve supply = T10 - L1

Lymphatic drainage =
Body - Int iliac LN (M/C)
" Ext. "
Cervix - Superficial ing LN
Fundus - Para Aortic LN

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CERNIX

- Length in preg = 4cms
- Blood supply = Descending cervical A
- Lymphatic Drainage
  - I = Int iliac
  - H = Hypogastric LN
  - O = Obturator
  - P = ~~Proximal~~ Paracervical / uretric
  - E = Ext iliac
- Nerve supply = S_2-S_4

Cervix : corpus Ratio

Infants	1 : 1
Before puberty	2 : 1
After puberty	1 : 2
Reproductive age	1 : 3
Menopause	1 : 1

pH = of Cx = 7.8

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Vagina

Embryologically derived:—
Upper 3/5 part = M. Duct
Lower 2/5 " = Endoderm of urogenital sinus (sinovaginal bulb)

- Stratified squamous epithelium is derived from Endoderm of urogenital sinus
- Vagina doesn't have any glands
- Vagina has Doderlein bacilli
- pH of vagina

Reproductive age 3.5–4.5 (4–4.5)
During menstruation, Before puberty, After menopause 6.5–7.5
Newborn = 5.5–6

Blood supply :-

Upper 1/3 = Descending br of uterine A
Middle 1/3 = Int. Pudendal A
Lower 1/3 = Middle Rectal A

Lymphatic drainage

Upper vagina = Same as Cx
Middle vagina = Int. iliac LN
Lower vagina = superficial inguinal LN

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Pain during Labor

(1)

Early stages :- Due to ut contraction — T_{10}-L_1
Later " — " " Cx dilatation = S_2-S_4.
" stretching

Painless Labor: Epidural Analgesia
↓↓
DOC - Bupivacaine
↓
Level = T_{10}

During cesarean = Spinal Anesthesia
↓
Level T_4

For instrumental delivery = Pudendal N block
↓
Site = Ischial spine
↓
Ligament pierced = Sacrospinous ligament.

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Labour Q's.

Q1 G_2P_1 = Admitted in Labor at 8am on Monday with Cx = 2cms, 40% effaced
Next day At Till 10 pm → Cx = 3cms,
80% effaced
FHR = Regular
Membs = Intact.

Next step in Mgt. Ans = Antenatal test

Q2:- G_2P_1 = Admitted in Labor at 8 am on Monday
Cx = 2cms dilated, 40% effaced
At 12pm (Midnight) = Cx = 6cms dilated
Station = 0
80% effaced
At 3pm = Cx = 7cms effaced 80% effaced
Station = +1.
Membs = Intact
FHR = Regular
Ut contraction = +nt

Next step — ARM

Q3:- G_2P_1 = admit in labor at 8 am on Monday
Membs Ruptured →
Cx = 6cms dilated, 80% effaced.
No Meconium
FHR = +nt, Regular.
At 12pm (midnight) same findings.
Ut contractions = +nt, Adequates
4hrs passed

Mgt :- C. section

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Q4 :- G2 P1 = admitted in labor Room = (8)

Time = 8 am P/V = Cx = Fully dilated.
Mems Ruptured
Liquor = clear.
station of fital head = +2 1 hr passed

At 9 am: Prolonged labour signs findings. | Mgt. forceps

At 10 am - same findings. | Mgt.
Caput tnt
Moulding tnt.
Hematoma tnt | C section.

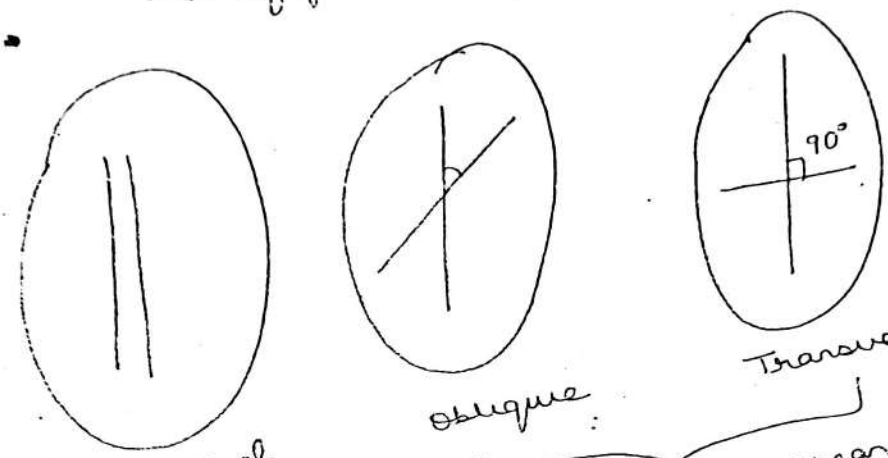
Second stage arrest
obstructed labour

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Important Terminology Related to Labor

Lie : Relationship b/w long axis of mother & long axis of fetus



Unstable lie

When lie is not fixed by 37 weeks

M/C cause = • Idiopathic
• Placenta previa
• Polyhydramnios
• Abn shape of pelvis.

Note - oligohydramnios can lead to malpresentation but it never leads to unstable lie).

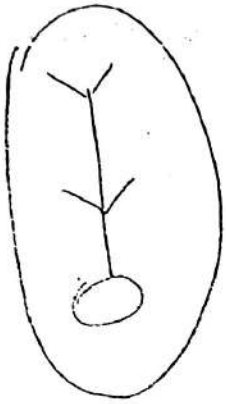
Mgt = cesarean section

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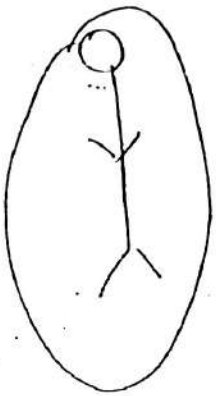
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(2) Presentation - Part of the fetus which lies in the lower part of uterus

→ Longitudinal lie



Cephalic presentation (M/C)

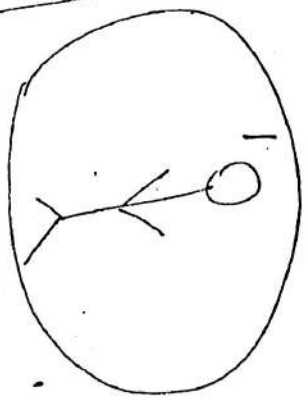


Breech presentation

Apart from cephalic presentation, rest all presentations are malpresentations.

M/C malpresentation - Breech.

→ Transverse lie



Presentation - Shoulder presentation

↓

Mgt

↓

cesarean section (whether the baby is alive / dead)

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Imp

Presenting part :-

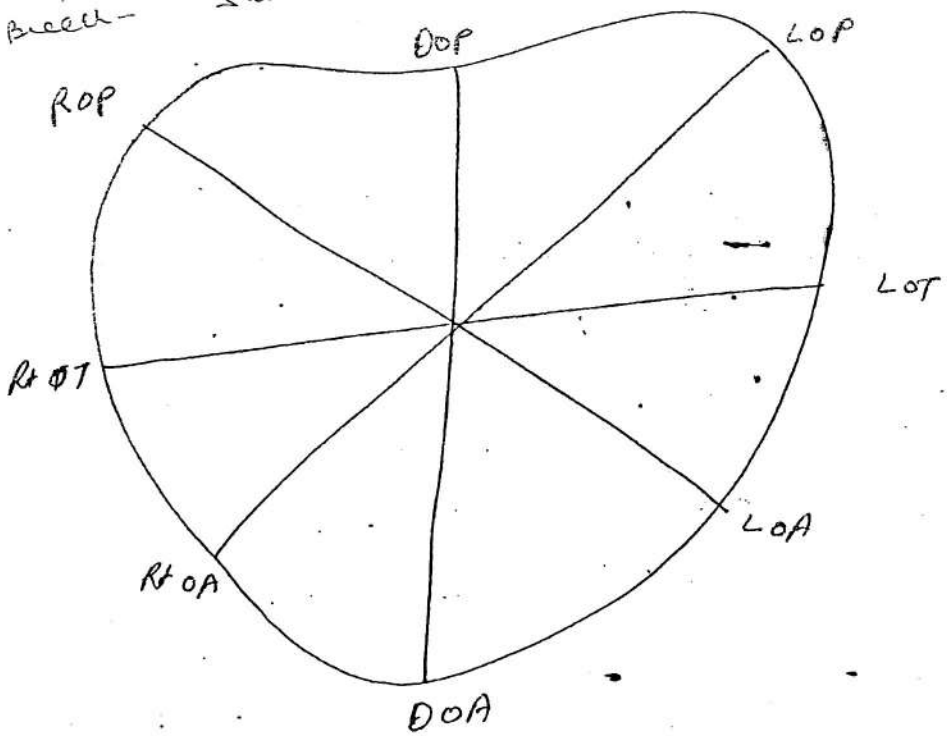
Part of the presentation which lies directly over the internal os -

- In well flexed head - vertex (MIC)
- In fully extended " - face
- In partial extended - Brow → always do cesarean section

Denominator

Bony fixed point of reference in presentation used to describe position -

Vertex - occiput
face - mentum
Brow - frontal bone
Breech - sacrum



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M/C position of fetus –

M/C " " " at onset of Labor/ Early Labor –

M/C " LOA " in Late Labor. LOA

M/C OA position – LOA

M/C OP " – ROP (6)

M/C position in breech – LSA

" " face – Left mento Anterior

" % of babies presenting with OP at the onset of Labor – 15–20%.

m/c Attitude of fetus is flexion

Parts of fetal skull vertex

└ part lie B/w Anterior fontanel and Posterior fontanel

(1) Vertex = B/w ant fontals and Post fontanels

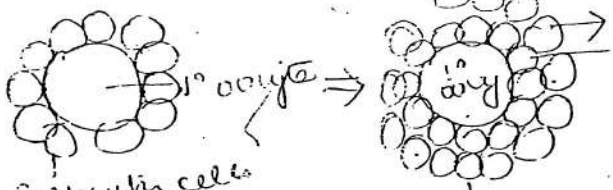
(2) Brow = ——— on one side and root of nose Supra orbital ridge on other side

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OVARIAN CYCLE

At puberty

Hypothalamus becomes functional
(" is formed @ 12wks of IUL)
↓
GnRH released in a pulsatile manner
↓
Ant Pituitary
↓
FSH released
↓
Acts on follicles of ovary



↓
Granulosa cells is released

- Estrogen
  - Proliferative effect on Endometrium (Regrows the function at layer of Endometrium which was shed during menstruation, from the basal layer)
  - Negative effect on FSH
  - +ve feedback on LH
    ↓
    LH suddenly ↑
- Inhibin B
  ↓
  Negative feedback on FSH

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Sudden ↑ in LH is called as LH surge

Q (For LH surge = Estrogen Levels should be 200 pg × 48 hrs)

↓

- Ovulation i.e 1° oocyte → 2° oocyte & rest of follicle is converted to Corpus Luteum
- Acts on Theca cells → Androgens released which are converted to Estrogen in Granulosa cells
- Luteinization of Granulosa cells → Progesterone released (Thus small amount of progesterone found before ovulation)

LH surge —(32-36 hrs)→ ovulation

LH peak —(24-36 hrs)→ ovulation

Q LH peak (serum) —12 hrs→ ovulation

Q In infertility = LH peak (urinary) —24 hrs→ ovulation

Q Initiation of LH surge is due to hormone → Estrogen

For maintainence of LH surge both = E + P needed

Note : At low levels

Q. Progesterone has a +ve feedback on LH & FSH :- just before ovulation there is both LH + FSH surge.

Q : When does synthesis of Progesterone begin in ovary = It begins at LH surge i.e 32-36 hrs before ovulation

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Corpus Luteum is formed.

↓ Secretes

↓ Progesterone (Mainly)

↓ Estrogen

↓ Inhibin A

∞ -ve feedback on FSH

↓ Supports the Endometrium (Secretory action)

↓ At high levels = Progesterone inhibits LH

↓ Levels of LH decrease

↓ CL Regresses (unless & until pt has become pregnant & hormone hCG rescued it)

↓ Progesterone ↓

↓ Release of PGF 2α (Vasoconstrictor)

↓ Cause constriction of Spiral arterioles

↓ Vasospasm

↓ Endometrial ischemia

↓ Necrosis of Endometrium

↓ Functionalis shed off (Menstruation)

↓ Hypomenorrhea (Pain)

↓ Estrogen ↓ → Inhibin A ↓

↓ Negative feedback on FSH lost

↓ FSH ↑

↓ New cycle begins.

> Corpus Luteum is maintained in non pregnant females due to hormone = LH

> Pregnancy due to hormone = LH

> Maximum size / activity of CL is 8 days after ovulation i.e (14+8 = 22) 22nd day of cycle

Q: When is progesterone Maximum ⇒ 22nd day of cycle.

Q Day of ovulation = Length of cycle - 14

Q Life span of CL = 12-14 days

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Remember

Estrogen

> Always has a negative feedback on FSH

> At LH
- Low conc → has negative feedback
- High conc → positive feedback

Progesterone

- At low conc has positive feedback on LH & FSH
- At high conc → negative feedback on FSH & LH.

Menstrual cycle

Normal length = ~~28-35 days~~ 24-35 days (population based) 24-38 days (study based)
~~(According to Novak gynae)~~
~~24-38 days~~
Avg = 28 days (only 15-1. of actually have cycle of 28 days)

Normal blood loss 5-80 ml
Avg blood loss = 35 ml (< 50 ml)

(N) Number of days women can bleed = 2-7 days.
Avg = 4.5 days

If there is any deviation from above it is called as Atypical uterine bleeding = AUB

(1) Menorrhagia = cycles at Regular interval.
Blood loss > 80 ml or ≥ 8 days of bleeding.

(2) Hypomenorrhea = cycles regular.
Blood loss < 20 ml or < 2 day of bleeding.

(3) Polymenorrhea = cycle length short < 24 days

(4) Oligomenorrhea = cycle length long > 38 days (or > 35 days)

(5) Amenorrhea = No bleeding for 90 days.

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(6) Metrorrhagia = Normal cycles + intermenstrual bleeding. ⑤

Causes of AUB : PALM COEIN classification

- P = Polyps
- A = Adenomyoma
- L = Leiomyoma
- M = Malignancy
- C = Coagulopathy
- O = Ovulatory dysfunction
- E = Endometrial cause
- I = Iatrogenic
- N = Not classified.

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Male & Female Differentiation (6)

What determines sex in a fetus:
others : Gene for Testis = SOX-9
" " ovary = Wnt T4 & Rspo1

SRY gene on short arm of Y chromosome. Main.

SRY gene +ve

Gonads → Testis (at 7 weeks)

Q: Gonads develop from = Genital Ridge

Q
1st feature which distinguishes 'T' & 'O' → formation of Testicular cords

Sertoli cells
- Müllerian inhibiting factor (7 weeks)
  - Glycoprotein
  - Has Paracrine action - acts locally
- Leads to Ipsilateral Regression of Müllerian duct in males by 9 weeks.

Leydig cells
- Testosterone (Production begins = 8-9 weeks. MAXM = 14-18 weeks)
  - Promote growth of Wolffian duct in males
  - 5α Reductase → dihydrotestosterone → Male External genitalia

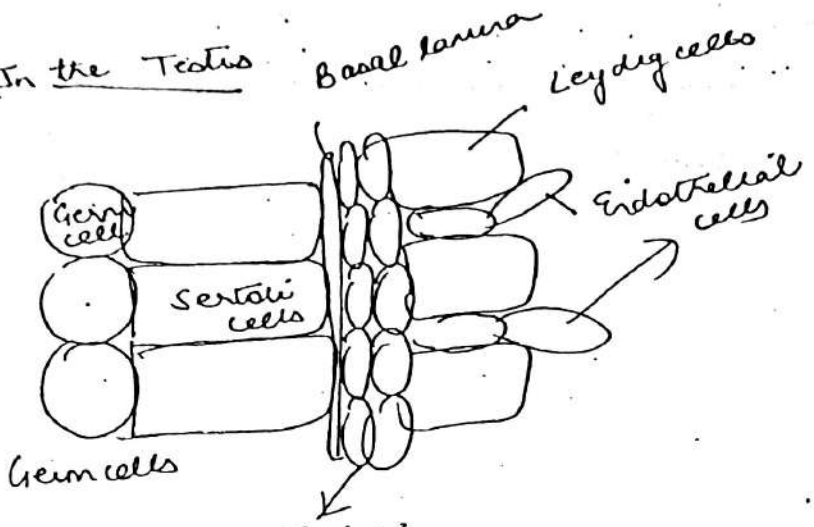
Remnant of MD in males
- → appendix of testis
- → Prostatic utricle

Wolffian duct in males
- S = Seminal vesicles
- E = Ejaculatory duct
- E = Epididymis
- D = Vas deferens

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In the Testis



The (tight junctions between sertoli cells create the blood testis barrier which keeps blood borne substances from reaching the germ cells & keeps

during spermatogenesis sperms are released

spermatogenesis occurs in seminiferous tubules of testes

↓ sperms

↓ Epididymis (attain maturity 10-14 days)

↓ Vas deferens

↓ ejaculation

↓ sperms released along with seminal vesicle fluid → Prostate gland fluid

bulbo urethral Gland (60:1 vol)

↓ ejaculatory duct → urethra

↓ Released

20-30 ml ejaculate

Healthy semen analysis = 1 ejaculate

but if no ejaculate till 60 min → do an urethral semen

If a woman's cervix is open (during ovulation) → ↓ volume → ↓ sperm motility → ↓ sperm survival in vagina

↓ CB & D

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In females :- ovaries are formed due to absence of Y chrosome
(i.e why in Turner = 45 XO = Gonads ovary)
↓
But for proper development of ovaries = XX needed
(i.e why in Turners = Gonads = streak)

> NO sertoli cells ⇒ NO Mulli nhib factor → MD grow into

Fallopian Tube
uterus
CX
upper 3/5 of vagina

> NO Testosterone :- { Wolffian duct Regresses
Ext genitalia of female develop

Remnants of WD in females

1. Hydatid of Morgagni
2 Kobelt Tubercle
3. Epo ophoron
4 Para-oophoron
5 Gartners duct

Q: All Remnants are present in Lateral part of Broad lig except Paraoophoron (medial part).

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Hermaphrodite

(8)

True –
Both ovary & testis in same individual.

Pseudo.
Gonads = one sex
Genitalia = other sex

Male Pseudo hermaphrodite
Genotype = 46XY
Gonads = Testis
External Genitalia = female
MC cause – Testicular feminizing syndrome (Androgen insensitivity syndr)

female Pseudo – hermaphrodite
Genotype = 46XX
Gonads = ovary
Ext Genitalia = Male
MC cause = Congenital Adrenal Hyperplasia
↓
MC Enzyme defect = 21 hydroxylase

Also Note
In CAH:- (classical cases)
17OHP = ≥ 3500 ng/dl (screening test)
After ACTH stimulation test = levels are ≥ 10,000 ng/dl

Q Pregnant female has CAH to prevent CAH in fetus → DOC → Hydrocortisone as it cannot cross the placenta and ∴ fetal HPO axis is not suppressed
Q In Pregnant female if female fetus has risk of / both CAH → DOC → Dexamethasone as it can cross the placenta

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TURNERS syndrome :-

(9)

Karyotype = 45XO

Gonads = streak gonads.

Most characteristic feature of Turner is short stature due to deletion of SHOX gene (short stature homebox containing gene)

Gonads are streak

↓

↓ Estrogen

↓

Lack secondary sexual characteristics
- nt Breast
- nt Pubic hair & axillary hair

↓

uterus hypoplastic

Note: Pure Gonadal dysgenesis = 46XX)

Mosaic = 46XO / 46XY
↓ streak
Abn functioning Testes

Stature is Normal (Ht = ")

Other features of Turners :

- webbing of neck
- Low post hair line
- shield shaped chest
- Widely separated Nipples
- CVS anomalies = M/C Bicuspid Aortic valve
- Renal anomalies = M/C Horse shoe shaped kidney
- Cubitus Valgus
- short 4th metacarpal
- Auto immune dis = Diabetes / Hashimotto Thyroiditis
- ↑ LH/FSH = ↑ (NO negative feedback). (Hypergonadotropic Hypogonadism)
- IQ = Normal
- lifespan ↓ = due to CV problem = decreased
- As they have ↓ estrogen :. they will have osteoporosis.
- In all pts of Turners syndr = Karyotype should be done CS if they are Mosaic = 45XO/46XY → then due to Y chr → gonads have high chances of developing GONADOBLASTOMA

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Puberty = ♀ = 10.5 years
♂ = 11.5 years.

Sequence in females

Growth spurt
↓
Thelarche
↓
Pubarche
↓
Peak height velocity
↓
Menarche

1st sign of puberty in females = Growth spurt

1st visible sign = Breast budding = Thelarche

1st sign of Puberty in males = Testicular growth

Tanner classified breast appearance & Pubic hair appearance in stages - Total 5 stages

Puberty
|
↓ Precocious Puberty (Males < 9 years)
[Females = Puberty < 8 years,
(M/C cause = 9 idiopathic
2nd M/C = Brain tumor ⇒ Hamartoma ∴ 9 in all cases do MRI)

2 types

Central
↑ GnRH
↑ LH/FSH
Premature activation of HPO axis
Always Isosexual
M/C cause = 9 idiopathic
DOC = Continuous GnRH

Peripheral
Not due to activation of HPO axis
↓
Some Estrogen/androgen producing Tm of ovary
↓
Can be isosexual/Heterosexual
Eg M/C cure Albright syndrome

↓ Delayed Puberty
♀ = No sec. sexual characteristic by 13 years / No menarche by 15 years
♂ = No testicular growth by 14 years

Note
Delayed Puberty is M/C in boys = M/C cause is constitutional

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Menopause

(11)

- Absence of menses for 12 months or 1 year
- Age = 51 years.
- In India = 47 years
- Delayed menopause = No menopause by 55 years.
- Premature menopause = < 40 years.

Menopause

↓

Ovarian failure

↓

Depletion of ovarian follicles

↓

↓ Estrogen

↓ Anovulation

↓ progesterone

↓ Amenorrhea

↓ Androgen produced by Theca cell of ovary

↓ Libido

↓ Negative feedback on FSH lost

↓ FSH ↑

Test for menopause

≥ 40 IU on 2 occasions 1 month apart.

↓ Bone Mass

↓ osteoporosis

↑ Chances of CHD

Senile vaginitis

Estrogen withdrawal

↓ Hot flashes (night sweat)

↓ MI/C symptom of Menopause

Note: Hot flashes is due to Estrogen withdrawal.
(Turners syndr pts have no hot flashes → because they never had Estrogen)

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In Menopause :-

Adrenals Normal → Androgen
↓ Peripherally converted to Estrogen

E1
↓
Main estrogen during Menopause

~~Hormonal~~ Estrogen

→ ~~Assessment~~

Diagnosis of Menopause

> FSH ≥ 40 IU
> Serum estradiol < 20 pg/ml
> Vaginal cytology = 100/0/0

Mgt of Hot flashes = HRT

D: Post hysterectomy pt = E
if with uterus intact = E + P
if with venous thrombo embolism with uterus intact has hot flushes = SSRI - Fluoxetine

Note: Raloxifene cannot be used in mgt of hot flushes as ~~the~~ de effect is ~~increase~~ hot flushes

Mgt of osteoporosis

DOC - Bisphosphonates.
other drug which can be used - Raloxifene.

Mgt of Senile Vaginitis -

Local estrogen cream.

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INFERTILITY

(10%)

In ability to Conceive Even after 1 year of Unprotected Intercourse

Female factors are responsible for Infertility by : 40-55%.

Male factors : 40%

Un Explained factors : 10%.

Female factors can be :

Ovulatory : 30% — Anovulation, Ovarian reserve

Tubular : 20-30%.

Uterine : 15%.

Cervical : 5%.

(Unexplained : 10%.)



Tests of ovulation :

1. Basal body temperature : Going to rise when progesterone given
   0.5°F (0.5-0.8)
   Biphasic curve indicate Ovulatory cycle

2. Cervical mucous : E - thin
   P - Scanty (Thick)

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(14)

D20 : Cork Screw Glands.
D22 : Maximum Stromal Edema
D24 : perivascular cuffing.
↓
Necrosis : D28.
on Histopath)

Classical Feature:
Lymphocytic Infiltration: C phase? → "Pre menstrual phase".
Seen by D24.

Endometrial Bx: L.P.D
• > 2 day lag b/w menstrual dating and HPE dating.

Ex:
D24 : Secretory change ≅ 20 day appearance : This is called as L.P.D
Luteal phase Defect

6th test
USG for follicular monitoring :
C USG? TVS.
Call pt alternate day
then
↓
Everyday



• Rate of growth: 2mm/day
• when will it rupture/mature follicle: 18-20 mm
• Sudden decrease in size of growing follicle & / Fluid in pouch of Douglas
↓
ovulated

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Mgt of Anovulation = Done in PCOS:
Ovulation inducing drugs

Q/ M.C.ly done for Infertility: TVS for follicular monitoring.
↓
- Antegrade test.
- Can tell pt. when to have Intercourse

Test of Ovarian Reserve:

① M.C. Test: Serum FSH level: on D_3 of M.Cycle
↓
Value: <10 IU : (N)
10-15 IU: Borderline reserve
>15 IU: poor reserve
>20 IU = Suggestive of premature failure
>40 IU: Dx of premature Ovarian failure

Follicle ↓ estrogen ↓
Inhibin ↓
FSH Level ↑

② S. Inhibin B Levels:
More follicle = more value
Done on D_3: level: <45 pg = poor reserve.

③ Clomiphene Citrate Challenge Test (CCCT):
On D_3 Sr. FSH measured
From: D_5 – D_9 : 100 mg of C.C:
on D_{10} : another Sample: S. FSH
High value on D_3 → which further ↑ on D_{10} : Indicates poor Ovarian reserve

(Theoritical) Not used these days

④ Best Test: Anti Mullerian hormone
produced by Granulosa cells of follicles
Best test for ovarian Reserve as it can be done on any day. In ↓ ovarian reserve = ↓ AMH

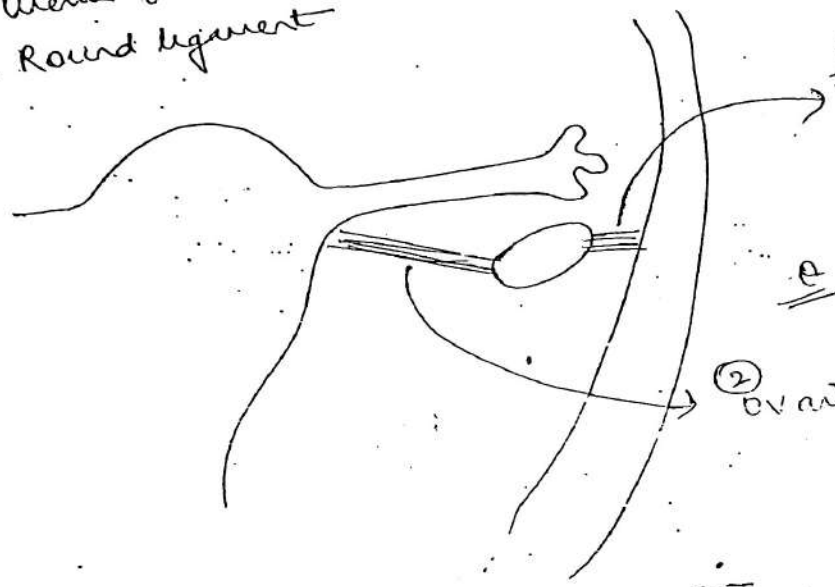
Male: TVF using Inner oocytes

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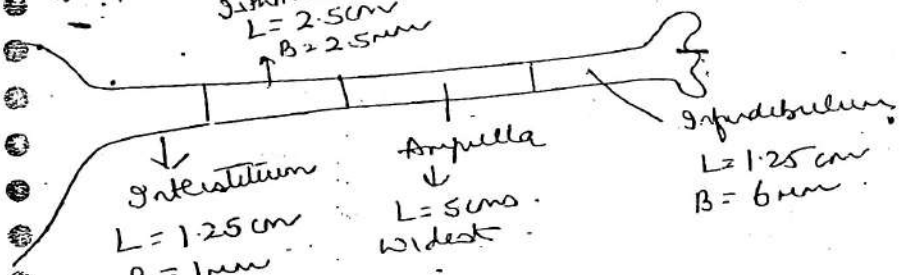
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Important Points on Anatomy of Ovary

1. Ovary lies in Pasat ovarian fossa of waldeyer in Lateral pelvic wall
2. In IUL it is present @ T10. & then descends down with help of Gubernaculum.
Uterus divides Gubernaculum into ovarian ligament and Round ligament



Important Points on Anatomy of FT.



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Fallopian tube contd

Phenomenon	site
fertilisation occurs	Ampulla
Ectopic preg M/C	"
Anatomic sphincter	Interstitial part
Physiological "	Isthmus
TB causes block in	Cornua
Gonococcus blocks	Fimbria

Lining epith = ciliated columnar epithelium
cells int = ciliated cells
secretory cells
Peg cells

TUBAL INFERTILITY

Cause = Endometriosis / PID / TB

IOC for assessing Tubal patency = HSG = (D5 - D11, Best - D10)

- OPD procedure
- Nonsurgical — Pt may have pain
- Cannula
  - Leech Wilkinson cannula

Absolute CI

1. Genital TB
2. Current pelvic infection
3. Pregnancy
4. Allergy to dye

Gold Standard :- Laparoscopic chromopertubation

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Tubal BLOCKS.

(NOTE = If one tube is patent & other blocked - No need for intervention) (18)

i) CORNUAL BLOCK
ii) MIDSEGMENT BLOCK
iii) DISTAL BLOCK.

(i) CORNUAL BLOCK.

Best prognosis as MC cause of BL cornual block is **Physiological**

↓

Mgt of cornual block

↓

Tubal cannulation under hysteroscopic or USG guidance (pass a guidewire).

+

Laparoscopic chromopertubation

↓

In most of cases block is seen Released.

↓

If not released

↓

It is pathological

MC cause of pathological BL cornual block - Genital TB. (Mgt = IVF).

(ii) Distal block

↓

Mild

↓

fimbrioplasty

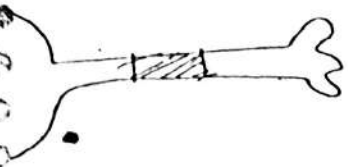
severe

IVF

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Midsegmental block



If in a pt of infertility
↓
HSG:- Midsegmental block
↓
pt has undergone sterilization
↓
Now wants Reversal

Prognosis of Reversal depends on
(1) Procedure for sterilization —
Ulps > Falloppe ring > Modified Pomeroy > Cautery
(2) Type of Reanastomosis —
Best with Isthmo-isthmic
(3) Total length of tube which can be reconstructed = ≥ 4cm - good

Q Before going for Reversal which Dx should be done
↓
Husband semen analysis

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Genital TB: Always a secondary infection.
M/C 1° site = lungs > Lymphnode.
M/C Route of spread = hematogenous
∴ If M/C PID in virgin ♀ = Genital TB
M.C. Site: 90% Fallopian Tubes > Endometrium (50-60%) > Ovary > Cervix

F.T : 90%.
Endo : 50-60%.
Ovary : 15-25%.
Cx. : 5-15%.

Least: Vulva/vagina : < 20%.

- Hematogenous route : ∴ B/L
- M.C: Ampulla (Least Common: Intramural part)
- In Acute phase: Tubes become Inflammed.
  Chronic phase: Tubes become Thick walled/ Adhesions
- HSG: findings: BIL cornual block
  LEAD PIPE Appearance
  But Remember: String of Bead appearance
  Tobacco pouch appearance
  Cobblestone appearance
  Hydrosalpinx
  Golf stick appearance

Endometrium: M.C. route of Spread: DIRECT SPREAD
- acute phase: absolutely (N) Looking
- Chronic Stage: Intra Uterine adhesions — Asherman syndrome or Pyometra

Ovaries: Presents as Tuboovarian abscess

Cx: TB of Cx is D/D for Ca. Cx. (∵ Bx & Culture always ⊖)

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(2)

M.C. presentation of Genital TB: Infertility > pain > Menstrual Irregularities

What % of pt's c TB are Infertile: Upto 75%

What % of pt's c Infertility have TB: { World: 10%, India: 17% }

1st Menstrual Complaint: Menorrhagia

M.C. Menstrual Complaint: Amenorrhea (or) Oligomenorrhea

B/c 1st phase is Endometrial => Menorrhagia (just like any infection)

Findings: P/v : M/C findings in reproductive age: Normal findings.

2nd M/C ✓ Tenderness

Reproductive age women & adolescent Girls

↓ B/L adenexal masses → Least Common Finding.

↓ Genital TB (M/C presents c: B/L Adenexal Masses)

Best Δx: Endometrial Bx (& for Acid Fast Bacilli Culture)

Rx: ATT x 6 months

Would she Conceive c ATT? → Yes (only in Mild Injury)

But Severe Injury: ATT can't help her: For Infertility: IVF.

ATT for: T.B

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Uterine Causes

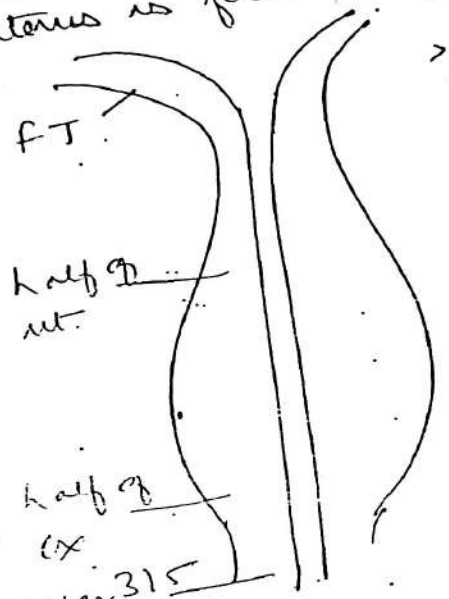
Can be due to congenital cause: septate / bicornuate uterus

Due to fibroid.

Important Points

Uterus is formed by MD.

- Both sides. MD begin to fuse = 7-8 weeks
- Fusion completes = 12 weeks.
- Septa formed.
- Septa Resolves by 18-20 weeks.
- Resolution & fusion begin from below upward direction.



Lower 2/5 vagina develops from sinovaginal bulb or urogenital sinus

Congenital Malformation

- Only 1 side MD is not → Unicornuate uterus
- Both side MD not fuse but fail to fuse → Uterus didelphys
  - FT = 2
  - Uterus = 2
  - CX = 2
  - Vagina = 2
- Both MD not fuse but fusion is incomplete → Bicornuate uterus (Always have single vagina)
  - 2 CX → Bicollis
  - 1 CX → Unicollis
- Septa lies b/w Re... → Se... fai... → uter...

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MIC in uterine Malformation

MIC malformation = septate > Bicornuate

MIC c/w abortion/infertility = septate.

Best Reproductive outcome = Arcuate uterus > Didelphys (Flat topped uterus)

Worst Reproductive outcome = Unicornuate

MC Ix for Sx on uterine malformation – if it leads to ≥3 abortions

IOC = MRI

Gold standard = Laparoscopy.

On USG Distinguishing features of septate & Bicornuate.

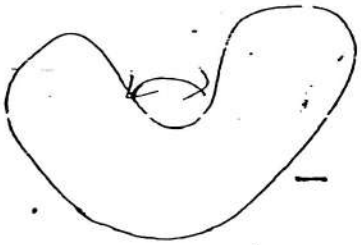


Bicornuate



septate

On USG



Angle = >60°

Distance b/w horns = > 4cms.

Mgt = Hysteroscopic inflation Sx



Angle <60°

Distance between horns <4cms.

Mgt - hysteroscopic Resection of septum

Earlier = Tompkins Jones Metroplasty.

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Male factor:

"HSA" - Husband semen analysis (128)

- Best way to obtain Semen for HSA: Masturbation.
- Done after Liquifaction of Semen.
- Max. time he can wait : 60 min (Should reach lab in 1 hour)
- avg time for Liquifaction : 30 min (20-30 min)
- After Liquifaction → Mixing → analysis.
- Ideal time of Abstinence : 2 days ; Max : 7 days.
- 2 Samples should be assessed (Dont Δx in One Sample)

WHO parameters: pH > 7.2

Lower limit Values:
- Volume > 1.5
- Total count > 39 million/ejaculate (Lower limit of (n))
- Sperm Concentration > 15 million/ml
- Total motility > 40%
- Progressive motility (forward) > 32%
- Morphology > 4%
- Vitality > 58%
- WBC count < 1 million/mL

(Ni) Avg count: 60-120 million/ejac.

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Confirmed ↓ if NO sperm on wet mount from centrifuged sediment x 2 times → Azoospermia

Check levels of LH / FSH Tests

Small vs Normal testes → Cryptozoospermia

↓	↑	↑
All ↓	LH ↑ FSH ↑ Test ↓	LH N FSH N Test N
↓	↓	↓
Cause is in HPO-axis	Testicular azoospermia	Post testicular obstructive
↓	↓	↓
Pretesticular azoospermia	Testicular biopsy DD a TESE + TESE Non obst	Fructose levels
↓		
Pubertile GnRH		

Fructose - nt several ↓

↓	↓
CBVAD (+ Amenorrhea iV)	Obst of ejaculatory duct
↓	↓
CFTR gene	TRUS / MRI

Mgt of Azoospermia

Vasectomy - Reversible & anastomosis

an obstructive - sperm retrieve by TESA MESA TESE PESA

↓

TESE

On TE NOA: Retrieve sperm from Testes only NT epididymis

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Ans:- (26)

Mild Sperm count = 15 million / ml → IUI

oligo spermia = IVF
(5–15 million)

< 5 million = ICSI

In azoospermia = sperms are not in semen
Not in testes if cause is obstruction

In that case :-

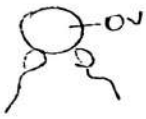
Sperms can be Retrieved from Testes using

1. MESA - Microsurgical epidymal sperm aspiration
2. PESA - percutaneous " " "
3. TESA - Percutaneous testicular " " "
4. TESE - Testicular sperm extraction

and then ICSI can be done

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Partial Mole	Complete Mole
Incidence In 695 Karyotype = • Triploid (69XXX or 69XXY) • Dispermic  Hence extra Genetic material is paternal in origin In 10% cases they are tetraploid or Mosaic	In 1945. • Diploid (46XX) • empty ova, Monospermic (90%) → sperm (duplicate). Hence all genetic material is paternal in origin. In 10% cases they are dispermic  empty ova (46XY)
All symptoms are less in partial mole. Presenting feature = Bleeding P/V in 1st trimester – nt	All symptoms more marked (same) → Pre eclampsia (27%) → Hyperemesis Gravidarum (25%) nt → Thyrotoxicosis (27%) → Resp. distress due to embolization (2%)
P/A: Ht of ut will be = or < POG.	Ht of uterus > POG Doughy consistency Fhs – nt Fetal parts not palpable

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Partial Mole	Complete Mole
IOC:- USG (TVS) USG picture similar to missed abortion	IOC- USG (TVS) Typical SNOW STORM appearance
BhCG levels- More than expected but not marked raised	BhCG >10.5 mIU/ml
Note: BhCG levels are used for follow up in Hmole after treatment	
Gold standard for diagnosis Histo pathological examination	
Findings:	
Hydropic degeneration - less marked	more marked
Some fetal part: +nt	-nt
Trophoblastic scalloping +nt	-nt
Inclusion bodies +nt	-nt
Theca lutein cyst -nt	B/L present
Progression to GT Neoplasia 3-5%	15-20%
choCA = <1%	4%

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Management

① Suction evacuation irrespective of gestational age

⇓

followed by curettage

⇓

Tissue sent for HPE (gold standard)

② If ♀ is more than 40 years & has completed family ~~- injection may be done~~ & is having excessive bleeding following evacuation → do hysterectomy.
Otherwise hysterectomy is not done as it doesn't eliminate the need for follow up

Other important points

- > Oxytocin drip s/not be started before evacuation as there is a risk of tumor embolization
- > Cannula size used = 10–12.
- > After procedure: Anti D given to Rh-ve females
- > Thela tablets, cysts don't need any separate T/t., may regress spontaneously after 2 months of uterine evacuation

Follow up :- in both partial & complete Mole.

Measure BhCG weekly

⇓

Till get 3 Normal values

⇓

Monthly till 6 months.

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In Partial Mole values of hcg come back to (4)
Normal in 7 weeks.
In complete Mole values of " " "
Normal in 9 weeks.

* Note
Pregnancy is CI × 6 months in Hmole.
contraception of choice = OCP's.

Prophylactic Chemotherapy
To prevent chorio CA. in Hmole patient is not given to all. It is given only in high risk patients

1. Pt age ≥ 40 years
2. hcg ≥ 10^5 IU
3. Large uterine size.
4. B/L Theca lutein cysts (≥ 6 cms)

DOC = Methotrexate
↓
If patient has Jaundice then DOC = Actinomycin D

Recurrence Rate after 1 molar pregnancy = 1% - 2%

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Gestational Trophoblastic Neoplasia.

Diagnosis:

If any 1 of the following is present:-

1) 4 consecutive values of hcg which show plateau (≤10%)
D1, D7, D14, D21

2) 3 consecutive rising values (>10%)
D1, D7, D14

3.) On HPE :- choCA

4) hcg levels remain high even after 6 months of suction evacuation.

Patient presents with:

- continuous bleeding even after evacuation
- can present with shock
- Uterine subinvolution.
- Persistence of Theca lutein cyst.
- Metastasis (lungs 80%.
  vagina 30%.
  pelvis 20%.
  liver / brain 10% each).

CHORIOCARCINOMA :-

- choCA develops M/C after which type of pregnancy - Hmole.
- high risk choCA develops M/C after ≤ type of pregnancy - Full Term pregnancy.

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Q. M/c GTN which is seen after full term pregnancy = choriocA.

histological characteristic - Absence of villi pattern
- Extensive proliferation of cytotrophoblast and syncitiotrophoblast with haemorrhage and necrosis.

Metastasis

M/c site lungs : on xray < M/c finding - cannon ball appearance
2nd M/c finding - snow storm appearance

- Metastasis of lungs means:- stage 3 of choriocA.

On vagina : M/c site of metastasis is submucosal.

- Metastasis of vagina means - stage 2 of choriocA.

Modified WHO scoring for choriocA

	Low Risk (score 1)	High Risk (score 2)
• Age	≤ 39 years	≥ 40 years
• hcg	$< 10^3$	$> 10^5$
• Type of antecedent pregnancy	Molar	Full Term
• Duration of antecedent pregnancy	< 4 months	> 8 months
• No. of metastasis	< 4	> 8
• site of metastasis	Lungs	Liver, brain
• size of Tm	< 3 cm	> 5 cm
• Previous H/O chemotherapy	single agent	Multiagent

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(7)

Management

If WHO score is ≤ 6 :- Low Risk choriocA

↓

single agent chemotherapy

↓

Methotrexate drug of choice

Follow up × 1 year

If WHO score is ≥ 7 : high Risk choriocA

↓

Multidrug chemotherapy

E = Etoposide
M = Methotrexate
A = Actinomycin
C = Cyclophosphamide
O = Oncovin

— called as Bagshawe Regime

Follow up × 2 years.

other important points

Figo staging

St. 1 — choriocarcinoma limited to Uterus

St. 2 — Vagina.

St 3 — lung mets

St. 4 — other mets like liver and Brain

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⇒ PSTT = It occurs in comby of Normal Pregnancy
tumour marker in PSTT is HPL and Human placental Alcaline phosphatase = It is chemoresistant tumour
Here the Tx is Hysterectomy
management of Invasive mole is Hysterectomy

⇒ PS2 kit 2 is a gene which is maternaly expressed

−	diploid	Komplete mole
+	Triploid.	Partial mole
+	diploid	abortion

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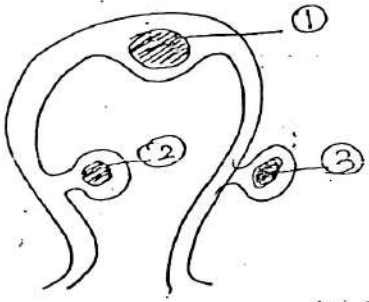
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(V)

Fibroid

> MC pelvic Tm in female
> MC solid benign Tm in female
* Estrogen + Progesterone dependant condition
  - Estrogen → causes proliferation
  - Progesterone → prevents apoptosis
  Hence fibroid
  MC in Nulliparous ♀, smoking is protective

Fibroid
- Intrauterine (MC)
- Extrauterine
  - cervical
  - Broad lig fibroid



1= Interstitial / Intramural
2) Submucous :-

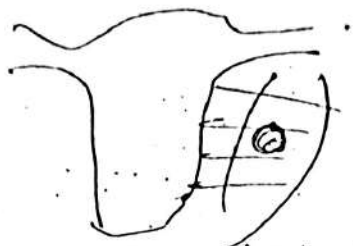
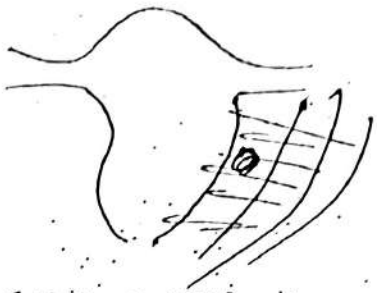
3 types

Type 0 :- fibroid entirely intracavitary [can be removed hysteroscopically
Type 1 = >50% intracavitary
Type 2 = <50% " (i.e >50% in the myometrium)

3 = subserosal

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Broad ligament fibroid can be (2)

True B.L. fibroid	Pseudo B.L. fibroid
lateral	medial
	
develops b/w layers of broad ligament.	Subserosal fibroid.

Important points

Age of fibroid = 35-45 years
MC in Nulliparous ♀
MC presentation in fibroid = Asymptomatic
MC symptom - Menorrhagia
(Menorrhagia seen in fibroid polyp)
To begin c all fibroids are - Intramural
MC variety of uterine fibroid - "
Fibroid c leads to maxm symptoms - Submucosal
" " has maxm chances of abortion - "
" c has maxm chances of undergoing malignancy - submucosal fibroid
MC extrauterine fibroid - cervical fibroid.
Fibroid with maxm urinary symptoms - cervical fibroid.

Ant Cx → ↑ urinary freq
Post Cx → urinary Retention

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(3)

Pain in fibroid

It is Never chief complain
- dysmenorrhea can be seen
- dyspareunia never seen
- Pain can be d/t Torsion
  ↓
  M/c in → subserosal pedunculated fibroid

Degerations :-

① fibroid is surrounded by a pseudo capsule: Blood vessels supplying the fibroid are seen in pseudo capsule, hence centre of fibroid is ~~too~~ least vascular ∴ degeneration begins from centre.

② M/c degeneration - Hyaline degeneration.

③ Least common - Sarcomatous change. (1c malignancy). seen in <0.5-1 cases.
- usually seen in postmenopausal female
- fibroid ↑ in size + pain
- ≥ 10 mitosis / HPF

④ Calcification
- begins from periphery as it is the most vascular part.
- seen in subserosal fibroid
  ↓ X ray
  Popcorn Kernel calcification

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Red degeneration

> seen during pregnancy (M/C in 2nd trimester) (9)
> Pt c/o acute abdomen in pain + Nausea + vomiting.
> Pathology:- Thrombosis in blood vessels supplying fibroid → Aseptic necrosis → fever + seen d/t Reactive causes cytokines and Oxygen

> Mgt: Conservative
No Role of
- Antibiotics
- Termination of pregnancy
- Myomectomy

Pregnancy complications of fibroid.

1. Recurrent pregnancy loss – M/C by submucosal fibroid.
2. Preterm labor.
3. Malpresentation
4. Abruptio
5. Abn Labour
6. PPH

fibroid can cause infertility ∵ (M/C = submucosal fibroid)

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IOC :- USG (5)

Fibroid	Adenomyosis
Chief complaints – menorrhagia. – uterus non tender	menorrhagia + dysmenorrhea tender uterus – Halban sign
– size + uterus = 16–20 wks Acc regular	not ⊙ > 10–12 wks. diffuse symmetrical enlargement of uterus.
regular contour of uterus	Regular contour + symmetrical thin uterus
IOC = USG	IOC → MRI. Gold standard → Histopath exam

USG appearance of Adenomyosis

1 salt & Pepper appearance.
2 myometrial cyst
3 Subendometrial cyst
4 Junctional blind appearance.
– Thickening of Junctional zone > mm

Management of Fibroid

Medical Mgt

↓ Sx's with improve symptoms but dont ↓ size of fibroid.
OC → OCPs (Low dose)
– LNG – IUCD – mirena
better OCP
– Tranexamic acid

↓ Sx's ↓ size of fibroid
GnRH agonist (Continuously)
↓ estrogen = menopausal
symp = add back up to prevent
small (2 + P)
– GnRH antagonist
– Mifepristone
– Ulipristal
– Aromatase inhibitor

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Drugs which ↓ blood loss but not size of fibroid.	Drugs which ↓ size of fibroid. (6)

Surgery

Conservative Sx → Myomectomy

Radical Sx → Hysterectomy

Myomectomy →
- Hysteroscopically (Morcellation) → Type 0, Type I submucous fibroid < 5 cms
  - Complications: Fluid Retention, Bleeding, Infection, Uterine perforation
- Laparoscopic
- Per abdominally
  - Best all fibroids
  - Laparoscopic better in all Respect except it can lead to higher recurrence rate

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Non Invasive method To treat fibroid (7)

1. Uterine artery embolisation
2. Magnetic Resonance guided high intensity focused ultrasound (MRgHIFU).

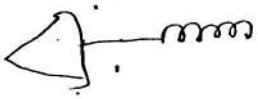
These both are done in females who dont desire future fertility

Ulipristal :-

→ selective Progest Receptor modulator.
— main use emergency contraception.
Brand — ⊕ ELLA taken ☐ in 5 days of UPSI.
— more effective than Levonorgestrel.
— also ↓ se bleeding & fibroid size.
(VE) → Abd pain

* ⊕ of Q want → to concieve infer → myomectomy.
— family incomplete.
→ Infertility + fibroid.
— Recurrent abortion d/t fibroid.

Bonney's myoma screw
Bonney's myoma clamp



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